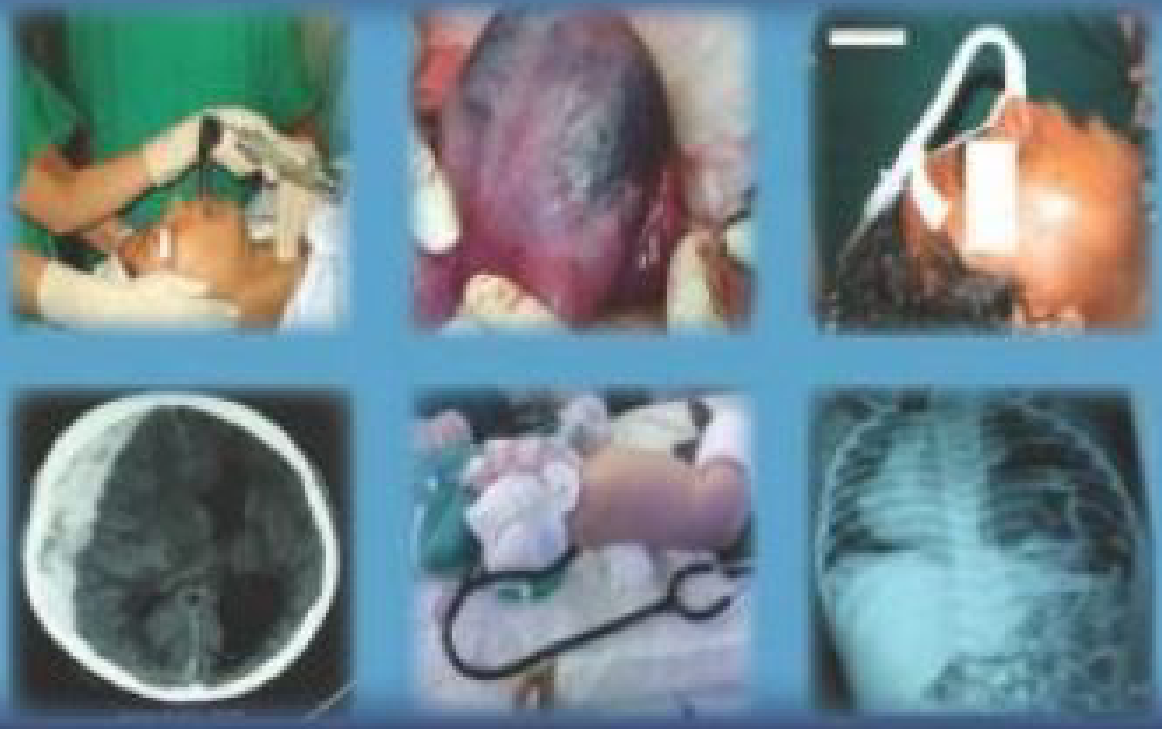
Manju N Gandhi
Anila D Malde
Amala G Kudalkar
Hemangi S Karnik

A Practical Approach to Anesthesia for Emergency Surgery



Forewords
Hemalata R Iyer
Sanjay Oak

JAYPEE

A Practical Approach to Anesthesia for Emergency Surgery

A Practical Approach to Anesthesia for Emergency Surgery

Editors

Manju N Gandhi MD DA
Ex-Professor and Head
Department of Anesthesiology
LTMMC and LTMGH, Sion, Mumbai, Maharashtra, India

Anila D Malde MD DA
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion, Mumbai, Maharashtra, India

Amala G Kudalkar DA MD DNB
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion, Mumbai, Maharashtra, India

Hemangi S Karnik MD
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion, Mumbai, Maharashtra, India

Forewords

Hemalata R Iyer

Sanjay Oak

JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD

New Delhi • Panama City • London

Published by

Jaypee Brothers Medical Publishers (P) Ltd

Corporate Office

4838/24, Ansari Road, Daryaganj, **New Delhi** 110 002, India
Phone: +91-11-43574357, Fax: +91-11-43574314
Website: www.jaypeebrothers.com

Offices in India

- **Ahmedabad**, e-mail: ahmedabad@jaypeebrothers.com
- **Bengaluru**, e-mail: bangalore@jaypeebrothers.com
- **Chennai**, e-mail: chennai@jaypeebrothers.com
- **Delhi**, e-mail: jaypee@jaypeebrothers.com
- **Hyderabad**, e-mail: hyderabad@jaypeebrothers.com
- **Kochi**, e-mail: kochi@jaypeebrothers.com
- **Kolkata**, e-mail: kolkata@jaypeebrothers.com
- **Lucknow**, e-mail: lucknow@jaypeebrothers.com
- **Mumbai**, e-mail: mumbai@jaypeebrothers.com
- **Nagpur**, e-mail: nagpur@jaypeebrothers.com

Overseas Offices

- **Central America Office, Panama City, Panama**, Ph: 001-507-317-0160
  e-mail: cservice@jphmedical.com, Website: www.jphmedical.com
- **Europe Office, UK**, Ph: +44 (0) 2031708910
  e-mail: info@jpmedpub.com

A Practical Approach to Anesthesia for Emergency Surgery

© 2011, Jaypee Brothers Medical Publishers

All rights reserved. No part of this publication should be reproduced, stored in a retrieval system, or transmitted in any form or by any means: electronic, mechanical, photocopying, recording, or otherwise, without the prior written permission of the editors and the publisher.

This book has been published in good faith that the material provided by contributors is original. Every effort is made to ensure accuracy of material, but the publisher, printer and editors will not be held responsible for any inadvertent error(s). In case of any dispute, all legal matters are to be settled under Delhi jurisdiction only.

First Edition : **2011**

ISBN 978-93-5025-070-9

Typeset at JPBMP typesetting unit

Printed in India

Contributors

Aklesh S Tandekar
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *drakleshtandekar@gmail.com*

Amala G Kudalkar
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *gurukudalkar@yahoo.co.in*

Anila D Malde
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *dradmalde@yahoo.com*

Aparna A Nerurkar
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *aparnan1@yahoo.co.in*

Deepali P Thakur
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *pankjat@rediffmail.com*

Deepti M Kotwani
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Maharashtra, India
E-mail: *drdeeptiagrawal@gmail.com*

Devangi A Parikh
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *sdevangi10@rediffmail.com*

Devendra W Thakare
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *devendra.thakre@gmail.com*

Dhawal R Wadaskar
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *dhawal104@gmail.com*

Geeta A Patkar
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *patkargeeta@gmail.com*

Hema B Gupta
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *drhemabg@gmail.com*

Hemangi S Karnik
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *dr_hemangi@hotmail.com*

Jyoti S Magar
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *jyotimagr@rediffmail.com*

Kamlesh G Gotiwale
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *kgotiwale@yahoo.com*

Kanchan R Rupwate
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
rupwatekanchan@rediffmail.com

Manish B Kotwani
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
drmanishkotwani@hotmail.com

Manju N Gandhi
Ex-Professor and Head of the
Department
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *drmanjugandhi@hotmail.com*

Naina P Dalvi
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *drnaina@rediffmail.com*

Nazmeen I Sayed
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *nazmeensayed@yahoo.co.in*

Nilam D Virkar
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *nilamvirkar@rediffmail.com*

Nishant S Bawankule
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
nishant_bawankule@yahoo.com

Preeti Rustagi
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
drpreetirustagi@rediffmail.com

Shakuntala N Basantwani
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
drshakuntala@hotmail.com

Shalaka A Gandhi
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
drgshalaka@yahoo.co.in

Shantanu B Kulkarni
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
drshan70@reiffmail.com

Smita S Lele
Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail:
smitaslele@rediffmail.com

Sohini S Shah
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *drsohini@rediffmail.com*

Swati S Chhatrapati
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *dr.swatich@gmail.com*

Trupti S Pethkar
Associate Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *truptipethkar@yahoo.co.in*

Urvi H Desai
Assistant Professor
Department of Anesthesiology
LTMMC and LTMGH, Sion
Mumbai, Maharashtra, India
E-mail: *urvi_desai@hotamail.com*

Foreword

It gives me great pleasure to write the foreword to this book *A Practical Approach to Anesthesia for Emergency Surgery*.

Anesthesia for patients with or without co-morbid conditions constitutes an important part of our emergency work. Different types of emergency cases that one can come across and all the perioperative aspects of emergency anesthesia are covered in great detail in this book and treatment options are provided. This will aid the residents to handle a wide range of problems that may arise in emergency situation.

The key points outlined at the beginning of each chapter are very interesting and is a welcome addition for a 'quick-read'.

The effort involved in compiling a book by a team of staff members from a single department is truly commendable. My congratulations to all the contributors and special appreciation to the editors who have done a great job! It is indeed a heartening fact that most of the contributors have been my students and/or colleagues at various points in my career.

I strongly recommend this book to all the residents in training and also to all members of our fraternity. The moderate pricing of the book should make it possible for it to be an addition in individual collection as well as in the library. A copy available in OR area 24×7 would be well appreciated by the resident community!

Hope you enjoy reading it as much as I did. Happy reading!

Hemalata R Iyer
Professor and Head
Department of Anesthesiology
TN Medical College and BYL Nair Charitable Hospital
Mumbai, Maharashtra, India

Foreword

As a clinical administrator of a busy university teaching hospital, one gets involved in various exercises which relate to planning, forecasting and execution. Civil projects take years to materialize but academic projects at times take even longer too (metamorphose). It really takes one's lifetime to put one's expertise and perspective into practice. For any good academician, "Publish or Perish" has to be motto and then one accordingly evolves oneself from case reports to review. It really takes an entire lifetime to pen down a book and that too when it deals about a vibrant and vividly important subject such as anesthesia. Therefore, it gives me immense pleasure to write a foreword for four of my brilliant anesthesiologists who gave me an opportunity to hold a scalpel while putting tiny tots to sleep in my lifetime. I am personally blessed to have been associated with all the four principal contributors of this book and I know how difficult it is to convince clinicians to write.

An emergency patient in the hospital presents multitude of problems. Surgical problems are overt but occult problems are the one that are to be addressed and answered by the anesthesiologists; at times, even diagnosis cannot be ascertained and the need to take instantaneous decision is pressing. Time at disposal is extremely short and every second counts. Such extraordinary, extremely demanding situation arises more than once in the life of an anesthesiologist and, therefore, it is of paramount significance that he/she is prepared for anytime, all the time and every time. He/she needs a scientific knowledge, also expected to use skill and at times with limited armament of monitoring devices. He/she is expected to rely on his/her clinical acumen. Hospital administration would spend for fancy gadgets and scopes and perhaps be little stingy when it comes to providing monitors. This attitude should be taken as a challenge by anesthesiologists to demonstrate their expertise.

I can certainly tell that each and every contributor in this title has a wealth of experience of managing patients from day one of life to even centurions and I am sure that this book will become relevant and a matter of ready reference for many of the students as well as practicing anesthesiologists. I only take pride in the fact that somewhere down in their sojourn I have been by-stander as a surgeon and have immensely benefited by their expertise. I wish the readers all the best.

Sanjay Oak
Director, Medical Education and Major Hospitals
Municipal Corporation of Greater Mumbai
Seth GS Medical College and KEM Hospital
Mumbai, Maharashtra, India

Preface

As faculty of Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai (LTMMC and LTMGH), we had ample opportunities to work on anesthesia for emergency surgeries. There is a large influx of trauma, general surgical, pediatric, neurosurgical and obstetric emergencies as the hospital is one of the major public hospitals located in a densely populated region sitting amidst major routes in the city. It also provides services for emergency cardiovascular, cerebrovascular interventions and cadaver transplants. Daily lessons from this tremendous emergency work inspired us to bring out this book.

There is plethora of reference material available in basic sciences related to anesthesiology, namely physiology, pharmacology, physics and anatomy. Similarly, a large number of textbooks are on techniques such as general, regional, monitored anesthesia care as well as super specialties like cardiac, neurosurgical and pediatric anesthesia, etc. But there is a limited availability of books and references for emergency anesthesia. One has to scan and integrate parts from various sources. This book is an attempt to assimilate the scattered information and add clinical expertise.

Contents of this book have been divided into nine sections to cover majority of the emergencies. General considerations for adults, obstetric and pediatric sections have been written separately to avoid duplication. An additional challenge is cases with various medical disorders for emergency surgeries. Efforts have been taken to accommodate them in first section and obstetric section.

The contributors of this book work together at LTMMC and LTMGH and have a well-balanced team with fresh talent bringing the latest technological knowledge and tenured practitioners supplementing it with applied experience. Part of the team worked on collection of the matter from different sources for an ideal scenario, while the senior faculty members used their vast clinical experience for a more practical and realistic application. These combined efforts have been truly beneficial to achieve our goal to rationally mix latest technology with clinical experience and identify potential ways to conduct anesthesia with best results, at least costs and minimum risk. We thank all our contributors for round-the-clock effort and still being alert for the next emergency.

We sincerely thank all our teachers, colleagues, Dr Sandhya Kamath, Dean, LTMMC and LTMGH and Dr Sanjay Oak, Dean, GSMC, KEMH and Director (Medical Education and Major Hospital), Mumbai, Maharashtra, India, for providing all training and infrastructure and thereby enriching us with this clinical experience. We are grateful to M/s Jaypee Brothers Medical Publishers for their prompt response for our first endeavor.

Manju N Gandhi
Anila D Malde
Amala G Kudalkar
Hemangi S Karnik

Contents

Section I: General Considerations

Section II: Cardiovascular Emergency Procedures

Section III: General Surgery

Section VI: ENT and Ophthalmic Emergencies

Section VII: Neurosurgical Emergencies

Section VIII: Trauma

Section I

General Considerations

1 General Principles for Emergency Anesthesia

Manju N Gandhi, Kanchan R Rupwate, Kamlesh G Gotiwale, Manish B Kotwani

KEY POINTS

- Anesthesia for emergency situations is unplanned, hence the time available to evaluate the patient preoperatively and prepare for surgery is short. The patient's condition may deteriorate rapidly, therefore both decision making and performance have to be achieved within short period in order to save life.
- The use of structured 'core' algorithm (based on mnemonic COVER ABCD A SWIFT CHECK) would diagnose and correct the problem in 60 percent of cases and provide a functional diagnosis in remaining 40 percent of cases.
- An altered level of consciousness may indicate the need for immediate evaluation of patient's oxygenation, ventilation and perfusion status.
- A quick look at patient's history, records, ease of intubation, investigations like hematocrit, serum creatinine, electrolytes, blood glucose, coagulation status and arterial blood gas analysis may give rough estimation of patient's condition.
- Prediction of difficult airway should be done quickly and difficult airway cart should be kept ready. Whenever possible expert assistance should be called to help. Patients with maxillofacial trauma, cervical spine fractures, burns, obesity and obstetric patient may have difficulty in ventilation and intubation.
- Certain bed side investigations can ascertain lot of required information like hemoglobin, blood glucose, urine ketones, ABG with serum electrolyte, ECG and portable X-ray.
- Adequate blood and blood products should be arranged as per nature of surgery. Whenever there is anticipation that large quantity of blood and blood products are going to be required, the blood bank officer should be alerted as soon as possible.
- Preoperative optimization is a vital component of safe conduct of anesthesia. The risk of operation can often be lowered by preoperative treatment of physiological derangements like hypoxia, hypoventilation, hypovolemia, dehydration, acidosis/alkalosis, electrolyte abnormalities and control of blood glucose levels, etc.
- Patients with co-existing medical diseases should be evaluated quickly and optimized as fast as possible. History may not be available regarding associated medication and/or medical disorders.
- The appropriate administration of intravenous fluid therapy to maintain an effective circulating volume and prevent inadequate tissue perfusion is a core element of the practice of anesthesia.
- H_2-blocker or proton pump inhibitor used to reduce gastric acidity and volume can reduce the morbidity of aspiration. Acid aspiration prophylaxis should also be given to the patient receiving regional or local anesthesia.
- Most of the patients coming for emergency surgery are either not starved or considered full stomach because of various pathologies or medical disorders. Proper aspiration prophylaxis should be given and rapid sequence intubation (RSI) should be performed while giving general anesthesia (GA). Endotracheal intubation is a gold standard for patient with full stomach undergoing surgery with GA.
- Changes in electrolyte disorders should be corrected and maintained within normal range in order to prevent life threatening arrhythmias, delayed recovery from anesthesia, disorientation, confusion, muscle weakness and inadequate ventilation, etc. Abnormalities in sodium, potassium, calcium, magnesium, and pH must be corrected simultaneously.

- Preoperative optimization with fluid therapy reduces the incidence of postoperative nausea and vomiting and acute deterioration of renal function especially in patients with hypotension, hypovolemia, dehydration and septicemia.
- All the emergency drugs, equipments, defibrillator, and monitors should be checked before starting of anesthesia.
- Choice of anesthesia technique depends on patient's condition, and type of surgery. Patient can be given any type of anesthesia from GA to peripheral nerve blocks, central neuraxial block or local infiltration.
- Anesthetic agents, sedatives and hypnotic drugs should be used judiciously with careful titration. Hemodynamically unstable patient may have increased sensitivity to these agents.
- Before performing peripheral nerve block or central neuraxial block patient's coagulation status should be known or whenever in doubt it is wiser to give GA.
- Patient's temperature should be maintained by using warming devices.
- All patients should be monitored for vital parameters, ECG, urine output, saturation of hemoglobin by pulse oximeter and end tidal carbon dioxide.
- Central venous pressure monitoring is needed in major surgery and patients with heart disease, renal disease, septicemic patient or shock, etc.
- Precautions should be taken to avoid aspiration at the time of extubation also.
- Patient who had difficulty in performing endotracheal intubation, the extubation should be delayed till the patient is fully awake. The extubation can be also performed using tube exchanger in case of anticipated difficulty in ventilation or maintenance of airway following extubation, the tube exchanger can be utilized for rapid and successful re-intubation.
- Postoperative analgesia is either planned preoperatively or decided at the end of procedure as per patient's hemodynamic stability.
- All patients should be observed in post-anesthesia care unit for few hours and then either transferred to ward, high dependency unit or intensive care unit for further management.

INTRODUCTION

Anesthesia for emergency situation can be termed as unplanned as the patients are not evaluated preoperatively and prepared for the surgery. This situations can occur anywhere inside the operation theater or outside the comfort of operation theater including ICU, high dependency units, coronary care units, fields and war situations.

DEFINITION OF EMERGENCY SURGERY

Immediate operation is needed usually within one hour of surgical consultation to save life. Resuscitation of patient is performed simultaneously with surgical management. Emergency cases require a great deal of logistical coordination between surgeons, anesthesiologists and various other disciplines of medicine. Emergency operations take precedence over all other cases and anesthesia is sometime administered in desperate circumstances, e.g. major thoracic, abdominal trauma, cardiac tamponade, major vessel trauma, obstetric emergencies.

The time available to accomplish this task is short and patient's condition may deteriorate rapidly. Therefore both decision-making and performance may be compromised in such circumstances.

In certain situations, the patient may need to be transferred to tertiary care centre, then one must ensure that optimal care is provided during evaluation, resuscitation and transfer process.[1]

DEFINITION OF URGENT SURGERY

The operation needs to be performed as soon as possible. The surgery can be differed for few hours to up to 24 hours, thereby one gets time for resuscitation and hemodynamic optimization of the patient, e.g. facial fractures without active nasal/oral bleeding, closed long bone fractures, etc.

ENVIRONMENT CONSIDERATION AND PREPAREDNESS

Emergency anesthesia is given in several environments. In prehospital emergency situations, either paramedics or emergency physicians are responsible for airway instrumentation and sedation/anesthesia, depending on the country and organization.

All anesthesiologists have to handle life-threatening crisis without any warning. However, some cognitive strategies and work practices that are appropriate for speed and efficiency under normal circumstances may become maladaptive in a crisis situation. It could be due to stress during high-risk patient management and suboptimal facilities available at that point of time.

The use of structured 'core' algorithm (based on mnemonic COVER ABCD A SWIFT CHECK) would diagnose and correct the problem in 60 percent of cases and provide a functional diagnosis in remaining 40 percent of cases.[2]

PREOPERATIVE ASSESSMENT

Once the decision has been made to proceed with operative management, a number of considerations must be addressed regarding the timing and site of surgery, the type of anesthesia, and the preoperative preparation necessary to optimize the patient. Direct communication with the consultant and with the patient's primary care provider, whenever possible, may provide meaningful clinical information.

By definition, emergency induction is needed when acuity of the patient's presentation does not allow anesthesiologist the normal preoperative assessment and optimization. The emergency patient is at higher risk for anesthesia and surgery in almost all surgical circumstances.

At bare minimum, one needs to have answers to the following questions:

- Why patient needs emergency surgery?
- How much time is available for resuscitation?
- Are there are signs indicating presence of hemorrhage?
- What are the results of quick airway assessment for difficult intubation?
- Whether patient will need ventilatory support postoperatively, ICU care, etc.?

A quick look at patient's history, records, ease of intubation, investigations like hematocrit, serum creatinine, blood glucose, coagulation status, arterial blood gas analysis and electrolytes may give rough estimation of patient's condition. Predicting anatomically difficult airway in a patient with critical condition is more difficult than in patient having an elective surgical operation. In addition the position of patient, facial and neck injuries, full stomach, saliva, blood and debris in the upper airway may worsen the intubating conditions and sometimes even makes it impossible to intubate the patient.

The anesthesiologist decides when the patient has recovered enough to be sent to a regular room, High dependency unit (HDU) or transferred to an intensive care unit (ICU). Mandating an ICU stay in advance makes no sense unless the operation itself demands ICU care.[3] In case of hemodynamically unstable patient, cardiac surgery, neurosurgery or septicemic patient, etc. who may need ICU care, the concerned department should be informed preoperatively for necessary arrangement.

EMERGENCY INVESTIGATIONS

Certain bedside investigations that can ascertain lot of required information like hemoglobin, blood glucose with glucometer/or glucose strips, urine ketones with ketostix, arterial blood gases with serum electrolytes, electrocardiogram (ECG) and portable X-ray' can be availed within short period of time.

Blood should be send for grouping and cross-matching as and when required. If requirement of more number of blood and blood products is anticipated, the blood bank should be alerted as soon as possible.

In addition, portable ultrasonography can be used for surgical diagnosis, portable 2-dimensional echocardiography for cardiovascular evaluation and thromboelastography (TEG) for determination of bleeding disorders.

Whenever the surgery is urgent or semiurgent, detailed investigations can be performed within few hours.

PREANESTHETIC PREPARATION

For many patients optimal perioperative care may require little or no additional medical management beyond that given by the anesthesiologist and surgeon. However, there is a continued existence of a group of surgical patients who are at a high-risk of morbidity and mortality which indicate an ongoing need to identify such patients and deliver optimal care throughout the perioperative period.

A group of patients may exist in whom the risk for death and serious complications after major surgery is in excess of 20 percent. The risk is related mainly to the patient's preoperative physiological condition and, in particular, the cardiovascular and respiratory reserves.[4]

The medical management of all co-existing disease processes should then be reviewed to ensure that current standards of best practice are adhered to. Various aspects of perioperative management should then be given consideration. It is recommended that all such patients be admitted to a critical care area ideally before surgery. There is evidence to suggest that this approach results in an overall reduction in consumption of resources.[5,6]

Certain special group of patients should be cared as per their co-existing diseases or physiologic alterations due to various reasons. Some of them are like neonates, pediatric patients, geriatric patients, obstetric patients, neurosurgical patients, cardiac surgery patients, motor

vehicular trauma victims and patient coming for cadaveric organ transplantation, etc. may need specialized care.

Preoperative Optimization and Management of Complications

Preoperative optimization is a vital component of safe conduct of anesthesia. The risk of operation can often be lowered by treatment of physiological derangements like hypoxia, hypoventilation, hypovolaemia, dehydration, acidosis/alkalosis, electrolyte abnormalities and control of blood glucose levels, etc.

Most of the patients who need emergency anesthesia outside the operation theatre have serious disturbances in respiratory and/or hemodynamic function. Anesthetic agents may worsen these disturbances.

Thus the following underlying conditions should be treated simultaneously with induction of anesthesia and intubation.

- Predict the problems like hypoxia and correct it with preoxygenation
- Manage hypotension with fluids and vasoactive drugs
- Aspiration prophylaxis for vomiting and regurgitation should be managed with rapid sequence intubation using cricoids pressure and using suction device whenever patient vomits
- Correct acidosis and electrolyte abnormalities
- Maintain effective circulatory blood volume to prevent inadequate tissue perfusion
- Cardiac arrhythmias are to be managed with drugs and defibrillator.

The appropriate administration of intravenous fluid therapy to maintain an effective circulating volume and prevent inadequate tissue perfusion is a core element of the perioperative practice of anesthesia. The choice of fluids in a variety of different clinical situations can now be rationally guided by an understanding of the physiochemical and biological properties of the crystalloid and colloid solutions available.

There is no difference in outcome between crystalloid or colloid administration when the amount infused is 3 to 4 liters but when volume of crystalloid infused reaches more than patient's blood volume there may be cellular edema. Therefore, it is wise to use balanced combination of crystalloid and colloid as per patient's requirement. However, one must be careful while giving colloids, especially when it is infused while waiting for blood to arrive. If the blood is transfuse as soon as it arrives and colloids still circulating in intravascular compartment at that point of time, it may lead to pulmonary congestion especially in geriatric patients.

Goal-directed "optimization" of intravascular volume and organ blood flow aims to ensure adequate tissue perfusion and cellular oxygenation. There is a substantial body of literature demonstrating that mortality following major surgery can be significantly reduced by goal-directed approaches to perioperative hemodynamic management.

Perioperatively patients may be 'water and solute depleted' owing to:

- Decreased intake (preoperative fasting, anorexia, altered consciousness level)
- Increased losses (vomiting, diarrhea, pyrexia, bleeding)
- In addition intraoperatively, both anesthesia and the surgical procedure may upset the fluid balance
- Intravenous and inhalational anesthetic agents commonly produce relative hypovolemia secondary to vasodilatation, and act as myocardial depressants, reducing cardiac output
- Surgical losses include direct hemorrhage, evaporative losses from exposed mucosal surfaces and third-space losses.

The traditional approach of an "X" ml/kg continuous fluid infusion with additional replacement of observed losses is clearly flawed in major surgery. It takes no account of preoperative fluid status.

Fluid therapy should be titrated to rational physiological end-points:

- Pulse, blood pressure
- Skin turgor, absence of thirst, etc.
- Monitoring of central venous pressure and whenever possible pulmonary capillary wedge pressure can be used to guide fluid therapy in critically ill patient.

One must not forget the ultimate goal of fluid therapy is maintenance of tissue perfusion and oxygenation.

In the study of fluid therapy for ambulatory surgery, it is suggested that preoperative or intraoperative crystalloid infusion of 20 ml/kg improves clinical outcome. In particular, postoperative nausea and vomiting is reduced following intraoperative crystalloid infusion, when compared with controls.[7]

Recently distinct sets of goals have been employed for fluid optimization:

- Intravascular pressure measurements (arterial pressure, central venous pressure, and pulmonary artery occlusion pressure)

- Indices of global blood flow (cardiac output or index; stroke volume or index, oxygen delivery, and mixed venous oxygen saturation)[8]
- Indices of tissue perfusion (gastrointestinal tonometry, tissue oxygen electrodes) [7,11]
- Dynamic ultrasonographic evaluation of the heart and great vessels in prediction of the fluid responsiveness of critical patient has been evaluated[9]
- Recent trial of patients recruited in the emergency room with early severe sepsis demonstrated a significant reduction in mortality when SvO_2 (from a CVP line) was used to direct therapy[10]
- Doppler flowmetry for splanchnic perfusion[12] and microdialysis catheters[13] have been use for guiding the fluid therapy
- Near infrared spectrometry[14] and tissue pH monitors.

There are now a number of well conducted studies that show that the use of perioperative goal directed therapy may improve outcome.[15-18]

Electrolyte Emergencies, Anion Gap and Osmolality

In the emergency department and acute surgical emergencies anesthesiologist have to deal with various fluid-electrolyte disturbances. Since fluid balance, electrolyte and acid-base derangements share common pathophysiological process, a full understanding of the ongoing interplay of homeostatic derangement and choosing the correct approach to a therapeutic strategy is necessary for the management of the critically ill patient.[19]

Hyperosmolality is present when plasma osmolality exceeds 295 mOsm/kg and that leads to water shift from ICF to ECF leading to thirst stimulation and ADH release, e.g. hyperosmolar diabetic ketoacidosis. In this case the patient will also tend to have metabolic acidosis, hyponatremia and hyperkalemias. Again because of metabolic acidosis patient will be in respiratory alkalosis. Here the main aim is to correct the hyperglycemia with insulin and dehydration with normal saline.

Hypoosmolality condition can be seen due to overloading or overhydration with crystalloids, hypotonic solution, and in patients with hypoproteinemia, heart failure and liver cirrhosis, etc. One of the major drawback of infusing 5 percent dextrose as replacement solution for major shift in fluid is that once glucose is utilized by the body then what will remain in intravascular compartment is water, which is hypotonic and may lead to tissue or pulmonary edema.

The target organ in any dysosmolal state is brain. As a general rule the faster the onset of disorder, the poorer the prognosis—owing to lack of time to compensate for the disorder at the metabolic and neuronal level. However, it may be possible to tolerate these disturbances provided enough time is elapsed to tolerate the 'water stress' that is hyponatremia.

Treatment of Various Electrolyte Abnormalities

Treatment of hyponatremia: Hyponatremia is usually asymptomatic unless serum sodium level falls below 120 mEq/L. The hyponatremia is commonly seen in patients with severe vomiting diarrhea, geriatric patient, TURP syndrome, burns, diabetic ketoacidosis and cerebral salt wasting syndrome.

Treatment of hyponatremia involves the administration of sodium and elimination of free water. Correction of hyponatremia should be gradual, usually an increase of 0.5 mEq/L/hr and to maximum of 10 to 15 mEq/L in first 24 hr. Rapid correction of serum sodium can cause pontine myelinosis, a lethal disorder caused by rapid shift of fluid in the brain. In hyponatremic states a loop diuretic can be given with hypertonic saline infusion, to enhance free water clearance. If serum sodium is above 120 to 124 mEq/L, it can be corrected slowly with 0.9 percent solution and loop diuretic furosemide injection. Whenever serum sodium is below 118 mEq/L then patient should be treated with 3 percent sodium chloride (513 Na^+ mEq/L) till serum sodium concentration is above 120 mEq/L or neurological symptoms improves. Check serum electrolyte frequently.

Ultimate correction of serum sodium requires calculation of the sodium deficit. The following formula can be used:

Sodium deficit = (Desired Sodium – Current Sodium) × 0.6 × Body Wt
(Use 0.6 for men and 0.5 for women)

Treatment of hypernatremia: Hypernatremia is defined as a serum sodium concentration above 145 mEq/L. Hypernatremia may be caused by a primary Na^+ gain or excess water loss. A common cause of hypernatremia is free water loss in excess of sodium loss, such as that which occurs with diabetes insipidus or hypernatremic dehydration. In the brain, decreased nerve cell volume can cause neurological symptoms, including altered mental status, weakness, irritability, focal neurological deficits, and even coma or seizures.

To treat hypernatremia, it is important to stop ongoing water losses (by treating the underlying cause) while correcting the water deficit. In hypovolemic patients the extracellular fluid (ECF) volume must be restored with normal saline.

The quantity of water in liters required to correct hypernatremia can be calculated by the following equation:

$$\frac{\text{Plasma sodium} - 140}{140} \times \text{Total body water}$$

Once the free water deficit is calculated, administer fluid to lower serum sodium at the rate of 0.5 to 1.0 mEq/L/hr with a decrease of no more than 12 mEq in the first 24 hours. Total correction should be achieved over 48 to 72 hours. The method of replacement of free water depends on the patient's clinical status. For stable, asymptomatic patients, replacement of fluid by mouth or through a nasogastric tube is effective and safe. If this is not possible or if the patient's clinical status demands more aggressive treatment, 5 percent dextrose in half-normal saline may be given IV. Check the patient's serum sodium and neurological function frequently to avoid overtly rapid correction.

Treatment of hypokalemia: Hypokalemia is defined as a serum potassium level <3.5 mEq/L. Hypokalemia results from one or more of the following reasons: decreased dietary intake, shift into cells, or increased net loss from the body. The most common causes of low serum potassium include gastrointestinal loss (diarrhea, laxatives), renal loss (hyperaldosteronism, potassium-losing diuretics, intracellular shift (alkalosis or a rise in pH) and malnutrition.

Symptoms of hypokalemia include weakness, fatigue, paralysis, respiratory difficulty, muscle pain, constipation, paralytic ileus, and leg cramps. Hypokalemia also exacerbates digitalis toxicity.

ECG changes suggestive of hypokalemia includes:

- U waves
- T-wave flattening
- ST-segment changes
- Arrhythmias (especially if the patient is taking digoxin)
- Pulseless electrical activity (PEA) or asystole.

The treatment of hypokalemia includes minimizing further potassium loss and giving potassium replacement. Intravenous administration of potassium is indicated when arrhythmias are present or hypokalemia is severe (K^+ <2.5 mEq/L). Rate of infusion of potassium chloride should be guided by ECG and serum electrolyte determination. Before starting the intravenous infusion of potassium one must ensure that the urine output is adequate.

The potassium deficit is calculated according to the following formula:

mEq potassium = kg body weight × 0.2 × 2 × (4.5 – current serum potassium)
(15 percent solution of potassium chloride: 1 ml = 2 mEq of potassium)

Concentrated potassium solution is for IV admixtures only; do not use undiluted. Direct injection may be instantaneously fatal.

Suitable vehicle solutions, e.g. 5 or 10 percent glucose solutions, isotonic sodium chloride solution, compound sodium lactate solution.

- The potassium concentration in the infusion solution must not exceed 40 mEq/L
- Do not infuse rapidly. Maximum infusion rate: Up to 40 mEq potassium per hour (corresponding to 0.3 mEq potassium/kg body weight/hour) in adults when the serum potassium is below 2 mEq/L
- Maximum daily dose should not exceed more than 2-3 mEq/kg body weight/day
- Children: IV infusion up to 3 mEq/kg/day. Adjust volume of administered fluids to body size
- In critical states, potassium chloride may be administered in saline (unless saline is contra-indicated), since dextrose may lower serum potassium levels by producing an intracellular shift along with glucose
- As a matter of principle, infusion pumps should be used for the infusion of potassium.

If cardiac arrest from hypokalemia is imminent (i.e. malignant ventricular arrhythmias), rapid replacement of potassium is required. Give an initial infusion of 2 mEq/min, followed by another 10 mEq intravenously after 10 minutes. Once the patient is stabilized, reduce the infusion to continue potassium replacement more gradually.

Treatment of hyperkalemia: Hyperkalemia (potassium >5.5 mEq/L) is common finding in patient with acute or chronic renal failure, oral/parenteral potassium supplementation and patients on angiotensin converting enzyme inhibitors (ACEI) or potassium sparing diuretic (Spironolactone). The hyperkalemia may also develop following succinylcholine in patients with severe burns, lower motor neuron paralysis or spinal cord trauma with paraplegia or quadriplegia. This is also observed in cases of crush injury, compartment syndrome, rhabdomyolysis or following tourniquet release, etc.

Changes in pH inversely affect serum potassium. Acidosis (low pH) leads to an extracellular shift of potassium, thus raising serum potassium. Conversely, high pH (alkalosis) shifts potassium back into the cell, lowering serum potassium.

Physical symptoms of hyperkalemia include, weakness, ascending paralysis, and respiratory failure. ECG changes suggestive of hyperkalemia include:

- Peaked T waves (tenting)
- Flattened P waves
- Prolonged PR interval (first-degree heart block)
- Widened QRS complex
- Deepened S waves and merging of S and T waves
- Idioventricular rhythm
- Sine-wave formation
- VF and cardiac arrest.

Emergency treatment of hyperkalemia may include any of the following measures:

- Discontinue medications that increase blood potassium levels
- Intravenous administration of glucose and insulin, which promotes movement of potassium from the extracellular space back into the cells. Mix 50 gm glucose and 10 U regular insulin and give IV over 15 to 30 minutes
- Intravenous calcium to temporarily protect the heart and muscles from the effects of hyperkalemia. (Calcium Chloride/Gluconate 10 cc of 10 percent over 2 to 5 min)
- Sodium bicarbonate administration to counteract acidosis and to promote movement of potassium from the extracellular space back into the cells (50 to 100 mEq over 5 min)
- Diuretic administration to decrease the total potassium stores through increasing potassium excretion in the urine, e.g. furosemide 1 mg/kg IV slowly
- Medications that stimulate beta-2 adrenergic receptors, such as albuterol and epinephrine have also been used to drive potassium back into cells
- Nebulized albuterol 10 to 20 mg nebulized over 15 minutes.
- Medications known as cation-exchange resins, which bind potassium and lead to its excretion via the gastrointestinal tract. Resins—Kayexalate 15 to 30 g in 50 to 100 ml of 20 percent sorbitol either orally or by retention enema (50 g of Kayexalate)
- Dialysis, particularly if other measures have failed or if renal failure is present.

Treatment of hyperkalemia naturally also includes treatment of any underlying causes (e.g. kidney disease, adrenal disease and tissue destruction) of hyperkalemia.

Treatment of hypomagnesemia: Hypomagnesemia is far more common clinically than hypermagnesemia. Defined as a serum magnesium concentration below the normal range of 1.3 to 2.2 mEq/L, hypomagnesemia usually results from decreased absorption or increased loss, either from the kidneys or intestines (diarrhea). Alterations in parathyroid hormone and certain medications (e.g. diuretics, alcohol) can also induce hypomagnesemia.

Symptoms of low serum magnesium include muscular tremors and fasciculations, ocular nystagmus, tetany, and altered mentation. Other possible symptoms include ataxia, vertigo, seizures, and dysphagia. A number of ECG abnormalities occur with low magnesium levels, including:

- Prolonged QT and PR intervals
- ST-segment depression
- T-wave inversion
- Flattening or inversion of precordial P waves.

Widening of QRS

- Torsades de pointes ventricular arrhythmias

Resistant VF (and other arrhythmias)

- Worsening of digitalis toxicity
- Treatment of hypomagnesemia depends on its severity and the patient's clinical status
- For severe or symptomatic hypomagnesemia, administer 1 to 2 g IV $MgSO_4$ over 15 minutes
- If torsades de pointes ventricular arrhythmias are present, administer 2 g of $MgSO_4$ over 1 to 2 minutes
- If seizures are present, administer 2 g IV $MgSO_4$ over 10 minutes
- Risks involved with intravenous magnesium therapy include hypermagnesemia, hypocalcemia, and sudden hypotension.

Treatment of hypermagnesemia: Magnesium balance is influenced by many of the same regulatory systems that control calcium balance. In addition, magnesium balance is influenced by diseases and factors that control serum potassium. As a result, magnesium balance is closely tied to both calcium and potassium balance.

The most common cause of hypermagnesemia is renal failure. Hypermagnesemia may also be iatrogenic (caused by overuse of magnesium) or caused by a perforated viscus with continued intake of food and use of laxatives/antacids containing magnesium.

The signs and symptoms are neurological symptoms like lethargy, muscular weakness, paralysis, ataxia, drowsiness, and confusion. Gastrointestinal symptoms include nausea and vomiting. Moderate hypermagnesemia can produce vasodilation, and severe hypermagnesaemia can produce hypotension. Extremely high serum magnesium levels may produce a

depressed level of consciousness, bradycardia, hypoventilation, and cardiorespiratory arrest.

ECG changes of hypermagnesemia include:

- Increased PR and QT intervals
- Increased QRS duration
- Variable decrease in P wave voltage
- Variable degree of T wave peaking
- Complete AV block, asystole.

Hypermagnesemia is treated by antagonizing magnesium with calcium gluconate/chloride, removing magnesium from serum, and eliminating sources of ongoing magnesium intake. Cardiorespiratory support may be needed until magnesium levels are reduced. Administration of calcium chloride (10 cc of 10 percent) intravenously will often correct lethal arrhythmias.

Dialysis is the treatment of choice for treatment of severe hypermagnesemia.

Treatment of hypocalcemia: Hypocalcemia is defined as a serum calcium concentration below the normal range of 8.5 to 10.5 mg/dl (or an ionized calcium below the range of 4.2 to 4.8 mg/dl). Hypocalcemia may develop with toxic shock syndrome, abnormalities in serum magnesium and tumor lysis syndrome. Symptoms of hypocalcemia usually occur when ionized levels fall below 2.5 mg/dl. Symptoms include paresthesias of the extremities and face, followed by muscle cramps, carpopedal spasm, stridor, tetany, and seizures. Hypocalcemic patients demonstrate hyperreflexia and positive Chvostek and Trousseau signs. Cardiac symptoms include decreased contractility and heart failure. Hypocalcemia can exacerbate digitalis toxicity.

ECG changes of hypocalcaemia include following:

- QT-interval prolongation
- Terminal T wave inversion
- Heart blocks
- Ventricular fibrillation.

Treatment of hypocalcemia requires administration of calcium. One ampoule of calcium chloride is equal to three ampoules of calcium gluconate. Treat acute symptomatic hypocalcemia with 10 to 20 cc of 10 percent calcium gluconate (90 to 180 mg of elemental calcium) IV over 10 minutes. Follow this with an IV drip of 540 to 720 mg of elemental calcium in 500 to 1000 ml D_5W 0.5 to 2.0 mg/kg per hour (10-15 mg/kg). Measure serum calcium every 4 to 6 hours and aim to maintain the total serum calcium concentration between 7 and 9 mg/dl.

Treatment of hypercalcemia: Hypercalcemia is defined as a serum calcium concentration above the normal range of 8.5 to 10.5 mg/dl (or an elevation in ionized calcium above 4.2–4.8 mg/dl). Symptoms of hypercalcemia usually develop when the total serum calcium concentration reaches or exceeds 12 to 15 mg/dl. Neurological symptoms include depression, weakness, fatigue, and confusion at lower levels. At higher levels patients may exhibit hallucinations, disorientation, hypotonicity, and coma. Hypercalcemia interferes with renal concentration of urine, causing dehydration.

Cardiovascular symptoms of elevated calcium levels are variable. Myocardial contractility may initially increase until the calcium level reaches 15 to 20 mg/dl. Above this level myocardial depression occurs. Automaticity is decreased and ventricular systole is shortened. Arrhythmias occur because the refractory period is shortened. Digitalis toxicity is worsened. Hypertension is common. In addition, many patients with hypercalcemia develop hypokalemia; both these conditions contribute to cardiac arrhythmias. Hemodialysis is the treatment of choice to rapidly decrease serum calcium in patients with heart failure or renal insufficiency. Chelating agents may be used for extreme conditions (e.g. 50 mmol PO_4 over 8–12 hours or EDTA 10–50 mg/kg over 4 hours).

Abnormalities in sodium, potassium, calcium, magnesium and pH must be corrected simultaneously.[20]

Preemptive Managment to Prevent Postoperative Acute Deterioration in Renal Function

Postoperative acute deterioration in renal function producing oliguria and/or increase in serum creatinine is one of the most serious complications in surgical patients. Most cases are due to renal hypoperfusion as a consequence of systemic hypotension, hypovolemia, and cardiac dysfunction. Hence all efforts should be made to identify patients and surgery that would most benefit from perioperative optimization with fluid therapy.[21]

Postoperative gastrointestinal function is influenced by preoperative fluid optimization, e.g. better splanchnic perfusion and lower gut edema with tetrastarches decrease the incidence of PONV.[22,23]

Selection of IV Fluid

Numerous fluid preparations are available for the replacement of perioperative fluid losses in patients undergoing surgery. The selection of particular fluid is influenced by multiple factors like tradition, systemic effects, postoperative outcome and cost.

Impact of IV fluids on coagulation: Whenever small volume is administered (1000 ml), there is no difference in outcome. The administration of large volume of any type of fluid will cause dilution of platelets and coagulation factors which may lead to coagulopathy. In

addition, fluids can have direct impact on blood clotting through effect on circulating components of coagulation cascade or altering platelet function. Several studies demonstrate hydroxyethyl starches 130/0.4 M and gelatins have fewer adverse effects on coagulation in comparison with higher molecular weight starches.

Replacement of Blood and Blood Components

Whenever patient is actively bleeding or has lost more than 30 to 40 percent blood volume and there is anticipation of major blood loss due to nature of surgical procedure, the transfusion of blood should be started as soon as possible. Blood components should be used rationally whenever indicated. The details of this will be dealt in another chapter of this book. Blood and blood products should be arranged as per the need and urgency before induction of anesthesia.

Once, the patient's co-morbidities are optimally managed, whether or not to proceed with surgery is a question for the anesthesiologist, surgeon and patient to decide after weighing the risks and benefits. No specific anesthetic is inherently safer than any other.

Coexisting Medical Diseases

Several medical diseases like uncontrolled hypertension, diabetes mellitus, asthma, congestive cardiac failure or other cardiac problems could complicate the course of surgery and anesthesia. Standard care requires that the inter–current medical disease is optimized prior to induction of anesthesia. This is often not possible in emergent situations. It is necessary that attempts should be made at optimizing the medical condition as much as time would allow.

Complicating diseases and conditions may strongly influence the outcome of procedure undertaken. In patients with coronary artery disease, congestive cardiac failure, respiratory failure, pulmonary emphysema, asthma, liver disease, renal disease and diabetes mellitus different methods of therapy and anesthesia must be employed, so that minimal delay without compromising the safety of patient is employed.[24]

Following Equipment and Drugs should be Readily Available

- **Emergency intubation cart:** Various sizes of laryngoscopic blades, endotracheal tubes, stylet, different types of laryngoscope, gum-elastic bougies, LMA/ILMA, fiberoptic bronchoscope, percutaneous cricothyroidotomy set or tracheostomy set, etc.
- **A table that can be rapidly placed in head down (Trendelenberg) position:** In case of vomiting or regurgitation the patient should be quickly rolled onto one side and the head of the table tipped down, using gravity to facilitate exit of vomitus from the airway and pharynx. **Caution:** patients who have received spinal anesthesia should never be placed head down, even if shocked. The reason is that further shock and respiratory failure may rapidly ensue if the spinal anesthetic agent is allowed to bathe the upper spinal cord during a head-down maneuver. For other patients, there is some debate whether the Trendelenberg position actually helps anyone who is hypotensive.
- **Have suction instantly ready, check equipment before starting**
- **Have resuscitation drugs (inotropes, vasopressors and vasodilators) tray ready with plenty of intravenous fluids**
- **Patient warming system**
- **Monitoring equipment like ECG, pulse oximeter, capnometer, NIBP/IBP, temperature, bispectral index monitor, etc.**
- **In special scenario cardiac output monitor, transesophageal echocardiography** can be used.
- **CVP/ PCWP** for guiding fluid therapy may be used.
- **Urine output should be monitored hourly.**

Choice of Anesthetic Technique

The anesthetic choice for emergency procedures is guided by the nature of surgical technique and the preferences of the anesthesiologist. They can be:

- General anesthesia
- Local anesthesia
- Central regional anesthesia
  - Peripheral nerve blocks
- Total intravenous analgesia (Bier's block)

Factors favoring the use of general anesthesia:

- Hemodynamic instability
- Uncertainty of diagnosis and duration of surgery
- Lack of time
- Patient's distress or confusion
- Strangulated or obstructed inguinal hernia
- Septicemic patient
- Fetal distress, cord prolapsed, uterine rupture, for LSCS.

In some cases either general or regional anesthesia may be administered:

- Amputation
- Debridement of wound
- Drainage of abscesses
- Fracture of long bones
- Patients who received general anesthesia for initial surgery due to hemodynamic instability may

become fit to receive regional anesthesia for subsequent surgery within few days, e.g. wound washing, redo surgery, etc.

Is there a best approach to induction of anesthesia in emergent situation?

Anesthetic agent/or sedative agents should be used judiciously with careful titration. Patients in shock have increased sensitivity to these agents. Sympathetic drive is at its maximum in patient with shock or hemodynamically compromised patient, therefore these patients tend to decompensate at the time of induction. Direct depressant effect of sedative and hypnotic agents may lead to vasodilatation and in addition positive pressure ventilation may reduce cardiac filling, as a result severe hypotension and sometimes cardiac arrest may occur since protection due to sympathetic drive is lost.

The risk of laryngospasm, hemodynamic instability and aspiration at the time of induction should be kept in mind.

The choice of induction agent is less important than the dose selected for particular patient in emergency. Initially full induction dose should not be given in order to prevent severe hypotension. The drug should be given in small aliquots, once the patient tolerates the first small dose well, the additional doses can be supplemented slowly by keeping watch on patient's hemodynamics parameters. Choice of neuromuscular blockade depends on clinical situation.

Care during Intubation

Intubation response should be attenuated in traumatic brain injury (TBI) or patients with intracerebral bleed and malignant hypertensive patient, etc. The intubation may be difficult, in patients with maxillofacial trauma and cervical spine injury especially with on line stabilization or cervical traction in place.

Full Stomach and Regurgitation Risk

As a general rule, all patients must come to the operating room starved (no solids for 6 hours, water allowed up to 2 hours preoperatively). One should assume that the stomach is not empty in injured or severely ill patients, in those who have received an opiate. Paralytic ileus is common following trauma and in patients with associated major medical diseases, therefore, delaying the anesthesia to allow the stomach to empty may not work. Hence, measures to reduce the risk of aspiration while otherwise proceeding with emergency induction, should be undertaken.[25]

Any method of anesthesia, including awakening techniques, can have an unexpected reaction that can, in theory, lead to unconsciousness, regurgitation and aspiration of stomach contents. One need to judge each case on its merits, balancing the risk of regurgitation and aspiration against the risks of general or spinal anesthesia. The general condition of the patient determines the risk of regurgitation more than the choice of technique.

Patients with high-risk of pulmonary aspiration of gastric contents or delayed gastric emptying are of following types:

- Patients with ileus, subileus, and bowel obstruction are considered nonfasting; irrespective of time elapsed since last meal or drink. Insertion of a nasogastric/duodenal tube in the ward prior to anesthesia induction is mandatory
- Presence of nasogastric tube itself can lead to regurgitation and aspiration
- Pregnant women of more than 20 weeks of gestation, including the first 24 hours postoperatively
- Patients with preoperative nausea/vomiting, e.g. newly started opioid pain treatment
- Patients with hiatal hernia, gastroesophageal reflux, or nausea or vomiting
- Morbidly obese patients (BMI > 35)
- Long standing diabetes mellitus (considering the risk of polyneuropathy and gastroparesis)
- Recently ingested full meal

Whenever possible, operate on a fasted patient: Aspiration is always possible. However, the risk of not operating is sometimes worse than the risk of aspiration, so the fasting rule is not an absolute one.

The risk of perioperative complications is reduced if patients can achieve the standards outlined below:

- H_2-blocker or proton pump inhibitor used to reduce gastric acidity and volume can reduce the morbidity of aspiration since acid is worse than neutral aspiration.
- Sodium citrate can be used to reduce acidity.
- Gastric prokinetic drugs like metoclopramide can be used. However, this should be avoided in intestinal obstruction or perforative peritonitis.
- Whenever a patient vomits, roll him to the side, drop his head downward, and suction his oropharynx immediately.

Preoxygenation and Cricoid Pressure

- Avoid a leak between the patients face and the oxygen mask
- Tidal volume breathing for 3 minutes or 8 deep breaths over 60 seconds with an oxygen flow of at least 10 L/min should be used

- Using 4 deep breaths over 30 seconds is a less effective procedure
- Noninvasive positive pressure ventilation or the application of positive end-expiratory pressure can be considered in the morbid obese or the critically ill hypoxic patients
- Preoxygenation in the obese patients should be performed in the head up position
- Use of cricoid pressure is not considered mandatory, but can be used on individual judgment
- Those choosing to use cricoid pressure in the patient at risk of aspiration must take care to apply the cricoid pressure correctly and release the pressure if ventilation or laryngoscopy and intubation prove difficult
- Cricoid pressure should be released before inserting the laryngeal mask airway (LMA) in case initial attempts at tracheal intubation prove unsuccessful
- Alternative plan of action should be ready for cannot ventilate cannot intubate (CVCI) situation
- Nasogastric tubes and the cricoid pressure may cause interference with airway management techniques. Sellick recommended that nasogastric tubes should be removed after final aspiration before induction of anesthesia as they might increase the risk of regurgitation and aspiration by tripping the esophageal sphincters[1] since nasogastric tube, occupying the part of the esophageal lumen, is not obliterated by the pressure on cricoid cartilage
- One may pull up the nasogastric tube in esophagus above the esophageal sphinter thereby making the esophageal sphincter competent. Once the patient is intubated the nasogastric tube can be advanced into the stomach.

Choice of Anesthetic Drugs

The induction agents with the most favorable pharmacological properties conferring hemodynamic stability appear to be ketamine and etomidate. Ketamine is traditionally contraindicated in the presence of brain injury. Ketamine represents a very rational choice for rapid sequence induction in hemodynamically compromised patients. If patient is stable any hypnotic drug can be used for induction and maintenance. The introduction of propofol and remifentanil has improved the recovery profile of patient. However, propofol does have dose dependent cardiovascular depression. Newer inhalational anesthetic agents like sevoflurane and desflurane with cardiovascular stability can also be used for induction and maintenance of anesthesia. Rapid recovery from newer inhalational agents may lead to emergence delirium due to delayed action of analgesics or regional techniques for postoperative pain relief.

Endotracheal intubation: The goal is to secure the airway with endotracheal tube without producing any regurgitation and vomiting.

The intubation procedure involves three objectives:

1. To prevent hypoxia during the induction intubation sequence.
2. To minimize the time between induction and tracheal intubation (Airway is unprotected by the patient's reflexes).
3. To apply measures to prevent pulmonary aspiration of gastric contents.

The first of these objectives is normally met by **preoxygenation of patient for at least 3 minutes.**

The second objective involves **minimization of the induction-intubation interval** which can be achieved by hypnotic agent followed by administration of rapidly acting neuromuscular blocking agent. Duration of laryngoscopy and intubation should be minimal.

Thirdly the chance of aspiration is diminished with **rapid sequence intubation (RSI)** by applying cricoid pressure, refraining from positive pressure ventilation after neuromuscular blocking agents and before tracheal intubation is accomplished. Laryngoscopy and intubation should be attempted only when neuromuscular blockade is adequate.

One must be ready with airway rescue plan in case intubation is not successful with three repeated attempts by experienced anesthesiologist.

Muscle relaxants are used to facilitate the intubation during rapid sequence intubation. Suxamethonium is the only drug which has rapid onset of action within 60 seconds. However, it does not come without its side effect like bradycardia, hyperkalemia, increased intracranial pressure (ICP) and in some sensitive cases it may precipitate malignant hyperthermia.

There is no substitute for the short duration of action of succinylcholine for aggressive airway management in the case of an unexpectedly difficult intubation in order to prevent life-threatening hypoxia.

Recently the intermediate acting nondepolarizing neuromuscular blocking drug like rocuronium in dose of 1 mg/kg and has been used for rapid intubation and it can be accomplished within 60 to 90 seconds.[26] With the introduction of sugamadex, new neuromuscular reversal agent the muscle relaxation can be reserved within 3 minutes. Thus can be helpful and life-savings in a situation, when patient cannot be ventilated and intubated after the administration of rocuronium

The **priming principle of relaxation** can also be used by giving 1/10th of the total dose 3 minutes prior to the

loading dose of muscle relaxant. This allows rapid onset of action of nondepolarizing muscle relaxant and one need not have to wait for 3 minutes for intubation. Earliest one can easily intubate the patient is 90 seconds.

Maintenance of anesthesia: Anesthesia can be maintained by either inhalational technique or total intravenous anesthesia with propofol. Narcotic sedation with fentanyl and sufentanil can be administered with caution. Intermediate acting neuromuscular blocking drugs are good choice for achieving muscle relaxation, e.g. vecuronium, atracurium, and rocuronium.

Reversal of Neuromuscular Blockade

At the end of surgery patients can be reversed with neostigmine. If patient condition demands post-operative ventilator support the patient need not be reversed and patient can be put on ventilator to provide controlled/assisted ventilation with or without PEEP.

Extubation

All patients who are extubated at the end of surgical procedure should be closely monitored till complete recovery. Take precautions to avoid aspiration at the end of anesthesia also. Patient who had difficulty in intubation, the extubation should be delayed till the patient is fully awake or extubation can be performed over the tube exchanger. In case of difficulty in ventilation or maintenance of airway following extubation, tube exchanger can be used for rapid and successful re-intubation.

Anesthesia Outside the Operation Room

For administration of anesthesia, all available induction agents can be used. Etomidate is a hemodynamically stable induction agent, however, the influence of possible etomidate-induced adrenocortical suppression must be considered. Total intravenous anesthesia can be used for maintenance of anesthesia. General anesthesia with endotracheal intubation and controlled ventilation can be given with usual monitoring. Rapid sequence intubation is considered the safest mode; however, awake intubation can also be performed in selected cases. Hypothermia should be taken care of in CT-Scan, MRI and cardiac catheterization department.[27] Patient under monitored anesthesia care should receive oxygen supplementation throughout the procedure.

Advantages of Local/Regional Anesthesia/ Analgesia

- Decreased blood loss
- Improved perioperative graft patency in vascular reconstruction
- Reduced incidence of venous thrombosis
- Combined regional and general techniques may improve outcomes in selected cases
- Can be used in significant cardiovascular disease, severe pulmonary disease and major abdominal or thoracic surgery
- Pre-emptive analgesia with epidural anesthesia enhances perioperative comfort of the patient
- Drug interaction with chronic medications can be minimized (antihypertensive, antiarrhythmics, and narcotics, sedatives and insulin) by avoiding general anesthesia.

Local anesthesia is always the safest technique whenever the patient's are with stomach.

The Limitations of Local Anesthetics

- Limited amount of local anesthetic agent safe to inject.
- The inability to inject through infected tissue.
- The difficulty of its use on children and anxious patients
- Supplement local anesthesia required with intra-venous anxiolytic agents whenever necessary.
- Allergy to local anesthetic agents.

The most commonly used local anesthetic agent is 2 percent plain lidocaine with or without addition of epinephrine. More concentrated solutions are needed for nerve blocks while solutions as dilute as 0.25 percent are effective for infiltration anesthesia. While injecting local anesthetic agent, use the finest possible needle available which will not break. Inject while pushing the needle forward (as well as when withdrawing the needle) and if needed, bend the long needle such that it remains parallel to the skin during injection thereby allowing injection in the correct plane. Ideally, a patient should feel only a single needle prick per wheal. Whenever possible, subsequent injections should be performed through skin that has already been anesthetized.

Field Blocks/Peripheral Nerve Plexus Block

Peripheral nerve block or field block is an alternative to simple infiltration anesthesia. This is most useful for blocking sensation of a large patch of skin, where as local infiltration would require multiple smaller injections, e.g. surgeries on extremities.

Performing the peripheral nerve block/plexus block under ultrasonic guidance (USG) improves the success and quality of block. The USG is very helpful in patient who cannot be given proper position to perform the plexus block in view of severe pain, burns contractures, rheumatoid arthritis, etc.

Central Regional Blocks

Spinal, epidural or combined spinal epidural anesthesia/analgesia can be given.

One must ensure before institution of central regional block that the patient is either hemodynamically stable or stabilized.

If patient is in severe respiratory compromise, severe abdominal distension, strangulated hernia or uncomfortable in supine position, it is advisable to give general anesthesia and control the ventilation, in order to maintain the oxygenation.

If patient coagulation status is in doubt or patient is on antiplatelet or anticoagulant therapy, it is better to proceed with general anesthesia.

Ketamine for Analgesia

In areas where morphine is expensive or not widely available, patients suffering with severe pain, as from cancer or multiply fracture ribs, can receive ketamine by low dose infusion. To infuse ketamine for analgesia, first give a small bolus (0.25 mg/kg) of ketamine intravenously and dose should be titrated upward carefully to eliminate pain but not produce dissociation, followed by 0.5 mg/ml ketamine in saline to be given at a rate of 0.5 to 1 mg/kg/hr.

Postoperative Care

Postoperative anesthetic issues are to be managed promptly in order to reduce the morbidity and mortality. Some of them are as follows:

- *Oxygenation:* Oxygenation and airway stability has to be monitored clinically as well as with pulse oximeter. All patients should be given oxygen supplementation until they are fully awake and stabilized.
- *Need for reintubation:* Some patients may need reintubation following the extubation. This may be either immediate or within few hours postoperatively. Patient's need to be monitored for adequacy of breathing very closely and emergency intubation tray must be kept ready next to patient in order to prevent hypoxia and its consequences.
- *Inadequate neuromuscular block reversal:* One must monitor the neuromuscular blockade following reversal of non-depolarizing muscle relaxants. The peripheral nerve stimulator helps in guiding the reversal of neuromuscular blockade and should be used whenever available. The inadequate reversal is observed during emergency cases since these patient may have acidosis, presence of electrolyte abnormalities especially hypokalemia and inadvertent hypothermia associated with infusion of large amount of intravenous fluid, blood and blood products within short period of time.
- *Mechanical ventilation:* There are innumerable causes for a patient to be mechanical ventilated. Few of the major reasons are inability to maintain oxygenation with spontaneous breathing, increased work of breathing, supramajor surgery, COPD patient and hypothermic patient. The mode of ventilation is decided on individual case basis.
- *Pain:* Pain is the major cause of agitation, hypertension, tachycardia, increased oxygen demand and hypoventilation due to diaphragmatic splinting in upper abdominal surgery. The postoperative pain relief should be planned preoperatively whenever possible. This can be provided either with parenteral acetaminophen, NSAIDS and narcotics like morphine, fentanyl or sufentanil. The drugs can be given intravenously either with intermittent doses, through infusion with or without patient controlled analgesia. Local infiltration at incisional site, peripheral nerve block, regional analgesia can be good option unless they are contraindicated.
- *Nausea, vomiting and aspiration:* There is high incidence of postoperative nausea, vomiting in patients who have received general anesthesia as compared to the one who received local or regional anesthesia. The incidence of nausea and vomiting can be reduced by giving optimum fluids, antacids (e.g. ranitidine, penteprozole), and prokinetic (e.g. metoclopramide, domperidone) and 5HT blockers (e.g. ondensetron, granisetron). The patient whose stomach was not empty preoperatively may be at risk of aspiration postoperatively as well, hence acid aspiration prophylaxis should be repeated postoperatively in those patients.
- *Temperature regulation:* Patient should be actively warmed using patient warming devices in case of hypothermia. Neonates, children and geriatric patients need special care as their temperature regulation mechanism is compromised. Hypothermic patient is at risk of hypoventilation, metabolic acidosis and increased bleeding.
- *Hypertension/hypotension:* Patient can have hypertension because of postoperative pain or pre-existing hypertensive patient. This should be treated promptly in order to prevent cardiac arrhythmias, myocardial ischemia, etc. There is a likely chance of postoperative bleeding due to sudden rise in blood pressure and inadvertent slippage of ligature of vessel. There can be postoperative hypotension due to residual effect of anesthetic agents, regional

block inadequate preload, myocardial infarction, concealed bleeding, etc. Therefore, a patient's blood pressure should be monitored at least every 15 minutes. In high-risk and hemodynamically unstable patient it is better to measure blood pressure invasively to monitor beat to beat variability in blood pressure.

- *Cardiac arrhythmias:* Patient's rhythm should be monitored with cardioscope and arrhythmias should be treated promptly.
- Acute metabolic disturbances, electrolyte derangements and fluid imbalance (dehydration or overhydration) are likely. If there is ongoing loss of fluid and blood from drains, nasogastric tubes and urine output. These need to be detected in time and treated promptly.
- The estimation of electrolytes should be done at least 4 to 6 hourly.
- The fluid should be administered with the monitoring of central venous pressure and urine output.
- Acute renal failure is observed many times in emergency cases with trauma patient, septicemic patient, preoperatively dehydrated or in patients with long standing hypotension. One should try to prevent perioperative renal failure by using renal protective strategy. This involves maintaining intraoperative blood pressure, fluid and electrolyte or renal replacement therapy. Low dose dopamine does not protect renal function but fenoldopam has been shown to protect it.
- *Postoperative cognotive dysfunction (POCD):* Postoperative delirium and confusion is commonly observed in geriatric patient and its incidence is more whenever atropine has been used intraoperatively. The delirium and confusion normally last from few hours to 24 hours. One should keep in mind that it can be due to cerebrovascular causes and may need to be thoroughly evaluated.
- *Deep vein thrombosis (DVT):* DVT prophylaxis should be given whenever heparin or low molecular weight heparin (LMWH) is not contraindicated because of ongoing bleeding issues. The high-risk patients for developing DVT are obese patient, geriatric patient, obstetric patient, chronic immobilation and lower limb trauma, etc. The patient can be given calf muscle exercises by sequential compression and decompression device, foot pump and physiotherapy.
- Use of aseptic precaution and barrier nursing will prevent infection.
- Anesthesiologist need to decide how long a patient can remain in postanesthesia care unit or will need to be cared in specialized intensive care units.

Regardless of choice and method of anesthesia management, one must adhere to certain general principles. These include maintenance of patent airway, adequate ventilation with proper oxygenation and normal carbon dioxide concentration with hemodynamic stability. The morbidity and mortality can be significantly reduced if expert personnel are available to manage unanticipated difficult intubation and for the crisis management.

Therefore, assess the risk and stress of the proposed surgery and it's relative to its benefit, in view of the physiological reserve of the patient. Thereby adjust the technique of anesthesia and choice of drugs accordingly for the optinal outcome.

REFERENCES

1. Amir AK, Imad Abu el-Naaj, Michal B. Maxillofacial tauma patient: coping with the difficult airway. World Journal of Emergency Surgery 2009;4:21.
2. Runciman WB, Kluger MT, Morris RW, Paix AD, Watterson LM, Webb RK. Crisis management during anesthesia: Crisis Management Manual, research @apsf.net.au
3. Michael A, José P. Perioperative care. Anesthesia, pain management and intensive care 2002;104.
4. Rupert MP, Andrew R, Michael RG. Clinical review. How to optimize management of high-risk surgical patients. Intensive Care Medicine Critical Care 2004;8:503-7.
5. Mythen MG, Webb AR. Perioperative plasma volume expansion reduces the incidence of gut mucosal hypoperfusion during cardiac surgery. Arch Surg 1995; 130:423-9.
6. Guest JF, Boyd O, Hart WM, Grounds RM, Bennett ED. A cost analysis of a treatment policy of a deliberate perioperative increase in oxygen delivery in high-risk surgical patients. Intensive Care Med 1997;23:85-90.
7. Denny Z, Levvett H. et al. The effects of fluid optimization on outcome following major surgery. Transfusion Alternatives in Transfusion Medicine 2002;4:(3)75.
8. Michand F, Teboule J-L. Predicting fluid responsiveness in ICU patients; A critical Analysis Evidence. Chest 2002; 121: 2000-8.
9. Jardin F, Viellard-Baron A. Ultrasonographic examination of venacavae. Intensive Care Med 2006;32:206-8.
10. Rivers EP, Ander DS, Powell D. Central venous oxygen saturation monitoring in the critically ill patient. Curr Opin Crit Care 2001;7:204-11.
11. Michael GM. Postoperative gastrointestinal dysfunction. Clivland clinic medical journal 2009;76(Supplement4):566-71.

12. Corbett EJ, Barry BN, Pollard SG, Lodge JP, Bellamy MC. Laser Doppler flowmetry is useful in the clinical management of small bowel transplantation. Gut 2000; 47:580-3.
13. Edsander-Nord A, Rojdmark J, Wickman M. Metabolism in pedicled and free TRAM flaps: a comparison using the microdialysis technique. Plast Reconstr Surg 2002;109:664-73.
14. Thorniley MS, Sinclair JS, Barnett NJ, Shurey CB, Green CJ. The use of near-infrared spectroscopy for assessing flap viability during reconstructive surgery. Br J Plast Surg 1998;51:218-26.
15. Lewis SA, Donalson P. Ion channels and cell volume regulation: chaos in an organized system. News physio Sci 1990;5:112-8.
16. Brienza N, Giglio MT, Marucci M, Fiore T. Does perioperative hemodynamic optimization protect renal function in surgical patients? A meta-analytic study. Critical Care Medicine 2009;37(6)2079-90.
17. Wilkes NJ, Woolf R, Mutch M, et al. The effect of balanced versus saline based hetastarch and crystalloid solution on acid-base and electrolyte status and gastric mucosal perfusion in elderly surgical patients. Anaesth. Analg 2001; 93($):811-6.
18. Moretti EW, Robertson KM, EL-Moaelem H, Gari TJ. Intraoperative colloid administration reduces postoperative nausea and vomiting and improves postoperative outcome compared with crystalloid administration. Anaesth. Analg 2003;96(2):611-7.
19. Nguyen NQ, Ng MP, Chapman M, M Fraser RJ, Holloway RH: The impact of admission diagnosis on gastric emptying in critically ill patients, Crit Care 2007;11R:16.
20. Ramos CG. Management of fluid and electrolyte disturbances in the burn patient. Annals of Burns and Fire Disasters 2000; vol. XIII: n4.
21. HJ Sparr. Choice of the muscle relaxant for rapid-sequence induction. European Journal of Anaesthesiology 2001; 18: 71-76.
22. Timmermann AR, SG Hollmann MW. Guidelines for RSI. Acta Anaesthesiol Scand 2008;52:897-907.
23. Shoemaker WC, Appel PL, Kram HB, Waxman K, Lee TS. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients. Chest 1988; 94:1176-86.
24. Wilson J, Woods I, Fawcett J, Whall R, Dibb W, Morris C, McManus E. Reducing the risk of major elective surgery: randomized controlled trial of preoperative optimisation of oxygen delivery. BMJ 1999;318:1099-1103.
25. Bishop MH, Shoemaker WC, Appel PL, Meade P, Ordog GJ, Wasserberger J, Wo CJ, Rimle DA, Kram HB, Umali R, et al. Prospective, randomized trial of survivor values of cardiac index, oxygen delivery, and oxygen consumption as resuscitation endpoints in severe trauma. J Trauma 1995;38:780-7.
26. Lobo SM, Salgado PF, Castillo VG, Borim AA, Polachini CA, Palchetti JC, Brienzi SL, de Oliveira GG. Effects of maximizing oxygen delivery on morbidity and mortality in high-risk surgical patients. Crit Care Med 2000; 28: 3396-3404.
27. Gordon JR, Weatherhead DSP, Van Bergen FH. Anesthesia for emergency surgery. Survey of Anaesthesiology 1958; 1(5):487-91.

2 Medical Disorders and Emergency Anesthesia

Manju N Gandhi, Amala G Kudalkar, Kamlesh G Gotiwale, Devendra W Thakare

KEY POINTS

- The average age of a patient has increased with the medical advances hence, we get more and more patients with medical disorders. We will highlight few of the common associated medical diseases and their management during emergency surgery and anesthesia.
- Thyroid hormones stimulate growth, increase oxygen consumption and produce heat. They speed up the activity of all systems of the body.
- Whenever feasible try to make patient euthyroid prior to surgery in order to avoid thyroid storm in hyperthyroidism and myxedema coma in patient with hypothyroidism. Thyroid patient may be a candidate for difficult intubation due to thyroid swelling or obesity.
- According to WHO guidelines, when random plasma glucose is >11.1 mmol/L or 200 mg/dl or fasting blood sugar >7 mmol/L or 126 mg/dl on two separate occasions, the diagnosis of diabetes is confirmed. More and more patients are diagnosed to have diabetes possibly because of the redefining diagnostic criteria as blood sugar of ≥126 mg percent.
- Diabetic patients have wide spread microangiopathy resulting in ischemic heart disease, cerebrovascular disease, peripheral vascular disease, renal disease, retinopathy and peripheral neuropathy.
- Stress of surgery results in release of counter-regulatory hormones such as epinephrine, nor-epinephrine, glucagon, cortisol and growth hormone. These stress hormones have anti-insulin action and counter the anabolic effect of insulin. This causes marked hyperglycemia. Hyperglycemia can also lead to hyperglycemic, hyperosmolar, non-ketotic coma. Starvation can produce ketosis and there can be associated electrolyte abnormalities, which has to be addressed promptly.
- Stress, sedation and general anesthesia often produce gastrointestinal instability leading to nausea, vomiting and dehydration. Acid aspiration prophylaxis has to be taken.
- Sedation and anesthesia can mask the signs of hypoglycemia and a high degree of alertness is required on part of the anesthesiologist.
- The choice of anesthesia can affect the outcome by modulating the secretion of the catabolic hormones and by interfering with the early resumption of enteral nutrition.
- Presence of nephropathy, ischemic heart disease, hypertension and autonomic neuropathy should prompt the anesthesiologist to choose the drugs carefully. Acid aspiration prophylaxis should be taken.
- Acute renal failure is a common and in many cases it is a preventable and/or eminently treatable problem. Thorough preoperative evaluation of acute renal failure (ARF) patient, whether it is due to prerenal, renal or postrenal cause is essential. Correct the cause preoperatively whenever possible.
- In CRF, correction of electolyte imbalance, metabolic acidosis, anemia, platelet correction should be done preoperatively.
- Intraoperatively try to maintain the urine output of at least 0.5 ml/kg/min. Use of fenoldopam or mannitol in case of acute oliguria may be helpful.
- Fluid administration is guided with central venous pressure. Hemodialysis may be needed if hypervolemia is present. Avoid nephrotoxic drugs. Postoperative recurirazation should be watched for in anephric patients.
- Hypertension is usually symptomless but if untreated it can result in enlargement of heart or failure, renal dysfunction and cerebrovascular accidents.
- Severe untreated hypertension (diastolic BP >120 mm Hg) in the perioperative period may lead to serious complications such as myocardial infarction, left ventricular failure, hypertensive encephalopathy or renal failure.

- Rapidly acting antihypertensive agents like nitroglycerine, β-blockers can be used to allow effective control of BP. Care should be taken not to cause precipitous fall of BP or any surges in rise in BP due to airway manipulation, light plane of anesthesia or during emergence from anesthesia.
- Spinal and epidural anesthesia can cause unpredictable and profound arterial hypotension in poorly controlled hypertensive patients.
- Patients with extensive three vessel coronary artery disease (CAD), a history of myocardial infarction (MI) or ventricular dysfunction are at greatest risk for cardiac complications.
- If the noncardiac surgery is urgent or emergent, then cardiac risk, the risk of bleeding and the benefit of coronary revascularization must be weighed. The anesthetic goal is to maintain the balance between myocardial oxygen demand and supply in order to prevent myocardial ischemia.
- β-blockers reduces perioperative ischemia and may reduce the risk of MI and mortality in patients with known CAD. The dose being titrated to achieve heart rate of 60 to 65 bpm.
- Hypoxia, tachycardia, pain, hypothermia, anemia and shivering should be treated aggressively in the postoperative period to prevent myocardial ischemia.
- Amongst all valvular heart lesions, the stenotic lesions are more dangerous then regurgitant lesions. Cardiac output depends on atrial kick for adequate LV filling. Tachycardia should be avoided in stenotic lesion and mild tachycardia is ok with regurgitant lesions.
- Preoperative antibiotic prophylaxis is an important aspect of patient management in patient with valvular heart disease.
- Hypoxia, hypercarbia and acidosis should be avoided as they exacerbate pulmonary hypertension.
- Chronic obstructive lung disease (COPD) patients should be evaluated thoroughly and optimized prior to surgery (whatever short period available) as they not only create problems intraoperatively but also contribute to a significant degree in the development of post-operative pulmonary complications.
- The quantification of respiratory dysfunction should be made on the basis of arterial blood gas (ABG) analysis, since it can be obtained within a short time.
- Anesthetic plan should include prevention of triggers which precipitate bronchospasm for, e.g. drugs (pentothal, morphine, muscle relaxants which releases histamine), light plane of anesthesia, endobronchial intubation, etc.
- Hypercarbia, hypoxemia and acidemia promote arrhythmias and may impair the response to bronchodilator therapy.
- Regional anesthesia is advocated as and when possible, however one should avoid higher level of regional block thereby avoiding the hypoventilation. The patient who is breathless at rest should be given general anesthesia in order to control the ventilation to maintain oxygenation.
- In selecting a ventilatory mode for COPD patient, attention should be given to providing an adequately long expiratory time to avoid the build up of intrinsic or auto-PEEP. This can be facilitated by using higher inspiratory flow rates or smaller tidal volumes.
- Postoperative pain control be it by the neuraxial route or PCA, bronchodilator therapy, incentive spirometry, deep breathing exercises, early mobilization and control of gastroesophageal reflux is beneficial in COPD patients.
- Patient with liver disease is assessed thoroughly and risk assessment is done with Child Pugh classification.
- Coagulation abnormality and metabolic acidosis should be corrected preoperatively.
- The kidneys are at risk of development of hepatorenal syndrome and pigment obstruction of the renal tubules leading to postoperative renal failure.
- Preoperative optimization includes correcting coagulopathy, ascites, hepatic encephalopathy and administering antibiotic prophylaxis.
- Presence of hypoalbuminemia impairs drug binding and doses of sedative, hypnotic and muscle relaxants should be meticulously titrated.

With the advances in new technology and therapeutics, the average age of a patient has increased. Therefore, more and more old age patients with associated medical disorders are exposed to surgeries. Also, as a result of the increased level of stress, lifestyle and dietary habits, the diseases of the middle age, such as hypertension, diabetes and ischemic heart disease are appearing at younger age. It is not surprising that the incidence of patients with medical disorders for routine surgeries is increasing. However, this does not pose a great problem for the anesthesiologist as there is enough time at disposal to treat or control these conditions. It is the emergency situation that is worrisome as the patient is neither investigated nor treated properly. In this

chapter, we are going to discuss certain commonly encountered medical disorders in an emergency patient. They are:

1. Patient with thyroid disease
2. Patient with diabetic mellitus
3. Patient with renal disease
4. Patient with hypertension
5. Patient with ischemic heart disease
6. Patient with valvular heart disease
7. Patient with chronic obstructive pulmonary disease (COPD)
8. Patient with liver disease

The detailed pathophysiology, diagnosis and treatment of each condition are beyond the scope in this book. Hence, only the salient points about the history, examination, investigations and treatment are mentioned. The stress is given on emergency management of the condition and its anesthetic implications.

PATIENT WITH THYROID DISEASE

The thyroid gland is responsible for maintaining the optimum level of metabolism in the tissues leading to their normal functioning. It stimulates growth, speeds the metabolic reactions, increases oxygen consumption and heat production.[1] Its absence causes physical, metabolic and mental slowing, mental retardation in children and dwarfism. Thyroid gland secretes two hormones tri-iodothyronine (T3) and tetra-iodothyronine or thyroxine (T4). Over or underproduction of these hormones leads to metabolic and hemodynamic derangement in the body.

A patient with thyroid disease can come for emergency surgery. Patient may have overt thyroid disease and may be already on medication for it or may have a subclinical disease, unmasked for the first time during emergency surgery. It is very important to achieve a euthyroid state while administering anesthesia as infection, fever, diabetes, trauma, stress of surgery as well as anesthesia, all can precipitate thyroid storm. Also, if the patient is hypothyroid, there may be prolonged recovery from anesthesia with a possibility of precipitation of myxedema coma.

Preoperative Evaluation in case of Thyroid Disease

Whenever a patient with suspected thyroid disease comes for emergency surgery, he should be quickly assessed in the following way.

History

- Origin, duration and progress of the disease. Whether it is long standing or any sudden increase in the size of the gland.
- History suggestive of hyperthyroidism, hypothyroidism and cardiac involvement. (Tables 2.1 and 2.2).

Table 2.1: Signs and symptoms of hyperthyroidism

Anxiety	Increased appetite
Restlessness	Weight loss
Increased warmth	Insomnia
Heat intolerance	Hyperdefecation
Tachycardia	Fatigue
Bounding pulse	Eye signs
Thyroid swelling	Muscle weakness

Table 2.2: Signs and symptoms of hypothyroidism

Lethargy	Bradycardia, hypotension
Somnolence	Hypoventilation
Decreased appetite	Enlarged tongue
Weight gain	Peripheral vasoconstriction
Cold intolerance	Myxoedematous appearance
Pretibial edema	Constipation
Stupor, coma	Carpel tunnel syndrome

- History of pressure symptoms due to thyroid swelling, e.g. dysphagia, dyspnea, dysphonia and giddiness, both in supine as well as sitting position.
- History of any drug treatment for the same or radioactive iodine therapy.
- Any thyroid surgery performed in the past and whether any hormone replacement therapy was prescribed to the patient.

Physical Examination

- **Airway examination**: Increased soft tissue deposition in tongue and pharynx as seen in myxedematous patients makes mask ventilation and intubation difficult. Presence of thyroid swelling itself may cause difficult airway from pressure and proximity causing deviation and compression of trachea.

- **Careful examination of the cardiovascular system**: As this is the system that bears the brunt of the hyperactivity of the thyroid gland.
  - *Pulse:* Tachycardia, bradycardia, dysrhythmias. The tachycardia may be due to the basic surgical pathology, pain, abdominal distension, reflex following hypotension and may falsely be considered to be due to hyperthyroidism.
  - *Blood pressure:* May have hypertension, or hypotension.
  - *Heart sounds :* Look for third heart sound in case of heart failure.
  - *Presence of murmur:* As in hyperdynamic circulation in hyperthyroidism.
  - *Pericardial rub:* May be present in myxedematous heart.
- Auscultation of thyroid bruit indicating hyperactivity of the gland.
- Auscultation of lung fields for crepitations to rule out ventricular failure.
- Any other sign of thyroid hyperactivity such as tremors, eye signs, etc. should be sought for.

Investigations

It is not possible to send elaborate investigations. As soon as the patient is admitted to the emergency room, after initial history and examination, blood should be sent for following investigations:

- **Hb, complete blood count:** Patient on antithyroid drugs can have leukopenia. Both hyper and hypothyroidism can have anemia.
- **Fasting blood sugar:** As the patients often have associated diabetes.
- **Renal function tests and serum electrolytes:** Patients with hyperthyroidism have severe dehydration and electrolyte imbalance. Hypothyroid patients often exhibit hyponatremia.
- **Arterial blood gases:** To know the acid-base status as the patients may have acidosis.
- **Thyroid function test:** Blood should be sent for thyroid function tests such as T3, T4 and TSH. The reports may not be available by the time of inducing anesthesia, but often guide in the further therapy in the postoperative period as well as help to substantiate the clinical diagnosis.
- **Chest X-ray:** To know pulmonary congestion secondary to heart failure, dilatation of the heart in hypothyroidism and may help in diagnosing the retrosternal goiter.
- **Neck X-ray:** Anterior and lateral view to look for deviation and compression of the trachea in case of thyroid swelling.
- **Cardiogram**: Absolutely necessary as often depicts tachycardia, tachyarrhythmias (hyperthyroidism), bradycardia, low voltage ECG (hypothyroidism).
- **Target echocardiography:** To know the heart function as there is often heart failure.
- Other investigations may be done as per the requirement of diagnosis and surgery contemplated.

A patient with thyroid disease[3] is likely to be on following medications:

- **Antithyroid drugs:** Carbimazole or methimazole
  Propylthiouracil
  They inhibit iodine trapping and coupling of tyrosine residues, thereby inhibiting the synthesis of thyroid hormones.
- **Inorganic iodine in Lugol's iodine, sodium or potassium iodide:** They inhibit the release of thyroid hormones.
- **β-adrenergic antagonists:** They mask the signs of adrenergic activity. They do not affect the thyroid gland function but do inhibit the peripheral conversion of T4 to T3.
- **Radioactive iodine therapy.**
- **T4 (Eltroxin)** in case of hypothyroid patient.

Hyperthyroid Patient

Thyrotoxicosis is the state that arises when tissues are exposed to excessive quantities of thyroid hormones. When it occurs as a result of hyperactivity of thyroid gland, it is termed as hyperthyroidism.[2] Signs and symptoms of hyperthyroidism are described in Table 2.1.

The main problem with a hyperthyroid patient is intense sympathetic nervous system stimulation and hypersensitivity of β-receptors. The signs and symptoms described above can be attributed to this stimulation.

Risk Factors in a Hyperthyroid Patient

- There is intense sympathetic nervous system stimulation leading to severe tachycardia, hypertension, dysrhythmias, the most important being atrial fibrillation. There may be congestive heart failure.
- These patients can have high circulating levels of endogenous catecholamines making them sensitive to the action of inhalational anesthetic agents like halothane on myocardium. It can readily precipitate dysrhythmias.
- Patient can be delirious and in altered state of consciousness.

- Patient not adequately rendered euthyroid, can develop severe exaggeration of symptoms of hyperthyroidism, a condition termed as 'thyroid storm' or 'thyroid crisis'. It is a condition characterised by severe hypermetabolism, hyperthermia, tachycardia and agitation, rapidly leading to hypotension, coma and death. It has mortality up to 20 percent. Usually it occurs in the postoperative period but can occur intraoperatively.
- As a result of hyperthermia (they often mimic malignant hyperthermia) and increased metabolism, they are dehydrated and severely volume depleted. There can be exaggerated hypertensive response to laryngoscopy and intubation. On the other hand, anesthetic agents like thiopentone sodium and propofol can cause severe hypotension due to peripheral vasodilatation in face of hypovolemia.
- These patients often have adrenocortical insufficiency.
- Proximal muscle weakness can significantly reduce respiratory reserve and this may come in way of postoperative adequate spontaneous breathing.

Preparing a Hyperthyroid Patient for Emergency Surgery

It is a dictum that unless the patient is rendered euthyroid, he should not be subjected to any operative procedures for the fear of inducing thyroid crisis. This is obviously not possible in emergency situation.

The Principles of Management[4]

- Rapid reduction of thyroxine production.
- Blocking the release of thyroid hormones into the circulation.
- Peripheral antagonism of thyroid hormone actions.
- Rapid correction of hyperthermia, dehydration, metabolic disturbances and acidosis.
- Correction of hyponatremia.

Rendering patients euthyroid by using antithyroid drugs takes minimum 2 to 6 weeks. When iodides are used, it takes 7 to 14 days for the thyroid hormone levels to recede. This approach may prove to be useful as it takes less time and shrinks the gland by causing thyroid constipation.[5] However, when confronted by a hyperthyroid patient for emergency surgery that must be undertaken within a matter of hours, these drugs are of little value.

The case should be undertaken after careful discussion with surgeons regarding the urgency of the surgery as even a few hours delay is sufficient to take remedial measures to reduce the thyroid activity. The risks of delay should be balanced against the risks of surgery in a poorly prepared hyperthyroid patient.[6]

Steps in Management of Thyroid Storm[3,5,7]

1. Establishment of venous access with wide bore intravenous (IV) lines and central venous catheter in place to guide the therapy. Every effort should be made to combat dehydration.
2. Administration of oxygen by mask as its requirement is increased tremendously.
3. Propylthiouracil (PTU) through Ryle's tube every 6 hourly. It is expected to initiate action within 2 to 3 hours.
4. Sodium or potassium iodides (saturated solution of KI) orally 1 gm over 12 hours only after PTU has been administered. Otherwise the iodine present in these preparations itself can precipitate thyroid crisis.
5. Iopanoic acid, Lugol's iodine is the other valuable preparations which can be started after PTU.
6. The nonselective β-adrenergic antagonists form the mainstay of therapy. They block the peripheral effects of thyroid hormones on the cardiovascular system. They improve the symptoms without actually decreasing the outflow of thyroid hormones. The commonly used drug is Inj. propranolol 1 to 2 mg given IV slowly, followed by 40 to 80 mgs orally. Inj. Metaprolol also may be given to reduce heart rate and sympathetic activity. Hyperthyroid patients having higher ventricular rate and who are in CHF actually benefit from b blockers. However, it should be noted that beta-adrenergic antagonists do not invariably prevent thyroid storm. If tachycardia and dysrhythmias persist in spite of beta-blockers, then it is advisable to add diltiazem 60 to 120 mg orally.
7. These patients often have adrenocortical insufficiency which is aggravated by the most stressful condition of emergency surgery and anesthesia. Therefore, steroid supplements should be given. Inj. hydrocortisone 100 mgs 8 hourly followed by maintenance with Inj. prednisolone.
8. Correction of the precipitating cause.

Anesthetic Considerations

- Render patient euthyroid as far as possible and achieve adequate β-blockade.
- Replacement of fluid deficit, electrolytes imbalance. Many a times the consequences of the surgical pathology itself can mimic hyperthyroidism, e.g. patient with abdominal sepsis can have

hyperthermia, tachycardia, hyperventilation as a result of pain and abdominal distension.
- Sedation and anxiolysis for anxiety.
- Regional and subarachnoid block[7] are preferable as they avoid the multiple drug administration. It also helps to reduce the sympathetic outflow. Care must be taken however, to preload the patient adequately.
- All drugs that cause sympathetic stimulation such as ketamine, pancuronium, etc. should be avoided.
- Metabolism of drugs is increased and therefore their requirement is increased.
- Adrenaline infiltration is contraindicated.
- Atropine is avoided as it increases the already existent tachycardia and also because it impairs the sweating mechanism, making the situation worse.
- Inhalational anesthetic like halothane is avoided as it sensitizes the myocardium to the high levels of circulating catecholamines that are present in hyperthyroid patient.
- Strict monitoring of fluid balance, temperature monitoring is mandatory. Sudden rise in temperature should make one suspicious about the beginning of the thyroid storm and all efforts must be directed at arresting its progress. Temperature should be brought down by tepid sponging, intravenous fluids, decreasing ambient temperature and anesthetic gases.
- Other monitoring device that can diagnose early the onset of burst of hypermetabolism is the $EtCO_2$. There is a sudden rise in $EtCO_2$ which can alarm the anesthesiologist about the possibility of the crisis ahead.
- Patient may need hemodynamic and ventilatory support in extreme cases.

Hypothyroid Patient

Risk Factors in a Hypothyroid Patient

This condition presents special challenge to the anesthesiologist especially, because it may not be diagnosed preoperatively and can only be suspected postoperatively when any complication occurs. All elective surgeries should be deferred till the patient becomes euthyroid. Patients with mild to moderate hypothyroidism do not pose increased risk as per clinical studies. However, possibility of adverse effects should be borne in mind.

- These patients are often obese, with short neck, large tongue, swollen oral cavity, edematous vocal cords and pad of fat at the back of the neck, making them a candidate for difficult airway.
- Cardiovascular depression: They often have bradycardia, decreased stroke volume, reduced cardiac output, and depressed baroreceptor reflexes.
- Intense peripheral vasoconstriction and hypovolemia.
- Presence of anemia.
- Delayed gastric emptying.
- Increased sensitivity to opioids, inhalational and intravenous anesthetics. Impaired ventilatory responses to hypoxia and hypercarbia.
- There is potentiation of action of sedatives, narcotics and anesthetic agents. Often there is prolonged recovery from anesthesia. Generalized decrease in the metabolic activity causes reduced metabolism of the drugs, thereby increasing their duration of action and increased sensitivity of their effects.
- The MAC of the inhalational anesthetic agent is not affected much but patient appears to have an increased sensitivity to them secondary to reduced cardiac output, blood volume, abnormal baroreceptor function, decreased hepatic metabolism and renal excretion.
- Decreased neuromuscular excitability and prolonged effects of both depolarizing as well as nondepolarizing muscle relaxants.
- Extremely prone to develop hypothermia, hyponatremia and hypoglycemia
- There is often adrenocortical insufficiency that blunts the stress response.

Preparing a Hypothyroid Patient for Emergency Surgery

Similar to the hyperthyroid patient, again it should be noted that failure to render patient euthyroid prior to anesthesia and surgery can prolong the recovery and may even precipitate myxedema coma. Hormone replacement with a combination of tri-iodothyronine and thyroxine appears to be the best approach. Levothyroxine 100 to 200 µgm/day would otherwise be a preferred therapy as it allows the controlling enzyme systems to regulate the TSH secretion. The decreased myocardial function and the ventilatory response come to normal within 3 to 6 months. Obviously in emergency setting this much time is not available. This is one indication of giving intravenous thyroxine therapy. Both T3 and T4 can be given intravenously. Intravenous T4 300 to 500 µgm is given as a single dose. Intravenous T3 may also be given in doses of 25 to 50 µgms every 8 hourly. It is effective within 6 hours with a peak in basal metabolism seen in 36 to 72 hours. It is probably superior to T4 because it is the physiologically active

form of thyroid hormone and bypasses the normal peripheral conversion of T4 to T3 which is markedly depressed in severely ill patients. Patients for emergency surgery should be treated by T3 rather than T4.[8] Steroid coverage with hydrocortisone or dexamethasone is necessary as decreased adrenal-cortical function is often present.

Hypothyroidism with IHD

The problem arises when hypothyroid patients have concomitant ischemic heart disease. The symptoms of IHD are often masked as a result of marked decrease in metabolism and hence oxygen consumption. As hypothyroidism is being corrected and metabolism returns to normal, patients with fixed coronary obstruction are unable to compensate for this increased demand. The underlying myocardial ischemia becomes unmasked and frank infarction can be precipitated. This may be a limiting factor while achieving adequate euthyroidism preoperatively. Patients presenting with both should have their angiographic evaluation of their coronary arteries done before hormone replacement is attempted. Achieving control of hypothyroid patient with well documented ischemic heart disease posted for emergency surgery is extremely challenging and one has to accept little less than optimum management of hypothyroidism. The need for thyroid replacement therapy should be carefully balanced against the risk of aggravating myocardial ischemia.

Patient may need coronary revascularization on emergency basis in presence of severe coronary artery disease and significant hypothyroidism. Thyroid replacement therapy may also be initiated in the recovery room postoperatively or a combination of both β-blockers and L-thyroxin can be given. This should be accompanied by adrenocortical replacement therapy as adrenocortical insufficiency often accompanies hypothyroidism.

However, whenever patient has to undergo another emergency noncardiac surgery, the priority should be given to the correction of surgical pathology, followed by slow titration of thyroid replacement therapy. Anti-anginal drugs will have to be added to the therapy.

Presence of hypothyroidism with ischemic heart disease is to be considered equivalent to the left main disease with unstable angina.

Anesthesia Considerations

- These patients are often hypovolemic and have hyponatremia. Initiation of thyroid replacement therapy corrects these problems.
- These patients are extremely sensitive to sedatives, narcotics and anesthetics. One should be cautious in administration of sedatives. Usually a preoperative visit and a good rapport will suffice. If required sedatives and narcotics can be added in the operation theater so that patient can be monitored well. Regional anesthesia is recommended when there are no particular contraindications.

Induction of Anesthesia

Owing to the increased sensitivity of the patient to the intravenous drugs, the drug should be titrated very judiciously. Ketamine is the preferred induction agent as it does not cause fall in heart rate or blood pressure. Barbiturates and propofol can cause unpredictable depression of CVS and CNS. Either depolarizing or non-depolarizing muscle relaxants can be used for intubation of trachea. But considering the likelihood of difficult airway and delayed gastric emptying, it is better to use depolarizing muscle relaxants.

Maintenance of Anesthesia

Endotracheal intubation with controlled ventilation is the norm. This could be with nitrous oxide supplemented by either short acting opioids such as fentanyl, a short acting benzodiazepine such as midazolam or by ketamine. The usual practice is to avoid the use of inhalational anesthetic agents for the fear of causing excessive cardiovascular depression. The vasodilation in presence of reduced blood volume and attenuated baroreceptor mechanism can cause severe reduction in the blood pressure. Intermediate acting muscle relaxants can be given safely but one has to be cautious about dosing. Hyperventilation should be avoided as the metabolism is reduced and this can lead to significant hypocapnia. Possible delayed emergence from anesthesia should be expected and patients often require postoperative ventilatory support.

Monitoring

This should be aimed at recognition of onset of congestive heart failure and hypothermia. Monitoring includes:

- ECG monitoring
- Continual blood pressure monitoring either invasively or noninvasively
- Temperature
- Pulse oximetry, capnometry
- Measurement of cardiac filling pressures
- Neuromuscular monitoring.

Should intraoperative hypotension occur, it is best treated with ephedrine, epinephrine or dopamine rather than using pure a adrenergic agonists. Unresponsive hypotension may require additional steroid administration. Phosphodiesterase inhibitor milrinone may be effective in the treatment of reduced myocardial contractility since its mechanism of action does not depend upon β-receptors whose number and sensitivity may be reduced in hypothyroidism.

Complications

- Difficult ventilation and intubation
- Cardiovascular decompensation—hypotension
- Prolonged recovery from anesthesia
- Hypothermia
- Water and electrolyte disturbance.

Myxedema Coma

It is a severe form of hypothyroidism with a mortality rate of around 50 percent and results because of defective hypothalamic function causing defective thermoregulation. It is characterized by delirium or unconsciousness, hypoventilation, hypothermia, bradycardia, hypotension, severe hyponatremia and paralytic ileus.

Treatment of Myxedema Coma

- Requires urgent treatment with IV thyroxin. Intravenous T4 in a loading dose of 300 to 500 µgm followed by a maintenance dose of 50 to 200 µgm/day is given. Alternately IV T3 is given in the loading dose of 25 to 50 µgm followed by a maintenance infusion. T3 is preferred as it has rapid onset of action. A combination of T3 and T4 also can be given.
- If parenteral treatment is not available, oral T3 can be started in the dose of 20 µgm thrice a day. If not, then oral T4 in a loading dose of 300 ugms followed by 100 µgm daily can be started.
- Mechanical ventilatory support.
- Glucocorticoid therapy: Hydrocortisone 100 mg three times a day.
- Glucose containing saline solutions.
- Correction of electrolyte imbalance.
- Temperature regulation.

Management of a hypothyroid patient in the perioperative period requires a lot of patience. It is always advisable to ventilate the patient in the postoperative period and support his rest of the systems, till patient shows signs of recovery.

PATIENT WITH DIABETIC MELLITUS

Diabetes mellitus is a disease characterized by chronic hyperglycemia with disturbance of carbohydrate, protein and fat metabolism due to relative or absolute deficiency of insulin or due to insulin resistance. It is characterized by wide spread microangiopathy resulting in ischemic heart disease, cerebrovascular disease, peripheral vascular disease, renal disease, retinopathy and peripheral neuropathy.[10]

According to WHO guidelines, when random plasma glucose is >11.1 mmol/L or 200 mg/dl or fasting blood sugar > 7 mmol/L or 126 mg/ dl on two separate occasions, the diagnosis of diabetes is confirmed.[11,12] More and more patients are diagnosed to have diabetes possibly because of the redefining diagnostic criteria as blood sugar of 126 mg percent or more. Therefore, more and more diabetic patients are surely going to require surgery. Many of them present for emergency surgery. Under this condition, the sugar levels are not always under control and this worsens the morbidity.

Diabetes affects the perioperative outcome mainly because of its end-organ effects rather than by itself. Though it causes accelerated aging (they have much higher physiological age), its effect mainly depends on the end organ damage that has occurred. The most important of these are cardiovascular insufficiency, renal insufficiency and collagen tissue abnormalities such as limited neck extension and neuropathies.[13]

Diabetes is roughly divided into two broad categories:

1. **Type 1:** This is due to absolute deficiency of insulin. It is immune mediated. Occurs at young age of about 20 years. It causes lipolysis, proteolysis and ketogenesis. They are dependent on insulin for their treatment and there is tendency to develop diabetic ketoacidosis.
2. **Type 2:** This is due to relative deficiency of insulin or because of peripheral insulin resistance. It develops after the 3rd decade of life and usually responds to oral hypoglycemic agents and exercise. They do not develop diabetic ketoacidosis, but are prone to develop hyperglycemic, hyperosmolar, nonketotic coma.[12] These are essentially two different diseases which share the end-organ abnormalities.

In addition, there are other types described:

- Diabetes associated with diseases of the exocrine pancreas
- Drug induced
- As a part of metabolic syndrome or syndrome X (a syndrome comprising impaired glucose tolerance or

diabetes, insulin resistance, hypertension, raised serum triglycerides, central obesity and microalbuminuria)[11] and gestational diabetes.

The clinical picture and effect of diabetes on various organ systems is enumerated in Table 2.3.

Patients with diabetes are more often subjected to surgery than the nondiabetic ones. In addition to the routine surgeries, very commonly they require surgery for debridement of ulcer on foot, commonly known as 'diabetic foot'. They also require incision and drainage of the abscesses, carbuncles excision, fournier's gangrene, necrotizing fasciitis, necrosectomy of the pancreas, emergency myocardial revascularization, etc. Such procedures are mainly carried out during emergency and have specific problems related to them such as:

- The timing of surgery can not usually be predecided. These patients are on oral hypoglycemic agents and they are kept starving for the surgery. It is a common practice to withhold oral hypoglycemic drugs (OHA). Starvation can produce ketosis.
- Stress of surgery itself results in release of counter-regulatory hormones such as epinephrine, norepinephrine, glucagon, cortisol and growth hormone. These stress hormones have anti-insulin action and counter the anabolic effect of insulin. This causes marked hyperglycemia. This in turn produces endothelial injury, impaired wound healing, development of cerebral ischemia and sepsis.[14] Hyperglycemia can also lead to hyperglycemic, hyperosmolar, nonketotic coma.

Table 2.3: Effect of diabetes on various organ systems

Polyuria, polydipsia and polyphagia	**Diabetic neuropathy**
Skeletal muscle wasting	Distal symmetrical neuropathy
Impaired wound healing	Sensory and motor involvement
Increased susceptibility to infection	Burning, tingling, numbness
	Autonomic neuropathy
Diabetic nephropathy	**Cardiac complications**
Osmotic diuresis	Accelerated atherosclerosis
Glomerular damage	Ischemic heart disease (silent)
Microalbuminuria	Hypertension
End-stage renal disease	Peripheral vascular disease
Stiff joint syndrome	**Retinopathy**
Involves joints of cervical spine, TM joint	Retinal hemorrhages, exudates Microaneurysms
Inability to extend neck	Retinal edema and detachment
Difficult laryngoscopy	
Gastrointestinal system	**Respiratory system**
Gastroparesis	Difficult airway
Reduced ability to coordinate swallowing	Impaired chemoreceptor activity Reduced hypoxic drive

- Sedation and anesthesia can mask the signs of hypoglycemia and a high degree of alert is required on part of anesthesiologist.
- Stress, sedation and general anesthesia often produce gastrointestinal instability leading to nausea, vomiting and dehydration.
- There is often osmotic diuresis with resultant dehydration and electrolyte imbalance, acidosis due to accumulation of ketone bodies and lactic acid.
- Diabetes usually has other comorbidities which need to be addressed and controlled.

Thinking of all these factors, it is better to defer even emergency surgery for few hours till control of diabetes and fluid and electrolytes is achieved. However, the problem is that, even blood sugar control is not possible unless the source of infection is removed.

Preoperative Evaluation of a Diabetic Patient for Emergency Surgery

The time for thorough assessment may not be available during emergency setting. However, certain information must be sought by the anesthesiologist.

- **Type of diabetes:** When did it originate? Is there any family history of diabetes or history of sudden cardiac death?
- Duration of diabetes.
- What treatment the patient is on? Is he controlled on oral hypoglycemic agents or is there a need to administer insulin? When did the patient take his antidiabetic medication last? Did he take food after that?
- How well controlled his diabetes is? Is blood sugar level monitored regularly? If possible, previous sugar reports should be checked.
- History is asked about the possible effects on the end organs and their functioning, e.g. history of hypertension, coronary heart disease, stroke, renal impairment, visual disturbances, etc.
- Any surgery in past related to diabetes such as amputation, debridement, etc. and the anesthesia for the same.

Physical Examination

- **General condition:** Whether obese, any skeletal muscle wasting.
- **Airway examination:** Neck extension, mouth opening.
- Prayer sign to know the joint involvement.
- Pulse, blood pressure, respiratory parameters.
- It is very important to rule out any autonomic neuropathy (Table 2.4).

Table 2.4: Diabetic autonomic neuropathy

Resting tachycardia
Silent myocardial ischemia
Orthostatic hypotension
Absence of beat-to-beat variation in the heart rate
Gastroparesis
Constipation and dysphagia, vomiting, diarrhea
Erectile dysfunction, impotence
Bladder atony
Hypoglycemia unawareness (asymptomatic hypoglycemia)
Cardiac dysrhythmias

- Rule out or document any peripheral neuropathy especially if regional anesthesia is contemplated.
- Quick but thorough systemic examination for cardiovascular, respiratory, gastrointestinal and central nervous system.
- Retinal examination if feasible.

Investigations

- Hemoglobin.
- **Complete blood count:** Increase in WBC indicates presence of infection.
- **Urine examination:** Presence of sugar, proteins and ketones. Glycosuria does not give proper idea about the level of glucose control as it depends on the renal threshold for glucose. However, the urine should be examined for pus cells (urine infection) and micro-albuminemia. Presence of microalbuminuria indicates diabetic nephropathy.
- **Blood sugar level:** A fasting sugar level (or random sugar) to know the blood sugar control. During perioperative period, a lot of fluctuations occur in the level of blood glucose as a result of starvation, vomiting, intestinal obstruction, stress hormones. Therefore, it is important that blood sugar be monitored at frequent intervals of may be ½ to 1 hour.
- To know the long-term control of diabetes, it is better to do glycosylated Hb. It gives an idea about glucose control over last 8 to 12 weeks. A level of less than 7 percent implies good control.
- Blood urea nitrogen and serum creatinine: To know renal involvement.
- Serum electrolytes: Sodium, potassium and magnesium.
- X-ray chest.
- ECG.
- **Echocardiography:** For LV function as the diabetic patients often have coronary artery disease.
- **Arterial blood gases:** To know if the patient is in acidotic state.

The aims of perioperative management in a diabetic patient:

- Avoidance of hypoglycemic events.
- Avoidance excessive hyperglycemia.
- Maintain fluid and electrolyte balance. Avoid loss of potassium and magnesium.
- Provide enough calories to prevent protein and fat breakdown and ketosis.
- Take care of other systems, mainly cardiovascular and renal system.

Preparing Patient for Surgery

In emergency cases, every effort must be made to optimize the condition of the patient in the available time.[15] It is important to:

- **Avoid and treat dehydration**: Hyperglycemia is often associated with dehydration due to osmotic diuresis. It should be corrected prior to induction of anesthesia by infusing 0.9 percent sodium chloride 20 ml/kg of this solution is infused to see the response. Many anesthesiologists avoid using lactated Ringer's solution as it forms glucose during metabolism.[14] However, according to another school of thought, Ringer's lactate can be safely administered to the patient.[12]
- **Correct electrolyte imbalance:** These patients often have a tendency to lose potassium. Plasma K^+ must be maintained in the range 4.0 to 4.5 mEq/L.
- **Correct hyperglycemia:** The management of peri-operative blood glucose level is a matter of debate. Conventional thinking was to maintain blood glucose levels to < 10 mmol/L (180 mg/dl). There is always a tendency to adopt the policy of 'permissive hyperglycemia'. Recently there are many proponents for 'tight control' therapy. According to this, the blood sugar level is strictly maintained between 4.4 to 6.1 mmol/L (80 to 110 mg /dl). This is a very intensive therapy and requires meticulous monitoring of blood glucose as there is always a risk of development of hypoglycemia. Hypoglycemia often undergoes unnoticed under anesthesia and it is recommended to keep blood sugar between 110 to 150 gm/dl. Insulin can be given as a separate solution from glucose, so that its rate can be changed independent of rate of glucose infusion. It is given at a rate of insulin (units) = blood sugar (mg)/150.
- **Avoid hypoglycemia:** If patient is starving for the surgery, withhold all his antidiabetic medication. If required blood glucose can always be controlled by

insulin infusion. Though in diabetes, there is hyperglycemia, the cells are unable to utilize glucose. This is facilitated by continuously providing a substrate in the form of glucose along with insulin. This is given as 'piggy backing' solution containing 500 ml of 5% glucose with 8 units of insulin and 20 mEq/L of potassium. This is slowly infused at the rate of 100 to 125 ml/hour. Blood glucose levels are repeated every hourly till patient stabilizes. This prevents lipolysis and proteolysis by providing continuous supply of glucose to the cells.

- **Treat acidosis:** The most feared part of diabetes is the development of diabetic ketoacidosis (DK) and development of hyperosmolar coma. DK should be suspected if patient has nausea, vomiting, abdominal pain and tenderness, intense thirst, signs of dehydration, tachypnea and dyspnea, lethargy and finally coma. The urine will test positive for ketones. The treatment of this condition consists of:
  1. **Correction of dehydration:** The total deficit may be 100 mg/kg is corrected over 24 hours. In the first hour, around 15 to 20 ml/kg 0.9 percent sodium chloride is infused, guided by the central venous pressure and the urine output. Ringer's lactate is avoided. Serum electrolyte estimation is done from time to time. Subsequently 0.45 percent sodium chloride also may be used.
  2. **Correction of hyperglycemia:** Separate insulin infusion, preferably in an infusion pump is started at the rate of 0.1 unit/kg/hr monitoring the blood sugar frequently. At this rate, the blood sugar level is expected to reduce by 50 to 75 mg/dl/hr. Once the blood glucose level drops to less than 300 mg percent, dextrose saline is added to prevent precipitous hypoglycemia. There is no role of subcutaneous insulin in this setting.
  3. **Correction of potassium level:** The usual practice is to start glucose-insulin-potassium infusion in these patients. If the K^+ is low (< 3.3 mEq/L), insulin is withheld. The disadvantage of this technique is that the rate of insulin and glucose adjustment cannot be done independently.
  4. **Correction of acidosis:** Use of sodium bicarbonate ($NaHCO_3$) is not routinely recommended unless the pH is <7.0. While administering $NaHCO_3$, serum K^+ should be monitored closely to prevent dysrhythmias. If K^+ is < 3.3 mEq/L then $NaHCO_3$ should not be added till hypokalemia is corrected.

Choice of Anesthetic Technique

The choice of anesthesia can affect the outcome by modulating the secretion of the catabolic hormones and by interfering with the early resumption of enteral nutrition. If the surgery permits, regional anesthesia is the preferred technique. It prevents stress response to surgery, patient is awake throughout the surgery and any symptoms of hypoglycemia can be immediately detected. It improves peripheral circulation therefore there are decreased chances of thromboembolism. The blood loss also is reduced. Another advantage of this technique is, by providing effective analgesia, it prevents increase in catabolism in the postoperative period by reducing the level of the stress hormones.

While selecting central neuraxial block as the technique of anesthesia, it is important to rule out autonomic neuropathy. Its presence should warn the anesthesiologist about the likelihood of cardiovascular instability upon institution of the block, especially the high level required for abdominal surgery. It is also very important to rule out peripheral neuropathy. There may be pre-existent neuropathy which needs to be demonstrated. There is a possibility of exaggeration of neuropathy postanesthesia. One is also apprehensive about the possibility of infection. Table 2.5 enumerates the anesthetic implication of autonomic neuropathy.

General Anesthesia

Diabetes *per se* would not dictate administration of a particular class of anesthetic agent. It is the end organ damage that decides the choice of anesthesia and the anesthetic. Presence of nephropathy, ischemic heart disease, hypertension and autonomic neuropathy should prompt the anesthesiologist to choose the drugs carefully. Prior aspiration prophylaxis is mandatory.

Midazolam and fentanyl appear to be safe. They reduce the requirement of the anesthetic agents. Carefully titrated doses of thiopentone or propofol or etomidate are used for induction. Ketamine is avoided

Table 2.5: Anesthetic implications of autonomic neuropathy

• Gastroparesis: Increased chances of aspiration of gastric contents
• Pressor response to laryngoscopy and intubation
• Asymptomatic hypoglycemia: Meticulous monitoring required
• Blunted tachycardic response to hypotension or any sympathetic response
• Profound hypotension under regional anesthesia
• More susceptible to the depressant effect of the anesthetics
• Possibility of sudden cardiac arrest

as it causes hyperglycemic response and sympathetic stimulation. These patients are considered as potentially full stomach and therefore mandate rapid sequence induction and intubation. The possibility of difficult intubation must be borne in mind, especially if documented by Prayer's sign and restricted neck extension. In such cases, use of difficult airway gadgets and in extreme conditions, awake fiberoptic intubation would seem prudent. If there is extensive peripheral neuropathy causing skeletal muscle wasting, then succinylcholine is best avoided. Injection rocuronium may be used instead.

Anesthetic agents also affect the glucose homeostasis in the perioperative period. Benzodiazepines and etomidate reduce the synthesis of cortisol whereas high dose opiate anesthesia produces hemodynamic, hormonal and metabolic stability. Halothane, enflurane and isoflurane cause dose dependent and reversible inhibition of insulin secretion in response to glucose load.
The perfect anesthetic plan would be:

- Aspiration prophylaxis.
- Premedication with midazolam and fentanyl.
- Induction with thiopentone, etomidate or propofol.
- Maintenance with vecuronium or atracurium depending on the renal profile.
- Analgesia by opioids or epidural local anesthetics. NSAIDS are avoided if renal impairment is suspected or present.
- Reversal and extubation as per the condition of patient.
- Monitoring at frequent intervals for blood sugar and ketonemia (blood hydroxybutyric acid) if feasible.
- Intravenous fluids to correct deficit and as maintenance fluid, preferably with a separate glucose insulin potassium drip.
- Reversal and extubation if patient is hemodynamically and metabolically stable and extubation criteria are satisfied.

PATIENT WITH RENAL DISEASE

Introduction

Kidney is generally associated with filtration function, but also responsible for other functions such as regulation of water balance, acids, bases, and electrolytes, erythropoietin hormone release like, regulation of blood count in circulation and clearance of waste.[16]

Anesthesiologists often care for patients with renal insufficiency or renal failure. These patients may present to the operating room for a minor procedure such as an inguinal hernia repair or an arteriovenous fistula/graft. Alternatively, they may present for major abdominal operations or coronary artery bypass grafting. Critically ill patients presenting to the operating room may have acute kidney injury. It is imperative that the anesthesiologist understands the ramifications of renal failure and adjusts the anesthetic plan accordingly.

Most drugs commonly employed during anesthesia are at least partly dependent on renal excretion for elimination. In the presence of renal impairment, dosage modifications may be required to prevent accumulation of these drugs or their active metabolites in the body. Moreover, the systemic effects of azotemia can potentiate the pharmacological actions of many drugs.[17]

Chronic Renal Failure (CRF)

CRF refers to a decline in the glomerular filtration rate (GFR) caused by a variety of diseases, such as diabetes, glomerulonephritis, and polycystic kidney disease. Patients with CRF have a high prevalence of hypertension. They have the traditional risk factors for coronary artery disease (CAD) such as advanced age, diabetes, hypertension, and lipid disorders, as well as a high prevalence of nontraditional risk factors, such as hyperhomocysteinemia, abnormal calcium phosphate metabolism, anemia, increased oxidative stress, and perhaps, uremic toxins.[17]

CRF can be associated with excess surgical morbidity, the most important of which include acute renal failure, hyperkalemia, volume overload, and infections. A study of more than 2000 patients with ESRD having operative procedures (majority for vascular access) found high degree of perioperative morbidity and mortality. Most common risk factors were arrhythmia, sepsis, emergency procedure, diabetes, advanced age and longer stay in intensive care unit.[18]

Acute Renal Failure (ARF)

Acute renal failure is a common and in many cases it is a preventable and/or eminently treatable problem seen in the operation theaters and intensive care units and the physician treating the critically ill patient should be well versed in the diagnosis and management of renal failure.[19]

ARF is in fact, responsible for at least one-fifth of all perioperative deaths among elderly surgical patients. The precise mechanism heralding the transition from compensated preserved renal function to uncompensated renal failure during the perioperative period remain poorly understood, in part because the methods

used to assess renal function are insensitive and non-specific.

Perioperative renal failure accounts for one-half of all patients requiring acute dialysis. Acute tubular necrosis accounts for nearly 90 percent of the cases of perioperative renal failure. Perhaps one reason for our inability to prevent renal failure is a shift in medical populations to older and more critically ill patients.[20]

The human response to stress results in a shift of renal blood flow away from the cortical regions and towards the juxtamedullary nephrons, which conserve water (the medulla is essential for concentrating urine). In extremely low blood flow states, reduced RBF decreases filtration, which lowers urine output further and can damage the kidneys permanently.

In a large clinical study, intra-abdominal pressure (IAP) has been shown to be an independent cause of renal impairment, and it ranks in importance after hypotension, sepsis, and age older than 60 years. Surgeons need to be aware of the importance of abdominal hypertension in postoperative period. Consideration to adopting a more routine approach to IAP measurement, every 8 hours in patients in the ICU, particularly those undergoing emergency surgery, should be undertaken. Perhaps IAP measurement should be undertaken in the operating room, at the end of abdominal closure, or in the recovery room.[21]

Cardiac surgery, aortic surgery, liver transplant, and emergency surgical procedures are thought by some to place the kidneys at risk. Trauma patients exhibit two types of renal failure, an early, oliguric form that carries a 90 percent mortality rate, and a late, nonoliguric form associated with multiorgan failure (MOF), toxins, or sepsis and which carries a 20 to 30 percent mortality rate. ARF occurs in 10 to 30 percent of patients admitted to ICU. It is associated with a mortality rate of 50 percent. When multiorgan failure develops, mortality rate increases to 50 to 80 percent.[22]

Pathophysiology

Knowledge of the pathophysiologic derangements as well as external (sometimes iatrogenic) insults that can arise in the perioperative period in patients with renal failure is vital in the evaluation and management of these patients. Impairment of the excretory function of the kidney results in an elevation in blood urea nitrogen (BUN), creatinine, and various protein metabolic products. Impairment in the synthetic function results in a decrease in the production of erythropoietin (causing anemia) and active vitamin D-3 (causing hypocalcemia, secondary hyperparathyroidism, hyperphosphatemia, and renal osteodystrophy). Impairment in synthetic function also results in a reduction in acid, potassium, salt, and water excretion (causing acidosis, hyperkalemia, hypertension, and edema) and in platelet dysfunction (leading to an increase in bleeding tendencies).

Principle of Anesthesia Management

One of the greatest challenges for the anesthesiologist is presented by patients who have undergone renal transplant or incidental surgery with insufficient renal function and whose renal function must be preserved during perioperative period. The whole process becomes trickier when these patients are to be taken up for emergency surgery.

Preoperative Preparation

Proper preoperative management of patients with renal failure include their identification, evaluation of their intravascular volume status, optimizing of preexisting medical conditions and discontinuation of any nonessential medications associated with renal insufficiency. The protecting strategies include recent treatment modalities to maintain adequate oxygen delivery, suppress renovascular constriction, produce renal vasodilatation, maintain renal tubular flow and decrease oxygen demand.[23]

- **Significant cardiovascular risk:** Elevated frequency of cerebrovascular disease, coronary artery disease, and peripheral vascular disease; GI dysfunction (including reflux) may cause chest pain and differentiation may become difficult (patient with neurologic dysfunction may not have symptoms of chest pain even with myocardial ischemia/infarction)
- Diabetes mellitus and systemic hypertension with left ventricular hypertrophy is common finding. CRF patient may be anemic and have hyperlipidemia with tendency for accelerated atherosclerosis
- Anorexia, hiccups, nausea and vomiting (hallmarks of acute uremia) and delayed gastric emptying increases the risk of regurgitation and aspiration.
- High incidence of hepatitis B and C (anicteric, carrier state) may be present in patients on chronic hemodialysis
- One must find out when the patient was last hemodialyzed. Blood volume status may be estimated by comparing body weight before and after hemodialysis and monitoring of vital signs (orthostatic hypotension, tachycardia) and measuring arterial filling pressures

- Depressed patients may not need preanesthetic premedication. Avoid heavy premedication in severely debilitated patients.

Investigations

Relevant laboratory studies include hematocrit, complete blood count, electrolytes, arterial blood gases, blood glucose, blood urea nitrogen, creatinine, bleeding time, electrocardiogram, and chest radiograph.[24]

Anesthetic Management

Identification of etiological causes of renal failure and prevention of further deterioration by various strategies is the goal of managing these patients. Hemodynamic monitoring and fluid management can be challenging in this patient population in the acute setting of the operating room.[25]

General vs regional anesthesia: One might expect regional anesthesia to have a better renal side effect profile, but there is no difference in creatinine changes following regional anesthesia.

Principles of Anesthetic Management in Renal Failure Patient

- Perioperative goal is to begin with serum potassium level < 5.5 mEq/L; consider arterial line despite challenge with vascular access and attentiveness to ventilatory management is important.
- Anemia and arteriovenous shunts cause a hyperdynamic circulation with fixed low systemic vascular resistance, and impaired circulatory reserve with poor tolerance of myocardial ischemia or sepsis. Anesthetic technique must minimize changes in renal blood flow (Renal blood flow: 25 percent of normal cardiac output).
- Most patients with CRF have chronic acidosis, surgical disease can further complicate the acidemia, e.g. sepsis, trauma and prolonged hypotension, etc. Such patients are at a higher risk for hyperkalemia, myocardial depression, and cardiac arrhythmia.
- All anesthetic techniques and agents tend to decrease glomerular filtration rate (GFR) and renal blood flow. They usually resolve rapidly with emergence of anesthesia.
- Toxicology of immunosuppressants and relevant drug interactions should be considered.
- Avoidance and curtailment of nephrotoxic insult by drugs and changes in hemodynamics is important for better outcome.
- Strict aseptic technique should be followed for all the invasive procedures and a broad-spectrum antibiotic coverage throughout the perioperative period. Pneumonia and sepsis are the most common serious infections.
- Hyperkalemia may be precipitated by tissue breakdown, transfusions, acidosis, ACE inhibitors, β-blockers, heparin, rhabdomyolysis, and the use of Ringer lactate solution as a replacement fluid. Hyperkalemia should be promptly treated.
- Third-space fluid loss, diarrhea, vomiting and nasogastric suction result in both volume contraction and hypokalemia. Hypokalemia is sometimes followed concomitantly with hypomagnesemia.
- Hypocalcemia and hyperphosphatemia may be caused by rhabdomyolysis. Hyponatremia may occur from hypotonic fluids or inappropriate secretion of antidiuretic hormone.
- Bleeding time may be prolonged due to uremic toxin. This can be treated with dialysis.
- Serial measurement of arterial blood gases and electrolyte should be done and correction of abnormalities should be done on a real time basis.
- Prophylaxis against acid aspiration should be taken, since there is delay in gastric emptying in renal failure or emergency patient may not be nil by mouth.
- Anticipate labile blood pressure: Hypotension (deep anesthesia, fluid losses, change of position) or hypertension (inadequate anesthesia). β-blockers or calcium channel blockers are helpful. α-agonist to control hypertension also can be used safely.
- Anticipate hyperkalemia (in patient on β-blockers), arrhythmias, and potential for digoxin toxicity.
- Avoid drugs that are totally dependent on renal clearance; loading dose unaltered, but maintenance dose should be drastically reduced; frequently used drugs include antibiotics (e.g. penicillin, cephalosporin), older muscle relaxants, and digoxin.[26]
- Drugs partially dependent on renal clearance: loading dose unaltered, but maintenance dose should be decreased significantly; includes anticholinergics (e.g. atropine, glycopyrrolate), reversal agents (e.g. neostigmine, edrophonium), muscle relaxants (e.g. vecuronium, pancuronium), cardiovascular drugs (e.g. milrinone), and some barbiturates. The doses of benzodiazepines and thiopentone may need to be reduced by 30 to 50 percent.[26]
- Propofol is hepatically metabolized into an inactive compound excreted by the kidneys. Although the pharmacodynamics of propofol are unchanged in CRF and the metabolites lack sedative activity, changes in volume of distribution and mental state

mean that a reduction in induction dose may also be appropriate.[26]

- Titration of effect and decrease in the dose of drugs with unbound fraction (e.g. thiopental, diazepam); active or toxic metabolites are common.[27]
- Nephrotoxicity is a theoretical possibility with enflurane (fluoride) or sevoflurane (Compound A) anesthetic agents.
- Opioids should be used cautiously in this patient population due to possible accumulation of the parent drug and/or its metabolites. Usual or adjusted doses may be appropriate for certain opioids (e.g. morphine, hydromorphone, hydrocodone). Oxycodone should not be used in dialysis patients, and others should be avoided at all times (e.g. codeine, meperidine, and propoxyphene).[27]
- The duration of action of a bolus dose of 0.6 mg/kg rocuronium under desflurane anesthesia was increased significantly in patients with end-stage renal failure compared to that of healthy controls and was prolonged according to the duration of renal failure.[28]
- Sugamadex is useful for reversal of vecuronium and rocuronium.[29]
- Drugs such as triamterene, spironolactone, NSAIDS, β-blockers, ACE inhibitors are to be avoided.
- Fluid and blood loss should be managed on real-time basis thereby preventing the overloading and pulmonary edema.
- The ventilation should be controlled as low colloid oncotic pressure promotes interstitial and pulmonary edema. Functional residual capacity and ventilatory reserve are decreased. (Hypoalbuminemia: dietary protein restriction, albuminuria and/or losses via continuous peritoneal dialysis—10 to 40 g/day protein).
- Regional anesthesia is not contraindicated if coagulopathy is absent or corrected, but there is increased risk of hypotension (autonomic neuropathy) and infection. Sudden increase in systemic vascular resistance, once the sympathetic block wears off can precipitate pulmonary edema.
- Monitoring: Avoid BP cuffs or arterial catheters on arm with arteriovenous fistula. Invasive hemodynamic monitoring is indicated if large fluid shifts are anticipated, or patient with sepsis or cardiopulmonary insufficiency. Temperature monitoring and urine output (hourly) should be measured.
- Use of active warming devices to prevent inadvertent hypothermia.
- Intraoperative renal replacement therapy may be required (on cardiopulmonary bypass). Protective interventions are very important in renal transplant patients undergoing coronary artery bypass graft surgery to prevent deterioration of renal function. Hemofiltration was performed routinely to prevent volume overload and excessive hemodilution. Low-dose dopamine infusion (renal dose) throughout the operation and phenylephrine infusion was used during cardiopulmonary bypass.[30]

Management of Acute Oliguria during Perioperative Period

- Check for the urinary catheter that it is not blocked (clots, debris) or kinked. Decreased cardiac output secondary to insufficient preload can lead to end-organ (including renal) failure. Unchecked fluid resuscitation does not necessarily improve outcome.
- Maintaining the adequate preload with central venous pressure monitoring. Also by observing the urine output by giving fluid challenge, at least twice given half an hour apart (150 to 250 ml for 10 min per dose).
- Diuresis: While there is a natural tendency to give diuretics or medications to improve urine output following perioperative oliguria, there is no evidence that this actually protects renal function or improves outcomes (i.e. one is treating the result, not the cause). Mannitol is an osmotic diuretic and has been used in various situations to improve urine output and prevent kidney injury.
- Renal replacement therapy may be needed and should be started early.

Postoperative Care

- Anesthetic emergence may be delayed, and complicated by vomiting, aspiration, hypertension, hypothermia, persistent neuromuscular blockade, respiratory depression or pulmonary edema
- Fluid management: Restrict the maintenance fluid therapy, replace only sequestration or overt losses
- Postoperative oxygen supplementation should be given
- Postoperative pain relief should be given with judicious use of opioids
- CO_2 retention causing respiratory acidosis, chronic metabolic acidosis with hyperkalemia may need urgent attention
- Whenever in doubt, a short period of postoperative mechanical ventilation allows controlled emergence, avoid side effects of reversal agents, and facilitates evaluation of neurologic and ventilatory function before extubating the patient

- Intraoperative monitoring should be continued in the postoperative care unit
- Indications for hemodialysis are: A-E-I-O-U[17]—acidosis, electrolyte abnormalities, intoxication, overload of fluids, uremia (pericaditis, mental status changes)
- Consider hemodialysis (HD) for severe uremia, however, difficult to do especially in the hypotensive postoperative or septic patient, requiring vasopressor support. In the ICU set up BUN and creatinine clearance is assessed daily and dialysis is usually started when the BUN level exceeds 100 mg/dl or the creatinine clearance is less than 15 ml/min/L. (these figures are arbitrary and vary from center to center).[31]
- If patient is hemodynamically unstable: Consider peritoneal dialysis (PD) or continuous venovenous hemodialysis (CVVH/D).[32]

Intermittent HD is the quickest therapy which allows large fluid offloading, but many patients become hypotensive. Since critically ill patients are usually intravascularly depleted, further insult from post-HD hypotension can cause ischemic damage to many organs including the already damaged kidneys.[33,34]

To safely manage renal failure patients, we need to understand the altered physiology and presence of associated diseases, altered pharmacology of commonly used anesthetic agents, perioperative medications in CRF and ARF and the benefits and limitations of dialysis.

PATIENT WITH HYPERTENSION

Introduction

Hypertension (HTN) is a common medical disease and patient with this condition is frequently encountered by the anesthesiologists. It is a heterogeneous disorder resulting from either a specific cause (secondary hypertension) or some unknown cause (primary or essential hypertension). It is frequently associated with other comorbid conditions. Ischemic heart disease is the most common form of organ damage associated with HTN. The others being, heart failure, renal insufficiency, and cerebrovascular disease.[35]

Every attempt is made to control the blood pressure (BP) prior to elective surgery by multimodal approach. This article will focus mainly on patients with hypertension for emergency surgery.

Definition

The Sixth Report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure has classified hypertension according to the degree of BP elevation[36] (Table 2.6). It defines a hypertensive patient as anyone with a systolic pressure of 140 mm Hg or higher, anyone with a diastolic pressure of 90 mm Hg or higher, or anyone taking antihypertensive medication. In the recent classification published by American Society of Hypertension, the stages of normal and high normal are merged and termed as prehypertension and moderate and severe hypertension are merged and termed as Stage II hypertension.

Hypertensive crisis is defined as having a diastolic BP greater than 120 mm Hg.The term 'Hypertensive urgencies' has been used for patients with severely elevated BP without acute end-organ damage. Hypertensive emergency is further characterized by end organ damage.

Etiology of Hypertension

Includes essential hypertension, renal, vascular, endocrine, neurogenic, pregnancy related, pharmacologic, central nervous system trauma, autoimmune disorders.[37] Drugs like oral contraceptives, glucocorticoids and mineralocorticoids, sympathomimetics and nasal decongestants can cause hypertension.

The predisposing factors for hypertension are familial, advancing age, stress, obesity, sedentary lifestyle, high sodium and low potassium diet, insulin resistance, high alcohol and tobacco consumption.

Pathophysiology

Hypertension involves peripheral blood vessels and end-organ damage of heart, kidney and brain. A variety of abnormalities including heredity, fetal undernutrition, abnormal sympathetic nervous system activity, cell membrane defects, renal retention of excess salt, microcirculatory alterations, endothelial cell dysfunction, hyperinsulinemia secondary to insulin resistance, vascular hypertrophy and altered renin-angiotensin system regulation are implicated.[37]

All these factors cause arteriosclerosis, i.e. loss of elastic tissue and replacement with fibrin, athero-

Table 2.6 : Classification of hypertension

	Systolic BP in mm Hg		*Diastolic BP in mm Hg*
Normal	< 120	and	< 80
Prehypertension	120-139	Or	80-89
Stage 1 hypertension	140-159	Or	90-99
Stage 2 hypertension	> 160	Or	> 100
Isolated systolic HT	> / = 140	And	< 90

Table 2.6 : Classification of Hypertension

sclerosis and plaque deposition in the arterial wall, reduced vascular lumen and lumen to thickness ratio, vascular hyperactivity of the vessel wall and reduced intravascular volume. The organs most involved are heart, kidneys, brain and retina.

The main effects on various organs are described in Table 2.7. The disease is usually symptomless but if untreated, hypertension may result in heart enlargement and failure, renal dysfunction and cerebrovascular accidents.

Treatment

Drugs used for emergency control of hypertension shown in Table 2.8.

Classification of drugs used in the management of hypertension is shown in Table 2.9.

Anesthesia for Emergency Surgery

We may get patient for emergency surgery with following status:

- Controlled hypertensive
- Uncontrolled hypertensive
- Hypertension detected for the first time on examination.

When dealing with hypertensive patients the following facts should be remembered:[38]

It is important to look for evidence of end-organ damage or complications, as they may influence the choice of anesthetic technique. Another very important factor is the presence of left ventricular hypertrophy as this will expose the patient to the risk of myocardial ischemia.

Table 2.7: Effect of hypertension on various tissues

Heart	*Brain*
• Increased afterload	• Atherosclerosis
• Myocardial hypertrophy	• Stroke/CVA
• Increased intracavitary pressure	• Cerebral ischemia
• Increased oxygen demand	*Retina*
• Myocardial ischemia	• Retinal hemorrhages
• Cardiac dilatation and failure	• Retinal exudates
Kidneys	
• Reduced GFR	
• Tubular dysfunction	
• Proteinuria	
• Glomerulosclerosis	
• Renal insufficiency	

Table 2.8: Antihypertensive drugs, action and duration of action

Drug	*Action*	*Duration of action*
Labetalol 10–200 mg IV	Alpha and beta-blocker	1–4 hours
Propranolol 1–4mg IV	Beta-blocker	1–2 hours
Hydralazine 5–20 mg IV	Vasodilator	3–6 hours
Nifedipine 10 mg sublingual or oral	Calcium channel blocker	2–5 hours
Diazoxide 30 mg boluses IV (max 300 mg)	Vasodilator	4–12 hours

Table 2.9: Antihypertensive agents

Mechanism of action	*Examples*	*Relevance to anesthesia*
Diuretics	Hydrochlorothiazide Frusemide	May produce hypokalemia resulting in dysrhythmias
Vasodilators	Hydralazine Diazoxide	Tachycardia (unpredictable when given IV)
Central sympathetic depression	Clonidine Methyldopa Reserpine	Rebound hypertension if withdrawn. Slow acting.
Adrenergic neuron blockers	Guanethidine	Sensitive to vasopressors. Postural hypotension.
β-blockers	Propranolol Atenolol Labetalol (alpha also)	Avoid in asthmatics and patients with heart failure. Cause bradycardias which usually respond to atropine.
α-blockers	Phenoxybenzamine Phentolamine	Tachycardia.
Calcium channel blockers	Nifedipine Verapamil	Vasodilator. Cardiac depressant—avoid combining with beta blockers as extreme hypotension can occur.
Renin-angiotensin connecting inhibitors	Captopril	Potentiate hypotensive action of an esthetic drugs.

Evaluation

The patient should be asked about:[39]

- Duration of hypertension
- Rule out or establish end-organ involvement, e.g. chest pain, sudden change in vision, weakness in extremity
- Family history
- Addictions

- Etiological factor to rule out secondary hypertension
- Drugs the patient is receiving and the level of control achieved.

Investigations

- Urine analysis for proteinuria
- Renal function tests and electrolytes
- Blood glucose level
- ECG
- X-ray chest
- Echocardiogram if feasible
- Patients with diabetes and hypertension are considered as coronary equivalents. It is important to assess the degree of blood pressure control achieved as severely untreated hypertension (a diastolic pressure >120 mm Hg) in the perioperative period may lead to serious complications, such as myocardial infarction, left ventricular failure, cerebral hemorrhage, hypertensive encephalopathy, and renal failure.
- Patients with untreated or inadequately treated hypertension develop marked swings in blood pressure during anesthesia, blood loss or pain. They may undergo a profound fall in arterial pressure in response to induction and maintenance of anesthesia. They also exhibit an exaggerated hypertensive response to stimuli such as laryngoscopy and intubation. They are prone to develop cardiac dysrhythmias and ischemia during anesthesia.

Adequate control of hypertension, smooth anesthesia, selection of appropriate anesthesia and anesthetic agents and awareness about possible drug interactions is essential. However, while conducting emergency surgery, there is limited time available to achieve this control. The treatment that the patient is already on should be continued as their discontinuation would result in rebound hypertension and tachycardia. There is exception of ACE inhibitors which most anesthesiologists prefer to withhold for the fear of sudden drop in BP. They pose problems because of their mode of action, i.e. reduction in angiotensin II and aldosterone formation, blockade of the effects of renin and inhibition of bradykinin breakdown.[40] Also the interaction between the drugs and the anesthetics should be anticipated. For emergency control of blood pressure sublingual or nasal nifedipine, β-blockers like esmolol and labetolol or intravenous nitroglycerine can be used.

Premedication

The aim is to ensure circulatory stability during surgery. So efforts should be made to control the BP before induction. Treat pain and anxiety with appropriate medication. The drugs listed in Table 2.9 may be used to carefully reduce the BP to around 160/100 mm Hg. However, these drugs can cause unexpected hypotension which may result in stroke, blindness and myocardial ischemia. The drugs should be given gradually, in small increments, while continuously assessing their effect.

Patients with well-controlled hypertension should be premedicated with benzodiazepines prior to surgery as this will help to allay anxiety. However, one has to be careful as this makes the patient prone to aspiration of gastric contents as a result of decreased tone of lower esophageal sphincter. Atropine should be avoided because of its tendency to cause tachycardia. Glycopyrrolate may be used instead. Preoperative administration of clonodine or β-blocker can reduce the intraoperative hemodynamic lability and myocardial ischemia.

Adequate monitoring should be started prior to induction of anesthesia. Continuous pulse measurement and frequent arterial pressure assessment are important. An ECG is useful to detect ischemia and dysrhythmias; pulse oximeter, end-tidal CO_2 analyzer are used. For major surgery, central venous pressure monitoring and measurement of urine output may be useful.

The choice of anesthesia depends on the control of blood pressure and the level of analgesia required. Central neuraxial blockade is well tolerated if the blood pressure is under control and the surgical level is not very high, i.e. below T6. Epidural anesthesia proves better than subarachnoid block as the resultant hypotension is well compensated. Minimum possible drug, judicious use of additives, insertion of epidural catheter to carefully titrate the level and adequate preloading and co-loading make the block successful without causing catastrophic fall in BP. Local blocks, e.g. brachial plexus blocks or ankle blocks, should always be considered in hypertensive patients, as the potential hazards of general anesthesia are thereby avoided.

Induction

Care should be taken not to cause a precipitous fall in arterial pressure at induction. The use of opioids, such

as morphine or fentanyl, will reduce the hypertensive response to laryngoscopy and intubation. Thiopentone may be used provided it is given slowly, and titrated against response. Similarly, propofol also would result in drop in blood pressure, sometime more extensively than thiopentone. Good BP control usually does not cause any major swings in blood pressure. Ketamine, which raises the arterial pressure and heart rate, is best avoided.

If tracheal intubation is necessary, the hypertensive response to laryngoscopy can be reduced by the use of intravenous opioids or lignocaine 1 mg/kg intravenously and an adequate depth of anesthesia. The duration of laryngoscopy should be kept to less than 15 seconds.

Maintenance

The use of opioids, which have minimal cardiovascular effects, will reduce the amount of volatile anesthetic agents required. High concentrations of volatile anesthetic agents can cause hypotension by decreasing the systemic vascular resistance and by depressing the myocardium. Nitrous oxide can be safely used. The combination of nitrous oxide and low to moderate doses of opioids and potent inhalation anesthetic agents may provide the most stable intraoperative course.[41] Local anesthetic nerve blocks or infiltration are useful either on their own or to supplement general anesthesia. Cardiostable muscle relaxant like vecuronium bromide is preferred.

The specific monitoring should include; electrocardiogram for heart rate, rhythm and ischemic changes. If possible two leads should be monitored simultaneously. Blood pressure should be monitored either manually or noninvasively. In extremely high-risk patient for major surgery, even invasive anesthetic BP can be considered. Central venous pressure can be monitored depending on the need of surgery.

The complications to be expected during surgery are, excessive hypertension/or hypotension, dysrhythmias, myocardial ischemia and infarct, congestive heart failure, excessive bleeding and intracerebral hemorrhage or stroke. Rapid increase or decrease in the BP should be avoided.

Hypertension during anesthesia may reflect inadequate depth of anesthesia, pain or hypercarbia (raised blood carbon dioxide level due to inadequate ventilation). These factors should be corrected before treating blood pressure with antihypertensive drugs such as labetalol or nifedipine. Most of the time it is possible to control hypertension using anesthetics drugs like propofol or isoflurane. Rarely continuous infusion of nitroglycerine or nitroprusside is needed to control hypertension during anesthesia.

Hypotension should be vigorously treated by reducing the depth of anesthesia (if it is excessive), and correcting any hypovolemia. Bradycardia should be treated using intravenous atropine. Occasionally, a small dose of a vasopressor such as ephedrine or phenylephrine may be required in patients not responding. The systolic pressure of controlled hypotension should not be lower than the diastolic pressure of the patient's usual pressure. The fall of > than 25 percent of original BP is considered severe hypotension and should be treated with fluid and judicious use of vasopressors.

Normal intravenous fluid replacement should be given. During emergency surgery in untreated hypertensives, it is important to maintain careful fluid replacement. One must be cautious, that a moderately low BP in a normal patient (e.g. 90-100 mm Hg systolic) may reflect more serious hypotension in the hypertensive patient. These patients tolerate hypovolemia poorly.

Recovery

Coughing on the tracheal tube during emergence will increase arterial pressure. Opioids given during anesthesia reduces this tendency. Hypertension may develop during the recovery phase. Hypoxia or inadequate breathing may be the cause. It is logical to use antihypertensive agents instead of anesthetic agents during extubation and emergence to awaken the patient at the end of the surgery. Lower doses of lidocaine, esmolol or labetalol can be given 2 minutes before extubation. If the hypertension is due to bladder distension, a urinary catheter is indicated. If it is caused by inadequately treated pain, analgesics should be administered and the patient reassured. The patient should be returned to the ward only when the circulation is stable.

PATIENT WITH ISCHEMIC HEART DISEASE

Anesthesia for patients with preexisting cardiac disease undergoing emergency surgery is an interesting challenge. Most common cause of perioperative morbidity and mortality in cardiac patients is ischemic heart disease (IHD).[42] Surgery stresses the cardiovascular system in the perioperative period. This stress leads to an increase in cardiac output which can be achieved easily by normal patients, but which results in

substantial compromise in those with cardiac disease. Postoperative events which cause death include myocardial ischemia and infarction (MI), arrhythmias, congestive heart failure and multiple organ failure secondary to low cardiac output. If the different mechanisms involved in different cardiac disease states are understood, then the most suitable anesthetic can be given. The skill with which the anesthetic agents are selected and delivered is more important than the drugs used. Care of these patients requires identification of risk factors, preoperative evaluation and optimization, medical therapy, vigilant monitoring and the choice of appropriate anesthetic technique and drugs.

The latest guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery in patients with cardiac disease have been formulated jointly by the American College of Cardiology (ACC). Foundation and the American Heart Association (AHA), and published in the year 2007[43,44] and modified in 2009.

Pathophysiology

Ischemic heart disease is the result of the build-up in larger coronary arteries of plaques of atheroma—consisting of cholesterol and other lipids. This causes narrowing of the vessels, restricting coronary blood flow. There may be insufficient myocardial blood supply during times of high demand, e.g. exercise, leading to the effort related chest pain of stable angina. The more serious conditions of unstable angina (pain at rest) and myocardial infarction are thought to be due to rupture of the atheromatous plaques causing total obstruction as well as due to vasoconstriction of the coronary vessels. Silent ischemia also can occur in diabetic patient. There are several factors in the perioperative period which precipitate such events:

- High levels of adrenaline and other catecholamines as a consequence of surgery, causing tachycardia, coronary vasoconstriction, and increasing platelet "stickiness".
- An increased tendency for blood to coagulate, making thrombosis in coronary vessels more likely.[45]

Assessment of Perioperative Risk

Several risk indices have been developed over the past 25 years based on multivariable analysis derived from history, physical examination or ECG review. The most popular ones in the past being perhaps the Goldman's risk criteria (1977), Table 2.10 and the Detsky's modification (1986). A simpler index for predicting cardiac risk in noncardiac elective surgery was given by Lee et al (1999) wherein six independent risk correlates were identified. These include IHD, congestive heart failure (CHF), cerebrovascular disease, high-risk surgery, preoperative insulin therapy, and serum creatinine > 2 mg percent. This revised cardiac risk index has become one of the most widely used indices presently.

Table 2.10: Goldman's risk index

No.	*Risk variables*	*Points*
1.	S3 gallop or raised jugular venous pessure	11
2.	Recent Myocardial infarction	10
3.	Non-sinus rhythm, paroxysmal atrial contractions (PAC)	7
4.	More than 5 ventricular premature beats (VPC)	7
5.	Age > 70 years	5
6.	Emergency surgery	4
7.	Poor general medical condition	3
8.	Intrathoracic, intraperitoneal surgery	3
9.	Aortic stenosis	3

ACC/AHA Guidelines

The guidelines are elaborate regarding preoperative risk assessment of these patients. The document considers effect of type of surgery, functional capacity and the clinical presentation of the patient for preoperative risk assessment and further workup accordingly. The aim is to identify the patients at risk from history, physical examination and simple noninvasive tests.

Since elaborate work-up is not possible during emergency, the stress is on knowing the severity of the symptoms, the functional capacity and the invasiveness of surgery. Depending on the results of evaluation, the risk stratification of the patient is done and interventions if any are prescribed.

History

The patient should be asked about history of symptoms such as

- Angina: Its nature, severity, the type and degree of activity that precipitates angina
- Dyspnea: Whether angina occurs at rest or not
- Palpitations
- Orthostatic intolerance.

In addition, the patient should also be asked about:

- Current medications
- Prior coronary evaluation and intervention if any
- Insertion of pacemaker
- Risk factors for coronary artery disease (Table 2.11)
- Other comorbid conditions.

Table 2.11: Risk factors for coronary artery disease

Advancing age	Smoking
Male sex and postmenopausal females	Dyslipidemia
Type A personality	Hypertension
Family history of CAD	Diabetes
Stress	Obesity

The points to be noted preoperatively are:

- The amount of myocardium at risk.
- The ischemic threshold.
- Patient's ventricular function.
- The stability of symptoms.
- Current medications and modifications required if any. Ask about coronary interventions in the past and in case of PCI, whether the patient is on any anticoagulants or platelet inhibitors.

These patients are likely to be on:

- β-blockers to favorably influence the myocardial oxygen balance
- ACE inhibitors to control blood pressure and to remodel left ventricle
- Anti-platelet agents to prevent thrombosis especially after PTCA
- Statins: To improve plaque stability
- Nitrates: Coronary vasodilators.

The physical examination should involve

- Pulse: Rate, rhythm, S3 gallop, bruit over carotids
- Blood pressure
- Basal rales, edema feet, jugular venous pressure, tender hepatomegaly
- Evidence of cardiomegaly and presence of murmurs.

The **investigations** during emergency can not be elaborate, however, following investigations can be performed rapidly and give an idea about the risk to the patient.

- Hemoglobin, CBC: Anemia is well-tolerated in the general population, but can cause a critical reduction in myocardial oxygen supply in those with IHD—a hematocrit of 30 percent or more is recommended.
- BUN, serum creatinine
- Blood glucose
- Liver function tests
- Serum electrolytes
- Arterial blood gases
- Coagulation profile
- X-ray chest: May reveal cardiomegaly, pulmonary congestion
- ECG: Rate, rhythm, axis, ischemic changes, chamber hypertrophy
- 2-D echocardiogram: It gives idea about ventricular function (ejection fraction), regional wall motion abnormalities, pulmonary hypertension, chamber hypertrophy and co-existing valvular heart diseases. Indications for preoperative noninvasive cardiac testing have been limited to the group in whom coronary revascularization may be beneficial, independent of noncardiac surgery. The aim of preoperative noninvasive cardiac testing is to provide objective measure of functional capacity, identify presence of preoperative myocardial ischemia or dysrhythmia, and to estimate pre-operative cardiac risk and long-term prognosis.

Stress test, thallium scintigraphy or angiography are not possible but information obtained from the above noninvasive and biochemical investigations is sufficient to manage the patient.

After the history, physical examination and investigations the clinical predictor of increased peri-operative risk is decided. It can be classified as shown in Table 2.12.

After establishing or ruling out clinical predictors of increased cardiac risk, the *next step is to determine the functional capacity* of the patient depending on the level of activity that the patient is capable of performing. It is defined in terms of 'Metabolic Equivalent'. One MET (metabolic equivalent) is defined as the resting or basal oxygen consumption of a 70 kg, 40-year-old male. All physical activities can be expressed as multiples of MET. The level of functional capacity can be established by the various activities the patient is able to perform, e.g. taking care of himself/herself, using toilet or dressing,

Table 2.12: Clinical predictors of increased perioperative cardiac risk

Major	*Intermediate*	*Minor*
Unstable/severe angina (class III or IV)	Angina (class I or II)	Advanced age
Decompensated congestive heart failure (CHF)	Previous myocardial infarction (MI)	Abnormal ECG
Significant arrhythmias such as high AV block, symptomatic VPCs, supraventricular tachycardia (SVT) with uncontrolled rate	Controlled or previous CHF History of cerebrovascular accidents (CVA)	Rhythm other than sinus
Severe valvular disease	Insulin dependent diabetes Renal insufficiency	Low functional capacity Previous CVA Uncontrolled hypertension

walk two flights of stairs, walking on level round or uphill, participating in strenuous sports, etc.

It is classified as excellent (>10 METs), good (7-10 METs), moderate (4-7 METs), and poor (< 4 METs). The general practice is to classify the patient as having functional capacity of less than or greater than 4 METs.

The next step is to decide the invasiveness of the surgical procedure:

Surgical procedures have been classified as low-risk (cardiac risk <1 percent), intermediate-risk (cardiac risk 1-5 percent), and vascular surgery (cardiac risk > 5 percent). The procedures included in each group are mentioned in Table 2.13.

The traditional division of MI into 3 and 6 months interval has not been done in the guidelines. Rather, acute and recent MI (i.e. ≤ 7 days, and > 7 days but < 1 month before examination) both been taken in as "active cardiac condition". However, presence of Q waves on ECG or a previous history of MI (prior to 1 month) is listed only as "clinical risk factor".[46]

As the emergency surgery cannot be deferred, the **risk reduction strategies** are adopted. These include:

- Perioperative β-blockade.
- Optimization of medical management.
- Limiting the extent of the planned procedure.
- Staged procedure.
- Maintaining normothermia.
- Treating anemia.
- Meticulous intraoperative monitoring and maintaining hemodynamic stability.
- Good postoperative analgesia.

If the noncardiac surgery is urgent or emergent, then cardiac risks, the risk of bleeding, and the long-term benefit of coronary revascularization must be weighed, and if coronary revascularization is absolutely necessary, coronary artery bypass grafting combined with the noncardiac surgery could be considered.

Table 2.13: Surgical risk stratification

High risk mortality >5%	*Intermediate <5%*	*Low risk <1%*
Emergency surgery	Carotid endarterectomy	Endoscopic procedures
Aortic or major vascular surgery	Head and neck surgery	Superficial surgery
Prolonged procedures	Intraperitoneal and thoracic surgery	Cataract surgery
Major fluid shifts	Orthopedic surgery	Breast surgery
Blood loss	Prostrate surgery	

Perioperative Medications to Decrease Cardiac Morbidity

Role of Perioperative β-Blocker Therapy

Current studies suggest that β-blockers reduce perioperative ischemia and may reduce the risk of MI and death in patients with known CAD. Patients who are already on β-blockers should receive β-blockers. The dose of β-blocker is titrated to achieve HR of 60 to 80 bpm rather than giving a fixed dose. Each 10 bpm decrease in the heart rate reduces the risk of cardiac death by 30 percent. Withdrawal of β-blockers can results in rebound increase in heart rate, MI and chest pain.

Perioperative Statin (Hydroxymethylglutaryl-Coenzyme A Reductase Inhibitors) Therapy

Patients currently taking statins and scheduled for non-cardiac surgery whether elective or emergency, should continue to receive them.

Antiplatelet Therapy in Patient with Percutaneous Intervention (PCI)

The decision to perform percutaneous coronary revascularization prior to emergency noncardiac surgery would depend upon, high-risk coronary anatomy, unstable angina, MI and hemodynamic or rhythmically unstable heart. If PCI is necessary, then urgency of noncardiac surgery and risk of bleeding in patients receiving dual antiplatelet agents should be weighed against each other.

In patients with a previous PCI with stent implantation, the management of antiplatelet therapy, in the setting of an emergency noncardiac surgery, represents a real challenge. In fact, premature discontinuation of a dual-antiplatelet therapy with aspirin and clopidogrel is associated with a risk of stent thrombosis. Several reports of drug-eluting stent (DES) thrombosis, occurring after cessation of antiplatelet therapy, have been documented, and are associated with a catastrophic case fatality rate of 20 to 45 percent. ACC/AHA guidelines recommend a combined antiplatelet treatment with aspirin and clopidogrel for a minimum of one month, for bare metal stent, and for at least one year, for drug-eluding stent (DES).

In the case of an angioplasty performed with a balloon catheter alone, it is recommended to continue the perioperative administration of aspirin, and to delay surgery for two to four weeks, in order to allow

healing of the injured vessel. The postponement of surgery by more than eight weeks could increase the risk of restenosis.

A global evaluation must consider the hemorrhagic risk, if the antiplatelet therapy is continued, vs the possible risk of a stent thrombosis, if the treatment is stopped when stents are *in situ*. It is strongly recommended to continue treatment with aspirin, at the very least during emergency surgery. The surgical risk of excessive bleeding is manageable with platelet transfusions, fresh frozen plasma and blood if required. Withdrawal of therapy may precipitate major adverse cardiac events.

Anesthetic Goals and Technique

Irrespective of the surgery and patient profile the anesthetic goals in such a patient are to maintain the balance between myocardial oxygen demand and supply so as to prevent myocardial ischemia. In case ischemia or infarction does develop, it should be detected and treated as soon as possible. The essential requirements of general anesthesia for IHD are avoiding tachycardia/arrhythmias and extremes of blood pressure, both of which adversely affect the balance between oxygen supply and demand.

Cardiologist's opinion needs to be integrated by the anesthesiologist, surgeon and postoperative caregivers in preparing an individualized perioperative management plan. All anesthetic techniques and drugs are known to have effects that should be considered in the perioperative plan. There is very little evidence to support an advantage of any one technique or agent over the other *per se*. The choice of anesthetic technique does not matter more than the maintenance of hemodynamics.

Premedication

A nervous patient may have tachycardia and require an anxiolytic premedication. Beta-blockers also reduce tachycardia, and prevent perioperative myocardial ischemia. In a similar fashion, alpha2-agonist drugs such as clonidine reduce noradrenaline release from synapses, causing both sedation and analgesia, also a reduction in intraoperative myocardial ischemia.

Induction

All intravenous anesthetic agents except ketamine have a direct depressant action on the myocardium, and may also reduce vascular tone. This causes hypotension (especially in the hypovolemic patient), often with a compensatory tachycardia, which may cause myocardial ischemia. Ketamine is best avoided as it causes tachycardia and hypertension. In general, all agents can be used safely if given slowly in small increments.

High-dose Opioid Based Anesthesia

The choice of anesthesia would depend on the left ventricular function. Patients with good ventricular function tolerate inhalational anesthesia better. Whereas patients with poor LV function require titrated doses of opioid. Use of high-dose opioid based anesthesia was previously popular due to its apparent hemodynamic stability. However, it can be associated with the need for postoperative mechanical ventilation.

Intubation

Laryngoscopy is a powerful stressor, causing hypertension and tachycardia. This can be avoided with a supplemental dose of intravenous induction agent or opioid, e.g. fentanyl, just prior to laryngoscopy. Other agents like lignocaine or esmolol can be given intravenously to achieve the same effect.

Maintenance

Volatile agents have minimal effects on cardiac output, although they do reduce myocardial contractility, especially halothane. Overall volatile agents are cardioprotective. The indicators of cardioprotection shown include decrease in troponin level, preservation of early LV function, decreased ICU stay, as well as decreased late cardiac events. The mechanism of cardioprotection with volatile agents is the postulated preconditioning and postconditioning of the heart against infarction by activating specific intracellular signal transduction pathways.

Monitoring

Online ST-segment monitoring, if done appropriately in high-risk patients is an early indicator of myocardial ischemia. ECG leads II, V5 are important. They should be monitored for any ischemic changes.

Blood pressure: Monitored invasively if surgical condition demands.

CVP, Pulse oximetry/ABG, End tidal CO_2, Foley's catheter—urine output.

There is no study to clearly demonstrate a change in outcome from routine use of pulmonary artery catheter, ST-segment monitor, transesophageal echocardiography or intravenous NTG. Accordingly, none of these therapies are given a class I recommendation in the

guidelines. Cardiac biomarkers for perioperative MI are reserved for those patients in whom severe hemodynamic perturbations occur and there is clinical or ECG signs of ventricular dysfunction.

In contrast, maintenance of normothermia is recommended (class I) for most surgeries except during periods of intentional mild hypothermia (e.g. high aortic cross-clamping).

Reversal and Recovery

Reversal of muscle relaxation with a combined anti-cholinesterase/antimuscarinic action causes tachy-cardia, and extubation in itself is a stressor. Problems in the recovery phase which can cause ischemia include; hypoxia, tachycardia, pain, hypothermia, shivering, and anemia should be treated in the immediate post-operative period. The use of supplemental oxygen in the postoperative period is most effective measures in preventing myocardial ischemia.

Regional Anesthesia

The use of regional anesthetic techniques has theoretical advantages. Epidural anesthesia reduces preload and afterload, coagulation responses, and in the case of thoracic epidurals causes coronary vasodilatation. These effects should reduce perioperative myocardial ischemia, however this is not supported by research. However, good epidural analgesia may reduce the incidence of tachycardia arising due to postoperative pain. In a patient with IHD, local anesthetic techniques such as brachial plexus block should be encouraged in order that the hemodynamic responses to general anesthesia are avoided. However, even under local anesthesia, the patient will be subject to the stresses of the surgical procedure itself, which can have marked hemodynamic effects.

Monitored Anesthesia Care

Monitored anesthesia care has been employed in patients with CAD. When combined with an adequate local anesthesia, suppress the stress response. When chosen as the anesthetic technique, it should be remembered that an adequate analgesia is mandatory.

Managing Intraoperative Complications

- Intraoperative ischemia
  - If patient is hemodynamically stable—β-blockers (IV metoprolol up to 15 mg), IV Nitroglycerine and heparin is given after consultation with surgeon.
  - If patient is hemodynamically unstable—Support with ionotropes, use of intraoperative balloon pump may be necessary. Urgent consultation with cardiologist to plan for earliest possible cardiac catheterization may be required.
- Other complications like dysrhythmias, pacemaker dysfunction should be managed accordingly.[46]

Postoperative Management

The postoperative goal in such patients is also the same, i.e. prevention and early detection and management of myocardial ischemia or infarction. Postoperatively, MI may often be painless, making its management even more difficult. Although most cardiac events occur within first 48 hours, delayed cardiac events (within first 30 days) still happen and could be the result of secondary stress.

- Pain relief at all cost. Pain costs postoperative catecholamine surges and hypercoagulability.
- Active management for hypothermia and shivering.
- Effective volume management.
- Despite all the care some incidence of MI is there and once it occurs the mortality is high (to the tune of 40 percent).
- Patients having symptomatic perioperative high ST-segment MI is treated with drugs like aspirin, β-blockers and ACE inhibitors as early as possible and after considering the risk-benefit can be subjected to balloon angioplasty.
- Continuous ECG monitoring is useful for detecting postoperative myocardial ischemia, which is often silent. Postoperative myocardial ischemia predicts adverse in-hospital and long-term cardiac events. It should be identified, evaluated, and managed aggressively, preferably in consultation with a cardiologist.

PATIENT WITH VALVULAR HEART DISEASE

Introduction

Patients with valvular heart disease undergoing noncardiac surgery need thorough preoperative evaluation. The goal of evaluation is to identify the most appropriate testing and treatment strategies to optimize care of the patient. But in emergency situations, where the anesthesiologist may not have sufficient time to make a detailed assessment has to:

- Assess the significance of the cardiac lesion for the proposed surgery.
- Plan anesthesia according to the hemodynamic status.
- Remember to administer antibiotic prophylaxis.

The anesthetic management requires an understanding of the natural history and pathophysiology of the valve disease.

Preoperative assessment in all patients with valve disease should include a recent (i.e. at least within 6 months) evaluation such as echocardiography, and a detailed assessment of symptom progression.

Pathophysiology

The ability of the left ventricle to generate the stroke volume (SV) depends on adequate filling or preload, the contractile state of the muscle, rate, rhythm and the impedance to ejection or 'afterload'.

Thus, stenotic lesions require the heart to force an adequate volume through a small orifice; regurgitant lesions require the heart to eject a large volume because part of the ejected volume returns backwards. The LV (left ventricle) is pressure overloaded in aortic stenosis and volume overloaded in aortic insufficiency and mitral regurgitation. In mitral stenosis, the LA (left atrium) has pressure overload whereas the LV is both volume-underloaded and pressure-underloaded. The RV (right ventricle) faces progressively increasing left atrial and pulmonary artery pressure.

In mitral regurgitation, LA has both pressure as well as volume overload. Compensatory mechanisms consist of chamber enlargement, myocardial hypertrophy, and variations in vascular tone and level of sympathetic activity. These mechanisms in turn, induce secondary alterations, including altered ventricular compliance, development of myocardial ischemia, chronic cardiac dysrhythmias, and progressive myocardial dysfunction. Myocardial contractility is often transiently depressed but may progress to irreversible impairment even in the absence of clinical symptoms. Conversely, the patient with aortic stenosis may complain of dyspnea, not because of impaired systolic function, but because of reduced ventricular compliance, increased left ventricular end-diastolic pressure, and increased pulmonary pressure.[48]

Anesthetic Considerations

The risk of cardiac events depends upon the severity of cardiac disease and surgery specific risk. Surgery specific risk is further related to type of surgery itself and to the degree of hemodynamic stress associated with surgery specific procedure. For example, those patients suffering from severe stenotic valvular lesions, balloon valvotomy must be considered prior to elective noncardiac surgery but for patients requiring emergency or urgent surgery, medical management such as digoxin and diuretic may have to be instituted in order to optimize the preoperative condition. Anesthetic goals include control of heart rate, maintenance of sinus rhythm (if present preoperatively), adequate intravascular volume and prevention of myocardial depression.[49]

In appropriate patients with cardiovascular disease, local infiltration, peripheral nerve blocks or plexus blocks provide satisfactory anesthesia with low-risk of side effects, allow a reduction in requirement for general anesthetic and improve postoperative analgesia. This can reduce cardiovascular depression and risk. Local anesthetic preparations which contain epinephrine may produce tachycardia and are best avoided in patients with severe cardiovascular disease. Similarly, central neuraxial blocks are administered with caution in patients with severe cardiac disease.[50] In general, epidural anesthesia is better tolerated in cardiac patient than subarachnoid block as the hypotension is not precipitous. Also, regurgitant lesions tolerate it better than the stenotic lesions. It also can be instituted only for the purpose of postoperative analgesia.

Preoperative Evaluation Includes

Detailed **history** of symptoms of reduced forward flow, increased back pressure, like angina, syncope, symptoms suggestive of congestive heart failure, embolization, cardiac medications and any previous admission to the hospital, etc. Also any rheumatic infection in past should be asked about.

Physical examination: The stress is on cardiovascular and respiratory system examination.

The important points are; pulse rate, rhythm-sinus or nonsinus, if other than sinus, ventricular rate, gallop rhythm, heart sounds, murmurs, evidence of cardiomegaly and basal crepitations.

Investigations: Biochemical and hematological investigations possible during emergency to know the liver and renal function tests, serum electrolytes especially if patient is on diuretics and digitalis should be carried out. Recent ECG and 2-D echocardiography to assess left ventricle function, valve status such as area, calcification, regurgitation, presence of pulmonary hypertension, chest radiograph to rule out cardiomegaly and features of pulmonary artery hypertension should be reviewed.

An informed consent is to be taken after consultation with cardiologist. The patient is given *infective endocarditis prophylaxis* prior to shifting to the operating room (OR) which should be repeated after 6 hours.

Monitoring includes ECG preferably lead II and V5, pulse oximetry, noninvasive blood pressure, central venous pressure (CVP), and urine output monitoring.

Pulmonary artery occlusion pressure (PAOP) may be considered in very high-risk patient. However, this is an invasive procedure and risk and benefit ratio must be carefully weighed prior to its insertion. An infusion of ionotropes and a defibrillator should be kept ready prior to induction.

Postoperatively the patient is referred to the cardiologist for further management of valvular heart disease.

In this section intraoperative anesthetic management for emergency surgery in patients with valvular lesions mainly aortic and mitral valve, is discussed in short.

Thus, check list for cardiac patients for emergency surgery includes:

- Diagnosis of the condition clinically and by echocardiographically.
- Rapid optimization using diuretics, ionotropes whenever indicated.
- Monitoring including cardioscope and filling pressures.
- Antibiotic prophylaxis.
- Cardiac drugs ready.
- Defibrillator checked and standby.
- Provision for postoperative intensive cardiac care.

Mitral Stenosis

Normal mitral valve area is 4 to 6 cm^2. Mitral stenosis is said to be present when the area becomes less than 2 cm^2. This impairs left ventricular filling and results in decreased cardiac output. Since, left atrial emptying is decreased, it causes back pressure effect and LA pressure increases. This results in left atrial enlargement and increased pulmonary artery pressures to maintain cardiac output. These patients may develop pulmonary edema and cardiac failure especially with higher heart rate. Atrial fibrillation may develop as a result of LA enlargement and stagnation of blood causes development of clot in LA. The main symptom of mitral stenosis is dyspnea. Patients having atrial fibrillation, experience dyspnea at rest and wake up at night with shortness of breath (paroxysmal nocturnal dyspnea). Asymptomatic patients usually tolerate noncardiac surgery well. Patients with poor functional capacity (less than 4 MET's) need to be considered for mitral valve dilatation prior to or simultaneously with emergency surgery. However, the incidence is very low. The anesthesiologist should avoid myocardial depressants, tachycardia (which reduces ventricular filling time), hypovolemia, hypotension and increased pulmonary vascular resistance (e.g. due to hypoxia, pain or hypercarbia). The aim is to maintain slow sinus rhythm, normal intravascular volume, normal cardiac contractility and normal systemic vascular resistance. Preoperative medication is given to decrease anxiety and associated tachycardia. Induction of anesthesia can be achieved with any available intravenous induction drug such as thiopentone sodium, propofol or etomidate. Sufficient time should be given for the drug to reach the central circulation as the circulatory time is increased in MS. Utmost care should be taken to prevent overdose of the drug. Ketamine, should be avoided because of its propensity to increase the heart rate. Tracheal intubation and muscle relaxation for the surgery is accomplished by administration of muscle relaxants that do not induce cardiovascular changes such as tachycardia and hypotension from histamine release. Nitrous oxide and narcotic anesthesia with low concentrations of a cardio-stable volatile anesthetic such as sevoflurane seems the ideal anesthetic agent.

If regional anesthesia technique is chosen, the epidural anesthesia may be safer than spinal anesthesia. Hypotension should be prevented by adequate preload under guidance of filling pressures and cautious use of vasopressors.

Patients in sinus rhythm who develop atrial fibrillation in the perioperative period should be cardioverted. Patients already in atrial fibrillation should have the rate controlled aggressively.

In the postoperative period, the risk of pulmonary edema and right heart failure continues, so cardiovascular monitoring should continue as well. Pain and hypoventilation with subsequent respiratory acidosis and hypoxemia may be responsible for increasing heart rate and pulmonary vascular resistance. Decreased pulmonary compliance and increased work of breathing may necessitate a period of mechanical ventilation, particularly after major thoracic or abdominal surgery. Relief of postoperative pain with neuraxial opioids can be very useful.[51]

Mitral Regurgitation

Whenever the left ventricle contracts some of the blood flows backwards into the left atrium. The regurgitant flow will increase with increased systemic vascular resistance and bradycardia. Most patients with chronic mitral regurgitation are well for many years without evidence of heart failure. Dyspnea and pulmonary edema are signs of severe mitral regurgitation. Forward cardiac output is best when the heart is full and reasonably fast, and the blood pressure is low-normal with peripheral vasodilatation. For induction of anesthesia, one should avoid myocardial depressants,

hypovolemia, bradycardia and increased systemic vascular resistance. Selection of a muscle relaxant should follow the same principles. Pancuronium produces a modest increase in heart rate, which can contribute to maintenance of forward left ventricular stroke volume.[51] One should aim for a normal or increased heart rate, decreased systemic vascular resistance and normal cardiac contractility and intravascular volume. Volatile anesthetics that do not cause dysrhythmias can be administered for maintenance of anesthesia.

Regional anesthesia particularly epidural block is well-tolerated.

Aortic Stenosis

Aortic stenosis (AS) is a major risk factor for peri-operative cardiac events in patients undergoing noncardiac surgery.[52] AS is usually a chronic condition with symptoms only occurring when the stenosis is severe. The main symptoms of aortic stenosis are dyspnea, angina and syncope. They occur as a result of severe myocardial hypertrophy, increased intra-cavitary pressures, and reduced forward flow. Coronary artery disease is commonly seen with aortic stenosis. The anesthesiologist must maintain sinus rhythm. Atrial contraction is vital to maintaining adequate ventricular filling. The heart rate should be on the lower side of normal. Tachycardia and bradycardia will both reduce coronary blood flow. There is a high risk of myocardial ischemia due to increased oxygen demand and wall tension in the hypertrophied left ventricle. Thirty percent of patients who have aortic stenosis with normal coronary arteries have angina. Subendocardial ischemia may exist as coronary blood supply does not increase in proportion to the muscular hypertrophy. Tachycardia is detrimental as it may produce ischemia because of increased demand in face of reduced supply. Cautious premedication of the anxious patient is indicated. Maintenance of diastolic blood pressure is crucial to maintain coronary perfusion.

The selected anesthetic technique should maintain afterload and avoid tachycardia to maintain the balance between myocardial oxygen demand and supply in the presence of a hypertrophied ventricle and reduced coronary flow. Induction agents, muscle relaxants, and potent inhalational anesthetic agents must be administered in such a way that the hemodynamic compensations are not lost. Induction of anesthesia can be accomplished with an intravenous induction drug that does not decrease systemic vascular resistance. An opioid induction may be useful if left ventricular function is compromised. Maintenance of anesthesia can be accomplished with a combination of nitrous oxide and volatile anesthetic and opioids or by opioids alone. Performing the operation using local anesthetic infiltration may be a safe method provided adequate pain relief is provided and measures taken to allay anxiety.[51]

The systemic vascular resistance should be kept normal. An increase in systemic vascular resistance will further reduce cardiac output and a reduction in systemic vascular resistance may reduce coronary blood flow. Myocardial depressants must be avoided as heart is already overworked. Similarly dysrhythmias are avoided at any cost.

Regional anesthesia can cause dangerous fall in systemic vascular resistance and heart rate. However, epidural anesthesia may be tolerated if performed slowly with careful monitoring and treatment of blood pressure and heart rate.

Aortic Regurgitation

Patients with aortic regurgitation may not have symptoms for many years. They may develop signs and symptoms of left ventricular failure. Aim is to maintain an adequate preload to assure filling of the hypertrophied, dilated LV, a high-normal heart rate to reduce the proportion of time spent in diastole, and low-normal systemic blood pressure to encourage forward rather than regurgitant flow.

Bradycardia should be prevented as this will increase the time for backwards flow and regurgitant factor. Anesthetic induction and maintenance must be designed to avoid these changes. Induction of anesthesia in the presence of aortic regurgitation can be achieved with standard intravenous induction drugs. The ideal induction drug should not decrease the heart rate or increase systemic vascular resistance. In the absence of severe left ventricular dysfunction, maintenance of anesthesia is often provided with nitrous oxide plus a volatile anesthetic agent and/or opioid. In patients with severe left ventricular dysfunction, high-dose opioid anesthesia may be preferred. Bradycardia and junctional rhythm may require prompt treatment with intravenous atropine.

While reductions in contractility are undesirable in almost all valvular disease conditions, for AR the maintenance of adequate preload and reduced afterload are most important. Therefore, one should avoid increased peripheral resistance and myocardial depressants and aim to maintain an increased heart rate, adequate intravascular volume and decreased systemic vascular resistance.

Regional anesthesia is well tolerated in patients with chronic aortic regurgitation.

Acute Aortic Regurgitation

Acute onset of AR is usually secondary to diseases such as endocarditis, aortic dissection, or valve trauma, without having time to develop compensatory LV dilatation. Acute AR is a hemodynamic 'emergency', requiring valve replacement. Maintenance of tachycardia and avoidance of myocardial depressants are anesthesiologist's goals for this condition.

PATIENT WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

The term chronic obstructive pulmonary disease indicates the progressive development of airflow limitation that is not fully reversible. It has significant extrapulmonary effects also. This entity includes:

1. Chronic bronchitis in which there is obstruction of small airways.
2. Emphysema in which there is enlargement of the airspaces and destruction of lung parenchyma. This causes loss of lung elasticity and obstruction of small airway.[53]

Many people erroneously include asthma as a part of COPD.

Chronic Bronchitis

It is defined as presence of productive cough for more than 3 months duration for more than 2 successive years. It consists of:

1. Hypersecretion of the mucus as a result of chronic irritation of the airways.
2. Inflammation of the mucosa of the airways.
3. Luminal narrowing.
4. Blockade of the airway by dried mucus.

Emphysema

It is more of a histopathological diagnosis, characterized by:

1. The destruction of the lung parenchyma.
2. Loss of elastic recoil of the lungs.
3. Airway collapse that occurs during exhalation
4. Air-trapping with formation of emphysematous bullae pressing on adjacent normal lung parenchyma.

It is very important that these patients be evaluated thoroughly and optimized prior to surgery as they not only create problems intraoperatively but also contribute to a significant degree in the development of postoperative pulmonary complications, reversing the benefit offered by the surgery.

Preoperative Evaluation and Preparation

The goals of preoperative evaluation in these patients are:

1. To diagnose the presence of a respiratory disease.
2. To identify the type of respiratory disease as parenchymal disease, airway disease, pulmonary vascular disease. One has to decide whether it is obstructive or restrictive disease.
3. Quantify the loss of function or the severity of the disease.
4. To check the reversibility of the disease.
5. To optimize the patient's pulmonary function within the time constraints.

All these factors will help reducing the post-operative pulmonary complications. The entire process of strict pulmonary regime requires around 6 to 8 weeks for optimization and this is not possible in the emergency cases. Still it is possible to improve the condition marginally within the stipulated time. As soon as the patient is posted for emergency surgery, he should be quickly evaluated as mentioned below.

History

Symptoms of respiratory disease should be asked for. Table 2.14 enumerates the history to be elicited in case of respiratory disorder.

The activity level should be defined: severe dyspnea may be a predictor of both poor ventilatory reserve and the need for postoperative mechanical ventilation.

Table 2.15 shows the physical findings in case of respiratory disease.

Table 2.14: History to be elicited for patients with respiratory diseases

Cough: dry/wet
Expectoration: Color, quantity, relation with position of the patient
Hemoptysis
Chest pain: site, type, relation with respiratory cycle
Breathlessness and wheezing: Ability to talk, effort tolerance
Occupational history: Asbestosis, industrial lung diseases
Smoking: Duration and quantity to find out pack years consumed
History of pulmonary tuberculosis in past or in family, with treatment details
History of any surgery in the past for pulmonary symptoms
Drug history: For example, amiodarone, ACE inhibitors, bleomycin, etc.
Ongoing treatment: Bronchodilators, steroids, mucolytics, etc.

Table 2.15: Physical findings in respiratory disorder

Respiratory rate, pattern	Breath sounds
Movement of the chest	Presence of ronchi, rales
Use of accessory muscles of respiration	Stridor
Obstructed purse lipped breathing	Signs of CHF
Paradoxical breathing	Signs of pulmonary hypertension
Dullness in the lung fields	
Bedside PFT's like	
Expiratory time	
Breathholding time	
Peak expiratory flow rates should be assessed	

Table 2.16: Investigations in respiratory disorder

Hb: To rule out polycythemia in case of chronic hypoxia
CBC: To rule out chronic infection, eosinophilia
X- ray chest: May show presence of consolidation, effusion, fibrosis, infiltration and emphysema
Blood biochemistry: Sugars, liver and renal function tests
ABG: To know diffusion and respiratory gas exchange
ECG: To rule out right ventricular hypertrophy and strain

Table 2.16 indicates the investigations that should be carried out in case of respiratory disease.

It is not possible to do pulmonary function tests on emergency basis. Stress should be given to assess the lung function by bedside clinical means. A patient who is breathless while talking, is not able to finish one sentence in one breath, cannot climb two flights of stairs, who uses accessory muscles of respiration while talking or doing simple work has very poor respiratory reserve. The quantification of dysfunction should be made on the basis of arterial blood gas (ABG) analysis, since it can be obtained within a short time.

In case of emergency surgery, where the time available for optimizing the patient's condition is critical, it is very important to meticulously decide the peri-operative plan of management. Preoperative preparation plays a major role.

Preoperative Preparation for Emergency Surgery:[53,54,55]

- **Withholding of irritant stimulus** such as smoking. Though it requires 6 to 8 weeks of smoking free interval for reversing all the bad effects of smoking, even a short smoke free period is useful to revert back some of the changes such as level of blood carboxy hemoglobin towards normal.
- **Ensuring adequate hydration** systemic and preferably local hydration is important to loosen the inspissated mucus. This is best achieved by giving normal saline, or distilled water nebulizations with bronchodilators added to it. With this method very small droplets of water and the medication are produced which get deposited in the airways causing better hydration and better action using smaller doses and less systemic side effects.
- **Relief of bronchial spasm with bronchodilators.** The commonly used ones are ipratropium bromide and salbutamol. Both have different mechanisms of action and hence can be used complimentary to each other. They are often given in the form of nebulization to increase efficacy and reduce the side effects. The patient's own meter dose inhaler (MDIs) should be brought to the operating theatre.[56, 57]
- **Use of corticosteroids** are known to reduce the airway edema and the reactivity of the airways. They are often given in nebulized form as MDI. This minimizes their dose and reduces the systemic side effects.[56]
- **Chest physiotherapy** to improve sputum clearance and bronchial drainage.

Other Modalities

- Reversal of uncompensated or borderline cor pulmonale with diuretics, digitalis, improved oxygenation and correction of hypoxia by preoperative low level oxygen therapy and ventilator therapy. It must be remembered that in patients with COPD the main respiratory drive is the hypoxic one. If hypoxia is corrected, there may be respiratory depression and patient may hypo-ventilate leading to hypercarbia.
- Correction of dehydration and electrolyte imbalance.
- Continuation of prophylactic cromolyn inhalation up to the time of surgery to prevent the degranulation of mast cells and the subsequent release of chemical mediators responsible for bronchoconstriction.
- Use of anticholinergic drug is controversial. They dry the secretions and suppress the upper airway vagal responses. Thus, though they decrease the airway reactivity, they also make the secretions thick forming plugs and blocking the airways. However, maintenance adequate hydration negates this.

Induction of Anesthesia

The principles of anesthetic management are threefold:

- To block airway reflexes before laryngoscopy and intubation
- To relax airway smooth muscles
- To prevent release of biochemical mediators.

Before induction of anesthesia, patient is given all the medications that he is routinely on, for his chest condition. He is asked to carry his MDI inhalers to the operation theater.

Regional anesthesia would seem to be the first choice if the surgery can be carried out under it. It prevents manipulation of airway, thereby preventing bronchospasm and increased airway secretions. One has to decide each case on its merit. A lower limb surgery such as fracture neck femur can be easily carried out under spinal or epidural anesthesia. But it may not be a good choice in patients with abdominal sepsis following ileal or duodenal perforation. A high spinal block can result in respiratory embarrassment.

If general anesthesia needs to be given, the aim would be to cause smooth induction of anesthesia without development of bronchospasm. The agents and techniques known to cause bronchospasm are:[58,59,60]

- Use of thiopentone as the anesthetic agent: It is known to induce both laryngospasm and bronchospasm in patients with reactive airway diseases.
- Stimulation in lighter planes of anesthesia: Every effort should be made to provide deep planes of anesthesia.
- Avoiding drugs that cause histamine release: Morphine, high doses of atracurium, etc.

The agent of choice for induction would be Inj. propofol or ketamine. Propofol has rapid onset, it is known to obtund the airway reflexes, does not accumulate in the body after repeated doses and good recovery profile. Therefore, it would seem to be the agent of choice, both for induction as well as maintenance. In children both propofol as well as halothane are used. Halothane has bronchodilator action and can be used judiciously for maintenance and for deepening the level of anesthesia, should bronchospasm develop intraoperatively. Alternately sevoflurane may be used for maintenance as it is nonirritant. Ketamine is preferred when the hemodynamic condition of the patient is unstable. Though ketamine has bronchodilator properties, one must remember that it also causes increased salivary and bronchial secretions. Intubation must be carried out in deeper planes of anesthesia after adequate relaxation. It is customary to spray vocal cords with lignocaine. Even if all precautions are taken, often the patient develops bronchospasm after intubation of trachea. This must be immediately treated by deepening the level of anesthesia, administration of bronchodilators, spraying of nebulized salbutamol in the airway and intravenous steroids.

Monitoring

- Pulse oximetry: To know saturation of hemoglobin. Always saturation on air and improvement after supplementing oxygen should be noted.
- Cardioscope: To monitor heart rate and rhythm.
- Blood pressure—manual/or noninvasive.
- $EtCO_2$: To know whether patient has airway obstruction and level of blood CO_2.
- CVP: COPD of long-standing can cause structural changes in the pulmonary vasculature causing increased workload on right ventricle. Any acute exacerbation in the pulmonary pressure can cause the right ventricle to fail. Therefore, monitoring CVP is essential in these patients.
- Airway pressures: It is recommended that whenever the lungs of these patients are mechanically ventilated, the airway pressures be measured. The respiratory rate should be on the lower side so as to reserve more time for expiration. This prevents auto-trapping of gases.[61] The peak airway pressures should be kept limited to prevent barotrauma especially in case of emphysema.

Muscle relaxants that cause histamine release should be avoided. Pancuronium, cisatracurium and vecuronium are preferred relaxants because the histamine release is insignificant.

In selecting a ventilatory mode, attention should be given to providing an adequately long expiratory time to avoid the build up of intrinsic or auto-PEEP. This can be facilitated by using higher inspiratory flow rates or smaller tidal volumes than usual.[61,62]

Intraoperative Bronchospasm

The causes of wheezing and increased airway pressure include:

- Kinked endotracheal tube
- Solidified secretions or blood
- Endobronchial intubation
- Persistent coughing and straining
- Pulmonary edema
- Tension pneumothorax
- Aspiration pneumonitis
- Pulmonary embolism.

Signs of Airway Obstruction

- Elevation of peak inspiratory pressure
- Prolonged expiratory phase
- Visible slowing or lack of chest fall
- Wheezing on auscultation.

Deepen the plane of anesthesia by ↑ sevoflurane, isoflurane and manual bag ventilation should

immediately be instituted to directly assess the compliance, the bag will not fill on exhalation if bronchospasm is severe. Sometimes the resistance is so severe that it may be impossible to press the bag. A thin suction catheter may be inserted in the endotracheal tube to rule out any mechanical obstruction of the tube. The chest should be auscultated to confirm wheezing and causes of bronchospasm should be ruled out. If none of the conditions exists or if bronchospasm persists after they have been corrected then treatment of the intraoperative bronchospasm should be instituted.

It includes:

- Inhaled bronchodilators, e.g. salbutamol puff.
- Intravenous bronchodilators, e.g. salbutamol, aminophylline.
- Inhalational and intravenous steroids.
- In severe cases, where bronchospasm does not resolve, subcutaneous terbutaline or adrenaline is injected.
- There should be frequent monitoring of ABGs to evaluate hypoxemia and hypercarbia. The adverse effects on the circulation can add a metabolic component to the respiratory acidosis.
- Hypercarbia, hypoxemia and acidemia promote arrhythmias and impair the response to bronchodilator therapy.

Postoperative Case[63-65]

To avoid bronchospasm triggered by coughing and bucking caused by laryngeal and pharyngeal reflexes during emergence and extubation, patients may be extubated during deeper planes of anesthesia. However, the risk of aspiration, airway obstruction and hypoventilation should be weighed against the benefits.

Those patient who cannot be extubated, will be shifted to recovery area with endotracheal tube *in situ*. However, the presence of tube in a conscious patient may itself induce bronchospasm. To increase the tolerance of the patient, the cuff may be instilled with lignocaine solution. β_2-agonists such as albuterol can be administered through MDI adapter to prevent bronchospasm. Alternatively, laryngeal mask airway (LMA) may be used to replace the endotracheal tube for control of ventilation and to avoid tracheal stimulation.

The key to minimizing postoperative pulmonary complications are—vigilance for managing bronchospasm and prevention of its causes.

Good pain control, be it by the neuraxial route or PCA, bronchodilator therapy, incentive spirometry, deep breathing exercises, early mobilization, control of gastroesophageal reflux is beneficial in COPD.

Noninvasive positive pressure ventilation is an option in some patients who have persistent bronchospasm after tracheal extubation.

Opioids should be used cautiously as they cause respiratory depression. Morphine is avoided because of possible histamine release and increased central vagal tone which may cause bronchospasm.

NSAIDs block the cyclooxygenase mediated conversion of arachidonic acid to prostaglandins, thereby shunting arachidonic acid towards formation of bronchoconstrictor leukotriens. Therefore it is prudent to avoid NSAIDs.

The best mode of analgesia would be epidural opioids and local anesthetics. Not only they provide analgesia but also allow the patient to perform physiotherapy exercises properly.

PATIENT WITH LIVER DISEASE

Introduction

Liver disease comprises a large spectrum of hepatic dysfunction. It includes aymptomatic transaminitis, jaundice, cirrhosis, and end-stage liver disease. It may or may not be accompanied by portal hypertension.

The most common causes of advanced liver disease are chronic viral infections [hepatitis C(HCV) and B(HBV)], alcohol abuse, NAFLD/NASH, autoimmune disease, drugs or toxins, metabolic disorders (e.g. alfa-1 antitrypsin deficiency, hemochromatosis and Wilson disease), and biliary tract diseases.

The patient may require anesthesia back up for various reasons, both for surgery related to the hepatic disease or other surgeries. These conditions include, banding of esophageal varices or injection of sclerosing agent into them, underrunning of the varices, devascularization of the stomach and the esophagus, creating porta-systemic shunts or any other nonhepatic surgery in a patient with hepatic disease. A full evaluation of the baseline liver status, preoperative optimization and close postoperative monitoring are required to reduce the risk of decompensation and improve survival.

Surgical Risk Assessment

Patient with liver disease are known to be at high-risk of morbidity and mortality following emergency surgery. The magnitude of the risk depends upon the type of liver disease, its severity, the type of surgery contemplated and anesthesia offered.

Evaluation of Patients

- Evaluation should include thorough history-taking and physical examination.
- Risk factors (e.g. previous blood transfusions, tattoos, illicit drug use, alcohol use, any adverse reaction to anesthesia, personal or family history of jaundice) for liver disease should be explored.
- Patient should also be asked about history of pruritus, fatigue, distension of abdomen, etc.
- Symptoms or physical signs suggestive of liver dysfunction (e.g. hepatosplenomegaly, spider angioma, jaundice, ascites, gynecomastia, palmar erythema, scleral icterus, asterixis and encephalopathy).
- The findings of examination should be further substantiated by liver function tests, coagulation studies, complete blood cell counts (CBC) and renal function tests. Detailed tests are not possible because of the emergency nature of the surgery. However, the following test results are available sufficiently rapidly and they provide a good assessment of liver function.

Various Liver Function Tests

- Serum proteins and albumin: The total protein level may be reduced; the major share of this decline is because of the reduction in serum albumin as the synthetic ability of the diseased liver is impaired. However, the half life of albumin is 21 days and therefore, it is not a satisfactory marker of acute liver dysfunction, as the value may still be normal in presence of acute liver failure.
- Prothrombin time (PT) and international normalize ratio (INR): This also tests the synthetic ability of the liver but since the half life of prothrombin is short, it can be used to monitor the liver function over a short time period, as well as the efficacy of the treatment.
- Serum bilirubin levels and the type of bilirubin that is raised: This will help in pointing towards the likely cause of the jaundice. Obstruction to the bile outflow will cause preferentially the direct bilirubin to rise.
- Liver enzymes transaminases and alkaline phosphatase levels: Their level increases in liver necrosis and obstructive jaundice respectively.
- Renal function tests, more importantly serum creatinine concentration as the kidneys are at risk of development of hepatorenal syndrome and pigment obstruction of the renal tubules.
- Arterial blood gases to know any degree of hypoxia and acidosis which can occur as a result of hepatopulmonary syndrome.

Most of these tests are available during emergency and hence, should be definitely done. The intraoperative monitoring mostly would involve repeated measurements of prothrombin time and INR.

Two risk stratifications schemes have been used to estimate the perioperative risk of patients with cirrhosis the *Child Turcotte Pugh score* and the model for end stage liver disease *(MELD)* score.

Table 2.17 shows the Child-Pugh's assessment of liver function.

- Serum bilirubin, albumin levels, prothrombin time represents the metabolic and synthetic functions of liver.
- Ascites and hepatic encephalopathy are related to the degree of portal hypertension and porta-systemic shunting, both reflecting severity of underlying liver disease.
- Patients with Class A have 10 percent mortality, Class B have 30-31 percent mortality and Class C have 76-82 percent mortality.

Model for End-stage Liver Disease Score (MELD)

- MELD score was originally developed to predict short-term mortality for patients undergoing transjugular intrahepatic porta-systematic shunt (TIPS) placement. It has since been adapted as the tool to prioritize patients with cirrhosis for liver transplant.
- MELD score is based on patient's serum bilirubin, creatinine and INR for PT and is calculated from a validated predictive equation as follows:
  - (3.8 × in bilirubin value) + (11.2 × in INR) + (9.6 × in creatinine value) where bilirubin and creatinine values are in milligrams per deciliter (mg/dl) and it represents natural logarithm.
- Score < 8 predicts good outcome after tips.
- Score > 18 predicts poor outcome.
- Best outcome with score < 14.

Table 2.17: Child-Pugh criteria for assessment of liver function

Components	*1*	*2*	*3*
Bilirubin mg/dL	<3.4	3.4-5.1	>5.1
Albumin, gm/L	>35	28-35	<28
PT prolongation (Sec)	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3
Ascities	None	Mild to moderate	Severe
Encephalopathy	None	Grade I or II	Grade III or IV

Class A: 5-6 points, Class B: 7-9 points, Class C: 10-15 points

Preoperative Preparation

- If surgery is essential and life saving, preoperative optimization should be instituted immediately to reduce the risk of development of postoperative complications.
- Preoperative optimization includes correcting coagulopathy, ascites, hepatic encephalopathy and administering antibiotics prophylaxis.

Coagulopathy

- In addition to hepatic synthetic dysfunction (all of the coagulation factors with the exception of Von-Willebrand factor are produced in liver) malnutrition and vitamin K malabsorption due to cholestasis contributes to this abnormality.
- Portal hypertension leads to hypertension with resultant platelet trapping and peripheral thrombocytopenia.
- Vitamin K supplementation and administration of fresh frozen plasma are recommended to correct coagulopathy.
- Cryoprecipitate might also be used to reduce PT.
- A prolonged bleeding time can also be corrected with deamino-8-D arginine vasopressin (DDAVP). Finally, platelet transfusion may be necessary based on the patient's platelet level and the desired level as dictated by the type of surgery.

Ascites

- It is important to assess and manage ascites before surgery because it can lead to wound dehiscence, abdominal wall herniation and respiratory compromise secondary to reduced lung expansion. In general ascites should be treated aggressively with diuretics. There is no time or need for paracentesis before surgery. However, ascites fluid invariably is lost intraoperatively at laparotomy. This is replaced with simultaneous administration of albumin to minimize worsening of renal functions.
- Antibiotic prophylaxis should be given to prevent subacute bacterial peritonitis.
- Patients on diuretics need to have their creatinine and electrolytes monitored.

Encephalopathy

- Multiple factors in the pre- and postoperative period precipitate encephalopathy such as infection, sepsis, diuretics, hypokalemia, metabolic alkalosis, constipation, use of CNS depressants such as narcotics and benzodiazepines, hypoxia, azotemia and gastrointestinal (GI) bleed causing increased gut protein load.
- Correction of electrolytes, treatment of infection, management of GI bleed, restriction of sedatives may help prevent encephalopathy.
- Hepatic encephalopathy is often treated by administering lactulose to enhance excretion of ammonia and ammonia producing bacteria.

Renal Dysfunction

- Risk of renal dysfunction is increased by diuretics, nephrotoxic drugs including NSAIDs, large volume paracentesis performed without albumin supplementation, infections, gastrointestinal bleeding.
- Hepatorenal syndrome is another concerning occurrence. Portal hypertension results in splanchnic vasodilatation with reduced effective circulatory volume. This causes activation of the renin-angiotensin-aldosterone system leading to renal vasoconstriction and renal dysfunction (Table 2.18).
- Renal dysfunction should be closely monitored pre and postoperatively with appropriate measures taken to address or eliminate potential insults.

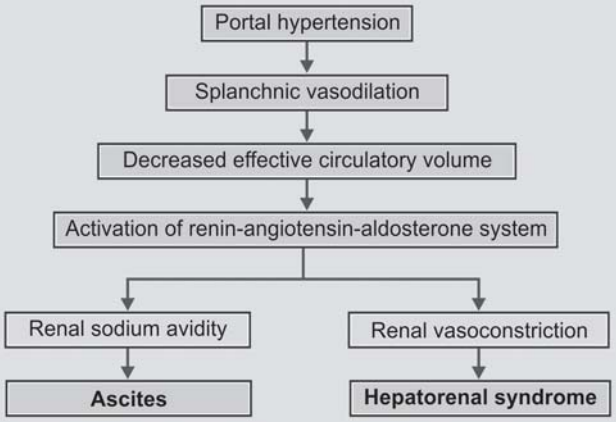
Pulmonary Disease

- Pulmonary complications of end-stage liver disease include hepatopulmonary and portopulmonary hypertension and hepatic hydrothorax.

Intraoperative Management

- The response to anesthetic agent is unpredictable in patient with cirrhosis.
- There is decreased synthesis of plasma binding proteins.

Table 2.18: Genesis of hepatorenal syndrome



- Hypoalbuminemia impairs drug binding and metabolism and elevates circulating free drug levels.
- Impaired drug metabolism, detoxification, and excretion by the liver can prolong drug half life. Thus, the absorption, distribution, metabolism and excretion of anesthetics, muscle relaxants, analgesics and sedative may be affected.
- Anesthesia causes a moderate reduction in hepatic arterial blood flow and hepatic O_2 uptake.
- The greater the degree of hemorrhage or hemodynamic instability with surgery, the greater the fall in hepatic blood flow and chance of ischemic liver injury.
- In addition catecholamine and other neurohormonal responses are impaired in patients with liver disease, therefore, intraoperative hypovolemia or hemorrhage may not trigger adequate compensatory mechanisms. Out of all the inhaled anesthetics, halothane and enflurane appears to reduce hepatic artery blood flow the most because of systemic vasodilation and mild negative ionotropic effect.
- Isoflurane has fewer effects on hepatic blood flow and less hepatic metabolism; hence, it is the preferred anesthetic agent in liver disease.
- Newer haloalkanes such as sevoflurane and desflurane also undergo less hepatic metabolism and can be used safely.
- Propofol can be used for induction. Thiopentone sodium causes enzyme induction. Opioid such as fentanyl is preferred as it reduces the dose of volatile anesthestic agents. The sedatives and anesthetics should be used very cautiously as they precipitate hepatic encephalopathy in patients with liver cell failure. The initial dose requirement may be more as the volume of distribution of these drugs is increased in portal hypertension. This to some effect is negated by the fact that the protein binding is also reduced. It is prudent to administer the drugs in titrated manner observing the clinical effects rather than giving a fixed dose.
- Atracurium has been recommended as the neuromuscular blocking agent of choice because it relies on neither liver nor kidney for excretion.
- Drugs such as morphine, meperidine, benzodiazepines and barbiturates should be used with caution because of their dependence on the liver for metabolism.
- In general the doses of these agents should be decreased by 50 percent.

Monitoring

- Close respiratory and cardiovascular monitoring is necessary in patient undergoing abdominal procedures.
- Pulse oximetry should be supplemented with ABG measurement to evaluate acid-base status.
- Intra-arterial pressure monitoring is indicated for most patients.
- Central venous pressure and pulmonary artery pressure may be necessary to assess the intravascular volume status.
- Urinary output must be followed closely.
- Intraoperatively coagulation profile may be repeated in case of substantial and ongoing blood loss.

Fluids

- Preoperatively, most patients are on sodium restriction, but intraoperatively preservation of intravascular volume and urinary output takes priority.
- The use of predominantly colloid intravenous fluids (albumin) may be preferable to avoid sodium overload and to ↑ oncotic pressure.
- Blood and blood products should be available promptly. Fluid warmers and cell saver may be of help.

Postoperatively

- Low threshold is maintained generally for postoperative transfer to the intensive care unit (ICU).
- Patients must be observed closely for signs of acute hepatic decompensation such as worsening of jaundice, encephalopathy and ascites.
- Sedatives and pain medications should be carefully titrated to prevent an exacerbation of hepatic encephalopathy.
- Renal function should be monitored because of the risk of hepatorenal syndrome and fluid shifts that occur due to surgery.
- These patients should also be monitored for surgical site complications such as infections, bleeding and dehiscence.
- Early enteral feeding has been suggested to improve outcome.
- Serious sequelae of decompensated cirrhosis include severe sepsis and secondary disseminated intravascular coagulation (DIC).

REFERENCES

1. Stoelting RK, Dierdorf SF (EDs). Endocrine diseases. In: Anesthesia and co-existing diseases. 4th edn. Pennsylvania: Churchill-Livingstone 2002;395-440.
2. Terry Davies, Reed Larsen. Thyrotoxicosis. Larsen, Kronenberg, Melmed, Polonsky, Catherine Caroll, Saith Voit (Eds). In : William's Textbook of Endocrinology. 10th ed. Philadelphia: Sunders Elsevier 2003; 374-421.
3. Satoskar RS, Bhandarkar SB, Rege Nirmala (Eds). Thyroid hormones and anti-thyroid drugs. In : Pharmacology and pharmacotherrapeutics. 21st revised edn Mumbai, Popular Prakashan Pvt. Ltd 2009;854-68.
4. Robson NJ. Emergency surgery complicated by thyrotoxicosis and thyrotoxic periodic paralysis. Anesthesia 1985;40: 27-31.
5. Michael Roizen, Lee Fleisher. Anesthetic implications of concurrent diseases. Miller, Eriksson, Fleisher, Kronish, Young, (Eds). In :Miller's Anesthesia. 7th edn. USA: Churchill-Livingstone; 2009;1067-40.
6. Dobyns BM. Prevention and management of hyperthyroid storm. World Journal of Surgery 1978;2:293-306.
7. Farling PA. Thyroid disease. British journal of anesthesia 2000;85:115-28.
8. Michael Roizen, Nadar Enany. Diseases of the endocrine system. Lee Fleisher (Ed). In: Anesthesia and Uncommon Diseases. 5th edn. USA: Saunders Elsevier 2006;413-54.
9. Merkin JM. Anesthesia and hypothyroidism: A review of thyroxin physiology, pharmacology and anesthetic implications. Anesthesia and analgesia 1982;61(4): 371-83.
10. Stoelting RK, Dierdorf SF (Ens). Endocrine diseases. In: Anesthesia and co-existing diseases. 4th edn. Pennsylvania: Churchill-Livingstone 2002;395-40.
11. McAnulty GR, Robertshaw HJ, Hall GM. Anesthetic management of patients with diabetes mellitus. Br.J. Anaesth. 2000;85(1):80-90.
12. Robertshaw HJ, Hall GM. Diabetes Mellitus: anesthetic management. Anesthesia 2006;61:1187-90.
13. Michael Roizen, Lee Fleisher. Anesthetic implications of concurrent diseases. Miller, Eriksson, Fleisher, Kronish, Young (Eds). In: Miller's Anesthesia. 7th edn. USA: Churchill-Livingstone 2009;1067-1140.
14. Dagogo-Jack, George Alberti. Management of diabetes mellitus in surgical patients. Diabetes spectrum 2000;15(1):44-48.
15. Hirsch IB, McGill JB, Cryer PE, White PF. Perioperative management of surgical patients with diabetes mellitus. Anesthesiology 1991;74(2): 346-59.
16. Mark Stafford-Smith. Caring for the patient with end-stage renal disease (ESRD) Audio Digest Summary, Volume 50, Issue 09;2008.
17. Ivan B. Perioperative Renal Insufficiency and Failure in Elderly Patients, Syllabus on geriatric anaesthesiology, ASA, 2010.
18. Moro O Salifu, Kenneth E Otah, Eseroghene Otah. Perioperative Management of the Patient With Chronic Renal Failure, eMedicine Specialties > Perioperative Care > Perioperative Care; Updated: Jan 11, 2008.
19. Sharrock NE, et al. Hypotensive epidural anesthesia in patients with preoperative renal dysfunction undergoing total hip replacement; BJA: British Journal of Anesthesia, Volume 96, (2); 207-212.
20. Wilkes BM, Mailloux LU. Acute renal failure: pathogenesis and prevention. Am J Med1986;80:1129-36.
21. Michael S, Jones F, Deane SA, Bishop G, Bauman A, Hillman. Intra-abdominal hypertension is an independent cause of postoperative renal impairment, Arch Surg. 1999;134:1082-85.
22. Acute renal failure http://anesth.unboundmedicine.com/anesthesia/ub/view/Pocket-ICU-management/105010/all/ 16 may 2010.
23. Agarwal RC, Jain RK, Yadava A. Prevention of perioperative renal failure. Indian Journal of Anesthesia 2008;52(1): 38-43.
24. Robert NS. Perioperative Care for the Patient with Renal or Hepatic Disease, http://www.anest.ufl.edu/articles/iars/sladen/sladen.html on 14 May 2010.
25. Perioperative Renal Insufficiency and Failure in Elderly Patients, Syllabus on geriatric anaesthesiology, ASA, May 14, 2010.
26. McDonald C, Milner Q, Devon R. Chronic Renal Failure and Anesthesia; ihttp://update. anesthesiologists.org/wp-content/uploads/2009/08/Chronic-Renal-Failure-and-Anesthesia-Update-18.pdf.
27. Mark Stafford-SmithAudio. Caring for the patient with end-stage renal disease (ESRD); Digest Summary, Volume 50, Issue 09 May7, 2008.
28. Sarah JJ, Pharm D. Opioids safety in patients with renal and hepatic dysfunction; www.Pain-Topics .org; June 2007; Updated: November 30, 2007.
29. Kyo SK, Jei TY, Hee JL. Time course of neuromuscular effects of rocuronium during desflurane anesthesia in patients with or without renal failure; Korean J Anesthesiol 2009; 57:566-71.
30. Gholam AM, Evaz H, Mohammad HKM, Rasool F, Kamal R. Anesthetic Management in Patients with Renal Transplant Undergoing Coronary Bypass Graft Surgery; Iranian Heart Journal 2002; 2(4) and 3(1):14-20).
31. Patrick HCW, Frances FC. Anesthesia for patient with chronic renal disease, Can Anasth. Soc. Journal: 1984;31(4);465-80.
32. Rebecca J. Acute renal failure; Indian J of Anaesth 2003;45(5):367-72.
33. Robert NS. Perioperative Care for the Patient with Renal or Hepatic Disease; http://www.anest.ufl.edu/articles/iars/sladen/sladen.html on 14 May 2010.
34. Oliver WC Jr, Nuttall GA, Orszulak TA, Bamlet WR, Abel MD, Ereth MH, Schaff HV. Hemofiltration but not steroids results in earlier tracheal extubation following cardiopulmonary bypass: a prospective, randomized double-blind trial. Anaesthesiology; 2004;101(2):327-39.
35. Bobbie JS. Overview of Preoperative Assessment and Management. In: Longnecker DE, Brown DL, Newman MF, Zapol WM (Eds). Anaesthesiology. 3rd edn. New York: Tata McGraw-Hill Companies; 2008;40-67.

36. The Sixth Report of the Joint National Committee on the Prevention, Detection, Evaluation and Treatment of High Blood Pressure. Arch Intern Med 1997;157:2413-46.
37. Kasper DL, Braunwald E, Fauci AS (Eds), et al. Harrison's principles of internal medicine, 16th edition, New York: McGraw-Hill 2005:1463-67.
38. Saddler JM. Anesthesia and Hypertension. Update in Anesthesia Issue 2 (1992) Article 3: Page 1 of 1.
39. Fleisher LA. Preoperative evaluation of the patient with hypertension. JAMA 2002; 287:2043-46.
40. McConachie I, Healy TEJ. ACE inhibitors and anesthesia. Postgraduate Medical Journal 1989;65:273-74.
41. Wallace MC, Ala SH. Systemic and pulmonary hypertension. In: Hines RL, Marschall KE (Eds). Stoelting's Anesthesia and Co-Existing Disease. 5th edn. Philadelphia: Churchill Livingstone 2008; 87-102.
42. Hall MJ, Owings MF. 2000 National Hospital Discharge Survey. Hyattsville, MD: Department of Health and Human Services; 2002. Advance Data from Vital and Health Statistics, No. 329.
43. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for non-cardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Non-cardiac Surgery). Circulation. 2007;116:1971-96.
44. Fleisher LA, Beckman JA, Brown KA, et al. 2009 ACCF/AHA focused update on perioperative Beta-blockers incorporated into guidelines on perioperative cardiovascular evaluation and care for non-cardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2009; e169-276.
45. Sock HK, James R. Anesthesia for patients with cardiac disease undergoing non-cardiac surgery. Update in Anesthesia; Issue 14 (2002) Article 4: Page 1 of 2.
46. Tej KK, Geeta T. Anesthetic Considerations in Cardiac Patients Undergoing Non Cardiac Surgery. Indian Journal of Anesthesia 2007;51(4): 280-86.
47. Shamsuddin A. Ischemic Heart Disease. In: Hines RL, Marschall KE (Eds). Stoelting's Anesthesia and Co-Existing Disease. 5th edn. Philadelphia: Churchill Livingstone 2008;1-26.
48. Nikolaos S, Lichtman AD, Aarti S, Thomas SJ. Anesthesia for Cardiac Surgery. In: Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anesthesia, 5th ed. Philadelphia, USA: Lippincott Williams & Wilkins 2009; 887-933.
49. Sarkar D, Goel S, Jain R, Deepak K. Tempe. Emergency Craniotomy in a Patient with Severe Mitral Stenosis and Aortic Stenosis: A Case Report. J Anaesth Clin Pharmacol 2004;20(2):183.
50. Grant IS, Nimmo GR. Intercurrent disease and anesthesia. In: Aitkenhead AR, Smith G, Rowbotham DJ (Eds). Textbook of Anesthesia. 4th edn. New York: Churchill Livingstone 2001; 429-54.
51. Adriana Herrera. Valvular heart disease. In: Hines RL, Marschall KE (Eds). Stoelting's Anesthesia and Co-Existing Disease. 5th edn. Philadelphia: Churchill Livingstone 2008;27-42.
52. Torsher LC, Shub C, Rettke SR, Brown DL. Risk of patients with severe aortic stenosis undergoing noncardiac surgery. Am J Cardiol 1998; 81:448-52.
53. Stoelting RK, Dierdorf SF. Anesthesia and coexisting disease, 5th edn, Philadelphia: Churchill Livingstone; 2008.
54. Barash PG, Cullen BF, Stoelting RK (Eds). Clinical anesthesia, 5th edn, Philadelphia, Lippincott, Williams & Wilkins 2006:484.
55. Bishop MJ, Editorial views. Preoperative costicostroids for reactive airway: Anaesthesiology 2004:100:147-48.
56. Silvanus MT, Groeben H, Peters J. Corticosteroids and inhaled salbutamol in patients with reversible airway obstruction markedly decrease the incidence of bronchospasm after tracheal intubation. Anaesthesiology 2004;100:1052-57.
57. Eameswo, Rooke G A, WURS, et al. Comparison of the effects of Etomedate, propofol, thiopental on respiratory resistance following tracheal intubation. Anaesthesiology 1996;63:844.
58. Rock GA, Choi JH, Bishop MJ. The effect of isoflurane, halothane, sevoflurane, thiopental, N_2O on respiratory system resistance after tracheal intubation Anaesthesiology 1997;86:1294-99.
59. Miller RD (Ed). Miller anesthesia, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005; 511-12.
60. Woods BD, Sladen RN. Preoperative consideration for the patients with asthma & bronchospasm. BJ vol103, PJ 157-165.
61. Kao CC, Janis, Gentapalli KK, Bandi V. Mechanical Ventialtion for asthma. J asthma 2008;45:552-6.
62. Laurence VA, Cornell JE, Smetana GW. Strategies to reduce postoperative pulmonary complications after noncardiac surgery: Systematic review for the American college of Physicians. Intern Med 2006;144:596-608.
63. Harding SM. Gastroesopheageal reflex, potential asthma trigger, Immunol Allergy clin north Am 2005;25:131-48.
64. Qaseem A, Snow V, Filterman N, et al. Risk assessment for strategies to reduce perioperative pulmonary complications for patients undergoing non cardiothorasic surgery: a guideline from the American college of Physicians from Intern med 2006;144:575-80.
65. Miller KH, Harkin CP, Bailey PL, Postoperative tracheal intubation Anesthesia. Analg 1995;80:148-72.
66. Marsh HM. Anesthesia for Patients with Chronic pulmonary disease. In Hershey SL (Ed). ASA refresher courses in Anaesthesiology Park Ridge, II: American Society of Anesthesiologists; 1984-133.
67. Hines, Marschall (Eds). Diseases of the liver and biliary tract, chapter 11, In Stoelting's Anesthesia and Co-existing diseases, 5th (edn), the Churchill Livington publishers, Philadelphia, USA, 2008.

3 Choice of Anesthetic Agents for Emergency Surgery

Aparna A Nerurkar, Deepti M Kotwani, Manju N Gandhi

KEY POINTS

- Choice of anesthetic agents in emergency surgery is dictated by many factors—patient factors, anesthesiologist preferences, drug pharmacology and environmental factors.
- Patient factors include type of surgery, urgency of surgery indication, fasting status, hemodynamic stability, ease of airway and presence of associated organ dysfunctions.
- Anesthesiologist's preference will depend on prior experience of the type of surgery that is being undertaken, anesthesia management in earlier patients with similar pathophysiology, familiarity with techniques and drugs available.
- Environmental factors contributing to decision making include the nature of the anesthetizing location-full-fledged operation theater versus remote location, availability of trained assistance, ready accessibility of drugs and relevant equipment.
- An organized approach to patient management and thorough knowledge of pharmacology inclusive of pharmacokinetics and pharmacodynamics of anesthetic drugs is crucial for a positive outcome.
- The 'ideal' emergency anesthetic induction agent is one which rapidly achieves unconsciousness, permits optimum intubating conditions, and is devoid of cardiovascular, respiratory, and cerebral side effects. However, no single anesthetic agent meets these criteria.
- Balanced anesthesia with multiple drugs—fast-acting intravenous induction agents and inhalational anesthetics with complementary properties are normally combined with a benzodiazepine, an opioid and neuromuscular blocking agent.
- Special precautions to prevent aspiration, infections and condition specific drugs like anticonvulsants, corticosteroid, drugs to blunt intubation response, etc. need consideration.
- In patients presenting for emergency surgery, the degree of hypovolemia, intensity of surgical stimulus, and the autonomic and somatic responses to surgical and traumatic injury and concomitantly used anesthetics vary widely, necessitating individualized dosages and careful titration.

INTRODUCTION

Patients coming for emergency anesthesia care comprise a wide range of unique management challenges for the entire medical team. First, these patients arrive at the hospital with minimal warning. Second, the patient's initial presentation can be vivid and associated with severe physiological and emotional stress. Finally, the most seriously ill patients are typically admitted directly to the operating room (OR) for life-saving surgery without evaluation and/or resuscitation.

No standard sequence or combination of induction and neuromuscular blockade (NMB) drugs are appropriate for all emergency situations. Choice of anaesthetic agents is dictated by many factors—patient factors, anesthesiologist preferences, drug pharmacology and environmental factors. Salient patient factors include type of surgery, urgency of indication, fasting status, hemodynamic stability, ease of airway and presence of organ dysfunctions. Experience of the anesthesiologist in managing similar type of patients in the past, familiarity with techniques, equipments and drugs plays a significant role in anesthesia plan and decision making. Perhaps the most important factor is thorough knowledge of the pharmacokinetics and pharmacodynamics of the drugs that are available. Major environmental factors contributing to decision making include the nature of the anesthetizing location-full-fledged operation theater versus remote location,

availability of trained assistance, ready accessibility of drugs and relevant equipment.

Modern anesthesia practice strives for "anesthesia, analgesia, amnesia, areflexia and autonomic stability". In *emergency* surgery, however, one may need to balance these needs with hemodynamic stability and organ dysfunction. Also the need for rapid sequence induction mandates modification of techniques with laryngoscopy and intubation performed earlier in the sequence and drugs administered in rapid succession. The 'ideal' emergency anesthetic induction agent is one which rapidly achieves unconsciousness and yet does not itself cause hemodynamic compromise. No single anesthetic agent meets these criteria. Thus, balanced anesthesia with multiple drugs is needed rather than relying on a single agent. Moreover, the use of separate drugs to modulate each desired effect allows both the anesthetist and the surgeon a greater measure of control.

Fast-acting intravenous induction agents and inhalational anesthetics with complementary properties are normally combined with a benzodiazepine, an opioid and neuromuscular blocking agent. Special precautions to prevent aspiration, prophylactic antibiotics, condition specific drugs like anticonvulsants and corticosteroid need consideration.

INTRAVENOUS INDUCTION AGENTS

The ideal intravenous induction agent for emergency airway management should be smooth and rapidly acting, painless on injection, permit optimum intubating conditions, and be devoid of cardiovascular, respiratory, and cerebral excitatory side effects. As yet, no single agent achieves all the requirements. Each agent has particular properties and for effective use in clinical practice, these are best tailored to each patient's condition and the requirements of the surgery.

Intravenous induction drugs are selected based upon a patient's neurological and hemodynamic condition. Intravenous induction drugs often require titration to be effected with careful attention to patient response. Reduced dosages of induction drugs are frequently required during emergency situations (for patients who are often hypovolemic).[1]

Thiopentone

Thiopentone is an ultra-short acting barbiturate. It acts on the $GABA_A$ receptor; increases chloride conductance in a concentration dependent manner thus potentiates the inhibitory effects of GABA.

Thiopentone is available as a sodium salt (0.5 g pale yellow powder) to promote dissolution of the drug in 20 ml water (to form a 2.5% solution). The use of a 5%. solution increases the likelihood of serious complications and is not recommended.[2]

Uses/indications in emergency: Because of its rapid action (within 5-15 seconds) and short duration, thiopentone is the historical gold standard for induction for general anesthesia especially in rapid sequence induction. It can be used in combination with other anesthetics or as the sole anesthetic agent for very short procedures. *However, thiopentone should never be administered without proper equipment, i.e. a secured intravenous access, tilting table, airway equipment with means of artificial ventilation and drugs for resuscitation.*

Contraindications/Precautions

Absolute contraindications:[2]

- History of hypersensitivity reactions to barbiturates
- Porphyrias
- Status asthmaticus.

Relative contraindications:

These relative contraindications do not preclude the use of thiopentone, but dosage adjustments must be considered and the drug must be given slowly and cautiously. 100% oxygen may be required to take care of associated complications. In some cases nitrous oxide supplementation may be required to reduce the dosage of thiopentone.

- Severe cardiovascular disease especially fixed cardiac output states like mitral stenosis, aortic stenosis (where compensation for hypotension is not possible), or pre-existing ventricular arrhythmias. As compared to propofol, hemodynamics (mean arterial pressure and systemic vascular resistance) are better maintained. in patients receiving thiopentone for induction and tracheal intubation,[3]
- Shock or severe cardiovascular instability
- *Myasthenia gravis*: Respiratory depression caused by thiopentone is prolonged in such patients.[2]
- *Asthma or reactive airway disease*: The availability of propofol has restricted the use of this drug in asthma and reactive airways in recent practice.
- Respiratory obstruction or an inadequate/difficult airway—as thiopentone may worsen the respiratory depression.[4]
- Conditions where hypnotic effects may be prolonged (e.g. severe hepatic disease, myxedema, severe anemia, excessive premedication, etc).
- Pregnancy—thiopentone readily crosses the placental barrier and should be used with caution.

Side Effects[2-4]

- Anaphylaxis
- *Allergic reactions:* facial edema, hives, bronchospasm
- *Hypotension:* Depending on dose and rate of injection
- Laryngospasm may occur if airway stimulation is performed in lighter plane of anesthesia, oral secretions, or blood in oral cavity and inflammation around the neck, jaw and oral cavity.[2]
- *Pentothal apnea:* Prolonged apnea and muscle relaxation may be seen in Huntington's chorea, hypokalemic periodic paralysis and muscular dystrophies.[4]
- Local tissue irritation and rarely tissue necrosis.
- Urticarial rash may develop on the head, neck, and trunk that last a few minutes.

Dosage and Administration

The usual dose of thiopentone is 4 to 7 mg/kg given intravenously over 5 to 15 seconds titrated to loss of eyelash reflex. If thiopentone is to be given during emergency anesthesia especially with hemodynamic instability, it may be given in small increments, after a test dose, while constantly monitoring hemodynamic stability and adequacy of respiration. The total dose has to be titrated to the underlying pathology. Interpatient dose variability is related to the presence of hemorrhagic shock, cardiac output, lean body mass, obesity, hepatic function concomitant drug therapy and age.[4,5]

Anticipate decrease in the required dose of thiopentone in the following conditions:[4,5]

- Presence of concurrent disease (e.g. cardiac dysfunction, COPD, hypovolemia)
- decreased blood volume (patients with shock or dehydration)
- Severe anemia, burns, malnutrition, widespread malignant disease, uremia, ulcerative colitis, or intestinal obstruction
- Previous premedication (e.g. with benzodiazepines and/or opioids)
- Less lean body mass (obese, elderly, female patients).

Higher doses of thiopentone will be required in following conditions:[5]

- Chronic alcoholics
- Acquired tolerance because of chronic use of barbiturates
- Cross-tolerance to benzodiazepines and anticonvulsants

Propofol

Propofol is 2, 6-diisopropylphenol available in an emulsion form. Propofol exerts its CNS effects primarily via the $GABA_A$ receptor. In recent practice it has become the most widely used intravenous anesthetic agent.

Propofol is 98% protein bound and undergoes hepatic metabolism to inactive glucuronide metabolites, which are ultimately excreted in urine.

The pharmacokinetics of propofol may be altered by various factors (e.g. gender, weight, pre-existing disease, age, and concomitant medication). Propofol may impair its own clearance by decreasing cardiac output and hepatic blood flow.

Uses/Indications in Emergency Surgery[1,2,4,5]

- *Induction and maintenance of anesthesia.*
  - An induction dose of 1 to 3 mg/kg is often given to the typical healthy patient undergoing elective surgery, but there is enormous variability between patients in the amount actually required. Coexisting factors, such as previous premedication (e.g. with benzodiazepines and/or opioids), advanced age, or presence of concurrent disease (e.g. cardiac dysfunction, COPD, hypovolemic or hemodynamically unstable patients), will decrease the required dose of propofol.
  - Older and sicker (ASA class III to IV) patients develop more profound hypotension, with propofol, especially when combined with opioids.
  - Propofol, when used for induction of anesthesia for short procedures/day care surgery, results in a significantly faster recovery and an earlier return of psychomotor functions compared with thiopental or other agents, making it most suitable induction agent for ambulatory procedures.
  - Following an induction bolus, infusion rates of 100 to 200 μg/kg/min typically are used to maintain general anesthesia in healthy patients. If nitrous oxide and/or opioids are administered concurrently, a reduction in the required propofol infusion rate by one third to half may be anticipated.
- *Sedation during Surgery*
  - Propofol is commonly used for procedural sedation as well as for long-term sedation in the intensive care unit (ICU). If loss of consciousness is to be avoided (as is the goal for most cases of

procedural sedation), a loading dose of 0.5 to 1 mg/kg is used, followed by an infusion of 25 to 75 µg/kg/min. Lower infusion rates will be required in persons given benzodiazepines and/or opioids, in elderly patients, and in those with coexisting cardiopulmonary disease.

– *As an antiemetic.* Propofol has intrinsic antiemetic activity and has been used successfully as an antiemetic. Dose: 10 to 20 mg IV, can repeat every 5 to 10 min or start infusion of 10 µg/kg/min.

Dosage

Induction of general anesthesia: 1 to 3 mg/kg IV (dose titrated to effect).

Maintenance of general anesthesia 50 to 200 mcg/kg/min IV combined with N_2O or an opiate.

Sedation 25-75 µg/kg/min infusion.[4]

- *Anticipate decrease in dose requirement of propofol in cases of:*[5]
  - Previous premedication (e.g. with benzodiazepines and/or opioids)
  - Advanced age
  - Presence of concurrent disease (e.g. cardiac dysfunction, COPD, hypovolemia)
  - In hepatic disease (decreased protein binding fraction)
  - Hemorrhage treated only with crystalloid solutions and/or packed red blood cells (decreased protein binding fraction)
- *Anticipate increase in dose requirement of propofol*:[5]
  - Patients with acquired tolerance due to chronic use of medications that exhibit cross-tolerance with propofol (e.g. benzodiazepines, barbiturates, anticonvulsants, or alcohol)

Adverse Effects/Cautions[2,4]

- *Cardiovascular depression and hypotension:*
  - Unless the drug is given very slowly, bolus dose of propofol causes profound hypotension (20-30%) in hypovolemic or untreated hypertensive patients and in those with cardiac disease[2]
  - Hypotension is augmented by the concomitant administration of opioids for the same reason. However, cardiovascular depression is modest if the drug is administered slowly or by infusion
  - The decrease in cardiac output and systemic vascular resistance (SVR) is dose dependent. As propofol causes reset or inhibition of the baroreflex,[2,5] it prevents the reflex correction of hypotension with tachycardia
  - Ephedrine has been given before rapid tracheal intubation using propofol and rocuronium bromide to combat hypotension[6]
- *Respiratory depression:* Apnea is more common and of longer duration than after thiopentone administration. Furthermore, addition of an opiate increases the incidence of apnea, especially prolonged apnea. Therefore, patients with potentially difficult airway require alternate plans for ventilation and intubation when propofol is used[1,2]
- *Excitatory phenomena:* Seizures after propofol administration have been reported, mainly on induction or emergence from anesthesia, rarely during maintenance, and occasionally postoperatively. The use of propofol in head injury and other neurosurgery and cerebral protection is thus controversial[2,5]
- *Pain on injection:* This occurs in up to 40% of patients. The incidence is reduced if a large vein is used, if a small dose (10 mg) of lidocaine is injected shortly before propofol, or if lidocaine is mixed with propofol in the syringe (up to 1 ml of 0.5 or 1% lidocaine per 20 ml of propofol). Accidental extravasation or intra-arterial injection does not result in adverse effects[4]
- *Allergic reactions:* Skin rashes occur occasionally. Anaphylactic reactions have also been reported, but appear to be no more common than with thiopentone
- *Propofol infusion syndrome* is a rare but lethal syndrome associated with infusion of propofol at 4 mg/kg/hr or more for 48 hours or longer[4]
- Propofol is not licensed for use in children aged less than one month age. There have been reports of unexpected deaths in children due to metabolic acidosis and myocardial failure after long-term use in the ICU.[4,7]
- *Others:*
  - Prolonged administration of propofol has been associated with the development of pancreatitis which is related to hypertriglyceridemia, hence cautious use has been recommended in patients with hyperlipidemias.
  - Hallucinations, and opisthotonus have been reported after propofol administration.[4]
  - Solutions of propofol support the growth of microorganisms. The drug must be drawn aseptically into a syringe and any unused solution should be discarded if not administered promptly. All opened vials and syringes containing propofol (irrespective of preservative) should

routinely be discarded after 6 hours in order to reduce the risk of bacterial contamination.[2]

Ketamine

Ketamine is a phencyclidine derivative and produces its inhibitory effects by blocking the NMDA receptor. The anesthetic state induced by ketamine is called "dissociative anesthesia" a functional/electrophysiological dissociation between the thalamocortical and limbic system, characterized by catalepsy in which the eyes remain open with slow nystagmic gaze, while corneal and light reflexes remain intact and patients do not respond to noxious stimuli or have any recall of events that occurred during the anesthesia.[5]

At subanesthetic concentrations, ketamine produces good analgesia (unlike propofol or barbiturates), that lasts even in the postoperative period.

Ketamine has a pKa of about 7.5, with a lipid solubility of 5–10 times that of thiopental, but with a lower proportion of the drug binding to plasma protein (45–50%).

Indications/Uses in Emergency Anesthesia

- *Induction and Maintenance of Anesthesia:*[1,4,5,7-9] Candidates for ketamine induction are:
  - High-risk patients (ASA class IV) with respiratory and cardiovascular system disorders (excluding ischemic heart disease)
  - Patients with reactive airway disease, susceptible to bronchospasm
  - Hemodynamically compromised patients due to either hypovolemia (e.g. ruptured abdominal aortic aneurysm, polytrauma) or septic shock or cardiomyopathy (not coronary artery disease)[8]
  - Cardiac diseases such as cardiac tamponade and restrictive pericarditis, congenital heart disease (right-to-left shunt)
  - Otherwise healthy trauma victims whose blood loss is extensive also are candidates for rapid-sequence of anesthesia induction with ketamine
  - Ketamine combined with propofol or midazolam can be given by continuous infusion to produce satisfactory cardiac anesthesia for patients with valvular and ischemic heart disease
  - Ketamine can be relatively safely administered in trauma or burn patients who are not in shock, and in whom spontaneous ventilation can be safely assured by their natural airway
  - A rapid onset after intramuscular injection makes ketamine a frequent choice for induction of anesthesia in children and adults with mental disabilities who will not tolerate a mask or the placement of an intravenous catheter.
- *Monitored Anesthesia Care and Sedation:* (especially in remote locations)
  - In subhypnotic doses (0.15 to 0.3 mg/kg)[4], ketamine is commonly used for monitored anesthesia care (as in short surgical procedures, anesthesia care at remote locations, etc.). It produces profound analgesia without the concurrent ventilatory depression produced by opioids. In combination with a low dose of propofol or a benzodiazepine, the unpleasant psychological effects are minimal, and the ventilatory depressant effect is much less than the combination with an opioid.
  - *Oral Premedication in Children:*[4] Ketamine is commonly used, either alone or in combination with midazolam, as an oral medication in children, especially as the sole agents for conscious sedation for a short but noxious procedure (e.g. upper endoscopy). Typical doses of the combination are 3–6 mg/kg of ketamine plus 0.25–0.5 mg/kg of midazolam
- *Supplement or an Adjunct to Regional Anesthesia:*[2,4]
  - In adults and children, ketamine can be used as a supplement or an adjunct to regional anesthesia, extending the usefulness of the primary (local anesthetic) form of anesthesia.
  - Ketamine can be used before the application of painful blocks, but more commonly it is used for sedation or supplemental anesthesia during long or uncomfortable procedures.
- *Analgesia*
  - The analgesic action of ketamine may be used when wound/burn dressings are changed, or while positioning patients with pain before performing regional anesthesia (e.g. fractured neck of femur).
  - Ketamine administered in small doses decreases postoperative analgesic consumption. Many meta-analyses have shown an overall decrease in opiate use or improved analgesia and a decrease in opiate-induced side effects, especially postoperative nausea and vomiting (PONV).[4]
  - The epidural/caudal administration of ketamine (0.5 to 1 mg/kg) has been increasingly reported. Although the efficacy of these doses of ketamine seems to be established, the safety of this technique has not yet received regulatory approval.

Dosage and Administration[2,4]

Ketamine has been administered intravenously, intramuscularly, transcutaneously, orally, nasally, and rectally, and as a preservative-free solution epidurally or intrathecally.

Dose for induction 0.5-2 mg/kg IV or 4-6 mg/kg IM

Dose for sedation: 0.2-0.8 mg/kg IV over 2-3 min, 2-4 mg/kg IM

Analgesia and sedation (0.15-0.25 mg/kg IV)

Oral premedication in children (3-6 mg/kg with/without 0.25-0.5 mg/kg midazolam).

Adverse Effects/Caution

- Ketamine causes direct myocardial depression, which can cause hemodynamic collapse when administered as a bolus in hypovolemic and/or catecholamine-depleted patient
- Ketamine had previously been the drug of choice for trauma patients due its sympathomimetic effects. It has now been known for almost two decades that the sympathomimetic effect of ketamine is negligible in already maximally stressed patients[1]
- Ketamine induces psychotomimetic activity and emergence reactions (e.g. vivid dreams, hallucinations, and delirium). These can occur in up to 30% of patients and especially in aged, females, patients with personality disorders, or with rapid intravenous injection. Benzodiazepines are probably the most effective drugs for attenuating psychic reactions[2,4]
- Ketamine produces marked salivation, especially in children; therefore an antisialogogue should be administered before it is used.
- Despite pharyngeal reflexes being preserved, and the upper airway remaining relatively patent with ketamine, airway management is still necessary, as with all intravenous induction agents. Laryngeal reflexes remain active (with the risk of laryngeal spasm) and regurgitation and aspiration are still possible[2]
- Sympathetic stimulation, with increased circulating concentrations of catecholamines, resulting in peripheral vasoconstriction and direct cardiac stimulation—this may be harmful in previously hypertensive patients and in those with ischemic heart disease and raised pulmonary artery pressures[1]
- Repeated administration or continuous infusion leads to prolonged recovery period; making ketamine is unstable suitable drug for maintenance of anesthesia
- Visceral stimulation: Ketamine poorly suppresses the response to visceral stimulation therefore supplementation (e.g. with an opioid) is indicated whenever visceral stimulation is anticipated.

Contraindications[4,5]

Contraindications to ketamine relate to specific pharmacologic actions and patient diseases.

- Patients with raised intracranial pressure (ICP) and with intracranial mass lesions breathing spontaneously should not receive ketamine because it can increase ICP and has been reported to cause apnea
- Open eye injury or other ophthalmologic disorder—in which a ketamine-induced increase in intraocular pressure would be detrimental
- Ischemic heart disease and vascular aneurysms—since ketamine has a tendency to cause hypertension and tachycardia which will increase the myocardial oxygen consumption, thereby further deteriorating the myocardial function.
- History of adverse reaction to ketamine, is an absolute contraindication

Etomidate

Etomidate is a nonbarbiturate imidazole derivative. It is water insoluble, unstable in a neutral solution, supplied as a 0.2% solution in propylene glycol (35% by volume).[2,4]

Etomidate is metabolized in the liver primarily by ester hydrolysis or by N-dealkylation

Only small fraction of the drug is excreted unchanged, the rest being excreted as metabolites by the kidney (85%) and bile (13%).[4]

Onset of anesthesia after a routine induction dose of 0.3 mg/kg of etomidate is rapid (one arm–brain circulation) and is equivalent to anesthesia obtained with an induction dose of thiopentone sodium.

The duration of anesthesia after a single induction dose is linearly related to the dose-each 0.1 mg/kg administered provides about 100-120 seconds of loss of consciousness. Recovery after multiple doses or an infusion of etomidate is still usually rapid.

Uses/Indications in Emergency Surgery

Induction and maintenance of anesthesia:[1,4,5] Etomidate has minimal hemodynamic effects in healthy patients and those with cardiac disease, which makes the agent suitable for patients with hypotension, hypovolemia or cardiovascular disorders. The hemodynamic stability of

etomidate is unique among the rapid-onset anesthetics used to induce anesthesia.

Etomidate has been used for anesthesia induction in following cases:

- Patients with compromised cardiovascular system who are undergoing coronary artery bypass surgery or valve surgery, coronary revascularization, aortic aneurysm repair, and thoracic surgery
- For cardioversion especially in hemodynamically unstable patients
- In many cases, the combination of etomidate with a rapid onset NMB drug (succinylcholine or rocuronium) will provide the best (safest and most efficacious) induction conditions for the trauma patient requiring rapid sequence induction. Though evidence both for and against etomidate has been reported.[10,11]
- Neurosurgical procedures such as giant aneurysm clippings for intracranial hypertension
- Trauma patients with questionable volume status especially the patients whose trauma may be related to drug or alcohol abuse
- Patients with reactive airway disease
- Any combination of disorders indicating the need for an induction agent with limited or beneficial physiologic side effects.

Sedation:

- Short-term sedation with etomidate is useful in hemodynamically unstable patients, such as patients requiring cardioversion, or patients requiring sedation after an acute myocardial infarction or with unstable angina for a minor operative procedure.

Dosage and Administration[4,5]

An average dose of 0.3 mg/kg iv induces anesthesia (0.2-0.6 mg/kg).

Maintenance of general anesthesia—10 µg/kg/min IV with N_2O and/or an opiate**.**

Adverse Effects/Caution[1,2,4]

- Although etomidate provides stable hemodynamics and minimal respiratory depression, it is associated with several adverse effects when used for induction, including nausea and vomiting (30%), pain on injection (10-80%), myoclonic movement (40%), and hiccups (10%)
- *Adrenocortical suppression:* Although there is substantial conflict in the literature on this issue, etomidate can transiently inhibit endogenous catecholamine/cortisol elaboration, which will lead to vasodilation and can cause a drop in blood pressure in the compromised patient
- *Emergence phenomena:* The incidence of severe restlessness and delirium during recovery is greater with etomidate than barbiturates or propofol
- Venous thrombosis is more common than with other agents (select a large vein; add lignocaine 0.5 -1%)
- Etomidate enhances the neuromuscular blockade of nondepolarizing neuromuscular blockers
- Although many *in vitro* studies have shown that etomidate inhibits aminolevulinic acid synthetase, it has been administered to patients with porphyria without inducing an acute attack of porphyria
- Propylene glycol, present as preservative in etomidate has been reported to be associated with a small degree of hemolysis. High-dose prolonged infusion is therefore not recommended.

INHALATIONAL ANESTHETIC AGENTS

Volatile anesthetics produce far greater cardiovascular depression than does other anesthetic agents. Furthermore, trauma patients or patients for emergency surgery have multiple reasons to display unpredictable or exaggerated responses to usual doses of these agents. They may have an altered volume of distribution, cerebral blood flow, cardiac output, as well as concomitant anemia and hypoxemia. The presence of other agents such as alcohol, "illict drugs" or pressor agents administered during resuscitation may also modify the minimum alveolar concentration (MAC) of inhaled anesthetics. The safe use of the inhaled anesthetic drugs in emergency situation requires both an understanding that only reduced concentrations will be initially tolerated, and careful titration to avoid adverse outcomes.

All of the commonly used inhaled drugs (halothane, isoflurane, desflurane, and sevoflurane) cause significant dose-dependent myocardial depression. These potentially deleterious cardiovascular effects are far more pronounced in the hypovolemic, or otherwise hemodynamically compromised person.[1,12] Physical characterstics and major systemic effects of inhalational anesthetic agents are summarised in Tables 3.1 and 3.2.

Sevoflurane

Sevoflurane is a highly fluorinated ether. Sevoflurane has a pleasant odor, low pungency and low blood solubility making it well tolerated for inhalation induction of anesthesia.

Table 3.1: Physical characterstics of inhalational anesthetic agents[12]

Agent	B.P (°C)	$\lambda_{blood/gas}$ #	MAC (in O_2)	MAC (in 70% N_2O)	Pungency
Sevoflurane	58.6	0.65	2.0	0.66	↑
Desflurane	22.8	0.45	6.0	2.5	↑↑↑↑↑
Isoflurane	48.5	1.4	1.28	0.56	↑↑↑↑
Halothane	50.2	2.5	0.75	0.29	↑↑
Nitrous oxide	-88.5	0.47	105	-	-

BP: Boiling point at 1 atmospheric pressure, $\lambda_{bloog/gas}$: Blood gas partition coefficient at 37°C, MAC: minimum alveolar concentration

Table 3.2: Cerebrovascular and cardiovascular effects of inhalational anesthetic agents[1]

Agent	Cerebrovascular effects		Cardiovascular effects		
	CBF & ICP	$CMRO_2$	HR	Myocardial depression	SVR
Sevoflurane	↑	↓	↑↑	Moderate	↓↓
Desflurane	↑↑	↓↓	↑↑	Moderate	↓↓↓
Isoflurane	↑↑	↓↓	↑↑↑	Moderate	↓↓↓
Halothane	↑↑↑↑↑	↓	↓	Severe	↓
Nitrous Oxide	↑	↑	↔	Modest (at high dose)	↔

CBF, cerebral blood flow; ICP, intracranial pressure; $CMRO_2$, cerebral metabolic rate of O_2 consumption; ↑ increased effect; ↓ decreased effect; ↔ No change; HR Heart rate; SVR systemic vascular resistance

Uses/Indications in Emergency Anesthesia

Induction of anesthesia can be achieved in approximately 1-3 minutes by using 1.5 to 3 percent sevoflurane in air or in oxygen, or by using 0.7 to 2 percent sevoflurane in 60 percent nitrous oxide.[4]

Sevoflurane does not cause coughing and excitation during induction and can be used without intravenous anesthetics. Maintenance of anesthesia can be achieved with 0.4 to 2 percent sevoflurane.

The low tissue solubility of sevoflurane results in rapid elimination and awakening.

Along with rocuronium, sevoflurane has been tried for rapid sequence induction using single breath (6-8%) technique providing satisfactory conditions.[13-15] However hemodynamic instability may be produced with such high concentrations.[16-18]

Adverse Effects/Caution

- The depth of anesthesia may be difficult to evaluate during mask induction with sevoflurane 8%. This may have major consequences in hypertensive patients. A paradoxic systemic hypertension has been observed during single breath vital capacity induction of anesthesia with sevoflurane in patients with previously controlled hypertension. This hypertension can occur despite two and a half minutes of inhalation of sevoflurane 8% in N_2O 50%.[19,20] The exact causes of such a hypertensive crisis remain to be elucidated. An increase in sympathetic reactivity in hypertensive patients as well as a nociceptive stimulus during light anesthesia may trigger this hypertension. Transient increase in sympathetic reactivity during induction with high concentrations of sevoflurane has been blamed by many studies.[20,21]
- Alkali (baralyme>sodalime>calcium hydroxide) can degrade sevoflurane, producing 'Compound A' (fluoromethyl-2,2-difluoro-1-[trifluoromethyl]vinyl ether) which is a nephrotoxic end product proven in rats.[4] The concentration of compound A is highest during low-flow anesthesia (<2 L/min) and is reduced by increasing fresh gas flow rate. Avoid its use in patients with poor renal function.
- Sevoflurane is defluorinated through oxidative metabolism with serum fluoride concentrations which peak >50 µmol/l even when sevoflurane is administered for average duration. However due to sevoflurane's low blood-gas solubility and its rapid elimination, fluoride concentrations fall very quickly after surgery, and renal toxicity is not expected from sevoflurane administration.[2]

Advantages[1,2,4,19,20]

- Unlike desflurane, sevoflurane does not irritate the airway. The absence of airway irritation and bronchodilatory effects of sevoflurane are effects beneficial to patients with asthma and reactive airway disease.

- Sevoflurane is less arrhythmogenic than halothane and maintains hemodynamic stability
- Due to its low solubility in blood it can be used for rapid induction of anesthesia without intravenous anesthetics. This is one of the reasons why it is currently replacing halothane for mask induction in pediatric patients
- Requires conventional vaporizers (particularly when compared with desflurane).

Halothane

It has a pleasant, nonpungent odor, it is least expensive volatile anesthetic and it is tolerated well during inhalation induction of anesthesia.

Recovery from halothane anesthesia is slower than with the other agents because of its high blood/gas solubility and recovery is prolonged with increasing duration of anesthesia.

Approximately 20% of halothane is metabolized in the liver, usually by oxidative pathways and small proportion of halothane may undergo reductive metabolism.

Adverse Effects/Caution during Emergency Anesthesia

- Many studies in human myocardium have demonstrated halothane to have the most profound negative inotropic effect.[1] Halothane also attenuates the baroreceptor reflex, blunting the ability to maintain cardiac output by the usual increase in heart rate. From this perspective, halothane can be viewed as an "inhalational beta-blocker".
  - Hypovolemic patients and some patients with severe cardiac disease (aortic stenosis) may not tolerate halothane's negative inotropic effects.[1,4]
  - Bradycardia, hypotension, and cardiac arrest may occur during induction of anesthesia specially in infants and children which may be due to the administration of excessive concentrations of halothane or due to an increased sensitivity of the myocardium in neonates to inhalational anesthetics.[22]
- Halothane is also known to sensitize the myocardium to catecholamines. This is particularly relevant in trauma patients, as they often present with high endogenous levels of these compounds and are far more likely than other patients to receive exogenous vasopressors and inotropic drugs. This effect is exacerbated in the setting of hypercarbia
- Like all other inhalational agents it can trigger malignant hyperthermia (MH) reactions (halothane > enflurane > isoflurane > methoxyflurane).[22]
- Great caution is required in patients with intracranial mass lesions as there is possibility of intracranial hypertension. Increase cerebral blood flow caused by halothane is highly significant and more than any other inhalational anesthetic agents
- Patients exposed to multiple halothane anesthetics at short intervals (<3 months), middle-aged obese women, and patients with a familial predisposition to halothane toxicity or a personal history of toxicity are considered to be at increased risk to halothane hepatitis.[23]

Isoflurane

Isoflurane (l-chloro-2,2,2-trifluoroethyl difluoromethyl ether), is an isomer of enflurane, a colorless, volatile liquid with a slightly pungent odor, stable (does not require preservatives for storage), nonflammable in clinical concentrations.

Isoflurane is metabolized (0.17% of the absorbed dose)[2] to trifluoroacetic acid. Because of the minimal metabolism, only very small concentrations of serum fluoride ions are found, even after prolonged administration so nephrotoxicity is extremely unlikely.

Adverse Effects /Caution during Emergency Anesthesia[1,2,4]

- Although, isoflurane preserves the baroreceptor response to decreased blood pressure and decreased cardiac output, patients who already have an increased heart rate may not manifest any further increases in heart rate. Most importantly, the direct vasodilatory effects of isoflurane will counter the normal hypovolemic shock-mediated increase in systemic vascular tone. Accordingly, profound hypotension may result from even low concentrations of volatile drug, and careful titration is again important.
- The safety of isoflurane in patients with coronary artery disease remains controversial because of the possibility of coronary steal syndrome. However, myocardial ischemia may be exaggerated by a large number of factors in addition to coronary vasodilatation, including tachycardia, hypotension, increase in left ventricular end-diastolic pressure and reduced ventricular compliance. Therefore attention should be directed to these factors before a diagnosis of isoflurane-induced coronary steal is considered.

Advantages[2]

- Rapid recovery
- Minimal biotransformation with little risk of hepatic or renal toxicity
- Very low risk of arrhythmias
- Muscle relaxation.

Disadvantages[2]

- A pungent odor which makes inhalation induction relatively unpleasant, particularly in children
- Coronary vasodilatation with the possibility of coronary steal syndrome at high inspired concentrations.

Desflurane

The structure of desflurane is very similar to that of isoflurane, the only difference being substitution of a fluorine atom for isoflurane's chlorine atom.

It is a colorless agent, nonflammable, pungent in odor, roughly one-fourth as potent as the other volatile agents, an ultrashort duration of action, with only 0.02% of inhaled desflurane being metabolized in the body. [4,23]

Desflurane has a high vapor pressure of 664 mm Hg at 20°C and boils at 22.8°C (73°F). To prevent boiling, desflurane is stored in special bottles with valves that open only when fitted into the filling port of a vaporizer.

Desflurane has a blood/gas partition coefficient of 0.42. Induction of anesthesia is therefore extremely rapid in theory but limited somewhat by a pungent nature. However, it is possible to alter the depth of anesthesia very rapidly and the rate of recovery of anesthesia is faster than that following any other volatile anesthetic agent.[12]

Adverse Effects/Caution during Emergency Anesthesia

- Desflurane and isoflurane appear to have very similar effects on systemic vascular resistance and arterial blood pressure. Cardiac output will be maintained or increased, but systemic vascular resistance decreases over time.
- Desflurane has been shown to cause sympathetic stimulation or hyperactivity during rapid increases in concentration of the drug. This will not significantly occur in patients with hemorrhagic shock because they already have maximum sympathetic stimulation. However, both hypertension and tachycardia does occur in euvolemic patients with desflurane, and rapid increases in concentration should be avoided in patients where this would be dangerous.
- Concentrations greater than 6%, may often lead to coughing, breath-holding and laryngospasm during gaseous induction of anesthesia.[2, 12,23]
- Desflurane, more than other volatile anesthetics, is degraded by desiccated carbon dioxide absorbent (particularly barium hydroxide lime, but also sodium and potassium hydroxide) into potentially clinically significant levels of carbon monoxide. Disposing of dried out absorbent or use of calcium hydroxide can minimize the risk of carbon monoxide poisoning.[23]

Advantages[2]

- It has low blood solubility; therefore it offers more precise control of maintenance of anesthesia and rapid recovery
- It is minimally biodegradable and therefore non-toxic to the liver and kidney
- It does not cause convulsive activity on electroencephalograph (EEG).

Disadvantages[2]

- It cannot be used for inhalation induction because of its irritant effects on the airway. Inhalational induction is associated with a high incidence of coughing and laryngospasm, especially in children
- Desflurane emergence has been associated with delirium in some pediatric patients
- It causes a tachycardia at higher concentrations
- It requires a special vaporizer. Although the TEC-6 vaporizer is reasonably easy to use, it is more complex than the more conventional vaporizers and the potential for failure may be higher
- It is expensive.

Nitrous Oxide

Nitrous oxide (N_2O; laughing gas) is the only inorganic anesthetic gas in clinical use with a low potency as an anesthetic agent. It must be delivered at nearly 0.7 atm (530 mm Hg) to ablate awareness in half of patients (ED_{50}) and at over 1 atm in most patients (ED_{95}) to prevent movement during an incision. Therefore, N_2O is frequently used in combination with other inhaled or intravenous anesthetic agents with or without opioids.

The addition of nitrous oxide decreases the requirements of other inhalational agents (70% nitrous oxide decreases the MAC of the volatile anesthetics by approximately 50%) (Table 3.1). Nitrous oxide potentiates neuromuscular blockade, but to a lesser extent than the volatile anesthetic agents:

- Nitrous oxide and its role in emergency anesthesia.[1,4,5,9,23]
  - Although nitrous oxide is insoluble in comparison with other inhalational agents, it (N_2O) will cause rapid distension of air containing spaces (due to nitrogen) and is therefore relatively contraindicated in many trauma patients or patients for emergency surgery.
  - Distension of air-filled structures occurs because the blood-gas partition coefficient of N_2O (0.47) is 34 times greater than that of nitrogen (0.014).[1] Thus, the capacity of N_2O to enter an air pocket exceeds the ability of nitrogen to exit. Pneumothorax, pneumocephalus, bowel obstruction, air embolus, or any hollow viscous injury with pneumoperitoneum will be exacerbated by the use of N_2O. Indeed, a pneumothorax can double in 10 minutes and triple in 30 minutes in the presence of 75% N_2O. Accordingly, trauma patients at risk for these injuries should not receive N_2O
  - The hemodynamic effects of N_2O are minimal. However, there is some sympathetic activation in normal patients with N_2O. At higher concentrations, N_2O is a mild myocardial depressant, a fact that may become evident in the hemodynamically compromised patient
  - Because the extent of injuries is often unknown during the initial resuscitation of the patient during emergency situation, N_2O should be avoided. It is always prudent to administer oxygen at 100% inspired concentration until the initial assessment has been completed and adequate ventilation and oxygen delivery to tissues is ensured
  - Because of the effect of N_2O on the pulmonary vasculature, it should be avoided in patients with pulmonary hypertension
  - Obviously, nitrous oxide is of limited value in patients requiring high inspired oxygen concentrations (patients in severe hemorrhagic shock, sepsis, etc.)
  - N_2O will even diffuse into tracheal tube cuffs, increasing the pressure against the tracheal mucosa
  - N_2O can support combustion and should be avoided if electrocautery is used near any distended segment of bowel containing gas.

In summary, the anesthesia provider must be ever-vigilant when administering volatile anesthetics, must communicate with the surgical team while observing the field for changes, and must be prepared to change care plan to suit the clinical conditions and to manage consequences of the agents administered.

NEUROMUSCULAR BLOCKING AGENTS

Neuromuscular blocking agents are required in emergency for two specific circumstances. They may be needed to facilitate tracheal intubation in the emergency department or prior to arrival in the hospital to provide oxygenation and ventilation to the unstable patient. Also, neuromuscular blocking agents may be needed in an otherwise stable patient as an adjunct to other anesthetic drugs for emergency surgery. In both cases, the major challenge is to choose the right drug for tracheal intubation. Neuromuscular blocking agents for maintenance of relaxation during surgery or mechanical ventilation are almost similar to those used in non-emergency cases. Finally, the indications for reversal in emergency and nonemergency cases do not differ significantly. Although many neuromuscular Blocking (NMB) drugs exist, only succinylcholine and rocuronium are recommended for rapid sequence induction (RSI) in the trauma and other emergency situations.[1,4,5,33]

Depolarizing Agents

Mechanisms of Action[4,24]

Several compounds (succinylcholine, decamethonium, imbretil) have an agonist action at the neuromuscular junction, much like the neurotransmitter acetylcholine. The mechanism of action of succinylcholine and other depolarizing agents is still poorly understood, but we know that, like acetylcholine, these drugs depolarize the muscle fiber at the endplate. However, contrary to acetylcholine, depolarizing drugs are not degraded by acetylcholinesterase. A persistent depolarization (longer than a few milliseconds) produces desensitization of acetylcholine receptors, and/or inactivation of nearby sodium channels, the net effect of which is to prevent further acetylcholine activation and/or action potential generation in the muscle cell.

Succinylcholine

The reason why succinylcholine is used clinically is not its depolarizing mechanism of action, but because it is the only neuromuscular blocking agent with rapid onset and rapid recovery.

After administration, succinylcholine first displays signs of its agonist properties at the neuromuscular junction. Disorganized muscle contractions can be observed, especially in young, muscular adult, after

injection of succinylcholine. These contractions, termed 'fasciculations', last for only a few seconds before flaccid paralysis is manifested.[24]

Uses/Indications in Emergencies[1,5,24,33]

Optimal intubating conditions occur within about 30-60 seconds of administration. This quality of producing a profound paralysis of short duration probably the most important factor in the selection of succinylcholine as the muscle relaxant of choice for RSI situations, when rapidly securing the airway is paramount for the survival of the patient. It is thus the gold standard for rapid sequence induction. The Cochrane review 2008 stated that intubation conditions provided by succinylcholine are superior to rocuronium.

Dosage[2,5,33]

Intubating conditions depend on dose. With 1.0 mg/kg, one can expect approximately 80% excellent conditions (no movement or cough). With 0.5 mg/kg, excellent conditions are found in only 50-60% of subjects. The probability of excellent conditions does not improve significantly if the dose is increased over 2 mg/kg.

Onset and Duration[2,5,24,33]

- Time to complete neuromuscular blockade (as measured at the hand muscles) after a 1 mg/kg dose is approximately one minute. Onset is shorter in children and patients with a hyperdynamic circulation and prolonged in the elderly and low-output states
- Duration of action with return of normal twitch height is 10-12 minutes.

Side Effects[2,24,33]

Frequent Side Effects

- Fasciculations, shortly after injection, are the result of the brief acetylcholine like action of succinylcholine
- Mild increase in potassium concentration (0.5 mEq/l or less) occurs as a result of potassium efflux from the cells induced by activation of cholinergic receptors
- Myalgias are muscle pains occurring 24-48 hours after succinylcholine administration.
- Arrhythmias are common, and range from bradycardia due to vagal effect, to tachycardia, which is the result of catecholamine release.

Uncommon Side Effects

- In a small proportion of susceptible individuals, contractures/fasciculations may be exaggerated and may be observed as masseter spasm, which may be of sufficient intensity to interfere with laryngoscopy and intubation
- Succinylcholine may also precipitate malignant hyperthermia. Although masseter spasm may be an early manifestation of malignant hyperthermia, most cases of masseter spasm do not lead to malignant hyperthermia.
- Severe hyperkalemia, leading to cardiac arrhythmias and asystole, may be seen after succinylcholine in patients with extensive denervation and/or muscle injury (e.g. after spinal cord injury, burns, extensive crush injuries, and muscle dystrophy)
- Patients with a genetic or acquired decrease in plasma cholinesterase activity have a prolonged response to the drug and may need to be ventilated for several hours after a usual dose of succinylcholine.

Contraindications

- Succinylcholine should not be given to patients with a documented history of malignant hyperthermia
- Extrajunctional receptors in burns, spinal cord injury, and trauma/crush injuries with extensive muscle damage take a few days to proliferate so succinylcholine should be avoided 24-48 hours after the injury. The drug is probably safe again upon resolution of the initial injury
- Hyperkalemic patients of any etiology
- Succinylcholine should not be given in patients with muscle disease, especially muscle dystrophy, in subjects who have a history of an allergic reaction to the drug, and in those with a personal history of prolonged blockade after receiving either succinylcholine or mivacurium.

Uses in Special Situations

- *In pediatric patients*, [22,24] the dose of succinylcholine is increased with decreasing age. As much as 2 mg/kg is suitable in infants. Onset and duration are shorter in pediatric patients. Whereas adults often have tachycardia after succinylcholine, bradycardia, often leading to asystole, is frequent in infants and children. Pretreatment with atropine is effective.
- *In obese individuals*[1,24-27] the same dose per kilogram actual body weight is recommended. As succinylcholine is water soluble, it has a reduced volume of distribution per kilogram, in obese patients. However, plasma cholinesterase activity is increased in obese subjects. Both effects tend to cancel out each other. Use of succinylcholine in obesity especially for bariatric surgery has been

recently debated though conclusive data is not yet available
- *In liver disease, malnutrition, and pregnancy*[24,33] plasma cholinesterase activity is decreased, requiring dose reduction of succinylcholine
- Patients with *myasthenia gravis* are resistant to the effects of succinylcholine and require more than the usual dose[33]
- In children and adults, repeat doses of succinylcholine may cause bradycardia and asystole. Pretreatment with an anticholinergic, such as atropine, 0.01 mg/kg, or glycopyrrolate, 0.004 mg/kg, is routine before administering a second dose of succinylcholine during RSI.[24,33]

Controversies[24,26,27]

- There have been sporadic reports of intractable cardiac arrests associated with hyperkalemia in otherwise healthy children (mainly male) receiving succinylcholine. These events, which have been attributed to undiagnosed muscle dystrophies, have prompted some centers to recommend a ban of succinylcholine in the pediatric population, except in cases of emergency
- A small dose of a nondepolarizing neuromuscular blocking agent given 2-4 minutes before succinylcholine is effective in the prevention of fasciculations and myalgia. This technique of'pre-curarization' has the potential to produce unpleasant symptoms if the patient is awake (diplopia, general feeling of weakness, difficulty swallowing or breathing, inability to protect airway with a possibility of pulmonary aspiration)
- Theoretical considerations and clinical studies suggest that the appropriate dose is one tenth the ED_{95} of the precurarizing drug. Larger doses, amounting to 0.2-0.4 times the ED_{95} should be avoided (as above symptoms of muscle weakness are frequent). With 0.1 times the ED_{95}, a larger dose of succinylcholine must be given (1.5-2 mg/kg) to obtain the same onset and duration characteristics as with 1 mg/kg without precurarization.

NONDEPOLARIZING NEUROMUSCULAR BLOCKING AGENTS

Mechanism of Action[2,24,33]

Nondepolarizing neuromuscular blocking agents bind to cholinergic receptors at the neuromuscular junction but do not produce activation (competitive antagonism).

Typical fade in responses can be seen with a nerve stimulator when the train-of-four (2 Hz for 2 seconds) or tetanic (30-100 Hz for 5 seconds) are used. Train-of-four or tetanic fade is not seen with depolarizing agents.

It is customary to classify neuromuscular blocking agents on the basis of their duration (discussed below).

Ultra-Short-Acting Drugs (Duration of Action 8-12 Minutes)

At present, there is no nondepolarizing neuromuscular blocking agent available for clinical use that is considered ultrashort acting.

Gantacurium[24] is an investigational compound in humans that may fit the criteria for an ultra-short-acting agent. It produces nondepolarizing blockade, its estimated ED_{95} is 0.12–0.19 mg/kg, and duration of action for twice the ED_{95} dose is less than 10 minutes in adults. It has some histamine-releasing properties. Gantacurium, if approved for clinical use, might become a succinylcholine replacement for tracheal intubation in future.

Short-Acting Drugs (Duration of Action 15-25 Minutes)

Mivacurium

Mivacurium is a benzylisoquinolone derivative that is broken down by plasma cholinesterase, like succinylcholine, but unlike succinylcholine, it produces a nondepolarizing block.

The ED_{95} of mivacurium is approximately 0.1 mg/kg in patients with normal plasma cholinesterase.[33]

The duration of action for doses in the 0.15–0.25 mg/kg range is 15-25 minutes. As with succinylcholine, decreased plasma cholinesterase activity may be associated with a blockade lasting many hours, requiring mechanical ventilation of the lungs.

Doses of 0.2-0.25 mg/kg are indicated for tracheal intubation. Infusion rates for maintenance of relaxation are 3-7 μg/kg/min and should be titrated by using a nerve stimulator.

Disadvantages

- Mivacurium has a surprisingly long onset time, with complete blockade taking 3-5 minutes after intubating doses (0.2-0.25 mg/kg). This slow onset of action has been attributed to the high potency of the drug. Thus, mivacurium is not recommended as a succinylcholine substitute for RSI.[24]
- Mivacurium has the propensity to release histamine in a dose-related fashion. At doses larger than or equal to 0.2 mg/kg, reddening of the skin, hypo-

tension, and reflex tachycardia are frequent. Rarely, bronchospasm may occur.[24,33]

Rapacuronium

Rapacuronium is a nondepolarizing agent with a steroid nucleus.

Rapacuronium enjoyed a brief moment of popularity after it was released in the United States for clinical use. However, the drug was withdrawn in 2001, a year after its introduction, because of reports of severe bronchospasm.[2]

Intermediate-Acting Drugs (Duration of Action 30-45 Minutes)

Rocuronium[1,24,28-31,33]

Rocuronium is an aminosteroid compound and its pharmacokinetics is also much like that of vecuronium, with an important redistribution phase after a bolus injection, and a slower elimination phase.

Rocuronium is actively taken up and eliminated primarily by the liver. Only part of the drug is metabolized. The rest is excreted unchanged, chiefly in the bile and to a lesser extent by the kidney.[4,5]

Elimination half-life is ~1-2 hours, but duration of action of a 2 × ED_{95} is much shorter (30-40 minutes).

ED_{95} is ~0.3 mg/kg (Rocuronium has one sixth the potency of vecuronium).[24]

Onset of action is ~1.5-2.5 minutes (two times faster than equipotent dose of vecuronium).

Duration of action is ~30-40 minutes (for 2 × ED_{95} dose). Compared with young adults, onset time and duration of action are shorter in children and longer in the elderly.

Rocuronium is also used for maintenance of relaxation by infusion at a rate of 4–8 mcg/kg/min or by bolus doses (0.1-0.2 mg/kg every 15-30 minutes).

Advantages[24,28-33]

- Rocuronium is the nondepolarizing neuromuscular blocking agent of choice for tracheal intubation when succinylcholine is contraindicated or not desired. However, compared with succinylcholine its duration of action is much longer, and this could be a major problem if tracheal intubation is not successful
- For comparable intubating conditions, the rocuronium dose must be increased to 1.0 to 1.2 mg/kg, in which case onset becomes comparable to that of succinylcholine. This resulted in advocacy of rocuronium for rapid sequence induction. (However, duration of action is increased to 60-75 minutes). The advent of Sugammadex may result in increasing use of rocuronium for rapid sequence induction particularly when difficult intubation is not anticipated
- Rocuronium is virtually devoid of cardiovascular effects, up to a dose of 1.2 mg/kg[2,5]
- Rocuronium does not trigger potassium release from muscle tissue and can be used safely in those patients (discussed previously) who may have proliferation of extrajunctional receptors
- As with other nondepolarizing NMB drugs, rocuronium does not cause fasciculations nor does it increase intracranial, intraocular or intragastric pressures.[1,24]

Vecuronium[1,4,5]

Vecuronium is a compound with a steroid nucleus (aminosteroid compound).

After a bolus injection, the drug concentration in plasma falls rapidly due to redistribution, mainly to the liver. Vecuronium also undergoes some metabolism in the liver and is partially excreted unchanged through bile and by the kidney.

Elimination half-life is ~1-2 hours. However, its duration of action is considerably less than the half-life because vecuronium is extensively redistributed.

The ED_{95} ~ 0.05 mg/kg, and doses of 0.1-0.15 mg/kg are recommended for intubation.

Maintenance doses are typically 0.4-1 µg/kg/min when given by infusion, or 0.02 mg/kg every 20-30 minutes when given as intermittent boluses.

Onset of action of vecuronium is 3-5 minutes for a 0.1 mg/kg dose. This interval can be shortened by increasing the dose, but at the expense of a prolonged duration of action.

Duration of action is heavily dose-dependent, ranging from 30-40 minutes after 0.1 mg/kg to 50-70 minutes after 0.15 mg/kg in young adults. Duration of action is shorter in children and longer in elderly, patient with hepatic failure or renal failure.

Fortunately, vecuronium is virtually devoid of cardiovascular side effects, even at doses as high as 0.4 mg/kg can be safely used even in hemodynamically unstable patients.

Disadvantages

- Long onset of action makes vecuronium a poor choice for RSI[1]
- Vecuronium has minimal effects on heart rate and blood pressure and is a good choice for patients with myocardial ischemia (where heart rate elevation is detrimental). However, in the trauma setting, drugs with vagolysis (pancuronium, rocuronium) are generally well tolerated[1]

- Vecuronium metabolite, 3-desacetylvecuronium has neuromuscular blocking properties at approximately half the potency of vecuronium and may account for the prolonged block when vecuronium is given as a continuous infusion in the ICU.[5,33]

Atracurium[1,2,24,32,33]

Atracurium is a benzylisoquinoline compound, like mivacurium. However, unlike mivacurium, it does not depend on plasma cholinesterase for its breakdown.

Metabolism is via two pathways, "Hoffmann elimination" (a pH and temperature dependent nonenzymatic breakdown) and ester hydrolysis (by nonspecific esterases).

Atracurium's unique mode of elimination provides a duration of action that is relatively independent of the function of traditional organs of elimination, such as the kidney and the liver.

Elimination half-life is ~20 minutes, and is independent of age, end-organ function, and weight.

The ED_{95} ~0.2-0.25 mg/kg, and recommended doses for intubation are 0.4-0.5 mg/kg.

Duration of intubating doses is 35-45 minutes.

It may be used for maintenance of anesthesia. The infusion rate is 3-7 µg/kg/min, or the equivalent in repeated bolus doses (0.1 mg/kg every 15–30 minutes).

Disadvantages

- Like mivacurium, atracurium releases histamine in a dose-related manner, with doses of 0.5 mg/kg and greater being associated with hypotension and tachycardia.[33]
- Hoffmann elimination and ester hydrolysis both produce an end-product called laudanosine, which is eliminated by the kidneys and has been found to produce seizures in high concentrations. However, doses of atracurium normally required for anesthesia and surgery are not high enough to lead to laudanosine seizures
- Onset time is longer than for succinylcholine (3-4 minutes). Thus, atracurium is not recommended for RSI. Timing principle may help in achieving intubating conditions fit for rapid sequence intubation for emergency anesthesia.

Cisatracurium[1,2,5,24,32,33]

Cisatracurium is one of the most potent isomers of atracurium. Hoffmann elimination and ester hydrolysis; both contribute to the degradation of cisatracurium.

The elimination half-life ~20-25 minutes, and is independent of the patient's organs of elimination.

The ED_{95} ~0.05 mg/kg, (histamine is not released unless the dose exceeds 0.4 mg/kg).

Onset of action is long ~5-7 minutes for a 2 × ED_{95} dose (0.1 mg/kg). Onset of action can be made shorter if the dose is increased to 0.15 or 0.2 µg/kg, at the expense of prolonged blockade.

Duration of action increases from 40-45 minutes with 0.1 mg/kg to 60-75 minutes with 0.2 mg/kg.

Infusion rates of 0.5-1.2 µg/kg/min or incremental bolus doses of 0.02 mg/kg every 15-20 minutes are recommended.

Advantages over atracurium are its increased potency without an increase in the threshold for histamine side effects. Moreover, as the amount of drug administered is less than in the case of atracurium, the amount of laudanosine produced is less, and this virtually eliminates concerns with the seizure-producing effects of laudanosine.

Long-Acting Drugs (Duration of Action > 60 minutes, usually 90-120 minutes)

Long-acting drugs have fallen into disfavor not because of their side effects, but because they tend to be associated with a high incidence of residual paralysis.

Representatives of the old group are d-tubocurarine, gallamine, pancuronium, alcuronium, and fazadinium fall in this class. Their use is now historical.

Long-acting drugs should be given only to patients who are likely to have their lungs ventilated postoperatively.

Pancuronium[1,24,33] is not recommended for tracheal intubation in emergency (RSI). Even modest doses produce hypertension and tachycardia. The ED_{95} is 0.07 mg/kg. Elimination half-life and duration of action are thus similar (1-2 hours). Maintenance doses are 0.01-0.02 mg/kg every 30-60 minutes.

Doxacurium though devoid of cardiovascular side effects, because of its high potency, onset time is extremely slow (7-10 minutes), making doxacurium useless for tracheal intubation.

ANTAGONISM OF RESIDUAL NEUROMUSCULAR BLOCKADE

Cholinesterase Inhibitors[1,5,7,24,31-33]

These agents inhibit the action of acetylcholinesterase at the neuromuscular junction, thus prolonging the half-life of acetylcholine and potentiating its effect, especially in the presence of residual amounts of non-depolarizing muscle relaxant at the end of surgery.

Commonly used cholinesterase inhibitors are neostigmine, edrophonium, pyridostigmine, and physostigmine.

The time required to fully reverse a nondepolarizing block depends on several factors such as choice and dose of cholinesterase inhibitor administered (edrophonium > neostigmine > pyridostigmine), the muscle relaxant being antagonized, rate of spontaneous recovery from the neuromuscular blocker, the extent of the blockade before reversal and concentration of inhaled anesthetic agent present during reversal.

In routine clinical practice, upon return of the TOF-response to a single twitch, adequate recovery from neuromuscular blockade to allow successful extubation usually is present within 20 minutes.

A muscarinic antagonist is simultaneously administered to limit any untoward effects of acetylcholine (bradycardia, bronchospasm, or gastrointestinal hyperactivity). Because the anticholinesterase and muscarinic antagonist should have similar durations, the usual drug pairs that are commonly given are neostigmine/glycopyrrolate or edrophonium/atropine.

Neostigmine

This drug combines reversibly with acetylcholinesterase by the formation of an ester linkage, and is excreted largely unchanged through the kidney.

The maximum effective dose is 60 to 80 µg/kg. The effects of neostigmine (40-60 µg/kg) are usually apparent in 5-10 min, peak at 10 min, and last more than 1 hour.

The onset of action of glycopyrrolate (0.2 mg glycopyrrolate per 1 mg of neostigmine) is similar to that of neostigmine and is associated with less tachycardia than is experienced with atropine (0.4 mg of atropine per 1 mg of neostigmine).

Renal failure decreases the plasma clearance of neostigmine.[33]

During profound (<3% twitch recovery) mivacurium-induced blockade, the administration of neostigmine may prolong recovery as it inhibits butyrylcholinesterase (the enzyme responsible for metabolism of mivacurium).[1,24,33]

Uses

- Antagonism of residual neuromuscular blockade
- Treatment of myasthenia gravis, urinary bladder atony, and paralytic ileus.
- Neostigmine (50-100 µg) has been used as an adjunct to intrathecal anesthesia for prolongation of sensory and motor blockade.

Precautions

- Bronchospasm may be precipitated—hence extreme caution while using in asthmatics
- Bradycardia may be potentiated by vagal stimulation, e.g. suction, pressure on the eye, etc.

Edrophonium[1,4,5,24,33]

Edrophonium has the most rapid onset of action (1-2 min) and the shortest duration of action among all cholinesterase inhibitors. Low doses should not be used, because longer-acting muscle relaxants may outlast the effects of edrophonium although higher doses prolong the duration of action to more than 1 hour.[33]

Edrophonium is less than 10% as potent as neostigmine.

The recommended dosage is 0.5-1 mg/kg.

Edrophonium is not as effective as neostigmine at reversing intense neuromuscular blockade, but is more effective in reversing a mivacurium blockade. It is a less potent inhibitor of butyrylcholinesterase so has little effect on the metabolism of mivacurium. 0.3 to 0.5 mg/kg of edrophonium will accelerate recovery from mivacurium.

Atropine is better suited for administration with the rapid-acting edrophonium, 7 to 10 µg/kg of atropine should be given with 0.5 to 1.0 mg/kg of edrophonium.[33]

In equipotent doses, muscarinic effects of edrophonium are less pronounced than those of neostigmine or pyridostigmine, requiring only half the amount of anticholinergic agent. Edrophonium's rapid onset is well matched to that of atropine (0.014 mg of atropine per 1 mg of edrophonium). Although glycopyrrolate (0.007 mg per 1 mg of edrophonium) can also be used, it should be given several minutes prior to edrophonium to avoid the possibility of bradycardia.

75% of the excretion of edrophonium occurs through kidney so renal failure decreases the plasma clearance of edrophonium.

Physostigmine[24,33]

It is lipid soluble and is the only clinically available cholinesterase inhibitor that freely passes the blood–brain barrier, dose being 0.01-0.03 mg/kg, completely metabolized by plasma esterases.

The lipid solubility and CNS penetration of physostigmine limit its usefulness as a reversal agent for nondepolarizing blockade.

However its other clinical uses are:

- Treatment of central anticholinergic toxicity caused by overdoses of atropine or scopolamine

- Reversal some of the CNS depression and delirium associated with use of benzodiazepines and volatile anesthetics
- Preventing postoperative shivering. (dose - 0.04 mg/kg)
- Partially antagonizes morphine-induced respiratory depression.

These effects are transient, and repeated doses may be required.

Bradycardia is infrequent in the recommended dosage range, but atropine or glycopyrrolate should be immediately available. Because glycopyrrolate does not cross the blood–brain barrier, it will not reverse the CNS effects of physostigmine.

Side Effects of Cholinesterase Inhibitors

- Bradycardia, salivation, sweating, bronchospasm, increased intestinal motility and blurred vision. These effects can be blocked by using atropine or glycopyrrolate
- Nausea and vomiting.

Sugammadex (ORG 25969, Modified γ-cyclodextrin)[5,24,28,31,33]

It is the first selective relaxant binding agent.

Sugammadex exerts its effect by forming very tight complexes in a 1:1 ratio with steroidal neuromuscular blocking agents (rocuronium > vecuronium > pancuronium).

Mechanism of action

- During rocuronium-induced neuromuscular blockade, intravenous administration of sugammadex results in rapid removal of free rocuronium molecules from plasma
- Also there is movement of the remaining rocuronium molecules from the neuromuscular junction back into plasma, where they are encapsulated by free sugammadex molecules
- Sugammadex also enter tissues and form a complex with rocuronium.

After the administration of sugammadex, the plasma concentration of free rocuronium decreases rapidly, but the total plasma concentration of rocuronium (both free and that bound to sugammadex) increases.

The soluble nature of the rocuronium-cyclodextrin complex makes urinary excretion of this complex the major route of elimination of rocuronium.

Advantages:

- Sugammadex causes a rapid and efficient reversal of rocuronium-induced neuromuscular blockade. With sugammadex (0.5 and 1.0 mg/kg) the recovery from rocuronium induced neuromuscular blockade ocurrs within 2-5 minutes without signs of residual blockade or re-curarization[31,33]
- Although the duration of paralysis produced by rocuronium is very much longer, the use of sugammadex as a reversal agent makes recovery from an NMBD as quick as that from succinylcholine and more predictable. This can be of utmost advantage in cases of unanticipated difficult airway managment situations. Thus, Sugammadex could solve the problems of residual paralysis and failed intubation
- It has no effect on acetylcholinesterase or any receptor system in the body, thus eliminating the need for anticholinergic drugs and their undesirable side effects[33]
- It has no cardiovascular or other major systemic effects so far.

Disadvantages and Side effects:

- Sugammadex is ineffective against succinylcholine and benzylisoquinolinium neuromuscular blockers such as mivacurium, atracurium, and cisatracurium as it cannot form inclusion complexes with these drugs
- Mild hypotension, coughing, nausea, vomiting, dry mouth, parosmia (an abnormal sense of smell), a sensation of a changed temperature are frequent side effects.

OPIOIDS

The opioids are quite useful in unstable or head-injured emergency patients because they do not cause direct myocardial depression, direct vasodilation, or cerebral vasodilation. However, sympathetic output may be decreased following opioid administration leading to a decrease in mean arterial pressure (MAP), heart rate (HR), and cardiac output. Infusions, as opposed to intermittent boluses, are frequently employed for TIVA and are believed to confer the benefits of achieving a more reliable steady-state, a lower total-dose administered, less side effects, hemodynamic stability, and a more rapid recovery.

Mechanisms of Action[1,34,35]

Opioids produce their actions at a cellular level by activating opioid receptors. These receptors are distributed throughout the central nervous system (CNS) with high concentrations in the nuclei of tractus solitarius, peri-aqueductal grey area (PAG), cerebral cortex, thalamus and substantia gelatinosa (SG) of the spinal cord.

Although both opioid agonists and antagonists bind to opioid receptors, only agonists are capable of receptor

activation. Opiate–receptor activation inhibits the presynaptic release and postsynaptic response to excitatory neurotransmitters (e.g. acetylcholine, substance P, etc.) from nociceptive neurons**.** Overall, the effect is a reduction in neuronal cell excitability that in turn results in reduced transmission of nociceptive impulses.

Pure opioid agonists (morphine, diamorphine, pethidine and fentanyl) bind to opioid receptors avidly and demonstrate high intrinsic activity at the cellular level as described above. Partial opioid agonists (buprenorphine, pentazocine), bind to opioid receptors but produce a sub-maximal effect compared to pure agonists. Opioid antagonists (naloxone, naltrexone), have receptor affinity but no intrinsic activity.

Pharmacokinetics

There is substantial variability (3-5 fold) in the clinical response to opioids due to their pharmacokinetics and pharmacodynamics. Pharmacokinetic properties like pKa, lipid solubility, unionized fraction, protein binding in the plasma and volumes of distribution are shown in Table 3.3.

Effects on Organ Systems

Cardiovascular: In general, opioids do not seriously impair cardiovascular function.

- Mild bradycardia is common as a result of decreased sympathetic drive and a direct effect on the sino-atrial (SA) node
- Peripheral vasodilatation caused by histamine release (morphine and pethidine) and reduced sympathetic drive may result in a fall in blood pressure that may be significant in hypovolemic patients. The effects of histamine release can be minimized in susceptible patients by slow opioid infusion, adequate intravascular volume, or pretreatment with H_1 and H_2 histamine antagonists
- The combination of opioids with other anesthetic drugs (e.g. nitrous oxide, benzodiazepines, barbiturates, volatile agents) can result in significant myocardial depression.

Respiratory

- Opioids depress ventilation (mediated by μ receptors in brain). In women > men
- Respiratory rate falls more than the tidal volume and the sensitivity of the brain stem to carbon dioxide is reduced. Resting $PaCO_2$ increases
- The apneic threshold—the highest $PaCO_2$ at which a patient remains apneic—is elevated, and hypoxic drive is decreased
- Concurrent use of other CNS depressants, for example benzodiazepines or halogenated anesthetic, may cause marked respiratory depression
- Opioids (particularly fentanyl, sufentanil, and alfentanil) can induce chest wall rigidity severe enough to prevent adequate ventilation. This centrally mediated muscle contraction is most frequent after large drug boluses and is effectively treated with neuromuscular blocking agents
- Opioids can effectively blunt the bronchoconstrictive response to airway stimulation during intubation.

Cerebral

- Opioids cause dose dependent cerebral depression. However, unlike the barbiturates or benzodiazepines, relatively large doses of opioids are required to render patients unconscious.
- Opioids reduce cerebral oxygen consumption, cerebral blood flow, and intracranial pressure, but to a much lesser extent than barbiturates or benzodiazepines.
- Regardless of the dose, however, opioids do not reliably produce amnesia.
- Analgesia: Opioids are most effective in relieving dull, continuous and poorly localized pain arising from deeper structures, for example the gut. They are less effective against superficial and sharp pain.

Table 3.3: Pharmacokinetic characterstics of commonly used opioids

Characteristic	*Morphine*	*Pethidine*	*Fentanyl*	*Alfentanil*	*Remifentanil*
pKa	8.0	8.5	8.4	6.5	7.1
Unionized at pH 7.4 (%)	23	5	9	90	68
Protein bound (%)	30	40	84	90	70
Terminal half life (hr)	3	4	3.5	1.6	0.06
Clearance (ml/min/kg)	15-30	8-18	0.8-1.0	4-9	30-40
Distribution(L/kg)	3-5	3-5	3-5	0.4-1.0	0.2-0.3
Relative lipid solubility	1	28	580	90	50

- Sedation: Drowsiness, feeling of heaviness and difficulty in concentrating are common. Sleep may occur with relief of pain, although they are not true hypnotics.
- Euphoria and dysphoria: Morphine and other opioids cause a sense of contentment and well-being (euphoria). If there is no pain, morphine may cause restlessness and agitation (dysphoria).
- Hallucination: These are more common with kappa agonists, but morphine and other μ-agonists may also cause hallucinations.
- Tolerance and dependence: Tolerence and physical dependence are significant problem associated with repeated opioid administration. Tolerance is the decrease in effect seen despite maintaining a given concentration of a drug. The mechanism is not fully understood but could involve down regulation of opioid receptors or decreased production of endogenous opioids. Dependence exists when the sudden withdrawn of an opioid, after repeated use over a prolonged period, results in various physical and psychological signs. These include; restlessness, irritability, increased salivation, lacrimation and sweating, muscle cramps, vomiting and diarrhea.
- Stimulation of the medullary chemoreceptor trigger zone is responsible for a high incidence of nausea and vomiting.

Gastrointestinal

Gastric emptying is delayed due to reduced gut motility.

Biliary colic may result from opioid-induced contraction of the sphincter of Oddi (effectively reversed with the pure opioid antagonist naloxone).

Endocrine

Opioids block the release of catecholamines and other stress related hormones more completely than volatile anesthetics. This is particularly true of the more potent opioids such as fentanyl, sufentanil, alfentanil, and remifentanil. In particular, patients with ischemic heart disease may benefit from attenuation of the stress response.

Secretion of antidiuretic hormone (ADH) is increased.

Histamine Release and Itching

Some opioids (morphine, pethedine) cause histamine release from mast cells resulting in urticaria, itching, bronchospasm and hypotension.

Itching occurs most often after intrathecal opioids and is more pronounced on the face, nose and torso. Mechanism is centrally mediated and may be reversed by naloxone.

Muscle Rigidity

Large doses of opioids (particularly fentanyl, sufentanil, and alfentanil) may occasionally produce generalized muscle rigidity especially of thoracic wall and interfere with ventilation.

Immunity

The immune system is depressed after long-term opioid abuse.

Effects on Pregnancy and Neonates

All opioids cross the placenta and if given during labor, can cause neonatal respiratory depression.

Chronic use by the mother may cause physical dependence *in utero* and lead to a withdrawal reaction in the neonate at birth that can be life, threatening.

There are no known teratogenic effects.

Drug Interactions

Barbiturates, benzodiazepines, and other central nervous system depressants can have synergistic cardiovascular, respiratory, and sedative effects with opioids.

The biotransformation of alfentanil, but not sufentanil, may be impaired following a 7-day course of erythromycin, leading to prolonged sedation and respiratory depression.

Contraindications and Cautions

Contraindications are—hypersensitivity to opioids; diarrhea caused by poisoning until toxins are eliminated; during labor or delivery of a premature infant (may cross immature blood brain barrier more readily); after biliary tract surgery or following surgical anastomosis; pregnancy; labor (respiratory depression in neonate; may prolong labor).

Use cautiously with head injury and increased intracranial pressure; acute asthma, COPD, cor pulmonale, pre-existing respiratory depression, hypoxia, hypercapnia (may decrease respiratory drive and increase airway resistance); lactation (wait 4-6 hr after administration to nurse the baby); acute abdominal conditions, CV disease, supraventricular tachycardias, myxedema, seizure disorders, acute alcoholism,

delirium tremens, cerebral arteriosclerosis, ulcerative colitis, fever, kyphoscoliosis, Addison's disease, prostatic hypertrophy, urethral stricture, recent gastrointestinal or genitourinary surgery, toxic psychosis, renal or hepatic dysfunction.

INDIVIDUAL OPIOIDS

Fentanyl[34,35]

Fentanyl is a synthetic phenylpiperidine derivative. It is 100 times more potent than morphine.

Extremely fat soluble (500 times more fat soluble than morphine), with rapid onset and short duration of action. The low molecular weight and high lipid solubility of fentanyl also allow transdermal absorption (the fentanyl patch). The amount of fentanyl released depends primarily on the surface area of the patch but can vary with local skin conditions (e.g. blood flow). Serum concentrations of fentanyl reach a plateau within 14-24 hours of application and remain constant for up to 72 hours.

Fentanyl is predominantly metabolized in the liver to norfentanyl which is inactive. The metabolite is excreted in the urine over few days.

Uses

Fentanyl produces minimal hemodynamic effects and is characterized by a rapid onset of sedation and analgesia, a relatively short duration of action (approximately 30 to 40 minutes), and rapid reversal with opiate antagonists. These properties make fentanyl and ideal drug for emergency department use

- When given in small doses (1-2 µg/kg), it has rapid onset and short duration of action (30 minutes). Peak effect after IV administration is in only 3-5 minutes. Such doses are used intravenously for pain associated with minor surgery. In small doses it has little sedative effect
- Higher doses (2-5 µg/kg) are used to obtund sympathetic response to laryngoscopy and intubation
- High-dose fentanyl (50 to 150 µg/kg) has been extensively used for anesthesia induction, particularly in cardiac surgery
- Fentanyl has been used to augment effects of local anesthetics in spinal and epidural analgesia at 10-25 µg and 25-100 µg doses respectively.
- Fentanyl is also available as transdermal patch for chronic pain conditions and as lollipop to premedicate the children. Experimental studies have explored the possibility of an inhalation delivery of liposome-encapsulated fentanyl.

Alfentanil[34-37]

Alfentanil is synthetic phenylpiperidine derivative structurally related to fentanyl; it has 10-20% of its potency and has an extremely rapid onset and short duration of effect with lower lipid solubility and high protein binding.

Peak analgesic and ventilatory depressant effects occur in less than 2 minutes.

Uses

Induction of anesthesia: High dose alfentanil has been used as an induction agent for patients with and without cardiac disease. It can be used alone or in combination with other drugs. In healthy patients, doses of about 120 µg/kg produce unconsciousness in 2 to 2.5 minutes, Premedication with a benzodiazepine is associated with a lower dose requirement, 40 to 50 µg/kg, and a faster onset of unconsciousness, within 1.5 minutes, but may also produce hypotension.

When high-dose alfentanil is used as the sole anesthetic agent, a range of infusion rates (0.05-2.0 µg/kg/min) are used during maintenance of anesthesia with controlled ventilation.

Adjunct for rapid sequence induction: To improve intubating conditions and hyperdynamic response.

Analgesia: In short surgical procedures (outpatient surgery), especially those associated with minimal postoperative pain, alfentanyl is used in loading dose of 5 to 10 µg/kg provides good analgesia with rapid recovery.

For longer procedures, alfentanil can be administered as needed in repeated small bolus doses, but its pharmacokinetic properties make it ideal for administration as a continuous infusion.

Sufentanil[34,35]

Sufentanil is closely related in structure to fentanyl (five to 10 times more potent), extremely lipophilic, with high degree of plasma protein binding and smaller degree of ionization at physiologic pH.

Plasma sufentanil concentration drops very rapidly after bolus intravenous dose, and 98% of the drug is cleared from plasma within 30 minutes.

Uses

- During induction, loss of consciousness is seen with total doses between 1.0-3.0 µg/kg
- Maintenance of anesthesia can be achieved with N_2O (60 to 70%) in O_2 and additional sufentanil

(intermittent boluses of 0.1 to 0.25 µg/kg or a constant infusion of 0.5 to 1.5 µg/kg/hr).
- Doses in the range of 0.3 to 1.0 µg/kg given 1 to 3 minutes prior to laryngoscopy blunts hemodynamic responses to intubation, but muscle rigidity can occur, particularly in the elderly, even at these lower doses
- Much higher bolus doses (10 µg/kg) and/or infusion rates (0.15 µg/kg/min) are required to achieve the plasma sufentanil concentration range of 6-60 ng/ml required during cardiac anesthesia using sufentanil as the sole agent.

Remifentanil[34,35,37]

Although chemically related to the fentanyl congeners, Remifentanil is structurally unique because of its ester linkages which are susceptible to rapid metabolism by plasma and tissue esterases.

Remifentanil is rapidly broken down by nonspecific plasma and tissue esterases resulting in a short elimination half-life (3-10 minutes). It is context insensitive, in that the half life, clearance and distribution are independent of duration and strength of infusion. It does not accumulate with repeated dosing or prolonged infusion.

Bolus doses produced a peak analgesic effect between 1-3 minutes. Duration of action is -10 minutes.

Because of its extremely short duration of action, remifentanil is best administered as a continuous infusion, although administration as repeated bolus doses has also been reported to be effective.

Uses

Certain properties of remifentanil like rapid onset, rapid offset, organ independent metabolism and lack of accumulation make it suitable for use during various surgical procedures. However, it should be used cautiously at higher rates of infusion as serious side effects like bradycardia, hypotension, apnea and muscle rigidity may occur.

- *Induction Dosage, Intubation, LMA Placement:*
  - Remifentanil alone has not been found to be a satisfactory single agent for induction of anesthesia because of unreliability in loss of consciousness as well as significant muscle rigidity. Combined with a potent inhalation agent, a loading dose of 1 µg/kg given over 60 seconds can provide adequate intubating conditions with hemodynamic stability
  - The most commonly used remifentanil-based regimen for anesthetic induction and laryngoscopy consists of remifentanil 0.5-1 µg/kg given over 60 seconds plus propofol 1-2 mg/kg, followed by remifentanil infusion at the rate of 0.25-0.5 µg/kg/min.
  - For high-dose opioid anesthesia for cardiac surgery, the remifentanil infusion is maintained at 1-3 µg/kg/min.
- *Monitored Anesthesia Care (MAC):* When used as an adjunct for sedation or analgesia during regional anesthesia, or for block placement, as part of MAC, a much lower maintenance infusion rate of 0.05-0.1 µg/kg/min provides adequate sedation and analgesia.

Disadvantages

The ultrashort duration of this analgesic can be a drawback and patients may experience substantial pain on emergence from anesthesia. Thus, if moderate to severe postoperative pain is anticipated, continuing the remifentanil infusion between 0.05-0.15 µg/kg/min ensures adequate analgesia in most patients or patients who are expected to have postoperative pain should receive a longer-acting opioid prior to stopping remifentanil.

Tramadol[34,35]

It is weak agonist at all opioid receptors with 20-fold preference for µ receptors. It inhibits neuronal reuptake of norepinephrine. It potentiates release of serotonin and causes descending inhibition of nociception.

Tramadol has high oral bioavailability of 70% which can increase to 100% with repeated doses due to reduction in first pass effect. It is 20% bound to plasma proteins. It is metabolized in the liver by demethylation into a number of metabolites, only one of them (O-desmethyltramadol) has analgesic activity.

Its volume of distribution is 4.0 liter/kg and its elimination half-life is 4-6 hours. Dose: 1-2 mg/kg IV/or IM.

In equianalgesic dose to morphine, respiratory and cardiovascular depression produced by tramadol is not significant. However, tramadol shares most of the common side effects of other opioids (e.g. vomiting, drowsiness and ambulatory dizziness). Tramadol is contraindicated in patients on MAOI or with a history of epilepsy.

Nalbuphine[34,35,38]

This phenanthrene opioid derivative, acts as an antagonist or weak partial agonist at the µ-receptor and as an agonist at the κ-receptor.

The onset of action is rapid (5 to 10 minutes), and its duration is long (3 to 6 hours) because of an extended plasma elimination half-life (5 hours).

Uses

Premedication: Nalbuphine (0.1 mg/kg) as a premedicant results in sedation, pain relief, and respiratory depression without causing any significant hemodynamic changes.

Analgesia: Nalbuphine can be administered as an analgesic suppliment for conscious sedation or balanced anesthesia and as an analgesic for postoperative and chronic pain problems. While nalbuphine has ceiling analgesic and respiratory depressant effects, they can be as effective as full μ agonists in providing postoperative analgesia.

Nalbuphine has been used to antagonize the respiratory depressant effects of full agonists while still providing analgesic effects, e.g. Nalbuphine is effective in antagonizing fentanyl-induced respiratory depression following high-dose (100 to 120 μg/kg) fentanyl anesthesia for cardiac surgery.

Nalbuphine (4 mg IV) is as effective as ondansetron (4 to 8 mg IV) for the prevention of intrathecal morphine–induced pruritus after cesarean delivery.

Nalbuphine provides a rapid and potent antishivering effect, similar to meperidine though there are trials which do not support this effect.

Disadvantage: Nalbuphine lacks the ability to attenuate cardiovascular and hormonal responses to tracheal intubation and surgical procedures.

Pentazocine[34,35,38]

Pentazocine is one half to one fourth as potent as morphine. Pentazocine is an agonist at the kappa and delta receptors, but it is also a partial agonist or antagonist at the μ-receptor.

The analgesia produced by pentazocine is primarily related to stimulation of the κ-receptor. Ceiling effect in both analgesia and respiratory depression occur after 30 to 70 mg of pentazocine.

Pentazocine depresses myocardial contractility and increases arterial blood pressure, heart rate, systemic vascular resistance, pulmonary artery pressure, and left ventricular work index secondarily by increasing blood catecholamine levels. Because these changes are likely to elevate myocardial oxygen consumption, pentazocine may be a poor choice for patients with ischemia or infarction.

Pentazocine has limited application because it is associated with a high incidence of postoperative nausea and vomiting, provides limited analgesia, partially antagonizes other opioids, and can produce undesirable cardiovascular and psychotomimetic effects.

Butorphanol[34]

Butorphanol is an agonist at κ-receptors and either antagonistic or partially agonistic at μ-receptors. It is five to eight times as potent as morphine and is available only in parenteral form.

After parenteral administration, the onset of effect is rapid, and peak analgesia occurs within 1 hour. The duration of action of butorphanol is similar to that of morphine; its plasma half-life is only 2 to 3 hours.

Side effects after butorphanol include drowsiness, sweating, nausea, and CNS stimulation. In healthy volunteers, butorphanol (0.03 or 0.06 mg/kg IV) produces no or minimal cardiovascular changes. However, in patients with cardiac disease, butorphanol causes significant increases in cardiac index, left ventricular end-diastolic pressure, and pulmonary artery pressure.

Acute biliary spasm can occur after butorphanol, but increases in biliary pressure are less than after equipotent doses of fentanyl or morphine.

Transnasal butorphanol is effective in relieving migraine and postoperative pain.

Butorphanol is indicated for use as a sedative and in treatment of moderate postoperative pain A dose as low as 0.5 mg can provide clinically useful sedation, while single analgesic doses range from 0.5 to 2 mg.[38]

Buprenorphine[34,35,38]

Buprenorphine is a μ-receptor partial agonist but approximately 33 times more potent to morphine.

The onset of action of buprenorphine is slow, its peak effect may not occur until 3 hours, and its duration of effect is prolonged (<10 hours). Buprenorphine has higher affinity for μ-receptor and takes much longer (half-life of 166 minutes) to dissociate from it. Because of its high affinity, it is difficult to reverse the effects with naloxone.

The cardiovascular effects of buprenorphine are similar to those of morphine. Buprenorphine has been used successfully for premedication (0.3 mg IM), as the analgesic component in balanced anesthesia (2 to 6 μg/kg), and for postoperative pain control (0.3 mg IM).

Buprenorphine, like the other agonist-antagonist compounds, is not acceptable as a sole anesthetic, and its receptor kinetic profile restricts its usefulness if other μ agonists are used. Opioid withdrawal symptoms develop slowly (5 to 10 days) when buprenorphine is discontinued after long-term use.

Buprenorphine can be effective in treatment of moderate to severe pain. A single dose of 0.3 to 0.4 mg appears to produce analgesia equivalent to 10 mg morphine.

Morphine Sulphate[34,35,38]

Morphine is a naturally occurring phenanthrene derivative. It is considered a standard drug against which all other opioids are compared. Morphine is highly hydrophilic (lowest lipid solubility), with poor penetration across the blood-brain barrier.

Parenteral dose is 0.01-0.2 mg/kg and onset of action is within 5 minutes.

Peak effects may be delayed for 10 to 60 minutes (main factor responsible for its latency being low lipid solubility and slow penetration of blood brain barrier).

Morphine is extensively metabolized by the gut wall and the liver to morphine-3-glucuronide (M3G) (70%), morphine-6-glucuronide (M6G) (10%) and to sulphate conjugates. M6G is 10-20 times more potent than morphine and is normally excreted in urine. It accumulates in renal failure and accounts for increased sensitivity to morphine seen in such patients.

Neonates are more sensitive than adults to morphine due to reduced hepatic conjugating capacity. In the elderly, owing to reduced volume of distribution, peak plasma levels of morphine are higher.

Uses

- Mainly used as a premedicant and for postoperative analgesia, and less often as a component of balanced or high-dose opioid anesthesia
- As a balanced anesthetic technique with N_2O—upto 3 mg/kg
- Combined with inhalation agents—1 to 2 mg/kg
- Treatment of acute myocardial infarction and dyspnea associated with acute left ventricular failure and pulmonary edema.
- Adjunct in central neuraxial blockade: Morphine may be given epidurally at 10% and intrathecally at 1% of the parenteral dose.

Advantage

There is remarkable hemodynamic stability with minimal cardiovascular effects when morphine is used as an induction agent.

Disadvantages

- Awareness under anesthesia is a risk if used as a sole anesthesia induction agent
- Delay in its action makes morphine more difficult to titrate as an anesthetic supplement than the more rapidly acting opioid
- Epidural, intrathecal or caudal morphine causes delayed respiratory depression, nausea, vomiting, pruritus, urinary retention.

Precautions

- Reduced dose required in elderly, hypovolemic, high risk surgical patients or concomitant use of other sedatives/ narcotics
- Morphine has active metabolites (M6G) that are dependent on kidney for elimination hence morphine may not be a good choice in patients with severely altered renal clearance mechanisms.

Naloxone[34,35,38]

Naloxone is a pure opioid agonist and will reverse opioid effects at μ, kappa and delta receptors, although its affinity is highest at μ-receptors. It is the drug of choice for the treatment of opioid induced respiratory depression.

Dosage

The usual dose is 200-400 μg intravenously, titrated to effect.

Smaller doses (0.5-1.0 μg/kg) may be titrated to reverse undesirable effects of opioids for example itching associated with the intrathecal or epidural administration of opioids, without significantly affecting the level of analgesia.

The duration of effective antagonism is limited to around 30 minutes and therefore longer acting agonists will outlast this effect and further bolus doses or an infusion (5-10 μg/kg/hr) will be required to maintain reversal.

Caution must be used in opioid addicts as giving naloxone may cause an acute withdrawal state with hypertension, pulmonary edema and cardiac arrhythmias.

Antanalgesic effects may be observed in opioid naïve subjects who are given naloxone.

BENZODIAZEPINES

Benzodiazepines have in common four basic pharmacological properties, i.e. anxiolytic, sedative-hypnotic, muscle relaxant, and anticonvulsant. For anesthesiology, the main interest lies in their anxiolytic and sedative-hypnotic properties because of their application in premedication preceding anesthesia and surgery and for anesthesia induction itself.

Practically all effects of the benzodiazepines result from their actions on the ionotropic $GABA_A$ receptors in the central nervous system.

Of the four benzodiazepines, widely used in clinical anesthesia, the agonists' midazolam, and diazepam and the antagonist flumazenil are metabolized by cytochrome P450 (CYP) enzymes and by glucuronide conjugation whereas lorazepam directly undergoes glucuronide conjugation.

In addition to pharmacokinetic interactions, benzodiazepines have synergistic interactions with other hypnotics and opioids.

Midazolam[4,24,39]

Midazolam is a water-soluble benzodiazepine that is available in an acidified (pH-3.5) aqueous formulation that produces minimal local irritation after IV or intramuscular (IM) injection. It has anxiolytic, amnestic, sedative, hypnotic, anticonvulsant, and spinally mediated muscle relaxant properties.

Midazolam is 1.5-2 times more potent than diazepam. Onset of sedation is in 90 seconds and peak effect at 2-5 minutes.[2]

Dosage and Administration

Intravenous Sedation: Midazolam (0.02 to 0.08 mg/kg IV/IM) is used for sedation as preoperative premedication, intraoperatively during regional or local anesthesia, and postoperatively. Peak effect is reached within 2 to 3 minutes of administration. The duration of action of these drugs depends primarily on the dose used.

Oral Sedation: Midazolam, 0.2 to 0.8 mg/kg administered orally 10 to 15 minutes before parental separation acts rapidly, providing reliable amnesia within 10 minutes and rendering children effectively sedated for anesthesia induction.

Induction and maintenance of anesthesia: Midazolam can be used to induce anesthesia due to its relatively short onset time after IV administration. Significant hypnotic synergism occurs when midazolam and opioid analgesics with or without other hypnotics are administered in combination.

The usual induction dose of midazolam in premedicated patients is 0.05 to 0.2 µg/kg IV, with infusion rates of 0.25 to 1 mcg/kg/min required to maintain hypnosis and amnesia in combination with inhalational agents and/or opioid analgesics. When midazolam is used with other anesthetic drugs (coinduction), there is a synergistic interaction and the induction dose is less than 0.1 mg/kg.[4]

Higher maintenance infusion rates and prolonged administration will result in dose-dependent respiratory depression, accumulation and prolonged recovery time.

Lower infusion rates are sufficient to provide sedation and amnesia during local and regional anesthesia.

Prevention of nausea and vomiting: Several studies have supported the role of midazolam (0.05 mg/kg) in the prevention of nausea and vomiting, specially after pediatric strabismus or head-neck surgery.

Side Effects

The most significant problem with midazolam is respiratory depression which is enhanced in patients with chronic respiratory disease. Synergistic depressant effects occur when benzodiazepines are co-administered with opioid analgesics. Benzodiazepines also depress the swallowing reflex and decrease upper airway reflex activity.

Midazolam produces decrease in systemic vascular resistance and blood pressure when large doses are administered for induction of anesthesia. However, the cardiovascular depressant effects are frequently "masked" by the stimulus of laryngoscopy and intubation.

Airway obstruction may follow inadvertent overdosage and is more likely in elderly or debilitated patients.

When used as sedative or for induction and maintenance of anesthesia, benzodiazepines can produce an undesirable degree or prolonged interval of postoperative amnesia, sedation, and, rarely, respiratory depression. These residual effects can be reversed with flumazenil.

Proposed usage of anesthetic drugs and dosing guidelines for endotracheal intubation under various emergency conditions are summarised in Table 3.4. These guidelines do not replace clinical judgment and should be evaluated within the clinical context on an individual basis.

Table 3.4: Proposed usage of anesthetic drugs for endotracheal intubation under various emergency conditions

Condition	*Analgesia*	*Amnesia*	*Hypnosis*	*Muscle relaxation*
Shock (SBP < 80 mm Hg)	Fentanyl (0.5-1 mcg/kg, titrated)	Midazolam (1-2 mg) (if BP increases with resuscitation)	None, until intravascular volume and tissue perfusion restored	SUX 1.5 mg/kg or ROC 1.2mg/kg
Hypotension (SBP 80-100 mm Hg) GCS 4-9	Not needed	Not needed	Not needed	SUX 1.5 mg/kg or ROC 1.2 mg/kg
Hypotension (SBP 80-100 mm Hg) GCS >9	Fentanyl (1-2 mcg/kg)	Midazolam (1-2 mg) If not used for hypnosis	Ketamine 1 mg/kg or Etomidate 0.1-0.2 mg/kg Midazolam 0.1-0.3 mg/kg (titrate to effect)	SUX 1.5 mg/kg or ROC 1.2 mg/kg
GCS = 3, Flaccid, unresponsive	Not needed	Not needed	Not needed	Not needed
GCS 4-9, Head injury with hypertension	Fentanyl (1-2 mcg/kg)	Midazolam (1-2 mg) If not used for hypnosis	Thiopental 2-5 mg/kg or Propofol 1-2 mg/kg or Midazolam 0.2-0.3 mg/kg	SUX 1.5 mg/kg or ROC 1.2 mg/kg
GCS >9, Combative, BP normal or elevated	Fentanyl (1-2 mcg/kg)	Midazolam (1-2 mg) If not used for hypnosis	Thiopental 2-5 mg/kg or Propofol 1-2 mg/kg or Sevoflurane 4-8% or Midazolam 0.2-0.3 mg/kg	SUX 1.5 mg/kg or ROC 1.2 mg/kg
Cardiac arrest	Not needed	Not needed	Not needed	Not needed

GCS, Glasgow Coma Scale; SBP, systolic blood pressure; SUX, succinylcholine (first choice for neuromuscular blockade unless contraindicated); ROC, rocuronium.

REFERENCES

1. Hilty C, Minokadeh A, Wilson WC. General Anesthesia for trauma and emergency situation. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma Emergency Resuscitation Perioperative Anesthesia Surgical Management. Vol. 1. New York: Informa Healthcare 2007; 365-81.
2. Aitkenhead AR. Intravenous anesthetic agents. In: Aitkenhead AR, Rowbotham DJ, Smith G (Eds). Textbook of Anesthesia, 4th edn. Edinburg: Churchill Livingstone 2001;169-83.
3. Vohra A, Thomas AN, Harper NJN, et al. Non-invasive measurement of cardiac output during induction of anesthesia and tracheal intubation: Thiopentone and propofol compared British Journal of Anaesthesia, 1991;67(1):64-68.
4. Reves JG, Glass PSA, Lubarsky DA, McEvoy MD, Ruiz MR. Intravenous Anesthetics. In: Miller RD, Eriksson LI, Fleisher Lee A, (Eds). Miller's Anaesthesia. 7th edn. United States of America: Churchill Livingstone Elsevier, 2010;719-68
5. Dershwitz M, Rosow CE. Pharmacology of Intravenous Anesthetics. In: Longnecker DE, Zapol WM, Newman MF, Brown DL (Eds). Anesthesiology. United States of America: The McGraw-Hill Companies 2008; 849-68.
6. Gopalakrishna MD, Krishna HM, Shenoy UK. The effect of ephedrine on intubating conditions and haemodynamics during rapid tracheal intubation using propofol and rocuronium. Br J Anaesth. 2007; 99(2): 191-4.
7. Johnson K, Egan TD. Blood Loss: Does It Change My Intravenous Anesthetic? In: Smith CE, Como JJ editors. Trauma Anesthesia 1st edn. New York: Cambridge University Press 2008; 133-41.
8. Morris C, Perris A, Klein J, Mahoney P. Anaesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent? Anaesthesia 2009; 532-9.
9. Imarengiaye C. Anaesthetic management of surgical emergencies. Journal of Postgraduate Medicine. December, 2009;11(1):43-47.
10. A J Oglesby. Should etomidate be the induction agent of choice for rapid sequence intubation in the emergency department? Emerg Med J 2004;21:655–659.
11. Skinner HJ, Biswas A, Mahajan RP. Evaluation of intubating conditions with rocuronium and either propofol or etomidate for rapid sequence induction. Anaesthesia.1998;53(7):702-6.
12. Forman SA, Mashour GA. Pharmacology of inhalational agents. In: Longnecker DE, Zapol WM, Newman MF, Brown DL (Eds). Anesthesiology. United States of America: The McGraw-Hill Companies 2008; 739-66.
13. Lowry DW, Carroll MT, Mirakhur R K, et al. Comparison of sevoflurane and propofol with rocuronium for modified rapid-sequence induction of anaesthesia. Anaesthesia 1999;vol 54(3):247-52.
14. El-Orbany MI, Wafai Y, Joseph NJ, et al. Tracheal intubation conditions and cardiovascular effects after modified rapid-sequence induction with sevoflurane-rapacuronium versus propofol-rapacuronium. J Clin Anesth 2002; 14(2):115-20.

15. Moore EW, Davies MW. Inhalational versus intravenous induction. A survey of emergency anaesthetic practice in the United Kingdom. Eur J of Anaesth 2000; 17 (1): 33-7.
16. Viitanen H, Baer G, Koivu H, et al. The hemodynamic and holter-electrocardiogram changes during halothane and sevoflurane anesthesia for adenoidectomy in children aged one to three years. Anesth analg 1999;89:1423.
17. Sloan MH, Conard PF, Karsunky PK, et al. Sevoflurane versus isoflurane: Induction and recovery characteristics with single-breath inhaled inductions of anesthesia. Anesth Analg. 1996;82(3):528-32.
18. Vial G, Nathan N, Benrhaiem M, et al. Paroxystic systemic hypertension during inhalation induction with sevoflurane 8%. Can J Anesth 2000;47(2):191-4.
19. Ebert TJ, Muzi M, Lopatka CW. Neurocirculatory responses to sevoflurane in humans. A comparison to desflurane. Anesthesiology 1995;83:88-95.
20. Yli-Hankala A, Randell T, Seppälä T, Lindgren L. Increases in hemodynamic variables and catecholamine levels after rapid increase in sevoflurane concentration. Anesthesiology 1993;78:266–71.
21. Hall JE, Ebert TJ, Harmer M. Sevoflurane adult gaseous induction: An evaluation of stage II and its haemodynamic consequences. Br J Anaesth 1999;82:A452.
22. Davis PJ, Lerman J, Tofovic SP, Cook RD. Pharmacology of Pediatric Anesthesia. In: Motoyama EK, Davis PJ editors. Smith's Anesthesia for Infants and Children, 7th edn. Philadelphia; Mosby Elsevier 2006;206-210.
23. Njoku D, Laster MJ, Gong DH. Biotransformation of halothane, enflurane, isoflurane, and desflurane to trifluoroacetylated liver proteins: Association between protein acylation and hepatic injury. Anesth Analg 1997;84:173.
24. Donati F. Pharmacology of neuromuscular blocking agents and their reversal in trauma patients. In: Smith CE, Como JJ (Eds). Trauma Anesthesia 1st Ed. New York: Cambridge University Press 2008;143-54.
25. Brodsky JB, Lemmens HJM, Morton JM. Succinylcholine: A drug useful in bariatric surgery. Obes Surg. 2009;19:537. doi: 10.1007/s11695-008-9795-1.
26. Schreiber JU, Buder TF Succinylcholine: The Dilemma with the evidence Obes Surg 2010;20(1): 134.
27. Tejirian T, Lewis CE, Conner J, et al. Succinylcholine: A Drug to Avoid in Bariatric Surgery. Obes Surg 2009;19: 534-6.
28. Larsen PB, Hansen EG, Jacobsen LS, et al. Intubation conditions after rocuronium or succinylcholine for rapid sequence induction with alfentanil and propofol in the emergency patient. Eur J Anaesthesiol. 2005; 22(10):748-53.
29. Di Filippo A, Gonnelli C. Rapid sequence intubation: a review of recent evidences. Rev Recent Clin Trials. 2009; 4(3):175-8.
30. Sasakawa T, Iwasaki H. Does rocuronium displace the position of suxamethonium? Masui. 2008; 57(7):860-8.
31. De Boer HD, Van Egmond J, Van de Pol1 F, Bom A. Sugammadex, a new reversal agent for neuromuscular block induced by rocuronium in the anaesthetized Rhesus monkey. Br J Anaesth 2006;96:473-9.
32. Koh KF, Chen FG. Rapid tracheal intubation with atracurium: The timing principle. Can J Anaesth. 1994;41(8):688-93.
33. Naguib M, Lien CA. Pharmacology of muscle relaxants and their antagonists. In: Miller RD, Eriksson LI, Fleisher Lee A, (Eds). Miller's Anaesthesia. 7th edn. United States of America: Churchill Livingstone Elsevier; 2010;859-912.
34. Fukuda K. Opioids. In: Miller RD, Eriksson LI, Fleisher Lee A, (Eds). Miller's Anaesthesia. 7th ed. United States of America: Churchill Livingstone Elsevier; 2010;769-824.
35. Rosow C, Dershwitz M. Pharmacology of Opioid Analgesics. In: Longnecker DE, Zapol WM, Newman MF, Brown DL editors. Anesthesiology. United States of America: The McGraw-Hill Companies 2008;869-896.
36. Abou-Arab MH, Heier T, Caldwell JE. Dose of alfentanil needed to obtain optimal intubation conditions during rapid-sequence induction of anaesthesia with thiopentone and rocuronium. Br J Anaesth 2007;98(5):604-10.
37. Kim JT, Shim JK, Kim SH, et al. Remifentanil vs. lignocaine for attenuating the haemodynamic response during rapid sequence induction using propofol: Double-blind randomised clinical trial. Anaesth Intensive Care. 2007;35(1):20-3.
38. Coda BA. Opioids. In; Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anesthesia, 5th edn. United States of America; Lippincott Williams & Wilkins 2006;654-684.
39. White PF, Romero G. Nonopioid Intravenous Anesthesia. In; Barash PG, Cullen BF, Stoelting RK (Eds.) Clinical Anesthesia, 5th edn. United States of America; Lippincott Williams & Wilkins 2006;634-53.

4 Blood and Blood Component Therapy in Emergency Anesthesia

Aparna A Nerurkar, Manish B Kotwani

KEY POINTS

- Blood management has been defined by Society for Advancement of Blood Management (SABM) as "the appropriate use of blood and blood components with a goal of minimizing their use."
- Hemoglobin triggers for transfusion are not to be taken as absolute indications. Patients should be transfused if signs or symptoms of inadequate myocardial oxygenation or organ insufficiency are present.
- All blood components including platelets should be transfused through a blood transfusion set with an integral mesh filter (170-200 micron).
- A new syringe and administration set should be used when administering different components.
- Aseptic technique must be employed during preparation and administration. If the container is entered in a manner that violates the integrity of the system, the component expires 4 hours after entry if maintained at room temperature (20-24°C), or 24 hours after entry if refrigerated (1-6°C).
- All unused components should be returned to the blood bank as soon as possible with information regarding the circumstances of return.
- If red cell units are out of temperature controlled storage for more than 30 minutes they should not be put back into storage for re-issue.
- The ideal solution to manage hypovolemia and anemia due to major blood loss is administration of fresh whole blood. However logistic and testing issues make it difficult for the blood bank to provide fresh whole blood.
- In an extreme situation where blood is required immediately and the patient's blood group is unknown, either type-specific or type O uncrossmatched red cells can be administered while awaiting a crossmatch.
- Type O Rh-negative, uncrossmatched packed RBCs should be used in preference to type O Rh-negative whole blood because packed erythrocytes have smaller volumes of plasma and are almost free of hemolytic anti-A and anti-B antibodies.
- When more than two units of type O Rh-negative, uncrossmatched whole blood have been administered, the patient probably cannot be immediately switched back to his or her blood type (A, B, or AB). Switching could cause major intravascular hemolysis of donor RBCs by increasing transfused anti-A and anti-B antibodies.
- The patient must not be transfused with his or her correct blood type until the transfused anti-A and anti-B has decreased to levels that permit safe transfusion of type-specific blood.
- Prophylactic administration of alkalinizing agents such as sodium bicarbonate or tris-hydroxymethyl aminomethane (THAM) is unnecessary and may cause serious alkalosis and hypernatremia.
- Hyperkalemia may occur, due to the high extracellular potassium concentration in stored red cell units. Treatment includes calcium gluconate, glucose insulin regimens (10 units of insulin with 50cc 50% dextrose over 20-30 mins) and beta-2 agonists together with bicarbonate to correct acidosis.
- Prophylactic platelet transfusion is rarely required for counts $\geq 100 \times 10^9$/L and always indicated at counts $\leq 50 \times 10^9$/L with massive bleeding. A platelet count increment of approximately 7 to10,000/mm^3 for each RDP (random donor platelet) given, or 30 to 60,000/mm^3 for each SDP (single donor platelet) given to an adult is expected.
- Treatment of microvascular bleeding due to congenital and acquired coagulopathies including massive blood loss, disseminated intravascular coagulation with depletion of clotting factors may require 10 to 20 ml/kg of fresh frozen plasma (FFP).

- FFP is contraindicated for increasing plasma volume or albumin concentration.
- One cryoprecipitate unit per 7 to 10 kg of body weight can be used in massive hemorrhage or disseminated intravascular coagulopathy (DIC) to raise plasma fibrinogen concentration by ~ 50 to 70 mg/dl.
- As an off-label use in massive hemorrhage, recombinant factor VIIa (rfVIIa) 90μg/kg as a slow intravenous bolus can be considered if all possible hemostatic measures like surgical control of bleeding, temperature correction and blood product administration have been carried out.
- Transfusion-Associated Circulatory Overload (TACO) resulting in pulmonary edema can accompany transfusion of any component at a rate more rapid than the recipient's cardiac output can accommodate.
- Transfusion-Related Acute Lung Injury (TRALI), indistinguishable from adult respiratory distress syndrome (ARDS) and having a mortality rate of approximately 5 to 10%, may develop within 6 hours of a transfusion. Supportive therapy in the form of high FiO_2, endotracheal intubation with ventilatory support, volume resuscitation and vasopressor support is needed.
- A checklist for checking blood and blood components must be developed for each institute and the final check should always be by the patient's bedside.
- The responsibility of implementing the Guidelines is a shared one and requires the support of health departments, clinicians and laboratory staff.

INTRODUCTION

Anesthesiologists in their role as perioperative physicians play a major role in the utilization of blood and blood components. Life-threatening hemorrhage, especially in an emergency scenario, mandates quick and careful decisions about transfusion of blood and blood components. Pre-existing hematological derangements require rational and effective use of blood component therapy to optimize outcome in emergency surgeries.

Patients with massive blood loss and patients with deficiency of blood components pose a major challenge for hematological and blood transfusion services. The imbalance in the demand and supply of blood makes voluntarily donated allogenic blood a limited and a valuable resource. Blood and blood products must be used carefully, rationally, safely and on the basis of evidence based guidelines.

Triaging of blood and blood components to prioritize supply to patients whose transfusion cannot be deferred is one strategy for optimizing the balance between available blood inventory and clinical use during critical shortages. Such triaging is integral to many disaster management plans. Effective triaging requires precise information regarding the clinical setting of transfusion, its urgency, and its potential deferability. Also one needs to address the possibility of using anesthetic and surgical techniques, volume expanders, etc. to reduce the need for blood.

Blood management has thus been defined by Society for Advancement of Blood Management (SABM) as "the appropriate use of blood and blood components with a goal of minimizing their use."

Technological advances in blood collection, separation, anticoagulation, and preservation have resulted in component preparation of red blood cells, platelets, white blood cells and plasma which are superior to whole blood used in the past. Blood component separation also augments the meager supply of blood by making available the exact component needed for the patient's hematological condition.

However, there are substantial differences in the clinical use of blood and blood components across the globe. This reflects a lack of consensus among clinicians about specific criteria for the appropriate use. Several studies have found that the amount of blood used and the percentage of admissions involving the use of blood components vary across different types of hospitals, even for the same procedure or diagnosis, and that the rate of use that can be classified as inappropriate is unacceptably high.[1,2,3,4,5]

Various strategies have now been developed to reduce the inappropriate use of blood. These include guidelines (ASA[6,7], BSH[8,9,10], AAGBI[11], ARC[12], GOI[13]), circulars (AABB[14]), handbooks (WHO[15,16]), and consensus conferences, as well as monitoring of transfusion practice (retrospective and prospective), education and self-audit[17] by clinicians. Physicians prescribing blood components should have a basic understanding of indications and contraindications of their use. They should be cognizant of methods of preparation, proper storage conditions, indications, contraindications, dosages and methods of proper transfusion of blood products to prevent potential adverse effects. The following tables of blood component use have been compiled from the above

mentioned learning material along with material in standard textbooks[18,19] (Tables 4.1 to 4.4).

TECHNICAL ASPECTS OF BLOOD COMPONENT ADMINISTRATION

Venous Access

- Blood components can be administered through peripheral intravenous cannula or most central venous access devices as per manufacturer's specifications.
- The size of the peripheral cannula depends on the size and integrity of the vein and the speed at which the blood component is to be transfused. In pediatric patients, special care must be taken to ensure blood transfusion through largest available venous access, to avoid hemolysis and consequent hyperkalemia.
- Peripherally inserted long central catheters (PICC lines) with narrow lumen diameter may lead to slower flow rates.
- When multi-lumen central venous access devices are used it is generally safe to coadminister other therapeutic solutions through a different lumen as rapid dilution occurs in the bloodstream.

Administration Equipment

Adult Administration

- All blood components should be transfused through a blood transfusion set with an integral mesh filter (170–200 micron).
- It is not essential to use the specially available platelet administration set that contains the same integral mesh filter (170–200 micron) as a blood administration set, but has a smaller lumen and thus a smaller priming volume.
- Platelets should be transfused through a fresh blood transfusion set. Blood sets utilized for other blood components should not be used.
- The administration set should be changed at least every 12 hours (or in accordance with manufacturer's instructions). This is intended to reduce the risk of bacterial growth.
- A new administration set should be used if another infusion is to continue after the transfusion. This is intended to reduce the risk of incompatible fluids or drugs causing hemolysis of residual red cells in the administration set or drip chamber.
- Leukodepletion filters can be used in special situations like cardiac surgery, oncology, etc.

Table 4.1: Packed red blood cells

Description	Packed Red Blood Cells consist of erythrocytes concentrated from whole blood donations by centrifugation or collected by apheresis method. 300–350 ml of RBCs in additive solution collected from 450 ml of whole blood Depending on the preservative-anticoagulant system used, the hematocrit of Red Blood Cells ranges from about 50–65% (e.g. AS-1, AS-3, AS-5) to about 65–80% (e.g. CPDA-1, CPD)
Storage	2–6°C in designated temperature controlled refrigerator.
Shelf Life	35 days (CPDA), 42 days (when Adsol® is added to CPDA)
Dosage/Yield	• One unit of PRBCs will increase the hematocrit by approximately 3% and the hemoglobin by about 1g/dl in the average adult. • In neonates, a dose of 5 ml/kg of packed red cells with a hematocrit of approximately 60% will increase the hemoglobin by about 1g/dl • In the case of a lower than expected transfusion yield, conditions causing the loss, sequestration or destruction of RBCs should be looked for. Such as occult bleeding; repeated blood sampling (particularly in children); fever; hypersplenism; primary and secondary immunological causes; mechanical or other type of hemolysis.
Indications	• Patients with values above 10 g/dl rarely require transfusion • Evidence supports transfusion in absence of physiologic signs with hemoglobin levels below 6 mg/dl in general patient population, below 7 mg/dl in patients > 80 years old or in febrile/ hypermetabolic patients, and below 8 mg/dl in patients with coronary artery disease or CHF. • The determination of whether intermediate hemoglobin concentrations (i.e. 6–10 g/dl) justify or require RBC transfusion should be based on any ongoing indication of organ ischemia, potential or actual ongoing bleeding, the patient's intravascular volume status, and the patient's risk factors for complications of inadequate oxygenation. • Cardiac patients should be transfused if signs or symptoms of inadequate myocardial oxygenation are present. Currently, red cell transfusion should be considered in following clinical scenario: – Anemic patients with relative hypotension/ tachycardia – New ST-segment depression > 0.1 mV or new ST-segment elevation > 0.2 mV – New wall motion abnormality – Mixed venous oxygen partial pressure less than 25 mmHg – Oxygen extraction rate more than 50% – Mixed venous oxygen saturation less than 50% and more than 10% decrease in oxygen consumption
Need for crossmatch	• A crossmatch is performed to ensure compatibility between the donor's RBCs and the recipient's plasma.

Table 4.2: Plasma (≈ 250 ml)

Description	• FFP contains normal levels of the stable clotting factors, albumin and immunoglobulins. It contains at least 70% of the original coagulant factor VIII and at least similar quantities of the other labile clotting factors and natural inhibitors of coagulation.
Storage	• Frozen at –18°C or colder in a designated temperature controlled freezer within 6–8 h of collection
Shelf Life	• 24 months (frozen)
Dosage	• When FFP is indicated, it should be administered in a dose calculated to achieve a minimum of 30% of plasma factor concentration. • Usually 10–20 ml/kg of FFP will generally result in a rise of most coagulation proteins by 25–30% (or increases in 0.25–0.3 U/ml) though more may be required depending upon the clinical situation. • A dose of 5–8 ml/kg may be adequate to urgently reverse warfarin anticoagulation.
Indications	• Frozen plasma is indicated for patients on warfarin only if there is serious bleeding or urgent reversal of warfarin effect is necessary. • Treatment of microvascular bleeding due to congenital and acquired coagulopathies including massive blood loss, disseminated intravascular coagulation with depletion of clotting factors resulting in a prolongation of either the activated partial thromboplastin time (aPTT) or prothrombin time (PT) greater than 1.5 times normal or a coagulation factor assay of less than 25 percent or empirically for more than 1 blood volume blood loss.
Comments	• Prior to the administration of FFP, the plasma must be thawed in a waterbath at 37ºC, which takes approximately 30 minutes. • Once thawed, FFP must not be re-frozen and should be transfused as soon as possible. Post-thaw storage will result in a decline in the content of labile coagulation factors • If stored at 1–6ºC post thawing, the FFP should be transfused within 24 hours. • FFP which has been thawed but not used within 24 hours can be relabelled as “Thawed Plasma” (TP) and stored at 1–6ºC for an additional 4 days. • ‘Thawed plasma’ can be used as a source of all stable coagulation factors except factor V which falls to 80% of normal and factor VIII which falls to 60% of normal. • FFP is contraindicated for increasing plasma volume or albumin concentration.
Need for crossmatch	• Because of anti-ABO antibodies in units of plasma, only ABO-compatible units can be used. Rh compatibility is not required. Thus, the recipient’s ABO type is needed for plasma transfusion. Because plasma from donors with type AB blood does not contain anti-ABO antibodies, it can be given to any patient and is used for emergency transfusion in patients with unknown blood types. Crossmatching of plasma is not required, since there are no RBCs in these products.

Table 4.3: Platelets

Description	• Platelet concentrate (random donor): Volume 50–70 ml Each unit contains approximately 5.5 × 10 platelets in 50 to 70 ml of plasma. • Pooled platelets: 5 to 10 units of random donor platelets may be pooled together in a single component bag. • Apheresis platelets (single donor): Volume 200 to 400 ml obtained by performing apheresis on volunteer donors. Each unit contains 15-50 × 10^{10} platelets, equivalent to 3–10 units of platelet concentrates.
Storage	• Room temperature (20°–24°C) with continuous gentle agitation to prevent platelet aggregation • Must not be refrigerated as it reduces platelet function
Shelf Life	• 5 days (7 days—in certain controlled circumstances)
Dosage/Yield	• Measure platelet count from 10 minutes to 3 hours after transfusion. • Generally, expect an adult platelet count increment of approximately 7–10,000/ mm^3 for each RDP given, or 30–60,000/ mm^3 for each SDP given. • In neonates and infants, a dose of 5–10 ml/kg of platelets (RDP or SDP) should result in a 50–100,000/mm^3 increment.
Indications	• Prophylactic preoperative transfusion is rarely required for counts ≥ 100 × 10^9/L, is usually required for counts < 50 × 10^9/L with bleeding • Factors to consider for the transfusion of platelets for counts between 50–100 × 10^9/L are the type of surgery, extent of actual blood loss or microvascular bleeding, presence of potent antiplatelet medications (e.g. clopidogrel, IIb/IIIa antagonists, etc.) and disorders like uremia known to affect platelet function and coagulation. Specific Procedures: • Procedures with insignificant blood loss or vaginal deliveries can be performed at counts <50 × 10^9/L without prophylactic transfusion. • Neurologic or ophthalmologic procedures require platelets near 100 × 10^9/L • In the absence of other coagulopathy, major invasive procedures require platelet counts of at least 40 to 50 × 10^9/L (including CVP placement, paracentesis/thoracentesis, respiratory tract/GI biopsies, closed liver biopsy, lumbar puncture, sinus aspiration and dental extraction). • A threshold of 80 × 10^9/L has been proposed for spinal epidural anesthesia. • Fiberoptic bronchoscopy without biopsy by an experienced operator may be safely performed in the presence of a platelet count ≈ 20 × 10^9/L • GI endoscopy without biopsy may be safely performed at platelet counts ≈ 20 × 10^9/L.[6] • Transfusion may be required with apparently adequate counts when known or suspected

Contd.

Contd.

	platelet dysfunction results in microvascular bleeding
Comments	• All platelet products should be tested for bacterial contamination prior to transfusion. • Platelet concentrates should not be transfused through administration sets which have already been used to administer other blood components • The infusion should be commenced as soon as possible after the component arrives in the clinical area • Typically administered over 30–60 minutes per dose. • Qualitative coagulation tests, such as bleeding time have been shown to have poor reproducibility and limited value.
Need for crossmatch	• ABO compatibility for platelet transfusion is desirable but not required because of the small amount of plasma present in a standard dose of platelets (each unit contains about 60 mL of plasma, and 5 or 6 units make up a standard dose)

Pediatric Administration

- Pediatric blood administration sets (with a smaller prime volume) are appropriate for small volume transfusions. Neonatal blood administration systems are also available which allow blood components to be delivered via a syringe driver. These systems should incorporate an integral three-way system allowing the blood component bag to remain attached throughout the transfusion.
- All administration systems should incorporate a 170 to 200 micron filter.
- A new syringe and administration set should be used when administering different components.
- Blood components from more than one donation should not be mixed in a syringe. Instead, it should be given sequentially using a new syringe in order to be able to identify the relevant donation in case of a reaction.

Infusion Devices

- Infusion devices should be used only by individuals competent in their use.
- Compatibility should be ensured between the blood component administration set and the infusion device as per manufacturers' recommendations.
- Administration sets used with infusion devices should incorporate an integral mesh filter (170-200 micron).
- The pre-administration checking procedure should include a check of the device and device settings.

Table 4.4: Cryoprecipitate

Description	• Cryoprecipitated antihemophilic factor or a cryoprecipitate unit is prepared by thawing one unit of FFP between 1–6°C and recovering the cold insoluble white precipitate which is removed following centrifugation and immediately refrozen • Each unit of cryoprecipitate contains 80 to 150 units of Factor VIII, 150 to 250 mg of fibrinogen, von Willebrand's factor, Factor XIII, and fibronectin in a volume of 5 to 15 ml • "Pooled Cryoprecipitate". Several units of cryoprecipitate are pooled into one bag and the volume of the pool is indicated on the label.
Storage/shelf Life	• Frozen at –18°C for 24 months
Dosage	• A typical dose for the treatment of hypofibrinogenemia is one cryoprecipitate unit per 7–10 kg of body weight. • One unit of cryoprecipitate per 10 kg of body weight raises plasma fibrinogen concentration by ~ 50 to 70 mg/dl in the absence of continued consumption or massive bleeding (assuming minimum fibrinogen content per bag of cryoprecipitate).
Indications	• Cryoprecipitate is used primarily to augment fibrinogen levels depleted because of massive hemorrhage or disseminated intravascular coagulopathy (DIC). • Rarely, it is used for the treatment of congenital or acquired Factor XIII deficiency. • Cryoprecipitate can also be administered prophylactically for non bleeding perioperative or peripartum patients with congenital fibrinogen deficiencies or von Willebrand's disease (deficient or abnormal von Willebrand's molecule) unresponsive to desmopressin.
Comments	• CMV testing and leukoreduction are not required. • Frozen cryoprecipitate is thawed in a protective plastic overwrap in a waterbath at 30–37°C up to 15 minutes. Thawed cryoprecipitate should be kept at room temperature and transfused as soon as possible or within four to six hours. • Should be infused through a 170 to 260 micron component filter • Because cryoprecipitate does not contain Factor V, it should not be the sole replacement therapy for disseminated intravascular coagulopathy (DIC), which is almost always associated with a variety of factor deficiencies and thrombocytopenia. Hence, FFP also needs to be administered along with platelet concentrates in those settings where a coagulopathy secondary to DIC is likely occurring.
Need for crossmatch	• Each unit of cryoprecipitate contains a small amount of plasma (15 mL/unit) and thus does not require ABO compatibility. Rh type need not be considered. It is preferable to use cryoprecipitate that is ABO-compatible with the recipient's red cells

- Infusion devices should be regularly maintained in accordance with manufacturers and/or organizational guidelines.
- Any adverse outcome as a result of using an infusion device to transfuse red cells should be reported to the appropriate authorities.

Infusion Rate Devices

- Precise infusion rate controlled, gravity-based or electronic infusion devices may be used for the administration of blood and blood components.
- Rapid infusion devices may be used when large volumes have to be infused quickly, as in massive hemorrhage. These typically have a range of 6 to 30 liters/hour and usually incorporate a blood warming device.
- Infusion devices should only be used if the manufacturer verifies them as safe for this purpose and they are CE marked.
- The volume delivered should be monitored regularly throughout the infusion to ensure that the expected volume is delivered at the required rate.

Pressure Devices

- External pressure devices make it possible to administer a unit of red cells within a few minutes. They should only be used in an emergency situation together with a large gauge venous access cannula or device.
- External pressure devices should exert even pressure over the entire bag, have a gauge to measure the pressure which should not exceed 300 mm Hg pressure and be monitored at all times when in use.

Blood Warmers

- Most published guidelines[20,13,15] only recommend the routine use of blood warmers in adult patients undergoing rapid or high volume transfusion of red cells in the context of major hemorrhage. Blood transfusion at a rate of > 50 ml/kg/hr in adults and >15 ml/kg/hr in pediatrics, exchange transfusion in infants or patients with clinically significant "cold" antibodies are some of the indications for use of blood warmers.
- In most other clinical situations where there is concern, it is sufficient to allow blood to come up to ambient temperature before transfusion.
- Special consideration should be given when rapidly transfusing large volumes to neonates, children, elderly patients, and patients susceptible to cardiac dysfunction.
- Platelet warming to 37°C was earlier recommended but there is no evidence that warming platelets before infusion improves postinfusion platelet viability[21].
- Blood should only be warmed using approved, specifically designed and regularly maintained blood-warming equipment with a visible thermometer and audible warning. Settings should be monitored regularly throughout the transfusion. Some blood warmers operate up to 43°C but are safe provided they are used and serviced according to manufacturers' instructions.
- Blood components should never be warmed using improvisations, such as putting the pack in warm water, in a microwave or on a radiator. These may increase the chances of hemolysis and infection.

Compatible Intravenous Fluids

- It is generally advised that no other intravenous fluids should be coadministered via an infusion line that is being used for a blood component (when multi-lumen central venous access devices are used, it is generally safe to coadminister other therapeutic solutions through a different lumen as rapid dilution occurs in the blood stream).
- Intravenous solutions which contain calcium, such as Ringer Lactate, and calcium-containing colloids, such as Hemaccel™ or Gelofusine™ may antagonize citrate anticoagulant and allow clots to form in the blood component.
- Hypotonic intravenous solutions, such as 5% dextrose in water, may cause hemolysis of red cells.
- The practice of priming or flushing administration sets used for the transfusion of blood components with isotonic (0.9%) saline is not evidence-based and is unnecessary. No other intravenous fluids should be used for this purpose.

Coadministration of Drugs with Blood Components

- The addition of a drug to an intravenous line containing blood or blood components raises concerns about compatibility of the drug and its carrier with the blood component and any preservatives or additives.
- A break in the integrity of the infusion line may also increase the risk of bacterial contamination of the component.

- Recent studies show that standard concentrations of morphine, hydromorphone or meperidine given by continuous infusion or single or multiple boluses have no significant deleterious effects on co-administered red cells [22,23]
- For the administration of any other drugs, wherever possible drugs should be timed to be administered between transfusions, or administered via a second venous access device.
- There is insufficient evidence to guide a policy change on the coadministration of drugs and red blood cells due to the lack of clinical applicability of *in vitro* experiments and diversity of clinical outcome measures used.
- Under no circumstances should drugs be directly added to a blood component bag.

Disposal of Equipment

- If there is any suspicion of a transfusion reaction, the component pack should be returned to the transfusion laboratory with full clinical details.
- If a transfusion is completed uneventfully, the empty blood component pack and administration set should be discarded according to the organization's policy for disposing of clinical waste. Generally the set is cut so as prevent misuse.
- If, in accordance with local policy, any paperwork is required for the traceability of blood components this must be done before disposal of the unit.

Return of Blood Components

- If a blood component is returned from a clinical area to a designated blood refrigerator/ storage location, the following information should be recorded for each individual unit:
- Identity of the person returning the component
- Date and time component placed in the blood refrigerator/storage location
- All unused components should be returned as soon as possible and the clinical areas should inform the laboratory of the circumstances of the return.
- If red cell units are out of temperature controlled storage for more than 30 minutes, they should not be put back into storage for reissue.

MASSIVE BLOOD TRANSFUSION

Definition

Massive blood transfusion is the replacement of blood loss equivalent to or greater than the patient's total blood volume in less than 24 hours which amounts to approximately 70 ml/kg in adults and 80 to 90 ml/kg in children or infants. Alternative definitions include 50% of the blood volume loss within 3 hours or the rate of blood loss >150 ml/min.

A dynamic definition of massive transfusion, such as the transfusion of four or more red cell concentrates within one hour when on going need is foreseeable, or the replacement of 50% of the total blood volume within three hours, may be more appropriate in the acute clinical setting.

Such definitions emphasize the importance of the early recognition of major blood loss and the need for effective action to prevent shock and its consequences. Massive blood transfusion is one of the main prognostic factor for mortality in trauma.[24]

Early consultation with surgical and hematology colleagues is advisable, and the importance of good communication and cooperation in this situation cannot be overemphasized.

The most common clinical situation leading to massive transfusion is extensive trauma; however, it also may occur in nontrauma settings during surgical procedures causing large blood loss especially after cardiothoracic and emergency obstetric surgery. In such situations, the ideal solution to manage hypovolemia and anemia due to major blood loss, involves administration of fresh whole blood since this approach restores not only oxygen carrying capacity and oxygen delivery but also hemostasis via maintenance of normal levels of coagulation factor and platelets. However due to logistic and testing issues, it is difficult for the blood bank to provide fresh whole blood every time. It may often be necessary to make a pragmatic decision regarding the relative risks of delaying transfusion or giving components that are not of the appropriate specification.

PRACTICAL APPROACH TO MASSIVE BLOOD TRANSFUSION

Priorities for treatment should be

- Maintenance of tissue perfusion and oxygenation by restoration of blood volume and hemoglobin.
- Achieving hemostasis by treating any surgical, obstetrical or traumatic source of bleeding and correcting coagulopathy by the judicious use of blood component therapy.

A successful outcome requires prompt action and good communication between clinical specialties, diagnostic laboratories, blood-bank staff and the local blood center. Blood component support takes time to organize and sometimes the blood center may be away from the hospital.

Table 4.5: Goal directed management of massive blood loss

Goal	*Approach*	*Comments*
Volume Resuscitation	Secure wide bore peripheral venous access or central single lumen or multi-lumen devices Administer warm intravenous fluids—crystalloid or colloid as needed Avoid hypotension or urine output <0.5 ml/kg/h	Preferably 14-gauge cannulae monitor CVP Keep patient warm Account for concealed blood loss
Clear communication with concerned personnel	Surgeon, Physician, in-charge Consultant anesthesiologist, Blood Bank Officer or Hematologist, Relatives/Caregivers	Early communication facilitates availability of blood, blood products, control of bleeding and postoperative intensive care support if required Communication with relatives and documentation is essential for medico-legal purposes
Control bleeding	Early surgical or obstetric intervention Interventional radiology Use of anesthetic techniques to reduce bleeding	
Obtain laboratory investigations	Blood group/crossmatch CBC including platelets PT, APTT, Thrombin time, Fibrinogen, FDP BS, RFT, LFT, Electrolytes Blood gases TEG, if available Repeat tests after blood component infusion	Ensure correct patient and sample identification Results may be affected by colloid infusion. May need to give components before results available
Maintain Hb >8 g/dl	Employ techniques to reduce bleeding and use blood salvage to minimize allogenic blood use Give Group O Rh D negative packed red blood cells in extreme emergency until ABO and Rh D groups known. If blood group is known use ABO group specific packed RBCs Fully compatible blood should be used if time permits Use blood warmer and/or rapid infusion device if flow rate >50 ml/kg/h in adult	Use cell savers if available. O positive is acceptable in males and postmenopausal females Further serological crossmatch not required after 1 BV replacement. Do not switch back to patient's group, after O group administration, till antibody titers confirmed to be low.Transfusion laboratory will complete crossmatch after issue.
Maintain platelet count >7.5 × 10^{10}/L	Allow for delivery time from blood center Anticipate platelet count <50 × 10^{9}/l. After 2 × blood volume replacement	Allows margin of safety to ensure platelet count >5.0 × 10^{10}/L Keep platelet count >10.0 × 10^{10}/L if multi-organ or CNS trauma or if platelet function is abnormal
Maintain PT and APTT <1.5 × mean control	Give FFP 12–15 ml/kg (Four units for an adult) guided by tests Anticipate need for FFP after 1–1.5 × BV replacement.	PT/APTT >1.5 × mean normal value correlates with increased microvascular bleeding Keep ionized Ca^{2+} >1.13 mmol/l
Maintain Fibrinogen >1.0 g/l	If not corrected by FFP give cryoprecipitate (2 packs of pooled cryoprecipitate for an adult)	Rarely needed except in DIC
Avoid DIC	Treat underlying cause (shock, hypothermia, acidosis)	Although rare, mortality is high

CVP, central venous pressure; CBC, complete blood count; PT, prothrombin time; APTT, activated partial thromboplastin time; FDP-Fibrin degradation products; BS-Blood Sugar, RFT-Renal function tests, LFT-Liver Function Tests, FFP, fresh frozen plasma; DIC, disseminated intravascular coagulation; BV, blood volume

The blood bank must be informed of a massive transfusion situation at the earliest possible opportunity. This will provide an opportunity to check stock, reschedule nonurgent work and call in additional staff if required out of hours.

Clear local protocols for management of massive blood loss should be accessible in all relevant clinical and laboratory areas and understood by all involved staff (Table 4.5).

Volume Resuscitation

The overriding first requirement is maintenance of tissue perfusion and oxygenation. This is critical in preventing the development of hypovolemic shock and consequent high mortality from multiorgan failure. Restoration of circulating volume is initially achieved by rapid infusion of crystalloid or colloid through large bore (up to 14 gauge) peripheral cannulae. The 2007 Cochrane review[25] stated that there is no evidence from

Table 4.6: Synthetic colloid solutions

Generic Name	*Contents*	*Intravascular Half Life*
Dextran 40	10% polysaccharide (MW 40,000) with normal saline	4–6 hrs
Dextran 60–70	6–7% polysaccharide (MW 70,000) with normal saline	6–8 hrs
Hydroxyethyl starch	6% hydroxyethyl starch solution in 0.9% saline (MW 45,000) or Ringer's lactate	24 hrs
Hydroxyethyl starch 0.4 molar substitution	6% hydroxyethyl starch 130/0.4 in 0.9% sodium chloride injection	5–6 hrs
Gelatin	3.5% gelatin polypeptide (MW 35,000) with Ringer's solution	3–5 hrs
Succinated Gelatin	4% succinylated (or modified fluid) gelatin, sodiumhydroxide	4–5 hrs

RCTs that resuscitation with colloids reduces the risk of death, compared to resuscitation with crystalloids, in patients with trauma, burns or following surgery. Larger bore central access devices can be used depending on local skills and availability. Similarly, crystalloids and colloids can be chosen as per availability, intravascular half-life (Table 4.6), hemodynamic conditions and time for arrival of cross-matched blood and components.

Blood Component Therapy

Red cells: The function of red cells is oxygen delivery to tissues; they should not be used as a volume expander. Red cells also contribute to hemostasis by their effect on platelet margination and function.

The optimal hematocrit to prevent coagulopathy is unknown, but experimental evidence suggests that a relatively high hematocrit, possibly ≈35% may be required to sustain hemostasis in patients with massive blood loss. Red cell transfusion is likely to be required when 30 to 40% blood volume is lost; over 40% blood volume loss is immediately life-threatening.

Blood replacement should be guided by clinical estimation of blood loss in conjunction with the patient's response to volume replacement. Hemoglobin and hematocrit levels should be measured frequently, with the knowledge that the hemoglobin level is a poor indicator of blood loss in the acute situation.

Red cells are rarely indicated when the hemoglobin concentration is >10 g/dl but almost always indicated when it is <6 g/dl.[1] Decisions on red cell transfusion at intermediate hemoglobin concentrations should be based on the patient's risk factors for complications of inadequate oxygenation, such as rate of blood loss, cardio-respiratory reserve, oxygen consumption and atherosclerotic disease. Measured physiological variables, such as heart rate, arterial pressure, pulmonary capillary wedge pressure and cardiac output may assist the decision-making process, but it should be emphasized that silent tissue or organ ischemia may occur in the presence of stable vital signs[26]

The requisition form for blood should indicate the time scale within which blood is needed at the theater, (i.e. immediately, within 20 minutes, within an hour) in order that the blood bank personnel to know how much time is available for ABO and D grouping and pre-transfusion testing. The number and type of blood components needed, the area where it is needed and reason for transfusion should be clearly communicated.

Which fluids should be administered while awaiting crossmatched blood?

While waiting for crossmatched blood to be available, emergency transfusion needs can be met in various ways, depending on the clinical status of the patient, the available resuscitation fluids and equipment and the anticipated delay in obtaining blood.

Most patients can tolerate an acute decrease in hemoglobin and oxygen-carrying capacity providing circulating volume is maintained. Crystalloid or colloid solutions may be infused for volume resuscitation and maintaining hemodynamic stability. Resuscitation with colloids does not reduce the risk of death, compared to resuscitation with crystalloids, in patients with trauma, burns or following surgery.[25] The Cochrane reviews[27,28] also suggest that there is no evidence that one colloid solution is more effective or safe than any other. Other studies state that trauma patients should continue to be resuscitated with crystalloids.[29] However, administration of crystalloid to normalize blood pressure should be titrated to the clinical condition. Penetrating torso injuries, head injuries may require careful crystalloid administration. In fact, studies have shown that attempting to achieve normal blood pressure in the setting of active bleeding through extensive fluid therapy is associated with disruption of haemostatic mechanisms, dilution of clotting factors, increased blood loss and decreased survival.[30] Therefore, maintaining blood pressure below normal (systolic 80 mmHg; mean 50–60 mmHg) and heart rate under 120 while closely monitoring indicators of organ ischemia are the suggested goals for early resuscitation in otherwise healthy patients.

In an extreme situation where blood is required immediately and the patient's blood group is unknown or if the patient has cardiac disease, pulmonary disease or cerebrovascular disease and acute anemia will pose increased risk, then either type-specific or type O uncrossmatched red cells can be administered while waiting for a crossmatch to be performed. Type O Rh-positive Red Blood Cells for males or postmenopausal females can be transfused in this setting as well. Administration of Group O uncrossmatched blood is safe provided that the patient is not already alloimmunized to any non-ABO red cell antigens.[18,31,32,33]

However, it should be remembered, that some type O donors produce high titers of hemolytic IgG, IgM, anti-A, and anti-B antibodies. High titers of these hemolysins in donor units are capable of causing destruction of A or B RBCs of a non-type O recipient. Type O Rh-negative, uncrossmatched packed RBCs should be used in preference to type O Rh-negative whole blood because packed erythrocytes have smaller volumes of plasma and are almost free of hemolytic anti-A and anti-B antibodies. Females of reproductive age (i.e. under 50 years) whose blood group is unknown must be given group O Rh D negative red cells in order to avoid sensitization and the risk of hemolytic disease of the newborn in subsequent pregnancy[18]

During emergency transfusion of more than two units of type O Rh-negative, uncrossmatched whole blood, the patient probably cannot be switched to his or her blood type (A, B, or AB) as soon as the blood bank determines the correct blood type. Switching could cause major intravascular hemolysis of donor RBCs by increasing titers of transfused anti-A and anti-B. Continued use of O Rh-negative whole blood results only in minor hemolysis of recipient RBCs, with hyperbilirubinemia as the only complication. The patient must not be transfused with his or her correct blood type until the blood bank determines that the transfused anti-A and anti-B has decreased to levels that permit safe transfusion of type-specific blood.[18]

If type specific crossmatched blood is not available then alternative blood group that is available in the blood bank can be given as per following guidelines[13] (Table 4.7).

Development of artificial oxygen-carrying agents (so-called blood substitutes) that can be easily stored and readily given to patients with no crossmatching is expected to revolutionize resuscitation in emergency setting. These products are not currently approved for use by the FDA.

Table 4.7: Choice of alternative blood (in absence of patient's blood group)

Recipient's ABO type	*Alternative Blood Ist Choice (Given as Packed Cells)*	*Alternative Blood IInd Choice (Given as Packed Cells)*
O	None	None
A	O	None
A_2 with anti A_1	O	None
B	O	None
A_1B*	A or B	O
A_2B*	A or B	O

*In Group AB patients, Group A Blood as an alternate source is preferred over Group B blood, as anti-B in Group A is weaker than anti-A in Group B.

Are prophylactic platelets and fresh frozen plasma indicated in massive transfusion?

Recent studies[34,35,36,37,38] have stated that the ratio of plasma, platelets, cryoprecipitates to red blood cells may influence survival, though further studies are recommended for establishing guidelines for the optimal transfusion ratios.

The role of platelets during massive transfusion is under review. Earlier guidelines stated that platelets are not indicated in massive transfusion.[39] While thrombocytopenia may develop in massively transfused patients, administration of platelets should be reserved for the patient exhibiting microvascular bleeding, and a platelet count less than 50×10^9/L. Platelet transfusion may be necessary for patients with intermediate platelet counts ($50–100 \times 10^9$/L) if more bleeding is anticipated.[6,19] However, platelet counts ≥100,000/mm^3 for those with multiple trauma or CNS injury has been recommended as also when >1.5 to 2 blood volumes have been replaced with red cells. In the presence of microvascular bleeding, transfusion may be appropriate when counts are known or suspected to be <100,000/mm^3.[12]

Similarly role of FFP is under review. FFP also should not be administered prophylactically for massive transfusion.[6] In the massively transfused patient, clinical bleeding associated with coagulation factor deficiencies is unlikely until factor levels fall below 20 percent of normal. In the clinical setting, this usually does not occur until greater than one to one-half blood volume has been replaced and the PT and PTT are greater than 1.5 to 1.8 times control values based on studies performed in the trauma and cardiac surgical settings. FFP to be used when PT is 1.5 times control value, INR or APTT 1.8.[19]

It should be noted that however, prophylactic administration of platelets and FFP is not warranted but

Table 4.8: Compatible blood products for transfusion according to recipient's ABO Type

Recipient's ABO type	*Compatible plasma*	*Compatible RBCs*	*Compatible platelets First choice*	*Compatible platelets Second choice*
A	A, AB	A, O	A, AB	B, O
B	B, AB	B, O	B, AB	A, O
O	O, A, B, AB	O	O*	A, B, AB
AB	AB	AB, A, B, O	AB	A, B, O

*Although type O red blood cells (RBCs) do not have A or B antigens, the recipient's plasma contains anti-A and anti-B antibodies, which could reduce the recovery of transfused platelets bearing A or B antigens.

rather "pre-emptive" use is advocated. For example, if a patient is massively bleeding one does not wait until the platelet count drops to less than 50000/mm^3 or until the INR is greater than 2 to infuse the blood components as it can take 30 to 45 minutes to thaw FFP. Also, in a trauma patient with massive bleeding, a rise in the PT may be a "late" sign that the patient is developing a severe dilutional coagulopathy. In the event that the PT and PTT tests cannot be obtained in time, FFP 10 to 15 ml/kg may be administered empirically for correction of microvascular bleeding in patients transfused with more than one blood volume.[6]

If compatible blood products are not available other alternative compatible groups may be used as above (Table 4.8).

Another thing to consider is that the practice of administering packed RBCs and FFP to the same patient adds to the cost and doubles the infection exposure. Hence, when conditions are appropriate, whole blood should be given.[18]

It has to be emphasized that once bleeding is controlled and the patient is hemodynamically stable, the transfusion of blood and blood components should be guided by bedside and laboratory tests as well as the clinical status of the patient.

What are the causes of coagulopathy during massive transfusion?

Coagulopathy during massive transfusion is of multifactorial origin. Dilutional coagulopathy due to crystalloid, colloid and red blood cells administration, hypothermia, tissue hypoperfusion with resultant lactic acidosis and disseminated intravascular coagulopathy due to tissue trauma contribute to coagulation abnormalities. Coagulopathy associated with massive transfusion is clinically characterized by the presence of microvascular bleeding or oozing from the mucosa, wound, and puncture sites. The development of acidosis, DIC, hypothermia and, rarely, a hemolytic transfusion reaction may accompany massive transfusion and complicate the ability to effectively manage the coagulopathy. Thus, empiric formulas using ratios of various components to volume lost or administered are inadequate to treat or prevent coagulopathy related to massive transfusion. Treatment of the coagulopathy should include restoration of systemic perfusion, maintenance of normal temperature, resolution of acid-base abnormalities and blood component therapy when supported by abnormal laboratory tests in the setting of active bleeding.

How should coagulation be monitored during massive transfusion?

There is no single coagulation test that will give complete information on coagulation function during massive transfusion. The use of routine coagulation tests to evaluate coagulation factor deficiencies such as PT, PTT and thrombin time, have not reliably predicted perioperative bleeding, but they can identify patients with deficiencies of coagulation factors. These standard laboratory tests along with platelet count and fibrinogen level should guide the component therapy. The bleeding time is not predictive of perioperative bleeding and is rarely accessible in the operating room setting. The activated clotting time (ACT) is influenced by hypofibrinogenemia, hypothermia, use of anti-fibrinolytic agents and coagulation factor deficiencies and this lack of specificity limits ACT as a useful test during massive transfusion.

Whole blood clotting analysis, as assessed with the Thromboelastograph® (TEG) and Sonoclot®, measure the viscoelastic properties of blood as it clots. Although the clinical utility of use of a TEG during liver transplantation and cardiac surgery to guide component therapy has been demonstrated their usefulness during massive transfusion has not been established.

Is it necessary to utilize blood warmers during massive transfusion?

Warming of red cells or blood components should be limited to patients receiving massive transfusions (i.e. adults at rates > 50 ml/kg/hr, children at rates > 15 ml/kg/hr), or patients with clinically significant "cold" antibodies.[13,15] Intravenous fluids (500 ml or more) and

blood products should be warmed to 37°C using a fluid warming device.[40] Warming blood also enhances the intracellular transfer of potassium. Keeping the patient warm is usually more important than use of a blood warmer.

Only specially designed and regulated blood warmers with a visible thermometer and audible warning must be used. Blood must not be warmed by any other method, such as immersion in warm water, because of the risk of contamination through minute holes in the bag and lack of control of the temperature which may lead to dangerous hemolysis.

When massive blood transfusion is administered, hypothermia (temperatures below 35°C) is likely to occur. Low ambient temperature, large open wounds, use of irrigating fluids as in hemoperitoneum, initial infusion of room temperature fluids, and blood stored at 4°C contribute to hypothermia. The potential effects of hypothermia include ventricular dysrhythmias, shivering, increased oxygen consumption, cardiac arrest and citrate toxicity secondary to reduced metabolism of citrate and lactate. Hypothermia contributes to coagulopathy in the operating room by causing reversible platelet dysfunction, altering coagulation, and enhancing fibrinolysis.

What are the alternatives to minimize Allogenic Blood Transfusion during emergency? [41]

In theater: The best way to avoid the need for transfusion is by minimizing blood loss. A number of simple anesthetic and surgical techniques may be used to achieve this objective. They include:

Anesthetic Techniques

- Avoid hypertension and tachycardia due to sympathetic overactivity by ensuring adequate levels of anesthesia and analgesia.
- Avoid coughing, straining and patient maneuvres, which increase venous blood pressure.
- Avoid hypercarbia causing vasodilatation which will increase operative blood loss.
- Use regional anesthesia, such as epidural and spinal anesthesia where appropriate.
- Avoid hypothermia in the perioperative period.
- Controlled hypotension in experienced hands.

Surgical Techniques

- Training, experience and care of the surgeon are the most crucial factors.
- Meticulous attention to bleeding points—use of diathermy.
- Posture: The level of the operative site should be a little above the level of the heart, e.g. Trendelenberg position for lower limb, pelvic and abdominal procedures. Head-up posture for head and neck surgery. Avoid air embolism if a large vein above heart level is opened during surgery.
- Tourniquets wherever applicable: The inflation pressure of the tourniquet should be approximately 100 to 150 mmHg above systolic blood pressure of the patient. Tourniquet should not normally be used in patients with sickle cell disease or trait.
- Vasoconstrictors : Infiltration of the incision site with adrenaline (with or without local anesthetic agent).
- Postoperative period: Give adequate analgesia because the postoperative pain can cause hypertension and restlessness, which can aggravate bleeding, e.g. following limb surgery, postoperative elevation will reduce swelling, control venous blood loss and reduce pain. Give iron supplements (ferrous sulphate 200 mg tds) to restore hemoglobin level.

Intraoperative Blood Salvage

- Blood salvage is the collection of blood shed from the wound or body cavity and its subsequent infusion into the same patient. Contraindications to salvage include blood contaminated with bowel contents, bacteria, fat, amniotic fluid, urine, malignant cells and irrigation fluids. One should not reinfuse salvaged blood more than 6 hours old, since hemolysis of red cells is likely to be complete.
- Intraoperative blood salvage usually becomes cost-effective when 1,500 ml or more of blood is collected. It is also indicated if the patient has a rare blood type and adequate amounts of allogenic blood cannot be found. Intraoperative blood salvage is often acceptable to Jehovah's Witnesses, provided the salvaged blood remains in continuity with the patient's circulation.
- Methods of blood salvage:
  - Gauze filtration: Using aseptic technique, blood is collected with a ladle or small bowl and filtered through a gauze into a bottle containing anticoagulant.
  - Simple suction collection systems: Suction pressure should be as low as possible to avoid hemolysis of red cells.
  - Automated suction collection systems (cell savers): These are commercially available and are routinely used for many operation associated with substantial blood loss in some countries. They collect, anticoagulate, wash, filter and

resuspend red cells in crystalloid fluid prior to re-infusion.

- Limitations or complications of cell salvage include:
  - Hemolysis via a dysfunctional centrifuge or use of high suction pressures (>150 mm Hg)
  - Inadequate removal of either cellular debris or anticoagulant via dysfunctional or low volume (< 3 bowl volumes) processing which can lead to either DIC, a generalized systemic inflammatory response or hypotension (i.e. with citrate)
  - Bleeding, (i.e. with heparin) related to inadequate removal of blood thinner used.
  - Infection via contamination of disposable circuits or loss of platelets or coagulation factors if process volumes are extreme (> 2–3 litres) in the setting of use to process cardiotomy bleeding during cardiac surgery.

Antifibrinolytic Drugs

Drugs, which inhibit the fibrinolytic system and encourage clot stability, have been used in certain operations (e.g. repeat cardiac operations) to reduce operative blood loss, but are not widely used. Aprotinin, tranexamic acid are used when indicated.

Recombinant Factor VIIa

- The exact role of off-label use of recombinant activated factor VII to manage bleeding that cannot be controlled by conventional measures remains to be clarified.
- A recent systematic review[42] concluded that the application of rVIIa in patients with severe bleeding is promising and relatively safe (1–2% incidence of thrombotic complications). Though studies[43,44,45,46,47,48,49] demonstrating various uses have been published sound evidence from controlled trials is not available so far. Until more safety data is published, clinicians should use this agent judiciously as a "rescue therapy" in patients with life-threatening (i.e. > 1 liter/hour) bleeding is unresponsive to routine hemostatic therapy (e.g. adequate platelet transfusion (where needed) to keep the platelet count $\geq 50 \times 10^9$/L, FFP10–15mls/kg if Prothrombin ratio >1.5, minimum of 10 units of cryoprecipitate if Fibrinogen <1g/L, etc.), adequate correction of hypothermia, if present and where bleeding does not have an identifiable surgical source.
- rVIIa is supplied in 3 vial sizes (1.2 mg, 2.4 mg and 4.8 mg). Clear instructions about reconstitution of rVIIa are given on the product insert supplied with every vial and should be followed meticulously.
- Initial dose is 90 µg/kg by slow intravenous bolus, a second dose can be given after 2 hours if there is still significant blood loss. Up to three doses (each 2 hours apart) of 90 µg/kg can be given if a clear reduction in bleeding has occurred after 2 doses but significant bleeding continues. After 3 doses a full clinical review by the consultant hematologist/surgeon/physician/anesthetist is required to determine further therapy.

What are the electrolyte and acid-base problems associated with massive transfusion?

The potential problems resulting from blood transfusion are hyperkalemia, citrate toxicity, hypomagnesemia, acidosis and impaired oxygen-carrying capacity of hemoglobin.

Hyperkalemia may occur, due to the high extra cellular potassium concentration in stored red cell units. After 21 days of storage, plasma potassium concentrations may approach 19 to 30 mEq/L.[6,18] However, in adults hyperkalemia does not usually pose a problem. This is because and there is very little plasma in packed red blood cells and.potassium leaves the intravascular space of the recipient and rapidly moves intracellularly unless there is a starting hyperkalemic state as related to renal insufficiency.

For significant hyperkalemia to occur clinically, bank blood must be given at a rate of 120 ml/min or more. Current technology allowing rapid blood infusion at rates greater than 500 ml/min may limit the time available for potassium redistribution to occur resulting in hyperkalemia. Also, infants may receive disproportionately higher volumes of blood cells rapidly and will be at increased risk of acute hyperkalemia from stored blood. Also, if blood is pushed rapidly through small gauge intravenous cannula in infants, resultant hemolysis may cause hyperkalemia. If blood needs to be infused rapidly it is preferable to use large bore intravenous access and other physical methods such as increasing the distance between the blood bag and table, etc.

The electrocardiogram should be monitored in all patients for signs of electrolyte abnormality during rapid infusions. Hyperkalemia exacerbates the cardiovascular effects of hypocalcemia. If serum potassium level >6 mEq/L it should be treated with calcium gluconate 10%, glucose insulin regimens (10 units of insulin with 50cc 50% dextrose over 20–30 mins) and beta-2 agonists together with bicarbonate to correct acidosis. Early hemofiltration is likely to be required after the arrest of bleeding in the most severe cases.

Citrate toxicity occurs when ionized calcium is significantly reduced by citrate present in anticoagulant

preservative solutions. The liver rapidly metabolizes citrate, and endogenous calcium stores are released to help prevent a fall in ionized calcium. However, in states of shock, these mechanisms may be impaired. Citrate toxicity is potentiated by the rapid administration of large volumes of citrated blood components. Low ionized calcium levels may cause hypotension, narrowing of the pulse pressure, increased cardiac filling pressures, gross muscle tremors and electrocardiogram changes (prolonged Q-T interval). Toxicity is enhanced by hypothermia and a depressed hepatic function that slows the normal rate of citrate removal by the liver. Administration of exogenous calcium is indicated during massive transfusion when the measured ionized calcium is low (< 1.13 mmol/l[1]) and there is evidence of cardiovascular compromise (hemodynamic or prolonged Q-T interval) not attributable to other causes. 10% calcium gluconate may be given, but since calcium gluconate requires liver metabolism to release ionized calcium, the action may be delayed. Intravenous infusion of calcium chloride may be preferred where available. Citrate also has an affinity for the magnesium ion; and the occurrence of hypomagnesemia in the setting of massive transfusion has been reported.

Acidosis is more likely to be the result of inadequate treatment of hypovolemia than the effects of transfusion. Stored blood is acidic (pH of 6.6 to 6.9) due to the citric acid in the anticoagulant and the accumulation of carbon dioxide and lactic acid from erythrocyte metabolism. The acid load resulting from transfusion is rapidly reversed in the presence of normal tissue perfusion. Patients with adequate perfusion are likely to become alkalotic as the lactate and citrate are metabolically converted to bicarbonate by the liver. However, monitoring of blood pH is helpful to ensure normal arterial pH since the acid-base response is variable during transfusion. Prophylactic administration of alkalinizing agents such as sodium bicarbonate or tris-hydroxymethyl aminomethane (THAM) is unnecessary and may cause serious alkalosis and hypernatremia.

What should be done if urticaria and/or fever develop during transfusion?

Urticaria is usually due to an idiosyncratic or allergic reaction involving transfused allergens in plasma that interact with the patient's tissue mast cells, causing them to degranulate and release various inflammatory mediators. Most reactions are mild. The transfusion should be stopped, antihistamine such as diphenhydramine administered. Patient must be monitored to rule out a more serious reaction or anaphylaxis. Administration of epinephrine and corticosteroid may be required. The remainder of the unit can often be administered successfully at a slower rate.

Fever is associated with several types of transfusion reactions and may be due to Febrile nonhemolytic transfusion reaction (FNHTR), Hemolytic transfusion reaction (HTR) or administration of a bacterially contaminated blood component.

Febrile nonhemolytic reaction is typically manifested by a temperature elevation of ≥ 1°C or 2°F occurring during or shortly after a transfusion and in the absence of any other pyrexic stimulus. FNHTRs are immunologically mediated reactions involving leukocyte antibodies in the patient's plasma (stimulated by previous transfusions or pregnancy) and antigens on donor leukocytes, causing release of endogenous pyrogens. Cytokines released during component storage are also implicated in causing FNHTRs.

If red blood cells are being administered, an HTR should be considered. If platelets are being transfused, bacterial contamination is more likely. Fever due to an HTR or bacterial contamination tends to occur following infusion of a small amount of blood and is rarely the only sign of a reaction. Hypotension accompanying fever should raise suspicion of an HTR or bacterial contamination.

Antipyretics usually provide effective symptomatic relief. Patients, who experience repeated, severe febrile reactions may benefit from receiving leukocyte reduced components or use of leukocyte filters.

What are the manifestations of hemolytic transfusion reactions?

Hemolytic transfusion reactions (HTRs) involve lysis of red blood cells. The hemolysis can occur intravascularly or extravascularly and can be caused by immunologic incompatibility between the donor and recipient or result from nonimmune mechanisms.

Immune Reactions

Most serious acute HTRs are caused by transfusion of ABO-incompatible red blood cells. The most common cause of severe transfusion reactions is patients being given the wrong blood. This may result from an incorrect sample being sent to the laboratory, a mix up in the blood bank, but most frequently the wrong blood being transfused at the patient level.

Even a small volume (10–20 ml) of incompatible blood can cause a severe reaction and larger volumes increase the risk. Acute transfusion reactions occur during or shortly after (within 24 hours) the transfusion. Rapid recognition and management of the reaction may save the patient's life.[50]

Signs and symptoms include chills, fever, chest and flank pain, and nausea. Patients often voice concerns that something is wrong with the transfusion. In the anesthetized patient, the only signs may be hemoglobinuria, a bleeding diathesis and unexplained hypotension.

Management

- Stop transfusion and treat as anaphylaxis.
- Replace infusion set with normal saline.
- Maintain airway, give high flow oxygen.
- If there is severe hypotension or bronchospasm give Inj adrenaline either IV (1:10,000 solutions in 0.5–1ml Inj aliquots) or subcutaneously (1:1000 as 0.01 ml/kg body weight). Consider IV corticosteroids and bronchodilators.
- Give IV diuretic, e.g. frusemide 1 mg/kg.
- Immediately notify blood bank and send the blood pack with the infusion set, fresh urine sample, fresh venous blood sample (1 clotted and 1 anticoagulated) from a vein opposite the infusion site.
- Assess and treat hypotension with saline 20 to 30 ml/kg over 5 minutes. Inotropes (e.g. dopamine) may be required.
- Monitor urine output. A falling urine output or a rising K+, urea, or creatinine are indicative of acute renal failure. Ensure a normal blood pressure (a CVP measurement may be required) and consider further frusemide. Renal dialysis may be required.
- If bacteremia is suspected (rigors, fever, collapse, and no evidence of a hemolytic reaction) IV broad spectrum antibiotics should be given.
- Laboratory evaluation includes the direct antigen test, urine and plasma hemoglobin determinations, other tests verifying hemolysis (elevations in LDH, bilirubin, and/or undetectable haptoglobin) and baseline coagulation studies (platelet count, prothrombin time, activated partial thromboplastin time and fibrinogen level).

Immune extravascular reactions, often referred to as delayed reactions, occur following transfusion of red blood cells containing an antigen other than ABO to a patient with an undetected alloantibody. Estimated risk of delayed HTR is much higher than acute HTR at about 1 in 1000 to 9000 units. The transfused red blood cells may survive initially, but may hemolyse within days to weeks. Laboratory findings may include a positive direct antiglobulin (Coombs) test and an unexplained rise in bilirubin. No treatment is generally indicated.

Nonimmune Reactions

Nonimmune hemolysis is generally preventable with strict adherence to proper handling and administration of blood components. Exposure to hypotonic or hypertonic solutions (e.g. 5-percent or 40–50% dextrose, respectively) thermal injury during blood transport, storage, processing (i.e. use of excessive suction exceeding 150 mm Hg for cell salvage systems or a dysfunctional cell salvage centrifuge) or administration, and inadequate deglycerolization of frozen Red Blood Cells can all cause hemolysis.

What other adverse reactions may occur as a result of transfusion?

Transfusion is associated with a number of other acute and delayed reactions, in addition to infection, fever, urticaria and hemolysis.

- *Allergic and Anaphylactic Reactions* involve interaction between an allergen (usually a protein in the plasma of the transfused blood component to which the recipient was previously sensitized) and IgE antibody present on the surface of mast cells and basophils in the tissues and circulation of the recipient. The severity ranges from mild urticaria to bronchospasm, laryngeal edema, severe hypotension and death — in general, the shorter the interval between initiation of transfusion and the onset of symptoms, the more severe the reaction. Minor allergic reactions occur in 1 per 20 to 2500 transfusions depending on the components used, definition of reaction and the studied population. Transfusion must be discontinued immediately if an anaphylactic reaction is suspected. Treatment is the same as for other anaphylactic reactions: epinephrine, diphenhydramine and corticosteroids, in addition to appropriate fluid therapy and airway management.
- *Transfusion Associated Circulatory Overload (TACO)* resulting in pulmonary edema can accompany transfusion of any component at a rate more rapid than the recipient's cardiac output can accommodate. Whole Blood creates more of a risk than Red Blood Cells because the transfused plasma adds volume without increasing oxygen-carrying capacity. Patients with chronic anemia have increased plasma volumes and are at increased risk for circulatory overload.
- *Transmitted Infections and Bacterial Contamination.*
- *Transfusion-Related Acute Lung Injury (TRALI)* is currently the leading cause of transfusion-related

death (30–50% deaths per million units) via FDA reporting mechanisms. Clinical presentation of TRALI, in its severe form, is indistinguishable from adult respiratory distress syndrome (ARDS) and is characterized by acute onset, bilateral pulmonary infiltrates and hypoxia without evidence of CHF. In comparison to ARDS, TRALI is characterized by a much shorter time interval between exposure to the precipitating risk factor (transfusion) and onset of clinical manifestations. TRALI usually develops within 6 hours (most often less than 2 hours) of a transfusion, usually resolves within 24 to 48 hours, and has a mortality rate of approximately 5 to 10 percent. Whereas, ARDS does not usually develop until at least 24 hours after exposure to one of its risk factors, has a duration often longer than 72 hours, and a mortality approaching 30 to 60 percent.

It can occur after the transfusion of a variety of blood components such as red blood cells, platelets and FFP but is most often seen after transfusion of the plasma-containing blood components such as FFP and platelets. Symptoms of TRALI can be confused with other transfusion and nontransfusion-related events such as anaphylaxis, hemolysis, circulatory overload and cardiac failure. Most cases of TRALI are due to passive transfer of donor-related anti-leukocyte antibodies directed at HLA or granulocyte-specific antigens on the patient's leukocytes. It can also be due to inflammatory response to the bioactive factors or white cell priming lipid-ligand released by platelets or several reactive lipid-like substances accumulating in red blood cells or platelets during storage. Supportive therapy in the form of high FiO_2, endotracheal intubation with ventilatory support, volume resuscitation and vasopressor support is needed.

- *Post-transfusion Purpura (PTP)* is a rare disorder characterized by severe thrombocytopenia 5 to 10 days after transfusion in a patient sensitized by prior transfusion or pregnancy. In most cases, PTP follows administration of Red Blood Cells. The estimated risk is around 1 in 150,000 to 300,000 red cell units. Platelet counts are often less than 10×10^9L. Patients usually recover spontaneously, although corticosteroids and intravenous immune globulin may be administered. The pathogenesis is unclear, but PTP is presumably related to the development of a platelet specific antibody in patients who are deficient of a common platelet antigen (e.g. PLA-1) following transfusion.
- *Transfusion-Associated Graft-versus-Host Disease (TA-GVHD)* occurs when immuno-competent donor lymphocytes are transfused to an HLA-incompatible recipient or host (e.g. immunocompromised patients or patients receiving a blood donation from a relative) who is immunologically incapable of eliminating the donor cells. Among the immunocompromised patients at risk are individuals with congenital cell-mediated immunodeficiencies or Hodgkin's disease, recipients of bone marrow transplants and patients receiving immunosuppressive therapy. Immunocompetent recipients of directed donations from biologic relatives may also develop TA-GVHD. Clinical manifestations are usually evident within 8 to 10 days after transfusion and include fever, skin rash, diarrhoea, liver dysfunction and pancytopenia. Death usually occurs within three to four weeks secondary to bone marrow failure. Irradiation of blood components virtually eliminates the risk of TA-GVHD in susceptible patients.
- Other adverse events and complications of transfusions include immunomodulation, alloimmunization, serologic reactions, or acid/based abnormalities and hemochromatosis.

TREATMENT OF OTHER FACTOR DEFICIENCIES

Patients with hemophilia A and B or congenital Factor 13 deficiency coming for emergency surgery may need specific factor infusions apart from cryoprecipitate and FFP. Dosages are decided depending on the pathology (Table 4.9).

A reasonable dosage calculation guide for factor VIII is provided by the following formula:

FVIII dose (U) = body weight (kg) × desired FVIII increase (%) × 0.5 U/kg

Approximately 50% of the initial dose is given as the second dose approximately 8 hours after the first; all subsequent doses are given every 12 hours. Continuous infusion of factor VIII can be used for treating patients after joint replacements or CNS bleeding, in which a continuous, steady level is desired. This can be achieved by an initial bolus dose, as discussed, followed by a maintenance infusion of 150 U/h, with monitoring of levels for adequacy.

Reasonable dose of Factor IX is body weight (kg) × desired FIX increase (%) × 1 to 1.2 U/kg based on the preparation.

Concomitant administration of EACA can help reduce the dose of concentrate that is required to treat such bleeding. For dental extractions, oral or intravenous EACA at 5 g is given, followed by

Table 4.9: Treatment of other factors deficiencies

Type of hemorrhage	*Desired FVIII-C and FIX Activity*	*Dose and duration of FVIII therapy*	*Dose and duration of FIX therapy*
Minor uncomplicated hemarthroses	20-50%	20-30 µ/kg IV every 12-24 hrs in adults, 6-8 hrs in children for 1-2 d	1-2 days of 1-1.2 µ/kg per % increase desired
Superficial large hematomas	Additional 15-25% for 2-3 days	Doses as required	
Moderate hematoma with dissection, target joint hematomas	25-50%, higher if clinically required	20-30 µ/kg IV every12-24h, 6-8 hrs in children for 3-7 d (shorter time for oral hemorrhages; higher dose for haematuria)	3-7 days of 1-1.2 µ/kg per % increase desired (2-5 days of 1-1.2 µ/kg per % increase desired in oral hemorrhages)
Oral/mucosal hemorrhages and epistaxis hematuria			
Dental extraction(s), oral surgery	50-100%	50-60 µ/kg IV initial dose followed by 20-30 U/kg every 8-12h for 2-5 d, shorter dosing interval in children	2-5 days of 1-1.2 µ/kg per % increase desired
Major pharyngeal/retropharyngeal Retroperitoneal GI bleeding CNS bleeding surgery Joint, bone surgeries	~50-100% until bleeding is controlled; then, gradually decrease the dosage to the minimum that is required to prevent rebleeding	50-60 µ/kg IV initial dose followed by 20-30 U/kg every 8-12h for 2-7 d, shorter dosing interval in children Continue for 2-6 weeks depending on healing	7-10 days of 1-1.2 µ/kg per % increase desired (5-10 days of 1-1.2 µ/kg per % increase desired in oral hemorrhages.

References are from Harrison and Stoelting

maintenance dose of 1 g/h, for 5 to 7 days, with a gradual taper.

Factor XIII-deficient patients can be treated with FFP, cryoprecipitate, or a plasma-derived factor XIII concentrate, Fibrogammin P. Acute hemorrhage should be treated with an infusion of 50 to 75 U/kg body weight. Factor XIII has a long circulating half-life of 7 to 12 days, and adequate hemostasis is achieved with even very low plasma concentrations (1%–3%).

CHECKLIST FOR GIVING BLOOD AND BLOOD COMPONENTS

Before you prescribe blood ask yourself:

- What improvement am I aiming to achieve in this patient's clinical condition? Can I reduce blood loss to minimize this patient's need for transfusion?
- Have I given other treatment (e.g. intravenous replacement fluids, oxygen) before making the decision to transfuse blood?
- What are the specific clinical or laboratory indications for transfusion?
- What are the risks of transmitting HIV, hepatitis, syphilis or other infectious agents through the blood products that are available for this patient?
- Do the benefits of transfusion outweigh the risks of blood transfusion for this particular patient? What other options is there if no blood is available in time?
- In the postoperative period will a trained person monitor and respond immediately if any adverse transfusion reactions occur?
- Have I recorded my decision and reasons for transfusion on the patient's chart and Blood Request Form?
- Finally, the most important question you should ask before making a decision— would I accept this transfusion in this clinical condition, if this blood was for me or my child?

Before you Give Blood - check

Correct patient?: Check patient identity against indoor paper and transfusion form/issue slip. If possible confirm identity from patient.

Correct group?: The blood group on the blood component pack label must be the same as on the laboratory produced label attached to the blood component and the transfusion/issue slip.

Correct blood?: Check donor blood according to transfusion form/issue slip. Check that all tests for infectivity are negative.

Correct date?: Check the expiry date of the component—unless a specific expiry

time is stated, the component expires at midnight of the date shown.

- The final administration check should always be conducted next to the patient (not in a remote clinical room or at the nursing station).
- Once all checks have been successfully completed, the transfusion should be started immediately. If the checking process is interrupted, the entire process should restart from the beginning.
- The healthcare professional who is going to administer the unit should himself undertake all these checks.
- Transfusion should only take place if the patient identification details on the blood component pack and the patient identification number (or equivalent) match. If they do not, the transfusion laboratory should be informed and the component must not be transfused until there has been an investigation and any discrepancies resolved. A repeat pre-transfusion blood sample may be required.
- If the healthcare professional is unsure that the blood component issued is correct, for example, an unexplained difference between blood groups in the donor and recipient or whether special requirements have been met, they should check with the hospital transfusion laboratory before starting the transfusion.

FUTURE GOALS

- Training and education of both junior and senior staff is imperative to successful implementation.
- Computerized systems are needed in the longer term and will assist with monitoring and evaluation.
- A national consumer information pamphlet should be developed. The pamphlet should include the risks and benefits of transfusion and frequently asked questions and answers.
- Comprehensive education resources must be developed appropriately and should include:
  - Pocket cards with transfusion guidelines;
  - Posters for display in hospital and clinician waiting rooms, and
  - Summary of guidelines that are followed in practise.
- Concerns should be raised about the resource implications of implementing the guidelines. Without the appropriate funding, the guidelines cannot be successfully implemented.
- The responsibility of implementing the Guidelines is a shared one and requires the support of health departments, professional colleagues, clinicians and laboratory staff.

As anesthesiologists we can make an impact on clinical use of blood beyond the care of our own patients. However small our contribution, we can play an important part in creating the conditions in which the appropriate clinical use of blood is possible. While progress may initially be slow, a regular and systematic review of transfusion practices should demonstrate the effectiveness of change and point to areas where further improvement may be needed.

REFERENCES

1. Wade M, Sharma R, Manglani M. Rational use of blood components — an audit. Indian J Hematol Blood Transfus 2009; 25(2):66-9.
2. Mirzamaniab N, Molanaab A, Pooraniab E. Evaluation of appropriate usage of fresh frozen plasma: Results of a regional audit in Iran. Transfus Apher Sci 2009;40(2); 109-13.
3. Matot I, Einav S, Goodman S, etal. A survey of physicians' attitudes toward blood transfusion in patients undergoing cesarean section. Am J Obstet Gynecol 2004;190(2):462-7.
4. Marti-Carvajal AJ, Munoz-Navarro SR, Pena-Marti GE, et al. An audit of appropriate use of blood products in adult patients in a Venezualan general university hospital. Int J Qual Health Care 1999;11(5):391-5.
5. Schofield WN, Rubin GL , Dean MG. Appropriateness of platelet, fresh frozen plasma and cryoprecipitate transfusion in New South Wales public hospitals. MJA 2003; 178:117-21.
6. Brauer SD, Cywinski JB, Downes K, Johnson LN, Kindscher JD, et al. Questions and Answers about Blood Management, 4th edn. Committee on Transfusion Medicine, American Society of Anesthesiologists 2008.
7. Practice Guidelines for Perioperative Blood Transfusion and Adjuvant Therapies: An Updated Report by the American Society of Anesthesiologists. Task Force on Perioperative Blood Transfusion and Adjuvant Therapies Anesthesiology 2006;105:198-208.
8. Harris AM, Atterbury CLJ, Chaffe B, Elliott C, et al; Guideline on the Administration of Blood Components, British Committee for Standards in Haematology. British Society of Haematology 2009;3-56.
9. Stainsby D, MacLennan S, Thomas D, et al. British Committee for Standards in Haematology—Guidelines on the management of massive blood loss. British Journal of Haematology 2006;135:634-41.
10. British Committee for Standards in Haematology. Blood Transfusion Task Force. Guidelines for the use of platelet transfusions. Br J Haematol 2003;122:10-23.
11. Blood transfusion and the anaesthetist blood component therapy. Published by the Association of Anaesthetists of Great Britain and Ireland December 2005.
12. Miller Y, Bachowski G, Benjamin R, Eklund DK, et al. Practice guidelines for blood transfusion: A compilation

from recent peer reviewed literature, 2nd edn. American National Red Cross April 2007;Section 1: 7-62.
13. Guidelines for setting up blood storage centres at first referral units, Maternal health division, Ministry of family Welfare, Government of India 2003.
14. Circular of Information for the Use of Human Blood and Blood Components. Prepared jointly by AABB, the American Red Cross, America's Blood Centers, and the Armed Services Blood Program. Revised Dec 2009.
15. Cherian MN, Emmanuel JC. Clinical use of blood. World Health Organisation-World Anaesthesia, Issue 14 2002; Article 6:1.
16. Clinical use of blood in Medicine, Obstetrics, Paediatrics, Surgery and Anaesthesia, Burns. World Health Organisation. Accessed from http://www.who.int/bloodsafety/clinical_use/en/ on 1 May 2010.
17. Shortt J, Polizzotto MN, Waters N, Borosak M, et al. Assessment of the urgency and deferability of transfusion to inform emergency blood planning and triage: The bloodhound prospective audit of red blood cell use. Transfusion 2009; 49: 2296-303.
18. Miller RD. Transfusion Therapy. In: Miller's Anaesthesia, Miller RD, Eriksson LI, Fleisher Lee A, (eds). 7th edn 2010. Philadelphia: Churchill Livingstone Elsevier;USA.
19. Nuttall GA. When are platelet/plasma transfusions indicated? In: Evidence-based practice of Anesthesiology. Fleischer LA (ed), 2nd edn,Saunders elseiver, Philadelphia 2009;166.
20. http://www.mdsr.ecri.org/summary/detail.aspx?doc_id=8269: Suggested guidelines for blood warmer use accessed on May 1,2010.
21. Slichter SJ, Christoffel T, Corson J, et al. Effects of pretransfusion warming of platelets to 35 degrees C on post-transfusion platelet viability. Transfusion 2009; 49(11):2319-25.
22. Murdock J, Watson D. Dorée CJ, et al. Drugs and blood transfusions: Dogma- or evidence-based practice? Transfus Med 2009;19(1):6-15.
23. Yousef HM, Padmore RE, Neurath DD, Rock GA. The effect of patient-controlled analgesia on coadministered red blood cells. Transfus 2006;46,372-76.
24. Huber-Wagner S, Qvick M, Mussack T, et al. Massive blood transfusion and outcome in 1062 polytrauma patients: A prospective study based on the Trauma Registry of the German Trauma Society. Vox Sang 2007; 92(1):69-78.
25. Perel P, Roberts I. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2007;17,(4):CD000567.
26. Mathru M, Solunki DR, Woodson LC. Splanchnic: Oxygen consumption is impaired during severe acute normovolemic anemia in anesthetized humans. Anesthesiology 2006;105:37.
27. Bunn F, Trivedi D, Ashraf S. Colloid solutions for fluid resuscitation. Cochrane Database Syst Rev 2008; (1):CD001319.
28. Human albumin administration in critically ill patients: Systematic review of randomised controlled trials Cochrane Injuries Group Albumin Reviewers BMJ 1998; 317(7153):235–40.
29. Sandro B. Rizoli. Crystalloids and Colloids in Trauma Resuscitation: A Brief Overview of the Current Debate. J Trauma 2003;54:S82-S88.
30. McCunn M, Dutton R. End-points of resuscitation how much is enough? Curr Opin Anaesthesiol. 2000;13(2):147-53.
31. Inaba K, Teixeira PG, Shulman I, et al. The impact of uncross-matched blood transfusion on the need for massive transfusion and mortality: Analysis of 5,166 uncross-matched units. J Trauma. 2008;65(6):1222-6.
32. Schwab CW, Shayne JP, Turner J. Immediate trauma resuscitation with type O uncrossmatched blood: A two-year prospective experience. J Trauma. 1986;26(10):897-902.
33. Dutton RP, Shih D, Edelman BB, et al. Safety of uncrossmatched type-O red cells for resuscitation from hemorrhagic shock. J Trauma 2005;59(6):1445-9.
34. Phan HH, Wisner DH. Should we increase the ratio of plasma/platelets to red blood cells in massive transfusion: What is the evidence? Vox Sang. 2010;98(3 Pt 2):395-402.
35. Shaz BH, Dente CJ, Nicholas J, et al. Increased number of coagulation products in relationship to red blood cell products transfused improves mortality in trauma patients. Transfusion 2010;50(2):493-500.
36. Teixeira PG, Inaba K, Shulman I, et al. Impact of plasma transfusion in massively transfused trauma patients. J Trauma 2009;66(3):693-7.
37. Zehtabchi S, Nishijima DK. Impact of transfusion of fresh-frozen plasma and packed red blood cells in a 1:1 ratio on survival of emergency department patients with severe trauma. Acad Emerg Med 2009;16(5):371-8.
38. Maegele M, Lefering R, Paffrath T, et al .Red-blood-cell to plasma ratios transfused during massive transfusion are associated with mortality in severe multiple injury: A retrospective analysis from the Trauma Registry of the Deutsche Gesellschaft für Unfallchirurgie. Vox Sang 2008; 95(2):112-9.
39. Stainsby D, MacLennan S, Hamilton PJ. Management of massive blood loss: A template guideline. British Journal of Anaesthesia 2000;85(3):487-91.
40. Rao SM. Blood conservation—how practical? Indian J. Anaesth 2002; 46(3):168-74.
41. Inadvertent perioperative hypothermia. The management of inadvertent perioperative hypothermia in adults. NICE clinical guideline 65 April 2008.
42. Stanworth SJ, Birchall J, Doree CJ, et al. Recombinant factor VIIa for the prevention and treatment of bleeding in patients without haemophilia [review]. Cochrane Database Syst Rev 2007;(2):CD005011.
43. Alten JA, Benner K, Green K, et al. Pediatric Off-label. Use of Recombinant Factor VIIa. Pediatrics 2009;123(3); 1066-72.

44. Elliott J, Smith M. The Acute Management of Intracerebral Hemorrhage: A Clinical Review. Anesth. Analg 2010; 110: 1419-27.
45. Levi M. Use of recombinant factor VIIa in the perioperative period. Hamostaseologie 2009;29(1):68-70.
46. Levi M, Peters M, Buller HR. Efficacy and safety of recombinant factor VIIa for treatment of severe bleeding: A systematic review. Crit Care Med 2005;33:883-90.
47. Boffard KD, Riou B, Warren B, et al. Recombinant factor VIIa as adjunctive therapy for bleeding control in severely injured trauma patients: Two parallel randomized, placebo-controlled, double-blind clinical trials. J Trauma 2005;59:8-15.
48. Bosch J, Thabut D, Bendtsen F, et al. Recombinant factor VIIa for upper gastrointestinal bleeding in patients with cirrhosis: A randomized, double-blind trial. Gastroenterology 2004;127:1123-30.
49. Diprose P, Herbertson MJ, O'Shaughnessy D, et al. Activated recombinant factor VII after cardiopulmonary bypass reduces allogeneic transfusion in complex non-coronary cardiac surgery: Randomized double-blind placebo-controlled pilot study. Br J Anaesth 2005;95:596-602.
50. Janatpour KA, Kalmin ND, Jensen HM, et al. Clinical Outcomes of ABO-Incompatible RBC Transfusions. Am J Clin Pathol 2008;129:276-81.
51. Robert I. Handin. Disorders of Hemostasis. In: Harrison's principles of Internal Medicine, Fauci AS, Kasper DL, Braunwald E, Hauser SL, (Eds) 17th ed. McGraw Hill Companies; USA 2008.
52. Rinder CS. Haematological Disorders. In: Stoelting's Anesthesia and Co-Existing Disease, Hines RL, Marschall KE, (Eds) 5th edn. Philadelphia: Churchill Livingstone Elsevier; USA 2008

Section II

Cardiovascular Emergency Procedures

5

Anesthesia for Emergency Myocardial Revascularization

Manju N Gandhi, Aparna A Nerurkar

KEY POINTS

- Cardiovascular disease (CVD) is one of the major causes of death in India and emergency myocardial revascularizations are showing an increasing trend.
- Patient may present as acute or complicated myocardial infarction (MI) or postangiography/angioplasty complication
- Mechanical complications like cardiogenic shock, rupture of the ventricular septum or free wall, acute mitral regurgitation, pericarditis, tamponade, and/or electrical complications like arrhythmia and conduction blocks may be present.
- Treatment options include percutaneous coronary interventions (PCI), conventional coronary artery bypass grafting (CABG) on cardiopulmonary bypass (CPB) or off-pump CABG or hybrid procedures.
- Stabilization includes MONA morphine, oxygen, nitrates and aspirin with the aim of optimizing oxygenation, decreasing cardiac workload, controlling pain, prevention and treatment of complications, rapid intravenous thrombolysis and/or interventional or surgical revascularization.
- The goal for patients with ST elevation myocardial infarction (STEMI) should be to achieve a door-to-drug time of within 30 minutes and a door-to-balloon time of within 90 minutes.
- Beta blockers are administered for heart rate (HR) control. Addition of intravenous platelet glycoprotein (GP) IIb/IIIa-receptor antagonists improves both early and late outcomes.
- Time is of essence. Consider risks versus benefits of all interventions—monitoring devices and lines, therapeutic drug infusions, heparinization and use of anesthetic agents.
- Aggressively treat ischemia/ dysrhythmias. Continue antianginal therapy—Nitroglycerin (NTG) infusion during acute myocardial ischemic event. Maintain coronary perfusion pressure with phenylephrine or norepinephrine boluses/infusions. Ionotropic/vasopressor infusions need to be maintained.
- Continue blood pressure (BP) monitoring, pulse oximetry and electrocardiographic (ECG) monitoring in transport. Triggers like ECG or BP should be maintained for auqmentation of intraoperative balloon pump (IABP).
- Continue heparin infusion until sternotomy to reduce risk of worsening thrombosis.
- In arrest situations go directly to CPB with doubling or tripling of heparin dose to ensure adequate heparinization. Lines can be taken while coming off bypass.
- If chest pain and ischemic ECG changes have resolved—cautious insertion of monitoring lines may be preferred. If ischemia is present induction precedes insertion of monitoring lines. If changes subside after anesthetic induction, insertion of monitoring lines can be done. If hemodynamic changes persist after induction, proceed to CPB emergently.
- Fibrinolytic/antiplatelet agents given in the catheterization laboratory may lead to increased bleeding. Adequate units of blood and blood products should be available. Cell salvage devices may be useful.
- Choice and dosage of anesthetic agents should be governed by the hemodynamic profile of the patient and the pharmacodynamics of the particular agent.
- Tranexamic acid or aprotinin can be given to control postoperative bleeding.
- Prolonged postoperative stay is anticipated depending on preoperative status.

INTRODUCTION

Cardiovascular disease (CVD) is one of the major causes of death in India.[1] The deaths due to CVD in India were 32 percent of all deaths in 2007.[2] According to recent estimates, cases of CVD may increase from about 2.9 crores in 2000 to as many as 6.4 crores in 2015; deaths from CVD will also more than double.[3] Thus, although a relatively new epidemic in India, it has quickly become a major health issue. Indians who have acute coronary syndromes have a higher rate of STEMI than do patients in developed countries.[4] Given the burden of cardiovascular disease with recent trends of young infarcts, the role of emergency revascularization especially percutaneous transluminal coronary angioplasty (PTCA) is increasing. Candidates unsuitable for thrombolytic therapy, PTCA or patients with failed PTCA/thrombolysis and medical management end-up requiring emergency coronary artery bypass grafting. Recent trends point to off pump interventions even for such high-risk patients.[5]

INDICATIONS FOR EMERGENCY REVASCULARIZATION

Some of the indications as per recent protocols[6] are:

- STEMI (ST elevation myocardial infarction) within 12 hours from onset of symptoms
- STEMI within prior 12 to 24 hours with severe heart failure (HF), persistent ischemic symptoms, or hemodynamic or electrical instability
- STEMI with presumed successful treatment with fibrinolysis with evidence of HF, recurrent ischemia, or unstable ventricular arrhythmias and single vessel involvement
- Asymptomatic STEMI with presumed successful treatment with fibrinolysis with depressed LVEF (left ventricular ejection fraction), three-vessel coronary artery disease (CAD) for semielective revascularization
- Unstable angina/non ST elevation myocardial infarction (UA/NSTEMI) with high-risk features for short-term risk of death or nonfatal MI for revascularization of 1 or more arteries (high-risk features include at least 1 of the following: Accelerating tempo of ischemic symptoms in preceding 48 hours, prolonged ongoing (greater than 20 minutes) rest pain, pulmonary edema, most likely due to ischemia, new or worsening mitral regurgitation murmur, S3 or new/worsening rales over the lungs, hypotension, bradycardia, tachycardia. Age greater than 75 years, angina at rest with transient ST-segment changes greater than 0.5 mm, bundle-branch block, new or presumed new, sustained ventricular tachycardia, elevated cardiac troponin T, troponin I, or creatine kinase-MB (e.g. troponin T or I greater than 0.1 ng per ml)
- Patients with acute myocardial infarction (STEMI or NSTEMI) with evidence of cardiogenic shock for revascularization of 1 or more coronary arteries.

PATIENT PROFILE

History

Patients present with angina-acute on chronic or unstable angina. The chest pain is typically present either in the anterior precordium described as tightness, pressure or squeezing, with radiation to the jaw, neck, arms, back, and epigastrium with sweating. Angina may be atypical. There may be dyspnea, which may accompany chest pain or occur as an isolated complaint. Dyspnea may be the patient's anginal equivalent. In geriatric or diabetic patients, dyspnea may be the only complaint. It indicates poor ventricular compliance in the setting of acute ischemia. Patients may have tachypnea and cough. Nausea with or without vomiting, abdominal pain, or both often present in infarcts involving the inferior or posterior wall. Patients may be anxious with a sense of impending death. There may be giddiness or altered mental status especially in geriatric cases.

MI may be "silent" not causing the classic symptoms described above. It is more common in elderly and diabetics with 15 to 35 percent of all MIs occurring as silent events. Apart from these two conditions, a high index of suspicion should be maintained for MI especially when evaluating women, patients with dementia and those with a history of heart failure. It may manifest as fatigue, rapid onset pulmonary edema, cardiac arrhythmias, syncope, feeling of indigestion or jaw pain. Patients with a permanent pacemaker need to be specially investigated as paced ventricular contractions impair recognition of STEMI by 12-lead ECG.

Other History

- Starvation status is to be noted—patients are likely to be full stomach candidates in view of emergency surgery
- History of other risk factors like age, prior MI , IHD, hypertension, diabetes, smoking, alcohol intake, renal or hepatic dysfunction, stroke, convulsions, asthma, COPD need to be assessed. Details of last medication schedule—including antiplatelet agents,

anticoagulants, ACE inhibitors, beta blockers, etc should be noted.
- Pastsurgical and anesthetic history with complications, if any, need to be noted.
- Drug allergies, allergy to fish (protamine reactions) need to be noted.

General Examination

- Patients with ongoing symptoms usually lie quietly in bed. There may be sweating and pallor. Low grade fever may be present. Jugular venous pressure may be elevated in congestive heart failure. With right ventricular failure, cannon jugular venous a waves may be noted.
- Pulse may be feeble, collapsing in cardiogenic shock, congestive heart failure. Dysrhythmias may present as an irregular heartbeat or pulse. Hypertension may reflect elevated catecholamine levels due to anxiety, pain, or exogenous sympathomimetics and may precipitate MI. Hypotension may indicate ventricular dysfunction due to ischemia. Hypotension in the setting of MI usually indicates a large infarct secondary to either decreased global cardiac contractility or a right ventricular infarct. Patients have tachypnea and may have rales with reactive rhonchi due to congestive heart failure.
- Pansystolic murmur of mitral regurgitation due to papillary muscle ischemia or necrosis may be present. Third heart sound (S3) may be present. A forth heart sound is a common finding in patients with poor ventricular compliance that is due to preexisting heart disease or hypertension.

Airway Examination

- Routine airway examination should be performed with assessment of mouth opening, neck movements, dentition and thyromental distance. Patients are generally cases of difficult airway due to risk-factors of obesity, diabetes, old age and obstructive sleep apnea.

Complicated Myocardial Infarction (MI)

- **Arrhythmias:** Tachyarrhythmias like sinus tachycardia, missed beats of PVCs (premature ventricular complexes) or bradyarrhythmias due to Type II blocks or bundle branch blocks may present as palpitations. NSVT/VT (nonsustained ventricular tachycardia/ventricular tachycardia) with or without pulse are indicative of ventricular dysfunction or failure.
- **Cardiogenic shock:** Patient may be obtunded, on multiple inotropes or have assist devices in place like pacemaker or IABP. Patient may be on assisted or controlled ventilatory therapy. Patients should undergo thrombolysis or PCI, placement of an intra-aortic balloon pump, or CABG.
- **Mitral regurgitation:** This may occur acutely when ischemia or an infarct of the papillary muscle occurs. It usually presents as flash pulmonary edema and hypotension. Papillary muscle rupture may require valve repair with ECG. Ischemia often responds to medical therapy and thrombolysis.
- **Congestive heart failure:** It can be due to systolic or diastolic dysfunction in MI. The severity of the heart failure and systolic dysfunction depends on the extent of the infarct and the presence of any other complications, such as acute mitral regurgitation. Treatment may include nitrates, morphine, diuretics, and ACE inhibitors.
- Right ventricular infarct: Occurs in the setting of an inferior wall infarction. These patients have profound hypotension and worsen with nitroglycerin as they are preload dependent. Careful volume loading is the treatment of choice.
- **Ventricular rupture:** Occurs commonly in the interventricular septum or the left ventricle free wall following acute massive infarction. Rupture represents a catastrophic event with mortality rates greater than 90 percent. Cardiogenic shock or pericardial tamponade may be the presenting features. Echocardiography and cardiac catheterization along with the clinical features aid the diagnosis. Right heart catheterization may show step-up of oxygenation in right ventricle with ventricular septal rupture. It is more common in women, patients with hypertension, and those receiving NSAIDs or steroids. Early recognition, stabilization, and surgical repair are needed for good outcome.
- **Other complications:** Include pericarditis, ventricular aneurysm, and mural thrombus.

Postangiography/Angioplasty Complications

In addition to the underlying ischemia, additional ischemic symptoms due to involvement of other coronary arteries during the intervention may be present. Ionotropes, vasopressors, vasodilator infusions may be given. Assist devices like pacemaker, IABP and "bailout" coronary perfusion catheter across the dissected artery may be in place. Patients may be intubated and on ventilatory support. Transport of such patients is critical as care needs to be taken that the bail-out catheter is not dislodged. Additional concerns in such patients include radiocontrast dye induced renal dysfunction and bleeding concerns due to antiplatelet

and thrombolytic administration. Massive platelet transfusions may be required.

INVESTIGATIONS

Hematological Investigations

- Complete blood count—CBC may show anemia either as a precipitating factor or a result of bleeding complications. Transfusion with packed red blood cells may be indicated. Leukocytosis may be observed within several hours after an AMI. It peaks in 2 to 4 days and returns to levels within the reference range within 1 week
- Coagulation profile—Patient is likely to be on heparin infusion/thrombolytics and/or platelet aggregation inhibitors and glycoprotein IIa/IIIb inhibitors leading to coagulation abnormalities.
- Blood Sugar levels may be elevated as a result of stress response or in diabetic patient
- S Electrolytes—Potassium and magnesium levels should be monitored and corrected
- Creatinine levels may be high in presence of acute on chronic renal dysfunction. Elevated creatinine levels may preclude use of angiotensin-converting enzyme (ACE) inhibitor and aminoglycoside group of antibiotics
- Liver function tests—Abnormal elevation of enzymes and bilirubin may be seen in right heart failure. Drugs and dosages may need to be titrated
- C-reactive protein (CRP)—Elevated CRP levels in patients without biochemical evidence of myocardial necrosis indicate increased risk of a subsequent ischemic event
- Erythrocyte sedimentation rate (ESR) rises above reference range values within 3 days and may remain elevated for weeks
- Serum lactate dehydrogenase (LDH) level rises above the reference range within 24 hours of MI, reaches a peak within 3 to 6 days, and returns to the baseline within 8-12 days
- Cardiac enzymes—Troponin I level is detectable in serum 3 to 6 hours after an AMI and its level remains elevated for 14 days. New sensitive cardiac troponin assays have greater diagnostic accuracy than the standard assays, especially for early diagnosis.[7] Creatine kinase—MB level begin to rise within 4 hours after injury, peak at 18 to 24 hours, and subside over 3 to 4 days. A level within the reference range does not exclude myocardial necrosis. Myoglobin level has high sensitivity but poor specificity and may be detected as early as 2 hours after MI
- Arterial blood gas analysis in complicated MI may help in detection and management of acidosis and oxygenation ventilation disturbances.

Other Tests

- Chest X-ray: Pulmonary edema secondary to heart failure, pericardial effusion, aortic dissection, presence of sternal wires in redo cases, pneumothorax or other lung pathologies may be seen
- Electrocardiography: ST-segment elevation greater than 1 mm in 2 anatomically contiguous leads or the presence of new Q-waves are strongly suggestive of MI. ST-segment depression, T-wave inversion, and other nonspecific ST-T abnormalities indicate intermediate probability of MI. Normal or nonspecific findings on may also be seen in some cases
  One can localize the affected region and coronary artery by analyzing the ECG (Table 5.1).
- Complications like sinus tachycardia, premature ventricular complexes, nonsustained ventricular tachycardia/ventricular tachycardia, bradyarrhythmia like Type I second-degree heart block, Type II second-degree heart block and new or pre-existing bundle branch blocks (BBB) may be seen. Complex PVCs and NSVT/VT need treatment with amiodarone, lidocaine or defibrillation, if unstable.

Table 5.1: Effect of ischemia on ECG

Region	*Leads*	*Artery*
Inferior wall	II, III, aVF	Dominant right coronary artery in 55% of individuals OR left circumflex artery in 45% patients, in which case changes of lateral leads will also be present.
Lateral wall	I, aVL, V_4 through V_6	Left circumflex coronary artery or lateral branch of the left anterior descending artery.
Anterior wall	I, V2-V4, and often in V1	Left anterior descending coronary artery
Anteroseptal walls	V_1 through V_3	Proximal left anterior descending artery
Anterolateral walls	I, L,V_4 through V_6 with possible involvement of V_2-V_3	Lateral branches of the left anterior descending artery
Right ventricular	RV_4, RV_5	Nondominant right coronary artery
Posterior wall	R/S ratio >1 in V_1 and V_2; T-wave changes (i.e. upright) in V_1	Right coronary artery with or without inferior infarction.

AIVR (accelerated idioventricular rhythm) is the most common reperfusion arrhythmia, but it usually is well tolerated and does not require treatment. Type I second-degree heart block (i.e. Wenckebach's phenomenon) is associated with inferior wall MI and is to be treated using temporary pacing or atropine only, if it is hemodynamically significant. Type II second-degree heart block is associated with anterior wall MI and may require a permanent pacemaker. Bundle branch blocks (BBB) that are new or pre-existing with new second-degree heart block may also mandate consideration for a permanent pacemaker

- Echocardiography can identify regional wall motion abnormalities (hypokinesia, dyskinesia and akinesia) and overall ventricular function—ejection fractions, presence of diastolic dysfunction which may decide the choice of anesthetic agents and cardiac medications. Chamber dimensions and pressures, wall and septal dimensions also aid in diagnosis. Complications of MI like valvular insufficiency, ventricular aneurysms, pericardial effusion can also be detected
- Coronary angiography—Exact anatomy of the coronary circulation with location and degree of stenosis, myocardial contractility and presence of regional wall anomalies should be noted. If the patient has had an abrupt closure during PCI or other complications like dissection, etc. Patient may have a "bailout" catheter in place. The catheterisation report should be reviewed to plan the type of intervention—PCI (percutaneous coronary interventions) or CABG (coronary artery bypass grafting) or hybrid procedures and also the number and sequence of grafts (if surgical management is planned). Also details regarding anesthetic management can be planned depending on the decision to use conventional on pump versus off-pump techniques
- Other imaging studies such as a contrast chest CT scan or transesophageal echocardiography can be used to differentiate MI from aortic dissection in patients in whom the diagnosis is in doubt. Stanford type A aortic dissections may dissect in a retrograde fashion causing coronary blockage and dissection, which may result in MI. Perfusion imaging has been used in risk stratification after MI and for measurement of infarct size to evaluate reperfusion therapies. However, its role in emergency revascularization is limited. Dual-source 64-slice CT scanning can do a full scan in 10 seconds and produce high-resolution images with fine details of the patient's coronary arteries.

TREATMENT OPTIONS

a. PCI—angioplasty/stents
b. CABG
   i. Conventional on CPB (cardiopulmonary bypass)
   ii. Off-pump
c. Hybrid procedures.[8-10]

Patients with single or two vessel involvement with proximal LAD (left anterior descending) stenosis can undergo PCI or CABG. PCI of the left main coronary artery with stents as an alternative to CABG may be considered in patients with anatomic conditions that are associated with a low-risk of PCI procedural complications and clinical conditions that predict an increased risk of adverse surgical outcomes. Primary PCI is more effective than thrombolysis and should be performed for confirmed STEMI, new or presumably new left bundle-branch block (LBBB), severe congestive heart failure, or pulmonary edema, if it can be performed within 12 hours onset of symptom. Door-to-balloon time should be 90 minutes or less.

Specific indications for surgical revascularization[11] are:

1. Cardiac catheterization complications — Acute closure due to flap dissection or dislodgement of thrombus, embolisation of platelet aggregates.[12]
2. Persistent ischemia with or without chest pain refractory to medical treatment or intra-aortic balloon pump.
3. Left main CAD.
4. Acute aortic dissection
5. Multiple high grade lesions with significant myocardium at risk.

Both conventional and off pump approaches have been successfully used.[5,13,14]

Choice depends on the surgical expertise, the anesthesiologist's ability to stabilize the patient and maintain acceptable hemodynamics during grafting, presence of competent perfusionists and patient factors.

Some patients may need hybrid procedures—surgical anastomosis of left internal mammary artery (LIMA) to LAD along with angioplasty/stents for other vessels in the same setting.

STABILIZATION

Patients with MI undergo medical management as per their hemodynamic stability. Hemodynamic instability or respiratory difficulty requires ABCD (airway-breathing-circulation-defibrillation) approach.

The main goals of management during this period are optimizing oxygenation, decreasing cardiac workload, controlling pain, prevention and treatment of complications and rapid intravenous thrombolysis and/or rapid referral for PCI or CABG depending on patient's condition. Evaluation, optimization and stabilization must proceed concurrently.

Empirical treatment of patients with suspected STEMI begins with morphine, oxygen nitrates and aspirin (MONA), ideally sequence being oxygen followed by nitrates, aspirin followed by morphine. However, therapy is initiated with whatever is available on site.

Oxygen is administered to all patients of suspected MI to maintain oxygen saturation >90 percent on pulse oximetry. Endotracheal intubation and IPPV with or without PEEP may be needed depending on hemodynamic stability.

Nitroglycerin sublingual (SL) tablet or spray 400 mcg or in the dose the patient is taking is given every 5 min and can be repeated up to 3 times. If symptoms persist, NTG is infused intravenously at a rate of 5 to 10 mcg/min. titrated to 10 percent reduction in MAP (mean arterial pressure) or limiting side-effects of hypotension (>30 percent reduction in MAP or systolic BP <90), or severe headache. Intravenous nitroglycerin is also indicated for control of hypertension and management of pulmonary congestion. RV infarction contraindicates use of nitrates. Nitrates should not be administered to patients who have taken any phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours as they may develop interactable hypotension.

Aspirin[15] 160 to 325 mg PO is given after confirming ability to swallow without aspiration and absence of contraindications to aspirin like GI bleed, allergy, etc. Clopidogrel 300 mg PO loading dose prior to PCI, then 75 mg PO qds is used in case of aspirin allergy.

Morphine is administered in the dose of 1 to 3 mg IV, repeated and titrated to pain relief.

At least two large-bore intravenous access should be ideally secured. Central venous access for hemodynamic monitoring and intravenous medications is obtained at the earliest. Blood is collected for hematological work-up. A chest radiograph is obtained soon after arrival to screen for alternative causes of chest pain and identify possible contraindications to thrombolysis (e.g. aortic dissection). Electrocardiogram for rapid diagnosis of ischemia, infarction and localization of artery is done.

Traditionally, unfractionated heparin 60 U/kg (max 4000 U) IV bolus; followed by a 12 U/kg/h (max 1000 U/h) maintenance infusion is used in ST elevation MI. In patients treated with fibrinolytic therapy, recommendations for heparin therapy depend on the fibrinolytic agent. Heparin has an established role as an adjunctive agent in patients receiving alteplase, reteplase, or tenecteplase but should not be used with nonselective fibrinolytic agents such as streptokinase and anistreplase. Heparin is also indicated in patients undergoing primary PCI. Low-molecular-weight heparins (LMWH) are commonly used because of convenient dosing and reliable therapeutic levels, but there have been no definitive trials of LMWH in patients with STEMI to provide a firm basis for recommendations. Low-molecular-weight heparin is be used as an alternative to unfractionated heparin, as ancillary therapy to fibrinolytics in patients aged older than 75 years or in patients with significant renal dysfunction (serum creatinine level >2.5 mg/dl in men or >2 mg/dl in women).

A platelet glycoprotein (GP) IIb/IIIa-receptor antagonist (eptifibatide 180 µg/kg IV loading dose; followed by 2 µg/kg/min IV for 72 h and for PTCA: 135 µg/kg IV bolus before procedure, followed by 0.5 µg/kg/min IV for 20 to 24 h, tirofiban 0.4 mcg/kg/min IV for 30 min; followed by 0.1 µg/kg/min, or abciximab 0.4 µg/kg/min IV for 30 min; followed by 0.1 µg/kg/min), is generally administered in addition to aspirin and unfractionated heparin. This combination is administered to patients with continuing ischemia or with other high-risk features and to patients in whom PCI is planned.[16] Studies suggest that the addition of intravenous platelet glycoprotein (GP) IIb/IIIa-receptor antagonists to aspirin and heparin improves both early and late outcomes, including mortality, Q-wave MI, need for revascularization procedures, and length of hospital stay.

Beta-blocker therapy for heart rate control and resultant decrease of myocardial oxygen demand should be given, if not contraindicated (asthma, etc.). Metoprolol 5 mg IV every 5 min 3 times; titrated to heart rate and SBP (systolic blood pressure) is the standard selective $beta_1$-adrenergic receptor blocker that is given. Esmolol in a loading dose of 500 mcg/kg/min IV over 1 min or 0.5 mg/kg slow IV infusion with a maintenance dose of 0.1 mg/kg/min IV initially; titrated in increments of 0.05 mg/kg/min every 10 to 15 min to a total dose of 0.2 mg/kg/min can be used. Beta-blockers reduce the rates of reinfarction and recurrent ischemia and may also reduce mortality.

An ACE inhibitor (Captopril 6.25 mg PO tid initially; may titrate to total 450 mg/d) is usually given orally within the first 24 hours of STEMI to patients with anterior infarction, pulmonary congestion, or left

ventricular ejection fraction (LVEF) less than 40 percent in the absence of hypotension. An angiotensin receptor blocker is administered to patients with STEMI who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 40 percent. Routine use of lidocaine as prophylaxis for ventricular arrhythmias in patients who have experienced an MI has been shown to increase mortality rates and its use is class indeterminate. Use of calcium channel blockers in the acute setting has come into question, with some randomized controlled trials and retrospective studies showing increased adverse effects. Diltiazem and verapamil should be avoided in patients with pulmonary edema or severe left ventricular (LV) dysfunction.

Reperfusion therapy with thrombolysis or primary PCI is considered in patients with persistent ST elevation. Thrombolysis is generally preferred to PCI in cases where the time from symptom onset is less than 3 hours and a delay of greater than 1 to 2 hours in the door-to-balloon time is expected. The goal for patients with STEMI should be to achieve a door-to-drug time of within 30 minutes and a door-to-balloon time of within 90 minutes. Thrombolytic therapy administered within the first 2 hours can occasionally abort MI and dramatically reduce the mortality rate. The optimal approach is to administer thrombolytic drugs as soon as possible after onset of symptoms up to 12 hours from symptom onset.

In patients without ST elevation, NSTEMI is diagnosed, if cardiac marker levels are elevated. If serum cardiac marker levels are not elevated, coronary angiography or imaging studies are needed to confirm the diagnosis of unstable angina. Both these group of patients generally receive anti-ischemic therapy and may be candidates for PCI urgently or sometime during the hospital stay.

Patients need to be simultaneously evaluated for anesthetic management with thorough history, examination and investigations as detailed above.

MONITORING

Availability of monitoring devices is crucial to the eventual outcome of these patients. Monitors include routine and special devices unique to cardiovascular set-ups. It is important to remember that time is of essence and risks versus benefits of all interventions—monitoring devices, insertion of central lines and arterial cannulation need to be considered. In arrest situations, one needs to resuscitate and go to institution of CPB as soon as possible. Therefore, femoral sheaths can be utilized, if available and additional lines can be taken while patient is weaned off bypass. If chest pain and ischemic ECG changes have resolved—cautious insertion of monitoring lines may be preferred followed by induction of anesthesia. If ischemia is present one should proceed to induction. If changes subside after anesthetic induction, insertion of monitoring lines can be done. However, if hemodynamic changes persist after induction, one should proceed to CPB emergently.

Pulse oximeter: One needs to target a $SpO_2 \geq 90$ percent

Cardioscope with defibrillator/pacing: 5 lead ECG with ST segment analysis and T-wave monitoring for ongoing or fresh ischemia is mandated. Simultaneous observation of inferior and anterior lead detects almost 90 percent of ischemic events. During automated ST segment analysis it is important to remember that the ST and J point should be rechecked before and after bypass and when there have been any significant persistent changes in the patient's heart rate. This is because the reference points chosen at the beginning of the procedure may not be accurate for conditions that may exist later. Rhythm needs to be closely observed as arrhythmias are quite common. Adhesive electrodes for defibrillation and pacing are required in thoracotomy approach.

Invasive blood pressures: Femoral sheaths can be utilized, if already in place, as in cases of post angioplasty complications. Consider radial or other arterial lines depending on hemodynamic stability and conduits planned for grafting.

Central venous/pulmonary arterial catheter monitoring (PAC): Depending on urgency and hemodynamic stability consider invasive monitoring. The use of PAC is controversial [17,18] especially in institutes where TEE is routine. However, majority of anesthesiologists are comfortable placing a PAC, if time permits. Extreme caution during insertion of PAC in patients with LBBB is mandated and transcutaneous pacemaker should be kept ready. Multiple lumen central venous access is preferred in patients with good ejection fraction and bundle branch block. Heparin administration for catheterization or as first line treatment for MI may complicate internal jugular venous access. In such cases pre-existing femoral venous sheaths or femoral venous access on contralateral limb may be preferred.

Capnography: End tidal CO_2 monitoring helps in early detection and treatment of hypercapnea. One can thus avoid detrimental-effects of hypercapnea on pulmonary arterial pressures.

Temperature monitoring: Nasopharyngeal and rectal/bladder probes can be used for CPB as well as off pump cases where temperature maintenance is crucial. Monitoring at the two sites with nasopharyngeal reflecting core temperature and rectal/bladder probes reflecting shell temperature is done taking care that the temperature lag does not exceed 10°C at any time. Insertion of nasopharyngeal probe should be done gently taking care not to cause any injury.

Urinary output: Urinary catheter aids in the detection and management of renal dysfunction. Renal dysfunction due to cardiogenic shock, complicated MI and post-catheterization radiocontrast induced nephropathy are common in emergency revascularizations

Neuromuscular monitoring: This can ensure adequate level of relaxation with prevention of inadvertent overdosing.

Transesophageal echocardiography (TEE): TEE can assess ventricular preload and contractility, detect myocardial ischemia induced regional wall motion abnormalities, evaluate the aortic cannulation site, detect concomitant valve pathology, pericardial effusion, ventricular aneurysms, ventricular septal defects and aid placement of catheters. It can help management of off-pump CABG.[19] However, pacing, bundle branch blocks and stunned myocardium may complicate the diagnosis of RWMA's using TEE.

Other cardiac output monitors: Noninvasive cardiac output using partial rebreathing, pulse contour analysis technology (PiCCO) and arterial pressure based cardiac output (Flotrac/Vigileo) can be used.

Arterial blood gas analysis and electrolytes monitoring: Useful for detection and treatment of acid-base, oxygenation-ventilation abnormalities and electrolyte like potassium, ionized calcium, sodium and magnesium crucial in prevention of further arrhythmias, ischemia and myocardial depression.

Hematocrit: Many ABG devices measure hematocrit and target hematocrits of 30 are recommended on cardiopulmonary bypass.

Coagulation: Activated clotting times are routinely used with target ACT's > 400 to 480 seconds with heparin dosage of 3 mg/kg. Institutes may individualize heparin dosages from 1 to 3 mg/kg. However, in cases of emergency revascularizations that mandate immediate access to cardiopulmonary bypass full heparinisation is indicated.

Thromboelastography: Functional information on platelets, clotting factors and fibrinolytic processes can be obtained and the use of blood and blood products can be rationalize.[20,21]

Blood glucose: Hyperglycemia has been associated with postoperative bacteremia[22] and worsened hospital outcome.[23] On the other hand tight control of blood glucose level may lead to dangerous hypoglycemia. Therefore, this strategy of tight control blood glucose has recommendations both for and against it.[24] It is reasonable to use insulin to maintain blood glucose levels less than 180 mg percent to avoid postoperative complications or dangerous hypoglycemia.[16] Also insulin infusions have been associated with increased neutrophil function[25] and reduced incidence of deep sternal wound infections.[26]

PERCUTANEOUS CORONARY INTERVENTION

Patients referred for PCI should ideally undergo intervention within 12 hours of STEMI with door-to-balloon time of 90 minutes. Depending on the patient status, the anesthesiologist may need to provide sedation and anlgesia with airway and hemodynamic monitoring or general anesthesia with same principles as any CABG patient or manage a patient with complete hemodynamic collapse on circulatory and ventilatory supports.

The choice of agents depends on the hemodynamic status of the patient and the pharmacodynamic profile of the anesthetic agent.

In otherwise stable patients, supplemental oxygen is administered, typically by nasal cannula or simple oxygen mask. Peripheral intravenous access is obtained for the administration of sedatives, analgesics and cardiac drugs. Sedation and analgesia are needed to reduce the discomfort associated with remaining supine and immobile for prolonged periods. Injection of contrast material also results in chest discomfort and burning sensation at times. Local anesthesia, typically lignocaine—bupivacaine combinations, can be used at the catheter insertion site to limit patient discomfort related to vascular access. Anesthetic agents used commonly include fentanyl 1 to 2 µg/kg and midazolam 0.02 to 0.05 mg/kg, sometimes supplemented with propofol 0.5 to 1 mg/kg. Pulse oximeter and noninvasive blood pressure monitoring is done. Arterial blood pressure can be directly transduced from the arterial introducer once the sheaths are introduced. The venous sheath also provides a ready route for rapid administration of fluid or medications or insertion of temporary pacing electrode, if needed. The ECG tracing must be continuously analyzed for fresh changes and arrhythmias which are common during wire and

catheter insertions and the patient monitored for the presence of angina or heart failure.

Sublingual and intravenous nitroglycerin should be readily available for administration, if myocardial ischemia develops, emergency resuscitation drugs including atropine, adrenaline, lignocaine, amiodarone, beta-blockers, ionotropes, vasopressors, vasodilatos, etc. equipment for airway control as well as cardiac arrhythmias like defibrillator and pacer must be immediately accessible.

Care must also be taken to carefully monitor the patient for reaction to the contrast media. The administration of steroid and antihistaminics, use of nonionic contrast media may decrease the incidence of allergic reactions. If severe reaction occurs, administration of 0.1 mg adrenaline repeated every two minutes until blood pressure and/or wheezing improves, may be helpful along with steroid and bronchodilator therapy. Another factor to be considered is the high incidence of renal dysfunction caused by pre-existing hemodynamic instability and further aggravated by the contrast media administered during PCI. Use of N-acetyl cysteine and sodium bicarbonate is helpful in preventing renal dysfunction.[27]

Heparin is administered in the doses of 10,000 IU intravenously, with a target ACT of greater than 300 seconds. Reversal doses of protamine may lead to hypotension due to peripheral vasodilation, anaphylactic and anaphylactoid reactions or the rare catastrophic pulmonary vasoconstrictive crisis. Platelet aggregation inhibitors and low-molecular-weight heparin are frequently administered during interventional cardiac catheterization and have resulted in improved outcomes despite the reduction in heparin dose.[28] Platelet aggregation inhibitors used have included, ticlopidine and clopidogrel and glycoprotein IIb/IIIa inhibitors abciximab, eptifibide and tirofiban. Abciximab may lead to elevation of the ACT independent of heparin.

If myocardial ischemia with angina occurs during angiography, the first action is to withdraw the catheter and temporarily suspend dye infusions until angina resolves. Intracoronary vasodilators such as nitroglycerin, intracoronary diltiazem or tirofiban infusions may be used to treat vasospasm and thrombosis. If marked hypertension is present and fails to respond to nitroglycerin, other vasodilators may be needed. If there is inappropriate tachycardia in the setting of angina with reasonable left ventricular systolic function, esmolol, metoprolol or propranolol may be cautiously used. If refractory failure develops intra-aortic balloon may be needed to augment stroke volume.

During balloon angioplasty or balloon inflation to optimize stent placement, transient coronary artery occlusion occurs, and the patient's hemodynamic status must be monitored closely. Further analgesia may be necessary because angina may develop during this time. PTCA has been supplemented by various techniques that remove the atheromatous plaque with atherectomy catheters that shear the plaque or with the excimer laser, as well as by placement of coronary stents to improve long-term patency.

During ischemia and frequently during reperfusion of the stenotic coronary artery, ventricular arrhythmias may develop and require treatment. As mentioned earlier AIVR is commonest reperfusion arrhythmia but usually requires no treatment. Hemodynamically significant premature ventricular contractions and nonsustained ventricular tachycardia should be initially treated with amiodarone. More severe arrhythmias may require cardioversion with the patient under general anesthesia.

Postprocedure care involves close monitoring of patient for hemodynamic stability, bleeding or hematoma at the catheter site, as well as for ischemia of the limb distal to the site. Patient should be discharged to the intensive cardiac care unit for further monitoring.

General anesthesia is reserved for hemodynamically unstable patients, failed sedation, uncooperative patients, patients with complicated MI or patients developing complications during the procedure. The principles are similar to management of patients for emergency coronary artery bypass grafting with attendant problems of full stomachs and hemodynamic instability.

Rupture of the coronary artery may result in hemopericardium and pericardial tamponade. Pericardial tamponade must be treated promptly with emergency pericardiocentesis and may involve placement of pigtail catheter, typically guided by transthoracic echocardiography. Once the tamponade is drained, patient usually complains of pain due to the constant friction of the pigtail catheter with each heartbeat. Additional fentanyl is usually needed at this stage. Emergency operative intervention may be required, if bleeding is not resolved. Ketamine may be a reasonable choice for induction of anesthesia.

Another rare complication of PTCA is coronary artery occlusion, which may result from coronary artery dissection, thrombus within the coronary artery, displacement of a plaque or vascular spasm caused by dysfunctional coronary artery endothelium. Vascular spasm may often be relieved by the injection of 200 µg of

nitroglycerin through the coronary artery. Intracoronary diltiazem has also been used. As mentioned earlier other measures like nitroglycerin, beta-blockers may be necessary.

Thrombosis of the coronary artery requires special therapeutic approaches. Prophylaxis in the form of heparin administration, therapy in the form of intracoronary injection of thrombolytic agents, such as urokinase to dissolve the thrombus and platelet aggregation inhibitors like tirofiban in infusion form may be administered. Emergency removal of thrombus or stenting across the thrombus may be needed.

Acute coronary occlusion not responding to percutaneuos intervention may require emergency coronary artery bypass grafting (CABG). The patient may have angina, hypotension, and arrhythmias and require an intra-aortic balloon pump. Endotracheal intubation may need to be performed emergently for securing airway, reduction of workload and ventilatory support. Full stomach precautions may be needed. In addition, the patient may require ionotropic support and nitroglycerin to improve collateral coronary flow and reduce preload. Adequate preload must be ensured, and monitoring of central pressures may be helpful.

Emergency "bail-out catheter" may be placed to allow some coronary blood flow and limit myocardial ischemia and salvage the myocardium. Great care must be taken in transport so that this catheter is not dislodged. Monitoring of blood pressure, ECG and SPO_2 must be continued in transport. Therapeutic NTG, Ionotrope and heparin infusions need to be continued. Maintenance of coronary perfusion pressure with phenylephrine or norepinephrine boluses and infusions needs to be considered. IABP triggers like ECG and BP need to be maintained. The patient must be transported to the operation theater as soon as possible and cardiopulmonary bypass initiated on an emergency basis for maximal myocardial salvage.

Fibrinolytic/antiplatelet agents given in the catheterization laboratory may lead to increased bleeding. Adequate units of blood and blood products especially platelets should be available. Cell salvage devices may be useful.

PREMEDICATION

Premedication depends on the hemodynamic status. Stable patients benefit by psychoprophylaxis, intravenous opioid-benzodiazepine sedation like fentanyl 1 to 2 µg/kg with midazolam 0.02 to 0.05 mg/kg titrated to hemodynamic stability and airway monitoring. Morphine 0.05 to 0.15 mg/kg with promethazine 0.5 mg/kg intramuscularly can be considered. Poorly compensated patients can be given 0.01 to 0.02 mg/kg midazolam intravenously titrated to hypotension. Continuation of infusions that the patient is receiving is important as is antibiotic prophylaxis. Aspiration prophylaxis in patient with full stomach precautions with histamine blockers/prokinetic agents, proton pump inhibitors and antiemetics are to be taken. Ranitidine 1 mg/kg, metoclopromide 0.2 mg/kg, Pantaprazole 0.8 mg/kg , ondansetron 0.08 to 0.16 mg/kg can be considered. Perioperative beta blockade with metoprolol may be given to reduce myocardial demand with target pulse rate 60 to 80 per minute provided there is no hypotension. Therapeutic drug infusions like NTG, ionotropes, vasopressors and heparin need to be continued in presence of ischemia and dysrhythmias. Maintenance of coronary perfusion pressure with phenylephrine or norepinephrine boluses and/or continuous infusions needs to be considered.

INDUCTION

No single anesthetic agent is found to be superior[29] as regards outcome though sevoflurane and desflurane have been recommended with emphasis on the need for further studies based on meta-analyses.[30,31] Both sevoflurane and propofol possess some, although different, cardioprotective properties. Propofol seems superior in patients with severe ischemia, cardiovascular instability, or in acute/urgent surgery.[32]

When choosing anesthetic agents and doses during induction and maintenance, one should consider any pharmacodynamic properties that might affect blood pressure, heart rate, or cardiac output. By being keenly aware of the impact of anesthetic agents on myocardial oxygen supply/demand dynamics and effectively monitoring and treating myocardial ischemia, one can accommodate any effects of an anesthetic agent. Most opioids, hypnotics, and volatile anesthetics have been used successfully in different combinations for induction and maintenance of anesthesia.

Emergency revascularizations in inadequately fasting patients require rapid sequence induction (RSI) with adequate preoxygenation. In such patients, anesthetic drugs with rapid onset of action with minimal hemodynamic-effects need to be chosen— High dose opioids or ketamine with succinyl choline or rocuronium may be a good choice. Inhalational agents may not be suitable. Though sevoflurane 8 percent vital capacity single breath technique has been used for RSI,[33,34] the associated hemodynamic instability[35-37] can make this technique unsuitable for emergent cases with hemodynamic instability.

Awake intubation may be considered in predicted difficult intubation with use of nebulized lignocaine or airway nerve blocks and transtracheal injections. However, it may be time consuming.

Anesthetic drugs and doses are selected according to LV function.[38] Patients with good LV function often have a strong sympathetic response to intense surgical stimulation such as sternotomy. If not adequately treated, tachycardia, hypertension, and ischemia result. Relatively large doses of anesthetics often combined with α-blockers, vasodilators, or both, are required. On the other hand, in patients with poor LV function, hypotension may develop with the administration of anesthetics because of a reduction in cardiac output or vasodilation (or both). Such patients may require vasopressor or inotropic pharmacologic support, or both.

Intravenous Drugs

Opioids: High dose opioids have been the mainstay of inducing anesthesia. This strategy has required rethinking recently due to an increasing trend of fast tracking. In patients in whom early extubation is not expected—as in most emergency revascularizations due to hemodynamic instability, difficult airways, obesity, severe pulmonary disease, poor ventricular function, etc. this technique is appropriate. 5 to 100 μgm/kg fentanyl or sufentanil 6 to 10 μgm/kg has been used. As little as 1 μgm/kg sufentanil has also been recommended. In case early extubation can be planned, combining low-dose opioids 2 to 5 μgm/kg fentanyl or sufentanil 0.1 to 1 μgm/kg or remifentanil 0.1 to 0.75 μgm/kg/min or bolus 0.5 to 1 μgm/kg with volatile agents (sevoflurane) or short acting intravenous agents (thiopentone sodium) may be helpful. Opioids have the main advantage of hemodynamic stability with disadvantages being chest rigidity and delayed extubation. A rapid acting neuromuscular blocker should always be ready prior to high-dose fentanyl induction.

Nonopioid Induction Agents

Thiopentone sodium: To be used cautiously with titration to effect on hypotension and tachycardia upto 2 to 4 mg/kg due to direct myocardial depression above 2 mg/kg.[39] Usually combined with opoid-benzodiazepine combinations to reduce required doses and maintain hemodynamic stability. Rapid onset is its only advantage in emergency situations.

Propofol: Induction dose of 2 mg/kg can drop blood pressure by 15 to 40 percent, with myocardial depression above 0.75 mg/kg. Due to its direct myocardial depression and hypotension it is to be used with extreme caution. Its use is reserved only for relatively stable patients.

Etomidate: Induction dose of 0.2 to 0.3 mg/kg maintains hemodynamics better (10-15 percent reduction in MAP, 3-4 percent increase in heart rate) than thiopentone and propofol. Reliable hypnosis when combined with opioid with additional advantage of attenuation of myoclonus. Hypotension may be seen with this combination at times. It can induce adrenal suppression rarely.

Ketamine: Drug of choice in patients presenting with cardiac tamponade, acute hypovolemia with hypotension or major hemorrhage. Centrally mediated rise in MAP. Tachycardia may be detrimental. Dose 2 mg/kg intravenously.

Other agents like midazolam 0.2 mg/kg, dexmedetomidine 0.5 to 1 μg/kg over 10 minutes have been used.

Inhalation Agents

Inhalational anesthetic agents decrease both myocardial oxygen demand and supply. Net effect depends on hemodynamic profile at the time of administration.

Sevoflurane with its pleasant aroma, hemodynamic stability and fast onset can be used. Other agents like desflurane with the advantage of rapid onset and offset with minimal hemodynamic compromise and isoflurane with cardiac stability need intravenous induction agent prior to their introduction due to their pungent odour. Desflurane and sevoflurane are more likely to reach 1.3 to 1.5 MAC levels to suppress the intubaton response earlier within 2 to 4 minutes. One meta-analysis demonstrated sevoflurane and desflurane reduced the postoperative rise in cardiac troponin I (cTnI) with Sevoflurane mediated reduction in cardiac troponin. It was not able to show that these positive effects on troponin were translated into improved clinical outcomes.[30,31]

However, isoflurane has been associated with better neurocognitive functions than desflurane or sevoflurane after on-pump CABG. Sevoflurane seems to be associated with the worst cognitive outcome as assessed by neuropsychological tests, and prolonged brain injury as detected by high S100 BP levels was seen with desflurane.[40]

Halothane with its potent myocardial depression and potential for dysrhythmias is not preferred.

Nitrous oxide: Not preferred especially when compressed air is available. It has probable myocardial depression action and increases pulmonary vascular resistance.

Muscle Relaxants

Suxamethonium chloride in a dose of 1.5 to 2 mg/kg with its potential for dysrhythmias may not be preferred except in difficult airway and rapid sequence induction. Nondepolarizing muscle relaxants are given early in the sequence. Rocuronium 1 mg/kg preferred for fast intubation and relative hemodynamic stability in full stomach situations. Vecuronium 0.1 to 0.2 mg/kg for rapid induction is ideal with low dose opioid with volatile agent. Bradycardia may limit it's usefulness in high dose opioid inductions. Pancuronium 0.1 mg/kg may be appropriate with high dose opioid induction.

OTHER CARDIOVASCULAR DRUGS TO BE KEPT READY

Other drugs that should be readily available include phenylephrine, ephedrine, epinephrine, atropine, lidocaine, nitroglycerin, dopamine, dobutamine, norepinephrine, vasopressin, nitroprusside, isoprenaline, milrinone, magnesium sulfate, potassium chloride, calcium gluconate, adenosine, beta blockers, calcium channel blockers, digoxin and amiodarone. Heparin 3 mg/kg must also be immediately available. Although, protamine should be accessible during the postbypass period, it should be stored in a separate, nearby location to prevent inadvertent inappropriate or premature administration.

Stress reponse to intubation may require to be blunted with esmolol 100 to 500 µg/kg or nitroglycerine 5 µg/min or lidocaine 1 to 1.5 mg/kg.

Hypotension requires phenylephrine 10 µg or ephedrine 5 mg boluses to maintain coronary perfusion pressures.

Tachyarrhythmias require β-blockers esmolol, metoprolol or propranolol, adenosine, amiodarone, Calcium channel blockers diltiazem and digoxin. electrolyte imbalances and acidosis may require correction in form of magnesium, calcium, potassium and sodium bicarbonate.

Bradyarrhythmias require atropine, low dose epinephrine and isoprenaline.

To be most prepared at least one ionotrope, one vasopressor , one vasodilator infusion should be set up and connected in a pump that is preprogrammed and ready to use. Syringes prepared for bolus administration should contain at least one vasopressor, one ionotrope, one vasodilator, a beta blocker, atropine, an antiarrhythmic and heparin.

MAINTENANCE

Opioid infusions fentanyl 0.03 to 0.1 µgm/kg/min or remifentanil 0.05 to 0.1 µgm/kg/min or sufentanil 0.01 µgm/kg/min or dexmedetomidine 0.5 to 1 µgm/kg/hr, with or without midazolam 0.25 to 0.5 µgm/kg/min, propofol infusion 20 to 100 µgm/kg/min, volatile inhalational agents sevoflurane, desflurane and isoflurane have been used.

Ischemic preconditioning with isoflurane, sevoflurane or desflurane may improve outcome.[41-45]

COAGULATION CONSIDERATIONS

Consensus has not been reached regarding whether aspirin and newer antiplatelet agents, if recently administered, increase the risk for bleeding after cardiac surgery. There is evidence for and against the ability of aspirin to increase mediastinal chest tube output and transfusion requirements.[46,47] ADP receptor antagonist clopidogrel has been associated with increased perioperative blood loss, an elevated risk for reoperation, increased mortality, and increased packed RBC and clotting factor use.[48,49] This trend has even been seen in the OPCAB population[50] However, one study did not show an association between the preoperative use of clopidogrel and increased bleeding and reoperation rates.[51] Hence, guidelines regarding cessation of clopidogrel and timing of urgent surgery continue to evolve.

Patients with a history of GP IIb/IIIa receptor blockade as a part of management of their acute coronary syndrome are at risk for increased bleeding and blood component use when given abciximab, especially within 12 hours of surgery.[52] Shorter-acting GP IIb/IIIa receptor antagonists have not been associated with increased bleeding or adverse outcomes. PURSUIT (platelet glycoprotein IIb-IIIa in unstable angina: receptor suppression using integrilin therapy) trial showed that eptifibatide reduced death and MI at 7 days to 6 months after surgery.[53] However, studies have reported safe use of tirofiban and abciximab in patients undergoing emergency CABG.[54,55]

Enoxaparin, an LMWH, has been associated with increased transfusion rates and an increased risk for surgical re-exploration.[56]

In summary, patients who undergo cardiac surgery with pre-existing, pharmacologically induced inhibition of the hemostatic system may have undesirable intraoperative and post-CPB bleeding.

FFP, platelets, cryoprecipitate, recombinant VIIa, cell savers and cell salvage devices need to be kept ready.

Heparinization: Most of the emergency revascularisation patients would be on heparin. Heparin infusion needs to be continued until sternotomy—bleeding may increase but continuation of heparin decreases risk of

worsening thrombosis. Initial doses need to be titrated from 2 to 3 mg/kg to higher doses to obtain ACT of >400 seconds. If celite based ACT is being used and aprotinin has been administered –ACT of 700 seconds is to be targeted. Off pump cases mandate ACT of 200 to 300 seconds with doses from 1 to 3 mg/kg. Considering heparin resistance in patient who is already receiving heparin, initial heparin dose needs to be increased to avoid delays in starting CPB due to low ACT's. In arrest situations as one needs to go directly to CPB, double or triple heparin dose are given to ensure adequate heparinization. FFP may need to be given, if antithrombin III deficiency is suspected.

Pharmacological Measures to Reduce Bleeding

Tranexamic acid can reduce bleeding in both on pump and off pump CABG.[57] This can be either given as 10 mg/kg preincision followed by 10 mg/kg on pump and 6 hours later can be given or higher dosage 20 mg/kg bolus followed by 1 to 2 mg/kg/hr or 5 gm bolus to a maximum of 15 gm has been recommended.[58]

Aprotinin though out of favor due to thrombosis of grafts, renal dysfunction and increased morbidity and mortality.[59-61] has been recently recommended for high-risk surgeries.[62]

Dose: 10000 KI units test dose followed by 1 or 2 million KI units as loading with same dose on pump and 25000 or 50000 units per hour infusion later not exceeding 7 million KI units.

PROTAMINE ADMINISTRATION

Reversal of heparin action by protamine has to be carefully titrated as per heparin dose and titrated against ACT. 1.3 times the heparin dose is usually administered. ACT should be within 10 percent of baseline values. One needs to watch for hypotension, pulmonary vasoconstriction and anaphylactoid reactions. Anaphylactoid reactions are more common with drug allergies, allergy to vertebrate fish, patients on NPH, vasectomized males and prior reaction to protamine. Treatment consists of slow injection, aggressive volume resuscitation, epinephrine, bronchodilators for anaphylactoid reactions and inodilators like milrinone, isoprenaline for pulmonary vasoconstriction. Re-institution of bypass with reheparinization may be required in severe cases of hemodynamic deterioration.

TEMPERATURE MAINTENANCE

Hypothermia is protective against further ischemia. Rewarming needs to be carefully managed so as to prevent arrhythmias as well as inadvertent cerebral damage due to sudden changes in temperature. In off pump cases, temperature maintenance is crucial as luxury of "rewarming on pump" is not available.

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

High dose steroids (dexamethasone 0.2 to 1 mg/kg IV/ methyl prednisolone 30 mg/kg on pump), heparin coated circuits, ultrafiltration, cell washers, protease inhibitors and off pump CABG have been used to prevent systemic inflammatory response syndrome.[63,64]

MYOCARDIAL PROTECTION

Apart from cardioplegia, use of hypothermia in on pump CABG cases, minimization of ongoing ischemia with use of oxygen with intubation, if necessary, nitrates, anticoagulants, antiplatelet agents, IABP and supports, avoidance of tachycardia, hypertension, hypotension, preoperative β-blockade in patients with good LV function in the preoperative period, rapid revascularisation with venous grafts[65] and nutritional repletion with glucose-insulin-potassium drip[66] can improve outcome in surgical revascularization.

CHOICE OF IONOTROPES/VASOPRESSORS/ VASODILATORS

Off Pump

- In off pump cases, prophylactic infusion of ionotropes—dopamine, dobutamine or low dose epinephrine may be appropriate.
- Prophylactic NTG may interfere with preload especially during verticalization of apex. However, it may be needed in patients showing fresh ischemic changes due to coronary vasospasm
- Norepinephrine or phenylephrine may be used during obtuse marginal and posterior descending-artery anastomoses.
- IABP may already be in place or may need to be placed emergently.
- Right ventricular failure management needs adequate preload, NTG infusion, ionodilators dobutamine, milrinone or isoprenaline, nitric oxide, sildenafil and hyperventilation with aggressive treatment of hypoxia and acidosis.
- Emergent conversion to CP bypass may be required in severe hemodynamic compromise unresponsive to treatment or IABP and is associated with poorer outcome.[67]

On Pump

In on pump cases, postbypass cardiovascular decompensation needs aggressive treatment. Causes of left ventricular failure may be ischemia due to graft problems, incomplete myocardial preservation, gas exchange problems, inadequate or excessive preload, reperfusion injury, ventricular septal defect, medications like β-blockade, calcium channel blockers, inhalational agents, acidemia, hypokalemia or hyperkalemia. Treatment includes detection and treatment of cause and use of ionotropes as stabilizing agents.

- Institutional policies vary with some preferring to use dopamine, dobutamine initially while others prefer epinephrine or milrinone. Ephedrine 5 to 20 mg or epinephrine 4 to 10 µgm can be given to increase contractility while commencing an ionotrope infusion
- Epinephrine or dopamine may be used, if HR is normal and SVR is low or normal
- Dobutamine or milrinone may be used, if SVR is increased
- Low dose epinephrine or milrinone may be used, if HR is elevated
- Dobutamine or dopamine may be used, if HR is low and pacing is not being used
- Norepinephrine or phenylephrine may be used, if SVR is low and CO normal or elevated
- Milrinone may require supplemental vasoconstrictor to combat reduction in SVR.
- RV failure needs to be treated as mentioned above.
- Emergent reconversion to CPB may be indicated for graft revisions.

POSTOPERATIVE MANAGEMENT

Early extubation may not be a choice in these hemodynamically unstable patients who have received thrombolytics and antiplatelet agents. However, single or double vessel revascularization with consequent hemodynamic stability may benefit from early extubation in absence of hypothermia or significant coagulopathy. Adequate pain relief is warranted with judicious use of opioids.

Generally these patients require prolonged ventilatory support. ICU sedation can be continued with fentanyl 1 to 2 µgm/kg/hour, Propofol 25 to 30 µgm/kg/min or ketorolac 15 to 30 mg IV 6 hourly with or without midazolam 0.01 to 0.03 mg/kg/hour.

Postoperative bleeding leading to re-exploration is a common but challenging complication of cardiac surgery, especially when it involves CPB. Coexisting pharmacologic inhibition in the form of antiplatelet agent therapy, residual heparin-effect due to inadequate protamine reversal, administration of glycoprotein IIb/IIIa inhibitors or low molecular weight heparin may contribute to the complication. Anesthetic considerations include mainly management of bleeding, stabilization of hemodynamics and judicious use of anesthetic agents. Platelets, FFP's, cryoprecipitate and recombinant factor VII A should be readily available apart from cell savers and other salvage devices.

Postoperative renal dysfunction, neurological dysfunction is anticipated.

ROLE OF REGIONAL ANESTHESIA

In view of patient being on anticoagulants and anti platelet agents and emergency surgery role of regional anesthesia is limited.

MANAGEMENT OF POST MI COMPLICATIONS

Ventricular Septal Rupture

Patients are anesthetized using a fentanyl-based regimen. Pancuronium is selected as the muscle relaxant so as to prevent bradycardia. Pulmonary bed vasodilators such as dobutamine are avoided to minimize the left-to-right shunt fraction. Preoperatively higher antibiotics need to be administered as prosthetic material may be left in the patient. Patients with multivessel coronary disease and critical coronary stenoses are revascularized before opening the heart in order to optimize myocardial protection. In most of these patients, saphenous vein rather than the left internal mammary artery is utilized. Low cardiac output and bleeding are problems while coming off bypass—IABP plus milrinone is a good strategy. Bleeding to be managed as discussed above.

Successful transcatheter closure of postinfarction ventricular septal rupture has been reported using several types of catheter-deployed devices. The largest experience is with the cardioseal device, a nitinol and double umbrella prosthesis.[68]

MI with Cardiogenic Shock or Severe LV Dysfunction

CABG in cardiogenic shock produces significant 1-year survival benefits and improvements in functional class of patient. Therefore, early surgical intervention is suggested where percutaneous coronary intervention is not possible or contraindicated for anatomical reasons.[69] Coronary artery bypass graft may be offered to patients with impaired ventricular function. Careful patient selection with assessment of potentially reversible

dysfunction and intensive perioperative management is needed.[70] Liberal use of ionotropes, IABP and left and right ventricular assist devices with rapid revascularization is mandated in these patients. Pericardial tamponade needs emergency pericardiocentesis.

CONCLUSION

Emergency myocardial revascularizations are expected to increase in the near future. These tremendously demanding surgeries require intensive involvement of anesthesiologist in the perioperative period. Time is critical and every intervention mandates a risk-benefit analysis. Anesthetic agents, cardiac drugs, monitors and devices are to be judiciously used for the maximum benefit of the patient. Surgical and interventional expertise, the Anesthesiologist's ability to stabilize the patient and maintain acceptable hemodynamics perioperatively, skilled intensivists, presence of competent perfusionists and patient factors all contribute to the better outcome.

REFERENCES

1. Gupta R, Joshi P, Mohan V, et al. Global burden of cardiovascular disease Epidemiology and causation of coronary heart disease and stroke in India. Heart 2008;94:16-26.
2. Gupta R. Escalating Coronary heart disease and risk factors in South Asians. Indian Heart Journal 2007;214-7.
3. NCMH Background Papers —Burden of Disease in India (New Delhi, India), 2005.
4. Xavier D, Pais P, Devereaux PJ, et al. Treatment and outcomes of acute coronary syndromes in India (CREATE): A prospective analysis of registry data. Lancet. 2008;371(9622):1435-42.
5. Varghese D, Yacoub MH, Trimlett R, et al. Outcome of non-elective coronary artery bypass grafting without cardiopulmonary bypass. Eur J Cardiothorac Surg 2001;19:245-8
6. ACCF/SCAI/STS/AATS/AHA/ASNC. Appropriateness Criteria for Coronary Revascularization A Report of the American College of Cardiology Foundation Appropriateness Criteria Task Force, Society for Cardiovascular Angiography and Interventions, Society of Thoracic Surgeons, American Association for Thoracic Surgery, American Heart Association, and the American Society of Nuclear Cardiology 2009.
7. Reichlin T, Hochholzer W, Bassetti S, et al. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. N Engl J Med 2009;361(9): 858-67.
8. Reicher B, Poston RS, Mehra MR, et al. Simultaneous "hybrid" percutaneous coronary intervention and minimally invasive surgical bypass grafting: feasibility, safety, and clinical outcomes. Am Heart J 2008;155(4):661-7.
9. Vassiliades TA, Jr, Douglas JS, Morris DC, et al. Integrated coronary revascularization with drug-eluting stents: Immediate and seven-month outcome. J Thorac Cardiovasc Surg 2006;131:956-62.
10. Zenati M, Cohen HA, Griffith BP. Alternative approach to multivessel coronary disease with integrated coronary revascularisation. J Thorac Cardiovasc Surg 1999; 117: 439-46.
11. Mehta AR, Romanoff ME, Licina MG. Anesthetic management in the precardiopulmonary bypass period. In: Hensley FA Jr, Martin DE, Gravlee GP (Eds), 4th edn. A practical approach to cardiac anesthesia. Wolters Kluver, Lippincott Williams & Wilkins, Philadelphia.
12. Bates ER, Ischemic complications after percutaneous transluminal angioplasty Am J Med March 2000;108(4): 309-16.
13. Capdeville M, Lee JH. Emergency off-pump coronary artery bypass grafting for acute left main coronary artery dissection. Tex Heart Inst J 2001;28(3):208-211.
14. Rastan AJ, Eckenstein JI, Hentschel B, et al. Emergency coronary artery bypass graft surgery for acute coronary syndrome beating heart versus conventional cardioplegic cardiac arrest strategies circulation. 2006;114:477-85.
15. Antithrombotic Trialists' (ATT) Collaboration. Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data from randomised trials. Lancet 2009;373 (9678):1849-60.
16. 2009 Focused Updates: ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction (Updating the 2004 Guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (Updating the 2005 Guideline and 2007 Focused Update): A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines.
17. Cohen MG, Kelly RV, Kong DF, et al. Pulmonary artery catheterization in acute coronary syndromes: insights from the GUSTO IIb and GUSTO III trials. Am J Med 2005;118(5):482-8SCA newsletter.
18. Harvey S, Harrison DA, Singer M, et al. Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): A randomised controlled trial. Lancet 2005;12,366(9484): 472-7.
19. Kapoor PM, Chowdhary U, Mandal B, et al. Transesophageal echocardiography in off-pump coronary artery bypass grafting. Ann Card Anaesth 2009;12:174.
20. Ronald A, Dunning J. Can the use of thromboelastography predict and decrease bleeding and blood and blood product requirements in adult patients undergoing cardiac surgery? Interact CardioVasc Thorac Surg 2005;4:456-63.
21. Vaneka T, Jares M, Snircova J, et al. Fibrinolysis in coronary artery surgery: detection by thromboelastography. Interact CardioVasc Thorac Surg 2007;6:700-04.
22. O'Brien JE Jr, Marshall JA, Tarrants ML, et al. Intraoperative Hyperglycemia and Postoperative Bacteremia in the Pediatric Cardiac Surgery Patient. Ann Thorac Surg 2010;89:578-84.
23. Ouattara A, Lecomte P, Manach YL, et al. Poor Intraoperative Blood Glucose Control Is Associated with

a Worsened Hospital Outcome after Cardiac Surgery in Diabetic Patients Anesthesiology 2005; 103:687-94.
24. Hogue CW Jr, Palin CA, Arrowsmith JE. Cardiopulmonary Bypass Management and Neurologic Outcomes: An Evidence-Based Appraisal of Current Practices Anesth Analg 2006;103:21-37.
25. Rassias AJ, Marrin CAS, Arruda J, et al. Insulin Infusion Improves Neutrophil Function in Diabetic Cardiac Surgery Patients Anesth Analg 1999;88:1011-6.
26. Furnary AP, Zerr KJ, Grunkemeier GL, et al. Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. Ann Thorac Surg 1999;67:352-60.
27. Brown JR, Block CA, Malenka DJ, et al. Sodium Bicarbonate Plus N-Acetylcysteine Prophylaxis A Meta-Analysis. J Am Coll Cardiol Intv 2009;2:1116-24.
28. Deutsch E. The emerging role of low-molecular weight heparin and antiplatelet therapies in the cardiac catheterization laboratory. Am Heart J 1999;138:S577-85.
29. Slogoff S, Keats AS. Randomized trial of primary anesthetic agents on outcome of coronary artery bypass operations. Anesthesiology 1989;70(2):179-88.
30. Yu CH, Beattie WS. The effects of volatile anesthetics on cardiac ischemic complications and mortality in CABG: a meta-analysis. Can J Anesth 2006;53:9,906-18.
31. Landoni G, Biondi-Zoccai G, Zangrillo A, et al. Desflurane and Sevoflurane in Cardiac Surgery: A Meta-Analysis of Randomized Clinical Trials Journal of Cardiothoracic and Vascular Anesthesia 2007; 21(4) 502-11.
32. Jakobsen CJ, Berg H, Hindsholm KB, et al. The influence of propofol versus sevoflurane anesthesia on outcome in 10,535 cardiac surgical procedures. J Cardiothorac Vasc Anesth 2007;21(5):664-71.
33. Lowry DW, Carroll MT, Mirakhur RK, et al. Comparison of sevoflurane and propofol with rocuronium for modified rapid-sequence induction of anaesthesia. Anaesthesia 1999;54(3):247-52.
34. Moore EW, Davies MW. Inhalational versus intravenous induction. A survey of emergency anesthetic practice in the United Kingdom. Eur J of Anaesth 2000;17(1):33-7.
35. Viitanen H, Baer G, Koivu H, et al. The Hemodynamic and Holter-Electrocardiogram Changes During Halothane and Sevoflurane Anesthesia for Adenoidectomy in Children Aged One to Three Years.Anesth Analg 1999;89:1423.
36. Sloan MH, Conard PF, Karsunky PK, et al. Sevoflurane versus isoflurane: induction and recovery characteristics with single-breath inhaled inductions of anesthesia. Anesth Analg 1996;82(3):528-32.
37. Vial G, Nathan N, Benrhaiem M, et al. Paroxystic systemic hypertension during inhalation induction with sevoflurane 8 percent. Can J Anesth 2000;47(2):191-4.
38. London M, Mittnacht A, Kaplan JA. Anesthesia for myocardial revascularization. In: Kaplan JA (Ed). Kaplan's Cardiac Anesthesia, Philadelphia. Saunders Elsevier 2006:585-643.
39. Howie MB, Gravlee GP, Induction of Anesthesia In: Martin DE, Gravlee GP, Hensley FA Jr (Eds), (4th edn). A practical approach to cardiac anesthesia. Wolters Kluver, Lippincott Williams & Wilkins, Philadelphia.
40. Kanbak M, Saricaoglu F, Akinci SB, et al. The Effects of Isoflurane, Sevoflurane, and Desflurane Anesthesia on Neurocognitive Outcome after Cardiac Surgery: A Pilot Study. The Heart Surgery Forum 2007;10(1):E36-41.
41. Landoni G, Biondi-Zoccai GG, Zangrillo A, et al. Desflurane and sevoflurane in cardiac surgery: a meta-analysis of randomized clinical trials. J Cardiothorac Vasc Anesth 2007; 21: 502-11.
42. Landoni G, Zangrillo A, Fochi O, et al. Cardiac protection with volatile anesthetics in stenting procedures. J Cardiothorac Vasc Anesth 2008; 22: 543-7.
43. Lange M, Redel A, Smul TM, et al. Desflurane-induced preconditioning has a threshold that is lowered by repetitive application and is mediated by beta(2)-adrenergic receptors. J Cardiothorac Vasc Anesth 2009; 23(5):607-13.
44. Lange M, Smul TM, Blomeyer CA, et al. Role of the beta1-adrenergic pathway in anesthetic and ischemic preconditioning against myocardial infarction in the rabbit heart *in vivo*. Anesthesiology 2006; 105:503-10.
45. Lee MC, Chen CH, Kuo MC, Kang PL, Lo A, Liu K. Isoflurane preconditioning-induced cardio-protection in patients undergoing coronary artery bypass grafting. Eur J Anaesthesiol 2006; 23: 841-7
46. Taggart DP, Siddiqui A, Wheatley DJ. Low-dose preoperative aspirin therapy, postoperative blood loss, and transfusion requirements. Ann Thorac Surg 1990; 50:424-8.
47. Tuman KJ, McCarthy RJ, O'Connor CJ, et al. Aspirin does not increase allogeneic blood transfusion in reoperative coronary artery surgery. Anesth Analg 1996;83:1178-84.
48. Kapetanakis EI, Medlam DA, Boyce SW, et al. Clopidogrel administration prior to coronary artery bypass grafting surgery. The cardiologist's panacea or the surgeon's headache?. Eur Heart J 2005;26:576-83.
49. Ascione R, Ghosh A, Rogers CA, et al. In-hospital patients exposed to clopidogrel before coronary artery bypass graft surgery: A word of caution. Ann Thorac Surg 2005; 79:1210-6.
50. Kapetanakis EI, Medlam DA, Petro KR, et al. Effect of clopidogrel premedication in off-pump cardiac surgery: Are we forfeiting the benefits of reduced hemorrhagic sequelae? Circulation 2006;113:1667-74.
51. Karabulut H, Toraman F, Evrenkaya S, et al. Clopidogrel does not increase bleeding and allogenic blood transfusion in coronary artery surgery. Eur J Cardiothorac Surg 2004; 25:419-23.
52. Gammie JS, Zenati M, Kormos RL, et al. Abciximab and Excessive Bleeding in Patients Undergoing Emergency Cardiac Operations. Ann Thorac Surg 1998;65:465-9.
53. Marso SP, Bhatt DL, Roe MT, et al. Enhanced efficacy of eptifibatide administration in patients with acute coronary

syndrome requiring in-hospital coronary artery bypass grafting. PURSUIT Investigators. Circulation 2000; 102:2952-58.
54. De Carlo M, Maselli D, Cortese B, et al. Emergency coronary artery bypass grafting in patients with acute myocardial infarction treated with glycoprotein IIb/IIIa receptor inhibitors. Int J Cardiol 2008;123(3):229-33.
55. Bizzarri F, Scolletta S, Tucci E, et al. Perioperative use of tirofiban hydrochloride (Aggrastat) does not increase surgical bleeding after emergency or urgent coronary artery bypass grafting. J Thorac Cardiovasc Surg 2001;122(6):1181-5.
56. McDonald SB, Renna M, Spitznagel EL, et al. Preoperative use of enoxaparin increases the risk of postoperative bleeding and re-exploration in cardiac surgery patients. J Cardiothorac Vasc Anesth 2005; 19:4-10.
57. Casati V, Valle PD, Benussi S, et al. Effects of tranexamic acid on postoperative bleeding and related hematochemical variables in coronary surgery: Comparison between on-pump and off-pump techniques. J Thorac Cardiovasc Surg 2004;128:83-91.
58. Shore-Lesserson L, Horrow JC, Gravlee GP. Coagulation management during and after Cardiopulmonary Bypass. In: FA Jr, Martin DE, Gravlee GP (Eds): 4th edn. A practical approach to cardiac anesthesia. Hensley Wolters Kluver, Lippincott Williams & Wilkins, Philadelphia.
59. Mangano DT, Tudor IC, Dietzel C. The risk associated with aprotinin in cardiac surgery. N Engl J Med 2006; 354(4):353-65.
60. Takagi H, Manabe H, Kawai N, et al. Aprotinin increases mortality as compared with tranexamic acid in cardiac surgery: A meta-analysis of randomized head-to-head trials. Interact Cardiovasc Thorac Surg 2009;9(1):98-101.
61. Sivasubramaniam S, Murali B, Smallpiece CJ, Smith B, Kalkat M. Intraoperative coronary vein graft thrombosis: Should we blame the use of aprotinin? Ann Card Anaesth 2009;12:84-6.
62. Karkouti K, Wijeysundera DN, Yau TM, et al. The risk-benefit profile of aprotinin versus tranexamic acid in cardiac surgery Anesth Analg 2010;110(1):21-9.
63. Day JRS , Taylor KM. The systemic inflammatory response syndrome and cardiopulmonary bypass. Int J Surg 2005; 3(2):129-40 .
64. Mazzone A, Gianetti J, Picano E, et al. Correlation between inflammatory response and markers of neuronal damage in coronary revascularization with and without cardiopulmonary bypass Perfusion 2003;18:3-8.
65. Hensley FA Jr, Martin DE, Gravlee GP, Entwistle JWC, Wechsler AS. Intraoperative myocardial protection, In: Henrley FA Jr, Martin DE, Gravlee GP (Eds), A practical approach to cardiac anesthesia. 4th edn. Wolters Kluver, Lippincott Williams & Wilkins, Philadelphia.
66. Quinn DW, Pagano D, Bonser RS, et al. Improved myocardial protection during coronary artery surgery with glucose-insulin-potassium: A randomized controlled trial. J Thorac Cardiovasc Surg 2006;131:34-42.
67. Landoni G, Pappalardo F. Crescenzia1 G, et al. The outcome of patients requiring emergency conversion from off-pump to on-pump coronary artery bypass grafting European Journal of Anaesthesiology 2007;24:317-322.
68. Landzberg MJ, Lock JE.Transcatheter management of ventricular septal rupture after myocardial infarction. Semin Thorac Cardiovasc Surg 1998;10(2):128-32.
69. Dar MI, Manan AU, Rasheed B, et al. Outcome of Patients after Coronary Artery Bypass Grafting in Cardiogenic Shock. Ann Thorac Cardiovasc Surg 2007;13(4):247-50.
70. Bouchart F, Tabley A, Litzler PY, et al. Myocardial revascularization in patients with severe ischemic left ventricular dysfunction. Long term follow-up in 141 patients. Eur J Cardiothorac Surg 2001;20:1157-62.

6 Anesthesia for Emergency Valvular Heart Procedure

Kanchan R Rupawate, Shalaka A Gandhi, Manju N Gandhi

KEY POINTS

- Normal cardiac valves permit unidirectional blood flow without causing obstruction or regurgitation, trauma to blood elements, thromboembolism, or excessive mechanical stress on the valve and heart. Any valvular pathology disrupts this relationship
- The most frequently encountered cardiac valve lesions produce pressure overload (mitral stenosis, aortic stenosis) or volume overload (mitral regurgitation, aortic regurgitation) on the left atrium or left ventricle
- Maintaining cardiovascular stability with optimal hemodynamic parameters and adequate systemic perfusion pressure during the anesthetic management of patients with valvular heart disease can be extremely challenging especially in emergent situations
- Drug selection during the perioperative period is based on likely effects of drug-induced changes in cardiac rhythm, heart rate, systemic blood pressure, systemic vascular resistance, and pulmonary vascular resistance
- The appropriate antibiotic prophylaxis, anticoagulation and antiarrhythmic drugs should be considered in the perioperative management of these patients
- Obstruction of prosthetic heart valves may be caused by thrombus formation, pannus ingrowths, or a combination of both. Prosthetic valve thrombosis can carry mortality as high as 60 to 80 percent hence anticoagulation is mandatory in all
- The physiological changes in the cardiovascular system during pregnancy can lead to early decompensation in case of a valvular heart disease and every attempt must be made to maintain the uteroplacental flow and fetal oxygen delivery.

INTRODUCTION

Emergency open heart surgery is only undertaken to relieve immediately life-threatening cardiac disease. Common indications for emergency valvular surgery are critical stenosis or regurgitation affecting one or more valves. Sudden deterioration can also occur by prosthetic valve dehiscence or thrombosis on its surface; perforation of valve cusp as a complication of subacute bacterial endocarditis, or by impaction of left atrial myxoma in the orifice of the mitral valve. Occasionally an aortic dissection extending to the ascending aorta and aortic valve needs urgent surgery. Myocardial trauma and ventricular septal rupture or intractable dysrhythmias associated with myocardial infarction account for a small percentage of emergency cases.

Important factors that govern blood flow across a valve include:

a. Valve area;
b. Square root of the hydrostatic pressure gradient across the valve; and
c. Duration of flow whether systole or diastole. The valve area of many regurgitant lesions changes in response to loading conditions (preload, afterload) whereas the valve area with stenotic lesions is generally fixed (Fig. 6.1).

Preoperative Evaluation in Valvular Heart Disease

These patients are almost always unprepared. Signs of circulatory failure are usually present and some patients are already intubated and on mechanical ventilatory support. Anesthesia assessment is often incomplete. Premedication should be minimal and whenever possible arterial and venous cannulae should be inserted under local anesthesia.

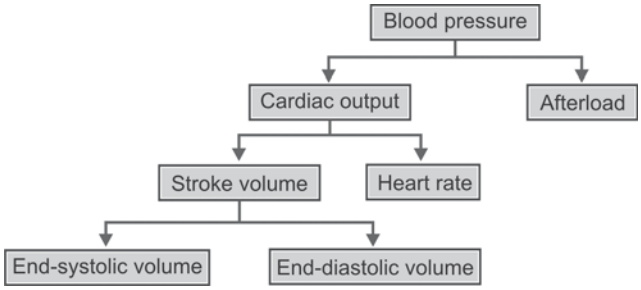


Fig. 6.1: Factors affecting the myocardial performance, cardiac output, systemic vascular resistance

Assessment should consist of;

- Severity of cardiac disease
- Degree of impaired myocardial contractility
- Presence of any major organ disease.

a. *History and physical evaluation to;*
   - Know the exercise tolerance of patient
   - Rule out the co-existence of congestive heart failure/ischemic heart disease
   - Know the presence of cardiac dysrhythmias, especially atrial fibrillation.

b. *Drug therapy*
   These patients can be on any of the following drugs and it is essential to know the interval since the last dose;
   - Anticoagulants
   - Antibiotics
   - Angiotensin-converting enzyme inhibitors
   - Calcium channel blockers
   - Beta-blockers
   - Diuretics
   - Nitroprusside
   - Inotropic agents.

c. *Blood Investigations*
   - Hemogram
   - Liver function tests
   - Blood urea nitrogen and serum creatinine
   - Arterial blood gases
   - Serum electrolytes—sodium, potassium and calcium.

d. *Recent electrocardiogram*
   - Chest X-ray
   - 2D-echocardiogram with color flow mapping
   - Cardiac catheterization laboratory data.

MITRAL STENOSIS (MS) (TABLE 6.1 AND FIG. 6.2)

Causes of mitral stenosis[1]

- Mitral stenosis in an adult is most commonly due to rheumatic heart disease.

Other causes of MS are:

- Congenital mitral stenosis
- Mitral annular calcification
- Rheumatoid arthritis
- Infective endocarditis.

Pathology

The latency period from acute rheumatic fever until the onset of cardiac symptoms due to MS was 19 years. Major cause of obstruction to diastolic flow in MS is usually due to fusion of commissures, it may be below the valve secondary to fusion of the chordae.

The cross-sectional area of the normal mitral valve is 4 to 6 cm^2. The normal mitral valve flow is 150 ml/s diastole in an adult patient.[1]

Table 6.1: Grades of severity of mitral stenosis

	Mild	*Moderate*	*Severe*
Mean gradient (mm Hg)	< 5	5-10	> 10
Pulmonary artery systolic pressure (mm Hg)	< 30	30-50	> 50
Valve area (cm^2)	> 1.5	1.0-1.5	< 1.0

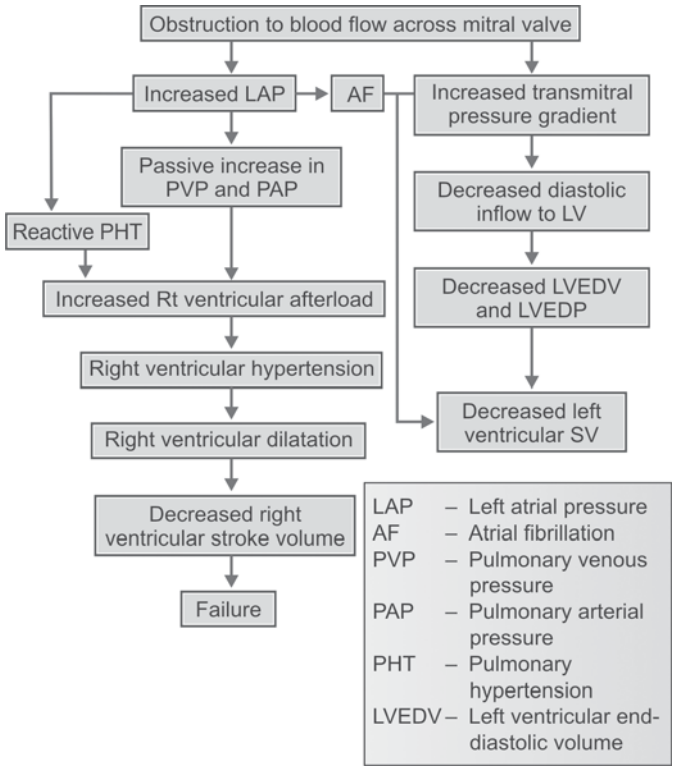


Fig. 6.2: Pathophysiology of mitral stenosis

Prime Symptoms[1]

A. Pulmonary venous hypertension: In the presence of tight MS (MVA < 1.0 cm^2), the mitral valve gradient at rest, with normal mitral flow, will be sufficient to cause symptomatic pulmonary venous hypertension. Patient may presents with:
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Pulmonary edema—may be the first overt sign of MS.

Causes of sudden onset of acute pulmonary edema:
- Atrial fibrillation (AF)
- Conditions that increase cardiac output, e.g. fever, anxiety, exercise, sepsis, etc.
- Pregnancy—effects are maximal at 25th to 27th weeks of gestation.

Causes of hemoptysis:
- Pulmonary apoplexy—from sudden increase in pulmonary venous pressure
- Profuse hemoptysis of this type is an indication for mitral valve surgery
- Bronchitis
- Pulmonary edema
- Pulmonary embolism causing pulmonary infarct.

B. Pulmonary vascular disease: This is in the form of reactive pulmonary hypertension which occurs when the pulmonary capillary pressure increases from 12 ± 2 mm Hg to 20 – 25 mm Hg at rest.

Other complications:
- AF
- Systemic embolism
- Infective endocarditis
- Ortner's syndrome.

Signs of MS

Inspection:
- Mitral facies
- Precordial bulge
- Left parasternal pulsations.

Palpation:
- Apex beat is tapping
- Right ventricular heave is present if pulmonary hypertension is present
- Apical diastolic thrill.

Percussion:
Cardiomegaly

Auscultation:
- Loud S1
- Mid-diastolic murmur
- Presystolic accentuation
- Opening snap.

Roentgenographic findings:
- Left atrial enlargement—double atrial shadow
- Redistribution of venous and arterial flow to upper lobes—Antler horn sign
- Calcification of mitral valve and left atrium
- Kerley B lines- in lower lung fields due to interstitial edema
- Enlarged pulmonary artery
- Enlarged right ventricle.

ECG
- P mitrale due to left atrial enlargement
- Right ventricular hypertrophy (RVH)—R/S in V1 > 1
- RVH with T inversion in V1 and V2—Right ventricular strain pattern
- Fibrillatory waves when atrial fibrillation is present
- Saw-tooth waves—in atrial flutter.

2D echocardiographic findings
- Increased echogenicity of mitral valve
- Reduced diastolic excursion of mitral leaflets
- Commissural fusion
- Diastolic doming of anterior mitral leaflet into left ventricle
- Reduction in mitral valve orifice area

Indications for transesophageal echocardiography (TEE)
- To evaluate the presence of left atrial thrombus
- To know the presence or severity of mitral regurgitation before balloon valvuloplasty
- To guide placement of catheters for atrial interseptal puncture.
  - For sizing and placement of balloon to be used.

Emergency Procedures in Mitral Valve Stenosis

1. **Performance of balloon mitral valvotomy (BMV)** on very sick patients with critical MS (Valve area <0.5 cm^2) necessitates the presence of an anesthesiologist. Such patients are in frank pulmonary edema and require ventilatory support to maintain the oxygenation. The anesthesiologist is required in such situations to execute endotracheal intubation and initiate ventilation before BMV, and manage the respiration during transport to the catheterization laboratory, during the BMV as well as post BMV period.[2] Percutaneous balloon mitral valvotomy is the procedure of choice when surgery is contraindicated or high-risk. It is performed by the interventional cardiologists in cardiac catheterization laboratory. It consists of advancement of a balloon

catheter through the interatrial septum and its inflation at the mitral orifice.[3] One should be prepared for open commissurotomy at any point of time in case of any eventually like acute mitral regurgitation or cordiac tamponade.

Contraindications[4]

- Thrombus in left atrium
- Moderate to severe MR
- Severe or bi-commissural calcification
- Absence of commissural fusion
- Concomitant severe aortic valve disease or combined tricuspid regurgitation and tricuspid stenosis
- Concomitant coronary artery disease requiring bypass surgery.

Complications[3]

- Severe acute mitral regurgitation
- Systemic embolism
- Residual atrial septal defect
- Risk of restenosis.

2. **Closed mitral commissurotomy** can be performed without cardiopulmonary bypass for pliable mitral valves without calcification and in absence of atrial thrombus or chordal fusion.[1,3] It has been replaced in the USA by BMV but in India, due to sheer number of patients and the economic conditions CMCV is still the preferred operation in India.[2]
3. **Open mitral commissurotomy** is done for pliable mitral valves without calcification but with left atrial clot.[1]
4. **Mitral valve replacement (MVR)** is done for associated mitral incompetence or calcified valve or above-mentioned contraindication for CMC.
   When chronic atrial fibrillation is present, scar tissue can be surgically created in the left atrium during open heart surgery to disrupt the re-entry circuits of atrial fibrillation. This procedure is called **Maze procedure**.[3]

GOALS OF PERIOPERATIVE MANAGEMENT

LV Preload

Forward flow across the stenotic mitral valve is dependent upon adequate preload. A reduction of preload from the venodilatory effects of anesthesia can markedly affect stroke volume, cardiac output and tissue perfusion.[5] On the other hand, overly aggressive fluids may cause congestive cardial failure (CCF) with florid pulmonary edema in these patients because of their elevated left atrial pressure (LAP). [3]

Heart Rate

Tachycardia shortens the duration of diastole thereby reducing the time for ventricular filling which then requires an increased transmitral pressure gradient—leading to pulmonary congestion. This is very relevant to perioperative events which can often lead to tachycardia. At the same time, excessive bradycardia can be dangerous because stroke volume is relatively fixed.

If atrioventricular pacing is initiated in these patients, a long PR interval of 0.15 to 0.20 msec is optimal to allow adequate time for blood to flow through the stenotic mitral valve[3] after atrial contraindication.

Maintenance of Sinus Rhythm

Patients in sinus rhythm who develop atrial fibrillation in the perioperative period should be cardioverted. Patients already in atrial fibrillation should have their heart rate controlled aggressively.

Contractility

Adequate forward flow depends on the right ventricular and left ventricular contractility. Many patients may require inotropic support before and especially after cardiopulmonary bypass (CPB).[3]

Systemic Vascular Resistance[3,5]

Patients with MS usually develop an increased systemic vascular resistance (SVR) in order to maintain the blood pressure in the presence of a limited cardiac output. Afterload reduction will reduce the forward flow and hence drug induced decreases in SVR should be avoided. It is recommended that after load should be kept in the normal range for these patients.

Pulmonary Vascular Resistance (PVR)

As these patients have elevated PVR and are prone to exaggerated pulmonary vasoconstriction. Therefore, hypoxia, hypercarbia, hypoventilation, acid-base balance should be avoided and probable use of pulmonary vasodilators may be considered.

Marked increase in central blood volume as associated with over-transfusion of fluids or head-down position should be avoided.

MONITORING

Monitoring includes standard noninvasive modalities and invasive monitoring of blood pressure, central

venous pressure (CVP) and intraoperative echocardiography (whenever available). Monitoring pulmonary artery pressure (PAP) and cardiac output (CO) with a pulmonary artery catheter (PAC) may be helpful. In order to maintain cardiac output through the tight mitral valve while avoiding pulmonary congestion, careful monitoring using a PAC is essential, the left ventricular end-diastolic pressure (LVEDP) will be significantly less than the pulmonary artery occlusion pressure (PAOP or "wedge"). Inotropic support may be needed for patients with secondary right ventricular (RV) dysfunction or failure. Epinephrine and milrinone are good therapeutic options to treat RV failure.[5]

ANESTHETIC MANAGEMENT

If the patient is in AF, it is necessary to continue digitalis preparations until the day of surgery and also maintain normal serum potassium levels.

Premedication[2,5]

Adequate premedication is necessary to prevent anxiety and tachycardia. Over sedation should be avoided since these patients may be sensitive to small doses of narcotics and hypnotics. A small dose of benzodiazepine may be considered for use in titrated doses. Pharmacologic agents or conditions that produce tachycardia, increased pulmonary vascular resistance, and decreased preload or decreased contractility should be avoided. Use of cardioversion is recommended if new atrial fibrillation should occur.

Induction and Maintenance

Anesthetic agents causing tachycardia or profound vasodilation should be avoided. For CMC, thiopental administered slowly or a narcotic (morphine 0.5 mg/kg, fentanyl 5-10 ug/kg) is a good choice.[2] Due to the common practice of extubating these patients at the end of surgery, the dose of the narcotic should be restricted to avoid excessive respiratory depression. However if higher doses of narcotics are administered for hemodynamic stability then elective postoperative ventilation for a few hours may be preferred.

Pancuronium is not a likely selection as it can lead to tachycardia. Vecuronium along with narcotics may produce severe bradycardia and hence the dose of the narcotics should be titrated slowly.[6] Atracurium may decrease blood pressure secondary to histamine release.[7] Vecuronium and rocuronium that decrease the HR, should be the prefered drugs in patients having faster baseline HR. In terms of intubating conditions, rocuronium and vecuronium provide best conditions but onset is quicker with rocuronium.[8] It thus appears to be a good choice for patients with MS.

Intraoperative use of vasodilator therapy (nitroglycerine or nitroprusside, 0.5-1 ug/kg/min IV[6]) is desirable in patients having severe pulmonary artery hypertension (PAH) and can be continued postoperatively.

In the period following CPB, inotropes such as dopamine, dobutamine, epinephrine may be considered to maintain cardiac output.

Postoperative Course

In the postoperative period, it is important to avoid increases in pulmonary vascular resistance (PVR). Therefore, it is desirable to electively ventilate the patients in the postoperative period and maintain normocarbia, acid-base balance and avoid hypoxia. The inotropic support and vasodilator therapy should be continued for a prolonged postoperative period up to 24-48 hours in case of severe PAH.

ACUTE MITRAL REGURGITATION

Mitral regurgitation from a number of causes is a relatively common valvular abnormality.[9] Acute mitral regurgitation can be classified based on pathophysiology;

- Primary nonischemic mitral regurgitation
- Primary ischemic mitral regurgitation
- Secondary mitral regurgitation.

Primary: Where one or more of valve components are responsible for valvular incompetence

Secondary: Implies ventricular dilatation or segmental wall motion abnormalities.

Acute Primary Nonischemic Mitral Regurgitation

Causes[1]

- Spontaneous chordal rupture and mitral valve perforation
- Infective endocarditis
- Myxomatous valvular degeneration
- Blunt chest trauma
- Hypovolemia in MVP
- After catheter valvuloplasty.

Pathophysiology of Acute Mitral Regurgitation (Fig. 6.3)

Symptoms

- Severe dyspnea
- Chest pain
- Orthopnea
- Paroxysmal nocturnal dyspnea (PND).

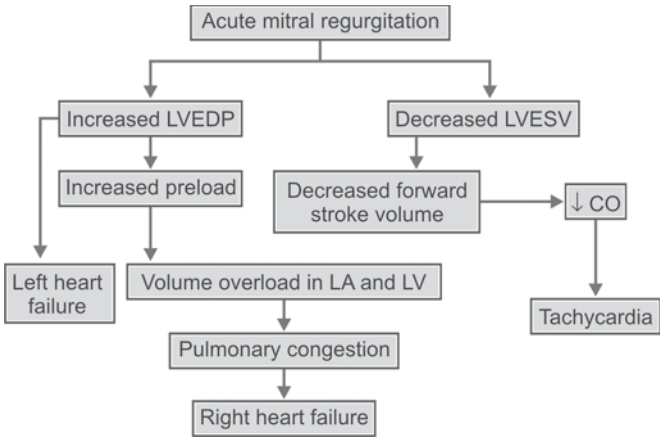


Fig. 6.3: Pathophysiology of acute mitral regurgitation

Signs
- Signs of right heart failure
- Cardiac palpation is unremarkable
- S1—soft intensity
- Murmur is an early systolic to holosystolic.

ECG—normal; atrial fibrillation is rare in acute mitral regurgitation

Chest X-ray—Normal cardiac silhouette, pulmonary edema may be seen

2D-Echo—Normal left atrium and left ventricular size

Surgical therapy
- Mitral valve repair
- Mitral valve replacement with chordal preservation

Acute Ischemic Mitral Regurgitation (AIMR)

Causes:[1]
- Acute ischemic MR occurs due to the effects of myocardial ischemia during an acute myocardial infarction (MI).
- It can also occur after the infarct as ventricular remodeling leads to papillary muscle displacement and annular dilation.
- It is more common during inferoposterior myocardial infarction.

Signs
- Signs of congestive cardiac failure
- Appearance of a systolic murmur

ECG—ST-T changes with q waves suggestive of an MI

Chest X-ray—Normal cardiac silhouette, pulmonary edema

2D-Echo—Wall motion abnormality, flail leaflet in case of papillary muscle rupture

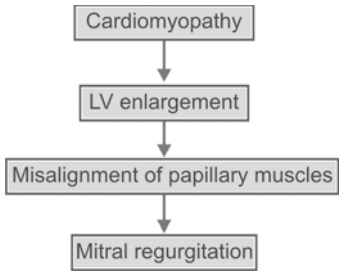


Fig. 6.4: Pathophysiology in MR due to cardiomyopathy

Surgical therapy[1]
- If AIMR is episodic with myocardial ischemia, revascularisation should be considered.
- If AIMR is persistent, mitral valve repair or replacement is the therapy.
- Mitral valve replacement with chordal preservation with myocardial revascularization.

Acute Secondary Mitral Regurgitation in Cardiomyopathy

Pathophysiology (Fig. 6.4)

Therapy[1]
Such cases should initially be treated with vasodilators and diuretics. If this fails then mitral valve annuloplasty may be considered.

Goals of Anesthetic Management

LV Preload

Augmentation and maintenance of preload frequently is helpful to ensure adequate forward stroke volume. But a decision on the best level of preload augmentation for a patient should be based on his hemodynamic and clinical response to a fluid load.[3]

Heart Rate[5,6]

Sudden decreases in heart rate should be avoided as forward left ventricular stroke volume is likely to be heart rate-dependent. Heart rate should be maintained in the high normal range (80-100 beats/min). Bradycardia not only increases the duration of systolic period but also decreases the diastolic filling interval. A normal sinus rhythm should be maintained.

Systemic Vascular Resistance[5,6]

Sudden increases in systemic vascular resistance can lead to increases in afterload which will reduce the forward left ventricular stroke volume and increase the

regurgitant volume. Hence, careful afterload reduction is normally desired. Monitor the magnitude of regurgitant flow with echocardiography whenever feasible.

Pulmonary Vascular Resistance[3]

As pulmonary vascular resistance is elevated, caution must be taken to avoid hypercapnia, hypoxia, nitrous oxide administration and light plane of anesthesia that might lead to increase in PVR.

Contractility[1,2,5]

Temporary use of small doses of ephedrine may be a better choice after which inotropes may be used to augment the pressure. Persistent hypotension may be treated with ionotropes like dobutamine, low-dose epinephrine and milrinone. In cardiogenic shock from ischemic MR, mechanical support with intra-aortic balloon counterpulsation which would reduce mean arterial blood pressure while reducing afterload and increasing the forward output may be considered. Arterial vasodilators such as sodium nitroprusside or venodilators like nitroglycerine which reduces vascular resistance thereby preferentially increasing forward flow may be useful.

Anesthetic Management

Premedication

Light premedication with use of titrated doses of midazolam may be given. A combination of opioids and benzodiazepines can be used to provide adequate hemodynamic stability. Combination of fentanyl and midazolam or sufentanil and midazolam either as an infusion or intermittant bolus can be used.

Induction and Maintenance of Anesthesia[1,2,5]

Careful titration of narcotics, hypnotics and volatile anesthetics are usually well tolerated. Tracheal intubation may lead to sudden rise in arterial blood pressure and increase in regurgitant volume followed by pulmonary edema. As slightly higher HR is desirable, pancuronium bromide is the preferred muscle relaxant in these patients. Atracurium or vecuronium may be used depending upon the basal heart rate. Inhalational anesthetic agents like halothane, isoflurane or sevoflurane may be used as they all produce vasodilation. Nitrous oxide can be used cautiously before the bypass if anesthetic induction has led to a decrease in the PAP is within normal range.

Central venous pressure—It is important to assess the RV function.

Pulmonary Artery Catheter

For any rise in PAP, nitric oxide may have an important role as a pulmonary arterial dilator.[3] Hyperventilation with minimal increases in intrathoracic pressures is another therapeutic modality for the same. PGE1 has also been used in dose of 0.1 ug/kg/min as an intravenous infusion[10] but is accompanied by a decrease in systemic pressure as well.

Transesophageal Echocardiography (TEE)

TEE is an invaluable tool for assessing the adequacy of valvular repair. After the valve replacement TEE can detect any perivalvular leak, prosthetic valve function or hemodynamically significant pressure gradient in immediate post-bypass period. It is also useful to confirm removal of air from LV before release of aortic cross clamp.

Weaning form CPB

The severity of regurgitation, left ventricular ejection fraction, pulmonary hypertension (PHT) and aortic cross clamp time are some factors that decide the need for inotropes. Patients with mitral valve repair less often require inotropes than replacement. Immediately following the weaning from CPB, the patient can be maintained in normal sinus rhythm by using amiodarone.[3]

Postoperative Course

Following MVR, LAP and PAP should decrease in case of acute MR. Use of inotropes and vasodilators are quite important during early postoperative period. Infusions of NTG and epinephrine may be started well before the bypass is terminated. Alternatively phosphodiesterase inhibitors such as enoximone, milrinone or a combination of dobutamine and NTG may be used.[5]

Finally, transverse midventricular disruption is the most dreaded complication after MVR and is usually fatal. This follows implantation of disproportionately large valve, direct surgical injury and sudden overdistention of LV after CPB.

AORTIC STENOSIS (AS)

Types

- Valvular
- Subvalvular
- Supravalvular

Causes

- Congenital—bicuspid aortic valve
- Rheumatic
- Atherosclerosis
- Degenerative calcification

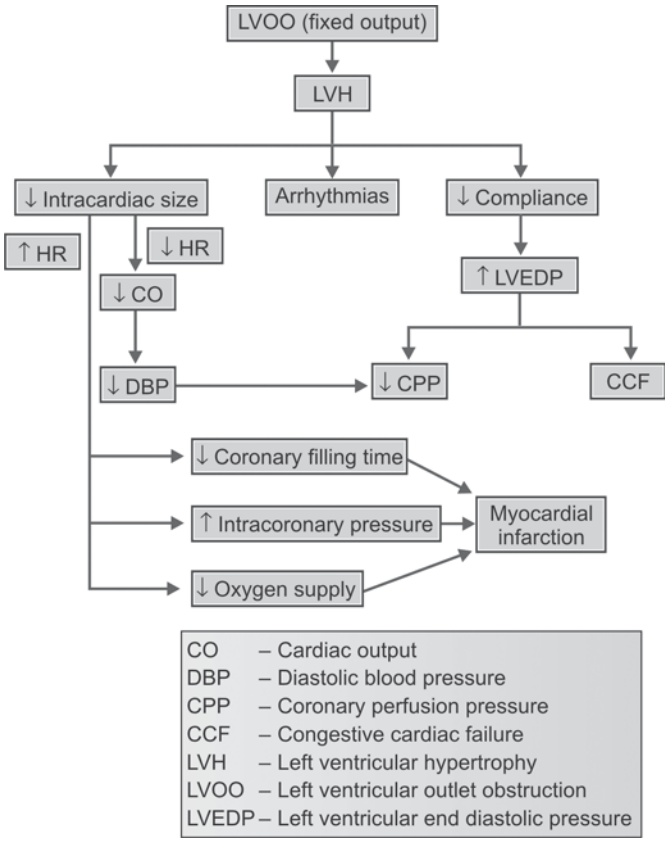


Fig. 6.5: Pathophysiology of aortic stenosis

Pathophysiology

The normal aortic valve area is 2.6 to 3.6 cm^2 and hemodynamically significant obstruction occurs at valve area of < 1.0 cm^2. Areas of 1.5 to 2.0 cm^2 represent mild AS; areas of 1.1 to 1.5 cm^2, moderate AS; areas 1.0 cm^2 or less, "surgical" stenosis. Critical aortic stenosis is defined as aortic valve area of 0.5 to 0.7 cm^2 with mean transvalvular gradient of > 50 mm Hg[11] (Fig. 6.5).

Symptoms

Classic triad of syncope, angina and dyspnea is usually present.

Signs

- Left ventriclular enlargement
- Systolic thrill in aortic area
- Ejection systolic click
- Ejection systolic crescendo decrescendo, conducted to carotids
- Pulsus parvus—low volume pulse.

Chest X-ray

- Prominent left ventricle
- Poststenotic dilatation
- Aortic valvular calcification.

ECG

- LVH
- Conduction abnormalities.

2D-Echo

- Restricted motion of aortic leaflet; leaflet thickening and calcification
- Aortic root dilation
- Concentric LVH.

Coronary angiography—To know the presence of coronary artery disease.

Therapy[1,3]

- Balloon aortic valvuloplasty
- Aortic valvulotomy
- Aortic valve replacement—If a patient undergoing aortic valve replacement (AVR) has significant coronary artery disease, coronary artery bypass grafting (CABG) should be simultaneously performed
- In young adults, Ross procedure is an alternative to AVR, where in patients AV is replaced by the pulmonary valve which is in turn replaced with a pulmonary homograft
- Percutaneous aortic valve insertion—Recent and undergoing clinical investigation.

Goals of Anesthetic Management

Left Ventricular (LV) Preload[3]

Due to decreased LV compliance as well as the increased left ventricular end-diastolic pressure (LVEDP) and left ventricular end-diastolic volume (LVEDV), preload augmentation is necessary to maintain a normal stroke volume.

Heart Rate[2,3]

As cardiac output (CO) is rate dependent, bradycardia can lower the CO. Tachycardia can lead to myocardial ischemia by disturbing the myocardial oxygen supply-demand ratio and reducing the coronary filling time. Heart rate between 50 and 70 beats/min may be essential to allow time for systolic ejection across a stenotic aortic valve. Arrhythmias such as AF or

junctional rhythm can lead to severe hypotension and hence it is important to maintain a sinus rhythm.

Contractility[5]

β-blockers are not well tolerated and can lead to an increase in left ventricular end daistolic volume and a decrease in cardiac output.

Systemic Vascular Resistance[3,5]

Systemic hypotension may develop with the anesthetic agents. The myocardium is at great risk for subendocardial ischemia because coronary perfusion depends upon maintenance of adequate diastolic perfusion pressure. Hence, early use of alpha-adrenergic agonists is indicated to prevent fall in blood pressure.

Severe AS reduces the usefulness of cardiopulmonary resuscitation to maintain adequate cardiac output.

Pulmonary Vascular Resistance[3]

PVR usually remains normal except in the end stage of aortic stenosis.

Percutaneous BAV[2]

BAV is usually a carried out in critically ill patients as a bridge to surgery. Anesthesiologist is usually required for monitored anesthesia care and for resuscitation, if necessary.

Anesthetic Management

Monitoring

This includes standard noninvasive modalities and invasive monitoring of blood pressure and CVP. Insertion of a PA catheter may be considered for monitoring PAP and cardiac output during surgery and particularly in postoperative period. A TEE may be desirable.

Premedication

Heavy premedication that would reduce the preload and afterload should be avoided. The dosage for premedication should be titrated for each patient. Morphine or benzodiazepine is required to reduce the preoperative anxiety and tachycardia.[2]

Induction and Maintenance

Narcotic based induction is desirable to reduce hemodynamic disturbances. For intubation, a muscle relaxant devoid of cardiovascular effects is desirable. Pancuronium can be advantageous if used with narcotics as it counters the bradycardia produced by narcotics.[2] Atracurium and vecuronium are reasonable alternatives but can potentiate bradycardia produced by the narcotics. Ocassionally, increase in systemic blood pressure during intubation or surgical stimulus can occur and cause severe increase in LV systolic pressure and may lead to myocardial ischemia. The adverse hemodynamic response to intubation should be treated by incremental doses of thiopentone or propofol. Low concentrations of inhalational agents may also be used. A potent alpha-adrenergic agent such as phenylephrine or norepinephrine should be available for early and aggressive treatment of any fall in blood pressure.[3]

For the patients who have a critical AS with LV dysfunction, a percutaneous femorofemoral bypass under local anesthesia can be instituted before administering general anesthesia.[2]

Supraventricular dysrhythmias should be aggressively treated with a synchronized DC shock. In the absence of preoperative ventricular dysfunction and associated coronary disease, inotropic support often is not required after CBP as AVR decreases the ventricular load.[3]

Postoperative Care

Once the AS is repaired, the pulmonary capillary wedge pressures (PCWP) and the LVEDP immediately decrease and stoke volume increases. LV hypertrophy regresses over a period of several months.

ACUTE AORTIC INCOMPETENCE (AAI)

Causes

- Acute aortic insufficiency may present suddenly in a chronic AI with CCF
- Infectious endocarditis
- Dissection of ascending aorta
- Trauma
- Spontaneous rupture of myxomatous valve
- After catheter valvuloplasty
- Prosthetic valve aortic insufficiency
- Connective tissue disorders like Marfans syndrome.

Acute severe AI is defined as hemodynamically important AI of sudden onset, occurring across a previously competent aortic valve into a left ventricle not previously subjected to volume overload. The appropriate treatment in a symptomatic patient with an aortic valve area <1.0 cm^2 is to proceed with an aortic valve replacement.[12]

Pathophysiology

Acute aortic regurgitation does not permit time for the LV to turn into a high-volume, high-compliance pump.[1] Following hemodynamic changes may be observed:

- Left ventricular diastolic volume—slightly increased
- LV afterload—modestly increased
- LVEDP—markedly increased
- LVEF—normal
- Forward stroke volume—decreased
- Peripheral vascular resistance—increased
- Coronary blood flow—modest decrease
- Left Atrial pressure—precipitous increase
- Pulmonary congestion—present
- Heart rate—tachycardia
- Sympathetic nervous system activity increased.

Symptoms

- Dyspnea on exertion
- Orthopnea
- Minimally productive cough
- PND
- Agitation
- Deterioration in mental function
- Abrupt chest pain.

Signs

- Signs of impaired regional blood flow
  - Oliguria
  - Pallor
  - Cold and clammy peripheries
- Signs of congestive cardiac failure
  - Pulsus Alternans.

Auscultatory Findings

- Soft S1
- Soft A2
- S3 can be heard
- Aortic systolic murmur—grade III or less
- Aortic regurgitant murmur—short, medium pitched
- Austin-flint murmur—heard mid-diastolic.

ECG

- Sinus tachycardia.

Chest X-ray

- Normal or modestly enlarged LV
- Usually normal aortic root and arch
- Redistribution of pulmonary venous flow to upper lobes.

2D-Echo

- Premature closure of mitral valve [1]
- Late opening of mitral valve
- Diastolic mitral regurgitation
- Normal systolic wall motion of left ventricle.

Goals of Anesthetic Management

Management centres on improving forward output. The degree of regurgitation depends on (1) the size of the incompetent orifice of aorta, (2) the pressure difference between the aorta and the ventricle, and (3) the duration of systole. Regurgitation increases with an increase in the area of valvular regurgitation with diastolic hypertension, and with bradycardia.

Heart Rate

Avoid bradycardia (low hr means long diastoles, more regurgitation and lower diastolic pressure). The atrial kick is relatively unimportant. Increased HR may decrease regurgitant flow by decreasing diastolic time. Even if increased HR fails to lower the regurgitant fraction, total cardiac output and therefore forward output may be improved. Tachycardia may also tend to reduce LVEDV.[5] A HR of 90 beats/min seems optimal, improving CO while not inducing ischemia.[3]

LV Preload

It should be augmented but without causing pulmonary edema. Pharmacological interventions that produce venous dilation may significantly impair CO in these patients by reducing preload.

Systemic Vascular Resistance

SVR should be kept lower to enhance forward output. A given aortic pressure can be achieved with a low or a high CO depending on SVR. A low SVR allows CO to rise without increasing aortic pressure. Decreased SVR tends to limit regurgitation by lowering diastolic pressure but the degree of afterload reduction may be limited by the decline in diastolic pressure.[5] Increases in afterload result in increased stroke volume and can significantly increase the LVEDP.[3]

Contractility

LV contractility must be maintained. Isoproterenol may improve contractility and cause vasodilatation and increase HR as well. Phophodiesterase inhibitors can also increase stroke volume through a combination of peripheral dilation and increased contractility. In the

absence of LV failure, CVP is likely to be an adequate measure of LV filling pressures. However, close control of SVR can only be achieved with a PA catheter and thermodilution cardiac output measurement.

A rise in peak airway pressure (PAW) pressure suggests LV dysfunction because one expects good LV compliance and therefore minimally elevated PAW.

Anesthetic Management

Monitoring

Pulmonary artery catheter: It is useful in evaluating the cardiac output of patients prior to repair of aortic valve and especially in post-bypass period for monitoring preload and myocardial function.[5] The measurement of CO and other hemodynamic parameters are also possible with the PAC so that the vasodilator therapy can be tailored to the patient's needs.[2]

TEE: It is beneficial in monitoring LV function, severity of regurgitation prior to valve repair and predicting the prosthetic valve size. Postoperatively it is used to assess the integrity of valve function, perivalvular regurgitation and pressure gradient across prosthetic valve.[3]

Intra-aortic balloon pump: Use of intra-aortic balloon pump is contraindicated in AI as augmentation of diastolic pressure will increase the amount of regurgitant flow.

Premedication

A light premedication is essential in patients with acute AI as myocardial depression is avoided and tachycardia if any is useful in these patients.

Induction and Maintenance[2,3]

Arterial vasodilatation caused by most of the anesthetic drugs is beneficial in AI and transient hypotension is well tolerated. The hemodynamic goals are directed at preserving the preload by adequately hydrating the patient, maintaining the arterial dilation, contractility and keeping the HR near 90/min. In particular bradycardia should be avoided as it can lead to LV distension that can further increase the LVEDP and produce myocardial ischemia and arrhythmias. Tracheal intubation in an inadequately anesthetized patient may cause a sudden rise in arterial pressure thereby increasing the regurgitant fraction leading to acute LV failure.

Narcotic based induction is preferred to ensure hemodynamic stability. Careful titration of narcotic and fluid balance is important. Pancuronium is the preferred muscle relaxant as it causes the beneficial rise in HR. Either nitroprusside or nitroglycerin infusion can be used for vasodilation in these patients.

Weaning from CPB

AVR leads to a mild transvalvular pressure gradient because a majority of prosthetic valves are intrinsically stenotic. Mild AS with a significantly dilated LV may result in increased afterload, low CO and may contribute to LV dysfunction. Inotropic support with preload augmentation is necessary during weaning from CPB as well as in postoperative period.

Postoperative Care

The LVEDP and LVEDV decrease immediately following AVR. In the early postoperative period, a decline in LV function may necessitate inotropic or Intra-aortic balloon pump support.

Management in Special Circumstances

Mixed Valve Lesions[3]

1. *Aortic stenosis and mitral stenosis (AS and MS):* This combination usually follows the course of a pure mitral stenotic lesion but is extremely serious because of the fixed output of blood flow at two points. Symptoms include dyspnea, hemoptysis and AF. It may underestimate the severity of AS (Table 6.2).
2. *Aortic stenosis and mitral regurgitation (AS and MR):* This is relatively rare combination and should be suspected in patients with aortic stenosis who also have left atrial enlargement. As aortic stenosis is a fixed cardiac output state, it should be given a priority when managing the hemodynamic variables (Table 6.3).
3. *Aortic stenosis and aortic regurgitation (AS with AR):* This combination is not well tolerated as it causes leads to severe pressure and volume load on left ventricle. Angina is an early symptom because of the increased myocardial O_2 consumption (Table 6.4). Generally, the hemodynamic profile should be consistent with the management of aortic stenosis as it is a more serious lesion.

Table 6.2: Hemodynamic management of AS with MS

	LV preload	*Heart rate*	*SVR*	*PVR*
Mitral stenosis	↑	↓	↑	↓
Aortic stenosis	↑	↓	↑	**Maintain**
Combination	↑	↓	↑	↓

Table 6.3: Hemodynamic management of AS with MR

	LV preload	*Heart rate*	*SVR*	*PVR*
Aortic stenosis	↑	↓	↑	Maintain
Mitral regurgitation	↑↓	↑	↓	↓
Combination	↑	Maintain	Maintain	↓

Table 6.4: Hemodynamic management of AS with AR

	LV preload	*Heart rate*	*SVR*	*PVR*
Aortic stenosis	↑	↓	↑	Maintain
Aortic regurgitation	↑↓	↑	↓	Maintain
Combination	↑	Maintain	Maintain	Maintain

Table 6.5: Hemodynamic management of MR with AR

	LV preload	*Heart rate*	*SVR*	*PVR*
Mitral regurgitation	↑↓	↑	↓	↓
Aortic regurgitation	↑	↑	↓	Maintain
Combination	↑	↑	↓	Maintain

Table 6.6: Hemodynamic management of MS with MR

	LV preload	*Heart rate*	*SVR*	*PVR*
Mitral stenosis	↑	↑	↓ Maintain	↓
Mitral regurgitation	↑↓	↑	↓	↓
Combination	↑	Maintain	↓ Maintain	↓

4. *Aortic regurgitation and mitral regurgitation (AR and MR):* This combination occurs more frequently and the patient can rapidly deteriorate due to the development of acidosis leading to peripheral vasoconstriction and increased impedance to LV outflow (Table 6.5).
5. *Mitral stenosis and mitral regurgitation (MS and MR):* Rheumatic heart disease commonly presents with a combination of MS and MR. Optimal hemodynamic stability may be obtained by normalization of after load, heart rate and contractility while avoiding conditions leading to pulmonary vasoconstriction (Table 6.6).

Considerations in Pregnancy

The challenges for managing a pregnant patient with valvular disease are three-fold;[13]

- The physiologic changes of pregnancy place a significant stress on the cardiovascular system and can lead to decompensation in previously asymptomatic patients.
- Labor, delivery, and puerperium add additional stresses to the maternal cardiovascular system.
- The presence of fetus should be borne in mind and every attempt must be made to maintain uteroplacental blood flow and fetal oxygen delivery.

Mitral Stenosis in Pregnancy

Rheumatic mitral stenosis is the most common clinically significant valvular abnormality in pregnant women.

Close follow-up is necessary in every pregnant woman with significant mitral stenosis, even if she was totally asymptomatic before pregnancy, with echocardiographic assessment of mean transmitral gradient and pulmonary artery pressure.

β-blockers should be started in patients who have symptoms or estimated systolic pulmonary arterial pressure more than 50 mm Hg, as well as low salt diet and reduction of physical activity.

Diuretics need to be added if signs of pulmonary congestion develop or persist due to autotransfusion by contracting uterus.

Choice of Therapy

- In patients who present with severe symptoms during pregnancy despite medical therapy, percutaneous balloon mitral valvuloplasty has to be considered. Performed during the second trimester, it has been associated with normal subsequent deliveries and excellent fetal outcomes.[14]
- One study compared percutaneous balloon mitral valvuloplasty with open mitral valve commissurotomy and showed that valvuloplasty is a better option, reducing fetal and neonatal mortality significantly.[15]

  When hemodynamic compromise persists despite appropriate medical treatment, percutaneous balloon valvuloplasty may be needed.[16]

Anesthetic Goals in Pregnancy with Mitral Stenosis

For Labor and Delivery

- Epidural anesthesia is recommended for labour and delivery as it eliminates maternal pain and sympathetic stimulation.
- Epinephrine containing local anesthetics should be avoided because of their potential for producing tachycardia and vasodilatation.
- Careful hydration is essential to prevent decreases in blood pressure that would cause tachycardia. Phenylephrine may be preferable to ephedrine to

prevent maternal hypotension because of its lack of positive chronotropic effects, but careful fetal surveillance is essential.
- Adequate coagulation function must be confirmed in case of patients receiving anticoagulants.

For Cesarean Section

- For a cesarean section, either epidural or general anesthesia is acceptable.
- Single shot spinal anesthesia is less desirable, due to exaggerated changes in maternal hemodynamics it can produce.
- If general anesthesia is selected, drugs such as ketamine, atropine, pancuronium and meperidine which cause tachycardia should be avoided.
- Full pharmacologic aspiration prophylaxis should be given and a rapid sequence induction may be done.
- For intraoperative AF, synchronized cardioversion, β-blockade, digitalization, or verapamil should be utilized to control the ventricular rate.
- If pulmonary hypertension worsens, correction of hypercarbia and hypoxia should be considered. Inotropes such as dobutamine or dopamine and vasodilatation with nitroglycerin may be used.
- Ergot alkalosis for augmenting the uterine contraction should be avoided.
- Patient diuretics like frusemide to prevent overloading of circulation and pulmonary edema form autotransfusion by contracting uterus.

As puerperium is a time of increased maternal mortality, all patients with moderate to severe mitral stenosis should be monitored in an intensive care setting for 24 to 48 hours postpartum.

Mitral Regurgitation in Pregnancy

MR is the second most common valvular lesion during pregnancy. The common causes of MR in pregnancy include rheumatic fever, mitral valve prolapse and ischemic coronary artery disease.

For Labor and Delivery

- Epidural anesthesia is recommended for labor and delivery, because it will prevent the increases in SVR that occur after painful contractions.
- In case of hypotension, positive chronotropic effects of ephedrine will be beneficial.

For Cesarean Section

- Epidural anesthesia is likewise preferred for cesarean section.
- If general anesthesia is administered, a rapid sequence induction should be carried out. Lignocaine 1 mg/kg may be used to ameliorate the sympathetic response to intubation.

Aortic Insufficiency (AI) in Pregnancy[13,16]

- AI is relatively less common in pregnancy.
- The anesthetic choice preferred for both labor and cesarean section is epidural anesthesia.
- Bradycardia should be avoided and increases in SVR should be aggressively treated, with either a rapid acting vasodilator or an inhalation agent if LV function is adequate.
- As in the nonpregnant women, acute aortic regurgitation—for example, owing to aortic dissection or aortic valve endocarditis—is a surgical emergency. Women with acute mitral regurgitation, due to a ruptured chord for example, may initially be stabilised with an intra-aortic balloon pump, but typically require urgent surgery.

Aortic Stenosis in Pregnancy

Aortic stenosis is the least common valvular heart disease seen during pregnancy, found in approximately 1% of parturients. Medical management of symptoms is challenging and includes bed rest, oxygen, treatment of exacerbating factors, α-blockers and cautious diuresis if volume overload is present. Drugs that decrease afterload may be hazardous, because of the relatively fixed obstruction at the valve level.

In patients with persistent hemodynamic compromise, percutaneous valvotomy is the preferred option.

Percutaneous aortic valve implantation is an emergent technique alternative to surgical aortic valve replacement in high risk patients with aortic stenosis that are undergoing rapid development and currently represent a dynamic field of research.[17]

For Labor and Delivery

- If appropriate monitoring and fluid replacement are ensured, a slowly titrated epidural anesthetic can be safely used for labor analgesia.
- Hypotension should be aggressively treated with fluids and an α-agonist such as phenylephrine.

For Cesarean Section

- In case of mild stenosis, a standard rapid sequence induction with thiopentone and succinylcholine or graded epidural anesthesia is acceptable.

- In presence of critical aortic stenosis, a standard rapid sequence induction may lead to increases in heart rate that causes significant myocardial infarction. Precaution should be taken to avoid hemodynamic response to laryngoscopy and intubation.

Anticoagulation in Pregnancy with Valvular Heart Disease[16,18,19]

The major difficulty in management of women with mechanical prostheses during pregnancy is the anticoagulation requirement. Pregnancy is a thrombogenic state, and so pregnant women with a mechanical prosthesis are at increased risk. The ideal goal is continuous effective anticoagulation that is safe for both the mother and fetus.

- Warfarin is probably safe during the first 6 weeks of gestation, but there is a risk of embryopathy if warfarin is taken between 6 and 12 weeks of gestation. Warfarin is also relatively safe during the 2nd and 3rd trimester of pregnancy, but needs to be discontinued and switched to a heparin compound several weeks before delivery. It is avoided in the third trimester since it crosses the placenta and can cause fetal hemorrhage. It also precludes regional anesthesia and its effects may be difficult to rapidly reverse in an emergency.
- In pregnant patients with mechanical prosthetic valves, warfarin should be discontinued and continuous IV unfractionated heparin may be given starting at 2 to 3 weeks before planned delivery.
- Low molecular weight heparin (LMWH) can be used instead of warfarin throughout the pregnancy. Regional anesthesia can be performed provided adequate time has elapsed since the last dose of LMWH.
- For women receiving prophylactic LMWH, regional anesthesia or removal of epidural catheter can be performed 12 hours after last dose of LMWH. After insertion of epidural or spinal a dose can be given 4 hours later.
- In women receiving therapeutic doses of LMWH, 24 hours should elapse after the last dose of LMWH before regional anesthesia or removal of epidural catheter. After insertion of epidural or spinal a dose can be given 4 hours later.

Prophylactic Antibiotics to Prevent Endocarditis

Infective endocarditis is uncommon in pregnancy, but when it occurs it presents difficulties in management. Antibiotics must be chosen to safeguard the life of the mother but also try to avoid damage to the fetus.[14]

American Heart Association (2007) guidelines and the UK National Institute for Health and Clinical Excellence (NICE) 2008 guidelines do not recommend administration of antibiotics solely to prevent endocarditis in patients who undergo a gynecological or obstetric procedure since there is no beneficial evidence of this practice.

In nonpregnant patients, surgery is indicated in patients with life-threatening heart failure or cardiogenic shock due to surgically treatable valvular heart disease with or without proven infective endocarditis (IE). Surgery is recommended for patients with annular or aortic abscesses, those with infections resistant to antibiotic therapy, and fungal endocarditis.[19]

Risk Stratification for Pregnant Women with Valvular Heart Disease[16]

High risk of adverse maternal and fetal outcomes is considered if any of the following is present;

- Prior cardiac event or arrhythmia
- New York Heart Association class >2 or cyanosis
- Systemic ventricular dysfunction (ejection fraction <40%)
- Pulmonary hypertension (pulmonary arterial systolic pressure >50 percent systemic pressure)
- Left heart obstruction
- Severe aortic or mitral regurgitation with NYHA class III or IV symptoms

Special Considerations of Redo Surgeries with Prosthetic Heart Valves

Prosthetic Valve Leak

Whenever a patient with a prosthetic heart valve has a hemodynamically significant leak through or around that valve, the problem is potentially serious. The first step is to exclude infective endocarditis. Subsequently full hemodynamic assessment is necessary. The occurrence of severe hemolysis in relation to a prosthetic leak contributes to the need for a reoperation.

Prosthetic Valve Thrombosis

This is a serious and often fatal complication of prosthetic valve replacement. Thrombosis of mechanical valves can carry mortality as high as 60 to 80 percent hence anticoagulation is mandatory in all patients with mechanical prosthetic valves.[20]

The clinical presentation is that of pulmonary edema, arrhythmias and cardiogenic shock. On

auscultation the valve sounds are often faint or may even be absent. New murmurs may also be audible. The diagnosis is confirmed echocardiographically. Thrombosis on a mechanical prosthetic valve is a grave emergency and most patients require urgent/semi-urgent surgery for either removal of the thrombus or replacement of the valve depending on the findings at the time of the surgery. The use of fibrinolysis has been considered in an attempt to avoid the high morbidity and mortality risks of surgery in a desperately ill patient. (Streptokinase in a loading dose of 2.5-5 lac units followed by 1 lac unit hourly). Fibrinolysis is the preferred treatment for patients with thrombosis of tricuspid valve prosthesis. In general, patients with aortic and mitral valve prosthesis are candidates for surgery unless it is contraindicated.

Prosthetic Valve Infective Endocarditis

It is a very serious complication of prosthetic valve replacement and can be early onset (within 2 months of surgery) or late onset. Most of the early onset infections are caused by organisms isolated from equipment such as cardiopulmonary bypass tubings or contaminated IV infusion lines. The organisms involved are *staphylococci (S. epidermitis and S. aureus)*, gram-negative bacilli and fungi. In late onset infection, the portal of entries is bacteremia caused by dental treatment, skin infection and deep-seated infections. The most common organisms involved here are *staphylococci*.

A high index of suspicion must be maintained in relation to all patients with prosthetic valves. The presence of pyrexia, a raised ESR, unexplained anemia or leukocytosis, spleenomegaly, petechiae, clubbing or systemic emboli in a patient with a prosthetic valve should alert the attending doctor to the possibility of prosthetic valve infective endocarditis.

Management

Any patient with a prosthetic heart valve and a suspected of prosthetic valve infective endocarditis should be admitted immediately to the cardiology or cardiac surgical unit. Antibiotic treatment should be initiated immediately after 3 to 6 blood cultures have been taken wherever there are reasonable clinical grounds for suspecting Prosthetic valve infective endocarditis. Those cases most likely to be cured medically are the late onset cases in which the infecting organism is a *Streptococcus*. Maintaining cardiovascular stability with optimal hemodynamic parameters and adequate systemic perfusion pressure during the anesthetic management of patients with valvular heart disease can be extremely challenging especially in emergent situations.

Those cases most likely to require urgent replacement are the early cases in which the infecting organism is a *Staphylococcus*.

The major indications for surgery are:

- Prosthetic valve dysfunction
- Prosthetic valve dehiscence
- Uncontrolled infection
- Moderate aortic regurgitation and congestive heart failure in prosthetic valve infective endocarditis
- Recurrent relapse of infection
- Persistently positive cultures despite appropriate antibiotic treatment
- Myocardial or valve ring abscesses.

The destructive effect of infection is particularly great around the sewing ring of the valve with destruction of the annulus, valve dehiscene, abscess formation and even rupture into cardiac chambers. Surgery must be radical involving total removal of infected valve. There is no question of just placing additional sutures along the area of valve dehiscence. Abscesses must be evacuated and all the infected tissue removed. In the worst cases valve dacron grafts may have to be used. Antibiotic treatment should be continued for a minimum of 6 weeks after surgery. The optimal antibiotic regime will depend upon the relevant bacteriological information, but large doses of appropriate bactericidal antibiotics are necessary. Once the antibiotic treatment has been instituted, it is essential to keep watch on the patient. Surgery should be undertaken immediately if there is an evidence of significant hemodynamic deterioration or of failures to control the infection.

All types of mechanical valves have suffered sudden mechanical failure. Two-third of all cases reported have been fatal with most of the patients having died before they could reach the hospital. Those patients who reach the hospital and were diagnosed immediately; emergency surgeries have saved there lives. The successful approach to long term care depends on the application of the following principles:

- Education of the patient
- Education of the general practitioner
- Routine clinical checks
- Immediate and optimal management of any problem revealed.

Left Ventricular Failure due to Malfunction of Prosthetic Valve

This can occur in significant number of patients with all types of prosthetic valves. It is generally gradual in

onset, but occasionally it occurs suddenly with fatal outcome. When a patient with a prosthetic valve develops new or unstable angina, syncope, dyspnea, increasing hemolysis or transient ischemic attacks, valve failure should be suspected. These patients will be taken up for removal of malfunctioning valve. Anesthesia management will be according to general condition and hemodynamic parameters of the patient.

CONCLUSION

Patients with valvular heart disease should have the severity of their valvular disease and functional class determined by a cardiologist before urgent or elective surgical operations or pregnancy. The cardiologist can then determine if the patients can undergo the operation with an acceptable risk. Continuous, skilled echo-cardiographic assessment and interpretation are important for optimal intraoperative management of the patients with valvular heart disease. The minimally invasive valve repair procedures are likely to offer encouraging outcomes.

REFERENCES

1. James ED, Paul E Fenster. Mitral Stenosis. In: James ED, Joseph SA (Eds). Valvular Heart Disease. Lipincott Williams and Wilkins; 2000. 1981; pp. 75-112.
2. Deepak KT. Anesthesia for Valvular Heart Disease. In: Deepak KT (Ed). Clinical practice of cardiac anesthesia, 2nd edn. Modern publishers; 2001; pp. 73-94.
3. Mikhail RS, Donald EM. Anesthetic Management for the surgical treatment of valvular heart diseases. In: Fredrick AH Jr, Glenn TG (Eds). A practical approach to cardiac anesthesia 4th edn. Lippincott, Williams and Wilkins. pp. 316-47.
4. Alec V, Helmut B. Guidelines on the management of valvular heart disease. European Heart Journal 2007; 28: 230-68.
5. Nancy AN, Michael CH. Anesthesia for Cardiac Surgical Procedures. In: Miller RD (Ed). Miller's Anesthesia 7th edn, New York, Churchill Livingstone; 2010; pp. 1889-1969.
6. Robert KS, Stoelting, Stephen FD. Valvular Heart Disease. In: Robert KS, Stephen F. Dierdorf (Eds). Anesthesia and Co-existing Disease 4th edn Churchill Livingstone; 2002 pp. 25-44.
7. Sheldon G, Michael ST. Mitral valve disease. In: Joseph IS (Ed). Anesthesia and the patient with co-existing heart disease, 1st edn, New York, Library of congress cataloging, 1993; pp. 129-72.
8. Sanjula V, Deepak KT. Effect of Muscle Relaxants on Heart Rate, Arterial Pressure, Intubating Conditions and Onset of Neuromuscular Block in Patients Undergoing Valve Surgery. Annals of Cardiac Anesthesia 2006;9:37–43.
9. Braunwald E. Valvular heart disease. In: Braunwald E (Ed). Heart Disease: A Textbook of Cardiovascular Medicine, 5th edn, WB Saunders Co., Philadelphia, 1997. pp. 1007-76.
10. Edith RS, Christoph B. Inhaled Nitric Oxide Versus Intravenous Vasodilators in Severe Pulmonary Hypertension After Cardiac Surgery. Anesthesia & Analgesia 1999;89(5):1108.
11. Mackson JM, Thomas SJ. Valvular Heart Disease. In: Kaplan JA, Reich DL, Konstadt SN (Eds). Cardiac Anesthesia, 4th edn, WB Saunders Co, Philadelphia 1999; pp 727-84.
12. Jean GD, Philippe P. Paradoxical low flow and/or low gradient severe aortic stenosis despite preserved left ventricular ejection fraction: implications for diagnosis and treatment. Eur Heart J 2010;31:281-9.
13. David W. The pregnant patient with cardiac disease. In: Joseph I. Simpson (Ed). Anesthesia and the patient with co-existing heart disease, 1st edn, New York, Library of Congress cataloging 1993. pp. 299-334.
14. Aikaterini C, Michael AG. Heart Disease and Pregnancy. Hellenic J Cardiol 2006;47:275-91.
15. Souza JA, Martinez E, Ambrose J, et al. Percutanous balloon mitral valvuloplasty in comparison with open mitral valve commissurotomy for mitral stenosis during pregnancy. J Am Coll Cardiol 2001;37:900-3.
16. Karen KS, Catherine M Otto. Pregnancy in women with valvular heart disease. Curr Cardiol Rep. 2006; 8(2): 83-9.
17. Rogério TT, Norberto TD, Percutaneous Balloon Aortic Valvuloplasty in a Pregnant Adolescent. Arq Bras Cardiol 2003;(82):98-101.
18. Matt R. Abbi W. Cardiac disease in pregnancy: tutorial 2 AT OTW 118, 10th November 2008.
19. Robert OB, Blase AC. Antithrombotic Management of Prosthetic Heart Valves: Pregnancy. In: Management of Patients With Valvular Heart Disease, ACC/AHA Pocket Guideline, American College of Cardiology Foundation and the American Heart Association, 2006; pp. 53.
20. Grotte GJ. Long-term management of patients with prosthetic heart valve. In: Recent advances in cardiology, Derec J. Rowland, 1992; pp. 271-90.

7

Anesthesia for Emergency Blalock-Taussig Shunt

Manju N Gandhi, Aparna A Nerurkar, Deepti M Kotwani

KEY POINTS

- Palliative shunts are indicated in cardiac disorders which are not generally amenable to initial corrective surgery, owing to either anatomic (Tetralogy of Fallot with pulmonary atresia or small pulmonary arteries) or physiologic causes (*tricuspid atresia* or another single ventricle situation).
- Modified Blalock-Taussig Shunt (MBTS) is currently the most commonly used type of shunt.
- The anesthesiologist should assess age at presentation, frequency of episodes, cyanosis, respiration, congestive heart failure and associated anomalies as these affect the anesthetic management.
- Coagulation abnormalities like hyperfibrinolysis, decrease in clotting factors and functional platelets may be present. Thorough evaluation of coagulation is indicated with appropriate blood products availability.
- Preoperatively, it is important to avoid dehydration by providing intravenous fluids before the patient's arrival in the operating room.
- Crying associated with intramuscular administration of drugs used for preoperative medication can lead to hypercyanotic attacks, therefore oral/nasal or intravenous premedication is preferred.
- Morphine, phenylephrine, propranolol and sodium bicarbonate or their substitutes should be readily available to treat hypercyanotic spell.
- Pulse oximeter and blood pressure monitoring devices should be preferentially placed on side opposite to proposed shunt.
- Main goal of induction in a cyanotic patient is to establish a necessary level of anesthesia without increasing the right-to-left shunt.
- Maintaining systemic vascular resistance, decreasing pulmonary vascular resistance, mild myocardial depression and euvolemia with a slow heart rate should be the main principles of induction.
- Ketamine increases systemic vascular resistance and decreases right-to-left shunt and hence is preferred choice of induction agent.
- A postshunt oxygen saturation of near 80 percent or a 10 percent increase as compared to preoperative level is optimal as this represents balanced pulmonary and systemic blood flow.
- Treatment of intraoperative hypercyanotic spells involves increasing the depth of anesthesia, hyperventilating with 100 percent oxygen, administering volume, increasing systemic vascular resistance with phenylephrine, decreasing infundibular spasm with β-blockers and administration of sodium bicarbonate to be considered to treat any ensuing metabolic acidosis.
- Extubation in emergency shunt placement with high probability of hemodynamic, metabolic, and pulmonary problems should wait until resolution of these issues. Mechanical ventilation is usually necessary for at least 12 to 24 hours after arrival in the intensive care unit.
- Intercostal or caudal blocks or suppositories (e.g. paracetamol) can be given for postoperative pain relief.

There are not many community-based data on prevalance of congenital heart diseases in India.[1] However, according to a hospital-based status report on congenital heart diseases (CHD) in north India, congenital heart diseases are relatively common with a prevalence ranging from 3.7 to 17.5 per 1000 live births. 10 percent of the present infant mortality is accounted by CHD. In one of the community-based studies from India the prevalence of CHD ranges from 0.8 to 5.2/1000 patients. Thus, the prevalence of CHD is not uniform

across the country and setting. Tetralogy of Fallot is the commonest cyanotic heart disease (4.6%).[2]

Due to improved diagnostic tools and outcome, the population of patients with congenital heart disease undergoing surgery is growing. The continued evolution in care of patient with CHD, advances in technology, modern perioperative supportive care and refinements in operative technique has increased the advent of initial corrective procedures during infancy. This has made palliative shunt procedures less common treatment for most cyanotic heart malformations.

Despite this there are a certain subsets of patients who require initial palliative surgery because their cardiac disorders are not generally amenable to initial corrective surgery, owing to either anatomic causes may make patient unsuitable for initial corrective surgery (TOF with pulmonary atresia or small pulmonary arteries) or physiologic causes (tricuspid atresia or another single ventricle situation). [3]

Indications for Palliative Systemic-to-pulmonary Shunt[3]

The indications for a systemic-to-pulmonary artery shunt include the following:

- *Tetralogy of Fallot (TOF):* Many centers advocate early (often neonatal) complete repair, However, due to concerns about potential attrition from palliative procedures, effects of chronic cyanosis, and better development of the pulmonary arteries with early repair; the timing of complete repair , however, remains controversial.

  The palliative shunt may initially be performed in some situations, such as:
  - In cases of TOF with hypoplastic arteries.
  - In cases of TOF in which an anomalous coronary artery, usually the left anterior descending coronary artery, crosses the right ventricular (RV) outflow tract.
  - In cases of TOF with associated complex cardiac lesions.
- *Tricuspid atresia:* Most patients with tricuspid atresia have pulmonic stenosis in the presence of normally related great arteries. These patients require an initial palliative systemic-to-pulmonary artery shunt prior to a Glenn or a Fontan procedure.
- *Pulmonary atresia***:** The systemic-to-pulmonary artery shunt is the method of choice to establish ductal independent pulmonary blood flow.
- *Ebstein's anomaly*
  This condition is often associated with functional pulmonary atresia which may necessitate a systemic -to-pulmonary artery shunt.
- *Single ventricle situation with pulmonary or aortic atresia.*

TYPES OF SHUNTS

There are several types of palliative shunts. An ideal shunt should have following characteristics:

- Be rapid and technically simple to perform
- Provide adequate but not excessive pulmonary blood flow, hence minimizing the risk of congestive cardiac failure and pulmonary hypertension
- Provide good long-term patency
- Be technically easy to close when complete repair is planned
- Result in no residual cardiopulmonary abnormalities after closure.

Although several modifications of a systemic-to-pulmonary artery shunt are known and are briefly discussed below, the modified Blalock-Taussig shunt is the most common type currently used.

- **Classic Blalock-Taussig shunt (CBTS):** It involves a direct anastomosis between the transected subclavian artery (or the innominate artery) and the pulmonary artery on the side opposite the aortic arch
- **Potts shunt:** A connection between the descending aorta and left pulmonary artery
- **Waterston shunt:** This connection is between the ascending aorta and right pulmonary artery
- **Cooley shunt:** This is an intrapericardial anastomosis from the ascending aorta to the right pulmonary artery
- **Modified Blalock-Taussig shunt:** Modified Blalock-Taussig shunt (MBTS) is currently the most commonly used.

An interposition polytetrafluoroethylene (PTFE, or Gore-Tex) graft between the subclavian artery and the pulmonary artery is used to prevent sacrificing the subclavian artery.

Advantages:
- Prevents the mutilating effects of the CBTS (Classic Blalock-Taussig shunt)
- When CBTS cannot easily be performed, such as on the same side as the aortic arch.
- Pulmonary artery distortion is less likely than with CBTS.
- Closure of these shunts is technically easy.

- Excellent patency rates of 90 percent at age 2 years have been reported.

- **Central shunt:** A central shunt is an anastomosis between the ascending aorta and the main pulmonary artery made of polytetrafluoroethylene (PTFE).

  Indications:
  - Neonates and children younger than 3 months.
  - It can be performed only in infants with a patent ductus arteriosus or some other source of pulmonary blood flow.
  - A central shunt may be especially useful with bilateral small branch pulmonary arteries, a concomitant procedure requiring a median sternotomy, or both.

  Advantages:
  - It is applicable to small children with small peripheral vessels.
  - Prevents distortion of pulmonary arteries.
  - Provides equal pulmonary blood flow to both lungs.
  - Lower occlusion rate (compared with the CBTS or MBTS techniques).
  - Avoids subclavian artery steal.
  - Easy to close during corrective repair.

  Disadvantages:
  - Entry into the pericardium.
  - Inapplicable for patients without a patent ductus arteriosus or other source of pulmonary blood flow.

- **Glenn Shunt:** It is end-to-side anastomosis between superior vena cava and the right pulmonary artery which provides unidirectional pulmonary blood flow. This is now termed the classic Glenn shunt. The modification in current use is termed the bidirectional Glenn which involves connection of the superior vena cava to the right pulmonary artery, which is still connected to the main pulmonary artery hence, blood from the superior vena cava can enter both the pulmonary arteries.

  Advantages: It does not increase the volume load on the ventricle.

  Disadvantages: Specific anatomic and physiologic limitations, the most important being pulmonary vascular resistance must be low. As a result, children younger than 3 to 6 months are generally excluded. Similarly, patients with any degree of elevated pulmonary vascular resistance are generally excluded.

- **Sano Shunt:** It is a right ventricle-to-pulmonary artery shunt in an attempt to overcome the obstacles noted with a systemic-to-pulmonary artery shunt.

  Advantages:
  - Improvement after Stage 1 mortality from 62 to 89 percent.
  - Overcome the obstacles noted with a systemic-to-pulmonary artery shunt, i.e. coronary insufficiency due to low diastolic pressures in the aorta caused by the run-off from the systemic circulation in favor of the pulmonary circulation.

  Disadvantages:
  - These shunts become obstructive over time, usually in approximately 3 months. This occurs sooner than that noted with MBTS.
  - The effects of a ventriculotomy on a systemic ventricle are of concern.

CLINICAL PRESENTATION

The initial presentation of the patient with TOF depends on the degree of right ventricular outflow tract (RVOT) obstruction. The severity of hypoplasia of RVOT obstuction varies from mild (pulmonary stenosis) to complete (pulmonary atresia).

Patients of Tetralogy of Fallot with pulmonary atresia often become symptomatic within the first hours to days of life depending on the source and volume of pulmonary blood flow which is usually via the ductus arteriosis (duct dependent circulation) and/or aorto-pulmonary collaterals.

Cyanosis is the most common clinical presentation. Most commonly cyanosis is mild at birth and gradually progresses with age as the obstruction increases as a result of increasing hypertrophy of the right ventricular infundibulum.

A few patients, however, have significant cyanosis at or shortly after birth. In this group, the RVOT obstruction is nearly always due to a hypoplastic pulmonary valve with or without severe right ventricular infundibular obstruction or hypoplasia. Cyanosis is constant in these patients because of the fixed nature of the obstruction to pulmonary blood flow.[4]

Also in case of pulmonary atresia severe cyanosis becomes apparent immediately after birth as the ductus arteriosus begins to close. Therefore, early diagnosis and treatment with prostaglandin (PGE_1) is life saving in this instance. Similar duct dependent circulation is present in tricuspid atresia and Ebstein's anomaly. In

these patients, cyanosis may be mild to moderate only, if they have adequate aortopulmonary collaterals or additional sources of pulmonary blood flow.[5]

The older infant and child usually presents with:

- History of cyanosis and hypoxic spells which are due to the labile infundibulum, obstruction of which leads to the characteristic crisis of hypoxemia (Tet spells - described later)
- Physical limitation with increased fatigability
- Shortness of breath in response to low oxygen levels and reduced pulmonary blood flow can occur
- Failure to thrive secondary to pulmonary hypertension and poor peripheral oxygenation and metabolite delivery
- Profuse prespiration particularly with feeds
- Irritability during feeding
- Infant often takes frequent small feeds and becomes rapidly exhausted (suck rest suck)
- Recurrent respiratory infections
- History of squatting.

Hypoxia usually progresses further as the child grows and the pulmonary blood flow is inadequate.

On rare occasions, patients with well-developed aorto-pulmonary collaterals or patent ductus arteriosus (PDA) may present with heart failure. Symptoms develop several weeks after birth as the pulmonary vascular resistance decreases and pulmonary blood flow (PBF) increases.[5]

Older children and adults usually do not experience the typical hypoxic episodes:

- Abnormal response to physical exercise is the common feature
- These patients have progressive myocardial hypertrophy which is more pronounced in older patients due to long-term cyanosis and pressure overload thereby increasing chances of myocardial dysfunction and arrhythmias
- The main physiological changes to be considered in these patients are arrhythmias, hypoxemia, hyperviscosity, endocarditis and coagulation abnormalities
- History of repeated admissions to the hospital to bleed (phlebotomies) adult patients with congenital cynotic heart disease is not uncommon.

However, natural history of the disease is such that, if TOF is not repaired, 70 percent of the children with this disorder die before the age of 10 years, but survival to adulthood is possible although after the age of 40 years, the rate of survival is not more than 3 percent.[6]

In patients with tricuspid atresia and Ebstein's anomaly the clinical presentation will vary depending on the anatomical aberration. Electrophysiologic abnormalities like paroxysmal supraventricular tachycardia (20 to 25% of children), AV nodal conduction disturbance, atrial fibrillation or flutter and other abnormalities are common. Noncardiac presentations are due to interatrial communication they are at a risk of paradoxical embolization, brain abscess, congestive heart failure, and sudden death.[7]

Associated cardiac anomalies are[8]:

- Multiple muscular ventricular septal defect (VSD) (about 3 to 15%)
- Right-sided aortic arch (25%)
- Atrial septal defect (ASD) (9%)
- Persistent left superior vena cava (8%)
- Abnormalities in the coronary artery origin and distribution (5%)
- Patent ductus arteriosis (4%)
- Partial anomalous pulmonary venous drainage (1%).

Associated syndromes and extracardiac malformations[5]:

- CATCH 22 (cardiac defect, abnormal face, thymic hypoplasia, cleft palate, hypocalcemia, microdeletion of band 22q11)
- VATER syndrome (vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, and renal and radial anomalies)
- CHARGE syndrome (coloboma, heart disease, atresia choanae, retarded growth and retarded development and/or CNS anomalies, genital hypoplasia, and ear anomalies and/or deafness)
- Maternal diabetes mellitus (20-fold higher risk); maternal *phenylketonuria*; and maternal ingestion of retinoic acid, trimethadione, or sex hormones increase the risk of conotruncal abnormalities
- Recurrence risk in siblings of patients with tetralogy of Fallot is 3 to 4 percent.

Physical Examination

Physical findings are not specific for TOF and will vary according to the source and volume of pulmonary blood flow:

- Cyanosis: Clinical cyanosis depends on the absolute concentration of deoxygenated hemoglobin rather than the oxygen saturation. The oxyhemoglobin saturation at which central cyanosis becomes clinically apparent varies from 62 percent with the hemoglobin level of 8 gm to 18 gm percent in the polycythemic infant with hemoglobin of 24 gram percent[9]
- Peripheral pulse and blood pressure are usually normal during the first few days of life. Patients with increased pulmonary blood flow may be noted to have bounding pulses

- Clubbing is a relatively late finding
- Auscultation reveals a normal first heart sound with a single second heart sound. A crescendo–decrescendo systolic ejection murmur is heard loudest in the second and third intercostal space that may radiate to the axilla. The typical right ventricular outflow tract murmur of classic tetralogy of Fallot is not heard. The intensity of the murmur will be diminished with increasing obstruction and absent during a hypercyanotic spell
- In Ebsteins anamoly triple or quadruple heart sound, often with a soft, high-pitched systolic murmur. A soft, scratchy mid-diastolic murmur heard best at the left sternal border and apex may be present. The second heart sound is widely split with little respiratory variation due to delayed emptying of the right ventricle[7]
- A soft continuous murmur from the ductus arteriosus may occur at the left base. A continuous murmur from the aortopulmonary collaterals may be heard in the back
- Growth and development are often delayed
- Pulse oximetry will demonstrate low hemoglobin saturation
- In Ebstein's anomaly with failure, the child may be diaphoretic, tachypneic, and irritable with rales present on chest auscultation and hepatomegaly on abdominal palpation.

LABORATORY STUDIES

Complete Blood Count[10]

- Polycythemia is seen in these patients. If hematocrit level is >70 percent and patient is symptomatic, phlebotomy may be indicated. Cyanotic infants do not develop the typical physiological anemia of infancy. Even poor nutrition and iron deficiency can prevent increase in hematocrit
- Acute infection leads to increase in leukocyte count predominantly polymorphs.

Coagulation[10]

- Peripheral sludging in polycythemic children induces hyperfibrinolysis due to stasis and thrombosis
- Decreased clotting factors
- Decreased number of functional platelets
- Vit K dependent factors may be reduced by hepatic dysfunction.

Other Laboratory Evaluations

- Chronic renal hypoxemia usually causes increased levels of serum blood urea nitrogen (BUN) and creatinine[6]
- Children on diuretic therapy are at risk for hypokalemia, particularly if they are digitalized
- Infants, particularly those in congestive heart failure, are also at risk for both hypoglycemia and hypocalcemia.[11]

Chest Radiograph

- Shows a characteristic "boot shaped" heart which is a reflection of RV hypertrophy and a concave upper left heart border from a small or absent main pulmonary artery
- The lung fields are oligemic from diminished blood flow
- When congestive heart failure is present the CXR demonstrates cardiac enlargement, increased pulmonary vascular markings and areas of atelectasis despite hyperexpansion of the lung.
- In Ebsteins anamoly moderate to severe cardiomegaly with a large right atrium and diminished pulmonary vascular markings will be seen. The heart often has a globular shape.[7]

Electrocardiogram

- Shows ventricular strain pattern (ST segment and T-wave changes), right ventricular hypertrophy and right axis deviation[10]
- In Ebstein's anomaly there is right atrial hypertrophy, an increased PR interval, and complete or incomplete right bundle branch block, the preexcitation patterns of Wolff–Parkinson–White syndrome (10–15% of individuals) is seen. [7]

Echocardiography is usually the definitive diagnostic modality for TOF with pulmonary stenosis. Detailed elucidation of anatomical features can be accomplished through echocardiography.

Cardiac Catheterization is occasionally required:

- Catheterization can provide information on PVR, the ratio of pulmonary blood flow to systemic blood flow (Qp: Qs), and degree of valvular pulmonary stenosis
- Angiography will display the coronary and pulmonary artery anatomy, pulmonary collateral circulation, and other cardiac anomalies. Much of this information can be gathered from transthoracic echocardiography.[8,12]

HYPERCYANOTIC (TET) SPELLS[8,10]

Hypercyanotic spells or "TET spells" are paroxysmal episodes in which the cyanosis acutely worsens. These spells can occur spontaneously, but are usually in response to crying, feeding, defecation, agitation, injury or fright which increases sympathetic tone leading to increased contractility producing infundibular spasm. The common pathway for all these activities is an increase in right-to-left shunting. "TET spells" in awake patient are usually accompanied by hyperventilation secondary to the metabolic acidosis and hypoxemia.

An infant with TOF and hypercyanotic spells is a medical emergency because a prolonged hypercyanotic spell can result in brain ischemia and death.

Three mechanisms can explain the increase in shunt:

1. Increase in PVR: This is associated with a reduction in pulmonary blood flow and increase in right-to-left shunt.
   - Treatment is to lower PVR
     - Through hyperventilation with 100 percent O_2
     - Sodium bicarbonate 1 mEq/kg to temper the effects of acidosis on PVR.
2. Dynamic right ventricular outflow obstruction (infundibular spasm):
   - Tachycardia, hypovolemia and increased myocardial contractility can cause infundibular spasm. Similar to an increase in PVR, spasm decreases blood flow into the pulmonary artery and worsens right-to-left shunt.
   - Treatment:
     - β-blockers (to relax the contracted infundibulum and to allow more time for right ventricular filling, improving pulmonary blood flow). Propranolol (0.1-0.2 mg/kg IV) or esmolol (0.5 mg/kg IV over one minute)
     - IV Fluids 10 to 20 ml/kg for volume expansion
     - Morphine (0.1 to 0.2 mg/kg IV/IM) can be given to diminish the hyperpneic response.
3. Decrease in systemic vascular resistance (SVR): This will favor right-to-left shunting through the VSD.
   - Treatment:
     - Volume administration to ensure adequate filling of the right ventricle
     - Adrenergic agonist to increase the SVR. (Phenylephrine 5-10 μg/kg and titrated to effect)
     - SVR can also be increased by flexing the legs or giving knee-chest position (older children usually squat spontaneously and do not develop TET spells).

If all medical therapies fail and the infant remains severely cyanotic, an emergency Blalock-Taussig shunt or extracorporeal membrane oxygenation may be necessary.

Propranolol has been used in the past in the outpatient setting of an infant with spells to delay surgery. Currently, an infant with a single spell is considered an indication for urgent surgical repair; however, oral propranolol 0.25 to 1.0 mg/kg q 6 hours may be used to prevent recurrences until surgery can be arranged.

Pink TET[13]

A pink TET is a patient with TOF with a source for adequate pulmonary blood flow. The additional pulmonary blood flow can come from a patent ductus arteriosus (PDA), aortopulmonary collaterals, or other naturally occurring collateral vessels to the pulmonary artery (bronchial, intercostal, or coronary arteries). In the rare case in which the right ventricular outflow obstruction is not significant, the degree of right-to-left shunt is reduced in favor of an increase in pulmonary blood flow, also causing a pink TET. With time, the degree of outflow obstruction worsens and this type of pink TET evolves into cyanotic TOF.

MANAGEMENT OF ANESTHESIA

Management of anesthesia is geared towards early extubation, mobilization and feeding of the infant.

Preoperative Preparation

NPO status: Preoperatively, it is important to avoid dehydration by providing intravenous fluids before the patient's arrival in the operating room. General NPO guidelines can be followed. The child is allowed any solid food and particulate fluid (like milk, formula, or breast milk) up to 6 hours before surgery and clear liquids up to 2 hours before surgery.[14]

Premedication: Crying associated with intramuscular administration of drugs used for preoperative medication can lead to hypercyanotic attacks therefore oral premedication is recommended in these patients.

- < 6-month-old infant: Premedication is not normally necessary
- 6-month-old infant: Oral midazolam 0.25 to 0.50 mg/kg (maximum, 20 mg) as used by various authors generally results in a very compliant child who will separate from parents without crying
- Oral ketamine (4 to 6 mg/kg) combined with atropine (0.02 mg/kg) and midazolam (0.5 mg/kg, maximum of 20 mg), will result in a deeply sedated child (Table 7.1).

Table 7.1: Common drugs for premedication

Medication	*Route*	*Dose (mg/kg)*	*Time of onset (min)*	*Elimination ½ time (hr)*
Midazolam	Oral	0.25–0.50 mg/kg (maximum 20 mg)	10	2
	Nasal	0.2–0.3 mg/kg, (conc. of 5 mg/mL 0.5 mg/puff)	<10	2–3
Ketamine	Oral	4 to 6 mg/kg	10–20	2–3
	Nasal	3 to 6 mg/kg	<10	3
	Intramuscular	2 to 4 mg/kg	5	

The disadvantages of oral or sublingual premedication are that they have slow onset of action and child may spit out the medication.

- Nasal midazolam 0.3 mg/kg can be used and a concentration of 5 mg/ml to minimize volume, although it is irritating to the nasal mucosa
- Intranasal ketamine (3 to 6 mg/kg) can also be used
- Intramuscular ketamine (2 to 4 mg/kg) combined with atropine (0.02 mg/kg) and midazolam (0.05 mg/kg). This combination is generally reserved for children who refuse oral premedication or those in whom lighter premedication regimens have failed in the past
- Higher doses of intramuscular ketamine (up to 10 mg/kg) combined with atropine and midazolam may be administered to children with anticipated difficult venous access or in whom an intravenous line is necessary for induction.
- Anticholinergic drugs are not routinely administered intramuscularly to children because they are painful on administration and do not significantly reduce laryngeal reflexes during induction of anesthesia. However, atropine (0.02 mg/kg) administered orally or intramuscularly less than 45 minutes before induction reduces the incidence of hypotension during induction with potent inhaled anesthetics, but only in infants younger than 6 months[14]
- Propranolol should be continued up to and including the day of surgery
- If patient is receiving infusion of PGE 1 to maintain ductal patency, it should be continued preoperatively
- Antibiotics should be administered intravenously before induction, if possible. In the event that intravenous access is obtained after induction, antibiotics should be given as soon as possible[15]
- Patients of Ebstein's anomaly with extreme cardiomegaly or perioperative ventricular arrhythmias should receive prophylactic antiarrhythmic treatment, such as amiodarone[7]
- Diuretics and digoxin for management of congestive heart failure should be continued[7]
- If intravenous access is absent placement of a topical anesthetic cream (EMLA) is usually helpful.

Operating Room Preparation

- The anesthesia machine must be checked and should have the capacity to provide air, oxygen, nitrous oxide to help balance pulmonary and systemic blood flow
- Intravenous tubing must be free from air bubbles to prevent paradoxical air embolism
- Standard emergency drugs for pediatric cardiac case should be available:
  - Epinephrine 10 mcg/kg
  - Atropine 20 mcg/kg
  - Calcium gluconate 10 mg/kg
- In addition, phenylephrine 2 to 10 mcg/kg and propranolol 10 to 50 mcg/kg should be available to assist in the treatment of hypercyanotic spells
- Infusions should be readily available, if inotropic support is expected as in high-risk cases. Dopamine, Dobutamine, Isoproterenol, Epinephrine, or phosphodiesterase inhibitors are all useful in these cases
- Sodium bicarbonate should be readily available.

Standard Monitoring Includes

- Electrocardiogram (ECG)
- Pulse oximetry: The pulse oximeters require pulsatile flow to work properly, hence the probe should be placed on the hand opposite to the side of the proposed shunt. Occasionally, two sites are selected – one probe on an upper extremity and a second probe on a lower extremity. Pulse oximetry also loses its accuracy in patients with severe hypoxemia[6]
- Blood pressure: An automated blood pressure cuff can be used during induction. After endotracheal intubation, an arterial line should be placed for continuous arterial pressure monitoring and arterial blood gas analysis. Radial/femoral arterial line is placed on the side opposite to that of the shunt
- End-tidal carbon dioxide: This helps determine correct placement of the endotracheal tube and assists in managing ventilation. Capnography greatly underestimates $PaCO_2$ and the degree of discrepancy is correlated with the degree of right-to-left shunt. The gradient (A- a gradient) usually decreases after successful shunt placement.[6]
- Rectal and esophageal temperature

- Urine output
- Central venous access is obtained for the infusion of fluids and vasoactive agents.

Induction of Anesthesia

Main goal of induction in a cyanotic patient is to establish a necessary level of anesthesia without increasing the right-to-left shunt. The stress of the induction can lead to a hypercyanotic spell.

Objectives

- Maintain SVR. Avoid drugs that decrease SVR and treat decreases in blood pressure promptly with vasoconstrictors
- Decrease PVR to maintain or improve pulmonary blood flow
- Favor mild myocardial depression and euvolemia because they can help prevent or limit a hypercyanotic spell
- Slow heart rate to reduce the likelihood of infundibular spasm.

If intravenous access is present: An intravenous induction is faster in patients with a right-to-left shunt because peak receptor site concentration is attained faster.

Induction of general anesthesia can be accomplished with Inj. Ketamine (1–2 mg/kg IV) or Inj. Fentanyl (up to 25 mcg/kg). Advantage of using ketamine is that it may be associated with improved arterial oxygenation, presumably reflecting increased pulmonary blood flow due to ketamine-induced increases in systemic vascular resistance, which can lead to a decrease in the magnitude of the right-to-left intracardiac shunt.[8,9,15]

For sicker and younger infants, particularly neonates, high-dose narcotic anesthesia has become a safe method. Ketamine has also been shown to be safe and to have relatively little effect on the hemodynamics in these patients.

A pure narcotic technique has the advantage of producing less myocardial depression and greater cardiac stability in the presence of pre-existing myocardial dysfunction.[8,10,15]

If intravenous access is absent: Induction can be accomplished with Inj. ketamine (4 mg/kg IM) especially in patients in whom a mask induction would be frightening.

Mask induction with oxygen and halothane or sevoflurane is always a possibility, keeping in mind that a significant right-to-left shunt may delay induction.[8,9,15] Also one must keep in mind that in case of relative overdose it may be difficult to remove the inhalational agent due to decreased pulmonary blood flow.[9]

The myocardial depressant effect of volatile anesthetic agents is also useful in limiting infundibular spasm. Although decreased pulmonary blood flow speeds the achievement of anesthetic concentrations, the hazard of decreased systemic blood pressure plus decreased systemic vascular resistance is great. Indeed, hypercyanotic attacks can occur during administration of low concentrations of volatile anesthetics.

Induction of anesthesia with sevoflurane, which has the least effect on *SVR* is acceptable in such cases, but must be accomplished with caution and careful monitoring of systemic oxygenation. Halothane may also be preferred inhalational anesthetic as it decreases contractility and maintains systemic vascular resistance.[9,16]

Once the induction of general anesthesia is completed and intravenous access is established, Inj. vecuronium (0.1 mg/kg) or pancuronium (0.1 mg/kg) can be given to facilitate endotracheal intubation.

Maintenance of Anesthesia

Maintenance of anesthesia is often achieved with nitrous oxide combined with ketamine.

Advantage of this combination is preservation of the systemic vascular resistance. Nitrous oxide may also increase pulmonary vascular resistance, but this potentially adverse effect is more than offset by its beneficial effects on systemic vascular resistance (no change or modest increase). The principal disadvantage of using nitrous oxide is the associated decrease in the inspired oxygen concentration thereby, increasing pulmonary resistance and decreased pulmonary blood flow and decreased PaO_2. Therefore, it seems prudent to limit the inspired concentration of nitrous oxide to 50 percent.

The use of an opioid or benzodiazepine may also be considered during maintenance of anesthesia, but the dose and rate of administration must be adjusted to minimize the fall in systemic blood pressure and systemic vascular resistance.

Intraoperative skeletal muscle paralysis may be provided with pancuronium in view of its ability to maintain systemic blood pressure and systemic vascular resistance. An increase in heart rate associated with pancuronium is helpful for maintaining left ventricular cardiac output. Alternative nondepolarizing neuromuscular blocking drugs like atracurium, vecuronium, rocuronium are often selected with consideration given to the ability of some of these drugs, when administered rapidly in high-doses, to evoke the release of histamine with associated decreases in systemic vascular resistance and systemic blood pressure.

Ventilation of the patient's lungs should be skillfully controlled to avoid excessive positive airway pressure which may adversely increase the resistance to blood flow through the lungs.

Intravascular volume must be maintained with intravenous fluid administration because acute hypovolemia tends to increase the magnitude of the right-to-left intracardiac shunt.

In view of the polycythemia, it is probably not necessary to consider blood replacement until approximately 20 percent of the patient's blood volume has been lost.[16]

Low SVR is treated with phenylephrine or norepinephrine; and preload is augmented with fluid boluses.

It is important to avoid most inotropes, as these will worsen infundibular spasm by increasing heart rate and contractility.[7]

A radial arterial line is placed on the side opposite to that of the MBTS in order to get a true assessment of the blood pressure because after the shunt is opened there may be significant "steal" from the ipsilateral subclavian artery. A femoral arterial line can also be placed due to technical difficulties with the radial artery and as long as care is taken to observe for evidence of distal lower extremity ischemia.[7]

OPERATIVE DETAILS OF A MODIFIED BLALOCK-TAUSSIG SHUNT

The operation may be performed on either side through a lateral thoracotomy in the forth intercostal space. The median sternotomy approach is used when the surgeon feels that the patient will not tolerate lung retraction or side-clamping of the pulmonary artery, when there is a possibility that cardiopulmonary bypass may be required, and for central shunt placement.

The pulmonary artery is dissected out, and a side-biting clamp is placed on the origin of the dissected subclavian artery. Before applying the vascular clamp, the vagus nerve and its recurrent laryngeal branch must be identified. The obliquely trimmed prosthesis is anastomosed 'end to side' on the subclavian artery.

Low-dose Heparin (100 U/kg) is administered intravenously before the Gore-Tex conduit is anastomosed to a transverse arteriotomy made on the anterior aspect of the pulmonary artery near its upper edge. The length of the graft must be adjusted so that it lies straight without kinking.[7]

Lung retraction can severely impair oxygenation and ventilation, and intermittent reinflation may be required. However, attempting to normalize PaO_2 during single lung perfusion may overdistend the dependent lung, increasing PVR and impairing venous return.

Similar decompensation can occur during partial clamping or obstruction of the PA during the construction of the anastomosis. Such decompensation is managed with fluids, vasopressors, and ventilation adjustments.

For central shunts, partial clamping of the ascending aorta is required, but it may be poorly tolerated in the presence of LV dysfunction. Inotropic support with dopamine is usually helpful.

Once the shunt is open, oxygen saturation usually improves immediately. However, blood pressure may drop significantly, requiring volume infusion and vasopressors. If the diastolic pressure becomes very low, coronary flow will be reduced and ischemic changes may be seen on the ECG.

Ventilation and inspired oxygen are adjusted to mimic spontaneous, nonanesthetized values for an accurate assessment of the shunt flow. An oxygen saturation of near 80 percent or a 10 percent increase as compared to preoperative level is optimal as this represents balanced pulmonary and systemic blood flow.

A high-saturation (> 80% or >10% increase) suggests pulmonary over circulation and the shunt size may have to be reduced. Conversely, a low-saturation suggests inadequate pulmonary blood flow (PBF), and a larger diameter shunt may be needed. Increased pulmonary blood flow (PBF) can cause unilateral pulmonary edema or pulmonary hemorrhage.[7]

In cases of persistent hypoxemia after apparently uneventful shunt placement, it is important to rule out the possibility of endobronchial intubation because failure to do so may lead to unnecessary shunt revision or even sternotomy.

De Leval et al (1981) stressed the importance of maintaining adequate systemic arterial pressure throughout the operation to prevent early thrombosis of the conduit.[3]

Patency of the shunt can be clinically confirmed by briefly disconnecting the patient from the ventilator and auscultating over the end of the endotracheal tube. The murmur is transmitted via the tracheal tube due to the proximity of the shunt to the bronchus.

A low-dose heparin infusion is started (8–10 U/kg/hour) to maintain shunt patency when the risk of post-surgical hemorrhage has diminished. Platelet transfusions are generally avoided for patients undergoing shunt placement due to the risk of shunt thrombosis.[7]

Blood is very rarely needed in shunt procedures. It should be present in the operating room in the event of any untoward events.[12]

Intraoperative hypercyanotic spells[8]*:* Manifests with a sudden decrease in the oxygen saturation. Without quick intervention, they can progress to bradycardia and systemic hypotension.

- Treatment:
  - Increase the depth of anesthesia
  - Hyperventilate with 100 percent oxygen
  - Administer volume
  - Increase SVR with phenylephrine
  - Decrease infundibular spasm with β-blockers
  - Sodium bicarbonate should be considered to treat any ensuing metabolic acidosis.

Requirements for Extubation

Spontaneous ventilation with normal blood gases, normothermia, adequate pain control, and stable hemodynamics are required before extubation.

Extubation following an elective shunt procedure can usually be accomplished soon after the completion of surgery. Extubation is usually performed in the operating room or soon after arrival in the intensive care unit.

Emergency shunt placement implies that the patient is unstable before surgery. The probability of hemodynamic, metabolic, and pulmonary problems is high. As such, extubation in these patients should wait until resolution of these issues and patient should be shifted to cardiac intensive care unit where mechanical ventilation is usually necessary for at least 12 to 24 hours. [16]

After enteral intake has begun, the patient is prescribed aspirin until the time of corrective surgery.[7] Role of low-dose aspirin in preventing shunt thrombosis has not been systematically studied. A small study from Germany found that aspirin effectively reduced the rate of shunt occlusion. However, another study showed no difference in patency rates.[3]

Postoperative Ventilation Issues[8,15]

Postoperative ventilation is directed at increasing pulmonary blood flow, i.e. increased FiO_2, mild hyperventilation ($PaCO_2$ around the low 30-35 mm Hg), euthermia, and mild alkalosis which will promote a decrease in PVR and increase pulmonary blood flow. The greatest benefit of this ventilatory management is seen following placement of a shunt, although, it helps after complete correction.

Disadvantages are pressures generated during mechanical ventilation are reflected to the pulmonary vascular bed and mediastinal structures. Positive pressure ventilation decreases venous return, increases PVR, and affects the relationship of pulmonary to systemic blood flow.

Prolonged ventilation is not advised in these patients.

Role of Regional Anesthesia

Regional anesthesia is more effective in inhibiting the stress response associated with surgery than intravenous narcotics.

Caudal epidural: As a "single shot" or via a small caudal catheter. Morphine or hydromorphone provides effective analgesia for 6 to 12 hours, with no significant respiratory depression. Caudal morphine diluted in 0.05 to 0.075 mg/kg delivered in a total volume of 1.25 ml/kg of sterile saline has been used. Relative contraindications to this technique include hemodynamic instability and patients with abnormal clotting profiles with continued active bleeding.

Intercostal nerve block—the surgeon usually performs the block under direct vision from inside the thorax while the chest is open. Bupivacaine 0.25 percent, in doses of 1.5 to 3 mg/kg as per age, can be used. This provides moderate pain relief for 6 to 24 hours.[7]

Larger volumes of local anesthetic (e.g. 5 to 10 ml) should not be used in the intercostal space because of the high-absorption rate and attendant systemic toxicity that can be produced, as well as the possibility of pushing the drug centrally and producing a paravertebral sympathetic or epidural block with central sympatholysis and severe hypotension. The intraoperative placement of catheters in intercostal grooves allows for a continuous postoperative intercostal nerve block. The technique reduces pain and improves pulmonary function. Although, bupivacaine has been used in most reports, use of lidocaine has also been described.

Complications of Shunt Surgery[3]

- A systemic-to-pulmonary artery shunt presents a volume load to the heart
- Low diastolic blood pressure has potential for coronary steal
- Conduit/PTFE is a stimulus for the coagulation system and a potential nidus for infection
- Sudden death among infants who had undergone shunting, especially in children with a single ventricle and in those with double ventricle anatomy. Cause being predominanly shunt thrombosis
- Surgical complications seen after shunt are:
  - Hematoma, pseudoaneurysm formation
  - Shunt leakage
  - Seroma is a rare complication seen after BT shunt.[17]

Occluded Modified Blalock-Taussig Shunt

Progressive stenosis and acute thrombosis, months to years after surgical creation, are the most known complications of the modified Blalock-Taussig shunt. The incidence of thrombotic occlusion of Blalock-Taussig shunts ranges from 1 to 17 percent.[18]

Shunt failure presents in two different clinical settings:

- Those occurring over a period of time result in gradual worsening of cyanosis, polycythemia, effort intolerance and progress to total occlusion
- The other presentation is an acute shunt occlusion in patients with shunt-dependent pulmonary blood flow, which can result in sudden onset life-threatening hypoxia, acute respiratory distress, acidosis, hypotension and even death.

Clinical diagnosis of acute shunt obstruction with sudden onset cyanosis and absent shunt murmur should prompt immediate action.

In suspected patients, echocardiography is a noninvasive reliable diagnostic tool. A second shunt may be required, if the pulmonary artery size, weight or age of the patient are suboptimal for the definitive surgery.

Treatment includes:

1. Thrombolytic therapy with tissue plasminogen activator (TPA) or streptokinase,
2. Balloon angioplasty,
3. Stent implantation and
4. Surgery is the other therapeutic options.

Anesthetic Implications

Management principles remain the same as discussed earlier except:

1. Radial pulse may be absent on the side of previous classical BT shunt.
2. Femoral arterial access may be preferred in view of shunt revision on same or opposite side.
3. Thrombolytic therapy mandates caution during the placement of invasive monitoring lines.
4. In case of midline approach and depending on hemodynamic instability which is common in these patients, postoperative ventilatory support may be needed.

REFERENCES

1. Saxena A. Congenital heart diseases in India-a status report. Indian J Pediatr 2005;72(7): 595-8.
2. Kapoor R, Gupta S. Prevalence of congenital heart disease in Kanpur, India. Indian Pediatr 2008; 45(4):309-11.
3. Pigula FA, Khalil PN, Mayer JE, et al. Repair of tetralogy of Fallot in neonates and young infants. Circulation 1999;100(19):157-61.
4. Hirsch JC, Mosca RS, Bove EL. Complete Repair of Tetralogy of Fallot in the Neonate - Results in the Modern Era. Ann Surg 2000;232(4): 508-14.
5. Leonard H, Derrick G, O'Sullivan J, Wren C. Natural and unnatural history of pulmonary atresia. Heart 2000;84(5):499-503.
6. Lorentz MN, Filho BG. Anesthesia for repair of tetralogy of Fallot in an adult patient. Case report. Rev. Bras. Anestesiol 2007;57(5):525-32.
7. Schmitz ML, Ullah S. Anesthesia for right-sided obstructive lesions. In: Andropoulos DB, Russell IA, Stayer SA (Eds). Anesthesia for Congenital Heart Disease. United States of America: Futura Blackwell Publishing 2005:328-44.
8. Lell WA, Pearce FB. Tetralogy of Fallot. In: Lake CL, Booker PD (Eds). Pediatric cardiac anesthesia 3rd edn. Stamford: Appleton and Lange 1998:303-6.
9. Davies LK, Knauf DG. Anesthetic management for patients with congenital heart disease. In: Hensley FA, Martin DE (Eds). A practical approach to cardiac anesthesia. 4th edn. Philadelphia: Wolters Kluver, Lippincott Williams and Wilkins 2009.
10. Lake CL. Anesthesia for patients with congenital heart disease. In: Kaplan JA, Reich D (Eds). Cardiac anesthesia 4th edn. Philadelphia: WB Saunders, 1999: 785-820.
11. Skubas N, Lichtman AD, Sharma A, Thomas SJ. Anesthesia for Cardiac Surgery. In: Barash PG. Cullen BF, Stoelting RK (Eds). Clinical Anesthesia, 5th edn. United States of America: Lippincott Williams and Wilkins 2006;888-933.
12. Greeley WJ, Steven JM, Nicolson SC. Anesthesia for Pediatric Cardiac Surgery. In: Miller RD, Anesthesia 5th edn. Philadelphia: Churchill Livingstone 2000:1814-5.
13. Castañeda A R. Cardiac surgery of the neonate and infant Philadelphia: WB Saunders 1994;219-20.
14. Cote CJ. Pediatric Anesthesia. In: Miller RD, Eriksson LI, Fleisher Lee A, (Eds) Miller's Anaesthesia. (7th edn). United States of America: Churchill Livingstone Elsevier 2010:2559-96.
15. Motoyama EK , Gronert BJ, Fine GF. Induction of Anesthesia and Maintenance of the Airway in Infants and Children. In: Motoyama EK, Davis PJ (Edn). Smith's Anesthesia for Infants and Children, (7th ed). Philadelphia: Mosby Elsevier 2008;319-53.
16. Cyanotic heart diseases. In: Hines RL, Marschall KE (Eds). Stoelting's Anesthesia and Co-Existing Disease. (5th edn). Philadelphia: Churchill Livingstone 2008.
17. Udyavar A, Dalvi B, Nand Kumar, D'souza R, Rajani R. Seroma Following Modified Blalock-Taussig Shunt resulting in subsequent occlusion of the shunt, which was opened by Balloon Angioplasty. Indian Heart J 2005;57:68-70.
18. Cemsit K, Gulendam K. Recanalization of occluded modified Blalock-Taussig shunt with balloon angioplasty and intraarterial streptokinase. The Anatolian Journal of Cardiology, 2008.

8

Anesthesia for Emergency Vascular Surgery

Kanchan R Rupwate, Kamlesh G Gotiwale, Manju N Gandhi

KEY POINTS

- Perioperative management of patients undergoing emergency vascular surgery is associated with a high incidence of morbidity and mortality due to associated pre-existing systemic diseases and advanced age of most of the patients.
- In patient undergoing emergency vascular surgery, preoperative evaluation should be fast and minimal with accurate assessment of the associated dysfunction of various systems.
- Early stabilization with large bore intravenous access is essential and adequate blood and blood products should be kept ready.
- The goal is to maintain the patients on their usual cardiovascular medication with normal hemodynamic throughout the perioperative period.
- Monitoring should include invasive blood pressure, central venous pressure and pulmonary arterial pressure monitoring along with standard monitoring.
- General anesthesia is most likely administered for thoracic and abdominal surgeries, whereas regional anesthesia can also be given for peripheral vascular surgeries.
- Maintenance of stable hemodynamic during perioperative period is more important than the choice of anesthetic agents and technique.
- Surgery on aortic aneurysm includes simple aortic cross-clamping, left heart bypass technique and deep hypothermic circulatory arrest (DHCA) with their associated pathophysiological changes.
- Spinal cord ischemia and renal ischemia should be taken care of by spinal cord protection and renal protection strategies.
- Endovascular aortic repair is used as it is less invasive and is suitable for patients who are poor surgical risk candidates for surgery.
- Vigilant monitoring and attention to detail in the postoperative management of these patients is essential for successful outcome.
- Anesthesia for the acutely ischemic lower limb is challenging because of inadequate time to evaluate and optimize the comorbidities.
- Cardiac injuries that result in bleeding, tamponade, myocardial ischemia, or structural intracardiac injury require immediate surgical intervention.
- Pulmonary embolism is difficult to diagnose, hence it is crucial to know clinical findings.
- Surgical management of pulmonary embolism includes emergency transvenous pulmonary embolectomy and open pulmonary embolectomy.

INTRODUCTION

Diseases of the aorta have been known since the time of Galen, who wrote "when the arteries are enlarged, the disease is called an aneurysm". In 1761, Morgagni reported the first description of both the clinical and the pathologic findings of aortic dissection. By 1948, surgical techniques had advanced to allow resection and end- to-end anastomosis of a coarctation of aorta. In 1953, DeBakey and Cooley described the successful resection of a thoracic aortic aneurysm and replacement with a homograft. The current surgical technique of leaving the aneurysmal sac intact and restoring flow with an indwelling permanent graft has been popularized by the group at Baylor University and at the Texas Heart Institute.[1]

Aneurysms pose an ever-present threat to life because of their unpredictable tendency to rupture or embolization. Small aneurysms of less than 5 cm rarely

rupture. Current guidelines are to offer operative intervention when the aneurysm exceeds 5.5 cm.

The provision of anesthesia for surgery on the aorta and its major branches is challenging in that it is associated with high morbidity and mortality. Perioperative management of patients undergoing emergency vascular surgery is demanding because of the frequent occurrence of coexisting diseases in elderly patients, the hemodynamic, metabolic stress associated with arterial cross clamping and unclamping and the ischemic insult to vital organs including the brain, heart, kidney and the spinal cord.[2]

PATHOPHYSIOLOGY OF AORTIC OCCLUSION AND REPERFUSION

Aortic cross-clamping and unclamping is associated with complex cardiovascular, renal, humoral and hemodynamic changes. The pathophysiology depends on many factors:

- Level of aortic cross clamp
- Duration of cross clamp
- Left ventricular function
- Degree of periaortic collateralization
- Blood volume distribution
- Body temperature
- Sympathetic nervous system activation
- Anesthetic agents and techniques.

AORTIC CROSS-CLAMPING

Aortic cross-clamping can be infrarenal (most abdominal aortic surgery) or supra-renal and supraceliac (thoraco-abdominal aorta, juxtarenal aneurysms, aortoiliac occlusive diseases with proximal extension).With supra-renal aortic cross-clamping, there is significant impact on cardiovascular system and other vital organs due to organ hypoperfusion and ischemia. The hemodynamic changes associated with cross-clamping are as follows:

- Increased arterial blood pressure above the clamp
- Decreased arterial blood pressure below the clamp
- Increased left ventricular wall tension
- Increased central venous pressure
- Increased pulmonary occlusive pressure
- Increased coronary blood flow
- Increase or decrease in cardiac output.

Changes in cardiac output and filling pressures with aortic cross-clamping are not consistent. The splanchnic circulation, an important source of blood volume reserve plays a vital role in blood volume distribution by altering splanchnic vascular tone and hence the changes in filling pressure of the heart.

The following metabolic changes are seen:

- Decrease in total body oxygen consumption
- Decreased total body CO_2 production
- Metabolic acidosis
- Increased epinephrine and norepinephrine levels.

AORTIC UNCLAMPING

The primary hemodynamic response to aortic cross clamp removal is hypotension along with metabolic acidosis, hyperkalemia and hypocalcemia.

The causes of hypotension are:

- Central hypovolemia due to blood volume redistribution to the lower extremities
- Hypoxia-mediated vasodilatation
- Release of vasoactive and myocardial-depressant metabolites from ischemic tissues.

The hemodynamic response depends on**:**

- Total clamp time
- Intravascular volume
- Level of aortic occlusion
- Use of diverting support.

Reactive hyperemia in tissues and organs distal to the clamp and relative hypovolemia are the main mechanisms of hypotension. Preoperative fluid deficit correction with appropriate fluids and vasoactive drug support can be accomplished before unclamping. Replacement of blood loss and gradual release of the aortic clamp are important measures in maintaining hemodynamic stability during unclamping. At the same time, hypertension should be avoided to prevent bleeding from or damage to the vascular anastomosis.

The major categories of emergency vascular surgical procedures are discussed separately:

1. Emergency abdominal aortic surgery for abdominal aorta rupture, impending abdominal aorta rupture, leaking abdominal aorta.
2. Emergency thoracoabdominal aorta repair for dissecting aneurysms.
3. Emergency endovascular aortic surgery.
4. Emergency lower extremity vascular surgery.
5. Cardiac and great vessels injury which includes blunt and penetrating cardiac trauma.
6. Emergency pulmonary embolectomy.

PREOPERATIVE EVALUATION

Patients undergoing emergency vascular surgery have a high incidence of coexisting disease including diabetes mellitus, hypertension, renal impairment, and pulmonary disease all of which should be assessed and optimized before surgery if possible.

Coronary artery disease (CAD) is the leading cause of perioperative mortality at the time of vascular surgery and long-term survival after emergency vascular procedure is significantly limited by the occurrence of morbid cardiac events.[3]

Cardiac Risk Assessment

The preoperative cardiac assessment presents an opportunity to initiate and optimize pharmacological management, to perform appropriate diagnostic and therapeutic interventions, and adjustment of overall care to decrease not only perioperative risk but also long-term risk from cardiovascular events.

After assessment of cardiac risk there is the additional challenge of modifying preoperative management to reduce the risk by adjusting or adding cardiac medications (e.g. β-blockers), direct coronary intervention (e.g. coronary artery bypass graft), modifying or intensifying perioperative management (e.g. invasive hemodynamic monitoring) or changing preoperative plans (e.g. performing endovascular aortic repair rather than open aortic surgery). [2]

Assessment of Pulmonary Function

Postoperative pulmonary complications are serious in patients undergoing emergency vascular surgery with the most significant morbidity seen in patients undergoing open aortic procedures.

The most important pulmonary complications are atelectasis, pneumonia, respiratory failure, and exacerbation of underlying chronic disease.

Preoperative blood gas determination can be used to establish a baseline values for postoperative comparison. Baseline hypercapnia $PaCO_2$ > 45 mm of Hg indicates a higher risk for postoperative morbidity. Bronchodilator therapy is continued, although the risk of β-agonist induced arrhythmia or myocardial ischemia must also be considered. Preoperative treatment with a short course of glucocorticoids may be helpful for patients with chronic obstructive pulmonary disease (COPD). Presence of pulmonary infection requires treatment with antibiotics.

Renal Function

Preoperative serum creatinine level higher than 2 mg/dl is an independent risk factor for cardiac complications after major noncardiac surgery.[4] Creatinine clearance less than 60 ml/min is an independent predictor of both short- and long-term mortality after emergency vascular surgery. Preoperative beta blocker use and statin use are associated with a lower risk of death in emergency vascular surgery patients with renal impairment.[5,6]

Renal ischemia occurs with interruption of renal blood flow from aortic cross-clamping. Even with infrarenal cross-clamp there can be significant reduction in renal blood flow. Fluctuation in intravascular volume and cardiac output can compromise renal perfusion during intraoperative and postoperative period.

Diabetes Mellitus

Diabetes mellitus is relatively common in vasculopathy. Patients with diabetes are more likely to suffer from autonomic neuropathy and may not tolerate the vasodilatation associated with induction of anesthesia or fall in venous return on commencement of positive-pressure ventilation.

β-blocker Therapy

Patients maintained on chronic β-blocker therapy should continue taking β-blocker throughout the perioperative period.

β-blocker should not be used as the initial or primary treatment of tachycardia caused by perioperative events such as hypovolemia, anemia, pain, or infection because these conditions require prompt treatment of the underlying cause. Acute initiation of high-dose of β-blocker treatment in the perioperative period in an effort to reduce cardiac risk should be avoided.[2]

EMERGENCY ABDOMINAL AORTA REPAIR (AAA)

Emergency abdominal aorta repair is required in patients with abdominal aorta rupture, impending abdominal aorta rupture or leaking abdominal aorta.

All emergency operative procedures on the abdominal aorta and its major branches require large incision and extensive dissection, clamping and unclamping of the aorta or its major branches, varying duration of organ ischemia- reperfusion injury significant fluid shifts and temperature fluctuations, and activation of neurohumoral and inflammatory responses are common during AAA.[2]

Diagnosis

History and Physical Examination

Abdominal or back pain, pulsatile abdominal mass and hypotension in a known case of abdominal aortic aneurysm is assumed to be due to ruptured aneurysm until proven otherwise.

CT scan: Only performed in hemodynamically stable patients.

Ultrasonography (USG)

USG is useful for emergent evaluation of patients.

Preoperative evaluation should be minimal. Decision of urgent operation is essential to minimize morbidity and mortality. Elderly patients with abdominal pain, an expanding pulsatile abdominal mass and in shock require immediate laparotomy as aortic cross-clamping is a life-saving maneuvre.

Preoperative Investigations

CBC, platelet count, serum electrolytes, BUN, serum creatinine, coagulation profile, arterial blood gas analysis, CXR, bedside spirometry and ECG. Preoperative laboratory tests should be correlated with positive findings on history and physical examination.[7]

Anesthetic Management

Two wide-bore intravenous cannulae should be inserted. The patient is induced with a rapid-sequence induction, taking due care for aspiration prophylaxis. Heparinization is not required.[8] When hemodynamic stability is obtained, arterial and central venous catheters, Ryle's tube can be inserted. Temperature probe and urinary catheter can be passed for temperature and urine output monitoring.

Aggressive preoperative fluid resuscitation is contraindicated as it increases bleeding and dilutes the clotting factors.

Anesthetic Drugs and Techniques

Anesthetic technique includes general anesthesia (GA). Induction of GA should proceed in a controlled fashion such that stable hemodynamic is maintained during loss of consciousness, laryngoscopy, intubation and the immediate postinduction period. A variety of intravenous anesthetics (thiopental, etomidate, propofol) are suitable. The addition of short acting opioids such as fentanyl (3 to 5 µg/kg) usually provides stable hemodynamic during and after induction.

Volatile anesthetics may be administered in low concentration before intubation during assisted ventilation as an adjuvant to blunt the hyperdynamic response to laryngoscopy and endotracheal intubation.

Esmolol (10 – 25 mg), sodium nitroprusside (5 – 25 µg), nitro glycerine (50 – 100 µg), and phenylephrine (50 – 100 µg) should be available for bolus administration during induction if needed to maintain stable hemodynamic.

After the aorta is cross-clamped, aggressive fluid resuscitation can be instituted with blood and colloid solutions. Fresh frozen plasma and platelets are kept ready to treat dilutional coagulopathy.

Maintenance of anesthesia is accomplished with a combination of potent opioid (fentanyl or sufentanil) and an inhaled anesthetic (sevoflurane, desflurane, or isoflurane) along with nitrous oxide for balanced anesthesia.

Intraoperative Hemodynamic Monitoring

All patients undergoing major emergency vascular surgery should be monitored with an intra-arterial catheter. It allows beat to beat monitoring, accurate determination of diastolic pressure and sampling of arterial blood for diagnostic purposes. The radial artery is most commonly selected for cannulation because of its superficial location and the presence of collateral circulation.

A noninvasive cuff should be placed on the arm contralateral to the arterial catheter. Central venous catheter should be used for all open aortic procedures. It allows monitoring of central venous pressure and administration of drug directly into the central circulation. Pulmonary Artery catheter should be considered for patients with significant left ventricular dysfunction (EF<60%), history of congestive cardiac failure (CCF), significant renal impairment (preoperative serum creatinine >2 mg/dl) or cor-pulmonale. Two dimensional transesophageal echocardiography (TEE) has been used intraoperatively to assess global ventricular function, guide fluid therapy and monitor myocardial ischemia if facilities are available.[2]

Emergence from Anesthesia

Hemodynamic, metabolic and temperature hemostasis must be normalized before the skin closure and any residual neuromuscular blockade is reversed. Hypertension, tachycardia are aggressively controlled during emergence by the use of short acting agents such as nitroglycerine or sodium nitroprusside. Patients are placed in recumbent position, nitrous oxide is discontinued, 100 percent oxygen is given and trachea is extubated provided spontaneous ventilation is adequate.

Postoperative Management

1. Extubation of trachea is not attempted and patients are shifted to ICU with controlled ventilation if hemodynamic, metabolic, and temperature homeostasis is not achieved, or in patients whom supraceliac aortic cross-clamp time is longer than 30 minutes, and patients with preoperative poor baseline function.[2]

2. Close monitoring of renal function is required as renal failure occurs commonly in this patient group in the postoperative period.
3. Recovery will depend on the success of the surgical repair, and the presence or absence of complications such as myocardial infarction, renal failure and respiratory failure.

ANESTHESIA FOR EMERGENCY THORACO-ABDOMINAL AORTA (TAA) REPAIR

Introduction

Surgical repair is required for aneurysms, acute dissection, penetrating aortic ulcer, coarctation, and traumatic aortic repair.

Open repair of the thoracoabdominal aorta is widely regarded as the most challenging surgical procedure in terms of overall anesthetic and perioperative management.

The anesthesiologist must be knowledgeable and expert in practicing following techniques:

- One lung ventilation
- Extracorporeal circulatory support including circulatory arrest
- Renal and spinal cord protection
- Induced hypothermia
- Invasive hemodynamic monitoring including TEE
- Massive blood transfusion and management of coagulopathy.

Aortic dissection with or without aneurysm formation is classified by DeBakey and colleagues as follows:

Type 1: Aneurysm begins in the ascending aorta and extends throughout the entire aorta.

Type 2: Aneurysm is confined to the ascending aorta.

Type 3: Aneurysms begin just distal to the left subclavian artery and extend either to the diaphragm (type 3 a) or to the aorto-iliac bifurcation (type 3 b).

Acute aortic dissection (less than 2 weeks duration) involving the ascending aorta, i.e. DeBakey type 1 and 2 is a surgical emergency that requires immediate cardiac surgical repair.[2] Preoperative evaluation, preparation and monitoring is same as discussed previously. Pulmonary function must be carefully evaluated as one lung ventilation is required in surgical repair of type I, II and III aneurysms. As large aneurysm can distort the left main bronchus the CXR and CT scan may give vital information about proper placement of double lumen tube (DLT).

Blood loss during TAA repair can be profound and need for massive transfusion must not be underestimated. There should be adequate blood and blood products ready before proceeding for surgery.

Large bore intravenous access is obviously important especially if partial (in contrast to full bypass) cardiopulmonary bypass is to be instituted. Central venous cannulation is done for rapid administration of fluids and drugs.

Radial artery cannulation is used for aneurysm involving the proximal descending thoracic aorta and for distal aortic perfusion techniques, arterial blood pressure distal to the cross-clamps are monitored via right femoral artery or distal aorta. This will help to monitor perfusion pressure to the kidneys, spinal cord and mesenteric circulation during the time when the cross-clamp is high on the descending aorta.

TEE is routinely used during TAA for assessment of LVEDV, evidence of myocardial ischemia and assessment of vascular functions.

A double lumen tube used for single lung ventilation provides optimal visualization and reduces retraction of lung during surgery. Left sided endobronchial tube is preferred. The double lumen tube is changed to single lumen at the end of surgery. It facilitates ICU management of pulmonary hygiene and reduces resistance to breathing during weaning from ventilator in the postoperative period.

Many centers use electrophysiological monitoring with somatosensory evoked potentials (SSEPs) or motor evoked potentials (MEPs) to monitor spinal cord ischemia, as paraplegia is a devastating complication of aortic surgery. A reduction in MEP amplitude to less than 25 percent of baseline is considered an indication of spinal cord ischemia and requires corrective measures.[2] Various strategies are used to prevent spinal cord damage but in general, prevention of this disastrous complication is helped by fast surgery and maintenance of best possible cardiac function during intraoperative period. Lower extremity and peripheral nerve ischemia can be avoided with the use of distal aortic perfusion techniques.[9]

ANESTHETIC MANAGEMENT

Simple Aortic Cross-clamping

- Descending thoracic and thoracoabdominal aortic surgery can be performed without extracorporeal support (i.e. left heart bypass or cardiopulmonary bypass)
- Large series of the "clamp-and-sew" techniques are used with favorable outcome because of the simplicity of its surgical technique
- The duration of cross-clamping on the aorta is the single most important determinant of paraplegia and renal failure with the clamp-and-sew technique
- Clamp time less than 20 to 30 minutes are associated with no paraplegia[10]

- When clamp time are between 30 to 60 minutes the incidence of paraplegia increases from 10 to 90 percent as time progresses
- As the clamp time is longer, generally specific adjustments directed against end organ ischemic complications are often used.[11] Such adjustments include epidural cooling for spinal cord protection, regional hypothermia for renal protection and in-line mesenteric shunting to reduce vascular ischemia[12]
- When the simple clamp-and-sew technique is used, application of the aortic cross clamp results in significant proximal hypertension which requires active pharmacologic intervention. Sodium nitroprusside and isoflurane have been used successfully to control the proximal hypertension associated with high aortic cross-clamping.

Left Heart Bypass

- Maintaining lower body perfusion with the use of retrograde distal aortic perfusion reduces ischemic injury and improves outcome, provided that the pressure is high enough to perfuse the organs
- The simplest method of providing distal aortic perfusion is a passive conduit or shunt
- Partial bypass also referred as left heart bypass or left atria to femoral bypass is the most commonly used distal aortic perfusion technique
- This technique allows adjustment of blood flow and usually draws blood from the left atrium and returns blood to the left femoral artery
- During left heart bypass it is essential that arterial blood pressure be monitored above and below the cross-clamp
- Simultaneous display of radial and femoral artery pressure and aim for a mean arterial pressure of 80 to 100 mm of Hg above the cross clamp and at least 60 mm Hg below the cross-clamp is desirable
- Careful control of intravascular volume, bypass pump flow and vasoactive drugs is required to achieve target blood pressure
- With sequential aortic clamping, intercostal arteries, visceral and renal arteries are reimplanted
- Routinely moderate hypothermia (32°C) is used during bypass to protect the vital organs during obligate periods of ischemia.

Deep Hypothermic Circulatory Arrest (DHCA)

- Complex aneurysm involving the aortic arch often require elective cardiopulmonary bypass with an interval of deep hypothermic (15°C) circulatory arrest (DHCA) at this point of time cerebral blood flow is transiently interrupted during surgery
- Bypass can be accomplished by cannulation of the femoral artery and femoral vein (femoro-femoral bypass)
- During the interval of DHCA some centers also use anterograde (i.e. innominate artery) or retrograde (i.e. internal jugular vein) selective cerebral perfusion with cold oxygenated blood to extend the safe maximum duration of circulatory arrest[11]
- Without selective perfusion technique, 45 to 60 minutes is thought to be safe limit of DHCA but 90 minutes have been reported with selective cerebral perfusion.[13]

Anesthetic Management

- Usually balanced anesthesia is provided with a combination of an opioid, a low dose potent volatile anesthetic, a benzodiazepine and a long acting muscle relaxant
- Induction of anesthesia should be in slow and controlled fashion
- During induction hypertension should be avoided because acute stress on the aneurysm can cause rupture of the aneurysm
- The heart rate should be maintained at or below baseline because myocardial ischemia is often related to the heart rate. Throughout in the intraoperative period, the patient should be monitored vigilantly. Extubation should always take place in ICU and only after significant period of hemodynamic and metabolic stability. Postoperative analgesic regimen focuses on pain control and stable hemodynamic.
- Paraplegia is a devastating complication of vascular surgery. Various methods and drugs have been used to prevent ischemic injury to spinal cord
- Distal aortic perfusion with extracorporeal support has been shown to reduce the incidence of paraplegia.[14] CSF drainage is frequently used to improve spinal cord perfusion during TAA repair and is often used in combination with distal aortic perfusion[15,16]
- Hypothermia is probably the most reliable method of neuroprotection from ischemic injury by reducing O_2 requirements by approximately 5 percent for each degree centigrade. A twofold prolongation of tolerated cross-clamp time is achieved by cooling even to mild hypothermia[6]
- Systemic hypothermia can be achieved by full cardiopulmonary bypass or partial bypass[17]
- Regional cooling is beneficial who receive epidural infusions of 4°C saline

- Variety of drugs have been studied to reduce spinal cord ischemic injury like barbiturates, corticosteroids, and calcium channel blocker.

Renal Ischemia and Protection

Renal failure after TAA repair results from pre-existing renal dysfunction, ischemia during cross-clamping, thrombotic or embolic interruption of renal blood flow, hypovolemia and hypotension. Retrograde distal aortic perfusion techniques are widely used to preserve renal function during the cross-clamps period along with systemic and regional hypothermia.

Renoprotective strategies can be tried which include:

- Maintenance of intravascular volume
- Minimizing the aortic cross-clamp time
- Avoiding high sodium load
- Adequate bypass flow and blood pressure for maintenance of renal function
- Mannitol is often given before cross-clamping as it improves renal cortical blood flow and the GFR. Loop diuretics are also tried. Low dose dopamine (3 µgm/kg/min) dilates renal blood vessels and increases renal blood flow and urine output
- Fenoldopam mesylate, a selective dopamine type I agonist that preferentially dilates the renal and splanchnic vascular beds can also be used as renoprotective agent.[18,19]

Spinal Cord Ischemia and Protection

Spinal cord ischemia is a rare but potentially serious complication of aortic vascular surgery. It causes flaccid paraparesis with dissociated sensory loss. The incidence of paraplegia increases in emergency infrarenal abdominal aortic repair surgery.

A multimodality approach for spinal cord protection during thoracic abdominal aneurysm repair is currently used by most institutions (e.g. left heart bypass, CSF drainage, aggressive intercostals artery reconstruction, motor evoked potential monitoring), but in general, prevention of this disastrous complication is helped by fast surgery and maintaining best possible cardiac function.

Coagulation and Metabolic Management

- Coagulopathy is a frequent complication during TAA repair
- A dilutional coagulopathy develops after approximately one blood volume replacement. Coagulation factors get diluted and platelets become deficient. With the early use of FFP and platelets transfusion severe coagulopathy can be avoided
- Prothrombin time, partial thromboplastin time, fibrinogen levels and platelet count should be measured frequently (6 hourly)
- When coagulopathy persists despite these efforts epsilon-amino caproic acid is beneficial
- Antifibrinolytic therapy and desmopressin can be given to increase circulating levels of von Willebrand factor and factror VIII
- Before coming off bypass normothermia should be achieved and ABG with serum electrolytes are maintained within normal limits
- The metabolic acidosis that occurs during and after cross-clamping should be treated with sodium bicarbonate along with the corrective measures of underlying pathology
- Hyperkalemia should be treated aggressively in oliguric and anuric patients with calcium chloride and sodium bicarbonate.

ANESTHESIA FOR EMERGENCY ENDOVASCULAR AORTIC REPAIR

Introduction

Endovascular surgery is one of the most exciting developments in the treatment of vascular disease and is revolutionizing current treatment modalities for aortic disease, including aneurysm, dissection, rupture and traumatic injury. The endovascular approach was developed as a less invasive alternative to open abdominal aortic aneurysm repair and to provide a treatment option for patients considered to be poor surgical candidates. The endovascular stent graft is designed to be deployed within the aorta to span the length of the eneurysm and exclude blood flow into the aneurysm cavity.

Anesthetic Management

Local, regional and general anesthesia techniques have been used for shortly after its introduction.

- Local and regional techniques are most often used with intravenous sedation
- GA is used for endovascular aortic repair in patients with extensive groin dissection or retroperitoneal dissection and those requiring complex repairs, where conversion to open repair is most likely. A balanced anesthesia technique using relatively short acting agents such as fentanyl 2 to 4 µgm/kg can be used. Esmolol, sodium nitroprusside, nitroglycerine and phenylephrine should be available and used to maintain stable hemodynamic

The possibility of acute aortic rupture necessitates the availability of fluids, blood and rapid infusion devices

along with invasive blood pressure monitoring. Two large bore peripheral intravenous catheters and urinary catheter insertion are recommended for these patients.

Monitoring of urine output can help to guide fluid management, particularly when high volumes of heparinized flush solution, radiographic contrast material and diuretic drugs are administered. To reduce the incidence of contrast induced nephropathy isotonic bicarbonate infusion along with appropriate fluids is often used.

Endovascular repairs involving the descending thoracic aorta require additional preparation and monitoring. These procedures are often performed in the operating room under general anesthesia with above mentioned precautions and care. Although current generation devices are much less prone to graft migration during deployment, pharmacological (i.e. sodium nitroprusside or nitroglycerine) induced hypotension (systolic blood pressure <100 mm of Hg) is commonly used during deployment. TEE monitoring is frequently used and can be extremely helpful in identifying proximal and distal, stent-graft landing zones, entry and exit points of dissections, true or false lumen and aneurysm exclusion.

ANESTHESIA FOR EMERGENCY LOWER EXTREMITY REVASCULARIZATION

Introduction

Most patients presenting with lower limb ischemia are elderly and often have extensive comorbidity. Anesthesia for acutely ischemic lower limb is challenging because of inadequate time to evaluate and treat comorbidities and the physiological disturbances resulting from an ischemic limb.

- Upper extremity peripheral arterial disease is less common as compared to lower extremity
- Acute peripheral arterial occlusion occurs primarily as a result of embolism and thrombosis
- The majority of embolic foci to the lower extremity originate in the heart, with intermittent atrial fibrillation and myocardial infarction being the most common causes of embolic events
- In the lower extremity the common site for thromboembolism include the iliac artery bifurcation femoral artery bifurcation, and popliteal artery.[8]

Acute Lower Extremity Ischemia

Clinical Manifestations

Pain, Pallor, Parasthesia, Paralysis, Pulselessness and Poikilothermia of the affected limb are the signs and symptoms of ischemia.

Diagnosis

Pulse examination may indicate the level of obstruction. Motor and sensory examination indicates severity of ischemia and will decide the plan of management.

Treatment

Hemodynamic instability should be treated with aggressive hydration and institution of heparin to prevent propagation of clot.

Urgent operative intervention is warranted in patients with significant sensory and minor motor function deficits if permanent limb damage or amputation is to be avoided. The thrombus is removed with embolectomy by passing catheter proximally and distally.

Preoperative Preparation and Monitoring

- If patient is on long-term cardiac and respiratory medications then they should be continued
- Chronic β-blocker therapy is particularly important because acute withdrawal can be associated with significant morbidity. Antiplatelet therapy with low dose aspirin should be continued
- Monitoring for lower extremity revascularization should include an intra-arterial catheter that permits continuous blood pressure monitoring to optimize coronary artery and lower extremity graft perfusion as well as blood sampling for diagnostic laboratory testing
- Urinary catheterization is indicated for assessing intravascular volume and cardiac output
- Central venous monitoring is considered for patients with significantly impaired ventricular dysfunction or congestive heart failure
- PA catheter is reserved for patients with active congestive heart failure or unstable angina
- Computerized ST segment monitoring is helpful in monitoring for myocardial ischemia in high-risk patients.

Anesthetic Management

- Almost all patients requiring surgery for acute limb ischemia may have received heparin and/or thrombolytic agents immediately before coming to the operation theater and hence, the use of regional anesthesia will be contraindicated
- GA is delivered with the use of a balanced technique consisting of opioids, potent inhaled anesthetics, nitrous oxide and neuromuscular blockade
- Induction of anesthesia should proceed in a controlled fashion such that a stable hemodynamic profile is maintained. Intraoperative complications such as

blood pressure instability, arrhythmias, ischemic changes, and hypoxia can occur which should be treated promptly
- Maintenance of anesthesia may be accomplished with a low dose inhaled anesthetic (i.e. isoflurane, desflurane or sevoflurane) with 50 percent nitrous oxide-oxygen and opioid (3 – 5 µg/kg of fentanyl)
- Judicious use of β-blockers and vasoactive drugs is often necessary[2]
- Anticoagulant and antiplatelet therapy is common in vascular surgery population and often precludes the use of spinal or epidural techniques.[20] If coagulation profile is normal, the epidural anesthesia technique can have advantage in its ability to continue drug delivery into the postoperative period for analgesia
- Infiltration of local anesthetic with sedation is a suitable technique in a cooperative patient.

Postoperative Care in Vascular Surgical Patients

- Pain and anxiety should be carefully controlled in the postoperative period because the stress response and myocardial ischemia are of great concern at this time
- Intravascular volume should be optimized
- Anemia should be avoided (Hb > 9 gm/dl should be maintained)
- Control of heart rate and blood pressure is achieved
- Computerized ST segment analysis is done to identify myocardial ischemic changes
- Peripheral pulses should be checked frequently to verify lower extremity graft patency
- Postoperative analgesia can be provided by intravenous or epidural uploads delivered by patient controlled analgesia (PCA) or epidural uploads with local anesthetic delivered by continuous infusion or PCA.

Postoperative Management of Emergency Vascular Surgical Patients

Vascular surgery patients require special attention during the postoperative period because most of the cardiac complications occur postoperatively and other problems may arise that require immediate attention. Conventional practice is to monitor all vascular surgery patients in an ICU setting after surgery.[2] Myocardial ischemia occurs most frequently in the postoperative period. The determinants of myocardial O_2 supply and demand should be optimized for all patients to prevent ischemia before it develops.β-blocker and statin therapy should be continued throughout the postoperative period.

Coagulopathy due to residual heparin or from dilutional coagulopathy after massive transfusion can occur. Fresh bleeding from anastomotic site can occur in case of uncontrolled hypertension.

Hypovolemia after aortic surgery (due to third space loss, and bleeding) may lead to hypotension and hypoperfusion of the coronary arteries and lower extremity vascular grafts.[2]

Lower extremity pulses should be checked at hourly interval and injectable dextran or heparin can be started to prevent thrombosis.

Residual hypothermia in the early postoperative period is associated with increased incidence of myocardial ischemia and cardiac morbidity; therefore body temperature should be carefully monitored and controlled in all vascular surgery patients post-operatively.

Stress response in the postoperative period should be controlled by paying attention towards pain, hypothermia, hemodynamic extremes, anemia and ventilatory insufficiency. In mechanical ventilated patients, weaning period is especially stressful and myocardial ischemia occurs frequently during this time. Careful sedation and expeditious weaning is desirable.[21]

CARDIAC TRAUMA AND GREAT VESSEL INJURY

Blunt trauma often occurs as a result of massive, sudden forces that can also injure the heart, vena cava, pulmonary vessels, thoracic aorta, and brachiocephalic branches.

Cardiac injuries that result in bleeding, tamponade, myocardial ischemia, or structural intracardiac injury require immediate surgical intervention. It includes blunt cardiac and penetrating cardiac trauma.

Blunt Cardiac Trauma

Motor vehicle crashes causes the majority of blunt cardiac injuries, with other causes include falls, crush or blast injuries, and street violence.

Screening for cardiac injuries begins with a chest radiograph and Electrocardiogram (ECG).

A normal troponin I levels increases the negative predictive value, and an elevated troponin significantly increases the specificity for blunt cardiac injury.

TEE improves resolution and allows for identification of aortic trauma.

The management of patients with blunt cardiac trauma is most often dictated by the presence of structural injury and hemodynamic compromise.

Stable patients with minor ECG changes with blunt or penetrating pericardial wound without cardiac injury, tamponade or herniation require close monitoring but no surgical intervention.

Disruption of right atrium or right ventricle may be tolerated for a brief period allowing patients to survive transport to the operating room.

Traumatic cardiac tamponade always warrants immediate sternotomy.[22] For more complicated cardiac disruptions cardiopulmonary bypass is necessary to facilitate hemodynamic stability and cardiac repair. Traumatic left sided valvular lesions typically require immediate intervention.

Penetrating Cardiac Trauma

Patients with penetrating cardiac injuries who survive transport are often intoxicated or combative and classic signs of cardiac tamponade (Beck's triad of hypotension, distended neck veins and muffled heart sounds) may be difficult to assess. Management of unstable patient with penetrating cardiac trauma begins with fluid resuscitation and immediate transfer to the operating room.

Pericardial decompression may be required to achieve enough stability to allow transfer to the operating room. Emergency thoracotomy can occasionally salvage unstable penetrating cardiac trauma patients by controlling bleeding.

Transthoracic echocardiography and chest CT scan to screen stable patients with suspected injury is of predictive value.[22]

If identified by noninvasive means, penetrating cardiac injury in a hemodynamically stable patient warrants sternotomy and exploration. Anesthesia management in such patients depends on patient's general condition and hemodynamic parameters rather than the choice of anesthetic agent.

ANESTHESIA FOR EMERGENCY PULMONARY EMBOLECTOMY

Introduction

Pulmonary embolism (PE), a consequence of venous thromboembolic disease, is a common medical problem. A spectrum of disease severity exists, ranging from subclinical to massive pulmonary embolism, which is life-threatening. Many clinical risk factors predispose to the development of pulmonary embolism which includes major and minor risk factors.[23]

Major risk factors are:
- Surgery: Major abdominal/pelvic surgery, hip or knee replacement
- Obstetrics: Late pregnancy, cesarean section, puerperium
- Lower limb problems: Fractures, varicose veins
- Malignancy: Abdominal or pelvic, advanced/metastatic
- Reduced mobility
- Miscellaneous: Previous proven venous thrombo-embolism.

Minor risk factors are:
- Cardiovascular diseases
- Patients taking oral contraceptive
- Hormone replacement therapy
- Obesity
- Neurological disability
- COPD
- Thrombotic disorder, etc.

As pulmonary embolism is difficult to diagnose, it is crucial to be especially alert to the possibility of disease.

Pathophysiology

The effect of a pulmonary embolism depends on the extent to which it obstructs the pulmonary circulation, the time over which the obstruction accumulates, and premorbid cardiopulmonary function. In massive PE, the increase in pulmonary vascular resistance (PVR) causes right ventricular failure and circulatory shock. There can be increase in alveolar dead space, ventilation/perfusion mismatch and subsequently the development of localized pulmonary edema, pulmonary infarction and pulmonary hemorrhage can occur.[23]

Clinical Findings

The presentation of massive pulmonary embolism often takes the form of acute circulatory collapse, with hypotension, altered consciousness and elevation of the jugular venous pressure.

Patient may give history of:
- Tachypnea
- Dyspnea
- Palpitation
- Syncope
- Chest pain
- Hemoptysis.

Physical Exam

- Pleural rub
- Wheezing
- Rales
- Fixed or split S2
- Rt Ventricle lift
- Evidence of venous thrombosis.

ECG will show S_1 Q_3 T_3 pattern.

Spiral CT or lung perfusion scan can be obtained to rule out diagnosis of PE.

Table 8.1: Classification of pulmonary thromboembolism[25]

Physiologic method for classification of patients using cardiorespiratory variables has been devised by Greenfield LJ and Stewart JP

Class	*Symptoms*	*PaO_2 (mm Hg)*	*$PaCO_2$ (mm Hg)*	*Hemodynamic*
I	None	Normal	Normal	Normal
II	Anxiety, hyperventilation	< 80	<35	Tachycardia
III	Dyspnea, collapse	<65	<30	CVP elevated, PA > 20 mm of Hg
IV	Shock, dyspnea	<50	<30	CVP elevated, PA > 25 mm of Hg
V	Dyspnea, syncope	<50	30-40	BP<100 mm of Hg PA- 40 mm of Hg CVP elevated, CO low

CVP– Central Venous Pressure, PA – Mean Pulmonary Artery, BP – Blood Pressure, CO – Cardiac Outputs.

Greenfield classified patients suffering from pulmonary embolism into two groups, based on physiologic parameter (Table 8.1).

The first group consists of class I, II and III patients who can be managed medically.

The second group includes class IV and V who have either a massive pulmonary embolus or a pulmonary embolus associated with pulmonary or myocardial disease.[24]

Management

Several methods of treatment for acute PE have been described including heparin therapy, streptokinase therapy and surgery (open pulmonary embolectomy).[26-29] Class IV and V patient's medical therapy has very little to offer because of associated high early mortality. These patients are treated with transvenous pulmonary embolectomy or open pulmonary embolectomy which has a very high mortality of up to 50 percent. Features of management which are specific to patients requiring pulmonary embolectomy are—the choice of methods of circulatory support and the management of anticoagulants.

Emergency Transvenous Pulmonary Embolectomy

A catheter of 100 cm length is inserted through the femoral venotomy to the RA and right ventricle to the pulmonary flow tracts. After angiographic localization of the embolus, suction is applied and embolus is extracted through femoral venotomy along with the catheter.

A Greenfield filter is inserted in the inferior vena cava at the completion of the procedure to protect against further embolization in an already hemodynamically compromised patient.

Complications

- Ventricular arrhythmias
- Inability to extract the embolus
- Perforation of pulmonary artery
- Fatal hemorrhage following reperfusion
- Perforation of lung.

Preoperative Preparation

- Appropriate monitoring should include arterial catheter placement for the second to second observation of blood pressure and to obtain serial ABGs as indicated
- A large bore peripheral or central venous cannulation is indicated for rapid infusion of volume and vasoactive drugs
- PA catheter should also be placed as early as possible both to determine pulmonary artery pressures and aid in evaluation of hemodynamic status
- Tachycardia both supraventricular and ventricular as well as bradycardia often result from pulmonary embolus manipulation and therefore, lead II or transesophageal lead is best selected.

Anesthetic Management

It requires a motionless patient. Hence, muscle paralysis with controlled ventilation can be employed with balanced anesthesia using sedative and narcotic agents. Midazolam is used for its amnesic properties and its cardiovascular stability. Narcotic is used for suppression of pain associated with the procedure. Pulmonary embolectomy is associated with significant blood loss.

There appears to be two periods when large volume infusions may be necessary:

A. The first is at the time the embolus is removed with reperfusion and its associated complications.

B. The second is when the filter is being inserted, due to the surgical incision and bleeding in an anticoagulated patient.

Open Pulmonary Embolectomy

Anesthesia management of patients undergoing pulmonary embolectomy may be challenging as a result of their hemodynamic instability. Patients with massive PE often presents with tachycardia, decreased left ventricular preload, and compromised systolic function. All these factor limits their ability to adjust to the physiological consequences associated with the induction of general anesthesia. Therefore, all patients undergoing surgical pulmonary embolectomy should be prepared and draped before general anesthesia induction and a cardiac surgical team should be present in the operating room ready to initiate CPB.[30]

Inferior vena cava filter is often placed prophylactically before surgery.

Surgery is performed via a midline sternotomy with CPB with or without periods of deep hypothermic circulatory arrest (DHCA). Support of systemic vascular resistance with norepinephrine or phenylephrine are usually required.

Monitoring includes femoral and pulmonary artery catheter, TEE, electroencephalography and rectal or bladder temperature.[31]

Anesthetic Management

These patients are at risk for hemodynamic collapse from right ventricular failure and hypotension during induction of general anesthesia (GA). In these patients, systemic blood pressure becomes predominantly dependant on compensatory increases in SVR and heart rate.[32]

Many anesthetic drugs used to induce GA are associated with varying degree of myocardial depression and direct or indirect systemic vasodilatation.[33, 34]

In healthy patients, cardiac output is maintained after GA induction by an increase in cardiac output that at least partially compensates for systemic hypotension. However, patients with massive PE often present with tachycardia, underlying reduced LV preload and compromised systolic function, which limits their ability to adjust to the physiological consequences associated with the induction of GA.

Preoperative measures should be taken so that patients are not exposed to conditions that increases PVR (e.g. hypoxia, hypercapnia, acidosis, lung hyperinflation, hypothermia).[35]

Induction may be performed with etomidate or ketamine to avoid hypotension, the drugs should be injected in graded doses.

There is no need for lung isolation, so airway management is achieved with standard endotracheal tube. Support of systemic vascular resistance with norepinephrine or phenylephrine is usually required.

If DHCA is used, it is preceded by mannitol, methylprednisolone and sodium thiopentone use to decrease cerebral edema and to achieve cerebral protection.

The speed of warming and cooling is controlled on CPB to maintain a temperature gradient less than 10°C between blood and bladder/rectal temperatures. Periods of DHCA are usually limited to 20 minutes. Massive pulmonary hemorrhage occurs rarely during CPB in these cases.

Management of Pulmonary Hemorrhage

General supportive measures should be taken**.**

The instillation of phenylephrine 10 µg and vasopressin 20 units diluted in 10 ml saline via the ET tube may be beneficial.

Postoperative Management

The patients are kept sedated, intubated and ventilated for at least 24 hours to decrease the risk of reperfusion pulmonary edema.

Norepinephrine or vasopressin infusion may be used to elevate the SVR and this may decrease the cardiac output.

CONCLUSION

Anesthesia for emergency vascular surgery continues to challenge the anesthesiologist due to involvement of relatively elderly population with a high incidence of coexisting diseases. Ruptured or leaking aneurysms are fatal if untreated and emergency repair is the patient's only chance of survival. Appropriate patient selection is imperative and only suitably experienced surgeons and anesthesiologist should be involved in the care of these patients. The mortality among patients who undergo emergency surgery on abdominal aortic aneurysm is 60 to 80 percent[36].

Various surgical bypasses are used in an attempt to revascularize lower limbs which are ischemic due to occlusive vascular disease. Outcome depends on the successful bypassing of the obstructions and the severity of the arterial disease distally. The patient's suffering should be minimized by providing adequate analgesia and nursing care.

REFERENCES

1. Schiff JA, Thoracic aortic diseases. In: Joseph Simpson (Ed). Anaesthesia and the patient with coexisting heart disease, 1st edn. NY 1993;11:241-2.
2. Edward J Norris. Anaesthesia for vascular surgery. In: Miller RD, Lee AF, Roger AJ, John JS, Jeanine WK, William LY, (Eds). Miller's Anaesthesia, 7th, 62: 1985-2043.
3. Mangano D T. Perioperative cardiac morbidity. Anaesthesiology 1990;72:153-84.
4. Lee TH, Mareantonio ER, Mangione CM. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation 1999; 100:1043-9.
5. Welten GM, Chonchol M, Hoeks, SE. Statin therapy is associated with improved outcomes in vascular surgery patient with renal impairment. Am Heart J 2007;154:954-61.
6. Barbato JE, Tzeng E. Vascular Emergencies. In: Peitzmam AB, Rhodes M, Schwab CW, Yealy DM, Fabian TC (Eds). Trauma manual: Trauma and Acute care surgery. 3rd edn. 59:626-35.
7. Ellis JE, Roizen MF, Mantha S. Anaesthesia for vascular surgery. Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anaesthesia, 5th edn. Philadelphia. Williams and Wilkins 2006;32:954-60.
8. Phillip J Cowlishaw, Richard J Telford. Anaesthesia and Intensive care medicine 2007;8;6:248-51.
9. MaConachie, Sebastin D. Vascular Surgery. In: MaConachie Edn. Anaesthesia for high risk patients. 2nd edn. 2009;17:263-75.
10. Katz NM, Blackstone EH, Kirklin J lo. Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transaction. J Thorax cardiovascular surge 1981;81:669-74.
11. Teferag, Acher CW, Wynm MM. Clamp and sew techniques in thoracoabdominal aortic surgery using naloxone and CSF drainage. Semin nase surg 2000;13:325-30.
12. Cambria RP. Thoracoabdominal aortic aneurysm repair: How I do it. Cardiovascular surge 1999;7:597-606.
13. Takamoto S, Matsuda T, Aardam. Simple hypothermic retrograde cerebral perfusion during aortic arch replacement: A Preliminary case report. J Thorax cardiovascular surge 1992;104:1106-9.
14. Coselli JS, Lemaitre SA. Left heart bypass reduces paraplegia rates after thoracoabdominal aortic aneurysm repair: Ann Thorac surg 1999;67:1931-4.
15. Estrera AL, Miller CC 3rd, Huynh TT, Porat EE, Safi HJ. Neurologic outcome after thoracic and thoracoabdominal aortic aneurysm repair. Ann Thoracic surg 2001;72:1225-30, discussion 1230-1.
16. Safi HJ, Miller CC 3rd, Huynk TT. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aorta repair: Ten years of organ protection. Ann Surg 2003;238:372-80.
17. Frank SM, Parker SD, Rrek P. Moderate hypothermia with partial bypass and segmental sequential repair for thoraco-abdominal aortic aneurysm. J Vasc Surg 1994;19: 687-97.
18. Gilbert TB, Hasnain JU, Flinn WR. Fenoldopam infusion associated with preserving renal function after aortic cross clamping for aortic aneurysm repair. J. Cardiovascular Pharmacology Ther 2001;6:336.
19. Halpenny M, Rushl C, Breen P. The effects of fenoldopam on renal function in patients undergoing electric aortic Surgery. Euro J Anaesthesiology 2002;19:32-9.
20. Horlocker TT, Heit IA. Low Molecular weight heparin: Bioermistry, pharmacology, perioperative phophylous regimens, guidelines for regional anaesthetic management. Anaesthesia Analg 1997;85:874-85.
21. Abalos A, Leibowitz AB, Distefano D. Myocardial Ischemia during the weaning period. Am J Criiticare 1992;1:32-6.
22. Cook CC, Gleason TG. Great vessel and cardiac trauma. In: Kerri j, Kron I. (Eds). Advances in cardiac surgery and aortic surgery: Surg Cli North Am 2009;89(4):797-815.
23. David G Sinclair. Anaesthesia and intensive care medicine. 2007;8:12:527-9.
24. Greenfield LJ. Pulmonary embolism, diagnosis and management. Curr Probl Surg 1976:13-52.
25. Stewart JP, Greenfield LJ. Transvenous vena canal filtration and pulmonary embolectomy. Surgical clinics of North America 1982:62:411-30.
26. Sharp EH. Pulmonary embolectomy successful removal of a massive pulmonary embolus with the support of CPB–case report. Ann surg 1962;156:1-4.
27. Glassford DM, Alford WC, Burrus GR, Stoney WS, Thomas CS. Pulmonary embolectomy. Ann Thorax surg 1981;32: 28-32.
28. Dalen JE, Alpert JS. Natural history of pulmonary embolism probe. Cardiovascular Dis 1975;17:259-70.
29. Mcdonald I G, Hirsh J, Hale G S. Early nature of resolution of major pulmonary embolism: A study of Angiographic and hemodynamic changes occurring during first 24 to 48 hrs. Br. Heart J 1971;33:432-7.
30. Rosenberger P, Shernan SK, Shekar PS, Tuli JK, Weissmuller T. Acute Hemodynamic Collapse after Induction of General Anaesthesia for Emergent Pulmonary Embolectomy. Anesth Analg 2006;102:1311-15.
31. Manecke G. Anaesthesia for pulmonary endarterectomy. Semin Thorac cardiovascular surg 2006;18:236-42.
32. Salter P. Pulmonary embolectomy with the aid of extracorporeal circulation. Thorac Cardiovascular Surg 1982;30:31-5.
33. Patschke D, Eberlein HJ, Hess W. Effect of high dosage of morphine and meperidine on hemodynamic coronary blood flow and myocardial O_2 consumption in comparison to fentanyl and piritramide. Anaesthesist 1977;26:239-48.
34. Chang KS, feng MG, Danis RF. Midazolam produces vasodilatation by mixed endothelium dependent and Independent mechanisms. Anaesth Analg 1994;78:710-7.
35. Domino KB, Westein L, Glasser S A. Influence of mixed venous oxygen tension PVO_2 on blood flow to atelectatic lung. Anaesthesiology 1983;59:428.
36. Hopkinson JM, Mortimer AJ. Abdominal aortic reconstruction-Emergency. In: Goldstone JC, Pollard BJ Eds. Handbook of clinical anaesthesia: 1st edn.1996;22:444-50.

9 Anesthesia for Cardioversion

Aparna A Nerurkar, Manish B Kotwani

KEY POINTS

- Cardioversion refers to a synchronized or a precisely timed delivery of electricity to convert organized (although abnormal) cardiac rhythm to normal rhythm.
- Synchronization in the early part of the QRS complex avoids energy delivery near the apex of the T wave in the surface ECG, which coincides with a vulnerable period for induction of ventricular fibrillation.
- As per advanced cardiac life support (ACLS) guidelines, any patient with narrow or wide QRS complex tachycardia (rate >150) who is hemodynamically unstable should be immediately treated with synchronized electrical cardioversion.
- Synchronized cardioversion is not used for treatment of VF, pulseless VT, or unstable polymorphic (irregular) VT. These rhythms require delivery of high-energy unsynchronized shocks (i.e. defibrillation doses).
- Since the skin can conduct away a significant portion of the current, conductive gel or pre-gelled pads are commonly used to ensure good contact. Under ideal circumstances, only 10 to 30 percent of the total current reaches the heart.
- Pacemakers and ICDs should be atleast 10 cm from direct contact with paddles and should eventually be interrogated for any malfunction after cardioversion.
- Many patients with atrial fibrillation requiring urgent or emergency cardioversion will classify as American Society of Anesthesiologists (ASA) III; that is a patient with severe systemic disease that is not incapacitating.
- Digitalis therapy predisposes to post-cardioversion arrhythmias; it should be withheld for 48 hours before elective cardioversion. If urgent cardioversion is required, the initial DC dose should be low.
- Cardioversion should be carried out only in areas specifically designed for the purpose and with a full range of drugs, resuscitation and monitoring equipment available.
- Adequate depth of sedation/anesthesia is important firstly to prevent recall of an unpleasant experience and attenuate the stress response; and secondly, to not precipitate hypoventilation, airway obstruction, or cardiac embarrassment.
- Rapid onset and offset, cardiovascular stability, no respiratory depression, and some analgesic component would be the ideal characteristics of the agents used.
- The duration of atrial fibrillation is inversely related to the probability of successful cardioversion.
- Pharmacologic cardioversion is used mainly for atrial fibrillation and flutter of relatively short duration. Dofetilide, flecainide, ibutilide, propafenone, amiodarone, and quinidine have been demonstrated to be useful in restoring sinus rhythm.
- Anticoagulation with warfarin to an international normalized ratio (INR) goal of 2.5 (range 2.0 to 3.0) is recommended for 4 weeks after cardioversion. Anticoagulation is not needed for atrial fibrillation of less than 48 hours duration.

Cardioversion refers to a synchronized or a precisely timed delivery of electricity to convert organized (although abnormal) cardiac rhythm to normal rhythm. It is important to distinguish this from defibrillation which refers to a high-energy electrical shock used to treat ventricular fibrillation or pulseless ventricular tachycardia.

The term cardioversion thus, does not apply to ventricular defibrillation or to the pharmacologic reversion of arrhythmias.[1]

Electrical countershocks can be delivered with either alternating current (AC) or direct current (DC) energy. Alternating current defibrillation can cause significant myocardial damage due to the greater energy flux and duration.[2] Direct current (DC) cardioversion uses a brief and calibrated discharge of electricity across the heart. The discharge depolarizes the entire heart, eliminating an abnormal rhythm and permitting the sinoatrial node to resume control.[3]

Delivery of DC shocks to the heart has long been used successfully to convert abnormal heart rhythms back to normal sinus rhythm. A variety of clinical scenarios are now encountered in which transthoracic and, more recently, intracardiac DC shock of variable energies is delivered.[4]

BASIC PRINCIPLES

For cardioversion the DC electrical discharge is synchronized with the R or S wave of the QRS complex. Synchronization in the early part of the QRS complex avoids energy delivery near the apex of the T wave in the surface ECG, which coincides with a vulnerable period for induction of ventricular fibrillation.[5,6] The peak of the T wave represents the terminal portion of the refractory state when adjacent heart fibers are in differing states of repolarization. Defibrillation refers to an unsynchronized discharge of energy and is only recommended for ventricular fibrillation (VF) or pulseless ventricular tachycardia.[1]

The energy (shock dose) used for a synchronized shock is lower than that used for unsynchronized shocks (defibrillation). These low-energy shocks should always be delivered as synchronized shocks because if they are delivered as unsynchronized shocks they are likely to induce VF. If cardioversion is needed and it is impossible to synchronize a shock (e.g. the patient's rhythm is irregular), use high-energy unsynchronized shocks.[3,6]

Transient delivery of electrical current causes a momentary depolarization of most cardiac cells allowing the sinus node to resume normal pacemaker activity. In the presence of re-entrant-induced arrhythmia, such as paroxysmal supraventricular tachycardia (PSVT) and ventricular tachycardia (VT), electrical cardioversion interrupts the self-perpetuating circuit and restores a sinus rhythm. Electrical cardioversion is much less effective in treating arrhythmia caused by increased automaticity (Table 9.1) (e.g. digitalis-induced tachycardia, catecholamine-induced arrhythmia) since the cause of the arrhythmia persists even after the arrhythmia is terminated and therefore is likely to recur.[6]

Table 9.1: Tachyarrhythmias based on predicted responses to cardioversion

Responsive (re-entrant)
Supraventricular arrhythmias
Atrial fibrillation
Atrial flutter
Sinoatrial nodal re-entrant tachycardia
AV nodal re-entrant tachycardia
AV reciprocating tachycardia
Ventricular arrhythmias
Monomorphic ventricular tachycardia due to scar/bundle branch re-entry
Unresponsive (automatic)
Supraventricular arrhythmias
Sinus tachycardia
Focal atrial tachycardias
Junctional tachycardia
Ventricular arrhythmias
Idiopathic monomorphic ventricular tachycardia
Accelerated idioventricular rhythm

INDICATIONS AND CONTRAINDICATIONS[4,6]

Delivery of synchronized shocks (cardioversion) is indicated to treat unstable tachyarrhythmias associated with an organized QRS complex and a perfusing rhythm (pulses).

Due to the potential risks involved, it is imperative that physicians be familiar with proper indications, precautions, techniques, and complications. The lists mentioned here are not all-inclusive and may be modified by clinical judgment in individual patients by advances in medical practice.[6]

Cardioversion on an elective basis (within a few weeks)

- Stable patients who have atrial fibrillation or flutter with significant and distressing symptoms or re-entrant tachycardia with significant symptoms that is resistant to therapy
- Asymptomatic patients with atrial fibrillation in whom long-term warfarin therapy is contraindicated or is rejected by the patient.

Cardioversion on an urgent basis (within 15 min)

- Patients with supraventricular arrhythmias with ongoing anginal pain from coexistent coronary disease or hemodynamic compromise
- Atrial fibrillation with rapid ventricular response in patients with Wolff-Parkinson-White syndrome
- Stable VT (monomorphic) after failure of intravenous antiarrhythmic agents.

Cardioversion on an emergency basis (immediately)

- Based on advanced cardiac life support (ACLS) guidelines, any patient with narrow or wide QRS complex tachycardia (ventricular rate >150) who is unstable (e.g. ongoing chest pain, pulmonary edema, lightheadedness, hypotension and other signs of shock or heart failure) should be immediately treated with synchronized electrical cardioversion.[4]

Extreme caution should be exercised in patients with digitalis toxicity or electrolyte imbalance because of their increased risk of ventricular tachycardia or fibrillation after being shocked. Patients with severe conduction system disease may develop significant bradyarrhythmia after cardioversion. In addition, patients who have been in atrial fibrillation for a prolonged or indeterminate length of time are at risk for thromboembolism due to cardioversion, appropriate measures should be taken to minimize this risk (see below).[8]

CONTRAINDICATIONS

Synchronized cardioversion is not used for treatment of VF, pulseless VT, or unstable polymorphic (irregular) VT. These rhythms require delivery of high-energy unsynchronized shocks (i.e. defibrillation doses).

OVERVIEW OF THE PROCEDURE[4,8,9]

Cardioversion, though a minor procedure, it is often needed in out-of-hours and often in remote sites. An understanding is required both of the pathophysiology underlying cardiac arrhythmias and of the technical side of defibrilltion equipment, including electrical safety.

The procedure is usually carried out in an area suitable for intravenous administration of a general anesthetic or sedative agent (discussed below) and for conduct of cardiopulmonary resuscitation, if necessary.

- Equipment for treatment of severe and clinically significant bradyarrhythmias should be available, and agents for treatment of malignant ventricular arrhythmias should be on hand
- A 12-lead electrocardiogram (ECG) is recorded and an intravenous cannula secured. Monitoring leads are attached in the standard manner with the electrodes away from the paddle sites
- Patient's ECG is monitored on the defibrillator to select the lead which properly senses the 'R' wave and not the 'T' wave of the ECG signal
- The defibrillator should be placed in the synchronized (sync) mode which permits a search for a large R or S wave. The desired energy is selected (Table 9.2)
- **Electrodes:** Until recently, hand-held paddles coated with conductive gel were the sole type of electrode used to deliver countershock. Self-adhesive pads have become more common in the past few years, although paddles are still used, especially in emergent cases. Limited data are available comparing the two modalities but one study suggested the superiority of paddles over pads in cardioverting atrial fibrillation.[8] Whichever, modality is used, impedance can be minimized by avoiding positioning over breast tissue by shaving or clipping body hair when it is excessive and by delivering the shock during expiration
- The optimal anatomic placement of pads and paddles is controversial. Anterior-lateral and anterior-posterior placements are both acceptable. The anterior paddle is placed on the right infraclavicular chest. In anterior-lateral placement, the lateral paddle should be located lateral to the left breast and should have a longitudinal orientation, since this results in a lower transthoracic impedance than horizontal orientation. When anterior-posterior positioning is used, the posterior pad is commonly located to the left of the spine at the level of the lower scapula, although some physicians favor placement to the right of, or directly over, the spine[8]
- Since the skin can conduct away a significant portion of the current, conductive gel or pre-gelled pads are commonly used to ensure good contact. Under ideal circumstances, only 10 to 30 percent of the total current reaches the heart
- Pacemakers and implantable cardioverters defibrillators should be at least 10 cm from direct contact with paddles and should eventually be interrogated for any malfunction after cardioversion
- The clearing chant "I am clear, You are clear, Everybody is clear" or some other equivalent is voiced and simultaneous visual inspection is done to confirm the same. This ensures that nobody is in contact with the patient as the shock is delivered. The activation button is then triggered to deliver the shock
- Immediately after the shock, the patient's rhythm is inspected on the monitor screen and then verified on a recorded ECG

Table 9.2: Suggested initial energy for cardioversion and defibrillation

Rhythm	*Biphasic*	*Monophasic*
Atrial flutter and PSVT	Unknown*	50 – 100 J
Atrial fibrillation	75 – 120 J	100 – 200 J
Ventricular tachycardia with pulse	100 J	200 J
Ventricular fibrillation, pulseless VT	150 – 200 J	360 J

* Studies are needed to confirm the exact biphasic equivalents. Approximately half the monophasic doses are usually recommended for many rhythms.

Table 9.3: Check list for cardioversion

Preparing the patient
• Ensure NPO status
• Obtain informed consent
• Apply self-adhesive pads (clip hair if needed)
• Achieve adequate sedation
• Monitor vital signs and cardiac rhythm throughout.
Performing the cardioversion
• Select initial energy appropriate for specific device
• Select the synchronization function
• Confirm that arrhythmia is still present
• Charge, clear, and deliver shock
• If no change in rhythm, escalate energy as appropriate
• Monitor ECG regularly.

- If the arrhythmia persists, a satisfactory level of general anesthesia or sedation is reaffirmed or reattained. The shock is repeated at a higher energy level until either normal rhythm is restored or a decision is made to abandon further attempts
- In the unlikely event that ventricular fibrillation follows the shock, the equipment is switched to the nonsynchronized mode and a defibrillating countershock of 300 J is delivered across the thorax
- All events are recorded and documented
- After the procedure, a 12-lead ECG is performed to document stability of the patient's QRS-T pattern (absence of evidence of injury).[8,4]

Table 9.3 gives the brief check-list before performing the cardioversion.

Energy requirements for atrial fibrillation using monophasic shocks are 100 to 200 J initially and 360 J for subsequent shocks. Biphasic shocks require a typical energy level of 75 to 100 J for correction of atrial fibrillation. Atrial flutter and PSVT require less energy: a monophasic shock of 50 J initially, then 100 J, if needed. Cardioversion of VT involves shocks of 50 to 100 J initially, then 200 J if unsuccessful.[10]

- In pediatric patients with PSVT or VT who are not hemodynamically stable, an initial synchronized shock of 0.5 J/kg is recommended. In subsequent attempts, the energy is increased
- During pregnancy, recommendations for other adults are applicable. [7,10]

Cardioversion devices using biphasic waveforms have higher success with less energy.[11]

ANESTHESIA FOR CARDIOVERSION

External cardioversion is a short, painful procedure with stimulus intensity similar to that of a surgical incision. This brief but distressing procedure should be carried out using sedation or sometimes general anesthesia. When TEE is being performed prior to cardioversion, the procedure may take 15 to 30 minutes. Elective cardioversion is often performed in areas near the operating room, usually in the PACU. Alternatively, there may be a requirement for the anesthesia team to provide sedation in the intensive care units (ICU) or sometimes out of hours in remote sites for urgent cardioversion in an unstable patient.[12]

Correct standards of care are important in these patients who have a risk of embolic events and may have critically compromised cardiac output.

Preprocedure Assessment

Accurate knowledge of the medical and drug history and thorough clinical examination is essential part of preprocedure evaluation. Patients may have other serious cardiovascular such as rheumatic disease, ischemic heart disease, recent myocardial infarction or cardiac failure. Other risk factors such as patients with predicted difficult airways and significant medical conditions should be looked for. Many patients with atrial fibrillation requiring urgent or emergency cardioversion will classify as American Society of Anesthesiologists (ASA) III; that is a patient with severe systemic disease that is not incapacitating. Such patients tolerate adverse events poorly and have a higher surgical mortality.[13] Digitalis therapy predisposes to post-cardioversion arrhythmias; in some centers, it is withheld for 48 hours before elecyive cardioversion. If urgent cardioversion is required in a patient receiving digoxin, the initial DC dose should be low (e.g. 10-25 J) and increased if necessary.

In some patients, there is a significant risk of embolic phenomena, e.g. those with:

- Mitral stenosis and atrial fibrillation of recent onset
- Atrial fibrillation and a dilated cardiomyopathy
- A prosthetic mitral valve
- A history of embolic phenomena.

Patient Preparation

In case of unconscious patients who are unstable due to tachyarrhythmia, countershock must be performed urgently. In more elective settings, patient safety and comfort become paramount. As with any procedure, informed consent should be obtained. Unless the procedure is emergent, all patients should refrain from eating and drinking for several hours in order to decrease the risk of aspiration. Constant heart rhythm monitoring should be used throughout the procedure and a 12-lead electrocardiogram should be obtained before and after the countershock.

Anesthesia Management

Treatment should be carried out only in areas specifically designed for the purpose and with a full range of drugs, resuscitation and monitoring equipment available.

The level of anesthesia required for cardioversion is either "deep sedation" or general anesthesia. Adequate depth of sedation is important firstly, to prevent recall of an unpleasant experience and attenuate the stress response; and secondly, to not precipitate hypoventilation, airway obstruction, or cardiac embarrassment. This is particularly important in a patient population with a high rate of myocardial ischemia and when rhythm stabilization is the ultimate goal.

Rapid onset and offset, cardiovascular stability, no respiratory depression and some analgesic component would be the ideal characteristics of the agents used.

The combination of a benzodiazepine, such as midazolam, with or without a narcotic, such as fentanyl, is a frequent choice in the absence of anesthesiology assistance.[8,14]

The usual anesthetic technique followed by most anesthesiologists for cardioversion is a small bolus of intravenous induction agent. All currently available induction agents are effective. Propofol is among the most preferred agent. Propofol produces hypotension more often than etomidate, although this can be attenuated by using an infusion or smaller titrated doses of propofol (1 mg/kg). Etomidate causes a high incidence of myoclonus, which can render interpretation of the ECG difficult. Recovery after midazolam tends to be longer than after the other agents, although this may be reversed with flumazenil. Propofol administered by a target controlled infusion has been compared with inhalational anesthesia with sevoflurane. Sevoflurane was found to provide greater hemodynamic stability than propofol. However, this technique is only practical if there is easy access to a sevoflurane vaporizer and an anesthesia machine. Fentanyl 1.5 μg/kg may also be administered 3 minutes before induction. When TEE is being performed before cardioversion, the patient needs to remain sedated for longer, and a propofol infusion may be useful. In general, patients do not require intubation for cardioversion unless there is a risk of regurgitation.(as in case of patients who require urgent cardioversion and in whom starvation status is unknown).

During TEE, local anesthetic is sprayed into the oropharynx to allow easy passage of the TEE probe. A bite block is inserted to prevent the patient from biting down on the probe, damaging both their teeth and the probe. The anesthesiologist can often assist the cardiologists by deepening the level of sedation in the initial stages to allow the TEE probe to be inserted more easily.[12,14]

Supplemental oxygen is delivered via nasal cannula, face mask, or in the case of heavier sedation seil inflating resuscitation bags (AmbuBag, Laerdal's resuscitator).[8] Less frequently, general endotracheal anesthesia may be required when patient is hemodynamically unstable, or with other associated risk factors requiring a rapid-sequence induction with cricoid pressure.[15]

The goal of sedation should be minimal or no response to verbal stimulus. In such a state, a patient may cry out during the actual cardioversion but will nonetheless usually have no recollection of the procedure after recovery.

The patient should be monitored carefully both during anesthesia and after recovery of consciousness, in particular for evidence of recurrent arrhythmia, hypotension, pulmonary edema, or systemic or pulmonary embolism. Existing hospital policies for monitoring during conscious sedation/general anesthesia should be followed, including frequent assessment of electrocardiogram, blood pressure and pulse oximetry.

Management of Resistant Arrhythmias

Electrical cardioversion is unsuccessful in 10 to 30 percent of cases of atrial fibrillation and up to 28 percent of cases of atrial flutter. The duration of atrial fibrillation is inversely related to the probability of successful cardioversion.

When cardioversion fails, the operator's technique should be reviewed and modified. Electrode position may be altered from anterior-posterior to anterior-lateral or vice versa. If paddles are being used, firmer pressure may be employed. If a device that delivers monophasic waveform shocks is being employed, it may be exchanged for one that delivers biphasic waveform shocks. Synchronized shocks from two separate defibrillators using electrical switches to coordinate the shocks may be performed. An antiarrhythmic medication may be initiated prior to another attempt at cardioversion. Finally, transvenous cardioversion may be attempted (see below).

Although some patients fail to achieve sinus rhythm, many who are successfully cardioverted revert to atrial fibrillation within minutes, hours, or days. The administration of antiarrhythmic pharmacologic therapy decreases this possibility significantly. However, given the adverse reactions associated with these medications, the necessity of maintaining sinus rhythm should be carefully considered. When atrial fibrillation is associated with substantial symptoms that are not alleviated by rate-controlling medications, antiarrhythmic therapy may be

indicated. However, patients in whom the arrhythmia is well tolerated may be served as well by a strategy of rate control and anticoagulation.[8]

Transvenous Cardioversion[8]

Cardioversion using high-energy shocks delivered internally via right atrial (RA) catheter and a backplate was described in 1988. This technique was demonstrated to be more efficacious than external cardioversion, especially in patients who are obese or who have chronic obstructive pulmonary disease.

Lower energy internal shock using an RA cathodal electrode and an anode in the coronary sinus or left pulmonary artery has also been described.

Pharmacologic Cardioversion

Cardioversion can be achieved not only electrically but also pharmacologically. Pharmacologic cardioversion is used mainly for atrial fibrillation and flutter of relatively short duration. Although electrical cardioversion is quicker and has a higher probability of success, pharmacologic cardioversion does not require sedation. The risk of thromboembolism with pharmacologic cardioversion has not been well established but is thought to be similar to that of electric countershock because it is the return of sinus rhythm rather than the shock itself that is believed to precipitate thromboembolism.

Dofetilide, flecainide, ibutilide, propafenone, amiodarone, and quinidine have been demonstrated to have some degree of efficacy in restoring sinus rhythm. Each of these medications has potential toxicities including malignant arrhythmias and hypotension. The risks and benefits should be carefully weighed when selecting a pharmacologic agent. Although beta-blockers and calcium channel antagonists are often believed to facilitate cardioversion, their efficacy has not been established in controlled trials.[8,15]

Patients with Implanted Pacemakers and Defibrillators[6-8,14,15]

Patients with implanted pacemakers and defibrillators may undergo external cardioversion and defibrillation safely in most cases. However, one must be aware of the possibility that external energy delivery may alter the programming of the internal device. Furthermore, energy may be conducted down an internal lead, causing local myocardial injury and a resultant change in the pacing or defibrillation threshold. The paddles or pads used for external electric countershock should never be placed over the internal device.

Pacemakers and ICDs should be at least 10 cm from direct contact with paddles and should eventually be interrogated for any malfunction after cardioversion.[6,7]

Cardioversion and Defibrillation in Pregnancy[7,8]

Cardioversion and defibrillation have been performed in all trimesters of pregnancy without obvious adverse fetal effects or premature labor. It has been suggested that the fetal heart rhythm be monitored during cardioversion.

Complications

Burns: Countershock can cause first-degree burns and pain at the paddle or pad site. The lowest effective energy should be used to minimize skin injury.

Thromboembolism: Cardioversion of atrial fibrillation and atrial flutter carries a risk of thromboembolism. One to seven percent of patients in atrial fibrillation who undergo cardioversion without receiving anticoagulation may experience this complication.[8]

Arrhythmia: Bradyarrhythmias such as sinus arrest and sinus bradycardia are common immediately after countershock and are almost always short-lived. However, patients who have atrial fibrillation with a slow ventricular response in the absence of medications that slow AV conduction should be suspected of having conduction disease and are at higher risk for sustained bradyarrhythmia after cardioversion. The prophylactic placement of a transvenous or transcutaneous pacemaker may be considered in this situation.

VT and ventricular fibrillation can occasionally be precipitated by countershock, particularly in patients with digitalis toxicity or hypokalemia. Elective cardioversion should therefore be avoided in patients with these conditions. If cardioversion or defibrillation must be performed urgently, one should anticipate the ventricular arrhythmias to be more refractory to shock than usual.

Myocardial damage: Occasionally, one may see transient ST elevations on postcountershock electrocardiograms. This is unlikely to signify myocardial injury. Nonetheless, it has been suggested that any two consecutive shocks be delivered no less than one minute apart to minimize the chance of myocardial damage. Of course, this recommendation applies only to nonemergent situations.[16]

Anticoagulation and Cardioversion

Patients with atrial fibrillation or flutter may develop thrombus in the left atrial appendage or left atrial cavity,

Table 9.4: Recommendations of American Association of chest Physicians for anticoagulation before and after cardioversion

- Warfarin for 3 weeks before nonemergency cardioversion of atrial fibrillation of >24-48 hours' duration
- Warfarin for 4 weeks after cardioversion
- Intravenous heparin followed by warfarin, if cardioversion cannot be postponed for 3 weeks
- Anticoagulants may not be needed for atrial fibrillation of less than 48 hours duration or for cardioversion of supraventricular tachycardia
- Consideration should be given to managing atrial flutter similar to atrial fibrillation.

leading to thromboembolism during or after cardioversion. There is a general agreement that cardioversion of patients who have been in atrial fibrillation for less than 24 to 48 hours are very unlikely to cause thromboembolism.[8] Current guidelines indicate that pericardioversion anticoagulation with heparin or low molecular weight heparin is optional in these patients.[17] Patients, in whom the arrhythmia has been present for longer than 24 to 48 hours, or for an undetermined length of time, are felt to be at higher risk. When these patients do not require urgent cardioversion for reasons of symptomatology, there are two reasonable approaches.

In the first case, one may perform a transesophageal echocardiogram to assess for the presence of thrombus in the left atrial appendage. If thrombus is not visualized, the patient is considered to be at low risk for thromboembolism, and cardioversion may be performed. Anticoagulation with warfarin to an international normalized ratio (INR) goal of 2.5 (range 2.0 to 3.0) is recommended for 4 weeks after cardioversion. The second approach is to defer cardioversion until the patient has been anticoagulated at a therapeutic level for at least 3 weeks. Cardioversion is then performed and the patient anticoagulated for a minimum of 4 weeks afterward (Table 9.4).[18]

Prognosis after Cardioversion

A long previous duration of arrhythmia, previous episodes of atrial fibrillation, and age >50 years predict unsuccessful maintenance of sinus rhythm and reversion to atrial fibrillation. In addition, the presence of coronary artery disease, hypertension, and other organic disease such as mitral valve disease, aortic stenosis, and cardiomyopathy-are detrimental to maintaining normal sinus rhythm. Recent studies suggest that left atrial size does not influence the outcome after cardioversion but that the duration of atrial fibrillation is the most important predictor for outcome.

CONCLUSION

The gold standard for cardioversion is a brief period of general anesthesia or deep sedation using short acting agents with a rapid recovery profile. It requires the presence of an airway specialist (i.e. an anesthesiologist) to provide controlled airway support, analgesia, amnesia and maintenance of the cardiac output if required. The patient population for cardioversion are not straightforward to deeply sedate or anesthetize, as they have coexistent medical problems and may have a reduced cardiac output after the procedure.

REFERENCES

1. Lown B. Defibrillation and cardioversion. Cardiovasc Res 2002;55(2):220-4.
2. Sucu M, Davutoglu V, Ozer O. Electrical cardioversion. Ann Saudi Med 2009;29(3):201-6.
3. Yurchak PM. Clinical competence in elective direct current (DC) cardioversion. A statement for physicians from the ACP/ACC/AHA Task Force on Clinical Privileges in Cardiology. J Am Coll Cardiol 1993;22(1):336-39.
4. Tracy CM, Akhtar M, DiMarco JP, Packer DL, Weitz HH, et al. American College of Cardiology/American Heart Association Clinical Competence Statement on Invasive electrophysiology Studies, Catheter Ablation, and Cardioversion. Circulation 2000;102: 2309.
5. Lown B. Electrical reversion of cardiac arrhythmias. Br Heart J 1967;29:469-89.
6. Akhtar M, Achord JL, Reynolds WA. Clinical competence in invasive cardiac electrophysiological studies. ACP/ACC/AHA Task Force on Clinical Privileges in Cardiology. J Am Coll Cardiol 1994;23:1258-61.
7. Part 5: Electrical Therapies. Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing. AHA/ACC Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2005;112:IV-35-46.
8. Botkin NF. Cardioversion and Defibrillation. In: Irwin RS, Rippe JM, Lisbon A, Heard SO, (editors). Procedures, Techniques, and Minimally Invasive Monitoring in Intensive Care in Intensive Care Medicine. 4th edn. United States of America: Lippincott Williams & Wilkins 2008; chapter 6.
9. Ewy GA. The optimal technique for electrical cardioversion of atrial fibrillation. Clin Cardiol 1994;17(2):79-84.
10. Levy S, Ricard P, Gueunoun M, et al. Low-energy cardioversion of spontaneous atrial fibrillation. Immediate and long-term results. Circulation 1997;96(1):253-9.
11. Deakin CD, Ambler JJ. Post-shock myocardial stunning: A prospective randomized double-blind comparison of monophasic and biphasic waveforms. Resuscitation 2006;68(3):329-33.

12. Souter KJ. Anesthesia Provided at Alternate Sites. In: Barash PG. Cullen BF, Stoelting RK (editors). Clinical Anesthesia, 5th edn. United States of America: Lippincott Williams and Wilkins 2006:1331-44.
13. Vacanti CJ, Van Houten RJ, Hill RC. A statistical analysis of the relationship of physical status to postoperative mortality in 68388 cases. Anesth Analg 1970;49:564.
14. Harrison SJ, Mayet J. Cardioversion and the use of sedation. Heart 2004;90:1374-76.
15. Thompson JP. Anaesthesia for plastic, endocrine and vascular surgery. In: Aitkenhead AR, Rowbotham DJ, Smith G (editors). Textbook of Anaesthesia, 4th edn. London: Churchill Livingstone 2001:680-83.
16. Dahl CF, Ewy GA, Warner ED, et al. Myocardial necrosis from direct current countershock: Effect of paddle electrode size and time interval between discharges. Circulation 1974;50:956-61.
17. Singer DE, Albers GW, Dalen JE, et al. Antithrombotic therapy in atrial fibrillation: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004;126 (Suppl 3):429-56S.
18. DiMarco JP, Tracy CM, Akhtar M, et al. American College of Cardiology/American Heart Association clinical competence statement on invasive electrophysiology studies, catheter ablation, and cardioversion. J Am Coll Cardiol 2000;36:1725-36.

Section III

General Surgery

10 Anesthesia for Intestinal Obstruction

Amala G Kudalkar, Trupti S Pethkar

KEY POINTS

- Intestinal obstruction is one of the commonest surgical emergency encountered in clinical practice.
- Intestinal obstruction can be defined as 'failure of normal forward transit of intestinal contents'. The obstruction can be either anatomical or functional, i.e. ileus.
- The anatomical obstruction can be inside the lumen, in the wall or outside the intestine. The commonest cause of ileus is postoperative status and electrolyte imbalance.
- Obstruction can be simple with only luminal obstruction and intact blood supply or strangulated where the blood supply to the obstructed intestine is compromised. Strangulated bowel carries high mortality.
- The main pathophysiological features include, bowel distension proximal to the obstruction, increase intra-luminal and intramural pressure, exudation of fluid in the bowel lumen and wall, bowel ischemia and peritonitis.
- The changes will differ according to the site of obstruction, small bowel obstruction is associated with severe fluid and electrolyte imbalance.
- The sequels of intestinal obstruction are intra-abdominal hypertension and abdominal compartment syndrome, fluid shifts, ileus, perforative peritonitis and sepsis.
- Intra-abdominal pressure more than >12mm Hg is termed as IAH and is clinically significant when more than 25 mm Hg. Various grades of IAH are described.
- IAH affects respiratory, cardiovascular, GI, renal, central nervous systems and eyes.
- Bowel ischemia results in breach in the anatomical mucosal barrier and translocation of bacteria into the peritoneum and from there into systemic circulation.
- Fluid shifts occur because of loss of fluid into the lumen of the bowel, bowel wall, in the peritoneum and outside the body and cause electrolyte imbalance. Prior to surgery, replacement of this loss is mandatory.
- Fluid resuscitation includes correction of deficit, maintenance and replacement of ongoing losses. The composition of fluid will depend upon the nature of loss.
- Investigations should include CBC, liver and renal biochemistry, electrolytes, sugars, ABG, coagulation studies if sepsis suspected, X-ray chest and ECG if indicated.
- These patients are at high risk for regurgitation and aspiration of stomach contents. Therefore, rapid sequence induction-intubation is mandatory.
- On opening peritoneum, there may be sudden drop in blood pressure and dysrhythmias. Intraoperatively patient should be extensively monitored which should be continued in the postoperative period.
- Patient with hemodynamic instability, IAH, sepsis and severe co-morbid conditions should be electively ventilated postoperatively.
- Development of sepsis and ARDS must be anticipated and treated in time.

Intestinal obstruction is one of the commonest surgical emergencies encountered in clinical practice. Often it results in perforation of intestine leading to peritonitis. It is associated with abdominal distension, increased intra-abdominal pressure, high risk of pulmonary aspiration, fluid and electrolyte disturbances and likelihood of development of sepsis.

Intestinal obstruction can be defined as 'failure of normal forward transit of intestinal contents either because of partial or complete blockage of the bowel

lumen or because of an ileus, i.e. the functional failure of progressive intestinal transit'.[1]

ETIOLOGY

As stated earlier, obstruction can be either mechanical or functional.[1,2]

1. **Mechanical Obstruction:** Actual physical barrier that blocks the intestinal lumen, preventing normal forward progression of intestinal contents.
   a. **Obstruction by extraneous substances in the lumen (intraluminal):**
      - Meconium
      - Intussusception
      - Gallstones
      - Impactions—fecal, barium, bezor, worms, foreign bodies, food bolus, etc.
   b. **Lesions of the bowel wall (intramural):**
      - Congenital—atresia, stenosis, imperforate anus, Meckel's diverticulum
      - Traumatic
      - Inflammatory—regional enteritis, diverticulitis, chronic ulcerative colitis, PID
      - Neoplastic
      - Infections—typhoid fever, pelvic abscess, appendicular mass
      - Miscellaneous—radiation strictures, endometriosis
   c. **Lesions extrinsic to bowel (extraluminal):**
      - Adhesive band of constriction or angulation by band either due to previous abdominal operations or abdominal sepsis of any kind
      - Hernias
      - Extrinsic masses—annular pancreas, anomalous vessels, abscesses or hematomas, neoplasms.
2. **Functional Obstruction:** Failure of downward progress of bowel contents because of disordered propulsive motility of the bowel due to:
   a. Neuromuscular defects—megacolon, paralytic ileus.
   b. Abdominal causes—intestinal distension, plastic tuberculous peritonitis.
   c. Systemic causes—electrolyte imbalance, toxemias.
   d. Vascular occlusion—arterial or venous.

The intestinal obstruction can be described in various ways as:

- **Simple:** There is an altered motility and lumen is obstructed causing distension of proximal bowel but there is no loss of blood supply. It causes progressive accumulation of fluid and gas above the level of obstruction with vomiting and systemic derangement
- **Closed loop:** A segment of intestine is obstructed both proximally and distally (e.g. with volvulus) with rapid progression to strangulation. Another classical example of this is carcinoma sigmoid colon with competent ileocecal valve

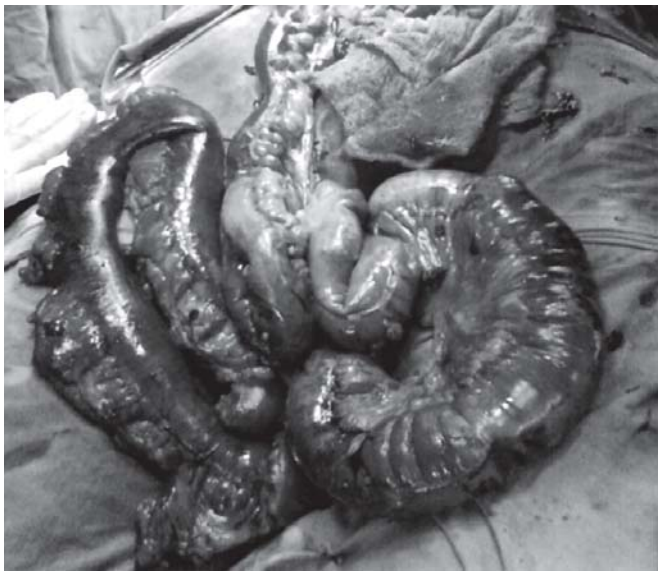


Fig. 10.1: Strangulated intestinal obstruction
(For color version, see Plate 1)

- **Strangulated:** This is intestinal obstruction with vascular compromise. (Fig. 10.1) It leads to leakage of blood, fluid and toxic substances into bowel lumen and peritoneal cavity. It is important to recognize strangulation preoperatively because the consequences are so severe that the mortality is high between 20 to 40 percent.
- **Partial/complete:** In partial obstruction, only a portion of the intestinal lumen is occluded, allowing passage of some gas and fluid. The progression of pathophysiologic events tends to occur more slowly than with complete small-bowel obstruction and development of strangulation is less likely. Continued passage of flatus and/or stool beyond 6 to 12 hours after onset of symptoms is characteristic of partial rather than complete obstruction
- **Acute/chronic:** This depends on the onset and progress of the symptoms. Patients having strictures, postoperative adhesions, tuberculous matting of intestines will have chronic symptoms of peritonitis over a long period
- **Small intestinal/colonic:** It is very important to know the site of obstruction as small and large intestinal obstruction as they have different clinical implications. Small intestine has both secretary and absorptive function. Small intestinal obstruction leads to accumulation of succus entericus above the level of obstruction, in addition to the deranged motility. Systemic derangements with fluid and electrolyte loss, bowel distension by intestinal gas is comparatively severe. While large bowel being a storage organ with less absorptive and secretary

functions, its obstruction is usually insidious in onset with less severe systemic derangements due to decreased vomiting and fewer propensities to strangulate except in volvulus, but progressive distension can cause rupture especially in the presence of competent ileocecal valve as it becomes a closed loop obstruction. Commonly it is due to neoplasms, diverticulitis and volvulus.

PATHOPHYSIOLOGY

Upon development of bowel obstruction, the bowel above the obstruction initially gets increased blood supply and develops hyper- peristalsis in an attempt to overcome the obstruction. The peristaltic wave may get reflected back from the point of obstruction giving rise to reverse peristalsis. If the obstruction is not relieved, the bowel above the obstruction dilates and becomes flaccid. This stagnation leads to bacterial overgrowth inside the lumen. It also causes sequestration of fluid (unabsorbed digestive juices, i.e. succus entericus) and gases (nitrogen and hydrogen sulphide) causing bowel distension. As bowel distension continues from the ongoing gas and fluid accumulation, intraluminal and intramural pressure rises. This increased intraluminal pressure initially causes to increase in secretary function of the bowel via prostaglandin release. But once the intraluminal pressure goes beyond 20 cm H_2O, the reabsorption is impaired. The increased intramural pressure leads to progressive interference with mesenteric blood supply, first venous followed by arterial. Venous obstruction prevents drainage of blood from the intestine leading to massive edema of the bowel. The intramural vessels become stretched. Obstruction to the arterial supply leads to necrosis of the bowel wall leading to perforation. Bowel contents leak out into the peritoneum and cause generalized peritonitis. In case of total vascular obstruction there is strangulated obstruction[1,2] (Table 10.1).

Table 10.1 Pathophysiology of intestinal obstruction[2]

Obstruction of a part of bowel
↓
Accumulation of gas and fluids in proximal bowel
↓
Dilatation of proximal bowel segment
↓
High intraluminal pressure
↓
Reduced reabsorption and mucosal edema
↓
Impairment of venous and arterial blood flow
↓
Ischemia, infarction and perforation of the gut wall
↓
Transmucosal and transperitoneal translocation of bacteria and endotoxins
↓
Intra-abdominal hypertension and systemic toxicity
↓
Massive loss of fluid, dehydration, electrolyte imbalance

The major complications of intestinal obstruction are:

1. Intra-abdominal hypertension.
2. Fluid shifts.
3. Ileus.
4. Perforation and sepsis.

Abdominal Compartment Syndrome

IAP is the steady state pressure concealed within the abdominal cavity. Normal IAP is 0 to 6.5 mm Hg. For most critically ill patients, an intra-abdominal pressure of 5 to 7 mm Hg is considered normal.[11] In a prospective study of 77 supine hospitalized patients, the intra-abdominal pressure averaged 6.5 mm Hg and was directly related to body mass index.[12] The normal range described above is not applicable for all patients. Patients with increased abdominal girth that developed slowly may have higher baseline IAPs. As an example, morbidly obese and pregnant individuals can chronically have IAP as high as 10 to 15 mm Hg without adverse sequelae.[11]Sustained increase in IAP of >25 mm Hg is clinically significant.

IAH is defined as a sustained intra-abdominal pressure of 12 mm Hg.[11] Although this value was established arbitrarily, it is used in many research studies and it distinguishes most patients whose IAP is inappropriately elevated. ACS represents the pathophysiologic consequence of a raised intra-abdominal pressure.

The elevated IAP is the net product of the rate of fluid accumulation within the abdominal cavity and the compliance of the abdomen. The pressure-volume curve for the abdominal cavity is nonlinear. Progressive accumulation of fluid within the peritoneal cavity with the decreasing compliance of the abdomen causes greater increase in IAP results. In 2000, Cheatham et al found **abdominal perfusion pressure (APP)** to be a much better predictor of end-organ injury than lactate, pH, urine output, or base deficit.[13] The APP is equal to the mean arterial pressure (MAP) minus the IAP. (APP = MAP-IAP). An APP of 50 mm Hg or higher is the optimum resuscitation goal in all critically ill patients.[14] In 1996, Burch et al developed a grading system

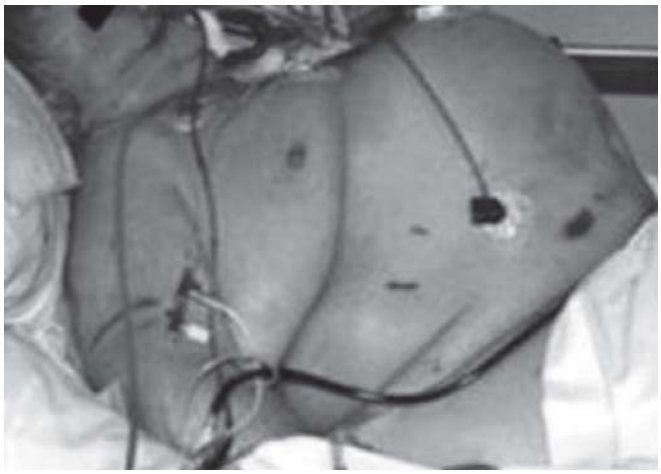


Fig. 10.2: Abdominal compartment syndrome
(For color version, see Plate 1)

according to which patients with higher grade ACS are shown to have end-organ damage, which is evidenced by splanchnic hypercarbia and elevated lactate levels, even if they appear clinically stable.

Grading of IAH

Table 10.2 shows the grading of IAH.[15-18] It is advocated that grades I and II have to be managed conservatively and grades III and IV with surgical decompression through a midline laparotomy termed 'decompressive laparotomy' (DL).

Table 10.2: Grading of IAH[15-18]

Grading	*IAP (cm H_2O)*	*Featmures*
Normal or Grade I	10-15 (7.5-11mm Hg)	No s/s
Mild or Grade II	15-25 (11-18 mm Hg)	- Cardiac index is usually maintained or even increased. Abdominal viscera are mildly squeezed and venous return increases. - Respiratory and renal symptoms are unlikely to occur. - Hepatosplanchnic blood flow may decrease. - Intravascular volume optimization will probably correct these alterations.
Moderate or Grade III	25-35 (18-25 mm Hg)	- Full syndrome may be observed - Bowel motility disrupted - Usually responds to aggressive fluid resuscitation. - Surgical decompression should be considered.
Higher Grade IV	>35 (>25 mm Hg)	- Oliguria followed by anuria and renal shutdown. - Surgical decompression associated with fluid resuscitation. Transient use of vasopressors is mandatory.

Etiology

Clinical settings which have been associated with the syndrome include:[3,6,14,19-22]

- Trauma: Patients with hepatic or intra-abdominal vascular injuries cause hemoperitoneum. Ruptured abdominal aortic aneurysm, massive intraperitoneal or retroperitoneal hemorrhage, pneumatic anti-shock garments, abdominal closure under excessive tension, ongoing surgical bleeding (missed injuries) or bleeding controlled with intra-abdominal packs are other conditions that lead to ACS. This condition is compounded by hypothermia and coagulopathy.
- Ovarian tumors
- Liver transplantation
- Intestinal obstruction
- Ascites under pressure
- Increasing bowel wall edema and third-space fluid losses due to large volumes of blood products and nonsanguinous solutions used in resuscitation without an associated intra-abdominal injury.

Abdominal compartment syndrome (ACS) refers to the development of physiologic dysfunction in intra-abdominal and extra-abdominal organs that occurs as a result of increased intra-abdominal pressure.[3-6] It is defined as:

An intra-abdominal pressure (IAP) of at least 20 mm Hg with dysfunction of at least one thoraco-abdominal organ or

IAP of more than 20 mm Hg complicated by one of the following:

- Peak airway pressure more than 40 cm H_2O
- Oxygen delivery index less than 600 ml O_2/min/m^2 or
- Urine output of less than 0.5 ml/kg/hr.[3,7-9]

It is probably under recognized because it primarily affects patients who are already quite ill and whose organ dysfunction may be incorrectly ascribed to progression of the primary illness. Since treatment can improve organ dysfunction, it is important that the diagnosis be suspected in the appropriate clinical situation. The incidence of ACS varies between 15 and 38 percent of all surgical patients admitted to intensive care units[8,10] (Fig 10.2).

Classification

ACS can be divided into three types as follows:[23]

- **Primary/acute:** Caused by intra-abdominal pathology like penetrating trauma, intraperitoneal hemorrhage,

pancreatitis, external compressing forces, such as debris from a motor vehicle collision or after a large structure explosion, pelvic fracture, rupture of abdominal aortic aneurysm or perforated peptic ulcer

- **Secondary:** Occurs without an intra-abdominal injury, when fluid accumulates in volumes sufficient to cause IAH like large-volume resuscitation (>3L), large areas of full-thickness burns develop ACS within 24 hours after receiving liberal fluid therapy
- **Chronic:** Occurs in peritoneal dialysis, morbid obesity, cirrhosis, meig's syndrome.

Pathophysiology

IAH causes impairment of respiratory, hemodynamic, renal and splanchnic function. It is characterized by:

- Tense and distended abdomen
- Hypotension and hypoventilation
- High airway pressures
- Hypercapnia
- Oliguria.

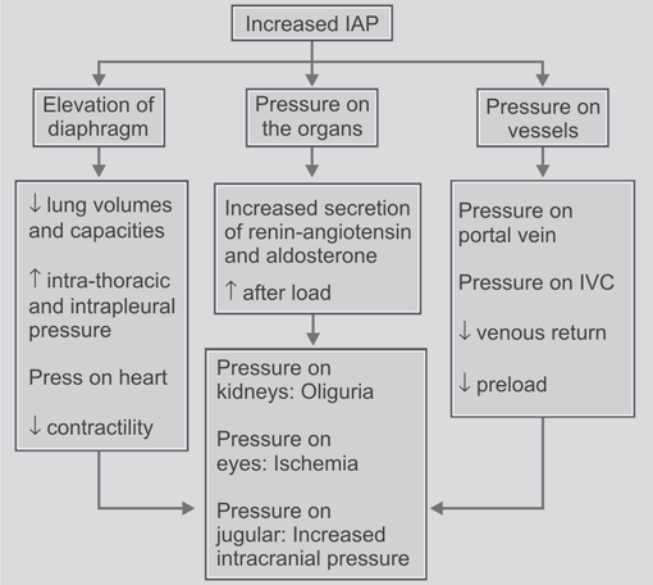
Abdominal compartment syndrome follows a destructive pathway similar to compartment syndrome of the extremity.

It represents the pathophysiologic consequence of a raised intra-abdominal pressure. Problems begin at the organ level with direct compression; hollow systems such as the intestinal tract and portalcaval system collapse under high pressure. Immediate effects such as thrombosis or bowel wall edema are followed by translocation of bacterial products leading to additional fluid accumulation, further increasing intra-abdominal pressure. At the cellular level, oxygen delivery is impaired leading to ischemia and anerobic metabolism. Vasoactive substances such as histamine and serotonin increase endothelial permeability, further capillary leakage impairs red cell transport and ischemia worsens. Although the peritoneal cavity and to a lesser extent, retroperitoneal space are distensible, they reach an endpoint at which the pressure rises dramatically. This is less apparent in chronic cases because the fascia and skin slowly stretch and thus tolerate greater fluid accumulation. Therefore, abdominal compartment syndrome should be recognized as a possible cause of decompensation in any critically injured patient.

Systemic manifestations of increased intra-abdominal pressure:

Primarily, increased IAP results in dysfunction of the respiratory, cardiovascular, renal, central nervous, abdominal and visceral system and eyes directly. They are shown in a nutshell in Table 10.3.

Table 10.3 Effect of intra-abdominal hypertension



Respiratory System[2]

Distended bowel causes pressure on diaphragm limiting its downward movement. The hemidiaphragms are elevated due to the increased IAP. The increased intra-abdominal pressure is transmitted to the pleural space so that intrathoracic pressure rises and lung compliance decreases. The work of breathing is increased with decrease in tidal volume and functional residual capacity. Peak inspiratory pressure (PIP) and pulmonary vascular resistance increase. Higher pressures are required to deliver a set tidal volume. The patient is hypoxic due to venous admixture caused by pulmonary shunting and ventilation perfusion mismatch and pulmonary atelectasis caused by abdominal distension and diaphragmatic splinting. Increasing positive end-expiratory pressure (PEEP) is required to maintain oxygenation of the patient. This can exacerbate cardiovascular and hemodynamic abnormalities in the patient with elevated IAP. This respiratory compromise with signs of poor perfusion will result in a decreased oxygen delivery and impairment in ventilation resulting in hypercarbia, acidosis and progressive hypoxemia. The oxygen requirement is also likely to be raised due to the local and systemic effects of the bowel obstruction.

The respiratory drive is increased and the arterial blood gas shows partially compensated metabolic acidosis. The potential causes for the metabolic acidosis are:

- Anaerobic metabolism with lactic acid production secondary to a global reduction in oxygen delivery (shock)
- Anaerobic metabolism in ischemic bowel
- Acidosis due to decreased renal perfusion (shock) and
- Loss of bicarbonate into the gut (balanced to some extent by a loss of acidic gastric secretions).

Cardiovascular System

We have already seen that increase in the IAP increases intrathoracic and airway pressures. Since the measured pulmonary artery wedge pressure (PAWP) and central venous pressure (CVP) are each the sum of pleural pressure and the intravascular filling pressures, they are spuriously elevated.[24] There is reduced venous return (↓ preload) to heart, causes direct compression of the heart (↓ contractility) and increases afterload (especially in the right ventricle).[25] Thus, there is elevation of CVP and pulmonary capillary wedge pressure (PCWP) even in hypovolemic or euvolemic patients. Cardiac output (CO) decreases progressively as the IAP increases. The magnitude of the depression of CO is dependent on the intravascular volume status. All effects of ACS are exacerbated by hypovolemia. Poor perfusion/shock is indicated by increased heart rate (HR), low blood pressure (BP), cold peripheries and acidosis. This is caused by fluid loss into the bowel and peritoneum and losses due to vomiting.[2] Intravenous volume expansion will increase the CO and central filling pressures in ACS, but will not correct the other manifestations of ACS, including depressed renal function and splanchnic blood flow. Septic shock may occur due to bacterial translocation across the gut wall or due to local complications such as perforation or strangulation.[2]

Renal System

Oliguria develops despite measured normal or mildly elevated CVP and PAWP. Oliguria occurs with IAP >15 mm Hg and anuria results with IAP >30 mm Hg. Blood flow and glomerular filtration in the kidney are diminished. The renal vein and inferior vena cava are compressed. In addition, renal vascular resistance increases several-fold in ACS. Direct compression of the renal parenchyma also contributes to the renal dysfunction. Oliguria is often the earliest sign of ACS and anuria follows if the IAP is not reduced. Abdominal decompression in combination with intravascular volume expansion reverses the effects upon renal function and normalises reninangiotensin - aldosterone system.[26] Renal failure is not prevented by intraureteral stents, which suggests direct compression of renal parenchyma and decreased renal perfusion as etiology.

Abdominal and Visceral Effects

- Clinically as the IAP increases, the abdominal girth increases and the abdomen becomes tenser which causes reverse peristalsis. Deeper planes of anesthesia and high doses of muscle relaxants are required to provide adequate operating conditions. The closure of the abdomen may be difficult
- Increased possibility of aspiration of gastric contents into tracheobronchial tree as a result of increased intragastric pressure and reverse peristalsis
- Bowel is distended as a result of accumulation of fluid and gas and has altered motility
- Blockage of venous outflow from the strangulated segment with subsequent extravasation of blood and fluid into the bowel wall. In addition, toxic breakdown products, exotoxins and endotoxins are released into peritoneal cavity
- Splanchnic blood flow decreases as ACS develops. Splanchnic ischemia is reflected by a decreased mucosal pH,[27,28] decreased liver metabolism, bowel ischemia and bacterial translocation.[29] There is decreased organ blood flow index (organ blood flow/cardiac output) in all major abdominal organs, except the adrenal glands.[3] Ileal and gastric mucosal blood flow are decreased with increased IAP.[29,30] Small bowel tissue oxygenation is decreased in ACS.[31]
- Also, perfusion of the abdominal wall may be decreased, so that wound healing may be impaired.

Central Nervous System

Elevation in intracranial pressure (ICP) and decrease in cerebral perfusion pressure (CPP) may also result from increased IAP. The proposed mechanism is functional obstruction of jugular venous drainage due to the elevated pleural pressures and CVP. Abdominal decompression results in a return toward baseline for ICP and an improvement in the CPP.[32] With the common association of abdominal injury and closed head injury, this observation is important. In these patients DL results in a dramatic reduction in ICP.[33]

Eyes

Sudden increased IAP has been associated with the rupture of retinal capillaries, resulting in the sudden onset of decreased central vision (valsalva retinopathy). The retinal hemorrhage usually resolves within days to months and no specific treatment is necessary. If a

patient with ACS develops visual changes, valsalva retinopathy should be considered and an appropriate ophthalmic examination performed.

Secondary effects of ACS occur immediately after evacuation. Care should be taken to prevent rapid escape of this fluid from the abdomen at the time of surgical incision, to minimize severe hypotension and even asystole. Theories to explain these effects include reperfusion syndrome and suddenly decreased systemic vascular resistance (SVR). Volume resuscitation immediately before decompression has been shown to significantly decrease this event. Preventing ACS is much more effective than treating it.

Diagnosis

To diagnose and intervene early in the course of ACS, a high index of suspicion must be maintained. It is difficult because it usually occurs in critically ill patients with other causes of circulatory or respiratory failure.

Clinically, the syndrome consists of the association of abdominal distension with increasing PIPs, increased CVP (if the patient is euvolemic), oliguria, hypercarbia and acute circulatory failure with wide systolic-diastolic pressure variation and elevated filling pressures.

On CT scan, ACS shows round-belly sign - abdominal distension with an increased ratio (>0.80) of anteroposterior-to-transverse abdominal diameter, collapse of the vena cava, bowel wall thickening with enhancement and bilateral inguinal herniation (Fig. 10.4).

Often, a diagnosis of ACS should be made on the basis of clinical suspicion and DL performed without attempts at measuring IAP. After exclusion of cardiac tamponade and increased pleural pressure (tension pneumothorax, status asthmaticus, etc), the IAP should be measured. In the early phases of ACS, when oliguria may be the only sign, measurement of IAP is useful.

Methods of measuring IAP:

- Measurement of bladder pressure, measurement of the rectal and gastric pressure
- Measurement of the IAP using a long femoral venous catheter placed in the inferior vena cava and direct measurement of the intra-abdominal pressure by direct puncture.

The most accurate and simple way to determine the IAP is indirectly by measurement of the bladder pressure using a Foley catheter. The bladder pressure is essentially equivalent to the IAP. It can be measured either using a pressure transducer or simply by a hand held manometer. In experimental conditions, bladder pressure is closely related to abdominal pressure.[4] Yol et al[34] found good agreement between bladder pressure and intra-abdominal pressure. However, Johna et al[35] found that bladder pressure systematically overestimated the true abdominal pressure; this could lead to an over-diagnosis of ACS. Bladder pressure increments reflect increments in IAP on an individual basis, therefore, it is used in patients at risk of developing an intra-abdominal compartment syndrome. Finally, most clinical studies have used bladder pressure measurements[3,28] so that clinical manifestations have been classified according to bladder pressure (cm H_2O) levels rather than directly measured intra-abdominal pressure (mm Hg) levels. The study by Johna et al[35] highlights that bladder pressure measurements require cautious interpretation, but it is still thought to be an easy, safe and valuable tool for diagnosing ACS in critically ill patients.

Treatment

- Oxygen is provided with a facemask
- Nasogastric tube is inserted to decompress the stomach and bowel
- Dehydration is treated with fluids with central venous pressure monitoring
- Inotropes may be required for hemodynamic instability. The goals of pharmacotherapy for ACS are to reduce intra-abdominal pressure
- Diuretics is the mainstay of the treatment
- Decompression results in immediate diuresis and physiological improvement.

Nonintervention may result in death. If ACS is present based on the measured IAP or clinical suspicion, DL should be performed.

During decompression of the abdomen, the following precautions should be taken to prevent hemodynamic decompensation:

- Restoration of the intravascular volume
- Maximization of oxygen delivery
- Correction of hypothermia
- Correction of coagulation defects.

The decompression may be carried out in the surgical intensive care unit on an emergency basis, however, the operating room (OR) is preferable. OR must be prepared to accept the patient if surgically correctable lesion is identified at the time of DL.

After decompression, prompt diuresis occurs and polyuria often develops. Peak airway pressure decreases as the abdomen is opened, necessitating simultaneous adjustments of the ventilator. Immediate aystole may occur upon opening the abdomen. Decompression of the abdomen results in acute, dramatic decrease in systemic vascular resistance resulting in an acute drop in blood pressure.[22] The second mechanism proposes a

reperfusion syndrome from the release of acid and metabolites of anaerobic metabolism from reperfused tissues. The effect of reperfusion injury can be attenuated by prior administration of Inj. Sodium bicarbonate.

After DL, a temporary abdominal closure is performed, followed by permanent abdominal closure at a later date. The fascia should not be closed primarily as this is associated with a high recurrence of ACS. Permanent abdominal closure is performed after hypovolemia, hypothermia, coagulapathy and acidosis have been corrected; which is usually 3 to 4 days after abdominal decompression.

FLUID SHIFTS

The volume of the fluid accumulated as transudate within the lumen, within the bowel wall and as transudation within the peritoneal cavity is very high. Because of the pressure built up within the gut, the proabsorptive fluxes become prosecretory. Thus water, electrolytes and proteins are translocated into the third space which is a nonfunctional extracellular fluid (ECF) compartment and is proportionate to the area of peritoneum. The volume of sequestration can be as large as 4 to 6 L/24 hrs. In the absence of proper fluid resuscitation, there is progressive dehydration, hypovolemia, hemoconcentration, renal insufficiency (oliguria and rising creatinine) leading to shock and death.

Normally, 7 L of fluid (saliva—1500 ml, gastric juice—2500 ml, bile—300 ml, pancreatic juice—750 ml, succus entericus—1000-3000 ml) are secreted daily into the upper gastrointestinal tract, most of which are absorbed so that only 400 ml pass the ileocecal valve. This is not possible in intestinal obstruction. Hence, accumulation of fluid occurs in obstructed bowel loop.

The volume of fluid lost could be as much as:

In early bowel obstruction	– 1500 ml
In well established obstruction with vomiting	– 3000 ml
Hypotension and tachycardia with circulatory insufficiency	– 6000 ml

Site of the obstruction

It is important to know the site of the obstruction[2] as the fluid and electrolyte abnormalities in bowel obstruction in each case varies.

Pyloric obstruction causes a loss of H^+ and Cl^- (and Na^+ and K^+) due to vomiting acidic gastric secretions. Alkaline pancreatic and duodenal secretions are retained and the result is a hypochloremic, hypokalemic, hyponatremic metabolic alkalosis.

Small bowel obstruction presents a different picture. Large volumes of fluid are lost (Na+, K+ and water) as the absorption of saliva, bile, gastric, pancreatic and duodenal secretions are impaired. The loss of a combination of alkaline intestinal secretions and acidic gastric secretions prevents the development of a metabolic alkalosis.

Large bowel obstruction: In this fluid loss tends to be less initially. Large bowel does not have secretary function. Therefore, not much water is accumulated in the gut.

In all the above situations if the obstruction is not relieved and intravenous fluid replacement does not take place, the combined effects of decreased fluid intake, vomiting, fluid loss into the bowel and peritoneum, bowel perforation, bowel ischemia, peritonitis, ketosis and sepsis leads to circulatory collapse and metabolic acidosis.

Thus, there is isotonic contraction of ECF volume causing acid-base disturbances and hypokalemia. Extracellular volume deficit may be considerable before clinical signs are apparent. Hematocrit rise is proportional to fluid loss. It is often helpful to crudely classify the extent of dehydration as 5, 10 or 15 percent rather than as mild, moderate or severe because this enables one to estimate roughly what the fluid deficit might be. A patient that is 5 percent dehydrated has lost 50 ml/kg of fluid and a patient that 10 percent dehydrated has lost 100 ml/kg of fluid, etc. This calculation serves as a useful starting point while prescribing rehydration fluid. It also depends on nature of surgical condition, duration of impaired fluid intake and presence and severity of symptoms associated with abnormal losses. Table 10.4 shows indices of extent of loss of ECF.[36]

Need of fluid resuscitation

The patient will need intravenous fluids to be administered to take account of the following:

Fluid deficit—As mentioned above, 5 percent dehydration is 50 ml/kg, 7.5 percent dehydrated then the deficit is 75 ml/kg and 100 ml/kg deficit with 10 percent dehydration.

Maintenance fluid—2000-3000 ml/day

Ongoing losses—initially difficult to quantify but includes NG loss and loss into bowel. A conservative estimate would be 2000 ml/day.

Therefore, the total fluid requirement for the first day is about 10 liters. Rehydration should therefore be started with 0.9 percent saline + 20 mmol/l KCl at

600 ml/hour for the first 8 hours. Before starting potassium, it is important to verify that the urine output is established and that the patient is not acidotic. Frequent reassessment will be necessary and the rate of fluid administration adjusted accordingly.[2]

Hemodynamic Effects

In healthy adults, compensatory mechanisms preclude a measurable decrease in BP until over 30 percent of the blood volume is lost. In general hypovolemia does not become apparent clinically until blood volume is reduced by at least 1000 ml (20% of blood volume). In elderly patients who have poor cardiac reserve and rigid vascular wall, signs of severe hypovolemia becomes evident with loss of 15 percent of blood volume. They exhibit less tachycardia for any degree of volume depletion as baroreceptor sensitivity decreases with age.

Diminished VR due to fall in circulating volume leads to a fall in CO, BP, O_2 transport and hence poor tissue oxygenation. This leads to development of metabolic acidosis. Expanding extracellular and intracellular compartments by volume resuscitation at this juncture can reverse further progress of acidosis. Table 10.5 shows clinical indices of extent of blood volume loss.[36]

Intestinal Strangulation

Whenever there is blockage of venous outflow and impairment of arterial supply, the strangulation occurs. This is a very dangerous condition and progresses rapidly with a mortality of 20 to 40 percent. Ileus of vascular obstruction is because of the immobility of strangulating bowel.

Effects of strangulation

There is increased motility of the gut in an attempt to push past the obstruction. Vomiting is frequent and bilious with higher levels of obstruction. There is subsequent extravasation of blood and fluid into the bowel wall followed by increased permeability of the bowel wall and loss of the red blood cells into the bowel cavity and peritoneal cavity. Patient is toxic with fever, tachycardia and distension.

After an initial period, regularly recurrent peristaltic bursts are seen interspersed with quiescent periods, duration of which depends on level of obstruction. In small intestine it is 3 to 5 minutes and with distal ileum it is 15 to 20 minutes. With strangulation it becomes severe and steady. These muscular contractions are traumatic to the bowel. After an initial surge, due to an inhibitory reflex, the bowel distal to the obstruction becomes progressively quiet. Hyperperistalsis results in diffuse, poorly localized, crampy abdominal pain. In strangulation, it is severe pain without any quiescent period. Whereas, ileus is not painful but results in generalized discomfort from distension.

Table 10.4 : Extent of ECF loss[36]

% of body wt. lost as water	*fluid lost (ml/70 kg)*	*Signs, symptoms*
>4% (mild)	>2,500	Thirst, decreased skin elasticity, decreased IOP, dry tongue, decreased sweating
>6% (mild)	>4,200	Orthostatic hypotension, decreased filling of peripheral veins, oliguria, nausea, decreased CVP, apathy, hemoconcentration
>8% (moderate)	>5,600	Hypotension, thready pulse, cool peripheries
10-15% (severe)	>7,000-10,500	Coma, shock, death

Table 10.5 : Clinical indices of extent of blood volume loss[36]

Class of hypovolemia	*1-minimal*	*2-mild*	*3-moderate*	*4-severe*
Blood volume lost (%)	10	20	30	>40
Volume lost (ml)	500	1000	1500	>2000
HR	N	100-120	120-140	>140, thready
BP	N	Orthostatic hypotension	<100 SBP	<80 SBP
Urine (ml/hr)	N	20-30	10-20	Nil
Sensorium	Alert	Anxious	Drowsy but restless	Impaired consciousness
Peripheral circulation	N	Cool and pale	Cold, pale, decreased capillary refill	Cyanosed
If left untreated	Progress to mild	Progress to moderate	Shock syndrome	Agitation, coma

Obstipation

Patient may spontaneously pass feces and flatus soon after obstruction as a part of bowel's hyperperistaltic surge, thereby evacuating distal segment. This presents as cramping followed by bouts of explosive diarrhea.

Systemic effects of absorption of bacteria and bacterial products:

Normal mucosa of the bowel is impermeable to bacteria and toxins produced by the bacterial degradation. But if blood supply is impaired as in case of strangulation the permeability is affected causing

absorption of toxic materials from peritoneum and transmucosal migration of bacteria. This leads to septic shock.

Ileus[1]

Ileus is caused by impaired intestinal motility and is characterized by symptoms and signs of intestinal obstruction in the absence of a lesion causing mechanical obstruction. Ileus is temporary and generally reversible if the precipitating factor can be corrected.

Etiology

- Abdominal operations
- Infection and inflammation
- Electrolyte abnormalities
- Drugs
- Inflammatory response- mediator release
- Anesthetic and analgesic effects, each of which can inhibit intestinal motility.

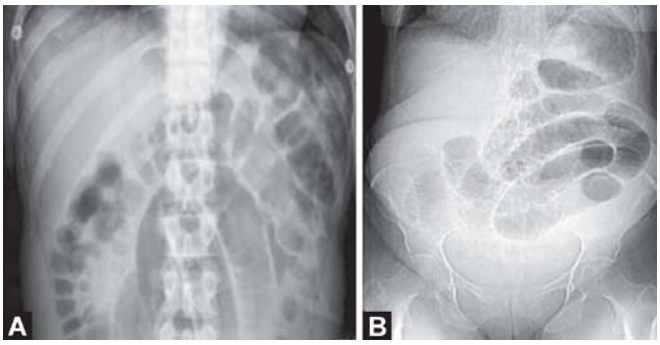
Pathophysiology

Clinical Presentation:
The clinical presentation of ileus resembles that of small-bowel obstruction.

- Inability to tolerate liquids and solids by mouth, nausea, and lack of passage of flatus or bowel movements are the most common symptoms
- Vomiting and abdominal distension may occur
- Bowel sounds are characteristically diminished or absent, in contrast to the hyperactive bowel sounds that usually accompany mechanical small-bowel obstruction. The clinical manifestations of chronic intestinal pseudo-obstruction include variable degrees of nausea, vomiting, abdominal pain, and distension.

Diagnosis

Routine postoperative ileus should be expected and requires no diagnostic evaluation. If ileus persists beyond 3 to 5 days postoperatively or occurs in the absence of abdominal surgery, diagnostic evaluation to detect specific underlying factors capable of inciting ileus and to rule out the presence of mechanical obstruction is warranted. Drugs known to be associated with impaired intestinal motility should be avoided. Measurement of serum electrolytes may demonstrate hypokalemia, hypocalcemia, hypomagnesemia, hypermagnesemia, or other electrolyte abnormalities commonly associated with ileus. Abdominal radiographs are often obtained, but the distinction between ileus and mechanical obstruction may be difficult based on X-ray abdomen alone. In the postoperative setting, CT scanning is the test of choice because it can demonstrate the presence of an intra-abdominal abscess or other evidence of peritoneal sepsis that may be causing ileus and can exclude the presence of complete mechanical obstruction.



Figs 10.3A and B: Plan X-ray abdomen showing triad of dilated small-bowel loops (>3 cm in diameter) air-fluid levels and a paucity of air in the colon' in supine (A) and in upright positions (B)

Treatment

The management of ileus consists of limiting oral intake and correcting the underlying precipitating factor. If vomiting or abdominal distension are prominent, the stomach should be decompressed using a nasogastric tube. Fluid and electrolytes should be administered intravenously until ileus resolves. If the duration of ileus is prolonged, TPN may be required. Prokinetic agents, such as metoclopramide are associated with poor efficacy. Cisapride has been associated with palliation of symptoms; however, because of cardiac toxicity and reported deaths, the use of it is restricted.

Differential Diagnosis

Conditions mimicking small bowel obstruction include.[37]

- Diabetic ketoacidosis
- Sickle crisis
- Porphyria
- Pancreatitis
- Ureteral and biliary colic
- Food poisoning
- Pseudo-obstruction.

Diagnosis

- **Plain X-ray abdomen:** In supine and upright positions, it shows triad of 'dilated small-bowel loops (>3 cm in diameter), air-fluid levels and a paucity of air in the colon' (Figs 10.3A and B).

- **USG**
- **CT scan abdomen (Fig. 10.4)**
  In intestinal obstruction there is discrete transition zone with dilatation of bowel proximally, constriction of bowel distally, intraluminal contrast that does not pass beyond the transition zone and a colon containing little gas or fluid. Strangulation is suggested by thickening of the bowel wall, pneumatosis intestinalis (air in the bowel wall), portal venous gas, mesenteric haziness, and poor uptake of intravenous contrast into the wall of the affected bowel. A limitation of CT scanning is its low sensitivity in the detection of low-grade or partial small-bowel obstruction.

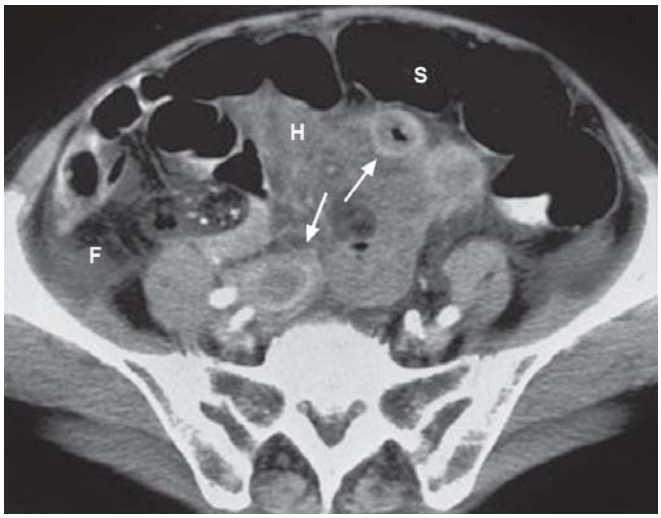


Fig. 10.4: CT scan abdomen

Anesthetic Management

Patients coming for emergency surgery can present with:

- Unclear surgical diagnosis
- Unevaluated or poorly optimized patient
- Unavailable history and previous medical record
- Untreated or uncontrolled preoperative medical conditions
- Risk of full stomach and regurgitation and vomiting
- Hypovolemia and hemorrhage
- Presence of acid base and electrolyte imbalance
- Renal impairment.

Preoperative assessment and optimization

The objective of emergency anesthesia is to permit correction of surgical pathology with minimum risk to the patient. Though it requires thorough preoperative workup guided by patient history and physical examination, the time available to achieve this is very limited.

Small-bowel obstruction is usually associated with a marked depletion of intravascular volume caused by decreased oral intake, vomiting and sequestration of fluid in bowel lumen and wall. Therefore, volume resuscitation should be done early during the course of treatment.

Ascertain the likely surgical diagnosis, the urgency of surgery, the expected duration and magnitude of surgery. Following points need special attention in addition to the surgical pathology:

1. Presence of co-morbid conditions.
2. Cardiovascular reserve.
3. A swift but thorough airway assessment in view of rapid sequence induction.

Preoperative Investigations

Laboratory investigations include:

- Complete blood count (Hb, hematocrit, total WBC count, differential count)
- Blood sugars
- Bun, serum creatinine, serum electrolytes
- Liver function tests
- X-ray chest, USG abdomen
- ECG
- Arterial blood gases needs to be done.

Though these investigations being most operative management can be started in emergency after blood collection, so that reports can be obtained at the earliest. Simultaneously volume resuscitation and correction of electrolyte imbalance can be started.

Broad-spectrum antibiotics are commonly administered because of concerns that bacterial translocation may occur in the setting of small-bowel obstruction.

Stomach decompression: The stomach should be continuously evacuated of air and fluid, using a nasogastric (NG) tube, to decrease nausea and distension before induction of anesthesia to reduce respiratory and circulatory derangements in addition to prevent the risk of vomiting and aspiration of gastric contents. There are two types of tubes available for abdominal decompression: short tubes placed in the stomach and long tubes placed in the small intestine.

Although gastric and intestinal tubes are employed to relieve abdominal distension, they do not function solely as definitive therapy for bowel obstruction except in postoperative ileus, partial small bowel obstruction and intestinal obstruction resulting from inflammation that is expected to subside with conservative therapy. For all other bowel obstructions, the mainstay of therapy remains surgical intervention. Percutaneous

fluid drainage (paracentesis) appears to be a superior alternative to DL in patients where anesthesia and surgery will pose unacceptably high risk to the patient. It may be performed quickly at bedside and avoids potential complications associated with larger incisions. It also serves to reduce the IAP and recover the organ function. The patient then can be taken up for definitive surgery later on once stabilized.

Fluid resuscitation: Primary goal of fluid management is the initial restoration of intravascular volume to a state of normovolemia. It is an integral part of treatment. In emergency situations, it may not be always possible to completely correct the deficit, but depending on the time available one must try to correct by rapid fluid replacement. This is guided by the central venous catheter monitoring. Insertion of a pulmonary artery catheter is advocated in patients with cardiac disease. However, the risk of inserting and maintaining the catheter and its probable benefit should be weighed. An indwelling bladder catheter is placed to monitor urine output. Monitoring urine output ensures early detection of hypovolemia, inadequacy of CO or mounting IAP.

Underestimated or unrecognized hypovolemia may lead to circulatory collapse during induction of anesthesia which attenuates the sympathetically mediated increases in arteriolar and venous constriction. In any patient in whom the fluid is sequestrated or lost or in whom hemorrhage has occurred, quantify the blood volume and correct the deficit.

Indications to insert a central venous line include:

- To guide fluid therapy especially in the elderly and those with impaired cardiac function
- To give inotropes if necessary
- To aspirate mixed venous blood and determine its oxygen saturation. Patients with a mixed venous oxygen saturation of less than 70% have a high global oxygen extraction due to a low oxygen delivery compared to oxygen consumption. They may benefit from attempts to increase oxygen delivery with further fluids and inotropes. An acidosis that persists in patients with a high mixed venous oxygen saturation (implying an adequate oxygen delivery) may be due to renal failure or ischemic bowel as seen in the late stages of septic shock and is due to microcirculatory abnormalities or due to cells that are unable to utilize the oxygen supplied to them.[2]

Choice of fluid replacement depends on:

- Nature of loss: This will essentially depend on the site of obstruction in the gut
- Hemodynamic status: Patient in shock will have to be aggressively treated by volume. Blood is sent for cross-match (4 units) and coagulation tests
- Co-morbidities in the patient:

These patients have fluid loss, electrolyte loss, protein loss and blood loss. A decrease in HR, increase in BP and improvement in peripheral perfusion are the objectives of fluid therapy. Each case should be treated on individual merit.

Patients with pyloric obstruction will have mainly loss of gastric juice that contains acid, i.e. H^+ , Cl^-, Na^+ and K^+ and is best corrected by isotonic solutions like 0.9 N saline with K^+ added. The intestinal fluid beyond pylorus resembles that of ISF and should be replaced with balanced salt solution like Ringer's lactate.

The fluid therapy should be guided by the central venous catheter and its efficacy should be decided by the parameters of peripheral perfusion such as, improvement in the mean arterial blood pressure, capillary perfusion, urine output (at least 30 to 50 ml/hr), orthostatic hypotension and decrease in the signs of dehydration such as dryness of mucous membrane and skin turgor.[1,38] It is prudent to initiate fluid therapy in small portions. The response to repeated fluid boluses (fluid challenge) is assessed. A sudden rise in CVP without any subsequent increase in the BP should caution the physician about the myocardial dysfunction. At this point, the strategy should be to reduce the rate of volume infusion, addition of an inotrope and administration of diuretics. Judicious use of vasopressors is required to maintain the cardiac output when fluid resuscitation does not serve the purpose. The drug of choice would be dopamine, noradrenaline, or dobutamine depending on the need of each patient. A colloid also can be used as this will substantially reduce the need for crystalloids. Blood should be used when it is available and if it is required.

Acidemia can potentiate the depressant effects of most sedatives and anesthetic agents on the central nervous and circulatory systems. Because most opioids are weak bases, acidosis can increase the fraction of the drug in the nonionized form and facilitate penetration of the opioid into the brain. Increased sedation and depression of airway reflexes may predispose to pulmonary aspiration. The circulatory depressant effects of both volatile and intravenous anesthetics can also be exaggerated. Moreover, any agent that rapidly decreases sympathetic tone can potentially allow unopposed circulatory depression in the setting of acidosis. Halothane is more arrhythmogenic in the presence of acidosis. Succinylcholine generally needs to be avoided in acidotic patients with hyperkalemia to

prevent further increases in plasma [K^+]. Lastly, respiratory—but not metabolic—acidosis augments nondepolarizing neuromuscular blockade and may prevent its antagonism by reversal agents.[39]

Replacement of blood is required to establish hemodynamic stability in surgical patients with blood loss for proper tissue oxygenation and to cope up with possible operative blood loss. This ensures proper post-operative healing and recovery.

Correction of electrolyte imbalance[40]

- Sodium requirement to correct hyponatremia to a safe level of at least 120 to 125 mEq/L is calculated. The targeted rate of correction should not exceed 0.5-1 mEq/L/hr. In such cases, the most commonly used fluid is 0.9 percent NaCl (154 mEq Na/L). Hypochloremia usually gets corrected with it.

  Na requirement = (Desired Na-Actual Na) × Total body water

  Total body water = 0.6 × body wt. in kgs

  If the patient is very hypernatremic (Na^+ >155 mmol/ l) rehydration should be over 48 hours with half strength saline because of the risk of cerebral edema.[2] Administration of free water through Ryle's Tube may not be possible in the setting of intestinal obstruction.
- Intravenous KCl preparations are usually used to correct hypokalemia. Potassium deficit needs to be corrected slowly over a period till S. K^+ reaches to at least 3 to 3.5 mEq/L. The rate should not exceed 0.5 mEq/Kg/hr. Before adding K^+ to any fluid or giving K^+ containing fluids, one should ensure establishment of proper urine output and correction of acidosis as both these conditions can lead to dangerous hyperkalemia.
- Acidosis if metabolic should be corrected with $NaHCO_3$, calculated as follows:

  $NaHCO_3$ required = 0.3 × body wt. × base deficit. Half of the required is given as IV bolus initially followed by remaining half after repeating the ABG. Acidosis is mainly metabolic in nature and gets corrected when the peripheral perfusion improves. Administration of $NaHCO_3$ without giving attention to improving the peripheral perfusion may not be a viable policy. Respiratory acidosis rarely occurs as patient is tachypneoic because of the pain and sepsis. However, the breathing may be shallow because of abdominal distension. Highly tachypneic and gasping patients need intubation and ventilatory support preoperatively to optimize respiratory parameters.

In severe deficit, the initial rate may be as high as 1000 ml/hr, reducing the rate as condition improves. Elderly and cardiac patients require careful correction with monitoring. Dextrose should be given in hypoglycemic patients with frequent monitoring of blood glucose levels. In addition there must be ongoing modifications in the management of fluid and electrolytes to achieve the physiologic goals.

Once the above treatment has been commenced the surgeon and anesthesiologist should decide together whether the patient is in an optimum condition to undergo the operation. Ideally the patient would go to theater when fully resuscitated and with his deficit corrected but surgical urgency may dictate otherwise. Patients with necrosed or perforated bowel will need to proceed to operation as early as possible and it may not be possible to rehydrate them fully before operation. A reasonable compromise in this case may be to plan to do the laparotomy after 8 to 10 hours of resuscitation when hopefully the shock and anemia will have been corrected and the patient would have received adequate rehydration fluid. Other factors that should be considered are the need for thromboembolism prophylaxis and antibiotics. If inotropic support has been started preoperatively, patient is shifted to OT along with it. After establishing adequate monitoring with cardioscope, pulse-oximeter, blood pressure apparatus, foley's catheter for urine output, two large bore intravenous lines are taken. CVP is monitored with indwelling central venous line.

Immediate preoperative period:

- Preoperative antibiotics
- Anticholinergics (glycopyrrolate IV/IM) to protect the heart from the potent vagal stimulation associated with the use of vagomimetic drugs and endotracheal intubation. In those patients with pre-existing tachycardia and hyperthermia, atropine or glycopyrrolate is avoided[1]
- Oxygen by mask or by nasal prongs
- NG tube aspiration and anti-aspiration prophylaxis: this is achieved with
  - Antacids like ranitidine IV to increase the pH of gastric contents and to reduce the gastric volume
  - Serotonin inhibitor, ondansetron IV to reduce vomiting.

However, because of the large volumes of fluid sequestrated in the bowel, antacids and/or H_2 blockers have little or no value in high mechanical intestinal obstruction compared to partial small bowel obstruction.[38]

- Since these individuals may have diminished respiratory reserve because of distended abdomen, any premedication that depresses respiratory drive will diminish the ability of the patient to breath. This will exaggerate the pre-existing hypoxia and

hypercarbia. It will also predispose the patient to aspiration of the gastric contents. Hence, sedative and narcotic premedication is avoided[1]

- Although the patient may be in considerable pain from abdominal distension, narcotic analgesics should be avoided.

Induction

Problems at induction

The most dreaded complications during induction and emergence of anesthesia are vomiting, regurgitation and aspiration of its contents into the tracheobronchial tree as well as hemodynamic instability.

The factors that predispose the patients with intestinal obstruction to aspiration are:

1. Full stomach (absent or abnormal peristalsis)
   a. Peritonitis of any cause.
   b. Postoperative ileus.
   c. Metabolic ileus (hypokalemia, uremia, diabetic ketoacidosis).
   d. Drug induced ileus (antichlolinergics).
2. Obstructed peristalsis
   a. Small or large bowel obstruction.
   b. Gastric carcinoma.
   c. Pyloric stenosis.
3. Delayed gastric emptying
   a. Trauma.
   b. Shock of any cause.
   c. Fear, pain, anxiety.
   d. Deep sedation, coma, narcotics.
   e. Recent solid food intake.

All these, with reduced lower esophageal sphincter (LES) tone makes the patient more prone for aspiration. LES is an area of 2 to 5 cms in length with higher resting intraluminal pressure situated in the region of cardia and is responsible for preventing reflux into the esophagus. It cannot be defined anatomically but detected only by manometry. Reflux depends on the difference between gastric and LES pressure termed as 'barrier pressure'. Factors that increase the later will decrease the reflux. Antichlolinergic drugs, sedatives and narcotics, anesthetic agents reduce the barrier pressure.

The volume and pH of gastric contents and the presence or absence of particulate matter appear to be the most important factors determining the degree of pulmonary injury following aspiration. The classical term 'at risk' is thought to imply a gastric volume greater than 25 ml, with the pH less than 2.5. Approximately 40 percent of the emergency surgical patients have a gastric pH below 2.5.[38]

PLAN OF ANESTHESIA

After adequate preparation and monitoring, the NG tube should be aspirated and general anesthesia should be induced using a **rapid sequence induction (RSI)** technique with **cricoid pressure.**[2]

If the patient's coagulation status is normal an **epidural catheter** may help provide high quality postoperative pain control and will decrease the likelihood of postoperative pulmonary morbidity. It may be prudent not to use the epidural immediately, but rather to establish the block cautiously during the operation or use it exclusively for postoperative period. The use of epidural anesthesia combined with general anesthesia in a partially rehydrated, elderly patient having emergency surgery with the potential to develop a systemic inflammatory response and intraoperative fluid shifts may cause profound hypotension and therefore caution is essential.[2]

Spinal or epidural analgesia only is not advisable for acute intestinal obstruction because the level of block required is very high, reduction in the sympathetic tone that they produce may cause profound hypotension in the presence of inadequate circulating blood volume. For the patient with massive abdominal distension and high spinal block, breathing may be extremely difficult. Though it may be performed under intercostals or abdominal field block combined with a splanchnic block.[41]

Rapid Sequence Induction and Intubation (RSI)

It is the technique of choice for induction of general anesthesia in these patients. Requirements are:

- Tiltable table with facility of head low positioning.
- Good, dedicated suction machine with a big bore catheter
- Cuffed ETTs of appropriate size. If difficult intubation is anticipated, then difficult intubation cart is kept ready.

Plan of General Anesthesia

Monitoring must begin preoperatively prior to induction of anesthesia and includes ECG, SaO_2, BP manual/noninvasive, $EtCO_2$ and urine output. A large bore intravenous access is established.

- **Stomach decompression** is done by sucking NG tube. Though the stomach contents are reduced, it is not completely evacuated. Hence it is repeatedly emptied every 15 minutes before the operation. Even aspirating air reduces the risk of aspiration. Then NGT is withdrawn in the esophagus and left open to drain. Whether to remove or leave the NG tube in

place before anesthesia induction is controversial. Removal increases the likelihood of aspiration of gastric contents and leaving in place does not reliably ensure an empty stomach and renders the LES incompetent by preventing its complete closure.

- **Preoxygenation** for 3 minutes to allow denitrogenation and improve the plasma stores of oxygen. This helps in RSI as the patient is not ventilated during the period of apnea that follows the administration of muscle relaxants.
- **Choice of the induction agent** will depend on general condition of the patient, hemodynamic stability and specific risk factors. It can be thiopentone sodium, propofol or ketamine intravenously. Care should be taken not to administer the drug too rapidly to cause circulatory collapse in a patient who is less than adequately volume resuscitated. The dose should be carefully titrated to the level of unconsciousness. Propofol will prevent development of bronchospasm in patient with reactive airway disease when it is not possible to optimally prepare the patient. However, it has a potential to cause severe hypotension.
- The **muscle relaxant of choice** to facilitate the intubation in emergency cases and patients with full stomach, is suxamethonium because of its rapid onset and offset and good intubating conditions. If there is no evidence of hyperkalemia and no contraindication to its use, it should be given. Normally a physiological rise of 0.5 mEq/L in S, K^+ is seen. In patients with renal failure, spinal cord injuries, burns, closed abdominal infections, it is known to cause exaggerated hyperkalemic response and therefore, best be avoided. It causes muscle fasciculations and increase intragastric pressure. But since it also increases LES tone, there is no increased risk of regurgitation after its use. When it is contraindicated, rocuronium is a suitable alternative as in doses of 0.9 mg/kg it provides intubating conditions within 60 to 90 seconds. Or else one may use NMBAs using priming principle.[42] Intubation can also be accomplished using propofol alone provided cardiovascular stability is maintained. The cricoid pressure should not be released unless the endotracheal tube (ETT) is confirmed to be in the trachea and the cuff is inflated.
- **Position for intubation:** Head low position promotes passive regurgitation. However, if massive reverse peristalsis and regurgitation occurs, vigorous suctioning and the head-down position (must be at least 10%) are the best methods to prevent soilage of the tracheobronchial tree.[43] Vomiting is an active process and requires contraction of the abdominal muscles and lighter planes of anesthesia.
- **Cricoids pressure (Sellick's maneuver)**: As soon as the patient loses consciousness, the trained assistant should apply a firm backward pressure on the patient's cricoid cartilage, at the same time stabilizing the cervical spine, to prevent regurgitation of stomach contents into the pharynx and inflation of the stomach by gases. Any amount of airway obstruction will cause the gases to take the path of least resistance into the stomach causing its inflation, increase in intragastric pressure, splinting of diaphragm, reduced ventilation and regurgitation. As soon as the ETT is placed under vision, the cuff of the tube should be inflated and then ventilation is started. The cricoids pressure then can be safely removed.
- **Maintainance of anesthesia** can be with oxygen, nitrous oxide, intermittent short acting narcotics or inhalational agents and NMBAs. The choice of the latter should depend on patient's renal, cardiac and hepatic functions. The use of nitrous oxide is again controversial since its administration is associated with an undesirable increase in intraluminal gas volume and pressure[43] that may lead to detrimental consequences. The blood-gas partition coefficient of nitrous oxide is 34 times that of nitrogen. Therefore, nitrous oxide in the blood can enter gas filled cavities 34 times more rapidly than nitrogen can leave those cavities to enter the blood. Thus, increased intraluminal pressure associated with nitrous oxide administration may lead to bowel ischemia and necrosis. It can also cause difficulties with abdominal closure at the conclusion of surgery. Therefore, anesthesia should be maintained with oxygen, air and a volatile agent and increments of opioids and muscle relaxants as needed.[44] Gastric distension though could be manageable with the help of Ryle's tube.
- **Intraoperative hypothermia prevention** can be done by the use of fluid and air warmers and a warming mattress.
- **Intraoperatively,** rapid decompression of the peritoneal cavity may cause sudden cardiovascular collapse. Malignant dysrrhythmias on decompression, secondary to sudden efflux of products of anaerobic metabolism from the abdomen which is prevented by volume resuscitation and gradual decompression.
- Intraoperative fluid requirements include correction of the preoperative deficit, maintenance fluids and replacement of ongoing loss such as blood loss, 'third space' loss and fluid loss outside the body.

(vomiting, urine output, RT aspirate). 'Third space' loss is the loss of extracellular fluid that occurs during a laparotomy due to the trauma, manipulation, resection and retraction of the abdominal contents. Since it is abdominal surgery with exposed large surface area of bowels and severe surgical trauma, the maintenance requirements should be 10 ml/kg/hr. Balanced salt solution such as Ringer's lactate is used to replace the intraoperative fluid losses since it is the most physiological fluid with composition similar to plasma. Isotonic saline also can be used when Ringer's lactate is contraindicated or when large volumes are to be replaced. Five percent dextrose can be used to provide calories as starvation fluid. Rapid replacement with dextrose can cause hyperglycemia and glycosuria causing osmotic diuresis. Since it is a hypotonic solution, massive resuscitation with dextrose alone will lead to cellular edema. Preoperative edema and fluid overload will mandate careful fluid replacement and use of colloids. Colloids will help to maintain the intravascular volume for a longer period with minimum fluids. Blood and blood products are given if required. Measurement of pulse rate, CVP and urine output will help to guide fluid therapy intraoperatively. An esophageal doppler monitor provides an additional noninvasive measure of cardiovascular output and filling and may be used if available.

- **At the end of surgery**, a decision to reverse or electively ventilate the patient will depend on his general condition, abdominal distension, other co-morbid conditions and breathing efforts. It is always beneficial to electively ventilate the hypothermic, septic and hemodynamically unstable or very acidotic patient to reduce work of breathing and improve the oxygenation in an intensive care unit. The other factor that is important is the development of intraabdominal hypertension as a result of the obstruction, volume resuscitation, surgical dissection, continued blood loss in the abdominal cavity, etc. This will greatly increase the airway pressures and work of breathing. Development of adult respiratory syndrome (ARDS) and multiorgan failure need to be closely watched for, investigated and treated thoroughly. This is discussed in detail in the chapter on 'Abdominal Sepsis'.
- If the patient is not in sepsis, diagnosed and operated well in time, the pathology corrected, does not have any fluid, electrolyte or acid base imbalance, without any pre-existent co-morbid conditions then he can be safely extubated. However, postoperatively he would need good analgesia that enables him to cough and breathe deeply and chest physiotherapy will help to prevent pulmonary atelectasis and infection. The analgesia is best provided by means of indwelling epidural catheter after ruling out sepsis. NSAIDs should be used with caution because of their propensity to precipitate acute tubular necrosis in elderly, dehydrated patients.
- **Postoperative care:**[1,38] The management principles remain the same as in the pre and intraoperative period. A bed in the high dependency unit, is the most appropriate place to care for this patient.

One has to take care of:

- Gastrointestinal decompression
- Oxygenation and ventilation
- Hemodynamic stabilization
- Infection
- Nutrition.

Indications for Continuation of Ventilator Assistance Postoperatively

- Severe sepsis
- Uncontrolled co-morbid conditions, e.g. recent MI, accelerated hypertension, uncontrolled diabetes
- Unstable hemodynamic condition
- Intra-abdominal hypertension with insufficient ventilation
- Risk of pulmonary aspiration of gastric contents
- Obesity—hypoventilation
- Prolonged shock/hypoperfusion state of any cause
- Severe ischemic heart disease
- Overt gastric acid aspiration
- Previously severe pulmonary disease and other co-morbid conditions.
- Nutritional support is very important in these patients because of the period without adequate nutritional intake preoperatively and because of the catabolic effect of sepsis and surgical trauma. NG feeding should be started as soon as the surgical procedure allows after return of the peristalsis and after ruling out any anastomotic leak.
- Postoperative oxygen, fluids, DVT prophylaxis and antibiotics should be prescribed as indicated.

In the immediate postoperative period, there continues to be a significant fluid loss, mostly secondary to third spacing which gradually diminishes over time. Usually by about the third postoperative day it reverses in direction as fluid goes back into the vascular compartment. This significant autoinfusion must be

accounted for in the computation of the daily fluid requirements of the patient. Otherwise congestive failure may follow, especially because patients with intestinal obstruction are usually older adults with limited reserve in several organ systems. As electrolyte loss continues postoperatively, serial determinations of serum sodium and potassium levels may be needed. Hyponatremia and hypokalemia causes prolonged postoperative ileus. Because return of normal intestinal motility is usually prolonged after surgical relief of bowel obstruction, abdominal decompression must often be continued for 5-6 days postoperatively, whereas bowel function returns after third postoperative day after a routine abdominal operation.

Finally, postoperative management should also include frequent monitoring of hemodynamic parameters, hemoglobin concentration and urine output. On second postoperative day, the blood investigations are sent. They include:

- Hemoglobin and the hematocrit
- Blood chemistry for liver and renal function
- Serum electrolytes
- Coagulation profile
- Serial arterial blood gases to assess the breathing adequacy or the ventilator settings.

Complications

1. Respiratory problems
   - Hypoventilation due to significant abdominal distension
   - Hypoxia and hypercarbia because of residual effects of anesthetic agents and sedatives
   - Reduction in lung volumes and capacities (TV, VC, FRC, RV, FEV_1)
   - Overzealous volume resuscitation
   - Pain.

   Hence, ETT may be left in place to decrease anatomical dead space and make it possible to ventilate the patient during the immediate postanesthesia period. This will also decrease residual atelectasis in basal portions of the lung which will decrease any pulmonary shunt and lessen the need for high inspired oxygen concentration (FIO_2). For those patients, not requiring ventilatory support, a T-piece attached to the ETT will increase the FIO_2 to maintain PaO_2 to acceptable level. As the patient gradually regains respiratory adequacy and ventilation returns to normal, extubation can be done safely.[37]
2. Chemical pneumonitis (Mendelson's syndrome) is caused by aspiration of gastric contents. It is characterized by hypoxemia, bronchospasm and atelectasis. It can progress to adult respiratory distress syndrome (ARDS) if treatment measures do not intervene. Treatment measures include improved oxygenation by use of positive end expiratory pressure in an intubated patient, lung condition monitoring by serial X-rays and comparing PaO_2 values with corresponding FIO_2 values on arterial blood gases, corticosteroid therapy, antibiotics.[43,45]
3. In addition, surgical complications such as anastomotic leak, wound gaping, retraction of stoma, gangrene of the bowel can occur that will have devastating effect on the postoperative course of the patient. Patient can be weaned off only when the surgical repair is intact.
4. Patient may develop sepsis and multiple organ dysfunction syndrome (MODS) depending on the initial insult, degree of invasion by the bacteria and the patient's immunity. This is discussed in detail in the following chapter.

REFERENCES

1. Whang EE, Ashley SW, Zinner MJ. Small intestine. In: Brunicardi FC, Schwartz's manual of surgery. 8th edn. New York: McGraw-Hill 2005;702-31. 1017-32.
2. Updates in Anaesthesia, Issue 17 (2003) Article 14. Available from: http://www.nda.ox.ac.uk/wfsa/html/u17/u1714_01.htm
3. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg 1984;199:28-30.
4. Iberti TJ, Kelly KM, Gentili DR, Hirsch S, Benjamin E. A simple technique to accurately determine intra-abdominal pressure. Crit Care Med 1987;15:1140-42.
5. Lacey SR, Bruce J, Brooks SP, Griswald J, et al. The relative merits of various methods of indirect measurement of intra-abdominal pressure as a guide to closure of abdominal wall defects. J Pediatr Surg 1987; 22:1207-11.
6. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med 1989;17:118-21.
7. Ivatury RR, Diebel L, Porter JM, Simon RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. Surg Clin North Am 1997;77:783-800.
8. Meldrum DR, Moore FA, Moore EE, Franciose RJ, et al. Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 1997;174:667-73.
9. Tiwari A, Haq AI, Myint F, Hamilton G. Acute compartment syndromes. Br J Surg 2002;89:397-12.

10. Nathens AB, Brenneman FD, Boulanger BR. The abdominal compartment syndrome *Can J Surg* 1997;40:254-62.
11. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 2006;32:1722.
12. Sanchez NC, Tenofsky PL, Dort JM, Shen LY. What is normal intra-abdominal pressure? Am J Surg 2001;67:243-48.
13. Cheatham ML, White MW, Sagraves SG, Johnson JL. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 2000;49:621-7.
14. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am. Aug 1996;76(4): 833-42.
15. Schiling MK, Redaelli C, Krähenbühl L, Signer C, Büchler MW. Splanchnic microcirculatory changes during CO_2 laparoscopy. J Am Coll Surg 1997;184:378-82.
16. Saggi BH, Bloomfield GL, Sugerman HJ, et al. Treatment of intracranial hypertension using nonsurgical abdominal decompression. J Trauma 1999;46:646-51.
17. Bloomfield G, Saggi B, Blocher C, Sugerman H. Physiologic effects of externally applied continuous negative abdominal pressure for intra-abdominal hypertension. J Trauma 1999;46:1009-14.
18. Richards WO, Scovill W, Shin B, Reed W. Acute renal failure associated with increased intra-abdominal pressure. Ann Surg 1983;197:183-7.
19. Bendahan J, Coetzee CJ, Papagianopoulos C, Muller R. Abdominal compartment syndrome. J Trauma 1995; 38:152-3.
20. Reeves ST, Pinosky ML, Byrne TK, Norcross ED. Abdominal compartment syndrome. Can J Anaesth 1997; 44:308-12.
21. Celoria G, Steingrub J, Dawson JA, Teres D. Oliguria from high intra-abdominal pressure secondary to ovarian mass. Crit Care Med 1987;15:78-9.
22. Shelly MP, Robinson AA, Hesford JW, Park GR. Haemodynamic effects following surgical release of increased intra-abdominal pressure. Br J Anaesth 1987;59:800-05.
23. Paula R. Compartment Syndrome, Abdominal: overview [article on the Internet]. [cited Feb 23, 2009]; Available from: http://emedicine.medscape.com/article/829008
24. Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. J Trauma 1995;39:1071-75.
25. Benumof JL (Ed). Anasthesia and uncommon diseases, 4th edn. Philadelphia: WB Saunders 1998;205.
26. Bloomfield GL, Blocher CR, Fakhry IF, Sica DA, Sugerman HJ. Elevated intra-abdominal pressure increases plasma renin activity and aldosterone levels. J Trauma 1997; 42:997-1005.
27. Chang MC, Miller PR, D'Agostino RJ, Meredith JW. Effects of abdominal decompression on cardiopulmonary function and visceral perfusion in patients with intra-abdominal hypertension. J Trauma 1998;44:440-5.
28. Ivatury RR, Porter JM, Simon RJ, et al. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: Prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 1998;44:1016-21.
29. Diebel LN, Dulchavsky SA, Brown WJ. Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. J Trauma 1997;43:852-5.
30. Diebel LN, Dulchavsky SA, Wilson RF. Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 1992;33:45-9.
31. Bongard F, Pianim N, Dubecz S, Klein SR. Adverse consequences of increase intra-abdominal pressure on bowel tissue oxygen. J Trauma 1995;39:519-25.
32. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. J Trauma 1996;40:936-43.
33. Bloomfield GL, Dalton JM, Sugerman HJ, Ridings PC, DeMaria EJ, Bullock R. Treatment of increasing intracranial pressure secondary to the acute abdominal compartment syndrome in a patient with combined abdominal and head trauma. J Trauma 1995; 39:1168-70.
34. Yol S, Kartal A, Tavli S, Tatkan Y. Is urinary bladder pressure a sensitive indicator of intra-abdominal pressure? Endoscopy 1998;30:778-80.
35. Johna S, Taylor E, Brown C, Zimmerman G. Abdominal compartment syndrome: Does intra-cystic pressure reflect actual intra-abdominal pressure? A prospective study in surgical patients. Crit Care 1999;3:135-8.
36. Turner DAB. Emergency Anaesthesia. In: Alan Aitkenhead, Graham Smith, David J Rowbothom (Eds). Textbook of Anaesthesia. 5th edn. UK: Churchill Livingstone, Elservier 2007; 540-53.
37. Slodzinski M, Merritt WT. Anesthesia for gastrointestinal Surgery. In: Longnecker DE, Brown DL, Newman MF, Zapol WM (Eds). Anesthesiology. New York: Mc Graw Hill 2008;1314-37.
38. Kaye AD, Riopelle JM. Intravascular Fluid and Electrolyte Physiology. In: Miller RD (Ed). Miller's anesthesia, 7th edn. New York: Churchill Livingstone 2010;1705-38.
39. Acid base balance. In: Morgan GE Jr, Mikhail MS, Murreay MJ (Eds). Clinical Anesthesiology. 4th edn. Boston: Mc Graw Hill 2008;708-24.
40. Fluid and electrolyte disorders. In: Pandya S (Ed). Practical guidelines on fluid therapy. 2nd edn. Rajkot: Pandya 2005, reprint 2008;56-127.
41. Kaufman L. Anaesthesia for abdominal surgery. In: Gray TC, Utting JE, Nunn JF (Eds). General anaesthesia, 4th edn. United Kingdom: Butterworth and Co 1980;1431-14528.

42. Wliiiam JS, Peter S. Rapid induction-intubation for prevention of gastric content aspiration. Anesthesia and analgesia 1970;49(4):633-6.
43. Barash PG, Cullen BF, Stoelting RK (Eds). Clinical anesthesia, 5th edn. Philadelphia: Lippincott Williams and Wilkins 2006;1393-5.
44. Gerald BH Lewis. Intestinal distension during nitrous oxide anesthesia. Canad. Anasth. Soc J 1975;22(2):200-01.
45. Salem MR. Anesthetic management of patients with a full stomach 'a critical view.' Anesthesia and analgesia. 1970; 49(1):47-55.

11 Anesthesia for Abdominal Sepsis

Amala G Kudalkar

KEY POINTS

- Sepsis is the major cause of perioperative morbidity and mortality in patients admitted to ICU.
- **Systemic inflammatory response syndrome** is the body's response to a variety of severe clinical insults such as trauma, infection, burns, etc. **Sepsis** is the development of systemic inflammatory response syndrome in response to infection.
- It is a procoagulant and proinflammatory state with multiple organ dysfunction and failure as a result of hypoperfusion and hypoxia.
- Abdominal infection forms a major etiological subset of sepsis. It can be in the peritoneal cavity, in the retroperitoneum or localized within the viscera, the most common site being intraperitoneal infection (peritonitis).
- The most common cause of peritonitis is inflammation followed by perforation of a portion of gut or reproductive system. Another important cause is abdominal trauma.
- Normal intestinal mucosa is impermeable to bacteria and their toxins. In low output and low immunity states, there is transmucosal, transperitoneal and transdiaphragmatic migration of bacteria causing infection.
- Postoperative peritonitis occurs as a result of infection, ischemia, foreign body and hemorrhage.
- The result of the bacterial invasion will depend upon the degree of contamination, the virulence of bacteria and the immunity of the host.
- Host responds by both blood born and cellular immunity to the causative agent. This leads to a widespread inflammatory response involving many inflammatory mediators and causing activation of complement system.
- Inflammation of peritoneum causes exudation of a large quantity of fluid into the peritoneal cavity. This, along with the depressive action of mediators on myocardium causes, significant hemodynamic instability.
- There is increase demand of oxygen, altered oxygen extraction and altered oxygen transport.
- It affects all systems of the body causing acute lung injury, renal failure, coagulopathy, myocardial depression, severe hypotension, gut ischemia and a dynamic ileus. Almost all organ systems of the body are involved leading to multiorgan failure (MOF).
- The prognosis is best indicated by the APACHE II score.
- The anesthesiologist is involved in preoperative resuscitation, administration of anesthesia as well as postoperative hemodynamic and ventilatory support.
- The patient should be resuscitated to a predefined early goal directed therapy and surviving sepsis guidelines should be bollowed.
- Crystalloids are as effective as the colloids and should be started early in the course of sepsis. Vasopressors are started when blood pressure does not respond to intravenous fluids.
- Inotropes are added to improve the cardiac output.
- Noradrenaline or dopamine are the drugs of first choice.
- Ventilation should be carried out using ARDSnet protocol- avoid volutrauma, barotauma and atelectrauma.
- Low tidal volumes, application of PEEP and avoiding excessive airway pressure.
- Renal replacement therapy if required.
- All systems should be supported till the sepsis resolves.

Sepsis is the second most common disease in the ICU, occurring in 30 to 40 percent of ICU admissions. Septic shock accounts for around 10 to 15 percent of intensive care admissions[1] with mortality between 50 and 60 percent. The major cause of death is multiple organ failure syndrome as a result of regional and systemic hypoperfusion and hypoxia. Abdominal sepsis forms a major subset of patients admitted in surgical ICU. Abdominal sepsis has problems unique to it. For optimum management of patient with abdominal sepsis, one has to know the normal defenses of the gut, genesis of sepsis in abdomen, the pathophysiology of sepsis, its clinical presentation and the guidelines for its management.

What is Sepsis?

To know the exact answer to this question, lets get familiarized with certain terminology.

Systemic inflammatory response syndrome[2,3] (SIRS): It is the body's response to a variety of severe clinical insults such as trauma, infection, burns, pancreatitis, etc. It is characterized by the presence of two or more of the following factors under appropriate circumstances:

- Temperature > 38.3°C or < 36°C
- Tachycardia: HR > 90 beats/min
- Tachypnea: RR > 20/min, $PaCO_2$ < 32 mm Hg or < 4.3 kpa
- WBC > 12,000/cmm (leukocytosis) or < 4,000/cmm (leukopenia).

Bacteremia:[2] It is the presence of bacteria in the blood cultures without systemic signs of infection.

Sepsis:[2-5] It is defined as infection plus the systemic manifestations of infection. In other words, it is the inflammatory response by the host to an infectious insult. It is characterized by a procoagulant and proinflammatory state and is frequently accompanied by organ dysfunction resulting from organ hypoxia and hypoperfusion. In addition to the criteria mentioned above, they have:

- Altered mental status.
- Significant edema with a positive fluid balance of > 20 ml/kg over 24 hours.
- Hyperglycemia with plasma glucose > 140 mg/dl in absence of diabetes.
- Plasma C-reactive protein > 2 SD above the normal value.
- Plasma procalcitonin > 2 SD above the normal value.

Severe sepsis:[2,3] It is associated with one or more organ dysfunction, hypoperfusion or hypotension.

- **Hypotension** is said to occur when the systolic BP is less than 90 mm Hg or MAP < 70 mm Hg or a decrease in SBP of more than 40 mm Hg or greater than 2 SD than normal.
- **Hypoperfusion** can be recognized by:[4]
  - The levels of the lactate more than 2.5 mmols/dl or more than 1.5 times the normal laboratory levels.
  - pH of ≤ 7.3 or a base deficit of ≥ 5.0 mEq/l
  - Decreased capillary refill or mottling.
- **Organ dysfunction[3]** is said to occur when:
  - Arterial hypoxemia with PaO_2/FiO_2 < 300
  - Oliguria < 0.5 ml/kg/hr for at least 2 hours despite adequate fluid therapy
  - Creatinine increase of > 0.5 mg/dl
  - Platelet count < 1,00,000/cmm
  - INR > 1.5
  - Plasma bilirubin > 4 mg /dl
  - Absent bowel sounds (ileus).

Septic shock:[3] It is said to occur when there is sepsis induced hypotension for at least 1 hour despite adequate fluid resuscitation or need for vasopressors to maintain normal blood pressure (SBP > 90 mm Hg).

Shock which is refractory to intravenous therapy and inotropic and vasoactive drugs within 1 hour of treatment is called as **Refractory Shock**.

MODS describes a state where dysfunction is seen in more than one organ requiring intervention to maintain homeostasis.

Some patients may present preoperatively with severe sepsis and need ICU care and resuscitation prior to surgery. Rest will require its postsurgery as a result of unstable hemodynamics, poor gas exchange or less than optimum muscle power. Some patients may develop abdominal sepsis post surgery as a result of anastomotic leak, wound infection, missed gallstones at laparoscopic cholecystectomy, presence of foreign body, etc.

Intra-abdominal sepsis can be located in the following places:[6]

- Peritoneal cavity: The most frequent site
- Retroperitoneal space
- Localized within the viscera to form an abscess.

Intra-abdominal infection and peritonitis are not the same. Peritonitis means inflammation of peritoneum or a part of it. It can be primary as in case of spontaneous bacterial peritonitis, related to peritoneal dialysis (CAPD) and tuberculous peritonitis. Secondary peritonitis develops secondary to an enteric source as in GI perforation, bowel wall necrosis due to ischemia, etc. Perforation of any portion of the gut within the abdominal cavity, whatever the etiology, causes bacteria to leak into the peritoneal cavity and leads to intra-abdominal sepsis and peritonitis. Similarly, obstruction or gangrene of the bowel from strangulation or from mesenteric vascular occlusion are important causes of peritonitis.[7]

Peritonitis can be generalized (diffuse) or localized to form an intra-abdominal abscess. The most common site is right lower quadrant as with acute appendicitis or perforated duodenal ulcer. It can also be in pelvic, subhepatic or subphrenic areas. Retroperitoneal abscesses occur in the space between the transversalis fascia and the retroperitoneum. Visceral abscesses can be in the liver, gallbladder (empyema), pancreas, kidneys or as tubo-ovarian mass.[5,6] The common causes of intra-abdominal sepsis are enumerated in Table 11.1.

Another severe form of peritonitis is postoperative peritonitis.

Postoperative peritonitis may occur due to:

- Anatomic dehiscence because of infection, ischemia and hemorrhage
- Excessive spillage of infected material and soiling of the peritoneum during handling of viscera during surgery.
- Excessive handling of the bowel and retraction, e.g. during pelvic surgery an undue pressure is exerted by the retractors on the ileocecal area during surgery. Usually, a small area of ileum is devitalized and suffers from pressure necrosis and perforates on 3rd or 4th postoperative day.[5]

Entry of Bacteria in the Peritoneal Cavity

Normally peritoneal fluid is secreted by the intestinal serosa and is absorbed through the terminal lymphatics present on the peritoneal surface of the diaphragm. From there, it reaches the central circulation via thoracic lymphatics. Thus, any fluid present in the peritoneal cavity finds its way into the central circulation within few hours. Peritoneal cavity is a sterile cavity. The development of peritonitis can be explained by following mechanisms.

- Intestines have a load of bacteria in their lumen, however, normal mucosa of the bowel is impermeable to bacteria and toxins produced by the bacterial degradation. In low cardiac output states and bowel wall edema, poor perfusion leads to gut ischemia resulting in breach of the anatomic integrity of the gut and **transmucosal migration** of bacteria takes place.
- When there is perforation, intestinal fluid is lost into the peritoneal cavity via the serosal perforation, from where, absorption of toxic materials takes place, allowing **transperitoneal migration** of bacteria.
- Absorption then takes place via **transdiaphragmatic** lymphatics into central circulation. These transport mechanisms are responsible for early systemic manifestations of peritonitis as the bacteria and their products are transported to the systemic circulation.[7]

Table 11.1: Common causes of intra-abdominal sepsis[6, 7]

1.	Gastrointestinal:
	– Perforated gastroduodenal ulcer
	– Intestinal obstruction leading to perforation
	– Perforation of any other hollow viscus or any part of the gut, e.g. gallbladder.
	– Acute appendicitis
	– Acute diverticulitis
	– Gangrene of the bowel because of strangulation, ischemia or obstruction
2.	Acute necrotizing pancreatitis.
3.	Cholecystitis (acute, acalculus, gangrenous)
4.	Liver abscess (pyogenic, amrebic)
5.	Pelvic pathologies
	– Acute salpingitis
	– Tubo-ovarian masses
	– Septic abortion or puerperial sepsis
6.	Perinephric or retroperitoneal abscess
7.	Multiple trauma (blunt/penetrating)
8.	Following abdominal surgery.
	Low perfusion states.

Pathophysiology

The causative agent could be any of these, the most common being:[2,6]

- Gram-positive bacteria
- Gram-negative bacteria, commonly *E.coli*
- Endotoxins: These are the heat stable polysaccharides derived from the cell wall of gram-negative bacteria
- Viruses and fungi.

The result of the interaction between the host and the microbe depends upon

Host related factors:

- Age of patient
- The immunity and nutrition of the host
- Other comorbid conditions of the patient such as chronic illnesses, burns, etc.

Microorganism related factors:

- The degree of contamination
- The lesion present, e.g. perforation of small bowel, colon, anastomotic leak has high mortality
- The duration of perforation
- The site of infection
- The virulence of invading organisms.

Depending on the relative strengths of the two, there will be localized abscess formation, sealed perforation, generalized peritonitis or septic shock.

The insult to the peritoneum is usually a bacterial toxin which is an endotoxin. Endotoxin is a major

component of cell wall of the bacteria and is composed of a lipopolysaccharide phospholipid and protein. The lipid A moiety of this lipopolysaccharide (LPS) binds to the lipoprotein binding protein present on host cells.[2]

Any insult to the peritoneal cavity invokes an inflammatory response from the host. The immune response could be blood borne or cellular. The blood borne immunity is mainly in the form of immunoglobulins, complement system, leukotrienes, autocoids and cytokines. Immunoglobulins are specific antibodies directed against specific antigens on bacteria or endotoxin. The cellular immunity is in the form of macrophages, leukocytes, platelets, endothelial cells, mast cells and lymphocytes.[7]

The invasion by endotoxin rapidly triggers production of proinflammatory mediators including Tissue Necrotizing Factor (TNF) and Interleukin 1 (IL- 1) and activation of complement cascade.[2,4] The complement system is a multicomponent triggered enzyme cascade that attracts phagocytes to microorganisms increasing capillary permeability, neutrophil chemotaxis and endothelial cell adhesion. This causes release of mediators of inflammation such as histamine, leukotrienes and acute phase cytokines. This serves to contain or eradicate the invading microbes. Because of the endothelial cell adhesion and release of platelet activating factor, both intrinsic and extrinsic clotting mechanisms are activated to generate microthrombi. This is further facilitated by depletion of protein C levels and impaired function of protein C. In addition, other factors, such as free oxygen radicals, nitric oxide, lysozymes, proteases and other enzymes play an important role. **Oxygen free radicals** are generated as a result of marked increase in oxygen consumption (respiratory burst)[7] and formation of superoxide anion and hydrogen peroxides. The other mechanism involves **nitric oxide.** The hypoperfusion in sepsis is caused by either an endothelial inflammatory process or by a systemic vasodilatory process in response to nitric oxide production by inducible form of NO synthase (iNOS), by the ATP- dependent activation of potassium channel or insufficiency of vasopressin. If this process continues, a state of refractory vasodilatation is reached.

In severe sepsis, the net effect could be actually anti-inflammatory resulting in leukopenia and hypothermia. Some patients appear immunostimulated while some are immunosuppressed. This is because of release of anti-inflammatory mediators such as IL-4 and IL-10, resulting in a negative feedback mechanism.[8]

Peritoneum has a total surface area of around 1.8 mt^2 which is almost same as body surface area. The consequences of any injury to the peritoneum will be similar to the burn injury of the skin.[7] Being a biological membrane, it allows passage of water, electrolytes, micromolecules and certain macromolecules such as proteins under specific conditions. Normally, there is minimal amount of fluid in the peritoneal cavity facilitating movement of the structures within. It is secreted by the serosa and is absorbed through the diaphragm via lymphatics reaching the central circulation.

When abdominal sepsis develops, there is:

- Increase in blood supply
- Increased permeability of vessels
- Increased osmolality of the peritoneal fluid as a result of presence of proteins and other macromolecules
- Increase in intra-abdominal pressure

All these factors cause exudation of fluid from the peritoneal surface into the abdominal cavity. This fluid is said to be in the 'third space'. Third space is nothing but non-functional extracellular compartment. This is a physiological concept, a place where fluid accumulates, when it is neither in the intracellular nor in the extracellular space and is not normally perfused. In fact, it causes depletion of fluid in the first and second compartment. Peritoneal cavity has a large capacity to sequester fluid, almost 9-18 L of fluid, which is similar to 100 percent burns of skin. Even 1 mm increase in the thickness holds around 18 L of fluids.[7]

Physiological Sequelae of Sepsis

They are characterized by imbalance between demand, extraction and transport of oxygen.[9]

Sepsis: It is a hypermetabolic state in which there is increased peripheral oxygen demand.

Increased oxygen demand: As a result of direct cellular activation and secondary to hormonal stress response. This causes increased metabolism with or without hyperthermia. This coupled with poor circulation causes shift to anaerobic metabolism causing lactic acidosis.

Altered oxygen extraction: Activation of leukocytes, platelets and other cellular elements causes capillary obstruction, decreased responsiveness of arteriolar smooth muscle to adrenergic stimulation and injury to the endothelial cells. This results in interstitial edema and maldistribution of blood flow.

Altered oxygen transport: It is insufficient to meet the demands of the tissue. There is reduced venous return to the heart and peripheral pooling as a result of peripheral vasodilatation. Because of this it is also called

as 'distributive shock'. The second cause is myocardial depression.

The clinical effects of sepsis:

- Severe vasodilatation
- Pooling of blood into the peripheral tissues
- Enhance the permeability of local blood vessels causing loss of fluids into the tissues.

All the above factors cause hypovolemia and hypovolemic shock.

- Microvascular thrombosis causing reduced blood supply to the tissues.
- If not treated in time, it causes irreparable damage and death.

The effect of sepsis on various organ systems: As the severity increases, multiple organs are involved. The lungs get involved early in the course of sepsis.

Respiratory System

The lungs are involved as a result of the infective agent reaching the lungs through the blood (blood borne infection), through the transdiaphrgmatic route and as a result of mediators of sepsis. In addition, abdominal distention causes intra-abdominal hypertension leading to increased intrapleural pressure, splinting of diaphragm. There is increased airway pressure, reduction of lung volumes and capacities and basal atelectasis. Presence of pain also limits respiratory excursions. These factors cause ventilation-perfusion mismatch and pulmonary shunting.[7]

There is widespread endothelial injury in sepsis. As a result fluid exudes out of the capillaries into the interstitium and the alveoli. Capillary membrane becomes permeable to the proteins which leave the capillaries to enter the interstitial tissue. Here they exert osmotic pressure and draw water along with them.

Presence of fluid and proteins in the interstitial tissue and alveoli reduces lung compliance and increases the barrier for diffusion of gases between the endothelium and the alveoli. This results in hypoxia. There is loss of surfactant, causing collapse of the alveoli. There are areas of consolidation formed. All factors culminate into development of acute respiratory distress syndrome. (ARDS) is the dreaded complication of sepsis. These patients have high glutamine efflux from the lungs.[11] Alterations in the pulmonary surfactant and increase in the inducible nitric oxide synthase (iNOS) have been implicated in the development of ARDS.[12]

Thus sepsis gives rise to Acute Lung Injury (ALI): ALI is said to be present when the following findings are present in the patient:[13]

- Acute onset of impaired oxygenation
- Severe hypoxemia with PaO_2: FiO_2 < 300 mm Hg. A more severe form of hypoxemia where the ratio is < 200 is termed as ARDS.
- Bilateral diffuse infiltration on chest X-ray
- Pulmonary capillary wedge pressure < 18 mm Hg, indicating thereby that it is not of cardiac origin.

Renal System

Acute renal failure occurs in approximately 19 percent of patients with moderate sepsis, 23 percent with severe sepsis and 51 percent patients in septic shock.[14] Mechanism of this renal failure is:

- Hypovolemia secondary to exodus of fluid from the vascular compartment
- Reduced cardiac output
- Reduced plasma levels of ADH and aldosterone.

These factors reduce the renal blood flow, more so in the cortical region and thereby the GFR. Hypoxia of the renal tubular cells causes acute tubular necrosis (ATN). There is gross oliguria. As the excretory products of metabolism are not thrown out of the body, it produces metabolic acidosis. Development of ARF in sepsis has grave prognosis.

Cardiovascular System

Sepsis causes severe hypotension and myocardial depression. Leaky capillaries allow the fluid to leave the vascular compartment on a large scale causing hypovolemia. There is reduced venous return to the heart and therefore, reduced cardiac output. This is also reduced due to direct myocardial depression caused by the factors release during inflammatory response such as myocardial depressant factor (MDF) and the acidosis that is present.

Prompt and adequate volume resuscitation prevents cardiovascular depression.

The effect on CVS can be considered in two stages,

Hyperdynamic stage: Cells are unable to utilize the oxygen properly and therefore, CVS tries to compensate by increasing the cardiac output and peripheral vasodilatation. The peripheral vascular resistance is reduced. This causes blood to be pooled in the peripheral tissues, reducing the central circulation. The extremities however, are warm and pink with bounding pulse.

Hypodynamic stage: As the shock progresses, the cardiovascular system becomes exhausted and is unable to meet the demands of the tissues. Compensatory vasoconstriction takes place reducing the peripheral blood flow to skin, muscles and splanchnic circulation.

Reduced renal blood flow decreases the GFR. As a result of peripheral vascular cut off and severe vasoconstriction, the extremities are cold and cyanosed.

Coagulation

There is often thrombocytopenia seen with altered platelet function. Because of the widespread endothelial damage and the effect of released inflammatory substances, significant changes occur in the coagulation system. In sepsis, Virchow's classical triad consisting of change in the coagulability, endothelial damage and abnormal blood flow comes into play.[15]

Gastrointestinal System

There is extensive bowel edema, ischemia and ileus. Initially as a response to peritoneal irritation, there is hyperemia and hypermotility of the bowel.[7] As the sepsis progresses the motility is depressed, finally resulting in a dynamic ileus. This causes distention of the bowel and as the contents can not move further, there is accumulation of gas and fluid in the bowel. The absorptive capacity of the intestine is reduced. There is gut wall edema. Because of the increased pressure, there is pressure on the vessels leading to ischemia and abnormal permeability of bowel mucosa to the invading organism. The mechanism of this is described in detail in the chapter on intestinal obstruction.

Metabolic and Endocrine Response

In face of the imbalance between demand and supply of oxygen as well as depressed function of the lungs and the cardiovascular system, there is development of oxygen debt leading to anaerobic metabolism and glycolysis. There is increased production of lactic acid and other acidic metabolites. They cannot be cleared off easily because of sluggish circulation and impaired renal function leading to progressive acidosis.[7] It could also be because of direct cellular insult by the invading bacteria or lactic acid produced as a result of bacterial metabolism.

Development of peritonitis induces an intense stress response and causes release of the stress hormones. Massive outpouring of adrenaline and noradrenaline from the adrenal mucosa is responsible for the majority of clinical features of shock. There is tissue breakdown and negative nitrogen balance because of ACTH and sodium and water retention because of aldosterone. All stress hormones are anti-insulinic and produce hyperglycemia. As the sepsis progresses, the stress hormones get exhausted leading to hypoglycemia and hypotension and hypothyroidism.

Clinical Features

Clinical features are the result of the presence of endotoxins as well as the anti-inflammatory mediators released and include the following:

Though **fever** is considered as the hallmark of infection, in severe cases there may be hypothermia. Increase in body temperature increases the fluid requirement and can easily push the patient in dehydration.

There is progressive development of **tachycardia** and **tachypnea** and inability to maintain normal ABG resulting in desaturation. Air hunger is present in order to gain more oxygen, hence, often there is CO_2 washout.

Altered sensorium and anxious look may be one of the earliest sign of sepsis related hypoperfusion and should be looked for.

There is tenderness, guarding, rigidity in patients with peritonitis.

Urine output is usually reduced, related initially to the degree of dehydration.

Petechial hemorrhages are seen in conjunctiva.

Progressive hypotension[9] can occur. The septic shock develops as a result of:

- Severe vasodilatation
- Hypovolemia as a result of loss of fluid in peritoneum, bowel wall and inside bowel lumen
- Altered distribution of blood flow
- Mayocardial depression.

Initially, the pulse is bounding and hyperdynamic, skin moist and pink. However, as the sepsis progresses, there is peripheral vasoconstriction, pulse becomes rapid and thready and cyanosis may develop because of peripheral cut off of blood supply.

A sudden development of hypotension and tachypnea in a postoperative patient indicates intestinal leak and development of sepsis. The effect of sepsis on various organ systems is enumerated in Table 11.2.

As the severity increases, multiple organs start getting involved. The first system to succumb is pulmonary system followed closely by the gastrointestinal tract, kidney, hemopoietic system, liver, central nervous system and finally cardiovascular system. Multiple scoring systems have been evolved to quantify the various organ dysfunction. The commonly used ones are proposed by Goris[10] and Knaus. Similarly APACHE scoring system is used to determine the prognosis. Each organ system is given scores from 0 to 2, depending on severity. Higher score indicates more severe sepsis.

In addition there will be leukocytosis/leukopenia, thrombocytopenia, disseminated intravascular coagulopathy, hyperglycemia/hypoglycemia. In severe cases, it

Table 11.2: Effect of sepsis on various system

Respiratory system	**Cardiovascular system**
Progressive hypoxia	Peripheral vasodilatation
Fluffy shadows on X-ray chest	Myocardial depression
ALI/ARDS	Hyperdynamic circulation followed by Hypodynamic circulation
Renal system	
Renal insufficiency	
Oliguria	**Gastrointestinal system**
Progressive increase in BUN/ creatinine	Gut ischemia
	Translocation of bacteria
	Hypermotility and hyperemia of bowel followed by
Coagulation system	Adynamic ileus
Altered platelet function	
Widespread endothelial damage	
Deranged coagulation	

may produce irreversible vascular damage, cellular hypoxia, shock and death.

Role of Anesthesiologist in the Management of Abdominal Sepsis

The anesthesiologist's role in the management of abdominal sepsis is not only confined to administering anesthesia intraoperatively but also in resuscitation of the patient prior to surgery as well as postoperative ventilatory and hemodynamic management. The prognosis depends on how early and how well the infection is limited and resuscitation is carried out. The anesthesia *per se* would not be different from other abdominal explorations. The principles remain the same and are described in the previous chapter on intestinal obstruction. The most important part, i.e. the resuscitation of a septic patient is described here at length.

Abdominal cavity is like Pandora's box throwing surprises after opening the abdomen. Patient with abdominal sepsis posted for emergency abdominal surgery poses a lot of challenges to the anesthesiologist. There may be great risk while inducing anesthesia in such patient. This is so because:

- The surgical diagnosis is often not clear. The extent of damage may vary from localized peritonitis in case of perforated appendix to a full blown pus inside the peritoneal cavity or fecal peritonitis.
- History and previous medical record is not available properly.
- Patient has hypovolemia and is less than optimally resuscitated.
- Preoperative medical conditions are not always treated or controlled.
- Patient may have raised intra-abdominal pressure with consequent effect on organ perfusion and function.
- Risk of full stomach with consequent regurgitation and vomiting.
- Presence of acid-base and electrolyte imbalance.

Preoperative Assessment and Optimization

The objective of emergency anesthesia is to permit correction of surgical pathology with minimum risk to the patient. Therefore, it requires a thorough preoperative work up guided by the patient's history and physical examination. To rush to deal with the underlying cause without careful yet speedy attention to his general condition and without making any attempt at restoring the fluid-electrolyte and acid-base balance will prove disastrous.

Ascertain the likely surgical diagnosis, the likely duration and magnitude of surgery, urgency of the surgery, as this will give the idea about the time period available to prepare the patient.

History

- Origin, duration and progress of the present symptoms. History of prolonged duration should make the physician suspect advanced sepsis and a lot of disturbance with milieu interior.
- Try and determine cause of sepsis.
- Symptoms and signs of the present illness, e.g. abdominal pain, vomiting, guarding, rigidity, etc. Site of the perforation should be determined as it guides to the likely volume and the composition of the contamination.
- Look for other comorbid conditions such as hypertension, diabetes, ischemic heart disease, valvular heart disease, COPD, convulsions, etc. Presence and severity of specific symptoms of, e.g. agina, DOE, orthopnea, nocturnal cough and productive cough should be sought. Patient's functional capacity should be determined. Basal crepitations, triple rhythm and raised JVP indicate impaired ventricular function and limited cardiac reserve.
- Pain in abdomen because of peritoneal irritation may cause respiratory distress. Degree of abdominal distention and respiratory embarrassment if any should be noted.

Examination

- A swift but thorough airway assessment is done in view of rapid sequence induction intubation that is

required in these patients. Difficult airway complicating a patient with vomiting risk is a perfect recipe for stormy induction and pulmonary aspiration of gastric contents. Prior airway assessment will ensure that proper intubation aids are kept ready.

- Evaluation of the intravascular volume status of the patient should be done as it will guide deficit therapy. Tachycardia and cutaneous vasoconstriction are the early signs of fluid loss. Cold extremities, rapid thready pulse, air hunger, anxiety, restlessness, sunken eyeballs, dry tongue and mucous membrane, oliguria indicate hypovolemia and intense sympathetic stimulation. Under-estimated or unrecognized hypovolemia may lead to circulatory collapse during induction of anesthesia which attenuates the sympathetically mediated increases in arteriolar and venous tone.
- Pallor, cyanosis, clubbing, icterus, edema, capillary filling, etc. should be looked for.
- Assessment of respiratory distress: Respiratory rate, labored breathing, use of accessory muscles of respiration, auscultation of chest for normal breath sounds as well as foreign sounds such as wheezing, crepitations, etc. Rapid, shallow breathing may be because of pain in abdomen or abdominal distention causing splinting of diaphragm. Rapid deep breathing is usually associated with septic shock. Simple bed side tests such as breath holding, expiratory time and peak expiratory flow rates should be measured. All these tests provide quick idea about the reserve capacity of the patient.
- Pulse rate, rhythm, volume, heart sounds, presence of murmurs, etc. Rapid, low volume pulse, raised JVP, basal crepitations indicate that the patient is in cardiac failure and may need perioperative cardiac support.

Investigations

As soon as the patient is admitted, after initial history and examination, certain investigations must be sent for, simultaneously as the resuscitation is carried out. The investigations include routine investigations, specific investigations and those required to confirm the diagnosis.

- Routine investigations include complete hemogram of the patient and urine analysis.

  Hemoglobin: Indicates the oxygen carrying capacity of the patient and will decide the transfusion trigger for the patient. Patients with intestinal strangulation often have blood lost in their peritoneal cavity.

  Total WBC count: Leukocytosis usually indicates infection. However, in case of fulminant infection neutropenia may be seen. Eosinophilia should raise the suspicion about allergic diasthesis and possible worm infestation.

  Platelet count: Platelet activating factor (PAF) is one of the mediators released during the genesis of sepsis. This causes platelet aggregation and adhesion with resultant consumption. Coagulopathy is the hallmark of sepsis. Both platelet count as well as bleeding time to test their function must be done.

  ESR will be raised in case of any chronic infection and inflammation.
- Blood should also be sent for evaluation of blood glucose, renal function and liver function.

  Blood glucose can be variable. There may be hyper- or hypoglycemia depending upon inflammatory or anti-inflammatory response.

  BUN and creatinine levels will indicate the renal function as the kidneys directly get affected by the reduced renal perfusion. A high creatinine level of > 3 mg percent or a rising creatinine will carry a high incidence of perioperative renal dysfunction.

  Deranged liver function tests such as high bilirubin, raised liver enzymes and low serum albumin indicate poor prognosis. Serum albumin also decides the intravascular stay of the fluid therapy given.

  Serum electrolytes are mandatory. Often, abdominal sepsis is secondary to the perforation of an abdominal viscus. There is loss of gastric and intestinal fluid into the peritoneal cavity. There may be associated vomiting as in case of intestinal obstruction. The renal function may be less than optimum. All these factors cause severe electrolyte derangement. This may lead to hypotonia, delayed emergence from anesthesia, dysrhythmias, abnormality on ECG and paralytic ileus.
- Often the **coagulation profile** is deranged in patients with sepsis[15] because of sepsis induced platelet dysfunction and consumption of coagulation factors. Bleeding time, clotting time, prothrombin time and INR must be done prior to taking up the patient for surgery. In case of any abnormality, attempt should be made to rectify the abnormality either prior or during the course of surgery. Blood should be sent for grouping and cross matching and when required should be readily available.
- **X-ray chest** shows abnormality in cardiovascular and respiratory systems. Pneumonic patches and

infiltrates, blunting of costophrenic angles, miliary mottling, cavities, prominent bronchopulmonay markings should caution the anesthesiologist about likelihood of patient developing postoperative respiratory insufficiency. ARDS is very common in patients with sepsis and is evident on X-ray chest.

- **ECG:** Whenever indicated depending on patient's age, coexisting condition such as hypertension, diabetes, ischemic heart disease should be done. A quick target radiography will help in identifying the patient at the risk of developing ischemia.
- **Arterial blood gases** will indicate any diffusion problem, i.e. hypoxia (PO_2 < 60 mm Hg), or ventilatory problem (CO_2 retention PCO_2 > 45 mm Hg). Often, in patients with sepsis, there will be diffusion barrier without much affecting the ventilation. So there is hypoxia with CO_2 washout. In addition, it will also indicate the presence of acidosis, serum bicarbonate levels and peripheral perfusion decided by the level of base deficit.
- **Serial serum lactic acid levels**[16] have prognostic value and should be measured in all patients. An elevated concentration (>2.5 mmol/l) of lactic acid (LA) in venous blood has been demonstrated to be indicative of recent or ongoing anaerobic metabolism, generally as a consequence of:
  - Insufficient oxygen delivery to meet normal tissue/organ demands (i.e. "shock") as a result of hypotension and hypoperfusion
  - Increased oxygen demands
  - Poor circulation leading to shift to anaerobic metabolism
  - Inability of kidneys to clear lactic acid and other acid metabolites from the blood
  - Bacterial metabolism causes production of lactic acid.

It is especially important in those patients who do not exhibit classical signs of shock such as tachycardia, hypotension, altered sensorium, reduced urine output (occult shock). In this way, patient can be treated early. Several recent studies in humans have demonstrated the value of lactic acid measurement for the prediction of adverse outcome after injury. Patients who are unable to resolve a lactic acidosis within 24 hours of injury have been shown to suffer significantly higher complication and mortality rates.[16-18] Serum lactate levels of ≥ 4 mmol/l is associated with higher acute phase (< 3 days) death incidence. Many intensivists use serum lactic acid as the marker of global oxygen debt while resuscitating a shocked patient. However, lactate should be measured to augment and not replace bedside assessment of mortality risk. The other markers that can be measured serially are blood pH and serum bicarbonate levels.

- Almost always investigations are also carried out to confirm the surgical diagnosis. These include USG abdomen, X-ray abdomen and CT scan abdomen. The anesthesiologist must take active effort to go through these. Knowing surgical diagnosis and knowledge about the surgical procedure contemplated makes the anesthesiologist more equipped to manage the case.

Diagnosis, resuscitation and preparation for surgery should go hand in hand.

Resuscitation is aimed at:

- Restoring blood volume by appropriate volume resuscitation
- Restoring tissue perfusion pressure
- Restoring oxygen delivery.

In October 2002, in Barcelona declaration, the **Surviving sepsis guidelines (SSG)** were introduced. They were revised in 2008[3] (Table 11.3).

Anesthesiologist can take help of these guidelines to resuscitate the patient. Any haste on his part will worsen the outcome.

The steps in the resuscitation prior to anesthetizing the patient are:

Initial Resuscitation

In every patient with hypotension and serum lactate levels above 4 mmol/l, the resuscitation should begin as early as possible. This is called as early goal directed therapy (EGDT) for septic shock.

The **therapeutic goal** is:

- CVP > 8 mm Hg. In mechanically ventilated patients, in patients with increased intra-abdominal pressure and patients with pre-existing decreased ventricular compliance, the CVP should be around 12 to 15 mm Hg.
- MAP > 65 mm Hg
- Urine output > 0.5 ml/kg/hr.

Table 11.3: Surviving sepsis guidelines (SSG) in a Nutshell

1. Initial resuscitation to the predefined goals	8. Inotropic therapy
2. Diagnosis by culture tests	9. Use of steroids
3. Antibiotic therapy	10. Mechanical ventilation
4. Source identification and control	11. Control of glucose
5. Fluid therapy	12. Renal replacement therapy
6. Use of blood products	13. Bicarbonate therapy
7. Use of vasopressors	14. DVT prophylaxis
	15. Sedation, analgesia, NM blockade

- SvO_2 > 70 percent or mixed venous saturation > 65 percent.
- Repeated lactate levels to < 2 mmol/l.

In 263 patients assigned to a study to know the usefulness of EGDT, it was noticed that the mortality was much less, the mean central venous oxygen saturation was better, the lactate levels were less, base deficit was less and the pH was higher as compared to those who receive standard therapy.[19] It was concluded that EGDT provides significant benefits with respect to outcome in patients with severe sepsis and septic shock. These benefits arise from the early identification of patients at high risk for cardiovascular collapse and from early therapeutic intervention to restore the balance between oxygen delivery and oxygen demand.

Volume of Resuscitation

In any patient in whom the fluid is sequestered or lost or in whom hemorrhage has occurred, quantify and correct the blood volume and body water deficit. In emergency situations, it may not be always possible to completely correct the deficit but depending on the time available one must try to correct by rapid fluid replacement. Choice of fluid replacement depends on nature of loss, hemodynamic status and condition of patient. Usually, isotonic solutions are used such as 0.9 percent saline, Ringer's lactate. It will also depend upon presence of electrolyte imbalance if any, e.g. hypokalemia, hyperkalemia, etc. and is directed towards correction of this imbalance. In addition, colloids and whole blood are used. Rate of fluid administration depends on the severity and type of fluid disturbance, the presence of continuing losses and hemodynamic and cardiac status. In severe deficit, the initial rate may be as high as 1000 ml per hour reducing the rate as condition improves. Elderly patients and patients with cardiac disease require careful correction with monitoring.

Fluid therapy is guided by the patient's central venous pressure, blood pressure, presence of orthostatic hypotension, heart rate, urine output and his mentation. The aim should be to maintain an hourly urine output of at least 30 to 50 ml. This is achieved by use of intravenous fluids. Rapid intravenous infusions are given to reach above mentioned goals. Initially a fluid challenge is given of 20 ml/kg rapidly or 500 to 1000 ml of crystalloids or 300 to 500 ml of colloids over 30 min. monitoring the hemodynamic parameters. Any decrease in heart rate and improvement in CVP, blood pressure, urine output, capillary filling, $ScvO_2$ is noted. If however, fluid challenge results in increasing cardiac filling pressures without hemodynamic improvement, rate of fluid administration should be reduced. Any further increase in filling pressure will push the patient in cardiac failure. In such cases, Dobutamine infusion may be started to augment the cardiac output. Patients with perforation of long duration or pancreatitis require massive volumes for resuscitation.

Overzealous fluid administration should be avoided. After routine surgery, there is usual weight gain of 3 to 5 kg postoperatively. This results in generalized edema impeding tissue healing and cardiopulmonary function. It also causes reduced SpO_2, delayed recovery of gastrointestinal function and poor survival.[20]

Variation in the pulse pressure with administration of IPPV can be monitored continuously and gives a useful idea about the intravascular status of the patient. These changes in pulse pressure with IPPV predict which patients will respond to fluid therapy, i.e. increased preload by increasing their cardiac output. In one study the fluid therapy was administered to keep this variation in the pulse pressure to < 10 percent. The volume required to achieve was substantially high and was associated with reduced duration of postoperative stay, reduced postoperative complications and reduced duration of mechanical ventilation.[21]

Colloids or Crystalloids

Whether colloid or crystalloid solutions should be used during resuscitation of septic patient has always been the matter of debate. However, in a large number of randomized controlled trials and meta-analysis, studying effect of albumin v/s isotonic saline or lactated Ringer's solution, it was noted that there is not much difference in the outcome between the two groups.[22] They were studied with respect to mortality, hospital stay and development of pulmonary edema. When crystalloids are used for resuscitation, the amount of fluid required is almost 3 to 4 times that of colloids. In fact, some of the studies had excess mortality of 4 percent in colloid group. The SAFE trial published in 2004 concluded that crystalloids were as efficacious as colloids and the volume of crystalloids required is 1.4 times that of colloids.[23] Also since they are freely permeable in all compartments of the body, their intravascular occupancy is limited. This, in addition to the fact that there are leaky capillaries in sepsis, leads to edema formation and discrepancy between input and output. Input is much higher than output. Colloid resuscitation has the problem of allergic reactions as well as high cost.

Blood products are transfused if Hb < 7 gm%.[3] The target hemoglobin should be between 7 to 9 gm%. In special cases like myocardial infarction, a higher hematocrit should be aimed for.

If SvO_2 of >70 percent is not achieved then packed RBCs are transfused to achieve a Hct of > 30 percent. It is unnecessary to use erythropoietin routinely to treat sepsis related anemia.[3] Or dobutamine infusion up to 20 µgms/kg is used in order to increase the cardiac output.[3] How fast fluids should be administered, depends upon the estimated deficit and the time available for restoring blood volume prior to anesthesia so also on the patient's cardiac compliance.

Diagnosis

Culture before antibiotic therapy to diagnose therapy of causative organism. At least two blood cultures should be drawn, one percutaneously and the other through the vascular access device that is > 48 hours old. Perform imaging studies to confirm source of infection.

Antibiotic Therapy

Parenteral antibiotics should be started within one hour of recognition of sepsis after sending blood for cultures. Initial drugs should be broad-spectrum and should include one or more drugs that have activity against the most likely pathogens, gram-positive, gram-negative and anaerobic bacteria. Start the antibiotic that is effective against the pathogen most likely to be encountered. The antibiotic should be able to penetrate the source of infection. Broad-spectrum antibiotics are continued till the causative agent and its susceptibilities are defined. A single large therapeutic dose is given intravenously preoperatively. Combination therapy should also be considered in neutropenic patients. Antimicrobial spectrum should be reassessed every 48 to 72 hours on the basis of microbiological and clinical data. This is to narrow the spectrum and prevent development of resistance. While administering antibiotics, it is important to know whether their action is concentration dependent or time dependent. Meticulous attention should be paid while deciding on the dose and timing of the antibiotic dose.[24] Duration of treatment should be minimum 7 to 10 days and guided by clinical response. Negative culture does not exclude sepsis.

Source Control

Evaluate the source of infection and eliminate it. This may require:

- I and D of an abscess
- Debridement of an infected tissue
- Washing the abdominal cavity
- Removal of an infected device or catheter.

Unless the source of infection is removed, the antibiotics are not going to help.

Use of Vasopressors and Inotropes

The ultimate goal of resuscitation is to restore the tissue perfusion. It is a common practice to equate this with arterial blood pressure. In sepsis, there is severe peripheral vasodilatation, abnormally high cardiac output and abnormal shunting of the cardiac output. Vasopressors should be started only after volume resuscitation. Vasopressors are used so as to maintain the MAP > 65 mm Hg. This value is selected because it is the lower limit of autoregulation. But more important than this is to follow the clinical indicators of tissue perfusion. These are, sensorium of the patient, peripheral skin temperature, capillary refill and urine output. In addition, other parameters such as blood lactate levels and $ScvO_2$ are monitored.

Sometimes, the hypotension is severe and there is not much time to wait for the effect of fluid resuscitation. In order to improve perfusion, pharmacological intervention in the form of vasopressors and inotropes may be carried out. Though most of the agents have both the actions, the agent is selected based on the intended purpose. Vasopressors raise the blood pressure while inotropes raise the cardiac output.[25]

Selection of vasopressor is important. They have positive impact on the mortality in septic patients. The choice would depend on the desired action, α or β. While α receptors cause purely vasoconstriction, β_1 receptors have predominant action on heart and cause tachycardia and increased myocardial contractility. β_2 receptors on the other hand have peripheral action and cause vasodilatation. It is always advisable to have an indwelling arterial catheter whenever high doses of vasopressors are being used as the effect of the therapeutic regime can be checked and doses can be varied if required.

The initial vasopressor / inotrope of choice is either norepinephrine or dopamine.

Septic shock is characterized by intense peripheral vasodilatation and there is often systemic hypotension in spite of aggressive fluid infusion and increased cardiac output. Traditionally vasopressors and inotropes are started in stepwise manner. Customarily dopamine or noradrenaline is started as the first drug.

- **Dopamine** has variable response according to the dose used, as it has action on dopaminergic (up to 3 µgm/kg/min), β (10 up to µgm/kg/min) and α receptors(10-20 µgm/kg/min) in dose dependent manner. It increases MAP by increase in stroke volume and heart rate. It is especially useful in patients with compromised cardiac function. However, there is no evidence that renal protective doses of dopamine improve the renal and splanchnic

blood flow in the sepsis and therefore, should never be used during sepsis. It is usually started in doses of 5 to 10 µgms/kg/min carefully watching the response. If blood pressure fails to respond after this, another vasopressor is added.

Noradrenaline would seem an ideal agent under these circumstances and will induce vasoconstriction in many beds such as skin, muscles, splanchnic bed, etc. There is a valid fear that this might decrease visceral and other organ blood flow impairing organ function. However, in severely vasodilated patients it may actually increase the visceral flow by improving the central aortic pressure. When started early in the septic shock it improves perfusion pressure. This actually increases the renal, gastric mucosal and splanchnic flow. This decreases mortality.[26,27]

- Noradrenaline should be administered through the central catheter and in the infusion form. Noradrenaline has less side effects than adrenaline and its main action is on α receptors leading to vasoconstriction. Also it does not cause tachycardia. It is more potent than dopamine and more effective in reversing the hypotension. It should be started in low dose of 0.05 µgm/kg/min and increased gradually till the decided goal is achieved. The dose range is 2 to 20 µgms/min. In high doses of > 3.3 µgms/kg/min it causes vasoconstrictor effect in the peripheral beds and may lead to distal necrosis.[28]
- In case the MAP fails to respond to noradrenaline , **adrenaline** is the alternate agent used. Adrenaline causes undesired tachycardia and unfavorable effects on splanchnic circulation.
- **Vasopressin:** Septic shock is associated vasopressin deficiency and a hypersensitivity to its exogenous use. It is started in very small doses of 0.01 to 0.05 units/min. in case of shock refractory to other agents. Higher doses cause splanchnic, cardiac and digital ischemia. Both noradrenaline and vasopressin cause peripheral vascular cut off in high doses.

Phenylephrine being pure α agonist, causes severe vasoconstriction and decreased stroke volume.[29] Phenylephrine when used as a vasopressor agent in septic shock patient, is effective in increasing MAP without compromising gastrointestinal and hepatosplanchnic perfusion as compared with norepinephrine. If intense vasoconstriction is suspected then **Dobutamine** is added if required. Dobutamine is also started in case of initial resuscitation in case the cardiac output is low with high filling pressures. Dobutamine causes decrease in the afterload. Though it improves cardiac output, the blood pressure may drop further. Therefore, prior to starting dobutamine, minimum blood pressure of 90 mm Hg systolic is required. Hypotensive patients can have low, normal or high cardiac output and therefore, it is advisable to start a combination therapy with vasopressors and inotropes.

Patient is ready to be wheeled inside the operation table when:

- He is adequately resuscitated by infusion of crystalloids, colloids and blood if required
- His acid- base and electrolyte balance has been corrected
- His oxygenation is improved
- The above mentioned investigations are done.

It may also be necessary to support his respiration preoperatively if needed.

The next step is patient's risk stratification. This is done by taking into account his severity of symptoms, his comorbid conditions and the values of investigations.

Risk stratification of patients with abdominal sepsis is usually carried out by using APACHE – II (Acute Physiology and Chronic Health Evaluation) scoring system. It provides objective discrimination of low and high risk groups of patients with abdominal sepsis and describes morbidity of patients. It can be used for patients above 15 years of age. Score is calculated during the first 24 hours of admission from 12 routine physiological parameters like blood pressure, body temperature, heart rate, etc. acute physiology score is the sum total of all 12 individual parameters. To this, are added points for age and chronic health status points. The addition of acute physiology score, age points and chronic health points gives the APACHE – II score. Minimum score could be 0 whereas maximum score could be 71. As the score increases, there is increasing risk of hospital deaths. An APACHE – II score of 11 to 20 is a better predictor of risk of mortality following peritonitis. In a study, it was seen that the mean APACHE II score in survivors was 9.88 as compared to 19.25 in nor-survivors.[30]

The morbidity and mortality increases with increasing age, the degree of contamination, duration of preoperative symptoms, high serum creatinine and bilirubin levels.

The anesthesia management should be tailor made to suit the hemodynamics of individual patient.

Premedication

- Preoperative antibiotics
- Oxygen by mask or nasal prongs
- Antiaspiration prophylaxis: Inj Ranitidine, Inj Ondansetron IV
- Sedative and narcotic premedication avoided
- Inj Glycopyrrolate IM/IV.

Choice of Anesthesia

General anesthesia is always considered as the first choice in case of abdominal sepsis particularly with perforative peritonitis. The reasons being:

- Unstable hemodynamics with subarachnoid block.
- High level of block required for abdominal surgeries with manipulation of bowels to allow proper relaxation.
- Preoperative respiratory embarrassment because of abdominal distention following bowel perforation.
- Possibility of precipitous hypotension because of sympathetic block adding to the severe vasodilatation resulting from septic shock.
- Sepsis is often associated with coagulation abnormalities and insertion of epidural catheter is not advisable.
- Insertion of an indwelling catheter in presence of sepsis would risk infectious complications in the epidural space.

Epidural anesthesia has the advantage that it provides excellent analgesia, reduces stress response to surgery, increases gastrointestinal blood flow, improved tissue oxygenation, reduced incidence of myocardial infarction, reduced recovery time from surgery.[31] These benefits continue for prolonged period into the postoperative period because of indwelling epidural catheter. However, in presence of sepsis, anesthetists are reluctant to put in catheters because no benefits are noticed and infectious complications are feared. The gut is an organ that is not only frequently injured through systemic inflammation, but is also believed to play an important role as a focus for septic complications in critically ill patients. Impaired microvascular perfusion and subsequent mucosal hypoxia are viewed as the probably the most important factors in the pathogenesis of gut dysfunction in sepsis. Endotoxemia decreases microvascular perfusion to both mucosa and the muscularis layers of the gut. However, it also causes redistribution of blood supply to mucosa.

Based on both experimental and clinical studies it is clear that epidural analgesia increases gastrointestinal mucosal blood flow.[31] However, thoracic epidural anesthesia prevents this protective redistribution to mucosa. Therefore, it was inferred that though epidural block induced sympathetic blockade may be useful in nonseptic conditions to improve intestinal perfusion, blocking this mechanism may worsen gut mucosal perfusion. Another animal study, however, showed that continuous thoracic epidural anesthesia induced sympathetic blockade improves microcirculation in gut mucosa.[32]

Unless definitive guidelines are provided whether to use epidural anesthesia will remain a matter of debate and individual preference.

General Anesthesia

General anesthesia with rapid sequence induction intubation (RSI) is the technique of choice for these patients.

Since patient with abdominal sepsis would have intestinal obstruction with perforative peritonitis as the likely pathology, the principles of anesthesia induction, intubation, maintenance and monitoring would remain essentially same as that for intestinal obstruction described in detail in the previous chapter.

Intraoperatively there may be hemodynamic fluctuations. These are very common if the patient is not resuscitated properly. A patient having a systolic blood pressure within normal range and a central venous pressure in the range of 10 to 12 cm water and with his acidosis corrected, is not likely to experience major fluctuations. If the patient is on vasopressor/inotropic support, the same should be continued throughout. The support must go through a dedicated line so as to avoid any inadvertent bolus of the agent.

Use of steroids in sepsis is controversial for the fear of flaring of infection. However, its use appears logical as sepsis is a syndrome characterized by uncontrolled proinflammatory response steroids on the other hand have anti-inflammatory properties. Lower doses lead to less damage to the host defense system. The incidence of secondary infection is less. Improved lung function is seen in patients with unresolving fibroproliferative ARDS. Use of steroids is associated with improved survival rate. It can be concluded that a 7-day period of steroid replacement therapy results in improved survival in patients with septic shock with acute adrenal insufficiency.[33]

Currently, there is enough evidence to conclude that early high dose corticosteroid therapy has no role in the treatment of severe sepsis and septic shock. However, lower physiological doses restore hemodynamic stability, resolution of organ dysfunction and reduced mortality. They exert this effect by inhibiting effects of interleukins, neutrophils, coagulation cascade and other inflammatory substances. 50 mg or 100 mg three times a day is usually sufficient.

The intraoperative monitoring will include:

Pulse: Rate, rhythm and volume.

Blood pressure: Cuff pressure—manual as well as non-invasive. However, if the patient is hemodynamically unstable and is on support, it is better to have indwelling arterial catheter.

Cardioscope: After decompression of abdomen, there may be sudden surge of acidic metabolites from the ischemic gut mucosa as well as sudden decrease in afterload to the left ventricle. This precipitates hypotension and dysrhythmias.

Urine output: A continued urine output of >1 ml/kg/hr is indicative of good organ perfusion.

Oxygen saturation: Signal strength may not be adequate in intense peripheral vasoconstriction and severe hypotension.

$EtCO_2$: Gives idea of lungs and circulation.

Blood loss: Intra-abdominal infection and adhesions, strangulation cause great deal of blood loss.

Temperature: Extremes of temperatures can develop in a septic patient and must be treated vigorously.

Biochemical tests such as serum electrolytes, PT/INR, ABG can be sent during surgery.

Whether to reverse and extubate the patient at the end of surgery would depend upon:

- Pre- and intraoperative hemodynamic instability
- Involvement of lungs and kidneys due to sepsis
- Comorbid conditions such as diabetes, IHD, etc.
- Nature and extensiveness of the surgery
- Intra-abdominal pressure, bowel edema, tension while closing the abdomen
- Blood loss.

Under unfavorable conditions, it is prudent to support patient's ventilation in the postoperative period. Mechanical ventilation of sepsis induced ALI.[34,35]

We have already seen that patients in sepsis often have acute lung injury. Improper ventilator setting and prolonged ventilatory support leads to a condition which resembles ALI and is termed as ventilator associated lung injury (VALI). There are certain predisposing factors for the development of VALI.

- High tidal volume with high plateau pressures are avoided. Large tidal volume cause overdistention of alveoli and direct physical damage to the alveolar-capillary architecture (Volutrauma). This leads to sudden and rapid increase in the permeability. It increases the alveolar water and protein content thereby impairing the action of surfactant and reducing the lung compliance. Tumor necrosis factor and interleukin 1 are believed to be the causative agents. The ventilator settings should be meticulously set and changed as per the change in the lung conditions.[34]
- Tidal volume should be roughly 6 ml/kg
- Avoid repeated opening and closure of alveoli and small airways during each breath as it produces shear forces causing **atelectrauma**. PEEP should be set to prevent lung collapse at the end of expiration.
- The third important principle to ventilate the patient with ARDS is to prevent '**Barotrauma'**. Application of excessive pressure causes alveolar cell damage and air leaks out into the interstitial and mediastinal spaces. Therefore, plateau pressure should be kept at minimum possible level. Excessive pressures may cause translocation of bacteria from alveoli into the blood. Prone position considered for patients with ARDS requiring high levels of FiO_2.
- All mechanically ventilated patients must be nursed in semi-recumbent position[3] and should be given trial of spontaneous breathing whenever they meet the weaning criteria in the form of 'T' piece or low level of pressure support with CPAP.

Sepsis is a double edged weapon where the patient suffers both because of the causative agent causing infection as well as the body's own defense mechanisms. In fact, these defense mechanisms may continue to act even after the causative agent is removed and infection is treated. We need to continue supporting the various systems involved till their full recovery ensues.

The measures taken are:

- Hemodynamic support in form of proper fluid management, inotropic and vasopressor infusions if required
- Measurement of intra-abdominal pressure and measures to reduce it
- Renal replacement therapy if required, in form of proper infusion, continuous veno-venous hemo-filtration or hemodialysis[3]
- Continue postoperative ventilator support. There is likelihood of development of ARDS postoperatively if not already developed. Therefore, the patient should be closely monitored for development of pulmonary findings
- Maintaining blood glucose level < 150 mg/dl[3]
- Deep vein thrombosis prophylaxis
- Stress ulcer prophylaxis
- The extensive monitoring should continue in the postoperative period.

The treatment essentially consists of supporting various systems till the process of sepsis is arrested. If the source of infection is removed, proper antibiotics are administered and patient is adequately hydrated, hemodynamics have improved because of vasopressors-inotropes, patient has very good prognosis. The management of sepsis is depicted in a Nutshell in Table 11.4.

Table 11.4 Schematic diagram for resuscitation of sepsis[36]

Suspected case of sepsis

Assess and maintain	
Airway:	Oxygenation
Breathing:	Intubation and ventilation
Circulation	

→ Sedation, paralysis
Peripheral and central venous lines
Urinary catheterization
Advanced monitoring
-Arterial line
-Pulmonary catheter
Blood investigations and culture
Broad-spectrum antibiotics

• CVP < 8 mm Hg:	Build up to 8-12 mm Hg by Crystalloids and colloids
• PCV to 30%:	RBC transfusion
• MAP > 65 mm Hg:	Vasopressors and inotropic support
• $ScvO_2$ < 70%:	cardiac output by inotropes

Ventilatory support:	Follow ARDS net protocol
Hemodynamic support:	Vasopressors and inotropes
Renal support:	Hemofiltration and hemodialysis
Iradication of infection:	Source control and antibiotics

REFERENCES

1. Jean-Louis Vincent, Carla Marie Clausi, Alejandro Bruhn: Novel therapies in critically ill septic patients, In Sepsis, Eds: Guillermo Ortiz Ruiz, Perafan, Faist, Castell, 2nd edn, Springer publication, USA 2006;25-32.
2. Robert S. Munford. Severe sepsis and septic shock, chapter 265, In Harrison's Principles of internal medicine, Eds : Fauci, Kasper, Longo, Braunwald, Hauser, Jameson , 17th edn., The McGraw Hill Publishers, USA 2008;1695-1702.
3. Phillip Dellinger R, Mitchell Levy, Jean Carlet, et al. Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock. Intensive Care Medicine 2008;34:17-68.
4. Okorie Nduka, Joseph E Parrillo. Pathophysiology of septic shock. Crit Care Clinics 2009;25:677-702.
5. Levy MM, Fink MP, Marshal JC, et al. International sepsis definition conference. Crit Care Medicine 2003;31:1250-56.
6. Udwadia F, Intra-abdominal sepsis. Chapter 14.2: In Principles of Critical care, first edn., Oxford University Press, New Delhi 1995;423-31.
7. Dietmar Wittmann, Alonzo Walker, Robert Condon. Peritonitis and intra-abdominal infection. Chapter 32, In Schwartz's Principles of Surgery; Ed S.Charles Brulicardi, 8th edn., McGraw Hill USA 2008;1449-80.
8. Daniel G Remick. Pathophysiology of Sepsis. American J Pathology 2007;170(50):1435-44.
9. Vincent JL. Recent developments in septic shock, chapter No. 10, In Recent developments in Anesthesia and Analgesia; Ed A P Adams, J N Cashnan, 19th edn, Churchill- Livingstone, London 1995:167-89.
10. Lefering R, Goris JA, Nieuwenhoven, Neugebauer. Revision of multiple organ failure score. Langenbeck's Arch Surg 2002;387:14-20.
11. Hulsewe Karel, Hulst Rene, Ramsay Graham, et al. Pulmonary glutamine production: Effects of sepsis and pulmonary infiltrates. Intensive Care Medicine 2003;29 (10):1833-6.
12. Bailey TC, Cavanagh C, Mehta S, Lewis JF, Veldhuizen. Sepsis and hyperoxia effects the pulmonary surfactant system in wild-type and iNOS knockout mice. European Respiratory Journal 2002;20:177-82.
13. Malarkkan N, Snook NJ, Lumb AB. New aspects of ventilation in acute lung injury. Anesthesia 2003;58:647-67.
14. Schrier RW, Wang Wei. Acute renal failure and sepsis. The new England Journal of Medicine 2004;351:159-69.
15. Remick Daniel. Pathophysiology of sepsis. American J of Pathology 2007;170(5):1435-44.
16. Dabrowski GP, Steinberg SM, Ferrara JJ, et al. A critical assessment of endpoints of shock resuscitation. Surg Clin North Am 2000;80:825-43.
17. Vincent JL, DuFaye P, Ber RE, et al. Serial lactate determinations during circulatory shock. Critical Care Medicine 1983;11:449-51.
18. Serum lactate as a predictor of mortality in patients with infection; Intensive Care Medicine 2007;33:970-7.
19. Emanuel Rivers, Bryant Nguyen,Suzanne Havstad, Julie Ressler, Alexandria Muzzin, et al. Early goal directed therapy in the treatment of severe sepsis and septic shock. The New England Journal of Medicine 2001;345(19):1368-77.
20. Birgitte Brandstrup, et al. Effect of IV fluid restriction on postoperative complications – comparison of 2 peri-operative fluid management. Annals of Surgery 2003;238 (5):641-48.
21. Lopez MR, Oliveira MA, Pareira, Lemos, et al. Goal directed fluid management based on pulse pressure variation monitoring in high-risk surgery: A pilot randomized controlled trial. Critical Care 2007;11(5):R 100.
22. Andrew Webb. Crystalloid or colloid for resuscitation. Are we any wiser? Critical Care 1999;3:R25-R28.
23. SAFE study. New Engl J Med 2004;350:2247-56.
24. Anand Kumar. Optimizing antimicrobial therapy in sepsis and septic shock. Critical Care Clinics 2009;25:733-51.
25. Steven Hollenberg. Inotrope and vasopressor therapy in septic shock. Critical Care Clinics 2009;25:781-802.
26. Maria Rudis. Update on vasopressors and inotropes in septic shock. Journal of Pharmacy Practice 2002;5(2):124-34.
27. Bellomo R, Giantomasso DD. Noradrenaline and kidney-friends or foe?, Critical Care 2001;5(6):294-8.
28. Martin C, Viviand X, Leone M, et al. Effect of norepinephrine on the outcome of septic shock. Critical Care Medicine 2000;28(8):2758-65.

29. Andrea Morelli, Christian Ertmer, Sebastian Rehberg, Matthias Lange. Phenylephrine versus norepinephrine for initial haemodynamic support of patients with septic shock in randomized controlled trial. Critical Care 2008;12:R143.
30. Kulkarni SV, Anita Naik, Subramanian N , APACHE – II scoring system in perforative peritonitis. The American Journal of Surgery 1994;(4):549-52.
31. Sielenkamper AW, Hugo Van Aken. Epidural anesthesia in sepsis: Too early to judge a new concept. Intensive Care Medicine 2004,30:1987-9.
32. Daudel Fritz, Hendrik F, Martin W, Stubbe, et al. Continuous thoracic epidural anesthesia improves gut mucosal microcirculation in rats with sepsis. Shock 2007; 28(5):610-4.
33. Annane, Bellissant, Bollaert, Briegel, et al. Corticosteroids for severe sepsis and septic shock: A systematic review and meta-analysis. BMJ 2004;329:480 ref 2.
34. Gabriella de durante, Monica Turco, Laura Rustichini, Patrizia Cosimini, et al. ARDSnet lower tidal volume ventilator strategy may generate intrinsic positive end-expiratory pressure in patients with ARDS. American J of respiratory and critical care medicine 2002;165:1271-4.
35. Malarkhan N, Snnok NJ, Lumb AB, New aspects of ventilation in acute lung injury Anesthesia 2003; 58: 647-7.
36. Bryant Nguyen H, Emanuel Rivers, Fredrick Abrahamian, Gregory Moran, et al. Severe sepsis and septic shock: Review of the literature and emergency department management guidelines. Annals of Emergency Medicine 2006;48(1):28-54.

12 Gastrointestinal Bleeding

Amala G Kudalkar, Devendra W Thakare

KEY POINTS

- Acute gastrointestinal (GI) Bleeding is a common major medical and surgical emergency.
- It constitutes an emergency only when gastrointestinal bleeding is acute.
- It manifests as hematemesis, melena or hematochezia.
- Note prior history of bleeding, the presence of liver disease and drug usage as well as the exact nature of the bleeding.
- Initial triage and assessment to identify life-threatening hemodynamic compromise and initiating appropriate resuscitation.
- Airway, breathing and circulation, recognition of shock and early aggressive fluid resuscitation is priority.
- Endoscopy and other investigations are chosen carefully for their usefulness. Control of bleeding is then tailored to the diagnosis and is usually with drugs, endoscopy, angio-embolization and surgery in that order.
- Peptic ulcers account for most cases of upper GI bleeding, but bleeding from varices has a much higher case-fatality rate and always demands aggressive treatment.
- Combinations of endoscopic therapy comprising of an injection of sclerosants coupled with either a thermal or mechanical treatment are recommended in preference to single modalities.
- Nonvariceal upper gastrointestinal hemorrhage not controlled by endoscopy should be treated by repeat endoscopic treatment, selective arterial embolization or surgery.
- Pharmacological therapy like acid suppression and agents to arrest bleeding.
- Surgery is indicated in massive, acute bleeding not amenable to endoscopic therapy or where endoscopic therapy fails to control active bleeding.
- The majority of patients with variceal bleeding have chronic liver disease.
- Variceal hemorrhage has a poor prognosis and prompt recognition and treatment are required.
- The initial approaches to treating patients presenting with variceal hemorrhage are endoscopic treatment, pharmacological therapy and balloon tamponade.
- Balloon tamponade is a temporary measure that can control massive variceal bleeding which does not respond to endoscopic therapy.
- After endoscopic treatment patients should receive vasoactive drug treatment and antibiotic therapy.
- Definitive endoscopic procedures, TIPSS or surgical treatment (esophageal transaction, surgical shunts like portacaval shunt) and liver transplantation can subsequently be administered once the patient has been stabilized.
- The severity of acute lower gastrointestinal bleeding (LGIB) is variable, but overall mortality is low.
- Acute lower gastrointestinal bleeds mostly stop spontaneously.
- The early use of colonoscopy and use of computed tomography scanning, computed tomographic angiography or digital subtraction angiographic embolization.
- Localized segmental intestinal resection or subtotal colectomy is recommended for the management of colonic hemorrhage uncontrolled by other techniques.
- Anesthesia may be in terms of monitored anesthesia care, mild to deep sedation or total intravenous anesthesia to general anesthesia according to the nature of procedure undertaken.
- Role of anesthesiologist involves assessment and resuscitation of the patient, management of airway, providing monitored anesthesia care for endoscopic and radiological procedures, anesthesia for shunt and surgical procedures.
- Prognosis depends on severity of the bleeding, the age of the patient, comorbidities, the diagnostic category, the endoscopic features and whether continued or recurrent bleeding is a feature.

INTRODUCTION

Acute gastrointestinal (GI) bleeding (or hemorrhage) is a common major medical and surgical emergency with a reported incidence of 170 cases per 1,00,000 population.[1] Over the years there have been a number of improvements in diagnosis and management of GI bleeding. The increased involvement of acute care specialists during resuscitation and follow-up, improved technique of diagnostic and therapeutic endoscopy, advances in diagnostic and therapeutic radiology, and use of powerful ulcer healing drugs, more selective and less invasive surgical approaches may all improve outcome for patients. These changes have altered the diagnostic and treatment pathways for patients presenting with nonvariceal and variceal upper GI bleeding and those with acute colonic bleeding.

National clinical guidelines for management of acute upper and lower GI bleeding published in September 2008 by Scottish Intercollegiate Guidelines Network provide recommendations based on current evidence.[2] They deal with the management of bleeding that is of sufficient severity to lead to emergency admission to hospital and serve as a reference to many institutes in Europe.

DEFINITIONS

Before proceeding with the topic, it is important to get familiarized with the related terms.

Upper and Lower Gastrointestinal Bleeding

GI bleeding is classified by its origin, from either the upper or the lower gastrointestinal tract, anatomically demarcated by the ligament of Treitz.[3] It often originates from the upper GI tract. Peptic ulcer disease (PUD) and variceal hemorrhage are the most common etiologies.

Most lower GI bleeding originates from the colon, with diverticula and angiodysplasias accounting for the majority of cases.[1]

It constitutes an emergency only when gastrointestinal bleeding is acute.

Hematemesis

Hematemesis is vomiting of blood from upper GI tract or vomiting the swallowed blood from a source in the nasopharynx. Bright red hematemesis usually implies active hemorrhage from the esophagus, stomach or duodenum. Coffee-ground vomitus refers to vomiting of brownish black material which is altered blood (acid hematin). It implies that bleeding has ceased or has been relatively modest.[4]

Melena

Melena is passage of black tarry stools usually due to acute upper GI bleeding but occasionally from bleeding within the small bowel or right side of the colon.[4]

Hematochezia

Hematochezia is passage of fresh or altered blood per rectum mixed with stools usually due to colonic bleeding.

OCCULT BLEEDING

Bleeding that is not symptomatic and is found incidentally in stools or with screening is called occult bleeding.

Shock

Shock is circulatory insufficiency resulting in inadequate oxygen delivery leading to hypoperfusion and tissue hypoxia. In context of GI bleeding, it is usually hypovolemic in nature.

Varices

Varices are abnormal distended veins usually in esophagus, less frequently in the stomach (gastric varices) or other sites (ectopic varices). They occur as a consequence of liver disease. Bleeding is characteristically severe and life threatening. The size of varices and their propensity to bleed is directly related to the portal pressure.

Endoscopy

Endoscopy is the visualization of the inside anatomy of the GI tract using telescope. Examination of the upper GI tract (esophagus, stomach and duodenum) is known as gastroscopy or upper gastrointestinal endoscopy. Examination of colon (large bowel) is called colonoscopy.

Triage

Triage is a system of initial assessment and management whereby a group of patients is classified according to the seriousness of their injuries or illnesses so that treatment priorities can be allocated between them.

UPPER GASTROINTESTINAL BLEEDING

Etiology

There are two broad etiological categories:

The **ulcer group** and the **variceal group**.

The common etiological factors for ulcer group are:

- *Helicobacter pylori* infection

- Ingestion of aspirin and nonsteroidal anti-inflammatory drugs (NSAID) causing ulcer disease and erosions
- Ulceration may also occur at the site of surgical enterostomies (stomal ulcers)
- In association with the Zollinger-Ellison syndrome.

Peptic ulceration at specific sites is associated with major hemorrhage due to the anatomical relation of major arteries—the posterior wall of the first part of the duodenum (gastroduodenal artery), the lesser curve of the stomach (left gastric artery) and posterior wall of stomach (splenic artery).

The Variceal Group

- Alcoholic liver disease and hepatitis
- Obstructive jaundice and portal hypertension due to any cause. They rarely affect the stomach and the remaining gastrointestinal tract.

Miscellaneous

- Mallory-Weiss tears are mucosal lesions at the esophagogastric junction associated with profuse vomiting; hematemesis occurs which is usually minor and always self-limiting
- *Malignancy:* The commonest variety is adenocarcinoma of the stomach and gastric lymphoma, but acute bleeding is an unusual presentation
- Benign tumors such as the stromal tumors (previously described as leiomyomas) which may bleed when the mucosal surface ulcerates, angiodysplasia (and other vascular lesions), aortoduodenal fistula, hemobilia, and trauma.[3]

Whenever a patient with GI bleeding comes to hospital, the management should be on the following lines:

1. Initial rapid assessment to know the gravity of the situation.
2. Initial resuscitation and stabilization of the patient.
3. Monitor the patient for ongoing blood loss.
4. Careful history and examination to know the likely etiology, any comorbid condition and complications. This allows for risk stratification and triage.
5. Localization of lesion.
6. Initiation of definitive therapy.

Initial Assessment

Initial assessment should be swift, individualized and should provide a quick assessment of patient's hemodynamic status and need for urgent resuscitation. It includes:

- Assessment of airway, breathing and circulation
- Assessment of magnitude of deficit and
- Ongoing loss.

Monitoring the hemodynamic parameters will help in this. A patient in shock will have following findings depending on its severity:

- A rapid pulse (tachycardia)
- Low blood pressure (hypotension) < 90 mm Hg in systolic position or a postural drop
- Anxiety or confusion (altered mentation)
- A high respiratory rate (tachypnea)
- Cool clammy skin (peripheral vasoconstriction)
- Low urine output (oliguria).

A point to note is that patient with normal blood pressure may still be in shock and will require resuscitation. At this juncture, a note is also made to quickly assess the additional risk factors such as:

- Age > 60 years
- Concomitant disease of other organs such as kidney, heart, liver and lungs
- Presence of malignancy
- Whether bleeding occurred during hospitalization for other purpose.

Resuscitation

Similar to initial assessment, even the resuscitation should occur in the same order. Opening airway and supporting breathing if required and normalizing circulation. Patient who has lost a considerable blood volume may be in a state of semi-consciousness with obtunded airway reflexes. The actual bleeding that has occurred is much more than the apparent bleeding as major portion is retained in stomach. Also, the presence of blood and clots in the stomach makes the patient more prone for vomiting. This poses a significant risk of pulmonary aspiration of blood in case of upper GI bleeding. With continued bleeding, the laryngoscopic visualization of the cords and intubation may become difficult.

Assessment and resuscitation should go hand in hand. After initial quick assessment, preferably two large bore (16G) intravenous lines are secured and blood is collected and sent for Hemoglobin, CBC including platelet count, LFT, RFT and coagulation profile as well as grouping and cross matching. Airway is secured and ventilation supported if required and volume resuscitation is begun. It is advisable to insert a central venous line as peripheral veins are often collapsed and also because it serves as a guide for intravenous therapy.

It is also indicated in elderly patients who are at the risk of over-infusion. A urinary catheter is inserted to know the perfusion. A wide bore nasogastric tube is inserted and the stomach is sucked of all blood and cold saline washes are given. Little ethamsylate added to the saline wash helps in controlling the bleeding.

Monitoring is also started simultaneously. Standard monitors include, cardioscope, pulse oximeter, blood pressure especially noninvasive, central venous pressure, urine output and temperature. Further monitoring may be added as per the availability and the need of the patient.

Initial volume resuscitation starts with rapid infusion of 1 to 2 liters of crystalloids preferably lactated Ringer's solution as it closely approximates the constitution of extracellular fluid. The response of the patient to volume therapy is noted. Lack of any response to volume therapy indicates either ongoing blood loss or cardiac insufficiency. In the later case, there will be signs of left ventricular dysfunction. Such patients would warrant early blood transfusion. Whether to infuse crystalloids or colloids always remains a controversial issue. Crystalloids have the advantage of easy availability, no allergic reactions, and easy diffusivity into the various compartments of the body. However, the intravascular retention time is limited and there is a lot of tissue edema. Also, the amount of crystalloids required will be much more than the colloids. Since these patients have lost their RBCs, ideally they should receive packed cell transfusions. But unless the cause of bleeding is treated, there will be continued blood loss.

The BLEED Classification Uses Five Criteria[5]

- Ongoing bleeding
- A systolic blood pressure of < 100 mm Hg
- PT > 1.2 times control
- Altered mental status
- An unstable comorbid disease.

Presence of any one of these factors increases the risk by almost three times.

CLINICAL FEATURES

- Patient presents with hematemesis or 'coffee-ground' vomiting
- *Melena:* It usually indicates that bleeding has occurred at a site above the cecum. Frank hematemesis indicates a severe bleed; melena will always follow a significant bleed
- *Signs of hypovolemia:* Tachycardia, peripheral vasoconstriction, sweating, hypotension (including a postural drop), tachypnea, and a low central venous pressure. Patients may experience hypotension and tachycardia if blood loss exceeds approximately 25 percent of the total blood volume (1500 ml in adults)
- If orthostatic hypotension is present, characterized by decreases in systolic blood pressure of 10 to 20 mm Hg and corresponding increases in heart rate, the patient usually has hematocrit less than 30 percent.[7] However, in acute setting the hematocrit may be normal as there is loss of both RBCs as well as plasma and not enough time has passed for intravascular shift of plasma or fluid resuscitation
- In rapid bleeding, symptoms of hypovolemia may precede hematemesis or melena. These include syncope, shock, and even death
- In patients with rapid hemorrhage, usually accompanied by shock, fresh blood may be passed per rectum (hematochezia) and may thus be difficult to distinguish from lower gastrointestinal hemorrhage. A mix of fresh blood and melena may indicate a lesion in the lower small intestine (e.g. Meckel's diverticulum).[3]

In most cases, the causative lesion will not be known until diagnostic endoscopy is undertaken.

DIAGNOSIS

History

A good history from the patient may give clues as to the site and cause of GI hemorrhage. Important points include:

- Prior history of bleeding episodes
- Exact nature of the bleeding—time of onset, number of episodes, amount vomited every time. This is important in estimating the amount of blood lost
- Color of the vomitus, precipitating factors such as prior vomiting, etc. which will help in estimating the site and cause of the bleeding
- History suggestive of liver disease, alcohol consumption
- History of drug usage: aspirin, nonsteroidal anti-inflammatory drugs, selective serotonin reuptake inhibitors. These drugs are associated with GI mucosal erosions. Patients on warfarin or LMWH are also at risk of bleeding.

Investigations

Acute GI bleeding is a clinical diagnosis. The cause of bleeding and the severity of bleeding should be determined by means of investigations.

- *Hemoglobin and hematocrit:* The initial hemoglobin estimation is not a useful indicator of the volume of

blood lost as the hematocrit may be normal early in the course of acute hemorrhage because of the insufficient time for equilibration of the plasma volume. Equally, the hemoglobin may be low in a patient with iron deficiency anemia resulting from chronic hemorrhage who presents with a small, acute bleed. The hemoglobin and haematocrit after volume resuscitation are more useful

- Platelet count and coagulation studies (BT/CT/PT/INR/APTT) are important to exclude a bleeding disorder, to rule out the medical cause of bleeding and are of particular relevance in patients receiving therapeutic anticoagulants and in those with liver disease[5]
- The blood urea nitrogen concentration is usually higher than 40 mg/dl because of the absorbed nitrogen load in the small intestine[6]
- Liver function tests: Complete LFT with proteins and enzymes to assess liver function
- Chest X-ray: To rule out pulmonary aspiration of blood in case of obtundation of airway reflexes.

Risk Factors Associated with Poor Outcome

The following factors are associated with a poor outcome, defined in terms of severity of bleed, uncontrolled bleeding, rebleeding, need for intervention and mortality.[3]

These factors should be taken into account when determining the need for admission or suitability for discharge.

- Age: Mortality due to UGIB increases with age across all age groups (> 60 years)
- Comorbidity: The absence of significant comorbidity is associated with mortality as low as 4 percent
- Liver disease: Cirrhosis is associated with a doubling of mortality and much higher risk of interventions such as endoscopic hemostasis or transfusion. The overall mortality of patients presenting with varices is 14 percent
- Inpatients have approximately a threefold increased risk of death compared to patients newly admitted with GI bleeding. This is due to the presence of comorbidities in established inpatients rather than increased severity of bleeding
- Initial shock (hypotension and tachycardia) is associated with increased mortality and need for intervention
- Continued bleeding after admission is associated with high-risk of intervention and up to a 50-fold increased mortality
- Elevated blood urea is associated with a need for intervention.

Nonsteroidal anti-inflammatory drugs (NSAIDs) and anticoagulants do not adversely affect the clinical outcomes of patients presenting with UGIB.

Pre-endoscopic Risk Assessment

Simple and widely validated scoring systems to identify patients at high-risk of rebleeding, death and active intervention are needed for optimum management. The Rockall scoring system was principally designed to predict death-based on a combination of clinical and endoscopic findings. The details of the marking are given in Table 12.1.[2,7]

The initial (pre-endoscopic) Rockall score is derived from age (0–2 points), shock (0–2 points) and comorbidity (0–3 points).

If the initial (pre-endoscopic) score is above 0, there is a significant mortality (score 1: predicted mortality 2.4%; score 2: predicted mortality 5.6%) suggesting that only those scoring 0 can be safely discharged at this stage. As the score increases, the risk of dying and that of rebleeding increases.

Post-endoscopic Risk Assessment

The full Rockall score comprises the initial score plus additional points for endoscopic diagnosis (0–2 points),

Table 12.1: Rockall numerical risk scoring system[2,7]

Variable	*Score 0*	*Score 1*	*Score 2*	*Score 3*
Age	< 60 years	60–79 years	≥ 80 years	
Shock	No shock	Pulse > 100	SBP < 100	
Comorbidity	Nil major		Cardiac failure, ischemic heart disease, any major comorbidity	Renal failure, liver failure, disseminated malignancy
Diagnosis	Mallory–Weiss tear, no lesion of SRH	All other diagnosis	GI malignancy	
Endoscopy	None or dark spot only		Blood, adherent clot, spurting vessel	

SBP – Systolic blood pressure; SRH – Stigmata of recent hemorrhage

and endoscopic stigmata of recent hemorrhage (SRH) (0–2 points) giving a maximum score of 11 points.

The Blatchford risk score was derived to predict death and the need for treatment (transfusion, endoscopic treatment, surgery). The full score was validated internally on 197 patients and performed better than the Rockall score in predicting the need for treatment.[8]

Assessment and Triage (Summary)

All patients presenting with acute upper gastrointestinal bleeding should have an initial (pre-endoscopic) rockall score calculated. Patients with a rockall score of 0 should be considered for nonadmission or early discharge with outpatient follow-up.

In patients with initial (pre-endoscopic) rockall score >0 endoscopy is recommended for a full assessment of bleeding risk.

Patients with a full (postendoscopic) rockall score < 3 have a low risk of rebleeding or death and should be considered for early discharge and outpatient follow-up.

Patients with score of 0, 1 or 2 have a lower risk of hemorrhage whereas approximately 50 percent of patients with a postendoscopy score of 8 or more will rebleed.[9]

The Rockall score should be taken into account with other clinical factors in assigning patients to different levels of care. It should not be used in isolation to assign patients to high-dependency care.

The Treatment of GI Bleeding Essentially Consists of

- Initial resuscitation and airway management
- Attempts to reduce bleeding by:
  - Pharmacological interventions
  - Local pressure
  - Surgical interventions
- Prevention of rebleeding.

EARLY PHARMACOLOGICAL MANAGEMENT

Unselected Patients with Gastrointestinal Bleeding Before Endoscopy

Maintaining gastric pH above 6 optimizes platelet aggregation and clot formation.[10] Patients at high-risk for rebleeding receive endoscopic therapy to achieve hemostasis and are subsequently treated with high-dose acid suppression to promote the formation of blood clots over the arterial defect that is responsible for bleeding. Although there is evidence of improved clinical outcome associated with postendoscopic pharmacological management of patients at high-risk of rebleeding, there is a lack of evidence to support pre-endoscopic treatment with proton pump inhibitors (PPI).[11]

Proton pump inhibitors should not be used prior to diagnosis by endoscopy in patients presenting with acute upper gastrointestinal bleeding.

Early Endoscopic Intervention

Endoscopy is an effective intervention for acute GI bleeding. The optimal timing of endoscopy has not been clearly established and there is no consistent definition of an "early" or "delayed" procedure. The literature describes early endoscopy as ranging from one to 24 hours after initial presentation.[12]

Timing of Endoscopy

The decision to carry out endoscopy will depend upon, the quantity of blood lost, the ongoing bleeding and the hemodynamic condition of the patient. Endoscopy is usually undertaken within 24 hours of presentation. Early endoscopy allows risk to be estimated for bleeding patients.

A small subgroup of patients is unstable because of active bleeding (active hematemesis and/or melena, tachycardia and/or hypotension). Early endoscopy and endoscopic therapy (<24 hours from admission) is associated with reduced transfusion requirements, a reduction in rebleeding and a lower need for surgery compared to patients in whom endoscopy is delayed.[13]

Optimum resuscitation is essential before endoscopy in order to reduce the potential cardiorespiratory complications of the procedure.

MANAGEMENT OF NONVARICEAL UPPER GASTROINTESTINAL BLEEDING

The most common cause of significant nonvariceal bleeding is universally reported to be peptic ulcer disease, which measures for up to half of all cases found at emergency endoscopy.

- *Risk stratification:* Endoscopic stigmata are integral to the Rockall scoring system (Table 12.1). Patients who are shocked and have active bleeding at endoscopy have an 80 percent risk of continued bleeding or rebleeding unless endoscopic intervention is undertaken
- *Endoscopy:* Certain lesions are at high-risk of developing rebleeding such as active arterial bleeding, nonbleeding visible vessels or an adherent blood clot. They should be treated endoscopically since only these are at risk of further bleeding.[14] Endoscopic

therapy should only be delivered to actively bleeding lesions, nonbleeding visible vessels and, when technically possible, to ulcers with an adherent blood clot.

Injection

Injection of sclerosants (polydochanol, sodium tetradecyl sulphate (STD) or ethanolamine) and absolute alcohol is also effective but is associated with a significantly increased risk of complications including mucosal perforation and necrosis compared with adrenaline.[14]

Thermal

Coagulation using the heater probe or multipolar coagulation has similar clinical efficacy to injection.

Mechanical

Definitive hemostasis was higher with clipping than injection. Use of clips significantly reduced rebleeding compared with injection and the need for surgery. Clipping and thermocoagulation had comparable efficacy.

Combination Therapies

Combinations of endoscopic therapy are superior to the use of a single modality therapy, and combination treatment does not increase the risk of complications. Combinations of endoscopic therapy comprising an injection of sclerosants coupled with either a thermal or mechanical treatment are recommended in preference to single modalities.

Repeat Endoscopy

Endoscopy and endotherapy should be repeated within 24 hours when initial endoscopic treatment was considered suboptimal (because of difficult access, poor visualization, technical difficulties) or in patients in whom rebleeding is likely to be life-threatening.

Rebleeding Following Endoscopic Therapy

Patients who rebleed after endoscopic therapy have increased mortality and require urgent intervention. The use of digital subtraction angiography to assist in the localization of bleeding point and simultaneous superselective coil transcatheter embolization using coils and polyvinyl alcohol, and gelatin sponge is reported.

Nonvariceal upper gastrointestinal hemorrhage not controlled by endoscopy should be treated by repeat endoscopic treatment, selective arterial embolization or surgery.

- Pharmacological therapy.[2]

ACID SUPPRESSION AND AGENTS TO ARREST BLEEDING

Acid Suppression

Patients at high-risk of rebleeding (active arterial bleeding, nonbleeding visible vessels, adherent clots) receive endoscopic therapy to achieve hemostasis. High-dose intravenous proton pump inhibitor therapy (e.g. omeprazole or pantoprazole 80 mg bolus followed by 8 mg/hour infusion for 72 hours) should be used in patients with major peptic ulcer bleeding (active bleeding or nonbleeding visible vessel) following endoscopic hemostatic therapy.

Tranexamic Acid, Somatostatin and its Analogs

The role of fibrinolytic inhibitors, somatostatin and its analogs in gastrointestinal bleeding is unclear.

There is insufficient evidence to make a recommendation for the use of tranexamic acid, somatostatin and its analogs in the treatment of non-variceal gastrointestinal bleeding.

Continuation of therapy for other medical conditions Medicines known to increase the risk of upper gastrointestinal complications should, where possible, be given in monotherapy and at the lowest effective dose to minimize the risk of upper gastrointestinal complication.

Nonsteroidal Anti-inflammatory Drugs (NSAIDs) and Cox-2 Inhibitors[2]

Patients with healed bleeding ulcers who test negative for *Helicobacteri pylori* require concomitant proton pump inhibitor therapy at the usual daily dose if nsaids, aspirin or Cox-2 inhibitors are indicated.

In patients in whom cardiovascular risk is a concern, naproxen with a proton pump inhibitor is recommended when alternative analgesic therapies fail. Cox-2 inhibitors are not recommended in patients with cardiovascular risk.

Aspirin and Clopidogrel

Aspirin and NSAIDs should be discontinued when patients present with peptic ulcer bleeding. Once ulcer healing and eradication of *Helicobacter pylori* are confirmed, aspirin and NSAIDs should only be prescribed if there is a clear indication.

Selective Serotonin Reuptake Inhibitors

Selective serotonin reuptake inhibitors (SSRI) should be used with caution in patients who have an increased risk of gastrointestinal bleeding, especially in patients taking NSAIDs or aspirin. A non-SSRI antidepressant may be an appropriate choice in such patients.

Anticoagulants and Corticosteroids

Anticoagulants or corticosteroids should be used with caution in patients at risk from gastrointestinal bleeding, especially in those taking aspirin or NSAIDs.[15]

SURGICAL MANAGEMENT

Surgery is indicated in massive, acute bleeding not amenable to endoscopic therapy or where endoscopic therapy fails to control active bleeding. Many units would attempt a second endoscopic therapy especially in young patients before resorting to surgery. There is no place for a third attempt at endoscopic therapy and surgery is indicated. In cases where endoscopic therapy has failed and surgery is deemed to be exceptionally high-risk, visceral angiography may allow for embolization (e.g. the gastroduodenal artery in duodenal ulcer).[3]

Surgical treatment of nonvariceal upper GI bleeding (oversewing an ulcer, gastrectomy for diffuse hemorrhagic gastritis) is used in patients who continue to bleed despite optimal supportive therapy and in whom endoscopic coagulation is unsuccessful.[6]

Management of Acute Variceal Upper Gastrointestinal Bleeding

Variceal hemorrhage occurs from dilated veins (varices) at the junction between the portal and systemic venous systems. These tend to be in the distal esophagus and/or the proximal stomach, but isolated varices may be found in the distal stomach, large intestine and rectum. The majority of patients with variceal bleeding have chronic liver disease. Patients with variceal hemorrhage will often present with overt upper GI bleeding with hematemesis and/or melena, but may also present with a decompensation of chronic liver disease including encephalopathy, renal involvement or with anemia.

Variceal hemorrhage has a poor prognosis and prompt recognition and treatment are required.

The outcome for patients with variceal hemorrhage is closely related to the severity of the underlying liver disease. The severity of liver disease is stratified by Child-Pugh's grade (Table 12.2). There is evidence that outcomes from variceal hemorrhage are improving over time as new treatment strategies (e.g. variceal band ligation and vasoactive drugs) are introduced.

Table 12.2: Child-Pugh's grading of chronic liver disease

Clinical/laboratory findings	*Score*		
	1	*2*	*3*
Encephalopathy	None	Mild (grade 1–2)	Severe (grade 3–4)
Ascites	None	Mild/Slight	Moderate/Large
Bilirubin (micromol/l)	<34	34–51	>51
Albumin (g/l)	≥35	28–35	<28
Prothrombin prolongation time (secs)	<4	4–6	>6
Or international normalized ratio (INR)	<1.3	1.3–1.5	>1.5

Table 12.3 Child-Pugh's grading

Total Points	*Child-Pugh class*
5–6	A
7–9	B
10–15	C

CHILD-PUGH'S GRADING OF CHRONIC LIVER DISEASE

Chronic liver disease is classified into Child-Pugh's class A to C (Table 12.3), employing the total score from the above table. A total score of 5 to 6 is considered grade A (well-compensated disease); 7 to 9 is grade B (significant functional compromise); and 10 to 15 is grade C (decompensated disease). These grades correlate with one- and two-year patient survival.[16]

Patients presenting with variceal hemorrhage should be assessed and resuscitated as for any other patient with evidence of UGIB. The initial approaches to treating patients presenting with variceal hemorrhage are endoscopic treatment, pharmacological therapy, and balloon tamponade.

Endoscopic Therapy for Acute Variceal Hemorrhage[2]

In patients with suspected variceal hemorrhage endoscopy should be performed once appropriate resuscitation has been undertaken.

Patients with confirmed esophageal variceal hemorrhage should undergo variceal band ligation. Banding may be technically difficult in cases of continued bleeding, and sclerotherapy may then be necessary.

Patients with confirmed gastric variceal hemorrhage should have endoscopic therapy, preferably with cyanoacrylate injection. The other solutions that is injected is sodium tetradecyl sulfate (STDS). It can be injected into the varices or in the paravariceal space.

The problems of including anesthesia in patient with bleeding varices are:

- Anemic, hypovolemic patient
- Full stomach with high-risk for aspiration
- Underlying liver disease
- Derangement of other systems.

The patient should be thoroughly evaluated and simultaneously resuscitated with crystalloids, blood and blood products. Central venous pressure and urine output should be catheterized. Though it is possible to perform variceal injection under only intravenous ketamine anesthesia, it is always better to give complete general anesthesia using rapid sequence injection intubation technique.

Vasoactive Drug Therapy for Acute Variceal Hemorrhage

In clinical practice the decision to use drug treatment is based either on suspicion of variceal hemorrhage or endoscopic confirmation of variceal hemorrhage.

Vasoactive drug Therapy Prior to Endoscopy

In the studies reviewed, vasoactive drug treatment was initiated prior to an endoscopic diagnosis of variceal hemorrhage.[2] Most patients went on to receive endoscopic treatment with either variceal band ligation or sclerotherapy.

Prior to endoscopic diagnosis, terlipressin should be given to patients suspected of variceal hemorrhage. The dose of terlipressin is 1 to 2 mg intravenously repeated at four and eight hours after the initial treatment. Somatostatin can be used as 250 mcg/hour after a 250 mcg bolus given intravenously.

The same treatment should be continued after the endoscopic treatment of acute esophageal variceal hemorrhage patients should receive vasoactive drug treatment (terlipressin for 48 hours, octreotide, or high-dose somatostatin each for three to five days).

Antibiotic Therapy

Antibiotic therapy should be commenced in patients with chronic liver disease who present with acute upper gastrointestinal hemorrhage.

Balloon Tamponade

Balloon tamponade (Sengstaken-Blakemore tube or Minnesota balloon) (Fig. 12.1) is a temporary physical or mechanical measure that can control massive variceal bleeding which does not respond to endoscopic therapy.[17] In these cases, bleeding tends to be torrential and resuscitative efforts, though ongoing, continue to fall behind in terms of maintenance of hemodynamic status and oxygen delivery. It is during this difficult circumstance that balloon tamponade can be a life-saving procedure by stabilizing the patient and allow a more definitive decompression procedure to be performed. The older Sengstaken-Blakemore tube, which is a three-lumen tube, is used for this purpose. The newer version of it is the four-lumen Minnesota tube. Both tubes have a gastric balloon, an esophageal balloon and a distal gastric port for aspiration of gastric contents. The Minnesota tube's fourth lumen is a proximal aspiration port above the esophageal balloon whose sole purpose is the aspiration of salivary secretions from the cervical esophagus. Because it lacks this proximal port, the Sengstaken-Blakemore tube requires placement of a proximal orogastric or nasogastric tube.[18]

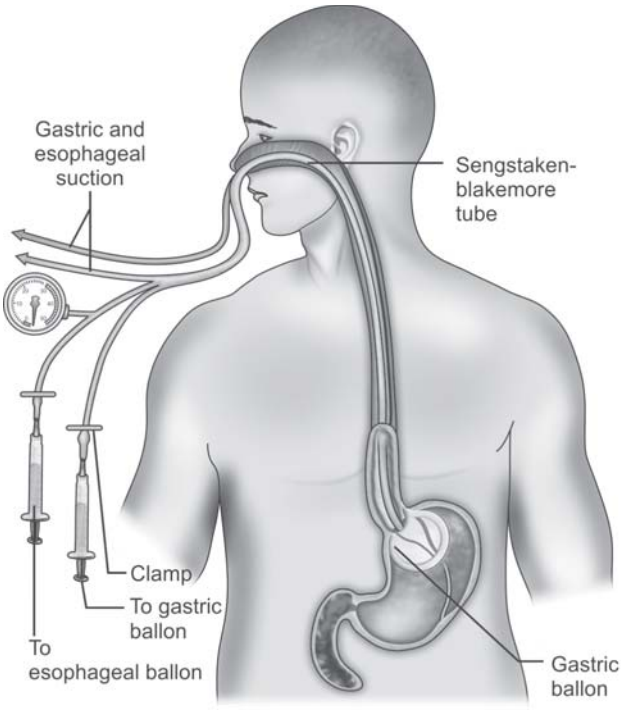


Fig. 12.1: Sengstaken-Blakemore tube

SENGSTAKEN-BLAKEMORE TUBE

Prior to placement of the tube, one should measure from the xiphoid process to the incisors the number of centimeters on the tube necessary to ensure complete placement of the gastric balloon within the stomach. This is usually more than 40 cm. Before insertion, one should check which balloons and aspirating channels connect to which tubes and inflate the balloons (esophageal 150 ml, gastric 250 to 300 ml) with air to confirm proper functioning. The patient is placed in the left lateral

position with the head raised at 45°, a small amount of intravenous sedation, such as midazolam, is given and a mouth guard is inserted. After spraying with local anesthetic, the tube is lubricated and inserted through the mouth. The tube is advanced until the 50 m mark is reached. The stomach balloon is inflated. The tube is then withdrawn until the balloon can be felt to impact in the esophagogastric junction and traction is maintained on the tube while the esophageal balloon is inflated. The patient may experience chest pain during inflation of the esophageal balloon and in consequence require additional sedation. An X-ray that includes both the lower chest and upper abdomen should be taken. After 24 hours the balloons are deflated. The balloon should not be left inflated for more than 48 hours. Institution of balloon tamponade mandates decompression of the varices via TIPS or surgical shunting within that time period.[19]

Securing airway with SB tube in place can be really challenging. Therefore, it is a usual practice to intubate trachea prior as the tube is mostly maintained in place by hitching it tightly and tying to a helmet worn by the patient. Presence of this helmet makes the mask holding and intubation not only difficult but almost impossible. Therefore, whenever patient is going to be intubated, the helmet is removed maintaining the traction on the stomach balloon. Esophageal balloon may have to be released temporarily if there is problem with the advancement of the endotracheal tube. Therefore, it is a common practice to insert the SB tube after the patient's airway has been secured. With this approach there is less risk of aspiration. In younger children the balloon causes tracheal displacement and compression. Nevertheless, it is a very effective means of controlling massive variceal bleeding when other modalities have failed.

MANAGEMENT OF BLEEDING VARICES NOT CONTROLLED BY ENDOSCOPY

On occasion acute variceal bleeding will continue despite the combination of endoscopic therapy and drug therapy. Expert opinion recommends managing such patients in two stages: initial emergency therapy to arrest the blood loss and second line therapy to address the underlying cause.[20]

Rates of hemostasis associated with balloon tamponade are reported to be 80 to 95 percent in patients with either esophageal or gastric varices. The complications of balloon tamponade including pneumonia, esophageal tears and discomfort were noted to be greater than drug treatments or sclerotherapy.[17]

Balloon tamponade is a temporary measure that can control massive variceal bleeding which does not respond to endoscopic therapy.

Definitive endoscopic procedures such as TIPSS (Transjugular intrahepatic portosystemic stent shunting) or surgical treatment (esophageal transection, surgical shunts like portacaval shunt) and liver transplantation can subsequently be administered once the patient has been stablized.

As surgical shunts are rarely performed and require specialized surgical skills, TIPSS should be considered the therapy of choice. TIPSS is recommended as the treatment of choice for uncontrolled variceal hemorrhage.[20]

Finally, esophageal transection is occasionally lifesaving where all other attempts at hemostasis have failed.

Prevention of Variceal Rebleeding

Once acute bleeding is successfully controlled, the recurrence of variceal rebleeding can be as high as 50 percent within the first day of the acute episode and 80 percent within one year. Due to the high-risk of mortality, consideration must be given to secondary prophylaxis of variceal hemorrhage.

Vasoactive Drug and Endoscopic Therapy

Variceal band ligation or sclerotherapy combined with a beta blocker is recommended as secondary prevention for esophageal variceal hemorrhage, in patients unsuitable for variceal band ligation. Combination of nonselective beta blocker and nitrate is recommended as secondary prevention for esophageal variceal hemorrhage.

LOWER GASTROINTESTINAL BLEEDING[2]

Acute lower gastrointestinal bleeds will stop spontaneously although 35 percent will require blood transfusion and 5 percent will require urgent surgical intervention.[3]

Etiology

Most causative lesions are colonic or anorectal and only 3 percent originate in the small bowel. They are:

- Diverticular disease
- Angiodysplasias
- Inflammatory bowel disease (including Crohn's, ulcerative colitis)
- Infectious colitis and ischemic colitis
- Neoplasia, benign anorectal disease and arteriovenous malformations
- Rare causes include radiation injury, Meckel's diverticulum, other small bowel pathology and varices.

Lower gastrointestinal bleeding of modest severity is a common problem in primary care. Rarely bleeding that is of sufficient severity warrants emergency admission to hospital.

Around 25 percent of patients presenting with GI hemorrhage in hospital have bleeding that originates in the lower GI tract. A large majority of these will stop bleeding spontaneously without any specific treatment. These patients should receive resuscitation and transfusion, if required, to restore circulatory volume.

All patients with rectal bleeding should have a full history taken, abdominal examination done and should undergo digital rectal examination and proctoscopy. The initial assessment in case of lower GI bleed also remains same as upper GI bleed.

Diagnosis[3]

A good history from the patient may give clues as to the cause of colorectal hemorrhage. Important points include a prior history of bleeding, the presence of liver disease and drug usage (aspirin, nonsteroidal anti-inflammatory drugs and warfarin) as well as the exact nature of the bleeding—specifically the duration, the color of the blood, the relationship to defecation, whether the blood is mixed with or separate from the stool, an associated change in bowel habit, or mucus discharge.

Timing of Endoscopy and Localizing Bleeding

Most patients who present with hematochezia are investigated when stable. Urgent colonoscopy is only considered in actively bleeding and shocked patients. It should only be done once resuscitation has been optimized. Localization of the site and determination of the cause of bleeding should be determined following the early use of colonoscopy and use of computed tomography scanning, computed tomography angiography, digital subtraction angiography or nuclear scintigraphy.

Interventions

In patients with poor localization and ongoing bleeding, early catheter mesenteric angiography and embolization using superselective techniques is often attempted.

Colonoscopic Hemostatic Techniques

The colonoscopic hemostatic techniques are adrenaline injections, bipolar coagulation or endoscopic hemoclipping.

Embolization

In patients with massive lower gastrointestinal hemorrhage, if colonoscopy fails to define site of bleeding and control hemorrhage, angiographic transarterial embolization is recommended as an effective means of controlling hemorrhage.

Surgery

Localized segmental intestinal resection or subtotal colectomy is recommended for the management of colonic hemorrhage uncontrolled by other techniques.

ANESTHETIC CONSIDERATIONS

Prior to hemostatis or radiological intervention procedure optimization of the patient includes the assessment (features of shock), resuscitation and hemodynamic stabilization of the patient as discussed earlier in this chapter. Anesthesia may be in terms of monitored anesthesia care, mild to deep sedation or total intravenous anesthesia to general anesthesia according to the nature of procedure undertaken.

Detailed history regarding duration and amount of blood loss, past history of similar episodes, history of any drug consumption, other comorbidities, addiction, liver disease should be noted. Physical examination, vitals, mental status, airway compromise, starvation status should be checked. Even though patient is adequately starving, he must be considered as full stomach.

Laboratory investigations hematological, biochemical including liver and kidney function tests and coagulation profile should be done simultaneously.

GENERAL APPROACH TO THE PATIENT WITH ACUTE GI HEMORRHAGE (TABLE 12.4)[1]

Role of Anesthesiologist

- Most procedures are done under oropharyngeal or local anesthesia in adults
- Aspiration prophylaxis
- IV with large bore needle, blood cross matched
- Monitoring
- Children: endotracheal anesthesia is preferred
- Extubate awake.

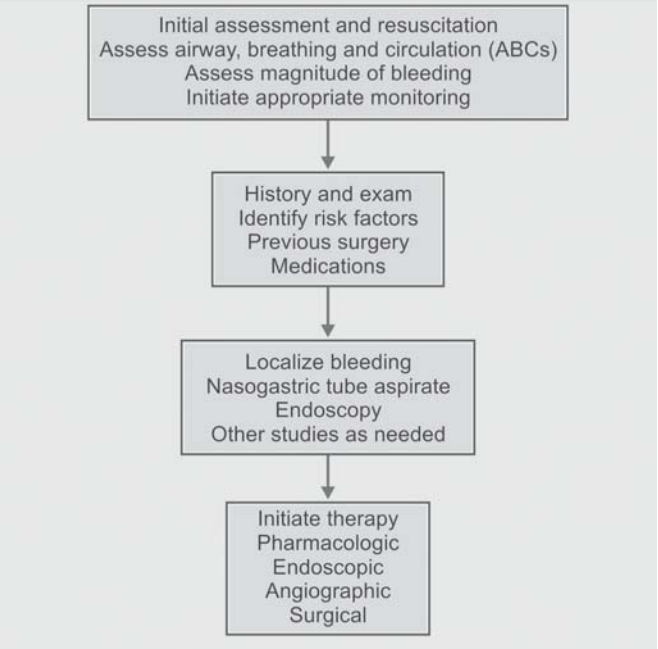
Sedation[21]

In most patients, an appropriate level of sedation can be reached through the use of a benzodiazepine combined with a narcotic. The benzodiazepine provides an amnesic effect while the narcotic provides an analgesic effect.

In older patients and those with decreased pharyngeal sensation, less sedation may be needed. In younger patients and those who may have increased pharyngeal sensation, additional sedation may be needed. This can be done through the use of pharyngeal anesthesia in addition to the standard benzodiazepine/narcotic combination.

Pharyngeal anesthesia includes local anesthetic viscous gargles, regional blocks, etc. anticholinergic agents are given to reduce the secretions.

Table 12.4: **General approach to acute gastrointestinal hemorrhage**



Whether to do the upper GI scopy under sedation or anesthesia would depend upon:

- *Age of the patients:* Children would not allow upper GI endoscopy with only sedation and upper airway analgesia.
- *Hemodynamic stability:* It is better to secure airway and give complete general anesthesia if the patient is unstable with obtunded airway reflexes.
- *Abdominal distension:* In patients with portal hypertension with bleeding varices there may be massive distension of abdomen with ascites. There is considerable increased intra-abdominal pressure. In such patients, it is wiser to secure airway.

Deep sedation can be accomplished through general anesthesia or by adding another medication (droperidol, diphenhydramine, or propofol) to the benzodiazepine/narcotic combination described above. Though propofol seems to be the agent of choice as it obtunds the airway reflexes, one has to remember that the patient might have swallowed considerable blood during the episode of hemetemesis. This will certainly lead to regurgitation when patient loses consciousness. Also the patient may be hemodynamically unstable and hypotensive. Therefore, ketamine seems to be the agent of choice as it maintains the airway reflexes and maintains the peripheral vascular resistance.

Contraindications to the use of propofol include patients with an American Society of Anesthesia status of 3 or 4, patients with potentially difficult airway management and patients at risk of aspiration, particularly those with upper gastrointestinal bleeding.

ANESTHESIA FOR INJECTION OF BLEEDING ESOPHAGEAL VARICES

Anesthetic management of patients with bleeding varices may be complicated by of circulatory shock, liver failure, or coagulopathy. The details of anesthesia management in a patient with liver disease are already discussed at length in Chaper 2 of this book. In addition, the presence of balloon tamponade may make endotracheal intubation difficult and/or impair ventilation.

PREANESTHETIC MANAGEMENT

Preanesthetic preparation of the patient begins with:

- Appropriate volume expansion as guided by central venous pressures.
- Metronidazole and lactulose are introduced into the stomach via the Sengstaken tube to reduce portal-systemic encephalopathy. When this tube is *in situ* both gastric and accessory nasogastric tubes are aspirated before induction.
- Preoxygenation and rapid sequence induction is performed. A cuffed endotracheal tube is placed to secure the airway. Aspiration of secretions is the most common complication of balloon tamponade, and is found in approximately 10 to 20 percent of cases.

It may be difficult to visualize the vocal cords owing to the presence of Sengstaken tube or blood and mucus in the pharynx. So the esophageal balloon is deflated to negotiate the endotracheal tube.

Anesthesia is maintained with oxygen with or without nitrous oxide with increments of analgesics and muscle relaxants permitted by the patients's liver function. If the patient is being maintained without endotracheal tube in place, extreme care has to be taken to prevent aspiration. A powerful dedicated suction with suction catheter attached should be ready. From time to time the endoscopist is encouraged to suck the stomach through the suction channel of the scope. Even if the endotracheal tube is in place, there may be movement of the head during esophagoscopy when the ETT and the anesthetic circuits have to be taken care of. Following an uneventful procedure the patients are extubated once completely awake.

Patients with liver cirrhosis tend to have a plethora of extrahepatic effects of end-stage liver disease which may affect the anesthetic management. The choice of drugs may vary based on the clinical situation of an individual patient.

POSTANESTHETIC MANAGEMENT

The presence of an esophageal leak is ruled out with a chest radiograph taken atleast 8 hours after injection. Esophagitis due to sclerosant may be seen in the postoperative period and the patients may complain of retrosternal pain or soreness in the absence of a leak.

For Definitive Surgical Procedure or Shunts (Liver Cirrhosis)

History, clinical examination, look for signs of liver failure, preoperative optimization to be done.

Monitoring

- ECG, SpO_2, $EtCO_2$
- Invasive BP-ABG/non-invasive BP
- CVP, pulmonary art catheter/TEE
- Urine output, temp. monitor
- Peripheral nerve stimulator
- Serum biochemistry.

Anesthesia Technique

- Premedication: Antisialogogues, sedatives if necessary
- Preoxygenation
- Induction: Thiopentone sodium.
- Ketamine may be used if hemodynamic instability
- Rapid sequence intubation with suxamethonium
- Controlled ventilation with atracurium/vecuronium
- Analgesia: Fentanyl/remifentanyl if available
- Air+ O_2+ isoflurane/desflurane
- Avoid hypo/hypercarbia
- Avoid lactate containing solutions, dextrose solution with saline in moderation minimum 4 units of blood should be available before proceeding surgery. In addition, blood products like plasma, platelets and cryoprecipitate also should be available.
- Fluid and blood warmers, body warmer
- Prophylactic ventilation till all parameters are acceptable.

Postoperative Management

- Pain relief: PCA morphine/fentanyl
- Watch for hepatorenal syndrome, DIC, hepatic encephalopathy
- Avoid IM injections, continued antibiotics
- Look for DIC.

Regional Analgesia

- INR < 1.5
- Dose of LA reduced
- During procedure and epidural block can reduce hepatic blood flow
- Possibilty of precipitous ciculatory collapse as hemodynamic responses are depressed
- Sometimes epidural varices that are present can bleed—risk of hematoma and risk of infection increases.

Prognosis

Prognosis depends on many factors including the severity of the bleed, the age of the patient, the associated comorbidity of the patient, the diagnostic category, the endoscopic features (stigmata of recent hemorrhage), and whether continued or recurrent bleeding is a feature. Overall, the crude mortality for patients presenting to emergency departments with acute upper gastrointestinal hemorrhage is about 10 percent. Most deaths occur in the elderly and those with severe comorbidity. Death in those under the age of 60 with no comorbidity is very low (0.1%) regardless of the severity of the hemorrhage.[3]

Elderly individuals with esophageal variceal bleeding, those with malignancy and those who develop bleeding after hospitalization for other comorbid diseases have an acute mortality rate higher than 30%. Multiple organ system failure, rather than hemorrhage, is the usual cause of death in these patients.[1]

REFERENCES

1. Tavakkolizadeh A, Goldberg J, Ashley S. Acute gastrointestinal hemorrhage. In: Courtney M, Townstead R, Daniel Beuchamp, Mark Evers B, Kenneth L, Mattox (Eds). Sabiston's book of Surgery. (18th edn) India: Saunders publications, imprint of Elsevier 2007; 1199-1223.
2. Scottish Intercollegiate Guidelines Network (SIGN). Management of acute upper and lower gastrointestinal bleeding. A national clinical guideline. Edinburgh (Scotland): Scottish Intercollegiate Guidelines Network (SIGN) 2008;57: (SIGN publication; no. 105). [194 references].
3. Rockall TA, Northfield T. Gastrointestinal bleeding. In: David A Warrell, Timothy M Cox, John D Firth, Edward J, Benz MD, (Eds). Oxford Textbook of Medicine. (4th edn) Oxford: Oxford Press 2003; 1468-70.
4. Gershman G. Approach to the child with gastrointestinal bleeding. In: Lucy MO (Ed). Pediatrics. (1st edn) Philadelphia, Pennsylvania: Elsevier Mosby Company 2005;639-45.
5. Kollef MH, O'Brien JD, Zuckerman GR, Shannon W. BLEED: A classification tool to predict outcomes in patients with acute upper and lower gastrointestinal hemorrhage. Crit Care Med 1997;25(7):1125-32.
6. Tantawy H. Causes of Upper and Lower Gastrointestinal Bleeding. In: Paul AK, Roberta L Hines, Katherine EM.

(Eds.). Stoelting's Anesthesia and Co-existing Disease. 5th edn. Noida (India): Saunders publications, imprint of Elsevier 2008. 324-25.

7. Rockall TA, Logan RFA, Devlin HB, Northfield TC. On behalf of the Steering Committee of the National Audit of Acute Upper Gastrointestinal Haemorrhage , Influencing the practice and outcome in acute upper gastrointestinal hemorrhage Gut 1997; 41: 606-11.
8. Rockall TA, et al. Risk Assessment after acute upper GI hemorrhage. Gut 1996;38:316-21.[abstract]
9. Oppong K, Thompson N, Wallis J. NHS Foundation trust: Protocol for the management for patients admitted with upper GI bleeding. Reviewed 2009; 1-4.
10. Green FW Jr, Kaplan MM, Curtis LE, Levine PH. Effect of acid and pepsin on blood coagulation and platelet aggregation. A possible contributor prolonged gastroduodenal mucosal hemorrhage. Gastroenterology 1978; 74(1):38-43.
11. Leontiadis GI, Sharma VK, Howden CW. Proton pump inhibitor therapy for peptic ulcer bleeding: Cochrane collaboration meta-analysis of randomized controlled trials. Mayo Clin Proc 2007;82(3):286-96.
12. Cooper G, Chak A, Connors A, Harper DL, Rosenthal GE. The effectiveness of early endoscopy for upper gastrointestinal hemorrhage. Med Care 1998;36(4):462-74.
13. Barkun A, Bardou M, Marshall JK. Consensus recommendations for managing patients with nonvariceal upper gastrointestinal bleeding. Ann Intern Med 2003;139(10): 843-57.
14. Rollhauser C, Fleischer DE. Current status of endoscopic therapy for ulcer bleeding. Baillieres Best Pract Res Clin Gastroenterol 2000;14(3):391-410. (pubmed abstract)
15. Shorr RI, Ray WA, Daugherty JR, Griffin MR. Concurrent use of nonsteroidal anti-inflammatory drugs and oral anticoagulants places elderly persons at high-risk for hemorrhagic peptic ulcer disease. Arch Intern Med 1993;153(14):1665-70. (pubmed abstract).
16. Child-Pugh Classification of Severity of Liver Disease. Available from www.medicalcriteria.com.
17. Panés J, Terés J, Bosch J, Rodés J. Efficacy of balloon tamponade in treatment of bleeding gastric and esophageal varices. Dig Dis Sci 1988;33(4):454-9.
18. Galloway JR. Emergency treatment of bleeding esophageal varices. In: Peter J Morris, William C Wood, (Eds). Oxford Textbook of Surgery. (2nd edn) Oxford: Oxford Press 2000; 1216-7.
19. Simon PB. Acute gastrointestinal bleeding. In: Hamilton B, Brian WE, Simon PB, (Eds). Hamilton Bailey's Emergency Surgery. (13th edn) Middlesex UK: A Hodder Arnold Publication 2009; 368-69.
20. Jalan R, Hayes PC. UK guidelines on the management of variceal hemorrhage in cirhotic patients. London: British Society for Gastroenterology; June 2000. Available from http://www.bsg.org.uk/pdf.
21. Curr Opin Gastroenterol: Sedation 2006;22(6):62-9.© 2006 Lippincott Williams and Wilkins Medscape anaesthesiology.

Section IV

Obstetric Emergencies

13 Anesthesia Management of Pregnant Patient for Emergency Surgery

Smita S Lele, Preeti Rustagi

KEY POINTS

- Anesthetic considerations for emergency surgery during pregnancy include concern for the safety of two patients, the mother and the fetus
- The fetus is a passive recipient of anesthesia administered to the mother
- In obstetric anesthesia, anesthetic management involves avoiding uterine relaxation and neonatal depression whereas prevention of premature delivery is the key element while administering anesthesia for nonobstetric surgeries in these patients. Also depression of fetal nervous system is well-tolerated as the transplacentaly transferred drugs can be excreted back to mother for disposal
- A thorough understanding of physiological changes in pregnancy is essential while giving anesthesia to these patients
- The pregnant patient should be transported in the lateral position, and left uterine displacement instituted when positioned on the operating table
- Regardless of the time of last oral intake, all obstetric patients are considered to have a full stomach and are at risk for pulmonary aspiration
- Regional anesthesia is safer in pregnant patient whether presenting for obstetric or nonobstetric surgery and should always be considered whenever possible
- Hypotension is the most common side effect of regional anesthetic techniques and must be treated aggressively with intravenous fluid boluses and vasopressors to prevent fetal compromise
- An experienced anesthesiologist should always be present and difficult intubation cart should be kept ready during obstetric as well as nonobstetric surgeries in these patients
- Preoxygenation with 100 percent oxygen administration for 3 to 4 min (or 4 vital capacity breaths if time is restricted) before a rapid sequence induction takes care of the tendency of pregnant patient to desaturate early
- Teratogenicity of a particular agent is dependent on the class of drug, dose, route of administration and timing of exposure. Drug exposure during the period of organogenesis (approximately days 15 to 70 after the first day of the last menstrual period) is best avoided if possible
- Nearly all parenteral opioid analgesics and sedatives readily cross the placenta and can affect the fetus
- Fetal oxygenation depends on maternal oxygen delivery (cardiac output, arterial oxygen tension and hemoglobin and uteroplacental perfusion. Transient mild-to-moderate decreases in maternal PaO_2 are well-tolerated by the fetus, because fetal hemoglobin has a high affinity for oxygen. Severe maternal hypoxemia results in fetal hypoxia and if persistent, may cause fetal death
- In a pregnant trauma patient the anatomic and physiologic changes associated with pregnancy may cause the clinician to underestimate the hypovolemia
- During neurosurgery, osmotic diuresis, controlled hypotension and hypothermia are commonly induced to decline the intracranial pressure (ICP). In the pregnant patients, those may adversely affect the fetus
- At 32 weeks cardiac output reaches maximum and the incidence of decompensation of a pregnant cardiac patient may peak at that time
- By maintaining higher pump flow rate and pulsatile flow, higher perfusion pressure, normothermic perfusion when feasible, fetal compromise during cardiopulmonary bypass can be minimized
- The anesthetist should also be an active participant in measures that might ameliorate isolated fetal compromise and even avert the need for cesarean section
- Extubation should be delayed until the patient is sufficiently awake to protect her airway from regurgitation and aspiration.

INTRODUCTION

Pregnant patient presenting for emergency surgery is a common scenario. This surgery may be directly or indirectly related to the pregnancy or may be totally unrelated. It may be required during any stage of pregnancy.

Type of Surgery

- Pregnancy related
  Emergency cesarean section
  Cervical incompetence surgery
  Ovarian cyst problems
- Nonpregnancy related
  Acute abdominal problems:
  – Appendicitis
  – Cholecystitis
  Trauma
  Surgery for malignancy
  Major surgeries like cardiac or neurosurgery.

Anesthetic considerations for emergency surgery during pregnancy include concern for the safety of two patients, the mother and fetus. Alterations in maternal anatomy and physiology induced by pregnancy have clinical anesthetic implications and present potential hazards for the mother and fetus undergoing anesthesia.

Additional risks for the fetus include the following:

1. Effects of the disease process itself or of related therapy.
2. Possible teratogenicity of anesthetic agents.
3. Intraoperative perturbations of uteroplacental perfusion and/or fetal oxygenation.
4. Risk of abortion or preterm delivery.[1]

In most circumstances, the fetus is a passive recipient of anesthesia administered to the mother, suffers no blood loss and undergoes passive changes rather than direct stress or hemodynamic alterations caused by surgery.[2] There is difference between obstetric anesthesia and anesthesia for nonobstetric surgeries during pregnancy. The goal in anesthetic management during labor is avoiding uterine relaxation that not only delays the delivery but also predisposes for postpartum hemorrhage. In contrast, prevention of spontaneous abortion or labor leading to premature delivery is the key element when administering anesthesia for non-obstetric cases to these patients. Also, obstetric anesthesia should avoid neonatal depression whereas during anesthesia for nonobstetric surgeries, depression of fetal nervous system is well-tolerated as the transplacentaly transferred drugs can be excreted back to mother for disposal.[3]

ALTERED MATERNAL PHYSIOLOGY AND ITS ANESTHETIC IMPLICATIONS

During pregnancy, profound changes in maternal physiology result from increased concentrations of various hormones, mechanical effects of the gravid uterus, increased metabolic demand and the hemodynamic consequences of the low-pressure placental circulation. Hormonal alterations are responsible for most of the changes that occur during the first trimester. Mechanical effects become apparent when the uterus becomes extra-pelvic, which occurs during the second half of gestation.

Respiratory System and Acid Balance-Base

Physiological changes in respiratory system have been summarized in Table 13.1. The level of the diaphragm rises as the uterus increases in size, and is accompanied by an increase in the antero-posterior and transverse diameters of the thoracic cage. The parturient's breathing pattern changes; it becomes more diaphragmatic as pregnancy progresses because of the effects of the gravid uterus.

Table 13.1: Physiological changes in the respiratory system

Parameter	*Change*	*Magnitude*
Functional residual capacity	Decrease	20%
Closing capacity	Unchanged	–
Minute ventilation	Increase	45%
Tidal volume	Increase	40%
Respiratory rate	Increase	15%
Oxygen consumption	Increase	20–50%

Anesthetic Implications

From the fifth month, the expiratory reserve volume, residual volume, and functional residual capacity (FRC) decrease.[4] Closing capacity (CC), however, remains unchanged. The resulting decrease in the FRC/CC ratio causes faster small-airway closure when lung volume is reduced; thus, parturient can desaturate at a much faster rate as compared with nonpregnant women. The rapid development of hypoxia as a result of decreased FRC, increased oxygen consumption, and airway closure may be minimized by administration of 100 percent oxygen for 3 to 5 minutes before the induction of anesthesia. In an emergency setting, four maximal capacity breaths with 100 percent oxygen should be sufficient.[5]

Minute ventilation increases primarily as a result of an increase in tidal volume. Alveolar ventilation increases 25 percent by the forth month of gestation and 45 to 70

percent by term. This results in a chronic respiratory alkalosis, with a $PaCO_2$ of 28 to 32 mm Hg, a slightly alkaline pH (e.g. approximately 7.44), and decreased levels of bicarbonates and buffer base. Although oxygen consumption increases during gestation, PaO_2 usually increases slightly or remains within the normal range.[1]

The decrease in FRC coupled with the increase in minute ventilation accelerates the uptake of all inhalational anesthetic agents.

Other changes in the respiratory tract and oropharynx during pregnancy may have profound anesthetic implications. Capillary engorgement of the mucosa and edema of the oropharynx, larynx, and trachea may result in a difficult intubation. Any manipulation of the upper airway such as suctioning, insertion of airways, or laryngoscopy may cause edema, bleeding, and upper airway trauma. Hence laryngoscopy should be gentle and be always be done by an experienced anesthesiologist. Also smaller endotracheal tubes should be used.

Because of the particularly friable mucosa of the nasopharynx, instrumentation of the nose should be avoided if possible.[5]

CARDIOVASCULAR SYSTEM

Table 13.2 highlights the physiological changes that occur in the cardiovascular system during pregnancy. Cardiac output increases from the fifth week of pregnancy and reaches its maximum levels at approximately 32 weeks, after which there is only a slight increase until labor, delivery, and the postpartum period.[6] Although this increase in cardiac output is due to an increase in both stroke volume and heart rate, the more important factor is stroke volume. Although the normal variability in heart rate does not change in pregnancy, there does appear to be a reduction in the sympathetic component. Tachyarrhythmias are more common, especially later in pregnancy as a result of both hormonal and autonomic factors.[5]

Anesthetic Implications

The increase in blood volume and cardiac output is associated with clinically significant implications in parturient who have concomitant cardiac disease, but they may also have an impact on healthy parturient. Many pregnant patients complain of symptoms like shortness of breath, palpitations, dizziness, edema, and poor exercise tolerance. Physical examination of the term pregnant women may also be abnormal when compared with the prepregnant state. As illustrated in Table 13.3 pregnancy has numerous effects on cardiac evaluation, including changes in the electrocardiogram, chest radiograph, and echocardiogram. Although these minor changes occur in healthy pregnant women at term, symptoms and signs such as chest pain, syncope, severe arrhythmias, systolic murmur more than grade 3, or diastolic murmur suggest severe disease and warrant further investigation.[7]

Table 13.2: The physiologic changes in the cardiovascular system

Parameter	*Change*	*Magnitude*
Blood volume	Increase	30–45%
Cardiac output	Increase	40%
Stroke volume	Increase	20–50%
Heart rate	Increase	20%
Peripheral resistance	Decrease	15%

Table 13.3: Effect of pregnancy on cardiovascular evaluation

Evaluation	*Findings*
Physical examination	Wide, loud and split S1 Presence of S3 Soft ejection systolic murmur
Chest radiography	Apparent cardiomegaly
Electrocardiography	Right-axis deviation Right bundle branch block T-wave inversion in leads III, V_2, and V_3
Echocardiography	Regurgitation of pulmonary, tricuspid and mitral valve. Increased left ventricle end-diastolic dimensions. Pericardial effusion (40% postpartum)

Supine Hypotension Syndrome

After 28 weeks of pregnancy supine position in a parturient is often associated with decrease in cardiac output. This decrease is secondary to decrease venous return to the heart as the enlarging uterus compresses the inferior vena cava. Some women at term develop the supine hypotension syndrome, which is characterized by hypotension associated with pallor, sweating, or nausea and vomiting. The cause of this syndrome is complete or near-complete occlusion of the inferior vena cava by the gravid uterus. Giving lateral position at this time restores venous return from the lower body and corrects hypotension. The Trendelenburg position may exacerbate caval compression. The gravid uterus also compresses the aorta in most parturients when they are supine. This latter effect decreases blood flow to the lower extremities and, more importantly, the uteroplacental circulation.

Vena caval compression also results in distension of the epidural venous plexus, which increases the chances of intravascular injection of local anesthetic during the

administration of epidural anesthesia. The capacity of the epidural space decreases leading to enhanced spread of small doses of epidural local anesthetic.[1]Anesthetics drugs that cause vasodilatation or anesthetic techniques (e.g. central neuraxial techniques) may exacerbate the impact of aortocaval compression. In the operating room, a small pillow or "wedge" should be used to provide left uterine displacement of 15 to 20 degree.

Changes in Blood Volume and Blood Constituents

Blood volume expands in the first trimester and increases 30 to 45 percent by term. A smaller increase in red blood cell volume (20%) than in plasma volume results in a dilutional anemia. Although moderate blood loss is well tolerated during pregnancy, pre-existing anemia decreases the patient's reserve when significant hemorrhage occurs. Pregnancy is associated with a benign leukocytosis, which makes the white blood cell count an unreliable indicator of infection. In general, pregnancy induces a hypercoagulable state, with increases in fibrinogen; factors VII, VIII, X, and XII; and fibrin degradation products. Pregnancy is associated with enhanced platelet turnover, clotting, and fibrinolysis and there is a wide range in the normal platelet count; thus pregnancy represents a state of accelerated but compensated intravascular coagulation.[8] There is decrease in serum albumin levels. Normal albumin to globulin ratio changes from 1.7:1 to 1:1.[9]

Pregnant patient is predisposed to risk of venous stasis and phlebitis, mainly because of venacaval obstruction by the gravid uterus. There is a risk of thromboembolism, specially in the postoperative period.

Gastrointestinal System Changes

Gastroesophageal reflux and esophagitis are common during pregnancy. Increased progesterone levels decreases the tone of the gastroesophageal sphincter. Mechanical effects of gravid uterus result in upward and anterior displacement of the stomach. At the same time placental gastrin secretion causes hypersecretion of gastric acid. There is some evidence suggesting delayed gastric emptying during pregnancy. All these factors have been associated with an increased risk of severe aspiration pneumonitis. Therefore ,irrespective of the time of oral intake, for all practical purposes pregnant patient is considered full stomach and aspiration prophylaxis should always be considered while administering anesthesia.

Altered Responses to Anesthesia

In addition to the decrease in MAC for inhaled anesthetic agents,[10] thiopental requirements begin to decrease early in pregnancy.[11] In addition, more extensive neural blockade is obtained with epidural and spinal anesthesia in pregnant patients. Pregnancy also enhances the response to peripheral neural blockade.

Plasma cholinesterase levels decrease by approximately 25 percent from early in pregnancy until the seventh postpartum day. Fortunately, prolonged neuromuscular blockade with succinylcholine is uncommon, because the larger volume available for drug distribution offsets the impact of decreased drug hydrolysis.[12] Nevertheless, the dose of succinylcholine and that of nondepolarizing muscle relaxants should be controlled carefully in the pregnant patient. It is desirable to have nerve stimulator to ensure adequate reversal before extubation.

Decreased protein binding associated with low albumin concentrations during pregnancy may result in a greater fraction of unbound drug, with the potential for greater drug toxicity during pregnancy. Cautious administration of all drugs is advisable, because their pharmacokinetic and pharmacodynamic profiles may differ from those in nonpregnant patients.

FETAL EFFECTS FROM ANESTHESIA AND SURGERY

Teratogenicity

Organs have different periods of sensitivity and vulnerability. Drug exposure during the period of organogenesis (approximately days 15–70 after the first day of the last menstrual period) is best avoided if possible. In addition, other factors during anesthesia and surgery (hypoxia, hypercapnia, stress, temperature, ionizing radiation: 5–10 rads) may be teratogenic themselves or enhance the teratogenicity of other agents. Important factors that determine the teratogenicity of a particular agent are the class of drug, dose, route of administration and timing of exposure.

Anesthetic agents and their adjuvants also may produce teratogenicity but the anesthetic exposure is mostly single and short-term.[13] Most medications used for sedation, analgesia or anesthesia have properties in favor of placental diffusion such as low molecular weight, high lipid solubility, low degree of ionization and low protein binding. When a drug crosses the placenta, its teratogenicity depends on the stage of fetal development. When organogenesis is completed, teratogen exposure will affect fetal growth, organ size or organ function.[14]

Concern about the potential harmful effects of anesthetic agents stems from their known effects on mammalian cells. These occur at clinical concentrations and include reversible decreases in cell motility,

prolongation of DNA synthesis, and inhibition of cell division.[15,16] Although there are no anesthetic agents proven to be teratogens,[2,13,17,18] ideally, pregnant patients should be exposed to the fewest drugs possible and at the lowest concentration clinically indicated.

Induction Agents

Teratogenesis has not been associated with the use of any of the commonly used induction agents—including the barbiturates, ketamine and the benzodiazepines—when administered in clinical doses during anesthesia.[18]

Similarly, no evidence supports the teratogenicity of opioids in humans. Benzodiazepine therapy became controversial after several studies reported an association between maternal diazepam ingestion during the first trimester and infants with cleft palate. Although the present consensus among teratologists is that diazepam is not a proven human teratogen, it is better to consider the risk/benefit ratio before initiating chronic benzodiazepine therapy during the first trimester. No evidence suggests that a single dose of a benzodiazepine (e.g. midazolam) during the course of anesthesia would prove harmful to the fetus.[1]

Analgesics

Nonsteroidal anti-inflammatory drugs (NSAIDs) can cause premature closure of the ductus arteriosus and are therefore contraindicated in late pregnancy. Relatively little is known however about possible teratogenic effects. Paracetamol can be used without danger for the developing fetus if the daily dose of 60 mg/kg is not exceeded.

Neuromuscular Blocking Agents

The commonly used depolarizing and nondepolarizing muscle relaxants do not reach fetal circulation in clinically significant amounts. They are water soluble, positively charged and have high molecular weights, which keeps them from crossing the placenta. As expected, no teratogenic effect has been reported after administration of neuromuscular blocking agents to pregnant women.

Local Anesthetics

Spinal anesthesia offers the least placental drug transfer for the degree of anesthesia achieved. Epidural anesthesia or plexus block bring along higher blood levels of local anesthetic and thus more fetal exposure. For clinical concentrations of local anesthetics, there has been no indication of teratogenicity in humans.[19] When using spinal anesthesia however, attention to maternal fluid volume and blood pressure is critical. Maternal hypotension has to be avoided or treated promptly.

Inhalational Agents

Clinical concentrations of volatile anesthetics have wide-ranging cellular effects, but so far, no clinical data link these cellular actions with teratogenic outcomes. Although some animal studies suggest that use of halothane and isoflurane during pregnancy is associated with congenital anomalies, there is not enough human data to support this. Sevoflurane and desflurane are considered safe.[1,19]

Nitrous Oxide

Proposed etiology for teratogenicity of nitrous oxide[5] is the inhibition of methionine synthase, which alters DNA. Although nitrous oxide is considered a weak teratogen in rats and mice, reproductive effects occur only after prolonged exposure to high concentrations that are unlikely to be encountered in clinical anesthesia.

Scientific evidence does not support avoiding nitrous oxide during pregnancy, particularly after the sixth week of gestation. Omission of nitrous oxide may increase fetal risk if inadequate anesthesia results or if a high-dose of a volatile agent results in maternal hypotension. A cautious approach would restrict nitrous oxide administration to a concentration of 50 percent or less and would limit its use in extremely long operations.

Other Potential Fetal Risks

Some studies show an increased risk of spontaneous abortions and low birth weight (LBW) infants in patients who underwent surgery during pregnancy. LBW results from both preterm delivery and intrauterine growth restriction. Although anesthesia and surgery are associated with an increased incidence of spontaneous abortion, IUGR, and perinatal mortality, these can be attributed to the procedure, surgical site, and/or the underlying maternal condition not necessarily exposure to anesthesia.[20]

Fetal Effects of Systemic Drugs

- Opioids and induction agents decrease FHR variability, to a greater extent than do the inhalation agents.[21-23] This most likely indicates the presence of an anesthetized fetus and is not a cause for concern in the absence of maternal hypotension or other abnormalities. Fetal respiratory depression is relevant only if cesarean delivery is to be performed at the same time as the surgical procedure. Even then, high-dose opioid anesthesia need not be avoided when it is

indicated for maternal reasons (e.g. cardiac disease). The pediatrician should be informed of maternal drug administration so that preparations can be made to ventilate the neonate mechanically if required

- Maternal administration of muscle relaxants and reversal agents has not proved to be problematic for the fetus[23]
- Atropine rapidly crosses the placenta and, when given in large doses, causes fetal tachycardia and loss of FHR variability. Both atropine and glycopyrrolate do not significantly affect FHR when standard clinical doses are administered, [24] still glycopyrrolate is often recommended, because it crosses the placenta less readily and may be a more effective antisialagogue. Although limited transplacental passage of neostigmine is expected, significant transfer occasionally may occur
- Sodium nitroprusside and esmolol have been used during pregnancy to induce hypotension during surgical procedures. Standard doses of nitroprusside have proved to be safe for the fetus, [1] and the risk of fetal cyanide toxicity appears to be low, provided tachyphylaxis does not occur and the total dose is limited
- Experiments on pregnant ewes have revealed that the volatile halogenated anesthetic agents can affect the fetus directly (by depressing the fetal cardiovascular system or CNS) or indirectly (by causing maternal hypoxia or hypotension). There is minimal fetal effect with maternal administration of moderate concentrations (1.0/I.5 MAC) of volatile agents. Higher concentrations (e.g. 2.0 MAC) given for prolonged periods induced marked maternal hypotension. Consequently, decreased uteroplacental blood flow resulted in fetal hypoxia, decreased fetal cardiac output, and fetal acidosis.[25]

UTEROPLACENTAL PERFUSION AND FETAL OXYGENATION

Fetal oxygenation depends on maternal oxygen delivery (cardiac output, arterial oxygen tension and hemoglobin and uteroplacental perfusion.

Maternal Oxygenation

Usually the fetus tolerates transient mild-to-moderate decreases in maternal PaO_2, because fetal hemoglobin is present in high concentration and has a high affinity for oxygen. Severe maternal hypoxemia results in fetal hypoxia and if persistent, may cause fetal death. Any complication that causes profound maternal hypoxemia (e.g. difficult intubation, esophageal intubation, pulmonary aspiration, high level of regional block, systemic local anesthetic toxicity) is a potential threat to the fetus.[1]

Maternal administration of increased inspired oxygen will increase fetal oxygenation; however, the fetus is never at risk for hyperoxia, because fetal oxygen tension rarely exceeds 65 mm Hg, even with maternal administration of 100 percent oxygen.[2] Thus, intrauterine retrolental fibroplasia and premature closure of the ductus arteriosus usually do not result from high levels of maternal PaO_2.

Maternal Carbon Dioxide and Acid-Base Status

Maternal **hypercapnia** can cause fetal acidosis, because fetal $PaCO_2$ correlates directly with maternal $PaCO_2$. Although mild fetal respiratory acidosis is of little consequence, severe acidosis can cause fetal myocardial depression and hypotension. Maternal **hyperventilation** with low maternal $PaCO_2$ and high pH can adversely affect fetal oxygenation by means of several mechanisms.[26-28] **Respiratory** or **metabolic alkalosis** can compromise maternal-fetal oxygen transfer by causing umbilical artery constriction[26] and by shifting the maternal oxyhemoglobin dissociation curve to the left.[27]In addition, positive-pressure hyperventilation, independent of changes in $PaCO_2$, may reduce uterine blood flow and cause fetal acidosis.[28] This most likely is a consequence of mechanical ventilation, whereby increased intrathoracic pressure decreases venous return and cardiac output, which in turn decreases uteroplacental perfusion. Thus hyperventilation should be avoided in the pregnant surgical patient. Rather, the $PaCO_2$ should be kept in the normal range for pregnancy.

Uteroplacental Perfusion

Maternal hypotension from any cause can jeopardize uteroplacental perfusion and cause fetal asphyxia .The most common causes of hypotension in the pregnant patient during surgery include the following: (1) deep levels of general anesthesia; (2) sympathectomy with high levels of spinal or epidural blockade; (3) aortocaval compression; (4) hemorrhage; and (5) hypovolemia. The asphyxiated fetus cannot increase oxygen extraction; rather, compensation is by redistribution of blood flow to vital organs. The uterine circulation is not auto regulated; it represents approximately 10 percent of cardiac output by full-term gestation and remains sensitive to vasopressors. Vasoactive medication that reduces uterine blood flow, such as adrenergic agents, dopamine, or epinephrine, are not ideal agents for treating maternal hypotension; al-

though blood pressure may increase, uterine blood flow may remain depressed. Of particular relevance to the pregnant surgical patient are drugs that cause uterine vasoconstriction, such as sympathomimetic agents with predominantly alpha-adrenergic effects[29] or toxic doses of local anesthetics.[30] Preoperative anxiety and light anesthesia increase circulating catecholamines, which may impair uterine blood flow.[31] Drugs that cause uterine hypertonus (e.g. ketamine in early pregnancy in doses greater than 2 mg/kg,[32] alpha-adrenergic agonists,[29] toxic doses of local anesthetics[30]) may increase uterine vascular resistance, which decreases uteroplacental perfusion.

PREVENTION OF PRETERM LABOR

Most epidemiologic studies of nonobstetric surgery during pregnancy have reported an increased incidence of abortion and preterm delivery.[33] It is unclear whether the surgery, manipulation of the uterus, or the underlying condition is responsible. Second trimester procedures and those that do not involve uterine manipulation carry the lowest risk for preterm labor.[20]

There is no evidence suggesting that any anesthetic agent or technique influences the risk of preterm labor. The prophylactic use of tocolytic agents is controversial; there are risks involved, and it is unclear whether they affect outcome. Selective administration to those patients at greatest risk (e.g. those undergoing cervical cerclage) has been suggested.

Indomethacin is used to prevent premature onset of labor; however, the potential risks (e.g. premature closure of ductus arteriosus and development of oligohydramnios) should be considered. Nitroglycerin can be used for uterine relaxation during short procedures or to manage refractory uterine activity. Beta-2 agonists (e.g. terbutaline) and magnesium are effective tocolytics, although their routine prophylactic use is controversial because of their potential risks and limited benefits on preterm labor prevention.[20]

PRACTICAL CONSIDERATIONS

Timing of Surgery

For the fetus, the second trimester is the optimal time to perform surgery. The theoretical risk of teratogenicity is increased during the period of organogenesis in the first trimester and the risk of preterm labor is higher during the third trimester. Maternal risk is greatest during the third trimester because of physiological changes of pregnancy. However, in an emergency setting ,the primary goal is to preserve the mother's life and remote fetal risks associated with anesthesia and surgery are of secondary importance in the event of a serious maternal illness. When planning surgery, it is important to ensure that her obstetrician is informed and that contingency plans have been discussed in the event of complications. The decision to perform simultaneous cesarean delivery depends on a number of factors, e.g. gestational age and maternal condition. Cesarean delivery may be performed immediately before the surgical procedure to avoid fetal risks associated with prolonged anesthesia and intraoperative cardiopulmonary changes and blood loss.

Diagnosis and Surgical Approach

Accurate diagnosis, especially of an acute abdominal crisis, can prove extremely difficult during pregnancy. Nausea, vomiting, constipation, and distension are common symptoms of both normal pregnancy and abdominal pathology. Abdominal tenderness may be indistinguishable from ligamentous or uterine contraction pain. Because the white blood cell count in normal pregnancy can reach 15,000/mm^3, it must be markedly elevated to be helpful diagnostically. During work-up of the acute abdomen, nonurgent radiologic testing should be avoided. Rare consequences of prenatal radiation exposure include a slight increase in the incidence of childhood leukemia and possibly a very small change in the frequency of genetic mutations.

ANESTHETIC MANAGEMENT FOR NONOBSTETRIC SURGERY

Preoperative Assessment

Patient coming for nonobstetric surgery should be assessed for:

- Urgency of the surgery (whether it can be deferred till second trimester of pregnancy)
- Whether the fetus has reached the age of viability
- Effect of the medical condition on the pregnancy
- If there is any underlying medical disease that makes nonobstetric surgery urgent, e.g. patient with RHD with MS. Patients with tight mitral stenosis may become critical as the pregnancy advances and urgent commisurotomy may have to be performed to prevent decompensation
- History of drug consumption and exposure to infection in the first trimester
- In addition to standard preoperative assessment, the pregnant woman requiring surgery needs counseling regarding the anesthetic risks and safety to the fetus and pregnancy. An obstetrician should be consulted if not already involved and matters regarding fetal

monitoring, the risk of preterm labor, use of tocolysis, should be discussed.

Anesthetic Approach before 24 Weeks of Gestation[34]

- Request preoperative assessment by obstetrician.
- Counsel the patient preoperatively
- Use a nonparticulate antacid preoperatively
- Monitor and maintain oxygenation, CO_2, normotension, and euglycemia
- Use regional analgesia for postoperative pain relief when appropriate
- Document fetal heart sounds before and after procedure.

Anesthetic Approach after 24 Weeks Gestation[34]

- Counsel the patient preoperatively
- Obtain obstetric consultation and discuss use of perioperative tocolysis
- Use aspiration prophylaxis
- Maintain uterine displacement perioperatively
- Monitor and maintain oxygenation, PCO_2, normotension, and euglycemia
- Consider use of fetal heart monitoring intraoperatively
- No outcome difference shown between anesthetic agents
- Monitor uterine contractions and fetal heart tones postoperatively.

PREMEDICATION

Aspiration Prophylaxis

As already explained pregnant patient whether presenting for obstetric or nonobstetric surgery, is always at risk for regurgitation and pulmonary aspiration of stomach contents. That is why we should take all efforts to prevent this. Adequate starvation also does not always guarantee protection against aspiration. Prophylactic administration of nonparticulate antacid (15–30 ml of 0.3 M sodium citrate orally 15-30 min prior to induction) can be considered. Although it increases gastric volume, it also increases gastric pH greater than 2.5 and may decrease the likelihood of severe aspiration pneumonitis. H2-receptor antagonist (e.g, ranitidine 150 mg IV at least 2 hrs prior induction) reduces gastric acid output thereby raising pH. Prokinetic drugs like metoclopramide accelerate gastric emptying, decreases gastric volume, and increases lower esophageal sphincter tone. An adult dose of 10 to 20 mg of metoclopramide (0.25 mg/kg) is effective orally, intramuscularly, or intravenously (injected over 5 min). The onset of action is much more rapid following parenteral (3–5 min) than oral (30–60 min) administration.

Anxiolysis

Anxiolytic drugs (e.g. midazolam 1 mg) may be necessary for the anxious parturient, as elevated catecholamines may decrease uterine blood flow (if delivery is not imminent). If delivery is imminent, then sedatives are withheld for the fear of causing neonatal depression in the baby and low APGAR scores.

Prevention of Aortocaval Compression

After 20 weeks gestation, the pregnant patient should be transported in the lateral position, and left uterine displacement instituted when positioned on the operating table. This is achieved by introducing a wedge below the right buttock. The effectiveness of left uterine displacement can be assessed by measuring the blood pressure on the right leg or observing the pulse oximeter waveform on the right foot. For emergency surgery in the prone position, the abdomen should hang unobstructed and any external compression should be avoided.

Preparation of Anesthesia Trolley

Due to the physiological changes in pregnancy (large breast, edema of the oropharyngeal tissues) the chances of difficult ventilation and intubation increase while giving anesthesia to these patients. Hence an experienced anesthesiologist should always be present and difficult intubation cart should be kept ready during obstetric as well as nonobstetric surgeries in these patients.

The following equipment should be ready on the trolley to take care of the likelihood of regurgitation and pulmonary aspiration and difficult intubation.

- Strong suction with wide bore suction catheter attached
- Table with a facility for giving head low position
- Wedge or folded gown to maintain left uterine displacement
- IV cannulae, infusions
- Equipment for difficult intubation
  - Laryngoscopes with different sized blades
  - Short handle as these patients are likely to have large breasts
  - Stylets and intubating bougie
  - Intubating LMA and pro- seal LMA
  - Small sized endotracheal tubes besides the normal one
  - At least one device suitable for emergency non-surgical airway ventilation, e.g. a transtracheal jet ventilator

– Equipment suitable for emergency surgical airway access (e.g. cricothyrotomy).

Monitoring

Monitoring must begin prior to induction of anesthesia. Maternal monitoring should include cuff blood pressure, preferably noninvasive BP measurement, cardioscope, pulse oximetry, capnography, urine output and temperature monitoring. The use of a nerve stimulator is desirable. The FHR and uterine activity should be monitored both during and after surgery when technically feasible.

Fetal Monitoring

Surgery and anesthesia can affect uterine activity and placental perfusion, and therefore fetal oxygenation and fetal heart rate. Fetal heart rate can also be affected directly by medications that readily cross the placenta or indirectly by their influence on maternal hemodynamics. Some researchers only measure fetal heart rate before and after surgery, others apply continuous monitoring.

The American College of Obstetricians and Gynecologists recently opined that "the decision to use intraoperative fetal monitoring should be individualized and each case warrants a team approach for optimal safety of the woman and her baby".[35]

Fetal well-being, as indicated by FHR variability, is present by 25 to 27 weeks gestation. Loss of beat-to-beat variability and decreased baseline FHR are common after administration of anesthetic agents but declerations suggest fetal hypoxemia. An unexplained change in FHR mandates the evaluation of maternal position, blood pressure, oxygenation, acid-base status, and inspection of the surgical site to ensure that neither surgeons nor retractors are impairing uterine perfusion. Maternal hypothermia during surgery may result in slowing of the fetal heart rate. Monitoring maternal temperature perioperatively and the use of warming devices to maintain normothermia are important. A multidisciplinary plan is necessary in the event of persistent fetal distress, e.g. performing an emergency cesarean delivery.

Choice of Anesthesia

The choice of anesthesia should be guided by maternal indications and should take into consideration, the site and the nature of the surgery. No study has correlated improved fetal outcome with any anesthetic technique. When possible, local or regional anesthesia (with the exception of paracervical block) is preferred; this permits the administration of drugs with no laboratory or clinical evidence of teratogenesis. In addition, maternal respiratory complications occur less frequently with local and regional anesthetic techniques. These techniques are suitable for cases involving cervical cerclage, urologic or lower extremity procedures, and operations on the arm or hand. Most emergency abdominal operations require general anesthesia, because the incision typically extends to the upper abdomen and surgery involves bowel handling which increases the chance of aspiration in a patient with unprotected airway.

Regional Anesthesia

Maternal hypotension associated with spinal or epidural anesthesia should be prevented or minimized by fluid preloading, preventing supine hypotension and avoiding high block. Pregnant patients may have reduced requirements for local anesthetics and appropriate dose reduction is necessary to prevent a high block. Patients are at higher risk for systemic local anesthetic toxicity because decreased protein binding during pregnancy results in a greater fraction of unbound drug.

Appropriate vasopressors should be available to treat hypotension if it occurs. Ephedrine has been shown to be the preferred vasopressor for the treatment of hypotension after neuraxial anesthesia. There is a risk though of uterine artery vasoconstriction and reduced uteroplacental circulation. Patients receiving magnesium are more prone to hypotension, which is often more resistant to treatment with vasopressors.

General Anesthesia

Induction

General anesthesia mandates endotracheal intubation beginning at approximately 16–20 weeks gestation. Preoxygenation with 100 percent oxygen administration for 3–4 min (or 4 vital capacity breaths if time is restricted) before a rapid sequence induction with cricoid pressure should be performed. This takes care of the tendency to desaturate early. Succinylcholine is the drug of choice for facilitating tracheal intubation because of its early onset and quick offset. In case of contraindication to the use of Succinylcholine, Inj. Rocuronium can be used. The use of a smaller endotracheal tube (6.0–7.0 mm) is recommended because of respiratory tract mucosal edema and engorgement associated with pregnancy. Avoid nasal intubations that may precipitate bleeding because of increased mucosal vascularity. Use drugs with a history of safe use in pregnancy including thiopentone,

succinylcholine, and most nondepolarising muscle relaxants. Although not similarly "time-tested," many consider propofol safe during pregnancy.[20]

Airway Management in Pregnant Patient

As already explained, because of the physiological changes in pregnancy these patients are often difficult to ventilate and intubate. A proper assessment of airway can predict the difficult airway, but many times anesthesiologist may encounter an unanticipated difficult airway and may fail to intubate a pregnant patient. Flow chart 13.1 suggests the steps to be followed in case of failed intubation in pregnant patients.

Maintenance

A moderate concentration of a volatile agent (1.5–2.0 MAC) with a high concentration of oxygen (FiO_2 0.5) is recommended. Restrict nitrous oxide administration to a concentration of 50 percent or less and limit its use in extremely long operations. Hyperventilation should be avoided for the fear of causing uterine artery vasoconstriction. Rather, end-tidal CO_2 should be maintained in the normal range for pregnancy. Long procedures should include a blood glucose assessment. Slow reversal of muscle relaxants with anticholinesterase drugs may decrease uterine irritability postoperatively.[34] Extubation should be done only after the patient is fully awake and after return of the protective airway reflexes.

Maintenance of normal maternal blood pressure is of great importance because of the relative passive dependence of the uteroplacental circulation. Except under the most unusual circumstances (i.e renal failure), intravenous fluid administration should be appropriate to the surgical blood loss requirements. Patients on magnesium may have prolonged muscle paralysis after the administration of muscle relaxants; therefore, reducing the dose of muscle relaxant is recommended.

Postoperative Care

The FHR and uterine activity should be monitored during recovery from anesthesia. If the fetus is viable and premature labour occurs, early pediatric consultation is advised and if necessary, the patient should be transferred to a hospital with a neonatal intensive care unit. Adequate analgesia should be obtained with systemic or spinal opioids. Regional anesthesia may be preferable because systemic opioids may reduce FHR variability. The routine and prolonged use of non steroidal anti-inflammatory drugs is best avoided because of potential fetal effects (e.g. premature closure of ductus arteriosus and development of oligohydramnios). Acetaminophen is safe to prescribe in this setting. Early mobilization should be considered as patients are at risk for thromboembolism.

SPECIAL SITUATIONS

Cervical Cerclage

This is a surgical procedure required occasionally for women with a history or active clinical features of an incompetent cervix, usually presenting as premature, precipitate labour. To reduce the risk of this occurring, a suture (Shirodkar suture) is placed around the cervix, in a procedure lasting 20 to 30 min. As this is usually performed in the second trimester or later, the anesthetic considerations for any pregnant woman apply. Regional (spinal) anesthesia is the technique of choice, with the required block height of T10-S5 being achieved using 7.5 mg hyperbaric bupivacaine with or without fentanyl 25 ug. If general anesthesia must be undertaken, use of rapid sequence induction is mandatory if the patient is at more than 12 weeks, gestation.[36]

Trauma

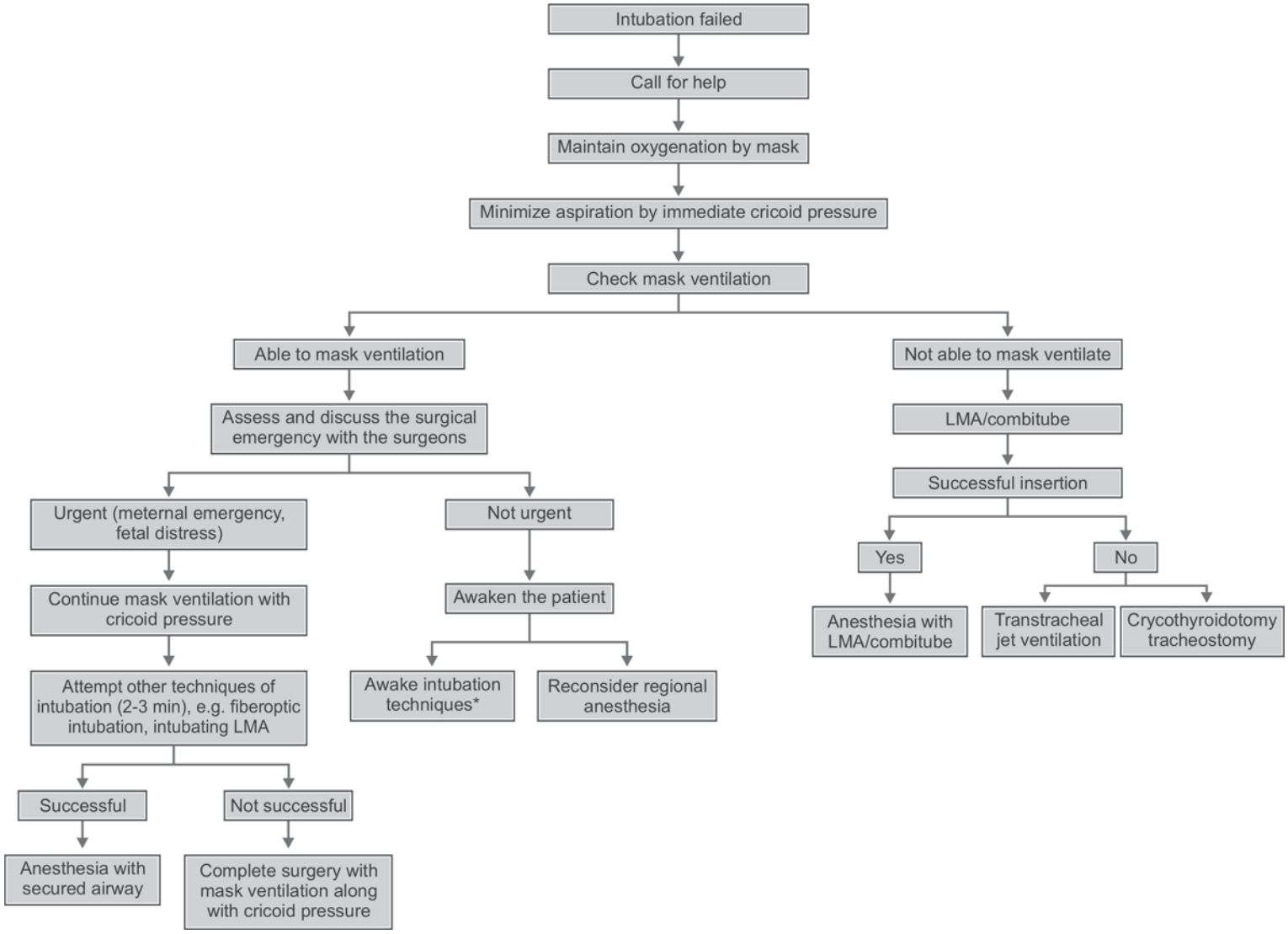
Injury related to trauma occurs in up to 6 to 7 percent of all pregnancies and is perhaps the most common cause of nonobstetric maternal mortality. In contrast with nonpregnant women, abdominal injury during pregnancy is more likely than head injury.[5]

Rapid assessment, hemodynamic stabilization and treatment of maternal injuries are essential for fetal survival. It is important to remember that the anatomic and physiologic changes associated with pregnancy may cause the clinician to underestimate the true extent of hypovolemia and hence clinical estimation of the following signs of hypovolemia should be undertaken:

- Hypotension
- Tachycardia >120 beat min^{-1}
- Urinary output <0.5 min^{-1} kg^{-1}
- Capillary refill time >5 seconds
- Anxiety, agitation or confusion
- Transient or minimal response to 1-2 L crystalloid or 500 ml colloid fluid challenge.[36]

In hemorrhagic shock, maternal blood is shunted away from the uterus to preserve perfusion to vital maternal organs at the expense of the fetus; such a physiologic response causes fetal hypoxemia and even death. A preoperative assessment that includes airway evaluation should be performed. The choice of regional or general anesthesia techniques should be based on the clinical status, surgical procedure, experience of the anesthesiologist and the psychological condition of the patient.

Flow chart 13.1: Failed intubation drill in pregnant patient[1]



*Awake fiberoptic intubation, awake intubation with topical anesthesia of the airway

Table 13.4: Indications for emergency cesarean delivery in a pregnant trauma patient[34]

1. Traumatic uterine rupture
2. Stable mother with viable fetus that is in distress
3. A unsalvageable mother who still has a viable fetus
4. A gravid uterus that is interfering with intraoperative surgical repair

Principles of aspiration prophylaxis remain the same Monitoring of the fetus perioperatively is important, but not always feasible, especially during abdominal surgery. External FHR monitoring is usually possible from 18 weeks onward. Alterations in FHR may indicate adverse maternal conditions before they become apparent with standard monitoring. Such alterations should therefore encourage evaluation of maternal oxygenation, hemodynamics, acid-base status and activities at the surgical field for compromise of uterine perfusion. After initial resuscitation and trauma surveys are complete, an early ultrasound in the emergency room is recommended to determine fetal viability. Sometimes emergency cesarean delivery may need to be performed in a pregnant trauma patient. Indications for emergency or urgent cesarean deliveries are outlined in Table 13.4. Resuscitation and optimisation of mother is important before obstetric management.

Neurosurgical Procedures in Pregnancy

In a neurosurgical procedure performed on a pregnant woman some specific precautions must be taken to avoid of fetal asphyxia, to avoid teratogenic drugs and to prevent preterm labour. Fetal oxygenation depends on maternal arterial oxygen content and placental blood flow. Although hypotension and hypocarbia are commonly induced during neurosurgery, those techniques may place fetus at risk for intrauterine asphyxia. This can be avoided by maintaining normal maternal PaO_2, $PaCO_2$ and uterine blood flow. The patients should be monitored intraoperatively and early postoperatively (at least 24–48 hours) for uterine tone. Drugs such as adrenergic vasopressors should be avoided, as should the rapid intravenous administration of anticholinesterase drugs. During neurosurgery, osmotic diuresis, controlled hypotension and hypothermia are commonly induced to decline the intracranial pressure (ICP). In the pregnant patients, those may adversely affect the fetus. Temporary clipping of a vessel may be preferred to reduce intra-aneurysmal pressure instead of hypotension.[37] Hypovolemia and very large doses of mannitol should be avoided as they have the potential to cause fetal dehydration. If endovascular treatments are undertaken, then uterine shielding during periods of radiation is necessary. In addition to the monitors used during neurosurgical interventions monitoring of the fetus and uterus should be used whenever possible. An external Doppler fetal heart rate monitor will be useful. Close observation of maternal blood pressure and prompt treatment of hypotension and hypoxia are essential for the well being of fetus. Extubation should be delayed until the patient is sufficiently awake to protect her airway from regurgitation and aspiration. The patient should be placed in a lateral position with her head slightly elevated. Fetal heart rate and uterine tone should be monitored at least 24 to 48 hours postoperatively.

Cardiac Surgery during Pregnancy

The incidence of heart disease in pregnancy ranges from 1 to 4 percent.[34] During pregnancy, the functional class of the patient can be considered as one class higher. Two periods in pregnancy are associated with decompensation. At 28 to 30 weeks cardiac output reaches maximum and the incidence of decompensation may peak at that time. Similarly, immediately after delivery the increase in venous return and uterine autotransfusion lead to volume overload. These changes are particularly problematic in stenotic valve lesions and pulmonary hypertension. The indications for cardiac surgery during pregnancy are few but include severe valve disease, malfunction of prosthetic valve, major vessel dissection, traumatic aorta rupture, pulmonary embolism, heart tumor, and open foramen ovale. Cardiac surgery in pregnancy is associated with maternal mortality of 3 to 15 percent [34] and a fetal mortality of 20 to 35 percent.[34] For some conditions, percutaneous cardiac intervention offers effective therapy with far less risk to the mother and her fetus. For others, cardiac surgery, including procedures that mandate the use of cardiopulmonary bypass, must be entertained to save the life of the mother. Given the extreme risks to the fetus, if the patient is in the third trimester, strong consideration should be given to delivery before surgery involving cardiopulmonary bypass.[38] Open heart surgery is associated with risks to both mother and fetus. Risk factors are summarized in Table 13.5. Maternal and fetal perioperative mortality rates may be reduced through such measures as early preoperative detection of maternal cardiovascular decompensation, use of fetal monitoring, delivery of a viable fetus before the operation and scheduling surgery on an elective basis during the second trimester. Additionally, fetal morbidity may be reduced during cardiopulmonary bypass by optimizing maternal oxygen-carrying capacity and uterine blood flow (Table 13.6).

Table 13.5: Risks factors for the mother and fetus during cardiac surgery[39]

Risk factors for mother	*Risk factors for fetus*
• Use of vasoactive drugs • Increased age • Type of surgery • Reoperation • Higher maternal functional class	• Maternal age >35 yr • Functional class • Emergency surgery • Type of myocardial protection • Anoxic time

Table 13.6: Current maternal bypass recommendations[40]

- Maintaining the pump flow rate >2.5 L · min^{-1} · m^{-2}
- Perfusion pressure >70 mm Hg.
- Maintaining the hematocrit > 28%
- Using normothermic perfusion when feasible
- Using pulsatile flow
- Using α-stat pH management

Electroconvulsive Shock Therapy

The treatment of major psychiatric disorders during pregnancy is problematic and optimal management remains controversial. Electroconvulsive shock therapy (ECT) avoids potential teratogenicity from psychotropic medications and it is often used to treat major depression and bipolar disorders during pregnancy especially when rapid control of depressive symptoms is needed.[20] Electroconvulsive therapy is a relatively safe and effective treatment during pregnancy, if steps are taken to decrease potential risks.[40]

Preparation for ECT during Pregnancy should include[41]

- A pelvic examination
- Discontinuation of nonessential anticholinergic medication
- Uterine tocodynamometry
- Intravenous hydration
- Use anesthetic agents (e.g. barbiturates, succinylcholine, anticholinergics) with a long history of safe use during pregnancy
- Confirm the absence of uterine contractions using tocodynamometry before and after ECT
- Monitor the FHR immediately before and after ECT
- Consider full stomach prophylaxis and endotracheal intubation after the first trimester
- Provide left uterine displacement in patients >20 weeks of gestation.

Anesthesia for Emergency Cesarean Section

Common indications for cesarean section[42]

- Previous cesarean section
- Malpositions (Breech)
- Fetal distress
- Dystocia (Failure to progress during labor)
- Maternal disease
  - Worsening of pre-existing disease (e.g. cardiac)
  - Associated with pregnancy (e.g. pre-eclampsia)
- Placenta previa or abruption
- Multiple pregnancy
- Cord prolapse
- Maternal choice.

What is an 'emergency' cesarean section? A four-point classification is used to categorize the urgency of the cesarean section[43] (Table 13.7).

Earlier a 30 min time period was defined as a standard for decision-delivery interval in the Category 1 situation. The 30 min time frame seems to be based on custom and practice, rather than on objective evidence in relation to condition of the newborn.[44] Each case should be managed according to the clinical evidence of urgency, with every single case being considered on its merits.

Table 13.7: Urgency of cesarean section

Category 1:	Immediate threat to the life of a woman or fetus. Examples: prolonged fetal bradycardia, umbilical cord prolapse, uterine rupture, fetal scalp pH <7.2, placental abruption.
Category 2:	Maternal or fetal compromise but not immediately life threatening. Examples: failure to progress labor, failed induction of labor
Category 3:	Needing early delivery but no maternal or fetal compromise. Examples: patient booked for planned cesarean section presenting with rupture of membranes
Category 4:	At a time to suit the woman and the cesarean section team. Examples: elective cesarean section for breech presentation, twins or previous cesarean section

In utero Fetal Resuscitation

The role of the anesthetist in maternal resuscitation and management of life-threatening obstetric emergencies has been reviewed recently. The anesthetist should also be an active participant in measures that might ameliorate isolated fetal compromise and even avert the need for cesarean section[45] (Table 13.8)

Table 13.8: *In utero* fetal resuscitation—role of anesthetist

1. Syntocinon off
2. Position full left lateral
3. Oxygen
4. IV infusion of 1 liter crystalloid
5. Low blood pressure: IV vasopressor
6. Tocolysis: Terbutaline 250 lg (SC), glyceryl trinitrate 400 lg (metered aerosol doses)

Anesthesia Options for Emergency Cesarean Section

The decision to use a particular anesthetic technique should be individualized based on anesthetic, obstetric or fetal risk factor, the preferences of the patient and the judgment of the anesthesiologist. The anesthetic technique of first choice for the woman who already has an epidural catheter in place will be top-up of that epidural. Unless contral indicated, single-shot spinal anesthesia is appropriate for the majority of women without labor epidural analgesia who require category 2 cesarean section. For all category 1 patients general anesthesia is recommended.

ANESTHETIC MANAGEMENT

General Principles

Preanesthetic Evaluation (ASA task force recommendations for obstetric anesthesia[46])

- Anesthesiologist should conduct a focused history and physical examination before sproviding anesthesia care, giving due attention to:
  - Maternal health and anesthetic history
  - Relevant obstetric history
  - Airway and heart and lung examination
  - Baseline blood pressure measurement
  - Back examination when neuraxial anesthesia is planned or placed
- A communication system should be in place to encourage early and ongoing contact between obstetric providers, anesthesiologists and other members of the multidisciplinary team
- A routine intrapartum platelet count is not necessary in the healthy parturient. However, depending on patient's history, physical examination and clinical signs platelet count is advised and platelets are ordered if required
- It is our institutional policy to order blood grouping and cross matching in all patients undergoing cesarian section
- The fetal heart rate should be monitored by a qualified individual before and after administration of neuraxial analgesia for labor.

Aspiration Prophylaxis

(Already discussed).

Large Gauge Intravenous Access

Reliable large gauge intravenous access should be established (ideally at least 18 G). A second IV should be considered in all individuals considered at additional risk of hemorrhage. Choice of insertion of central line would depend mainly upon the medical disease complicating the pregnancy, e.g. mitral stenosis etc, unstable hemodynamics because of obstetric complications like ruptured uterus or placenta accreta.

Patient Positioning

- Supine positioning should be avoided in all women presenting for cesarean section
- Left lateral tilt reduces aortocaval compression and hypotension secondary to reduction in preload.

Patient Monitoring

- Continuous pulse oximetry, noninvasive blood pressure monitoring, ECG, urine output, blood loss, temperature are monitored. It is also desirable to monitor end tidal CO_2 to confirm tracheal intubation as well as maintaining it at normal level intraoperatively to prevent uterine arterial vasoconstriction
- Fetal monitoring should ideally continue during preparation for surgery where there are concerns with fetal well-being.

Prophylactic Antibiotics

Prophylaxis may reduce the risk of fever, endometritis, wound infection, urinary tract infection and serious infection postcesarean section.

Use of Antiemetics

- Emesis is often a response to hypotension secondary to regional anesthesia; this should be actively managed and where possible, prevented.

Use of Oxytocic Drugs

- Administration of syntocinon (oxytocin) following delivery reduces the risk of postpartum hemorrhage.
- Syntocinon causes vasodilatation and tachycardia and bolus injection has been associated with catastrophic collapse in vulnerable parturients. Therefore, it should be given slowly following cesarean delivery
- In women at very high-risk, e.g. the parturient with significant cardiac disease, syntocinon may sometimes be best avoided; instead other practices such as uterine massage and hot fomentation should be encouraged.

GENERAL ANESTHESIA

Conditions which pose an immediate threat to the life of mother or fetus, e.g. uterine rupture, severe fetal acidosis, etc. demand an urgent delivery and hence early

induction of anesthesia. For such conditions general anesthesia is the choice. The anatomic changes to the airway increase the risk of failed intubation. The principles of aspiration prophylaxis and airway management remain the same.

Conduct of General Anesthesia[47]

- Metoclopramide 10 mg IV and H_2 blocker.
- Nonparticulate antacid
- Left uterine displacement
- Denitrogenation with 100 percent O_2
- Cricoid pressure
- Rapid sequence intravenous induction with thiopental and succinylcholine
- Intubation (the principles of airway management remain the same)
- Verification of tube placement
- Communication with surgeons to start the surgery
- Administration of 30 to 50 percent nitrous plus 0.5 MAC of volatile agent after delivery
- Increase nitrous oxide to 70 percent
- Turn off (or turn down to very low levels) the volatile agent
- Administer intravenous opioid and benzodiazepine
- Administer muscle relaxant (short acting non-depolarizing or intermittent bolus doses of succinylcholine)
- Empty stomach with orogastric tube
- Reversal of muscle relaxant
- Awake extubation after verification of intact airway reflexes.

Depth of Anesthesia

The effects on the fetus of anesthetics and opioid analgesics are 'innocuous and reversible'. The choice of drug regimen or doses used for women with cardiac or cerebrovascular disease should not be restricted on account of concerns for the fetus. Ensure presence of a neonatologist and the resuscitation equipment prior to induction of anesthesia. Dose-dependent respiratory depression is predictable and readily treatable by a neonatologist. There is no justification for administration of low inspired vapour concentrations that risk awareness. However, using inhalational anesthetics in higher concentrations may produce uterine hypotonia. Use of benzodiazepines and opioids at this point will help to achieve the desired depth of anesthesia and amnesia. There is no evidence that neonatal 'outcome' is adversely influenced by greater depth of maternal anesthesia; the relaxant effect of modern, insoluble vapours on uterine tone is readily reversible. In the event of severe hypovolemia, anesthesia can be induced and maintained with intravenous ketamine, which has a useful sympathomimetic effects.[48]

REGIONAL ANESTHESIA

Epidural Anesthesia

Women receiving epidural analgesia in labour should be reviewed regularly to identify suboptimal blocks (e.g. missed segments) that predict potentially inadequate surgical anesthesia for cesarean section. 0.5 percent bupivacaine is the most commonly used local anesthetic. The efficacy of epidural anesthesia is consistently reported as inferior to that of spinal anesthesia in both elective and emergency situations. Blockade of light touch sensation from S5 to T5 should avoid the need for supplementation or conversion to general anesthesia. The addition of epidural fentanyl 50 mg minimizes pain from visceral traction.

Subarachnoid Block

Active bleeding, cardiac disease, uncorrected coagulopathy and a high suspicion of bacteremia are contraindications to single-shot spinal anesthesia. To establish a satisfactory block, 0.5 percent hyperbaric bupivacaine (1.8-2.2 ml) is appropriate for most Indian women Intravenous fluid preloading reduces the frequency of maternal hypotension after spinal anesthesia for cesarean delivery. Although fluid preloading reduces the frequency of maternal hypotension, initiation of spinal anesthesia should not be delayed to administer a fixed volume of intravenous fluid, in an emergency situation. Obsessive maintenance of left-lateral tilt to offset aortocaval compression and prompt use of vasopressors can minimise the complications.

Postoperative Concerns

Patient should be monitored postoperatively. The aims of postoperative monitoring are to ensure:

- Recovery from regional or general anesthesia
- Detection of PPH and other complications of procedure
- Adequate pain relief. Unless there is hepatic dysfunction, paracetamol can be given Diclofenac can be prescribed provided there are no contraindications (notably renal dysfunction, e.g. in pre-eclampsia).

REFERENCES

1. Naughton NN, Cohen SE. Non obstetric Surgery During Pregnancy. In: Chestnut DH, (Ed). Obstetric Anesthesia: Principles and Practice 3rd edn. Philadelphia: Elsevier Mosby 2004; 255-72.

2. Rosen Mark A. Management of Anesthesia for the Pregnant Surgical Patient. Anesthesiology 1999;91: 1159-63.
3. Steinberg Ellen S, Santos Alan C. Surgical Anesthesia During Pregnancy. International Anesthesiology Clinics Winter 1990;28(1):58-66.
4. Santos AC, Braveman FR, Finster M. Obstetric Anesthesia. In: Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anesthesia. (5th edn). Philadelphia: Lippincott Williams and Wilkins 2006;1153-81.
5. Birnbach DJ, Browne IM. Anesthesia for Obstetrics. In: Miller RD (Ed). Miller's Anesthesia (7th edn). New York: Churchill Livingstone 2007; 2203-40.
6. Mabie WC, DiSessa TG, Crocker LG, et al. A longitudinal study of cardiac output in normal human pregnancy. American Journal of Obstet Gynecol 1994;170:849-56.
7. Bhagwat AR, Engel PJ. Heart disease and pregnancy. Cardiology Clinics 1995;13:163.
8. Gerbasi FR, Bottoms S, Farag A, et al. Increased intravascular coagulation associated with pregnancy. Obstet Gynecol 1990;75:385-9.
9. Physiological changes during pregnancy. In: Hiralal Konar, editor. Dutta DC: Textbook of Obstetrics. Calcutta: New Central Book Agency Pvt Limited 2004;546-56.
10. Gin T, Chan MTV. Decreased minimum alveolar concentration of isoflurane in pregnant humans. Anesthesilogy 1994;81:829-32.
11. Gin T, Mainland P, Chan MTV, et al. Decreased thiopental requirements in early pregnancy. Anesthesiology 1997; 86:73-8.
12. Leighton BL,Cheek TG, Gross JB, et al. Succinylcholine pharmacodynamics in peripartum patients. Anesthesiology 1986;64:202-5.
13. Brodsky JB. Anesthesia and surgery during early pregnancy and fetal outcome. Clinical Obstetrics and Gynecology 1983;26:449-57.
14. Van de Velde M, De Buck F. Anesthesia for nonobstetric surgery in the pregnant patient. Minerva Anestesiologica 2007;73(4):235-40.
15. Sturrock JE, Nunn JF. Mitosis in mammalian cells during exposure to anesthetics. Anesthesiology 1975;43:21-33.
16. Sturrock J, Nunn JF. Effects of halothane on DNA synthesis and the presynthetic phase (G1) in dividing fibroblasts. Anesthesiology 1976;45:413-20.
17. Fanzago E. Anesthesia for nonobstetric surgery in pregnant patients. Minerva Anestesiologica 2003;69: 416-27.
18. Littleford J. Effects on the fetus and newborn of maternal analgesia and anesthesia: a review. Canadian Journal of Anesthesia 2004;51:586-609.
19. Allaert S, Carlier S, Weyne L, et al. First trimester anesthesia exposure and fetal outcome. A review. Acta Anaesth Belg 2007;58:119-23.
20. Brendan Carvalho. Nonobstetric Surgery During Pregnancy IARS 2006. Review Course Lectures
21. Johnson ES, Colley PS. Effects of nitrous oxide and fentanyl anesthesia on fetal heart-rate variability intra-and postoperatively. Anesthesiology 1980;52:429-30.
22. Katz JD, Hook R, Barash PG. Fetal heart rate monitoring in pregnant patients undergoing surgery. Am J Obstet Gynecol 1976;125:267-9.
23. Liu PL, Warren TM, Ostheimer GW, et al. Fetal monitoring in parturients undergoing surgery unrelated to pregnancy. Can Anaesth Soc J 1985;32:525-32.
24. Abboud T, Raya J, Sadri S, et al. Fetal and maternal cardiovascular effects of atropine and glycopyrrolate. Anesth Analg 1983;62:426-30.
25. Palahniuk RJ, Shnider SM. Maternal and fetal cardiovascular and acid-base changes during halothane and isoflurane anesthesia in the pregnant ewe. Anesthesiology 1974;41:462-72.
26. Motoyama EK, Rivard G, Acheson F, et al. The effect of changes in maternal pH and $PaCO_2$ on the PO_2 of fetal lambs. Anesthesiology 1967;28:891-903.
27. Kamban JR, Handte RE, Brown WU, et al. The effect of normal and pre-eclamptic pregnancies on the oxyhemoglobin dissociation curve. Anesthesiology 1986;65:426-7.
28. Levinson G, Shnider SM, de Lorimier AA, et al. Effects of maternal hyperventilation on uterine blood flow and fetal oxygenation and acid-base status. Anesthesiology 1974;40:340-7.
29. Ralston DH, Shnider SM, de Lorimier AA. Effects of equipotent ephedrine, metaraminol, mephentermine and methoxamine on uterine blood flow in the pregnant ewe. Anesthesiology 1974;40:354-70.
30. Greiss FC, Still JG, Anderson SG. Effects of local anesthetic agents on the uterine vasculature and myometrium. Am J Obstet Gynecol 1976;124:889-99.
31. Shnider SM, Wright RG, Levinson G, et al. Uterine blood flow and plasma norepinephrine changes during maternal stress in the pregnant ewe. Anesthesiology 1979;50:524-7.
32. Oats JN, Vasey DP, Waldron BA. Effects of ketamine on the pregnant uterus. Br J Anaesth 1979;51:1163-6.
33. Mazze RI, Källén B. Appendectomy during pregnancy: A Swedish registry study of 778 cases. Obstet Gynecol 1991; 77:835-40.
34. Cheek Theodre G, Emily Baird. Anesthesia for Nonobstetric Surgery: Maternal and Fetal Considerations. Clinical Obstetric and Gynecology 2009;52(4):535-45.
35. American College of Obstetricians and Gynecologists Committee on Obstetric Practice. Nonobstetric Surgery in Pregnancy. ACOG Committee Opinion No. 284. Obstetric and Gynecology 2003;102(2):431.
36. Obstetric anesthesia and analgesia. In: Aitkenhead AR, Rowbotham DJ, Smith G (Eds). Textbook of Anesthesia 4th ed. New York: Churchill Livingstone 2001;629- 49.
37. Cirak Bayram Kiymaz Nejmi, et al. Neurosurgical Procedures in Pregnancy Acta Cirúrgica Brasileira 2003;18 (1):5.
38. Barth William H. Cardiac Surgery in Pregnancy. Clinical Obstetrics and Gynecology 2009;52(4):630-46.
39. Talwar S, Kale SC, Kumar L, et al. Open heart surgery during pregnancy IJTCVS 2003;19:184-5.
40. Chandrasekhar S, Christopher R, et al. Cardiac Surgery in the Parturient. Anesth Analg 2009;108:777-85.

41. Miller LJ. Use of electroconvulsive therapy during pregnancy. Hospital and Community Psychiatry 1994; 45(5):444-50.
42. Brown JPR. Recent Developments in Anesthesia for Cesarean Section in the UK. Update in Anesthesia 2008; 23.
43. Lucas DN, Yentis SM, Kinsella SM, et al. Urgency of cesarean section: a new classification. Journal of the Royal Society of Medicine 2000;93:346-50.
44. Wee MYK, Brown H, Reynolds F. The National Institute of Clinical Excellence (NICE) guidelines for cesarean sections: implications for the anesthetis. International Journal of Obstetric Anesthesia 2005;14:147-58.
45. Thurlow JA, Kinsella M. Intrauterine resuscitation: active management of fetal distress. International Journal of Obstetric Anesthesia 2002;11:105-16.
46. Practice Guidelines for Obstetric Anesthesia An Updated Report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Anesthesiology 2007; 106:843-63.
47. Birnbach David J. General Anesthesia for Cesarean Section—Who needs it? European Society of Anesthesiologists Refresher Course Euroanesthesia 2003.
48. Levy DM. Emergency Cesarean Section Anesthesia 2006;61:786-91.

14 Pregnancy Induced Hypertension and Anesthesia

Shantanu B Kulkarni, Urvi H Desai, Manju N Gandhi

KEY POINTS

- Preeclampsia and eclampsia (Pregnancy induced hypertension) constitute a significant cause of maternal and fetal morbidity and mortality. These two conditions are responsible for multiorgan dysfunction and failure, and require a multidisciplinary approach to treat them
- The primary target organ sites of involvement in preeclampsia include the brain, liver, lungs and the kidneys
- Acute pulmonary edema is one of the most critical clinical presentations of preeclampsia—in such cases intravenous fluid management needs close monitoring
- HELLP syndrome is considered as a variant of severe preeclampsia with dominant but not isolated involvement of the hepatic parenchyma
- Magnesium sulfate ($MgSO_4$) is the drug of choice in the following settings—seizure prophylaxis in severe preeclampsia and the treatment of seizures and recurrent seizures in eclampsia
- Antihypertensive drugs, which may be safely used, include labetalol, nifedipine and hydralazine
- Lumbar epidural analgesia is beneficial during labor as it limits hypertensive responses to pain and provides ready opportunity for block extension for delivery by cesarean section
- Regional blockade is the preferred method of anesthesia for cesarean section. The risk of spinal hematoma following regional anesthesia is very low if the platelet count is $>100,000/mm^3$
- When general anesthesia is required measures should be taken to ensure that the intubation response is minimized.

Pregnancy induced hypertension (PIH) is a complex disorder, resulting from multifactorial causes which produce multiple end organ dysfunction, and is a major cause of obstetric and perinatal mortality.

Classification[1]

Hypertension during pregnancy is distinctively classified into two major categories:

- Pregnancy induced hypertension (PIH)
- Chronic hypertension

PIH is further divided clinically into preeclampsia, eclampsia and HELLP syndromes.

Preeclampsia

Preeclampsia is a disorder that occurs after 20 weeks of pregnancy. A triad of signs and symptoms characterizes it, which include the following:

- Hypertension, BP $\geq$ 140/90 mm Hg
- Proteinuria > 300 mg in 24 hrs
- Generalized edema.

Eclampsia

Eclampsia is defined as the occurrence of convulsions in pregnancy or puerperium in association with preeclampsia.

HELLP Syndrome

HELLP syndrome comprises of hemolysis, elevated liver enzymes and low platelet count. It is usually associated with preeclampsia or eclampsia.

Chronic Hypertension

Chronic hypertension consists of persistent hypertension before the 20th week of pregnancy or after 6 weeks of delivery.

Incidence and Predisposing Factors

Preeclampsia usually abates within 48 hours of termination of pregnancy and placental delivery. The incidence of PIH is more in the following sub-groups of patients:

- Young primigravida
- Multiple gestations
- Polyhydraminos
- Diabetic patient
- Chronic hypertensive
- Family history of PIH.

Etiology

The cause of PIH continues to defy our complete understanding of the disease. It could be secondary to the following causes:

- Immunological
- Genetic
- Placental.

Decreased placental perfusion from compromised implantation, hyperplacentosis and/or impaired placental vascularization causes global endothelial dysfunction. Damaged endothelial cells release substances like fibronectin, endothelins, tumor necrosis factor, etc. which result in systemic inflammatory response (SIR). It also causes decrease in vasodilators like prostacyclin and nitric oxide, leaky capillaries and platelet aggravation. There is imbalance in the production of two prostaglandins, prostacyclin and thromboxane. Therefore, the development and severity of preeclampsia can be decreased with a low dose regimen of aspirin 60 mg/day from second trimester of pregnancy.

The collaborative low dose aspirin study pregnancy trial[2] involved pregnant women randomized to receive aspirin or placebo. Aspirin did not decrease the incidence of preeclampsia but it delayed the onset until a later gestational age. Its use is not a contraindication for regional anesthesia.

Classification of Preeclampsia

Mild preeclampsia is characterized by a sustained systolic blood pressure equal to, or greater than 140 mm Hg, a diastolic blood pressure equal to, or greater than 90 mm Hg, and proteinuria of 300 mg in a 24-hour urine collection.

Preeclampsia is considered severe when systolic pressure is 160 mm Hg or higher, diastolic pressure is 110 mm Hg or higher and there is proteinuria of at least 5 gm in 24-hour urine collection. Other features of *severe preeclampsia* include the following:

- Oliguria of less than 500 ml in 24 hrs
- Headache
- Visual disturbance (photophobia, diplopia, blurred vision)
- Cerebral disturbances (drowsiness, altered sensorium)
- Grand mal seizures
- Pulmonary edema
- Thrombocytopenia
- Impaired liver functions.

The triad of physiological derangements in preeclampsia is:

- Intense vasospasm—excess production or sensitivity to vasoconstrictors
- Plasma volume contraction following vasospasm, capillary leakage, and reduced plasma osmotic pressures
- Local or disseminated intravascular coagulation.

Pathophysiology[3]

There is multiorgan systemic hypoperfusion, leading to many systemic manifestations such as the following:

Cardiovascular System

Hypertension is a characteristic finding in preeclampsia which is abrupt and often progressive. Due to an increased sensitivity to catecholamines, majority of the patients have high systemic vascular resistance with resultant reduced circulating volume and increased total body water. There is considerable increase in the cardiac output (CO) resulting in a hyperdynamic circulatory state. However, with increasing severity of preeclampsia, the CO normalizes or even decreases in association with left ventricular dysfunction. CVP is essentially normal or slightly elevated in severe PIH. CVP may not correlate well with pulmonary capillary wedge pressure (PCWP). However, a CVP of less than 6 mm Hg is not associated with increased PCWP. Plasma volume is reduced in preeclamptic patients; however, fluid overload must be avoided. Decreased plasma protein levels reduce the colloid osmotic pressures. This along with endothelial damage and marked vasoconstriction will result in the development pulmonary edema. Sometimes patient may develop myocardial ischemia and ST-T changes.

Anesthetic Implications

After regional anesthesia, preeclamptic patients are more likely to suffer from hypotension due to decreased intravascular volume. Large fluid boluses used to minimize hypotension, are poorly tolerated by the patients. Due to exaggerated responses to vasopressors, ephedrine should be cautiously used to treat hypotension. An increased pressor response during intubation causes

large increase in mean arterial pressures and heart rate. This can lead to cerebral hemorrhage, left ventricular dysfunction and pulmonary edema.

Respiratory System

Preeclamptic patients have edematous airway (pharynx and larynx) causing difficult intubation which may be required for induction of general anesthesia, airway protection, or treatment of respiratory distress. Increased capillary permeability with low colloid osmotic pressures will further lead to interstitial fluid accumulation in the lungs.

Anesthetic Implications

Edema of face, lips and tongue causes difficult mask ventilation. Narrowing of the airway requires intubation with small sized endotracheal tube. Special equipments like bougie, McCoy laryngoscope, stubby handle, laryngeal mask airway, fiberoptic bronchoscope should be available.

Central Nervous System

Severe preeclamptic patients can present with headache, decreased vision, altered sensorium or seizures. Seizures may occur abruptly without underlying hypertension. Seizures are effects of cerebral ischemia due to vasospasm, cerebral edema, and microinfarcts. Raised intracranial pressures may lead to coma. Magnesium being a cerebrovasodilator is an effective treatment of choice.

Anesthetic Implications

Prophylactic administration of antihypertensive is essential for controlling of blood pressure. Intracranial hypertension can be exaggerated in the presence of hypercarbia acidosis and hypoxia.

Kidney

Preeclampsia is associated with decreased renal blood flow and glomerular filtration rate, along with increase in serum creatinine concentrations. Oliguria may occur in severe preeclampsia due to hypovolemia, decreased renal perfusion and renal artery vasospasm. Progression to acute renal failure (ARF) is rare. ARF is reversible in cases of acute tubular necrosis. However, in HELLP syndrome, patient may require hemodialysis.

Anesthetic Implications

Serum creatinine more than 1 mg/dl indicates severe renal involvement. It is associated with decreased elimination of drugs in renal failure patients. Hydration of patients for regional anesthesia should be CVP-guided to prevent fluid overload. Persistent oliguria may require diuretics, vasodilator therapy and dialysis.

Liver

Involvement of the liver will result in raised liver enzymes with or without epigastric and right upper quadrant pain. Pathologically, hepatic lesions include periportal hemorrhages, necrosis, generalized swelling, fibrin deposition, and rarely subcapsular hematomas. In HELLP syndrome, liver enzymes (SGOT/SGPT) are very highly elevated.

Anesthetic Implications

Hepatic involvement results in impaired drug metabolism. Decreased production of clotting factors may affect the coagulation profile of the patient. The prothrombin time may be increased. Blood and blood products should be available. Regional anesthesia may be contraindicated.

Fetoplacental Unit

Hypoperfusion caused by placental disease and vasospasm will result in intrauterine growth retardation and placental abruption. Abruptio placenta is defined as premature separation of the normally implanted placenta. In preeclamptic patients, it has been proposed that thrombotic lesions develop in the placental vasculature, causing decidual necrosis and placental separation. Thus, there is increased perinatal mortality associated with preeclampsia. Decrease uterine blood flow also predisposes to a hyperactive uterus and premature labor. Infants born to preeclamptic mothers are small and premature.

Hematological

Severe preeclampsia and eclampsia are commonly associated with coagulation abnormalities, primarily involving reduction in platelet number and function. Additionally, increased platelet aggregation causes a low-grade disseminated intravascular coagulation. Increased concentrations of fibrin degradation products are consistent with disseminated intravascular production. Causes of thrombocytopenia in preeclampsia are the following:

- Autoimmune mechanism
- Platelet aggregation at the site of endothelial dysfunction and activation due to imbalance between prostacyclin and thromboxane
- Platelet consumption
- Decreased life span of platelets.

Incidence of thrombocytopenia ranges from 15 to 20 percent and up to 50 percent in severe eclampsia.

Anesthetic Implications

Platelet count of 100000/mm^3 is adequate for administration of neuraxial block. It is controversial to give regional anesthesia for platelet count between 75000 – 100000/mm^3. Platelet count <50000/mm^3 requires platelet transfusion.

Other investigations which will give some idea about the patient's hemostasis, are:

- Bleeding time correlates with thrombocytopenia but not with platelet dysfunction
- Thromboelastography examines clotting function
- Prothrombin time, partial thromboplastin time and fibrinogen levels deteriorate with DIC.

Pathophysiology of PIH as shown in Table 14.1

Management

The definitive therapy for preeclampsia is delivery of the fetus and placenta. Thus, the main objective is to first stabilize the patient and then proceed with the delivery. Before any anesthetic intervention, it is important to ensure that hypertension is well controlled and other problems such as coagulopathy, eclampsia and pulmonary edema are treated. If not, there is an increased risk of fetoplacental and or maternal deterioration. Preeclampsia and or eclampsia account for 20 percent of the maternal and perinatal deaths.[4]

Table 14.1 Pathophysiology of preeclampsia

End-organ	*Mild*	*Severe*
CNS		
Seizures	None	Present
Headache	None	Present
Visual disturbances	None	Present
Papilledema	None	Present
Clonus	None	Present
GIT		
Liver tenderness	None	Present
Nausea and vomiting	None	Present
Epigastric pain	None	Present
Elevated liver enzymes	> 40 IU/L	> 70 IU/L
Hematological system		
Low platelets	< 1.5 lakhs/mm^3	< 1.0 lakhs/mm^3
Hemolysis	None	Present
DIC	None	Present
Cardiorespiratory system		
Pulmonary edema	None	Present
Renal		
Proteinuria	0.3/g per 24 hours	> 5g in 24 hours
Urine output	Normal	Oliguria

Preeclamptic patients present either for an emergency cesarean section, elective cesarean section or labor analgesia.

While anesthetising these patients we are especially worried about:

- Hypertensive crisis leading to intracerebral bleed and left ventricular failure
- Interstitial volume overload causing pulmonary edema
- Maternal hypotension causing placental hypoperfusion and neonatal morbidity
- Thrombocytopenia
- Airway edema causing difficult intubation.

Preoperative Assessment

Preoperatively, detailed history of the patient and physical examination should emphasize on airway examination, intravascular fluid status, control of blood pressure and coagulopathy. Laboratory investigations[5] should include complete blood count, coagulation profile, liver functions, renal functions and urine analysis. Routine coagulation screening is not recommended by all authors but if coagulopathy is suspected clinically, coagulation study must be done.

Antihypertensives[1] used for controlling blood pressure help not only the mother but have been proved to be beneficial for the neonate, if it is started well in advance 28 to 32 weeks of gestation. Preoperatively, patients may be on one or many antihypertensives like methyldopa, nifedepine and hydralazine, of which nifedepine is gaining importance as being safe and effective. It is given orally or sublingually as 5 to 10 mg dose 8 hourly. It is potent uterine relaxant and also increases renal blood flow. It is not associated with decreased uterine perfusion or fetal deterioration. Methyldopa (Aldomet) acts on α-2 receptors to decrease sympathetic outflow and hence reducing blood pressure. However, because of its sedating qualities and other side effects like fluid retention and postural hypotension its use may likely to be reduced.

Rapidly acting antihypertensives[1] include nitroglycerine, (NTG), sodium nitroprusside, labetalol and hydralazine.

- Nitroglycerine is a venodilator. Constant intravenous infusion is given at the rate of 5 to 50 μg/min. Infusion is prepared as 25 to 50 mg in 500 ml. Its duration of action is less than 2 min. It may cause fetal deterioration. We may need arterial line for monitoring blood pressure.

- Labetelol is β_1 and α-blocker, given as IV bolus 10 to 20 mg up to 1 to 3 mg/kg. Onset of action is within 1 to 2 min and duration of action is 2 to 3 hours. It improves uterine and placental blood flow. No special monitoring is required. Use cautiously in asthmatic patient and patients with compromised ventricular function.
- Hydralazine is an arterial vasodilator. It is used as 5 to 10 mg IV bolus. Maximum effect requires 20 to 30 minutes. It can cause maternal tachycardia, nausea, vomiting and headache. It is slower in onset and unreliable and can cause fetal distress.
- Nitroprusside is a direct acting arterial vasodilator. It is given as constant IV infusion at the rate of 0.05 to 10 μg kg/hr. Time of onset is usually less than 1 minute. Its duration of action lasts only for few minutes. No ill effects on fetus are seen. It is available as unstable solution. It needs arterial line monitoring. It can cause tachyphylaxis. Long-term use can cause cyanide toxicity.

Monitoring

In patients with mild preeclampsia routine monitoring with ECG, noninvasive blood pressure cuff and pulse oximetry is sufficient. For those with severe eclampsia radial arterial line is recommended for accurate measurement of blood pressure and collection of blood for ABGs and other laboratory investigations like coagulation profile and liver functions. CVP (central venous pressures) and PCWP (pulmonary capillary wedge pressures) monitoring should be initiated in patients with refractory hypertension, oliguria and pulmonary edema.

However, invasive hemodynamic monitoring is not essential for safe fluid management of all cases of severe preeclampsia.[6] Patients with severe preeclampsia with adequate urine output will tolerate prophylactic hydration. If the urine output is inadequate, a fluid challenge is done with 250 to 500 ml of crystalloid infused over 20 minutes. If the patient responds with an increase in urine output, additional fluid boluses may be given cautiously before the regional block. If there is no response to the initial fluid bolus, CVP or PCWP monitoring becomes necessary. If CVP monitoring alone is thought of, then a volume expansion to CVP of at least 6 to 8 mm Hg is considered to be safe and effective in mild preeclampsia. However, the CVP–PCWP gradients in severe preeclampsia may be as high as 8 to10 mm Hg. Therefore, CVP of 8 mm Hg might correspond to a PCWP as high as 18 mm Hg. This results in volume overload and possibly pulmonary edema. Therefore, if CVP alone is being monitored for fluid management, volume expansion to achieve a CVP of 4 mm Hg or less is sufficient.

Monitoring with a pulmonary artery catheter is indicated in patients with pulmonary edema or oliguria unresponsive to fluid therapy or intractable hypertension for better patient care.

Analgesia for Labor and Vaginal Delivery

Epidural Analgesia

Epidural analgesia is most suitable for preeclamptic patients. Pain relief causes decrease in the serum concentration of catecholamines resulting in increase in the uteroplacental and intervillous blood flow. This is of great benefit because preeclamptic patients have vasospasm of placental circulation. Another main advantage is that it can be rapidly extended to provide adequate anesthesia in emergency cesarean sections. With judicious use of fluids and slow induction of block hemodynamic stability can be maintained.

Technique

Patients with platelet count of 100,000/mm^3 and above can be safely given regional anesthesia. Hydration with 500 to 1000 ml of crystalloid is required. Mother should be monitored continuously. She should be oxygenated with facemask or nasal prongs. Local anesthetic of choice is low concentration of bupivacaine 0.0625 to 0.125 percent with fentanyl 2 μg/ml can be given as infusion at the rate of 10 to 12 ml/hour. Adrenaline containing local anesthetics should be avoided. Ephedrine 5 to 10 mg can be used to treat hypotension along with uterine displacement and additional fluid. Epidural infusion is preferred over intermittent boluses of local anesthetics (bupivacaine) as it gives continuous pain relief and causes less hypotension.

Combined Spinal and Epidural Analgesia (CSE)

Intrathecal opioid like fentanyl 25 mcg alone or in combination with bupivacaine 1.25 mg or 2.5 mg, followed by epidural infusion as mentioned above can be given. In a study done by Ramanathan et al,[7] use of CSE with low intrathecal doses of bupivacaine and epidural supplementation produced adequate anesthesia for cesarean delivery and analgesia for labor in patients with severe preeclampsia. However, there was significant fall in the mean arterial pressures (MAP) 16% ± 9% from the baseline with no significant correlation with umbilical artery pH of the neonate.

Choice of Anesthesia for Cesarean Section

- Regional
  1. Spinal
  2. Epidural
  3. Combined spinal epidural
- General.

Regional Anesthesia

Single shot spinal anesthesia is appropriate for the majority of women without labor epidural analgesia who require cesarean section. The anesthetic technique of choice for patients with epidural catheter *in situ* will be top up of that epidural anesthesia.[5] It is controversial that whether spinal anesthesia is safe for preeclamptic patients. With spinal anesthesia, there is a risk of severe hypotension since patients have depleted intravascular volume. Vasodilatatory actions of magnesium and antihypertensives may exaggerate hypotension.

A study conducted by Hood et al[8] compared the blood pressure effects of spinal and epidural anesthesia in severely preeclamptic patients requiring elective cesarean section. Spinal anesthesia produced blood pressure decreases that were similar to epidural anesthesia in these patients. Maternal and fetal outcomes also were similar. Total fluid resuscitation in spinal patients was 400 ml more than epidural patients. Total ephedrine used was small but not significantly different in both groups. This large retrospective study supports the use of either technique in the anesthetic management of these patients. However, a study done by Visalyaputra et al[9] shows that spinal anesthesia for cesarean delivery in severely preeclamptic patients causes slightly more hypotension than epidural anesthesia during the induction to delivery period. The duration of hypotension, however, was short and there was no difference in the neonatal outcome.

Compared to general anesthesia, regional anesthesia maintains uteroplacental circulation and causes decrease in the perinatal mortality. Anesthesia-related complications are the sixth leading cause of pregnancy-related death. Maternal mortality due to general anesthesia is 16 times more than that with regional anesthesia.[10] Preeclamptic patients have increased airway edema and possible difficult airway. Also there is exaggerated hypertensive response to endotracheal intubation.

Hyperbaric bupivacaine 0.5 percent is the local anesthetic of choice. The dose is approximately 10 to 12 mg. Doses as low 7.5 mg in combination, with 25 µg fentanyl have also been used combined with epidural anesthesia to extend the level and duration of the block. Van de Velde et al[11] performed a retrospective chart analysis to evaluate the effects of combined spinal-epidural anesthesia on maternal hemodynamics and fetal outcome compared to conventional epidural anesthesia. Combined spinal-epidural anesthesia appears to be safe as anesthetic technique for preeclampsia and severe preeclampsia. In the combined spinal-epidural anesthesia group more ephedrine was used compared to the epidural group (14.6 ± 4.4 vs. 3.6 ± 4.6 mg, $P < 0.05$). However, more lactated Ringer's was used in the epidural group. Umbilical artery pH was lower in the epidural group (7.26 ± 0.01 vs. 7.29 ± 0.02, $P < 0.05$).

Epidural anesthesia alone is also a preferred technique in severe preeclampsia especially if the cesarean section is not urgent. Epidural anesthesia usually involves a mixture of a local anesthetic and an opioid. This is associated with a slower and more gradual onset of action and hence a gradual and less dramatic drop in blood pressure. This has traditionally made it the regional anesthetic of choice in severe preeclampsia. In an urgent cesarean section either single shot spinal or general anesthesia is indicated.

Technique for Regional Anesthesia[6]

When a preeclamptic patient comes for cesarean section and she is stable with controlled blood pressure, normal coagulation and platelet count >100000/cmm:

- Two Large bore intravenous accesses are taken and monitors attached
- Aspiration prophylaxis with injection ranitidine 50 mg IV is given
- Confirm availability of blood and blood products.
- CVP monitoring is done as indicated
- 500 to 1000 ml of fluid is infused taking care not to increase the CVP more than 4 mm Hg
- Fetal monitoring to be continued till the beginning of the surgery
- Epidural catheter is inserted and after confirming its placement start administering either 2 percent lignocaine plain or 0.5 percent bupivacaine in 5 cc increments with or without 50 to 100 µgm fentanyl till blockade of light touch sensation up to T5 segments is achieved.[12]

General Anesthesia

In emergency cesarean sections, general anesthesia in PIH patients is indicated in cases of coagulopathy, fetal distress (no pre-existing epidural catheter), uncontrolled blood pressure, altered sensorium (mental irritability, drowsiness, blurred vision), respiratory failure and

patient refusal. The advantages of general anesthesia are its rapid onset of action and its reliability as a mode of anesthesia.

The hazards of general anesthesia[13] are:
- Potentially difficult intubation due to presence of laryngeal edema
- Aspiration of gastric contents
- Prolonged neuromuscular blockade if the patient is on $MgSO_4$
- Pressor response to laryngoscope
- Impaired intervillous blood supply
- Severe hypotension.

Technique for general anesthesia:
- Check anesthesia equipment for difficult intubation and suction machine
- Keep drugs like antihypertensives (labetalol, nifedepine, nitroglycerine patch and injections) to tackle hypertensive crisis intra and perioperatively.
- Ionotropes like adrenaline, noradrenaline and dopamine should be available
- Blood and blood products should be available
- Administer aspiration prophylaxis in the form of ranitidine 50 mg IV
- 2 peripheral lines to be taken
- Invasive hemodynamic monitoring (CVP and arterial line) should be done as indicated
- Preoxygenate the patient for three minutes. Apply standard monitors
- If the blood pressure is very high inspite of optimization with drugs like nifedepine and methyldopa, then to blunt the pressor response, one of the following drugs can be given with fetal heart rate monitoring
  - Hydralazine 5 to 10 mg IV 10 to 15 min before intubation
  - Labetalol 10 to 20 mg bolus up to 1 mg/kg IV 5 min before intubation
  - Fentanyl 2 to 2.5 µg/min IV 3 min prior to intubation
  - Lignocaine 1.5 mg/kg IV 90 seconds before laryngoscopy and intubation
  - NTG 1 to 2 µg/kg IV just before intubation
  - Sodium nitroprusside 0.1 to 3 µgm/kg/min IV just prior to laryngoscopy
  - NTG patch 5 to 10 mg can be attached transdermally
- Induction of anesthesia with thiopental 4 to 5 mg/kg and succinylcholine 1.5 mg/kg. Rapid sequence intubation with cricoid pressure. Anesthesia can be maintained with 50 percent nitrous oxide and isoflurane/sevoflurane 0.5 percent until delivery. Use vecuronium or atracurium for muscle relaxation in reduced doses
- Ergometrine should be avoided due to its propensity to cause a hypertensive crisis
- If the condition of the patient is stable then patient can be reversed and extubated.

Preeclamptic patients may be in respiratory distress due to pulmonary edema. In a study by Mushambi MC et al, 70 percent of the cases developed pulmonary edema 71 hours after delivery.[13]As maternal oxygen reserve is decreased in pregnancy, significant arterial desaturation will occur if the patient becomes apneic for even a short time. Such an episode increases the hypoxic risk to the fetus as well. Therefore, patients have to be treated with oxygen administration, diuretics, digitalis glycosides, fluid restriction and intermittent positive pressure ventilation preoperatively.

Management of Pulmonary Edema

Pulmonary edema[14] may be due to left ventricular dysfunction secondary to high systemic vascular resistance, iatrogenic volume overload as a result of contracted intravascular space, decreased plasma colloid oncotic pressure, or pulmonary capillary membrane injury. Colloid oncotic pressure may decrease further following intravenous fluid replacement with crystalloid and as a result of rapid intravascular mobilization of edema fluid after delivery.

Patients are usually in respiratory distress. They require resuscitation preoperatively with oxygen, diuretics and fluid restriction to reduce the preload and after load. Inj. Furosemide 1 to 2 mg/kg IV is given. At many instances severe preeclamptic patients may be started postoperatively on mannitol to reduce the cerebral edema. Mannitol causes increase in the intravascular volume. This can result in pulmonary edema. Hence, mannitol should be given cautiously. Patient should be started 100 percent FiO_2 and PEEP must be added to the assist control mode of ventilation.

Eclampsia

Eclampsia is defined as the occurrence of one or more grandmal convulsions in association with the syndrome of preeclampsia. It can occur in the prepartum (17%), intrapartum (52%) and postpartum (34%) period.[1] Eclampsia usually terminates 48 hours after delivery.

The pathophysiology of eclampsia is thought to involve cerebral vasospasm leading to ischemia, disruption of the blood–brain barrier and cerebral edema. Neurological complications may include coma, focal motor deficits and cortical blindness.

Cerebrovascular hemorrhage is a complicating factor in 1 to 2 percent of patients. Other complications of eclampsia include pulmonary edema, acute renal failure, placental abruption, coagulopathy and aspiration.

Severe and persistent headache, blurred vision, photophobia, irritability, transient mental changes, epigastric or right upper-quadrant pain, nausea, and vomiting are warning signals for the occurrence of eclampsia.

Principles of Treatment of Eclampsia[15]

- Protection of maternal airway
- Control of convulsions
- Prevention of further convulsions
- Treatment of severe hypertension
- Monitoring the fluid balance
- Deliver the baby safely as soon as possible
- Treatment of any complications that may arise

If the patient is convulsing, intravenous (IV) diazepam 5 mg boluses, repeated as required, upto a maximum of 20 mg can be given. Other drugs like midazolam 1 to 2 mg and sodium thiopentone 50 to 100 mg and phenytoin can also be used. This should be followed by IV infusion or IM injection of $MgSO_4$. The airway should be secured and the patient should be placed in the recovery position and given oxygen supplementation. Since convulsions are associated with metabolic acidosis, the determination of ABG is important. During resuscitation maternal blood pressure, SpO_2, electrocardiogram, and fetal heart rate should be monitored continuously.

There is enough evidence to favor the use of $MgSO_4$ rather than diazepam or phenytoin, for the treatment of eclampsia. Also one must aim to lower blood pressure and control fluid balance simultaneously to prevent further complications. In the Collaborative Eclampsia trial[16] women allocated $MgSO_4$ had a 67 percent lower risk of recurrent convulsions than those allocated phenytoin and a 52 percent lower risk of recurrent convulsions than those allocated diazepam. In the MAGPIE trial,[17] preeclamptic patients were prophylactically given $MgSO_4$ and the control group placebo. It was found that 24 percent of the women given $MgSO_4$ reported side effects versus 5 percent given placebo. Women allocated $MgSO_4$ had a 58 percent lower risk of eclampsia than those allocated placebo. Maternal mortality was also lower among women allocated $MgSO_4$. For women randomized before delivery, there was no clear difference in the risk of the infant mortality. The only notable difference in maternal or neonatal morbidity was for placental abruption.

$MgSO_4$ acts as vasodilator and a membrane stabilizer. This not only reduces cerebral ischemia but also blocks some of the subsequent neurological damage that may be associated with it. $MgSO_4$ may also exert its effects by blocking the *N*-methyl-D-aspartate receptor in the hippocampus, thus acting as a central anticonvulsant.

Magnesium Therapy [15]

Loading dose of $MgSO_4$ is 4 to 6 gm IV followed by 1 to 2 gm/hour IV infusion for at least 24 hours after the last seizure. $MgSO_4$ can also be given intramuscularly but it is painful. Loading dose is 4 gm IV bolus plus 5 gm IM in each buttock. This is then maintained by 5 gm IM every 4 hours, till 24 hours after the delivery or from the last convulsion.

Therapy can be monitored safely by hourly measurement of the patellar reflex and respiratory rate. If there are any signs of magnesium toxicity further doses must be withheld until these disappear. Significant respiratory depression should be treated with 1 gm calcium gluconate given IV over 10 minutes. As, magnesium is excreted by the kidneys regular monitoring of the serum levels should be considered in women with renal disease or in women with oliguria. The therapeutic range is believed to be between 2 and 4 mmol/l. If repeated seizures occur despite $MgSO_4$ therapy, options include diazepam (10 mg IV) or thiopentone (50 mg IV). Intubation may become necessary in such women in order to protect the airway and ensure adequate oxygenation. Further seizures should be managed by intermittent positive pressure ventilation and muscle relaxation.

Plasma magnesium levels and clinical effects as shown in table Table 14.2

Anesthetic Management

Unconscious patients with obtunded reflexes with evidence of raised CVP should have general anesthesia. The patient can be extubated at the end if awake and conscious, or else ventilated till BP is control and controlled weaning is done over a few hours.

Table 14.2 Clinical effects of magnesium

Plasma Magnesium levels (mEq/L)	*Clinical Effects*
1.5–2.0	Normal Level
4.0–4.8	Therapeutic range
5.0–10.0	Prolonged P-Q interval, Wide QRS
> 10.0	Respiratory depression / arrest
25.0	Cardiovascular collapse

Regional anesthesia to facilitate labor or delivery should be considered in patients who are seizure free, conscious with no evidence of raised CVP and absence of coagulopathy.

Epidural anesthesia is preferred in patients who are hemodynamically stable and with platelets > 100000 mm.^{3} Regional analgesia is contraindicated, if there is evidence of actual or incipient disseminated intravascular coagulation. Occasionally, a cesarean section has to be done too quickly to consider using epidural anesthesia. Although general anesthesia allows more precise control of the speed and timing of surgery, it carries its own complications. Intubation may be difficult, or impossible because of laryngeal edema, which may also cause postoperative respiratory obstruction and cardiac arrest. Laryngoscopy is a well-known cause of extreme transient reflex hypertension in all individuals. The problem is aggravated in eclamptic women and may be so extreme so as to cause acute pulmonary edema.

Management of Patient with HELLP Syndrome

It is associated with high maternal and perinatal morbidity and mortality. It is a variant of preeclampsia consisting of hemolysis, elevated liver enzymes and low platelet count. HELLP syndrome occurs in 2 to 12 percent of patient's with severe preeclampsia and 30 to 50 percent in eclampsia

Diagnostic features[5]

- Hemolysis
  - Presence of microangiopathic hemolytic anemia
  - Abnormal blood smear and increased bilirubin > 1.2 mg/dl
- Elevated liver enzymes
  - Increased aspartate aminotransferase (AST) > 70 IU/L
  - Lactate dehydrogenase (LDH) > 500 IU/L
- Platelet count < 100000/mm^3

Two types of HELLP syndrome are known:

1. Partial HELLP syndrome—one or two abnormalities present
2. Full HELLP syndrome—all abnormalities present.

Complications of HELLP syndrome include stroke, DIC, placental abruption, pleural effusion, acute renal failure, subcapsular hematoma, intraparenchymal hemorrhage, and need for blood transfusion. Clinical signs and symptoms include right upper quadrant pain, jaundice, nausea, vomiting, hypertension, headache, seizures.

The aim is to deliver the fetus as early possible. If fetus < 34 weeks steroid course is given for fetal lung maturity 48 hours before delivery. Platelet transfusions are indicated either before or after delivery in all patients with HELLP syndrome in the presence of significant bleeding from puncture sites, wound site, intraperitoneal regions, and extensive ecchymosis. Transfusion is indicated in all antepartum patients whose platelet count is less than 20000/mm^3. Platelets, red cell and fresh frozen plasma may also be needed in patients with more severe coagulopathies. Recombinant factor VIIa has been used in selected patients who have organ failure and refractory to treatment. Patients who develop postpartum HELLP syndrome have a higher incidence of pulmonary edema and renal failure. Dexamethasone increases the platelet number significantly. If the patient is seizure free, conscious with no evidence of ICP and absence of coagulopathy regional anesthesia can be given to facilitate labor or delivery. Epidural anesthesia is preferred in patients who are hemodynamically stable with platelets count more than 100000/mm^3. General anesthesia is preferred over regional anesthesia in the setting of complete HELLP syndrome.

REFERENCES

1. Gaiser RR, Gutsche BB, Cheek TG. Anesthetic considerations for Hypertensive disorders for Pregnancy. In: Hughes SC, Levinson G, Rosen MA, (Eds). Schnider and Levinson's Anesthesia for obstetrics. (4th edn). Philadelphia: Lippincott William Wilken's 2002;297-321.
2. CLASP (Collaborative Low-dose Aspirin Study in Pregnancy) Collaborative Group. CLASP: A randomised trial of low-dose aspirin for the prevention and treatment of preeclampsia among 9364 pregnant women. Lancet 1994;343(8898):616-7.
3. Stoelting RK, Dierdorf SF. Diseases associated with pregnancy. In: Anaesthesia and coexisting disease. (4th edn). New Delhi: Elsevier 2002;657-64.
4. Malinow AM. Preeclampsia. In: Roizen MF, Fleisher LA, (Eds). Essence of anaesthesia practice. (2nd edn). USA: WB Saunders 2002;268.
5. Birnbach DJ, Ingrid M. Browne IM. Anaesthesia for obstetrics. In: Miller RD, (Eds). Miller's Anaesthesia, (6th ed). Philadelphia: Chruchill Livingstone 2005;2329-33.
6. Ramanathan J, Bennett K. Preeclampsia: Fluids, drugs, and anesthetic management. Anesthesiology Clinics of N Am. 2003;21:145-63.
7. Ramanathan J, Vaddadi AK, Arheart KL. Combined spinal and epidural anaesthesia with low doses of intrathecal bupivacaine in women with severe preeclampsia: A preliminary report. Reg Anesth Pain Med. 2001;26(1):46-51.
8. Hood, David D, Curry, Regina RN. Spinal versus Epidural Anaesthesia for Cesarean Section in Severely Preeclamptic Patients: A Retrospective Survey Anesthesiology 1999;90 (5);1276-82.

9. Visalyaputra S, Rodanant O, Somboonviboon W, Tantivitayatan K, Thienthong S, Saengchote W. Spinal Versus Epidural Anaesthesia for Cesarean Delivery in Severe Preeclampsia: A Prospective Randomized, Multicenter Study. Anesth Analg 2005;101:862-8.
10. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anaesthesia-related deaths during obstetric delivery in the United States. Anesthesiology 1997; 86(2):277-84.
11. Velde V, Berends M, Spitz NB, Teunkens A, Vandermeersch E. Low-dose combined spinal-epidural anaesthesia vs. conventional epidural anaesthesia for Caesarean section in pre-eclampsia: A retrospective analysis. European Journal of Anesthesiology 2004;21 (6): 454-9.
12. Levy DM. Emergency Caesarean section: Best practice. Anaesthesia 2006;61:786-91.
13. Mushambi MC, Halligan AW, Williamson K. Recent development in the pathophysiology and management of preeclampsia BJA 1996;76:133-48.
14. Wahjoeningsih S. Management of perioperative pulmonary edema in a patient with preeclampsia/ eclampsia undergoing C-section. Folia Medica Indonesiana 2005;Vol. 41(2):138-41.
15. Salha O, Walker JJ. Modern management of eclampsia. Postgrad Med J 1999;75:78–82.
16. The Eclampsia Trial Collaborative Group. Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial. The Lancet 1995;345 (8963):1455-63.
17. The Magpie Trial Collaborative Group. Do women with preeclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: A randomised placebo-controlled trial. The Lancet 2002;359(9321):1877-90.

15 Anesthesia for Hemorrhagic Obstetric Patient

Smita S Lele, Urvi H Desai, Manju N Gandhi

KEY POINTS

- Hemorrhagic complications of pregnancy represent one of the extreme challenges to the anesthesiologist.
- These are life-threatening to the mother-to-be, the fetus, or both.
- Bleeding can be catastrophic.
- Maternal response to bleeding may be misleading and pregnant patients are at high-risk of developing DIC.
- The visual estimate of vaginal bleeding often does not reflect the extent of intravascular volume deficit in patients with obstetric hemorrhage.
- The incidence of placenta accreta is increasing due to higher cesarean delivery rate.
- Patients with placenta accreta have high-risk of postpartum hemorrhage.
- Uterine atony is the most common cause of postpartum hemorrhage.
- Aggressive drug therapy must be instituted in cases of severe uterine atony.
- Uterine atony and placenta accreta are the common indications for obstetric hysterectomy.
- Prophylactic placement of internal iliac artery balloon catheters by interventional radiologist may decrease blood loss and morbidity when patient with known placenta accreta is to undergo cesarean section.
- Assessment of urine output and laboratory studies (Hemoglobin, arterial blood gases) should be used to assess intravascular volume deficit and to guide fluid and blood replacement therapy.
- Arterial ligation, uterine compression sutures, and embolization therapy may reduce the need for peripartum hysterectomy.

INTRODUCTION

There is progressive decrease in maternal mortality and morbidity[1]due to advances in obstetric care. Though better prenatal care, more accurate diagnostic methods, and availability of intensive care during parturition has improved maternal outcome, however the precipitating factors for obstetric hemorrhage have not been eliminated. Some parturients sustain profound peripartum blood loss that overwhelms compensatory mechanisms.

Maternal mortality secondary to hemorrhage has not decreased and hemorrhagic complications remain the most common reason for intensive care unit admission.[2]

Several risk factors should be remembered:

- Older maternal age at childbirth is associated with higher incidence of pregnancy related disorders
- Assisted reproductive technology has led to higher incidence of multiple gestational pregnancy
- Cesarean delivery results in uterine scarring and increases risk of placenta previa and placenta accreta in subsequent pregnancies.

Common problems leading to morbidity and mortality are failure to recognize risk factors, failure to estimate blood loss accurately and failure to initiate treatment quickly.

Anesthesia providers have the skills to manage these patients, but understanding of maternal physiology and an appreciation of rapidity with which parturients can become unstable is very important. Early involvement of anesthesiologist in the care of bleeding pregnant woman is must for the safety of mother.

Timely and effective communication among all obstetric caregivers is essential.

Etiology and Classification

Divided by 20th week of gestation, causes of obstetric hemorrhage can be classified as follows:[3]

- **Early pregnancy** (<20 weeks of gestation)
- Miscarriage
- Ectopic pregnancy
- Vesicular mole
- Reproductive tract tumors
- **Late pregnancy** (>20 weeks of gestation)
  It can be antepartum or postpartum hemorrhage.

Antepartum Hemorrhage

- Placenta previa
- Abruptio placentae
- Vasa previa
- Uterine rupture.

Postpartum Hemorrhage

- Retained products of placenta
- Uterine atony
- Uterine inversion
- Trauma to genital tract.

Predisposing Factors for Hemorrhage

There are a number of clinical conditions which increase the risk of hemorrhage in the obstetric patient.[4,5] These are listed below:

A. Abnormalities of placenta—Congenital abnormalities: bicornuate uterus, Abnormal location: placenta previa, Abnormal invasion: placenta accreta/increta/percreta, Aquired structural abnormalities: leiomyoma, previous surgery, Peripartum problems: uterine inversion, uterine rupture, placental abruption.
B. Coagulation disorders—Congenital: von Willebrand's disease, Acquired: disseminated intravascular coagulation (DIC), dilutional coagulopathy, heparin.
C. Trauma during labor and delivery—Episiotomy, complicated vaginal delivery, low or mid-forceps delivery, cesarean section or hysterectomy, uterine rupture.
D. Uterine atony—Overdistended uterus, e.g. Large or multiple fetuses, hydramnios, distension due to clots; Anesthesia-Halogenated agents, conduction analgesia with hypotension; Exhausted myometrium—Rapid labor, prolonged labor, oxytocin or prostaglandin stimulation, chorioamnionitis; Previous uterine atony.
E. Retained uterine contents—Blood clots, products of conception.
F. Other factors—Obesity, previous postpartum hemorrhage.

Hemorrhage in Early Pregnancy

Obstetric disease of early pregnancy may result in significant maternal mortality and morbidity.

Specific obstetric problems of early pregnancy include ectopic pregnancy, abortion and gestational trophoblastic disease. These will be discussed in following section.

Ectopic Pregnancy

Ectopic pregnancy occurs when fertilized ovum implants outside the endometrial lining of the uterus. Death, infertility and recurrent ectopic pregnancy are possible sequelae. Rupture of ectopic pregnancy is the leading cause of pregnancy related maternal death in first trimester.

Risk factors[6] for above condition are:

1. Prior ectopic pregnancy.
2. Prior tubal surgery.
3. Pelvic inflammation.
4. Congenital anatomic distortion.
5. Previous pelvic or abdominal surgery.
6. Use of intrauterine contraceptive device.
7. Delayed ovulation.
8. History of infertility and assisted reproductive technology procedures, e.g. zygote transfer to fallopian tubes.[7]

Possible Sites of Ectopic Pregnancy

Tubal—infundibular or fimbrial, ampullary, isthmic, interstitial or corneal. Cervix, vagina, ovary, abdomen, cesarean scar.

Clinical Presentation

Classic clinical signs of impending rupture or ruptured ectopic pregnancy include abdominal pelvic pain, delayed menstrual cycle and vaginal bleeding. Pain often precedes vaginal bleeding. Patients with hemorrhage often experience dizziness or syncope. Urge to defecate and shoulder pain may be present.

Physical findings include abdominal tenderness with or without rebound, uterus that is smaller than expected for date, a tender adnexal mass. A bulging cul-de-sac suggests hemoperitoneum.

With significant hemorrhage, orthostatic changes in blood pressure and heart rate or frank shock may occur.

Diagnosis: Pelvic pain with a positive pregnancy test result indicates the need to rule out an ectopic pregnancy.

Diagnostic modalities available are:

- Ultrasonography[8]—transvaginal, transabdominal
- Serial beta-HCG concentrations
- Serum progesterone
- Uterine curettage.

Obstetric Management

Management options for ectopic pregnancy are expectant, medical and surgical. Choice depends on symptoms and diagnostic findings.

Expectant management: It is used for selected, asymptomatic patients with early tubal ectopic pregnancies and stable or decreasing beta-HCG levels. If it fails medical or surgical approach is required.

Medical management: Systemic, intramuscular, oral or intragestational forms of chemotherapy have been tried for medical management[9] of ectopic pregnancy. Methotrexate is commonly used for this purpose. Side effects include abdominal pain, vomiting, stomatitis, neutropenia and pneumonitis.

Surgical management: It depends on location of ectopic pregnancy, hemodynamic stability of the patient, available equipment and surgeons expertise.[10]

Laparoscopy or laparotomy is performed.

Anesthetic Management

Patients with unruptured tubal pregnancy have normal intravascular volume and minimal bleeding during procedure. The risk of anesthesia and surgery is low. Laparoscopy or laparotomy can be performed safely with spinal, epidural or general anesthesia.

Ruptured ectopic pregnancy may be associated with significant preoperative blood loss.

General anesthesia is required for these patients. Suggested plan for general anesthesia:

General Considerations

1. Blood grouping and cross matching.
2. Pulmonary aspiration prophylaxis.
3. Monitoring—ECG, blood presture, pulse oximetry, temperature, etc.
4. One or two large bore intravenous lines.
5. Foley catheter.

General anesthesia—Rapid sequence induction with cricoid pressure for acid aspiration prophylaxis.

Induction with thiopentone, propofol or ketamine

Muscle relaxant (Succinylcholine or Rocuronium) for intubation and Atracurium or Vecuronium for surgery.

Maintenance with oxygen, nitrous oxide, opioid, and volatile anesthetic agent or propofol infusion as tolerated.

Placement of Ryles tube.

Reversal and extubation if patient is awake.

Intraoperative autologous blood transfusion can be used.

Invasive monitoring must be used when patient is hemodynamically unstable.

Gestational Trophoblastic Disease

In normal pregnancy trophoblastic tissue forms the placenta. Abnormal trophoblastic proliferation of the human placenta results in gestational trophoblastic neoplasia (GTN).

The spectrum of the GTN shows four histologic patterns:

- Hydatidiform mole
- Invasive mole (chorioadenoma destruens)
- Choriocarcinoma
- Placental site tumor.

Clinical Classification

- Hydatidiform mole—complete or classic
  partial or incomplete
- Malignant gestational trophoblastic neoplasia—
  Nonmetastatic (confined to uterus).

Metastatic

1. Low-risk—no high-risk factors
2. High-risk
   a. Initial serum beta-HCG levels >40000 mIU/lit
   b. Long duration of disease (>4 months)
   c. Brain or liver metastasis
   d. Failed prior chemotherapy
   e. Antecedent term pregnancy
   f. Placental site tumor.

Diagnosis

Patients with partial mole usually have preoperative diagnosis of incomplete or missed abortion.[11]

Patients with complete mole present with vaginal bleeding after delayed menstrual period, and they often spontaneously pass hydropic vesicles.

Absence of fetal heart, a uterus that is large for gestational age and markedly elevated concentrations of beta-HCG strongly suggest diagnosis of hydatidiform mole. Ultrasonography shows characteristic multiechogenic regions that indicate hydropic villi or hemorrhagic foci.

Complications of complete molar pregnancy:

1. Excessive uterine size.
2. Ovarian theca lutin cysts.
3. Hyperemesis gravidarum—Volume depletion and electrolyte disturbances.
4. Pregnancy induced hypertension (PIH)—GTN should be strongly suspected in any patient presenting with PIH in early pregnancy.
5. Anemia.
6. Hyperthyroidism—Due to thyrotropin like effect of beta-HCG.
7. Acute cardiopulmonary distress—It is usually due to trophoblastic embolization. Other causes are high output cardiac failure due to thyrotoxicosis, pulmonary congestion due to anemia, PIH, aspiration pneumonitis, or iatrogenic fluid overload.
8. Malignant sequelae (metastasis).
9. DIC.
10. Infection.

Obstetric Management

- Suction evacuation
- Hysterectomy (for malignant GTN).

Anesthetic Management

- Evaluation for specific complications
- Evaluation for cardiopulmonary problems.

Preoperative tests—complete blood count including platelet count, coagulation function studies, renal and liver function tests, blood grouping and cross matching, beta-HCG levels, and chest radiogram.

General anesthesia is preferred due to potential for rapid blood loss during suction and evacuation. Thiopental, ketamine or etomidate is used for induction. Maintenance is achieved with oxygen, nitrous oxide, benzodiazepine and an opioid. Volatile anesthetic agents are avoided.

Intravenous oxytocin[12] infusion is started during procedure (20 U/L) for contraction of uterus and safe curettage.

Monitoring should continue in postoperative period for any evidence of uterine bleeding or cardiopulmonary distress.

Abortion and Intrauterine Fetal Demise

Abortion refers to a pregnancy loss or termination, either before 20 weeks of gestation or when the fetus weighs less than 500 gm. It can occur spontaneously, or is performed electively for personal or medical reasons.

Etiology of Spontaneous Abortion

Chromosomal abnormalities,[13] Immunologic mechanisms, maternal infections, endocrine abnormalities, uterine anomalies, incompetent cervix, debilitating maternal disease, trauma, environmental exposure (radiation, smoking, and certain drugs).[14]

Clinical Presentation

Definition of types of abortion:

Threatened abortion: It is defined as uterine bleeding without cervical dilatation before 20 weeks of gestation. Cramping or backache may accompany bleeding. Activities are restricted once the diagnosis is confirmed and till the symptoms resolve.

Inevitable abortion: It is defined as cervical dilatation or rupture of membranes without expulsion of the fetus or placenta. Spontaneous expulsion usually occurs but infection is a complication.

Complete abortion: It is defined as complete, spontaneous expulsion of the fetus and placenta. Partial expulsion of uterine contents may occur which is called incomplete abortion. It is common at 8 to 12 weeks of gestation. Persistent bleeding and cramping after expulsion of tissue are signs of incomplete abortion. Dilatation and evacuation (D&E) is required for this condition.

Missed abortion: In this condition fetal death may go unrecognized for several weeks. Some times coagulation defects may complicate intrauterine fetal death. If spontaneous expulsion of uterine contents does not occur, then evacuation of the uterus is required.

Recurrent or habitual abortion: In this condition three or more consecutive spontaneous abortions occur.

Septic abortion: Abortion is complicated by serious infection. It is more common with illegal and induced abortion. It causes significant morbidity and can be life threatening.

Treatment of Septic Abortion

- Send blood culture and start broad spectrum antibiotics
- Fluid therapy, blood transfusion and vasoactive drug therapy is needed if hemodynamics of the patient is unstable (septic shock)
- Exclude lower genital tract and bowel injury
- Prompt re-evacuation of the uterus
  Occassionally hysterectomy is required.

Obstetric Management

Dilatation and evacuation (curettage)—D and E

Complications of D and E procedure are cervical laceration, uterine perforation, hemorrhage, retained products of conception, infection, vasovagal events, post abortal syndrome and DIC.

Anesthetic Management

Several anesthetic techniques are appropriate for dilatation and evacuation.

Choice depends on different factors like dilatation of cervix, presence of blood loss, sepsis, full stomach condition and emotional state of the patient.

Dilatation is painful; suction evacuation is less painful.

This procedure is performed with patient in lithotomy position. After the procedure patient's legs are lowered to supine position. If the patient has lost significant amount of blood then hypotension may develop with change of position.

SUGGESTED PLAN FOR ANESTHESIA

General Considerations

- Blood grouping and cross matching must be ready
- Aspiration prophylaxis
- Routine noninvasive monitors should be used – ECG, BP, pulse oximetry, temperature, peripheral nerve stimulator
- One peripheral IV catheter with crystalloid solution
- Neuraxial anesthesia for hemodynamically stable patient without sepsis
  - Prehydration
  - Oxygen supplementation
  - Minimal sedation
- Oxytocin and ergot alkaloids should be ready
- Postprocedure monitoring especially for hypotension.

Monitored Anesthesia Care

Intravenous analgesia with fentanyl along with Midazolam or propofol for sedation with paracervical block is well-tolerated during D and E.

Spinal Anesthesia

Bupivacaine (0.5%) along with/without fentanyl is used to achieve a block up to T8 to T10 level.

Epidural Anesthesia

Mid lumbar epidural catheter

Lignocaine with epinephrine and fentanyl is given to get blockade up to T8 to T10 level.

General Anesthesia

Thiopentone, propofol or ketamine is used for induction depending on patient's hemodynamic condition.

If patient is not starving then rapid sequence induction with cricoid pressure and endotracheal intubation is performed.

If patient is with empty stomach then face mask or LMA can be used.

End tidal CO_2 monitoring should be used.

Maintenance

O_2 and N_2O are used along with opioid, benzodiazepines and/or propofol.

Low concentration (< 0.5% MAC) inhalational agent can be added if no evidence of uterine atony.

Extubation should be done only when patient is fully awake.

ANTEPARTUM HEMORRHAGE (APH)

Causes of APH range from cervicitis to abnormalities of placenta including placenta previa and placental abruption.

Following causes will be discussed.

- Placenta previa
- Abruption of placenta
- Uterine rupture
- Vasa previa

Placenta Previa

When placenta implants in advance of presenting part, it is placenta previa.

Classification can be made on basis of relationship of placenta with cervical os:

Total—completely covers cervical os

Partial—covers part of the cervical os

Marginal—lies close to but does not cover cervical os.

Etiology—Exact etiology is not known.

Prior uterine trauma is a common element in associated conditions.

Conditions/Risk factors[1] are:

1. Multiparity.
2. Advanced maternal age.
3. Previous cesarean section or other uterine surgery.
4. Previous placenta previa.

Diagnosis

Classic sign is painless vaginal bleeding during second or third trimester.

First episode is typically preterm, not associated with abdominal pain or abnormal uterine tone, rarely causes shock and characteristically stops spontaneously. Fetal compromise or demise is uncommon with first episode of bleeding.

Obstetric Management

Ultrasonography—Mainstay of diagnosis and it is useful for assessment of gestational age.

MRI is useful but not practical.

Obstetric management depends on severity of vaginal bleeding and maturity of fetus.

Active labor, mature fetus and/or persistent bleeding should prompt abdominal delivery.

Fetus is at risk due to two distinct pathophysiologic processes:

1. Progressive or sudden placental separation that causes uteroplacental insufficiency.
2. Preterm delivery and its consequences.

Expectant Management

Goal is to delay delivery until fetus is mature.[15] It includes following steps:

1. Prevention of hemorrhage by limiting physical activity and avoiding per vaginal examination.
2. Monitoring—maternal vital signs and Hb - Fetal evaluation: nonstress test, biophysical profile, USG, fetal lung maturity studies.
3. If diagnosed between 24 to 34 weeks of gestation corticosteroid to accelerate fetal lung maturity is administered.
4. Tocolytic therapy—obstetrician must balance potential cardiovascular consequences of tocolytic therapy in presence of maternal hemorrhage against consequences of preterm delivery.[16]

Anesthetic Management

Anesthesiologist should evaluate the patient with special emphasis on:

- Airway examination
- Evaluation of volume status
- History of previous cesarean section.

One large bore intravenous line must be started and blood should be sent for hematocrit, grouping and cross matching. Anesthesiologist must start volume resuscitation with nondextrose containing balanced salt solution.

Double Set-up Examination

During vaginal examination for diagnosis of placenta previa, sudden and severe maternal hemorrhage may occur, necessitating immediate cesarean section. Therefore before the examination, with patient in lithotomy position, the abdomen is prepared and draped and all preparations for cesarean section are completed. One large bore intravenous line should be started and two units of blood should be available before examination. Preparation for general anesthesia like administration of nonparticulate antacid and presence of skilled assistant to give cricoid pressure should be available.

Cesarean Delivery

Choice of anesthetic technique depends on indication and urgency of delivery and severity of maternal hypovolemia.

If no active bleeding

Neuraxial anesthesia may be given.

There is a risk of increased intraoperative blood loss due to following reasons:

1. Obstetrician may cut into the anteriorly placed placenta during uterine incision.
2. After delivery, lower segment uterine implantation site does not contract well.
3. Patient with placenta previa is at increased risk of having placenta accreta.

Two large bore IV lines should be started and two to four units of packed RBCs must be ready in the operating room.

If Active Bleeding

When the mother is actively bleeding and may be in hemorrhagic shock, resuscitation may be extremely difficult. It may not be possible to correct blood loss completely before surgery as the bleeding will continue till the placenta is removed. Blood, crystalloid or colloid should be infused as rapidly as possible.

Simultaneous evaluation, resuscitation and preparation for operative delivery is must.

Anesthesia Management

Induction: Rapid sequence induction with an appropriate dose of thiopental, etomidate or ketamine followed by succinylcholine or rocuronium for facilitating endotracheal intubation.

Maintenance: Depends on maternal cardiovascular stability.

O_2, N_2O and low-dose inhalational agents; or

O_2, N_2O and opioids with benzodiazepines after delivery of baby.

Muscle relaxants atracurium or vecuronium can be used for optimum operating conditions.

Ketamine stimulates the sympathetic nervous system centrally to increase heart rate and BP.

In severely hypovolemic patient in whom peripheral vasoconstriction is already maximal, ketamine will not cause any increase in BP. In these patients direct cardiac depressant effect of ketamine may produce decrease in BP.

In some situations with profound hypotension, endotracheal intubation should be facilitated with succinylcholine alone. Possibility of maternal recall should be secondary to maternal safety.

Placenta Accreta, Increta and Percreta

Placental implantation directly onto or into the myometrium gives rise to one of the three conditions:

- *Placenta accreta:* Implantation onto myometrium.
- *Placenta increta:* Implantation into myometrium.
- *Placenta percreta:* Penetration through full thickness of myometrium.

With placenta percreta implantation may occur onto bladder, bowel, other pelvic organs and vessels. This produces markedly adherent placenta, which cannot be removed without tearing myometrium .These abnormal implantations occur more frequently in patients with placenta previa. Placenta increta and percreta are rare but can be diagnosed antenatally by ultrasound.

Massive intraoperative blood loss is common. These patient's are likely to develop coagulopathy and may need cesarean hysterectomy. Placenta accreta is not reliably diagnosed until uterus is open. The anesthesiologist must keep in mind this possibility and be prepared to treat sudden, massive blood loss. For patients with placenta increta likelihood of massive intraoperative blood loss and cesarean hysterectomy are markedly higher than placenta accreta. With placenta percreta maternal hemorrhage and death can occur despite adequate preparation and expert management.

Cardiovascular collapse secondary to amniotic fluid embolism like syndrome may occur.

If diagnosis of placenta percreta is made antenatally, uterine incision remote from placenta can be made, umbilical cord is clamped, baby is delivered, placenta is allowed to remain *in situ* and uterus is closed. Controlled hysterectomy can then be performed or the abdomen is closed and patient is watched carefully with or without methotrexate administration to facilitate placental involution.

Abruptio Placentae

It refers to separation of normally implanted placenta after 20 weeks of gestation and before the birth of fetus.

Etiology- Not well-defined. It is commonly associated with hypertensive disorders of pregnancy, high parity, uterine abnormalities, previous placental abruption, cocaine use and trauma.

Clinical manifestations depend on site and degree of placental separation and amount of blood loss. Placental abruption leads to fetal compromise because surface area for maternal-fetal exchange of oxygen and nutrients is lost.[17]

Classification

A. *External:* Bleeding can be revealed (appears through vagina)
   Internal: Bleeding can be concealed (in uteroplacental unit)
   Degree of revealed bleeding is often misleading and concealed bleeding is one of the main problem for anesthesiologist caring for these patients. Amount of blood loss is commonly underestimated.
B. Abruption can be *mild, moderate or severe*
   Mild to moderate—No maternal hypotension, no coagulopathies and no fetal distress.
   Severe—Maternal hypotension, uterine irritability, hypertonicity, pain, fetal distress or death and clotting abnormalities.

Clotting Abnormalities Associated with Abruption

Two theories:

1. Abruption causes circulating plasminogen to be activated, which enzymatically destroys circulating fibrinogen (fibrinolysis).
2. Thromboplastin from placenta and decidua triggers the activation of extrinsic clotting pathway, causing thrombin to convert fibrinogen to fibrin *disseminated intravascular coagulation* (DIC).

End result is hypofibrinogenemia, platelet deficiency, and decreased factor V and VIII.

Once clotting mechanism is activated, degenerating products of fibrin-fibrinogen system appear in circulation. Patient then manifests widespread bleeding from intravenous sites, gastrointestinal tract, subcutaneous tissues as well as uterus.

Coagulation profile to be monitored.

When blood is drawn for clot observation test, a sample must be sent for hemoglobin (Hb), hematocrit

(HCT), platelet count, prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen level, fibrin degradation product (FDP).

Management

Definite management: To empty the uterus

Method by which it is accomplished depends on degree of abruption, time of gestation, stability of maternal hemodynamics and status of the fetus.

Mild-to-moderate abruption—Exclude placenta previa, if fetus is mature then amniotomy is performed to induce labor. If hemodynamics and clotting parameters are normal then epidural or spinal anesthesia can be administered.

Severe abruption—For emergency cesarean section regional anesthesia is contraindicated.

General anesthesia is given. After delivery, intensive resuscitation of newborn is needed. After delivery of infant and removal of placenta, subsequent management of mother may continue to be difficult. If blood has extravasated into myometrium, uterus may not contract and bleeding may continue. This condition can be treated with the following steps:

1. Oxytocin, ergot preparations or prostaglandins.
2. Interventional radiological procedures like selective uterine artery embolization.
3. Hypogastric or internal iliac artery ligation.
4. Postpartum hysterectomy.

To maintain circulating blood volume rapid blood transfusion, crystalloid or colloid administration is needed. These patients regardless of mode of delivery are extremely susceptible to postpartum hemorrhage resulting from uterine atony. Frequent monitoring of maternal vital signs, urine output and fundal firmness is necessary.

Uterine Rupture

Rupture of gravid uterus can be disastrous to both mother and fetus. Fortunately it is rare event.

Risk factors are previous uterine surgery, trauma (blunt trauma, excessive manual fundal pressure, extension of cervical lacerations, intrauterine manipulation and forceps application), inappropriate use of oxytocin, grand multiparity, uterine anomalies, placenta percreta, tumors (trophoblastic disease, cervical carcinoma) and fetal problems (macrosomia, malposition anomalies).[18,19]

Diagnosis

Presentation is variable. It should be suspected when vaginal bleeding, hypotension, cessation of labor and fetal compromise is present. A nonreassuring fetal heart rate pattern is considered to be the most reliable sign of uterine scar dehiscence or scar rupture.

Treatment

- Uterine repair
- Arterial ligation
- Hysterectomy.

Anesthesia Management

Patient evaluation and resuscitation begins simultaneously as the patient is prepared for emergency laparotomy.

General anesthesia is necessary except in some stable patients with pre-existing epidural catheter. Aggressive volume replacement and maintenance of urine output is very important.

Vasa Previa

It is defined as velamentous insertion of fetal blood vessels over the cervical os (Fetal vessels traverse the fetal membranes ahead of presenting part). The fetal vessels are not protected by placenta or umbilical cord and can get compressed by presenting part leading to fetal hypoxia.[20] Rupture of membranes is accompanied by tearing of fetal vessels, which may lead to exsanguination of fetus.

It is a condition that occurs rarely. There is no threat to the mother because it involves loss of fetal blood. It is associated with highest fetal mortality. This condition is associated with multiple gestation.

Diagnosis

Vasa previa should be suspected whenever bleeding occurs after rupture of membranes and is accompanied by FHR decelerations or fetal bradycardia. Antenatal USG[21] may sometimes diagnose the condition. If time permits, diagnosis can be made by examination of shed blood for fetal Hb.

Obstetric Management

Treatment is directed solely towards ensuring fetal survival.

- Hospitalization.
- Delivery of fetus by abdominal route.
- Neonatal resuscitation requires immediate attention to neonatal volume replacement.

Anesthetic Management

General anesthesia with rapid sequence induction is required.

Other causes of APH are cervical polyps, malignancy, vaginal or vulval varicosities, circumvallate placenta, vasa previa. The last two conditions present threat to fetus as bleeding is primarily from fetal vessels. Management of vasa previa is discussed above.

Other obstetric problems like severe pre-eclampsia, maternal infection, amniotic fluid embolism and intrauterine death can result in DIC and hemorrhage if not diagnosed and treated early.

POSTPARTUM HEMORRHAGE (PPH)

Hemorrhage following delivery is due to excessive bleeding from placental implantation site, trauma to the genital tract and adjacent structures or both. Postpartum hemorrhage is an event and not the diagnosis.

It can be:

Primary: Occurs during first 24 hours after delivery.

Secondary: Occurs between 24 hours to 6 weeks postpartum.

Traditionally PPH is defined as blood loss greater than 500 ml after delivery. The real definition of clinical value would be a blood loss leading to hemodynamic instability and compromised oxygen carrying capacity.

Deaths from PPH can be prevented by understanding the common problems:

- Failure to recognize risk factors
- Failure to estimate the degree of blood loss
- Failure to initiate treatment of severe hemorrhage
- Delay in surgical management
- Nonavailibility of blood and blood products
- Delay in involving other specialities

Etiology

1. Retained placenta
2. Uterine atony
3. Trauma to genital tract
4. Uterine inversion.

Severe PPH can occur with little or no warning. For this reason, immediately after delivery the anesthesiologist should observe the patient closely and be prepared to institute resuscitation of mother and to give an anesthetic at a few seconds notice.

Retained Placenta

After delivery of baby if there is failure to deliver placenta completely then it is called retained placenta.[22] Fragments of the placenta may remain inside the uterus and it can be a cause of early as well as late postpartum hemorrhage.

Treatment: This requires manual exploration of the uterus. This can be accomplished without an analgesic or anesthetic, but the mother can be severely disturbed and obstetrician can encounter difficulty in exploring uterus that is partially contracted.

Anesthesia Technique

- Regional block if epidural is already in place
- Benzodiazepines and ketamine
- Inhalational analgesia
- Judicious use of opioids

Careful observation of mother to prevent oversedation and potential aspiration both during and after removal of placenta.

Uterine relaxation is required for removal of placenta.

Nitroglycerine (NTG) 50-100 µg can be administered intravenously[23] or sublingually[24] for uterine relaxation.

Careful monitoring of maternal vital signs is must.

Advantages of NTG are avoidance of general anesthesia; removes the risk of failed intubation and aspiration and immediate onset of uterine relaxation; rapid maternal recovery.

Disadvantages of NTG are hypotension and headache but effect wears off within few minutes.

Uterine Inversion

Uterine inversion is a rarely encountered condition. It may happen when removal of placenta is attempted by traction on umbilical cord without careful pressure applied to uterus through the abdomen.

Uterus and placenta appear at the introitus. Severe hypotension and bradycardia can occur to the mother due to vasovagal reflex, hemorrhage from placental site and endometrium.

Treatment

To replace the uterus as quickly as possible with steady pressure before the cervix constricts and prevents repositioning of the uterus. Prompt diagnosis and immediate correction will reduce morbidity and mortality of the patient.[22]

- Use of NTG for relaxation of uterus
- GA with endotracheal intubation.

In rare cases laparotomy may be required.

Uterine Atony

Failure of uterus to contract after delivery of baby is common cause of severe postpartum hemorrhage. Risk factors are multiparity; multiple births; polyhydramnios;

large infants; retained placenta; operarive intervention such as internal version and extraction of baby and chrioamnionitis.

Resuscitation of Mother

- Replacement of blood loss initially with crystalloid and colloid solution then with packed red blood cells (PRBC's) as soon as possible
- IV infusion of oxytocin to cause contraction of uterus
- General supportive measures, e.g. oxygen by mask with point in Trendlenberg position
- Close monitoring of vital signs and CVP line if profound hemorrhage.

Treatment

- Oxytocin infusion
- Obstetric maneuvers, e.g. uterine massage, douch with warm water, bimanual compression of uterus, uterine packing, B lynch suturing, internal iliac artery ligation and lastly hysterectomy may be performed to save the mother
- Other pharmacological methods
  - Ergot alkaloids
  - Prostaglandins.

Drugs Used in Treatment of Uterine Atony

Oxytocin: Dose 10 to 40 units in 1000 ml of normal saline or Ringer lactate. It can be given intravenous, intramuscular or intramyometrial routes. It should be given in infusion form. Intravenous bolus can cause decrease in systemic vascular resistance (SVR), mean arterial pressure (MAP), increase in pulmonary vascular resistance (PVR), and severe fall in BP. Therefore dilution is recommended. It also has antidiuretic effect. It rarely causes nausea and vomiting.

Ergot alkaloids: Dose is 0.2 mg IM or IMM (intra-myometrial). Hypertension, vasoconstriction and coronary artery vasospasm are its side effects. Nausea and vomiting is associated with its use.

Prostaglandins: Prostaglandins E and F are used to treat oxytocin resistant uterine atony. Concentrations of endogenous prostaglandins rise during labor and it peaks at the time of placental separation. Failure of such rise in some parturients may lead to uterine atony. Carboprost (15-methyl prostaglandin F_{2alpha}) is preferred drug. Misoprostol is other prostaglandin that can be tried. Side effects associated with use of prostaglandins are malaise, diarrhea, nausea and vomiting and branchospasm.

Dinoprostone: (Prostaglandin E_2) Dose is 20 mg given per rectally. It decreases SVR and MAP. There is no significant change in PVR. It causes bronchodilatation, nausea and vomiting.

Genital Trauma

It can go undetected and can cause hemorrhagic shock, if bleeding goes unnoticed in labor room and patient is shifted to ward. Patient should be resuscitated and transferred to operation theater.

For drainage of vulval hematoma: Local infiltration and IV opioid is administered.

For repair of extensive lacerations and drainage of vaginal hematoma following options can be used:

1. Residual spinal or epidural block.
2. O_2 and N_2O.
3. Pudendal block.
4. Opiates and sedatives.
5. Ketamine can be used.

Retroperitoneal hematoma: It is not common but it is a dangerous condition. Exploratory laparotomy is done. General anesthesia with rapid sequence induction is required.

Detailed description of anesthesia management for these emergency and semiemergency D and E, suction evacuation, cesarean section, laparotomy and laparotomy surgeries can be found in previous chapter.

While administering anesthesia for bleeding obstetric patient, anesthesiologist has to address to following problems:

1. Altered maternal response to hemorrhage
2. Hemorrhagic shock and
3. DIC.

Maternal Response to Hemorrhage

During pregnancy there is physiological increase in blood volume and cardiac output and decrease in systemic vascular resistance resulting from vasodilator effects of pregnancy—hormones and low resistance uteroplacental circulation.[1] Hence pregnant women tolerate blood loss better than nonpregnant patient.

As the gravid uterus receives 12 percent of cardiac output, when hemorrhage occurs it is extremely rapid.[1] Massive bleeding can be dangerous to the mother because of lack of vascular autoregulation. It happens because normal vasoconstrictor response to elevate SVR is impeded by A-V fistula of uteroplacental circulation.

Fetus is at great risk due to maternal hemorrhage. Maternal hypotension reduces uteroplacental blood flow.

Severe anemia reduces oxygen delivery. Hence fetal mortality is very high.

Estimation of Blood Loss

Visual inspection is most often used but is inaccurate. In obstetric patient, part or all of the hemorrhage may be concealed. It is important to realize that in acute hemorrhage, the hematocrit may not reflect actual blood loss. Urine output is one of the most important parameter to monitor in bleeding patient. When carefully measured, rate of urine formation in absence of diuretics, reflects adequacy of renal perfusion and in turn perfusion of vital organs.

Assessment of severity of blood loss (hemorrhagic shock) is shown in Table 15.1.[3]

MANAGEMENT OF HEMORRHAGIC SHOCK IN OBSTETRIC PATIENTS

Pathophysiology

Shock from hemorrhage evolves through several stages. Early in the course of massive bleeding, there are compensatory mechanisms like decrease in mean arterial pressure, stroke volume, cardiac output, central venous pressure and pulmonary capillary wedge pressure. Relative increase in tissue oxygen extraction leads to increase in arteriovenous oxygen content difference. Catecholamine release during hemorrhage leads to generalized increase in venular tone, resulting in an autoransfusion from capacitance vessels. This is accompanied by compensatory increase in heart rate, systemic and pulmonary vascular resistance, and myocardial contractility. There is redistribution of cardiac output and blood volume that results in diminished perfusion to kidneys, splanchnic bed, skin and uterus with relative maintenance of blood flow to heart, brain and adrenal glands.

As blood volume deficit exceeds 25 percent, compensatory mechanisms are usually inadequate to maintain cardiac output and blood pressure. At this point, a small additional loss of blood results in rapid clinical deterioration. Maldistribution of blood flow results in local tissue hypoxia and metabolic acidosis. It produces a vicious cycle of vasoconstriction, organ ischemia, and cellular death. Hemorrhage also activates the CD-18 locus of lymphocytes and monocytes which mediates leukocyte—endothelial cell interactions. There is loss of capillary membrane integrity and additional loss of intravascular volume. There is also increased platelet aggregation in hypovolemic shock, that results in release of vasoactive mediators leading to small vessel occlusion and impaired microcirculation. One must not overlook the importance of extracellular fluid and electrolyte shifts and its role in pathophysiology of hemorrhagic shock.

Table 15.1: Assessment of severity of blood loss (hemorrhagic shock)[3]

Severity of shock	*Symptoms and signs*	*Estimated blood loss*
Grade I/None	Normal pulse and blood pressure	15-20%
Grade II/Mild	Tachycardia (≤100 beats/min) Mild hypotension Peripheral vasoconstriction	20-25%
Grade III/ Moderate	Tachycardia (100-120/min) Hypotension (SBP: 80-100 mm Hg) Restlessness, Oliguria	25-35%
Grade IV/Severe	Tachycardia (>120 beats/min) Hypotension (SBP <60 mm Hg) Altered consciousness Anuria	>35%

Treatment of Shock

A. Provide early diagnosis: Diagnosis is usually self evident, but one must be aware of concealed bleeding. Signs of cardiovascular decompensation may be delayed because pregnant women are usually young and there is pregnancy induced intravascular volume expansion.
   History, examination and investigations will help to identify the etiology of hemorrhage.
B. **Identify and treat the underlying cause**
C. **General principles of management**
   - Resuscitation (Airway, breathing and circulation)
   - Call for help
   - Insert large bore intravenous lines (14G/16G)
   - Send blood samples for investigations: Hb, HCT, BUN, electrolytes, arterial blood gases, coagulation profile
   - Order blood and blood products (hematology consultation)
   - Oxygen by mask at 6-8 lit/min
   - Infuse crystalloid or colloid to maintain normovolemia
   - Use fluid warmers and blood warmers to maintain normothermia
   - Prompt treatment of clotting disorders
   - Consider use of vasopressors
   - Monitoring pulse, BP (noninvasive and/or invasive), oxygen saturation. CVP, urine output, investigations
   - Correction of acid base and electrolyte abnormalities

- Remember that patient may need postoperative ICU care and ventilatory support.

D. **Fluid therapy**
Treatment of serious hemorrhage demands prompt and adequate refilling of intravascular compartment. Initial volume resuscitation is done with crystalloid solution (Ringer lactate or Normal saline) and colloid solution (Hetastarch or polygeline). Do not use Dextran as it can cause allergic reaction and it interferes with blood grouping and cross matching.

E. **Blood and component replacement**
Compatible whole blood is ideal for treatment of hypovolemia from catastrophic hemorrhage. It has shelf life of 40 days and 70 percent of transfused red cells remain viable for at least 24 hours following transfusion. It replaces many coagulation factors, and especially fibrinogen, and its plasma expands the blood volume.
Fractionation of the whole blood makes available specific components—clotting factors and platelets. Component therapy is advisable because it conserves blood resources.

Components

1. Packed red blood cells (PRBC): A unit of packed red cells will raise hematocrit by 3 to 4 volume percent. PRBC and crystalloid infusion are the mainstays of transfusion therapy in obstetric hemorrhage.
2. Platelets: Platelet transfusion is considered in a bleeding patient with platelet count below 50,000 mm^3. 6 to 10 units are usually transfused. Each unit transfused should raise the platelet count by 5000, 10,000/mm^3.
3. Fresh frozen plasma: It is prepared by separating plasma from whole blood and then freezing it. It contains all stable and labile clotting factors, including fibrinogen. It should not be used as a volume expander. It is to be given in obstetric bleeding when fibrinogen level is below 100 mg/dl, and prothrombin time and partial thromboplastin time is abnormal.
4. Cryoprecipitate: It is prepared from fresh frozen plasma. Factor VIII, von Willebrand factor, fibrinogen, factor XIII, and fibronectin. It is indicated in states of general factor deficiency. Volume overload could be a problem. It is useful in specific factor deficiency. A major indication is for severe hypofibrinogenemia due to placental abruption.

F. **Red cells substitutes**
Three varieties are available:
1. Perflurochemicals
2. Liposome encapsulated hemoglobin and
3. Hemoglobin-based oxygen carriers.

Complications of blood transfusion

1. Immunologic—hemolytic transfusion reaction, allergic reaction, fever
2. Infection
3. Hypokalemia, hypocalcemia, hypothermia and citrate intoxication due to storage
4. Hypothermia
5. Metabolic acidosis
6. Volume overload
7. Transfusion related acute lung injury (TRALLI)
8. Dilutional coagulopathy: Replacement of massive blood loss with crystalloid solutions and packed red cells results in depletion of platelets and soluble clotting factors leading to a coagulopathy that clinically is indistinguishable from DIC. Most frequent coagulation defect in obstetric patient with blood loss and multiple units of blood transfusion is thrombocytopenia. Severe hemorrhage without factor replacement can also cause hypofibrinogenemia and prolongation of prothrombin time and partial thromboplastin times. In some cases, shock is associated with consumptive coagulopathy and it is difficult to differentiate between dilutional and consumptive coagulopathy. Fortunately treatment is same in most of the situations.

G. **Systemic hemostatic agents**
1. Aprotinin.
2. Vitamin K.
3. Tranexamic acid.
4. Recombinant Factor VIIa.

MANAGEMENT OF DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

Consumptive Coagulopathy

In 1901, DeLee reported a temporary hemophilia like condition in a woman with placental abruption and another with a long-dead macerated fetus. It was observed that hypofibrinogenemia is associated with placental abruption and other obstetrical accidents. These syndromes are termed as consumptive coagulopathy or DIC.

Pregnancy hypercoagulability- During pregnancy coagulation factors I, VII, VIII, IX, X are increased and

plasminogen levels are also high.There is increased activation of platelets, clotting and fibrinolytic mechanisms. This compensated, accelerated intravascular coagulation may serve for maintenance of the uteroplacental interface.

DIC is always seen as a complication of an identifiable, underlying pathological process and treatment must be directed to reverse the cause and defibrination. Identification and elimination of the source of coagulopathy is the first priority. In addition to bleeding and circulatory obstruction, that can cause ischemia from hypoperfusion, DIC may be associated with microangiopathic hemolysis. This process is likely to contribute to HELLP syndrome.

In obstetrical syndromes involving DIC, the importance of restoration and maintenance of the circulation to treat hypovolemia and persistent intravascular coagulation cannot be overemphasized.

Clinical and Laboratory Findings

Clinical Findings

Excessive bleeding at sites of moderate trauma indicates defective hemostasis. Persistent bleeding from venepuncture sites , trauma from insertion of a urinary catheter, and spontaneous bleeding from gums or nose are signs of coagulation defects. Purpuric areas at pressure sites indicate significant thrombocytopenia. During surgical procedure; oozing from skin, subcutaneous and facial tissues, and retroperitoneal space should suggest coagulopathy.

Laboratory Findings

- Hypofibrinogenemia
- Fibrin and its degradation products in serum
- Thrombocytopenia
- Prolongation of prothrombin time and partial thromboplastin times.

In short, DIC results from abnormal activation of coagulation system, which leads to (1) formation of thrombin, (2) depletion of coagulation factors, (3) activation of fibrinolytic system, and (4) hemorrhage.

Causes of DIC in obstetric patients are:

1. Pre-eclampsia.
2. Placental abruption.
3. Sepsis.
4. Retained dead fetus syndrome.
5. Amniotic fluid embolism.

Etiopathology

Pre-eclampsia: Microangiopathic hemolysis is caused by mechanical disruption of erythrocyte membrane within small vessels in which fibrin is deposited. It is contributory to the HELLP syndrome associated with pre-eclampsia.

Placental abruption: It is one of the most common causes of clinically significant coagulopathy. There is severe hypofibrinogenemia along with elevated levels of fibrinogen-fibrin degradation products, d-dimer, and variable decreases in other coagulation factors. The mechanism is the induction of coagulation intravascularly and to some extent retroplacentally. An important consequence of intravascular coagulation is activation of plasminogen to plasmin, which lyses fibrin microemboli, and maintains patency of microcirculation.

Fetal death: If the delivery of dead fetus is delayed for more than one month, there is disruption of maternal coagulation mechanism. Fibrinogen concentration falls to levels that are normal in nonpregnant state and sometimes to dangerously low levels. Fibrin degradation products are increased and platelet count is decreased. The consumptive coagulopathy is initiated by dead products of conception.

Amniotic fluid embolism: This is a complex disorder characterized by abrupt onset of hypotension , hypoxia, and consumptive coagulopathy. Classically, a woman in late stages of labor or immediately postpartum begins to gasp for air, and then rapidly suffers a seizure or cardiorespiratory arrest which is complicated by DIC, massive hemorrhage, and death. There is great variation in clinical presentations of this condition.

Amniotic fluid embolism was originally described in 1941 by Steiner and Luschbaugh, who found evidence of fetal debris in pulmonary circulation of a group of women dying during labor.

Amniotic fluid enters the circulation as a result of a breach in physiological barrier that normally exists between maternal and fetal compartments. There may be maternal exposure to various fetal elements during pregnancy termination, following amniocentesis, during labor or delivery and cesarean section. In most cases these events are innocuous but in some women it leads to initiation of complex series of physiological reactions likely to be caused by chemokines and cytokines.

After a brief initial phase of pulmonary and systemic hypertension, there is decreased systemic vascular resistance and left ventricular stroke work index (Clark and colleagues, 1988). Transient but profound oxygen desaturation is often seen in the initial phase, resulting in neurological injury in most survivors (Harvey and associates, 1996). In women who live beyond the initial cardiovascular collapse, a secondary phase of lung injury and coagulopathy often ensues. Treatment of this near

fatal condition is cardiopulmonary resuscitation, circulatory support, blood and component therapy.

Sepsis: Infections that lead to sepsis in obstetric patients are mostly due to septic abortion, antepartum pyelonephritis, or puerperal sepsis. Coagulopathy is mediated by disruption of vascular endothelium due to endotoxins. Treatment of inciting cause will be followed by reversal of coagulopathy. In some cases fresh frozen plasma, platelets packs may be useful.

Abortion: Remarkable blood loss may occur as a consequence of abortion. Hemorrhage during early pregnancy is less severe unless abortion is induced and procedure is traumatic. If pregnancy is advanced then hemorrhage occurs due to disruption of a large number of maternal blood vessels at the site of placental implantation. As a consequence of abortion coagulation defects may develop in following circumstances-prolonged retention of dead fetus, sepsis, intrauterine instillation of hypertonic saline or urea, medical induction with prostaglandins, and instrumental termination of pregnancy. Thromboplastin release initiates coagulation within maternal circulation. Consumptive coagulopathy has been a serious complication among women with septic abortion. Management consists of prompt restoration and maintenance of circulation, and appropriate steps to control infection.

Therapeutic goals are to treat or remove the precipitating cause, to stop ongoing proteolytic activity, to replace depleted coagulation factors, provide multi-system support if required.

In obstetric patients, evacuation of uterine contents often removes the precipitating cause.

Considerable controversy exists regarding medical management of patients with DIC.

It varies according to etiology of the disorder.

Treatment includes use of fresh frozen plasma, cryoprecipitate, and platelets.

Use of heparin is controversial.

Antifibrinolytics like epsilon aminocaproic acid and aprotinin are used when other therapies fail and bleeding continues. There has to be documented evidence of raised circulating plasma D-dimer and reduced levels of plasminogen.

Antithrombin III—It is used in moderate to severe DIC associated with reduced antithrombin III levels.

Recombinant factor VIIa (rf VIIa) which is used in variety of patients with bleeding problems can be tried in obstetric patients. It is most useful in hemophilic patients.

Patients with DIC have multiorgan system failure and require ventilator support.

DIC always mandates administration of general anaesthesia for cesarean section.

REFERENCES

1. David CM, Kathelin AS. Antepartum and postpartum haemorrhage. In: Chestnut DH, Polley LS, Tsen LC, Wong CA (Eds). Chestnut's Obstetric anaesthesia Principles and Practice. 4th edn, Philadelphia: MOSBY, Elsevier 2009;811.
2. Liston W. Haemorrhage. In: Lews G (Ed). The confidential enqiry into maternal and child health. Saving mothers lives. Reviewing Maternal deaths to make Motherhood safer-2003-2005. The seventh report of confidential enquiries into maternal deaths in U.K., London (CEMACH) 2007;78-85.
3. Tsen LC. Anaesthesia for obstetric care and gynaecological surgery. In Longnecker DE, Brown DL, Pneuman MS, Zepol WM (Eds). Anaesthesiology. McGrawHill: Newyork 2008;1486-8.
4. Cunnigham SG, Gant NS, Leveno KJ, Gilstrap LC, Hauth J, Katherine NS (Eds). Obstetrical haemorrhage. In Williams Obstetrics. 21st International Edition. USA: McGraw Hill 2003;619-69.
5. Dureja GP. Obstetric Haemorrhage. In: Sunanda Gupta. (Ed). Obstetric anaesthesia. 1st edn, India: Arya Publications; Sirmour HP 2004;297-315.
6. Pisarska MD, Carson SA. Incidence and risk factors for ectopic pregnancy. Clinic Obstet Gynecol 1999;42:2-8.
7. Clayton Hb, Schieve LA, Peterson HB, et al. Ectopic pregnancy risk with assisted reproductive technology procedures, Obstet Gynecol 2006;107:595-604.
8. Levine D. Ectopic pregnancy. Radiology 2007;245:385-97.
9. Medical management of tubal pregnancy. ACOG Practice Bulletin No. 3. Washigton, DC, ACOG 1998.
10. Chantigian RC, Chantigian PDM. Anaesthesia for laparoscopy. In Corfman RS, Diamond MP, DeCherney A (Eds). Complications of Laparoscopy and Hysteroscopy. 2nd edn, Boston, Blackwell Scientific Publications 1997;5-13.
11. American college of Obstetricians and Gynecologists. Diagnosis and treatment of gestational trophoblastic disease. ACOG Practice Bulletin No. 53 Obstet Gynecol 2004;103:1365-77.
12. Berkowitz RS, Goldstein DP. Diagnosis and management of primary hydatidiform mole. Obstet Gynecol clinics of North America 1988;15:491-503.
13. Simpson JL, Jauniaux ERM. Pregnancy loss. In Gabby SG, Niebyl JR, Simpson JL (Eds). Obstetrics: Normal and Problem pregnancies. 5th edn Philadelphia, Churchil Livingstone 2005;629.
14. Cunnigham FG, Leveno KJ, Bloom SL, et al. Willams Obstetrics. 22nd edn, New York, McGraw- Hill 2005;231-51.
15. Silver R, Depp R, Sabbagha RE, et al. Placenta previa: Aggressive expectant management. Am J Obstet Gynecol 1984;150:15-22.
16. Sharma A, Suri V, Gupta I. Tocolytic therapy in conservative management of symptomatic placenta previa. Int J gynecol Obstet 2004;84:109-13.

17. Oyelese Y, Ananth CV. Placental abruption. Obstet Gynecol 2006;108: 1005-16.
18. Kaczmarczyk M, Sparen P, Terry P, Cnattingius S. Risk factors for uterine rupture and neonatal consequences of uterine rupture; a population based study of successive pregnancies in Sweeden. British J Obstet Gynecol 2007;114:1208-14.
19. Walsh CA, Baxi LV. Rupture of the primigravid uterus: A review of the literature. Obstet Gynecol Surv 2007;62:327-34.
20. Oyelese Y, Smulian JC. Placenta previa, placenta accreta and vasa previa. Obstet Gynecol 2006;107:927-41.
21. Catanzarite V, Maida C, Thomas W, et al. Prenatal sonographic diagnosis of vasa previa: ultrasound findings and obstetric outcome in ten cases. Ultrasound Obstet Gynecol 2001;18:109-15.
22. You WB, Zahn CM. Postpartum haemorrhage: abnormally adherent placenta, uterine inversion, and puerperal hematomas. Clin Obstet Gynecol 2006;49:184-97.
23. DeSimone CA, Noris MC, Leighton BL. Intravenous nitroglycerine aids manual extraction of retained placenta. Anaesthesiology 1990;73:787.
24. Bullarbo M, Tjugum J, Ekerhovd E. Sublingual nitroglycerin for management of retained placenta. Int J Gynaecol Obstet 2005;91:228-32.
25. Crochetiere C. Obstetric emergencies. Anaesthesiology Clinics of North America 2003;21:111-15.

16 Anesthesia for Pregnant Patient with Medical Disorders

Shantanu B Kulkarni, Preeti Rustagi, Smita S Lele, Manju N Gandhi

KEY POINTS

- The presence of shunt flow between the right and left heart requires meticulous exclusion of air bubbles or clots from intravenous fluids to prevent paradoxical embolism into the cerebral or coronary circulation.
- The clinical manifestations of Eisenmenger syndrome include the features of arterial hypoxemia and right ventricular failure (e.g. dyspnea, clubbing of the nails, polycythemia, engorged neck veins and peripheral edema).
- In aortic stenosis, it is important to maintain a normal heart rate and sinus rhythm as these patients have a low fixed stroke volume and a slow heart rate decreases cardiac output.
- Aortic regurgitation results in left ventricular volume overload, which over time leads to left ventricular dilatation and hypertrophy.
- The increased heart rate of pregnancy limits the time available for filling of the left ventricle and results in increased left atrial and pulmonary arterial pressures and an increased likelihood of pulmonary edema in patient with mitral stenosis.
- Pregnant patient with mitral regurgitation usually tolerate the increased blood volume and heart rate of pregnancy, provided sinus rhythm is maintained.
- By avoiding instrumentation of the hypereactive airway, regional anesthesia is preferred to general anesthesia in asthmatic patients.
- The cardiac output does not rise till hemoglobin concentrations decrease to approximately 7 g/dl. In anemic parturient when blood transfusion should be considered.
- Pregnancy usually is characterized by an increase in sickle crises and its complications like pneumonia, pyelonephritis, pulmonary emboli, congestive heart failure and by obstetric complications, e.g. IUGR (intra-uterine growth retardation), preterm birth, pre-eclampsia.
- Large breasts, increased anteroposterior diameter of the chest, airway edema, and decreased chin-to-chest distance, left lateral position, all increase the incidence of difficult laryngoscopy and failed intubation in obese pregnant patient.
- Drugs that stimulate the sympathetic nervous system should be avoided in the hyperthyroid patient (i.e. ketamine, pancuronium, atropine, ephedrine, epinephrine).
- Hypothyroidism is associated with an increased incidence of the following obstetric complications: anemia, pre-eclampsia, IUGR, placental abruption, postpartum hemorrhage, and fetal distress during labor.
- The diabetic parturient should be evaluated for adequate temporomandibular joint and cervical spine mobility to anticipate difficult intubation.
- Factors that reduce hepatic blood flow such as hypotension, excessive sympathetic activation and high mean airway pressure during controlled ventilation should be avoided in hepatic diseases.
- Atracurium and cisatracurium undergo Hoffmann degradation and nonspecific esterase hydrolysis, hence are safely used in renal failure.
- There was no evidence of accelerated disease progression or increased infectious or neurologic complications after regional anesthesia in HIV-positive parturient.

INTRODUCTION

We are often called by our obstetric colleagues to give anesthesia to pregnant ladies who may have a cardiac murmur and dyspnea, wheezing, uncontrolled blood pressure and seizures, very high blood sugars, severe anemia, neck swelling, yellowish discoloration of skin and conjunctiva, facial puffiness and low urine output, or is HIV-positive. To optimize these patients, and give relevant anesthesia is very important to both the mother and the newborn. This chapter will describe the management of such patients for emergency cesarean section.

CARDIOVASCULAR DISEASES

The pregnant patients coming for emergency cesarean section have cardiac problems like mitral valve stenosis, mitral valve regurgitation, aortic stenosis, atrial septal defect and so on. We will describe the various congenital and valvular cardiac defects and their anesthetic management.

Congenital Heart Disease

The congenital heart diseases are atrial septal defect, ventricular septal defect, patent ductus arteriosus, tetralogy of Fallot and transposition of great vessels. Improvement in the diagnosis and treatment have led to an increase in the number of women who survive to childbearing age. These women may be asymptomatic with relatively normal intracardiac pressures and blood flow patterns. Such patients usually require two considerations.

- Antibiotic prophylaxis.
- Neonatologist at delivery (there is increased incidence of congenital cardiac lesions in babies of these women).

Some pregnant women have an uncorrected or a partially corrected defect. The anesthetic management of these patients is more challenging.

Left to Right Shunts

Small atrial septal defect (ASD), ventricular septal defect (VSD), or patent ductus arteriosus (PDA) produce a modest degree of left-to-right intracardiac shunting, which commonly is well-tolerated during pregnancy.[1]

Anesthetic management of these patients should include attention to the following detail:

- There should be meticulous exclusion of air bubbles or clots from intravenous fluids to prevent paradoxical embolism into the cerebral or coronary circulation
- If epidural anesthesia is used, the anesthesiologist should use a loss-of-resistance to saline rather than air to identify the epidural space as epidural injection of air can cause systemic embolization
- Labor pain causes increased maternal concentrations of catecholamines and increased maternal systemic vascular resistance (SVR). This increases the severity of left-to-right shunt resulting in pulmonary hypertension and right ventricular failure. Epidural anesthesia causes a pain-free labor and prevents the increase in maternal catecholamines level and SVR
- Epidural anesthesia is preferred over spinal anesthesia as a rapid decrease in SVR could result in a reversal of shunt flow (asymptomatic left-to-right shunt may become a right-to-left shunt with maternal hypoxemia)
- The patient should receive supplemental oxygen, and monitor the hemoglobin oxygen saturation.
- Even mild hypoxemia can result in increased pulmonary vascular resistance and reversal of shunt flow. It is important to avoid hypercarbia and acidosis, which increases pulmonary vascular resistance.

Coarctation of the Aorta (COA)

Patients who have undergone corrective surgery and who have normal arm and leg blood pressures do not require special precautions or monitoring. An arm-to-leg pressure gradient of less than 20 mm Hg is associated with a good pregnancy outcome. Pregnant women with uncorrected coarctation are at high-risk for left ventricular failure, aortic rupture or dissection, and endocarditis. In such pregnancy, the fetal mortality rate increases because of decreased uterine perfusion distal to the aortic lesion.

Patients with aortic coarctation are more likely to have a bicuspid aortic valve (hence the increased risk of endocarditis) or an aneurysm in the circle of Willis. Thus, these patients are at an increased risk of a cerebrovascular accident.

Physical examination includes comparison of the right versus left-sided blood pressures and upper versus lower extremity pressures. The electrocardiogram (ECG) shows left ventricular hypertrophy.

The pathophysiologic manifestations include a fixed obstruction to aortic outflow and distal hypoperfusion. The increased cardiovascular demands of pregnancy exacerbate the pathophysiology of this lesion. The blood pressure gradient of more than 50 mm Hg between upper arm and lower limb is considered high-risk for mother and fetus.

Anesthetic Consideration

- Attention should be toward maintaining normal to slightly elevated SVR, a normal to slightly increased heart rate, and adequate intravascular volume
- In patients with uncorrected coarctation, neuraxial anesthesia should be avoided
- It is advisable to give general anesthesia for LSCS (lower segment cesarean section) since it is low fixed cardiac output state[2]
- Fentanyl helps maintain hemodynamic stability
- Invasive hemodynamic monitoring is used for the administration of intravenous fluids. Uterine perfusion pressure is shown accurately by using a postductal intra-arterial catheter instead of a preductal catheter
- If there is no time for invasive monitoring, the blood pressure should be recorded in upper arm and lower limb
- Ephedrine and dopamine are the vasopressors of choice because of their mild positive chronotropic effects for the managing hypotension
- If anesthetic technique involves the use of high-dose fentanyl, the naloxone should be readily available for the management of neonatal respiratory depression.

Right-to-Left Shunts

Tetralogy of Fallot (TOF)

This lesion includes four components: (1) VSD, (2) right ventricular hypertrophy, (3) pulmonic stenosis with right ventricular outflow tract obstruction, and (4) an overriding of aorta (i.e. the aortic outflow tract receives blood from both the right and left ventricles). Patients typically present with cyanosis.

The cardiovascular changes of pregnancy (e.g. increased blood volume, increased cardiac output and decreased SVR) may unmask these previously asymptomatic patients. The severity of symptoms depends on the size of the VSD, the magnitude of the pulmonic stenosis, and the contractility of the right ventricle. Patients with tetralogy of Fallot, should undergo echocardiography before and during early pregnancy.

Most pregnant women with TOF have had corrective surgery. The surgical treatment involves closure of the VSD and widening of the pulmonary outflow tract. In some cases, a small VSD may recur, or progressive hypertrophy of the pulmonary outflow tract may occur slowly.

Anesthetic Consideration

- Patients with successful correction of TOF are similar to normal woman
- Patients with corrected TOF can manifest various atrial and ventricular arrhythmias, owing to surgical injury to the cardiac conduction channels. Thus, a 12-lead ECG and ECG monitoring during cesarean section are desirable
- In a parturient with uncorrected or corrected TOF with residua, the anesthesiologist should avoid technique which decreases SVR, which increases the severity of right to left shunt[3]
- It is important to maintain adequate intravascular volume and venous return
- In the presence of right ventricular compromise, high filling pressures are needed to increase right ventricular outflow and adequate pulmonary blood flow
- Administration of an epidural block during labor is advisable and helps prevent an increase in pulmonary vascular resistance and consequent right to left shunting
- For cesarean section, epidural anesthesia is preferred, as the fall of BP is gradual.

Eisenmenger Syndrome

A chronic, uncorrected left-to-right shunt may produce right ventricular hypertrophy, raised pulmonary artery pressures, right ventricular dysfunction (this syndrome was first described by Eisenmenger in 1897). The primary lesion can be either an ASD, VSD, PDA or truncus arteriosus. The pulmonary and right ventricular musculature undergoes remodeling in response to chronic pulmonary volume overload. The high, fixed pulmonary arterial pressure gradually limits flow through the pulmonary vessels. A reversal of shunt flow occurs when pulmonary artery pressure exceeds the systemic blood pressure. The primary left-to-right shunt becomes a right-to-left shunt. Initially the shunt may be bidirectional; acute changes in pulmonary vascular resistance or SVR may influence the primary direction of intracardiac blood flow. However, the pulmonary vascular occlusive disease ultimately leads to irreversible pulmonary hypertension. The correction of the primary intracardiac lesion is not helpful at this stage.

The clinical manifestations of Eisenmenger syndrome include the features of arterial hypoxemia and right ventricular failure (e.g. dyspnea, clubbing of the nails, polycythemia, engorged neck veins and peripheral edema).

These pregnant women are unable to increase the oxygen demand required during pregnancy. Maintenance of adequate oxygenation requires adequate pulmonary blood flow. The decrease in pulmonary vascular resistance seen in normal pregnancy does not occur in these women as the pulmonary vascular resistance is irreversible. The decrease in SVR associated with pregnancy tends to exacerbate the severity of the right-to-left shunt.[4] The pregnancy-associated decrease in functional residual capacity also can predispose the patient to maternal hypoxemia. Maternal hypoxemia results in decreased oxygen delivery to the fetus, which results in a high incidence of IUGR and fetal death. The thromboembolic phenomenon are responsible for about 43 percent of all maternal deaths in patients with Eisenmenger syndrome. Many of these deaths occur postpartum (4 to 6 weeks after delivery).

The goals of anesthetic management are as follows:

- Maintain adequate SVR
- Maintain intravascular volume and venous return. Avoid aortocaval compression
- Prevention of pain, hypoxemia, hypercarbia, and acidosis, which may cause an increase in pulmonary vascular resistance
- Avoid myocardial depression during general anesthesia
- Supplemental oxygen should be given. The pulse oximeter is used to detect acute changes in shunt flow.

An intra-arterial catheter helps in rapid detection of sudden changes in blood pressure, and a central venous pressure (CVP) catheter can detect clinically significant changes in cardiac filling pressures. CVP catheter occasionally produces complications (e.g. air emboli, infection, hematoma, pneumothorax), which can be harmful in these patients.

Pulmonary artery catheter may be relatively contraindicated for following reasons:

1. It is difficult to properly position the balloon-tipped, flow-directed catheter within the pulmonary artery.
2. If the catheter does go into the pulmonary artery, the risks of pulmonary artery rupture and hemorrhage are great.
3. These patients may not tolerate catheter-induced arrhythmias.
4. Measurements of cardiac output by thermodilution method are unpredictable in the presence of a large intracardiac shunt.
5. Pulmonary artery pressure monitoring rarely gives clinically useful information in the presence of severe, fixed pulmonary hypertension.
6. The pulmonary artery catheter may predispose to pulmonary thromboembolism.

Anesthetic Consideration

- Effective labor analgesia is necessary to prevent labor-induced increase in plasma catecholamines, which can further increase pulmonary vascular resistance.
- During the first stage of labor, intrathecal opioid administration is ideal, as it produces profound analgesia with minimal sympathetic blockade.
- For the second stage of labor, epidural or intrathecal doses of a local anesthetic and an opioid will provide satisfactory analgesia; alternatively, a pudendal block can be given early in the second stage of labor.
- Maternal anticoagulation may contraindicate the use of regional anesthetic techniques. Historically, anesthesiologists have avoided regional anesthesia for LSCS because the vasodilatation that accompanies sympathectomy can worsen a right-to-left shunt.
- However, good results have been achieved with epidural anesthesia, which has become the technique of choice. It is important to avoid aortocaval compression and maintain adequate venous return. Intravenous crystalloid and small doses of phenylephrine are needed to maintain maternal preload, SVR, and oxygen saturation.

Disadvantages Associated with General Anesthesia

Positive-pressure ventilation results in decreased venous return, which compromises cardiac output. The volatile halogenated agents can cause myocardial depression and decreased SVR and uterine atony may lead to postpartum hemorrhage. Rapid-sequence induction with thiopentone or propofol characteristically decreases both cardiac contractility and SVR, which can exacerbate a right to left shunt. Hence, it should be avoided. The risk of anesthesia-related aspiration can be prevented by IV Ranitidine 50 mg, IV metoclopramide 10 mg, cricoid pressure. Intravenous opioid (fentanyl) helps maintain hemodynamic stability, however one should be prepared to manage respiratory depression in newborn.

Regardless of the anesthetic technique used, these women are at high-risk for hemodynamic compromise immediately after delivery. Blood loss should be replaced adequately with crystalloid, colloid or blood transfusion. Cautious fluid therapy is important when the blood loss is minimal, because the postpartum autotransfusion can cause intravascular volume overload with consequent myocardial dysfunction and pulmonary edema.

Infective Endocarditis

It is the invasion and colonization of the cardiac valves, endocardium, and prosthetic cardiac tissue by an infectious pathogen. Colonization results in the development of friable vegetations, which can produce emboli, hemodynamic compromise, and a fulminant clinical course. Infective endocarditis usually occurs in association with a pre-existing cardiac lesion (which provides a roughened surface for bacterial growth). The risk factors include dental and urologic procedures, renal dialysis, prolonged intravenous therapy, and intravenous drug abuse.

Pathogens reported during pregnancy include *Streptococcus viridians* (74%), group B streptococcus, *Staphylococcus aureus, Neisseria gonorrhoeae, Chlamydia trachomati,* enterococcus and *Salmonella enteritidis.* Women who are immune-deficient (e.g. AIDS) are susceptible to uncommon and less-virulent pathogens.

Infective endocarditis is divided into two groups: subacute and acute.

Subacute Endocarditis

It is characterized by an insidious onset of fever, weakness, malaise, and unexplained embolic phenomena. Blood cultures are positive in 90 percent cases. Murmurs usually result from the underlying cardiac lesion. Systemic embolism can develop at any time, giving rise to splinter hemorrhages and mucosal petechiae. Septic abscesses can lead to atrioventricular nodal dysfunction, conduction block, and arrhythmias. Other manifestations include splenomegaly and immune complex nephritis due to antigen-antibody complex deposition on the glomerular basement membrane with delayed renal failure. The major causes of death include congestive heart failure, embolic cerebral infarction, arrhythmias, and renal failure.

Acute Infective Endocarditis

It has an acute onset of symptoms like high grade fever, shaking chills, and early onset of embolic phenomena. Skin and mucosal petechiae can occur. Murmurs occur in two-thirds of patients with vegetations on the left side of the heart. Cardiac decompensation appears early. Cardiovascular function may worsen suddenly with erosion of a valve or rupture of the chordae tendinae. Aortic ring abscesses may produce conduction disturbances or ventricular septal defects. The ECG may be normal or may show arrhythmias or conduction disturbances. Echocardiography helps localize valvular vegetations, show valvular incompetence or left ventricular failure. The major cause of death is due to congestive heart failure, arrhythmias, uncontrolled sepsis, septic emboli, and mycotic aneurysm formation with rupture.

Patients should receive parenteral antibiotic therapy according to culture and sensitivity. Complications such as congestive heart failure and arrhythmias are managed accordingly.

Anesthetic Consideration

- Anesthetic management depends on the clinical presentation
- Patients with evidence of cardiac decompensation require invasive haemodynamic monitoring and general anesthesia
- Avoidance of regional anesthesia in patients with sepsis or acute infective endocarditis is advocated
- However, in a patient with a recent history of infective endocarditis, administration of regional anesthesia can be considered, if the patient is afebrile, culture test is negative, and hemodynamically stable.

Antibiotic prophylaxis: Patients at high-risk for endocarditis (e.g. surgically constructed systemic-pulmonary shunts or conduits, prosthetic valves, rheumatic heart disease, history of endocarditis) should receive antibiotic prophylaxis before LSCS. Prophylaxis typically consists of intravenous ampicillin, amoxicillin and gentamicin. Vancomycin may be substituted for penicillin in patients who are allergic to penicillin.

Valvular Heart Diseases

These include Mitral stenosis, Mitral regurgitation, Aortic stenosis, Aortic regurgitation.

Aortic Stenosis (AS)

In women of childbearing age, valvular aortic stenosis is usually rheumatic in origin. Subvalvular and supravalvular aortic stenosis are usually congenital.

AS does not become hemodynamically significant until the valve diameter is one third of its normal size (The normal aortic valve area is 2.6 to 3.5 cm^2). A valvular gradient of 50 mm Hg signals severe stenosis. Patients with a gradient that exceeds 100 mm Hg are at high-risk for myocardial ischemia. Patients with moderate-to-severe AS have a relatively fixed stroke volume and they are unable to maintain adequate coronary or cerebral perfusion during exertion. The onset of angina, dyspnea, or syncope indicates poor prognosis.

A coarse systolic murmur, which reaches its maximal intensity at midsystole and radiates to the apex and the neck, is characteristic of aortic stenosis. The

ECG may show evidence of left ventricular hypertrophy, conduction disturbances, and ischemia. In patients with severe aortic stenosis, chest X-ray may show left ventricular enlargement, aortic valve calcification, and poststenotic dilatation of the ascending aorta. Echocardiography is preferred for monitoring the cardiac status of parturients throughout the labor and delivery.

Interaction with pregnancy

The increased blood volume of pregnancy allows women with mild aortic stenosis to tolerate pregnancy well. Women with severe aortic stenosis have limited ability to compensate for increased demands during pregnancy. They may develop dyspnea, angina, or syncope. The incidence of perinatal fetal loss is increased in these cases.

Anesthetic consideration

The goals of anesthetic management are as follows:

- Maintain a normal heart rate and sinus rhythm
- Maintain adequate SVR
- Maintain intravascular volume and venous return. Avoid aortocaval compression
- Avoid myocardial depression during general anesthesia
- In AS, it is important to maintain a normal heart rate and sinus rhythm as these patients have a low fixed stroke volume. A slow heart rate decreases cardiac output. Severe tachycardia increases myocardial oxygen demand and decreases time for diastolic perfusion of the hypertrophic left ventricle. Patients with aortic stenosis do not tolerate arrhythmias well. Left atrial systole is important for the maintenance of adequate ventricular filling and cardiac output. Prompt treatment of arrhythmias is essential during the management of these patients
- Patients with aortic stenosis do not tolerate hypotension as it causes significant decrease in SVR and decreased perfusion of the hypertrophic left ventricle. Normal patients compensate for decreased SVR by increasing stroke volume and heart rate. Patients with aortic stenosis have a low fixed stroke volume, hence they must increase their heart rate to increase cardiac output. However, severe tachycardia decreases coronary blood flow and is detrimental to coronary perfusion
- Patients with aortic stenosis do not tolerate decreased venous return and left ventricular filling pressure. Adequate end-diastolic volume is necessary to maintain left ventricular stroke volume
- Monitoring is done with intra-arterial line and CVP or pulmonary artery catheter (care is taken to prevent arrhythmias). The left ventricle is relatively noncompliant, and the anesthesiologist may observe an increased pulmonary capillary wedge pressure (PCWP) with a normal left ventricular end-diastolic volume (LVEDV)
- During labor, hypovolemia is a greater threat than pulmonary edema. Thus, CVP or PCWP should be maintained at high-normal levels (e.g. PCWP of 18 mm Hg) to protect cardiac output during unexpected peripartum hemorrhage
- Avoid spinal and epidural anesthesia in pregnant women with aortic stenosis. Moderate-to-severe aortic stenosis remains a relative contraindication for single-shot spinal anesthesia. However, many reports have described the safe use of continuous epidural for LSCS in women with aortic stenosis.[5,6] Do not use local anesthetic solutions with epinephrine for patients with moderate-to-severe aortic stenosis. An unintentional intravascular injection of epinephrine can precipitate tachycardia, whereas systemic absorption of epinephrine from the epidural space can decrease SVR and lower venous return. It is essential to maintain left uterine displacement during the induction and maintenance of anesthesia. Maintenance of venous return and LVEDV are important
- For general anesthesia, a combination of etomidate and a modest dose of opioid is a good choice for the induction of anesthesia and is generally preferable to sodium thiopentone (which causes myocardial depression) and ketamine (which causes tachycardia). A combination of low-dose thiopentone and ketamine can be used if etomidate is unavailable.

Aortic Regurgitation (AR)

Rheumatic heart disease is the cause in almost 75 percent of affected patients. Women with rheumatic aortic regurgitation usually have mitral valve disease. Fifteen percent patients with VSD develop prolapse of an aortic cusp and **chronic aortic regurgitation**. Congenital fenestrations of the aortic valve occasionally produce mild aortic regurgitation. Syphilis or ankylosing spondylitis are associated with AR. Cystic medial necrosis of the ascending aorta, idiopathic dilatation of the aorta, and severe hypertension may widen the aortic annulus and lead to progressive aortic regurgitation.

There is regurgitation of blood from the aorta to the left ventricle when the aortic valve fails to close normally. Aortic regurgitation results in left ventricular volume overload, which over a time period leads to left ventricular dilatation and hypertrophy. Initially, the

enlarging left ventricle tolerates the increased work. Gradually left ventricular contractility decreases, the ejection fraction (forward stroke volume) progressively decrease, and LVEDV (left ventricular end diastolic volume) continues to rise. Deterioration of left ventricular function precedes the development of symptoms.[7] A competent mitral valve can protect the pulmonary circulation from the initial increases in LVEDV and LVEDP (left ventricular end diastolic pressure). However, as the ventricle begins to fail, further increases in LVEDV and LVEDP occur, which leads to pulmonary edema.

Equilibration between aortic and left ventricular pressures may occur toward the end of diastole, particularly when the heart rate is slow. The LVEDP may increase to very high levels (greater than 40 mm Hg). Rarely the left ventricular pressure exceeds the left atrial pressure toward the end of diastole. This may cause premature closure of the mitral valve or diastolic mitral regurgitation.

Myocardial ischemia occurs in patients with aortic regurgitation because left ventricular dilatation and increased left ventricular systolic pressure result in increased myocardial oxygen demand, in addition decreased coronary flow during diastole results in decreased myocardial perfusion.

In **aortic regurgitation,** the first complaint often is a pounding sensation in the chest, especially when the patient is lying down. Exertional dyspnea is the first symptom of diminished cardiac reserve. This is followed by orthopnea, paroxysmal nocturnal dyspnea, and diaphoresis. Symptoms of left ventricular failure are more common than symptoms of myocardial ischemia. Late in the course of the disease, peripheral edema, congestive hepatomegaly, and ascites can develop.

The arterial pulse pressure is widened. The patients characteristically have a rising water hammer pulse. A diastolic thrill is palpable along the left sternal border, and a third heart sound is common. The murmur of AR typically is a high-pitched, blowing decrescendo diastolic murmur that is heard best along the left sternal border in the third intercostal space. The ECG may show left ventricular hypertrophy and myocardial ischemia in women with severe disease. The presence of atrial fibrillation is suggestive of coexisting mitral valve disease.

The left ventricular failure of chronic aortic regurgitation initially responds to treatment with digoxin, salt restriction, and diuretics. Cardiac arrhythmias and infection are poorly tolerated and require prompt therapy.

Aortic regurgitation usually is well-tolerated during pregnancy for three reasons:

- Pregnancy results in a modest increase in maternal heart rate, and this decreases the time for regurgitant blood flow during diastole
- Pregnancy results in decreased SVR, which favors the forward flow of blood and decreases the amount of regurgitant blood flow
- The increased blood volume of pregnancy helps maintain adequate filling pressures.

Anesthetic consideration

The goals of anesthetic management are as follows:

- Maintain a heart rate that is normal or slightly increased
- Prevent an increase in SVR
- Avoid aortocaval compression
- Avoid myocardial depression during general anesthesia
- Epidural anesthesia may result in decreased afterload and is preferred for cesarean delivery. During labor, epidural anesthesia prevents the pain-associated increase in SVR, which can precipitate acute left ventricular volume overload in women with aortic regurgitation. These patients do not tolerate bradycardia, which should be treated promptly.

Mitral Stenosis

Mitral stenosis is the most common lesion associated with rheumatic heart disease.

The rheumatic mitral valve is fused along the edges of the cusps or the chordae tendinae. The valve thickens and becomes funnel-shaped or fish-mouthed.

The normal mitral valve orifice has a surface area of 4 to 6 cm^2. Symptoms typically develop when the size of the orifice is 2 cm^2 or less. A reduction to 1 cm^2 or less is considered severe and often requires surgical intervention.

Mitral stenosis prevents filling of the left ventricle, which results in decreased stroke volume and decreased cardiac output. Mitral stenosis prevents emptying of the left atrium. This results in left atrial dilatation and increased left atrial and pulmonary arterial pressures. Atrial fibrillation can occur and mural thrombi may develop. Increased pulmonary arterial pressure results in dyspnea, hemoptysis, and pulmonary edema.[8]

Progressive pulmonary hypertension results in compensatory right ventricular hypertrophy. Increased pulmonary vascular resistance worsens with exercise

and may lead to right heart failure. Severe, fixed pulmonary hypertension limits the compensatory changes in pulmonary vascular resistance that normally accompany changes in cardiac output and SVR.

Approximately 25 percent women with mitral stenosis first experience symptoms during pregnancy. Symptoms and signs of mitral stenosis include dyspnea, hemoptysis, chest pain, right heart failure, and thromboembolism. Auscultation may reveal a diastolic murmur, an accentuated S1, an audible S4, and an opening snap. The ECG may show left atrial enlargement, atrial fibrillation, and right ventricular hypertrophy. Echocardiography helps confirm the diagnosis, although careful measurements are important because mitral valve-area calculation by 2-D echocardiography may be inaccurate during pregnancy.

Women with severe mitral stenosis do not tolerate the cardiovascular demands of pregnancy. Mitral stenosis limits the patient's ability to increase cardiac output during pregnancy. The increased heart rate of pregnancy limits the time available for filling of the left ventricle and results in increased left atrial and pulmonary arterial pressures and an increased likelihood of pulmonary edema in mitral stenosis. Atrial fibrillation is associated with an increased maternal morbidity and mortality in women with mitral stenosis. Both the loss of atrial systole and the increased ventricular rate result in decreased cardiac output and an increased risk of pulmonary edema. Approximately 80 percent of cases of systemic emboli occur in patients with atrial fibrillation.

Women with mitral stenosis who are asymptomatic before pregnancy usually tolerate pregnancy well. Women with previous pulmonary congestion have an increased incidence of pulmonary edema and mortality during or after pregnancy. The risk of maternal death is greatest during labor and during the postpartum period. The sudden increase in preload immediately after delivery may flood the central circulation and result in the development of severe pulmonary edema. However, hypovolemia and a sudden decrease in venous return, which can occur with hemorrhage, should be avoided.

Beta receptor blockade is useful to prevent tachycardia during pregnancy.

Atrial fibrillation requires aggressive treatment with digoxin and beta blockers. If pharmacologic therapy fails to control the ventricular response, cardioversion should be performed. After cardioversion, pulmonary edema typically responds well to bed rest in the left lateral decubitus position and to administration of a diuretic.

Pregnancy usually is well tolerated by women with mild mitral stenosis. However, in women with moderate to severe stenosis (valve area <1.5 cm^2), pregnancy worsens the functional NYHA (New York Heart Association) status by an average of 1 or 2 classes. Parturients with symptomatic mitral stenosis require invasive hemodynamic monitoring during labor or LSCS.

Anesthetic Consideration

The goals of anesthetic management are as follows:

- Maintain a slow heart rate
- Maintain a sinus rhythm, if present. Aggressively treat acute atrial fibrillation
- Avoid aortocaval compression. Maintain venous return and PCWP to maximize LVEDV without causing pulmonary edema
- Maintain adequate SVR
- Prevent pain, hypoxemia, hypercarbia, and acidosis, which may increase pulmonary vascular resistance.
- A slow heart rate allows increased diastolic filling time through the fixed, obstructed mitral valve. It is important to prevent a significant fall in SVR, because of the patient's limited ability to increase cardiac output to maintain perfusion pressure.[9] Give supplemental oxygen and monitor oxygen saturation with a pulse oximeter
- Intrathecal opioid provides excellent analgesia during the first stage of labor without causing sympathetic blockade. Epidural anesthesia is the most reliable method for providing anesthesia during the second stage of labor analgesia
- An alternative approach is to use a CSE (Combined Spinal Epidural) technique. The anesthesiologist can give intrathecal opioid with a small dose of bupivacaine, followed by slow epidural administration of a dilute solution of local anesthetic, with an opioid (e.g. 0.125% bupivacaine with fentanyl 2 mcg/ml). The epidural catheter facilitates the provision of analgesia during the second stage of labor. Phenylephrine rather than ephedrine is the preferred vasopressor in patients with mitral stenosis. Small bolus doses of phenylephrine help maintain SVR and do not cause maternal tachycardia
- Epidural anesthesia is preferred for cesarean section. Invasive hemodynamic monitoring, judicious intravenous crystalloid administration, slow induction of anesthesia, and administration of small bolus doses of phenylephrine help maintain maternal hemodynamic stability
- If general anesthesia is required, do not use drugs that produce tachycardia (e.g., atropine, ketamine, pancuronium, meperidine). A beta-blocker and a

modest dose of opioid should be administered before the induction of general anesthesia. Esmolol is a good choice in these patients; it has both a rapid onset and a brief duration of action. However, esmolol may cause adverse fetal effects. The fetal heart rate (FHR) should be monitored until delivery when possible

- After delivery, the bolus administration of either oxytocin, methylergonovine, or 15-methyl prostaglandin $F_{2\text{-alpha}}$ can result in increased pulmonary vascular resistance. The patient is at risk of hemodynamic compromise and pulmonary edema during the postpartum period. Some obstetrician routinely practice the administration of the IV frusemide following the delivery of baby in order to prevent volume overload from contracting uterus. Cesarean section does not eliminate the haemodynamic stress of the puerperium, except that the greater blood loss during cesarean section may be beneficial for women with mitral stenosis. These patients require intensive care in postpartum period. Postoperatively patient may need ventilator and hemodynamic support for few hours.

Mitral Regurgitation

The common causes of mitral regurgitation are rheumatic heart disease, myxomatous degeneration, ischemic papillary muscle disease, and endocarditis.

The factors that affect atrial and ventricular enlargement include the severity of the systolic regurgitant flow and the duration of mitral regurgitation. Chronic mitral regurgitation causes back flow of blood in the left atrium during systole. The left atrium accommodates the regurgitant blood flow by gradual dilatation and increased compliance of left ventricle.[10] The left atrial dilatation predisposes to atrial fibrillation. The onset of atrial fibrillation may produce palpitations. However, patients with mitral regurgitation withstand atrial fibrillation better than patients with mitral stenosis as there is no obstruction to diastolic blood flow. Pulmonary hypertension is less common in chronic mitral regurgitation than in patients with mitral stenosis. Typically, there is only a modest increase in left atrial pressure. Severe, long-standing mitral regurgitation results in increased left atrial pressure and pulmonary congestion.

With severe, acute mitral regurgitation, the pulmonary artery catheter tracing shows a V wave with a wide pulse pressure.

Symptoms of chronic mitral regurgitation include chronic weakness and fatigue secondary to a low cardiac output. Physical examination reveals a pansystolic murmur, an accentuated pulmonary component of the second heart sound and in severe cases, an S3 gallop rhythm. ECG findings include left ventricular hypertrophy and atrial arrhythmias. The ECG may demonstrate atrial fibrillation. Left ventricular dilatation and hypertrophy is more common in chronic mitral regurgitation than in patients with acute mitral regurgitation. Prominent atrial waves reflect atrial hypertrophy. Chest X-ray may show a moderately enlarged heart and marked left atrial enlargement.

Interaction with pregnancy

Mitral regurgitation usually is well tolerated during pregnancy. Pregnant women with mitral regurgitation usually tolerate the increased blood volume and heart rate of pregnancy, especially if sinus rhythm is maintained.

There is an increased risk of atrial fibrillation. Some physicians recommend prophylactic digoxin therapy to decrease the risk of a rapid ventricular response, if atrial fibrillation should occur.

The hypercoagulability of pregnancy increases the risk of systemic embolization. Anticoagulation may be indicated if (1) cardioversion is planned, (2) there is a history of embolic phenomena, or (3) a new onset of atrial fibrillation occurs.

During labor, SVR is increased by pain, expulsive efforts (e.g. Valsalva maneuver), and aortic compression by the uterus. Increased SVR is poorly tolerated by parturients with regurgitant valvular lesions.

These women are at increased risk for infective endocarditis, and antibiotic prophylaxis is indicated.

Anesthetic Consideration

The goals of anesthetic management are as follows:

- Prevent an increase in SVR
- Maintain a heart rate that is normal or slightly increased
- Maintain sinus rhythm, if present. Aggressively treat acute atrial fibrillation
- Avoid aortocaval compression and maintain venous return, but prevent an increase in central vascular volume
- Avoid myocardial depression during general anesthesia
- Prevent pain, hypoxemia, hypercarbia, and acidosis, which may increase pulmonary vascular resistance
- Maternal monitoring should include continuous ECG monitoring. Invasive monitoring is required in cases of severe mitral regurgitation. If pulmonary edema or refractory hypotension develops, pulmonary artery catheter helps in treatment

- Continuous epidural anesthesia is preferred for labor or cesarean section. Epidural anesthesia prevents the increase in SVR that is associated with pain. Epidural anesthesia may result in some decrease in SVR, which promotes the forward flow of blood and helps prevent pulmonary congestion.[11] However, epidural anesthesia may result in decreased venous return. Careful administration of intravenous crystalloid and left uterine displacement are necessary to maintain venous return and left ventricular filling. In contrast to patients with mitral stenosis, patients with mitral regurgitation may benefit from the chronotropic effect of ephedrine if a vasopressor is required
- If general anesthesia is required, the anesthesiologist should give attention to the maintenance of adequate heart rate and decreased afterload. The increased heart rate associated with ketamine and pancuronium is desirable in these patients. Myocardial depression should be avoided. Hypoxemia, hypercarbia, acidosis, and hypothermia produce an undesirable increase in pulmonary vascular resistance and should be avoided.
- Acute atrial fibrillation must be treated promptly and aggressively. Hemodynamic instability warrants the immediate performance of cardioversion.

Mitral Valve Prolapse (MVP)

Myxomatous degeneration of the mitral valve affects the cusps, chordae tendinae, and annulus and may cause the valve to billow into the left atrium during systole. Involvement of the chordae tendinae can lead to chordal rupture and subsequent mitral regurgitation. MVP can result from papillary muscle ischemia or endocarditis.

Most patients are asymptomatic. However, some women develop palpitations, chest pain, anxiety, fatigue, and light headedness.

The auscultatory findings are the midsystolic click and late systolic murmur. The intensity of these auscultatory findings may decrease during pregnancy because of the expansion of the maternal intravascular volume and decreased SVR. These conditions increase ventricular volume, enhance forward blood flow, and lessen the prolapse of the mitral valve.

The diagnosis is made by echocardiography. The most common echocardiographic finding is abrupt posterior movement of both valve leaflet or only the posterior leaflet in midsystole. These patients frequently have exaggerated motion of the anterior leaflet; however, actual prolapse into the left atrium appears to be more common with the posterior leaflet.

The ECG is usually within normal limits in asymptomatic patients. However, nonspecific changes in the inferior and anterolateral leads and a variety of arrhythmias may occur. Paroxysmal supraventricular tachycardia is the most common tachyarrhythmia.[12] Ventricular arrhythmias have been implicated in rare cases of sudden death. There also is a high incidence of MVP among patients with Wolff-Parkinson-White syndrome.

Neurologic complications include acute hemiplegia, transient ischemic attacks, cerebellar infarcts, amaurosis fugax, and retinal arteriolar occlusions. These complications usually result from an embolic etiology. Cardiac arrhythmias may contribute to the embolic events.

A beta blocker may be necessary to treat arrhythmias, chest pain, and palpitations. Because progressive mitral regurgitation occurs in approximately 15 percent of patients with MVP, treatment of left ventricular failure with digoxin and a diuretic therapy may be required.

Most patients tolerate pregnancy very well, with no increased risk of obstetric complications or fetal stress.

Anesthetic consideration

- The severity of coexisting disease often dictates whether invasive hemodynamic monitoring is required
- Regional anesthesia is an excellent choice for labor and cesarean delivery. The sympathectomy and decreased concentrations of catecholamines are beneficial for these patients
- In some cases, sedation with small doses of an opioid or a benzodiazepine may be required
- When general anesthesia is required, sympathomimetic agents (e.g. ketamine, pancuronium) should be avoided because of the high incidence of arrhythmias
- Avoid agents (halothane) that sensitize the myocardium to catecholamines
- Ephedrine may precipitate or exacerbate tachyarrhythmias
- Hypotension can be treated with small bolus doses of phenylephrine.

Asthma

Asthma produces the clinical manifestations of wheezing, cough, and dyspnea.

The pathophysiological mechanisms include:

- An increased contractility or an impaired relaxation of airway smooth muscle
- A neural imbalance with the bronchoconstricting parasympathetic system domination
- Airway inflammation
- Changes in the function of airway epithelium.

Effect of Pregnancy on Asthma

Pregnancy has no consistent effect on asthma. Asthma is a variable disorder whose severity depends on many factors like exposure to allergens, respiratory infections, season and patient's emotional state.

Factors Improving the Asthma in Pregnancy

Progesterone, increased production of bronchodilating prostaglandin and increased levels of circulating cortisol produces relaxation of bronchial smooth muscles.

Factors Worsening Asthma in Pregnancy

Decreased sensitivity to beta-adrenergic agonists, increased production of bronchoconstricting prostaglandins and reduced sensitivity to circulating cortisol due to binding of steroid hormones to cortisol receptors.[13]

Effect of Asthma on Parturient and Fetus

There is an increased incidence of pre-eclampsia, cesarian delivery, low birth weight (LBW) infants, preterm labor, antepartum and postpartum hemorrhage, and perinatal mortality. Premature rupture of membranes, neonatal hypoxia, transient tachypnea of newborn. The adverse outcomes is higher in patients with severe or poorly controlled asthma and corticosteroid dependent patients.[14]

Medical management

Whenever asthma is carefully managed by a physician, the risk of adverse outcomes does not increased significantly. The drugs that are routinely used in pregnant ladies are given below.

Bronchodilators

Beta adrenergic agonists (e.g. albuterol, salmeterol): Inhaled beta adrenergic agonists are not associated with congenital malformations, increased incidence of intrauterine growth restriction, preterm delivery, or perinatal mortality.[15]

Methylxanthines (e.g. theophylline, aminophylline): These drugs have been used for many years in pregnant asthmatic subjects without detrimental effects on the fetus. Serum concentrations of these agents should be monitored carefully, especially in the third trimester, when theophylline clearance decreases.

Anticholinergic agents (Ipratropium bromide): The aerosol drug to the lungs causes less systemic absorption and reduced serum concentrations in the fetus.

Anti-inflammatory Agents

C**orticosteroid** action include (1) decreases in cellular infiltration and mediator release, (2) decreases in airway permeability, and (3) up-regulation of the beta-adrenergic system.

Corticosteroids do not increase the risk of congenital malformations or perinatal risk in humans.[16] Corticosteroids with beta-adrenergic agonist may aggravate maternal glucose intolerance. Hence monitoring of maternal glucose is indicated.

The potential for adrenal insufficiency in infants of mothers taking corticosteroids appears to be very low. In the mother, prednisone is converted rapidly to prednisolone, which crosses the placenta to a very limited extent.

Cromolyn sodium and **nedocromil sodium**: An increased risk of teratogenicity with cromolyn sodium, is not seen.

Obstetric Management

Three aspects of obstetric management of the asthmatic parturient differ from normal patient: (1) induction of labor, (2) management of postpartum hemorrhage, and (3) treatment of hypertension:

Induction of labor: Prostaglandins should be avoided. Prostaglandin $F_{2\text{-alpha}}$ constricts respiratory smooth muscles and cause bronchospasm when used to induce labor. Prostaglandin E_2 dilates airways in vitro but aerosols can precipitate bronchoconstriction in asthmatic subjects due to irritant effect. Similarly 15-methyl prostaglandin F2-alpha (carboprost) causes bronchoconstriction in asthmatic patients. [17]

Antihypertensive agents: In asthmatic patients, beta blokers can provoke bronchospasm, hence should be avoided. Other **antihypertensive agents** (e.g. hydralazine, sodium nitroprusside, trimethaphan) do not enhance airway responsiveness and can be used.

Anesthetic Consideration

- Preoperative evaluation: A clinical history to elicit the severity of the patient's asthma and treatment should be taken
- The clinical history should include duration of disease, triggering factors and allergens, cough and sputum characteristics and medications.

- Blood eosinophil counts often parallel the degree of airway inflammation, and airway hyper-reactivity
- Chest physiotherapy, antibiotic and bronchodilator therapy during the preoperative period often improve asthma
- Hypothalamic-pituitary-adrenal suppression is unlikely with inhaled corticosteroids. However, preoperative corticosteroids supplementation should be given if patient is on long-term oral steroids therapy
- Ideally patients should be free of wheezing and have a peak expiratory flow greater than 80 percent of predicted or at patient's best value prior to surgery
- The use of anticholinergic drugs (atropine) for premedication should be individualized, as these drugs can increase the viscosity of airway secretions, making them more difficult to remove from the airway
- However in case of emergency, time may not be available to prepare the patient
- General anesthesia will be required for urgent LSCS (fetal distress), hemorrhage, and presence of coagulopathy
- Induction of anesthesia with propofol is preferred compared to thiopentone. Thiopentone may inadequately suppress upper airway reflexes so airway instrumentation may trigger bronchospasm. Propofol is effective in blunting airway response and also may have weak bronchodilating action
- Ketamine gives greater protection against reflex-mediated bronchoconstriction than thiopental, due to its sympathomimetic properties. Ketamine also produce respiratory smooth muscle relaxation
- Intravenous lignocaine (1 mg/kg) attenuates airway reflexes and hemodynamic responses to endotracheal intubation, hence is used as an adjunct during induction. Aerosolized lignocaine can be an airway irritant therefore it should not be used
- Succinylcholine is commonly used for rapid sequence intubation. Newer agent like Rocuronium is also safe alternative. Atracurium and rapacuronium can worsen bronchospasm by histamine release. Unlike atracurium, cisatracurium does not cause histamine release. Vecuronium also does not exhibit this property and is safe
- Inhalational agents (Halothane, Isoflurane and Sevoflurane) are preferred for the maintenance of anesthesia as they cause reduction in airway resistance by direct effect on airway smooth muscle. Halothane may cause cardiac irritability when used with adrenaline or aminophylline
- Halothane produces greater bronchodilatation than isoflurane at lower concentrations (0.6 to 1.1 MAC), and is preferred.The lesser pungency of halothane and sevoflurane (compared with isoflurane and desflurane) decreases coughing and bronchospasm[18]
- Halothane, enflurane, and isoflurane relax uterine smooth muscle at high concentration. Hence, nitrous oxide, an intravenous opioid, and a low concentration of volatile agent are preferred for maintenance of anesthesia
- Adequate oxygenation and ventilation should be maintained. A slow inspiratory flow rate provides better ventilation. Sufficient time for exhalation is necessary to prevent air trapping. Humidification and warming of inspired gases is beneficial
- Reversal of neuromuscular blockage with neostigmine may exacerbate asthma by increasing secretions and bronchospasm. This response is attenuated by glycopyrrolate or atropine
- At the end of surgery, remove the endotracheal tube while anesthesia is adequate to suppress hyperreactive airway reflexes. Whenever there is risk of aspiration, extubation should not be performed unless patient is fully awake. One can decrease airway reflexes and the risk of bronchospasm by administration of intravenous lignocaine or inhaled bronchodilators just prior to extubation
- Postpartum hemorrhage: Asthmatics have a high incidence of postpartum hemorrhage as compared to nonasthmatics
- Ergot alkaloids (ergometrine) should be avoided in asthmatics as it causes bronchospasm due to tryptaminergic action or alpha receptor stimulation of respiratory smooth muscles. Oxytocin do not have any effect on airway muscle tone, and hence is the preferred ecbolic agent in asthmatic patients.[19]

Hypertension, Pre-eclampsia, and Asthma: All beta blockers especially nonselective agents (Labetalol) should be used with caution in asthmatics. Vasodilators like hydralazine, calcium-channel blockers, nitroglycerine and nitroprusside can be used in asthmatic pregnant patient with PIH. Magnesium infusions, commonly used for seizure control, also have beneficial effect by relaxation of bronchial smooth muscles.

Regional Anesthesia for Cesarean Section

By avoiding instrumentation of the hyperactive airway, regional anesthesia is preferred to GA in asthmatic patients. Careful attention must be paid to avoid higher level of block (above T6) which may compromise respiratory muscle function. The level of motor block must be kept up to T6-8 dermatome. If intrathecal or epidural opioid is used, the patient must be monitored for respiratory depression. In addition, the use of a dilute concentration of bupivacaine combined with a

low dose of fentanyl is safe for the fetus and produces satisfactory analgesia with less motor block than bupivacaine alone.

ANEMIA

Anemia is defined as a hemoglobin (Hb) concentration <12 g/dl in nonpregnant women. During pregnancy, plasma volume expands proportionately more than Hb or red blood cell volume, resulting in Hb dilution, such that anemia is defined as Hb concentration <10 g/dl. The initial work-up consists of a history, physical examination as well as an examination of the red blood cell indices and a peripheral smear, with additional tests as indicated.

Iron Deficiency Anemia

Iron deficiency is the most common cause of anemia in pregnant women.

Iron deficiency anemia shows the following parameters; Mean corpuscular volume (MCV) is <80/mm^3 an elevated total iron-binding capacity (TIBC), a low serum iron level, a serum iron-to-TIBC ratio <20 percent, and a low ferritin level.

The total iron requirement in pregnancy is 1000 mg: 500 mg increases the maternal red blood cell mass, 300 mg is used by the fetus and placenta, and 200 mg compensates for blood loss at delivery.

The iron requirements of pregnancy increase steadily toward term but average 3.5 mg per day which cannot be obtained from diet only.

Anemic pregnant patient (Hb of 8 or 9 g/dL) should take 300 mg ferrous sulate (contain 60 mg elemental iron) two or three times per day. Vitamin C facilitates iron absorption.

The severely anemic patient (Hb <8 g/dl) may require parenteral therapy in the form of intramuscular or intravenous iron dextran. Adequate parenteral therapy should result in a marked increase in the reticulocyte count within 7 to 14 days.

Effect of Anemia on Fetus and Mother

The incidence of neonatal anemia is increased. Iron is transported actively across the placenta, and fetal iron and ferritin levels are three times higher than maternal levels. Severe anemia (Hb <8 g/dl) is associated with intrauterine growth restriction (IUGR).

In pregnant women with anemia, symptoms including fatigue, headache, light headedness, and reduced exercise tolerance. Blood loss at delivery may be tolerated poorly in anemic patients, and postpartum tissue healing may be compromised.

Increased 2,3-DPG (Diphosphoglyceraldehyde) concentrations in RBCs are principally responsible for maintaining oxygen-carrying capacity in the presence of chronic anemia.

The cardiac output does not rise till Hb concentrations decrease to approximately 7 g/dL in anemic parturient and at this point blood transfusion should be considered.

Packed RBCs can be administered to increase Hb concentrations without significantly increasing blood volume and volume overload on a compromised heart. Approximately 24 hours is needed to restore intravascular fluid volume. In comparison with similar volumes of whole blood the packed RBC's produce approximately twice the increase in Hb concentration.

Anesthetic considerations

- It is important to avoid factors that can further decrease oxygen delivery to tissues, for example, drugs causing tachycardia (atropine, ketamine) should be avoided and drug induced depression of cardiac output must be prevented
- A leftward shift of the oxyhemoglobin dissociation curve due to respiratory alkalosis from iatrogenic hyperventilation during GA can interfere with tissue oxygen delivery
- The patient must be kept warm, as decreased body temperature shifts the oxyhemoglobin dissociation curves to the left causing less oxygen delivery to peripheral tissues
- Measures to decrease the impact of surgical blood loss by normovolemic hemodilution and intraoperative blood salvage should be considered in selected patients
- Effects of anesthetic drugs on the sympathetic nervous system and cardiovascular depression may blunt the increase in cardiac output associated with acute normovolemic anemia
- The effect of decreased solubility of volatile anesthetics due to anemia is probably offset by the impact of increased cardiac output. Clinically there is no change in the rate of induction of anesthesia or overdose with inhalational anesthetic
- It is advisable to replace intraoperative blood loss with whole blood or packed RBC when Hb concentrations decrease acutely to less than 7 g/dl, especially if there is coexisting cardiovascular or cerebrovascular disease.[20]

Megaloblastic Anemia

Megaloblastic anemia is characterized by red blood cells with increased Mean Corpuscular Volume (MCV) and white blood cells with altered morphology (hypersegmented neutrophils, anisocytosis, and poikilocytosis). It is commonly caused by folate deficiency, it can also occur after exposure to sulfa drugs, hydroxyurea or, rarely, vitamin B_{12} deficiency.

Folate deficiency can develop in a relatively short time, as liver stores of folate is limited (meet body's needs for only 1 to 2 months). Associated malnutrition, malabsorption, anticonvulsant therapy, oral contraceptive use, or pregnancy can rapidly deplete the body's folate stores. A pregnant woman needs 300 to 400 mg.

In contrast, vitamin B_{12} deficiency is rare because very little of the body's stores are used each day.

Sickle Cell Disease

A mutation leading to a single amino acid substitution of valine for glutamic acid at the sixth amino acid residue on the β2 chain protein changes normal Hb to sickle Hb. An individual who is homozygous for this mutation has sickle cell anemia, or sickle cell disease, producing only HbS and a small quantity of HbF but no HbA. Sickle Hb functions well in the oxygenated state but aggregates, forming rod-shaped polymers in the deoxygenated state. Polymerized Hb precipitates in the red blood cell, changing the cell from a biconcave disk to an elongated crescent or sickle shape. Sickled red blood cells are rigid and cannot squeeze through the microcirculation. Microvascular obstruction results in local hypoxia that leads to a vicious cycle of further sickling and obstruction. Localized ischemia and infarction cause tissue damage.

Patients with sickle cell anemia usually produce increased quantities of HbF. HbF is present in concentration of 0 to 20 percent per cell. In cells containing HbF, restoration of normal oxygen tension may reverse the sickling and halt the destructive process. Cells containing little or no HbF become irreversibly sickled and are rapidly cleared from the system in a process leading to hemolytic anemia.

Hydroxyurea increase both the number of red blood cells and the quantity of HbF per cell. However, it is a known teratogen and not used in pregnancy.

Any pathologic state causing acidosis, dehydration, or hypoxemia can precipitate sickling, hemolysis, vaso-occlusion, and infarction. Pregnancy usually is characterized by an increase in sickle crisis and its complications like pneumonia, pyelonephritis, pulmonary emboli, congestive heart failure and pregnancy related complications (e.g. IUGR, preterm birth, pre-eclampsia).[21]

The goal of pregnancy management should be to maintain adequate hydration and oxygen delivery to the tissues and to avoid or rapidly control infections or other stressors that could precipitate a sickle cell crisis.

Patients should ingest 1 mg of folate per day to support increased erythropoiesis because of chronic hemolysis and should receive the polyvalent pneumococcal vaccine because chronic splenic infarction leads to a functional spleen by adulthood.

Iron supplementation should be prescribed if there is evidence of iron-deficiency anemia. Asymptomatic bacteriuria (ASB) and other infections should be treated aggressively.

- Blood transfusion should be considered prior to delivery or surgery, because the stresses of labor, anesthesia, operative delivery, and any associated complications (e.g., pre-eclampsia, chorioamnionitis) can precipitate a serious sickle cell crisis
- Transfusions should achieve a hematocrit above 30% and an HbA above 50 percent.

OBESITY

Obesity is a disease of modern civilization. Obesity is defined as an excess of body fat

Ideal weight (kg) = Height (cm) – 100

Other measure is Body Mass Index (BMI)

BMI = Weight (kg)/Height (meter square)

A person with less than 20 percent in excess of ideal weight is described as **overweight.**

A person with more than 20 percent of ideal weight is **obese**.

A person is **morbidly obese**, when his body weight is more than twice the normal weight or when it exceeds by more than 100 lbs, the ideal weight for age and height.

A BMI of less than 25 is normal, 25 to 29.9 is overweight, and over 30 is obese. A BMI more than 40 is morbid obesity.

There is an increased incidence of hypertension, coronary artery disease, cerebrovascular disease, diabetes mellitus, gallbladder disease, and liver disease in obese, patients. Pregnancy-associated weight gain also affects the weight of women. Fortunately, pregnancy primarily results in an accumulation of lower body fat, which has less health risk than upper body fat.

Physiologic Changes of Obesity

Pulmonary Changes

Excess body weight requires increased energy expenditure proportionate to the increase in body mass and surface area. Increased energy expenditure increases oxygen consumption and CO_2 production independent of the metabolic rate, which remains constant in obese patients. Oxygen consumption and CO_2 production double as weight doubles. Minute ventilation increases to meet increased oxygen demand.[22]

Patients with Pickwickian syndrome or Obese Hypoventilation Syndrome (OHS) have hypoventilation leading to hypercapnia, hypoxemia, polycythemia and somnolence. They have raised pulmonary artery pressure and cardiac failure can occur.

Increased chest wall weight requires greater energy expenditure during ventilation. The oxygen requirement of breathing rises proportionately with weight. The respiratory work efficiency falls in obese patients (measured as a ratio of mechanical work divided by the oxygen cost of breathing). The weight gain associated with pregnancy further increases the work of breathing in obese patients. Frequent shallow respirations are seen. The obese patient conserves energy by decreasing tidal volume. This ventilatory pattern contrasts with the increased tidal volume that typically accompanies pregnancy. Nonetheless, most morbidly obese pregnant women have a P_aCO_2 that is normal for pregnancy. However, pulmonary reserve is decreased.

Lung Volume

Obesity alters lung volumes. Increased abdominal weight restricts diaphragmatic movement, especially in the supine or Trendelenburg position; this causes smaller tidal volumes. Increased chest wall weight decreases the chest wall expansion. Functional residual capacity (FRC) decreases, and it may be less than closing capacity. This results in airway closure during tidal ventilation in morbidly obese patients. Similarly, expiratory reserve volume, vital capacity, inspiratory capacity, total lung capacity, and maximum minute ventilation all decrease in the morbidly obese patient. Although total compliance decreases, pulmonary compliance and airway resistance remain normal. In addition pregnancy also alters lung volumes. In pregnant patient, expiratory reserve volume and FRC decline approximately 20 to 25 percent by term.[23]

Oxygenation

The pulmonary diffusion remains normal. Decreased chest wall compliance and the increased weight of abdominal contents cause airway closure in the dependent lung. Ventilation preferentially occurs in the more compliant, nondependent portion of the lung while pulmonary blood flow preferentially occurs in the dependent portion of the lung. This results in ventilation-perfusion mismatch and hypoxemia. Similarly oxygenation worsens in the supine and Trendelenburg positions.

Cardiovascular Changes

Obesity increases blood volume and cardiac output. The cardiac index remains normal. Increased cardiac output primarily results from increased stroke volume. The systemic arteriovenous oxygen difference remains normal. Pulmonary blood volume increases in proportion to cardiac output and total blood volume. Pulmonary hypertension can occur and it may be position dependent. Chronic hypoxemia causes increased pulmonary vascular resistance. Airway obstruction may also increase pulmonary artery pressure. Hypertension occurs more commonly in obese patients. A clear relationship exists between obesity and death from cardiovascular causes.

There is an association between increased left ventricular mass and increased weight. In the morbidly obese pregnant women, left atrial size, left ventricular thickness, interventricular septal thickning and an increase in left ventricular mass. Fatty infiltration of the heart can occur, especially the right ventricle. Eccentric LVH is a major cause of increased heart size. Inadequate hypertrophy and chamber dilatation predispose some patients to myocardial decompensation. There is an increase in premature ventricular contractions in obese patients with eccentric left ventricular hypertrophy (LVH).

Gastrointestinal Changes

It is observed that obese patients have an increased risk of developing aspiration pneumonitis. They have a gastric pH less than 2.5 and gastric volume more than 25 ml. The combination of pregnancy and obesity further increases gastric volume and decreases gastric pH. Hiatal hernia is more common in obese patients. The intragastric pressure is greatly raised by omental fat and the weight of the panniculus. Hence there is an increased risk for pulmonary aspiration of gastric contents.[24]

Endocrine Changes

Diabetes mellitus occurs more commonly as there is a relative insulin resistance in obese parturients.

Coagulation Changes

It is likely that obesity increases the risks of deep vein thrombosis and pulmonary thromboembolism associated with pregnancy.

Interaction with Pregnancy

Obesity adversely affects pregnancy outcome. Obesity is associated with an increased risk of chronic hypertension, pregnancy induced hypertension (PIH), and diabetes mellitus during pregnancy.[25]

Thus, obesity increases the risk of maternal death during pregnancy and during anesthesia.

Obstetric Management

There is an increased incidence of cesarean section in pregnant obese patients due to abnormal presentation, fetal macrosomia, prolonged labor and associated medical complications. There is an increased incidence of meconium-stained amniotic fluid, umbilical cord accidents, and late fetal heart rate (FHR) decelerations during labor in obese patients.

Perinatal Outcome: Obese women are at decreased risk for preterm delivery, LBW infant or delivery of small-for-gestational-age (SGA) infant. Higher maternal weight increases the risk of late fetal death.

Anesthetic Consideration

- The high incidence of associated medical diseases in obese pregnant women requires careful preanesthetic assessment
- Patient should be investigated for Hb, CBC, Platelet count, ECG, thyroid function test, blood glucose (fasting and postprandial), BUN, serum creatinine, BT, CT, PT and echocardiogram
- Using an appropriate size blood pressure cuff for correct blood pressure measurement is necessary. Unless the length of the sphygmomanometer cuff exceeds the circumference of the arm by 20 percent, systolic and diastolic blood pressure measurements may overestimate true maternal blood pressure
- Appropriately sized labor beds, transportation trolleys, and operating tables are needed. Have a sufficient number of personnel to help in patient transport
- It is difficult to establish intravenous access in some obese patients. However, the increased concentrations of steroid hormones during pregnancy cause venodilatation, which may lessen the technical challenge.

Regional Anesthesia for Emergency LSCS

Regional anesthesia is preferred to GA as it avoids airway difficulties and associated respiratory complications. Spinal and epidural needles longer than those commonly used are needed.

Lumbar Epidural Anesthesia

It is preferred if the coagulation profile is normal. It has the following advantages (1) the ability to titrate the dose of local anesthetic agent, (2) a decreased incidence of hypotension, and (3) a decreased potential for high motor blockade.

In the obese patient, the level of the block is proportional to BMI and weight but not height. The sitting position decreases the cephalad spread of neuraxial block in obese patients.

The obese patient may requires more attempts to identify the epidural space. Use of the sitting position helps in identification of the midline of the spinal column and subsequent subarachnoid or epidural space. Also, the distance from the skin to the epidural space is less when the patient is sitting. The increased depth of the epidural space contributes to the high failure rate of epidural analgesia. There is increased incidence of unilateral blockade. Ultrasonographic guidance helps in identification of the epidural space.

When the patient moves from the sitting to the lateral position, there is increased chance of catheter displacement if the catheter is secured before the change in patient position. This can be avoided by securing the epidural catheter after positioning the patient. The block should be bilateral and total. Otherwise, the catheter should be removed and reinserted.

In cases of unintentional dural puncture, continuous spinal analgesia can be used for LSCS. In such cases small titrated doses of hyperbaric local anesthetic agent should be used. The intradural catheter must be promptly removed after surgery to prevent misuse.

Spinal Anesthesia for LSCS

Obesity can cause an unpredictable, exaggerated spread of the local anesthetic agent due to following reasons; Hormonal and mechanical factors cause a decreased local anesthetic dose requirement in pregnant women. Raised abdominal pressure may cause compression of the inferior vena cava, which increases blood flow through the epidural venous plexus and reduces the

cerebrospinal fluid volume in subarachnoid space, lower CSF volumes in obese patients increase the risk of a high spinal block. The large buttocks of obese patients place the vertebral column in a Trendelenburg position and result in an exaggerated spread of regional anesthesia causing high spinal anesthesia.

Spinal anesthesia causes a profound thoracic motor blockade as compared to epidural anesthesia. This may adversely affect oxygenation and ventilation of the obese parturient.

The duration of surgery in obese patients may extend beyond the duration of single-shot spinal anesthesia and intraoperative induction of general anesthesia may be required which is undesirable and hazardous.

General Anesthesia for LSCS

- An urgent LSCS due to coagulopathy, hemorrhage or fetal distress will require GA
- A careful, airway assessment is essential. Failed intubation with pulmonary aspiration is a common cause of anesthetic death in obese parturients
- Obese pregnant patients require aggressive prophylaxis to prevent aspiration. The following measures should be taken; 30 ml of 0.3 M solution of sodium citrate increases gastric pH within 5 minutes. Administration of an H_2 antagonist and metoclopramide provide additional protection
- It is difficult to position the patient appropriately. The protuberant abdomen may shift when the patient is tilted toward the left. Hence the patient must be secured to the operating table before she is tilted leftward. It is important to initiate left uterine displacement to prevent vena caval-compression and hypotension[26]
- The anesthesia team can be asked to help in cephalad retraction of the large panniculus. The anesthesiologist must be aware of the risk of hypotension and fetal compromise during cephalad retraction of the panniculus in a morbidly obese patient. This decreases chest wall compliance causing dyspnea. The fetal heart rate must be monitored
- Obese patients commonly have a short neck. Increased fat in the neck and shoulders makes it more difficult to position the patient appropriately for intubation. Large breasts, increased anteroposterior diameter of the chest, airway edema, and decreased chin-to-chest distance all increase the incidence of difficult laryngoscopy and failed intubation in obese pregnant patient
- Endotracheal intubation may be difficult or impossible with standard techniques. The history of a previous successful intubation does not guarantee the same result during a subsequent procedure
- Patient position before intubation is important. The shoulders are elevated, allowing the breasts to fall away from the neck and chin, while folded towels support the occiput and place the head in sniffing position. Further, the fat pads on the back of the shoulders often restrict the range of neck movements which makes mask ventilation, laryngoscopy, and intubation difficult
- Mask ventilation and intubation: The potential for failed intubation and difficult mask ventilation highlights the need for an experienced assistant. The primary anesthesiologist fatigues rapidly while attempting mask ventilation in an obese patient. Further, the jaw-thrust manoeuvre may require the use of both hands, and a second person must be available to provide positive-pressure ventilation and cricoid pressure
- Difficult mask ventilation causes gastric distension with ventilating gases, which increases the risk of regurgitation and pulmonary aspiration of gastric contents
- Obesity impairs accurate identification of the cricoid ring; making it difficult for the assistant to apply cricoid pressure correctly during rapid-sequence induction of general anesthesia. The prepared anesthesiologist must have a short-handled laryngoscope, assorted laryngoscope blades including adjustable-angle blade, endotracheal tubes of smaller sizes, and equipment for performing percutaneous cricothyrotomy and transtracheal ventilation immediately available. Other devices like intubating laryngeal mask airway (LMA) using fiberoptic light source such as Bullard laryngoscope can be helpful
- Obese patients are prone to laryngeal edema hence smaller-diameter endotracheal tube must be used[27]
- It is difficult to perform cricothyrotomy or transtracheal jet ventilation in obese patient. In failed intubation, hypoxia and acidosis develop rapidly in the obese parturient
- In elective LSCS, awake fiberoptic intubation using mild sedation and adequate topicalization of airway can be done. In emergency situations awake direct laryngoscopy following topicalizing the upper airway is done. However, catecholamine release and blood pressure elevation can occur
- Pregnant obese women become hypoxemic more rapidly than nonobese patients. Therefore, adequate denitrogenation (preoxygenation) is necessary before general anesthesia. There is rapid onset of hypoxemia in patients who had four maximal

inspirations of 100 percent oxygen as compared to similar patient who had. Hence, the 3 minutes of tidal ventilation with 100 percent oxygen method of denitrogenation should be used in emergencies

- Obesity alters the distribution and response of anesthetic drugs. An increased volume of distribution prolongs the elimination half-life of thiopental in obese patients. Thiopental administration of less than 4 mg/kg may increase the risk of maternal awareness, hypertension, and decreased uterine blood flow during light anesthesia, while administration of a larger dose may be associated with delayed arousal in the event of failed intubation
- Succinylcholine remains the muscle relaxant of choice for rapid-sequence induction. Administer succinylcholine on the basis of total rather than lean body weight in adult (at least 1.0–1.5 mg/kg ups to a maximum dose of 200 mg)
- Adequate muscle relaxation by nondepolarizing relaxants is necessary. Their doses are guided by peripheral nerve stimulator. Morbidly obese patients usually have a normal response to nondepolarizing muscle relaxants
- No evidence suggests that obesity alters the minimum alveolar concentration (MAC) for inhalation agents in pregnant women. In theory, increased body fat serves as a reservoir for inhalation and intravenous agents. Likewise, the body fat reservoir could increase the risk of biotransformation of the volatile halogenated agents, which would increase the risk of organ toxicity
- Isoflurane is a better choice than halothane or enflurane because of its decreased biotransformation in morbidly obese patients. The newer inhational agents—desflurane, sevoflurane reduce the time to extubation, as compared to isoflurane in obese patients. However, sevoflurane is associated with higher serum inorganic fluoride concentrations in obese patients
- High concentrations of volatile halogenated agent increase neonatal depression, uterine atony, and maternal blood loss, hence nitrous oxide, low dose of opioid and decreased concentration of a volatile halogenated agent should be used
- The morbidly obese patients may require higher inspired concentration of oxygen thus reducing nitrous oxide concentration. Administration of general anesthesia results in decreased functional residual capacity (FRC). Further, the supine and Trendelenburg positions may further decrease the FRC and increase the likelihood of intraoperative hypoxemia

Techniques that may improve intraoperative oxygenation include:

- Use of a large tidal volume
- Administration of positive end-expiratory pressure (PEEP): PEEP may increase maternal PaO_2, but it may decrease cardiac output, decreased uterine blood flow and oxygen delivery to fetus in case of prolonged induction-to-delivery interval
- Elevation of the panniculus
- Airway obstruction may increase PCWP and precipitate cardiovascular decompensation. Thus, it is important to avoid airway obstruction during induction and emergence from anesthesia.

Postoperative management: In obese patients, postoperative respiratory complications are frequent and hypoxemia can persist for several days hence, supplement of oxygen is given. The obese patient is placed in sitting position, and intensive chest physiotherapy is given. To prevent atelectasis, encourage the patient for deep breathing and provide adequate postoperative analgesia. Epidural anesthesia decreases the risk of thromboembolic complications by providing better pain relief and early mobilization.

THYROID DISORDERS

Thyroid Hormone Physiology

The thyroid hormones are highly protein bound in the blood. In euthyroid nonpregnant humans, the normal total serum concentrations of T_4 and T_3 are 50 to 150 nmol/L and 1.4 to 3.2 nmol/L respectively. The unbound or free fractions of T_4 and T_3 are 0.03 percent and 0.3 percent, respectively. The serum T_4 and T_3 are bound to three plasma proteins: (1) thyroxine-binding globulin (TBG) (70–80%), (2) thyroxine-binding prealbumin or transthyretin (10–20%), and (3) albumin (10–15%).

In pregnancy: The serum concentration of TBG (thyroxine-binding globulin) steadily increases until it reaches a plateau at 20 weeks' gestation, when it is 50 percent greater than the nonpregnant level. The increased concentration of TBG results from a prolonged half-life (not increased synthesis) during pregnancy. The normal pregnant woman is euthyroid because the serum concentrations of free T_4 and T_3 are in the normal (or low-normal) range for nonpregnant female. However, the increased concentration of TBG means that the total serum concentrations of T_4 and T_3 during pregnancy are at higher levels.

Maternal iodine availability is decreased during pregnancy because of increased fetal uptake and

increased maternal renal clearance. Patient living in the region with marginal iodine supply may predispose the mother to goiter unless she has a dietary iodine supplement.

Hyperthyroidism

The common etiologies are:
Abnormal thyroid stimulator: Graves' disease, Gestational trophoblastic neoplasia, TSH-secreting pituitary tumor.
Intrisic thyroid autonomy: Toxic adenoma or toxic multinodular goiter.
Inflammatory disease: Subacute thyroiditis.
Extrinsic hormone source: Ectopic thyroid tissue, Thyroid hormone ingestion.

Clinical Features

Hyperthyroidism presents clinically as a physiologic state with increased metabolic rate. A hyperthyroid symptoms scale has been developed based on 10 clinical factors: nervousness, sweating, heat intolerance, hyper-activity, tremor, weakness, hyperdynamic precor-dium, diarrhea, appetite, and degree of incapacitation. Exophthalmus or infiltrative ophthalmopathy is common in Graves' disease. Other physical signs seen at low frequency: pretibial myxedema, dermopathy and nail changes or acropachy. Hyperthyroidism stimulates the cardiovascular system causing an hyperkinetic circulatory state. There is increase in myocardial contractility, heart rate, stroke volume, and ventricular size and peripheral vascular resistance decreases in skin and muscle. A cardiomyopathy can occur which is reversible with normalization of thyroid function. The thyroid function tests (TFT) show an increase in the serum concentration of free T_4.

Interaction with Pregnancy

Graves' disease is the predominant cause of hyperthyroidism during pregnancy. The prevalence of hyperthyroidism during pregnancy is lower than that in the general population because of the beneficial effect of immunotolerance of pregnancy on autoimmune disorders like Graves' disease.[28] Pregnancy is associated with an increase in number and size of thyroid nodules. Pregnancy commonly does not affect the development or progress of thyroid carcinoma.

Fortunately, pregnancy appears to attenuate the severity of hyperthyroidism and doses of antithyroid drugs can be kept low, i.e. propylthiouracil (PTU) <200 mg/day. If doses greater than 300 mg/day of PTU are needed during the first trimester, a subtotal thyroidectomy should be performed in the second trimester.

Obstetric Management

Hyperthyroidism is associated with increased rates of spontaneous abortion, preterm delivery, and congenital goiter. Poorly controlled hyperthyroidism is associated with a higher incidence of pre-eclampsia. The early diagnosis and treatment of hyperthyroidism during pregnancy is associated with better maternal and fetal outcome.

Fetal goiter may develop as a result of the placental transfer of antithyroid medications. Fetal goiter can interfere with vaginal delivery, or it may lead to airway obstruction in the neonate.

Anesthetic Consideration

Many features of hyperthyroidism can affect anesthetic management:

- The hyperdynamic cardiovascular system and the possibility of cardiomyopathy
- Partial airway obstruction secondary to an enlarged thyroid gland
- Respiratory muscle weakness
- Electrolyte abnormalities
- Preoperative preparation: In an emergency LSCS, a hyperthyroid patient can be prepared for surgery with oral propylthiouracil and intravenous gluco-corticosteroid, sodium iodide, and propra-nolol. The anesthesiologist should be prepared to treat peri-operative thyroid storm in patients with uncontro-lled or poorly controlled disease for emergency cesarean section
- Premedication should include a barbiturate, benzodiazepine, and/or a narcotic. Anticholinergic drugs (i.e. atropine) should be avoided since they may precipitate tachycardia and alter heat-regulating mechanisms
- Adequate anesthetic depth is important to avoid exaggerated sympathetic nervous system (SNS) responses. Drugs that stimulate the SNS should be avoided in the hyperthyroid patient (i.e. ketamine, pancuronium, atropine, ephedrine, epinephrine)
- For induction of anesthesia thiopental, due to its thiourylene nucleus, decreases the peripheral conversion of T_4 to T_3 have a slight advantage over other induction agents
- Succinylcholine and the nondepolarizing muscle relaxants with limited hemodynamic effects (e.g.

vecuronium, rocuronium) can be used safely for intubation
- Hyperthyroid patients having coexisting muscle disease (e.g. myasthenia gravis) may require reduced doses of nondepolarizing muscle relaxants with careful titration using peripheral nerve stimulator
- Eye protection (eyedrops, lubricant, eye pads) is important for patients with proptosis
- For maintenance of anesthesia, any of the inhalation anesthetic agent may be used. Nitrous oxide and opioids are safely used in hyperthyroid patients.
- Reversal of muscle relaxants should include glycopyrrolate instead of atropine in combination with an acetylcholinesterase inhibitor to prevent excessive tachycardia
- For the treatment of intraoperative hypotension, direct-acting vasopressor (phenylephrine) is preferred. Ephedrine, epinephrine, norepinephrine, and dopamine are avoided or administered in extremely low doses to prevent exaggerated hemodynamic responses
- **Regional anesthesia for emergency LSCS** is preferred to GA. Preoperatively adequate preloading with IV fluids should be done. Epine-phrine containing local anesthetic solutions should be avoided. Fluids and phenylephrine are used to treat hypotension due to regional anesthesia.
- The $T_{1/2}$ of T_4 is 7 to 8 days; therefore, beta blocker therapy should be continued in the postoperative period.

Thyroid storm and malignant hyperthermia can present with similar signs and symptoms (i.e. hyperpyrexia, tachycardia, hypermetabolism). Thyroid storm is a life-threatening exacerbation of pre-existing hyperthyroid state. Thyroid storm presents with the following signs and symptoms: (1) fever, (2) mental and emotional disturbances, (3) tachycardia, (4) tachypnea, (5) diaphoresis, and (6) diarrhea. Without any treatment, thyroid storm may progress to coma, multiorgan system failure, and death. Precipitating events for thyroid storm during pregnancy include infection, thyroid cancer, normal labor, hemorrhage, cesarean section, and eclampsia.

The management of thyroid storm includes supportive measures like cold sponging, cooled IV fluids, adequate oxygenation and hydration, maintain blood glucose and electrolytes and correction of metabolic acidosis.

Chlorpromazine (25–50 mg IV) or meperidine (25–50 mg IV) to decrease shivering. Glucocorticoids: Dexamethasone (2–4 mg IV) or hydrocortisone (100–300 mg IV).

To reduce synthesis and secretion of thyroid hormone; antithyroid medications: Propylthiouracil (600–1000 mg orally per day) or methimazole (60–100 mg orally or rectally per day).

Iodine: Sodium iodide (1 g IV or Lugol's solution 30 drops orally) or supersaturated potassium iodide solution (SSKI 3 drops orally) to reduce the peripheral conversion of thyroxine (T_4) to 3,5,3-triodothyronine (T_3). Iopanoic acid (telepaque) 3 g orally or sodium ipodate (oragrafin) 1 g orally.

Hypothyroidism

Followings are the various causes of hypothyroidism.

Primary

Autoimmune: Hashimoto's thyroiditis, atropic hypothyroidism.

Iatrogenic: Radioiodine therapy, subtotal thyroidectomy.

Drugs: Iodine deficiency or excess, lithium, amiodarone, antithyroid drugs.

Congenital: Dyshormonogenesis, thyroid gland agenesis/dysgenesis.

Secondary

Pituitary dysfunction: Irradiation, surgery, neoplasm, Sheehan's syndrome, idiopathic.

Hypothalmic dysfunction: Irradiation, granulomatous disease, neoplasm.

Clinical Presentation

The clinical signs and symptoms are: hoarseness of voice, paresthesias, cold intolerance, delayed relaxation of deep tendon reflexes, slow movements, coarse skin and hair, periorbital puffiness, and bradycardia.

During the preanesthetic history and physical examination, the following may suggest the diagnosis of hypothyroidism: (1) a history of neck irradiation or radioiodine therapy (2) the use of lithium, iodine, amiodarone, antithyroid medications, or thyroid replacement medications; and (3) a history of thyroid surgery or surgical scar overlying the site of the thyroid gland.

Thyroid function test shows a decreased serum concentration of free T_4 and a corresponding increase in TSH in patients with primary hypothyroidism.

Interaction with Pregnancy

Pregnant women usually exhibit overt or symptomatic hypothyroidism at a much lower rate than general population. Hypothyroid women have a lower fertility

rate than euthyroid women, which reflects neuroendocrine and ovarian dysfunction. The immunosuppressive effects of pregnancy may lead to a temporary improvement of Hashimoto's thyroiditis during pregnancy.

Effect on Fetus

Hypothyroidism is associated with an increased incidence of the following obstetric complications: anemia, pre-eclampsia, IUGR, placental abruption, postpartum hemorrhage, and fetal distress during labor. Early diagnosis and treatment is associated with improved maternal and fetal well-being.

Medical Management

Hypothyroidism is treated by replacement therapy with oral thyroid hormone. The drug commonly used is levothyroxine, which has a half-life of 7 days.

Anesthetic Consideration

- Patients with mild to moderate disease should receive levothyroxine (100–200 µg/day) in the preoperative period
- Elective surgery is differed until these patients are euthyroid. Decreased myocardial function and ventilatory drive return to normal within 3 to 6 months on levothyroxine 150 µg/day
- If **emergency cesarean section** is necessary, the potential for intraoperative cardiovascular instability and myxedema coma in the postoperative period is high. If surgery can be delayed for 24 to 48 hours, intravenous levothyroxine will be more effective
- Intravenous tri-odothyronine is effective in 6 hours (peak basal metabolic rate in 36-72 hours.) and is used in emergency LSCS. (I-triiodothyronine 25-50 µg intravenously). However acute intravenous replacement therapy carry a significant risk of myocardial ischemia
- Steroid coverage with hydrocortisone or dexamethasone is necessary because of decreased adrenal cortical function often seen in hypothyroidism
- Hypothyroid patients are at high-risk for aspiration due to delayed gastric emptying
- General anesthesia with rapid sequence induction is done. Ketamine is the preferred induction agent as it supports blood pressure and heart rate due to its sympathomimetic activity
- Succinylcholine can be used for rapid sequence induction. For maintenance, nitrous oxide with small doses of short-acting opioid or benzodiazepine or ketamine, and an intermediate-acting nondepolarizing muscle relaxant (vecuronium, rocuronium) can be used. Pancuronium is a preferred muscle relaxant, however, reduced skeletal muscle activity in these patients along with a reduction in hepatic metabolism requires lower doses and close monitoring with peripheral nerve stimulator
- Anticipate difficult intubation as there is airway compromise secondary to a swollen oral cavity, edematous vocal cords, or goitrous enlargement of thyroid gland
- Hypothyroid patients are sensitive to the myocardial depressant effects of the inhalation agents and anesthetic drugs
- The effect of thyroid activity on the MAC of inhalation agents is not clinically significant. However the increased sensitivity is secondary to reduced cardiac output, decreased blood volume, abnormal baroreceptor function, decreased hepatic metabolism, and decreased renal excretion
- Intraoperative hypotension is best treated with ephedrine, dopamine, or epinephrine and not with pure alpha-adrenergic agonist (phenylephrine). Unresponsive hypotension may require supplemental steroid administration
- Decreased ventilatory response to hypoxia and hypercarbia are enhanced by anesthetic agents. Hypothermia occurs quickly and is difficult to treat
- Hematologic abnormalities such as anemia, platelet dysfunction and coagulation factor (especially factor VIII) dysfunction are usually seen
- Electrolyte imbalances (hyponatremia), and hypoglycemia are common and require ABG and electrolyte monitoring perioperatively
- Phosphodiesterase inhibitors such as milrinone is effective in the treatment of reduced myocardial contractility since its mechanism of action does not depend on alpha-receptors, whose number and sensitivity are reduced in hypothyroidism patient
- These patients are very sensitive to narcotics and sedatives. Hence, these drugs are avoided or given in titrated doses in perioperative period
- Reversal is done with neostigmine and glycopyrrolate. Postoperative ventilatory support may be required to manage possible delayed emergence. Postoperative analgesia is best managed with regional techniques or NSAIDs
- Regional anesthesia is recommended if there are no contraindications (e.g. coagulation abnormalities, skin infection at spinal anesthesia site)
- A hypodynamic cardiovascular system may require intra-arterial blood pressure monitoring and a central venous pressure or pulmonary artery (Swan-Ganz) catheter or transesophageal echocardio-

graphy to monitor intravascular volume and cardiac status
- Dextrose in normal saline is the recommended intravenous fluid to avoid hypoglycemia and minimize hyponatremia secondary to impaired free water clearance.

DIABETES MELLITUS

Clinically diabetes mellitus (DM) is characterized by polyuria, polydipsia, and polyphagia with unexplained weight loss. DM results from either a decrease in insulin secretion (type 1 or insulin dependant DM or a resistance to insulin in target tissues (type 2 or noninsulin dependant DM. Gestational DM refers to DM or glucose intolerance that is first diagnosed during pregnancy. Insulin is an important anabolic regulator of carbohydrate, lipid and amino acid metabolism.

Normal hepatic glucose metabolism is a balance between the effects of insulin and several counter-regulatory hormones (e.g. glucagon, cortisol, epinephrine, growth hormone).

The three major acute complications are diabetic ketoacidosis, hyperosmolar hyperglycemic nonketotic coma, and hypoglycemia.

Diabetic ketoacidosis (DKA) occurs with type 1 DM. DKA may occur due to infection, trauma, stress or inadequate insulin dose. There is metabolic acidosis, hyperglycemia, and dehydration secondary to an osmotic diuresis.

Hyperosmolar hyperglycemic nonketotic coma (HHNC) occurs predominantly with type 2 DM. The precipitating medical event is more serious than DKA. The laboratory findings in HHNC are hyperglycemia (often more than 600 mg/dL), hyperosmolarity (greater than 310 mOsm), and moderate azotemia (serum blood urea nitrogen [BUN] of 70–90 mg/dl), without ketonemia or significant acidosis.

Hypoglycemia is common in diabetic patients on insulin therapy. Symptomatic awareness of hypoglycaemia may be inadequate in patients with autonomic neuropathy. Beta blockers should be avoided in diabetic patients for the same reason.

Diagnosis

The random blood sugar is > 200 mg/dl. The fasting plasma glucose is >126 mg/dl.

The two-hour postprandial plasma glucose > 200 mg/dL with oral glucose tolerance test (OGTT) using glucose load of 75 gm of anhydrous glucose dissolved in water.

Gestational DM is associated with (1) advanced maternal age (2) obesity (3) a family history of DM and (4) a history of prior stillbirth, neonatal death, or fetal malformation or macrosomia.

The life of RBC is 100 days hence, glycosylated hemoglobin shows glucose control of 2 to 3 months. The normal range for hemoglobin A_{1C} in nondiabetic pregnant women is 4.1 to 5.9 percent.

Interaction with Pregnancy

In pregnant women maternal insulin requirements increase progressively during the second and third trimester. It decreases with the onset of labor, increases again during the second stage of labor and decreases markedly during the early postpartum period.

There is increasing peripheral resistance to insulin in the second and third trimesters. The presumed mechanism is an increase in counter-regulatory hormones (e.g. placental lactogen, placental growth hormone, cortisol and progesterone) during pregnancy.

Gestational DM develops when a patient cannot mount a sufficient compensatory insulin response during pregnancy. After delivery most patients have a normal glucose tolerance but remain at increased risk for DM (predominantly type 2) in later life and subsequent pregnancies.

At term, the daily insulin requirement typically is about 1.0 IU/kg, compared with 0.7 IU/kg at first trimester.

In nondiabetic parturients, glucose production markedly increases during painful labor. During labor, plasma insulin concentrations remain unchanged except for a brief increase during the third stage of labor and immediately postpartum. These observations suggest that glucose use during labor is largely independent of insulin. In patients with type 1 DM, insulin requirements decrease with the onset of the first stage of labor.

Insulin requirements increase during the second stage of labor, the mechanism is unknown. The use of epidural analgesia or oxytocin does not affect exogenous insulin requirements during the first and second stages of labor. After delivery (either vaginal or caesarean) insulin requirements in patients with type 1 DM decrease markedly for at least several days. The decreased insulin requirement results from the loss of counter-regulatory hormones of the placenta.[29] The insulin requirements gradually return to pre pregnancy levels within several weeks of delivery in patients with type 1 DM.

Despite improved obstetric and medical care, maternal mortality is 10 times higher in diabetic patients than in nondiabetic patients.

DKA occurs almost exclusively in type 1 DM. DKA results in an increased perinatal mortality rate. DKA occurs commonly during the second and third trimester. It is associated with the following factors:

- Beta-agonist therapy
- Vomiting
- Decreased caloric intake
- Poor medical management
- Patient noncompliance
- Infection.

There is fetal distress during episodes of maternal DKA. FHR responses normalized and preterm uterine contractions stop after adequate management of maternal DKA. Maternal metabolic acidosis decreases uterine blood flow.

The management of DKA includes the following measures:

- Intravenous hydration
- Intravenous insulin
- Treatment of the underlying cause of DKA
- Monitoring of serum glucose and electrolytes
- Restriction of bicarbonate therapy to severe acidosis
- In addition, left uterine displacement should be maintained
- Supplemental oxygen should be administered.

Effect of Diabetes on Mother and Fetus

Both pregestational and gestational DM is associated with an increased incidence of PIH, polyhydramnios, and cesarean section. In women with type 1 DM, the risk of pre-eclampsia is increased with severity of diabetes. Proteinuria in early pregnancy is associated with an increased risk of adverse outcome. Pregnancy is an independent risk factor for diabetic retinopathy.[30]

Pregnancy does not accelerate the progression of diabetic nephropathy.

It is unclear whether pregnancy accelerates the progression of somatic or autonomic neuropathy in diabetic patients. Myocardial infarction is a rare complication in pregnant diabetics.

Fetal complications of maternal DM during pregnancy are as follows:

- Fetal macrosomia: Excessive birth weight >90 percent for gestational age or as a birth weight >4000 g
- Shoulder dystocia, birth injury/trauma
- Structural malformations
- CNS: Anencephaly, encephalocele, meningomyelocele, spina bifida, holoprosencephaly
- CVS: Transposition of great vessels, VSD, situs inversus, single ventricle, hypoplastic left ventricle.
- Skeletal: Caudal regression
- Renal: Agenesis, cystic kidney, ureter duplex
- Gastrointestinal: Anal/rectal atresia, small left colon
- Pulmonary: Hypoplasia
- Other complications: Intrauterine/neonatal death, neonatal respiratory distress syndrome, neonatal hypoglycemia, neonatal hyperbilirubinemia, neonatal hypocalcemia (calcium level at or below 7 mg/dL) and neonatal polycythemia (venous hematocrit that exceeds 65%)[31]
- Postdelivery: Glucose intolerance.

Obstetric Management

Frequently determine the capillary blood glucose concentration with timely adjustments in diet and insulin therapy. The target glucose concentration in patients with pregestational DM is a fasting blood glucose concentration of 60 to 95 mg/dL. The long-term metabolic control of diabetes is best monitored by measuring glycosylated hemoglobin (Hb_{A1c}).

Regular insulin (crystalline zinc insulin) is a fast-acting preparation and is the only insulin that can be administered intravenously as well as subcutaneously. Hence, regular insulin is used for perioperative treatment of DM.

As insulin requirements decrease abruptly at delivery, it is important to modify the insulin doses administered, 24 hours before delivery to avoid maternal postpartum hypoglycemia.

Diet and exercise are the initial therapeutic approaches for glycemic control in patients with gestational DM. Insulin therapy is started if the fasting glucose exceeds a threshold of 80 to 105 mg/dl. In general, oral hypoglycemic agents are not used during pregnancy. These agents cross the placenta, and there is concern of fetal hyperinsulinemia and they may be teratogenic.[32]

Anesthetic Consideration

- The preanesthetic evaluation of the patient with DM should include a history and physical examination to identify acute and chronic complications of DM
- The diabetic parturient should be evaluated for adequate temporomandibular joint and cervical spine mobility to anticipate difficult intubation
- The effects of chronic hyperglycemia (coronary artery disease, myocardial infarction, congestive heart failure, peripheral vascular disease, hypertension, cerebrovascular accident, chronic renal failure, infection and neuropathy) are frequently

present on preoperative evaluation and should be medically optimized before surgery if time permits
- Acute hyperglycemia causes dehydration, impaired wound healing, an increased rate of infection, worsening central nervous system/spinal cord injury with ischemia, and hyperviscosity with thrombogenesis
- Tight control of serum glucose in the perioperative period is important in managing the consequences of acute and chronic hyperglycemia
- A high index of suspicion should exist for myocardial ischemia and infarction. Silent ischemia is possible if an autonomic neuropathy is present
- For renal disease, adequate control of hypertension is important. Meticulous attention to hydration status, avoiding nephrotoxins, and preserving renal blood flow is essential. Cardiovascular autonomic dysfunction predisposes the patient to perioperative dysrhythmias and intraoperative hypotension. There is loss of compensatory sympathetic response to intraoperative hypotension. Hence, these patients need meticulous haemodynamic monitoring with adequate hydration and vasopressors to mantain blood pressure[33]
- Gastroparesis: A manifestation of autonomic neuropathy causes increased risk of gastric regurgitation and pulmonary aspiration in diabetic parturient. Premedication with metoclopramide (10 mg intravenously) minimize the risk of aspiration secondary to diabetic gastroparesis
- The diabetic **stiff-joint syndrome or diabetic scleroderma** may cause difficult laryngoscopy and intubation in patients with DM. This syndrome occurs in patients with long-standing type 1 DM and is associated with nonfamilial short stature, joint contractures, and tight skin. Limited movement of the atlanto-occipital joint may result in difficult direct laryngoscopy and intubation. Preoperative evaluation should focus on limited joint mobility of the neck from nonenzymatic glycosylation of proteins and abnormal cross-linking of collagen. There is firm, woody, nonpitting edema of the posterior neck and upper back. Thus, there is limited range of neck movement. Also it makes the epidural spaces noncompliant and this makes regional anesthesia difficult
- Perioperative peripheral nerve injuries are common in diabetic patients. It is important to perform a brief neurological examination and documentation of peripheral nerve injuries. There should be proper perioperative positioning and padding of the extremities to prevent further nerve injuries
- Intraoperative Management: Serum glucose should be monitored periodically and IV insulin given according to sliding scale to mantain blood glucose in normal range (80–100 mg/dL)
- Avoidance of hypoglycemia is very important as its recognition is masked by anesthetic drugs, sedatives, analgesics, β-blockers, sympatholytics and an autonomic neuropathy
- Neonatal acidosis does not occur during **spinal** or **epidural anesthesia** for cesarean section in diabetic parturients if: (1) maternal glycemic control is satisfactory, (2) the patient receives aggressive preanesthetic volume expansion with balanced salt solution (3) hypotension is treated aggressively with ephedrine
- Epidural anesthesia is preferable to spinal anesthesia as it causes slower onset of sympathetic blockade and hypotension. The dose and volume of epidural anesthetic drugs should be reduced and repeat doses should be titrated carefully in order to prevent high sympathetic blockade and inadvertent hypotension.

VIRAL HEPATITIS

Acute viral hepatitis is the most common cause of jaundice during pregnancy. It is one of the most serious infections of pregnant women. More than 80 percent cases of viral hepatitis are caused by hepatitis viruses A, B or C; hepatitis viruses D, E and G also have been reported.

Hepatitis A Virus (HAV): This RNA virus is responsible for nearly half of viral hepatitis cases. This virus replicates in the liver, is secreted in the bile, and is then shed through feces. Transmission occurs almost exclusively through fecal-oral contamination. Occasionally, it is spread through intravenous drug use or sexual contact. Rare cases of vertical transmission have been reported. Clinical illness commonly is mild and limited to 2 or 3 weeks, It is never associated with a chronic carrier state. Fulminant hepatic necrosis is a rare but devastating complication of HAV infection.[34]

Hepatitis B Virus (HBV): This DNA virus is transmitted through parenteral or sexual exposure. In contrast to HAV infection, 5 to 10 percent of those infected with HBV progress to a chronic carrier state. Thirty percent of the chronic carrier persons continue to have active hepatocellular destruction and are at risk of cirrhosis, hepatocellular carcinoma and death. Vertical transmission of active HBV infection to the newborn is common in chronic HBV hepatitis. When the mother is chronic HBsAg carrier and positive for viral DNA in serum, the risk of neonate becoming chronic carrier is 80 to 90 percent. If the mother is negative for viral DNA in serum, the transmission rate is about 10 to 30 percent.[35]

Hepatitis C Virus (HCV): This virus is responsible for most cases of non-A, non-B viral hepatitis and is predominantly transmitted through parenteral, sexual, nasal, and intrauterine routes. Most infections with HCV are asymptomatic. Nevertheless, up to 90 percent of infected individuals slowly develop cirrhosis, hepatocellular carcinoma, hepatic failure, and death over two decades or longer.[36] Women of reproductive age are at particular risk for unrecognized, asymptomatic HCV infection. Approximately 7 to 8 percent HCV positive women transmit the virus to their offsprings. A higher rate of incidence is seen in HIV coinfected women.[37]

Hepatitis D Virus (HDV or delta agent): It is an incomplete RNA virus that is dependent on coinfection with HBV. Chronic HDV infection carries an increased risk of fulminant hepatic failure. Although vertical transmission of HDV does occur, measures that protect against HBV infection also protect the neonate against HDV infection.

Hepatitis E Virus (HEV): This RNA virus is transmitted by the fecal-oral route and is responsible for major epidemics of viral hepatitis in developing countries. In nonpregnant ladies, HEV infection typically is self-limited and does not lead to chronic carrier state. However, HEV infection during pregnancy is associated with a high-risk of maternal and fetal morbidity and mortality.[38] Vertical transmission of this virus has been reported.

Hepatitis G Virus (HGV; also called **hepatitis GB virus C** or **HGBV-C):** This RNA virus is closely related to the HCV virus. Vertical transmission of HGV from mother to infant has been reported. However, the clinical significance is unknown, and no causal relation between HGV and hepatitis has been seen.

Effect on the Fetus

Preterm labor and delivery is more common due to viral hepatitis. Vertical transmission of HBV from mother to fetus is a significant factor.[39] The vertical transmission rate for HCV is significantly less than that for HBV infection. Hepatitis E viral infection is associated with a very high-risk of maternal and fetal morbidity and mortality.

Diagnosis

Clinical symptoms range from vague constitutional symptoms (e.g. malaise, nausea, anorexia) to overt jaundice. Physical examination often reveals tender hepatomegaly. Bilirubinuria and acholic stool are noted. Hepatic transaminase enzymes rise (>1000 IU range) in acute infection. Serologic testing confirms the diagnosis of individual virus infection.

Medical and Obstetric Management involves supportive care and prevention of newborn transmission. Vigorous medical management is indicated for severe nausea and vomiting, hypoglycemia, encephalopathy, coagulopathy, hepatorenal shut down.[40] HBV vaccination for all newborns, regardless of maternal serology is also recommended. If the mother is HBsAg positive or has unknown serology, initial vaccination should be performed within 12 hours of delivery and hepatitis B immune globulin (HBIg) should be administered.

Intrahepatic Cholestasis of Pregnancy

It is the second most common cause of jaundice during pregnancy. It commonly occurs in the third trimester. Nulliparous women with multiple gestation are at high-risk. The disorder resolves promptly after delivery. It commonly recurs in subsequent pregnancies or with the administration of estrogenic oral contraceptives. The specific etiology is unknown; however, it seems to represent an enhanced sensitivity to the cholestatic effects of estrogenic steroids.[41]

Effect on the Mother and Fetus

Intrahepatic cholestasis of pregnancy has minimal impact on maternal health during gestation. Vitamin K malabsorption, if uncorrected, may lead to clinical coagulopathy. There is an increased incidence of postpartum hemorrhage in women with intrahepatic cholestasis of pregnancy.

The fetus is at increased risk of spontaneous preterm labor, meconium-stained amniotic fluid and fetal distress. Neonatal hypoprothrombinemia places the infant at increased risk of intracranial hemorrhage.

Medical and Obstetric Management

It is directed toward improving bile secretion and reducing intestinal reabsorption of bile salts to provide symptomatic relief.[42]

Acute Fatty Liver of Pregnancy

Idiopathic acute fatty liver of pregnancy (AFLP) or reversible peripartum liver failure is an uncommon disorder of late pregnancy. It is characterized by impaired hepatic metabolic activity, which may progress to liver failure, disseminated intravascular coagulation (DIC), profoundly depressed antithrombin III level, hypoglycemia, and renal insufficiency (is it correct). The liver failure resolves shortly after delivery. Histologic examination of the liver reveals microvesicular fatty infiltration of the liver cells.[43] Similarities between AFLP and pre-eclampsia or eclampsia is

striking. Both disorders primarily occur near term and are associated with nulliparity and multiple gestation. Hepatic involvement occurs with pre-eclampsia in the form of the HELLP syndrome (i.e. Hemolysis, Elevated Liver enzymes, and Low Platelets). AFLP may represent a severe variant on the pathologic continuum of pre-eclampsia/eclampsia.[44]

The symptoms are nausea and vomiting, malaise, abdominal pain, fever, jaundice or dark urine, headache, pruritus and sore throat. Pruritus is uncommon. Hepatic encephalopathy is a late and ominous finding.

Effect on the Mother and Fetus

With improved medical management, maternal and fetal mortality rates have decreased. Maternal complications may include DIC, profound hypoglycemia, hepatic encephalopathy, pancreatitis, acute renal failure and hepatic rupture. Fetal distress and death may occur secondary to uteroplacental insufficiency.[45]

AFLP is a medical emergency that demands rapid evaluation and treatment. Hepatic failure and fetal death may develop within a few days. Supportive care includes fluid and electrolyte support, treatment of hypoglycemia, and attention to coagulopathy and anemia.[46] The maternal glucose level should be monitored and dextrose infusions administered.

Portal Hypertension

It is uncommon during pregnancy. Clinical manifestations include the development of portal-systemic collaterals, splenomegaly and ascites. The portal-systemic collaterals within the gastric and esophageal mucosae are common sites for hemorrhage.

During pregnancy, uterine compression of the inferior vena cava causes diversion of venous return through the azygos and vertebral venous systems.

Medical and Obstetric Management

Endoscopic sclerotherapy is the preferred management of variceal hemorrhage during pregnancy. If uncontrolled hemorrhage occurs despite sclerotherapy, portosystemic shunting may be considered.

Anesthetic Consideration

It depends on the extent of hepatic impairment. The woman with inactive viral hepatitis, mild intrahepatic cholestasis of pregnancy, can be managed similar to healthy parturient, provided synthetic and metabolic function of liver are intact. Coagulopathy should be excluded or corrected before giving regional anesthesia. In contrast, the parturient with acute viral hepatitis, advanced cirrhosis, portal hypertension, or AFLP presents many challenges to the anesthesiologist.

Systemic Abnormalities Associated with Hepatic Disease

There is impaired synthesis of clotting factors I, II, V, VII and X. Cholestasis leads to malabsorption of vitamin K, an important cofactor required for the synthesis of factors II, VII, IX and X. The plasma half-life of factor VII is 5 hours thus, coagulopathy may develop rapidly. Vitamin K administration corrects coagulopathy if malabsorption is the primary cause. However, impaired hepatic synthesis does not respond to vitamin K administration. Fresh frozen plasma or cryoprecipitate may be needed if clinical bleeding develops.[47]

Cardiovascular manifestations include increased cardiac output and low systemic vascular resistance (SVR) due to extensive arteriovenous shunting. Cardiomyopathy may develop. Central venous or pulmonary artery pressure monitoring should be considered in those patients with ascites or cardiomyopathy.

Hypoxemia impaired hypoxic pulmonary vasoconstriction and portopulmonary venous communication lead to significant hypoxemia. Ascites and the gravid uterus cause diaphragmatic elevation, decrease functional residual capacity, and lead to further intrapulmonary shunting. Patients with liver disease have increased 2,3-diphosphoglycerate levels in red blood cells and a right shift of the oxyhemoglobin dissociation curve.

Hepatic encephalopathy is a reversible neuropsychiatric disorder that occurs with advanced hepatic failure. Inadequate hepatic clearance of ammonia and mercaptan toxins, altered gamma-amino-butyric acid levels cause this condition. Impairments can range from mild confusion to coma. These patients are at risk for pulmonary aspiration of gastric contents.[48] The integrity of the blood-brain barrier is altered, hence anesthetic agents should be titrated carefully.

Metabolic abnormalities include hypoglycemia, hyponatremia, hypokalemia, and acid-base disturbances. Hypoglycemia is due to impaired hepatic gluconeogenesis and glycogenolysis (common in AFLP).[49] Blood glucose levels should be monitored frequently in these patients.

Albumin is synthesized exclusively by the liver. Its half-life is approximately 15 days. Acute hepatic failure is associated with increased plasma triglyceride and abnormal lipoprotein concentrations. These metabolic changes may be sensitive markers for hepatic function.

Increased renal sodium retention often accompanies hepatic disease and contributes to ascites formation. The pathogenesis involves increased plasma aldosterone, increased sympathetic activity, altered renal prostaglandins and kinins and reduced renal blood flow. Overt oliguric renal failure may occur, which heralds the onset of the **hepatorenal syndrome**. Renal vasoconstriction along with central hypovolemia is primarily responsible for this syndrome. The prognosis for these patients is poor.

Effect of Anesthesia on Hepatic Blood Flow and Oxygenation

Failure to maintain hepatic blood flow or oxygenation may lead to further hepatic necrosis and decompensation in a parturient with compromised hepatic function. Isoflurane is the inhalational agent of choice as it has the least effect on hepatic blood flow. Factors that reduce hepatic blood flow such as hypotension, excessive sympathetic activation and high mean airway pressure during controlled ventilation should be avoided in hepatic diseases. It is wise to avoid halothane as its metabolic products are hepatotoxic. Enflurane, isoflurane, and desflurane can form trifluoroacetyl metabolites resulting in cross-sensitivity with halothane. However, the degree of anesthetic metabolism is substantially less. Sevoflurane does not undergo metabolism to trifluoroacetylated metabolites, hence, sevoflurane does not produce immune-mediated hepatotoxicity. There is no indisputable proof that general anesthesia of any type causes postoperative hepatic dysfunction.

Anesthetic Consideration

Regional Anesthesia for Emergency LSCS

- Regional anesthesia also may reduce hepatic blood flow due to sympathetic blockade and associated central hypovolemia. Thus, judicious hydration, epidural anesthesia, and control of BP with intravenous fluids and inotropes should prevent clinically significant reduction in hepatic blood flow.
- In the absence of coagulopathy, regional anesthesia can be used. In patients with cirrhosis, the half-life of lignocaine is increased almost three times (from 108-296 minutes), and the volume of distribution is increased from 1.3 to 2.3 L/kg. An expanded volume of distribution offers some protection against toxicity despite impaired clearance.
- Ascites and portal hypertension lead to engorged epidural veins; hence, the use of a test dose to exclude intravascular injection is essential. On theoretic basis, spinal anesthesia is safer than epidural anesthesia because of less drug requirement. However, spinal anesthesia can cause profound sympathetic blockade and hypotension.

General Anesthesia for Emergency LSCS

- Coagulopathy, obstetric hemorrhage, fetal distress, or altered sensorium may necessitate the use of general anesthesia for cesarean section
- These patients are prone to pulmonary aspiration. Premedication with IV ranitine and metoclopramide and rapid sequence induction is the technique of choice
- Intravascular volume should be monitored with a central line and adequate hydration maintained. In the presence of ascites or cardiovascular compromise, arterial and central venous pressure monitoring may be helpful. Large-gauge intravenous access should be established
- Blood glucose must be monitored and dextrose containing IV fluids given to prevent hypoglycemia.
- Patients with bleeding esophageal varices should have awake intubation. Nasogastric suction is contraindicated
- Rapid-sequence induction is done with thiopental, propofol, ketamine or etomidate, depending on the patient's hemodynamic status. Reduced pseudocholinesterase levels may delay succinylcholine metabolism, but this is clinically insignificant. Thus, succinylcholine remains the muscle relaxant of choice for rapid-sequence induction and should be given in the same bolus dose as for healthy parturients
- Airway trauma must be avoided because of bleeding tendency. Deep neuromuscular blockade facilitates atraumatic tracheal intubation
- Muscle paralysis can be maintained with atracurium or cisatracurium. Anesthesia is maintained with isoflurane and nitrous oxide. Adequate reversal of neuromuscular blockade must be done before extubation
- The clearance of opioids is delayed in patients with severe liver disease hence they must be used cautiously.

RENAL DISEASES IN PREGNANCY

Two groups of renal disorders are: 1) **Glomerulopathies** and 2) **Tubulointerstitial disease**.

Glomerulopathies are further subdivided into disorders that involve inflammatory or necrotizing

lesions—the **nephritic syndromes,** and disorders that involve abnormal permeability to protein and other macromolecules the—**nephrotic syndromes**.

- Blood pressure measurement to moniter the degree of hypertension and effectiveness of treatment
- Urinalysis for the presence of renal casts and bacteriuria
- Serum creatinine and BUN concentrations defines the extent of renal regurgitation. A serum creatinine concentration greater than 0.8 mg/dl although normal in the nonpregnant patient, may represent significant renal insufficiency during pregnancy
- Complete biochemical evaluation for proteinuria, hematuria, or azotemia
- Renal biopsy is done only when sudden deterioration in renal function occurs. It is not practical during emergency surgery.

Effect on Fetus

When renal disease has progressed to end-stage renal failure (i.e. the GFR is less than 5 ml/min) dialysis may be required. Fetal complications of dialysis include IUGR fetal death, and preterm labor. BUN levels are kept below 80 mg/dl predialysis and 30 mg/dl postdialysis.

At birth, neonatal azotemia is similar to that of the mother.

Effect on Pregnant Patients: Chronic renal failure causes many systemic changes, which are enumerated below:

- Metabolic and Endocrine: Hyperkalemia, metabolic acidosis, hyponatremia, hypocalcemia, hypermagnesemia, hypoglycemia and decreased protein binding of drugs
- Hematologic: Anemia, platelet dysfunction, decreased coagulation factors and leukocyte dysfunction
- Neurologic: Autonomic neuropathy, mental status changes, peripheral neuropathy, restless leg syndrome and seizure disorders
- Gastrointestinal: Delayed gastric emptying, increased gastric acidity, hepatic venous congestion, hepatitis (viral or drug) and malnutrition
- Pulmonary: Increased risk of difficult airway and recurrent pulmonary infections
- Cardiovascular: Hypertension, fluidoverload, ventricular hypertrophy, accelerated atherosclerosis, uremic pericarditis and uremic cardiomyopathy.[50]

Anesthetic Consideration

- The anesthetic management depends on the extent of renal dysfunction and hypertension. The parturient with stable renal disease, mild-to-moderate renal insufficiency, well-controlled hypertension, and euvolemia requires minimal special consideration. In contrast, patient with end-stage renal failure presents many anesthetic challenges
- Poorly controlled hypertension leads to left ventricular hypertrophy and dysfunction and may require echocardiography
- The drugs which are completely excreted by the kidneys are: gallamine, metocurine, digoxin, inotropes, aminoglycosides, vancomycin, cephalosporin and penicillin. These drugs should be avoided
- The drugs which are partially excreated by the kidneys are: barbiturates, pancuronium, vcuronium, neostigmine, edrophonium, atropine, glycopyrollate, milrinone, hydralazine and sulphonamides. They should be used judiciously
- Preoperative evaluation includes consideration of concomitant drug therapy and evaluation of the changes which are characteristic of chronic renal failure
- The need for right atrial (central venous pressure) or pulmonary artery (pulmonary artery occlusion pressure) pressure monitoring will depend on associated cardiac disease or pulmonary edema. Strict asepsis must be maintained when taking these lines as these patients are extremely prone to infection
- These patients are prone to uremia-induced delayed gastric emptying and to be dealt accordingly. Premedication with IV H_2-receptor antagonist (ranitidine 50 mg) and metoclopramide 10 mg intravenously can prevent acid aspiration syndrome.[51]
- Hemodialysis fistulae should be padded carefully to prevent thrombosis perioperatively. Blood pressure cuffs should not be placed on these extremities. Similarly IV line should not be taken on the limb having an AV-fistula. The arterial pressure or the arterial blood gases will not be accurate if the cannula is placed in the same limb with a functioning or partially patent fistula
- As diabetes is common in these patients, glucose management is important
- Antihypertensive therapy should be continued preoperatively for adequate control of BP. Beta-blockers, calcium channel blockers, hydralazine are commonly used. Clonidine is occasionally useful in refractory hypertension. Doxazosin and prazosin are used if required. ACE inhibitors are contraindicated. (they cause fetal and neonatal oliguria-anuria, malformations like microcephaly and encephalocele)

- These patients have increased sensitivity to central nervous system depressant drugs hence, they should, be used judiciously
- Preservation of renal function depends on maintaining an adequate intravascular fluid volume and minimizing drug-induced cardiovascular depression
- Patients on hemodialysis should undergo dialysis during the 24 hours preceeding elective surgery. A common recommendation is that the serum potassium concentration should not exceed 5.5 mEq/L on the day of surgery
- Anemia is evaluated preoperatively and managed accordingly with hematinics or blood transfusion. The recombinant human erythropoietin therapy has decreased the severity of anemia in CRF patients. Patients on ACE inhibitors may be at increased risk of hypotension with acute blood loss.
- The sympathetic nervous system function is attenuated by uremia. This impairs compensatory peripheral vasoconstriction. Positive-pressure ventilation of patient's lungs, abrupt changes in body position, or drug-induced myocardial depression can cause an exaggerated fall in systemic blood pressure even with minimal blood volume loss
- There is uremia-induced disruption of the blood-brain barrier. The decreased plasma proteins result in more free drugs at receptor sites thus, aggravating their action. Therefore, hypnotic and sedative agents should be use in titrated doses
- Induction of anesthesia and tracheal intubation can be safely done with intravenous drugs like propofol, etomidate, or thiopentone. However, decreased blood albumin level leads to increased concentration of free thiopentone causing profound effect. Protein binding of propofol is unaffected by renal failure. These changes may require a decrease in the dose of thiopental or propofol for induction
- Muscle relaxants: Renal disease may slow the excretion of pancuronium, vecuronium and rocuronium, whereas clearance of mivacurium, atracurium, and cisatracurium is independent of renal function
- Atracurium and cisatracurium undergo Hoffmann degradation and nonspecific esterase hydrolysis, hence are safely used in renal failure.[52] It is wise to decrease the initial dose of the relaxant drug and give subsequent doses according to peripheral nerve stimulator response. Magnesium-containing antacids may cause hypermagnesemia, which potentiates neuromuscular blockade
- Succinylcholine will cause a 0.5 to 0.7 mEq/L increase in potassium concentration, which is similar to the rise seen in normal persons. If the patient is already hyperkalemic, this mild elevation may be sufficient to precipitate cardiac arrhythmias, hence should be avoided. Plasma cholinesterase concentrations are normal, even after dialysis
- Anesthesia is maintained with nitrous oxide combined with isoflurane, desflurane and short-acting opioids. Sevoflurane may be avoided because of concerns related to fluoride nephrotoxicity or production of compound A, although there is no evidence that patients with renal disease are at increased risk of renal dysfunction
- Total intravenous anesthesia with remifentanil, propofol, and cisatracurium has been recommended for patients with end-stage renal failure
- Potent inhalational agents are used to control intraoperative hypertension. Maintain adequate BP as the associated anemia will jeopardize oxygen delivery to peripheral tissues. Opioids decrease the likelihood of cardiovascular depression and avoid the concern of hepatotoxicity or nephrotoxicity
- Fluid management: Patients with severe renal dysfunction but not on hemodialysis may benefit from preoperative hydration with normal saline. Lactated Ringer's solution or other potassium-containing fluids should not be used. Administration of normal saline to maintain adequate CVP and urine output is recommended.
- Stimulation of urine output with osmotic (mannitol) or tubular (furosemide) diuretics without adequate intravascular fluid volume replacement will cause further intravascular volume contraction. Altough mannitol or furosemide predictably increases urine output, there is no evidence of corresponding improvements in the GFR
- Patients dependent on hemodialysis require special attention to perioperative fluid management as the safety margin between insufficient and excessive intravascular fluid is narrow
- Blood transfusions may be considered if anemia is severe or blood loss is excessive. Central venous pressure monitoring is useful for fluid replacement
- Renal excretion accounts for approximately 50 percent clearance of neostigmine. As a result, the elimination half-time of these drug is prolonged in renal failure but the volume of distribution remains the same and standard doses are used for reversal of neuromuscular blockade

- Correction of factors like nephrotoxic antibiotics, metabolic acidosis, electrolyte imbalance, diuretics should be considered when neuromuscular blockade persists or reappears in patients with renal dysfunction
- Continuous ECG monitoring is helpful for detecting cardiac dysrhythmias due to hyperkalemia
- Provide supplemental oxygen in postoperative period as these patients are normally anemic. Patient may require hemodialysis in the postoperative period.

Regional Anesthesia for LSCS

The factors to be considered for regional anesthesia (spinal and epidural):

- Uremic toxins cause functional platelet dysfunction and a prolonged bleeding time. Thrombocytopenia may occur because of increased peripheral destruction of platelets
- Adequacy of coagulation should be checked by platelet count, BT, CT, PT, INR. The presence of uremic neuropathies should be excluded to avoid medicolegal suits
- Uremic patients are usually hypovolemic. Hypovolemia and autonomic neuropathy may lead to profound hypotension during regional anesthesia. This can be minimized by adequate prehydration and epidural anesthesia
- Coexisting metabolic acidosis may decrease the seizure threshold for local anesthetics hence must be corrected.

Acute Renal Failure

Acute renal failure (ARF) is a rapid deterioration of renal function leading to an accumulation of fluid and nitrogenous waste products with impaired electrolyte regulation. ARF is an uncommon but serious complication of pregnancy.

Causes of Acute Renal Failure in Pregnancy

Prerenal: Hyperemesis gravidarum, uterine hemorrhage.

Intrarenal: Acute tubular necrosis, septic abortion, amniotic fluid embolism, acute glomerulonephritis, drug-induced acute interstitial nephritis, bilateral renal cortical necrosis.

Postrenal: Urolithiasis, ureteral obstruction by gravid uterus, acute pyelonephritis, pre-eclampsia, eclampsia, HELLP syndrome and acute fatty liver of pregnancy.

ARF is also associated with **HELLP syndrome** (Hemolysis, Elevated Liver enzymes, and Low Platelets). The majority of patients had a derangement of multiple organ systems and other obstetric complications (e.g. placental abruption, intrauterine fetal death, disseminated intravascular coagulation DIC, postpartum hemorrhage and sepsis). Renal histology reveals thrombotic microangiopathy and acute tubular necrosis, suggesting pathogenesis of acute renal failure associated with HELLP syndrome.

Effect on the mother and fetus: There is an increase in both maternal and fetal mortality. The prognosis for the fetus is worse than that for the mother, and more than 40 percent of these pregnancies end in fetal death.

Medical and obstetric management: Many obstetric causes of ARF are associated with DIC, therefore, coagulation abnormalities should be excluded.

As urea and other metabolic products cross the placenta, hemodialysis or peritoneal dialysis should be done to maintain the postdialysis BUN concentration below 30 mg/dl. The pediatrician must be alerted to the presence of high fetal BUN levels which may lead to osmotic diuresis and neonatal dehydration.

Anesthetic Consideration for LSCS

- Optimize the patient's condition. Hypovolemia, hypotension, and low cardiac output should be corrected by fluid resuscitation and vasopressor therapy.
- Regional anesthesia can be administered after ruling out coagulopathy, thrombocytopenia, or hypovolemia. As the sympathetic blockade dissipates, the mother should be monitored for evidence of volume overload and pulmonary edema.
- General anesthesia may be required for urgent cesarean delivery or in patients with coagulopathy or hemorrhage. The drug dosing is according to the creatinine clearance since elimination of drugs by the kidneys is proportional to the GFR.
- The supportive measures for ARF include maintenance of an adequate mean systemic blood pressure and cardiac output and the avoidance of further renal insults by hypotension, hypovolemia, hypoxia, and nephrotoxic drugs. Meticulous hemodynamic monitoring, periodic blood gas analysis and electrolyte measurement help in patient management
- The anesthetic drug management for CRF can be followed for ARF parturient.

MALARIA

There are four species of *Plasmodium* that cause human malaria: *vivax, ovale, malariae,* and *falciparum.* The

disease is characterized by high grade fever and flu-like symptoms, including chills, headaches, myalgia, which may occur at periodic intervals. Symptoms are less severe with recurrences. Malaria may be associated with anemia and jaundice and falciparum infections may cause kidney failure, coma and death.

Effects on Pregnancy

Malarial episodes increase significantly during the later two trimesters of pregnancy and two months postpartum. Pregnancy enhances the severity of falciparum malaria, especially in nonimmune primipara. The incidence of abortion and preterm labor is increased with malaria. Increased fetal loss can be related to placental and fetal infection. In nonimmune women, congenital malaria may develop in up to 7 percent of neonates. In heavily endemic (hyper- and holoendemic) areas, falciparum malaria is associated with low birth weight (average reduction, ~170 g) and increased infant and childhood mortality.

The infected mothers in areas of stable transmission remain asymptomatic despite intense accumulation of parasitized erythrocytes in the placental microcirculation.

Maternal HIV infection predisposes pregnant women to malaria, predisposes their newborns to congenital malarial infection, and exacerbates the reduction in birth weight associated with malaria.

In areas with unstable transmission of malaria, pregnant women are prone to severe infections with anemia, hypoglycemia, and acute pulmonary edema. Fetal distress, premature labor, and stillbirth or low birth weight and fetal death are common results.[53]

Treatment: Commonly used antimalarial drugs are not contraindicated during pregnancy. Chloroquine is the treatment of choice for malaria caused by Plasmodium species sensitive to the drug. For chloroquine-resistant infection, mefloquine is given orally. At high doses, mefloquine is teratogenic and embryotoxic in some laboratory animals. However, limited data indicate the drug to be safe and effective in pregnancy.

For severe or complicated malaria, quinine is given parenterally**.** Its main side effect is hypoglycemia and cardiac dysrhythmias. Artemisinin and its derivatives have been used in a limited number of cases in the second half of pregnancy with success and without apparent complications.

Anesthetic Consideration

- The anesthetic management is on similar lines as anemic patient.
- The renal function may be compromised because of massive hemolysis and hemoglobinuria.
- The mortality and morbidity is high.

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

HIV, previously known as lymphadenopathy-associated virus (LAV) and human T-cell lymphotropic virus type III (HTLV-III), is a member of the lentivirus subfamily of human retroviruses. As a retrovirus, HIV carries the enzyme reverse transcriptase. This enzyme converts the single-stranded viral RNA into double-stranded DNA, which subsequently is integrated into the DNA of the infected cell. For infection of the host cell to occur, HIV must bind to a cell-surface receptor, the CD4 antigen complex. It is found on placental cells and may provide a route of vertical transmission to the fetus during early pregnancy. Infection of helper T-cells causes immune suppression in HIV disease. This makes the HIV patient vulnerable to bacterial, viral, fungal, parasitic, and mycobacterial infection.

Diagnosis

Commonly, the diagnosis is made on the basis of one of two antibody detection tests—either enzyme immunoassay (EIA) or the Western blot test.

Quantitative PCR (polymerase chain reaction) techniques, through the amplification of viral RNA, can detect very low levels of infection. This technique can detect viremia as early as two weeks after exposure, during the period of primary symptomatic infection. [54]

There may be an interval of several weeks to months after the initial infection and before detectable levels of antibody are present. A patient infected with HIV, who is tested during this window period will have a negative test result but will be fully capable of infecting others. Hence, universal precautions must be taken.

Clinical Features

The effect of HIV on various systems is described below:

- The neurologic manifestations of HIV infections:
- Early (initial infection): Headache, photophobia, aseptic meningoencephalitis, cognitive changes (depression, irritability) cranial and peripheral neuropathy
- Latent phase: Demyelinating neuropathy, CSF abnormalities (HIV antibody, HIV particles, viral nucleic acid)
- Late (clinical aids): Meningitis, diffuse encephalopathy, focal brain lesions, myelopathy (segmental or diffuse), peripheral neuropathy and myopathy

- Autonomic neuropathy: It can present with mild postural hypotension or severe cardiovascular instability during invasive procedures
- An inflammatory myopathy resembling dermatomyositis has been reported
- Pulmonary abnormalities are commonly caused by the opportunistic infections. The most common of these is *P. carinii,* a fungal organism. *Pneumocystis carinii* pneumonia (PCP) typically is seen with severe immune suppression. The clinical picture is similar to that of adult respiratory distress syndrome (ARDS) with severe hypoxemia and diffuse interstitial infiltrates on chest radiograph. The mortality rate may be as high as 75 percent. Patients who survive the disease are at risk for the development of pneumatoceles; subsequent rupture leading to pneumothorax is common
- Reactivation of latent tuberculosis is common with HIV infection because of the impairment of cellular immunity. There is an increased incidence of bacterial pneumonia caused by encapsulated organisms (e.g. *Streptococcus pneumoniae, Haemophilus influenzae*), pneumonia secondary to other fungal organisms (e.g. aspergillus, cryptococcus, coccidioides) is common in HIV patients
- Gastrointestinal Abnormalities: Painful or difficult swallowing is caused by herpetic, CMV or candida **esophagitis**. **Severe diarrhea** resulting from infection with CMV, HSV, candida, cryptosporidia, *Mycobacterium avium* complex (MAC), or HIV leading to significant cachexia and electrolyte abnormalities
- Hepatobiliary disease is common. Causes of parenchymal liver disease include hepatitis B and C, CMV, mycobacterial infection (both *Mycobacterium tuberculosis* and *M. avium* complex), and cryptococcus. Cryptosporidiosis and CMV can cause cholangitis
- Hematologic abnormalities: Hematologic abnormalities affect each of the peripheral cell lines. Leukopenia is a hallmark of the disease, especially the depletion of CD4 lymphocytes; qualitative alterations in the function of neutrophils and macrophages also occur. Anemia is common
- Coagulation disturbances are common in HIV patients. Platelet production may be impaired because of direct infection of megakaryocytes with HIV. The activated partial thromboplastin time (aPTT) may be prolonged because of the presence of the lupus anticoagulant; this has no clinical significance. Many antiretroviral agents used have hematologic toxicity
- Cardiovascular abnormalities: Clinically significant cardiovascular disease is rare. Pericarditis is the most prevalent cardiovascular disorder in these patients. The most common etiology is mycobacterial infection Pulmonary hypertension can develop secondary to repeated episodes of PCP (*Pneumocystis carinii* pneumonia) and cytokine-mediated endothelial injury. Clinical myocarditis or cardiomyopathy is rare. Infective endocarditis occurs almost exclusively in intravenous drug users
- Endocrine abnormalities: Patients with AIDS frequently have abnormal thyroid function tests. Hyperglycemia secondary to insulin resistance has been reported in patients receiving protease inhibitors. Intravenous pentamidine therapy for CMV infection may cause islet cell injury, which may lead to hypoglycemia caused by the release of insulin from damaged islet cells. If sufficient islet cells are destroyed, diabetes mellitus can develop
- Renal abnormalities: Patients with HIV are at risk for acute renal failure secondary to sepsis, dehydration, and drug toxicity.

Interaction with Pregnancy

The diagnosis of HIV infection in the offspring of HIV-infected mothers is affected by the presence of passively acquired maternal antibody in the newborn for about 18 months. Until 18 months of age, an infant's HIV status must be confirmed by viral culture or DNA PCR. Measurement of circulating p24 antigen has been used for rapid diagnosis of neonatal HIV infection.

Prophylactic antibiotics must be used because of increased risk of postoperative infectious morbidity.

The effect of HIV infection on pregnancy outcome: HIV seropositivity was associated with an increased risk of preterm delivery and low birth weight. The incidence of serious infectious complications (e.g. PCP, CNS toxoplasmosis) is increased with advanced HIV infection (CD4 counts <300 cells/mm^3). The fetal implications of such infections are obvious. There is no evidence that drug therapy affects pregnancy outcome. There is no evidence that pregnancy accelerates clinical deterioration in the HIV-infected patient, or that viral RNA load changes significantly during pregnancy.[55]

Drug Therapy: Antiretroviral medications are used in pregnancy to achieve two goals; to maintain the mother's health and to prevent mother-to-child transmission (PMTCT) of HIV.

Currently, there are numerous nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), and PIs (protease inhibitors) approved for therapy. Antiretroviral drugs are not listed in FDA pregnancy category A, which signifies a lack of fetal effect in controlled human trials. Hence, they should be used cautiously.

Fetal side effects: NRTIs (nucleoside reverse transcriptase inhibitors) are well tolerated and have not been demonstrated to be teratogenic in humans. However, concerns have been raised about potential adverse effects on both mothers and infants related to the avidity of these drugs for mitochondria. Clinical disorders associated with mitochondrial toxicity include neuropathy, myopathy, cardiomyopathy, pancreatitis, hepatic steatosis, and lactic acidosis.

Anesthetic Consideration

- The HIV-positive parturient have a high-risk of sexually transmitted diseases
- Out of all syphilis is significant because of its neurologic effects in its later stages
- If regional anesthesia is performed, a careful neurologic examination should be documented. Hepatitis B also is a sexually transmitted disease, and should be investigated
- Severe hepatic impairment affects anesthetic management
- Regional anesthesia: There was no evidence of accelerated disease progression or increased infectious or neurologic complications after regional anesthesia in HIV-positive parturient
- There is no postoperative change in viral load or CD4+/CD8+ counts after spinal anesthesia. In the immediate postpartum period, the immune function remained essentially unchanged, as did the severity of the disease
- The natural history of HIV includes central nervous system involvement early in the clinical course and expression of this infection varies widely. The safety of epidural blood patches for treatment of postdural puncture headache has been reported in HIV-seropositive patients, but, given the very small theoretical risk of introducing virus to the central nervous system, an alternative is the epidural infusion of normal saline or colloidal solutions such as hetastarch. PDPH management consists of bed rest, analgesics, and oral hydration, intravenous or oral caffeine and 5-HT receptor agonist sumatriptan
- Regional anesthesia can be given safely unless there is contraindication to regional blocks like coagulopathy, local infection, blood dyscrasias, etc
- General anesthesia may be required due to obstetric causes, bleeding, coagulopathy. HIV patients are more prone to the pulmonary complications of general anesthesia. Hence take aseptic precautions perioperatively. Handle the endotracheal tube in a sterile manner and minimize the duration of postoperative ventilation
- Steroid supplementation may decrease hemodynamic instability and should be considered for unexplained hypotension
- HIV infection does not increase the risk of post procedural complications up to 30 days
- During anesthesia, tachycardia is more frequently seen in HIV seropositive patients, and, postoperatively, high fever, anemia and tachycardia are more common
- Immunosuppression from general anesthesia may persist for about 3 to 11 days.

Operation Theater Precautions

Postoperatively all the disposable items must be segregated and properly disposed.

Reusable anesthesia equipment like laryngoscope blades, endoscopes, should be thoroughly washed to remove gross contamination followed by either high-level disinfection or autoclaving.

Disposable carbon dioxide absorbers and unidirectional valves are used when anesthetizing HIV patients with active pulmonary tuberculosis. If a disposable absorber is not used, the entire assembly distal to the fresh gas source must be disassembled and sterilized.[56]

HIV transmission to medical personnel can be prevented by the mandatory use of universal blood and body fluid barrier precautions whenever contact with infectious material is anticipated, like blood, amniotic fluid, CSF, synovial fluid, pleural fluid, and pericardial fluid. Saliva is not thought to be infectious, but manipulations of the oral mucosa (e.g. laryngoscopy and endotracheal intubation) may lead to the contamination of saliva with blood. The barrier protection should include gloves, mask, full body gown and eye shields. The avoidance of needle stick injuries is important.

REFERENCES

1. Oakley CM. Pregnancy and Congenital Heart Disease. Heart 1997;78:12-4.
2. Davis Gregory AL, Herbert William N P. Congenital heart disease in pregnancy. J Obstet Gynaecol Can 2007;29:409-14.

3. Jahan S, Das T, Rahman S, Hossain Md Z, et al. Pregnancy and its outcome in women with and without surgical correction of Tetralogy of Fallot. University Heart Journal 2009;5:42-3.
4. Severe cardiac disease in pregnancy, Part-2: Impact of congenital and acquired cardiac diseases during pregnancy. Curr Opin Crit Care 2005;11:435-48.
5. Easterling Thomas R, Chadwick HS , Otto Catherine M, J Benedetti Thomas J. Aortic stenosis in pregnancy. Obstet Gynecol 1988;72:113-8.
6. Hadi I, Parkin J. Aortic stenosis and pregnancy: A case report and review of peripartum anesthetic management. Kuwait Medical Journal 2008;40:64-6.
7. Davis Gregory AL, Herbert William NP . Acquired heart disease in pregnancy. J Obstet Gynaecol Can 2007;29:507-9.
8. Reimold Sharon C, Rutherford John D. Valvular heart disease in pregnancy. N. Engl J Med 2003;349:52-9.
9. Desai DJ, Golwala MP, Dimar AA, Swadia VN. Emergency LSCS in a patient with severe Mitral Stenosis with Pulmonary Hypertension with Aortic Regurgitation. (NYHA-3). Indian J Anaesth 2002;46:483-5.
10. Gambling David R, Huckell Victor F. Structural heart disease. In: Gambling David R, Douglas Joanne M , (Eds). Obstetric Anesthesia and Uncommon Disorders. Philadelphia: WB Saunders 1998;1-20.
11. Valvular heart disease. In: Stoelting Robert K, Dierdorf Stephen F. (Eds). Anesthesia and Co-existing diseases. (4th Edn). Philadelphia. Churchill Livingstone 2002;25-44.
12. Mangano Dennis T. Anesthesia for the pregnant cardiac patient. In: Huges Samuel C, Gershan Levinson, Rosen Mark A., (Ed). Shnider and Levinsons Anesthesia for Obstetrics. 4th edn, Philadelphia. Lippincott Williams & Wilkins 2001;455-86.
13. Suryakant, Ojha Shashank. Management of asthma in pregnancy. Asthma Immunol Indian J Allergy 2006;20:112-5.
14. Nelson-Pierrcy C. Asthma in pregnancy. Thorax 2001; 56:325-8.
15. Abou-Gamrah A, Refaat M. Bronchial asthma in pregnancy. ASJOC 2005:2;226-30.
16. NAEPP (National Asthma Education and Prevention Program) Expert Panel Report. Managing Asthma during pregnancy: Recommendations for Pharmacologic treatment-2004 Update. J Allergy Clin Immunol 2005;115:34-46.
17. Howell Paul R. Acute Severe Asthma. In: Gambling David R, Joanne M, (Ed). Obstetric Anesthesia and Uncommon Disorders. Philadelphia: WB. Saunders 1998;119-24.
18. Asthma. In: Stoelting Robert k, Dierdorf Stephen F. (Eds). Anesthesia and Co-existing diseases. 4th edn, Philadelphia. Churchill Livingstone 2002;193-204.
19. Carlisle S. Perioperative management of the pregnant patient with asthma. In: Huges Samuel C, Levinson G, Rosen Mark A. (Eds). Shnider and Levinsons Anesthesia for Obstetrics. 4th edn, Philadelphia. Lippincott Williams & Wilkins 2001;487-95.
20. Krakow D. Hematologic diseases. In: Gibbs Ronald S, Karlan Beth Y, Haney Arthur F, Nygaard I. (Eds). Danforths Obstetrics and Gynecology. 10th edn, and Philadelphia: Lippincott Williams & Wilkins 2008;276-283.
21. Firth Paul G, Head A. Sickle cell disease and Anesthesia. Anesthesiology 2004;101:766-85.
22. Dewan D. Obesity. Principles and Practice. In: Chestnut DH, (Ed). Obstetric Anesthesia. 6th edn, St Louis: Mosby 1994;942-53.
23. Bimbach David J, Browne Ingrid M. Anesthesia for Obstetrics. In: Ronald D. Miller, (Ed). Millers Anesthesia. 7th edn, Philadelphia. Churchill Livingstone Elsevier 2010;2203-40.
24. Ogunnaike Babatunde O, Whitten Charles W. Evaluation of the Obese Patient. In: Longnecker David E, Brown David L, Newman Mark F, Zapol Warren M, (Eds). Anesthesiology. New York: McgrawHill Medical 2008; 374-95.
25. Shenkman YS, Brodsky JB. Perioperative Management of the Obese Patient. Br J of Anesthesia 1993;70:349-59.
26. Cohen Sheila E. Anesthesia for the morbidly obese pregnant patient. In: Huges Samuel C, Gershan Levinson, Mark A. Rosen, (Ed). Shnider and Levinsons Anesthesia for Obstetrics 4th edn, Philadelphia. Lippincott Williams & Wilkins 2001;545-558.
27. Diseases associated with pregnancy. In: Stoelting Robert K , Dierdorf Stephen F. (Eds). Anesthesia and Co-existing diseases. 4th edn, Philadelphia. Churchill Livingstone 2002;655-85.
28. Wissler Richard N. Thyroid disorders. In: Chestnut David H. (Eds). Obstetric Anesthesia Principles and Practice 3rd edn, Philadelphia. Mosby 2004;744-50.
29. Kausar R. Diabetes in Pregnancy. Indian Journal for the Practicing Doctor 2004;1:11-2.
30. Reece E Albert, Homko Carol J. Diabetes Mellitus and Pregnancy. In: Gibbs, Karlan Ronald S, Haney Beth Y, Nygaard Arthur F, Ingrid E. (Eds). Danforth's Obstetrics and Gynecology (10th edn). Philadelphia: Lippincott Williams & Wilkins 246-56.
31. Krishnamurty E, Pavlic-Renar I, Metalko Z. Diabetes & Pregnancy. Diabetologia Croatica 2002;31:131-46.
32. Couston Donald R. Management of gestational diabetes. In: E. Reece A, Coustan Donald R, Gabbe Steven G. (Eds). Diabetes in Women Adolescence, Pregnancy and Menopause 3rd edn, Philadelphia: Lippincott Williams & Wilkins 227-33.
33. Datta S. Anesthesia for the pregnant diabetic patient. In: Huges Samuel C. Levinson G, Rosen Mark A, (Eds). Shnider and Levinsons Anesthesia for Obstetrics (4th edn). Philadelphia. Lippincott Williams & Wilkins 2001;497-507.
34. Sookoian S. Liver disease during pregnancy: Acute viral hepatitis. Annals of Hepatology 2006;5:231-36.
35. Sookoian S. Effect of Pregnancy on Pre-existing Liver disease: Chronic Viral Hepatitis. Annals of Hepatology 2006;5:190-7.
36. Holden E Thomas, Donald M Sherline. Hepatitis and hepatic failure in pregnancy. Obstetrics and Gynecology 1972;40:586-93.

37. A Kumar, Sharma K Aparna, Gupta RK, Kar P, Chakravarti A. Prevalence and risk factors for hepatitis C virus among pregnant women. Indian J Med Res 2007; 126: 211-5.
38. Floreani A. Viral Hepatitis and Pregnancy. Current Women's Health Reviews 2009;5:8-13.
39. Beniwal M, Kumar A, Kar P, Jilani N, Sharma JB. Prevalence and severity of acute viral hepatitis and fulminant hepatitis during pregnancy: A prospective study from North India. Indian Journal of Medical Microbiology 2003;21:184-5.
40. Tran Tram T, Keeffe Emmet B. Management of the pregnant hepatitis B patient. Current Hepatitis Reports 2008;7:12-7.
41. Sato T, Hashiguchi A , Mitsuse T. Anesthesia for cesarean delivery in a pregnant woman with Acute Hepatic Failure. A and A 2009;91:1441-2.
42. Lee Noel M, Brady Carla W. Liver Disease in Pregnancy. World J Gastroenterol 2009;15:897-906.
43. Riely Caroline A. Liver disease in the pregnant patient. The American Journal of Gastroenterology 1999;94:1728-32.
44. Brunt Elizabeth M, Pathology of Fatty Liver Disease. Modern Pathology 2007;20:40-8.
45. Rathi U, Bapat M, Rathi P, Abraham P. Effect of liver disease on maternal and fetal outcome A prospective study. Indian J Gastroenterol 2007;26:59-63.
46. Vora Kalpana S, Shah Veena R, Parikh Geeta P. Acute fatty liver of pregnancy. Indian J Crit Care Med 2009;13:34-6.
47. Breen Kerry J, Perkins Kenneth W, Schenker S, Dunkerley Robert C, Noore HC. Uncomplicated subsequent pregnancy after idiopathic fatty liver of pregnancy. Obstetrics and Gynecology 1972;40:813-5.
48. Holden Thomas E, Sherline Donald M. Hepatitis and hepatic failure in pregnancy. Obstetrics and Gynecology 1972;40:586-93.
49. Stravitz R Todd, Kramer Andreas H, Dravern T, S Shaikh A Obaid, et al. Intensive care of patients with Acute liver failure. Crit Care Med 2007;35:2498-2508.
50. Cereda M, Horak J, Neligan Patrick J. Renal diseases. In: Lee A. Fleisher, (ed). Anesthesia and Uncommon Diseases. (5th Edn), Philadelphia: Saunders Elsevier 2006;229-60.
51. Gaiser R. Evaluation of the Pregnant Patient. In: Longnecker David E, Brown David L, Newman Mark F, Zapol Warren M, (Ed). Anesthesiology. New York: McgrawHill Company 2008;358-73.
52. Miller Ronald D, Rupp Stephen M, Fisher Dennis M, Cronnelly R, Fahey Mark R, Sohn Yung J. Clinical pharmacology of Vecuronium and Atracurium. Anesthesiology 1984;61:444-53.
53. Osler W. Malaria. In: Fauci, Braunwald, Kasper, Hauser, Longo, Jameson, Loscalzo. (Eds). Harrisons Principles of internal medicine. 17th Edn, New York: Mcgraw Hill Company 2008;1280-94.
54. Lindsay Keenan L, Yudin Mark H. HIV Screening in Pregnancy. JOGC 2006;185:1103-7.
55. Shah I. HIV and Pregnancy- is vaginal delivery a safe and viable option? Indian Pediatrics 2008;45:603.
56. Hughes Samuel C, Dailey Patricia A. Human Immunodeficiency Virus in the delivery suite. In: Huges Samuel C, Gershan Levinson, Rosen Mark A (Eds). Shnider and Levinsons Anesthesia for Obstetrics 4th edn, Philadelphia. Lippincott Williams & Wilkins 2001;583-95.

Section V

Neonatal and Pediatric Emergencies

17 General Consideration in Neonatal and Pediatric Emergency Surgeries

Anila D Malde, Nilam D Virkar

KEY POINTS

- Hypoxia, hypercapnia, acidosis, hypothermia, anesthesia-induced changes in peripheral or pulmonary vascular tone, prematurity and infection can cause reversion to fetal circulation.
- In neonates cardiac output is heart rate dependent. Parasympathetic dominance leads to increased incidence of bradycardia.
- Neonates exhibit biphasic response to hypoxemia, i.e. initial hyperventilation followed by sustained hypoventilation.
- Newborns respond to hypercapnia by increasing ventilation but less so than do older infants.
- Premature infants less than 50 weeks postconceptional age at the time of surgery are prone to postoperative episodes of obstructive and central apnea for up to 24 hours.
- Neonates and infants have a proportionately larger head and tongue, narrow nasal passages, an anterior and cephalad larynx, a long epiglottis, and a short trachea and neck. They are obligate nasal breathers. The cricoid cartilage is the narrowest part. They are prone to dangerous tracheal edema.
- Negative intrathoracic pressure is poorly maintained thus leading to lung collapse, especially under general anesthesia. Lower amount of Type I fibers in respiratory muscles make them more prone for fatigue.
- Alveolar ventilation is 130 ml/kg/min for neonate versus 60 ml/kg/min for adults to meet, increased oxygen consumption and carbon dioxide production.
- Fetal hemoglobin which is present up to 3 month has higher affinity for oxygen.
- GFR, tubular mechanisms, concentrating, and diluting capacity is not well-developed in infants.
- Higher fluid requirement is due to higher insensible fluid loss, larger surface area, immature skin, and higher metabolism.
- There are two peaks in the occurrence of neonatal hypocalcemia. Neonates who are not breastfed are supplemented with 1ml/kg 10 percent Ca gluconate 8 hourly.
- Functional maturity of the liver is incomplete at birth.
- Due to limited glycogen stores there is tendency towards hypoglycemia.
- Blood brain barrier is immature; nerves are the thinner with less myelination in the neonates; CSF volume is relatively high in infants requiring higher drug volume.
- Changes in blood flow, pressure, and osmolality are associated with the development of intraventricular hemorrhage (IVH) in preterm newborns.
- Hypothermia is a serious problem in the neonates. Adequate measures are needed to prevent heat loss.
- Larger body water content, lesser proteins and fats, immature hepatic and renal function alters drug pharmacology in neonates.
- Both induction and emergence from inhalational anesthesia are more rapid in children than in adults.
- The blood pressure of neonates and infants tends to be more sensitive to volatile anesthetics.
- Children are more susceptible than adults to cardiac arrhythmias, hyperkalemia, rhabdomyolysis, myoglobinemia, masseter spasm, and malignant hyperthermia after administration of succinylcholine.
- Vitamin K is usually given to all newborns.
- Intravenous induction is most commonly used in emergency.
- Awake intubation may cause intraventricular hemorrhage in fragile, premature newborns.
- For infants, especially those younger than six months, intubation is indicated because upper airway obstruction occurs commonly.

- Precordial stethoscope, ECG, pulse oximetry, NIBP, and temperature are minimum essential monitors. Microstream capnograph can give idea about circulation, respiration, metabolism and machine related problems. It is important to identify at least one peripheral pulse site which can be monitored throughout. If not, axillary artery should be palpated intermittently.
- Fluid therapy includes replacement of deficit fluid, maintenance fluid by Holliday/Segar formula and third space losses. Lactated Ringer is the fluid of choice for intraoperative period. Dextrose 10 percent, 5 percent, 2.5 percent is added in premature, term neonates and children respectively.
- A minimum hematocrit of 30 percent in older children and 40 percent for neonates have to be maintained.
- Paracetamol, NSAIDs, fentanyl, local bupivacaine infiltration, caudal, epidural local anesthetics and narcotics are the mainstay of postoperative analgesia.
- Maintenance of normothermia, normoglycemia, normovolemia and normal hematocrit are very essential in premature babies.
- Choanal atresia is commonly associated with other congenital anomalies. Oral airway is needed during induction. Baby should be awake at the time of extubation.

INTRODUCTION

This chapter outlines the essential principles of safe pediatric anesthesia for emergency surgeries covering the basics of anatomy, physiology and pharmacology, suggested techniques, monitoring methods, regimens for fluid management and new advances in pediatric pain management.

PHYSIOLOGY

Cardiovascular System

Maturation of the cardiovascular occurs throughout fetal development and continues into the neonatal period and infancy.

Fetal circulation:[1] In the fetus, gas exchange occurs at the placenta. Fetal intracardiac and extracardiac shunts (foramen ovale, ductus arteriosus, and ductus venosus) exist to minimize flow to the lungs. At birth, fetal shunts are no longer needed and must close to permit the efficient transition to neonatal circulation with resultant increase blood flow to the lungs. Fetuses have parallel circulation unlike series circulation of adults.

Transitional circulation:[1] Though functional closure of the foramen ovale occurs at birth, anatomical closure occurs between 3 months and one year of age. Probe patency of the foramen ovale is demonstrated in 50 percent of children under five years of age.[2] Therefore care has to be exercised in prevention of air injection. Functional closure of the ductus arteriosus occurs with removal of the placenta and a consequent decrease in the levels of circulating prostaglandins and increasing oxygen tension. Final anatomic closure results from thrombosis and fibrosis over the first few months of life. Ductus arteriosus remains patent in 50 percent of the preterm newborns which is unlikely to close spontaneously in extremely low gestational age newborns. Therefore they are at risk for the development of congestive heart failure, intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), renal insufficiency, pulmonary edema, or hemorrhage. Management in the neonatal intensive care unit (NICU) starts with fluid restriction, diuresis, and packed red blood cell (PRBC) administration to keep the hematocrit at 40 percent. If these measures fail, indomethacin is given.[3]

Persistent fetal circulation:[4] Many factors like hypoxia, hypercapnia, acidosis, hypothermia, anesthesia-induced changes in peripheral or pulmonary vascular tone, prematurity and infection leads to increase in the pulmonary artery pressure up to systemic levels. This causes reopening of the intracardiac shunts. A rapid downhill spiral may occur and lead to severe hypoxemia, which explains why hypoxemic events in infants are often prolonged despite adequate pulmonary ventilation with 100 percent oxygen.

Developmental aspects of myocardial function: Cardiac calcium stores are reduced that produces increased susceptibility to myocardial depression by potent anesthetic agents. This also makes neonates dependent on exogenous calcium (blood ionized calcium).[4]

Neonatal myocardium has higher percentage of noncontractile protein in fetal myocytes (60 percent versus 30 percent in the adult myocardium),[5] which render the myocardium stiff and noncompliant. This may impair filling of the left ventricle and limit the ability to increase the cardiac output by increasing stroke volume (Frank Starling mechanism). Stroke volume is therefore relatively fixed and the only way of increasing cardiac output is by increasing heart rate.

Parasympathetic innervation is noted very early in development. Sympathetic innervation may begin in the area of the sinoatrial node and proceed to the ventricle but is definitely not complete at birth leading to increased incidence of bradycardia in neonates. Arterial blood pressure increases with age.

Respiratory System

To introduce air into the fluid-filled lungs at birth, the newborn infant must overcome large surface force with the first few breaths. Usually fluid is rapidly expelled via the upper airways. The residual fluid leaves the lungs through the pulmonary capillaries and lymphatic channels. If this fails to occur baby may go in respiratory distress also known as transient tachypnea of newborn, which gradually disappears. Premature babies are deficient in surfactant predisposing them to respiratory distress syndrome and require respiratory support leading to increased chances of barotraumas and bronchopulmonary dysplasia. Ventilation should be gentle, allowing modest degrees of hypercarbia and SpO_2 of 85 percent.

Control of Breathing[6]

Response to hypoxemia in infants: During the first 2 to 3 weeks of age, both full-term and premature infants in a warm environment respond to hypoxemia (15 percent oxygen) with a transient increase followed by sustained decreased in ventilation.[7] In infants born at 32 to 37 weeks gestation, the initial period of transient hyperpnea is abolished in a cool environment, indicating the importance of maintaining a neutral thermal environment.[8-10] When 100 percent oxygen is given, a transient decrease in ventilation is followed by sustained hyperventilation.[11] By 3 weeks after birth, hypoxemia induces sustained hyperventilation, as in older children and adults. The biphasic depression in ventilation has been attributed to central depression rather than to depression of peripheral chemoreceptor.[12] Premature infants continue to show a biphasic response to hypoxemia even at 25 days after birth.[13]

Response to carbon dioxide in infants: Newborn infants respond to hypercapnia by increasing ventilation but less so than do older infants. The slope of the CO_2 response curve increases appreciably with gestational age as well as with postnatal age. This increase in slope may represent an increase in chemosensitivity, but it may also result from more effective mechanics of the respiratory system. In adults the CO_2 response curves both increases in slope and shifts to the left with the severity of hypoxemia. In contrast, in newborn infants breathing 15 percent oxygen, the CO_2 response curve decreases in slope and shifts to the right. Inversely, hyperoxemia increases the slope and shifts the curve to the left.[14]

Periodic breathing, in which breathing is interposed with repetitive short apneic spells lasting 5 to 10 seconds without desaturation, occurs frequently in neonates and young infants. Incidence of periodic breathing was reported to be 78 percent in full-term,[15] and 93 percent in preterm infants.[16] The frequency of periodic breathing diminishes with increasing postconceptual age and decreases to 29 percent by 10 to 12 months of age.[15,17]

Apnea of prematurity and hypoxia: Central apnea of infancy is defined as unexplained cessation of breathing for 20 seconds or longer or a shorter respiratory pause associated with bradycardia (heart rate <100), cyanosis, or pallor.[18] Apnea is common in preterm infants and may be related to an immature respiratory control mechanism.

Postoperative apnea:[19] Premature infants who are less than 50 (some authorities would say 60) weeks postconceptional age at the time of surgery are prone to postoperative episodes of obstructive and central apnea for up to 24 hours. In fact, even term infants can experience apneic spells following general anesthesia (GA). Risk factors include: low gestational age at birth, anemia, hypothermia, sepsis, and neurological abnormalities. Therefore careful monitoring in the postoperative period is required. Both theophylline (10 mg/kg) and caffeine have been effective in reducing apneic spells in preterm infants.

Anatomical Differences[4,6,20-22]

- The head is relatively large with a prominent occiput, the neck is short.
- The tongue is large. The airway is prone to obstruction because of these differences.
- Infants and neonates breathe mainly through their nasal airway, and their nostrils are small and can be easily obstructed. The cephalad position of the epiglottis and close approximation of the soft palate to the tongue and epiglottis in neonates may be a reason why mouth breathing is more difficult than nose.
- The larynx is higher in the neck, being at the level of C3 in a premature infant and C4 in a child compared to C5-6 in the adult.
- The epiglottis is short, stubby, floppy, omega shaped, and angled over the laryngeal inlet; control with the laryngoscope blade is therefore more difficult.[4]
- The trachea is short and the right main bronchus is less angled and wider than the left therefore right mainstem intubations are more likely.
- The glottic opening (laryngeal opening) is more anterior and the narrowest part of the airway is at the cricoid ring. (In the adult airway the narrowest point is the vocal cords). Therefore uncuffed

endotracheal tubes have generally been preferred for children younger than 6 years. However, the development of better endotracheal tube design and several prospective studies have combined to allow more common use of cuffed endotracheal tubes, even in infants.[21] However, there should be leak around the cuff (with or without inflation) because injury to the tracheal mucosa is still possible.

- The diameter of the trachea in the newborn is 4-5 mm. The resistance to airflow through a tube is inversely related to the fourth power of the radius of the tube. Tracheal edema of just 1 mm can dramatically increase resistance to breathing.
- The vocal cords are angled, so a "blindly" passed endotracheal tube may easily lodge in the anterior commissure rather than slide into the trachea.[4]
- The airway of infants is highly compliant and poorly supported by surrounding structures. The chest wall is also highly compliant, so the ribs provide little support for the lungs; that is, negative intrathoracic pressure is poorly maintained. Thus, each breath is accompanied by functional airway closure. This makes infants more prone to lung collapse, especially under general anesthesia.[6]
- Another important factor is the composition of the diaphragmatic and intercostal muscles. Type I muscle fibers provide the ability to perform repeated exercise. These diaphragmatic and intercostals muscles do not achieve the adult configuration of Type I muscle fibers until the child is approximately 2 years old.[22] As a result, they are more easily fatigued.[4]

As metabolic rate is high, it leads to increased oxygen consumption and carbon dioxide production. Alveolar minute ventilation is increased to meet the increased oxygen demands and to maintain normal $PaCO_2$ level. Alveolar ventilation is 130 ml/kg/min for neonate versus 60 ml/kg/min for adults. Tidal volume is similar for adults and children on ml/kg basis, so that the increased alveolar ventilation is achieved by an increase in respiratory rate. All of these factors give the lungs less reserve of oxygen so that a well oxygenated infant with upper airway obstruction can become cyanotic in a matter of seconds.

The breathing pattern of infants less than six months of age is predominantly abdominal (diaphragmatic) and the contribution of the rib cage (external intercostal muscles) to tidal volume is relatively small (20 to 40 %). After nine months of age, the rib cage component of tidal volume increases to a level (50%) similar to that of older children and adolescents.

Oxygen Delivery in Fetus and Neonate[6,20,23]

Fetal hemoglobin (HbF) (which is present in fetal life and up to three months) is not able to deliver oxygen to the tissues as efficiently as adult hemoglobin (HbA). Oxygen competes with 2,3-diphosphoglycerate (2,3-DPG) for binding on the hemoglobin molecule. Levels of 2,3-DPG are lower in the fetus than in the adult. In addition, HbF has a low affinity for 2,3-DPG compared with HbA and therefore HbF preferentially binds oxygen. The P_{50} occurs at a lower PO_2 in the fetus as a result of HbF.[2] Neonates have a higher hemoglobin concentration (17g/dl) and blood volume and this together with the increased cardiac output compensates for the decreased release of oxygen from hemoglobin in the tissues. Replacement of fetal hemoglobin with adult hemoglobin begins at 2 to 3 months of age and this period is known as physiological anemia as hemoglobin concentrations may fall to 11 g/dl. Anemia sufficient to jeopardize oxygen carrying capacity of the blood is possible if the hemoglobin concentration is less than 13 g/dl in the newborn and less than 10 g/dl in the infant under six months of age.[20] By 4 to 6 months, the oxyhemoglobin dissociation curve approximates that of adults.[23] Cardiovascular and respiratory differences are shown in Table 17.1.[6,20,24,25]

Renal System and Fluid Balance[1,26-30]

Although all nephrons of the mature kidney are formed by 36 weeks of gestation of normal intrauterine life, hyperplasia continues until the sixth postnatal month; thereafter, cell hypertrophy is responsible for increase in renal size.[26]

- Fetal kidney receives 3 to 7 percent of cardiac output.[27] Renal blood flow (RBF) increases gradually after birth and reaches adult levels at 2 years.
- GFR in the full-term newborn infant averages 40.6 ± 14.8 ml/min per 1.73 m^2 reaches adult levels after 2 years of age. Premature newborns have a lower GFR that increases more slowly than that in full-term infants.[28]
- The tubular mechanisms involved in the excretion of organic acids are poorly developed in neonates. Proximal tubular secretion reaches adult values by six months of age. Excretory capacity is usually similar to older children by six months of age.[29]
- Concentrating ability is low at birth, especially in premature infants. After water deprivation in the full-term newborn, urine concentrates to only 600 to 700 mOsm/kg, or 50 to 60 percent of maximum adult levels.

Table 17.1: Cardiovascular and respiratory differences[6,20,24,25]

Age	*HR/ Min*	*SBP (mm Hg)*	*Blood Vol. (ml/kg)*	*Hb (g/100 ml)*	*Hct (%)*	*P_{50} (mm Hg)*	*RR /min*	*FRC ml*	*O_2 consumption ml/kg/min*
Neonate	133 ± 18	80 ± 16	85	16.5 ± 1.5	55 ± 7	19.4	50 ± 10	80	6.0 ± 1.0
6 months	120 ± 20	89 ± 29	80	11.5 ± 1.0	37 ± 3	27.8	30 ± 5	-	5.0 ± 0.9
1 year	120 ± 20	96 ± 30	80	12.0 ± 0.75	35 ± 2.5	30.0	24 ± 6	-	5.2 ± 0.9
2 years	105 ± 25	99 ± 25		12.5 ± 0.5	40 ± 3		24 ± 6	490	6.4 ± 1.2
5 years	90 ± 10	94 ± 14	75	12.5 ± 0.5	40 ± 2	29.0	23 ± 5	680	6.0 ± 1.1
9 to 12 years	70 ± 17	113 ± 18		13.5 ± 1.0	42 ± 2	27.9	18 ± 5	1970	3.3 ± 0.6
Adult	75 ± 5	120	70	14.0 ± 1.0	43-48	27.0	12 ± 3	3000	3.4 ± 0.6

Table 17.2: Maturation of renal function with age[29]

Measurement	*Premature newborn*	*Full-term newborn*	*1 to 2 weeks*	*6 Months to 1 year*	*1 to 3 years*	*Adult*
GFR (ml/min per 1.73 m^2)	14 ± 3	40.6 ± 14.8	65.8 ± 24.8	77 ± 14	96 ± 22	Male: 125 ±15 Female: 110 ± 15
RBF (ml/min per 1.73 m^2)	40 ± 6	88 ± 4	220 ± 40	352 ± 73	540 ± 118	620 ± 92
Maximal concentration ability (mOsm/kg)	480	700	900	1200	1400	1400
Serum creatinine (mg/dl)	1.3	1.1	0.4	0.2	0.4	0.8 to 1.5
Fractional excretion of Na (%)	2 to 6%	<1	<1	<1	<1	<1

- A water-loaded infant can excrete dilute urine with osmolality as low as 50 mOsm/kg. In the first 24 hours of life however, the infant may be unable to increase water excretion to approximate water intake.[30] The diluting capacity becomes mature by 3 to 5 weeks of postnatal life. Maturation of renal function with age is shown in Table 17.2.[29]

Fluid and Electrolyte Balance in Infants and Children[29]

Body Water Composition and Changes with Gestational and Postnatal Age

Neonates are born with an excess of total body water (TBW), primarily extracellular fluid (ECF). They lose this excess ECF in the first postnatal week. Term neonate have 75 percent water (40% ECF, 35% ICF), and usually lose 5 to 10 percent of their weight in the first week of life, which is water loss. Preterm neonates have more water (at 23 weeks' gestation, 90% water composed of 60% ECF and 30% ICF), and they lose 10 to 15 percent of their weight in the first week of life. Small for gestational age (SGA) preterm infants may have higher proportional body water content (90% for SGA infants vs 84% for appropriate for gestational age [AGA] infants at 25-30 weeks' gestation). Evaporative losses through their immature skin can be quite large, up to 5 to 6 ml/kg/hour. Maintenance water requirement in these tiny, immature infants is high, 120 ml/kg/day.

Infants have greater fluid needs because of:

- Higher rates of metabolism and growth
- Surface area-to-weight ratio that is about three times greater
- Immature skin results in higher insensible fluid loss. In infant and in premature babies skin is even more delicate, leading to larger evaporative fluid loss
- Greater urinary excretion of solutes combined with lower tubular concentrating ability, which increases obligatory fluid loss. Loss is even greater in premature newborns.

Na^+ = 2.5 mmol/100 kcal expended. The high fractional excretion of Na^+ (FE^+_{Na}) in premature infants can lead to negative Na^+ balance, hyponatremia, neurologic disturbances, and poor growth unless an Na^+ intake of 3 to 5 mmol/kg per day is given.[29,31]

K^+ = 2.5 mmol/100 kcal expended.[31] In the early postnatal period especially immature infants serum potassium concentration is higher than older persons. Etiology of the hyperkalemia is multifactorial which involves the developmentally functional difference in the Na-K-ATPase activity and hormonal milieu. K^+ supplementation should be started after volume contraction is complete and urine output is well-established usually during 2nd day of life.[32]

Ca^{++}= There are two peaks in the occurrence of neonatal hypocalcemia. An early form occurs during the first few days after birth, with the lowest concentrations

Table 17.3: Normal electrolyte levels in neonates[34,35]

Electrolyte	*Cord*	*1-12 hr*	*12-24 hr*	*24-48 hr*	*48-72 hr*	*3-10 d*
Na (mEq/L)	147 (126-166)	143 (124-156)	145 (132-159)	148 (134-160)	149 (139-162)	
K (mEq/L)	7.8 (5.6-12)	6.4 (5.3-7.3)	6.3 (5.3-8.9)	6.0 (5.2-7.3)	5.9 (5.0-7.7)	
Cl (mEq/L)	103 (98-110)	101 (80-111)	103 (87-114)	102 (92-114)	103 (93-112)	
Ca (millimol/L)	2.33 (2.1-2.8)	2.1 (1.8-2.3)	1.95 (1.7-2.4)	2.0 (1.5-2.5)	1.98 (1.5-2.4)	
Ca (I) (millimol/L)		1.05-1.37	1.05-1.37	1.05-1.37	1.10-1.44	1.20-1.48

of serum Ca being reached at 24 to 48 hours of age; late neonatal hypocalcemia occurs toward the end of the first week. Reasons include abrupt discontinuation of placental Ca supply at birth, limited or no dietary Ca, smaller skeletal reservoir available for mineral homeostasis, high requirement for Ca for the most rapid period of postnatal skeletal growth, transient limited increase in the serum PTH concentration, possibly end-organ resistance to PTH and 1,25 (OH) 2D and elevated serum calcitonoin concentration. Treatment include intravenous 10 to 20 mg elemental Ca/kg (10% Ca gluconate or 10% Ca chloride provides 9 mg elemental Ca/ml or 27.2 mg/ml, respectively) with dextrose water or normal saline infused over 5 to 10 minutes under constant ECG monitoring. For neonates who are not breastfed calcium is given in the dose of 1 ml/kg 10 percent Ca gluconate 8 hrly.[33] Serum calcium levels of 6.5 to 8 mg/dl are normal for premature infants because these babies have lower protein concentration, and more unbound calcium available for the cell. Normal electrolyte levels in neonates are shown in Table 17.3.[34,35]

Liver[4,36,37]

- At term, the functional maturity of the liver is incomplete. The cytochrome P450 system is responsible for Phase I drug metabolism of lipophilic compounds. This system reaches approximately 50 percent of adult values at birth, thus suggesting an overall reduced capacity for drug metabolism (e.g. caffeine).[36]
- Phase II reactions involve conjugation, which makes the drug more water soluble to facilitate renal excretion. These reactions are often impaired in neonates. It reaches the adult value by the age of one year.[37]
- Liver stores of glycogen (and iron) are made up primarily in the last trimester of fetal life. A preterm infant has minimal glycogen stores and is unable to handle large protein loads. This difference accounts for the neonate's tendency toward hypoglycemia and acidemia and for the failure to gain weight when the diet contains too much protein. There is intolerance to any degree of hypoglycemia due to their very limited ketogenesis and lipolysis capability to create energy from alternate sources.
- Due to decreased synthetic capacity of liver, plasma levels of albumin and coagulation factors are low in full-term newborns (and are even lower in preterm infants). This leads to greater levels of unbound drug and increased coagulopathy (e.g. the need for vitamin K at birth).

Gastrointestinal System[4]

The ability to coordinate swallowing with respiration does not fully mature until infants are 4 to 5 months of age, thus resulting in a high incidence of gastroesophageal reflux in newborns and more so in preterm infants.[4]

Central Nervous System[4,38]

Several anatomical features distinguish central nervous system of neonate from that of adult:

- Soft and pliable cranium, nonfused sutures, open fontanels (posterior fontanel closes by 6-9 month of postnatal age, and anterior by 18 months of age)
- Composition of brain changes dramatically with age. Water content of the brain decreases as myelin and other protein content increases. This leads to increase in the blood gas partition coefficient as the child grows
- The blood brain barrier is immature in neonate, facilitating the passage of large lipid soluble compounds such as anesthetic drugs and free bilirubin into the brain
- Cerebral blood flow (CBF) in healthy neonate is (30-40 ml/100 g of brain tissue/min) less than adult (55 ml/100 g of brain tissue/min)
- In neonates and infants (under 1 year) the spinal cord (ends at the level of L3) and dural sac (ends at S2-S4), are located more caudal than in the adults. Therefore there is increased risk of trauma to the spinal cord and inadvertent puncture of dura. Lower approach to the epidural space is favoured. At

approximately one year the spinal cord ends at L1, and the dural sac at S2, same as in adult position
- The intercristal line is found at L5 in children and the L5-S1 interspace in prematures and neonates compared to adult level of L4
- The vertebral column forms a single shallow anteriorly concave curve extending from the C1 to L5 at birth. The cervical curve appears when the head is held upright (approximately 6 months) and the lumber curve develops with weight bearing (1 year)
- Due to cartilaginous structure of bones and vertebrae, there is decreased resistance to penetration of the needle during insertion, which can cause direct trauma to the ossification centre in the bone compromising further growth. Therefore excessive force should be avoided
- The spinous process is more parallel and horizontal facilitating epidural puncture at all levels
- CSF volume is relatively high in infants weighing less than 1.5 kg, i.e. 4 ml/kg body weight, in contrast to the adults and older children (2 ml/kg). CSF production is also increased (0.35 ml/min). This may explain why infants require proportionately more local anesthetic for spinal block than older children and the incidence of the postspinal headache is extremely low in children
- Nerves are thinner with less myelination in the neonates and young infants, allowing a lower concentration of local anesthetic to be effective. The nerve trunks to the lower limbs are fully myelinated by approximately the second year of the life. The degree of myelination of nerve fibers considerably influences the pharmacodynamic effects of local anesthetic
- The germinal matrix surrounding the ventricles in the brain is immature and delicate in preterm newborns. Changes in blood flow, blood pressure, and even serum osmolality have all been associated with the development of intraventricular hemorrhage (IVH) in preterm newborns.

Temperature Regulation [4,19,23]

- Neonates and infants have a large surface area to body mass ratio (normal ratio for term neonate is 1 and for adult about 0.40) and therefore a greater area for heat loss, especially from the head
- Body fat for insulation is insufficient therefore heat is lost more rapidly
- Infants less than three months of age do not shiver and rely primarily on nonshivering thermogenesis in which brown fat is used and occurs only in infants. The fat is located primarily around the scapula, in the mediastinum, around the adrenal glands and kidneys, around blood vessels in the neck, and in large deposits in the axilla. Metabolism of brown fat is severely limited in premature infants and in sick neonates who are deficient in fat stores.[17] The inhibition of nonshivering thermogenesis by inhalational anesthetics begins as early as five minutes after turning on the vapour and starts to wean off within approximately 15 minutes after discontinuation of the anesthetic.[2] Nonshivering thermogenenesis disappears by the age of two years.

Mechanisms of heat loss in neutral thermal environments (by percent): Radiation-39 percent, convection-34 percent, evaporation-24 percent, and by conduction-3 percent.[23]

Hypothermia is a serious problem that has been associated with delayed awakening from anesthesia, altered drug responses, cardiac irritability, bradycardia, respiratory depression, increased pulmonary vascular resistance, metabolic acidosis and hypoglycemia.[19,23]

To minimize oxygen consumption, the neonate must be in a neutral thermal environment. Neutral temperature is defined as the ambient temperature that results in the least oxygen consumption. The critical temperature is that ambient temperature below which an unclothed, unanesthetized person cannot maintain a normal core body temperature.[23] Neutral and critical temperatures for various age groups are shown in Table 17.4.

Measures to Prevent Heat Loss[4]

- For the very small neonate, operating room temperatures must be increased
- Place the baby on a warming mattress, which minimizes heat lost through conduction
- Heat loss may be reduced by wrapping the limbs and head in wool or foil
- Warming and humidification of inspired gases
- Warming of intravenous fluids
- Use of plastic wraps to decrease water loss through the skin
- Warming of skin disinfectant solutions decreases the heat loss through evaporation.

Table 17.4: Neutral and critical temperatures for various age groups

Age	*Neutral temperature (°C)*	*Critical temperature (°C)*
Preterm neonate	34	28
Term neonate	32	23
Adults	28	1

- Heat lost from radiation is decreased with the use of an incubator during transport
- Keeping the infant in an incubator, covered with blankets, minimizes heat lost through convection.

PHARMACOLOGY

Developmental Pharmacology[4]

The body compartments (fat, muscle, water) change with age. Total body water content is significantly higher in preterm than in term infants and in term infants than in 2-year-old. Fat and muscle content increases with age. These changes lead to:

- A drug that is water soluble has a larger volume of distribution and usually requires a larger initial dose (mg/kg) to achieve the desired blood level (e.g. most antibiotics, succinylcholine)
- Lower total plasma protein levels and lower levels of specific proteins especially alpha-1-acid glycoprotein lead to less protein binding. The free fraction of many drugs in the plasma is high. Lower doses of some drugs (e.g. barbiturates) are needed. Persisting fetal albumin has a reduced affinity for drugs. Increased concentrations of unconjugated bilirubin compete for binding sites with acidic drugs
- As there is less fat, a drug that depends on redistribution into fat for termination of its action will have a longer clinical effect (e.g. thiopental); and drug that redistributes into muscle may have a longer clinical effect (e.g. fentanyl)
- Delayed excretion is secondary to the larger volume of distribution
- Immature hepatic and renal function.

Inhalational agents: Both induction and emergence from anesthesia are more rapid in children than in adults, probably because of a smaller lung functional residual capacity, higher minute ventilation-to-FRC ratio with relatively higher blood flow to vessel-rich organs, contributes to a rapid rise in alveolar anesthetic concentration and speeds inhalation induction. Furthermore, the blood/gas coefficients of volatile anesthetics are lower in neonates than in adults, resulting in even faster induction and potentially increasing the risk of overdosing.[17]

MAC of inhalational agents is greatest in the young and decrease with age. However neonates require lower concentrations of volatile anesthetics than infants[18] (Table 17.5). The blood pressure of neonates and infants tends to be more sensitive to volatile anesthetics, probably because of not fully developed compensatory mechanisms (e.g. vasoconstriction, tachycardia) and an immature myocardium that is very sensitive to myocardial depressants.[17]

Sevoflurane: Children older than 3 years usually experience an increase in heart rate and no change in systolic blood pressure with sevoflurane, whereas neonates and infants <6 months of age, there is least increase in the heart rate and greater decrease in systolic blood pressure. In addition the sevoflurane is associated with higher incidence of emergence reaction and agitation.

Halothane: It is commonly used for the gaseous induction of anesthesia. Halothane is a potent myoca-rdial depressant that can have profound effects on neonates and children with congenital heart disease. Halothane also sensitises the heart to the arrythmogenic effect of adrenaline. Doses of adrenaline should be kept under 10 µ/kg when halothane is used. Incidence of halothane hepatitis is less in children.

Isoflurane: Disadvantage of isoflurane is its noxious smell, which is unacceptable to many pediatric patients, and a greater incidence of airway-related events (laryngospasm, coughing). With rapid increase in concentration, hypertension has been reported due to stimulation of pulmonary irritant receptor leading to sympathetic and renin angiotensin activation. Isoflurane has advantages over halothane: It has less myocardial depression, preservation of the heart rate and a greater reduction in the cerebral metabolic rate for oxygen.

Table 17.5: Pharmacokinetic characters[19,39]

Agent	*Odor*		*Neonates*	*Infants*	*Children*	*Adults*	*Myocardial depression*	*Respiratory depression*
Halothane	Non-Pungent	MAC	0.87	1.1-1.2	0.87	0.75	++	+
		Blood gas coe.	2.1			2.3		
Sevoflurane	Non-pungent	MAC	3.2	3.2	2.5	2.0	+	+
		Blood gas coe.	0.7			0.7		
Isoflurane	Pungent	MAC	1.60	1.8-1.9	1.3-1.6	1.2	+	++
		Blood gas coe	1.2			1.4		
Desflurane	Pungent	MAC	8-9	9-10	7-8	6.0	+	++
		Blood gas coe	-			0.4		

Intravenous Anesthetics and Sedatives[4,40,41]

Propofol: It is used for induction, maintenance of anesthesia, and sedation. Propofol is preferred to thiopental for induction in short cases. Because of the improved quality of early recovery from anesthesia, recovery is initially due to redistribution but metabo-lism and elimination are quicker than with thiopental. It is metabolised in the liver by glucuronidation and sulfation. Glucuronidation is decreased in the neonate, but this will be compensated by the sulfonation. The major drawback of propofol is pain on intravenous administration, particularly through small veins. The drug is administered through a large antecubital vien and as little as 2 mg/kg of lidocaine (mixed with propofol) is effective in reducing but not eliminating this discomfort. The induction dose is higher in younger children (2.9 mg/kg for infants younger than 2 years) than in older children (2.2 mg/kg for children 6 to 12 years of age). A modest reduction in systolic blood pressure often accompanies bolus administration. Propofol has been associated with a reduced rate of postoperative vomiting. Because propofol contains egg and soy products, it should be used with caution in children with egg or soy allergies. Propofol infusion can be used to sedate the children in ICU. But, unexplained metabolic acidosis and cardiac failure occurred in some patients. This may be due agglutination of the lipid vehicles of the propofol and lipid microemboli production.[41]

Thiopentone sodium: Short-acting agent for induction of anesthesia, potent anticonvulsant by bolus or infusion. Intravenous bolus administration of 2.5 percent thiopental (5 to 6 mg/kg) is sufficient to induce anesthesia in most healthy, unpremedicated pediatric patients. Reduced clearance and prolonged elimination half-life of 19 hours in neonates compared with 6 to 12 hours in older children. Termination of effect occurs through redistribution of the drug in muscle and fat; thiopental should be used in reduced doses (2 to 4 mg/kg) in children who have low fat stores, such as neonates or malnourished infants. On the other hand, older, healthy children require higher doses of thiopentone to achieve intravenous induction of anesthesia (5-7 mg/kg in children versus 3-5 mg/kg in adults).[20]

Ketamine: It has sympathomimetic effects which usually prevent hypotension. It is induction agent of choice in hemodynamically unstable patients. Airway reflexes are better preserved, however there is no guaranteed protection against aspiration. It causes minimal respiratory depression compared to thiopentone and propofol. There is high incidence of PONV.

Midazolam: Midazolam is the only benzodiazepine approved by the Food and Drug Administration for use in neonates; in this population the half-life is much longer (6 to 12 hours).[42] Midazolam has the fastest clearance of all the benzodiazepines. The combination of midazolam and fentanyl can cause profound hypotension.[19]

Doses: Premedication (PO) — 0.5 mg/kg, Maximum dose (PO) – 20 mg, Sedation (IM) — 0.1-0.15 mg/kg, Sedation (IV) –0.05 mg/kg.

Opioids

Fentanyl: Most commonly used in children because of its rapid onset and brief duration of action as it is more lipid soluble. Termination of the effect of low doses of fentanyl results primarily from redistribution, whereas termination of the effect of high doses depends on elimination. Because the cardiac output of neonates is determined by the heart rate, fentanyl-induced bradycardia may require administration of a vagolytic drug.

Doses: As analgesic 2 µg/kg, as main anesthetic (IV) 50 to 100 µg/kg, as maintenance infusion 2 to 4 µg/kg/h.

Sufentanyl: Sufentanyl has been used primarily for cardiac emergencies; must be administered with caution because severe bradycardia and asystole have been reported when a vagolytic drug was not administered simultaneously.[43]

Remifentanyl: It is a unique potent opioid in neonates. Unlike every other medication used in newborns, its half-life is shorter rather than longer as in older children. This difference may in part relate to the larger volume of distribution with equivalent half-life. Dose is —0.25/1 µg/kg IV bolus, and infusion —0.05/2µg/kg/min.

Muscle Relaxants

Succinylcholine: Infants require significantly higher doses of succinylcholine (2–3 mg/kg) than older children and adults because of the relatively larger volume of distribution (extracellular space). The skeletal muscle relaxation produced by intramuscular administration may last up to 20 minutes.[4] Children are more susceptible than adults to cardiac arrhythmias, hyperkalemia, rhabdomyolysis, myoglobinemia, masseter spasm, and malignant hyperthermia after administration of succinylcholine. If a child unexpectedly experiences cardiac arrest following administration of succinylcholine, immediate treatment for hyperkalemia should be instituted.[19,44]

The response of neonates to nondepolarizing muscle relaxants is quite variable. Immaturity of the neuromuscular junction (particularly in premature neonates)

tends to increase sensitivity, whereas a disproportionately large extracellular compartment dilutes drug concentration. The relative immaturity of neonatal hepatic function prolongs the duration of action for drugs that depend primarily on hepatic metabolism (e.g. pancuronium, vecuronium, and rocuronium).[20] Doses of commonly used nondepolarizing agents are given in Table 17.6.

PREOPERATIVE ASSESSMENT

History and Review of Systems

- Chief complaint and history of present illness: A brief account of the ailment should be made, noting its onset and duration, principle clinical features and its associated symptoms, precipitating, aggravating and relieving factors, the course of illness with attention to the functional limitations it imposes, and the child's response to therapy
- Time of last feed and micturition should be noted
- A quick but systematic history can rule out other major illnesses
- Nurse's charts should be thoroughly checked for fluid intake, urine output, gastric tube aspirations, drains, trends in temperature and other vital parameters.

Physical Examination

- Postconceptional and postnatal age, weight, hemodynamic parameters, respiratory rate, temperature and status of hydration should be noted
- Cry, tone and activity of the neonate, playfulness or otherwise of the infant and consciousness of older child should be assessed
- Hemodynamic instability in the form of cold peripheries, feeble or absent distal pulses and poor capillary refill should be assessed and treated. Central venous pressure (CVP) if available should be noted
- One should look for evidence of respiratory distress in the form of grunting, tachypnea, labored breathing. One can easily check SpO_2 in the preoperative holding area
- Degree of abdominal distention and amount of gastric tube aspiration should be noted
- Type and severity of pain and distress should be noted
- One should look at the profile view of face to rule out receding mandible
- Evidence of sepsis should be looked for especially in abdominal emergencies
- Sites for peripheral and central venous access should be checked.

Table 17.6: Doses, onset and duration of commonly used nondepolarizing agents

Muscle relaxant	*Dose*	*Onset*	*Duration*
Atracurium	0.5 mg/kg	1.5-2 min	30 min
Pancuronium	0.1 mg/kg	2-3 min	60-120 min
Vecuronium	0.1 mg/kg	90 sec-3 min	30-50 min
Rocuronium	0.6 mg/kg	2-3 min	30-45 min
Rocuronium (for rapid sequence induction)	0.9-1 mg/kg	60-90 sec	60 min

Investigations

Hemoglobin, blood urea nitrogen or creatinine, serum electrolytes, blood gases (venous or arterial) and platelet count especially if there is evidence of sepsis suffices in majority of cases.

PREOPERATIVE PREPARATION

Psychological Preparation

The child should be told about the impending surgery with complete honesty depending on the emergency of the situation. The information should be broad and general and not detailed. The child should be introduced to the hospital environment and its personnel in an unthreatening manner.

Premedication

- Premedication is usually not offered in emergencies because of either poor hemodynamic, respiratory or CNS status or lack of time
- However, majority of children would need antisialoggues
- Depending on the case, aspiration prophylaxis is needed in older children
- Vitamin K is usually given to all newborns and this should be verified from the notes before surgery. Neonates are relatively deficient in clotting factors II, VII, IX, and X and at risk of excessive bleeding during surgery.

Preparation for Induction

Operating Room Preparation

- Warm the operating room
- Turn on warming devices (e.g. warming blanket, intravenous line warmer, radiant light heat source)

Anesthesia Equipment (Table 17.7)

- Anesthesia machine check-up
- Monitoring equipment (pulse oximeter, capnograph, anesthetic gas monitor) turned on and checked properly
- Precordial stethoscope with double-stick adhesive should be kept ready
- Proper-size blood pressure cuff, pulse oximeter probe, temperature probe
- Proper-size facemask
- Adequate size oral and nasal airways
- Laryngeal mask airway (LMA) if planned
- Laryngoscope handle and at least two blades of appropriate size
- Three sizes of endotracheal tubes with stylet
- Suction turned on with suction catheter attached to it. Additional sterile endotracheal suction catheters (6F to 10F, depending on the patient's age)
- Nasogastric tubes (10F to 18F)
- Adhesive tape torn and ready for use for ET tube, intravenous line. Authors use micropore stickings for fixation of endotracheal tubes and intravenous lines for neonates
- Intravenous fluid bag connected to appropriate tubings and injection ports
- Intravenous catheters
- Padded or foam rubber head ring or a small pillow should also be at hand.

INDUCTION OF ANESTHESIA[40,45-48]

Intravenous induction is most commonly used as majority of the patients in emergency have intravenous line in place.

In few minor emergency cases where IV access is not in place or in suspected difficult airway cases, inhalation induction will be needed. Two most important pieces of monitoring equipment during an inhalation induction are a precordial stethoscope and a pulse oximeter.

The first sign of anesthetic induction usually is the appearance of nystagmus; then the eyes usually close, respiration becomes slower, regular, and deeper, then shallower and more rapid, and the child becomes still. For some time after that the child may be only half asleep and responds to verbal command. Until he or she no longer reacts to one's voice and the eyelash reflex disappears, nothing should be done to move or stimulate the patient, unless airway obstruction or a similar arises. As soon as anesthesia is induced and the patient can tolerate moderately painful stimuli, an intravenous infusion can be started. An intravenous dose of atropine (0.01 to 0.02 mg/kg) may be given to infants to prevent bradycardia and hypotension caused by inhaled anesthetics, especially with halothane.

For infants, especially those younger than six months, ET intubation is indicated because upper airway obstruction occurs commonly. In addition, vigorous manual ventilation with a mask by inexperienced hands tends to inflate the stomach, resulting in compression of the lower lungs and an increase in the danger of regurgitation and aspiration of gastric secretions. Between 7 to 12 months of age, ET intubation is optional, although it is still recommended unless one is well experienced in the management of infant airways.

A newborn rapidly desaturates following only 15 to 20 seconds of apnea, and beacuse of that, in the past many anesthesiologists believed it was safer to intubate the newborn "awake." However, recent evidence indicates that awake intubation may cause intraventricular hemorrhage in fragile, premature newborns.[45,46] Furthermore, awake intubations are technically more difficult to accomplish and often result in trauma to the vocal cords, hemorrhage, bradycardia, and desaturation secondary to breath holding.

Table 17.7: Criteria for equipment selection in children

	Neonate				*Infant*	*Toddler*	*Small child*	*Large child*
Age	<28 wk	28-34 wk	34-38 wk	>38 wk	1-12 month	1-3 years	3-8 years	8-12 years
Weight (kg)	<1	1-2	2-3	3-5	4-10	8-16	14-30	25-50
Tracheal (ET) tube (mm i.d.)	2.5	3	3.5	3.5	3.5-4	4-4.5	4.5-5.5	5.5-6 (cuffed)
ET depth (cm at lips)	6.5-7.0	7-8	8-9	>9	10-12	12-14	14-16	16-18
Suction catheter (F)	6	6	6	6	8	8	10	12
Laryngoscope blade	00	0	0-1	1	1	1.5	2	3
Mask size	00	0	0	0	0	1	2	3
Oral airway	000-00			00	0 (40 mm)	1 (50 mm)	2 (70 mm)	3 (80 mm)
Laryngeal mask airway	-	-	-	1	1	2	2.5	3

It is preferable to use a "rapid sequence" induction in infants requiring "full stomach" precautions, that is, infants who are at risk for aspiration (e.g. intestinal obstruction, necrotizing enterocolitis) and who have a normal airway on physical examination. After fluid volume resuscitation (10–40 mL/kg of lactated Ringer solution), preoxygenation, pretreatment with atropine (0.15 mg), intravenous administration of thiopental 4–7 mg/kg or propofol 2–3 mg/kg, suxamethonium, and with application of gentle cricoid pressure, ETT is inserted.[47]

Factors which limit its usefulness in children include:[40]

- Difficulty in securing IV access
- Difficulty in application of monitoring pre-induction
- Difficulty in preoxygenation and denitrogenation Neonates and infants have high oxygen consumption relative to FRC and rapidly become hypoxic when apneic. This problem is compounded if preoxygenation has been incomplete because the child is struggling and distressed
- Discomfort associated with cricoid pressure
- Distortion of the view of the laryngeal inlet by cricoid pressure.

In author's experience, unlike adults, majority of the neonates and infants do not tolerate 60 seconds of apnea and they do require gentle puffs with 100 percent O_2. Avoidance of hyperinflation, repeated gastric suction and appropriate anesthetic depth can reduce risk of regurgitation and pulmonary aspiration.

Newborns who do not require precautions for full stomach are in the minority (e.g. myelomeningocele or bladder exstrophy), and in these patients anesthesia can be induced by inhalational or intravenous agents without cricoids pressure.[47] Intubation can be facilitated using rocuronium or atracurium.

Because of the higher position of the larynx and the shape of the epiglottis, intubation may be easier with straight blade laryngoscope. ETT should be properly fixed and should be visible to detect disconnection and kinking.[19,48]

EARLY POSTINDUCTION PERIOD

ETT: ETT should be properly secured with adhesive tapes. Corrugated delivery tubing of the anesthesia breathing circuit should be anchored securely near the ET tube connection to prevent to-and-fro movement with positive pressure ventilation.

Intravenous Catheters: Securing the intravenous insertion site is particularly important in children because they often emerge in an uncooperative and agitated state.

Positioning: The relative lack of subcutaneous adipose tissue, poorly developed musculature, and more superficially located neurovascular structures place infants and children at greater risk for injuries caused by incorrect positioning. Cushioning with cotton gamjees can prevent pressure injuries, especially during procedures that require a long period of time.

Ventilation: Controlled ventilation has traditionally been used during most anesthetics in infants and children even in the absence of neuromuscular blckade (NMB). Authors prefer use of Jackson Rees circuit for ventilation in neonates and thoracic surgeries. With the availability of sophisticated anesthesia ventilators one can use/the same in children. Piston based ventilator can deliver accurate tidal volume. Pressure controlled ventilation has been used in favor of volume control mode in pediatric anesthesia. In part, this is because uncuffed tubes are used or to avoid barotraumas.

Monitoring

Monitoring gadgets are essential as, once positioned and draped on the operating room table, observation, palpation, and even auscultation are difficult, if not impossible. However they cannot replace a vigilant anesthesiologist who will evaluate, interpret, and analyze the patient's condition.[47] Precordial stethoscope, ECG, pulse oximetry, NIBP, and temperature monitoring are minimum essential.

Precordial stethoscope should monitor quality of heart sounds and breath sounds. Whenever patient is operated in the lateral position authors simply tuck the stethoscope under the dependent chest.

SpO_2 of 90 to 95 percent (PaO_2 50–70 mm Hg) is adequate in neonates.[47] If condition demands, authors monitor preductal and postductal SpO_2. Periphery should be kept warm to pick up the signals properly. Whenever there is loss of SpO_2 signals underlying circulatory problems should be suspected unless proved otherwise. One should immediately check pulse volume and blood pressure rather than just attributing to faulty monitor or sensor. In prolonged surgery it is important to change probe site to avoid prolonged pressure and burns; as such poor peripheral perfusion increases the risk of the same.

In author's institution use of non-invasive blood pressure monitor is a must as it gives very good idea about circulation as most of the time peripheral pulses are not accessible (either under drapes or inside limb splints).

Microstream capnograph can give idea about circulation, respiration, metabolism and machine related problems.

In majority of abdominal emergency central venous pressure monitoring may be needed. In few major emergencies urinary bladder is catheterized, otherwise in majority cases soakage is monitored. Monitoring of blood loss is very important. It is important to identify at least one peripheral pulse site which can be monitored throughout. If not, axillary artery should be palpated intermittently. In majority of emergencies one may need at least one pre- and postoperative blood gas, Na^+, K^+, Ca^{2+} (if available), and glucose levels.

Fluid and Electrolyte Management

Fluid requirements can be considered as deficit fluids, maintenance fluids and replacement fluids.[49]

Deficit fluid: This refers to the management of fluid and electrolyte loss that occurs prior to presentation for surgery. This may be due to disease process or preoperative restriction of orals. The therapy has three components: (i) estimation of dehydration severity (ii) determination of fluid deficit type, and (iii) deficit repair.

Dehydration severity: Estimated from the history, clinical and laboratory findings (Table 17.8).

For the same degree of dehydration, clinical symptoms are worse for hyponatremic dehydration than for hypernatremic dehydration. Treatment of severe dehydration must begin with infusion of balanced salt solution like ringer lactate (RL) or normal saline (NS) 20 ml/kg even before the lab results are available. Potassium should be replaced only after adequate renal perfusion is established, acidosis is corrected and child starts passing urine.

Unsuspected hypovolemia when combined with anesthetic drugs can be catastrophic. Thus, prior to induction of general anesthesia in neonates requiring emergency surgery, fluid bolus of at least 20 ml/kg of lactated Ringer solution should be given in an attempt to ensure an adequate preload.[41]

Maintenance fluid requirements: The fluid for maintenance therapy replaces ongoing fluid and electrolyte losses during surgery due to insensible/evaporative and urinary losses. Insensible water loss (IWL) forms about 30 to 35 percent of ongoing losses. In the newborn infant 1/3 IWL occurs via the respiratory tract and 2/3 via the skin. Maintenance of fluid requirement is calculated on an hourly basis depending on the body weight by Holliday-Segar formula.[1] (Table 17.9). Fever increases calorie needs by 10 to 12 percent per degree centigrade elevation.

Neonatal regimens: Fluid requirement in neonates for first day according to birth weight is as follows: >2.5 kg – 60 ml/kg/day; 2-2.5 kg – 70 ml/kg/day; 1.5-2 kg – 80 ml/kg/day; 1-1.5 kg – 90 ml/kg/day. Then, for everyday add 10 ml/kg/day till fifth day. With use of overhead warmer, phototherapy and double surface phototherapy additional 10 percent, 20 percent and 30 percent is added in fluid requirement. Whenever there is severely raised C reactive protein or sepsis 40 percent extra fluid is given. Fluid restriction (less by one-third) is needed in presence of associated cardiac or renal disease.

Electrolyte requirements: Na^+ = 2.5 mmol/100 kcal expended, K^+=2.5 mmol/100 kcal expended, Cl^- = 5 mmol/100 kcal Expended.

Glucose requirement in maintenance fluids: Dextrose = 25 g/100 kcal expended. Glucose containing fluids are usually unnecessary other than in premature babies, neonates, and some other at risk children, e.g. those

Table 17.8: Dehydration severity

	Infant < 10 kg		*Child > 10 kg*		*Clinical signs and symptoms*
% dehydration	*% dehydration*	*Estimated fluid deficit*	*% dehydration*	*Estimated fluid deficit*	
Mild	5%	50 ml/kg	3-4%	30 ml/kg	Increased thirst, tears present, mucous membrane moist, external jugular visible when supine, capillary refill time < 2 secs, urine specific gravity > 1.020
Moderate	10%	100 ml/kg	6-8%	60 ml/kg	Dry mucous membrane, decreased tears, pulse rate may be increased, fontanelles may be sunken, oliguria, capillary refill time 2-4 secs, decreased skin turgor.
Severe	15%	150 ml/kg	10%	90 ml/kg	Tears absent, mucous membrane dry, eyes sunken, tachycardia, slow capillary refill, poor skin turgor, cold extremities, orthostatic hypotension to shock, apathy, somnolence.
Shock	> 15%		> 10%		Physiologic decompensation, insufficient perfusion to meet end organ demand, poor oxygen delivery, decreased blood pressure.

Table 17.9: Average fluid/energy requirement (Holliday-Segar formula)

Body weight (kg)	*Kcal or ml/kg/hr*	*Kcal or ml/kg/day*
0 to 10	4 ml/kg/hr	100 ml/kg/day
10 to 20	40 ml + 2 ml/kg/hr for weight > 10 kg	1000 + 50 ml/kg/day for weight > 10 kg
>20	60 ml + 1 ml/kg/hr for weight > 20 kg	1500 + 25 ml/kg/day for weight > 20 kg

receiving parenteral nutrition. The present day recommendation is to use replacement fluids which are free of dextrose or should not have more than 1 percent dextrose. Whatever the case may be, hourly blood sugar estimations are recommended to ensure adequate blood sugar levels without inducing hyper/hypoglycemia.[45] It is not practical to measure sugar every hour. Therefore it is preferable to use 10 percent glucose for premature neonate, 5 percent glucose for term neonates, and 2.5 percent glucose for older infants.

Replacement fluids: Third space loss, i.e. certain disease processes (GI illness), surgical handling and tissue trauma leads to capillary leak which translocates the fluid to non-functional compartment. This fluid resembles ECF with small amount of proteins. This fluid needs to be replaced with balanced salt solution. Lactated Ringer is the fluid of choice for intraoperative period. The replacement for third space losses depends on the duration and severity of surgical handling. For mild, moderate and severe tissue trauma 2, 4, or 6 ml/kg fluid is given.

As a general rule, once a child has received one blood volume of crystalloids, some colloids must be given to minimize critically low levels of colloidal oncotic pressure.

Replacement of other losses: Other perioperative losses like GI fluids, sweat and burns losses have to be replaced ml for ml and mEq for mEq. For optimum replacement of electrolytes, composition of these body fluids must be known. Burns patients in addition also loose proteins. In cases of GI illness, the site and duration of obstruction determines the nature of the fluid loss and influences body response. Obstructions which are < 3 days will have more of ECF deficit (80%) than ICF deficit (20%). Where as in obstructions for more than 3 days have ICF deficit of 40 percent and ECF deficit of 60 percent. Knowledge of composition of body fluids can guide in appropriate replacement[43] (Table 17.10).

For the gastric loss one can provide hydrogen donor, i.e. ammonium. But that is hardly ever necessary. One can simply provide KCL, which will allow kidney to retain H^+. For the replacement of lost gastric juice, a good choice would be 5 percentD1/2 NS + KCL. Another solution is to administer Isolyte G in ml to ml basis. The electrolyte pattern of Isolyte G [Cl^- 149 mEq/l, Na^+ 65 mEq/l, K^+ 17 mEq/l, NH^+ 70 mEq/l (a hydrogen ion donor)] is the perfect answer for replacement of gastric juice.

An acceptable approximation for bile would be lactated Ringer with perhaps additional potassium. Plasmalyte R with 5 percent Dextrose and Isolyte E are virtually identical: [Cl^- 103 mEq/l, Na^+ 140 to 141 mEq/l, K^+ 10 mEq/l, acetate 47 to 49 mEq/l (gets converted to bicarbonate)] and are the solution for replacement of bile.

Replacement of blood loss: In children, blood loss should be replaced either with crystalloids, colloids or blood and blood products like packed RBC (PRBC). The intraoperative blood loss can be judged with clinical observation and suction bottle estimation or swab weighing and hematocrit estimation. With the increasing awareness of risks and cost of transfusion in the past decade, there is increased trend towards restricted blood transfusion practices. According to which a minimum hematocrit of 30 percent in older children and 40 percent for neonates is accepted as safe enough to ensure an adequate O_2 transport without the need for blood transfusion.

Under 10 percent of blood loss of estimated blood volume, no blood is required and it can be managed with crystalloid. For each ml of blood loss, 3 ml of balanced salt solution can be given safely. Colloid solutions like Dextrans, Gelatins and Hetastarch are given in the dose of 1 ml per 1 ml blood loss. Human albumin can also be used for maintaining the intravascular compartment. In cases of capillary leak syndrome, albumin does not help as it does not remain in intravascular compartment and hence first the capillary leak should be tackled.

Estimated blood volume (EBV) can be calculated by knowing approximate blood volume/kg in different

Table 17.10: Composition of various body fluids

Body fluid or IV fluid	*Electrolytes (mEq/l)*			
	Na^+	*K^+*	*Cl^-*	*HCO_3^-*
Gastric	70	5-15	120	0
Pancreas	140	5	50-100	100
Small bowel	100-140	5-15	90-130	-
Bile	130	5	100	40
Ileostomy	130	15-20	120	25-30
Diarrhea	50	35	40	50
Burns	140	5	110	-
Sweat	10-30	3-10	10-35	-

age groups. When the blood loss is more than 10 percent of the estimated blood volume it has to be replaced with blood to maintain the oxygen carrying capacity.

Temperature maintenance: Accurate and close monitoring of body temperature is essential for keeping pediatric patients in the normothermic range. Rectal or esophageal thermistor probes reliably measure core temperature. Axillary probes, which are more easily placed, record temperatures 0.7 to 1.0°C less than core readings and are more appropriate for older children undergoing surgery without expected major hemodynamic changes. For the best results with this method, the sensing tip should be located as near the axillary artery as possible by taping the probe to the lateral chest wall and then adducting the arm down close to the body. The safe range for a child's core temperature is approximately 35.5 to 37.5°C.

EMERGENCE AND EXTUBATION

- Carried out when the child is normothermic, normovolemic and has a minimal concentration of expired volatile agent.
- Neuromuscular blockade is reversed with neostigmine 50 µg/kg and atropine 20 µg/kg or glycopyrronium bromide 10 µg/kg.
- Gentle suction of pharynx and nostrils (nasal breathing).
- Nasogastric tube aspirated if present.
- Child is extubated 'awake' when breathing adequately and moving vigorously.

BASIC POSTOPERATIVE CARE

The anesthetist should report to the recovery room personnel any intraoperative problems that occurred. The airway should be maintained to assure adequacy of ventilation and oxygenation and any unexpected findings reported to the anesthetist. Vital signs should be taken frequently in the first hour and pain treated. The child may return to the ward when the observations are stable, he is fully conscious and his pain is controlled.

Postoperative Pain Management

Methods of treating postoperative pain in children include the use of systemic analgesics and local anesthetic agents. The systemic analgesics can be divided into nonopioids and opioids.

Nonopioid Analgesics (For Mild or Moderate Pain)

Paracetamol (acetaminophen): Oral loading dose 20 mg/kg followed by 15 mg/kg 4-hourly, IV: 15 mg/kg 6-hourly, Rectal: loading dose 40 mg/kg (20 mg/kg in neonates) followed 20 mg/kg 6-hourly. Maximum dose by oral and rectal routes 90 mg/kg/day above 3 months of age (4 g/day in adolescents); 60 mg/kg/day in neonates and infants up to 3 months of age.

NSAIDS: This group of drugs has become extremely popular for treating postoperative pain in children as they are effective with few side effects and produce an opioid sparing action. They should be avoided in patients with coagulopathy (because of a tendency to prolong postoperative bleeding), nephropathy, gastropathy and asthma. Diclofenac 1 to 3 mg/kg per day in divided doses is widely used. It is also available as a suppository. **NB**: Aspirin should not be used for children under 12 years because of the association with Reye's syndrome. Maximum by all routes 3 mg/kg/day or 150 mg/day.

Opioids (for severe pain): Opioids may be administered by IM, IV or oral routes. Children are sensitive to opioids and doses should be reduced accordingly. They should not be given to children < 5kg. Slow IV administration avoids the need for painful intramuscular injections but the child should be closely observed whilst this is given.

Local Anesthetic Techniques

Especially useful in emergency situation as it decreases need of systemic anesthetic requirement and therefore hastens the recovery and decreases the need for postoperative ventilation.

Local wound infiltration with bupivacaine 0.25 percent at the conclusion of surgery is very effective and is extremely simple and safe. It reduces the need for additional measures.[1]

Other regional blocks are used in specific situations, e.g. intercostal blocks following thoracotomy.

Caudal anesthesia, caudal narcotics and child-parent-nurse–controlled analgesia have all been accepted by anesthesiologists and children. Volume required = 0.5 ml/kg – lumbos caral, 1.0 ml/kg – thoracolumbar, 1.5 ml/kg – midthoracic and should not exceed 20 ml.

Epidural analgesia: Epidural anesthesia is recommended for all major abdominal, retroperitoneal, pelvic, and thoracic surgeries requiring long-lasting pain relief, thus requiring placement of an epidural catheter to allow repeat injections or continuous infusion of local anesthetic. The volume of anesthetic solution depends on the upper level of analgesia required for completion of the surgery; around 0.1 ml per year of age is necessary to block one neuromere.[46] Usual volumes of injectate range from 0.5 to 1 ml/kg (up to 20 ml) .

Consideration for Premature Newborns[33]

An infant delivered before 37 week from the first day of the last menstrual period is defined as premature infant. Degree of prematurity is by the gestational age and birth weight. When baby is born between 36 to 37 wks and < 2.5 Kg it is said to be borderline or near term, when born between 31 to 36 wks and <1.5 Kg it is considered as moderately premature and when born between 24 to 31 wks with birth weight < 1 Kg, considered as severely premature.

Premature babies usually come for PDA ligation, laparotomy for NEC or spontaneous bowel perforation, fundoplication for unresolving gastroesophageal reflux, vitrectomy or laser surgery for retinopathy of prematurity, V-P shunt for obstructive hydrocephalus after IVH.

The Conduct of Anesthesia

- Two pulse oximeters should be used, one for preductal other for the postductal circulation, with less adhesive to minimize skin damage.
- The noninvasive blood pressure measurement can fracture very poorly ossified, calcium-deficient bones.
- The adhesive on electrocardiogram (ECG) stickers can also damage the skin.
- Because of very small tidal volumes and low maximum expiratory flow rate, end-tidal CO_2 measurement will be rendered less accurate.
- Invasive blood pressure or central venous pressure measurement, although desirable, is so technically challenging and potentially dangerous that often these are not employed.
- While maintenance of normothermia is very important, temperature monitoring itself poses risks for these tiny patients. Perforation is possible despite meticulous and careful placement of rectal or esophageal probes.
- Intravenous fluids and blood products: Hypoglycemia should be expected unless supplemental glucose is supplied. Maintenance glucose for the premature babies is 4 to 6 mg/kg/minute. Estimated maintenance fluid requirement in a preterm baby is 100 ml/kg/day. During surgery maintenance fluid should be isotonic, e.g. Hartmanns solution, 0.9% NS.[49]
- Replacement of blood products must be done promptly but also slowly.[13,14] A 10 ml syringe of PRBCs rapidly delivered into the vascular system over < 1 minute will increase the blood volume by 10 percent. This is analogous to administering 2 units of PRBC to an adult over 1 minute. It is also very important that the blood products be warmed to minimize hypothermia in these patients. Calcium supplementation is essential.
- Anesthesia can be induced with ketamine or thiopentone sodium depending on the severity of the illness. Hypertension and increased levels of stress hormones will have deleterious effects on the patients.

Choanal Atresia[47,48]

Choanal atresia occurs in approximately 1 in 7, 000 live births. Classification of the blocked segment may be divided into bony atresia (30%) and bony-membranous atresia (70%). Choanal stenosis is not a blockage but rather a narrowing of < 6 mm. Fifty percent of patients with choanal atresia have other congenital anomalies.[48] Choanal atresia may be partial or one of a constellation of congential abnormalities known as the CHARGE association (coloboma, heart disease, Artesia [choanal], retarded growth, genital abnormalities, ear deformity). Other associated anomaly can be Apert syndrome: craniosynostosis,[50] syndactylism, difficult airway; Fraser syndrome: laryngeal/tracheal stenosis, congenital heart disease, genitourinary anomalies, renal agenesis/hypoplasia; Treacher Colins syndrome; DiGeorge syndrome.

Choanal atresia can be unilateral or bilateral. Because neonates are obligate nose breathers, bilateral choanal atresia frequently presents as immediate onset of respiratory distress. Obstruction of the nasal cavity can present with apneic episodes and "cyclic" cyanosis, which are exacerbated by feeding and improved with crying. The use of an oral airway or McGovern nipple (a nipple modified with enlarged perforations at the tip) acts as an alternative temporary airway. Unilateral choanal atresia is usually asymptomatic.

Inability to pass a 6-Fr catheter through the nasal cavity to more than 32 mm, coupled with an endoscopic examination, verifies the suspected diagnosis. Axial computed tomography (CT) remains the study of choice to delineate the type of atresia and aid with operative planning (transpalatal vs. transnasal approach). For bilateral choanal atresia surgical correction occurs in the neonatal period and involves a transnasal correction using CO_2 or neodymium:yttrium-aluminum-garnet (Nd:YAG) lasers. The nasal passage is stented open for 3 to 5 weeks to improve airway patency. The surgical technique generally involves an endoscopic approach where a vertical mucosal incision is made in the posterior bony septum and a perforation within the atretic plate is created.[47] Once the atretic areas are

removed, nasal stents are placed to prevent reclosure from scarring. The stents are left in place for up to 3 weeks. Disadvantages of the transpalatal approach are the procedure's long operative time and large blood loss. Additionally, malocclusion occurs in 50 percent of patients and oronasal fistulas are not uncommon. In patients with unilateral choanal atresia surgical treatment is usually carried out at any time during childhood. The surgical approach can be transnasal or transpalatal.

Anesthesia Consideration

For infants having the CHARGE association any underlying cardiac issue must be addressed. Oral airway is needed during induction. Oral RAE tube is inserted after an inhalational or intravenous induction. The anesthetic agent is titrated to allow the patient to be extubated as awake as possible with the patient's airway reflexes intact. However, if the procedure has been lengthy, airway edema is present, or hemodynamic instability is present, then the patient should remain intubated until these issues have resolved. Proper humidification and maintenance of stent patency is essential.

REFERENCES

1. Strafford MA. Cardiovascular Physiology in Infants and Children. In: Motoyama EK, Davis PJ, (Eds). Smith's Anesthesia for Infants and Children. 7th Edn. Philadelphia: Mosby Elsevier; 2006;70-104.
2. Scammon RE, Norris EH. On the time of obliteration of the fetal blood passages (foramen ovale, ductus arteriosus, ductus venosus). Anat Rec 1918;15:165.
3. Mancuso TJ. Anesthesia for the Preterm Newborn. In: Holzman RS, Mancuso TJ, Polaner DM, (Eds). A Practical Approach to Pediatric Anesthesia. 1st edn. Boston: Lippincott Williams and Wilkins; 2008;602-09.
4. Coté CJ. Pediatric Anesthesia. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL, (Eds). Millers Anesthesia. 7th edn. Philadelphia: Mosby Elsevier; 2009;2559-98.
5. Rychik J. Fetal cardiovascular physiology. Pediatr Cardiol 2004;25:201.
6. Motoyama EK. Respiratory Physiology in Infants and Children. In: Motoyama EK, Davis PJ, (Eds). Smith's Anesthesia for Infants and Children. 7th edn. Philadelphia: Mosby Elsevier; 2006;12-52.
7. Rigatto H, Brady JP, Chir B, Rafael de la Tone Verduzco R. Chemoreceptor reflexes in preterm infants. The effect of gestational and postnatal age on the ventilatory response to inhaled carbon dioxide. Pediatrics 1975;55:614.
8. Cross KW, Oppe TE. The effect of inhalation of high and low concentrations of oxygen on the respiration of the premature infant. J Physiol (Lond) 1952;117:38.
9. Ceruti E. Chemoreceptor reflexes in the newborn infant. Effect of cooling on the response to hypoxia. Pediatrics 1966;37:556.
10. Perlstein PH, Edward NK, Sutherland JM. Apnea in premature infants and incubator-air-temperature changes. N Engl J Med 1970;282:461.
11. Dripps RD, Comroe Jr JH. The respiratory and circulatory response of normal man to inhalation of 7.6 and 10.4 percent CO_2 with a comparison of the maximal ventilation produced by severe muscular exercise, inhalation of CO_2 and maximal voluntary hyperventilation. Am J Physiol 1947;149:43.
12. Albersheim S, Boychuk R, Seshia MMK, et al. Effects of CO_2 on immediate ventilatory response to O_2 in preterm infants. J Appl Physiol 1976;41:609.
13. Rigatto H. Apnea. In: Thibeault DW, Gregory GA, (Eds). Neonatal pulmonary care. Norwalk, CT: Appleton-Century-Crofts; 1986.
14. Rigatto H, Brady JP, de La Torre Verduzco R. Chemoreceptor reflexes in preterm infants. The effect of gestational and postnatal age on the ventilatory response to inhalation of 100 and 15% oxygen. Pediatrics 1975;55:604.
15. Kelly DL, Stellwagen LM, Kaitz E, Shannon DC. Apnea and periodic breathing in normal full-term infants during the first twelve month. Pediatr Pulmonol 1985;1:215.
16. Glotzbach SF, Tansey PA, Baldwin RB, Ariagno RL. Periodic breathing in preterm infants: Influence of bronchopulmonary dysplasia and theophylline. Pediatr Pulmonol 1989;7:78.
17. Fenner A, Schalk U, Hoenicke H, Adelheid W, Thomas R. Periodic breathing in premature and neonatal babies: Incidence, breathing pattern, respiratory gas tensions, response to changes in the composition of ambient air. Pediatr Res 1973;7:174.
18. Brooks JG. Apnea of infancy and sudden infant death syndrome. Am J Dis Child 1982;136:1012.
19. Morgan GE, Mikhail MS, Murray MJ, (Eds). Pediatric anesthesia. In: Clinical Anesthesiology. 4th edn. McGraw-Hill Companies; 2006; 922-50.
20. Rusy L. Pediatric Anesthesia Review. 1998;8(2):1-8.
21. Bernet V, Dullenkopf A, Maino P, Weiss M. Outer diameter and shape of paediatric tracheal tube cuffs at higher inflation pressures. Anesthesia 2005;60:1123-28.
22. Keens TG, Bryan AC, Levison H, Ianuzzo CD. Developmental pattern of muscle fiber types in human ventilatory muscles. J Appl Physiol 1978;44:909-13.
23. Lee C, Luginbuehl I, Bissonnette B, Mason LJ. Peditric Diseases. In: Hines RL, Marschall KE, (Eds). Stoelting's Anesthesia and Co-Existing Disease. 5th edn. America: Churchill Livingstone; 2008; 658-71.

24. Crone RK. Pediatric Intensive Care. ASA Refresher Courses in Anesthesiology, vol 9. Philadelphia: JB Lippincott; 1981.
25. O'Rourke PP, Crone RK. The respiratory system. In: Gregory G, (ed). Pediatric Anesthesia. 2nd edn. New York: Churchill Livingstone; 1989;63.
26. Schultz DM, Giordano DA, Schultz DH. Weights of organs of fetuses and infants. Arch Pathol 1962;74:244.
27. Rudolph AM, Heymann MA, Teramo AW, et al. Studies on the circulation of the previable human fetus. Pediatr Res 1971;5:452.
28. Schwartz GL, Brion LP, Spitzer A. The use of plasma creatinine concentration for estimating glomerular filtration in infants, children and adolescents. Pediatr Clin North Am 1987;34:571.
29. Demetrius Ellis. Regulation of Fluids and Electrolytes in Infants and Children In: Motoyama EK, Davis PJ, (Eds). Smith's Anesthesia for Infants and Children. 7th edn. Philadelphia: Mosby Elsevier; 2006;117-19.
30. Aperia A, Zetterstrom R. Renal control of fluid homeostasis in the newborn infant. Clin Perinatal 1982;9:523.
31. Winters RW. Principles of pediatric fluid therapy. 2nd edn. Boston: Little Brown; 1982.
32. Seri I, Ramanathan, Evans JR. Acid-base, fluid, and electrolyte management. In: Taeusch HW, Ballard RA, Gleason CA, Avery ME, (Eds). Avery's diseases of the newborn. 8th edn. Philadelphia: Elsevier Saunder's; 2005;385.
33. Koo WWK, Tsang RC. Calcium and Magnesium Homeostasis. In: Avery GB, MacDonald MG, Seshia MMK, Mullett MD, (Eds). Avery's neonatology: Pathophysiology and management of the newborn. 6th edn. Philadelphia: Lippincott Williams and Wilkins; 2005;862.
34. Rennie JM, Roberton NRC, (Eds). Textbook of Neonatology. 3rd edn. Edinburgh: Churchill Livingstone; 1999.
35. Taeusch HW, Ballard RA, (Eds). Avery's Diseases of the Newborn 7th edn. Philadelphia: W. B. Saunders Company; 1998.
36. Ward RM, Mirkin BL. Perinatal/neonatal pharmacology. In: Brody TM, Larner J, Minnema KP, (Eds). Human pharmacology: Molecular to clinical. 3rd edn. St Louis: Mosby -Year Book; 1998;873-83.
37. Gow PJ, Ghabrial H, Smallwood RA, et al: Neonatal hepatic drug elimination. Pharmacol Toxicol 2001;88:3-1.
38. Lerman J. Neonatal anesthesia. In: Healy T, Cohen PJ, (Eds). Wylie And Churchill- Davidsons A practice of Anesthesia. 7th edn. London: Arnold; 2003;941-56.
39. Meakin GH. Developmental Pharmacology. In: Holzman RS, Mancuso TJ, Polaner DM, (Eds). A Practical Approach to Pediatric Anesthesia. 1st edn. Boston: Lippincott Williams and Wilkins; 2008; 37.
40. Lesch V. Pediatric Pharmacology and Fluid Administration. In: Doyle E, (Eds). Pediatric Anesthesia. 1st edn. Oxford University Press; 2007;26-40.
41. Parke TJ, Stevens JE, Rice AS, et al. Metabolic acidosis and fatal myocardial failure after propofol infusion in children: Five case reports. BMJ 1992;305:613.
42. Burtin P, Jacqz-Aigrain E, Girard P, et al. Population pharmacokinetics of midazolam in neonates. Clin Pharmacol Ther 1994; 56:615-25.
43. Starr NJ, Sethna DH, Estafanous FG. Bradycardia and asystole following the rapid administration of sufentanil with vecuronium. Anesthesiology 1986;64:521-23.
44. Rosenberg H, Gronert GA. Intractable cardiac arrest in children given succinylcholine. Anesthesiology 1992;77:1054.
45. Friesen RH, Honda AT, Thieme RE. Changes in anterior fontanel pressure in preterm neonates during tracheal intubation. Anesth Analg 1987;66:874-78.
46. Friesen RH, Honda AT, Thieme RE. Perianesthetic intracranial hemorrhage in preterm neonates. Anesthesiology 1987;67:814-16.
47. Yaster M, Rossberg MI. Anesthesia for newborn surgical emergencies. In: Longnecker DE, Brown DL, Newman MF, Zapol WM, (Eds). New York: McGraw-Hill Companies; 2008;1502-10.
48. From Niermeyer S, Kattwinkel J, Van Reempts P, et al. International Guidelines for Neonatal Resuscitation: An excerpt from the Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science. Contributors and Reviewers for the Neonatal Resuscitation Guidelines. Pediatrics 2000;106:E29.
49. APA Consensus Guidelines on Perioperative Fluid Management in Children v l. l. Available from http://www.apagbi.org.uk/docs/ Perioperative_Fluid_ Management_2007.pdf (September 2007).
50. Papay FA, McCarthy VP, Eliachar I, et al. Laryngotracheal anomalies in children with craniofacial syndromes. J Craniofacial Surg 2002;13:351-64.

18 Anesthetic Management of Neonatal Thoracic Emergencies

Anila D Malde

KEY POINTS

- Congenital diaphragmatic hernia (CEH), tracheoesophageal fistula (TEF) and congenital lobar emphysema (CLE) are three most important neonatal thoracic emergencies.

CDH

- Most commonly occurs through left posterolateral foramen of Bockdalek.
- Associated CVS abnormalities increases mortality.
- Pulmonary hypoplasia, hypertension and immaturity, deficiency of surfactant and antioxidant enzymes and cardiac impairment leads to hypoxia, hypercarbia, acidosis, myocardial dysfunction and right to left shunting.
- Preoperative stabilization and a delayed surgical approach are preferred now.
- Medical management consists of conventional or high-frequency oscillatory ventilation, permissive hypercarbia, nitric oxide and extracorporeal membrane oxygenation.
- Anesthetic management comprises of: gastric decompression, avoidance of mask ventilation and nitrous oxide, rapid-sequence induction, opioid-based anesthetic and prevention of barotrauma.
- Contralateral pneumothorax, pulmonary hypertension and abdominal compartment syndrome are the potential complications.
- In the perioperative period, sudden, unexplained return to a state of persistent pulmonary hypertension and clinical deterioration is frequent.
- Measures to prevent increases in pulmonary vascular hypertension (PVR) and promote a decrease in PVR, should be instituted.
- Postoperatively they require NICU care and ventilatory support.

TOF

- Esophageal atresia (EA) with distal TEF is the most common type.
- TEF can be part of the VATER syndrome or VACTERL association.
- EA is diagnosed soon after birth when an oral catheter cannot be passed beyond 10 to 12 cm into the stomach or when the neonate exhibits cyanosis, coughing, and choking during oral feedings.
- Leakage of gas through the fistula has to be avoided by proper positioning of ETT. In practice, the anesthetist often inserts the tube 'too deep', slowly withdrawing it until both lungs are ventilated.
- Air/O_2/opioid (e.g. fentanyl 1–2 μg/kg/h), propofol or low-dose volatile technique and manually controlled ventilation with low PIP are used.
- Desaturation can occur as a result of the patient's anatomy, operative positioning, and surgical manipulations. Intermittent release of surgical retraction is essential.
- Loss of breath sounds and the end-tidal carbon dioxide ($ETCO_2$) tracing can be because of secretions or blood in the ETT or from kinking of the trachea during surgical manipulation.
- Multimodality analgesic approach consisting of IV fentanyl 0.5 to 1 μg/kg, rectal paracetamol 30 mg/kg and caudal 0.125 percent, 1.25 ml/kg bupivacaine is recommended.
- Babies which are good weight, full-term without CHD or intraoperative cardiopulmonary complications, and are normothermic are extubated; others are continued with intubation and ventilation.

CLE

- CLE refers to the postnatal abnormal overdistention of an otherwise anatomically normal lobe of the lung.
- It is characterized by expiratory air trapping within the lobe, which produces compression and atelectasis of adjacent lobes. If it is severe, mediastinal shift and impaired venous return can occur. Impaired gas exchange is common in CLE.
- It presents with tachycardia, tachypnea, retractions, asymmetric chest expansion with focal hyperresonance and diminished breath sounds over the affected lobe.
- Chest radiograph may reveal hyperinflation of the involved lobe, atelectasis of adjacent lung, mediastinal shift, and flattening of the ipsilateral diaphragm.
- Lobectomy via thoracotomy or thoracoscopy is done.
- A smooth inhalation induction with sevoflurane and oxygen is often used, with positive-pressure ventilation minimized until the chest is open.
- Nitrous oxide is avoided.
- If the lobe expands suddenly, the surgeon should be ready to open the chest immediately and relieve the pressure and prevent sudden cardiopulmonary decompensation.
- Extubation is desirable because an air leak can develop at the bronchial suture (or staple) line with IPPV.

CONGENITAL DIAPHRAGMATIC HERNIA

Congenital diaphragmatic hernia (CDH) is the most challenging and demanding of all neonatal surgical emergencies. It is characterized by a defect in the diaphragm that allows the herniation of abdominal contents into the thoracic cavity. The incidence is approximately 1 in 3000 to 5000 births.[1] Classification is shown in Table 18.1.

Associated anomalies are present in 10 to 50 percent of patients with CDH (Table 18.2),[4-9] these anomalies confer a 2-fold relative risk of mortality when compared with patients with isolated CDH.[8,9] The most common of these involve the central nervous system (CNS) and the cardiovascular system (CVS). In Greenwood et al's[4] series of 48 infants, 23 percent had associated CVS abnormalities. Mortality in this group was 73 percent in contrast to 27 percent in those without CVS abnormalities.

Morbidity and Mortality

Traditionally, the mortality rate from CDH was in the range of 40 to 50 percent. The new strategy of permissive hypercapnia and delayed surgical repair has resulted in survival rates of >75 percent in some centers. However, the morbidity remains high in survivors.[10]

Indicators of high-morbidity and mortality are:[9,11-15]

- Diagnosis prior to 25 weeks' gestation
- Large defects (lung to head ratio <1)
- Liver herniation into the thorax
- Pulmonary hypoplasia
- Persistent fetal circulation (PFC)
- Associated anomalies
- Identification of the stomach within the chest
- Pneumothorax (reflecting lung hypoplasia)
- Right-sided defect
- Symptoms severe enough to require endotracheal intubation immediately after birth
- Weight less than 1000 g
- Infants born at less than 33 weeks' gestation
- PAO_2-PaO_2 gradient greater than 500.

Favorable features include:[12,14,16]

- Presence of aerated ipsilateral lung
- Aeration in the contralateral lung of more than 50 percent
- Antenatal diagnosis
- Neonatal stabilization and delayed surgery
- Avoidance of ventilator-induced lung injury.

Table 18.1: Classification[1-3]

Name	*Defect*	*Organs involved*	*Incidence*
Left Bochdalek	Left posterolateral foramen of Bochdalek	Small and large bowel as well as solid organs into the thoracic cavity	85%
Right Bochdalek	Right posterolateral foramen of Bochdalek	Liver and large bowel	13%
Bilateral Bochdalek	Bilateral foramen of Bochdalek		Extremely rare
Morgagni	Anterior midline through the sternocostal hiatus of the diaphragm. 90% of cases occurring on the right side		2%
Paraesophageal	Esophageal hiatus	Stomach	Rare

Table 18.2: Associated anomalies[4-9]

Organ system	*Anomalies*	*Incidence*
Cardiovascular	Hypoplastic left heart syndrome, atrial and ventricular septal defects, coarctation, tetralogy of Fallot	23%
Genitourinary	Hypospadias, hydronephrosis, renal dysplasia	23%
Gastrointestinal	Tracheoesophageal fistula, malrotation, various atresias, omphalocele	17%
Central nervous system	Spina bifida defects, meningomyelocele, hydrocephalus, cerebral dysgenesis	
Musculoskeletal	Syndactyly, amelias	
Chromosomal	Trisomy 18, trisomy 21, trisomy 13	10%
Syndromes	Fryn (autosomal recessive syndrome with CDH, cleft lip or palate, and distal digital hypoplasia), Cornelia de Lange (an autosomal dominant syndrome with characteristic facial features, hirsutism, and developmental delay), Brachmann-de Lange, Pallister-Killian or tetrasomy 12p mosaicism (coarse facial features, aortic stenosis, cardiac septal defects, and abnormal genitalia)	10%

Clinical Presentation

Severe cases present in the first minutes to hours of life. Less severe ones present within 24 hours after birth. The classic triad of CDH consists of cyanosis, dyspnea, and apparent dextrocardia.[2]

Physical examination reveals a scaphoid abdomen, bulging chest, decreased breath sounds, distant or right-displaced heart sounds, and bowel sounds in the chest. Signs of respiratory distress (retractions, cyanosis, grunting respirations) can be evident. Profound hypoxemia reflects reopening of the ductus arteriosus and persistent fetal circulation.[17,18]

Diagnosis

Radiographic examination of the chest (Fig. 18.1) shows a bowel gas pattern in the chest, a gastric tube in the affected chest, mediastinal shift, little lung tissue at the right costophrenic sulcus, and most ominously, a contralateral pneumothorax. Right-sided lesions are difficult to differentiate from diaphragmatic eventration and lobar consolidation.[13]

Continuous pulse oximetry is valuable in the diagnosis and management of primary pulmonary hypertension (PPHN). Place oximeter probes at preductal (right-hand) and postductal (either foot) sites to assess for a right-to-left shunt at the ductus arteriosus level.

Assess ABG. Hypoxemia, hypercarbia, and respiratory or metabolic acidosis depend on the degree of pulmonary hypoplasia, PPHN, right-to-left shunting, and ventricular function.

Early echocardiography, which may reveal cardiac defects, decreased left ventricular mass, poor ventricular contractility, pulmonary and tricuspid valve regurgitation, and right-to-left shunting through the ductus arteriosus and/or the foramen ovale.[19] Repeated echocardiography is recommended to measure changes in the pulmonary artery pressure, and flow across the ductus arteriosus.

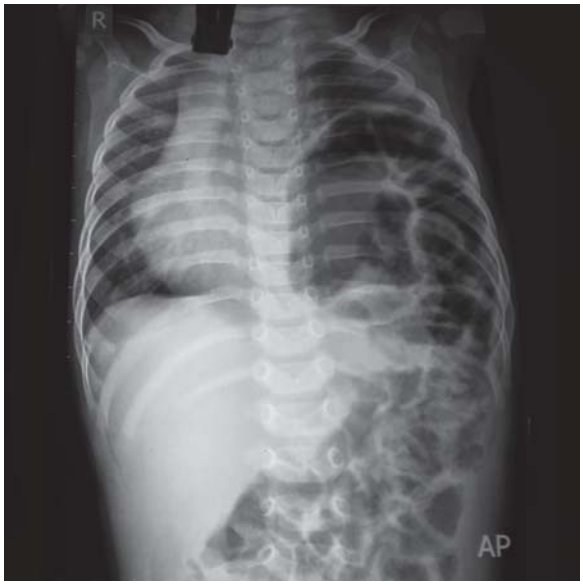


Fig. 18.1: Chest X-ray of a neonate with CDH

Renal and Cranial ultrasonography should be done to rule out associated anomalies.

Antenatal diagnosis can be made prenatally by fetal ultrasonography or ultrafast fetal magnetic resonance imaging (MRI). One of the hopes of prenatal diagnosis is to identify predictors of poor postnatal outcome which include: early gestation diagnosis, severe mediastinal shift, polyhydramnios, a small lung-to-thorax transverse area ratio, and the herniation of liver or stomach.[20]

Three-dimensional fetal ultrasonography and MRI can be used for direct volume estimation of the fetal lung and definitive diagnosis of pulmonary hypoplasia.[21-28]

Doppler ultrasonographic determination of pulmonary artery blood velocity waveforms is one tool used to diagnose pulmonary hypoplasia in the fetus. The pulsatility indices are high, and the peak systolic flow is significantly lower than normal. These findings are attributed to the higher impedance and a delay in pulmonary vessel development.

Pathophysiology

The pathophysiology of congenital diaphragmatic hernia involves:

- Pulmonary hypoplasia
- Pulmonary hypertension
- Pulmonary immaturity
- Potential deficiencies in the surfactant and antioxidant enzyme system
- Cardiac impairment.

Pulmonary Hypoplasia

In the fetus, during the second month, the pleuroperitoneal membrane begins to form, separating the pleural and peritoneal cavities. The last portion of this membrane to form is the posterolateral portion, the right side closing before the left side. The fetal gut is outside the pleuroperitoneal cavity in the yolk sac during the first month of fetal life and returns to the peritoneal cavity during the second month of development. If the gut returns prior to full closure of the pleuroperitoneal membrane, any or all portions of the gut may migrate up into the pleural cavity.[29]

Lung development is impaired by the presence of abdominal contents in the pleural cavity during fetal growth. The greater the amount of abdominal contents in the pleural cavity and the earlier the migration, the greater the degree of pulmonary hypoplasia that will be present at birth. Not only is the ipsilateral lung affected but there are developmental changes in the contralateral lung as well.[15]

Because of bowel herniation into the chest during crucial stages of lung development, airway divisions are limited to the 12th to 14th generation on the ipsilateral side and to the 16th to 18th generation on the contralateral side. Normal airway development results in 23 to 35 divisions. Because airspace development follows airway development, alveolarization is similarly reduced. Lung size is reduced, cell numbers are decreased, branches of airways are narrower and fewer, alveolar differentiation is reduced.

The surfactant system is demonstrably deficient in the lamb model of CDH. Postnatal administration of surfactant in these lambs is associated with dramatic increases in gas exchange, lung compliance, and pulmonary blood flow. However, in human neonates, reports on the status of the surfactant system are inconsistent.[30,31]

Infants with CDH also have impairment of the pulmonary antioxidant enzyme system and are more susceptible to hyperoxia-induced injury.

Severe reduction in lung mass and a relative deficiency of surfactant causes decrease in lung compliance, ultimately leading to hypercarbia and hypoxemia. Supranormal inflating pressures and respiratory rates to provide adequate tidal volume and minute ventilation often results in significant barotraumas and contralateral pneumothorax is not uncommon.

Pulmonary Vascular Hypertension

Development of the pulmonary arterial system parallels development of the bronchial tree, and, therefore, fewer arterial branches are observed in CDH.[32] Abnormal medial muscular hypertrophy is observed as far distally as the acinar arterioles. Pulmonary vessels are more sensitive to stimuli of vasoconstriction. Pulmonary hypertension leads to right-to-left shunting at atrial and ductal levels. This is referred as persistent fetal circulation or PPHN.[18,33,34] Acidosis, hypothermia, and elevated airway pressures also may contribute to sustained elevations in pulmonary vascular resistance (PVR). In the face of the significantly elevated PVR, flow will be preferentially directed right to left across the ductus arteriosus, further contributing to systemic hypoxemia. Elevated RV afterload causes increase in right atrial pressure which causes a right-to-left shunt at atrial level. Simultaneous decrease in pulmonary venous return causes fall in left atrial pressure, further contributing to an atrial pressure gradient favoring right to left shunting of blood. This persistent fetal circulation leads to right-sided heart strain or failure and to the vicious cycle of progressive hypoxemia, hypercarbia, acidosis, and pulmonary hypertension observed in the neonatal period. Primary myocardial dysfunction occurs as a consequence of elevated afterload, persistent hypoxemia and acidosis.

These physiologic changes can be divided into two components:

1. Irreversible: Due to pulmonary hypoplasia and abnormal vasculature.
2. Reversible: Due to vasoconstriction of the abnormal muscularized arteries.

The greater the irreversible component, the poorer the prognosis.

Imbalance in the production, release, and/or circulating levels of vasoconstrictors (leukotrienes C_4 and D_4, thromboxane A_2, platelet-activating factor) and vasodilators seems to be central to the right-to-left shunting observed with PPHN associated with CDH.[35] Elevated levels of endothelin 1 have been described in infants with CDH.[36,37] These peptides are vasoconstrictors that are produced in response to inflammation, ischemia, and other stimuli. Multiple receptors have been localized both in the vascular smooth muscle and in the vascular endothelial cell. Ventilator-induced hyperinflation of the hypoplastic lungs causes epithelial and endothelial damage resulting in inflammatory cascade and release of these vasoactive agents.

Cardiac Impairment

Relative left ventricular (LV) hypoplasia with an attenuated muscle mass and cavity size have been described. Many studies have confirmed that a calculated left ventricular (LV) mass less than 2 g/kg on pre-ECMO echocardiography was predictive of subsequent death.[38] This is believed to arise from decreased *in utero* blood flow to the LV, the mechanical compression of the herniated viscus similar to that observed in the lungs, and/or a primary yet unidentified developmental defect that simultaneously causes the diaphragmatic hernia and lung problems. LV hypoplasia is perhaps the most important determinant of outcome following nitric oxide administration. The markedly diminished LV performance causes a right-ventricular-dependent systemic circulation.

The combined effect of these pathophysiologic alterations is the establishment of a vicious cycle of hypoxemia, hypercarbia, pulmonary hypertension, myocardial dysfunction, and right-to-left shunting of blood through persistent fetal channels. If uninterrupted, this sequence of events ultimately proves fatal. Optimum ventilation, myocardial support and metabolic correction may reduce PVR and thereby interrupt this cycle.

Medical Management of Congenital Diaphragmatic Hernia

Goal: The goal is to maintain adequate oxygenation and ventilation, and prevent iatrogenic barotrauma from mechanical ventilation.[1] Bag-mask ventilation has to be minimized to reduce the risk of gastric expansion. Baby should be endotracheally intubated. Immediately after intubation, the gut should be decompressed.

Monitoring: Vascular access should be obtained. The umbilical vein and artery may be utilized or a right radial arterial catheter (for preductal arterial blood gas analysis) and a central venous catheter may be placed. Shunting through the ductus arteriosus is suggested, if the preductal PaO_2 is 15 to 20 mm Hg higher than the postductal PaO_2. Shunting at the level of the foramen ovale will decrease the predicted value of the preductal PaO_2 and will not produce a gradient when compared with the postductal PaO_2. Preductal saturation also reflects cerebral oxygenation.

Conventional ventilation: Treatment is aimed at immediate stabilization with sedation, paralysis, and moderate hyperventilation.[39] The ventilatory strategy should achieve a preductal oxygen saturation of greater than 85 percent, while maintaining a $PaCO_2$ of 45 to 55 mm Hg and a pH greater than 7.3, with peak inspiratory pressures less than or equal to 25 cm H_2O.[40] Pneumothorax in the unaffected lung can occur and is often the cause of death during resuscitation. Hypotension and shock are often seen. They can be secondary to prolonged systemic hypoxemia, cardiac impairment caused by shifting of the mediastinal contents by the hernia, and gastrointestinal fluid losses.

High-frequency oscillatory ventilation (HFOV): Neonates who require peak pressures greater than 25 cm H_2O for adequate oxygenation may need to be ventilated with high-frequency oscillatory ventilation (HFOV) to minimize the risk of ventilator associated barotraumas. In fact, some neonatologists advocate not only preoperative stabilization but also endotracheal intubation and high-frequency oscillatory ventilation (HFOV) immediately at birth.

Permissive hypercarbia: It was first proposed by Wung[41] in 1985 for infants with persistent fetal circulation. In many centers this strategy has been adopted for neonates with CDH.[42,43] Baby is stabilized with a strategy of "gentle ventilation." Low peak inflating pressures and permissive hypercapnia, prevents barotrauma to the already hypoplastic lungs. After a period of stabilization, when pulmonary arterial pressure falls, elective surgery is done.

Circulatory support: Provided there is adequate right-sided heart function and systemic perfusion, hypercarbia and ductal shunting is tolerated by the neonates with CDH. Decreased cardiac output leads to reduction in pulmonary perfusion and further hypoxemia. Venous return with lower oxygen content potentiates the hypoxemia caused by right-to-left shunting. Normal lactate levels, mixed venous saturation greater than 70 percent, absence of metabolic acidosis and normal echocardiography may reflect adequate right heart function.

Continuous monitoring of oxygenation, blood pressure, and perfusion is needed. Adequate volume

and inotropic support with dopamine, dobutamine, or milrinone may be needed. To prevent elevation of PVR a minimal stimulation approach is suggested. Maintain glucose and ionized calcium concentrations within reference range.

In the perioperative period, some infants exhibit a "honeymoon period", which is then followed by a sudden, often unexplained return to a state of PPHN and clinical deterioration (acidosis, hypoxemia, hypercapnia, pulmonary hypertension, and right-to-left shunting through the foramen ovale and the ductus arteriosus). Measures to both prevent further increases in PVR and promote a decrease in PVR, thereby increasing pulmonary blood flow, should be instituted. These include increased oxygenation, hypocarbia, alkalosis, avoiding sympathetic stimulation, and normothermia. In the 1970s/early 1980s, such measures included hyperventilation (often maintaining the pH > 7.50 and the Pco_2 < 25 mm Hg), ligation of the PDA, and pharmacologic therapy (e.g. isoproterenol, tolazoline). However, resulting barotraumas, volutrauma and systemic hypotension can be harmful.

Vasodilator therapy: It has been advocated for perioperative control of increased pulmonary arterial pressures. Suggested intravenous agents include isoproterenol, nitroglycerin, tolazoline, adenosine, and adenosine triphosphate. These agents rarely are effective because the pulmonary vasodilatation produced is matched by an equal fall in systemic vascular resistance. They have been largely replaced by inhaled nitric oxide (NO).[44,45]

Alkalinization: It is occasionally used for rapid pulmonary vasodilation. Hyperventilation or alkali infusions are used for this. However, the use is controversial. Cerebral ischemia and neurodevelopmental deficits can occur. Walsh-Sukys et al have shown increased use of extracorporeal membrane oxygenation (ECMO) and oxygen at age 28 days with use of alkali infusions.[46]

During the 1980s, other pharmacologic therapies were introduced, including prostacyclin, prostaglandins, NO, and ECMO. More recently, therapeutic aims have been directed toward NO and ECMO.

Nitric oxide (NO): The FDA approved nitric oxide for the treatment of PPHN in December 1999. NO diffuses across the alveolar capillary membranes and stimulates cyclic guanylate cyclase, which increases cyclic GMP, thereby causing vascular smooth muscle to relax. When inhaled, NO decreases PVR and improves lung blood flow. Optimal dose is unknown, although most investigators agree that doses >20 ppm are not beneficial and may be harmful. Administration should occur under controlled conditions, with access to ECMO if needed. NO_2 and methemoglobin levels should be frequently monitored, and weaning should gradually occur. Abrupt discontinuation may be associated with severe rebound pulmonary hypertension. Although there may be a response to inhaled NO (iNO) in neonates with CDH, there are no clear data that this impacts on survival.[47] A recent meta-analysis on inhaled nitric oxide for PPHN (all causes) suggests that the outcome of infants with CDH is not improved, and recommends its use in term and near-term infants with hypoxic respiratory failure who do not have CDH.[48] The immediate short-term improvements in oxygenation, however, seen in some treated infants may be of benefit for transport, or until initiation of ECMO.[40]

Phosphodiesterase inhibitors: Phosphodiesterase inhibition using dipyridamole results in transient improvement in oxygenation with some side-effects on systemic blood pressure.[49-51] The relatively lung-selective phosphodiesterase-5 inhibitor sildenafil has been successfully used in the treatment of adult pulmonary hypertension. Case reports in infants with CDH are emerging, suggesting some acute improvement in oxygenation or decrease in PVR either with sildenafil alone, or in combination with iNO to potentiate its pathway.[52-55] There are no randomized controlled trials or prospective studies on the use of sildenafil in infants with CDH.

Prostaglandins: Decreased LV performance in patients with severe CDH is common, and is perhaps the most important determinant of outcome following NO administration. The markedly diminished LV performance causes a right-ventricular-dependent systemic circulation. In this setting, both prostaglandin E1 to maintain ductal patency (thus enhancing RV contribution to systemic blood flow), and milrinone for pharmacologic reduction of LV afterload may be beneficial.[45] Therefore, serial ultrasound to detect a restrictive ductus and initiate a trial of prostaglandin E1 to reopen the ductus and decrease RV pressure overload is increasingly used in some centres.[33] There is also evidence that prostaglandin E1 augments the endogenous production of cyclic AMP and thus, could potentiate the effect of cyclic GMP mediated vasodilatation. There are, however, no randomized controlled trials on the use of prostaglandin E1 in infants with CDH.

Neonates with right ventricular dysfunction and low systemic pressures may require intravenous fluids and inotropic support. Predictors of outcome during the initial resuscitation are inexact. The inability to achieve a preductal PaO_2 greater than 100 mm Hg predicted 100 percent mortality in one study.[56]

ECMO: The benefit of ECMO on the morbidity and mortality of CDH is controversial. Some centers reported significant improvement in survival with the introduction of ECMO.[57] However, others experienced the same survival rates without the use of ECMO.[58] There is significant morbidity associated with the use of ECMO. Anticoagulation with heparin to prevent clot formation in the ECMO circuit and platelet activation and consumption increase the risk of bleeding. Bleeding may cause significant morbidity if this occurs in the CNS, and bleeding may complicate attempts at surgical correction while on ECMO.

Inclusion criteria for ECMO include:[59]

- Gestational age greater 34 weeks
- Weight greater than 2 kg
- Presence of reversible disease
- Predicted mortality of greater than 80 percent. Neonates with an oxygenation index (OI = Fio_2 × mean airway pressure × 100/Pao_2) greater than 40 to 50 may represent those at greatest risk (> 80%) of mortality
- Progressive hypoxia, hypercarbia, and PPHN who have failed other attempts at medical correction, including iNO, inotropic support, or opening the ductus with prostaglandin E_1.[40]

Contraindication:

- Intraventricular hemorrhage more than grade II
- Another life-threatening congenital anomaly.[59]

A review of all of the published data has indicated that there may be a short-term benefit with the use of ECMO but there may not be a long-term benefit because of the associated morbidity.[60] The use of ECMO has been shown to improve survival in the infants at highest risk of mortality (80% or greater), but overall, infants with CDH placed on ECMO have a lower survival rate than those not placed on ECMO (52.9 versus 77.3%).[61] Infants placed on ECMO may, of course, be sicker and have more hypoplastic lung than infants not placed on ECMO; their increased mortality may be the result of their underlying lung disease.

Infants who would not survive according to Bohn criteria [$PaCO_2$ > 40 and ventilatory index (mean airway pressure × respiratory rate) >1000] or who develop a persistent fetal circulation pattern following a "honeymoon" period can be placed on ECMO in order to allow the infant's lungs time to develop and restructure. Neither there is consensus for the the timing of initiation, and withdrawal of ECMO therapy nor for the timing of surgery during ECMO.[62]

Surfactant: As rescue therapy it is administered within 24 hours of birth in neonates with CDH and a poor prognosis. As prophylactic therapy, surfactant (50-100 mg/kg of Infasurf R) is administered prior to the first breath in neonates with CDH who are given a poor prognosis antenatally. Rescue or prophylactic therapy is associated with an improvement in oxygenation in some neonates with CDH.[63-65] Prophylactic surfactant therapy and natural surfactants are thought to be more efficacious. Negative results are shown by Colby et al.[66]

Surgical Considerations

Timing of Surgery

CDH is no more considered a neonatal emergency. Preoperative stabilization and delayed surgery decreases morbidity, mortality and is preferred now. Medical problems of pulmonary hypoplasia and PPHN are largely responsible for the outcome of CDH and that the severity of these pathophysiologies is largely predetermined *in utero*. Herniated viscera in the chest does not appear to exacerbate the pathophysiology as long as bowel decompression with a nasogastric tube is continuous.[67] Bohn et al[68,69] advocate the avoidance of the "mad dash" to the OR and recommend instead a 24 to 48 hours period of stabilization. Furthermore, they contend that infants who do not respond to this therapy will fail to survive with surgery or any other therapy, including ECMO. Some suggest that repair 24 hours after stabilization is ideal, but delays of up to 7 to 10 days are typically well-tolerated, and many surgeons now, adopt this approach. Some surgeons prefer to operate on these neonates when normal pulmonary artery pressure is maintained for at least 24 to 48 hours based on echocardiography.

Several reports indicate that circulatory stability, respiratory mechanics, and gas exchange deteriorate after surgical repair.[70]

Typically supine position with transabdominal subcostal incision is used. Intrathoracic approach is usually through right posterior thoracotomy for right-sided defects. By using laparotomy, herniated viscera is removed from chest cavity and relocated to abdomen. Primary repair of diaphragm is done, if there is adequate mobilization, otherwise, prosthetic (Silastic or Gortex) material, rotational muscle flaps or fascial flaps are used. Visceral adhesions may need to be lyzed or the Ladd procedure is carried out to fix mobile viscera postreduction of the malrotation. Primary closure of abdomen is done, if tension is minimal; otherwise, Silastic pouch closure is used.[71]

After the abdominal contents are replaced, there may be a significant elevation in abdominal pressure with surgical wound closure resulting in cephalad displacement of the diaphragm, decreased functional residual capacity, and compression of the inferior vena

cava (IVC).[1,70] A pulse oximeter applied to the lower extremity at the time of anesthetic induction may forewarn of abdominal compartment syndrome and circulatory compromise.

After reduction, an attempt to inflate the hypoplastic lung is not recommended, as it is unlikely to expand and the contralateral lung may be damaged by excessive positive airway pressures.[17]

The patient with a right-sided defect and an intrathoracic liver presents unique problems to the surgeon. The neonatal liver is extremely friable, and kinking of the hepatic veins and the IVC can accompany the return of the liver to the abdomen. Careful manipulation of the liver into the abdomen must be accompanied by hemodynamic monitoring. Occasionally, a 2-cavity (right chest and abdomen) approach may be necessary to reduce the viscera.

Chest tube placement: Chest tube drainage is necessary when a tension pneumothorax is present; however, whether routine chest drainage has a role is controversial. Some clinicians report improved survival when chest drainage is not used. Others think that balanced intrathoracic drainage, in which a closed gated pressure system is used to maintain intrathoracic pressure within the normal physiologic range, may minimize-risk of pulmonary injury.

In some institutions, surgery for CDH may be performed in the neonatal intensive care unit to avoid the stresses of transport and sudden changes in ventilation parameters.

Increasingly a laparoscopic or thoracoscopic approach is being utilized.[72] General balanced anesthesia without regional analgesia is used. Lung separation is not done. Near right lateral decubitus position is used. For better surgical exposure, carbon dioxide is insufflated into the left chest, starting at pressures of 5 mm Hg and flows of 0.5 L/min. Immediate (and immediately reversible) decrease in tidal volumes, increase in CO_2, and eventually desaturation are reported with surgical manipulations. However, no hemodynamic instability was noted with insufflation pressures as high as 8 mm Hg.[73] In children with significant hypercarbia and/or pulmonary hypertension, insufflation with carbon dioxide may not be tolerated, precluding this approach.

Maintenance of platelet counts above 150,000/µl, activated clotting times (ACT) 160 to 180 seconds, and aminocaproic acid in the perioperative period decreases hemorrhagic complications and fibrinolysis respectively during ECMO. Otherwise surgery should be done just before or after decannulation from ECMO.

Fetal Surgery

Fetal repair in human was first described in 1990.[74] Overall success of the open fetal approach was limited by maternal morbidity, which included premature rupture of membranes and preterm labor. Fetal intervention was only considered for those fetuses at highest risk of mortality. Research during the late 1970s introduced the concept of tracheal occlusion to reverse the lung pathophysiology from diaphragmatic herniation. This concept resulted in a fetal strategy in humans to temporarily occlude the trachea *in utero* until birth (PLUG—Plug the Lung Until it Grows).[67,75-77] The fetal lung secretes fluid by active ion transport through gestation, and this lung fluid provides a template for lung growth. Occlusion of the fetal trachea traps this fluid and stimulates lung growth, either by retention of growth factors within the lung or stimulation of local growth factors by the gentle distension provided by the fluid. However, this strategy and variations of this strategy have not demonstrated any survival advantage over standard postnatal medical management.[67] Currently three randomized control trial of fetoscopic endoluminal tracheal occlusion are going on.

Anesthetic Considerations

Preoperative assessment includes evaluation of the degree of respiratory compromise, pulmonary hypertension, type of ventilatory support and blood gas values. One may need neonatal intensive care unit (NICU) ventilator or HFOV for intraoperative ventilation. Cardiovascular evaluation should focus on identifying congenital heart defects, degree of right-to-left shunting, pulmonary hypertension, and right ventricular performance. Severe pulmonary hypertension can result in severe hypoxia, decreased cardiac output, and metabolic acidosis. This can be inferred from echocardiography and preductal and postductal blood gas analysis. Premature neonates are at risk for the development of intraventricular hemorrhages. This will exclude them from ECMO because of the anticoagulation. Head ultrasounds can detect hydrocephalus. Hematologic issues require maintaining an adequate hemoglobin (approximately 12 gm%) and checking for vitamin K administration at birth. Some of these patients may be on diuretics. An electrolyte panel can rule out hypokalemia. The neonate will already have a nasogastric or orogastric tube in place. If not, this should be placed to decompress the stomach.[1]

Anesthesia concerns are:

- Hypoxemia due to lung compression, primary pulmonary hypoplasia, pulmonary hypertension, and high-pressure induced iatrogenic contralateral pneumothorax.
- Hypotension caused by overdistension of the stomach and herniation across the midline, pulmonary hypertension causing RV failure, contralateral pneumothorax, kinking of major blood vessels, particularly those of the liver and both primary and secondary myocardial dysfunction.
- Hypercarbia and respiratory acidosis. $PaCO_2$ reflects the severity of the lung pathology and therefore survival. An inability to reduce $PaCO_2$ is associated with a poor prognosis.[78]

Neonate should be transported from NICU to OR by anesthesia team. If infant is already intubated, confirm paralysis prior to transport to lessen the risk of patient movement and inadvertent extubation. Transport with full monitoring, airway equipments, and resuscitation drugs. Prior to any further anesthetic administration, re-establish all monitoring in OR.[72] For nonintubated patients, rapid-sequence induction using sodium thiopental or propofol should be carried out. Aspirate gastric contents with nasogastric tube before induction. Atropine (0.02 mg/kg IV) is given prior to induction to counteract bradycardia. Muscle relaxation is achieved using succinylcholine. If necessary to mask ventilate, avoid high-inflation pressures, as this may further dilate the bowel. Many now choose to administer rocuronium or mivacurium in doses designed to provide laryngeal relaxation within 60 to 90 seconds; however, significant oxygen desaturation may occur in these stressed neonates. Avoid N_2O and maintain PIPs as low as possible. For patients with late herniations and healthy lungs, consider caudal epidural catheter advanced to thoracic space instead of IV opioids, and early extubation (OR or NICU); If surgery is performed on ECMO, give high-dose iv opioids (may be given in ECMO circuit) and muscle relaxant.

In these infants, venous access in the lower extremities should be avoided because the IVC may be compressed after reduction of the hernia, limiting venous return.[2] Some suggest internal jugular cannula to provide more reliable access. This access allows CVP monitoring both intraoperatively and postoperatively and may be essential to deliver total parenteral nutrition (TPN) postoperatively. However, in the author's institute, this is not the practice.

In addition to the standard monitors, many recommend a preductal or umbilical artery catheter.[71] Author uses and recommends both preductal and postductal pulse oximeters. A precordial stethoscope on the contralateral chest can be used to identify a pneumothorax.

Hypothermia must be avoided. Complications associated with hypothermia include increased PVR with resultant right-to-left shunting. Hypothermia also causes increased oxygen consumption, which may result in inadequate oxygen delivery and acidosis, which then further increases PVR and worsens arterial desaturation.[2] Intraoperative management consists of first ensuring adequate room temperature and utilizing either warming lights or a forced warm air blanket to maintain normothermia.

Anesthetic Maintenance

Size of the defect and the anticipated postoperative respiratory status determines the anesthetic technique. For infants with a small defect without respiratory distress, regional analgesia is provided and narcotics are avoided in anticipation of extubation.[10]

In those infants who will remain intubated after surgery, inhalational agents and narcotics may be used as tolerated. Nitrous oxide is avoided in infants with CDH;[2] (1) most require high, inspired oxygen concentrations, (2) N_2O can diffuse inside the viscera and exaggerate lung compression, (3) N_2O can cause pulmonary vasoconstriction. Low concentrations of inhalation anesthetics (sevoflurane or isoflurane) can be administered and increased, if the patient is hemodynamically stable. However, they are myocardial depressants and are avoided until the chest is decompressed.[78] An opioid-based anaesthetic has been described and may minimize the surgical stress and pulmonary vascular lability.[79] In most cases, high-dose narcotics (usually, fentanyl) are administered and the narcotic infusion is continued into the postoperative period.

Muscle relaxation is typically employed to facilitate surgical exposure and abdominal closure. With rare exceptions, infants with a moderate or large CHD require ventilatory support preoperatively and most receive neuromuscular blockade. The goals of ventilation in the OR are the same as preoperatively, i.e. to optimize pH and pulmonary blood flow with minimal barotrauma. Peak pressures should not exceed 25 to 30 cm H_2O. Hyperventilation is reserved to initially treat an acute episode of pulmonary hypertension. If a sudden deterioration occurs in ventilation, hemodynamic status, or both, pulmonary hypertension must be quickly differentiated from a contralateral pneumothorax, because the treatment for a pneumo-

thorax is needle thoracostomy and chest tube placement rather than hyperventilation.

Pain Management

- Continuous fentanyl infusion
- Epidural placed transcaudally or through lumbar or thoracic sites. This option for intraoperative and postoperative management may best be suited in those with smaller defects who likely will not require prolonged ventilation or anticoagulation for ECMO[80]
- Intrapleural analgesia
- Infiltration of local anesthetic at incision
- Paracetamol suppository.

Intraoperative Complications

Contralateral pneumothorax is a potential intraoperative complication and needs to be considered, if there is an acute clinical deterioration. A sudden fall in lung compliance, blood pressure, or oxygenation may signal a contralateral (usually right-sided) pneumothorax and necessitate placement of a chest tube.[18]

Pulmonary hypertension can be managed by maintaining a normal pH, PaO_2, and $PaCO_2$ and minimizing hypothermia and surgical stress.[39] Sodium bicarbonate may need to be administered to treat acidosis and/or to alkalinize the blood and thereby treat pulmonary hypertension. If iNO is used preoperatively, it should be continued in the operating room.

Finally, the anesthesiologist should anticipate the possibility of a cardiac arrest during this operation. Vasopressors, including dopamine (4 to 10 µg/kg/min) and epinephrine (0.1 to 1.0 µg/kg/min), should always be available for emergency intraoperative administration.[18]

Postoperative Care

Most infants with CDH require NICU care.[10] Recovery depends on the degree of pulmonary hypertension and pulmonary hypoplasia. There is no effective treatment for pulmonary hypoplasia, other than keeping these neonates alive with the hope that lung maturation will occur.[17] It was previously believed that pulmonary hypoplasia was responsible for most deaths; however, it is now believed that potentially reversible pulmonary hypertension may be responsible for as much as 25 percent of reported deaths.[81]

The postoperative course, after surgical reduction of CDH, is often characterized by rapid improvement, followed by sudden deterioration with profound arterial hypoxemia, hypercapnia, and acidosis, resulting in death.[17] The mechanism for this deterioration is the reappearance of fetal circulation patterns, with right-to-left shunting through the foramen ovale and ductus arteriosus. Proper sedation is necessary as any stressful stimulus can further exacerbate already elevated pulmonary pressures with resultant increases in shunt flow and further desaturation. ECMO may be required in the perioperative period because of pulmonary vascular reactivity and respiratory distress refractory to conventional or even high-frequency or oscillatory ventilation.[71]

Monitoring for hypoglycemia, metabolic and hematologic status is essential.

Pain management can be with continuous fentanyl infusion or epidural analgesia.

ESOPHAGEAL ATRESIA AND TRACHEOESOPHAGEAL FISTULA

Esophageal atresia (EA) is the most frequent congenital anomaly of the esophagus, with an approximate incidence of 1 in 4000 neonates. Greater than 90 percent of affected individuals have an associated tracheoesophageal fistula (TEF).[17]

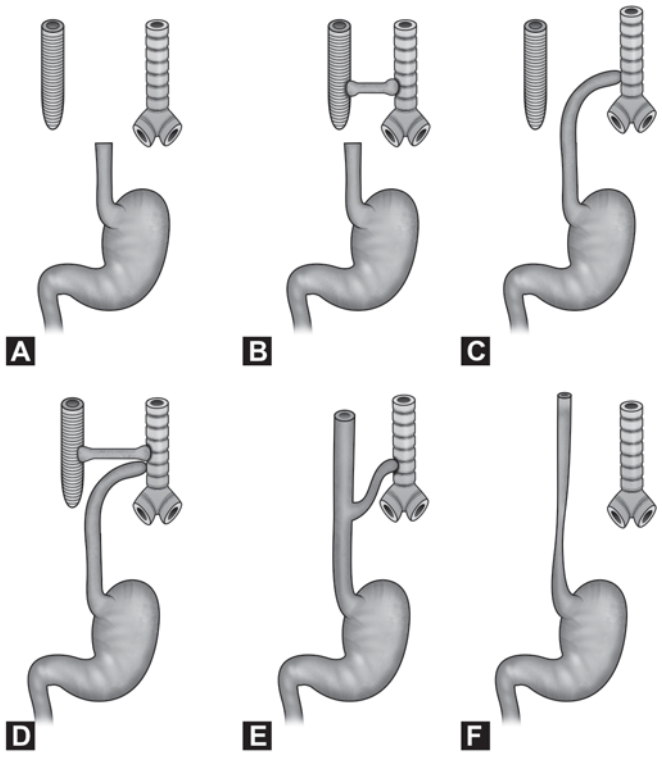
Embryology and Anatomy

Both the esophagus and the trachea originate from the median ventral diverticulum of the primitive foregut. The TEF lesion results from failure of the two structures to separate during division of the endoderm. Esophageal atresia results when the tracheal structures assume most of the endoderm, and TEF results when the esophageal and tracheal ridges fail to develop, leaving a communication between the two structures.[2]

Gross created a classification system in 1953[82] (Figs 18.2A to F).

Classification and Incidences[83-94]

- Type A: Esophageal atresia without fistula or so-called pure esophageal atresia (8%)
- Type B: Esophageal atresia with proximal TEF (1%)
- Type C: Esophageal atresia with distal TEF (84%)
- Type D: Esophageal atresia with proximal and distal TEFs (3%)
- Type E: TEF without esophageal atresia or so-called H-type fistula (4%)
- Type F: Congenital esophageal stenosis (<1%)



Figs 18.2A to F: The gross classification of congenital anomalies of the trachea and esophagus. A: EA without fistula. B: EA with proximal fistula. C: EA with distal fistula. D: EA with proximal and distal fistula. E: TEF with no EA. F: Esophageal stenosis. EA, esophageal atresia; TEF, tracheoesophageal fistula.

Associated Anomalies

The VATER syndrome, described in 1973, is an association of the following anomalies: V, vertebral defects; A, anal defects; T, TEF; E, esophageal atresia; and R, radial or renal anomalies.[95] Another acronym includes a "C" and "L" because cardiac and limb anomalies are also common. VACTERL: V Vertebral (vertebral malformations, hemivertebrae); A Anal (imperforate anus, also midgut malrotation, Meckel's diverticulum); C Cardiac (VSD, PDA, TEF, ASD, coarctation of aorta); T Trachea (TEF); E Esophagus (EA); R Renal (renal agenesis, hydronephrosis, renal lobulation); L Limb (radial aplasia, polydactyly, wrist anomalies). As many as 20 to 25 percent of infants with esophageal atresia have at least three of the lesions included in VACTERL.[96] Between 50 and 65 percent of infants with esophageal atresia with or without a TEF have at least one additional anomaly. Anomalies are more common in the isolated esophageal atresia type and least common in the H-type fistula.

CHARGE syndrome is associated in 6 percent of patients.[97] It includes: coloboma of the eye, heart disease, choanal atresia, retarded growth, ear abnormalities and deafness. Potentially fatal anomalies, e.g. tracheal agenesis or stenosis, laryngeal atresia, are uncommon but associated with TEF plus EA but not H type fistulas.

Incidence of Associated Anomalies[2]: Cardiovascular—35 percent; Musculoskeletal—30 percent; Gastrointestinal—20 percent; Genitourinary—10 percent; Craniofacial—4 percent.

The following anomalies also occur with increased frequency in esophageal atresia:

Neurologic defects: Neural tube defects, hydrocephalus, tethered cord, holoprosencephaly.

GI defects: Duodenal atresia, ileal atresia, hypertrophic pyloric stenosis, omphalocele, malrotation, Meckel's diverticulum.

Pulmonary defects: Unilateral pulmonary agenesis, diaphragmatic hernia.

Genitalia defects: Undescended testicles, ambiguous genitalia, hypospadias.

Also, trisomies 13, 21, or 18 and Fanconi syndrome may be present. The overall incidence of associated anomalies is approximately 50 percent.

Another 20 percent to 30 percent of infants with tracheoesophageal fistula (TEF) are premature, weighing less than 2000 g.

Anesthetic Implications

- Look for other associated anomalies
- Get cardiology opinion and echocardiogram preoperatively in suspected cases
- Choanal atresia may cause respiratory distress
- Retrognathia is common with CHARGE syndrome, so be prepared for difficult intubation
- A tethered cord is usually detectable with ultrasonography in the newborn period. If present, one cannot plan for epidural anesthesia
- Mortality associated with TEF generally depends on the severity of the underlying lung disease and associated anomalies. Improvements in anesthetic and surgical techniques allow greater than 90 percent survival in otherwise healthy full-term infants.[98] In high-risk infants (<1800 g or with pneumonia) with TEF, mortality ranges from 15 percent to 60 percent.

Clinical Presentation

Classically, the neonate with EA presents with copious, fine, white, frothy bubbles of mucus in the mouth and, sometimes, the nose. These secretions may clear with aggressive suctioning but eventually return. The infant may have rattling respirations and episodes of coughing, choking and cyanosis. These episodes may be exaggerated during feeding. Aspiration of saliva or milk, if the baby is allowed to suckle, can lead to an aspiration pneumonitis. In a baby with esophageal atresia and a distal TEF, the lungs may be exposed to gastric secretions. Infants with an isolated TEF in the absence of EA may elude diagnosis until later in life when the patients come to medical attention for recurrent pneumonias and refractory bronchospasm.[17] Air from the trachea can pass down the distal fistula when the baby cries, strains, or receives ventilation. As a result, the stomach and small intestine can become dilated, which elevates the diaphragm and makes respiration more difficult. Occasionally, it can lead to an acute gastric perforation, which is often lethal. The abdomen will be scaphoid, if no fistula exists.[99,100] In the delivery room, the affected infant may have the sonorous seal-bark cough that indicates concomitant tracheomalacia.

Diagnosis

Prenatally, EA should be suspected, if maternal polyhydramnios is present. However, EA is usually diagnosed soon after birth when an oral catheter cannot be passed beyond 10 to 12 cm into the stomach or when the neonate exhibits cyanosis, coughing, and choking during oral feedings.

Plain radiographs of the chest and abdomen will reveal coiling of a nasogastric tube in the esophageal pouch and possibly an air-filled stomach in the presence of a coexisting TEF (Figs 18.3 and 18.4). In contrast, pure EA may present as an airless, abdomen. The chest radiograph provides information about the cardiac

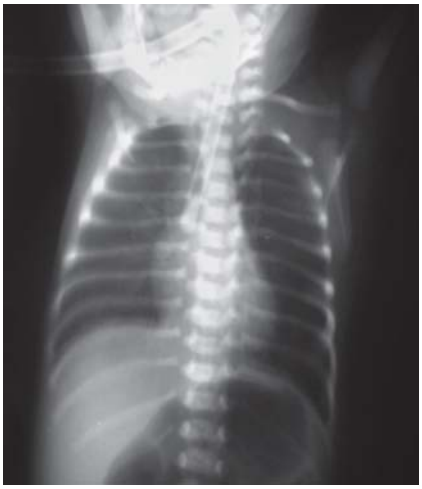


Fig. 18.3: X-ray chest and abdomen PA view of a baby with EA and TEF

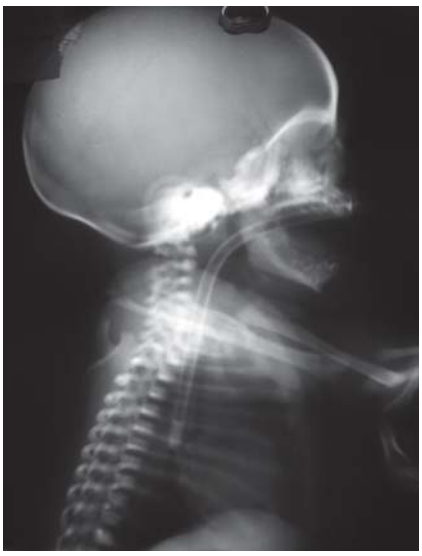


Fig. 18.4: X-ray chest and abdomen lateral view of a baby with EA and TEF

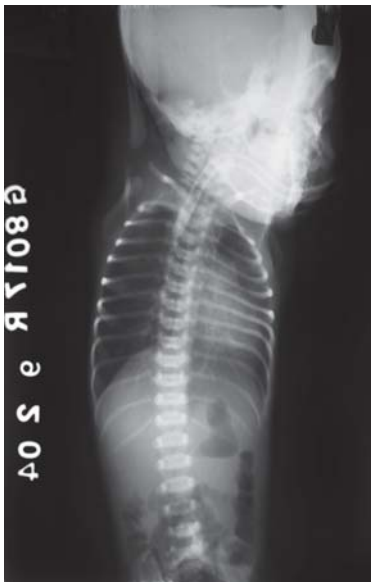


Fig. 18.5: X-ray chest and abdomen PA view of a baby with EA and TEF with 13 ribs

silhouette, the location of the aortic arch and the presence of vertebral and rib anomalies, as well as the presence of pulmonary infiltrates (Fig. 18.5).[101-104]

Computed Tomography (CT) Findings

CT is not typically used in the evaluation of EA and TEF; however, CT does allow 3-dimensional (3D) visualization of the entire length of the esophagus, complete with atresias, fistulas, and gap length. Virtual endoscopy can be used to traverse stenoses, unlike traditional endoscopy.[105-114]

Ultrasonography

The ultrasonographic finding of an absent or small fetal stomach bubble in combination with maternal polyhydramnios is suggestive of EA and/or TEF. The presence of a dilated blind-ending esophageal pouch on a sonogram is suggestive of EA. This pouch sign has been confirmed with direct visualization after 26 weeks' gestation, but its onset has been suggested as early as the 22nd week.[115-117]

Preoperative Management

It certainly depends upon: Type of TEF/EA, duration between birth and presentation to clinician. 90 percent of cases come to the anesthesiologist after first feed with aspiration pneumonitis and respiratory distress. For management of anesthesia the infant's feedings are withheld, a catheter is placed in the esophagus to drain saliva, and the infant is placed prone in a head-up position.[78] If the infant has pneumonia, treatment should be initiated; surgery may be postponed until the pneumonitis improves or clears. The infant may be a candidate for gastrostomy to provide a means of nutrition during recovery from pneumonitis.

Rule out associated congenital anomalies, especially cardiac defects.[3] An echocardiogram should be obtained along with a chest X-ray and renal ultrasonography. Intravenous fluid containing an adequate glucose concentration (i.e. 10 percent glucose) is administered at a rate appropriate for the neonate's gestational age and weight. Prophylactic broad-spectrum antibiotics (e.g. ampicillin, gentamicin) are intravenously administered. The neonate is kept warm by using an incubator or overhead warmer. Oxygen therapy is used as needed to maintain normal oxygen saturation. In infants with respiratory failure, endotracheal intubation should be performed.

Surgical Management

Healthy infants without pulmonary complications or other major anomalies usually can undergo primary repair in the first few days of life. Survival rates in this group of patients approach 100 percent.[83-91,118,119]

Surgery usually involves a right thoracotomy in the lateral position, identification and ligation of a tracheoesophageal fistula, and anastomosis of the two ends of the esophagus.[3]

Debate continues as to whether the best approach is retropleural or transpleural. The former is slower, but it may diminish the chances of empyema when the esophageal anastomosis leaks transiently.[72]

Dividing the azygous vein is necessary to find the subjacent fistula, branching off the posterior aspect of the trachea (Type C). The right bronchus, aorta, and (rarely) left bronchus may be mistaken for this structure.[72]

Division of the fistula may dramatically improve ventilation; until this moment it is sometimes necessary to operate in short 3 to 5 min bursts, relaxing lung and mediastinal retraction for 1 to 2 min when saturations descend to critical levels.[72]

The anesthetist may be asked to help identify the distal end of the proximal pouch within the thorax by manipulating the Replogle tube from above. The distance from the mouth to the distal tip is noted. Only catheters of this length should be used for suction in the postoperative period.[3]

Once the two ends of the esophagus are approximated the anesthetist usually passes a fine transana-

stomotic nasogastric tube before the anastomosis is fashioned to allow enteral feeding in the postoperative period. This tube must be fixed in place, because it has a tendency to become dislodged.

Typical surgical time: 2 to 4 hours for primary repair of TEF/EA.

A thoracoscopic technique using a transpleural approach is possible.

A fistula may not be present. In some cases, the esophagus cannot be made continuous and a gastrostomy is performed.

A bronchoscopy may be performed prior to the thoracotomy to help identify the position of the tracheo-esophageal fistula.

A staged procedure, initial gastrostomy with deferred thoracotomy may be used in babies <1 kg, those with pure EA, or with more critical associated anomalies. The survival rate in this group is lower but in the range of 80 to 95 percent. Cardiac anomalies typically are the cause of death in this more complicated cases.[72]

Anesthetic Management

Main anesthesia goals are to maintain adequate oxygenation, ventilation and avoidance of gastric distension. Avoid excessive positive pressure ventilation (PPV) especially before the placement of the Fogarty balloon catheter or the ligation of the fistula.[120]

The degree of prematurity, episodes of aspiration, and associated congenital heart disease (CHD) are most relevant to anesthetic management.[3] Limb anomalies may make venous access and arterial cannulation difficult. If there is no tracheoesophageal fistula present then gastric distension during induction of anesthesia will not occur.

Premature babies or those who have aspirated may already be intubated. In this case, the positioning and length of the ETT are checked. Ideally, the distal end will be distal to the tracheoesophageal fistula so that distension of the stomach by ventilation through the fistula is avoided.

Patient should have intravenous access before induction and receive 20 µg/kg atropine (minimum 0.15 mg). The Replogle tube (tube in the upper esophageal pouch) is aspirated.[121]

Leakage of gas through the fistula may cause preferential ventilation of the stomach during PPV, which will decrease the functional residual capacity (FRC), impair ventilation and oxygenation by diaphragmatic splinting, and increase the chance of aspiration. Clinically, however, this is seldom a problem and most anesthetists use a gaseous or intravenous induction, facilitating tracheal intubation with a non-depolarizing relaxant. Irrespective of mode of induction, ventilation by face mask should be at inflation pressures less than 10 to 15 cm H_2O. In babies with associated significant airway abnormalities, some prefer to insert the tracheal tube while the baby breathes spontaneously, anesthetized with a volatile agent. 'Awake intubation' is seldom used because it is difficult, distressing for the baby and associated with significant oxygen desaturation and oropharyngeal trauma. Increases in intracranial pressure must be considered in a vigorous neonate. It can contribute to the occurrence of intraventricular hemorrhage in premature infants.[122] Cote[78] generally perform an "awake sedated" intubation. He usually administer 0.5 to 1 µg/kg of fentanyl and 25 to 50 µg/kg of midazolam and topicalize the tongue, larynx, and vocal cords with no more than 5 mg/kg of lidocaine (1.0%). The ETT is intentionally passed into the right main bronchus and then slowly withdrawn until breath sounds are heard on the left.

Occasionally massive gastric distension can occur, resulting in respiratory compromise and cardiovascular collapse, requiring an emergency gastrostomy.[1] Risk of gastric distension is proportional to the size of the fistula. Though, insertion of a gastrostomy may be lifesaving, it has its own problem. Airflow resistance through the fistula–stomach–gastrostomy may be so low that ventilation of the lungs becomes impossible. The gastrostomy may need to be intermittently clamped and unclamped or left partially clamped.[18]

It is critical, and sometimes difficult, to establish an airway in patients with a TEF. Surgeons should be readily available during the induction should emergent decompression of the stomach be required.[123]

The presence of a gastrostomy reduces the potential for reflux of gastric juice during the surgical procedure. If a gastrostomy is present, the gastrostomy tube should be open to air and left at the head of the table under the anesthesiologist's observation to avoid kinking and obstruction. Presence of a gastrostomy may slow mask inductions, requiring transient partial clamping of the tube.[1]

Positioning the tracheal tube: Most fistulas lie posteriorly in the mid or low trachea. Salem and others[124] suggest distal positioning of the ETT, with the bevel facing anteriorly and the posterior wall of the ETT occluding the fistula, but this manoeuvre is challenging to achieve and maintain. In practice, we insert the tube 'too deep', slowly withdrawing it until both lungs are ventilated. During surgery, inadvertent bronchial

intubation is a possibility because of the low lying tip. Bilateral air entry should be confirmed after intubation and whenever, the baby is repositioned. It is important to check proper ETT placement. Accidental right main stem bronchus placement of the tracheal tube results in a precipitous decrease in arterial oxygenation, especially during surgical retraction of the lung.

Once positioned, the ETT should be carefully secured. After the patient is positioned in the left lateral decubitus position, reconfirmation of the position of the ETT may be necessary.

Proper positioning of ETT between fistula and carina is impossible, if the fistula connects to the carina or a mainstem bronchus. In these situations, intermittent venting of a gastrostomy tube that has been placed preoperatively may permit positive-pressure ventilation without excessive gastric distension.[39] Even with adequate positioning of the ETT, in some patients ventilation through the fistula still occurs. In patients without a gastrostomy, gastric distension may impair ventilation.

Filston et al[125] have suggested occluding the fistula with a Fogarty catheter placed through a bronchoscope. Once the infant is adequately anesthetized, the surgeon is able to perform rigid bronchoscopy with a ventilating bronchoscope, following removal of the ETT. At this point, the exact location and size of the fistula can be determined and it can be occluded using a Fogarty balloon catheter. The ETT can then be replaced under direct visualization.[120] However, bronchoscopy, itself is quite challenging in newborn, leave aside proper placement of a Fogarty catheter.

The use of a cuffed ETT to minimize the risk of gastric distension and aspiration has been described.[126] Proper placement of the ETT can be confirmed with fiberoptic bronchoscope.[97] After intubation, the fiberoptic bronchoscope is passed through the ETT and the carina is visualized. Upon withdrawal of the bronchoscope, if the fistula is not visualized then the ETT is appropriately positioned. If the fistula is visualized, the ETT is advanced making sure that its tip remains above the carina. Keep air leak around ETT to a minimum (leak at 18–35 cm H_2O) to minimize alterations in ventilation secondary to changes in chest and pulmonary compliance.[72]

In patients with a gastrostomy, proper positioning of the ETT can be monitored by submerging the gastrostomy tube in a container of water so that gas bubbles are evident during ventilation of the fistula. If gas bubbling occurs, the ETT must be repositioned. The gastrostomy tube may be left to water seal during the surgery, which allows for continued monitoring for ventilation through the fistula.[2]

Alternatively, the gastrostomy tube can be connected to a capnograph. When the ETT is proximal to the fistula, carbon dioxide is detected. When the ETT is distal to the fistula, no expiratory gases are detected.

In patients with a gastrostomy, gastric decompression may serve as a low resistance vent through which most of the tidal volume escapes. If this occurs, the gastrostomy tube should be clamped or, as Karl[127] has reported, a retrograde Fogarty catheter can be inserted. Although this sounds good, precise positioning is nearly impractical in neonates. If at all done, maintenance of this exact position for two hours is almost impossible. Displacement of the balloon can cause complete occlusion of the trachea, or high pressure on small pulmonary vessels and/or airways with pulmonary blood flow or ventilation compromise.

Inadvertent entry of ETT into the fistula, kinking of bronchus during surgical manipulation, and lung retraction can cause drop in oxygen saturation and end-tidal carbon dioxide ($ETCO_2$). The surgeon must stop the procedure while the situation is clarified. The surgeon will be able to palpate the tip of the tube in the fistula if this is the problem.

Because of these myriad problems with PPV, many anesthesiologists recommend an anesthetic technique that uses spontaneous ventilation with sevoflurane. Alternatively, others believe that paralysis may be a safe and effective alternative, as long as the fistula can be effectively isolated by careful positioning of the ETT. In our experience, sufficient anesthetization with sevoflurane of a spontaneously breathing newborn without compromising blood pressure and oxygenation is rarely possible. This is particularly true in the repair of an esophageal atresia with tracheoesophageal fistula because this surgery is performed in the lateral position.

A new option, of the general inhalational plus caudal (or thoracic) epidural anesthetic have been described.[128,129] Caudally placed catheter can be advanced upto the thorax. Either 0.5 to 1 ml/kg of 0.25 percent bupivacaine with epinephrine (5 µg/ml) or 0.5 ml/kg of 3 percent chloroprocaine (15 mg/kg) with epinephrine is administered, and the inspired sevoflurane concentration is significantly reduced.[130] With this combination, spontaneous breathing can be maintained without hemodynamic compromise. In addition, postoperative analgesia can be supplemented.

Intraoperative Maintenance

Nitrous oxide may distend the stomach, compromising ventilation, and is best avoided. Avoid high FiO_2, if possible in premature neonates at risk for retinopathy of prematurity (ROP). Use air/O_2 mixture for ventilation

to maintain O_2 saturation between 95 to 100 percent. At times they require ventilation with 100 percent oxygen, despite the risk of the ROP.[17] Healthy infants may tolerate spontaneous ventilation, but most often neuromuscular blockade is necessary especially once the chest is opened and the lungs are retracted.[120] It can be difficult to obtain adequate oxygenation, ventilation and surgical conditions in a spontaneously breathing patient during open thoracotomy.[131]

Use low PIPs to avoid gastric distension by gases passing through fistula. Careful adjustment of ventilation will be necessary during surgical retraction of lung or during insufflation, if procedure is done thoracoscopically. Manual ventilation is recommended as surgical traction can easily occlude the neonate's soft trachea. It can be helpful in assessing pulmonary compliance.[132] IPPV is usually done by hand using a Jackson Rees circuit at a high-frequency of 30 to 40 per minute and low tidal volumes.[3]

Air/O_2/opioid (e.g. fentanyl 1 to 2 µg/kg/h), propofol or low-dose volatile technique is preferred because of better hemodynamic stability. Muscle relaxation (atracurium) is usually necessary. If epidural is used, GA drug requirements will be reduced. Once satisfactory ventilation is ensured, the chest is opened and the lungs are retracted.

Lung retraction impairs ventilation, especially in infants with respiratory dysfunction from immature lungs, pneumonia, or congenital heart disease.[2] Intermittent release of pressure by the surgeon to allow inflation of the right lung improving oxygenation and ventilation. Close communication between the surgeon and anesthesiologist is mandatory.[17] Blood clots or secretions may block the ETT, and frequent ET suctioning may be required. Rarely, the ETT may become completely occluded by a clot that cannot be removed by suctioning, necessitating immediate replacement of the tube.[17] This author has seen two such cases. Because the trachea is a soft structure in the newborn, surgical manipulation may kink the airway and further obstruct ventilation.[2] Thus, interference with adequate oxygenation can occur as a result of the patient's anatomy, operative positioning, and surgical manipulations. Inspired concentration of oxygen must be closely monitored and adjusted, balancing the risks of oxygen toxicity with those of hypoxia. Loss of breath sounds and the end-tidal carbon dioxide ($ETCO_2$) tracing commonly occurs during surgery secondary to airway obstruction.[97] It may be due to the accumulation of secretions or blood in the ETT. More often, however, it results from kinking of the trachea during surgical manipulation. The surgeon should immediately be instructed to release the surgical traction. Cardiac output can fall dramatically, if surgical maneuvres compress major vessels. Good communication and cooperation with the surgeon are essential. ETT placement may interfere with TEF closure. I have seen inadvertent inclusion of ETT in sutures while repair of tracheal tear. This was detected at the time of extubation as the ETT could not be pulled out. Migration of ETT above fistula may lead to leak through gastrostomy and difficult ventilation.

Fluids: Two intravenous cannulas are advisable, though significant bleeding is uncommon. Third-space losses can be replaced with (6–8 ml/kg/h) LR with 5 percent dextrose.[72]

Intraoperative monitoring must be carefully planned. A precordial stethoscope should be placed in the dependent (left) axilla, since obstruction of the mainstem bronchus during surgical retraction is not uncommon. It also helps in detection of endobronchial intubation.[2] Surgical retraction can also compress the great vessels, trachea, heart, and vagus nerve.[39] In infants with an unstable cardiorespiratory status or CHD, an arterial catheter (umbilical or right radial) should be placed.[2] If an arterial catheter is not available, a noninvasive device is used. Other monitoring consists of an electrocardiogram, pulse oximetry, and end-tidal gas monitoring. A change in the distance of insertion of the endotracheal tube of as little as 1 to 2 mm may determine whether the anesthesiologist is ventilating both lungs, one lung, or the fistula. Pulse oximeter may give early warning of some problem. A preductal and postductal location (two pulse oximeters) will diagnose intracardiac shunting.[78] The patient's temperature must also be monitored, and efforts must be made to prevent hypothermia. Blood gas monitoring is recommended to check pH, PO_2, PCO_2, hematocrit, glucose, electrolytes, and possibly coagulation.

Pain management: Intravenous opioids are effective for intraoperative and postoperative pain management. Generous doses can be used, if there is definite indication for postoperative ventilatory support. Infusion (fentanyl 2 to 4 µg/kg/hour; remifentanil 0.25-0.5 µg/kg/min) can be started.[1] The author uses fentanyl in titrated doses for such cases. Babies who are expected to be extubated, regional anesthesia is advantageous to avoid opioids and the risk of postoperative respiratory depression. Providing there are no significant vertebral anomalies, a caudal catheter may be advanced to T6 to T7 to supplement the general anesthetic (iso-, des-, or sevoflurane/air/oxygen) and provide excellent postoperative analgesia without the use of opioids and to facilitate extubation.[121] The catheter's position can be confirmed by injecting low ionic strength

contrast medium (0.5 ml Omnipaque 180).[133] Either intermittent bupivacaine (1 to 2 ml of 0.125% with epinephrine 1:200,000) can be administered every 6 to 8 hours or a continuous infusion of chloroprocaine (1.5%) with fentanyl (0.4 µg/ml) can be infused at 0.3 to 0.8 ml/kg/hr.[78] This is only possible with good ICU support. Surgeon can block intercostal nerves before wound closure. I use multimodality approach consisting of IV fentanyl 0.5 -1 µg/kg, rectal paracetamol 30 mg/kg and caudal 0.125 percent, 1.25 ml/kg bupivacaine after anesthetic induction.

Emergence and extubation: There is a constant debate whether to extubate the patient or to continue intubation and ventilation.[121] Extubation has advantage that it minimizes manipulation of the anastomosis from the ETT.[17] However, approximately 30 percent will require reintubation for clearing of secretions. With laryngoscopy and reintubation there are chances of trauma to the fistula site and traction on the oesophageal repair. Other advantage of ventilation is that adequate amounts of analgesia can be given. I take following approach: extubate babies which are good weight, full term without CHD, and without intraoperative cardiopulmonary complications, and are normothermic; I continue intubation and ventilation for babies < 2 kg, premature, hypothermic, with CHD or intraoperative complications or, if postoperative adequacy of ventilation is doubtful. They are weaned from ventilatory support when adequate gas exchange and respiratory effort are demonstrated.

Postoperative Considerations

Nasopharyngeal and oropharyngeal suctioning catheters should be carefully marked to avoid insertion down to the level of the anastomosis. Oral suctioning is performed every half hour for the first day, then every hour or more frequently as necessary on the second day. Thereafter, it is performed as needed. Suctioning is required to handle the copious oral secretions that can build up in the first day or so after surgery. As the swelling of the esophagus settles, the secretions taper. Head extension can put tension on the anastomosis and should be minimized. The chest draining tube is placed in 2 cm of water only to seal it; it is not connected to a suction device, which could encourage an anastomotic leak. Antibiotics are continued until the chest drain is removed, and the ETT is suctioned as necessary.

Postoperative complications include anastomotic leak, tracheomalacia or bronchomalacia, stricture, pneumonia, and pneumothorax.[134] Complications can also occur secondary to underlying medical conditions and result in significant morbidity and mortality.

A chest film is required.[3] The child is fed by nasogastric tube or parenterally for 5 to 7 days until a contrast study confirms anastomotic integrity. If the esophagus is patent and reasonably sized, the baby may be orally fed; starting with expressed breast milk is ideal. Then, the chest tube is removed. As soon as the baby is feeding well, the intravenous line is discontinued, and the baby can be discharged.

An anastomotic leak tends to occur 3 to 4 days after surgery. This leak has been reported in approximately 15 percent of cases. Pain and distress are often evident. Signs of sepsis may be present. The chest tube drains saliva. Treatment is supportive; appropriate antibiotics should be used, and the child should be given nothing by mouth. Surgery is not indicated, even with huge leaks. If the leak persists, esophagography may be performed with water-soluble contrast material to assess its magnitude. The usual protocol is to wait and let the leak close. If an extrapleural approach was used, the child is usually less ill than with other approaches, and the resultant esophagocutaneous fistula closes within days. If a transpleural approach was used, then the child is more ill and has an empyema that may require further treatment and drainage.

Recurrent TEF may occur within days; most often, it occurs weeks later. Its incidence has been variously reported as 3 to 14 percent. Its first manifestation may be pneumonia, although the child may cough and have respiratory distress with feeding. The diagnosis is made by means of an esophagography.

CONGENITAL LOBAR EMPHYSEMA (CLE)

CLE refers to the postnatal abnormal overdistension of an otherwise anatomically normal lobe of the lung. It is characterized by expiratory air trapping within the lobe, which produces compression and atelectasis of adjacent lobes. If it is severe, mediastinal shift and impaired venous return can occur. Impaired gas exchange is common in CLE.[120]

CLE is rare, with a 2:1 or 3:1 male preponderance.[120] Congenital heart disease or abnormalities of the great vessels occur in approximately 15 percent of infants. Ventricular septal defect and patent ductus arteriosus are the most common.[135,136]

Embryology/Anatomy[120]

CLE classically develops when the cartilage of the affected bronchus fails to develop, resulting in focal bronchial collapse and air trapping on expiration. However, other causes of bronchial obstruction, either intrinsic or extrinsic, may be responsible for the development of CLE. Intrinsic

causes include bronchial stenosis, bronchial cysts, or redundant bronchial mucosa. Extrinsic bronchial compression may be due to abnormal vessels (such as an aberrant or enlarged pulmonary artery or ductus arteriosus), mediastinal lymphadenopathy, or mediastinal cysts or tumors. Polyalveolar lobe syndrome has also been identified as a cause of CLE. In this syndrome, the involved lobe has three to five times the normal number of alveoli present and air trapping occurs postnatally within these many alveoli.

Usually only one lobe is involved, with 40 percent to 50 percent of cases affecting the left upper lobe, 30 percent to 40 percent affecting the right middle lobe, and 20 percent affecting the right upper lobe. The lower lobes are affected in only 1 percent of cases. Occasionally more than one lobe is involved.

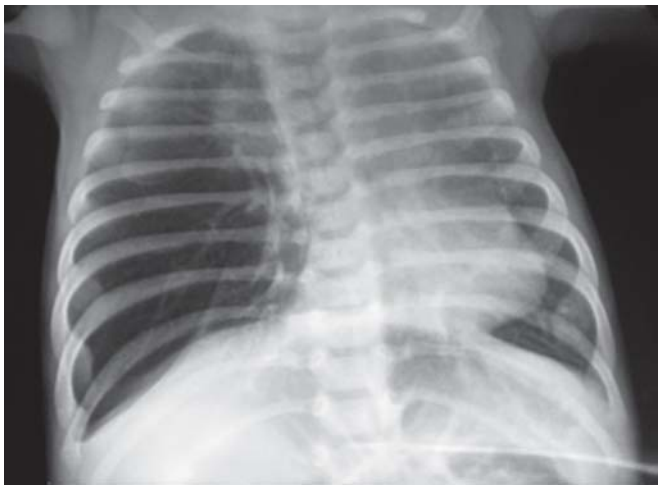


Fig. 18.6: Chest X-ray of a 27 days neonate with CLE of right upper and middle lobe causing mediastinal shift to left. Note the presence of bronchovascular markings till periphery of right lung

Clinical Features

The clinical manifestations of congenital lobar emphysema usually become apparent in the neonatal period, with 25 percent of cases diagnosed at birth and 50 percent of cases diagnosed by 1 month of age.[17] Presentation is variable, depending on the degree of bronchial obstruction and the time it takes for overexpansion to occur. Many infants present with tachycardia, tachypnea, and retractions. If hypoxemia ensues, the infant may become increasingly agitated and anxious. Other symptoms include cyanosis, grunting, and coughing. Physical examination may reveal asymmetric chest expansion with focal hyperresonance and diminished breath sounds over the affected lobe.[120]

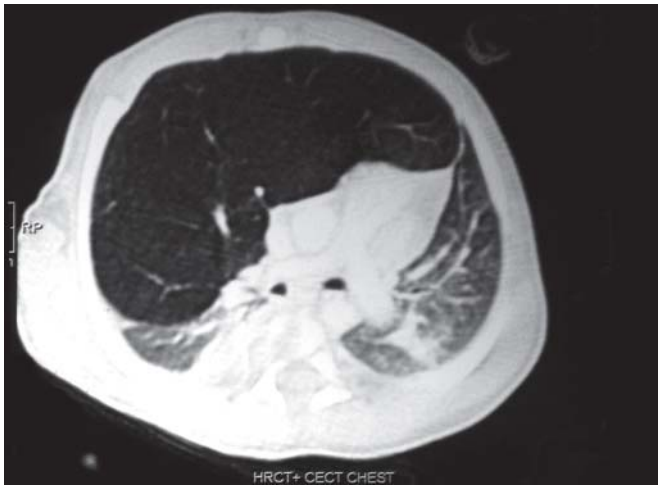


Fig. 18.7: High resolution CT scan from the same neonate showing right upper lobe herniation encroaching left side

Diagnosis

The diagnosis is confirmed by plain chest radiograph and CT scan with typical findings including hyperinflation of the involved lobe, atelectasis of adjacent lung, mediastinal shift, and flattening of the ipsilateral diaphragm (Figs 18.6 and 18.7). The chest radiograph can help differentiate lobar emphysema from pneumothorax or congenital cysts by the presence of faint bronchovascular markings and herniation of the affected lobe across the midline.[137]

Surgical Approach

- Thoracotomy and complete lobectomy are usually required for CLE.[120]
- Thorascopic lobectomy has been described but is infrequently practiced.[120] It can be performed safely and effectively, and avoids the morbidity and poor cosmesis of a thoracotomy. Upper-lobe resections can be technically more challenging, but are still possible with thoracoscopy. Insufflation of the hemithorax with 7 mm Hg carbon dioxide can aid in lung collapse.[72]
- Preresection bronchoscopy has been advocated to determine the cause of bronchial obstruction and its reversibility before sacrificing an otherwise normal lung lobe.[120]
- Significant blood loss is possible (infrequent).
- Often ventilation improves when the aberrant lung segment is removed.
- Chest tube placement at the end of the procedure
- Position: Lateral decubitus for thoracotomy and thoracoscopy.
- Typical surgical time: 2 to 3 hours.

Anesthetic Management

Preoperative evaluation: It depends on the degree of patient distress.[138] If there is rapid deterioration, evaluation is limited. If the patient is stable and there is any question about the diagnosis, procedures such as radioisotope perfusion scans, angiography, or CT imaging can be used before proceeding with definitive surgery. During preanesthetic evaluation, cardiopulmonary stability of the patient is the prime concern. The degree of distress, its progression, and the need for supplemental oxygen are key components of the examination. Identify associated congenital anomalies. Echocardiogram may be needed to rule out CHD. Blood loss is usually minimal for most thoracic surgery, but given the proximity of the great vessels, it may occur suddenly and unexpectedly. Hence, blood should be readily available.[120]

Induction: Induction of anesthesia in infants with CLE is a critical phase in the anesthetic management. The crying, struggling infant can increase the amount of trapped gas.[139] Positive-pressure ventilation or positive airway pressure by the anesthesiologist can also increase the emphysema (gas enters but cannot leave due to a ball-valve effect), with sudden mediastinal shift and cardiac arrest.[17] A smooth inhalation induction with sevoflurane and oxygen is often used, with PPV minimized until the chest is open.[140] Intubation is performed with or without muscle relaxants, depending on the patient's tolerance of PPV. Spontaneous ventilation should be maintained until either the chest is opened or one lung ventilation (OLV) of the contralateral lung is achieved. [141] Controlled or assisted ventilation is added if unacceptable hypoventilation develops. Intubation of the contralateral bronchus, facilitated by the use of muscle relaxants, and the use of PPV have been suggested as an alternative means of airway management by some.[17] If the lobe expands suddenly, the surgeon should be ready to open the chest immediately and relieve the pressure and prevent sudden cardiopulmonary decompensation.[17] General anesthesia may be supplemented with local anesthesia until the chest is opened and the emphysematous lobe is delivered through the incision (Fig. 18.8). Thereafter, these infants may be paralyzed and their lungs mechanically ventilated.[17] Nitrous oxide should not be used, as its diffusion into the diseased lobes can cause further distension.[138]

Raghavendran et al[142] have described a technique involving caudal epidural catheter threaded to the thoracic level in spontaneously breathing patients who were anesthetized with potent inhaled anesthetic agents. An alternative induction approach, especially for unstable infants, is sedation with intravenous ketamine (1 to 2 mg/kg) and local anesthetic infiltration of the incision site.[140] After the intrathoracic pressure has been relieved, general anesthesia can proceed with any technique appropriate to the patient's underlying status. Severely decompensated infants may require emergency needle aspiration or thoracotomy for decompression of the affected lobe or lobes. [17]

Maintenance: OLV is not generally indicated for open thoracotomies in neonates and infants because the surgeon is usually able to manually retract the lung.[120] Nitrous oxide is contraindicated in the case of CLE.[143] Isoflurane may be the preferred volatile agent because it affects hypoxic pulmonary vasoconstriction (HPV) less than other inhaled agents, though this is based on studies in adults.[144] Regardless of which inhalation agent is used, the inspired concentration should be limited (0.5 to 1.0 minimum alveolar concentration [MAC] to minimize the attenuation of HPV.[120] If OLV is not established in patients with CLE, either spontaneous ventilation or gentle, cautious PPV with minimal PIP should be employed.[140] Hypoxia or hypercarbia may occur during OLV or with surgical retraction of the lung. Re-expansion of both lungs periodically may be required. Some degree of hypercarbia may be permissible. Particularly in neonates and small infants, there is a risk of dislodgement of the bronchial blocker with positioning of the patient and during the surgical procedure itself. The tracheal lumen may be occluded by the balloon tip. The position of the blocker should be continuously checked by monitoring airway pressures, auscultating the chest, and ultimately confirming position with direct visualization using a fiberoptic

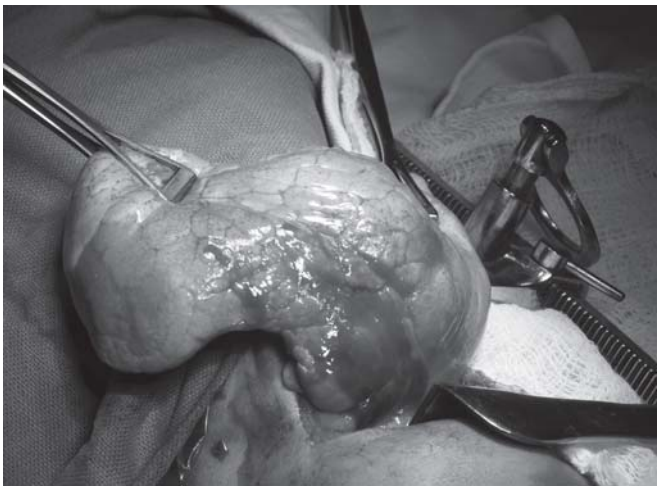


Fig. 18.8: Intraoperative picture from the same baby. Note the herniation of the right upper lobe as soon as chest was opened *(For color version, see Plate 1)*

bronchoscope.[120] High-frequency ventilation has been used successfully in infants with CLE and should be considered, if the practitioner is familiar with the technique. The low airway pressures are especially suitable for these patients.[145]

Monitoring: It includes pulse oximetry to detect rapid changes in oxygenation, especially with induction.[139] Placement of an arterial catheter allows serial blood gas monitoring and earlier detection of hemodynamic changes and hence is recommended by some.[17] In deteriorating patients, there may be little time to establish intra-arterial monitoring before incision. Author does not use invasive arterial monitor. Doppler-assisted or automated blood pressure cuffs increase the accuracy of measurements and are especially useful in neonates. After intubation, capnography is helpful.[139] As mentioned earlier, blood loss is minimal so transfusion is rarely required. Arterial blood gas monitoring is advised to check pH, PO_2, PCO_2, glucose, hematocrit.

Emergence: After most thoracic procedures, the patient's trachea can be extubated at the end of the procedure. Extubation is desirable because an air leak can develop at the bronchial suture (or staple) line with PPV.[120] Adequate oxygenation and ventilation must be confirmed before extubation. All neonates and small infants should be monitored in an intensive care unit (ICU) setting. In patients with limited cardiopulmonary reserve, continued postoperative intubation and ventilation may be required.

Postoperative care: A chest radiograph should be obtained soon after surgery to detect any significant pneumothorax or atelectasis. Humidified oxygen is given to minimize atelectasis.

Pain management: Postoperative pain can cause significant splinting. A smooth postoperative course may be facilitated by regional anesthetic, intercostal block, local anesthetic infiltration and paracetamol suppository.

REFERENCES

1. Soliman D, Cladis F, Davis P. The Pediatric Patient. In: Fleisher LA, Dripps RD (Eds). Anesthesia and Uncommon Diseases, 5th edn, Philadelphia: Saunders, An Imprint of Elsevier 2005:601-39.
2. Brett CM, Davis PJ, Bikhazi G. Anesthesia for Neonates and Premature Infants. In: Motoyama EK, Davis PJ (Eds). Smith's Anesthesia for Infants and Children, 7th edn, Philadelphia: Mosby, An Imprint of Elsevier 2005:550-2.
3. Dickson E. Emergency Surgery: Neonatal, General, and Airway. In: Doyle E (Eds). Paediatric Anaesthesia, 1st edn, New York: Oxford University Press 2007;396-8.
4. Greenwood RD, Rosenthal A, Nadas AS. Cardiovascular abnormalities associated with congenital diaphragmatic hernia. Pediatrics 1976;57:92-7.
5. Fauza DO, Wilson JM. Congenital diaphragmatic hernia and associated anomalies: Their incidence, identification, and impact on prognosis. J Pediatr Surg 1994;29:1113-7.
6. Iriani I. Experimental study on the embryogenesis of congenital diaphragmatic hernia. Anat Embryol 1984; 169:133.
7. David TJ, Illingworth CA. Diaphragmatic hernia in the southwest of England. J Med Genet 1976;13:253.
8. Tonks A, Wyldes M, Sommerset DA, et al. Congenital malformations of the diaphragm: findings of the West Midlands congenital anomaly register 1995 to 2000. Prenatal diagnosis 2004;24:596-604.
9. Tibboel D, Gaag AV. Etiologic and genetic factors in congenital diaphragmatic hernia. Clin Perinatol 1996; 23:689-99.
10. Berry FA, Castro BA. Neonatal Anesthesia. In: Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anesthesia, 5th edn, USA: Lippincott Williams and Wilkins 2006;1181-1204.
11. Flake AW, Crombleholme TM, Johnson MP, et al. Treatment of severe congenital diaphragmatic hernia by fetal tracheal occlusion: Clinical experience with fifteen cases. Am J Obstet Gynecol 2000;183:1059-66.
12. Saifuddin A, Arthur RJ. Congenital diaphragmatic hernia—a review of pre and postoperative chest radiology. Clin Radiol 1993;47(2):104-10.
13. Swischuk LE. Imaging of the Newborn, Infant and Young Child. 4th edn Lippincott Williams and Wilkins 1997:68-72,398-99,412-23.
14. Mitanchez D. Antenatal treatment of congenital diaphragmatic hernia: An update. Archives de Pediatrie 2008;15(8):1320-5.
15. Gucciardo L, Deprest J, Done' E, Van Mieghem T, Van de Velde M, Gratacos E, et al. Prediction of outcome in isolated congenital diaphragmatic hernia and its consequences for fetal therapy. Best Pract Res Clin Obstet Gynaecol 2008;22(1):123-38.
16. Van Loenhout RB, Tibboel D, Post M, Keijzer R. Congenital Diaphragmatic Hernia: Comparison of Animal Models and Relevance to the Human Situation. Neonatology 2009;96(3):137-49.
17. Lee C, Luginbuehl I, Bissonnette B, Mason LJ. Pediatric Diseases. In: Hines RL, Marschall KE (Eds). Stoelting's Anesthesia and Co-Existing Disease, 5th edn, Philadelphia: Churchill Livingstone 2008;658-72.
18. Yaster M, and Rossberg MI. Anesthesia for Newborn Surgical Emergencies. In: Longnecker DE, Brown DL, Newman MF, Zapol DM (Eds). Anesthesiology. New York: The McGraw-Hill Companies, Inc 2008;1502-19.

19. Dillon PW, Cilley RE, Mauger D, et al. The relationship of pulmonary artery pressure and survival in congenital diaphragmatic hernia. J Pediatr Surg 2004;39:307-12; discussion 307-12.
20. Wilcox DT, Irish MS, Holm BA, Glick PL. Prenatal diagnosis of congenital diaphragmatic hernia with predictors of mortality. Clin Perinatol 1996;23(4): 701-9.
21. Deprest JA, Flemmer AW, Gratacos E, Nicolaides K. Antenatal prediction of lung volume and *in utero* treatment by fetal endoscopic tracheal occlusion in severe isolated congenital diaphragmatic hernia. Semin Fetal Neonatal Med 2009;14(1):8-13.
22. Gerards FA, Twisk JW, Fetter WP, Wijnaendts LC, van Vugt JM. Predicting pulmonary hypoplasia with 2 or 3 dimensional ultrasonography in complicated pregnancies. Am J Obstet Gynecol 2008;198(1):140.e1-6.
23. Tsukimori K, Masumoto K, Morokuma S, et al. The lung-to-thorax transverse area ratio at term and near term correlates with survival in isolated congenital diaphragmatic hernia. J Ultrasound Med 2008;27(5):707-13.
24. Taylor GA, Atalabi OM, Estroff JA. Imaging of congenital diaphragmatic hernias. Pediatr Radiol 2009;39(1):1-16.
25. Matsushita M, Ishii K, Tamura M, et al. Perinatal magnetic resonance fetal lung volumetry and fetal lung-to-liver signal intensity ratio for predicting short outcome in isolated congenital diaphragmatic hernia and cystic adenomatoid malformation of the lung. J Obstet Gynaecol Res 2008;34(2):162-7.
26. Liu X, Ashtari M, Leonidas JC, Chan Y. Magnetic resonance imaging of the fetus in congenital intrathoracic disorders: preliminary observations. Pediatr Radiol 2001;31(6):435-9.
27. Mahieu-Caputo D, Sonigo P, Dommergues M, et al. Fetal lung volume measurement by magnetic resonance imaging in congenital diaphragmatic hernia. Br J Obstet Gynecol 2001;108(8):863-8.
28. Paek BW, Coakley FV, Lu Y, et al. Congenital diaphragmatic hernia: prenatal evaluation with MR lung volumetry: preliminary experience. Radiology 2001; 220(1):63-7.
29. Yudkowitz FS. Congenital Diaphragmatic Hernia. In Reed AP, Yudkowitz FS (Eds). Clinical Cases in Anesthesia, 3rd edn. Philadelphia: Elsevier, Churchill Livingstone 2005;399-402.
30. Janssen DJ, Tibboel D, Carnielli VP, et al. Surfactant phosphatidylcholine pool size in human neonates with congenital diaphragmatic hernia requiring ECMO. J Pediatr 2003;142(3):247-52.
31. Lotze A, Knight GR, Anderson KD, et al. Surfactant (beractant) therapy for infants with congenital diaphragmatic hernia on ECMO: evidence of persistent surfactant deficiency. J Pediatr Surg 1994;29(3):407-12.
32. Levin D. Morphologic analysis of the pulmonary vascular bed in congenital left-sided diaphragmatic hernia. The Journal of Pediatrics 1978;92(5):805-9.
33. Mohseni-Bod H, Bohn D. Pulmonary hypertension in congenital diaphragmatic hernia. Semin Pediatr Surg 2007; 16:126-33.
34. Miniati, D. Pulmonary vascular remodelling. Semin Pediatr Surg 2007;16:80-87.
35. Furchgott RF, Zawadzki JV. The obligatory role of endothelial cells in relaxation of arterial smooth muscle by acetylcholine. Nature 1980;288:373-6.
36. Rosenberg AA, Kennaugh J, Koppenhafer SL, et al. Elevated immunoreactive endothelin-1 levels in newborn infants with persistent pulmonary hypertension. J Pediatr 1993;123:109-14.
37. Kobayashi H, Puri P. Plasma endothelin levels in congenital diaphragmatic hernia. J Pediatr Surg 1994; 29:1258-61.
38. Schwartz SM, Vermillion RP, Hirschl RB. Evaluation of left ventricular mass in children with left-side congenital diaphragmatic hernia. J Pediatr 1994;125:447.
39. Morgan GE, Mikhail MS, Murray MJ (Eds). Pediatric Anesthesia. In: Clinical Anesthesiology, 4th edn. USA: McGraw-Hill Companies, Inc 2005;922-50.
40. Bohn D. Congenital diaphragmatic hernia. Am J Respir Crit Care Med 2002;166:911-5.
41. Wung JT, James LS, Kilchevsky E, James E. Management of infants with severe respiratory failure and persistence of fetal circulation, without hyperventilation. Pediatrics 1985;76:488-94.
42. Wung JT, Sahni R, Moffitt ST, et al. Congenital diaphragmatic hernia: survival treated with very delayed surgery, spontaneous respiration, and no chest tube. J Pediatr Surg 1995;30:406-9.
43. Boloker J, Bateman DA, Wung JT, et al. Congenital diaphragmatic hernia in 120 infants treated consecutively with permissive hypercapnia/spontaneous respiration/ elective repair. J Pediatr Surg 2002;37:357-66.
44. Kinsella JP, Abman SH. Inhaled nitric oxide therapy in children. Paediatr Respir Rev 2005; 6:190-8.
45. Kinsella JP, Ivy DD, Abman SH. Pulmonary vasodilator therapy in congenital diaphragmatic hernia: acute, late, and chronic pulmonary hypertension. Semin Perinatol 2005;29:123-8.
46. Walsh-Sukys MC, Tyson JE, Wright LL, et al. Persistent pulmonary hypertension of the newborn in the era before nitric oxide: practice variation and outcomes. Pediatrics 2000;105(1 Pt 1):14-20.
47. Neonatal inhaled nitric oxide study group (NINOS): Inhaled nitric oxide and hypoxic respiratory failure in infants with congenital diaphragmatic hernia. Pediatrics 1997;99:838-45.
48. Finer, NN, Barrington KJ. Nitric oxide for respiratory failure in infants born at or near term. Cochrane Database Syst Rev 2001(4):CD000399.
49. Buysse C, Fonteyne C, Dessy H, et al. The use of dipyridamole to wean from inhaled nitric oxide in congenital diaphragmatic hernia. J Pediatr Surg 2001;36: 1864-5.

50. Kinsella JP, Torielli F, Ziegler JW, et al. Dipyridamole augmentation of response to nitric oxide. Lancet 1995;346 (8975):647-8.
51. Thebaud B, Saizou C, Farnoux C, et al. Dypiridamole, a cGMP phosphodiesterase inhibitor, transiently improves the response to inhaled nitric oxide in two newborns with congenital diaphragmatic hernia. Intensive Care Med 1999;25:300-3.
52. Filan PM, McDougall PN, Shekerdemian LS. Combination pharmacotherapy for severe neonatal pulmonary hypertension. J Paediatr Child Health 2006;42:219-20.
53. Keller RL, Hamrick SE, Kitterman JA, et al. Treatment of rebound and chronic pulmonary hypertension with oral sildenafil in an infant with congenital diaphragmatic hernia. Pediatr Crit Care Med 2004;5:184-7.
54. Keller RL, Moore P, Teitel D, et al. Abnormal vascular tone in infants and children with lung hypoplasia: Findings from cardiac catheterization and the response to chronic therapy. Pediatr Crit Care Med 2006;7:589-94.
55. Noori S, Friedlich P, Wong P, et al. Cardiovascular effects of sildenafil in neonates and infants with congenital diaphragmatic hernia and pulmonary hypertension. Neonatology 2007;91:92-100.
56. Stolar C, Dillon P, Reyes C. Selective use of extracorporeal membrane oxygenation in the management of congenital diaphragmatic hernia. J Pediatr Surg 1988;23:207-11.
57. Semakula N, Stewart DL, Goldsmith LJ, et al. Survival of patients with congenital diaphragmatic hernia during the ECMO era: An 11-year experience. J Pediatr Surg 1997; 32:1683-9.
58. Al-Shanafey S, Giacomantonio M, Henteleff H. Congenital diaphragmatic hernia: Experience without extracorporeal membrane oxygenation. Pediatr Surg Int 2002;18:28-31.
59. Stolar CJ, Snedecor SS, Bartlett RH. Extracorporeal membrane oxygenation and neonatal respiratory failure: Experience from the extracorporeal life support organization. J Pediatr Surg 1991;26:563.
60. Elbourne D, Field D, Mugford M. Extracorporeal membrane oxygenation for severe respiratory failure in newborn infants: Cochrane review. Cochrane Database Syst Rev 2002;CD001340.
61. The Congenital Diaphragmatic Hernia Study Group: Does extracorporeal membrane oxygenation improve survival in neonates with congenital diaphragmatic hernia? J Pediatr Surg 1999;34:720-4.
62. Rothenbach P, Lange P, Powell D. The use of extracorporeal membrane oxygenation in infants with congenital diaphragmatic hernia. Semin Perinatol 2005; 29:40-4.
63. Bos AP, Tibboel D, Hazebroek FW, Molenaar JC, Lachmann B, Gommers D. Surfactant replacement therapy in high-risk congenital diaphragmatic hernia. Lancet 1991;338(8777):1279.
64. Glick PL, Leach CL, Besner GE, et al. Pathophysiology of congenital diaphragmatic hernia. III: Exogenous surfactant therapy for the high-risk neonate with CDH. J Pediatr Surg 1992;27(7):866-9.
65. Katz AL, Wiswell TE, Baumgart S. Contemporary controversies in the management of congenital diaphragmatic hernia. Clin Perinatol 1998;25:219.
66. Colby CE, Lally KP, Hintz SR, et al. Surfactant replacement therapy on ECMO does not improve outcome in neonates with congenital diaphragmatic hernia. Journal of Pediatric Surgery 2004;39(11):1632-7.
67. Harrison MR, Keller RL, Hawgood SB, et al. A randomized trial of fetal endoscopic tracheal occlusion for severe fetal congenital diaphragmatic hernia. N Engl J Med 2003; 349(20):1916-24.
68. Bohn D, Tamura M, Perrin D, et al. Ventilatory predictors of pulmonary hypoplasia in congenital diaphragmatic hernia, confirmed by morphologic assessment. J Pediatr 1987;111:423-31.
69. Bohn DJ, James I, Filler RM, et al. The relationship between $PaCO_2$ and ventilation parameters in predicting survival in congenital diaphragmatic hernia. J Pediatr Surg 1984; 19:666-71.
70. Sakai H, Tamura M, Hosokawa Y, Bryan A, Barker G, Bohn D. Effect of surgical repair on respiratory mechanics in congenital diaphragmatic hernia. The Journal of Pediatrics 1987;111(3):432-8,731-4.
71. Holzman RS. The Body Cavity and Wall. In: Holzman RS, Mancuso TJ, Polaner DM (Eds). A Practical Approach to Pediatric Anesthesia, 1st end, Boston, Massachusetts: Lippincott Williams and Wilkins 2008;286-305.
72. Dutta S, Smith BM, Albanese C, Claure RE, Golianu B, Hammer GB. Pediatric General Surgery. In: Jaffe RA, Samuels SI, Schmiesing CA, Golianu B (Eds). Anesthesiologist's Manual of Surgical Procedures, 4th edn. Philadelphia: Lippincott Williams and Wilkins 2009;1251-1319.
73. Chatterjee D, Seefelder C, Tassone R, Wilson J. Anesthesia for Thoracoscopic Repair of Congenital Diaphragmatic Hernia in Neonates. Anesthesiology 2004;101:A1420.
74. Harrison MR, Adzick NS, Longaker MT, et al. Successful repair *in utero* of a fetal diaphragmatic hernia after removal of herniated viscera from the left thorax. N Engl J Med 1990;322:1582-4.
75. Hedrick MH, Estes JM, Sullivan KM, et al. Plug the Lung Until it Grows (PLUG): A new method to treat congenital diaphragmatic hernia *in utero*. J Pediatr Surg 1994;29:612-7.
76. Albanese CT, Jennings RW, Filly RA, et al. Endoscopic fetal tracheal occlusion: Evolution of techniques. Pediatr Endosurg Innovative Tech 1998;2:47-55.
77. Vanderwall KJ, Skarsgard ED, Filly RA, et al. Fetendo-clip: A fetal endoscopic tracheal clip procedure in a human fetus. J Pediatr Surg 1997;32:970-2.
78. Coté CJ. Pediatric Anesthesia**.** In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL (Eds). Miller's anesthesia, 7th edn, Philadelphia: Churchill Livingstone 2009;2559-98.
79. Vacanti JP, Crone JD, Murphy JD, et al: The pulmonary hemodynamic response to perioperative anesthesia in the

treatment of high-risk infants with congenital diaphragmatic hernia. J Pediatr Surg 1984;19:672-9.
80. Hodgson RE, Bosenberg AT, Hadley LG: Congenital diaphragmatic hernia repair impact of delayed surgery and epidural analgesia. S Afr J Surg 2000;38:31-5.
81. Iocono JA, Cilley RE, Mauger DT, et al. Postnatal pulmonary hypertension after repair of congenital diaphragmatic hernia: Predicting risk and outcome. J Pediatr Surg 1999;34:349.
82. Gross RE. The Surgery of Infancy and Childhood, Philadelphia: WB Saunders 1953;76.
83. Spitz L, Kiely EM, Morecroft JA, Drake DP. Oesophageal atresia: At-risk groups for the 1990s. J Pediatr Surg 1994; 29:723-5.
84. Engum SA, Grosfeld JL, West KW, Rescorla FJ, Scherer LR 3d. Analysis of morbidity and mortality in 227 cases of esophageal atresia and/or tracheoesophageal fistula over two decades. Arch Surg 1995;130:502-8.
85. German JC, Mahour GH, Woolley MM. Esophageal atresia and associated anomalies. J Pediatr Surg 1976;11:299-306.
86. Sillen U, Hagberg S, Rubenson A, Werkmaster K. Management of esophageal atresia: review of 16 years' experience. J Pediatr Surg 1988;23:805-9.
87. Holder TM, Ashcraft KW, Sharp RJ, Amoury RA. Care of infants with esophageal atresia, tracheoesophageal fistula, and associated anomalies. J Thorac Cardiovasc Surg 1987; 94:828-35.
88. Poenaru D, Laberge JM, Neilson IR, Guttman FM. A new prognostic classification for esophageal atresia. Surgery 1993;113:426-32.
89. Chittmittrapap S, Spitz L, Kiely EM, Brereton RJ. Oesophageal atresia and associated anomalies. Arch Dis Child 1989;64:364-8.
90. Ein SH, Shandling B, Wesson D, Filler RM. Esophageal atresia with distal tracheoesophageal fistula: Associated anomalies and prognosis in the 1980s. J Pediatr Surg 1989; 24:1055-9.
91. Rokitansky A, Kolankaya A, Bichler B, Mayr J, Menardi G. Analysis of 309 cases of esophageal atresia for associated congenital malformations. Am J Perinatol 1994; 11:123-8.
92. Weaver DD, Mapstone CL, Yu PL. The VATER association. Analysis of 46 patients. Am J Dis Child 1986;140:225-9.
93. Brown AK, Roddam AW, Spitz L, Ward SJ. Oesophageal related malformation and medical problems: A family study. Am. J Med Genet 1999;85(I):31-7.
94. Budhiraja S, Rattan KN, Gupta S, Pandit SK. Tracheooesophageal fistula with hydrocephalous. Indian J Paediatr 1997;64(5):727-9.
95. Quan L, Smith DW. The VATER association: Vertebral defects, anal atresia, tracheooesophageal fistula with oesophageal atresia, radial dysplasia. Birth Defects 1973;8:75.
96. Rittler M, Paz JE, Castilla EE. VACTERL association, epidemiologic definition and delineation. Am J Med Gen 1996;63:529-36.
97. Chietero M. Tracheoesophageal Fistula. In Reed AP, Yudkowitz FS (Eds). Clinical Cases in Anesthesia, Third edn, Philadelphia: Elsevier, Churchill Livingstone 2005;405-8.
98. Choudhury SR, Ashcraft KW, Sharp RJ, et al. Survival of patients with esophageal atresia: Influence of birth weight, cardiac anomaly and late respiratory complications. J Pediatr Surg 1999;34:70-4.
99. Cozzi DA, Mele E, Totonelli G, Ceccanti S, Frediani S, Cozzi F. Asymmetric sweating and flushing in infants with esophageal atresia. J Pediatr Surg 2009;44(6): E27-9.
100. Gardella C, Tomà P, Sacco O, Girosi D, Panigada S, Battistini E, et al. Intermittent gaseous bowel distention: Atypical sign of congenital tracheoesophageal fistula. Pediatr Pulmonol 2009;44(3):244-8.
101. Altman AR, Ball WS Jr, Kosloske AM. Radiographic evaluation of the postoperative neonatal chest. Curr Probl Diagn Radiol 1984;13(1):1-40.
102. Berdon WE, Baker DH. Radiographic findings in esophageal atresia with proximal pouch fistula (type B). Pediatr Radiol 1975;3(2):70-4.
103. Berdon WE, Baker DH, Schullinger JN. Plain film detection of right aortic arch in infants with esophageal atresia and tracheoesophageal fistula. J Pediatr Surg 1979;14(4):436-7.
104. Sumner TE, Auringer ST, Cox TD. A complex communicating bronchopulmonary foregut malformation: Diagnostic imaging and pathogenesis. Pediatr Radiol 1997;27(10):799-801.
105. Day DL. Aortic arch in neonates with esophageal atresia: Preoperative assessment using CT. Radiology 1985; 155(1):99-100.
106. Fitoz S, Atasoy C, Yagmurlu A. Three-dimensional CT of congenital esophageal atresia and distal tracheo-esophageal fistula in neonates: preliminary results. AJR Am J Roentgenol 2000;175(5):1403-7.
107. Griscom NT, Martin TR. The trachea and esophagus after repair of esophageal atresia and distal fistula: Computed tomographic observations. Pediatr Radiol 1990;20(6):447-50.
108. Halber MD, Daffner RH, Thompson WM. CT of the esophagus: I. Normal appearance. Am J Roentgenol 1979;133(6):1047-50.
109. Hupscher DN. Computed tomography of the esophagus. In: Radiology of the Esophagus. Thieme medical publishers 1988;53-8.
110. Inoue K, Yanagihara J, Ono S. Utility of helical CT for diagnosis and operative planning in tracheomalacia after repair of esophageal atresia. Eur J Pediatr Surg 1998; 8(6):355-7.
111. Kao SC, Smith WL, Sato Y. Ultrafast CT of laryngeal and tracheobronchial obstruction in symptomatic postoperative infants with esophageal atresia and tracheoesophageal fistula. Am J Roentgenol 1990; 154(2):345-50.
112. Quint LE, Glazer GM, Orringer MB. Esophageal imaging by MR and CT: Study of normal anatomy and neoplasms. Radiology 1985;156(3):727-31.

113. Schmalfuss IM, Mancuso AA, Tart RP. Postcricoid region and cervical esophagus: normal appearance at CT and MR imaging. Radiology 2000;214(1):237-46.
114. Tam PK, Chan FL, Saing H. Diagnosis and evaluation of esophageal atresia by direct sagittal CT. Pediatr Radiol 1987;17(1):68-70.
115. Kalache KD, Chaoui R, Mau H. The upper neck pouch sign: A prenatal sonographic marker for esophageal atresia. Ultrasound Obstet Gynecol 1998;11(2):138-40.
116. Kalache KD, Wauer R, Mau H. Prognostic significance of the pouch sign in fetuses with prenatally diagnosed esophageal atresia. Am J Obstet Gynecol 2000;182(4):978-81.
117. Rossi C, Domini M, Aquino A. A simple and safe method to visualize the inferior pouch in esophageal atresia without fistula. Pediatr Surg Int 1998;13(7):535-6.
118. Spitz L. Esophageal atresia: Past, present, and future. J Pediatr Surg 1996;31:19-25.
119. Dillon PW, Cilley RE. Newborn surgical emergencies. Gastrointestinal anomalies, abdominal wall defects. Pediatr Clin North Am 1993; 40:1289-314.
120. Kang BV, Koka BV. The Foregut and Chest. In: Holzman RS, Mancuso TJ, Polaner DM, (Eds). A Practical Approach to Pediatric Anesthesia, 1st edn, Boston, Massachusetts: Lippincott Williams and Wilkins 2008; 383-90.
121. Maxwell LG. Anesthetic Management for Newborns Undergoing Emergency Surgery. In: Schwartz AJ, Butterworth JF, Gross JB (Eds). Asa Volume Thirty-Five. Philadelphia: Lippincott Williams and Wilkins 2007;107-26.
122. Koka BV, Chacko SK. Airway management of a newborn with a tracheoesophageal fistula. In: Murphy M, Hung O, (Eds). Airway management and monitoring manual. New York: McGraw-Hill 2006.
123. Roberts JD, Hobai I, Cronin JH. Anesthesia for Surgical Emergencies in the Neonate. In: Dunn PF, Alston TA, Baker KH, Davison JK, Kwo J, Rosow CE (Eds), Clinical Anesthesia Procedures of the Massachusetts General Hospital, 7th edn, Lippincott Williams and Wilkins 2007:492-514.
124. Salem MR, Wong AY, Lin YH, et al. Prevention of gastric distention during anesthesia for newborns with tracheoesophageal fistulas. Anesthesiology 1973;38:82-3.
125. Filston HC, Chitwood Jr WR, Schkolne B, et al. The Fogarty balloon catheter as an aid to management of the infant with esophageal atresia and tracheoesophageal fistula complicated by severe RDS or pneumonia. J Pediatr Surg 1982;17:149-51.
126. Greemberg L, Fisher A, Katz A. Novel use of neonatal cuffed tracheal tube to occlude tracheo-oesophageal fistula. Paediatr Anaesth 1999;9(4):339-41.
127. Karl HW. Control of life-threatening air leak after gastrostomy in an infant with respiratory distress syndrome and tracheoesophageal fistula. Anesthesiology 1985;62:670.
128. Bosenberg AT, Bland BA, Schulte-Steinberg O, et al. Thoracic epidural anesthesia via caudal route in infants. Anesthesiology 1988;69:265-9.
129. Bosenberg AT, Hadley GP, Murray WB. Epidural analgesia reduces postoperative ventilation requirements following esophageal atresia repair. J Pain Symptom Management 1991;6:209.
130. Tobias JD, Rasmussen GE, Holcomb GW 3, et al. Continuous caudal anaesthesia with chloroprocaine as an adjunct to general anaesthesia in neonates. Can J Anaesth 1996;43:69-72.
131. Andropoulos DB, Rowe RW, Betts JM. Anaesthetic and surgical airway management during tracheo-oesophageal fistula repair. Paediatr Anaesth 1998;8(4):313-9.
132. Aitkenhead AR, Rowbotham DJ, Smith G (Eds). Paediatric anaesthesia and intensive care. In: Textbook of Anaesthesia, 4th edn, Edinburgh London: Churchill Livingstone 2001;650-63.
133. Vas L, Kulkarni V, Mali M, Bagry H: Spread of radiopaque dye in the epidural space in infants. Paediatr Anaesth 2003; 13:233-43.
134. Konkin DE, O'hali WA, Webber EM, Blair GK: Outcomes in esophageal atresia and tracheoesophageal fistula. J Pediatr Surg 2003;38:1726-9.
135. Pierce WS, DeParedes CG, Friedman S, et al. Concomitant congenital heart disease and lobar emphysema in infants: Incidence, diagnosis, and operative management. Ann Surg 1970;172(6):951-6.
136. Jones JC, Almond CH, Snyder HM, et al. Lobar emphysema and congenital heart disease in infancy. J Thorac Cardiovasc Surg 1965;49:1-10.
137. Pinkerton HJ, Oldham KT. Lung. In: Oldham KT, Colombani PM, Foglia RP (Eds). Principles and practice of pediatric surgery, Vol. 2. Philadelphia: Lippincott Williams and Wilkins 2005;951-62.
138. Payne K, Van Zyl W, Richardson M. Anaesthesia for congenital lobar emphysema resection. South Afr Med J 1984;66:421.
139. Hammer G, Hall S, Davis PJ. Anesthesia for General Abdominal, Thoracic, Urologic, and Bariatric Surgery. In: Motoyama EK, Davis PJ (Eds). Smith's Anesthesia for Infants and Children, 7th edn Philadelphia: Mosby, An Imprint of Elsevier 2005;706-7.
140. Cote CJ. The anesthetic management of congenital lobar emphysema. Anesthesiology 1978;49(4):296-8.
141. Tobias JD. Anaesthesia for neonatal thoracic surgery. Best Pract Res Clin 2004;18(2):303-20.
142. Raghavendran S, Kiwan R, Shah T, Vas L. Continuous caudal epidural analgesia for congenital lobar emphysema: A report of three cases. Anesth Analg 2001; 93:348.
143. Hammer GB. Pediatric thoracic anesthesia. Anesthesiol Clin North America 2002;20(1):153-80.
144. Benumof JL, Augustine SD, Gibbons JA. Halothane and isoflurane only slightly impair arterial oxygenation during one-lung ventilation in patients undergoing thoracotomy. Anesthesiology 1987;67(6):910-5.
145. Goto H, Boozalis ST, Benson KT, et al. High-frequency jet ventilation for resection of congenital lobar emphysema. Anesth Analg 1987;66:684.

19 Anesthesia for Neonatal and Pediatric Abdominal Emergencies

Anila D Malde, Trupti S Pethkar, Aklesh S Tandekar

KEY POINTS

Abdominal Wall Defects

- It includes gastroschisis (without cover) and omphalocele (with cover). Later is divided into exomphalos major and minor.
- Prematurity is more common with gastroschisis. While associated anomalies are more common with omphalocele, hence preoperative cardiac evaluation is must.
- Fluid resuscitation, prevention of heat loss, treatment of sepsis, and protection of herniated viscera are major concerns.
- Watch for development of abdominal compartment syndrome after tight abdominal closure. Intravenous lines are usually taken in upper limbs as abdominal distension can impair venous return from the lower body.
- It is safer to continue ventilatory support postoperatively for large gastroschisis and exomphalos major.

Congenital Hypertrophic Pyloric Stenosis

- These patients show hypochloremic, hypokalemic, hypovolemic metabolic alkalosis.
- Give 5% dextrose with 0.45% saline as indicated by serum electrolytes and fluid deficits. Add potassium chloride supplementation once the urine flow is established.
- Surgery should be delayed until biochemical values reach: pH 7.3 to 7.5, serum HCO_3 < 28-30 mEq/l, Na^+ > 132 mEq/l, K > 3.5 mEq/l, Cl^- > 90 mEq/l and urinary Cl^- > 20 mEq/l with adequate preoperative hydration.
- Due to higher incidence of postoperative apneoic spells, apnea monitoring is necessary.

Necrotizing Enterocolitis

- These babies are premature and have coagulative or ischemic necrosis leading to pneumatosis intestinalis and hypovolemic shock.
- Thrombocytopenia, deranged coagulation profile and metabolic acidosis need correction before induction. Fluid, blood, FFP and platelets may be needed.
- Hypoxia is a greater danger than retinopathy and must be avoided. Arterial oxygen tensions need to be maintained at 50 to 70 mm Hg intraoperatively.

Atresia

- The higher prevalence of associated congenital malformations with duodenal atresia warrants preoperative cardiology evaluation and echocardiography.
- Coexisting tracheoesophageal fistula is repaired before correction of duodenal atresia.
- Orogastric decompression of the stomach and fluid resuscitation should be promptly initiated. Umbilical arterial lines are replaced with a peripheral arterial line so that mesenteric blood flow is not compromised.
- Nonbilious and bilious nasogastric tube aspirates are replaced by Isolyte G and Isolyte E respectively.

Meconium Ileus

- Meconium ileus is the earliest clinical manifestation of cystic fibrosis.

Malrotation and Volvulus

- This is a true neonatal emergency and surgery should proceed as reasonably fast as possible. Delay in surgery may result in necrosis of the entire small intestine.
- These infants may have hypotension, hypovolemia, and electrolyte abnormalities.
- The intravascular deficits may be exacerbated by the presence of barium in the gastrointestinal tract.

- Fluid and electrolyte resuscitation begins preoperatively and continues during surgery.
- Blood and blood products should be available in the operating room.

Anorectal Malformations

- Decompressive colostomy before definitive surgery.
- Echocardiography is needed to rule out associated congenital heart disease.
- Spinal abnormalities should be ruled out before giving caudal anesthesia.

Intussusception

- Intussusception results in bowel obstruction, which progresses to arterial obstruction and subsequent necrosis of the bowel, fluid sequestration and bleeding from the GI tract, perforation, and sepsis.
- Regional anesthesia is avoided as it may lead to exaggerated bowel movements and precipitate perforation.

Roundworm Obstruction

- Associated with malnutrition, iron-deficiency anemia, and impairments of growth and cognition.
- Child is kept NBM and 25% dextrose is supplemented IV to provide calories.
- Calcium gluconate 1 ml/kg increases GI motility which helps to propel the worms distally.
- Anaphylactic reaction leads to severe hypotension and metabolic acidosis.

All these babies are considered full stomach. Aspiration of the gastric contents with a wide bore vented orogastric tube and rapid sequence induction are necessary.

Abdominal procedures which requires emergency management includes abdominal wall defects, congenital hypertrophic pyloric stenosis, necrotizing enterocolitis, atresias, anorectal malformations, malrotation, volvulus, meconium ileus, intussusception and intestinal obstruction.

ABDOMINAL WALL DEFECTS

Introduction

Congenital abdominal wall defects permit external herniation of abdominal viscera including stomach, bladder, uterus and rarely liver. Peritoneal cover is either absent (gastroschisis: <5 cm) or present (omphalocele).[1,2] Although both appear to be similar, these are distinct from each other.[3] Omphalocele is further classified as exomphalos minor (5-8 cm) and exomphalos major (10-12 cm, includes liver, with poorly developed abdominal and thoracic cavities and pulmonary hypoplasia) (Figs 19.1 and 19.2).

Incidence

Overall incidence is 0.3 to 2:10, 000 births.[3] Incidence of herniation of intestines is approximately 1 in 5000 and that of liver and intestines is 1 in 10,000 live births. Cardiac defects and prematurity are the major causes of the 30% mortality among newborns with an omphalocele.[2]

Comparison of Gastroschisis and Omphalocele[1-6] is in Table 19.1.

Gastroschisis is a stand-alone congenital defect where babies are often small for gestational age and born to mothers with a history of cigarette, alcohol, and drug intake during the first trimester. A change in paternity, sexually transmitted disease, urinary tract infection and use of oral decongestants in early pregnancy shows the association. In these patients, short gut syndrome may be a consequence of the attenuated intestinal length. Even with adequate length, the remnant bowel may be damaged to the point that motility, digestion, and absorption are markedly impaired. This prenatal intestinal injury accounts for most of the postoperative morbidity and mortality.

Diagnosis

In vivo: Elevated maternal serum α-fetoprotein.

Fetal ultrasound detects anatomic defects in about 95% of cases which allows elective cesarean section at 38 weeks and immediate surgical repair at a medical centre with resources for high-risk obstetric, surgical/anesthetic, and neonatal care where surgery can be performed, or transferred immediately after surgery.[3]

Preoperative Assessment

Embryologically, though these are two separate conditions, both present similar challenges to the anesthesiologist. Following points are considered[2]:

- Gestational age, birth history and vitamin K administration. Premature neonates may require surfactant administration.
- Difficult intubation in neonates may be associated with micrognathia or abnormal tissues (large tongue in BWS).

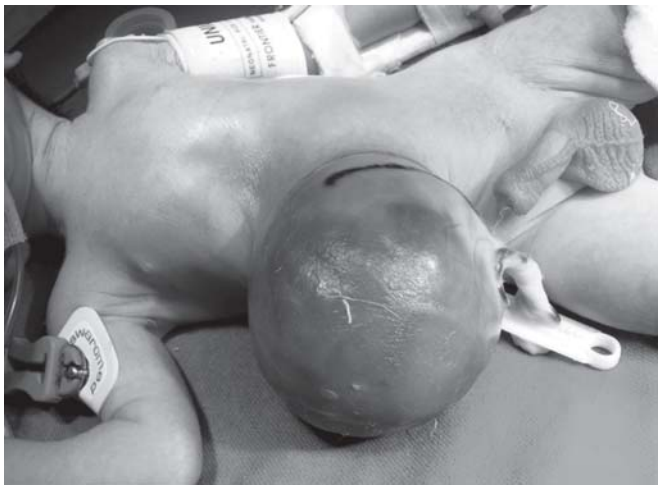


Fig. 19.1: Exomphalos major *(For color version, see Plate 1)*

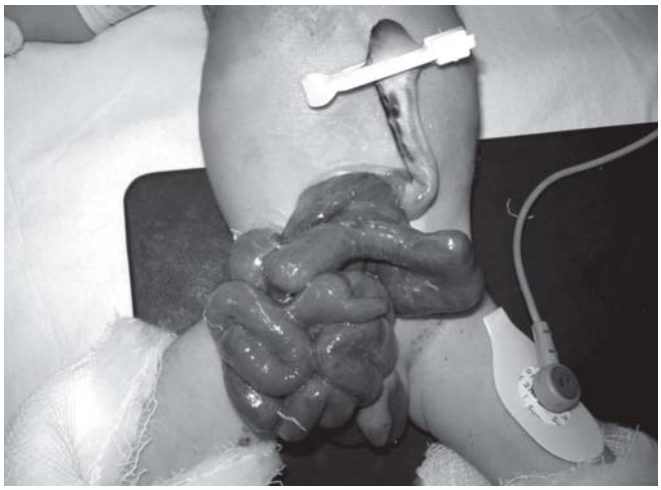


Fig. 19.2: Gastroschisis *(For color version, see Plate 1)*

- Exomphalos major is associated with a scaphoid abdomen and small thorax. Respiratory distress may present with signs of increased work of breathing, i.e. tachypnea, tachycardia, and nasal flaring or apnea. Distressed infants may desaturate easily. Chest X-ray (CXR) may show intrapulmonary shadowing suggesting of infection due to aspiration; position of the tracheal tube and NGT if present.
- Gastroschisis and ruptured exomphalos major may present with significant fluid loss requiring fluid resuscitation. Assess the baby clinically—skin perfusion, capillary refill, heart rate (HR), blood pressure (BP) and blood gases. Fluid challenge may be required. A baby requiring significant volume resuscitation should be electively intubated.
- Examine for associated abnormalities, especially in exomphalos.

Investigations

- Full blood count and crossmatch of one adult unit of blood.
- Arterial blood gas (ABG) to assess the adequacy of resuscitation and ventilation.
- Chest X-ray for all infants with cardiorespiratory symptoms.
- Echocardiogram if the physical examination suggests underlying congenital heart disease.
- Renal ultrasound, especially if syndromic.
- Head ultrasound to exclude intracranial hemorrhage in premature infants.

Preoperative Management

The herniated abdominal contents offer a large surface area by which fluid and heat can be lost. Dehydration and hypothermia are usually more serious in gastroschisis, as the protective hernial sac is absent.[7]

There is severe dehydration and massive fluid loss, both from the exposed visceral surfaces and from third-space losses caused by partial bowel obstruction. An IV line in the upper limb is secured for administration of fluids and broad-spectrum antibiotics. The initial fluid requirement in these neonates is increased and should be administered at a rate 2 to 4 times the daily maintenance requirements (8–16 ml/kg/hr) to ensure adequate hydration and to compensate for a combination of peritonitis, edema, ischemia, and protein loss. Without such vigorous fluid resuscitation, hypovolemic shock, hemoconcentration, and metabolic acidosis may develop. To maintain normal oncotic pressures, protein containing solutions (5% albumin) should constitute approximately 25% of the replacement fluid which is usually a balanced salt solution (lactated Ringer's solution). Urine output of 1 to 2 ml/kg/hr indicates adequate hydration. Hypovolemia is indicated by hemoconcentration and metabolic acidosis. If severe metabolic acidosis develops despite fluid delivery, sodium bicarbonate, colloids, ventilatory support, and vasopressors may be needed to maintain the pH >7.20. Because of the large fluid requirements, acid-base status and electrolyte levels should be monitored serially.[1-3]

Decompression of the stomach with an orogastric tube or nasogastric tube (NGT) is done to prevent regurgitation, aspiration pneumonia, and further bowel distension due to swallowed air.[6]

Table 19.1: Comparison of gastroschisis and omphalocele

	Gastroschisis	*Omphalocele*
Pathophysiology	Occlusion of the right omphalomesenteric artery or right umbilical vein development	Failure of midgut migration from the yolk sac into the abdomen
Incidence	<1 in 15,000 births	<1 in 6000 births
Incidence of prematurity	Higher (58%)	Comparatively lower (33%)
Etiology	Abnormal development of right omphalomesenteric artery or umbilical vein with ischemia to right paraumbilical area. Maternal age <20 yrs (11 fold increased risk)[4]	Failure of the cephalic, lateral, and caudal folds to fuse (closure of the exocelomic space) and abnormal fusion and differentiation of myotomes to form abdominal wall musculature (7 to 12 weeks of gestation). Abdominal cavity is primarily underdeveloped. Maternal age >40 yrs.[5]
Incidence of associated anomalies	<10-15%	<40-60%
Location	Periumbilical (lateral defect usually to right of cord)	Central defect through the base of the umbilical cord
Covering	None (exposed viscera)	Membranous sac with peritoneal membrane internally and the amniotic membrane externally, without overlying skin, unless the sac ruptures *in-utero*.
Contents	Small or large bowel, rarely liver	Stomach, loops of small and large intestine, and, in about 30 to 50%, the liver
Condition of bowel	Bowel wall may be thickened, edematous, matted together, and foreshortened with a fibrin 'peel' due to exposure to amniotic fluid, the umbilical cord is normal and separate from the defect	Bowel looks normal
Problems associated with the defect	Inflammation of exposed gut, edema, dilated and foreshortened gut (chemical peritonitis), malrotation of GIT, prematurity	Syndromes of midline defects often include an omphalocele. Congenital heart disease (<20%), exstrophy of bladder, BWS (omphalocele, profound hypoglycemia, hyperinsulinemia, hyperviscosity syndrome, visceromegaly, macrosomia, macroglossia, gigantism), Reiger and prune belly syndromes, trisomies 13, 18, 21 (30%), malrotation of GI tract, exstrophy of bladder, Edward's syndrome, pentalogy of Cantrell (omphalocele, diaphragmatic hernia, sternal abnormallities, ectopic and anomalous heart, and gene abnormalities at Xq25 to q26.1) Another syndrome involving the lower abdomen include omphalocele, bladder or cloacal exstrophy, imperforate anus, colonic atresia, sacrovertebral anomalies, and meningomyelocele[3,6]
Complications	Moalrotation, volvulus, poor gut motility, intestinal atresia, stenosis (15%)	Malrotation (uncommon), volvulus, intestinal atresia, stenosis
Management: immediate	Cover bowel to reduce evaporative loss, IV fluids, NGT, antibiotic; urgent surgery.	Bowel covered by sac; semi-urgent surgery unless sac ruptures, then as for gastroschisis
Surgical	Staged surgery common.	Single stage surgery may be possible—staged for exomphalos major

IV: intravenous, NGT: nasogastric tube, GIT: gastrointestinal tract, BWS: Beckwith-Wiedemann syndrome

Vigorous efforts are taken to maintain normothermia. To prevent hypothermia, dehydration and infection, exposed viscera must be covered with sterile, moist dressings, cellophane or polythene bag immediately after birth. The exposed bowel can simply be placed into the abdomen with a planned reoperation after several weeks. The sooner the bowel is reduced, the more likely primary closure can be achieved and the less severe the degree of bowel wall edema and accumulation of fibrinous coating (Fig. 19.3). Gastroschisis requires urgent repair.[6] Single stage surgery with reduction of the bowel and closure of the abdominal wall defect may be possible for exomphalos minor. But exomphalos major and gastroschisis generally require staged surgery as

Fig. 19.3: Gastroschisis with edematous bowel
(For color version, see Plate 1)

'Abdominal compartment syndrome' (ACS) may occur if the abdominal contents are reduced under pressure, particularly in exomphalos major if the abdominal cavity is small.

While omphalocele also requires urgent corrective surgery, the frequency of associated cardiac anomalies warrants preoperative cardiology evaluation and echocardiography. Surgical management of omphalocele is dictated by its size. Usually, the contents within the sac are reduced back into the abdomen, the sac is excised and the fascia and skin are closed. In giant omphaloceles, due to visceroabdominal disproportion gradual reduction to the peritoneal cavity is done with a silastic silo over a several-day period, followed by a second procedure a few days later for complete closure.[8] Suggested criteria for a staged closure include intragastric or intravesical pressure >20 cm H_2O, peak inspiratory pressure >35 cm H_2O, or an end-tidal carbon dioxide >50 mm Hg.[7] In either situation, postoperative ventilation will be necessary until the abdominal wall has had time to stretch to accommodate the viscera as the compliance is reduced by return of the viscera to the peritoneum. Improved outcome has been claimed using the delayed repair after the nonoperative placement of a spring-loaded silo.[9,10] This procedure is accomplished in the neonatal intensive care unit (NICU) or delivery room and requires "no anesthesia", thus minimizing repeated trips to the operating room and the need for postreduction ventilation.[10] The prosthesis is then removed or reduced under general anesthesia, and eventually the defect is closed. Monitoring of intra-abdominal pressure during reduction may prevent the development of an abdominal compartment syndrome. After the contents are returned to the abdomen, the infant is taken back to the operating room for formal fascia and skin closure.[6]

Effects of Increased Abdominal Pressure after a Tight Closure

Tight surgical abdominal closure can result in ACS which includes hypotension secondary to tension on a major organ (liver) and cava. Decrease in venous return not only leads to reduction in cardiac output but also congestion of organs, ultimately resulting in impaired perfusion. End result is bowel necrosis, perforation, necrotizing enterocolitis, acidosis, anuria, hepatic impairment, altered drug metabolism and excretion.[1,3] Decreased diaphragmatic excursion, lung compression and decreased pulmonary compliance results in impaired ventilation.[2] During abdominal closure, the anesthesiologist must monitor airway pressures and watch for decreased pulmonary compliance. The surgeon and the anesthesiologist should cooperate to assess the feasibility of a primary closure. Increases in intragastric pressure measured through a gastrostomy tube >20 mm Hg, increases in central venous pressure (CVP) of >4 mm Hg above baseline and failure of saphenous IV line (if *in situ*) fluid to flow by gravity were frequently associated with reductions in venous return and cardiac index, requiring surgical decompression of the abdomen.[11,12]

Forceful closure of the abdominal wall will also cause significant tension of the skin resulting in a high incidence of necrosis with secondary infection. Blood pressure and pulse oximetry measurements from a lower extremity will help identify circulatory problems. Lower extremity congestion and cyanosis may also be seen if venous return from the lower body is impaired.[7]

Anesthetic Management

- Explain anesthetic plan and risk to the parents.
- No sedative premedication; administer only atropine prior to induction.
- Take measures for adequate hydration and to prevent hypothermia.
- Monitoring of BP, ECG and SpO_2
- Large bore IV access in the upper limbs is preferable as abdominal pressure may restrict venous return temporarily in the postoperative period. Place a second line after induction.
- Though Vane and others have demonstrated utility of spinal anesthesia for the repair of gastroschisis, general anesthesia with or without caudal analgesia is preferred by most.[13]

Induction: Awake intubation is traumatic and is not preferred. After decompression of the stomach and pre-

oxygenation, a modified rapid sequence induction with thiopentone (2-3 mg/kg) or sevoflurane is done. Suxamethonium or atracurium should be used to facilitate intubation.[7] Ventilation is done with air and oxygen with low concentrations of an inhalation anesthetic maintaining peak inspiratory pressures (PIPs) <20 cm H_2O in order to prevent compression damage to tracheal mucosa. Correct placement of the endotracheal tube is confirmed by chest auscultation and end tidal carbon dioxide measurements. Nitrous oxide is avoided as it distends the bowel and interferes with reduction of hernia. The inspired oxygen concentration must be adjusted to maintain oxygen saturation between 95 and 97%. Atracurium is the muscle relaxant of choice as it is eliminated by Hoffmann's degradation. The liberal use of muscle relaxants provides optimal surgical conditions for closure of the defect. Low to moderate concentrations of volatile anesthetics are administered to avoid hypotension.[2,3]

Intraoperative analgesia: Usually with opioids such as fentanyl in increments of 1 µg/kg is used if postoperative ventilation is anticipated. A caudal epidural catheter requires an experienced anesthesiologist but provides very good intra and post-operative analgesia and has the added benefit of abdominal wall relaxation. Catheter tip is placed at the midpoint of incision. A single shot caudal epidural block may be performed. The maximum dose of 0.25% bupivacaine is 0.8 to 1 ml/kg. Hepatic clearance of local anesthetics may be reduced if there is any abdominal compression.

Intraoperative monitoring: An arterial catheter facilitates continuous BP monitoring and blood sampling. A CVP catheter is important for evaluation of changes in blood volume, visceral compression during abdominal closure and for provision of total parenteral nutrition (TPN) till recovery of gut function. Serial intraoperative glucose monitoring is indicated as hypoglycemia can occur in infants with BWS. Intragasrtric and intravesical pressure monitoring can determine the surgical course.

Intraoperative fluid management: Intraoperative fluid requirement is usually at least 25% of estimated blood volume during surgical repair of large abdominal wall defects. Fluid therapy consists of 5 to 10% dextrose in 0.2% saline at the maintenance rates and lactated Ringer's solution (8 to 15 ml/kg, or more, per hour) for third-space loss. Extreme clinical vigilance and intensive monitoring are recommended. Blood loss should be replaced with warmed blood and Hb is maintained at approximately 10 to 12 g/dl. Fresh frozen plasma is administered in 10 ml/kg aliquots and platelets are given if the platelet count or coagulation screen is abnormal.

Termination of anesthesia: Some babies with small defects can breathe spontaneously after surgery, but the majority requires ventilation due to the increased intra-abdominal pressure with compromised respiratory function. Neonates should only be extubated when they are fully awake, with regular spontaneous breathing and vigorous movements of all limbs, well saturated and with stable hemodynamics.

Postoperative Care

Postoperative management is continued in neonatal intensive care unit, except for infants with a small defect who could be extubated at the conclusion of surgery.

Fluid management: Fluids should be restricted to 60% maintenance immediately postoperatively as fluid restriction will already be in place if surgery is on the first day of life. Check fluid balance and electrolytes to determine subsequent fluid requirements. 10% dextrose or 4% dextrose in 0.18% saline is used initially, but gastrointestinal losses should be replaced with 0.9% saline ml for ml; only isotonic fluids should be used for correction of hypovolemia. Colloids (gelatins, starch and albumin depending on local preference) may be used to replace third space losses, including those following placement of an abdominal wall silo. Daily maintenance fluid is continued.[3]

Glucose control: Newborn babies less than 48 hrs, especially premature, small for gestational age and those born to diabetic mothers are prone to hypoglycemia. It is important to measure blood glucose levels regularly and to treat hypoglycemia with 1 to 2 ml/kg of 10% glucose. Some may require a continuous infusion of glucose.

Respiratory system: Primary closure may require the ability to ventilate the patient with high PIPs into the postoperative period. Evidence of unacceptable intra-abdominal pressure requires removal of fascial sutures and closure of only the skin or addition of prosthesis. Neonates with exomphalos major or gastroschisis may require mechanical ventilation due to temporary deterioration of lung function due to the raised intra-abdominal pressure; some may require muscle relaxation for 24 hrs and are gradually weaned from the ventilator. Thereafter, respiratory compliance usually improves dramatically. Body temperature needs to be measured and maintained. Analgesia is provided with intravenous fentanyl or an epidural infusion of local anesthetic. Paracetamol (10 mg/kg) 6 hourly IV or 20 mg/kg PR should also be administered. All of these patients must be carefully monitored for respiratory complications in intensive care unit. Inferior vena caval compression, evident as "blue" lower limbs or bowel ischemia

(necrotizing enterocolitis) can occur as a result of increased abdominal pressure and may require surgical treatment. These infants are also at high risk for developing sepsis in the postoperative period.

The onset of peristalsis after repair of omphalocele or gastroschisis is usually delayed, and the resulting ileus may be prolonged, so that TPN is generally required for days to weeks in the postoperative period until full feeds are established and plays a vital role in the rapid recovery of these children. TPN is associated with the development of cholestasis, cirrhosis, portal hypertension, and ultimate liver failure.[6] Despite adequate volume replacement, hypotension may persist, and dopamine infusion is often needed to maintain a normal BP and urine output (if infant is severely compromised perioperatively). Early complications include: necrotising enterocolitis, renal insufficiency, pneumonia, patent ductus arteriosus, cellulitis of the abdominal wall, abdominal wall breakdown, gastroesophageal reflux and cholestasis.

Elective delayed midgut reduction can be done without anesthesia or sedation in the incubator in the NICU. In this, bowel reduction, which is pain free, can be undertaken conventionally with the same attention and with no greater difficulty than under general anesthesia. Delaying midgut reduction for >4 hrs, led to more stable cardiovascular, respiratory, and renal parameters. Moderate lower limb congestion seen, cleared rapidly.[14,15]

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

Introduction

Congenital hypertrophic pyloric stenosis (CHPS) is one of the most common causes of gastric outlet obstruction during early infancy, with an incidence of 1.4 to 8.8:1000 live births.[16] Though this presents in the first 6 months of life, it is most common between the ages of first 3 to 6 weeks life.[1] Treatment of this condition is by surgical mechanical distraction of the pyloric ring. Approximately 7% of pyloric stenosis infants have associated anomalies, like cleft palate and esophageal reflux with esophageal hiatal and inguinal hernias being the most frequent.[16]

Clinical Features

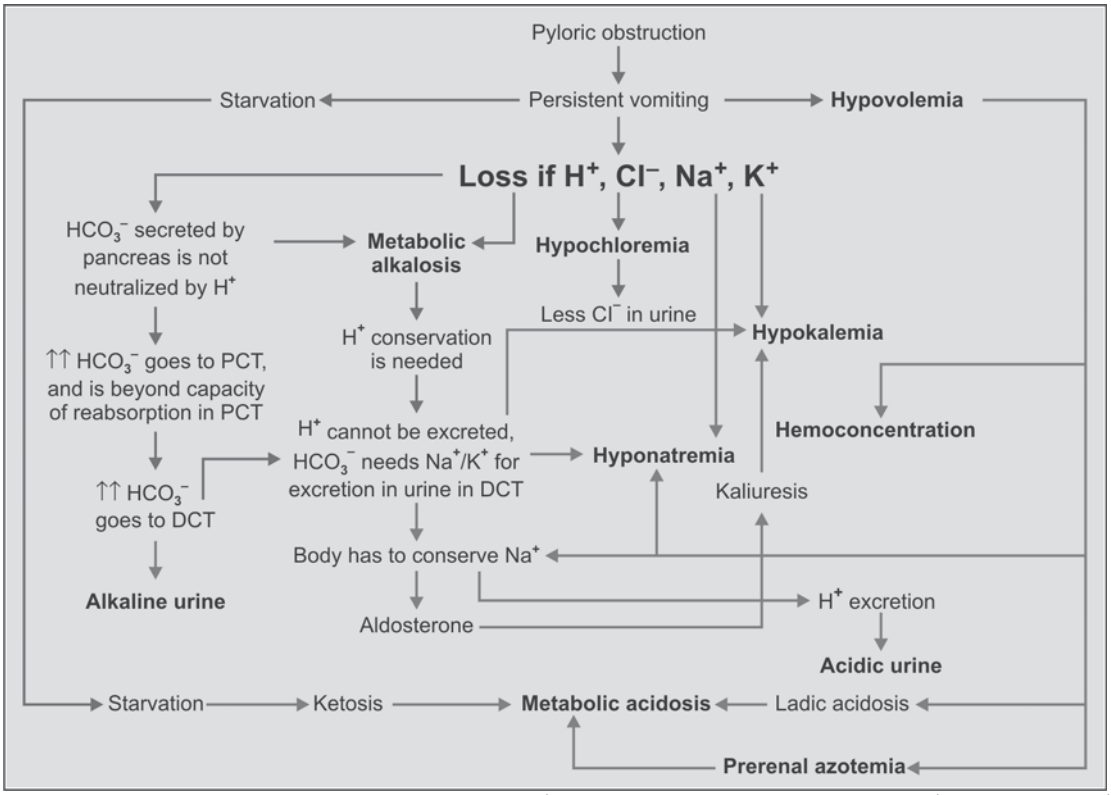
The cardinal features of pyloric stenosis condition are nonbilious (because the pyloric obstruction prevents entry of duodenal contents containing bile into the stomach) projectile vomiting, visible peristalsis, hypochloremic, hypokalemic metabolic alkalosis, with dehydration and lethargy. The vomitus may be brown or blood streaked and may not be frequent or projectile initially, but progressively becomes forceful and occurs within 30 to 60 minutes after feeding.[2] Body weight is reduced. There is decrease in frequency of stool or urine passage. Untreated, the child develops a hypokalemic, hypochloremic metabolic alkalosis. Jaundice (indirect hyperbilirubinemia) occurs in <5% of patients and is thought to be either associated with caloric deprivation and hepatic gluconyltransferase deficiency associated with starvation.[16] Histochemical studies indicate defective hepatic glucuronyl transferase activity. The condition is comparable to Gilbert syndrome. No specific treatment is necessary because the jaundice resolves spontaneously within 5 to 7 days after pyloromyotomy.

Pathophysiology

This has been explained in Flow chart 19.1.

The metabolic alkalosis can cause compensatory respiratory acidosis, shift of oxygen dissociation curve to left, hypocalcemia and convulsions. While some potassium is lost in the vomitus, its concentration is generally <15 mEq/l. Of greater importance is the potassium loss in the urine in exchange for hydrogen in an effort to maintain a normal serum pH. The potassium shift intracellularly with rising plasma pH, represent only a small portion of the plasma potassium (K^+) loss. Along with considerable hydrogen ion (H^+) loss from the gastric secretions, chloride (Cl^-) is also lost. The resulting hypochloremia results in maximal Cl^- conservation, with urinary Cl^- <20 mEq/l. The urine Cl^- concentration is usually similar to that of Na^+ in hypovolemic states since sodium (Na^+) and Cl^- are reabsorbed together. The finding of a low or absent urinary urine Na^+ concentration is virtually pathognomonic of reduced tissue perfusion and is diagnostic of hypovolemia.[17] However, an exception occurs when Na^+ is excreted with another anion. A patient who has vomited delivers more bicarbonate ions (HCO_3^-) to the distal tubule and collecting duct than can be reabsorbed. As some Na^+ (and K^+) loss is obligatory with the HCO_3^- delivered to the distal tubule and the collecting ducts, the urine contains Na^+ and K^+ which is not reabsorbed despite the contracted extracellular fluid volume (ECFV). Therefore, urinary Na^+ cannot be relied on as an indirect measure of volume status. In contrast, since all Cl is reabsorbed in exchange for HCO_3^-, the urinary Cl^- concentration provides a much more accurate estimate of the volume status in pyloric stenosis. It should be measured when an apparently hypovolemic patient has high urinary Na^+ concentration. The urine will not contain Cl^- when the

Flow chart 19.1: Pathophysiology of congenital hypertrophic pyloric stenosis



DCT: distal convoluted tubule, PCT: proximal convoluted tubule, H^+: hydrogen ions, Cl^-: chloride ions, Na^+: sodium ions, K^+: potassium ions, HCO_3: bicarbonate ions

ECFV is contracted and severe metabolic alkalosis is present.[18] Patients with ECFV contraction and Cl^- and K^+ depletion respond to Na^+, K^+, and Cl^- replacement. A urine Cl^- concentration >20 mEq/l, suggests volume status has been corrected. Urine Cl^- results are more relevant than serum electrolytes when assessing volume status in infants awaiting a pyloromyotomy.[18-20]

Pyloromyotomy, either open or laparoscopic, is the definitive and curative treatment for pyloric stenosis.[2] Fluid deficit and degree of dehydration is calculated as described in chapter 17. Before surgery, it is important that the infant is adequately hydrated with IV fluids to establish a normal urine output.

The following are major concerns for the anesthesiologist: (1) a full stomach, occasionally filled with contrast material can cause pulmonary aspiration; (2) metabolic alkalosis with hypochloremia and hypokalemia; and (3) severe dehydration.[1]

Preoperative Management

Once the patient is admitted, a large bore multiholed NGT is placed for aspiration of stomach contents. Pediatricians are now adapting at the early diagnosis with ultrasound so it is rare to find an infant with severe fluid and electrolyte derangements. However, an infant is occasionally seen whose problem has developed over a week leading to severe derangement. Severely dehydrated infants should receive an initial 20 ml/kg bolus of isotonic saline to re-expand the intravascular volume. Further resuscitation is given as 5% dextrose in 0.45% normal saline at 1.5 times the maintenance rate, i.e. 6 ml/kg/hr to prevent rapid changes in volume and electrolyte to avoid seizures and other complications.[16] Potassium chloride (KCl) 10 to 40 mEq/l can be added to the fluids if necessary when adequate urine output is demonstrated. Fluid resuscitation should be guided by serial measurement of serum electrolyte concentrations, which is essential for estimating the degree of dehydration, alkalosis, and electrolyte derangements. Pyloric stenosis is a medical emergency and should not be converted into a surgical nightmare by premature surgical repair before adequate resuscitation.

Let us see an example for fluid and electrolyte correction as follows:

A diagnosed case of pyloric stenosis, neonate of 2.5 kg with serum Na^+ = 125 mEq/l, K^+ = 2.5 mEq/l, Cl^- =

86 mEq/l, pH = 7.54 and mild dehydration. Fluid and electrolytes requirement of this baby are shown in Table 19.2. This deficit can be sufficed by administering 375 ml of 5 percent dextrose with 0.45 percent saline plus 3.2 ml (= 6.4 mEq) of KCl over 24 hrs.

Metabolic alkalosis must be corrected prior to surgery.[2] The child is ready for surgery between 24 and 48 hrs after commencing fluid resuscitation. Surgery should be delayed until biochemical values reach: pH 7.3 to 7.5, serum HCO_3^- <28 to 30 mEq/l, Na^+ >132 mEq/l, K^+ >3.5 mEq/l, Cl^- >90 mEq/l and urinary Cl^- >20 mEq/l. If deficits are not corrected as above, compensatory respiratory acidosis coupled with residual anesthetics may affect recovery from GA and increase the incidence of postoperative apnea.[20-25]

Table 19.2: Fluid and electrolytes requirement of CHPS baby

	Deficit	*Maintenance*	*Total*	*Actual*
Fluid	50 ml/kg	100 ml/kg/day	125 + 250	375 ml
Na^+	(140-125) × 0.6 × 2.5	3 mEq/kg/day	22.5 + 7.5	30 mEq
K^+	(4.5-2.5) × 0.3 × 2.5	2 mEq/kg/day	1.5 + 5	6.5 mEq
Glucose		4-6 mg/kg/min		18 gms

Anesthetic Management

Premedication: Once the child is satisfactorily hydrated, stomach contents thoroughly evacuated; appropriate monitors (electrocardiogram, pulse oximeter, blood pressure cuff, and precordial stethoscope) are placed. In theater, after giving atropine, NGT replaced with a fresh orogastric tube and the stomach is sucked out in the supine and both the right and left lateral positions immediately before induction of anesthesia and as much of the stomach contents are removed as possible.[26] This suctioning technique will remove 98% of the gastric contents.[27] This decreases the possibility of aspirating gastric contents during induction of anesthesia.[16] Though stomach washes with warm saline are advised until the aspirated fluid is clear, it is not practiced by us.

Induction: Inhalation induction is not advised.[28] Cook-Sather and others[28] compared three techniques: awake intubation, rapid sequence intubation, and modified rapid sequence intubation (ventilation through cricoid pressure). Awake intubation was not superior to anesthetized, paralyzed intubations. Awake intubation prevented neither bradycardia nor oxygen desaturations.[2,16] We do rapid sequence IV induction with cricoid pressure. After preoxygenation for 10 minutes, thiopentone sodium and succinylcholine are injected. Cricoid pressure is given. As babies are usually low weight, they do not tolerate complete apnea for 60 seconds. We do give gentle puffs with 100% oxygen.

Maintenance: Intraoperatively, we give lactated ringer's solution with 5% dextrose and continue the same in the postoperative period. Postoperative hypoglycemia has been reported. After endotracheal intubation, one dose of muscle relaxant is given which is usually sufficient for the procedure, as it last shorter. So usually, we maintain anesthesia with sevoflurane. Desflurane possesses useful characteristics for recovery conditions in these infants who are prone to apnea and ventilatory depression.[29] In case of nonavailability, isoflurane is another option. Avoid intraoperative hyperventilation as it can cause cerebrospinal fluid alkalosis leading to lethargy, drowsiness and respiratory depression in the postoperative period. Opioids are avoided. These patients have high incidence of postoperative apnea and opioids can aggravate the same. If at all, opioids are to be used, remifentanil is the drug of choice.[24,30,31] Majority of time, local bupivacaine (maximum dose, 1 ml/kg of 0.25%) infiltration with rectal paracetamol provides sufficient analgesia.[32] After intubation of the trachea, an orogastric tube is repositioned and left in place during surgery so that air can be insufflated in the stomach, to test for mucosal perforation after the hypertrophied muscle is split.[2] A small volume of air is injected down the NGT, and the surgeon manipulates the air bubble into the duodenum and occludes the bowel lumen both proximal and distal to the incision. Mucosal perforation is indicated if there is air leakage. After the operation, which usually requires less than 30 minutes, the effects of any nondepolarizing muscle relaxant are reversed.[16]

Emergence and extubation: Infants should be fully awake with intact protective airway reflexes, vigorous, with a stable general condition, adequate and regular breathing pattern before tracheal extubation.

Postoperative Management

Incidence of postoperative apnea is higher.[24,29,31] Hence, apnea monitoring along with pulse oximetry is indicated for the first 12 hours after surgery irrespective of the drugs used.

After the operation, the gastric tube is removed as soon as the infant awakens from the anesthetic. For infants in whom the duodenal mucosa is perforated, the gastric tube may be left for an additional 24 hours to ensure gastric decompression. Parenteral fluids are administered to maintain adequate hydration and urine output until oral intake is sufficient. Oral feedings usually are initiated 4 hours after surgery, provided that the infant is alert and has a good sucking reflex.

NECROTIZING ENTEROCOLITIS

Introduction

Necrotizing enterocolitis (NEC) is the most common neonatal gastrointestinal (GI) surgical emergency, resulting in substantial perinatal morbidity and mortality. It is a life-threatening intestinal inflammation.[16] It most frequently involves the terminal ileum and proximal colon.[2] Although centered in the GIT, it is a systemic process primarily related to the sepsis.

Classically, it occurs in small premature (80-90%) and low birth weight (LBW) infants with gestational age <32 weeks and weight <1500 gm. However, NEC can also occur in full-term infants on the first day of life or months after birth. It can occur in fed or unfed infants, in a single patient, or as a nursery epidemic. Of note, term infants develop symptoms by day 2 to 3 of life, whereas the extremely low birth weight (ELBW) infants are more likely to develop NEC in the second week of life.[16]

Incidence

The incidence, severity of the symptoms, complications, and mortality are inversely related to gestational age and birth weight.[2] Survivors often have significant long term nutritional and developmental problems.[1] The incidence of NEC is 0.3 to 2.4 cases per 1000 live births (10 to 20%, in infants <1500 gm) which has not decreased and overall mortality has not improved over the past two to three decades. As the survival of the premature, especially the ELBW, infant has improved, the incidence of NEC has increased in some populations. The increased number of susceptible infants may explain the lack of improved overall survival in infants with NEC.[16] The mortality rates for term versus preterm infants are 4.7 and 11.9% respectively.[33,34]

Etiology and Pathophysiology

It is multifactorial; ischemia and/or reperfusion injury, exacerbated by activation of proinflammatory intracellular cascades, may play a significant role. This has been explained in Flow chart 19.2.[2,16,35-39]

NEC is frequently discontinuous, with patchy occurrence in both the small and large intestine (50%) (Fig. 19.4). Perforations often are multiple. The inflammatory response seems to be unique in that abscesses do not form, as seen in inflammatory bowel disease or infectious colitis or as a result of acute arterial occlusion. The combination of ischemia and bacteria seems to be essential. NEC does not evolve after an episode of vascular injury *in utero*, when the bowel is sterile. Instead, an intestinal atresia or stenosis may develop. However, NEC is not associated with a specific organism or with particularly virulent bacteria. A wide range of organisms have been identified.[16]

Stages

Bell and others[16] have described three stages of NEC as shown in Table 19.3.

Although these three stages are not distinct and are actually a continuum of clinical disease, the three-stage concept helps to define management strategies.

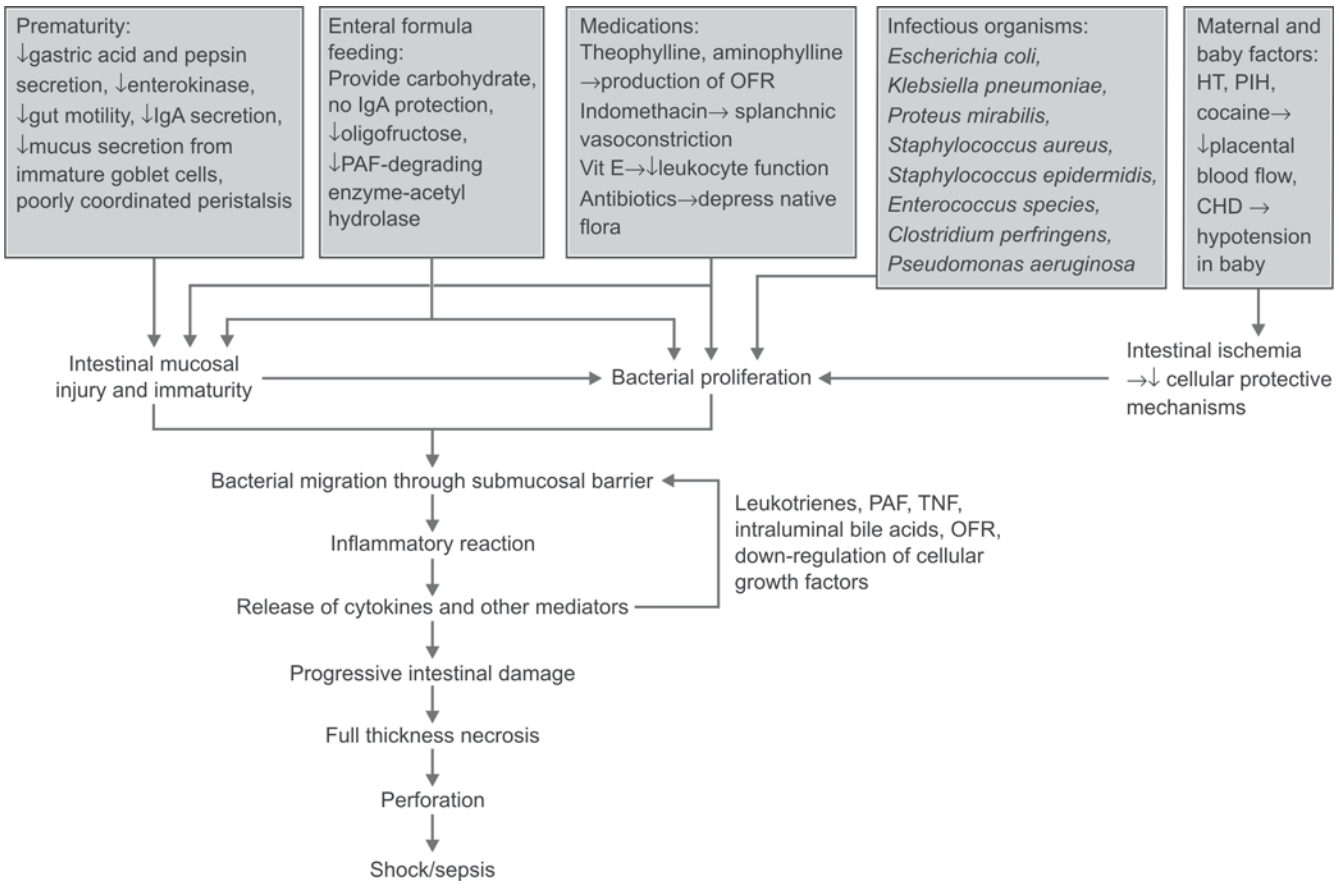
Clinical Features

Early clinical findings include abdominal distension and tenderness recurrent apnea, lethargy, temperature and glucose instability. More specific signs are abdominal distension, abdominal wall erythema, high gastric residuals after feeding (may be bile-tinged), feeding intolerance leading to vomiting and bloody or mucoid diarrhea. The GI signs appear between the 1st and 10th days of life in more than 90% of these babies. Bowel necrosis and perforation follow, and sepsis occurs along with hyponatremia, jaundice, decreased peripheral perfusion, hemodynamic instability, anemia, thrombocytopenia (50,000 to 75,000/mm^3), coagulopathy, disseminated intravascular coagulation, prerenal azotemia and metabolic acidosis secondary to generalized peritonitis and hypovolemic shock. There is bleeding diathesis with prolonged prothrombin and partial thromboplastin time, decreased fibrinogen, and rise in fibrin split products. There is leukopenia or leukocytosis.[2] Respiratory distress syndrome (RDS) is present in severe cases.

Diagnosis[40,41]

- An abdominal anteroposterior (AP) and a left lateral decubitus radiograph shows abnormal gas pattern, dilated loops, and thickened bowel walls (suggesting edema/inflammation). A fixed and dilated loop and sometimes scarce or absent intestinal gas, are more worrisome than diffuse distension that changes over time. Classic pnuematosis intestinalis[1] is seen as characteristic train-track lucency configuration within the bowel wall (Fig. 19.5). The free air can be difficult to differentiate from intraluminal air.
- Pneumoperitoneum (abdominal free air) is ominous and indicates intestinal perforation.[2] Withdrawing intestinal fluid by a needle from the abdomen indicates the intestinal perforation. Left-side down decubitus radiography is essential for detection of intraperitoneal air, which rises above the liver shadow (right-side up) and can be visualized easier than on other views.

Flow chart 19.2: Pathophysiology of necrotizing enterocolitis



IgA: immunoglobulin A, PAF: platelet activating factor, OFR: oxygen free radicles, HT: hypertension, PIH: pregnancy induced hypertension, CHD: congenital heart disease, TNF: tumor necrosis factor

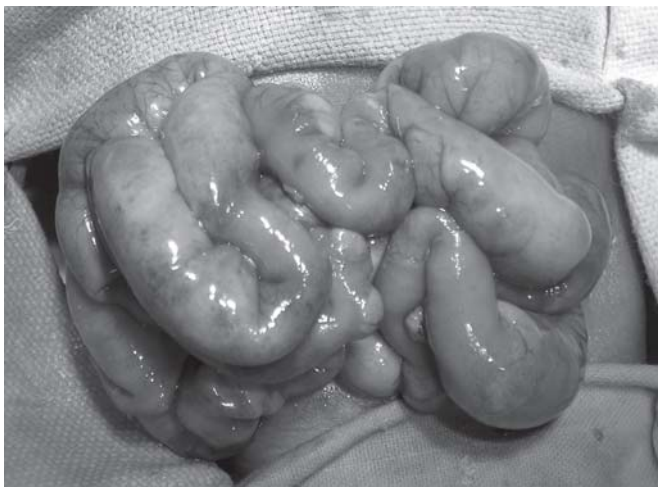


Fig. 19.4: Necrotizing enterocolitis. Note the multiple necrotic and gangrenous areas *(For color version, see Plate 2)*

- Portal gas appears as linear branching areas of decreased density over the liver shadow and represents air present in the portal venous system which is a poor prognostic sign. It is much more dramatically observed on ultrasonography as bubbles present in the venous system.
- Ascites is a late finding that indicates either peritonitis or bowel perforation and is observed on an AP radiograph as centralized bowel loops that appear to be floating on a background of density. It is better appreciated on ultrasonography.
- Doppler study of the splanchnic arteries and GI tonometry early in the course of necrotizing enterocolitis can help to distinguish developing NEC from benign feeding intolerance in a mildly symptomatic baby. Markedly increased peak flow velocity (>1) of arterial blood flow in the celiac and superior mesenteric arteries is seen in early necrotizing enterocolitis.[42]

Table 19.3: Staging of NEC

Stages	*Disease*	*Symptoms*	*Radiologic evidence*	*State of the bowel*
I	Mild	Nonspecific (vomiting, gastric residuals, apnea, bradycardia, guaiac-positive stools)	No definitive	Completely unknown
II	Definitive	Similar to the infants in stage I	Pneumatosis intestinalis or portal venous air[39]	Suitable for medical management
III	Advanced	Intestinal necrosis and/or perforation along with clinical signs of hemodynamic, respiratory, and/or hematologic instability	Pneumoperitoneum, gas under diaphragm or air-fluid levels indicates intestinal perforation	Surgical treatment is advised

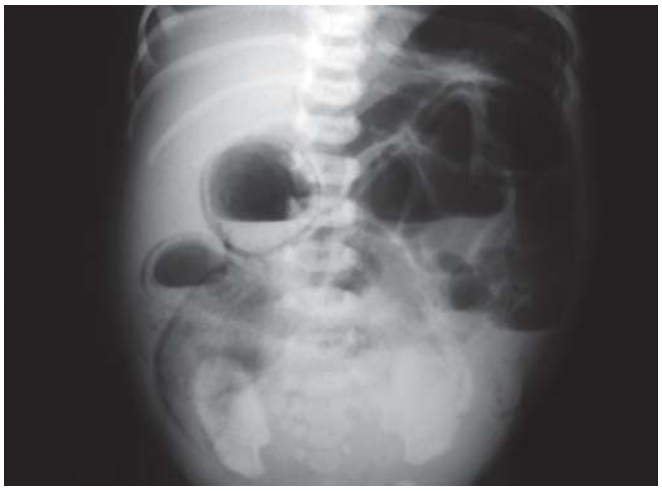


Fig. 19.5: X-ray abdomen in necrotizing enterocolitis. Note the pneumatosis intestinalis

- Other contemporary imaging techniques includes: contrast radiography, portal vein ultrasonography, MRI, and radionuclide scanning.

Treatment

Unless there is evidence of intestinal necrosis or perforation, the initial treatment for NEC is nonoperative. Medical treatment, consisting of, no enteral feedings for 10-14 days, intermittent suction of NGT for gastric decompression, fluid and electrolyte therapy, including parenteral nutrition, correction of hematologic abnormalities and broad-spectrum antibiotics, is often successful in the management. Mechanical ventilation is indicated if abdominal distension is contributing to hypoxia and hypercapnia. Hypotension is treated with crystalloid and blood products. Supportive therapy, including inotropic agents and steroids, may be used to treat endotoxic shock[3] and to improve cardiac output and bowel perfusion. Umbilical artery catheters should be removed, if present, to avoid compromise of mesenteric blood flow.[2]

Indications for Operation[3]

Absolute Indications include Pneumoperitoneum following bowel perforation and intestinal gangrene (positive results of paracentesis).

Relative Indications are clinical deterioration, metabolic acidosis, ventilatory failure, oliguria, hypovolemia, leukopenia, leukocytosis, thrombocytopenia, portal vein gas, erythema of abdominal wall, fixed abdominal mass, persistently dilated loop, peritonitis, bowel wall edema, ascites, and a progressively deteriorating cardiorespiratory status indicating bowel necrosis.[3]

While severe gastrointestinal hemorrhage, abdominal tenderness, intestinal obstruction, gasless abdomen with ascites are nonindications.

The effectiveness of peritoneal lavage in "buying time" for an infant to achieve hemodynamic, acid-base, and hematologic stability is actively debated. This procedure seems to be "palliative" because most infants eventually require surgery. The ELBW infant may benefit from such a temporizing procedure that would allow resuscitation and stabilization. If surgery is performed after the hemodynamic status improves, adequate gut perfusion to marginal segments of bowel might be established and, consequently, less bowel is resected. This might avoid excessive bowel resection leading to short gut syndrome, [16] complications related to central venous catheters for TPN, and cholestatic jaundice.[2]

Preoperative Assessment

A typical infant with NEC has perinatal asphyxia or other respiratory complications in the early postnatal period. Prenatal complications include premature rupture of the membranes, placenta previa, maternal sepsis, and toxemia of pregnancy. A breech delivery or cesarean section is associated with 15 to 20% of cases. Infants may be acidotic, hypoxic, hypothermic, and in shock. Thus anesthesiologist is confronted with the problems of prematurity, hypovolemia, sepsis, acidosis,

and hepatic portal gas. The preoperative assessment should focus on evaluating and correcting the respiratory, circulatory, metabolic, and hematologic disorders. One anomaly mandates a search for others. Murmurs necessitate a cardiology consult.[43]

Laboratory Testing Includes[41]

1. CBC count, with manual differential to evaluate the WBC, hematocrit, and platelet count, is usually repeated at least every 6 hours if the patient's clinical status continues to deteriorate.
   - WBC count: Marked elevation or leukopenia with left shift. Moderate-to-profound neutropenia (absolute neutrophil count <1500/µl) strongly suggests established sepsis.
   - RBC count: Anemia due to prematurity, iatrogenic blood draws, hematochezia and/or a developing consumptive coagulopathy.
   - Platelet count and coagulation study: Thrombocytopenia (<100,000/µl). Neutropenia and thrombocytopenia are associated with gram-negative sepsis and platelet binding by endotoxin. It is profound in severe cases that become complicated with consumption coagulopathy. Consumption coagulopathy is characterized by prolonged PT, prolonged aPTT, and decreasing fibrinogen and increasing fibrin degradation products concentrations.
   - Blood culture: Obtaining a blood culture is recommended before beginning antibiotics.
2. Serum electrolytes (Na^+, K^+, and Cl^-) is done serially at least every 6 hours depending on the acuity of the patient's condition.
   - Serum sodium: Hyponatremia is a worrisome sign that is consistent with capillary leak and "third spacing" of fluid within the bowel and peritoneal space. Depending on the baby's age and feeding regimen, baseline sodium levels may be low-normal or subnormal, but an acute decrease (<130 mEq/l) is alarming and heightened vigilance is warranted.
   - Metabolic acidosis: Low serum bicarbonate (<20 mEq/l) in a baby with a previously normal acid base status is also concerning. It is seen in conjunction with poor tissue perfusion, sepsis, and bowel necrosis.
3. Arterial blood gasses
   - Depending on presentation acuity, hypoventilation and frank apnea, ABG can aid in the determination of the infant's need for respiratory support in addition to acid-base status.
   - Acute acidosis is worrisome. Lactic acidosis results from decreased cardiac output (as in cardiovascular collapse and shock), leading to poor perfusion of peripheral tissues. Tissue necrosis may also add to the observed metabolic acidosis.

An arterial blood sample is a convenient way to simultaneously obtain a blood culture, CBC count, serum electrolytes, and ABG for the initial evaluation. Depending on presentation acuity, inserting a peripheral arterial line while peripheral perfusion and intravascular volume is still within the reference range may be prudent. This peripheral arterial line facilitates serial blood sampling and invasive blood pressure monitoring that is essential if the baby's condition deteriorates.

Preoperative respiratory status must be carefully evaluated. Profound hypovolemia from peritonitis, sepsis and massive third-space fluid losses require vigorous fluid resuscitation with crystalloid and colloid solutions before induction of anesthesia. Intraoperative fluid resuscitation is critical. Blood and platelet transfusions are often necessary. Perioperative fluid administration can approach 150 to 200 ml/kg. Because of varying degrees of hypovolemia it is not possible to recommend rigid guidelines for fluid replacement during operation. Infusion rates for crystalloid solutions (5% dextrose in lactated Ringer's solution) approach 8 to 10 ml/kg/hr.[43] Inotropes such as dopamine may be required to maintain adequate cardiac output and bowel perfusion. Adequate monitoring of fluid resuscitation is done by catheter placed in a peripheral artery to measure systemic BP, ABG (pH), hematocrit, and electrolytes continuously. Rapid fluid administration to preterm neonates may cause intracranial hemorrhage or reopening of the ductus arteriosus with congestive heart failure.[2,16] Thrombocytopenia and the associated coagulopathy may require blood and platelet transfusions. The normal platelet count for the preterm infant is 280 $x10^9$ cells/l which is similar to that of the normal adult. Normal bleeding times are maintained in the adult when the platelet count is above 100 x 10^9 cells/l. Although preterm infants usually have a platelet count above this level they do have a propensity for bleeding which may be secondary to vessel fragility or reduced platelet function. For transfusion with platelet concentrates the following formula is used to calculate the expected increase in platelet count:

$$\text{Expected increase (cells/l)} = 2\ (0.7 \times 10^{11} \times N)/\text{Blood volume}$$

Where the blood volume is given in liters and N is the number of units of platelet concentrate administered.[44] Using 85 ml/kg as the blood volume for

newborns this formula suggests that the administration of 0.18 unit/kg would increase the platelet count by 100 x 10^9 cells/l. The newborn has low levels of coagulation factors II, VI, IX, X, XI, and XII. Factors II, VII, IX, and X are all vitamin K dependent factors. Therefore, it is recommended that vitamin K be administered routinely to all newborns. In the event that coagulation needs to be restored to normal immediately, fresh plasma, fresh-frozen plasma, or prothrombin complex can be administered. The administration of 10 ml/kg of fresh frozen plasma or platelet concentrate will supply the minimum hemostatic factors for most newborn hemostatic problems.

Management of Anesthesia

Monitoring: Routine monitoring during neonatal anesthesia includes continuous electrocardiogram, core temperature by esophageal or nasopharyngeal probe, esophageal or precordial stethoscope, BP, serial ABGs, CVP and urine output.

Because of the large fluid requirements and rapid heat loss, hypothermia is a frequent. Normothermia is achieved by administration of warmed blood and IV solutions, humidified anesthetic gases. Later reduces heat loss and minimizes the risk of tracheal tube occlusion from inspissated secretions.[45] Temperature controlled blankets and overhead radiant warmers keep the operating room warm.[2]

BP monitoring is done with an indwelling arterial catheter. Intraoperative fluid therapy is guided by acid base status and CVP. Thus central venous cannulation is also helpful. Patient needs to be catheterized.

Induction: These infants are usually on mechanical ventilation prior to surgery. If not already intubated on arrival in the operating room, full stomach precautions are in order. Preoxygenation and premedication with atropine should be given prior to induction and laryngoscopy. Following rapid sequence induction an endotracheal tube should be chosen to allow ventilation with PIPs >20 cm H_2O, due to high intra-abdominal pressures and decreased pulmonary compliance.

Prematurity has its own problems like retinopathy (retrolental fibroplasia) and hyaline membrane disease requiring varying amounts of supplemental oxygen and mechanical ventilation. High oxygen tension in the retinal vessels interferes with normal vascularization of the developing retina. Newly formed capillaries are destroyed and abnormal vessels are formed. Though severe cases of retinopathy can ultimately progress to retinal scaring and blindness, 80 to 90% of them show regression.[46] To minimize the risk of retinopathy arterial oxygen tensions need to be maintained at 50 to 70 mm Hg intraoperatively. Hypoxia is a greater danger than retinopathy and must be avoided. Ventilatory requirement often increases during surgery as handing of bowel reduces lung compliance.[47] Intraoperative ventilatory settings should be oriented to the maintenance of preoperative oxygen tensions. The use of a continuous inline oxygen monitor and an air flowmeter on the anesthetic machine will permit the fine control of inspired oxygen concentrations. Continuous direct intra-arterial monitoring of oxygen tensions and pulse oximetry (SpO_2=90-95%) will allow better control of arterial oxygen tensions.[16] It is advisable not to use nitrous oxide due to expansion of intramural and portal gas pockets.[48] Mural gas is composed of hydrogen, nitrogen, carbon dioxide, and traces of oxygen. Due to the greater blood solubility of nitrous oxide (0.47) relative to nitrogen (0.0122) and hydrogen (0.0149) the size of bubbles could increase. This expansion could further compromise bowel blood supply. An additional hazard of mesenteric vein gas is the risk of gas embolism from the passage of gas through the ductus venosus into the inferior vena cava. The ductus venosus may be patent for two to three weeks after birth in term infants.

Maintenance: Volatile anesthetics can produce significant myocardial depression and hypotension particularly in presence of sepsis and hypovolemia. Therefore, decreased maintenance doses of anesthetics are needed. Fentanyl, remifentanil or ketamine combined with low-dose, inhaled anesthetics can provide analgesia and amnesia as well as cardiovascular stability. Neuromuscular blocking agents facilitate surgical exposure. Inotropic agents occasionally are needed to support the cardiovascular system when fluid therapy alone fails to maintain adequate perfusion.[16]

Postoperative Management

Postoperatively, mechanical ventilation and cardiovascular support are usually continued because of abdominal distension and coexisting RDS.[2] TPN is essential even after sepsis is controlled and metabolic stability is established.[16]

INTESTINAL OBSTRUCTION

Intestinal obstruction is one of the most common surgical emergencies encountered in the neonatal period and is characterized by feeding intolerance, vomiting, and abdominal distension. Common causes of intestinal obstruction in neonatal period are duodenal, jejunal or ileal atresia, Meckels diverticulum, meconium ileus, necrotising enterocolitis, malrotation

with volvulus, Hirschprungs disease, and anorectal malformation intussusception, round worm infestation, adhesions, bands, etc. usually present in infancy or childhood. Irrespective of type of obstruction majority have fluid, electrolyte imbalance and abdominal distension (Fig. 19.6), which reduces respiratory compliance and venous return. They have to be treated like full stomach.

Atresia

Overall incidence of small intestinal atresia is 1.3 cases per 10, 000 live birth.[49] Neonates with intestinal atresia are frequently preterm or may have other associated anomalies such as malrotation of the gut, volvulus, and abdominal wall defects.[4] Healthy newborn infants have gastric aspirates that measure less than 5 ml. Congenital intestinal obstruction is associated with gastric aspirates that measure greater than 30 ml.[50,51] Sustained vomiting (bilious or nonbilious) is the most common symptom, occurring in approximately 85% of cases. [49]

Duodenal Atresia

Incidence of duodenal atresia is 1 case per 5, 000-10, 000 live births.[49] Duodenal atresia typically presents within the first hour of life. Associated anomalies are extremely common: Down syndrome (30%), isolated cardiac defects such as atrial septal defect, ventricular septal defect, or atrioventricular canal (23-34%), esophageal atresia (7-12%), and polyhydramnios (32-59%).[49] Duodenal atresia with associated cardiac anomalies warrants preoperative cardiology evaluation and echocar-diography.[49] Coexisting tracheoesophageal fistula is repaired before correction of duodenal atresia.

Neonates with duodenal atresia present so early in life, usually within the first 12 hours, that they rarely are dehydrated or hypochloremic. Nonbilious vomiting occurs when atresia is present above the papilla of Vater. Vomiting is associated with variable dehydration, changes in serum electrolytes, and weight loss. An abdominal X-ray film will show the classic "double bubble" sign (i.e. dilated air-filled stomach and proximal duodenum) (Fig. 19.7).

Jejunoileal Atresia

Site of atresia is proximal jejunum (31%), distal jejunum (20%), proximal ileum (13%), and distal ileum (36%).[52] It can be single (>90%) or multiple (6-20%).[53] With jejunoileal atresia, air-fluid levels are observed throughout the abdomen. With distal ileal obstruction, a barium enema may demonstrate a microcolon. Patients are frequently premature (35%).[53] Majority are low birth

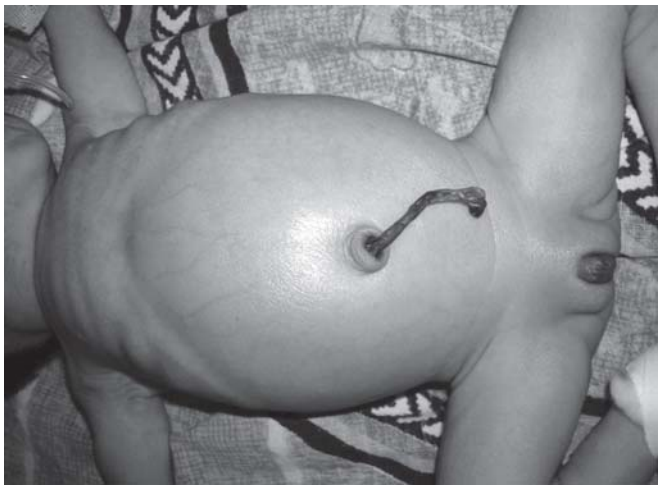


Fig. 19.6: Neonate with massive distention of abdomen in case of ileal atresia *(For color version, see Plate 2)*

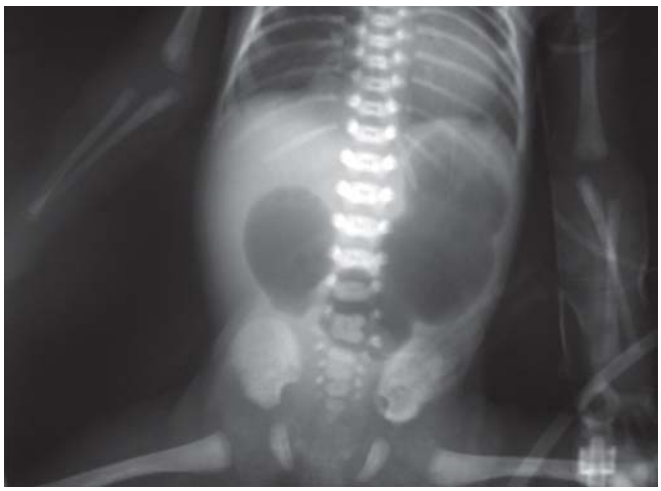


Fig. 19.7: X-ray abdomen in duodenal atresia. Note the double bubble sign

weight: jejunal (33%), ileal (25%), multiple (>50%). Deaths are attributed to associated malformations, respiratory complications, prematurity, and anastomotic complications.[53,54]

Investigations

- CBC, Electrolytes, Crossmatch of blood.
- Chest X-ray for all infants with cardiorespiratory symptoms.
- Echocardiography to exclude endocardial cushion defects and patent ductus arteriosus (PDA).
- Ultrasound, useful in detecting renal anomalies and an annular pancreas.
- Head ultrasound to exclude intracranial hemorrhage in premature infants.

- Esophagogastroduodenoscopy may reveal esophageal irritation, reflux gastritis, megaduodenum, delayed emptying.

Preoperative Management

Vomiting leads to dehydration (elevated hematocrit) and hypoglycemia (especially in premature). Parenteral nutrition is instituted on the first day of life via a peripherally inserted central catheter. Neonates tolerate surgical procedures best when they are metabolically and hemodynamically stable. Attention should be directed to prevent or correct hypothermia, hypovolemia, hypoglycemia, and hypoxemia. The patient is admitted to the neonatal intensive care unit and restricted to nothing by mouth (NPO). The baby should be kept in a warm environment (e.g. an incubator) with humidified air, and the oxygen saturation should be monitored. Vital signs should be frequently assessed. The airway is kept clear with frequent nasopharyngeal aspiration. Initial fluid requirement in these neonates is 2 to 4 times the maintenance requirements (8–16 ml/kg/hr) to ensure adequate hydration and to compensate for vomiting and large gastric aspirates. Hypovolemic shock, hemoconcentration, and metabolic acidosis may develop with inadequate resuscitation. Acid-base status and electrolyte levels should be monitored serially.[18] Dopamine infusions may be required to increase the cardiac output and improve intestinal perfusion. Broad-spectrum antibiotics and 1 mg vitamin K are administered.[49] Use of umbilical lines should be avoided because of the increased risk of infection, mesenteric blood flow compromise and because they become suboptimal for the transverse incision for laparotomy.[55-57]

A proximal small-bowel obstruction results in loss of fluids that resemble gastric juice and thus produces hypokalemic and hypochloremic metabolic alkalosis. With distal small bowel obstruction, fluid losses are usually isotonic, so serum electrolytes are normal until sufficient dehydration results in metabolic acidosis, as demonstrated by tachypnea, low serum bicarbonate levels, and elevated serum chloride values. Nasogastric tube losses are replaced by Isolyte G in proximal and Isolyte E in distal atresias.[58]

Abdomen is scaphoid in duodenal, diffusely distended in ileal, and the combination of two in jejunal atresia (upper distension, lower scaphoid). Intubation for respiratory support in patients with severe abdominal distension or sepsis may be necessary.

Bypass procedures for duodenal atresia or stenosis include duodenoduodenostomy or duodenojejunostomy.

Anesthetic Management

Anesthetic management proceeds with decompression of the stomach and preoxygenation. Avoid sedative premedication; only atropine is administered prior to induction. All the patients should be considered full stomach. Awake intubation is generally advocated in volume-depleted or actively vomiting infants. Rapid-sequence induction may be used with thiopentone (2-3 mg/kg) or sevoflurane in hemodynamically stable neonates with normal airway anatomy. Ventilation is done with air and oxygen avoiding nitrous oxide to minimize intestinal distension. Muscle relaxation with Suxamethonium or atracurium is generally necessary to facilitate abdominal exploration. Use of a neuromuscular blocking agent also decreases or avoids the need for large concentrations of potent inhalation agents, which are poorly tolerated by hypovolemic neonates.[7]

Intraoperative analgesia: Opioids if elective ventilation is planned.

Intraoperative fluid management: Nasogastric tube aspirates are replaced by Isolyte G in duodenal atresia and Isolyte E in jejunoileal atresia in the perioperative period. This is because Isolyte G has similar constituents as gastric juice. Further, ammonium releases H^+ ion. Similarly, Isolyte E has similar constituents as small intestinal juice. Further, acetate gets converted to bicarbonate (Tables 19.4 and 19.5). Lactated Ringer's solution (8 to 15 ml/kg, or more, per hour) is administered for surgical exposure.[58,59]

Postoperative care: The orogastric tube is left on free drainage. The patient is not given oral feedings until bowel sounds are heard, stool is passed, and the gastric drainage is limited (<1 ml/kg/h of clear or pale-green fluid) . This may take 7 to 10 days but can be prolonged in the premature infant with other significant anomalies that require venous access for parenteral nutrition.[49] Transfusion is administered if indicated. Glucose, hemoglobin, electrolytes, and bilirubin levels are frequently monitored during the first postoperative days, and adjustments are made accordingly. Phototherapy to avoid kernicterus is sometimes necessary.

MECONIUM ILEUS

Introduction

Meconium ileus (MI) accounts for 9 to 33% of neonatal intestinal obstructions and is the earliest clinical manifestation of cystic fibrosis.

Clinical Features

Patients with simple meconium ileus usually present with abdominal distension at birth, eventually progressing to failure to pass meconium, bilious vomiting, and progressive abdominal distension. Often, examination reveals dilated loops of bowel with a doughy character that indent on palpation. Patients with complicated meconium ileus present more dramatically at birth with severe abdominal distension, sometimes accompanied by abdominal wall erythema and edema. Abdominal distension may be severe enough to cause respiratory distress. Signs of peritonitis include tenderness, abdominal wall edema, distension, and clinical evidence of sepsis.[60] Cystic fibrosis is characterized by the triad of chronic obstruction and infection of the respiratory tract, exocrine pancreatic insufficiency, and elevated sweat chloride levels.

Diagnosis

Abdominal radiography reveals a characteristic pattern which includes the following:[61] (1) great disparity in the size of the intestinal loops because of the configuration of the different segments of the bowel; (2) no or few air fluid levels on the erect film because swallowed air cannot layer above the thickened inspissated meconium and (3) a granular, "soap bubble" or "ground glass" appearance seen frequently in the right half of the abdomen, a finding that requires swallowed air bubbles to intermix within the sticky meconium (Fig. 19.8). When meconium ileus is suspected based on clinical and radiographic evidence, a contrast barium enema may be performed with a therapeutic Gastrografin enema. Immunoreactive trypsinogen (IRT) is a pancreatic enzyme that can help with diagnosing cystic fibrosis (CF) in neonates with meconium ileus.[62]

Treatment

Manage both simple and complicated meconium ileus (MI) in newborns as an intestinal obstruction.[49,63] Perform resuscitative measures, including mechanical respiratory support, if necessary. Initiate intravenous hydration and gastric decompression. Evaluate and correct any coagulation disorders, and begin empiric antibiotic coverage. Immediate surgical evaluation is necessary when meconium ileus is suspected or diagnosed.[64] Surgical management is necessary in patients with persistent or worsening abdominal distension, persistent bowel obstruction, enlarging abdominal mass, intestinal atresia, volvulus, perforation, meconium cyst formation with peritonitis and bowel necrosis.

Management of Anesthesia

The Bishop-Koop enterostomy is the procedure of choice.[65,66] Intraoperative management involves fluid

Table 19.4: Composition of various body fluids

Body fluid	*Electrolytes (mEq/l)*			
	Na^+	K^+	Cl^-	HCO_3^-
Gastric	70	5-15	120	0
Pancreas	140	5	50-100	100
Bile	130	5	100	40
Ileostomy	130	15-20	120	25-30
Diarrhea	50	35	40	50

Table 19.5: Constituents of various IV fluids

	Fluid Therapy					
Composition	*Ringer's Lactate*	*Normal Saline*	*Isolyte-P*	*Isolyte-G*	*Isolyte-E*	*Isolyte-M*
Sodium	130 mEq	154 mEq	25 mEq	65 mEq	140 mEq	40 mEq
Potassium	4 mEq		20 mEq	17 mEq	10 mEq	35 mEq
Chloride	109 mEq	154 mEq	22 mEq	150 mEq	103 mEq	38 mEq
Calcium	3 mEq				5 mEq	
Lactate	28 mEq					
Ammonium				69 mEq		
Glucose			50 g	50 g	50 g	50 g
Magnesium					3 mEq	
Citrate			3 mEq		8 mEq	
Acetate			23 mEq		47 mEq	20 mEq
Phosphate			3 mEq			15 mEq

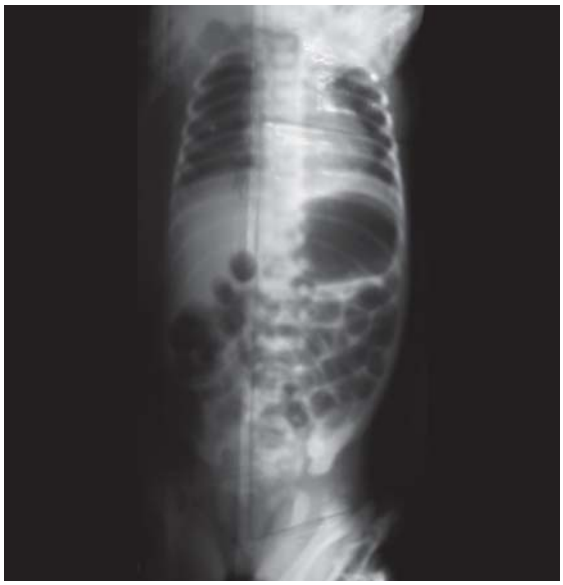


Fig. 19.8: X-ray abdomen in meconium ileus

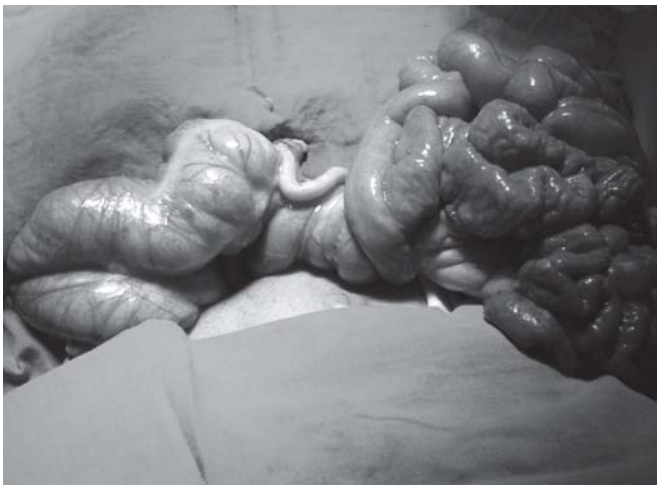


Fig. 19.9: Intraoperative picture of intestinal malrotation with volvulus *(For color version, see Plate 2)*

resuscitation, carefully replacing the fluid losses caused by surgery and by preoperative diuresis and diarrhea (if a Gastrografin enema is attempted). Adjust ongoing maintenance fluids and replace insensible fluids lost, as well as GI losses (i.e. losses from NG suction and ileostomy).[18] Instillation of *N*-acetylcysteine via an NG tube or via ileostomy helps solubilize residual meconium.[49] These infants may be posted for stoma closure within 6 weeks to avoid prolonged problems with fluid, electrolyte, and nutritional losses.

MALROTATION AND VOLVULUS

Introduction

Incidence is 1 in 500 live births.[67] Of those in whom volvulus occurs, 68-71% are neonates.[49] Most cases occur by age 2 months, 75% present before age 1 year, and the remaining 25% present after age 1 year. Malrotation is also associated with duodenal atresia, Meckel diverticulum, intussusception, small bowel atresia, prune belly syndrome, gastric volvulus, persistent cloaca, Hirschsprung disease, and extrahepatic biliary anomalies.[67] In contrast to midgut volvulus, sigmoid volvulus is usually the result of a dilated rectosigmoid colon on a narrow pedicle.

Pathophysiology

Rotation of the intestine around the mesentery may produce the abnormal location of the ileocecal valve in the right upper quadrant and kinking or compression of its vascular supply (Fig. 19.9). If the malrotation occurs during development, atretic segments of bowel are formed. If the kinking or compression occurs after the bowel is normally developed, bowel necrosis results. Intestinal ischemia progresses to gangrene and bowel distension from gas-producing organisms within the intestine occurs.

Clinical Features

In the first month of life, the most typical presentation includes feeding intolerance or bilious vomiting and sudden onset of abdominal pain, increasing abdominal girth; bloody stools are an ominous sign. Bilious vomiting is the hallmark presentation and is observed 77 to 100% of the time. Sigmoid volvulus typically presents with abdominal pain, distension, and inability to pass stool or flatus (obstipation). Vomiting may be a late presenting feature, and cases may progress to peritonitis, sepsis, and death. Signs of intraluminal blood loss, such as hematochezia or stool guaiac testing, are usually positive.[49,67]

Diagnosis

- Plain radiography shows evidence of small bowel obstruction, including dilated small-bowel loops; marked gastric or proximal duodenal dilatation, with or without intestinal gas and air-fluid levels.
- Upper GI imaging shows corkscrew tapering of contrast signifying proximal intestinal obstruction.
- Lower GI imaging reveals dilated rectosigmoid loops with an abrupt inability to pass contrast beyond obstruction in patients with sigmoid volvulus.
- Ultrasonography may reveal intraluminal fluid or edema in the bowel wall.

- CT scanning may reveal The "whirl sign" (clockwise or counterclockwise twisting of the bowel that extends for at least 180°).

Management of Anesthesia

Preoperative assessment: These infants may have hypotension, hypovolemia, and electrolyte abnormalities. Because delay in surgery may result in necrosis of the entire small intestine, fluid and electrolyte resuscitation begins preoperatively and continues during surgery. This is a true neonatal emergency and surgery should proceed as reasonably fast as possible.[49] Blood and blood products should be available in the operating room. Hematocrit may be falsely increased secondary to marked intravascular volume depletion. The indications for intra-arterial monitoring depend on the severity of the infant's illness. Central venous pressure monitoring may improve assessment of intravascular volume status, and fluid management; however, if there is adequate peripheral venous access, the operation should not be delayed to insert the central venous pressure catheter.[49] CBC count, clotting studies, electrolyte levels, and blood glucose levels are usually sufficient for preoperative evaluation. Nasogastric decompression may be successful in alleviating vomiting and discomfort associated with obstruction. Rectal tube decompression of the sigmoid volvulus can be achieved. This may be aided by endoscopic placement.

Intraoperative management: During operation, the midgut volvulus is reduced by untwisting the bowel in a counterclockwise fashion. Viability of the small bowel loops can then be assessed.

Postoperative management: Postoperatively, patients still require aggressive fluid resuscitation and intravenous (IV) antibiotics. IV parenteral nutrition is begun in patients that have undergone resection of a significant length of bowel. Broad-spectrum antibiotics, such as ampicillin, clindamycin, and gentamicin or cefotetan, should be administered to the patients in whom vascular compromise, bowel necrosis, perforation, or sepsis is suspected. These agents have proven effective in decreasing the rate of postoperative wound infection and improving outcome. Empiric initial antimicrobial therapy must be comprehensive and should cover all likely pathogens in the clinical setting.[49]

ANORECTAL MALFORMATION

Introduction

Anorectal malformations occur in approximately 1 per 5000 live births.

Clinical Features

Imperforate anus is generally recognized at the initial physical examination or by failure to pass meconium within the first 48 hours of life. There is high incidence of associated anomalies of the urogenital sinus, renal agenesis, vesicoureteral reflux, neurogenic bladder, renal dysplasia, megaureter, hydronephrosis, and ectopic ureter. Entire spectrum of the VACTERL syndrome is common.[68]

Diagnosis

The radiologic evaluation of a newborn with imperforate anus includes abdominal ultrasonography to evaluate for urologic anomalies. In patients with persistent cloaca, a distended vagina (hydrocolpos) may be identified. Plain radiography may reveal spinal anomalies, such as spina bifida and spinal hemivertebrae, a hemisacrum and sacral hemivertebrae which may also affect the lumbar and thoracic spine, leading to scoliosis. Spinal ultrasonography may be done to find evidence of a tethered spinal cord and other spinal anomalies which may result in motor and sensory disturbances of the lower extremities, syringomyelia and myelomeningocele.[69,70]

Treatment

For neonates born with an anorectal anomaly, early treatment is crucial.[71] After birth, an intravenous line is placed for fluids and antibiotics. A nasogastric tube is inserted to keep the stomach decompressed to avoid the risk of vomiting and aspiration. Abdominal distension does not develop during the first few hours of life but is required to force meconium through a rectoperineal fistula, as well as through a urinary fistula.[72] The intra-abdominal pressure must be high enough to overcome the tone of the muscles that surround the rectum to force meconium through the fistula. Therefore, the decision to perform a colostomy or an anoplasty must be delayed for 16-24 hours.

Some of these infants require a decompressive colostomy before definitive surgery.[73] Before anesthetic induction, it may be prudent to perform echocardiography to rule out associated congenital heart disease. Preductal and postductal oxygen saturation determinations may be of value.[74]

Management of Anesthesia

Intraoperative management: As they have massive abdominal distension, they are to be treated like "full stomach". Slight head up tilt may help in breathing.

Spinal abnormalities should be ruled out before giving caudal anesthesia.

Postoperative management: Hyperchloremic metabolic acidosis can occur with rectovesical fistula caused by urine absorption. Urinary tract infections are common.[75]

INTUSSUSCEPTION

Introduction

Intussusception is produced by the invagination or telescoping of one portion of the intestine into another (Fig. 19.10). Over 50% of cases occur in children under 1 year of age, and less than 10% of cases occur in children older than 5 years.[76,77] About 90% of intussusceptions are ileocolic, with the remainder being ileoileal and colocolic.

Pathophysiology

Ninety percent of cases have idiopathic causes, which are frequently seen in children less than 1 year of age. Older children are more likely to have Meckel's diverticulum, intestinal polyp, lymphoma, adhesions, trauma hemolytic uremic syndrome, or ectopic pancreatic nodule as an etiology. Intussusception results in bowel obstruction, followed by congestion and edema with venous and lymphatic obstruction. This progresses to arterial obstruction and subsequent necrosis of the bowel. Ischemia and then necrosis results in fluid sequestration and bleeding from the GI tract. If untreated, the bowel may perforate, resulting in sepsis.

Clinical Features

The clinical presentation of acute intussusception involves sudden paroxysms of abdominal pain, bloody stools and an abdominal mass. Intussusception can also present with neurologic findings (lethargy, apnea, seizures, hypotonia, opisthotonus) similar to a picture of septic encephalopathy. Other symptoms and signs may include diarrhea, vomiting, fever, and dehydration.[78] In other children, intussusception can present as a chronic entity that may mimic gastroenteritis, whereas in neonates, intussusception may mimic necrotizing enterocolitis.

Major Criteria

- Evidence of intestinal obstruction: History of bile-stained emesis, along with abdominal distension or abnormal or absent bowel sounds.
- Features of intestinal invagination: Abdominal mass, rectal mass, or rectal prolapse, as well as an abdominal radiograph, sonogram, or CT scan showing visible intussusceptum or a soft-tissue mass.[79]
- Evidence of intestinal vascular compromise or venous congestion: This manifests as rectal bleeding or "red currant jelly" stool or blood on rectal examination.

Fig. 19.10: Intraoperative picture of intussusception
(For color version, see Plate 2)

Minor Criteria

Minor criteria includes any of the following:
- Male infants younger than 1 year
- Abdominal pain
- Vomiting
- Lethargy
- Pallor
- Hypovolemic shock
- Abdominal radiograph showing nonspecific abnormality.

Diagnosis

Plain radiography findings suggestive of intussusception include dilated loops of small bowel with or without air-fluid levels, an airless or opacified right lower quadrant.

Treatment

Treatment for intussusception involves the administration of appropriate fluids to combat dehydration. Radiologic reduction (hydrostatic or pneumatic) is attempted.[80,81] Laparotomy with manual reduction and/or resection as well as laparoscopic approaches have been described for the surgical management in patients with unsuccessful radiologic reduction and in patients with signs of intestinal perforation, peritonitis, and shock.

Management of Anesthesia

Preoperative management: Laboratory investigations may reflect dehydration, anemia, leukocytosis. Shock should be treated before commencing anesthesia. The intravascular deficits may be further exacerbated by the presence of barium in the gastrointestinal tract.[80]

Intraoperative management: The child with an intussusception should also be considered at risk for aspiration. Rapid sequence induction of anesthesia should take place because of the risk of regurgitation. If hemodynamic instability is a concern, ketamine or etomidate should be used as the hypnotic agent for induction. Nitrous oxide should be avoided. Regional anesthesia is avoided as it may lead to exaggerated bowel movements and precipitate perforation.[17]

ROUNDWORM OBSTRUCTION

Introduction

Intestinal roundworm, Ascaris lumbricoides is the most common and is associated with malnutrition, iron-deficiency anemia, and impairments of growth and cognition.[82]

Clinical Features

Diffuse or epigastric abdominal pain, nausea, vomiting, "tingling throat", frequent throat clearing, and dry cough are the usual manifestations. Biliary and intestinal obstruction, appendicitis, pancreatitis are common. Initial manifestation may be hepatic abscess or tropical pulmonary eosinophilia. Complications attributed to larval migration typically involve the lungs, and intestinal tract, kidneys, brain, and heart. Severe complications later in disease are usually attributable to mechanical obstruction of intestines or biliary tree secondary to high worm burden and allergic reaction to ascaris infection (urticaria).

Diagnosis

CBC may show eosinophilia. Sputum analysis may reveal larvae or Charcot-Leyden crystals.[83] Stool examination findings are typically normal in absence of previous infection (during the first 40 days). Increase in IgE and later IgG occurs. Abdominal radiography may reveal adult worms (especially with contrast). Obstructing Ascaris lesions cause cylindrical filling defects on contrast computed tomography (CT) scans. ERCP or MRCP may detect adult worms in bile or pancreatic ducts. Ultrasonography may detect worms in the gallbladder.

Fig. 19.11: Intraoperative picture of roundworm bolus
(For color version, see Plate 2)

Treatment

It includes intravenous hydration, nasogastric suctioning, electrolyte monitoring, and laparotomy if conservative measures fail (Fig. 19.11). Colonoscopy and esophagogastroduodenoscopy (EGD) may be useful in removing obstructing masses of worms.

Management of Anesthesia

Preoperative assessment: Child is kept NBM to deprive roundworms of nutrients and 25% dextrose is supplemented IV to provide calories. Calcium gluconate 1 ml/kg increases gastrointesinal motility which helps to propel the worms distally.

Intraoperative management: Anaphylactic reaction leads to histamine release resulting in vasodilatation, severe hypotension and metabolic acidosis. Inhaled beta-agonists and steroids may be indicated for intraoperative bronchospasm.

REFERENCES

1. Cote CJ. Pediatric Anesthesia. In: Mller RD, Ed. Miller's Anesthesia. 7th edn. New York: Churchill Livingstone 2010;2559-98.
2. Lee C, Luginbuehl I, Bissonnette B, Mason LJ. Pediatric Diseases. In: Paul AK, Hines RL, Marschall KE, editors. Stoelting's Anesthesia and Co-existing Disease. 5th edn. India: Saunders, imprint of Elsevier 2008;658-721.
3. Brett CM, Davis PJ, Bhikhazi G. Anaesthesia for neonates and premature infants. In: Motoyama EK, Davis PJ, Eds. Smith's Anesthesia for infants and children, 7th edn. Philadelphia: Mosby Elsevier 2006;521-70.
4. Weber T, Au-Fliegner M, Downard C, Fishman S. Abdominal wall defects. Curr Opin Pediatr 2002;14: 491-7.

5. Langer J. Abdominal wall defects. World J Surg 2003; 27:117-24.
6. Turnage RH, Richardson KA, Li BD, McDonald JC. Abdominal Wall, Umbilicus, Peritoneum, Mesenteries, Omentum, and Retroperitoneum. In: Courtney M. Townstead R, Beuchamp D, Evers BM, Mattox KL, Editors. Sabiston's book of Surgery. 18th edn. India: Saunders, imprint of Elsevier 2007;1129-54.
7. Morgan GE Jr, Mikhail MS, Murray MJ, Editors. Pediatric anesthesia. In: Clinical Anesthesiology. 4th edn. Boston: Mc Graw Hill 2008;922-50.
8. Towne BH, Peters G, Chang JHT: The problem of "giant" omphalocele. J Pediatr Surg 1980;15:543.
9. Davis MW, Kimble RM, Cartwright DW et al. Gastroschisis: ward reduction compared with traditional reduction under general anesthesia. J Pediatr Surg 2005;40:523-7.
10. Schlatter et al. Improved outcomes in the treatment of gastroschisis using a preformed silo and delayed repair approach. J Pediatr Surg 2003;38(3):459-64.
11. Wesley JR, Drongowski R, Coran AG. Intragastric pressure measurement: a guide for reduction and closure of the silastic chimney in omphalocele and gastroschisis. J Pediatr Surg 1981;16(3):264-70.
12. Yaster M, Buck JR, Dudgeon DL, et al: Hemodynamic effects of primary closure of omphalocele/gastroschisis in human newborns. Anesthesiology 1988;69:84.
13. Vane DW, Abajian JC, Hong AR: Spinal anesthesia for primary repair of gastroschisis: A new and safe technique for selected patients. J Pediatr Surg 1994; 29:1234.
14. Bianchi A, Dickson AP. Elective reduction and no anesthesia: minimal intervention management for gastroschisis. J Pediatr Surg 1998;33:1338-40.
15. Bianchi A, Dickson AP, Alizai NK. Elective delayed midgut reduction - no anesthesia for gastroschisis: selection and conversion criteria. J Pediatr Surg 2002;37:1334-6.
16. Hammer G, Hall S, Davis PJ. Anesthesia for General Abdominal, Thoracic, Urologic, and Bariatric Surgery. In: Motoyama EK, Davis PJ, Eds. Smith's Anesthesia for infants and children, 7th edn. Philadelphia: Mosby Elsevier 2006;685-722.
17. Spicer RD. Infantile pyloric stenosis: a review. Br J Surg 1982;69:128-35.
18. Halperin ML, Goldstein MB. Fluid, Electrolyte and Acid-base Emergencies. 18th edn. Philadelphia: W.B. Saunders Co. 1988.
19. Rose BD. Clinical Physiology of acid-base and electrolyte disorders. 2nd edn. New York: McGraw-Hill Book Co. 1984.
20. Bissonnette B, Sullivan PJ. Pyloric stenosis. Can J Anaesth 1991;38:668-76.
21. Daly AM, Conn AW. Anaesthesia for pyloromyotomy: a review (The Hospital for Sick Children, Toronto). Can Anaesth Soc J 1969;16:316-20
22. MacDonald NJ, Fitzpatrick GJ, Moore KP, et al. Anaesthesia for congenital hypertrophic pyloric stenosis. Br J Anaesth 1987;59:672-7.
23. Peutrell JM, Wilkins DG. Pyloric stenosis in full-term babies: a postal survey of the management by paediatric anaesthetists. Paediatr Anaesth 1994;4:93-7.
24. Andropoulos DB, Heard MB, Johnson KL, et al. Postanesthetic apnea in full-term infants after pyloromyotomy. Anesthesiology 1994;80:216-9.
25. Chipps BE, Moynihan R, Schieble T, et al. Infants undergoing pyloromyotomy are not at risk for postoperative apnea. Pediatr Pulmonol 1999;27:278-81.
26. Cook-Sather SD, Tulloch HV, Liacouras CA, Schreiner MS: Gastric fluid volume in infants for pyloromyotomy. Can J Anaesth 1997;44:278-183.
27. Steven IM, Alien TH, Sweeney DB. The congenital hypertrophic stenosis: the anaesthetist's view Anaesth intensive care 1973;1:544-6.
28. Cook-Sather SD, Tulloch HV, Cnaan A, et al. A comparison of awake versus paralyzed tracheal intubation for infants with pyloric stenosis. Anesth Analg 1998;86:945-51.
29. Davis PJ, Galinkin J, McGowan FX, et al. A randomized multicenter study of remifentanil compared with halothane in neonates and infants undergoing pyloromyotomy. I. Emergence and recovery profiles. Anesth Analg 2001;93:1380-6.
30. Galinkin JL, Davis PJ, McGowan FX, et al. A randomized multicenter study of remifentanil compared with halothane in neonates and infants undergoing pyloromyotomy. II. Perioperative breathing patterns in neonates and infants with pyloric stenosis. Anesth Analg 2001;93:1387-92.
31. Wolf AR, Lawson RA, Dryden CM, Davies FW. Recovery after desflurane anesthesia in the infant: comparison with isoflurane. Br J Anaesth 1996;76:362-4.
32. McNicol LR, Martin CS, Smart NG, et al. Perioperative bupivacaine for pyloromyotomy pain. Lancet 1990;335: 54-5.
33. Kovacs L, Papageorgiou, A. Incidence, Predisposing Factors and Outcome of NEC in Infants <32 Weeks' Gestation [dissertation/master's thesis]. Presented at PAS 2007, Toronto: SMBD-Jewish General Hospital, McGill University, Montreal 2007.
34. Wiswell TE, Robertson CF, Jones TA, Tuttle DJ. Necrotizing enterocolitis in full-term infants. A case-control study. Am J Dis Child. May 1988;142(5):532-5.
35. Stevenson JK, Stevenson DK. Necrotizing enterocolitis in the neonate. Surgery Annual 1977;147-69.
36. Bell MT, Shackelford P, Femin RD, Ternaerg JL, Brotherton T. Epidemiologic and bacteriologic evaluation of neonatal necrotizing enterocolitis. J. Pediatr. Surg 1979;14:1-4.
37. Touloukian RJ. Neonatal necrotizing enterocolitis: an update on etiology, diagnosis, and treatment. Surg. Clin. N. Amer 1976;56(2):281-97.
38. Arnon RG, Fishbein JF. Portal venous gas in the pediatric age group. J. Pediatr 1971;79:255-9.
39. Vollman JH, Smith WI, Tsang TC. Necrotizing enterocolitis with recurrent hepatic portal venous gas. J Pediatr 1976;88:486-7.

40. Leonidas JC, Hall RT, Amoury RA. Critical evaluation of the roentgen signs of neonatal necrotizing enterocolitis. Ann. Radiol. 1976;19:123-32.
41. Springer SC, Annibale DJ. Necrotizing Enterocolitis: Overview, Differntial diagnosis and workup, Treatment and Medication, Followup, Multimedia. [Article on the Internet]. [cited : Sep 22, 2009]; Available from: http://emedicine.medscape.com/article/977956.
42. Deeg KH, Rupprecht T, Schmid E. Doppler sonographic detection of increased flow velocities in the celiac trunk and superior mesenteric artery in infants with necrotizing enterocolitis. Pediatr Radiol 1993;23(8):578-82.
43. Haselby KA, Dierdorf SF, Krishna G, Rao CC, Wolfe TM and Mcniece WL. Anaesthetic implications of neonatal necrotizing enterocolitis Can. Anaesth. Soe J 1982; 29(3):255-60.
44. Miller DR, Pearson HA, Baehner RL, Mcmillan CW. Smith's Blood Diseases of Infancy and Childhood, 4th edn. St. Louis: CV Mushy 1978.
45. Battersby EF, Hatch DJ and Towey RM. The effects of prolonged naso-endotracheal intubation in children. Anesthesia 1977;32:154-7.
46. Merritt JC, Sprague DH, Merritt WE. and Ellis RA. Retrolental fibroplasia: a multifactorial disease. Anesth. Analg 1981;60:109-11.
47. Dickson E. Emergency surgery: Neonatal, general and airway. In: Doyle E (Ed). 1st edn. Edinburg: Oxford University Press 2007; 388-426.
48. Yaster M, Roseberg MI. Anesthesia for newborn surgical emergencies. In: Longnecker DE, Brown DL, Newman MF, Zapol WM (Eds). Anesthesiology, New York: The McGraw-Hill Companies 2008; 1502-19.
49. Roberts JD, Romanelli TM and Todres ID. Neonatal emergencies. In Cote CJ, Lerman J, Todres ID, Editors. Practice of naesthesia for Infants and children, fourth edition. Philadelphia: Saunders Elevier 2009;583-6.
50. Cook-Sather SD, Liacouras CA, Previte JP, et al. Gastric fluid measurement by blind aspiration in paediatric patients: a gastroscopic evaluation. Can J Anaesth 1997; 44:168-72.
51. Britton JR, Britton HL. Gastric aspirate volume at birth as an indicator of congenital intestinal obstruction. Acta Paediatr. Aug 1995;84(8):945-6.
52. DeLorimier AA, Fonkalsrud EW, Hays DM. Congenital atresia and stenosis of the jejunum and ileum. Surgery. 1969;65(5):819-27.
53. Ashcraft KW, Holcomb GW, Murphy JP. Intestinal atresia and stenosis. In: Pediatric Surgery. 4th edn. Philadelphia, Pa: Elsevier-Saunders 2005.
54. Spigland N, Yazbeck S. Complications associated with surgical treatment of congenital intrinsic duodenal obstruction. J Pediatr Surg. Nov 1990;25(11):1127-30.
55. Erkan V, Blankenship W, Stahlman MT. The complications of chronic umbilical vessel catheterization. Pediatr Res 1968;2:317.
56. Brans YW, Ceballos R, Cassady G: Umbilical catheters and hepatic abscesses. Pediatrics 1974; 53:264-6.
57. Goetzman BW, Stadalnik RC, Bogren HG, et al: Thrombotic complications of umbilical artery catheters: a clinical and radiographic study. Pediatrics 1975;56:374-9.
58. Pandya S. Practical guidelines on fluid therapy 2008;27-31.
59. Hill LL. Body composition, normal electrolyte concentrations, and the maintenance of normal volume, tonicity, and acid-base metabolism. Pediatr Clin North Am 1990; 37:241-56.
60. Chaudry G, Navarro OM, Levine DS, Oudjhane K. Abdominal manifestations of cystic fibrosis in children. Pediatr Radiol 2006;36(3):233-40.
61. Ziegler M. Meconium ileus. In: Grosfeld JL, O'Neill JA, Jr, Fonkalsrud EW, and Coran AG. Pediatric surgery, 6th edn. Philadelphia: Mosby Elsevier 2006;1289-1303.
62. Steven LC, Gavel G, Young D, Carachi R. Immunoreactive trypsin levels in neonates with meconium ileus. Pediatr Surg Int 2006;22(3):236-9.
63. Santulli TV, Blanc WA. Congenital atresia of the intestine: pathogenesis and treatment. Ann Surg 1961;154:939-48.
64. Ein SH, Shandling B, Reilly BJ, Stephens CA. Bowel perforation with nonoperative treatment of meconium ileus. J Pediatr Surg 1987;22(2):146-7.
65. O'Neill JA Jr, Grosfeld JL, Boles ET Jr, Clatworthy HW Jr. Surgical treatment of meconium ileus. Am J Surg 1970; 119(1):99-105.
66. Bishop HC, Koop CE. Management of meconium ileus; resection, Roux-en-Y anastomosis and ileostomy irrigation with pancreatic enzymes. Ann Surg 1957;145(3): 410-4.
67. Dott NM. Anomalies of intestinal rotation: Their embryology and surgical aspects with report of five cases. Brit J Surg 1923;11:251.
68. Rich MA, Brock WA, Pena A. Spectrum of genitourinary malformations in patients with imperforate anus. Pediatric Surg Intl 1988;3:110-3.
69. Beals RK, Robbins JR, Rolfe B. Anomalies associated with vertebral malformations. Spine 1993;18(10):1329-32.
70. Levitt MA, Patel M, Rodriguez G. The tethered spinal cord in patients with anorectal malformations. J Pediatr Surg 1997;32(3):462-8.
71. Levitt MA, Pena A. Outcomes from the correction of anorectal malformations. Curr Opin Pediatr 2005;17(3): 394-401.
72. Parrott TS. Urologic implications of anorectal malformations. Urol Clin North Am 1985;12(1):13-21.
73. Pena A. Current management of anorectal anomalies. Surg Clin North Am 1992;72(6):1393-416.
74. Kouwenberg M, Severijnen RS, Kapusta L. Congenital cardiovascular defects in children with intestinal malrotation. Pediatr Surg Int 2008;24(3):257-63.
75. Shaul DB, Harrison EA. Classification of anorectal malformations—initial approach, diagnostic tests, and colostomy. Semin Pediatr Surg 1997;6(4):187-95.

76. Stringer MD, Pablot SM, Brereton RJ. Paediatric intussusception. Br J Surg 1992;79(9):867-76.
77. Dennison WM, Shaker M. Intussusception in infancy and childhood. Br J Surg 1970;57(9):679-84.
78. Avansino JR, Bjerke S, Hendrickson M, Stelzner M, Sawin R. Clinical features and treatment outcome of intussusception in premature neonates. J Pediatr Surg 2003;38(12):1818-21.
79. Parienty RA, Lepreux JF, Gruson B. Sonographic and CT features of ileocolic intussusception. AJR Am J Roentgenol 1981;136(3):608-10.
80. Shekherdimian S, Lee SL, Sydorak RM, Applebaum H. Contrast enema for pediatric intussusception: is reflux into the terminal ileum necessary for complete reduction? J Pediatr Surg 2009;44(1):247-9; discussion 249-50.
81. Collins DL, Pinckney LE, Miller KE, et al. Hydrostatic reduction of ileocolic intussusception: a second attempt in the operating room with general anesthesia. J Pediatr 1989;115(2):204-7.
82. Cappello M, Hotez PJ. Intestinal Nematodes. Philadelphia: Churchill-Livingstone 2003;1331-3.
83. Sellimoglu MA, Ozturk CF, Ertekin V. A rare manifestation of ascariasis: encephalopathy J Emerg Med 2005; 28(1):87-8.

Section VI

ENT and Ophthalmic Emergencies

20 Anesthesia for Post-tonsillectomy Bleeding Patient

Swati S Chhatrapati, Nilam D Virkar, Anila D Malde

KEY POINTS

- Post-tonsillectomy bleeding is a surgical emergency. Patients may be profoundly hypovolemic and tachycardic. Anesthetic care includes both fluid resuscitation and meticulous airway management.
- Vigorous fluid resuscitation is the key to improve cardiac output and achieve hemodynamic stability before anesthesia to prevent fatal outcome.
- Baseline hemoglobin, hematocrit, platelet count, bleeding time and clotting time should be checked. If abnormal, hematology-consult and additional coagulation testing is required. Blood grouping and cross matching must be obtained prior to surgery.
- Post-tonsillectomy hemorrhage patients must be considered to have full stomach.
- Difficult intubation trolley including equipment for tracheostomy, trained assistance and senior surgeon must be available at the time of induction.
- The technique chosen to secure the airway will depend on the experience of the anesthesiologist and the physiologic and the airway status of the patient.
- Options for anesthetic induction include a rapid sequence induction with cricoid pressure or an inhaled induction.
- A large bore gastric tube is passed after intubation to decompress the stomach at the beginning and at the end of the surgery.
- Patients are extubated awake in lateral position when protective airway reflexes have returned.

Tonsillectomy is one of the most frequently performed surgical procedures in children. Tonsillectomy and adenoidectomy should not be considered minor procedures as they involve a shared airway, often in a small child, with difficult access, obstructive airway symptoms and the potential for blood contamination of the lower airway. Mortality associated with tonsillectomy ranges from 1:40,000 to 1:12,000.[1,2]

Postoperative hemorrhage is the most common complication related to adenotonsillectomy and is responsible for the majority of postoperative fatalities. Reported postoperative hemorrhage rates range from 0.5 to 10 percent although variable rates have been reported with different surgical techniques.[3]

Surgical techniques for tonsillectomy include guillotine resection (rare), cold dissection, bipolar dissection, and cold ablation (coblation) dissection. Each approach has its particular merits. Blood loss is greater but pain is less with the first two techniques. Bipolar dissection allows immediate coagulation and less blood loss. However, thermal injury to the surrounding healthy tissue is responsible for greater postoperative discomfort. Coblation is a relatively new technique. Coblation offers the advantages of both cold dissection and bipolar diathermy, with less postoperative pain and blood loss.[3] However, the incidence of post-tonsillectomy hemorrhage with coblation tonsillectomy has been reported up to 11.1 percent. Postoperative bleeding after adenotonsillectomy originates from the tonsillar fossa in 67 percent, and from nasopharynx in 26 percent and from both in 7 percent.[4]

Post-tonsillar bleeding is classified as primary or secondary bleeding. Primary bleeding occurs within first 24 hours after surgery. It usually results from significant emesis, retching, or straining secondary to swallowed blood or pain in addition to inadequate surgical technique.[5] Meticulous attention to surgical technique and intraoperative hemostasis along with

adequate treatment of pain and emesis reduce the risk of hemorrhage in the immediate postoperative period. Approximately 75 percent of post-tonsillar hemorrhage occurs within 6 hours of surgery.[3]

It is therefore desirable to observe the patient for 8 to 10 hours after the surgery in the day-care surgery setting. Secondary bleeding usually occurs 24 hours to 5 to 10 days after surgery and may be associated with sloughing of the eschar, loosened vessel ties or infection from underlying chronic tonsillitis. The most common time for secondary bleeding is between 5th and 7th postoperative days.[3]

Intraoperative hemorrhage during adenotonsillectomy is usually related to underlying coagulopathy or major arterial damage in severe cases. It can be controlled using suction cautery or ligation or by placing a pack in the tonsillar fossa and oversuturing the tonsillar pillars to provide constant compression of the fossa. In severe cases, ligation of larger arteries through an open neck exploration may be necessary. Postoperative bleeding after adenoidectomy may be controlled initially with topical decongestants nose drops. Patients with severe bleeding are taken back to the operating room for examination of the nasopharynx and hemostasis where adenoid remnants are removed.[3]

In the National Prospective Tonsillectomy Audit (July 2003 to September 2004), the incidence of post-tonsillectomy bleeding was 3.5 percent and the overall rate of return to theater was 0.9 percent. The incidence of primary hemorrhage was 0.6 percent and the majority of these occurred within the first 6 hours after the operation. Factors influencing the hemorrhage rates were age (lower rates in children than adults), indication for surgery (highest rates with quinsy and recurrent tonsillitis, lowest with obstructive symptoms), and surgical technique (higher rates with use of diathermy and disposable equipment, lowest with blunt dissection).[6]

Generally, in children the tonsils can be dissected and removed more easily than in adults. In adults, the tissue is often scarred and fibrotic from repeated infections. This leads to greater incidence of post-operative hemorrhage in the older age group.[1]

Richmond reported a series of 794 adenotonsillectomy patients of whom 4.2 percent had variable degrees of postoperative bleeding.[3] In 9409 pediatric patients undergoing adenotonsillectomy, Crysdale and Russel reported an overall hemorrhage rate of 2.15 percent with 0.06 percent of the patients requiring a second general anesthetic for hemostasis.[7]

Diagnosing patients with underlying coagulation disorders either by history, physical examination, or laboratory studies help avert significant perioperative blood loss. Certain analgesics, e.g. ketorolac and drugs like aspirin, have been associated with increased hemorrhage rates when given in the perioperative period and should be avoided or stopped well in advance in patients undergoing adenotonsillectomy.[8]

A study by Brown et al noted that nearly half of post-tonsillectomy hemorrhage cases occurred in patients with previously undiagnosed coagulation disorders.[9]

Post-tonsillectomy bleeding is an emergency procedure. It often requires dealing with anxious parents, upset surgeons, and frightened, anemic hypovolemic children with stomach full of blood. The management of anesthesia can be challenging even in the hands of an experienced anesthesiologist.

THE ANESTHETIC CONSIDERATIONS IN POST-TONSILLECTOMY BLEEDING INCLUDE

1. Hypovolemia: Adequate fluid resuscitation is essential before induction. Care should be exercised when using anesthetic agents that may cause vasodilatation and hypotension.
2. Pulmonary aspiration: These patients must be considered to have a full stomach due to swallowed blood and therefore are at increased risk of pulmonary aspiration.
3. Potential for a difficult laryngoscopy and intubation: Laryngoscopy is likely to be difficult due to presence of blood clots in the pharynx, bleeding tonsillar bed, and reduced venous and lymphatic drainage causing intraoral swelling and edema. Earlier airway instrumentation may lead to vocal cord edema with or without subglottic edema. If so, a smaller tracheal tube should be considered.
4. The effects of the previous general anesthetic and perioperative opioids.
5. Abnormal clotting: Nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided.
6. Airway obstruction: Presence of blood in the airway or a large clot in the oral cavity may produce airway obstruction.

Preoperative Assessment

A thorough history and review of the anesthetic record of the original surgery will provide pertinent information about pre-existing medical conditions (especially bleeding diathesis), use of medications such as aspirin, difficulty with airway management and a rough estimate of intraoperative blood loss and fluid replacement as well as duration of known bleeding and the volume of blood vomited since the bleeding began.

Assessment of the child's volume status must be carried out. If the child is sitting up and is talking without feeling dizzy, hypovolemia is only mild to moderate. With 10 to 15 percent loss of blood volume, blood pressure may remain normal due to compensatory increase in heart rate. The patient may be tachycardic with elevated blood pressure due to release of endogenous catecholamines from hemorrhage, hypovolemia or fear and excitement. Tachycardia, weak or thready pulse, tachypnea, delayed capillary refill, decreased urine output, cold extremities with mottling or cyanosis and altered sensorium are early indicators of hypovolemia in children. History of dizziness and presence of orthostatic hypotension suggests 20 to 30 percent decrease in blood volume. Pallor, poor skin turgor, listless child, and hypotension are indicators of advanced volume depletion. Increased swallowing, coffee ground emesis, and airway obstruction are indirect indicators of bleeding. When there is redevelopment of earache, six or more days after tonsillectomy, one must look out for secondary hemorrhage.

Signs of blood volume loss are shown in Table 20.1.[10]

Table 20.1: Stages of pediatric blood volume loss (shock) and associated clinical signs[10]

Blood volume loss	*System*	*Clinical signs*
<20%	CVS	Tachycardia; weak, thready pulses
	Skin	Cool to touch, capillary refill 2 to 3 seconds
	Renal	Slight decrease in urine output, increase in specific gravity
	CNS	Irritable, may be combative
25%	CVS	Tachycardia; weak, thready distal pulses
	Skin	Cold extremities, cyanosis and mottling
	Renal	Decrease in urine output
	CNS	Confusion, lethargy
40%	CVS	Frank hypotension; tachycardia may progress to bradycardia
	Skin	Pale, cold
	Renal	No urine output
	CNS	Comatose

Investigations

Baseline hemoglobin, hematocrit, platelet count, bleeding time, and clotting time must be checked. Hematocrit value should be checked after rehydration to have a more accurate estimate of blood loss. If any abnormality is detected in baseline investigations, hematology consult and additional coagulation testing should be obtained. Blood grouping and cross-matching should be obtained prior to surgery.

Anesthetic Management

The management of anesthesia can be considered under two heads:

1. Resuscitation
2. The procedure in the operating room

Resuscitation

Post-tonsillectomy bleeding is almost always venous or capillary ooze rather than arterial. Although it may be quite brisk, resuscitation rather than immediate operation must be the first step in dealing with it. The child with the bleeding tonsil is hypovolemic and has a decrease in cardiac output secondary to ongoing blood loss. If blood loss is severe and/or fluid resuscitation is not vigorous, lactic acidosis and an eventual state of shock will develop. The compensatory response to acute blood loss is release of catecholamines. This causes peripheral vasoconstriction which delays the clinical onset of hypotension in the awake child. When anesthesia induced vasodilation occurs, profound hypotension is observed. Significant cardiovascular collapse can occur if a general anesthetic is induced in the severely hypovolemic patient. Hence, assessment of volume status and aggressive fluid resuscitation should be carried out before taking the patient to the operating room. Typically blood loss during tonsillectomy averages 4 ml/kg.[11]

However, it is often difficult to measure the exact blood loss as it occurs over several hours and is partly swallowed. Also, children have excellent autoregulation due to which vital signs do not change until a potentially fatal blood loss occurs.

The child must be kept in the lateral position with head low to prevent aspiration. Oxygen must be given via Hudson's mask. Two larger bore intravenous lines must be established for rapid hydration. An intraosseous infusion or surgical cutdown may be indicated in severely hypovolemic child with extreme cutaneous vasoconstriction. Vigorous fluid resuscitation with crystalloids in repeated boluses of 20 ml/kg of balanced salt solution and/or colloids is the key to improve the cardiac output and achieve hemodynamic stability before induction of anesthesia. If hematocrit is less than 25 percent, blood should be administered before surgery since crystalloid or colloid will further drop the hemoglobin and hematocrit.[11]

Other colloids like fresh frozen plasma may be needed depending on the clotting status and medical

history of the patient. More blood and blood products should be available for surgery. Once vital signs have improved and are stable, then the child is taken to the operating room, the degree of tachycardia being a useful sign of the efficacy of fluid replacement.

Restlessness is often due to hypovolemia, hence preoperative sedation must be avoided.

The Procedure in the Operating Room

The patient is brought into the operating room and monitors are placed before induction of anesthesia.

Essential monitoring consists of precordial stethoscope, electrocardiogram, pulse oximeter, non-invasive blood pressure monitoring, capnograph, and thermometer. A nerve stimulator may be used to track the depth of paralysis.

Before the induction of anesthesia, all the precautions and preparations for the patient with a full stomach and difficult laryngoscopy and intubation should be made. These include well-functioning suction apparatus with large bore suction tubes, extra-laryngoscope handles and blades with good light, and several cuffed endotracheal tube with lubricated stylets. A selection of smaller tracheal tubes may be needed if airway and tracheal edema is significant. The otolaryngologist should be present before anesthetic induction. Equipment for an immediate tracheostomy should be readily available. An experienced assistant should always be available to help the anesthesiologist and to apply cricoid pressure during the rapid sequence induction.

Preoxygenation can be done with the patient in the lateral, head down position to encourage blood to drain out of the mouth. The child is then turned supine. Consideration should be given to adopting left lateral position for induction if bleeding is excessive.

Options for induction of anesthesia include a rapid sequence induction with cricoid pressure or an inhaled induction. The technique chosen to secure the airway will depend on the experience of the anesthesiologist and the physiologic and the airway status of the patient.

Rapid Sequence Induction

In the absence of respiratory distress and the absence of distorted airway anatomy (e.g. tracheal deviation), a rapid sequence induction will be safe.

The advantage of rapid sequence induction with cricoid pressure is rapid induction and control of the airway with less chance of regurgitation during induction. The disadvantages are the potential to inhale blood and cardiovascular depression from induction. Also, there may be difficulty in visualizing the cords after giving muscle relaxant.

The choice of induction drug should be based on the patient's volume status and hemodynamic stability. If the volume status is borderline or if there is rapid ongoing blood loss, etomidate 0.3 to 0.4 mg/kg is a good choice. While ketamine 1 to 2 mg/kg may also be safe, myocardial depression and hypotension can still be seen in hypovolemic patients. The adequately resuscitated patients will tolerate propofol (2 mg/kg) or thiopentone sodium 3 to 6 mg/kg. The reduced doses of thiopentone sodium 2 to 3 mg/kg or propofol 1 to 2 mg/kg may be used in hypovolemic patients. The neuromuscular blockers that give the best intubating conditions in less than 60 to 90 seconds would be succinyl choline 2 mg/kg or rocuronium 1.2 mg/kg. However, rocuronium may result in paralysis outlasting the surgical procedure. In children older than 4 years, a cuffed endotracheal tube is selected to minimize the chance of blood aspiration around the tube. The cuffed tube which is 0.5 to 1 mm smaller in diameter than the appropriate sized uncuffed tube should be chosen. The use of a stylet is recommended despite a previous history of easy intubation.[12]

Both sides of the chest are carefully auscultated and the endotracheal tube is suctioned to rule out aspiration of blood or gastric contents. After intubation, a large bore gastric tube should be placed to decompress the stomach at the beginning and at the end of the procedure.

Inhaled Induction

If a patient has respiratory distress before the induction of anesthesia, using inhalation anesthesia and a FiO_2 of 1.0 will allow the anesthesiologist to determine if positive pressure ventilation can be administered before giving muscle relaxant.

An inhaled induction can be challenging in the lateral or the head down position. The advantage of this technique is that spontaneous respiration is maintained and is helpful in patients with anticipated difficult intubation. However, this technique can be slow and blood can be inhaled precipitating laryngospasm. Also, hypotension can be precipitated if there is any degree of hypovolemia.

Maintenance of Anesthesia

Controlled ventilation provides good conditions for hemostasis. A nondepolarizing muscle relaxant such as vecuronium 0.05 to 0.1 mg/kg after the effects of succinylcholine have worn off helps maintain a lighter

level of anesthesia. Continuation of the resuscitation and maintenance of anesthesia should be tailored according to hemodynamics and the patient's response to induction. Titration of a volatile anesthetic such as sevoflurane or desflurane with nitrous oxide and oxygen supplemented with an opioid such as fentanyl 1 to 2 µg/kg will facilitate rapid recovery at the end of the surgery while limiting reflex induced tachycardia, hypertension, arrhythmia, coughing and retching on the tube.

Great care must be taken when the surgeon places a mouth gag in the oral cavity to expose the tonsils. Some surgeons disconnect the anesthetic circuit prior to placing the mouth gag; others work around the endotracheal tube. After the mouth gag is in place, one should recheck to verify bilateral breath sounds and rule out compression, kinking or displacement of the tube. Breath sounds, chest excursion, peak airway pressure, end tidal CO_2, and oxygen saturation should be monitored at all times to detect any mishaps at the earliest.

These surgeries are not excessively painful as surgery is limited to the area of bleeding. Controlling the bleeding vessel in the tonsillar bed can be accomplished rapidly by the surgeon if the blood pressure is maintained in the normal range. Fluid resuscitation and transfusion of blood and blood products should continue intraoperatively as necessary. All post-tonsillectomy bleeding cannot be fully corrected surgically. Occasionally, an unrecognized bleeding diathesis results in oozing without an obvious source or no evidence of clot formation. Obtaining clotting studies and hematology consult may be indicated in such cases. Administering desmopressin (DDAVP) may show improvement in patients with von Willebrand's disease.[13]

At the end of the surgery, the gastric tube is suctioned but this does not guarantee an empty stomach as blood clots may still remain in the stomach. This, along with pharyngeal mucosal irritation predispose to postoperative vomiting. Hence, the use of prophylactic antiemetic therapy in the form of ondansetron 0.1 mg/kg or metoclopramide 0.15 mg/kg is indicated in these patients. Topical lidocaine 4 mg/kg in concentrations of 2 to 4 percent can be used to reduce the incidence of postextubation laryngospasm and is as effective as 1 mg/kg intravenous lidocaine without the sedative effects. Intravenous magnesium has also been used for the same purpose.[14,15]

Extubation

After the procedure, the anesthesiologist should again auscultate both sides of the chest to rule out the presence of aspirated blood or secretions. One should remember to verify removal of the throat pack before extubation. The pharynx must be suctioned under direct vision with atraumatic suction catheter to prevent bleeding caused by agitation of raw mucosal surfaces by a suction catheter. The anesthesiologist should never suction blindly the naso or oropharynx. The patient is then turned on to the side in the tonsillar position. After the neuromuscular block is antagonized, patients are extubated awake when protective airway reflexes have returned. Stormy emergence predisposes patients to rebleeding from the surgical site.

The patient is then shifted to the high quality recovery room in tonsillar position. If the non-dependent hip is flexed, the patient easily remains in this position. This position allows pooling of blood and secretions to occur on the side of the mouth rather than midline, thus decreasing the chances of laryngospasm. Also, the upper airway of a child widens in the lateral position and is less likely to obstruct.

Postoperatively, a repeat determination of the hemoglobin level may be indicated.

REFERENCES

1. The Royal College of Surgeons: National Prospective Tonsillectomy Audit. London, 2005.
2. Cohen D, Dor M. Morbidity and mortality of post-tonsillectomy bleeding: Analysis of cases. J Laryngol Otol 2008;122:88-92.
3. Wiatrak BJ, Woolley AL. Pharyngitis and Adenotonsillar disease. In: Cummings CW, Flint PW, Harker LA, Haughey BH, Richardson MA, Robbins KT, Schuller DE, Thomas JR, (Eds). Cummings Otolaryngology Head and Neck surgery. Volume four (4th edn). Philadelphia: Elsevier Mosby 2005;4135-65.
4. Ferrari LR, Gotta AW. Anesthesia for otolaryngologic surgery. In: Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MG, (Eds). Clinical Anesthesia (6th edn). New Delhi: Wolters Kluwer/Lippincott Williams and Williams 2009;1305-20.
5. Herlich A. Adenotonsillectomy. In: Reed AP, editor. Clinical Cases in Anesthesia. (2nd edn) New York: Churchill Livingstone 1995;211-4.
6. Ravi R, Howell T. Anaesthesia for paediatric ear, nose, and throat surgery. In: McCormick, editor-in-chief. Update in Anaesthesia. Vol-24, number-1. COS printers 2008;18 - 23.
7. Crysdale WS, Russel D. Complications of tonsillectomy and adenoidectomy in 9409 children observed overnight. Can Med Assoc J 1986;135:1139.
8. Gallagher JE, Blauth J, Fornadley JA. Perioperative ketorolac tromethamine and postoperative hemorrhage in cases of tonsillectomy and adenoidectomy. Laryngoscope 1995;105:606.

9. Brown KA, Morin I, Hickey C, et al. Urgent adenotonsillectomy. Anesthesiology 2003;99:586-95.
10. Reynolds PI, Solders O, Malviya S, Davis PJ. Perioperative Management of the Pediatric Trauma Patient. In: Motoyama EK, Davis PJ, (Eds). Smith's Anaesthesia for infants and children (7th edn). Philadelphia: Mosby Elsevier 2006;991-1015.
11. Fee WE Jr, Terris DJ, Gabalski EC, Troell RJ, Riley RW, Powell NB, Li KK, Roberson JB. Surgeons. Brock-Utue JG, Gaeta RR, Baer ER, Champeau MW. Anesthesiologists. Otolaryngology—Head and Neck Surgery. In: Jaffe RA, Samuels SI (Eds). Anesthesiologists manual of surgical procedures (2nd edn). Philadelphia: Lippincott Williams and Wilkins 1999;120-22.
12. Verghese ST, Hannallah RS. Pediatric otolaryngologic emergencies. Anesthesiology clinics of North America 2001;19(2):237-56.
13. Berlucchi M, Tomenzoli D, Nicolai P, et al. Adenotonsillectomy in children with von Willebrand's disease: how and when. A case report with review of the literature. Int J Pediatr Otorhinolaryngol 2002;65:253-56.
14. Koc K, Kocaman F, Aygenc E, et al. The use of preoperative lidocaine to prevent stridor and laryngospasm after tonsillectomy and adenoidectomy. Otolaryngol Head Neck Surg 1998;118:880-2.
15. Gulhas N, Durmus M, Demirbilek S, et al: The use of magnesium to prevent laryngospasm after tonsillectomy and adenoidectomy: A preliminary study. Pediatr Anesth 2003;13:43-7.

21 Anesthetic Considerations in Deep Neck Space Infections

Anila D Malde, Swati S Chhatrapati

KEY POINTS

- Deep neck space infections (DNSIs) are most commonly odontogenic and pharyngotonsillar in origin.
- The free communication of potential spaces of the head and neck and the minimal resistance offered by cervical fascia can have devastating consequences. Infections in any of the potential spaces and fascial planes of the neck, spreads rapidly and easily and may envelop vital structures within the neck including the larynx, the great vessels and lower cranial nerves.
- The danger space provides a conduit for invasion into the mediastinum, often leading to pleural and pericardial involvement.
- Older patients and patients with systemic disease are at increased risk of complications.
- Advanced cases of DNSI lead to narrowing and eventually to loss of airway.
- A thorough evaluation of the airway is an absolute necessity and should include use of the CT scan.
- Patients with DNSI present challenging airways for an anesthesiologist. Securing the airway must be a joint venture involving surgeon and anesthesiologist.
- Pharyngeal wall abscesses increase the risk of rupture and pus aspiration during intubation.
- During intubation, the surgeon must always be ready to perform an emergency tracheostomy.
- Awake fiberoptic intubation is safe and effective in patients with advanced DNSI but it requires additional skills and practice. When fiberoptic intubation is not feasible, not available or has failed, an awake tracheostomy may be the preferred option.
- Death from loss of an airway still occurs in patients of advanced DNSI with difficult airway.

The diagnosis and treatment of deep neck space infections (DNSIs) have challenged physicians and surgeons for centuries.

The DNSI may arise from several focuses in the head and neck; and those of dental and adenotonsillar origin are the most common.[1] In the preantibiotic era, most cases of DNSI (70%) were secondary to an oropharyngeal infection. The widespread use of antibiotics has reduced the role of pharyngotonsillitis in deep neck space infections. Today, odontogenic sources seem the most common origin of deep neck infections in adults.[2,3]

DEEP NECK INFECTIONS: A CONSTANT CHALLENGE

Although the advancement of antibiotics, improved dental care, the development of sophisticated diagnostic tools (e.g. CT, MRI) and continued development of medical intensive care protocols and surgical techniques have markedly reduced the incidence and mortality rates, DNSI remain a challenging problem due to the complex anatomy and potentially lethal complications that may arise.[4]

Management of patients with a deep neck abscess consists of airway management, IV antibiotics, and incision and drainage of abscess as necessary. Our anesthetic expertise is called upon for help in the management of the airway as well as for the administration of anesthesia for surgical incision and drainage.[5]

Patients with DNSI present challenging airways for an anesthesiologist. These infectious processes may progress rapidly to airway obstruction that could be lethal. A common cause of death in patients with DNSI is acute loss of airway during interventions to control it. Various techniques are available to secure the airway but the success and safety of these techniques in patients with DNSI have not yet been established.[6]

To understand the problems of patients with DNSI and its implications to airway management, one has to have a clear knowledge of the anatomy of the neck spaces and fascial relationships.[5] Within the deep neck space are 11 spaces created by planes of greater and lesser resistance between the fascial layers. These spaces may be real or potential and may expand when pus separates layers of fascia. The deep neck spaces, although have discrete boundaries, communicate with each other, forming avenues by which infections may spread[1,5] (Figs 21.1 and 21.2).

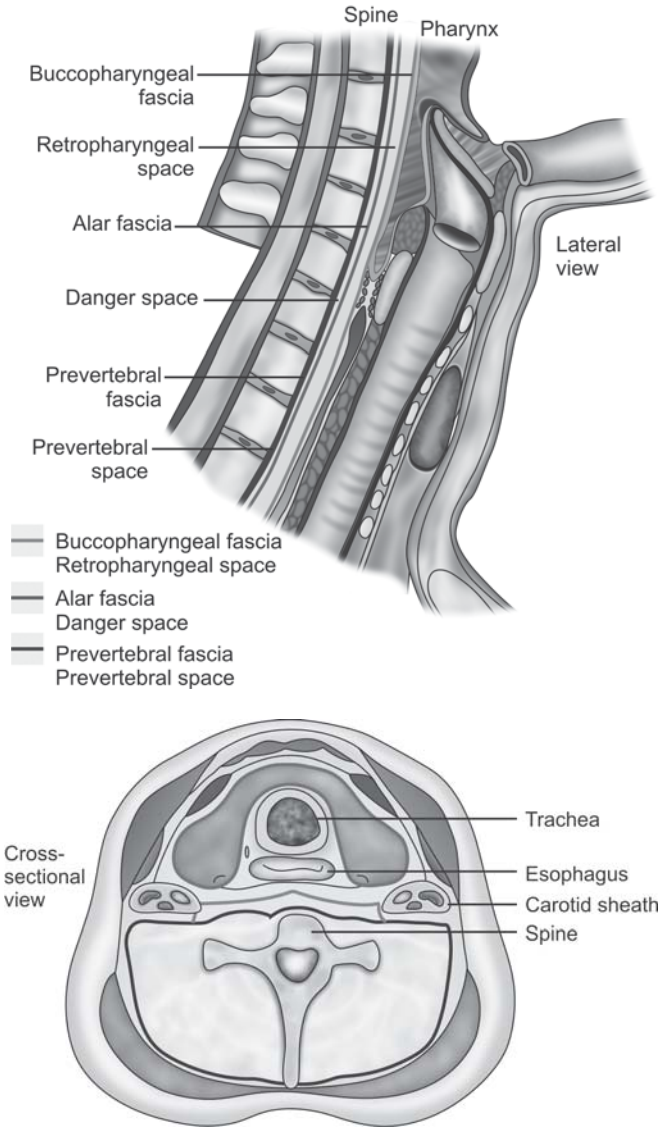


Fig. 21.1: Fascial spaces of the neck: Lateral and cross-sectional view

In general, the hyoid bone is the most important structure in the neck which limits the spread of infection.[5,7-9] Levitt[7] divides the fascial planes into:

- Those involving the entire length of the neck:
  - Retropharyngeal space
  - Danger space
  - Prevertebral space
  - Carotid or visceral vascular space.
- Those above the hyoid bone:
  - Submandibular space
  - Pharyngomaxillary or parapharyngeal or lateral pharyngeal space
  - Masticator space
  - Parotid space
  - Peritonsillar space
  - Temporal space.
- Those below the hyoid bone:
  - Anterior visceral space or pretracheal space.

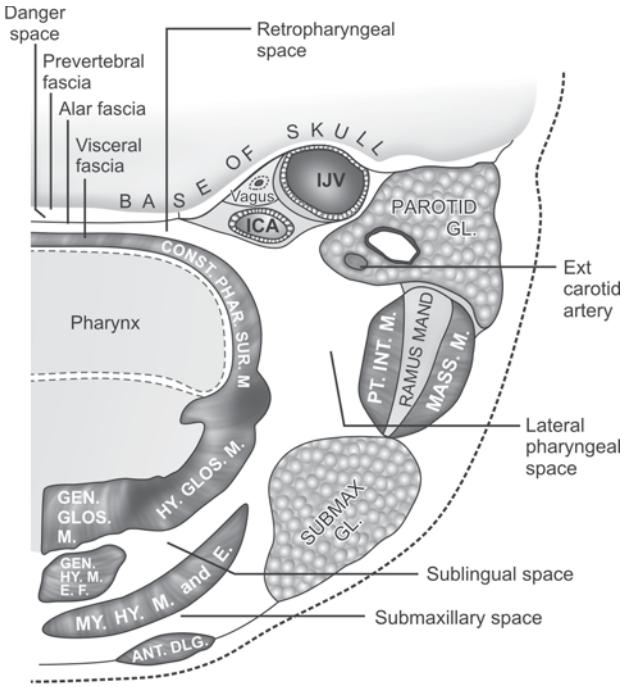


Fig. 21.2: Fascial spaces of the neck: Cross-sectional view at the level of pharynx illustrating sublingual and submaxillary spaces: IJV-Internal jugular vein; ICA-Internal carotid artery; PAROTID GL-Parotid gland; CONST.PHAR.SUP.M-Superior pharyngeal constrictor muscle; GEN. GLOS. M.-Genioglossus muscle; Hy. GLOS. M.-Hioglossus muscle; GEN. Hy. M.& F-Geniohyoid muscle and fascia; MY. HY. M. & F-Mylohyoid muscle and fascia; ANT. DIG-Anterior digastric muscle; PT. INT. M-Pterygoid internal muscle; RAMUS MAND-Ramus mandible; MASS. M-Massater muscle; SUBMAX. GL-Submaxillary gland

RETROPHARYNGEAL SPACE

Retropharyngeal space is located immediately posterior to the pharynx (nasopharynx, oropharynx, hypopharynx), larynx and trachea. It extends from the base of the skull to the upper mediastinum at the level of the tracheal bifurcation.

The visceral (buccopharyngeal) fascia, which surrounds the pharynx, trachea, esophagus and thyroid gland forms the anterior border of the retropharyngeal space. It is bounded posteriorly by the alar fascia and laterally by the carotid sheaths and parapharyngeal spaces. The danger space and the prevertebral space are in close proximity to the retropharyngeal space. The danger space is formed anteriorly by the alar fascia and posteriorly by the prevertebral fascia. The prevertebral space is bounded anteriorly by the prevertebral fascia and posteriorly by the longus colli muscles of the spine. The danger space extends down the mediastinum to the level of the diaphragm, whereas the prevertebral space continues to the insertion of the psoas muscles. These anatomic relationships can allow an infection of the retropharyngeal space to spread to the mediastinum, leading to potentially fatal mediastinitis.

The retropharyngeal space contains two chains of lymph nodes on either side of the midline that drain the nasopharynx, adenoid and paranasal sinuses.[7-11]

Etiology of Retropharyngeal Abscess

Retropharyngeal space is the second most common location for deep neck abscess in children.

- Acute retropharyngeal abscess is usually seen in children less than 4 to 5 years of age. It is due to spread of infection from nasopharynx, oropharynx and rarely from mastoid infection. Lymph nodes in the retropharyngeal space usually disappear due to recurrent infection and fibrosis after the age of 4 to 5 years. Hence, retropharyngeal abscess due to naso-oropharyngeal infections is rare in adults.[12,13]
- In adults, common causes of retropharyngeal abscess are some penetrating injury or foreign body piercing in the posterior pharyngeal wall (fish bones) or iatrogenic instrumentation such as esophagoscopy, endotracheal intubation, feeding tube insertion and dental injections.[9-11]
- The retropharyngeal infections also spread from contiguous spaces such as the parapharyngeal space, submandibular space, or prevertebral space (e.g. osteomyelitis).[9-11]
- Of special interest is the group of lateral retropharyngeal nodes at the base of the skull (Rouviere nodes) which constitute the primary lymphatic drainage of the nasopharynx. These become significant in cases of nasopharyngeal cancer. With secondary bacterial infection, they can lead to retropharyngeal abscess.
- Chronic retropharyngeal abscess is usually seen in adults or slightly elder children. It is due to tuberculous infection of the cervical spine. In children below 4 to 5 years, it occurs due to tuberculous infection of the retropharyngeal lymph nodes secondary to tuberculosis of deep cervical nodes.[12]

LATERAL PHARYNGEAL SPACE

This space occupies a critical area in the neck, as it communicates with all other fascial spaces. It sits as an inverted cone with its base at the base of the skull and apex at the hyoid bone. The lateral pharyngeal space is bounded by the pharynx medially, the styloid muscles and carotid sheath posteriorly, the parotid gland posterolaterally, the mandible, pterygoids and masetter anterolaterally, and the pterygomandibular raphe anteriorly. The lateral pharyngeal space connects posteromedially with the retropharyngeal space, and inferomedially with the submandibular space. Laterally, it connects with the masticator space. The carotid sheath courses through this space into the chest. This space communicates directly with the brain by way of foramina of the skull. It is directly involved by lateral extension of peritonsillar abscesses and was the most commonly affected space before the advent of modern antibiotics.

The lateral pharyngeal space can be divided into anterior (prestyloid) and posterior (poststyloid) compartments by the styloid process. The anterior compartment is also referred to as the muscular compartment. The posterior compartment is also referred to as the neurovascular compartment, as it contains the carotid sheath, cranial nerves[9-12] and the cervical sympathetic chain.[7-9,11,13]

Etiology of Lateral Pharyngeal Abscess

- Infections can arise from the tonsils, adenoids, pharynx, teeth, salivary glands, nasal infections or Bezold abscess.[13]
- Infection may spread from contiguous spaces.

PERITONSILLAR SPACE

The peritonsillar space is located lateral to the fibrous wall of the tonsillar capsule. Peritonsillar abscess originates as acute tonsillitis that extends through the fibrous capsule of the tonsil into the peritonsillar space from where it often extends into the lateral parapharyngeal space.[7-9,11,13]

SUBMANDIBULAR SPACE

The submandibular space extends from the hyoid bone to the mucosa of the floor of the mouth. It is bounded anteriorly and laterally by the mandible and inferiorly by the superficial layer of the deep cervical fascia. The myelohyoid muscle divides the space into sublingual and submaxillary spaces. These two subdivisions freely communicate around the posterior border of the myelohyoid.[7-9,11]

Ludwig's Angina

Ludwig's angina is a potentially lethal, rapidly expanding cellulitis of the floor of the mouth characterized by brawny induration of the upper neck, usually unaccompanied by obvious fluctuation.[14]

The word "Angina" is derived from the Latin word "*Angere*" which means "to strangle".[15,16] Ludwig's angina appropriately describes the subset of deep neck abscesses in which swelling of critical spaces threatens to elevate the floor of the mouth, displace the tongue upwards and posteriorly and thereby strangle the patient. Other authors used the ominous labels of angina maligna, morbus strangulatorius, and garotillo (Spanish for "hangman's loop") to describe the condition.[17]

Although one can find mention of the symptoms of this condition in writings dating back to Hippocrates and Galen, Ludwig's angina was best described initially in 1836 by its namesake, Karl Friedrich Wilhelm von Ludwig. He described this disease as a rapidly progressive gangrenous cellulitis originating in the region of the submandibular gland that extends by continuity rather than lymphatic spread.[14]

Grodinsky proposed four criteria to distinguish Ludwig's angina from other forms of deep neck abscesses. The infection must:

- Occur bilaterally
- Produce gangrenous serosanguinous infiltration with or no pus
- Involve connective tissues, fascia, and muscle but not glandular structures
- Spread by continuity, not by lymphatics.

Etiology of Ludwig's Angina

1. Odontogenic procedures and poor dental hygiene account for 70 to 80 percent of the cases of Ludwig's angina.[18] The second and third mandibular molars are most frequently involved. As their roots extend below the mandibular insertion of the myelohyoid muscle, the infection of the pulp of these teeth spreads to the submaxillary space after penetrating the inner cortex of the mandible. Owing to the communication around the posterior margin of the myelohyoid muscle, rapid involvement of the sublingual space occurs, followed quickly by involvement of the contralateral spaces. The unyielding presence of the mandible, hyoid and superficial layer of the deep cervical fascia limit tissue expansion as edema develops and progresses. This resistance leads to superior and posterior displacement of the floor of the mouth and the base of the tongue. Therefore, these patients have an open-mouth appearance, with a protruding or elevated tongue and exhibit marked neck swelling.[14] In effect, the path of least resistance towards the airway becomes the most vulnerable area anatomically, which is the genesis of the inevitable airway crisis.
2. Other risk factors for the development of Ludwig's angina include sublingual lacerations, penetrating injuries to the floor of the mouth, sialadenitis, compound mandibular fractures, osteomyelitis of the mandible, otitis media, infected malignancy and abscesses located under thyrohyoid membrane.[14]
3. Ludwig's angina in children has been described after infection of a lymphangioma and after tongue piercing.[19,20]

RISK FACTORS FOR THE DEVELOPMENT OF DEEP NECK INFECTIONS[1,2,11,12,21]

- Low socioeconomic status
- Poor oral hygiene
- Immune dysfunction (including HIV, diabetes, immunosuppression, patients on chemotherapy and on steroids)
- IV drug abuse
- Recent dental work
- Chronic or recurrent tonsillitis
- Alcohol abuse
- Cocaine abuse
- Systemic diseases—chronic glomerulonephritis, SLE, aplastic anemia

In patients with recurrent deep neck infections one should have a high suspicion for underlying congenital anomalies like 1, 2, 3, and 4 branchial cleft cysts, lymphangiomas, thyroglossal duct cysts and cervical thymic cysts.[22,23]

Microbiology[2,20,22-25]

Bacteria are often polymicrobial, with gram-positive organisms and anaerobes predominanting but gram-negative bacteria have also been isolated. The source is usually oropharyngeal flora.

Complications of Deep Neck Infections[1,2,7-11,13,24]

Patients at risk for complications are older patients and patients with systemic disease, including HIV/AIDS, myelodysplasia, cirrhosis, and diabetes. Huang et al found that 33 percent of diabetic patients had complications.[1]

1. The most urgent complication involves the abscess expanding against the pharynx or trachea with mass effect causing airway compression. In Ludwig's angina, soft tissue swelling in the suprahyoid region combined with lingual displacement and associated laryngeal edema can occlude the airway and abruptly asphyxiate the patient.[14]
2. Rupture of the abscess either spontaneously or during manipulation can cause aspiration of pus, resulting in asphyxiation, pneumonia or lung abscess.
3. Spread of the infection to the mediastinum can result in mediastinitis, purulent pericarditis and tamponade, pyopneumothorax, plueritis, empyema and bronchial erosion. Patients with—mediastinitis present with increased respiratory difficulty, tachycardia, chest pain, back pain, erythema/edema of the neck and chest, crepitus and shock. Hemodynamic instability may result from cardiac tamponade, and septicemia requiring the prompt institution of hemodynamic support, invasive monitoring and surgical intervention. A catastrophic caudad spread of a peritonsillar abscess with lateral pharyngeal space extension with tracheal deviation, bilateral pleural and pericardial effusion with airway compromise and septicemia has been reported.[21]
4. Spread of the infection to the carotid sheath can cause jugular vein thrombosis, carotid artery rupture or vocal cord paralysis. Jugular vein thrombophlebitis may manifest as tender induration at the anterior sternocleidomastoid border, or sepsis of an unknown source. Lemierre's disease, inwhich infection from the oropharynx extends to cause septic thrombophlebitis of the internal jugular vein and metastatic abscesses in the lungs has been described. The causative pathogen is *Fusobacterium necrophorum*, an anaerobic bacterial constituent of the oropharyngeal flora. The typical presentation is that of a previously healthy adolescent or young adult with a history of recent pharyngotonsillar disease who becomes acutely ill with fever and pulmonary symptoms. Chest radiography demonstrates multiple cavitary nodules, often bilateral, and often accompanied by pleural effusion.[26,27] Carotid artery rupture can be heralded by sentinel bleeding from the ear, nose or mouth. Ecchymosis may be detected in the lateral cheek.
5. Posterior spread of the infection to the vertebral canal can result in osteomyelitis and erosion of the spinal column, causing vertebral subluxation and spinal cord injury.[28]
6. The infection itself can evolve into necrotizing fasciitis, sepsis and death.
7. Horner's syndrome and 9 to 12 cranial nerve palsy due to involvement of lateral pharyngeal space have also been described.

Clinical Presentation[7-11,24]

- Constitutional symptoms such as fever, chills, malaise, decreased appetite and irritability
- Sore throat, dysphagia, odynophagia, otalgia, toothache, neck swelling or neck pain along with neck stiffness or torticollis
- Trismus due to spasm of pterygoid muscles is usually seen inpatients with peritonsillar, lateral pharyngeal or masticular space involvement
- Muffled, hyponasal or "Hot-potato" voice due to inflammation of posterior pharyngeal wall or palate is usually seen inpatients with retropharyngeal abscess, peritonsillar abscess or Ludwig's angina
- Parents and spouses may note worsening of snoring and sleep apnea
- Croupy cough, difficulty in breathing, inability to lie supine, drooling, inspiratory stridor, chest retraction, anxiety and cyanosis are ominous complaints that portend impending airway obstruction
- Dehydration due to fever and lack of oral intake
- Chronic retropharyngeal abscess due to tuberculous caries of the cervical vertebrae cause forward bulging of the posterior pharyngeal wall in the midline, in contrast to the acute retropharyngeal abscess in which the bulge is a little to one side of the midline due to distribution of the retropharyngeal lymph nodes
- The association of pharyngeal abscess with atlantoaxial subluxation and compression of brainstem resulting in quadriplegia and somnolence have been reported.[29]

Imaging Studies[10,11,13,30,31]

1. The radiographs of the neck and chest may demonstrate the extent of soft tissue swelling or gas in the tissue compatible with anaerobic infection (Fig. 21.3).
2. The lateral neck plain film is often enough to make the diagnosis of retropharyngeal abscess. Abscess cavity may be evident on lateral neck radiography

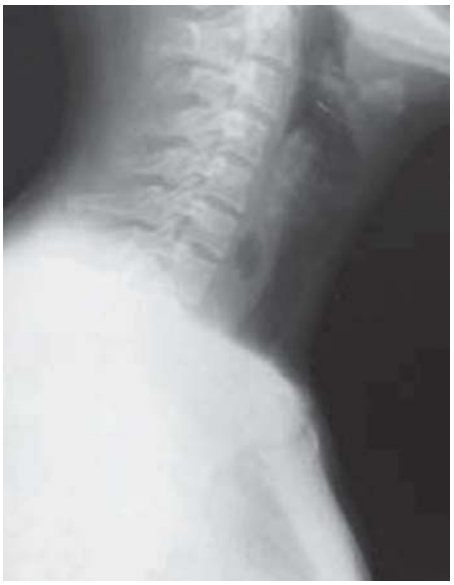


Fig. 21.3: Retropharyngeal abscess with air pocket within

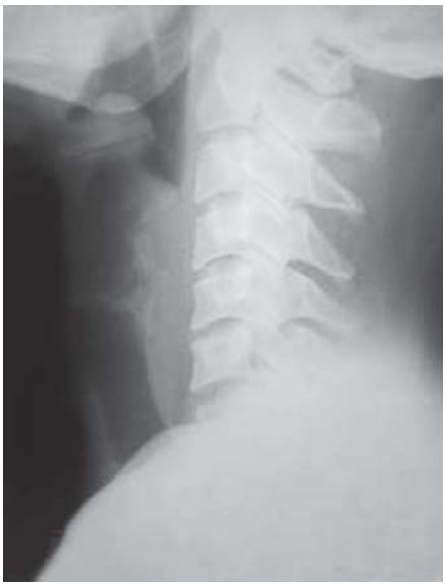


Fig. 21.4: Retropharyngeal abscess with loss of cervical lordosis

with anterior displacement of esophagus and upper pharynx (Fig. 21.4). The normal dimensions of the retropharyngeal space were defined by Wholey in 1958. The normal dimensions of the retropharyngeal space in both children and adults is between 4 to 7 mm measured from the most anterior aspect of C2 to the soft tissues of the posterior pharyngeal wall. The retrotracheal space measured from the anteroinferior aspect of C6 to the posterior pharyngeal wall should be no more than 14 mm in children and 22 mm in adults. Other useful radiological signs are loss of the normal cervical lordosis with straightening of the cervical spine and the presence of air or foreign body in the soft tissue. Roentgenographic evidence of caries of the cervical vertebrae may be seen.

3. A chest radiograph may demonstrate intrathoracic extension of the infectious process. Patients with mediastinitis will have a widened mediastinum superiorly, mediastinal emphysema and pleural effusion.
4. Ultrasound studies: It is particularly helpful in children and can identify location and size of the abscess and its relationship to surrounding structures.
5. Computed tomography: CT of the neck with contrast is the most used imaging modality because of its ability to delineate cellulitis versus abscess. Anatomic boundaries of the abscess can be defined along with involvement of airway and vascular structures.
6. Magnetic resonance imaging (MRI): It is superior in assessing the origin of infection and also has decreased interference from dental artifacts. Also, MRI decreases toxic exposure to radiation and iodine based contrast. It may be indicated to define abscess extension in difficult cases. It has the potential to delineate any complications more accurately, such as internal jugular vein thrombosis. But is more expensive and requires good patient cooperation.

Management[2,10,11,32]

There are four principle components in the treatment of deep neck infections:

1. Adequate nutrition and hydration—especially in patients with significant oropharyngeal edema.
2. Airway management.
3. Antibiotic therapy.
4. Surgical therapy.

The mainstay of treatment for deep neck abscesses has been for decades, open surgical incision and drainage. In the last few years, some authors have proposed antibiotic therapy alone or needle aspiration as an alternative to surgical incision and drainage in cases of small abscesses. Paola et al recommend treating all patients with broad-spectrum intravenous antibiotics until culture results identify the causative organism.

In cases of large abscesses and/or multiple space involvement, an open surgical incision and drainage is promptly performed.

In cases of small abscesses, watch and wait for 48 hours and in cases of easily reachable abscesses, perform needle aspiration, eventually CT guided for culture and drainage.

If lack of response to conservative treatment is clinically and radiologically noted after 48 hours, the patient is treated with an open surgical drainage. The choice between external and intraoral approach mainly depends on the size and site of the abscess and its relationship with the great vessels of the neck and the number of the neck spaces involved. Transoral approaches have been shown to be safe in patients with retropharyngeal, lateral pharyngeal and prevertebral abscesses that are medial to the great vessels. Lesions that extend lateral to great vessels should be approached externally. Similarly, for more serious infection and abscesses that have spread to multiple spaces external drainage is necessary.

Peritonsillar abscesses are most commonly managed by incision and drainage or by needle aspiration. Most children undergo general anesthesia for treatment of peritonsillar abscess by incision and drainage.

In older children and adolescents, it is possible to perform incision and drainage of peritonsillar abscess with intravenous sedation using an opioid and topical anesthesia with the patient in a head down position and head turned to the side of the abscess.[33] Most commonly, interval tonsillectomy is performed 4 to 12 weeks after resolution of the infection. Quinsy tonsillectomy is indicated, if the abscess is small and well confined and in children who do not tolerate awake needle aspiration and who have indications for tonsillectomy such as history of recurrent tonsillitis.[13]

Treatment of descending necrotizing mediastinitis should include some sort of drainage procedures along with IV antibiotics. Consultations with thoracic surgeon should be obtained. Access to superior mediastinum from a cervical incision is adequate for fluid collections above the tracheal bifurcation. Transthoracic drainage should be performed for abscesses that extend below T4. Abscesses in the anterior mediastinum may be approached by a subxyphoid incision. Thoracostomy tubes should be placed for pleural effusions.[11,21,31,32]

Anesthetic Management

Preanesthetic Evaluation

1. History—with special stress:
   a. To know the etiology
      - URTI
      - Recent dental procedures, toothache
      - Neck or oral cavity trauma
      - Tuberculosis
      - High risk population.
   b. To know the presence of airway compromise
      - Ability to lie supine
      - Sleep position
      - Change in voice quality
      - Respiratory difficulties
      - Intolerance to own secretions
      - Croupy cough
      - Inability to phonate or talk in monosyllables.
   c. To know the presence of complications
      - Weakness in upper limbs and/or lower limbs
      - Persistence of fever inspite of antibiotics
      - Sentinel bleeding from ear/nose or mouth
      - Chest pain/breathlessness.
2. Physical examination—with special attention to:
   a. Potential airway problems and airway assessment including the signs of airway compromise. Tachypnea, stridor, tracheal tug, intercostals indrawing and use of accessory muscles suggest presence of airway obstruction. Before surgery, full assessment of the airway is necessary. Fixed anatomic airway parameters predictive of a difficult intubation must be assessed. Facial, neck and dental abnormalities including the presence of face or neck swelling, limited neck and jaw movements, limited mouth opening, protruding maxillary incisors, lack of visualization of pharyngeal structures and tongue edema were found to predict a difficult tracheal intubation. The presence of pus and secretions in the oral cavity may complicate the airway management.[34] Stridor indicates critical airway obstruction with more than 50 percent reduction in airway diameter and in adults an airway diameter of 4 to 5 mm.[35] High airway pressure is required to create flow in patients with stridor which may not be possible with bag mask ventilation. Hence, bag-mask ventilation is likely to be difficult in patients with stridor. The presence of secretions may precipitate laryngospasm at the time of induction. The presence of tongue swelling precludes the use of oral airway and also suggests the likely presence of glottic edema in which case supraglottic devices will not be of much use. Tongue swelling and oral secretions make direct laryngoscopy difficult irrespective of the type of laryngoscopic blade used.[36]
   b. Neurological examination—in patients with atlantoaxial instability, involvement of cranial nerves and Horner's syndrome.
   c. Rule out presence of complications like pleural effusion, pericardial effusion and pneumothorax.
   d. Vital parameters—hemodynamic instability may suggest cardiac tamponade or septicemia.
   e. Dehydration from lack of oral intake may be present.

The assessment must also include reviewing the relevant X-rays and CT scans with the surgeon to determine the extent of soft tissue swelling and airway compromise. The lateral X-ray neck, CT scan and nasopharyngoscopy can assess the dynamic aspect of airway obstruction.[36] After complete assessment, the surgeon and anesthesiologist must jointly decide on the best method of securing the airway.

Airway management options will depend on the clinical severity of the disease, including the presence of trismus and airway obstruction, surgical preference, the urgency of situation, available resources, skill and experience of anesthesiologist, ability of the patient to cooperate and other factors (e.g. CT scan findings). For example, if the oropharynx cannot be visualized by CT, a fiberoptic nasotracheal approach is advised.[14]

Investigations: As per the age of the patient and associated morbidities.

General Considerations for Anesthetic Management

1. Monitoring: Cardioscope, pulse oximeter, capnometer, manual blood pressure and peripheral nerve stimulator. Consider more invasive monitoring like CVP and arterial line if impending sepsis or if significant comorbidities exist. If central access is desired potential for jugular vein thrombosis which may make line placement difficult if not impossible must be kept in mind.[37]
2. Preanesthetic medication: Consider
   a. H_2 receptor antagonists and prokinetic drugs to reduce the risk of aspiration.
   b. Anticholinergic drugs to reduce secretions.
   c. Nebulized adrenaline to reduce mucosal swelling especially in patients with Ludwig's angina is recommended by few authors.[38] This is not practiced in our institute.
   d. No sedative premedication should be given in patients with pre-existing or potential airway obstruction.
3. The pharyngeal swelling, the distortion of normal anatomy and excess mouth secretions may make laryngoscopy and intubation difficult. Hence, the operation theater should be prepared with fully tilting table, fully checked anesthesia machine, all available equipment for difficult intubation, emergency drugs, two adequate and functioning Yankauer suction cannulas and appropriate monitoring.[39]
4. Whenever, it is decided to manage the airway non-surgically, following options are available: Awake vs. asleep and oral vs. nasal. While attempting awake intubation, sedation may be necessary in some cases. However, it is preferred to keep the patient cooperative and spontaneously breathing. Sudden deterioration may occur even with the use of minimal or no sedation. Oral intubation is usually preferred over nasal intubation unless it appears impossible due to massive tongue edema or presence of trismus as it avoids the risk of nasal bleeding and endotracheal tube size will only be limited by the size of glottis. Whichever technique is chosen, keep plan B and sometimes plan C ready.[36]
5. Double setup: The surgeon must be present in the OT fully prepared for immediate tracheostomy. Surgical airway represents plan B. However, surgical access could prove difficult. Loss of airway during tracheostomy has been reported and such an eventuality must be prepared for with plan C. This will typically involve sedation (IV or inhalation) to facilitate patient cooperation, but in rare circumstances could require muscle relaxation and an attempt at direct laryngoscopy while attempts at obtaining surgical access continue.[36]
6. The most skilled and experienced staff must be available for anesthetic management of difficult airway.
7. Rupture of the abscess and possible aspiration of pus during the induction of anesthesia should be avoided. Some authors recommend preoperative decompression and drainage of the peritonsillar abscess by needle aspiration to decrease the risk of abscess rupture.[40]
8. To avoid aspiration of pus during intubation and drainage, the patient may be positioned in a slightly head down position and a cuffed endotracheal tube should be inserted carefully without touching the abscess.
9. The inherent danger in using an intravenous induction is apnea in a patient with a compromised airway and inability to intubate or ventilate the patient.[41] In such a scenario, an induction technique that maintains spontaneous respiratory drive until the efficacy of assisted mask ventilation is established or until tracheal intubation is performed is preferred.
10. After the abscess is drained, the surgeon irrigates and suctions the oral cavity multiple times in case of intraoral approach for the drainage of peritonsillar and retropharyngeal abscess.[29] The cuffed endotracheal tube and throat pack decreases the chances of aspiration. However, packing must be done with extreme caution and gentleness to avoid rupturing the access.

11. These cases are not lengthy and an awake extubation is indicated.[29] In patients where the induration is more than the pus with associated airway compromise (e.g. Ludwig's angina), extubation may need to be deferred.
12. In patients with pharyngeal abscess with atlantoaxial instability, meticulous attention should be given to cervical spine integrity while securing the airway with head held in neutral position by an assistant.
13. Owing to the potential for obstruction of the PVC tracheal tube due to compression from surrounding structures, it seems prudent to insert an armoured tube to better protect the airway, especially when upper airway obstruction is managed with fiberoptic intubation.[14,38,42]
14. Any soft tissue infection of the floor of the mouth has the potential to spread along the fascial planes of the neck. Descending necrotizing fasciitis with mediastinal, pulmonary and cardiac involvement is a rare but potentially fatal complication of Ludwig's Angina. It is therefore advisable to avoid non-steroidal anti-inflammatory drugs given their possible association with necrotizing fasciitis.[38]

MANAGEMENT OF AIRWAY

A review of the literature shows that a variety of techniques have been advocated for managing the airway. These include awake tracheostomy under local anesthesia, awake fiberoptic laryngoscopy, awake blind nasal intubation, and intravenous or gaseous induction followed by laryngoscopy and intubation. Blind nasal intubation is to be avoided as besides having a high failure rate, it could cause catastrophic bleeding, abscess perforation with soiling of the airway, airway edema and laryngospasm. Complete airway obstruction could be precipitated, potentially necessitating an emergency cricothyrotomy.[6,36,38,42]

1. If there is minimal trismus and the airway assessment indicates minimal distortion, a rapid sequence intravenous induction after adequate preoxygenation may be the best way to avoid trauma to the pharyngeal structures while struggling with a mask induction, needing to insert an oropharyngeal airway and possibly causing the abscess to rupture.[39] If the abscess is right sided, a left-sided approach for laryngoscopy may be indicated.
2. If trismus is present without any signs or symptoms of airway compromise, a slow inhalational induction performed with sevoflurane and oxygen allows the anesthesiologist to assess the jaw relaxation under anesthesia. Sevoflurane allows rapid control of anesthetic depth with cardiovascular stability and the maintenance of spontaneous breathing.[38] It is important to maintain the maximum possible inspired oxygen percentage throughout the induction. Nitrous oxide should not be used. Usually, the apparent trismus often resolves with adequate depth of anesthesia. When this is confirmed or if trismus was minimal to begin with, a short acting muscle relaxant is given to allow for an atraumatic laryngoscopy and intubation after confirming ventilation. Another alternative is to perform tracheal intubation without muscle relaxation under adequate depth of anesthesia.
3. Inhalational induction in adults with airway obstruction may be more difficult in that the relatively longer excitement phase predisposes to aspiration, laryngospasm or both. As anesthetic depth increases complete airway obstruction can also occur due to loss of muscle tone. The preservation of spontaneous ventilation has been shown to be difficult when inhalational agents are used to achieve adequate depth of anesthesia.
4. Remifentanil may be a more attractive IV sedation option.[36] Machata et al have found excellent intubating conditions with Remifentanil when used for conscious sedation in a dose of 0.75 µg/kg followed by the infusion of 0.075 µg/kg/min.[43] The authors do not have any experience with Remifentanil.
5. In advanced cases, the induction of general anesthesia is dangerous because it may precipitate complete airway closure and make face mask ventilation and tracheal intubation impossible, thus necessitating emergency tracheostomy.[6] Elective awake tracheostomy was considered the gold standard of airway management in patients with deep neck infections in order to avoid the dangers of emergency tracheostomy in a severely compromised airway. This notion has been questioned for few years.[44] Although bypassing the supraglottic swelling, tracheostomy is itself associated with a high complication rate. Tracheostomy conveys substantial risk of infection spread to the mediastinum and tracheal stenosis as a late complication. Aspiration of pus, rupture of the innominate artery, spread of infection to the thorax, airway loss and death have been reported. Surgical access is likely to be difficult in patients with a severely compromised airway, distorted anterior neck anatomy and the patient's inability to lie supine. However, in cases with complete upper

airway obstruction, emergency tracheostomy may be the only way to prevent death by hypoxia. Cricothyroidotomy with jet ventilation could be an alternative but in the presence of the severe distortion of the anatomy, it might prove very difficult.[6,38]

6. Awake fiberoptic laryngoscopic intubation has been widely recommended as a first line approach in upper airway obstruction.[6,44] Although tissue edema and immobility, a distorted airway and copious secretions contribute to the difficulty of fiberoptic intubation, it has a high success rate. It is possible to see "around the obstruction" and is well tolerated by awake patients. Fiberoptic bronchoscope does not induce pain, can be applied through oral or nasal routes and can be used in any position comfortable to the patient.

Nasal bleeding can be decreased by one of the following techniques:

- Liberal topical vasoconstrictors like xylometazoline
- Small endotracheal tube
- Reinforced endotracheal tube
- Soften the regular endotracheal tube by putting in warm saline
- Dilating the nasal passage.[36]

Topical anesthesia of the larynx and trachea can be achieved with the spray-as-you-go technique. Airway blocks are contraindicated due to poor anatomic landmarks, presence of infection and risk of aspiration. The topical anesthesia of the larynx is achieved within 1 minute after lidocaine spray. A shorter time interval between application of topical anesthesia and tracheal intubation lessens the potential of aspiration. Inflammation and infection can in theory decrease the efficacy of local anesthetics due to changes in local pH. However, this is generally not of any clinical significance. Heavy secretions, bleeding or both can decrease the amount of local anesthetic that actually reaches the mucosa and this should be taken into account.

Topical anesthesia can also be achieved by applying approximately 2 cm of 5 percent Lidocaine ointment to the back of the tongue with a tongue depressor. The De Vilbiss atomizer may be used to deliver 15 ml of 0.45 percent tetracaine as an aerosol.[36]

An analysis and review by Ovassapian et al concluded that an awake fiberoptic intubation is the first choice for airway control in adult patients with advanced DNSI. If an awake fiberoptic intubation is not feasible, an awake tracheostomy under local anesthesia should be preferred.[6]

In summary, death from loss of an airway still occurs in patients with advanced DNSI. Securing such an airway is challenging and dangerous and must be a joint venture involving surgeon and anesthesiologist. Sound clinical judgment is critical for timing and for selecting the method for airway intervention. The choice of airway maneuvers must be individualized, depending on the judgment and experience of the physician in charge.

REFERENCES

1. Coelho MS, Ramos G, Prestes LC, Soccol A, Oliveira MS, Lobo P. Deep Neck Infections – Classification in levels of severity. International Archives of Otolaryngol 2009; 13(2):184-8.
2. Boscolo-Rizzo P, Marchiori C, Montolli F, Vaglia A, Da Mosto MC. Deep neck infections: A constant challenge. Otorhinolaryngol 2006;68:259-65.
3. Kremer MJ, Blair T. Ludwig's Angina – forewarned is forearmed. AANA Journal 2006;74(6):445-51.
4. Baba Y, Kato Y, Saito H, Ogawa K. Management of deep neck infection by a transnasal approach: a case report. Journal of Medical Case Reports 2009, 3: 7317 doi: 10.4076/1752-1977-3-7317. Published 31 July 2009. Available from: http://www.jmedicalcasereports.com/content/3/1/7317.
5. Heindel DJ. Deep neck abscesses in adults: Management of a difficult airway. Anesth Analg 1987;66:774-6.
6. Ovassapian A, Tuncbilek M, Weitzel EK, Joshi CW. Airway management in adult patients with deep neck infections: A case series and review of the literature. Anesth Analg 2005;100:585-89.
7. Levitt GW. Cervical fascia and deep neck infections. Otolaryngol Clin. North Am 1976;9:703-16.
8. Paonessa DF, Goldstein JC. Anatomy and physiology of head and neck infections (with emphasis on the fascia of the face and neck). Otolaryngol Clin North Am 1976;9(3): 561-80.
9. Lindner HH. The anatomy of the fasciae of the face and neck with particular reference to the spread and treatment of intraoral infections (Ludwig's) that have progressed in to adjacent fascial spaces. Ann Surg 1986;204:705-14.
10. Goldenberg D, Golz A, Joachims HZ. Retropharyngeal abscess: a clinical review. The Journal of Laryngology and Otology 1997;111:546-50.
11. Weed HG, Forest LA. Deep neck infection. In: Cummings CW, Flint PW, Harker LA, Haughey BH, Richardson MA, Robbins KT, Schuller DE, Thomas JR, (Eds). Cummings Otolaryngology Head and Neck Surgery. Vol 3. 4th edn. Philadelphia: Elsevier Mosby 2005;2515-24.
12. Velankar H. Retropharyngeal abscess. Bombay Hospital Journal 2001;43(3):371-3. Available from http://www.bhj.org/journal/2001_4303_july01/review_371.htm.
13. Nicklaus PJ, Kelley PE. Management of deep neck infection. Pediatric Clinics of North America 1996;43(6): 1277-94.

14. McGoldrick KE. Eye, ear, nose and throat diseases. In: Fleisher LA, (Eds). Anesthesia and uncommon diseases. 5th edn. Philadelphia: Saunders Elsevier 2006;23-4.
15. Fritsch DE, Klein DG. Curriculum in critical care: Ludwig's angina. Heart Lung 1992;21:39-47.
16. Moreland LW, Corey J, McKenzie R. Ludwig's angina report of a case and review of the literature. Arch Intern Med 1988;148(2):461-6.
17. Patterson HC, Kelly JH, Strome M. Ludwig's Angina: An update. Laryngoscope 1982;92:370-6.
18. Hamza NS, Farrel J, Strauss M, et al. Deep fascial space infection of the neck: a continuing challenge. South Med J 2003;96:928-32.
19. Tasca RA, Myatt HM, Beckenham EJ. Lymphangioma of the tongue presenting as Ludwig's angina. Int J Pediatr Otorhinolaryngol 1999;51(3):201-5.
20. Perkins CS, Meisner J, Harrison JM. A complication of tongue piercing. Br Dental J 1997;182:147-8.
21. Manecke GR, Marghoob S, Finzel KC, Madoff DC, Quinjano IH, Poppers PJ. Catastrophic caudad spread of a peritonsillar abscess: a case report. Anesthesiol 1999; 91:156-8.
22. Stalfors J, Adielsson A, Ebenfelt A, Ethander G, Westin T. Deep Neck Space Infections Remain a Surgical Challenge. A study of 72 patients. Acta Otolaryngol 2004;123:1191-6.
23. Parhiscar A, Har-El G. Deep neck abscess: a retrospective review of 210 cases. Annals of Otology, Rhinology and Laryngology 2001;110(11):1051-4.
24. Pappas DE, Hendley JO. Retropharyngeal abscess, lateral pharyngeal (parapharyngeal) abscess, and peritonsillar cellulitis/abscess. In: Behrman RE, Kleigman RM, Jenson HB, (eds). Behrman: Nelson textbook of pediatrics. (17th edn). Philadelphia: Saunders Elsevier 2004;1394-6.
25. Dryden M, Dalgltesh D. Pastuerella multocida from a dog causing Ludwig's angina (letter). Lancet 1996;347:123.
26. Golpe R, Marin B, Alonso M. Eponyms in medicine revisited. Lemierre's syndrome (necrobacillosis). Postgrad Med J 1999;75:141-4.
27. Lustig LR, Cusick BC, Cheung SW, Lee KC. Lemierre's syndrome: Two cases of postanginal sepsis. Otolaryngol Head Neck Surg 1995;112(6):767-72.
28. Brookes A, Moriarity A. Pharyngeal abscess presenting with upper airway obstruction and atlantoaxial subluxation in a small infant. Anesthesia 2000;55:469-74.
29. Ragan B. Anesthesia for otorhinolaryngolic (Ear, Nose, and Throat) Surgery. In: Longnecker DE, Brown DL, Newman MF, Zapol WM, (Eds) Anesthesiology. New York: McGraw Hill 2008;1590-1.
30. Philpott CM, Selvadurai D, Banerjee AR. Pediatric retropharyngeal abscess. The Journal of Laryngology and Otology 2004;118:919-26.
31. Grodinsky M. Retropharyngeal and lateral pharyngeal abscesses. Annals of Surgery 1939;110(2):177-99.
32. Collin J, Beasley N. Tonsilitis to mediastinitis. The Journal of Laryngology and Otology 2006;120:963-6.
33. Smith-Landsman IS, Werkhaven JA, Motoyama EK. Anesthesia for Pediatric otorhinolaryngologic surgery. In: Motoyama EK, Davis PJ, (Eds). Smith's Anesthesia for infants and children. (7th edn). Philadelphia: Mosby Elsevier 2006;789-822.
34. Cozine K, Gilbertstone J. The take back patient in ear, nose and throat Surgery. Anesth Clinics of North America 1993; 11:655.
35. Feldman MA, Patel A. Anesthesia for eye, ear, nose and throat surgery. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL, (Eds). Miller's Anesthesia (7th edn). Philadelphia Churchill Livingstone; Elsevier 2010;2357-88.
36. MacQuarrie KJ, Kirckpatrick. Airway management in a patient with retropharyngeal abscess. In: Hung O, Murphy MF, (Eds) Management of the difficult and failed airway. New York: McGraw Hill Medical 2008;283-8.
37. Penner J. Ludwig's Angina. In: Roizen MF, Fleisher LA, (Eds) Essence of Anesthesia practice, (2nd edn). Saunders Philadelphia 2002;210.
38. Neff SPW, Merry AF, Anderson B. Airway management in Ludwig's angina. Anesth Intensive Care 1999:27:659-61.
39. Verghese ST, Hannallah RS. Pediatric otolaryngologic emergencies. Anesthesiology Clin N Am 2001;19:237-56.
40. Dougherty TB. The difficult airway in conventional head and neck surgery. In: Benunof JL, (Ed). Airway management: principles and practice. St. Louis: Mosby Publishers 1996;686-97.
41. Loughnan TE, Allen DE. Ludwig's Angina. The Anesthetic management: of nine cases. Anesthesia 1985;40:295-7.
42. Chung RA, Liban JB. Ludwig's angina and tracheal tube obstruction (letter). Anesthesia 1991;46:228-9.
43. Machata AM, Gonano C, Holzer A, et al. Awake nasotracheal fibreobtic intibuation: patient comfort, intubating conditions and hemodynamic stability during conscious sedation with remifentanil. Anesth Analog 2003;97:904-8.
44. Kulkarni AH, Pai SD, Bhattarai B, Rao ST, Ambareesha M. Ludwig's angina and airway considerations: a case report. Cases Journal 2008, 1: 19 doi: 10.1186/1757-1626-1-19. 2008 June. Available from http://www.casesjournal.com/content/1/1/19.

22 Anesthesia for Removal of Foreign Body from Airway and Upper Digestive Tract

Anila D Malde, Swati S Chhatrapati

KEY POINTS

- Aspiration of foreign body accounts for an important cause of morbidity and mortality in children between 1 and 3 years of age.
- Vegetable foreign bodies like nutty substances or plants are more dangerous as they can be easily fragmented during removal and cause gradual mechanical obstruction and pneumonitis.
- In adults, the foreign body aspiration occurs more commonly in the right main bronchus than the left. However, in the pediatric age group, both right and left bronchi are equally susceptible for foreign body aspiration.
- Anesthesia for bronchoscopy to remove a foreign body from the airway is a challenging procedure for the anesthesiologist who must share the airway with the bronchoscopist and maintain adequate depth of anesthesia. Avoiding mishaps demands skill, communication and teamwork between the endoscopist and anesthesiologist.
- The severity of respiratory embarrassment following foreign body aspiration depends on the size, location, and nature of the foreign body. Distal foreign bodies are more difficult to remove whereas proximal ones are more likely to obstruct the airway.
- Complete airway obstruction demonstrates atelectasis whereas partial airway obstruction demonstrates obstructive emphysema on the affected side on the chest radiograph.
- The guiding principle of treatment should be "Do not convert a partial airway obstruction into a complete airway obstruction".
- The surgeon must be prepared to perform an emergency tracheostomy if partial obstruction suddenly becomes complete intraoperatively.
- The urgency to proceed with anesthesia is dictated by severity of respiratory distress and the location and nature of the aspirated material.
- For laryngotracheal foreign bodies, inhalational induction and maintenance of spontaneous breathing is preferred to prevent displacement of the foreign body and further obstruction of the airway.
- For bronchial foreign bodies, both the induction of anesthesia by either inhalational or intravenous (IV) agents are acceptable.
- Both, spontaneous and controlled ventilation techniques have been used successfully for bronchoscopic removal of the foreign body. There is no strong evidence for choosing one technique over another.
- Ventilation during bronchoscopy in a paralyzed patient can be carried out by means of apneic oxygenation, high frequency positive pressure ventilation, a ventilating bronchoscope, or a venturi injector device.
- Any rapid deterioration of cardiac function during bronchoscopy should make one highly suspicious of tension pneumothorax.
- Treatment of postoperative laryngeal edema and stridor include administration of humidified oxygen, steroids, nebulized racemic adrenaline and rarely, reintubation for 1 or 2 days.
- Endotracheal intubation and airway protection should precede foreign body extraction from the esophagus under general anesthesia.
- In the absence of airway obstruction, it is advisable to follow the fasting guidelines before removal of foreign body from the upper digestive tract.

FOREIGN BODY ASPIRATION

Foreign body aspiration is a common problem in children and accounts for an important cause of morbidity and mortality. It is potentially a life-threatening event and may also cause chronic lung injury if not properly managed.[1] Most deaths occur at the time of aspiration. In most series, the mortality is zero for patients who reach the hospital alive.[2]

The diagnosis and treatment of the problem requires awareness and highest degree of suspicion of signs and symptoms of foreign body aspiration. In the absence of a history of trauma or infection, the onset of respiratory distress in a toddler who has no underlying airway abnormalities should raise suspicion of an acute aspiration of a foreign body.[3]

Foreign body aspiration occurs commonly in children between 1 and 3 years of age who often place small objects in their mouths and who lack molars for grinding hard food. It consists most frequently of peanuts seeds and other food particles and less frequently of plastic and metal particles.[3] In adults the aspiration of fish or chicken bones or aspirations resulting from dental surgery accidents are more common.[4] Vegetable items (nutty substances or plants) are more dangerous than other kinds of foreign bodies. They swell with the bronchial secretions, thus causing gradual mechanical obstruction, and they also cause allergic and chemical bronchitis (so called "Vegetable Bronchitis") as the result of absorption of antigenic proteins, organic acids and oils which they contain.[5]

Where the foreign body lodges in the airway depends on its size and shape. In adults, it occurs commonly in the right main bronchus than the left. This is because of anatomical position of the right main bronchus, as it is more vertical and has larger diameter leading to more air entry than the left bronchus.[1,4]

However, in the pediatric age group, the aspirated foreign bodies lodge frequently in the proximal airways due to smaller bronchial tree diameter and both right and left bronchi are equally susceptible for foreign body aspiration.[4]

Types of Bronchial Obstruction

Four types of bronchial obstructions by foreign bodies have been described by Chatterji and Chatterji[6] (Fig. 22.1):

1. The check valve mechanism, by which air is inhaled but cannot be expelled, causes unilateral hyperaeration (emphysema) on the affected side.
2. The bypass valve mechanism, caused by partial obstruction of the bronchial lumen, results in diminished aeration and opacity on the affected side.
3. The stop valve mechanism, caused either by a large foreign body or by a gradually swollen small foreign body leads to distal atelectasis.
4. The ball valve mechanism, by which the foreign body dislodges during expiration and reimpacts during inspiration, leads to early atelectasis on the affected side.

The gold standard for diagnosis and management of foreign body aspiration is rigid open tube bronchoscopy under general anesthesia.[2]

Specific Problems of Rigid Bronchoscopy[7-10]

1. There is competition between the bronchoscopist and anesthetist for control of the airway.
2. Instrumentation of the respiratory tract is a potent cause of bronchospasm, laryngospasm, dysrhythmias and rise in the intrathoracic and cerebrospinal fluid pressure.
3. It is often difficult to maintain adequate ventilation and oxygenation in these patients. Bronchoscopy interferes with the gas exchange by reducing alveolar ventilation and altering ventilation/perfusion relationships. Ventilation can be further impaired during the procedure if a lobar bronchus is obstructed by the bronchoscope. The rate of suction is important as a suction of 12.5 cm H_2O may reduce the tidal volume by 40 percent. Hence, suctioning must be intermittent and short.
4. The procedure is sometimes indicated as an emergency on an unprepared patient with full stomach

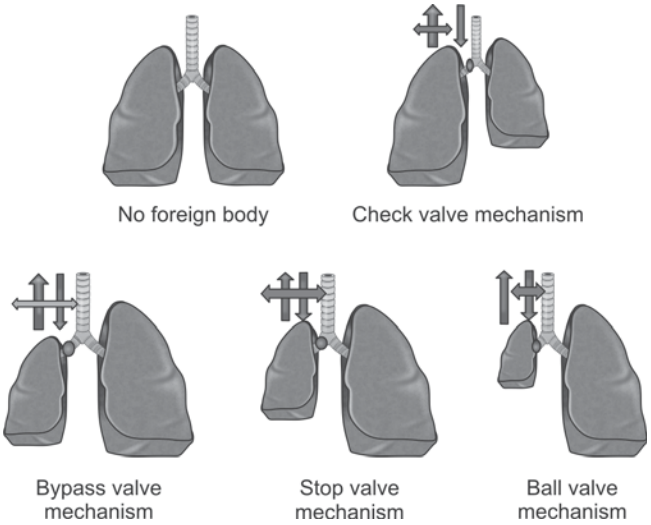


Fig. 22.1: Bronchial foreign body obstruction: Mechanisms
Downward pointing arrows: Inspiration; Upward pointing arrows: Expiration; Double sided horizontal empty arrow: Partial obstruction; Double sided horizontal filled arrows: Complete obstruction *(For color version, see Plate 2)*

and who has impaired cardiovascular or respiratory function. An increase in catecholamine levels and hypoxia produce a rise in cardiac output, heart rate, blood pressure and pulmonary artery pressure by 30 to 50 percent. This may be hazardous in patients with coronary artery disease. Major dysrhythmias may occur in 4 to 11 percent of patients. Though atrial dysrhythmias are well- tolerated, ventricular dysrhythmias may be life- threatening as they are commonly the result of hypoxia.

5. There is increase in airway pressure and work of breathing due to small caliber of bronchoscope. There is obstruction to expiratory flow during suctioning, while insertion of telescope or foreign body forceps which ultimately may cause barotraumas.
6. The problems are compounded in children due to small size of the glottis, an even smaller lumen of the bronchoscope, low functional residual capacity resulting in poor tolerance to apnea, a very high vagal tone and increased susceptibility to all potential dysrhythmias.
7. It is often difficult to maintain an adequate depth of anesthesia during the procedure, as there is a constant leak of anesthetic gases through the proximal end and around the bronchoscope.

Goals of Anesthesia for Bronchoscopy[7,11]

1. Maintenance of adequate ventilation and oxygenation while maintaining a clear view and appropriate access for the bronchoscopist.
2. Adequate depth of anesthesia, amnesia, analgesia and sufficient muscle relaxation to allow easy passage of the instrument with abolition of cardiorespiratory reflexes.
3. Prevention of pulmonary aspiration.
4. Quick return of consciousness, respiratory drive and upper airway reflexes at the end of the procedure.
5. To allow longer time duration for the procedure.
6. Minimization of secretions.

Preoperative Evaluation[2,12-14]

An assessment must be made of the location, suspected type and degree to which the foreign body is obstructing the airway because these factors influence the approach for removal and thus the anesthesia technique. Distal foreign bodies are more difficult to remove whereas proximal ones are more likely to obstruct the airway. Signs of airway obstruction include obvious distress, stridor, tachypnea, nasal flaring and chest retraction.

Presenting symptoms of an inhaled foreign body depends on time since aspiration. The child with a foreign body in the airway usually presents with a history of a choking episode, the aptly named "penetration syndrome". If the child is unable to give history or if the episode is not witnessed, it may not be identified. Also, the initial symptoms of inhaled foreign body like coughing, wheezing, or raspy breathing may be missed in these cases.

One must look for signs of airway obstruction, quantify the respiratory distress, note the respiratory rate, characteristics of breathing (such as stridor, retractions), presence or absence of cyanosis, anxiety and finally evaluate the child's sensorium.

The severity of respiratory embarrassment following aspiration of a foreign body depends on its location and nature. A foreign body with sharp edges can tear the mucosa resulting in pneumothorax, pneumomediastinum or subcutaneous emphysema. Also, sharp objects cause bleeding into the airway in addition to the obstruction. Encapsulated dry vegetable substances such as beans can swell in the presence of moisture and cause respiratory embarrassment in previously asymptomatic child. Also, it can create difficulties in delivering the foreign body in one piece. This type of foreign body must be broken into smaller pieces to avoid total obstruction of the trachea when attempts are made to remove it intact from the bronchus. Close observation is necessary when breaking up the foreign body to prevent some of the pieces from occluding the originally unobstructed bronchus.

If possible, attempts should be made to determine whether the foreign body is in the larynx, trachea or bronchus. Laryngeal foreign bodies are always symptomatic and are more likely to cause airway obstruction. Prominent stridorous breathing is indicative of supraglottic or glottic involvement. Patients with foreign body within the larynx and trachea present with acute dyspnea, stridor, coughing, and cyanosis. Wheezing is heard more often with subglottic obstruction.[15] Laryngeal obstruction may cause aphonia or dysphonia. A normal voice with a brassy cough and bidirectional stridor occur with tracheal foreign body.[14]

Expiratory wheezing, pneumonia, atelectasis, chest pain or cough suggests a distal location. In these cases physical findings may include asymmetric breath sounds with decreased breath sounds in the post-obstructive areas and adventitious sounds like rhonchi or crepitations. Foreign bodies located in the bronchi may dislodge from cough or change in position and cause total airway obstruction. Therefore, prompt removal is important.

Fever and the symptoms and signs of a chest infection are typical presenting symptoms in those who are first seen more than 24 hours after aspiration. In the presence of longstanding aspirated foreign bodies, the possible complications include pneumonitis, atelectasis, emphysema, massive hemoptysis, lung abscess and bronchiectasis. This means that if there is no acute obstruction, then the situation is unlikely to be life-threatening. Thus, there is usually time to fast the child, perform chest X-ray, and if necessary, to arrange referral and management by a specialty team. However, late diagnosis is associated with a higher complication rate.[2]

The child who has been previously endoscoped is likely to have laryngeal edema. This child is likely to benefit with antibiotics, humidified air and steroids, all of which should be started in the preoperative period.[2,12]

Radiological Findings[3,16-18]

All patients suspected of a foreign body in the airway should have posteroanterior and lateral chest films and a lateral soft tissue neck radiograph.

Because the majority of inhaled objects are radiolucent, secondary signs of complete or partial airway obstruction give clues as to their location on X-ray. When bronchial obstruction is complete, air is absorbed distal to the obstruction and atelectasis is seen on chest radiography. Most commonly, the bronchial obstruction is partial; air can enter around the foreign body on inspiration when the bronchus normally dilates, but during expiration with associated bronchial constriction, exhalation is completely obstructed because the foreign body acts like a unidirectional valve. Therefore, X-ray findings will be of obstructive emphysema with unilateral hyperaeration, a depressed and flattened diaphragm, and mediastinal shift away from the affected side (Fig. 22.2). Chest radiographs should be obtained during both inspiration and expiration, since the latter are more likely to demonstrate obstructive emphysema (overinflation) when compared with the opposite lung. Fluoroscopy has also been advocated for diagnosis by observation of mediastinal swing during rapid (crying) respiration.

However, it is not uncommon to have normal chest X-ray within the first 24 hours following aspiration. Despite a normal radiographic appearance, a positive history plus clinical symptoms of aspiration are sufficient to justify endoscopy for diagnosis and retrieval of the foreign body.

Pneumothorax may be present due to spontaneous rupture of hyperaerated peripheral alveoli or a sequelae to the pneumomediastinum. Disruption of the bronchial wall can lead to mediastinal emphysema. The presence

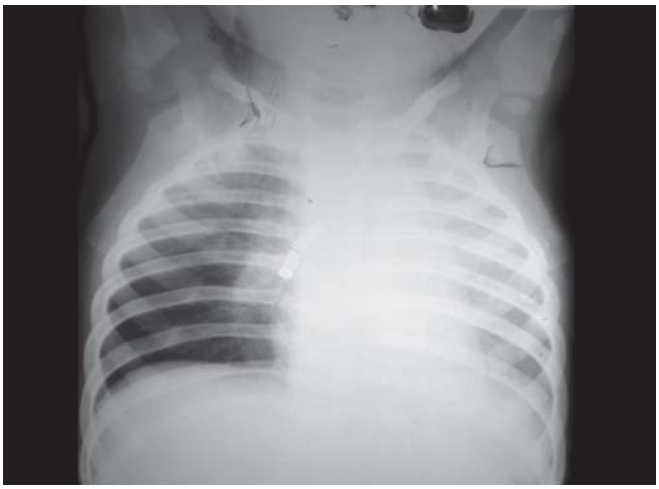


Fig. 22.2: Bronchial foreign body obstruction: Right main bronchial spring causing obstructive emphysema of right lung in a child

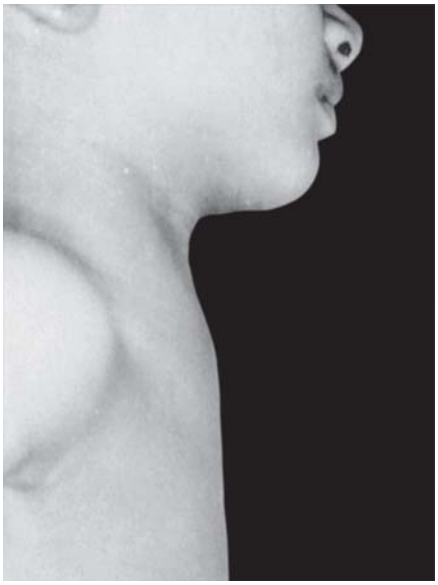


Fig. 22.3: Surgical emphysema on right side of neck because of foreign body in left main bronchus. The child had left pneumothorax and pneumomediastinum *(For color version, see Plate 3)*

of subcutaneous emphysema with streaks of air in the mediastinum point to the diagnosis of pneumomediastinum (Fig. 22.3). In patients with subcutaneous emphysema with pneumomediastinum, it is advisable to insert an intercostal drain on the affected side preoperatively as there is a distinct possibility of the occurrence of a pneumothorax intraoperatively. A quick smooth induction of anesthesia is necessary to prevent further rise in mediastinal tension. Also, nitrous oxide

being highly soluble compared to nitrogen can expand the pneumomediastinum and hence should be avoided.[3]

Laryngotracheal foreign bodies frequently cause laryngeal subglottic edema which can be seen on the lateral neck films.[14]

Other investigations – Depending on the urgency of the procedure, investigations appropriate for the age of the patient and comorbid conditions should be asked for.

Resuscitation

Complete airway obstruction is rarely seen by the tertiary care team. In such a scenario, primary intervention by a Heimleich maneuver is the therapy of first choice, followed by digital extraction. One should be aware that digital manipulation can push an obstructing foreign body further into the airway. If the patient has become hypoxic, cyanotic and moribund, the only life saving option is tracheotomy. Partial airway obstruction is the clinical entity most frequently encountered. The guiding principle of treatment should be "Do not convert a partial airway obstruction into a complete airway obstruction".[15] The surgeon must be prepared to perform an emergency tracheostomy or cricothyrotomy if partial obstruction suddenly becomes complete.

Prior to performing bronchoscopy, the characteristics of the foreign body are reviewed. If possible, a duplicate foreign body is examined and manipulated with the forceps and bronchoscopes to determine the behavior of the object with this instruments.[19]

Fasting: When possible, removal of the foreign body should be done as an urgent, not emergent, procedure in a well-prepared patient. The urgency to proceed with anesthesia depends on the severity of respiratory distress, and the location and nature of the aspirated material. Laryngotracheal foreign bodies cause considerably more distress and are associated with higher mortality than objects lodged peripherally. Similarly, objects located at precarious sites, such as carina, demand prompt attention.[3]

Even in the absence of marked respiratory distress, objects such as pea nuts, which can fragment, or sharp objects should be aggressively removed. When an aspirated foreign body is stable and causes no or minimal distal airway obstruction, normal fasting guidelines should be followed. If necessary, one may arrange for referral and management by a speciality team and complete other preparations for bronchoscopy. Optimal fasting times are 4 to 6 hours for solids and 2 hours for clear fluids. Fasting is important to decrease the risk of further aspiration because the airway cannot be fully protected during the procedure.[2]

An initial trial of bronchodilators, postural drainage, and chest physiotherapy to dislodge and expel the foreign body before attempting bronchoscopy is no longer recommended.[3]

Preparation: Rigid bronchoscopy should only be undertaken by experienced staff in an operating room fully equipped for managing pediatric airway emergencies. A minimum of two anesthetists is critical to a successful outcome, and at least one of the anesthetists should be a pediatric anesthetist if the patient is an infant. Lack of experience was recently cited as a contributing factor to poor outcome of bronchoscopy.[2] There must be a clear plan of action for treatment of airway obstruction (i.e. equipment including emergency cricothyrotomy, drugs and personnel immediately available). After careful assessment of the patient an anesthetic plan can be developed with backup plans if a "surprise" occurs.

Anticholinergic agents: Atropine (20 µg/kg) or glycopyrrolate (10 µg/kg) are often recommended for rigid bronchoscopy to attenuate the volume of airway secretions and to prevent vagally mediated arrhythmias associated with mechanical stimulation of the airway. However, their routine use has been questioned. In a survey of Australian anesthetists, very few used anticholinergics routinely. In a study comparing three groups of patients undergoing laryngoscopy and bronchoscopy, those in the placebo group had a lower incidence of cardiac arrhythmias compared with those in the atropine or the glycopyrrolate group.[2,13]

Aspiration prophylaxis: The airway must be protected from aspiration of gastric contents during prolonged airway manipulation. Metoclopramide 0.15 mg/kg IM or IV may be used to facilitate gastric emptying and to increase the tone of cardiac sphincter. H_2 receptor antagonist cimetidine 2 mg/kg IM should be considered to reduce the risk of aspiration pneumonitis. IV ondansetron 0.1 mg/kg can be given for antiemesis.

Sedative premedication should be avoided because it may precipitate total airway occlusion.

Monitoring

Standard monitors include pulse oximetry, capnographs, electrocardiography, and blood pressure reading and a PNS. A precordial stethoscope is useful in detecting any changes in airway sounds or regional ventilation. A second stethoscope is sometimes, useful in monitoring air entry over the lung fields as the endoscopy proceeds.[2]

Particular attention should be paid to pulse oximetry readings which will show desaturation before a change in skin color. In addition, the rate of change in saturation serves as a guide to how well the patient might tolerate an apneic episode.[2] Before induction oxygen saturation must be noted on room air to evaluate the degree of right to left intrapulmonary shunting due to airway obstruction.

Capnography gives an unreliable estimate of endtidal CO_2, because most expired gas exits via or around the scope rather than the side arm. A low CO_2 value is actually due to dilution by dead space gas and air trapping and does not reflect hyperventilation. The patient could, in fact be hypercarbic.[13]

Transcutaneous CO_2 monitors may be useful when the usual endtidal expired CO_2 is not available (Venturi jet ventilation and apneic oxygenation). During jet ventilation technique, the motion of the chest wall should be observed throughout the procedure.[13]

Documentation of the extent of muscle relaxation is essential to avoid sudden movements, coughing and bucking during the foreign body removal as these can cause significant tracheal or laryngeal damage. A train of four or double burst stimulus pattern may be used.[13]

Rigid Bronchoscope[14,20]

The rigid ventilating bronchoscope with an optical telescope forcep is used most commonly for foreign body removal. Rigid telescopes with 0 to 90 degree lenses passed through the rigid bronchoscope improve visualization in the more distal and upper lobe bronchi. The standard adult rigid bronchoscope is 40 cm in length and has an internal diameter of 8 mm. The size of a bronchoscope refers to the internal diameter and is typically 2 to 3 mm less than the corresponding external diameter. Because the external diameter may be significantly greater than in an endotracheal tube of similar size, care must be taken to select a bronchoscope of proper external diameter to avoid damage to the laryngeal structures[21] (Table 22.1).

The anesthesia circuit is connected to the bronchoscope through a side-arm, and a closed circuit is achieved by either placing the thumb, a glass eyepiece, or a telescope at the proximal end of the bronchoscope. A second side port provides a means of introducing flexible suction catheters or delicate instruments into the lumen of the bronchoscope. Increased airway resistance may occur when using the telescope or forceps through a bronchoscope with a small lumen. This increased resistance during expiration may require the telescope to be regularly removed with subsequent manual ventilation to avoid air trapping and its complications. If intrathoracic pressure is sufficiently increased, cardiac output can be impaired. Maintaining adequate expiratory time (greater than 5 seconds) is essential to allow complete expiration. When a 2.5 mm bronchoscope is used in small infants, lung emptying is facilitated by removing the telescope frequently, and only occasional positive pressure breaths using low flow oxygen (1 l/min) and leaving the pop off valve open.[13]

Anesthetic Management

Laryngeal Foreign Body[11]

In case of acute respiratory distress and hypoxemia with laryngeal foreign body, anesthesia is induced with the patient in a sitting position with an inhaled anesthetic and oxygen while the patient is monitored with a precordial stethoscope, pulse oximeter and ECG. Spontaneous breathing is preferable, as positive pressure ventilation may cause the foreign body to be displaced and further obstruct the airway. Sevoflurane is the drug of choice for inhalational induction because of its tendency not to induce coughing and better cardiovascular stability. The use of nitrous oxide is contraindicated in these patients as it reduces the FiO_2. Also, if significant air trapping is present nitrous oxide can increase the gas volume and pressure in the affected lung.[3]

After inhalational induction, intravenous access is established (if not already available). A vagolytic dose of atropine 0.02 to 0.03 mg/kg or glycopyrrolate 5 to 10 µg/kg is administered. The inhalational induction in a patient with marginal airflow is slow and 15 to 20 minutes may be required to reach a sufficient depth of general anesthesia to suppress airway reflexes. Gentle application of CPAP helps to overcome upper airway obstruction. Before endoscope insertion, topical local anesthesia of the oropharynx helps prevent airway reflexes. The endoscopist does the laryngoscopy to find and extract the laryngeal foreign body. Inhalational anesthesia may be switched to total intravenous

Table 22.1: Comparison of external diameter of standard endotracheal tube versus rigid bronchoscope

Internal diameter (mm)	*External diameter (mm)*	
	Endotracheal tube	*Rigid bronchoscope*
2.5	3.5	4.2
3.0	4.3	5.0
3.5	4.9	5.7
4.0	5.5	6.7
5.0	6.8	7.8
6.0	8.2	8.2

anesthesia with graded doses of propofol with or without opioids (fentanyl, remifentanil) or the anesthesiologist must reoxygenate and re-anesthetize by mask with spontaneous respiration if the patient becomes light.[13] Sometimes, it may be necessary to push the foreign body peripherally into a mainstem bronchus with improvement in ventilation and relief of the immediate crisis.[3]

Bronchial Foreign Body

For children in stable condition with foreign body aspiration presumed to be in the bronchus, induction of anesthesia by the inhalational or IV route are both described in literature.[2,11] The choice is often-based on the institution's protocol and the anesthetist's training. However, spontaneous ventilation must be maintained until it is certain that the child can still be ventilated under anesthesia. Rapid sequence IV induction is more likely to risk loss of the airway. The risk of aspirating gastric contents is small with either induction technique and if aspiration occurs, the consequences are relatively mild.[2] Sevoflurane is the agent of choice for inhalational induction because of its tendency not to induce coughing and better cardiovascular stability.

Maintenance of Anesthesia

To adequately ventilate the patient, the bronchoscope must be in direct alignment with the airway. Precautions against hyperextension of the head, especially in the very young infant, are mandatory. Hyperextension causes the bronchoscope to be directed anteriorly and makes ventilation difficult to impossible.[12]

Historically, there have been two schools of thought regarding the ventilatory management: spontaneous versus controlled. Fearson et al, Chatterjee et al, Baraka, Kim et al, Perrin et al and Ahmed prefer spontaneous ventilation, where as Kosloske, Blazer et al, and Puhakka et al advocate neuromuscular blockade and controlled ventilation during removal of foreign body.[8]

Advantages and Disadvantages of Spontaneous Ventilation

Advantages: The advantage of spontaneous ventilation is lower-risk of foreign body being moved more distally, increasing the difficulty to remove and possibly leading to ball-valve obstruction of the airway (as compared to positive pressure ventilation). Spontaneous ventilation also allows for continued ventilation during removal of the foreign body with the bronchoscope's ocular piece open and rapid assessment of the adequacy of the airway after removal of the foreign body.[2]

Disadvantages: Increased depth of anesthesia is required during spontaneous ventilation to permit insertion of instruments into the airway, which could decrease both cardiac output and ventilation. In addition, increased resistance to ventilation during the use of the telescope or forceps could worsen the hypoventilation leading to hypercarbia and hypoxia. Also, a variable amount of room air is entrained, diluting the FiO_2 as well as the concentration of anesthetic gases. The incidence of coughing, bucking, breath holding, and apnea is more during spontaneous ventilation due to uneven depth of anesthesia.[2,8]

Advantages and Disadvantages of Positive-pressure Ventilation

Advantages: The main advantage of using a muscle-relaxant technique is that the airway is immobilized, which facilitates removal of the foreign body and decreases the chances of trauma to the airway. A muscle-relaxant technique also allows the lighter level of anesthesia which in turn decreases anesthetic effects on cardiac output. In addition, positive-pressure ventilation may decrease atelectasis, improve oxygenation and overcome the increased airway resistance that occurs when a telescope is used.[2] Muscle relaxation is particularly useful for bronchoscopy involving the removal of the foreign body distal to the carina, especially because the duration of these procedures can extend to more than an hour.[11] The time taken for induction of anesthesia and the recovery times are also significantly shorter in patients whose respiration is controlled.[8]

Disadvantages

1. With ventilating bronchoscope, leakage of gases around the lumen may not permit adequate ventilation and ventilation is further interrupted when the sliding eyepiece of proximal end is removed during surgical maneuvers.[2,8]
2. IPPV may push the foreign body further down.[2,8]
3. Jet ventilation is more likely to dislodge the foreign body or cause barotraumas.[2]
4. In view of early recovery and awakening, the incidence of postoperative cough is higher as compared to spontaneous respiration.[8]

In a report of four patients in whom the bronchoscopist had the foreign body slip from the forceps back into the airway, neither controlled ventilation with muscle paralysis nor spontaneous breathing under a deep plane of anesthesia played a role in the mishaps. It was thought that the experience of the endoscopist and the availability of proper equipment were more important factors than the method of ventilation.[3]

Maintenance

Anesthesia is usually maintained by connecting anesthesia breathing circuit to the sidearm of the bronchoscope. Ventilation can be assisted or controlled with 100 percent oxygen and halothane or isoflurane or a propofol-based total intravenous anesthetic technique with or without muscle relaxants. Halothane and isoflurane have some advantage over sevoflurane during maintenance because they are more soluble and are associated with slower elimination which allows more time for airway manipulations without the possibility of the patient becoming too lightly anesthetized and reacting to the procedure.[22]

A propofol-based total intravenous anesthetic technique is more superior as it allows a steady level of anesthesia that is independent of ventilation and does not expose the operating room personnel to waste anesthetic agents that inevitably spill around the bronchoscope.[22] Topical anesthesia of the vocal cords and trachea is used as an adjunct to general anesthesia. Lidocaine 1 percent has two advantages for this application: (1) larger volumes can be used, and (2) it has short (10-min) duration of action. Doses up to 4 mg/kg have been used without complications but the dose may need to be reduced in patients younger than 2 years old and in those with dry mucosa.[2]

The use of short-acting (e.g. mivacurium) or intermediate-acting (e.g. vecuronium and atracurium) muscle relaxants mandates positive pressure ventilation, but enables a lighter level of anesthesia and ensures a quieter field for the surgeon.[22]

Ventilation During Bronchoscopy

Ventilation during bronchoscopy in the paralyzed patient can be carried out by means of apneic oxygenation, high-frequency positive-pressure ventilation (HFPPV), a ventilating bronchoscope, or a Venturi injector device.[7,23,24]

Apneic Oxygenation

A fine catheter is passed into the trachea and oxygen insufflated at 10 to 15 liters per minute throughout bronchoscopy. The patient is hyperventilated to achieve profound hypocapnia before presenting him to the surgeon. Satisfactory arterial oxygenation is maintained because oxygen is taken up from the alveoli where the concentration remains high because of mass movement of oxygen from the insufflating catheter. However, there is a progressive rise in arterial carbondioxide tension (mean rise 3 mm Hg per minute – 0.4 kPa) and therefore this method is unsuitable for prolonged brochoscopy. Not more than 10 minutes of bronchoscopy is permitted in adult patients and much lower limits are permitted in children. This technique works well in patients without significant pulmonary disease.

High Frequency Positive-pressure Ventilation

High frequency jet ventilation (HFJV) and High frequency positive-pressure ventilation (HFPPV) have also been used during bronchoscopy. High frequency positive-pressure ventilation (HFPPV) rates between 60 and 100 per minute have been found to provide adequate alveolar ventilation at a low airway pressure. The principle has evolved from systems designed for use during endoscopy and a particular benefit during bronchoscopy is that there is no entrainment of air and so anesthetic gases can be delivered at known concentrations.

The advantages of HFJV at ventilation rates of 100 to 150 jet per minute are smaller tidal volumes, less vocal cord motion and reduced airway barotraumas.[2] Carbon dioxide retention can occur with HFJV at frequencies above 300 to 360/min. At frequencies of 150 to 300 bpm, HFJV produced adequate but not superior gas exchange when compared with manual jet ventilation at 20 bpm.[20]

Ventilating Bronchoscope

The ventilating bronchoscope was first described by Muendrich and Hoflehner in 1953. Anesthetic gases are administered through a sidearm of the bronchoscope and the proximal lumen is occluded by a glass window. It is important to remember that with extremely high-flow resistance through the sidearm of the bronchoscope spontaneous breathing is all but ineffective. High fresh gas flows, large tidal volume and high inspired volatile anesthetic concentrations are often necessary to compensate for leaks around the ventilating bronchoscope and the high-resistance encountered when the viewing telescope is in place. During long procedure, carbondioxide accumulates and predisposes the patient to dysrrhythmias particularly in the presence of light anesthesia. Hypoxia and hypercarbia due to inadequate ventilation are remedied by manual ventilation at higher than normal tidal volume, frequent removal of the telescope and withdrawal of the bronchoscope to the midtrachea.[21] It also deepens the anesthetic.

The respiratory rate especially the expiratory phase must be kept very slow to allow sufficient time for passive exhalation.[11] The sliding eyepiece at the proximal end has to be removed during surgical maneuvres, e.g. suctioning, foreign body removal, etc. Ventilation in a paralyzed patient is interrupted during these maneuvers, leading to hypoxia, hypercapnia and

lightening of anesthetic plane. So these maneuvres should be intermittent and short. The use of oxygen flush should be kept to a minimum as the flush bypasses the anesthetic vaporizer and dilutes the inhalational agent.

Venturi Injector Device

Its use during bronchoscopy was first described by Saunders in 1967. Oxygen from a high-pressure source is injected intermittently through a narrow needle placed at the proximal end of the bronchoscope. The venturi effect which this creates entrains atmospheric air so that the lungs can be inflated with oxygen-enriched air as long as the distal end of the bronchoscope is beyond the larynx.

The system consists of a high-pressure source of oxygen (usually pipeline pressure at 410 kPa (approximately 60 psi), an on-off tap, high-pressure tubing and a needle of suitable size. It is essential that the size of the needle should match that of the bronchoscope so that good air entrainment is achieved without creating excessive airway pressure. A 16 SWG needle coupled to a driving gas pressure of 410 kPa (approximately 60 psi) is usually used with an adult Negus bronchoscope and gives a maximum inflation pressure of 25 to 30 cm water (2.5-3.0 kPa). Typical maximum pressures achieved with various sizes of needles and bronchoscopes are summarized in Table 22.2.[7]

Using this system it is possible not only to maintain arterial oxygenation but also keep arterial carbon dioxide values within normal limits in a normal subject. However, excessive pressure can be created if the proximal end of the bronchoscope is obstructed and the distal end is a tight fit at the larynx or elsewhere in the tracheobronchial tree. Complications can also occur if the driving gas pressure and venturi needle size are not matched appropriately.

Jet ventilation techniques rely on passive recoil of the lung for exhalation. This exhalation phase needs adequate egress and time to prevent air trapping and pressure build up in small airways which may lead to barotraumas.[13] Pediatric patients are less likely to have a significant gas leak around the bronchoscope and higher inflation pressures may develop leading to barotraumas. Miyasaka et al reported that in 6 months to 3 years old children, maximal inflation pressures and the volume of ventilation are vulnerable to small alterations in variables like the size of the jet and the bronchoscope, the length and angle of the jet, the shape of the bronchoscope (tapered or straight), and the introduction of a suction canula. Tidal volumes in excess of 10 ml/kg were achieved with driving pressures of 24 to 45 psi. However, the results of this study are applicable only when the Sanders No.19 adapter is used.[3] Nitrous oxide-oxygen jet mixtures have also been used. In addition to environmental pollution, nitrous oxide can produce optical distortion because of an alteration of the refractive index of the gas mixture.[20]

Table 22.2: Maximum inflation pressure achieved with various venturi bronchoscope injector systems (driving pressure 410 kPa or 60 psi)[7]

Negus bronchoscope	*Injector needle size*		*Typical maximum pressure*
	SWG	*Internal diameter (mm)*	*(cm) H_2O*
Adult	14	1.2	50
Adult	16	1.2	25-30
Adult	17	1.10	23-25
Child	19	0.69	14-18
Suckling	19	0.69	15

Oxygen (100%) is used as the jet gas. The effective FiO_2 is about 0.8 to 0.9 because of dilution from entrainment of ambient air.[13] Usually TIVA technique is used in association with jet ventilation equipment. Anesthesia is provided by a mixture of either thiopentone sodium or propofol with a potent narcotic such as fentanyl or alfentanil and muscle relaxation. After induction, a propofol drip at the rate of 100 to 200 µg/kg/minute can provide the deep sedation component of TIVA. Narcotics are necessary to suppress airway reflexes and blunt adrenergic responses to pain. Sudden patient motion, coughing or bucking during bronchoscopy can have disastrous consequences such as mucosal bleeding or perforation. Hence, muscle relaxation is an essential part of this procedure. Muscle relaxation is also essential to ensure low resistance to inspiration and adequate tidal volumes at reasonable airway pressures. Patient recall during TIVA can be decreased by adding benzodiazepine such as midazolam to the anesthetic management.

Patient selection is important when considering the use of jet ventilation. Patients with significantly compromised airways are susceptible to barotraumas, pneumothorax and hypoventilation and are not good candidates for jet ventilation. Obese patients and patients with poor lung compliance are difficult to adequately ventilate using jet ventilation techniques and hypercarbia or hypoxia may develop.

To decrease the incidence of barotrauma in children, the inspiratory/expiratory ratio of ventilation should be

at least 1:4 to allow adequate time for complete passive exhalation. The respiratory frequency for small children should be about 20 breaths per minute. Driving pressures of only 5 to 10 psi are used initially and increased gradually until sufficient expansion of the thorax occurs with each jetted ventilation.[13]

The use of a cylinder with an adjustable regulator allows far greater flexibility in the technique of pulmonary ventilation than piped oxygen. It is desirable in certain patients to set the pressure at which oxygen flows through the injector to a level above or below 60 lb/sq in provided by piped oxygen. Patients with noncompliant lungs or with a large gas leak around the bronchoscope will require a higher pressure, while patients with small airways, such as children, will require a lower pressure.[25]

Airway pressures and end-tidal CO_2 values cannot be monitored during jet ventilation. The adequacy of the mechanics of ventilation is determined by observing the chest wall or sternum rise and fall, with each jet ventilation. So, the patient's chest must be clearly visible throughout the procedure. Also, the system does not allow precise control of FiO_2. Other disadvantages include potential contamination of operating room, gastric dilatation, uncertainty of oxygen concentrations delivered, inability to monitor end-tidal CO_2 and mucosal drying. The FiO_2 and the air entrainment depend on jet location, nozzle size, driving pressure and airway resistance.

Giesecke et al[26] and Morales et al[27] have compared mechanical ventilation with the ventilating bronchoscope and with the Sander's injector during bronchoscopy in anesthetized, fully relaxed patients and found that significant progressive respiratory acidosis developed intraoperatively in those patients in whom ventilating bronchoscope was used. However, PCO_2 remained in the normal range in those patients in whom the Sander's injector was used, although the PO_2 levels in patients being ventilated with the Sander's injector technique were lower than that of the patients being ventilated with the ventilating bronchoscope, they were clinically not significant.

If the endoscopist is taking too long to grasp the object with the bronchoscope in a distal airway the nonventilated lung may become atelectatic, resulting in hypoxia, hypercarbia and light anesthesia may ensue. The endoscopist needs to retreat momentarily to the midtrachea so that the patient can be adequately ventilated.[22] However, during the crucial moment of foreign body retrieval, ventilation sometimes must be held until the oxygen saturation begins to fall.[11]

If the child has aspirated vegetable matter, it may be fragmented during removal and may occlude the originally unobstructed bronchus or trachea. Hence, working with the patient in the lateral position with the affected side down is advantageous in this situation. If this is not feasible, thoracotomy instruments and a surgeon skilled in pediatric thoracic surgery should be immediately available. A Fogarty No. 3 embolectomy balloon catheter may help dislodge impacted foreign bodies.[3]

When the foreign body is in the grasp of the forceps, the upper airway and glottis need to be quiet and relaxed. The airway should be anesthetized adequately so that the foreign body is not dropped from the forceps by a cough or a closing glottis. At this point, the depth of anesthesia may need to be increased if the child is breathing spontaneously or manual ventilation may need to be stopped temporarily in the paralyzed patient. Supplemental intravenous lidocaine 1 mg/kg is helpful to attenuate the airway reflexes. When the foreign body is retrieved, it may prove too large to pass through the bronchoscope. This requires that both instruments and the foreign body be removed as a unit. After removal of the foreign body, the airway should be re-evaluated for any other foreign bodies and the impact side should be assessed for trauma, bleeding or granulations. Once the endoscopy and foreign body removal are complete, beta agonist bronchodilators such as albuterol may help with postoperative wheezing.[22] Lidocaine 1.5 mg/kg given intravenously at the end of the procedure decreases the incidence of coughing in the postbronchoscopy period.[8]

Sometimes, the bronchoscope may need to be reinserted multiple times before the foreign body and secretions are successfully removed. This trauma to the airway may produce mucosal edema with respiratory distress following bronchoscopy. Measures to minimize postoperative stridor and distress include administration of steroids (dexamethasone 0.5 to 1 mg/kg with smaller doses repeated at intervals as dictated by the individual situation), humidified oxygen, and nebulized racemic adrenaline, and rarely, reintubation for 1 or 2 days until the edema subsides. Racemic epinephrine 0.5 ml 2.25 percent is given in a 1:6 to 1:10 dilution through a nebulizer accompanied by ECG monitoring and is repeated as necessary every 2 hours.[3]

The Dropped Foreign Body

One of the most serious complications of attempts to remove a foreign body is obstruction of the airway

caused by movement of the foreign body. This may occur if the foreign body is dropped or if it fragments proximally. Management by the endoscopist includes pushing the foreign body more distally into one of the main bronchi preferably the affected side because placing it in the unaffected lung could result in unreliable ventilation of either lung.[2,3]

After extraction of the foreign body the airway is intubated, the patient is ventilated with 100 percent oxygen. Tracheal intubation allows for tracheobronchial suction, lung expansion and for oxygenation and ventilation until adequate reversal of muscle relaxation and return to spontaneous breathing. A nasogastric tube is placed to decompress the stomach. Extubation is performed with the patient awake and with protective airway reflexes present. A chest X-ray should be obtained before the patient is discharged from the postanesthesia care unit to rule out the presence of pneumothorax or atelectasis. Postural drainage and chest percussion enhance clearance of secretions and decrease subsequent risk of infections.

Complications

Several complications are associated with rigid bronchoscopy such as coughing and bucking, pneumothorax, mediastinal and subcutaneous emphysema, laryngospasm, bronchospasm, laryngeal edema, cardiac arrhythmias, cardiac arrest, airway rupture with massive hemoptysis, unpleasant recall, convulsions and death. These could be due to many reasons such as inadequate depth of anesthesia, hypoxia, inadequate ventilation, vagal stimulation, metabolic and electrolyte disturbances.[8]

Any rapid deterioration of cardiac function during bronchoscopy should make one highly suspicious of tension pneumothorax which justifies immediate thoracostomy before obtaining a chest radiograph. Complete obstruction of the airway may occur. If foreign body cannot be removed and ventilation is inadequate, an emergency thoracotomy and bronchotomy may be necessary.[14] Adrenergic responses to the stimuli of airway instrumentation may produce hypertension, tachydysrhythmias, coronary ischemia, and changes in cardiovascular dynamics. These adrenergic responses are best prevented by good topical local anesthesia of the airway, by maintaining adequate depth of anesthesia and with beta blocking drugs like esmolol or labetalol. Lidocaine is rapidly absorbed from the trachea and small airways leading to local anesthetic toxicity. The dose of lidocaine placed in tracheal areas below the glottis should be limited to 1 to 1.5 mg/kg.[13] Teeth can be damaged or dislodged if the bronchoscopist is not vigilant in protecting dental structures. Loose and damaged teeth can become foreign body. Postobstructive pulmonary edema may occur in the recovery room and should be treated aggressively.[14]

FOREIGN BODY IN THE UPPER DIGESTIVE TRACT

Ingestion of foreign bodies is common, especially among the pediatric age group. According to the National Safety Council, suffocation from foreign body ingestion and aspiration is the third leading cause of accidental death in children younger than 1 year and the fourth leading cause in children between 1 and 6 years.[11] In adults, it occurs more commonly among those with psychiatric disorders or mental retardation, prisoners and alcoholics. Fortunately, most ingested foreign bodies pass through the gastrointestinal tract harmlessly. However, 10 to 20 percent will require non-operative intervention and only 1 percent or less requires surgery.

The type of esophageal foreign body typically relates to the age of the patient. Infants and children tend to swallow toy parts and coins. Adults experience difficulty with meat, often poorly masticated, fish bones and occasionally swallowed dentures.[19]

Clinical Presentation

The most frequent symptoms of upper esophageal foreign body include dysphagia, drooling, gagging, retching and vomiting.[11] An esophageal foreign body can present with signs of airway obstruction due to distension of the anterior wall of the esophagus in to the posterior membranous wall of the trachea.[19]

Foreign bodies lodged in the digestive tract at the level of the cricopharyngeus may cause respiratory effects. If large, they may cause respiratory arrest; otherwise, they may cause inflammation and subsequent compression of the airway. Esophageal foreign bodies constitute an emergency when actual or potential airway obstruction occurs or when there is impending perforation. Disk batteries are particularly hazardous if ingested and lodged in the esophagus. Perforation may occur within 8 to 12 hours.[14] Nonreactive foreign bodies, such as coins, can be removed nonemergently if airway obstruction or significant esophageal obstruction is not present.[19]

The commonest site of lodgement of ingested foreign body is cricopharynx (C6 vertebra). Fish bone is commonly lodged in tonsillar crypts or vallecula.[28] Big foreign bodies like meat bones or chicken bones

commonly lodge in cricopharynx and at various constrictions of the esophagus.[11]

1. The proximal esophagus at the level of the cricopharyngeal muscle (Figs 22.4 and 22.5) and thoracic inlet (Fig. 22.6)—the foreign body is seen at the level of the clavicles on chest radiograph. If the foreign body is a coin, it will be oriented in a transverse position because the opening of the esophagus is widest in a transverse position.
2. The middle esophagus at the level of the carina and the aortic arch.
3. The distal esophagus just proximal to the esophageal gastric junction. The foreign body is seen 2 to 4 vertebral bodies above the stomach bubble on chest radiograph.

Investigations[28]

1. A chest X-ray (PA view) and lateral.
2. Plain X-ray lateral and AP view of the neck, should be obtained in all cases of suspected foreign body ingestion. Radio opaque foreign body would be seen on the X-ray. If the foreign body is radiolucent, there may be prevertebral widening or anteriorly displaced airway.
3. Fine radiolucent foreign body gets covered with barium during fluoroscopy with thin barium swallow.

Complications[3]

As with chronic airway foreign bodies, retained esophageal foreign bodies can lead to bronchoesophageal fistula, aortoesophageal fistula, retropharyngeal and parapharyngeal abscess, mediastinitis, esophageal perforation with surgical emphysema, esophageal diverticulum and lobar atelectasis. These retained foreign bodies may require a thoracotomy to remove them.

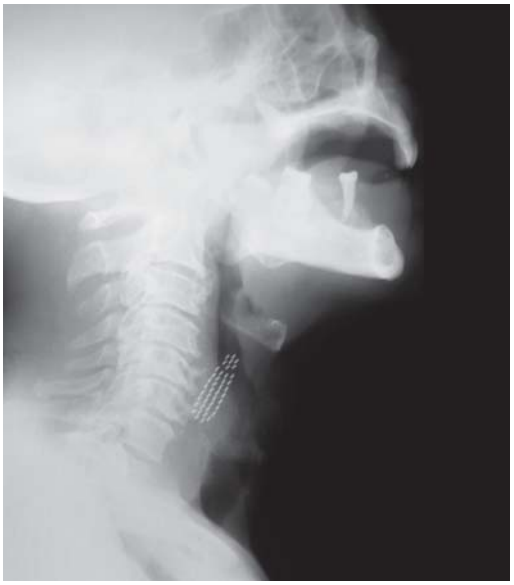


Fig. 22.5: Foreign body (tooth brush) at the level of cricopharynx in an elderly

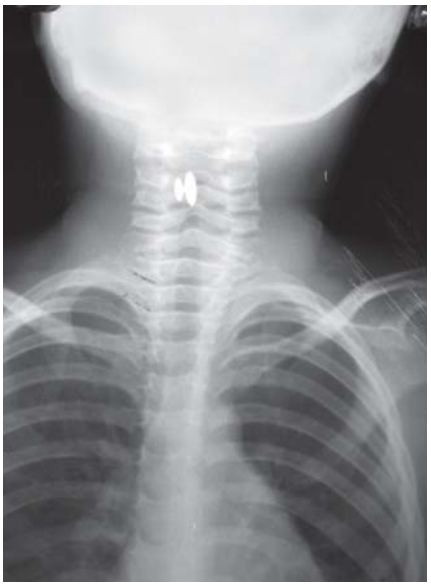


Fig. 22.4: Foreign body (button) at the level of cricopharynx in a child

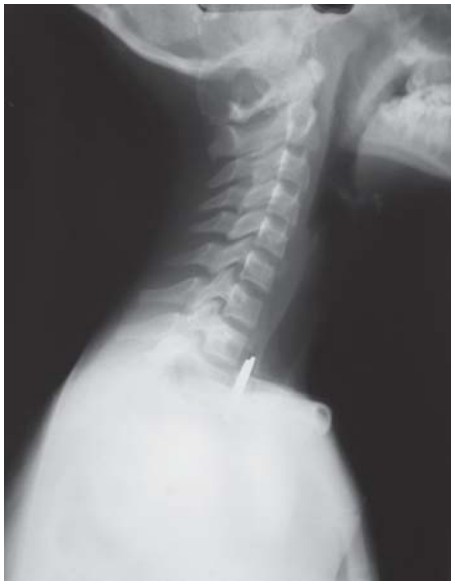


Fig. 22.6: Foreign body (coin) at the level of thoracic inlet

Anesthetic Management[3,11]

Esophageal foreign bodies can be removed with rigid or flexible esophagoscopes. Adults may tolerate rigid endoscopy with intravenous sedation. Sharp objects are best managed with rigid scopes, since the scope can be used to sheath the object during extraction.

Removal of esophageal foreign bodies in infants and children should be performed with a rigid esophagoscope using general anesthesia.[19] Accidentally dropping an esophageal foreign body in to an unprotected larynx can cause a disaster. Hence, endotracheal intubation and airway protection should precede foreign body extraction from the esophagus.[3] General anesthesia with laryngotracheal intubation provides the conditions to produce excellent muscle relaxation and protection of the airway during foreign body removal. As long as the child is not dyspneic, the anesthetist should wait 4 to 6 hours after the last meal depending on the child's age until the stomach is empty.[11]

Monitoring

ECG, pulse oximeter, capnography and manual blood pressure.

If the airway is not compromised, after giving anticholinergics and sedation, a rapid sequence induction with cricoid pressure is performed.[15] During esophagoscopy, the mucosa over the cricoid cartilage may be traumatized by compression between the endotracheal tube anteriorly and the rigid esophagoscope posteriorly. This increases the incidence of postintubation croup and stridor. To avoid this complication, a reduced size endotracheal tube may be helpful. Dexamethasone (0.4 to 1 mg/kg up to 20 mg) may also reduce the incidence of postoperative stridor.[11]

After intubation, the endotracheal tube is fixed at the left angle of the mouth as the surgeon will insert the esophagoscope down the right side of the mouth. The anesthetist along with the anesthesia machine stands towards the left side of the patient. The anesthesia is maintained with oxygen, nitrous oxide (33:66) and inhalational agent or a propofol-based total intravenous technique with intermediate acting muscle relaxant (vecuronium or atracurium). Appropriate muscle relaxation reduces the incidence of esophageal perforation by preventing unwanted movement or bucking during the procedure.[15]

Inadvertent extubation can occur during withdrawal of the esophagoscope from the oral cavity. The endoscopist must be aware of this possibility and maintain a firm hold of the foreign body during this part of the procedure. The anesthetist should be prepared for a premature extubation and possibility of managing an unprotected airway.[19]

When the foreign body is held in the hypopharynx, one must avoid gagging and coughing at the time of induction. This may dislodge the foreign body which may slip into the larynx, completely occluding the airway. In such a scenario, the child is sedated heavily with an opioid and a sedative to avoid excitement, gagging and coughing. After IV access is established and atropine is given (20 µg/kg), the trachea is intubated under deep inhalational anesthesia or propofol with or without muscle relaxation after confirming the ability to ventilate the child. Cricoid pressure is avoided in these circumstances because it may irritate the upper airway or dislodge the foreign body.[11]

Intraoperative Complications

1. Esophageal perforation which may lead to pneumothorax (Right > Left).
2. Compression of endotracheal tube.
3. Dysrhythmia.
4. Aspiration.
5. Accidental extubation.
6. Stridor secondary to subglottic edema.

At the end of the procedure, after adequate reversal of muscle relaxation, extubation is performed when the patient is awake, has spontaneous breathing efforts, and protective airway reflexes.

REFERENCES

1. Kalyanappagol VT, Kulkarni NH, Bidri LH. Management of tracheobronchial foreign body aspirations in paediatric age group—A 10 years retrospective analysis. Indian J. Anaesth 2007;51(1):20-3.
2. Farrell PT. Rigid bronchoscopy for foreign body removal: Anesthesia and ventilation. Pediatric Anaesthesia 2004;14: 84-9.
3. Brett CM, Zwass MS. Eyes, Ear, Nose, Throat and Dental Surgery. In: Gregory GA (Eds). Pediatric Anaesthesia. 4th edn. Philadelphia: Churchill Livingstone 2002;663-705.
4. Baharloo F, Veyckemans F, Francis C, Biettlot M, Rodenstein DO. Tracheo bronchial foreign bodies: Presentation and management in children and adults. Chest 1999;115:1357-62.
5. Aytac A, Yurdakul Y, Ikizler C, Olga R, Saylam A. Inhalation of foreign bodies in children Report of 500 cases. The Journal of Thoracic and cardiovascular surgery 1997;74(1):145-51.
6. Chatterji S, Chatterji P. The Management of foreign bodies in air passages. Anaesthesia 1972;27(4):390-5.

7. Gothard JW. Thoracic Anaesthesia. In: Churchill-Davidson HC (Eds) Wylie and Churchill Davidson's A Practice of Anaesthesia. 5th edn, Singapore: PG Asian Economy 1990; 377-8.
8. Soodan A, Pawar D, Subramanium R. Anesthesia for removal of inhaled foreign bodies in children. Pediatric Anesthesia 2004;14:947-52.
9. Ghosh BR, Das AK, Dutta S. Paediatric bronchoscopy. Ind J Anaesth 2000;44:40-5.
10. Sqn. Ldr. Lalla RK. Bronchoscopy for foreign body removal in children. Ind J Anaesth 1995;43:360-3.
11. Landsman IS, Werkhaven JA, Motoyama EK. Anesthesia for Pediatric otorhinolaryngologic surgery. In: Motoyama EK, Davis PJ (Eds). Smith's Anaesthesia for infants and children. 7th edn, Philadelphia: Mosby Elsevier 2006;789-822.
12. Cohen SR, Herbert WI, Lewis GB JR, Geller KA. Foreign bodies in the airway. Five years retrospective study with special reference to management. Ann. Otol 1980;89:437-42.
13. Donlon JV Jr. Anesthetic and airway management of laryngoscopy and bronchoscopy. In: Benumof JL (Eds). Airway management: Principles and practice. St Louis: Mosby Publishers 1996;666-83.
14. Salcedo L. Foreign body aspiration. Anesthesiology Clinics of North America 1998;16(4):885-92.
15. Ragan B. Anesthesia for otorhinolaryngolic (Ear, Nose, and Throat) Surgery. In: Longnecker DE, Brown DL, Newman MF, Zapol WM (Eds). Anesthesiology. New York: McGraw Hill 2008;1591.
16. Malde AD, Iyer HR, Pantavaidya SH, Jagtap SR. Pneumomediastinum complicating foreign body aspiration: Anaesthetic Considerations—A Case report. Ind. J. Anaesth 1998;42:51-3.
17. Rothmann BF, Boeckman CR. Foreign Bodies in the larynx and tracheobronchial tree in children. A review of 225 cases. Ann. Otol 1980;89:434-6.
18. Baraka A. Bronchoscopic removal of inhaled foreign bodies in children. Br J Anaesth 1974;46:124-6.
19. Magit AE, Davidson TM. Performance of rigid bronchoscopy. In: Benumof JL (Eds). Airway management principles and practice. St Louis: Mosby Publishers 1996; 475-83.
20. Pereira KD, Hessel AC. Performance of rigid bronchoscopy. In: Hagberg CA (Eds). Benumof's Airway Management. 2nd edn. Philadelphia: Mosby Elsevier 2007.
21. Ferrari LR, Gotta AW. Anesthesia for otolaryngologic surgery. In: Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MG (Eds). Clinical Anesthesia. 6th edn,. New Delhi: Wolters Kluwer/Lippincott Williams and Williams 2009;1305-20.
22. Verghese ST, Hannallah RS. Pediatric otorhinolaryngologic emergencies. Anesthesiology clinics of North America 2001;19(2):237-56.
23. Brodsky JB. Anesthesia for Thoracic Surgery. In: Healy TEJ, Cohen PJ (Eds). Wylie and Churchill Davidson's A Practice of Anesthesia. 6th edn. London: Edward Arnold 1995;1148-69.
24. Brown Allan CD. Anesthesia for Ear, Nose and Throat Surgery. In: Healy TEJ, Cohen PJ (Eds). Wylie and Churchill Davidson's A Practice of Anesthesia. 6th edn, London: Edward Arnold 1995;224-50.
25. Hart SM. A further modification of a simple apparatus for pulmonary ventilation during bronchoscopy. Brit. J. Anaesth 1970;42:78.
26. Giesecke AH Jr, Gerbershagen HU, Dortman C, Lee D. Comparison of the ventilating and injection bronchoscopes. Anesthesiology 1973;38(3):298-303.
27. Morales GA, Epstein BS, Cinco B, Adkins PC, Coakley CS. Ventilation during general anesthesia for bronchoscopy: Evaluation of a new technique. Journal of Thoracic and Cardiovascular surgery 1969;57(6):873-8.
28. Hazarika P, Nayar DR, Balkrishnan R (Eds). Foreign Body upper digestive tract. In: Textbook of Ear, Nose, Throat and Head and Neck Surgery (Theory, clinical and practical). New Delhi: CBS Publishers and Distributors 2007;609-12.

23 Anesthesia for Ophthalmic Emergencies

Swati S Chhatrapati, Nishant S Bawankule

KEY POINTS

- True ophthalmic emergencies, such as chemical burns and central retinal artery occlusions, must be treated within minutes to avoid permanent vision loss.
- Open globe injuries and other urgent procedures can be scheduled after appropriate NPO status and improvement of patient's general condition.
- Eye compression from a facemask or positioning can lead to central retinal artery occlusion and sudden rise in IOP.
- With intraocular procedures, profound akinesia and meticulous control of intraocular pressure (IOP) are requisite. However, with extraocular surgery, the significance of IOP fades, whereas concern about elicitation of the oculocardiac reflex assumes prominence.
- Pressure on the globe or any orbital contents can result in a oculocardiac reflex (trigeminovagal) with bradycardia, atrioventricular block, or asystole and is more common during general than regional anesthesia. Although it is a fatigable reflex, the oculocardiac reflex must be blunted with atropine or glycopyrrolate.
- The blood supply to the retina is driven by the ocular perfusion pressure, which is determined by the arterial blood pressure and the intraocular pressure (IOP).
- Sudden increases in IOP in the setting of open globe injury can lead to vitreous loss and blindness.
- Deep inhaled anesthesia, thiopentone and propofol reduces IOP, whereas ketamine and succinylcholine can increase IOP.
- Succinylcholine increases intraocular pressure by 5 to 10 mm Hg for 5 to10 min after administration, principally through prolonged contracture of the extraocular muscles.
- Prior administration of diazepam, lignocaine, acetazolamide, self-taming dose of succinylcholine or a small dose of nondepolarizing muscle relaxant can attenuate the rise in IOP caused by succinylcholine.
- Rocuronium in the dose of 0.9 mg/kg produces ideal intubating conditions after 60 seconds.
- The key to inducing anesthesia in a patient with an open eye injury is controlling intraocular pressure with a smooth induction. Specially, coughing during intubation must be avoided by achieving a deep level of anesthesia and profound paralysis.
- In children moderate sedation with opioid lollipop, topical anesthetic cream at IV site and gentle sevoflurane induction may be needed.

Most urgent ophthalmologic procedures do not need to be done on an emergency basis. This is important because the anesthetic plan must consider the NPO status and general medical condition of the patient. This does not apply to true eye emergencies. In these rare conditions, therapy should be started within minutes. Other urgent ophthalmologic conditions can start within 1 to several hours without change in outcome.

TYPES OF OPHTHALMOLOGIC EMERGENCIES

True Emergencies

These are chemical burns and central retinal artery occlusion.

Urgent Situations

Urgent situations include open-globe injuries, endophthalmitis, acute narrow-angle glaucoma, acute retinal detachment, corneal foreign body, and lid laceration.

Semiurgent Situations

Therapy should be started within days, but sometimes can be rescheduled after several weeks. Semiurgent situations include ocular tumours, blow-out fractures of the orbit, congenital cataract, and chronic retinal detachment.[1]

An emergency is defined as an event that has to be dealt with immediately, usually within the first hour after presentation. The commonest eye emergencies that fall into this category are chemical burns of the eye and retinal artery occlusion. Neither of these requires surgery as part of the initial management. Therapy should be started within minutes for chemical burns of the cornea and central retinal artery occlusion. Management involves immediate, copious, prolonged irrigation and removal of particulate matter as well as careful evaluation of the initial damage and visual acuity.[2] Anesthesia is not required for this situation.

Nontraumatic surgical emergencies include spontaneous retinal detachment, infections, and complications of previous surgery. One of the factors which determines the degree of urgency for retinal detachment surgery is the condition of the macula. The risk of a detachment progressing and resulting in loss of the macula increases the sense of urgency. There is usually enough time however to allow for fasting prior to surgery.

An injury to the eye or its surrounding tissues is the most common cause for attendance at an eye hospital emergency department. Ocular trauma is one of the most preventable causes of visual impairment in the world. Children and adolescents account for a disproportionate share of ocular trauma. Traumatic injuries can be blunt or penetrating. The incidence is highest in young adult males and children. Boys are the most vulnerable as compared with the girls.[3] The majority of cases presenting as emergencies can be defined as urgent cases.

COMMON TYPES OF EYE INJURY

- Corneal abrasions
- Foreign bodies
- Radiation damage
- Chemical damage
- Blunt injuries with hyphema
- Penetrating injuries.

ANESTHETIC MANAGEMENT OF PENETRATING EYE INJURY

If the patient has an open eye injury there is the risk of vitreous loss, retinal detachment, and infection causing endophthalmitis. Urgency of surgery depends on the size of the laceration, risk of loss of ocular contents, status of the wound, and risk of infection. Risk to the eye and risk to life because of inadequate starvation should be weighed. The case should be discussed with the surgeons. In many open injuries it is advisable to delay the operation until a patient is adequately fasted prior to anesthesia. If there is severe damage to the eye and chances of improvement in sight are minimal, then there is no point in risking the life. It may be worth anesthesing a patient with inadequate starvation, of course with full precautions, if eye is still largely intact and vision prognosis is good.[4-6]

NPO Status

Rule of 8-6-4-2, i.e. eight hours for solids, six for non clear liquids, four for breast milk and two for water or clear liquid are followed. In the patients who have had trauma or received opioids, gastric emptying time may be delayed up to 24 hours. Time interval between the last meal and injury is important. If trauma occurs soon after a large meal, the patient still has a full stomach after the standard 6 hour fast. Alcohol also delays the gastric emptying. If surgery is necessary in a patient with full stomach then principles of rapid sequence induction may be followed.[4]

Intraocular Pressure (IOP)

Normal IOP is that between 10 mm Hg to 20 mm Hg.

It is important for anesthesiologist to control IOP. A rise in IOP will impair the operating conditions and may cause an expulsion of intraocular contents with permanent damage to the eye. On the other hand a mild reduction in IOP will improve the operating condition for the surgeon.

Factors Increasing IOP

1. *Arterial pressure*: Rise in blood pressure causes increase in IOP. Though this is transient, it can cause detrimental effect on the eye. Hypotension (systolic BP below 90) may reduce IOP as choroidal blood volume (CBV) decreases.[1]
2. *Central venous pressure (CVP)*: Flexion of the head, a valsalva maneuver, coughing, bucking, straining and vomiting causes marked increase in the intraocular pressure by raising CVP.[6]
3. *Changes in blood gas:* Hypoxia and hypercarbia causes vasodilatation of intraocular blood vessels raising the IOP.
4. *External pressure:* Face mask can cause pressure on eye ball and raise IOP. So gentleness is required during the procedure. A forceful lid squeeze can increase IOP to more than 50 mm Hg.[7]

5. *Suxamethonium:* It causes increase in IOP. The precise mechanism is unknown but may be due to contraction of extraocular muscles during the fasciculation or dilatation of blood vessels. It produces 7 to 12 mm Hg increase in intraocular pressure. The effect is maximum at 2 to 4 minutes and returning to normal within 7 minutes.[8,9]
6. *Ketamine:* It produces a small increase in IOP. But intramuscular ketamine may even lower IOP in children. It causes blepharospasm and nystagmus, which make it anyway unsuitable for ophthalmic surgery.
7. *Laryngoscopy and endotracheal intubation:* Both raise IOP. An increase in IOP during induction may cause expulsion of intraocular contents and the permanent damage to the eye.
8. *Miscellaneous stimuli:* Nasogastric tube insertion can cause multifactorial rise in IOP. Coughing, straining, or vomiting can increase IOP to 30 to 40 mm Hg.[10]

Factors Lowering IOP

When the globe has been penetrated the IOP is reduced to atmospheric pressure:

1. Induced hypotension <90 mm of Hg.
2. Head up tilt lowers venous pressure and thereby IOP.
3. Hypocarbia lower IOP by constricting the choroid vessels.
4. Induction agents reduce IOP. Deep inhaled or thiopental anesthesia causes a dose-related reduction in IOP by 30 to 40 percent.[10]
   - Thiopental significantly decreases IOP by its central depressive effect on the diencephalic control of IOP. Etomidate also lower IOP.
   - The effects of propofol are similar to that of thiopental.
   - Major tranquilizers, including intravenous doses of midazolam (0.03 mg/kg), lower the IOP.
   - Inhalational agents cause fall in IOP which is proportional to the inspired concentration.
   - Dexmedetomidine, could attenuate increase in the IOP after succinylcholine and intubation along with hemodynamic fluctuations.[11]
5. Nondepolarizing muscle relaxants reduces the IOP.
6. Diuretics: Mannitol by osmotic diuresis and acetazolamide by inhibiting aqueous humor production.

Preoperative Assessment

Timing of injury: NPO status, i.e. interval between meal and timing of injury is important.

Type of injury: If there is any concern about the possibility of a penetrating injury, no pressure is applied to the eye because it may cause extrusion of the intraocular contents. The patient is kept under constant observation so that there is no pulling, pressing or scratching of the injured eye. The traumatized eye is covered with a soft, sterile dressing that does not put pressure on the eye.[3,6]

Coexisting injuries: As with all trauma cases, priority is given to any associated abdominal, thoracic, or cerebral trauma; and the patient is stabilized before considering any repair of the ocular injury. Life-threatening problems should be dealt before sight threatening problems.

Comorbid conditions: Eye trauma requiring surgery may also be associated with other injuries that may or may not need surgery. Patients with other disease process such as diabetes or ischemic heart disease should be optimized prior to surgery if the time allows.[6] History of medications and allergies should be obtained.

Local Anesthesia

Routinely extraocular, anterior segment and vitreoretinal eye surgeries are performed using local anesthesia. However, it has many disadvantages and practical problems for emergency cases:

- Uncooperative, intoxicated, anxious patients and children may not lie flat, and still for the duration of the procedure. Children and younger adults tend to tolerate surgery with a local anesthesia technique poorly compared with elderly patients.[6,12]
- Spread of local anesthetic agents is poor in patients with eye and orbital infections.
- Injection of local anesthetic using peribulbar and retrobulbar techniques is associated with an increase in intraocular pressure which may lead to vitreous loss.
- Ocular compression after the block is also not an option if the patient has an open eye injury.

In some patients it may be possible to operate on small open eye injuries using topical anesthesia, subtenon blocks or a careful peribulbar or retrobulbar block.

Sedatives

If sedation is to be used then small doses of a short acting agent such as midazolam should be given. Diazepam in small doses may also be an option. Some anesthetists use small doses of alfentanil or fentanyl.[6] Propofol in small 10 mg increment doses can also be

used especially prior to performing a local anesthetic eye block.

The key to good sedation is to maintain verbal contact with the patient. Oversedation can easily turn a cooperative patient into a difficult to manage patient due to airway problems and patient confusion. Sedation should not be used as an alternative to a general anesthetic in a patient with a full stomach. If a patient develops pain during surgery using a local anesthetic technique the patient requires analgesia and not sedation. The surgeon should supplement the block using local anesthesia or small doses of intravenous analgesia should be given.

General Anesthesia

Adults

Premedication

Adults will allow intravenous line placement. Early administration of an H_2 receptor antagonist such as ranitidine (1 mg/kg IV) will decrease the gastric volume and provide some protection. Use of anticholinergic and antisialogogue agent such as glycopyrrolate (0.004 mg/kg iv) is useful for preventing the oculocardiac reflex. Traction on the extraocular muscles or pressure on the globe causes bradycardia, atrioventricular block, ventricular ectopy, or asystole. It is especially seen with traction on the medial rectus muscle, but can occur with stimulation of any of the orbital contents, including the periosteum. The reflex is trigeminovagal. The afferent limb is from orbital contents to ciliary ganglion to ophthalmic division of the trigeminal nerve to the sensory nucleus of the trigeminal near the forth ventricle. The efferent limb is via the vagus nerve to the heart. In the event of arrhythmia, the anesthesiologist first should ask the surgeon to stop manipulations. The ventilatory status is assessed. If significant bradycardia persists or recurs, intravenous atropine is administered in 7 µg/kg increments. Rarely, severe bradycardia or asystole occurs. Pretreatment with intravenous atropine or glycopyrrolate can be effective. Pretreatment may be indicated in patients with a history of conduction block, vasovagal responses, or α-blocker therapy.[1,12]

Induction

For an emergency ophthalmic procedure the patient must be considered to have a full stomach and requires a rapid-sequence induction. Care must be taken not to exert pressure on the eye by the face mask during preoxygenation. Preoxygenation for 3 to 4 minutes with the patient holding the mask himself can build confidence and relieve anxiety.

Before rapid-sequence intubation, several precautions must be taken to blunt the pressor, IOP response to laryngoscopy and tracheal intubation. When the globe has been penetrated the IOP is reduced to surrounding atmospheric ambient pressure. Any attempt which causes increase in IOP may cause expulsion of the eye contents, may it be vomiting, Ryle tube insertion, placemeant of oxygen mask, laryngoscopy, intubation, bucking, coughing, raised arterial or venous pressure.

The patient is preoxygenated with 100 percent oxygen for 3 minutes. After administration of anticholinergic agent like glycopyrrolate (0.004 mg/kg), patient is sedated with intravenous midazolam (0.02 mg/kg). Intravenous administration of lidocaine (1.5 mg/kg) and remifentanil (0.7 µg/kg) 3 to 5 minutes before induction may help to attenuate the increase in IOP after tracheal intubation. A beta adrenergic receptor blocking drug such as labetalol (0.05 to 0.10 mg/kg) may also be useful in blocking the cardiovascular response to tracheal intubation, especially in patients with angina or hypertension.[6] Anesthesia is induced by propofol (2-2.5 mg/kg) or sodium thiopental (5-7 mg/kg) over 20 to 30 seconds, cricoid pressure (Sellick's maneuver) is applied, a nondepolarizing agent vecuronium (0.2 mg/kg) is given. Other nondepolarizing muscle relaxants, e.g. rocuronium (0.9 mg/kg) can also be used in sufficient and equipotent doses.[13-17] The depth of anesthesia is maintained by using O_2+N_2O+ sevoflurane. Endotracheal intubation is performed within 90 seconds in case of vecuronium and after 60 seconds with rocuronium.[15] A temptation of intubation too early with incomplete relaxation for performing laryngoscopy may stimulate coughing or bucking and cause sudden and significant rise in IOP. Etomidate is an imidazole derivative used in a emergency setting for rapid sequence induction of anesthesia, especially in elderly patients and patients who have cardiovascular compromise. It has a rapid onset of effect and a rapid offset even after a continuous infusion. Prolonged infusion results in inhibition of adrenocortical synthesis and potential mortality in intensive care unit (ICU) patients. The major advantage of etomidate is its minimal effect on the cardiovascular and respiratory systems. It may cause myoclonus which may ultimately cause rise in IOP and is associated with a high incidence of burning on injection, thrombophlebitis, and postoperative nausea and vomiting (PONV), limiting its popularity. The induction dose is 0.2 to 0.3 mg/kg.[1,18]

Use of succinylcholine as muscle relaxant is theoretically is contraindicated and controversial. But there are no published report of eye damage following its use. In rapid sequence induction, Libonati and coworkers[19] did not encounter aspiration of gastric

contents or extrusion of eye contents. Bourke[5] used succinylcholine successfully and carefully without encountering much problem. When an open globe injury has occurred, it is often associated with crying, Valsalva's maneuver, forceful blinking and rubbing the eyes. All these create a much larger rise in IOP than associated with the use of succinylcholine. Increase in IOP produced by succinylcholine can be attenuated by sedatives and the induction agents. Profound paralysis of the muscles of chest and thus prevention of cough will be the advantage.

Various modifications have been suggested to attenuate the rise in IOP caused by succinylcholine. These comprise of prior administration of diazepam, lignocaine, acetazolamide, self-taming dose of succinylcholine or a small dose of nondepolarizing muscle relaxant. The self-taming involves the injection of subparalytic doses of succinylcholine, i.e. one-tenth of a paralyzing dose prior to the administration of the paralyzing dose with a view to desensitize the neuromuscular junction to the subsequent large dose.[20]

Among the pretreatments tested, 0.06 mg/kg rocuronium followed by the intubating dose of 0.6 mg/kg is the best to prevent muscular fasciculations following succinylcholine injection.[15] The patient can be intubated after 60 seconds. The other agents used for pretreatment are vecuronium (0.01mg/kg) followed by intubating dose of 0.1 to 0.15 mg/kg which provides the ideal intubating condition after 90 seconds. The priming dose of cisatracurium is 0.01mg/kg and intubating dose is 0.25 mg/kg and the intubation can be performed after 90 seconds. For rapid sequence induction of anesthesia using propofol and fentanyl, rocuronium did not cause as great an increase in IOP as succinylcholine and may be an alternative in open eye injury cases.[13,16]

The muscular relaxation must be monitored with train of four to prevent accidental coughing caused by carinal stimulation. Whenever a paralytic agent is used, the train of four stimulus is the test used to measure the degree of neuromuscular blockade. Do a baseline measurement before paralytic agent is started to determine current necessary to obtain twitch. The trachea is often intubated when the response to TOF stimulation disappears.[1]

Maintenance

Good monitoring of vital parameters should be done. Patient should be maintained with controlled ventilation with O_2+N_2O+ sevoflurane or isoflurane during the procedure aiming for low to normal end-tidal carbon dioxide. During the procedure adequate depth of anesthesia should be maintained to avoid any movement of the patient. A little head up tilt is helpful in further lowering the IOP.

Emergence and Extubation

At the end of the procedure once airway protective reflexes have returned and the patient starts breathing spontaneously, the patient should be extubated on their side. To avoid coughing and to have smooth emergence from anesthesia lidocaine (1.5 mg/kg IV) or remifentanil (0.5 µg/kg) should be administered 5 minutes before the awakening. Application of lidocaine spray or gel may be helpful but with the risk of numbness of vocal cords and some chances of aspiration.[6] Postoperative nausea, vomiting and pain should be kept to a minimum as they can cause increase in IOP. The antiemetic agent ondansetron (0.1 mg/kg) is administered 30 min prior to the extubation.

Children

Special care is needed in the children having open eye injury. Pain, crying, rubbing of the eyes, anxiety are the causes of concern. The patient should be nicely sedated.[3] Whenever there is doubt of head injury, narcotics should be avoided. Topical instillation of local anesthesia for conjunctiva and cornea may be helpful for relieving the pain for sometime.

Like adults all precautions have to be taken for full stomach. However, small children may not allow insertion of IV cannula and hence one may not be able to administer antacids and prokinetics. If such is the case, one should apply topical anesthetic cream (EMLA) while assessing the child. By the time other preparations are through, it would act and then one can secure intravenous access. Other alternative is to do gentle induction of anesthesia by mask (with 7 to 8% of sevoflurane) with good preoxygenation for a period of time without disturbing the patient. However, positive pressure must be avoided.

The ideal induction should avoid making the child cry or struggle. If the child is fasted then an 'elective' induction can be performed. A sedative premedication is given and a smooth gaseous or intravenous induction is performed. A nondepolarizing relaxant is used to facilitate endotracheal intubation. A bolus of opioid, e.g. fentanyl 1 to 2 µg/kg IV or lidocaine 1.5 mg/kg IV may be given to attenuate the rise in IOP in response to laryngoscopy.

A hysterical child with a penetrating eye injury and a full stomach provides an anesthetic challenge due to the importance of avoiding increases in intraocular

pressure and yet minimizing the risk of aspiration. The screaming and crying can lead to tremendous increases in intraocular pressure. Attempting to sedate children with rectal suppositories or intramuscular injections, however, often heightens their state of agitation and may worsen the eye injury. An ideal strategy would be to administer enough sedation painlessly to allow placement of an intravenous line yet maintain a level of consciousness adequate to protect airway reflexes. The opioid-containing lollipops are acceptable alternatives. Primary aim should be to avoid aspiration and related complications.[21]

This operation may be of long duration. Maintenance IV fluids are required. Dextrose containing IV fluids are used. Temperature is monitored and a warming mattress is used. A prophylactic antiemetic ondansetron 0.1 mg/kg IV is given since the likelihood of PONV is high. Emergence from anesthesia and extubation should be as smooth as possible to minimize coughing or straining as that will raise IOP.

REFERENCES

1. Feldman MA, Patel A. Anesthesia for eye, ear, nose and throat surgery. In: Miller RD (Ed). Miller's Anesthesia, 7th edn. Philadelphia: Churchill Livingstone 2010;2357-88.
2. Meyer RF, Sweet ER. Chemical injuries of the eye. Journal of the American College of Emergency Physicians 1973; 2(3):194-5.
3. Rukaiya KA, Hamid. Pediatric eye emergencies. Anesthesiology Clinics of North America 2001;19(2):257-64.
4. Wilson A, Soar J. Anaesthesia for Emergency Eye Surgery 2000; Issue 11: Article 10:1-2. httpl/www.nda.ox.ac.uk/wfsa/html/u11/u1110-0L, html 1/19/2008(s).
5. Bourke DL. Open eye injuries. Anesthesiology 1985;63(6): 727.
6. Gupta S, Mehta A. Open Eye Injury with Full Stomach. The Internet Journal of Anesthesiology 2010;22(2).
7. Gouws P, Moss EB, Trope GE, Ethier CR. Continuous intraocular pressure (IOP) measurement during glaucoma drainage device implantation. J Glaucoma 2007;16:329-33.
8. Panday K, Badola P, Kumar S. Time course of intraocular hypertension produced by suxamethonium. Br J Anaesth 1972;44:191-5.
9. Taylor TH, Mulcahy M, Nightingale DA. Suxamethonium chloride in intraocular surgery. Br J Anaesth 1968;40:113-8.
10. Kohli R, Ramsingh H, Makkad B. The anesthetic management of ocular trauma. Int Anesthesiol Clin 2007; 45:83-98.
11. Fujii Y, Tanaka H, Toyooka H. Granisetron and dexamethasone provides more improved prevention of post operative emesis than granisetron alone in children. Can J Anaesth 1996;43:1229-32.
12. Mcgoldrick KE, Gayer SI. Anesthesia for ophthalmological surgery. In: Barash PG, Cullen BF, Stoelting RK, (Eds). Clinical Anesthesia. 6th edn. Philadelphia: Wolters Kluwer. Lippincott Williams and Wilkins 2009;1321-45.
13. Chiu CL, Jaais F, Wang CY. Effect of rocuronium compared with succinylcholine on intraocular pressure during rapid sequence induction of anaesthesia. British Journal of Anaesthesia 1999;82(5):757-60.
14. Malik P, Rao S, Malhotra N, Chugh JP, Hooda S. Comparative evaluation of effect of rocuronium and succinylcholine on intraocular pressure. Journal of Anaesthesiology and Clinical Pharmacology 2004; 20(3):255-61.
15. Martin R, Carrier J, Pirlet M, Claprood Y, Tetrault JP. Rocuronium is the best non-depolarizing relaxant to prevent succinylcholine fasciculations and myalgia. Canadian Journal of Anaesthesia 1998;45(6):521-5.
16. Vinik H. Intraocular pressure changes during rapid sequence induction and intubation: A comparison of rocuronium, atracurium, and succinylcholine. J Clin Anesth 1999;11(2):95-100.
17. Perry JJ, Lee JS, Sillberg VAH, Wells GA. Rocuronium versus succinylcholine for rapid sequence induction intubation. Cochrane Database of Systematic Reviews 2008; Issue 2. Art. No: CD002788. DOI: 10.1002/14651858.CD002788.pub2.
18. Thompson MF, Bean P. Anaesthesia and intraocular pressure: A comparison of total intravenous anaesthesia using etomidate and conventional inhalational anaesthesia. Anaesthesia 1968;37:758-61.
19. Libonati MM, Leahy JJ, Ellison N. The use of succinylcholine in open eye surgery. Anesthesiology 1985; 62:637-40.
20. Murphy DF. Anaesthesia and intraocular pressure. Anaesth Analg 1985;64:520.
21. Morgan GE, Mikhail MS, Murray MJ, (Eds). Anesthesia for ophthalmic surgery. In: Clinical Anesthesiology 4th edn. New York: Tata Mcgraw-Hill 2009;826-36.

Section VII

Neurosurgical Emergencies

24 Anesthesia for Neurosurgical Decompression

Hemangi S Karnik, Naina P Dalvi, Dhawal R Wadaskar

KEY POINTS

- The brain uses 20 percent of total oxygen and 25 percent of the glucose required by the body at rest.
- Very high increases in intracranial pressure (ICP) may lead to either reduction in cerebral blood flow or cerebral herniation.
- The Autoregulation keeps cerebral blood flow (CBF) constant between mean arterial pressures of 65 to 150 mm Hg.
- Most intravenous anesthetic, analgesic, and sedative drugs except ketamine, reduce CBF and cerebral metabolic rate (CMR) and do not adversely affect ICP.
- Inhaled anesthetic agents produce a dose dependant increase in cerebral blood flow.
- The net effects of inhalational anesthetics on blood flow are the overall balanced result of the drug's individual direct effects on cerebral vasomotor tone and on the CMR.
- During preoperative assessment, special attention should be given to the patient's level of consciousness, presence or absence of evidence of increased ICP, the extent of focal sensory and motor neurologic deficits.
- It is important how the anesthetic agents are given and which other measures are taken to maintain CPP and control ICP and blood pressure.
- Use of N_2O has been found to be associated with an increased risk for the development of delayed ischemic neurological deficits.
- Mild hyperventilation to decrease the $PaCO_2$ to near 35 mm Hg is appropriate.
- Extreme neck flexion and rotation can obstruct venous drainage and should be avoided.
- Normovolemia, guided by central venous pressures, should be maintained.
- In anesthetised, vasodilated patient, any change in position involves sudden change in circulatory dynamics. Partly this is minimized by adequate hydration, use of elastic stockings and gradual positioning with simultaneous fluid loading.
- The amount of air trapped during venous air embolism (VAE) can be limited by increased intravascular volume to maintain higher CVP, use of PEEP, avoiding N_2O or flooding the surgical field with saline.
- In posterior fossa lesions, pressure on the brain stem may produce bradycardia, irregular respiration and lower cranial nerve palsies.
- In acute hydrocephalus, rapidly rising intracranial pressure can deteriorate the patient's condition fast.
- Cerebral Abscesses may be present in any area of the brain, often occurring secondary to adjacent extracranial infection, previous trauma with open wound, meningitis or due to dissemination of distant systemic infections including tuberculosis. They are common in children with cyanotic heart disease.
- During spinal surgeries, blood pressure should be controlled balancing the spinal cord perfusion with the requirement to produce a bloodless surgical field.

A number of neurosurgical diseases require to be treated within minutes or few hours to prevent permanent neurological disability or death. Such patient may come up for emergency or urgent neurosurgical interventions in precarious state, often without preoperative optimization. The administration of anesthesia to these patients requires an understanding of the basic neurophysiology and effects of anesthetic agents on intracranial dynamics.

APPLIED NEUROPHYSIOLOGY

The human brain constitutes only 2 to 3 percent of total body weight in adults, where as it requires about

15 percent of resting cardiac output.[1] This disproportionately large blood supply is needed by brain to meet its high metabolic demands. The brain uses 25 percent of the glucose and 20 percent of total oxygen required by the body at rest.[2] In spite of such high demands for oxygen and glucose by brain, it can store energy only to a minimal extent and hence it is essential for each part of the brain to receive adequate, continuous blood supply. Cessation of blood supply even for few minutes can cause permanent brain damage. Therefore, it is important for an 'occasional neuroanesthesiologist' to know about basic neurophysiology to understand the effect of various anesthetic agents and different stimuli on brain, to protect a diseased brain from these stimuli and to prevent it from further insult during perioperative period.

Intracranial Pressure (ICP)

The cranial cavity is a closed structure with a fixed volume. It contains the brain, blood and cerebrospinal fluid (CSF). If there is increase in volume of any one of these, volume of another should reduce (Monro-Kellie hypothesis). Usually in presence of a space occupying intracranial lesion, first cerebrospinal fluid from cranium will shift to spinal subarachnoid space without any increase in ICP. Next the venous blood will reduce in quantity. However in case of very large intracranial masses or when these compensations fail, ICP will increase making the brain prone for ischemia and cerebral herniation. Normal ICP in adults is 5 to 15 mm Hg in horizontal position.[3] The pressure volume curve (Fig. 24.1) shows relationship between intracranial pressure and volume expansion. In a patient with intracranial lesion, as compensatory mechanisms have already been exhausted, even a small increase in volume as may occur during hyperdynamic response to tracheal intubation can produce very high increases in ICP.

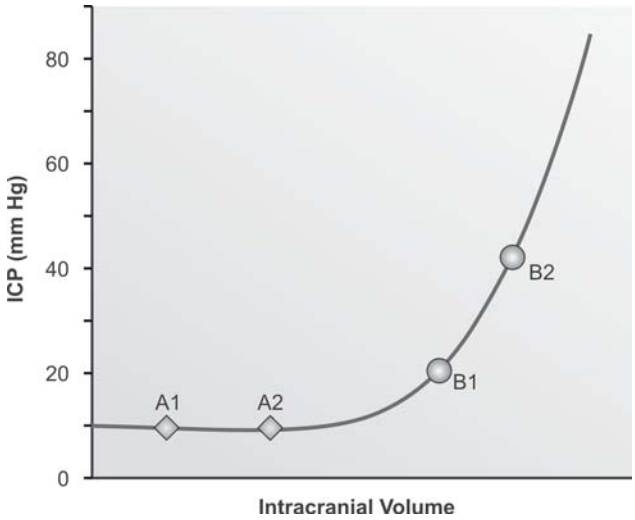


Fig. 24.1: Intracranial pressure volume curve showing effect of volume increase on intracranial pressure. A1 and A2 – In normal person. B1 and B2 – In patient with Intracranial SOL *(For color version, see Plate 3)*

The cerebral perfusion pressure is the pressure at which blood enters the cerebral arteries. The cerebral perfusion is dependant on the mean arterial pressure (MAP) and the ICP.

Cerebral perfusion Pressure (CPP) = MAP – ICP

Therefore when ICP rises, correspondingly cerebral perfusion reduces, if blood pressure remains unchanged.

Intracranial Pressure Monitoring

ICP monitoring helps in guiding therapy in patients who are at risk of secondary brain damage. It is also useful in deciding the need of specific surgical intervention.

Various modalities for ICP measurements are intraventricular or subdural catheters, subdural bolt, intraparenchymal sensor or epidural transducer. Intraventricular catheter is most common method to directly measure ICP. Even though it is more invasive with increased risk of infection, it is a reliable, relatively less costly technique. It can also be used therapeutically to drain CSF to reduce pressure from time to time. The subdural catheters are easier to place, but may show damping with cerebral edema. The new transducer tipped systems, though costly, are accurate in measuring CSF or intraparenchymal pressure. These can be placed in tissues 'at risk' and may be placed for extended period of time to assess effectiveness of therapy. ICP monitoring is commonly indicated in severe head injury, benign intracranial hypertension with papilledema, brain swelling, subarachnoid hemorrhage, intracerebral hematoma or cerebral ischemia and postoperative patients in whom persistent high ICP is expected.

Intracranial Hypertension

Intracranial Hypertension is defined as a sustained elevation in pressure above 20 mm Hg. Intracranial hypertension may produce cerebral ischemia or cerebral herniation and therefore is an acute emergency, requiring immediate reversal. The common causes are cerebral edema, intracranial space occupying lesions, hydrocephalus, pneumoencephalus and cerebral venous obstruction. Several variables can cause intracranial hypertension (Table 24.1). Common clinical presentation includes headache, nausea, vomiting, blurred vision, diplopia, altered consciousness and papilloedema. Arterial hypertension and bradycardia occur secondary to ischemia and pressure on brainstem. There may be

Table 24.1: Causes of intracranial hypertension

	Causes
↑ **Volume of CSF**	Hydrocephalus
↑ **Volume of Blood**	Venous obstruction, ↑ intrathoracic Pressure, ↑ $PaCO_2$, ↑ blood volume, ↓ PaO_2, some anesthetic agents, vasodilators, seizures
↑ **Volume of brain tissue**	Cerebral edema
Space occupying lesion	Intracranial hematoma, tumors, cysts

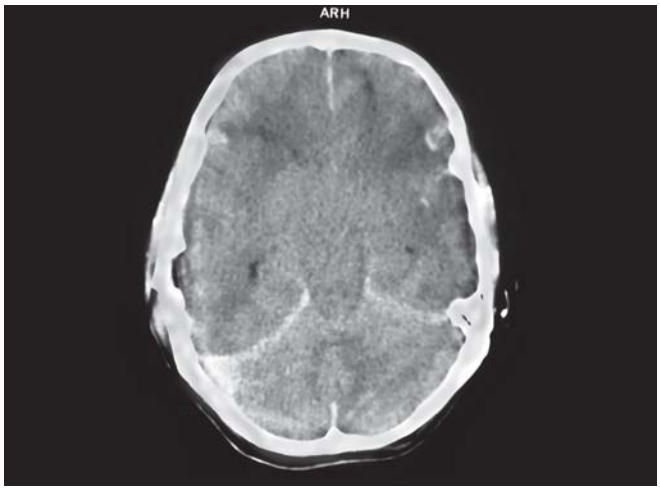


Fig. 24.2: CT scan showing cerebral edema

Chyne-Stoke respiration or sustained hyperventilation due to pons or midbrain involvement or shallow, rapid respiration when upper medulla is involved (Cushing's Triad).[4] On computed tomography obliteration of ventricles, loss of sulci or hypodensity in presence of edema may be seen with or without midline shift (Fig. 24.2).

Management: The goal of management is to reduce ICP to maintain adequate cerebral perfusion to prevent ischemia to brain tissues. This is done by ensuring a patent airway, adequate breathing and circulation. Additional general measures include removal of space occupying lesion, head up position, sedation, analgesics, hyperventilation, administration of mannitol, hypertonic saline, CSF drainage or pressor agents to maintain CPP. Decompressive craniectomy may be indicated in severe cases.

Cerebral Herniation

When pressure gradient becomes large enough to overcome resistance, shift of intracranial contents from one compartment to another occurs. This is known as

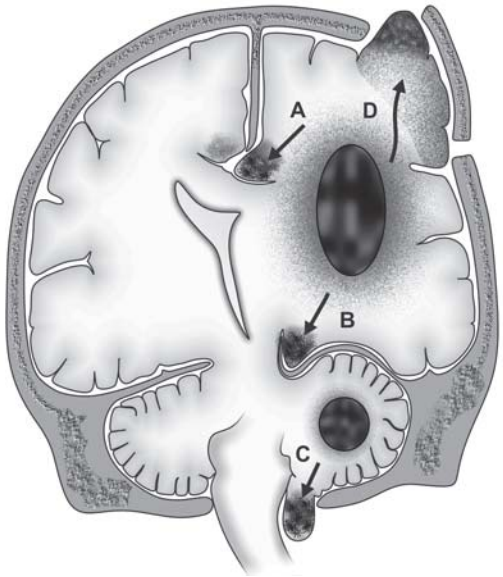


Fig. 24.3: Types of cerebral herniation: (A) Subfalcine, (B) Uncal, (C) Tonsillar, (D) Transcalvarial

cerebral herniation. There are four types of cerebral herniations[2] (Fig. 24.3).

- Subfalcine
- Transtentorial- uncal, upward or central (coning)
- Tonsillar
- Transcalvarial.

Within a very short time span, an impending cerebral herniation can progress to irreversible neurological damage or death. Therefore, once it is diagnosed, immediate attempts should be taken to reduce the brain bulk by medical or surgical measures.

Cerebral Metabolism

About 60 percent of energy by the brain is consumed for electrophysiologic functions. Remaining amount is used for cellular homeostasis. The main substance used by the brain for energy production is glucose. Normally, when oxygen supply is adequate, glucose is converted to pyruvate by normal glycolysis and then further metabolized by oxidative phosphorylation through the Kreb's cycle to produce energy. Each molecule metabolized will produce 38 ATPs. If oxygen supply is insufficient or absent, by anerobic pathway, pyruvate gets converted to lactic acid. This produces H^+ ion as byproduct and can generate only 2 ATPs per molecule. Presence of acid ions enhances cellular damage, which will be greater if more glucose is available in hypoxic situations. Therefore, in patients who are at high risk of hypoxic injury, intraoperative hyperglycemia is

associated with long-term cognitive and neurological dysfunction.[5]

Normally brain utilizes 3.5 ml of O_2/min/100 g brain tissue[1] (Range: 3.0—3.8)[6] and 5.5 mg of glucose/min/100 g.[7]

In presence of hypoxia, there will be rapid cessation of spontaneous electrophysiological activity.[7] This will reduce the brain oxygen demand significantly. The small amount of available energy is used to maintain the structure and internal function of the cells, predominantly to pump ions across the cell membranes. If this critical energy demand is also not met, the irreversible cellular damage will occur.

Cerebral Blood Flow (CBF)

The cerebral blood supply is provided principally by the internal carotid arteries (80%) and the vertebral arteries (20%) and the flow is linked through the circle of Willis. The brain receives average of 55 ml/100g/minute of continuous blood supply and 80 percent of it is supplied to the grey matter.[8] This blood supply needs to be balanced with the cerebral metabolic rate (CMR).

CBF is dependent on cerebral perfusion pressure (CPP) and cerebral vascular resistance (CVR).

Thus, CBF = CPP/CVR.

In a patient with CVP equal or less than ICP, CBF = (MAP – ICP)/CVR.

Thus, the CBF will change with any change in blood pressure or resistance. There are several chemical and neurohumoral mechanisms which play a regulatory role in maintaining CBF (Fig. 24.4).

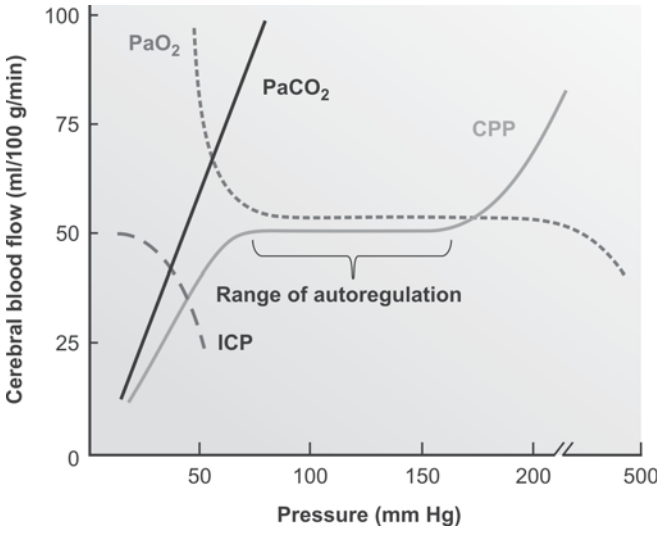


Fig. 24.4: Regulation of cerebral blood flow

Factors Affecting CBF

Autoregulation

Autoregulation is the ability of cerebral circulation to maintain a constant flow over a moderate range of perfusion pressures. It is the capacity of the cerebral circulation to adjust its resistance to maintain constant CBF over a wide range of mean arterial pressure (MAP). As blood pressure changes, the cerebral vasculature dilates or constricts to maintain CBF. The CBF remains constant between mean arterial pressure of 50 to 150 mm Hg.[8] Below this range, there is sharp reduction. When the pressure is increased beyond 150 mm Hg, there is steep increase in cerebral blood flow. In chronically hypertensive patients, this autoregulation limits appear to be reset at higher level and may produce ischemia at pressures which are considered adequate in a normal individual.[9] Various mechanisms that possibly contribute in governing autoregulation are vascular myogenic responses, endothelial factors, direct effects of metabolic by-products neural mechanisms.[10]

Carbon Dioxide (CO_2)

Carbon dioxide, being a vasodilator, is a powerful modulator of cerebral blood flow. CBF proportionately changes by 3 to 5 percent (1 to 2 ml/100 g/min) per mm Hg change in $PaCO_2$ within clinical range.[1] Therefore, hyperventilation reduces cerebral blood flow and severe hyperventilation may make the patient prone for cerebral ischemia. Hypoventilation will cause vasodilatation and increase the blood flow till $PaCO_2$ of 55 mm Hg when full vasodilatation is seen. This may increase the ICP in compromised patients.

Oxygen

The CBF remains nearly constant over wide range (60-300 mm Hg) of oxygenation. When PaO_2 falls below 60 mm Hg, CBF increases exponentially. In adults breathing 100 percent O_2, about 10% decrease in CBF is noted.[3] In ischemic hemisphere however, increasing the PaO_2 may not have any effect.

Cerebral Metabolism

Increase in cerebral metabolic activity is closely associated with proportionate increase in CBF (Metabolic coupling). Cerebral metabolic rate (CMR) increases during arousal, sensory stimulation, pain or hyperthermia. During seizures it increases severely. Reductions in cerebral metabolic rate may be observed during sleep, hypothermia, regionally after brain injury and globally with coma. Hypothermia causes reduction in CMR and offers

measurable degree of brain protection. At moderate levels (> 27°) of hypothermia, both autoregulation and CO_2 reactivity remains intact.[11]

Intracranial Pressure

The ICP influences the CBF through the cerebral perfusion pressure. Rise in ICP would lead to a fall in CPP.

Neural Regulation

The cerebral circulation is innervated by sympathetic, parasympathetic and sensory nerves. There is evidence that sympathetics play a role in reducing CBF by causing vasoconstriction. Moreover, sympathetic denervation produced by blockade of the stellate ganglion can increase CBF in humans.[12] However, its significance in controlling CBF is yet to be determined.

Blood Viscosity

Hematocrit is a major determinant of blood viscosity. In anemia, cerebral vascular resistance is reduced and CBF increases where as dehydration will increase the viscosity and reduce the flow. Therefore, hemodilution is useful to increase blood supply in focal cerebral ischemia.

Metabolic Substances

Nitric oxide (NO) is a short lived and diffusible molecular mediator and is known to be a potent relaxant of cerebral arteries.[13] Hydrogen ions and potassium ions are arteriolar vasodilator and increase CBF. Adenosine is a potent vasodilator and produce slower change in CBF.

Vasoactive Drugs

Vasodilators and calcium channel blockers also dilate cerebral vasculature and depending upon the blood pressure will affect the CBF. Sympathetic agonist and antagonist agents can cause direct effect on cerebral vasculature or may act indirectly via autoregulation by causing changes in systemic blood pressure. In a patient with intact autoregulation, these drugs will have minimal effects within the autoregulation range.

Effect of Anesthetic Agents on Cerebral Physiology

During a neurosurgical procedure, there are simultaneous stimuli and drugs administration going on. Hence, individual drug effect on one parameter may get complicated by multiple factors like blood pressure, hypocapnia, surgical stimulation, etc. In general, most anesthetic agents have favourable effects on cerebral physiology. Table 24.2 shows comparative effects of commonly used anesthetic agents on cerebral physiology.

Table 24.2: Comparative effects of anesthetic drugs on cerebral physiology

Drug	*CBF*	*$CMRO_2$*	*ICP*
N_2O	↑	↑	↑↑
Halothane	↑↑↑	↓↓	↑↑
Isoflurane	↑	↓↓↓	↑
Enflurane	↑↑	↓	↑↑
Savoflurane	↑	↓↓↓	↑↑
Desflurane	↑	↓↓↓	↑↑
Barbiturates	↓↓↓	↓↓↓↓	↓↓↓
Etomidate	↓↓	↓↓↓	↓↓
Droperidol	↓	=	=
Propofol	↓↓↓↓	↓↓↓	↓↓
Benzodiazepines	↓	↓↓	↓
Opioids	=	=	=
Ketamine	↑↑	=	↑

Inhalational Agents

Inhaled anesthetic agents produce a dose dependant increase in cerebral blood flow. Sevoflurane and desflurane have less pronounced effect than isoflurane. Halothane causes most vasodilatation. The increase in blood flow is generalized throughout all parts of the brain. Both halothane and isoflurane increase CBF at 0.5 and 1 MAC concentrations. Addition of N_2O further increases the CBF.[14,15] Nitrous oxide, when administered alone, increases both CBF and CMR. However, when used with another inhalational agent, it further increases CBF without changing CMR.

Inhaled anesthetics retain CO_2 reactivity but impair autoregulation in a dose-dependent manner. Halothane, at concentrations greater than 1 percent, nearly abolishes cerebral autoregulation. Desflurane at 1 MAC and above causes marked and significant impairment in auto-regulation and at 1.5 MAC it nearly abolishes it.[8] Hypocarbia may be less effective in decreasing ICP with desflurane compared with other inhaled agents. The carbon dioxide reactivity is better preserved with isoflurane and sevoflurane than with propofol.[16] N_2O impairs cerebral autoregulation and thus increases CBF.[17]

Halothane, desflurane, sevoflurane, and isoflurane produce dose-dependent decreases in CMR. Isoflurane produces the greatest depression (up to 50% reduction), whereas halothane has the least effect (< 25% reduction).[1]

Inhalational agents alter but do not uncouple the normal relationship of CBF and CMR. The combination of decreased CMR (metabolic demand) with an increased CBF (metabolic supply) has been termed luxury perfusion. This state may be beneficial during induced

hypotension and supports the use of an inhalational agent, particularly isoflurane, during this technique.

The net effects of inhalational anesthetics on blood flow are the overall balanced result of the drug's individual direct effects on cerebral vasomotor tone and on the cerebral metabolic rate (CMR). A decrease in metabolic rate induces cerebral vasoconstriction, which counteracts the direct vasodilatory effect of the drug. These two parameters can get affected to a different extent at different concentrations. Thus, despite the direct cerebral vasodilatory effects of inhalational anesthetics, CBF may remain relatively unchanged at concentrations up to 1 MAC.[18]

The net effect of inhalational anesthetics on ICP is secondary to changes in cerebral blood volume and $PaCO_2$. Studies have indicated that desflurane can cause greater ICP increases than isoflurane in patients with altered intracranial elastance. Desflurane in 1 MAC concentration significantly reduces autoregulation and almost abolishes it at 1.5 MAC concentrations.[8]

In presence of focal ischemia a circulatory steal phenomenon may occur with these agents. Inhalational agents can increase blood flow in normal areas of the brain but not in ischemic areas, where arterioles are already maximally vasodilated. The end result may be a redistribution of blood flow away from ischemic to normal areas.[8]

Intravenous Agents

Thiopentone sodium causes dose dependant reduction in CBF and CMR. In anesthetic doses it produces about 30 percent reduction in both and in larger doses produces complete EEG suppression with 50 percent reduction in CBF and CMR. Barbiturates can substantially reduce MAP, which, if not controlled, can reduce CPP. Barbiturates also reduce elevated ICP and control seizure activity.

Propofol will produce dose dependant reduction in CBF and metabolism. Propofol significantly increases, and sevoflurane significantly decreases cerebral vascular tone as assessed by changes in zero flow pressure. Thus, despite moderate decreases in mean arterial blood pressure, the estimated cerebral perfusion pressure is preserved during sevoflurane, but not during propofol anesthesia.[19]

As barbiturate-induced cerebral vasoconstriction occurs only in normal areas, there is redistribution of blood flow from normal to ischemic areas in the brain (Robin Hood, or reverse steal phenomenon). The cerebral vasculature in ischemic areas remains maximally dilated and is unaffected by the barbiturates. Therefore, such agents may be beneficial in presence of focal ischemia.

The cerebral effects of an anesthetic drug are further modified by its effect on the blood pressure. When a drug causes hypotension in the presence of preserved cerebral autoregulation, cerebral vessels dilate, resulting in an increase in cerebral blood volume and possibly an increase in ICP.[18] The administration of etomidate is associated with the least cardiovascular side-effects compared with other anesthetic drugs. It may decrease ICP while preserving CPP.[18]

Opioids in clinical doses, produce minimal decrease in CBF and $CMRO_2$ and cause minimum effect on ICP, if ventilation is maintained.

Pathophysiology of Cerebral Ischemia

Global Ischemia

Global ischemia develops due to complete cessation of cerebral blood flow. Cerebral ischemia develops at a CBF of 20 to 25 ml/100g/minute.[20] When CBF reduces below 18 ml/100 g/min, brain cells are at risk of electrical failure and membrane failure and cell death occurs at flows less than 10 ml/100 g/min.[20,21]

In presence of ischemia, an inflammatory cascade is initiated (Fig. 24.5). These processes include failure of ATP dependant transmembrane ion pumps, leading to increased intracellular Na^+ and Ca^{++}, which depolarize the neurons. This causes further neuronal damage by releasing excitatory amino acids, free fatty acids and free radicals. These chemicals produce intense vasoconstriction which may persist even after reperfusion starts. Furthermore tissue edema develops further reducing the blood flow. The cerebral edema can occur due to different mechanisms.[4]

- *Cytotoxic edema*: It is usually caused by impaired cellular transport of ions and fluids.
- *Vasogenic edema*: Extracellular edema secondary to increased permeability of blood- brain barrier.
- *Interstitial edema*: Occurring secondary to osmotic and oncotic differences between blood and brain tissue.

Commonly, all three types are present together.

Two major independent processes may start during ischemia that can lead to neuronal death.[7]

A. *Necrosis*: It occurs in response to severe ischemic damage. As the ischemic cell gradually disintegrates, it causes activation of microglia and the immune response. This activates and recruits neutrophils and macrophages that produce free radicals and damage adjacent neurons expanding the lesion in volume.[22]
B. *Apoptosis*: It is a programmed cell death, involving an active process. Apoptosis uses a metabolic process that is normally used to kill off unneeded neurons.

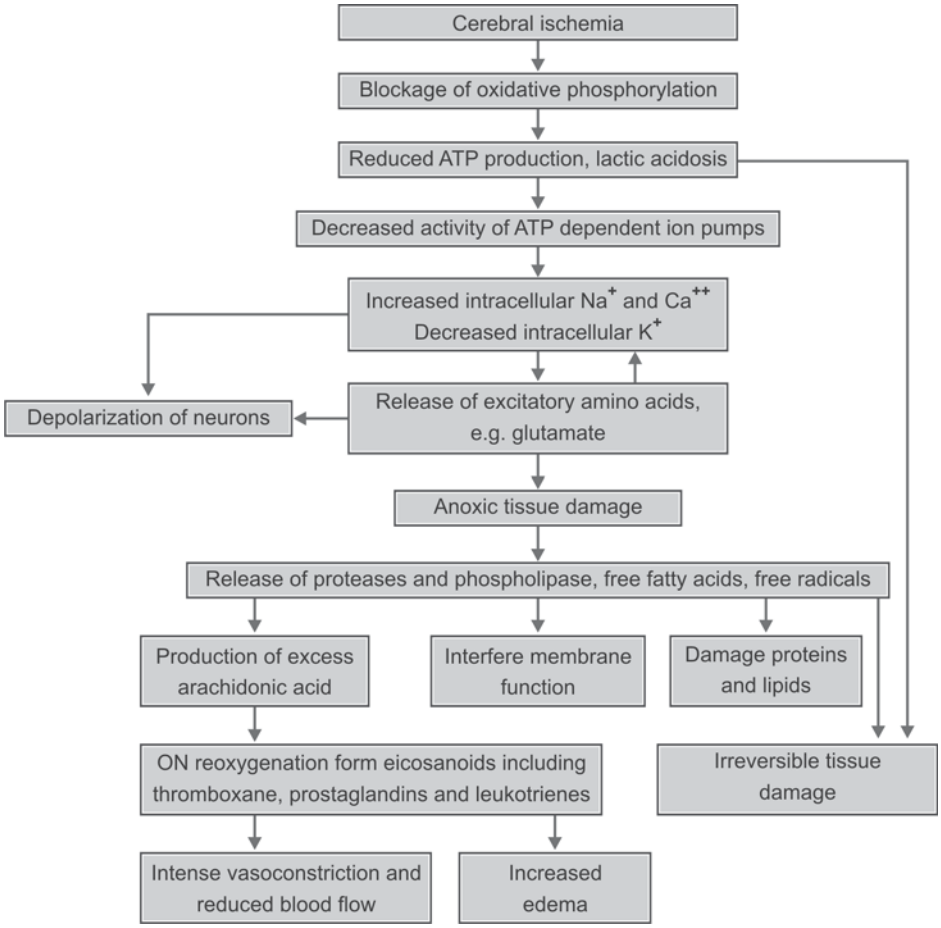


Fig. 24.5: Changes occurring during cerebral ischemia

This process is active during the development and can be reactivated when neurons are damaged subsequent to ischemia.[23] This regulated cell death does not injure adjacent neurons.

Focal Ischemia

Focal ischemic event occurs, usually due to a thromboembolic event or due to external compression. It is characterized by a 'core' area of severe ischemia/infarction which is surrounded by a 'penumbra' zone of moderate ischemia which has partial blood supply from collaterals (Fig. 24.6). The ischemia in the penumbra zone tissue may be reversed if blood flow is improved promptly. A severe insult, as seen in the core of an ischemic area, leads predominantly to necrosis, while less-compromised areas undergo a greater degree of apoptosis.

Generally, anesthetic agents with vasodilator properties can produce the steal phenomenon and the anesthetics producing vasoconstriction have the opposite

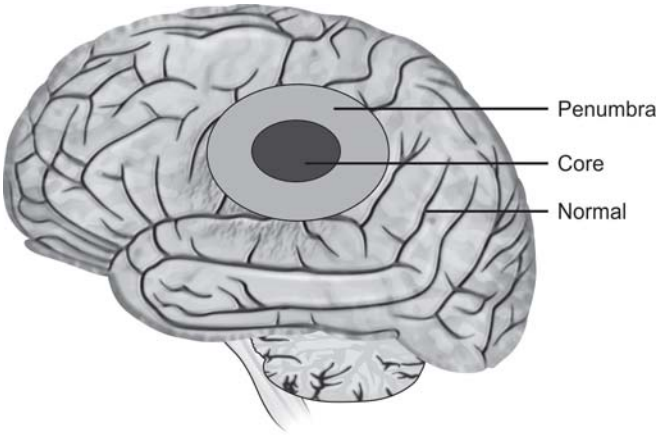


Fig. 24.6: Zones of injury

effect, resulting in inverse steal, and may protect the "At risk" brain tissue in case of focal ischemia.[7]

Therefore, neuroprotection is required during any surgical procedure in which blood supply to the brain is

hampered, either globally (as in some cardiac surgeries) or focally (carotid or cerebral vascular procedures.)

Cerebral Protection

Various clinical trials have been done to study various strategies in reducing risk of ischemic damage and offer neuroprotection during various surgeries where ischemic insult is likely to occur, e.g. major vascular or cardiac surgery, neurosurgery. Several strategies are mentioned in Table 24.3.

Protection essentially means prevention and mitigation of an anticipated insult likely to occur in given circumstances. Usually multipronged strategies are applied.

Cerebral Blood Flow Promotion[24]

This is employed in all patients as first line of management. Commonly done by:

- Maintaining adequate arterial blood pressure-using fluid therapy, if needed vasopressors and avoiding agents that may cause hypotension.
- Reducing ICP- Head up position (30°), mannitol (0.25-1 g/kg), furosemide, surgery. Mannitol is the most widely used osmotic agent in management of raised ICP. It reduces brain bulk due to its hyperosmolarity which draws water across intact blood brain barrier and reducing cerebral blood volume.
- Promoting cerebral vasodilatation.
- Avoiding hypocarbia.

Table 24.3: Strategies of brain protection

Goal	*Method*
Improving blood supply	Revascularization ↑ MAP, ↓ ICP ↓ Blood viscosity Reperfusion
Decreasing metabolic demands	Barbiturate, etomidate, propofol Control of seizures Control of infection Hypothermia
Reducing the effects of ischemic Injury	Mannitol Glucose control Blocking Na or Ca influx Free radical scavenging Blocking the release of excitatory amino acids Inhibiting proteins that activate or contribute to damage Activating proteins that induce repair or rescue from apoptosis and necrosis

Decreasing Metabolic Demands

Traditionally it is believed that if reduction in neuronal firing as measured by burst suppression in EEG is achieved, the cerebral metabolic rate will reduce and the ischemic area will become less vulnerable to the insult. This suppression reduces energy requirement, and this in turn should allow tissue to preserve energy balance better during a transient interruption of substrate delivery.[25]

Control of seizures: Seizures increase the metabolic activity several fold and worsen the ischemia. Antiepileptics are essential to prevent increased electrophysiological activity. They are commonly employed prophylactically in patients likely to develop or have history of seizures.

Anesthetic agents: They reduce brain activity by producing sedation and unconsciousness and thus decrease metabolism. Thus they are considered essential part of neuroprotection. All barbiturates and propofol cause burst suppression, in varying degrees.

These anesthetic agents will be protective only if given before or during ischemia but not if given after restoration of substrate delivery.[25] The efficacy of an anesthetic is dependent upon the severity and duration of the ischemic insult.

Barbiturates have been used extensively for brain protection. They offer modest neuroprotection in doses less than burst-suppression doses.[26] They are more effective on focal injury than during global ischemia. As very large doses of barbiturates are needed for EEG burst suppression, the recovery from its effect is delayed. It can cause peripheral vasodilatation and may be associated with low blood pressure after restoration of blood supply.

Propofol can reduce $CMRO_2$ by about 50 percent and offers sustained neuroprotection against mild insults. However, these effects are not sustained in moderate to severe ischemia.[26]

Etomidate remains the standard regimen for cerebral protection at several institutions as its beneficial effects are not accompanied by hypotension or myocardial depression. But there is clinical evidence that the standard propylene glycol formulation of etomidate induces more cerebral tissue hypoxia, tissue acidosis, and neurological deficits than an EEG-equivalent dose of desflurane.[7] It has been found that etomidate adversely affects focal cerebral ischemia, probably by inhibiting nitric oxide.

Various studies have shown that the inhalational anesthetics provide major improvement in ischemic outcome.[25] The dose required for this protection is within a clinically relevant range, with higher doses potentially worsening outcome.[27] They protect against both focal and

global ischemia. However, the improvement in outcome is transient in global ischemia,[28] whereas it is persistent in focal ischemia.[29] Sevoflurane has also been shown to provide long-term protection in one experimental model.[30]

Reducing the Effects of Ischemic Injury

Sodium channel blockers: Sodium influx is one of the first steps in ischemic cascade. Lignocaine blocks the Na channel and appears to reduce ischemic cell damage.[7] It also reduces postnecrotic injury by blocking apoptotic cell death pathway in the penumbra zone by blocking the sodium influx in the cell, which is the first step of ischemic cascade in experimental studies.

Calcium channel blockers: Magnesium appears to offer brain protection by causing cerebral vasodilatation as well as critically reducing entry of calcium into cells in experimental studies. However, till now there is no conclusive data for neurosurgical or head injury patients. Experiments with nimodipine have been inconclusive and failed to show any benefit in ischemic injury.[7]

Hypothermia: Hypothermia can reduce cerebral metabolism and thus can offer brain protection. Experimental studies have shown that reduction of temperature by 33 to 34°C can reduce the brain's vulnerability to ischemic injury. Therefore mild hypothermia in the operation theatre is advocated. However, this assumption has been proved experimentally and evidence of benefit in large clinical trials is still lacking. Currently it is not recommended as a tool to offer cerebral protection for neurosurgical procedures that do not require total circulatory arrest. Aggressive control of fever is recommended.

NMDA receptors blockade and release of excitatory amino acids: Ketamine is a potent N-methyl-d-aspartate (NMDA) receptor antagonist which has shown significant neuroprotection.[26] Remacemide reduces glutamate release, and so excitotoxicity, by blocking NMDA channels. N_2O antagonizes NMDA receptors and thus reduces damage from release of excessive glutamate release. However, it also blocks GABA release producing generalized disinhibition. Therefore, it has potential to cause neuronal damage rather than protection.[31] Different experimental studies have shown that N_2O increases ischemic effects on brain and also reduces efficacy of neuroprotection offered by thiopentone or isoflurane.[31,32] There is a strong doubt raised about its use in neuroanesthesia. Anesthetic agent Xenon produces its effects by blocking NMDA receptors like ketamine and can offer neuroprotection.[1] It has been shown to enhance neuroprotection in combination with either hypothermia or isoflurane.

Tirilazad: Tirilazad is a 21-aminosteroid used for vasospasm and stroke patients. Results from various studies have been inconclusive so far.[33]

THAM or tris-hydroxymethyl aminomethane is used in some centers to maintain normal pH in presence of intracranial hypertension, but its efficacy is still equivocal.[34]

Current evidence cautions against the use of prophylactic etomidate prior to temporary vessel occlusion, magnesium loading in ischemic stroke patients, intraoperative nitrous oxide and ketamine, intraoperative moderate hypothermia in subarachnoid hemorrhage (SAH) patients, postoperative nimodipine and tirilazad, and postoperative hypothermia in head-trauma patients.[7]

Brain Monitoring

Monitoring of cerebral oxygenation or metabolism are increasingly used perioperatively to assess adequacy of blood flow and to assess the effects of various pharmacological and surgical interventions on the same.

Assessment of Adequacy of CBF

Though several methods like Transcranial Doppler, thermal diffusion flowmetry, Xenon rCBF study or PET scan can monitor CBF qualitatively or quantitatively, their use in emergency neurosurgical procedures is very limited, as blood flow measured in one vessel or area may not reflect the status in other areas including the one which is prone to ischemic injury. Besides, rather than assessing the absolute blood flow or flow velocity, it is more important to know the adequacy of flow with respect to the cerebral metabolic demand. This can be done by assessing cerebral oxygenation invasively or noninvasively or indirectly by calculating arterial to jugular venous oxygen content difference ($AJvDO_2$) and jugular venous saturation.[35] However, these measures have more importance in ICU settings than during emergency neurosurgery.

Near Infrared Spectroscopy

This method noninvasively assesses cerebral oxygen by measuring absorption of light by the oxyhemoglobin in cortical vessels. The values obtained with near infrared spectroscopy (NIRS) represent primarily the oxygenation status of the venous compartment (75%). Declines of cerebral oxygen saturation of 25 to 30 percent seem to be associated with correctable neurological dysfunction. The technique measures local cortical oxygenation and does not indicate status of other areas.

Brain Tissue Oxygenation ($PbtO_2$)

Brain Tissue Oxygen Tension ($PbtO_2$) is partial pressure of oxygen in the extracellular fluid of brain and reflects availability of ATP production. This can be monitored by placing microsensors in the deep white matter of brain parenchyma. Monitoring $PbtO_2$ along with ICP and brain temperature is increasingly used where ICP monitoring is indicated. This technique can be used in ICU in cases of head injury and subarachnoid hemorrhage and intraoperatively during aneurysm and tumor surgeries.

Normal $PbtO_2$ is approximately 32 mm Hg. Studies have shown that the patients with $PbtO_2$ of less than 15 mm Hg had poor outcome.[36] Ideally the probe is placed in the "penumbra zone", so that the therapy can be directed to the tissues at risk. Brain tissue oxygen monitoring improves patient outcome by detecting secondary injuries that occur due to cerebral ischemia or hypoxia. Major drawback is invasiveness which is partially reduced by using specially designed triple lumen cranial access devices that allow simultaneous ICP, intracerebral microdialysis and $PbtO_2$ monitoring.

Interventions[37] Affecting $PbtO_2$:

- Increase in FiO_2 increases $PbtO_2$.
- Enhancing CPP by decreasing ICP or increasing MAP can increase $PbtO_2$.
- Hyperventilation may decrease brain tissue oxygenation to as low as 10 mm Hg despite dramatically decreased ICP and increased CPP.
- Increased temperature decreases $PbtO_2$.

Jugular Venous Oximetry

Jugular venous bulb oximetry involves placing a sampling catheter in the internal jugular vein, directed upwards, so that its tip rests in the jugular venous bulb at the base of the brain. As jugular bulb contains blood coming predominantly from brain, it is indicative of the oxygen extraction by the brain. This is used for patients with acute increases in ICP, who require hyperventilation therapy. Since hyperventilation is associated with reduction in CBF, falling jugular oxygen (jvO_2) indicates the need to employ other means to reduce ICP or increase CPP. Normally, $SjvO_2$ is in the range 50 to 75 percent. Factors affecting $SjvO_2$ include CBF, cerebral oxygen consumption, arterial oxygen content, and hemoglobin concentration.

$SjvO_2 = SaO_2 -$ (Oxygen consumption/Cardiac output $\times$ Hb $\times$ 1.39)

The $SjvO_2$ will fall when there is an imbalance between oxygen consumption and delivery. If the $SjvO_2$ falls below 50 percent (without a fall in arterial oxygen saturation - SaO_2), it implies either a fall in CBF or a rise in oxygen utilization (higher $CMRO_2$). An increase in $SjvO_2$ to 85 percent + implies either a hyperemia with a rise in cerebral blood flow, shunting of blood away from neurons or a decrease in $CMRO_2$ (impending cell death/brain death).

$AjvDO_2$ is the difference in oxygen content measured in simultaneously drawn blood samples from an artery and jugular bulb. Normal value is 6 ml O_2 /100 ml blood in adults.[38] The demand of oxygen by brain may change in various situations like hyperthermia, hypothermia, effect of anesthetic agents or ischemia. Hence, the values will change. A difference of 4 ml O_2/100 ml blood indicates hyperemia and values more than 9 ml O_2/100 ml blood indicates increased oxygen extraction.[2] This technique reflects global balance of oxygen and focal ischemia can be missed. It may also hamper venous drainage from brain, and raise ICP.

Cerebral Microdialysis (MD)

Microdialysis is a technique of measuring the concentration of neurochemicals in the extracellular space of the brain by fine semipermeable membrane tubes placed directly in the brain and perfusing them with a physiologic solution at ultra-low flow rates.[39] Smaller molecules diffuse from the ECF into the perfusion fluid, which is collected into microvials and then analysed for measurements of glucose, lactate, pyruvate, glutamate and glycerol. The measurement of lactate and pyruvate concentrations provide information on the extent of anerobic glycolysis and the extracellular lactate: Pyruvate ratio (LPR) is a reliable biomarker of tissue ischemia. Glutamate and aspartate are considered to have neurotoxic potential for their role in facilitating excessive calcium influx into brain cells. Glycerol is a useful MD marker of tissue hypoxia and cell damage. Thus, with the help of microdialysis, one can detect the harmful physiological events earlier, before clinical signs appear and secondary irreversible brain damage occurs.

ANESTHESIA FOR EMERGENCY CRANIOTOMY

Commonly emergency neurosurgical procedures are required to drain cerebrospinal fluid, to remove blood clots, to remove diseased brain tissue or to increase the capacity of cranium by removal of bone flaps and expanding duroplasty.

The anesthetic management varies with the individual procedure and may include a preplanned surgical position and use of positioning aids. It may also involve use of steroids, diuretics, anticonvulsants and antibiotics. The management depends upon the remaining intracranial compliance reserve, the need to manipulate blood pressure, $PaCO_2$, body temperature, anticipated blood loss and the likely use of neurophysiologic monitoring, which may place several limitations on the use of anesthetic agents or neuromuscular blocking agents and the risk of venous air embolism.

Preoperative Assessment

During the preoperative evaluation, the patient's overall medical condition must be considered and integrated into the formulation of an anesthetic management plan. A clear understanding of the intracranial pathology and problems associated with it during anesthesia and surgery is essential for proper planning and management. It should also include complete neurological evaluation, medications received and thorough assessment of associated diseases.

While performing complete neurologic examination special attention should be given to the patient's level of consciousness, presence or absence of evidence of increased ICP and the extent of focal sensory and motor neurologic deficits.

A patient with increased intracranial pressure may present with:

- Nausea and vomiting
- Altered levels of consciousness
- Mydriasis and decreased reactivity of pupils to light
- Papilledema
- Bradycardia, systemic hypertension, and breathing disturbances.

Absence of these signs does not rule out raised ICP. With advanced stages of intracranial hypertension, the patient may exhibit a depressed level of consciousness and irregular respiration. The patient may have systemic complications due to intracranial pathology. Pulmonary aspiration, hypoxia, hypertension, cardiac arrhythmias, fluid and electrolyte disturbances should be diagnosed and the patient should be optimized in the available time period. Most neurosurgical emergencies except acute trauma can wait for few hours and medically unstable conditions can be treated after consultation with neurosurgeons.

Medications history should be checked. The patient may be getting anticonvulsants, which can substantially alter anesthetic drug metabolism. During the examination, one should assess for intravascular volume status, as often these patients are on diuretics, especially mannitol, have inadequate intake or have recurrent vomiting. Treatment with corticosteroids should also be confirmed, and continued. These patients are prone for hyperglycemia and are at increased-risk of infection.

Laboratory Investigations

Specific attention should be provided to complete hemogram with platelet count, PT and INR, Blood sugar levels, (corticosteroid induced hyperglycemia) and serum electrolyte levels (diuretic and ADH effects). Electrocardiogram may show ischemic changes secondary to hypertension or increased ICP.

The CT scan or MRI should be assessed and the location and size of the mass, presence and extent of brain edema, midline shift and size of ventricles should be noted. The location of the lesion in the supratentorial or infratentorial compartment will affect the patient's clinical presentation and plan of surgery which in turn will determine the anesthetic management. Supratentorial space occupying mass often have intracranial hypertension, whereas infratentorial lesions create problems related to pressure effects on vital brainstem structures and secondary hydrocephalus.

The location specific and type specific consequences may be different with various lesions and they should be noted and the individual problems should be addressed, e.g. a mass in posterior fossa producing obstructive hydrocephalus with rapid clinical deterioration, loss of vision with tumors in third ventricular or occipital region, etc.

Further investigations may be needed for co-morbidities.

Premedication

Patients with increased intracranial pressure may have depressed level of consciousness and hence, all sedative or respiratory depressant drugs are best avoided for emergency decompressive surgery. Rarely, if the patient is alert and anxious, with clinically normal ICP, smaller doses of midazolam may be given to alleviate anxiety without affecting ventilation. Corticosteroids and anticonvulsants should be continued till surgery. Anticholinergic drugs or H_2-receptor antagonists drugs may be given.

Intraoperative Monitoring

Standard monitoring includes a 5-lead electrocardiogram, pulse oximetry, capnography, urinary output, core body temperature and neuromuscular blockade. Insertion of a

central venous catheter helps in judging intravascular volume status and can be used for the administration of mannitol and for injection of potent cardiovascular drugs in case of severe cardiovascular instability. When there is a risk of venous air embolism, the cannula should be placed in right atrium, for easy removal of air.

Many of the major neurosurgical procedures are associated with either large blood loss or need very strict control of blood pressure. It may be needed to manipulate blood pressure as per the steps of the surgical procedure. Therefore, most often continuous intra-arterial blood pressure monitoring is needed. In others frequent blood pressure monitoring using noninvasive blood pressure monitor is sufficient. When the patient's heart and brain are at different levels, and it is important to monitor cerebral perfusion pressure, with the help of the transducer placed at the level of external auditory meatus. The arterial line also helps in getting arterial blood samples for repeated blood gas analyses. Intraoperatively blood glucose should be monitored and both hypo- and hyperglycemia are to be avoided. Serum electrolytes, arterial blood gases, hematocrit estimation and monitoring of coagulation status are commonly required during all major neurosurgical procedures.

In patients who are at risk of venous air embolism, it is preferable to use sensitive diagnostic monitors like precordial Doppler or transesophageal echocardiography, however their availability and use in emergency situations are not common.

In elderly patients and in presence of clinically relevant cardiorespiratory comorbidities, pulmonary artery catheterization and cardiac output monitoring may be indicated.

Specific brain monitoring to ensure cerebral oxygenation may be used during the surgery as described earlier in the chapter. Bispectral Index Monitoring or entropy may not reliably detect focal ischemia but have been reported to be useful in detecting global ischemia, secondary to acute reduction in CPP.[40] Sometimes the surgery involves specific neural pathways and it may be necessary to check their integrity during the manipulation. This may require use of electrophysiological monitoring like somatosensory evoked potentials, visual evoked potentials or motor evoked potentials during the surgery. As anesthetic management affects these tests, their need during the procedure should be planned before the surgery so as to alter the anesthesia technique in a more favorable way.[18]

Choice of Anesthetic Agents

Under normal circumstances, the minor changes in CNS physiology produced by common anesthetic agents may not be significant but it is important to identify clinical situations in which these effects may cause significant impact on patient outcome. The characteristics of ideal agent for neuroanesthesia are described in Table 24.4. Most intravenous anesthetic, analgesic and sedative drugs except ketamine, reduce CBF and cerebral metabolic rate (CMR) and do not adversely affect ICP. On the other hand, all inhalational anesthetic agents produce dose-dependent cerebral vasodilation. Halothane causes most increase in cerebral blood flow and increase in ICP and hence is not suitable. Both sevoflurane and desflurane are preferred as they have minimum effects on cerebral physiology. Isoflurane is preferred when blood pressure lowering effect is needed without reducing CBF.

Rather than individual agent choice, it is more important how these agents are given and which other measures are taken to maintain CPP and control ICP and blood pressure. We routinely use intravenous agents for induction of anesthesia and maintenance is done with either propofol or inhalational agents along with other adjuncts.

Use of N_2O has been found to be associated with an increased-risk for the development of delayed ischemic neurological deficits in patients with likelihood of intraoperative cerebral ischemia.[41] When N_2O is not used, sufficient analgesia needs to be provided, preferably with opioids.

Succinylcholine produces small and transient increases in ICP. However, this can be prevented by pretreatment with nondepolarizing neuromuscular blocking agent. Agents causing histamine release should be used with caution. Rocuronium and vecuronium

Table 24.4: Characteristics of ideal agent for neuroanesthesia

Characteristics
Rapid onset and rapid offset
Maintains hemodynamic stability
Decreases CBV
No change in CSF dynamics
Decreases ICP
Maintains autoregulation
Maintains CO_2 reactivity
Easy titrability
Allows neuromonitoring
Does not increase $CMRO_2$
Anticonvulsant
Decrease edema
Offers brain protection
Provide analgesia

provide satisfactory neuromuscular blockade with hemodynamic stability.

Light sedation with remifentanil does not result in a higher ICP than propofol in patients undergoing stereotactic brain tumor biopsy. CPP might be better preserved with remifentanil.[42]

Propofol with fentanyl causes lower ICP and $AJvDO_2$ and higher CPP as compared to Isoflurane-fentanyl or sevoflurane-fentanyl. However, additional benefit is not seen if hyperventilation is instituted. The carbon dioxide reactivity in patients anesthetized with isoflurane and sevoflurane was found to be significantly higher than with propofol.[43]

Dexmedetomidine significantly attenuates the hemodynamic responses to intubation and the emergence from anesthesia. In addition, it increases intraoperative cardiovascular stability. Most of the effects were concentration-dependent, and the higher-dose was more effective than the lower dose.[44]

Intraoperative Management

Induction

Before induction, the gross mental status of the patient should be reassessed and documented. Goals of induction of anesthesia for emergency neurosurgical procedures are smooth induction, gentle intubation and avoidance of arterial hypertension.

- Usually, intravenous induction is preferred with agents that produce a rapid, reliable onset of unconsciousness without increasing ICP, e.g. thiopentone, propofol. In elderly or dehydrated patient sudden hypotension may occur, which can be reduced by giving smaller doses and in case of propofol, slower drug administration.
- Mild hyperventilation to decrease the $PaCO_2$ to near 35 mm Hg is appropriate. If N_2O is to be used, this should be instituted before starting the agent.
- Adequate depth of anesthesia should be ensured by additional doses of intravenous induction drugs before tracheal intubation.
- Esmolol, lignocaine or potent short-acting opioids are used to blunt the intubation responses.
- Skeletal muscle paralysis should be confirmed with the help of peripheral nerve stimulator to prevent coughing at tracheal tube insertion.
- One may aim for an initial reduction in baseline blood pressure (approximately 20%) during induction of anesthesia, blunt the hypertensive response to laryngoscopy and tracheal intubation by esmolol, labetalol, or lignocaine and then perform laryngoscopy and tracheal intubation.
- Alternatively, a deep level of anesthesia while balancing the associated decrease in blood pressure by a continuous infusion of a vasopressor (e.g. phenylephrine or norepinephrine) from the beginning of induction of anesthesia may be used. The deep level of anesthesia will prevent the hypertensive response to laryngoscopy and tracheal intubation, whereas the continuous infusion of a vasopressor will ensure adequate CPP.[18]
- As neck flexion and rotation are required commonly for neurosurgery, a reinforced, nonkinkable tracheal tube is preferred many times. Neck flexion can shift the tracheal tube endobronchially which should be prevented and watched for.
- A nasogastric tube is inserted in all intracranial cases to aspirate the stomach contents and a pharyngeal packing is appropriate when oral bleeding is expected.

Maintenance of Anesthesia

There is no evidence that any particular anesthetic combination is significantly different from another in terms of effects on ICP and short-term patient outcome. The commonly used techniques are:

- N_2O in oxygen with propofol-opioid neuromuscular blocking agent infusions
- Air-Oxygen with propofol-opioid neuromuscular blocking agent infusions
- Inhalational agent, N_2O in oxygen with opioid-neuromuscular blocking agent infusions
- Inhalational agent, Air-Oxygen with opioid- neuromuscular blocking agent infusions.

The use of N_2O is controversial in neuroanesthesia and many centers do not use it. Periods of greatest stimulation are during laryngoscopy, intubation, scalp pin insertion, skin incision, dural opening, periosteal manipulation and closure. At these times, anesthesia is needed to be deepened. Inhalational anesthetic agents at 0.6 to 1.0 MAC help in preventing or treating rise in systemic blood pressure related to noxious surgical stimulation. They increase the anesthetic depth and decrease the physiologic responses to noxious stimuli and preserve CBV and ICP. Isoflurane or sevoflurane 0.5 to 1 percent and fentanyl 2 µg/kg/hour are commonly employed combinations.

As the brain itself is insensitive to pain, deeper planes of anesthesia are not needed. Awareness needs to be prevented at all times. Adequate neuromuscular blockade should be ensured with at least a peripheral nerve stimulator.

Management principles of patients with increased ICP are summarized in Table 24.5.

Table 24.5: Management principles for patients with raised ICP

- Minimal or no premedication
- Continue anticonvulsants and steroids
- Normovolemia
- ICP monitoring
- Ventriculostomy/CSF drainage
- Mild Hypocarbia
- 30° head up position with neck neutral
- Maintain CPP > 70 mm Hg
- Mannitol, furosemide
- Barbiturates/low-dose inhalational agents.

Fluid Management

- Normovolemia, guided by central venous pressures, should be maintained.
- Isotonic solutions are used for correcting pre-existing deficits and as maintenance fluids (e.g. normal saline, lactated ringer solution). As large volumes of crystalloid alone increase cerebral edema, a combination of both colloids and crystalloids is commonly used. Among the various colloids available, newer generation lower molecular weight starches are preferred as they have least side effects of hypersensitivity reactions and coagulation impairments.
- Intraoperative blood loss may be replaced with packed red cells transfusion.
- Glucose containing fluids are avoided as hyperglycemia may increase ischemic brain injury.

Blood Pressure Management

Acute hypertension can elevate the intracranial pressure and therefore should be avoided or rapidly controlled. Use of additional small dose of propofol, fentanyl or agents like esmolol is helpful in controlling sudden increases. Vasodilators like nitroglycerine or sodium nitroprusside are rarely used now to control persistent hypertension, as they can increase cerebral vasodilatation and hence ICP. Isoflurane is preferred as it maintains the CPP. Hypotension should be avoided in most neurosurgical emergencies as it can reduce cerebral perfusion. In some vascular cases deliberate hypotension may be needed. They are discussed in another chapter.

Positioning

Proper positioning of a patient is an important aspect of neurosurgery. Both the anesthesiologist and the neurosurgeon must have full understanding of the implications and possible hazards of each position so as to avoid complications. The aims of positioning in neuroanesthesia are:

- Ease of access to deeper lesions
- Keeping the surgical site uppermost and easy venous drainage- to reduce bleeding and swelling.

Common positions in neurosurgical procedures are:

1. Supine, with its various modifications.
2. Prone position.
3. Lateral position.
4. Sitting position.

In anesthetised, vasodilated patient, any change in position involves sudden change in circulatory dynamics. This is further aggravated by controlled ventilation. The result is fall in cardiac output and blood pressure. This is more prominently seen in hypovolemic and elderly patients. Under anesthesia, intubation and paralysis of respiratory muscles affect normal respiratory dynamics unfavorably setting the stage for V/Q mismatch. In different positions, changes in pulmonary blood volume, lung volume and restricted movements of chest wall can aggravate the mismatch and over prolonged period of times may produce adverse effects. Pre-existing pulmonary pathology add to this insult. Increased ICP may result from increased abdominal pressure, venous congestion or when the head is placed below the level of the heart.[45]

Basic principles during positioning include:

1. Proper support to the upper chest and pelvis to prevent respiratory impairments which produce hypoventilation.
2. The position should be changed gradually to avoid sudden changes in blood pressure. At special risk are elderly and hypertensive patients. For patients who can tolerate infusion of crystalloids, preloading with intravenous fluids should be done to treat hypotension.
3. All pressure points should be padded and protected properly to avoid nerve injuries and pressure injuries.
4. Proper positioning of the neck to prevent venous obstruction is important.
5. Care of eyes to prevent pressure on eye ball or abrasion to cornea should be taken.
6. Elevation of the head and lower limbs may aid in venous drainage.

Prone Position: This position is used for midline lesions of the posterior fossa and all lesions of the spine and spinal cord that are approached posteriorly. In most cases head is fixed using skull pins or on a horse-shoe head rest in straight position with the tracheal tube hanging downwards. The anesthesiologist is often at the foot end of the patient.

- The light weight, nonkinkable or armoured tracheal tube should be used and must be firmly fixed. The breathing system should be well supported and should not drag down the tracheal tube. Use of antisialagogue agents, pharyngeal packing and careful presurgical scrubbing assist in keeping the tube fixing tapes dry. Conventional tracheal tube holders cannot be used in most cases, as they encroach on the surgical area.
- All the intravenous lines and breathing system should be long and kept away from surgical area.
- The abdomen should be free for diaphragmatic movements. The airway pressure should be continuously monitored.
- Eyes should be properly protected and no pressure should be placed on the eyeballs. The forehead and chin may be rested leaving the eyes free.
- Upper limbs should be placed in such a way as not to hinder the surgeon and avoid nerve injury. The access to intravenous site and at least one peripheral pulse is essential.

Lateral position: Modified lateral position is used for craniotomies involving temporal or posterior fossa regions:

- Stability of the patient in this position is the main concern.
- Care should be taken to avoid pressure on dependant limb affecting the circulation. Excessive downward pull on the shoulder should be avoided to prevent injury to brachial plexus.
- V/Q mismatch can occur in the lateral position and over a period atelectasis of the dependent lung may occur.

Sitting Position: This position is commonly used for surgeries involving infratentorial regions. This is the most controversial position in neurosurgery with several inherent problems:

- It is associated with severe reductions in cardiac output and hemodynamic instabilities, due to reduced venous return. Severe hypotension may occur while elevating the head end. Partly this is minimized by adequate hydration, use of elastic stockings and gradual positioning with simultaneous fluid loading.
- Extreme neck flexion and rotation, as often considered necessary by surgeons, can either kink the tracheal tube or may hamper jugular venous drainage. Use of nonkinkable armoured tube and limiting neck flexion should be helpful.
- As operative area is higher than the heart in this position, the pressures in the veins of the surgical field is usually negative. This may lead to entrapment of air and pulmonary venous embolism. To some extent this occurs in almost all patients. The amount of air trapped can be limited by increased intravascular volume to maintain higher CVP, use of PEEP, avoiding N_2O or flooding the surgical field with saline. The incidence of venous air embolism in sitting position is not influenced by N_2O use. Once a venous air embolism has been detected, N_2O use must be discontinued to prevent increase in the size of embolus.

Alternatively use of prone or lateral position for the surgery is commonly recommended to avoid the complication. If large amount of air blocks the pulmonary circulation, the patients can have hypoxia with sudden circulatory collapse necessitating resuscitation. Rapid change in position to supine may be needed in severe cases and the provision to do so should be planned while fixing the patient in sitting position. Many centers do not use this position and in case of emergent surgery it is not indicated.

General Care

Active methods to prevent deep vein thrombosis like use of unfractionated heparin are not used conventionally in neurosurgery for fear of increased risk of intracranial hematoma. Alternatively, use of graduated elastic stockings and active sequential compression- decompression devices are encouraged.

Temperature Management

As these procedures tend to be longer, heat loss can be significant, especially in children. This can be prevented by using warming mattress, warming blankets covering the patient, heating intravenous fluids and by use of heat and moisture conserving breathing systems. Hyperthermia is strictly prevented or corrected as it increases cerebral metabolism and oxygen demand. When it is necessary to reduce the cerebral metabolism, active or passive cooling methods are used.

Management of Tight Brain

- Check ventilation. Ensure tracheal tube patency and placement.
- Ensure adequate venous drainage. Head elevation.
- Moderate hypocarbia ($PaCO_2$ 25-30 mm Hg).[46]
- Ensure normal oxygenation.
- Control blood pressure. Target within 10 percent of basal pressure.[46]
- Ensure adequate anesthesia. If appropriate deepen it by additional boluses of barbiturates or propofol.
- Ensure adequate neuromuscular blockade.
- Administer mannitol.
- CSF drainage through lumbar drain, if already placed.

- Rule out pneumothorax (especially, if central lines are placed).

If the brain is still tight:

- Make sure that the inhalational agents are being given is less than 0.5 MAC.
- Discontinue N_2O.
- Discontinue inhalational agents and change over to propofol–opioid infusion.
- If brain swelling is still persistent, consider- presence of hematoma underlying, CSF drainage, postoperative barbiturate burst suppression.

Evoked Potential Monitoring

When intraoperative evoked potential monitoring is likely to be used, an anesthesia technique under which a consistent recordable waveform can be obtained, should be used. It should minimally affect the amplitude. For somatosensory evoked potential (SSEP) monitoring, use of inhalational agents either alone in doses higher than 1 to 1.3 MAC or, higher than 0.5 MAC along with N_2O is not suitable. Propofol infusion with opioids has lesser effect on SSEP amplitude. Commonly a low concentration of isoflurane (up to 1 MAC) without N_2O is appropriate for the monitoring. When cortical motor evoked responses are to be obtained during the procedure, use of neuromuscular blocking agent should be avoided, if the patient has preoperative muscular weakness. If there is no preoperative deficit, a partial blockade with 20 to 50 percent EMG response is acceptable. Hypothermia may affect nerve conduction and should not be employed.

Analgesia

Earlier it was thought that the craniotomy involves minimal pain, this has been refuted and the need for analgesia in neurosurgery is without a doubt. Various pain management regimens are used during craniotomy. Use of preincisional infiltration with local anesthetic agents is one of the commonest method of providing near complete analgesia for a limited time period. Scalp nerve blocks may be given before or after the surgery for postoperative analgesia. We have found this method to be convenient and superior to other conventional systemic methods. Opioids either alone or in combination with NSAIDs may be used in neurosurgical patients. Dexmedetomidine can act as adjuvant to analgesic regime while providing sedation.

Emergence

The effects of anesthetics and muscle relaxants are allowed to dissipate or pharmacologically reversed at the conclusion of intracranial surgery.

It is important to limit reaction to the tracheal tube as patient is waking up, as straining and coughing on the tracheal tube may cause increase in ICP, cerebral edema or intracranial hemorrhage. Therefore, continue anesthesia till skin suturing. The aim should be to wake up the patient at the end of head dressing and not at the last suture. After the dressing, discontinue all inhalational anesthetics and provide 100 percent oxygen. Reverse the residual neuromuscular blockade. If needed, one can administer intravenous lignocaine 1.5 mg/kg. Smooth extubation and rapid awakening are desirable. This facilitates monitoring the neurologic status and recognizing any adverse effects of the surgery. There can be increased sensitivity of injured neurons to the depressant effects of anesthetic agents. This may make preoperative deficit appear more severe. These deficits usually disappear and neurologic function returns to its baseline state with time.

Other causes of slow postoperative recovery may be hypothermia, residual neuromuscular block, residual effects of sedative drugs such as narcotics, benzodiazepines, volatile anesthetics or central nervous system event such as ischemia, hematoma, and tension pneumocephalus.

If consciousness is depressed preoperatively or new onset neurologic deficits are anticipated, it may be best to delay the tracheal extubation until return of airway reflexes is confirmed and spontaneous ventilation is sufficient to prevent CO_2 retention. Patients who remain intubated should be sedated, paralyzed and ventilated.

Postoperative Care

Postoperatively, all patients following emergency neurosurgery should be monitored in the neurosurgical ICU for monitoring of respiratory adequacy, hemodynamic stability and frequent assessment of neurological functions.

Posterior Fossa Space Occupying Lesions

Posterior fossa lesions constitute 60 percent of all tumours in children.[47] Many of these may have to be dealt with in emergency. Due to its unique location, the lesions in the posterior fossa pose several challenges for the management.

- Posterior fossa being a smaller compartment these lesions can cause obstruction to CSF circulation and produce obstructive hydrocephalus. The resultant increase in ICP can deteriorate the patient needing emergency decompression. As a temporizing measure a ventriculoperitoneal shunt may be planned in such situations, followed by the major surgery after proper patient preparation.

- The brain stem along with the vital centers lies in close proximity to these lesions. Hence, pressure on the brain stem may produce bradycardia, irregular respiration and lower cranial nerve palsies. Loss of protective airway reflexes makes these patients prone for pulmonary aspiration. Often they need ventilator support to treat hypoxia, while surgery is being arranged.
- Congenital anomalies like Arnold-Chiari malformation in which cerebellar tonsils herniate through foramen magnum, can cause lower cranial nerves dysfunction with stridor, respiratory distress and aspiration. It can further lead to quadriparesis and death in 30 percent of patients.[47]
- Bleeding inside a tumor can increase the size of lesion rapidly. Middle ear infections often get complicated and produce cerebellar abscesses. Emergency decompression may be needed.

The preoperative evaluation should include assessment of lower cranial nerves and also the pulmonary status. Sedative premedication should be avoided, as these patients are very sensitive to respiratory depressant effect. The decompressive surgery is commonly done in prone or lateral position. Rarely sitting position is used. In addition to routine monitoring, often arterial pressure monitoring is indicated for close monitoring of systemic blood pressure. As most of these patients are at risk of venous air embolism, a central venous cannula should be inserted in right atrium. The goals of anesthesia remain the same as described earlier, but here, rise in ICP is less tolerated. No specific anesthetic plan is considered better than others. Common intraoperative concerns are:

- Manipulations close to the brain stem can produce cardiac arrhythmias – especially bradycardia, which may get corrected after the stimulus is stopped. The anesthesiologist has to be vigilant throughout and should guide the surgeons during the excision of the lesion, by continuous information. In severe cases treatment with atropine or glycopyrrolate may be needed.
- Respiratory pattern is a sensitive indicator of these brain-stem manipulations. Hence, many anesthesiologists prefer to keep the patient spontaneously breathing during the surgery.
- Venous air embolism (VAE) is a frequent complication of posterior fossa surgery, especially in sitting position. Dural venous sinuses remain open during craniotomy and air can get sucked in the pulmonary circulation. The first evidence is usually seen as sudden reduction in end tidal CO_2 levels, as pulmonary gas exchange gets affected. In severe cases the patient may show hypoxia, hypotension or ischemic changes. In presence of patent foramen ovale, paradoxical air embolism may take place. More sensitive indicators for VAE are Doppler ultrasound and transesophageal echocardiography. If VAE is detected or suspected, inform the surgeons, who in turn, attempt to close open sinuses and flood the area with saline. Jugular compression can increase the venous pressure and stop further air entrapment. Attempts should be made to aspirate air via the central venous catheter. PEEP can be instituted if the patient is hemodynamically stable. N_2O should be stopped.
- Postoperatively airway edema may be extensive and it may be wise to keep the tracheal tube till edema subsides. Hypertension should be avoided in the emergence phase as risk of hematoma formation is high.
- Patients who were unconscious or had lower cranial nerves palsies should not be extubated at the end and ventilator support with gradual weaning is indicated. Some patients who remain in poor state and no intact airway reflexes, usually go for elective tracheostomy.

The Principles of Management in Neurosurgical ICU

1. Position: Neutral position with head elevation of 15 to 30°.
2. Monitoring of vital signs, GCS and pupillary changes.
3. Maintaining O_2/Ventilation: PaO_2 around 100 mm Hg.
4. Maintain $PaCO_2$ around 35 mm Hg.
5. Maintenance of MAP between 70 to 100 mm Hg.
6. Diuretics to reduce ICP should be continued (Mannitol 20% or Furosemide).
7. Maintain Normovolemia.
8. Use of steroids is controversial as no benefit in neurosurgery is found.
9. Prevent hyperglycemia: Start insulin.
10. Electrolytes: Regular monitoring of electrolytes, urea, creatinine, blood sugar, serum osmolality to determine fluid and electrolyte therapy.
11. Temperature control: Maintain normothermia. Prevent hyperthermia.
12. Nutrition: Early enteral feeding with high nutrient and proteins (to prevent infection).

SPECIFIC EMERGENCY CONDITIONS

Acute Hydrocephalus

Acute hydrocephalus may develop due to blockage of CSF circulation by a posterior fossa tumor, or blood clot

in ventricles or due to blockage of a previously functioning shunt. Rapidly rising intracranial pressure can deteriorate the patient condition fast and rapid decompression is required. The ventriculoperitoneal shunt is the most commonly performed procedure. The procedure is more commonly performed in children than in adults, in the supine position with head turned to one side:

- All patients will have some degree of increased ICP and hence, avoid further increases in ICP
- Avoid inhalational induction in a patient who is already stuporous
- Intravenous induction is preferred
- For children in whom cannulation of a peripheral vein cannot be accomplished readily, an inhalation induction with sevoflurane is a common approach, with initiation of controlled ventilation, by bag and mask, as rapidly as possible
- Mild hyperventilation ($PaCO_2$ 30 to 35 mm Hg) is customary
- Anesthesia is most commonly maintained thereafter with 60 percent to 70 percent N_2O, mechanical hyperventilation and isoflurane or sevoflurane as required
- As the surgery does not involve major blood loss, invasive monitoring is not required
- Blood pressure may decrease abruptly (as brainstem pressure is relieved) when the ventricle is first punctured
- Sometimes brief vasopressor support is required
- As the procedure involves large area of the body (head, neck, chest and abdomen), cold cleaning solutions can initiate hypothermia in infants. The fluids should be warmed and other measures to prevent heat loss should be taken
- Postoperatively, patients are kept supine after their procedures in an attempt to prevent an excessively rapid collapse of the ventricular system
- Administer 2 to 3 µg/kg of fentanyl as this procedure is not entirely pain-free, and a smoother emergence can be accomplished with a narcotic administration.

Other emergency surgical procedures for this condition may be ventricular tapping or external ventricular drainage. Anesthesia management should follow same principles, though often the procedure may be done under local infiltration anesthesia if the patient has preserved sensorium with patent airway.

Cerebral Abscesses

The incidence of brain abscesses is approximately 8 percent of intracranial masses in developing countries, whereas in the West the incidence is around 1 to 2 percent.[48,49,50] They may be present in any area of the brain, often occurring secondary to adjacent extracranial infection, previous trauma with open wound, meningitis or due to dissemination of distant systemic infections including tuberculosis. Commonest cause is extension from middle ear infection.

The presence of micro-organisms in brain tissue stimulates the immune system to mobilize large numbers of white blood cells (pus) that produce antimicrobial chemicals like cytokines, free radicals, etc. Some of these chemicals are also toxic to brain tissue. Hence, there is significant injury to brain tissue around an abscess. These also contribute to the formation of cerebral edema surrounding the abscess leading to an increase in intracranial pressure which can progress to cerebral herniation and death.

The patient may present initially with headache, fever or seizures. There may be sudden deterioration in consciousness. In suppurative otitis media with intracranial complications, commonly the neurosurgical procedure is performed first, followed by mastoidectomy at a later date. A combined procedure involving craniotomy and mastoidectomy in the same sitting has also been conducted and has been observed to be safe.[51]

Patients with congenital cyanotic heart disease are at risk for developing a brain abscess, with the incidence being higher in children.[48] Most of these patients present with headache without any other CNS symptoms, hence a CT scan should be performed with slightest suspicion. In patients with cyanotic heart disease, a right-to-left shunt allows the venous blood to leave the heart, bypassing the pulmonary circulation. Thus, bacteria in the bloodstream are not filtered through the pulmonary circulation, where they would normally be removed by phagocytosis. In addition, these patients can have low-perfusion areas in the brain due to chronic severe hypoxemia and metabolic acidosis.[48] Increased blood viscosity due to secondary polycythaemia may further reduce perfusion, making these patients prone to seeding by micro-organisms present in the bloodstream. They may have a variety of coagulation defects.

Anesthesia management to such patients can be challenging, as hypoxia or low arterial oxygen saturation along with high ICP can render the brain ischemic. Anesthetic drugs like ketamine, commonly preferred in cyanotic heart disease have tendency to further increase the ICP. Usually, an opioid based technique is used balancing the needs of both –the brain and the heart.

Immunosuppression can predispose patients to the development of brain abscesses. It can result from

systemic or hematological malignancy, infections like human immunodeficiency virus, or due to long-term steroid therapy, chemotherapy or immunosuppressive agents in patients who have undergone organ transplants. These patients are prone to the development of brain abscesses secondary to organisms that may not be seen in immunocompetent individuals, and because of this, empirical therapy in these patients should be avoided.[48] Attention should be directed to obtaining a microbiological diagnosis so that appropriate antibiotic therapy can be initiated without delay. During the surgical procedure extra care should be taken for asepsis, especially nosocomial respiratory infection should be avoided.

Stereotactically guided aspiration of cerebral abscess may be done for abscesses >3 cm in diameter. There may be high failure rate due to inadequate aspiration, especially in larger abscesses (Figs 24.7A and B). Immunosuppressed status and lack of higher antibiotics may contribute to recurrence of abscess.[52] Good outcomes in patients have been reported with long term external drainage following stereotactic aspiration.[53] This surgery may be performed with local anesthetic infiltration with monitered anesthesia care. The conventional excision surgery involving craniotomy will require general endotracheal anesthesia as described earlier. Preoperative seizures also make the patient prone for postoperative seizures and hence, anticonvulsants should be continued.

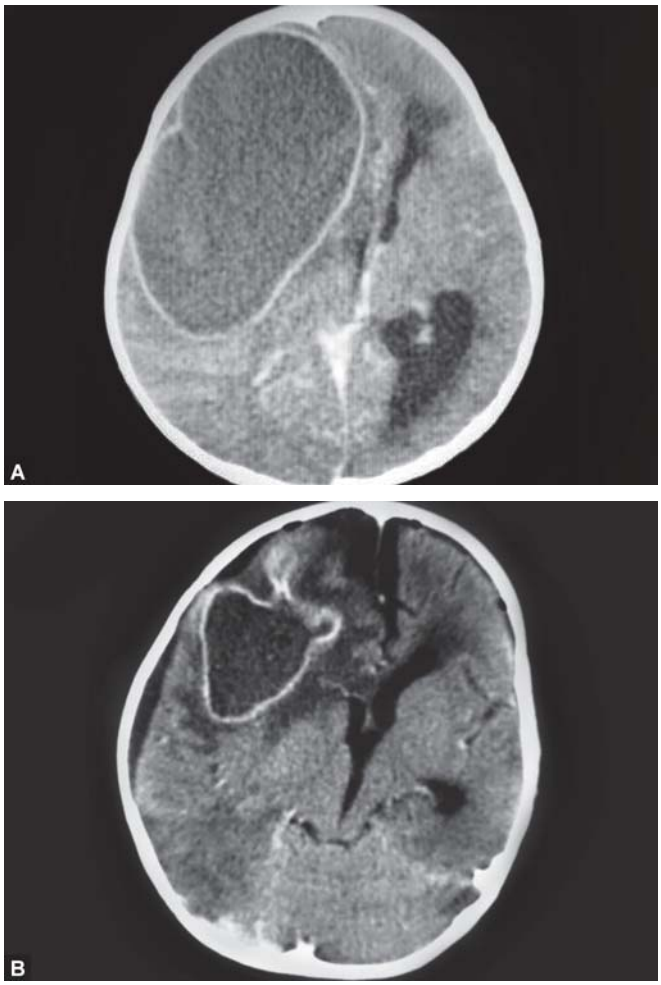
Subdural Empyema

Pus in the subdural space overlying the surface of the brain can cause a thrombophlebitis which can progress to cerebral venous thrombosis and possible hemorrhagic infarct. These may develop in chronic subdural hematoma with superadded infection.

Spinal Cord Decompressions

Sustained compression of the spinal cord can result in irreversible injury to this central nervous structure. Severe, sustained spinal cord compression can result in para- or quadriplegia with spasticity and loss of bladder and bowel control after only a few hours. Until the functional deficit is "complete" one must assume there is still function left to preserve with surgical intervention.

Hence, most emergency spinal surgeries are performed to relieve compression with discectomies, corpectomies or similar procedures and to provide spine stabilization with fusion or fixation to prevent further secondary cord damage. Most cervical spine procedures are done in supine position and thoracolumbar procedures in prone position. Position related care should be taken as discussed above.



Figs 24.7A and B: CT Scan showing intracranial abscess: (A) Before needle aspiration, (B) After needle aspiration

All pre-existing neurological deficit should be recorded. Respiratory reserve and cardiovascular instability should be assessed. Assessment must include careful examination of airway. In cervical spine compression patients, tracheal intubation technique should not pose a risk to the compromised spinal cord. If the risk to the spinal cord during intubation is high, often awake fiberoptic technique is used with minimal movement of cervical spine. At many places it is customary to wake up the patient after tracheal intubation and surgical positioning to assess the neurological deficit, before starting the procedure, if the patient is previously well preserved.

Standard monitoring may be used for most cases, but arterial and central venous pressure catheters are useful for more prolonged procedures. Additionally, spinal cord monitoring using evoked potentials may be required.

Anesthesia principles include avoidance of hypoxia and maintenance of spinal cord blood flow. Blood

pressure should be controlled balancing the spinal cord perfusion with the requirement to produce a bloodless surgical field. Many of these procedures can have major blood loss. Induced hypotension is not commonly employed as risk of reduced perfusion to spinal cord is major. Incidences of postoperative visual defects are rare but serious and occur due to ischemic optic neuropathy. The intraoperative hypotension is a contributing factor.

Extubation may be problematic and is best performed with the patient awake and able to support their own airway. Any new neurological deficits should be noted. Airway obstruction, respiratory inadequacy and bradycardia with or without hypotension are potential postoperative problems thus necessitating continuous monitoring of patients.

Postoperative pain is usually severe and adequate pain relief must be provided with local infiltration, opioids and NSAIDs combination. Use of epidural analgesia may not be appropriate for emergency decompressive surgeries with cord damage and pre-existing neurodeficits as postoperative assessment is affected.

REFERENCES

1. Patel PM, Drummond JC. Cerebral Physiology and the Effects of Anesthetic Drugs. In Miller RD, (Ed). Miller's Anaesthesia. (7th edn). Philadelphia: Churchill. Livingstone 2009;305-39.
2. Fitch W. Brain Metabolism. In Anesthesia and Neurosurgery. Cottrell JE, Smith DS (Eds). (3rd edn). Mosby; St. Louis 1994; 1-16.
3. Sulek CA. Intracranial Pressure. In: Cucchiara RF, Black S, Michenfelder JD, (Eds). Clinical Neuroanesthesia. (2nd Edn). New York; Churchill Livingstone. 1998;73-123.
4. Timofeev I. The Intracranial Compartment and Intracranial Pressure. In: Gupta AK, Gelb AW, (Eds). Essentials of Neuroanesthesia and Neurointensive Care. Philadelphia; Saunders Elsevier, 2008. pp. 26-31.
5. Pasternak JJ, McGregor DG, Schroeder DR, et al. Hyperglycemia in Patients Undergoing Cerebral Aneurysm Surgery: Its Association With Long-term Gross Neurologic and Neuropsychological Function. Mayo clinic proceedings. 2008;83:406-17.
6. Black S, Michenfelder JD. Cerebral Blood Flow and Metabolism. In: Cucchiara RF, Black S, Michenfelder JD, (Eds). Clinical Neuroanesthesia. (2nd Edn) New York; Churchill Livingstone 1998;1-40.
7. Bendo AA, Kass IS, Hartung J, Cottrell JE. Anesthesia for Neurosurgery. In: Barash PG, Cullen BF, Stoelting RK, (Eds). Clinical Anesthesia. (5th Edn). Philadelphia; Lippincott Williams and Wilkins 2006;747-90.
8. Bedforth NM, Girling KJ, Skinner HJ, Mahajan RP. Effects of desflurane on cerebral autoregulation. Br J Anaesth. 2001;87:193-97.
9. Mishra LD. Cerebral Blood Flow and Anesthesia – A Review. Indian J Anaesth, 2002; 46:87-95.
10. Simpson PJ. Neurosurgical Anesthesia. In: Aitkenhead AR, Rowbothom DJ, Smith G, (Eds). Textbook of Anesthesia. (4th edn). Edinburgh; Churchill Livingstone. 2001. pp. 688-98.
11. Gupta MM, Bithal PK, Dash HH, et al: Effects of stellate ganglion block on cerebral hemodynamics as assessed by transcranial Doppler ultrasonography. Br J Anaesth 2005; 95:669-73.
12. Schweitzer JS, Bergsneider M, Becker DP. Intracranial pressure Monitoring. In: Cottrell JE, Smith DS, (Eds). Anesthesia and Neurosurgery. (3rd Edn). St. Louis; Mosby 1994;117-35.
13. Shibutani T, Hirota Y, Niwa H, Matsuura H. Cerebral arterial blood flow velocity during induction of general anesthesia: Rapid intravenous induction versus awake intubation. Anesth Prog 1993;40(4):122–26.
14. Hansen TD, Warner DS, Todd MM and Vust LJ. Effects of Nitrous oxide and volatile anesthetics on cerebral blood flow. Br J Anaesth 1989;63:290-95.
15. Strebel S, Kaufman M, Anselmi L, Schaefer HG. Nitrous oxide is a potent cerebrovasodilator in humans when added to isoflurane. A transcranial Doppler study. Acta Anesthesiol Scand 1995;39:653-58.
16. Petersen KD, Landsfelt U, Cold GE, Petersen CB, Mau S, Hauerberg J, et al. Intracranial pressure and Cerebral Hemodynamic in Patients with Cerebral Tumors: A randomized Prospective Study of Patients Subjected to Craniotomy in Propofol-Fentanyl, Isoflurane-Fentanyl, or Sevoflurane-Fentanyl Anesthesia. Anesthesiology 2003;98:329-36.
17. Girling KJ, Cavill G, Mahajan RP. The effects of Nitrous oxide and oxygen on Transient Hyperemic Response in Human volunteers. Anesth Analg 1999; 89: 175-80.
18. Priebe H-J. Aneurysmal Subarachnoid Hemorrhage and the Anesthetist. Br J Anaesth 2007; 99:102-18.
19. Marval PD, Perrin ME, Hancock SM, Mahajan RP. The effects of Propofol or Sevoflurane on the Estimated Cerebral perfusion Pressure and Zero Flow Pressure. Anesth Analg 2005;100:835-40.
20. Patel PM. Cerebral Ischemia. In: Gupta AK, Gelb AW, (Eds). Essentials of Neuroanesthesia and Neurointensive Care. Philadelphia; Saunders Elsevier 2008;36-42.
21. Markus HS. Cerebral perfusion and stroke. J Neurol Neurosurg Psychiatry 2004;75:353-61.
22. Minghetti L, Levi G. Microglia as effector cells in brain damage and repair: Focus on prostanoids and Nitric oxide. Prog Neurobiol 1998; 54:99-125.
23. Friedlander RM. Apoptosis and caspases in neurodegenerative diseases. N Engl J Med 2003;348:1365-75.
24. Kupnik D, Grmec Š. Erythropoietin in post-resuscitation neurological recovery: Is there light at the end of the tunnel? Signa Vitae 2007;2:11-14.

25. Fukuda S, Warner DS. Cerebral Protection. Br J Anaesth 2007; 99(1):10-17.
26. Patel PM. Intraoperative Brain Protection. In: Gupta AK, Gelb AW, (Eds). Essentials of Neuroanesthesia and Neurointensive Care. Philadelphia; Saunders Elsevier 2008;43-8.
27. Nasu I, Yokoo N, Takaoka S, Takata K, Hoshikawa T, Okada M, Miura Y. The dose-dependent effects of isoflurane on outcome from severe forebrain ischemia in the rat. Anesth Analg 2006;103:413-8.
28. Elsersy H, Sheng H, Lynch JR, Moldovan M, Pearlstein RD, Warner DS. Effects of isoflurane versus fentanyl-nitrous oxide anesthesia on long-term outcome from severe forebrain ischemia in the rat. Anesthesiology 2004; 100:1160-6.
29. Sakai H, Sheng H, Yates RB, Ishida K, Pearlstein RD, Warner DS. Isoflurane provides long-term protection against focal cerebral ischemia in the rat. Anesthesiology 2007;106:92-99.
30. Pape M, Engelhard K, Eberspacher E, Hollweck R, Kellermann K, Zintner S, et al. The long-term effect of sevoflurane on neuronal cell damage and expression of apoptotic factors after cerebral ischemia and reperfusion in rats. Anesth Analg 2006;103:173-9.
31. Jevtovic-Todorovic V, Wozniak DF, Benshoff ND, Olney JW. A comparative evaluation of the neurotoxic properties of ketamine and nitrous oxide Brain Research 2001;859 (1-2):264-67.
32. Jevtovic-Todorovic V, Benshoff N, Olney JW. Ketamine potentiates cerebrocortical damage induced by the common anesthetic agent nitrous oxide in adult rats. Br J Pharm, 2000; 130: 1692-8.
33. Kassell NF, Haley EC Jr, Apperson-Hansen C, Alves WM. Randomized, double-blind, vehicle-controlled trial of tirilazad mesylate in patients with aneurysmal subarachnoid hemorrhage: A cooperative study in Europe, Australia, and New Zealand. J Neurosurg 1996;84:221-28.
34. Seppelt I. Intracranial hypertension after traumatic brain injury. Indian J Crit Care Med 2004; 8(2):120-26.
35. Todd MM, Warner DS, Maktabi MA, Vagnerova K. Neuroanesthesia. In: Longnecker DE, Brown DL, Newman MF, Zapol WM, (Eds). Anesthesiology. New York; McGraw-Hill. 2008;1081-1139.
36. Kiening KL, Hartl R, Unterberg AW, Schneider GH, Bardt T, Lanksh WR. Brain tissue PO_2 monitoring in comatose patients: Implications for therapy. Neurol Res 1997;19: 233-40.
37. Littlejohns LR, Bade MK, March K. Brain Tissue Monitoring in severe brain Injury I. Critical Care Nurse 2003; 23: 17-25.
38. Capan LM, Miller SM. Trauma and Burns. In: Barash PG, Cullen BF, Stoelting RK, (Eds). Clinical Anesthesia, (5th edn). Philadelphia: Lippincott Williams and Wilkins; 2006, 1262-97.
39. Tisdall MM, Smith M. Cerebral microdialysis: Research technique or clinical tool. Br J Anaesth 2006;97(1):18-25.
40. Prabhakar H, Ali Z, Rath GP, Singh D. Zero Bispectral Index During Coil Embolization of an Intracranial Aneurysm. Anesth Analg 2007;105:887-88.
41. Pasternak JJ, McGregor DG, Lanier WL, et al. Effect of nitrous oxide use on long-term neurologic and neuropsychological outcome in patients who received temporary proximal artery occlusion during cerebral aneurysm clipping surgery. Anesthesiology 2009;110: 563-73.
42. Girard F, Moumdjian R, Boudreault D, Chouinard P, Bouthilier A, Ruel M. The effect of sedation on intracranial pressure in patients with an intracranial space occupying lesion: Remifentanil versus propofol. Anesth Analg. 2009; 109:194-8.
43. Petersen KD, Landsfeldt U, Cold GE, Petersen C, Mau S, Hauerberg J, et al. Intracranial Pressure and Cerebral Hemodynamic in Patients with Cerebral Tumors: A Randomised Prospective Study of Patients Subjected to Craniotomy in Propofol-Fentanyl, Isoflurane-Fentanyl or Sevoflurane-Fentanyl Anesthesia. Anesthesiology 2003; 98:329-36.
44. Tanskanen PE, Kytta JV, Randell TT, Aantaa RE. Dexmedetomidine as an anesthetic adjuvant in patients undergoing intracranial tumour surgery: A double blind, randomized and placebo-controlled study. Br J Anaesth 2006; 97:658-65.
45. Du R, Lee CZ. Surgical positioning In: Gupta AK, Gelb AW, (Eds). Essentials of Neuroanesthesia and Neurointensive Care. Philadelphia; Saunders Elsevier 2008; 98-105.
46. Patel P. Neurosurgical anesthesia- Does the choice of anesthetic agents matter? Revista Mexicana de Anesthesiologia 2007; 30:S126-32.
47. Craen RA, Pellerin H. Anesthesia for posterior fossa Lesions. In: Gupta AK, Gelb AW, (Eds). Essentials of Neuroanesthesia and Neurointensive Care. Philadelphia; Saunders Elsevier 2008;36-42.
48. Moorthy RK, Rajshekhar V. Management of Brain Abscess: An Overview. Neurosurg Focus 2008;24(6):1-6.
49. Loftus CM, Osenbach RK, Biller J. Diagnosis and management of brain abscess. In: Wilkins RH, Rengachary SS (Eds). Neurosurgery, Volume 3, 2nd edn, New York: McGraw Hill 1996;3285-98.
50. Sharma BS, Gupta SK, Khosla VK. Current concepts in the management of pyogenic brain abscess. Neurol India 2000;48:105-11.
51. Kurien M, Job A, Mathew J, Chandy M. Otogenic intracranial abscess: Concurrent craniotomy and mastoidectomy—changing trends in a developing country. Arch Otolaryngol Head Neck Surg 1998;124:1353-56.
52. Kondziolka D, Duma CM, Lunsford LD. Factors that enhance the likelihood of successful stereotactic treatment of brain abscesses. Acta Neurochir (Wien) 1994;127:85-90.
53. Itakura T, Yokote H, Ozaki F, Itatani K, Hayashi S, Komai N: Stereotactic operation for brain abscess. Surg Neurol 1987;28:196-200.

25 Anesthesia for Cerebrovascular Emergencies

Hemangi S Karnik

KEY POINTS

- Cerebrovascular emergencies arise due to disorders like ischemic stroke, hemorrhagic stroke, rupture of intracranial aneurysms and arteriovenous malformations (AVMs)
- The most common sign of aneurysm rupture is subarachnoid hemorrhage (SAH)
- The major complications of SAH are rebleeding, cerebral vasospasm, hydrocephalus, cardiopulmonary dysfunction, and electrolyte disturbances
- Neurological deterioration, after a period of stabilization, in a patient with SAH usually occurs due to vasospasm.
- Therapy of cerebral vasospasm includes maintenance of Cerebral Perfusion Pressure (CPP), administration of nimodipine, Triple-H therapy, balloon angioplasty and intra-arterial papaverine
- Hyponatremia following SAH may be associated with hypo- or hypervolemia
- Acute hypertension should be prevented during the anesthetic management of aneurysm surgery to minimize the risk of aneurysm rerupture
- If temporary clips are used during aneurysm surgery, maintenance of CPP and reduction of cerebral oxygen consumption ($CMRO_2$) are needed
- In the event of aneurysm rupture, induced hypotension may be needed immediately and accurately
- AVMs may produce large shunts and induce intracerebral steal phenomena
- During surgical resection of AVM, there may be rapid and massive blood loss
- Removal of AVM can result in cerebral edema and possible hemorrhage
- Intracerebral hemorrhage is the most dramatic and likely to result in death than other emergencies
- Correction of coagulation defects is indicated before surgery for spontaneous intracranial hemorrhage
- Major ischemic stroke with severe edema may need emergency decompressive craniectomy
- Cerebral venous thrombosis may be associated with intracranial hemorrhage or infarct.

Cerebrovascular emergencies arise due to some of the most common and devastating disorders like ischemic stroke, hemorrhagic stroke, rupture of intracranial aneurysms and arteriovenous malformations (AVMs). They cause high number of deaths and are a major cause of disability. The incidence of cerebrovascular diseases increases with age and number of strokes are projected to increase.

An anesthesiologist often faces the challenge of caring and providing anesthesia for such patients in emergency or within a limited time frame.

INTRACRANIAL ANEURYSMS

Intracranial aneurysms are saccular dilatations that most commonly occur at bifurcation points[1,2] of the major intracranial cerebral vessels and occur in about 5 percent of people. Common contributing factors include arteriosclerosis, hypertension, and hereditary connective tissue disorders including Ehlers-Danlos syndrome, coarctation of aorta, polycystic kidney disease and fibromuscular dysplasia.[3] Most cerebral aneurysms lie outside the brain parenchyma close to circle of Willis, about 90 percent of them occur in anterior circulation[2] and when they rupture, they cause subarachnoid hemorrhage (SAH).[3,4] SAH occurs most commonly between the ages 40 to 60 years and slightly more common in women.

About a third of these patients die or become severely disabled after the first bleed and of the remaining patients, only one-third patients are 'functional' survivors.

Diagnosis

Unruptured aneurysm is usually asymptomatic and may remain undiagnosed. Rupture of aneurysm produces abrupt rise in intracranial pressure leading to the acute onset, severe headache. The classic presentation of aneurysmal SAH is that of severe headache associated with stiff neck, photophobia, nausea, vomiting and often transient loss of consciousness. On examination, systemic hypertension and dysrhythmias are common. In about one-third of patients, a small bleed or "warning leak" precedes a major aneurysmal rupture by approximately 2 to 3 weeks.[3] Warning symptoms and signs are milder and nonspecific like headache, dizziness, orbital pain, slight motor or sensory disturbance and are generally ignored or misdiagnosed by both patient and physician.

Presence of subarachnoid hemorrhage on CT scan is usually indicative of ruptured intracranial aneurysm. In addition, intracerebral or intraventricular hemorrhages may be present (Fig. 25.1). One of the following investigations is advised to confirm the diagnosis and to find out the location of aneurysm (Fig. 25.2).

- Four Vessel CT angiogram scan
- Magnetic resonance angiography
- Cerebral angiography.

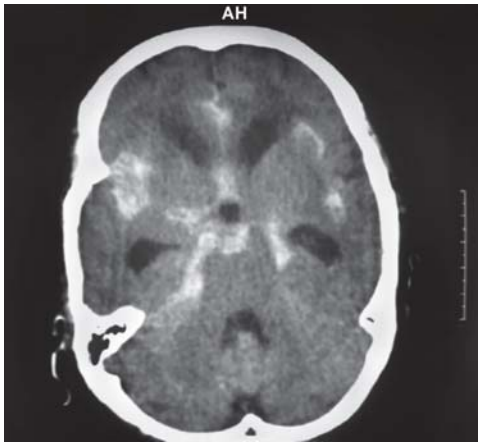


Fig. 25.1: CT scan showing subarachnoid hemorrhage

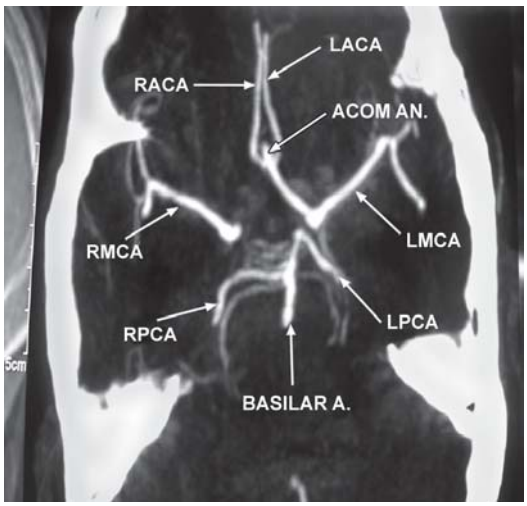


Fig. 25.2: CT angiography showing aneurysm of anterior communicating artery

Clinical Grading

The SAH has been graded in several ways. Hunt and Hess classification (Table 25.1) and World Federation of Neurosurgeons SAH scale are most commonly used grades (Table 25.2). These grades help in planning therapy and interventions and also act as predictors of outcome. Early interventions are advocated in grade I to III. grade 0, being asymptomatic, is usually accidentally discovered while being investigated for different reasons.

Complications of SAH

There are several potential complications of SAH[1] (Table 25.3).

Table 25.1: Modified hunt and hess scale for SAH

Grade	*Criteria**
Grade 0	Unruptured Aneurysm
Grade I	Asymptomatic or minimal headache with normal neurological examination
Grade II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
Grade III	Lethargy, confusion or mild focal deficit
Grade IV	Stupor, moderate to severe hemiparesis, possible early decerebrate rigidity, vegetative disturbances
Grade V	Deep coma, decerebrate rigidity, moribund appearance

*Serious systemic diseases (hypertension, coronary artery disease, chronic pulmonary disease, diabetes) and severe vasospasm on angiography cause assignments of the patient to the next less favorable category

Table 25.2: World federation of neurosurgeons (WFNS) SAH scale

WFNS grade	*GCS*	*Motor deficit*
I	15	Absent
II	13-14	Absent
III	13-14	Present
IV	7-12	Present or Absent
V	3-6	Present or Absent

GCS = Glasgow Coma Scale

Table 25.3: Potential complications of subarachnoid hemorrhage

- Vasospasm
- Rebleeding
- Intracranial hypertension
- Hydrocephalus
- Hyponatremia/volume contraction
- Seizures
- Electrocardiographic changes

Cerebral Vasospasm

It is a major cause of morbidity and mortality in SAH patients. It can be radiologically detected in up to 70 percent of patients and clinical vasospasm with ischemic deficits is observed in approximately 30 percent of patients, most often between 4 to 12 days.[1]

Neurological deterioration in a patient with SAH after a period of stabilization usually occurs due to vasospasm. The cerebral arterial vasospasm is caused by breakdown products of oxyhemoglobin and endothelin is considered to be a likely mediator.[3] The vasospasm after SAH directly correlates with amount of blood in the subarachnoid space and removal of the blood reduces the occurrence and severity of ischemic deficits.[5]

Vasospasm can be diagnosed by cerebral angiography or by Transcranial Doppler Ultrasonography (TCD). Flow velocity >120 cm/sec in the middle cerebral artery correlate well with angiographic narrowing of vessels.[3] TCD is also useful to judge the severity of vasospasm as well as the response to therapy.

Management

The aim of management in case of established vasospasm is to improve cerebral blood flow to the areas which have become ischemic by vasospasm.

"Triple–H" therapy: The standard treatment is "Triple-H" therapy, i.e. hypervolemia, hypertension, and hemodilution.[6] This therapy increases the cardiac output, improves cerebral perfusion pressure and reduces viscosity of the blood. For this purpose, systolic arterial pressure is increased by administration of crystalloids and colloids to approximately 120 to 150 mm Hg in non-operated and 160 to 200 mm Hg in operated aneurysm patients; central venous pressure is maintained at 8 to 12 mm Hg[1] and hematocrit is decreased to approximately 30 to 35 percent. If hematocrit is very low, packed red blood cells may also be used.[3] Triple-H therapy has been shown to reverse ischemic neurological deficits secondary to vasospasm in 70 percent of patients.[7,8] If needed, a vasopressor (phenylephrine or dopamine) is introduced to raise systemic blood pressure. Triple-H therapy may increase risk of rebleeding and may cause cerebral edema or increase in intracranial pressure. Systemic complications like pulmonary edema and cardiac failure may be seen in patients at risk. Hemodilution decreases blood viscosity and improves cerebral blood flow. The optimal hematocrit thought to maximize the oxygen delivery to tissues has been estimated to be 33 percent, but may be higher in ischemic brain.[5]

Calcium channel antagonists: After SAH oral nimodipine has been known to reduce morbidity due to cerebral ischemia. It is given at a dosage of 60 mg 4 hourly for 21 days following SAH. As there is no radiological effect on vasospasm, probably this drug acts directly on neurons rather than on vascular smooth muscles. Nimodipine therapy may interact with inhalational anesthetic agents, particularly isoflurane, to produce enhanced hypotensive effect.[9] Another intravenous alternative agent nicardipine has shown some benefit in symptomatic vasospasm without any difference in outcome. Currently it is approved only for control of blood pressure.[10]

Trials of magnesium sulfate have shown a trend towards improved neurological outcome in patients of SAH.[11] The trials involving endothelin antagonist clazosentan showed reductions in vasospasm, delayed ischemic deficits and improved outcome.[12] Vitamin E has also been used widely for neuroprotection. Recently Tirilazad, an antioxidant appears to be useful.[10]

Transluminal angioplasty: Symptomatic vasospasm can be alternatively treated by cerebral balloon angioplasty in refractory patients.[13] Transluminal angioplasty procedures are usually performed under general anesthesia to minimize movement and permit accurate placement of the intra-arterial balloon to dilate cerebral vessels. The risks of angioplasty include aneurysm rupture, intimal dissection, vessel rupture, ischemia and infarction.

Intra-arterial injections: Superselective intra-arterial infusion of papaverine (2 mg over 10 seconds, maximum 300 mg per hemisphere) has been used to dilate distal vessels not accessible to angioplasty. However, since intra-arterial papaverine is short acting and may be neurotoxic, many centers no longer use it.[13] Currently intra-arterial verapamil, a calcium channel blocker, and nicardipine, a dihydropyridine calcium channel blocker, are increasingly being tried.[13] Intrathecal and intraventricular infusion of sodium nitroprusside has also been reported to be effective.[14]

Rebleeding: The risk of rebleeding without surgery is about 30 to 50 percent in the first two weeks with a mortality of

more than 50 percent.[3] There is high-risk of rebleeding during cerebral angiography. The common signs are deterioration in consciousness, development of new focal deficits, hypertension, bradycardia, irregular respiration or electrocardiographic changes. To reduce the incidence, the patients with SAH should be placed in quiet surroundings and if needed, sedated. Other measures are analgesics, strict control of blood pressure and anticonvulsants.[8] Early surgery reduces the risk of rebleeding.

Administration of tranexamic acid up to 72 hours from time of rupture until the repair of aneurysm has decreased the rebleeding by 80 percent.[15] Use of antifibrinolytics may reduce risk of rebleeding but can increase the risk of pulmonary complication or deep venous thrombosis. They can also increase the risk of vasospasm.[10] Short-term epsilon aminocaproic acid (EACA) treatment has resulted in decreased rebleeding without any increase in serious side effects in select group of patients.[16] However, these may increase incidence of hydrocephalus.

Intracranial Hypertension

Intracranial Hypertension of varying degree is commonly present in patients with SAH in the first week. If it is severe or is complicated with intracerebral or intraventricular hemorrhage, vasospasm, cerebral edema or hydrocephalus, it needs to be treated. Along with blood pressure control, the patient often needs to be treated with ventriculostomy, steroids, diuretics, intubation and controlled ventilation.

Fluid and Electrolyte Imbalances

Fluid and electrolyte imbalances are frequently observed in patients following subarachnoid hemorrhage due to hypothalamic disturbances. SAH is frequently accompanied by hyponatremia, hypokalemia, hypocalcemia, and hypomagnesemia. Hyponatremia is most common, which may be associated with either syndrome of inappropriate antidiuretic hormone secretion (SIADH) or the cerebral salt wasting syndrome.[3] SIADH is characterized by dilutional hyponatremia and increased or normal intravascular volume and needs to be treated with fluid restriction. The cerebral salt wasting syndrome which occurs due to release of natriuretic peptide, is more common after SAH.[3,17] It consists of a triad of hyponatremia, volume contraction and high urinary sodium concentration (> 50 mmol/l). Fluid restriction in this disease will be deleterious to the patient. Isotonic fluids with NaCl should be administered till hypovolemia is corrected. In severe cases hypertonic saline may be used. Correction of hypontremia should be slow as rapid correction may lead to central pontine myelinosis.[3]

Hydrocephalus

Acute noncommunicating hydrocephalus occurs in 15 to 20 percent of patients commonly due to a blood clot in ventricles causing obstruction to CSF flow. This may lead to progressive unconsciousness and often requires prompt treatment with ventricular drainage.[8]

Seizures

Occur in 13 percent of patients and can increase chances of rebleeding.[5] Therefore, they should be immediately treated and prophylactic anticonvulsants are advocated.

Definitive Management Options

- Direct surgical clipping, ligation and/or wrapping
- Endovascular coil embolization.

Surgical Management

Current management of intracranial aneurysm involves surgical clipping within 48 hours with aggressive removal of blood, preferably within 12 to 24 hours of subarachnoid hemorrhage, usually applied in better neurological grades.[7,12]

Advantages of early surgery are:

- Less likelihood of rebleeding
- Reduced incidence of vasospasm, as it is directly proportional to presence of blood near basal cisterns
- The management of vasospasm becomes easy as hypertension and hypervolemia no longer pose threat for rebleeding
- Early surgery reduces incidence of medical complications of prolonged bed rest.

Early surgery may be technically difficult due to recent bleeding, brain edema and mild hydrocephalus. If, for any reason surgery can not be done in first 72 hours, usually it is deferred for about 2 weeks to eliminate the period of vasospasm and the fibrinolytic phase. Patients operated between days 7 and 10 have fared worst in major international studies.[18]

Preoperative Assessment and Preparation

Preoperative evaluation should include assessment of the duration and severity of SAH, neurological deficit, assessment of comorbidities, complications of the disease

and treatment received in intensive care unit (ICU). Adequacy of airway, breathing and circulation should be ensured.

Neurological Status

It is important for anesthesiologist to assess neurological status preoperatively, as they can suddenly deteriorate, hastening the urgency of surgery. The Patients with lower SAH grades may have deteriorating autoregulation and CO_2 response along with raised intracranial pressure (ICP). Patients with SAH grade I and II are likely to have normal ICP, intact autoregulation and normal response to carbon dioxide. Patients with altered level of consciousness may be candidates for postoperative ventilatory support.

Fluid and Electrolyte Status

Degree of hyponatremia and fluid status should be evaluated. The distinction between SIADH and cerebral salt wasting syndrome must be done as management and preoperative workup differs.

Vasospasm

One should look for evidence of vasospasm. Surgery is usually deferred in presence of vasospasm. However, if it is to proceed, note the therapy patient is receiving for vasospasm and maintain mean arterial pressure (MAP) to high normal range to maintain distal perfusion. Avoid hypovolemia, hypocarbia and induced hypotension, even in Hunt and Hess grade I patients. These patients should be given some form of 'Triple –H' therapy perioperatively.

Electrocardiogram (ECG)

Electrocardiographic abnormalities are common after subarachnoid hemorrhage.[19] Commonly described changes are—Cannon T-waves, nonspecific T-wave changes, Q-T prolongation, S-T segment depression, and presence of U-waves.[10] These may be due to severe hypertension and autonomic discharge resulting in sub-endocardial, myocardial injury. There may be elevation of troponin levels[1], increase in myocardial dysfunction and decreased cardiac index. There is an association between the extent of neurological injury and cardiac injury and the degree of neurological injury is more accurate predictor of myocardial morbidity than ECG changes.

These changes seem to recover rapidly and do not contribute to morbidity and mortality and usually do not influence anesthesia management. Therefore, surgery need not be deferred if there is no evidence of congestive cardiac failure or angina and enzymes are negative.[10] However, presence of ECG changes may indicate a risk of perioperative dysrhythmia. Particularly an increased Q-T interval has been associated with increased incidence of malignant ventricular rhythms including torsades de pointes.[7,20] This also predicts increased need of inotropes. The prognostic implications of recent myocardial infarction due to SAH are still unclear.

Pulmonary Complications

Following SAH, pulmonary complications commonly seen are—nosocomial pneumonia, congestive heart failure, aspiration pneumonia, acute respiratory distress syndrome and neurogenic pulmonary edema secondary to initial loss of consciousness, obtunded state[3] and sympathetic hyperactivity.[21] The chest condition should be optimized within the available time period. If respiratory failure is present, tracheal intubation, positive pressure ventilation with PEEP is advocated.

One should also look for evidence of hepatic dysfunction, renal dysfunction, thrombocytopenia or GI bleeding occurring after SAH.

Anesthesia Management

The anesthetic management for aneurysm clipping depends upon the timing of surgery, presence of hypertension, vasospasm and severity of the disease. Any technique that allows proper blood pressure control may be suitable. Main considerations while providing anesthesia for emergency aneurysm clipping are:

- Maintaining adequate cerebral perfusion and oxygenation
- Avoidance of acute rises in blood pressure
- Providing brain relaxation to facilitate dissection
- Preparedness to precisely manipulate blood pressure to facilitate clipping
- Preparedness to control bleeding in case of intra-operative rupture of aneurysm.

Premedication

Calcium channel blockers, anticonvulsants and steroids are to be continued. Drugs to reduce gastric acidity and to enhance gastric emptying should be given before induction. In patients with SAH grade I and II, as intracranial pressure is expected to be normal, anxiety related hypertension may be prevented by providing modest sedation. Patients with neurological deficits should not receive any sedative premedication outside

operation theatre. If needed, it should be given intravenously by the anesthesiologist.[10]

Monitoring

For emergency intracranial aneurysm surgery, monitoring should include 5-lead electrocardiography, invasive arterial pressure preferably secured prior to induction, pulse oximetry, capnography and core temperature. Central venous pressure monitoring and indwelling urinary catheter are helpful in maintaining normo- or hypervolemia. They are also useful when mannitol is needed to reduce brain bulk. Pulmonary artery catheter and transoesophageal echocardiography may be indicated in presence of pre-existing severe cardiac disease. ICP monitoring started prior to induction is useful in maintaining adequate cerebral perfusion though it is not widely used. Jugular venous oximetry ($SjvO_2$) reflects the global balance between cerebral oxygen demand and supply. Along with arterial blood gases, it is useful to assess adequacy of cerebral blood supply.

When available, somatosensory evoked potentials (SSEP) and brain stem auditory evoked potential (BAEP) monitoring may be used for posterior circulation aneurysms to assess neurological deficits. The anesthesiologist needs to know whether these monitoring will be used during surgery so as to use a suitable anesthesia technique.

Choice of Drugs

The choice of anesthetic agents should be guided by the effect of each agent on cardiovascular and cerebral dynamics. Both thiopentone sodium and propofol are satisfactory induction agents along with nondepolarizing neuromuscular blocking agent. Etomidate is useful for unstable patients. Even though laboratory studies suggest that N_2O aggravates brain injury after ischemia or hypoxia, the clinical effects are controversial and it is still being used in neurosurgery.

Both total intravenous anesthesia and inhalational agents provide satisfactory anesthesia. Sevoflurane preserves autoregulation and gives easy titrability during various phases of surgery. Fentanyl 25 to 50 μg boluses or 1 to 2 μg/kg/h infusion improves brain relaxation during craniotomy. Remifentanil provides intense analgesia and faster wear off.

Induction and Maintenance

One must weigh the risk of full stomach versus adequate depth of anesthesia and relaxation. Apart from the individual drug choice, it is important how the drugs are given using above mentioned principles. To avoid sudden hypertensive episodes, during laryngoscopy and intubation, usually patients are kept in deeper planes of anesthesia using additional doses of thiopentone or propofol, additional inhalational agent or large doses of fentanyl (upto 12 μg/kg). We also use lignocaine 1 to 1.5 mg/kg prior to laryngoscopy. Arterial pressure is continuously monitored and if blood pressure is rising, noxious stimulus is stopped and anesthesia is deepened. Shorter acting β-blockers like esmolol (0.5 mg/kg) are always kept ready and used if needed. In some patients, vasodilators may be needed for control of blood pressure. Usually during induction of anesthesia, N_2O is avoided in patients with decreased intracranial compliance and may be given after other measures like hypocapnia or cerebral vasoconstricting drugs are given. The scalp infiltration with local anesthetic agent at skull-pin sites, is used to attenuate hemodynamic response. For maintenance of anesthesia propofol, opioids and neuromuscular blocking agents are used along with any newer inhalational agents.

Control of Blood Pressure

SAH preserves CO_2 reactivity of brain but interferes with autoregulation.[22] Avoid more than 10 degree head tilt to prevent venous occlusion and subsequent increase in ICP. The optimum mean arterial pressure (MAP) to be maintained is best judged by $SjvO_2$. The transmural pressure across the aneurysm wall must be kept constant. Sudden increases in MAP as well as sudden decreases in ICP are to be avoided to prevent intraoperative aneurysmal rupture. It is not a routine practice to provide induced hypotension to all the patients. However, it may be necessary to reduce blood pressure at the time of clipping and one should be ready to provide that if required. In most cases MAP of 80 to 90 mm Hg are satisfactory during initial dissection and further lowered around 60 mm Hg at the time of direct manipulation of aneurysm. Deeper planes of inhalational anesthesia, nitroglycerin, sodium nitroprusside or nicardipine may be used to produce controlled hypotension.[7] We generally use isoflurane for lowering blood pressure, if needed. Induced hypotension is not provided in presence of vasospasm and in presence of associated renal insufficiency or cerebral vascular disease.

Fluid Administration

Hypovolemia should be corrected before induction using isotonic crystalloids and colloids to maintain cerebral perfusion. Normovolemia to mild hypervolemia

should be maintained. Use of colloids has not shown any particular benefit in aneurysm surgery.[3] Hyperglycemia should be avoided.

Brain Relaxation

Reduction in brain bulk during aneurysm dissection helps to provide good surgical exposure and reduced brain retraction. The brain relaxation may be provided by maintaining adequate depth of anesthesia, stable hemodynamics, careful surgical positioning, avoiding jugular obstruction, moderate hyperventilation ($PaCO_2$ 30 to 35 mm Hg), adequate analgesia and judicious use of mannitol and frusemide. Mannitol should be preferably given in such a way that its effect (15-30 minutes) will be seen after the duramater is open. If it is given early, sudden reduction in ICP may lead to rebleeding of aneurysm. Lumbar or ventricular cerebrospinal fluid (CSF) drainage may be useful to facilitate exposure when other measures fail. Though, this needs to be placed before surgery with caution.

Brain Protection

In case, temporary clipping of proximal vessel is needed, brain protection must be provided. This is done by maintaining cerebral perfusion, reducing brain metabolism and decreasing brain bulk so as to have less pressure by retractors. Various agents can be used to reduce cerebral metabolic rate for oxygen ($CMRO_2$) without causing sudden reductions in blood pressure. Barbiturates, usually thiopentone 3 to 5 mg/kg, may be used to produce burst suppression on EEG. Alternatively, isoflurane, propofol or etomidate may be used with lesser hemodynamic consequences. If available, Electroencephalogram should be monitored—preferably over the area most likely to be affected. If significant slowing occurs, usually the clip needs to be repositioned with elevation of MAP. If this is not possible, recirculation should start within 10 minutes of EEG changes to avoid postoperative neurological sequelae.[23] Mannitol is commonly used for brain protection. Combination of mannitol, vitamin E, phenytoin and dexamethazone has been found to offer cerebral protection during ischemia.[24] One should be aggressive in maintaining perfusion pressure, limiting pressure on brain tissue and minimizing duration of vessel occlusion.

Hypothermia

Though mild hypothermia (2-4°C) can provide significant cerebral protection in laboratory settings, clinically, in a multicentric trial (IHAST, the International Hypothermia in Aneurysm Surgery Trial) no neurological benefit attributable to hypothermia has been observed.[7,25] It is a general practice not to warm the patient actively during surgery.

Intraoperative Rupture of Aneurysm

Access to ipsilateral carotid artery should always be checked before anterior circulation aneurysm surgery.

Prevention: Some of the preferred measures to prevent intraoperative rerupture of aneurysm are:

- Control of systolic hypertension and to minimize transmural pressure. (MAP-ICP)
- Use of shorter acting antihypertensive agents, e.g. esmolol to prevent sudden surges in the blood pressure that may occur due to various interventions
- Maintenance of blood pressure to the lowest acceptable limit to maintain CPP
- Adequate pain relief and sedatives in titrated doses perioperatively
- Avoidance of sudden drop in ICP, e.g. rapid drainage of CSF as this can increase transmural pressure
- Monitoring CVP and maintaining euvolemia.
- Avoidance of seizures
- Normocapnia ($PaCO_2$ 35-40 mm Hg)
- Judicious use of mannitol and/or CSF drainage after craniotomy, if needed
- If temporary clip is applied, maintenance of high normal blood pressure to enhance distal perfusion and revert to low-normal blood pressure when clip is removed if aneurysm sac is still not ligated/clipped.
- Prevention of hypertension during emergence.

Management of intraoperative rupture: Following steps are to be followed:

- Always have blood ready in the operating room
- Start blood transfusion and call for help
- Help surgeons to get control over bleeding by lowering blood pressure, if needed
- Use hypotensive agents if needed. Massive bleeding will produce hypotension without any drugs
- Ipsilateral carotid compression can reduce bleeding
- Provide brain protection after the bleeding is controlled.

Intraoperative angiography may be needed and the anesthesiologist has to be careful about the radiation hazard, patient positioning, vascular access with its complications and the quantity of 'Heparin Flush' used.

Emergence and Postoperative Care

A patient may have another undetected aneurysm which can bleed at emergence, if sudden rise in blood pressure occurs. Both hypertension and tachycardia should be

avoided using adequate analgesia and if needed, using esmolol, labetalol or vasodilators like hydralazine. The use of low-dose fentanyl is found to have advantage over low dose anesthetics given during craniotomy closure.[26]

The patient should be awake and responding to commands at the earliest, so that postoperative neurological assessment can be done and early CT scan may be ordered, if needed. Failure of a patient to return to baseline neurological status can be due to residual effects of anesthetic agents, hypothermia or surgical concerns and these should be distinguished.[10] The presence of new focal neurological deficit may indicate improper clip placement, hematoma formation or development of vasospasm, all of which require prompt treatment.[3] Patients with higher grades of disease and intraoperative complications should be shifted to ICU and mechanically ventilated. Prophylactic hypervolemic fluid therapy has been used in patients who are at risk of developing vasospasm.

Special Situations

In aneurysms involving vertebro-basilar system, there is a risk of venous air embolism in lateral position. As brain stem is at risk, cardiovascular responses should be monitored. Spontaneous respiration strategies, though difficult, are preferred by many to identify brain stem ischemia. Auditory and somatosensory evoked responses have been employed to know impending neurological damage. Rarely, deep hypothermic cardiac arrest with cardiopulmonary bypass may be used for giant basilar aneurysms.[3]

ARTERIO-VENOUS MALFORMATION (AVM)

AV Malformations are congenital vascular abnormalities that involve shunting of blood between arteries and veins within the brain through low-pressure, high-flow shunts. They are present from birth and with time blood flow through the lesion increase enlarging the AV shunt. Cerebral AVMs may be associated with systemic conditions like Osler-Weber-Rendu disease, Sturge-Weber disease, and Wyburn-Mason syndrome.[27] These patients are prone to have intracerebral steal phenomenon leading to ischemia in surrounding brain tissue with neurological deficits. AVMs can also provoke epilepsy and local compression symptoms.

The symptoms at the time of presentation are varied and include hemorrhage, seizures and hydrocephalus or congestive cardiac failure.[28] It is an important cause of hemorrhage in young adults.[27] Untreated AVMs carry 2 to 4 percent annual risk of hemorrhage with morbidity between 38 to 53 percent and mortality between 10 to 18 percent.[4] These hemorrhages have little correlation with hypertension and show increased incidence during pregnancy.[29]

The treatment modalities for AVMs are surgical excision, conventional radiotherapy, stereotactic radiosurgery and endovascular embolisation.[3] There is significant operative morbidity and mortality, especially for larger and complex lesions.

Surgical Excision

The indications for emergency or urgent surgical excision of AV malformation are intracranial hemorrhage, intractable epilepsy and progressive neurologic deficits. Often adjunct endovascular superselective angiography and embolisation procedures of feeder vessels are used prior to surgical excision to reduce bleeding and facilitate the surgery.[30]

Anesthetic Considerations

During preoperative evaluation, pre-existing medical conditions and neurological status should be assessed. Special attention must be given to preoperative history of seizures, neurological deficits, symptoms of intracerebral steal and mass effect due to intracerebral hemorrhage. In patients with large doses of anticonvulsant and sedative therapy for intractable convulsions, possibility of excessive sedation and drug interactions with neuromuscular blocking agents should be kept in mind. If the patient has undergone embolisation before surgery, abnormalities and complications associated with the procedure should be looked for. Location specific possible intraoperative and postoperative complications should be discussed with surgeons.

General anesthesia techniques, usually employing similar principles as for aneurysm surgery, are used. Depending upon the location of AVM, if intraoperative neurophysiological testing like EEG or evoked potential monitoring is indicated, the drugs and doses should be appropriately chosen. In patients with large AVMs and resultant congestive cardiac failure, monitoring of pulmonary artery pressures may be appropriate[30]. Hypertension can increase ICP and edema and hypotension may produce ischemia. Hence, both should be avoided. For fluid replacement, role of crystalloids is limited and often colloids are preferred to maintain intravascular volume with reduced risk of developing cerebral edema.[29]

Rapid and massive intraoperative bleeding may occur which is often difficult to control and is a major cause of morbidity and mortality in AVM surgeries. Potentially life threatening hemorrhage can occur even after preoperative embolisation. Bleeding may also result in

malignant cerebral edema.[29] Rapid transfusion anticipation and preparation should be done in advance along with direct arterial pressure monitoring and adequate vascular access for rapid transfusion. Deliberate hypotension may be needed to reduce bleeding, though risk of ischemic changes is high. In addition, devascularization of surrounding brain tissue that can occur during surgery is better appreciated at normal perfusion.

During emergence hypertension is to be avoided. Patient should be awake and responding early for neurological assessment to diagnose and treat operative complications. Postoperatively hyperemic complications like perioperative edema or hemorrhage constitute major source of morbidity.

Normal Perfusion Pressure Breakthrough Syndrome

A unique phenomenon with AVM surgery is normal perfusion pressure breakthrough syndrome. The arteriovenous shunting effect of AVMs lowers the cerebral perfusion pressure in the areas of immediate vicinity. This leads to a dilated and fragile vasculature. Probably, there is loss of vasomotor tone and cerebral autoregulation.[3] Therefore, once the AVM is surgically removed, the CPP gets restored and may lead to significant postoperative edema, hyperemia and hemorrhage in this fragile area. The patients who are showing evidence of local cerebral ischemia prior to surgery are more likely to develop this syndrome postoperatively. This complication can be partly decreased by planning staged surgery, induced hypotension or barbiturate treatment.[29] In addition, osmotic diuresis, head-up position and hyperventilation have been attempted with questionable usefulness.

Interventional Neuroradiology

Interventional neuroradiology is emerging as a specialty itself due to advanced radioimaging modalities and used for a variety of neurovascular diseases. In emergency, it is being used in both, hemorrhagic stroke to embolise the bleeding vessel and in ischemic stroke to thrombolize the blocked vessel. It is frequently employed for endovascular coiling of cerebral aneurysms. Endovascular embolisation of AV malformation can completely obliterate and offer protection from hemorrhage with associated lower rates of morbidity and mortality. Alternatively, it may be used prior to elective surgery for AVM excision.

Anesthetic Considerations

Anesthetic considerations to provide anesthesia for patients undergoing emergency interventional neuroradiological procedures are maintenance of patient immobility, hemodynamic and respiratory stability, management of anticoagulation and treating sudden complications during the procedure. The management of critically ill patients during transport to and from radiology areas is equally important.

Thorough preoperative evaluation must be done to assess neurological status including GCS, neurological deficits, presence of raised ICP, hemodynamic stability and status of renal function. Any untoward incidents during previous angiography like allergies, excessive bleeding, adverse reaction to contrast medium should be noted. The procedure can be done either under general anesthesia or local anesthesia with sedation. The choice of anesthesia technique depends upon the need for intraoperative neurologic testing like speech or vision, need for controlled ventilation and ability of patient to lie supine for prolonged periods.[29]

Monitored Anesthesia Care

The procedure may be long and uncomfortable without general anesthesia but allows constant neurological evaluation and neurophysiological testing during an embolisation procedure. Midazolam, propofol, dexmedetomidine, droperidol and fentanyl have been used for sedation satisfactorily. Monitoring includes electrocardiogram, blood pressure and pulse oximetry. Oxygen should be administered to all patients via nasal prongs. Capnometry is optional if general anesthesia is not contemplated. Unprotected airway can pose a risk for aspiration, hypoxia and hypercarbia.

General Anesthesia

General anesthesia is commonly used as it allows patient immobility, better patient comfort, improved image quality and better control of physiological parameters. The anesthetic agents chosen for general anesthesia induction and maintenance should provide rapid recovery for neurological assessment. Generally, anesthetic agents with vasodilator properties can produce the steal phenomenon and the anesthetics producing vasoconstriction have the opposite effect, resulting in inverse steal, and may protect the "At risk" brain tissue.[30] Thiopentone is most commonly used for induction and fentanyl/isoflurane or propofol are commonly used for maintenance of anesthesia along with nondepolarising neuromuscular blocking agents using tracheal intubation and controlled ventilation technique. Laryngeal mask airway may be used as an alternative. N_2O is generally avoided as it can expand intravascular microbubbles.[31] Anticonvulsants should be provided to patients with history of seizures. In addition to usual monitors, arterial

pressure monitoring, either a separate line or using a side port of femoral cannula is done. It is customary to catheterise urinary bladder as the procedure often takes long time.

Normally, both hypertension and hypotension are avoided; however any of these may have to be provided as per need of the procedure. During embolisation with glue, hypotension is needed to slow the flow through the artery to reduce inadvertent systemic embolisation.[30] Hypocarbia is also used to reduce CBF at this point of time. As large amount of heparin is often needed, it is advisable to monitor Activated clotting time (ACT). During the procedure it may be necessary to maintain ACT 2 to 3 times the baseline value.

As the procedure is done in neuroradiology set-up, special precautions need to be taken to maintain patient position in such a way as to have rapid access to patient's airway. Low levels of lighting may hamper visualization of patient's colour and respiration. It is important to realise that digital subtraction angiography delivers considerably more radiation than fluoroscopy.[31] The radiation exposure drops off proportional to the square of the distance from the source. Therefore, activity near the head of the patient should be kept to a minimum during fluoroscopy, and the use of long extension tubings is required for infusion and monitoring lines. This also facilitates the patient table movement and easy access for drug administration. Hypothermia should be avoided. Precautions against prolonged radiation need to be taken for both—the patient and the anesthesiologist.

Postoperatively rapid recovery and continued hemodynamic monitoring are recommended.

Complications

Commonest complications are neurologic deficits occurring due to inadvertent blockage of a vessel supplying normal area. If the patient is under mild sedation, this can be noted by occurrence of speech disturbances, aphasia, hemianopia, hemiplegia, seizures or by neurological testing. Seizures during embolisation or coiling can be very harmful and disturbing and need to be treated with benzodiazepines or barbiturates. Pulmonary embolisation may occur especially with large AVMs. The contrast medium can produce adverse reactions as well as increased osmotic load.[29] Therefore, close monitoring for hypovolemia and electrolyte imbalance is required.

Acute bleeding may occur due to perforation of an artery. Systemic anticoagulation increases the amount of bleeding significantly. Small perforations may be treated conservatively. Hemorrhage will manifest as extravasation of contrast medium on fluoroscopy, with a sudden rise of systolic blood pressure. In some cases a proximal vessel may be embolised or the patient may have to undergo emergency craniotomy for aneurysm clipping. The outcome of a patient with severe hemorrhage is very poor. The anesthetic management of such patient depends upon the general and neurologic status. The anticoagulants like heparin which have been given during the procedure should be reversed immediately with protamine. Awake patients should be given antiemetics and analgesics to prevent vomiting and to treat the acute headache. Anticonvulsant therapy should be started as extravasation of contrast material can produce seizures. Rapid blood and blood components transfusions will be needed. The blood pressure is decreased as low as possible. Thiopentone boluses may be administered for cerebral protection. In addition, the brain edema is treated by the elevation of the head, the infusion of mannitol and moderate hyperventilation.[32]

If vasospasm occurs, it can be immediately treated with intra-arterial papaverine through the catheter and blood pressure is raised by 15 to 20 percent from baseline value.

SPONTANEOUS INTRACEREBRAL HEMORRHAGE (SICH)

Spontaneous intracerebral hemorrhage (SICH) is collection of blood that occurs in the brain parenchyma in the absence of trauma or surgery. This entity accounts for 20 to 30 percent of all strokes in Asian populations[33] and is associated with a higher mortality rate than either ischemic stroke or subarachnoid hemorrhage. Common causes include hypertension, amyloid angiopathy, coagulopathy, vascular anomalies, tumors and various drugs. It may also be present along with SAH in case of rupture of aneurysm or AV malformation. Hypertension, however, remains the single greatest modifiable risk factor for SICH and therefore is often termed as "Hypertensive Intracerebral Bleed".

Intracerebral hemorrhage (ICH) is the most dramatic, among cerebrovascular emergencies. Though the improved treatment of arterial hypertension has decreased its frequency, it still carries a 30 to 55 percent mortality rate.[34] Intracerebral hemorrhage is more than twice as common as subarachnoid hemorrhage (SAH) and is much more likely to result in death or major disability than cerebral infarction or SAH.[35] Though the benefit of surgical clot evacuation remains uncertain,[4] many of these patients need emergency surgery for decompression in cases of large hematoma or when the cause of the bleeding can be surgically corrected.

Presentation[35]

- Sudden onset of a focal neurological deficit that progresses over minutes to hours

- Accompanying headache
- Decreased consciousness
- Nausea, vomiting
- Elevated blood pressure.

The initial diagnostic modality of choice is CT scan (Fig. 25.3). Angiography should also be considered.

The management of these patients has two aims:
A. Management of acute hemorrhage.
B. Prevention of rebleeding.

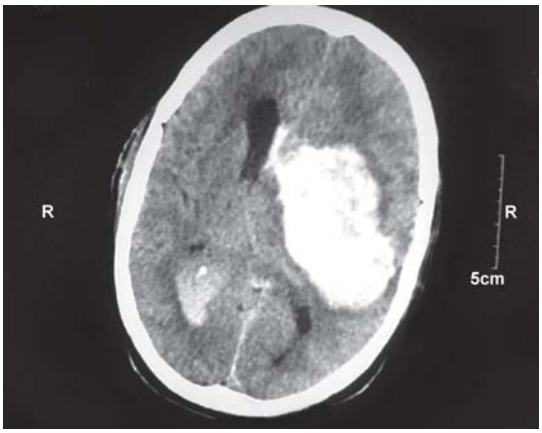


Fig. 25.3: Intracerebral hemorrhage with midline shift

Management

Treatment predominantly includes supportive measures and control of general medical risk factors. As patients of SICH are often unconscious, initial management should first be directed towards the basic airway, breathing, circulation and detection of focal neurological deficits. The patient with Glasgow Coma Scale (GCS) < 9, respiratory insufficiency (PaO_2 < 60 mm Hg on FiO_2 0.4, $PaCO_2$ >55 mm Hg) and those who are at obvious risk of aspiration with or without hypoxemia should be intubated and ventilated with supplemental oxygen.

Control of blood pressure: Hypertension should be treated only if mean arterial pressure is >130 mm Hg or systolic BP is >185 mm Hg.[35] Table 25.4 shows several commonly used agents for acute control of blood pressure. Nicardipine 2.5 mg/h IV may be given initially; dose is increased by 2.5 mg/h every 5 min to a maximum of 15 mg/h as needed to decrease systolic BP by 10 to 15 percent from baseline.

Control of ICP: ICP monitoring should be done for patients with GCS < 9 and should be maintained below 20 mm Hg.[35]

Table 25.4: Agents used for intraoperative control of blood pressure

Drug	*Dosage*	*Advantages*	*Disadvantages*
Sodium nitroprusside	0.5-10 μg/kg/min	Rapid onset Good titrability	Cyanide toxicity ↑ ICP Rebound hypertension Coagulation abnormalities ↑ Pulmonary shunting
Nitroglycerin	1-10 μg/kg/min	Rapid onset Good titrability	↑ ICP Rebound hypertension Coagulation abnormalities ↑ Pulmonary shunting
Trimethaphan	1–5 mg/min	Rapid onset	Histamine release Cerebral compromise below autoregulation range □ Pseudocholinesterase
Esmolol	0.2-0.5 mg/kg loading dose 50-200 μg/kg/min	Rapid, short effect	Limited efficacy Myocardial depression Bronchospasm
Labetalol	After test dose 0.5-2 mg/min (total 300 mg)	Reduced side effects	Limited efficacy Bronchospasm
Nicardipine	5 mg/h infusion, up to 15 mg/h	Rapid onset Less tachycardia	Slow offset ↑ Pulmonary shunting
Isoflurane/ sevoflurane/desflurane	Titrated dose	Provides surgical Anesthesia Reduce $CMRO_2$	□ ICP □ Cerebral edema ↓ Vital organ blood flow

Fluid Balance: Normovolemia should be maintained. Generally, CVP should be maintained between 5 and 12 mm Hg or pulmonary wedge pressure at 10 to 14 mm Hg.

Fever should be aggressively treated, if present. The role of anticonvulsant is controversial but many centers prescribe those for short-terms. Anticoagulants and antithrombotic drugs are contraindicated.

Concomitant Oral Anticoagulant/Antithrombotic Therapy (OAT)

Oral anticoagulant therapies increase the incidence of spontaneous intracerebral hemorrhage by 6 to 7 times, worsen the severity by hematoma enlargement and may increase the mortality rate to as high as 67 percent.[34,36]

Warfarin is most commonly associated oral anticoagulant with intracranial hemorrhage.[37] Although international normalized ratio (INR) values exceeding 3.5 to 4 are associated with a higher-risk of ICH, majority of OAT-related ICH occurs when the INR is within the therapeutic range.[38]

The primary aim of management is reversal of the anticoagulant effect to limit ongoing bleeding and hematoma expansion. The traditional methods involving Vitamin K (10 mg IV daily for 3 days) and Fresh Frozen Plasma (FFP) (15-30 ml/kg) are slower and less effective. Agents such as prothrombin complex conjugates (PCC) (15 ml/kg) and recombinant activated factor VII (rFVIIa) are being increasingly used to achieve more rapid correction of INR in warfarin-associated coagulopathy.[37] Although, rFVIIa can reverse the elevated INR measurements rapidly, its use in this setting remains investigational.[39]

Patients receiving aspirin or clopidogrel are also at higher-risk of spontaneous hemorrhage as well as hematoma expansion that has occurred due to any cause. Patients with dual therapies are at even higher-risk.[38] Most of these patients are also at high-risk of myocardial infarction as well as of thromboembolism. Platelet transfusion may be useful in such cases. It is uncertain whether procoagulant therapy given in ICH patients put them at more risk of thrombotic complications.[40] There is no role of these agents in limiting the size of hematoma in patients without coagulopathy.

In patients with OAT associated ICH, the coagulation status should be monitored with INR and brought down to or below 1.5 prior to surgery when needed.

Surgical Management

Evacuation of clot within four hours of bleeding is complicated with increased incidence of rebleeding.[41,42] Patients with small hematomas (<10 cm^3) or minimal neurological deficits should be treated conservatively. Surgery should be considered in patients with moderate to large lobar or basal ganglia hemorrhages and those suffering progressive neurological deterioration. The surgery may prevent death but neurological outcome may or may not improve. Rabinstein et al studied the outcome of clot evacuation in patients with worsening intracranial hemorrhage.[43] They found that about a quarter of patients showed functional independence but those with absent upper brain stem reflexes died.

Most posterior fossa clots occur in cerebellum and exert effects by compressing the adjacent vital areas rather than direct destruction of surrounding tissue. They also have marked tendency to cause hydrocephalus.[4] Cerebellar hematomas that are >3 cm in diameter or large cerebral hematomas may need early evacuation as life-saving procedure but risk of rebleeding is high, sometimes even increasing neurologic deficits. Elderly patients with the Glasgow Coma Scale score <5, brainstem hemorrhages, or small hemorrhages do not typically benefit from surgery. Early evacuation of deep cerebral hematomas is associated with high surgical mortality and severe neurologic deficits.[44]

The brain damage due to ICH may be minimized by removal of the hematoma. It reduces the mass effect, blocks the release of toxic products and prevents early hematoma enlargement occurring after onset of ICH.[45] In a review it was concluded that in the modern era of CT, good neuroanesthesia, intensive care, and the operating microscope, surgery is beginning to find a therapeutic role in the treatment of supratentorial intracerebral hemorrhage.[46]

The goals of surgical treatment of ICH should be to remove as much blood clot as possible with the least amount of brain trauma. If possible, surgery should also remove the underlying cause of ICH, such as an arteriovenous malformation, and prevent complications of ICH such as hydrocephalus and mass effect of the blood clot.[35]

Conventionally open craniotomy and removal of hematoma is done. New strategies focusing on early hemostasis, improved critical care management, and less invasive surgical techniques for clot evacuation are likely to decrease secondary neurologic injury.[47] These include stereotactic aspiration, special ultrasonic aspiration, aspiration with double cannulae under CT monitoring sometimes with local fibrinolytic instillation to dissolve the clot. Intraventricular injection of urokinase has been found to be safe and beneficial for thrombolysis after spontaneous, nonaneurysmal intraventricular hemorrhage.[48]

Anesthetic Considerations

Control of hypertension and correction of coagulation defects form the mainstay of anesthetic management for emergency surgery to evacuate primary intracranial hematoma. Stereotactic clot evacuation may be done using local anesthesia and sedation if patient is stable. In most emergency craniotomies general anesthesia with basic neurosurgical principles should be employed.

Preoperative assessment includes patient's general status, neurological condition and associated systemic diseases. Chronic hypertension is often associated with ischemic heart disease and diabetes mellitus, therefore cardiac function should be evaluated. Acute hypertension can be controlled preoperatively and the management can be continued intraoperatively. Systolic blood pressure should be brought down to less than 180 mm Hg as described earlier. In addition, drugs like clonidine, dexmedetomidine and propofol may be used perioperatively as adjuncts for control of hypertension.

Detailed history should be obtained about current medications. Patients on anticoagulants or antiplatelet agents should be investigated for coagulation status. Any coagulation defects will need to be corrected prior to surgery using blood products like fresh frozen plasma or platelets if needed.

Use of fibrinolytics like tranexamic acid is useful during surgery and should be continued postoperatively for at least 24 hours to reduce intraoperative nonsurgical bleeding and risk of postoperative hematoma.

Adequate analgesia extending into postoperative period and continued control of blood pressure during emergence are important aspects in preventing rebleeding or hematoma formation. Patients who had decreased levels of consciousness prior to surgery and those who need controlled ventilation should be monitored in neurosurgical ICU.

ACUTE ISCHEMIC STROKE

Stroke is a neurological emergency characterized by a rapidly evolving episode of focal or global loss of cerebral function with symptoms lasting for more than 24 hours or leading to death, with no apparent cause other than vascular in origin. Around 80 percent of strokes are caused by acute ischemic stroke and 20 percent are caused by intracerebral hemorrhage.[49]

The common risk factors are older age, prior stroke, hypertension, atrial fibrillation, established cardiac disease, carotid stenosis, hyperlipidemia, cigarette smoking, alcohol intake and diabetes. It may be triggered by inflammatory stimuli through immunologically mediated platelet activation and endothelial dysfunction. Preventive measures include treatment with antihypertensive agents, statins, aspirin, cessation of smoking and carotid endarterectomy.

When the brain becomes ischemic, the electrical activity disappears in 10 to 20 seconds; sodium-potassium pump fails within 30 seconds; glucose level rapidly decreases with intracellular water and sodium influx, causing cytotoxic edema in 3 minutes. In 5 to 10 minutes, intracellular lactate levels increase five fold and cellular glucose is exhausted. To this point, the changes are reversible. Prolonged ischemia causes progressive and irreversible cellular death and edema worsens. Within few hours, due to disruption of Blood Brain Barrier, vasogenic edema ensues with accumulation of fluid in extracellular compartment. The combined edema is maximum at 24 to 48 hours after an ischemic event. Mass effect due to edema can often lead to increased intracranial pressure, midline shift and uncal or cingulate herniation. Reduction in cerebral perfusion pressure may further enhance ischemic injury.

The patient typically presents with sudden onset neurological deficit along with cognitive dysfunction, secondary to disruption of blood supply. If the blood flow is quickly restored, symptoms may subside completely with full recovery. This is labeled as transient ischemic attack (TIA). Generalized low blood flow may produce syncopal attack and if continues for longer duration, can progress to ischemic stroke.

Management of Acute Stroke

The goals of therapy are providing neuroprotection to reduce ischemic damage in penumbra zone and to restart perfusion in the ischemic area.

All patients should be monitored by cardioscope for ischemic changes or arrhythmia. Oxygen should be supplemented, if SpO_2 is less than 93 percent. Dehydration should be corrected. Hemodilution may be useful to improve blood flow through narrowed vessels. Hyperglycemia is associated with local lactic acidosis and jeopardizes the recovery of the ischemic brain tissue in penumbra zone and therefore, should be avoided. Calcium channel blockers like nimodipine reduce neuronal damage that accompanies ischemia.

Anticoagulation appears to be the mainstay of reperfusion management in acute stroke. Aspirin, low molecular weight heparin and intravenous thrombolytic therapy using urokinase, streptokinase or tissue plasminogen activator (rTPA) have all been found useful. Intravenous rTPA (0.9 mg/kg, maximum 90 mg) with

10 percent of the dose given as a bolus followed by an infusion lasting 60 minutes is the most beneficial proven treatment, if given within 3 hours of onset of ischemic stroke.[50] Hemorrhagic complications are observed in 20 percent of patients following thrombolysis. This can be reduced with blood pressure control with SBP limits <195 mm Hg and DBP < 110 mm Hg.

Hypothermia has been used to reduce ischemic damage. Currently, it can not be recommended for ischemic stroke. Fever, which is observed in 25 percent of patients, should be aggressively treated. In addition, glutamate antagonists, sodium channel antagonists, glycine antagonists, free radical scavengers, membrane stabilizers like citicholine, etc. have been tried for minimizing the damage.

Surgical Interventions

Surgery has limited role in acute ischemic stroke. Immediate decompressive surgery may be life-saving for patients with severe edema with impending cerebral herniation or in patients with "Malignant" cerebral infarction. Endovascular radiological procedures may be planned for angioplasty, thrombectomy or intra-arterial instillation of vasodilators.

Cerebral Venous Thrombosis

Cerebral venous thrombosis results from occlusion of a venous sinus and/or cortical vein by a partial thrombus or by extrinsic compression that subsequently progresses to complete occlusion. Once the vein is occluded, there is obstruction of cortical venous drainage and further pressure causes breakdown of the brain-blood barrier with vasogenic edema and hemorrhage (Fig. 25.4). Finally, venous infarct with cytotoxic edema ensues. This may lead to raised ICP or ischemic stroke which does not follow any arterial distribution. They are more common in patients with hypercoagulable state or infections. Most frequent causes are oral contraceptives and pregnancy/ puerperium. Hence, 75 percent of patients are females.[51] The presentation varies from mild headache to focal deficits or even coma and seizures.

Management

Supportive measures include intravenous fluids, anticonvulsants and control of ICP. Patients of central venous or sinus thrombosis without concomitant hemorrhage is not a contraindication for heparin therapy. They may be treated with either body-weight adjusted subcutaneous low molecular weight heparin or intravenous heparin.[52] Other specific measures include endovascular thrombolysis. In severe cases decompressive craniectomy may be indicated.

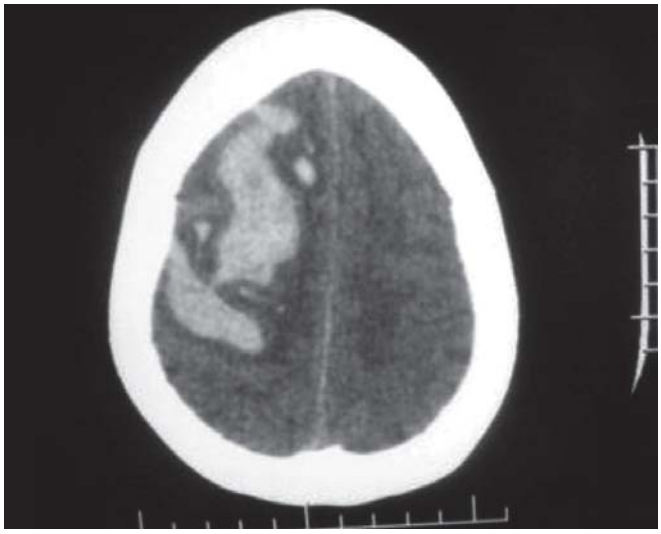


Fig. 25.4: CT scan showing cerebral venous infarct

Endovascular thrombolysis is done in patients who are deteriorating in spite of systemic anticoagulation. This is done via transvenous femoral route under general anesthesia with full systemic anticoagulation, using pharmacological means or balloon dilator or microsnare.[53] There is a risk of major hematoma or venous sinus rupture. The success rate is also low.

Decompressive Craniectomy

In some patients of ischemic stroke with severe cerebral edema, ischemia and increased ICP, decompressive craniectomy appears to be a life-saving procedure.[54] Parenchymal hypodensity in greater than 50 percent of MCA territory on CT scan (also known as 'Malignant CVA') predict poor collateral circulation and in such patients decompressive surgery may be useful. Intracranial pressure more than 30 mm Hg is usually considered as an indicator for surgical intervention. If craniectomy is performed early, especially in young patients, a satisfactory functional outcome can be achieved in a significant proportion of cases.

Patients should be medically treated with steroids, mannitol and hyperventilation before consideration is given to a decompressive craniectomy. Those who show transient clinical improvement following these measures are likely to benefit from surgery. Monitoring brain tissue oxygenation ($PbtiO_2$) could be an important tool for deciding craniectomy in the future.[55]

In decompressive craniectomy, a large bone flap along with duroplasty is essential for adequate ICP reduction. As these patients are having impending cerebral herniation, any further rise in ICP should be prevented during anesthesia. Usually, most of these patients are already intubated and mechanically ventilated. Use of mannitol

and acute hyperventilation ($PaCO_2$ 25-30 mm Hg) for short period can buy crucial time for decompression. Other anesthetic principles remain same as described in previous chapter for decompressive surgeries.

REFERENCES

1. Priebe H-J. Aneurysmal Subarachnoid Hemorrhage and the Anesthetist. Br J Anaesth 2007; 99:102–18.
2. Crespo JL. Anesthesia for the surgery of Intracranial Aneurysms: Part I. The Internet Journal of Anesthesiology. 1998; vol.2. Number 1.
3. Black S, Sulek CA and Day AL. Cerebral Aneurysm and Arteriovenous Malformation. In: Cucchiara RF, Black S. Michenfelder JD (Eds) Clinical Neuroanesthesia. 2nd edn. New York: Churchill Livingstone; 1998;265-318.
4. Mitchell P, Gregson BA, Vindalacheruvu RR, Mendelow AD. Spontaneous Intracerebral Hemorrhage. In: Loftus CM (Ed.). Neurosurgical Emergencies. 2nd edn. New York: Thieme; 2008;68-77.
5. Bendo AA, Kass IS, Hartung J and Cottrell JE. Anesthesia for Neurosurgery. In: Barash PG, Cullen BF, Stoelting RK, (Eds). Clinical Anesthesia 5th edn. Philadelphia: Lippincott Williams and Wilkins; 2006;746-89.
6. Origitano TC, Wascher TM, Reichman HO, Anderson DE. Sustained Increased Cerebral Blood Flow with Prophylactic Hypertensive Hypervolemic Hemodilution (Triple-H, Therapy) after Subarachnoid Hemorrhage. Neurosurgery 1990;27:729-40.
7. Drummond JC. Anesthesia for intracranial aneurysm surgery. In: ASA annual meeting refresher course Lectures 2004;219:1-7.
8. Kotapkar MJ, Flamms ES. Cerebral Aneurysms- Surgical Considerations. In: Cottrell JE, Smith DS (Eds). Anesthesia and Neurosurgery, 3rd edn. St. Louis: Mosby; 1994;364-75.
9. Simpson PJ. Neurosurgical Anesthesia. In: Aitkenhead AR, Rowbothom DJ, Smith G (Eds). Textbook of Anesthesia. 4th edn. Edinburgh: Churchill Livingstone 2001;688-98.
10. Todd MM, David SW, Maktabi MA, Vagnerova K. Neuroanesthesia. In: Longnecker DE, Brown DL, Newman MF and Zapol WM (Eds). Anesthesiology. New York: McGraw Hill 2008;1081-139.
11. Van den Bergh W, Dijkhuizen RM. Rinkel GJE. Potentials of magnesium treatment in subarachnoid hemorrhage. Magnesium Research 2004;17:301-13.
12. Drummond JC. Patel PM. Neurosurgical Anesthesia. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL (Eds). Miller's Anesthesia. (7th edn). Philadelphia: Churchill Livingstone 2009;2045-87.
13. Mindea SA, Yang BP, Bendok BR, Miller JW, Batjer HH. Endovascular Treatment Strategies for Cerebral Vasospasm. Neurosurg Focus 2006;21(3).
14. Thomas JE, Rosenwasser RS, Armonda RA, Harrop J, Mitchell W and Galaria I. Safety of intrathecal sodium nitroprusside for the treatment and prevention of refractory vasospasm and ischemia in humans. Stroke 1999;30:1409-16.
15. Menon GR, Nair S, Rao RM, Abraham M, Easwer HV, Krishnakumar K. Patterns and predictors of in-hospital aneurismal rebleed: An institutional experience and review of literature. Annals of Indian Academy of Neurology 2007;10:247-51.
16. Starke RM, Kim GH, Fernandez A, Komotar RJ, Hickman ZL, Otten ML et al. Impact of a Protocol for Acute Antifibrinolytic Therapy on Aneurysm Rebleeding After Subarachnoid Hemorrhage. Srtoke 2008;39:2617-21.
17. Sivakumar V, Rajshekhar V, Chandy MJ. Management of neurosurgical patients with hyponatremia and natreuresis. Neurosurgery 1994;34:269-74.
18. Kassel NF, Torner JC, Haley EC, Jane JA, Adams HP, Kongable GL. The international Co-operative study on the timing of aneurysm surgery. Part I: Overall management results. J Neurosurg 1990;73:18-36.
19. Davis TP, Alexander J, Lesch M. Electrocardiographic changes associated with acute cerebrovascular disease: A clinical review. Prog Cardiovasc Dis 1993;36:245-60.
20. Takenaka I, Aoyama K, Iwagaki T, Ishimura H, Kadoya T. Development of Torsade de pointes caused by exacerbation of QT prolongation during clipping of cerebral artery aneurysm in a patient with subarachnoid hemorrhage. Br J Anaesth 2006;97:533-5.
21. Friedman JA, Pichhelman MA, Piepgras DG, McIver JI, Toussaint LG, McClelland RL, et al. Pulmonary complications after aneurysmal subarachnoid hemorrhage. Neurosurgery 2003;52:1025-32.
22. Newfield P and Bendo AA. Anesthetic Management of Intracranial Aneurysms. In: Newfield P, Cottrell JE (Eds). Handbook of Neuroanesthesia. 4th edn. Philadelphia: Lippincott Williams and Wilkins 2006;143-72.
23. Mizoi K, Yoshimoto T. Permissible temporary occlusion time in aneurysm surgery as evaluated by evoked potential monitoring. Neurosurgery 1993;33:434-40.
24. Suzuki J, Abiko H, Mizoi K, Oba M and Yoshimoto T. Protective effect of phenytoin and its enhanced action by combined administration with mannitol and vitamin E in cerebral ischemia. Acta Neurochirurgica 1987;88:56-64.
25. Todd MM, Hindman BJ, Clarke WR, Torner JC. Mild Intraoperative Hypothermia during surgery for Intracranial Aneurysm. N Engl J Med 2005;352:135-45.
26. Bhagat H, Dash HH, Bithal PK, Chouhan RS, Pandia MP. Planning for Early Emergence in Neurosurgical patients: A Randomised Prospective Trial of Low dose Anesthetics. Anesth Analg 2008;107:1348-55.
27. Achrol AS, Guzman R, Varga M, Adler JR, Steinberg GK, Chang SD. Pathogenesis and Radiobiology of Brain Arteriovenous Malformations: Implications for Risk Stratification in Natural History and Post-treatment Course. Neurosurg Focus 2009;26(5):E9.
28. Millar C, Bissonnette B, Humphreys RP. Cerebral arteriovenous malformations in children. Can J Anaesth 1994;41:321-31.

29. Dodson BA. Interventional Neuroradiology and the anesthetic management of patients with Arteriovenous Malformations. In: Cottrell JE, Smith DS (Eds). Anesthesia and Neurosurgery 3rd edn. St. Louis: Mosby 1994;407-24.
30. Sinha PK, Neema PK, Rathod RC. Anesthesia and intracranial arteriovenous malformation. Neurology India 2004;52:163-70.
31. Varma MK, Price K, Jayakrishnan V, Manickam B, Kassell G. Anesthetic considerations for interventional neuroradiology. British Journal of Anesthesia 2007;99: 75-85.
32. Asouhidou, Asteri T, Katsaridis V, Georgiadis G. Immediate Anesthesia Management of Complications During Embolization of Cerebral Vascular Lesions. The Internet Journal of Anesthesiology 2007 Volume 14 Number 2.
33. Cheung RT. Update on medical and surgical management of intracerebral Hemorrhage. Rev Recent Clin Trials 2007; 3:174-81.
34. Steiner T, Rosand J, Diringer M. Intracerebral Hemorrhage Associated With Oral Anticoagulant Therapy-Current Practices and Unresolved Questions. Stroke 2006;37: 256-62.
35. Broderick JP, Adams HP, Jr, Barsan W, Feinberg W, Feldman E, Grotta J, et al. Guidelines for the management of spontaneous intracerebral Hemorrhage. AHA Scientific statement. Guidelines for the management of spontaneous intracerebral Hemorrhage. A statement for healthcare professionals from a special writing group of the stroke council, American Heart Association. Stroke 1999;30: 905-15.
36. Toyoda K, Yasaka M, Nagata K, Nagao T, Gotoh J, Sakamoto T, et al. Antithrombotic therapy influences location, enlargement and mortality from intracerebral hemorrhage. The bleeding with Antithrombotic Therapy (BAT) Retrospective study. Cerebrovasc Dis 2009;27: 151-9.
37. Aguilar MI, Hart RG, Kase CS, Freeman WD, Hoeben MBJ, Garcia RC, et al. Treatment of Warfarin-Associated Intracerbral Hemorrhage: Literature Review and Expert Opinion. Mayo Clinic Proceedings 2007;82:82-92.
38. Marietta M, Pedrazzi P, Girardis M, Torelli G. Intracerebral hemorrhage: An often neglected medical emergency. Intern Emerg Med 2007;2:38-45.
39. Broderick J, Connolly S, Feldmann E, Hanley D, Kase C, Kreiger D, et al. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage in Adults: 2007 Update: A Guideline From the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke 2007;38:2001-23.
40. Iorio A. Iatrogenic causes of an ICH: OAT therapy. Eur J Anesthesiol Suppl 2008;42:8-11.
41. Morgenstern NB, Demchuk AM, Kim DH, Frankowski RF, Grotta JC. Rebleeding leads to poor outcome in ultra-early craniotomy for intracerbral hemorrhage. Neurology 2001; 56:1294-9.
42. Mayer SA. Ultra-early hemostatic therapy for intracerebral hemorrhage. Stroke 2003;34:224-9.
43. Rabinstein AA, Atkinson JL, Wijdicks EFM. Emergency craniotomy in patients worsening due to expanded cerebral hematoma: To what purpose? Neurology 2002; 58:1367-72.
44. Fewel ME, Thompson BG Jr, Hoff JT. Spontaneous Intracranial Hemorrhage: A Review. Neurosurg Focus 2003;15(4): Article 1, 1-16.
45. Minematsu K. Controversies in stroke. Evacuation of Intracerebral hem atoma is likely to be beneficial. Stroke 2003;34:1567.
46. Fernandes HM, Gregson B, Siddique S, Mendelow AD. Surgery in Intracerebral Hemorrhage. The uncertainty continues. Stroke 2000;31:2511-6.
47. Manno EM, Atkinson JL, Fulgham JR, Wijdicks EF. Emerging medical and surgical management strategies in the evaluation and treatment of intracerebral hemorrhage. Mayo Clin Proc 2005;80:420-33.
48. Coplin WM, Vinas FC, Agris JM, Buciuc R, Michael DB, Diaz FG, Muizelaar JP. A Cohort Study of the safety and Feasibility of Intraventricular Urokinase for Non-aneurysmal Spontaneous Intraventricular Hemorrhage. Stroke 1998;29:1573-9.
49. Special report from the National Institute of Neurological Disorders and Stroke. Classification of cerebrovascular diseases III. Stroke 1990;21:637–76.
50. Adams HP, Jr., del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, et al. Guidelines for the Early Management of Adults With Ischemic Stroke. A Guideline From the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke 2007;38:1655-711.
51. Agostoni E, Aliprandi A, Longoni M. Cerebral Venous Thrombosis. Expert Rev Neurother 2009;9:553-64.
52. Masuhr F. Einhaupl K. Treatment of cerebral venous and sinus thrombosis. Front Neurol Neurosci 2008;23:132-43.
53. Renowden S. Interventional Neuroradiology. J Neurol Neurosurg Psychiatry 2005;76:iii48-iii63.
54. Lanzino DJ, Lanzino G. Decompressive Craniectomy for Space-Occupying Supratentorial Infarction: Rationale, Indications and Outcome. Neurosurg Focus 2000;8(5). Article 3.
55. Reithmeier T, Lohr M, Pakos P, Ketter G, Ernestus RI. Relevance of ICP and $ptiO_2$ for indication and timing of decompressive craniectomy in patients with malignant brain edema. Acta Neurochir (Wien). 2005;147(9):947-51.

Section VIII

Trauma

26 Anesthesia for Head Injuries

Hemangi S Karnik, Jyoti S Magar, Devangi A Parikh

KEY POINTS

- Primary brain injury occurs at the time of actual trauma. Secondary brain injury involves additional damage that occurs to the brain after the traumatic event.
- In infants, large scalp wounds and major hematoma (subgaleal blood collection) can result in anemia and hypotension.
- Rapid and directed neurological examination should be performed as soon as cardiopulmonary status is stabilized, for all trauma patients on arrival.
- If the patient's systolic BP cannot be brought above 100 mm Hg despite aggressive fluid resuscitation, the priority should be given to establish the cause of hypotension and neurosurgical diagnosis takes a second priority.
- Never presume that the altered mental status is due to alcohol or other intoxicants.
- After traumatic seizures, postictal state will also worsen the patient's responsiveness for significant duration.
- Best motor response elicited is more accurate prognostic indicator than worst response.
- Emergency head CT scan must be obtained as soon as possible after hemodynamic stability is ensured even in multisystem injury patient.
- About 3 percent of minor head injury (HI) and 20 percent of moderate HI have unexpected deterioration resulting in severe neurological dysfunction.
- Early tracheal intubation should be performed in comatose patients with GCS 8 or lower.
- Current management principles of management of head injured are based on both—reduction of ICP and improving cerebral perfusion. Normovolemia is maintained.
- Maintain normocarbia (30-35 mm Hg) in a patient with head injury. Routine use of PEEP is not advocated.
- The main goals of anesthesia are to maintain the cerebral perfusion to optimum, minimize increasing in ICP, to prevent secondary brain damage and to provide optimum surgical conditions.
- Opioids in clinical doses, produce minimal decrease in CBF and $CMRO_2$ and cause minimum effect on ICP, if ventilation is maintained.
- Use of N_2O is contraindicated in presence of pneumoencephalus.
- The patients who were comatose with unfavorable GCS score prior to surgery and those who had intraoperative high ICP, should remain intubated for neurosurgical purpose in postoperative period.
- Treatment with fresh frozen plasma (FFP), Vitamin K and activated recombinant factor VII may be needed as immediate therapeutic measures to reverse drug-induced coagulopathies.
- Non-neurosurgical procedures in a head injury patient are highly controversial as these are associated with increased incidences of hypoxia and hypotension.

Trauma is the leading killer of young, productive people between 1 to 45 years of age.[1] Few patients offer greater challenge to the anesthesiologist than the critical trauma patient. The suddenness of the situation and uncertainty about the extent of injury necessitates that the anesthesiologist is knowledgeable, keenly observant, vigilant and methodical in approaching such patients. Advances in anesthesia techniques, improvements in monitoring technology, development of intraoperative monitoring standards and awareness of patient safety have substantially reduced the perioperative mortality and morbidity.[2]

Trauma Patient

Trauma patient management requires a multidisciplinary approach. Initial resuscitation plays an important role in successful outcome. As in our hospital, in many institutions, an anesthesiologist is an integral part of the trauma team and is available to care for a multiple injury patient.[3] The initial management is based on the Advances Trauma Life support protocols.

The ATLS scheme for trauma management is as follows:[4]

1. *Primary survey:* Involves rapid evaluation of patients to identify problems in vital functions which are crucial to survival and pose immediate threat to life, such as airway obstruction, major chest trauma hampering the breathing, cardiovascular collapse or coma.
2. *Resuscitation of vital functions:* Includes management of the problems found in primary survey in rapid and efficient manner. It includes airway management, breathing support, hemorrhage control, intravenous access and volume resuscitation.
3. *Secondary survey:* Reassessment of patients from head to toe along with the history taking should be done again as soon as resuscitation is started to identify all detected and possible injuries. Some of these can be potentially life-threatening such as major bleeding by hemoperitoneum or pelvic disruption, spine or intracranial injuries, multiple fractures, etc.
4. *Definitive care:* Steps 1 and 2 are performed simultaneously. The processes of immediate care are enumerated in Table 26.1.

HEAD INJURY

Traumatic brain injuries (TBI) are among the commonest cause of death and disabilities.[5,6] The overall mortality in severe TBI is 23 percent.[7] Even those who survive of traumatic brain injuries are often left with major neurological impairment resulting in long-term disability. Therefore, even a small reduction in mortality and morbidity after brain injury should have a major impact on public health.

In the recent years, the treatment of CNS trauma has improved significantly due to:

a. Development and improvement of emergency medical systems, techniques of rapid diagnosis enabling early operative decompression.
b. Better understanding of pathophysiology of brain injury leading to development of measures to prevent secondary brain damage.

Development of evidence-based protocol has helped to significantly reduce the mortality.[8] The key to management is to identify these patients as emergencies. This involves early recognition of patients with potential intracranial injuries, early diagnosis with CT scan and immediate definitive management including surgery.

Table 26.1: ATLS protocol of primary survey

Assess	*Manage*
Airway:	
Response to verbal commands	Chin lift, jaw thrust, airway insertion, tracheal intubation, O_2 supplementation, tracheostomy
Breathing:	Intercostal drainage, positive pressure ventilation
Clinical assessment	
Pulse oximetry	
Arterial blood gases	
Chest X-ray	
Circulation:	Control of external hemorrhage, Intravenous access,
Heart rate, blood pressure,	Administration of fluids, pelvic binders, fracture splinting,
ECG, Capillary refill, skin turgor,	Emergency surgery, blood transfusion
Pelvic X-ray, FAST, blood grouping and X-matching	
Disability:	Oxygenation, controlled ventilation, adequate perfusion, surgical
GCS, pupils, motor, sensory examination, spine X-rays,	decompression, ICP monitoring
CT scan	
Exposure:	Expose completely, review test results and definitive care
Detailed examination, laboratory studies	

An anesthesiologist is often involved in the care of head injured patient in various ways:

- As a first responder at site
- As a member of trauma ambulance team
- As a resuscitator in emergency department
- As an intensivist in trauma ICU
- As an anesthetist in OR
- As pain management specialist.

Therefore, an anesthesiologist should have knowledge of relevant anatomy and cerebral physiology for rational management of head injured patients in various capacities.

Pathophysiology

Traumatic brain injury usually has two components:

1. *Primary brain injury* that occurs at the time of actual trauma. The brain tissue is disrupted by mechanical force-either by direct contact or by sudden acceleration, deceleration forces. These injuries can not be minimized except by preventive strategies. Primary injuries can be skull fractures, focal neuronal injuries or diffuse injuries. Each of these may be present in a single patient.[9] Primary injuries are irreversible and its outcome depends upon its severity.
2. *Secondary brain injury* involves additional damage that occurs to the brain after the traumatic event. It can develop within minutes, hours or few days after the primary event due to cerebral hypoxia or ischemia.[10] Usually it occurs due to potentially preventable reasons like hypotension, hypoxia, hypocarbia, hyperthermia, hypoglycemia or increased intracranial pressure (ICP) and, all of which have been shown to independently worsen survival after TBI.[6] If appropriate measures are taken before and after hospitalization, this component of injury can be significantly reduced.

Primary Head Injury

The type and mechanism of injury has an important effect on clinical course after TBI. High velocity injuries involving rapid acceleration and deceleration, particularly if there is a rotational element, may result in shearing forces which can lead to widespread disruption of axonal processes.[11] The primary injury may occur due to direct impact (coup) or by sudden movement of brain within the skull by counteracting forces (countercoup). The countercoup injuries are located at a distance from the point of injury and are not an extension of original injury.

Scalp lacerations: Scalp has generous vascular supply and when lacerated or cut, may result in major blood loss, especially in infants and children.

Skull fractures: Skull fractures occur due to direct impact on head with force. These may be associated with underlying intracranial injuries, especially hematomas. Their presence should raise a high index of suspicion. However, some patients show no evidence of brain damage and show uneventful recovery. Probably this occurs due to dissipation of forces by the fracture.[12]

There may be linear, depressed or basilar skull fractures. Depressed fractures (Fig. 26.1) can cause pressure on the brain and need to be dealt urgently. Open wounds and sinus tear can cause significant bleeding. Early surgery is indicated to control hemorrhage and to prevent meningitis. Fractures of skull base can be suspected when the patient presents with cerebrospinal fluid leak from nose or ears or raccoon eyes. There may be ecchymosis over orbital or mastoid region. These patients predominantly need nonoperative management with measures to prevent meningitis and other complications. Countercoup fractures are seen on roof of orbit and ethmoidal plates following a fall on the back of the head.

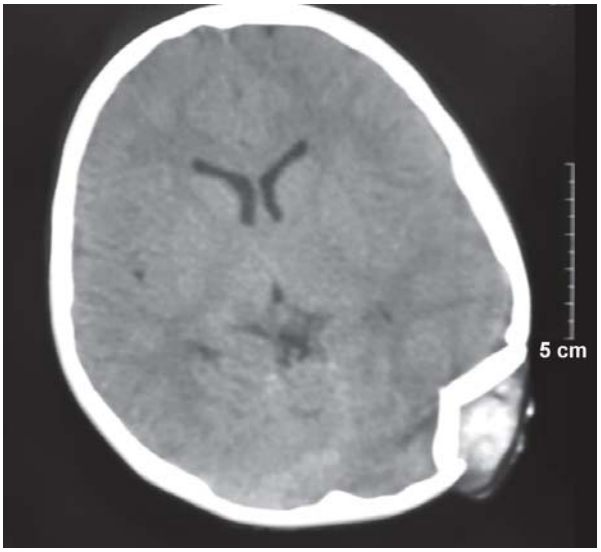


Fig. 26.1: CT Scan showing depressed fracture skull

Subdural hematoma (SDH): These are most common mass lesions occurring in 20 to 40 percent of severe brain injury patients.[13] SDH occurs due to injury to the bridging vessels. This injury carries high mortality rate due to compression of brain parenchyma underneath it and decreased cerebral blood flow. The hematoma is located between the dura and the brain, appearing as a crescent shape, more often taking the contours of brain on CT scan. (Fig. 26.2) It can rapidly create a mass effect

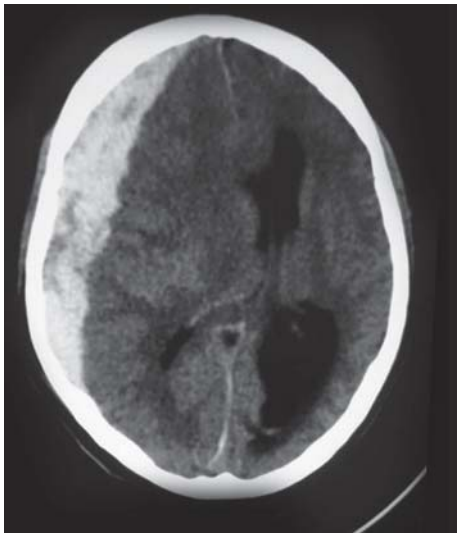


Fig. 26.2: CT scan showing subdural hematoma

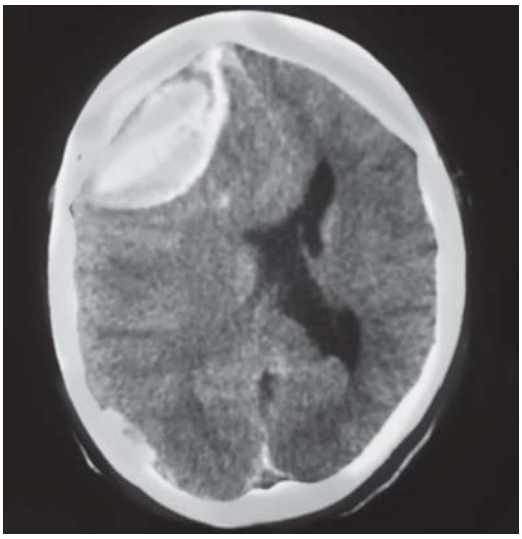


Fig. 26.3: CT scan showing epidural hematoma

and can grow to cover the entire brain surface of a hemisphere. Immediate surgery is required for brain decompression.

Epidural hematoma: Epidural hematomas are located between the skull and the duramater and usually caused by lacerations of middle meningeal vessels, often near the venous sinuses. They are biconvex or lenticular in shape on CT scan (Fig. 26.3). They generally occur in about 9 percent of comatose trauma patients[4] and have better prognosis than subdural hematomas, as their size is often limited by attachments of duramater. The classic presentation is an initial loss of consciousness at the time of injury followed by a 'lucid interval' in which the patient is awake and alert; followed by neurological deficits and sometimes coma due to expanding hematoma reaching the critical volume causing sudden deterioration. This can lead to sudden coma, focal deficits like hemiparesis, aphasia or intracranial herniation. Surgical evacuation is required if its approximate volume is more than 30 ml for supratentorial or more than 10 ml for infratentorial hematomas, thickness on CT scan is more than 15 mm or more than 5 mm of midline shift. Prognosis is good if it is promptly treated before neurological deterioration. Therefore all patients should be closely monitored, even if the patient seems stable.

Intracerebral hematomas and contusion: Cerebral contusions are relatively common in severe as well as moderate brain injury. Contusion can occur beneath the area of injury (coup) or in the area remote from the impact (counter coup). Tips of frontal and temporal lobes are commonest sites. Contusions consist of mixed areas of bruised brain tissue and blood collection (Fig. 26.4). Contusions may evolve over the period of hours to form intracerebral hematoma or coalescent contusion to produce focal deficit or to produce mass effect requiring urgent evacuation. Therefore these patients should be admitted and a repeat CT scan after 12 to 24 hours is recommended. They are likely to develop cerebral edema with reduced cerebral blood flow in the area surrounding the contusion[14] leading to neurological deterioration.

Diffuse brain injuries: Diffuse brain injuries result due to rapid head motion occurring with acceleration and deceleration causing wide spread disruption. Diffuse brain injuries can be classified as concussion, if the loss of consciousness is less than 6 hours and diffuse axonal injury if it is more than 6 hours.[10] Sometimes, severe diffuse axonal injuries (DAI) cause microscopic structural damage occurring throughout the brain which can be permanent. Clinically, DAI is recognized by GCS <8 after resuscitation and a CT scan without focal mass lesion but signs of brain swelling along with presence of a consistent mechanism of injury (rapid acceleration/deceleration/rotation, seen in road traffic accidents).[11] Diffuse axonal injury often takes time to become apparent on CT scan and is usually diagnosed by absence of any mass lesion on CT scan in deeply comatose patient who often has decerebrate or decorticate posturing. It has been suggested that there may be therapeutic window to prevent these changes, however this remains speculative.[15,16]

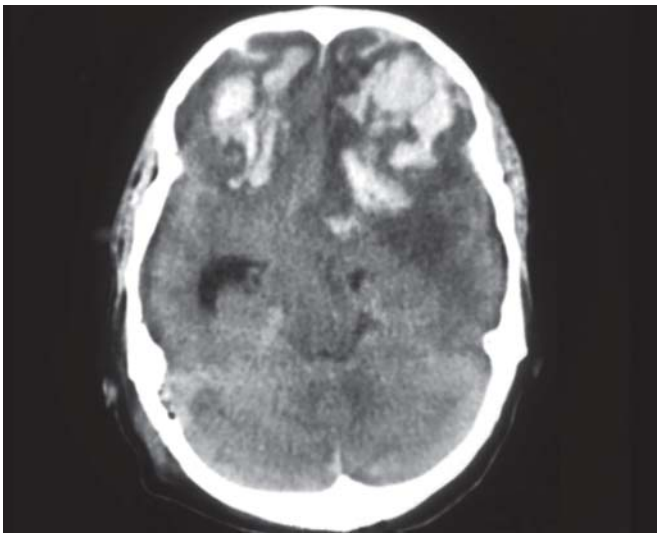


Fig. 26.4: CT scan showing bilateral cerebral contusions

Autonomic dysfunction producing high fever, hypertension and sweating are common following diffuse injuries. These lesions can not be corrected by surgical intervention and have high mortality rates due to increased ICP secondary to cerebral edema.

Penetrating brain injuries: May have associated depressed fractures. The foreign body should be left *in situ* and removed only during the formal operation. (Fig. 26.5) Bullet injuries have high mortality rates, especially with larger bullet and higher velocity. Patient may present with initial GCS < 6. CT scan helps in planning surgical approach, if necessary.

Secondary Brain Injury

Secondary brain insults can arise from both systemic and intracranial causes and may occur at any time during initial resuscitation and stabilization and during intensive care[15] (Table 26.2).

Most common extracranial causes are respiratory dysfunction, inadequate ventilation or pulmonary aspiration leading to hypoxia.[17] There are several intracranial factors aggravating the traumatic brain injury.[18]

Changes in CNS Physiology after Trauma

Pressure autoregulation: In a normal brain autoregulation is maintained between mean arterial pressures (MAP) of 50 to 150 mm Hg. At MAP above 150 mm Hg, the cerebrovascular volume may be markedly increased and the blood brain barrier disrupted often leading to cerebral edema or hemorrhages.[9] This pressure autoregulation may be abnormal or even absent in patients with severe head trauma[19] and such detrimental changes can occur with relatively smaller increase in blood pressures.

Table 26.2: Causes of secondary brain injury

Extracranial causes	*Intracranial causes*
Hypotension	Hydrocephalus
Hypoxia	Vasospasm
Hypoventilation	Cerebral infection
Hyperthermia	Cerebral herniation
Convulsions	Reperfusion injury
Hyperglycemia	Cytotoxic cellular edema
Severe Hypertension	Vasogenic/hydrostatic cellular edema

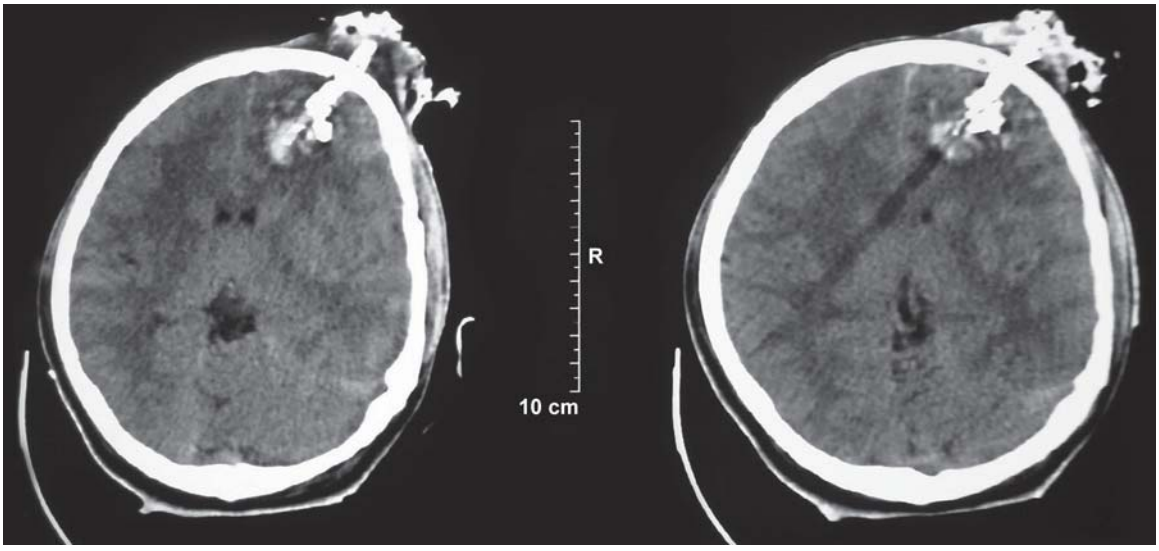


Fig. 26.5: CT scan showing penetrating injury with foreign body in the brain

More importantly, these patients are more at risk from low CPP leading to ischemia.

Metabolic vasoreactivity: In a normal brain, regional and global blood flow is closely linked with the metabolic demands of the brain. Increased metabolic activities produce immediate increase in blood flow by several neural and chemical mechanisms. This coupling is markedly impaired in case of CNS trauma and coma resulting in relative hyperemia contributing to the increased ICP.[19,20]

CO_2 reactivity: In a normal brain CO_2 causes vasodilatation in a linear relationship. Therefore in severe head injury, hypoventilation leads to increased ICP. This concept is an important aspect of management of patients in ICU. In injured areas of brain, such reactivity may be lost in severely head injured patients who had poor outcome. In most other patients CO_2 reactivity is likely to be intact.[19,20]

Due to all above-mentioned changes, in traumatic brain tissue, following effects are seen in three distinct phases.[21,22]

- A severe reduction in CBF occurs in the initial period (3–8 hours) following injury. The CBF can fall below the threshold of cerebral ischemia (18 ml/100G/min) following severe head injury[23]
- In next 1 to 3 days, this is followed by a gradual increase in CBF until eventually, cerebral metabolic needs are exceeded. The metabolism itself will remain depressed in comatose patients
- Vasospasm, which occurs in 20 to 40 percent of patients with head injury, may reduce CBF after this initial hyperemic phase (days 4 to 15), with an increase in the incidence of noncontusion related cerebral infarction.[19]

Both extremely low CBF and high CBF after head injury are associated with poor outcome.

Focal Effects of Head Trauma

In the initial stages following trauma, there is direct tissue damage and impaired regulation of CBF and metabolism initiating an inflammatory cascade (See chapter 24). This 'Ischemia-like' pattern may lead to lactic acidosis, increased membrane permeability and edema formation.[9,19] The intracellular calcium influx occurring after brain trauma is considered as the trigger for further damage by releasing vasoconstrictor agents. These changes result in worsening of tissue edema.[24] Classically, two major types of traumatic brain edema exist: "vasogenic" due to disruption of blood-brain barrier (BBB) resulting in extracellular water accumulation and "cytotoxic" due to sustained intracellular water collection. A third type, "interstitial" edema is caused by osmotic and oncotic imbalances between blood and brain tissue.[11] In the second step there will be membrane depolarization, release of free radicals and release of neurotransmitters that aggravate tissue injury. Following TBI, various mediators are released which enhance vasogenic and/or cytotoxic brain edema. These include glutamate, lactate, H^+, K^+, Ca^{++}, nitric oxide, arachidonic acid and its metabolites, free oxygen radicals, histamine, and kinins.[11]

As ICP and edema increase, within few hours, cerebral perfusion gets compromised and CBF decreases. Any hypotension will correspondingly further reduce CBF and cause ischemia. There is severely reduced cerebral blood supply along with reduced cerebral metabolism in the core area of injury.[10] This may lead to necrosis of the brain tissue. Surrounding the core area a significant zone of the injured brain will have moderately reduced blood supply. If ICP increases or hypotension occurs, this area can become ischemic and the tissue damage can spread to a larger area.

Systemic Effects of Head Trauma

Cardiovascular system: In early stages following head injury, hypertension, tachycardia, increased cardiac output are common findings. Following severe head injury bradycardia may be noted in late stages. In presence of polytrauma or multisystem injuries, hypotension and decreased cardiac output will be more common. Ventricular arrhythmias and heart rate variability are observed frequently following moderate to severe head injury.[25]

Respiratory system: Respiratory insufficiency, spontaneous hyperventilation or apnea, aspiration of gastric contents and neurogenic pulmonary edema are associated with severe head injury. Respiratory complications may account for up to 50 percent of deaths following brain injury.[26]

Hyperthermia: Hyperthermia may occur due to post-traumatic cerebral inflammation, direct hypothalamic damage, or secondary infection resulting in fever. Hyperthermia increases metabolic rate, glutamate release, and neutrophil activity to levels higher than those occurring in the normothermic brain-injured patient. This may further compromise the injured brain and increase the vulnerability to secondary neuronal damage.[27] Diffuse axonal injury and frontal lobe injury of any type are predictive of an increased risk of hyperthermia following severe TBI.[28]

Endocrine dysfunction: Post-traumatic posterior pituitary dysfunction is common. In most cases it is seen as tran-

sient Syndrome of Inappropriate Antidiuretic hormone (SIADH).[29]

Prevention of secondary CNS injuries depends upon prompt diagnosis, early and goal-directed therapy. Thus, the initial management of these patients can significantly affect the outcome.

IMMEDIATE ASSESSMENT

Assessment of all head injured patients should be considered an emergency. The victim should be examined systematically as per advanced trauma life support protocol.[4,30] Prompt attention should be provided to airway, breathing and circulation.

Airway and breathing: The Airway should be rapidly assessed for its patency. It may be jeopardized in head injured patient due to either unconsciousness or due to direct injury to face and upper airway causing bleeding or airway edema. Airway obstruction should be removed and oxygen should be supplemented. All major head injury patients have likelihood of developing respiratory depression and associated pulmonary aspiration.[9] Such patients should be identified at the earliest. Their management is described later.

Circulation: Any external bleeding from scalp should be controlled with external pressure and suturing at the earliest. Hypotension in a head injured patient suggests either end-stage brain injury or a major injury involving other regions. The degree and duration of hypotension can significantly affect the extent of secondary brain injury and the outcome.[31] It has been reported that hypotension of SBP <90 mm Hg in severe brain injury significantly reduced the likelihood of good outcome.[32] Therefore, hypotension should be aggressively treated with intravenous fluids. The goal should be to have euvolemic patient. The older concept of 'dry out the patient' has fallen into disfavor.[9] If the patient's systolic BP cannot be brought above 100 mm Hg despite aggressive fluid resuscitation, the priority should be given to establish the cause of hypotension and neurosurgical diagnosis takes a second priority.[4]

History taking should not delay the basic airway management and circulation support. However the information is important to plan correct line of management. The mode of injury should be asked to judge the possible injuries. High speed vehicular accidents, fall from height more than 10 feet and accidents involving multiple casualties with death of another victim from same compartment are likely to be associated with severe injuries, care need to be taken in these patients even when the patient does not show any obvious signs. Information about the patient's condition immediately after the accident and duration of unconsciousness, if present, should be asked.

Neurological assessment: As soon as cardiopulmonary status is stabilized, rapid and directed neurological examination is performed in 1 to 3 minutes to identify patients in need of urgent management.

Level of consciousness can be assessed in few seconds during the primary survey by AVPU score. This identifies patients who need urgent attention.

- A = Alert
- V = Responds to verbal commands
- P = Responds to painful stimuli
- U = Unresponsive

Glasgow coma scale (GCS) provides a quantitative measure of patient's level of consciousness and should be performed as soon as time permits the examination. It is based on eyes opening, best motor response and verbal response. Table 26.3 shows the modified glasgow coma scale. The maximum score in a normal patient is 15 ($E_4M_6V_5$), whereas deeply unconscious patient without any response will have GCS score of 3 ($E_1M_1V_1$). Based on GCS, head injury patients can be classified as minor head injury (GCS 13 to 15), moderate head injury (GCS 9-12) and severe head injury (GCS 3-8).

It is also important to look for confounding issues during evaluation of traumatic brain injury including presence of drugs, alcohol, intoxicants and other injuries. After traumatic seizures, postictal state will also worsen the patient's responsiveness for significant duration.

Pupillary examination: Both pupils should be examined for size and reaction to light. A difference of 1 mm diameter is considered abnormal. Normal pupil responds briskly to light. If the response is absent or sluggish, it suggests intracranial injury. Dilatation and sluggish response of the pupil is a sign of compression of the occulomotor nerve by the medial portion of the temporal lobe (uncus).[2] A maximally dilated and unresponsive pupil suggests uncal herniation under the falx cerebri. However, such pupillary changes may be present in cases of ocular injuries.

Motor response: To look for evidence of unilateral lesion, spontaneous movements on both sides are compared. If patient is not moving, response to pain may be compared. Delay in onset of movement, less or no movements on one side or need for a stronger stimulus is considered significant. Clearly lateralized weakness suggests possibility of intracranial mass lesion. Best

Table 26.3: Glasgow coma scale

Eye opening		*Best motor response*		*Best verbal response*	
Never	**1**	None	**1**	None	**1**
To pain	**2**	Extension	**2**	Incomprehensible words	**2**
To speech	**3**	Abnormal flexion	**3**	Inappropriate words	**3**
Spontaneously	**4**	Withdrawal	**4**	Confused conversation	**4**
		Localizes	**5**	Oriented	**5**
		Obeys commands	**6**		

motor response elicited is more accurate prognostic indicator than worst response.

Convulsions are common following head injury, especially in pediatric patients. They are more commonly associated with frontal or temporal subdural hematomas or contusions. Patients with concussion may present with persistent vomiting.

In addition, in unconscious patients, cough, gag and corneal reflexes, doll's eyes sign should be assessed. Bleeding or CSF leak from ears, or nose suggest possibility of skull base fracture. Insertion of nasogastric tube should be avoided in these cases. Any patient having GCS < 9 should have a secured airway to protect against aspiration of gastric contents and GCS < 6 should be mechanically ventilated to maintain normocarbia.

It is important to assess GCS and to perform pupillary examination prior to sedating or paralyzing the patient as it is important in determining subsequent treatment. Long acting sedatives or neuromuscular blocking agents should not be used during primary survey.

Sedation should be avoided except when the patient is in agitated state. The shortest acting agents available are recommended when pharmacologic paralysis or brief sedation is necessary for safe tracheal intubation or obtaining good quality diagnostic studies.

Thorough neurological assessment allows the physician to identify patients at high-risk severe injury which may need immediate attention. In these patients, accelerated work-up may be done so as to diagnose and treat the patient sooner, if needed with surgical decompression.

One should never presume that the altered mental status is due to alcohol or other intoxicants. In such cases, the patient should be observed until the mental status becomes normal.

DIAGNOSIS OF HEAD INJURY

Computed tomogram scan (CT scan): Availability of computed tomography has revolutionalized the diagnosis of intracranial abnormalities. It is the diagnostic modality of choice for patients suspected to have head injury. Emergency head CT scan must be obtained as soon as possible after hemodynamic stability is ensured even in multisystem injury patient. Early diagnosis allows precise management planning including the surgical management of the space occupying injuries.

The crucial findings are intracranial hematoma, contusion, shift of midline (mass effect) and compression of basal cisterns. A shift of 5 mm or greater is often indicative of need for surgery to evacuate blood clot or contusion causing shift. If initial CT scan is abnormal or if the patient remains in altered sensorium, a repeat CT scan is performed after 12 to 24 hours to assess the progress of the focal lesion or to identify new abnormalities.

However for the diagnosis, the patient needs to be transferred to the CT scan area, in precarious condition, often with tracheal tube and assisted breathing along with possibility of other, still undiagnosed, associated injuries. First, the patient needs to be hemodynamically stabilized and hypoxemia should be corrected before the transfer and should be continuously monitored during the transfer and in the CT scan suite. A trained physician should always be with the patient at all the times. CT scan of head is indicated in any patient with history of loss of consciousness, headache, amnesia, nausea, vomiting, convulsions, GCS < 15 or focal neurological deficits.

The prognosis of patients with TBI depends upon on admission GCS score, initial findings on CT scan, patient's age and secondary brain injury.

X-ray skull: It can show features like skull fractures or intracranial air, which suggest the increased likelihood of intracranial pathology. In the post CT scan era, its role is limited to highly unstable patients or at places where CT scan is not available.

Cerebral angiography: Cerebral angiography is used in head injured patient when intracranial vascular injury is suspected, e.g. Gunshot wounds or penetrating injuries. Sometimes, when patient shows focal neurological deficits without any evidence on CT scan, angiography may be done to rule out vertebral or carotid injury.[13]

Air ventriculography: It is rarely used in resuscitation area in unstable patients. It may show midline shift suggesting presence of mass lesion, requiring urgent evacuation.[13]

Magnetic resonance imaging (MRI): It has no role in emergency head injured patient.

IMMEDIATE MANAGEMENT

All head injury patients are managed as per the severity of injury as assessed by GCS status on first presentation (Fig. 26.6).

Minor head injury (GCS 13-15): Minor head injury is typically seen as disorientation, amnesia, or transient loss of consciousness in a patient who has regained consciousness and is able to communicate on first presentation. Brief history of unconsciousness is difficult to confirm in presence of alcohol or other intoxicants. Most patients with minor head injury make uneventful recovery with relatively minor sequelae like headache, sleep disturbances or memory loss, but about 3 percent have unexpected deterioration resulting in severe neurological dysfunction. If decline in mental status is detected early, further deterioration may be prevented. Therefore, secondary survey is particularly important in these patients during assessment.

CT scan should be performed in mild head injury if:

- Failure to reach GCS score of 15 within 2 hours of injury
- Clinically suspected open or depressed skull fractures
- Any sign of basal skull fracture
- More than 2 episodes of vomiting
- Age more than 65 years
- Unconsciousness for more than 5 minutes
- Retrograde amnesia for longer than 30 minutes
- Dangerous mechanism of injury
- Severe headache
- Focal neurological deficit
- Patients on anticoagulants.

The patient should be admitted if CT scan is abnormal, the patient remains symptomatic or if neurological deficits are persistent.

If the patient is fully awake, alert, asymptomatic and without any neurological deficits, he/she can be observed for several hours and if still asymptomatic, can be safely discharged, preferably under care of a

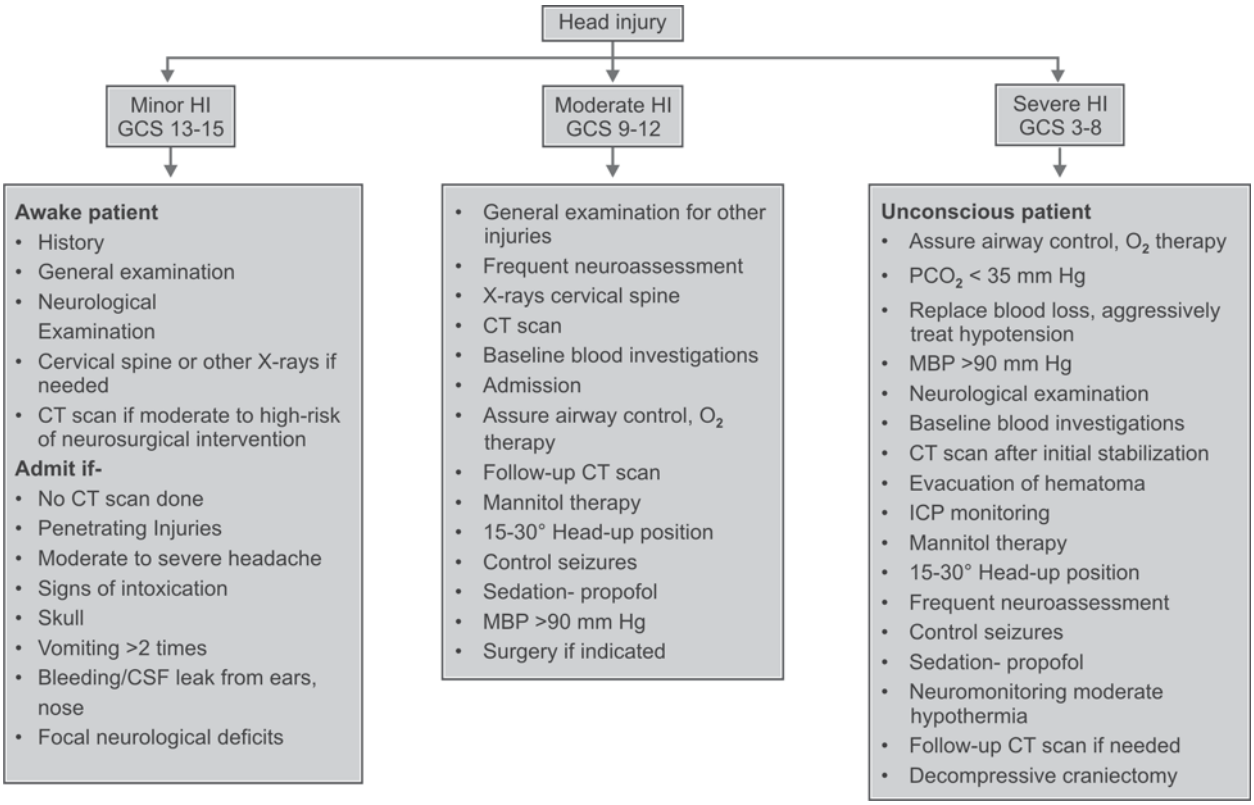


Fig. 26.6: Early management of head injury

companion who can observe the patient continuously for next 24 hours. The patient should be instructed to return to emergency department if headache develops or in case of decline in mental status or appearance of focal deficits. During the observation period, if patient is not alert or oriented enough to answer verbal instructions, decision to discharge may be reconsidered.

Mild head injury patients may appear neurologically normal but continue to be symptomatic for some time. One has to be extra careful that these patients avoid second impact during symptomatic period which can lead to devastating edema. Emphasis must be given for complete follow-up and clearance before resumption of normal activities especially contact sports.

Moderate head injury (GCS 9-12): Moderate head injury constitutes about 10 percent of all head injuries. On admission to emergency department, these patients are able to follow simple commands, but often are confused or drowsy. There may be focal neurological deficits. On admission, rapid primary survey will ensure adequate airway, breathing and circulation. Brief history should be obtained including the prehospital status after injury. Complete neurological assessment should be performed. Serial neurological evaluations are important as about 20 percent of them may deteriorate and lapse into coma. Many of these patients have surgically correctable lesions and therefore, all patients with moderate head injury should undergo an early head CT scan and should be admitted for observation in intensive care area for at least 12 to 24 hours. A follow-up CT scan should be done if the first CT scan is abnormal or if the patient worsens neurologically.

Sudden deterioration in moderate head injury can occur secondary to hypoxia, hypoventilation or hypotension leading to increased cerebral edema, enlargement of hematomas. Sedative or analgesic drugs given can also contribute to hypoventilation and aspiration of gastric contents and add to hypoxia and secondary brain injury. Repeated assessments and timely intubation and ventilator support may be life-saving at times.

Management of severe head injury (GCS 3-8): Patients with severe brain injury are usually comatose on presentation, having Glasgow coma scale less than 9 and they remain so even after cardiopulmonary stabilization. This group includes patients who are at greatest risk of morbidity and carry approximately 34 percent early mortality.[33] These patients are at risk of suffering from additional insult by secondary brain injury. Mortality rate for patients with severe brain injury who have hypotension on admission is more than double that of patient who do not have hypotension.[4] Very young children and older adults have worse outcome with apparent equivalent injuries. Therefore, it is important to aggressively treat hypotension in such patients.

Immediate management of severe brain injury is as follows:

Airway and Breathing

Transient respiratory arrest and hypoxia are common and are important causes of secondary brain injury. Early endotracheal intubation should be performed in comatose patients with GCS 8 or lower. Any further hypoxemia, hypocarbia and hypercarbia must be avoided. A single episode of hypoxia (PaO_2 < 60 mm Hg) in severe TBI is associated with increased mortality. Therefore, oxygen saturation < 90 percent or PaO_2 < 60 mm Hg should be either avoided or treated immediately. If patient arrives in emergency department, tracheally intubated one must confirm proper placement of tracheal tube.

Patient with severe traumatic brain injury may require tracheal intubation for following reasons:

1. Decreased level of consciousness.
2. Increased risk of aspiration.
3. Risk of hypoxemia, hypercarbia.
4. To minimize increases in ICP.
5. To facilitate further diagnostic studies.

Several considerations must be kept in mind prior to intubation of patient with traumatic brain injury including:

1. Skill and expertise of the physician performing the intubation—The most experienced person available should perform the tracheal intubation.
2. About 5 to 10 percent patients of head injury patients have associated unstable spine injury. Therefore, all attempts at intubation should include a trained assistant applying in line manual neck stabilization to minimize further neurological injury.[4,34] Any attempt to immobilize cervical spine degrades the laryngoscopic view.
3. Possibility of skull base fractures—Patients with TBI should generally be intubated orally and not nasally because of potential risk of aggravating the injury. A nasogastric tube can migrate in the brain through basal skull fracture defect.
4. Multiple facial trauma—A surgical airway in form of either cricothroidotomy or tracheostomy, remains a viable and appropriate option in setting of severe facial and neck trauma.
5. Aspiration of stomach contents is a major risk. The patient may have aspirated prior to presentation to the emergency department. Further aspiration can be minimized by employing Sellick's maneuver, i.e.

elevating the patient's chin without displacing the cervical spine and posteriorly displacing the cricoid cartilage to occlude the esophagus.[35] Even though its benefit has never been proved in trauma, it is routinely practiced during rapid sequence induction and intubation as there is little harm.

6. Traditionally positive pressure ventilation is avoided during rapid sequence ventilation to avoid stomach insufflation. However, these patients often have pre-existing hypoxia and increased oxygen consumption due to trauma. It is necessary to correct pre-existing hypoxia by preoxygenation and gentle ventilations preferably before laryngoscopy, so that the patient can have sufficient oxygen reserve during emergency airway management.

Choice of Drugs to Facilitate Intubation

Patients in respiratory arrest and those on hypovolemic shock may not require any induction drug for tracheal intubation. At times in comatose patients, intubation may be performed only with neuromuscular blocking agents. When rapid sequence intubation technique with general anesthesia is needed, each drug should be considered-based on their effects on hemodynamic stability, ICP and the rapidity of onset of action.

Hypotension is extremely detrimental to injured brain while hypertension may increase intracranial pressure (ICP) worsening cerebral ischemia and cerebral herniation. Therefore choice of drugs must be based upon status of each individual patient.

Effects of anesthetic agents on brain have been described in chapter 24. Both propofol and thiopentone sodium are suitable drugs for tracheal intubation in normovolemic stable patients due to their favorable effects on CBF, ICP and $CMRO_2$. However, both can cause severe hypotension particularly in hypovolemic patients due to their vasodilatory and negative inotropic effects. If these drugs are to be given, then the dose should be reduced as their therapeutic effect is exaggerated in hypovolemia.[36] Only 10 to 20 percent of normal dose may be sufficient.

Etomidate is another suitable drug to facilitate intubation in dosage of 0.2 to 0.3 mg/kg in trauma patients. This drug also reduces CBF, ICP and $CMRO_2$. Care must be taken in acutely unstable patient with even etomidate can produce profound hypotension. Ketamine is not preferred due to its ability to increase CBF, and ICP for head injury patients, though it has sympathomimetic effect which is advantageous in a hemorrhagic patient.[37] It can also cause hypotension by its direct myocardial depressant action. Use of fentanyl is of little help during rapid sequence intubation as it has slower onset time. It may be used later for sedation and anlgesia.

Lignocaine is useful to blunt effect of laryngoscopy and intubation, in dosage of 1.5 mg/kg. This drug may decrease ICP with minimal hemodynamic effect.

Choice of neuromuscular blocking agent is somewhat controversial during trauma resuscitation. Administering the agent prevents coughing and resultant spikes of ICP. Agents with faster onset of action like rocuronium (0.9-1.2 mg/kg) or vecuronium (0.1-0.2 mg/kg)[35] are commonly chosen if no apparent difficulty in intubation is expected. However in these doses, long duration of action may preclude neurological examination for 1 to 2 hours. In addition, one must be able to maintain the airway for long duration, should there be problem with tracheal intubation.

Though succinylcholine (1-2 mg/kg) has fastest onset of action for rapid sequence intubation, the main argument against use of this drug in patient with TBI is potential increase in ICP. This can be offset by short-term hyperventilation before its administration. After 72 hours of injury, succinylcholine should be avoided for fear of excessive increase in potassium in presence of spine injuries. It is also contraindicated in cases with crush injury.

In specific conditions with anticipated difficult intubation, spontaneous breathing technique without use of any neuromuscular blocking agent may be preferred during tracheal intubation.

Initial ventilation parameters should include 100 percent oxygen and appropriate adjustments in fraction of inspired oxygen (FiO_2) are made later-based on arterial blood gases. There is no absolute contraindication to the use of positive end expiratory pressure in hypoxemic patients unless the increase in thoracic venous pressure causes an unacceptable increase in ICP.[11] Pulse oximetry is useful adjunct and oxygen saturation of > 98 percent (PaO_2 > 70 mm Hg) are desirable.[4]

Hyperventilation should be used cautiously in patient with severe brain injury and only to prevent acute neurological deterioration for brief periods.

Circulation

Goal of resuscitation in any trauma patient is to maintain organ perfusion. Isotonic intravenous fluids should be administered as necessary to restore intravascular volume and avoid hypotension. The aim is to maintain cerebral perfusion pressure in the range of 60 mm Hg as recommended by updated guidelines from Brain Trauma Foundation in 2007[30] (Table 26.4).

Hypotonic crystalloid solutions may aggravate cerebral edema and therefore are not preferred. Isotonic fluids like 0.9 percent saline (NS) and Lactated Ringer's solution (LR) are commonly used for fluid resuscitation. Rapid and adequate restoration of the intravascular

Table 26.4: Recommendations for management of head injured patient (BTF)

- Blood pressure should be monitored and hypotension below 90 mm Hg systolic should be avoided
- Oxygenation should be monitored and hypoxia < 90 percent or PaO_2 < 60 mm Hg should be avoided
- Mannitol is effective in controlling intracranial pressure in the doses of 0.25-1 g/kg
- Prophylactic hypothermia is not significantly associated with reduced mortality
- Periprocedural antibiotics for intubation should be administered to reduce incidence of pneumonia
- In the first 24 hours following TBI, prolonged hyperventilation ($PaCO_2$ < 35 mm Hg) is not recommended
- Early tracheostomy reduces length of stay on ventilator
- Graduated compression stockings or intermittent compression stockings are recommended to prevent deep vein thrombosis
- ICP monitoring should be done in all patients with severe head injury (GCS 3-8 after resuscitation) and abnormal CT scan
- ICP monitoring should be done in all patients with severe head injury (GCS 3-8 after resuscitation) and normal CT scan if age > 40 years, unilateral posturing or SBP < 90 mm Hg
- Treatment for ICP reduction should be initiated at ICP above 20 mm Hg
- Aggressive attempts to maintain CPP above 70 mm Hg with fluids and pressures should be avoided because of the risk of ARDS. CPP of < 50 mm Hg should be avoided
- Jugular venous saturation < 50 percent and brain tissue oxygen monitoring pressure < 15 mm Hg are treatment thresholds
- Prophylactic administration of barbiturates to induce burst suppression EEG is not recommended
- Barbiturates or propofol administration is reserved for control of elevated ICP refractory to standard medical or surgical treatment
- Early nutrition should be provided to attain full caloric replacement by day 7 postinjury.

volume with isotonic crystalloids and if necessary, with colloid solutions should be done aiming to maintain the CPP > 60 mm Hg, while attempting to minimize further brain swelling. Ringer's lactate solution, which is slightly hypotonic, may promote swelling in uninjured areas of the brain if it is given in large quantities. Edema tends to occur in injured brain regions regardless of the type of solution administered due to increased permeability of the blood-brain barrier.[2]

Colloids maintain oncotic pressures and therefore are beneficial, especially when large volumes of fluids are needed. Drummond et al[38] demonstrated that reduction in colloid oncotic pressure *per se* can also aggravate brain edema after a mild to moderate mechanical head injury. Colloid solutions like hetastarch are associated with lower cerebral edema than crystalloid solutions. Both hyperglycemia and hypoglycemia are detrimental to the brain and hence should be avoided. Hyperglycemia increases the adverse effects of hypoxia on brain by increasing lactic acidosis and therefore should be avoided. Blood glucose levels should be regularly monitored. Fresh whole blood, when available, is ideal replacement solution for hemorrhagic polytrauma patient with ongoing blood loss.[24]

Hypertonic saline infusion is useful to achieve rapid volume resuscitation, with smaller quantities of fluid. It draws water from tissues and hence can reduce cerebral edema. It is found to be useful in prehospital management. In resuscitation of hypotensive patients with TBI, 3 percent NaCl with or without dextran was associated with improved outcome, as compared to patients with isotonic resuscitation.[39] However, in a more recent study such benefit was not observed.[40] Hypertonic fluids such as 3 percent saline may be useful during emergency room resuscitation, although there is insufficient evidence to justify their routine use.

Intracranial hemorrhage does not cause hemorrhagic shock. Primary source of hypotension must be urgently sought and treated. Hypotension is a marker of severe blood loss which may not be always obvious. Associated spinal cord injury (neurogenic shock), cardiac contusion or tamponade and tension pneumothorax are also possible causes requiring immediate treatment.

While efforts are in progress to determine cause of hypotension, volume replacement should be initiated. Normovolemia should be established as soon as possible. If a patient with persistent hypotension has to go to OR for emergency laparotomy, CT scan of head may be done after the surgical hemorrhage control and hemodynamic stability is achieved. In presence of clinical evidence of an intracranial mass, diagnostic burr hole may be undertaken in OR while the surgery is being performed.

FAST or DPL may be used in comatose patient with hypotension to rule out abdominal bleeding as clinical symptoms cannot be assessed in such patient.

It must be emphasized that neurological examination of patient with hypotension is unreliable. These patients should undergo repeat neurological assessment after hypotension is corrected. Patients, who were unresponsive to any form of stimulation during hypotension, may revert to near normal neurological status soon after normal blood pressure is restored.

Vasopressors and ionotropes may be needed after fluid resuscitation to achieve desired CPP. Aggressive attempts to maintain CPP above 70 mm Hg with fluids

and pressures should be avoided due to risk of acute respiratory distress syndrome (ARDS). CPP < 50 mm Hg should be avoided.[41] In the absence of ICP monitoring with known TBI, MAP should be maintained in the 70 to 80 mm Hg range. This recommendation is based on assumption that ICP is between 10 to 20 mm Hg.

Critical care: Most of the management in emergency room while awaiting surgery, operating room and in intensive care unit is directed at minimizing further damage to neural tissue caused by secondary injury.

Earlier, the management was based on reduction of ICP, at times even at the cost of reduction in the cerebral blood flow. However, current management principles of head injured are based on both – reduction of ICP and improving cerebral perfusion. It is now evident that normal or slightly higher systemic blood pressure improves cerebral perfusion and if autoregulation is intact, can reduce the ICP by reflex vasoconstriction. Therefore, cerebral perfusion pressure should be maintained above 70 mm Hg. However, severe hypertension can aggravate cerebral ischemia and edema.

Position of the patient: As raised ICP is associated with poor outcome methods undertaken to reduce ICP and maintain CPP are:

- Elevation of head: Head elevation of 15 to 30° enhances cerebral venous drainage. It should be given gradually. Head elevation in hypovolemic/hypotensive patient will be hazardous and may aggravate hypotension
- Along with head elevation, the head should be kept in neutral position, i.e. not to turn on left or right, as this may decrease jugular venous outflow and may further elevate ICP.

Ventilation: Inadequate ventilation is associated with hypercarbia and hence will increase intracranial pressure. Therefore, all severe injury patients should be monitored for adequacy of respiration. E_TCO_2 is not an accurate indicator, especially with pulmonary pathology and arterial blood gases should be preferred. If needed, mechanical ventilatory support should be provided. All patients with GCS 6 or less should be mechanically ventilated. Thus, current recommendations advise to maintain normocarbia ($PaCO_2$ 35-40 mm Hg) in a patient with head injury.[21,30]

Hyperventilation is an effective way to reduce ICP. However, excessive or prolonged hyperventilation may cause cerebral ischemia by decreasing cerebral blood flow especially in area of low cerebral blood flow.

Patients with isolated head injury can be managed with traditional ventilatory strategy. Those patients with chest trauma, aspiration and massive resuscitation after shock are at high-risk for development of acute lung injury and may be dependent on positive end expiratory pressure (PEEP) for maintaining adequate oxygenation. Though PEEP is not used routinely for head injured patients, to avoid further increases in ICP, in fact, PEEP on the other hand may actually decrease ICP as result of improved cerebral oxygenation and hence improves cerebral perfusion in such patients.[42]

Sedation and analgesia: Adequate sedation and analgesia can alleviate pain, anxiety and agitation, reduce the $CMRO_2$, and facilitate mechanical ventilation. This is commonly achieved by shorter acting drugs like midazolam or propofol along with opioids. Propofol may have benefits over midazolam like its better metabolic suppressive effects and short half-life. However, it has a tendency to accumulate and precipitate hyperlipidemia. Propofol may cause precipitous cardiovascular collapse and 'Propofol infusion syndrome' of metabolic acidosis, rhabdomyolysis, and bradycardia is described in children and in adults.[11]

Blood Pressure and Fluid Management

Maintenance of hemodynamic stability is essential to the management of severe TBI as the altered or absent autoregulation makes these patients vulnerable to hyperemia or ischemia with relatively minor changes in blood pressure, either globally or locally. Hypotension can cause reduction in CBF and therefore can produce cerebral ischemia. On the other hand hypertension can exacerbate vasogenic edema and increase ICP.[11]

Combination of hypotension (systolic blood pressure < 90 mm Hg) and hypoxemia (PaO_2 < 60 mm Hg) at time of hospital arrival has dramatic negative implications for neurologic outcome. Even brief periods of hypotension (systolic blood pressure <90 mm Hg), during first 72 hours post-TBI correlates with doubling the mortality.[43] Therefore, systolic blood pressure < 90 mm Hg should be avoided with goal of MAP > 70 mm Hg until ICP monitoring is instituted and CPP can be directly targeted. The most challenging of all trauma patients are those with severe traumatic brain injury coexisting with hemorrhagic shock.

The earlier practice of 'keep the patients dry' is not consistent with the goal of maintaining CPP and is no longer recommended.[21] Thus, the current recommendation is to maintain the patient with severe brain injury in isovolemic state and fluid resuscitation is mainstay of therapy followed by vasoactive infusion as needed. Initially, intravascular volume should be maintained targeting a central venous pressure of 5 to 10 mm Hg.[44]

Patients with severe cardiac dysfunction after brain injury require invasive cardiovascular monitoring (e.g. pulmonary artery catheter) to accurately guide therapy.[26]

Ideal fluid is not yet been identified. Fluids that provide free water as 5 percent dextrose solution and thereby reduced serum osmolarity will cause edema in normal and injured brain. Reduction in colloid oncotic pressure in traumatic brain has been associated with increased brain edema.[38] Therefore, a reasonable approach would be to avoid profound reduction of colloid oncotic pressure by limiting large amounts of crystalloids alone and using a mixture of isotonic crystalloids and colloids when needed. We commonly use tetrastarches –either saline-based or Lactated Ringer-based as they minimally affect coagulation and do not produce tissue edema. Albumin is expensive and does not offer any advantage over other colloids. In states of increased vascular permeability, colloids tend to leak into the extravascular space, which leads to increases in colloid oncotic pressure in the interstitium, potentially increasing total lung water and pulmonary dysfunction.[45]

Most appropriate colloid to be used is blood when indicated, if ongoing cerebral ischemia is suspected. It may be prudent to maintain higher (27 to 30 percent) hematocrit.

Neurogenic pulmonary edema (NPO) requires aggressive management with positive pressure ventilation and careful restoration of the systemic circulating volume. Even though, patients with NPO and myocardial stunning often appear moribund, they have a good chance of recovery if appropriately managed.[26]

Plasma glucose concentration should be done frequently as hyperglycemia in severe TBI is associated with poor prognosis and reasonable target would be 140 mg percent, though practically tight control is difficult to achieve. Iatrogenic hypoglycemia may increase the morbidity.

Hyponatremia may lead to increased brain edema and seizures, which should be prevented. Most common cause is syndrome of inappropriate antidiuretic hormone (SIADH). Serum sodium levels should be maintained at 140 to 145 mEq/L along with monitoring by serial measurements of plasma sodium. It is important to exclude glucocorticoid deficiency as a treatable and dangerous cause of acute hyponatremia after TBI.[29] Prolonged use of hyperosmolar solutions may cause very high serum sodium levels. A patient with persistent vomiting may have hypokalemia. Potassium supplementation to intravenous fluids will be indicated to correct it.

Mannitol: Mannitol is most widely used osmotic agent in management of raised ICP. It reduces brain bulk due to its hyperosmolarity (20 percent solution – 1280 mOsm/L) by drawing water across intact BBB and reducing cerebral blood volume. Acute neurologic deterioration, such as development of dilated pupil, hemiparesis or loss of consciousness is a strong indication of administrating mannitol.

Most widely accepted dose is 0.25 to 1 gm/kg IV over 15 to 30 minutes. Doses higher than 2 gm/kg may precipitate intravascular volume overload, electrolyte and acid base disturbances. These may also cause pontine hemorrhage and myelin disruption.

Mannitol may be used in repeated doses with monitoring of serum osmolarity. Prolonged administration of mannitol can lead to hypovolemia, hypotension, prerenal azotemia as well as reduction of cerebral blood flow.[46] Systemic dehydration should be avoided by providing adequate intravenous fluids. Mannitol therapy longer than four days is commonly associated with hypernatremia.

Mannitol may have significant rebound effect on ICP in patient with severe head injury with damaged blood brain barrier. Additional therapy may be indicated if intracranial hypertension is refractory to mannitol therapy. In a recent Cochrane review it was noted that mannitol therapy for raised ICP may have beneficial effect on mortality when compared with pentobarbital treatment.[47]

Furosemide reduces CSF production and improves cellular water transport. Some individual may benefit from use of furosemide in combination with mannitol.

Hypertonic saline (HTS): Hypertonic saline can reduce intracranial pressure as well as support circulation.[48] Either 3 percent or 7.5 percent solution of hypertonic saline is used at rate of 20 to 40 ml/hr titrating against serum osmolarity, serum sodium levels and ICP.

Following effects have been noted.

- Plasma volume expansion and increased cardiac output[48]
- Reduction in intracranial pressure following bolus dose as well as infusion
- No diuretic properties, hence does not cause hypotension
- Increase in extracellular sodium helps to restore normal cell polarity and hence, transport across the cell membrane
- Increases vessel diameter and counters cerebral hypoperfusion and vasospasm
- A 10 to 15 mEq/L rise in serum sodium was found to lower ICP for about 72 hours[49]

- Can reduce inflammation around the injured brain area by increasing the circulating levels of cortisol and ACTH[50] and thus, can decrease the interstitial edema and protease mediated cell death.

In refractory intracranial hypertension 7 percent hypertonic saline was found to be more effective than 20 percent mannitol.[51] Horn et al studied the effects of a bolus dose of 7.5 percent HTS on elevated ICP refractory to both barbiturates and mannitol. A decrease in ICP from a mean of 33 mm Hg to 19 mm Hg was observed after 1 hour and lasted for 3 hours.[52]

Adverse effects: Rapid rise in serum sodium can lead to osmotic demyelination syndrome (ODS), acute renal insufficiency and hematological abnormalities.[48] Rebound increase in ICP has also been noted.[49]

Temperature Management

Hypothermia decreases ICP by decreasing cerebral metabolic rate and thus cerebral blood flow. There is no clear evidence that hypothermia is beneficial in the treatment of head injury as per recent Cochrane analysis. The benefit was only observed in low quality trials. Routine use of hypothermia is not recommended for head injured.[53,54] In contrast to hypothermia: devastating effects of hyperthermia are well-established and fever in brain injured patient must be treated promptly and vigorously.

Pharmacotherapy

Use of steroids: Steroids have profound anti-inflammatory action and can reduce brain edema. They have been used for brain trauma in the past without evidence for their benefit. However, the results of multicentric CRASH trial involving study of corticosteroids in head injury showed that the risk of death was higher in the corticosteroid group than in the placebo group. It was recommended that corticosteroids should not be used routinely in the treatment of head injury.[55]

It quiet possibly increases mortality and morbidity because of increased incidence pneumonia and infection.

Barbiturates: Barbiturates are effective in reducing ICP refractory to other measure. They should not be used in the presence of hypotension or hypovolemia as hypotension often results from their use. Propofol may function in similar manner as barbiturates. Both barbiturates and propofol can be used in dose response manner to provide ICP control ranging from mild sedation to pharmacologically induced coma. Thiopental is most commonly used agent in operating room.

Anticonvulsant: Seizures produce rapid and sustained rise in ICP. It may precipitate cerebral herniation. Post-traumatic seizures occur in 5 percent patients admitted to hospital with closed head injury and in 15 percent of those with severe head injuries. Prophylactic phenytoin therapy reduces incidence of seizure in first week of injury but no thereafter.

For patients with prolonged seizures, diazepam or lorazepam are used in addition to phenytoin until seizure stop. The seizures should be controlled as soon as possible, as prolonged seizures (30-60 minutes) may cause secondary brain injury. This can be reduced with antiseizure therapy and not with neuromuscular blocking agents. Seizures, fever, pain all increase $CMRO_2$ and cerebral blood flow.

THAM or Trishydroxymethyl-aminomethane is used in some centers to maintain normal pH in presence of intracranial hypertension, but its efficacy is still equivocal.[21]

Surgical decompression: It has been used to manage intractable intracranial hypertension after TBI. Wide bilateral fronto-parietal craniotomy with lax duroplasty is commonly done in absence of any operable mass. This has shown to improve brain tissue oxygenation. Potential complications are increased cerebral edema and infection.

Oral anticoagulants and head injury: Increasing number of patients on warfarin, clopidogrel, and aspirin therapy for various reasons are presenting with head injury. There is high mortality rate associated with aspirin or clopidogrel or both in elderly patients who have head trauma resulting in intracranial hemorrhage.[56] Age was a significant predictor of mortality independent of the drug use. The triad of anticoagulation with warfarin, age greater than 65 years, and traumatic head injury frequently produces a lethal brain hemorrhage.[57] All patients on warfarin should have a CT scan and coagulation profile done. All patients with supratherapeutic INR or abnormal CT scan should be admitted for neurological observation and short-term reversal of anticoagulation should be considered.[58] Treatment with fresh frozen plasma (FFP) and vitamin K is indicated when INR exceeds 1.4. Activated recombinant factor VII may be useful as immediate therapeutic measure under these circumstances, though the effects are short lived. Platelet transfusion is indicated when count is <100,000/cmm. Platelets may be given when a patient is receiving aspirin and is clinically bleeding despite normal platelet count though there is no consensus for this. The patient should be followed-up clinically and with repeat CT scan to identify enhancement of hematoma.

Systemic hypothermia can aggravate coagulopathy, therefore is avoided in these patients with TBI.

MONITORING INJURED BRAIN

ICP Monitoring

The decision to measure ICP in a TBI patient not scheduled for surgery is most closely related to patient's neurological status and ability to provide clinical examination. Unless there is severe coagulation derangements, ICP should be monitored in presence of severe head injury GCS ≤ 8 and abnormal CT scan. It should be inserted early during the management of patients with normal CT scan if age is >40 years, in presence of motor posturing and systolic blood pressure <90 mm Hg.[21] Other possible indications are—in patients with head injury who are unable to follow neurologic examination due to:

1. Tracheal intubation and deep sedation or paralysis.
2. Immediate non-neurosurgical procedure (duration >2 hours) in mild to moderate TBI.

Intracranial hematoma and cerebral edema may appear or worsen after initial CT scan up to 48 hours after injury. Therefore, frequent neurological examination or continuous ICP monitoring is justifiable in this time period.

Intracranial pressure can be measured by various techniques. The most common one is insertion of intraventricular catheter. This method is accurate, can be easily calibrated and relatively less expensive. Along with pressure monitoring, it also allows continuous or intermittent drainage of CSF to control intracranial hypertension.[44] Its main disadvantage is increased risk of infection. Alternatively, a microsensor may be placed in brain parenchyma (Codman). Another system uses fiberoptic tipped pressure monitoring technique. Multiparameter monitoring system can monitor pressure, temperature (Camino Multiparameter monitor) and pH or microdialysis. As some monitors cannot be recalibrated, a drift may impair the accuracy over longer periods. These systems are relatively easy to insert and are associated with fewer complications. Other less accurate techniques for ICP monitoring include, subdural bolt, epidural transducer or subdural catheter. With the advent of newer systems these are now rarely used.

Normal upper limit of ICP is 10 to 15 mm Hg and treatment is recommended when ICP exceeds 20 to 25 mm Hg. Based upon the ICP and MAP, arbitrarily CPP >60 mm Hg is targeted.[21]

Jugular Venous Oxygen Saturation

Jugular venous saturation can be used to guide the therapy for head injured. The catheter is inserted retrograde into the jugular bulb. Venous oxygen saturation is measured either continuously or intermittently.[1] $SjvO_2$ < 50 percent is considered as critical desaturation, most commonly due to inadequate O_2 or blood supply and measures should be employed to increase the same. On the other hand, nonfunctioning brain extracting little oxygen will result in high $SjvO_2$ value (>50 percent) and both the situations are associated with poor outcome. This monitoring is particularly useful when measures such as hyperventilation which reduce global CBF without reducing cerebral metabolic rate are used.

The brain's oxygen extraction can be measured by calculating the difference between arterial and jugular venous oxygen content ($AjvDO_2$).

$$AjvDO_2 = 1.34 \times Hb \times (SaO_2\text{-}SjvO_2)$$

Normal values are approximately 6 ml O_2/100 ml blood. Higher value indicates insufficient cerebral blood flow and lower values are present with hyperemia.[1] A reduction in ICP with increase in CPP will increase $SjvO_2$ and reduce $AjvDO_2$. Following severe head injury the cerebral $AjvDO_2$ increases in the first few hours and then gradually decreases. After 24 hours of injury reduction in CBF is not accompanied by increased $AjvDO_2$.[44] However, these changes reflect the global perfusion and can not be applied to assess adequacy of regional blood flow.

In addition, lactate levels on blood samples drawn from this catheter can reveal anerobic metabolism in brain if they are higher than that in arterial blood drawn simultaneously.

This technique provides information of global balance of cerebral blood flow and metabolism; and does not reflect status of injured area. $SjvO_2$ values can be in the normal range despite small regional areas of ongoing ischemia.

Brain tissue oxygenation: Brain Tissue Oxygen Tension ($PbtO_2$) is monitored by placing microsensors in the deep white matter of brain parenchyma. This monitor provides a continuous measurement of oxygen tension in brain parenchyma which reflects local balance between supply and demand of oxygen. Some intraparenchymal catheter may combine ICP monitoring with brain tissue oxygenation ($PbtO_2$). Normal $PbtO_2$ are approximately 32 mm Hg. Studies have shown that the patients with $PbtO_2$ of less than 15 mm Hg had poor outcome.[59] A $PbtO_2$ of 10 mm Hg is considered as a critical value, below which aggressive attempts should be made to increase O_2 delivery to the brain.

The best method for intervening for low $PbtO_2$ has not been established, however presumably any inter-

vention that increases CBF, decreases $CMRO_2$ or increases oxygen content of blood would improve $PbtO_2$.

Cerebral microdialysis (MD): Cerebral MD is a well-established laboratory tool and is being increasingly used as a bedside monitor. It allows sampling and collecting of small-molecular-weight substances from the interstitial space of the brain. From the sample common markers of metabolism like glucose, lactate, pyruvate, glycerol and glutamate are analyzed. As during ischemia, various pathological processes lead to failure of cellular metabolism, calcium overload, increased production of free radicals and release of neurotoxic levels of excitatory amino acids,[60] their levels reflect presence of hypoxic oxygenation and metabolic disturbances after brain injury. Elevated Lactate-pyruvate ratio (LPR) is a sensitive marker of ischemia after ABI. The brain metabolism may be altered without changes of cerebral oxygenation.[61] A several-fold increase is noted in severe ischemia after head injury. As cerebral MD measures changes at the cellular level, it also has the potential to detect hypoxia/ischemia before changes can be detected by the patient's neurological status or by more conventional monitoring techniques such as ICP measurement. Hence, the specific treatment can be started early probably before irreversible cellular damage takes place. The MD catheter should be placed in the 'at-risk' tissue, such as the area surrounding a mass lesion after traumatic brain injury (TBI), so as to detect this focal neurochemical changes.[60]

Anesthesia Management

Surgical intervention may be needed with all types of head injury. Immediate surgery is usually required for patients with large intracranial hematomas, causing mass effect and in those patients showing rapid deterioration.

Intraoperative anesthesia management in head injured patient is actually a continuation of resuscitation, which has been started in the emergency room and extends into the postoperative phase in the neurointensive care unit.

Preoperative assessment: An anesthesiologist, who arrives on the scene at this point of time, needs to reassess the patient completely. The information about the condition on arrival and the treatment received should be sought. After ensuring the stability of airway and circulation, neurological examination should be quickly carried out and documented. Possibility of alcohol or other intoxicants affecting the level of consciousness should be noted. However, if the patient has been intubated or given medications, this may not be possible.

All associated injuries should be identified. Surgery for uncontrolable bleeding gets priority over neurosurgery.

Pulmonary, cardiovascular and renal status along with laboratory parameters should be evaluated. Isolated head injury generally does not result in systemic hypotension in adults, although this can occur in small children with major blood loss from the scalp. The patients with increased ICP may have hypertension, bradycardia and papilledema (Cushing's response). Serum electrolytes and arterial blood gases will identify metabolic derangements. Endocrine and electrolyte abnormalities like hypokalemia, hyponatremia, hypernatremia or hyperglycemia often accompany severe head injury for which corrective measures should be started. It is important to assess coagulation function for neurosurgical procedures to avoid perioperative bleeding problems. Electrocardiogram may show ischemic changes or arrhythmias. They are more common with severe head injuries. Chest radiograph should be obtained, especially in polytrauma patients.

Sedative premedication is avoided prior to emergency craniotomy. Pre-existing hypoxemia should be corrected at the earliest.

Intraoperative monitoring: Intraoperative routine monitoring includes electrocardiogram, pulse oximetry, capnography, invasive arterial blood pressure, central venous pressure, core temperature and urine output. In addition specific brain monitoring like ICP, pulmonary artery catheter, ABG or other monitors may be used as per the need. When it is technically possible to place electrodes on skull, either BIS or Entropy monitoring should be used to guide the administration of the anesthetic agents, aiming for a value below 60. Monitoring cerebral oxygenation using Near infrared spectroscopy, brain tissue oxygen monitoring or jugular venous oximetry may be continued inraoperatively and are helpful in management of ventilation and blood pressure.

Whenever possible, the cervical spine should be 'cleared' before the patient needs tracheal intubation. If it is not done, precautions should be taken to minimize cervical spine movements during the patient transfer, laryngoscopy and tracheal intubation.

As the surgical procedure involves sudden blood loss, two large bore peripheral intravenous cannulae should be inserted. A central venous access preferably through subclavian vein allows CVP measurement for fluid balance and also provides a route for medication and faster fluid administration.

Induction and intubation: The main goals of anesthesia are to maintain the cerebral perfusion to optimum,

minimize further increases in ICP, to prevent secondary brain damage and to provide adequate surgical conditions.

Choice of anesthetic agents will depend upon the individual patient status and the most critical injury. Factors that should be considered when anesthetizing head trauma patients include the effects of anesthetic agents on the cardiac and respiratory systems, their effects on cerebral blood flow (CBF), ICP and possible neuroprotective benefits offered by certain agents.

The basic principles of neuroanesthesia are still applicable while choosing drugs for anesthesia induction and maintenance. As described earlier, both propofol and thiopentone in titrated doses are satisfactory agents for head injured patient as they reduce intracranial pressure and cerebral oxygen requirements. If the patient has associated major trauma involving other systems, these agents may cause sudden hypotension. Adequate volume loading is needed to avoid the complication. Alternatively the patient may be induced with opioids with benzodiazepine- usually midazolam. Etomidate is another suitable alternative, but may cause hypotension in hypovolemic patients. Use of ketamine is debatable. It should be noted that in hypovolemic patients, due to reduction in circulating blood volume, reduced dosage of anesthetic agents are needed to produce anesthesia.

Opioids in clinical doses produce minimal decrease in CBF and $CMRO_2$ and cause minimum effect on ICP, if ventilation is maintained. They do not cause direct myocardial depression or systemic vasodilatation. Therefore, opioids like fentanyl provide good analgesia and permit lower concentrations of anesthetic agents to be used in unstable head injury patients.[9] Alternatively remifentanil infusion may be started along with appropriate dose reduction of propofol or other agents. Sufentanil and alfentanil may increase ICP.

Use of neuromuscular blockade facilitates tracheal intubation, mechanical ventilation, help in preventing rise in ICP and avoid coughing and straining.[9] All depolarising neuromuscular blocking agents like vecuronium, pancuronium, atracurium or rocuronium have minimal or no direct effect on ICP and hence may be used for head injured.

Preoxygenation for 3 minutes should be done. Care should be taken not to increase intracranial pressure further during laryngoscopy and intubation. This is commonly achieved by:

- Correction of hypoxia and hypercarbia by preoxygenation and if needed, by mask ventilation
- Avoiding agents which adversely effect brain dynamics
- Adequate depth of anesthesia
- Supplemental fentanyl 1 to 2 mg/kg if adequate time is available
- Use of adequate neuromuscular blocking agents to prevent coughing response
- Use of agents like lignocaine (1-1.5 mg/kg IV) or esmolol (0.5 mg/kg IV) before laryngoscopy
- Use of faster intubation techniques.

If patient arrives in or with a tracheal tube, its correct position in trachea must be checked and if needed, it may be changed with another appropriate tracheal tube.

Maintenance: The anesthetic technique that reduces ICP, maintain blood supply to brain and protect it against ischemic-metabolic insult is suitable for a head injury patient. Both intravenous (propofol-opioid) and inhalational techniques (Isoflurane/Sevoflurane with opioids) may be used for maintenance of anesthesia. A target controlled infusion of propofol may be started at 2 mg/ml and then adjusted as per patient response provides satisfactory anesthesia.

All inhalational anesthetic agents cause dose dependent increase in CBF through direct vasodilatation and thereby can increase ICP. These effects can be offset by employing mild hyperventilation. Halothane causes significant vasodilatation and increase in ICP and therefore should be avoided. Sevoflurane has less influence on CBF and ICP than halothane, and to a lesser extent than even isoflurane or desflurane. Desflurane in higher dosage appears to increase ICP. Commonly low concentrations (< 0.5 MAC) of inhalational agents like sevoflurane are used for maintenance.

Nitrous oxide also increases $CMRO_2$, CBF and ICP in head injury patient and hence often avoided. When it is used, it should be started only after employing mild hypocarbia. As it can rapidly enlarge air pockets, N_2O is contraindicated in presence of pneumoencephalus, which is often present with frontal bone fractures. Boluses of fentanyl may be given before skull pin insertion or similar noxious stimuli.

General care during the surgery includes, care of eyes and covering with soft padding, proper fixation of tracheal tube and avoiding kinking or other obstruction as these areas are not accessible to anesthesiologist, once the surgery starts. Venous drainage from the brain should not be impeded with excessive neck flexion or rotation.

Mild hyperoxia can improve cerebral oxygenation during treatment of systemic hypotension or increased ICP.[62]

Normocarbia (PCO_2 30-35 mm Hg) should be maintained. Routine use of PEEP is not advocated. If patient has probable impending cerebral herniation, hyperventilation (PCO_2 25-30 mm Hg) may be used for short duration till craniotomy is performed and duramater is

opened. Elevation of ICP is more detrimental than effect of short term hyperventilation.

Patients with increased ICP often have hypertension as a compensatory mechanism to maintain adequate perfusion of brain. This hypertension should be accepted before cranium has been opened. When ICP is relieved surgically as happens with removal of extradural hematoma or opening of duramater, sudden loss of sympathetic drive can result in profound hypotension especially in hypovolemic patients, if adequate intravenous fluid has not been restored. Volume loading in early stage of anesthesia is appropriate particularly in a patient with other injuries and significant blood loss as hypertensive response often mask hypovolemia. Patients with poor GCS, absence of mesencephalic cisterns on CT scan and dilated pupils are 'at risk' of sudden hypotension with > 20 percent reduction in MAP on dural opening.

Hypotension during this period should be treated aggressively. Mean arterial blood pressure should be at least 80 mm Hg. Normovolemia should be maintained. Combination of fluids and vasopressor should be accomplished this goal. Until adequate volume resuscitation is accomplished, small bolus dosage of epinephrine may be necessary. A hematocrit of 30 percent provides optimum oxygen delivery and judicious blood transfusion should be used to achieve this goal.

Use of vasodilators like sodium nitroprusside or nitroglycerin to treat intraoperative hypertension is associated with unacceptable cerebral vasodilatation. Instead, use of barbiturates, propofol or opioids is preferred. Small doses of esmolol may be used to prevent sudden increases in blood pressure.

Emergence: Patients, who were comatose with lower GCS score prior to surgery and those who had intraoperative high ICP, should remain intubated for neurosurgical purpose in postoperative period. When the anesthetic agents are discontinued, additional opioids are commonly given to provide transitional analgesia as well as sedation. The pulse oximetry, capnometry, electrocardiography and ICP monitoring is continued during the transfer and in the ICU.

Postoperative management: After evacuation of hematomas, there may be hyperemia with regional edema in the tissue which was initially compressed. This should be anticipated and measures to prevent rise in ICP should be taken. If swelling is apparent at the time of surgical closure, 'open craniotomy' without replacement of bone graft may be considered. It may be appropriate to control ventilation for few hours.

Post-traumatic vasospasm may occur in as many as 40 percent of severely brain injured patients.[9] Usually this occurs early and is less severe than aneurysm patients.

Non-neurosurgical Procedures

Management of a trauma patient, with extracranial life-threatening injuries, but who also have concurrent TBI is complex and can be an anesthetic challenge. These patients are often critically ill and at high risk for significant morbidity and mortality.

At times, a bleeding patient may have to be taken directly to operation room for immediate life saving procedure prior to undergoing adequate neurologic examination or head CT. In this situation, main goal is resuscitation and correction of hypotension. The most immediate life threatening condition must be taken priority but potential presence of TBI should be kept in mind. Specifically history on level of consciousness should be obtained and pupil examined which suggest high likelihood of intracranial injury. Consultation with neurosurgery should be obtained to initiate ICP monitoring. During the surgery the goal should be to do least harm to the patient. After the patient is hemodynamically stabilized, management of head injury gets priority.

Timing of surgery for non-life threatening extracranial injuries in a head injured patient is highly controversial as these are associated with increased incidences of hypoxia and hypotension. Following factors should be considered to plan the timing of surgery.

- Severity of brain injury
- Presence of hypotension and the patient's response to therapy
- Presence and severity of pulmonary dysfunction coagulopathy and hypothermia
- Risks of delaying the surgery

The need and urgency should be discussed with the surgeons. The non-urgent procedures like long bone fractures are delayed till head injury status is stabilized-usually for 48 to 72 hours.

When an urgent non-neurological surgery, e.g. limb saving vascular injury, cleaning and debriding of compound fractures, etc. is needed for associated injuries, e.g. crush injury, fractures, in presence of severe head injury, one should continue all the critical management the patient is receiving prior to surgery.

General anesthesia is commonly used and central neuraxial blockade is not recommended in presence of moderate to severe head injury. Increase in ICP and

hypotension both are avoided. Monitoring includes ECG, pulse oximetry, capnography, arterial blood pressure and CVP monitoring. In addition, continuation of brain monitoring especially ICP monitoring allows the anesthesiologist to do precise adjustments in anesthesia, fluid and blood pressure management. Monitoring brain tissue oxygenation can identify brain ischemia that may occur secondary to inadvertent hyperventilation ($PaCO_2$ < 30 mm Hg) or hypotension. The anesthetic agents chosen should be in accordance with the principles of neuroanesthesia and should not aggravate the secondary brain injury. Head low position is avoided. Whenever possible, 15 to 20° head up position should be used. Normovolemia should be maintained using crystalloids and in volume and colloids. Analgesia with opioids, with or without additional NSAIDs may be used. Peripheral procedures may be performed using regional anesthesia.

Head Injury in Children: Children with head injuries usually present with normal BP and tachycardia. In infants, large scalp wounds and major hematoma (subgaleal blood collection) can result in anemia and hypotension. Maintaining adequate basic airway, breathing and circulation is more important initially than sending a child with a head injury straight to the CT scan. If the child is alert and fully conscious, CT scan should be done if fall from a significant height, fall on to a hard surface, presence of tense fontanelle and suspected nonaccidental injury. The optimum CPP recommended for children is lower than in adults. (approximately 60 mm Hg.) There should be low threshold for admission in an ICU close monitoring. Hyperventilation is easy in children and may lead to severe hypocarbia and dangerously low cerebral perfusion.

Head injury in pregnant patient: The pregnant trauma victim presents a unique spectrum of challenges. Even mild, head injury during pregnancy can threaten either the maternal or the fetal life. Initial management is directed at resuscitation and stabilization of the mother that taken precedence over that of fetus.[63] Radiological examinations should not be deferred because of the presence of the fetus, though the fetal loss is possible if radiation exposure occurs within 2 weeks of conception. Radiological examinations should be interpreted in the context of pregnancy-related changes, e.g. increased AP diameter of chest, cardiomegaly, and a slightly widened mediastinum. All pregnant trauma victims of 20 to 24 weeks of gestational age or greater should be placed on a fetal monitor. Fetal distress may be the first sign of maternal hemodynamic compromise because the mother will maintain her vital signs by shunting blood away from the relatively low-resistance uterus. Monitoring for 24 hours is suggested with major trauma or any signs of obstetric decompensation.

The airway mucosal edema, increased oxygen requirements, decreased functional residual capacity and the 'full stomach' status increase the difficulty of airway management and decrease the margin of safety.[64] One should balance the health and well-being of the fetus against the mother's need for surgery. An increase in risk to the fetus occurs from surgery alone especially in the first and third trimesters. The large crystalloid infusions may cause pulmonary edema earlier in pregnant women. There is increased risk of pulmonary embolism in pregnant patient. Special care should be taken to position the patient in third trimester to avoid supine hypotensive syndrome and optimize venous return. Hypotension should be rapidly treated as uterine blood flow can reduce. Hypoglycemia can occur rapidly especially in head injured patient due to glucose restrictive strategies of fluid replacement. Therefore blood glucose monitoring and the clinical monitoring of patient is essential. Other perioperative management principles remain same.

Perimortem cesarean delivery may be needed for two indications:

- In the case of certain maternal death due to severe head injury or other nonrevivable cause: to save the fetus. Here the fetal well-being takes the precedence.
- In the case of maternal cardiac arrest due to a potentially resuscitatable cause such as a cardiac event or severe blood loss: to remove the fetus from the maternal circulation and therefore, facilitate the maternal resuscitation.

SUMMARY

All head injured patients should be treated as emergencies. No time should be wasted in diagnosing and treating head injury. When in doubt, perform the CT scan. All pathophysiological insults that may cause secondary brain injury should be avoided or treated. Minimise ICP and maintain CPP throughout the management. Provide definitive management early. In case of multiple traumas manage the most life threatening situation first. Be prepared to change your plans as per the patient's physiological status and the response to therapy.

REFERENCES

1. Capan LM, Miller SM. Trauma and Burns. In: Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anesthesia, 5th edn. Philadelphia: Lippincott Williams and Wilkins; 2006; 1262-97.

2. Duke JC. Anesthesia. In: Feliciano DV, Mattox KL, Moore EE (Eds). Trauma. 6th edn. New York: McGraw Hill; 2008. pp. 339-361.
3. Turner DAB. Emergency Anesthesia. In: Aitkenhead AR, Rowbothom DJ, Smith G (Eds). Textbook of Anesthesia. 4th edn. Edinburgh: Churchill Livingstone; 2001;619-28.
4. Advanced Trauma Life Support for Doctors. Student Course Manual. 8th edn. Chicago: American College of Surgeons; 2008.
5. Hulme J. Resuscitation of patients after traumatic brain injury. Trauma 2008; 10(1): 55-63.
6. Helmy A, Vizcaychipi M, Gupta AK. Traumatic brain injury: intensive care management. Br J Anaesth 2007; 99(1):32-42.
7. Hyam JA, Welch CA, Harrison DA, Menon DK. Case mix, outcomes and comparison of risk prediction models for admissions to adult, general and specialist critical care units for head injury: a secondary analysis of the ICNARC Case Mix Programme Database. Crit Care 2006; 10:1–11.
8. Clayton TJ, Nelson RJ, Manara AR. Reduction in Mortality from severe head injury following introduction of a protocol for intensive care management. Br J Anaesth 2004; 93:761-7.
9. Atkinson JLD, Faust RJ. Central Nervous System Trauma. In: Cucchiara RF, Black S, Michenfelder JD (Eds). Clinical Neuroanesthesia, 2nd edn. New York: Churchill Livingstone; 1998; pp. 539-55.
10. Sakabe T, Bendo AA. Anaesthetic Management of Head Trauma. In: Newfield P, Cottrell JE (Eds). Handbook of Neuroanesthesia, 4th edn. New Delhi: Lippincott Williams and Wilkins; 2007; pp. 91-110.
11. Unterberg AW, Stover J, Kress B, Kiening KL. Edema and brain trauma. Neuroscience 2004; 129:1021-9.
12. Granacher RP, Jr. Traumatic Brain Injury: Methods of Clinical and Forensic Neuropsychiatric assessment. (2nd edn). Bica Raton, FL: CRC Press; 2008.
13. Letarte P. The Brain. In: Feliciano DV, Mattox KL, Moore EE (Eds). Trauma. 6th edn New York: McGraw Hill Medical; 2008. pp. 397-418.
14. Steiner LA, Coles JP, Johnston AJ et al. Responses of post-traumatic pericontusional blood flow and blood volume to an increase in cerebral perfusion pressure. J Cereb Blood Flow Metab 2003; 23:1371-7.
15. Povlishock JT, Becker DP. Fate of reactive axonal swellings induced by head injury. Lab Invest 1985;52:540-52.
16. Povlishock JT. Traumatically induced axonal injury: pathogenesis and pathological implications. Brain Pathol 1992; 2:1.
17. Popp AJ, Feustel PJ, Kimelberg HK. Pathophysiology of Traumatic Brain Injury. In: Wilkins RH, Rengachary SS, (Eds). Neurosurgery. Volume III 2nd edn. New York: McGraw Hill; 1996. pp. 2623-37.
18. Sutcliff AJ. Traumatic Brain Injury: Critical Care Management. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma: Critical Care. New York: Informa Healthcare; 2007; 201-19.
19. Werner C, Engelhard K. Pathophysiology of traumatic brain injury. Br J Anaesth 2007; 99:4-9.
20. Lee JH, Kelly DF, Oertel M, McArthur DL, Glenn TC, Vespa P, et al. Carbon dioxide reactivity, pressure autoregulation, and metabolic suppression reactivity after head injury: a transcranial Doppler study. J Neurosurg 2001; 95:222-32.
21. Seppelt I. Intracranial hypertension after traumatic brain injury. Indian J Crit Care Med 2004; 8(2):120-6.
22. Martin NA, Patwardhan RV, Alexander MJ, Africk CZ, Lee JH, Shalmon E, et al. Characterization of cerebral hemodynamics phases following severe head trauma: hypoperfusion, hyperemia and vasospasm. J Neurosurg 1997; 87:9-19.
23. Bouma GJ, Muizelaar JP, Choi SC, Newton PG, Young HF. Cerebral circulation and metabolism after severe traumatic brain injury: the elusive role of ischemia. J Neurosurg 1991; 75(5):685-93.
24. Bendo AA, Cass IS, Hartung J, Cottrell JE. Anesthesia for Neurosurgery. In: Barash PG, Cullen BF, Stoelting RK, (Eds). Clinical Anesthesia 5th edn. Philadelphia: Lippincott Williams and Wilkins; 2006. pp. 746-789.
25. Rotem M, Constantini S, Shir Y, Cotev S. Life-threatening torsade de pointes arrhythmia associated with head injury. Neurosurgery 1988; 23:89-92.
26. Adams JP. Non-Neurological Complications of Brain Injury In: Adams JP, Bell D, McKinlay (Eds). Neurocritical Care: A Guide to Practical Management. London; Springer 2010. pp. 77-87.
27. Thompson HJ, Tkacs NC, Saatman KE, Raghupathi R McIntosh TK. Hyperthermia following traumatic brain injury: a critical evaluation. Neurobiology of disease 2003;12:163-73.
28. Thompson HJ, Pinto-Martin J, MR Bullock Neurogenic fever after traumatic brain injury: an epidemiological study. J Neurol Neurosurg Psychiatry 2003;74:614-9.
29. Agha A, Sherlock M, Phillips J, Tormey W, Thompson CJ. The natural history of post-traumatic neurohypophysial dysfunction. European Journal of Endocrinology 2005;152 (3): 371-7.
30. Povlishock JT, Bullock MR (Eds). Guidelines for the management of Severe Traumatic Brain Injury, 3rd edn. Brain Trauma Foundation, New York Journal of Neurotrauma 2007; supplement-1: S1-S106.
31. Jeremitsky E, Omert L, Dunham CM, Protetch JBS, Rodriguez A. Harbingers of Poor Outcome the Day after Severe Brain Injury: Hypothermia, Hypoxia, and Hypoperfusion. J Trauma. 2003;54(2):312-9.
32. Chesnut RM. Avoidance of hypotension: condition sine qua non of successful severe head-injury management. J Trauma 1997;42:S4-S9.
33. Miller JD, Jones PA, Dearden NM, Tocher JL. Progress in the management of head injury. Br J Surg 1992; 79:60-4.
34. Scannell G, Waxman K, Tominaga G, Barker S, Annas C. Orotracheal Intubation in Trauma Patients With Cervical Fractures. Arch Surg 1993; 128(8):903-6.
35. Dutton RP, McCunn M, Grissom TE. Anesthesia for Trauma. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-

Kronish JP, Young WL (Eds). Miller's Anesthesia. 7th edn. Philadelphia: Churchill Livingstone; 2009; 2277-2311.
36. Shafer SL. Shock Values. Anesthesiology 2004; 101: 567-8.
37. Morris C, Perris A, Klein J Mahoney P. Anesthesia in hemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent? Anesthesia 2009; 64:532-9.
38. Drummond JC, Patel PM, Cole DJ, Kelly PJ. The Effect of the reduction of colloid reduction of oncotic pressure, with and without reduction of osmolality, on post-traumatic cerebral edema. Anesthesiology 1998; 88: 993-1002.
39. Vassar MJ, Perry CA, Holcroft JW. Prehospital resuscitation of hypotensive trauma patients with 7,5 percent NaCl versus 7,5 percent NaCl with added dextran : a controlled trial. J Trauma 1993; 34(5):622-33.
40. Cooper DJ, Myles PS, McDermott FT, Murray LJ, Laidlaw J, Cooper G, et al. Prehospital hypertonic saline resuscitation of patients with hypotension and severe traumatic brain injury: a randomized controlled trial. JAMA. 2004; 291(11):1350-7.
41. Guidelines for the management of severe traumatic injury. Cerebral perfusion Thresholds. Brain Trauma Foundation, American Association of Neurological surgeons, Congress of Neurological Surgeons. J Neurotrauma 2007; 24 (Suppl 1):9-S64.
42. Huynh T, Messer M, Sing RF, Miles W, Jacobs DG, Thomason MH. Positive end-expiratory pressure alters intracranial and cerebral perfusion pressure in severe traumatic brain injury. J Trauma 2002; 53:488-92.
43. Chesnut RM, Marshall LF, Klauber MR, Blunt BA, Baldwin N, Eisenberg HM, et al. The role of secondary brain injury in determining outcome from severe head injury. J Trauma 1993;34,216-22.
44. Ghajar J. Traumatic brain injury. Lancet 2000; 356:923–9.
45. Rudra A, Chatterjee S, Sengupta S, Wankhade R, Sirohia S, Das T. Fluid resuscitation in trauma. Indian J Crit Care Med 2006; 10: 241-9.
46. Arai T, Tsukahara I, Nitta K, Watanabe T. Effects of mannitol on cerebral circulation after transient complete cerebral ischemia in dogs. Crit Care Med 1986; 14: 634-7.
47. Wakai A, Roberts IG, Schierhout G. Mannitol for acute traumatic brain injury. Cochrane Database of Systematic Reviews, 2007 Issue 1. Art. No: CD001049. Accessed from: http://www.thecochranelibrary.com/Springboard WebApp/userfiles/ccoch/file/CD001049.pdf
48. Sandhu K, Murthy TVSP, Prabhakar T. Hypertonic saline in Traumatic Brain Injury: Current Status. Indian Journal of Neurotrauma 2004; 1:15-20.
49. Peterson B, Khanna S, Fisher B, MarshallL. Prolonged hypernatremia controls elevated intracranial pressure in head-injured pediatric patients. Crit Care Med 2000; 28: 1136-1143.
50. Cudd TA, Purinton S, Patel NC, Wood CE. Cardiovascular adrenocorticotropin and cortisol responses to hypertonic saline in euvolemic sheep are altered by prostaglandin synthetase inhibition. Shock 1998; 10: 32-6.
51. Vialet R, Albanese J, Thomachot L, Antonini F, Bourgouin A, Alliez B, et al. Isovolume hypertonic solutes (sodium chloride or mannitol) in the treatment of refractory post-traumatic intracranial hypertension: 2 ml/kg 7 percent saline is more effective than 2 ml/kg 20 percent mannitol. Crit Care Med 2003; 31: 1683-7.
52. Horn P, Meunch E, Vajkocry P, et al. Hypertonic saline solution for control of elevated intracranial pressure in patients with exhausted response to mannitol and barbiturates. Neurol Res 1999; 21: 758-64.
53. Sydenham E, Roberts I, Alderson P. Hypothermia for traumatic head injury. Cochrane Database of Systematic Reviews 2009, Issue 2. Art. No: CD001048. Accessed from: http://www2.cochrane.org/reviews/en/ab001048.html
54. Hutchison JS, Ward RE, Lacroix J. Hypothermia Therapy after Traumatic Brain Injury in Children. N Engl J Med 2008; 358:2447-56.
55. Edwards P, Arango M, Balica L, Cottingham R, El-Saved H, Farrel B, et al. CRASH Trial collaborators. Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. Lancet 2005;365:1957-9.
56. Ivascu FA, Howells GA, Junn FS, Bair HA, Bendick PJ, Janczyk RJ. Predictors of mortality in trauma patients with intracranial hemorrhage on preinjury aspirin or clopidogrel. J Trauma 2008 Oct;65(4):785-8.
57. Karni A, Holzman R, Bass T, Zorman G, Carter L, Rodriguez L, et al. Traumatic head injury in the anticoagulated elderly patient: A lethal combination. The American surgeon 2001; 67:1098-1100.
58. Cohen DB, Rinker C, Wilberger JE. Traumatic Brain Injury in Anticoagulated Patients. J Trauma 2006; 60:553-7.
59. Kiening Kl, Hartl R, Unterberg AW, Schneider GH, Bardt T, Lanksh WR. Brain tissue PO_2 monitoring in comatose patients: Implications for therapy. Neurol Res 1997; 19: 233-40.
60. Tisdall MM, Smith M. Cerebral microdialysis: research technique or clinical tool. Br J Anaesth 2006 ;97(1):18-25.
61. Meixensberger J, Kunze E, Barcsay E, Vaeth A, Roosen K. Clinical cerebral microdialysis: brain metabolism and brain tissue oxygenation after acute brain injury. Neurol Res 2001;23(8):801-6.
62. Thiagarajan A, Goverdhan PD, Chari P, Somasunderam K. The Effect of hyperventilation and hyperoxia on cerebral venous oxygen saturation in patients with traumatic brain injury. Anesth Analg 1998; 87:850-3.
63. Rudra A, Ray A, Chatterjee S, Bhattacharya C, Kirtania J, Kumar P, et al. Trauma in Pregnancy. Indian J Anaesth 2007;51:100-5.
64. Kuczkowski KM. Trauma in pregnancy: Perioperative anesthetic considerations for the head-injured pregnant trauma victim. Anaesthetist 2004; 53:180-1.

27 Anesthesia for Acute Spine Injury

Naina P Dalvi, Devangi A Parikh, Hemangi S Karnik

KEY POINTS

- The diagnosis of an unstable spinal injury and its subsequent management can be difficult and a missed spine injury can result in aggravation of injury leading to devastating long-term consequences.
- Spinal cord injuries most frequently involve the lower cervical spine and the thoracolumbar junction.
- The goal of care in the patient with SCI is to prevent secondary injury, thereby maintaining the viability of the neurological tissue in the spinal cord.
- All trauma patients should be placed in spinal precautions and presumed to have a spinal injury before they are evaluated and treated.
- Neck hyperextension and excessive axial traction must be avoided in suspected cervical spine instability. "Manual in-line stabilization"(MILS) of the head and neck by an assistant should be used to stabilize the cervical spine during laryngoscopy and tracheal intubation.
- The best immobilization method is to secure the patient to a hard board from the head to feet, place sandbags at either side of the head and put a rigid collar around the neck. This decreases movement to about 5 percent of normal.
- Respiratory distress can occur following SCI either due to concomitant thoracoabdominal trauma, or secondary to SCI itself.
- Acute spine injury leads to massive sympathetic discharge which causes intense peripheral vasoconstriction and systemic hypertension.
- Beyond the usual causes of hypotension and shock in trauma patients, the SCI patient may be complicated further by the presence of neurogenic shock.
- Methylprednisolone should be administered to all acute SCI patients if diagnosed within eight hours of injury.
- For stable patients with acute cervical injuries requiring cervical spine surgery, an awake fiberoptic intubation is ideal because it allows intubation to occur without disturbing spine alignment.
- Intraoperatively the goal is to maintain adequate spinal cord perfusion and oxygenation to prevent further damage.
- Intraoperatively blood pressure control is important, balancing the need to ensure spinal cord perfusion with the requirement to produce a bloodless surgical field.
- Postoperatively the patients may need ventilatory support in view of their unstable cardiovascular and respiratory status as well as concomitant facial edema.

INTRODUCTION

Spinal cord injury (SCI) is a common traumatic injury that requires hospitalization and surgical intervention. Care of the patient with an acutely injured spinal column with or without spinal cord involvement is challenging for all members of the trauma team. Anesthesiologists are often involved in the initial resuscitation and management of trauma victims with possible spine injuries. They have a role on site, during transport, during resuscitation, in operation theatre and in the intensive care unit. As a result, the anesthesiologist encounters many patients with SCI at various stages of their hospitalization, both in and out of the operating room.

The diagnosis of an unstable spinal injury and its subsequent management can be difficult and a missed

spine injury can result in aggravation of injury leading to devastating long-term consequences. Therefore, one should recognize the situations in which such injuries are likely, be familiar with evaluation of the spine, understand the pathophysiology of the spinal cord injuries and consider the risks and benefits of various approaches to their management. There are several aspects of their care, including airway management, initial resuscitation and intraoperative management, that require a particular understanding of their disease and for which the anesthesiologist is particularly suited.

EPIDEMIOLOGY

Vertebral column injury with or without neurological deficits must always be considered in patients with multiple injuries.

Anatomic Considerations

Approximate incidences of spinal injuries are as follows:[1]

- Cervical region 55 percent
- Thoracic region 15 percent
- Thoracolumbar region 15 percent
- Lumbosacral region 15 percent.

Mode of Injuries

The causes of spinal cord trauma in the adult population approximately are as follows:[2]

- Motor vehicle collisions (MVCs) 40 percent
- Falls 21 percent
- Violence 15 percent
- Sports related injuries 13 percent.

Etiologies for pediatric SCIs parallel those in adults, with water-related injuries accounting for 13 percent of the cases and sports-related injuries comprising 24 percent of cases.

In India the causes of spine injury differ significantly with railway accidents, fall from trees or construction sites being more common ones.

Age Distribution

Spine injury in India is usually seen in the third and fourth decade.[3]

MECHANISM OF SPINAL CORD INJURIES (FIG. 27.1)

Injuries to the spinal cord occur as a result of excessive forces applied to the spine. Injuries to the cervical spine can be due to:[4]

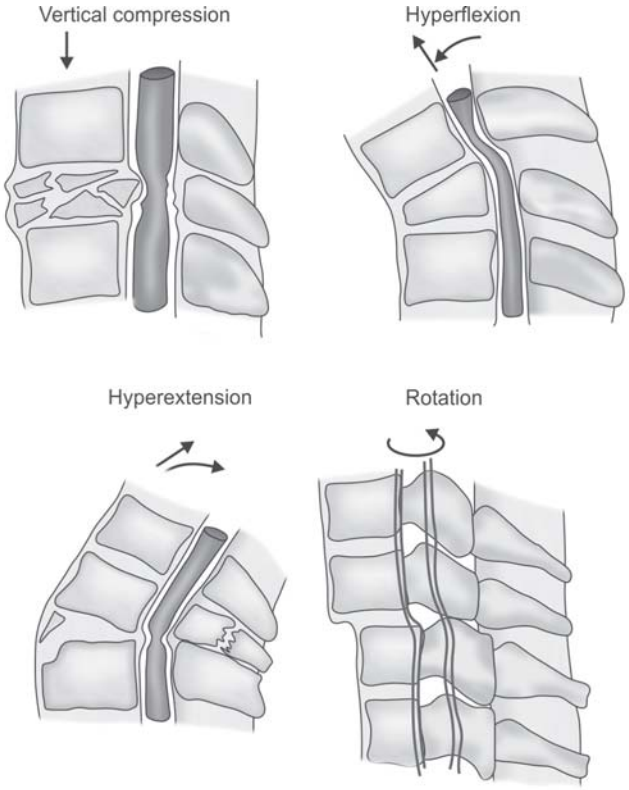


Fig. 27.1: Mechanisms of spinal cord injuries

Hyperflexion: Usually result from blows to the back of the head or forceful decelerations as might occur in MVC's. They are usually stable and rarely associated with neurological injury.

Hyperflexion-rotation: Disruption of the posterior ligamentous complex occurs and although cervical nerve root injury is common, the spine is stable and not usually associated with spinal cord damage.

Vertical compression or axial loading: Depending on the magnitude of the compression forces, the resulting injury ranges from loss of vertebral body height with relatively intact margins, to complete disruption of the vertebral body. Posterior displacement of comminuted fragments may result, producing cord injury. Despite cord injury, the spine is usually stable.

Hyperextension: Usually result from a blow to the anterior part of the head or from a whiplash injury. They are twice as common as flexion injuries and are more often associated with cord damage. Violent hyperextension with fracture of the pedicles of C2 and forward movement of C2 on C3 produces the "Hangman's fracture".

Extension-rotation: Seen in diving injuries. Because the anterior and posterior columns are disrupted, this injury is both unstable and associated with a high incidence of cord dysfunction.

Lateral flexion: Often associated with extension and flexion injuries.

The mechanism of injury of the thoracolumbar spinal cord is similar, except that there is less mobility; and injuries are more likely to be due to compression and rotational forces. The spinal canal is narrower in the thoracic segment relative to the width of the cord, so that when vertebral displacement occurs it is more likely to damage the cord. Until the age of 10 years, the spine has increased physiological mobility due to lax ligaments that affords some protection against acute spinal cord injury (ASCI). In contrast, elderly patients are at an increased risk due to osteophytes and narrowing of spinal canal. The three column concept of the structure of the vertebral column helps to clarify the mechanism of injury[5] (Fig. 27.2). The posterior column is formed by the posterior neural tract, spinous process, facetal articular processes and their corresponding posterior ligamentous complex. The middle column consists of the posterior one-third of the vertebral body and annulus fibrosus and the posterior longitudinal ligament. The anterior column comprises of the anterior longitudinal ligament and the anterior two-thirds of the vertebral body and annulus fibrosus. Flexion injury causes disruption of the posterior column, where as extension injury causes disruption of the anterior column. Acute spinal instability exists, if two or more columns are disrupted. This also predicts the possibility of late instability. Spinal cord injuries most frequently involve the lower cervical spine and the thoracolumbar junction; the former because of its mobility and the latter because it is the junction between the rigid thoracic spine and the flexible lumbar spine.[1]

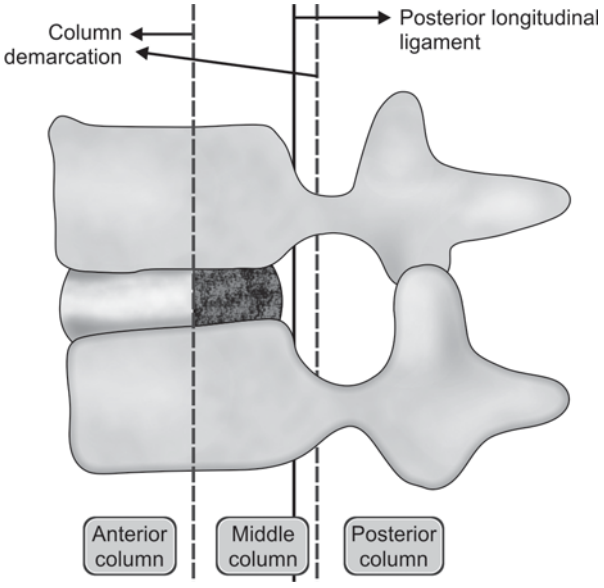


Fig. 27.2: Three column concept of the vertebral column

PATHOPHYSIOLOGY

Primary Injury

The primary injury[6] (mechanical injury) results from the original impact and compression against the spinal cord. This results in damage to the small intramedullary vessels causing hemorrhage in the central gray matter and perhaps vasospasm. The primary insult to the spinal cord results from fractures, severe ligamentous injury or spinal column dislocation (Fig. 27.3). A foreign body or bone fragment may directly damage the cord by producing lacerations, contusions, or compression. Burst fractures are more precarious than compression fractures because bony fragments retropulse into the spinal canal and may cause neurologic injury. Fracture dislocations, which involve all three support columns of the spine, are even more likely to cause complete cord injury than simple burst or compression fractures. The effects of the primary injury depend on the severity and the site. The cervical spine because of its increased mobility is commonly injured after trauma, falls and accidents. The primary injury cannot be treated and can only be prevented with educational programs aimed at reducing the incidence of ASCI.

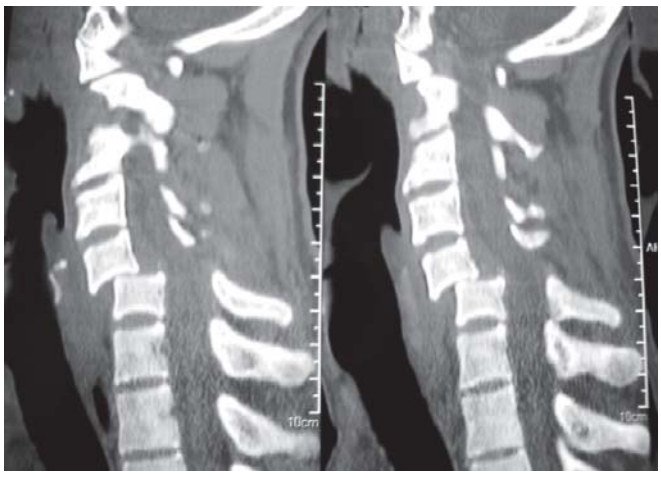


Fig. 27.3: CT scan showing spinal column dislocation

Secondary Injury

The reduction in spinal cord blood flow (SCBF) which may be associated with the primary injury leads to ischaemia of the cord. This triggers a biochemical cascade promoting a secondary injury, eventual infarction of the spinal cord and permanent loss of function. Secondary injury[7] occurs within minutes to hours following the primary injury and is thought to involve the release of a myriad of biochemical mediators including:

- Calcium accumulation intracellularly
- Potassium accumulation extracellularly
- Phospholipase A2
- Arachidonic acid metabolites
- Free oxyradicals
- Excitatory amino acids (glutamate, aspartate)
- Eicosanoid production
- Catecholamine accumulation.

The long-term results of these secondary events are loss of cord conduction and synaptic transmission, membrane damage and cytoskeleton disruption and eventual loss of structural integrity.

Other secondary injury mechanisms include: loss of autoregulation, vasospasm, thrombosis, edema, hypotension, reduced SCBF and loss of energy metabolism. The goal of care in the patient with SCI is to prevent secondary injury, thereby maintaining the viability of the neurological tissue in the spinal cord.

CLASSIFICATION

Spinal cord injuries can be classified according to:

1. Spinal cord syndromes[8] (Table 27.1).
2. Morphology.
   Spinal injuries can be described as:
   - Fractures
   - Fracture-dislocations
   - Spinal cord injuries without radiographic abnormalities (SCIWORA)
   - Penetrating injuries.

   Each can be further classified as stable or unstable.[1]
3. American Spinal Injury Association (ASIA) Classification.

   In an effort to categorize the nature of the injury, the American Spinal Injury Association (ASIA) classification was developed.[8] This system rates cord injuries by using an impairment scale (letters A through E) (Table 27.2).

ROLE OF ANESTHESIOLOGIST IN ASCI

SCI is often one of the several injuries in a patient with multiple trauma. The immediate role of anesthesiologist is in providing resuscitation and stabilization. Subsequently, the main goal of anesthesiologist involved in care of SCI is to prevent secondary damage to spinal cord by immobilizing the spine and airway management.

Management at the Scene of the Accident

The trauma patient's neck must be immobilized (Fig. 27.4) as soon as help arrives at the scene of the accident until complete evaluation shows that there is no injury. If the neck is not in the neutral position, an attempt should be made to achieve alignment. If the patient is awake and co-operative, they should actively move

Table 27.1: Spinal cord syndromes

Syndrome	*Description*
Central cord	Cervical lesion with upper— greater than lower-extremity paresis
Anterior cord	Anterior spinal artery disruption with loss of motor below lesion
Posterior cord	Rare, with loss of touch, vibration, and proprioception below lesion
Brown–Sèquard	Interruption of lateral half of cord, with loss of ipsilateral motor and touch, and loss of contralateral pain and temperature
Cauda equina	Compression of nerve roots below conus, with saddle anesthesia, urinary retention, and fecal incontinence

Table 27.2: American Spinal Injury Association (ASIA) Classification

ASIA A	Complete	No motor or sensory in sacral segments S4–S5
ASIA B	Incomplete	Sensory spared below lesion, including S4–S5
ASIA C	Incomplete	Motor score ≤3 for >50% of major muscle groups
ASIA D	Incomplete	Motor score >3 for >50% of major muscle groups
ASIA E	Intact	No motor or sensory deficit

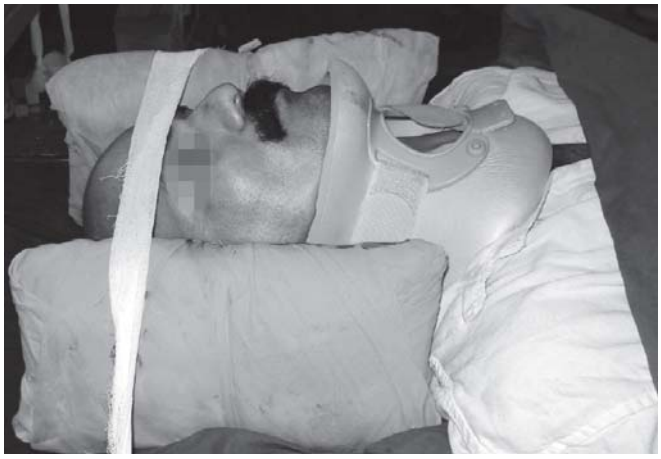


Fig. 27.4: Cervical spine immobilization with Philadelphia collar *(For color version, see Plate 3)*

their neck into line. If patient is unconscious or unable to co-operate then this is done passively. If there is any pain, neurological deterioration or resistance to movement, the procedure should be abandoned and the neck splinted in the current position. Soft collars are unsatisfactory for immobilization because they permit 75 percent of normal neck movement. Rigid collars, such as the Philadelphia and the extrication collars should be applied before extrication. These devices reduce flexion and extension to about 30 percent normal and rotation and lateral movement to about 50 percent. The best immobilization method is to secure the patient to a hard board from the head to feet, place sandbags at either side of the head and put a rigid collar around the neck. This decreases movement to about 5 percent of normal.[9] However, the application of definitive immobilisation devices should not take precedence over life-saving procedures. Thoracolumbar injury must also be assumed and treated by carefully straightening the trunk and correcting rotation. It is vital that the whole spine is maintained in the neutral position.

Management in Hospital

An anesthesiologist may first encounter the spine-injured patient during the initial resuscitation, in which case the primary survey may be ongoing. The primary survey of these patients, like all trauma patients, begins with the airway, breathing, circulation, disability, exposure, and environmental concerns (ABCDEs). If the patient has arrived from the scene of accident on a spine board then it should be removed as soon as possible once the patient is on a firm trolley. Prolonged use of spine boards can rapidly lead to pressure injuries. Full immobilization should be maintained.

Primary Survey

Airway: Airway may be compromised due to concomitant facial trauma, head trauma, or other injuries. Also, patients may need to be intubated before the presence or location of spinal cord injury can be confirmed. As a result, many acute trauma patients who require urgent or emergency endotracheal intubation are treated as if they have a cervical spine injury. The goal of intubation is to secure the airway with as little movement of the cervical spine as possible. The standard emergency intubating technique for an awake patient with a presumed or known cervical spine injury is a rapid sequence induction with cricoid pressure and manual inline stabilization. Precise cervical spine in-line immobilization should be maintained throughout the intubation maneuvers. This technique, also called manual in-line axial traction is an active process done by a second individual who is responsible for applying a varying amount of force to counteract the movements of the laryngoscopist, in an attempt to stabilize the cervical spine. The patient lies supine with the head in neutral position; an assistant applies manual in-line immobilization by grasping the mastoid processes, whereupon the front of a rigid collar can be removed safely; the collar can impede mouth opening, does not contribute significantly to neck stabilization during laryngoscopy, and will be an obstruction if surgical airway is required (Fig. 27.5). This technique of emergency airway management involves a minimum of three, but ideally four individuals: the first to preoxygenate and intubate, the second to apply cricoid pressure, the third to maintain manual in-line immobilization of the head and

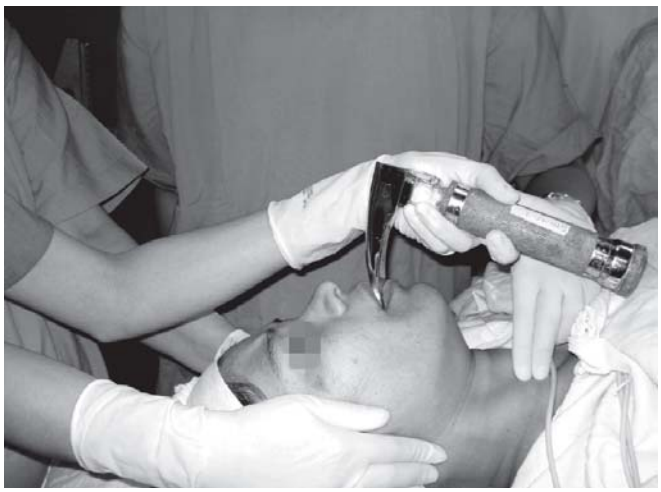


Fig. 27.5: Intubation with cricoid pressure and manual inline stabilization *(For color version, see Plate 3)*

neck and the fourth to give intravenous drugs and assist.[10] A gum elastic bougie is kept at hand and is used immediately if the view is obscured and/or intubation proves difficult.[11] In nonemergent cases, or in cooperative patients with unstable fractures, awake or asleep fiberoptic intubation may be appropriate. Succinylcholine is still the preferred drug for rapid sequence intubation. Succinylcholine can be safely used when given within 24 hours of spinal cord or head injury.[12] Tracheostomy should be considered when oral intubation fails in view of coexisting injuries or blood in the oral airway.

At our institute, most of the major trauma patients who require emergency tracheal intubation during early resuscitation are intubated in neutral position, with a trained assistant firmly immobilizing the head and neck, using direct laryngoscopy preferably with a McCoy blade. A gum elastic bougie is passed behind the epiglottis over which the tracheal tube is threaded. It is not necessary and often harmful to attempt to visualize full length of vocal cords in these patients. Intubating laryngeal mask airway involves less neck movements than regular laryngoscope and may be used. Newer rigid fiberoptic devices may help in visualization of vocal cords and can be used in emergency.

Breathing: Respiratory distress can occur following SCI either due to concomitant thoracoabdominal trauma, or secondary to SCI itself. If several days have elapsed since the injury (e.g., patients transported from remote areas), the victim may have suffered from aspiration pneumonitis during transport, especially with associated head trauma. These patients often require ventilatory support. Breathing adequacy in SCI patient may be compromised in acute cervical or high thoracic SCI. In these patients the chest paradoxically collapses (from the loss of intercostal muscle innervation below the injury level) as the abdomen expands upon inspiration from contraction of the diaphragm. Consequently, the vital capacity, functional residual capacity (FRC) and also the ability to cough or clear secretions[13] are reduced. Therefore these patients need to be put on ventilatory support early as they may deteriorate with time. In cases with high cervical injury, diaphragm may be paralysed leading to apnea or severe respiratory insufficiency, necessitating urgent intubation and mechanical ventilation. The patient's pulmonary status may also be compromised by neurogenic pulmonary edema. Acute spine injury leads to massive sympathetic discharge which causes intense peripheral vasoconstriction and systemic hypertension, forcing blood in the low resistance pulmonary circulation. The resultant increase in pulmonary vascular pressures increases the permeability of pulmonary capillaries and can cause pulmonary hemorrhage and persistent edema even after the sympathetic discharge resolves.[14] Although the catecholamine release may resolve in only a few minutes, the pulmonary edema may persist for 24 to 48 hours, and can exacerbate other pulmonary injuries. Neurogenic pulmonary edema is managed by supportive care, mechanical ventilation with PEEP and fluid restriction.

Circulation: Circulation may be impaired due to spinal shock, concomitant thoracoabdominal trauma or other injuries. Neurogenic (spinal) shock is characterized by bradycardia, systemic vasodilatation, and hypotension and results from the loss of sympathetic innervation to the cardiac sympathetic plexus due to the high cervicothoracic spinal cord injury. Neurogenic shock is associated with complete injuries at the T6 level and above.[15] Extreme bradycardia and even cardiac arrest can occur in the presence of the unopposed parasympathetic tone. Approximately two-thirds of cervical spine injury patients with a systolic blood pressure (SBP) <100 mm Hg will have neurogenic shock.[16] Treatment for both forms of shock begins with volume resuscitation. If active hemorrhage is not detected or suspected, persistent hypotension despite the administration of 2L or more of fluid should raise the suspicion of neurogenic shock. Patients with hypovolemic shock usually have tachycardia, whereas those of neurogenic shock classically have bradycardia. If the blood pressure does not improve after a fluid challenge, the judicious use of vasopressors may be indicated. Use of phenylephrine hydrochloride, dopamine or norepinephrine is recommended. Use of vagolytics such as atropine and glycopyrrolate may be considered.When the fluid status is uncertain, invasive monitoring for central venous pressure and arterial blood pressure is helpful.

Disability: A thorough neurological examination should be performed for all patients suspected of spine or cord injury. Minimally, a Glasgow Coma Scale score, pupillary examination, sensory examination and presence of paralysis or paresis should be documented.

Exposure and Environmental Protection: Exposure, the first "E" means all body surfaces must be exposed so that they can be examined for missed injuries. Environmental is the second "E" and means beware of environmental exposure, which could cause hypothermia or hyperthermia. This is particularly important in the SCI

patient because of the thermoregulatory dysfunction that occurs. After thorough assessment the patient should be covered properly to prevent further hypothermia.

Secondary Survey

This mainly focuses on a detailed neurological assessment. The following steps are followed:

Step 1: Obtain a detailed history of the mechanism of injury, medical history, and also drugs given prior to patients arrival, during assessment and management phases.

Step 2: Reassess level of consciousness and pupils.

Step 3: Reassess GCS score.

Step 4: Assess the spine.

The entire spine should be palpated by carefully log rolling the patient. Logrolling is discussed later in the chapter. The spine is examined for deformity, swelling, crepitus, pain, contusion, laceration. Sensation to pin prick in all dermatomes is tested and the most caudal dermatome where pinprick is felt is recorded. Similarly motor function is also assessed. Deep tendon reflexes are measured.

All physicians caring for the patient should verify the findings of their own examinations. The examination results should be documented in the anaesthetic record.

Step 5: Revaluate—assess for associated or occult injuries.

CLINICAL CLEARANCE OR RADIOLOGICAL DIAGNOSIS OF SPINE INJURY

The diagnosis of spinal cord injury is of paramount importance in the acute setting. Clinically cervical spine can be clinically cleared without radiography in patients with a GCS of 15, with no evidence of drug or alcohol abuse, normal neurological examination, no midline cervical pain and absence of distracting or significant injuries. (another injury which may 'distract' the patient from complaining about a possible spinal injury).[17]

In all the remaining patients who don't meet above criteria, radiological evaluation is indicated. Lateral, anteroposterior (AP) and open mouth odontoid views of the cervical spine should be obtained. On the lateral view, the base of the skull, all seven cervical vertebrae, and the first thoracic vertebra must be visualized. The patient's shoulders may need to be pulled down when obtaining lateral cervical spine x-ray film, to avoid missing fractures or fracture-dislocations in the lower cervical spine. If all seven cervical vertebrae are not visualized on the lateral x-ray film, a swimmer's view (raising the arm closest to the film over the head and depressing the opposite arm) of the lower cervical and upper thoracic vertebrae should be obtained. The open mouth odontoid view should include the entire odontoid process and bilateral C1 and C2 articulations. Axial CT scans should be obtained at 3 mm intervals if suspicious areas are not adequately visualized on plain films.

Approximately 10 percent of patients with a cervical spine fracture have a second, non contiguous vertebral column fracture. This warrants a complete X-ray screening of the entire spine in patients with cervical spine fracture. Such screening is also advised in all comatose trauma patients. The indications for screening radiography of the thoracic and lumbar spine are same as that of cervical spine.

In the presence of neurological deficits, magnetic resonance imaging (MRI) is recommended to detect any soft tissue compressive lesion, spinal cord contusions or disruptions, and paraspinal ligamentous or soft tissue injury.[1]

The North American Emergency X-Ray Usage (NEXUS) database however has shown that screening radiography using three cervical views (anteroposterior, lateral and odontoid views) can identify only 61 percent of the injuries.[18] Computed tomography with 3 mm slices using helical scanning and multiplanar reconstruction has been shown to have a much higher sensitivity of 97 to 100 percent. Spinal cord injury may occur without any radiological abnormality in 2.8 to 3.8 percent of all spinal injuries.[18,19] MRI is the investigation of choice to detect this condition. (Figs 27.6 and 27.7)

GENERAL MANAGEMENT

The goal of treatment of spinal cord injuries is:

i. To protect the spinal cord from further damage.
ii. To maintain alignment of the bony structures to allow maximal recovery in incomplete lesions.
iii. To achieve stability of the bony column to allow rehabilitation.

Resuscitation of the Spinal Cord

It is now known that though the initial primary or mechanical injury is irreparable, the spinal cord undergoes additional biochemical and pathological injury, and this secondary injury is amenable to treatment. Anesthesiologists are often involved in the initial resuscitation of patients with ASCI and therefore

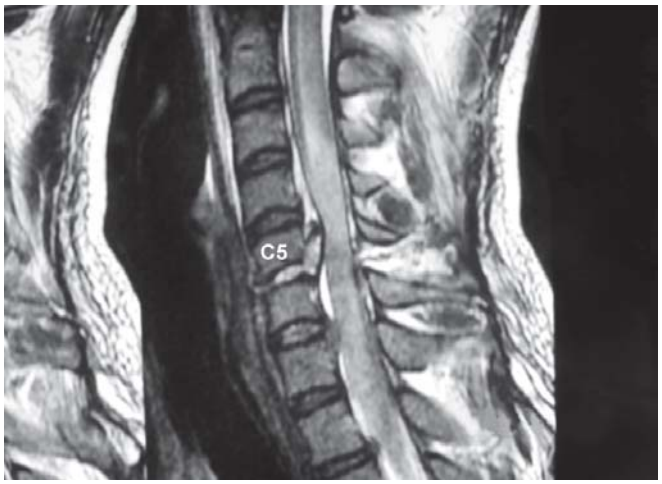


Fig. 27.6: MRI showing cord compression in a case of C5 to C6 dislocation

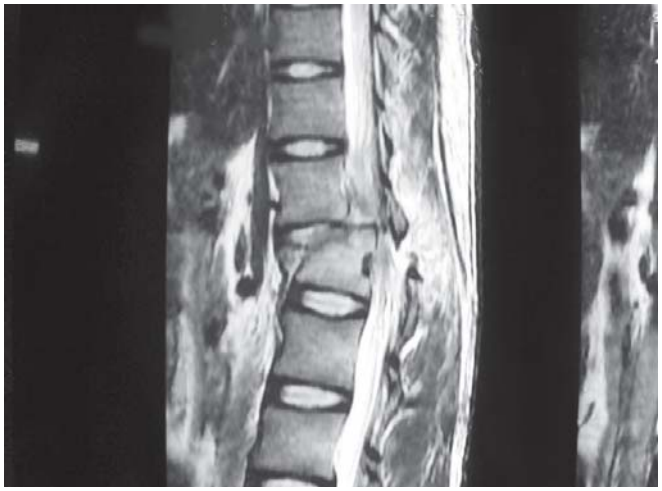


Fig. 27.7: MRI showing severe spinal cord damage at D12- L1 fracture dislocation

are in an ideal position to influence the degree of functional recovery that may take place. General management of ASCI includes immobilization, intravenous fluids, medications and transfer, if appropriate.

Immobilization

Any patient with a suspected spine injury should be immobilized above and below the suspected injury site until a fracture is excluded by x-ray examination. Spinal protection should always be maintained until a cervical spine injury is excluded. Technique of immobilization has been discussed above. Of special concern is the maintenance of adequate immobilization of restless, agitated and violent patients. This condition could be due to pain, confusion associated with hypoxia or hypotension, alcohol or drug abuse or a personality disorder. The cause should be searched for and corrected if possible. If necessary a sedative or paralytic agent may be administered.

Logrolling

Once the patient arrives in the emergency department, every effort should be made to remove the spine board as early as possible. Paralyzed patients should be removed from the long spine board as soon as possible (i.e. within 2 hours) as they are at particular risk for pressure points injuries and decubitus ulcers. Removal of the board is done as a part of the secondary survey as the patient is logrolled for inspection. Four people are needed to perform the modified logrolling procedure and to immobilize the patient on a long spine board—one person to maintain manual in line immobilization of the head and neck; one for the torso; one for the pelvis and the legs; one to direct the procedure and move the spine board. This procedure maintains the entire patient's body in neutral alignment, thereby minimizing any untoward movement of the spine. This procedure assumes that any extremity suspected of being fractured has already been immobilized.[1]

Intravenous Fluids

Traumatic ASCI may cause a decrease in local SCBF and loss of autoregulation leading to ischemia and tissue hypoxia. Superimposed on this background, systemic arterial hypoxemia and hypotension are not well tolerated by injured neural tissue and may lead to further secondary damage. Hence, the first and probably most important step in resuscitating the injured spinal cord is to correct hypotension and hypoxemia. A urinary catheter should be inserted to monitor urinary output and relieve bladder distension. A gastric catheter should also be inserted in all patients with paraplegia and quadriplegia to prevent gastric distension and aspiration.

Medications

Methylprednisolone is a synthetic steroid with anti-inflammatory effects at the site of injury, decreasing edema and the extravasations of leukocytes through the capillary endothelium within the injury zone. Methylprednisolone is administered to all acute SCI patients, if diagnosed within eight hours of injury. However, the data supporting its use is still controversial. Its use was precipitated by the NASCIS (National Spinal Cord

Injury Study) II and III studies from the 1990s, which showed clinically important improvement in the motor function of patients with SCI.[20,21] The results of the studies confirming benefit of methylprednisolone have been criticized[22] however, and some centers do not regard steroids as standard of care. There are numerous reasons why avoidance of systemic steroids might be desirable if they are not indicated, such as their association with hyperglycemia, infection and polyneuropathy of critical illness. In addition, because the SCI patient population has a high incidence of comorbid head injury, it is worthwhile to consider a large randomized study of methylprednisolone therapy in patients with head injury, which found an increased risk of death in patients receiving methylprednisolone both at two weeks[23] and at six months.[24] Despite the continued controversy, the accepted protocol at ours and many other institutions is in accordance with the NASCIS II trial, which is to administer Methylprednisolone 30 mg/kg bolus over 15 minutes and a 5.4 mg/kg/hr continuous infusion for 24 hours if treatment is initiated within three to six hours of injury. If started within six to eight hours of injury, the infusion is continued for 48 hours.

In addition to steroids, GM-1 gangliosides have also been found useful in the treatment of patients with SCIs.[25] Oxygen free radical scavengers are also being investigated for the treatment of patients with SCIs.

Transfer

Patients with spine fractures or neurological deficits should be transferred to a definitive care facility preferably a tertiary care trauma center. Unnecessary delay should be avoided. The spine should be immobilized and protected for the transfer. Split-scoop stretchers and vacuum mattresses are more appropriate for transfer than rigid spinal (rescue) boards, which should be reserved for primary extrication from vehicles, rather than as devices for transporting patients.

SURGICAL INTERVENTION

Surgical indications for treatment during the acute phase include:

i. Decompression with or without fusion in a neurologically deteriorating patient. This includes complete as well as incomplete lesions.
ii. Reduction and stabilization, when conservative management fails to achieve these objectives.
iii. Surgical treatment for other life-threatening conditions, unrelated to the cord injury.

Decompression is usually accomplished by the anterior approach in cervical spine injuries. It can involve single-level diskectomies to multiple-level corpectomies. Rarely does the injury pattern necessitate a posterior approach. Thoracic injuries usually do not require decompression owing to the stiff nature of the spine and if necessary it can be done effectively by a transpedicular approach posteriorly. Decompression of the thoracolumbar (T10–L2) spine remains controversial among providers. Most providers feel that the anterior approach affords a more thorough decompression. Surgery usually involves a retroperitoneal approach. The anterior procedure is often augmented by posterior stabilization. If the neurologic injury is purely radicular, this can be effectively decompressed from a standard midline posterior approach.[8]

Stabilization in cervical spine injuries can involve diskectomy and/or corpectomy with bone graft reconstruction and fusion with or without hardware. Stabilization procedures for the thoracic and lumbar spines usually follow similar principles but are performed through a posterior approach.

Timing of Surgical Intervention

Even more controversial than steroid administration is the timing of surgery in spinal cord injuries. Management of the patient with SCI frequently requires intervention for comorbid life-threatening trauma, therefore surgical decompression of the spinal cord with fixation of the spinal column must wait until the patient is clinically appropriate for the procedure. Early fixation (within 24 hours) is probably most appropriate for cervical spine injuries with quadriplegia.[26,27] Surgeons feel that decompression of the neurologic elements can benefit patients with traumatic injuries. The optimal time of the decompressive surgery remains controversial. Incomplete injuries are usually decompressed in an emergent or urgent manner. The prognosis of complete injuries remains poor and urgent decompression does not appear to affect functional recovery[8]. Current clinical studies have provided conflicting data, some showing no benefit from early surgery[28-30] and others showing positive effect.[28-30,31-33]

INTRAOPERATIVE MANAGEMENT

As discussed above, operative management of cervical and thoracic spine injury is seldom emergent. However, these patients may have emergent needs for other procedures (e.g., exploratory laparotomy and emergency thoracotomy). Whatever may be the nature of surgery, securing the airway is the most crucial step during the anaesthetic management of a patient with cervical spine

Table 27.3: Effect of basic airway maneuvers on cervical spine mobility

Airway Maneuver	*Effect*	*Comments*
Chin Lift and Jaw Thrust[9]	↑ 5 mm distance space at C5-C6 in cadaver	Collar does not prevent this widening.
Anterior neck pressure (for nasotracheal intubation)	Posterior subluxation > 5 mm	Contraindicated if cervical spine trauma
Head tilt	May increase displacement	Contraindicated if cervical spine trauma
Insertion of airway	No significant displacement	Safe in cervical spine trauma
Cricoid pressure[34]	Negligible spine movement	Safe in cervical spine trauma

injury. The urgency of airway intervention is the most important factor in planning airway management for patients with potential C-spine injuries. Other considerations include the assessment of the risk of cord injury with head and neck movement, the airway anatomy, the patient's degree of cooperation, and the anaesthesiologist's expertize.

Anesthesia Induction and Airway Management

The ultimate goal is to establish tracheal intubation without causing further injury to the spinal cord. The ideal induction agent probably does not exist, and it is down to operator experience. Propofol is to be used with caution because of the potential for hypotension. Ketamine is a very cautiously used induction agent which maintains cardiovascular stability better than the other intravenous agents. As a noncompetitive NMDA receptor antagonist, it has neuroprotective effects. Its use is currently contraindicated in patients at risk from raised intracranial pressure (ICP) as it has been shown to increase cerebral blood flow and so ICP in head injured patients. However, evidence is accumulating that this may not be the case, especially in hypotensive patients, and its effects on ICP may be modulated by agents such as propofol. Orotracheal intubation under thiopentone and suxamethonium combination is recommended in most centers. If this technique is used, hyperkalemic response to suxamethonium should be anticipated from 48 hours onwards post injury. Prior to induction, the cervical spine stability needs to be documented along with the neurological status. The clearance of the cervical spine both clinically and radiologically has been discussed.

The anesthesiologist should be aware of the effect of basic airway maneuvers on cervical spine mobility (Table 27.3). The anesthetist may also face problems during intubation due to the spine immobilisation techniques employed in the emergency resusitation room. Immobilization of neck with collars, straps and sand bags may restrict the mouth opening and cause a poor laryngoscopic view (grade 3 and 4) in 64 percent of the patients.[35]

Techniques of Securing Airway in Patients with Cervical Spine Injury

Direct Laryngoscopic Intubation

Direct laryngoscopic oral tracheal intubation with in-line stabilization, either performed after induction of general anaesthesia or with the patient awake, remains an excellent option for elective airway management in patients with cervical spine injuries.[36] During normal direct laryngoscopy and oral intubation, significant extension occurs between occipital bone and C1 and also between C-1 and C-2.[37,38] Manual inline neck stabilization reduces this head extension by 50 percent in anaesthetized patients.[39] However, in a cadaver study of injuries at C4, this type of stabilization did not reduce the movement, suggesting the limitation of this maneuver in preventing movement of the spine in patients with cervical spine injury.[37] Axial traction on spine should be avoided during laryngoscopy and intubation as this could increase the spinal cord injury. Gum elastic bougie is an important adjunct to avoid displacement of the fractured spine during direct laryngoscopic intubation.[40]

Influence of the type of laryngoscope on cervical movement: The cervical spine movement caused by McIntosh curved blade or Miller's straight blade are not significantly different during direct laryngoscopic intubation.[41] In a comparison of McIntosh and McCoy laryngoscopes, McCoy laryngoscope improved visualization of the larynx by at least one grade in 49 percent of cases.[42] In another study, Miller and McIntosh blades were compared with Bullard laryngoscope.[43] Head extension and neck movements were less and laryngeal visualization was better with Bullard laryngoscope. However, there were problems associated with Bullard laryngoscope, which included prolonged time for intubation, fogging, and occasional inability to pass the tracheal tube through the glottis. Angulated video

intubating laryngoscope significantly improved the laryngeal view compared with direct laryngoscopy with cricoid pressure.[44]

Awake Intubation

In a cooperative patient who does not require emergency intubation, awake intubation has been recommended. The fibreoptic technique allows intubation under direct vision with minimal neck movement. The technique requires a sufficiently skilled anaesthesiologist, a cooperative patient, secretion and blood free airway and adequate topical anaesthesia. Coughing and bucking will result in failure and might threaten an unstable spine. The nasal route should not be used in cases of suspected basal skull fracture. In cadavers with a C5-C6 instability, blind nasal intubation caused least cervical spine movements.[45] With C1-C2 instability both oral and nasal intubations produced similar cervical spine movement.[46] In cadavers with C3 injury, awake fibreoptic technique produced no movement of unstable segments as assessed by video fluoroscopy.[47]

Laryngeal Mask Airway (LMA)

Intubating LMA has been used successfully for intubation in patients undergoing cervical spine surgery.[48] It has also been used in conjunction with rapid sequence intubation and also for awake oral intubation with fibreoptic bronchoscope.[49] Both standard LMA and intubating LMA have been shown to cause a temporary pressure of 250 cm H_2O against the posterior pharyngeal wall during insertion. The pressure is sufficient to cause up to 2 mm of displacement of C3.[50] The cervical spine movement that occurs during insertion of LMA and intubation through LMA is less than that produced during direct laryngoscopy.[51]

Surgical Airway

Cricothyroidotomy is usually performed when all other attempts to secure the airway have failed. Tracheostomy is technically difficult without neck extension and is avoided due to proximity of operative field and risk of contamination when other options are possible.

PLAN FOR AIRWAY MANAGEMENT IN A PATIENT WITH CERVICAL SPINE INJURY

No single technique of airway management has been shown to be superior to others. Therefore, the fear of inflicting cord damage should not prevent securing the airway with the technique that the operator is conversant with.

A practical approach for airway management in a patient with suspected cervical spine injury is shown in the Figure 27.8.

Anesthesia Maintenance

The main goal here is to maintain adequate spinal cord perfusion to prevent further damage. In the acute stage, this consideration applies equally to complete and incomplete lesions, since in both instances deterioration as well as improvement can occur. Because of loss of autoregulation, spinal cord perfusion becomes dependent on systemic perfusion, and systemic hypotension may cause further secondary injury. On the other hand, hypertension may lead to hemorrhage and edema formation. Any standard maintenance regimen is acceptable. However, blood pressure control is important, balancing the need to ensure spinal cord perfusion with the requirement to produce a bloodless surgical field. Maintenance of normal systemic perfusion and gas exchange are the prime objectives. Hyperventilation may decrease perfusion and cause ischemia, therefore normocapnia is recommended. No single anesthetic technique has been shown to be superior. All anesthetic drugs that decrease metabolism appear to have a protective effect. All drugs should be given slowly by titration because of the cardiovascular lability. Bradycardia is a frequent occurrence in patients with cervical cord injuries and atropine is the drug of choice. It is recommended that hypotension should be treated with fluid and inotropic agents rather than direct vasoconstrictors. Usually in elective spine surgeries, induced hypotension is used for a good surgical field but in the emergency setting this may not be feasible. Paralytic ileus is relatively common after ASCI and may last for 2 to 3 days. The patient is at risk of gastric aspiration of stomach contents and a distended abdomen can impede respiration. Gastric distension and paralytic ileus require the insertion of a nasogastric tube. An orogastric is used if there is associated basal skull fracture. Acute retention of urine can develop in paraplegic and tetraplegic patients unless the sacral segments are spared. In the absence of urethral trauma a smaller size Foley's catheter is passed under aseptic conditions and taped to the anterior abdominal wall to prevent any unnecessary movement of and injury to the urethra. The urine output should be measured and maintained at greater than 0.5 ml/kg/hour. Thermoregulation is also affected in patients with spinal cord injury. Core temperature should be monitored and warming devices used to prevent heat loss. Intravenous fluids should be warmed. Experimental evidence suggests that hyperglycemia aggravates ischemic injury and dextrose containing infusions should

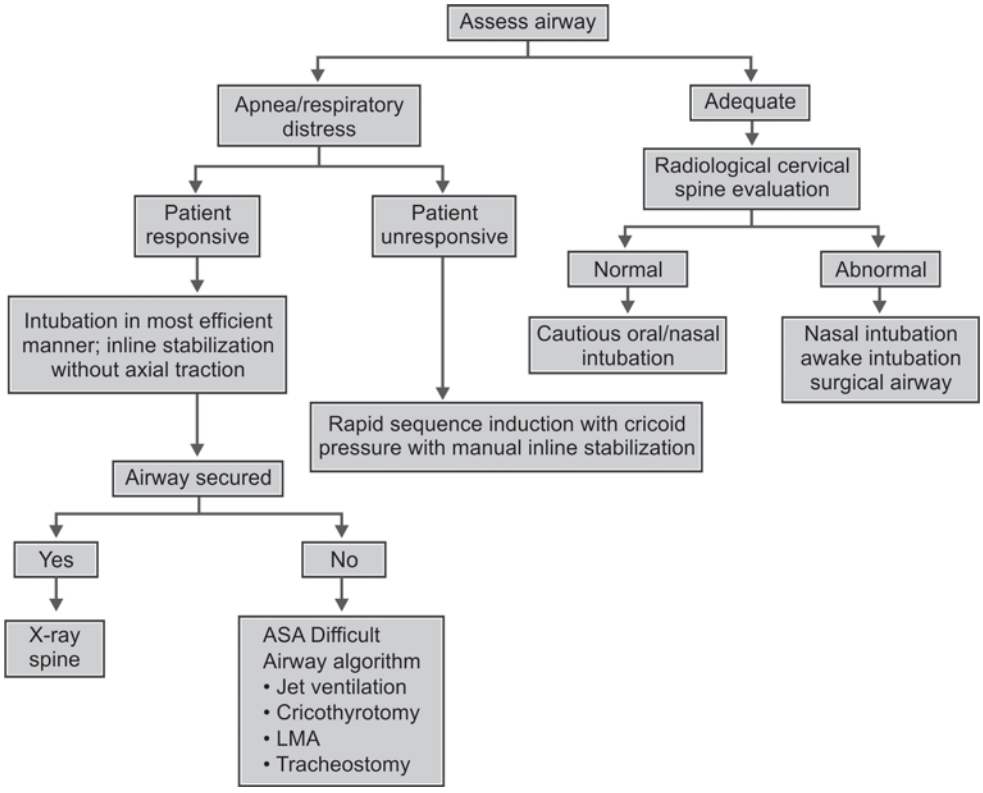


Fig. 27.8: Algorithm for airway management

therefore be avoided. Special precautions should be taken during patient positioning. Pressure points should be padded well. In prone position, care should be taken not to twist the neck while turning the patient, to protect the eyes from compression, proper positioning of the head to avoid jugular venous obstruction, and to avoid abdominal compression. Traction using skull calipers or manual traction may be needed during the fixation.

Handling of spinal cord during the surgical procedure, additional hypotensive or hypoxic insults and blood loss can make the patient prone for neurological deterioration in the immediate postoperative phase. In high cervical spine procedures, there are chances of airway edema or worsening of spontaneous respiratory efforts. Therefore, extubation may be problematic and is best performed when the patient is awaken and able to support their own airway. If the risk of reintubation is high, a tracheal tube exchange catheter (e.g. Cook catheter) may be useful. The catheter can be introduced into the tracheal tube and left *in situ* when the patient is extubated. If urgent reintubation is necessary, the new tracheal tube can be rapidly railroaded over the exchange catheter. However, most patients with acute cord injuries have unstable cardiovascular and respiratory status as well as concomitant facial edema hence their tracheas cannot be extubated at the end of the surgical procedure. Instead they should be transferred directly to an intensive care unit for further care. An intubated patient can be allowed to awaken to perform a neurologic examination and then sedation can be restarted. Sedation overnight can be accomplished with a propofol infusion and extubation can be planned in the next 24 to 72 hours after swelling has subsided with diuresis and head-up positioning. Extubation can be considered if patient is awake, having adequate respiratory attempts with good chest excursions, arterial blood gases showing no CO_2 retention and adequate oxygenation, no hemodynamic instability. When in doubt it is better to leave the tube in situ to allow the cord edema to settle down and extubate a few hours later.

Monitoring

In addition to the standard monitoring which includes pulse oximetry, electrocardiography, noninvasive blood pressure monitoring, capnometry, in acute quadriplegic patients, direct intra-arterial pressure monitoring and the insertion of a central venous catheter is advised

because of cardiovascular instability and an unpredictable response to fluid challenge and the risk of pulmonary edema. For central venous access and pressure monitoring, the subclavian vein is preferred over internal jugular vein because placement does not require the neck to be turned. In addition a good peripheral access is mandatory for blood loss replacement as central veins may be inaccessible in the prone position.

Intraoperative Monitoring of Spinal Cord Function

The aim of surgery in spinal cord injury is to stabilize the spine as well as prevent further damage to the cord. However the surgery itself has potential for causing further injury. Despite apparently optimal surgical and anesthetic management, complications still occur during spine surgery which explains the use of intraoperative monitoring of spinal cord function. The modalities for this purpose are:

Wake up Test

This test involves discontinuation of anesthesia during specific time of surgery, waking up and asking the patient to make certain movements of limbs. The ability of the patient to perform the test ensures the surgeons about the integrity of the motor pathway. The main advantage of this test is that it assesses anterior spinal cord (i.e. motor) function. The disadvantage is that it is difficult to perform, can be performed at one time point only, and cannot be performed in children or in unconscious patients. The risks involved are distress to the patient, dislodging of monitoring equipment, intravenous catheters or spinal instrumentation, accidental extubation and air embolism as negative pressures are generated during spontaneous ventilation. It is the anesthesiologists' duty to provide a faster, smoother, pain free awakening.[52] The patient needs to be explained about the procedure prior to anesthesia. Surgical anesthesia is typically provided with a balanced technique of a volatile drug (e.g. sevoflurane) and opioids along with shorter acting neuromuscular blocking agents with or without nitrous oxide. The opioids are important to provide analgesia while the patient is awake and to permit the patient to tolerate the endotracheal tube.[53]

Somatosensory and Motor Evoked Potentials

It is the most common type of intraoperative monitoring during spine surgery. In patients who have suffered vertebral column injuries without neurological deficit, spinal cord monitoring may be required. However, its use in the emergency setting may not be feasible. Although the interpretation of SSEP remains imprecise, still its use is gaining popularity. The major limitation at present is that it monitors only dorsal column function and, theoretically, motor paralysis can occur despite unchanged SSEP signals. In patients with incomplete lesions and abnormal preoperative SSEP interpretation of intraoperative assessment is impossible. SSEP signal is susceptible to anaesthetic influence and inhaled anaesthetics in high doses can abolish these responses. The recommended anesthetic regimen for intraoperative monitoring of SSEP is continuous intravenous infusion of a narcotic (fentanyl or sufentanil) supplemented with low-dose inhaled anesthetic (isoflurane or halothane) or with nitrous oxide. The combination of nitrous oxide and an inhaled anaesthetic should be avoided. In cases wherein epidural electrode can be placed, the choice of anesthetic becomes a minor matter, since epidural recordings are more resistant to anesthetic influence than cortical recordings. Recent advances have made it possible to record motor-evoked potentials using either electrical or magnetic stimulation of the motor cortex. This complements SSEP and allows simultaneous monitoring of both anterior and posterior column functions.[2]

COMPLICATIONS IN SPINE SURGERY

Exacerbation of spinal cord injury and massive blood loss in emergency setting are known complications. Intraoperatively complications due to prone position should be avoided. They include impaired ventilation due to abdominal compression, venous obstruction due to improper positioning of the head and neck, optic neuropathy due to compression of the eyes, postoperative blindness due to anterior spinal artery syndrome and corneal abrasion if eyes are not taped properly. Postoperative complications include the risk of respiratory failure increased by thoracotomy, diaphragmatic injury and fat embolism. The risk of infections is also very high as these patients are on steroids, hence antibiotic cover post operatively is a must. Anterior cervical procedures lasting more than five hours, exposing three or more levels involving C2, C3, and C4 and 300 ml blood loss are at higher risk for airway compromise postoperatively.[54] Great care should be exercised when moving and transferring patients to prevent dislodgement of spinal fixation. Careful documentation of neurological status is important because postoperative neurological deterioration is a major concern.

REFERENCES

1. Advanced Trauma Life Support for doctors. Student Course manual. 8th edn. Chicago, American college of Surgeons 2008.
2. Nieman CC, Dhawan R, Wilson WC. Cervical and Thoracic Vertebral Spine Trauma. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma: Emergency Resuscitation, Perioperative Anesthesia, Surgical management. Volume I. New York: Informa Healthcare; 2007; pp.501-15.
3. Singh R, Sharma SC, Mittal R, Sharma A. Traumatic Spinal Cord Injuries In: Haryana: An Epidemiological Study. Indian Journal of Community Medicine 2003;28(4):184-6.
4. Crosby ET, Lui A. The adult cervical spine: implication for airway management. Can J Anaesth 1990;37:77-93.
5. Denis F. Spinal instability as defined by the three-column spine concept in acute spinal trauma. Clin Orthop Relat Res 1984;189:65–76.
6. Tator CH. Acute management of spinal cord injury. Br J Surg 1990;77:485-6.
7. Tator CH, Fehlings MG. Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. J Neurosurg 1991;75:15-26.
8. Kincaid MS, Lam AM. Anesthesia for spinal cord trauma. In: Smith CE (Ed). Trauma Anesthesia. New York: Cambridge University Press; 2008; pp.213-24
9. Aprahamian C, Thompson BM, Finger WA, Darinz JC. Experimental cervical spine injury model; evaluation of airway management and splinting techniques. Ann Emerg Med 1984;13:584-7.
10. Criswell JC, Parr MJA, Nolan JP. Emergency airway management in patients with cervical spine injuries, Anaesthesia 1994;49:900-3.
11. Nolan JP, Wilson ME. Orotracheal intubation in patients with potential cervical spine injuries. An indication for the gum elastic bougie. Anaesthesia 1993;48:630-3.
12. John DA, Tobey RE, Homer LD, Rice CL. Onset of succinylcholine induced hyperkalemia following denervation. Anesthesiology 1975;43:89–99.
13. Cardozo CP. Respiratory Complications of Spinal Cord Injury. Journal of Spinal Cord Medicine 2007(30);4:307-8.
14. Theodore J, Robin ED. Pathogenesis of neurogenic pulmonary oedema. Lancet 1975;2:749–51.
15. Chiles BW, Cooper PR. Acute spinal injury. N Eng J Med 1996;334:514–20.
16. Soderstrom CA, McArdle DQ, Ducker TB, Militello PR. The diagnosis of intra-abdominal injury in patients with cervical cord trauma. J Trauma 1983;23:1061–5.
17. Jankowitz BT, Welch WC, Donaldson WF. Injuries to the spinal cord and spinal column. In: Peitzman AB, Rhodes M, Schwab CW, Yealy DM, Fabian TC, (Eds). Trauma manual: Trauma and acute care surgery. 3rd edn. Philadelphia: Lippincott Williams & Wilkins; 2008; pp.145-64.
18. Mower WR, Hoffman JR, Pollack CV, Zucker MI, Browne BJ, Wolfson AB. NEXUS Group. Use of plain radiography to screen for cervical spine injuries. Ann Emerg Med 2001; 38:1-7.
19. Demetriades D, Charalambides K, Chahwan S, et al. Nonskeletal cervical spine injuries: epidemiology and diagnostic pitfalls. J Trauma 2000;48:724-7.
20. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. N Eng J Med 1990;322(20):1405-11.
21. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. JAMA 1997; 277(20):1597-604.
22. Sayer FT, Kronvall E, Nilsson OG. Methylprednisolone treatment in acute spinal cord injury: The myth challenged through a structured analysis of published literature. Spine J 2006;6(3):335-43.
23. Roberts I, Yates D, Sandercock P, et al. Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head injury (MRC CRASH trial): Randomised placebo-controlled trial. Lancet 2004; 364(9442):1321-8.
24. Edwards P, Arango M, Balica L, et al. Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury outcomes at 6 months. Lancet 2005;365(9475):1957-9.
25. Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal cord injury-A randomized, placebo controlled trial with GM-1 ganglioside. N Eng J Med 1991; 324(26):1829-38.
26. Harris MB, Sethi RK. The initial assessment and management of the multiple-trauma patient with an associated spine injury. Spine 2006;31 Suppl-11:S9-15.
27. Fehlings MG, Perrin RG. The timing of surgical intervention in the treatment of spinal cord injury: A systematic review of recent clinical evidence. Spine 2006;31 Suppl-1: S28–35.
28. Waters RL, Adkins RH, Yakura JS, Sie I. Effect of surgery on motor recovery following traumatic spinal cord injury. Spinal Cord 1996;34:188-92.
29. Collons WF. Surgery in the acute treatment of spinal cord injury: A review of the past 40 years. J Spinal Cord Med 1995;18:3-8.
30. Vaccaro AR, Daugherty RJ, Sheehan TP, et al. Neurologic outcome of early versus late surgery for cervical spinal cord injury. Spine 1997;22:2609-13.
31. Fehlings MG, Tator CH. An evidence-based review of decompressive surgery in acute spinal cord injury: Rationale, indications, and timing based on experimental and clinical studies. J Neurosurg 1999;(91suppl 1):1–11.
32. Chen TY, Dickman CA, Eleraky M, Sonntag VK. The role of decompression for acute incomplete cervical spinal cord injury in cervical spondylosis. Spine 1998; 23:2398-2403.
33. Mirza SK, Krengel WF III, Chapman JR, et al. Early versus delayed surgery for acute cervical spinal cord injury. Clin Orthop 1999;359:104-14.

34. Hellivel V, Gabbott DA. The effect of single handed cricoid pressure on cervical spine movement after applying manual inline stabilization. A cadaver study. Resuscitation 2001;49:53-57.
35. Heath KJ. The effect on laryngoscopy of different cervical spine immobilization techniques. Anesthesia 1994;49:843-5.
36. Suderman VS, Crosby ET, Lui A. Elective oral tracheal intubation in cervical spine injured adults. Can J Anaesth.1991;38:785-9.
37. Lennarson PJ. Smith D, Todd MM, et al. Segmental cervical spine motion during orotracheal intubation of the intact and injured spine with and without external stabilization. J Neurosurg 2000; 92(Suppl 2): 201-6.
38. Sawin PD, Todd MM, Traynelis VC, et al. Cervical spine motion with direct laryngoscopy and orotracheal intubation. An in vivo cinefluoroscopic study of subjects without cervical abnormality. Anesthesiology 1996;85:26-36.
39. Hastings RH, Wood PR. Head extension and laryngeal view during laryngoscopy with cervical spine stabilization maneuvers. Anesthesiology 1994;80:825-31.
40. Nolan JP, Wilson ME. Orotracheal intubation in patients with potential cervical spine injuries. An indication for the gum elastic bougie. Anaesthesia 1993;48:630-3.
41. Hastings RH, Vigil AC, Hanna R, Yang BY, Sartoris DJ. Cervical spine movement during laryngoscopy with Bullard McIntosh and Miller laryngoscopes. Anesthesiology 1995;82:859-69.
42. Laurent SC, de Melo AE, Alexander-Williams JM. The use of the McCoy laryngoscope in patients with simulated cervical spine injuries. Anaesthesia 1996;51:74-5.
43. Abrams KJ, Desai N, Katsnelson T. Bullard laryngoscope for trauma airway management in suspected cervical spine injuries. Anesth Analg 1992;74:623.
44. Weiss M, Hartmann K, Fischer JE, Gerber AC. Use of angulated intubating laryngoscope in children undergoing manual inline neck stabilization. Br J anaesth 2001; 87:435-58.
45. Donaldson WF, Towers JS, Doctor A. A methodology to evaluate motion of the unstable spine during intubation techniques. Spine 1993;18:2020-23.
46. Donaldson WF, Heil BV, Donaldson VP, Silvaggio VJ. The effect of airway maneuvers on the unstable C1-C2 segments. A cadaver study. Spine 1997;22:1215-8.
47. Benumof JL. Management of the difficult adult airway. With special emphasis on awake tracheal intubation. Anesthesiology 1991;75:1087-110.
48. Nakazawa K, Tanaka N, Ishikawa S, et al. Using the intubating laryngeal mask airway (LMA-Fastrach) for blind endotracheal intubation in patients undergoing cervical spine operation. Anesth Analg 1999; 89:1319-3121.
49. Wong JK, Tongier WK, Armbruster SC, White PF. Use of the intubating laryngeal mask airway to facilitate awake orotracheal intubation in patients with cervical spine disorders. J Clin Anesth 1999;11:346-8.
50. Keller C, Brimacombe J, Keller K. Pressure exerted against the cervical vertebrae by the standard and intubating laryngeal mask airways: a randomized controlled cross over study in fresh cadavers. Anesth Analg 1999;89:1296-1300.
51. Kihara S, Watanabe S, Brimacombe J, Taguchi N, Yaguchi Y, Yamasaki Y. Segmental cervical spinal movement with the intubating laryngeal mask during manual inline stabilization in patients with cervical pathology undergoing cervical spine surgery. Anesth Analg 2000; 91;195-200
52. Goto T, Crosby G. Anaesthesia and the spinal cord. Anesthesiology Clinics Of North America 1992;10(3):493-513.
53. Horlocker TT, Wedel DJ. Anesthesia for Orthopaedic Surgery. In: Barash PG, Cullen BF, Stoelting RK, (Eds) Clinical Anesthesia, 5th edn. Philadelphia: Lippincott Williams & Wilkins; 2006. pp. 1112-28.
54. Sagi HC, Beutler W, Carroll E, Connolly P. Airway complications associated with surgery on the anterior cervical spine. Spine 2002;27:949-53.

28 Anesthesia for Thoracic Trauma

Jyoti S Magar, Deepali P Thakur

KEY POINTS

- Chest trauma ranks third behind head and extremity trauma in major accidents and motor vehicle accident is the most common etiology.
- In patients sustaining blunt cardiothoracic injuries, multisystem injuries involving, head, abdomen, spine and extremities frequently coexist.
- A high index of suspicion coupled with an aggressive diagnostic and therapeutic approach, remains the cornerstone of treatment to minimize the morbidity and mortality.
- Assessment and treatment of a trauma patient follows the ABCDE algorithm: Airway, breathing, circulation, neurologic disability, and exposure/environment.
- Most of these patients can be successfully treated with tube thoracostosmy and respiratory and hemodynamic support and very few require open thoracotomy.
- Tracheal intubation is frequently required in chest trauma for respiratory insufficiency. Preferred protocol for securing airway in patient without sign of difficult intubation is rapid sequence intubation within line cervical stabilization.
- If airway appears difficult or with direct injury to airway, trachea is intubated awake with maintaining spontaneous respiration ideally with fiberoptic bronchoscope.
- Laryngeal injury is suspected if patient has hoarseness of voice, subcutaneous emphysema and palpable crepitus over neck and/or hemoptysis.
- The presence of subcutaneous emphysema, pneumomediastinum, pneumopericardium, or pneumoperitoneum, on plain chest radiography or chest CT without apparent cause, is strongly suggestive of possible tracheobronchial injuries.
- Tension pneumothorax is a life-threatening condition that should be diagnosed in a trauma patient clinically by presence of shock associated with the absence of breath sounds and hyperresonance note.
- A trauma patient in shock, associated with the absence of breath sounds and/or dullness on one side of the chest should be treated for massive hemothorax until proven otherwise.
- Rib fractures are usually associated with flail segment, lung contusions, pneumothorax and hemothorax. Upper-rib fractures (1st and 2nd) may be associated with thoracic aortic injuries and lower-rib fractures with liver and spleen injuries.
- Epidural analgesia provides the best pain relief for patients with multiple rib fractures.
- Limiting peak and plateau pressure during mechanical ventilation are important management strategies in patient with lung injury including pulmonary contusion.
- In a patient with history of chest trauma, if the examination findings of distended neck veins, hypotension and muffled heart sound or pulses paradoxus[Becks triad] is found, then it indicates cardiac tamponade.
- Fluid administration in resuscitation should be balanced to limit hypoperfusion against an undesirable increase in blood pressure and thus blood loss.
- The most commonly reported complications following blunt cardiac injury are dysrhythmias and cardiac failure. Echocardiography is the diagnostic method of choice.
- Thoracic CT with contrast is the initial test of choice to diagnose aortic injury in the setting of a widened mediastinum noted on chest X-ray.
- Risk factors for developing paraplegia after aortic surgery include duration of aortic cross-clamping, intra-operative hypotension and surgical technique.

- The most common error in diaphragmatic trauma is failure to suspect the possibility of diaphragmatic injury.
- Esophageal trauma is lethal if unrecognized because it leads to mediastinitis.
- Anesthetic management of video assisted thoracoscopic surgeries requires the use of one lung ventilation.
- Efficient pain management in cardiothoracic trauma is a must to prevent neurohumoral stress response and to improve ventilatory mechanics.
- Mortality has been found to be higher with blunt injuries as compared to penetrating injuries.
- ARDS, multiple organ system failure and sepsis are the major late causes of death in cardiothoracic trauma.

Trauma is a major cause of morbidity and mortality worldwide. Chest trauma ranks third behind head and extremity trauma in major accidents in the United States and it accounts for approximately 20 to 25 percent of traumatic deaths.[1,2] The motor vehicle accident is the most common etiology (70%).[1] Significant number of cardiothoracic injuries at the most urban trauma center is due to increasing use of high speed travel and continued interpersonal violence.[1,2]

Critically injured trauma patients comprise wide range of unique management challenge for entire medical team because they have multiple injuries to multiple body systems. In patient sustaining blunt cardiothoracic injury, multisystem injuries such as head, face, spine, extremities, and abdomen can coexist. Of these, most commonly associated injuries are head and abdomen.[2,4] Cardiothoracic trauma includes injury to chest wall, trachea, bronchus, lung, pleura, heart, thoracic great vessels, diaphragm and esophagus. Most significant injuries to the heart or great vessel structures and lung are immediately fatal while early death within few hours are secondary to airway obstruction, hypoxia, hemorrhage, cardiac tamponade, hemopneumothorax or aspiration.[5,6] Two-third of these cardiothoracic trauma victims, reach hospital alive but many of these die after reaching hospital. Some of these deaths can be prevented by prompt diagnosis and treatment. Most of these patients can be successfully treated with tube thoracostomy and respiratory and hemodynamic support. Very few potentially fatal traumatic cardiothoracic injuries require open thoracotomy for emergency surgical intervention.

Anesthesiologists practicing at designated trauma centers are involved in the care of trauma patients beginning with airway management and resuscitation in the emergency department (ED) and proceeding through the operating room (OR) to the intensive care unit (ICU). Early involvement of anesthesia provider is particularly important and continued reassessment of overall patient is essential because surgical intervention may be required urgently at any time of evaluation. Major thoracic injuries can occur without a clear external damage or skin wound. Also, there can be extra thoracic injuries which can delay diagnosis of chest trauma.[2] A high index of suspicion coupled with an aggressive diagnostic and therapeutic approach, remains the cornerstone of treatment to minimize the morbidity and mortality of these injuries. The approach to diagnosis and treatment of injuries to the chest depends greatly on the mechanism of injury and the evidence and type of associated injury. The history of mechanism of injury is important as it can predict type of injuries patient can get.[3]

Cardiothoracic trauma is classified into blunt and penetrating trauma:

- Penetrating wounds of chest occur with gunshot or stab injury. Thus, they result in fracture ribs, pneumothorax, hemothorax, pulmonary and/or cardiac injury
- Blunt forces to chest wall cause injury by rapid deceleration, direct impact and compression. Rapid deceleration is involved in high speed motor vehicular collision or fall from height. Direct impact by blunt object can cause localized fracture of bony chest wall with underlying lung parenchymal injury, blunt cardiac injury, pneumothorax, and/or hemothorax.

Both of these injuries, penetrating and as well as blunt can occur in patients with bomb blast injury or with fall from moving vehicle.

Initial approach to assessment and management should follow principle of American College of Surgeons's (ATLS) advanced trauma life support protocol.[7] Time factor is critical in trauma. Organized management approach helps to avoid chaos and promote appropriate life sustaining therapy in time.

Initial assessment and treatment of patient with trauma consists of:

- Primary survey along with resuscitation of vital organs followed by
- Detailed secondary survey and definitive care.

Complete primary survey can be challenging in emergency room. It must be performed in such a manner as to include quick physical examination and integrate resuscitation procedure. Primary survey

involves assessment and management of airway, breathing, circulation and disability and exposure with global search for injuries which are immediate threat to life and which require immediate therapy. Continuous reassessment of overall patient status is essential because surgical intervention may be required urgently.

Early intervention in cardiothoracic trauma includes oxygen supplementation, rapid tracheal intubation, large bore intravenous assess, fluid resuscitation and if required chest tube insertion to prevent and correct hypoxia. Preoperative assessment and management of specific thoracic injuries is done as per ATLS protocol.

AIRWAY ASSESSMENT AND MANAGEMENT

Airway assessment and management are of primary importance because tissue hypoxia is the most immediate threat to life. The goal of emergency airway management is to ensure adequate oxygenation and ventilation while protecting the patient from the risks of aspiration and airway obstruction. Airway patency and adequacy of ventilation is assessed by checking patient's verbal response. If appropriate it suggests that airway is patent, ventilation is adequate with adequate cerebral oxygenation. Respiratory movement and quality of respiration should be assessed by observation, palpation and listening. Important but often subtle sign of chest injury or hypoxia include increased respiratory rate, change in breathing pattern especially progressively shallow respiration. Airway patency and adequacy of ventilation should be monitored with continuous oxygen saturation monitoring with pulse oximeter and end tidal CO_2 monitoring. If required arterial blood gas can be monitored.

- Preferred protocol for securing airway in patient without sign of difficult intubation is rapid sequence intubation within line cervical stabilization. Regardless of whether endotracheal tube is already inserted, proper placement must be objectively confirmed by direct laryngoscopy.
- If airway appears difficult or with direct injury to airway, awake fiberoptic intubation can be used.
- If intubation is not possible then definitive airway is achieved with cricothyroidotomy or tracheostomy.[8] All airway manipulation must be performed with cervical spine immobilization until cervical spine injury is ruled out. Postintubation X-ray is required to further confirm endotracheal tube position. The same protocol is followed for intubation in operation theatre for emergency surgery if patient is not intubated.[9-12]

Following injuries must be identified and treated at the earliest in patients with cardiothoracic trauma. Patients may come for surgery primarily for these injuries or these injuries may be present as associated injuries.

Laryngeal Injury

Laryngeal injuries are less common but clinically important injuries as their initial management has a tremendous impact on the probability of survival of the patient, as well as long-term quality of life.[13-15] It can be caused by blunt or penetrating trauma. Penetrating trauma commonly occurs as a result of gunshot wounds or knife injuries. With penetrating injury as in cut throat, presentation is overt and requires emergent operative exploration and is usually associated with injuries of esophagus, carotid artery, jugular vein. Motor vehicle accidents are the most common cause of anterior blunt trauma to the larynx. Clothesline injuries typically occur in motorcyclists with direct blow to neck. Strangulation injuries occur as a result of compressive forces from assaults with blunt objects or from attempted suicides by hanging. With blunt trauma, presentation is subtle therefore history is important. High index of suspicion is needed for rapid diagnosis and successful surgical intervention. Laryngeal injury is suspected if patient has hoarseness of voice, subcutaneous emphysema and palpable crepitus over neck and /or hemoptysis. In the absence of airway compromise, when symptoms are subtle, diagnosis is confirmed by examination of upper airway with computed tomography (CT). Bronchoscopy is necessary to determine degree of damage following blunt laryngeal or neck trauma.[13-15] Acute airway obstruction from neck trauma is life-threatening and emergency cricothyrotomy may be required.

In patient with laryngeal trauma and airway compromise with stridor, method of choice for securing airway is fiberoptic bronchoscope assisted intubation while maintaining spontaneous ventilation. Intubation over FOB allows visualization of airway as ETT is passed beyond site of injury and eliminates risk of danger associated with conventional laryngoscope. Blind intubation technique is contraindicated in airway injury. Over sedation and neuromuscular relaxant are best avoided as they may result in loss of airway.[13-15] Early surgery for repair of laryngeal injury is indicated to avoid complications such as delayed airway obstruction and voice compromise associated with late repair. Laryngeal injuries can be associated with fracture of skull base, intracranial damage, open neck wound and injury to cervical spine, esophagus or pharynx.[16]

Tracheobronchial Injuries

Tracheobronchial injury can result from either blunt force or penetrating trauma. Penetrating injuries are usually more promptly diagnosed and treated. In blunt trauma with rapid deceleration as in direct blow in unrestrained driver, trachea and major bronchi may rupture due to acute intraluminal pressure elevation or may be lacerated after shear forces usually applied to the level of the carina. Blunt trauma most commonly results in an injury to the tracheobronchial tree within 2.5 cm of the carina and may initially be unrecognized. The presence of subcutaneous emphysema, pneumomediastinum, pneumopericardium or pneumoperitoneum on plain chest radiography or chest CT, without apparent cause, is strongly suggestive of possible tracheobronchial injury.[17] Other findings are hemoptysis, dyspnea, hypoxia and/or stridor. A large pneumothorax is present if there is free communication between the rupture of the tracheobronchial tree and the pleural cavity (bronchopleural fistula). Tube thoracotomy thus shows persistent large air leak. Loss of more than one-third of the delivered tidal volume in mechanically ventilated patient is also strongly suggestive of tracheobronchial injuries. Tracheobronchial injuries are also suspected in patient with fracture upper ribs, clavicle or sternum and in patient with lung contusion. Helical CT scans are helpful in establishing diagnosis but fiberoptic bronchoscopy is the most reliable means of diagnosing and staging tracheobronchial injury.[17] Massive air leak is indication for thoracotomy if suction fails to re-expand the lung. Tracheobronchial injuries should be repaired surgically with thoracotomy as soon as possible in order to diminish risk of repeated pulmonary infection, severe bronchial stenosis or mediastinitis.

Anesthesia care is directed towards airway control, maintenance of adequate ventilation and management of blood loss.[17] Inflation pressure should be minimized and spontaneous respiration maintained if possible until surgical repair can be performed. Airway management of patient with bronchial injury may require placement of double lumen tube, endobronchial tube or bronchial blocker or temporary intubation of opposite mainstem bronchus to bypass lesion and to provide adequate oxygenation and for surgical exposure.[17] Tolerance of single-lung ventilation will depend on the presence of significant pathology in the ventilated lung and thus may require increased FiO_2 and high levels of PEEP to maintain adequate oxygenation.

Following thoracic injuries that impact breathing must be recognized and treated promptly.

Tension Pneumothorax

It is caused by injury to lung parenchyma or airway in the following circumstances:

- Penetrating or blunt chest injury
- Misguided attempt at subclavian or internal jugular venous catheter insertion
- Displaced thoracic spine fracture.

It develops when air enters pleural space from lung or from chest wall via one way valve like opening which allows entry of air but no exit. Progressive increase in intrathoracic pressure leads to complete collapse of affected lung, shift of mediastinum to contralateral side and severe impairment of central venous return may be present. Compression of contralateral lung occurs by displaced mediastinum which further impairs ventilation capacity resulting in hypoventilation and hypoxia. Decreased venous return by elevated intrathoracic pressure leads to profound hypotension and cardiac arrest if untreated. Tension pneumothorax is clinically characterized by chest pain, respiratory distress with dyspnea. On examination there will be tachycardia, hypotension, neck vein distention, tracheal deviation, unilateral absent or decreased air entry with hyper-resonant note on affected side. There will be decreased saturation on pulse oximeter and increased airway pressure or resistance on ventilation. X-ray chest will show hyper lucency with widened intercostals spaces on affected side, depressed ipsilateral diaphragm, shift of mediastinum and trachea to opposite side (Fig. 28.1). CT scan may reveal even small pneumothorax which is not identifiable on conventional chest radiographs.

Tension pneumothorax, is a life-threatening condition that should be diagnosed clinically. Treatment should not be delayed while waiting for radiological confirmation. Tension pneumothorax requires immediate decompression and is managed initially by rapidly inserting large caliber needle into second intercostal space in midclavicular line of affected hemithorax. This maneuver converts tension pneumothorax into simple pneumothorax. However, possibility of subsequent pneumothorax as a result of a needle stick injury may occur and repeated reassessment of patient is necessary. Definitive treatment usually requires tube thoracostomy with insertion of chest tube in 5th intercostals space (usually at nipple level), just anterior to midaxillary line. Suction may be applied to the pleural space until large air leak resolves spontaneously. Chest X-ray is needed to confirm expansion of lung. If the lung does not fully re-expand after tube thoracostomy and there is a large ongoing air

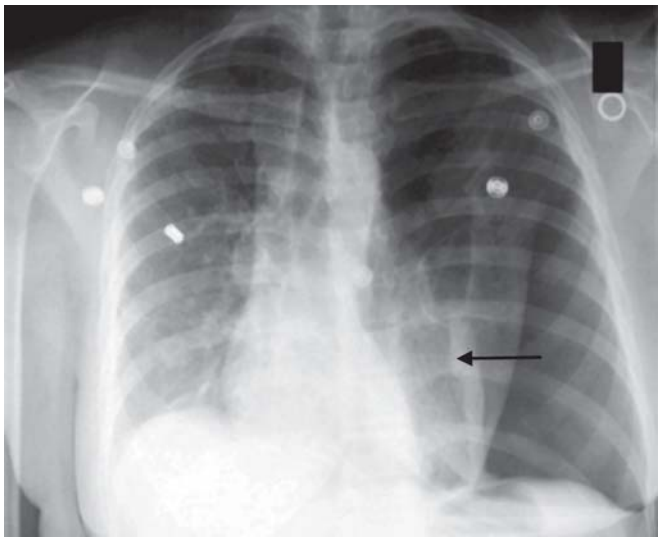


Fig. 28.1: Tension pneumothorax with collapse lung

leak, the airways should be evaluated bronchoscopically to exclude airway injury. Usually in most cases no other treatment than chest tube thoracostomy is required. Thoracotomy may be necessary when tracheal or bronchial injury and massive air leak are evident.

In patient with small pneumothorax or subcutaneous emphysema, chest tube should be inserted on the same side before positive pressure ventilation for general anesthesia or for mechanical ventilation and positive end expiratory pressure (PEEP) and nitrous oxide should be avoided with anticipation of tension pneumothorax.[18]

Open Pneumothorax

It is usually caused by wound with a large defect of chest wall that remains open. This creates a communication between pleural space and external environment and thus results in open pneumothorax or sucking chest wound. As size of this chest wall defect approaches 2/3 diameter of trachea, air passes preferentially through low-resistance injury tract with each respiratory effort rather than normal airways.

In the open or sucking wound of chest wall, there is immediate equilibration of intrathoracic and atmospheric pressure resulting in collapse of lung on affected side and shifting mediastinum to unaffected side. This leads to profound ventilation perfusion mismatch which leads to hypoxia and hypercapnia. This is an immediate life-threatening condition. In spontaneously breathing patient, open pneumothorax is managed by application of sterile adhesive dressing which must be large enough to cover the entire wound and is taped securely on three sides. This will then act as a one way valve so that air can escape pleural space but cannot renter. Taping all edges of dressing before chest tube insertion will lead to development of tension pneumothorax. Tube thoracostomy should be performed as soon as possible at remote site away from wound. If chest wall defects are relatively small, pleura may soon seal and no further intervention is necessary. In patient with airway or breathing difficulty, early intubation and initiation of positive pressure ventilation should be considered. For large open chest wall defects, surgical debridement of dead and devitalized tissue and closure of wound with or without prosthetic patch is often required under general anesthesia.

Fracture Ribs, Pulmonary Contusion and Flail Chest

Rib fracture is the most common injury resulting from blunt chest trauma. It usually results from direct impact of chest wall as in motor vehicular collision or anterior chest compression. Rib fractures contribute significantly to the morbidity and mortality associated with chest injuries.[19-21] The elderly and patients with poor respiratory reserve are particularly vulnerable.[22] Rib fractures are usually associated with flail segment, lung contusions, pneumothorax and hemothorax. Upper rib fractures (1st and 2nd) may be associated with thoracic aortic injuries and lower rib fractures with liver and spleen injuries. A chest X-ray will not only identify the number and the extent of rib fractures, but also to determine whether there is an associated pneumothorax, hemothorax or lung contusion.

A flail chest is defined by fractures of two or more ribs at two or more sites on each rib or fracture of multiple neighboring ribs (Fig. 28.2). It is an indicator of severe blunt trauma to the chest. It is characterized by paradoxical chest wall motion during spontaneous ventilation. Flail chest causes pain with respiratory movement and is almost always associated with pulmonary contusion. Pulmonary contusion is a loss of vessel integrity due to concussion, resulting in intraparenchymal and alveolar hemorrhage and/or edema. Frequency and extent of lung contusion are proportional to severity of thoracic injury. Pulmonary contusion will cause shunting, which will lead to hypoxemia. Symptoms and signs of pulmonary contusion are dyspnea, hypoxemia, cyanosis, and tachycardia and decreased or absent breath sounds. An initially clear chest radiograph does not exclude the possibility of a pulmonary contusion, but usually shows patchy, undefined densities or homogeneous consolidation. In lung contusion, chest X-ray changes tend to

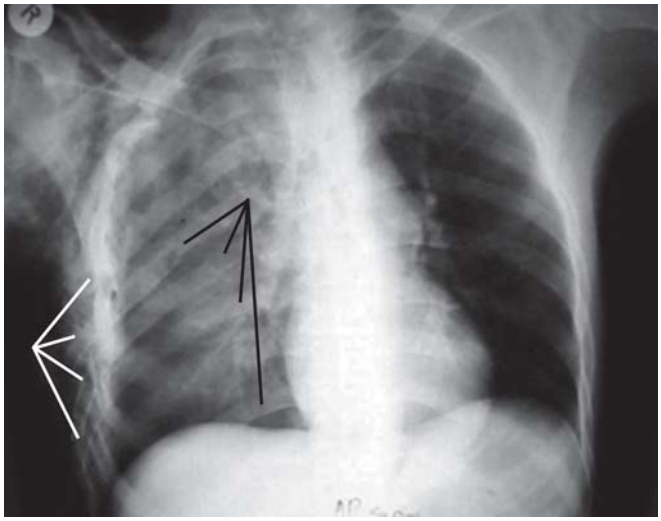


Fig. 28.2: Flail chest with underlying lung contusion

lag behind the patient's condition and the extent of lung injury is usually greater than suspected radiologically (Fig. 28.2). Chest CT scan permits a more complete evaluation of the pulmonary lesion and again, close observation is warranted if signs of significant chest wall trauma are noted.[23] No specific treatment of pulmonary contusion is available and therapy is directed at the associated injuries or resultant hypoxemia. The natural history of pulmonary contusion is variable; contusions may resolve without sequels or may lead to either pneumonia or ARDS.[24-26] Respiratory distress is common with hypoxemia and hypercarbia is greatest on about third day.[25,26] Younger trauma victims usually have lung contusion without flail segment due to more compliant chest wall.

Multiple rib fractures lead to severe pain. Pain limits patient's ability to cough and breathe deeply, resulting in sputum retention, atelectasis and a reduction in FRC. These factors in turn result in decreased lung compliance, V/Q mismatch and hypoxemia. Small flail segment without respiratory impairment generally do well with pain control without ventilator assistance. Failure to control pain, compounded by the presence of pulmonary contusion, flail segment and other insults, can result in serious respiratory complications, including respiratory failure and subsequent pneumonia which often requires intubation, mechanical ventilation and aggressive suctioning. The primary indication for tracheal intubation is respiratory decompensation.[24-26]

Pneumonia is the most common complication after rib fractures. The entire management should be targeted to the prevention of lung infection rather than the actual treatment of the rib fracture. Therapy consists of adequate treatment of pain in order to allow the patient deep breathing and proper lung expansion.[27,28] Chest physiotherapy is equally important. The best physiotherapy is both active and passive mobilization of the patient. The patients should be encouraged to breathe deeply, cough and use incentive spirometry. There is no role for prophylactic antibiotics in this condition but antibiotic therapy may be indicated to treat pneumonia and other infections.

Pain management goals are focused upon allowing patient to breathe deeply and cough effectively to mobilize and clear secretions, thus improving ventilatory mechanics and preventing atelectasis. Pain treatment consists of:

- Oral medication for ambulatory patients or patients with minimal to moderate pain
- Adequate pain control is provided in patient with flail segment, ideally by means of epidural analgesia which is proven to offer the best pain relief. Epidural analgesia minimizes complications of splinting with pain such as hypoxia, hypoventilation, need for intubation and possibility of pneumonia[27-29]
- Patient controlled analgesia (PCA) offers another good method for pain relief
- Continuous intravenous analgesia is reserved for mechanically ventilated patients
- Other analgesic techniques are nerve blocks and interpleural catheter. Early application of continuous positive airway pressure (CPAP) improves V/Q mismatch, FRC, lung compliance, thus enhances efficiency of gas exchange and spontaneous ventilation.[30]

Endotracheal intubation is reserved for patients who are unable to maintain oxygenation or breathe adequately or who require protection of airway. In patients, who subsequently require intubation for surgical procedure, an anestheologist will have to determine safety of extubation postoperatively and postoperative need of adequate analgesic. Early extubation and application of continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) is one of the successful weaning technique.[30] Early and aggressive implementation of lung protective strategy is crucial in treatment of patient with significant pulmonary contusion to minimize progression to ARDS with ventilator associated lung injury (VALI). Prevention of VALI should be a standard part of the anesthetic management of all severely injured chest trauma patients intraoperatively to prevent further lung injury. Limiting peak and plateau pressure and tidal volume and avoiding over distention during mechanical ventilation are important manage-

ment strategies in these patients. Pressure controlled ventilation minimizes peak and plateau airway pressures and may help prevent barotrauma.[31,32] Pulmonary parenchymal repair or resection including tractotomy and repair, wedge resection, lobectomy and pneumonectomy may be required in lung contusion.[32]

Massive Hemothorax

Rapid accumulation of more than 1500 ml of blood in pleural space causes massive hemothorax. Such a massive hemorrhage results either secondary to blunt or penetrating trauma of chest, more common being penetrating trauma. Bleeding occurs either from large pulmonary laceration or injury to great vessel like descending thoracic aorta, innominate artery, pulmonary vein or artery or internal mammary or intercostals artery or even heart. There can be bleeding from an abdominal structure like liver or spleen when diaphragm ruptures especially with blunt trauma. Massive hemothorax causes hemodynamic instability due to loss of intravascular blood volume and due to decreased central venous return because of increased intrathoracic pressure and mediastinal shift. Hemothorax also causes respiratory compromise due to compression of lung with blood accumulation. Chest X-ray will show evidence of fluid in pleural space (Fig. 28.3). Diagnosis is readily evident from clinical picture. Thus, a trauma patient in shock associated with absence of breath sound and/or dullness on one side of chest should be treated for massive hemothorax until proven otherwise. Initial management includes simultaneous resuscitation of blood volume and decompression of chest cavity with large (36-40 Fr) chest tube. Blood collected from pleural space may be auto transfused as it is devoid of clotting factor.[33]

Persistent hemorrhage usually arises from intercostal or internal mammary artery. Bleeding from lung generally stops within few minutes after lung expansion although initially it may be profuse. Thus most cases of hemothorax can be adequately managed by tube thoracostomy and restoration of circulating blood volume.[34] Urgent thoracotomy is strongly indicated if initial chest tube output is greater than 1500 ml in adult or 20 ml/kg in pediatric patient or with continued bleeding of more than 250 ml/hr in adult or more than 2 to 3 ml/kg /hr in pediatric patient for more than 3 to 4 consecutive hour.[34]

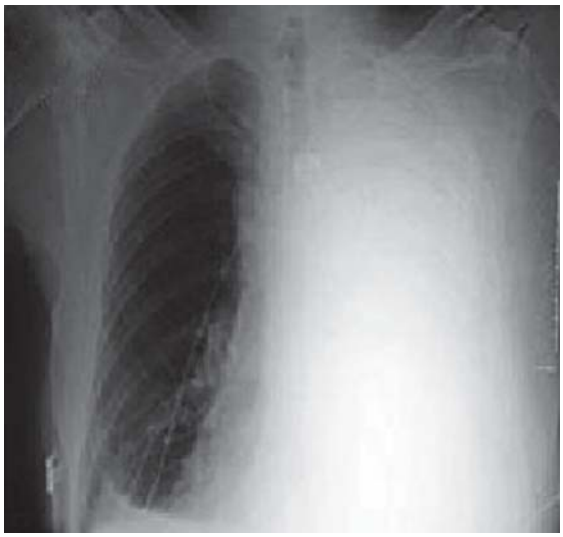


Fig. 28.3: Massive hemothorax

CIRCULATION

Circulation is assessed by palpating pulse (radial, carotid or femoral artery) and with blood pressure measurement. Absent radial pulse suggests hypovolemic shock with systolic BP less than 70 mm Hg. Neck veins are usually distended with cardiac tamponade or due to tension pneumothorax but may be flat if associated hypovolemic shock. Early ECG monitoring required for early diagnosis of dysrhythmias or pulseless electrical activity and pulse oximeter monitoring is required for early diagnosis of hypoxia. Pulseless electrical activity is present in many cases such as cardiac tamponade, tension pneumothorax, profound hypovolemia and hypoxia. FAST [Focussed Assessment by Sonography in Trauma] can immediately diagnose tamponade and abdominal hemorrhage.[35] If FAST is inconclusive then more formal transthoracic or transesophageal ECHO is extremely useful for the diagnosis cardiac tamponade.

Treatment of hemorrhagic shock associated with cardiothoracic trauma requires rapid administration of crystalloids, colloids and blood. Fluid administration to a patient who is actively hemorrhaging may be counterproductive because it dilutes red cell mass which reduces oxygen delivery and contributes to both hypothermia and coagulopathy. Elevation of blood pressure before definitive treatment of thoracic vascular injury leads to increased bleeding as a result of disruption of clots and reversal of compensatory vasoconstriction.[36-38] Thus in absence of head injury moderate hypotension, i.e. systolic blood pressure 90 mm Hg is maintained before hemostasis. Thus, fluid administration is balanced to limit hypoperfusion against an undesirable increase in blood pressure and thus blood loss.[39]

Following life-threatening cardiovascular injuries must be identified and treated.

Cardiac Injuries

Penetrating injury to precordium has high mortality. Classic precordium is bounded by sternal notch superiorly, bottom of anterior ribs inferiorly and nipple laterally. Majority of stab wounds to the heart enter through this zone while majority of gunshot wound strike heart without entry in this zone.[40] Direct gunshot wound to heart almost always has 100 percent mortality immediately due to exsanguination. However, low velocity stab wound victim usually survives if injury to vital cardiac structure such as valves and coronary arteries are missed. Eighty to ninety percent of patients with stab wounds to heart present with cardiac tamponade.[40] Diagnosis is easily made with high index of suspicious of cardiac injury in cardiothoracic trauma. If in patient with history of chest trauma has examination finding of Becks triad, that is distended neck veins, hypotension and muffled heart sounds or pulsus-paradoxus, then it indicates tamponade. Transthoracic echocardiography (TTE) is noninvasive, rapid and accurate in rapid diagnosis of even occult cardiac tamponade.[41] FAST may be equally effective and fast but requires experienced sonologist.[41] In chest X-ray, radiological signs suspicious of cardiac injury are enlarged, globular cardiac shadow, widened upper mediastinum due to dilated major veins as a result of the tamponade and venous stasis. ECG may be diagnostic, may show low QRS complexes, elevated or depressed ST segments and inverted T-waves. Pericardiocentesis recommended by ATLS protocols however has very limited value in organized, modern trauma centers.

Pericardiocentesis or alternately subxiphoid pericardial window may be still useful for diagnosis and temporary release of tamponade in situation where TTE is not available.[42,43] However, once tamponade is diagnosed and relieved by subxiphoid window or pericardiocentesis, median sternotomy is needed to repair damage and control the bleeding structure. Immediate treatment of cardiac tamponade in trauma consists of aggressive fluid replacement and open surgical drainage. If patient has high likelihood of tamponade and is hemodynamically compromised, an emergent thoracotomy should be considered. Repair of many penetrating cardiac injuries can be done without cardiopulmonary bypass. Temporary asystole can be induced with adenosine. This allows surgeon time to accurately place required number of sutures in semi-bloodless and motionless field to adequately control hemorrhage. Owing to ultrashort half-life of adenosine (approximately 5-20 sec); it leads to prompt restoration of sinus rhythm afterwards.[44,45] ECG monitoring, pacing back up, corrective measures for untoward hemodynamic effect (e.g. phenylephrine, ephedrine) and defibrillation capabilities are required while using adenosine. Right ventricular injuries have the best prognosis. Certain injuries like pulmonary artery laceration, ventricular septal defect may require cardiopulmonary bypass.[46]

Blunt Cardiac Injury

Blunt cardiac injury (BCI) can result from deceleration injuries, such as in high speed traffic accidents or falls with direct precordial blow. Fractured sternum or anterior ribs should increase the suspicion of blunt cardiac trauma. Blunt cardiac injury can result in ventricular dysfunction, cardiogenic shock, dysrhythmia, free wall or septal wall rupture, valvular tear, coronary artery thrombosis simulating like myocardial infarct.[47-51] Damage occurs most frequently to right ventricle and septum.[47] The most commonly reported complications following BCI are dysrhythmias and cardiac failure.[48] Sinus tachycardia is the most common abnormality noted but can only be attributed to myocardial contusion when other causes of tachycardia have been ruled out, notably hypovolemia and pain.

TEE is useful tool in blunt cardiac injury in detecting complication of BCI such as severe focal or global ventricular dysfunction, pericardial effusion and/or valvular dysfunction. It is especially indicated in suspected blunt chest injury with ECG abnormality or unexplained cardiovascular instability. There is no correlation between troponin levels and clinical presentation or the severity of myocardial injury.[52]

Patients with suspected blunt chest injury mainly those with ST segment changes, should be observed for dysrhythmias and hypotension with cardiac monitoring in surgical intensive care unit and need at least 24 hours monitoring in ICU. In patient with blunt chest trauma with normal ECG, normal BP, no dysrhythmias on admission, do not require further investigation for BCI with echocardiography.[53]

Presence of BCI may increase perioperative morbidity and mortality due to intraoperative arrhythmias and hypotension. Urgent surgery in patients with BCI can be safely performed with aggressive hemodynamic monitoring and patient may require inotropic and/or vasopressor support perioperatively to treat hypotension but nonemergent surgery however should be delayed for 48 hours to allow improvement of

myocardial function.[54,55] Cardiopulmonary bypass is required for successful repair of myocardial rupture.

Resuscitative Thoracotomy

It is thoracotomy employed to aid resuscitation of trauma patient. It is generally performed in patient with thoracic or abdominal injuries sustained either by blunt or penetrating trauma that goes onto develop cardiac arrest, decompensating cardiac tamponade or hypotension unresponsive to aggressive fluid administration. Although, overall survival rate for all patients undergoing resuscitation thoracotomy is low, some patients survive solely because of this procedure.[56]

Objectives of resuscitation thoracotomy are:

- To relieve cardiac tamponade
- To perform open cardiac massage as closed massage is ineffective in providing adequate cardiac output in hypovolemic patient
- Occlude thoracic aorta in an attempt to improve cerebral and coronary circulation and decrease intra-abdominal hemorrhage
- Control of life-threatening intrathoracic hemorrhage
- Control of bronchovenous air embolism.[57]

Thus, in general there are three main indications of thoracotomy after traumatic injury, i.e. shock or arrest with suspected correctable intrathoracic lesion, specific diagnosis like cardiac tamponade, penetrating cardiac lesion or aortic injury, evidence of ongoing thoracic hemorrhage.[57-60] On review of survival, resuscitative thoracotomy is futile in patients with no signs of life such as supraventricular electrical cardiac activity, pupillary reaction or agonal respiration and blunt trauma requiring more than 5 minutes of CPR and penetrating trauma requiring more than 15 minutes CPR, provided airway has been secured with endotracheal tube.[58]

Damage Control Thoracotomy

Complete repair of all injuries in patient with multiple injuries at the end of their physiologic reserve often result in repaired but dead patient, therefore damage control measures such as abbreviated thoracotomy to restore suitable physiology during single operation are occasionally necessary in acidotic, hypothermic and coagulopathic patients. Pulmonary tractotomy and repair is an abbreviated method often employed in the patient with trauma to the lung.[61]

Traumatic Aortic Injury

Traumatic aortic injury is caused by sudden and violent deceleration. Mechanism of injury includes high speed motor vehicular collisions, autopedestrian collisions, falls, crush injuries as many as third of patients with these injuries expire on scene. Fifty percent of survivors die within 48 hours if injury is not recognized or treated.[62] Aortic injury occurs most commonly just distal to the left subclavian artery and is the result of shear forces between the mobile heart and aortic arch and the immobile descending thoracic aorta. Traumatic aortic injury encompasses a continuum of injury from a small intimal flap to free transection contained by the surrounding mediastinum and pleura. A contained hematoma around the site of disruption is the reason for most survivors reaching the hospital alive.

Aortic injuries are often missed because those that make it to the hospital alive, often present with minimal external physical finding. A high index of suspicion for an associated aortic injury and its appropriate treatment are keys to survival.[63] The diagnosis is made by screening chest radiography. Radiological signs on X-ray chest are widened mediastinum (more than 8 cm), right tracheal shift, elevation and right ward shift of right bronchus, depression of left bronchus, blurring of aortic knob outline, and deviation of nasogastric tube to right (Fig. 28.4). Other associated radiographic evidence of multiple ribs especially 1st and 2nd rib fracture, scapular, thoracic spine fracture. Routine helical CT is an excellent diagnostic tool and has to a greater extent replaced the aortogram in high risk blunt trauma patients. In many centers it has become the first line investigation for suspected aortic injury.[64-67] Aortography, once dominant modality is still used where new generation CT is not available or if the CT scan has questionable findings.[68] MRI is helpful, but it cannot be done during an acute phase of resuscitation. Transesophageal echocardiogram (TEE) is a good adjunct to the diagnosis of aortic disruption by blunt trauma. It is useful in the setting of critically ill trauma patient because it can be performed in the OR, SICU or the trauma resuscitation unit.[69,70] With TEE, the distal ascending aorta and the aortic arch are difficult to visualize because of the intervening trachea and left main bronchus and an injury in this area may be overlooked.[68-71]

Surgical repair is indicated for most patients with traumatic aortic injury (TAI) because of the high-risk for rupture in the hours and days after injury.[72] Surgical repair of TAI can be done with or without CPB depending on extent of damage and location of tear. Various techniques have been described for this highly morbid surgery, with the best reports recently attributed to partial bypass techniques using inflow from the left atrium, a centrifugal pump and outflow to the descending aorta.[73] Ascending

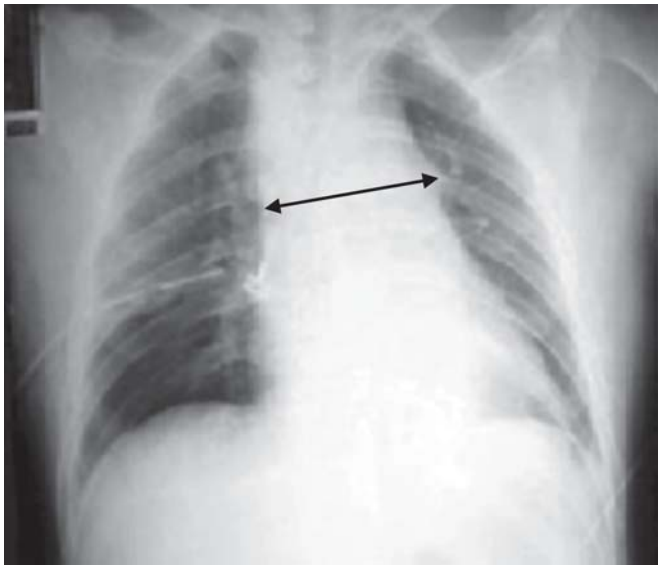


Fig. 28.4: Widened mediastinum

aorta that may require replacement of aortic valve and re-implantation of coronary arteries (i.e. Bentall repair), a period of deep hypothermic circulatory arrest may be required. Repair of descending thoracic aorta tear requires left thoracotomy.[74] A double lumen endotracheal tube should be inserted if possible to facilitate collapse of the left lung and improve visualization of the disrupted aorta. One lung ventilation may not be tolerated in presence of pulmonary contusion mainly in right lung.[74-76]

Hemodynamic monitoring lines should be placed, arterial line in the right radial artery and a right femoral arterial line or dorsalis pedis artery to monitor distal perfusion. Distal perfusion should be maintained at 50 to 70 mm Hg. Major consideration for thoracic aortic surgery include massive blood loss, hemodynamic alteration induced with aortic cross clamp, spinal cord and visceral ischemia during cross clamp, hypothermia, coagulopathy. Major complications after aortic repair are respiratory failure, pneumonia, renal failure, suture line failure and paraplegia. Risk factors for developing paraplegia after aortic surgery include duration of aortic cross clamp (> 30 minutes), intraoperative hypotension and surgical technique.[75,76] Nonoperative management or endovascular stent grafting for blunt TAI should be considered in patients with severe traumatic brain injury (TBI), significant right side pulmonary contusion, hemodynamic instability (systolic blood pressure less 90 mm Hg), coagulopathy or other severe injuries.[77] Endovascular repair has been increasingly shown to be well-tolerated. Furthermore, early and midterm results are promising, although long-term durability and complications of these endovascular stents remain unknown.[78,79] Reports of selective nonoperative management of high-risk patients with traumatic aortic injury have appeared in the recent literature. Treatment consists of β-blockade to minimize the cardiac ratepressure product.[74-81]

Diaphragmatic Trauma

In the acute setting, diaphragmatic injuries are generally not life-threatening but can be associated with a significant morbidity and mortality due to associated injuries or herniation with cardiopulmonary compromise. In addition, if undetected in the acute setting, delayed presentation of diaphragmatic hernias carries an increased risk of complications.

These injuries usually result from either direct penetration of diaphragm as in gunshot and stab injury or accidental perforation or blunt trauma to chest or abdomen as in motor vehicular accident. Left hemidiaphragm as it is little supported from adjacent structures is more susceptible to rupture, while right hemidiaphragm is protected by liver. The most common error in diaphragmatic trauma is failure to suspect the possibility of diaphragmatic injury. Diagnosis is often missed because of associated intra-abdominal injuries to liver, spleen and bowel and sometimes only incidentally discovered during exploratory laparotomy.[82, 83]

Diaphragmatic injury should be suspected in any penetrating thoracic injury at or below 4th intercostal space at the nipple level anteriorly, 6th intercostals space laterally or 8th intercostals space posteriorly.[84] The stomach and other abdominal viscera may herniate into left thorax collapsing left lung, shifting mediastinum and trachea to the right and compressing the right lung. If not diagnosed in time, hernia becomes strangulated leading to ischemia, necrosis, and sepsis. The diagnosis of diaphragmatic injuries can be very difficult especially in the asymptomatic patient.[82-85] Patient may present with dyspnea, chest or shoulder pain, left upper quadrant pain. If stomach is herniated into thorax, dyspnea is relieved dramatically by insertion of nasogastric tube.

The diagnosis is suspected when X-ray chest shows atelectasis with elevation of the ipsilateral diaphragm, evidence of a viscus, air bubble, air-fluid level in the thorax, nasogastric tube above the level of the diaphragm (Fig. 28.5). The diagnosis may be confirmed by ultrasound, CT scan, or laparoscopy. Computerized tomography may allow visualization of diaphragmatic hernia but may not be visualized if not associated with intestinal herniation. Due to the unreliability of physical

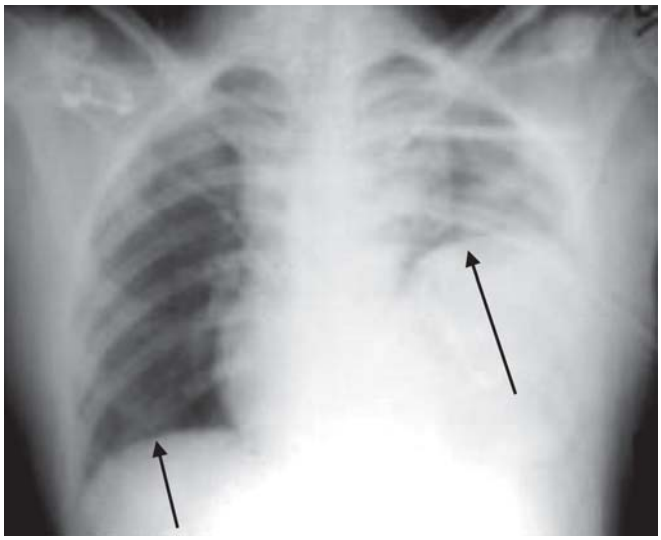


Fig. 28.5: Diaphragmatic eventration

examination and radiographic findings, the only method currently available to definitively diagnose a diaphragmatic injury in the acute setting is by direct visualization. This can be done with laparotomy, laparoscopy, or thoracoscopy.[83,86,87] Video assisted thoracic surgery (VATS) has been found to be the most accurate method (accuracy rate 98%) in diagnosing diaphragmatic injury.[88] Surgical repair may be required to prevent incarceration and strangulation of herniated abdominal contents and to prevent respiratory compromise. Any patient with intrathoracic herniation of abdominal contents must be considered at risk for regurgitation, aspiration, hypoxemia and hemodynamic compromise.

Esophageal Rupture

Penetrating injuries are the most common causes of esophageal trauma. Blunt injury to the esophagus is quite rare because esophagus is in relatively protected location in posterior mediastinum. Severe blow in upper abdomen may result in linear tear in lower esophagus. Multiple associated injuries are the rule rather than the exception.

Most common and obvious are penetrating injuries of esophagus. Cervical esophageal injuries are generally associated with injuries to the major blood vessels of the neck, trachea, cervical spine and spinal cord. Associated injuries occurring in conjunction with thoracic esophageal injuries include major thoracic vascular, cardiac, pulmonary and bony thoracic structures such as ribs, thoracic spine and neurological injuries.[89]

Iatrogenic esophagus perforation has been associated with endotracheal intubation, use of combitube, intubating LMA, nasogastric tube, esophagoscopy, esophageal dilator, insertion of TEE probe.[90, 91]

The diagnosis of esophageal injury also requires a high index of suspicion. Physical examination may be characterized by minimal findings. Physical clue to the diagnosis may include subcutaneous emphysema, pneumomediastinum, pneumothorax or intra abdominal free air. Esophageal trauma is lethal if unrecognized because it will lead to mediastinitis due to contamination of mediastinal space by esophageal content. Patients who present after a significant time after injury may manifest signs and symptoms of systemic sepsis. Mortality rates can triple in patients undergoing surgical procedures after 24 hours.[89]

Diagnosis is often confirmed by esophagogram with contrast study to demonstrate leak and may be performed when the patient is stable. Esophagoscopy with visualization of localized blood or an actual laceration in the esophagus is diagnostic. Esophageal injury in neck if seen early is repaired and reinforced with adjacent muscle or tissue and drained. If leak is diagnosed late, only abscess is drained without any attempt to repair. Esophageal leak in chest is managed with thoracotomy and primary repair if feasible, is done using lung separation techniques along with wide drainage of both mediastinum and pleural space. When above is not feasible, i.e. if inflammation is substantial and injury is extensive, esophageal exclusion and diversion is the most prudent choice. Cervical esophagostomy is necessary followed by eventual esophageal replacement.[92-94]

Video Assisted Thoracoscopic Surgery (VATS)

Use of VATS is expanding in stable trauma patient for diagnosis and treatment because as compared to thoracotomy, it is less invasive operation, patients experience less pain and less impairment in pulmonary function thus need decreased length of stay in hospital. It is useful for the diagnosis and treatment of continued chest tube bleeding, retained hemothorax, post-traumatic empyema, suspected diaphragmatic injury, persistent air leak, mediastinal injuries. Contraindication to VATS include hemodynamic instability, injuries to heart and great vessel, inability to tolerate one lung ventilation, prior thoracotomy, coagulopathy, indication for emergent thoracotomy or sternotomy. Conversion to thoracotomy may be necessary in few patients.[88,95]

Anesthesia management of VATS requires use of one lung ventilation. Left side double lumen endo-

tracheal tube is preferred to right side DLT because of ease of placement, avoids risk of right upper lobe collapse and stability in lateral decubitus position. Alternatively, right side DLT or brochial blocker technique can be used. Proper tube placement is confirmed with auscultation and visualization using fiberoptic bronchoscope. It is prudent to reconfirm proper position of DLT with FOB after patient is positioned in lateral decubitus position as these tubes may be dislodged during patient's positioning.[96] Single lumen ETT can be advanced into right or left mainstem bronchi with or without fiberoptic guidance. This allows one lung ventilation but does not permit suctioning or application of CPAP to atelectactic lung.

Anesthetic Considerations for Cardiothoracic Trauma

Preoperative anesthetic and critical care management of patient with thoracic trauma is challenging because victim may have multiple life-threatening injuries and may be unstable and time for evaluation and resuscitation before surgery is often inadequate. ATLS protocol provides invaluable guidelines for immediate preoperative assessment and management of patient. Critical ATLS evaluation end point must be addressed prior to induction of anesthesia. Anesthesia plan must be formulated for each patient with full awareness that sudden change in patient's condition may justify modification of proposed plan.

Those patients who on arrival meet criteria for resuscitative thoracotomy (e.g. witnessed cardiac arrest in patient with penetrating chest injury) are taken directly to operation room for simultaneous resuscitation and emergency surgery. In this situation, trauma team including the anesthesiologist assesses and treats patients in operation room. Here, the time for information gathering and patient preparation is quite less and none of data or investigation may be available. Patient who does not meet these criteria is brought to resuscitation suite for initial assessment and management. Results of primary and secondary survey will determine whether the patient is to be transported to computed tomography for further evaluation or to operation room for emergency surgery or surgical intensive care unit for nonoperative management. If the patient requires surgery after secondary survey is completed then appropriate preparation including monitoring and blood product requirement can be provided ahead of time. In semiemergent surgeries all laboratory investigations and radiological imaging films should be reviewed to formulate appropriate plan.

Causes of hemodynamic instability and persistent hypotension in cardiothoracic trauma are:

- Undetected or underestimated blood loss
- Pneumothorax, hemothorax
- Cardiac tamponade
- Air embolism
- Spinal cord injury
- Acidosis, hypothermia, hypoglycemia
- BCI with myocardial contusion and right ventricular dysfunction
- Pre-existing medical disease like cardiomyopathy, valvular heart disease.[97,98]

Hypovolemia is common and requires early and aggressive correction. Most trauma patients will benefit from volume resuscitation prior to initiating general anesthesia and surgery. However, some patients require emergency surgery to locate major source of bleeding in order to achieve hemostasis. Aggressive fluid resuscitation of hypotensive patient may result in rebleeding and subsequent increase in mortality. Thus, permissive arterial hypotension (systolic blood pressure of about 80 to 100 mm Hg) been suggested for bleeding patients in absence of CNS injury to limit hypoperfusion and prevent undesirable increase in blood pressure and thus blood loss. Systolic arterial blood pressure of about 110 to 120 mm Hg is good for patient with traumatic head injury and known hypertensive atherosclerotic cerebrovascular episode patients but arterial hypertension avoided especially in aortic injury.[99,100] Administration of bolus doses of vasopressor medication may transiently elevate BP, but does not improve end organ perfusion which is achieved with volume resuscitation. Vasopressor infusion may be used to reach target blood pressure when fluid volume replacement is adequate. In stable patients, volume status can be guided by urine output, hematocrit, central venous pressure, base deficit and plasma lactate. Most important is to avoid triachy of doom, i.e. hypothermia, coagulopathy and acidosis and is best avoided by maintaining MAP, temperature, tissue perfusion during perioperative period and the priority is for definitive surgical arrest of hemorrhage from major vessels.

Preoxygenation is important because of pre-existing hypoxia and/or respiratory distress. Anesthesia plan is formulated as per specific injuries of patient, urgency of induction and surgical consideration. Understanding of direct action or side effect of each drug is essential for safe intraoperative and postoperative management. No standard sequence or combination of induction and neuromuscular blockade drug is appropriate for all trauma induction situations. It is impossible to prescribe

universal recipe for all trauma patients. Intravenous induction drugs are selected based on patient's neurological and hemodynamic condition. Intravenous induction drug often requires titration with careful attention to patient's response. Reduced dosages of induction drug are frequently required for hypovolemic trauma patients. Patients in cardiac arrest or with GCS of 3 or less, do not require any drug for analgesia, amnesia, hypnosis and muscle relaxation. Victim of trauma with cardiovascular collapse will be hemodynamically too unstable to tolerate even most judiciously titrated general anesthetic. This may lead to intraoperative awareness. Recall experience often causes post-traumatic stress disorder[101] but patient who is in shock or comatose at presentation is less likely to have recall. In order to preserve life at cost of recall, small doses of anesthetic drugs with having property of amnesia, i.e. benzodiazepine or volatile anesthetics should be titrated as tolerated.[102-104]

In many cases, combination of etomidate with rapid onset neuromuscular blocker like succinylcholine or rocuronium will provide the best and safe conditions for induction in trauma patient. Amnesia is primary goal in unstable patient which is provided by IV midazolam 1 to 2 mg increments or scopolamine 0.4 mg as per hemodynamics and fentanyl 0.5 to 2 µgms/kg added for analgesia, as per need. Etomidate is devoid of any significant cardiac depressant effect and has minimal effect on circulation and blood pressure. Etomidate is therefore the preferred induction agent especially in hemodynamically compromised patient with cardiothoracic trauma.[105]

Ketamine is theoretically useful for inducing anesthesia in trauma patient's especially in patients with cardiac tamponade because it produces sympathetic nervous system stimulation with increase in heart rate, blood pressure and cardiac output. However in maximal stress states as in severely hypovolemic patient and/or catecholamine depleted patients, sympathomimetic effect of ketamine does not occur but rather it causes direct myocardial depressant effect with hemodynamic collapse especially when administered as bolus.[106] Thus, IV etomidate 0.1 to 0.2 µg/kg or ketamine 1 mg/kg or Midazolam 0.1 to 0.3 mg/kg are used for induction of hypnosis in titrated doses. If blood pressure is normal or elevated, both propofol in dose 1 to 2 mg/kg or thiopentone in dose 2 to 5 mg/kg can be used for induction but both these induction agents may produce hypotension due to direct myocardial depression and vasodilatation and should be avoided or administered in reduced dosage to unstable trauma patients. Significant alteration in pharmacodynamics (e.g. changes in volume of distribution, protein binding) occurs with trauma, hypothermia, acid base disorder. Therefore, initial titration of drug should be done carefully as drug effect may be unpredictable and profound. In many cases neuromuscular blocking drugs are used to facilitate tracheal intubation. Succinylcholine is used in dose of 1.5 mg/kg, rocuronium in dose of 1.2 mg/kg. Succinylcholine should be avoided due to potential for exaggerated K^+ release and subsequent hyperkalemia induced cardiac arrest in certain high risk patients. This response is typically seen in burn victims and patients with muscle pathology caused by direct trauma, denervation (as with spinal cord injury) or immobilization. Hyperkalemia is not seen in the first 24 hours after these injuries and succinylcholine may be safely used for acute airway management.[107] There is also theoretical risk relating to increasing intracerebral, intraocular, intragastric pressure with use of succinylcholine, however risk benefits ratio must be assessed. Furthermore, a benefit of rapidly obtaining optimum intubating condition with loss of laryngeal reflex often outweighs temporary and usually small increase in pressure.[108] Rocuronium has provided valuable alternative to succinylcholine over last decade with onset of intubating condition within a minute. As with other nondepolarizing neuromuscular blocking drug, rocuronium does not cause increase in intracerebral, intraocular or intragastic pressure. It does not trigger K^+ release and can be used safely in those patients with contraindication for succinylcholine.[109] While maintaining spontaneous respiration during intubation, it is preferred to proceed with slow induction with ketamine or inhaled sevoflurane with cricoid pressure which will enable tracheal intubation without compromising patient safety. Fiberoptic intubation can also be performed under such circumstances.

MAINTENANCE OF ANESTHESIA

Anesthesia can be maintained with inhalational agent or intravenous drug such as propofol and/or with opioid supplementation. Nitrous oxide (N_2O) will cause rapid distention of air containing spaces (due to its higher solubility than nitrogen) and is therefore relatively contraindicated in many trauma patients.[110] Pneumothorax, pneumocephalus, bowel obstruction, air embolus or any hollow viscus injury with pneumoperitoneum will be exacerbated by the use of N_2O. In hemodynamically compromised patient mild myocardial depressant action of N_2O may become evident. All commonly used volatile agents (halothane, isoflurane,

desflurane, sevoflurane) cause significant dose dependant myocardial depression. Their potentially deleterious cardiovascular effects are more pronounced in hypovolemic or otherwise hemodynamically compromised patient.[111,112] Isoflurane, desflurane and sevoflurane maintain cardiac output better than do enflurane and halothane. Hypoxic pulmonary vasoconstriction impaired by volatile agents in dose dependant manner can be clinically significant in patients with respiratory problems requiring one lung ventilation.

Fluids and Blood Transfusion

Major goals of fluid and blood resuscitation in cardiothoracic trauma are normovolemia, normalization of tissue oxygen delivery and control of bleeding. Cardiothoracic trauma patients often present with hemorrhagic shock. Decision regarding transfusion should take into account cardiovascular and pulmonary status, blood loss and hemoglobin count. Hemoglobin is maintained at 7 to 8 gm/dl in trauma patients with no risk of myocardial ischemia, cardiac injuries, and pulmonary contusion whereas in patients with cardiac ischemia, hemoglobin is maintained at 9 to 10 gm/dl. Hematocrit, ABG and coagulation factor evaluation are recommended to guide transfusion of blood products. Blood products are transfused to maintain INR ≤1.5, platelet count ≥ 50000/cmm if no head injury and platelet count ≥ 100000/cmm if head injury, serum fibrinogen ≥ 100 mg/dl.[113] Improvement in organ perfusion and function is judged by increase in pulse pressure, decrease in heart rate, increase in urine output, resolution of lactic acidosis and base deficit, brisk capillary refill, definitive control of hemorrhage, restoration of normothermia, and correction of coagulopathy.[114] Effective fluid warmers are routinely employed to prevent iatrogenic hypothermia.

Intraoperative Problems

Hypotension and hypoxemia are common in patients with cardiothoracic trauma. Utmost care and vigilance is must to diagnose and treat life-threatening complications resulting from cardiothoracic trauma.

Hypoxemia

Intraoperatively hypoxemia results from accidental endobronchial intubation, hypoventilation, airway obstruction, aspiration of foreign material into tracheobronchial tree, pulmonary contusions, pulmonary edema, pulmonary embolism, bronchospasm, hemopneumothorax and problems related to anesthesia circuit or low inspired oxygen. Low cardiac output and low hemoglobin can also produce tissue hypoxia.

Hypotension

Hemorrhage and hypovolemia are the most common causes of hypotension in trauma. Other causes are tension pneumothorax, anaphylaxis, neurogenic shock from high spinal cord injury, cardiogenic shock from blunt cardiac injury, cardiac tamponade, air embolism, valvular rupture, coronary ischemia or infarction, anesthesia drugs.

Hypothermia

Adverse effect of hypothermia is major coagulation derangement by slowing enzymatic rate of clotting factor and reduced platelet function. Other effects are peripheral vasoconstriction, lower cardiac output, metabolic acidosis and impaired immune response. It also causes cardiac arrhythmia if temperature <30°C, impairs citrate, lactate and drug metabolism and causes shift of oxygen dissociation curve to left. Therefore, hypothermia must be prevented by increasing ambient temperature >28°C, using forced air warmer, warming all fluids and blood to 37°C, evaporation from respiratory tract can be prevented by use of active airway humidifier or passive heat and moisture exchanger.[115, 116]

Monitoring

Standard Monitoring

- ECG
- Noninvasive blood pressure monitoring
- Pulse oximeter and end tidal CO_2
- Core temperature monitoring with esophageal/nasopharyngeal temperature probe
- Peripheral nerve stimulator is used to assess degree of neuromuscular block
- Continuous peak inspiratory pressure is monitored for all mechanically ventilated patients. Sudden increase in PIP suggests tension pneumothorax. Risk of barotrauma especially in patient with rib fracture, pulmonary contusion, and pneumothorax is prevented by limiting PIP to 35 cm of H_2O and using pressure controlled mode
- BIS monitoring (bispectral analysis of EEG) is extremely valuable to monitor awareness as most of these patients are hemodynamically too compromised to tolerate sufficient anesthetic agent.[117, 118]

INVASIVE MONITORING

Invasive arterial BP monitoring is indicated in all major cardiothoracic surgeries especially thoracotomy as it gives beat to beat measurement of BP. It also allows for

minimally invasive cardiac output determination using LIDCOTM system. It also allows sampling of ABG, blood chemistry, hematocrit, hemoglobin and coagulation parameters.

Central venous pressure catheter is useful for central venous pressure monitoring. Trend in CVP is useful in patients requiring massive fluid resuscitation and those who require infusion of vasopressors and inotropes. It provides secure access for fluid therapy and drug infusion. It is also useful in diagnosis of cardiac tamponade.

Pulmonary artery catheter is useful for accurate assessment of intravascular volume and cardiac function. Routine insertion of pulmonary artery catheter is controversial due to costs of PA catheter monitoring and risks related to its insertion, use and misuse, e.g. misinterpretation of data.[119]

Transesophageal echocardiography (TEE) is a diagnostic tool for diagnosing aortic injury and cardiac tamponade. TEE is excellent monitor of ventricular performance and volume.[120]

Pain Management in Cardiothoracic Trauma

Cardiothoracic trauma patients are frequently associated with other multiple injuries like head, abdominal or extremity injuries. This necessitates need for monitoring of central and peripheral neurological functions. Thus, it requires flexibility of pain management techniques. Inadequate pain management often characterizes trauma care. This may be due to fear regarding hemodynamic fluctuations and respiratory depression associated with treatment or under-recognition of pain. This adds to physiologic insult to injury due to secondary effects of the neurohumoral stress response. This can lead to increase in the incidence of hypertension, tachycardia, deep venous thrombosis (DVT), pulmonary embolism, immobility, splinting, V/Q mismatch, reduced gastrointestinal motility, water and salt retention, hypoxia and infections.[121] Adequate thoracic pain management by reducing pain improves ventilatory mechanics, allowing patients to breathe more deeply and to cough more effectively. This will decrease the likelihood of atelectasis, thus decrease respiratory infections. This also prevents episodes of hypoxia thus decreases need for mechanical ventilation.[122] Adequate pain management facilitates earlier rehabilitation and may reduce the incidence of long-term chronic pain syndromes.[121] The need for analgesic is also influenced by the schedule of physical therapy especially pulmonary physiotherapy. One of the goals of analgesia, therefore, is the provision of adequate medication to facilitate physical therapy.

Individual patients have widely variant requirements for pain medications, so analgesia must be carefully titrated, ideally in a closely monitored environment such as the postanesthesia care unit (PACU). In acutely traumatized patients, therapy will begin with a maximal dose of acetaminophen and then include a nonsteroidal anti-inflammatory drugs (NSAIDS), then a short-acting narcotic and then a long-acting narcotic. Acetaminophen is well tolerated in doses up to 3 to 4 g/day, but it should be avoided in hepatic or renal failure. NSAID therapy begins with injection ketorolac during the perioperative period. NSAIDs may cause gastrointestinal upset, gastritis and ulceration. Prophylaxis against peptic ulceration is thus indicated. All narcotics cause dose dependent respiratory depression. IV patient-controlled opioid analgesia (IV PCA) is preferred over IM or IV infusions as pain and over sedation cycles are reduced using this technique that allows for immediate delivery and self titration of pain medication. Overdosing is extremely rare because of the self-regulating nature of the device. This technique also is less demanding on nursing resources. Injection morphine or fentanyl is a common choice and morphine when used in dosage of 0.05 to 0.1 mg/kg, the onset is within 1 to 2 min when given IV and 10 to 30 min when given IM the duration is 4 to 5 hours. The side effects are nausea, vomiting, respiratory depression, hypotension, histamine release. Fentanyl used in dosages 0.5 to 1 μg/kg has onset in < 1 min, duration 0.5 to 1 hour and side effects are same as morphine but it causes less histamine release, hence less hypotension.

Pain relief can also be achieved by regional anesthesia like intercostals nerve block to alleviate rib fracture pain but limitation with this technique is relief of pain is temporary (6-12 hours), there is also risk of pneumothorax and risk of local anesthetic toxicity. Interpleural catheter has a risk of local anesthetic toxicity and pain relief achieved is not constant.[123,124] Epidural analgesia provides excellent pain relief for patients with multiple rib fractures and thus helps facilitate an effective cough. Quality of pain relief reported by the patient is also superior in patients receiving epidural analgesia.[125] Epidural analgesia can be delivered in intermittent boluses or as continuous infusion or as patient-controlled epidural analgesia (PCEA). Benefits of epidural analgesia include improved vital capacity, functional residual capacity, airway resistance, and dynamic lung compliance.[125] Patients receiving continuous epidural analgesia (narcotics with or without local anesthetics) have been shown to have decreased ventilator days, shorter ICU stays, and

shorter hospitalizations and decreased incidence of tracheostomy.[126] Epidural analgesia leads to a lower incidence of respiratory depression, utilizes smaller doses of narcotics, allows earlier ambulation and discharge when compared to other modes of analgesia.[121,127,128] Mortality rate, frequency of respiratory infection is less with epidural.

Limitations of this technique are:

- Insufficient time for insertion of epidural catheter (e.g. unstable or emergent presentation)
- Inadequate positioning for catheter placement (uncleared spine/pelvic fracture)
- Use of local anesthetic may be inappropriate in patient with unstable hemodynamics, patients with associated spinal cord injuries and patients with coagulopathy
- Other contraindications to its use are septicemia, untreated bacteremia, alteration in patient's mental status.

Drug selection is determined by lipid solubility, catheter insertion site, pain location. Hydrophilic drug like morphine has delayed onset, prolonged duration and increased incidence of respiratory depression. Lipid soluble agent such as fentanyl has faster onset of action, shorter duration of analgesia and fewer respiratory side effects. Complications of epidural opioids are pruritus, sedation, nausea, vomiting, urinary retentions, apnea and seizures. Rare complication are epidural hematoma or abscess with spinal cord compression which must be recognized and requires surgical evaluation immediately if paraplegia is to be prevented.[129,130] Potential for epidural hematoma is increased if coagulation is impaired at time of insertion or removal of catheter especially if patient is on low molecular weight heparin, warfarin, antiplatelet drugs.[131]

Postoperative Considerations

Rapid termination of general anesthesia is desirable particularly in patients with altered level of consciousness or with evidence of traumatic brain injury before surgery. Change in mental status from preoperative baseline is indication for repeat CT and search for possible metabolic or toxic derangement.

Indications for Postoperative Ventilation

- Patients with unfavorable GCS prior to surgery require postoperative mechanical ventilation for neurosurgical purpose.
- Victims with significant cardiothoracic trauma remain intubated and ventilated postoperatively because of the obvious concern about patient's breathing reserve capacity.
- There is always a risk of postoperative respiratory failure in patients with fracture ribs and lung contusion.
- Patient with massive blood loss and fluid shifts may need to remain intubated postoperatively.
- Surgeon's concern about doubtful postoperative course or planned reoperation.

Whenever, there is significant amount of doubt, it is safer to keep patient intubated rather than premature extubation in tenuous cardiopulmonary state or in setting of possible airway swelling, compromising patient's outcome. Usually 12 to 24 hours of support allows confirmation of successful resuscitation, surgical repair, hemodynamic stability, titration of analgesia, resolution of intoxication. Many patients can get extubated easily and safely by this time.

Mortality Following Cardiothoracic Trauma

Mortality has been found to be higher with blunt injuries as compared to penetrating injuries. ARDS, multiple-organ system failure and sepsis are the major late causes of death in cardiothoracic trauma.[1, 2]

REFERENCES

1. Locicero J, Mattox KL. Epidemiology of Chest Trauma. Surg Clin North Am 1989;69:15-6.
2. Shor RM, Crittenden M, Indeck M, et al. Blunt Thoracic Trauma. Ann Surg 1987;206:200-5.
3. Engelhardt S, Hoyt D, Coimbra R, et al. The 15-year evolution of an urban trauma center: What does the future hold for the trauma surgeon? J Trauma 2001;51:633-8.
4. Besson A, Saegesser F. Color atlas of chest trauma and associated injuries. New Jersey: Oradell, 1983;12-4.
5. Lockey D, Crewdson K, Davies G. Traumatic cardiac arrest: Who are the survivors? Ann Emerg Med 2006; 48(3):240.
6. Devitt JH, McLean RF, Koch JP. Anesthetic management of acute blunt thoracic trauma. Can J Anaesth 1991;38:506-10.
7. American College of Surgeons Committee on Trauma. Advanced trauma life support program for physicians. Student and Instructor Manual, Chicago: American College of Surgeons, 2004.
8. Devitt JH, Boulanger BR. Lower airway injuries and anaesthesia. Can J Anaesth 1996;43:148-59.
9. Smith CE, Walls RM, Lockey D, Kuhnigk H. Advanced airway management and use of anesthetic drugs. In: Grande CM, Soreide E, (Edn). The International Textbook on Prehospital Trauma Care. New York: Marcel Dekker, 2001;203-53.
10. Smith CE. Rapid sequence intubation in adults: indications and concerns. Clin Pulm Med 2001;8:147-65.
11. Stene JK, Grande CM, Barton CR. Airway management for the trauma patient. In: Stene JK, Grande CM, (Edn).

Trauma Anesthesia. Baltimore: Williams & Wilkins, 1991;64-99.
12. Wilson WC. Trauma airway management: As a difficult airway algorithm modified for trauma—and five common trauma intubation scenarios. ASA Newsl 2005;69(11).
13. Schaefer SD. The treatment of acute external laryngeal injuries. Arch Otolaryngol Head Neck Surg 1991;117:35-9.
14. Leopold DA. Laryngeal trauma. Arch Otolaryngol Head Neck Surg 1983;109:106-8.
15. Meredith JW, Riley RD. Injury to the esophagus, trachea, and bronchus. In: Mattox KL, Feliciano DV, Moore EE, (Edn). Trauma. 4th edn. New York: McGraw-Hill; 2000;473-82.
16. Jewett BS, Shockley WW, Rutledge R. External laryngeal trauma analysis of 392 patients. Arch Otolaryngol Head Neck Surg 1999;125:877-80.
17. Balci AE, Eren N, Eren S, Ülkü R. Surgical treatment of post-traumatic tracheobronchial injuries: 14-year experience. Eur J Cardiothorac Surg 2002;22:984-9.
18. Johnson G. Traumatic pneumothorax: Is a chest drain always necessary? J Accid Emerg Med. 1996;13(3): 173-4.
19. Sirmali M, Türüt H, Topçu S, Gülhan E, Yazici U, Kaya S, Tatepe A. Comprehensive analysis of traumatic rib fractures: morbidity, mortality and management. Eur J Cardiothorac Surg 2003;24(1):133-8.
20. Mayberry JC, Trunkey DD. The fractured rib in chest wall trauma. Chest Surg Clin North Am 1997;7:239-61.
21. Ziegler DW, Agarwal NN. The morbidity and mortality of rib fractures. J Trauma 1994;37:975-9.
22. Bulger EM, Arneson MA, Mock CN, Jurkovich GJ. Rib fractures in the elderly. J Trauma 2000;48(6):1040-7.
23. Livingston DH, Shogan B, John P, Lavery RF . CT diagnosis of rib fractures and the prediction of acute respiratory failure. J Trauma 2008;64(4):905-11.
24. Yanping D, Charles ES, John JC. Cardiothoracic Trauma. In: William CW, Christopher MG, David H. (Eds). Trauma: Emergency resuscitation, perioperative anesthesia, surgical management. NY 2007;25: 469-99.
25. Cohn SM. Pulmonary contusion: review of the clinical entity. J Trauma 1997;42(5):973-9.
26. Easter A. Management of patients with multiple rib fractures. Am J Crit Care 2001;10(5):320-7.
27. Patel N, Smith CE. Pain management in trauma. Anesthesiol Clin North Am 1999;17(1):295-309.
28. Moon MR, Luchette FA, Gibson SW. Prospective, randomized comparison of epidural versus parenteral opioid analgesia in thoracic trauma. Ann Surg 1999; 229:684-92.
29. Schweiger JHW. The pathophysiology, diagnosis and management strategies for flail chest injury and pulmonary contusion: A review (review course lectures). Anesth Analg 2001;86-93.
30. Beltrame F, Lucangelo U, Gregori D, et al. Noninvasive positive pressure ventilation in trauma patients with acute respiratory failure. Monaldi Arch Chest Dis 1999;54:109-14.
31. Amato MBP, Barbas CSV, Medeiros DM, et al. Effect of a protective-ventilation strategy on mortality in the respiratory distress syndrome. N Engl J Med 1998;338:347-54.
32. Karmy-Jones R, Jurkovich J, Shatz DV, et al. Management of traumatic lung injury: a western trauma association multicenter review. J Trauma 2001;51:1049-53.
33. McGhee A, Swinton S, Watt M. Use of autologous transfusion in the management of acute traumatic hemothorax in the accident and emergency department. J Accid Emerg Med 1999;16:451-2.
34. Wall MJ, Storey JH, Mattox KL. Indications for thoracotomy. In: Mattox KL, Feliciano DV, Moore EE, (Eds). Trauma. 4th edn. New York: McGraw-Hill, 2000;473-82.
35. Rozycki GS, Feliciano DV, Ochsner MG, et al. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. J Trauma 1999; 46:543-51.
36. Stern A, Dronen SC, Birrer P, Wang X. Effect of blood pressure on hemorrhagic volume in a near-fatal hemorrhage model incorporating a vascular injury. Ann Emerg Med 1993;22:155-63.
37. Owens TM, Watson WC, Prough DS. Limiting initial resuscitation of uncontrolled hemorrhage reduces internal bleeding and subsequent volume requirements. J Trauma 1995;39:200-9.
38. Burris D, Rhee P, Kaufmann C. Controlled resuscitation for uncontrolled hemorrhagic shock. J Trauma 1999;46: 216-23.
39. Wall MJ, Hirschberg A, Lemaire SA, Holcomb J, Mattox K. Thoracic aortic and thoracic vascular injuries. Surg Clin North Am 2001;81(6):1375-93.
40. Ivatury RR. The injured heart. In: Mattox KL, Feliciano DV Moore EE (Eds). Trauma. 4th edn. New York: McGraw-Hill 2000;545-8.
41. Rozycki GS, Feliciano DV, Ochsner MG. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. J Trauma 1999; 46:543-51.
42. Buckman RF, Buckman PD, Badellino MM. Heart. In: Ivatury RR, Cayten CG (Eds). The Textbook of Penetrating Trauma. Baltimore: Williams & Wilkins 1996:499-511.
43. Thourani VH, Feliciano DV, Cooper WA, et al. Penetrating cardiac trauma at an urban trauma center: a 22 years perspective. Am Surg 1999; 65:811-6.
44. Lim R, Gill IS, Temes RT, Smith CE. The use of adenosine for repair of penetrating cardiac injuries: A novel method. Ann Thorac Surg 2001;71:1714-5.
45. Lim R, Gill IS, Temes RT, Smith CE. Adenosine for repair of penetrating cardiac injuries: reply to the editor. Ann Thorac Surg 2002;73:342.
46. McAleese P, Young S, Cohen M. Penetrating cardiac injuries. Am Surg 1999;65:444-8.
47. Southerland GR, Calvin JE, Driedger AA. Anatomic and cardiopulmonary responses to trauma with associated blunt chest injury. J Trauma 1981;21:1-13.
48. Robert E, de ja Coussaye JE, Aya AGM. Mechanism of ventricular arrhythmias induced by myocardial contusion. Anesthesiol 2000;92:1132-43.
49. Barach P. Perioperative anaesthetic management of patients with cardiac trauma. Anesthesiol Clin North Am 1999;17(1):197-209.

50. Cooney RN, Haluck R, Smith JS, Mucha P. Blunt cardiac injury: is there a standard of care? Eur J Emerg Surg Intensive Care 1997;20:82-6.
51. Mattox KL, Flint LM, Carrico CJ. Blunt cardiac injury. J Trauma 1992;33:649-50.
52. Feliciano DV. Advances in the diagnosis and treatment of thoracic trauma. Surg Clin North Am 1999;79(6):1417-29.
53. Nagy KK, Krosner SM, Roberts RR, Joseph KT, Smith RF, Barrett J. Determining which patients require evaluation for blunt cardiac injury following blunt chest trauma. World J Surg 2001;25:108-111.
54. Orliaguet G, Ferjani M, Riou B. The heart in blunt trauma. Anesthesiol 2001;95:544-8.
55. Flancbaum L, Wright J, Siegel JH. Emergency surgery in patients with post-traumatic myocardial contusion. J Trauma 1986;26:795-803.
56. Rhee PM, Bridgeman A, Acosta JA. Survival after emergency department thoracotomy: review of published data from the past 25 years. J Am Coll Surg 2000; 190(3):288-98.
57. Boyd M, Vanek V, Bourguet C. Emergency room resuscitative thoracotomy: when is it indicated? J Trauma 1992;33(5):714-21.
58. Powell DW, Moore EE, Cothren CC, Cielsa DJ, Burch JM,Moore JB, Johnson JL. Is emergency department resuscitative thoracotomy futile care for the critically injured patient requiring prehospital cardiopulmonary resuscitation? J Am Coll Surg 2004;199(2):211-5.
59. Kish G, Kozloff L, Jospeph WL, Adkins PC. Indications for early thoracotomy in the management of chest trauma. Ann Thorac Surg 1976;22(1):23-8.
60. Zieren HU, Muller JM, Pichlmaier H. Thoracotomies in thoracic injuries–indications and results. Langenbecks Arch Chir 1991;376(6):330-4.
61. Wall MJ, Villavicencio RT, Miller CC. Pulmonary tractotomy as an abbreviated thoracotomy technique. J Trauma 1998;45:1015-23.
62. Rupture of the thoracic aorta by roadside impacts in road traffic and other collisions: further angiographic observations and preliminary autopsy findings. J rauma 1993;35:363-7.
63. Kram HB, Appel PL, Wohlmuth DA, Shoemaker WC. Diagnosis of traumatic thoracic aortic rupture: a 10-year retrospective analysis. Ann Thorac Surg 1989;47:282-6.
64. Lee J, Harris JH, Duke SH, Williams JS. Noncorrelation between thoracic skeletal injuries and acute traumatic aortic tear. J Trauma 1997;43:400-4.
65. Vignon P. Comparison of multiplane transesophageal echocardiography and contrast-enhanced helical CT in the diagnosis of blunt traumatic cardiovascular injuries. Anesthesiol 2001;94:615-22.
66. Chen MY, Miller PR, McLaughlin C. The trend of using computed tomography in the detection of acute thoracic aortic and branch vessel injury after blunt thoracic trauma: single center experience over 13 years. J Trauma 2004; 56(4):783-5.
67. Parker MS, Metheson TL, Rao AV. The role of helical CT in the evaluation of potentially acute thoracic aortic injuries. Am J Roentgenol 2001;176:1267-72.
68. Ben-Menachem Y. Assessment of blunt aortic-brachiocephalic trauma: should angiography be supplanted by transesophageal echocardiography? J Trauma 1997;42:969-72.
69. Cohn SM, Burns GA, Jaffe C, Milner KA. Exclusion of aortic tear in the unstable trauma patient: The utility of transesophageal echocardiography. J Trauma 1995; 39;1087-90.
70. Kearney PA, Smith DW, Johnson SB. Use of transesophageal echocardiography in the evaluation of traumatic aortic injury. J Trauma 1993;34:696-701.
71. Cinnella G, Dambrosio M, Brienza N. Transesophageal echocardiography for diagnosis of traumatic aortic injury: an appraisal of the evidence. J Trauma 2004;57(6):1246-55.
72. Cowley RA, Turney SZ, Hankins JR. Rupture of thoracic aorta caused by blunt trauma. J Thorac Cardiovasc Surg 1990;100:652-61.
73. Read RA, Moore EE, Moore FA, Haenel JB. Partial left heart bypass for thoracic aortic repair. Survival without paraplegia. Arch Surg 1993;128:746-50, discussion 750-752.
74. Galli R, Pacini D, Di Bartolomeo R, Fattori R. Surgical indications and timing of repair of traumatic ruptures of the thoracic aorta. Ann Thorac Surg 1998;65:461-4.
75. Devitt JH, McLean RF, McLellan BA. Perioperative cardiovascular complications associated with blunt thoracic trauma. Can J Anaesth 1993;40:197-200.
76. O'Connor CJ, Rothenberg DM. Anesthetic considerations for descending thoracic aortic surgery: part II. J Cardiothorac Vasc Anes 1995;9:734-47.
77. Karmy-Jones R, Carter YM, Nathens A. Impact of presenting physiology and associated injuries on outcome following traumatic rupture of the thoracic aorta. Am Surg 2001;67:61-6.
78. Fujikawa T, Yukioka T, Ishimaru S. Endovascular stent grafting for the treatment of blunt thoracic injury. J Trauma 2001;50:223-9.
79. Peterson BG, Matsumura JS, Morasch M. Percutaneous endovascular repair of blunt thoracic aortic transection. J Trauma 2005;59(5):1062-5.
80. Hirose H, Gill IS, Malangoni MA. Nonoperative management of traumatic aortic injury. J Trauma 2006; 60:597-601.
81. Pate JW, Gavant ML, Weiman DS. Traumatic rupture of the aortic isthmus: Program of selective management. World J Surg 1999;23:59-63.
82. Murray JA, Demetriades D, Cornwell EE. Penetrating thoracoabdominal trauma: The incidence and clinical presentation of diaphragm injuries. J Trauma 1997;43:624-6.
83. Haciibrahimoglu G, Solak O, Olcmen O. Management of traumatic diaphragm rupture. Surg Today 2004;34:111-4.
84. Ahmad J, Beattie GC, Kennedy R, Kennedy JA, Clements WDB. Penetrating trauma to the junctional zone needs aggressive management. BMJ 2007;334(7587):257-8.

85. Demetriades D, Kakoyiannis S, Parehk D. Penetrating injuries to the diaphragm. Br J Surg 1988;75:824-6.
86. Lowdermilk GA, Naunheim KS. Minimal access surgery: Thoracoscopic evaluation and treatment of thoracic trauma. Surg Clin North Am 2000;80(5):14-36.
87. Simpson J, Lobo DN, Shah AB, Rowlands BJ. Traumatic diaphragmatic rupture: associated injuries and outcome. Ann R Coll Surg Engl 2000;82:97-100.
88. Manlulu AV, Lee TW, Thung KH, Wong R, Yim APC. Current indications and results of VATS in the evaluation and management of hemodynamically stable thoracic injuries. Eur J Cardiothorac Surg 2004; 25:1048-53.
89. Juan A Asensio, Esteban Gambaro. Esophageal Injury. In: Demetrios Demetriades, Juan A. Asensio (Eds) Textbook of trauma management, 2000;23: 249-53.
90. Vezina D, Lessard MR, Bussieres J, Topping C, Trepanier CA. Complications associated with the use of the esophageal tracheal Combitube. Can J Anaesth 1998; 45:76-80.
91. Branthwaite MA. An unexpected complication of the intubating laryngeal mask. Anesthesia 1999;54:166-7.
92. Richard GY, Alexander G Obeid, Farouck N, Horan, Paul D. Traumatic Esophageal Injuries: 12-year Experience at Henry Ford Hospital,YAP. The Journal of Trauma, 1994;24(7);623-5.
93. Asensio JA, Chahwan S, Forno W, MacKersie R. Penetrating esophageal injuries: multicenter study of the American Association for the Surgery of Trauma. J Trauma 2001 Feb; 50(2):289-96.
94. Weiman DS, Walker WA, Brosnan KM, Pate JW, Fabian TC. Discussion 849-50. Noniatrogenic esophageal trauma. Ann Thorac Surg 1995;59(4):845-9.
95. Potaris K, Migos P, Gakidid I. Role of video-assisted thoracic surgery in the evaluation and management of hemodynamically stable thoracic injuries. Interactive Cardiovasc Thorac Surg 2005;4(4):292-4.
96. Wilson WC, Benumof JL. Anesthesia for Thoracic Surgery. In: Miller RD, Lee AF, Roger AJ, John JS, Jeanine WK, William LY, (Eds). Miller's Anesthesia. 6th edn. NY: Churchill Livingstone 2004; 49:1847-1940.
97. Stene JK, Smith CE, Grande CM. Evaluation of the trauma patient. In: Longnecker DE, Tinker JH, Morgan GE, edn. Principles and Practice of Anesthesiology. Chapter 31, 2nd edn. Mosby: St. Louis l997:572-600.
98. Grande CM, Smith CE, Stene JK. Trauma anesthesia. In: Longnecker DE, Tinker JH, Morgan GE (Eds). Principles and Practice of Anesthesiology. Volume 2, 2nd edn. Mosby: St.Louis 1997;39:2138-64.
99. Dutton RP, Mackenzie CF, Scalea TM. Hypotensive resuscitation during active hemorrhage: Impact on in-hospital mortality. J Trauma 2002;52:1141-6.
100. Siegel JH, Gens DR, Mamantov T. Effect of associated injuries and blood volume replacement on death, rehabilitation needs, and disability in blunt traumatic brain injury. Crit Care Med 1991;19:1252-65.
101. Jones JG. Perception and memory during general anesthesia. Br J Anaesth 1994;73:31-7.
102. Bogetz MS, Katz JA. Recall of surgery for major trauma.Anesthesiol 1984;61:6-9.
103. Sandin RH, Enlund G, Samuelsson P, Lennmarken C. Awareness during anesthesia: a prospective case study. Lancet 2000;355:707-1.
104. Richard PD, Maureen McCunn. Anesthesia for Trauma. In: Miller RD, Lee AF, Roger AJ, John JS, Jeanine WK, William LY, (Eds). Miller's Anesthesia. 6th edn. NY, Churchill Livingstone 2004;63:2451-96.
105. Gooding JM, Corssen G. Effect of etomidate on the cardiovascular system. Anesth Analg 1977;56:717-9.
106. Waxman K, Shoemaker WC, Lippmann M. Cardiovascular effects of anesthetic induction with ketamine. Anesth Analg 1980;59:355-8.
107. Gronert GA, Theye RA. Pathophysiology of hyperkalemia induced by succinylcholine. Anesthesiol 1975;43:89-99.
108. Kovarik WD, Mayberg TS, Lam AM. Succinylcholine does not change intracranial pressure, cerebral blood flow velocity, or the electroencephalogram in patients with neurological injury. Anesthesiol, 1994;78:469-73.
109. Andrews JI, Kumar N, van den Brom RH. A large simple randomized trial of rocuronium versus succinylcholine in rapid-sequence induction of anesthesia along with propofol. Acta Anaesthesiol Scand 1999;43(1):4-8.
110. Eger EI, Saidman LJ. Hazards of nitrous oxide anesthesia in bowel obstruction and pneumothorax. Anesthesiol 1965;26:61-6.
111. Ebert TJ. Cardiovascular and autonomic effects of sevoflurane. Acta Anaesthesiol Belg 1996;47:15-21.
112. Azad SS, Bartkowski RR,Witkowski TA. A comparison of desflurane and isoflurane in prolonged surgery. J Clin Anesth 1993;5:122-8.
113. Homer Tien, Bartolomeu Nascimento, Jr. Jeannie Callum, Sandro Rizoli. An approach to transfusion and hemorrhage in trauma: current perspectives on restrictive transfusion strategies. Can J Surg 2007;50(3):202-9.
114. Ivatury RR, Simon RJ. Assessment of tissue oxygenation (evaluation of the adequacy of resuscitation). In: Ivatury RR Cayten CGC, (Eds). The Textbook of Penetrating Trauma. Baltimore: Williams & Wilkins 1996;935.
115. Smith CE, Yamat RA. Avoiding hypothermia in the trauma patient. Curr Opin Anaesthesiol 2000;13:167-74.
116. Smith CE, Patel N. Hypothermia in adult trauma patients: Anesthetic considerations. Part II. Prevention and treatment. Am J Anesthesiol 1997;24:29-36.
117. Sebel PS, Lang E, Rampil I. A multicenter study of bispectral EEG analysis for monitoring anesthetic effect. Anesth Analg 1997;84:891-9.
118. Glass PSA, Bloom M, Kearse L. Bispectral analysis measures sedation and memory effects of propofol, midazolam, isoflurane, and alfentanil in healthy volunteers. Anesthesiol 1997;86:836-47.
119. Varon AJ. Arterial, central venous and pulmonary artery catheters. In: Civetta JM, Taylor RW, Kirby RR, (Eds). Critical Care. 3rd edn. Philadelphia: Lippincott-Raven, 1997;847-65.

120. Otto CM. Textbook of Clinical Echocardiography. 2nd edn. St. Louis: WB Saunders 2000;59-79.
121. Patel N, Smith CE. Pain management in trauma. Anesthesiol Clin North Am 1999;17(1):295-309.
122. Simon BJ, Cushman J, Barraco R. For the EAST Practice Management Guidelines Work Group: pain management guidelines for blunt thoracic trauma. J Trauma 2005; 59(5):1256-67.
123. Karmakar, Manoj K, Ho, Anthony MH. Review Article Acute Pain Management of Patients with Multiple Fractured Ribs. The Journal of Trauma: Injury, Infection, and Critical Care: March 2003 - Volume 54 - Issue 3-615-25.
124. Bachmann-Mennenga B, Biscoping J, Kuhn DF, Schürg R, Ryan B, Erkens U, Hempelmann G. Intercostal nerve block, interpleural analgesia, thoracic epidural block or systemic opioid application for pain relief after thoracotomy? Eue J Cardiothorc Surg 1993;7(10):561.
125. Mackersie RC, Shackford SR, Hoyt DB. Continuous epidural fentanyl analgesia: Ventilatory function improvement with routine use in treatment of blunt chest trauma. J Trauma 1987;27:1207-12.
126. Rankin AR, Comber RE. Management of fifty cases of chest injury with a regimen of epidural bupivacaine and morphine. Anaesth Intensive Care 1984;12:311-4.
127. Dittman M, Steenblock U, Kranzlin M. Epidural analgesia or mechanical ventilation for multiple rib fractures. Intensive Care Med 1982;8:89-92.
128. Moon MR, Luchette FA, Gibson SW, Crews J, Sudarshan G, Hurst JM. Prospective, randomized comparison of epidural versus parenteral opioid analgesia in thoracic trauma. Ann Surg 1999;229(5):684.
129. Scherer R, Schmutzler M, Giebler R. Complications related to thoracic epidural analgesia: a prospective study in 1071 surgical patients. Acta Anaesthesiol Scand 1993;37:370-4.
130. Tanaka K,Watanabe R, Harada T, Dan K. Extensive application of epidural anesthesia and analgesia in a university hospital: incidence of complications related to technique. Reg Anesth 1993;18:34-8.
131. Terese T, Horlocker, Honorio T Benzon, David L, Brown F, Kayser Enneking, John A Heit, Michael F Mulroy, Richard W Rosenquist. Regional Anesthesia in the Anticoagulated Patient-Defining the Risks. Developed from: American Society of Regional Anesthesia and Pain Medicine Consensus Conference held during the Annual Spring Meeting on Regional Anesthesia, Chicago, IL, 2002;25-28.

29 Anesthesia for Abdominal Trauma

Hemangi S Karnik, Nazmeen I Sayed

KEY POINTS

- Uncontrolled concealed hemorrhage is often the cause of death immediately following abdominal trauma.
- Immediate tracheal intubation and controlled ventilation are needed for patients in hypovolemic shock to improve oxygen delivery to vital organs.
- History taking should not delay resuscitation in unstable patients.
- In hemodynamically unstable patients, focussed assessment sonography in trauma (FAST) is the investigation of choice.
- In hemodynamically unstable patients, a positive FAST result indicates an emergency laparotomy and an indeterminate FAST result leads to a diagnostic peritoneal lavage (DPL) or a CT scan.[12]
- A nonworking pulse oximeter may indicate inadequate resuscitation.
- Shock decreases the size of the central compartment and systemic clearance for drugs, resulting in increased plasma concentrations.
- Adverse effects of various anesthetic agents in a hemorrhagic patient can be reduced by providing resuscitation to restore the hemodynamic status to near normal.
- Rapid Infusion devices should be kept available in all operation theaters where trauma cases are taken up.
- Coagulation abnormalities are imminent in patients with major blood loss due to loss of coagulation factors and is further aggravated due to hemodilution post-fluid resuscitation.
- If a patient has multiple injuries requiring surgical management, in unstable status, only life-threatening surgeries should be undertaken with ongoing resuscitation.
- Damage control surgery is an effort to salvage patient, who are at the end of their physiological reserve.
- Damage control surgery can reduce the mortality in critically injured patients by 50 percent.
- Acute abdominal compartment syndrome (ACS) may occur either due to primary abdominal trauma or secondary reperfusion of tissues after prolonged ischemia.

INTRODUCTION

Abdominal trauma is one of the major causes of early mortality following severe trauma. With increasing incidence of road traffic accidents and violent assaults, there is a rise in blunt as well as penetrating abdominal trauma cases. Unlike injuries to many other regions, abdominal injuries can often remain unrecognized due to its concealed nature. Uncontrolled concealed hemorrhage is often the cause of death immediately following abdominal trauma. Early identification, resuscitation and timely surgical intervention may be life-saving in the immediate phase and can prevent many systemic consequences responsible for multiorgan failure.

Understanding the mechanism of injury helps to suspect primary organ of injury and associated injuries. To be able to do so, one needs to know the anatomy relevant to trauma and common abdominal injuries.

Relevant Anatomy

There are three basic regions of the abdomen: the peritoneal cavity with its intrathoracic and intra-abdominal components, the retroperitoneum and the pelvic region.[1] The intra-abdominal part of the peritoneal cavity is called 'the true abdomen'. The abdomen is separated from the chest cavity by the diaphragm, which may ascend as high as third thoracic vertebra. The intrathoracic component lies beneath the rib cage and includes the diaphragm, liver, spleen and stomach. Liver and spleen lie in 'true abdomen' during inhalation. Any injury to lower thoracic cage, i.e. fractures of lower ribs or penetrating trauma can have

associated trauma to these organs. The intra-abdominal component contains hollow viscera like stomach, small and large bowels, omentum, gravid uterus and dome of bladder when full. The retroperitoneal part contains kidneys, great vessels, colon, pancreas and part of duodenum. This region can hold large amount of blood loss without being apparent, often seen along with spine or pelvic trauma. Pelvic cavity is surrounded by the bony pelvis and contains the urogenital organs. These organs get involved in pelvic fracture.

Trauma to abdomen can be broadly classified into blunt or penetrating injuries.

Blunt abdominal trauma: Blunt abdominal trauma may involve compression injuries or deceleration injuries. Compression of the abdominal cavity against a fixed object results in rapid increase in intraluminal pressure, leading to bowel rupture and tears or hematomas of solid organs.[2] Improperly worn seat belts are associated with duodenal or jejunal perforations along with pancreatic injuries as these organs get compressed against the belt and the vertebral column. Injuries caused by bicycle handlebar or animal kick can also result in small bowel perforation.

Deceleration forces as seen with high speed collisions cause shearing and stretching of junctions between fixed and mobile structures. These forces typically result in injury to the mesentery, large vessels and solid organs near the capsule.[2] Spleen and liver are most commonly injured organs during blunt abdominal trauma.

Unlike penetrating abdominal injury where the penetrating object or its wound is visible, blunt abdominal trauma can be very deceiving and may remain undiagnosed.

Penetrating abdominal trauma: This may occur in gunshot injuries, vehicular accidents, assaults, blasts or building collapse. It can be of three types:

- Low-energy penetrating injuries, resulting from a knife, low-velocity bullet or fall on sharp object
- Medium energy injuries
- High-energy injuries.

Medium and high-energy penetrating injuries are caused by missile fragments and higher velocity bullets.

In low-energy penetrating trauma, injuries are limited to the tract created by the wounding object. In high-energy penetrating injury, destruction occurs to a much larger extent. With gunshot injury the bullet causes primary injury by the bullet along with a cavitatory effect greatly expanding the zone of injury.[3] In very high velocity gunshot wounds, there is also a forward or leading edge shock wave, which contributes to the injury. Penetrating abdominal trauma cases are predominantly associated with bowel, liver and major vascular injuries.

Specific Organ Injuries

Liver Injuries

The liver is the most commonly injured organ from blunt abdominal trauma, and is the most common abdominal injury resulting in about 10 percent mortality[4] usually due to massive blood loss. Usually associated with fractures of the right lower ribs. Patient may have an elevated right hemidiaphragm, right pleural effusion, right pneumothorax and right upper quadrant tenderness. Nonoperative management is now the initial treatment of choice for hemodynamically stable patients with isolated blunt liver trauma, with nearly 95 percent of success rates.[2] Absence of continued transfusion requirement is another criteria for nonoperative management. Though penetrating wounds in liver region has been treated conservatively, this is not accepted by all.[5] However in unstable patients, immediate exploration is usually indicated. Surgical procedures are hepatorrhaphy, occlusion of portal triad by Pringle maneuver, anatomical hepatic resection or selective hepatic artery ligation.[4] For uncontrollable hemorrhage, tight packing of perihepatic region may be needed along with staged surgeries as damage control (see below-damage control surgery). The bile leak can be treated by surgery or by percutaneous drainage or by stenting. Postoperative complications include rebleeding, biliary leak, intrahepatic or perihepatic abscesses, septicemia and hemobilia. Hemobilia classically presents with right hypochondriac pain, jaundice and hemorrhage. Treatment is angiogram and embolization. Angiographic embolization of bleeding vessel has revolutionalized the nonoperative management.

Splenic Injuries

The spleen may be involved in 25 percent of blunt abdominal injuries.[4] After the liver, the injured spleen is the next important cause of life-threatening hemorrhage following blunt trauma. It is firmly connected to the retroperitoneal space by the splenorenal and splenophrenic ligaments and thus can lead to retroperitoneal hematoma. The spleen receives 5 percent of cardiac output and has an open microcirculation without endothelium. Therefore, it can bleed torrentially when injured.

Management

- Nonoperative treatment is successful in 90 percent of children and 65 to 75 percent of adults

- Angiography and embolization procedures help in nonoperative management
- Severe lacerations and shattered spleen in unstable patients need operative management.

Smaller tears can be managed either conservatively if patient is stable or by surgery using local hemostatic agents, cautery and suturing. In unstable patients with severe splenic injury, immediate splenectomy is indicated. For large tears or shattered spleen, attempts for splenic preservation may pose life-threatening bleeding and other postoperative complications. Postoperative thrombocytosis and overwhelming postsplenectony infection (OPSI) are well-known following splenectomy. OPSI syndrome is suspected by acute onset flu-like symptoms, progressing rapidly to respiratory and renal failure, cardiovascular collapse and finally death within hours of onset, if appropriate treatment is not effectively instituted. The incidence of infections can be minimized by giving H. influenza, pneumococcal and meningococcal vaccines.[4]

Stomach and Small Bowel Injuries

They are more common in penetrating injuries than in blunt trauma. Duodenal injuries are associated with handlebar, steering wheel or seat belt injuries as these objects compress the duodenum against the vertebral column. Many times they are associated with spinal injuries. Majority of the cases have associated injuries commonly involving liver, pancreas, small bowel, and colon. Immediate surgery is indicated for hemorrhage control and suturing of perforation.

Genitourinary Trauma

This occurs in about 10 percent of abdominal trauma patients.[6] In blunt trauma patients with liver or spleen tear, associated kidney injury on same side should be suspected. The presentation varies depending on the type and severity of trauma. Hematuria, tenderness or wound in flank are common signs. Penetrating trauma patients are likely to undergo surgery without CT scan. Surgical procedures include renal exploration with or without nephrectomy, ureteral reconstruction, bladder tear suturing. Major hemorrhage and postoperative urinary leak are common complications.

Vascular Injuries

These are seen more frequently with penetrating abdominal injuries. Underlying vessel injury should be suspected as per the location of the wound. The patient presentation may range from stable to more frequent cardiovascular collapse. Stable patients may undergo CT scan and unstable patients should be taken directly to operating room, without removing the foreign object, if still present. Immediate airway management and fluid resuscitation should be instituted. Emergency blood replacement with non-cross matched, type specific group may be indicated, while efforts to control hemorrhage are being done.

Immediate Assessment and Resuscitation

Assessment and resuscitation of the trauma victim involves:

a. Quick primary survey.
b. Identification of most life-threatening injury and associated injuries.
c. History.
d. Evaluation of ongoing resuscitation.
e. Specific investigations.

Airway and Breathing

Abdominal trauma patients may have an obstructed airway or inadequate respiration due to associated airway or brain injury or in case of intoxication. As abdominal trauma is often associated with major blood loss, even in absence of other injuries, the victim may have obtunded consciousness and may require assisted breathing. Immediate tracheal intubation and ventilation may be needed for patients in hypovolemic shock to improve oxygen delivery to vital organs. One should specifically look out for subtle signs of shock like anxiety, gradually increasing disorientation, tachypnea, pallor, cyanosis, excessive sweating and prepare for airway intervention at the earliest. Oxygen supplementation should be provided for all abdominal trauma patients until considered unnecessary following initial resuscitation.

Circulation

An abdominal trauma victim may have varying amount of occult or overt blood loss. The resuscitating physician should assess the vital parameters, peripheral pulses, orientation, urine output and depending on these signs, estimate the approximate blood loss (Table 29.1). One has to remember that a patient may bleed more than 30 percent of blood volume before hypotension occurs. Any external bleeding should be controlled preferably by external compression. Blood volume lost needs to be replaced at the earliest with crystalloids, colloids and blood products as per the need through large bore intravenous (IV) cannulae. Severe Hypovolemia may

Table 29.1: Estimation of blood loss[1]

Parameters	*Class I*	*Class II*	*Class III*	*Class IV*
Blood loss*	< 750 ml	750-1500 ml	1500-2000 ml	>2000 ml
	0-15%	15-30%	30-40%	> 40%
Heart rate	< 100	> 100	>120	> 140
Pulse pressure	Normal/Increased	Decreased	Decreased	Decreased
Blood pressure	Normal	Normal	Decreased	Decreased
Urine output	> 30 ml/hr	20-30 ml/hr	5-15 ml/hr	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and Lethargic
Fluids	Crystalloids	Crystalloids	Crystalloids + blood products	Crystalloids + blood products

*For a 70 kg male

lead to hemorrhagic shock (See below). Shock in acute trauma is presumed to result from hemorrhage until proved otherwise. However there may be other simultaneous causes for shock.

Physical Examination

All major systems should be examined to suspect, diagnose or rule out common injuries. The patient's consciousness and other disabilities should be assessed. The abdominal examination should be done, though a normal examination does not rule out abdominal injury. Intra-abdominal injury should be suspected in presence of following signs:

- Increased tenderness and guarding
- Abdominal wall hematoma
- Patterned abrasion, e.g. seat belt sign
- Fracture of the lumbar spine- L1 (pancreatic injury)
- Fractures of ribs 9 to 11 on right side (liver laceration)
- Fractures of ribs 9 to 11 on left side (splenic laceration, diaphragm tear)
- Pelvic injury (bladder injury, retroperitoneal hematoma).

The patient should be fully undressed and all regions of abdomen including perineum should be inspected for any external marks of injury. A cautious log roll will facilitate the complete examination. Abdomen should be palpated to check guarding, rigidity and rebound tenderness. Auscultation confirms the presence or absence of bowel sounds. The findings may change over a period of time necessitating frequent reassessments. Perineal and per rectal examination are important aspects of assessment and management of abdominal trauma.

Assessment of Other Injuries

Consider associated brain injury if altered consciousness is noted. Chest and spine trauma are also common with abdominal trauma. The most critical injury should be identified and patient should be managed accordingly. All abdominal trauma victims should be assessed for pelvic fractures, however repeated pelvic manipulations should be avoided as it can aggravate hemorrhage.[1]

Adjuncts to Physical Examination

Nasogastric tube should be inserted early during the resuscitation process to relieve gastric distension and to remove gastric contents to reduce risk of aspiration. Presence of blood on gastric aspiration suggests injury to upper gastrointestinal tract if upper airway is not bleeding. Avoid nasogastric tube in case of suspected skull base fracture or mid face injury. Oral route may be used. Urinary catheter should be inserted to relieve retention, decompress bladder before diagnostic peritoneal lavage and to monitor urine output. The inability to void, unstable pelvic fractures, blood at meatus, scrotal hematoma may suggest urethral injuries and urinary catheter should not be inserted without ruling out the injury by retrograde urethrography. If urethral injury is likely to be present a suprapubic cystostomy may be performed.[1]

History

History taking should not delay resuscitation in unstable patients. In addition to all relevant medical history, it must include mode of injury so that certain probable injuries can be suspected and looked for. This can be obtained from any person accompanying the victim—bystander, police, other passengers, etc. If the

patient has been attended by paramedics or other physician prior to admission, information should be asked about the patient's status when first observed, injuries noted, type of treatment received and the patient's response to such treatment.

Diagnosis of Abdominal Trauma

As large numbers of abdominal trauma patients are unstable at initial assessment, bedside investigations play a significant role in the diagnosis.

Plain Radiographs

Abdomen with pelvis (AP view): Thoracolumbar spine fractures suggest higher likelihood of bowel injury. Pelvic fractures of anterior elements may suggest associated genitourinary tract injury. Injuries to posterior pelvic elements may have retroperitoneal hematoma.

Chest (AP view): In addition to detecting chest trauma, bowel gas shadow in thorax, displaced nasogastric tube or disrupted normal diaphragmatic contour suggests ruptured diaphragm. This is associated with other abdominal organ injury in 90 percent of patients.[7] Presence of free air under diaphragm suggests a bowel perforation.[8] Splenic injury is commonly associated with left sided lower rib fractures and liver injuries are to be expected with right sided lower rib fractures.

Focused Assessment Sonography in Trauma (FAST)

It is a noninvasive, speedy and sensitive and inexpensive technique which can be used in the resuscitation room itself within 3 to 4 minutes and can be repeated if necessary. This is useful in pregnant patients. In hemodynamically unstable patients, FAST is the investigation of choice.

A trauma clinician is usually trained to identify free fluid in peritoneum and pericardium. The technique has 60 percent sensitivity, 98 percent specificity and 82 percent positive predictive value.[9]

FAST involves assessment of four common views.[10]

- Subxyphoid cardiac view to assess for pericardial fluid and cardiac motion
- Right upper quadrant (RUQ) view to assess for free fluid in Morrison's pouch
- Left upper quadrant (LUQ) view to assess for fluid in the splenorenal and subphrenic space
- Pelvic view to assess for free pelvic fluid
- Free fluid appears anechoic (black) compared to surrounding structures.

Disadvantages

- Minimum of 200 ml of fluid is needed to diagnose
- Cannot distinguish ascites from hemorrhage
- Specific type of injury cannot be diagnosed
- Cannot exclude hollow viscous injury
- Not useful in presence of subcutaneous emphysema.

Hemodynamically stable patients with positive or indeterminate FAST results should undergo CT scan. Hemodynamically stable patients with negative FAST results should be followed by close clinical observation[11] and repeat FAST after 30 minutes (control scan) to confirm the absence of organ injuries or development of hemoperitoneum. In hemodynamically unstable patients, a positive FAST result indicates an emergency laparotomy and an indeterminate FAST result leads to a DPL or a CT scan.[12]

Diagnostic Peritoneal Lavage (DPL)

This is a long established technique for identifying blood in peritoneal cavity. It is employed in the emergency resuscitation area. This technique takes little longer time than FAST and is invasive, hence, its use is decreasing where FAST is available and very few centers are practicing it. A catheter is placed infraumbilically into the peritoneal cavity for fluid aspiration. If the aspirate yields more than 10 ml of blood or gastrointestinal contents, it is considered positive. If it is inconclusive, a liter (10 ml/kg in children) of normal saline is infused into the abdomen and allowed to drain by gravity. It is considered positive if any of the following are present in the aspirate.[4]

- 10 ml of gross blood on aspiration
- > 100,000 RBC/cmm
- > 500 WBC/cmm
- Bacteria
- Bile
- Food particle.

DPL is indicated in blunt trauma in patients with lumbar spinal injury, those with multiple injuries and unexplained shock, obtunded or intoxicated patients with possible abdominal injury and patients with potential intra-abdominal injury who will undergo prolonged anesthesia for another procedure.[12]

Computerized Tomography (CT) Scan

The CT scan of abdomen with pelvis is the standard diagnostic modality for detection of solid organ injuries. Intra-abdominal organs like liver, spleen, kidneys and retroperitoneal injuries are better revealed by CT scan than DPL or FAST.[2] It is indicated in blunt abdominal trauma in hemodynamically stable patients with

equivocal findings on physical examination, neurological injury or impaired consciousness due to drugs or alcohol, multiple extra-abdominal injuries and when the mechanism of injury is suggestive of duodenal or pancreatic injury.[13]

However, CT requires transfer of the patient to another area, which is not appropriate with patients in hemodynamic instability. The procedure is expensive and time consuming and involves injection of contrast material with its attendant risks. Often hollow viscous injuries may be missed. CT scan is contraindicated in a blunt abdominal trauma patient with clear indication of laparotomy and in hemodynamically unstable patient.

Pathophysiology of Hemorrhagic Shock

Hypovolemia

In the early stages of hypovolemia, the injured vessels vasoconstrict to limit hemorrhage, while the collateral vessels dilate to increase perfusion to ischemic areas. The blood loss causes increased sympathetic discharge resulting in increased chronotropic and inotropic actions of heart and shunting of blood away from ischemia tolerant tissues like skin and muscles by vasoconstriction and into the central circulation.[14] Enhanced activity of antidiuretic hormone (ADH) system and reninangiotensin system (RAS) is known to occur in trauma. This retains water and sodium to restore intravascular volume to some extent.[15] Anxiety following blood loss may be partly related to release of catecholamines and slightly reduced cerebral blood flow.[16] Severe hypovolemia may progress further to hypovolemic shock.

Hemorrhagic Shock

Hemorrhagic shock is a condition produced by rapid and significant loss of intravascular volume, which may lead sequentially to hemodynamic instability, decrease in oxygen delivery, decreased tissue perfusion, cellular hypoxia, organ damage and eventually death.[16]

Selective perfusion to organs occurs due to regional variation in these responses. While perfusion to the brain, heart and kidneys are preserved, there is decreased cellular perfusion to systems like skin, muscles and splanchnic circulation. Ischemic tissues resort to anaerobic metabolism which leads to rise in lactate levels, metabolic acidosis and oxygen debt.

The resultant lactic acidosis interferes with ca^{++} mediated cellular functions, leading to accumulation of Na^+ intracellularly and leakage of K^+ extracellularly. Increased Na^+ in the cells is accompanied by increased intracellular edema.[14] Cellular edema may choke off adjacent capillaries and may hamper perfusion even after adequate circulation is restored. Ischemic cells produce lactate and free radicals and release factors like prostacyclin, thromboxane, prostaglandins, leukotrienes, endothelin, complement, interleukins and tumor necrosis factor. These compounds cause direct damage to the cell and get washed back to the systemic circulation after reperfusion.[6] These inflammatory reactions further trigger immune responses causing injury to even nonischemic cells eventually leading to cellular death. As a result, even after adequate volume resuscitation a patient of hemorrhage shock may develop multisystem organ failure.[6]

Resuscitation

The main goals of resuscitation should be to stop the bleeding, replacement of lost blood volume and restoration of organ perfusion with tissue oxygenation. Rapid treatment of shock is crucial for prevention of later complications like ARDS and multiorgan failure.

General signs and symptoms of shock[14] should be looked for (Table 29.2). If signs of hypovolemia are present or suspected, immediate fluid resuscitation should be started without waiting for blood pressure to fall. The priority of management is to stop the ongoing hemorrhage, if needed by emergency surgery to reduce the blood transfusion needs.

Establish two 16 gauge intravenous lines preferably in the antecubital fossa and transfuse 1 to 2 liters of intravenous fluids in an adult or 20 ml/kg in pediatric patients.[1]

In a patient with major active bleeding, occurrence of hypotension reduces further blood loss. Early aggressive efforts to increase blood pressure by rapid crystalloid infusions may dislodge the clot and restart bleeding, leading to further increased infusion and transfusion requirements, dilution of clotting factors,

Table 29.2: Common signs of shock

- Visible evidence of blood loss or long-bone fracture
- Anxiety, progressing to lethargy and coma
- Pallor, diaphoresis, cool skin
- Decreased skin turgor
- Hypotension with narrowed pulse pressure
- Tachycardia
- Prolonged capillary refill
- Diminished urine output
- Nonfunctioning pulse oximeter
- Decreased end-tidal CO_2 after tracheal intubation (a late sign)
- Unusual sensitivity to anesthetic doses

electrolyte imbalance and increased risk of hypothermia eventually causing further deterioration in clinical status.

Permissive Hypotension

The concept of permissive hypotension has evolved to reduce the ill effects of aggressive fluid management. In uncontrolled hemorrhagic shock, resuscitation is aimed at restoration of radial artery pulse, restoration of mental function, and systolic blood pressure of 80 mm Hg, until the bleeding is surgically controlled.[17] The hypotension facilitates *in vivo* coagulation, while lower volumes of crystalloids preserve normothermia and avoid excessive dilution of clotting factors. Once the active bleeding is controlled full resuscitation to optimize perfusion to all the tissues is done.

It is important to know that when hematocrit is very low, oxygenation may be compromised while maintaining permissive hypotension. Clinical trials of deliberate hypotensive resuscitation have avoided the application of this technique to populations perceived to be at greater risk for ischemic complications, including patients with known ischemic coronary disease, elderly patients, and those with injuries to the brain or spinal cord.[17] In patients with head injury and pregnant patients, the systolic blood pressure should be maintained above 100 mm Hg.

Choice of Fluids

There is a major controversy concerning which intravenous solutions should be used for resuscitation. The age old crystalloid/colloid controversy has still not been resolved but has been enlarged to a colloid/colloid debate. Since, in addition to apparent blood loss, fluid deficits may also occur secondary to diffuse capillary leakage and fluid shifts from the intravascular to the interstitial compartment,[15] the type of fluid used plays an important role in circulating volume replacement.

Crystalloids: Isotonic crystalloid solutions such as lactated Ringer's (LR) solution or 0.9 percent saline (NS) are most commonly used for resuscitation. They are readily available, inexpensive and nonallergenic. Lactated Ringer's solution is preferred in hemorrhagic patient over normal saline as the latter is associated with hyperchloremic acidosis. ATLS mentions LR as initial fluid of choice.[1] On the other hand LR being slightly hypotonic, NS is preferred in head trauma and through the blood transfusion lines.

Crystalloids are freely permeable to the vascular membrane and only 25 percent of the infused crystalloid solution remains in the intravascular compartment, whereas 75 percent is extravasated into the interstitium. Therefore, they are shorter acting and have tendency to cause tissue edema. Nearly 3 to 4 times quantities are needed to replace lost volume and such large quantities can produce fluid overload. Even a massive crystalloid resuscitation is less likely to achieve adequate restoration of microcirculatory blood flow.[18]

Glucose containing solutions are generally avoided as hyperglycemia is associated with aggravation of central nervous system injury.

The effects of crystalloid solutions on the coagulation system are complex. With hemodilution up to 20 to 40 percent, crystalloids produce a hypercoagulable state because of dilution of anticoagulant factors such as antithrombin and by platelet activation. After 60 percent hemodilution, both crystalloids and colloids produce a hypocoagulable state.[19]

Colloids: Colloid solutions are more effective plasma expanders than crystalloids. They increase the plasma oncotic pressure, which serves to retain water in the intravascular compartment and minimize interstitial edema in vital organs such as the lung, heart, and brain.[19,20] In spite of these results, often crystalloids are recommended as first choice of fluids for trauma resuscitation. American College of Surgeons currently does not advocate use of colloids in trauma resuscitation even for blood loss > 2000 ml.[1]

Most colloids produce coagulopathy at relatively lower degrees of hemodilution compared with crystalloids. They also prevent naturally occurring platelet activation and hypercoagulability.[19]

Albumin is generally considered to be safe in terms of transmission of infectious diseases with little effect on coagulation. Albumin may have some additional specific effects such as transport function for various drugs and endogenous substances or effects on membrane permeability secondary to free radical scavenging.[15] In patients with impaired vascular endothelial integrity, albumin may pass into the interstitial compartment leading to tissue edema and impaired microcirculatory perfusion. Both 5 and 20 percent are used in trauma, though they are expensive.

Hydroxyethyl starches (HES): Hydroxyethyl starches are high polymeric glucose compounds available as solutions in either saline or balanced salt solution. Different agents are available with different degrees of molecular substitution having varying molecular weight. They give volume expansion of 100 percent and remain in circulation for 3 to 9 hours depending upon the type used. Smaller molecular weight hydroxyethyl

starches and those suspended in balanced salt solutions preserve coagulation better than large molecular weight starches and saline-based colloids.[15,19]

Large amounts (>15-20 ml/kg) of high-molecular-weight HES are avoided because of the well-documented risk of coagulopathy, leading to increased blood loss and transfusion requirements.[21] Life-threatening anaphylactic reactions may occur with different HES preparations, but seem to be rare. Hetastarch (200/0.6) has been associated with impaired renal functions, however, this adverse effect was not observed with lower molecular weight preparations like HES (200/0.5) and HES (130/0.4).[15] A dose limitation exists for all HES preparations ranging from 20 ml/kg (Pentastarch, 10% HES 200/0.5) to 50 ml/kg (Tetrastarch, 6% HES 130/0.4).[19]

It has been observed that intravascular volume replacement with 6 percent HES 130/0.4 improves tissue oxygenation during and after major surgical procedures compared with a crystalloid-based volume replacement strategy.[22]

Dextrans: Dextrans are available as 6 percent dextran 70 and 10 percent dextran 40 solutions. They have largely been abandoned for fluid resuscitation because of the negative effects on coagulation and high anaphylactic potential.[19]

Gelatins: Gelatins have almost equal volume expanding effect as lower molecular starches. Due to their low molecular weight, their effect is short lasing, i.e. 3 to 4 hours. They do not have significant damaging effect on renal functions.[20] They are commonly used for trauma resuscitation in India. However, they are also associated with high incidence of hypersensitivity reactions.

Hypertonic fluids: Even a smaller volume of hypertonic saline will draw intracellular water into the circulation. Thus volume expansion with hypertonic saline is both more efficient and better sustained than with iso-osmolar fluids.[19] This effect is utilized in achieving "small volume resuscitation" in short span of time (4 ml/kg), mainly in prehospital trauma settings. Both 3 and 7.5 percent NaCl either alone or in combination with 6 percent HES (HyperHES) or dextran (HSD) have been used with success.[15] They also reduce tissue edema and intracranial pressure in head injured patients.

Disadvantages:

- Increased bleeding from open vessels
- Hypernatremia,
- Hyperchloremia
- Rebound intracranial hypertension.

Response to resuscitation: After initial fluid infusion, patients need to be categorized depending on their initial response (Table 29.3). The nonresponders and transient responders probably have ongoing major blood loss. Attempts for early control of hemorrhage must be done in all these patients. These patients require constant monitoring for signs of deterioration and may need surgical exploration for control of bleeding.

Blood transfusion: The use of blood products is necessary when the blood loss exceeds 30 percent of estimated blood volume.[16] However, this point is extremely difficult to determine clinically. Hence, any hypotensive patient, who fails to respond to two liters of crystalloid infusion, in presence of probable hemorrhage should be considered for blood and blood product transfusion. Transfusing one unit of packed red cells in an adult patient who is not actively bleeding will increase hemoglobin by 1 to 1.5 gm percent depending on patient's weight. However, in a patient who is actively bleeding, such estimation is impossible.[16]

Emergency 'O' negative blood may have to be transfused in nonresponders along with crystalloids and colloids if typed and cross matched blood is not readily available.

Markers of Resuscitation

Vital signs: Altered vital signs are nonspecific but sensitive for shock. Pain, anxiety, temperature, therapeutic medications, and illicit drugs can influence them. Some patients have physiologic reserves that allow them to maintain vital signs in the normal range

Table 29.3: Response to initial fluid resuscitation[1]

	Rapid response	*Transient response*	*No response*
Vital signs	Return to normal	BP drops after initial rise with tachycardia	Remain abnormal
Estimated blood loss	10-20%	Ongoing-20-40%	> 40%
Need for more crystalloids	Low	High	High
Need for blood	Low	Moderate to high	Immediate
Blood preparation	Type and cross match	Type specific	Emergency blood release
Need for operative intervention	Possibly	Likely	Highly likely

Table 29.4 Signs of adequate resuscitation

- Heart rate below 120/minute
- Systolic blood pressure 80-100 mm Hg
- Hematocrit at 25-30%
- Core temperature higher than 35°C
- Urine output > 0.5-1 ml/kg
- Normalization of PT and PTT
- Platelet count > 50,000 / mm^3
- SpO_2 > 95%
- pH > 7.3

until terminal cardiovascular collapse. Vital signs still are the most commonly used parameters in assessing adequacy of resuscitation. (Table 29.4)

A decrease in heart rate, an increase in blood pressure, a decrease in the FiO_2 needed to maintain an adequate PaO_2, an increase in urinary output and longer time intervals between therapeutic interventions signal a movement toward normal homeostasis. In spite of achieving normal hemodynamics it is not guaranteed that perfusion to all organs and tissues is maintained adequately.[15,23]

Arterial pressure: As arterial blood pressure reflects the patient's volume status, cardiac contractility as well as systemic vascular resistance, it is strongly recommended during resuscitation of hemorrhagic shock. Excessive respiratory variations in systolic blood pressure during mechanical ventilation may be a sensitive indicator of hypovemia.[23]

Central venous pressure (CVP): Central venous pressure reflects changes in cardiac filling pressures in response to the fluid resuscitation. Persistently low central venous pressure suggests hypovolemia or ongoing blood loss. Higher CVP associated with low blood pressure may indicate presence of myocardial failure or presence of chest trauma with cardiac tamponade or tension pneumothorax. CVP does not reflect status of microcirculation.

Pulse oximetry: A nonworking pulse oximeter may indicate poor peripheral perfusion, though this may be due to other reasons also. SpO_2 > 95 percent with good waveforms suggest adequate perfusion.

Exhaled CO_2: End tidal CO_2 (E_TCO_2) reflects effectiveness of gas exchange during resuscitation. Increase in E_TCO_2 levels may indicate improvement in pulmonary capillary blood flow even during CPR. However, E_TCO_2 may get altered by hypoventilation, CO_2 production, pulmonary embolus or collapse. Along with $PaCO_2$ it can be used as a guide for adequacy of resuscitation.

Urine output: A urine output > 0.5 to 1 ml/kg/hour is considered adequate output, though this does not always indicate adequate renal perfusion. Urine output lesser than this amount is considered as an indicative of inadequate perfusion and need for more fluid administration.

Arterial blood gases (ABG): Arterial blood gas analysis provides information on the global acid base cv bstatus, efficiency of oxygenation and ventilation and hemoglobin. Normalization of each of these parameters suggests that resuscitation is reversing the shock state. A decreasing pH in the trauma patient is generally due to metabolic acidosis secondary to hypoperfusion or a respiratory acidosis secondary to hypoventilation. Fluid replacement and increasing the minute respiration will correct these abnormalities. Improvement of oxygenation is manifested by an ability to maintain an adequate PaO_2 with lower FiO_2 levels.

Base deficit: Base deficit has emerged as an important tool for measuring adequacy of resuscitation and is more sensitive than vital signs in detecting shock. Base deficit more negative than -5 is predictive of severe injury and mortality.[24] Failure to clear base deficit is an additional indicator of outcome, associated with higher mortality.[24] Infusion of large quantities of 'normal' saline can produce hyperchloremia and may mimic inadequate resuscitation.[23]

The use of bicarbonate during resuscitation and any pre-existing medical conditions that result in chronic elevations or reductions in bicarbonate levels may alter the results.

Serum lactate: Serum lactate is a biochemical marker of choice for hypoperfusion.[25] The amount of lactate produced is believed to correlate with the total oxygen debt, the degree of hypoperfusion and the severity of shock. It is a better predictor of risk of infection as well as patient survival than other markers like pH, base deficit or anion gap.[25] Lactate is cleared by the liver, therefore, liver injury or hepatic disease can decrease lactate clearance leading to high levels which is not associated with ongoing tissue hypoxia. Nonperfused tissues during true ischemia do not contribute to serum lactate levels. Estimation of serum lactate does not offer information on regional tissue hypoxia or blood flow.

Gastric tonometry: Hypovolemia and shock shunt blood from other tissues to the heart and brain. Therefore, stomach mucosa is used to assess tissue hypoperfusion. A tonometer is a nasogastric tube that has a fluid filled balloon distally. It measures the partial pressure of carbon dioxide in the gastrointestinal

mucosa by allowing the equilibration of the partial pressure of carbon dioxide in the fluid filled balloon with that of gastric mucosa.[25] Assuming that excess production of CO_2 occurs during hypoxia, an increasing value of CO_2 should reflect tissue hypoperfusion. Because the gut is highly susceptible to hypoperfusion, reduction in pH may be an early indicator of concurrent global hypoperfusion and impending shock.

Echocardiography: Both transthoracic as well as transoesophageal echocardiography have been used to assess volume status, cardiac function and need for further infusion, vasopressors or inotropes. In addition, these also provide information about pericardial effusion or tamponade.[26]

Cardiac output and derived variables: Other methods to assess adequacy of fluid resuscitation include assessment of cardiac output by pulmonary artery catheter, minimally invasive (Lithium Dilution cardiac output and peripherally inserted continuous cardiac output)[14] or noninvasive (using exhaled CO_2 rebreathing technique-NICO) methods. They all can provide global measurements of cardiac output and do not reflect perfusion in critical vascular beds.[25] The role of newer lesser invasive methods in acute trauma setting is yet to be established. Cardiac output monitoring may be invaluable in presence of severe cardiac disease.

All these measures do not rule out regional hypoperfusion.

Surgical Management

Surgery may be required in absence of definite diagnosis in certain situations. In case of unexplained persistent hypotension with inconclusive investigations, exploratory laparotomy to detect intra-abdominal injury may be done, though the incidence is decreasing with improved diagnostic modalities. Gun shot wounds or penetrating injuries to abdomen with uncontrolled bleeding requires emergent exploratory laparotomy, even without diagnostic tests. In stable patients, diagnostic laparoscopy may be done to detect and possibly treat specific injury like diaphragmatic trauma. However, its scope is limited and may have its own hazards like injury and bleeding due to trocar, problems of pneumoperitoneum with chest injury or hypovolemia.

Indications for laparotomy in a patient with blunt abdominal injury include the following:

- Signs of peritonitis
- Uncontrolled shock or hemorrhage
- Clinical deterioration during observation
- Hemoperitoneum findings after FAST or DPL examinations
- Continued requirement of blood components.

Preoperative Investigations

Following investigations are considered in patients of abdominal injury when surgical intervention is likely to be needed:

- Blood group-type and cross matching
- Hemoglobin/hematocrit estimation
- Platelet count
- Prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR)
- Blood urea nitrogen (BUN) and serum electrolytes
- Arterial blood gases (ABG)
- Serum Amylase and lipase if pancreatic injury is suspected
- Hemoccult test for occult rectal blood
- Urinalysis for hematuria
- Urine toxicology screening, when indicated
- Pregnancy testing for women of childbearing age
- Chest X-ray to rule out or diagnose chest injury
- Pelvic X-ray to rule out pelvic/femur fracture.

In patients who continue to bleed internally and thus remain unstable, in spite of initial fluid resuscitation, emergent laparotomy is needed to control bleeding. In such instances, one should not wait for any laboratory investigations except blood grouping and cross matching. Samples for other investigations may be sent as soon as time permits. Clinical judgement is time and life saving in such cases. Coagulation abnormalities are imminent in patients with major blood loss due to loss of coagulation factors and are further aggravated due to hemodilution post-fluid resuscitation. Thus, platelet count, PT, PTT and INR are important markers to assess the coagulation status.

Anesthetic Management

Preoperative Assessment

An anesthesiologist may be the primary care physician in assessing and resuscitating the patient as a part of the trauma team or may be a new member called in for providing anesthesia for surgical intervention. In the latter situation, though information can be sought from the primary care physician or from the notes, reassessment of patient can never be over emphasized. The patient needs to be assessed thoroughly as per ATLS guidelines and one may have to initiate or continue resuscitation.

A quick assessment of patient's airway and breathing should be done. A patent airway may need to be established in emergency or to be rechecked if it is already established. Unlike external bleeding which is obvious, internal bleeding can not be estimated. Status of intravascular volume should be assessed by

palpating peripheral pulses, blood pressure, skin color and turgor. If central line is already placed, central venous pressure gives good estimate of volume status. Associated injuries should be noted. Most life-threatening injury should be identified and its management should be continued. Care should be taken not to aggravate any injury. In presence of pneumothorax, however small, an intercostal drainage tube should be placed prior to institution of positive pressure ventilation. Any external bleeding should be controlled. Repeated assessment of level of consciousness, vital parameters, pallor and capillary fill will help in determining any deterioration in patient status.

All diagnosed and likely injuries should be reviewed; their management rechecked and continued perioperatively, if needed. All laboratory and radiological investigations should be reviewed.

Monitoring

Commonly, following monitoring is used for an abdominal trauma surgery:

- *Electrocardiogram*: Heart rate is a nonspecific indicator of volume status
- *Pulse oximeter*: A nonworking pulse oximeter may indicate inadequate resuscitation
- *Capnography*: To assess adequacy of ventilation. This is a valuable aid to maintain normocarbia in presence of associated head trauma
- *NIBP/Invasive arterial pressure monitoring*: To monitor accurate systemic blood pressure
- *Central venous pressure monitoring*: To judge volume status. Patients with severe cardiac disease or heart failure may need pulmonary artery pressure monitoring
- *Temperature monitoring*: To prevent hypothermia. Both core temperature and skin temperature should be monitored to assess perfusion to peripheral organs
- *Arterial blood gases*: To assess oxygenation and acidosis
- *Urine output monitoring*: To judge renal function
- *Peripheral nerve stimulator*: To avoid overdosage of neuromuscular blocking agents in the event of delayed elimination due to reduced organ function.
- *Bispectral index monitoring*: To prevent awareness during surgery, as unstable patients are often given minimum amounts of cardiodepressant anesthetic drugs for fear of unpredictable or exaggerated response.

Intravenous Access

Securing two 16 G or larger, short, intravenous lines in the antecubital fossae is probably the anchor of resuscitation. No amount of colloids or blood can save the patient if rapid infusing lines are not available. With two large peripheral lines and a triple lumen central line one is adequately armed to handle most bleeding patients. The use of central vein allows access to a larger vein for rapid fluid infusions and also provides an easy means to administer medications. Additionally, central venous pressure can also be measured. In abdominal trauma, central venous access should be preferably obtained in upper extremities. The use of veins draining into the inferior vena cava (IVC) is also avoided when the IVC is severed by the trauma or if surgery involves clamping the vein.[8] Internal jugular and subclavian veins are most commonly used. Risk of pneumothorax with both the routes should be kept in mind and appropriate precautions should be taken. Access to internal jugular vein may not be feasible in case of associated cervical spine trauma due to the cervical collar and inability to turn the neck.

Effect of Hemorrhage on Anesthetic Drugs

It has been observed since long that the trauma patients who have bled need smaller, incremental doses of anesthetics. Full doses of certain anesthetics may lead to pronounced and often unwanted side effects with potentially disastrous consequences. Such effects have been noted with sedative-hypnotics, opioids and local anesthetics.[27] Various studies have shown that equivalent dosing leads to higher drug concentrations with severe blood loss when compared with unbled controls. Decrease in circulating blood volume and cardiac output along with compensatory changes in regional blood flow are likely physiologic mechanisms.[27] Shock decreases the size of the central compartment and systemic clearance for drugs, resulting in increased plasma concentrations. Lower circulating plasma proteins can further lead to higher free drug concentrations in blood causing markedly higher therapeutic effects.

Intravenous anesthetic agents: The most commonly used intravenous anesthetic agents—sodium thiopentone and propofol—both are poorly tolerated by patients with major blood loss probably due to their vasodilator and negative inotropic properties. These intravenous anesthetic agents, administered to a trauma patient in hemorrhagic shock may potentiate profound hypotension and even cardiac arrest as a result of inhibition of

circulating catecholamines. Studies have shown that in hemorrhagic subjects, higher plasma concentrations of propofol were observed than in normal subjects.[28] There is lower volume of distribution and lower clearance of drugs in shock patients. Hence, the anesthetic agents reach the brain in higher concentration leading to more pronounced anesthetic effect. The loss of response to various stimuli and lower bispectral index values were observed in hemorrhagic patients even at lower plasma concentrations.[28,29] If these agents are to be used, doses should be markedly reduced to achieve desired clinical endpoints in sedation and hypnosis. It is recommended that a patient in hemorrhagic shock should receive only 10 to 20 percent of the propofol dose that a healthy patient would receive.[30]

If a patient is partially resuscitated, these exaggerated responses still persist but on a smaller scale.[27]

Such exaggerated cardiac depressant effect is not commonly seen with etomidate[31] and ketamine.[32] Ketamine increases sympathetic tone and is a potent analgesic. Therefore, both these drugs are useful for patients suffering from life-threatening blood loss with minimal dose adjustments.[27] It is important to remember that ketamine is direct myocardial depressant. In hemodynamically stressed patients, the cardiac depression may be unmasked and lead to cardiovascular collapse.[33]

Opioids: Opioids have been found to cause minimal cardiovascular depression in both normal and hemorrhagic patients. Infusions of fentanyl and remifentanil have been well-tolerated in presence of blood loss.[34,35]

Inhalational agents: All inhalational anesthetics produce dose-dependent depression of myocardial contractility. Desflurane, isoflurane, and sevoflurane maintain cardiac output better than older agents such as enflurane or halothane, mainly through a peripheral vasodilatory effect. There are no absolute contraindications of any inhalational agent for abdominal trauma. Yet, halothane and sevoflurane have been occasionally avoided due to a theoretical potential for liver and renal injury, respectively. Nitrous oxide (N_2O) should be avoided to limit bowel and closed space gas accumulation.

Effects of simultaneously administered drugs in these situations remain to be studied.[27]

Neuromuscular blocking drugs: The site of action of neuromuscular blocking agents is at the neuromuscular junction in muscle tissue. Because the fraction of cardiac output irrigating muscle is reduced when cardiac output falls, in favor of vital organs, the amount of neuromuscular drug reaching its target might be less. However, the effect of reduced muscle blood flow is compensated by the increased drug concentration in arterial blood, because of dilution of the drug into a small total blood volume.

As a result, the dose of neuromuscular blocking agent in hypovolemia is not markedly different from normovolemia. But, the onset time, that is, interval from injection until maximum neuromuscular blockade, is increased.[36]

Thus, adverse effects of various anesthetic agents can be reduced by providing resuscitation to restore the hemodynamic status to near normal. The altered behavior of various drugs with respect to duration of effect, peak concentrations and extent of cardiovascular depression should be considered while choosing agents for emergency anesthesia.

Central neuraxial block: The use of central neuraxial block is contraindicated in hemodynamically unstable patients, in view of technical difficulties in placing the patient in lateral or sitting position, sudden hypotension that may occur following the block, development of coagulopathies due to major blood loss and most importantly, need to provide controlled ventilation and oxygenation to treat tissue hypoxia.

Induction of General Anesthesia

In hypovolemic patients the administration of any anesthetic may cause interruption of compensatory sympathetic overactivity leading to hypotension. This is a common and often avoidable complication at induction. Previously, healthy young patients can lose up to 40 percent of their blood volume before experiencing a decrease in blood pressure, thereby leading to potentially catastrophic circulatory collapse with induction of anesthesia, regardless of the drug chosen. As mentioned above the dose of intravenous anesthetic must be decreased in the presence of hemorrhage. In extremely obtunded patients rapid-sequence induction of anesthesia and tracheal intubation may be done with neuromuscular blocking agent alone. Small doses of midazolam can reduce the incidence of patient awareness but can also contribute to hypotension. Only opioids in reduced doses may be given for narcosis and analgesia. In relatively stable patients induction may be done with reduced dose of intravenous anesthetics.

Succinylcholine remains the neuromuscular blocker with fastest onset less than 1 minute and shortest duration of action 5 to 10 minutes. These properties make it popular for rapid-sequence induction of anesthesia. Alternatives to succinylcholine include

rocuronium (0.9 to 1.2 mg/kg) and vecuronium (0.1 to 0.2 mg/kg). Because these drugs have no significant cardiovascular toxicity, larger doses can be administered to achieve rapid (1 to 2 minute) neuromuscular blockade. Unfortunately, at this dose the duration of action of either rocuronium or vecuronium will be prolonged, which may be of significance if inadequate sedation leads to patient awareness of paralysis or if it prevents ongoing neurologic assessment. Such techniques should not be used for anticipated difficult intubation cases.

Tracheal intubation: Patients who are unstable with depressed consciousness are usually intubated before reaching the operating room (OR). Patients requiring intubation in OR are usually the ones that are reasonably stable and oriented.

A trauma patient is always considered to have a full stomach and at risk for aspiration during induction of anesthesia. Reasons include ingestion of food or liquids before the injury, swallowed blood from oral or nasal injuries, delayed gastric emptying associated with the stress of trauma, and sometimes, administration of liquid contrast medium for abdominal CT scanning. Gastric decompression with a nasogastric tube should be done at the earliest. Rapid sequence induction or modified RSI is used in these cases.

During emergency airway management, cricoid pressure should be applied continuously from the time the patient loses protective airway reflexes until tracheal tube placement and cuff inflation are confirmed. If there is no evidence of associated chest trauma and patient is having normal breathing preoperatively, then chances of desaturation during induction are minimal. Preoxygenation without positive pressure ventilation till tracheal tube insertion is commonly employed. The APL valve of breathing system kept in the semi-closed position helps in oxygen insufflation during apneic phase. In presence of associated chest trauma, obesity or in elderly patients chances of desaturation exist. Such patients may be ventilated intermittently while maintaining cricoid pressure. The presence of an "uncleared" cervical spine mandates the use of in-line manual stabilization throughout the intubation procedure.

Maintenance of Anesthesia

In hemodynamically unstable patients, oxygen, nitrous oxide and neuromuscular blockers alone may be sufficient to prevent awareness, however, routine use of N_2O should be avoided. Opioids are added for analgesia. If hemodynamic stability permits, inhalational agents may be added.[8] Inhalational agents provide satisfactory anesthesia and the dose and depth can be changed easily as per patient's clinical status. In a hypothermic and hypovolemic patient pulse oximeter may be unreliable. One may have to depend on ABG for oxygenation and every effort made to prevent hypothermia and hypovolemia.

Intraoperative Concerns

Surgical Exposure

For exploratory laparotomies, a large incision is taken to see all abdominal organs. Stomach should be deflated using nasogastric tube. Avoiding use of N_2O will prevent bowel distension and expansion of closed air cavity. Provide adequate muscle relaxation. Avoid excessive use of crystalloids to prevent further tissue edema.

Antibiotics

Broad-spectrum antibiotics should be given preoperatively to cover gram-positive and gram-negative bacteria including anaerobes and enterobectors. Commonly, third generation cephalosporins with metronidazole infusion are used. Higher dosage may be indicated if contamination is high.

Hypotension

Sudden hypotension is common at induction due to effects of multiple anesthetic and other depressive agents given within a short time span. This can be limited by choosing agents with minimal depressant properties, adjusting the timing of drug administration and titrating dose of each agents as per patient response. Sudden cardiovascular collapse can also occur when abdomen is decompressed at opening of peritoneum. This occurs due to release of pressure on intra-abdominal vessels leading to increased perfusion to lower body and abdominal organs. Associated injuries can also contribute to hypotension. Of these, important ones are undiagnosed chest injuries, development of tension pneumothorax or hemothorax or cardiac tamponade, restarting of bleeding from pelvic fractures and spinal shock in case of spine injuries.

Management of Blood Loss

Major blood loss is expected during emergency abdominal exploration. The resuscitation should be continued in the intraoperative period. In addition, blood loss needs to be replaced with rapid fluid and blood administrations.

Flow of fluids through an intravenous line can be increased by using larger diameter and short length cannula and increasing the infusion pressure. Methods of increasing the infusion pressure are:

- Increasing pressure gradient by raising the IV pole
- Use of a mechanical pressurizing infusion devices.

Pressure infusion devices range from hand-inflated airbags that squeeze the fluid bag manually to those driven by compressed air systems or electronic devices. Rapid Infusion systems can warm IV fluids to physiologic temperature and pump them at rates from 10 to 500 ml/min. Rapid infusers are especially useful in liver trauma and polytrauma where one can expect rapid and massive blood loss. Such devices should be kept available in all operation theatres where trauma cases are taken up.

Blood transfusion is indicated if:

- Hemoglobin < 6 gm percent
- Hemoglobin 6 to 8 gm percent with ongoing loss
- Persistent hypotension with visible blood loss
- Tachycardia > 140/min with decreased urine out
- Hypothermia and persistent acidosis
- Clinical pallor with visible blood loss
- With greater than 30 to 40 percent blood loss, pulse rate more than 160 /minute and major vessel injury, non-cross matched O-ve blood may be life saving.
- Existing coagulopathy has to be corrected and fresh frozen plasma (FFP) and platelet transfusion may be required in such patients.

Under certain circumstances, use of salvaged blood from abdominal cavity through cell saver devices for autotransfusion has been found to be effective, though there is a risk of the possibility of bacterial or fecal contamination.[2]

Coagulopathy

Though primary cause of bleeding is trauma or surgical, secondary cause is hypothermia and coagulopathy. The coagulopathy sets in during massive blood transfusion due to acute consumption of coagulation factors. It is likely in any patient losing more than a single blood volume (4-5 L) or receiving more than 10 units of red blood cells. The fresh frozen plasma, cryoprecipitate and factor concentrates may be indicated to correct specific factor deficiencies, usually after the patient has lost about 70 to 80 percent of blood volume. Platelet transfusion is indicated when the platelet count goes below 50,000/cmm. Prothrombin time, activated partial thromboplastin time, fibrinogen and fibrin degradation products are to be monitored. Thromboelastography (TEG) is an integrated assessment of the whole coagulation with interaction of separate coagulation components. Hence, TEG and platelet function testing are useful. Supplement calcium, if large amounts of citrated blood has been given.

Hemostatic Agents

Various hemostatic agents have been used to reduce blood loss during surgery.

Tranexamic acid: Tranexamic acid is an antifibrinolytic drug that reduces nonsurgical bleeding and general oozing. Tranexamic acid is not expensive and has been reported to reduce postoperative bleeding and transfusion requirements. It is used as 10 mg/kg intraoperatively and continued postoperatively for 24 hours.

Desmopressin: (1-deamino-8-D-arginine vasopressin [DDAVP]), is a synthetic analog of the antidiuretic hormone vasopressin. It increases the levels of factor VIII and von Willebrand factor and is a well-established therapy for hemophilia and von Willebrand disease. Desmopressin can enhance platelet adhesion promoting stronger clots and reduce fluid requirement in hemorrhagic shock.[37] It may reduce blood loss and transfusion requirement in patients if given in early hemorrhagic period. The ultimate role of desmopressin remains to be determined. It can cause hypotension, hyponatremia, and increased platelet adhesion.

Recombinant factor VII (rFVIIa) : The mechanism of action is activation of factors IX and X, inducing a thrombin burst and faster formation of the fibrin clots at the site of vascular injury. This "thrombin burst" depends heavily on adequate levels of fibrinogen being available, so it is advisable to give it after administering fresh-thawed plasma or any available cryoprecipitate. Acidosis and hypothermia are known to inactivate rFVIIa. Its effect on generalized ooze on the surgical field can be quite impressive. However, it is very expensive.

In trauma patients rFVIIa may play a role as an adjunctive hemostatic measure, in addition to surgical hemostatic techniques. Moribund patients in whom standard surgical and medical treatment failed to control diffuse bleeding that resulted from trauma related coagulopathy and massive transfusion may benefit from 100 µg/kg rFVIIa. Administration of rFVIIa has resulted in cessation of the diffuse bleed, with significant decrease of blood requirements, shortening of prothrombin time and activated partial thromboplastin time.[38]

Topical fibrin glue may be used for control of minor surgical bleeding.

Acidosis

Acidosis occurs frequently with hypoperfusion of tissues. It reduces myocardial contractility and shifts oxygen dissociation curve to right, favouring oxygen delivery to tissues. Improved tissue perfusion will correct acidosis, however, if pH is < 7.1, administration of sodium bicarbonate may be appropriate.

Hypothermia

Hypothermia is a significant risk in early hemorrhagic shock. Decreased body temperature has the potential to change the rate constants of important biochemical processes, including coagulation.[23] Core temperature should be monitored. The anesthesiologist should do everything possible to preserve and support a normal core body temperature. These may include warming the environment, keeping the patient covered, use of warming mattresses and forced air blower blanket for upper body, humidifying gases, use of breathing systems that conserve heat and warming all administered fluids. Hypothermia can cause vasoconstriction and inhibit the establishment of adequate tissue perfusion.

In major trauma hypothermia, coagulopathy and acidosis are best prevented, rather than treated.

Perioperative Analgesia

Adequate analgesia should be provided preferably using multimodal approach. Some of the commonly used methods either alone or in combination include:

- Surgical site infiltration of local anesthetics
- Nonsteroidal Anti-inflammatory agents like diclofenac sodium- intravenous or per rectal.
- Fentanyl or remifentanil infusion in reduced doses
- Intravenous butorphanol, pentazocine or nalbuphine
- Intravenous tramadol
- Intravenous or epidural buprenorphine.

In patients with deranged coagulation epidural drugs and NSAIDs are generally avoided.

Abdominal Closure

Patients, who have been in hypotension for longer period and were resuscitated with large amounts of fluids, are likely to develop bowel edema and often paralytic ileus. As the edema can further aggravate postoperatively, it may not be appropriate to close the abdomen under tension. The intestines may need to be decompressed or at times, 'Open abdomen' seal may be indicated. (See below).

Multiple Surgeries

If a patient has multiple injuries requiring surgical management, in unstable status, only life-threatening surgeries should be undertaken with ongoing resuscitation. Injuries which do not pose threat to the patient immediately should not be performed simultaneously as this will increase the duration of surgery, anesthesia and bleeding leading to the triad of acidosis, hypothermia and coagulopathy. Such injuries can be dealt with later after the patient stabilizes.

Emergence and Tracheal Extubation

Many abdominal trauma patients often require post-operative ventilatory support because of hypothermia, severe blood loss with massive transfusion, residual acidosis and coexiting CNS trauma, direct pulmonary or chest wall trauma. If there is any doubt about the patient's ability to maintain tissue oxygenation, it is appropriate that the patient be transported to the trauma intensive care unit with the tracheal tube in place. Appropriate analgesic medication should be administered, with sedation if necessary. 12 to 24 hours of ventilatory support allows confirmation of successful resuscitation and surgical repair, hemodynamic stability, titration of appropriate analgesia and resolution of intoxication. Many patients can be extubated easily and safely at this time; those who cannot are at high risk for the development of multisystem organ failure heralded by the development of post-traumatic ARDS and will usually require prolonged ventilatory support.

Probable postoperative complications:

- Abdominal compartment syndrome
- Anemia
- Sepsis
- ARDS and MODS
- Leak at bowel anastomotic site
- Intra-abdominal/pelvic abscess
- Complications of multiple transfusion
- Deep venous thrombosis.

Damage Control Surgery (DCS)

With the advances in anesthesia and critical care and improved understanding of pathophysiology of severe hemorrhage, it is now possible to provide care for patients who are at the end of their physiological reserve on arrival. Damage control surgery is an effort to salvage such patient, who otherwise may not be able to survive a major abdominal surgery and further blood

loss without going into potentially fatal cycle of acidosis, hypothermia and coagulopathy.

This is planned during the initial stages of resuscitation itself to salvage the patient's life and no further time is wasted in diagnostic procedures. Damage control surgery is a systematic approach performed in three distinct phases.

Phase I

Limited operation to control hemorrhage and contamination. The abdomen is opened and packed tightly in all four quadrants.[39] Systematic exploration is undertaken in each quadrant.

Hemorrhage Control

- Goal is to identify and control of life-threatening hemorrhage
- Bleeding is controlled by application of vascular clamps, suture ligature, vascular stents and packing
- Complex reconstructions are not done
- Splenic and renal injuries are managed by rapid resection
- Hepatic injury may require manual compression with surgical hands, packing, plugging with procoagulant material, partial resection, Pringle maneuver of hepatic artery and portal vein occlusion
- Vascular injuries are managed frequently by ligating, as end-to-end anastomosis or grafting is time consuming
- Surgery is stopped when there are no signs of surgical bleeding.

Contamination Control

- The goal is to rapidly stop spillage of gastrointestinal contents
- Bowel injury is closed by simple techniques like rapid suturing, staple closure or resection without anastomosis
- Bile duct injuries are managed by ligation, end choledochostomy or simple drainage.

Aggressive resuscitation and warming is done during the procedure. At the end of the surgery, rapid and temporary abdominal closure is provided, if needed with large abdominal packs inside near bleeding sites of liver. Often the abdomen is not closed primarily if bowel loops are dilated and edematous. These are accomodated inside a silastic or other similar pouch sutured along the incision. Drains are placed in abdomen to remove fluid and blood often assisted with vacuum device. If pelvic fracture is associated, then the pelvic ring is reduced and stabilized with external fixator to decrease further bleeding.

Phase II

Secondary and continued resuscitation in the ICU is performed for 24 to 72 hours to:

- Maximize hemodynamic stability
- Optimize oxygen delivery
- Provision of blood components
- Correction of acidosis (pH >7.3, Normal lactate levels)
- Correction of hypothermia (core temperature >35 C)
- Correction of coagulopathy (Normal PT, INR)
- Ventilatory support
- Monitoring and reduction of ICP, Maintaining CPP in head injury
- Monitoring of abdominal compartment syndrome
- Continued injury identification and appropriate management
- Management of open abdomen.

Phase III

After the patient improves and becomes normothermic, reoperation is performed for removal of packs, effective hemostasis and definitive repair of abdominal injury including debridement of nonviable tissue, reconstruction of bowel, feeding procedures, colostomy along with attempted fascial closure. Damage control surgery can reduce the mortality in critically injured civilian patients by 50 percent.[40] Hence, with the advances in critical care management, the concept of damage control surgery is more commonly being adapted for critical trauma patients.

Abdominal Compartment Syndrome (ACS)

Compartment syndrome is defined as 'a condition in which increased pressure in a confined anatomical space adversely affects the circulation and threatens the function and viability of the tissues within'.[41] The abdominal compartment syndrome is a condition of increased intra-abdominal pressure and has been described in detail in chapter 10. In trauma settings, acute ACS may occur either due to primary abdominal trauma or secondary reperfusion of tissues after prolonged ischemia.

Primary ACS

This occurs due to a primary abdominal pathology. Most ACS patients belong to this category due to intraperitoneal, retroperitoneal or pelvic hemorrhage following trauma. This leads to state of shock due to massive hemorrhage and reduced venous return.

Resuscitation with large volumes of fluids and blood products further aggravate blood loss and tissue edema. Immediate surgical intervention is needed to break this cycle.

Primary ACS can also develop in a postoperative patient due to edema developing after resuscitation, reperfusion, revascularization, intra-abdominal packing or excessive surgical handling.

Secondary ACS

This occurs due to accumulation of fluid in 'Third space' as occurs in massive edema due to capillary leak following resuscitation without a primary abdominal pathology. This entity needs to be kept in mind for all major trauma patients, who have bled due to nonabdominal trauma and were in hypotension for longer period of time requiring resuscitation with large volume of crystalloids causing visceral edema. This may require decompressive laparotomy to reverse the pathological consequences of intra-abdominal hypertension (IAH).

Combined lesions of the abdomen and the pelvis revealed a significantly increased incidence of ACS compared with isolated injuries of the abdomen or the pelvis.[42] Furthermore, the combination of abdominal and pelvic injuries showed the most rapid occurrence within 4 to 5 hours after trauma. The incidence of ACS is also high following damage control surgery as many of these patients need to be treated for shock. Centers which use supranormal resuscitation [goal to maintain oxygen delivery index (DO_2I) > 600 ml/min m^2] have shown that this results in significantly increased incidence of IAH or ACS.[43]

The first clinical signs may be decreasing urine output and increased airway pressure or respiratory failure. Pressures exceeding 15 mm Hg can lead to oliguria and splanchnic hypoperfusion. In addition, increased intra-abdominal pressure causes decreased tidal volume, increased ventilatory pressures, and increased atelectasis. Increased intra-abdominal pressure can also cause venous hypertension and elevate intracranial pressure. IAH or ACS is commonly diagnosed by measuring bladder pressure. Alternatively, direct intra-abdominal, inferior vena caval or gastric pressure may also be measured.[41] In patients with severe hemorrhagic shock, the increase in the intra-abdominal pressure through ACS can further augment the preexisting ischemia/reperfusion injury of the gut, leading to intestinal infarction and necrosis. Abdominal pressures greater than 20 to 25 mm Hg, accompanied with organ failure require decompression (Table 29.5).

Table: 29.5 Grading of abdominal compartment syndrome

Grade	*Bladder pressure (mm Hg)*	*Recommended treatment*
I	10-15	Maintain Normovolemia
II	16-25	Hypervolemic resuscitation
III	26-35	Decompression
IV	> 35	Decompression and re-exploration

Early laparotomy and hemorrhage control form the mainstay of management of primary traumatic ACS. Opening the abdomen can result in rapid decrease in intra-abdominal pressure, with a resultant reperfusion syndrome. This can lead to sudden hypotension and possible cardiac arrest unless proper fluid loading is done. As bowel loops are often dilated, primary closure of abdomen may lead to IAH. In such patients, open abdomen seal as described earlier will be useful. Re-exploration and abdominal closure can be done after edema subsides. Along with the surgery, adequate hydration, ionotropic support, correction of acidosis and electrolyte imbalance and maintenance of adequate minute ventilation are needed.

Associated Conditions

Chest Injury

A major chest trauma may hamper breathing and contribute significantly to hemorrhage. If necessary, early intubation and ventilation should be instituted before surgery. In case of pneumothorax, an intercostal drainage tube should be placed prior to institution of positive pressure ventilation, even if it is small. Decision to provide postoperative ventilatory support should be based on the extent of chest injury as well as abdominal injury.

Head Injury

Polytrauma patients who need emergency surgery for both the head and the abdomen trauma are rare.[44] Any patient with altered sensorium after correction of hypovolemia and hypoxia is usually due to head trauma. In stable patients with altered mental status and potential injuries to both head and the abdomen, the abdomen should be evaluated by FAST or by DPL. If it is negative, CT scan of the head should be done. If on FAST or DPL gross blood is obtained and there are no focal neurological signs, it is best to proceed to exploratory laparotomy.[44]

Stabilization of uncontrollably bleeding abdominal trauma usually gets precedence over the diagnosis of head injury. Care should be taken to prevent rise in ICP

during intubation and resuscitation. The patient may be sent for CT scan after hemodynamic stabilization probably following laparotomy is achieved. Extra care needs to be taken to avoid hypoxia, hypertension and coughing during the surgery. Mild hypocarbia to normocarbia should be maintained. The mean arterial pressure should be maintained above 70 mm Hg for cerebral perfusion.

Pelvic Injury

Concomitant pelvic injury may lead to progressive retroperitoneal hemorrhage. Abdominal trauma should always be suspected with pelvic injury and FAST or DPL should be carried out. In shocked patient with combined injuries pelvic stabilization with external fixator and exploratory laparotomy may have to be done together to control hemorrhage as an emergency resuscitative procedure. Usually such patient is intubated on admission before surgery and resuscitations is continued during surgery.

Pregnancy

Trauma is the leading cause of nonobstetric death in women between the ages of 14 and 44 years. Fetal injury and death following blunt and penetrating abdominal trauma is common, as the gravid uterus displaces viscera and acts as a shield. Aggressive maternal resuscitation remains the best chance for fetal survival. Indeed, the major cause of fetal death is maternal death.[2] Ultrasonography should be done as soon as possible. There is a potential for amniotic fluid embolism.

Future Advances

Newer multislice CT can provide faster images, with patient remaining in the area for very short time. Portable CT scans placed close to the resuscitation areas will help in faster diagnosis in unstable patients. Easy availability of newer hemostatic agent rFVIIa will help in reducing nonsurgical bleeding. Advances in interventional radiology can be more commonly employed in trauma for embolization of bleeding vessel especially in liver and may thus reduce the need for emergent laparotomy. Availability of oxygen carrying blood substitutes can revolutionize the resuscitation procedure in massive blood loss. The advent of sublingual capnometry will help to assess adequacy of perfusion to tissues during resuscitation and use of near-infrared spectroscopy can indicate adequacy of oxygen supply to the brain to guide the ICU management.

REFERENCES

1. Advanced Trauma Life Support for Doctors. Student Course Manual (8th edn). Chicago, American College of Surgeons. 2008.
2. Richards JR, Acosta JA, Wilson WC. Abdominal Trauma. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma-Emergency Resuscitation, Perioperative Anesthesia, Surgical Management. Volume I, New York: Informa Healthcare 2007;517-31.
3. Potenza B, Nolan J. Mechanisms and Epidemiology of Trauma. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma- Emergency Resuscitation, Perioperative Anesthesia, Surgical Management. Volume I, New York: Informa Healthcare 2007;25-42.
4. Simeone AA, Frankel HL and Velmahos G. Abdominal Injury. In: Peitzman AB, Rhodes M, Schwab CW, Yealy DM, Fabian TC (Eds). The trauma Manual: Trauma and acute care surgery (3rd edn). New Delhi: Lippincott Williams and Wilkins 2008;243-72.
5. Fabian TC, Bee TK. Liver and Biliary Tract. In: Feliciano DV, Mattox KL, Moore EE (Eds). Trauma (6th edn). New York: McGraw Hill 2008:637-60.
6. Harbrecht BG, Forsyth RM, Peitzman AB. Management of Shock. In: Feliciano DV, Mattox KL, Moore EE (Eds). Trauma (6th edn). New York: McGraw Hill 2008;213-34.
7. Rodriguez-Morales G, Rodriguez A, Shatney CH. Acute rupture of the diaphragm in blunt trauma: Analysis of 60 patients. J Trauma 1986;26:438-44.
8. Wilson WC. Anesthesia Considerations in Abdominal Trauma. In: Smith CE (Ed). Trauma Anesthesia. New York: Cambridge University Press 2008;155-71.
9. Richards JR, Schleper NH, Woo BD, Bohnen PA, MCGahan JP. Sonographic assessment of blunt abdominal trauma: A 4-year prospective study. J Clin Ultrasound 2002;30:59-67.
10. Logan P, Lewis D. Focussed Assessment with Sonography in Trauma. Emergency Ultrasound UK 2004.
11. Kornezos I, Chatziioannou A, Kokkonouzis I, et al. Findings and limitations of focused ultrasound as a possible screening test in stable adult patients with blunt abdominal trauma: A Greek study. European Radiology. 2010;20:234-8.
12. Amer MS, Ashraf M. Role of FAST and DPL in assessment of Blunt Abdominal Trauma. Professional Med J 2008; 15:200-04.
13. Radwan MM, Abu-Zidan FM. Focussed Assessment Sonography Trauma (FAST) and CT scan in blunt abdominal trauma: Surgeon's perspective. Afr Health Sci 2006;6:187-90.
14. Dutton RP. Shock Management. In: Smith CE (Ed). Trauma Anesthesia. New York: Cambridge University Press 2008;55-68.
15. Boldt J. Fluid choice for resuscitation of the trauma patient: A review of the physiological, pharmacological and clinical evidence. Can J Anesth 2004;51:500-13.
16. Reines HD and Wulf-Gutierrez ME. Clinical review: Hemorrhagic Shock. Critical Care 2004;8:373-81.

17. Kreimeier U, Prueckner S, Peter K. Permissive Hypotension. Schweiz Med Wochenschr 2000;130:1516-24.
18. Funk W, Baldinger V. Microcirculatory perfusion during volume therapy. A comparative study using crystalloid or colloid in awake animals. Anesthesiology 1995;82: 975-82.
19. Novikov M and Smith CE. Fluid and Blood Therapy in Trauma. International Trauma Care 2008;18:42-56.
20. Boldt J. Fluid Choice for Resuscitation in Trauma. International Trauma Care 2008;18:57-65.
21. Kozek-Langenecker SA. Effects of Hydroxyethyl Starch Solutions on Hemostasis. Anesthsiology 2005;103:654-60.
22. Lang K, Boldt J, Suttner S and Haisch G. Colloids versus Crystalloids and Tissue Oxygen Tension in Patients Undergoing Major Abdominal Surgery. Anesth Analg 2001; 93:405-09.
23. Dutton RP, Howard RS. Fluid Resuscitation Strategy. In: Wilson WC, Grande CM, Hoyt DB (Eds) Trauma-Emergency Resuscitation, Perioperative Anesthesia, Surgical Management. Volume I, New York: Informa Healthcare 2007;215-34.
24. Randolph LC, Takacs M, Davis KA. Resuscitation in the Pediatric Trauma Population: Admisssion Base Deficit Remains an Important Prognostic Indicator. J Trauma 2002;53:838-42.
25. Kaplan LJ, Bailey H. Ongoing Resuscitation Endpoints and Strategies. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma-Emergency Resuscitation, Perioperative Anesthesia, Surgical Management. Volume I, New York: Informa Healthcare 2007;353-63.
26. Mandavia DP, Hoffner RJ, Mahaney K, Henderson SO. Bedside Echocardiography by Emergency Physicians. Annals of Emergency Medicine 2001;38:377-82.
27. Johnson K and Egan TD. Blood Loss: Does it change my Intravenous Anesthetic? In: Smith CE, (Ed). Trauma Anesthesia. New York: Cambridge University Press 2008; 133-41.
28. Johnson KB, Egan TD, Kern SE, et al. The influence of hemorrhagic shock on propofol: A pharmacokinetic and pharmacodynamic analysis. Anesthesiology 2003;99: 409-20.
29. Johnson K, Egan T, Kern S, McJames S, Cluff M, Pace N. The influence of hemorrhagic shock followed by crystalloid resuscitation on propofol: A pharmacokinetic and pharmacodynamic analysis. Anesthesiology 2004;101:647-59.
30. Shafer SL. Shock Values. Anesthesiology 2004;101:567-8.
31. Johnson KB, Egan TD, Layman J, Kern SE, White JL, McJames SW. The influence of hemorrhagic shock on etomidate: A pharmacokinetic and pharmacodynamic analysis. Anesth Analg 2003;96:1360-68.
32. Black I, Grathwohl K, IB T, Martini W, Johnson K. The influence of hemorrhagic shock on ketamine: A pharmacokinetic analysis. Anesthesiology 2006;105:A203.
33. Duke JC. Anesthesia. In: Feliciano DV, Mattox KL, Moore EE, (Eds) Trauma. 6th (edn) New York: McGraw Hill 2008: 339-61.
34. Egan TD, Kuramkote S, Gong G, Zhang J, McJames SW, Bailey PL. Fentanyl pharmacokinetics in hemorrhagic shock: A porcine model. Anesthesiology 1999;91:156-56.
35. Johnson KB, Kern SE, Hamber EA, McJames SW, Kohnstamm KM, Egan TD. Influence of hemorrhagic shock on remifentanil; a pharmacokinetic and pharmacodynamic analysis. Anesthesiology 2001;94:322-32.
36. Kuipers JA, Boer F, Olofsen E, Bovill JG, Burm AG. Recirculatory pharmacokinetics and pharmacodynamics of rocuronium in patients: The influence of cardiac output. Anesthesiology 2001;94:47-55.
37. Rezende-Neto JB, Rizolli SB, Andrade MV, et al. Permissive hypotension and desmopressin enhance clot formation. J Trauma 2010;68:42-50.
38. Martinowitz U, Kenet G, Segal E, et al. Recombinant Activated Factor VII for Adjunctive Hemorrhage Control in Trauma J Trauma 2001;51:431-9.
39. Toschlog EA, Sagraves SG, Rotondo MF. Damage Control. In: Peitzman AB, Rhodes M, Schwab CW, Yealy DM, Fabian TC, (Eds). The trauma Manual: Trauma and acute care surgery (3rd edn). New Delhi: Lippincott Williams and Wilkins 2008;283-92.
40. Brookside Associates. Emergency war Surgery. 3rd United States Revision 2004. US Army 2004.
41. Anjaria DJ, Hoyt DB. Abdominal Compartment Syndrome. In: Wilson WC, Grande CM, Hoyt DB, (Eds) Trauma: Critical Care. Volume II. New York: Informa Healthcare 2007;619-32.
42. Ertel W, Oberholzer A, Platz A, et al. Incidence and clinical pattern of the abdominal compartment syndrome after "damage-control" laparotomy in 311 patients with severe abdominal and/or pelvic trauma. Crit Care Med 2000;28: 1747-53.
43. Balogh Z, McKinley BA, Cocanour CS, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg 2003;138:637-42.
44. Wisner DH, Victor DS, Holcroft JW. Priorities in management of Multiple Trauma: Intracranial versus Intra-Abdominal Injury. J Trauma 1993;35:271-6.

30 Anesthesia for Pelvic and Long Bone Fractures

Jyoti S Magar, Hema B Gupta

KEY POINTS

- Delay in the stabilization of fractures results in increased morbidity due to increased chances of infection, delayed mobilization, pulmonary complications and increased length of hospital stay.
- Hence, the emphasis in trauma management of a patient with multiple injuries includes early stabilization of long bone, spine, pelvic and acetabular fractures.
- One of the important predictors of mortality in pelvic fractures is the initial hemodynamic status.
- Management's goal in these patients is to prevent early death resulting from exsanguinations and later mortality due to sepsis and multiple organ failure.
- Bleeding from sacral venous plexus can be extensive with blood loss of up to 3 to 10 liters. The retro peritoneum of the intact pelvis can hold 4 liters of blood before tamponade occurs.
- The pubic symphysis may fracture and widen leading to "open–book" pelvic fractures. In these cases a high index of suspicion for significant bleeding is essential.
- In vascular injuries, early operative revascularization is essential, as muscle and nerves are very sensitive to anoxic environment. Muscular necrosis and loss of neurological function will begin within 6 hours of ischemia.
- In patients with pelvic trauma, intravenous access should be obtained in the drainage area of superior vena cava as there may be possible disruption of the pelvic veins or inferior vena cava. Therefore, fluids administered through lower extremity intravenous access may not enter the central circulation.
- An effective method for fixing the pelvic ring is with circumferential wrapping with a sheet or belt encircling the hips at the level of the greater trochanter.
- The concept of damage–control orthopedics has emerged. In this, patients are treated with temporary stabilizing surgeries, instead of longer definitive procedures, thereby avoiding prolonged duration of surgery and greater blood loss.
- Patients with hip fracture are often dehydrated and anemic because the fracture site can accommodate a large amount of extravasated blood.
- Regional blocks should be used cautiously in patients with acute fractures and extremity trauma who are at risk of developing compartment syndrome.
- The triad of hypothermia, coagulopathy, and acidosis may be lethal for the patient.
- Methylmethacrylate cementing may lead to arterial oxygen desaturation, hemodynamic instability, cardiac and pulmonary complications and, even, cardiac arrest.
- Fat embolism syndrome (FES) is a well-known complication of skeletal trauma and surgeries involving instrumentation of femoral medullary canal. The presentation of FES can be gradual, developing over 12 to 72 hours, or can be fulminant leading to ARDS and cardiac arrest.
- The risk of deep venous thrombosis (DVT) is as high as 60 percent in trauma patients with pelvic fractures. Venous thromboembolism is a major cause of death after surgery or trauma to the lower extremities.
- Pain relief is an important aspect in the management of patients with orthopedic trauma. Uncontrolled and constant pain contributes to continued immobility leading to thromboembolic events, pulmonary complications, and even death.
- Femoral nerve block can provide significant perioperative analgesia. Placing a femoral nerve block prior to moving a patient to the surgical table can considerably decrease pain on movement.

Injuries to the musculoskeletal system occur in 85 percent of patients who sustain blunt trauma. Even when they do not pose acute threat to life or limb, they should be assessed and managed properly to avoid further damage to life or limb.[1]

Delay in the stabilization of fractures results in increased morbidity due to increased chances of infection, delayed mobilization, pulmonary complications and increased length of hospital stay. Hence, the emphasis in trauma management of a patient with multiple injuries includes early stabilization of long bone, spine, pelvic and acetabular fractures.[2] A study was conducted in patients with multiple musculoskeletal injuries, where the relationship between the length of time from injury to the operative stabilization of major fractures and the incidence of ARDS was studied. This study showed that a delay in orthopedic surgery of greater than 24 hours is associated with a fivefold increase in the incidence of ARDS.[3]

PELVIC FRACTURES

Pelvic fractures remain a challenge for all those involved in their assessment and management. According to a study conducted by Mucha et al,[4] the overall mortality resulting from pelvic fractures was about 6.4 percent. One of the important predictors of mortality is the initial hemodynamic status. Patients who were hemodynamically stable on arrival had a death rate of 3.4 percent compared with 42 percent in patients who had arrived hemodynamically unstable. Apart from hemorrhage from pelvic vessels, other identifiable causes of death in patients with pelvic fractures include other associated injuries particularly closed head injury and sepsis.[5] Hence, management goals in these patients is to prevent early death resulting from exsanguinations and later mortality due to sepsis and multiple organ failure.

The pelvis is a ring structure made up of three bones: The sacrum, and the two innominate bones. The pelvic ring is formed by the connection of the sacrum to the innominate bones at the sacroiliac joints and the pubic symphysis. Factors important in maintaining pelvic stability are posterior sacroiliac complex, pelvic floor and pubic symphysis.[6] Because of the ring structure, if the pelvis is broken in one location, another fracture or dislocation may be present. The pelvic ring is much more solid than many other bone structures, and high energy trauma is required to disrupt this complex. Because of this, patients with pelvic fractures generally have injuries involving other organ systems.

High energy injuries are usually caused by motor vehicle accidents, motor cycle collisions or fall from heights. They may be associated with neurological injury, chest injury, abdominal trauma, urogenital injury, extremity fracture, and massive hemorrhage. The mortality rate in patients sustaining high energy trauma range from 15 to 25 percent.[7,8]

Low energy injuries may be isolated, and may be produced by a fall from a low height and is often seen in elderly, osteoporotic patients.

Vascular supply of pelvis is composed of four interconnected collateralized arterial loops. The veins are arranged in a large plexus, closely applied to the pelvic walls. They are valve less, thereby allowing bidirectional flow. The pelvic venous system is extensive; therefore significant hemorrhage can occur following disruptions, even at normal venous pressures.[8]

If the integrity of the pelvic floor is compromised, then blood leaks in large volumes into the retroperitoneal space.

Bleeding in pelvic fractures may be from:
1. Fractured bony surfaces.
2. Pelvic arterial injury.
3. Pelvic venous plexus injury.

Bleeding from sacral venous plexus can be extensive with blood loss of up to 3 to 10 liters. The retroperitoneum of the intact pelvis can hold 4 liters of blood before tamponade occurs.[9]

Types of Pelvic Fractures[7,9,10]

Pelvic fractures are classified by the mechanism of injury, the force involved, and the disruption caused by the force into the following types:

a. Anteroposterior injuries due to direct force to the anterior pelvis or external rotation force to the hemipelvis through the femur. Commonly seen in head-on motor vehicle accidents, crush injuries or auto-pedestrian collisions. The pubic symphysis may fracture and widen leading to "open–book" pelvic fractures. In these cases, a high index of suspicion for significant bleeding is essential (Fig. 30.1).
b. Lateral compression injury due to lateral impact to the pelvis. Seen in motor vehicle accidents or fall on the side. This is the most common pelvic fracture pattern. The rotation of the hemipelvis drives the pubis into the lower genitourinary system, causing injury to the bladder or urethra. Because of the compression of the pelvic volume in these injuries, life-threatening hemorrhage is not common[1] (Fig. 30.2).
c. Vertical shear injuries due to axial force to the pelvis. They result from a fall from height or motorcycle accidents. Concomitant stretching of the lumbosacral

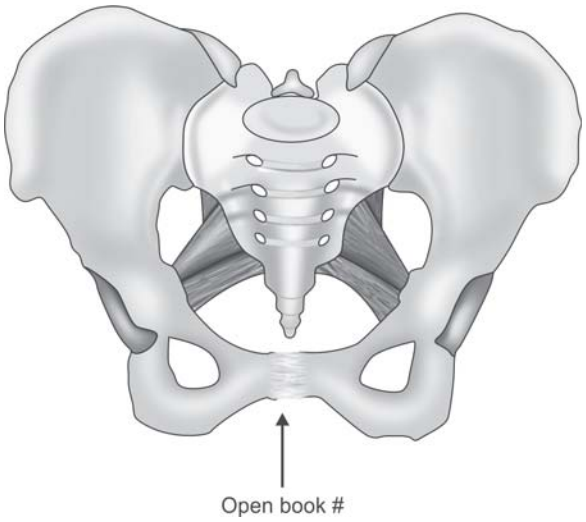


Fig. 30.1: Diagram of pelvic "open-book" fracture

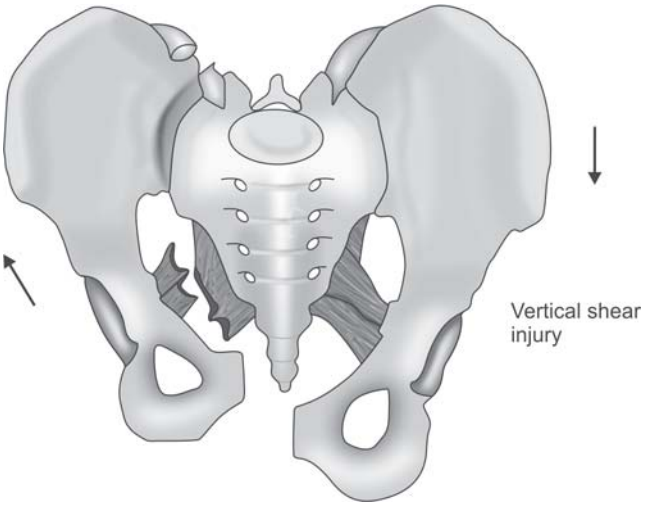


Fig. 30.3: Diagram of vertical shear injury

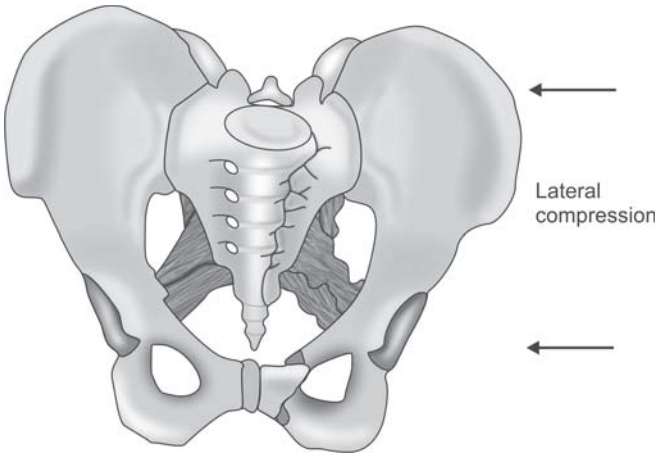


Fig. 30.2: Diagram of lateral compression injury

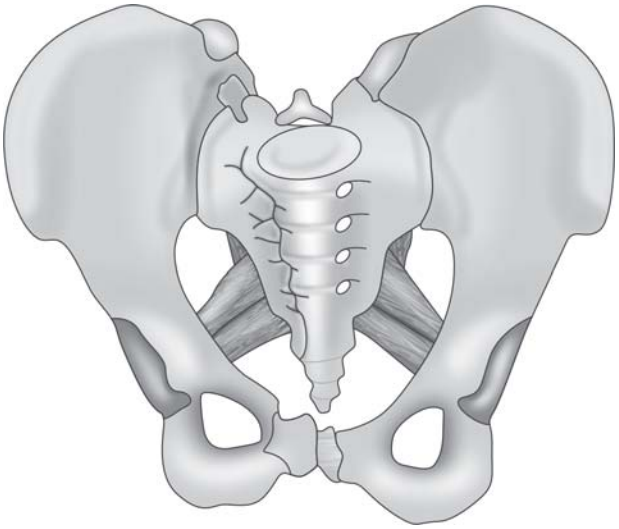


Fig. 30.4: Diagram of combined mechanical injury

plexus and vascular structures may be present (Fig. 30.3).

d. Combined mechanical injuries due to combination of forces. These injuries may be unstable (Fig. 30.4).

Open pelvic fractures are uncommon but devastating injuries, requiring massive blood transfusions. According to a study conducted by Brenneman et al[11] open pelvic fractures occur more commonly in young, male patients, more likely to have been involved in motorcycle crash and more likely to have unstable pelvic ring disruption. Since they are associated with major bleeding, control of hemorrhage followed by debridement and packing of open wounds is necessary.

ASSOCIATED INJURIES

Urogenital Injury

Rupture of the bladder and urethra occurs in 20 percent of patients with a fractured pelvis and pubic rami fracture. Urethral injuries occur in up to 15 percent of men but are rare in women.[12] Bladder rupture may be intraperitoneal or extraperitoneal. Extraperitoneal injury occurs more commonly and is due to laceration of bladder from pelvic fractures. Intraperitoneal injury occurs mostly from contusion to the lower abdomen or the symphyseal region. It may also occur due to pelvic ring disruptions as a result of seatbelt or steering wheel injury. It requires prompt diagnosis and treatment in the

form of suprapubic drainage or laparotomy to avoid complications such as hyperkalemia, hypernatremia, uremia, acidosis and peritonitis. Urogenital injury is promptly diagnosed by an urethrocystogram. In patients at high risk of urogenital injury, as indicated by blood at the meatus or high-riding prostate on rectal examination, retrograde urethrograms may have to be performed prior to instrumentation of urethra.

Anorectal Injury

Anorectal injuries occur more commonly in association with open pelvic fractures. They may include lacerations of the rectum and perforations of small or large bowel. Since these are open fractures, they require urgent surgery in the form of laparotomy and diverting colostomy. Delay in obtaining fecal diversion may lead to contamination of the fracture site resulting in sepsis.[12]

Neurological Injury

The sciatic nerve is particularly vulnerable to damage with acetabular fractures associated with posterior dislocation of the hip.[12] Careful documentation of the presence of a complete or partial foot drop with any associated numbness is important for clinical as well as medico legal reasons.

LONG BONE FRACTURES

Multiple long bone fractures are usually associated with high energy trauma and may be accompanied by injuries to the other organ systems. Isolated extremity fractures may occur with low energy trauma, such as minor fall, in elderly patients.

Life-threatening hemorrhage can occur with bilateral femoral shaft fractures or multiple long bone fractures.[9] Patients with bilateral femoral fractures have a significantly higher risk of death, ARDS and associated injuries than patients with unilateral femoral fractures[13] (Table 30.1).

Open Fractures

Open fractures occur most commonly as a result of high energy trauma and may be associated with injury to other systems. Open fractures are classified by their associated soft tissue injury and level of contamination. As the severity increases, so does the risk for infection and other complications.[14]

Table 30.1: Relationship of average blood loss and the bone fractured

Bone fractured	*Average blood loss*
Femoral shaft fracture	1500 ml
Humerus shaft fracture	750 ml
Tibia shaft fracture	750 ml

Traumatic Amputation

It is a severe form of open fracture resulting in loss of an extremity. Certain open fractures with prolonged ischemia, neurologic injury, and muscle damage may require amputation. Amputation may also have to be carried out as a life saving measure in patients with hemodynamic instability, who are difficult to resuscitate.[1]

Vascular Injury

A vascular injury should be strongly suspected in the presence of vascular insufficiency associated with a history of blunt, crushing, twisting, or penetrating injury to an extremity. More than 75 percent occur from penetrating trauma.[9] Clinical signs in patients with a major arterial injury include pallor, cold limb and decreased and/or absent pulses in an extremity. When a pulse is palpable or dopplerable but asymmetrical, vascular injury is suspected. Ankle brachial indices (ABIs) should be obtained in these patients. The ABI is the ratio of the systolic pressure of the ankle over the systolic pressure of the arm. If an ABI is less than 0.9 an arteriogram is indicated.[15] In vascular injuries early operative revascularization is essential, as muscle and nerves are very sensitive to anoxic environment. Muscular necrosis and loss of neurological function will begin within 6 hours of ischemia.

PRIMARY SURVEY AND RESUSCITATION

Primary survey and resuscitation are initially carried out as per ATLS protocols which include, airway, breathing, circulation, disability and exposure.

Because pelvic and bilateral femur fractures may be responsible for massive hemorrhage; once the airway and breathing are established, control of hypovolemic shock should be carried out aggressively.

The important physical signs associated with pelvic hemorrhage are progressive flank, scrotal or perianal swelling and bruising. Open fracture wounds around the pelvis, blood at the urethral meatus and demonstrable mechanical instability are signs of unstable pelvic ring injury. Mechanical instability of the pelvic ring is tested by manual manipulation of the pelvis.[1]

It is necessary to establish a venous access with minimum two 14 or 16 G intravenous cannulae. In patients with pelvic trauma intravenous access should

be obtained in the drainage area of superior vena cava as there may be possible disruption of the pelvic veins or inferior vena cava. Therefore, fluids administerd through lower extremity intravenous access may not enter the central circulation.[8]

Fluid resuscitation should be carried out with an initial volume of 2 to 3 liters of crystalloids. Further management depends on whether the patient responds to the initial fluid challenge. If no response is seen, attempts should be made for provisional reduction of the fracture. Provisional reduction of the pelvic fractures provides early stabilization of the pelvic ring, thereby reducing hemorrhage. An effective method for fixing the pelvic ring is with circumferential wrapping with a sheet or belt encircling the hips at the level of the greater trochanter. This is mainly effective in reducing bleeding of venous origin.[16]

In spite of the above measures, if patient remains hemodynamically unstable, external fixation of the pelvis can be done to immobilize the fracture sites to achieve hemostasis. For unresponsive patients, laparotomy may be required for surgical hemostasis.

In cases of major arterial injury, emergency angiography and embolization may prove to be life saving. Aim here is to achieve immediate vascular occlusion by decreasing pressure upstream from the leak; this facilitates physiological hemostasis, thrombus formation and healing of the dissected vessels.[17]

Similarly, hemorrhage from long bone fractures can be significant. Appropriate splinting should be done as it decreases bleeding by reducing motion and enhancing a tamponade effect of the muscles. The proper application of a splint helps to control blood loss, reduces pain and prevents further soft tissue injury.[1]

For management of unstable pelvic injury, refer to Figure 30.5.

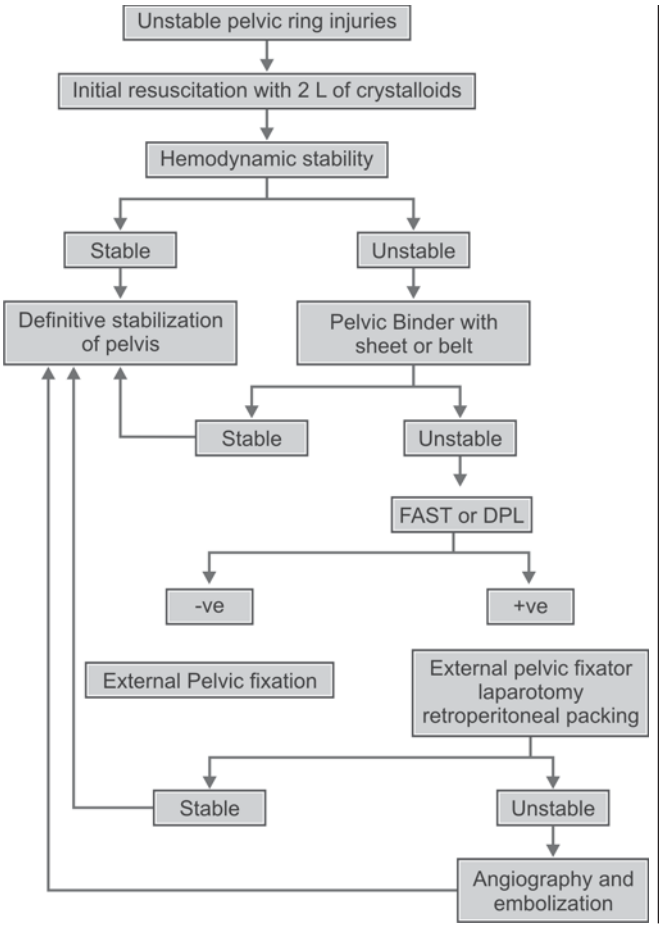


Fig. 30.5: Schematic plan of management of unstable pelvic ring injury

Radiographic Evaluation

Radiographs required for pelvic ring fractures include A-P pelvis, inlet view, outlet view[8] (Figs 30.6 to 30.8).

For diagnosis of urogenital injury, a micturating cystourethrogram is useful.

CT scan is a routine imaging study obtained on most pelvic fractures. It helps to classify the fracture and plan fixation. Also it will reveal associated injury to pelvic and abdominal structures in addition to acetabular and femoral head and neck injuries.[18]

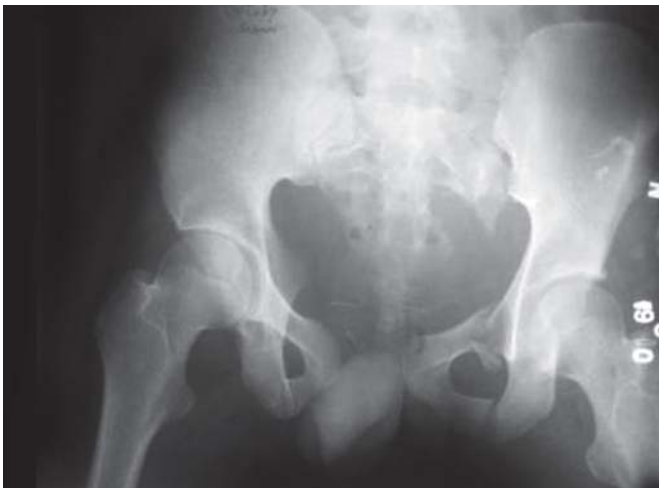


Fig. 30.6: Anteroposterior radiograph of pelvis fracture

DEFINITIVE MANAGEMENT

The concept of early fracture fixation was introduced in the 1980's, when La Duca et al[19] published a report

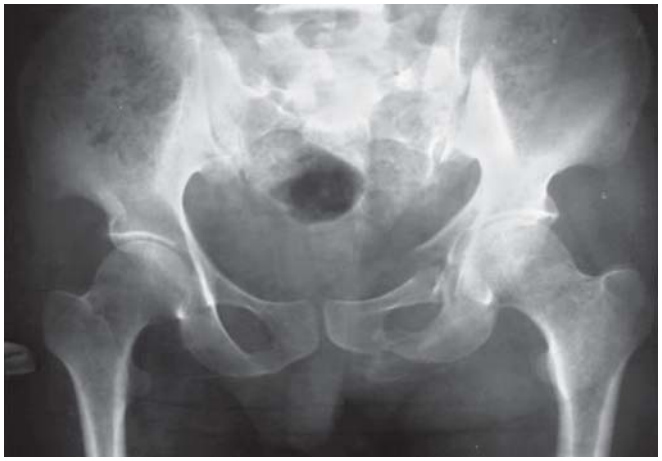


Fig. 30.7: Radiograph of lateral compression injury

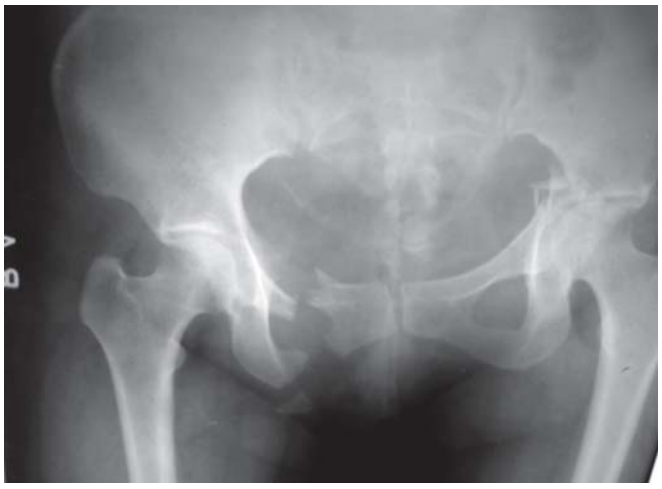


Fig. 30.8: Radiograph of vertical shear injury

describing only a 4 percent incidence of infection and no cases of fat embolism or post-traumatic cardiopulmonary failure in 42 patients with 50 open fractures undergoing immediate repair. Another similar study was carried out by Johnson et al.[3] These studies demonstrated advantages of early fracture fixation including decreased pain, improved mobility, leading to decreased incidence of fat embolism and pulmonary complications. Also, chances of wound infection and sepsis are reduced. Some fractures such as displaced femoral neck fracture may lead to avascular necrosis of femoral head necessitating early fixation.

Indications for and Timing of Fixation of Pelvic and Long Bone Fractures[9]

Injuries that are immediately threatening to life and limb should be dealt with on an emergent basis.

Emergency Procedures

- Life-threatening injuries
  - Pelvic ring injuries associated with massive hemorrhage not responding to fluids.
  - Bilateral femoral shaft fracture and multiple long bone fractures with massive hemorrhage.
- Limb threatening injuries
  - Traumatic amputation—emergent surgery is recommended to examine the affected area, to perform surgical debridement and to achieve hemostasis.
  - Vascular injury—when an extremity remains pulseless despite reduction and splinting, emergency surgical exploration is essential to preserve the limb.
  - Compartment syndrome.

Urgent Procedures

- Surgery within 6 to 8 hours
  - Open fractures—after a delay of 6 to 8 hours infection rates begin to rise, hence debridement and irrigation is recommended. If possible this should be combined with provisional or definitive fixation of the fracture.
  - Traumatic arthrotomy—in these cases also, surgical debridement and irrigation of the joint are recommended to reduce the risk of infection.
  - Dislocations—a dislocated joint should be anatomically reduced as soon as possible as it is associated with significant pain and deformity and impairment of motion. Hip dislocations require emergent reduction to decrease the risk of avascular necrosis.
  - Displaced femoral neck fracture in young adult—these fractures need immediate management in order to decrease the potential for osteonecrosis of the femoral head which may lead to advanced arthrosis of the hip joint.
- Surgery within 24 hours
  - Unstable pelvis/acetabulum or femur fracture—as described above, early fixation of these fractures is associated with a decreased incidence of pulmonary complications and infection rates.
  - Proximal femoral fracture in elderly patient.
  - Femoral neck fracture.
  - Inter trochanteric fracture.

Associated Injuries

Though the recent emphasis in trauma management is on early fracture fixation, due consideration should be given to the presence of other associated injuries;

especially head and chest trauma. Severely injured patients with massive hemorrhage develop a systemic inflammatory response syndrome, which is described as the first hit. If the patient is not adequately resuscitated, orthopedic fracture fixation may result in a secondary insult leading to multiple organ failure. Hence, the concept of damage—control orthopedics has emerged. In this, the patients are treated with temporary stabilizing surgeries, instead of longer definitive procedures, thereby avoiding prolonged duration of surgery and greater blood loss, e.g. Provisional external fixation for fracture femur instead of intramedullary nailing.[20,21] Even in patients with coexistent chest trauma, early femur fixation (< 24 hours) is associated with an improved outcome.[22,23]

Associated Head Injury[24]

In patients with combined head injuries and orthopedic injuries, consultation is required among the trauma surgeon, neurosurgeon, orthopedic surgeon and anesthesiologist before the surgery for appropriateness of orthopedic management and intracranial pressure monitoring. Patients with minor head injury (GCS 14-15) with normal CT scan can be taken up for appropriate fixation. Other patients (i.e. GCS 11-13 with normal CT scan or GCS 11-15 with CT scan findings) may be taken up for urgent orthopedic injuries with intraoperative ICP monitoring. Patients with severe head injury (GCS 3-10) may be taken up with ICP monitoring but minimum tolerable intervention should be carried out, such as, fasciotomy, fracture reduction, rapid external fixation. In case ICP becomes unstable, surgery should be terminated and head CT scan should be obtained.[25]

ANESTHETIC CONSIDERATIONS

Preoperative Assessment and Work-up

Preoperative evaluation of the trauma patient requires knowledge and comprehensive understanding of the mechanism of injury, other known and potential collateral injuries, patient's premorbid condition and current physiological status. Adequacy of the primary resuscitation should be judged. A note should be made of the Glasgow Coma Scale. A pupillary examination and evaluation of gross sensory and motor status should be determined. Documentation of the neurological deficits should be done prior to the administration of sedatives or neuromuscular blocking drugs. Hypovolemia should be managed with adequate volume resuscitation prior to taking up for surgery, whenever possible. If possible a medical history is obtained from the patient or family members regarding past medical conditions, medications, allergies, etc.[24]

Patients with hip fracture are often dehydrated and anemic because the fracture site itself can accommodate a large amount of extravasated blood. Due to the dehydration and blood loss hematocrit values may be normal in these patients as a result of hemoconcentration. Therefore, a normal intravascular volume should be restored before anesthesia and surgery.[26]

The standard laboratory investigations in trauma patients include hemoglobin, platelet count, arterial blood gas, prothrombin time, international normalized ratio, activated partial thromboplastin time, biochemical investigations, urinanalysis, and blood group and cross match.[24]

In the case of life threatening injury requiring emergency surgery, time may not be available for a detailed evaluation and preparation of the patient. In these cases after initial survey and resuscitation, samples must be drawn for the laboratory investigations prior to taking up the patient.

When trauma patient is stable, a detailed preoperative evaluation should be carried out. All the required investigations should be checked. Appropriate intravenous access should be obtained and hypovolemia or anemia, if present, need to be corrected. Perioperative management plan should be formulated.

Choice of Anesthesia

The choice of regional or general anesthesia depends upon the patent's general condition, associated injuries, patient preference, ability to give informed consent, duration of the surgery, surgical preference, expected blood loss, and risk of development of compartment syndrome.

In case of pelvic fractures, general anesthesia is preferred as these injuries are often associated with major blood loss with hemodynamic instability. Also, regional block may be difficult to perform because of the patient's inability to lie in proper position. Another factor to be considered is the presence of nerve damage, which may be present from the time of accident or caused by surgery. There is a potential for compartment syndrome, the symptoms and signs of which may be masked by a regional block.[12]

Regional techniques are most appropriate for isolated extremity injuries, in cooperative and stable patients. Whenever significant hypovolemia is present or hemorrhage is expected, central neuraxial blocks are relatively contraindicated because of the high likelihood of associated sympathetic blockade and subsequent hypotension. Regional blocks should be used cautiously in patients with acute fractures and extremity trauma who are at risk of developing compartment syndrome.[27] In patients requiring amputation, there may be severe

hemorrhage leading to coagulopathy caused by consumption of coagulation factors. In such cases general anesthesia is preferable over regional anesthetic techniques. The type of hip fracture may help to determine the anesthetic technique. Procedures such as plating for intertrochanteric fractures may require longer duration, more manipulation of the leg, and greater blood loss. In these cases a continuous epidural technique may be required.

Monitoring

Intraoperative monitoring consists of electrocardiogram, pulse oximetry, capnometry, urine output, central venous pressure, arterial blood pressure monitoring (especially in unstable pelvic ring injuries).

Intravenous Access

Minimum of two large bore intravenous cannulae; preferably 14 or 16 G should be placed. Lower extremity intravenous access should be avoided in patients with pelvic trauma. Central venous access should be obtained in one of the veins draining into the superior vena cava. Whenever possible care should be taken to avoid fractured or potentially fractured extremities when inserting intravenous cannulae or monitoring lines.

Full Stomach

All trauma patients are considered to be full stomach and must undergo intubation with a rapid sequence induction and cricoids pressure must be applied.

Cervical Spine

Till the cervical spine is completely cleared, care must be taken to prevent any further damage; hence intubation should always be carried out with manual-in-line stabilization.

Induction Agents

In patients undergoing emergency surgery for hemorrhage control, any intravenous anesthetic may precipitate profound hypotension and even cardiac arrest because of interruption of compensatory sympathetic outflow.[8]

Ketamine continues to be popular for induction because it causes release of catecholamines. But caution is required as in hypovolemic patients this release might be attenuated and direct myocardial depressant effect may lead to cardio vascular collapse.

Etomidate minimally affects the cardiovascular system and hence is frequently used in trauma patients, especially if patients are presumed to be hemodynamically unstable.

Propofol is known to cause a fall in the systolic, mean and diastolic blood pressures, hence it must used with caution in the hypovolemic patient.[28]

Thiopentone may be acceptable induction agent in patients with combined head and orthopedic injuries, but care should be taken to maintain the mean arterial pressure in the normal range.

Intraoperative Concerns

Major Blood Loss

As pelvic fractures may be associated with blood loss greater than 2.5 L, it is necessary to keep adequate cross matched blood ready. It may be necessary to infuse large volumes of crystalloids, colloids, blood and blood products.

Hypothermia

All measures should be taken to keep the patient warm. Hypothermia can lead to coagulopathy, decreased platelet aggregation and acidosis.[29] The triad of hypothermia, coagulopathy and acidosis may be lethal for the patient.[30] It is important to cover the patients with warm blankets and minimise unnecessary patient exposure. Warming blankets, intravenous fluid warmers and transfusion devices are useful for this purpose.

Neurological Monitoring

It may be necessary to perform intraoperative neurological monitoring when nerve entrapment or damage is possible. Integrity of sciatic nerve may be checked using evoked potential monitoring. Because of interference with evoked potential tracings, benzodiazepine medications and high concentrations of inhalational agents should be avoided.[31] A nitrous oxide, oxygen, narcotic, muscle relaxant technique may be used. But in cases where nitrous oxide may be contraindicated, such as associated head, chest and abdominal trauma, an infusion of propofol or low dose inhalational technique may be used.

Methylmethacrylate Cement

Patients with hip fractures who undergo cemented hemiarthroplasty or total joint replacement are at risk of complications of methylmethacrylate cement. Cementing may lead to arterial oxygen desaturation, hemodynamic instability, cardiac and pulmonary complications and,

even, cardiac arrest. The mechanism of impaction of cement into the femoral shaft can force fat and marrow emboli, as well as thromboplastic elements into the circulation. It is necessary to monitor the preoperative oxygen saturation of the patient and provide supplemental oxygen during the procedure. Clinical suspicion, arterial blood gas analysis and echocardiographic evaluation can aid in the diagnosis and management of emboli before their disastrous consequences occur.[29]

Tourniquet

Tourniquets are often used to minimize blood loss and provide a bloodless operating field. In general, a cuff pressure 100 mm Hg above a patient's measured systolic pressure is adequate for the thigh, and 50 mm Hg above systolic pressure is adequate for the arm. The recommended duration for inflation of tourniquet is maximum 2 hours. Nerve injury after extended tourniquet inflation has been attributed to the combined effects of ischemia and mechanical trauma.

Limb exsanguination and tourniquet inflation causes an increase in cardiac preload and afterload. This may not be well tolerated in patients with diminished cardiac reserve. After deflation as blood re-enters the affected extremity, preload decreases which is accompanied by an acute decrease in the afterload often producing hypotension.[32] Transient systemic metabolic acidosis and increased arterial carbon dioxide levels have been demonstrated after tourniquet deflation, and do not cause deleterious effects in healthy patients.[33,34] Measurable changes include a 10 to 15 percent increase in heart rate, a 5 to 10 percent increase in serum potassium, and a rise of 1 to 8 mm Hg in carbon dioxide tension in blood.[35] The rise in carbon-dioxide production is compensated for by increase in minute ventilation, but this can produce deleterious effects in patients with raised intracranial pressure due to increase in cerebral blood flow.

Drugs administered before tourniquet inflation can become sequestered in the ischemic limb and a bolus of that particular drug may be delivered to the central circulation after tourniquet deflation. Antibiotic administration must be coordinated with tourniquet inflation for adequate penetration at the surgical site. It is necessary to inject the antibiotic prior to the inflation of the tourniquet.[32]

Tourniquet pain presents as dull aching pain which occurs in spite of apparently adequate level of anesthesia. The postulated mechanism is differential conduction block of large myelinated A-δ fibers and small unmyelinated C fibers. The treatment for this pain is tourniquet deflation.

Fat Embolism Syndrome

Fat embolization is a well known complication of skeletal trauma and surgeries involving instrumentation of femoral medullary canal. Fat embolism syndrome (FES) is a physiological response to fat within the systemic circulation. Incidence of FES in isolated long bone fractures is 3 to 4 percent and mortality associated with this condition ranges from 10 to 20 percent.[35] The presentation of FES can be gradual , developing over 12 to 72 hours, or fulminant leading to ARDS and cardiac arrest.

Gurd and Wilson suggested major and minor criteria to be used for diagnosis of FES. The major features include petechial rash, mainly axillary and subconjunctival, hypoxia, pulmonary edema and CNS depression. Minor features include pyrexia, tachycardia, retinal fat emboli, jaundice, urinary fat globules, fat microglobulinemia, anemia, thrombocytopenia and raised ESR. The presence of one major and four minor findings are required for diagnosis of FES.[26] Pulmonary hypertension and decreased cardiac function (right heart failure) may occur.

Treatment is early recognition of signs, oxygen administration, and reversal of aggravating factors such as hypovolemia, early surgical correction of fracture and ventilator support, if needed. Acute right heart failure may necessitate invasive monitoring, inotropes, and other vasoactive drugs. Although 10 percent of these patients may require mechanical ventilation, in most of these patients symptoms resolve within 3 to 7 days.[26] Corticosteroids have been used in the treatment of FES, but the beneficial role of corticosteroids needs to be further studied.

Compartment Syndrome

This occurs due to an increase in pressure within a relatively non compliant fascial compartment resulting in ischemia compromising nerve and muscle function. It is diagnosed by the pain out of proportion to the degree of injury, pain on passive motion and stretch pain. The only effective treatment for this condition is emergency surgical fasciotomy.[31]

Deep Vein Thrombosis and Pulmonary Thrombo-embolism

The risk of DVT is as high as 60 percent in trauma patients with pelvic fractures.[8] Venous thromboembolism is a major cause of death after surgery or trauma to the lower extremities. Without prophylaxis, venous thrombosis develops in 40 to 80 percent of orthopedic patients, and 1 to 28 percent show clinical or laboratory

evidence of pulmonary embolism.[35] Therefore, DVT prophylaxis with anticoagulants should be initiated as early as possible in the postoperative period.

Low molecular weight heparin (LMWH) is recommended over unfractionated heparin for initial therapy of DVT. Administration of LMWH 6 hours post operatively was effective in DVT prophylaxis without increasing chances of bleeding. This prophylaxis should be continued for at least 10 days in routine orthopedic cases, and may have to be extended to 28 to 35 days in patients with DVT or at high risk of DVT. Warfarin is used in the treatment of DVT maintaining a target INR of 2.5. Fondaparinux, a synthetic pentasaccharide may also be used as alternative to LMWH.[26]

Analgesia[29]

Pain relief is an important aspect in the management of patients with orthopedic trauma. Certain orthopedic procedures require mobilization immediately after surgery. Uncontrolled and constant pain contributes to continued immobility leading to thromboembolic events, pulmonary complications, and even death. In addition to the intravenous and intramuscular routes of administering analgesic drugs, various regional techniques can be used to provide analgesia. While using regional techniques, due consideration should be given to the presence of coagulopathy, use of anticoagulants for DVT prophylaxis and the risk of development of compartment syndrome.

Patient Controlled Analgesia

This system permits administration of a continuous background infusion superimposed on patient controlled boluses. By setting a loading dose, intermittent dose, lockout period, basal rate, and maximum dose per hour, the clinician can adjust the analgesia to the needs of the patient. This is advantageous in maintaining serum drug levels within the analgesic range and preventing fluctuation in levels, resulting in improved analgesia and patient satisfaction with minimum side effects. Only the patient should be permitted to initiate a bolus dose from a PCA device.

Continuous Epidural Analgesia

Analgesia delivered through an indwelling epidural catheter is a safe and effective method for management of pain in the postoperative period. Use of epidural analgesia can reduce the incidence of postoperative gastrointestinal, pulmonary and cardiac complications. Careful patient selection and vigilance is necessary, especially with regard to thromboprophylaxis. In patients with continuous infusion or patient controlled epidural analgesia careful monitoring of the neurological status is required.

Nerve Block Analgesia

Brachial plexus blocks are used for providing analgesia for the upper extremity. Interscalene block is utilized for postsurgical pain involving the shoulder down to the mid shaft of humerus. Infraclavicular blocks are used for pain below humerus mid shaft including the elbow, forearm and hand. Axillary blocks are used for pain involving the forearm and hand. Continuous analgesia can also be achieved using catheters placed via the above mentioned approaches.

For lower extremity pain involving the distribution of the femoral, obturator, and lateral femoral cutaneous nerves, lumbar plexus blocks can be used. Femoral nerve block can provide significant perioperative analgesia. Placing a femoral nerve block prior to moving a patient to the surgical table can considerably decrease pain on movement. Femoral and lateral femoral cutaneous nerve blocks can be utilized for femoral neck fractures. Combined femoral and sciatic nerve blocks are effective for surgeries of the knee or below.

REFERENCES

1. Advanced Trauma Life Support for Doctors, Student Course Manual, 8th edn, Chicago; American College of Surgeons: 2008.
2. Dutton RP, McCunn M, Grissom TE. Anaesthesia for trauma. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young W (Eds). Miller's Anesthesia, 7th edn. Philadelphia, PA: Churchill Livingstone Elsevier; 2009;2277-2312.
3. Johnson KD, Cadambi A, Seibert GB. Incidence of acute respiratory distress syndrome in patients with multiple musculoskeletal injuries. Effect of early operative stabilization of fractures, J Trauma 1985;25:375-84.
4. Mucha P, Farnell, Michael B. Analysis of pelvic fracture management. J Trauma 1984;24(5):379-86.
5. Naam NH, Brown WH, Hurd R, Burdge RE, Kaminski DL. Major pelvic fractures. Arch Surg 1983;118(5):610-6.
6. Vrahas M, Hern TC, Diangelo D, Kellam J. Tile M. Ligamentous contributions to pelvic stability. Orthopedics 1995;18(3):271-4.
7. Dalal SA, Burgess AR, Siegel JH, Young JW, Brunback RJ, Poka A, Dunham CM, Gens D, Bathon H. Pelvic fracture in multiple trauma: Classification by mechanism is key to pattern of organ injury, resuscitative requirements, and outcome. J Trauma 1989;29(7):981-1000.
8. Schwartz AK, Dreier J, Hughes TH, Mangar D, Camporesi EM. Pelvic trauma. In Wilson WC, Grande CM, Hoyt DB (Eds). Trauma Emergency Resuscitation Perioperative Anesthesia Surgical Management, Volume 1. New York Inc:.Informa healthcare; 2007;533-51.

9. Vallier HA, Jenkins MD. Musculoskeletal Trauma. In: Smith CE (Ed). Trauma Anaesthesia. New York: Cambridge University Press; 2008;225-44.
10. Burgess AR, Eastridge BJ, Young JW, Ellison TS, Ellison PS Jr, Poka A,Bathon GH, Brumback RJ. Pelvic ring disruptions: effective classification systems and treatment protocols. J Trauma 1990;30(7):848-56.
11. Brenneman FD, Katyal D, Boulanger BR, Tile M. Long-term outcomes in open pelvic fractures. J Trauma 1997; 42:773-7.
12. Umarji S, Bircher M. Pelvic and acetabular trauma. Recent Advances in Anesthesia and Intensive Care 24, Cambridge University Press 2007;87-107.
13. Copeland CE, Michell KA, Brumback RJ, Gens DR, Burgess AR. Mortality in patients with bilateral femoral fractures. J Orthop Trauma 1998;12(5):315-19.
14. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type 3(severe) open fractures: A new classification of type 3 open fractures. J Trauma 1984; 24(8): 742-6.
15. Mills WJ, Barei DP, McNair P. The value of the ankle-brachial index for diagnosing arterial injury after knee dislocation: A prospective study. J Trauma 2004;56(6): 1261-65.
16. Simpson T, Kreig JC, Heuer F, Bottlang M. Stabilization of pelvic ring disruptions with a circumferential sheet. J Trauma, 2002;52:158-61.
17. Velmahos GC, Toutouzas KG, Vassiliu P, Sarkisyan G, Chan LS, Hanks SH, Berne TV, Demetriades D. A prospective study on the safety and efficacy of angiographic embolisation for pelvic and visceral injuries. J Trauma 2002;53(2):303-8.
18. Guillamondegui OD, Pryor JP, Gracias VH, Gupta R, Reilly PM, Schwab CW. Pelvic radiography in blunt trauma resuscitation: A diminishing role. J Trauma 2002; 53(6):1043-47.
19. La Duca JN, Bone LL, Seibel RW, et al. Primary open reduction and internal fixation of open fractures. J Trauma 1980;20(7):580.
20. Pape HC, Grimme K, Van Griensven M, Giannoudis P, Morley J, Roise O, Elisabeth, Frank, Wiese B, Krettek C. Impact of intramedullary instrumentation versus damage control for femoral fractures on immunoinflammatory parameters. Prospective Randomized Analysis by the EPOFF study group. J Trauma 2003;55:7-13.
21. Nowotarski PJ, Turen CH, Brumback RJ, Scarboro JM. Conversion of external fixation to intramedullary nailing for fracture of the shaft of femur in multiply injured patients. J Bone Joint Surg Am 2000;82(6):781-8.
22. Brundage SI, McGhan R, Jurkovich GJ, Mack CD, Maier RV. Timing of femur fracture fixation: Effect on outcome in patients with thoracic and head injuries. J Trauma 2002; 52(2):299-307.
23. Handolin L, Pajarinen J, Lassus J, Tulikoura I. Early intramedullary nailing of lower extremity fractures and respiratory function in polytraumatized patients with a chest injury: A retrospective study of 61 patients. Acta Orthop Scand 2004;75(4):477-80.
24. Newhouse BJ, Laurent CS. Preoperative Evaluation, Preparation, and Monitoring. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma Emergency Resuscitation Perioperative Anesthesia Surgical Management, Volume 1. New York Inc:.Informa healthcare; 2007;325-52.
25. Tenenbein P, Kincaid MS, Lam AM. Head trauma- Anesthetic considerations and management. In: Smith CE (Ed). Trauma Anesthesia. New York: Cambridge University Press 2008;172-82.
26. Urban MK. Anesthesia for orthopaedic surgery. In: Miller RD (Ed). Miller's Anesthesia, 7th edn. Philadelphia, Pennsylvenia: Churchill Livingstone Elsevier; 2009;2241-60.
27. Agarwal S, Schwartz AK, Wilson WC, Jacoby IJ, Bramhall J, Acosta JA. Extremity Trauma. In Wilson WC, Grande CM, Hoyt DB. Trauma Emergency Resuscitation Perioperative Anesthesia Surgical Management, Volume 1. New York Inc:.Informa healthcare; 2007;553-72.
28. Claeys MA, Gepts E, Camu F. Hemodynamic changes during anesthesia induced and maintained with propofol. Br J Anaesth 1988;60(1): 3-9.
29. Conatiello RM, Rosenberg AD, Smith CE. Anesthetic considerations for orthopaedic trauma. In: Smith CE (Ed). Trauma Anesthesia. New York: Cambridge University Press; 2008;245-58.
30. Spahn DR, Rossaint R. Coagulopathy and blood component transfusion in trauma. Brit J Anaesth 2005; 95(2):130-39.
31. Rosenberg AD, Bernstein RL. Perioperative anesthetic management of orthopedic injuries. Anesthesiology Clinics of North America, 1999;17:171-80.
32. Martin G, Breslin D, Stevens T. Anesthesia for orthopedic surgery. In: Longnecker DE, Brown DL, Newman MF, Zapol WM (Eds). Anesthesiology. North Carolina: The McGraw- Hill Companies, Inc; 2008;1541-55.
33. Kadoi Y, Ide M, Saito S, et al. Hyperventilation after tourniquet deflation prevents an increase in cerebral blood flow velocity. Can J Anaesth 1999;46:259.
34. Bourke DL, Silberberg MS, Ortega R, Willock MM. Respiratory responses associated with release of intraoperative tourniquets. Anesth Analg 1989;69:541.
35. Horlocker TT, Wedel DJ. Anesthesia for orthopedic surgery. In: Barash PG, Cullen BF, Stoelting RK (Eds). Clinical Anaesthesia. 5th edn. New York Lippincott Williams & Wilkins 2006;1113-28.

31 Anesthesia for Patients with Crush Injury

Naina P Dalvi, Dhawal R Wadaskar

KEY POINTS

- As there is enormous increase in automobile utility, there is simultaneous increase in road traffic accidents and trauma cases
- Treatment of trauma victim starts at the trauma scene and during the time of extrication from trauma site.
- Early fluid resuscitation is the mainstay in management of crush injury
- Adequate fluid resuscitation, diuretics, sodium bicarbonate, mannitol is mainstay of treatment in crush injury patients
- Various electrolyte abnormalities like hyperkalemia, hypocalcemia, hypomagnesemia and hyperphosphatemia can occur in crush injury patients
- Because of myoglobin and various other toxins released in crush injury and associated hypovolemia, acute renal failure (ARF) is common in crush injury patients
- Compartment syndrome and rhabdomyolysis are not uncommon in crush injuries. High index of suspicion is necessary as clinical features can be misleading in cases with compartment syndrome and rhabdomyolysis.
- Crush injury patients generally come for debridement, amputation and fasciotomy. Role of early fasciotomy is still controversial
- Avoiding succinylcholine and nephrotoxic drugs, maintaining fluid balance, alkalotic pH and good urine output are the important anesthetic considerations in crush injury patients.

INTRODUCTION

Trauma victim care and management is a multidisciplinary area and require interdepartmental coordination. It was observed that, in road traffic accidents extremities were the most affected parts of the body (41% lower extremity and 21% upper extremity). Superficial injuries were found to be most common (47%), followed by fractures (20%), crush injuries (14%) and concealed injuries (12.4%).[1]

Major reason of morbidity and prolonged hospital stay after traffic accidents is musculoskeletal injury. Victims who survive after the major traumatic injuries can succumb to the various life threatening complications. Most of these injuries are directly related to the bone and soft tissue injuries.

Goal of an anesthesiologist is to address initial musculoskeletal insult and treat or avoid secondary complications. In this chapter, the primary resuscitation and anesthetic management of crush injuries and related complications have been dealt with.

As there is enormous increase in automobile utility; road/railway traffic accidents are the major reasons for crush injuries. Other reasons are fall from height, disaster situations, such as earthquakes, bombings, building collapse and mine accidents.

Every trauma patient has a possibility of crush injury and they should be searched for the same thoroughly. Early diagnosis and treatment of problems like hypovolemia, crush syndrome, rhabdomyolysis can avoid further metabolic, cardiovascular and renal complications. Thus early diagnosis and management of crush injury can modulate the overall outcome of a trauma patient.

PATHOPHYSIOLOGY OF CRUSH INJURY

Crush injuries are caused by continuous prolonged pressure on the body. It involves mainly lower and upper extremities. Chest, abdomen or face can also have crush injuries. The direct pressure after crush injury causes muscle cell to become ischemic. Continuous

pressure causes muscle damage resulting in an influx of fluid into the muscles resulting in edema and elevation in compartment pressure.[2] The cells then switch to anaerobic metabolism, generating large amounts of lactic acid. Prolonged ischemia then causes the cell membranes to leak.

Continuous inflow of fluids (edema fluids, bleeding) or too little outflow (venous obstruction) can cause increased pressure in the compartment. This increased compartment pressure will cause tissue pressure to increase more than total capillary pressure and ultimately cause capillary collapse. All this will cause increased diffusion of fluid from the intravascular space to the extravascular space and lead to further increase in compartmental pressure. This edema-outflow obstruction – more edema vicious cycle if continued will hamper oxygenation of muscle tissue. If not treated early this will proceed to tissue ischemia, muscle necrosis and nerve dysfunction, eventually irreversible cell damage/death.[3]

TOXINS RELEASED IN CRUSH INJURY

The toxin leak may continue for as long as 60 hours after the crush injury. Some of these substances and their consequences are listed in Table 31.1.[4,5]

CLINICAL PRESENTATION

- Pain is the main presenting symptom and it is deep and aching in nature and is worsened by passive stretching of the involved muscles. Pain can be out of proportion to the injury.[2]
- Immediately following extrication, a severe neurologic deficiency, mainly flaccid paralysis of the injured limb, may be present. Sensory loss to pain and touch is seen in a patchy pattern.
- Pallor, pulselessness and poikilothermia (hypothermia) may be present but they are usually late signs.[6]
- Limb edema is initially not present. Gross edema takes time to develop and can progress to compartment syndrome
- Distal pulses may be present even in the presence of gross edema. Investigation for additional injuries is warranted if pulses are not present
- Uncontrolled bleeding in mangled extremities may be present and it can lead to severe hypovolemic shock and death
- Even if skin and subcutaneous layers are not injured still the underlying muscles can be severely damaged
- Associated injuries elsewhere may be present
- Unlike the adult, the signs of hypovolemia or significant hemorrhage in a child are subtle and difficult to identify. The best early sign of hypovolemia in pediatric victims is a weak pulse as opposed to tachycardia in adults.

PREHOSPITAL CARE

- Prehospital care begins with first assessing trauma scene safety by the care provider. It is important to move the victim away from the trauma scene and to get medical aid as early as possible
- The primary focus for trauma resuscitation in the field is airway, breathing, circulation, (the ABCs of the primary survey) and spinal stabilization
- Shock, respiratory distress, and altered mental status are associated with high mortality and must be rapidly identified in the field with subsequent rapid transport to the nearest appropriate receiving center
- For entrapped victims, venous access can sometimes be established during extrication. For trauma victims in shock, venous access should be attempted during transport to a receiving center.[7]
- Resuscitation fluid of choice at trauma scene is Lactated Ringers solution. But if one suspects crush injuries then it is prudent to use isotonic saline for

Table 31.1: Toxins released after crush injury

Agent	*Effect*
Amino acids and other organic acids	Acidosis, Aciduria, Dysrhythmia
Creatinine phosphokinase (CPK)	Laboratory marker for crush injury
Free radicals, superoxides, peroxides	Secondary tissue damage
Histamine	Vasodilation, Bronchoconstriction
Lactic acid	Acidosis, Dysrhythmia
Leukotrienes	Lung injury
Lysozymes	Cellular injury
Myoglobin	Renal failure
Potassium	Renal failure, Dysrhythmia, Cardiac arrest
Thromboplastin	Disseminated intravascular coagulation

resuscitation fluid till victim reaches hospital as there are chances of fatal arrhythmias because of hyperkalemia

- Tying of obviously bleeding vessels or applying direct pressure bandages on crushed or mangled extremities can stop ongoing hemorrhage
- Application of tourniquets above the injured extremity can itself cause limb ischemia hence routine use of tourniquets should be avoided. Tourniquet can be used temporarily in victim who is actively bleeding so that hypovolemic shock can be avoided before reaching hospital
- Spinal stabilization, i.e. securing of a victim to a rigid spine support, of not only the cervical spine but whole spine is an important aspect of the prehospital care of trauma victims
- Remember that the aim of primary resuscitation is not to treat the trauma but stabilization of the patient till transfer to hospital for treatment.

EMERGENCY MANAGEMENT

Emergency management is aimed at stabilization of hemodynamic status, treatment of crush injury and prevention of its complications.

As patient arrives at EMS, airway, breathing, circulation and hemodynamic status should be checked. Secondary survey follows primary survey and associated injuries should be evaluated and treatment plan decided accordingly. Crush injury in more than one extremity should raise anticipation of crush syndrome. All routine investigations including serum electrolytes and urine for routine and myoglobin should be sent to laboratory after patient arrives at hospital.

STABILIZATION OF HEMODYNAMIC STATUS

Intravenous Fluids

The mainstay of treatment for crush injury is administration of intravenous fluids. At least two 14 or 16 G intravenous access should be established as soon as patient arrives at emergency area and fluid resuscitation should be started immediately. Initially a colloid or crystalloid such as normal saline is used. Potassium containing fluid, e.g. lactated Ringer's solution should be avoided in suspected crush injury patients as it may worsen hyperkalemia.[8] Once the patient is rescued from trauma site, it is critical to maintain a high urine output. Foley catheter placement is very important as it allows more accurate measurements of urine output as well as urine pH.

Treatment of Hyperkalemia

Mode of treatment used to treat hyperkalemia depends upon its severity. Administration of calcium gluconate is one of the fastest method to decrease blood potassium, but it will act only for a short period of time. Usual dose is 10 ml of 10 percent solution infused over 3 to 5 minutes.

Insulin will shift extracellular K^+ to intracellular side. Infusion of 50 g of dextrose combined with 10 units of insulin will decrease blood K^+ immediately and effect will last for some hours.

Sodium bicarbonate can be used in cases with severe hyperkalemia associated with metabolic acidosis.

Other modalities which can be used are β_2 agonists, loop diuretics, cation exchange resins like sodium polystyrene sulfonate and ultimately hemodialysis as last resort.[9]

Alkaline Diuresis

Alkalinization of urine will increase solubility of myoglobin and promote its excretion. It also prevents oxidative damage resulting from cycling of myoglobin by stabilizing the more reactive ferryl form. Sodium bicarbonate will reverse the pre-existing acidosis that is often present and also treat hyperkalemia. It will also increase the urine pH, thus decreasing the amount of myoglobin precipitated in the kidneys. Ion trapping via alteration of urine pH may prevent the renal reabsorption of poisons that undergo excretion by glomerular filtration and active tubular secretion. Since membranes are more permeable to nonionized molecules than to their ionized counterparts, acidic (low-pK_a) poisons are ionized and trapped in alkaline urine, whereas basic ones become ionized and trapped in acid urine. Urine output of 3-6 ml/kg/hr and urinary alkalinization by adding sodium bicarbonate to an IV solution enhances the excretion of acidic toxins. Infusion of 12 L/day of normal saline with 50 mEq of sodium bicarbonate per liter of fluid will maintain an alkaline urine output of 8 L/day.[8] This can be achieved with intravenous fluids, mannitol, and sodium bicarbonate and frusemide at 1 mg/kg. Acetazolamide, 250 to 500 mg, may be used if the patient becomes too alkalotic.

Alkalinization of urine is contraindicated in patients with congestive heart failure, renal failure, and cerebral edema. Acid-base, fluid, and electrolyte parameters should be monitored carefully. The patient with crush injury syndrome should maintain a urine output of at least 300 ml/h with a pH higher than 6.5.

Mannitol

Intravenous mannitol has several beneficial actions for the victim of crush injury. It protects the kidneys from the effects of rhabdomyolysis, increases extracellular fluid volume, and increases cardiac contractility.

Mannitol can be given in doses of 1 gm/kg added to the patient's intravenous fluid as a continuous infusion. The maximum dose is 200 gm/d; doses higher than this can cause renal failure. Mannitol should be given only after good urine flow has been established with IV fluids.

Mannitol should be avoided in patients with congestive heart failure and pulmonary congestion as it may cause frank pulmonary edema. It is contraindicated in patients with active cranial bleeding and in patients with anuria. Electrolyte monitoring is essential during mannitol administration as it will cause excretion of many electrolytes including Na^+, K^+, Ca^+, Mg^+, Cl^-, HCO_3 and phosphate.[10]

SURGICAL MANAGEMENT OF CRUSH INJURY

Wounds should be cleaned, debrided, and covered with sterile dressings in the usual fashion. Splinting the limb at heart level will help to limit edema and maintain perfusion. Application of the pneumatic anti-shock garment (PASG) should be avoided. The use of PASG has been reported to cause compartment syndrome and crush injury syndrome.[11] There are case reports of hyperbaric oxygen improving the outcome of victims of crush injury.[12]

Treatment of closed crush injuries is conservative. They should not be routinely explored since the intact skin acts as a barrier against infection. The use of fasciotomies is controversial. Routine use is not to be advocated. Fasciotomies will not reverse muscle necrosis in the absence of compartment syndrome.

COMPLICATIONS AFTER CRUSH INJURY

- Hemorrhage and shock
- Hypothermia
- Hyperkalemia
- Acute compartment syndrome
- Rhabdomyolysis
- Acute renal failure
- Hepatic dysfunction
- DIC.

COMPARTMENT SYNDROME

Richard von Volkmann in 1872 was first to describe compartment syndrome. He proposed that "The paralysis is caused by too long continued isolation of the arterial blood".[13]

Compartment syndrome may occur in the abdomen, chest and face but the majority of cases are diagnosed within the extremities. The majority of cases of compartment syndrome (roughly 45%) are due to tibial fractures. These fractures generally involve high levels of energy with many being open fractures.[3]

Compartment syndrome develops when increased tissue pressure in a myofascial compartment increases to a point that blood flow to the muscles and nerves is impaired. The resultant ischemia causes tissue and nerve damage leading to cellular death. Symptoms worsen acutely, and if the condition is not quickly reversed, individuals develop irreversible damage to nerves and muscles leading to permanent deficits.

Treatment of compartment syndrome is emergency fasciotomy. Ideally it should be done before appearance of painlessness or paralysis in extremity.[4]

MEASUREMENT OF INTRACOMPARTMENTAL PRESSURES

Mainly Stryker STIC Device (Stryker Corporation, Kalamazoo, Michigan) is used to measure the intra compartmental pressures.[3]

The normal pressures within a compartment range from 0 to 4 mm Hg when muscle is at rest but during exertion it can rise up to 8 to 10 mm Hg. In normotensive patients cut off point for emergency fasciotomy is taken generally as 30 mm Hg. In hypotensive or hypertensive patients comparison with diastolic pressures is more justified. Whitesides postulated that a patient could be hypotensive and have a value less than 30 mm Hg but still have an elevated compartment pressure being within 20 mm Hg from the diastolic number.[14]

Some authorities consider that field fasciotomy can increase chances of infection, bleeding and sepsis. It converts a closed injury to an open one, risking infection and sepsis. Several studies indicate a worse outcome in patients who received fasciotomy compared with those who did not.

Hence it is advocated that fasciotomies should be done only in patients not responding to conservative and medical line of treatment.[2,8,15]

RHABDOMYOLYSIS

Crush syndrome is a form of traumatic rhabdomyolysis that occurs after prolonged continuous pressure and characterized by systemic involvement.[16] Rhabdomyolysis is the breakdown of muscle fibers with leakage of potentially toxic cellular contents, e.g. in Table 31.1 into the systemic circulation.

PATHOPHYSIOLOGY

In rhabdomyolysis, there is extensive muscle breakdown and release of toxins in systemic circulation mainly myoglobin. Two crucial factors for development of myoglobinuric renal failure are hypovolemia and aciduria. Renal vasoconstriction with diminished renal circulation, intraluminal cast formation and direct heme protein induced cytotoxicity are main mechanisms behind heme protein induced renal toxicity.[17]

It has been suggested that ARF is caused by tubular obstruction causing increased intraluminal pressures and thus opposing glomerular filtration. Other mechanism suggested is heme protein precipitated in kidneys itself providing substrate for generating toxic free radicals. The propensity for cast formation is determined by the pH, the filtered load of myoglobin and the flow through the renal tubules.[18-20]

Heme causes free radical induced oxidative damage to the renal tubule. It has been suggested that myoglobin is central to the oxidative injury manifested as lipid peroxidation, and that this may be inhibited by an alkaline pH. Other reasons implicated in acute renal failure are, renal vasoconstriction secondary to circulatory shock and pigment nephrotoxicity.[8]

CLINICAL FEATURES

- General: Malaise, fever, tachycardia, nausea and vomitings
- Musculoskeletal: Pain, tenderness, paraesthesia, weakness
- Complications: Dark urine, oliguria, anuria, hepatic dysfunction, disseminated intravascular coagulation

Hepatic dysfunction occurs in 25 percent of patients with rhabdomyolysis. Proteases released from injured muscle lead to hepatic injury. ARF and diffuse intravascular coagulation are late complications, developing 12 to 72 hours after the acute insult.[21]

INVESTIGATIONS

- Serum myoglobin levels
- Serial serum creatinine kinase (CK) levels
- Blood urea nitrogen
- Serum K^+ levels
- Blood coagulation profile
- Urinalysis to determine myoglobin and CK

MANAGEMENT

Early diagnosis and treatment can prevent complications due to crush injury and rhabdomyolysis. Fluid replacement should start at the site of extrication of the trapped victim. Initial fluid should be preferably isotonic saline at the rate of 1.5 L/hr. It has been recommended as a prophylactic treatment.[15]

In our institute, immediately after arrival of victim at hospital, we send all routine blood investigations to laboratory along with serum electrolytes. Hyperkalemia can develop within hours of crush injury and renal failure may develop. Patients often die of hyperkalemia unless they are treated rapidly. Other electrolyte imbalances which can be encountered are hypocalcemia and hyperphosphatemia.

Arterial blood gases, blood and urine pH should be measured. Empirical 1 mEq/kg sodium bicarbonate can be given to decrease pre-existing acidosis; later alkaline diuresis can be instituted to avoid myoglobinuric renal failure. The patient with crush injury syndrome should maintain a urine output of at least 300 ml/h with a pH higher than 6.5.[8]

Mannitol can be given in doses of 1 gm/kg or added to the patient's intravenous fluid as a continuous infusion. The maximum dose is 200 gm/day with continuous monitoring of urine output, urine pH, ABG, and serum electrolytes.

Following algorithm can be used for a suspected rhabdomyolysis patient[22,23] (Flow chart 31.1).

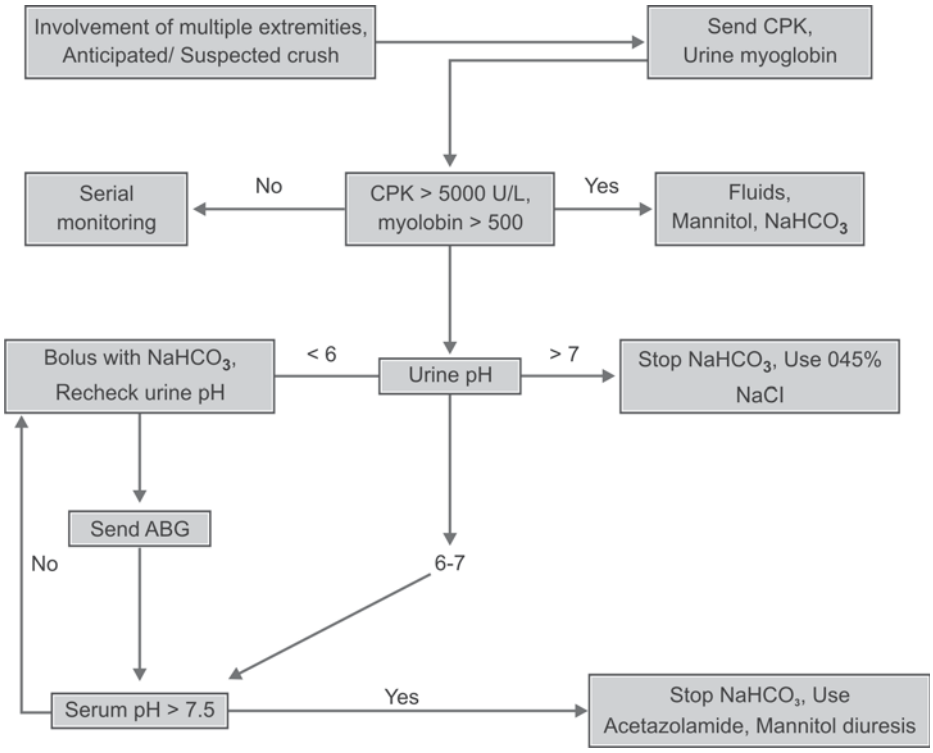
ANESTHESIA IN CRUSH INJURY PATIENTS

Emergency fasciotomy, debridement, refashioning and amputation of involved extremity are some of the common surgical procedures required in crush injury patients. Muscle ischemia from acute crush injury can cause muscle necrosis within the 3-hour post-trauma period that was previously considered safe and irreversible tissue damage ensues after 8 hours of injury.[5] Hence it is very important to give emergency medical help at the earliest.

If compartment pressures are elevated, fasciotomies should be performed. At the time of fasciotomy, extensive resection of all dead muscle should be performed at the first operation. Dead muscle cannot be identified by lack of bleeding. Identification of dead muscle is by its reaction to direct physical or electrical stimulation.

During preoperative assessment, along with general examination, volume status should be assessed. All patients with crush injury are required to be resuscitated and optimized well, before any surgical procedure. All patients are to be investigated with hemogram to estimate blood loss, serum electrolytes and renal functions. Hyperkalemia if present may lead to arrhythmias, which should be diagnosed and treated. Adequate cross matched blood and blood products are to be kept ready in blood bank. Additional

Flow chart 31.1 Treatment of rhabdomyolysis[22,23]



investigations may be needed as per patients' medical status. Associated injuries should be noted.

Regional anesthesia can be used for fasciotomy but patient may not cooperate because of associated injuries or deranged mental status. It may also be difficult to execute a regional block because of edematous extremities. Regional or Neuraxial blockade can be used for amputation cases where patient is hemodynamically stable and has no associated major injuries. Different peripheral nerve blocks like sciatico-femoral, popliteal, brachial plexus block can be used for such procedures. Neuraxial blockade is not advocated in hemodynamically unstable patients for both amputation and fasciotomy. One should avoid using adjuvants and intense blockade for fasciotomies as it is a short procedure, and it may obscure signs of inadequate fasciotomy for a long time.

Hemodynamically unstable patient and associated major injuries will mandate general anesthesia with or without endotracheal intubation or laryngeal mask airway. Thiopentone sodium, propofol, ketamine can be used for induction of anesthesia before endotracheal intubation. These agents should be given in smaller titrated doses, in hypovolemic patients. One should avoid use of succinylcholine in extensive crush injury provided there is no anticipated airway difficulty as it can exacerbate existing hyperkalemia to dangerous levels. A short acting agent like atracurium or mivacurium may be used. Atracurium, cisatracurium, mivacurium are also beneficial in patients in ARF as these are least excreted by renal route. Maintenance of anesthesia is commonly done with inhalational agents or propofol infusion with opioids. It should be remembered that fasciotomy is a short procedure and hence unnecessary long acting anesthetic agents should be avoided.

Intraoperative monitoring includes continuous electrocardiogram, pulse oximetry, capnography, blood pressure and urine output. Central venous line helps in assessing adequacy of fluid status and also offers route for fluid and drugs administration, e.g. mannitol. Serum electrolytes and arterial blood gases estimations should be done during the procedure, if needed.

Intraoperatively, normovolemia and normal blood pressure should be maintained to optimize perfusion to ischemic tissues. Hypotension should be avoided. Hypertension, on the other hand, is associated with increased bleeding from muscles being excised. Most often, surgeries with crush injuries do not involve use of intraoperative tourniquets. When a patient arrives in the theater with a limb tourniquet, sudden blood loss and hypotension should be anticipated when they are removed at the start of surgical procedure.

To avoid myoglobinuric renal failure intraoperatively, it is important to maintain central venous pressures, maintain alkaline pH of urine by producing alkaline diuresis, use of diuretics and mannitol during surgery. It is very important to monitor hemoglobin, serum electrolytes, serum myoglobin, serum CPK (if found raised initially) and blood coagulation during intraoperative period. Some authors suggest that serum myoglobin rather than CK levels should be used to guide therapy in such patients.[23]

Thus early resuscitation, hemodynamic stability, adequate diuresis and prompt treatment of complications will help in better management of crush injuries.

Prevention and treatment of ARF: Adequate intravascular volume expansion is the cornerstone of treatment of rhabdomyolysis. Exact amount of preloading is not known but it should be adequate to ensure good urine output. Alkalinization of as described above will prevent formation of pigmented casts in kidneys and thus prevent renal failure.

Diuretics can be used to maintain adequate urine flow as well mannitol can be used as described above. While using diuretics like mannitol patient should be adequately hydrated as these agents themselves can cause renal dysfunction otherwise. Early renal replacement therapy use in case of suspected acute renal failure had good results. Hemodialysis and continuous or intermittent peritoneal dialysis are modes available for renal replacement therapy.

SUMMARY

Thus management of crush injury patient is a multidisciplinary work and requires good interdepartmental coordination for successful outcome. More sophisticated biochemical studies and devises to measure intercompartmental pressures are required to avoid unnecessary fasciotomies.

REFERENCES

1. Verma PK, Tiwari KN. Epidemiology of Road Traffic Injuries in Delhi: Result of Survey. Regional Health Forum-Volume 8, Number 1, 2004.
2. Gail T. Tominaga. Crush Syndrome. In: Demetrios Demetriades, Juan A. Asensio, (Eds). Trauma Management, Texas; Landes Bioscience 2000;61;618-22.
3. John FD. Compartment Syndrome of the Lower Extremity: Blast Injuries in a Ranger Battalion Don't Miss The Diagnosis. Journal of special operations medicine; ISSN 1553-9768;2006; Vol 6, Edition 2.
4. Darren J. Malinoski, Matthew S. Slater, Richard J. Mullins. Crush Injury and Rhabdomyolysis. Crit Care Clin 2004;20: 171-92.
5. Christian Vaillancourt, Ian Shrier, Alain Vandal, Markus Falk, Michel Rossignol, Alan Vernec, Dan Somogyi. Acute compartment syndrome: How long before muscle necrosis occurs? CJEM 2004;6(3):147-54.
6. Wallace S, Goodman S. Compartment Syndrome, Lower Extremity. Emedicine 2005. Updated on Feb 9, 2009 http://www.emedicine.com/orthoped/topic596.htm
7. Samuel JS, Mark Eckstein. Prehospital Trauma Care; In: Demetrios Demetriades, Juan A. Asensio, (Eds). Trauma Management, Texas; Landes Bioscience 2000;1: 2-15.
8. Jason Smith, Tan Greaves. Crush Injury and Crush Syndrome: A Review; The journal of trauma 2003;54: S226-30.
9. Gary GS, Barry MB. Fluid and Electrolyte Disturbances. In: Anthony S Fauci, Eugene Braunwald, Dennis L Kasper, Stephen L Hauser, Dan L Longo, J. Larry Jameson, Joseph Loscalzo (Eds). Harrison's principle of internal medicine, 17th edition. New York: McGrawHill Medical 46:274-85.
10. Edwin K, Jackson Diuetics. In: Goodman LA, Gilman A, Hardman JG, Limbard LE (Eds). The Pharmacological Basis of Therapeutics, 10th edn. New York; The McGraw Hill 2006;29:757-87.
11. Godbout B, Burchard KW, Slotman GJ, Gann DS. Crush syndrome with death following pneumatic anti-shock garment application. J Trauma 1984;24:1052-6.
12. James PB. Hyperbaric oxygen treatment for crush injury. BMJ 1994;309(6967):1513.
13. Hatch W, Hatch V. Richard von Volkmann and surgical school of Halle from 1867 to 1889. Zentralbl Chir 2001;126: 822-7.
14. Whitesides TE, Heckman MM. Acute Compartment Syndrome: Update on Diagnosis and Treatment; J Am Acad Orthop Sueg 1996;4:209-18.
15. Demirkiran, Y Dikmen, T Utku, S Urkmez. Crush syndrome patients after the Marmara earthquake. Emerg Med J 2003;20:247-50.

16. Hiraide A, Ohnishi M, Tanaka H, et al. Abdominal and lower extremity crush syndrome. Injury 1997;28:685-6.
17. Ana L Huerta-Alardín, Joseph Varon, Paul E Marik. Bedside review: Rhabdomyolysis—an overview for clinicians. Critical Care 2005;9:158-69.
18. Flamenbaum W, Gehr M, Gross M, Kaufman J, Hamburger R. Acute renal failure associated with myoglobinuria and hemoglobinuria. In: Brenner B, Lazarus J (Eds). Acute Renal Failure. Philadelphia: WB Saunders 1983;269-82.
19. Salahudeen A, Wang C, Bigler S, Dai Z, Tachikawa H. Synergistic renal protection by combining alkaline-diuresis with lipid peroxidation inhibitors in rhabdomyolysis: possible interaction between oxidant and non-oxidant mechanisms. Nephrol Dial Transplant 1996; 11:635-42.
20. Zager R, Burkhart K. Differential effects of glutathione and cysteine on Fe^{2+}, Fe^{3+}, H_2O_2 and myoglobin-induced proximal tubule cell attack. Kidney Int 1998;53:1661-7.
21. Akmal M, Massry S. Reversible hepatic dysfunction associated with rhabdomyolysis. Am J Nephrol 1990;10: 49-52.
22. Brown, Carlos VR, Rhee, Peter , Chan, Linda, Evans, Kelly, Demetriades, Demetrios, Velmahos, George C. Preventing Renal Failure in Patients with Rhabdomyolysis: Do Bicarbonate and Mannitol Make a Difference? The Journal of Trauma: Injury, Infection, and Critical Care 2004; 56:(6)1191-6.
23. Helena Lappalainen, Eero Tiula, Lasse Uotila, Matti Manttari. Elimination kinetics of myoglobin and creatine kinase in rhabdomyolysis: Implications for follow-up. Crit Care Med 2002;30:2212-5.

32 Anesthetic Considerations in Blast and Burn Injuries

Jyoti S Magar, Deepali P Thakur

KEY POINTS

- Bombs and explosions can cause unique patterns of injury seldom seen outside combat. These can have the potential to inflict multi-system life-threatening injuries on many victims simultaneously.
- Four basic mechanisms of blast injury are termed as primary, secondary, tertiary and quaternary. A primary blast injury almost always affects air-filled structures such as the lung, ear, and gastrointestinal (GI) tract.
- Expect half of all initial casualties, to seek medical care over one-hour period.
- Explosions in confined spaces (buildings, large vehicles, mines) and/or structural collapse are associated with greater morbidity and mortality.
- Anesthesiologist's response includes: A—Assess incident size and severity; B—Alert backup; C—Casualty care; D—Definitive care.
- All bomb events have the potential for chemical and/or radiological contamination.
- Blast lung injury signs are usually present at the time of initial evaluation, but may be delayed up to 48 hours. Suspect in anyone with dyspnea, cough, hemoptysis, or chest pain following blast with "butterfly" pattern on chest X-ray.
- Administer high flow O_2 sufficient to prevent hypoxemia. Ensure tissue perfusion but avoid volume overload. Consider tracheal intubation for massive hemoptysis, impending airway compromise or respiratory failure.
- Consider selective bronchial intubation for significant air leaks or massive hemoptysis. Positive pressure may risk alveolar rupture or air embolism.
- Prompt decompression of pneumothorax or hemothorax is needed. Consider prophylactic chest tube before general anesthesia or air transport.
- Air embolism can present as stroke, myocardial infarction, acute abdomen, blindness, deafness, spinal cord injury. High flow O_2; prone, semi-left lateral, or left lateral position should be given. Consider transfer for hyperbaric O_2 therapy.
- History may be asked by written question in presence of acoustic trauma.
- They often have limited choice for venous access. Invasive monitoring is reserved for patients with blast lung, major burns and unresponsive shock requiring inotropic support.
- Succinylcholine is to be avoided in patients with burns, crush injury, compartment syndrome due to risk of life-threatening hyperkalemia. Rapid sequence induction with modified Sellick's maneuver is preferred. Rocuronium may be used if succinylcholine is contraindicated.
- Avoid the deadly triad of hypothermia, coagulopathy and metabolic acidosis, as each condition worsens both of the others.
- Prophylactic postoperative ventilatory support may be required in patients with hemodynamic instability, moderate to severe blast lung, multiple rib fractures with flail segment, massive blood transfusion, and impaired coagulation.
- In burns patient evaluation for any emergency surgery, anesthetist should avoid tunnel vision as burns injury causes multisystem involvement.
- Assessment should include age of patient, etiology, depth and extent of burn, associated inhalational injury and other injuries, adequacy of resuscitation and patients response to resuscitation.
- In presence of airway edema, intubation difficulty is anticipated and appropriate difficult airway gadgets are to be kept ready.

- Initial resuscitation should include ABCDE of trauma resuscitation. Fluid deficit is calculated as per one of the various formulae available.
- Practical difficulties in conduction of anesthesia are difficult AIM, i.e. difficult airway, difficult intravenous access and difficult monitoring.
- Anesthesia principles to be followed are adequate analgesia preferably opioids, avoid hypothermia, avoid succinylcholine after 24 hours of burns, replace blood loss with colloids and blood products and delay extubation in case of airway edema or massive blood transfusion.

Bomb blast injuries to civilians in a non-combat setting have become increasingly common over the last decade, mainly as acts of terrorism. Well-known examples of such acts of terrorism include the Madrid commuter train bombings (March, 2004), the London Underground bombings (July, 2005), the serial train blasts in Mumbai (2006) and the recent terrorist attack in Mumbai (November, 2008). With the ever-present threat of terrorist acts, it is important for anesthesiologist to be well-versed in the spectrum of injuries that are associated with mass casualty incidents resulting from bomb blasts and other similar explosive devices and their efficient management. When they do occur, they have the potential to inflict multi-system life-threatening injuries on many persons simultaneously. The injury patterns following such events are a product of the composition and amount of the materials involved, the surrounding environment, delivery method (if a bomb), the distance between the victim and the blast, and any intervening protective barriers or environmental hazards.[1] Because explosions are relatively infrequent, blast-related injuries can present unique triage, diagnostic and management challenges to providers of emergency care and anesthesiologist. Careful observation for signs and symptoms of exposure to poisonous chemicals, screening for radiation contamination and decontamination of patients as needed are important steps in the management of victims of explosions. Blast injury is now specifically addressed in Advanced Trauma Life Support 8th Edition Guidelines (ATLS) because of the recent military experience with improvised explosive devices.[2]

ETIOLOGY

Classification of Explosive

Explosives can be classified into two broad categories, called "high order explosives" or "low order explosives". High order explosives (HE) produce a defining supersonic over-pressurization shock wave. Detonation rates are usually between 1000 to 10000 yards/second. Examples are TNT (trinitrotoluene), PETN (pentaerythritol tetra nitrate), RDX (cyclonite), plastic-bonded explosives such as C-4 or Semtex, dynamite, nitroglycerine and ammonium nitrate/fuel oil (ANFO) mixtures.[3]

Low order explosives (LE) create a subsonic explosion and undergo deflagration (very rapid burning). Burn rates may be on the order of inches to yards per second. The characteristic over pressurization shock wave is not produced. Examples are gunpowder, pipe bombs and ignition of gasoline vapors, Molotov cocktails and aircraft used as guided missiles.[3]

Explosion Physics

An explosion is defined as the release of mechanical, chemical or nuclear energy in a sudden and often violent manner with generation of high temperatures and winds.[1] Detonation of an explosive material results in instantaneous conversion of a solid or liquid into gas.[4] Rapid expansion of this gas in outward direction causes displacement of the surrounding medium, either air or water. This expansion of gas causes an immediate rise in pressure creating a blast wave. The maximum pressure is called the peak or blast overpressure.[1] An overpressure of 60 to 80 PSI is considered potentially lethal. After the initial energy of the explosion has dissipated, a phase of negative underpressure develops due to the vacuum created by the displaced air, before normalization of pressure.[4]

Factors Affecting Blast Overpressure

- *Medium:* Water being noncompressible has a greater potential for injury than air.
- *Distance:* The person closer to an explosion experiences greater blast overpressure.
- *Reflecting surface:* Pressure waves reflect back from solid surfaces and amplify the effective blast over pressure. A person close to a wall is subjected to increased blast overpressure thus he is at raised risk of blast injury.[4]

- *Site of explosion:* Open or enclosed space. In open space circumferential spread of blast wave from its origin leads to quick dissipation of energy. An explosion that occurs in an enclosed space including a building, a mine or a relatively lightly constructed enclosed space such as a bus tends to cause more serious injury.[5]

MECHANISM OF INJURY IN BLAST

Explosion causes multidimensional injuries in multiple victims at the same time. For theoretical understanding, four basic mechanisms of blast injury are termed as primary, secondary, tertiary, quaternary and quinary[3,4,6] (Table 32.1).

- A primary blast injury is caused solely by the direct effect of blast overpressure on tissue. Air is easily compressible, unlike water. As a result, a primary blast injury almost always affects air-filled structures such as the lung, ear and gastrointestinal (GI) tract.[3]
- A secondary blast injury is caused by flying objects that strike people.
- A tertiary blast injury is a feature of high-energy explosions. This type of injury occurs when people fly through the air and strike other objects.
- Quaternary blast injuries encompass all other injuries caused by explosions, such as burns, crush injuries and toxic inhalations. It also includes secondary consequences of trauma leading to exacerbation of pre-existing condition. For example, exacerbation of angina, hypertension, asthma, COPD, hyperglycemia.[3]
- Quinary pattern of blast injury is recently suggested in the form of hyperinflammatory response following blast exposure.[6]

Table 32.2 summarizes blast inflicted injuries on multiple systems of the body.

Primary Blast Injury

- Primary blast injury (PBI) is organ and tissue damage caused solely by the blast wave associated with HEs.
  - The leading edge of a blast wave is called the blast front. When a blast front reaches a victim, it causes an enormous, almost instantaneous rise in ambient pressure.[4]
  - Both the positive overpressure and the negative under pressure are capable of causing significant PBI.[4]
  - Blast overpressure is transmitted into three explosive forces: spallation, implosion and inertia.[4,5]
- Since air is easily compressible by pressure while water is not, organ systems with air-tissue interfaces, especially the pulmonary, gastrointestinal and auditory systems are most susceptible to PBI.
- Auditory injury occurs at lowest blast overpressure (35 kPa), whereas pulmonary and GI injury occurs at increased pressures (75-100 kPa).[5]
- Global shock-like response with transient bradycardia, bradypnea and systemic hypotension uncompensated by vasoconstriction can follow a thoracic blast overpressure. This response is mediated by pulmonary C fibers in the vagus nerve.[7]

Table 32.1: Mechanism of blast injury

Category	*Characteristics*	*Organs affected*	*Type of injury*
Primary	Unique to HE, results from the impact of the over-pressurization wave with body surfaces.	Gas filled structures are most susceptible–lungs, GI tract, and middle ear	- Blast lung - TM rupture and middle ear damage - Abdominal hemorrhage, perforation - Globe (eye) rupture - Concussion without physical signs of head injury
Secondary	Results from flying debris and bomb fragments	Any body part may be affected	- Penetrating ballistic or blunt injuries - Eye penetration
Tertiary	Results from individuals being thrown by the blast wind	Any body part may be affected	- Fracture and traumatic amputation - Closed and open brain injury
Quaternary	- All explosion-related injuries, illnesses, or diseases not due to primary, secondary, or tertiary mechanisms. - Includes exacerbation or complications of existing conditions.	Any body part may be affected Systemic responses from massive trauma	- Burns, crush injuries - Closed and open brain injury - Radiation exposure - Asthma, COPD, or other breathing problems from dust, smoke, or toxic fumes - Angina, hyperglycemia, hypertension.
Quinary	Hyperinflammatory response	Systemic responses from massive trauma	- Hyperpyrexia, Diaphoresis, low central venous pressure, positive fluid balance

Table 32.2: Explosive related injuries[3]

System	Injury
CNS injury	Concussion, head injury, stroke, spinal cord injury, air embolism-induced injury
Auditory	TM rupture, ossicular disruption, cochlear damage, foreign body
Eye, Orbit, Face	Perforated globe, foreign body, air embolism, fractures
Respiratory	Blast lung, hemothorax, pneumothorax, pulmonary contusion and hemorrhage, aspiration pneumonitis, sepsis
Circulatory	Cardiac contusion, myocardial infarction from air embolism, shock, vasovagal hypotension, vascular injury, air embolism-induced injury, penetrating injuries
Digestive	Bowel perforation, hemorrhage, ruptured liver or spleen, sepsis, mesenteric ischemia from air embolism
Renal Injury	Renal contusion, laceration, acute renal failure due to rhabdomyolysis, hypotension, and hypovolemia
Extremity injury	Traumatic amputation, fractures, crush injuries, compartment syndrome, burns, acute arterial occlusion, air embolism-induced injury

Pulmonary System

"Blast lung" is a direct consequence of the HE overpressurization wave which can give rise to pulmonary hemorrhage and contusions, direct barotraumas, arterial air embolism, free radical damage such as thrombosis, DIC.[3] ARDS may be due to direct lung injury or shock from other body injuries. Blast lung is the most common fatal primary blast injury among initial survivors. The incidence of blast lung increases three-fold with closed-space explosions and high blast overpressure generation.

Presentation: Immediate or delayed (12-24 hours) due to progressive vascular leak and inflammatory changes in injured lung.[8]

Pulmonary contusion: Spallation and implosion forces cause disruption of the alveoli, capillary walls and lung parenchyma leading to perivascular pooling of blood, alveolar hemorrhage and pulmonary edema.[9] Blast lung syndrome refers to the clinical picture of dyspnea, cough and hypoxia due to impaired gas exchange and ventilation perfusion mismatch.

Pulmonary barotraumas: Pleural tears or lacerations can lead to pneumothorax, hemothorax, pneumomediastinum or subcutaneous emphysema. Pulmonary barotraumas may be exacerbated by high altitude air transport.[3,9]

Signs and Symptoms

- Apnea, tachypnea, hypopnea, dyspnea.
- Cough, hemoptysis, chest pain
- Hypoxia, cyanosis, wheezing
- Decreased air entry, dullness on percussion

Arterial Air Embolism

Disruption of bronchovascular tree due to shearing forces leads to formation of bronchopulmonary fistulas. Arterial air emboli can develop immediately after blast or after starting positive pressure ventilation, PEEP.[1,9]

Signs and Symptoms

- Confusion, mental status changes
- Visual disturbances, air in retinal arteries
- Tongue blanching
- Livedo reticularis (mottled skin discoloration).

Signs and symptoms of massive emboli may be due to spinal cord ischemia, myocardial infarction, stroke, intestinal ischemia and may lead to death. Resulting neurologic symptoms must be differentiated from the direct effect of trauma.

Diagnosis and Treatment of Blast Lung Injury

Imaging: A chest X-ray is recommended for all exposed persons with pulmonary complaints, evidence of other primary blast injuries or suspected exposure to a high blast overpressure. Blast lung produces a characteristic "butterfly" pattern on chest X-ray due to bilateral pulmonary infiltrates.[3] But X-ray may be normal initially; hence patients with persistent pulmonary signs and symptoms with normal X-ray chest, CT chest should be done.

Pulse oximetry may be used for screening mass casualty patients for desaturation.

Arterial blood gas analysis may show hypoxemia and hypercarbia with respiratory acidosis.

Management

Management of pulmonary blast injury is a challenge as therapies for different injuries often conflict.[5]

- Hemodynamic instability demands volume resuscitation but excessive crystalloids can cause pulmonary edema in patients with pulmonary contusions.[1,10]
- Prefer using non-invasive ventilation techniques like continuous positive airway pressure, bilevel positive airway pressure via facial or nasal masks and adequate analgesia for maintaining adequate respiration.
- Prompt chest drainage of pneumo or hemothorax.

- Prophylactic intercostal drainage tube should be considered in severe blast lung injury before positive pressure ventilation and air transport.[1,3]
- Pizov el al have classified BLI into mild (PaO_2/FiO_2 >200, localized lung infiltrates on chest radiograph, no evidence of bronchopleural fistula), moderate (PaO_2/FiO_2 60-200, bilateral and asymmetric chest infiltrates), and severe (PaO_2/FiO_2 <60, severe bilateral chest infiltrates, and bronchopleural fistula present) (Table 32.3).[11] Ventilatory support is likely to be necessary in moderate-to-severe BLI because of lung or coexisting injury.
- Positive pressure ventilation (PPV) and positive end expiratory pressures (PEEP) should be avoided whenever possible in the setting of pulmonary blast injury due to the risk of pulmonary alveolar rupture and subsequent formation of air emboli.[9,10] However, mechanical ventilation often cannot be avoided. Due to the nonhomogeneous pulmonary compliance that characterizes the blast lung, localized over inflation of the more compliant lung segments occurs when high ventilatory pressures are used. Whenever possible, reduce the tidal volume to limit peak inspiratory pressure (PIP) and minimize ventilator-induced lung barotrauma injury. If necessary, consider permissive hypercapnia ventilation: reduce the tidal volume to maintain PIP less than 40 cm H_2O; make no attempts to control $PaCO_2$ levels until the arterial pH falls below 7.25. When respiratory acidosis becomes too severe, increase the respiratory rate until the arterial pH rises above 7.25.[12]
- Patients thought to have arterial gas embolism (AGE) require recompression treatment. Place patients on 100% oxygen by tight-fitting face mask and, if possible, place them in the left lateral recumbent position with head low to minimize the risk of travel of the air embolism out of the heart. If the side of the lung responsible for the AGE can be identified, unilateral lung ventilation may prevent further introduction of air into the vascular system during positive pressure ventilation.[1,9]
- In case of acute mental status changes, cerebral AGE should be considered as well as other causes of symptoms (e.g. traumatic CNS injury).
- Hyperbaric oxygen (HBO) treatment is the definitive procedure for AGE and cerebral AGE. Transfer of the patient to a facility with HBO therapy may be required.[1]

Gastrointestinal System

Abdominal injury is more common with underwater blast and closed space explosions. Colon and ileocecal junction are at greatest risk of perforation. Due to the implosion and shearing forces, intestinal wall contusion, edema, hemorrhage leads to impaired intestinal perfusion.[3] Arterial air embolism can further lead to intestinal ischemia. Abdominal solid organ injury can be due to primary, secondary or tertiary mechanism. Primary blast injury can lead to immediate or delayed bowel perforation, mesenteric shear injuries, solid organ laceration, hemorrhage and testicular rupture.[5,9]

Signs and symptoms: Nausea, vomiting, pain in abdomen, hematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia, acute abdomen.

Diagnosis and Treatment

- If significant abdominal pain is present, consider an immediate abdominal radiographic series (flat and upright films) or abdominal CT to detect pneumoperitoneum from enteric rupture.
- The Focused Abdominal Sonography for Trauma (FAST) examination is a potentially useful tool for rapidly screening patients, especially in the setting of multiple seriously injured victims.
- A positive FAST examination in an unstable patient is an indication for surgical exploration of the abdomen in the operating room (OR). Volume resuscitation is to be continued until emergency surgery can be done. In patients with associated lung injury, permissive hypotension (systolic BP: 80-90 mm Hg) during resuscitation may be beneficial. But

Table 32.3: Severity of blast lung injury[11]

	Mild BLI	*Moderate BLI*	*Severe BLI*
Bronchopleural fistula	–	+/–	+
Chest radiograph	Localized lung infiltrates	Bilateral asymmetric lung infiltrates	Severe diffuse bilateral infiltrates
PaO_2/FIO_2	>200	60–200	< 60
Positive pressure ventilation	+/–	+	+
Positive end expiratory pressure (cm,H_2O)	≤ 5	5-10	> 10

in patients with concomitant head injury blood pressure should be normalized to avoid cerebral hypoperfusion.[5,9]
- In stable patients, a positive FAST examination can facilitate prioritization for CT imaging. A negative FAST examination is unreliable in the setting of penetrating trauma to the abdomen; flank, buttocks or back, and it should be followed up with CT examination of the abdomen and pelvis.
- No practical, sensitive test exists for intestinal hematoma. The diagnosis is often missed even when performing the best available test—abdominal CT. Because intestinal hematoma can take 12 to 36 hours to develop, symptoms such as increased pain or vomiting should determine decisions about testing.

Auditory System

The ear is the organ most susceptible to primary blast injury. Acoustic barotrauma commonly consists of tympanic membrane (TM) rupture. Hemotympanum without perforation also has been reported. Ossicle fracture or dislocation may occur with very high-energy explosions.[3,9]

Patient communication may be difficult while obtaining history and consent due to hearing problem. History taking may be performed in writing.

Signs and symptoms: Hearing loss, tinnitus, otalgia, vertigo, external ear bleed, and otorrhea.

All patients exposed to blast must undergo otoscopic and ocular examination. X-ray chest should be taken for patients with TM perforation to rule out pulmonary injuries.

Central Nervous System

Primary blast wave can cause cerebral concussion and lead to post-traumatic stress disorder.[5,9]

Signs and symptoms: Headache, memory impairment, poor concentration, lethargy, fatigue.

Secondary Blast Injury

Flying objects (shrapnel) mainly cause penetrating ballistic or blunt injuries to many victims.[3] Hand grenades are specifically designed to fragment and increase damage from shrapnel. Terrorist bombers deliberately use screws and metal objects to increase secondary injuries. Penetrating injuries can affect any part of body. Penetrating cardiac and vascular injuries can be fatal and need urgent intervention with simultaneous resuscitation.

Tertiary Blast Injury

These injuries are due to individuals being thrown away by blast wave.[3] Most commonly seen with high energy explosions, focused energy (through door or hatch) and in persons close to explosion. Affected individuals may sustain skull fractures, blunt abdominal trauma, long bone fractures, traumatic amputations and head injuries. Pediatric age group may be more affected by this mechanism.

Quaternary Blast Injury

Injuries other than above three mechanisms are termed as quaternary blast injuries. They include burns (thermal, chemical), crush injuries, toxic substance exposure (radiation, carbon monoxide poisoning, cyanide poisoning), asphyxia, injuries due to falling structures, etc. It also includes acute exacerbation of pre-existing conditions like hypertension, angina, asthma, COPD, diabetes. They may further complicate perioperative management of blast victims undergoing surgeries and those in ICU.

Quinary Blast Injury

There is a hyperinflammatory state after exposure to blast. Patients may have hyperpyrexia, diaphoresis, low central venous pressure and a positive fluid balance.[6]

On Site Management

- Airway with C-Spine control is most effective in saving most of salvageable patients with unconsciousness with inadequate airway or ventilation.
- Improve oxygenation by O_2 via mask
- Urgent decompression of pneumothorax either by insertion of large needle in 2nd intercostal space anterior axillary line with under water seal or chest tube in 5th intercostal space.[13]
- Control of hemorrhage by direct pressure or tourniquet.
- IV fluids to be started in hemodynamically unstable patients.
- Alignment of fracture using available splints (Wood, cardboards).

Management at Emergency Department

Following blast event there are multiple patients with multiple injuries hence activation of hospital and regional disaster management plan is essential.

Expect "upside-down" triage – the most severely injured arrive after the less injured, who bypass EMS triage and go to local hospitals for treatment.[3]

- Double the first hour's casualties for a rough prediction of the total expected number of acute casualties.[3]
- Obtain information on nature of explosive, potential toxic exposures and environmental hazards.
- Screen patients as needed to rule out radiation (dirty bomb).

Four phases have been described:[13]

1. *Chaotic phase:* Lasts for 15 to 25 minutes due to lack of leader. A chain of command (Institutional and intra-departmental) is essential to control this phase. Upside down triage as less injured arrive first.
2. *Reorganization phase* (1–1 ½ hours)*:* Most important phase as EMS commander should indentify patients with immediate life threatening injuries needing urgent explorations in operation theater. Senior most surgeon and anesthetist must be available to do this job. They decide about disposition of these patients either to OR, radiology, ICU, recovery room.
3. *Site clearing phase:* (2–3 hours) Patients of prolonged extrication may arrive in this phase. Occult injuries may be scrutinized again for delayed development of symptoms.
4. *Late phase:* After site clearing to 24 to 48 hours risk of another bomb (serial bombs) may be present hence EMS must be prepared during this phase by resupply of medical equipments used.

Anesthesiologist should follow response ABCD[14]

A—Assess incident size severity
B—Alert backup.
C—Casualty care.
D—Definitive care.

- Effective triage at ambulance bay by senior surgeon helps indentifying urgent and nonurgent patients.
- Optimal care for every patient with preference to salvageable patient.
- Conservation of critical hospital resources until exact number of mass casualties is known.

Urgent patients: Screening of patients for injuries is done and treatment according to advance trauma life support is started.

A. Hemodynamically unstable patients are transferred to operating room for emergency explorations (liver, spleen, kidney lacerations).
B. Stable patients are transferred to radiology suite for X-ray, CT, FAST, angiography, MRI with conservative management (IV fluid, antibiotics, analgesia nasogastric tube).

Nonurgent patients: After evaluation may be admitted to wards or discharged. All patients before discharge must be evaluated for phonal trauma (TM rupture) and penetrating ocular injuries.

Sequence of Surgeries Performed

According to priority:

- Emergency life saving explorations for solid organ damage or major vascular damage
- Craniotomies for close and open brain injuries
- Explorations for bowel perforation.
- Extremity injuries with bone, vessel injuries—amputations, time consuming vascular and nerve repairs are inappropriate in this situation.
- Debridement of contaminated wounds.

Laboratory Investigations

- All patients—Urinalysis
- Closed space explosion and fire:
  ABG - For carboxy hemoglobin estimation for carbon monoxide (CO) toxicity
  - Acid base status.
  - Anion gap metabolic acidosis seen in cyanide toxicity
  - Serum electrolytes (K^+, Na^+, Ca^{++}, PO_4^-)
- Patients with major trauma should have:
  Hb, CBC, platelet count
  Blood grouping cross-matching
  DIC screening—PT, INR, aPTT, serial CBC for platelet count, fibrinogen, fibrin split products, D-Dimer.
- Patients with crush injury, compartment syndrome or severe burns:
  BUN, Serum creatinine
  Serum K^+ to rule out hyperkalemia
  Urine for myoglobin, hemoglobin
- Patients with white phosphorous burns:
  Serial estimation of serum Ca^{++} for hypocalcemia and serum PO_4 for hyperphosphatemia.

Imaging Studies

X-ray chest is performed on all patients exposed to high blast over pressure to rule out primary blast injury.

- All patients with TM rupture
- With respiratory symptoms
- Abnormal findings on auscultation.
- Visible signs of thoracic trauma.

Look for pneumothorax, hemopneumothorax, primary blast lung with butterfly infiltrates, fracture ribs, cardiomegaly and gas under diaphragm.

X-ray abdomen (standing) for detection of pneumoperitoneum due to bowel perforation.

FAST (focused abdominal sonography in trauma) is useful for rapid screening of mass casualty patients.

- Positive FAST in unstable patients is an indication for surgical exploration of abdomen in OR.
- Positive FAST in stable patients can be sent for CT abdomen.
- Negative FAST is unreliable in penetrating trauma, further follow-up with CT abdomen.

Radiation contamination can be detected with the help of radiation safety officer using Geiger counter or similar radiation dosimeter.

Anesthesiologist's Perspective to Blast Event

A: Assess Incident Size and Severity

After blast event latent period of 20 minutes is there in which events are occurring outside the hospital. This period may be used for departmental wakeup for mass casualty and equipment preparedness for O_2 airway equipment including difficult airway cart, suction, IV supplies, drugs, monitoring devices and mobile mass casualty carts.

B: Backup

On call anesthetists and recruiting additional anesthetists with trauma expertise may be required.

C: Casualty Care

- Patient evaluation is done
- Airway assessment and protection
- Confirmation of tracheal tube position inserted at scene
- Intubation
- Initiation of mechanical ventilation
- Venous assess and volume resuscitation.
- Send laboratory investigations.
- Nasogastric tube/Foley's catheter
- Injection tetanus toxoid (TT) and analgesics
- Intercostal drainage (ICD) tube insertion.

D: Definitive Treatment

Anesthesiologist may be involved in one of the following areas:[10]

Operation theatre: For surgical procedures.

Radiological suite Patients may be taken up for diagnostic or interventional radiological procedures for vascular damage from shrapnel injuries. There is risk of renal damage due to contrast used in angiography.

ICU: To provide close monitoring and critical care. Intensivist should identify shiftable patients to the wards and other areas.

Anesthesia Management

Preoperative Evaluation

History: May be asked by written question in presence of acoustic trauma:

- Age, weight, married status
- Symptoms suggestive of systemic, abdominal, airway, extremity injuries
- Presence of ICD, prophylactic ICD may be inserted prior to general anesthesia
- Details of blast
- Response to resuscitation, inotrops, urine output
- Past and personal history.

Examination

General Examination

Detailed general and systemic examination is to be done to rule out other injuries. Pulse, BP, pallor, CVP, airway assessment, facial burns, and cervical spine fracture must be noted. Do not forget to examine patient posteriorly for penetrating entry and exit wounds which may contribute to significant blood loss.

Systemic Examination

- RS—For blast lung and barotraumas, ICD status
- CVS—For penetrating trauma and cardiac tamponade
- CNS—Glasgow Coma Scale score and power in all four limbs
- P/A—Distention, guarding, rigidity, penetrating trauma.

Laboratory investigations: as described earlier.

Consent and starvation: All patients are considered full stomach as starvation period is only between food intake and time of injury. Nasogastric tube if *in situ* is aspirated prior to anesthesia. Antiaspiration prophylaxis must be given.

Premedication

- Antisialogouge—Glycopyrrolate or Atropine (in presence of bradycardia)
- H_2 recepter blocker—Ranitidine
- Antiemetic and prokinetics—Ondensetron, Metoclopramide
- Analgesics—Opioids like fentanyl, tramadol, buprenorphine, morphine, etc. as per requirement of

patient and severity of injury. NSAIDs are to be avoided if coagulation profile is deranged.
- Antibiotics should be given after test dose.
- Tranexamic acid (10 mg/kg) may be used to reduce intraoperative blood loss.
- Inotropic support if required is to be continued and dose required for drugs used (dopamine, dobutamine, adrenaline, noradrenaline) should be noted.

IV access: Two large bore (18 G, 16 G) peripheral veins must be secured.

Central venous access for CVP monitoring is obtained in patients with unresponsive shock, blast lung, major burns and for giving inotropic support. In blast patients there can be limited choice for venous access depending upon the type of injury and the area involved. The presence of edema further makes this difficult.

Monitoring

There should be high index of suspicion for intraoperative development of pnuemothorax and systemic air embolism especially after intubation and IPPV.[1,12]
- ECG—arrhythmias
- Pulse oximetry—blast lung
- $ETCO_2$—blast lung
- Airway pressure
- Blood pressure—noninvasive or invasive BP depending on patients hemodynamic status and magnitude of injury
- Temperature—nasal, esophageal, rectal to avoid life-threatening hypothermia
- Urine output—to know adequacy of tissue perfusion and for myoglobinuria, hemoglobinuria, blood transfusion reactions.
- ABG, serum electrolytes.

Choice of Anesthesia

Monitored Anesthesia Care

Diagnostic and interventional angiographies for vascular injuries, bed side debridements in ICUs, pediatric central venous access, etc. may require monitored anesthesia care. Starvation is usually inadequate, hence avoid deep sedation if airway is not protected. Midazolam, fentanyl, ketamine (rule out head injury), propofol (in hemodynamically stable patients), sevoflurane may be used.

Regional Anesthesia

Extremity surgeries with normal platelet count and coagulation profile without head injury may be preferably done under regional anesthesia to avoid intubation and IPPV. But regional anesthesia is contraindicated in polytrauma patients with hypovolemic shock, coagulopathy, suspected head injury, etc.

General Anesthesia

General anesthesia is required for all exploratory laparotomies, craniotomies, thoracotomies, hemodynamically unstable patients, surgery in prone position, coagulation derangement, etc.

Induction of Anesthesia

- Consider prophylactic ICD prior to induction of general anesthesia.[7]
- Adequate preoxygenation to avoid desaturation in already compromised pulmonary function due to blast lung.
- Induction agent is selected depending on hemodynamic parameters, head injury component, etc. Commonly used agents are thiopentone sodium, propofol, ketamine in slow graded doses.
- Tracheal intubation may be done via oral or nasal route. If prolonged ventilatory support is anticipated, nasal route may be preferred.
- One lung ventilation may be required in suspected bronchial injury with risk of systemic air embolism.
- In anticipated difficult airway awake fiberoptic or awake blind nasal intubation may be tried depending on anesthetist's expertise.
- In unanticipated difficult airway proseal LMA (laryngeal mask airway) can be life saving. Other difficult airway equipments like intubating LMA, intubating stylet, different types of laryngoscope blades and handles, needle cricothyrotomy set and tracheostomy tubes must be available in emergency mass casualty situation.
- Succinylcholine is to be avoided in patients with burns, crush injury, compartment syndrome due to risk of life-threatening hyperkalemia.
- Rapid sequence induction with modified Sellick's maneuver is preferred as trauma patients are considered to be full stomach. Rocuronium may be used if succinylcholine is contraindicated.
- Peak inspiratory pressure is to be limited to 35-40 cm H_2O by low tidal volume and high respiratory rate if blast lung is suspected. ICU type ventilator may be required inside the operation theater.[1,12]

Maintenance

Hemodynamically unstable patients requiring maximal dose of inotrops should be maintained on 100% O_2 till bleeding is controlled to maintain vital organ perfusion.

Inhalational agents and propofol may be used depending on hemodynamic parameters. Avoid N_2O in nondecompressed suspected pneumothorax, pneumomediastinum and pneumocephalus. Avoid flammable inhalational agents and give low O_2 for patients with white phosphorous burns. Sedation and analgesia can be repeated as per duration of surgery and hemodynamic status. Avoid the deadly triad of hypothermia, coagulopathy and metabolic acidosis, as each condition worsens both of the others. Intraoperative blood loss should be minimized by use of extremity tourniquets. Proper estimation of blood loss and its timely replacement as per blood transfusion guidelines is important for maintaining hemodynamic stability.

Reversal and Extubation

Prophylactic postoperative ventilatory support may be required in patients with poor preoperative GCS, hemodynamic instability with inotropic support, moderate to severe blast lung, multiple rib fractures with flail segment, massive blood transfusion, and impaired coagulation with risk of DIC and further bleeding, perioperative cardiorespiratory arrest revived with CPCR. These patients may be kept in SICU, PACU, Trauma ICU, MICU or recovery room wherever facilities are available.

Postoperative Care

Continue same monitoring in postoperative period for patients on ventilator.

Pregnancy and Blast Injury

There is risk of placental abruption due to shear injuries. All pregnant patients exposed to the blast in second and third trimester should be admitted for fetal monitoring and further testing and evaluation.

BURNS

While evaluating burns patient for any emergency surgery, an anesthetist should avoid tunnel vision as burns injury causes multisystem involvement. Major burns may be defined as follows depending on the extent, depth, location, etiology and associated trauma.[15]

- Third degree full thickness burns involving >10% TBSA (total body surface area) burn
- Second degree partial thickness burns involving >20% TBSA at extremes of age and >25% TBSA in adults
- Burns on face, hands, feet, perineum, major joints
- Inhalational burns
- Chemical burns
- Electrical burns
- Burns patients with coexisting medical disorders
- Burns with associated trauma.

Assessment should include following points:

1. Age of patient, weight and general health. Mortality and morbidity is high in extremes of age groups (children < 2 years and elderly). They have low immunity and fluid resuscitation and monitoring is difficult.
2. Cause of burn – thermal, chemical, electrical, others (radioactive).
3. Depth of burn:[16,17]
   - Ist degree – superficial burns involving only epidermis.
   - IInd degree – superficial—injury to epidermis and superficial papillary dermis.
     – deep—injury to epidermis and deep reticular dermis
   - IIIrd degree – full thickness injury to epidermis and dermis.
   - IVth degree – injury involving skin, subcutaneous fat with muscle or bone injury.

   This assessment is important to determine the analgesia requirement, surgical treatment required in the form of escharotomy, fasciotomy, excision and grafting, amputation, etc.
4. Extent of burn injury:[16,17]
   a. Wallace's "rule of nines" is appropriate for accurate estimation of the total body surface area (TBSA) burn in adult patients (Fig. 32.1). Patient's palm is approximately 1% TBSA and can be used for estimating patchy areas.
      Head/neck—9% TBSA
      Each arm—9% TBSA
      Anterior thorax—18% TBSA
      Posterior thorax—18% TBSA
      Each leg—18% TBSA
      Perineum—1% TBSA
   b. The Lund and Browder method covers all age groups and is considered most accurate in pediatric age group.
   c. If Lund and Browder chart is not available:
      - For child < 1 year; head = 18%; leg = 13.5%
      - For child > 1 year; add 0.5% to leg, subtract 1% from head, for each additional year until adult value.

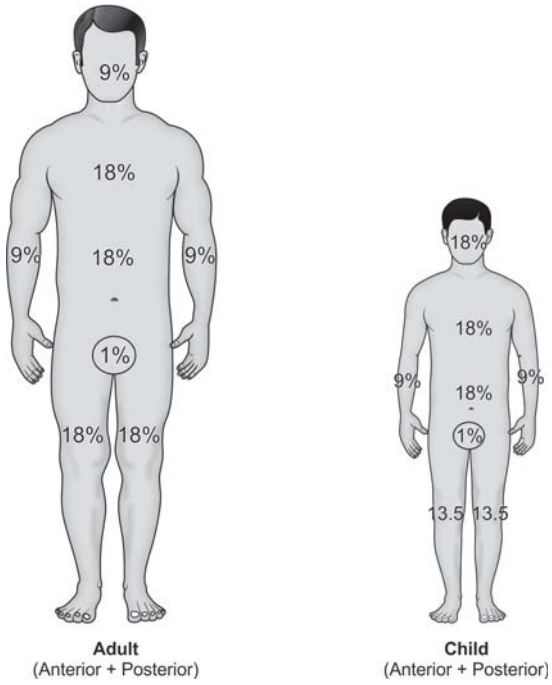


Fig. 32.1: Rule of nine

5. TBSA% calculation is important guide for fluid replacement therapy, as % TBSA burned determines the loss of fluid electrolytes and thus the severity of burn shock.
6. Localization of burns: Burns on face, neck may cause airway edema and inhalational injury, vital sense organ injury (eye, ear, nose). Burns over perineum has high infection rate.
7. Associated inhalational injury is to be suspected if there is fire in an enclosed space, disturbed consciousness, facial burns involving nose, lips, mouth, singed eyebrow or nasal hairs, carbonaceous sputum, cough, wheezing, hoarseness, stridor, tachypnea, apnea, convulsion and coma.[17,18] In presence of airway edema, intubation difficulty is anticipated and appropriate difficult airway gadgets are to be kept ready including smaller size tracheal tubes, different types of laryngoscope blades and handles, intubating stylet, fiberoptic bronchoscope and tracheostomy tubes, etc.
8. Other associated injuries like head injury, fractures of cervical spine and long bones.
9. Comorbid conditions like hypertension, diabetes, epilepsy, addictions, drug allergies, pregnancy in females, psychiatric illness.
10. Adequacy of resuscitation and patients response to resuscitation.
11. Infection.

Anesthetist may be involved in burn care as follows:

a. Acute resuscitation
   - Airway protection
   - IV access (Peripheral and central)
b. Monitored anesthesia care and pain relief—Burn dressings
c. Anesthesia for
   - Extensive burn dressings
   - Escharotomies
   - Fasciotomies in compartment syndrome
   - Amputations – mainly with external electrical injury
   - Excision and skin grafting
   - Fiberoptic broncoscopy
   - Burns with other injuries like craniotomies, explorations, extremity fractures
   - Tracheostomy standby.
d. Intensive care management:

Burns injury leads to multisystem involvement hence knowledge of burn pathophysiology is must before anesthetizing burn patient for any surgery.

PATHOPHYSIOLOGY OF BURNS

Hemodynamic Changes

Burn injury triggers local as well as systemic inflammatory response due to release of inflammatory mediators like histamine, serotonin, kinins, cytokines, oxygen free radicals.[16] This results in increased capillary permeability to proteins and water, leading to edema formation in burnt tissue and non-burnt tissue in > 25% TBSA burn. Capillary leakage leads to hemoconcentration, hypovolemia, hypoproteinemia, electrolyte imbalances and acid base disturbances which may lead to burn shock.[15]

In early phase, there is fall in cardiac output with increase in systemic and pulmonary vascular resistance with or without development of pulmonary edema.

In late phase (3-5 days), there is hypermetabolism and hyperdynamic circulation with tachycardia, hypertension, hyperthermia and increased protein catabolism.

Increased systemic vascular resistance is due to release of endogenous catecholamines, vasopressin, angiotensin II, neuropeptide Y and increased blood viscosity.

Increased pulmonary vascular resistance and pulmonary capillary wedge pressure may be due to

release of serotonin in developing pulmonary edema, inhalational injury, hypoproteinemia, left heart failure and late restoration of capillary integrity (48-72 hours) after injury with edema fluid reabsorption.[18] Fall in cardiac output is also attributed to ventricular dysfunction from myocardial depressant factors.

Respiratory Changes

Wheezing, bronchospasm may be due to inhalational injury or systemic inflammatory response. There may be airway edema (due to inhalational burns, systemic response, postresuscitation), pulmonary edema, atelectasis, collapse, consolidation due to secondary infection, ARDS.[15,18] Increased capillary permeability with mucosal irritation, decreased surfactant level and mucociliary clearance may lead to above pulmonary complications.

Gastrointestinal

Paralytic ileus and Curling's ulcers may cause endotoxemia due to disruption of gastric mucosal barrier in extensive burns. Nasogastric tube placement with early enteral feeding, antacids and H_2 receptor blockers can minimize this damage.[16]

Renal

Decrease cardiac output with hypovolemia leads to decrease in renal blood flow and glomerular filtration rate. This may lead to oliguria, acute tubular necrosis with renal failure. Other causes of renal failure may include myoglobinuria, septicemia, nephrotoxic drugs and contrast media.[15] There is activation of renin angiotensin system with resultant retension of sodium, water and excretion of potassium, calcium and magnesium.[18] Extensive tissue damage with hemolysis may cause hyperkalemia.

Cerebral

Hyponatremia complicating resuscitation may result in burn encephalopathy syndrome with cerebral irritability.[18]

Hematologic

Hemoconcentration leads to increase in hematocrit in first 48 hours. Due to increase erythrocyte destruction (hemoglobinemia), decrease production and survival of erythrocytes, burn patients are often anemic.[16] Sepsis, blood loss during surgery may further aggravate anemia. Platelets and coagulation factors are often decreased due to dilution and consumption.[18]

Metabolism

Release of catabolic harmones like glucocorticoids, catecholamines, glucagon leads to hypermetabolism in patients of severe burns. In this phase, there is tachycardia, increased cardiac output, increased oxygen consumption, proteolysis, lipolysis gluconeogenesis and negative nitrogen balance. This leads to increased chance of infection and impaired wound healing.[16] Total parenteral nutrition may be required to meet these increased metabolic demands.

Thermoregulation

Loss of protective skin barrier leads to extensive evaporative water loss leading to hypothermia in early stage. In hypermetabolic stage, body core temperature may be slightly raised above normal.[18]

Sepsis

There is global depression in immune system. Loss of protective skin barrier leads to invasion of bacteria. Burn patients are susceptible for number of infectious complications like bacterial wound infection, pneumonia, fungal, viral infections, central line sepsis etc.[18]

At Site Management

Triage[19]

Triage is the cornerstone of effective burn disaster management and is done at the disaster site by staff with knowledge of burn treatment. Triage takes into consideration the total number of patients, bed availability and transportation capacity.

Triage should be prognostic and patients should be categorized on the basis of age, extent of burns, site of burns and presence of inhalational injury:

- Group I—Minor burns (< 10% of total surface area in children, < 20% in adults) to non-critical sites.
  Assigned to—Outpatient care, dressing, tetanus prophylaxis
- Group II—Minor burns to critical sites (face, hands, genitalia)
  Assigned to—Short hospital stay, special wound care or operation
- Group III—Major burns (20-60%)
  Assigned to—Admission to burn unit, intravenous resuscitation
- Group IV—Extensive burns (>60%)
  Assigned to—Lower priority for transfer
- Group V—Major burns with inhalational injury or associated injury

Assigned to—Oxygen, intubation, transfer to intensive care unit.

The patients in groups III and V are evacuated first, followed by group IV. Group II cases are evacuated at the end. Group I cases are either discharged after first aid or asked to make their own way to the nearest primary care centre.

Stop Burning Process

- Use fire extinguisher to extinguish fire.
- Turn off electricity.
- Remove all clothing and constricting ornaments.
- Brush off chemical powders from contaminated clothes and wounds in case of chemical burns.
- Cool the burnt area with cold water (15°C) for 20 to 30 minutes, this limits the extent and depth of burn.
- Ice should not be applied directly on burnt area as it may increase tissue damage by vasoconstriction and lead to hypothermia.
- Do not stand upright to avoid inhalational injury.
- In electrical burns, look for entry and exit wounds.
- Wrap the patient in warm, clean and dry linens to avoid hypothermia.

RESUSCITATION

Initial approach of burn patient should be as per ATLS guidelines for polytrauma patient.[2]

Primary survey includes:

A – Airway
B – Breathing
C – Circulation
D – Disability
E – Exposure.

A—Airway with Cervical Spine Control [20,21]

1. Assess for:
   - Airway injury
   - Airway obstruction
   - Pre-existing airway abnormality
   - Post resuscitation airway edema
   - Unconsciousness due to trauma or CO poisoning or smoke inhalation
2. Facial and neck burns—difficult mask holding and difficult laryngoscopy (Fig. 32.2).
3. Difficult intubation gadgets to be kept ready along with basic airway adjuncts.[22]
4. There should be low threshold for elective intubation in cases of airway edema. Identify 'at risk' airway and intubate early before airway control is lost. For patients with markedly difficult airways, immediate supervised transport of the patient to the operating room may be appropriate. In this location, the anesthesiologist has skilled assistance, a full range of intubation equipment, an anesthesia machine and the best environment in which to secure the airway surgically should that be necessary.[15]

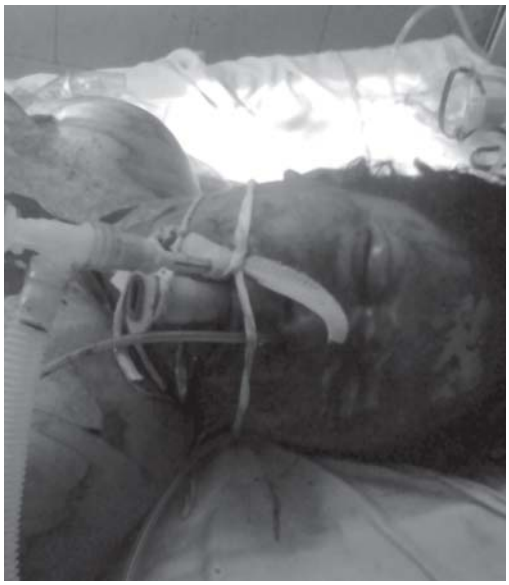


Fig. 32.2: Severe facial edema leading to difficult intubation
(For color version, see Plate 3)

- *Tracheal Intubation of the Adult with 'at risk' Airway:* With difficult airway or upper airway obstruction, the safest way to secure the airway is with the patient awake.[20] Key prerequisites include effective topical anesthesia, proper patient positioning, and supplemental oxygen administration. Intravenous opioid administration is appropriate for the alert patient in pain, but sedatives may worsen airway obstruction and should be used cautiously, if at all.[15] Although the best technique will depend on the operator's expertise, the flexible fiberoptic scope is well suited to this situation. Alternatives include direct laryngoscopy, the laryngeal mask airway, and the Bullard laryngoscope. In uncooperative patients when general anesthesia is required, an inhalational induction with spontaneous ventilation may be performed before tracheal intubation. When the upper airway is badly damaged and tracheal intubation is not possible, a direct surgical approach to the airway is indicated.
- *Tracheal Intubation of the Child with 'at risk' Airway:* For children, an inhalational induction with oxygen and a volatile agent before airway

manipulation is probably the safest technique. Sevoflurane, with a lower solubility in blood and minimal airway irritation, may offer the advantage of a more rapid induction.[15] Once the patient is anesthetized, pediatric fiberoptic and Bullard laryngoscopes may be useful when direct laryngoscopy is difficult. The laryngeal mask airway may act as a valuable adjunct in maintaining airway patency and as a guide for fiberoptic intubation in patients with severe airway edema. The uncuffed tracheal tubes should be used in infants and young children. However, in larger children, particularly those requiring high inspiratory pressures during mechanical ventilation, place cuffed tracheal tubes. Postextubation stridor is a recognized complication of long-term tracheal intubation in pediatric burn and trauma patients. It is best to wait until an air leak occurs around the endotracheal tube before tracheal extubation, because this indicates resolution of edema. If there is still no air leak, direct laryngoscopy may be necessary to determine the extent of residual edema prior to extubation. After extubation the patient should be closely monitored for progressive airway obstruction during the subsequent 24-48 hours.[15]

- *Tracheal Intubation for Patients with a Normal Airway:* In the absence of an airway difficulty tracheal intubation will usually be achieved using a rapid sequence technique with an intravenous induction agent and a rapidly acting muscle relaxant. The conventional wisdom is that administration of succinylcholine is safe only within 24 hours of a burn.[20] How long the hyperkalemic response to succinylcholine persists is also unclear. Rocuronium or high doses of other nondepolarizing muscle relaxants (e.g. vecuronium or cisatracurium) are attractive alternatives to succinylcholine.[15,21]
- *Long-term Airway Management:* Most patients with respiratory failure can be managed with tracheal intubation. The advantages of tracheostomy include easier oral and tracheal hygiene and easier replacement if the tube is dislodged. But it has some surgical risk, the potential for airway scarring from both the tracheal tube and tracheotomy, and it leaves a permanent neck scar. The long-term upper airway sequelae such as tracheal stenosis, tracheoesophageal fistula, and tracheoarterial fistula may occur in these patients.[15]

5. In presence of severe upper airway damage, laryngoscopy and tracheal intubation may be difficult or impossible, hence surgical airway in the form of needle cricothyroidotomy, surgical cricothyroidotomy, or tracheostomy must be kept ready in case of failed intubation and severe laryngeal edema. In this situation surgical airway can be life saving.
6. After securing airway, give 100% humidified oxygen via non-rebreathing mask or tracheal tube.
7. Propped up position 30 to 90° to decrease facial and airway edema after ruling out cervical spine injury.
8. Intubation and mechanical ventilation may be required if PaO_2 < 60 mm Hg, $PaCO_2$ > 50 mm Hg, PaO_2/FiO_2 ratio < 200, with impending respiratory/ventilatory failure and severe upper airway edema.[16]

   Mechanical ventilation is indicated in:
   - Deep extensive burns > 60% TBSA
   - Tracheobronchial thermal injury
   - Facial and cervical burns
   - Severe inhalational burn injury from smoke inhalation
   - Carbon monoxide intoxication
   - Moderate to severe blast lung injury.

B—Breathing

In burns patients breathing may be affected due to presence of inhalational injury.

- Circumferential chest wall burns may require escharotomies to avoid respiratory compromise.
- Blast injuries may involve pulmonary system as discussed earlier.
- Signs and symptoms include disturbed consciousness, facial burns involving nose, lips, mouth, singed eyebrow or nasal hairs, carbonaceous sputum, cough, wheezing, hoarseness, stridor, tachypnea, apnea, convulsion and coma.

Inhalational injury may occur due to following mechanisms:[15,18]

- Direct thermal injury from hot gases leading to damage from upper airway to carina and chest wall.
- Damage due to cellular and oxygen transport process — carbon monoxide (CO) and cyanide (CN) poisoning.
- Chemical injury to lower airways—toxic products from fire.

Carbon monoxide poisoning:[18,21,23] Carbon monoxide has 200 times high affinity for hemoglobin leading to formation of carboxyhemoglobin (COHb). It hence causes shift of oxyhemoglobin dissociation curve to the

left. There is decrease in oxygen carrying capacity of blood thereby decrease oxygen delivery to the tissues, leading to tissue hypoxia and metabolic acidosis. Carbon monoxide binding to myoglobin also leads to myocardial stunning due to mitochondrial dysfunction. Patient may present with headache, confusion at 15 to 20 percent COHb level, nausea, vomiting, fatigue at 20 to 40 percent COHb level, hallucinations, ataxia, seizures, coma, cardiovascular instability at 40 to 60 percent COHb level and death above 60 percent COHb level. Patients may have cherry red mucous membranes.

Half life of carbon monoxide at room air — 4 hours;
100% oxygen — 40 to 60 minutes;
hyperbaric oxygen — 23 minutes.

Pulse oximeter may show normal readings even with lethal concentration (>50%) of COHb. A cooximeter, which measures the percentage of hemoglobin, oxyhemoglobin, carboxyhemoglobin, and methemoglobin, is needed to obtain an accurate pulse oximetry saturation.[18] PaO_2 on arterial blood gas (ABG) may be normal with raised COHb levels. Hence, ABG for estimation of COHb levels should be obtained. More than 15 percent COHb levels are toxic and more than 50 percent are lethal. Treatment is with 100 percent humidified oxygen via non-rebreathing mask, ventilatory support as and when required and monitoring for cardiac arrythmias. Severe cases may need hyperbaric oxygen therapy.

Cyanide poisoning: Hydrogen cyanide is toxic gas produced by burning of nitrogenous materials like wool, silk, synthetic polymers. Cyanide causes tissue asphyxia by inhibiting mitochondrial cytochrome oxidase which is final step in oxidative phosphorylation (adenosine triphosphate). This avoids use of oxygen by mitochondria leading to anaerobic metabolism for ATP formation and metabolic (lactic) acidosis.[14] Along with CO poisoning, cyanide toxicity may be difficult to diagnose. Signs include headache, dizziness, tachycardia, tachypnea. Above 100 ppm seizures, coma, respiratory failure, death may occur. Cardiac toxicity may mimic myocardial ischemia with ST segment elevation on ECG. Anion gap metabolic acidosis not responding to 100 percent oxygen and high mixed venous oxygen saturation may suggest CN toxicity.[15,18,23]

Treatment may include thiosulfate, vitamin B_{12} and sodium nitrite.

Chemical injury: Toxic products of combustion may damage lower airway with damage to the epithelium and capillary endothelium of alveoli. This leads to decrease in mucociliary clearance, atelectasis and collapse.

Diagnosis of inhalation injury is based on relevant history, signs and symptoms specified earlier in evaluation, ABG for COHb levels, X-ray chest, fiberoptic bronchoscopy and X^{133} (Xenon) lung scan.[23,24]

Treatment of inhalation injury includes ventilatory support, early and aggressive pulmonary toilet, bronchoscopic removal of casts, and nebulization therapy, including acetylcysteine, heparin (300-1000 IU/kg/day for 3-5 days), and albuterol.[16,23,24]

C—Circulation

Rapid assessment of volume status is to be done in burn patients. Securing 2 large bore IV access in unburnt area is essential for institution of fluid resuscitation.[25] Unusual sites may have to be selected for this purpose. Preferred sites are cubital, femoral, neck veins, saphenous veins. Circumferential burns in extremities may require escharotomies and fasciotomies.

Applying monitors may be difficult. BP cuff, ECG leads may be stapled to skin. Various formulae are available for fluid resuscitation. They are only guide and assessment should be based on clinical parameters like pulse, blood pressure, central venous pressure (CVP), urine output, capillary refill, temperature, conscious status.[15] Crystalloid formulae most commonly used are given in Table 32.4.

The Parkland formula is most commonly used for resuscitation. In children, maintenance fluid at hourly basis is given according to 4 ml/kg for first 10 kg + 2 ml/kg for 2nd 10 kg + 1 ml/kg for every kg thereafter. In children more than 10% TBSA burn, IV fluids are required. Oral feeds or breast/feeding can be continued if facial burns are absent. Urine output of 0.5-1 ml/kg/hr in adult and 1-2 ml/kg/hr in children is considered adequate resuscitation.

Recently, American Burn Association guidelines have accepted 'Rule of Ten' i.e. initial fluid rate in ml/hr

Table 32.4: Most commonly used crystalloid formulae[18,25]

*F*ormulas	IVF	Rate 1st 24 hours	Next 24 hours
Parkland	RL/NS	4 ml/kg/% TBSA burn*	Colloid at 20-60% of calculated plasma volume to maintain adequate urine output
Modified Brooke	RL	2 ml/kg/% TBSA burn*	Colloid at 0.3-0.5 ml/kg/% TBSA burn + 5% D to maintain urine output.

* 50% of calculated volume is given in first 8 hours, 25% in next 8 hours and remaining 25% is given during third 8 hours.

= % TBSA X 10. For every 10 kg above 80 kg, 100 ml is to be added.[26] Fluid calculated is from the time of injury and not from the time of admission.

- Patient requiring > 6 ml/kg/% TBSA in 24 hours may require invasive monitoring in the form of CVP, pulmonary artery pressure (PAP), arterial line and inotropic support, acidosis correction, early use of colloid (12-18 hours instead of 24 hours).[18]
- Additional fluids are commonly needed in patients with inhalational injuries, diuretic use, electrical burns, associated trauma and escharotomy, delayed resuscitation, patients under influence of alcohol.
- Blood for serial determination of hematocrit, complete blood count, serum electrolytes, glucose, albumin, osmolality, blood urea and creatinine, COHb levels and arterial blood gas analysis should be sent. Urine for myoglobin and hemoglobin is tested and if present, forced alkaline diuresis with IV sodium bicarbonate or acetazolamide with IV mannitol is to be done. Chest X-ray should be done to rule out inhalational injury, ECG is to be done for electrical burns.

D — Defibrillation: May be required in electric burns for ventricular tachycardia and fibrillation.

D—Disability

Assess neurologic status of patient with Glasgow Coma Scale score (GCS). It may be low due to hypoxia, hypovolemic shock or head injury.

E—Exposure

Remove all constricting clothes and ornaments as burns may lead to rapid edema formation. Cover patient in clean, warm linen to avoid hypothermia due to evaporative loss from burnt area.

Analgesia

Analgesics mainly opioids (like fentanyl, morphine), partial opioid agonists (such as nalbuphine or butorphanol) ketamine, etc. can be used.[24] Analgesics should be given by intravenous route only and in small incremental doses as intramuscular, subcutaneous injections may have erratic absorption and delayed effect.[15]

Catheterize urinary bladder to monitor urine output.

Nasogastric tube is to be inserted to decompress stomach and avoid risk of aspiration pneumonitis in patients with >20% TBSA burn with:

- Inhalational injury, facial burns.
- Patients with paralytic ileus with nausea, vomiting, abdominal distension.
- For nasogastric feeding as early as 24 to 48 hours or once adynamic ileus resolves. Causes of adynamic ileus can be large burns, hypokalemia, narcotic overuse, sepsis, etc.

Tetanus prophylaxis for all patients should be given. Antibiotics are reserved for treatment of infection. Barrier nursing care is must in burns patient management for infection control.

Secondary Survey

This includes assessment of concomitant injuries of other systems.

Neurologic	–	Head injury, fracture cervical spine.
ENT	–	Airway burns, corneal burns.
Chest	–	Circumferential burns leading to decreased chest wall compliance.
Abdomen	–	Paralytic ileus, Curling's ulcer.
Extremities	–	Compartment syndrome.
Renal	–	Urine output, pigments.

Anesthesia Considerations for Emergency Surgeries in Burns

Complete preanesthetic evaluation with history and physical examination should be done for systemic involvement of burn injury. Airway assessment for difficult intubation should include mallampatti class, thyromental distance, mouth opening, neck mobility, upper airway edema and facial burns.[27] Larynx may be fixed anteriorly due to edema, scarring and development of neck contracture later on leading to progressive intubation difficulty as the contracture undergoes fibrosis (Fig. 32.3).

Laboratory investigations should include complete blood count with hemoglobin and platelet count, PT, PTT, serum electrolytes, blood glucose, COHb levels and arterial blood gases, serum urea, creatinine, blood grouping and cross matching, urine analysis, chest X-ray and ECG.

Starvation in the form of last oral intake and enteral feed must be confirmed. Avoid overstarvation of burns patients as it may affect caloric intake. Starvation period of six hours is considered adequate in our institute. Parenteral nutrition can be continued.

IV Access

Peripheral veins may be difficult to find and must be preciously conserved. Two large bore IV cannulae should be inserted, preferably in non-burned area and not on donor site extremity, to replace blood and blood products in case of excessive bleeding.[27,28] Neck veins,

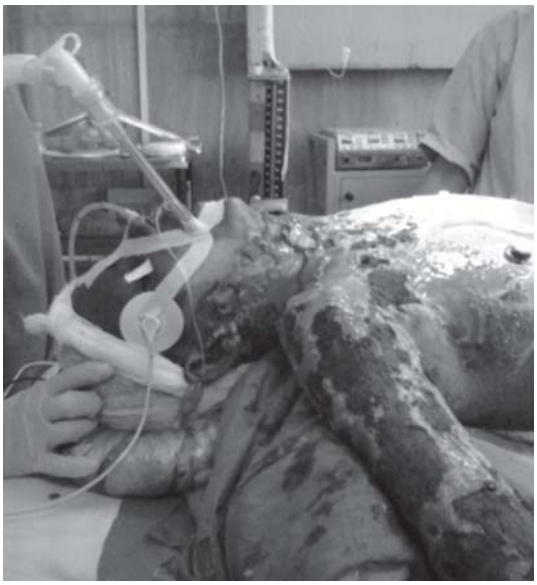


Fig. 32.3: Difficult intubation due to anteriorly fixed larynx
(For color version, see Plate 3)

saphenous, femoral veins can be accessed under strict aseptic precautions. Arterial and CVP lines should only be inserted after due consideration of the infection risk. In case of difficulty, USG guidance may be used.[15]

Monitoring

Cutaneous burns may make conventional monitoring difficult (placement of ECG electrodes, BP cuff, pulse oximeter probes as fingers and toes are burned).

- Consider stapling ECG leads to skin or use of needle electrodes. Alternative sites for pulse oximetry like ear, nose, lip, tongue can be used.[28,29]
- Routine monitoring must include ECG, blood pressure, pulse oximetry, esophageal stethoscope, endtidal CO_2, temperature, neuromuscular monitoring, urine output.
- If large blood loss is anticipated, CVP line and arterial line is considered. Pulmonary artery catheter and transesophageal echocardiography is reserved for patients with underlying cardiovascular disease or hemodynamic unstability.[15]

Preoperative Operation Room Preparation

- To avoid hypothermia:
  - Elevate room temperature 28°C to 32°C with 50% humidity.[28,29]
  - Consider warming devices like fluid and blood warmers, warming blankets, gas humidifiers, etc.
- Difficult intubation:

Fiberoptic bronchoscope, different types of laryngoscope blades and handles, intubating stylets, LMA, intubating LMA, smaller size tracheal tubes, tracheostomy tube, etc.

Premedication

Antisialogouge, antacid, histamine receptor antagonist and antiemetic drugs must be given. Antibiotic prophylaxis is must.

Analgesia and Sedation

Due to development of tolerance, these patients need large doses of opioids.[27] Fentanyl, pentazocin, buprenorphine may be used according to requirement of patient. Avoid deep sedation and analgesia before securing airway in case of difficult airway. Ketamine has been used successfully for analgesia without causing loss of airway reflexes especially in children.[20] Lumbar epidural or femoral nerve block may be given for skin donor site.

Regional Anesthesia

There are several indications for regional anesthesia for burn surgery, either alone or combined with general anesthesia, but these are limited primarily to the patient with small burns. For the patient having surgery below the umbilicus, a lumbar epidural or caudal anesthetic can provide excellent postoperative analgesia. Extensive debridement with the potential for massive blood loss is a relative contraindication to epidural local anesthetic use during operation, but epidural opioids can be used. Probably the greatest limitation to the use of regional techniques is the extent of the surgical field; most patients with major burns will have multiple injuries or need skin harvested from areas too extensive to be easily blocked by a regional technique.[15] In addition, regional techniques should not be performed through burned tissue because of the potential for infection to spread.

Induction

- Mask holding may be difficult due to wet burn surface and ointment. Use wet gauze pads to achieve tight mask seal.[20]
- Presence of upper airway burn is a contraindication to the use of muscle relaxants. Tracheal intubation is to be done with the patient awake, with or without use of fiberoptic laryngoscopy or following an inhalational induction with sevoflurane.[20]
- General anesthesia with an opioid, muscle relaxant and a volatile agent (except halothane as repeated exposure carries risk of halothane hepatitis) is widely used for burn excision and grafting.

Ketamine can be used as primary agent for induction of general anesthesia and analgesia.[24,27] Thiopentone sodium, propofol can be used in adequately volume resuscitated and non-septic patients.[28]

Muscle Relaxants

- Succinylcholine can be used in the first 24 hours for tracheal intubation. Avoid its use for upto two years. It may lead to life-threatening hyperkalemic response due to efflux of potassium from increased acetylcholine receptor sites on muscle membranes. Exact duration of this response is also unclear. Rocuronium can be used as an alternative for rapid sequence induction.[15]
- High doses of non-depolarizing muscle relaxants are required due to development of resistance. This is attributed to high volume of distribution, increased drug elimination and proliferation of acetylcholine receptors.[15,27,29]

Maintenance

Increased oxygen consumption and carbon dioxide production from increased metabolic rate along with ventilation perfusion mismatch due to inhalational injury may require sophisticated ICU-type ventilator with high levels of PEEP and high minute ventilation (as much as 30 L/min) intraoperatively.[28]

Blood and Fluid Requirement

Blood loss may be rapid, diffuse, massive and difficult to control and estimate. Blood loss may be approximately 200 ml/1% BSA excised and grafted. Blood loss can be controlled by restricting the escharotomy to 15 to 20%, use of extremity tourniquets, applying dilute adrenaline (1:10,000) solution topically and using compression bandage. Intraoperatively 8-10 ml/kg/h of intravenous fluids like Ringer lactate or Normal saline may be required to keep urine output of 0.5-1 ml/kg/h.[28] Colloids like tetrastarch or pentastarch (20 ml/kg) can be given if blood loss is within tolerable limit of 10% of total blood volume and while awaiting for the blood products.

When blood products are given rapidly, citrate-induced hypocalcemia may occur requiring treatment with calcium chloride (2.5-5.0 mg/kg) or calcium gluconate (7.5-10.0 mg/kg).[29]

Positioning

Patient may be in supine, lateral or prone position and position may be changed intraoperatively for harvesting skin graft. Take care of tracheal tube, IV cannulae and monitoring devices during position change.

Postoperatively

Avoid extubation in case of suspected airway edema mainly in prone position and massive blood loss.[28] Continue monitoring of vital parameters in postoperative period. Avoid shivering and hypothermia in postoperative period by measures discussed above.

Intravenous fentanyl or morphine may be used in titrated does for postoperative analgesia. Repeat laboratory investigations (CBC, ABG, electrolytes, PT, INR) in case of massive transfusion.

SUMMARY

Providing anesthesia care to blast and burns patients is challenging, but it can be satisfying when the anesthesiologist is successful in taking a severely ill patient through a devastating, deforming, painful, and emotionally stressful process. Once the patient is out of this emergency phase, much prolonged rehabilitative phase begins which requires tender family support and social acceptance.

REFERENCES

1. Leissner KB, Ortega R, Beattie WS. Anesthesia Implications of Blast Injury. Journal of Cardiothoracic and Vascular Anesthesia 2006;20:872-80.
2. Advanced Trauma Life Support for doctors. Student Course manual. 8th Edition. Chicago, American College of Surgeons 2008.
3. CDC Mass Casualties. Explosions and Blast Injuries: A Primer for Clinicians. Available at: http://www.bt.cdc.gov/masstrauma/pdf/explosions-blast-injuries.pdf. Accessed January 2010.
4. Stuhmiller JH, Phillips YY III, Richmond DR. The physics and mechanisms of primary blast injury. In: Bellamy RF, Zajtchuk R, (Eds). The Textbook of Military Medicine-Conventional Warfare: Ballistic, Blast, and Burn Injuries. Washington, DC: Department of the Army, Office of the Surgeon General 1991:241-70.
5. Wolf SJ, Bebarta VS, Bonnett CJ, Pons PT, Cantrill SV. Blast injuries. Lancet 2009;374:405-15.
6. Sorkin P, Nimrod A, Biderman P. The quinary (Vth) injury pattern of blast. J Trauma 2004;56:232.
7. Ohnishi M, Kirkman E, Guy RJ, Watkins PE. Reflex nature of the cardiorespiratory response to primary thoracic blast injury in the anaesthetised rat. Exp Physiology 2001; 86:357-364.
8. Chaloner E. Blast injury in enclosed spaces. BMJ 2005;331: 119-20.

9. Phillips YY III, Richmond DR. The management of primary blast injury. In: Bellamy RF, Zajtchuk R, (Eds). The Textbook of Military Medicine- Conventional Warfare: Ballistic, Blast, and Burn Injuries. Washington, DC: Department of the Army, Office of the Surgeon General 1991:295-335.
10. Sandra Wanek , John C. Mayberry. Blunt thoracic trauma: Flail chest, pulmonary contusion, and blast injury. Crit Care Clin 2004;20:71– 81.
11. Pizov R, Oppenheim-Eden A, Matot I, Weiss YG, Eidelman LA, Rivkind AI, et al. Blast lung injury from an explosion on a civilian bus. Chest 1999;115:165-172.
12. Sorkine P, Szold O, Kluger Y. Permissive Hypercapnia ventilation in patient with severe pulmonary blast injury. J Trauma. 1998;45:35-8.
13. Stein M, Hirshberg A. Medical consequences of terrorism. Surgical Clinics of North America 1999;79(6):1537-52.
14. Shamir MY, Weiss YG, Willner D, Mintz Y, Bloom AI, Weiss Y, et al. Multiple casualty terror events: The anesthesiologist's perspective. Anesth Analg 2004;98:1746-52.
15. MacLennan N, Heimbach DM, Cullen BF. Anesthesia for Major Thermal Injury. Anesthesiology 1998;89:749-70.
16. Gallagher JJ, Wolf SE, Herndon DN. Burns. In: Townsend CM, Beauchamp DR, Evers MB, Mattox KL (Eds). Sabiston Textbook of Surgery. The biological basis of modern surgical practice. 18th edn. Philadelphia: Saunders Elsevier 2008;559-85.
17. Clarke J. Burn. In: Ellis BW, Paterson-Brown S (Eds). Hamilton Bailey's Emergency surgery. 13th edn. Arnold Publishers 2000;137-42.
18. Bittner EA, Grecu L, Martyn JA. Evaluation of the burned patient. In: Longnecker DE, Brown DL, Newman MF, Zapol WM, Editors. Anesthesiology. New York: McGraw Hill 2008;1674-85.
19. Ahuja RB. Burns in the developing world and burn disasters. BMJ 2004;21:447–9.
20. Lamb JD. Anesthetic considerations for major thermal injury. Can Anaesth Soc J 1985; 32: 84-92.
21. Blanding R, Stiff J. Perioperative anesthetic management of patients with burns. Anesthesiology Clinics of North America. March 1999;17:237-250.
22. The Royal College of Anaesthetists. Burns and plastics anaesthesia. Guidelines for the Provision of Anesthetic Services 2009.
23. Mlcak RP. Respiratory management of inhalation injury. Burns 2007;33:2–13.
24. Wesley RL. Burn injuries. In: Zajtchuk R, Christopher M, Editors. The Textbook of Military Medicine—Anesthesia and Perioperative Care of the Combat Casualty. Washington, DC: Department of the Army, Office of the Surgeon General, 1995:521-42.
25. Yowler CJ. Burn injuries critical care in severe burn injury. In: Smith CE (Ed). Trauma anesthesia. New York: Cambridge University Press 2008;314-21.
26. Alvarado R, Chung KK. Burn Resuscitation. Burns 2009;35:4-14.
27. Lovich-Sapola JA. Anaesthesia for Burns. In: Smith CE Editor. Trauma Anesthesia. New York: Cambridge University Press 2008;322-42.
28. Giffard RG, Press BH, Solem LD. Free skin graft for burn wound. In: Jaffe RA, Samuels SI (Eds). Anesthesiologist's manual of surgical procedures, 2nd edn. Philadelphia: Lippincott Williams & Wilkins 1999;817-21.
29. Capan LM, Miller SM. Burns. In: Barash PG, Cullen BF, Stoelting RK (Eds) Clinical Anesthesia. 5th edn. Philadelphia: Lippincott Williams & Wilkins 2006;1279-93.

33

Anesthesia for Maxillofacial and Upper Airway Trauma

Geeta A Patkar, Hema B Gupta

KEY POINTS

- A patient presenting with a facial fracture is likely to be a young intoxicated male, who has sustained a road traffic accident (RTA) or interpersonal violence. The incidence of pre-existing comorbidities is likely to be low.
- Maxillofacial trauma (MFT) may compromise the upper airway and airway injuries may be associated with MFT.
- Facial bones, the base of the skull and the upper airway are closely related anatomically and have important clinical implications.
- MFT due to blunt injuries are more common. They are usually associated with RTAs, interpersonal trauma, sports-related trauma, occupational injuries, and falls. The midfacial bones can be easily fractured by low impact forces.
- Blunt injuries to the upper airway most commonly occur due to a direct blow often seen in the "Padded Dash Board Syndrome".
- Penetrating maxillofacial and upper airway injuries occur due to gunshot wounds, stabbing, and explosions. They are usually associated with severe bleeding, hematoma formation and airway obstruction.
- The mandible is commonly fractured in a MFT. A bilateral fracture of the body of the mandible is known as the "Andy Gump" fracture. This fracture can be dangerous as in the supine position causing airway obstruction at the oropharynx.
- Midface fractures are commonly classified according to the three Le Fort types.
- Diagnosis of a base of skull fracture is essential as it has important clinical implications, due to communication between the airway and the intracranial cavity.
- Head injury and cervical spine injury is common along with MFT. Airway management should be done with minimal movement of the cervical spine.
- Airway patency is the priority in acute management of maxillofacial and upper airway injuries. Loss of airway kills more quickly than does the loss of the ability to breathe or any circulatory problems. Hemorrhage from the midface or base of skull may occasionally be massive, and in severe cases, difficult to control.
- Simple measures to clear the airway by suction, lateral posture (provided cervical spine injury is excluded) and insertion of an oropharyngeal airway will suffice in many cases.
- An alert clinician anticipates a respiratory emergency, does a prophylactic intubation or tracheotomy, to avoid complete respiratory obstruction and a chaotic crash intubation or a surgical airway.
- Nasotracheal intubation should not be attempted when midfacial injuries are present, and is absolutely contraindicated when basal skull fractures are suspected.
- Blunt or penetrating injuries to the larynx require immediate attention to the airway. Maintain spontaneous ventilation when possible. Use of direct visualization techniques is preferable when possible.
- Elective surgery for maxillofacial fractures can be planned in 10-15 days after life-threatening emergencies are tackled. In some cases, particularly orbital injuries when ocular function is at risk, early surgery is mandatory.
- The anesthetic management of a patient with maxillofacial trauma can be a challenge. They are long surgical procedures; involve significant blood loss, besides the airway is shared by the surgeon and the anesthesiologist.
- An oral endotracheal tube may not always be acceptable. Alternative airways are therefore required for definitive surgery.
- Patients require a high dependency unit for postoperative care. Endotracheal tube may be retained postoperatively till the patient is fully awake.

INTRODUCTION

Accidents are on the increase in India. It has a world's highest fatality rate in road traffic accidents (RTAs) which is 20 times that of developed countries. In India, eight people get killed for every 100 vehicles, where as in developed countries it is as low as one person for every 1000 vehicles.[1]

Facial fractures occur commonly in such accidents. Trauma to the maxillofacial anatomy carries great significance as it incorporates vital structures in the head and neck region, including systems that control specialized functions like seeing, hearing, smelling, breathing, eating and talking. Injury to the face along with the disfigurement caused can have a great psychological impact.

In developing countries highway discipline is poor and vast majority of facial fractures result from RTA.[2] In a five year retrospective study, Chandra Shekar and Reddy concluded that, RTAs were the common cause for maxillofacial injuries.[3] Men sustained more injuries compared to women. The injuries were mostly sustained in the age group of 11-40 years, constituting about 78 percent of all the injuries. Two wheelers were most commonly involved compared to other vehicles. Influence of alcohol at the time of injury was found in about 58 percent of the patients with maxillofacial injuries. The accidents occurred commonly during the weekends. The most common fractured facial bone was the mandible.

Many studies in the west have studied maxillofacial trauma (MFT). Shapiro and others reviewed 13,000 level one trauma center patients.[4] It was noted that 10.5 percent of the patients had sustained at least one facial fracture. The mortality was 8.7 percent. The male to female ratio was 3:1. RTAs caused 60 percent of the injury followed by blunt interpersonal trauma (19 percent). Facial fractures were more in unrestrained RTA victims, 15.4 percent against 9.5 percent in restrained victims. Non helmeted motorcyclists had a greater incidence of facial fractures (18.4% vs. 4.3%) than helmeted ones. They found that 45 percent of patients with facial fractures were intoxicated upon arrival in the trauma center.

Iida and others studied more than 1500 patients over a 15-year period.[5] The male to female ratio was 2.8. The largest subgroup of patients was between 10 and 29 years of age. Most of the injuries were due to RTAs (52 percent), 16 percent due to falls, 15 percent due to assaults, 10 percent due to sport related accidents. Therefore a patient presenting with a facial fracture is likely to be a young intoxicated male, who has sustained a RTA or interpersonal violence. The incidence of pre-existing comorbidities is likely to be low.

Upper airway injury may be associated with MFT. Displacement of bony structures, hemorrhage, foreign bodies (teeth, etc.), aspiration or soft tissue swelling itself can obstruct the upper airway in a patient with MFT. A stable patient can deteriorate suddenly necessitating emergency airway management. The mortality is mainly due to airway obstruction and hypoxia.[6]

Blunt trauma can injury the larynx or the cervical trachea in < 1 percent of trauma patients.[7] Penetrating injuries to the upper airway are less common and may be associated with injuries to the neighbouring structures. Often death occurs during attempts to intubate the trachea, when the injury is unsuspected, which leads to complete airway obstruction. In patients who survive the injury the diagnosis is often delayed increasing the incidence of late complications.[8]

RELATED ANATOMY OF UPPER AIRWAY AND MAXILLOFACIAL STRUCTURES[8,9]

The Face and the Base Skull

The face is made up of 6 paired bones— maxilla, nasal, lacrimals, zygomatic, palatines, inferior nasal conchae and the unpaired vomer, and mandible (Fig. 33.1). It is closely associated with the base of the skull, which consists of the ethmoid, frontal, sphenoid, temporal, and occipital bones. The base of the skull is divided into anterior, middle, posterior cranial fossa (Fig. 33.2). Facial bones, the base of the skull and the upper airway are closely related anatomically. The sphenoid and the ethmoid bones are usually, damaged with midfacial fractures and thus, there may be a communication between the upper airway and the intracranial cavity

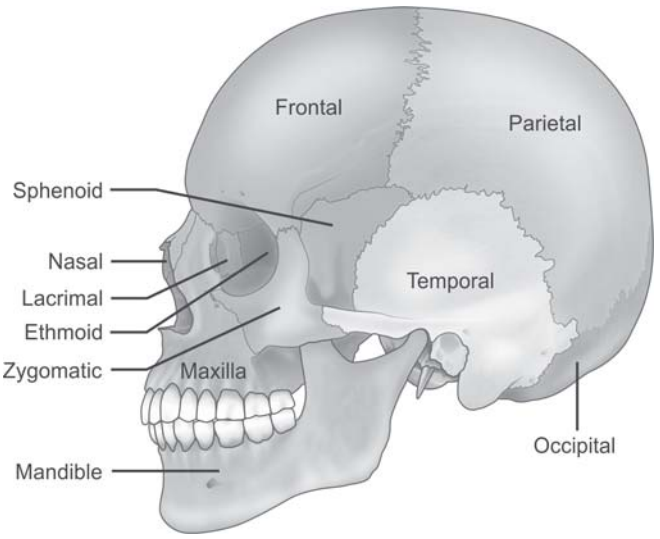


Fig. 33.1: Facial bones

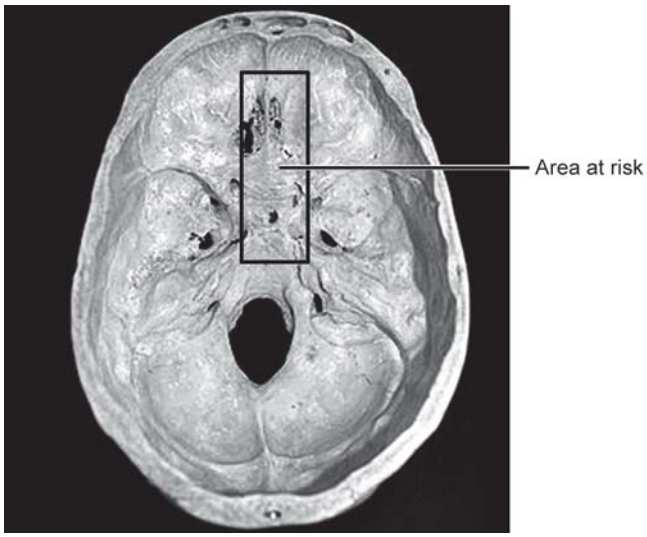


Fig. 33.2: Anatomy of base skull

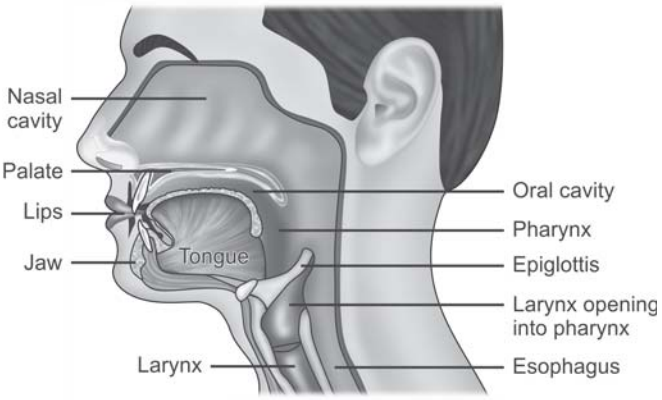


Fig. 33.3: Anatomy of the pharynx

following MFT which have important clinical implications.

The Upper Airway

The airway is divided into the pharynx and the airway proper which is composed of the larynx, trachea and bronchi.

The Pharynx

The soft tissues of the pharynx are supported anteriorly by the maxilla, mandible and teeth; laterally by the mandible and the mastoid processes and posteriorly by the base of the skull and cervical spine (Fig. 33.3). Any damage to these structures following MFT can fatally obstruct the upper airway. Besides the face has a rich blood supply, as a result bleeding following MFT can be profuse, eventually causing airway obstruction either by hematoma formation or by free bleeding into the pharynx with resultant aspiration and respiratory obstruction.

The Airway Proper

The airway proper is fairly free and mobile. It is attached at its superior margin only to the hyoid bone (Fig. 33.4). Intrathoracically it is attached only to the lungs. The bronchi are anchored to some extent by their passage under the great vessels. The trachea in the neck is covered by the thyroid gland, the strap muscles and the cervical fascia, which in turn are surrounded by the structures of the carotid sheath and the sternocleidomastoid muscles. Posteriorly the membranous portion

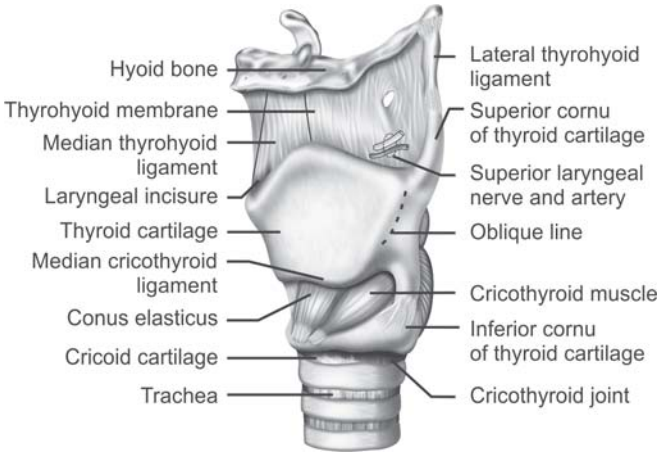


Fig. 33.4: Anatomy of the airway proper

of the trachea is in close approximation to the esophagus. Internally all the structures of the larynx are connected by a submucosal sheath of elastic tissue which is thick in the anterior and posterolateral margin and is termed as the conus elasticus and it attaches the thyroid, cricoid and arytenoid cartilages to one another. The attachment of the larynx to the trachea however consists of a thin elastic membrane called the cricotracheal ligament. The cricotracheal ligament is quite weak and is the most likely point of airway separation. The individual tracheal cartilages are connected by fibrous tissue and smooth muscles and the entire trachea is encased internally and externally by layers of elastic tissue. The superior laryngeal nerve enters the larynx through the posterior portion of the thyrohyoid membrane where it is relatively protected by soft tissue. The recurrent laryngeal nerve however, passes directly between the

cricoid and thyroid cartilages and is frequently damaged when these structures are injured.

MECHANISMS OF INJURY TO THE MAXILLOFACIAL STRUCTURES AND UPPER AIRWAY

Blunt Injury

MFT due to blunt injuries are more common. They are usually associated with RTAs, interpersonal trauma, sports-related trauma, occupational injuries, and falls. The force causing the injury is transmitted through the stronger portions of the facial skeleton and fractures tend to occur at weaker portions. The amount of force required to fracture various facial bones may be classified as high impact [greater than 50 times the force of gravity (G)] or low impact (less than 50 G) The midfacial bones can be easily fractured by low impact forces while the fontal bone and mandible are stronger bones and require high impact forces to be damaged (Fig. 33.5).[9]

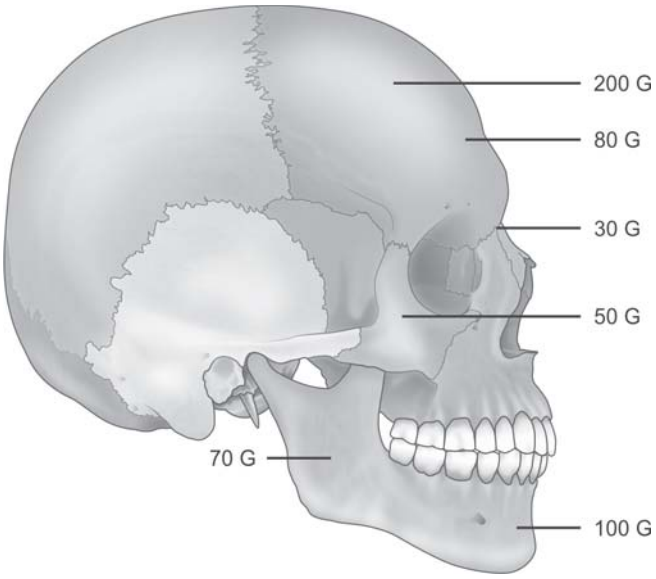


Fig. 33.5: Forces that fracture the facial bones

Blunt injuries to the upper airway most commonly occur due to a direct blow most commonly seen in the "Padded Dash Board Syndrome". Where the head and neck of a seat belt restrained victim on impact continue in the forward motion and the neck makes contact with the dashboard.[10] Severe flexion/extension injuries can also injure the upper airway causing laryngotracheal separation. They are usually associated with esophageal and cervical spine injury. Patients survive as the airway remains patent as long as spontaneous negative pressure ventilation is maintained. Airway obstruction may ensue during intubation techniques.

Blunt injury to the larynx commonly fractures the thyroid cartilage which can distort and also obstruct the airway making intubation difficult. The mortality is around 11 percent.

Cricoid injuries are less frequent. They can be associated with injury to the recurrent laryngeal nerve which can paralyse the vocal cords. Cricoid pressure can cause complete airway obstruction in an unsuspected cricoid injury. Mortality from cricoid injury is 43 percent.[11,12.]

Penetrating Injury[9,13]

Penetrating maxillofacial and upper airway injuries occur due to gunshot wounds, stabbing and explosions. Penetrating injuries causing MFT commonly fracture the mandible followed by the maxilla and zygoma, orbit and nasal bones. They are usually associated with severe bleeding, hematoma formation and airway obstruction. Tissue, teeth or bone fragments can also obstruct the airway. Most of these patients require intubation or a surgical airway to maintain patent airway or prevent aspiration.

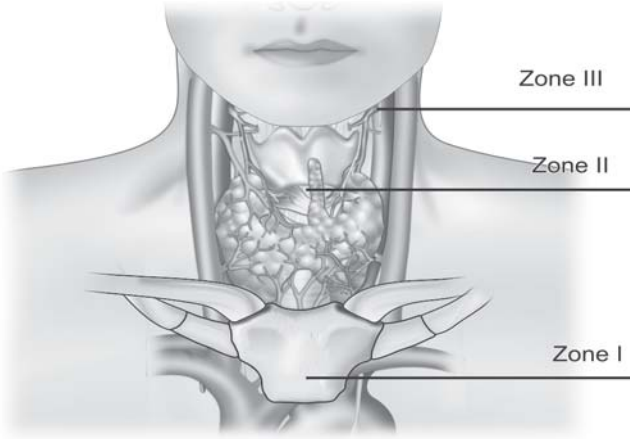


Fig. 33.6: Zones in the neck

Penetrating injury to the neck can damage the airway along with damage to other vascular structures and esophagus. The injuries are described in terms of their location in three anatomic zones (Fig. 33.6).

Zone I: It extends from the cephalad border of the clavicle to the cricoid cartilages. Around 3-7 percent of the penetrating neck injuries occur in this region. They are associated with great vessel or pulmonary injuries which can be life-threatening. Most of these patients require emergency airway procedures.

Zone II: It extends from the cricoid cartilages to the angle of the mandible and is the most common location of neck wounds and amounts to 82 percent of injuries. Airway compromise can occur due to laryngeal injury, an expanding hematoma or subcutaneous emphysema. One third of these patients require an emergency airway.

Zone III: It extends from the angle of the mandible to the base of the skull and is most often associated with vascular and pharyngeal injuries. They amount to 15 percent of the injuries. They are less likely to require emergency airway procedures or explorations.

MAXILLOFACIAL FRACTURES

Mandibular Fractures[7,8,14]

The mandible is commonly fractured in a MFT. The other commonly fractured bones are the nasal bone and the zygoma. The mandible is flexible due to its mobility around the temporomandibular joint (TMJ). The TMJ also absorbs some forces that cause trauma. However the mandible though flexible, forms a semi rigid ring and along with the TM joint. The stiffness of the ring increases the chances that it breaks in more than one place. Therefore like the pelvis, fractures often occur in two places. It gets fractured at the condyles, the angle or the symphysis as the cortex is thinnest here. 34 percent of the mandibular fractures are single. A bilateral fracture of the body of the mandible is known as the "Andy Gump" fracture. (Andy Gump was a chinless cartoon character developed by Sidney Smith, first appearing in 1917.) This fracture can be dangerous as in the supine position, the fractured segment, along with the attached muscles of the tongue, falls backwards, causing airway obstruction at the oropharynx. Grasping the fracture segment and pulling it forward easily relieves the obstruction. Positioning the patient in the lateral position will also enable gravity to help prevent the obstruction. However care in lateral positioning should be taken in patients at risk of cervical spine injury. Mask ventilation may be difficult with this injury, but intubation is not generally difficult.

Hematoma at the floor of the mouth may cause airway obstruction by displacing the tongue upwards and backwards against the palate. An artificial airway will relieve this obstruction. Any Injury to TM joint will limit the mouth opening making intubation difficult.

Le Fort Fractures[9] (Fig. 33.7)

Midface fractures are commonly classified according to the three Le Fort types.

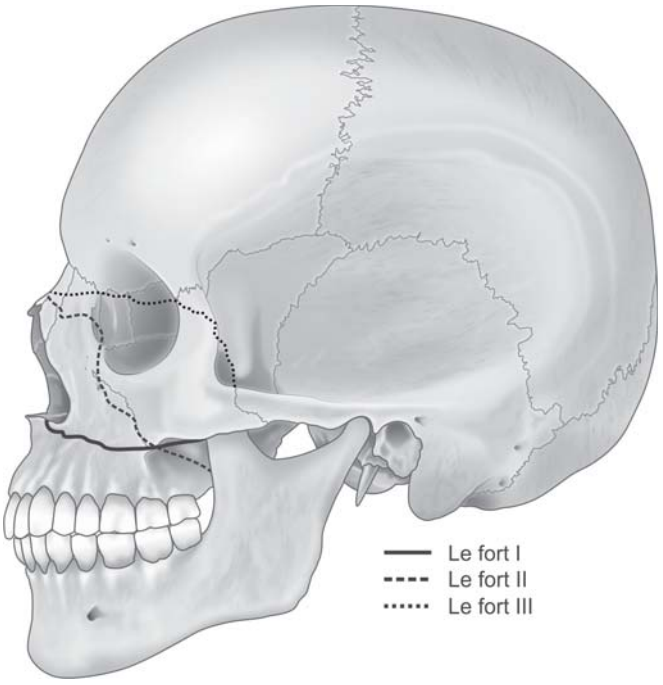


Fig. 33.7: Le Fort fractures

Le Fort I

It is a horizontal fracture of the maxilla that separates the hard palate from the rest of the maxilla. The fracture segment appears like a complete upper denture. It has minimal impact on the airway.

Le Fort II

It is a pyramidal fracture that starts at the nasal bone, extends through the lacrimal bone, and through the zygomaticomaxillary suture, posteriorly through the maxilla and below the zygoma into the upper pterygoid plates. It also has minimal impact on the airway but can be associated with fracture of the skull base. Therefore, any nasal instrumentation in the form of an airway, endotracheal tube or a nasogastric tube is contraindicated until base skull fracture is ruled out.

Le Fort III

This fracture separates facial bones from cranium, causing the face to appear long and flat (i.e. dish face). This fracture is also known as craniofacial disjunction. Like the Le Fort II fractures, this fracture also starts at the nasal bridge, but then extends posteriorly through the ethmoid bones and laterally through the orbits below the optic foramen, through the pterygomaxillary suture into the sphenopalatine fossa. These fractures are

associated with loss of airway and fracture base of skull is common. Bleeding from the midface fractures can be profuse. Manual repositioning of the fracture, nasal packing with gauze or inflated Foley catheter may tamponade bleeding till definitive treatment can be given. A maxillofacial fracture that extends into the frontal bone is referred to as the Le Fort IV fracture with clinical condition same to Le Fort III.[14]

Other Facial Fractures[9]

Simple nasal fractures are the most common of all facial fractures. They are usually seen in direct trauma. Fractures of the nasoethmoid (bones of the nose, orbit, maxilla, and skull) are prone to CSF rhinorrhea due to dural tears. These fractures are frequently reduced openly via a coronal scalp flap.

Zygomatic arch fractures occur in two to three places along the arch. The fracture segment may impinge on the temporalis muscle causing trismus. Zygomatic fractures may extend into the orbital floor. Approximately 60 percent to 70 percent of all facial fractures will involve the orbit and eye. Direct blows can cause a blow out fracture of the orbit. The impact of the injury causes increased pressure within the orbit leading to a fracture at its weakest point—the floor. When there is an intraocular injury, issues of increase in intraocular pressure during intubation have to be taken care of. Surgery on the eye can also stimulate an oculocardiac reflex.

ASSOCIATED INJURIES AND THEIR IMPLICATIONS

Fracture Base Skull[9]

Two to four percent of patients with facial fractures have an associated fracture of the base of skull. The positive clinical signs for skull base fractures are "Battle's sign" (post auricular ecchymosis), "Raccoon eyes" (unilateral or bilateral periorbital hematoma) and bloody otorrhea.[16]

Diagnosis of a base of skull fracture is essential as it has important clinical implications, due to communication between the airway and the intracranial cavity. An associated dural tear increases the risk of meningitis due to easy access to nasopharyngeal bacteria into the intracranial cavity.

The roof of the nose is formed anteriorly by the nasal bones and nasal spine of the frontal bone, centrally by the cribriform plate of the ethmoid bone and posteriorly by the body of the sphenoid. A nasotracheal tube, a nasogastric tube or an nosopharyngeal airway can pass into the cranial cavity through the nose when there is fracture of these midline structures in the base of the skull.[16,17] Besides there is a risk of introducing infection intracranially. There is a high incidence of a nasogastric tube entering the cranial cavity and is attributed to their small diameter and lack of control over direction during insertion. ATLS recommends the oral route of gastric intubation, if needed in patients with facial trauma.

Head and Cervical Spine Injuries[8,9]

Head injury is common along with MFT. It is important to look for pneumocephalus. It can occur in paranasal sinus fractures, fractures of the base of the skull or of the mastoid air cells. Expansion of pneumocephalus can ensue with positive pressure mask ventilation which may cause air to enter the cranial vault under pressure, causing a tension pneumocephalus. Low flow oxygen used during fiberoptic intubation may also cause gas to enter the cranial vault under pressure. Tension pneumocephalus may also occur if nitrous oxide is used during anesthesia.

Cervical spine injury is also common with MFT.[18] They occur in high velocity injuries (RTA) and have associated head injury too. The diagnosis of cervical spine injury can be missed or delayed in 25 percent of cases especially in those who have an altered level of consciousness. About 5-10 percent of cases do not have any neurological deficit but may deteriorate while undergoing treatment. However prevention of hypoxia and airway management remains the high priority in managing trauma patients. Airway management should be done with minimal movement of the cervical spine. Unfortunately the fiberoptic bronchoscope has minimal use during emergency airway management. Therefore airway management is best done by stabilising the cervical spine by manual in line axial traction (MIAT).

ACUTE MANAGEMENT[8,9,19]

Airway patency is the priority in acute management of maxillofacial and upper airway injuries. Loss of airway kills more quickly than does the loss of the ability to breathe or any circulatory problems. Therefore an alert clinician anticipates a respiratory emergency, does a prophylactic intubation or tracheotomy, to avoid complete respiratory obstruction and a chaotic crash intubation or a surgical airway. Once the problems of airway management have been solved, management of other life-threatening injuries will follow.

Thus, treatment priorities are to:

- Clear and secure the airway, provide oxygenation
- Control hemorrhage, treat hypovolaemia, and
- Evaluate for associated life-threatening injuries.

The severity of facial injuries may distract the team from the diagnosis and appropriate management of other associated injuries. Therefore, following a primary and secondary survey approach, as guided by the trauma ATLS, the facial, neck and other injuries can be dealt with appropriately.

The Airway in MFT

Hutchison et al[20] described six specific situations with maxillofacial trauma, which may make the airway management difficult:

1. Posterior inferior displacement of a fractured maxilla parallel to the inclined plane of the skull base may block the nasopharyngeal airway.
2. A bilateral fracture of the anterior mandible (Andy Gump fracture) may cause the fractured symphysis to slide posteriorly along with the tongue. In the supine patient, the base of the tongue may drop back, thus blocking the oropharynx.
3. Fractured or exfoliated teeth, bone fragments, vomitus and blood as well as foreign bodies – dentures, debris, shrapnel, etc. may block the airway anywhere along the upper aerodigestive tract.
4. Hemorrhage, either from distinct vessels in open wounds or severe nasal bleeding from complex blood supply of the nose, may also contribute to airway obstruction.
5. Soft tissue swelling and edema resulting from trauma to the head and neck may cause delayed airway compromise.
6. Trauma to the larynx and trachea may cause swelling and displacement of structures, such as the epiglottis, arytenoids and vocal cords thereby increasing the risk of cervical airway obstruction.

Besides the above situations, the maxillofacial trauma patient has the following problems that can create problems in airway management:

- A problem of difficult mask holding and ventilation and difficult intubation. The trauma usually disrupts the normal anatomy and causes edema and bleeding in the oral cavity
- Furthermore, an injured airway may prevent efficient air transfer from the mask to the lungs
- There is a difficulty in visualizing the vocal cords. The oral cavity, pharynx and larynx may be filled with blood, secretions, debris, soft tissue and bone fractures, all of which preclude good visualization of the vocal cords
- Associated C-spine injury and MIAT may degrade the laryngoscopic view
- The maxillofacial trauma patient, is considered to have a "full stomach", since there was no time for stomach emptying prior to intubation. In addition, this patient often bleeds from the upper aerodigestive tract, blood is swallowed and accumulates in the stomach and the risk of regurgitation and aspiration is high. Studies have shown that cricoid pressure applied to prevent aspiration may significantly worsen the laryngeal view, making endotracheal intubation even more difficult[21]
- In emergency situations, the medical personnel are often less experienced. This is the "inverse care law", meaning that the care for those who are most critically ill is provided by those who are not- yet the most expert. And in an emergency both decision-taking and performance are impaired.

Thus, management of airway in such circumstances may be challenging.

Airway Evaluation and Management[14,19,22]

Airway evaluation should be thorough and quick. The airway must be assessed in the early triage, to exclude airway obstruction, rupture or bleeding. Determine level of consciousness, presence of a full stomach and cervical, skull or other associated injuries. The airway should be observed regularly in the acute phase of the injury, for increasing edema, swelling and hematoma may obstruct a previously patent airway.

Active external bleeding, stridor, voice changes, dysphagia, dyspnea, hemoptysis or surgical emphysema, air bubbling through wound, retropharyngeal air on plain films following blunt injury to the neck, may indicate serious injury to the larynx, pharyngeal airspaces, or extra-thoracic trachea.

Airway Management in MFT

Simple measures to clear the airway by suction, lateral posture (provided cervical spine injury is excluded) and insertion of an oropharyngeal airway will suffice in many cases. An oropharyngeal airway is best avoided in the conscious patient, as it may stimulate vomiting. Nasopharyngeal airway should be avoided in all injuries to the middle third of the face or with basal skull fractures. Supraglotic ventilatory device, such as the LMA, may cause stomach inflation and may be displaced when the patient is moved and managed. Thus, it is not suitable. The Combitube (esophageal-tracheal twin-lumen airway device) is also inserted blindly and can increase the risk of causing false passage and further damage to the airway.

Conscious patients are usually able to control their own airway; sedation in these patients may be catastrophic as the airway may suddenly be lost. A spontaneously breathing patient with a an unobstructed

airway is best observed till one reaches the hospital, preferably taken to the operating room and the airway is managed under the best conditions and by the most experienced medical personnel. Failed attempts at endotracheal intubation by non-qualified persons could be disastrous and could worsen the patient's condition. The American Society of Anesthesiologists (ASA) Practice Guidelines for management of the difficult airway recommend that spontaneous breathing should be preserved in patients with anticipated difficult endotracheal intubation. Emergencies are not good opportunities for trying out novel techniques. Stick to simple techniques that you know. It may be possible to open the airway simply by applying a jaw thrust or by applying traction to the mandible.

Immediate orotracheal intubation by a skilled person under direct vision is indicated in cases of:

- Severe obstruction
- Respiratory depression
- When general anesthesia is required to manage concomitant injuries.

The Bullard laryngoscope is a rigid fiberoptic laryngoscope. It can be used to visualize of the glottic opening even when there is an inability to align the oral, pharyngeal and laryngeal axes. This minimizes head manipulation and positioning. Conversely severe MFT especially with gunshot injuries may make intubation very easy, as the tissue obstructing the larynx has been traumatically removed. Therefore, a direct digital intubation may be possible in these circumstances.[14]

If the upper airway is closed or obliterated and intubation is likely to be difficult and time-consuming a surgical airway is recommended. An emergency cricothyroidotomy or laryngeal jet ventilation may be life-saving. Both can be difficult in a struggling patient. An emergency tracheotomy by an unskilled person may be futile and can be dangerous.

Nasotracheal intubation should not be attempted when midfacial injuries are present and is absolutely contraindicated when basal skull fractures are suspected. The use of a fiberoptic laryngoscope may be difficult because blood may obscure vision. Furthermore, suctioning through it may be inadequate to remove secretions, blood and foreign material. Blind intubation techniques are contraindicated in the acute phase of injury, because a disrupted anatomy may be present. If intubation difficulties are anticipated, tracheotomy under local anesthesia should be considered. Formal tracheotomy is best performed as a planned procedure in the operating room under local or general anesthesia provided airway patency and protection can be maintained.

Airway Management in Upper Airway Trauma[8,14,22]

Simple contusions of the anterior neck can be managed by neck stabilization, head-up posture and humidified oxygen therapy by face mask. Swallowing may be painful and may be accompanied by spasm or tracheal aspiration and oral fluids or solids should not be given for 48 hours.

Blunt or penetrating injuries to the larynx require immediate attention to the airway.

Maintain spontaneous ventilation when possible. Use of direct visualization techniques is preferable when possible. In the presence of an experienced and skilled laryngoscopist an awake fiberoptic bronchoscopic intubation is generally the best choice for patients who are cooperative and stable (these are not teaching patients). Surgeons experienced and trained in tracheotomy should be available at bedside during intubation attempts. Intubation attempts should be performed in the operating room when possible. Ensure that the ETT cuff is below the site of injury before positive pressure ventilation is carefully initiated. Avoid cricoesophageal compression when the injury is in proximity to the cricoid ring.

Alternatively an awake tracheotomy with local anesthesia is well tolerated by cooperative, stable patients. Perform a tracheotomy at least one tracheal ring below the level of injury. Do not place a nasal or orogastric tube until the patient is under general anesthesia.

If intubation is expected to be difficult, and the patient is uncooperative or unstable, a surgical airway may be the best first choice. An emergency tracheotomy or cricothyroidotomy may be preferable to blind or hasty intubation, which may misplace the endotracheal tube or extend the injury. Gaping wounds of the larynx can be temporarily intubated under direct vision. The clinician should always be prepared for a plan in case the airway is lost due to complete separation of the trachea; this may require an immediate upper third sternotomy. After the airway is secured, a detailed examination including direct laryngoscopy should be performed. When the laryngeal skeleton is disrupted, surgical exploration and repair is indicated.

Anesthetic Considerations

Adequate preoxygenation and ensuring spontaneous ventilation is a safe method during induction of anesthesia in a patient with airway injuries. This can be established with the use of a potent volatile agent such as sevoflurane. In uncooperative patients judicious use of propofol can be considered. Use of neuromuscular

blockers is avoided till the airway is properly secured beyond the point of injury. Positive pressure mask ventilation in these circumstances may be dangerous as it may worsen subcutaneous emphysema.

Control of Hemorrhage

Hemorrhage from the midface or base of skull may occasionally be massive, and in severe cases, difficult to successfully control. Provided the airway is secured, the use of topical vasoconstrictors, nasopharyngeal packs or a Foley balloon catheter inflated in the nasopharynx, may control or reduce blood loss. If bleeding persists, coagulation studies should be performed and appropriate replacement therapy is given. Operative reduction of fractures and direct ligation of bleeding vessels may be attempted when simple measures fail to control bleeding. When these measures are unsuccessful, more radical measures, including ligation of the external carotid artery or intra-arterial embolization performed under angiographic control, should be considered.

Clinical Evaluation of Injuries[9]

A history of how the injury occurred should be taken from the patient, bystanders, police or paramedics. Physical examination includes inspection for facial deformity or asymmetry, malocclusion of dentition, palpation of facial bones (including orbital margins), instability and movement of facial fragments, motor and sensory function, visual disturbances (i.e. diplopia, limitation of eye movements and loss of vision) and presence of cerebrospinal fluid (CSF) rhinorrhea.

Specific Investigations

Most facial fractures can be easily diagnosed with a minimum of X-ray studies. Useful studies include stereo Water's view, stereo Caldwell's view, postero-anterior, lateral oblique and Panorex views. Two-dimensional and 3-dimensional CT provide additional information about specific patterns of fracture, and may facilitate surgical care. CT may also be useful when laryngeal injury is suspected.

ELECTIVE MANAGEMENT[9,23]

After life-threatening conditions have been tackled elective reduction and fixation of maxillofacial fractures can be planned in 10-15 days. Early surgery improves the aesthetic results and patient comfort. In some cases, particularly orbital injuries when ocular function is at risk, early surgery is mandatory.

Gross facial swelling may delay surgery. Facial swelling can be reduced by, debridement of open wounds, removal of foreign bodies, closure of facial lacerations. Non-definitive stabilization measures, use of ice packs and head-up nursing of the patient also help to reduce venous pressure and encourage fluid resorption. Prophylactic antibiotics should be used for patients with CSF rhinorrhea, compound wounds and when operative fixation of fractures is performed.

The goals of surgery in maxillofacial fractures include:

- Restoration of functional occlusion
- Stabilization of the major facial skeletal supports
- Restoration of the normal facial contours.

This involves aligning of the upper and lower teeth followed by interdental wiring to maintain proper occlusion. Once the occlusion has been established, open reduction and internal fixation of the fracture can occur.

The anesthetic management of a patient with maxillofacial trauma can be a challenge. They are long surgical procedures and involve significant blood loss. Besides, the airway is shared by the surgeon and the anesthesiologist. Therefore detailed discussion with the surgeon regarding the severity of the injury, duration and extent of surgery, method of securing the airway, route of intubation, type and size of the tracheal tube, is essential as it helps in planning anesthesia. Airway has to be protected to prevent any aspiration of blood (throat pack) as surgery involves intraoral manipulations. The throat pack has to be removed without fail at the end of the procedure.

An oral endotracheal tube may not always be acceptable. Alternative airways are therefore required for definitive surgery. Options available include:

Nasotracheal Intubation (Fig. 33.8)

Nasal intubations can be carried out with the patient awake, anesthetised or fibreoptically depending on the condition of the patient, the difficulty in intubation and expertise of the anesthesiologist. In those cases where nasal intubation is considered, base of skull fracture should be ruled out by a CT scan. In patients with a fractured base of skull this may cause nasopharyngeal bacteria to pass through the fracture into the skull, increasing the risk of meningitis. Lateral skull base fractures do not pose a hazard.

Tracheotomy

Tracheotomy is especially suitable for patients severe head injuries which may require longer term mechanical ventilation in intensive care or as an aid to weaning from such ventilation.

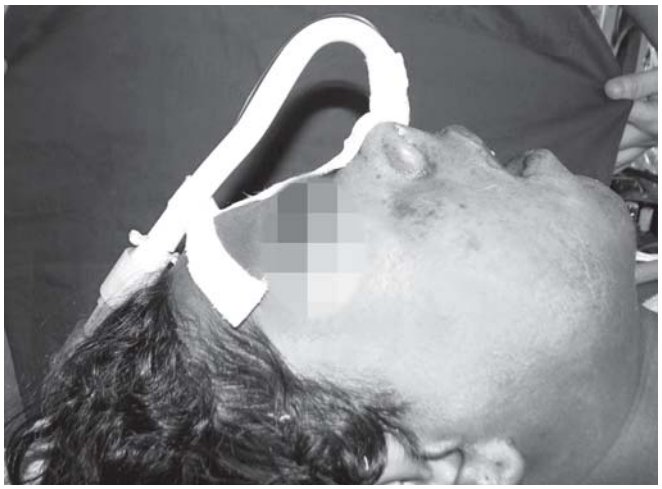


Fig. 33.8: Nasotracheal intubation

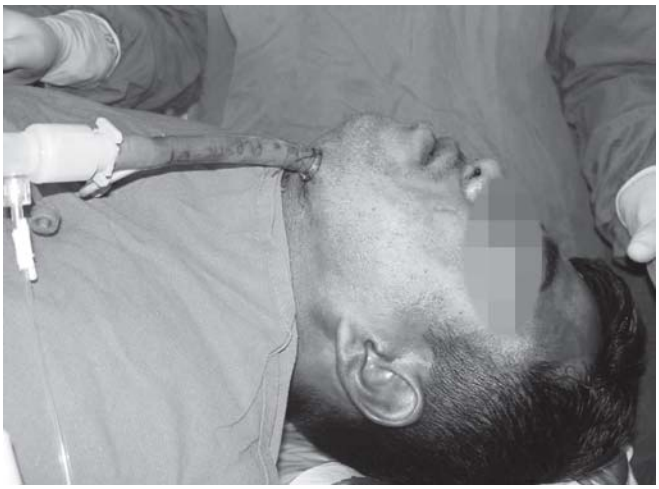


Fig. 33.9: Submental intubation

Submental Intubation (Fig. 33.9)

Submental Intubation has been described as an alternative to tracheotomy in some patients with maxillofacial trauma. We do it in our center. The patient is intubated orally with a flexible endotracheal tube. A passage is created through the submental portion of the floor of the mouth using blunt dissection. Care needs to be taken to avoid the submandibular duct and the lingual nerve damage. The proximal end of the endotracheal tube (ETT) is pulled through the incision after removing the connector. The connector is reconnected, and the function of the ETT is confirmed. The ETT is then sutured in place giving a reliable and secure airway which is out of the surgical field and also allows for occlusion to be achieved. There is a possibility of endobronchial intubation during the positioning and surgery. The ETT can be kept post operatively till the patient is fully awake and the patient can be extubated and the incision sutured.

Retromolar Intubation[24]

Retromolar positioning of the tracheal tube in the retromolar trigone during intermaxillary fixation provides an optimal intraoperative control of dental occlusion. The tube is fixed at the angle of the mouth. At the end of the procedure, extubation can be achieved from the retromolar space, when the patient is awake.

Postoperative Care

Patients require a high dependency unit for postoperative care. Endotracheal tube may be retained postoperatively till the patient is fully awake. If interdental wiring is in place, wire cutters must always be kept next to the patient for emergency (vomiting, airway obstruction, bleeding) and the staff must be taught its use. The head end is elevated to improve venous drainage and limit soft tissue swelling. A tongue stitch may be a safety measure used to prevent airway obstruction.

REFERENCES

1. Park K. Epidemiology of Chronic Non Communicable Diseases. In Park K, (Ed). Textbook of Preventive and Social Medicine, 17th edn. Jabalpur: Banarsidas Bharot; 2005; pp. 303-7.
2. Ugboko VI, Odusanya SA, Fagade OO. Maxillofacial fractures in a semi-urban Nigerian teaching hospital: A review of 442 cases. Int J Oral Maxillofacial Surg 1998; 27: 286-9.
3. BR Chandra Shekar, CVK Reddy. Indian Journal of Dental Research 2008;19(4):304-8.
4. Shapiro AJ, Johnson RM, Miller SF, McCarthy MC. Facial fractures in a level I trauma centre: the importance of protective devices and alcohol abuse. Injury 2001;32:353-6.
5. Iida S, Kogo M, Sugiura T, et al. Retrospective analysis of 1502 patients with facial fractures. Int J Oral Maxillofac Surg 2001;30(4):286-90.
6. Dolin J, Scalea T, Mannor L, et al. The management of gunshot wounds to the face. Trauma 1992;33:508.
7. Gussack GS, Jurkovich GJ, Luterman A. Laryngotracheal Trauma: protocol approach to rare injury. Laryngoscope 1986;96:668.
8. Cicala RS. The traumatised airway. In: Benumof JL, (Ed). Benumof's Airway Management Principles and Practise. Philadelphia, PA: Mosby 1996;736-59.
9. Moloney JT. Maxillofacial Injuries. In: Wilson WC, Grande CM, Hoyt DB, (Eds). Trauma- Emergency Resuscitation, Perioperative Anesthesia, Surgical Management. Volume I. New York: Informa Healthcare 2007;417-32.

10. Butler RM, Moser FH. The Padded Dash Syndrome: Blunt Trauma to the Larynx and Trachea. Laryngoscope 1968; 78:1172.
11. Mathison DJ, Grillo. Laryngotracheal Trauma. Ann of Thoracic Surgery 1987;43:254.
12. Cicala RS, Kudsk K, Nguyen H. Airway Injuries in Multiple Trauma Patients: A review of 48 cases. Clin J Anaesth 1991;2:91.
13. Roon AJ, Christianson N. Evaluation and treatment of Penetrating cervical injuries. J Trauma 1979;9:397.
14. Skerman KH. Anesthetic Management of Craniofacial Trauma and Trauma to the Airway. Middle East Journal of Emergency Medicine 2002;2:2.
15. Flores P, De Almeida CS; Casulari LA. Positive predictive values of selected clinical signs associated with skull base fractures. J Neurosurg Sci 2000;44(2):77-82.
16. Marlow TJ, Goltra DD, Schabel SI. Intracranial placement of a Nasotracheal Tube after Facial Fracture: A Rare Complication. J Emerg Med 1997;15:187-91.
17. Spurrier EJ, Johnston AMD. Use of Nasogastric Tubes in Trauma Patients (M) A Review. JR Army Med Corps 2008; 154(1):10-3.
18. Davidson John SD, Birdsell Dale C. Cervical Spine Injury in Patients with Facial Skeletal Trauma. Journal of Trauma 1989;29(9):1276-78.
19. Krausz A, Abu el –Naaz, Michal B. MaxilloFacial Trauma Patient: Coping With The difficult Airway. World Journal of Emergency Surgery 2009, 4,doi:10. 1186/1749-7922-4-21. http://www.wjes.org/content/4/1/21.
20. Hutchison I, Lawler M, Skimmer D. ABC of Major Trauma: Major Maxillofacial injuries BMJ 1990;301:595-99.
21. Haslam N, Parker L, Duggan JE. Effect of cricoid pressure on the view at laryngoscopy. Anesthesia 2005;60:41-7.
22. Hurford WE, Peralta R. Management of Tracheal Trauma. Can J Anaesth 2003; 50(6):R1-R6.
23. Sood J. Maxillofacial and Upper Airway Injuries: Anesthetic Impact. Indian J Anesth 2008;52(5):688-98.
24. Dutta A, Kumar V, Saha SS, Sood J, et al. Retromolar tracheal tube positioning for patients undergoing faciomaxillary surgery. Can J Anesth 2005;52:341.

Section IX

Miscellaneous

34 Anesthesia for Emergency Microvascular Surgeries

Shakuntala N Basantwani, Sohini S Shah

KEY POINTS

- Microvascular surgeries are time consuming and lengthy; and the patient needs to be rendered immobile for long periods, sometimes in excess of 8 to 12 hours.
- Anesthetic management of patients undergoing microvascular surgeries requires a sound knowledge of circulatory physiology and pharmacology.
- It is important to maintain adequate blood pressure, perfusion pressure and cardiac output of the patient. The degree of any induced vasodilatation should be balanced against the effect on systemic arterial pressure. Maintenance of central venous pressure at about 2 to 3 mm Hg above the baseline, helps maintaining a high cardiac output.
- Normovolemic/Hypervolemic hemodilution to a hematocrit of 35 percent, increases cardiac output and improves the flow in microcirculation.
- Heat loss should be minimized in the intraoperative as well as postoperative period and the transplant itself kept warm. Thus, hypothermia should be strictly avoided.
- Hypocapnia/Hypercapnia should be avoided.
- Analgesia should be adequate in the intraoperative as well as in the postoperative period. Use of continuous epidural/ brachial plexus catheters for postoperative analgesia and sympatholysis is beneficial.
- Measures taken in the intraoperative period to produce optimum circulatory conditions should be continued in the postoperative period as well and patient monitored in a high dependency/intensive care unit.
- Dextran-40 infusion is started postoperatively and continued for 3 to 4 days, to prevent thrombosis at the anastomotic site.
- Thus, in general it requires maintenance of adequate tissue oxygenation by optimization of blood flow and oxygen content.

INTRODUCTION

Our institute sees a lot of trauma and accident victims and emergency replantations and revascularizations are quite commonly done procedures. Every anesthetist needs to be aware of both the theoretical and practical aspects while giving anesthesia to such patients.

DEFINITION[1]

Microvasclar surgery: These include surgeries where, arteries and veins of less than 1.5 mm in diameter may be anastomosed with the aid of magnification from loupes or an operating microscope.

Replantation: If a part is completely amputated, i.e. cut off without any attaching structures whatsoever, it will be *replanted.*

Revascularization: If a part has been deprived of its main blood supply, but there are connecting structures remaining such as tendons or nerve, the part has been incompletely amputated and will be *revascularized.*

Emergency microvascular surgeries would include:

1. Replantation/Revascularization of amputated limb/ digits.
2. Emergency re-exploration of an operated case of free flap surgery.
3. Emergency free flap cover for tissue loss due to trauma.

Revascularization/Replantation procedure is quite common, especially in cases of upper limb, e.g. assault by sharp instruments like a sword or for a laborer working with machinery or even with other type of accidents. Usually, clean cut amputations or guillotine

amputations have a better viability and good ultimate function. Avulsion and crushing injuries produce a zone of injury proximally and distally to the amputation site that requires more debridement, have more chances of infection and have a poor prognosis in terms of survival.[1] Certain other structures may also be replanted for example, scalp which maybe avulsed by long hair being caught in a rotating machinery. Similarly, the lip, nose or ear which may have been traumatically amputated *en bloc*, may be replanted.

Emergency re-exploration could be required for a patient who has already undergone a free flap surgery for example, for resection of a tumor followed by a free flap for reconstruction of the defect. Emergency re-exploration would be required for flap failure, the causes of which are classified as arterial, venous or resulting from edema. The arterial anastomosis may be inadequate, in spasm or thrombosed. Similarly venous anastomosis may be defective, in spasm or compressed (by tight dressings or poor positioning).[2] Edema reduces the flow to the flap and may be a result of excessive crystalloid administration, extreme hemodilution, trauma from excessive handling or prolonged ischemia time. Patients who have undergone such major and lengthy procedures are usually in the intensive care unit for observation. They would have had considerable blood loss in their primary surgery and, if they need to be taken up again for re-exploration they would require at least a basic hemogram, serum electrolytes and an arterial blood gas analysis if possible.

Although, emergency microvascular surgery, for emergency free flap cover is not commonly done, it could be required in certain cases where major important or vital structures are exposed and where primary cover with either skin grafting or flap other than free flap is not possible.

Emergency Room Care

When the patient arrives at the hospital with a history of amputation, full attention has to be given initially to other associated major life-threatening injuries, as for any other major trauma case. Since the replantation/revascularization procedures are very lengthy, other major problems must be diagnosed and cared for before the beginning of microvascular surgery. The higher the level of amputation greater the chance that significant injuries may have occurred to other systems. Major limb amputations could have also resulted in considerable blood loss. Self-inflicted amputating injuries may require urgent psychiatric consultation.[1]

All blood investigations needed for providing anesthesia should be sent immediately, blood should be sent for grouping and cross-matching. X-ray chest postero-anterior view should be done. Intravenous access should be established on the other hand/lower extremity. Patient should be monitored for all the vital parameters. Any bleeding from the amputated area should be controlled by direct compression and not by hemostats, ligations or proximal tourniquets.

Ischemia Time

The warm ischemia time sustained by the amputated part will influence the chance of revascularizing the part and ultimate function. Hypoxia will lead to progressive cellular damage resulting in no reflow to the ischemic tissues and necrosis. The permissible warm ischemia time for more proximal amputations which contains more muscle is 6 hours, whereas for more distal amputations it is 8 to 12 hours. Replantation may be partly successful after a longer warm ischemia time, but necrosis and impaired function will surely follow. Cold ischemia is tolerated longer than warm ischemia. A finger or hemi hand amputation may be maintained in a cold ischemic state for more than 24 hours, whereas a proximal arm or forearm amputation may only be replantable for up to 12 hours after amputation, despite proper cold storage.[1]

Care of the Amputated Part

The amputated part must be cooled from the time it is retrieved. The part should be wrapped in moist saline gauze, placed in a sealed plastic bag and immersed in an iced saline container. The part should not be water logged or frost-bitten. Any bleeding from the amputation site should be controlled by direct compression and not by hemostats, ligations or proximal tourniquets.[1]

Physiological Considerations of Circulatory Control

Management of patients undergoing microvascular surgery, whether it is elective or emergency, requires a sound knowledge about cardiovascular and circulatory physiology. This will help one understand the principles and guidelines of management of these patients.

Poiseuille-Hagan Formula

The interrelation of factors influencing the laminar flow of Newtonian fluids through a rigid, nondistensible cylindrical tube is defined by Poiseuille-Hagan formula,[3]

Blood flow = $(P_A - P_B) \times \pi/8 \times 1/n \times r^4/l$

Where, $P_A - P_B$ = Pressure difference between two ends of a tube

n = Viscosity, r = Radius of the tube, l = Length of the tube

Thus, from the above formula, changes in perfusion pressure, viscosity and cross-sectional area (radius) will all influence the blood flow. From this we can infer that the goals of anesthesia for microvascular surgery are vasodilatation, good perfusion pressure and a low viscosity.

The systemic arterial pressure is the major determinant of the pressure gradient across the transplanted tissue. Also, since blood vessels are distensible, the intraluminal pressure will itself influence the diameter, according to the Law of Laplace. The transmural pressure will decrease if either the intravascular pressure decreases or the extravascular pressure increases (pressure from dressings, hematoma or edema).

As blood flow is related to fourth power of radius, even a small change in the cross-sectional area, produces a large change in the blood flow. In vessels with internal diameter > 1.5 mm, the viscosity is closely related to the hematocrit.[4] This relationship is not linear and as hematocrit increases above 40 percent, the viscosity begins to increase dramatically.

Peripheral and Central Control of Blood Pressure and Vascular Resistance

Blood pressure is maintained by sympathetically mediated increase in peripheral vascular resistance, at the expense of a reduction in cardiac output and oxygen delivery. During surgery, a fall in cardiac output is predominantly due to decreased cardiac preload from hypovolemia (absolute-fluid deficit, blood loss from the injury site or relative from vasodilatation by anesthetic agents). Blood pressure must be maintained by fluids and if required blood replacement to restore the preload and hence the cardiac output.

The reflex response to hypovolemia and a falling venous pressure is mediated by "low pressure" cardiopulmonary baroreceptors. This results in an increase in sympathetic tone and peripheral vascular resistance with arteriolar and venular constriction and can lead to peripheral vasoconstriction. This reflex can be effectively blocked by inhalational agents like isoflurane and intravenous agents like propofol which attenuate these reflexes. Benzodiazepines and opioids have less effect.

The "high pressure" baroreceptor reflexes are activated by sensors located in the arch of aorta and respond to hypotension (from any cause) by increased sympathetic activity, leading to increase in vascular resistance and peripheral vasoconstriction.

Local Control of Blood Flow

The regulation of blood flow and oxygen delivery to tissues is provided by three different portions of the microcirculation, the resistance vessels (arterioles), the exchange vessels (capillaries) and capacitance vessels (venules).

Regulation of blood flow through capillary beds is mainly concerned with the supply of adequate oxygen and nutrient to cells requiring them. At any one time, only small sections of capillary beds are perfused and the precapillary sphincters, which are located at the arterial end of the capillaries are responsible for the control of blood flow within the capillaries.

As metabolism continues to proceed, hypoxia, hypercarbia supervene along with accumulation of metabolic products like H^+ ions, K^+ ions, adenine and lactic acid. These substances cause relaxation of precapillary sphincters to allow blood flow to be re-established. Thus, given an adequate supply of blood to the tissue bed, it can be appropriately distributed by local factors. Other vasoactive hormones like vasopressin, renin, angiotensin, kinins, adrenaline and nor-adrenaline also have a role in microvascular blood flow.

Transplanted vessels in a free flap do not have a sympathetic innervation, but they do respond to local and humoral circulating factors, described above.

Microcirculation

In the microcirculation of the flap itself, the same principles of the Poiseuille–Hagan formula apply. The blood viscosity however plays a more important role. Blood is not a Newtonian fluid, which means its viscosity is not constant and is determined by the flow. In very small blood vessels, rapid laminar flow results in axial streaming of the cells, where the cells line-up with their discoidal surfaces facing each other and pass through in a column and effective viscosity is reduced.

The pressure generating flow is known as shear stress. In the microcirculation it can decrease due to a decrease in blood pressure, increase in venous pressure and an increase in the resistance to flow upstream as a result to vasoconstriction or increase in whole blood viscosity. This decrease is most likely to occur first at the level of postcapillary venule and extend upstream to the capillaries.[3]

If the flow rate decreases below a critical value, axial streaming of the red cells does not occur and the cells are

evenly distributed throughout the vessel. The viscosity increases in proportion to hematocrit. Red cells may come in contact with the vessel wall and as the flow further reduces, they will clump together in rouleaux which are much more rigid than individual cells and greatly increase the resistance to flow.

The tendency to rouleaux formation is also related to the concentration in the plasma of large molecules such as fibrinogen and α_2-macroglobulin. Dextrans with low molecular weight (dextran-40), cause break up of such rouleaux.

Metabolic autoregulation of regional blood flow can only function in presence of an adequate perfusion pressure. A good pulse pressure in the microcirculation ensures an effectively greater period of capillary patency for equivalent values of mean blood pressure. It also improves the circulation of lymph and interstitial fluid and also helps enhance cellular metabolism.

In planning anesthesia, the aim must therefore be to produce a hyperdynamic circulation with a high cardiac output, adequate vasodilatation and a large pulse pressure.[5,6] This will ensure adequate microcirculatory perfusion and minimize the risk of platelet deposition at the sites of anastomosis.[3]

PRACTICAL CONSIDERATIONS IN MANAGEMENT OF PATIENTS

Initial Evaluation and Assessment of the Patient

Patients requiring emergency microvascular surgery should be evaluated thoroughly for any other severe or life-threatening injuries which need immediate attention. In cases of traumatic amputation, the mode of injury and time elapsed since the injury are to be noted, as time elapsed is critical for the survival of the amputated part. Also, when the patient brings the amputated part along, one should ensure whether it is properly stored or not.

Patient needs to be stabilized, vital parameters should be checked. Good intravenous access should be obtained. The required basic investigations should be sent and X-ray chest should be done. Blood should be sent for grouping and cross-matching. The patient may not be adequately fasting hence, all measures to hasten the gastric emptying and to prevent aspiration should be taken into account.

Control of Blood Pressure

An adequate blood pressure with vasodilatation ensures good tissue perfusion by providing rapid regional blood flow, improving patency of the microvasculature and maintaining the fluidity of blood in the microcirculation. One has to balance the benefits of a hyperdynamic circulation against the need to provide good operating conditions and adequate hemostasis.[3]

Deliberate controlled hypotension which is used during dissection and removal of a tumor prior to taking of the flap with help of vasodilators, in routine free flap and other microvascular surgeries may or may not be possible in emergency situations depending on the condition of the patient.

Adequate blood pressure varies from patient to patient. In general the systolic blood pressure should not be less than 90 to 100 mm of Hg in healthy young adults, while in hypertensive and older patients it should be maintained to at least 100 mm of Hg.[3] Certain drugs like sodium nitroprusside, act as arteriolar smooth muscle relaxants and are used for providing both controlled hypotension as well as preventing vascular spasm in routine microvascular surgeries, may not be possible in a patient who has already bleed a lot due to trauma/injury.

Hypovolemia is usually the cause of persistent low blood pressure and should be avoided.

Control of Carbon Dioxide

An increase in carbon dioxide causes depression of myocardium and relaxation of vascular smooth muscle, however, a concomitant increase in sympathetic activity more than compensates for the cardiac effects. However, in anesthetized patients the response is modified depending on the agents used and the degree of myocardial depression and sympathetic stimulation they themselves produce. For example, even with halothane which produces minimal sympathetic activity, the sympathetic response to carbon dioxide is sufficient to overcome the direct myocardial depressant effect.

The overall effects of hypercapnia are increases in cardiac output, heart rate and blood pressure with a reduction in peripheral vascular resistance. Hypocapnia on the other hand, causes an increase in peripheral vascular resistance and a decrease in cardiac output.[6,7] Thus, hypocapnia, which occurs very commonly during controlled ventilation, should be avoided at all costs. An end-tidal CO_2 monitoring is therefore required for all these surgeries and it should be maintained in the range of 30 to 35 mm of Hg.

Temperature Control

Under general anesthesia, heat production and conservation are impaired especially in elderly and infants.[8]

Surface radiation and evaporation are main sources of heat loss during surgery. General anesthetics such as halothane, along with opioids depress the threshold for peripheral vasoconstriction under anesthesia by 2 to 3ºC.[9] Also by promoting peripheral vasodilatation, inhalational anesthetics increase the heat loss by radiation, conduction and evaporation. Neuromuscular blockers also decrease the heat production by impeding the muscle activity. Ventilation with dry gases increase the heat loss, for example, ventilation with dry gases will at 10 L/min removes approximately 40 KJ/hour of heat from the body. Infusion of cold intravenous fluids and blood can also cool the patient. The cold stress of one unit of bank blood at 4 to 8°C is equivalent to that of 1 liter of colloid or crystalloid at 16 to 20°C and it decreases body temperature by 0.25°C.[3]

On awakening from anesthesia, there is a greater sensitivity to thermal change. This accounts for the excessive shivering seen frequently in the postoperative period. Therefore there is an increase in the oxygen demand in postoperative period due to shivering. This might prejudice the flap/graft survival. In a free tissue transfer surgery, the flap is denervated and ischemic, which would actually predispose to vasodilatation, but local decrease in skin temperature can lead to proportionate decrease in blood flow. This reduction in flow is due to various factors, cold causes vasoconstriction, an increase in hematocrit, increased aggregation of red cells and increased whole blood/plasma viscosity.

Thus, above discussion stresses, that the patients temperature should be maintained near normal as possible.[3] All measures should be taken to conserve heat from commencement of anesthesia and should continue during transport, recovery and postoperative period. The most important measure is to maintain the ambient temperature in the operating theater to about 22 to 24°C. The anesthetic gases used should be humidified; a heat moisture exchanger filter should be attached to the circuit. The patient should be wrapped in plastic sheeting which helps maintain the body temperature. Warming blankets, which are very effective in infants and children, make very little contribution to patient warmth in adults.[10] Forced air blankets may prove to be effective in conserving heat. In surgeries where blood loss is expected to be excessive (e.g. in replantations of multiple digits or hand an in-line warmer should be used. It is also advisable to warm all intravenous fluids administered to the patient.

Positioning of the Patient

In view of the long durations of these surgeries and often awkward positions required for surgical access, extra care should be taken during positioning of the patient, right at the beginning of the surgery.

Liberal use of soft padding should be employed. Nerve and muscle damage have been reported after such prolonged surgeries, if pressure points have not been adequately padded. The use of ripple type of water mattress and frequent passive limb movements have been advised to avoid this complication. Pneumatic leggings used intraoperatively should help prevent the occurrence of deep vein thrombosis.

Hematocrit and Hemodilution

The effects of hematocrit on viscosity, flow, tissue oxygen delivery following hemodilution have been reviewed.[11] A reduction in viscosity, reduces afterload and increases cardiac output with little pressure change. Although oxygen content of blood is reduced by fall in hematocrit, oxygen delivery (flow X content) is maintained due to increased cardiac output. Local blood flow also increases thus maintaining tissue oxygen delivery during normovolemic/hypervolemic hemodilution and this is probably maximum at hematocrit of 30 to 35 percent.

In patients with adequate cardiac reserve an increase in cardiac filling pressure to about 2 mm of Hg greater than the control measurement will double the cardiac output and at the same time produce skin and muscle vasodilation.[12]

Normovolemic or hypervolemic hemodilution has been shown clinically and experimentally to improve the chances of survival of tissues in which the circulation is compromised.[13-15]

Either crystalloid or colloid solutions are used for hemodilution. Crystalloid solutions are alone ineffective in producing volume expansion and hemodilution, and in fact may be detrimental to the survival of the flap by the virtue of the edema produced. Colloids hold fluid in the vascular compartment and provide a more stable circulation.[16]

There is always a danger of overloading the circulation especially if left ventricular function is already impaired. Hence central venous pressure (CVP) monitoring is mandatory for such cases. Also a low hematocrit increases myocardial work and therefore care should be taken in patients with poor cardiac function.

Fluid Balance and Blood Loss

The aim of intravenous fluid therapy in microvascular surgery is to maintain intravascular fluid volume for optimal tissue blood flow and oxygen transport to all tissues, including the free transferred tissue. A meticulous fluid balance is necessary in such protracted surgeries.

These patients could have already lost a significant amount of blood from the amputation site or in cases like re-exploration of free flaps, patients could have already have had loss in previous surgery. A fresh sample for hemoglobin and hematocrit should be sent to the laboratory and adequate units should be cross-matched and kept ready. The choice of fluids is important, since free flaps and replants are at increased risk of developing edema, due to lack of lymphatic drainage and a decreased ability to reabsorb excessive interstitial fluid. Therefore, it is suggested that crystalloids be used only for insensible fluid loss, but that synthetic colloids like pentastarch[17] or tetrastarch be used for the replacement of plasma constituents.

Guide to Fluid Management

Crystalloids – 2 to 3 ml/kg/hr, to replace preoperative deficit
– 4 to 8 ml/kg/hr, to replace insensible losses and for maintenance fluid
Colloids – 10 to 15 ml/kg for hemodilution (if planned)
To replace blood loss
Blood – To maintain hematocrit at 30 percent
Dextran – Often given postoperatively as infusion

Control of Pain

Pain and anxiety both increase the sympathetic vasomotor tone and thus it is important from circulatory as well as humanitarian point of view to relieve pain adequately both during and after the surgery.

Regional analgesia (epidural/plexus blocks) used as a lone anesthetic technique or in conjunction with general anesthesia produces good analgesia, reduces the requirement of anesthetic drugs needed during the maintenance period and also produces sympathetic blockade which helps to dilate the blood vessels.[18,19]

Continuous epidural and brachial plexus (supraclavicular or axillary) catheters with infusion of local anesthetic and opioids if required, used intraoperatively and postoperatively are helpful. There are some concerns that the sympathetically denervated transplanted vessels in the free flap would be unable to dilate after lumbar epidural blockade resulting in "steal" effect, reducing the flap blood flow. However, provided any hypotension due to sympathetic blockade is avoided or treated promptly, blood flow to the flap improves as a result of the increased flow through the feeding recipient artery.[20] Other advantages are reduced incidence of vessel spasm, improved diaphragmatic function especially after prolonged surgery, and more rapid postoperative recovery. The muscle relaxation produced helps immobilize the extremity. Good analgesia also reduces the level of circulating catecholamines, avoids vasoconstriction, increases skin temperature and improves circulation. All of these help healing especially in replanted digit or hand. A study demonstrated that axillary brachial plexus block with continuous infusion of 0.75 percent ropivacaine can increase the skin temperature, an index of tissue perfusion, of the reconstructive digits for 24 hours after microvascular surgery of the crushed fingers.[21] In case of low molecular weight heparin for DVT prophylaxis, removal of the catheter has to be timed as per the last dose of heparin and aPTT values.

Vasodilatation

There are a lot of studies supporting use of local/regional and systemic vasodilatation in microvascular surgery, to enhance flap survival.

Local: At local level vasodilatation will be maximal in the flap itself due hypoxia, hypercarbia and accumulation of metabolites. However, following devascularization, feeding vessels are still subjected to systemically induced vasoconstriction.

Surgeons may use topical vasodilators such as papaverine, lidocaine or verapamil to relieve spasm of blood vessels locally.

Intra-arterial guanethidine has been used to produce prolonged (3 days) and localized vasodilatation in experimental studies of flaps in rabbit ear.[22] Applications in humans could include an intravenous regional sympathetic block with guanethidine,[23] when using radial artery forearm flap. Elsewhere it would be difficult to limit the spread of guanethidine.

Systemic: Many drugs used in anesthesia, involving all volatile anesthetic agents have a vasodilatation action, which can be helpful. Isoflurane produces a marked reduction in peripheral vascular resistance with minimal cardiac depression.

Systemic vasodilators like sodium nitroprusside and nitroglycerine cause vasodilatation by action on vascular smooth muscles.[24] These vasodilators might be actually harmful as they could steal the blood away from maximally vasodilated flap. Sodium nitroprusside can cause reflex increase in plasma renin activity and angiotensin II secretion, unless patient is adequately beta blocked. It could also cause vasoconstriction when discontinued in the postoperative period. The use of vasodilators would require invasive blood pressure monitoring and an arterial line cannulation.

Role of Dextran in Microvascular Surgery

- Dextrans are polysaccharides built up of glucose molecules.
- The physico-chemical and biological properties of dextran solutions depend upon average molecular weight, molecular weight distribution, concentration and molecular structure. On an average, complications increase with higher molecular weight, broader molecular weight distribution and more the degree of branching of the molecule.
- Commonly used dextran preparations are dextran-70, average molecular weight of 70,000 daltons and dextran-40 with molecular weight of about 40,000 daltons.
- The colloid osmotic effectivity of dextran is based on the fact that every intravascular gram of dextran binds to 20 to 25 ml of water.
- In humans the dextran plasma concentration after infusion of 0.5 to 1.5 litres is approximately 0.5 to 1.5 gm percent. Smaller dextran molecules are excreted quickly and are completely broken down and excreted by the kidneys at the rate of 70 mg/kg/24 hours.
- Dextran infusion does not impair renal function. The only caution is that when infusing large amounts, one must assure normal conditions of water and electrolyte balance, as they displace interstitial fluid into the vascular compartment.
- The commercial preparations of dextran available for clinical use have no antigenic effect, however its structure is similar to other antigenic polysaccharides. Some polysaccharide reacting antibodies if present in a patient in sufficient quantities, may cross react with dextran and trigger allergic side effects or anaphylaxis may occur.[25]
- Dextran-40 and 70 commonly used clinically do not influence blood group determination in any way.
- Dextran reduces the blood viscosity, reduces platelet adhesiveness and sludging and prevents red blood cell rouleaux formation. This contributes to the antithrombotic effect of dextran. The mechanism of proposed action is formation of negative charge on platelet surface or inactivation of von Willebrand factor.
- Dextran-40 is usually given postoperatively after a microvascular surgery for up to 3 to 4 days to reduce the chances of thrombosis at the anastomotic site. Usually an initial test dose of < 5 ml of 10 percent solution of dextran-40 is given. If there are no signs and symptoms of any allergy in 1 hour, the patient is given 20 to 30 ml of 10 percent dextran-40 as a loading dose followed by a maintenance dose of 15-25 ml/hr for 3 to 5 days.

Anesthesia Management and Selection of Anesthetic Technique

Preoperative Evaluation and Investigations

As emergency microvascular surgeries are usually done for posttraumatic cases, thorough evaluation of the patient should be done to rule out other associated injuries. Patient needs to be stabilized hemodynamically and analgesics needs to be administered to reduce pain induced sympathetic stimulation.

Depending upon the circumstances, minimum basic investigations—Hemoglobin, Blood urea nitrogen, Serum electrolytes, Random blood sugar and if time permits an ECG, X-ray chest—should be done and blood should be cross matched if blood loss is expected. In cases of replantation and revascularization depending upon the time elapsed since injury and the ischemia time (warm/cold ischemia) cases should not be delayed due to want of investigations unless certain specific history or examination demand a specific investigation.

It needs to be kept in mind that the replantation and revascularization procedures are limb saving procedures and not life saving procedures. So, if there is associated life-threatening injury then they need to be addressed first. There have been incidents of unrecognized major injuries in patients referred for microvascular surgeries, which needed to be stabilized first.[26] Also, if the risk of anesthesia and prolonged surgery outweighs the benefits from replantation/revascularization then risk benefit ratio should be informed to the patient after discussing with the surgeons.

The emergency revascularisation procedure will require close attention to preoperative blood loss and repletion.

Monitoring

- *Standard/minimum basic monitoring:* Cardioscope, Pulse-oximeter, Noninvasive blood pressure monitoring and capnometry.
- *Central venous pressure monitoring:* It reflects cardiac filling pressures and can be monitored in intraoperative as well as postoperative period.
- *Core and peripheral temperature monitoring:* It should be used during microvascular surgeries as mentioned before, especially when active warming is instituted. A rectal or nasopharyngeal probe for continuous core temperature monitoring can be used

intraoperatively. Peripheral temperature should also be measured, as a fall in skin temperature can reflect hypovolemia and vasoconstriction. A difference of less than 2°C between core and peripheral temperature indicates a warm, well-filled patient.

- *Urine output:* It is a good indicator of the volume status of the patient. A urine output of 1 to 2 ml/kg/hr should be maintained intraoperatively and postoperatively with appropriate fluid management. It is especially important, in patients who have had crush injuries with impending renal shut down.
- *Invasive blood pressure monitoring:* Whenever indicated, invasive blood pressure monitoring should be used.
- *Neuromuscular junction monitoring:* As it is a prolonged surgery, the requirement of muscle relaxant should be titrated as per TOF response of the patient.

Anesthesia Techniques

Certain surgeries like thumb/digit replantations can be carried out by using regional anesthetic techniques only. A brachial plexus block (supraclavicular/axillary) can be given, followed by axillary catheter placement for continuous use intraoperatively and for postoperative pain relief.[3]

However, in majority of the cases, as these surgeries are quite prolonged and patient may not be comfortable for a very long period under regional anesthesia, general anesthesia will then be required.[5]

A balanced general anesthetic technique with endotracheal intubation and controlled mechanical ventilation with use of muscle relaxants, maintenance by intravenous or inhalational agents and adequate analgesic supplementation with blocks or parenteral analgesic is used. The aim is to reduce the stress of surgery and associated catecholamine secretion which might tend to reduce the peripheral blood flow in certain procedures like replantion or revascularization where the ischemia time is crucial, even if the patient is not adequately fasting, anesthesia has to be given by rapid sequence induction and cricoid pressure, along with measures to hasten gastric emptying.

Maintenance of anesthesia with intravenous agents like propofol or inhalational agents like isoflurane is preferred over halothane as it does not decrease the cardiac output as much as halothane. Epidural anesthetic supplementation has been advocated to prevent vascular spasm for lower limb surgeries and brachial plexus block supplementation for upper limb surgeries.

Following induction and intubation, Ryle's tube and Foley's catheter should be inserted and central venous line should be cannulated. An arterial line should be cannulated if invasive blood pressure monitoring is deemed necessary. An end tidal CO_2 analyser should be attached to the circuit along with use of a HME filter. Temperature probes should be placed in position. All pressure points should be adequately padded with soft padding and ripple-water mattress should be used wherever possible. Limbs should be well supported to avoid neurological damage or vascular compression. Eyes of the patient should be taped and moisture retaining eye drops should be used to prevent corneal drying and abrasion. Also measures to avoid postoperative deep vein thrombosis should be taken as per discussion with the surgeon. Body temperature should be maintained with warming blankets, use of humidified anesthetic gases, warm intravenous fluids and maintaining ambient temperature of the operating theater at 22 to 24°C.

The surgeons may use a pneumatic tourniquet during the surgery, the timing of inflation and deflation of the tourniquet should be noted by the anesthetist. There could be repeated inflation and deflation of tourniquet and adequate rest time must be allowed for the limb before reinflation of tourniquet. Following deflation of the tourniquet there could be hypotension due to release of accumulated metabolites like lactic acid, K^+ ions, etc. and hence patient must be given adequate fluids before the deflation of the tourniquet. Certain drugs like antibiotics and muscle relaxants should be given before inflation of the tourniquet.

After the revascularization procedure is complete, a lot of metabolic substances like lactic acid, H^+ ions, K^+ ions released into the systemic circulation which had accumulated in the ischemic area.

These metabolites can cause acid-base imbalance and hypotension. Hence an arterial blood gas analysis during that period would be helpful.

After surgery is completed, the neuromuscular blockade is reversed and patient is extubated as per the spontaneous regular breathing attempts, oxygenation status as seen by the ABG, Train of Four (TOF) response and temperature of the patient. Unless there are other associated medical conditions, risk factors or complications these patients can be usually extubated, but they need monitoring and close observation in a high dependency unit/ICU setting for next 24 to 48 hours.

Postoperative Care

Even if the operation has been successfully completed, the circulation to the transplanted/replanted tissue is still precarious. The period of initial recovery from anesthesia, is the one when patient's cardiovascular and respiratory state is particularly labile and does not take much to initiate a vicious cycle of microvascular stasis and thrombosis.

The patient should be kept warm, well-perfused, regular central venous pressure monitoring and fluid replacement continued. The patient should be given adequate analgesia via plexus/epidural catheter with 0.125 to 0.25 percent bupivacaine solution or more recently available ropivacaine solution with or without opioids, with either continuous infusion or intermittent top-ups or via parenteral (intravenous/intramuscular) analgesics. Postoperative hemogram and hematocrit, serum electrolytes, ABG must be sent. Regular examination for the patency of the anastomosis is usually done by the surgical team, to detect any thrombosis at the anastomotic site or hematoma which would need reexploration. Low molecular weight dextran 40 infusion may be started in the recovery area and continued for 3 to 4 days, in the postoperative period.

The surgical team may use antithrombotic agents to prevent microvascular thrombosis at the anastomotic site. Usually, either aspirin, alone or in combination with dipyridamole or low molecular weight heparin may be used. However a study has demonstrated that there is a similar outcome with either of the drug, when used as a single drug to prevent thrombosis.[27] In addition, low molecular weight heparin, also provides prophylaxis against deep vein thrombosis. If the patient is on heparin, the removal of the catheter has to be timed with the last dose of heparin.

REFERENCES

1. Gallico GG III. Replantation and Revascularization of the Upper Extremity. In: Littler JW, May JW Jr. (Eds). Plastic surgery-volume 7 Hand I, Philadelphia: WB Saunders. 1990;4355-83.
2. Hidalgo DA, Jones CS. Role of emergent exploration in free tissue transfer: A review of 150 consecutive cases. Plastic and Reconstructive Surgery 1990;86:492-6.
3. Macdonald DJF. Anesthesia for microvascular surgery. A physiological approach. Br J Anaesth 1985;57:904-12.
4. Guyton AC, The Circulation, Part V. In: Guyton AC, (Ed). Textbook of medical physiology. 6th edn. Philadelphia: WB Saunders 1981;206-57.
5. Wilton TN, Cochrane DF. Anesthesia for plastic and maxillofacial surgery. In: Gray TC, Nunn JF, Utting JE, (Eds). General anesthesia-Vol II. 4th edn. London: Butterworth and Co Publishers 1980;1267-88.
6. Patel DK. Free Flap Surgery. In: Goldstone JC, Pollard BJ, Editors. Handbook of anesthesia. New York: Churchill Livingstone 1996;335-7.
7. Robins DW. The anesthetic management of patients undergoing free flap transfer. British Journal of Plastic Surgery 1983;36:231-4.
8. Hammel HT. Anesthetics and body temperature regulation. The Journal of Anesthesiology 1988;68:833-5.
9. Sessler DJ, Olofsson CI, Rubinstein EH, Beebe JJ. The thermoregulatory threshold in humans during halothane anesthesia. Anesthesiology 1988;68:836-42.
10. Morris RH, Kumar A. The effect of warming blankets on maintenance of body temperature of anesthetised, paralysed adult patient. Anesthesiology 1972;36:408-11.
11. Konrad MK. Hemodilution. Surgical Clinics of North America 1975;55 (suppl 3):659-79.
12. Abboud FM. Integration of reflex responses in the control of blood pressure and vascular resistance. The American Journal of Cardiology 1979;44:903-11.
13. Bailey MJ, Johnston CLW, Yates CJP, Somerville PG, Dormandy JA. Preoperative haemoglobin as a predictor of outcome of diabetic amputations. Lancet 1979;2:168-70.
14. Yates CJP, Berent A, Andrews V and Dormandy JA. Increase in the leg blood flow by normovolemic haemodilution in intermittent claudication. Lancet 1979; 2:166-8.
15. Earle AS, Fratianne RB, Nunez FD. The relationship of hematocrit levels to skin flap survival in the dog. Plastic and Reconstructive Surgery 1974;54:341-4.
16. Laks H, O'Connor NE, Anderson W, Pilon RN. Crystalloid versus colloid hemodilution in man. Surgery, Gynaecology, Obstetrics 1976;142:506-12.
17. Sigurdsson H. Perioperative Fluid Management in Microvascular Surgery. Journal of Reconstructive Microsurgery 1995;11(1):57-65.
18. Berger A, Tizian C, Zenz M. Continous plexus blockade for improved circulation in microvascular surgery. Annals of Plastic Surgery 1985;14(1):16-9.
19. Kurt E, Ozturk S, Isik S, Zor F. Continuous brachial plexus blockade for digital replantations and toe-to-hand transfers. Annals of Plastic surgery 2005;54(1):24-7.
20. Weber S, Bennet CR, Jones NF Improvement in Blood flow during lower extremity microsurgical eree tissue transfer associated with epidural anesthesia. Anesth Analg 1988;67:703-05.
21. Su HH, Lui PW, Yu CL, Liew CS, Lin CH, Lin YT, Chang CH, Yang MW. The effects of continuous axillary brachial plexus block with ropivacaine infusion on skin temperature and survival of crushed fingers after microsurgical replantation. Chang Gung Medical Journal 2005;28(8):567-74.
22. Aarts HF. Regional intravascular sympathetic blockade for better results in flap surgery: an experimental study of free flaps, island flaps and pedicle flaps in rabbit ear. Plastic and Reconstructive Surgery 1980;66:690-8.
23. Holland AJC, Davies KH, Wallace DH. Sympathetic blockade of isolated limbs by intravenous guanethidine. Canadian Anaesthetists Society Journal 1977;24:597-602.
24. Aps C, Cox RG, Mayou BJ, Sengupta P. The role of anesthetic management in enhancing peripheral blood flow in patients undergoing free flap transfer. Annals of Royal College of Surgeons of England 1985;67:177-9.
25. Gruber UF. Dextran and the prevention of postoperative thromboembolic complications. Surgical Clinics of North America 1975;55:679-94
26. Partington MT, Lineaweaver WC, O'Hara, et al. Unrecognized injuries in patients referred for emergency microsurgery. The Journal of Trauma 1993;34 (suppl 2): 238-41.
27. Ashjian P, Chen CM, Pusic A, Disa JJ, Cordeiro PG, Mehrara BJ. The effect of postoperative anticoagulation on microvascular thrombosis. Annals of Plastic Surgery 2007; 59(1): 36-40.

35 Anesthesia for Emergency Renal Transplant

Shakuntala N Basantwani, Geeta A Patkar, Nazmeen I Sayed

KEY POINTS

- Hypervolemia, acidemia, hyperkalemia, cardiorespiratory dysfunction, anemia, and bleeding disturbances are manifestations of chronic renal failure.
- Although dialysis improves some of the abnormalities of ESRD and associated complications, renal transplantation is the preferred method of therapy for most patients as it reverses most of the abnormalities, is cost effective and allows a return to a more normal lifestyle than maintenance dialysis does.
- The low transplantation rate in India is due to the lack of an appropriate cadaver transplant program and the inability of many to afford a transplant due to their socioeconomic status.
- Cadaver organ donation is a family-driven process and can be affected by various social, educational and spiritual considerations.
- Diagnosis of brain death has to be done. Effort to identify potential donors, obtain consent, and convert a potential donor into an actual organ donor is the need of the hour. The organs to be transplanted should be maintained in a state of good function until the moment of recovery.
- Recent improvements in combined immunosuppression therapy have improved the overall outcome in transplant recipients.
- Proper anesthetic management is facilitated by understanding the physiology and pharmacology of ESRD.
- The pharmacokinetics of many drugs is altered in CRF due to decreased renal excretion, with changes in plasma protein binding, alterations in volume of distribution and altered drug metabolism.
- Fifty percent of patients with ESRD have one or more of the comorbid conditions which greatly influences the approach to anesthesia.
- Preoperative hemodialysis may render patients hypovolemic and put them at risk for significant hypotension on induction of anesthesia.
- Intraoperatively, before the release of vascular clamps CVP of 10 to 15 mm Hg, blood volume greater than 70 ml/kg and a plasma volume greater than 45 ml/kg to optimize cardiac output and renal blood flow are associated with immediate graft function.
- The success of cadaveric renal transplantations depends on many factors: Type of donor, length of cold ischemia, and maintenance of adequate hemodynamic parameters at the time of reperfusion.

INTRODUCTION

Patients with end-stage renal disease (ESRD) can have two treatment options:

Dialysis or Transplantation

Although some patients respond well to dialysis, many do not. Children maintained with chronic dialysis do not grow. Adults frequently must give up their jobs and lose their roles as providers and heads of their families as dialysis treatment is done 2 to 3 times a week; 2.5 to 5 hours per session worldwide. Physical and financial dependency often leads to emotional dependency and depression. For those patients who adapt poorly to dialysis, the only hope for a normal life is a new kidney —a real kidney.

Renal transplantations are associated with better quality of life, better cost/benefit ratio, and possibly longer survival. In children, early transplantation promotes better growth and development.[1] Organ donation and transplantation is one of the most powerful and dramatic practices in modern medicine. Renal transplantations have been performed since 1906; however, only after 1960, with the development of new immuno-

suppressive agents, especially calcineurin inhibitors, recognition of brain death, transplant has become the treatment of choice for patients with end-stage renal disease.[2]

In India, about 80,000 patients are added annually to the pool of ESRD, however, only 2.4 percent undergo transplant. This low transplantation rate in India is due to the lack of an appropriate cadaver transplant program and the inability of many to afford a transplant due to their socioeconomic status.[3]

Still, India is one of Asia's leading countries in the field of renal transplants done annually. India started its kidney transplant program in 1970s. To regulate transplant activities, the transplantation of human organ (THO) Act was passed in the Indian parliament in 1994, which heralded a new era in Indian medicine. The act provides for the regulation of removal, storage, and transplantation of human organs for therapeutic purposes and for the prevention of commercial dealings in human organs. The act accepted brain death, which gave a boost to the development of solid organ transplant programmes other than kidney, such as of liver, heart, lungs and pancreas. Since the act has been passed, approximately 1300 such transplants from cadaver donors have been performed of various organs and averages at 100 cadaver transplants a year.[4]

TYPES OF RENAL TRANSPLANTATION[5]

- Living Related kidney transplantation (Genetically related)
- Living nonrelated kidney transplantation (Genetically nonrelated)
- Cadaveric kidney transplantation.

Living Related Kidney Transplantation

Following legal definition this includes first degree consanguinity-parents and children. Under administrative order from the Department of Health it extends the definition of living related donors to include siblings, cousins, nephews, nieces and other blood relatives.

Living Nonrelated Kidney Transplantation

A living nonrelated transplant is a kidney transplant from a donor that is not related to the recipient. These may be spouse, in-law, friend or altruist donor.

Cadaveric Kidney Transplantation

A cadaveric donor is a kidney that is donated by someone who has just died. Cadaveric organ donors are previously healthy patients who have suffered irreversible catastrophic brain injury of known etiology. The brain dead donor should have effective cardiovascular function.

An ideal cadaveric renal donor would be:[6]

- Age between 6 to 50 years
- No history of hypertension, diabetes mellitus
- No bacterial or viral infection
- Normal renal anatomy and function
- No malignancy other than a primary brain tumor or treated superficial skin cancer.

The scarcity of ideal donors and the growing waiting list have forced the transplant community to liberalize donor criteria and to use marginal kidneys and donors also called expanded donors.

Expanded Criteria Donor (ECD)

The term "expanded criteria donor" (ECD) was introduced by Kaufman et al in 1997[7] Marginal kidneys are transplantable deceased donor (DD) kidneys for which the average patient survival, graft survival, and renal function are inferior when compared to standard criteria.

Criteria for expanded cadaveric donors are as follows:[3]

- All donors greater than 60 years
- Donors older than 50 years with any two of the following criteria:
  - Hypertension
  - Cerebrovascular cause of death
  - Preretrieval serum creatinine (SCr) level >1.5 mg/dl
  - With a degree of glomerulosclerosis >15 percent
  - Prolonged cold ischemia >36 hours
  - Non-heart-beating donor (NHBD).

The NHBD is a donor who has suffered an irreversible brain injury (usually from trauma or stroke) and does not have effective cardiovascular function. The patient is pronounced dead only after sustained cardiac asystole, which results in prolonged warm ischemia and damage to the procured organs. This is in contrast to a standard cadaver organ donor who has been pronounced brain dead and the heart is still beating until the very moment of flushing of the donor organs with cold preservation solution. This method usually results in less stress in the procured organs.[3]

CONTRAINDICATIONS FOR CADAVERIC ORGAN DONATION[8]

- An overwhelming infectious process
- An active malignancy.

Fig. 35.1: Organ donor card

Consent for organ retrieval[8]

The public attitude survey indicated a positive attitude of the people towards eye donation. It was suggested that eyes should be requested first and only if the relatives were willing other organs should be requested. This was less likely to upset the relatives in a brain death situation. It also gives an idea about the family's attitude towards the sensitive issue like organ donation in the difficult brain death situation. The request should be made by experienced personnel to properly address all the concerns of the family members so as to increase conversion of the potential donors to an actual donor.

The organs can be lawfully retrieved from a cadaver:[9]

If a person has stated whilst he was alive that his organs could be removed.

Or

If a person has expressed a wish before his death either in writing or orally in the presence of two witnesses during his last illness that his body may be used for therapeutic purposes.

Or

If a donor card signed by brain-damaged patient is available (Fig. 35.1).

If no permission has been given by the patient then the person lawfully in possession of the body, may authorize the removal of any part from the body.

First relative (father, mother, brother, sister, son, daughter and wife) is authorized to consent for organ donation without permission from the government.

In event of the donor not being a first relative an approval has to be obtained by a government appointed authorization committee in each state of the country.

Brain Death[5]

The number of fatal road traffic accidents every year in India is constantly rising. At any given time there are many brain dead patients in different ICU's in any major city of the country. There is hence potentially a huge pool of brain dead donors available in India. The diagnosis of brain death is made in ICUs where facilities exist for sustaining the other organ systems of a brain dead patient.

The criteria for brain death, was first proposed by the Harvard Committee in 1968.

Medical professionals differ regarding the criteria by which death is defined. Usually death is a process, rather than an instantaneous event, therefore, the diagnosis of death can be made when a person has undergone irreversible brain injury. Such a person's existence depends on a machine, he does not have a personal identity, and his chances of regaining an independent life are nil. Therefore, first, the donor must be shown to suffer brain stem destruction as well as cortical death. Second, the diagnosis of death can be made while the heart is still beating and while the function of transplantable organs such as the kidneys, liver and heart, is still normal.

The fact that the diagnosis of death can be made while the heart is still beating and the function of transplantable organs, is still normal, allows time (a few hours only are necessary) for the transplant team to take all necessary steps to fulfil the legal and ethical rules described above before the patient is moved to the operating theater for organ retrieval. It is also possible during this interval to verify and ensure that blood pressure and renal function are optimal by appropriate adjustment of fluid and electrolyte balance, and to administer diuretics and drugs to prevent renal vasospasm.

Brain dead patients have traditionally been given low priority in ICUs and treated with neglect. Therefore, for a good cadaveric transplant program it is essential that when brain dead patients become donors, they should be given the same attention as that given to any critically ill patient.

DIAGNOSIS OF BRAIN DEATH[10]

Before the diagnosis of brain death is considered, certain clinical states must be excluded:

- Severe electrolyte, acid–base or endocrine abnormalities
- Hypothermia (absolute temperature (≤32°C)
- The presence of muscle relaxants or drugs with central nervous system depression.

Other coexisting conditions that may affect interpretation of the clinical examination are severe facial trauma, ocular or pupillary pathology, sleep apnea and

pulmonary disease with CO_2 retention. In such cases confirmatory laboratory testing is recommended.

Evaluation of Cortical Function and Brainstem Reflexes[10]

The cortical functions are evaluated by the lack of motor response to a standard noxious stimulus such as pressure on the temporomandibular joint or the nail bed of a finger. If no movement is elicited then the patient has lost his cortical function.

The assessment of the brain stem function is to be done by testing the brainstem reflexes.

- Pupillary Reflex: The pupils should be dilated and fixed at midposition (4–6 mm) and unresponsive to light
- Corneal Reflex: Touching the cornea with a cotton swab elicits no protective blink
- Oculocephalic and Oculovestibular reflex: No movement of the eyes in response to an acute turn of the head, nor to irrigation of the external auditory canal with ice water
- Gag and Cough reflex: The absence of gag reflex may be observed with stimulation of the posterior pharynx using a tongue blade. The cough reflex is ideally provoked by suctioning the endotracheal tube
- Positive apnea test: It is the last clinical examination that is performed. The recommended protocol for performing this is very specific and is meant to minimize a false-positive result.

Apnea test:[1] Briefly, after preoxygenation with 100 percent oxygen to a Po_2 200 mm Hg and a Pco_2 40 mm Hg, the ventilator is disconnected and 100 percent O_2 is insufflated at 6l/min into the endotracheal tube. The patient is observed carefully for respiratory movements. The appearance of respiratory movements defines a negative apnea test and does not support a diagnosis of brain death. After approximately 8 minutes if no respiratory efforts are noted, an arterial blood sample is drawn, and the ventilator is reconnected. If the measured Pco_2 is 60 mm Hg or 20 mm Hg greater than baseline, the apnea test is considered positive. If the patient desaturates earlier or becomes unstable during the apnea test, an arterial blood sample should immediately be drawn and the patient reconnected to the ventilator. This also indicates a positive apnea test.

In general, two examinations separated by at least six hours are required to establish the diagnosis of brain death and the clinical diagnosis should be confirmed by two or three physicians who are independent of the transplantation team, and at least one of the physicians is required to be a specialist in neurology, neurosurgery, or anesthesiology.[11]

Electroencephalogram and isotope or dye angiography can be used to support the diagnosis, but they are not mandatory.[10]

Certain movements or hemodynamic responses like spontaneous limb movement, deep tendon reflexes, diaphoresis, tachycardia, and hypertension observed particularly during organ procurement are mediated by spinal reflexes rather than by an intact brainstem.

ORGAN DYSFUNCTION IN CADAVER AND ITS MANAGEMENT

It may take several days for identification of a possible organ donor, declaration of brain death, consent, and eventual organ recovery. However, cessation of cerebral function invariably results in a sequence of pathophysiologic changes leading to death of the potential organ donor within a few days, unless appropriate intervention is undertaken.

The pathophysiological changes include:[8]

1. *Hypovolemia:* Regardless of the events surrounding brain death, the majority (up to 91 percent) of brain-dead patients become hypovolemic and hypotensive. Hypovolemia is a common culprit due to inadequate volume resuscitation from the inciting traumatic event, unintended consequences of osmotherapy with mannitol for neurologic trauma, and unrecognized diabetes insipidus. Before considering pharmacologic therapy, volume status must be assessed by CVP measurement and it should be maintained at 6 to 10 mm Hg. Choice of fluid for resuscitation is guided by the patient's hematocrit and electrolyte status. Hemoglobin of 10 g/dl should be accepted as a target level.
2. *Autonomic dysfunction:* Autonomic dysfunction with peripheral vasodilation and left ventricular dysfunction occur due to rapidly declining catecholamine levels after brain death. Volume replacement alone cannot be expected to compensate for the loss of sympathetic tone. Volume overload should be avoided, as it is detrimental to the lungs, liver, pancreas and heart. If, despite good hydration, blood pressure remains low, low-dose dopamine and other inotropic agents, such as dobutamine or norepinephrine should be used. If urine output decreases to less than about 40 ml per hour in a well-hydrated donor, furosemide or mannitol may be given.
3. *Hormonal Insufficiency:* There is a fall in thyroid hormone, antidiuretic hormone (vasopressin), cortisol, and insulin in the hours following brain death. It is believed that profound hormonal insufficiency plays a role in the hemodynamic collapse. A change from aerobic to anerobic metabolism results in decreased

high-energy stores (phosphate and glucagon), lactic acidosis, and progressive organ dysfunction. Insulin administration may be necessary to minimize hyperglycemia and glycosuria; a vasopressin infusion may be required if the hypotonic urine volume is massive (more than 500 ml per hour). Hormonal resuscitation with methylprednisolone, tri-iodothyronine (T3), and arginine vasopressin may improve the quality of recovered organs.

4. *Activation of inflammatory mediators:* Activation of complement system, thromboxanes, platelet and leukocyte factors occur after onset of brain death. As time progresses, there is a progressive leukocyte influx into solid organs with a continuous inflammatory deterioration that enhances the immunogenicity of the graft. Hence, cytoprotective therapy has to be started in order to reduce the initial inflammatory process in the donor.[8]
5. *Hypothermia:* It develops in approximately half of all donors due to lack of hypothalamic temperature regulation and generalized vasodilatation. Hypothermia can cause cardiac irritability and coagulopathy. Prevention of heat loss by application of forced air blankets, heated mattresses and administration of warmed intravenous fluids is effective.[8]
6. *Coagulopathy:* This develops in many patients and is secondary to thrombocytopenia, hypothermia, massive release of plasminogen activator, and other tissue compounds, frequently requiring transfusion of different blood components. International normalized ratio below 2.0 and a platelet count over 50,000/mm^3 should be accepted for organ retrieval.[8]

Briefly, the management of cadaver involves optimization by conventional management followed by assessment of cardiac function by echocardiography. If at this point the ejection fraction (EF) is more than 45 percent, the donor is considered acceptable and may proceed to procurement. If EF is below 45 percent, Hormone replacement therapy (HRT) is recommended, followed by pulmonary artery catheter (PAC) placement (if not already done) and further adjustment of volume and pressor support to meet target criteria. If the patient continues to fail to meet target criteria, it is recommended that the heart not be recovered, although other solid organs may yet be transplant-worthy. Wheeldon et al reported a 35 percent incidence of initially unacceptable donors in their series. Of those, 92 percent were transformed into acceptable donors by aggressive management with HRT and PAC guidance.[1]

Ideal cadaver should be having the following target criteria:[12]

- Systolic blood pressure greater than 100 mm Hg (mean 70 – 110 mm Hg)
- Po_2 greater than 100 mm Hg
- Urine output greater than 100 ml/hr (1 – 1.5 ml/kg/hr)
- Hemoglobin concentration greater than 100g/L
- Central venous pressure (CVP) 5 to 10 mm Hg
- FIO_2 less than 40 percent (if tolerated) for lung retrieval
- Glucose concentrations less than 200 mg/dl (or even <150 mg/dl).

Role of Anesthetist during Organ Retrieval[8,13]

Donor care during the organ retrieval surgery presents a unique combination of various challenges beyond the scope of the anesthesiologist's daily practice. The clinical focus shifts from patient preservation, to that of organ preservation. Interdisciplinary communication must be maintained.

The anesthesiologist has a special role to play in order to maintain cardiovascular function, oxygenation, acid-base balance, electrolyte levels, glucose and temperature within the physiologic range.

- Urine output should be >100 ml/hour. Mannitol, in doses of up to 1 g/kg, is given to ensure diuresis and possibly to minimize ischemic injury
- Heparin, phentolamine (vasodilator), antibiotics and corticosteroids are administered according to the consensus of the multispecialty team. Phentolamine (Regitine), 10 to 15 mg, may be used just before crossclamping of the aorta to decrease systemic vascular resistance. Large doses of corticosteroids are given to deplete circulating donor lymphocytes[14]
- Brain-dead patients do not have pain perception, so analgesics are not required. However, volatile anesthetics or narcotics may facilitate hemodynamic stability
- Neuromuscular blocking agents are given to provide an optimal surgical exposure and suppress the possibility of spinal-reflex induced patient movement. Generally, there is no need for mechanical ventilation and the presence of the anesthesiologist after the crossclamping of the aorta. Obviously, this is not considered the "time of death"[8]
- Significant bradycardia in brain-dead patients which does not respond to atropine should be treated with, direct-acting chronotrope such as isoproterenol

- If thymoglobulin is to be the form of antibody induction that is prescribed, it should be administered during the procedure.

Monitoring

Ideally the anesthesiologist should use standard monitors, measure urine output, and use invasive measurements of arterial pressure and CVP (frequently with a pulmonary artery catheter).[13]

Surgical technique involves wide exposure of the surgical field. Cannulae are placed for *in situ* perfusion, and the organs to be removed are isolated with preservation of their central vascular structures. Removal of the organs is performed under cold protection by applying ice to the surgical field. Heparin is administered (approximately 20,000 – 30,000 IU) when requested by the recovery team. If multiple organs are to be removed; the standard organ removal sequence is as follows: Heart and lungs first, followed by liver or pancreas second, and kidneys last.

If recovery of the heart or lung is anticipated, pulmonary artery catheters or central venous catheters need to be withdrawn before cross-clamping. If lung recovery is anticipated, ventilation is continued well beyond cross-clamping and initiation of organ recovery other than the lung. The kidneys are protected against ischemia by the cold flush and surface cooling during 10 to 15 minutes that it takes to remove the other organs. Excellent communication between the abdominal organ team, thoracic organ team and the anesthesiologist is crucial to ensure optimal organ quality.[13]

Preservation of Donor Kidney

Three main distinct periods have been identified that contribute to injury to the donor kidney:

1. Storage of recovered organs *ex vivo* during transport (generally under hypothermic conditions) from the donor to the recipient.
2. The implantation phase (rewarming phase) without reperfusion.
3. The initial reperfusion phase with the reintroduction of oxygen.

Ex vivo storage involves immediate cooling and flushing of the organ after retrieval with several available preservation solutions. Quick and uniform cooling considerably minimizes warm ischemic damage. However, only cooling to about 4°C is not enough as it does not completely arrest metabolic processes in the cells. Utilization of a correct washout technique and an appropriate preservatve solution is crucial for organ viability. Complete washout of all blood cells from the vascular space ensures uniform distribution of the preservative throughout micro vessels resulting in decreased immune reaction and formation of oxygen-free radicals upon reperfusion.

The preservative is responsible for creation of a colloid-oncotic, osmotic and electrolyte balance across cellular membranes in the preserved organ. Such balance is necessary for maintaining a constant cellular volume, membrane integrity, physiologic pH and nutrients. Lack of Na^+ and K^+ gradients across the membrane decreases ATP-ase activity and conserves energy.[8]

Kidney can be successfully preserved for up to 48 hrs by flushing the organs with the University of Wisconsin (UW) organ preservation solution and storing them at hypothermia (0–5°C). UW solution contains 120 mmol/l of potassium versus only 10 mmol/l in the Histidin-Tryptophan-Ketogluterat (HTK) solution. These differences may have clinical implications, such as a greater potential for hyperkalemia after reperfusion of organs mainly when the preservation solution is not adequately flushed with colloids before implantation. Various other cold storage solutions used worldwide include collins, euro-collins, bretschneider, and celsior perfadex.[13]

Techniques discussed above significantly prolong the functional viability of procured organs and reduce the time constraints of the entire transplant process like transportation, preparation and surgery.

Ischemia Times[13]

Warm ischemia time: It refers to the period between circulatory arrest and commencement of cold storage. With modern *in situ* perfusion techniques, the warm ischemia time is essentially zero, although there is warm ischemia if hemodynamic deterioration or cardiac arrest occurs before harvest. A kidney may function after up to 20 minutes of warm ischemia, but rates of delayed function and nonfunction increase markedly thereafter.

Cold ischemia time: It refers to the period of cold storage or machine perfusion.

Rewarm time: It is the period from removal of the kidney from cold storage or perfusion to completion of the renal arterial anastomosis. Rewarm time can be minimized by cooling the kidney during surgery.

KIDNEY RECIPIENT

Cadaveric renal transplant is an emergency procedure and it is not possible to predict when patients will be offered a DDK. All patients should be kept in optimal medical condition, to decrease the chance of last moment

cancellation, or proceeding at higher recipient risk. Patients of ESRD are on a waiting list for cadaveric renal transplantation, which is maintained by the Zonal transplant coordination center (ZTCC). Patients are selected as per ABO typing and lymphocyte cross-matching from this waiting list. HLA matching is extremely important in living related donor transplants. There is considerable controversy evolving around the role of HLA tissue matching in determining the prognosis of the cadaveric allograft.[15] The overall graft survival rate among cadaver kidney transplant recipients at 3 years is greater than 88 percent, and in live related it is approximately 93 percent.

End Stage Renal Disease (ESRD)[13,16]

The most common causes of ESRD are diabetes, systemic hypertension, glomerulonephritis, polycystic kidney disease, interstitial nephritis, obstructive uropathy, lupus nephritis and congenital malformations of the kidneys and urinary tract.

Patients of CRF can be classified into five stages according to GFR:[17]

1. GFR 90 ml/minute/1.73 m^2 with evidence of kidney damage.
2. GFR 60 to 89 ml/minute/1.73 m^2 with evidence of kidney damage
3. GFR 30 to 59 ml/minute/1.73 m^2.
4. GFR 15 to 29 ml/minute/1.73 m^2.
5. GFR < 15 ml/minute/1.73 m^2 or dialysis-dependent.

Patients with GFR 15 ml/min/1.73 m^2 or less are known as having ESRD.

Absolute Contraindications for Transplant in Recipient

Active infection, active drug abuse, complete thrombosis of the vena cava and iliac veins, and disseminated malignancies.[18]

The perioperative management of patients with ESRD is complicated by both the underlying renal disease, with associated disturbances of fluid and electrolyte homeostasis, altered drug clearance, and the presence of associated comorbid conditions, including diabetes mellitus, chronic hypertension and cardiovascular and cerebrovascular disease.

The perioperative and anesthesia management of these patients must therefore, take into account the specific changes related to renal dysfunction as well as the increased anesthesia and operative risks associated with these comorbidities.[18]

PATHOPHYSIOLOGY OF CHRONIC RENAL FAILURE (CRF)

The kidneys are essential for adjusting body fluid volumes, electrolyte composition, acid-base balance, and hemoglobin concentration. The kidneys receive about 25 percent of cardiac output and function as filters for toxins and drugs in the circulation. When kidney function declines, these functions are impaired.[19]

Anemia[13,18]

Anemia is a common feature of patients with CRF. Erythropoietin deficiency is the predominant cause as kidney is a major source of erythropoietin. This leads to chronic normocytic normochromic anemia and a rightward shift of oxyhemoglobin dissociation curve thus, improving oxygen unloading. Hyperparathyroidism, aluminium toxicity, iron deficiency and decreased red blood cell survival also contributes to anemia.

Prior to treatment with recombinant human erythropoietin (rHuEPO), hemoglobin concentrations of less than 8 g/dl were common although remarkably well tolerated. Many patients were, however, transfusion dependent.

The introduction of replacement therapy with rHuEPO and newer analogues Darbepoetin alfa allows maintenance of hemoglobin concentrations above 11 g/dl and has eliminated the need for transfusion in the majority of patients with CRF. The response to rHuEPO takes 2 to 6 weeks. Therefore, it is effective in the chronic management of anemia of renal disease; it is of little value in the acute management of anemia.

Acute blood loss or severe anemia in the acutely ill patient with ESRD should therefore, be treated with blood transfusion following the same criteria used for patients without renal disease. Careful monitoring of volume status and blood chemistry is required, however, as many patients with chronic renal disease are at increased risk for volume overload and hyperkalemia following blood transfusion. Perioperative hematocrit should be maintained at 30 percent.

Bleeding Diathesis[1]

Patients with chronic renal disease may have an increased risk of bleeding complications. Renal failure *per se* is not associated with abnormalities of clotting factors or of altered platelet number but is associated with platelet dysfunction.

Retained uremic toxins, abnormal binding of von Willibrand factor, ineffective production of factor VIII,

abnormal platelet arachidonic acid metabolism and excess vascular prostacyclin and nitric oxide production have all been implicated in the pathogenesis. In addition, anemia may exacerbate the platelet dysfunction by altering the rheological properties of the circulation, thereby decreasing contact between platelets and the endothelium.

The bleeding time provides the best correlation with risk of clinical bleeding in patients with ESRD. Prothrombin Time (PT), may be normal in patients with clinical bleeding associated with a prolonged bleeding time due to renal failure. Several therapeutic strategies may be employed.

- Preoperative initiation of dialysis usually shortens the bleeding diathesis
- Anemic patients should be transfused to maintain a hemoglobin concentration of at least 10 g/dl in order to optimize the rheologic conditions for hemostasis
- Intravenous desmopressin a synthetic analogue of vasopressin (dDAVP; 0.3 μg/kg of body weight) having a rapid onset of action and duration of approximately 6 to 8 hours may be used. However, tachyphylaxis may develop with repeated doses. It stimulates the release of von-Willibrand-factor VIII complex from the endothelium into the plasma, where it binds and activates the platelets
- Oestrogens are also effective at reducing the bleeding time and may be administered intravenously, orally or transcutaneously. When administered intravenously at a dose of 0.6 mg/kg of body weight daily for 5 days, conjugated oestrogen shortens the bleeding time within 24 to 48 hours and has duration of action of approximately 14 days
- Cryoprecipitate has also been demonstrated to improve the bleeding time and reduce hemorrhagic complications but as a pooled blood product it carries an increased risk of transmitting viral agents.

Cardiovascular System[18]

Cardiovascular disease is the predominant cause of death in patients with ESRD, even after renal transplantation. Causes being acute myocardial infarction, cardiac arrest of unknown etiology, cardiac arrhythmia, and cardiomyopathy.

Hypertension:[13] Hypertension in patients with ESRD is due to:

- Deranged renal handling of Na^+ and fluids leading to volume expansion
- Alterations in levels of vasoactive substances resulting in systemic and local changes in arterial tone.

Hyper-reninemia may lead to increased systemic vascular resistance and elevated systemic blood pressure. If untreated, the elevated systemic pressure within the kidney causes damaging sclerotic changes in the renal vasculature. A vicious cycle ensues in which hypertension damages the kidney and creates the conditions for escalating hypertension.

Cardiomyopathy: Dilated cardiomyopathy and concentric hypertrophy can develop in response to increase in intravascular volume and afterload leading to increased myocardial oxygen requirements. Fluid overload and congestive heart failure occur when the kidneys cannot excrete the daily fluid intake, and hypervolemia ensues. The accumulation of uremic toxins and metabolic acids contributes to poor myocardial performance. ESRD with significantly depressed ventricular function is not a contraindication to renal transplantation, but it may complicate the anesthetic management.

Pericarditis: Pericarditis is now observed more often in under dialyzed patients than in predialysis. Pericarditis may coexist with hemorrhagic pericardial effusion. Cardiac ultrasonography determines the size of an associated pericardial effusion and its effect on myocardial contractility.[20]

Dyslipidemia:[16] Renal failure accelerates the progression of atherosclerosis, especially in the coronary circulation. Uremia causes changes in lipid metabolism, leading to increased concentrations of serum triglycerides and reduced levels of protective high-density lipoproteins. Therefore, the patient's are prone for coronary artery disease.

Arrhythmias: The occurrences of arrhythmias are due to electrolyte abnormalities, i.e. hyperkalemia hypocalcemia, or may represent episodes of myocardial ischemia. Atrial cardiac dysrhythmias are common in the presence of uremic pericarditis.[20]

Congestive cardiac failure: Abnormal cardiac function secondary to myocardial ischemic disease and/or left ventricular hypertrophy, together with salt and water retention in uremia, often result in congestive heart failure and/or pulmonary edema.[17]

Ischemic heart disease: CRD, at all stages, constitutes a major risk factor for ischemic cardiovascular disease, including occlusive coronary, cerebrovascular, and peripheral vascular diseases.

Dialysis is the indicated treatment of patients who are hypertensive because of hypervolemia (remove volume to attain "dry weight") and those who develop uremic pericarditis. Cardiac tamponade and hemodynamic instability associated with uremic pericarditis

and effusion is an indication for prompt drainage of the effusion via placement of a percutaneous pericardial catheter. Rarely surgical drainage with creation of a pericardial window or pericardiectomy is necessary.[20]

Increasing doses of antihypertensive drugs are recommended in patients with hypertension due to activation of rennin angiotensin aldosteron system. ACE inhibitors are used cautiously in patients in whom the GFR is dependent on increased efferent arteriolar vasoconstriction (bilateral renal artery stenosis, transplanted kidney with unilateral stenosis), which is mediated by angiotensin II. Administration of ACE inhibitors to these patients can result in efferent arteriolar dilatation and decreased GFR, which results in sudden deterioration in renal function.[18,20]

Respiratory System

Patients with CRF are prone to develop pulmonary edema secondary to volume overload. However, a unique form of pulmonary congestion and edema may occur even in the absence of volume overload and is associated with normal or mildly elevated intracardiac and pulmonary capillary wedge pressure and is due to increased permeability of alveolar capillary membranes. It is characterized radiologically by peripheral vascular congestion giving rise to a "butterfly wing" distribution. This "low-pressure" pulmonary edema responds promptly to vigorous dialysis.[1]

Central Nervous System

Uremia may cause central nervous system disturbances like decreased concentration, drowsiness, memory loss, myoclonus, seizure, stupor, and coma. Signs of peripheral or autonomic neuropathy are considered strong indications for dialysis.[13]

Fluid and Electrolyte Disturbances[20]

Total-body contents of Na^+ and H_2O are increased in chronic renal failure (CRF) due to deranged renal handling of Na^+ and fluids leading to volume expansion. In ESRD, volume overload may develop as the GFR is reduced to very low levels. Weight gain is usually associated with volume expansion and is offset by the concomitant loss of lean body mass. Administrations of loop diuretics coupled with restriction of salt intake are the mainstays of therapy.

When the GFR falls to 5 to 10 ml/min per 1.73 m^2, even high doses of combination diuretics are ineffective. Extracellular fluid volume (ECFV) expansion under these circumstances usually means that dialysis is indicated.[20]

Hyperkalemia:[20] Maintenance of the extracellular potassium concentration is dependent upon both total body potassium balance and on the distribution of potassium between the extracellular and intracellular compartments. Renal potassium excretion is primarily dependent upon potassium secretion in the collecting duct and is not directly impaired by reductions in GFR.

However, hyperkalemia may be seen with, augmented dietary intake, protein catabolism, hemolysis, hemorrhage, transfusion of stored red blood cells and metabolic acidosis. The medications that inhibit K^+ entry into cells or K^+ secretion in the distal nephron (beta-blockers, ACE inhibitors and angiotensin receptor blockers, potassium-sparing diuretics amiloride, triamterene, spironolactone, and nonsteroidal anti-inflammatory drugs may also cause hyperkalemia.

Hypermagnesemia usually accompanies hyperkalemia (GFR < 10 ml/minute) and can cause neuromuscular weakness, respiratory failure, bradycardia, hypotension and heart block.[17]

Calcium and Phosphate Metabolism[20]

- Decreased GFR leads to reduced inorganic phosphate (PO_4^{3-}) excretion and consequent retention
- Retained PO_4^{3-} has a direct stimulatory effect on parathyroid hormone synthesis and on cellular mass of the parathyroid glands
- Retained PO_4^{3-} also indirectly causes excessive production and secretion of PTH through lowering of ionized Ca^{2+} and by suppression of calcitriol (1,25-dihydroxycholecalciferol) production
- Reduced calcitriol production in CRD results both from decreased synthesis due to reduced kidney mass and from hyperphosphatemia. Low calcitriol levels, in turn, lead to hyperparathyroidism via both direct and indirect mechanisms. In addition, reduced calcitriol leads to impaired Ca^{2+} absorption from the gastrointestinal tract, thereby leading to hypocalcemia, which then increases PTH secretion and production.

Taken together, hyperphosphatemia, hypocalcemia, and reduced calcitriol synthesis all promote the production of PTH and the proliferation of parathyroid cells, resulting in secondary hyperparathyroidism.

Gastrointestinal System[18]

Chronic uremia causes delayed gastric emptying. The mechanism for this may be gastric dysrrhythmia with discoordinated myoelectric activity.

Metabolic Acidosis[20,21]

Acidosis is a common disturbance during the advanced stages of CRD. Decreased renal mass results in decreased NH_4^+ production and excretion of protein load. In the early stages, the organic anions are excreted in urine, and the metabolic acidosis is of the nonanion gap variety. With advanced renal failure, a fairly large "anion gap" may develop however, with a reciprocal decrease in plasma HCO_3^-.

In renal failure, total urinary net daily acid excretion is usually limited to 30 to 40 mmol, and an anion gap of 20 mmol/l with a reciprocal fall in plasma HCO_3^-. It can be corrected by 20 to 30 mmol of $NaHCO_3$ or sodium citrate daily. This will further increase in Na^+ load leading to volume expansion and the potential need for diuretic agents. Citrate enhances aluminum absorption in the large bowel, and citrate-containing agents should be avoided if aluminum-toxicity is seen.

Glucose Metabolism[20]

Glucose metabolism is impaired in CRF, which is seen by a slowing of the rate at which blood glucose levels decline after a glucose load. The mild glucose intolerance seen in uremic patients does not require specific therapy. Because insulin levels are slightly to moderately elevated in most uremic subjects this leads to reduction in doses of many hypoglycemic drugs.

DIALYSIS

Ideally, renal transplantation should precede the need for dialysis. The success of transplantation is negatively affected by lengthy pretransplantation dialysis dependence.[1]

Criteria for Placing Patients on Dialysis Include[20]

- Presence of the uremic syndrome
- Presence of hyperkalemia unresponsive to conservative measures
- Extracellular volume expansion
- Acidosis refractory to medical therapy
- Bleeding diathesis
- Creatinine clearance of 10 ml/min per 1.73 m^2.

The principle of dialysis involves equilibration of waste products in the patient's blood across a semi- permeable membrane to the dialysis bath. Dialysis improves most of the signs and symptoms of uremia (e.g. volume overload, acid-base and electrolyte imbalance, abnormal mental function, coma, pericarditis, CCF, uremic lung, metabolic acidosis, peripheral neuropathy, muscle weakness, and defective coagulation). Hypertension is improved except in patients with high renin levels. However, it does not improve increased lipoprotein level, accelerated atherosclerosis, or pruritus.[21]

Hemodialysis and peritoneal dialysis are renal replacement therapies.

Peritoneal Dialysis[21]

In peritoneal dialysis patient's peritoneum is used as the exchange membrane.

Indications of Peritoneal Dialysis

- Recent cerebral surgery, a cerebrovascular accident, or trauma, as in these cases the risk of fluid shifts or bleeding after heparinization is greater with hemodialysis
- Recent cardiac surgery or a myocardial infarction because the risk of hypotension and arrhythmias
- Recent acute hemorrhage, severe coagulopathy
- Children.

Advantages

- Cost effective
- Clearance of large molecules such as creatinine is good
- Continuous ambulatory peritoneal dialysis can be performed at home.

Disadvantages

- Clearance of small molecules such as urea is less than with hemodialysis
- Peritoneal dialysis is less efficient than hemodialysis, so dialysis times are longer
- Chances of infection and peritonitis

Hemodialysis

Hemodialysis is the most commonly used dialysis technique today.

During dialysis, solutes are removed by diffusion across a semi-permeable membrane within a dialyzer, or artificial kidney, from blood circulated through an extracorporeal circuit. Fluid retained during the interval between treatments is removed by regulating the hydrostatic pressure across the membrane of the dialyzer.[20]

Vascular access is usually obtained by the creation of an end-to-side arteriovenous fistula in the forearm or by insertion of a prosthetic arteriovenous graft when the vessels are inadequate. During hemodialysis, which is performed two or three times weekly, the patient and the external circuit are heparinized to prevent clotting. Flow rates are usually 500 ml/min, so the patient's

blood is exposed to 120 l of fluid during a standard 4-hour dialysis. Variation in ionic content and osmolarity of the dialysate permits correction of abnormalities in fluid and electrolyte balance.

If fluid and electrolyte shifts are too rapid, dialysis dysequilibrium may occur. This syndrome is characterized by weakness, nausea, and vomiting, and occasionally convulsions and coma. In the interval between treatments, clotting does not occur across the arteriovenous fistula because the shunted circuit is short, and blood flow is rapid (150 to 300 ml/min).

Advantages

- Short treatment time
- Highly efficient for small solute such as urea removal.

Disadvantages

- Need for heparin
- Need for vascular access
- Hypotension with fluid removal
- Muscle cramps
- Anaphylactoid reaction
- Infection.

PREOPERATIVE ASSESSMENT

The aim of preoperative preparation of patients with ESRD is to identify and optimize any pre-existing pathophysiology in order to minimize the risk of anesthesia and surgery. Efforts should be made to determine the cause of underlying renal disease. Because ESRD affects all organ systems, it is important to identify and optimize existing organ pathology. A systematic approach is, therefore, useful.

Cardiovascular System

A detailed cardiovascular history is mandatory for all recipients. History of hypertension, antihypertensive drugs and their side effects has to be noted.

Patients should receive their usual blood pressure medications prior to surgery. However, angiotensin system inhibitors administered immediately before operation have been associated with a greater incidence of hypotension upon induction of general anesthesia in hypertensive patients. As a result, these drugs should be withheld in patients undergoing kidney transplantation, particularly if they have other risk factors for hypotension upon induction of general anesthesia.[18]

There is no absolute consensus at this point on the optimal cardiac workup for ESRD patients coming for kidney transplant.[11] So, in newly diagnosed ESRD unrelated to diabetes patients who are young, a preoperative ECG and stress test may be sufficient, whereas a stress echocardiogram or a cardiac catheter may be indicated for symptomatic patients or patients with long-standing ESRD associated with diabetes. Many older and diabetic patients are unable to undergo exercise ECG testing and may have "silent" cardiac ischemia. Noninvasive testing in the form of chemical (dobutamine) stress echocardiography should be done in these patients.[13]

Patients with a positive stress test should proceed to a coronary angiogram. Patients who have critical lesions should probably undergo coronary angiography for possible correction prior to transplantation.[1] However, the decision to perform cardiac catheterization should not exclusively depend on the results of the stress test as the sensitivity and specificity of stress tests are relatively low.

Calcific aortic stenosis valvular heart disease, pericardial effusion, LVH, left ventricular dysfunction is common in transplant candidates. High incidence (40 percent) of pulmonary hypertension is present in patients receiving long-term hemodialysis through an arteriovenous fistula and when suspected it is important to perform an echocardiogram to elicit systolic or diastolic dysfunction because this may have important prognostic implications. ESRD with significantly depressed ventricular function is not a contraindication to renal transplantation, but it may complicate the anesthetic management and improvement in the ejection fraction can be documented after transplantation.[16]

Cerebrovascular Disease

Signs and symptoms of cerebrovascular disease in transplant candidates must be evaluated. Risk factors for cerebrovascular disease include a history of smoking, diabetes, hypertension, hyperlipidemia, and old age. Patients who have had recent transient ischemic attacks or other cerebrovascular events should be evaluated by a neurologist. Patients receiving anticonvulsant medications for a seizure disorder should undergo neurologic assessment to determine if these medications should be continued in the perioperative period and their interaction with immunosuppressants.[16]

Peripheral Vascular Disease[13]

Males, diabetics, patients with hypertension, lipid abnormalities, a history of vascular disease elsewhere, and chronic smokers are at higher risk for peripheral vascu-

lar disease. In these patients noninvasive evaluation of the peripheral vasculature (doppler) should be done. Angiography should be considered if noninvasive studies suggest the presence of large-vessel disease.

Diabetes Mellitus[16,18]

DM is the cause of ESRD in nearly 30 to 40 percent of cases. Nephropathy develops in nearly 60 percent of insulin-dependent diabetics. Patients with ESRD and diabetes mellitus have higher cardiovascular risk than patients with uremia alone because of the associated acceleration of small-vessel atherosclerosis.

Doses of oral hypoglycemic agent, insulin, and their side effects if any should be noted. Hypoglycemic agents should be withheld on the morning of the surgery. These patients also have autonomic neuropathy, peripheral neuropathy, a higher incidence of CAD and silent MI. Symptoms of gastroparesis, such as heartburn, bloating, and explosive diarrhea, indicate autonomic neuropathy and these patients are at risk for aspiration of gastric contents upon induction of general anesthesia.

The evaluation of the airway is particularly important for patients in whom diabetes is the cause of renal failure. Patients with long-standing diabetes often develop stiff joints due to glycosylation of the connective tissue that results from elevated blood sugar levels. The inability to oppose the palms of the hands suggests that stiff connective tissue may be present in a diabetic patient. Patients with stiff joints may be difficult to intubate and may require an awake, fiberoptic intubation.

Infections[1,16]

A detailed history of infectious disease should be obtained. Respiratory tract, skin, ENT, dental, and female genital tract infection should be ruled out and treated. History of tuberculosis, prior treatment of antitubercular drugs, and its duration should be elicited. Evidence of other possible infections including hepatitis and endemic fungal infections should be sought. The presence of chronic infection precludes transplantation and the use of immunosuppressive therapy. Whenever possible, transplant candidates should receive immunization for infections that are prevalent.

Patients with HIV/acquired immune deficiency syndrome (AIDS) were long regarded as inappropriate transplant candidates because of the fear of immunosuppressant-induced opportunistic infection and a short life span. The onset of effective antiviral therapy has radically altered the prognosis of infected patients. Patients who are consistently receiving and tolerating an effective antiviral regimen (with an undetectable viral load and normal T-cell counts) can be considered as candidates.

PREOPERATIVE INVESTIGATIONS AND PREPARATION

Patients scheduled for renal transplant should be dialyzed on the day preceding surgery. A longer interval between the dialysis procedure and surgery increases the risk of volume overload, acidosis or hyperkalemia.[16] Details and adequacy of dialysis needs to be checked. Fluid balance often is difficult to assess in patients on chronic dialysis. Patients who are dialyzed the day prior to surgery and with a weight loss of more than 2 kg have a high incidence of hypotension upon induction of general anesthesia because of hypovolemia. Hence, we must be prepared to rapidly replace the intravascular volume in those patients.[18]

The recipient should have post dialysis blood investigations such as Hb, CBC, platelet count, electrolytes, BUN, serum creatinine, PT, PTT, INR, and ABG. Other investigations include serum glucose, liver function tests, urinanalysis, ECG, chest radiograph and 2 D Echocardiogram.

Screening for hepatitis B and C, syphilis, human immunodeficiency virus (HIV) and cytomegalovirus (CMV) should be done as patients with renal disease are at increased risk for acquiring hepatitis C because of frequent transfusions and exposure to HCV-contaminated dialysis equipment.[19]

Doppler of major vessels should be done to rule out vascular abnormalities.

ANESTHETIC MANAGEMENT

Both regional and general anesthesia have been successfully used for renal transplantation. General anesthesia is most commonly used technique. General anesthesia provides the advantage of mechanically maintaining the patient's ventilation, which may become compromised by surgical retraction in the area of the diaphragm.[23]

General Anesthesia

Premedication: Atropine and glycopyrrolate are eliminated 20 to 50 percent by kidney. Because they are administered as single doses accumulation with toxic effects is unlikely to be a significant problem.[19] Anxiolysis is achieved with water soluble benzodiazepine midazolam. It is preferred over lipid soluble diazepam as it is less affected by reduced plasma proteins, volume

of distribution and clearance. However, the dose should be reduced because of the increased sensitivity in these patients.[16]

Patients of ESRD are at a risk of aspiration under anesthesia. Thirty ml of 0.3 molar sodium citrate as an oral antacid is beneficial. Inj metoclopramide 10 mg IV and H_2 receptor blocker ranitidine should be given ½ hour prior to induction of anesthesia. However, their doses should be reduced as they are excreted through kidney.[18]

Monitoring: Minimum monitoring recommended by ASA, i.e. 5 lead ECG, blood pressure, oxyhemoglobin saturation by pulse oximetry, end tidal CO_2, temperature and urine output.[16]

- Central venous pressure[1,16] monitoring is very important as these patients have fluid abnormalities, and associated cardiac disease. It determines the volume status of the recipient at the time of reperfusion of the allograft. Thus, it reduces the risk of acute tubular necrosis and graft failure caused by hypovolemia. It also provides convenient vascular access to obtain blood samples and for administration of immunosuppressive drugs that must be administered centrally
- Neuromuscular junction monitoring[16] is required as the reported recovery times from neuromuscular blockade are highly variable in renal failure patients
- Protection and monitoring of the hemodialysis vascular access (shunts or fistula) is mandatory. No intravenous catheters, including central venous catheters, should be placed on the same side as an arteriovenous access. Blood pressure should be measured only in the contralateral arm. The limb with the arteriovenous access should be carefully positioned to prevent inadvertent occlusion. It is important that armbands, which may migrate up the arm and occlude a forearm access, not be placed on the access arm. Access patency needs to be carefully monitored both intraoperatively and postoperatively by palpation or by doppler and any alteration in access function addressed promptly. In the event of delayed graft function or even non function following kidney transplantation, a functioning dialysis shunt is invaluable in the postoperative period.[17]
- In diabetic patients blood glucose determinations should be made every hour. Blood sugar levels should be tightly controlled to reduce the incidence of wound infections
- Potassium levels also should be monitored, as these patients are prone for hyperkalemia. It should be treated with calcium gluconate, sodium bicarbonate, glucose with insulin or with hyperventilation.[18]
- Invasive blood pressure monitoring[16] is done in patients with refractory systemic and pulmonary hypertension
- Pulmonary artery catheters[16,18] are indicated in patients with CAD, left ventricular dysfunction, poorly controlled hypertension and valvular heart disease where CVP may not reflect left atrial pressure
- Transesophageal echocardiography:[16] It is very rarely required to differentiate hypotension caused by hypovolemia or left ventricular dysfunction.

Induction: At the time of induction, volume status, pharmacokinetics of induction agent and comorbid conditions must be considered. Low serum albumin levels leads to an increase in free fraction of the protein bound drug in plasma. Uremia is associated with altered blood brain barrier and can increase the levels of unbound drug crossing the blood brain barrier into CNS receptors. Hence, the dose of induction agents may need to be adjusted according to the volume status, and pH. There is increased sensitivity of the nervous system to this drugs.[19]

Propofol 2 mg/kg is the most popular induction agent which should be given cautiously.[22] It may cause peripheral vasodilatation and profound hypotension.[22]

Thiopentone in reduced doses (2–2.5 mg/kg) in view of reduced protein binding can also be used.[18]

Etomidate (0.2 mg/kg) can be safely used in hemodynamically compromised patients because it causes minimal myocardial depression and its pharmacokinetics are not significantly affected in CRF.[13].

Ketamine may not be a suitable drug due to its hypertensive response.

Intubation: In patients who do not have a history of gastroparesis or acid reflux disease, intermediate-duration nondepolarizing muscle relaxants that do not depend on renal excretion for elimination, such as atracurium (0.75 mg/kg), rocuronium (0.5 mg/kg), cisatracurium (0.2 mg/kg), can be administered to facilitate tracheal intubation. In patients with gastroparesis or acid reflux disease, a rapid sequence induction, including the Sellick maneuver to prevent regurgitation of gastric contents, should be performed.[18] The depolarizing agent succinylcholine (1.5 mg/kg IV) can be used to provide rapid skeletal muscle relaxation for immediate tracheal intubation provided serum potassium level is <5.5 mmol/l. In patients with elevated potassium level (>5.5 mmol/L)[1] nondepolarizing agent, rocuronium can be used for rapid sequence induction. At high doses (1.2 mg/kg) it has an onset of action of 60 to 90 seconds and is metabolized by the liver and does not require renal elimination. Fentanyl (50–100 µg/kg IV) can be administered during induction to blunt the hypertensive response to tracheal intubation.[18]

Maintenance of Anesthesia

A balanced technique combining volatile anesthetics with opioids and muscle relaxants is used.

- Volatile agents: Nitrous oxide can be used with the potent inhaled agent. It has minimal side effects, no renal toxicity, and rapid elimination. Isoflurane is the inhalational agent of choice because of its mild cardiodepressive effects, peripheral vasodilatation, preservation of renal blood flow and minimum metabolism to inorganic fluoride which is nephrotoxic.[19] Sevoflurane is rarely used for renal transplantation. It is metabolized by the liver to produce inorganic fluoride which causes renal toxicity. Although fluoride toxicity has not been reported with sevoflurane, the possibility still exists particularly in a donor organ that likely has sustained some degree of ischemic damage.[15] Sevoflurane also reacts with soda lime to form a substance (Compound A) that is in nephrotoxic in some human studies. Therefore, the safety of sevoflurane in renal transplantation is unclear.[1] Desflurane biodegradation does not increase fluoride concentration. No evidence of deterioration in renal function has been noted.[18]
- Muscle Relaxants:[1,16,18] Ongoing skeletal muscle relaxation can be provided with nondepolarizing muscle relaxants that do not depend on the kidney for elimination, such as atracurium, cisatracurium, rocuronium, and vecuronium. They have minimal effects on heart rate and blood pressure. Atracurium and cisatracurium are broken down in the plasma by Hoffman elimination, which does not depend on renal or hepatic function. Its duration of action is not prolonged in renal failure. However, laudanosine, a metabolite of atracurium, has CNS-stimulating properties. Unlike atracurium, laudanosine is dependent on the liver and kidney for its elimination and has a long elimination half-life. Although the liver metabolizes rocuronium and vecuronium primarily, the duration of blockade with these two agents may be prolonged if large doses are used. Pancuronium should not be used in kidney transplant recipients because the drug depends primarily on the kidney for elimination. The effect of mivacurium is prolonged by approximately 50 percent in patients with renal failure, as the activity of cholinesterases are reduced, leading to a prolonged drug half-life.[17] For all muscle relaxants, the reported recovery times from neuromuscular blockade are highly variable in renal failure patients, and careful monitoring of the degree of neuromuscular blockade is recommended.
- Opioid Analgesics:[1,16,18] The pharmacokinetics and pharmacodynamics of fentanyl, alfentanil, sufentanil, and remifentanil are not significantly altered by kidney disease and are not associated with significant histamine release. Therefore, they can be used without modifying the dose. However, large doses or prolonged administration of meperidine and morphine is of concern. M-6-Glucuronide, a metabolite of morphine, accumulates in renal failure and mediates CNS and respiratory depression. Meperidine is metabolized in liver to normeperidine, also excreted by kidneys and its accumulation leads to excitatory CNS effects such as convulsions. In addition, meperidine and morphine cause histamine release, which may result in hypotension and hemodynamic instability.

Reversal and Extubation[13]

Towards the end of surgery when good attempts at respiration are seen and TOF ratio is 0.9, residual neomuscular blockade is reversed using anticholinesterase drugs (neostigmine, physostigmine) and anticholinergic drug (atropine, glycopyrrolate). However, in some patients due to hypothermia or due to variable response to neuromuscular blocking agents, recovery times are prolonged. In such patients ventilation is to be continued and patients are transferred to intensive care unit. Elimination half-life of anticholinesterases are prolonged in patients with ESRD and plasma clearance may exceed clearance of nondepolarizing agent, therefore, recurarization is unlikely.

Methods to Improve Graft Perfusion

- Hypotension can result from hypovolemia or from products of ischemia from the graft or lower extremity when the vascular clamps are released. The microvasculature of the graft and lower extremity are also vasodilated maximally with reperfusion after a period of ischemia and can result in low peripheral resistance[18,23]
- Intraoperative volume expansion is associated with increased renal blood flow and an improvement in immediate graft function. Increased hydration works by atrial distension and subsequent release of atrial natriuretic peptide and increased renal perfusion.[19] Central venous pressure maintained in the range of 10 to 15 mm Hg usually achieves this goal and decreases the incidence of acute tubular necrosis after transplantation. This can be achieved by infusion of intravenous 0.9 percent saline, 0.45 percent saline or 5 percent albumin prior to perfusion of the al-

lograft.[1] In cases of cadaveric donors, volume expansion with albumin in doses of 1.2 to 1.6 g/kg is associated with improved outcome.[24] If the patient is anemic (hemoglobin <10 g/dl), transfusion with packed cells that are saline washed, irradiated and cytomegalovirus negative can be done[16]

- Adequate volume expansion and maintaining the systolic blood pressure 130 or 140 mm Hg by reducing the concentration of inhaled anesthetic agents prevents the hypotension seen with reperfusion of the allograft. Injection sodium bicarbonate (1 mEq/kg) should be given to offset the effects of clamping of renal and iliac vessels. Vasopressors with alpha agonist activity should be avoided as they can compromise blood flow to the transplanted organ
- Hypothermia: Cooling of the kidney is done by keeping ice around the kidney during surgery. This along with other causes of hypothermia under general anesthesia predispose the patients for hypothermia. Hypothermia is one of the factor responsible for delayed graft function. It also prolongs the duration of action of nondepolarizing muscle relaxants and hence delays recovery. Therefore, hypothermia should always be treated prior to reversal
- Other measures to improve urine production and improve kidney viability immediately after reperfusion include use of loop diuretic (furosemide), osmotic agents (mannitol), dopamine and sometimes calcium channel blockers (verapamil)[1]
- The use of mannitol (50 gm) infused IV prior to revascularization of transplanted kidney (cadaveric) is associated with reduced incidence of acute tubular necrosis and hence the need for dialysis.[25] It also acts as a free radical scavenger and increases the release of intrarenal prostaglandins. However, side effects of mannitol like rapid intravascular expansion leading to pulmonary edema and heart failure should be kept in mind. High doses (>200 gm/day) may cause acute renal failure due to renal vasoconstriction
- DA1 and DA2 receptors are two classes of receptors having opposing effects on renal vasculature. Dopamine exerts its effects by acting on both DA1 and DA2 receptors, resulting in increasing renal blood flow, glomerular filtration rate, and sodium excretion. DA1 receptors are postsynaptic and, when activated, elicit vasodilation and inhibition of sodium-potassium adenosine triphosphatase, consequently promoting diuresis and natriuresis, whereas DA2 receptors are less well understood. They are located presynaptically and, when activated, inhibit adenylate cyclase activity (in contrast to DA1 receptors) and norepinephrine release. DA2 receptors activation decreases renal blood flow. Low dose dopamine is commonly used to stimulate DA1 dopaminergic receptors in the kidney vasculature to induce vasodilatation and increased urine output.[26] However, the utility of this approach is questioned in that a newly transplanted, denervated kidney may not respond to low dose dopamine like normal kidneys do[19]
- Fenoldopam:[26] Fenoldopam is a direct-acting systemic and renal vasodilator which is now under evaluation at smaller doses as a renal protectant. At low doses fenoldopam (of 0.01 µg/kg) selectively binds to DA1 receptors without any interaction with DA2 and α1 receptors and acts as renal vasodilator. At larger concentrations (of 0.1 µg/ kg), fenoldopam is an α receptor antagonist, with greater activity at α2 than at α1 receptors. Della et al studied the effects of Fenoldopam 0.1 µg/kg/min on renal function in patients undergoing liver transplant.[26] They concluded that fenoldopam, at a dose that does not interfere with hemodynamics, seems to preserve creatinine and BUN values in patients undergoing liver transplant. Therefore, fenoldopam may have a role as a new pharmacological option as a renal vasodilator
- Hyperacute rejection occurs within minutes to hours of release of clamps of the circulatory system of kidneys during the surgical procedure. The effects produced are so fast that the damage caused is irreversible. This may be a result of preformed circulating antibodies. Corticosteroids infusion prevents interleukin (IL)-1 and IL-6 production by macrophages and inhibit all stages of T-cell activation and prevents hyperacute rejection.

Role of Regional Anesthesia

- The first renal transplant which was performed in 1960 between identical twins was under spinal anesthesia.[11] The advantages of regional anesthesia being:
  - Less incidence of metabolic and electrolyte imbalance.
  - Neuromuscular blocking agents are avoided.
  - Less risk of pulmonary infection.
  - Less incidence of drug induced hepatic or renal dysfunction.[27]
  - Extended postoperative pain relief.

  Combined spinal–epidural anesthesia (CSE) can be used for both surgical anesthesia as well as postoperative pain relief. Before giving regional anesthesia certain points should be kept in mind[28]

- Many uremic patients have at least subtle coagulation defects. Besides, intraoperatively patients may receive heparin. Heparin may also be used for dialysis in the preoperative or postoperative period leading to risk for epidural hematoma
- Epidural anesthesia may complicate hemodynamics due to sympathetic blockade and increase perioperative fluid requirements[1]
- If immunosuppressant azathioprine or antithymocyte globulin (ATG) have been started, platelet count must be seen as they cause thrombocytopenia, which increases the risks associated with central neural blockade
- Although decreased renal function may result in the risk of increased toxic effects of bupivacaine, in clinical doses this does not seem to be an issue.

SURGICAL STEPS

Usual approach is to use the side contralateral to the donor kidney; that is, left kidney is put on the right side, and vice versa. The vessels then lie without kinking when the kidney is placed in position. In repeat transplantations, the side opposite the original transplant is generally used.

A curvilinear incision is taken along the lateral margin of the rectus muscle approximately 8 to 10 inches from just above the pubic bone to just above the umbilicus. The transplanted kidney usually is placed in the extraperitoneal iliac fossa.

The common and external iliac arteries and veins are exposed retroperitoneally.

The renal vein is anastomosed before the renal artery. The renal vein is anastomosed to the external iliac vein in end-to-side or end-to-end manner, depending upon the anatomy of the vessels.

The renal artery is then anastomosed to the internal or external iliac artery. In deceased donor kidney transplantation, the donor renal artery or arteries are usually kept in continuity with a patch of donor aorta called a Carrel patch.

Renal revascularization involves clamping of the iliac artery and vein. This process results in ischemia to the lower extremity for as long as 60 minutes. After the vascular anastomoses are completed, the clamps are released resulting in perfusion of the kidney graft and lower extremities. After reperfusion of the allograft, the urine output, intravascular volume, and overall circulatory status should be monitored carefully.

Finally, the donor ureter is anastomosed to the patient's bladder.[13]

Postoperative care: Postoperatively, after extubation patients are usually monitored in a high dependency area (postkidney transplant care unit). Only 1 percent requires admission to an Intensive Care Unit (ICU) secondary to fluid overload or sepsis.[18] Postoperatively the goals should be: to maintain a well perfused patient with a sufficient blood pressure to allow good graft function. New oliguria (<50 ml/hr) in a hemodynamically stable patient requires assessment of graft blood supply with doppler ultrasound or surgical reexploration.

Postoperative pain: Postoperative pain after renal transplant is usually mild to moderate. Postoperative analgesia using epidural infusions of local anesthetics and opioids have found a valuable place in the management of kidney recipients. Intravenous narcotics such as fentanyl, morphine, can be used as postoperative analgesics. However, morphine (already discussed) must be used cautiously. These agents also can be used for patient-controlled analgesia following discharge from the recovery room.

Inj tramadol can be safely used as it is the least problematic, although dose reduction and increased dosing interval are required, and caution should be exercised.[29]

Nonsteroidal anti-inflammatory drugs (NSAIDs) are absolutely contraindicated as they inhibit prostaglandins synthesis which are integral for renal blood flow and glomerular filtration rate autoregulation and cause gastrointestinal bleeding.[19]

Postoperative Complications

Cardiovascular events are the most common cause of death after renal tansplantation.[1] However, continuous α-blockade, maintaining normothermia, hematocrit >30 percent and good postoperative analgesia helps to reduce cardiac complications in high- risk patients.[18]

Common postoperative anesthetic complications are vomiting and pulmonary aspiration, delayed respiratory depression, pulmonary edema, hypotension, hypertension and cardiac arrhythmias which can lead to cardiac arrest.[19]

Rejection, infection, vascular thrombosis, and urinary obstruction are the commonest surgical complications that may occur in the postoperative period.

Immunosuppression

Immunosuppression in renal transplantation typically consists of two phases.[13]

Induction phase: Induction immunosuppression is important and involves use of large, tapering doses of corticosteroids (e.g. methylprednisolone), large doses of calcineurin inhibitors (e.g. cyclosporine, tacrolimus),

and a polyclonal, e.g. antithymocyte globulin) or monoclonal antibody (e.g. basiliximab, daclizumab, or muromonab-CD3).

Maintenance phase: Less intense than induction immunosuppression and is used for the life of the allograft. It involves a triple-drug combination comprising: a calcineurin inhibitor, an antiproliferative agent such as azathioprine, mycophenolic acid, or sirolimus. Many a times a corticosteroid, such as prednisone.

Different centers' in the world use different regimens to decrease the incidence of graft rejection.

Complications of Immunosuppressant[18]

1. Multiple immunosuppressive drugs predispose patients to infectious and malignant complications.
2. Cyclosporine use may cause hypertension and worsen atherosclerosis in kidney transplant recipients.
3. Nephrotoxicity is an important side effect of both calcineurin inhibitors.
4. Cyclosporine and OKT_3 are associated with renal vascular thrombosis.

Interactions between immunosuppressive and anesthetic drugs: As immunosuppressants are started preoperative it is important to know the common anesthetic interaction.

Cyclosporine enhances the effects of neuromuscular blockade. Hence, requirements of nondepolarizing muscle relaxants are reduced in patients taking cyclosporine.[13]

Transplant Patient Coming for Nontransplant Surgery[18,30]

As the survival rate of most transplant recipients is increasingly approaching 80 – 90 percent (1-year) and continues to improve annually, an increasing number of patients who received a transplant present for either elective or emergency nontransplant surgery.

The general considerations related to any transplant recipient coming for nontransplant surgery are physiological and pharmacological problems of allograft denervation, the side effects of immunosuppression, the risk of infection, and the potential for rejection.

Pharmacological considerations: Transplant recipients are on various regimens of immunosuppression. Various side effects, already discussed, should be kept in mind.

Cyclosporine and tacrolimus may cause a dose-related decrease in renal blood flow and glomerular filtration rate due to renal vasoconstriction as they increase thromboxane A2, and perhaps endothelin production.

Azathioprine and antithymocyte globuline (ATG) have major side effect of bone marrow suppression, therefore, the drug dose may require adjustment for leukopenia or thrombocytopenia.

Graft function: In recipient function of the graft, presence of infection, and function of other organs, particularly those which may be compromised due to either immunosuppressive therapy or dysfunction of the transplanted organ should be evaluated.

The presence of rejection should always be ruled out preoperatively. Rejection leads to a progressive deterioration in organ function tests, and is the main cause of late mortality in the transplant recipients. It should be suspected if functional tests of the kidney, i.e. BUN, creatinine are abnormal.

Infection is a significant cause of morbidity and mortality after transplantation. Therefore, it should be ruled out preoperatively. Immunosuppressed patients are at risk of infections that may be bacterial, viral or fungal. However, the dose of immunosuppressive drugs in the perioperative period cannot be reduced as that may increase the risk of rejection.

Recipients may still suffer from their other systemic disease, such as diabetes or hypertension that initially resulted in renal insufficiency. Use of cyclosporine may aggravate hypertension and worsen atherosclerosis.

Anesthetic technique: A variety of anesthetic techniques (general, regional, neuroleptic) have been successfully used in patients with a transplant history.

General anesthesia: Standard premedication may be used. As in nontransplant patients oral endotracheal intubation is preferred over nasal intubation because of the potential of infection caused by nasal flora. Laryngeal mask airway can be used as an alternative. If perioperative invasive monitoring is required, full aseptic techniques with antibiotic cover should be used. When hepatic and renal function is normal, there is no contraindication to the use of any anesthetic. However, the glomerular filtration rate and effective renal plasma flow in transplanted kidney are likely to be significantly lower than those of healthy subjects, and the activity of drugs excreted from the kidney may be prolonged. Therefore, drugs that do not rely on the kidney for excretion (e.g. atracurium) should be used. Nephrotoxic drugs should be avoided. Nonsteroidal anti-inflammatory drugs should be avoided because of the risk of adverse interactions (e.g. gastrointestinal hemorrhage, nephrotoxicity, hepatic dysfunction).

They augment nephrotoxicity of cyclosporine, as both drugs affect the renal microcirculation. Because of

long-term steroid use, most patients have some degree of adrenal suppression Therefore, a stress dose of steroids may be necessary in renal transplant.[1]

Diuretics should not be given without careful evaluation of the patient's volume status. Renal hypoperfusion from inadequate intravascular volume should be prevented.

If an epidural or spinal technique is planned, clotting studies and platelet count should be normal. Patients taking azathioprine or antithymocyte globuline (ATG) may have thrombocytopenia, which increases the risks associated with central neural blockade.

REFERENCES

1. Harry JM, Lemmens. Kidney transplantation: Recent developments and recommendations for anesthetic management. Anaesthesiology Clin N Am 2004; 22: 651–62.
2. Hirata, SE, et al. Influence of the anesthetic technique on the hemodynamic changes in renal transplantation. A retrospective study. Rev Bras Anestesiol 2009; 59 (2):166-76.
3. Gopalakrishnan G, Gourabathini S. Marginal kidney donor. Indian journal of urology 2007; 23(3): 286-93.
4. Shroff S. Organ donation and transplantation in India-legal aspects and solutions to help with shortage of organs. JNRT 2009; 2(1):23-34.
5. Dossetor JB. Ethics Issues in Kidney Transplantation. Indian J Transplant 2005;1:4-11.
6. Berry JM, Jordan ML, Collin MJ. Renal transplantation. In: Wein AJ, Kavoressi LR, Novick AC, (Eds). Campbell-Walsh urology. 9th edn. Philadelphia: Saunders Elsevier 2007;1295-1324.
7. Ojo AO. Expanded criteria donors: Process and outcomes. Seminars in Dialysis 2005;18:463-8.
8. Murthy T. Organ Donation: Intensive care issues in managing brain dead. Medical Journal Armed forces India (MJAFI) 2009;65:155-60.
9. Sells RA. Live organs from dead people. Journal of the Royal Society of Medicine 1979;72:109-17.
10. Practice parameters for determining brain death in adults: report of the quality standards subcommittee of the American Academy of Neurology. Neurology 1995;54: 1012-4.
11. Mori K, Shingu K, Nakao S. Brain death. In: Miller RD, Erksson LI, Fleisher LA, (Eds). Miller's Anesthesia. 7th edn. Philadelphia: Churchil Livingstone Elsevier; 2010. 3003-23.
12. Gelb AW, Robertson KM. Anaesthetic management of the brain dead for organ donation. Can J Anaesth 1990; 37: 806-12.
13. Yost CS, Niemann CU. Anesthesia for abdominal organ transplantation. In: Miller RD, Erksson LI, Fleisher LA, (Eds). Miller's Anesthesia. 7th edn. Philadelphia: Churchil Livingstone Elsevier 2010; 2155-84.
14. Kandrick E, Singer J, Gritsch HA. Medical and Surgical aspects of kidney donation. In: Danovitch GM, McAllister L, Bersin J, (Eds). Handbook of Kidney Transplantation. 4th edn. Crawfordsville: Lippincott Williams & Wilkins; 2005;166-7.
15. Richie RE, Johnson HK, Tallent MB, Turner B, Vaughn W. The role of HLA tissue matching in cadaveric kidney transplantation. Ann. Surg 1979;189(5):581-5.
16. Sprung J, Kapurral L. Anesthesia for kidney transplant surgery. Anaesthesiology Clin N Am 2000;18(4): 919-44.
17. Palevsky PM. Perioperative management of patients with chronic kidney disease or ESRD. Research Clinical Anaesthesiology 2004;18(1):129-44.
18. Beebe DS, Belani KG. Anesthesia for kidney, pancreas, or other organ transplantation. In: Longnecker DE, Brown DL, Newman MF, (Eds). Anaesthesiology. 3rd edn. New York: McGraw Hill Companies; 2008;1396-1419.
19. Baxi V, Jain A, Dasgupta D. Anesthesia for renal transplantation: An Update. Indian Journal of Anesthesia 2009;53(2):139-47.
20. Skorecki K, Green J, Brenner BM. Chronic Renal Failure. In: Kasper DL, Fauci AS, Longo DL, (Eds). Harrison's Principles of Internal Medicine. 16th edn. New York: McGraw Hill Companies 2005:1653-65.
21. Malhotra V, Sudheendra V, O'Hara J, Diwan S. Anesthesia and the renal and genitourinary systems. In: Miller RD, Erksson LI, Fleisher LA, (Eds). Miller's Anesthesia. 7th edn. Philadelphia: Churchil Livingstone Elsevier;2010; 2105-34.
22. Ickx B, Cockshott ID, Byttebier L. Propofol Infusion for induction and maintenance of anesthesia in patients with end stage renal disease. British journal of Anesthesia 1998; 81:854-60.
23. Garwood S. Renal Disease. In: Hines and Marschall, (Eds). Stoelting's anesthesia and co-existing disease. 5th edn. New York: Churchil Livingstone Elsevier 2008;340-1.
24. Dawidson, Ingemar J, Sandor A. Intraoperative albumin administration affects. The outcome of cadaver renal transplantation. Transplantation 1992;53(4):774-8.
25. Tiggeler R, Berden HM. Prevention of acute tubular necrosis in cadaveric kidney transplantation by the combined use of mannitol and moderate hydration. Ann Surg 1984;201:246-51.
26. Della RG, Pompei L, Costa MG, Coccia C. Fenoldopam mesylate and renal function in patients undergoing liver transplantation: A Randomized, Controlled Pilot Trial. Anesthesia and Analgesia 2004;99(6):1604-9.
27. Linke CL. Anesthesia for renal transplantation. Bulletin of New York academy of medicine (Bull N.Y. Acad Med) 1980;56(5):458-66.
28. Bhosale G, Shah V. Combined spinal-epidural anesthesia for renal transplantation. Transplant Proc 2008;40(4): 1122-4.
29. Murtagh FE, Donohoe P, Wolf CL. The Use of opioid analgesia in end-stage renal disease patients managed without dialysis. Journal of pain and palliative care pharmacotherapy 2007;21(2):5-16.
30. Kostopanagiotou G, Smyrniotis V, Arkadopoulos N. Anaesthetic and perioperative management of adult transplant recipients in nontransplant surgery. Anesthesia and Analgesia 1999;89(3):613-35.

36

Role of Anesthesiologist in Disaster Management

Kanchan R Rupwate, Shalaka A Gandhi, Manju N Gandhi

KEY POINTS

- The various types of disasters include man-made such as fire, bomb explosions, chemical and biological warfare and natural disasters like earthquakes, floods and tsunamis, etc.
- The most commonly expected injuries include burns, crush injuries, hypoglycemia, acidosis and dehydration. The anesthesiologist must be geared up to tackle all of these.
- The administrator plays a very crucial role in managing the manpower optimally and utilizing the available resources in the best possible manner.
- Anesthesiologists are acute care physicians with special expertise in airway management, physiologic monitoring, patient stabilization and life support, fluid resuscitation and crisis management. These are the most important aspects of emergency medical care of the trauma or disaster patient.
- As such, anesthesiologists can serve crucial roles not only in the anesthetic management of civilian and combat casualties in the familiar setting of the operating room, but also, if called upon, can function adequately as team members in field of medical teams, the emergency room, or in the management of intensive care patients.
- Other roles of anesthesiologists are well qualified to perform in disaster include but are not limited to the following:
  - Sort, triage, stabilize and resuscitate casualties.
  - Establish definitive airway control.
  - Provide external hemorrhage control.
  - Diagnose and treat life-threatening conditions commonly observed in victims of disasters (i.e. acute respiratory failure, acute renal failure, poisoning with organophosphates, hemopneumothorax, etc.).
  - Establish intravenous access.
  - Guide fluid resuscitation and blood component therapy.
  - Perform regional and general anesthesia wherever needed or feasible.
  - Transport critically ill patients.
  - Management of acute pain.
  - Alleviate pain and suffering among patients triaged to die.
  - Manage intensive care patients in the ICU or in nonintensive care areas when the number of intensive care patients exceeds ICU bed capacity.

INTRODUCTION

Disaster medicine is a new specialty medical science that deals with the delivery of emergency health and medical services to the injured or ill victims of medical or environmental disasters under extreme or hazardous conditions in the community. The primary aim of disaster medicine is to prevent or mitigate morbidity, mortality, and long-term disability among the victims of disaster by immediately restoring emergency medical and public health services.[1]

Disaster reanimatology, a term coined by Safar, is the study of modern techniques of resuscitation as applied to the critically injured or ill victims of disaster.[1]

The twentieth century has been witness to a marked escalation in the frequency and magnitude of man-made disasters, such as world wars, regional armed conflicts and civil wars, famines, complex cross-border humanitarian emergencies causing the displacement of millions of people, and the epidemic of terrorisms. The latter has brought the spectre of the terrorist use of

weapons of mass destruction.[1] There have been other disasters in India, such as earthquakes, floods, cyclones, as well as tsunamis.[2] In Mumbai itself, we have witnessed disasters such as widespread riots, terrorist bomb blasts, floods, building collapse and fires.

Definition of Disaster

A disaster is defined as a sudden massive disproportion between hostile elements of any kind and the survival resources that are available to counterbalance these in the shortest period of time.[2]

Types of Disaster

Disasters are commonly classified as is shown in Table 36.1

How is a disaster different from other medical emergencies?
Disasters are the events in which the need of patients overextends or overwhelms the resources required to care for them.[3] The disaster response is beneficial in saving lives of the critically injured but treatable victims in the first few hours, the (golden hour). The barriers to prompt response include infrastructure damage directly caused by the disaster (i.e. roadways, lifeline, communications, community services, etc.), poor preplanning, organization, and coordination of mass casualty management. As a result, a second disaster unfolds. This is an important difference that distinguishes a disaster from other medical emergencies.[4]

What is difference between multiple casualty and mass casualty disasters?

Multiple casualty disasters are disasters in which the patient care resources are overextended but are not overwhelmed. For example, in an automobile crash involving five patients can stress local resources such that triage focuses on identifying patients with most life-threatening injuries.[3]

Mass casualty disasters are disasters in which the patient care resources are overwhelmed and cannot be supplemented. For example, natural or man-made disasters involving twenty or more patients that can exhaust local resources such that triage focuses on identifying patients with greatest possibility of surviving.[3]

Phases of Disaster Management

In general terms, the disaster management phases, concomitant activities and the timing of the delivery of emergency medical services will vary according to disaster type.[5] It is divided into following three phases:

I. Initial phase (the first 24 hours): Period of greatest lifesaving potential:
   - Alert and notification

Table 36.1: Classification of disasters

Natural disasters		*Man-made disasters*	
Natural phenomena beneath the earth surface	• Earthquake • Tsunami • Volcanic eruption	Caused by warfare	• Conventional warfare • Nuclear, biological and chemical warfare
Natural phenomena on the earth surface	• Avalanche • Land slide	Caused by accidents	• Vehicular (plane, train, ship, car, etc.) • Drowning • Collapse of building • Explosion • Fire • Biological • Chemical, including, poisoning
Meteorological phenomena	• Wind • Storm (Cyclone, typhoon, hurricane) • Tornado • Hailstorm/snow storm, sea surge • Flood • Drought		
Biological phenomenon	• Locust swarm • Disease epidemic		

- Search and rescue (detection and evacuation of victims)
- Emergency medical service response.

II. Intermediate phase (1-12 days)
- Heavy rescue (victims trapped under heavy rubble)
- Public health measures (postdisaster epidemic surveillance, vector control, etc.

III. Final phase (>12 days)
- Continuation of public health measures
- Rehabilitation of injured
- Reconstruction of affected community.

Activities Involved in Disaster Management

Disaster Preparedness

This involves the activities that a hospital undertakes to build capacity and identify resources that may be used if disaster occurs.

Principles of Disaster Preparedness[1-5]

- Disaster plans should be made.
- Disaster management plan should be (simple), (flexible), (clear and concise) and most importantly an extension of the normal hospital working so that people can act on it immediately.
- Community planning is made at local, regional and national level.
- Acute care specialists such as emergency medicine physicians, anesthesiologists, trauma surgeons, critical care physicians should be involved.
- Well-equipped and trained first responders such as emergency medical services (EMS), police, fire and other professional emergency health workers and services capable of rapid deployment and field operations in the local community should be chosen.
- There should be early warning systems linked with pre-designated evacuation routes for populations at risk (e.g. in coastal areas at risk for hurricanes and tsunamis).
- Epidemiologic or disease surveillance systems (especially in preparing for bioterrorism) should be carried out.
- A significant proportion of the civilian population (> 30%) knowledgeable in what to do in disaster and trained in life supporting first aid should be available.
- In addition, an organized citizen response component such as the Community Emergency Response Teams (CERT), volunteers trained in providing effective support to emergency medical services (EMS) operations should be available.
- A disaster-resistant telecommunications infrastructure (with back-up systems, such as amateur radio network), and a unified management system designed to allow multijurisdictional coordination (police, fire, EMS, hospital, and so forth) enhanced by robust information management and decision support systems are essential.
- The organization should have the flexibility to escalate the delivery of health resources effectively to match resources with demands, the ability to predict and prevent secondary hazards (e.g. fires produced by ruptured gas lines in earthquakes).
- Hospital and departmental planning and training is necessary, i.e. structural and nonstructural integrity of health care facilities are capable of operating in the aftermath of disaster.
- Periodic mock disaster drills (every month) should be carried out to maintain the staff on alert, to check that the equipment is functioning optimally, and to ensure coordination between the participating fellow departments.[4] Drills should be scheduled for different shifts to get as many personnel trained as possible. The importance of drills can ensure disaster preparedness and allow suitable modifications to be incorporated for betterment[5] of the victims.

Disaster Activation Plan

Initial alert

- The first to responder for any disaster in Mumbai is the fire brigade or the police or both, activated through public calls. The calls are then sorted depending on the nature and location of the disaster and passed on to the appropriate authority.[4]
- The moment information regarding a disaster reaches the medical superintendent or emergency medical officer on duty, he alerts all those who are involved in the operation of the plan through a well-defined channel like through telephone operator who then informs all concerned persons about the disaster. The person in the hospital who receives information about the disaster should gather details regarding casualties which include METHANE.[6]
  - M — declaring a major incident
  - E — It's exact location
  - T — Type of incident
  - H — Identified hazards
  - A — Accessibility to disaster area
  - N — Assessment of the number of people affected
  - E — Emergency services required.

Activation of the hospital disaster management plan[5]

- Authorized individuals including chief executive officer, Medical Director, and Director of Nursing or Senior physician in emergency room are called to declare (Doctor Major); a code used to indicate a disaster of undetermined magnitude.
- Depending upon seriousness and scale of emergency, physicians and other hospital staff who are off duty are recalled.
- Specific instructions are given to the chiefs of disaster, emergency room, triage area, emergency waiting room, radiology, operating theater, blood bank, laboratory, medical store supportive services like security, sterile supply, ambulance, etc.
- The plan should be given complete details of resources mobilization. Maximum number of staff should be available within ten minutes of disaster notification. The matron should prepare a pre-arranged ward to receive casualties.
- The place and time of accident determine the reaction time of the hospital while the type of casualty dictates the type of preparation required by the hospital.

Emergency supplies stock

The emergency supplies are stocked up in a dedicated disaster room which is opened in case of mass casualties. The hospital administrator provides for adequate quantities of intravenous cannulae, airways, lines, intravenous fluids, drainage tubes, nasogastric tubes, anesthetic agents, splints, drugs, and dressing materials by opening the stores or through fresh procurement. The need for additional supplies is determined by the senior medical officers and consultants at the hospital. The supplies are replenished from the hospital medical stores.

The routine operation theaters are made available for emergency surgeries. Additional equipments like patient monitors, ventilators, and pulse oximeters are redeployed and organized from the fellow departments.

Triage

The word (triage) is derived from the French verb *trier*, meaning to shift or to sort. Triage is the process of prioritizing and allocating patient treatment during mass-casualty events.[3] Credit for initiating triage is attributed to Baron Dominique Jean Larrey, chief surgeon to Napoleon Bonaparte.[7]

Principles of triage

These are applied when the number of casualties exceeds the medical capabilities that are immediately available to provide customary care.

- The triage occurs at multiple levels such as at the disaster site for those entrapped, again at a primary receiving facility or collection point typically situated close by, and then in the emergency room and operating theaters. It allows the allocation of priorities for initial treatment, transport and in hospital management, including access to operating rooms and intensive therapy unit bed-space.[3,7]
- The triage officer is the decision maker who rapidly assesses the scene by focusing on individuals briefly and makes triage determinations for each patient. A surgeon is the ideal hospital triage officer as he understands all components of hospital.[3]
- Optimal triage decisions are based upon the knowledge and best distribution of the available resources at each stage.[3]
- Triage is a continuous process as some patients may have to be upgraded to the higher triage category while others may deteriorate.[3]
- Upside down or reverse triage may occur as the most severely injured arrive after less injured who bypass EMS and self-transport to closest hospitals.
- As corpses do not cause epidemics, survivors are the priority. However, it is important to identify and tag cadavers and issue death certificates as soon as possible.[8]

The following systems for triage categorization types have been described:

1. Triage categories;[7]
   - T1: Immediate—Casualties needing immediate resuscitation but exclude those with severe and life-threatening injuries where outcome is likely to be poor or where treatment would overburden facilities in terms of time and material.
   - T2: Seriously injured but can tolerate a short delay (30-60 minutes).
   - T3: Can tolerate a delay for as long as necessary
   - T4: Expectant—Includes patients with severe, multisystem injury where prognosis is poor and treatment is unduly time-consuming and demanding.
2. Color coded triage categorization[2,7,9]
   - *Red tag:* Critical; likely to survive if simple care given within minutes; needing immediate management in emergency department. Examples include airway obstruction, tension pneumothorax or catastrophic hemorrhage. Treatment must take place within 60 minutes.
   - *Blue tag:* Catastrophic; unlikely to survive and/or extensive or complicated care is needed within minutes. Examples include burns > 90% or head-

injured patients with GCS 3 despite basic airway and ventilatory measures.
- *Yellow tag:* Urgent; likely to survive if simple care provided within hours; needing indoor admission and treatment. Casualties with serious injury (e.g. long bone fractures) but who can tolerate limited delay. Treatment is suggested within two hours.
- *Green tag:* Minor; likely to survive even if care be delayed by hours to days; may be walking or on stretcher; may be treated as on outpatient basis. These are casualties with minor wounds, small burns or simple fractures (not femur or pelvis). Treatment may be deferred for up to 4 hours.
- *White tags:* Dismiss; are given to those with minor injuries for whom a doctor's care is not required.
- *Black tag:* Expectant; is used for deceased and for those whose injuries are so extensive that they will not be able to survive given the care that is available.

3. Triage sieve and triagesort system (Flow chart 36.1)[7] This is the "sieve-sort" approach of triage categorization based on two-part physiological variables that now gaining widespread popularity. Triage sieve is a simple approach using the ATLS ABC system, while triage sort is based on the triage revised trauma score. The triage sieve will determine priority on field for transport to hospital, where the process will be rapidly repeated and supplemented by the application of triage sort.
4. Quick triage[10]
   - *Priority one:* Needing immediate resuscitation
   - *Priority two:* Immediate surgery
   - *Priority three:* Needing first aid and possible surgery
   - *Priority four:* Needing only first-aid.

Mitigation

Disaster mitigation can be defined as pre-event medical or nonmedical interventions aimed at reducing injury or damage once the event has occurred.[1] It involves all the activities that a hospital undertakes in attempting to lessen the severity and impact of a potential disaster.[3]

Two basic personnel use elements were identified: maintaining a chain of command and perioperative anesthetic management (forward deployment).[11]

Chain of command

A chain of command (institutional and intradepartmental) is essential to control chaos. This incident command system that establishes clear hierarchical lines of responsibility, authority, reporting, and communication for all health care personnel.[11]

Formulation of the command nucleus[5]

The command nucleus which includes the hospital controller, the hospital administrator and the matron should be immediately formulated and it should be near the casualty department.

The roles of members of command nucleus are:
- Appointing the triage officer and medical controller for the ward, casualty department and operation theater (OT).
- Coordinating organizing, communicating and assigning duties to medical officers.
- A senior general surgeon performs triage at the door of the emergency department (ED).
- Another experienced general surgeon acts as the surgical command officer who guides the trauma teams.
- The senior triage surgeon, the senior-most anesthesiologist, the senior-most orthopedic surgeon, and a physician-administrator (hospital management) maintain a log of the most severely injured victims.

Chain of command in anesthesiology

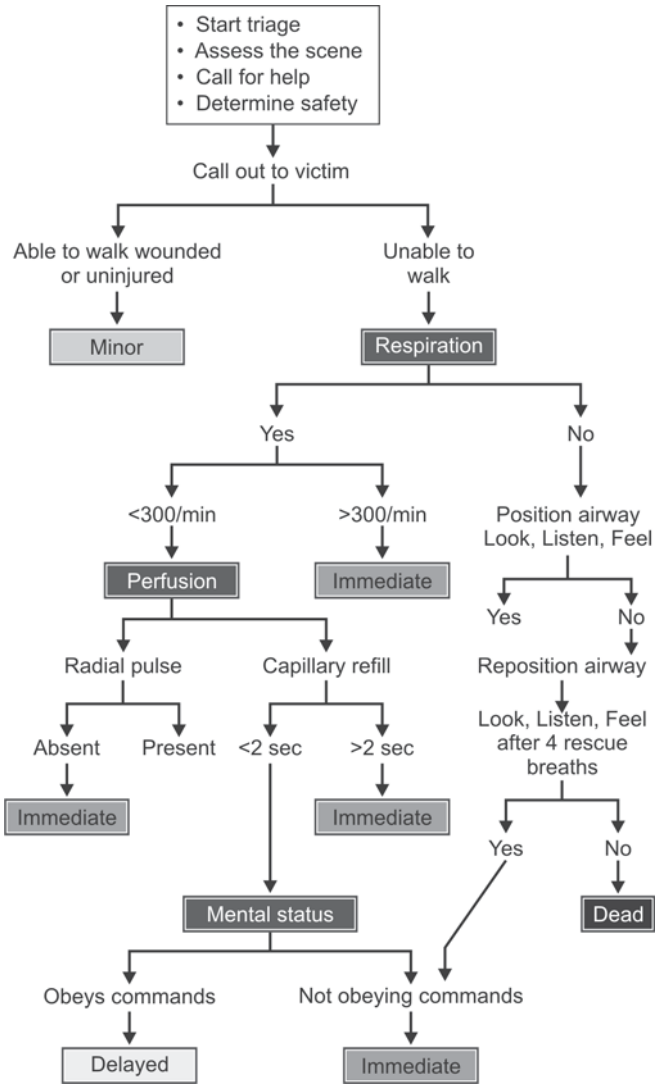
In the disaster scenario, a large number of anesthesiologists have to work in different places and move continuously between locations with the patients. Therefore, the need to have an anesthesiology clinical coordinator and ED supervisor (arises to form a chain of command.[1,11]

Role of anesthesiology clinical coordinator:
- Dispatches anesthesiologists to needed areas
- Discharges patients from recovery
- Prepares an operating room (OR) schedule along with nursing supervisor.

Role of anesthesiology "ED supervisor"
- Receives anesthesiologists from the clinical coordinator and assigns them to specific patients.
- Procures information regarding new arrivals and proposed investigational/therapeutic procedures.
- Assigns anesthesiologists to identify under-triaged or deteriorating patients in lower intensity areas of the ED and also in the remote anesthesia locations (e.g. the radiology department).

Forward deployment means that an anesthesiologist continuously cares for a severely injured patient from admission to the ED, through imaging studies in the radiology department, and during surgery. The advantage of forward deployment is that it permits the anesthesiologist to continuously assess, treat, and prepare the patient for either surgery or ICU while diagnostic or interventional procedures are being performed.

Flow chart 36.1: Triage process



Response

This involves the activities a hospital undertakes to treat the victims of actual disaster. The American College of Surgeons Committee has identified the following service levels of trauma centers that are based on a number of factors, including resources and location.[2]

- **A Level I trauma center** is a lead hospital that is designated as a regional resource leader within a service area and generally serves large cities or population-dense areas.
- **A Level II center** serving a less populated area and must have an outreach program that involves smaller institutions in its service area.
- **A Level III trauma center** must have continuous general surgical coverage, must be capable of managing the initial care of the majority of injured, and must have transfer agreements with other trauma hospitals for patients that exceed its patient care resources.

The Level I trauma center can provide the hospital disaster response in the form of:

A. Prehospital response service
B. Emergency reception, triage and resuscitation service
C. Emergency operative and postoperative service
D. Recovery and rehabilitation after-care service.[12]

Prehospital Response Service

The prehospital trauma team is constituted by the rescue elements such as:

- At least one doctor
- Paramedics
- Fire service personnel
- Emergency medical technicians
- Ambulance officers
- Flying squads—part of accident and emergency department or fire and ambulance service

The survival and morbidity of a patient at the accident site depends upon the timely treatment given by this team.

Role of Prehospital Trauma Team (PHTT)

- Prevention of additional injury by careful patient handling during extrication, loading and unloading onto stretchers and ambulances.
- Splintage of the injured parts must be effective and comfortable for the patients.
- Initiation of treatment—Measures to stabilize the patient like intravenous infusions and endotracheal intubation are carried out.
- Rapid transport—The treatment started at the accident site must be continued during evacuation and transportation to the trauma center. Geographical distance and congested urban traffic contributes to delays in patient transportation.
- To provide a good communication system:
  - A notification function which alerts the emergency department and allows the physician to prepare the resuscitation room and receive the casualty.
  - Relaying of information to other hospitals for specialists intervention.
  - Field management guidance provided by the emergency physician to field personnel.

Prehospital Fluid Resuscitation

Although early cannulation is desirable, priority should be given to transfer of the patient to a center where definitive care can be provided. The on-scene time should not be prolonged by attempts to gain a intravenous line. Intravenous access during transit should be considered with a limit of two attempts en route. But in cases of entrapment, circulatory access should be gained on scene. The entrapped patients are likely to be dehydrated, hypoglycemic or in starvation ketoacidosis. At present, isotonic saline is recommended as the first line fluid in the resuscitation of a hypovolemic trauma patient in aliquots of 250 ml titrated against the radial pulse volume.[13]

Prehospital Anesthesia on Field[14]

- Early analgesia administration should be a priority for trauma cases.
- Regional anesthesia may be used by trained anesthesiologists in the case of fractures of the extremities.
- General anesthesia has many advantages in the management of major trauma. All trauma patients should be assumed to have a full stomach, and a rapid sequence induction is used routinely with succinylcholine. In trauma cases, cricoid pressure with manual in-line stabilization is used only after the anesthesiologist has considered the possibility of risking further cervical spine injury against that of aspiration pneumonia. General anesthesia requires drugs suitable for a hypotensive trauma patient such as ketamine, etomidate and low doses of midazolam. Indications for general anesthesia include extrication of a trapped patient, intractable restlessness, and severe head trauma with presumed elevated intracranial pressure.
- Before extricating the entrapped patients, one should start aggressive fluid hydration in them as they are likely to develop crush syndrome.

Emergency Reception, Triage and Resuscitation Service

This team receives the patients after notification from the PHTT. Triage is done at the doorstep to determine the severity of the injury and to which area of the emergency department the patient be best sent for further managment.

Emergency Department Trauma Team (EDTT)[12]

- A trauma physician—Captain of the trauma team
- Anesthesiologist—To manage the airway, ventilation and fluid therapy
- Assistant to take specimens to the laboratory and return with the results
- A nurse to collect the inventory and store valuables
- A clerk to record vital signs and begin resuscitation chart
- A nurse to help with intravenous solutions and other medications
- Orderlies to move patients.

Inpatient Trauma Team (IPTT)

The trauma surgeon, physician and anesthesiologist decide the course of action for the patient.

Role of the EDTT and IPTT

Trauma score system

Trauma scoring is a useful tool for triage, assessing the injury severity and for trauma system evaluation and comparison and to provide appropriate treatment.

- The acute physiological and chronic health evaluation (APACHE scores) the severity of illness as a predictor of outcome.
- Trauma score injury severity score (TRISS): The TRISS method predicts trauma patient outcomes on the basis of their injuries and enables comparisons of patient outcomes among trauma systems.
- Organ injury scale
- Abbreviated injury scale
- Revised trauma score
- Glasgow coma scale.

Surveys

- **Primary survey (PS)**

The primary survey is performed in a prescribed sequence which reviews the most life-threatening conditions first including evaluation of airway, breathing, circulation, disability and exposure (ABCDE).

Airway maintenance: Ensure patency of the airway with in-line C-spine immobilization.

Breathing: Verify breathing and address life-threatening conditions.

Circulation: With hemorrhage control.

Check for external and internal bleeding, heart rate, pulse character, blood pressure, and capillary refill.

Disability: Evaluate neurological status (GCS and pupils).

Exposure: With environmental control.

Completely undress the patient, examine the entire surface for injuries.

- **Secondary survey (SS)**

The secondary survey entails a comprehensive assessment of the patient and constitutes the initiation of overall treatment planning. The ABCDE reviewed in the PS are reassessed during the SS as standard practice, including careful attention to any perturbations in the vital signs. A useful acronym for this information is - AMPLE

A—Allergies
M—Medications and mechanism of injury
P—Past medical history
L—Last meal
E—Events of the trauma

- **Tertiary survey**

The tertiary survey is an additional head-to-toe evaluation of the trauma patient, usually conducted in the surgical intensive care unit (SICU) 24 hours after admission. The purpose is to completely re-evaluate the patient with the goal of identifying injuries that may have been missed at the initial resuscitation.

Identification of emergent, urgent and nonurgent injuries. Some of the common emergent, urgent and nonurgent injuries are as follows:

Emergent injuries

Airway injury causing obstruction, exsanguinating hemorrhage, intracranial hematoma causing midline shift, tension pneumothorax, pericardial tamponade, flail chest, sucking chest wound.

Urgent

Traumatic near-amputation, peripheral vascular injury, peripheral compartment, unstable spine, open globe injury, perforated stomach or bowel, massive soft tissue injury, open fracture or joint.

Nonurgent

Closed long bone fracture, stable spinal fracture, closed facial fracture, unstable spine soft tissue wound or laceration, joint dislocation.

In order to maximize lifesaving efforts in disasters emergency care should be provided as early as possible and certainly within a critical time period. This period of time can be defined as the 'critical time to treat', which translates into the amount of time an injured or ill patient in a disaster can wait before complications can be expected. Delays in definitive treatment beyond the critical time to treat may cause complications such as loss of limb, other disability, or death.[4]

Table 36.2 shows the critical time for treatment of some common type of injuries.

Emergency Operative and Postoperative Service

A patient's checklist should be followed before proceeding to operating room.[15]

- **History:** AMPLE as explained in primary survey
- **Airway:** If airway not secured, airway examination should be performed. If airway appears difficult to intubate, get difficult airway cart (including fiberoptic bronchoscope), and extra help. High flow oxygen should be administered to all patients
- **Cervical spine:** Status of cervical spine must be known before the induction and precautions must be taken to prevent further injury

Table 36.2: Critical time for treatment of common injuries

Type of injury	*Critical time for treatment in hours*			
	0-0.1	0-1	1-6	6-12
Crush	Head	Chest abdomen		Limb
Uncontrolled hemorrhage				
External		Arterial	Venous	
Internal		Arterial	Venous	
Burns			2nd/3rd degree (>45% BSA)	

BSA—Body surface area

- **Neurological examination:** Assess level of consciousness and brief motor and sensory exam of each extremity.
- **Cardiopulmonary examination:** Auscultate heart, lungs, and perform focused cardiovascular examination. ECG indicated for patients with history of cardiac illness, thoracic trauma, age > 50 years.
- **Injuries and radiographic results:** Be aware of all other injuries, review of all pertinent studies, and anticipate any impact on anesthetic management.
- **Laboratory results:** Abnormal lab values corrected if possible and if time permits. Appropriate access of large bore intravenous accesses, arterial and central lines if indicated.
- **Blood:** Typed and cross-matched: The appropriate number of units, confirm when blood and blood products will be available.
- **Consent:** Obtain consent and risks/benefits/alternatives explained to patient and/or family members.
- **Vital signs stability:** Check if there is need for further resuscitation before the induction of anesthesia. External hemorrhage must be controlled by infusing warm fluid volume through two large bore catheters.

The goal of the anesthesiologist has always been to facilitate surgery (which is often potentially life-saving) in these patients but, in addition, choices and techniques chosen may have a significant effect on long-term outcome.

Practical conduct of anesthesia in critically ill disaster patients.[16,17]

- Conventional assessment of fitness for anesthesia and surgery may not be helpful.
- It is never possible to ensure that the patient has an empty stomach. They should be considered to be full-stomach and given anti-aspiration prophylaxis.
- Many of these patients require ongoing resuscitation. Inotropes and vasopressors should not be given as a substitute for fluids in the hypovolemic patients. However, if the coronary and cerebral perfusions need to be maintained, then short-term use of such drugs while one "catches up" with blood loss is advisable.
- *Perioperative fluid therapy*
  A more rational approach would suggest that crystalloids should be limited in volume, blood loss replaced largely with colloids and packed red blood cells, and that balanced salt solutions such as Ringer's lactate should be preferred to 0.9% saline.
- Perioperative hypothermia is very common in the traumatized patients. Hence, all measures should be undertaken to minimize heat loss and maintain the patients' body temperature.
- *Anesthetic agents*
  Every available technique and drug combinations have been used to anesthetize the critically ill patients. The mode of a drug administration, for examples, the dose of injection, combination of drugs, the rate of administration, route of administration, etc. may be more important than the choice of drug. A novel combination of ketamine and propofol (ketofol) has been used by physician in some emergency departments and may be worth exploring further.[18]
- *Postoperative considerations*
  Critically ill patients may require a period of post-operative ventilatory support and gradual return to spontaneous ventilation.

Psychological Rehabilitation Aftercare Service

Of paramount concern in a traumatized patient is the development of psychological conditions, which may or may not manifest in hospital. Reactions to traumatic experiences tend to be short-lived, but prolonged or excessive maladaptation to trauma requires psychiatric rehabilitation. Post-traumatic sequelae may arise from problems existing before trauma, may be related to the effects of trauma, or may follow stressful medical, surgical, or rehabilitative procedures.

Psychological disturbances attributable to trauma include:[12]

- Survival guilt
- Phobic avoidance (isolation, withdrawal)
- Bereavement
- Loss of future plans; loss of limbs; loss of function

- Sleep problems
- Hypervigilance
- Dissociation

Some post-traumatic stress (PTS) is related to the surgical or ICU experience. Sleep deprivation, terror from the pain of procedures, drug effects, inability to communicate (secondary to paralysis while on ventilator), and disorientation are the factor responsible for PTS. Other sources are worries about child care, financial crises, adverse physical and social outcomes, and generalized uncertainty.

More intensive therapies use desensitization techniques and, if necessary, hospitalization to treat severe psychological symptoms. Counseling and group therapy with specialists in post-traumatic stress management is helpful.

Mental healthcare workers at disaster sites assist people suffering from acute traumatic stress by implementing critical event debriefing.

Specific Conditions of Disaster

Blast Injury[19]

Common blast injuries:

- *Ear injury*: The organ that is most sensitive to the primary blast effect is the ear. 50-80 percent of tympanic membrane ruptures resolve spontaneously. Steroids may be helpful in sensorineural hearing loss.
- *Pulmonary injury*: Second most common is "Blast Lung" which is a consequence of the over-pressurization wave. Prophylactic chest tubes are recommended before general anesthesia or air transport if blast lung is suspected.
- *Abdominal injury*: Most commonly injured is the colon or ceacum. Consider blast abdominal injury in any exposed patient with abdominal pain, nausea, vomiting, hematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia, or acute abdomen.
- *Brain injury*: In addition to more severe coup-contracoup brain injuries, primary blast waves can cause concussions or mild traumatic brain injury. Brain injury should be suspected with complaints of headache, fatigue, poor concentration, lethargy.

The rest of the management of the blast injuries has already been discussed.

Hydrological/Meteorological Events[8]

Ninety percent of hurricane fatalities are caused by drowning associated with storm surges. Other causes of death and injury include the collapse of buildings and resultant penetrating trauma from broken glass or wood, blunt trauma from floating debris, and entrapment in mudslides associated with hurricane floods.

- During tornadoes, the leading causes of death are head trauma and crush trauma to the chest and trunk. Fractures, lacerations, and foreign materials (splinters, tar, dirt, etc.) embedded in soft tissue are very common injuries. Wounds contaminated with gram-negative organisms found in soil are also common and contribute to postoperative sepsis.

Near-drowning[8,15]

The term "near-drowning," alternately, has been assigned to those patients who experience an immersion or submersion event in a liquid medium of sufficient severity to require medical attention to survive, atleast temporarily, the initial postrescue period-usually 24 hours. Drowning can lead to tissue damage due to hypoxia, affecting the brain and causing pulmonary gas exchange abnormalities. Low body temperatures predispose the immersion victim to cardiac dysrhythmias, coagulation derangements, and altered mental status.

Treatment: Hypoxia from pulmonary parenchymal injury unresponsive to supplemental O_2 therapy and bronchodilator therapy requires intubation and mechanical ventilation. Recruitment of collapsed alveoli with mechanical ventilation and positive end expiratory pressure (PEEP) reduces intrapulmonary shunt, improves oxygenation and carbon dioxide clearance.

Geophysical Events[8]

After a volcanic eruption, the high concentrations of volcanic ash can be very irritating to the eyes (causing, e.g. corneal abrasions), the mucous membranes, and the respiratory system. Upper-airway irritation, cough, and bronchospasm are common findings, as is exacerbation of chronic lung conditions. It may cause severe tracheal injury, pulmonary edema, and bronchial obstruction, which could lead to death from acute pulmonary injury or suffocation.

In severe earthquakes, death may be instantaneous owing to severe crushing injuries of the head or chest, exsanguination from external or internal hemorrhaging, or drowning from earthquake-induced tidal waves (tsunamis). Death may also occur within minutes or hours due to asphyxia, hypovolemic shock, or exposure (e.g. hypothermia), or it may be delayed for days and be secondary to dehydration, hypothermia, hyperthermia,

crush syndrome, or postoperative sepsis. Less than 4 percent of people injured by earthquakes require inpatient care. That group will include people with serious multiple fractures or internal injuries, hypothermia, sepsis from wound infections, multiple organ failure, and renal failure secondary to crush syndrome.

Treatment: If lacerations have not been addressed for more than 6 to 12 hours or appear contaminated, they should be treated by debridement and left open for delayed primary closure after approximately three days. Appropriate tetanus prophylaxis should also be administered. Admission of patients with respiratory distress to an intensive care unit is advised.

Warfare Agents

Weapons of mass destruction (WMD) include chemical, biological, and radiological (CBR) agents designed to produce mass casualties against unprotected persons.[15] Dealing with chemical terrorism needs to be well organized with a back up of antidotes and personal protective equipment (PPE).

The primary route of infection or "portal of entry", for biological agents is by inhalation (pulmonary). The symptoms that develop after biological warfare attacks are usually delayed and nonspecific, thereby making the initial diagnosis difficult. Nerve agents exert their biological effects by inhibiting acetylcholinesterase. Nerve agents act so rapidly that treatment must be immediate.[15]

Strategy for chemical and biological disaster response:[6]
Setting up of zones in disaster area (Fig. 36.1):

- *Hot zone*: The affected site is called the hot zone and should be cordoned off. It being the center of the disaster is controlled by the fire service. The main job of team (police force, fire fighters, rescue personnel, medical personnel) inside the hot zone is to contain spread of contamination and restrict spread of damage.
- *Warm zone*: This may be contaminated and due care must be taken to protect the area from further spread. Advance medical posts and medical Evacuation centers are set up by the medical staff. Use of personal protective equipment is mandatory.

  After triage and decontamination in warm zone, the casualties enter the cold zone. START triage is used for the quick assessment of victim's condition using airway, respiratory, capillary refill and age as triage criteria in classifying victims into: dead, immediate and urgent category for initiating treatment.

  Decontamination is done in specially created decontamination stations. The treatment includes disrobing the victims to wash the body with clean warm water, rinse with detergents for disinfection, wipe and again rinse body with clean water. The fact that some chemicals behave differently when in contact with water should be borne in mind while decontaminating the victims.
- *Cold zone*: Doctors and paramedics are permitted to this zone for treatment. It should not be in downward wind direction of Hot zone so as to ensure that gaseous toxins blown by the wind do not contaminate the cold zone thereby impeding the recovery and treatment of the victim (Table 36.2).

Personal Protective Equipment (PPE)[6,20]

Special PPE is provided to the health workers to deal with hazardous chemicals. They have powered filters and prevent direct exposure to the chemicals.

HAZMAT—This is an United Nations controlled Hazardous Materials Control System that gives detailed information to emergency services about the properties and management of toxic substances. HAZMAT databases provide information for protection and decontamination and clinical management protocols. It is a valuable tool and all anesthetists involved in disaster management should be aware of the stages in the overall management of a HAZMAT incident

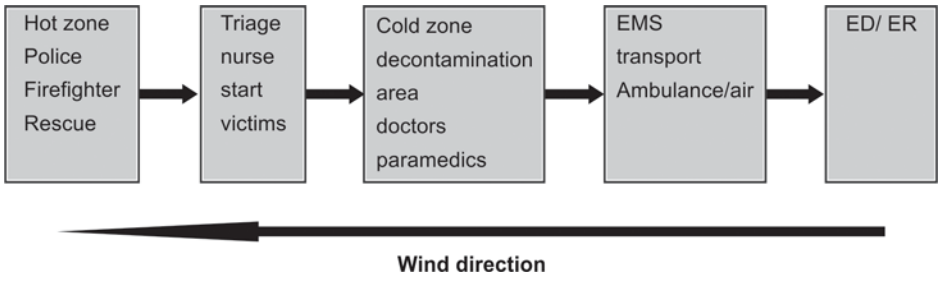


Fig. 36.1: Different disaster zones

including the risks related to the toxicity, latency of action, persistence and transmissibility of the agents involved.

A HAZMAT suit is a garment worn as protection from hazardous materials or substances. A HAZMAT suit is generally combined with breathing apparatus to provide clean, uncontaminated air for the wearer and may be used by fire fighters, emergency personnel responding to toxic spills, researchers, specialists cleaning up contaminated facilities, or workers in toxic environments.

It is sometimes referred to as, an NBC (Nuclear, Biological, Chemical) suit, which is a military version intended to be usable in combat.

Radiation Management

Radiation Dispersal Device (RDD): The RDD is more commonly known as the "dirty bomb." A dirty bomb combines a conventional explosive, such as dynamite, with radioactive material. The conventional explosive itself would cause more casualties than the radioactive material. As radiation death is delayed, the management of conventional injuries and acute life threats takes precedence over radiation exposure. Injury should be treated before decontamination.

Special Considerations

Elderly

- May be at a higher risk of mortality and the in-hospital stay may be longer and more complicated
- Orthopaedic injuries may be more prevalent
- Blunt chest trauma should be of special consideration
- Decontamination methods may need modification due to limited mobility
- Technical decontamination of medical equipment such as wheelchairs, walkers and other walking aides may be needed.

Children

- History of event or patient's complaints may be difficult to obtain.
- Pulmonary contusion is one of the most common injuries from blunt thoracic trauma. The injury may not be clinically apparent initially and should be suspected when abrasions, contusions, or rib fractures are present. A chest X-ray is essential in diagnosis especially when blast lung is suspected.
- Specialized equipment
- Identification of regional pediatric trauma facilities.

Pregnancy

- Injuries to the placenta are possible and must be detected
- Second or third trimester of pregnancy should be admitted for continuous fetal monitoring
- The placental attachment is at risk for primary blast injury
- Screening test for fetal-maternal hemorrhage in second or third trimester of pregnancy
- Positive test requires mandatory pelvic ultrasound, fetal non-stress test monitoring, and obstetrics/gynecology (OB/GYN) consultation.

Disabled

- Consideration should be given to patients with underlying medical conditions
- Untreated or inadequately treated fractures may lead to severe and long lasting disabilities.

Language Barriers

- Diverse population speaking multiple languages may be an unforeseen obstacle
- Interaction with the deaf, hard of hearing, late-deafened and the deaf-blind
- History of the event may be difficult to obtain as well as the individual history for the patient
- Translation
- On scene resources
- Pool of medical interpreters including sign language
- Telephone translation services.

CONCLUSION

The emergency preparedness system lacks trained disaster medicine leaders. Moreover, the field of disaster medicine has not been universally standardized and institutionalized. Education and training programs in disaster medicine are not widely available or firmly rooted in science.

Reduction in mortality and morbidity in mass disaster can be achieved by a well-organized, concise, but flexible, pre-disaster protocol and frequent training. Every hospital must have a disaster management committee with flexible disaster management plan. Elaborate disaster management curriculum needs to be included in the undergraduate medical program of every college. A well-established plan, especially trained staff and regular drills, can make significant change in the outcome of such commonly occurring mass disasters.

REFERENCES

1. Ernesto AP Jr, Paul B. What every anesthesiologist should know about responding to medical disasters advances in anesthesia. Principles and Practice of Disaster Medicine 2008;26:177-99.
2. Avinash S, Rajeev S. Health services responses to disasters in Mumbai sharing experiences. Ind med sci 2008;26:Issue 6:242-51.
3. Advanced trauma life support for doctors. Student Course Manual (8th edn). Chicago, American College of Surgeons 2008.
4. Ernesto AP. Framework for mass casualty management and the role of the anesthesiologist. Anesthesia and Disaster Medicine Part II, 2003;1:2.
5. Kunders GD. Disaster and disaster preparedness. In: Hospitals facilities planning and management. Tenth Reprint 2008;483-8.
6. Bleetman A. EMS response to chemical and nuclear bio-terrorism. EMS India 2009;Issue 11:26-27.
7. Ryan J. Triage: Principles and pressures. European journal of trauma and emergency surgery 2008;34:427-32.
8. Ruben JA, Dauryne S. Disasters produced by natural phenomenon. In: Fundamental Disaster Management. 1999;1-9-16.
9. Melissa CS. Medical triage: Code tags and triage terminology. www.medicinenet.com. Accessed on 2010;31-3.
10. Cedric F, DK Sharma. Disaster Management. In: A practical guide to hospital planning and management (1st edn). Voluntary Health Association of India Press; 1999;68-80.
11. Micha YS, Yoram GW. Multiple casualty terror events: The Anaesthesiologist's Perspective. Anaesth Analg 2004;98:1746-52.
12. Lim SK, Anantharaman V. Organization of a trauma centre. Sing Med J 1988;29:367-70.
13. Greaves I, Porter K. Fluid resuscitation in prehospital trauma care: a consensus view. Emerg Med J 2002;19:494-8.
14. David JB, Caroline T. Multiple casualty incidents: The prehospital role of the anesthesiologist in Europe. Anaesthesiology Clinics of North America 2007;25:179-88.
15. Beverly JN, Carla St L. Preoperative evaluation, preparation, and monitoring. In: Wilson WC, Grande CM, Hoyt DB (Eds). Trauma: Emergency resuscitation, perioperative anesthesia, surgical management. Volume I. New York: Informa Healthcare 2007;332.
16. McConachie I. The critically ill patient undergoing surgery. In: Ian McConachie (Ed). Anaesthesia for high risk patients (2nd edn). Cambridge University Press 2009;198-224.
17. Joshi GP. Intraoperative fluid restriction improves outcomes after major elective gastrointestinal surgery. Anaesth Analg 2005;101:601-5.
18. William EV, Andalfatto. A prospective evaluation of use of "ketofol" for procedural sedation and analgesia in the emergency department. Ann Emerg Med 2007;49:23-30.
19. Bomb and blast injuries. www.emsrb.state.mn.us/docs/Bomb_Blast_Injuries-Final-193.pdf as accessed on 6/1/2010.
20. Baker DJ. Management of casualties from terrorist chemical and biological attack: A key role for the anaesthetist. Br J Anaesth 2002;89:2:212-4.

Index