

Anesthesia

EMERGENCIES



EDITED BY

KEITH J. RUSKIN

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Anesthesia Emergencies

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Anesthesia Emergencies

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Contents

Series Preface	vii
Preface	ix
Managing Emergencies: Lessons Learned from Aviation	xi
Contributors	xv
1 Airway Emergencies	
<i>Sekar S. Bhavani and D. John Doyle</i>	1
2 Cardiovascular Emergencies	
<i>Ajoy Katari and Benjamin A. Kohl</i>	25
3 Equipment Problems	
<i>James B. Eisenkraft</i>	63
4 Ethical Considerations	
<i>Robert B. Schonberger and Stanley H. Rosenbaum</i>	77
5 Metabolic and Endocrine Emergencies	
<i>Lewis J. Kaplan</i>	85
6 Miscellaneous Problems	
<i>Keith J. Ruskin</i>	127
7 Neurosurgical and Neurologic Emergencies	
<i>Ira J. Rampil</i>	143
8 Obstetric Emergencies	
<i>Robert Gaiser</i>	165

9	Pediatric Emergencies <i>Jessica L. Wagner</i>	203
10	Postanesthesia Care Unit <i>Sean M. Quinn and Keith A. Candiotti</i>	239
11	Procedures <i>Ramachandran Ramani and Ala Haddadin</i>	267
12	Regional Anesthesia Complications <i>Raymond S. Sinatra and Dan B. Froicu</i>	297
13	Respiratory Emergencies <i>Vivek K. Moitra and Tricia E. Brentjens</i>	313
14	Surgical Emergencies <i>Linda L. Maerz and Stephen M. Luczycki</i>	337
15	Thoracic Emergencies <i>Marc S. Azran and Michael Nurok</i>	363

Index 389

Series Preface

Emergency physicians care for patients with any condition that may be encountered in an emergency department. This requires that they know about a vast number of emergencies, some common and many rare. Physicians who have trained in any of the subspecialties – cardiology, neurology, OBGYN and many others — have narrowed their fields of study, allowing their patients to benefit accordingly. The Oxford University Press Emergencies series has combined the very best of these two knowledge bases, and the result is the unique product you are now holding. Each handbook is authored by an emergency physician and a sub-specialist, allowing the reader instant access to years of expertise in a rapid access patient-centered format. Together with evidence-based recommendations, you will have access to their tricks of the trade, and the combined expertise and approaches of a sub-specialist and an emergency physician.

vii

Patients in the emergency department often have quite different needs and require different testing from those with a similar emergency who are in-patients. These stem from different priorities; in the emergency department the focus is on quickly diagnosing an undifferentiated condition. An emergency occurring to an in-patient may also need to be newly diagnosed, but usually the information available is more complete, and the emphasis can be on a more focused and in-depth evaluation. The authors of each Handbook have produced a guide for you wherever the patient is encountered, whether in an out-patient clinic, urgent care, emergency department or on the wards.

A special thanks should be extended to Andrea Seils, Senior Editor for Medicine at Oxford University Press for her vision in bringing this series to press. Andrea is aware of how new electronic media have impacted the learning process for physician-assistants, medical students, residents and fellows, and at the same time she is a firm believer in the value of the printed word. This series contains the proof that such a combination is still possible in the rapidly changing world of information technology.

Over the last twenty years, the Oxford Handbooks have become an indispensable tool for those in all stages of training throughout the world. This new series will, I am sure, quickly grow to become the

standard reference for those who need to help their patients when faced with an emergency.

Jeremy Brown, MD

Series Editor

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Preface

Anesthesiology is unique among medical specialties, in that most anesthetics and surgical procedures are uneventful. Critical events can, however, occur without warning, are usually sudden, and may be life-threatening. Anesthesiologists must be ready to detect and manage unpredicted emergencies at any time.

The initial response to a critical event may determine its outcome. A recent study found that early recognition and effective management of complications were as important as their avoidance in improving mortality during surgery.¹ The use of checklists and established procedures, long an accepted practice in aviation, can help health care providers quickly establish a diagnosis and begin treatment. *Anesthesia Emergencies* can be used to prepare for emergencies that may occur in the future, and to deal with critical events as they happen.

Anesthesia Emergencies is written to help an anesthesia provider deal with common complications or unforeseen emergencies during the perioperative period. Chapters have been organized alphabetically, and each section within a chapter is arranged alphabetically by type of problem. Critical information has been highlighted. Each chapter contains one or more references to textbook chapters or review articles that will provide additional information.

Emergency readiness requires that the caregiver have the knowledge, skills, and equipment necessary to resolve the problem. This book will ideally be used to prepare for emergencies before they occur. The authors recommend that if a specific type of problem can be anticipated (e.g., massive hemorrhage in a postpartum patient being brought to the operating room) the appropriate chapter should be reviewed beforehand. All personnel on the care team should be briefed on the critical events most likely to occur, and the actions that will be taken during an emergency. This book can be used outside of the operating room to prepare for future events by reviewing a section and thinking through the steps that would be needed for successful resolution of the problem.

Ideally, *Anesthesia Emergencies* will be one part of an organized approach that includes simulation and training in crisis resource management. The authors recommend that when an emergency occurs, the anesthesia provider call for help as quickly as possible and

1. Ghaferi AA, Birkmeyer JD, Dimick JB. Variation in hospital mortality associated with inpatient surgery. *N Engl J Med*. 2009 Oct 1;361(14):1368–1375.

delegate tasks as personnel arrive. There should be one person who is clearly in charge and directs the others, but that person should ask for advice and help as needed. When an anticipated critical event occurs, the “Immediate Management,” “Differential Diagnosis,” and “Subsequent Management” sections can be used as checklists, to help the anesthesia provider remember each of the steps that must be taken. If an unforeseen emergency occurs, the same sections can be used as a “Do” list. Follow the suggested procedure, and then refer to the article in “Further Reading” for more information after the patient has been stabilized.

This book would not have been possible without the help of many people. The authors would first like to thank their families for their constant support. We would like to thank our editors, Andrea Seils and Staci Hou, for their advice and guidance. We also thank our authors, who produced outstanding manuscripts and turned them in on time. We thank Ashley Kelley, MD; Jorge Galvez, MD; Glenn Dizon, MD; Emilio Andrade, MD; Michele Johnson, MD; and Viji Kurup, MD for their assistance with the illustrations. Lastly, we thank the residents and faculty of the Yale University School of Medicine, Department of Anesthesiology, for their critical review of the manuscript and their thoughtful comments.

Managing Emergencies: Lessons Learned from Aviation

Dan Gryder

A “crash” is never a good thing, whether it happens to an airline flight or to a patient in the operating room. When an airline flight crashes, the entire world knows about it, and everyone can read about it. Keith Ruskin and I have had the opportunity to fly together; we talked a lot about the similarities between the OR and the cockpit, and in those discussions were able to draw many parallels. In the airlines, the captain used to be the “boss” of the flight and often could never be told that he was wrong. This might sometimes still be true in the operating room, where one or more members of the patient care team may appear to be unapproachable, or become hostile when questioned.

In one tragic aviation accident, the aircraft was dangerously low on fuel. The captain insisted that they had enough, but the other two pilots, who knew the truth, were afraid to disagree with him. Because two members of the flight crew were afraid of being fired, the aircraft ran out of fuel and crashed near New York, killing all aboard. In another infamous airline accident, the left engine caught fire after takeoff. The captain mistakenly insisted that the right engine was on fire. The flight attendant in the back could have told the captain where the problem was, had he or she been asked. The captain elected to shut down and secure the right engine and run the left engine at full power, adding more fuel to the fire. Everyone died.

In yet another accident, one of the green landing-gear indicator light bulbs had burned out, falsely indicating that one of the gears was unsafe for landing. All three pilots bent over the instrument panel in flight, trying to determine if the gear was safe for landing, but no one paid attention to what the airplane was doing. The airplane began a slow descent and eventually crashed in the Florida Everglades west of Miami. Many aboard died.

The National Transportation Safety Board (NTSB) is the government agency tasked with the responsibility of determining causes of airplane crashes and implementing methods of assuring that the same types of accidents do not happen again. In the late 1970s the NTSB

determined that a significant number of accidents were caused by problems related to “human factors.” In other words, there was no mechanical failure. The trained flight crew made time-critical mistakes that in many cases could have been prevented. As a result, the airline industry has evolved and grown safer in recent years. Professional pilots are now required to undergo detailed training in the classroom and in a simulator to learn what to do when an abnormal event takes place. Cockpit resource management (CRM) has been largely credited with the dramatic improvement in airline safety.

The common thread of more than 50 fatal aircraft accidents is very simple: lives were lost that could have easily been spared. It may be possible to draw parallels to similar events in the operating room that are caused by human factors. The only difference between the two professions is that nobody outside the hospital may ever know what really happens during a “crash” in the operating room. The patient can’t tell his story. The family may never know. The personnel who were there have to sleep at night knowing that a preventable chain of errors may have been responsible for that patient’s death. Like aviation, the outcome of decisions made in the operating room is frequently irrevocable. There are, however, lessons that anesthesiologists could possibly adapt from aviation and place into practice that will improve their performance during an abnormal event. The following is a summary of what we in the airline industry have learned:

- The situation always turns from NORMAL to ABNORMAL at some clearly defined point. Learn to recognize that point and announce that point in time.
- If there are any aural or visual warnings that have been activated, acknowledge what they represent, and then silence them. Studies have shown that flight crew performance, and their ability to think and reason, is impaired by as much as 60% by the constant noise from audible alarms.
- During any ABNORMAL scenario, the captain should quickly verbally review the role of each person and assign tasks. For instance, he or she may say, “You fly the aircraft and let the controllers know about our situation. I will run the appropriate checklists and coordinate with the company maintenance department.” The physician primarily responsible for the patient can perform the same role.
- Stabilize the situation and find a way to buy time.
- Once stabilized, solicit as many inputs as possible given the possible time constraints. Even information from a flight attendant or passenger could be useful. Learn to differentiate data that arrives, as “what I saw or smelled or felt” as opposed to “what I think we should do.” Almost every accident review has revealed that someone, somewhere, had the missing piece of information

that could have solved the problem. There is a passenger, a flight attendant, a mechanic, another pilot, or a dispatcher who saw, smelled, or felt something. That person may have the missing clue if only someone would ask.

- Ask for advice. It is not necessary to comply with every suggestion that is offered, but it is best to solicit that information and at least consider each opinion.
- After careful consideration, and using all available resources as tools, the captain will ultimately select the best course of action and implement it, monitoring the situation closely as he or she does so.
- Start the sequence again from the top as necessary.

There are some general strategies that will improve your ability to manage a critical event:

- Announce the abnormal condition! You may be the only person that realizes what you see and the significance of it. It is not necessary to offer a solution at the same time. Simply state that at this point in time, something is wrong.
- Encourage every member of your team to announce a problem. Charge them with the responsibility of announcing *anything* seen as abnormal at any time. You will deal with what you see later, but it is imperative that someone announce an abnormal condition as soon as it is recognized.
- Build time. Find a way to stabilize the situation while resources can be mobilized.
- You are not a fool if you ask for the opinions of others. You are a fool if you do not.
- At some point, a decision must be made. Consider the consequences of each choice and make the best choice after having considered all options and opinions.

The Federal Aviation Administration has developed a “3P” model of decision-making, and this may be applied to the management of critical events in the operating room. The 3P model has three steps: *Perceive*, *Process*, and *Perform*. The first step is to *perceive* that a problem has occurred. Next, all available information is *processed* to determine if a change in management is necessary and, if so, what intervention is likely to solve the problem. The last step is to *perform*, or to make the intervention. After making the intervention, return to the *perceive* step to determine if the intervention had the desired result, and the entire process starts all over again. If the intervention did not have the desired result, consider the possibility that the intervention was not performed correctly, or that the intervention was not appropriate for the problem.

Simple changes in the way you approach a problem and work with members of your team may prevent an adverse event from occurring

and maybe even save your patient's life. Using the information in this introduction will help you be more effective when you manage the emergencies in this book.

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Chapter 1

Airway Emergencies

Sekar S. Bhavani and D. John Doyle

- Airway Fire 2
- Airway Obstruction in the Spontaneously Breathing Patient 3
- Aspiration 4
- Bleeding Following Tonsillectomy 8
- Blood in the Airway 9
- Bronchial Intubation 13
- Cannot Intubate/Can Ventilate 14
- Cannot Intubate/Cannot Ventilate 16
- Difficult Mask Ventilation 17
- Difficult Ventilation through an Endotracheal Tube 18
- Laryngospasm 19
- Ludwig's Angina 20
- Rapid-Sequence Intubation (RSI) 22

Airway Fire

Definition

Ignition of combustible materials (e.g., plastic tracheal tube) in the airway.

Presentation

Any fire requires three components: a fuel source (e.g., alcohol-based skin prep solution, plastic tracheal tube), oxygen, and a source of ignition (e.g., cautery, laser beam).

Immediate Management

- **Immediately disconnect the breathing circuit from the endotracheal tube.**
- Inform the surgeons that a fire has occurred.
- Remove the endotracheal tube.
- Stop flow of all airway gases, especially N_2O .
- Remove all other flammable materials from the airway.
- Pour saline into the airway to extinguish any flaming debris.
- Reintubate the patient, even if injury is not immediately apparent.

Subsequent Management

- After an airway fire has occurred, the patient should be reintubated and examined by fiberoptic bronchoscopy to determine the extent of airway injury.
- Admission to an ICU is often required.

Prevention

- Determine whether there is risk of a surgical fire before every procedure and formulate a plan of action that will be taken if a fire occurs.
- Display a protocol for fire prevention and management in every operating room.
- Whenever the surgical site is near the airway, a 60 mL syringe filled with saline solution should be immediately available.
- Ensure that the surgeon does not enter the trachea with electrocautery during a tracheostomy. Use the lowest FiO_2 that the patient will tolerate.
- When using lasers on the airway, use the lowest possible FiO_2 (40% or less).
- Avoid use of N_2O for surgery near the airway.

Special Considerations

- Alcohol-based skin cleansing solutions are easily ignited.
- The only indication of an airway fire may be a puff of smoke and a flash of light.
- Fires caused by alcohol-based prep solutions may be invisible under ordinary room lighting and can spread within seconds.

Further Reading

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Airway Obstruction in the Spontaneously Breathing Patient

3

Definition

Partial or complete airway blockage that increases upper airway resistance and results in difficult ventilation and impaired oxygenation.

Presentation

- Dyspnea
- Hypercarbia (can contribute to obtundation)
- Hypoxemia (can contribute to obtundation)
- Snoring
- Wheezing
- Stridor
- Use of accessory muscles of respiration
- Tracheal tug
- Apnea
- Agitated patient

Pathophysiology

Airway obstruction in the spontaneously breathing patient has many causes, including aspirated foreign bodies, infections (e.g., epiglottitis, diphtheria Ludwig's angina), laryngospasm, bronchospasm, tumors impinging on the airway, hematomas impinging on the airway, trauma to the airway, tonsillar hypertrophy, obstructive sleep apnea, and airway edema (e.g., anaphylaxis, smoke inhalation, or burn injury).

DIFFERENTIAL DIAGNOSIS

- Ordinary snoring
- Bronchospasm
- Bradypnea or apnea from drug overdose or other causes

Immediate Management

- Increase FiO_2 to 100% to ensure adequate oxygenation.
- Attempt to open the airway with a jaw thrust, a nasopharyngeal airway, an oropharyngeal airway, or a supraglottic airway device (e.g., Laryngeal Mask Airway).
- Consider nebulized racemic epinephrine and/or IV dexamethasone.
- Administer an inhaled bronchodilator (e.g., albuterol) if bronchospasm is suspected.
- Consider endotracheal intubation for unremitting obstruction.
- Consider heliox as a bridge therapy in patients with stridor when intubation is not feasible.

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Aspiration

Definition

Inhalation of material into the airway below the level of the true vocal cords.

Risk Factors for Aspiration with General Anesthesia

Patient Related

- Emergency surgical procedures or surgery at night
- Inability to clear pharyngeal secretions/poor or absent gag reflex
 - Altered mental status
 - Head injury
 - Medications

Risk Factors for Aspiration with General (continued)

- Bulbar palsy
- Multiple sclerosis
- Parkinson's disease
- Guillain-Barre syndrome
- Muscular dystrophies
- Cerebral palsy
- Increased volume of food and acid in the stomach.
 - Recent ingestion of food or fluids
 - Poor gastric emptying/gastroparesis
 - Obesity
 - Diabetes
 - Autonomic neuropathy
 - Pregnancy
 - Ileus
 - Renal failure
 - Head injuries
 - Pain
 - Stress
 - Intestinal Obstruction
 - Pyloric stenosis
- Incompetence of the lower esophageal sphincter.
 - Esophageal reflux
 - Achalasia cardia
 - Hiatal hernia
- Increased intra-abdominal pressure
 - Obesity
 - Pregnancy
- Patients with a history of prior upper abdominal surgery
- Extremes of age

Surgery and Anesthesia Related

- Poor induction technique
 - Large tidal volume or high airway pressure during mask ventilation
 - Incorrectly performed Sellick maneuver
- Difficult intubation resulting in:
 - Gas insufflation into the stomach during mask ventilation
 - Inability to intubate and protect the airway with a cuffed tube.

Risk Factors for Aspiration with General (continued)

- Medications and drugs
 - Opioids
 - Topicalization of the airway leading to suppression of the gag reflex
- Depth of anesthesia
 - Manipulation of upper airway under light anesthesia, leading to gagging and vomiting
- Patient positioning
 - Trendelenberg position
 - Lithotomy position
- Increased intra-abdominal pressure
 - Intra-abdominal air insufflation
 - External pressure on the abdomen

6 Presentation

- Material found in the oropharynx
- Wheezing
- Elevated airway pressure
- Infiltrates seen on chest X-ray
- Clinical findings of ARDS as syndrome develops

Pathophysiology

Factors affecting outcome after aspiration include:

- pH
 - In general, the extent of injury increases with the acidity of the aspirated material.
 - Aspiration of bile is associated with extensive tissue injury.
- Volume of aspirated material
- Particulate matter
 - Particulate matter increases the mortality and incidence of pneumonitis and bacterial overgrowth.
- Bacterial load
- Blood in the airway
 - Blood in the airway generally produces minimal injury, but may predispose the patient to infection
- Host responses

DIFFERENTIAL DIAGNOSIS

- Laryngospasm or airway obstruction during intubation
- Bronchospasm, wheezing, or crackles following intubation
- Hypoventilation, dyspnea, apnea
- Reduced pulmonary compliance (ARDS)

Immediate Management

- Increase FiO_2 to 100%.
- Position the patient with the head down.
- Maintain cricoid pressure.
- Aspirate the nasopharynx and oropharynx.
- Intubate the patient (consider rapid-sequence induction if the patient is not cooperative)
- Suction the lower airway.
- Initiate mechanical ventilation as indicated (may require PEEP of at least 5 cm H_2O).
- Administer bronchodilators (e.g., nebulized albuterol) as indicated.
- Perform bronchoscopy and remove particulate matter.
- Routine prophylactic steroids should *not* be administered.
- Avoid empirical antibiotics unless there is a clear risk factor (e.g., aspiration of feculent matter). In most cases, initiate antibiotic therapy when there is a clear diagnosis of pneumonia.
- Do not administer H_2 blockers or proton pump inhibitors. Antacids and prokinetic drugs have not been shown to improve outcome after aspiration.
- Ensure careful fluid management (since volume shifts may occur that lead to pulmonary edema).
- Defer planned or noncritical surgery where feasible.
- Initiate CXR and ABGs as indicated.

Prevention

- Strictly follow NPO guidelines.
- Ensure early control of the airway in patients with poor gag reflex or sensorium.
- Practice good induction techniques.
- Exercise increased vigilance during intubation *and* extubation.
- Use H_2 blockers/proton pump inhibitors at least 90–120 minutes prior to the surgical procedure.

- Use nonparticulate buffered salts like Bicitra that decrease the gastric pH for over 7 hours.
- Administration of prokinetic agents prior to surgery may decrease the risk of aspiration by decreasing the volume of gastric contents.
- Use rapid-sequence intubation where appropriate.
- Use an awake fiberoptic intubation technique if a difficult airway is anticipated.

Special Considerations

- The incidence of aspiration in adults is roughly 1 in 3000 anesthetics. In patients undergoing emergency surgery, this risk increases to 1 in 600–800, and for caesarean sections under general anesthesia the incidence is 1 in 400–900.
- Children are at increased risk for aspiration with an overall incidence of 1 in 2600 and an incidence of 1 in 400 cases in the setting of emergency surgery.
- The consequences of aspiration can be catastrophic: patients requiring ventilation for more than 48 hours post-aspiration have a 50% mortality rate.

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Bleeding Following Tonsillectomy

Definition

Bleeding from the surgical field after tonsillectomy surgery.

Presentation

Although the most common period for a child to bleed after tonsillectomy is several days following surgery, bleeding may occur within the first few hours after surgery.

Pathophysiology

Causes of bleeding after tonsillectomy include incomplete surgical hemostasis, and various coagulopathic states (antiplatelet agents, anticoagulant therapy, hemophilia, etc.).

DIFFERENTIAL DIAGNOSIS

- Hemoptysis
- Nontonsillar bleeding

Immediate Management

- Evaluate the patient's airway rapidly.
- Consider reintubation in the setting of brisk bleeding or if the patient is unable to protect his or her own airway.
- Ensure that adequate suction is available. Blood in the oropharynx may obscure the view during laryngoscopy.
- Assume that the patient has a full stomach. Regurgitated blood or postoperative oral intake may result in extensive pulmonary aspiration. Rapid-sequence induction of general anesthesia is recommended.
- Request an emergency evaluation by ENT service.
- Restore intravascular volume if the patient has signs of hypovolemia.
- Consider surgical re-exploration of the tonsillar bed.

Special Considerations

The blood supply to the tonsils comes from the external carotid artery and its branches. It is sometimes necessary to embolize or ligate the external carotid artery in patients with severe hemorrhage.

Further Reading

Henderson J: Airway Management in the Adult. In: Miller RD, ed. *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1573–1610.

Blood in the Airway

Definition

Massive hemoptysis is defined as more than 600 mL of blood loss in 24 hours, and exsanguinating hemoptysis is considered to be the loss of at least 1000 mL of blood at a rate of more than 150 mL/h.

Presentation

- Hemoptysis in patients who are unintubated.
- Frothy or frank blood in the ETT of intubated patients.
- Chest X-ray may show signs of aspirated blood.

Etiology**Infection**

- Bronchitis
- Necrotizing pneumonia
- Lung abscess
- Tuberculosis
- Fungal infection
- Parasitic infection

Neoplastic

- Primary lung cancer
- Bronchial adenoma
- Metastatic lung cancer

Cardiovascular

- Pulmonary embolism
- Mitral stenosis
- Left ventricular failure
- Atrioventricular fistula
- Congenital heart disease
- Pulmonary hypertension

Pulmonary

- Cystic fibrosis
- Bronchiectasis
- Tuberculosis
- Trauma during intubation, endoscopy or endobronchial surgery
- Related to tracheostomy
- Arteriovenous fistula
- Tumors

Hematologic

- Upper airway bleeding
- Disorders of coagulation
- Disseminated intravascular coagulopathy
- Thrombocytopenia
- Uremia
- Platelet dysfunction

Traumatic

- Aortic aneurysm
- Ruptured bronchus
- Chest injury
- Foreign body aspiration
- Tracheal-innominate artery fistula

Iatrogenic

- Bronchoscopy
- Lung biopsy
- Pulmonary artery catheterization
- Endobronchial brachytherapy
- Pulmonary hypertension

Alveolar Hemorrhage Syndromes

- Antiphospholipid syndrome
- Bechet syndrome
- Goodpasture syndrome
- Henoch Schonlein purpura
- Systemic lupus erythematosus
- Wegener's granulomatosis
- Hematemesis and aspiration into the airway
- Idiopathic pulmonary hemosiderosis

Cryptogenic**Immediate Management**

- Increase FiO_2 to 100%.
- Ensure that adequate supplies of blood products are available. In the setting of exsanguinating hemoptysis, consider activating the massive transfusion protocol.
- Initiate aggressive resuscitation with IV fluids.
- Support blood pressure as needed with ephedrine (5 mg IV) or phenylephrine (100 mcg IV) boluses. If refractory, consider phenylephrine or epinephrine infusion.
- Control the airway—intubate if ETT is not in place.
- Use a single lumen ETT in order to facilitate evacuation of blood from the airway, bronchial lavage, and fiberoptic bronchoscopy.
- Convert to a double lumen endotrachealtube (DLT) or use of a bronchial blocker (BB) to isolate the lungs if indicated.
- In an emergency when a DLT or a BB is not available, it is possible to push the ETT into a mainstem bronchus on the nonbleeding side to achieve long isolation.
- Rigid bronchoscopy facilitates identification of site of bleeding and may offer an opportunity for immediate treatment by cauterization, ablation or submucosal injection of vasoconstrictors.
- Consider ECMO as a bridge to definitive treatment.

Table 1.1 Comparing Bronchial Blockers to DLTs

	Bronchial Blocker	Double Lumen Tube
Age	Applicable at all ages	Not applicable in children
Difficult Intubation	Easier	Difficult to position
Positioning	Difficult	Relatively easier
Time to isolation	Longer	Quick
Selective lobar collapse	Possible	Not possible
Airway resistance with one-lung ventilation	Lower	Higher
Migration during surgery	Common	Uncommon
Suction and clearing of secretions	Difficult distal to balloon	Easier to accomplish
Deflation of lung	Takes longer	Easy to accomplish
Repeated inflation and deflations	Difficult	Easy to accomplish
Repositioning during pneumonectomy	Required	Not required
Interrupt ventilation during resection	Needed	Not needed
Risk of inclusion in suture line	Possible	Unlikely
Dislodgement during manipulations	Possible	More difficult

Adapted with permission from Lee KG, *Anaesthesia and Intensive Care Medicine* 2008; 9(12):538–541.

Diagnostic Studies

- Coagulation profile (PT/INR/aPTT etc.)
- Rigid/Fiberoptic bronchoscopy
- Chest X-Ray
- CT scan of the chest and neck as indicated
- Bronchial arteriogram
- ECHO to R/O cardiac origin
- Right heart catheterization

Subsequent Management

- Correct any coagulation defects. (See Coagulopathy in Chapter 5, page 93.)
- Recombinant activated factor VII is sometimes used with diffuse alveolar bleeding.
- Treat the primary cause of bleeding.

- Bronchial artery embolization is effective in the management of life-threatening massive hemoptysis.
- Surgery is indicated when bronchial artery embolization is not feasible. This could be a pneumonectomy, lobectomy, bilobectomy or a segmental wedge resection.
- Adopt a multidisciplinary approach to management of life-threatening massive intrapulmonary hemorrhage and hemoptysis.

Further Reading

Shigemura N, et al. Multidisciplinary management of life-threatening massive hemoptysis: a 10-year experience. *Ann Thorac Surg.* 2009;87(3):849–853.

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Godfrey S. Pulmonary hemorrhage/hemoptysis in children. *Pediatr Pulmonol.* 2004;37:476–484.

Seijo LM, Daniel H, Sterman DH. Interventional pulmonology. *N Engl J Med.* 2001;344(10):740–749.

Bronchial Intubation

13

Definition

Inadvertent endobronchial intubation (usually into right mainstem bronchus) occurs commonly. Early detection and correction avoids potential complications.

Presentation

- Hypoxemia caused by one-lung ventilation
- Increased airway pressure
- Unilateral breath sounds
- Atelectasis on CXR

Pathophysiology

The average distance from the larynx to the carina is 12–14 cm and changes with age, height, and head position. The tube moves cephalad when the neck is extended and caudad when the neck is flexed and can move as much as 5 cm with maximal cervical range of motion. Small changes in head position can cause endobronchial migration of the ETT in infants and small children. One-lung ventilation as a result of endobronchial ETT placement can cause atelectasis and hypoxemia.

Diagnostic Studies

- Chest X-ray
- Fiberoptic bronchoscopy
- Auscultation

Prevention

- Observe the endotracheal tube passing through the glottis and ensure that the upper end of the cuff is no more than 3–4 cm beyond the glottis.
- As a general rule, do not pass the endotracheal tube more than 21 cm in women and 23 cm in men (measured at the teeth).
- Ensure equal bilateral breath sounds.
- Chest radiography shows the tip of the tube overlying the 3rd or 4th thoracic vertebral body.
- Fiberoptic endoscopy should show the tip of the endotracheal tube 5–7 cm above the carina.

Further Reading

Owen RL, Cheney FW. Endobronchial intubation: a preventable complication. *Anesthesiology*. 1987;67(2):255–257.

Verghese ST, et al. Auscultation of bilateral breath sounds does not rule out endobronchial intubation in children. *Anesth Analg*. 2004;99(1):56–58.

Cannot Intubate/Can Ventilate

Definition

Inability to intubate the patient. Adequate face mask ventilation is still possible.

Immediate Management

- **Call for help.**
- Ensure FiO_2 is 100%.
- Consider inserting a supraglottic airway (e.g., Laryngeal Mask Airway) if appropriate.
- If a supraglottic airway is not feasible, and if another attempt at intubation is not appropriate, awaken the patient. Consider deferring the surgery or proceeding with awake intubation.
- If ventilation becomes difficult, proceed to “Cannot Intubate/ Cannot Ventilate” (Page 16)
- If the patient becomes hypoxic and cannot be ventilated, consider a surgical airway. (i.e., cricothyrotomy)
- If another attempt at intubation is warranted, consider the options listed in Box 1.1.
- Refer to the ASA Difficult Airway Algorithm, Inside Front Cover

Box 1.1 When an Initial Attempt at Intubation Fails

Place the patient's head in an optimal position (e.g., head in "sniffing position," use of pillows or towels to "ramp up" obese patients).

Make a decision as to how to approach the next attempt.

- External laryngeal manipulation
- A larger blade (e.g., MAC 4)
- Use of a straight blade (e.g., Miller)
- Consider a video laryngoscope such as the GlideScope
- Consider a Gum Elastic Bougie (Eschman stylet)
- Is asleep fiberoptic intubation a practical option?

Alternatives to consider in this setting include the following:

- Use a Trachlight
- Insert an intubating LMA (Fastrach) and then an ETT via the intubating LMA (See intubating Laryngeal Mask Airway, Page 282)
- Insert an LMA ProSeal or a Combitube to allow application of high ventilatory pressures and to help prevent aspiration
- Use a Syracuse-Patil face mask to facilitate fiberoptic intubation (keeping patient asleep)

As a general rule, awaken the patient after three unsuccessful attempts and proceed with intubation using an FOB technique or other method (e.g., retrograde intubation technique) while the patient breathes spontaneously.

Subsequent Management

- Document the intraoperative events carefully, with special attention to those techniques that were successful.
- Explain the sequence of events to the patient and advise him or her to warn future anesthesia providers.
- Write the patient a "difficult airway" letter. Instruct the patient to obtain a "difficult airway" bracelet.

Further Reading

Henderson J. Airway Management in the Adult. In: Miller RD, ed. *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1573–1610.

American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report. *Anesthesiology* 2003;98:1269–1277. Available at: <http://www.asahq.org/publicationsAndServices/Difficult%20Airway.pdf> Accessed: February 25, 2010.

Cannot Intubate/Cannot Ventilate

Definition

Inability to intubate the trachea. Inability to ventilate the patient by facemask.

Immediate Management

- **Call for help. A surgical airway may become necessary.**
- Ensure FiO_2 is 100%.
- Call for the difficult airway cart.
- Reposition the patient's head and jaw.
- Treat suspected laryngospasm with propofol 0.25–0.8 mg/kg or succinylcholine 0.1–2 mg/kg.
- Insert an airway of some kind (oropharyngeal, nasopharyngeal, supraglottic etc.) In many such cases the airway can be rescued with insertion of a supraglottic airway.
- Consider two-person ventilation. One person holds the mask in position and delivers a jaw thrust using both hands while the other ventilates the patient by hand using the reservoir bag and the emergency O_2 flush as needed.
- If oxygenation remains satisfactory, consider the use of a video laryngoscope (e.g., GlideScope) or fiberoptic intubation.
- Consider a surgical airway (e.g., cricothyrotomy) or transtracheal jet ventilation (TTJV). See Figure 1.1.
- Refer to the ASA Difficult Airway Algorithm, Inside Front Cover



Figure 1.1 A 14g needle with catheter (with a 10 mL syringe filled with saline) is inserted into the trachea through the cricothyroid membrane.

Subsequent Management

- Document the intraoperative events carefully, with special attention to those techniques that were successful.
- Explain the sequence of events to the patient and advise him or her to warn future anesthesia providers.
- Write the patient a “difficult airway” letter and instruct him or her to obtain a “difficult airway” bracelet.
- If a surgical airway has been attempted, request an emergency ENT consultation.

Special Considerations

Ensure that extra help is available before starting an anesthetic when airway management may be difficult. Be certain that the patient is adequately preoxygenated. Position the patient’s head correctly (“sniffing position,” use of pillows or towels to “ramp up” obese patients). Have special intubation equipment (e.g., GlideScope, McGrath, FOB, etc.) readily at hand.

Further Reading

- Henderson J. Airway Management in the Adult. In: Miller RD, ed. *Miller’s Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1573–1610.
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- Hsiao J, Pacheco-Fowler V. Videos in clinical medicine. Cricothyroidotomy. *N Engl J Med*. 2008;358(22):e25.
- Scrase I, Woollard M. Needle vs surgical cricothyroidotomy: a short cut to effective ventilation. *Anaesthesia*. 2006;61(10):962–974.

Difficult Mask Ventilation

Definition

Inability to provide adequate face mask ventilation due to inadequate mask seal, excessive gas leak, or excessive resistance to the gas flow.

Presentation

Absent or inadequate chest movement or breath sounds, signs of severe obstruction, cyanosis, gastric air entry or dilatation, hypoxemia,

absent or inadequate exhaled CO_2 , or low gas flows on spirometry (reservoir bag does not fill).

Pathophysiology

There are five independent predictors for difficult face mask ventilation: (1) age over 55 years; (2) body mass index (BMI) over 26 kg/m^2 ; (3) presence of a beard; (4) edentulous patient; and (5) history of snoring.

Immediate Management

- Increase FiO_2 to 100%.
- Administer a jaw thrust.
- Ensure that an appropriately sized face mask is in use.
- Consider the use of two-person ventilation, with one person using both hands to get a good face mask fit and with the second person doing the ventilation.
- Consider the use of an oral airway or a nasopharyngeal airway.
- Consider inserting a supraglottic airway, such as the laryngeal mask airway (LMA).
- Consider tracheal intubation.

Further Reading

American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report. *Anesthesiology* 2003; 98:1269–1277. Available at: <http://www.asahq.org/publicationsAndServices/Difficult%20Airway.pdf>

El-Orbany M, Woehlick HJ. Difficult mask ventilation. *Anesth Analg*. 2009 Dec;109(6):1870–1880.

Langeron O, Masso E, Huraux C, Guggiari M, Bianchi A, Coriat P, Riou B. Prediction of difficult mask ventilation. *Anesthesiology*. 2000;92:1229–1236.

Difficult Ventilation through an Endotracheal Tube

Definition

High airway pressure and/or inability to ventilate well with a tracheal tube *in situ*.

Presentation may include

- Stiff rebreathing bag during manual ventilation
- High airway pressure during manual or mechanical ventilation
- Hypercarbia/elevated end-tidal CO_2 levels

DIFFERENTIAL DIAGNOSIS

- Kinked endotracheal tube
- Blood or secretions occluding endotracheal tube
- Bronchospasm
- Pneumothorax
- Anaphylaxis
- Endobronchial intubation
- Other causes (ARDS, pulmonary edema etc.)

Immediate Management

- Administer 100% oxygen.
- Auscultate the lung fields to rule out wheezes, crackles and unilateral ventilation.
- Pass a suction catheter down ETT to rule out kinking or the presence of blood or secretions.
- To rule out a problem with the anesthesia machine and patient breathing circuit, check that ventilation with a self-inflating bag (“Ambu bag”) is satisfactory.
- Examine the patient for signs of anaphylaxis (erythema, urticaria, hypotension, tachycardia, etc.).

Diagnostic Studies

- Auscultation of lung fields
- CXR
- FOB

Subsequent Management

Will depend on the nature of the underlying problem.

Further Reading

Henderson J. Airway Management in the Adult. In: Miller RD, ed. *Miller's Anesthesia*. 7th ed. Philadelphia, Elsevier Churchill Livingstone; 2010:1573–1610.

Laryngospasm

Definition

Reflex closure of the upper airway due to spasm of the glottic musculature.

Presentation may include

- Difficult or impossible face mask ventilation
- Difficult or impossible ventilation with supraglottic airway
- “Crowing” sound on inspiration.

Pathophysiology

Laryngospasm is especially common in children and is associated with light planes of anesthesia and the presence of foreign matter (e.g., blood or secretions) irritating the vocal cords and nearby structures.

Differential Diagnosis

- Bronchospasm
- Stridor
- Foreign body in the airway
- Airway obstruction from edema, infection, tumor, hematoma, etc.

Immediate Management

- Administer 100% oxygen with positive pressure ventilation.
- Administer propofol or other drugs to deepen the level of anesthesia.
- Consider succinylcholine 0.1 mg/kg IV.

Special Considerations

- Failure to rapidly diagnose and treat laryngospasm can quickly produce hypoxemia and hypercarbia.
- Patients who generate high negative inspiratory pressures in attempting to breathe against the obstruction may develop negative-pressure pulmonary edema.

Further Reading

Tsui BC, Wagner A, Cave D, et al. The incidence of laryngospasm with a “no touch” extubation technique after tonsillectomy and adenoidectomy. *Anesth Analg*. 2004;98:327–329.

Visvanathan T, Kluger MT, Webb RK, Westhorpe RN. Crisis management during anaesthesia: laryngospasm. *Qual Saf Health Care*. 2005 June;14(3):e3.

Ludwig’s Angina

Definition

Ludwig’s angina is a multispace infection of the floor of the mouth. The infection usually starts with infected mandibular molars and

spreads to sublingual, submental, buccal, and submandibular spaces.

Presentation

- Edema and distortion of airway structures
- Signs of airway obstruction, such as the use of the accessory muscles of respiration
- Dyspnea
- Drooling
- Fever
- Leukocytosis

Pathophysiology

The tongue becomes elevated and displaced posteriorly, which may lead to loss of the airway, especially when the patient is placed in the supine position.

DIFFERENTIAL DIAGNOSIS

- Retropharyngeal abscess
- Submandibular abscess
- Epiglottitis
- Dental abscess

Immediate Management

- Airway management depends on clinical severity, surgical preferences, and other factors (e.g., CT scan or MRI findings).
- If the clinical situation permits, transport the patient to the operating room for airway management.
- A skilled surgeon and emergency cricothyroidotomy equipment must be present.
- Consider awake fiberoptic intubation if at all possible (see Page 279), but elective tracheostomy prior to incision and drainage may be necessary in the setting of significant airway compromise.

Subsequent Management

- Initiate antibiotic therapy, either empirical or based on culture and sensitivity testing.
- Transfer the patient to the ICU.

Special Considerations

- Loss of the airway is the leading cause of death in patients with Ludwig's angina.

- Extubation may be hazardous. Special precautions, such as the use of a tube exchange catheter, may be appropriate.
- The abscess may rupture spontaneously or after attempts at laryngoscopy and intubation, flooding the hypopharynx with pus. This may result in lung soiling.

Further Reading

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;100:585–589.

Boscolo-Rizzo P, Da Mosto MC. Submandibular space infection: a potentially lethal infection. *Int J Infect Dis*. 2009;13(3):327–333.

Marple BF. Ludwig angina: a review of current airway management. *Arch Otolaryngol Head Neck Surg*. 1999;125(5):596–599.

Rapid-Sequence Intubation (RSI)

Definition

A technique of inducing general anesthesia so as to reduce the risk of pulmonary aspiration of gastric contents. An induction agent (e.g., propofol or etomidate) is administered, immediately followed by a neuromuscular blocking agent (e.g., succinylcholine or rocuronium) to quickly render the patient unconscious and facilitate airway management. Pressure is applied to the cricoid area (Sellick's maneuver) to compress the esophagus, reducing the risk of regurgitation.

Indications

- Emergency surgery in which a patient has not been fasting
- Patients with paralytic ileus or acute abdomen
- Patients with significant reflux or achalasia cardia
- Patients with acute trauma requiring immediate surgery
- Women presenting for surgery in the last trimester of pregnancy

Contraindications

- Patients with anticipated difficult airway
- Situations where laryngeal injury may be present

Consider awake intubation in these situations.

The Rapid Sequence Induction (“The 9 Ps”)

Preparation

Prepare all necessary equipment and drugs, and have a backup plan.

- A working laryngoscope and different types of blades. A GlideScope or other video laryngoscope may be desirable if a difficult airway is anticipated.

- Styletted ETTs of the desired size.
- Device to confirm proper placement of ETT (e.g., capnograph)
- Equipment for emergency tracheotomy/cricothyrotomy where appropriate.
- Establish IV access.
- Attach appropriate monitoring equipment

Patient Evaluation

- Evaluate the airway to rule out possible difficult intubation.
- Ensure that ventilation by facemask will not be difficult.
- Review possible contraindications to medications.

Preoxygenation

Administer 100% oxygen for 3–5 minutes with a tight seal around the mask. If the patient is cooperative, five vital-capacity breaths are nearly as effective.

Premedication

Should be used judiciously. These medications are used to prevent the adverse effects of endotracheal intubation.

- Midazolam: 0.02–0.05 mg/kg. Use with caution in patients with head injury or who may need to be rapidly awakened.
- Fentanyl: 3 mcg/kg IV 2–3 minutes prior
- Lidocaine: 1.5 mg/kg IV 2–3 minutes prior

Paralysis and Induction

Rapidly administer an anesthetic followed by a neuromuscular blocking agent. Do not titrate medication to effect.

- Choose an induction agent:
 - Etomidate: 0.3 mg/kg IV
 - Ketamine: 1–2 mg/Kg IV
 - Propofol: 1–2 mg/Kg IV
 - Thiopental: 3–5 mg/kg IV
- Choose a neuromuscular blocking agent:
 - Succinylcholine: 1–2 mg/kg
 - Rocuronium: 1–1.2 mg/kg

Position and Protect the Patient

- Position the head and neck into the sniffing position by flexing the neck and extending the atlanto-occipital joint. Reposition the head if an adequate view of the glottic opening is not achieved.
- Apply cricoid pressure (Sellick's maneuver) after the patient has gone to sleep. (Do not release the cricoid pressure until correct ETT position is confirmed.)
- Wait for 45–60 seconds to allow full effect of the muscle relaxant.

- If the patient will tolerate apnea, do not ventilate the patient at this time to prevent gaseous distension of the stomach.

Pass the ETT

Visualize the tube going through the vocal cords.

Proof of Placement

Establish that the ETT is in the correct position by clinical and capnographic means.

Post-intubation Care

Secure the tube, ventilate.

Further Reading

Reynolds SF, Heffner J. Airway management of the critically ill patient: rapid-sequence intubation. *Chest*. 2005;127(4):1397–412.

Langeron O, Birenbaum A, Amour J. Airway management in trauma. *Minerva Anesthesiol*. 2009 May;75(5):307–311.

Chapter 2

Cardiovascular Emergencies

Ajoy Katari and Benjamin A. Kohl

- Asystole 26
- Cardiac Tamponade 27
- Pathophysiology 28
- Cardiac Trauma 29
- Congestive Heart Failure 31
- Dysrhythmias: Atrial Fibrillation 33
- Dysrhythmias: Bradycardia 35
- Dysrhythmias: Narrow Complex Tachycardia 37
- Dysrhythmias: Wide Complex Tachycardia 39
- Hypertension 41
- Hypotension 43
- Gas Embolism 45
- Myocardial Ischemia 47
- Postoperative Hemorrhage 49
- Valvular Disease: Aortic Regurgitation 51
- Valvular Disease: Aortic Stenosis 53
- Valvular Disease: Mitral Regurgitation 55
- Valvular Disease: Mitral Stenosis 56
- Thoracic Aortic Dissection 58
- Ventricular Fibrillation 60

Asystole

Definition

Complete absence of electrical and/or mechanical cardiac activity.

Presentation

Usually preceded by other arrhythmias. Vagal stimulation, caused by insufflation of the abdominal cavity for laparoscopic surgery or excessive intraocular pressure, can sometimes be the initial trigger.

Pathophysiology

Primary asystole has a poor prognosis. It is usually secondary to other conditions such as hypoxia, hyper- or hypokalemia, acidosis, or myocardial infarction.

DIFFERENTIAL DIAGNOSIS

- Monitor lead disconnection
- Severe bradycardia
- Ventricular fibrillation
- Low-voltage ECG

Immediate Management

- Confirm asystole in more than 1 ECG lead
- Begin CPR
- Establish an airway (endotracheal intubation preferred)
- Epinephrine (1 mg IV every 3–5 minutes)
- Vasopressin (40 U within the first 10 minutes of CPR)
- Atropine (1 mg every 3–5 minutes for three doses)

Diagnostic Studies

- Electrocardiogram
- Echocardiogram

Subsequent Management

- Treat underlying causes (hypoxia, acidosis, etc.)
- Sodium bicarbonate as necessary to correct metabolic acidosis
- Temporary transcutaneous or transvenous cardiac pacing

Risk Factors

- Hypoxia
- Electrolyte imbalances (e.g., hyperkalemia)
- Hypovolemia

Risk Factors (continued)

- Hypothermia
- Ocular surgery
- Hypersensitive carotid sinus
- Other underlying cardiac conduction abnormalities

Prevention

Early identification of underlying conditions such as hypoxia, acidosis, and ensuing arrhythmias.

Special Considerations

Transcutaneous pacing has not been shown to favorably affect survival unless instituted early.

Several factors must be considered before terminating resuscitation efforts. The most commonly used parameters include:

- Resuscitation for more than 30 minutes without a sustained, perfusing rhythm
- Asystole as an initial finding
- Advanced age with severe comorbid disease
- Ensure that the patient is normothermic before resuscitation is terminated.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.2: Management of cardiac arrest. *Circulation*. 2005;112:IV-58-IV-66.

Cardiac Tamponade**Definition**

Acute accumulation of fluid in the pericardial space. (Normal volume is 25–50 mL.) Gradual accumulation of this fluid allows time for compensation and may not result in symptomatic tamponade.

Presentation

- Dyspnea
- Orthopnea
- Tachycardia
- Jugular venous distention
- Distant heart sounds
- Pulsus paradoxus (Systolic drop of 10 mm Hg with inspiration)

- Beck's triad (small, quiet heart, rising venous pressure, falling arterial pressure)

Pathophysiology

External pressure on the heart reduces the ventricular preload.

DIFFERENTIAL DIAGNOSIS

(Some of these conditions can also cause pericardial tamponade)

- Acute myocardial infarction
- Postoperative bleeding
- Aortic dissection
- Iatrogenic (e.g., catheter insertion)
- Connective tissue disorders
- Uremia
- Positive end expiratory pressure ("auto-peep")

Immediate Management

- In the setting of profound hypotension, emergency pericardiocentesis may be necessary.
- Consider IV fluid administration to increase preload.
- Maintain heart rate to maintain cardiac output.
- Maintain normal sinus rhythm.

Diagnostic Studies

- Electrical alternans on ECG (variation of R-wave axis with alternate beats)
- Globular heart on chest radiography
- Accumulation of fluid on echocardiography
- Equalization of diastolic pressures in all four chambers of heart

Subsequent Management

- Request a surgical pericardiectomy.
- Administer ketamine for induction of general anesthesia (may help to maintain heart rate and blood pressure).
- Maintain spontaneous ventilation until the pericardial sac is opened.

- Consider low tidal volumes with high ventilation rate to minimize intrathoracic pressure.
- Keep heart rate high.
- Maintain adequate preload.

Risk Factors

- Trauma
- Myocardial infarction
- Connective tissue disorders
- Uremia

Prevention

- Control of uremia
- Early recognition and control of cardiac surgical bleeding
- Awareness of risk of cardiac injury during pacemaker electrode implantation or removal
- Early recognition and intervention are critical to successful management.

Special Considerations

- Pneumothorax and cardiac perforation may occur during cardiocentesis.
- Pulmonary edema and global systolic dysfunction may occur after relief of tamponade

Further Reading

Soler-Soler J, et al. Management of pericardial effusion. *Heart*. 2001;86:235–240.

Cardiac Trauma

Definition

Penetrating or blunt injury to the myocardium.

Presentation

- Dyspnea
- Tachycardia
- Chest pain
- Flail chest

Pathophysiology

Severe blunt injury due to high impact force is usually required to cause thoracic organ injury.

DIFFERENTIAL DIAGNOSIS

- Pulmonary and myocardial contusion
- Pneumothorax
- Esophageal disruption
- Myocardial laceration
- Cardiac tamponade
- Dissection of the thoracic aorta
- Coronary laceration
- Diaphragmatic injury

Immediate Management

- Assess airway, breathing, and circulation.
- Provide supplemental O₂ as required to maintain oxygenation.
- Request a surgical consultation for chest tube insertion and/or pericardiocentesis.
- Emergency surgery, possibly requiring cardiopulmonary bypass, may be necessary.
- Supportive management.

Diagnostic Studies

- Chest radiography
- CT scan
- Bronchoscopy
- Esophagoscopy
- Transesophageal echocardiography (TEE)

Subsequent Management

- Request a surgical consultation for possible chest exploration.
- Evaluate the tracheobronchial tree to rule out concomitant lung injury.
- Use lung isolation techniques as required to optimize ventilation.

Risk Factors

- Trauma

Prevention

Although cardiac trauma is not truly a preventable phenomenon, early recognition and diagnosis of underlying pathology followed by timely intervention is key.

Special Considerations

Nitrous oxide should be avoided in patients undergoing trauma surgery because an undiagnosed pneumothorax may be present. Be alert for impaired ventilation of the dependent lung secondary to a tension pneumothorax during one-lung ventilation. Some degree of cardiac involvement should be anticipated in blunt injury, so the ECG should be monitored continuously in the postoperative period.

Further Reading

Bastos R, et al. Penetrating thoracic trauma. *Semin Thorac Cardiovasc Surg.* 2008;20:19–25.

Sybrandy KC, et al. Diagnosing cardiac contusion: old wisdom and new insights. *Heart.* 2003;89:485–489.

Congestive Heart Failure

Definition

A structural or a functional cardiac disorder with impaired ability of the ventricle to fill with (diastolic) or eject (systolic) blood.

New York Heart Association (NYHA) classification of severity:

- Class I: symptoms of heart failure (HF) only at activity levels that would limit normal individuals
- Class II: symptoms of HF with ordinary exertion
- Class III: symptoms of HF with less than ordinary exertion
- Class IV: symptoms of HF at rest

Presentation

- Dyspnea
- Fatigue
- Edema

Pathophysiology

For systolic HF, about 50% of cases are idiopathic. Other causes include myocarditis, ischemic heart disease, infiltrative disease (amyloidosis), peripartum cardiomyopathy, hypertension, HIV infection, connective tissue disease, substance abuse, drugs (e.g., doxorubicin). Diastolic HF risk factors include hypertrophic and restrictive cardiomyopathies and are otherwise similar to systolic HF.

DIFFERENTIAL DIAGNOSIS

- Myocardial ischemia
- Primary pulmonary pathology
- Cardiomyopathy

Immediate Management

- Increase FiO_2 to maintain oxygenation.
- Administer a loop diuretic (furosemide 20–40 mg IV)
- Consider an ACE inhibitor (enalapril 2.5 mg IV every 6 hours)
- Consider nitroglycerine (infusion starting at 0.5 mcg/kg/min, increase every 3–5 minutes to desired effect)
- Consider nesiritide (2 mcg/kg IV bolus, then 0.01 mcg/kg/min to a maximum of 0.03 mcg/kg/min)
- Consider beta-blockers (esmolol 500 mcg/kg over 1 minute, then 50 mcg/kg/min to a maximum of 300 mcg/kg/min)
- Consider digoxin 0.125–0.25 mg IV
- If the patient is awake and can take oral medication, consider angiotensin II receptor blockers (candesartan)
- Aldosterone antagonist (spironolactone)

Diagnostic Studies

- Chest X-ray shows cardiomegaly, Kerley B lines, pleural effusions
- ECG to evaluate for ischemic or hypertrophic changes
- Echocardiogram to evaluate cardiac function
- Serum brain natriuretic peptide (BNP) (Elevated in heart failure)

Subsequent Management

- Correction of contributing systemic disease
- Lifestyle modification
- Discontinue drugs implicated in HF

Specialized management for HF that is refractory to pharmacologic therapy:

- Intra-aortic balloon pump
- Left ventricular assist devices
- Heart transplantation

Risk Factors

- Coronary artery disease
- Smoking
- Hypertension
- Obesity
- Valvular heart disease

Prevention

Careful fluid management in the patient at risk for congestive heart failure

Special Considerations

Implantable cardiac defibrillators are used to detect and treat arrhythmias associated with heart failure, and may also be used for cardiac resynchronization therapy via biventricular pacing.

Cardiac wraps have been used to prevent further deterioration of heart function by preventing further dilation of the ventricles.

Further Reading

Hunt, SA, et al. 2009 focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, developed in collaboration with the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol.* 2009;53(15):e1–e90.

Dysrhythmias: Atrial Fibrillation

Definition

An irregularly irregular heart rhythm with the absence of P waves on ECG. This is the most common cardiac arrhythmia.

Presentation

- May be asymptomatic
- Palpitations
- Chest pain/angina
- Congestive heart failure
- Syncope
- Transient ischemic attacks

Pathophysiology

Atrial fibrillation is often seen in patients with otherwise normal hearts. It is also associated with excessive alcohol consumption, hypertension, hyperthyroidism, and myocardial ischemia. Atrial dilatation and fibrosis are the primary changes observed.

DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia
- Multifocal atrial tachycardia
- Reentrant tachycardia
- Junctional tachycardia
- Atrial flutter

Immediate Management

- DC cardioversion if the patient is hemodynamically unstable

(Rate Control)

- Administer beta blocking drugs (esmolol 500 mcg/kg over 1 minute, then 50 mcg/kg/min to a maximum of 300 mcg/kg/min)
- Calcium channel blockers (diltiazem)
- Amiodarone (150 mg IV over 10 minutes, then 1 mg/min IV for 6 hours, then 0.5 mg/min for 18 hours.) **Be alert for hypotension when administering a bolus dose.**
- Digoxin 0.125–0.25 mcg IV

Diagnostic Studies

- ECG
- Electrophysiology study
- Echocardiogram
- Holter monitoring

Subsequent Management

Rhythm Control

- Cardioversion (electrical or chemical)
- Amiodarone in resistant cases
- Electrophysiology and cryoablation
- Surgical ablation

Risk Control (Stroke)

Anticoagulation with heparin and warfarin

Risk Factors

- Age (8% of people over 80)
- Recent cardiothoracic surgery (particularly valvular surgery)
- Atrial dilatation
- Hyperthyroidism
- Male > Female
- Smoking
- Alcohol consumption
- Coronary artery disease
- Stress

Prevention

- Avoid acute beta-blocker discontinuation perioperatively.

- Maintain normal electrolytes (particularly potassium and magnesium).
- Reduce stress.

Special Considerations

- Atrial fibrillation increases the risk of stroke sevenfold.
- Cardioversion should only be performed for new-onset atrial fibrillation within less than 48 hours of onset, or if a transesophageal echocardiogram (TEE) shows no evidence of thrombus.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation*. 2005;112:IV-67–IV-77).

Echahidi N. Mechanisms, prevention, and treatment of atrial fibrillation after cardiac surgery. *J Am Coll Cardiol*. 2008;51:793–801.

National Institute for Health and Clinical Excellence. Guidelines for atrial fibrillation. Available at: <http://www.nice.org.uk/nicemedia/pdf/CG036niceguideline.pdf>. Accessed August 9, 2009.

Dysrhythmias: Bradycardia

Definition

A heart rate less than 60 beats per minute.

Presentation

- Hypotension
- Nausea
- Altered mental status
- Pulmonary edema
- Chest pain

Pathophysiology

May be secondary to cardiac ischemia, atrioventricular node disease, hypoxemia, acidosis, or drugs (e.g., narcotics, beta-blockers)

A sick sinus node or conduction abnormalities may cause bradycardia. The above presentations are suggestive of organ malperfusion.

DIFFERENTIAL DIAGNOSIS

- Excessive beta or calcium channel blocker therapy
- Excessive vagal stimulation
- High dose narcotic administration
- Hypersensitivity of carotid sinus

- Excessive intraocular pressure
- Elevated systemic vascular resistance
- Hypoxia
- Acidosis
- Cardiac tamponade
- Electrolyte abnormalities

Immediate Management

- Remove possible causes. (E.g., during laparoscopic surgery, ask the surgeons to reduce intra-abdominal pressure.)
- Initiate immediate transcutaneous pacing if there is evidence of tissue hypoperfusion.
- Administer atropine while preparing for pacing (0.5 mg IV, repeat every 5 minutes to a total dose of 3 mg).

Diagnostic Studies

- ECG
- Echocardiogram
- Angiogram (rule out acute myocardial infarction as cause)

Subsequent Management

- Transvenous pacing if transcutaneous pacing fails to capture
- Epinephrine (2–10 micrograms/min) or dopamine (2–10 micrograms/kg/min) infusion
- Permanent pacemaker placement

Risk Factors

- Pharmacologic (beta-blockers, calcium channel blockers, narcotics)
- Alpha-agonists
- Ocular surgery
- Surgery near carotid sinus (e.g., carotid endarterectomy)
- Laparoscopic surgery
- Age
- Elevated blood pressure
- Elevated cholesterol
- Smoking
- Heavy alcohol consumption
- Use of recreational drugs
- Psychological stress or anxiety

Prevention

- Maintain normal plasma electrolyte levels.
- Inject the carotid sinus with lidocaine during carotid endarterectomy.
- Release intraocular or intra-abdominal pressure.
- Exercise cautious use of alpha-agonist agents.

Special Considerations

Atropine works at the atrioventricular node and is unlikely to be effective in heart transplant patients or for bradycardia due to a block below the bundle of His. Glucagon 3 mg IV followed by an infusion at 3 mg/h can be used if bradycardia is attributed to beta-blocker therapy.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation*. 2005;112:IV-67–IV-77).

Dysrhythmias: Narrow Complex Tachycardia

Definition

Heart rate > 100 beats per minute (may be regular or irregular) with a narrow QRS complex (<120 msec)

Presentation

- Hypotension
- Palpitations
- Altered mental status
- Chest pain
- Pulmonary edema

Pathophysiology

A narrow QRS complex implies a rapid activation of the ventricles via the normal His-Purkinje system. This suggests that the arrhythmia originates within or above the AV node (supraventricular). Narrow QRS complex tachycardia is most commonly sinus tachycardia, or due to a reentry phenomenon.

DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia
- Atrial tachycardia

- Multifocal atrial tachycardia
- Reentrant tachycardia (e.g., Wolff-Parkinson-White syndrome)
- Junctional tachycardia
- Atrial fibrillation
- Atrial flutter

Immediate Management

- Administer adenosine (6 mg rapid IV push, repeat 12 mg \times 2).
- Stable regular narrow complex tachycardia: treat for underlying cause (fever, anemia, shock, sepsis, pain, etc.).
- Stable, irregular, narrow complex tachycardias: control heart rate with diltiazem (15 mg IV over 20 minutes) or metoprolol (5 mg IV q 5 minutes).
- Consider DC cardioversion if hemodynamically unstable.
- Treat unstable arrhythmias with immediate electrical cardioversion.

Diagnostic Studies

- ECG
- Electrophysiology testing
- Echocardiography

Subsequent Management

- If adenosine fails, initiate rate control with either intravenous calcium-channel blockers or beta-blockers.
- Chemical cardioversion: Administer procainamide (50 mg/min IV, up to a dose of 18–20 mg/kg) or amiodarone (5 mg/kg IV over 15 minutes).
- Use electrical cardioversion for tachycardia resistant to pharmacological interventions and/or in patients who are hemodynamically unstable.

Risk Factors

- Fever
- Inadequate anesthesia
- Hypovolemia
- Myocardial ischemia
- Hyperthyroid
- Vagolytic drugs

Prevention

- Maintain normothermia.
- Volume resuscitation.
- Avoid beta-blocker discontinuation perioperatively.
- Avoid vagolytic drugs (e.g., atropine, pancuronium).

Special Considerations

- Regular narrow complex tachycardia is most often sinus tachycardia.
- Irregular narrow complex tachycardia may be atrial flutter, atrial fibrillation, or multifocal atrial tachycardia.
- Transient chest discomfort, dyspnea, and flushing may occur with adenosine administration.
- Consider the risk of embolic stroke before cardioversion.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation*. 2005;112:IV-67–IV-77).

Dysrhythmias: Wide Complex Tachycardia

Definition

Heart rate greater than 100 beats per minute (may be regular or irregular) with a wide QRS complex (>120 msec)

Presentation

- Hypotension
- Altered mental status
- Palpitations
- Chest pain

Pulmonary Edema Pathophysiology

A widened QRS complex implies slow ventricular activation. Commonly the arrhythmia originates outside of the normal conduction system, but may be ventricular or supraventricular. A regular, wide complex tachycardia is most often ventricular tachycardia.

DIFFERENTIAL DIAGNOSIS

Regular

- Ventricular tachycardia
- Supraventricular tachycardia with aberrant conduction
- Artifact

- Paced rhythm with atrial tachycardia (atrial sense, ventricular paced)

Irregular

- Atrial fibrillation with aberrancy (bundle branch block),
- Atrial fibrillation with preexcitation (e.g., Wolff-Parkinson-White syndrome)
- Polymorphic ventricular tachycardia

Immediate Management

- DC cardioversion if hemodynamically unstable

Regular Rhythm

- Amiodarone (150 mg IV given over 10 minutes, repeated as needed to a total of 2.2 g IV over the first 24 hours)

Irregular Rhythm

- Procainamide (20 mg/min IV up to a total of 17 mg/kg)
- Lidocaine (1 mg/kg IV, may repeat 0.5 mg/kg every 5 minutes as needed to total of 3 mg/kg)

Diagnostic Studies

- ECG
- Echocardiogram
- Electrophysiology testing

Subsequent Management

- Cardioversion for regular tachycardia resistant to pharmacological intervention, or if hemodynamically unstable.
- If the patient has a history of a preexcitation syndrome (e.g., Wolff-Parkinson-White syndrome), or evidence of preexcitation on the ECG (e.g., delta wave), procainamide is the preferred treatment (20 mg/min continuous infusion until the arrhythmia is suppressed, the patient is hypotensive, the QRS widens 50% beyond baseline, or a maximum dose of 17 mg/kg is administered).

Risk Factors

- Age > 50
- Myocardial ischemia (or old infarction)
- Known reentrant pathway

Prevention

- Maintain normal electrolytes (particularly potassium and magnesium).
- Prescribe anti-arrhythmic (Class I and III) drugs.
- Request an interventional cardiology consultation to perform a catheter ablation.
- Consider Implantable cardiac defibrillator (ICD).
- Change pacemaker to ventricular-paced, ventricular-sensing, inhibition (VVI) or atrial-paced, atrial-sensing, inhibition (AAI)
- Although amiodarone is an alternative treatment, beware of irregular wide complex dysrhythmia due to preexcitation converting to unstable ventricular tachycardia or ventricular fibrillation with the use of amiodarone.

Special Considerations

AV nodal blockers are contraindicated in wide complex, irregular tachycardias, especially when the etiology is unknown, as they may precipitate ventricular fibrillation and death.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation*. 2005;112:IV-67–IV-77).

Hypertension

Definition

A blood pressure greater than 160/100 on a series of measurements.

Presentation

- Chest pain
- Headache
- Palpitations
- Stroke
- Pulmonary edema

Pathophysiology

The pathophysiology of hypertension is often multifactorial. Anything that increases preload, afterload, or contractility can cause hypertension.

DIFFERENTIAL DIAGNOSIS

- Inadequate anesthetic depth
- Agitation

- Vasopressor error (inadvertent administration or overdose)
- Pheochromocytoma
- Thyrotoxicosis
- Aortic cross clamp
- Elevated intracranial pressure
- Transection of the spinal cord at or above T5
- Eclampsia in pregnancy
- Post electroconvulsive therapy

Immediate Management

- Increase depth of anesthesia
- Check for medication error
- Administer nicardipine: Start at 5 mg/h IV and increase by 2.5 mg/h every 5–15 minutes. Maximum dose is 15 mg/h.
- Administer sodium nitroprusside for acute, life-threatening hypertension: Start at 0.3 mcg/kg/min and increase slowly. Maximum dose is 10 mcg/kg/min.
- Administer beta-blockers (labetolol 5 mg q 2 minutes is preferred because it has both alpha- and beta-blocking properties)

Diagnostic Studies

- ECG
- Echocardiogram
- Angiogram
- Intra-arterial catheterization

Subsequent Management

- Long-acting beta-blockers
- Hydralazine ACE inhibitors or angiotensin receptor blockers
- Clonidine
- Anxiolytics

Risk Factors

- Age > 60
- Male > Female
- Race
- Weight
- Stress

Risk Factors (continued)

- Alcohol
- Smoking
- Family history
- Renal disease
- Diabetes
- Sleep apnea

Prevention

- Maintain adequate depth of anesthesia.
- Avoid rapid termination of antihypertensive medications.

Special Considerations

An acute rise in BP is of more significance than the actual numbers, but systolic pressure > 220 mm Hg and diastolic pressure > 120 mm Hg must be treated promptly.

Preexisting elevated blood pressure should be decreased by no more than 20–30% of baseline to compensate for a right shift of the autoregulation curve in patients with chronic hypertension.

Sodium nitroprusside is very rapid in onset and offset and can produce wide variations in blood pressure. Infusion rates above 2 mcg/kg/min result in accumulation of cyanide and can lead to cyanide toxicity. Consider cyanide toxicity in patients who become confused and develop unexplained metabolic acidosis and/or increased mixed venous PaO₂. Tachyphylaxis may also occur.

Further Reading

Laslett L. Hypertension: Preoperative assessment and perioperative management. *West J Med.* 1995;162:215–219.

Varon J. The diagnosis and treatment of hypertensive crisis. *Postgrad Med.* 2009;121:5–13.

Hypotension**Definition**

There are no universally accepted criteria. Commonly accepted definitions include:

- Systolic BP < 80 mm Hg
- Mean arterial pressure less than 60 mm Hg
- Decline of SBP or MAP by 20% from baseline

Presentation

- Chest pain
- Shortness of breath
- Nausea
- Altered mental status
- Oliguria

Pathophysiology

The pathophysiology is often multifactorial. Anything that decreases heart rate, preload, afterload, or contractility can cause hypotension.

DIFFERENTIAL DIAGNOSIS

- Deep anesthesia
- Hypovolemia
- Spinal/epidural anesthesia
- Pericardial tamponade
- Pneumothorax
- Acute heart failure
- Endocrine dysfunction
- Anaphylaxis

Immediate Management

- Administer IV fluid.
- Check for medication error.
- Administer ephedrine (5 mg IV bolus).
- Administer phenylephrine (100 mcg IV bolus).
- Administer epinephrine.
- Depending on suspected etiology, consider infusion of a vasopressor (e.g., phenylephrine) or an inotrope in cases of refractory hypotension.

Diagnostic Studies

- ECG
- Echocardiogram
- Angiogram
- Intra-arterial catheterization

Subsequent Management

- Establish a differential diagnosis and treat the underlying cause if known.
- Consider vasopressin infusion for catecholamine-resistant hypotension (recommended rate is 0.01–0.04 units/min).
- Administer a corticosteroid (hydrocortisone 100 mg IV) if adrenal insufficiency is suspected.

Risk Factors

- Dehydration
- Age > 65
- Beta-blocker or calcium channel blocker therapy
- Neuraxial analgesia
- Cardiac disease
- Medications

Prevention

- Provide adequate volume resuscitation accounting for preoperative and intraoperative fluid loss and third-spacing.
- Monitor the level of anesthesia in patients who have undergone neuraxial blockade.
- Administer vasodilating medications (e.g., phenytoin, protamine, vancomycin) slowly.

Special Considerations

Hypotension has many etiologies. Rapid differential diagnosis can narrow the therapeutic options and improve patient outcome.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.4: monitoring and medications. *Circulation*. 2005;112:IV-78–IV-83).

Gas Embolism

Definition

Gas (air) bubbles in the vascular system that disrupt the continuity of the blood stream.

Presentation

- Minor gas embolism is often asymptomatic
- Dyspnea
- Hypotension
- Tachypnea
- Tachycardia

Pathophysiology

Two components must be present for a gas embolism to occur: vascular access to air, and a pressure gradient between air and the vascular system.

DIFFERENTIAL DIAGNOSIS

- Acute anemia
- Stroke
- Angina
- COPD
- Cardiogenic shock

Immediate Management

- Increase FiO_2 to 100% to ensure adequate oxytation.
- Alert the surgeons and request that the field be flooded with saline solution.
- Support the blood pressure with fluids and vasopressors as required.
- Remove air from the right atrium if a central venous catheter is available.
- Place the patient left side down in Trendelenburg position if possible.
- Begin CPR if necessary.
- Provide supportive care until the episode resolves.

Diagnostic Studies

- Echocardiogram
- Precordial Doppler ultrasound
- Respiratory alkalosis, decreased pCO_2 , hypoxia

Subsequent Management

- Consider hyperbaric therapy in the face of massive gas embolism refractory to other treatment

Risk Factors

- Lung barotrauma
- Craniotomy in the sitting position
- Disruption of blood vessels located above the level of the heart

Prevention

Tilt the head of the bed down to reduce the pressure gradient between site of entry and right atrium when attempting to place or remove a central venous access. Maintain normovolemia when the surgical site is located above the right atrium. PEEP does not appear to reduce the

risk of gas embolism and should be used only to improve oxygenation. Avoid nitrous oxide in patients at risk for gas embolism.

Special Considerations

20–30% of the population has an asymptomatic patent foramen ovale, which can complicate a venous gas embolism (in which the bubbles are usually trapped in the lungs) because bubbles can migrate to the systemic arterial circulation.

Further Reading

Mirski M, et al. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. 2007;106(1):164–77.

Myocardial Ischemia

Definition

Oxygen supply inadequate to meet myocardial demand.

Presentation

- Chest pain, pressure or tightness, often behind the sternum (in an awake patient)
- Radiates to either arm, neck, jaw, back or abdomen
- Usually associated with increase in activity, either physical or emotional
- Dysrhythmias, ST segment changes, or hypotension may be the only findings in an anesthetized patient.

Pathophysiology

An inability to supply enough oxygen to meet myocardial demands. This deficiency may be due to coronary obstruction (physical obstruction from plaque and/or thrombus, or physiologic from coronary spasms), a decrease in oxygen carrying capacity, or a diminished coronary perfusion pressure.

DIFFERENTIAL DIAGNOSIS

- Heartburn/dyspepsia
- Myopathic pain
- Thoracic (bone and cartilage) or pleuritic pain

Immediate Management

- Increase FiO_2 to 100%.
- Administer pain control (IV morphine) if the patient is awake.
- Begin a nitroglycerine infusion (start at 0.5 mcg/kg/min and increase as tolerated until ECG changes or symptoms reverse)

Immediate Management (continued)

- Control heart rate with esmolol (50 mcg/kg/min) or metoprolol (1–2 mg/IV q 5 min)
- Administer aspirin 325 mg PO if the patient is awake and there are no contraindications.
- Heparin or LMWH (1 mg/kg every 12 hours for non ST-elevation myocardial infarction) if the surgical procedure permits.
- Consider clopidogrel (if awake and the surgical procedure permits). The first dose is 300 mg by mouth
- Manage dysrhythmias aggressively

Diagnostic Studies

- ECG
- Echocardiogram
- Coronary artery angiogram.

Subsequent Management

- Initiate primary percutaneous coronary intervention (coronary angioplasty and/or stents)
- Initiate percutaneous cardiopulmonary support (pacemaker, intra-aortic balloon pump)
- Prepare for emergency coronary artery bypass grafting (CABG) surgery to preserve myocardium.
- Long-term care should include antiplatelet therapy with clopidogrel to reduce mortality.

Risk Factors

- Age > 50
- Male > Female
- Hypertension
- Diabetes mellitus
- Hyperlipidemia
- Smoking
- Family history
- Drug use (cocaine)

Prevention

Perioperative myocardial ischemia is multifactorial, and may result from catecholamines, inflammation, altered hemodynamics, or coronary artery vasoconstriction. The role of preoperative

revascularization is controversial. Prevention focuses on risk stratification and early intervention in at-risk patients.

Special Considerations

Mortality for emergency CABG for acute coronary syndrome (due to left main coronary) approximates 9%.

Although outcomes of emergency CABG intervention remain unclear for acute MI, cardiogenic shock prior to CABG intervention has been identified as a significant predictor of mortality.

Despite concerns of excessive bleeding during CABG, clopidogrel is recommended as an early intervention (on admission) even for those who proceed to CABG during the initial hospitalization.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 8: Stabilization of the patient with acute coronary syndromes. *Circulation*. 2005;112:IV-89–IV-110).

Postoperative Hemorrhage

49

Definition

Excessive postoperative bleeding (>300 mL/h in the first few post-op hours)

Presentation

- Increased output from chest tubes or other drains
- Hemodynamic instability

Pathophysiology

Coagulopathy after cardiothoracic surgery may be a result of anticoagulation therapy, resulting in chest wall bleeding. Inadequate surgical hemostasis resulting in suture line bleeds, and elevated blood pressure causing generalized bleeding, are also contributory.

DIFFERENTIAL DIAGNOSIS

- Hypovolemia
- Cardiac dysfunction
- Pericardial tamponade

Immediate Management

- Consider reoperation if bleeding is greater than 300 mL/h over the first few hours.
- Maintain normovolemia with crystalloids, colloid solutions, and blood products.

Immediate Management (continued)

- Administer fresh frozen plasma and cryoprecipitate as necessary to correct coagulopathy.
- Maintain normothermia.
- Fluid resuscitation

Diagnostic Studies

- Coagulation studies
- Temperature

Subsequent Management

- Blood component therapy
- Antifibrinolytic therapy

Risk Factors

- Elevated blood pressure
- Inadequate surgical hemostasis
- Coagulopathies
- Hypothermia

Prevention

- Adequate surgical hemostasis is the best prevention.
- Adequate reversal of heparin (with protamine) confirmed with activated clotting time (ACT) is mandatory.
- Adjuncts like fibrin glue at specific surgical sites can be useful.

Special Considerations

If chest tube output decreases suddenly, chest tubes should be carefully examined for clots. Occluded chest tubes can result in cardiac tamponade. If patient requires massive transfusion, check and replace calcium and avoid hypothermia by warming all infusions. Consider scopolamine, midazolam and ketamine for anesthetic management in patients with hypovolemia and hemodynamic instability.

Further Reading

Despotis G, et al. Prediction and management of bleeding in cardiac surgery. *J Thromb Haemost* 2009;7 Suppl 1:111–117.

Valvular Disease: Aortic Regurgitation

Definition

Abnormalities in the leaflets or supporting structures of the aortic valve resulting in retrograde flow into the left ventricle during diastole.

Presentation

- Dyspnea
- Fatigue
- Palpitations
- Angina

Pathophysiology

Aortic insufficiency leads to increased LV systolic and diastolic volumes. A regurgitant fraction of less than 40% is well tolerated with minimal symptoms, as it approaches 60% there is an increase in LV end diastolic pressure (LVEDP). LVEDP gradually increases, indicating worsening dilation and hypertrophy of the LV eventually leading to pulmonary edema.

DIFFERENTIAL DIAGNOSIS

- Other causes of diastolic murmurs (e.g., mitral stenosis)
- Pulmonary edema
- Coronary artery disease
- Congestive heart failure

Immediate Management

- Mildly vasodilate the patient to minimize afterload and promote forward flow.
- Avoid anesthetic induction with ketamine (may increase afterload).
- Consider vasodilator administration (e.g., nicardipine infusion, start at 5 mg/hr)
- Administer fluid to maintain preload.
- Increase heart rate (goal 90) to minimize diastolic time

Diagnostic Studies

- ECG
- Echocardiogram
- Angiogram

Subsequent Management

- Decrease afterload with vasodilators (e.g., nicardipine 5 mg/hr)
- Consider central venous catheter placement if major fluid shifts are anticipated.
- Consider pulmonary artery catheter placement in the setting of severe aortic insufficiency and decompensated CHF.
- Refer the patient for valve repair or replacement.

Risk Factors

- Marfan syndrome
- Bacterial endocarditis
- Cystic medionecrosis
- Trauma
- Aortic dissection
- Bicuspid aortic valve

52 Prevention

Early detection and medical management of symptoms

Special Considerations

Regurgitant fraction is calculated as follows:

$$\text{Regurgitant Fraction} = \frac{\text{Backward flow}}{\text{Total Flow}}$$

Total flow = (end diastolic vol. – end systolic vol.) × HR

Forward flow = cardiac output

Backward flow = (total flow – forward flow)

Intra-aortic balloon pump is contraindicated in the patient with aortic insufficiency.

Further Reading

Bonow RO, Carabello BA, Chatterjee K, et al. 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease). Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2008 Sep 23;52(13):e1–e142.

Frogl J, Galuska D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. *Anesthesiol Clin*. 2010; Mar;28(1):67–85.

Valvular Disease: Aortic Stenosis

Definition

Narrowing of the aortic opening. May be either at the valvular, sub-valvular, or supravalvular level. Normal valve area 2.6–3.5 cm². Aortic stenosis is graded as mild (1.5–2.5 cm²), moderate (1.0–1.4 cm²), severe (0.6–0.9 cm²), or critical (< 0.6 cm²).

Presentation

- Angina
- Syncope
- Dyspnea

Pathophysiology

A congenital bicuspid valve is the major predisposing factor (>75% of cases) followed by rheumatic fever and senile degeneration.

Mild aortic stenosis is compensated by ventricular hypertrophy and high LV systolic pressure to overcome stenosis. An increase in LV end diastolic pressure is compensatory. Progression of LV dilation and LV hypertrophy further increases LV end diastolic volume, thus increasing workload for the LV. Pulmonary edema eventually develops and increases the risk of sudden death.

DIFFERENTIAL DIAGNOSIS

- Pulmonary Hypertension
- Coronary artery disease
- Congestive heart failure
- Systolic Murmurs (mitral regurgitation)

Immediate Management

- Treat hypotension immediately with a vasoconstrictor.
- Maintain normovolemia.
- Avoid drugs that decrease systemic vascular resistance.
- If necessary, begin a phenylephrine infusion to augment preload and maintain afterload.
- Decrease heart rate (goal 50–70)
- Preserve sinus rhythm—immediate cardioversion for supraventricular dysrhythmias

Diagnostic Studies

- ECG
- Echocardiogram
- Chest radiograph

Subsequent Management

- Afterload reduction is not helpful because the stenotic valve still resists forward flow.
- Administer alpha-adrenergic agents (e.g., phenylephrine) to help maintain systemic vascular resistance (SVR). SVR is important to maintain a diastolic pressure high enough to perfuse the hypertrophied myocardium.
- Long-term beta blockade is usually not well tolerated in aortic stenosis, as it decreases the necessary heightened contractile state, but it may be beneficial in a patient with idiopathic hypertrophic subaortic stenosis (which is a dynamic stenosis).
- TEE for intraoperative monitoring

Risk Factors

- Bicuspid Valve
- Rheumatic Fever
- Age > 70
- Male > female

Prevention

Early detection and medical management of symptoms

Special Considerations

- LV dysfunction (CHF) with aortic stenosis is associated with high risk for sudden death.
- Myocardial hypertrophy is a risk factor for subendocardial ischemia. Myocardial ischemia may be difficult to detect because ECG signs of left ventricular hypertrophy may mask changes due to ischemia.

Further Reading

Bonow RO, Carabello BA, Chatterjee K, et al. 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease). Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2008 Sep 23;52(13):e1–142.

Frogel J, Galusca D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. *Anesthesiol Clin*. 2010; Mar;28(1):67–85.

Valvular Disease: Mitral Regurgitation

Definition

- Blood flowing from the left ventricle back into the left atrium during systole. Regurgitant fraction = regurgitant volume/LV stroke volume
- Mild MR: Regurgitant fraction < 30%
- Moderate MR: Regurgitant fraction 30–60%
- Severe MR: Regurgitant fraction > 60%

Presentation

- Fatigue
- Dyspnea
- Orthopnea

Pathophysiology

- Mitral regurgitation (MR) can be primary due to a defective valve or secondary due to LV dilation.
- The most common cause of MR is mitral valve prolapse. Myocardial infarction with papillary muscle dysfunction presents as acute-onset MR.
- Acute MR leads to increased left atrial volumes and pressures that are transmitted to the pulmonary circuit. Compensatory tachycardia maintains cardiac output at the expense of increased oxygen consumption.
- Chronic slow-onset MR causes compensatory left ventricular hypertrophy and left atrial dilation that maintains forward flow and normal pressures to the pulmonary circuit; hence, the relative absence of symptoms. When the regurgitant fraction increases to 60% the hypertrophic and dilated LV fails to compensate, and heart failure ensues.

DIFFERENTIAL DIAGNOSIS

- Primary pulmonary hypertension
- Myocardial ischemia
- Cardiomyopathy

Immediate Management

To ensure forward flow:

- Maintain preload with volume resuscitation
- Avoid bradycardia (increases LV and regurgitant volume)
- Decrease afterload
- Avoid further increases in pulmonary vascular resistance (hypoxia, hypercapnia, acidosis)

Diagnostic Studies

- Echocardiogram
- Giant V wave on pulmonary artery occlusion pressure tracing

Subsequent Management

- Nitric oxide is a pulmonary vasodilator
- Consider prostaglandin E1 (alprostadil)
- Surgical intervention

Risk Factors

- Mitral valve prolapse
- Myocardial ischemia with papillary muscle dysfunction
- Bacterial endocarditis

Prevention

Once LV dysfunction has become established, it may be irreversible; hence early intervention is necessary.

Special Considerations

Atrial fibrillation is seen in about 75% of cases of MR.

Further Reading

Bonow RO, Carabello BA, Chatterjee K, et al. 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease). Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2008 Sep 23;52(13):e1–142.

Frogel J, Galusca D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. *Anesthesiol Clin*. 2010; Mar;28(1):67–85.

Valvular Disease: Mitral Stenosis

Definition

- Normal Valve area 4–6 cm² (Valve Index 4–4.5 cm²/m²)
- Symptomatic at 1.5–2.5 cm² (Valve Index 1–2 cm²/m²)
- Critical < 1 cm²
- Incompatible with life < 0.3 cm²

Presentation

- Dyspnea
- Fatigue
- Palpitations
- Paroxysmal nocturnal dyspnea
- Hemoptysis
- Angina

Pathophysiology

Usually secondary to rheumatic heart disease, with fusion of valvular commissures and progressive scarring.

Mild disease with good physiologic compensation is asymptomatic. The increased filling pressure in the left atrium with mild mitral stenosis is not usually transmitted to the pulmonary circulation. Progression of stenosis gradually increases pulmonary vascular resistance and right ventricle pressure. Hypertrophy and dilation of the RV further compromise LV function by shifting the interventricular septum toward the LV, decreasing LV volumes.

DIFFERENTIAL DIAGNOSIS

- Primary pulmonary hypertension
- Other causes of diastolic murmurs (e.g., aortic regurgitation)
- Myocardial ischemia

Immediate Management

- Maintain preload (increases can precipitate pulmonary edema)
- Slow heart rate (maximize time spent in diastole)
- Maintain pulmonary vascular resistance (avoid increases in pulmonary vascular resistance by hypoxia, hypercarbia, and acidosis)
- Immediate cardioversion to maintain sinus rhythm

Diagnostic Studies

- Transesophageal echocardiogram (TEE)
- Pulmonary artery catheter—large A wave on PA tracing

Subsequent Management

- Afterload reduction is not helpful, as the proximal stenosis at the mitral valve is the limiting factor.
- AV pacing with long PR intervals (to allow adequate filling) may be necessary.
- Beta-blockade

- Maintenance of sinus rhythm
- Surgical intervention

Risk Factors

- Female > Male
- Rheumatic fever

Prevention

Early detection and medical management of symptoms, and early intervention to avoid irreversible ventricular dysfunction.

Special Considerations

- Atrial contribution to the LV stroke volume is extremely important, and as high 30% of the stroke volume.
- Even though mitral commisurotomy results in a restenosis rate of 30% in five years, the early morbidity is lower than that of valve replacement.

Further Reading

Bonow RO, Carabello BA, Chatterjee K, et al. 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease). Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol.* 2008 Sep 23;52(13):e1–e142.

Frogel J, Galusca D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. *Anesthesiol Clin.* 2010; Mar;28(1):67–85.

Thoracic Aortic Dissection

Definition

Dissection of the intimal and medial layers of the thoracic aortic wall by penetrating blood.

Presentation

- Chest pain
- Back pain (often described as “ripping”)
- Congestive heart failure with aortic insufficiency
- Pericardial tamponade
- Elevated blood pressure

- Dyspnea and hoarseness due to recurrent laryngeal nerve compression and direct tracheal compression
- Hemoptysis due to tracheal erosion
- Cardiac ischemia

Pathophysiology

Blood most commonly penetrates an intimal tear and separates the intima and media, unlike an aneurysm where all three layers of vessel wall are dilated. Tears in the ascending aorta and aortic arch make up 70% of all dissections. Vasa vasorum rupture has been implicated in a minority of cases without intimal tear.

DIFFERENTIAL DIAGNOSIS

- Acute MI
- Cardiogenic shock
- Pancreatitis
- Thoracic outlet syndrome

Immediate Management

- Administer vasodilators to maintain systolic BP 105–115 mm Hg
- Administer beta blockers (e.g., labetalol or an esmolol infusion) to maintain HR 60–80
- Transfuse packed red blood cells and/or factors as necessary.
- Surgical correction

59

Diagnostic Studies

- Chest X-ray (mediastinal width greater than 8 cm,)
- Chest CT
- Transesophageal echocardiogram
- Angiogram

Subsequent Management

Assessment of organ function is important. Neurologic changes, deteriorating kidney function and gastrointestinal perfusion (which may manifest as metabolic acidosis) are all indications for acute surgical intervention.

Risk Factors

- Hypertension
- Age > 60
- Male > Female
- Marfan syndrome and other connective tissue disorders
- Pregnancy

Prevention

Aggressive BP control.

Special Considerations

Surgical mortality greater than 30% is considerably better than an untreated 2-day mortality of 50% and 6-month mortality as high as 90%.

Judicious use of preoperative pain medication so as not to obtund the patient is necessary to be able to monitor the patient for propagation of dissection into head vessels and associated mental changes.

A balance between full-stomach precautions requiring rapid sequence induction and slow, controlled induction to maintain hemodynamic stability is required.

Femoral artery cannulation may be required if the entire ascending aorta is involved, so as to allow for perfusion of the major vessels.

Left radial artery is preferred for the arterial line placement with ascending aortic dissections, as the right subclavian artery may be involved.

Further Reading

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Subramanian S, Roselli EE. Thoracic aortic dissection: long-term results of endovascular and open repair. *Semin Vasc Surg.* 2009;22(2):61–68.

Ventricular Fibrillation

Definition

A nonperfusing rhythm of the ventricles with disorganized and non-coordinated electrical activity.

Presentation

Either a witnessed cardiac arrest, or noted on the ECG

Pathophysiology

Usually secondary to myocardial ischemia or underlying conditions such as hypoxia and acidosis.

DIFFERENTIAL DIAGNOSIS

- Drug toxicity (cocaine, digitalis, antidepressants)
- Pulmonary embolism
- Hypoxia
- Acidosis

- Cardiac tamponade
- Electrolyte abnormalities (hyper/hypokalemia)
- Hypovolemia
- Shivering

Immediate Management

- 360 J* monophasic (200 J* biphasic) cardioversion—may repeat every 2 minutes if necessary (**avoid delay—administer shocks before airway management**)
- Endotracheal intubation
- CPR after shock to maintain 30:2 ratio for 2 minutes without pausing to check rhythm or pulse
- Epinephrine 1 mg IV (repeat every 3–5 minutes) OR 40 U IV vasopressin (one-time dose)

Diagnostic Studies

- ECG
- Serum electrolyte levels
- Arterial blood gas
- Cardiac enzymes
- Echocardiogram

Subsequent Management

- Airway management
- ACLS secondary survey
- Amiodarone 300 mg IV (repeat 150 mg in 3–5 minutes if VF/PVT persists)
- Lidocaine (if amiodarone unavailable) 1.0–1.5 mg/kg IV, may repeat to a 3 mg/kg max. loading dose
- Magnesium sulfate 1–2 g IV diluted in 10 mL 5% dextrose in water for torsades de pointes or suspected/ known hypomagnesemia

Risk Factors

- Cardiac comorbidities
- Advanced disease states

Prevention

Early detection of precipitating underlying conditions and therapy.

Special Considerations

ACLS guidelines have been updated to emphasize the importance of early cardioversion; hence, the recommendation of 360 J instead of an escalating energy to cardiovert. Current guidelines recommend cardioversion **before** definitive airway management.

Further Reading

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Chapter 3

Equipment Problems

James B. Eisenkraft

Breathing Circuit Malfunction: Low Pressure Condition 64
Electric Power Failure 66
Oxygen Pipeline Failure 69
Ventilator Failure 72

Breathing Circuit Malfunction: Low Pressure Condition

Definition

Failure to generate or sustain a positive pressure that is sufficient to ventilate the patient's lungs

Presentation

- Breathing system fails pre-use checkout.
- During spontaneous ventilation, reservoir bag empties; FIO_2 is less than intended, leading to low FIO_2 alarm.
- During bag-assisted ventilation, bag empties easily when squeezed but lungs do not inflate.
- During positive pressure ventilation by ventilator:
 - Properly set breathing system low pressure alarm is annunciated during case.
 - Capnogram is abnormal or absent (apnea) leading to alarm.
 - Ventilator standing bellows fails to fill; sinks to bottom of bellows housing.
 - Spirometry alarm for low tidal volume (TV)/minute volume (MV) is annunciated.

Pathophysiology

Breathing system must be gas tight to ensure that patient receives gas mixture produced by the machine, and that positive pressure is transmitted to the lungs during inspiration.

DIFFERENTIAL DIAGNOSIS

- Leak in anesthesia machine low-pressure system
- Breathing system disconnection or misconnection
- Breakage of, or leakage from, a breathing system component that may be obvious or concealed
- Leak around airway management device (tracheal tube, laryngeal mask airway (LMA))
- Leak from bronchial tree (e.g., bronchopleural fistula)

Immediate Management

- Inspect anesthesia workstation/breathing system for obvious site of leak and correct if possible.
- If the leak is small, increase fresh gas flow (FGF) to compensate
- If the leak cannot be compensated for by increasing FGF, disconnect patient from breathing system at Y-piece/elbow and

Immediate Management (continued)

ventilate lungs with self-inflating resuscitation (e.g., Ambu) bag connected to an O₂ source if available—otherwise use room air.

- Maintain anesthesia with intravenous agents.
- Call for help to repair the leak while continuing to care for the patient.

Diagnostic Studies

- If possible, disconnect patient from breathing system and repeat manufacturer-recommended pre-use checkout to identify likely point of leakage, i.e., bag circuit, ventilator circuit, or both. Trace gas pathway from machine common gas outlet, through all breathing circuit components (including tracheal tube or other airway device), to waste gas scavenging.
- Measure inspired and expired tidal volumes at Y-piece. A difference indicates leak distal to Y (e.g., tracheal tube cuff leak; poorly seated LMA).
- Does set ventilator TV agree with inspired and expired TVs?

Subsequent Management

- If source of leak has been determined, correct if possible.
- If source is not determined, replace anesthesia breathing system and/or workstation.
- Refer to authorized service personnel.

Risk Factors

- Failure to perform pre-use checkout procedures recommended by manufacturer
- Failure to correctly assemble breathing system components

Prevention

- Educate personnel in the correct operation of the workstation, including pre-use checkout.
- Use all available breathing system monitors and alarms. Early detection and intervention will avoid an adverse outcome.

Special Considerations

Any connection can become disconnected or misconnected.

Further Reading

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- Eisenkraft JB. Hazards of the anesthesia delivery system. In: Rosenblatt MA (ed.). *ASA refresher courses in anesthesiology*, Vol. 37. Lippincott, Williams & Wilkins; 2009:37–55.
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Electric Power Failure

Definition

- Failure of delivery of electrical power to the OR. May be due to failure of electric utility company supply to the facility, or failure within the facility.
- Most facilities have backup generators that turn on automatically when a utility company supply failure occurs. This backup may fail during a fire or during natural disasters such as storms, earthquakes, terrorism act, etc.

Presentation

- Room lighting fails.
- All wall-powered electrical devices turn off, unless they have a backup power supply or are connected to an uninterruptible power supply, UPS.
- Some or all functions of an electronic workstation may cease to operate, depending on the model.
- Physiologic monitoring fails (ECG, noninvasive blood pressure (NIBP), pulse oximeter, gas monitoring, capnograph).
- Anesthesia information management (record-keeping) system and other computers shut down.
- Local telephone and paging systems may shut down.
- Cardiopulmonary bypass machine, cell saver, electrocautery, robotic systems, etc. will fail.
- If failure is in the supply to the facility and emergency generators are activated, electricity should resume within a short period of time. During the transition, computer-based equipment may need to power up and/or be reset. Only certain outlets and lights in the OR are energized from the emergency power supply; these are usually identified with a red wall plate.

DIFFERENTIAL DIAGNOSIS

- Failure of supply outside the facility (e.g., excessive power demand on utility company, weather-related power line failure, natural disasters, fire, earthquake, construction work).
- Unannounced construction work or to failure of individual circuits in the OR (e.g., turned off accidentally, or by a tripped circuit breaker) causing shutdown of one or more electrical devices.

Immediate Management

- Obtain emergency portable lighting (flashlight, laryngoscope light, emergency flashlights mounted on wall in most ORs)
- Call for help. If the OR telephone system fails, use a cellular telephone.
- Ensure that power loss is not due to patient or personnel electrocution.
- If an electrical panel is in the room, check for tripped circuit breakers.
- Ensure that all essential electrically powered equipment is connected to emergency power outlets in OR
- Check that oxygen pipeline gas supply and suction are functioning; otherwise follow protocol for oxygen pipeline failure.
- Check that the anesthesia workstation is functioning and that the patient's lungs are being ventilated. If not, switch to manual ventilation by breathing system reservoir bag, or self-inflating resuscitation bag.
- Discuss the situation with surgeon/proceduralist. Abort the procedure if possible. Awaken patient or not?
- Re-establish patient monitoring. If possible, obtain a battery powered transport monitor for ECG, NIBP, capnography, pulse oximetry. Otherwise, individual battery powered monitors may be available.
- ECG can be monitored using the monitoring mode of a defibrillator
- Use a precordial or esophageal stethoscope to monitor ventilation
- Obtain a manual BP cuff and sphygmomanometer to monitor BP. Palpate the pulse.
- Consider using colorimetric CO₂ detector or battery powered semi-quantitative CO₂ mainstream analyzer (e.g., Stat cap, Phasein EMMA) to monitor CO₂

Immediate Management (continued)

- Most electronic workstations have a backup battery that will supply power for 30–45 minutes. Conserve battery power by switching off the ventilator and manually ventilate using the reservoir bag. Adjust the screen brightness control to the lowest possible setting.
- On workstations that normally have electronic display of gas flows, revert to 100% O₂ from backup rotameter (Dräger Apollo®, Dräger Fabius® GS, GE S5/ADU®). If a GE Aisys® workstation, switch to “Alternate Oxygen” flowmeter.
- Maintain anesthesia using intravenous agents.
- Reassign personnel to provide manual power (e.g., hand-ventilating patient’s lungs; cranking the cardiopulmonary bypass machine).
- Elective surgical procedures should be postponed until a reliable electrical supply has been restored.

Diagnostic Studies

Refer to authorized engineering and service personnel.

Risk Factors

- Failure to regularly check emergency backup power supply systems and to maintain/repair as required.
- Unannounced construction or maintenance in the vicinity of the institution
- Unannounced construction or maintenance in the vicinity of the ORs

Prevention

- Regularly test emergency power supply to institution to ensure that automatic transfer to this source occurs without delay. Service and repair as needed.
- Provide adequate warning to all staff of any planned interruption of electrical power supply (e.g., maintenance or construction).
- Educate OR personnel about the power supply to the OR, how to react to a power supply failure, and about electrical safety.
- During checkout of the anesthesia workstation, note the charge status of the backup battery.
- Regularly check all anesthesia devices and monitors to ensure that the batteries are charged and can maintain their charge. Replace backup batteries as necessary.

- Regularly check that battery-powered lighting/emergency flashlights are present, obvious, functioning, and charging in each anesthetizing location.
- Consider obtaining uninterruptible power supply (UPS) units for electrically powered devices such as OR computers and anesthesia information management systems (AIMS) that have no backup battery.

Further Reading

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Welch RH, Feldman JM. Anesthesia during total electrical failure, or what would you do if the lights went out? *J Clin Anesth* 1989;1:358–362.

Yasny J, Soffer R. A case of power failure in the operating room. *Anesth Prog*. 2005;52:65–69.

Oxygen Pipeline Failure

Definition

Oxygen pipeline supply pressure to the workstation is either absent or below the minimum oxygen pressure required for normal function of the anesthesia workstation.

Presentation

- Workstation fails pre-use checkout.
- Low oxygen supply pressure alarm is annunciated in workstation.
- If due to a central supply problem, low oxygen supply pressure alarm sounds at the OR control center.
- O₂ flow at main and auxiliary flow meters decreases or ceases; oxygen flush fails.
- Other gases supplied to machine (N₂O, heliox, possibly air) stop flowing.
- Oxygen-powered pneumatic ventilator stops working, leading to alarms for apnea (low pressure, absent capnogram).

Pathophysiology

- Pipeline supply of oxygen at 50 psig enters workstation high pressure system and then supplies the following:
 - High pressure diameter indexed safety system (DISS) oxygen take-off (to drive jet ventilator, venturi suction device)

- Pneumatically powered ventilator
- Auxiliary flowmeter (for nasal cannula, etc.)
- Alternate oxygen flow meter (GE Aisys® workstation)
- Low oxygen pressure alarm
- Fail-safe mechanism
- O₂ flush
- Main oxygen flow meter
- In the absence of an open cylinder supply, the latter functions fail.
- Failure of central oxygen storage supply or pipeline to OR (empty, leaking), pipeline system closed off or obstructed (e.g., by debris), or obstruction in hose connecting oxygen wall outlet to workstation.

DIFFERENTIAL DIAGNOSIS

- Leak in anesthesia machine high-pressure system
- Obstruction/kinking of hose between wall oxygen outlet and workstation
- Debris in pipeline system obstructing wall oxygen outlet
- Shutoff valve outside OR is in OFF position
- Unannounced maintenance of pipeline system
- Failure of connection between bulk oxygen storage vessel and pipeline system

Immediate Management

- Confirm loss of pipeline oxygen supply/pressure by checking pipeline supply pressure gauge on workstation. Check O₂ flush operation.
- Open reserve O₂ cylinder on workstation
- Take steps to minimize oxygen use
 - Use lowest O₂ flow possible (closed-circuit technique if possible).
 - Switch off a pneumatic ventilator; use spontaneous or manual ventilation.
 - Ensure that auxiliary oxygen flow is off.
- Announce the failure, call for help and for additional backup oxygen tanks.
- Alert surgeon, personnel in other ORs, and the engineering department.
- If workstation has pipeline supply of air and the patient will tolerate a lower FIO₂, consider decreasing FIO₂ to conserve oxygen.

Immediate Management (continued)

- If the ventilator is driven by O_2 and use of compressed air is not possible, ventilate with room air using a self-inflating resuscitation bag and maintain anesthesia using intravenous agents.
- Elective surgical procedures should be postponed until pipeline oxygen supply has been restored, and an adequate supply of backup oxygen cylinders confirmed.

Diagnostic Studies

- Ensure adequate FIO_2 by oxygen analyzer.
- Ensure adequate SpO_2 .
- Refer to authorized engineering and service personnel.

Risk Factors

- Failure to perform pre-use checkout procedures recommended by manufacturer
- Unannounced construction in vicinity of OR
- Unannounced pipeline maintenance
- Filling of bulk oxygen storage vessel by unqualified personnel.

Prevention

- Educate personnel in correct operation of workstation, including pre-use checkout.
- Check for normal pipeline pressure.
- Check adequacy of all hose connections between wall and workstation.

Special Considerations

- Before starting a case, ensure that backup tank of oxygen on the workstation is tightly secured in the hanger yoke.
- Ensure that the tank is full, and then turn off the tank. If the tank is left open, oxygen may leak out between the tank and the hanger yoke. If the pipeline pressure decreases to less than 45 psig, oxygen will be drawn from an open tank, silently depleting the backup tank oxygen supply.
- Ensure that all anesthesia personnel have been instructed and have practiced changing the oxygen tank on the workstation.
- Consider, for backup supply, E size O_2 tanks that are filled to 3000 psig and contain 1000 liters of gaseous O_2 , and that have a

regulator and DISS oxygen connection that can provide oxygen at 50 psig. If the pipeline supply fails, the oxygen hose can be disconnected from the wall outlet and connected to the high-pressure outlet on the tank.

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Ventilator Failure

Definition

Inability of the anesthesia ventilator to deliver the set inspired volume to the patient's lungs

Presentation

- Workstation fails pre-use checkout for ventilator.
- Ventilator cannot be turned on.
- Ventilator bellows movement is abnormal (irregular, sticking) or absent.
- Ventilator does not sound normal during positive pressure inspiration.
- Ventilator/ventilation alarm(s) is/are annunciated (e.g., low/high TV, MV; low/high/continuing pressure).
- Physiologic monitors indicate inadequate ventilation (low/high end-tidal carbon dioxide; low SpO₂).
- Clinical signs of hypoventilation/apnea include failure of chest to move normally with ventilation, absence of breath sounds on auscultation, and patient attempting to breathe spontaneously.

Pathophysiology

- Bellows ventilators are powered pneumatically (by compressed oxygen, or in some cases air); piston ventilators are driven by an electric motor. In both designs, the controls are electronic. Ventilator operation may not be intuitively obvious; the uneducated user may inadvertently select an incorrect mode, resulting in failure to ventilate.

- The ventilator must be connected correctly to a properly configured breathing system.
- No fresh gas flow, or flow is inadequate to overcome a small leak in the breathing system
- Change in ventilatory needs during the procedure associated with decrease in thoracic compliance
- Breathing system or airway obstruction
- Failure of pneumatic and/or electrical power supply to the workstation
- Failure of a ventilator component (may be mechanical, electronic, computer control system, or pneumatic)
- Failure of breathing system flow sensor. Properly functioning flow sensors are required for the normal operation of some ventilators (e.g., GE Aisys® Carestation).
- Failure of decoupling valve in a workstation that uses fresh gas decoupling (e.g., Drager Fabius® GS, Drager Apollo®, Datascope Anestar®)

DIFFERENTIAL DIAGNOSIS

- Ventilator turned off
- Failure of ventilator pressure relief valve (stuck open, causing leak; stuck closed, causing high pressure condition in breathing system)
- Obstruction to inspiratory side of circle system

Immediate Management

- Call for assistance. Have an anesthesia technician or other anesthesia caregiver troubleshoot and correct the problem while you care for the patient.
- Change from ventilator to bag circuit, fill the circuit using oxygen flush control, and attempt to ventilate using the breathing system reservoir bag. If this is successful, continue bag ventilation.
- If the breathing circuit cannot be filled by operating the oxygen flush, ventilate the patient with a self-inflating resuscitation (“Ambu”) bag. If a source of oxygen or air is available, a Bain circuit may be used. In the absence of back-up ventilation equipment, mouth-to-tracheal tube ventilation may be necessary.
- Check circuit for disconnections, misconnections, and other sites of gas leakage. Check that the pipeline gas supply is functioning; if not, switch to backup cylinder supplies.
- If the circuit does fill from the oxygen flush/flowmeters, but the patient can not be ventilated, revert to the “Ambu” bag, Bain,

Immediate Management (continued)

or other alternative ventilation system, and look for possible obstruction on the inspiratory side of the circle system. The inspiratory unidirectional valve could be stuck, or the lumen of the inspiratory limb may be obstructed (e.g., by plastic wrapping).

- If unable to ventilate with the Ambu, Bain, or other backup system, consider an obstruction to/in the patient's airway (e.g., kinked tracheal tube, herniated cuff, foreign matter in the tube or bronchial tree, endobronchial intubation, bronchospasm, tension pneumothorax).
- If inhalational agents cannot be administered, anesthesia should be maintained using a total intravenous anesthesia (TIVA) technique.
- Ensure that ventilation and oxygenation are maintained using the backup system if necessary, until the failure has been identified and the problem corrected.
- If the problem cannot be corrected by an anesthesia caregiver or anesthesia technician, the workstation should be withdrawn from clinical use until repaired by manufacturer-authorized service personnel.

Diagnostic Studies

- Ensure adequate FIO_2 by oxygen analyzer.
- Ensure adequate SpO_2 .
- Refer to authorized engineering and service personnel.

Risk Factors

- Failure to perform pre-use checkout procedures recommended by manufacturer
- Failure to maintain/service workstation according to manufacturer's recommendations
- Inadequate education of anesthesia caregiver in the operation of the workstation/ventilator.

Prevention

- The anesthesia caregiver must be adequately trained in the use of the ventilator.
- Some newer workstations use fresh gas decoupling to prevent changes in tidal volume when fresh gas flow rate, respiratory rate,

or I:E ratio are changed. It is important that the user understand the differences between this design, and that of the traditional systems.

- Equipment should be properly maintained and serviced according to manufacturer's recommendations.
- Educate personnel in the correct operation of the workstation.
- Perform proper pre-use checkout of the workstation/ventilator
- Confirm correct assembly of the breathing circuit, function of unidirectional valves, and ability to ventilate a test lung (in the form of a reservoir bag) connected at the Y-piece of the circle system in both manual/bag and automatic/ventilator modes, and proper function of bag/ventilator selector switch.
- Ensure that an alternative means to ventilate the patient's lungs is immediately available and functioning.
- Consider, for backup supply, E size oxygen tanks that are filled to 3000 psig (containing 1000 liters of gaseous oxygen), and have a regulator and DISS oxygen connection that can provide oxygen at 50 psig. If the pipeline supply fails, the workstation's oxygen hose can be disconnected from the wall outlet and connected to the 50 psig outlet on the O₂ tank.

Special Considerations

- Standard E cylinders on the anesthesia machine are filled to 2200 psig and contain 684 liters of O₂.
- To estimate the time remaining at a given flow rate, use the formula:

$$\text{Time remaining (hours)} = \text{Pressure (psig)} / (200 \times \text{O}_2 \text{ flow rate (l/min)})$$

Further Reading

ASA Sub-Committee on Equipment and Facilities. Recommendations for pre-anesthesia checkout procedures (2008). Available at: www.asahq.org/clinical/FINALCheckoutDesignguidelines02-08-2008.pdf. (Accessed March 14, 2010).

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Chapter 4

Ethical Considerations

Robert B. Schonberger and Stanley H. Rosenbaum

Informed Consent 78

Refusal of Blood Transfusion 80

Organ Harvesting and the Declaration of Brain Death 81

Key Points

Informed Consent

- As a general rule, informed consent is required prior to all invasive procedures.
- In an emergency in which no decision maker is available, physicians should act in accordance with what a reasonable person would consider to be in the best interests of the patient.

Refusal of Blood Transfusion

- With few exceptions (see below) patients may refuse blood transfusion for any reason.
- There are numerous strategies for managing a patient who refuses transfusion. Some major modalities include deliberate hemodilution, removal of blood that is kept in a continuous circuit with the patient, cell saver systems, and pharmacologic treatments such as erythropoietin, desmopressin (ddAVP), and recombinant factor VII (if indicated).
- Specific strategies should be discussed with the patient, as individual acceptance varies.

Organ Harvesting and Brain Death

- With the exception of a competent and consenting living donor, organ harvesting may occur only after the declaration of cardiac or brain death.
- In the United States, the declaration of brain death requires death of the entire brain, including the brainstem. Patients who are persistently vegetative, minimally conscious, or exhibit spontaneous respiratory efforts are not brain dead and may not undergo organ harvesting unless donation after cardiac death has been arranged.
- Donation after cardiac death involves the removal of life support in anticipation of death within a short time. Strict institutional protocols govern such procedures. If the donor does not become apneic and pulseless within a specified time, the organ harvest does not proceed, and appropriate care of the critically ill patient should resume.

Informed Consent

The Ethical Standard of Care

- As both a legal and ethical matter, informed consent must be obtained prior to the performance of any invasive procedure.

- Competent patients may refuse to give such consent for any procedure for any reason.
- For competent patients, informed consent must be obtained from the patient and must include an explanation of the purpose of the proposed procedure as well as the major risks, benefits, and alternatives to the procedure.
- In the case of incompetent patients who were formerly competent, a surrogate decision maker must be sought. The essential elements and requirements of the informed consent process are the same when dealing with surrogates, as with patients themselves. The surrogate's role is to act in substituted judgment, i.e., to authorize what the patient's wishes would be if the patient were in a position to express them.
- In the case of a minor child, or individual who has never been competent, informed consent must be obtained from the legal guardian or conservator whose role is to represent the best interests of the incompetent person.
- For emergency situations in which delay would result in the loss of life, limb, or other serious morbidity, and in which a competent decision maker cannot be located, physicians have an ethical duty to act in accordance with what a reasonable person would hold to be in the best interest of the patient until such time as informed consent can be sought.

Exceptions to the Need for Informed Consent

- Patients may be incapable of informed consent due to acute injury, chronic brain disease, medication, intoxication, mental impairment, mental illness, or young age. In all of these cases, the physician's obligation to obtain informed consent does not go away; the process simply transfers from the patient onto the surrogate decision maker.
- Physical disability or language barrier may make obtaining consent inconvenient, but has no impact on a patient's competency or on the obligation of the caregiver to obtain consent from the patient.
- The emergency exemption to obtaining informed consent may be conceptualized as a reasonable presumption of consent. If the presumption of consent is later contravened by the patient or a surrogate, the emergency exemption no longer applies.
- In the case of minor children, some treatments may be mandated by the courts despite the parents' refusal to give informed consent. Keeping in mind the emergency exemption, it is prudent for a care provider to seek institutional administrative support in such situations.

Further Reading

For a discussion of the typology of informed consent as distinct from shared decision making and simple consent, see:

Whitney SN, McGuire AL, McCullough LB. A typology of shared decision making, informed consent, and simple consent. *Ann Intern Med.* 2004;140(1):54–59.

Refusal of Blood Transfusion

The Ethical Standard of Care

- Competent adult patients may refuse blood transfusion or any other treatment for any reason, even if withholding such treatment would entail death.
- Patients should be asked before their surgery whether they have any objections to receiving blood products. Any such wishes should be honored.
- Individual patient preferences may differ regarding cell saver systems and extracorporeal blood circuits. The specifics of a patient's preferences should be clarified prior to surgery.
- A care provider who objects to withholding appropriate blood products may defer care to a colleague who is able to honor the patient's autonomy. In cases where alternative providers do not exist, physicians may have an obligation to provide care despite their own objections.

Exceptions to the Refusal of Blood Transfusion

- United States case law generally holds that parents of preadolescent children may NOT refuse blood transfusions on behalf of their minor children.
- Older adolescent children with objections to blood transfusion fall into a gray area in which an ethics consult is best pursued.
- Gravid patients with viable fetuses present another ethical dilemma for which conflicting case law exists, and for which an ethics consult may be warranted.

Treatment Modalities for Patients Who Refuse Transfusion

- A variety of techniques may reduce risk to patients who refuse blood transfusion.
- Preoperatively, patients may receive recombinant erythropoietin in an effort to increase hemoglobin concentration. Some insurance carriers will not pay for this treatment without sustained advocacy on behalf of the patient.

- Occasionally, patients who refuse anonymous-donor banked blood may still consent to autologous blood banking.
- Patients who refuse autologous blood banking may sometimes consent to the withdrawal of blood if it is kept in a continuous circuit with the body. Such patients may undergo intraoperative withdrawal of blood into a blood donation bag that is left connected to the IV circuits. The blood can be slowly reinfused until a transfusion is needed.
- Deliberate hemodilution techniques are commonly used, but evidence-based guidelines have yet to be developed.
- Steps to reduce blood loss include modifications of surgical technique, controlled hypotension, use of ddAVP, and use of recombinant factor VII.
- Cell saver systems can be useful if patients consent to them. In situations, such as oncologic surgery, where cell saver is normally contraindicated, surgical blood loss may still be collected in a cell saver system. This blood should then be discarded unless the need for blood becomes critical.
- Postoperative recombinant erythropoietin has been used. In addition, there are case reports of salvage therapy using hypothermia, sedation, and neuromuscular blockade to reduce oxygen consumption in patients with otherwise fatal anemia.

Further Reading

For a discussion of some relevant case law, see:

Benson KT. The Jehovah's Witness patient: considerations for the anesthesiologist. *Anesth Analg*. 1989 Nov;69(5):647–656.

Organ Harvesting and the Declaration of Brain Death

The Ethical Standard of Care

- Outside the domain of the consenting living donor, organ harvesting may occur either after the declaration of cardiac or brain death.
- The declaration of brain death in the United States requires documentation that the entire brain, including the brain stem, has permanently ceased to function.
- The declaration of brain death must generally be made by at least one physician who is not connected to the transplantation process.

- Comatose patients with residual brain function, including persistently vegetative patients, do not meet the legal definition of brain death and may not undergo organ harvesting unless a donation after cardiac death has been arranged.
- A patient who shows evidence of spontaneous respiration or other brainstem activity is not legally dead.

The Determination of Brain Death

- Reversible causes of apparent coma must be ruled out both by history and exam. Specifically, severe metabolic derangement, intoxication, hypothermia, and residual neuromuscular blockade must be considered and, where appropriate, treated.
- The clinical determination of brain death requires:
 - 1) unresponsiveness, including to painful stimuli in cranial nerve territories;
 - 2) the absence of all brainstem reflexes;
 - 3) apnea in response to a hypercarbic challenge.
- Most United States jurisdictions hold that the clinical determination of brain death is sufficient in adults for the determination of death without the need for further neurophysiologic testing.
- In cases where there remains some doubt, or where the clinical tests for the determination of brain death cannot be performed, brain death may also be based on an EEG showing absent brain activity, or on perfusion studies that demonstrate the absence of brain blood flow.
- Legal and institutional guidelines vary but may require the determination of brain death be performed by more than one physician or at more than one point in time.
- Some United States jurisdictions allow for the next of kin to refuse a neurological standard of death entirely.

Pitfalls in the Declaration of Brain Death

According to the American Academy of Neurology, brain death may be diagnosed even if any of the following are present:

- Spontaneous movements of limbs other than pathologic flexion or extension
- Intercostal expansion without significant tidal volumes
- Sweating, blushing, or tachycardia
- Hemodynamic stability
- Absence of diabetes insipidus
- Deep tendon reflexes
- Babinski reflex

Donation after Cardiac Death (DCD)

DCD refers to the practice of removing a severely ill patient from active life support in the expectation that the patient will have a cardiac arrest within a few minutes. This is distinct from donation after brain death, in which asystole is not required. In a DCD situation, when asystole has lasted for a specified period (usually taken to be 5 minutes), the patient is pronounced dead. Subsequently, the transplant team removes organs, generally just the liver and kidneys, for transplantation. This procedure must satisfy strict constraints:

- The patient's surrogate decision makers must have given formal consent for withdrawal of active life support.
- To avoid any potential conflicts of interest, there must be a sharp separation between the medical team caring for the patient and the transplant team.
- There must be a formal institutional protocol describing the details of the process, including the acceptable participants, the venue for the withdrawal of active support, the waiting duration for the patient to expire, and the period of asystole needed for the declaration of death.
- If the patient does not become apneic and pulseless during the waiting time after withdrawal, generally chosen to be one hour, the patient is returned to regular hospital care appropriate for a standard end-of-life patient.

Further Reading

American Academy of Neurology, Quality Standards Subcommittee. Practice parameters: Determining brain death in adults, *Neurology*. 1995;45:1012–1014.

Van Norman GA. A matter of life and death: what every anesthesiologist should know about the medical, legal, and ethical aspects of declaring brain death. *Anesthesiology*. 1999;91(1):275–287.

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Chapter 5

Metabolic and Endocrine Emergencies

Lewis J. Kaplan

- Acidosis 86
- Adrenocortical Insufficiency (AI) 88
- Alkalosis 90
- Anaphylaxis 91
- Coagulopathy 93
- Diabetic Ketoacidosis (DKA) 96
- Hypercalcemia 99
- Hyperkalemia 101
- Hypermagnesemia 102
- Hyernatremia 104
- Hypocalcemia 105
- Hypokalemia 107
- Hypomagnesemia 109
- Hyponatremia 110
- Hypothermia 112
- Malignant Hyperthermia (MH) 114
- Pheochromocytoma 116
- Porphyria 119
- Sickle Cell Crisis 121
- Thyroid Storm 123
- TURP Syndrome 125

Acidosis

Definition

Blood pH less than 7.36 (normal = 7.36–7.45)

Presentation

- Finding on arterial blood gas analysis
- Respiratory acidosis: signs of CO₂ retention, including vasodilation, narcosis, and possibly cyanosis.
- Metabolic acidosis: hypotension, vasodilation, myocardial depression and dysrhythmias, compensatory hyperventilation in a spontaneously breathing patient

Pathophysiology

Respiratory acidosis is caused by an acute increase in PaCO₂, most commonly from respiratory failure. May also occur in hypermetabolic states (e.g., malignant hyperthermia).

Metabolic acidosis is caused by accumulation of acids (e.g., lactic acid, ketoacids, toxins) faster than the liver or kidneys can eliminate them.

DIFFERENTIAL DIAGNOSIS

- Laboratory error
- Diagnostic studies
- Arterial blood gas analysis, lactic acid level, plasma electrolytes, studies to determine the underlying cause (if unknown)
- Subsequent management
- Continue to monitor and treat the underlying cause.

Immediate Management

- Establish large-bore IV access.
- Consider intra-arterial and central venous catheter placement.
- Plasma Volume Expansion
 - Generally lactated Ringer's solution (LR) is preferred to 0.9% normal saline solution NSS because its lower chloride content reduces the risk of hyperchloremic acidosis.
- PaCO₂ Regulation
 - 10 torr decrease in PaCO₂ results in an approximate increase of 0.08 in pH
 - Metabolic acidosis can be buffered acutely by inducing a respiratory alkalosis to raise the pH. Determine the desired change in pH and calculate the necessary change in PaCO₂, then adjust the patient's minute ventilation.

Immediate Management (continued)

- NaHCO_3 Administration
 - Administer NaHCO_3 to rapidly correct severe acidosis ($\text{pH} < 7.2$) using the base deficit derived from an arterial blood gas (ABG):
- NaHCO_3 (mEq) = $0.5 \times \text{weight (kg)} \times (24 - \text{HCO}_3^- \text{ (mEq/L)})$
 - Administer only half the calculated dose and repeat the ABG in 30 minutes to determine the need for additional therapy.
- Maintenance IVF Electrolyte Prescription
 - Administer isotonic crystalloids or colloid solutions as appropriate to maintain normal circulating blood volume.

Risk Factors

- Respiratory acidosis:
 - Respiratory failure
 - Malignant hyperthermia
- Metabolic acidosis:
 - Acute illness
 - Hypoperfusion states (e.g., shock)
 - Hypermetabolic states (e.g., thyrotoxicosis)
 - Massive resuscitation with hyperchloremic fluids (normal saline)
 - Hypothermia
 - Severe diarrhea
 - Diabetic ketoacidosis

Prevention

No specific prevention. Aggressive treatment of underlying cause.

Special Considerations

- Monitor the patient closely during management of acid base disorders and adjust treatment for a change in clinical status. (e.g., aggressive treatment of lactic acidosis after a seizure may cause alkalosis when the seizure ends).
- Do not reduce PaCO_2 to less than 25 torr, as this may produce hypocarbia-induced vasoconstriction and ischemia.
- Consider using colloid solutions (e.g., hetastarch) when large-volume plasma volume expansion is required. (less shift into the extravascular space.)

- Synthetic colloids and fresh frozen plasma (FFP) are better retained in the intravascular space than albumin in the presence of injury, inflammation, and capillary leak. This is due to albumin's lower molecular weight.
- As plasma volume expansion corrects hypoperfusion, lactic acid levels decrease from both hepatic metabolism and reduced generation, and anaerobic metabolism is replaced with aerobic respiration.
- Use of $\frac{1}{2}$ NSS + 75 mEq NaHCO_3 for plasma volume administration and D_5W + 75 mEq NaHCO_3/L in patients with acidosis (especially with preexisting hyperchloremic metabolic acidosis) may be beneficial because of its reduced chloride content relative to plasma.

Further Reading

Fidkowski C, Helstrom J. Diagnosing metabolic acidosis in the critically ill: bridging the anion gap, Stewart, and base excess methods. *Can J Anaesth*. 2009 Mar;56(3):247–256.

Neligan PJ, Deutschman CS. Perioperative acid-base balance. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1557–1572.

Adrenocortical Insufficiency (AI)

Definition

Primary adrenal insufficiency due to destruction of the adrenal cortex, adrenal suppression, or pituitary insufficiency.

Presentation

- Hypotension or shock refractory to fluids and vasopressors
- Hyponatremia, hyperkalemia, and hypoglycemia
- An awake patient may present with abdominal pain and vomiting.

Pathophysiology

Corticotrophin-releasing hormone is released from the hypothalamus and stimulates the pituitary to release ACTH. ACTH then causes the adrenal glands to release cortisol. Cortisol provides negative feedback, inhibiting the release of CRH and ACTH. AI can be caused by destruction of the adrenal cortex (autoimmune or HIV), pituitary disease, or exogenous steroids.

DIFFERENTIAL DIAGNOSIS

- Septic shock
- Acute abdomen

Immediate Management

- Administer fluids to expand plasma volume. (NSS or LR to replete Na⁺ using isotonic fluids)
- Administer dextrose (D₅₀W bolus) to correct hypoglycemia.
- Support blood pressure with inotropes or vasopressors. If resistant to catecholamines, consider vasopressin (0.01–0.04 units/min)
- Administer an initial dose of 100 mg hydrocortisone in the OR. Follow with 50 mg hydrocortisone every 6 hours.

Diagnostic Studies

- Baseline cortisol and ACTH levels (draw **before** administering steroids when there is sufficient time, but do not delay therapy when hypotension is life-threatening!)
- Dexamethasone suppression test may be appropriate outside of the OR
- Plasma electrolytes and glucose
- Imaging studies to rule out acute abdomen

Subsequent Management

Manage the underlying cause of AI.

Risk Factors

- Corticosteroid use for conditions other than primary AI (e.g., transplant patients)
- Corticosteroid use (more than prednisone 20 mg/day or equivalent) for more than 4 weeks within the preceding year)
- Patients being treated for diagnosed AI

Prevention

Careful history should be taken for steroid use before surgery.

Special Considerations

- The efficacy of providing a mineralocorticoid in addition to the large doses of glucocorticoid is controversial.
- Acute AI is more common with patients undergoing emergency surgery for infection-related disorders. Septic shock is a common presenting finding. Examples of patients at risk for acute AI include those with necrotizing soft tissue infection, intra-abdominal sepsis with feculent peritonitis, and mesenteric ischemia.

- Suspect acute AI in older patients who are vasopressor dependent after general surgical procedures despite adequate volume resuscitation.
- Septic patients with hypotension unresponsive to vasopressor therapy may also have acute AI.

Further Reading

Connery LE, Coursin DB. Assessment and therapy of selected endocrine disorders. *Anesthesiol Clin North Am*. 2004 Mar;22(1):93–123.

Alkalosis

Definition

Blood pH greater than 7.45 (normal = 7.36–7.45)

Presentation

Vasoconstriction, pallor, lightheadedness, paresthesias

Pathophysiology

Alkalosis is caused by loss of acid from the extracellular space (e.g., hypochloremia), excessive HCO_3^- loads, or hyperventilation acutely reducing PaCO_2 .

Immediate Management

- Consider intra-arterial catheter placement for severe alkalosis (pH > 7.5) (facilitates ABG analysis).
- Venous blood gas analysis may also be reliable in patients with central venous access and is a reasonable alternative to arterial cannulation.
- PaCO_2 Regulation
 - Buffer metabolic alkalosis by inducing a deliberate acute respiratory acidosis. Adjust minute ventilation to attain a desired pH.
 - PaCO_2 is inversely related to pH by the following formula: A 10 torr increase in PaCO_2 produces an approximate decrease of 0.08 pH
 - Use permissive hypercapnia as necessary to restore pH to a safe physiologic range while ensuring adequate oxygenation.
- For patients who are able to tolerate volume loading, bolus administration of 0.9% normal saline solution (NSS). For those who cannot tolerate the volume, providing an identical amount of chloride using hypertonic saline is equally efficacious.

Diagnostic Studies

Arterial blood gas analysis.

Subsequent Management

Investigation to determine underlying causes.

Risk Factors

- 2nd most common acid-base disorder in hospitalized adults
- Loop diuretic administration
- Severe hypoproteinemia
- Hypocarbica
- Volume contraction
- Hypochloremia
 - Vomiting
 - Proximal enteric fistula
- Iatrogenic causes
 - Hyperventilation of patients with chronic respiratory failure
 - Administration of weak ions (acetate, citrate)

Prevention

Avoid inadvertent hyperventilation.

Special Considerations

- Regardless of cause, alkalosis generally responds to either PaCO₂ regulation or chloride administration.
- Metabolic alkalosis is commonly treated in the ICU with carbonic anhydrase inhibitors (e.g., acetazolamide) but these drugs take too long to act to be of benefit in the acute setting.

Further Reading

Khanna A, Kurtzman NA. Metabolic alkalosis. *J Nephrol.* 2006 Mar-Apr;19(Suppl 9):S86–S96.

Neligan PJ, Deutschman CS: Perioperative acid-base balance. In: Miller RD (ed.). *Miller's Anesthesia.* 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1557–1572.

Anaphylaxis

Definition

An acute, severe, hypersensitivity reaction to a trigger such as an antibiotic or latex.

Presentation

- Urticaria
- Hypotension
- Tachycardia
- Wheezing
- Flushing

Pathophysiology

Massive release of leukotrienes, histamine, and prostaglandins in response to release of IgE from mast cells, causing potentially life-threatening vasodilation, myocardial suppression, and bronchoconstriction.

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Nonimmunologic drug reaction
 - “Red man syndrome” with vancomycin
 - Morphine-induced histamine release

Immediate Management

- Ensure an adequate airway
- Supplemental O₂ (FiO₂ 100% if severe)
- Establish large-bore IV access
- Treat bronchospasm (inhaled albuterol 4–8 metered doses)
- Search for triggering agent
- If mild anaphylaxis
 - Epinephrine 0.1 mg-0.5 SQ or IV every 10–20 minutes
 - Hydrocortisone 100 mg IV
 - Diphenhydramine 50 mg IV (H₁ antagonist)
 - H₂ antagonist (e.g., famotidine 20 mg IV)
- If severe anaphylaxis with hypotension
 - Above interventions **plus**
 - Consider intubation and mechanical ventilation
 - Arterial blood gas for pH, PaCO₂ and PaO₂ assessment
 - Fluid resuscitate with isotonic crystalloid or colloid solutions
 - Epinephrine infusion (titrate to systolic blood pressure)
 - If cardiac arrest occurs, administer epinephrine 1 mg IV and begin advanced cardiac life support (ACLS).
- Perform a careful evaluation of agents administered immediately prior to the anaphylactic event, including antibiotics, latex, and neuromuscular blocking agents.
- Inform the surgeons; consider terminating the procedure

Diagnostic Studies

Clinical diagnosis—no diagnostic studies

Subsequent Management

- Careful review of the patient's chart for known drug allergies
- Prominently document the event to prevent another exposure to the same drug (if identifiable)
- If the trigger is unknown, consider skin testing

Risk Factors

- History of atopy
- History of allergic rhinitis
- Prior exposure to certain drugs (e.g., intravenous contrast)

Prevention

- Review each patient's chart carefully for known allergies.
- Limit the use of latex products in the operating room.

Special Considerations

- Neuromuscular blocking agents are the most common trigger in the perioperative period.
- Latex and perfume allergy (undisclosed) is probably more common than is reported in the medical literature.

Further Reading

Mertes PM, Lambert M, Guéant-Rodriguez RM et al. Perioperative anaphylaxis. *Immunol Allergy Clin North Am.* 2009 Aug;29(3):429–451.

Coagulopathy

Definition

Uncontrolled bleeding resulting from a failure of the coagulation cascade.

Presentation

Diffuse hemorrhage from the surgical wound, areas of dissection (i.e., retroperitoneum), raw organ surfaces after partial resection, catheter insertion sites, or other locations.

Pathophysiology

Failure of a complex interaction between clotting factors, the endothelium, and the platelet surface that must undergo a conformational change. This can be due to a deficiency of one or more factors, the presence of an inhibitor, or the deficiency of ions including Mg^{2+} and Ca^{2+} .

DIFFERENTIAL DIAGNOSIS

- Surgical bleeding
- Medications: warfarin, heparin, fish oil, vitamin E, aspirin, all nonsteroidals
- Herbal preparations: ginko bilboa, ginseng, saw palmetto, garlic
- Liver disease
- Renal disease: chronic renal failure
- Congenital bleeding disorder: von Willebrand's disease, hemophilia
- Acquired coagulation disorders: disseminated intravascular coagulation, immune thrombocytopenia, thrombotic thrombocytopenic purpura, heparin-induced thrombocytopenia, chronic renal failure

Immediate Management

- Establish large-bore IV access.
- Review medications and history.
- Correct factor deficiencies (if laboratory studies are not immediately available, consider empiric administration of fresh frozen plasma and platelets).
 - If warfarin use is suspected, administer 4 units FFP and Vitamin K 10 mg IV. (**Caution: Vitamin K may cause severe hypersensitivity reactions or anaphylaxis!**)
 - Protamine may partially reverse low molecular weight heparin. (Heparin and LMWH are cleared by the kidney.)
 - If aspirin or NSAID use is suspected, transfuse 10 units of platelets (delivers approximately 100,000 functional platelets/ μL).
 - If renal insufficiency, administer 0.3 mcg/kg ddAVP.
 - If PT and aPTT are elevated, administer 4 units FFP.
 - If PT is elevated or liver disease is suspected, administer vitamin K 10 mg IV followed by 4 units FFP.
 - If fibrinogen level is less than 100 mg/dl, administer 10 units of cryoprecipitate.
 - Transfuse platelets if platelet count less than 50,000/ μL (minor procedures such as catheter insertions) or $< 100,000/\mu\text{L}$ (major procedures such as laparotomy with partial colectomy).
- Consider platelet transfusion for unexpected and persistent bleeding from small vessels. (May be due to aspirin or clopidogrel use)
- If diffuse microvascular hemorrhage occurs or the cause is unknown, support the entire cascade including clotting factors,

Immediate Management (*continued*)

platelets, calcium, magnesium, and fibrinogen (if hypofibrinogenemia is present).

- Use Activated Clotting Time (ACT) to assess coagulopathy after intraoperative heparin therapy. Reverse heparin with protamine in patients without a salmon allergy.
- If massive coagulopathy occurs, strongly consider converting to a “damage control” type of procedure to control bleeding and stabilize the patient. Consider initiating a massive transfusion protocol that may include activated factor VII. (See Massive Hemorrhage, Chapter 14.)

Diagnostic Studies

- Complete blood count with platelets
- PT, aPTT
- Fibrinogen level
- Activated clotting time
- Consider thromboelastography if equipment is available.

Subsequent Management

- Continue to monitor coagulation studies.
- Obtain a hematology consultation if the cause of the coagulopathy is unknown or if the massive transfusion protocol is activated and activated factor VII is administered

Risk Factors

- Uncontrolled surgical hemorrhage
- Consumption or dilution of coagulation factors
- Platelet dysfunction or consumption
- Fibrinolysis
- Congenital bleeding disorder
- Disseminated intravascular coagulation
- Unreported use of aspirin or NSAIDs
- Use of “herbal” preparations

Prevention

- Current recommendations for massive transfusion therapy in traumatic injury include minimal use of crystalloid solutions and use of plasma, packed red blood cells (PRBCs) and platelets in a 1:1:1 ratio.

- Patients with specific clotting factor deficiencies should have those factors replaced before surgery and may need ongoing replacement during and after surgery.
- Hypothermia creates a functional clotting deficit by reducing enzyme kinetics. Maintain normothermia for most procedures to the extent that clinical conditions permit.

Special Considerations

- Coagulopathy that occurs in association with acidosis and hypothermia implies a poor prognosis.
- Thromboelastography is sensitive measure of the interaction of all aspects of clotting but is time consuming, may be difficult to interpret, and is neither universally accepted nor commonly used.
- Consider recombinant activated factor VII in unexplained severe coagulopathy. This is an “off-label” use of the drug and is extremely expensive. Efficacy does not correlate with measured PT and aPTT, and undesirable clotting has been reported. The optimal dose for trauma and many other conditions has yet to be established, and Hematology consultation is often helpful.

Further Reading

- Rossaint R, Duranteau J, Stahel PF, Spahn DR. Nonsurgical treatment of major bleeding. *Anesthesiol Clin*. 2007 Mar;25(1):35–48.
- Spahn DR, Rossaint R. Coagulopathy and blood component transfusion in trauma. *Br J Anaesth*. 2005 Aug;95(2):130–139.
- Spinella PC, Holcomb JB. Resuscitation and transfusion principles for traumatic hemorrhagic shock. *Blood Rev*. 2009 Nov;23(6):231–240.

Diabetic Ketoacidosis (DKA)

Definition

The failure of glucose homeostasis in an undiagnosed diabetic, a diabetic with poor compliance, or a compliant diabetic with a stress-inducing condition (most often infection).

Presentation

- 2–3 day history of gradual deterioration, including polydipsia, nausea, vomiting, and abdominal pain.
- Hyperglycemia, ketosis, and acidemia

Pathophysiology

DKA is generally caused by actual or functional insulin deficiency. Fluid, electrolyte, and acid-base balance are affected. Osmotic diuresis causes a severe free-water deficit in almost all patients.

Severe acidosis increases the required minute ventilation for pH buffering.

DIFFERENTIAL DIAGNOSIS

Other conditions that can cause severe metabolic acidosis include:

- Sepsis (lactic acidosis)
- Renal failure
- Hyperosmotic coma (markedly elevated glucose, no ketones)
- Aspirin overdose
- Alcoholic ketoacidosis.

Immediate Management

- Assess airway, breathing, circulation, and level of consciousness.
- Correct acid-base status.
- If pH is less than 7.2, calculate required NaHCO_3 load using base deficit and give 50% IV prior to sedation for ETT placement.
- Correction of lesser degrees of acidosis is controversial but certainly reasonable in patients requiring emergency surgery, as large-volume fluid resuscitation may induce a hyperchloremic metabolic acidosis.
- If the clinical situation permits, consider briefly delaying urgent surgery to resuscitate and improve pH and perfusion in a critical care setting.
- Strongly consider cancelling any elective procedure if the patient presents with DKA in order to correct the metabolic abnormality and the underlying cause.
- Management is guided by serial blood gas and ketone measurements.
- Water and electrolytes
 - Establish large bore IV access.
 - Rapidly resuscitate hypotensive patients with dextrose free, normotonic fluids as boluses.
 - Normotensive patients should receive approximately 2–3 times their maintenance fluid rate.
 - Avoid colloids because they do not correct the free water deficit. Appropriate fluids include 0.9% NSS, LR, and “designer” fluids such as $\frac{1}{2}$ NSS + 75 mEq/L NaHCO_3 (the author’s preference).
 - Assess electrolyte status hourly.
- Insulin Deficiency
 - Establish a continuous intravenous infusion of short-acting insulin. (Suggested initial dose 0.1 unit/kg bolus, then 0.1 unit/kg/h.)

Immediate Management (continued)

- Assess glucose and K^+ at least hourly and adjust infusion as required.
- Electrolyte Disorders
 - Administer KCl 40 mEq slow IV if K^+ is less than 3.0 mg/dL.
 - Replace Mg^{2+} as $MgSO_4$ 1 g IV
 - Check electrolyte levels at least hourly
 - Consider replacing some potassium as KPO_4 to avoid hypophosphatemia.

Diagnostic Studies

- Serial arterial blood gas analysis, lactic acid level
- Serial plasma electrolyte and ketone levels
- To determine possible etiology:
 - Troponin (myocardial ischemia)
 - Complete blood count, blood cultures (sepsis)

Subsequent Management

- Add dextrose to IV solution and reduce to standard as appropriate for maintenance fluid when the glucose falls below 250 mg/dL.
- Begin subcutaneous insulin sliding scale when stable.

Risk Factors

- Usually occurs in type I diabetics, but may occur in type II diabetics when under stress
- Discontinuation of insulin in a diabetic patient
- Acute illness in a diabetic patient
- Emergency surgery

Prevention

Early detection and treatment of diabetes mellitus. Early intervention with insulin even in non-insulin-requiring diabetics who are acutely ill and/or undergoing surgery.

Special Considerations

- Avoid subcutaneous insulin administration in patients with DKA because absorption is variable.
- Record and manage on an hourly basis: insulin infusion rate, fluids, electrolyte levels and replacement, glucose, urine, ABGs.

- Hyperglycemia and dehydration may induce hyponatremia that is often artifactual and insignificant.
- Severe hypokalemia may induce life-threatening dysrhythmias. K^+ and Mg^{2+} are cotransported and should be simultaneously repleted.
- Hypocalcemia may also occur but is overshadowed in frequency by hypophosphatemia. Acute respiratory compromise occurs when phosphate level is less than 1 mg/dL.
- Delivering totals of 200 mEq K^+ and 12 grams of Mg^{2+} is not atypical in severe DKA.

Further Reading

Kohl BA, Schwartz S. Surgery in the patient with endocrine dysfunction. *Med Clin North Am.* 2009 Sep;93(5):1031–1047.

Hypercalcemia

Definition

- Normal calcium is 8.5–10.5 mg/dL (ionized 2.0–2.5 mEq/L)
- Mild: 11–14 mg/dL
- Severe: Greater than 14 mg/dL

Presentation

- Incidental laboratory finding
- Mental status changes
- Hyperreflexia
- Hypertension
- Bradycardia
- Vomiting
- Polyuria
- Renal calculi
- Oliguric renal failure

Pathophysiology

Hypercalcemia is most commonly a manifestation of secondary hyperparathyroidism, paraneoplastic syndrome, immobility, or lithium toxicity.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- IV fluid resuscitation (generally NSS)
- In patients with normal renal function, administer furosemide 40 mg IV
- Hemodialysis in patients with renal failure
- Calcium channel blockade (e.g., diltiazem, nifedipine), or
- Beta-blockade (e.g., metoprolol, labetalol)

Diagnostic Studies

- Plasma electrolyte levels
- Metabolic panel (including liver function tests)
- Amylase
- Electrocardiogram

Subsequent Management

- Bisphosphonate therapy
- Calcimimetic agents (i.e., cinacalcet),
- Calcitonin therapy
- Investigate underlying cause if unknown
 - Parathyroid hormone levels
 - Workup for malignancy
- Follow amylase levels until Ca^{2+} returns to normal

Risk Factors

- Malignancy (breast, lung, kidney, multiple myeloma, lymphoma)
- Hyperparathyroidism
- Kidney failure
- Granulomatous diseases: tuberculosis, sarcoid

Prevention

Maintain adequate hydration and urine output with sodium-containing fluids in vulnerable patients.

Special Considerations

- Avoid thiazide diuretics in patients at risk for hypercalcemia.
- Patients with muscle weakness should receive reduced doses of neuromuscular blocking agents.
- Hypercalcemia can cause pancreatitis.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1711–1712.

Hyperkalemia

Definition

- Normal potassium is 3.5–5.5 mEq/L.
- Mild: 5.5–6.0 mEq/L
- Moderate: 6.0–7.0 mEq/L
- Severe: More than 7.0 mEq/L

Presentation

- Incidental laboratory finding
- Nausea, vomiting
- Muscle weakness, paresthesias
- Peaked T waves on ECG indicate impending cardiac arrest. The degree of hyperkalemia loosely correlates with the height of the T wave elevation.

Pathophysiology

High plasma concentration of potassium impairs myocardial conduction and may potentially result in cardiac arrest.

DIFFERENTIAL DIAGNOSIS

- Improper sample handling (cell lysis)
- May be artificially elevated in severe leukocytosis or thrombocytosis

Immediate Management

- Establish IV access and ECG monitoring
- Administer CaCl_2 (**not calcium gluconate**) 1 IV
- Administer 10 units of regular insulin IV with 50 cc of D_{50}W IV
- Force renal excretion
 - 0.9% NSS 2000 cc
 - Furosemide 20–40 mg IV (depending on baseline creatinine) between the 1st and 2nd liters of saline

Diagnostic Studies

- Plasma electrolytes (used to follow treatment progress)
- Electrocardiogram

Subsequent Management

Reduce the total body K^+ load with a cation exchange resin (e.g., sodium polystyrene [Kayexelate]) suspended in sorbitol. Consider this therapy in patients with abnormal renal function or renal failure. Induction of diarrhea indicates effective therapy.

Risk Factors

- Iatrogenic (inadvertent overdose, massive transfusion)
- Renal failure
- Diabetic ketoacidosis
- Succinylcholine use in patients with upper motor neuron disorders or severe burn injury
- Cell destruction: skeletal muscle crush, massive tissue necrosis, massive hemolysis

Prevention

- Avoid iatrogenic causes
- Monitor K^+ carefully in patients with risk factors

Special Considerations

- Therapy addresses three major aims: (1) supporting myocardial polarization and depolarization; (2) relocating potassium from the plasma space to the intracellular space; and (3) reducing the total body potassium load.
- Consider acute hemodialysis in patients with life-threatening hyperkalemia (generally $K^+ > 7.0$ mEq/L) or renal failure.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1710

Hypermagnesemia

Definition

Mg^{2+} level greater than 2.5 mEq/L. (Normal Mg^{2+} level 1.5-2.0 mEq/L.)

Presentation

- Muscle weakness
- Bradycardia with widened QRS complex, prolonged PR interval
- Hypotonia

- Hyporeflexia
- Acute respiratory failure

Pathophysiology

Most commonly caused by excessive intake of Mg^{2+} (especially laxatives) in patients with renal impairment. High magnesium levels depress the central nervous system and interfere with neurotransmitter release.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Intubate and initiate mechanical ventilation if indicated.
- Control the underlying cause (i.e., stop the infusion).
- Administer $CaCl_2$ 1 g IV over 5 minutes (temporarily reverses effects of Mg^{2+}).
- Expand plasma volume with an isotonic solution (LR or NSS).
- Administer furosemide 20 mg IV.
- Acute hemodialysis in severe hypermagnesemia or in patients with renal failure.

Diagnostic Studies

Plasma Mg^{2+} , electrolytes, BUN, creatinine

Subsequent Management

Monitor Mg^{2+} levels as appropriate.

Risk Factors

- Aggressive treatment of pre-eclampsia
- Use of Mg^{2+} containing antacids or laxatives

Prevention

Monitor Mg^{2+} levels carefully when using it therapeutically.

Special Considerations

- Magnesium and calcium antagonize each others' effects.
- Mg^{2+} will produce profound muscle weakness in patients with myasthenia gravis or Lambert-Eaton syndrome.
- Mg^{2+} prolongs the action of neuromuscular blocking agents.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1713–1714.

Whang R, Hampton EM, Whang DD. Magnesium homeostasis and clinical disorders of magnesium deficiency. *Ann Pharmacother*. 1994 Feb;28(2):220–226.

Hypernatremia

Definition

- Normal sodium is 135–145 mEq/L
- Mild: 145–150 mEq/L
- Moderate: 150–160 mEq/L
- Severe: Greater than 160 mEq/L

Presentation

- Profound dehydration
- Laboratory finding
- Central nervous system symptoms: confusion, seizures, focal neurologic deficits
- Severe hypernatremia: brain shrinkage causing intracerebral hemorrhage

Pathophysiology

Either excessive free water loss or excessive sodium administration. Free water loss is the most common cause.

DIFFERENTIAL DIAGNOSIS

- Laboratory error
- Acute liver failure (use of lactulose or mannitol)

Immediate Management

- Hypovolemic hypernatremia:
 - Administer fluids to correct hypovolemia
 - .45% saline or D₅W to correct Na⁺
- Normovolemic hypernatremia:
 - Free water (.45% saline or D₅W to correct Na⁺)
 - Correct underlying cause (DDAVP for diabetes insipidus)
- Hypervolemic hypernatremia:
 - Discontinue Na⁺ containing solutions
 - Consider furosemide 20 mg IV or as appropriate for the patient's renal function

Diagnostic Studies

- Plasma electrolytes and osmolality
- Urine electrolytes and osmolality

Subsequent Management

- Monitor electrolyte intake carefully.
- Determine the cause of hypernatremia and treat appropriately.

Risk Factors

- Hypovolemic patients: open wounds, GI losses, insufficient ACTH, excessive mannitol administration, loop diuretic use in conjunction with a salt restricted diet
- Euvolemic patients: diabetes insipidus (nephrogenic or central), lithium toxicity
- Hypervolemic patients: iatrogenic (NaHCO_3 or hypertonic saline administration)

Prevention

Follow plasma electrolyte levels closely when administering large quantities of sodium-containing fluids.

Special Considerations

- NOTE: Correct half of the deficit over 8 hours and the remainder over the subsequent 16 hours
- Free water deficit may be calculated using the following equation:

$$\text{Free H}_2\text{O deficit} = \text{Body water (L/kg)} \times \text{weight (kg)} \times ((\text{serum Na}^+/140)-1)$$

- Estimate body water as 0.6 L/kg in males and 0.5 L/kg in females.

Further Reading

Bagshaw SM, Townsend DR, McDermid RC. Disorders of sodium and water balance in hospitalized patients. *Can J Anaesth.* 2009 Feb;56(2):151–167.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia.* 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1706–1709.

Hypocalcemia

Definition

Calcium less than 8.5 mg/dl (ionized less than 2.0 mEq/L).

Presentation

- Mental status changes
- Tetany
- Chvostek and Trousseau signs
- Laryngospasm
- Hypotension
- Prolongation of Q–T interval or heart block
- Dysrhythmias

Pathophysiology

Ca²⁺ mediates muscle contraction, multiple endocrine functions, and the transport and secretion of fluids and electrolytes.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Administer CaCl₂ 1 g IV over 5 minutes
- Repeat as needed to return ionized Ca²⁺ levels to normal, or for symptoms to abate.

Diagnostic Studies

- Plasma electrolytes
- Metabolic panel (including protein and liver function tests)
- Parathyroid hormone level

Subsequent Management

- Investigate the cause of hypocalcemia if unknown.
- Maintain normocalcemia with calcium gluconate g IV by bolus or continuous infusion (less irritating to veins).

Risk Factors

- Acute hyperventilation
- Rapid transfusion of citrated blood products (1.5 mL/kg/min)
- Low albumin levels (critically ill patient, sepsis, burns, acute renal failure)
- Hyperparathyroidism
- Sepsis
- Thyroidectomy or parathyroidectomy
- Acute panhypopituitarism
- Hypomagnesemia

Prevention

- Common practice is empiric administration of 500 mg CaCl_2 per 8–10 units of PRBC during rapid transfusion.
- Alternatively, measure ionized Ca^{2+} every 5 units and treat as necessary.

Special Considerations

- CaCl_2 provides immediately available calcium. Calcium gluconate requires degluconation before the calcium is biologically available.
- Hypocalcemia may be accompanied by hypomagnesemia when caused by large-volume resuscitation with isotonic saline.
- If ionized calcium measurement is unavailable, correct for hypoalbuminemia using the relationship that a 1 g/dL decrease in albumin causes a 0.8 mg/dL drop in total Ca^{2+} ; therefore:

$$\text{Corrected Ca}^{2+} \text{ (mg/dL)} = \text{Measured Ca}^{2+} \text{ (mg/dL)} + 0.8 \times (4 - \text{albumin (g/dL)})$$

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1711–1712.

Hypokalemia

Definition

- Normal potassium is 3.5–5.5 mEq/L.
- Mild: 3.0–3.5 mEq/L
- Moderate: 2.5–3.0 mEq/L
- Severe: Less than 2.5 mEq/L

Presentation

- Severe weakness.
- ECG may show prolonged PR interval, ST segment depression, inverted T waves, or U waves.

Pathophysiology

Arrhythmia is the most common consequence of hypokalemia, and includes atrial fibrillation and premature ventricular contractions.

DIFFERENTIAL DIAGNOSIS

- Hypocalcemia
- Hypomagnesemia
- Cushing syndrome (manifestations of hypercortisolemia)

Immediate Management

- **Slowly** administer KCl 20 mEq IV.
- **Limit KCl infusion to 20 mEq/h except in the setting of life-threatening dysrhythmias with careful ECG monitoring.**
- The patient must have ECG monitoring.
- Consider administration through a central venous catheter because KCl-containing solutions are extremely irritating.
- Consider withholding potassium-wasting diuretics (e.g., furosemide) or switching to potassium-sparing diuretics (e.g., spironolactone).

Diagnostic Studies

- Plasma electrolytes including magnesium (renal cotransport)

Subsequent Management

- Consider postoperative telemetry monitoring after large doses of potassium have been administered.

Risk Factors

- Plasma volume expansion
- Normal postoperative diuresis
- Diuretic administration
- Insulin administration
- Alkalosis (including respiratory alkalosis due to hyperventilation)
- GI losses (diarrhea, vomiting, intestinal suction, ileal bladder)
- Salt-wasting nephropathies
- Beta-agonist use

Prevention

Monitor plasma electrolytes carefully when patients are at risk for hypokalemia.

Special Considerations

- Mild or moderate hypokalemia rarely requires emergency treatment, although K^+ is generally a part of IV maintenance fluids.
- Severe hypokalemia may require as much as 200–300 mEq of potassium to restore normal plasma and intracellular and may require several days of intravenous therapy to correct.
- Potassium is principally an intracellular cation. Low plasma levels imply that the intracellular store is depleted because transmembrane raises plasma levels.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1710.

Hypomagnesemia

Definition

Mg²⁺ level less than 1.5 mEq/L. (Normal Mg²⁺ level 1.5 - 2.0 mEq/L.)

Presentation

- Central nervous system: mental status change, tetany, Chvostek and Trousseau signs
- Hypertension and angina (coronary artery spasm)
- Torsade des pointes, ventricular tachycardia, ventricular fibrillation
- Dysrhythmias

Pathophysiology

Hypomagnesemia is commonly associated with hypokalemia and with large-volume plasma volume expansion.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- For acute arrhythmias:
 - Monitor blood pressure and heart rate
 - Administer MgSO₄ 2–4 g IV over 5 minutes.
- To correct a deficit found on laboratory studies:
 - Administer MgSO₄ 2–4 g IV over 15 minutes.

The amount of replacement can be estimated from the plasma level:

Mg level	Replacement dose (IV)
2.0–2.25	2.0 gm
1.75–1.9	4.0 gm
1.5–1.74	6.0 gm
1.25–1.49	8.0 gm
1.0–1.24	10.0 gm

Diagnostic Studies

Plasma Mg²⁺ level, plasma electrolyte levels

Subsequent Management

Follow Mg^{2+} level and maintain within normal range.

Risk Factors

- Hypokalemia
- Large volume plasma volume expansion
- Mild hypomagnesemia is normal in athletes, pregnancy, and other hypermetabolic states.
- Renal losses: loop diuretics, diuretic phase of acute tubular necrosis
- Common in critically ill patients

Prevention

Monitor magnesium levels carefully during resuscitation.

Special Considerations

- Hypomagnesemia increases the risk of perioperative arrhythmias.
- Hypomagnesemia is associated with bronchial smooth muscle constriction. Supplementation may be a useful modality for those with reactive airway disease.
- Maintaining plasma Mg^{2+} approximately 2.5 mg/dL is helpful in atrial dysrhythmia management as well.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1712–1713.

Whang R, Hampton EM, Whang DD. Magnesium homeostasis and clinical disorders of magnesium deficiency. *Ann Pharmacother*. 1994 Feb;28(2):220–226.

Hyponatremia

Definition

- Normal sodium is 135–145 mEq/L.
- Mild: 125–134 mEq/L
- Moderate: 120–124 mEq/L
- Severe: Less than 120 mEq/L

Presentation

Depends on patient's fluid status. Symptoms may include headache, ataxia, altered mental status, nausea, vomiting.

Pathophysiology

Most surgical patients have a dilutional hyponatremia and not true total body Na^+ deficiency. Dilutional hyponatremia is characterized

by low Na^+ but nearly normal Cl^- accompanied by dilute urine and a normal to high urine Na^+ .

DIFFERENTIAL DIAGNOSIS

- Laboratory error
- Sample dilution (drawing from the same arm as an IV with hyponatremic fluid)
- Hyperglycemia

Immediate Management

- Establish IV access.
- Monitor ECG or hemodynamics if indicated.
- In patients with dilutional hyponatremia:
 - Free water restriction
 - Furosemide 20–40 mg IV if patient is hypervolemic.
 - For severe, symptomatic hyponatremia, consider 3% hypertonic saline
- Calculate sodium deficit using the formula:

Sodium deficit (mEq) = $0.6 \times \text{body weight (kg)} \times (\text{pt's Na}^+ - \text{desired Na}^+)$

Diagnostic Studies

Plasma electrolyte levels

Subsequent Management

After acute correction to 125 mEq/L over the first 8 hours, additional correction should be limited to 0.5 mEq/L per hour.

CAUTION: Rapid correction may lead to central pontine myelinolysis, an irreversible demyelinating disorder that causes permanent neurologic injury.

Risk Factors

- Excess free water administration (resuscitation with D5 ½ NS)
- Absorption of irrigation fluid (TURP syndrome, endoscopic GU or GYN procedures)
- Thiazide diuretic administration
- Adrenocortical insufficiency
- Syndrome of inappropriate antidiuretic hormone (SIADH)
- Liver failure

Prevention

Measure plasma electrolytes frequently

Special Considerations

- Total body water is 0.6 L/kg in males and 0.5 L/kg in females
- When ADH antagonist therapy (i.e., aquaretics) is readily available in the US, those agents will replace furosemide, because ADH induces free water loss with minimal changes in solute excretion.

Further Reading

Bagshaw SM, Townsend DR, McDermid RC. Disorders of sodium and water balance in hospitalized patients. *Can J Anaesth.* 2009 Feb;56(2):151–167.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD (ed.). *Miller's Anesthesia.* 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1706–1709.

Hypothermia

Definition

A mild (32–35° C) or severe (less than 32° C) decrease in temperature.

Presentation

- 32–35° C
 - Lethargy and confusion
 - Weakness
 - Shivering
- 28–32° C
 - Myocardial depression
 - Dysrhythmias
 - Metabolic acidosis (may get worse on rewarming)
 - Hyperkalemia
 - Coagulopathy
- Less than 28° C
 - Unconsciousness
 - Electrically silent EEG (if less than 18° C)
 - Ventricular fibrillation or cardiac arrest

Pathophysiology

Heat is lost in a patient with impaired thermal regulation (e.g., after injury or drug overdose). During surgery, heat is transferred from the core to the periphery due to anesthesia-induced vasodilation. Heat is also lost during surgery through radiation, evaporation, and convection. Cold-induced diuresis may lead to profound intravascular volume depletion.

DIFFERENTIAL DIAGNOSIS

Myxedematous coma (hypothyroidism)

Immediate Management

- Airway, breathing, circulation.
- Establish an airway and ventilate with 100% O₂.
- If necessary, begin resuscitation according to ACLS guidelines. (**NOTE: The patient may not respond until core temperature reaches 30° C.**)
- Consider Mg²⁺ for dysrhythmias.
- Actively rewarm the patient.
- Mild to moderate hypothermia:
 - Warm IV fluids
 - Warming blankets
 - Forced-air warmers.
- Severe hypothermia:
 - In addition to above:
 - GI lavage via NGT
 - Body cavity lavage (in particular bladder, and on occasion abdomen)
 - Consider cardiopulmonary bypass support and rewarming (**best option**)
- In severe hypothermia, active surface warming may be dangerous. Warming-induced peripheral vasodilation in the setting of hypovolemia may lead to life-threatening hypotension.

Diagnostic Studies

- Monitor core temperature.
- Send thyroid function tests if hypothyroidism is suspected.

Subsequent Management

- It may be necessary to buffer “washout acidosis” with sodium bicarbonate during rewarming.
- Consider limiting intraoperative time to less than 2 hours in patients who arrive with or develop hypothermia.

Risk Factors

- Prolonged exposure to cold ambient environment (e.g., after injury).
- Immersion in cold water.
- Impaired level of consciousness.
- Failure to actively warm the patient during surgery (especially trauma, craniofacial procedures, extensive body cavity surgery)
- Acute alcohol intoxication
- Major tranquilizers (suppress shivering)

Prevention

- Use active warming devices in the OR and preoperative holding area.
- Warm the OR.
- Cover the patient to the extent possible.
- Place warming pads on the OR bed.
- Use a forced air warming device during the surgical procedure.
- Use active warming devices for fluids.

Special Considerations

- Patients who are hypothermic on arrival in the OR are generally suffering from sepsis, environmental exposure, or injury with acute hemorrhage. In each circumstance, take precautions to prevent further heat loss.
- Shivering, a common sign of core hypothermia is not typically seen in the OR due to the use of anesthetics (which blunt the response to hypothermia) and neuromuscular blocking agents.
- Separation from cardiopulmonary bypass may require norepinephrine, epinephrine, or other vasoactive drugs.
- Temperature monitoring is recommended as part of the ASA Guidelines on Intraoperative Monitoring.

Further Reading

Insler SR, Sessler DI. Perioperative thermoregulation and temperature monitoring. *Anesthesiol Clin*. 2006 Dec;24(4):823–837.

Malignant Hyperthermia (MH)

Definition

A relatively rare inherited disorder of skeletal muscle that causes a hypermetabolic response to a triggering anesthetic agent. Characterized by hyperthermia, body rigidity, and increased CO₂ production, it may be accompanied by cardiovascular collapse or hypertension.

Presentation

- Tachycardia
- Hypertension
- Increased CO₂ production (absorbent canister may become warm)
- Muscular rigidity (especially masseter muscle spasm)
- Hyperthermia is a late sign.

Pathophysiology

Inborn error of calcium metabolism in skeletal muscle.

DIFFERENTIAL DIAGNOSIS

- Light anesthesia (hypertension and tachycardia without CO₂ production)
- Thyrotoxicosis

Immediate Management

- Call for help!
- Immediately discontinue triggering anesthetic agent(s) (succinylcholine and potent volatile anesthetics—N₂O is safe)
- Increase FiO₂ to 100%.
- Hyperventilate the patient with a new anesthesia circuit.
- Establish large-bore IV access.
- Insert an arterial catheter (blood pressure monitoring and frequent blood gas measurements for acid-base status).
- Expand plasma volume with 15 cc/kg per bolus × 3 using cool fluids.
- Administer sodium dantrolene (2.5 mg/kg IV until signs and symptoms are controlled).
- Cool the patient aggressively with cold IV solutions. Ask the surgeon to lavage any open body cavity/wound surface with cold irrigating solution. Insert a nasogastric tube and irrigate with ice-cold solution.
- Terminate the surgical procedure as quickly as possible using intravenous anesthetics only.

Diagnostic Studies

- Frequent arterial blood gas and plasma electrolyte determination
- Diagnosis may be confirmed obtaining a muscle biopsy for caffeine halothane contracture testing (CHCT) or genetic testing at a separate sitting.

Subsequent Management

- Maintain urine output at least 2 mL/kg to minimize risk of renal tubular injury from rhabdomyolysis.
- Severe metabolic acidosis may require management with sodium bicarbonate.
- Continue dantrolene 1 mg/kg every 4–6 hours for 36 hours after the episode, because the recurrence rate is 25%.

- Discuss the episode with the patient's family and add the patient to the MH national database maintained by the Malignant Hyperthermia Association of the United States (www.mhaus.org).

Risk Factors

- Use of triggering agents (succinylcholine, potent volatile anesthetics) in a patient with a known history of MH.
- Family history, especially an unexplained death of a relative during an anesthetic.
- **MH may occur despite prior uneventful exposure to triggering agents.**
- Rate of occurrence is approximately 1 in 10,000 patients.

Prevention

Avoid the use of triggering agents in patients with a known history of MH or a suggestive family history.

Special Considerations

- Sodium dantrolene inhibits calcium egress from the sarcoplasmic reticulum, and thus serves to restore a normal myocyte calcium concentration.
- Do not administer calcium channel blockers. In the presence of dantrolene, these drugs may cause hyperkalemia and cardiac arrest.
- Because MH is an inherited disorder, consider testing family members for susceptibility.

Further Reading

Benca J, Hogan K. Malignant hyperthermia, coexisting disorders, and enzymopathies: risks and management options. *Anesth Analg*. 2009 Oct;109(4):1049–1053.

Pheochromocytoma

Definition

A catecholamine secreting chromaffin cell tumor usually found in the adrenal medulla.

Presentation

- Sustained or paroxysmal hypertension, tachycardia, and tachydysrhythmias
- History of headaches, chest pain, and palpitations
- Myocardial ischemia

- Acute crisis may occur during induction of anesthesia or surgical manipulation of the tumor.

Pathophysiology

Excessive catecholamines secreted by a chromaffin cell tumor cause tachycardia and vasoconstriction. Cardiac manifestations may be due to increased myocardial oxygen demand or to catecholamine-induced myocarditis.

DIFFERENTIAL DIAGNOSIS

- Light anesthesia
- Malignant hyperthermia (fever, mixed respiratory and metabolic acidosis)
- Malignant hypertension
- Cocaine or amphetamine use (toxicology screen)
- Thyrotoxicosis (fever, acidosis, tachycardia)

Immediate Management

- Immediately discontinue any noxious stimulation.
- Deepen the anesthetic with opioids or potent volatile anesthetics (volatile anesthetics cause vasodilation and myocardial depression, which will help to lower the blood pressure).
- Return the blood pressure to a safe level.
 - Insert an intra-arterial catheter to monitor blood pressure.
 - Sodium nitroprusside infusion (start at 1 mcg/kg/min), fenoldopam infusion (0.2–0.8 mg/kg/min), nicardipine infusion (start at 5 mg/h).
 - Labetalol (20–40 mg IV every 10 minutes), or esmolol infusion (500 mcg/kg IV, then 50 mcg/kg/min titrated to heart rate). **Use beta-blocking agents to control heart rate only after adequate control of blood pressure.** Beta blockade combined with unopposed alpha-adrenergic activity may lead to severe hypertension and vasoconstriction.
 - Titrate medications to return the blood pressure to the patient's baseline.
- Administer fluids as necessary.
- Discontinue surgery as soon as practical.
- If myocardial ischemia refractory to beta-blockade is suspected, nitroglycerine should be used with caution.

Diagnostic Studies

- Plasma free metanephrines and urinary fractionated metanephrines (highest sensitivity)
- Urinary vanillylmandelic acid (highest specificity)
- Subsequent management
- Careful postoperative blood pressure monitoring (consider transfer to the SICU)
- Consider Mg^{2+} (40–60 mg/kg bolus), then infusion 2 g/h. (Magnesium inhibits catecholamine release and has antiarrhythmic and vasodilator effects.)
- Endocrine workup to confirm diagnosis, including 24-hour urine collection for free catecholamines.

Risk Factors

- Associated with multiple endocrine neoplasia (MEN) type 2 (pheochromocytoma, medullary thyroid carcinoma, hyperparathyroidism), von Hippel-Lindau syndrome, or neurofibromatosis type 1.

Prevention

- Careful preoperative workup in patients with unexplained symptoms suggestive of pheochromocytoma.
- Careful manipulation of the adrenal glands in patients with known pheochromocytoma.

Special Considerations

- Labetalol is a vasodilator in addition to its cardiac effects and is preferred over metoprolol or other cardio-specific beta-blockers.
- Hydralazine causes reflex tachycardia and should not be used alone.
- Avoid venodilators and diuretics in the unanticipated pheochromocytoma patient. These patients generally have reduced intravascular volume due to renal excretion of both salt and water. Reducing preload with venodilation or attempted diuresis may be ineffective.
- Patients who have undergone preoperative preparation for resection of a known pheochromocytoma generally have an adequate circulating blood volume.

Further Reading

Connery LE, Coursin DB. Assessment and therapy of selected endocrine disorders. *Anesthesiol Clin North Am*. 2004 Mar;22(1):93–123.

Porphyria

Definition

A family of related enzyme disorders of heme pathway intermediates that cause pathologic accumulation of porphyrins or porphyrin precursors. Classified as erythropoietic (bone marrow) or hepatic (liver) depending on where the accumulation occurs. The three most common types are porphyria cutanea tarda, erythropoietic porphyria, and acute intermittent porphyria (AIP). AIP is the porphyria of relevance to anesthesiologists.

Presentation

- Porphyria cutanea tarda: skin blistering related to sun exposure.
- Erythropoietic porphyria: skin lesions, occasional hemolysis, and rarely liver failure.
- Acute Intermittent Porphyria:
 - Abdominal pain (possibly related to autonomic neuropathy)
 - Sympathetic hyperactivity
 - CNS symptoms including confusion, hallucinations, seizures, autonomic neuropathy
 - Progressive polyaxonal motor neuropathy that may lead to acute postoperative respiratory failure and reintubation. (An anesthetized patient may fail to regain motor function after a long procedure)
 - Reddish colored urine that darkens after exposure to light occurs during an acute attack.
 - In between attacks, diagnosis is difficult, but laboratory testing is particularly helpful during an attack.

Pathophysiology

The heme precursors are believed to be neurotoxic; delta-aminolevulinic acid and porphobilinogen are two characteristic intermediates in the heme biosynthetic pathway that are increased during acute attacks of porphyria. None of these elements accumulate in significant quantity in between attacks.

DIFFERENTIAL DIAGNOSIS

- Acute abdomen (may present with severe abdominal pain).
- May mimic Guillain-Barre syndrome.

Immediate Management

- Withdraw any triggering agents.
- Begin aggressive hydration with dextrose-containing fluids.
 - Administer a bolus of D₅₀W. Begin D₁₀W 1 mL/kg/h to deliver at least 3 L per day.
- Anticipate cardiovascular instability.
 - Consider beta-blockade (treats hypertension and tachycardia and may decrease ALA synthetase activity).
- If a triggering drug has been used:
 - Immediately discontinue use of that drug.
 - Give oral carbohydrates or intravenous D₁₀W infusion (200 kCal/24 h).
 - Monitor closely, with daily urine porphyrin levels.

Diagnostic Studies

- Urine porphobilinogen (diagnostic of an acute attack).
- Frequent plasma Na⁺, K⁺, and Mg²⁺ levels during acute attack.
- Measurement of both porphyrin precursors and porphyrins in RBC, plasma, and/or urine will help to make the diagnosis in the OR or PACU.

Subsequent Management

- A hematology consultation is strongly recommended for acute diagnosis and management.
- Administer hemin (Panhemin®); Lundbeck Inc., Deerfield, IL, 3–4 mg/kg delivered as a single daily dose. Large-bore peripheral or central IV administration is preferred.
- Admit patients with acute porphyria.
- Genetic testing is helpful for diagnosis confirmation and family analysis, but is not useful in the acute setting.

Risk Factors

- Drugs: Alcohol, angiotensin converting enzyme inhibitors, anticonvulsants except gabapentin, barbiturates, calcium channel blocker (**especially nifedipine**), ergots, etomidate, progesterone, sulfonamide antibiotics (**NOT A COMPLETE LIST**)
- Reduced caloric intake (especially carbohydrates)
- Dehydration

Prevention

- Avoid use of known triggering agents.
- Patients with a family history of porphyria should be scheduled early in the day and undergo plasma volume expansion prior to elective procedures.
- Consider regional anesthesia if feasible and after evaluating the patient's neurologic status. There is no evidence to suggest that local anesthetics can trigger an acute attack.

Special Considerations

- Propofol, ketamine, and benzodiazepines are probably safe.
- Inhaled anesthetics, including N₂O are considered safe.
- Neuromuscular blocking agents (including succinylcholine) are widely considered to be safe but should be used with caution.
- Morphine, fentanyl, and sufentanil are safe.
- In general, a single exposure to even potent inducers may be tolerated, but repeat exposure or use during an acute attack is considered to be unsafe.
- Acute blood loss does not seem to provoke a porphyric attack.
- Attacks may last for days, generally followed by complete recovery.
- A missed diagnosis of porphyria may lead to a nontherapeutic laparoscopy or laparotomy, although frank peritonitis is quite rare.
- Anhematin restores normal hepatic heme levels and suppresses ALA synthetase activity.

Further Reading

James MF, Hift RJ. Porphyrias. *Br J Anaesth*. 2000 Jul;85(1):143–153.

Sickle Cell Crisis

Definition

An inherited disorder characterized by abnormal hemoglobin structure that undergoes a conformational change at low oxygen tension, resulting in a characteristic sickle shape to the cells.

Presentation

- Painful crisis (especially bones or joints)
- Chest pain with possible respiratory failure
- Abdominal pain (GI dysfunction, liver or spleen infarction)
- Jaundice, hematuria, gallstones
- Priapism

Pathophysiology

Deoxyhemoglobin S polymerizes, distorting RBCs and causing hemolysis and capillary thrombosis. This leads to chronic anemia and vaso-occlusive disease.

DIFFERENTIAL DIAGNOSIS

Acute abdomen (e.g., appendicitis). Sickle crisis may cause an acute abdomen.

Immediate Management

- Therapy is directed at restoring plasma volume and RBC flow as well as enhancing O_2 delivery.
- Increase FiO_2 to 100%
- Administer analgesics (morphine or hydromorphone) as necessary to control pain.
- Plasma volume expansion with crystalloid or colloid solutions

Diagnostic Studies

- Complete blood count (sickle bodies)
- Urinalysis
- Chest X-ray

Subsequent Management

Exchange transfusions to reduce the hemoglobin-S concentration may be indicated in the setting of severe anemia or acute CNS ischemia.

Risk Factors

- 8% of African-Americans are heterozygous and have sickle cell trait. Approximately 1 in 600 has sickle cell disease.

Prevention

- Maintain normothermia
- Maintain adequate hydration.
- Avoid acidosis.
- Preoperative exchange transfusion is controversial, with no improvement in outcome.

Special Considerations

- Although patients homozygous for Hgb-S (sickle cell disease) are more likely to have an acute crisis, heterozygous patients (sickle trait) may also have crises.
- Patients with sickle cell disease often have chronic pain and may be habituated to opioids. High doses of multiple analgesics are

usually required for optimal analgesia. Postoperative pain control may be difficult and require multimodal therapy.

- Pulse oximetry may underestimate SaO₂ by 2% due to a high concentration of methemoglobin.

Further Reading

Firth PG. Anesthesia and hemoglobinopathies. *Anesthesiol Clin*. 2009 Jun;27(2):321–336.

Thyroid Storm

Definition

A hypermetabolic state characterized by high levels of circulating catecholamines driven by excessive thyroid hormone production and release.

Presentation

- Hyperthermia
- Tachycardia (with or without dysrhythmia and ischemia)
- Hypertension
- Tremulousness
- High fever (105–106 F)
- Diaphoresis

Pathophysiology

A state of severe sympathetic overactivity in the setting of clinical hyperthyroidism, generally precipitated by the stress of surgery or infection.

DIFFERENTIAL DIAGNOSIS

- Malignant hyperthermia (mixed metabolic and respiratory acidosis, creatine kinase is elevated in MH)
- Pheochromocytoma (no fever)

Diagnostic studies

- T₃, free T₄, TSH
- Plasma electrolytes

Immediate Management

- Establish large-bore peripheral IV or central venous access.
- Begin fluid resuscitation.
- Administer beta blockers: labetalol 20–40 mg IV every 10 minutes, metoprolol 5 mg IV every 10 minutes, or esmolol 80 mg over 5 minutes, then 150 mcg/kg/min infusion.

Immediate Management (*continued*)

- Administer glucocorticoids (possibility of adrenal insufficiency; glucocorticoids block conversion of T_3 to T_4): dexamethasone 4 mg IV every 6 hours or hydrocortisone 100 mg IV every 6 hours.
- Supplement Mg^{2+} as needed.
- Administer acetaminophen or NSAIDs to decrease fever.
- If necessary, institute topical cooling methods (less desirable since they might cause shivering).

Subsequent Management

- If the patient is already intubated, consider postoperative intubation and mechanical ventilation
- Load with anti-thyroid medication: propylthiouracil (PTU) or methimazole. (Both are oral preparations.)
- Begin elemental iodine after starting antithyroid medication.
- An endocrinologist should guide subsequent management.

Risk Factors

- Recent iodine therapy (radioiodine or iodine-containing contrast agents)
- Cessation of antithyroid medications
- Infection or serious illness (e.g., myocardial infarction) in a patient with preexisting hyperthyroidism
- Trauma
- Preeclampsia
- Excessive thyroid hormone replacement
- Excessive manipulation of a hypertrophic thyroid gland during surgery

Prevention

Establish an adequate depth of anesthesia in a patient with hyperthyroidism to avoid an exaggerated sympathetic nervous system response.

Special Considerations

- Emergency therapy consists of circulatory support and blockade of the end-organ effects of the excess thyroid hormone.
- Intraoperative hypotension should be treated with a direct-acting vasoconstrictor (e.g., phenylephrine).

Further Reading

Connery LE, Coursin DB. Assessment and therapy of selected endocrine disorders. *Anesthesiol Clin North Am.* 2004 Mar;22(1):93–123.

TURP Syndrome

Definition

Systemic absorption of fluids used for bladder distension during a transurethral resection of a prostate mass. Occurs in approximately 2% of patients.

Presentation

Symptoms of hyponatremia, including:

- CNS manifestations:
 - Confusion
 - Nausea and vomiting
 - Diplopia
- Cardiovascular symptoms:
 - Hypertension
 - Bradycardia
 - Myocardial ischemia and dysrhythmia

Pathophysiology

Nonionic irrigating solution (mannitol, glycine, or sorbitol) enters the circulation through open venous sinuses. Excessive absorption of irrigating solution leads to hyponatremia, circulatory overload, or neuro toxicity (if glycine-containing solution is used).

DIFFERENTIAL DIAGNOSIS

- Congestive heart failure (CHF)

Immediate Management

- Discontinue administration of irrigation fluid.
- Send blood for plasma electrolyte levels. (Na^+)
- Administer furosemide (20 mg IV); adjust dose for preoperative creatinine.
- TURP syndrome is usually time-limited (generally resolves within 6 hours post-op).

Diagnostic Studies

- CBC
- Plasma electrolytes

Subsequent Management

- Careful postoperative monitoring until symptoms abate.
- Consider hypertonic (3%) saline in patients with severe hyponatremia (<120 mEq/L, or those with neurologic symptoms).

Risk Factors

- Prolonged operative time (Approximately 1 liter of fluid absorbed per 40 minutes operating time)
- High irrigating pressure (height of bag above patient)
- Large prostate, extensive resection
- Glycine-containing solution (may cause transient blindness)

Prevention

Consider use of regional anesthesia (permits early detection of CNS changes)

Special Considerations

- Acute reduction in plasma sodium content forces migration of water along its concentration gradient out of the plasma space and into the interstitium as well as the extravascular intracellular space. Acute cell swelling and lysis may occur.
- Careful attention should be paid to replacement of both K^+ and Mg^{2+} if diuretics are administered. Use of glycine-containing solution may cause transient blindness.

Further Reading

Malhotra V. Transurethral resection of the prostate. *Anesthesiol Clin North Am.* 2000 Dec;18(4):883–897.

Chapter 6

Miscellaneous Problems

Keith J. Ruskin

- Acute Transfusion Reaction 128
- Bone Cement Implantation Syndrome (BCIS) 130
- Burns 131
- Drug Extravasation 133
- Intra-arterial Injection 135
- Occupational Exposure 137
- Operating Room Fire 139

Acute Transfusion Reaction

Definition

Intravascular hemolysis of red blood cells caused by recipient antibody and complement. Usually occurs in response to a major (ABO) type mismatch.

Presentation

- Variable symptoms, many of which are masked by anesthesia.
- Hypotension
- Hemoglobinuria
- Bleeding diathesis
- An awake patient may complain of nausea, fever, chills, and chest and flank pain.

Pathophysiology

Intravascular hemolysis of red blood cells caused by recipient antibody and complement. The precise cause of renal failure is controversial, but the prevailing hypothesis is that hemoglobin precipitates in the distal tubules. Disseminated intravascular coagulation (DIC) commonly occurs when RBC products are released and activate the intrinsic system of coagulation.

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Delayed transfusion reaction (transfusions within 2–21 days)
- Febrile transfusion reaction (direct antiglobin test)

Immediate Management

- **Stop the transfusion.**
- Maintain urine output at 75–100 cc/hour with generous IV fluid administration.
- Consider mannitol 12.5–50 g IV.
- Consider furosemide 20–40 mg IV.
- Alkalinize the urine to pH of 8 with sodium bicarbonate. (0.5–1 mEq/kg, then additional doses as necessary to achieve urine pH of 8).
- Maintain blood pressure as needed.

Diagnostic Studies

- Send suspected unit to blood bank with another sample of patient's blood for repeat crossmatch.

- Send a blood sample to blood bank for direct antiglobulin test, and hemoglobinemia.
- Send urine for hemoglobinuria.

Subsequent Management

- Maintain normotension (using fluids and vasoactive drugs as necessary) to ensure adequate renal blood flow.
- Send urine and serum samples for hemoglobin concentration.
- Send blood samples for platelet count, partial thromboplastin time, and fibrinogen level.
- Consider a hematology consult.

Risk Factors

- The majority of transfusion reactions are due to ABO incompatibility. Most errors occur AFTER the blood products have left the blood bank and are committed by physicians and nurses.
- Rushed or incomplete check-in, especially during rapid blood loss or trauma surgery.
- Labeling errors—check the patient's unit number and blood serial number carefully.

Prevention

Use extra caution when administering blood products.

Special Considerations

Delayed hemolytic transfusion reactions may occur between 2 days and 3 weeks after administration of blood products, and usually manifest as a drop in hematocrit. They are difficult to prevent, since they require very low levels of antibody that may be undetectable. Severe hemolytic reactions are fatal in 20%–60% of patients.

Further Reading

Miller RD. Transfusion therapy. In: Miller RD (ed.). *Miller's Anesthesia*. 6th ed. Philadelphia: Elsevier Churchill Livingstone; 2005:1815–1817.

Bone Cement Implantation Syndrome (BCIS)

Definition

Hypoxia, hypotension, cardiac dysrhythmias, or increased pulmonary vascular resistance following methyl methacrylate cement implantation.

Presentation

- Decreasing end-tidal carbon dioxide (ETCO₂) in ventilated patients
- Dyspnea and/or altered mental status in patients under regional anesthesia
- Cardiac dysrhythmias or cardiac arrest may occur.

Pathophysiology

The pathophysiology of BCIS is unknown. Proposed mechanisms include embolic showers causing either a direct mechanical effect or release of vasoactive or pro-inflammatory mediators. Complement activation has also been proposed as a mechanism.

DIFFERENTIAL DIAGNOSIS

- Anaphylaxis
- Massive pulmonary thromboembolus (prolonged hypoxemia)
- Myocardial infarction or cardiac failure (ECG changes, prolonged hypotension, pulmonary edema, minimal response to fluids or vasopressors)

Immediate Management

- Increase FiO₂ to 100%.
- Initiate aggressive resuscitation with IV fluids.
- Support blood pressure with ephedrine (5 mg IV) or phenylephrine (100 µg IV) boluses. If refractory, consider phenylephrine or epinephrine infusion.

Diagnostic Studies

- BCIS is a clinical syndrome without specific confirmatory diagnostic studies.
- Transesophageal echocardiography or precordial Doppler ultrasonography may reveal the presence of emboli.

Subsequent Management

- Advise the surgical team of the event and make a decision as to whether to proceed if a second joint replacement is planned.
- BCIS is usually transient and resolves spontaneously

- Prolonged episodes may occur and should be treated as right ventricular failure. In this case, consider placement of a central venous catheter for monitoring and administration of vasoactive drugs.

Risk Factors

- Rare in healthy patients
- More common in elderly or debilitated patients
- May be associated with surgical technique

Prevention

The severity of BCIS may be reduced by generous fluid administration, increased vigilance during and immediately after prosthesis implantation, and consideration of invasive blood pressure monitoring in high-risk patients.

Special Considerations

Intravascular emboli of air, bone marrow, or fat can be seen on transesophageal echocardiography during implantation of orthopedic prostheses. Some of these patients develop transient hypotension or hypoxemia.

Further Reading

Donaldson AJ, Thomson HE, Harper NJ, Kenny NW. Bone cement implantation syndrome. *Br J Anaesth*. 2009 Jan;102(1):12–22.

Burns

Definition

First-degree burns involve epidermis and upper dermis and heal spontaneously. Second-degree burns involve the deep dermis and require excision and grafting. Third-degree burns involve complete destruction of the dermis and must be excised and grafted. Fourth-degree burns involve muscle, fascia, and bone.

Presentation

- Thermal trauma after exposure to flames in an enclosed space
- Thermal trauma after airplane, motor vehicle, or industrial accidents
- Chemical burns after industrial accidents
- Partial-thickness burns are red, blanch when touched, and heal spontaneously. Full-thickness burns do not blanch and are insensate.

- Airway injury from smoke inhalation present with dyspnea and airway obstruction (airway injury may not be immediately apparent)

Pathophysiology

Severe burns cause multiple systemic reactions, including release of interleukins and tumor necrosis factor, resulting in immunosuppression, sepsis, multiple organ failure, and protein catabolism. Hypoxemia may result from lung injury, atelectasis, and airway edema. Extensive fluid loss from the injury and massive fluid shifts may cause hypovolemic shock.

Immediate Management

- Immediately administer 100% O₂ by facemask in patients with a patent airway.
- Secure the airway with an endotracheal tube. Awake fiberoptic intubation with topical anesthesia is preferred in patients with severe facial or airway injury, but other techniques may be considered.
- After intubation, maintain a high FiO₂ due to the risk of CO toxicity.
- Begin aggressive fluid resuscitation in patients with burns greater than 15% total body surface area (TBSA). Crystalloid resuscitation is preferred in the first 24 hours following burn injury. Estimate requirements according to the Parkland formula: Fluid Requirements = TBSA burned (%) × Wt (kg) × 4 mL
- Administer 1/2 of total requirement in first 8 hours; give second half over next 16 hours.
- Fluid management is guided by urine output, CVP, or pulmonary artery pressures.
- If cyanide toxicity is suspected, administer sodium thiosulfate, sodium nitrate 3% solution, and hydroxycobalamin.
- If a chemical burn is suspected, use caution to prevent contamination of unit or staff.

Diagnostic Studies

- Surface area can be estimated by the Rule of Nines: Head 9%; each upper extremity 9%; each lower extremity 18%; torso front and back 18% each.
- Blood electrolytes
- Arterial blood gas, including co-oximetry to determine carboxyhemoglobin level
- Lactic acid level (lactic acidosis may indicate cyanide poisoning from burning plastics).

Subsequent Management

- Maintain normothermia. Use warming blankets, forced-air warmers, fluid warmers as necessary. Keep the room temperature as high as possible.
- Use topical antibiotics to prevent infection.
- Consider hyperbaric oxygen therapy if the patient is stable, a pressure chamber is available, and severe CO poisoning is suspected.

Risk factors

Fires in the operating room due to electrocautery or lasers. “Fire resistant” plastic drapes will burn in the presence of O₂ and release toxic smoke.

Prevention

See “Operating Room Fires” On page 139.

Special Considerations

- Full-thickness burns appear white, waxy, or leatherlike and may be confused with unburnt skin. Full thickness burns do not bleed.
- Succinylcholine is generally safe to use within the first few hours after a burn, but after that must be avoided for 12 months after the burn injury.
- Resistance to nondepolarizing neuromuscular blocking agents may occur for up to 10 weeks postinjury.

Further Reading

Hettiaratchy S, Papini R. Initial management of a major burn: I—overview. *BMJ*. 2004 Jun 26;328(7455):1555–1557.

Hettiaratchy S, Papini R. Initial management of a major burn: II—assessment and resuscitation. *BMJ* 2004 Jul 10;329(7457):101–103.

Drug Extravasation

Definition

Unintentional injection of drugs or fluids into the subcutaneous tissue or perivascular space.

Presentation

- Pain on injection or during infusion of fluids.
- Discomfort, swelling or hyperemia at the site of the catheter.

- Paresthesias or local induration of the skin (late signs).
- Severe cases: Compartment syndrome; muscle, tendon, or nerve injury.

Pathophysiology

Tissue injury occurs for a variety of reasons, including hydrostatic pressure, fluid osmolality or cytotoxicity, vasoconstriction.

DIFFERENTIAL DIAGNOSIS

Intra-arterial injection (severe burning pain, discoloration, or absence of pulse distal to injection site)

Immediate Management

- There is no definitive treatment for extravasation injury.
- Stop drug injection or fluid administration if the patient complains of severe pain or signs of extravasation are noted.
- Consider vascular or plastic surgery consultation.
- Specific treatment depends on the extravasated substance.
- In the case of vesicants (e.g., adriamycin), stab incisions and flushing with 500 mL of normal saline has been recommended.
- Extravasation of vasopressors has been treated with phentolamine infiltration.

Diagnostic Studies

- Clinical diagnosis—no diagnostic studies are necessary.
- Subsequent management
- Document the injury in the patient's chart.
- Observe the site carefully for at least several days.

Risk Factors

- Location of catheter
- High infusion pressure
- Multiple punctures of the same vein
- Access sites in close proximity to tendons, nerves, or arteries

Prevention

Avoid placing IV catheters over joints (e.g., antecubital fossa). Whenever possible, place IVs where they can be visually inspected throughout the surgical procedure. Avoid using "positional" IVs. If there is any doubt, administer a small test dose of drug first.

Special Considerations

Although elevating the extremity and applying warm or cold compresses are commonly recommended, there is little evidence to support these practices. Extravasation can occur even when a central venous catheter is used because the proximal port may exit the vessel if the catheter is withdrawn even a few centimeters. Vessicants should therefore be given through the distal lumen.

Further Reading

Schummer W, Schummer C, Bayer O, Müller A, Bredle D, Karzai W. Extravasation injury in the perioperative setting. *Anesth Analg*. 2005 Mar;100(3):722–727.

Intra-arterial Injection

Definition

Unintentional injection of drugs or fluids into an artery, usually by injection into an indwelling intra-arterial catheter, or after accidentally inserting an intravenous catheter into an artery.

Presentation

- Pain on injection or during infusion of fluids, possibly in the distribution of the vessel.
- Anesthesia or muscle weakness may occur distal to the injection site in an awake patient.
- Skin pallor or cyanosis distal to the injection site.

Pathophysiology

Pallor, ischemia, and pain may occur due to vasospasm, chemical arteritis, or drug-induced tissue injury.

DIFFERENTIAL DIAGNOSIS

- Drug extravasation (discomfort, swelling or hyperemia at the site of the catheter; late signs include paresthesias or local induration of the skin)
- Some drugs (e.g., propofol) cause pain on injection. This will not be associated with distal blanching or mottling of the skin.

Immediate Management

- Stop drug injection or fluid administration if the patient complains of pain, or distal pallor is noted.

Immediate Management (continued)

- Leave the catheter in place for subsequent diagnostic studies and treatment. Begin a slow infusion of isotonic saline solution if the catheter can be flushed and there is no clot.
- Consider anticoagulation with intravenous heparin infusion.
- Consider intraarterial lidocaine 2 mg/kg and papaverine (30 mg).

Diagnostic Studies

- Clinical diagnosis
- Transduce the catheter. High pressures or an arterial waveform implies that the catheter is intra-arterial. (If the patient has an arteriovenous fistula, this test is nondiagnostic.)
- Subsequent management
- Document the injury in the patient's chart.
- Extremity should be elevated to decrease edema and reduce the risk of compartment syndrome.
- Consider sympathectomy with a stellate ganglion block or continuous brachial plexus block (if catheter is located in upper extremity).
- Consider intra-arterial thrombolytic injection.
- Observe the site carefully for at least several days.

Risk Factors

- Morbid obesity
- Darkly pigmented skin
- Thoracic outlet syndrome (pulse decreases with internal rotation of the arm)
- Pre-existing vascular anomaly

Prevention

- Always verify that drugs are being injected into the correct tubing in a patient with an arterial catheter.
- Never assume that an indwelling catheter is in the correct location.
- When inserting an intravenous catheter, the tourniquet should not be so tight as to occlude arterial blood flow.
- Before injecting a drug, carefully observe IV tubing for backflow of blood.

Special Considerations

Although many treatment strategies have been recommended, none have been definitively proven to work. All recommendations are based on individual case reports or small series. Vigilance, immediate recognition of the problem, discontinuation of the irritant, and rapid initiation of treatment offer the best chance of a good outcome.

Further Reading

Sen S, Chini EN, Brown MJ. Complications after unintentional intra-arterial injection of drugs: risks, outcomes, and management strategies. *Mayo Clin Proc.* 2005 Jun;80(6):783–795.

Occupational Exposure

Definition

Inoculation of a healthcare worker (HCW) with infectious blood or body fluids by puncturing the skin with a contaminated object (e.g., needle) or splashing fluids into exposed mucosa.

Presentation

- Injury with a needle or other sharp object.
- Blood or body fluids splashed into the eyes, mouth, or an open wound.

Pathophysiology

Exposure to blood-borne pathogens results in infection of a health care worker.

Immediate Management

- Wash the wound liberally with soap and water. Use of antiseptics is not contraindicated, but there is no evidence that use will reduce risk of infection.
- If exposure was through exposed mucous membranes, they should be irrigated copiously with normal saline.
- All healthcare institutions have an occupational exposure protocol; this protocol should be followed.
- Federal (US) and state reporting requirements must also be followed.
- Post-exposure prophylaxis depends on the type of suspected pathogen.

Diagnostic Studies

- The patient should be tested for the presence of blood borne pathogens hepatitis B surface antigen (HBsAg), hepatitis C virus antibody (anti-HCV), and HIV antibody) if his or her status is unknown. Testing usually requires patient consent.
- If exposure to HCV is suspected, the healthcare worker should undergo baseline testing for anti-HCV and alanine aminotransferase with follow-up in 4–6 months.
- If exposure to HIV is suspected, the healthcare worker should undergo baseline testing for HIV antibody and further testing at 6–12 weeks and 6 months.
- Subsequent management
- If exposure was to hepatitis B and the HCW is susceptible, hepatitis B immune globulin should be administered within 24 hours, and the hepatitis B vaccine should be offered to confer active immunity.
- There is no prophylaxis for exposure to hepatitis C.
- If the source patient is HIV positive, the HCW should be placed on a 2- or 3-drug regimen depending on the risk of HIV transmission. Prophylaxis should begin within 24 hours of exposure and be continued for 4 weeks if tolerated.

Risk Factors

- Use of large, hollow bore needles without engineered protection
- Use of straight suture needles for securing vascular access devices
- Two-handed recapping of needles (13% of exposures in one study)
- Long work hours and fatigue have been reported to increase the risk of needle stick injuries in medical personnel.

Prevention

- Always use standard precautions.
- Do not recap needles if possible. If recapping is necessary, a one-handed technique should be used.
- Needleless or protected needle devices should be used when available.
- Sharp objects such as needles or scalpels with engineered safety systems should be used when practical.
- Needles should not be held while tying sutures.

- Staples should be used instead of sutures with straight needles to secure vascular access devices.
- Barrier precautions (gloves, masks, eye shields) should be used whenever risk of exposure is present.

Special Considerations

A copy of the healthcare institution's occupational exposure protocol is maintained in the policy and procedures manual. A copy of this document should be available at all clinical locations. There is no need to modify the patient care responsibilities of an individual who has been exposed to hepatitis B or C or HIV, although the exposed individual should be counseled regarding infection control. Exposed health care workers should not donate blood, plasma, or tissue during the post exposure follow-up period. A health care worker who develops hepatitis B or C should modify his or her patient care responsibilities as recommended by the CDC.

Further Reading

Berry AJ. Needle stick and other safety issues. *Anesthesiol Clin North Am*. 2004 Sep;22(3):493–508, vii.

Fisman DN, Harris AD, Rubin M, Sorock GS, Mittleman MA. Fatigue increases the risk of injury from sharp devices in medical trainees: results from a case-crossover study. *Infect Control Hosp Epidemiol*. 2007 Jan;28(1):10–17.

Operating Room Fire

Definition

An infrequent but catastrophic event that can involve the airway, head and neck, or any other portion of the surgical field.

Presentation

- A puff of smoke and possibly a flash of light in an endotracheal tube
- Appearance of smoke on the surgical field or from under the drapes
- A “popping” sound may be heard.
- Flames may not be visible, especially if alcohol (from prep solutions) is the fuel.

Pathophysiology

Three elements must be present: fuel (e.g., alcohol or plastic), oxidizer (O_2 or N_2O), and an ignition source (electrocautery or laser). Operating room fires can produce significant amounts of toxic smoke, but not enough heat to activate overhead sprinkler systems. Burning plastic (e.g., surgical drapes) can produce CO, hydrogen chloride (HCl), and cyanide.

Immediate Management

- Specific actions depend upon the location of the fire and source of fuel and oxidizer.
- **RACE: Rescue, Alert, Contain, Extinguish.**
- Rescue patients or staff in immediate danger. Activate the fire alarm.
- Contain the fire by removing the oxidizer from the fuel. (Disconnect anesthesia circuit from patient for airway fire, discontinue O₂.)
- Extinguish flames if it is safe to do so.
- Evacuate the room if necessary.
- **PASS: Pull** the pin to activate the extinguisher, **Aim** at the base of the fire, **Squeeze** the trigger, and **Sweep** the extinguisher back and forth across the fire.

DIFFERENTIAL DIAGNOSIS

There is no true differential diagnosis. It is important to determine the ignition source, fuel, oxidizer, and location of the fire immediately.

Subsequent Management

- Subsequent management depends on the location of the fire.
- Determine whether evacuation of the operating room is necessary.
- Abort the surgical procedure as soon as it is safe to do so.

Risk Factors

- Use of laser surgical devices
- Use of electrocautery after application of alcohol-based prep solutions
- Use of O₂ enriched gas adjacent to electrocautery or laser
- Bringing hot items (i.e., halogen lamps or camera light sources) into contact with other flammable items

Prevention

- Minimize FiO₂ whenever surgery will take place near the airway.
- Be certain that flammable prep solutions have been removed from the patient before using electrocautery.
- A response plan should be formulated before the patient is brought into the operating room. Know the location of fire extinguishers and fire blankets.

Special Considerations

There are three types of fire extinguishers: A (paper, cloth, plastics), B (liquids or grease), and C (electrical). Be sure to use the right extinguisher for the fire in progress. Most extinguishers in the OR are type ABC (can be used on all fires).

Further Reading

Ehernwerth J, Siefert HA. Electrical and fire safety. In: Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2006:169–173.

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Chapter 7

Neurosurgical and Neurologic Emergencies

Ira J. Rampil

- Autonomic Hyperreflexia 144
- Closed Head Injury 145
- Dystonic Reactions 148
- Intracranial Hypertension 150
- Penetrating Head Injury 152
- Spinal Cord Injury 155
- Subarachnoid Hemorrhage (SAH) 157
- Venous Air Embolism (VAE) 161

Autonomic Hyperreflexia

Definition

A medical emergency that is caused by massive autonomous discharge of sympathetic spinal neurons below the level of chronic spinal cord injury.

Presentation

- Autonomic hyperreflexia can occur during general anesthesia in patients with an old spinal cord injury. It is unlikely during subarachnoid block.
- Sudden significant increase in systolic blood pressure with associated baroreceptor-reflex bradycardia
- Diaphoresis, erythema and piloerection above the dermatome of spinal injury
- Headache in awake patients
- Blurred or spotty vision in awake patients

Pathophysiology

Spinal cord injury releases spinal preganglionic sympathetic motor neurons below the level of the injury from descending control by the rostral CNS. A series of abnormal reflexes develop (e.g., the plantar reflex) as new synaptic circuits form in the independent cord below the lesion. In some cases, a reflex arc develops and any form of sensory stimulus may cause a massive sympathetic discharge. Most commonly, this reflex is triggered by stretch of a hollow organ like the bowel or bladder. Severe hyperreflexia is common (50–90%) when the spinal injury is above the T6 level, but hyperreflexia may occur when the injury is as low as T10.

DIFFERENTIAL DIAGNOSIS

- Pheochromocytoma
- Essential hypertension
- Light anesthesia

Immediate Management

- Insert a Foley catheter to empty the bladder.
- If the patient is anesthetized, increase concentration of potent volatile agent.
- If anesthesia is planned for a patient with a history of hyperreflexia, consider neuraxial local anesthesia.
- If the patient is awake, consider nifedipine 10 mg, bite and swallow; if not awake, consider nitroprusside or nitroglycerine infusion.

Subsequent Management

If long-term therapy is required, consider oral therapy with diazoxide, mecamylamine, or phenoxybenzamine.

Risk Factors

- Spinal cord injury above T6
- Male gender (4:1)

Prevention

If surgery involves an area enervated below the cord injury level, consider a subarachnoid block with a local anesthetic of sufficient concentration to provide motor blockade.

Further Reading

Paralyzed Veterans of America/Consortium for Spinal Cord Medicine. Acute management of autonomic dysreflexia: individuals with spinal cord injury presenting to health-care facilities. Washington DC: Paralyzed Veterans of America (PVA); 2001 Jul. 29 p. [138 references]. Available from <http://guidelines.gov>.

Karlsson AK. Autonomic dysfunction in spinal cord injury: clinical presentation of symptoms and signs. *Prog Brain Res.* 2006;152:1–8.

Closed Head Injury

Definition

Abrupt concussive or shearing injury to brain tissue. Secondary injuries occur as a result of vascular interruption, cardiovascular instability, respiratory depression, and increased intracranial pressure (ICP) leading to ischemia. Biochemical derangements such as excitotoxicity lead to delayed neuronal necrosis and secondary arterial vasospasm.

Presentation

- History of impact or sharp acceleration of head
- Brief loss of consciousness or “seeing stars”
- Lethargy
- Possible seizure
- Possible autonomic signs including bradycardia, pallor, hypotension
- May have sluggish papillary light reflex

Pathophysiology

Injury is proportional to force of impact. A small impact may not lead to detectible anatomic injury, but may cause transient neuronal dysfunction. More forceful injuries may cause a cerebral surface

contusion with local destruction of neurons, petechial hemorrhage, ischemia and edema. Injured areas will often be in a coup-contrecoup configuration (skull-brain interface on the opposite site of impact). Increasing the force of impact will lead to deeper injuries as the relatively free-floating cerebrum moves in the skull while the falx and tentorial dura and brainstem are fixed in position. These motions shear the neuropil and white matter tracts along with their vascular supply.

DIFFERENTIAL DIAGNOSIS

- Chronic subdural hematoma

Immediate Management

- Definitive airway management: Oral endotracheal intubation with axial cervical spine stabilization. Nasal intubation is contraindicated because of risk of basilar skull fracture. Consider rapid-sequence induction if the clinical situation permits.
- Increase FiO_2 to maintain adequate oxygenation.
- Initiate mechanical ventilation to maintain PaCO_2 between 30–35 mmHg.
- Administer sedation when neurologic assessment is complete.
- Aggressively treat hypotension: fluid resuscitate with normal saline or colloid solutions to restore normal blood pressure. Avoid glucose-containing or hypotonic solutions.
- Patients with severe head injury may require higher than normal BP to maintain cerebral perfusion pressure (CPP). Maintain mean arterial blood pressure (MAP) > 90 mmHg or CPP > 60 mmHg. Use inotropes or vasopressors as needed.
- Obtain blood for type and crossmatch.
- Transfuse platelets if patient has a history of NSAID, clopidogrel or other anti-platelet drug use.
- Administer mannitol 0.5 g/kg as needed to decrease ICP as a bridge to definitive therapy.
- Obtain an emergency CT scan to determine extent of intracranial injury.
- Patients with severe injury require evacuation of hematoma or placement of an intraventricular catheter for monitoring ICP and possible drainage of cerebrospinal fluid (CSF).

- Penetrating head injury (including any skull fracture)
- Subarachnoid hemorrhage from vascular malformation or aneurysm.

Diagnostic Studies

- Plasma electrolytes, complete blood count, toxicology screen
- Serial arterial blood gas analysis

- Serial CT or MRI if symptoms worsen over time

Subsequent Management

- Supportive care
- Patients with severe head injury may have additional injuries. If the patient is neurologically stable, consider workup for occult injuries (e.g., CT scan). Consider occult bleeding if the patient appears to be hypovolemic despite adequate fluid resuscitation.
- Consider seizure prophylaxis: load with phenytoin 15 mg/kg over 1 hour.
- Rehabilitation if neurological deficits persist

Risk Factors

- Youth and male gender. Motor vehicle accidents are a leading cause of brain injury.
- Lack of helmet during motorcycling
- Shaken baby syndrome

Prevention

Patients typically present with this injury; prevention is not feasible in the hospital setting.

Special Considerations

- Even minor head trauma with brief alteration of consciousness may evolve into a life-threatening lesion over hours to a few days. All patients with a history of head injury should be carefully evaluated.
- If surgery is indicated, treat patients with precautions for intracranial hypertension.
- The severity of the injury may progress over the first few post-injury days.

Further Reading

Ropper AH, Gorson KC. Clinical practice: concussion. *N Engl J Med.* 2007;356:166–172.

Table 7.1 Glasgow Coma Scale

Eye Opening	
Spontaneous	4
To sound	3
To pain	2
None	1
Motor Response	
Obeys commands	6

Table 7.1 (continued)

Localizes pain	5
Normal flexion (withdrawal)	4
Abnormal flexion (decorticate)	3
Extension	2
None	1
Verbal Response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1

Reprinted with permission from Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale. *Lancet* 1974; 304(7872): 81–84.

Dystonic Reactions

Definition

Involuntary, sustained, or periodic muscle contractions causing abnormal postures, facial expressions or other movements. The dystonic reaction is a side effect common to several classes of drugs, including neuroleptics, that affect striatal dopaminergic (D_2) transmission.

Presentation

- Sustained muscle contractions, frequently causing twisting and repetitive movements or abnormal postures.
- Onset during or within 3 months of treatment with a triggering agent.
- History of exposure to phenothiazines (thorazine, thioridazine, prolixin), butyrophenones (haldol, droperidol), some antiemetics (prochlorperazine, promethazine, and metaclopramide) and other dopamine receptor antagonists.

Pathophysiology

Etiology is unknown, but some studies suggest that altered sensitivity of dopaminergic receptors (especially D_2 and perhaps D_3) may be responsible.

Immediate Management

- Taper or discontinue the offending drug.
- Request a psychiatric consult to suggest alternative drug therapy (i.e., atypical antipsychotic drugs).

Immediate Management (continued)

- In the case of acute onset (within 7 days) dystonia, consider benztropine, trihexyphenidyl, or diphenhydramine (i.e., anticholinergics).
- For cases with onset between one week and 3 months, consider amantadine and/or a beta-blocker such as propranolol.

DIFFERENTIAL DIAGNOSIS

- Wilson's Disease
- Degenerative CNS disease (e.g., Parkinson's, Huntington's Disease, etc.)
- Perinatal injury
- Head trauma, stroke, brain tumor
- Lipid storage diseases
- Ataxia telangiectasia
- Lesch-Nyhan syndrome

Diagnostic Studies

- Detailed drug history
- Serum ceruloplasmin and slit lamp exam
- CNS imaging
- Serum electrolytes including Mg^{++} , Ca^{++}
- ANA (antinuclear antibody titers), VDRL (syphilis test), HIV titers
- Detailed neurologic exam and psychiatric history

Subsequent Management

- Therapy for chronic dystonia is problematic.
- Botulinum toxin (Botox) may provide symptomatic relief for facial or ocular muscles.

Risk Factors

- Use of older neuroleptic drug therapy (about 4:1) rather than newer atypical neuroleptic drugs
- Younger adults are more likely to have this reaction than older patients
- Risk increases with the duration of treatment with neuroleptic drugs.

Prevention

- Prophylaxis with anticholinergic drugs
- Use of new agents at minimum effective dosing

Special Considerations

Anesthesia providers and postoperative nursing personnel should be alert to the possibility of dystonic drug reactions in the recovery room. Many antiemetic drugs currently used to prevent nausea and vomiting have been reported to trigger dystonia. Many antiemetic drugs currently used to prevent nausea and vomiting have been reported to trigger dystonia.

Tardive dystonia closely resembles acute dystonia, but it occurs only in prolonged treatment with triggering drugs, and its symptoms do not respond to anticholinergic therapy.

Further Reading

van Harten PN, Hoek HW, Kahn RS. Acute dystonia induced by drug treatment. *Brit Med J* 1999;319(7210):623–626

Intracranial Hypertension

Definition

Cerebrospinal fluid pressure that exceeds the normal value (approximately 7–20 mmHg in supine adults).

Presentation

- Headache
- Nausea, vomiting
- Blurred vision, diplopia
- Depressed level of consciousness
- Systemic hypertension, bradycardia, and periodic breathing may be seen in severe cases.
- If an ICP monitor is in place:
 - A waves: abrupt increases in ICP to 50–100 mmHg that last up to 20 minutes
 - B waves: abrupt increases in ICP to 35 mmHg that last for several minutes

Pathophysiology

There are three major components within the nearly sealed and poorly compliant intracranial vault: Brain parenchyma, blood, and CSF. If any component increases in volume, the pressure in the vault will rise and be reflected in the CSF pressure. Intracranial hypertension impairs cerebral blood flow and may cause ischemic injury. Increased volume may also push the brain against available dural or bony edges in the skull, causing herniation.

DIFFERENTIAL DIAGNOSIS

- Headaches of other etiology, e.g., migraine, cluster, etc.

- Drug intoxication
- Idiopathic intracranial hypertension (pseudotumor cerebri)

Immediate Management

- **Optimize positioning:** head up 30° and looking straight ahead to ensure open jugular drainage.
- Mannitol 20% 0.5–1.0 g/kg IV rapidly, or hypertonic saline to osmotically reduce brain water content.
- Consider general anesthesia for airway protection, to allow mild hyperventilation, and to decrease cerebral metabolic rate and cerebral blood flow.
- Control systemic blood pressure to maintain cerebral perfusion pressure between 60 and 70 mmHg. (CPP = MAP – ICP)
- Avoid hyperglycemia.
- Neurosurgical treatment (e.g., decompressive craniectomy or hematoma evacuation) or ventricular drainage

Diagnostic Studies

- CT scan to assess ventricular size, compression of basal cisterns, mass effects, edema, hemorrhage, etc.
- Invasive ICP monitoring
- Lumbar puncture (after CT scan shows NO mass effect)

Subsequent Management

- Control underlying cause.
- Consider mild (35°C) hypothermia.
- Treat fever if present.
- Consider ventriculostomy to drain CSF and allow continuous ICP monitoring.
- If CSF circulation is obstructed, a ventriculoperitoneal shunt may be indicated.

Risk Factors

- Neural tube defects
- Prior subarachnoid hemorrhage (SAH)
- Chronic anticoagulant therapy
- Anoxic brain injury
- Tumors
- Infection (brain abscess)
- Cerebral edema
- Intracranial hemorrhage
- Vasodilation (due to drugs, hypercapnia or hypoxia)
- Hydrocephalus (obstructive or idiopathic)

Special Considerations

Initiate treatment rapidly because the brain has a very low tolerance to ischemia.

Further Reading

Bershad EM, Humphreis III WE, Suarez JI. Intracranial hypertension. *Semin Neurol.* 2008;28(5):690–702.

Penetrating Head Injury

Definition

Any injury that fractures or otherwise violates the cranial vault of the skull.

Presentation

- Depends upon the type of impact and the kinetic energy or velocity involved
- Epidural hematomas (EDH) (Figure 7.1) are frequent after high-energy impacts and usually are accompanied by diffuse parenchymal damage. Patients with EDH may progress rapidly to coma and typically have a poor outcome.
- Acute subdural hematoma (SDH) (Figure 7.2) is also common after high-velocity impacts such as motor vehicle accidents, falls and interpersonal violence. Associated with high mortality (> 50%).
- If the patient is conscious, severe, sudden headache is very common ($\approx 90\%$) as is confusion ($\approx 60\%$), hemiparesis ($\approx 60\%$), and depressed level of consciousness ($\approx 40\%$). Papilledema, abnormal reflexes, nuchal rigidity, and seizures may occur.
- Hematomas initially appear in CT imaging as hyperdense, but over the course of a few weeks will transition to isodense and finally hypodense.
- Chronic SDH is defined as presenting more than 21 days after injury.

Pathophysiology

Two main agents of injury are:

- 1) Contusion and shear from mechanical motion of brain relative to the skull.
- 2) Disruption of vascular supply. A depressed bone fragments and the possibly the object that caused the trauma (e.g., knife or bullet, etc.) may also be present and cause additional injury.

Both of these disrupt brain tissue and cause edema, vascular compromise, ischemia, and necrosis that can propagate and that will be followed by secondary injuries. Epidural hemorrhage is less likely than SDH to be associated with contusion and disruption of brain tissue.

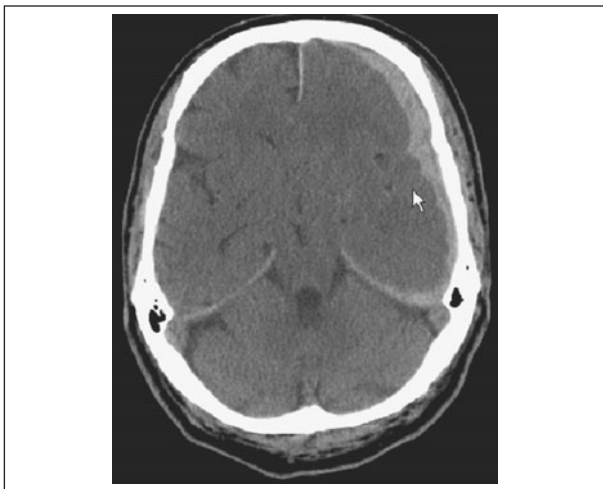


Figure 7.1 A left frontal epidural hematoma causing mass effect. (Image courtesy of Michele Johnson, MD)

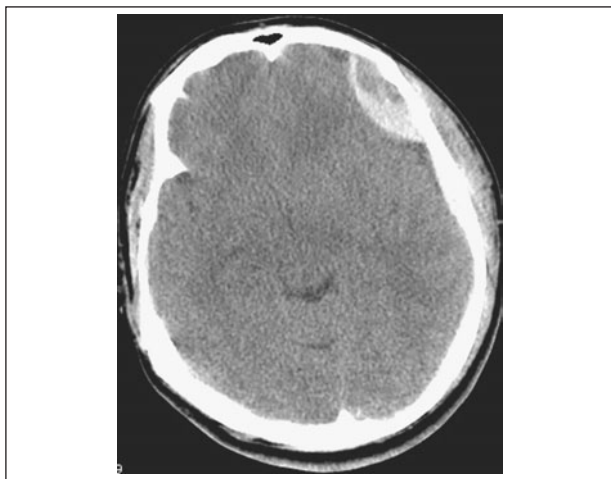


Figure 7.2 A left sided subdural hematoma and mass effect. (Image courtesy of Michele Johnson, MD)

DIFFERENTIAL DIAGNOSIS

- Brain contusion
- Leaking vascular malformation (aneurysm or arteriovenous malformation [AVM])
- Hemorrhage into existing neoplasm or infectious mass lesion of brain

Diagnostic Studies

- Plasma electrolytes, complete blood count, toxicology screen
- Serial arterial blood gas analysis
- Serial CT or MR imaging studies

Immediate Management

- Assess the patient's Glasgow Coma Score before managing the airway.
- Definitive airway management if GCS less than 8, or otherwise indicated: oral endotracheal intubation with axial cervical spine stabilization. Nasal intubation is contraindicated because of risk of basilar skull fracture. Consider rapid-sequence induction if the clinical situation permits.
- Presume cervical spine instability during acute airway management.
- Increase FiO_2 to maintain oxygenation.
- Initiate mechanical ventilation to maintain PaCO_2 between 30 and 35 mmHg.
- Aggressively maintain $\text{CYP} < 60$ mmHg and $\text{SIP} < 90$ mmHg.
- Use hyperventilation ($\text{PaCO}_2 \approx 30$ mmHg) and mannitol (0.5–1 g/kg) or hypertonic saline to control elevated ICP until definitive therapy is initiated.
- Surgical decompression; guideline indications include midline shift > 5 mm, hematoma thickness > 1 cm, decline in $\text{GCS} \geq 2$, fixed pupils, or $\text{ICP} > 20$ mmHg.
- Assess coagulation; delayed injury is highly correlated with presence of coagulopathy.

Subsequent Management

- Maintain cerebral perfusion pressure (CPP) > 60 mmHg to ensure adequate cerebral blood flow for at least 7 days
- Consider ventricular drainage to control intracranial pressure.
- Seizure prophylaxis with anticonvulsants for 7 days (load with phenytoin 15 mg/kg)
- CAUTION: If intravenous phenytoin should be administered over at least 1 hour to minimize risk of bradycardia and hypotension.

- Use of high-dose corticosteroids increases mortality and is not indicated.

Risk Factors

- Preexisting coagulopathy increases risk of delayed neuronal deficits.
- Male gender
- Age over 60 years increases risk of SDH.

Prevention

Avoid risk-taking behavior.

Special Considerations

- Depressed fractures of the temporal-parietal area may lead to laceration of the middle meningeal artery and formation of an epidural hematoma (EDH).
- Epidural hematomas form in the space created by stripping dura from its usual adherence to the skull. These patients may sometimes present with the classic lucid interval before symptoms worsen (Figure 7.1).
- SDH collect between the dura and arachnoid after disruption of the bridging veins. Acute SDH occurs after traumatic head injury and may rapidly compress the brain and its blood supply. Chronic SDH accumulates more slowly in aged patients, ethanol abusers, and patients on anticoagulant therapy (Figure 7.2).
- Epidural hematomas are limited by cranial suture lines and are convex with respect to the brain, whereas SDH are not limited in extent by suture lines and are concave relative to the brain.
- Subarachnoid hemorrhage can be due to traumatic injury of brain parenchyma or to a leaking vascular malformation like an aneurysm or an AVM.

Further Reading

Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury. *J Neurotrauma*. 2007;24(Suppl 1):S1–S106.

Spinal Cord Injury

Definition

Disruption of spinal cord function following traumatic, mechanical disruption of the spinal column, vascular compromise, or compression by an expanding hematoma.

Presentation

- Any combination of sensory and motor loss including sympathetic efferents, depending on the extent of injury
- Cervical injuries above C4 will compromise diaphragmatic ventilation.
- Upper thoracic level injuries will compromise accessory muscles of ventilation and may also lead to bradycardia.
- Lower thoracic and lumbar injuries can cause loss of bowel and bladder control and sympathectomy.
- Hypotension and spinal shock

Pathophysiology

The spinal cord contains white matter tracts that pass ascending and descending neural traffic. In addition, the central grey matter contains areas that perform local sensory signal processing, and central pattern generators that locally coordinate complex motor activity.

DIFFERENTIAL DIAGNOSIS

- Soft tissue injury of neck leading to muscle spasm
- Vascular thrombosis, neoplasia, infection, etc.

Immediate Management

- Maintain spinal alignment to avoid worsening the injury.
- Administer steroids to reduce edema. Some centers use a high-dose methylprednisolone infusion.
- Maintain close monitoring to detect cardiopulmonary compromise.
- If ventilator support is required, intubate the trachea with in-line cervical traction or stabilization, avoiding extension or flexion. Consider use of an indirect visualization device such as a Glidescope® videolaryngoscope or a fiberoptic bronchoscope, especially if a difficult airway is anticipated.
- Maintain mean arterial pressure (MAP) 85–90 mmHg for first 7 days following injury.
- Surgically decompress cord and stabilize spinal column.

Diagnostic Studies

- 3-view cervical spine radiographs (AP, lateral, odontoid views)
- CT scan if suspicious areas are seen on plain radiographs, or if plain radiographs are of poor quality

Subsequent Management

- Deep vein thrombosis (DVT) prophylaxis
- Pressure ulcer prophylaxis

- Neuropathic pain management
- Monitoring for autonomic hyperreflexia
- Control hypothermia

Risk Factors

- Risk-taking behavior, especially in motor vehicles
- Frequent falls
- Violence
- Sports injuries
- Male gender
- Age 16–30 years

Prevention

This is a surgical emergency. There is no specific preventive strategy in the hospital.

Further Reading

Miko I, Gould R, Wolf S, Afifi S. Acute spinal cord injury. *Int Anesthesiol Clin*. 2009 Winter;47(1):37–54.

Stevens RD, Bhardwaj A, Kirsch JR, Mirski MA. Critical care and perioperative management in traumatic spinal cord injury. *J Neurosurg Anesthesiol*. 2003;15:215–229.

Subarachnoid Hemorrhage (SAH)

Definition

Extravasation of blood into the CSF. Nontraumatic SAH is usually secondary to bleeding from an arteriovenous malformation (AVM), or from an arterial aneurysm (Figure 7.3).

Presentation

- About half of SAH patients present with symptoms of a sentinel bleed, which is a small leak of blood from aneurysm. This is often a warning that a catastrophic bleed will shortly follow. SAH symptoms are graded by the Hunt-Hess or the WFNS (World Federation of Neurosurgeons) system. Both scores have prognostic value.
- Acute, severe headache is a classic sign of SAH.
- Nausea, vomiting
- Photophobia
- In some cases, seizures or transient loss of consciousness may occur.
- Hypertension and elevated temperature are common signs.
- Cranial nerve palsy



Figure 7.3 Diffuse subarachnoid hemorrhage, filling the basal cisterns and causing resultant early hydrocephalus. (Image courtesy of Michele Johnson, MD)

Pathophysiology

Arterial bleeding may significantly increase the ICP, which could potentially reach systemic arterial blood pressure. This reduces the CPP significantly (possibly to zero) and causes major ischemic injury. Extravasated blood appears to also have a role in local nitric oxide regulation and in the etiology of vasospasm.

DIFFERENTIAL DIAGNOSIS

Intraparenchymal hemorrhage from nonvascular sources including infectious masses, tumor necrosis and hemorrhage. (Figure 7.4).

Immediate Management

- Obtain large-bore peripheral IV access and insert an intra-arterial catheter for blood gas analysis and continuous blood pressure monitoring.
- Stabilize the blood pressure: Control MAP ψ | 130 mmHg and HR as low as consistent with CPP \geq 60 mmHg.
- Reduce MAP to maintain CPP of 60–70 mmHg with intravenous short-acting beta blockers (e.g., esmolol) and/or calcium channel blockers (e.g., nicardipine). Do not use nitrates. They are cerebral vasodilators and increase ICP. Hydralazine increases ICP less than nitroglycerine or nitroprusside.
- Sympathetic tone may be reduced with midazolam and/or fentanyl. These drugs should only be used in consultation

Immediate Management (continued)

with the neurosurgical team because they will obscure the underlying neurologic clinical examination.

- Consider emergency endotracheal intubation if the patient is obtunded and cannot protect his or her airway, or hyperventilation will be used to manage intracranial hypertension.
- Maintain tight control of blood pressure, heart rate, and ICP while intubating the patient. An abrupt increase in blood pressure might catastrophically rupture the aneurysm. Judicious premedication with midazolam and fentanyl are strongly advised, followed by an appropriate dose of propofol or thiopental and a rapid-acting neuromuscular blocking agent followed by expeditious laryngoscopy. It is prudent to have esmolol drawn up connected to the IV line in order to promptly treat an undesirable sympathetic response.
- Respiratory gas monitoring should be used to assure normocapnic to slightly hypocapnic ventilation.
- Keep patient euvolemic while maintaining adequate CPP.
- Obtain an emergency neurosurgical consult.
- Current choices for definitive therapy include surgical management (i.e., aneurysm clipping or AVM resection) or intravascular therapy (e.g., coiling or embolization).

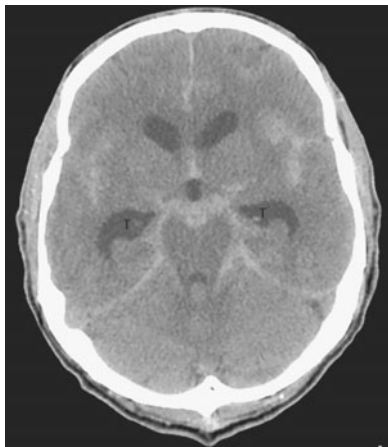


Figure 7.4 A large right temporo-parietal intraparenchymal hematoma with surrounding edema. There is compression of the ipsilateral ventricle and intraventricular hemorrhage. (Image courtesy of Michele Johnson, MD)

Diagnostic Studies

- Plasma electrolytes, complete blood count, coagulation profile, arterial blood gas analysis
- Blood type and crossmatch
- CT scan without contrast, CT angiography
- Direct intravascular angiography
- ECG (will often reveal repolarization abnormalities)

Subsequent Management

- Rebleeding prior to definitive therapy is often fatal.
- Antifibrinolytics may reduce rebleeding, but may increase risk of ischemia.
- Consider prophylaxis against cerebral vasospasm with oral nimodipine.
- If symptomatic vasospasm occurs *after* aneurysmal rupture, consider both intravascular angioplasty or stenting, and classic triple-H therapy (Hypertension, Hypervolemia, and Hemodilution). HHH therapy can only be instituted after definitive aneurysmal therapy. A central venous catheter is usually needed to titrate HHH therapy.
- Patients with SAH have a moderate risk of developing obstructive hydrocephalus.

Risk Factors

Abrupt increase in blood pressure in a patient with a known intracranial aneurysm or arteriovenous malformation. Tobacco and alcohol abuse. Recent data indicates that there may be a genetic component to aneurysm formation.

Special Considerations

- Intraparenchymal hemorrhage is not considered SAH.
- The presence of blood in the CSF detrimentally causes cerebral vasoconstriction and impairs cerebral perfusion.

Further Reading

Molyneux AJ, Kerr RS, Yu LM. International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomized comparison of effects on survival, dependency, seizures, rebleeding, subgroups. *Lancet*. 2005;366:809–817.

Venous Air Embolism (VAE)

Definition

Air enters the venous circulation through an open wound located above the heart and travels to the right side of the heart or pulmonary vasculature. Gas bubbles may enter the systemic circulation in patients with an intracardiac defect; gas bubbles may move to the left ventricle and then become lodged in vital organs.

Presentation

- In an awake patient, the earliest signs include coughing and chest pain.
- Arterial desaturation
- End-tidal CO_2 will decrease and end-tidal nitrogen will increase.
- If systemic embolization occurs, ECG signs of ischemia may occur.
- In awake patients, acute neurologic deficits may be noted.
- The surgeon may note intra-arterial bubbles.
- If a large volume of gas is rapidly entrained, cardiovascular collapse will occur.

Pathophysiology

Gas bubbles move to the pulmonary circulation and cause progressive right heart failure. Gas bubbles can also collect in the right ventricular outflow tract and block cardiac output. Gas emboli can also cause ischemic injury in any organ when the bubbles pass through a right-to-left shunt.

DIFFERENTIAL DIAGNOSIS

- Myocardial infarction
- Thrombotic pulmonary embolism

Immediate Management

- Increase FiO_2 to 100%
- Tell the surgeon to flood the field or cover the wound with soaking wet sponges.
- Put the OR table flat or head-down to place the surgical wound below the level of the right atrium
- If VAE occurs during cranial surgery, apply sufficient bilateral pressure to the neck to obstruct flow in the jugular veins (but not the carotid arteries)

Immediate Management (continued)

- Ask the surgeons to find and occlude the point of gas entrainment using bone wax and/or electrocautery.
- Maintain systemic blood pressure with vasopressors or inotropes as needed.
- Volume resuscitate with isotonic crystalloid or colloid solutions to support systemic blood pressure and increase right atrial pressure.
- If cardiovascular collapse occurs, perform CPR with the patient in head-down and rolled-to-the-left lateral decubitus position.

Diagnostic Studies

- Arterial blood gas
- Hematocrit

Subsequent Management

- Hemodilute to hematocrit of 30. Colloid solutions mitigate cerebral edema.
- If systemic VAE occurred, strongly consider hyperbaric oxygen therapy.

Risk Factors

- VAE classically occurs in sitting craniotomy where the surgical wound is elevated above the level of the right atrium.
- VAE is a risk in many other surgical procedures, including Caesarean section, arthroscopic surgery of the shoulder, and total hip arthroplasty.
- The CO₂ tension pneumoperitoneum that is created during laparoscopic surgery may also cause gas embolization into the venous system. Carbon dioxide bubbles are more soluble and disappear more quickly than nitrogen or oxygen, but if large gas entrainment occurs, ischemic injury and right heart failure can still occur.

Prevention

- Preoperative fluid loading may be useful.
- Use of positive end-expiratory pressure (PEEP) may increase CVP and decrease the incidence of VAE, but at the risk of increased risk of right-to-left shunting of bubbles.

Special Considerations

Entrainment of air occurs when the height of the opened vein above the atrium, in cm, is greater than the CVP (as measured in cm H₂O). In the past, a special central venous catheter designed to aspirate bubbles from the right atrium was strongly advocated. These catheters may serve a diagnostic role if end-tidal gas analysis, precordial Doppler and pulse oximetry are not available, but aspiration of blood with bubbles rarely recovers enough volume to cause a therapeutic effect.

Further Reading

Mirski MA, Lele AV, Fitzsimmons L, Toung TJ. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. 2007;106:164–177.

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Chapter 8

Obstetric Emergencies

Robert Gaiser

- Accidental Dural Puncture 166
- Breech Presentation 168
- Embolism: Thrombus, Amniotic Fluid, Air 170
- Failed Intubation 174
- Fetal Bradycardia 177
- Local Anesthetic Toxicity 179
- Maternal Cardiac Arrest 181
- Maternal Hemorrhage 184
- Neonatal Resuscitation 187
- Preeclampsia/Eclampsia 189
- Shoulder Dystocia 192
- Total/High Spinal Anesthesia 195
- Umbilical Cord Prolapse 197
- Uterine Rupture 199

Accidental Dural Puncture

Definition

Penetration of the dura mater with an epidural needle or catheter. The majority of accidental dural punctures are accompanied by the visible return of cerebrospinal fluid (CSF) through the epidural needle. The dura mater may also be nicked by the epidural needle or punctured by the catheter. There may be no CSF return, but the patient is at risk for complications from the dural puncture.

Presentation

Any one of the following signs indicates accidental dural puncture:

- Free flowing cerebrospinal fluid from the epidural needle;
- Test dose (1.5% lidocaine with epinephrine, 1:200,000) causes intrathecal block;
- Able to aspirate cerebrospinal fluid from the catheter;
- Frontal-occipital headache 24–48 hours after epidural anesthesia.

Pathophysiology

If an epidural needle is advanced beyond the epidural space (distance from the ligamentum flavum to the dura mater in the lumbar spine averages 4–6 mm) or if the catheter is advanced through the dura, an accidental dural puncture occurs. The larger amounts of local anesthetic used for epidural anesthesia can produce high sensory and motor levels if unintended intrathecal injection occurs. Headache tends to occur 24–48 hours after accidental dural puncture and is caused by cerebrospinal fluid leaking into the epidural space. The CSF leakage decreases intracranial pressure, causing traction on pain-sensitive structures including the dura and meninges.

DIFFERENTIAL DIAGNOSIS

Hypotension and high sensory level from a small amount of local anesthetic (subdural injection). A subdural injection results in a patchy, weak block, unlike the dense blockade from intrathecal injection.

Immediate Management

- Identify the presence of an accidental dural puncture by observing CSF dripping from the needle, by aspirating CSF from the epidural catheter, or by observing the presence of a sensory level from a test dose.
- If accidental dural puncture with the needle occurs, there are two courses of action:

Immediate Management (*continued*)

- Resite the epidural needle to place the catheter and slowly titrate the local anesthetic.
- Thread an intrathecal catheter and manage as a continuous spinal catheter.
- If the provider aspirates cerebrospinal fluid from the catheter or if the test dose suggests that the catheter is intrathecal, manage as a continuous spinal catheter.

Diagnostic Studies

- CSF contains glucose, which may be detected with a glucometer.
- Cerebrospinal fluid contains protein which may be detected with a urine dipstick.

Subsequent Management

- Decrease the amount of local anesthetic injected, as there is a hole in the dura.
- Follow the patient for 48 hours postoperatively for the presence of headache.
- Conservative management includes intravenous hydration, caffeine 500 mg IV, bed rest, and analgesia.
- The definitive treatment for a postdural puncture headache is an epidural blood patch. 20 mL of autologous blood is injected into the epidural space. This compresses the thecal sac, forcing cerebrospinal fluid cephalad and immediately relieves the headache. The injection of blood forms a clot at the dural hole, preventing further leakage.

Risk Factors

For accidental dural puncture:

- Previous accidental dural puncture

For postdural puncture headache:

- Female gender
- Young age (10–40 years)
- Vaginal delivery

Prevention

There are no good means to prevent an accidental dural puncture other than meticulous technique. For prevention of postdural puncture headache, intrathecal catheters have been recommended (but never studied) or 3 mg epidural morphine, two doses 24 hours apart.

Special Considerations

Accidental dural puncture may result in postoperative headache. Postdural puncture headaches tend to be positional, with symptoms improving when the patient lies flat. Other symptoms include nausea, vomiting, visual disturbances, and hearing alteration. Visual disturbances (diplopia) are due to the dysfunction of the extraocular muscles from the transient paralysis of the cranial nerves III, IV, and VI.

Further Reading

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Breech Presentation

Definition

Breech presentation occurs when the fetal buttocks descend into the maternal pelvis before the fetal head.

Presentation

Breech presentations are classified according to the presentation of the legs and buttocks. A frank breech presentation is the most common type of breech presentation and occurs when both hips are flexed and both knees are extended. In a complete breech presentation, both the knees and the hips are flexed. In an incomplete breech presentation, one or both hips are not flexed, and one or both feet are lowermost in the birth canal.

Pathophysiology

The major concern with delivery of the breech fetus is fetal head entrapment. The head of the fetus is the largest portion of the fetus and may not deliver through the cervix, especially if the cervix is partially dilated. During the time period from delivery of the fetal abdomen to delivery of the fetal head, there is compression of the umbilical cord, which leads to decreased blood flow and fetal hypoxia.

DIFFERENTIAL DIAGNOSIS

The obstetrician determines the type of breech presentation and decides whether to proceed to cesarean delivery. There are circumstances in which a parturient and her obstetrician will decide to attempt a vaginal breech delivery such as for the delivery of a nonvertex second twin, or if cesarean delivery is refused.

Immediate Management

- Prepare for emergency cesarean delivery in instances of footling breech or a premature breech fetus.
- Both the anesthesiologist and the obstetrician must be prepared to perform rapid cesarean delivery.
- Unless the mother has an epidural catheter in place, general anesthesia is usually administered.

Diagnostic Studies

Ultrasound is used to confirm physical examination.

Subsequent Management

Following delivery, the infant should be resuscitated by individuals skilled in neonatal resuscitation. Breech infants have more neonatal complications than babies that have a vertex presentation, regardless of the route of delivery.

Risk Factors

- Maternal
 - Uterine anomalies
 - Multiparity
 - Pelvic tumors
- Fetal
 - Anencephaly
 - Microcephaly
 - Neuromuscular disorders
 - Polyhydramnios
 - Oligohydramnios
- Placental
 - Placenta Previa

Prevention

Prior to delivery, the obstetrician may attempt an external version. The procedure involves an attempt to maneuver the fetus into a cephalic presentation by applying pressure to the maternal abdomen. The procedure is done usually around 36–37 weeks gestation. Later attempts have a lower success rate because of the increase in fetal size.

Success for this technique may be improved with the use of regional anesthesia.

Special Considerations

Before 28 weeks gestation, as many as 40% of fetuses are in the breech presentation. At term, however, only 3–4% of singleton pregnancies have a breech presentation. Breech delivery increases the risk of infection and perineal laceration for the mother. There are two basic options for management of a breech presentation: somehow converting to a vertex presentation and then allowing labor, or performing a cesarean section.

The Term Breech Trial randomized parturients with breech fetuses at 37 or more weeks gestation to either planned cesarean delivery or planned breech vaginal delivery. There was an increased risk of neonatal morbidity and mortality with attempted vaginal delivery. Cesarean delivery is now the preferred route for breech presentations except for a nonviable fetus, delivery of a second nonvertex twin, or advanced labor with a breech presentation.

Further Reading

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Embolism: Thrombus, Amniotic Fluid, Air

Definition

Embolism is the leading cause of maternal mortality in the United States. Embolism involves entry of solid (thrombus), liquid (amniotic fluid), or gas (air) into the maternal vascular system. Thromboembolism is the most common in parturients.

Presentation

- Common to all:
 - Dyspnea
 - Chest pain
 - Tachypnea
 - Tachycardia
 - Increased central venous pressure
 - Dilated external jugular veins
- Amniotic fluid embolism includes all of the above, and:
 - Bleeding
 - Disseminated intravascular coagulopathy

Pathophysiology

After entry into the venous system, emboli become lodged in the pulmonary vascular bed. This obstruction may produce right ventricular failure. It also creates a ventilation/perfusion mismatch with resulting hypoxemia. An anaphylactoid reaction and coagulopathy with hemorrhage occur during amniotic fluid embolism.

Immediate Management

- Thrombus
 - Increase FiO_2 to maintain oxygenation. Consider endotracheal intubation if respiratory failure is imminent.
 - Hemodynamic support with fluids, inotropes, or vasopressors
 - Anticoagulation with unfractionated heparin
 - For episodes with profound hemodynamic compromise, consider thrombolysis or surgery.
- Amniotic fluid
 - Increase FiO_2 to maintain oxygenation. Consider endotracheal intubation if respiratory failure is imminent.
 - Hemodynamic support with fluids, inotropes, or vasopressors
 - Consider placement of arterial catheter and establishing central venous access.
 - Correct coagulopathy with fresh frozen plasma, cryoprecipitate, and platelets.
 - Uterine atony is common and requires administration of medications to improve uterine tone.
- Air
 - Inform the obstetrician and position the patient with surgical site below the level of the heart.
 - Request irrigation of the surgical field with normal saline to cover open venous sinuses.
 - Increase FiO_2 to maintain oxygenation. (Rarely requires intubation.)
 - Hemodynamic support with fluids, inotropes, or vasopressors.
 - Consider placement of a multi-orifice central venous catheter to aspirate air from the right atrium.
 - Consider hyperbaric oxygen after the event in severe cases if available.

DIFFERENTIAL DIAGNOSIS

- Asthma
- Anaphylaxis
- Sepsis
- Aspiration
- Peripartum cardiomyopathy
- Uterine rupture
- Placental abruption

Diagnostic Studies

Thrombus

- Arterial blood gas analysis, looking for a decrease in oxygen partial pressure and an increase in carbon dioxide partial pressure
- V/Q scan
- Spiral computed tomographic pulmonary angiography
- The majority of thrombi originate in the lower extremities and require Doppler ultrasound or MRI to locate the source

Amniotic fluid

- Diagnosis of exclusion
- Consider sampling blood from central venous catheter looking for epithelial squamous cells, lanugo hair, or mucin in the maternal circulation.

Air

- Arterial blood gas analysis looking for a decrease in oxygen partial pressure and an increase in carbon dioxide pressure
- If general anesthesia, there is an abrupt decrease in end-tidal carbon dioxide.
- Transesophageal echocardiography is both sensitive and specific, but requires general anesthesia and the presence of a person skilled in the interpretation.

Subsequent Management

- The most common cause of embolism in pregnancy is thrombus.
- Parturients with thromboembolism should be anticoagulated.
- If event occurs prior to delivery, the parturient is anticoagulated with low-molecular-weight heparin, as warfarin is a teratogen. The appropriate time periods for withholding this drug prior to regional anesthesia must be considered when providing labor analgesia.
- The hemodynamic and hematologic consequences of amniotic fluid embolism mandate that the patient be managed in the intensive care unit.

- Inhaled nitric oxide has been used for pulmonary hypertension accompanying amniotic fluid embolism.
- With air embolism, stop vascular entrainment of air. Reposition the patient with the surgical site lower than the heart and direct the surgical team to flood the operative site with normal saline irrigation.

Risk Factors

- **Thrombus:** Age > 35 years, obesity (BMI > 30), cesarean delivery, current infection, parity > 3, immobility, thrombophilia
- **Amniotic Fluid:** Advanced maternal age, multiparity, tumultuous labor, trauma, multiple gestation, polyhydramnios, fetal macrosomia, augmentation of labor
- **Air:** Cesarean delivery (usually between delivery of infant to closure of hysterotomy), uterine exteriorization

Prevention

- Thrombus
 - Use of compression stockings during cesarean delivery
 - Mobility
 - Antepartum pharmacologic thromboprophylaxis in patients with several risk factors or a history of thrombus
- Amniotic fluid
 - No proven means of prevention
 - Maintenance of a high index of suspicion
 - Amniotic fluid embolism tends to be a diagnosis of exclusion.
- Air
 - Maintenance of surgical site below the level of the heart

Special Considerations

Pregnancy results in a hypercoagulable state with increased levels of coagulation factors I, VII, VIII, X, and XII. This increase is thought to be the cause of the high incidence of thromboembolism in pregnancy. Air embolism is also common during cesarean delivery, but typically does not result in clinical consequences. Air can be demonstrated by transesophageal echocardiography in 93–100% of cesarean deliveries. Although air embolism is relatively common, the incidence of complications is low. The pathophysiology of amniotic fluid embolism syndrome may be due more to the immunologic reaction than to the embolism itself.

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- Marik PE, Plante LA. Venous thromboembolic disease and pregnancy. *N Engl J Med*. 2008;359:2025–2033.

Failed Intubation

Definition

Inability to intubate the parturient, most commonly due to pharyngeal, laryngeal, or tracheal edema. Due to increased awareness of difficulty with intubation, airway emergencies are occurring more commonly in the postoperative period than during at induction of anesthesia.

Presentation

- Hypoxemia
- Difficulty with mask ventilation
- Difficulty with intubation
- Tachycardia followed by bradycardia
- Hypertension

Pathophysiology

During pregnancy, capillary engorgement of the mucosa throughout the respiratory tract causes swelling of the nasal and oral pharynx, larynx, and trachea. At 38 weeks, the incidence of grade IV airways increases by 34% suggesting an increased potential for difficult intubation. Not only is the parturient more difficult to intubate, but there is less time to accomplish it. The gravid uterus displaces the diaphragm 4–7 cm cephalad, causing a decrease in functional residual capacity, and may result in a profound decrease in oxygen saturation during periods of apnea.

DIFFERENTIAL DIAGNOSIS

Failed intubation may occur during induction of general anesthesia or may be caused by a total spinal anesthetic, oversedation, or other etiologies. Loss of the airway may also occur at the end of a general anesthetic if the patient is extubated prematurely.

Immediate Management

- If intubation is not possible:
 - Call for help.
 - Attempt mask ventilation.

Immediate Management (continued)

- If mask ventilation is possible, consider using cricoid pressure and mask ventilation for the procedure.
- If mask ventilation is not possible, follow the difficult airway algorithm. (Inside front cover)
- Consider using a laryngeal mask airway that allows for passage of a gastric tube.
- Consider inserting another type of supraglottic airway.
- Consider transtracheal oxygenation.
- Consider a surgical airway.

Diagnostic Studies

- Mallampati classification and other features of the airway examination
- Able to visualize
 - Class 1: faucial pillars, soft palate, uvula
 - Class 2: faucial pillars, soft palate
 - Class 3: soft palate only
 - Class 4: hard palate

Examine the airway in every parturient. The Mallampati classification may increase as labor progresses, and this increase correlates with a decrease in upper airway volume. The change did not correlate with the amount of intravenous fluids or the length of labor. The airway (including oropharyngeal structures) must therefore be examined before inducing general anesthesia, even if the airway had been examined previously.

Subsequent Management

Patients with difficult intubation should not be extubated until they have recovered sufficiently from anesthesia and physiologic perturbations have resolved. Parturients who have received fluid or blood resuscitation for hemorrhage may require postoperative intubation until airway edema has decreased.

Risk Factors

- Obesity
- Previous airway surgery
- Diabetes mellitus
- Preeclampsia
- Inability to visualize oropharyngeal structures
- Receding mandible
- Short neck

Prevention

The obstetric care team should be alert to the presence of risk factors that place the parturient at increased risk for complications from general anesthesia. For those patients at risk, consider placement in early labor of an epidural catheter and confirm that the catheter is functional. The American Society of Anesthesiologists has established an algorithm for failed intubation that must be readily available. Labor and Delivery Units should have equipment and personnel readily available to manage airway emergencies. Basic airway management equipment should be immediately available during the initial provision of regional anesthesia, such as:

- Laryngoscope and assorted blades;
- Endotracheal tubes with stylets;
- Oxygen source;
- Suction;
- Self-inflating bag and mask for positive pressure ventilation;
- Medications for blood pressure support, muscle relaxation, and hypnosis.

In addition, portable equipment for difficult airway management should be readily available in the operative area of labor and delivery units.

- Rigid laryngoscope blades and handles of alternate design and shape
- Endotracheal tubes of assorted size
- Laryngeal mask airways of assorted sizes
- At least one device suitable for emergency nonsurgical airway ventilation.
 - Retrograde intubation equipment
 - Hollow jet ventilation stylet
 - Cricothyrotomy kit with or without a transtracheal jet ventilator
- Endotracheal tube guides
- Equipment suitable for emergency surgical airway access
- Topical anesthetics and vasoconstrictors

Special Considerations

- Obstetricians should be encouraged to provide appropriate consultation and early epidural placement in patients with potentially difficult airways. Anesthesiologists must be prepared for management of the unexpected difficult airway. Appropriate training in emergency airway management will improve mortality from failed intubation.

- Pregnancy is associated with a shift in the position of the stomach caused by the gravid uterus, which changes the angle of the GE junction, resulting in an incompetent GE sphincter. Stomach emptying may also be prolonged in pregnancy. All parturients are therefore at increased risk of aspiration and should receive aspiration prophylaxis (sodium bicarbonate solution) before induction. If general anesthesia is planned, or in the case of a failed regional anesthetic, rapid sequence induction should be performed.

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Fetal Bradycardia

Definition

The baseline fetal heart rate (FHR) is determined by approximating the FHR for a 10-minute window, but excluding accelerations or decelerations. Bradycardia is defined as an abnormal baseline heart rate less than 110 bpm. Fetal bradycardia with baseline variability is considered to be indeterminate. Absence of baseline variability is highly predictive of abnormal fetal acid-base status.

Presentation

Clinical diagnosis based on FHR tracings.

Pathophysiology

Fetal bradycardia, when accompanied with decreased baseline variability or prolonged deceleration (abrupt ≥ 15 bpm decrease in the baseline lasting 2–10 minutes) is associated with fetal hypoxia. The etiology may be maternal (hypoxemia, hypotension, aortocaval compression, decreased hemoglobin), uterine (placental abruption), or fetal (umbilical cord occlusion from knot or compression).

DIFFERENTIAL DIAGNOSIS

- Maternal heart rate
- Non-reassuring fetal heart rate
- Maternal administration of beta-blockers

Immediate Management

- Consult the obstetrician regarding plan for delivery and prepare for urgent cesarean section.
- Treat factors that could be contributing to fetal distress:
 - Administer supplemental O₂
 - Treat hypotension with fluid loading and incremental doses of ephedrine 5 mg IV
 - Left uterine displacement to treat aortocaval compression
- If urgent delivery is required, consider aspiration prophylaxis with sodium bicarbonate.
- If a functioning epidural catheter is present, extend the sensory level with 3% 2-chloroprocaine or 2% lidocaine depending upon the urgency (2-chloroprocaine requires 2 minutes to achieve a satisfactory sensory level; lidocaine requires 4–6 minutes to achieve an appropriate sensory level).
- If no epidural catheter is in place, evaluate the maternal airway.
 - If airway exam is suggestive of possible difficult intubation, consider regional anesthesia or fiberoptic intubation to avoid maternal injury.
 - If airway exam does not suggest a possible difficult intubation, consider rapid sequence induction with cricoid pressure.
- If non-urgent delivery is planned, supplemental maternal oxygen is administered and maternal position is altered to prevent aortocaval obstruction.

Diagnostic Study

Fetal heart rate may be confirmed with ultrasonography.

Subsequent Management

If non-urgent delivery is planned, in-utero resuscitation includes maintaining blood pressure, left uterine displacement, and supplemental oxygen should be continued. In the case of urgent delivery, either general or regional anesthesia is administered depending upon the amount of time available and the maternal airway examination.

Risk Factors

- Maternal hemorrhage
- Hypovolemia
- Maternal asthma
- Polyhydramnios
- Placental abruption
- Maternal cardiac disease
- Maternal cocaine use
- Maternal trauma
- Rupture of membranes with the fetal head not engaged
- Prematurity

Prevention

While specific prevention is not possible, maintenance of uterine perfusion and fetal oxygen delivery is critical. Left uterine displacement helps to prevent aortocaval compression. Administer incremental doses of phenylephrine (100 mcg IV) or ephedrine (5 mg IV) to treat the hypotension caused by neuraxial anesthesia. The use of supplemental oxygen is debatable because the impact on fetal oxygenation is minimal.

Special Considerations

Despite the presence of fetal bradycardia, the anesthesiologist may elect to perform a regional anesthetic to avoid failed intubation and maternal mortality. Early placement of epidural analgesia during labor decreases the possibility of an urgent general anesthetic and should be considered in parturients if the fetal heart rate tracing is nonreassuring.

Further Reading

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Local Anesthetic Toxicity

Definition

Unintended systemic effects of local anesthetic administration, usually due to overdos or accidental intravascular injection.

Presentation

CNS toxicity, including seizures, and myocardial depression, dysrhythmias, and cardiovascular collapse.

- Low blood concentration: Tongue numbness, tinnitus, lightheadedness, visual disturbances
- Higher blood concentration: Convulsions, coma

Cardiovascular toxicity—impaired electrical conduction in the myocardium and myocardial depression

- Ventricular tachycardia
- Ventricular fibrillation

Pathophysiology

Local anesthetics reversibly block nerve conduction by blocking sodium channels. These drugs act on the central and peripheral nervous system, myocardial muscle, and the conduction system in the myocardium. Seizures are caused by loss of inhibition in the central nervous system. Cardiovascular toxicity results from blocking the sodium channels in the heart, leading to potentially fatal dysrhythmias.

Immediate Management

- Halt the injection of local anesthetic.
- Hyperventilate the patient.
- Administer a benzodiazepine (e.g., midazolam 2 mg IV) or sodium thiopental for seizures. Prepare to secure the airway.
- If bupivacaine toxicity is suspected administer 20% Intralipid 1.5 mL/kg IV bolus followed by 0.25 mL/kg/min for 30–60 minutes.
- Bolus or infusion of 20% intralipid should be continued if symptoms persist.

DIFFERENTIAL DIAGNOSIS

- Eclampsia
- Underlying seizure disorder
- Toxic/metabolic
- Alcohol/drug withdrawal
- Cardiac arrest
- Thromboembolism
- Myocardial infarction
- Underlying cardiac disease

Diagnostic Studies

If a patient seizes or has ventricular tachycardia during or shortly after injection of local anesthetic, assume local anesthetic toxicity.

Subsequent Management

- Administer intralipid for persistent symptoms.
- Perform neurologic examination.
- If cardiovascular toxicity is present, obtain a series of cardiac troponin levels to rule out myocardial injury.

Risk Factors

Parturients are at risk for intravascular placement of epidural catheters due to dilation of epidural veins.

Prevention

Administer a test dose of 3 mL 1.5% lidocaine with epinephrine 1:200,000 prior to injection of large volume local anesthetics. Fractionate the dose of local anesthetic and ask the patient about tinnitus and check for tachycardia during the injection. Subsequent doses of epidural anesthesia should also be fractionated. Administer in 3–5 mL increments, waiting several minutes between doses. An intravascular injection may be detected prior to giving a large dose.

Special Considerations

Any anesthetizing location in which local anesthetic is administered should have 20% intralipid in sufficient quantities to treat toxicity, with instructions for its use readily available.

Further Reading

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Maternal Cardiac Arrest

Definition

Absence of a palpable maternal pulse requiring cardiopulmonary resuscitation.

Presentation

No palpable pulse. The electrocardiogram will reveal pulseless electrical activity, ventricular fibrillation, or asystole.

Pathophysiology

The most common cause is venous thromboembolism. Other causes include sepsis, amniotic fluid embolism, trauma, and maternal cardiac disease. Bupivacaine toxicity can also cause cardiac arrest. The

anatomic and physiologic changes of pregnancy and labor result in significantly decreased cardiovascular and pulmonary reserves, which may complicate the resuscitation. The fetus must also be considered during the resuscitation.

DIFFERENTIAL DIAGNOSIS

- Hemorrhage
- Total spinal anesthesia
- Local anesthetic toxicity
- Anaphylaxis
- Embolism
- Maternal cardiac disease
- Sepsis

Immediate Management

- The goal of resuscitating the parturient is the return of maternal circulation. Follow the American Heart Association ACLS algorithm. During resuscitation, consider maintaining left uterine displacement to improve venous return. The traditional approach to emergency cardiac care has been the *primary survey* followed by the *secondary survey*.
- *Primary survey*: **A**irway (open the airway); **B**reathing (provide positive-pressure ventilation); **C**irculation (give chest compressions); and **D**efibrillation (shock VF/pulseless VT) are used.
- *Secondary survey*, **A**irway (establish airway control); **B**reathing (assess the adequacy of ventilation); **C**irculation (obtain IV access to administer fluids and medications), and **D**ifferential Diagnosis (identify possible reasons for the arrest) follow the primary survey.
- The energy required for defibrillation probably does not change significantly during pregnancy. The energy settings recommended in the ACLS algorithm apply for pregnant patients.

Diagnostic Studies

An automated external defibrillator will determine whether the cardiac rhythm is treatable with electricity. The etiology of pulseless electrical activity should be determined by obtaining arterial blood gases and transesophageal echocardiography if available.

Subsequent Management

If the maternal circulation has not been restored within 4 minutes, the infant should be delivered by STAT cesarean section if the gestation is greater than 20 weeks.

Risk Factors

Parturients with congenital heart disease are at particular risk for cardiac arrest during labor and delivery. Pregnancy is associated with an increase in coagulation factors, including fibrinogen (Factor I), proconvertin (Factor VII), antihemophilic factor (Factor VIII), Christmas factor (Factor IX), Stuart-Prower factor (Factor X), and Hageman factor (Factor XII). The concentrations of Factors I and VIII increase by more than 100%. Parturients are more likely to be hypercoagulable and at risk for thromboembolism because factor concentrations are increased and prothrombin and partial thromboplastin times are decreased.

Prevention

Appropriate testing of the epidural catheter may reduce the incidence of total spinal anesthesia and local anesthetic toxicity but these complications are not totally preventable. Keep a high level of suspicion in parturients with cardiac disease, hemorrhage, or hypertensive disorders of pregnancy.

Special Considerations

The estimated incidence of cardiac arrest is 1 in 30,000 pregnancies. The most important step in the resuscitation of the parturient is the early delivery of the fetus if the resuscitation is not successful. If the circulation has not returned by 4 minutes, the infant should be delivered by minute 5. Early delivery decreases the risk of neurological injury in the infant and may improve the likelihood of successful maternal resuscitation. In late pregnancy, cardiopulmonary resuscitation is compromised by aortocaval compression and inadequate chest compressions. Delivery allows for improved venous return and more effective chest compressions. Estimated gestational age is an important factor in predicting prognosis for infants after perimortem cesarean deliveries. The threshold for expected fetal viability is considered to be around 24 week gestation. Although emergency delivery performed between 20 and 23 weeks may not result in a viable infant, it may enable successful resuscitation of the mother.

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- Katz V, Balderston K, DeFreest M. Perimortem cesarean delivery: were our assumptions correct? *Am J Obstet Gynecol.* 2005;192:1916–1920.

Maternal Hemorrhage

Definition

The American College of Obstetricians and Gynecologists defines maternal hemorrhage as any loss of blood that results in hemodynamic instability.

Presentation

Parturients with obstetric hemorrhage

- Increased maternal heart rate
- Sinus tachycardia
- Maternal hypotension
- Fetal tachycardia

Pathophysiology

In the nonpregnant state, uterine blood flow is approximately 50 mL/min. At term, uterine blood flow is approximately 500–700 mL/min. Hemorrhage during delivery is characterized as antepartum or postpartum. The two major causes of antepartum hemorrhage are placenta previa and placental abruption. The major causes of postpartum hemorrhage are uterine atony, retained placenta, and placenta accreta. *Placenta previa* is a placenta overlying the cervical os, or is proximate to the internal os of the cervix. *Placental abruption* refers to separation of the placenta after 20 weeks gestation and before the birth of the fetus. *Placenta accreta* defines implantation directly onto the myometrium (accreta), into the myometrium (increta), or through the myometrium (percreta). If placenta is implanted into the myometrium, the uterus cannot contract, resulting in hemorrhage. A soft, poorly contracted uterus is referred to as *uterine atony*.

DIFFERENTIAL DIAGNOSIS

- Antepartum hemorrhage: Placenta previa results in painless vaginal bleeding with no concealed bleeding. Placental abruption is very painful, but the blood loss may be retroplacental and concealed.
- Postpartum hemorrhage: The obstetrician will inform the anesthesia provider of retained placenta. The most common cause of postpartum hemorrhage is uterine atony.

Immediate Management

Management of hemorrhage:

- Type and crossmatch packed red blood cells.
- Obtain large-bore intravenous access
- Initiate fluid resuscitation

Immediate Management (continued)

- Consider activating the massive transfusion protocol

Management of uterine atony:

- Administer oxytocin 20–40 units in 1 liter crystalloid solution IV. May cause hypotension
- Methylergonovine 0.2 mg IM. Preeclampsia is a relative contraindication
- Prostaglandin $F_{2\alpha}$ 0.25 mg IM. Asthma is a contraindication

Management of placenta accreta:

- Obstetrician will do a gravid hysterectomy.
- Prepare for significant hemorrhage.
- Consider general anesthesia. Although a gravid hysterectomy may be performed under regional anesthesia, the provider would have to manage the airway and the fluid resuscitation at the same time.

Diagnostic Studies

- Placenta previa is diagnosed by transvaginal ultrasonography.
- Placental abruption and retained placenta are diagnosed by transabdominal ultrasonography.
- Uterine atony is a diagnosis of exclusion.

Subsequent Management

- If uterine contractility is maintained, no further intervention is required. The administration of oxytocin may be continued to maintain contraction. If retained products are suspected, intravenous nitroglycerin provides uterine relaxation and allows the obstetrician to explore the uterus manually, potentially avoiding dilation and evacuation.
- For continued bleeding that does not cause hemodynamic instability, the obstetrician may request an interventional radiology consultation for arterial embolization.

Risk Factors

- Placenta Previa
 - Previous cesarean section
 - Previous uterine surgery
 - Older maternal age
 - Multiple pregnancy
- Placental Abruption

Risk Factors (continued)

- Preeclampsia
- Hypertension
- History of placental abruption
- Cocaine
- Trauma
- Placenta Accreta
 - Previous cesarean section and placenta previa
- Uterine Atony
 - Multiparity
 - Multiple gestation
 - Infection
 - Magnesium
 - Fetal macrosomia
 - Polyhydramnios

Prevention

There are no specific means to prevent obstetric hemorrhage. Optimal management includes preparation and identification of risk factors. In patients at risk for hemorrhage, large-bore IV access and blood for transfusion should be available. In patients with placenta accreta, consider placement of balloon catheters in the iliac arteries by interventional radiology prior to cesarean delivery. These catheters may be inflated after delivery to decrease blood flow to the uterus, decreasing blood loss.

Special Considerations

The use of a cell saver in obstetrics is controversial because of the theoretical risk of infection or amniotic fluid embolism. Both American College of Obstetricians and Gynecologists and the American Society of Anesthesiologists guidelines state that a cell saver should be considered if available. The use of recombinant Factor VIIa in obstetrics has been described, but may exacerbate a preexisting hypercoagulable state.

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Neonatal Resuscitation

Definition

During the transition from intrauterine to extrauterine life, the neonate is forced to make rapid and profound physiologic changes. Approximately 10% of newborns require some assistance to initiate respiration, while about 1% of newborns need extensive resuscitative measures.

Presentation

- Failure to initiate ventilation after being stimulated
- Heart rate less than 100 bpm
- Total body cyanosis

Pathophysiology

Before birth, the fetus depends upon uterine blood flow for oxygen. After birth, the lungs become the only source of oxygen. During this transition, the fluid in the alveoli is absorbed into the lung tissue and replaced by air. The umbilical arteries and vein are clamped, removing the low-resistance placental circuit and increasing systemic blood pressure. Pulmonary vasodilation decreases pulmonary artery resistance, resulting in increased pulmonary blood flow and decreased flow through the ductus arteriosus. The ductus arteriosus begins to constrict. At the completion of the transition, the baby is breathing air and using the lungs for oxygen. If this sequence is interrupted, the pulmonary arterioles may remain constricted, and the systemic arterial blood does not become oxygenated.

Respirations are the first vital sign to cease when a newborn is deprived of oxygen. After an initial period of rapid attempts to breathe, there is a period of primary apnea during which stimulation, such as drying the infant will cause a resumption of breathing. If oxygen deprivation continues, the baby gasps and then enters a period of secondary apnea. During secondary apnea, stimulation will not restart respiration.

DIFFERENTIAL DIAGNOSIS

Maternal opioid consumption may cause neonatal apnea, but this does not change the need for resuscitation. Naloxone 0.1 mg/kg is given to the infant if the mother has received opioids within the past

4 hours. Other causes include general anesthesia administered to the mother, congenital heard defects or other malformations, meconium aspiration, and neonatal sepsis.

Diagnostic Studies

Management is based upon physical exam, evaluating for respirations, heart rate (should be more than 100 bpm determined by palpating umbilical cord or with a stethoscope) and color (infant should have pink lips and, in light-skinned infants, a pink trunk).

Immediate Management

- Place the infant on a radiant warmer.
- Dry and stimulate the infant for 20 seconds.
- If apneic, initiate positive pressure ventilation at a rate of 40–60 breaths per minute at a pressure no greater than 20 cm H₂O.
- Assess heart rate (listening with a stethoscope is most accurate).
- Initiate chest compressions if heart rate is less than 60 bpm despite 30 seconds of effective positive pressure ventilation (Two thumbs are used to depress the sternum while the hands encircle the torso and the fingers support the spine. With the two finger technique, the tips of the middle finger and the index or ring finger of one hand are used to compress the sternum.)
- The rate of compression is one breath after every 3rd compression, for a total of 30 breaths and 90 compressions per minute.
- If the heart rate is greater than 60 bpm, chest compressions are stopped but ventilation is continued.
- If the heart rate is greater than 100 bpm and the baby begins to breathe spontaneously, positive ventilation is stopped.
- If the heart rate is less than 60 bpm, epinephrine 1:10,000 0.1–0.3 mL/kg is administered intravenously.
- If the infant is pale, replace volume with normal saline or lactated Ringer's solution at 10 mL/kg.

Subsequent Management

If the infant responds to drying or to positive pressure ventilation, no further work-up is necessary. If a baby does not begin breathing immediately after being stimulated, the infant has secondary apnea and requires positive pressure ventilation. If the infant requires chest compression or medications, the infant should be admitted to the neonatal ICU for further monitoring.

Risk Factors

- Maternal diabetes
- Preeclampsia
- Maternal infection
- Maternal drug overdose
- Post-term gestation
- Fetal malformation
- Meconium
- Non-reassuring fetal heart tracing during delivery
- Emergency cesarean section
- Prolonged rupture of membranes
- Instrumental delivery (forceps or vacuum)

Prevention

Be aware of risk factors that place the neonate at risk for requiring resuscitation. The absence of a risk factor does not guarantee that the infant will not require resuscitation.

Special Considerations

At least one person (not the anesthesia provider) whose primary responsibility is the neonate and who is capable of initiating resuscitation should be present at every delivery. Laryngeal mask airways have been shown to be effective for ventilating newborns. Neonates should be ventilated with the lowest FiO_2 required to produce adequate oxygenation.

Further Reading

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Preeclampsia/Eclampsia

Definition

A multisystem disorder unique to pregnancy that can cause edema, increased blood pressure, and proteinuria. The American College of Obstetricians and Gynecologists defines preeclampsia as a sustained systolic blood pressure of at least 140 mmHg or a sustained diastolic blood pressure of 90 mmHg. Eclampsia includes these findings as well

as central nervous system involvement leading to seizures or grand mal convulsions not related to other cerebral conditions.

Presentation

- Preeclampsia
 - Requires both:
 - Systolic blood pressure \geq 140 mmHg or diastolic blood pressure \geq 90 mmHg
 - Proteinuria \geq 300 mg in a 24-hour collection
- Severe Preeclampsia
 - Presence of one of the following
 - Headache
 - Visual changes
 - Epigastric pain
 - Oliguria | 400 mL in a 24-hour period
 - Proteinuria \geq 5000 mg in a 24-hour collection
 - Pulmonary edema
 - Hemolysis, elevated liver enzymes, low platelets (HELLP)
 - Systolic blood pressure \geq 160 mmHg
 - Diastolic blood pressure \geq 110 mmHg
 - Intrauterine growth retardation

Pathophysiology

The etiology of preeclampsia remains unknown, but the disease seems to begin at implantation, well before the clinical manifestations appear and seems to involve the placenta. Preeclampsia is seen in patients with molar pregnancies and also in patients with abdominal pregnancies. Most theories implicate an abnormal response to angiogenic proteins produced by the placenta.

Immediate Management

- Preeclampsia at term, or the presence of severe preeclampsia, is an indication for delivery either by induction of labor or cesarean section.
- Anticonvulsants are administered to women with preeclampsia to prevent eclampsia. Predicting who is at risk for eclampsia is difficult, so prophylactic anticonvulsants are used in all preeclamptic patients. Magnesium sulfate (MgSO_4) remains the best medication for the prevention of seizures intra- and postpartum.
- Check the platelet count before attempting regional anesthesia.
- Consider epidural labor analgesia or regional anesthesia for cesarean delivery.

DIFFERENTIAL DIAGNOSIS

- Local anesthetic toxicity
- Preexisting seizure disorder
- Hypertension
- Acute cocaine toxicity
- Pain
- Preexisting kidney disease
- Toxic/metabolic
- Alcohol/drug withdrawal

Diagnostic Studies

- In a hypertensive parturient, urinalysis for protein is the most important test. In a 24-hour collection, proteinuria up to 300 mg in a 24-hour period is normal. Proteinuria greater than 300 mg in a 24-hour period is abnormal.
- Complete blood count to rule out thrombocytopenia.
- The hemoglobin/hematocrit may be increased with a decrease in intravascular volume, or it may be decreased if there is hemolysis.
- If the patient seizes, a head CT scan may be obtained to rule out anatomic causes of the seizure.

Subsequent Management

- Start MgSO_4 for seizure prophylaxis. In patients with normal renal function, load with 4 gm followed by an infusion of 1–2 gm/h.
- The diagnosis of severe preeclampsia indicates organ involvement and is an indication for delivery.
- Parturients with preeclampsia are at risk for the development of eclampsia in the postpartum period, especially during the first 24 hours. Patients must be monitored and MgSO_4 continued for 24 hours postpartum.
- In parturients with epidural catheters who develop thrombocytopenia, consider delayed removal of the catheter until the platelet count begins to normalize.
- Platelet counts tend to normalize within 60 hours postpartum.

Risk Factors

- Maternal age (< 20 years or > 40 years)
- Multiple births
- Hypertension before pregnancy
- First pregnancy
- Previous pregnancy with preeclampsia
- Diabetes mellitus
- Asthma

Risk Factors (continued)

- Kidney disease
- Systemic lupus erythematosus
- Scleroderma

Prevention

There is no known intervention that prevents preeclampsia. With the discovery of the angiogenic/antiangiogenic proteins, it may be possible to predict those who will develop preeclampsia before the onset of symptoms. Aspirin does not prevent preeclampsia and is no longer indicated.

Special Considerations

In industrialized countries, preeclampsia complicates 4.5–11.2% of pregnancies and is more likely to occur at both extremes of reproductive age. Of those parturients with preeclampsia, 1% of them develop eclampsia. While once considered absolutely contraindicated, spinal anesthesia has proven to be a reliable technique without a higher incidence of hypotension. Spinal anesthesia does not increase the risk of pulmonary edema. Thrombocytopenia is the most common hematologic abnormality with preeclampsia. Its incidence depends on the severity of the disease and the presence or absence of placental abruption. A platelet count should therefore be checked prior to neuraxial analgesia/anesthesia in a parturient with preeclampsia. The lowest acceptable platelet count will depend upon the clinical situation. $MgSO_4$ potentiates neuromuscular blockade. If the patient receives general anesthesia for cesarean delivery, extreme care must be used in the dosing of non-depolarizing neuromuscular blockers.

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Shoulder Dystocia**Definition**

Gentle downward traction on the fetal head by the obstetrician fails to result in delivery of the shoulders. The most common etiology

is impaction of the anterior shoulder above the pubic bone. Less commonly, the posterior shoulder becomes impacted on the sacral promontory.

Presentation

- Prolonged second stage of labor.
- Following delivery of the fetal head, gentle traction on the head fails to result in delivery of the shoulder.
- The obstetrician must avoid excessive force to prevent brachial plexus injury.

Pathophysiology

During a normal delivery, the fetal head and shoulders rotate to allow for its descent and passage through the maternal pelvis. If the shoulder should rotate into the anterior-posterior diameter before entering the pelvis, the shoulders may become impacted on the pelvic bone. The obstetrician must be prepared to perform several maneuvers to relieve this impaction or must return the fetal head into the pelvis and perform an urgent cesarean delivery.

DIFFERENTIAL DIAGNOSIS

- Poor maternal pushing effort
- Absence of uterine contraction

Immediate Management

- Delivery must be rapid in order to prevent hypoxic brain injury.
- If attempts to deliver the neonate fail, the obstetrician may do the Zavenelli Maneuver (returning the fetal head into the maternal pelvis and performing cesarean delivery)
- Provide analgesia for delivery.
- Prepare for emergency cesarean delivery.

Diagnostic Studies

Clinical diagnosis is made by the obstetrician.

Subsequent Management

Following delivery, the infant should be resuscitated by a neonatal resuscitation team. The mother should be informed of the event. The record should include the times of delivery of the head and shoulders, the arrival times of team members, and maneuvers used for the delivery.

Risk Factors

- Previous shoulder dystocia
- Fetal macrosomia
- Maternal obesity
- Maternal diabetes
- Prolonged second stage
- Precipitous labor and delivery
- Vacuum or forceps assisted delivery

Prevention

Fetal position should be assessed during labor and delivery. Consider elective cesarean delivery in patients at risk for shoulder dystocia. Training, especially simulation-based training, has been demonstrated to improve the management of shoulder dystocia.

Special Considerations

Shoulder dystocia requires an immediate and coordinated response from the entire team. The anesthesia provider must be prepared to provide analgesia if an epidural catheter is present and to provide anesthesia if urgent cesarean delivery is required. Shoulder dystocia complicates an estimated 0.6–1.4% of deliveries.

- Potential maternal complications:
 - 4th degree laceration
 - Pubic symphyseal separation
 - Femoral neuropathy
 - Hemorrhage
- Potential neonatal complications:
 - Fractured clavicle
 - Brachial plexus injury
 - Birth asphyxia with neurologic impairment

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Total/High Spinal Anesthesia

Definition

Extensive spread of local anesthetic within the intrathecal space caused by an excessive volume of local anesthesia or high rostral spread.

Presentation

- The patient may be conscious but have difficulty speaking, or the patient may be unconscious.
- The patient is unable to move upper extremities.
- Bradycardia from blockade of cardiac accelerator fibers (level higher than T1)
- Hypotension
- Respiratory arrest from motor blockade of muscles of respiration
- Patients with a total spinal anesthetic may have fixed and dilated pupils.

Pathophysiology

Total or high spinal anesthesia may result from the injection of large amounts of local anesthetic into the intrathecal space, such as may occur when an epidural catheter is located intrathecally and is not recognized by the provider. A total or high spinal may occur when a hyperbaric solution is used and the patient is placed in Trendelenburg position, or when a hypobaric solution is used and the patient is in a head-up position. Symptoms of total/high spinal depend upon the extent of blockade. Levels higher than T1 cause bradycardia due to blockade of the cardiac accelerator fibers. If the blockade extends to the cranial nerves, the patient's pupils may be fixed and dilated.

DIFFERENTIAL DIAGNOSIS

- Myocardial infarction
- Local anesthetic toxicity
- Hemorrhage
- Intracerebral bleed

Immediate Management

- Recognize that the patient has a total/high spinal anesthetic.
- Increase FiO₂ to maintain oxygenation.

Immediate Management (continued)

- Prepare for urgent airway management in the setting of respiratory insufficiency. Administer vasopressors (ephedrine or epinephrine) to treat hypotension; bradycardia may occur if the level of the block extends beyond T1, making phenylephrine relatively contraindicated.
- Administer additional intravenous fluid.
- Recognize that the patient has a total/high spinal anesthetic.
- Increase F_{iO_2} to maintain oxygenation.
- Use caution when administering an induction agent in a hypotensive patient.

Diagnostic Studies

- No diagnostic studies are indicated for the diagnosis of total/high spinal.
- If the patient does not respond within the expected time frame following the intrathecal injection, a computed tomography scan is indicated.

Subsequent Management

- Hypotension resulting from sympathetic blockade may require continuous administration of a vasopressor.
- Small amounts of volatile anesthetic agents should be used to provide amnesia during surgery.
- If fetal bradycardia occurs, urgent cesarean delivery should be performed.
- Reverse Trendelenburg position is not recommended to prevent further rostral spread, as it will only worsen venous pooling and further decrease venous return to the heart.
- Some practitioners choose to manage the airway with assisted mask ventilation until the patient is able to protect the airway, but this may increase the risk of aspiration and make it difficult to perform other tasks if this approach is chosen.

Risk Factors

- Short stature
- Maternal obesity
- Spinal anesthesia following failed epidural anesthesia

Prevention

- Maintain vigilance while performing neuraxial blocks.
- Reduce the dose of intrathecal local anesthesia in short or obese parturients

- Reduce the dose of local anesthesia in patients with failed epidural anesthesia
- Always administer a test dose of local anesthetic through an epidural catheter.
- Fractionate the dose of epidural local anesthetic and always solicit the patient for symptoms of intrathecal injection

Special Considerations

Cardiac arrest may occur in patients with high/total spinal anesthesia. The most common sign preceding the arrest is bradycardia.

Further Reading

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Umbilical Cord Prolapse

Definition

The umbilical cord descends in advance of the fetal presenting part during labor, protruding into or through the cervix in the vast majority of cases. It may also become compressed between the fetus and uterine wall.

Presentation

Umbilical cord prolapse typically presents with persistent fetal bradycardia or severe variable decelerations in the setting of ruptured membranes.

Pathophysiology

Two mechanisms have been postulated:

1. The umbilical cord becomes limp after repeated compression and can more easily prolapse.
2. Fetal acidosis increases the stiffness of the umbilical cord, which then leads to decreased adaptability and predisposes to cord prolapse.

While these two postulated mechanisms are contradictory, both require ruptured membranes and a fetus who is not engaged in the pelvis prior to rupture of the membranes.

DIFFERENTIAL DIAGNOSIS

- Fetal bradycardia
- Placental abruption
- Maternal hypotension
- Oligohydramnios

Immediate Management

- Ask the obstetrician to elevate the presenting part of the fetus, moving it away from the umbilical cord.
- The obstetrician should assess the fetal heart rate by feeling umbilical cord pulsations and determine to the urgency of delivery.
- Prepare for emergency caesarean delivery.

If the parturient does not have an epidural catheter in place, consider general anesthesia using rapid-sequence induction.

Diagnostic Studies

- Suspect umbilical cord prolapse in a parturient with fetal bradycardia and ruptured membranes.
- The diagnosis is confirmed by palpation of umbilical cord during vaginal examination.

Subsequent Management

- Prepare for emergency cesarean delivery.
- The obstetrician must follow the fetal heart rate and assist in the decision-making process.
- Following delivery, the infant should be resuscitated by individuals skilled in neonatal resuscitation, as there is a high possibility that the infant is acidotic and hypoxic.

Risk Factors

- Fetal malpresentation
- Preterm delivery
- Low birth weight
- Contracted pelvis
- Multiparity
- Amnioinfusion
- Polyhydramnios
- Twin gestation
- Amniotomy

Prevention

- A careful vaginal examination should be performed prior to rupture of membranes to insure that the fetus is engaged in the pelvis.

- If there is bradycardia after rupture of the membranes, vaginal examination should be performed to check for the presence of umbilical cord.

Special Considerations

The incidence of umbilical cord prolapse varies between 0.14% and 0.62% and has a perinatal mortality rate of 50%. If umbilical cord prolapse occurs in a hospital and a monitored setting, the incidence of perinatal mortality is low (0–3%). The currently accepted management for umbilical cord prolapse is emergency cesarean delivery. Although spinal anesthesia may be possible, it is technically difficult to position the patient with the obstetrician's hand in the patient's vagina.

Further Reading

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Uterine Rupture

Definition

Uterine rupture is the complete separation of all layers of the uterine wall, resulting in free communication between the uterine and abdominal cavities. Uterine dehiscence is an incomplete disruption of the uterine wall, usually with serosa overlying the defect in the uterine muscle. A dehiscence is also known as a uterine window.

Presentation

Uterine rupture usually occurs in parturients who have a uterine scar, although it may occur in an unscarred (no previous surgery) uterus. Uterine rupture usually presents with:

- Abdominal pain; (If an epidural labor analgesia is in use, parturient with uterine rupture will experience breakthrough pain.)
- Vaginal bleeding;
- Loss of fetal station;
- Abnormal fetal heart rate (usually bradycardia).

Pathophysiology

In women with an unscarred uterus, uterine rupture is due to uterine anomalies or connective tissue disease. In parturients with a previous cesarean section, the risk of uterine rupture with a prior low transverse scar is approximately 1%. With a low vertical scar, the risk is 2%, and with an inverted T-shaped or classic incision, the risk is 4–9%.

Uterine rupture may occur following myomectomy. While it generally occurs during labor with uterine contractions, it can occur before the onset of labor.

DIFFERENTIAL DIAGNOSIS

- Placental abruption
- Placenta previa
- Fetal bradycardia
- Uterine tetany

Diagnostic Studies

Immediate Management

- If uterine rupture is suspected, expeditious cesarean delivery is performed.
- Establish large-bore intravenous access in anticipation of hemorrhage.
- Send a maternal blood specimen for type and crossmatch.
- Consider use of a fluid warmer and rapid infusion system.
- Epidural anesthesia may be used for gravid hysterectomy as long as adequate fluid resuscitation is maintained. General anesthesia is also an option.

Ultrasound may be used, but generally the obstetrician will proceed with urgent cesarean delivery if uterine rupture is suspected.

Subsequent Management

The presence of uterine rupture does not necessitate gravid hysterectomy. Usually the uterus can be repaired, but any subsequent pregnancy must be managed with elective cesarean delivery. If gravid hysterectomy is performed, close attention to adequate fluid and blood resuscitation is mandatory.

Risk Factors

- Prior uterine surgery (risk increases in direct correlation with number of surgeries)
- Maternal obesity
- Type of scar (vertical incision has the highest risk)
- Pregnancy within 2 years of previous cesarean delivery
- Advanced maternal age
- Fetal macrosomia
- Induction of labor in parturient with previous cesarean section

Prevention

- Uterine rupture is a rare event.
- A parturient considering a vaginal delivery after cesarean delivery should not undergo induction of labor with prostaglandins.
- Prolonged use of oxytocin is associated with uterine rupture.
- An anesthesiologist and obstetrician must be present during any attempted vaginal birth after cesarean section in case uterine rupture occurs.

Special Considerations

Despite the low incidence of uterine rupture during attempted vaginal birth after cesarean delivery, the number of women attempting a trial of labor has decreased. American College of Obstetricians and Gynecologists guidelines state that an obstetrician and anesthesia provider must be immediately available when a parturient attempts a vaginal birth after cesarean delivery.

Further Reading

American College of Obstetricians and Gynecologists. ACOG committee opinion no. 342: induction of labor for vaginal birth after cesarean delivery. *Obstet Gyencol.* 2006;108:465–468.

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Chapter 9

Pediatric Emergencies

Jessica L. Wagner

- Anaphylaxis 204
- Asthma, Status Asthmaticus 206
- Burns 209
- Drowning and Near Drowning 212
- Epiglottitis 215
- Inhaled Foreign Body 217
- Major Trauma 219
- Neonatal Resuscitation 223
- Pediatric Basic Life Support 227
- Pediatric Advanced Life Support 230
- Stridor 234

Anaphylaxis

Definition

Acute, potentially life threatening, type I hypersensitivity reaction to a specific antigen with multisystemic manifestations resulting from rapid release of inflammatory mediators. Anaphylaxis is mediated by immunoglobulin E (IgE).

Presentation

- Dyspnea
- Bronchospasm with wheezing
- Hoarseness or stridor caused by laryngeal edema
- Tachypnea with use of accessory respiratory muscles
- Erythematous rash, urticaria, and pruritis
- Facial, lip, and tongue edema
- Nausea, vomiting, and abdominal pain
- Hypotension (**may be the only sign of anaphylaxis in the anesthetized patient**)
- Dizziness or altered mental status
- Cardiac arrest (rare, usually reversible)

Pathophysiology

Anaphylaxis is an immediate hypersensitivity reaction caused by IgE activation of mast cells and basophils, following exposure to an allergen in a previously sensitized person. Exposure triggers the production and release of multiple inflammatory and vasoactive mediators, including histamine, prostaglandins, leukotrienes, cytokines, heparin, tryptase, and platelet-activating factor. Increased vascular permeability causes transudation of fluid into the skin and viscera, causing hypovolemia and shock. Arterial vasodilation produces decreased systemic vascular resistance and tissue hypoperfusion.

Immediate Management

- Identify and discontinue any possible antigen.
- Maintain a patent airway; if airway obstruction occurs, intubate immediately (laryngeal edema may make endotracheal intubation or cricothyroidotomy impossible).
- Support ventilation and oxygenation with FiO_2 100%.
- Epinephrine 1–10 mcg/kg IV, depending on severity of reaction, and repeat as necessary.
- Establish large-bore IV access.

Immediate Management (continued)

- Begin aggressive resuscitation with IV fluids to support intravascular volume (10–20 mL/kg, repeat until systolic blood pressure stabilizes).
- Stop or decrease dose of anesthetic agents to maintain blood pressure.
- Treat bronchospasm with inhaled bronchodilators (e.g., albuterol). If the patient is hemodynamically stable, increasing inhaled anesthetic concentration will also treat bronchospasm.

DIFFERENTIAL DIAGNOSIS

- Cutaneous manifestations of allergic reaction not associated with anaphylaxis
- Acute asthma exacerbation
- Aspiration of a foreign body or gastric contents
- Anaphylactoid reactions (present with similar clinical manifestations but are not immunologically mediated)

Diagnostic Studies

- Anaphylaxis is diagnosed based on clinical manifestations; at least two organ systems must be involved to make the diagnosis.
- If the diagnosis is unclear, a blood sample should be analyzed for mast cell tryptase.

Subsequent Management

- Corticosteroids (dexamethasone 0.2 mg/kg IV bolus, or methylprednisone 2 mg/kg IV bolus)
- Antihistamines (diphenhydramine IV 1 mg/kg)
- Advise the surgical team of the event and make a decision as to whether to proceed; terminate the procedure if there is no response to treatment.
- Continuous infusion of epinephrine (0.01–0.2 mcg/kg/min) may be required for several hours after severe reactions.
- Follow-up evaluation by an allergy specialist

Risk Factors

- Food allergies, particularly to peanuts, eggs, and dairy, are the most common cause of anaphylactic reactions in children.
- Drug allergies (especially penicillin)

Risk Factors (continued)

- Muscle relaxants are the most common cause of anaphylaxis during anesthesia (even with no previous exposure).
- Latex allergy (more common in children with spina bifida or congenital genitourinary abnormalities who have had multiple surgical procedures)
- Insect bites (bees, wasps) or contact with animals
- Higher incidence of anaphylaxis in children with asthma

Prevention

Obtain a detailed history of previous allergic reactions, atopy, and asthma. If there is any question of a latex allergy, treat the child in a latex-free environment. Prophylactic medications to prevent anaphylaxis are not recommended because they may mask a true reaction and delay immediate diagnosis and treatment. Follow-up with an allergy specialist is important to reduce the incidence of future anaphylactic reactions.

Special Considerations

Doses of epinephrine used to treat anaphylaxis are lower than the doses recommended for treating cardiac arrest. Overdosing epinephrine can lead to severe hypertension, supraventricular tachycardia, or ventricular dysrhythmias. Between 5%–20% of patients will have a recurrence of anaphylaxis 8–12 hours after initial presentation. Respiratory abnormalities are the predominant finding with anaphylaxis in children, in contrast with adults where cardiovascular instability is more commonly seen.

Further Reading

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Asthma, Status Asthmaticus**Definition**

Reversible airway obstruction caused by bronchial smooth muscle constriction, mucosal edema, airway inflammation, and secretions. This airway obstruction increases resistance to air flow within the lower airways. Occurs in response to a variety of stimuli including airway instrumentation.

Presentation

- Expiratory wheezing caused by airway obstruction
- Dyspnea and/or tachypnea with prolonged expiratory phase
- Hypoxemia resulting from ventilation-perfusion mismatch
- Increasing cough with sputum production
- Chest pain or tightness
- As the child fatigues, air movement may decrease to the point that wheezing is no longer heard.

Pathophysiology

Acute asthma is caused by the local release of various chemical mediators, immune mechanisms that lead to degranulation of bronchial mast cells and overactivity of the parasympathetic nervous system.

Immediate Management

- Increase FiO_2 to 100% to maintain oxygenation.
- Administer a nebulized β_2 -agonist, such as albuterol; continuous use at 5–10 mg/h may be required
- Intravenous or oral corticosteroids (e.g., methylprednisolone 1–2 mg/kg) to reduce inflammation
- Methyxanthines (e.g., theophylline or aminophylline) may be given, but are considered a second-tier treatment.
- Parasympathetic antagonists (atropine, nebulized ipratropium) may also be used in the management of severe asthma.
- Asthma refractory to initial medical therapy may require endotracheal intubation and mechanical ventilation.
- For emergency surgery, anesthesia may be induced with standard anesthetic agents; consider a rapid-sequence induction for a full stomach.
- Ketamine acts as a bronchodilator and may be beneficial as an induction agent, but an anticholinergic agent (atropine, glycopyrrolate) should be given first to decrease secretions.

DIFFERENTIAL DIAGNOSIS

- Bronchiolitis (viral infection, infants less than 1 year old, tachypnea, retraction, wheezing)
- Inhalation injury (based on exposure history)
- Anaphylaxis (sudden onset, exposure to allergen, urticaria, hypotension)
- Foreign body aspiration (trachea or esophagus; history of choking while eating with persistent cough, object may be identified on neck or chest radiograph)

- Recurrent pulmonary aspiration of gastric contents
- Tracheomalacia/bronchomalacia
- Bronchial stenosis
- Mediastinal mass
- Vocal cord dysfunction

Diagnostic Studies

- Hyperinflation of the lungs due to air trapping may be seen on chest radiograph.
- Arterial blood gas will help assess oxygenation and carbon dioxide elimination (only necessary for severely compromised patients).

Subsequent Management

- A short course of oral steroids (prednisone) may be necessary.
- Intravenous bronchodilators, such as terbutaline, may also be considered.
- If conventional therapies fail in the intubated child, administration of potent volatile anesthetics or extracorporeal membrane oxygenation may be beneficial.
- Following emergency surgery (or in the presence of a full stomach), extubation should only occur once the child is completely awake and upper airway reflexes are restored.
- Monitor for hypokalemia, which is a side effect of high-dose β -agonist therapy.
- If no risk factors for aspiration are present, suction the patient's stomach and extubate under deep anesthesia.

Risk Factors

- Sensitivity or allergies to pollens, dusts, pollutants, animal dander, or other airborne substances (approximately 80% of children who have asthma also have allergies)
- Exercise (especially in cold, dry conditions), emotional excitement, and exposure to strong odors (chemicals or cigarette smoke) can precipitate an asthma exacerbation.
- Upper respiratory infections
- Children of parents with asthma are at much higher risk of developing the disease (6% general population, 30% if one parent has asthma, 70% chance if both parents have asthma).

Prevention

Medical management and minimizing exposure to triggers can reduce the incidence of acute attacks. Children with chronic asthma

are treated with daily inhaled corticosteroids (such as fluticasone) and long-acting bronchodilators (salmeterol), along with leukotriene inhibitors (such as montelukast) and cromolyn sodium, as needed.

Special Considerations

A near-normal or increasing PaCO_2 in the presence of significant tachypnea is a sign of impending respiratory failure.

Further Reading

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Burns

Definition

Thermal injury to the skin that disrupts the body's ability to regulate temperature, provide a barrier to infection, and maintain fluid and electrolyte balance. First-degree burns are limited to the epithelium, second-degree burns involve the dermis, and third-degree burns are full-thickness.

Presentation

- Variable injuries to the skin or deeper tissues
- Massive fluid shifts from vascular compartment into the burned tissues causing significant hemoconcentration and edema
- Inhalation injury that may be associated with hypoxemia, carbon monoxide poisoning, or airway edema and obstruction
- Pulse oximetry values may be normal despite carbon monoxide poisoning.
- CNS dysfunction caused by inhalation of toxic chemicals, or hypoxic encephalopathy (hallucinations, delusions, seizures, coma)
- Small surface injury with massive underlying tissue damage (electrical burns)

Pathophysiology

- Generalized increase in vascular permeability and hypovolemia due to sequestration of fluid in the burned area and evaporative losses
- Decreased preload and decreased cardiac output causing hypoperfusion, hypotension, metabolic acidosis, and renal failure if resuscitation is inadequate
- Myocardial depression also occurs after extensive burns.

- Smoke inhalation and thermal injury directly damages respiratory epithelium, resulting in sloughing of respiratory tract mucosa, impairment of normal mucociliary function, bronchial edema, loss of surfactant, and necrotizing bronchitis.
- Sepsis, metabolic derangements, and neurohumoral responses are also common.

DIFFERENTIAL DIAGNOSIS

- Severe smoke inhalation and carbon monoxide or cyanide poisoning (even in the absence of external injuries)
- Severe unexplained metabolic acidosis may be due to cyanide inhaled from burning plastics.
- Chemical injury: pain or numbness, blisters or necrotic skin, may have extensive underlying injury
- Electrical burns: small surface injury with massive underlying tissue damage and necrosis, myoglobinuria
- Stevens-Johnson Syndrome: severe inflammatory eruption of the skin and mucous membranes caused by allergic reaction or following a respiratory infection
- Non-accidental burns (“glove and stocking” appearance due to immersion in hot water)

Immediate Management

- Administer 100% humidified oxygen to decrease carboxyhemoglobin concentration
- Early intubation and mechanical ventilation for any child with facial burns or potential inhalation injury
- Aggressive resuscitation with IV fluids (estimate with Parkland formula, crystalloid fluids: $4 \text{ mL/kg} \times \text{percent burn} \times \text{wt (kg)}$; give half in first 8 hours and remainder during next 16 hours)
- Full stomach precautions (decreased gastrointestinal function immediately following thermal injury). Rapid sequence induction with succinylcholine is appropriate within the first 24 hours after a burn (succinylcholine is contraindicated after 24 hours and for a prolonged period thereafter due to a profound hyperkalemic response).
- Induction of anesthesia with thiopental, propofol, or ketamine; ketamine may be preferred if hypovolemia is suspected. Slow inhalation induction with sevoflurane and fiberoptic intubation for children with compromised airway. Cuffed endotracheal tubes (with little or no cuff inflation) do not need replacement as edema decreases (if continued postoperative intubation is planned).
- Maintenance of anesthesia with oxygen, nitrous oxide, muscle relaxant, and an opioid or inhalation agent

Immediate Management (continued)

- Aggressive warming to maintain normal body temperature—use warming blankets, warm intravenous fluids, elevated ambient room temperature, and forced-air warming devices.
- Analgesia and sedation with IV opioids, such as fentanyl (minimize histamine release), ketamine, and midazolam.

Diagnostic Studies

- Direct measurement of carboxyhemoglobin and cyanide level in the blood (to guide treatment)
- Serial blood gas measurements (to monitor for adequacy of ventilation, oxygenation, and resuscitation)
- Chest radiograph if inhalation injury is suspected

Subsequent Management

- Establish arterial and central venous access (to monitor for major blood loss and fluid shifts); large-bore single lumen catheters may be more helpful than a multilumen catheter.
- Placement of urinary catheter (with hourly measurements of urine output)
- Fluid resuscitation is guided by systolic blood pressure, heart rate, urinary output (0.5–1 mL/kg/h), central venous pressure, and pH (excessive fluids may result in pulmonary edema and increased tissue edema). Administer blood products as necessary.
- Topical antibiotic and antibacterial therapy (to prevent burn wound sepsis)
- Hyperbaric oxygen treatment is only indicated for acute, symptomatic CO poisoning in a hemodynamically stable child who is not wheezing and does not require ongoing burn resuscitation.
- Treatment for cyanide toxicity includes sodium thiosulfate, sodium nitrate 3% solution, and/or hydroxycobalamin.

Risk Factors

- Children less than 5 years old (50% of burn patients)
- Inadequate supervision of small children around hot liquids, candles, campfires, electrical cords, and other hot appliances. Scalding caused by reaching for hot liquids is the most common burn injury in children.

Prevention

Children are admitted and present to the operating room with this injury. The only preventive strategy is to avoid operating room fires.

Special Considerations

The half-life of COHb is approximately 5 hours while breathing room air, but decreases to 90 minutes when breathing 100% oxygen. Most common indications for surgical intervention in burn patients include wound debridement, skin grafting, and correction of contractures. Children with burn injuries rapidly develop tolerance to most opioids and sedatives and eventually require higher doses. Burn patients also demonstrate marked resistance to nondepolarizing muscle relaxants. Chronic ionized hypocalcemia is common and should be monitored and corrected. Pulmonary complications are a major contributing factor or cause of death in patients with major burn injuries.

Further Reading

Campo TD, Aldrete JA. The anesthetic management of the severely burned patient. *Intensive Care Med.* 1981;7:55–62.

Shank ES, Sheridan RL, Cote CJ, Jeevendra Martyn JA. Burn Injuries. In: Cote CJ, Lerman J, Todres ID (eds.). *A Practice of Anesthesia for Infants and Children.* 4th ed. Philadelphia: Saunders Elsevier; 2009:715–733.

Drowning and Near Drowning

Definition

Suffocation by submersion in a liquid, usually water. Near drowning is suffocation while submerged with recovery for at least some period of time.

Presentation

- Varies greatly, ranging from no symptoms to coma and multiple organ impairment
- Hypoxemia due to intrapulmonary shunting
- Hypothermia (may provide some brain protection if the patient was in very cold water)
- Metabolic acidosis
- Hypovolemia due to brisk diuresis (hypothermia decreases ADH production early in submersion)
- Clinically significant alterations in fluids and electrolytes in less than 15% of submersion victims
- Dysrhythmias or cardiac dysfunction (due to hypoxemia, hypothermia, or acidosis)

Pathophysiology

90% of patients aspirate water or some other debris into the lungs (“wet drowning”). Laryngospasm without aspiration of water may also occur

(“dry drowning”). Pulmonary injury, particularly intrapulmonary shunting of blood through poorly ventilated areas, is the primary pathophysiologic process. Loss of surfactant due to denaturation (fresh water) or washout (salt water) also occurs. Alveolar injury and pulmonary edema cause hypoxemia due to intrapulmonary shunting and ventilation-perfusion mismatch. Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) commonly develop after resuscitation.

DIFFERENTIAL DIAGNOSIS

Spinal cord injury (may occur after diving in shallow water)

Immediate Management

- Cervical spine immobilization for all suspected diving accidents
- Remove debris or fluid from the airway.
- Intubate the patient if apneic, in severe respiratory distress, or has loss of protective airway reflexes.
- Increase FiO_2 to maintain oxygenation.
- Continuous positive airway pressure (CPAP) or positive pressure ventilation with positive end-expiratory pressure (PEEP) improves ventilation-perfusion mismatching in children with severe hypoxia.
- Perform nasogastric suctioning to empty the stomach after airway is protected.
- Avoid abdominal thrusts (which may increase the risk of aspirating gastric contents) unless a foreign object is obstructing the airway.
- Assess adequacy of circulation; if child is pulseless begin chest compressions.
- Obtain large-bore IV access; administer 20 mL/kg IV fluids; boluses of balanced salt solution if child is hypotensive or shows signs of poor perfusion.

Diagnostic Studies

- Diagnosis is made by history; useful laboratory data include electrolytes, arterial blood gas, and toxicology screens.
- Chest radiograph to evaluate pulmonary involvement.

Subsequent Management

- If patient is hypothermic, rewarm over a few hours with heating blankets or radiant warmer. If body temperature is less than 32° use gastric lavage, cardiopulmonary bypass, or other core warming techniques.

- Surface warming using blankets or radiant warmers may induce peripheral vasodilation and profound hypotension.
- Continue advanced cardiac life support (ACLS) until the core temperature has reached 37°.
- Bronchospasm may be treated with bronchodilators (e.g., albuterol).
- Correct electrolyte and metabolic abnormalities.
- Children who are asymptomatic but have a history of submersion or resuscitation should be observed for a minimum of 4 hours prior to discharge.
- Children with any lower respiratory involvement should be admitted and observed for progressive respiratory failure.

Risk Factors

- Most common in previously healthy patients, especially ages 0–4 years old
- More common in males in all pediatric age groups
- Use of alcohol while participating in water activities
- Children with seizure disorders, history of syncope, or mental retardation in a pool or near water, especially if unsupervised
- Patients with congenital prolonged QT syndrome (swimming in very cold water can trigger a fatal arrhythmia)

Prevention

Patients are admitted to the emergency department, intensive care unit, or operating room with this condition.

Special Considerations

- Infants less than one year of age are most likely to drown in bathtubs, buckets, or toilets.
- Adolescents drown most commonly in rivers, lakes, and canals. Alcohol or drugs are involved in 50% of cases.
- The type of water (fresh versus salt) makes little difference in the management of the patient.

Further Reading

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Epiglottitis

Definition

Sudden onset inflammatory edema of supraglottic structures, including the arytenoids, aryepiglottic folds, and epiglottis, causing swelling and stiffening that can rapidly lead to complete airway obstruction.

Presentation

- Abrupt appearance of high fever, severe sore throat, and difficulty swallowing
- Inspiratory stridor with little or no hoarseness
- Child will appear toxic and insist on sitting up and leaning forward in a sniffing position.
- Mouth may be open with drooling and protruding tongue.
- Slow to normal respiratory rate
- Cough is usually absent.

Pathophysiology

The most common causative organism was *Haemophilus influenzae* type B; however, since the widespread administration of the *H. influenzae* vaccine, the incidence has drastically decreased. Group A β -hemolytic *streptococcus*, *staphylococcus*, *candida*, and other fungal pathogens are now more frequent causes of this rare infection.

Immediate Management

- Transport patient to the operating room or ICU as soon as the diagnosis is made. Examination of the pharynx and larynx should only be performed in a controlled setting (ideally, the OR), not in the emergency department.
- Patient should be transported by a physician and with adequate equipment to immediately establish an airway.
- OR setup should include equipment and personnel capable of establishing a surgical airway if needed.
- Inhalation induction of general anesthesia with sevoflurane (or halothane). Child should be kept in the sitting position during induction and spontaneous ventilation should be maintained. Avoid use of IV induction agents or muscle relaxants that may cause apnea.
- Establish IV access after surgical anesthesia has been established.
- Endotracheal intubation following direct laryngoscopy with gentle cricoid pressure. Carefully lift the base of the tongue without touching or traumatizing the epiglottis.

Immediate Management (continued)

- ETT should be one-half size smaller than normal (to decrease risk of pressure necrosis). A stylet may be helpful in facilitating intubation though partially obstructed glottis aperture. Nasotracheal intubation is preferred.

DIFFERENTIAL DIAGNOSIS

- Croup (gradual onset, barking cough, hoarseness, rapid respiratory rate)
- Foreign bodies in the airway or esophagus (history of choking while eating; sudden, persistent cough)
- Bacterial tracheitis (barking cough, no dysphasia, sore throat, or drooling)
- Retropharyngeal abscess (airway obstruction, stridor)

Diagnostic Studies

- Lateral neck radiograph will show thumb print-like mass (not required for diagnosis, should only be attempted in stable children).
- Definitive diagnosis is made on direct visualization; however, direct laryngoscopy should only be attempted in the operating room with airway equipment and physician skilled in airway management immediately available.

Subsequent Management

- IV hydration with 10–30 mL/kg of normal saline or lactated Ringer's solution (child may be dehydrated from decreased oral intake)
- Ventilation with humidified gases
- Obtain blood cultures and begin aggressive antibiotic therapy.
- Transport the child to ICU postoperatively; sedation with opioids and benzodiazepines. Ideally, child should be breathing spontaneously.
- The child can usually be extubated within 1–3 days (when the supraepiglottic and periepiglottic swelling has significantly decreased).

Risk Factors

- Decreased occurrence with the widespread use of the *H. influenzae* vaccine
- Most commonly seen in children age 3–6 years
- Occurs rarely (0.63 per 100,000 in 1990)

Prevention

Patients are admitted with this condition. Patients should be managed cautiously in order to prevent exacerbation of airway obstruction.

Special Considerations

Use of lateral neck films is controversial since complete airway obstruction can occur during the time it takes to obtain the film. The urgency of securing the airway in this situation takes precedence over the risks associated with a “full stomach.”

Further Reading

Verghese ST, Hannallah RS. Pediatric otolaryngologic emergencies. *Anesthesiol Clin North America*. 2001 Jun;19(2):237–256.

Inhaled Foreign Body

Definition

Aspiration of a foreign body into the respiratory tract, or posterior compression of the trachea caused by an object obstructing the esophagus.

Presentation

- History of choking and cyanosis while eating
- Respiratory distress, tachypnea, and tachycardia
- Sudden onset, persistent cough
- Monophonic wheezing or stridor
- Absent breath sounds on one side
- Emotional distress, agitation (may be due to underlying hypoxemia)
- Cyanosis (suggests foreign body in trachea or multiple foreign bodies obstructing both bronchi)

Pathophysiology

Acute onset airway obstruction may be supraglottic, glottic, or subglottic. Food is the most common foreign body aspirated by young children, but any small object that can be put into a child's mouth (e.g., beads, coins, toys, etc.) may result in airway obstruction if it is aspirated into the trachea or becomes lodged in the esophagus.

Immediate Management

- Administer 100% oxygen to correct hypoxemia.
- Keep child calm to prevent airway collapse associated with agitation.

Immediate Management (continued)

- If the child is stable, radiographic examination may help identify and localize the foreign body.
- **If there is any doubt as to whether airway obstruction is life-threatening, immediately transport the child to the operating room, accompanied by a physician skilled in airway management and emergency airway equipment, including rigid bronchoscope.**
- If complete airway obstruction occurs, the foreign object should be immediately removed by rigid bronchoscopy, or dislodged distally to allow for adequate ventilation and oxygenation.
- For supraglottic and glottic obstruction, maintain spontaneous respirations and perform an inhalation induction with sevoflurane.
- For subglottic obstruction, a rapid-sequence induction or inhalation induction is followed by rigid bronchoscopy, or careful endotracheal intubation and flexible bronchoscopy.
- Maintain anesthesia with 100% O₂ and propofol infusion (steady state of anesthesia independent of ventilation), with or without muscle relaxant (ensure that the patient does not move or cough).
- Intubate with an ETT following bronchoscopic removal of foreign body.

DIFFERENTIAL DIAGNOSIS

- Croup (gradual onset, barking cough, hoarseness, rapid respiratory rate).
- Foreign body in the esophagus (drooling, dysphasia, dyspnea).
- Epiglottitis (acute onset, toxic appearance, sore throat, stridor).
- Acute asthma exacerbation (wheezing, respiratory distress, prolonged expiratory phase).
- Anaphylaxis (sudden onset, exposure to allergen, urticaria, hypotension).
- Retropharyngeal abscess (airway obstruction, stridor).

Diagnostic Studies

- Chest and neck radiographs with lateral views will help confirm the presence and location of the foreign body; distal hyperinflation from air trapping or atelectasis may be seen.

Subsequent Management

- Administer a beta agonist bronchodilator (e.g., albuterol) to decrease postoperative wheezing.

- Nebulized racemic epinephrine (0.5 mL of 2.25% solution diluted to 1:6) or steroid therapy (dexamethasone 0.5–1 mg/kg) may be necessary to treat severe airway edema.

Risk Factors

- More common in infants or toddlers (age 1–3 years old) who tend to put objects in their mouths (popcorn and peanuts are particularly dangerous)
- May be associated with dislodgement of teeth during airway manipulation.

Prevention

Careful laryngoscopy and removal of loose teeth prior to laryngoscopy.

Special Considerations

The majority of foreign bodies (95%) become lodged in the right mainstem bronchus. Complications can include aspiration pneumonia or bacterial infection, airway rupture, hemoptysis, pneumothorax, or severe bronchospasm. Peanuts can cause an intense inflammatory reaction if aspirated.

Further Reading

Verghese ST, Hannallah RS. Pediatric otolaryngologic emergencies. *Anesthesiol Clin North America*. 2001;19:237–256.

Major Trauma

Definition

The “disease of injury,” usually affecting multiple organ systems, depending on mechanism and severity of the trauma. Major trauma involves a constellation of the most critical and severe injuries, often resulting in death.

Presentation

- Extremely variable depending on type and extent of injury
- Altered level of consciousness, confusion (75% of children presenting with a major trauma have concomitant head injury)
- Potentially unstable spinal cord injury
- Blunt or penetrating abdominal or thoracic injury (ruptured liver or spleen are common)
- Acute bleeding (internal or external)

- Hypoxia (from pulmonary contusions, aspiration, or pneumothorax)
- Tachycardia (initial symptom with acute blood loss)
- Hypotension (late finding, only after significant blood loss)
- Severe pain (especially from unstable fractures)

Pathophysiology

The stress response associated with multisystem trauma stimulates the sympathetic nervous system, increasing the levels of circulating catecholamines. Metabolic derangements, hyperglycemia, and coagulopathy are common.

Primary brain injury (PBI) results from contusions, lacerations, diffuse axonal injury and dural tears which are caused by acute acceleration and/or deceleration of the head.

Secondary brain injury is caused by complications of PBI such as local or global brain ischemia, hypercapnia, hyper- or hypotension, electrolyte or metabolic disturbances, or intracranial hematomas.

DIFFERENTIAL DIAGNOSIS

- Spinal cord injury.
- Traumatic brain injury (including intracranial, subdural, or epidural hematoma).
- Intraperitoneal or retroperitoneal injury or bleeding.
- Vascular or orthopedic injuries.
- Child abuse (injuries inconsistent with history, multiple burns, intracranial bleeding, rib fractures, eye contusions).

Immediate Management

- Primary survey, initial resuscitation, and cervical spine immobilization begin simultaneously.
- Provide 100% oxygen by face mask to maintain $\text{SaO}_2 > 90\%$; if inadequate, begin bag-valve-mask ventilation.
- Endotracheal intubation is required for severely injured children (Glasgow Coma Score [GCS] < 8); if the patient was intubated at the scene, confirm proper ETT size and placement.
- Awake intubation (no medications) for infants with airway obstruction (blood, foreign body, vomitus) and infants or children with sufficiently depressed consciousness.
- Rapid-sequence intubation indicated for all trauma patients; administer analgesic (fentanyl), vagolytic, (e.g., atropine) to avoid bradycardia, and lidocaine to reduce hemodynamic response to intubation. Selection of a specific induction agent depends on individual circumstances. Etomidate is

Immediate Management (continued)

recommended for hemodynamically unstable patients with head injury. Muscle relaxation with either succinylcholine (2 mg/kg) or rocuronium (1.2 mg/kg).

- Intubate with manual in-line stabilization.
- If difficult airway, consider fiberoptic bronchoscope, GlideScope[®], or Bullard[®] laryngoscope or blind placement of LMA or light wand (emergency situation). Use LMA as a conduit to definitively secure the airway. Emergency cricothyroidotomy or tracheostomy may be required.
- Assess and control bleeding (may require emergent surgical intervention).
- Insert orogastric tube to decompress the stomach.
- Obtain vascular access, at least 2 large-bore IV lines (usually in the antecubital fossa and femoral veins); if difficult access, consider intraosseous cannulation (tibia, femur, iliac crest, clavicle, or sternum) or scalp veins in neonates and infants.
- Begin IV fluid boluses with a balanced salt solution, 20 mL/kg to stabilize systolic blood pressure. Consider blood products if bleeding is suspected or if infusion of IV fluids surpasses 80–100 mL/kg (overhydration can worsen pulmonary and cerebral edema).
- Placement of invasive monitors (arterial line, central venous catheter) and bladder catheter.

Diagnostic Studies

- Chest radiograph for all patients with major trauma
- Head CT is indicated for GCS <9, skull fractures, any neurologic symptoms, or Cushing's response (bradycardia, hypertension, irregular respirations).
- Depending on injuries and hemodynamic stability, skeletal survey, CT scans, ultrasonography (FAST scan), or angiography may be indicated.

Subsequent Management

- The goal of management in head trauma is to prevent secondary brain injury. Prevent hypotension and maintain adequate cerebral perfusion pressure (>60 mmHg).
- Decrease intracranial pressure (ICP), if necessary, through hyperventilation (PaCO₂ between 25–35 mmHg), mannitol administration (0.25–1 g/kg), drainage of cerebrospinal fluid (CSF), diuretic therapy (furosemide), sedation, analgesia,

pentobarbital, or bolus administration of hypertonic saline (if serum Na^+ is <150 and osmolality is <300 mOsm/L).

- Seizure prophylaxis for head injuries (phenobarbital or phenytoin)
- Corticosteroids are indicated for spinal cord injuries.
- Carefully monitor and correct for ongoing electrolyte disturbances, metabolic acidosis, coagulation disorders, and acute blood loss.
- Avoid using glucose-containing solutions (especially for traumatic brain or spinal cord injuries) unless hypoglycemia is documented. The optimal range for plasma glucose is between 80 and 120 mg/dL.
- Maintain normothermia with forced-air warming systems, blood-warming devices, and increased ambient OR temperature.
- Manage acute pain with opioids, nonsteroidal anti-inflammatory drugs (NSAIDS), acetaminophen, low-dose ketamine infusions, or regional anesthetic techniques (particularly for orthopedic injuries). Patient-controlled analgesia (PCA) may be appropriate for children 5 years or older.

Risk Factors

- Motor vehicle collisions, particularly when seat belts are not in use
- Home accidents (falls, electrical injuries, burns, drowning)
- Homicide, suicide
- For infants under one year of age, non-accidental injury (child abuse) is the most common cause of head injury.

Prevention

Government policies, road safety campaigns, and educating children and their families are the best ways to reduce the incidence of preventable injuries.

Special Considerations

Traumatic brain injury (TBI) is the most common cause of death from injury in all age groups. Optimizing cerebral perfusion and minimizing extension of injury (i.e., controlling ICP) are of paramount importance. Hyperventilation should be used only as a bridge to definitive treatment (e.g., surgery) and should be avoided unless intracranial hypertension is severe because the resulting vasoconstriction and decreased cerebral blood flow may worsen outcome) Children can sustain major blood loss ($>25\%$ of their blood volume) with minimal change in vital signs; therefore, hypovolemic shock may ensue

with little warning. Goals of care include avoiding hypovolemia, coagulopathy, and hypothermia (the “fatal triad”).

Further Reading

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Lam WH, Mackersie A. Paediatric head injury: incidence, aetiology, and management. *Paed Anaesth.* 1999;9:377–385.

Neonatal Resuscitation

Definition

Resuscitation of a neonate undergoing the transition from intrauterine to extrauterine life; any newborn requiring cardiopulmonary resuscitation during the first few weeks to months of life.

Presentation

- Preterm delivery (less than 37 weeks gestation)
- Respiratory distress or apnea (caused by immature lungs or low level of pulmonary surfactant)
- Cyanosis (due to hypoxia)
- Bradycardia (less than 100 bpm)
- Intracranial hemorrhage (due to immature blood vessels in the brain)
- Hypothermia (due to large surface area and thin skin)
- Congenital abnormalities or syndromes

Pathophysiology

Neonatal depression is most commonly caused by intrauterine asphyxia during labor, which may be due to maternal hypotension or hypoxia, umbilical cord compression, or uteroplacental insufficiency leading to progressive fetal hypoxia and lactic acidosis. Other causes for neonatal depression include congenital cardiac or pulmonary diseases, prematurity, maternal fever or infection, maternally administered drugs (opioids, barbiturates, etc.), and anemia secondary to Rh isoimmunization.

DIFFERENTIAL DIAGNOSIS

- Neonatal asphyxia or hypovolemia (late decelerations in utero, hypotension, loss of palpable pulse)
- Diaphragmatic hernia (difficult to ventilate, scaphoid abdomen, bowel sounds present in the chest)
- Hydrops fetalis (leading to hemolytic anemia)

- Congenital complete heart block (suspect when heart rate does not increase above 60 bpm)
- Pneumothorax (can develop during resuscitation, decreased breath sounds)
- Residual drug effects or withdrawal (i.e., opioids administered to the mother prior to delivery or maternal addiction to drugs or alcohol)

Immediate Management

- Warm the patient immediately after birth (radiant heat source, preterm babies should be covered in plastic wrapping).
- Position head in “sniffing” position to open the airway, clear airway secretions (using bulb suction or suction catheter), dry, and stimulate the baby.
- Meconium-stained infants require endotracheal suctioning immediately after birth (not intrapartum) ONLY if they are not vigorous (heart rate less than 100 bpm with poor muscle tone and respiratory effort).
- Allow 30 seconds for initial assessment; spend the next 30 seconds simultaneously evaluating heart rate, respirations, and color.
- Begin positive pressure ventilation (using self-inflating bag, flow-inflating bag, or T-piece) at a rate of 40–60 breaths per minute if infant is apneic or gasping, heart rate is between 60 and 100 bpm, or persistent central cyanosis is present.
- Supplementary oxygen is recommended whenever positive pressure ventilation is indicated (also reasonable to begin with room air, but oxygen should be readily available and used if hypoxia does not improve).
- If bag-mask ventilation is unsuccessful, endotracheal intubation is indicated (LMA may be used as an alternative if unable to place ETT). Avoid excessive tidal volumes and peak airway pressures. Use initial inflation pressures of 20–25 cm H₂O; higher pressures may be necessary if heart rate does not improve or chest wall movement is not seen.
- Begin chest compressions if heart rate is less than 60 bpm despite adequate ventilation with oxygen for 30 seconds. Compress the lower one third of the sternum to a depth one third the anterior-posterior diameter of the chest at a rate of 120 bpm. Three chest compressions should be given for each breath. The two thumb-encircling hands technique is recommended.
- Give epinephrine (0.01–0.03 mg/kg IV is preferred, or 0.1 mg/kg via ETT) if heart rate remains below 60 bpm despite adequate

Immediate Management (continued)

ventilation with 100% oxygen and chest compressions. Repeat every 3–5 minutes.

- Continue coordinated chest compressions and ventilations until spontaneous heart rate is greater than 60 bpm. Reassess respirations, heart rate, and color every 30 seconds.

Diagnostic Studies

Monitor arterial blood gas (monitor adequacy of ventilation) and glucose level (maintain within the normal range) regularly during resuscitation.

Subsequent Management

- Consider volume expansion with isotonic crystalloid 10 mL/kg if neonate appears to be in shock or is not responding to other resuscitative measures.
- Consider naloxone (0.1 mg/kg IM or IV) if respiratory depression persists despite adequate heart rate and color.
- Use plastic wrapping and radiant heat to maintain normothermia.
- Most infants less than 28 weeks gestation will require supplemental surfactant (most effective if administered as early as possible).
- If no signs of life are present after 10 minutes of adequate and continuous resuscitation, discontinuing resuscitation is appropriate.

Risk Factors

- Preterm or instrumented (suction, forceps) delivery
- Fetal distress, hypoxia, and acidosis (noted by prolonged late decelerations and decreased heart rate variability)
- Thick meconium present at delivery
- Fetal hypovolemia (due to maternal hemorrhage, sepsis, twin-to-twin transfusion, etc.)
- Infants born to febrile mothers (increased incidence of respiratory distress, seizures, and cerebral palsy)
- Maternal opioid administration near time of delivery (or drug and alcohol use during pregnancy)
- General anesthetic required for cesarean section

Table 9.1 Apgar Score

Clinical Feature	0	1	2
Color	Pale/blue	Pink body, blue extremities	Pink
Heart Rate (beats/minute)	Absent	<100	>100
Response to Stimulation	None	Grimace	Pulls away, crying
Muscle Tone	Limp, flaccid	Some flexion of extremities	Active movement
Respiratory Rate	Absent	Poor effort, slow, irregular	Good, crying

Table 9.2 Endotracheal tube diameters and lengths according to gestation and weight

50% weight by gestation		Tube diameter and length at the lip	
Gestation (weeks)	Body weight (kg)	Diameter (mm)	Length (cm)
23/24	0.5	2.5	6
26	0.75	2.5	6.5
27	1	2.5	7
30	1.5	2.5	7.5
33	2	2.5–3	8
35	2.5	3	8.5
37	3	3–3.5	9
40	3.5	3.5	9.5

Special Considerations

Potential adverse effects of 100% oxygen administration should be balanced with risk of hypoxic injury (no clear data to support a specific FiO_2). Heart rate and pulse oximetry guide therapy. Naloxone should be avoided in neonates whose mothers may have a history of chronic opioid use (may cause seizures). The 1-minute Apgar score correlates with survival; the 5-minute score is related to neurological outcome. Withholding resuscitation is reasonable with extreme prematurity (less than 23 weeks or birth weight less than 400 grams),

anencephaly, and chromosomal abnormalities incompatible with life (trisomy 13 or 18).

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 13: neonatal resuscitation guidelines. *Circulation*. 2005;112:IV-188–IV-195.

The International Liaison Committee on Resuscitation (ILCOR). Consensus on science with treatment recommendations for pediatric and neonatal patients: neonatal resuscitation. *Pediatrics*. 2006;117(5):978–984.

Pediatric Basic Life Support

Definition

Initial management of sudden arrest of an infant (less than 1 year of age) or child (age 1 year until start of puberty), including basic cardiopulmonary resuscitation (CPR).

Presentation

- Unresponsive infant or child (may have no signs of respiration, with or without a pulse)
- Sudden respiratory arrest (choking, foreign body aspiration)
- Sudden cardiac arrest (usually caused by hypoxia)
- Ventricular fibrillation or pulseless ventricular tachycardia (e.g., child suddenly collapsing at a sporting event)

Pathophysiology

Asphyxia leading to hypoxia is the most common cause of cardiac arrest in children. Various mechanisms—for example, trauma, foreign body airway obstruction, drowning, inhalational injuries, or pre-existing cardiac or respiratory disease—may cause hypoxia. VF is the cause of cardiac arrest in 7% to 15% of infants and children.

DIFFERENTIAL DIAGNOSIS

- Foreign body causing airway obstruction (child unable to vocalize in the setting of complete obstruction)
- Traumatic injury (consider the possibility of brain or spinal cord injury)
- Cardiac arrest, VF, or pulseless VT
- Anaphylaxis (sudden onset respiratory distress, urticarial rash)
- Poisoning, exposure to toxic gases

Immediate Management

- If the child is responsive but in need of medical attention, call for help. If alone, leave the child to call for assistance and return immediately.
- If unresponsive, call for help and begin CPR. If alone, perform 5 cycles of CPR, then call EMS (if outside the hospital) or the code team and get an automated external defibrillator (AED). If the collapse was witnessed, call for help and apply the AED before beginning CPR (etiology is most likely to be VF).
- Open airway with head tilt—chin lift maneuver (use jaw thrust without head tilt if cervical spine injury is suspected). Look, listen, and feel for breathing for no more than 10 seconds.
- If the child is breathing, turn the child onto the side and continue to monitor for breathing.
- If child is not breathing, maintain open airway, pinch the nose closed, and give 2 breaths mouth-to-mouth (be sure to watch chest rise). For infants, use mouth-to-mouth-and-nose technique. Use bag-mask ventilation with 100% oxygen if available.
- Check for pulse (brachial in an infant, carotid or femoral in a child). If pulses are present but not breathing, continue to deliver 12–20 breaths per minute and reassess pulse every 2 minutes.
- If no pulse is palpated after 10 seconds, or if pulse is less than 60 bpm with signs of poor perfusion, begin chest compressions.
- Chest compressions should depress the chest one third to one half the anterior-posterior diameter of the chest, at a rate of 100 compressions per minute. Release completely to allow chest to fully recoil.
- For *infants*, the 2 thumb-encircling hands technique is recommended. Place thumbs together over lower sternum and forcefully squeeze the thorax. If alone, or unable to encircle infant's chest with your hands, use the 2-finger (place just below intermammary line) technique to facilitate rapid transition between ventilation and compressions.
- In a *child*, compress the lower half of the sternum with heel of one or two hands (do not press on ribs or xiphoid).
- If alone, perform cycles of 30 chest compressions followed by 2 breaths. For 2-rescuer CPR, the ration should be 15:2. Simultaneous compressions (100 per minute) and ventilation (8–10 per minute) only after advanced airway is in place.
- Apply the AED and follow the prompts (for children over 1 year old). Resume chest compressions immediately after

Immediate Management (continued)

shock and continue CPR until pediatric advanced life support is initiated or patient begins to move.

- If pulse is present, but absent or inadequate respirations, continue giving 12–20 breaths per minute.
- For foreign body airway obstruction, if incomplete and the child is able to cough do not interfere. If severe (unable to make a sound):
 - In an *infant*, deliver 5 back blows (between shoulder blades) followed by 5 chest thrusts (to lower sternum); repeat until object is expelled or infant becomes unresponsive (then begin CPR).
 - In a *child*, perform subdiaphragmatic abdominal thrusts (Heimlich maneuver) until object is expelled or child becomes unresponsive.
- If unresponsive, look in the mouth before giving a rescue breath. If you see a foreign body, remove it, but do not perform a blind finger sweep.

Diagnostic Studies

ECG waveform or prompts on the AED.

Subsequent Management

- Transfer patient to a hospital if the event occurs at an outside facility, initiate pediatric advanced life support if needed.
- If resuscitation is successful, continue to closely monitor the child and transfer to an intensive care unit.
- Obtain IV access (for fluid and drug administration) if not already available.

Risk Factors

- Preventable injuries (motor vehicle, pedestrian, bicycle, and firearm injuries, burns, drowning); often due to inadequate supervision of infants and children.
- Primary respiratory, cardiac, or metabolic disease
- Failure to use proper safety restraints and seat belts in cars, drinking and driving, adolescent drivers with teen passengers (nearly half of all pediatric deaths in the US are caused by motor vehicle-related injuries)
- Sudden infant death syndrome (SIDS) in infants 2–4 months old (increased risk with prone sleeping position, sleeping on soft surface, and second-hand smoke)

Special Considerations

Anticipate airway obstruction from broken teeth or other debris. Stabilize the head and neck and minimize movement if cervical spine injury is suspected. Minimize gastric inflation by avoiding excessive peak inspiratory pressures (ventilate slowly) and applying cricoid pressure. Avoid hyperventilation, which will decrease venous return and therefore decrease cardiac output, cause air-trapping and barotrauma, and increase the risk of aspiration.

Short interval time from collapse to initiation of effective CPR is associated with improved survival and neurologic outcomes. Children with cardiac arrest caused by blunt trauma or septic shock tend to have a poor prognosis.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 11: Pediatric basic life support. *Circulation*. 2005;112:IV-156–IV-166.

The International Liaison Committee on Resuscitation (ILCOR). Consensus on science with treatment recommendations for pediatric and neonatal patients: pediatric basic and advanced life support. *Pediatrics*. 2006;117(5):955–966.

Pediatric Advanced Life Support

Definition

Algorithm for resuscitation of an infant or child with cardiac arrest.

Presentation

- Unresponsive child. Pulses may or may not be present.
- Cardiac arrest (usually due to hypoxia)
- Respiratory failure (tachypnea, gasping, grunting, cyanosis)
- Hypotension, tachycardia (decompensated shock)
- Trauma drowning, burn injury, or toxic ingestion
- Under general anesthesia, ECG will indicate a nonperfusing rhythm, sudden drop in $ETCO_2$ and oxygen saturation on pulse oximetry.

Pathophysiology

Various mechanisms—for instance: trauma, massive hemorrhage, drowning, inhalation injury, shock, or primary cardiac or respiratory disease—can lead to hypoxia, the most common cause of cardiac arrest in a child. Ventricular fibrillation (VF) is the cause of cardiac arrest in 7–15% of infants and children. Intraoperatively, extensive blood loss, compression of great vessels, decreased venous return

to the heart, and vagus nerve stimulation may precipitate cardiac arrest.

DIFFERENTIAL DIAGNOSIS

- Respiratory failure (caused by airway obstruction, asphyxiation, etc.)
- Shock (usually hypovolemic)
- Primary cardiac or respiratory disease (i.e., congenital heart disease)
- Metabolic disease or derangement (i.e., DKA, hypothermia)
- Anaphylaxis (urticaria, airway obstruction, cardiovascular collapse)
- Vagal stimulation (due to surgical manipulation)

Immediate Management

- Initiate basic life support (BLS); assessment of airway, breathing, and circulation. Ask the surgical team to discontinue any action that might cause vagal stimulation (e.g., ocular compression, neck compression).
- Attempt defibrillation immediately; do not wait to secure the airway (increased duration of fibrillation decreases the chance of restoring an organized rhythm).
- Increase FiO_2 to 100%.
- Intubate the trachea (equal safety with cuffed and uncuffed tracheal tubes). Confirm ETT placement with ETCO_2 (lack of ETCO_2 may reflect either incorrect ETT placement or lack of effective chest compressions).
- If pulseless arrest, continue CPR (strive to minimize any interruptions in chest compressions) while attaching monitors and defibrillator and obtaining IV access.
- Assess rhythm—
 - If shockable (VT/VF), give 1 shock (manual, 2 J/kg, or apply an AED if available and patient is greater than one year old).
 - Resume 5 cycles (approximately 2 minutes) of CPR and recheck rhythm.
 - If a shockable rhythm is present, give a second shock (4 J/kg) and give epinephrine (IV/IO: 0.01 mg/kg or 0.1 mg/kg through ETT) and immediately resume 5 cycles of CPR.
 - Repeat epinephrine every 3–5 minutes.
- Recheck rhythm—
 - If persistent VF/VT, give another shock (4 J/kg) and consider giving amiodarone (IV/IO: 5 mg/kg) or lidocaine (IV/IO: 1 mg/kg).

Immediate Management (continued)

- Administer magnesium (IV/IO: 25–50 mg/kg) for torsades de pointes (polymorphic VT with prolonged QT interval).
- If defibrillation is successful but VT/VF recurs, continue CPR and give another dose of amiodarone before repeating defibrillation.
- If rhythm is not shockable (asystole or PEA), resume CPR and give epinephrine (IV/IO: 0.01 mg/kg or 0.1 mg/kg through ETT). Repeat epinephrine dose every 3–5 minutes. After every 5 cycles of CPR, recheck rhythm to determine if it is shockable.
- For bradycardia (HR less than 60) causing cardiovascular compromise, increase FiO_2 to 100% and support airway, breathing, and circulation. If poor perfusion persists, begin chest compressions and give standard dose of epinephrine. Repeat epinephrine every 3–5 minutes or begin an infusion, if needed.
- Give atropine (0.02 mg/kg, minimum dose 0.1 mg) if bradycardia is due to vagal stimulation or primary AV block.
- Transcutaneous pacing may be lifesaving for refractory bradycardia due to complete heart block or sinus node dysfunction.
- For tachycardia with a pulse, assess the QRS duration; narrow-complex tachycardia has QRS less than 0.08 seconds and wide-complex tachycardia has QRS duration greater than 0.08 seconds.
- For narrow-complex tachycardia, obtain a 12-lead ECG to differentiate sinus tachycardia and supraventricular tachycardia (SVT). If rhythm is sinus, search for and treat reversible causes.
- For hemodynamically stable SVT, attempt vagal maneuvers first (apply to ice to the face of young children, carotid sinus massage or Valsalva maneuver in older children), then try chemical cardioversion with adenosine (0.1 mg/kg rapid bolus for first dose, 0.2 mg/kg for second dose).
- For unstable SVT, provide synchronized cardioversion (0.5–1 J/kg for first shock, increase to 2 J/kg if second shock is required). Consider amiodarone or procainamide if other treatments are unsuccessful.
- Wide-complex tachycardia is most likely VT. Treat with synchronized cardioversion (2 J/kg, if unsuccessful, increase subsequent doses to 4 J/kg). If second shock is unsuccessful, give amiodarone (procainamide is an alternative) before the third shock.

Diagnostic Studies

- Check arterial blood gases (ABGs), serum electrolytes, glucose, and calcium frequently during resuscitation (consider toxicology screen and carboxyhemoglobin level).
- Obtain chest radiograph (evaluate ETT placement, heart size, and pulmonary status).

Subsequent Management

- Give crystalloid fluid boluses (20 mL/kg) to treat signs of shock.
- Insert an intraarterial catheter to continuously measure blood pressure and allow for frequent blood sampling.
- Evaluate and treat reversible causes of cardiac arrest, as well as any metabolic or electrolyte abnormalities.
- Vasoactive drugs may be necessary to maintain cardiac output (myocardial dysfunction after cardiac arrest is common).
- Avoid hyperventilation, unless signs of impending cerebral herniation are present.
- If patient remains comatose after resuscitation, consider cooling to 32°C to 34°C for 12–24 hours (to improve neurologic outcome).
- Hyperthermia in the peri-arrest period should be aggressively treated (post-arrest fever is associated with a worse outcome).

Risk Factors

- Cardiac arrest is uncommon in children and is usually the terminal event of progressive respiratory failure or shock.
- Reversible causes of pulseless electrical activity (PEA) cardiac arrest:
 - Hypoxia
 - Hypovolemia
 - Hydrogen ions (acidosis)
 - Hypo- or hyperkalemia
 - Hypoglycemia
 - Hypothermia
 - Toxins (drug ingestion)
 - Tamponade, cardiac
 - Tension pneumothorax
 - Thromboembolism, coronary or pulmonary

Special Considerations

All resuscitation drugs, including fluids and blood products, may be given through intraosseous access if IV access cannot be obtained. Lidocaine, epinephrine, atropine, and naloxone (“LEAN”) may be given through ETT. When using a manual defibrillator, use largest paddles or self-adhering electrodes that will fit on the chest without touching (use adult paddles for children over 10 kg). Place paddles over right side of upper chest and to the left of the nipple over left lower ribs. Biphasic defibrillators have greater than a 90% first shock success rate. There is no survival benefit from high-dose epinephrine; it should only be considered in exceptional circumstances, such as β -blocker overdose. Insufficient evidence is available to recommend for or against use of vasopressin during cardiac arrest in children.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 12: Pediatric advanced life support. *Circulation*. 2005;112:IV-167–IV-187.

The International Liaison Committee on Resuscitation (ILCOR). Consensus on science with treatment recommendations for pediatric and neonatal patients: pediatric basic and advanced life support. *Pediatrics*. 2006;117(5):955–966.

Stridor

Definition

Partial to complete obstruction of the respiratory tract causing turbulent airflow.

Presentation

- Noisy breathing, usually high pitched (due to airway obstruction)
- Hypoxemia and hypercarbia (due to respiratory distress)
- Tachypnea and tachycardia
- Cyanosis
- Paradoxical movement of chest wall inward with inspiration
- Suprasternal, intercostal, and substernal retractions

Pathophysiology

Stridor can occur during inspiration, expiration, or both, depending on the level of the lesion. Upper airway obstruction or extrathoracic lesions most commonly cause inspiratory stridor, while lower airway disease or inflammation may cause expiratory stridor. Intrathoracic and mid-tracheal lesions cause biphasic stridor. Etiology can range from viral or bacterial infections to congenital or acquired laryngeal, subglottic, or tracheal stenosis.

Immediate Management

- Administer 100% oxygen by face mask to correct hypoxemia.
- Humidify inhaled gases to prevent drying of secretions.
- Obtain thorough history and physical exam to determine possible etiology of stridor.
- If viral laryngotracheitis (croup) is suspected, begin nebulized racemic epinephrine (0.5 mL of 2.25% solution is standard dose for all children). Repeat every 1–2 hours, as needed.
- Corticosteroids are indicated for croup and airway edema; a single dose is usually sufficient (dexamethasone 0.15–0.6 mg/kg).
- Continuous positive airway pressure (CPAP) may decrease stridor by lowering the pressure gradient across obstructed airway segment.
- Helium-oxygen mixture (Heliox) decreases airflow turbulence and is of benefit in the setting of severe respiratory distress.
- Transport to OR for definitive diagnosis by direct laryngoscopy or bronchoscopy.
- Use caution when administering sedatives. Deep sedation may cause respiratory depression and worsen airway obstruction.
- Keep the child breathing spontaneously in whatever position is most comfortable for him or her.
- Inhalation induction with sevoflurane or halothane (nitrous oxide may be used with caution to speed induction, but should be discontinued prior to examination). Rigid bronchoscope and surgeon capable of treating total airway obstruction should be immediately available.
- Obtain IV access after induction of general anesthesia.
- After spontaneous movement of the vocal cords has been observed, anesthetic level may be increased.
- After completion of rigid bronchoscopy, intubate the trachea, preferably using a nasotracheal ETT (may require smaller than expected ETT size due to subglottic narrowing).

DIFFERENTIAL DIAGNOSIS

- Croup (usually under age 3, gradual onset, bark-like cough, low grade fever, hoarseness)
- Bacterial tracheitis (high fever, toxic appearance, poor response to nebulized epinephrine)
- Epiglottitis (sudden onset, high fever, dysphasia, drooling)

- Acute allergic reaction (rapid onset of stridor and dysphasia, urticarial rash)
- Reflex laryngospasm (in patients with GERD, active upper airway infection (URI), or mechanical irritants in the airway)
- Vocal cord dysfunction (possibly secondary to Arnold-Chiari type II malformation).
- Aspiration of foreign body (sudden onset of stridor, history of choking or cyanosis while eating)
- Large esophageal foreign body (causing tracheal compression)
- Obstructive laryngeal papillomatosis (hoarseness, stridor, aphonia, chronic cough, caused by human papilloma virus)
- Tracheomalacia/laryngomalacia (prematurity, prior tracheostomy)
- Subglottic stenosis (history of endotracheal intubation or tracheostomy)
- Enlarged tonsils or adenoids
- Tonsillitis or peritonsillar abscess

Diagnostic Studies

- Anteroposterior and lateral chest radiographs; symmetric narrowing of the subglottic air shadow on AP film (“church steeple” sign) is characteristic of croup.
- Barium swallow study may be helpful in identifying lesions compressing the trachea.
- Defer radiologic studies if the patient is unstable.
- Definitive diagnosis is obtained by direct laryngoscopy or bronchoscopy.

Subsequent Management

- IV hydration to prevent thickening of tracheal secretions
- Antibiotics are not indicated for croup, but may be appropriate if bacterial superinfection or abscess is suspected.
- The child can usually be extubated in 2–4 days.

Risk Factors

- Most commonly due to croup in infants less than 6 months of age
- May be associated with congenital narrowing of subglottic region
- Edema and inflammation following (usually prolonged) endotracheal intubation

Prevention

No specific prevention for croup. Appropriately sized, uncuffed ETT (approximately the size of the patient's fifth finger) may reduce incidence of postextubation stridor.

Special Considerations

An apneic anesthetic technique without an ETT tube in place provides the best view for the surgeon to make a diagnosis and/or correct the airway obstructing lesion. Due to the short half life of epinephrine, rebound edema may occur. Children should be observed for at least 2–3 hours after treatment with racemic epinephrine.

Further Reading

- Bjornson CJ, Johnson DW. Croup in the paediatric emergency department. *Paediatr Child Health*. 2007;12:473–477.
- Holzman RS. Advances in pediatric anesthesia: implications for otolaryngology. *Ear Nose Throat J*. 1992;71:99–105.

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Chapter 10

Postanesthesia Care Unit

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- Altered Mental Status 240
- Chest Pain 242
- Hypoxia 244
- Myocardial Ischemia 247
- Oliguria/Acute Renal Failure 249
- Postoperative Hypertension 251
- Postoperative Hypotension 253
- Postoperative Nausea and Vomiting 255
- Prolonged Neurologic Impairment after Regional Anesthesia 257
- Respiratory Depression or Failure 259
- Severe Postoperative Pain 261
- Stroke 263

Altered Mental Status

Definition

A clinical spectrum that includes inappropriate or unexpected behaviors, ranging from emergence delirium through delayed awakening.

Presentation

- Many patients exhibit some somnolence, disorientation, and mental sluggishness immediately after emergence.
- Emergence excitation may result in a highly agitated state with wild thrashing that is resistant to treatment.
- Combativeness, confusion, and disorientation may be signs of hypoxia.
- Delayed awakening is said to occur when a patient fails to respond to stimulation within 60 minutes following anesthesia with no obvious underlying cause.

Pathophysiology

Changes in mental status following surgery are generally not the result of organic brain disease. Pain may cause agitation, confusion, and aggressive behavior during emergence. Endotracheal tubes, drains, and catheters, as well as gastric and urinary distention are painful and may cause agitation or combativeness. Confusion and delirium may also be due to metabolic and electrolyte derangements, hypothermia, and poor analgesia.

DIFFERENTIAL DIAGNOSIS

- Hypoxia or hypocarbia
- Hypotension
- Electrolyte derangement (i.e., hyponatremia, hypercalcemia)
- Hypoglycemia
- Hypothermia
- Acidemia
- Stroke
- Seizure
- Infection
- Central cholinergic syndrome

Immediate Management

- Increase FiO_2 to maintain oxygen saturation.
- Ensure that the patient has an adequate respiratory rate and tidal volume.
- Consider drawing an arterial blood gas to measure PaO_2 and PaCO_2 .

Immediate Management (continued)

- Assess the patient's blood pressure and heart rate.
- Verbally reassure and reorient the patient.
- Provide adequate analgesia and anxiolysis if indicated.
- Consider administration of physostigmine 0.5 mg IV if cholinergic syndrome is suspected.
- Restrain patient only if patient or staff safety is at risk.

Diagnostic Studies

- Arterial blood gas analysis
- Fingerstick for blood glucose level
- Plasma electrolyte measurements Toxicology screen
- CT of brain (rule out acute bleed, stroke)

Subsequent Management

- Continue to provide reassurance to the agitated or combative patient.
- Correct all metabolic causes and ensure normothermia.
- If indicated, administer naloxone and/or flumazenil, and ensure adequate neuromuscular reversal for patients with delayed emergence.
- Consider judicious administration of a butyrophenone (e.g., haloperidol 2.5 mg IM) to treat agitation without an obvious underlying cause.
- Consider further neurological consultation and workup (CT scan, MRI, EEG) if mental status does not improve.

Risk Factors

- Age (children and elderly)
- Organic brain dysfunction
- Mental retardation
- History of substance intoxication or withdrawal

Prevention

Ensure adequate pain control in the postoperative period. Maintain hemodynamic and metabolic stability throughout the anesthetic and into the postoperative period. There is no clear strategy for preventing postoperative excitation and delirium in a susceptible patient.

Special Considerations

A high percentage of elderly patients (up to 30–50%) may experience some degree of postoperative confusion, delirium, or cognitive

decline. General anesthesia, combined with the stress of surgery and preexisting cognitive abnormalities may all exacerbate this problem. Postoperative cognitive dysfunction may be seen immediately in the PACU or may occur days after surgery.

Further Reading

Chang YL, Tsai YF, Lin PJ, et al. Factors for postoperative delirium in a cardiovascular intensive care unit. *Am J Crit Care*. 2008 Nov;17(6):567–575.

Chest Pain

Definition

Pain located in the thoracic region, attributable to multiple etiologies, may be cardiac or respiratory in nature, frequently described as heavy or pressing, substernal with possible radiation, and is rarely localized. Intensity may vary with respiration.

Presentation

- Acute presentation of substernal chest pain or pressure, with frequent radiation to arm or jaw, described as squeezing pressure
- Blood pressure changes may also be noted (decreased or elevated).
- May be associated with tachypnea, shortness of breath, dyspnea, cyanosis, or diaphoresis

Pathophysiology

Postoperative chest pain is common and has a varied etiology. Classic symptoms of chest pain and tightness may be due to myocardial ischemia or infarction. Cardiac chest pain may also radiate to the arms, epigastric area, back, shoulders, neck or jaw, and may follow skin dermatomes. Gastroesophageal reflux may also be reported as chest pressure or burning. Pulmonary thromboembolism may produce sharp, pleuritic pain that varies with respiration.

DIFFERENTIAL DIAGNOSIS

- Myocardial ischemia or infarction (MI)
- Cardiac arrhythmia
- Pericarditis
- Pericardial tamponade
- Pneumothorax
- Pulmonary embolism (PE)
- Pneumoperitoneum
- Gastroesophageal reflux disease

- Esophageal spasm or rupture
- Thoracic aneurysm
- Anxiety

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Aggressively treat hypotension or hypertension.
- Provide analgesics (morphine) for relief of pain.
- Consider sublingual nitroglycerin or an IV infusion (start at 0.5 mcg/kg/min).
- Obtain an electrocardiogram and chest X-ray.
- Initiate review of surgical and anesthetic procedures.

Diagnostic Studies

- 12-lead electrocardiogram
- Chest X-ray
- Cardiac enzymes (troponin, creatine phosphokinase (CPK), CPK-MB)

Subsequent Management

- ECG interpretation should take into account the quality of chest pain, patient cardiac history, baseline ECG, and risk index.
- If a PE is suspected, obtain a pulmonary perfusion test, high-resolution CT, or angiography. Anticoagulation may be indicated, but risks of bleeding need to be considered.
- Treat significant pneumothorax (tube thoracostomy) or pericardial tamponade (pericardiocentesis).
- Consider administering a beta-blocker for control of tachycardia and hypertension (labetalol 5 mg IV q 2 min or metoprolol 1–2 mg IV q 5 min).
- Treat hypotension with fluids and/or vasopressors.
- Consider administering aspirin and/or sublingual nitroglycerin if an MI is suspected.
- If an MI is suspected, obtain a cardiology consultation for possible intervention.

Risk Factors

- A cardiac etiology is more likely in the presence of coronary artery disease, peripheral vascular disease, advanced age, male sex, hyperlipidemia, obesity, and diabetes.

Risk Factors (continued)

- PE diagnosis is supported in patients with known deep vein thrombosis (DVT), hypercoagulable states, malignancy, advanced age, high risk (e.g., orthopedic) or long procedures and prolonged bed rest.
- A pneumothorax may occur in almost any setting, but should especially be considered after central line placement and surgical procedures involving the chest, neck, and upper abdomen. Also suspect a pneumothorax in any patient with recent trauma.

Prevention

Appropriate preoperative evaluation, cardiac risk stratification, and medical optimization identifies patients at risk for cardiac events. Appropriate DVT prophylaxis and early ambulation is also useful in reducing the incidence of deep vein thrombosis and PE. The use of ultrasound may decrease the risk of pneumothorax with central line placement.

Special Considerations

The initial evaluation of chest pain should always rule out life-threatening conditions first. Once those causes have been considered and ruled out, other etiologies (e.g., gastroesophageal reflux) may be considered. Abdominal laparoscopic procedures with pneumoperitoneum can often cause referred pain into the chest, and esophageal pathology may also mimic cardiac pain.

Further Reading

Eagle KA, Berger PB, Calkins H, et al. Guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *Anesth Analg.* 2002;94:1052–1064.

Hypoxia**Definition**

Decreased arterial oxygen content in blood as measured by pulse oximetry or arterial blood gas analysis, usually transient as a result of atelectasis and/or alveolar hypoventilation in the early postoperative period.

Presentation

- $SpO_2 < 90\%$ or $pO_2 < 60$ mmHg
- Patient is frequently lethargic and may be uncooperative or agitated.

- Tachycardia and hypertension may be associated with hypoxia.
- Tissue cyanosis may be seen in severe cases.

Pathophysiology

In the patient breathing room air, even mild hypoventilation can result in significant hypoxia. A healthy patient with normal lungs will become hypoxic in the setting of severe hypoventilation. A right-to-left intrapulmonary shunt (i.e., atelectasis) or ventilation/perfusion mismatch may also lead to arterial hypoxemia. Alveolar hypoventilation, from residual anesthetics or residual neuromuscular blocking agents, can also occur.

DIFFERENTIAL DIAGNOSIS

- Hypoventilation due to residual anesthetics
- Hypercarbia
- Atelectasis
- Impaired diffusion (pulmonary edema, pulmonary fibrosis, aspiration)
- Increased oxygen consumption (shivering)
- PE
- Pneumothorax
- Transfusion-related lung injury
- Acute respiratory distress syndrome
- Mainstem bronchial or esophageal intubation
- Carboxyhemoglobin (normal PaO₂ but impaired carrying capacity)
- Methemoglobin (normal PaO₂ but impaired carrying capacity)
- Inadequate FiO₂
- Obstructive sleep apnea

Immediate Management

- Provide supplemental O₂ via nasal cannula or face mask to maintain adequate oxygenation.
- Insert an artificial airway if airway obstruction is present.
- Intubation and mechanical ventilation may be required if the patient does not respond to supplemental O₂.
- Arterial blood gas analysis is useful to determine PaO₂ and PaCO₂.
- Cardiovascular support may be necessary in extreme cases of arterial hypoxia.

Diagnostic Studies

- Chest X-ray
- Arterial blood gas
- Arterial co-oximetry

Subsequent Management

- Continue supplemental oxygen.
- Consider intubation and mechanical ventilation for significant hypoxia or hypercarbia.
- Consider reversing opioids, residual neuromuscular blockade, and benzodiazepines.
- Provide analgesia if splinting is the cause of hypoventilation.
- Insert a chest tube to decompress a pneumothorax.
- Administer diuretics (furosemide 20 mg IV) to treat volume overload causing pulmonary edema.
- Consider initiating an anticoagulant (e.g., heparin) if PE is suspected. Consult the surgical team before administering anticoagulants.

Risk Factors

- Advanced age
- Obesity
- Preexisting pulmonary disease (emphysema, fibrosis, pulmonary hypertension)
- Congestive heart failure
- Increased production of carbon dioxide (i.e., malignant hyperthermia, shivering)

Prevention

Prevention of postoperative hypoxia is largely based on appropriate administration of supplemental oxygen in the immediate postoperative period. Appropriate levels of positive end-expiratory pressure and recruitment maneuvers may help to decrease atelectasis and right-to-left shunt. Care must also be taken to use judicious amounts of sedatives, narcotics, and fluids intraoperatively. Hypoxia may also be prevented by appropriate postoperative respiratory care to prevent and treat atelectasis.

Special Considerations

Diffusion hypoxia can contribute to postoperative hypoxia after a nitrous oxide anesthetic, and may persist for up to approximately 10 minutes, emphasizing the need for supplemental oxygen immediately after emergence and during patient transport.

Further Reading

Lumb AB. *Nunn's Applied Respiratory Physiology*. 6th ed. Philadelphia: Butterworth-Heinemann; 2005.

Myocardial Ischemia

Definition

Myocardial oxygen supply insufficient to meet metabolic requirements. Ischemia can be caused by decreased regional perfusion or increased oxygen demand.

Presentation

- Chest pain or pressure reported by an awake patient
- ST-segment elevation or depression on ECG
- Possible tachycardia and hypertension or profound hypotension
- Arrhythmias, congestive heart failure, acute mitral regurgitation
- Hypoxia, dyspnea
- Dizziness, or syncope

Pathophysiology

Platelet aggregation, vasoconstriction, and thrombus formation at the site of a plaque in a coronary artery that decreases or interrupts blood flow. In patients with ischemic coronary artery disease, a sudden increase in myocardial oxygen demand (tachycardia) or decrease in oxygen supply (hypotension, hypoxia) can precipitate acute myocardial ischemia and/or infarction.

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Ensure adequate ventilation.
- Treat pain with narcotics (morphine IV).
- Aggressive fluid resuscitation and/or vasopressors for hypotension.
- Initiate beta-blockers (metoprolol 1–2 mg IV q 5 min or esmolol IV infusion started at 50 mcg/kg/min to a max of 300 mcg/kg/min) for treatment of tachycardia and hypertension. Titrate to a target heart rate of 60 if possible.
- Administer aspirin after consulting with surgeon to determine risk of bleeding.
- Request a cardiology consultation for a possible interventional procedure.

DIFFERENTIAL DIAGNOSIS

- Coronary vasospasm
- Pericarditis
- Aortic dissection
- PE
- Anxiety

Diagnostic Studies

- 12-lead electrocardiogram
- Cardiac enzymes (troponin, CPK, CPK-MB)
- Complete blood count, metabolic profile
- Arterial blood gas
- Chest X-ray

Subsequent Management

- Continuous monitoring and serial ECGs and cardiac enzymes to assess for ST segment changes, new Q waves, or conduction defects
- Echocardiography for new wall motion defects, ejection fraction, thrombus evaluation
- Beta-blockers (e.g., labetalol or metoprolol IV push or esmolol infusion) for control of heart rate and blood pressure
- Consider addition of nitroglycerin for coronary vasodilatation.
- Cardiology consultation and discussion with surgical team are important for possible interventions, including emergent coronary artery stenting and initiation of anticoagulation and antiplatelet therapy.

Risk Factors

- Known history of coronary artery disease (CAD), previous myocardial infarction, or significant risk factors of CAD
- Major abdominal, thoracic, vascular, or emergency surgeries carry the highest risk of postoperative myocardial infarction.

Prevention

Preoperative optimization of the patient with known or suspected coronary artery disease is essential for prevention of postoperative complications. Avoiding hemodynamic lability is important to ensure adequate myocardial oxygen supply and minimize myocardial demand. With possible exception of angiotensin converting enzyme (ACE) inhibitors and diuretics, all cardiac medications should generally be continued on the day of surgery.

Special Considerations

Interpretation of ECG changes, including ST segment elevation or depression, must be interpreted taking into account a patient's history and cardiac risk. ST segment depression and chest pain in a low-risk patient rarely indicates myocardial ischemia, but rather other, more benign causes such as anxiety, hyperventilation, and hypokalemia, and generally only requires observation in the PACU. High-risk patients with ST segment changes should undergo an immediate evaluation for myocardial ischemia or infarction. While monitoring of leads II and V5 will detect 80% of ischemic events, a 12-lead ECG should always be performed for verification and evaluation.

Further Reading

Badner NH, Knill RL, Brown JE, et al. Myocardial infarction after noncardiac surgery. *Anesthesiology*. 1998;88:572–578.

Feringa HH, Bax JJ, Poldermans D. Perioperative medical management of ischemic heart disease in patients undergoing noncardiac surgery. *Curr Opin Anaesthesiol*. 2007 Jun;20(3):254–260.

Oliguria/Acute Renal Failure

Definition

Urine output less than 0.5 mL/kg/h that may signify loss of kidney function due to prerenal, renal, or postrenal etiologies.

Presentation

- Often associated with a preexisting renal insufficiency that is exacerbated by an intraoperative event.
- Urine output less than 0.5 mL/kg/h.
- May be associated with electrolyte abnormalities (hyperkalemia), acidosis, and rising blood urea nitrogen and creatinine.

Pathophysiology

Prerenal causes of oliguria include hypovolemia due to ongoing bleeding, third-space fluid loss, and inadequate fluid replacement. Preoperative or intraoperative insults, such as radiographic contrast dye, hypotension, or exposure to nephrotoxic medications may result in acute tubular necrosis, particularly in a patient with baseline renal insufficiency. Postrenal causes of oliguria include obstruction of the ureters, surgical injury to the ureter, and obstruction of a urinary catheter.

DIFFERENTIAL DIAGNOSIS

- Hypovolemia
- Low cardiac output

- Abdominal hypertension
- Acute tubular necrosis
- Nephrotoxic agent exposure
- Ureter obstruction
- Mechanical catheter obstruction.

Immediate Management

- Insert a urinary catheter and evaluate for patency.
- Administer a fluid challenge (10–20 mL/kg crystalloid).
- Consider diagnostic administration of a diuretic (furosemide 10–40 mg IV) after ruling out hypovolemia.
- Discontinue administration of nephrotoxic agents.

Diagnostic Studies

- Basic metabolic panel
- Arterial blood gas
- Chest X-ray
- Urine electrolytes

Subsequent Management

- Consider inserting a central venous or pulmonary artery catheter if volume status is unclear.
- Consider starting a dobutamine or norepinephrine infusion for low cardiac output or hypotension to ensure adequate renal perfusion.
- Treat severe metabolic acidosis and hyperkalemia if present.
- Consider hemodialysis for worsening renal failure refractory to treatment.

Risk Factors

- Chronic renal insufficiency
- Left ventricular dysfunction
- Advanced age
- Significant intraoperative blood loss
- Intraoperative hypotension

Prevention

Careful perioperative fluid balance is important in patients with baseline renal insufficiency. Avoidance of nephrotoxic agents and maintenance of adequate renal perfusion may also be useful in preventing postoperative renal failure.

Special Considerations

Aortic cross-clamping, severe hypotension, massive transfusion, possible ureteral injury, and other intraoperative events may predispose to postoperative renal failure. Aggressive early rehydration and maintenance of blood pressure are useful in preventing further renal insult.

Further Reading

Josephs SA, Thakar CV. Perioperative risk assessment, prevention, and treatment of acute kidney injury. *Int Anesthesiol Clin*. 2009 Fall;47(4):89–105.

Noor S, Usmani A. Postoperative renal failure. *Clin Geriatr Med*. 2008 Nov;24(4):721–729

Postoperative Hypertension

251

Definition

Elevated systemic blood pressure, frequently seen with tachycardia.

Presentation

Moderate elevation in systolic and/or diastolic blood pressure in postoperative patients. Pain is often present and is typically the precipitating factor of hypertension, commonly associated with tachycardia.

Pathophysiology

Patients with a history of preexisting hypertension have an exaggerated blood pressure response due to noncompliant vasculature, elevated peripheral vascular tone, and increased endogenous sympathetic nervous system activity. Peripheral arterial and venous constriction is mediated by alpha-adrenergic stimulation causing increased systemic vascular resistance and venous return. Increased contractility and heart rate results from an increase in beta-1 receptor stimulation.

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Ensure adequate ventilation.
- Administer analgesics (morphine 1–2 mg IV) or sedatives (midazolam 1–2 mg) to control pain and anxiety.

Immediate Management (continued)

- Treat acidosis or hypoxemia if present.
- Ensure ability to void urine.
- If hypertension persists, consider other interventions such as intravenous labetalol, esmolol, hydralazine, nicardipine or clevidipine for control.

DIFFERENTIAL DIAGNOSIS

- Preoperative essential hypertension
- Postoperative pain and agitation
- Arterial hypoxemia
- Hypercapnea
- Hypervolemia
- Gastric or bladder distention
- Increased intracranial pressure
- Emergence delirium
- Inappropriately small blood pressure cuff or improperly calibrated arterial line.

Diagnostic Studies

Initially, none indicated. Further workup should be based on clinical evidence of ruling out an underlying etiology (i.e., acidosis, hypercapnea).

Subsequent Management

- Continued assessment and treatment of postoperative pain.
- Resume usual antihypertensive medication as soon as possible.
- Persistent hypertension may require intermittent boluses of a beta blocker (e.g., labetalol), continuous IV infusions (e.g., nicardipine, clevidipine), invasive blood pressure monitoring, and ICU admission.
- Potent vasodilators such as nitroprusside or nitroglycerine are reserved for refractory or life-threatening hypertension.

Risk Factors

- Presence of essential hypertension preoperatively.
- Cessation of antihypertensive medications in the perioperative period.
- Inadequate pain control, either during or after surgery.
- Patients undergoing intracranial procedures.

Prevention

Antihypertensive medications should generally be continued throughout the perioperative period (ACE inhibitors and diuretics

are often held on the morning of surgery) because abrupt withdrawal may precipitate rebound hypertension. Pain should be treated with judicious use of narcotics, regional anesthetic techniques, or other forms of analgesia.

Special Considerations

Postoperative hemodynamic instability in the PACU is somewhat frequent; however, postoperative systemic hypertension and tachycardia are more predictive of an adverse outcome than are hypotension and bradycardia. While the exact etiology of hypertension cannot always be found, it is crucial to treat common postoperative causes (i.e., pain, hypoxia) rapidly, and then use pharmacotherapy to restore hemodynamic stability as quickly as possible.

Further Reading

Varon J, Marik PE. The diagnosis and management of hypertensive crises. *Chest*. 2000;118:214–227.

Postoperative Hypotension

253

Definition

Decreased systemic blood pressure that can cause tissue hypoperfusion and hypoxia.

Presentation

- Decreased arterial blood pressure.
- May be associated with either bradycardia or tachycardia.
- Alteration in mental status may be seen in severe cases.
- Organ system failure may be seen after prolonged hypotension (myocardial infarction, acute renal failure, hepatic ischemia).

Pathophysiology

Systemic hypotension can be categorized as either hypovolemic (decreased preload), cardiogenic (pump failure), or distributive (decreased afterload). Hypotension in the PACU is usually caused by decreased preload and ongoing fluid losses due to third-spacing and blood loss. These factors lead to decreased ventricular filling and cardiac output, sympathetic-mediated tachycardia, increased systemic vascular resistance, and venoconstriction. Neuraxial anesthetic techniques may also lead to a loss of sympathetic tone causing hypotension.

Immediate Management

- Ensure adequate oxygenation and ventilation. Provide supplemental oxygen or ventilatory support if necessary.
- Initiate aggressive resuscitation with IV fluids (crystalloid or colloids). Many patients will often respond to a moderate fluid challenge.
- Support blood pressure with phenylephrine, ephedrine, or epinephrine boluses. Consider beginning an infusion of phenylephrine (0.5–1 mcg/kg/min) or epinephrine (0.03–0.05 mcg/kg/min) in the setting of refractory hypotension.
- Consider placing the patient in Trendelenberg position (benefits controversial).

DIFFERENTIAL DIAGNOSIS

- Hypovolemia
- Hemorrhage
- Myocardial ischemia/infarction
- Cardiac arrhythmia
- Cardiac tamponade
- Tension pneumothorax
- Pulmonary embolus
- Anaphylaxis
- Spinal shock
- Sepsis
- Drug-induced (beta blocker, calcium channel blocker)

Diagnostic Studies

- Chest X-Ray if clinical suspicion of pneumothorax.
- Complete blood count
- Arterial blood gas analysis to determine hematocrit and acid-base status (hematocrit may be inaccurate during acute blood loss).
- 12-lead ECG and/or echocardiogram may be useful if a cardiogenic origin is suspected.

Subsequent Management

Continue supportive care with fluids, blood products, and vasopressors as necessary. Arterial and/or central venous pressure monitoring may be useful for continued monitoring and infusion of fluids and drugs. Determination and treatment of primary etiology is essential (i.e., myocardial ischemia or pneumothorax).

Risk Factors

- Inadequate replacement of fluids from ongoing fluid losses (bowel preparation, gastrointestinal losses) or significant bleeding.
- Pre-existing hypertension and cardiac disease.
- Neuraxial anesthetic techniques are often associated with hypotension.

Prevention

Postoperative hypotension is most often prevented by careful intraoperative volume replacement. Blood and fluid losses are often underestimated in many surgical procedures. A fluid load prior to neuraxial anesthetic techniques may also prevent hypotension. Critically ill patients rely on increased sympathetic tone to maintain normotension and may be more sensitive to anesthetic agents.

Special Considerations

While relative hypovolemia is usually the reason for systemic hypotension, shock resulting from more catastrophic causes must be considered. Significant hemorrhage can develop rapidly in the PACU, often without obvious signs, and must be suspected in any patient with unexplained hypotension. Tension pneumothorax, pulmonary embolus, or pericardial tamponade may occur suddenly and may be lethal if not immediately treated. Finally, anaphylaxis is often overlooked as a cause of hypotension and should always be considered.

Further Reading

Barash PG, Cullen, BF, Stoelting RK, Cahalan MK, Stock ME (eds.). *Clinical Anesthesia*. 6th ed. Philadelphia: Lippincott, Williams & Wilkins; 2009:1452–1456.

Postoperative Nausea and Vomiting

Definition

Significant nausea and/or emesis experienced either in the PACU or after discharge following surgery. Without prophylaxis, 10–80% of patients may experience postoperative nausea and vomiting (PONV) after a volatile anesthetic technique.

Presentation

- Range of symptoms from mild nausea to retching and vomiting in the postoperative period.
- Associated with increased intra-abdominal pressure, and may risk abdominal or inguinal suture lines.
- Increased risk of gastric aspiration, especially when airway protective reflexes are not completely intact.
- Increased sympathetic nervous system response, producing hypertension and tachycardia.

Pathophysiology

PONV is the result of multiple of perioperative factors such as starvation, autonomic imbalance, pain, and the anesthetic effects on chemotactic centers. The emetic centers are located in the lateral reticular formation of the medulla. Pharmacological treatment aims to target specific receptors including dopaminergic, histaminergic, cholinergic, substance P, and serotonergic.

Immediate Management

- Increase FiO_2 to ensure adequate oxygenation. Exclude potentially serious causes of PONV (i.e., impending shock, increased intracranial pressure).
- Ensure adequate hydration with intravenous crystalloids.
- Administer medication for rescue (i.e., ondansetron 4 mg IV). In general, do not repeat the same class of agents used for prophylaxis.

DIFFERENTIAL DIAGNOSIS

- Anesthetics (narcotic, inhalational agents)
- Pain
- Anxiety
- Hypoxia
- Hypotension
- Hypoglycemia
- Increased intracranial pressure
- Gastric bleeding
- Bowel obstruction

Diagnostic Studies

- Clinical diagnosis
- Perform additional diagnostic studies as clinically indicated to rule out other more severe etiologies.

Subsequent Management

- Continue to treat severe PONV with multimodal therapy.
- Phenothiazines (phenergan, compazine) are good second-line agents.
- Droperidol (0.625 mg) is highly effective but carries a “black-box” warning for QTc prolongation.
- Propofol (10–20 mg) may be also be beneficial for refractory nausea.

Risk Factors

- Female sex
- Nonsmoking status
- Prior history of motion sickness or PONV
- Laparoscopic surgery
- Strabismus and middle ear surgery
- Opioid-based and nitrous oxide anesthetic

Prevention

Prophylaxis of PONV is often more effective than rescue therapy. Patients who have the highest risk of developing PONV should ideally receive triple agent prophylaxis, Prophylaxis in patients at very low risk may not be indicated.

Special Considerations

Patients with a history of protracted PONV, despite adequate prophylactic therapy, may benefit from an alteration in anesthetic technique in addition to the usual prophylactic treatment. Regional techniques will reduce the incidence of PONV significantly. When a general anesthetic is required, limited use of narcotics, volatile anesthetics and nitrous oxide may be possible with a total intravenous anesthetic technique.

Further Reading

Gan TJ, Meyer TA, Apfel CC, et al. Society for ambulatory anesthesia guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2007;105:1615–1628

Prolonged Neurologic Impairment after Regional Anesthesia

Definition

Prolonged paralysis or sensory deficit after a neuraxial anesthesia technique, which may have resulted from the anesthetic, its contaminants,

surgical procedure, positioning, or exacerbation of a preexisting condition.

Presentation

- Prolonged motor and sensory blockade greater than expected for local anesthetic injected into the spinal or epidural space
- Sensory deficits such as numbness and paresthesias

Pathophysiology

The most serious neurological complication of neuraxial anesthesia is paraplegia due to ischemia, infection, or drug toxicity. Spinal cord ischemia or infarction may be due to arterial hypotension or compression of the cord by an expanding epidural hematoma or abscess. Neuro toxicity can also occur as a result of either intrathecal local anesthetics or preservatives.

DIFFERENTIAL DIAGNOSIS

- Spinal/epidural hematoma
- Spinal/epidural abscess (not acute)
- Neuropathy due to positioning (i.e., lithotomy position)
- Toxicity of anesthetic or additive in the intrathecal space
- Cauda equina syndrome
- Longer than expected effect of anesthetic agents

Immediate Management

- Neurological examination to evaluate extent of residual effect.
- Emergency MRI of the spinal cord to evaluate for hematoma or other pathology.
- Emergency neurosurgical consultation

Diagnostic Studies

Emergency MRI of spinal cord after an intrathecal or epidural block.

Subsequent Management

- Perform emergency surgical decompression if there is evidence of hematoma or abscess.
- Other than surgical decompression, further treatment of significant neurologic injury is largely supportive.
- Most transient paresthesias resolve over about 6 months.
- Numerous case reports show unexplained prolonged block with no known nerve damage (i.e., clonidine patch prolonging blockade).

Risk Factors

- Leukemia, blood dyscrasias, thrombocytopenia
- Antiplatelet therapy or anticoagulation
- Traumatic or difficult needle placement

Prevention

While it is impossible to prevent all neurological complications of spinal and epidural anesthetics, proper patient selection is important in limiting the potential of catastrophic complications. Avoiding regional anesthesia in patients who are treated with antiplatelet agents (aspirin alone is usually acceptable) and therapeutic anticoagulation, as well as patients with ongoing significant infections or those who are severely immunocompromised is recommended.

Special Considerations

An association between paresthesias and radiating pain on injection of local anesthetics has been suggested. While it appears safe to redirect a needle if a paresthesia develops during placement, it may be safer to stop the procedure or relocate the needle if a paresthesia develops during local anesthetic injection.

Further Reading

Horlocker TT, Wedel DJ, Benzon H, et al. Regional anesthesia in the anticoagulated patient: defining the risks. *Reg Anesth Pain Med.* 2003 May-Jun;28(3):172–197

Respiratory Depression or Failure

Definition

Impaired ventilation, oxygenation, and airway maintenance due to a variety of mechanical, hemodynamic, and pharmacologic factors seen in the postoperative period.

Presentation

- Airway obstruction and loss of pharyngeal muscle tone
- Decreased minute ventilation and possible hypoxia
- Excessive sedation and somnolence
- Residual neuromuscular blockade with muscle weakness

Pathophysiology

The residual effects of intravenous and inhalational anesthetics blunt the normal ventilatory responses to both hypercarbia and hypoxemia.

Benzodiazepines and narcotics act synergistically to further decrease the ventilatory drive, and can cause upper airway obstruction. Residual neuromuscular blockade may cause further compromise of the upper airway. While pharyngeal muscles normally contract with the diaphragm to open the airway during negative inspiratory pressure, this function is often decreased in the sedated patient, which may lead to airway obstruction and respiratory depression.

DIFFERENTIAL DIAGNOSIS

- Residual anesthetics (benzodiazepines, opioids, inhaled anesthetics)
- Residual neuromuscular blockers
- Upper airway obstruction, edema, or hematoma
- Laryngospasm
- Bronchospasm
- Pulmonary edema
- PE
- Pneumothorax
- Obstructive Sleep Apnea

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Provide jaw thrust, artificial oral, nasal, or laryngeal mask airway to provide a patent airway.
- Intubate the trachea in the setting of respiratory failure or complete airway obstruction that does not immediately resolve with the above interventions.

Diagnostic Studies

- Chest X-ray
- Arterial blood gas
- Assessment for the presence of residual neuromuscular blockade

Subsequent Management

- Reverse residual opioids or benzodiazepines with judicious use of the appropriate antagonist (i.e., naloxone or flumazenil).
- Reverse residual neuromuscular blocking agents.
- Administer beta-2 agonists for treatment of bronchospasm or reactive airway disease.
- Initiate continuous positive airway pressure (CPAP).
- Mechanical ventilation

Risk Factors

- Advanced age
- Obesity
- Chronic obstructive pulmonary disease (COPD)
- Severe asthma
- Obstructive sleep apnea

Prevention

The synergistic effects of respiratory depressants (e.g., residual anesthetic agents, opioids) are a common cause of hypoventilation. Titrate sedatives and opioids carefully, especially in patients who are prone to postoperative respiratory failure (e.g., those with obstructive sleep apnea and morbid obesity)

Special Considerations

Hypoventilation and airway obstruction may be seen after the removal of noxious stimuli (i.e., extubation or infiltration of a wound with local anesthetic solution). Slow titration of naloxone (40 mcg incremental doses) can reverse opioid-induced respiratory depression without affecting analgesia. An overdose of naloxone will result in severe pain, tachycardia, hypertension, and possibly myocardial ischemia. Consider the possibility of a hematoma obstructing the airway in a patient who has just undergone neck surgery.

Further Reading

Arozullah AM, Daley J, Henderdon, WG, et al. Multifactorial risk index for predicting postoperative respiratory failure in men after major noncardiac surgery. *Ann Surgery*. 2000;232(2):242–253

Severe Postoperative Pain

Definition

Postoperative pain is a complex physiological response to tissue trauma, visceral distention, and ongoing disease processes. The aim of treatment for postoperative pain is to provide subjective comfort in addition to inhibiting trauma-induced nociceptive signals in order to blunt autonomic and somatic reflex responses to pain.

Presentation

- Constant pain near the surgical site, and aching in nature
- Frequent acute exacerbation of pain with normal postoperative activities such as coughing, ambulating, dressing changes

- Usually is a self-limiting condition with progressive improvement over a relatively short period of time
- Hypertension, tachycardia, myocardial ischemia, increased oxygen consumption, and increased sympathetic tone may be present.

Pathophysiology

Nociception involves the recognition and transmission of noxious stimuli via afferent sensory nerves through the dorsal horn of the spinal cord to the contralateral cortex via the thalamus. Modulation of this signal may occur at any level of the pathway with medications such as opioids, nonsteroidal anti-inflammatory drugs (NSAIDs), cyclooxygenase-2 inhibitors (COX-2), and local anesthetics.

Immediate Management

- Evaluate the quality and level of pain using a visual analog scale or similar method.
- If the pain is severe and refractory to appropriate management, reevaluate the patient and notify the surgical team.
- For severe postoperative pain, opioids are the mainstay of therapy.
- Administer incremental doses of opioids (e.g., morphine or hydromorphone) until patient appears comfortable. A pain score of 3–4 is usually acceptable.
- Consider patient-controlled analgesia (PCA) once pain is at an acceptable level and patient is comfortable.
- Provide supplemental oxygen to ensure adequate oxygenation should hypoventilation occur following administration of opioids.

DIFFERENTIAL DIAGNOSIS

- Inadequate analgesic regimen (inadequate or infrequent dosing)
- Wound complications (infection, hematoma, nerve injury)
- Chronic pain disorder
- Drug-seeking behavior

Diagnostic Studies

Clinical diagnosis

Subsequent Management

- Intrathecal or epidural administration of local anesthetics and opioids may provide superior pain relief in many abdominal or lower-extremity orthopedic procedures.

- NSAIDs and COX-2 inhibitors have no hemodynamic effects and do not cause respiratory depression, but will decrease levels of inflammatory mediators at the site of tissue injury. COX-2 agents will not affect platelet function. At the present time, celecoxib is the only COX-2 inhibitor available in the United States.
- Consider switching opioids if pain control is inadequate (i.e., morphine to hydromorphone).
- Transition to oral route as soon as feasible with adequate control of pain.

Risk Factors

- Male sex
- Preoperative pain
- Prior history of poor pain management
- Coexisting medical conditions (substance abuse, withdrawal, anxiety)
- Opioid addiction

Prevention

Preemptive analgesic therapy may significantly help in controlling postoperative pain in many patients. Local and regional anesthetic techniques can be used to decrease the intensity and duration of postoperative pain.

Special Considerations

In addition to patient comfort, adequate control of pain may have a further benefit in improving clinical outcome by preventing myocardial ischemia or infarction, impaired wound healing, atelectasis, and thromboembolic events. Severity of postoperative pain has been shown to predict persistent postoperative pain.

Further Reading

Caplan RA, Carr DB, Connis RT. Practice guidelines for acute pain management in the perioperative setting. *Anesthesiology*. 2004;100:1573–1581.

Stroke

Definition

Development of a new acute focal neurological deficit due to either an ischemic or embolic event in the brain, diagnosed either immediately after emergence or early in the postoperative period.

Presentation

- Weakness or paralysis in one or more extremities
- Cranial nerve deficits and/or dysarthria
- Severe confusion and altered mental status
- Blurred vision or diplopia

Pathophysiology

A stroke may occur as the result of an occlusion of a cerebral artery and subsequent infarct of brain tissue due to either profound hypotension, a thrombotic event, or an embolism (e.g., thrombus). Embolic strokes may be of cardiac origin. Hemorrhagic strokes due to aneurysm or arteriovenous malformation are also possible, but far less common in the perioperative setting.

DIFFERENTIAL DIAGNOSIS

- Thrombo-embolic stroke
- Ischemic stroke
- Hemorrhagic stroke
- Intracerebral hemorrhage
- Gas embolism (through a patent foramen ovale)
- Seizure

Immediate Management

- Ensure adequate oxygenation and ventilation.
- Emergency CT of brain without contrast.
- Ensure hemodynamic stability, using vasoactive drug infusions as needed for tight hemodynamic control.
- Emergency neurology consultation.

Diagnostic Studies

- Clinical diagnosis
- CT of brain to rule out bleeding
- Possible MRI of brain

Subsequent Management

- Consider insertion of an intra-arterial catheter to facilitate continuous hemodynamic monitoring.
- Consider initiating tissue plasminogen activator (t-PA) or antiplatelet therapy if there is no surgical contraindication
- Request a neurosurgical or neurology consultation for possible endovascular intervention

Risk Factors

- Recent stroke
- Transient ischemic attack (TIA) or amaurosis fugax
- Cardiac, carotid, or neurosurgical procedure
- Atrial fibrillation
- Significant hypotension

Prevention

Preoperative screening for significant carotid disease or risk of cardiogenic embolus may be of benefit. Careful surgical technique during cardiac and carotid surgery is also crucial to prevent embolism of arterial plaques, thrombus, or air entry. Avoidance of hypotension in at-risk patients is also important to reduce the risk of stroke due to hypoperfusion and ischemia.

Special Considerations

Many patients exhibit slurred speech or behavior that under other circumstances might indicate a primary neurologic event while awakening after a potent volatile anesthetic. Close observation is necessary to assure that patients return to their expected level of functioning prior to discharge from PACU.

Further Reading

Selim M. Perioperative stroke. *N Engl J Med*. 2005 Feb;356:706–713.

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Chapter 11

Procedures

Ramachandran Ramani and Ala Haddadin

- Anesthetic Implications of Pacemakers 268
- Blind Nasal Intubation 270
- Bronchial Blocker Placement 271
- Cricothyroidotomy 273
- Double Lumen Endotracheal Tube (DLETT) 276
- Femoral Vein Catheter 278
- Complications 279
- Fiberoptic Intubation 279
- Intubating Laryngeal Mask Airway 282
- Retrograde Intubation 284
- Transcutaneous Pacing 286
- Transport of a Critically Ill Patient 288
- Transvenous Pacing 290
- Ultrasound-Guided Central Venous Access 292

Anesthetic Implications of Pacemakers

Artificial pacing is generally indicated for the treatment of symptomatic bradycardia of any origin. The two major indications for permanent pacing are failure of impulse formation, and failure of cardiac conduction.

The complete pacemaker code contains 5 letters, of which only the first 3 are commonly used. The first letter represents the cardiac chamber paced (A for atrium, V for ventricle, D for dual). The second letter represents the chamber sensed (A,V,D and O for none). The third letter represents the mode of the pacemaker, also described as the response of the pacemaker to the sensed chamber (I for inhibited, T for triggered, D for dual and O for none). The fourth letter indicates programmability and the fifth letter indicates anti-dysrhythmia functions.

Table 11.1 Generic Pacemaker Code (NBS) NASPE (now known as the Heart Rhythm Society)/BPEG Revised (2002)

Position I	Position II	Position III	Position IV	Position V
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	I = inhibited	R = rate modulation	A = atrium
V = ventricle	V = ventricle	T = triggered		V = ventricle
D = dual (A + V)	D = dual (A + V)	D = dual (T + I)		D = dual (A + V)

Source: Reprinted with permission from Bernstein AD, et al. The Revised NASPE/BPEG Generic Code for antibradycardia, adaptive-rate, and multisite pacing. *PACE* 2002;25:260-264.

Modern pacemakers are programmable into one of three modes of pacing: asynchronous, single-chamber demand, and dual-chamber atrioventricular (AV) sequential demand.

A *demand pacemaker* discharges only when there is no intrinsic electrical activity. The primary advantage of demand pacemakers is that they will not interfere with the intrinsic rhythm when the ventricular rate is adequate. Extrinsic electrical activity (e.g., electrocautery) may inhibit this pacemaker even when no intrinsic rhythm is present.

An *asynchronous pacemaker* discharges continuously, regardless of the heart's intrinsic rhythm. This mode is not generally used outside the operating room.

No special monitoring or anesthetic technique is required for a patient with a pacemaker, but there are some important points to remember:

1. Electrocardiographic monitoring of a patient must include the ability to detect pacing discharges.
2. Ensure that paced electrical activity is converted to mechanical systole. Mechanical systole is best evaluated by pulse oximetry, plethysmography, or the arterial pressure waveform.
3. There is limited experience with intraoperative biventricular (BiV) pacing in the perioperative period. Loss of ventricular pacing for any reason will cause an immediate drop in cardiac output. With the exception of transesophageal echocardiography (TEE), no other intraoperative monitor has been shown to detect loss of BiV pacing behavior.
4. Appropriate equipment must be on hand to provide backup pacing or defibrillation (or both) if needed.
5. *A monopolar electrosurgical unit (ESU) is more problematic than a bipolar ESU because the current travels through the body to the return electrode.* Pacemakers can be inhibited by ESU current, which the pacemaker may interpret as QRS complexes. The generator may detect significant electromagnetic interference (EMI) and begin pacing asynchronously at a programmed lower rate (*noise reversion mode pacing*).
6. Placing a magnet over the generator might prevent aberrant pacemaker behavior, but it might also allow reprogramming of an older (pre-1990) generator. More recently manufactured generators are relatively immune to spurious reprogramming.
7. If a monopolar ESU is to be used, place the electrosurgical current-return pad so that electricity does not cross the generator-heart circuit.

If the pacemaker malfunctions:

- Be prepared to begin temporary transthoracic (transcutaneous), transvenous, or transesophageal pacing if necessary.
- Sympathomimetic drugs may be useful if the pacemaker malfunctions because they decrease depolarization threshold or increase chronotropicity (or both). Consider epinephrine or dopamine. Isoproterenol is not widely used because it may cause hypotension. Antimuscarinic drugs may be also used as long as the block is not below the bundle of His. Anticholinergics do not affect the heart rate in heart transplant patients because the transplanted heart is not innervated.
- Rule out myocardial ischemia. Myocardial ischemia can substantially increase the energy required for ventricular capture.
- Correct acid-base and electrolyte disturbances.
- Check antiarrhythmic drug levels (e.g., amiodarone or digoxin).

Further Reading

- Rozner MA. The patient with a cardiac pacemaker or implanted defibrillator and management during anaesthesia. *Curr Opin Anaesthesiol*. 2007 Jun;20(3):261–268.
- Stone ME, Apinis A. Current perioperative management of the patient with a cardiac rhythm management device. *Semin Cardiothorac Vasc Anesth*. 2009 Mar;13(1):31–43.

Blind Nasal Intubation

Nasal intubation can be carried out under direct vision (e.g., with a laryngoscope and Magill's forceps) or it can be performed as a blind technique.

Indication

Blind nasal intubation can be done when other techniques of intubation under vision fail.

Contraindications

Maxillofacial fracture

Skull base fracture with or without cerebrospinal fluid leak

Equipment Checklist

The only equipment necessary for blind nasal intubation is a soft, flexible endotracheal tube. A nasotracheal tube is 2–4 cm longer than that required for an orotracheal tube.

Procedure

- This technique is only practical if the patient is breathing spontaneously.
- Position the patient supine and in the sniffing position, with slight flexion of cervical joint and extension of the atlantooccipital joint.
- Spray the nasal passages with a vasoconstrictor (e.g., phenylephrine, oxymetazoline, or cocaine) to decrease the risk of bleeding.
- Inject 2 mL lidocaine 2% through a 25 g needle placed through the cricothyroid membrane into the trachea to anesthetize the tracheal mucosa.
- Consider bilateral superior laryngeal block to anesthetize the larynx above the glottis (may not be necessary in anesthetized patients).
- Lubricate a 7.0 or 7.5 mm cuffed ETT with lidocaine jelly and pass it through the nostril with the bevel of the tube facing laterally (prevents trauma to the nasal turbinates).
- Gently pass the ETT into the nasopharynx and then further down into the glottis.

- When the ETT has passed 15 cm from the external nares, the distal end of the nasotracheal tube will be just above the glottis.
- With the mouth closed, spontaneous ventilation will be audible at the proximal end of the nasotracheal tube.
- Gently pull the mandible forward and close the mouth to pull the epiglottis away from the path of the nasotracheal tube.
- With gentle side to side manipulation of the larynx with the left hand and the nasotracheal tube with the right hand, at the point where breath sounds are heard with maximum intensity, **gently** advance the nasotracheal tube into the trachea during inspiration.
- Entry of the tube into the trachea may cause coughing or breath-holding.
- ETT position is confirmed with capnography and auscultation.

Complications

- Trauma to the mucosa with the nasotracheal tube passing into a submucosal pocket. If resistance is encountered, gently pull the tube back and redirect it.
- Bleeding from the nares is not uncommon following nasotracheal intubation. This complication is less likely if the nasal passages are sprayed with a topical vasoconstrictor before attempting a nasotracheal intubation.
- Respiratory tract infection is more common after nasotracheal intubation in comparison to oral intubation.
- Prolonged nasotracheal intubation can cause nasal sinus infection.

Special Considerations

- With the availability of intubating devices such as the fiberoptic bronchoscope, blind nasal intubation technique is very rarely performed but may be invaluable when more advanced tools are unavailable.
- Always use a soft, well-lubricated tube.
- Overextension of the neck directs the tube anterior to the larynx, impinging on the vallecula, and extreme flexion directs the tube towards the esophagus.

Bronchial Blocker Placement

Definition

A device that permits one-lung ventilation (OLV) by selective occlusion of a mainstem bronchus.

Indications

- Inability to insert a double-lumen ETT
- OLV in a patient with a known difficult airway
- Selective lobar blockade

Contraindications

- Lung isolation (e.g., hemoptysis, empyema)
- Inability to use fiberoptic bronchoscope (these devices must be placed under direct vision)
- Presence of a bronchopleural fistula

Equipment Checklist

- Bronchial blocker device (Arndt® wire guided, tip-deflecting, or other type) or enclosed bronchial blocker (Torque Control Blocker Univent®; Vitaid, Lewinston, NY)
- Fiberoptic bronchoscope
- Lubricant

Preparation

- Discuss use of a bronchial blocker vs. double lumen endotracheal tube with surgical team.
- Ensure that the ETT is large enough to permit passage of both a fiberoptic bronchoscope and a bronchial blocker (Internal diameter 7.5 mm in most cases.).

Technique

- Thoroughly lubricate the device prior to insertion into the ETT.
- Pass the fiberoptic bronchoscope through the ETT.
- Identify the bronchus in which the blocker is to be placed.
- Pass the bronchial blocker, manipulating it such that the entire cuff is placed within the bronchus to be occluded.
- Inflate the cuff with the volume of air recommended by the manufacturer.
- Open the lumen of the blocker to room air to deflate the lung.

Complications

- Rupture of the bronchus with the cuff, causing a pneumothorax or bronchopleural fistula
- The inflated balloon can migrate proximally and occlude the trachea (e.g., inability to ventilate, unexplained hypoxia)
- Slow deflation of the occluded lung. (Deflate the cuff, allow both lungs to deflate, then inflate the cuff and re-expand the ventilated lung.)
- Malposition or dislodgement of the blocker. (Reposition under direct vision.)

Special Considerations

- Bronchial blockers do not allow for suctioning of the deflated lung.
- Occasionally, secretions, blood, or pus occlude the small lumen of the bronchial blocker, preventing the application of continuous positive airway pressure (CPAP). This can be managed by injecting saline.
- Bronchial blockers are more easily used when collapse of the left lung is indicated. The takeoff of the right mainstem bronchus is variable and the right upper lobe may even originate in the trachea, making controlled collapse of the right lung difficult or impossible.

Further Reading

Campos JH. Update on selective lobar blockade during pulmonary resections. *Curr Opin Anaesthesiol.* 2009 Feb;22(1):18–22.

Slinger PD, Campos JH. Anesthesia for thoracic surgery. In: Miller RD (ed.). *Miller's Anesthesia.* Philadelphia: Churchill Livingstone; 2010:1833–1841.

Cricothyroidotomy

Indications

- Cricothyroidotomy is the surgical airway access of choice in an airway emergency.
- This procedure should only be performed by personnel who have received proper training. The authors recommend that personnel who plan to perform surgical cricothyroidotomy receive formal training in this technique (e.g., an Advanced Trauma Life Support course).

Contraindications

- Distorted anatomy
- Transected airway
- Laryngeal injury
- Surgical cricothyroidotomy is not recommended in children younger than 12 years.

Equipment Checklist

- Many standard commercial cricothyroidotomy kits are available and contain a number 11 blade, tracheal hook, dilator and 6 mm endotracheal tube.
- Needle cricothyroidotomy: 14 g intravenous catheter, 10 cc syringe, jet ventilator system (see Figure 11.2)

Procedure

Anatomical landmarks: The cricothyroid membrane is one finger-breadth below the thyroid cartilage. The larynx can be immobilized with the left thumb and middle finger, and with the index finger, thyroid cartilage and the cricothyroid membrane is identified.

Standard Surgical Cricothyroidotomy

- Clean and drape the anterior aspect of neck (if time permits).
- Palpate the larynx and identify the cricothyroid membrane.
- Stabilize the larynx with the thumb and index finger and incise the skin and subcutaneous tissue over the cricothyroid membrane.
- Again palpate with the index finger and identify the cricothyroid membrane. Make a horizontal incision on the cricothyroid membrane.
- Insert the tracheal hook along the superior edge of the incision and retract the trachea in the upward, cephalad, and anterior direction.
- Insert the tracheal dilator and dilate the trachea in a superior-inferior direction.
- Keep the tracheal dilator in place and pass the cricothyroidotomy tube. Remove the dilator and confirm the position of the tube by ventilating the patient.

Percutaneous Cricothyroidotomy

- Clean and drape the anterior aspect of the neck.
- Immobilize the trachea with the thumb and middle finger and feel the cricothyroid membrane with the index finger.
- Make a transverse incision over the cricothyroid membrane—first through the skin and subcutaneous tissue, and then a small incision through the cricothyroid membrane.
- Attach an 18 g needle to a syringe filled with saline and insert it through the cricothyroid membrane into the trachea. Aspiration of air confirms that the needle is in the trachea.
- Remove the syringe and pass a guide wire through the needle.
- Ensure that the wire is directed inferiorly.
- Pass the dilator over the guide wire to widen the tracheal opening.
- Pass a number 6 tracheostomy tube over the dilator and remove the dilator.
- Confirm the position of the tracheostomy tube by ventilating the patient.

Needle Cricothyroidotomy

This is a lifesaving procedure in any patient in a “cannot intubate, cannot ventilate” situation. This is also the preferred technique if a cricothyroidotomy must be placed in children less than 12 years of age.



Figure 11.1 A 14 g needle with catheter (with a 10 mL syringe filled with saline) is inserted into the trachea through the cricothyroid membrane.

- Palpate the cricothyroid membrane.
- Connect a 14 g intravenous catheter to a 10 mL syringe filled with saline.
- Insert the catheter into the trachea through the cricothyroid membrane (See Figure 11.1).
- Aspiration of air confirms the position of the needle in the trachea.
- Tilt the needle to a 45° angle inferiorly, and thread the catheter into the trachea.
- Connect the catheter to a jet ventilator (the Luer lock connector at the end of the jet ventilator connects to the 14 g cannula). (Figure 11.2)
- When jet ventilation is being used, discontinue O₂ insufflation as soon as the patient's chest begins to rise.

Complications

- Bleeding from the cricothyroid artery or anterior thyroid vein
- Barotrauma or pneumothorax (jet ventilation)
- Subcutaneous emphysema (high-pressure air forced into the subcutaneous space)
- Injury to the posterior wall of the trachea

Special Considerations

- In a “cannot ventilate, cannot intubate” situation, cricothyroidotomy provides a definitive airway more quickly than a tracheostomy.
- Percutaneous cricothyroidotomy requires prior experience.

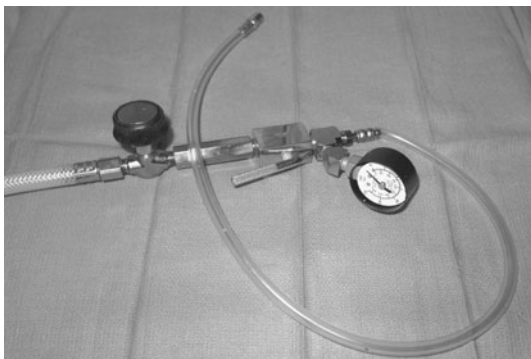


Figure 11.2 Luer lock connector at the end of the jet ventilator connects to the 14 g cannula.

Double Lumen Endotracheal Tube (DLETT)

276

Indications

- Lung isolation: Pulmonary hemorrhage, unilateral lung lavage, purulent bronchial secretions.
- Control of ventilation: Giant unilateral cyst or bulla, independent lung ventilation.
- Elective lung surgery, thoracoscopy, bronchopleural fistula, nonpulmonary thoracic surgery.
- Surgery on the esophagus, thoracic spine, descending thoracic aorta, etc.

Contraindications

Abnormality in the tracheobronchial tree (e.g., high bifurcation of the trachea, early takeoff of the right upper lobe bronchus).

Equipment Checklist

- Appropriate size double lumen tubes:
 - 4 sizes are available: 35 Fr, 37 Fr, 39 Fr, 41 Fr
 - 39 Fr is preferred in male patients
 - 35 Fr is preferred in female patients
- Y connectors, Kelly clamps
- Fiberoptic bronchoscope (A 3.6 mm or 4.9 mm pediatric bronchoscope will pass through the bronchial lumen.)
- Confirm that the bronchoscope is lubricated and that it will pass through the double lumen tube.
- Suction catheters

Procedure

Insertion of a left side double lumen tube will be described here, since this tube can be used for both left- and right-sided procedures.

Assess the patient's airway before attempting intubation with a double lumen endotracheal tube. In patients who have a difficult airway, consider alternative techniques (e.g., single lumen ETT with bronchial blocker).

- Preoxygenate the patient prior to induction of anesthesia.
- Induce general anesthesia.
- Visualize the vocal cords with a laryngoscope.
- Pass a lubricated DL tube with the stylet in place and the curvature of the tube facing anteriorly.
- Remove the stylet after the bronchial cuff passes through the vocal cords. Rotate the tube 90° to the left (this turns the bronchial orifice towards the left side).
- Advance the DL tube until slight resistance is encountered. In an adult of average height (170–180 cm) the 29 cm mark on the tube should be at the incisors.
- Connect the DL tube to the breathing circuit using the Y connectors. Inflate the tracheal cuff, ventilate the lungs, and check for air entry. Bilateral air entry confirms that the tracheal lumen is positioned correctly.
- Inflate the bronchial cuff with 2 mL of air.
- Clamp the bronchial lumen and ventilate through the tracheal lumen—air entry should be heard only on the right side (if the bronchial cuff is in the trachea, it will not be possible to ventilate the patient through the tracheal lumen).
- Occlude the tracheal lumen and ventilate the bronchial lumen. Air entry should be heard only on the left side.
- Pass the bronchoscope through the tracheal lumen. The bronchoscope should emerge above the carina and the top of the bronchial cuff (blue color) should be barely visible.
- If necessary, deflate the bronchial cuff and gently advance the tube so that the bronchial lumen is completely within the left bronchus. The entry to the left upper lobe bronchus should be visible when the bronchoscope is passed through the bronchial lumen.

Complications

- Misplacement of the DL tube. Listen to breath sounds and confirm placement with the fiberoptic bronchoscope.
- Because of its large size and length, trauma to the larynx and trachea is possible.

Special Considerations

- Choose the correct size DL tube and fiberoptic bronchoscope.
- Check the position of the DL tube after any change in position, with a bronchoscope.

Further Reading

Campos JH. Lung isolation techniques. *Anesthesiol Clin North America*. 2001 Sep;19(3):455–474.

Femoral Vein Catheter

Vascular access through the femoral vein minimizes the risk of complications associated with other sites, including pneumothorax, hemothorax, arrhythmias, thoracic duct laceration, and damage to the phrenic, recurrent laryngeal, and vagus nerves. Another benefit of the femoral route is that a radiograph is not necessary to verify proper position.

Indications

- Emergency venous access for administration of drugs or fluids
- Difficult peripheral venous access
- Patient who will not tolerate the supine or Trendelenburg position
- Introduction of a transvenous pacemaker

Contraindications

- Presence of an IVC filter
- Relative contraindications:
 - Infection, burn, or skin damage at puncture site
 - Trauma to ipsilateral groin or lower extremity
 - Suspected proximal vascular injury, especially of inferior vena cava (IVC)

Equipment Checklist

- Central venous catheter kit
- Anatomy and patient positioning
- The femoral vein is punctured in the femoral triangle (inferior to the inguinal ligament, lateral to the adductor longus, and medial to the sartorius muscles), where it lies medial to the femoral artery.

Technique

- **Before attempting this procedure, verify that the patient does not have an IVC filter.**
- If the catheter is to be placed into the right femoral vein, use the ipsilateral hand to hold the needle and syringe. Palpate the femoral artery with the left hand while guiding the needle. Hands

should be in the opposite position if the left femoral vein is being cannulated. (The femoral vein is medial to the artery.)

- Aspirate dark (venous) blood from the vein and remove the syringe. Verify that blood flow is not pulsatile.
- Pass the guidewire through the needle. Do not advance the wire if any resistance is encountered.
- One hand should hold the guidewire at all times.
- Make a stab incision along the wire with a #11 blade.
- Pass the dilator over the wire. Avoid excessive dilation of the vessel. The dilator should only be passed slightly farther than the depth at which blood was first aspirated from the needle. Excessive dilation increases the risk of bleeding and arteriovenous fistula or pseudoaneurysm formation.

Special Considerations

- Always use adequate local anesthesia at the puncture site when the procedure is done on an awake patient.
- Unless access must be obtained urgently, the operator attempting the procedure must wear a cap, eye shield, and mask with sterile gown and gloves.
- Do not attempt to insert the needle above the inguinal ligament. This will increase the risk of retroperitoneal hematoma or intra-abdominal injury.
- If the location of vessel is in doubt, a finder (25-gauge) needle attached to 5-mL syringe can be used to note the depth and location of the vessel.
- Always occlude the open hub of needle to prevent air embolism.

Complications

- Infection.
- Arterial bleeding.
- Retroperitoneal hematoma. (Always insert a femoral vein catheter below the inguinal ligament).

Further Reading

Tsui JY, Collins AB, White DW, Lai J, Tabas JA. Videos in clinical medicine. Placement of a femoral venous catheter. *N Engl J Med.* 2008 Jun 26;358(26):e30.

Fiberoptic Intubation

Fiberoptic intubation can be done either orally or nasotracheally. Fiberoptic intubating scopes are smaller in diameter than bronchoscopes

used for procedures. Only antero-posterior movement of the tip is possible in a fiberoptic intubating bronchoscope. The eye piece of the fiberoptic scope must be rotated for side-to-side movement.

Indications

- Anticipated/unanticipated difficult endotracheal intubation.
- Unstable cervical spine (to minimize movement of the cervical spine during intubation).

Contraindications

- Consider other techniques or a surgical airway if the patient is hypoxic and immediate access to the airway is required.
- Excessive secretions or blood in the airway is a relative contraindication because this distorts the view through the fiberoptic scope.

Equipment Checklist

- A working fiberoptic bronchoscope (ideally with a video display), oxygen connected to the side port, and suction.
- Appropriate size endotracheal tube threaded over the fiberoptic scope.
- Intubating airway (e.g. Ovassapian airway, Richards Medical Equipment, Wheeling, IL, USA)
- 2% lidocaine and viscous lidocaine.

Procedure

The success of an awake fiberoptic intubation depends upon effective airway anesthesia. Hence local anesthesia of the airway is discussed first, followed by fiberoptic intubation.

- Administer an antisialagogue (glycopyrrolate 0.2 mg IV) to dry the mucosa and improve the efficacy of the local anesthesia.
- Consider mild sedation depending on the clinical status of the patient and operator's choice (e.g., incremental doses of midazolam 0.5 mg IV or fentanyl 25 mcg IV).
- Topical anesthesia should be applied to three regions:
 - Nasal cavity and nasopharynx—supplied by the greater and lesser palatine nerves and anterior ethmoidal nerves.
 - Oral cavity, posterior third of the tongue and the pharynx—posterior third of the tongue, anterior surface of epiglottis, vallecula, lingual tonsil and pharyngeal wall are innervated by the branches of the glossopharyngeal nerve.
 - Hypopharynx, larynx and trachea—innervated by the branches of the vagus nerve (superior laryngeal and recurrent laryngeal nerves).
- If a nasal intubation is planned, anesthetize the nasal cavity and nasopharynx by placing cotton pledgets soaked in 2% lidocaine on

the posterior wall of the nasopharynx along the upper border of the middle turbinate (greater and lesser palatine nerves) and in the roof of nasal cavity close to the cribriform plate (anterior ethmoidal nerve) bilaterally.

- Anesthetize the oropharynx with viscous 2% lidocaine. Cotton pledgets soaked in lidocaine are placed close to the inferior aspect of the palatoglossal fold to anesthetize the glossopharyngeal nerve and suppress the gag reflex.
- Transtracheal injection of 2% lidocaine (with 25 g needle inserted through the cricothyroid membrane) anesthetizes the trachea.
- If the patient is cooperative, consider blocking the superior laryngeal nerve. Displace the hyoid bone toward the side being blocked while displacing the carotid artery laterally and posteriorly. “Walk” the needle off the hyoid cartilage until it pierces the thyro-hyoid membrane. The needle should be directed in the anterior and cephalic direction. Aspirate before injecting to ensure that the needle has not entered the pharynx or a blood vessel and then inject 2% lidocaine 2 mL.
- Fiberoptic intubation:
 - Lubricate the bronchoscope and thread the appropriate size endotracheal tube over it, securing the endotracheal tube with a piece of tape.
 - Insert an oral airway designed for fiberoptic intubation into the oral cavity (e.g., Ovassapian, Patil Syracuse, Berman, etc.). The airway directs the scope toward the larynx and the patient’s ability to tolerate the airway confirms airway anesthesia. In an anesthetized patient, the distal end of the airway goes behind the tongue and opens the space between the tongue and the pharyngeal wall.
 - The bronchoscope is held in the nondominant hand while the dominant hand guides the shaft of the scope. The thumb of the nondominant hand operates the lever controlling the movement of the tip of the scope in the anterior posterior direction. O₂ flowing at 2 L/min is connected to the side channel. This enriches the inhaled oxygen concentration and helps blow away any secretions from the tip of the scope.
 - The bronchoscope is then gently introduced through the oral airway.
 - The epiglottis is visualized at a depth of 10 cm. The tip of the bronchoscope will be just above the glottis when the 15 cm mark is at the lips. The position of the tip of the scope is adjusted to get clear visualization of the glottis.
 - With the scope just above the glottis, an epidural catheter can be threaded down the side port of the bronchoscope to spray the glottis with 2% lidocaine.

- The bronchoscope is then advanced through the larynx into the trachea. Avoid touching the carina because this is very irritating and will induce coughing in an awake patient.
- Gently pass the endotracheal tube along the bronchoscope. If resistance is felt, the most likely cause is that the tip of the tube is caught on the arytenoid cartilage. Rotate the tube 90 degrees counterclockwise and attempt to pass the tube again.
- Before advancing the endotracheal tube, consider administering propofol 20–30 mg IV to sedate the patient and permit the passage of the tube.

Special Considerations

- The endotracheal tube should be advanced during an inspiration when the cords abduct.
- Always use a topical vasoconstrictor (phenylephrine or oxymetazoline spray) and a smaller endotracheal tube if a nasal intubation is planned.
- In anesthetized patients, gently lifting the jaw moves the tongue away from the pharyngeal wall and opens the pharynx. If this procedure is not effective, the tongue may have to be pulled forward to open a path through the pharynx.

Intubating Laryngeal Mask Airway

The laryngeal mask airway can be used for oral endotracheal intubation in an emergency. The intubating LMA (ILMA, also referred to as LMA Fastrach®) is a rigid LMA with a wider lumen (13 mm internal diameter) and was specifically designed for this purpose. It is shorter in length and has a handle that permits manipulation of the shaft and orifice, while limiting movement of the patient's head and neck. A silicone endotracheal tube has been designed for use with the ILMA.

Indications

- Difficult intubation where conventional techniques of intubation fail (e.g., direct laryngoscopy, fiberoptic intubation)
- Intubation in situations when good visualization of the larynx is expected, but visualization of the vocal cord is expected to be difficult (e.g., lingual tonsillar hyperplasia or immobility of cervical spine)
- In a “cannot intubate, cannot ventilate” scenario, ILMA may facilitate definitive airway management

Contraindications

- Severe trismus or inability to open the mouth for other reasons
- Airway pathology involving the tongue, pharynx or larynx

Equipment Checklist

- ILMA (Four sizes are available. Size 3 is preferred in children, size 4 in adults 50–70 kg, and size 5 in adults 70–100 kg.)
- Cuffed silicone endotracheal tube
- Stabilizer rod (for stabilizing the endotracheal tube when the LMA is being withdrawn over the endotracheal tube)

Procedure

- The ILMA is generally used in anesthetized patients.
- Check the patency of the cuff and lubricate the ILMA with a water-soluble lubricant.
- Position the patient supine with the head and neck in neutral position.
- Hold the device in the left hand while opening the patient's mouth with the right hand.
- Insert the ILMA with the handle of the ILMA parallel to the chest wall, and cup of the LMA orifice facing down resting against the palate.
- Exerting gentle pressure on the palate, advance the ILMA along the contour of the palate until some resistance is encountered at the level of the hypopharynx.
- Once the device is in place, inflate the cuff and confirm the position of the LMA by ventilating the patient.
- Pass the silicone endotracheal tube through the ILMA with the longitudinal black line facing cephalad (this ensures that the bevel of the endotracheal tube faces to the left).
- Insert the endotracheal tube 15 cm (horizontal black line on the tube) and then advance it another 1.5 cm. At this point the tip of the ET tube lifts up the epiglottis elevator in the LMA. If no resistance is encountered, the ETT is gently advanced into the trachea.
- If resistance is encountered, grasp the ILMA by its handle and gently lift it (the “Vergheze” maneuver) while advancing the ETT into the trachea. This maneuver seals the cup of the LMA against the laryngeal orifice.
- Once the ETT is in place, inflate its cuff and confirm its position by ventilating the patient and observing for CO₂ return.
- Remove the ILMA over the endotracheal tube. Deflate the cuff of the ILMA and withdraw the ILMA while holding the ETT in place. When the LMA is over the proximal end of the ET tube, the stabilizer rod is introduced through the ILMA into the proximal end of the ET tube to stabilize the ET tube, and the ILMA is gently removed over it.
- Confirm the position of the ET tube again by ventilating the patient.

Complications

- The epiglottis may become folded as ILMA is advanced, thus occluding the airway. If this occurs, gently withdraw the ILMA while rotating it along the axis of the airway, and then reintroduce it.
- Do not inflate the ILMA for longer than 15 minutes because it can exert excessive pressure on the surrounding tissues.

Special Considerations

- Select the appropriate size ILMA to ensure correct placement of the device.
- If the distal end of LMA tends to get folded, insert it partially inflated.
- Use the stabilizer device while removing the ILMA over the ET tube. If the physician is unfamiliar with the procedure for removing the ILMA it can be left in place (with the cuff deflated) until the end of the surgical procedure.

Retrograde Intubation

Definition

An intubation technique in which a guide wire is inserted through the cricothyroid membrane and retrieved from the mouth (or nose). An ETT is then threaded over the guide wire into the trachea.

Indications

Retrograde intubation is indicated when conventional techniques for intubation either fail or are not feasible.

- Unstable cervical spine
- Excessive secretions or blood in the airway
- Oropharyngeal malignancy
- Small mouth opening (direct laryngoscopy and fiberoptic intubation requires that the mouth open 3–4 cm)

Contraindications

- Inability to access the cricothyroid membrane
- Coagulopathy, local infection
- Tumor or trauma involving the larynx
- Retrograde intubation is an elective procedure and is not a preferred technique in an emergency.

Equipment Checklist

- 18 g intravenous catheter (or Tuohy needle)

- .035" guidewire (75 cm long—2–2.5 times the length of a standard endotracheal tube)
- 3 mL syringe
- Magill forceps
- Endotracheal tube

Procedure

- Position the patient supine with the neck slightly extended.
- Spray the posterior part of the tongue and pharynx with 4% lidocaine. (A bilateral superior laryngeal block can also be performed for laryngeal anesthesia.)
- Clean and drape the anterior part of the neck.
- Identify the cricothyroid membrane and infiltrate the skin and subcutaneous tissue over the cricothyroid membrane with 1% lidocaine.
- Puncture the cricothyroid membrane with a 26 g needle and inject 2 mL of 2% lidocaine into the trachea.
- Insert the 18 g catheter into the trachea through the cricothyroid membrane. Confirm its position by aspirating air.
- Direct the catheter cephalad. The .035" guide wire is threaded through the catheter and retrieved from the mouth. A laryngoscope and Magill forceps may be required to reach the proximal end of the guide wire.
- Remove the catheter and place a small clamp over the distal end of the guide wire to prevent it from slipping out.
- Thread the ETT over the proximal end of the guide wire. Maintain tension by gently pulling on both ends of the wire while the ETT is being passed.
- Remove the guide wire from the cricothyroid membrane and pass the ETT further down into the trachea.

Special Considerations

- The guidewire can be threaded through the Murphy's eye of the endotracheal tube to provide better control over the distal end of the endotracheal tube.
- An epidural catheter can be used instead of a guidewire, but it is easier to retrieve a wire than an epidural catheter from the proximal end.
- Once the ETT enters the trachea, a soft bougie can be passed before advancing the tube further into the trachea.
- If resistance is encountered when passing the ETT, the most likely cause is that the tube is impinging on the pyriform fossa. Gently rotate the tube while advancing it.

Transcutaneous Pacing

Definition

External cardiac pacing is a potentially life-saving measure in patients with hemodynamic compromise due to a disturbed conduction system. This is a bridge to more definitive treatment (e.g., transvenous pacing).

Indications

- Acute myocardial infarction:
 - Asystole
 - Symptomatic second- or third-degree atrioventricular (AV) block
 - New trifascicular block
 - Second or third degree AV block without symptoms
- Not related to an acute myocardial infarction:
 - Asystole
 - Symptomatic second or third degree AV block (hypotension, pulmonary edema, angina or other signs of end-organ hypoperfusion)
 - Symptomatic sinus or junctional bradycardia
 - Tachyarrhythmias secondary to bradycardia
 - As a precaution in patients undergoing alcohol septal ablation who have a high risk of developing acute third-degree heart block
 - As a precaution in patients undergoing percutaneous coronary rotational atherectomy (rotablation), rheolytic thrombectomy (Angio Jet®), or a generator replacement in a patient who is pacemaker-dependent
 - After cardiac surgery, especially valvular heart surgery
- Overdrive suppression of tachyarrhythmias:
 - To prevent or treat torsades de pointes
 - Certain atrial tachycardias, such as atrial flutter, can be terminated with rapid atrial pacing

Contraindications

- Absolute: None
- Relative:
 - Severe hypothermia (due to propensity for induction of ventricular fibrillation)
 - Digoxin toxicity
 - Bradysystolic arrest for more than 20 minutes (minimal chance of successful resuscitation)

Complications

- Pain. May be minimized by proper pad placement, use of the lowest effective current, and judicious administration of sedatives and analgesics
- Atrial or ventricular dysrhythmia
- Pacemaker Syndrome: When temporary pacing starts, loss of synchrony between the atria and ventricles or retrograde activation of the atrium may occasionally result in undesirable hemodynamic changes including loss of atrial kick, reverse blood flow with cannon A waves, and decreased cardiac output.
- Coughing and hiccups may occur secondary to stimulation of the diaphragm and thoracic muscles.
- Skin burns have been reported with prolonged use.
- Failure to capture—potential causes include improper pad placement, poor skin contact (excessive hair, wet skin, or pad loosely applied), inadequate current output, and faulty or improperly set up equipment. Anatomic impediments to current delivery may include fluid (pericardial effusion) or air (pneumothorax, chronic obstructive pulmonary disease).

Equipment Checklist

- External pacemaker (Most defibrillators have this capability.)
- 2 pacing electrode pads
- Assistants to roll an uncooperative patient

Pad Application

Pacemaker pads, often labeled “front/back” or “anterior/posterior,” are applied over the cardiac apex and just medial to the left scapula. It may be necessary to shave excessive body hair to ensure a good electrical connection to the patient.

Pacemaker Operation

Set the heart rate to 80 bpm. Set the current to 0 mA if the patient is conscious. The pacemaker unit is then turned on, and the current is increased in 10-mA increments until capture is achieved. **In the unconscious patient, begin with a high current output (200 mA) to quickly achieve capture and then decrease the output to the level needed to maintain capture.**

Synchronous/Asynchronous Modes

In the asynchronous (fixed-rate) mode, the pacemaker delivers an electrical stimulus at preset intervals, independent of intrinsic cardiac activity. This mode may induce dysrhythmias if stimulation occurs during the vulnerable period of the cardiac cycle (R on T). Synchronous pacing is a “demand mode” in which the pacer fires only when no complex is sensed for a predetermined amount of time. Pacing generally should be started in the synchronous mode.

Confirmation of Capture

Electrical capture is documented on the ECG by the presence of a widened QRS complex that should correlate with palpable carotid pulses. In the hypotensive patient, echocardiography may be used to confirm cardiac contraction that correlates with electrical activity.

Transport of a Critically Ill Patient

Definition

Intrahospital transport of a critically ill patient.

Problems During Transport

- Critically ill patient requiring cardiorespiratory care that involves continuous monitoring
- Need to provide critical care during transport
- Limited monitoring and ventilation capabilities during transport
- Anticipate and be prepared to manage problems such as hypotension, hypoventilation. Be prepared to identify and manage new problems as they arise.
- Equipment used for transport may have unique failure modes and the alarms may sound different, making some problems difficult to identify.

Principles of Management

- If possible, stabilize the patient's hemodynamic status and ventilation before transport.
- Ensure that the equipment required for transporting the patient (e.g., monitor, transport ventilator, infusion pumps) is functional.
Check battery power.
- Anticipate which drugs and medications will be required during transportation and pre-fill syringes for rapid administration.
- Attempt to continue the same mode of ventilation in order to avoid abrupt changes in ventilation status during transport.
- If the patient is on hemodynamic support, it is advisable to continue these infusions during transport and make changes at the patient's destination.
- Ideally, the physician caring for the patient should manage the patient during the transport and hand over the care of the patient to the physician at the patient's destination.

Equipment Check

- Transport monitor
- Transport ventilator (O₂ tank should be full—2200 PSI)
- Infusion pumps

- Endotracheal tube, laryngoscope, and other airway management equipment (Be ready to reintubate or mask ventilate if the endotracheal tube is dislodged during transport.)
- Confirm that all battery-powered devices are fully charged and have enough power to last at least as long as the anticipated transport time, plus a generous reserve. Use fully-charged batteries whenever possible.

Procedure

Preparation for Transport

- The transport team should be familiar with the clinical status of the patient.
- To the extent possible, stabilize the patient prior to transfer.
- If the patient requires sedation or neuromuscular blockade, administer these drug before transporting and bring an extra supply.
- Confirm that the care team at the destination knows that the patient is en route and is ready.

Checklist for Transportation

- **Airway:** Ensure that the patient's airway is secure. If patient is intubated, check the position and security of the endotracheal tube.
- **Breathing:** If the patient is on respiratory support, connect the transport ventilator and make sure patient is being ventilated adequately.
- **Circulation:** To the extent possible, stabilize the patient's vital signs before transport. If patient is on vasopressors, confirm that the infusion is flowing. Confirm that intravenous drug and fluid infusions (especially vasoactive drugs) are flowing well. Be certain that drugs and battery power will last for the entire trip. Plan to have adequate reserves in the event that there is a delay while en route (e.g., an elevator gets stuck).
- **Position in bed:** Most patients can be transported with the head end of bed slightly elevated. Patients with an unstable spine may have to remain flat.

Transfer of Care

When the patient reaches the ICU:

- Connect the patient to the monitor in the ICU.
- Connect the patient to the ventilator on appropriate settings.
- Make sure that all the infusions are flowing.
- Check the vital signs and record one set of vital signs in the transfer note.
- Hand over care to the ICU physician with a detailed description of the management of the patient (both verbal as well as a written record).

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Transvenous Pacing

Definition

Invasive electrical pacing is used to initiate myocardial contractions when intrinsic stimulation is insufficient, the native impulses are not being conducted, or the heart rate is too slow to maintain an adequate cardiac output.

Indications

- See Transcutaneous Pacing (Page 286).
- Patients who do not tolerate or are refractory to transcutaneous pacing
- As a bridge when a permanent pacemaker is not functioning properly in a pacemaker-dependent patient

Contraindications

- Hypothermia (increases the irritability of the myocardium and may predispose to ventricular fibrillation when the pacing wires contact the myocardium)
- Digoxin toxicity and other drugs that may increase the irritability of the myocardium are considered relative contraindications.
- A patient who has been asystolic for an extended period of time is not a candidate for transvenous pacer placement.

Equipment

- Airway management equipment
- “Crash cart” with resuscitative drugs
- Defibrillator
- Flexible transvenous pacing catheter
- Pacemaker generator
- Spare battery for the pacemaker
- Percutaneous sheath introducer kit
- Cardiac monitor
- Local anesthetic solution
- Suture material
- Alligator clips and connecting wire
- Shoulder roll

Technique

- **The physician must be familiar with the pacemaker generator prior to using it in an emergency situation.**
- Access the central venous circulation by placing a percutaneous sheath introducer.
- Attach V1 lead to the pacemaker catheter negative terminal. This is done using an insulated wire with an alligator clip at each end. Attach one alligator clip to the negative pacemaker wire and attach the clip to the V1 lead on the ECG monitor. Inflate the balloon. Set the ECG monitor to lead V1.
- Insert the pacemaker catheter through the rubber diaphragm of the introducer. Advance the catheter 10 centimeters to ensure that the balloon is within the vascular system. As with all balloon-tipped catheters, the balloon should always be deflated prior to withdrawal.
- Slowly advance the catheter while observing the ECG monitor at all times. In the subclavian or internal jugular vein, the P wave and the QRS complex are both small in amplitude and inverted. In the superior vena cava, the P wave increases in amplitude (still inverted), while the QRS complex is unchanged.
- When the pacing catheter reaches the right atrium, a large P wave with a negative polarity and a small QRS complex will be seen. Continue to advance the catheter.
- As the right atrium is entered, the P wave becomes upright and the QRS complex increases in amplitude. Now continue to advance the catheter to the right ventricle, where the QRS complex should appear normal in V1. When the catheter is floating freely in the right ventricle, the P wave will be upright with a large-amplitude QRS complex.
- Deflate the balloon and advance the catheter until ST-segment elevation is observed. This indicates that the catheter is abutting the right ventricular wall.
- Connect the pacemaker generator to the catheter. Disconnect the negative terminal of the pacemaker catheter from the ECG lead. Connect the pacemaker catheter terminals on the proximal end of the catheter to the negative and positive terminals of the pacemaker generator.
- Set the pacemaker generator on demand mode (to fire only when a spontaneous beat has not occurred) with a rate of 60–80 beats per minute. Start with 5 mA of energy on the output dial.
- Turn on the pacemaker. Increase the energy until capture is seen on the monitor. This is indicated by pacing spikes and a wide QRS complex in lead V1.
- The energy needed for successful pacing (the pacing threshold) is assessed by reducing the energy until the pacemaker fails to

stimulate contraction (loss of capture). The output is then set at three times the threshold value to prevent inadvertent loss of capture.

- Chest radiographs should be obtained after the patient is stabilized to ensure proper tip placement and to rule out a pneumothorax from the central venous catheter placement.
- Firmly affix the pacing catheter to the insertion site prior to transferring the patient.

Complications

- Perforation of the ventricular septum, the myocardium of the atria, or the free ventricular wall. This is more common with the rigid catheters. Suspect septal perforation if the pattern on the ECG changes from a left to a right bundle branch block, or if an abrupt increase in the pacing threshold occurs. Ventricular perforation can present as failure to capture or as cardiac tamponade. Perforation of the inferior wall could stimulate and pace the diaphragm.
- If cardiac arrhythmias occur, immediately withdraw the catheter a few centimeters and observe the rhythm.
- Infection can be a delayed complication.
- Air embolism
- Complications related to obtaining central venous access

Special Considerations

- Fluoroscopic guidance can be used to ensure accurate placement of the pacing wire in the right ventricle.
- Electrocardiographic guidance is useful in patients with narrow complexes and/or P waves when fluoroscopy is unavailable.
- In a true emergency, the pacemaker electrodes are connected to the power source and the catheter advanced blindly so that the tip will encounter the endocardium of the right ventricle and capture will result. Vascular access should be through the right internal jugular vein so that the catheter traverses a straight line into the right ventricle and does not curl up in the atrium or deflect into the inferior vena cava.
- A flexible catheter should always be used to decrease the risk of myocardial injury.

Ultrasound-Guided Central Venous Access

Definition

The use of ultrasonography to facilitate placement of vascular catheters into the internal jugular, subclavian, or femoral veins. This technique can also be used to access peripheral veins and arteries.

Contraindications

None

Anatomy

- Internal and external jugular veins
- Femoral vein
- Antecubital vein
- Subclavian vein (difficult to visualize on ultrasound because of air)
- Veins are nonpulsatile, easily compressible, and distended when the patient is in head-down position or performs a Valsalva maneuver. Arteries are noncompressible when moderate pressure is applied with the probe.
- Fluid appears black, while bone and air appear white on ultrasound.

Equipment Checklist

- Ultrasound equipment:
 - Linear probes are used most commonly, although curved probes may also work.
 - Vessels are usually superficial, so most frequencies between 2–10 MHz are adequate.
 - Sterile sheath for the probe with gel and elastic band (usually included together in a set).
- Usual equipment for central line placement

Technique for Right Internal Jugular Central Line Placement

- Clean and drape the area using standard aseptic technique.
- Prepare the probe:
 - Place gel on the distal end of sheath and gently roll over the probe. Air bubbles between the probe and the inner surface of the sterile sheath will degrade the image.
 - Secure with the elastic band.
- If possible, place the patient in head-down (Trendelenburg) position at around 15 degrees to maximize the vessel diameter
- Place the ultrasound machine at the head of the bed. The probe will be placed in a transverse position on the patient's neck, caudal to the needle insertion site.
- Perform ultrasonographic examination of the neck to determine the best site for needle puncture and central line placement.
- Track the internal jugular vein from the angle of the mandible down into the supraclavicular fossa using the linear ultrasound probe in the transverse orientation.

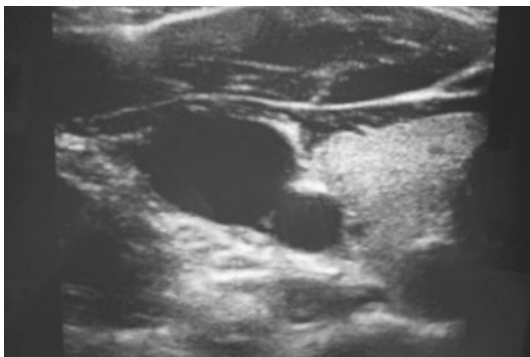


Figure 11.3 The internal jugular vein lateral to the internal carotid artery.

- During the initial scan of the internal jugular vein, make note of vessel patency, diameter, degree of collapse with respiration, overlap with the internal carotid artery, and depth from the skin. Figure 11.3 shows the larger, thin-walled internal jugular vein lateral to the smaller, thick-walled internal carotid artery
- Determine whether the internal jugular vein is patent throughout its course. Internal jugular vein size of less than 7 mm may be an independent risk factor for unsuccessful venous cannulation. Attempt to find a location where the internal jugular vein only minimally overlies the internal carotid artery. This will reduce the chance of inadvertent arterial puncture.
- Do not turn the patient's head more than 30 degrees from midline. (Increased risk of arterial puncture.)
- Infiltrate the skin with local anesthesia (1% lidocaine).
- The needle should be angled at 40–60 degrees from the plane of the neck and 1–2 cm back from the middle of the ultrasound probe. During initial skin puncture, the operator should note a depression of the skin on the ultrasound monitor. If the needle is aligned correctly, this soft tissue depression should be located directly over the target vessel. Always look at the patient's neck and the needle during any adjustments in needle position
- Gently advance the needle in 1 cm increments; entry into the vein occurs with a distinct “pop” and aspiration of blood will be possible.
- Once blood is aspirated, the guide wire can be passed through the needle into the vein. Confirm position of the guidewire with ultrasound. When the subclavian vein is being cannulated, ensure that the wire has passed into the superior vena cava.

- Continue placement of the catheter using the Seldinger technique.
- Confirm the position of the catheter with chest x-ray.

Complications

As with central line insertion

Further Reading

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Chapter 12

Regional Anesthesia Complications

Raymond S. Sinatra and Dan B. Froicu

- Bupivacaine Cardiotoxicity 298
- Epidural Abscess 300
- Epidural Hematoma 301
- Globe Injury 304
- Local Anesthetic Toxicity 305
- Nerve Injury 308
- Total Spinal Anesthesia 310

Bupivacaine Cardiotoxicity

Definition

Profound depression of heart rate and/or contractility caused by high plasma concentrations of bupivacaine and subsequent myocardial uptake. Cardiac effects are life threatening.

Presentation

- *Neurotoxicity.* Tinnitus, perioral numbness, tremors, seizures, lethargy, coma. These symptoms appear first, and are rapidly followed by cardiovascular effects.
- *Cardiac dysrhythmias.* Initially widened QRS and bradyarrhythmias, which may be quickly followed by ventricular tachycardia, ventricular fibrillation and asystole.
- *Hemodynamic changes.* Hypotension, cardiovascular collapse.

Pathophysiology

Bupivacaine is highly protein-bound and is rapidly taken up by contractile and conducting cardiac cells. Bupivacaine decreases conductance of sodium channels through a fast-in, slow-out mechanism that affects impulse conduction and cardiac contractility. Blockade of myocardial sodium channels results in decreased cardiac output, fatal arrhythmias and cardiovascular collapse.

Immediate Management

- Airway, breathing, circulation. Administer 100% O₂.
- Intubate the patient and initiate mechanical ventilation.
- Begin advanced cardiac life support (ACLS) if indicated.
- Promptly treat ventricular arrhythmias: amiodarone 150 mg slow IV (may cause hypotension).
- Support blood pressure with vasopressors (e.g., vasopressin, epinephrine). Consider CaCl₂.
- Check arterial blood gases (ABG) frequently. Treat acid-base disturbances aggressively.
- Administer lipid emulsion. One recommended protocol is lipid emulsion 20% 1.5 mL/kg over 1 minute, followed by a continuous infusion of 0.25 mL/kg/min for 30–60 minutes.
- Consider open chest massage and/or cardiac bypass for patients unresponsive to intravenous lipid emulsion.

DIFFERENTIAL DIAGNOSIS

- Vasovagal episode
- Acute myocardial infarction
- Intrathecal injection and total spinal anesthesia
- Profound hypotension caused by preexisting hypovolemia.

Diagnostic Studies

Arterial blood gas analysis, plasma electrolytes to correct acidosis and electrolyte abnormalities

Subsequent Management

- **Myocardial depression may not resolve for 80–90 minutes. Do not discontinue resuscitation efforts!**
- If the patient does not respond to standard resuscitation techniques, consider open chest cardiac massage and/or cardiac bypass.

Risk Factors

- Pregnancy
- Hypokalemia, hypercarbia
- Cardiac disease

299

Prevention

- Careful needle placement, frequent aspiration and bupivacaine administration in divided doses. Always use a test dose containing epinephrine 1:200,000.
- 20% lipid emulsion in sufficient quantity to treat bupivacaine toxicity should be available wherever local anesthetics and regional anesthesia are used. The regional anesthesia cart should include a 500 mL vial of 20% intralipid, and a 60 mL syringe.
- Careful monitoring of ECG and ongoing patient assessment is essential.
- Accidental intravenous injection is the most common cause of cardiac toxicity.

Special Considerations

Do not administer more than 3 mL of 0.5% bupivacaine or 6 mL of 0.25% bupivacaine at one time. Do not exceed 1.6–1.7 mg/kg with plain bupivacaine solution, or 2 mg/kg with epinephrine-containing solution.

Further Reading

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- Weinberg G. Intralipid for control of bupivacaine cardiotoxicity> Available at: www.lipidrescue.org. Accessed: October 5, 2009.

Epidural Abscess

Definition

Epidural abscess formation, either spontaneously or after attempted or successful epidural anesthesia.

Presentation

- Back pain, pain along nerve root distribution, weakness.
- Progressive weakness with abscess formation; if abscess left untreated paraplegia can develop.
- Neck stiffness, headache, signs of meningitis.
- Fever, tenderness at epidural site.
- Leucocytosis, sometimes present after a variable delay of days, weeks, or even months after epidural anesthesia.

Pathophysiology

Local infection of the epidural needle/catheter site, most often with *S. aureus*, causes an abscess with associated inflammation and mass effect that cause spinal cord symptoms (pain, incontinence, paresthesias).

DIFFERENTIAL DIAGNOSIS

- Epidural hematoma
- Muscular or ligamentous injury related to needle placement
- Residual local anesthetic effect
- Spinal artery thrombosis

Immediate Management

- Stop epidural drug administration.
- Emergency MRI
- Emergency neurosurgical consultation for drainage of the abscess

Diagnostic Studies

Emergency MRI of lumbar and/or thoracic spine

Subsequent Management

- Close neurologic monitoring until function is restored
- Repeat MRI if warranted

Risk Factors

- Existing infection
- Diabetes mellitus
- Cancer
- Steroid use
- Alcohol abuse

Prevention

- Use careful aseptic technique when placing neuraxial blocks.
- Infection at the procedure site is a contraindication for epidural placement.
- Check the skin at the site of indwelling catheters for signs of infection daily.
- Promptly remove the catheter if erythema or local discharge is noted.
- Antibiotic prophylaxis and/or bacterial filter use may be warranted.

Special Considerations

Specifically inform the patient of the risks of the procedure.

Further Reading

Pradilla G, Ardila GP, Hsu W, Rigamonti D. Epidural abscesses of the CNS. *Lancet Neurol.* 2009 Mar;8(3):292–300.

Epidural Hematoma

Definition

An expanding collection of blood related to damage to an epidural blood vessel that causes progressive spinal cord compression and paralysis

Presentation

- Presentation is often slow, insidious and progressive.
- The initial symptom is back pain at the site or immediately adjacent to the site of epidural needle or catheter placement.

- Worsening back pain with loss of posterior column sensation (vibration sense, two point discrimination). Loss of sphincter tone and urinary incontinence or retention may also be observed.
- Motor weakness progressing to paralysis. Pain, temperature, and light touch sensation may remain intact despite motor deficits.

Pathophysiology

Spinal or epidural needle placement may damage an epidural vein or artery, resulting in hematoma formation. The hematoma irritates nerve endings in the epidural space, resulting in acute back pain, and may eventually cause spinal cord compression. The posterior columns are the first structure to be affected, impairing vibration, two point discrimination, and position sense. As the hematoma expands, the cortical spinal motor tracts are compromised and the patient becomes paraplegic. The anterior lateral spinal thalamic tract that conveys pain, temperature, and light touch is furthest from the hematoma and last to be affected.

Immediate Management

- Delayed recognition of epidural hematoma may result in permanent, devastating neurologic injury.
- Do not administer opioids to patients complaining of worsening back pain during or following epidural catheter placement.
- Emergency MRI to rule out hematoma in patients with unexplained sensory deficits or motor weakness.
- Emergency neurosurgical consultation for surgical decompression.

DIFFERENTIAL DIAGNOSIS

- Muscular or ligamentous injury related to needle placement
- Residual neural blockade
- Epidural abscess
- Spinal artery thrombosis

Diagnostic Studies

- MRI or CT to detect collections of blood or abscess in the epidural space. MRI is more sensitive, and is the preferred study if available and the clinical situation permits.
- Physical examination: Assess ability to detect vibration and position in the lower extremities. Pain, temperature, and light touch are the last sensory modalities to be affected.
- Assess rectal tone.

Subsequent Management

- After obtaining a baseline neurologic examination, follow patients carefully for 24–36 hours.
- Consider an early neurosurgical consultation in settings of abnormal neurological assessment.

Risk Factors

- Anticoagulation including heparin infusion, coumadin, and low-molecular-weight heparin
- Herbal preparations: ginkgo bilboa, saw palmetto, and garlic
- NSAID use
- Epidural placement for pain following cardiac surgery
- Hypertension
- Pregnancy with engorged epidural veins
- Spondylolisthesis, spinal stenosis
- Hematoma may occur in the absence of risk factors.

Prevention

- Avoid epidural placement in patients who are treated with anticoagulants or will receive them after surgery.
- Do not insert an epidural catheter within 12 hours of the last dose of low-molecular-weight heparin.
- Minimize needle placement attempts.
- Minimize the duration of epidural catheter placement.
- Avoid removal of epidural catheters in anticoagulated patients.

Special Considerations

- The occurrence of back pain with posterior column deficits after placement of an epidural catheter represents a true surgical emergency.
- The anesthesiologist may be the only member of the patient care team to recognize the symptoms and their implications.
- Surgical intervention must be performed before the patient develops motor deficits, or the injury may become irreversible.

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Globe Injury

Definition

Accidental penetration (needle entry) or perforation (needle entry and exit) of the globe of the eye with a needle during attempted ophthalmic regional anesthesia.

Presentation

- Sudden loss of vision, hypotonia, poor red reflex, vitreous hemorrhage.
- Pain, more frequent with intraocular anesthetic inadvertent injection.
- Intraocular hypertension occurs rarely if local anesthesia is injected into the globe. Awake patients may complain of severe pain at the time of injection.

Pathophysiology

Mechanical injury to the eye structures. Proliferative vitreoretinopathy and/or retinal detachment may occur after initial injury.

Immediate Management

- Emergency ophthalmological consultation for surgical treatment
- If injury is limited to a retinal tear, the ophthalmologist may elect to perform laser photocoagulation, cryotherapy. Minor cases may only require close observation.
- Patients with significant intraocular hemorrhage may benefit from early vitrectomy.

DIFFERENTIAL DIAGNOSIS

- Preexisting retinal/intraocular hemorrhage
- Optic nerve injury
- Retinal detachment
- Preexisting painful eye

Diagnostic Studies

Funduscopy, when ocular media are sufficiently clear.

Subsequent Management

- Complex, directed by ophthalmologist.

- Observation and additional surgery as vitreous hemorrhage caused by penetration often leads to proliferative vitreoretinopathy and/or retinal detachment.

Risk Factors

- Severe myopia.
- Surgery for retinal detachment
- Deep sedation

Prevention

- Avoid deep sedation in patients undergoing refractive or retinal detachment surgery.
- Insert the needle with the eyes in primary gaze.
- Always aspirate before injecting local anesthesia.
- Use short, sharp needles.

Special Considerations

- Other complications include optic nerve injury, retrobulbar hemorrhage, and brainstem anesthesia.
- The rate of complications is similar for both intraconal and periconal blocks.
- Ocular injury is more frequent in myopic patients because of the ovoid shape of the globe. This complication is rare in patients undergoing cataract extraction.
- There is no difference in the rate of perforation between anesthesiologists and ophthalmologists.

Further Reading

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- Goldberg M. Complications of anesthesia for ocular surgery. *Ophthalmol Clin North Am*. 2006 Jun;19(2):293–307.

Local Anesthetic Toxicity

Definition

Central nervous system excitatory response to local anesthetic overdose or intravascular injection.

Presentation

- CNS manifestations generally precede cardiovascular symptoms. Early symptoms include tinnitus, metallic taste, and tongue and

circumoral numbness. These symptoms may be followed by light-headedness, tremor, visual disturbances, and muscle twitching.

- With increased plasma levels, obtundation, seizures, coma, respiratory depression, or cardiac arrest may occur.

Pathophysiology

Central nervous system toxicity is both biphasic and proportional to levels of anesthetic in plasma and CNS. Local anesthetics initially block inhibitory CNS pathways and then produce diffuse CNS depression.

DIFFERENTIAL DIAGNOSIS

- Hypoxia
- Altered mental status (e.g., oversedation)
- Seizure of different etiology
- Preexisting neurological disease (e.g., CNS mass lesion, ambliopia, nystagmus)

Immediate Management

- Discontinue injection.
- Airway, breathing, circulation. Administer 100% O₂.
- Intubation is often not needed if the airway is adequate during the seizure, but may be necessary if postictal depression occurs.
- Administer sedatives to control the seizure (e.g., midazolam, diazepam, propofol).
- Be prepared to begin ACLS.
- Treat bradycardias with atropine.
- If bupivacaine toxicity is suspected and cardiac arrest is imminent, administer intravenous lipid emulsion. (See Bupivacaine Cardiotoxicity, page 298) One recommended protocol is intravenous lipid emulsion 20% 1.5 mL/kg over 1 minute, followed by a continuous infusion of 0.25 mL/kg/min for 30–60 minutes.

Diagnostic Studies

Clinical diagnosis. There are no specific diagnostic tests.

Subsequent Management

- If symptoms resolve rapidly, consider whether to continue with surgery.
- Provide supportive care.
- Consider admission to telemetry unit or intensive care unit if cardiovascular symptoms are present.
- Correct metabolic abnormalities.

Risk Factors

- Large volumes or high concentration of local anesthetic solution
- Intravenous regional anesthesia
- *Pregnancy.* Pregnant patients are at increased risk of intravascular epidural vein placement, and higher blood flow increases the rate of local anesthetic uptake. Progesterone may increase the risk of cardiotoxicity of ropivacaine and bupivacaine.
- *Hepatic failure.* Amide local anesthetics are metabolized in the liver. Use caution in patients with liver disease, because their ability to clear the drug may be significantly impaired.

Prevention

- Aspirate carefully after positioning the needle when performing a nerve block.
- Adding epinephrine to the local anesthetic solution will cause tachycardia if injected into a blood vessel, and speed the diagnosis of intravascular injection.
- Be prepared to treat profound CNS depression, cardiovascular collapse, respiratory depression or respiratory arrest whenever a block is being placed.
- The maximum recommended dose of lidocaine is 3–5 mg/kg. With the addition of epinephrine, 1:200,000, up to 7 mg/kg may be given.

Special Considerations

- Local anesthetic induced seizures are usually the result of intravascular injection, and less often the result of absorption from the administration site.
- Administration of sedatives (e.g., benzodiazepines) before a block may hide the early CNS signs of toxicity and increase the risk of cardiac toxicity.
- Symptoms are common, yet rarely clinically significant, with chloroprocaine, which is rapidly metabolized, and more problematic with bupivacaine and other slowly metabolized amide-based local anesthetics.
- The rate of absorption and risk of toxicity depend upon the injection site. Uptake and rise in plasma levels from slow to rapid is as follows: spinal, intra-articular, subcutaneous, femoral-sciatic, brachial plexus, epidural, caudal, intercostal, intrapleural. Lidocaine toxicity has a faster onset and resolution, while bupivacaine toxicity may start slowly but last much longer.
- Allergy to local anesthetics is extremely rare and is more common with ester local anesthetics.

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Nerve Injury

Definition

Injury to spinal cord, nerve root, or peripheral nerve caused by spinal, regional or epidural needles.

Presentation

- Patient complains of sudden “electric shock” during needle placement.
- Worsening “electric shock” or burning pain radiating into an extremity
- Nonsedated patients may report severe pain during injection.

Pathophysiology

Nerve injuries are a recognized complication of regional anesthesia. Quincke-tipped or cutting needles can transect axonal fibers, and symptoms often persist until the fibers regenerate, which may take months. Injection of local anesthetic solution exacerbates nerve injury, particularly if high concentrations are used.

DIFFERENTIAL DIAGNOSIS

- Nerve root injury
- Spinal injury
- Local anesthetic toxicity
- Underlying neurologic disease: myleopathic disease, preexisting radiculopathy, peripheral neuropathy

Immediate Management

- Immediately stop advancing a needle or catheter if the patient complains of pain or paresthesia.
- Do not inject local anesthetic if the patient complains of persistent paresthesias.
- Carefully withdraw the needle and catheter (if one was placed).
- Perform a full neurological assessment to rule out a focal deficit.
- Provide analgesics and anxiolytics to patients with painful paresthesias.

Diagnostic Studies

- Complete neurologic examination
- Consider an emergency MRI scan.
- Consider EMG testing to rule out nerve injury.

Subsequent Management

- Consider anti-neuropathic analgesics (gabapentin, pregabalin, tricyclic antidepressants).
- Consider a course of steroids to reduce neural inflammation.

Risk Factors

- Difficult technique due to patient anatomy (obese or elderly patients)
- Nerve block in an anesthetized or sedated patient
- Use of paresthesia eliciting techniques to facilitate regional nerve block
- Placement of spinal needles above the L1 interspace.

Prevention

- Careful needle and catheter placement
- Avoid direct nerve penetration
- Consider nerve stimulation or ultrasound needle guidance for peripheral neural blockade.
- Never inject local anesthetic to patients complaining of pain at the needle site.
- Do not administer more than 3 mL of local anesthetic as a test dose. Stop administration if the patient reports pain.

Special Considerations

- Many anesthesiologists elicit paresthesias to confirm local anesthetic injection and increase the success rate of the block. It is unclear whether this technique increases the risk of nerve injury.
- Nerve penetration with blunt or round tipped needles rarely results in permanent injury. In general, the nerve is not transected, but the Schwann cells and myelin sheath can be damaged. This form of injury is repaired fairly quickly and symptoms of numbness or mild neuropathic discomfort generally resolve over a period of days.
- It is unclear whether placement of an indwelling peripheral nerve or plexus catheter increases the risk of nerve injury. In a series of over 500 patients treated with continuous brachial plexus

catheters, only 3 (0.5%) experienced long-lasting paresthesia (see Further Reading, Lynch et al.)

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Liguori GA. Complications of regional anesthesia: nerve injury and peripheral neural blockade. *J Neurosurg Anesthesiol.* 2004 Jan;16(1):84–86.

Lynch NM, Cofield RH, Silbert PL, Hermann RC. Neurologic complications after total shoulder arthroplasty. *J Shoulder Elbow Surg.* 1996 Jan-Feb;5(1):53–61.

Total Spinal Anesthesia

Definition

Profound sensory motor and autonomic blockade and associated cardiovascular collapse or cardiac arrest related to unintentional or excessive intrathecal local anesthetic blockade.

Presentation

- The severity of symptoms may vary depending upon the concentration, volume, and baricity of the local anesthetic employed.
- Early symptoms include rapid and unexpected sensory motor blockade with associated hypotension and tachycardia.
- Progressive increases in the density and the number of dermatomes blocked—symptoms include upper extremity weakness, bradycardia, respiratory and CNS depression.
- Higher levels of dermatomal (above C4-5) and brainstem blockade, termed “total spinal”—dysarthria or aphonia, unconsciousness, cardiovascular collapse, respiratory arrest and death.

Pathophysiology

Patients experience rapid and dense sensorimotor blockade that can inhibit or prevent ventilation, as well as intrathecal sympatholysis that results in cardiovascular collapse and cardiac arrest.

DIFFERENTIAL DIAGNOSIS

- Dense epidural blockade
- Subdural injection (usually asymmetric blockade, with maintenance of respiration and ventilation)
- Preexisting conditions such as myelopathy, neuropathy, myasthenia gravis, hypocalcemia
- Hypermagnesemia

Immediate Management

- Intubate the trachea and begin mechanical ventilation. Increase FiO_2 to maintain adequate oxygenation.
- Attempt to assess the level dermatomal blockade if time permits.
- Patients treated with hyperbaric spinal solutions should be taken out of Trendelenburg position.
- Aggressively treat hypotension and bradycardia with fluids, vasopressors (epinephrine), and atropine.
- If cardiac arrest occurs, begin ACLS.
- The rapid onset of bradycardia or asystole combined with the loss of sympathetic tone caused by spinal blockade may explain why subsequent cardiac arrest may be refractory to treatment.

Diagnostic Studies

- Clinical diagnosis based on reported symptoms and physical examination
- No specific diagnostic tests other than assessment of dermatomal blockade

Subsequent Management

- Supportive care includes careful sedation and controlled ventilation followed by gradual weaning as motor strength returns (usually 90–120 min if bupivacaine was injected).
- Administer fluids and vasopressors as required.
- Correct metabolic abnormalities (see Metabolic Emergencies).
- Test epidural catheter—to confirm the diagnosis, attempt to aspirate cerebrospinal fluid (CSF), then carefully remove the catheter.
- Administer naloxone infusion if the patient was treated with intrathecal or epidural morphine.

Risk Factors

- Pregnancy: increased chance of high dermatomal blockade secondary to diminished intrathecal space, and neural blockade effects of progesterone and possibly magnesium infusion.
- Patients with difficult epidural landmarks (greater chance of unintentional intrathecal placement and injection).
- Patients who cough or retch excessively, or are quickly placed in Trendelenburg position following spinal injection.
- Preexisting disturbances (e.g., COPD, myasthenia, disautonomia)

Prevention

- When placing an epidural block, aspirate for CSF frequently after positioning the needle or catheter.
- If hyperbaric bupivacaine plus epinephrine is injected, the dermatomal level may continue to ascend for 12–15 minutes after administration. During this time, the level of blockade should be followed carefully.
- Dermatomal levels above C7 may be life threatening if not diagnosed and treated quickly.
- Symptoms may occur during epidural anesthesia with accidental intrathecal placement of the needle or catheter.
- Avoid placing the patient in the Trendelenburg position soon after injection of hyperbaric local anesthetic solutions.
- High spinal anesthesia has been reported in the setting of combined spinal and epidural anesthesia, and is caused by migration of the epidural catheter into the intrathecal space, or diffusion of local anesthetic into the intrathecal space through the dural puncture.

Special Considerations

- Dilated pupils indicate a high level of blockade, not CNS injury.
- Intrathecal blockade with lidocaine has a faster onset and resolution, while bupivacaine or bupivacaine plus epinephrine have a delayed onset and much longer duration of action.
- 14% of the neuraxial cardiac arrest claims from the 1980s and 1990s were associated with unintentional intrathecal blockade.

Further Reading

Bernards CM. Epidural and spinal anesthesia. In: Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC (Eds.), *Clinical Anesthesia*, 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:948.

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Chapter 13

Respiratory Emergencies

Vivek K. Moitra and Tricia E. Brentjens

- Acute Lung Injury (ALI) and Acute
Respiratory Distress Syndrome (ARDS) 314
- Bronchospasm 316
- Decreased ETCO_2 (Intraoperative) 317
- Difficult Controlled Ventilation 319
- Hemoptysis 322
- Hypercarbia (Intraoperative) 323
- Hypoxemia (Intraoperative) 326
- Pneumothorax 328
- Pulmonary Edema 330
- Pulmonary Embolus 332
- Respiratory Precautions 334

Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS)

Definition

Acute onset of bilateral pulmonary infiltrates on noted chest X-ray with pulmonary edema, poor systemic oxygenation and absence of left atrial hypertension.

Presentation

Patients with ALI or ARDS are intubated and mechanically ventilated. Their physical exam is notable for reduced breath sounds and possibly wheezing. Increased peak inspiratory and plateau pressures may be seen with positive pressure ventilation. Arterial blood gases may show hypercarbia in the setting of poor lung compliance.

Pathophysiology

The early phase of ARDS is characterized by pulmonary capillary leak and by interstitial and alveolar edema. There is a loss of surfactant activity. During the late phase of ARDS, pulmonary fibrosis and decreased lung compliance can develop.

Immediate Management

- Increase FIO_2 to maintain adequate oxygenation.
- Consider ventilation with low tidal volume (6–8 cc/kg predicted body weight).
- Avoid plateau pressures > 30 cm H_2O .
- Permissive hypercapnia may be necessary.
- Consider alternative ventilatory strategies (e.g., airway pressure release ventilation [APRV]).

DIFFERENTIAL DIAGNOSIS

- Pulmonary edema
- Multilobar pneumonia
- Diffuse alveolar hemorrhage
- Pneumonitis
- Pulmonary embolus
- Transfusion related acute lung injury (TRALI)
- Bronchiolitis obliterans-organizing pneumonia (BOOP)

Diagnostic Studies

- Chest X-ray (shows patchy infiltrates that extend to the periphery)

- Right heart catheterization (CVP or pulmonary artery catheter)

Subsequent Management

- Treat the precipitating cause of ALI/ARDS.
- Ventilator management of respiratory abnormalities
- Consider high-dose steroid therapy.
- Transfer the patient to the intensive care unit for further management.
- Neuromuscular blockade may rarely be required to facilitate ventilation and oxygenation.

Risk Factors

- Sepsis
- Pneumonia
- Pneumonitis
- Pancreatitis
- Toxic drug reaction
- Inhalational injury
- Massive Transfusion

Prevention

Early and aggressive treatment of precipitating causes may prevent progression to lung injury.

Special Considerations

- Patients who come to the operating room with ARDS can present with increased peak airway pressures and high levels of positive end-expiratory pressure (PEEP).
- Patients often require specialized transport from an ICU. (See “Transportation of Critically Ill Patients,” Page 288.)
- The anesthesiologist must be familiar with the patient’s mode of ventilation in order to ensure safe transport. Arterial blood gas samples can guide changes in ventilator strategy in the operating room.

Further Reading

Jason D. Christie Paul N. Lanken. Acute lung injury and the acute respiratory distress syndrome. In: JB Hall, G Schmidt, LDH Wood (eds.). *Principles of Critical Care*, 3rd ed. New York: McGraw-Hill; 2005:515–547.

Bronchospasm

Definition

Spasmodic contraction of bronchial smooth muscle.

Presentation

Decreased SpO₂ or an upsloping of the ETCO₂ tracing on the capnograph. An increase in peak airway pressure (PIP) may also be seen if the patient is mechanically ventilated. Wheezing or decreased breath sounds may be heard. Hypotension is a late sign in severe bronchospasm due to hypoxia or auto-PEEP leading to decreased venous return.

Pathophysiology

Bronchospasm can occur after a mechanical (intubation) or chemical (anaphylatoxin) stimulus activates mast cells, eosinophils, lymphocytes, epithelial cells and macrophages to release various mediators, i.e., histamine, to constrict bronchial smooth muscle. The hyperirritable airway is often edematous and produces mucus, which further increases airway resistance.

DIFFERENTIAL DIAGNOSIS

- Mechanical obstruction (kinked endotracheal tube)
- Pulmonary Edema
- Tension Pneumothorax
- Aspiration Pneumonitis
- Pulmonary embolus
- Pulmonary edema
- Endobronchial intubation

Immediate Management

- Increase FIO₂ to 100%.
- Increase depth of anesthesia with inhalational agents.
- Administer β -agonist bronchodilators.
- Consider terbutaline 0.25–0.5 mg subcutaneous injection.
- Consider epinephrine 10–30 mcg IV in refractory cases, titrate to effect.

Diagnostic Studies

Clinical presentation. No specific diagnostic studies.

Subsequent Management

- If surgery has not started, consider postponing an elective procedure if the patient is unstable.

- Consider administration of steroids.
- Maintain an adequate depth of anesthesia to prevent further bronchospasm.
- Avoid unnecessary airway manipulation.
- Avoid triggering agents, i.e., histamine-releasing drugs.

Risk Factors

- History of asthma, COPD, emphysema
- Recent upper airway infection
- Can occur in healthy patients

Prevention

- If the clinical situation permits, avoid endotracheal intubation in at-risk patients.
- Consider use of a regional anesthesia technique if the patient has a history of reactive airway disease.
- Patients with a history of asthma have bronchial hyperreactivity, and may benefit from preoperative corticosteroid treatment.
- IV agents, including propofol, ketamine and lidocaine, may decrease airway resistance.

Special Considerations

Even with adequate preparation and implementation of preventative measures, bronchospasm may still occur in the operating room.

Further Reading

Yao F. Asthma and chronic obstructive pulmonary disease. In: F Yao (ed.). *Anesthesiology: Problem-Oriented Patient Management*. 5th ed. New York: Lippincott Williams & Wilkins; 2003:3–31.

Decreased ETCO₂ (Intraoperative)

Definition

End tidal CO₂ < 30 mmHg

Presentation

A decrease in ETCO₂ in a mechanically ventilated patient may occur with either hypocarbia (hyperventilation) or hypercarbia (problem with CO₂ elimination). A sudden decrease in ETCO₂ may indicate cardiovascular collapse or an embolic phenomenon.

Etiology

Most commonly caused by hyperventilation during mechanical ventilation. It may also reflect increased dead space with a normal PaCO₂. Sudden, catastrophic decrease in cardiac output will decrease the ETCO₂ because of decreased perfusion (CO₂ is not being carried to lungs).

Immediate Management

- Assess cause of decreased ETCO₂
- Sudden:
 - Consider cardiovascular collapse.
 - Assess perfusion by other means (blood pressure, presence of pulse oximeter waveform).
 - Assess volume status.
 - Consider providing cardiovascular support using inotropes or vasopressors.
 - If an embolic event is suspected:
 - Adjust head of bed down.
 - Flush field with saline if air embolus is suspected.
 - Provide hemodynamic support.
- Gradual:
 - Decrease minute ventilation (if patient is being ventilated).

DIFFERENTIAL DIAGNOSIS

- Air leak in sample line
- Gas analyzer error
- Low ETCO₂ with hypocarbia (PaCO₂ < 35 mmHg)
- Hyperventilation
 - High minute ventilation in a mechanically ventilated patient
 - Pain
 - Anxiety
 - Compensation for metabolic acidosis
- Low ETCO₂ with hypercarbia (PaCO₂ >45 mmHg)
 - Pulmonary thromboembolus
 - Air embolus
 - Fat embolus
 - CO₂ embolus (laparoscopic surgery)
 - Amniotic fluid embolus
- Obstruction
 - Mechanical (kinking of tube)

- Bronchospasm
- COPD
- Low cardiac output state
- Esophageal intubation

Diagnostic Studies

- Arterial blood gas (ABG) to determine PaCO₂
- Transthoracic or transesophageal echocardiogram to assess cardiac function
- Spiral CT if thromboembolic event is suspected

Subsequent Management

- Correct the underlying cause.
- Provide hemodynamic support.
- Intubate the trachea and initiate mechanical ventilation.

Risk Factors

- Sitting craniotomy or any surgical procedure in which the operative site is above the heart: air embolus
- Bone cement implantation: fat embolus (See “Bone Cement Implantation Syndrome,” Page 130.)
- Hemorrhagic or cardiogenic shock: low cardiac output state

Further Reading

Eisenkraft J, Leibowitz A. Hypoxia and equipment failure. In: F Yao (ed.). *Anesthesiology: Problem-Oriented Patient Management*. 5th ed. New York: Lippincott Williams & Wilkins; 2003:1116–1136.

Difficult Controlled Ventilation

Definition

Inability to effectively oxygenate and/or ventilate a patient who is mechanically ventilated.

Presentation

High peak airway pressures and hypercarbia are observed with difficult controlled ventilation. Patients are often hypoxemic and at risk for hemodynamic compromise secondary to increased intrathoracic pressures that may decrease venous return.

Etiology

- High peak airway pressures result from poor compliance of the lung parenchyma and increased resistance to airflow due to:
 - Mucous plug
 - Bronchospasm
 - Pulmonary edema
 - Autopeep
 - Pneumothorax
- Elevated plateau pressure (the pressure applied to the small airways and the alveoli during positive pressure) suggests poor lung compliance.
- Low peak airway pressures and loss of measured tidal volumes may indicate an air leak in the circuit or in the tracheobronchial tree (i.e., a bronchopulmonary fistula).

Immediate Management

- Increase FiO_2 to 100%.
- Auscultate breath sounds.
- Begin manual ventilation.
- Suction the endotracheal tube.
- Administer a bronchodilator if bronchospasm is suspected. (albuterol 2–4 puffs into the ETT).
- Administer a diuretic if pulmonary edema is present (furosemide 20 mg IV).
- Exclude machine failure.
- Increase sedation and consider neuromuscular blockade if necessary.

DIFFERENTIAL DIAGNOSIS

- Increased resistance to flow
 - Bronchospasm
 - Obstruction
 - Kinked endotracheal tube
 - Mucus plug
- Decreased lung compliance
 - Inadequate muscle relaxation
 - Tension pneumothorax
 - Autopeep
 - Acute lung injury/acute respiratory distress syndrome
 - Pulmonary edema

- Aspiration
- Opioid-induced chest wall rigidity
- Pulmonary hemorrhage
- Intra-abdominal hypertension/abdominal compartment syndrome
- High insufflation pressures in laparoscopic surgery

Diagnostic Studies

- Measure peak airway pressures
- Measure plateau pressure
- Chest X-ray

Subsequent Management

- Decrease tidal volume to limit volutrauma.
- Increase respiratory rate to ensure adequate minute ventilation.
- Monitor for evidence of autopeep (sudden hypotension, clinical evidence of “breath stacking”).
- Consider CT scan of the chest, if underlying cause is unknown.
- Treat the underlying cause of decreased compliance.
- If conventional mechanical ventilation is inadequate, consider high frequency oscillatory ventilation (HFOV), airway pressure release ventilation (APRV), or extracorporeal membrane oxygenation (ECMO).

Risk Factors

- Patients with COPD are at risk for autoPEEP
- Patients with diffuse pulmonary injury (infection, sepsis) may develop ARDS as their disease progresses.

Prevention

- Maintain adequate sedation.
- Consider use of neuromuscular blocking agents if necessary.
- Monitor for signs of autoPEEP.

Special Considerations

Patients who are difficult to ventilate may have increased peak airway pressures and high levels of PEEP and often require transport to or from an ICU. The anesthesiologist must be familiar with the patient's mode of ventilation in order to ensure a safe transport. ABG samples can guide changes in ventilator strategy in the operating room.

Further Reading

Schmidt G. Ventilator waveforms: clinical interpretation. In: JB Hall, G Schmidt, LDH Wood (eds.). *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:427–443.

Hemoptysis

Definition

Cough productive of blood or bloody sputum. Massive hemoptysis is the production of 300–600 cc of blood in a 12–24 hour period.

Presentation

Reduced breath sounds. In an intubated patient, blood may appear in the endotracheal tube. Diffuse pulmonary infiltrates on chest X-ray. Hypoxia with increased peak inspiratory pressures with positive pressure ventilation may occur with massive hemoptysis. Coagulopathy can trigger hemoptysis, and anemia may be present. Hemoptysis may be the presenting symptom for pulmonary infection or malignancy.

Pathophysiology

Disruption of the pulmonary vessels lining the trachea, bronchi or alveoli. Disruption can occur in the setting of infection, tumor, vascular disorders and trauma. Coagulopathy may exacerbate these conditions.

DIFFERENTIAL DIAGNOSIS

- Nasal trauma
- Pharyngeal trauma
- Gastrointestinal bleeding

Immediate Management

- Increase FiO_2 to maintain oxygenation.
- Inspect the nose and pharynx to exclude an upper-airway source of bleeding.
- Consider bronchoscopy to identify bleeding site.
- Consider placement of a double lumen endotracheal tube or bronchial blocker to isolate the bleeding site.
- For life-threatening hemoptysis, consider endobronchial ablation, bronchial artery embolization, external beam irradiation, or surgical resection.
- Consider the presence of a gastrointestinal source of bleeding.

Diagnostic Studies

Chest CT and bronchoscopy may localize the source of pulmonary bleeding.

Subsequent Management

- Identify and treat the cause of hemoptysis.
- Identify and treat the cause of coagulopathy.
- Consider a surgical consultation if bronchial embolization is unsuccessful or multiple bleeding vessels are seen with angiography.
- Consider a course of steroids in patients with vasculitis.
- Consider plasmapheresis in patients with Goodpasture syndrome.

Risk Factors

- Infection
- Tumor
- Pulmonary vascular abnormalities
- Cardiac causes (e.g., mitral stenosis)
- Trauma
- Coagulopathy
- Pulmonary-renal syndromes (Goodpasture syndrome)

Prevention

- Avoid unnecessary instrumentation of the airway.
- Correct underlying coagulopathy. (See “Coagulopathy”)

Special Considerations

Frequent suctioning of the endotracheal tube may cause hemoptysis.

Further Reading

- Albert R. Massive hemoptysis. In: JB Hall, G Schmidt, LDH Wood (eds.). *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:583–585.
- Sakr L, Dutau H. Massive Hemoptysis: An Update on the Role of Bronchoscopy in Diagnosis and Management. *Respiration*. 2010 Jan 8.

Hypercarbia (Intraoperative)

Definition

Increased arterial partial pressure of carbon dioxide ($\text{PaCO}_2 > 45$ mmHg)

Presentation

Tachycardia, agitation, hypertension, and eventually obtundation.

Etiology

Hypercarbia is caused by hypoventilation or increased CO_2 production. Hypoventilation due to decreased respiratory drive or airway obstruction in sedated patients often leads to hypercarbia. Poor lung compliance may reduce minute ventilation and cause hypercarbia. Residual anesthetic effects or inadequate reversal of muscle relaxants can cause postoperative hypercarbia. Splinting due to pain can lead to increased dead space, hypoventilation and hypercarbia. Hypermetabolic states and fever may contribute to increased CO_2 production.

Immediate Management

- Intubate the trachea and initiate mechanical ventilation if necessary.
- Increase minute ventilation to reduce PaCO_2 .
- Ask the surgeon to lower insufflation pressure during laparoscopic surgery.
- In a spontaneously breathing patient, consider judicious reversal of opioids with naloxone. (Naloxone 0.04 mg IV increments)

DIFFERENTIAL DIAGNOSIS

- Hypoventilation
 - Low minute ventilation
 - Narcotics or oversedation
 - Inadequate reversal of muscle relaxant
 - Splinting
- Malignant hyperthermia (see “Special Considerations” below)
- Bronchospasm (COPD or asthma exacerbation)
- Acute lung injury, acute respiratory distress syndrome
- Severe pneumonia
- Aspiration
- Shivering
- Sepsis
- CO_2 insufflation during laparoscopy
- Bicarbonate administration
- Thyrotoxicosis

Diagnostic Studies

Arterial blood gas analysis (ABG) to quantify degree of hypercarbia and acidosis.

Subsequent Management

- Treat the underlying cause of hypercarbia
- Endotracheal intubation or noninvasive positive pressure ventilation (CPAP, BiPap) if necessary

Risk Factors

- Laparoscopic surgery (insufflation of peritoneal cavity with CO₂)
- Obesity
- Obstructive sleep apnea (OSA)
- Chronic CO₂ retainers
- COPD
- Asthma
- Poor lung compliance
- Narcotic administration

Prevention

- Judicious use of narcotics and other sedatives
- Adequate reversal of muscle relaxants
- Adequate minute ventilation, especially in laparoscopic surgery

Special Considerations

- Hypercarbia causes respiratory acidosis that cannot be compensated for in the acute period. Hypercarbia may cause severe hypertension, hyperkalemia, arrhythmias, myocardial depression, altered mental status, increased intracranial pressure and increased pulmonary vascular resistance.
- **Rapidly rising ETCO₂ in conjunction with tachycardia and rising temperature may be caused by malignant hyperthermia.** MH must be diagnosed quickly and treatment initiated immediately. (See “Malignant Hyperthermia,” Page 114.)

Further Reading

Lane J. Postoperative respiratory insufficiency. In: JL Atlee (ed.). *Complications in Anesthesia*. 2nd ed. Philadelphia: Saunders Elsevier; 2007:877–880.

Hypoxemia (Intraoperative)

Definition

Decreased partial pressure of oxygen in the blood ($\text{PaO}_2 < 60$ mmHg) often manifested by a decrease in SpO_2 .

Presentation

Decreased SpO_2 , cyanosis, and possibly hypertension and agitation. Left untreated, hypoxemia may progress to hypotension, bradycardia, arrhythmias, and neurological and myocardial ischemia.

Etiology

- Oversedation and/or narcotic overdose can cause hypoventilation and airway obstruction in patients undergoing surgery with monitored anesthetic care (MAC).
- Decrease in functional residual capacity (FRC)
- Position (supine position decreases FRC)
- Atelectasis and alveolar shunting
- Blunted hypoxic pulmonary vasoconstriction (may be caused by inhaled anesthetics)
- Ventilation perfusion mismatching
- Intrapulmonary shunt due to mainstem bronchus intubation
- Anesthesia machine malfunction

Immediate Management

- Increase FIO_2 to 100% while assessing patient.
- Ascultate the lung fields to assess breath sounds.
- Check ETCO_2 to ensure that the ETT is in the trachea.
- If the patient is not already intubated, consider intubation and mechanical ventilation for severe hypoxia or if respiratory failure is imminent.

DIFFERENTIAL DIAGNOSIS

- Esophageal intubation
- Mechanical disconnect from ventilator or O_2 source
- Right mainstem intubation
- Airway obstruction
- Hypoventilation
- Atelectasis
- Presence of a mucus plug
- Bronchospasm

- Pneumothorax
- Pulmonary embolus
- Pulmonary edema
- Acute lung injury
- Aspiration
- Low cardiac output state

Diagnostic Studies

- Arterial blood gas analysis (ABG) to quantify PaO₂
- Chest X-ray
- Consider bronchoscopy (assess ETT placement, find a possible obstruction).

Subsequent Management

- Administer supplemental O₂ to maintain oxygenation.
- Treat the underlying cause of hypoxemia.
- Prepare for endotracheal intubation and mechanical ventilation.

Risk Factors

- Underlying pulmonary disease
- Obstructive sleep apnea (OSA)
- Aspiration risk
- Use of narcotics
- Advanced age
- Obesity
- Shivering

327

Prevention

- Confirm ETT placement with capnography and auscultation.
- Use narcotics and sedatives carefully in patients who are breathing spontaneously.
- Ensure that the patient is adequately ventilated.

Special Considerations

Intraoperative hypoxemia is one of the most common problems that an anesthesiologist encounters, and should be considered life threatening. Prompt diagnosis and treatment are essential to preventing further complications, i.e., hypotension, arrhythmias and end organ damage.

Further Reading

Lane J. Postoperative respiratory insufficiency. In: JL Atlee (ed.). *Complications in Anesthesia*. 2nd ed. Philadelphia: Saunders Elsevier; 2007:877–880.

Pneumothorax

Definition

Presence of gas, usually air, in the pleural cavity that leads to collapse of the lung. This condition may be life threatening if the gas cannot escape (i.e., tension pneumothorax).

Presentation

A small pneumothorax is often asymptomatic. As the pneumothorax becomes larger, hypoxia, tachypnea, tachycardia, and chest pain may occur. It may be possible to hear hyperresonance on the affected side with percussion. Decreased or absent breath sounds may also be heard on the affected side. Increased peak airway pressures and plateau pressures occur in mechanically ventilated patients. A tension pneumothorax is often associated with hypotension.

Pathophysiology

A tension pneumothorax occurs when a one-way valve mechanism occurs after injury to the pleural space. With each inspiration, gas is trapped in the pleural space causing collapse of the lung. If intrapleural pressure increases significantly, mediastinal shift causes kinking of major veins at the thoracic inlet of the neck and inferior vena cava, resulting in decreased venous return and hypotension.

Immediate Management

- Increase FIO_2 to 100%.
- Decompress the pleural space with a large bore needle in the mid-clavicular line in the 2nd intercostal space (see Figure 13.1).
- Insert a chest tube.

DIFFERENTIAL DIAGNOSIS

- Hemothorax
- Mucus plug
- Endobronchial intubation
- Severe bronchospasm

Diagnostic Studies

- Auscultation (absent breath sounds on the affected side)
- Percussion (hyperresonance).
- Chest X-ray (lung collapse and mediastinal shift)

Subsequent Management

- Bronchoscopy if bronchial tree injury is suspected

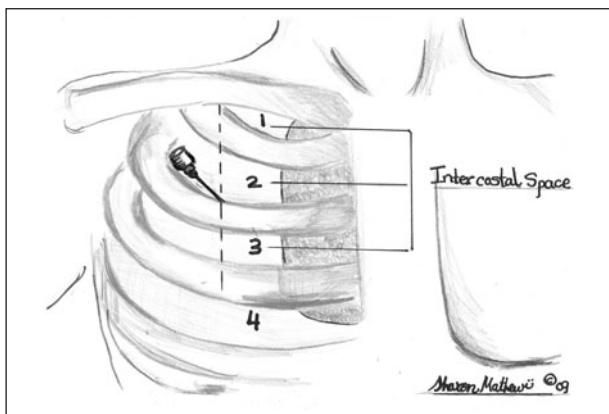


Figure 13.1 Decompression of tension pneumothorax.

- Surveillance chest X-ray to evaluate progression of pneumothorax
- Chest tube management

Risk Factors

- Central line placement
- Laparoscopic surgery
- Excessive tidal volumes or peak airway pressures
- Blebs

Prevention

- Avoid excessive tidal volume or peak airway pressure.
- Chest X-ray or imaging after central line placement to prevent progression of a small pneumothorax to a tension pneumothorax.

Special Considerations

Tension pneumothorax should be considered in patients who develop a pulseless electrical activity (PEA) cardiac arrest.

Further Reading

Ali J. Torso Trauma. In: JB Hall, G Schmidt, LDH Wood (eds.). *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:1421–1441.

Pulmonary Edema

Definition

The abnormal accumulation of extravascular fluid in the lung parenchyma.

Presentation

The first signs of pulmonary edema in an anesthetized patient are often hypoxemia and decreased SpO₂. Rales or wheezing are heard over the lung fields. Frothy sputum may be noted in the endotracheal tube. In an awake patient, respiratory distress, tachycardia and agitation. Jugular venous distension may be seen on physical examination.

Pathophysiology

- Occurs due to high pulmonary and venous hydrostatic pressure (cardiogenic) or increased capillary permeability (non-cardiogenic).
- Cardiogenic pulmonary edema is caused by impaired venous drainage from the pulmonary vasculature to the left atrium. This often occurs when the left atrial pressure is high in the setting of left ventricular dysfunction and/or valvular abnormalities.
- Negative pressure or postobstruction pulmonary edema occurs when negative intrapleural pressure increases the pulmonary hydrostatic pressure gradient, causing fluid to move from the pulmonary vasculature to the interstitium.

Immediate Management

- Increase FIO₂ to 100%.
- Initiate diuresis (start with furosemide 20 mg IV).
- Intubate the trachea and begin positive pressure ventilation if the patient is hypoxic or respiratory failure is imminent.
- If cardiogenic pulmonary edema is suspected, consider afterload reduction with nitroglycerine (Infusion starting at 0.5 mcg/kg/min) and support blood pressure with vasopressors. (See Congestive Heart Failure, in chapter 2, Page 31)
- Treat the underlying cause.

DIFFERENTIAL DIAGNOSIS

- Aspiration pneumonitis
- Acute lung injury/ARDS
- Neurogenic pulmonary edema
- Aspiration pneumonitis

- Fat embolism
- TACO (transfusion associated circulatory overload)
- Transfusion Related Acute Lung Injury (TRALI)

Diagnostic Studies

- Chest X-ray (bilateral pulmonary infiltrates and edema around pulmonary arteries)
- Pulmonary artery catheter or echocardiogram can differentiate between cardiogenic and noncardiogenic pulmonary edema.

Subsequent Management

- Ventilatory support including PEEP
- Serial arterial blood gas measurements.
- Central venous pressure monitoring may aid in medical management, i.e. diuresis.

Risk Factors

- Cardiogenic
 - Systolic dysfunction
 - Diastolic dysfunction
 - Volume overload
 - Myocardial Infarction
 - Valvular abnormalities
- Negative Pressure
 - Laryngospasm
 - Upper airway obstruction
 - Upper airway tumor or foreign body
 - Tonsillar hypertrophy

Prevention

- Avoid fluid overload in a patient with compromised myocardial function.
- Ensure adequate perfusion pressure and avoid tachycardia in patients with coronary artery disease.
- Identify patients at risk for airway obstruction.

Special Considerations

Negative pressure pulmonary edema often resolves within 24 hours. Cardiogenic pulmonary edema may occur 2–3 days postoperatively when fluids are mobilized.

Further Reading

Ali J. Special considerations in the surgical patient. In: JB Hall, G Schmidt, LDH Wood (eds.). *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:1321–1330.

Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, et al: 2009 Focused update incorporated into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines Developed in Collaboration With the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol*. 2009 Apr 14;53(15):e1–e90.

Pulmonary Embolus

Definition

Obstruction of a pulmonary artery or one of its branches, most commonly by a venous thrombus that becomes dislodged and eventually travels to the lungs. Pulmonary embolus may also be caused by fat, air, carbon dioxide or amniotic fluid emboli.

Presentation

The signs and symptoms may be subtle. Small emboli may go undetected; unexplained fever, tachycardia, and rales may be the only presenting symptoms. Some patients may present with a triad of dyspnea, hemoptysis, and chest pain. Patients with large emboli may present with sudden hypoxia, hypercarbia, tachypnea, decreased ETCO_2 , and circulatory collapse.

Pathophysiology

Pulmonary thromboembolism occurs when a venous thrombus is dislodged and travels to the lungs. This occurs most commonly in the setting of venous stasis or injury, and most thrombi originate in the lower extremity deep vein system. Pulmonary embolism can also be caused by air, fat, carbon dioxide or amniotic fluid emboli. Gas exchange becomes impaired as dead space increases. A widened alveolar to arterial gradient is often seen. Right ventricular afterload increases.

DIFFERENTIAL DIAGNOSIS

- Thromboembolism
- Air embolism
- Fat embolism
- Amniotic fluid embolism
- Acute myocardial infarction
- Severe bronchospasm

- Anaphylaxis
- Pneumothorax

Immediate Management

- Increase FIO_2 to 100% to maintain oxygenation.
- Consider intubation and mechanical ventilation if hypoxia is severe.
- Support circulation with fluid, vasopressors, and inotropes.
- Consider right ventricular afterload reduction with nitric oxide.
- Begin systemic anticoagulation if not contraindicated and a thromboembolism is diagnosed. (Heparin 80 units/kg IV bolus, then 18 units/kg/h IV, titrated to therapeutic INR)
- *Consider thrombolytic therapy or embolectomy in the setting of hemodynamic instability.

Diagnostic Studies

- Helical chest computerized tomography angiography
- D-dimer is of limited value, as levels are often elevated in the perioperative setting.
- Ventilation-perfusion scan
- Noninvasive venous doppler studies to assess for deep vein thrombosis
- Echocardiography to evaluate for right ventricular dilation or strain

Subsequent Management

- Identify the source of the embolism.
- Continue anticoagulation if indicated.
- Consider inserting an inferior vena cava filter if anticoagulation is contraindicated.

Risk Factors

- Thromboembolism
 - Malignancy
 - Surgery and trauma
 - Immobility
 - Pregnancy
 - Hypercoagulable states
 - Obesity
 - Indwelling central lines

Risk Factors (continued)

- Other embolic events
 - Sitting craniotomy (air embolus)
 - Laparoscopic surgery (CO₂ embolus)
 - Hip replacement (fat embolus)

Prevention

- Intermittent compression stockings
- Subcutaneous heparin in high-risk patients
- Early mobilization after surgery

Special Considerations

- The decision to administer anticoagulants or thrombolytics may be complicated in patients who recently underwent surgery, and must take into consideration the risk of postoperative bleeding
- Brain natriuretic peptide levels predict right ventricular dysfunction and mortality.
- Pulmonary embolus should be considered in patients with PEA.

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Respiratory Precautions

Bioterrorism, SARS, multi-drug resistant tuberculosis, and H1N1 influenza have brought new concerns to the health care provider, and especially the anesthesiologist. Anesthesiologists must provide rapid and appropriate care when managing an airway, while at the same time making sure to protect themselves from communicable diseases.

The CDC recommends use of N95 respirator masks while caring for patients with suspected H1N1. The N95 mask is tighter-fitting than a traditional face mask. (Figure 13.2) An N95 respirator covers the nose and mouth and is designed to have a tight fit. If worn correctly, it should filter out at least 95 percent of particles as small as 0.3 micrometers.

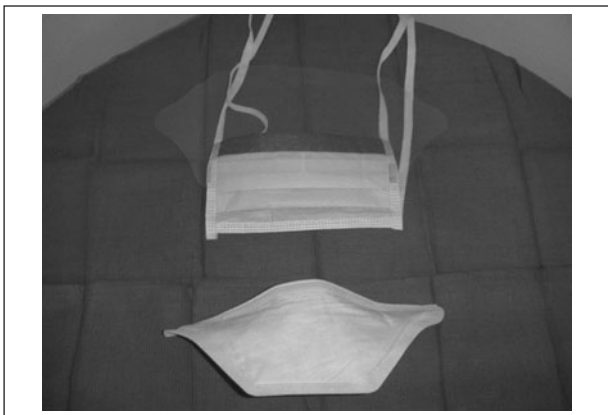


Figure 13.2 Standard fluid shield mask (top) and N95 respirator (bottom).

The use of eye protection in the form of fluid shields or goggles is also recommended to protect against contact with sputum, gastric contents or other bodily fluids while securing a patient's airway. Additionally, wearing a gown may further protect the anesthesiologist, as well as other patients.

If a bioterrorism strike is suspected, gown, gloves, N95 respirators and fluid shields should be worn. Immediately contact local authorities and the CDC for likely pathogens and take further proper precautions, including potentially a splash protective suit and a self-contained breathing apparatus.

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Chapter 14

Surgical Emergencies

Linda L. Maerz and Stephen M. Luczycki

- Bleeding after Carotid Endarterectomy 338
- Bleeding after Thyroid Surgery 340
- Facial Trauma 342
- Laparotomy in the Critically Ill Patient 344
- Massive Hemorrhage 347
- Neck Injury 350
- Ruptured Abdominal Aortic Aneurysm 352
- Ruptured Ectopic Pregnancy 355
- Upper GI Bleeding 358

Bleeding after Carotid Endarterectomy

Definition

Bleeding from the carotid artery, internal jugular vein, smaller vessels or raw tissue after carotid endarterectomy (CEA). Postoperative hemorrhage is rare (occurs in less than 1%–4%).

Presentation

- In the awake patient, anxiety may be the first symptom of early airway compromise.
- Neurologic symptoms may rarely occur as a result of compression or disruption of the internal carotid artery. Tachycardia and tachypnea may occur, possibly due to anxiety. Decreased oxygen saturation is a late occurrence. Hypotension due to exsanguination from disruption of the suture line occurs rarely.
- New onset of dysphonia and stridor are diagnostic of airway compromise.
- An expanding hematoma may develop, which may or may not be pulsatile.

Anatomy and Pathophysiology

- Sites of operative hemorrhage after CEA include both venous and arterial bleeding at sites remote from the carotid artery itself. Diffuse microvascular bleeding from residual heparin effect and/or effects of anti-platelet agents also occurs. The suture line of the endarterectomy site is another potential source. In those instances in which vein-patch angioplasty is employed, vein patch rupture may occur. Arterial bleeding may result in exsanguinating hemorrhage.
- An acute neck hematoma exerts pressure on the larynx and may cause life-threatening airway compromise.

Immediate Management of Acute Respiratory Distress

- Increase FiO_2 to 100%.
- Elevate the head of the bed.
- Immediate re-exploration is mandatory. Even a hematoma that initially appears benign can rapidly progress and compromise the airway. Transport the patient to the operating room if the clinical situation permits.
- It may be necessary to evacuate the hematoma first in order to accomplish endotracheal intubation.
- Rapid establishment of a surgical airway may rarely necessary if endotracheal intubation is not possible in spite of hematoma evacuation.

DIFFERENTIAL DIAGNOSIS

- Anaphylaxis
- Laryngeal edema
- Acute pulmonary edema
- Tension pneumothorax
- Acute coronary syndrome
- Pulmonary embolus

Diagnostic Studies

- This is primarily a clinical diagnosis. Physical examination and a high index of suspicion are critical.
- Because the patient can deteriorate rapidly, diagnostic studies (i.e., imaging) are not usually feasible.
- In more subtle hemorrhage without acute airway compromise, carotid duplex ultrasound and diagnostic cervical ultrasound may be used to establish the diagnosis.

Subsequent Management

- After a definitive airway is established, transport the patient to the OR for exploration of the operative site.
- Be prepared for a major vascular operation, including carotid isolation and repair.

Risk Factors

- Surgical technique
- Perioperative hypertension
- Perioperative use of aspirin or other anti-platelet agents in the perioperative period
- Failure to reverse heparin with protamine sulfate
- Postoperative anticoagulation with heparin or warfarin
- The use of a small vein may increase the risk of rupture after a vein patch procedure.

Prevention

- Meticulous hemostasis at the time of the original operation
- Consider reversal of heparin that was administered during the procedure.
- The incidence of vein patch rupture can be minimized by using greater saphenous vein harvested from the thigh.

Special Considerations

This complication is potentially life-threatening because of associated airway compromise or, rarely, exsanguination from arterial suture line disruption.

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Bleeding after Thyroid Surgery

Definition

Bleeding from the raw thyroid surface, dissected muscles, or blood vessels after thyroid surgery.

Presentation

- In the awake patient, anxiety may be the first symptom of early airway compromise.
- Tachycardia and tachypnea due to anxiety or airway compromise may occur. Decreased oxygen saturation is a late occurrence.
- New onset of dysphonia and stridor indicate airway compromise.
- A new neck mass in proximity to the operative site, which may or may not be associated with bleeding from the incision, represents a hematoma until proven otherwise.

Pathophysiology

- Sources of postoperative hemorrhage after thyroid surgery include the raw surfaces of residual thyroid tissue, venous bleeding and arterial bleeding. The bleeding may originate from vessels deep or superficial to the cervical strap musculature. When an acute hematoma occurs in the neck, the resulting pressure results in compression of the larynx, which can lead to life-threatening airway compromise.
- Up to 3% of patients with thyroid diseases undergoing thyroid surgery have various acquired abnormalities of coagulation. The most common abnormality resembles von Willebrand's disease. Patients who have hypothyroidism are the most likely to have an abnormal bleeding tendency, but coagulopathy may also occur in patients with thyroid malignancies.

DIFFERENTIAL DIAGNOSIS

- Anaphylaxis
- Laryngeal edema
- Acute pulmonary edema
- Tension pneumothorax
- Acute coronary syndrome
- Pulmonary embolus

Immediate Management for Acute Respiratory Distress

- Administer 100% FiO_2
- Immediate surgical exploration and evacuation of the hematoma is mandatory. If possible, transport the patient to the operating room unless the airway compromise requires immediate management.
- After the hematoma is evacuated and laryngeal anatomy is restored, endotracheal intubation is usually possible.
- A surgical airway may rarely be necessary if endotracheal intubation is not possible after hematoma evacuation.

341

Diagnostic Studies

- Physical examination and a high index of suspicion.
- The urgency of the situation precludes imaging or other diagnostic studies.

Subsequent Management

After a definitive airway is established, exploration of the operative site is performed and the source of hemorrhage is controlled.

Risk Factors

- Surgical technique is most commonly responsible.
- Extensive surgery
- Underlying bleeding diathesis or the use of anticoagulants (e.g., heparin, warfarin, clopidogrel)
- Male gender
- Advanced age

Prevention

Meticulous hemostasis at the time of surgery. Preoperative coagulation screening to identify patients at increased risk of bleeding. Early detection and treatment of reversible coagulopathy.

Special Considerations

Although bleeding and wound hematomas have an occurrence rate of <1%, these complications may be life-threatening due to airway compromise.

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Facial Trauma

Definition

Maxillofacial injuries may be dramatic in appearance but are rarely life-threatening, unless airway, breathing, or circulation are compromised as a result of the injury.

Presentation

- Compromise of airway and breathing:
 - Airway obstruction may result in early death in the setting of multiple mandibular fractures or the combination of nasal, maxillary and mandibular fractures.
 - Aspiration of teeth, blood, vomitus, or foreign bodies can cause airway obstruction.
 - Signs of impending respiratory obstruction include stridor, cyanosis, drooling or ineffective gag reflex.
- Compromise of circulation resulting in hemorrhage:
 - Epistaxis
 - Scalp lacerations
 - Tongue lacerations
 - LeFort fractures

Anatomy and Pathophysiology

Blunt trauma may cause panfacial fractures, significant soft tissue injury and multiple remote sites of injury. Penetrating trauma ranges from simple lacerations to high velocity missile injuries that may also involve the brain or neck.

ASSOCIATED INJURIES

- Airway obstruction
- Brain injury
- Cervical spine injury
- Hemorrhage from other sites

Immediate Management

- Secure the airway: orotracheal intubation if needed; surgical airway if orotracheal intubation is not possible. Nasotracheal intubation is contraindicated.
- Maintain cervical spine stabilization during airway management.
- Local control of hemorrhage.
- Establish large-bore peripheral intravenous access.

Diagnostic Studies

CT scan is the standard of care. Panorex films are a useful adjunct for defining mandible fractures.

Subsequent Management

- Management of associated life-threatening injuries is undertaken first.
- Early tracheostomy should be considered in selected patients as follows:
 - pan-facial fractures
 - profuse nasal bleeding
 - severe soft tissue edema in the proximity of the airway
 - patients with altered mental status
 - severe facial burns
 - high spinal cord injuries
 - difficult airway characteristics
 - need for prolonged intubation
- Assume that the cervical spine is unstable. A cervical collar should remain in place until definitive clearance by a combination of physical examination and radiographic examination.
- Definitive management of facial injuries, particularly facial fractures, usually occurs in a delayed timeframe.

Prevention

Maxillofacial injuries themselves are seldom life-threatening. Associated injuries, however, are serious and must be managed first in order to prevent loss of life.

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Laparotomy in the Critically Ill Patient

Definition

Indications for laparotomy in the critically ill include life-threatening infection, hemorrhage, ischemia, and abdominal compartment syndrome.

Presentation

- Signs and symptoms vary with etiology and indication for laparotomy.
- Shock (hemorrhagic, septic or traumatic) is usually present.
- Symptoms may include signs of impaired end-organ perfusion, including altered mental status, oliguria, tachypnea, hyperlactacidemia and increased base deficit.
- Significant tissue hypoperfusion can occur in spite of a normal arterial blood pressure. The presence of hypotension implies severe physiologic decompensation.

Pathophysiology

- Shock is defined as the inadequate delivery of oxygen and nutrients to tissues, a result of hypoperfusion. Shock is characterized in various ways based on etiology. A clinically useful scheme is as follows:
 - Hypovolemic/hemorrhagic (acute hemorrhage of any etiology, peritonitis, pancreatitis, bowel obstruction)
 - Vasodilatory/distributive (sepsis, adrenal insufficiency, high spinal cord injury, liver failure, anaphylaxis)
 - Cardiogenic (myocardial infarction, tamponade, arrhythmias)
 - Obstructive (pulmonary embolus, pneumothorax)
 - Traumatic (a combination of hemorrhage, ischemia, reperfusion, activation of proinflammatory cascades)
- Persistent hypothermia and progressive metabolic acidosis in the setting of massive transfusion are associated with life-threatening coagulopathy.

DIFFERENTIAL DIAGNOSIS

- Cardiovascular collapse from extra-abdominal source of sepsis
- Hemorrhage from extra-abdominal trauma (pelvic fractures, long-bone fractures, thoracic injuries, open extremity wounds, scalp lacerations)
- Massive myocardial infarction with acute cardiac failure and cardiovascular collapse
- Massive pulmonary embolus with cardiovascular collapse

Immediate Management

- Intubate the trachea and initiate mechanical ventilation.
- Increase FiO_2 to maintain adequate oxygenation.
- Establish large-bore peripheral IV access or central venous access.
- Begin aggressive resuscitation with IV fluids. Transfuse with packed red blood cells (PRBCs) if indicated (i.e., for hemorrhagic shock)
- Promptly identify and correct coagulopathy, thrombocytopenia, and platelet dysfunction
- Support blood pressure with vasopressors if indicated (septic shock).
- Consider epinephrine infusion starting at 0.03–0.05 mcg/kg/min.
- Begin broad-spectrum empiric or culture-directed antibiotic therapy (septic shock)

Diagnostic Studies

- Abdominal and pelvic CT scan is the standard for diagnosis of intra-abdominal catastrophes.
- If the patient is too unstable for CT scan, emergency surgery may be indicated based on a diagnosis made from the clinical scenario and physical examination.
- In the unstable trauma patient, focused abdominal sonography for trauma (FAST) and diagnostic peritoneal lavage can be expeditiously performed in the trauma bay to confirm intra-abdominal hemorrhage.

Subsequent Management

- Communication with the surgical team is essential.
- Anesthetic management should focus on maintaining oxygenation and perfusion. Induction agents are chosen based on the patient's volume status and medical condition. Consider etomidate (0.3 mg/kg IV) if the patient is hemodynamically unstable. Sympathomimetic agents (e.g., ketamine) may cause profound hypotension in critically ill patients who have high circulating

levels of endogenous catecholamines (hypovolemia from sepsis or hemorrhage; myocardial dysfunction; pain). Hypotension is exacerbated by the transition from spontaneous ventilation to positive pressure mechanical ventilation.

- Instability during the surgical procedure is most likely due to surgical manipulation and the patient's underlying pathophysiology. A narcotic based technique is associated with less vasodilation and negative inotropy and may be appropriate for hemodynamically unstable patients and those with underlying myocardial dysfunction. Pay close attention to the patient's volume status, body temperature (fluid warmer, forced hot air blanket, elevated room temperature, humidification of the mechanical ventilation circuit), and prevention of positioning injuries.
- Patients undergoing emergency laparotomy typically emerge from anesthesia more slowly and are more likely to have hemodynamic instability in the postoperative period. Most patients remain intubated and mechanically ventilated after surgery to allow for delayed emergence and possible subsequent extubation. Depending on the degree of physiologic impairment, many patients will require prolonged mechanical ventilation and ongoing resuscitation in the ICU.

Risk Factors

- The lethal triad: hypothermia, acidosis, coagulopathy
- Advanced age
- Chronic illness; comorbidities

Prevention

Early diagnosis of impending intra-abdominal catastrophe prior to development of shock may prevent significant hypoperfusion, associated end organ dysfunction, complications, and death.

Special Considerations

- Abdominal compartment syndrome:
 - Abdominal hypertension with associated end organ dysfunction (most commonly oliguria and elevated peak airway pressures)
 - Bladder pressure is used to assess intra-abdominal pressure.
 - Etiologies include blunt and penetrating abdominal trauma, pelvic fractures, severe burns, massive resuscitation, and ischemia-reperfusion of the abdominal viscera.
 - Treatment is abdominal decompression, typically via decompressive laparotomy, leaving the abdomen open.

- Mortality rates of 60%–70% reflect delayed diagnosis and underlying pathophysiology.
- Early decompression, prior to irreversible end organ ischemia and cardiovascular collapse, is essential.
- Damage control surgery:
 - Damage control is the preferred management strategy for trauma patients with abdominal injuries complicated by hypothermia, coagulopathy and acidosis. This strategy can also be used for critically ill nontrauma patients.
 - Limited initial operation for the immediate control of hemorrhage and contamination; the abdomen is left open with a temporary abdominal closure in place.
 - Ongoing resuscitation and correction of physiologic derangements occurs in the ICU.
 - Subsequent operation for correction of anatomic abnormalities occurs in the stabilized patient.

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Massive Hemorrhage

Definition

- Major categories of massive hemorrhage include traumatic, gastrointestinal and obstetric.
- Classes of hemorrhagic shock:
 - Class I: blood loss up to 750 cc (up to 15% of blood volume)
 - Heart rate <100
 - Blood pressure normal
 - Pulse pressure normal or increased
 - Class II: blood loss 750–1500 cc (15–30% of blood volume)
 - Heart rate >100
 - Blood pressure normal
 - Pulse pressure decreased

- Class III: blood loss 1500–2000 cc (30–40% of blood volume)
 - Heart rate >120
 - Blood pressure decreased
 - Pulse pressure decreased
- Class IV: blood loss >2000 cc (>40% of blood volume)
 - Heart rate >140
 - Blood pressure decreased
 - Pulse pressure decreased

Presentation

- Symptoms depend on the etiology of the hemorrhage. Gastrointestinal blood loss is typically painless, while blood loss associated with trauma is associated with pain due to the injury.
- Perturbation of vital signs depends on the degree of blood loss.
- Severe shock is notable for cool, moist, pallid or cyanotic skin. Mental status changes progress from anxiety to confusion to lethargy as the degree of shock progresses. Tachypnea progresses as the patient's spontaneous minute ventilation increases in order to meet increased metabolic demands. Oliguria is caused by renal hypoperfusion.
- The source of hemorrhage may or may not be visible on external examination.

Pathophysiology

Hemorrhagic shock is a complex spectrum of events:

- Acute massive blood loss resulting in circulatory collapse
- Ischemia-reperfusion injury
- Inflammatory and anti-inflammatory responses
- Multiple organ dysfunction

Immediate Management

- Intubate the trachea and initiate mechanical ventilation.
- Establish large-bore peripheral and central IV access.
- Transfuse with packed red blood and factors cells as indicated. Consider activating the massive transfusion protocol.
- Interstitial resuscitation with crystalloid solutions (e.g., lactated Ringer's solution or normal saline solution)
- If laboratory studies are not feasible, current recommendations for massive transfusion therapy in traumatic injury include minimal use of crystalloid solutions and use of plasma, PRBCs and platelets in a 1:1:1 ratio. Administer cryoprecipitate for continued microvascular bleeding.
- If time permits, identify and correct specific deficits with serial PT, PTT, INR, fibrinogen and platelet count.

DIFFERENTIAL DIAGNOSIS

Includes other causes of acute circulatory collapse:

- Hypovolemic shock of nonhemorrhagic etiology (bowel obstruction, pancreatitis)
- Vasodilatory/distributive shock
- Obstructive shock
- Cardiogenic shock

Diagnostic Studies

- *Gastrointestinal source*: endoscopy, interventional radiology, nuclear medicine.
- *Traumatic source*: CT scan, focused abdominal sonography in trauma (FAST), diagnostic peritoneal lavage, immediate operative intervention in hemodynamically unstable patients.
- *Obstetric source*: usually apparent on physical examination or ultrasound examination.

Subsequent Management

- Prompt surgical control of the bleeding is of paramount importance.
- Continue resuscitation postoperatively or post-procedure.
- The goals of resuscitation are optimization of preload, cardiac performance, blood pressure, oxygen delivery and end-organ perfusion. No single parameter is universally applicable to every patient. Therefore, multiple endpoints should be optimized:
 - Clinical endpoints (heart rate, respiratory rate, blood pressure, urine output, level of consciousness, pulse pressure)
 - Cardiac output measurement
 - Metabolic parameters (lactate, base deficit)
 - Regional perfusion (gastric tonometry, sublingual capnography, near-infrared spectroscopy)

Risk Factors

- *GI source*: Advanced age, comorbidities.
- *Traumatic source*: Young age (injury is the leading cause of death for persons younger than 44 years of age in the US) and lifestyle issues.
- *Obstetric source*: Postpartum hemorrhage is largely due to uterine atony.

Prevention

Prevention of shock and resultant hypoperfusion and organ dysfunction is dependent on early control of the bleeding and appropriate resuscitation, regardless of the etiology of the hemorrhage.

Special Considerations

- Massive transfusion is usually defined as the complete replacement of the patient's entire blood volume—10 units PRBC—in a 24-hour period.
- Role of recombinant-activated factor VII (rFVIIa):
 - Approved in the US only for bleeding associated with hemophilia.
 - Significant off-label use has been noted, including for the reversal of the coagulopathy of trauma.
 - Generates a thrombin peak, which in turn leads to formation of a fibrin plug.
 - May not be efficient in acidosis (consider biochemical correction of acidosis prior to administration).
 - Hypothermia has little effect on efficacy.

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Neck Injury

Definition

The neck contains a high density of vital structures located in a small and unprotected area. This may result in multisystem injury as a consequence of a single traumatic event.

Presentation

- “Hard signs” warrant urgent surgical intervention:
 - Active bleeding
 - Expanding or pulsatile hematoma
 - Subcutaneous emphysema or air bubbling from wound
- “Soft signs” warrant a more selective or expectant approach:
 - Dysphagia
 - Voice change

- Hemoptysis
- Wide mediastinum

Anatomy and Pathophysiology

- Structures located in the neck:
 - Sternocleidomastoid muscles
 - Carotid artery and internal jugular vein
 - Pharyngeo-esophageal junction
 - Larynx and proximal trachea
 - Thyroid and parathyroid glands
 - Thoracic duct—enters the jugulosubclavian system in the left neck
 - Cervical vertebra and spinal cord
 - Long cervical musculature (posterior)
- Anterior triangles of the neck: the area between the sternocleidomastoid muscles
- Zones of the neck:
 - Zone I: the area bounded by the cricoid cartilage superiorly, the thoracic inlet inferiorly and the sternocleidomastoid laterally
 - Zone II: the area between the cricoid cartilage and the angle of the mandible
 - Zone III: the area bounded by the angle of the mandible inferiorly and the base of the skull superiorly

ASSOCIATED INJURIES

- Spinal cord injury
- Brain injury
- Facial trauma

Immediate Management

- Secure the airway if it is compromised. Orotracheal intubation is preferred, surgical airway if orotracheal intubation is not possible.
- Maintain cervical spine stabilization during airway management.
- Establish large-bore IV access. If central venous access is required, consider femoral venous cannulation.
- Resuscitation with crystalloid and PRBC if indicated.
- Control active hemorrhage with local application of pressure on the way to the operating room.

Diagnostic Studies

- Selected radiographic and endoscopic studies (see below).
- Immediate surgery in the setting of active hemorrhage.

Subsequent Management

- Formal neck exploration is currently performed in the presence of “hard signs” that indicate major vascular or aerodigestive tract injury.
- Penetrating wounds should not be explored locally. Exploration should only be performed in the operating room as part of a formal neck exploration.
- In the absence of “hard signs,” a selective approach for management can be undertaken. This includes thorough physical examination and specific diagnostic studies to identify vascular and aerodigestive tract injuries:
 - Esophagoscopy
 - Esophagography
 - Laryngoscopy/tracheoscopy
 - Arteriography (conventional and CTA)
 - Doppler ultrasonography
 - CT scan
- Definitive operative management of identified injuries
- Endovascular management approaches are in evolution.

Special Considerations

Immediate protection of the airway and control of exsanguinating hemorrhage prevent loss of life.

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Ruptured Abdominal Aortic Aneurysm

Definition

Rupture is a potentially lethal complication of abdominal aortic aneurysm (AAA).

Presentation

- Most patients who present with a ruptured AAA are unaware that they have aneurysmal disease. Patients may present with a symptomatic aneurysm (pain without evidence of rupture), a contained leak, or free rupture.

- The classic clinical presentation of AAA rupture includes abdominal pain, hemodynamic instability and a pulsatile abdominal mass. The abdominal pain is usually acute, unremitting, and radiates to the back. If the rupture is in the retroperitoneum adjacent to the ureter, pain may be referred to the ipsilateral testicle or groin.
- The patient may experience light-headedness or collapse because of acute hypovolemia.
- If the aneurysm perforates into the duodenum or colon, massive gastrointestinal hemorrhage may occur.
- If rupture occurs directly into the inferior vena cava, high-output congestive heart failure results.

Pathophysiology

Once an aneurysm develops, regardless of the etiology, enlargement is governed by the Law of Laplace, $T=PR$. T is tangential stress (which disrupts the wall of a sphere), P is the transmural pressure, and R is the radius. Large aneurysms are therefore more likely to rupture than small ones.

Immediate Management

- Intubate the trachea if the patient is in severe shock and is unable to protect the airway.
- If the clinical situation permits, transport the patient to the operating room and induce anesthesia after the patient is prepped and draped, because of the possibility of severe hypotension. Establish large-bore peripheral and central venous access.
- Initiate aggressive resuscitation with IV fluids and PRBC.
- Patients who present with the classic triad—pain, hemodynamic instability and a pulsatile abdominal mass—must be transferred immediately to the operating room while being resuscitated. Any delay in control of the hemorrhage may be life-threatening.

DIFFERENTIAL DIAGNOSIS

- Acute infectious or inflammatory abdominal process (hollow viscus rupture, ischemic/infarcted bowel, acute cholecystitis, acute pancreatitis) causing septic shock or exaggerated systemic inflammatory response
- Hemorrhage from another intra-abdominal source, including ruptured visceral artery aneurysm, solid organ rupture (liver, spleen, kidney), hepatobiliary tumor rupture
- Massive gastrointestinal hemorrhage

- Aortic dissection or aortic occlusion
- Massive myocardial infarction with resultant acute cardiac failure, cardiogenic shock and hemodynamic collapse
- Massive pulmonary embolus with resultant obstructive shock and hemodynamic collapse

Diagnostic Studies

- Hemodynamically stable patients should undergo CT of the abdomen and pelvis with intravenous contrast.
- Duplex ultrasound (which may be performed at the bedside) can rapidly determine the presence of an AAA but may not distinguish between a ruptured and a nonruptured AAA.

Subsequent Management

- All patients with ruptured AAA require urgent surgery.
- Hemodynamically unstable patients must undergo immediate surgery. Resuscitation occurs during surgery because operative intervention cannot be delayed.
- Assess and treat coagulopathy, thrombocytopenia and platelet dysfunction. Baseline laboratory studies include CBC, PT, PTT, comprehensive chemistry panel and cardiac enzymes. Coagulopathy is treated with fresh frozen plasma and platelets, as indicated by laboratory parameters.
- In hemodynamically stable patients, timing of repair depends on CT scan results. Emergency surgical management is necessary for a contained peritoneal rupture. In symptomatic patients who do not demonstrate CT evidence of rupture, repair can be postponed for up to 24 hours while the patient's medical condition is optimized.

Risk Factors

- The most important risk factor for rupture is the maximum diameter of the aneurysm. Aneurysms 4.0–5.4 cm in diameter have a yearly risk of rupture of 0.5–1%. Aneurysms of 6–7 cm have a 6.6% risk of rupture per year.
- Hypertension
- COPD
- Smoking
- Female gender
- Eccentric saccular aneurysms
- Rate of expansion of the AAA as an independent risk factor has been implicated but not proven.

Prevention

- Avoid abrupt episodes of hypertension until the aneurysm has been secured.
- The goal of elective repair of AAAs is to avoid rupture. The mortality of elective repair is 6% compared with >48% for repair of ruptured AAA.
- The overall mortality rate for ruptured AAA is 90%, since 60% of patients die prior to reaching the operating room.
- Elective operative repair is recommended for AAAs 5.5 cm or greater in males and 4.5–5.0 cm in females and patients with greater than average rupture risk.

Special Considerations

- More than 90% of aneurysms are associated with atherosclerosis, but 75% of patients with aneurysmal disease do not have occlusive vascular disease. Multiple factors contribute to the destruction of the media of the aortic wall, leading to aneurysm formation. Alterations in the connective tissue of the aortic wall, proteolytic enzymes and inflammatory changes have been implicated.
- Less frequent etiologies of AAA formation include infection, arteritis, cystic medial necrosis, trauma, inherited connective tissue disorders and pseudoaneurysm formation. AAAs in young adults and children occur in the setting of tuberous sclerosis, Behcet's disease, Marfan syndrome, Ehlers-Danlos syndrome, and infection associated with umbilical artery catheters.

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Ruptured Ectopic Pregnancy

Definition

- Implantation of a fertilized ovum outside of the endometrial cavity. The most common site of ectopic pregnancy is the fallopian tube (approximately 98% of all ectopic gestations). Abdominal, ovarian and cervical ectopic pregnancies comprise the remaining 2%. Heterotopic pregnancy is the simultaneous occurrence of intrauterine and extrauterine gestation.

- In ruptured tubal ectopic pregnancy, trophoblastic proliferation extends through the tube. Hemorrhage occurs when the pregnancy extends into the large blood vessels in the broad ligament.

Presentation

- Classic symptoms: abdominal or pelvic pain and vaginal bleeding with an associated positive pregnancy test. Pain radiating to the shoulder, syncope, and shock are caused by hemoperitoneum secondary to ruptured ectopic pregnancy and occur in 20% of patients.
- Hemorrhagic shock dependent on the degree of blood loss (See Massive Hemorrhage, Page 347)
- Abdominal tenderness in 90% of patients. Peritonitis in 70% (typically rebound tenderness). Cervical motion tenderness is present in approximately 30% of patients, while a tender adnexal mass is evident in up to 50%.

Pathophysiology

In tubal implantations (the most common type of ectopic gestation), the proliferating trophoblast first invades the luminal mucosa, then the muscularis and lamina propria, and finally the serosa. Invasion into the large blood vessels in the broad ligament results in hemorrhage that distorts the tube and causes pain. Although some of these pregnancies are clinically silent and result in spontaneous tubal abortion, significant and life-threatening bleeding can result.

Immediate Management

- Early diagnosis of hemorrhage is critical. Hypotension does not occur until 30% of the circulating blood volume is lost. Mild tachycardia may be the first sign of significant blood loss.
- Establish large-bore peripheral IVs and/or central venous access.
- Begin aggressive fluid resuscitation. Class I and Class II hemorrhagic shock can usually be treated with crystalloid infusions. Transfuse PRBCs in patient with Class III and Class IV shock. (See “Massive Hemorrhage,” Page 347.)

DIFFERENTIAL DIAGNOSIS

- Any pathologic condition of the abdomen and pelvis that can cause pain and shock.
- Infectious or inflammatory intra-abdominal and pelvic diagnoses involve the gastrointestinal tract (perforated viscus, peptic ulcer

disease, intestinal ischemia, appendicitis, colitis, cholecystitis, diverticulitis, pancreatitis), the urinary tract (urosepsis of any etiology, including pyelonephritis, cystitis, obstructive nephroureterolithiasis) and the reproductive tract (pelvic inflammatory disease, salpingitis, endometritis).

- Hemorrhagic intra-abdominal and pelvic diagnoses include ruptured solid organ (liver, spleen, kidney), ruptured aortic or visceral aneurysm, ruptured hemorrhagic ovarian cyst and uterine rupture (may occur with traumatic injury).

Diagnostic Studies

- Routine non-emergent tests to establish ectopic pregnancy include serial measurements of beta-hCG, ultrasonography, uterine sampling, and occasionally progesterone levels.
- In patients presenting in hemorrhagic shock without a previous diagnosis of ectopic pregnancy, a single beta-hCG measurement and ultrasound will guide definitive management.

Subsequent Management

- Resuscitation is guided by CBC, PT, PTT, and INR. In cases of massive hemorrhage, check baseline fibrinogen level and obtain periodic fibrinogen level. Infusion of fresh frozen plasma, cryoprecipitate, and platelets is guided by laboratory parameters and surgical bleeding.
- Either laparotomy or laparoscopy with salpingectomy is performed for ruptured ectopic pregnancy.
- When significant hemorrhagic shock is present and rapid entry into the peritoneal cavity for source control is needed, emergency laparotomy is usually the best choice.

Risk Factors

- High risk: tubal surgery, tubal ligation, previous ectopic pregnancy, in utero exposure to DES (altered fallopian tube development), use of IUD, tubal pathology, assisted reproduction.
- Moderate risk: infertility, previous genital infections, multiple sexual partners.
- Low risk: previous pelvic infection, cigarette smoking, vaginal douching, young age at first intercourse.

Prevention

Based on risk stratification and a high index of suspicion, ectopic pregnancy can usually be diagnosed and treated before rupture and hemorrhage develop.

Special Considerations

- Serial beta-human chorionic gonadotropin (beta-hCG) levels are used in diagnostic algorithms. Progesterone levels are lower in ectopic pregnancies than in intrauterine gestations, but there is no established cutoff to use to distinguish between the two.
- Although multiple-dose systemic methotrexate is the first-line medical treatment of ectopic pregnancy, operative management is always the treatment of choice for ruptured ectopic pregnancy.

Further Reading

Seeber BE, Barnhart KT. Ectopic pregnancy. In: Gibbs RS, Karlan BY, Haney AF, et al. (eds.). *Danforth's Obstetrics and Gynecology*. New York: Lippincott Williams & Wilkins; 2008:71–86.

Upper GI Bleeding

Definition

Upper gastrointestinal (UGI) hemorrhage originates proximal to the ligament of Treitz. This includes etiologies attributable to the esophagus, stomach, duodenum, and, more rarely, the hepatobiliary tree, pancreas and aortoenteric fistulae.

Presentation

- Hematemesis and/or melena. Abdominal pain rarely occurs.
- Hemorrhagic shock. Classification is dependent on the degree of blood loss (see “Massive Hemorrhage”). Manifestations of shock, such as tachypnea, oliguria, and mental status changes may be present. In patients presenting with variceal hemorrhage, signs of portal hypertension and jaundice may be noted.

Anatomy and Pathophysiology

- Peptic ulcer disease is the most common etiology of UGI hemorrhage and accounts for greater than 50% of cases. *Helicobacter pylori* and NSAIDs are implicated in most cases.
- Gastric and esophageal varices are the second most common etiology and account for 15% of cases of UGI hemorrhage. Cirrhosis causes portal hypertension, which in turn results in formation of varices.
- Less common etiologies include stress ulceration, esophagitis, Mallory-Weiss tear, Dieulafoy's lesion, AVMs and tumors. Unusual etiologies include hemobilia and hemosuccus pancreaticus; unlike most patients presenting with UGI hemorrhage, these patients may have abdominal pain.

Aortoenteric fistula is another infrequent etiology of UGI hemorrhage.

DIFFERENTIAL DIAGNOSIS

Lower gastrointestinal hemorrhage originates below the ligament of Treitz and is generally less life threatening than UGI hemorrhage; shock is less likely and transfusion requirements are typically lower. 80% of all patients with gastrointestinal hemorrhage pass blood in some form from the rectum. 20% of all cases of apparent lower gastrointestinal hemorrhage have an upper gastrointestinal source, including massive nasal or oropharyngeal hemorrhage resulting in swallowed blood.

Immediate Management

- Intubate the trachea if the patient has hematemesis or altered mental status, or if shock is imminent.
- Be prepared to suction copious blood from the airway.
- Establish large-bore peripheral and/or central venous access. Resuscitate aggressively with crystalloid IV fluids and PRBC. Class I and Class II hemorrhagic shock can usually be treated with crystalloid infusions. PRBC are necessary in Class III and Class IV shock.
- Draw baseline laboratories: CBC, PT, PTT, INR and comprehensive metabolic panel, including liver function tests.
- In cases of massive hemorrhage or known hepatic dysfunction, check fibrinogen level at baseline and periodically throughout the resuscitation.
- Correct coagulopathy, thrombocytopenia, platelet dysfunction (medication induced or pathologic) with blood products and ddAVP.

Diagnostic Studies

- After initial stabilization, the source of bleeding must be determined. Esophagogastroduodenoscopy is the modality of choice. Findings are useful in predicting the risk of rebleeding:
- Active arterial bleeding
 - 90–100% risk of rebleeding without endoscopic intervention
 - 15–30% risk of rebleeding with endoscopic intervention
- Visible nonbleeding vessel
 - 40–50% risk of rebleeding without endoscopic intervention
 - 15–30% risk of rebleeding with endoscopic intervention
- Adherent clot in ulcer base
 - 20–30% risk of rebleeding without endoscopic intervention
 - 5% risk of rebleeding with endoscopic intervention

- Angiography: bleeding rates must be at least 0.5 mL/minute for adequate visualization.
- Nuclear medicine scans (tagged RBC scan): bleeding rates must be at least 0.1 mL/minute to be detected.

Subsequent Management

- In general, 80% of cases of UGI hemorrhage stop spontaneously.
- 90% hemostasis rates are achieved with endoscopic therapy.
- Interventional radiology may be used to deliver intra-arterial vasopressin or embolize the lesion in poor surgical candidates.
- Surgical indications in patients with UGI hemorrhage attributable to peptic ulcer disease include two failed attempts of endoscopic hemostasis, rapid deterioration attributable to exsanguination, large visible vessels not amenable to endoscopic coagulation, and documented malignant ulcers.
- Medical management of variceal hemorrhage includes vasopressin (bolus 0.4 U with 0.4–1 U/minute infusion) plus nitroglycerin (10–50 mcg/minute) or octreotide (50 mcg bolus with 50 mcg/h infusion for 5 days).
- Endoscopic management of variceal hemorrhage includes sclerotherapy and variceal band ligation.
- Esophageal variceal bleeding may be treated with transjugular intrahepatic portosystemic shunt (TIPS) when medical and endoscopic therapy fail or are not feasible, as a bridge to hepatic transplantation. There is an associated risk of worsening hepatic encephalopathy.

Risk Factors for Mortality (6–10%)

- Advanced age
- Renal insufficiency
- Hepatic failure
- Disseminated malignancy

Prevention

Treatment of helicobacter pylori infection and limitation of NSAID use may help to prevent complications of peptic ulceration. Major indications for stress ulcer prophylaxis in the critically ill include respiratory failure and coagulopathy.

Further Reading

- Cheung FK, Lau JY. Management of massive peptic ulcer bleeding. *Gastroenterol Clin North Am.* 2009 Jun;38(2):231–243.
- Donahue PE. Foregut. In: Britt LD, Trunkey DD, Feliciano DV (eds.). *Acute Care Surgery: Principles and Practice.* New York: Springer; 2007:450–470.
- Sass DA, Chopra KB. Portal hypertension and variceal hemorrhage. *Med Clin North Am.* 2009 Jul;93(4):837–853.

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Chapter 15

Thoracic Emergencies

Marc S. Azran and Michael Nurok

- Bleeding During Mediastinoscopy 364
- Bronchopleural Fistula (BPF) 365
- Cardiac Herniation after Pneumonectomy 368
- Inhaled Foreign Body (Adult) 371
- Intrathoracic and Mediastinal Lesions Causing Tracheal, Bronchial, Cardiac, and/or Vascular Obstruction 374
- One-Lung Ventilation: Hypoxemia 378
- One-Lung Ventilation: Increased Airway Pressure 380
- Tension Pneumothorax 381
- Tracheal Injury 384

Bleeding During Mediastinoscopy

Definition

Bleeding greater than 500 cc or requiring exploration through a median sternotomy or thoracotomy. The incidence is 0.4%.

Presentation

- Arterial or venous surgical bleeding
- Hypotension
- Tachycardia
- Cardiovascular collapse

Etiology

- Accidental biopsy of a vascular structure.
- Azygous vein injury
- Innominate vein or artery injury
- Pulmonary artery injury
- Injury to the aortic arch
- Patient movement

Immediate Management

- Surgical compression and wound packing—apply pressure with mediastinoscope, or gauze soaked in epinephrine.
- Establish large-bore IV access in the *lower extremities* if innominate vein injury is suspected.
- Obtain crossmatched blood and set up rapid infusers.
- Begin volume resuscitation.
- Secure arterial access and equipment for invasive blood pressure monitoring.
- Support blood pressure with ephedrine (5 mg IV) or phenylephrine (100 μ g IV) boluses. If refractory, consider phenylephrine infusion (0.5–1 mcg/kg/min).

Subsequent Management

- Surgical exploration and repair is the definitive treatment for refractory bleeding. The surgical approach will depend on the injured vessel.
- Midline sternotomy: Innominate vein, pulmonary artery
- Right posterolateral thoracotomy: Azygous vein, right pulmonary artery or bronchial artery
- Lung isolation may enhance surgical exposure.

Lung Isolation for Right Thoracotomy

- *Previously easy intubation & control of bleeding:*
 - Confirm adequate muscle relaxation.
 - Change the ETT to a left-sided double-lumen tube (with or without an airway exchange catheter).
- *Previously difficult intubation or uncontrolled bleeding:*
 - Left mainstem intubation using the existing endotracheal tube over a fiberoptic bronchoscope.
 - Alternatively, a right-sided bronchial blocker may be used.

Risk Factors

- Aberrant vessels
- Superior vena cava (SVC) syndrome causing engorged vasculature
- Mediastinal inflammation as a result of prior chemotherapy, radiation therapy, or mediastinal procedure

Prevention

- Surgical palpation of the lesion or needle aspiration prior to biopsy to identify a vascular structure.
- Adequate muscle relaxation should be confirmed before biopsy to prevent movement during this critical period.

Special Considerations

- Persistent hemodynamic instability with only minor blood loss may be caused by cardiac tamponade. Bleeding from the bronchial artery into the pericardial sac has occurred as a result of biopsy. Transesophageal echocardiography should be performed to rule out pericardial effusion and tamponade physiology.

Further Reading

Park BJ, Flores R, Downey RJ, Bains MS, Rusch VW. Management of major hemorrhage during mediastinoscopy. *J Thorac Cardiovasc Surg.* 2003 Sep;126(3):726–731.

Bronchopleural Fistula (BPF)

Definition

A communication between the bronchial tree and pleural space causing a pneumothorax. Immediate postoperative blow-out of the stump will result in the inability to ventilate effectively. This situation is different from the remote small, parenchymal BPF, for which the mortality rate may reach 50%.

Presentation

- Inability to ventilate (complete stump blow-out)
- Dyspnea
- Oxygen desaturation
- Increased inspiratory pressures
- Cyanosis
- Hypotension
- Tachycardia

Etiology

- Surgical
 - Any pulmonary resection
 - Lung volume reduction surgery
 - Pneumonectomy or lobectomy
- Infectious
 - Pneumonia, empyema, or lung abscess
 - Tuberculosis
- Iatrogenic
 - Central line placement
 - Baro- or volutrauma during mechanical ventilation (often in the setting of acute lung injury)
- Penetrating chest trauma may cause a bronchopleuralcutaneous fistula.

Immediate Management

- Immediate postoperative stump blow-out
 - Isolate the lungs with a double-lumen endotracheal tube or bronchial blocker.
 - Thoracostomy tube placement as described below
 - Surgical re-exploration
- Remote BPF
 - Immediate thoracostomy tube insertion
 - A BPF may permit air flow up to 16 liters per minute (LPM) across the defect. A thoracostomy tube with an internal diameter of at least 6 mm is required to evacuate this degree of flow and avoid tension pneumothorax.
 - High volume thoracostomy tube drainage system ideally capable of evacuating up to 35 LPM with a suction pressure of -20 cm H₂O.

Subsequent Management

- Can be managed conservatively by treating underlying lung pathology and weaning mechanical ventilation. Positive pressure ventilation creates a gradient between the airways and pleural space, allowing air to pass through the fistula. During mechanical ventilation, minimizing the volume of the leak promotes healing.
- Conventional mechanical ventilation
 - Minimize alveolar distension and airway pressure.
 - Decrease minute ventilation (both rate and tidal volume).
 - Decrease inspiratory time by altering the I:E ratio or by increasing inspiratory flow rates.
 - Utilize weaning modes to encourage spontaneous ventilation.
 - Minimize positive end-expiratory pressure (PEEP).
 - Decrease chest tube suction as tolerated.
 - Aggressively treat bronchospasm and other airflow obstruction.
- Alternative modes of ventilation may decrease mean airway pressures.
 - Chest tube occlusion during the inspiratory phase and chest tube PEEP may minimize the egress of air during ventilation. Tension pneumothorax is a risk.
 - Lateral decubitus ventilation with the BPF in the dependent position theoretically decreases BPF airflow.
 - Differential lung ventilation with a double-lumen endotracheal tube. The normal lung may be ventilated conventionally, while the injured lung may be managed with any of the above techniques. Two synchronized ventilators are required.
 - High-frequency jet ventilation may be used to maintain oxygenation, although respiratory acidosis may occur because CO₂ elimination is compromised.

Invasive Management

- Bronchoscopy may be used to localize and manage BPFs. A proximal BPF may be sealed with fibrin, autologous blood or cautery.
- Occlusion balloons may be left in place for distal BPFs that are not visualized.
- Chest tube or thoroscopic sclerosis may incite an inflammatory response, thereby sealing a BPF.
- Surgical pleurodesis or bronchial stapling remains an option for refractory cases.
- If 100% oxygen is required, the surgical team should be alerted because electrocautery may cause a fire.

DIFFERENTIAL DIAGNOSIS

- Endotracheal tube malposition
- Bronchospasm
- Extrinsic lung compression from masses or fluid
- Hemothorax
- Cardiac tamponade

Diagnostic Studies

Chest radiograph following chest tube insertion of the affected lung

Risk Factors

- High-risk pulmonary surgery or lung volume reduction surgery
- Pulmonary infection
- Central line placement
- Excessive tidal volumes (more than 10 mL/kg) in the setting of lung injury
- Obstructive lung disease with autoPEEP

Prevention

- Decrease tidal volumes to 6 mL/kg
- Permissive hypercapnia
- Reduce inspiratory pressures
- Use judicious PEEP by monitoring static respiratory compliance during incremental PEEP increases. Decrease PEEP if compliance falls.
- Wean mechanical ventilation early
- Institute measures to avoid nosocomial pneumonia
- Suspend mechanical ventilation while inserting a needle into the internal jugular or subclavian veins.
- Surgical reinforcement of stumps with flaps (intercostal muscle, omental tissue, etc.)

Further Reading

Baumann MH, Sahn SA. Medical management and therapy of bronchopleural fistulas in the mechanically ventilated patient. *Chest*. 1990 Mar;97(3):721–728.

Cardiac Herniation after Pneumonectomy

Definition

Herniation of the myocardium into an empty hemithorax through a pericardial defect created during pneumonectomy. Both

left- and right-sided cardiac herniation may occur, causing severe hemodynamic instability. Mortality is 50% if recognized, and 100% if left untreated.

Presentation

- Hypotension
- Tachycardia
- Cardiovascular collapse
- Dysrhythmias
- ECG changes (ST segment or axis changes)
- Right-sided herniation
 - Jugular venous distension
 - Cyanosis of the face
 - Absence of a left-sided cardiac impulse
- Usually occurs during transfer of the patient. May occur within the first 24 hours after surgery.

Etiology

- Pneumonectomy with an empty hemithorax and unclosed pericardial defect places the patient at risk for herniation.
- Intrapericardial pneumonectomy performed for hilar tumors and masses invading the pericardium creates a pericardial defect.
- Extrapleural pneumonectomy is performed for resection of mesothelioma involving the construction of a pericardial patch after excision of the lung. A loosely constructed patch on the right side may predispose to herniation by allowing the mediastinum to shift.

Pathophysiology

- Right-sided cardiac herniation results in cardiac malposition.
 - Torsion of the atrio caval junction severely impedes venous return.
 - Torsion of the great vessels or ventricular outflow tract, causing obstruction to blood flow.
 - The end result is myocardial ischemia and cardiovascular collapse.
- Left-sided cardiac herniation involves prolapse of the ventricles through the defect.
 - Cardiac orientation and venous return is preserved.
 - Strangulation of the myocardium and epicardial vessels results in myocardial ischemia.
 - Ventricular outflow tract obstruction may also occur.

DIFFERENTIAL DIAGNOSIS

- Myocardial infarction
- Hypovolemic shock
- Contralateral pneumothorax
- Pulmonary embolus
- Cardiac tamponade

Immediate Management

- Clinical scenario can help to differentiate between cardiac herniation (which requires immediate surgical intervention) and hypotension due to hypovolemia or deep anesthesia (which will respond to volume administration and/or vasopressors).

Management of Presumed Cardiac Herniation

- Alert the surgeon immediately.
- Reposition the patient with the operative side up
- Decrease tidal volumes and positive end-expiratory pressure (PEEP).
- Begin aggressive volume resuscitation to correct relative preload deficiency.
- Support blood pressure as necessary with vasopressors. Severe hypotension may require aggressive treatment with an infusion of phenylephrine (0.5–1 mcg/kg/min) or epinephrine (0.03–0.05 mcg/kg/min).
- If complete cardiac arrest occurs, reposition and have surgeon intervene immediately.
- CPR may make herniation worse.
- Surgical correction with reduction of myocardial prolapse and pericardial patch construction.

Diagnostic Studies

- The diagnosis should be clinical and management should not be delayed.
- Chest radiograph
 - Right-sided herniation
 - Opacification of the right hemithorax from mediastinal shift
 - Abnormal (clockwise) configuration of a pulmonary artery catheter
 - Left-sided herniation may show a rounded opacity in the lower left hemithorax due to ventricular strangulation.
- Echocardiography (TTE or TEE) may reveal prolapsed myocardium or a malpositioned heart.

Risk Factors

- Pericardial defect of any size and an empty hemithorax
- Chest tubes placed to suction or even water seal
- Positive pressure ventilation and PEEP
- Coughing secondary to suctioning or extubation
- Position changes in operating room or during transport from the operating room (especially operative side down)

Prevention

A pericardial sling or patch should be used to close large pericardial defects. Primary closure of the pericardium has been used for smaller defects, although herniation can occur from a ruptured suture line.

Further Reading

Chambers N, Walton S, Pearce A. Cardiac herniation following pneumonectomy—an old complication revisited. *Anaesth Intensive Care*. 2005 Jun;33(3):403–409.

Inhaled Foreign Body (Adult)

Definition

Aspiration of organic or inorganic material into the tracheobronchial tree.

Presentation

- At the laryngeal inlet: coughing, choking, hoarseness or cyanosis.
- Below the cords: inspiratory stridor and coughing.
- In a bronchus: unilateral wheezing and coughing.
- In cases of delayed diagnosis, may present with recurrent pneumonia, empyema, hemoptysis or bronchopleural fistula.

Etiology

- Organic material
 - Beans or other organic material may become engorged causing complete airway obstruction.
 - Inspissated oral secretions may cause respiratory arrest.
- Inorganic material may be tolerated for years with minimal symptoms.

Immediate Management

- In adults, FB aspiration is rarely a true emergency.
 - Monitor closely and give supplemental oxygen while the operating room is prepared.
 - The ability to convert from flexible to rigid bronchoscopy to thoracotomy should be immediately available.
- Emergency FB asphyxiation usually involves material at the supraglottic larynx or subglottic trachea.
 - Direct laryngoscopy can be performed while the patient is awake and the offending object is grasped with Magill forceps.
 - Intubation with an ETT may be used to push the object into a distal bronchus, enabling life-saving ventilation while the patient is prepared for bronchoscopy.

Subsequent Management

- *Rigid bronchoscopy* is the most effective therapeutic intervention and usually requires general anesthesia.
 - Positive pressure ventilation can wedge the FB distally, creating a ball-valve obstruction.
 - Spontaneous ventilation is preferable using an inhaled anesthetic technique. Spontaneous breathing enables ventilation when the bronchoscopist's ocular window is open.
 - Neuromuscular blockade may be necessary to facilitate laryngoscopy. Short-acting agents are preferable. Intermittent ventilation is possible through the bronchoscope. If positive pressure ventilation is necessary, consider an intravenous anesthetic technique to reduce operating room pollution with volatile agents.
- *Flexible bronchoscopy* is the tool of choice when the FB is wedged in a distal segment, in patients with cervical spine pathology, or when the patient is intubated and mechanically ventilated.
 - Avoids general anesthesia and preserves spontaneous ventilation.
 - Preservation of the cough reflex is imperative in patients with full stomachs and also aids in FB expulsion. Use local anesthetics sparingly.
 - Sedatives should be administered judiciously.
 - A laryngeal mask airway may be inserted in an awake patient after topical anesthesia of the oropharynx, and can be used as a conduit for the flexible bronchoscope. This provides some degree of airway control and is large enough to allow for extraction of the FB.

- Special forceps and snares are passed through the working port of the bronchoscope. The bronchoscope, grasping device, and FB are all removed as a unit. If the FB dislodges in the proximal trachea, utilize the Trendelenberg position and ask the patient to cough.
- If an ETT is in place, the FB may not pass through the ETT—necessitating extubation along with FB and bronchoscope extraction.

DIFFERENTIAL DIAGNOSIS

- Pneumothorax
- Bronchospasm
- Tracheal injury
- Airway compression from the intrathoracic mass

Diagnostic Studies

Chest radiographs are useful only in patients with radio-opaque FBs. Findings may include air-trapping, atelectasis, pulmonary infiltrates, or mediastinal shift.

Risk Factors

- Advanced age
- Neurologic disorders with impairment of swallowing
- Alcohol and sedative use
- Trauma with loss of consciousness
- General anesthesia
- Seizures

Special Considerations

- Massive hemoptysis may occur with FB removal. Consider lung isolation with a double-lumen ETT or bronchial blocker if bleeding cannot be controlled. Preparations should be made for thoracotomy or bronchial artery embolization.
- Prolonged rigid bronchoscopy may lead to postoperative laryngeal edema. Treatment options include:
 - Nebulized racemic epinephrine
 - Helium/oxygen mixtures
 - Dexamethasone 10 mg IV bolus
- Failed bronchoscopy may ultimately require thoracotomy and bronchotomy to remove the impacted object.

Further Reading

Rafanan AL, Mehta HC. Adult foreign body removal. What's new? *Clin Chest Med.* 2001 Jun;22(2):319–330.

Intrathoracic and Mediastinal Lesions Causing Tracheal, Bronchial, Cardiac, and/or Vascular Obstruction

Definition

Extrinsic or intrinsic obstruction of intrathoracic structures caused by tumor, tracheal disease, or vascular compression, with potential to lead to cardiovascular collapse and/or the complete inability to oxygenate and ventilate.

Presentation

- Dyspnea
- Cough
- Orthopnea
- Inspiratory stridor or expiratory wheeze
- Hoarseness
- Hemoptysis
- Obstructive pneumonia

Etiology

Causes of obstruction include:

- Malignancy
 - Primary airway tumors (usually intrinsic): squamous cell carcinoma, adenoid cystic carcinoma or carcinoid tumor
 - Primary adjacent tumor (usually intrinsic): anterior or middle mediastinal tumors, lung carcinoma, or esophageal carcinoma
 - Metastatic cancer (extrinsic): renal, breast, colon or sarcoma
- Tracheal disease related to stenosis or tracheomalacia (intrinsic)
- Vascular compression or ring (extrinsic)

General anesthesia can cause cessation of spontaneous ventilation, increased intrapleural pressure, and decreased functional residual capacity. As a result, airways that had been patent can collapse and cause worsening or complete obstruction.

DIFFERENTIAL DIAGNOSIS

- Foreign body aspiration
- Bronchospasm
- Pneumothorax

Immediate Management

Pre-procedure:

- Identify location of lesion on imaging.
- Identify relationship of lesion to adjacent structures.
- Anticipate the potential for compression of the trachea, bronchi, and cardiovascular structures (greater than 50% compression by tracheal lesions on imaging warrants a conservative approach).
- Insert an intraarterial catheter if there is a risk of cardiovascular compromise.
- Obtain large-bore IV access.
- If SVC involvement or syndrome, place a large-bore IV in a lower extremity.
- Ensure a mechanism for administering intravenous fluids rapidly.
- Ensure that vasopressors are immediately available.
- Rigid bronchoscopy should be immediately available.
- Surgical team must be present in the room.
- Femoral arterial and venous access is established, with standby extracorporeal support device in extreme cases.

Procedure:

- A local anesthetic technique in the awake, spontaneously breathing patient is safest in patients with severe or potentially severe obstruction.
- Avoid sedatives, or administer judiciously.
- Maintain patient in the sitting position if possible.
- Stepwise airway approach:
 - Awake fiberoptic examination of airway, trachea, and bronchi by anesthesia and surgical team to characterize the lesion and plan an approach to definitive airway management (may be facilitated by the use of a laryngeal mask airway—maintaining patient awake throughout—following topical local anesthesia)
 - Awake fiberoptic intubation with passage of tube distal to lesion if possible
 - If not possible, alternative strategies for securing the airway while maintaining spontaneous ventilation should be considered, including the use of extracorporeal support.
 - Securing the airway *after* induction should be attempted only with great caution, and with appropriately skilled individuals immediately available.

Induction:

- The airway is most safely managed *prior* to the induction of general anesthesia.

Immediate Management (continued)

- Gradually administer an inhalational agent or small doses of intravenous agents while maintaining spontaneous ventilation.
- Attempt to assist with bag-ventilation
 - If successful
 - Overtake spontaneous ventilation with positive pressure.
 - Gradually increase the depth of anesthesia.
 - If not successful
 - Shifting the lesion by placing the patient in the lateral decubitus or prone position may improve ventilation.
 - Awaken the patient and reconsider the airway approach and/or using extracorporeal support.
- If muscle relaxation is required, use a small dose of succinylcholine *after* the airway is secure and the ability to provide positive pressure ventilation has been confirmed. **Neuromuscular blockade may cause airway collapse in a marginal patient.** If succinylcholine is tolerated, longer-acting agents can be safely administered.
- In the event of total airway obstruction that is not responsive to patient repositioning (lateral decubitus or prone position), a rigid bronchoscope or armored endotracheal tube may be used in an attempt to secure the airway distal to the collapse. An alternative rescue strategy is to pass a jet ventilator cannula distal to the lesion.
- An intravenous anesthetic is preferable for maintenance, as inhalational agents will contaminate the operating room with surgical manipulation of the airway.

Therapeutic Approaches

- Surgical airway resection and reconstruction
- Tracheal (or bronchial) stent placement for symptomatic relief.
- Awake tracheostomy with a long-length prosthesis allows for control of the airway distal to the lesion.
- Endobronchial brachytherapy or external beam radiotherapy
 - Preoperative radiation can significantly reduce tumor burden.
- Nd:YAG laser therapy in conjunction with rigid bronchoscopy can vaporize lesions and achieve hemostasis. Tracheal perforation and hemorrhage, and airway fire, are risks with this procedure.
- Photodynamic therapy
 - IV photosensitizer is administered and retained in tumor cells.
 - Specific wavelength light activates the agent and generates cytotoxic oxygen radicals.

Diagnostic Studies

- Neck and chest radiographs may detect tracheal deviation, endoluminal narrowing, and obstructive pneumonia.
- CT of the neck and chest can determine the exact location, length, and nature of the obstruction.
- Both MRI and CT angiography are useful in characterizing vascular malformations.
- TTE may be useful in evaluating the pericardium for malignant effusion or tumor.
- Flow-volume loops are neither sensitive nor specific in characterizing obstruction.

Risk Factors

Prolonged intubation or tracheostomy may lead to stenosis or tracheomalacia.

Prevention

Daily spontaneous breathing trials and sedation holidays may reduce the incidence of complications from prolonged intubation.

Special Considerations

- If the clinical scenario is concerning for an inability to maintain a patent airway during instrumentation or other stages of the procedure, prior to proceeding the femoral artery and vein should be cannulated to permit extracorporeal oxygenation and ventilation.
- Superior vena cava (SVC) syndrome
 - Obstruction by tumor burden of the SVC
 - Requires lower extremity IV access
 - May have significant upper airway edema and friable tissue
- Postoperative airway obstruction
 - May occur from tumor swelling after manipulation
 - Tracheal narrowing to less than 50% of normal confers a 7-fold increased risk.
- There is a risk of acute intraoperative airway obstruction if the patient develops positional dyspnea while supine.

Further Reading

Wood DE. Management of malignant tracheobronchial obstruction. *Surg Clin North Am.* 2002 Jun;82(3):621–642.

One-Lung Ventilation: Hypoxemia

Definition

Low PaO₂, Low SvO₂

Presentation

- Low oxygen saturation by pulse oximetry
- Dark arterial blood
- Cyanotic patient
- Cardiac dysrhythmias

Pathophysiology

Pathophysiology is multifactorial. Shunt develops in the nonventilated lung following resorption of residual oxygen. In the ventilated lung, regions of low ventilation to perfusion ratios develop as a result of atelectasis and West zone III conditions from compression by the nonventilated lung, mediastinal structures, and the diaphragm. Other causes of hypoxemia from the ventilated lung include hypoxic pulmonary vasoconstriction (causing redistribution of blood to the nonventilated lung and increasing shunt), secretions, and double lumen tube or lung isolation device malposition.

DIFFERENTIAL DIAGNOSIS

- Increased oxygen utilization
- Decreased oxygen delivery (low cardiac output states)

Immediate Management

- Eliminate causes proximal to the double lumen tube including ventilator disconnect or failure.
- Mild hypoxemia (SpO₂ >90%)
 - Increase FiO₂ to 100%.
 - Fiberoptic bronchoscopy to ensure correct position of double lumen tube or lung isolation device
 - Check the ventilated lung for obstruction or secretions—passage of suction catheter is more efficient than using the narrow port on fiberoptic bronchoscope.
 - Ensure adequate cardiac output and oxygen carrying capacity.
 - Recruit ventilated lung. Follow with addition of PEEP if using low tidal volume ventilation (will only work if lung is being ventilated in a noncompliant region on the low end of its pressure volume curve).
 - Continuous positive airway pressure to nonventilated lung (start with 5 cm H₂O to avoid distention of operative lung)

Immediate Management (continued)

- Consider administration of total intravenous anesthetic to promote hypoxic pulmonary vasoconstriction in the nonventilated lung (impaired by potent volatile anesthetics).
- Consider increasing tidal volume to 6–10 cc/kg.
- No improvement or significant hypoxemia ($SpO_2 < 90\%$)
 - Inform surgeon and ventilate both lungs.
 - If no improvement, or the above steps are not possible, ask surgeon to clamp pulmonary artery in the event of a planned pneumonectomy of nonventilated lung.
 - If no improvement, consider high-frequency jet ventilation to operative lung.
 - If no improvement, consider nitric oxide or almitrine (not available in the US).
 - If no improvement, consider extracorporeal membrane oxygenation (ECMO).

Diagnostic Studies

- Arterial and mixed venous blood gas measurement
- Fiberoptic bronchoscopy

Risk Factors

- Patients with increased or normal ventilation and perfusion to the operative lung will have a larger shunt during one-lung ventilation.
- Right-sided operations (right lung normally receives 60% of blood flow)
- Low PaO_2 during two-lung ventilation
- Normal or high FEV1/FVC ratio

Prevention

- Maintain two-lung ventilation as long as possible.
- Ensure proper double lumen tube or lung isolation device position following significant changes in patient positioning.
- Ensure appropriate ventilator settings when beginning one-lung ventilation (in patients with normal lungs, pressure control ventilation, 4–6 cc/kg ideal body weight with 5–10 cm H_2O PEEP, respiratory rate 10–15/min, FiO_2 0.5–0.8).
- Adapt ventilatory strategy to lung pathology.
- Avoid high concentration of potent volatile anesthetic agents, which blunt hypoxic pulmonary vasoconstriction.

- Avoid large tidal volume or high PEEP to the ventilated lung, which increases pulmonary vascular resistance and may shunt blood to the nonventilated lung.

Further Reading

Lohser J. Evidence-based management of one-lung ventilation. *Anesthesiology Clin.* 2008;26:241–272.

One-Lung Ventilation: Increased Airway Pressure

Definition

Increase in peak or plateau inspiratory airway pressure

Presentation

- Elevated airway pressures during volume-controlled mode of ventilation
- Low tidal volume during a pressure-controlled mode of ventilation
- Low blood pressure

Pathophysiology

The most common cause of elevated airway pressure during one-lung ventilation is malposition of the double lumen tube or lung isolation device, such that a larger volume of gas is displaced into a smaller portion of lung, leading to elevated pressures. Other causes include excessive tidal volume delivery to one lung, secretions, bronchospasm, and tension pneumothorax.

Immediate Management

- Inspect the ventilator tubing and endotracheal tube for kinks.
- Disconnect the patient from the ventilator and allow to exhale fully to exclude air trapping/autoPEEP.
- Manually ventilate the patient to evaluate compliance.
- If high airway pressure is accompanied by hypoxemia, resume two-lung ventilation.
- Perform fiberoptic bronchoscopy to ensure proper positioning of double lumen tube or lung isolation device.
- Pass a suction catheter through the lumen leading to the ventilated portion of the lung to eliminate obstruction.

Immediate Management (continued)

- Ensure appropriate tidal volume and PEEP settings for one-lung ventilation (in patients with normal lungs, pressure control ventilation, 4–6 cc/kg ideal body weight, with 5–10 cm H₂O PEEP, respiratory rate 10–15/min, FiO₂ 0.5–0.8).
- Address other causes individually.

DIFFERENTIAL DIAGNOSIS

- Double lumen tube or lung isolation device malposition
- Obstruction to double lumen tube (blood, secretions, foreign body)
- Excessive tidal volume to one lung
- Bronchospasm
- Tension pneumothorax
- Air trapping/AutoPEEP (allow patient to exhale fully)

Risk Factors

- Surgical manipulation of the airway
- Change in patient position
- Right-sided double lumen tube placement (orifice to right upper lobe may become misaligned, leading to exclusion of right upper lobe and entire tidal volume being delivered to only the right lower and middle lobes)
- Obstructive lung disease is a risk factor for air trapping (use long expiratory times).

Prevention

Confirmation of correct placement of double lumen tube or lung isolation device after position changes or significant surgical manipulation.

Tension Pneumothorax**Definition**

Abnormal presence of gas in the pleural cavity with inability to escape, causing pressure on intrathoracic structures.

Presentation

- High peak airway pressure
- Decreased tidal volume

- Decreased SpO₂, SvO₂
- Hypotension
- Tachycardia
- Distension of neck veins
- Subcutaneous emphysema
- Contralateral tracheal deviation
- Hyperresonance of the affected chest
- Hyperexpansion of the affected chest
- Reduced breath sounds in the affected chest
- Compression of bronchi on fiberoptic inspection of affected side
- Elevation of the mediastinum in the surgical field

Pathophysiology

Gas accumulates in the pleural space through passage from the ventilated lung as a result of high airway pressure, rupturing of a bleb, or through the chest wall. A one-way valve effect prevents gas from escaping the pleural space. Increasing pressure in the pleural cavity from accumulation of gas results in clinical symptoms by compressing intrathoracic structures including the mediastinum, lung, and blood vessels.

DIFFERENTIAL DIAGNOSIS

- Hyperinflation of the ventilated lung with intrinsic PEEP (disconnect patient from ventilator circuit and allow exhalation)
- Double lumen tube or lung isolation device malposition
- Bronchospasm
- Extrinsic lung compression
- Hemothorax
- Cardiac tamponade

Immediate Management

- Inform surgical team.
- Resume two-lung ventilation.
- Increase FiO₂ to 1.0.
- Consult surgeon about the ability to access the lung from the surgical field:
 - Enquire about the possibility to surgically dissect a plane to the affected pleural space between the aorta and esophagus posteriorly, and the pericardium anteriorly.
 - Accessing this space will immediately decompress the pneumothorax.

Immediate Management (*continued*)

- If the above steps are not possible, roll patient supine and decompress the affected side with a large-bore needle or a long 14 gauge intravenous catheter in the second intercostal space at the midclavicular line. Proceed immediately to formal tube thoracostomy.
- If there is uncertainty about the diagnosis, and the patient is hemodynamically stable, fiberoptic bronchoscopy may show compression of major bronchi, and a chest radiograph will provide a definitive diagnosis.

Diagnostic Studies

Chest X-ray following decompression of affected lung

Subsequent Management

Tube thoracostomy

Risk Factors

- Elevated peak airway pressures
- Malposition of the double lumen tube leading to high airway pressure
- Obstructive lung disease
- Acute lung injury
- Pleural blebs
- Penetrating chest wall injury
- Recent central venous catheter insertion

Prevention

- Appropriate ventilatory management to avoid elevated airway pressures
- Appropriate double lumen tube or lung isolation device placement

Special Considerations

Tension pneumothorax has been reported during one-lung ventilation in the absence of the classic signs of hypoxemia and hypotension.

Further Reading

Malik S, Shapiro WA, Jablons D, Katz JA. Contralateral tension pneumothorax during one-lung ventilation for lobectomy: diagnosis aided by fiberoptic bronchoscopy. *Anesth Analg*. 2002 Sep;95(3):570–572.

Tracheal Injury

Definition

Injury to any portion of the extrathoracic or intrathoracic trachea. May involve complete or partial disruption of the trachea. Mortality estimates vary widely depending upon the etiology.

Presentation

- Dyspnea
- Hoarseness/stridor
- Signs of external trauma
- Subcutaneous emphysema
- Pneumothorax, pneumomediastinum or pneumopericardium
- Cyanosis and oxygen desaturation
- Hemoptysis

Etiology

- *Iatrogenic* causes include endotracheal intubation, use of tube exchange catheter, percutaneous dilational tracheostomy, and cricothyroidotomy.
 - Often results in tearing of the posterior membranous trachea
- *Blunt trauma*
 - Frequently injures the trachea within 2 cm of the carina
 - Chest trauma may result in complete transection or posterior membranous tracheal tear.
 - Upper airway trauma may result in fractured laryngeal cartilages.
 - Flexion-extension injury may precipitate full laryngotracheal separation.
- *Penetrating trauma* mostly occurs to the cervical trachea, but one-third may affect the larynx.
- *Mucosal tears* are often self-limited—through-and-through tears require specialist care.

Management of Iatrogenic Injury

Iatrogenic trauma that results in small tears to the middle and upper third of the trachea may be managed conservatively.

- Advance the tracheostomy or endotracheal tube beyond the lesion using a fiberoptic bronchoscope.
- Inflate the cuff to eliminate airway pressure on the proximal tear.
- Long-term ventilation may be required to allow healing.

Airway Management of Blunt or Penetrating Trauma

Airway manipulation may quickly turn a stable situation into a life-threatening one by precipitating complete obstruction.

- *General guidelines*

- Tracheostomy equipment and a skilled surgeon should be present.
- Maintain spontaneous ventilation. Avoid intravenous sedatives and neuromuscular blockers until the airway is secure.
- Positive pressure ventilation before a cuff has excluded the injury can worsen existing pneumothorax, pneumomediastinum, or air dissection around the airways.
- Awake fiberoptic bronchoscopy is the diagnostic and interventional procedure of choice for determining the nature of the injury as well as securing the airway.
- The goal is to inflate a cuffed airway device or cannula distal to tracheal disruption to permit positive pressure ventilation.
- If 100% O₂ is required to maintain oxygenation, the surgical team should be alerted, as concomitant use of electrocautery may cause a fire.

- *Cervical tracheal and upper airway lesions*

- Patients with open tracheal disruptions may be oxygenated via facemask and cannulated with the aid of a bronchoscope. A jet ventilator cannula may also be used for oxygenation.
- Rigid bronchoscopy may be diagnostic when blood in the airway precludes flexible bronchoscopy. An inhaled anesthetic technique with spontaneous ventilation can be used to facilitate this approach, but there is a possibility of aspiration of gastric contents. The clinical scenario should dictate the best anesthetic approach.
- With blunt laryngeal injuries, attempted conventional laryngoscopy and endotracheal intubation may fracture the cricoid cartilage or provoke complete transection. Avoid cricoid pressure. Awake oral fiberoptic intubation or awake tracheostomy distal to the lesion are safe airway management options.

- *Lower tracheal lesions*

- Blunt chest trauma disrupts the trachea within several centimeters of the carina. Tracheostomy will not permit ventilation in this situation.
- Flexible fiberoptic bronchoscopy is essential in diagnosing and safely crossing the lesion. Intubation is best achieved over the bronchoscope.
- The ETT cuff should be placed distal to the lesion and proximal to the carina if possible. Alternatively, consider endobronchial intubation or jet ventilation through a catheter located distal to the lesion.

Surgical Considerations

- The approach for high lesions is a collar incision. The mediastinal trachea is repaired via a right posterolateral thoracotomy. The left approach is sometimes used, so the surgeon should be consulted.
- One-lung ventilation may be required to facilitate surgical exposure via a thoracotomy approach.
 - An airway isolation device or blocker may be used.
 - Alternatively, bronchial intubation with a double lumen ETT may safely be achieved over a fiberoptic bronchoscope or airway exchange catheter of adequate length.
- The ETT may need to be pulled back to facilitate visualization of the surgical anastomosis. An armored ETT and sterile circuit should be used to provide ventilation across the surgical field.
- At the conclusion of surgery, bronchoscopy should be used to position the cuff of the ETT distal to the anastomosis.

DIFFERENTIAL DIAGNOSIS

- Pneumothorax
- Foreign body aspiration
- Pulmonary hemorrhage
- Bronchospasm

Diagnostic Studies

- Airway management should not be delayed by studies.
- Radiographic findings may include cervical emphysema, pneumothorax, pneumomediastinum or air column disruption.
- CT is sensitive, but lying flat may prove difficult and sedatives should be used with great caution before the airway is secure.
- Esophagoscopy should eventually be pursued in all patients with tracheal injury to rule out esophageal perforation.

Associated Pathology

- Neurologic trauma including cervical spine and closed head injury
- Related airway pathology
 - Maxillofacial trauma
 - Laryngotracheal hematoma and edema
 - Subcutaneous emphysema of upper airway and epiglottis
- Esophageal injury
- Vascular injury with associated hemodynamic instability

Prevention

Iatrogenic injury during endotracheal intubation can be minimized by ensuring that the tip of a stylet does not protrude past the tip of an ETT.

Special Considerations

- If it is impossible to maintain a patent airway during instrumentation or other stages of the procedure, the femoral artery and vein should be cannulated to permit extracorporeal oxygenation and ventilation prior to proceeding.
- Cricothyroid pressure (Sellick's maneuver) is contraindicated if upper tracheal or laryngeal injury is suspected, because it can cause complete tracheal disruption.

Further Reading

Hurford WE, Peralta R. Management of tracheal trauma. *Can J Anesth*. 2003 Jun;50:R4–R7.

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Index

Note: Page references followed by “f” and “t” denote figures and tables, respectively.

A

Abdominal aortic aneurysm, ruptured, 352
Abscess, epidural, 300–301
Accidental dural puncture, 166–68
Acidosis, 86–88
Acute intermittent porphyria, 119
Acute lung injury (ALI), 314–15
Acute renal failure, 249–51
Acute respiratory disease, management of, 356
Acute respiratory distress syndrome (ARDS), 314–15
Acute transfusion reaction, 128–29
Adenosine, for narrow complex tachycardia, 38
Adrenocortical insufficiency (AI), 88–90
Advanced cardiac life support (ACLS), 214
for bupivacaine cardiotoxicity, 298
Airway
blood in, 9–13
obstruction in the spontaneously breathing patient, 3–4
Airway fire, 2–3
Albuterol
for airway obstruction, 4
for anaphylaxis, 92
for asthma/status asthmaticus, 207
for difficult controlled ventilation, 320

Alkalosis, 90–91
Altered mental status, 240–42
Amantadine, for dystonic reactions, 149
American Academy of Neurology, 82
American Society of Anesthesiologists, 194
Aminophylline, for asthma/status asthmaticus, 207
Amiodarone
for atrial fibrillation, 34
for narrow complex tachycardia, 38
for pediatric advanced life support, 231
for ventricular fibrillation, 61
for wide complex tachycardia, 40
Anaphylaxis, 91–93, 204–6
Anesthesia information management systems (AIMS), 69
Antepartum hemorrhage, 184
Antiemetics, and dystonic reactions, 148
Antihistamines, for anaphylaxis, 205
Antiplatelet therapy, for stroke, 264
Aortic regurgitation, 51–52
Aortic stenosis, 53–54
Apgar score, 226t
Artificial pacing, 268
Aspiration, 4–8
Aspirin
for bleeding after carotid endarterectomy, 339

for myocardial ischemia, 48
for preeclampsia/eclampsia, 192
Asthma, 206–9
Asynchronous pacemaker, 268
Asystole, 26–27
Atrial fibrillation, 33–35
Atropine
for asthma/status asthmaticus, 207
for asystole, 26
for bradycardia, 36
for major trauma, 220
for pediatric advanced life support, 232
for total spinal anesthesia, 311
Automated external defibrillator (AED), 228
Autonomic hyperreflexia, 144–45

B

Bellows ventilators, 72
Benzodiazepine
for local anesthetic toxicity, 180
for porphyria, 121
Benztropine, for dystonic reactions, 149
Bleeding. *See also* Coagulopathy; Hemorrhage
after carotid endarterectomy, 338–40
after thyroid surgery, 340–42
during mediastinoscopy, 364–65
following tonsillectomy, 8–9

- Blind nasal intubation, 270–71
- Blood transfusion.
See also Acute transfusion reaction
refusal of, 78, 80–81
treatment modalities for, 80–81
- Bone cement implantation syndrome (BCIS), 130–31
- Botulinum toxin (Botox) for dystonic reactions, 149
- Bradycardia, 35–37
definition of, 177
fetal, 177–79
- Brain death, declaration of, 81–83
determination of, 82
donation after cardiac death (DCD), 83
pitfalls in, 82
- Breathing circuit malfunction, low pressure condition, 64–66
- Breech presentation, 168–70
- Bronchial blocker (BB) vs. double lumen endotracheal tube, 12t
- Bronchial blocker placement, 271–73
- Bronchial intubation, 13–14
- Bronchopleural fistula (BPF), 365–68
- Bronchospasm, 316–17
- Bupivacaine, for total spinal anesthesia, 311
- Bupivacaine cardiotoxicity, 298–300
- Burns, 131–33, 209–12.
See also Operating room fire
- Butyrophenones, and dystonic reactions, 148
- C**
- Cardiac arrest
maternal, 181–83
- Cardiac herniation after pneumonectomy, 368–71
- Cardiac tamponade, 27–29
- Cardiac trauma, 29–31
- Cardiopulmonary resuscitation (CPR), 227, 228
- Carotid endarterectomy, bleeding after, 338–40
- Chest pain, 242–44
- Clevidipine, for postoperative hypertension, 252
- Clopidogrel, for myocardial ischemia, 48
- Closed head injury, 145
- Coagulopathy, 93–96
- Cocaine, for blind nasal intubation, 270
- Compazine, for postoperative nausea and vomiting, 257
- Congestive heart failure, 31–33
- Continuous positive airway pressure (CPAP), 213
for respiratory depression or failure, 260
- Corticosteroids, for anaphylaxis, 205
- Cricothyroidotomy, 273–75
- Critically ill patient intrahospital transport of, 288–90
laparotomy in, 344–47
- Cyclooxygenase-2 inhibitors (COX-2), for severe postoperative pain, 262
- D**
- Deep vein thrombosis (DVT) prophylaxis, 156
- Demand pacemaker, 268
- Dexamethasone
for airway obstruction, 4
for anaphylaxis, 205
for inhaled foreign body, 373
for stridor, 235
for thyroid storm, 124
- Diabetic ketoacidosis (DKA), 96–99
- Difficult controlled ventilation, 319–21
- Difficult mask ventilation, 17–18
- Digoxin
for atrial fibrillation, 34
for congestive heart failure, 32
- Diltiazem, for narrow complex tachycardia, 38
- Diphenhydramine
for anaphylaxis, 92, 205
for dystonic reactions, 149
- Disseminated intravascular coagulation (DIC), 128
- Dobutamine, for oliguria/acute renal failure, 250
- Donation after cardiac death (DCD), 83
- Dopamine, for bradycardia, 36
- Double lumen endotracheal tube (DLET), 276–78
vs. bronchial blocker, 12t
- Droperidol
and dystonic reactions, 148
for postoperative nausea and vomiting, 257
- Drowning, 212–14
- Drug extravasation, 133–35
- Dysrhythmias
atrial fibrillation, 33–35
bradycardia, 35–37
narrow complex tachycardia, 37–39
wide complex tachycardia, 39–41
- Dystonic reactions, 148–50
- E**
- Eclampsia, 189–92
- Ectopic pregnancy, ruptured, 355–58

- Electric power failure, 66–69
- Embolism, 170–74
- Enalapril, for congestive heart failure, 32
- Endobronchial intubation. *See* Bronchial intubation
- Ephedrine
for bleeding during mediastinoscopy, 364
for bone cement implantation syndrome, 130
for fetal bradycardia, 179
for hypotension, 44
- Epidural abscess, 300–301
- Epidural hematoma (EDH), 152, 153f, 301–4
- Epiglottitis, 215–17
- Epinephrine
for airway obstruction, 4
for anaphylaxis, 92, 204
for asystole, 26
for bradycardia, 36
for bronchospasm, 316
for bupivacaine cardiotoxicity, 298
for cardiac herniation after pneumonectomy, 370
for foreign body inhalation, 219
for hypotension, 44
for laparotomy in the critically ill patient, 345
for local anesthetic toxicity, 307
for neonatal resuscitation, 188, 224
for pediatric advanced life support, 231
for postoperative hypotension, 254
for stridor, 235
for total spinal anesthesia, 311
for ventricular fibrillation, 61
- Erythropoietic porphyria, 119
- Esmolol
for atrial fibrillation, 34
for congestive heart failure, 32
for myocardial ischemia, 48
for pheochromocytoma, 117
for postoperative hypertension, 252
for thyroid storm, 123
- ETCO₂, decreased (intraoperative), 317–19
- Etomidate
for laparotomy in the critically ill patient, 345
for major trauma, 220
for rapid-sequence intubation, 23
- F**
- Facial trauma, 342–44
- Failed intubation, 174–77
- Famotidine, for anaphylaxis, 92
- Femoral vein catheter, 278–79
- Fenoldopam, for pheochromocytoma, 117
- Fentanyl
for burns, 211
and porphyria, 121
for rapid-sequence intubation, 23
for subarachnoid hemorrhage, 158
- Fetal bradycardia, 177–79
- Fetal heart rate (FHR), 177
- Fiberoptic intubation, 279–82
- Focused abdominal sonography for trauma (FAST), 345
- Foreign body, inhalation of
adult, 371–73
pediatric, 217–19
- Furosemide
for congestive heart failure, 32
for difficult controlled ventilation, 320
for hypercalcemia, 100
for hyperkalemia, 101
for hyponatremia, 111
for hypoxia, 246
for oliguria/acute renal failure, 250
for pulmonary edema, 330
for TURP syndrome, 125
- G**
- Gas embolism, 45–47
- Glasgow Coma Scale, 147t
- Globe injury, 304–5
- Glucagon, for bradycardia, 37
- Glycopyrrolate
for asthma/status asthmaticus, 207
for fiberoptic intubation, 280
- H**
- Haemophilus influenzae*, and epiglottitis, 215
- Haldol, and dystonic reactions, 148
- Haloperidol, for altered mental status, 241
- Head injury
agents of, 152
closed, 145
penetrating, 152–55
- Heart failure. *See* Congestive heart failure
- Hematoma. *See* Epidural hematoma; Subdural hematoma
- Hemin, for porphyria, 120
- Hemodialysis, 250
for oliguria/acute renal failure, 250
- Hemoptysis, 322–23
- Hemorrhage
massive, 347–50
maternal, 184–87
postoperative, 49–50
subarachnoid, 157–60
- Heparin
for myocardial ischemia, 48
for pulmonary embolus, 333

- HHH therapy, for subarachnoid hemorrhage, 160
- High spinal anesthesia. See Total spinal anesthesia
- Hydralazine, for postoperative hypertension, 252
- Hydrocortisone
for anaphylaxis, 92
for thyroid storm, 124
- Hyperbaric oxygen therapy
for burns, 133
for venous air embolism, 162
- Hypercalcemia, 99–101
- Hypercarbia, intraoperative, 323–25
- Hyperkalemia, 101–2
- Hypermagnesemia, 102–4
- Hyponatremia, 104–5
- Hypertension, 41–43
intracranial, 150–52
postoperative, 251–53
- Hypocalcemia, 105–7
- Hypokalemia, 107–9
- Hypomagnesemia, 109–10
- Hyponatremia, 110–12
- Hypotension, 43–45
postoperative, 253–55
- Hypothermia, 112–14
- Hypoxemia, 378–80
- intraoperative, 326–27
- Hypoxia, 244–47
- I**
- Increased airway pressure, 380–81
- Informed consent, 78–80
- Insulin, for diabetic ketoacidosis, 97
- Intra-arterial injection, 135–37
- Intracranial hypertension, 150–52
- Intrahospital transport of critically ill patient, 288–90
- Intrathoracic obstruction, 374–77
- Intravenous corticosteroids
for asthma/status asthmaticus, 207
- Intubation inability, 174–77
with ventilation ability, 14–15
with ventilation inability, 16–17
- Ischemia, myocardial, 47–49, 247–49
- K**
- Ketamine
for asthma/status asthmaticus, 207
for burns, 210
for porphyria, 121
for rapid-sequence intubation, 23
- L**
- Labetalol
for chest pain, 244
for hypertension, 42
for myocardial ischemia, 248
for pheochromocytoma, 117
for postoperative hypertension, 252
for thyroid storm, 123
- Laparotomy in critically ill patient, 344–47
- Laryngeal mask airway (LMA), 18
intubation, 282–84
- Laryngospasm, 19–20
- Law of Laplace, 353
- Lidocaine, epinephrine, atropine, and naloxone (“LEAN”), 234
- Lidocaine
for blind nasal intubation, 270
after intra-arterial injection, 136
for local anesthetic toxicity, 307
for major trauma, 220
for pediatric advanced life support, 231
for rapid-sequence intubation, 23
for ventricular fibrillation, 61
for wide complex tachycardia, 40
- Local anesthetics, for severe postoperative pain, 262
- Local anesthetic toxicity, 179–81, 305–8
- Low pressure condition, 64–66
- Ludwig’s angina, 20–22
- M**
- Magnesium sulfate ($MgSO_4$), for preeclampsia/eclampsia, 190
- Major trauma, 219–23
- Malignant hyperthermia (MH), 114–16
- Massive hemoptysis, 9
- Massive hemorrhage, 347–50
- Maternal cardiac arrest, 181–83
emergency cardiac care, 182
- Maternal hemorrhage, 184–87
- Mediastinal obstruction, 374–77
- Mediastinoscopy, bleeding during, 364–65
- Mental status, altered, 240–42
- Metaclopramide, and dystonic reactions, 148
- Methylprednisolone, for asthma/status asthmaticus, 207
- Methyxanthines, for asthma/status asthmaticus, 207
- Metoprolol
for chest pain, 244
for myocardial ischemia, 48, 247, 248
for narrow complex tachycardia, 38
for thyroid storm, 123
- Midazolam
for burns, 211
for fiberoptic intubation, 280
for postoperative hypertension, 251
for rapid-sequence intubation, 23
for subarachnoid hemorrhage, 158

- Mitral regurgitation, 55–56
- Mitral stenosis, 56–58
- Morphine
for myocardial ischemia, 47
for porphyria, 121
for postoperative hypertension, 251
for sickle cell crisis, 122
- Multimodal therapy, for postoperative nausea and vomiting, 257
- Myocardial ischemia, 47–49, 247–49
- ## N
- Naloxone, for hypercarbia, 324
- Narrow complex tachycardia, 37–39
- Nausea and vomiting, postoperative, 255–57
- Near drowning, 212–14
- Nebulized ipratropium, for asthma/status asthmaticus, 207
- Neck injury, 350–52
- Neonatal resuscitation, 187–89, 223–27
endotracheal tube diameters and lengths, 226t
- Nerve injury, 308–10
- Nicardipine
for hypertension, 42
for pheochromocytoma, 117
for postoperative hypertension, 252
- Nifedipine, for autonomic hyperreflexia, 144
- Nonsteroidal anti-inflammatory drugs (NSAIDs), 222
for severe postoperative pain, 262
- ## O
- Occupational exposure, 137–39
- Octreotide, for upper GI bleeding, 360
- Ocular injury, 305
- Oliguria, 249–51
- Ondansetron, for postoperative nausea and vomiting, 256
- One-lung ventilation hypoxemia, 378–80
increased airway pressure, 380–81
- Operating room fire, 139–41. *See also* Burns
- Opioids, for severe postoperative pain, 262
- Oral corticosteroids, for asthma/status asthmaticus, 207
- Organ harvesting, and brain death declaration, 81–83
- Oxygen pipeline failure, 69–72
- Oxymetazoline, for blind nasal intubation, 270
- Oxytocin, for uterine rupture, 201
- ## P
- Pacemakers, anesthetic implications of, 268–70, 268t
- Packed red blood cells (PRBCs), for laparotomy in the critically ill patient, 345
- Panorex films, 343
- Papaverine, for intra-arterial injection, 136
- Patient-controlled analgesia (PCA), 222
- Pediatric advanced life support, 230–34
- Pediatric basic life support, 227–30
- Penetrating head injury, 152–55
- Phenergan, for postoperative nausea and vomiting, 257
- Phenothiazines and dystonic reactions, 148
for postoperative nausea and vomiting, 257
- Phenylephrine, 130
for bleeding during mediastinoscopy, 364
for blind nasal intubation, 270
for bone cement implantation syndrome, 130
for cardiac herniation after pneumonectomy, 370
for fetal bradycardia, 179
for hypotension, 44
for postoperative hypotension, 254
- Phenytoin, for intracranial pressure, 147
- Pheochromocytoma, 116–18
- Physostigmine, for altered mental status, 241
- Piston ventilators, 72
- Placental abruption, 184
- Placenta previa, 184
- Pneumonectomy, cardiac herniation after, 368–71
- Pneumothorax, 328–29
tension, 381–83
- Porphyria, 119–21
types of, 119
- Positive end-expiratory pressure (PEEP), for venous air embolism, 162
- Postoperative hemorrhage, 49–50
- Postoperative hypertension, 251–53
- Postoperative hypotension, 253–55
- Postoperative nausea and vomiting (PONV), 255–57
- Postoperative pain, 261–63
- Postpartum hemorrhage, 184
- Prednisone, for asthma/status asthmaticus, 208
- Preeclampsia, 189–92
- Primary brain injury (PBI), 220

- Procainamide
 for narrow complex tachycardia, 38
 for wide complex tachycardia, 40
- Prochlorperazine, and dystonic reactions, 148
- Prolixin, and dystonic reactions, 148
- Prolonged neurologic impairment after regional anesthesia, 257–59
- Promethazine, and dystonic reactions, 148
- Propofol
 for burns, 210
 for fiberoptic intubation, 282
 for laryngospasm, 20
 for porphyria, 121
 for postoperative nausea and vomiting, 257
 for rapid-sequence intubation, 23
- Pulmonary edema, 330–32
- Pulmonary embolus, 332–34
- Pulseless electrical activity (PEA), 233
- R**
- Rapid sequence induction, 22–24
- Rapid-sequence intubation (RSI), 22–24
- Regional anesthesia, prolonged neurologic impairment after, 257–59
- Regurgitant fraction, 52
- Respiratory depression, 259–61
- Respiratory failure, 259–61
- Respiratory precautions, 334–35
- Retrograde intubation, 284–85
- Rocuronium
 for major trauma, 221
 for rapid-sequence intubation, 23
- Rule of Nines, 132
- Ruptured abdominal aortic aneurysm, 352–55
- Ruptured ectopic pregnancy, 355–58
- S**
- Shoulder dystocia, 192–94
- Sickle cell crisis, 121–23
- Sodium nitroprusside
 for hypertension, 42
 for pheochromocytoma, 117
- Spinal cord injury, 155–57
- Status asthmaticus, 206–9
- Stenosis, aortic, 53–54
- Stevens-Johnson syndrome, 210
- Stridor, 234–37
- Stroke, 263–65
- Subarachnoid hemorrhage (SAH), 157–60
- Subdural hematoma (SDH), 152, 153*f*
- Sublingual nitroglycerin, for chest pain, 243
- Succinylcholine
 for burns, 133
 for major trauma, 221
 for malignant hyperthermia, 116
 for rapid-sequence intubation, 23
- Sudden infant death syndrome (SIDS), 229
- Sufentanil, for porphyria, 121
- T**
- Tachycardia
 narrow complex, 37–39
 wide complex, 39–41
- Tardive dystonia, 150
- Tension pneumothorax, 381–83
- Terbutaline
 for asthma/status asthmaticus, 208
 for bronchospasm, 316
- Theophylline, for asthma/status asthmaticus, 207
- Thiopental
 for burns, 210
 for rapid-sequence intubation, 23
- Thioridazine, and dystonic reactions, 148
- Thoracic aortic dissection, 58–60
- Thoracotomy, right lung isolation for, 365
- Thorazine, and dystonic reactions, 148
- Thyroid storm, 123–25
- Thyroid surgery, bleeding after, 340–42
- Tissue plasminogen activator (t-PA), for stroke, 264
- Tonsillectomy, bleeding following, 8–9
- Total spinal anesthesia, 195–97, 310–12
- Tracheal injury, 384–87
- Transcutaneous pacing, 286–88
- Transesophageal echocardiography, 172
- Transjugular intrahepatic portosystemic shunt (TIPS), for upper GI bleeding, 360
- Transvenous pacing, 290–92
- Traumatic brain injury (TBI), 222
- Trihexyphenidyl, for dystonic reactions, 149
- TURP syndrome, 125–26
- U**
- Ultrasound-guided central venous access, 292–95, 294*f*
- Umbilical cord prolapse, 197–99
- Upper gastrointestinal bleeding, 358–61
- Uterine dehiscence, 217
- Uterine rupture, 199
- V**
- Valvular disease
 aortic regurgitation, 51–52

- aortic stenosis, 53–54
 - mitral regurgitation, 55–56
 - mitral stenosis, 56–58
 - Vasodilators, for upper GI bleeding, 360
 - Vasopressin
 - for asystole, 26
 - for upper GI bleeding, 360
 - for ventricular fibrillation, 61
 - Venous air embolism (VAE), 161–63
 - Ventilation
 - difficult controlled, 319–21through an endotracheal tube, 18–19
 - difficult mask, 17–18
 - Ventilator failure, 72–75
 - Ventricular fibrillation (VF), 60–62, 230
 - Vitamin K, for coagulopathy, 94
- W**
- Warfarin, for coagulopathy, 94
 - Wide complex tachycardia, 39–41
- Z**
- Zavenelli Maneuver, for shoulder dystocia, 193