EMERGENCY MEDICINE:
A Focused Review of the Core Curriculum

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Joel M. Schofer

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Amal Mattu

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ACKNOWLEDGMENTS

During my residency and board preparation, I was frustrated by the lack of an affordable, unifying resource that provided an appropriate level of detail, asked board-style questions, and included all the required images and figures without referring readers to other documents. It is my hope that through our efforts this unifying resource finally exists. I hope readers find it useful in their studies.

No one involved in the preparation of this book has received or will receive any monetary reward. All the effort involved was for the benefit of the American Academy of Emergency Medicine Resident and Student Association.

Linda Kesselring, our copy editor, was invaluable and of the utmost importance to this project. Her attention to detail enhanced the continuity among the chapters and the readability of the text. Linda’s refinement of the manuscripts was a considerable contribution to the quality of the presentation of this book.

Special thanks go to Andreas Alfer, our images editor, who compiled and formatted the collection of photos presented as Chapter 22, one of the most valuable components of this book.

Jody Bath (Communications and RSA Program Manager), Janet Wilson (AAEM/RSA Executive Director), and Laura Ludwig (Director of Creative Services at AAEM) applied their admirable skills to the layout and design of this text and extended inordinate patience during our many e-mail exchanges during the page-proof stage of production.

Finally and most importantly, during the two years spent working on this book, I was reminded what truly matters in life. I thank my wife, Wendy, and my children, Nicholas and Erin, for their patience and support during the hours I spent staring at a computer screen. I look forward to more time sharing red wine on the porch, playing Barbies, and building trains.

Joel M. Schofer, MD, FAAEM
Editor-in-Chief
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PREFACE

Anyone who practices emergency medicine has a healthy respect for the breadth of knowledge required to practice this specialty. The health care provider must be knowledgeable about the urgencies and emergencies that span the entire spectrum of medicine. Moreover, this core knowledge must include more than just emergencies but also benign conditions and everything in between, from costochondritis to myocardial infarction, from gastroenteritis to mesenteric infarction, from tension headache to subarachnoid hemorrhage. Not surprisingly, attempts to master this core curriculum often cause significant angst for young practitioners and for those studying for certification and recertification examinations.

Numerous textbooks have been written for emergency practitioners interested in reviewing the curriculum. Unfortunately, over time, these textbooks have expanded to become reference books rather than review books. Practitioners interested in a focused review of the essentials of the core curriculum have been lacking a concise text for clinical and examination review. Emergency Medicine: A Focused Review of the Core Curriculum was created to meet this need. The chapters have been adapted from the syllabus of the American Academy of Emergency Medicine’s Written Board Review Course. A brief outline format has been retained in order to ensure the “readability” of the text, and the authors and editors refrained from including esoterica and details of pathophysiology, which would have increased the length of the chapters and quickly turned the text into yet another reference book. This text is intended to be one that can be read cover-to-cover in a short period of time. It is our sincere hope that this text does not become just another book relegated to dusty bookshelves, only to be looked at from afar but never actually read.

The authors and editors have focused their efforts on creating a text that is relevant not only to practicing clinicians and those studying for certification and recertification examinations but also to residents interested in reviewing the core curriculum for in-training examinations and medical students interested in an easy-to-read curriculum primer for their emergency medicine rotations.

Many thanks go to the contributors of the chapters, all of whom are all accomplished educators. Thanks also go to the American Academy of Emergency Medicine (AAEM) and to their Written Board Review Course speakers and course directors for their critical contributions and support of this text. Many thanks also to the medical editors and copy editors for their hard work and attempts to maintain consistency between the chapters. Special thanks go to Linda Kesselring for her incredible copy-editing skills and dedication to this project. Finally, I’d personally like to recognize the tremendous work of Editor-in-Chief Joel Schofer. This text could not have been completed had it not been for Dr. Schofer’s passion and commitment to this project during the past two years.

On behalf of the AAEM Resident and Student Association, I extend best wishes and hopes that this textbook helps to improve your patient care and your ability to prepare for upcoming certification and recertification examinations.

Amal Mattu, MD, FAAEM
Senior Associate Editor
FOREWORD

As a founding board member of the American Academy of Emergency Medicine, I am honored and truly pleased to be able to write the foreword for *Emergency Medicine: A Focused Review of the Core Curriculum*, a product of the AAEM Resident and Student Association. When AAEM was formed in 1993, the board had a vision of an organization focused on securing a better future for EM residents. We now see the AAEM Resident and Student Association, through efforts like this publication and other activities, “giving back” to that mission by helping their colleagues become better physicians. The intent of the original board—to join together to help our colleagues as best we could—now permeates the organization from student to emeritus member.

The other central intent of the founders of the Academy was to create an organization that could effectively promote the simple concept that board certification is an essential element of the title “specialist” in emergency medicine. As a tool to help our colleagues reach that goal, this book is also true to the original mission. With the leadership of Joel Schofer and the able guidance of Amal Mattu, this book further strengthens the Academy’s record of service to its members and the specialty in the area of education.

The impressive record of the educational contributions of AAEM to the specialty was not foreseen at the time of its creation but is a truly wonderful by-product of its formation. I commend the efforts of Joel and Amal and all those who contributed to this work. It is very encouraging to see AAEM members doing today exactly what was planned at the beginning of the Academy—helping our colleagues who then in turn can better serve their patients.

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INTRODUCTION:

Emergency Medicine Examinations

Joel M. Schofer, MD

From the time you start your emergency medicine (EM) residency until you near retirement from active practice as an emergency physician, you will prepare for and take many certification examinations. This book can help you prepare for all of them. This introduction provides an overview of the timing, content, and format of each examination and offers advice intended to guide your study strategies.

During Residency: The Inservice Examination
The American Board of Emergency Medicine (ABEM) in-training examination (the “inservice exam”) is offered on the last Wednesday of February by most EM residencies (although they are not required to participate). It consists of 225 multiple-choice questions and takes 4.5 hours to complete. Unlike the qualifying examination, it is still taken with paper and pencil. It tests the expected knowledge base of a third-year EM resident and, like the ABEM qualifying examination, draws its content from The Model of the Clinical Practice of Emergency Medicine (the EM Model).

In my opinion, this examination is the closest thing you will get to a true practice test for the ABEM written board examination. It should be used as a guide for how well you are preparing for the qualifying examination. As stated at the ABEM website:

There is a strong relationship between in-training and qualifying examination scores. Physicians with higher in-training scores have a higher likelihood of passing the qualifying examination and those with lower scores have a lower likelihood of passing the qualifying examination.

After Residency: The Written Boards
Formerly known as the written certification examination, the ABEM qualifying examination (still commonly referred to as the “written boards”) is offered annually in late October or early November. It covers the breadth of emergency medicine, as defined by the EM Model, and takes up to 8 hours to complete, including up to 6.5 hours of testing time. It consists of approximately 305 single-best-answer multiple-choice questions, with 10% to 15% utilizing accompanying pictorial stimuli. The stimuli can take many forms, the most common being electrocardiograms, radiographs, ultrasounds, CT scans, and photographs of medical conditions. This is a computerized test offered at Pearson VUE computer testing centers.
The content of the qualifying examination is weighted as detailed below (source, www.abem.org):

<table>
<thead>
<tr>
<th>Listing of Conditions and Components</th>
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<tr>
<td>1.0  Signs, Symptoms, and Presentations</td>
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<td>2.0  Abdominal and Gastrointestinal Disorders</td>
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<td>3.0  Cardiovascular Disorders</td>
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<td>4.0  Cutaneous Disorders</td>
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<td>5.0  Endocrine, Metabolic, and Nutritional Disorders</td>
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<td>6.0  Environmental Disorders</td>
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<td>7.0  Head, Ear, Eye, Nose, and Throat Disorders</td>
<td>5%</td>
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<td>8.0  Hematologic Disorders</td>
<td>2%</td>
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<td>9.0  Immune System Disorders</td>
<td>2%</td>
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<td>10.0 Systemic Infectious Disorders</td>
<td>5%</td>
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<tr>
<td>11.0 Musculoskeletal Disorders (Non-traumatic)</td>
<td>3%</td>
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<td>12.0 Nervous System Disorders</td>
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<td>13.0 Obstetrics and Gynecology</td>
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<td>14.0 Psychobehavioral Disorders</td>
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<td>15.0 Renal and Urogenital Disorders</td>
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<td>16.0 Thoracic-Respiratory Disorders</td>
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<td>Lower Acuity</td>
<td>27%</td>
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<tr>
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A minimum of 8% of the questions will be related to the care of pediatric patients and a minimum of 4% to the care of geriatric patients.

All candidates achieving a score of 75 or higher pass the examination. The scores are released by mail within 90 days after administration of the examination. Since 1995, the qualifying exam was passed by 90% of EM-residency–trained candidates when taking it for the first time, with annual passing rates ranging from 88% to 93%.
After the Written Boards: The Oral Boards
After you pass the qualifying examination, you must take the ABEM oral certification examination (“oral boards”). The oral certification examination, offered twice a year, examines the candidate’s ability to apply emergency medicine knowledge to patient scenarios based on actual clinical cases. There are seven scenarios in total—five single-patient encounters and two multiple-patient encounters, in which you must manage multiple simulated patients simultaneously.

The content of the oral examination is also based on the EM Model, but the test emphasizes cardiovascular, traumatic, and toxicologic content. Pediatrics will be emphasized as well. Approximately two thirds of the simulated patients will be critical and one third will be emergent. Of the seven cases, one of the single-patient encounters is a test case and will not count toward your score.

Scoring of the examination is a complex process. Check the ABEM website (www.abem.org) for specific details. The passing rate for EM-residency–trained first-time test takers since 1995 is 92%, with annual pass rates ranging from 86% to 97%. The results are mailed within 90 days after the examination is complete.

You Passed…Now What?
The process for maintaining ABEM board certification involves multiple components, but only two examinations are required. One is the Lifelong Learning and Self-Assessment, an on-line “open book” test based on annual readings selected by ABEM. These annual self-assessments do not require “study,” so this board review book is not designed to help you prepare for them.

The second required examination is the Continuous Certification (ConCert) examination. This is a half-day computer-based examination typically taken every 10 years after initial board certification. It is similar to the qualifying examination in that it is based on the EM Model. This board review book should serve as an excellent study guide for the ConCert examination.

ABEM.org
There is a wealth of information on all of the tests discussed above at ABEM’s website, www.abem.org. I encourage you to visit the website and read more information as you prepare to take a specific test.

How Should I Study?
Some EM board review books lay out specific schedules for study over varying time frames. I will simply give you a little advice based on common sense and my own experiences.

Know Thyself
Since you are reading this book, you have probably completed high school, college, and a majority (if not all) of medical school. You have taken countless tests, attended seemingly millions of lectures, and read hundreds of books. By now, you should have some idea of how you learn. I, for instance, learn much more effectively by reading than by attending a lecture. Other people learn better by listening than by reading.

Take a moment to think about it. Do you learn from reading, from listening to lectures, or from hands-on practice? Whichever is your most efficient method of learning, make that the focal point of your test preparation. I’d love to say that everyone will learn best by reading this book, but that is not true. Use what works best for you.

Use the Inservice Examination
The inservice examinations are the closest thing you will get to the written board exam. If you are studying for the written boards and you “aced” the inservice exams, you probably don’t need to study much, if at all. You will, in all likelihood, pass with flying colors.

On the other hand, if you scored low or marginally on the inservice examination, then you need to study as much as you can to minimize your risk of failing.
If you are like most people and scored between the 25th and 75th percentiles on the inservice exam, then study as much as you feel you need to or reasonably can. More preparation is certainly better than less; it is difficult to you tell you exactly how much to study.

If you’re not sure about your learning style, or if you never took the inservice exams, then ask your residency director to advise you as to how to prepare for the exam. He/she should have a good idea of how much you need to study.

*If You are Studying for the Oral Boards, Practice as Many Cases as Possible*

Do not rely only on this book to prepare for the oral boards. I don’t care how smart you are, the oral boards can be daunting. You know you are well prepared because you already passed the qualifying examination, but there is something unnerving about the unknown case behind Door Number 1. Practicing cases, whether during an evening with your buddies who are also studying for the exam or at a formal course, is invaluable. Even though my friends and I had practiced over 40 cases together before our oral boards, we kept practicing them even after we had become confident. The last one we did as practice was on the exam. Do as many as you can.

*Make Chapter 20, the Rapid Board Review, the Final Thing You Read*

Chapter 20 presents a rapid review of topics commonly encountered on the boards. If you read nothing else in this book, read Chapter 20 before you take any of the certification tests. You won’t regret it.
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CHAPTER 1

Traumatic Disorders

David E. Manthey, MD, and Bret A. Nicks, MD

1.1 TRAUMA OVERVIEW

Epidemiology
- Leading cause of death between the ages of 1 and 44
- Motor vehicle crashes account for >50,000 deaths/year (more than half involve alcohol)
- Trimodal distribution of trauma deaths
  - Immediate (within minutes)
    - Greatest number of fatalities
    - Neurotrauma (head or spinal cord)
    - Massive exsanguination (rupture of great vessels)
    - Airway compromise
  - Early (within hours)
    - Origin of the “golden hour” concept
    - Target group of emergency medical services (EMS) and trauma systems
    - Deaths in this group are caused by
      - Hemopneumothorax
      - Tension pneumothorax
      - Cardiac tamponade
      - Subdural/epidural hematoma
      - Massive hemorrhage
  - Late (days to weeks)
    - Multiple organ dysfunction; sepsis

Mechanism of Injury
- Motor vehicle crashes
  - Frontal crash
    - Most common type of motor vehicle crash
    - Cervical spine injuries in unrestrained occupants
    - Posterior hip dislocations for front-seat occupants
o Seat belts
  ▪ Half-belted practice (shoulder only without the lap belt component) results in liver/spleen injuries
  ▪ Lap belt only – lumbar spine and small intestine injury
o T-bone crash
  ▪ Second most common type of motor vehicle crash
  ▪ Intrusion of at least 6 inches is associated with a 16% risk of significant injury
o Roll-over
  ▪ Gradual dissipation of energy reduces risk of injury as long as lower speed, no single defining impact, and no ejection
o Auto–pedestrian
  ▪ Run “under” not over
    □ Tibia (most common)
    □ Pelvis
    □ Head

• Firearms
  o Handguns are considered low velocity (<1000 feet/sec)
  o Hunting and military rifles are high velocity
  o Extensive internal damage due to kinetic energy
    ▪ Cavitation
    ▪ Sonic pressure wave
    ▪ Velocity, yaw, deformation, and fragmentation determine wound
  o Close-range shotgun
    ▪ Massive injury, contamination

• Falls
  o Major cause of death in pediatrics
  o Most common cause of injury in the United States
  o Average floor is 12 feet
    ▪ The median lethal dose (LD50) is 4 floors (6 floors in children)
    ▪ The lethal dose for 90% of test subjects (LD90) is 7 floors (84 feet)
  o Upright impact is associated with calcaneal fractures; thoracolumbar spine fractures; pelvic fractures; and severe, comminuted leg and femur fractures.

• Domestic violence
  o Leading cause of injury to women aged 15 to 44
  o 30% to 50% of female victims are killed by current or former partner
  o Affects all racial and socioeconomic backgrounds
  o Clues
    ▪ Cover story
    ▪ Ever-present, overly concerned, or domineering partner
    ▪ Defensive wounds or assault to abdomen when pregnant
  o Reporting is mandatory if the injury was caused by a gun, knife, or deadly weapon.
  o Routine screening is essential.
• Strangulation
  o Crushed larynx, fractured hyoid, carotid-intimal injury
• Cave-in (e.g., mining accidents)
  o Asphyxia
  o Environmental inhalation injury
• Bicycle handlebar
  o Intramural duodenal hematoma; bowel injury
  o Pancreatic injury

1.2 ADVANCED TRAUMA LIFE SUPPORT (ATLS) GUIDELINES

Primary Survey (ABCs) – Assess and stabilize life-threatening issues.
• Airway (with C-spine precautions)
  o Single most important prehospital/arrival therapy
  o Assume C-spine injury in patients with multisystem trauma, especially those with injury above the clavicle or with altered mental status.
  o Assessment
    ▪ Patency
    ▪ Maintainability
  o Intubate trauma patients for
    ▪ Inability to oxygenate despite oxygen
    ▪ Inability to ventilate
    ▪ Inability to clear secretions or blood
    ▪ Head injury with Glasgow Coma Scale score <8
    ▪ Impending airway compromise (hematoma, swelling)
  o Technique
    ▪ Ootracheal intubation (with in-line immobilization) is the procedure of choice (even with suspected C-spine injury).
    ▪ Nasotracheal intubation is an option in patients older than 12 years of age who are breathing.
      □ Contraindications to nasotracheal intubation: apnea, mid-face fractures, basilar skull fracture
    ▪ Surgical cricothyroidotomy
      □ May be necessary in the case of unsuccessful orotracheal intubation
      □ Procedure of choice in patients with severe mid-facial injuries
    ▪ Endotracheal tube placement verification
      □ Most reliable: end tidal CO₂
      □ “Seeing” the tube go through cords
      □ Listening over the axillae for breath sounds
      □ Chest radiograph to confirm distance into trachea
  o Pediatric airway considerations
    ▪ Cuffed tracheal tubes can be used in all except newborns, provided appropriate tube size and cuff pressure are used.
    ▪ For patients younger than 12 years of age, needle cricothyroidotomy is preferred over surgical cricothyroidotomy.
• Breathing
  o Oxygenation
    ▪ Pulse oximeter to determine adequacy
    ▪ Administer oxygen (FIO₂ >85%) to all trauma patients
  o Ventilation
    ▪ Assess depth, rate, and pattern to determine adequacy
    ▪ Correct any injuries that may impede oxygenation or ventilation
      □ Tension pneumothorax
        — Needle thoracostomy followed by tube thoracostomy
      □ Sucking chest wounds
        — Three-sided seal
      □ Hemothorax/pneumothorax (see Image #3)
        — Tube thoracostomy
      □ Pulmonary contusion – positive-pressure ventilation
    ▪ Ventilator setting adjustments in trauma
      □ Low tidal volumes – 5 to 7 cc/kg
      □ Respiratory rate ≥ 20 breaths/min
• Circulation with hemorrhage control
  o Shock is defined as inadequate tissue perfusion and oxygenation.
  o Assess for decreased blood pressure, narrowed pulse pressure, capillary refill >2 seconds, decreased urine output, diminished peripheral pulses, and increased heart rate.
  o Elderly patients, athletes, and those on rate-control medications may not develop tachycardia.
• Mental status
  o Alteration is assumed to be caused by poor perfusion of brain
  o Treatment
    ▪ 2 to 3 liters of Ringer’s Lactate (or normal saline)
    □ 20 cc/kg in children
    ▪ Partial responders or non-responders should be given blood.
    ▪ Control all external hemorrhage.
      □ Direct pressure or pneumatic splint
      □ A trauma pelvic orthotic device (an external corset-like device designed to mechanically stabilize major pelvic ring injuries) or a sheet wrapped around the pelvis may be used to close open-book pelvic fractures.
    ▪ Pericardiocentesis under ultrasound for temporary relief of pericardial tamponade, followed by definitive pericardial window
  o Consider ED thoracotomy for penetrating thoracic trauma, victims who lose vitals in the ED, those who arrive recently pulseless with myocardial activity, and blunt trauma victims who experience cardiac arrest in the emergency department. (ATLS guidelines state that a surgeon must be present.)
    The survival rate when a thoracotomy is performed in blunt thoracic trauma victims without signs of life prior to arrival at the hospital is almost zero.
• Disability
  o Pupil function: size, reactivity, and symmetry
  o Glasgow Coma Scale (GCS)
  o Motor function, rectal tone to establish spinal cord function
  o Sensory level if motor function is abnormal

1.3 HEAD TRAUMA

General
• Accounts for half of all trauma deaths
• Males, ages 15 to 30: motor vehicle crashes, assaults, falls, bicycles
• When evaluating a patient with serious head trauma, rule out cervical injuries.
• An understanding of head trauma physiology is essential.
  o Cerebral perfusion pressure (CPP) = mean arterial pressure (MAP) – intracranial pressure (ICP)
  o Autoregulation: constant blood flow despite changes in intracranial pressure

Intracranial Injury
• Unca l herniation
  o Compression of cranial nerve (CN) III, which presents clinically as an ipsilateral fixed, dilated pupil
  o Compression of ipsilateral corticospinal tract presents clinically as contralateral hemiplegia, which eventually progresses to bilateral decerebrate posturing.
  o Kernohan’s notch syndrome – occurs in as many as 25% of patients; the contralateral cerebral peduncle is forced against the opposite edge of the tentorial hiatus
    ▪ Hemiparesis is noted ipsilateral to the dilated pupil and the mass lesion.
• Central transtentorial herniation
  o Less common than uncal herniation
  o Progression of rostrocaudal neurologic deterioration, resulting in increased motor tone, decorticate posturing followed by decerebrate posturing, and a progression of respiratory symptoms culminating in respirator arrest
• Cerebellotonsillar herniation
  o Cerebellar tonsils herniate downward through the foramen magnum
  o Pinpoint pupils
  o Flaccid quadriplegia
  o Mortality approaches 70%
• Upward transtentorial herniation
  o Secondary to an expanding posterior fossa lesion
  o Rapid decline in LOC
  o Pinpoint pupils
  o Downward conjugate gaze accompanied by absence of vertical eye movements

Scalp Lacerations/Avulsions
• Scalp has a rich blood supply and therefore can bleed profusely.
• Carefully palpate lacerations for possible underlying fractures.
• Shaving hair prior to repair is not necessary (increases infection rate).
• If possible, close wide galeal defects with buried sutures to prevent sub-galeal hematoma and scar retraction.
Skull Fractures
- CT scan is the imaging study of choice.
- Skull films have limited utility.
- Nondepressed linear skull fractures – no specific treatment
- Depressed and open fractures require operation.
- Basilar skull fractures
  - CSF rhinorrhea/otorrhea (ring sign or halo on filter paper)
  - Hemotympanum
  - Battle’s sign: retroauricular/mastoid ecchymosis (see Image #52)
  - Raccoon’s eyes: infraorbital or periorbital ecchymosis
  - Up to approximately 20% may develop meningitis

1.4 FACIAL FRACTURES

General
- Up to 60% of patients with significant facial trauma have trauma to other organs.
- In urban areas, up to 70% of facial fractures are secondary to assault.
- Maxillofacial trauma is associated with elder/child abuse and domestic violence.
- The most common facial fracture is nasal, then mandibular.
- 25% to 50% of patients with facial fractures have associated brain injury
- With isolated facial trauma, associated C-spine injury is rare (1%–4%).

Dental
- Ellis classification of dental fractures
  - Class I – enamel only
    - No hot/cold sensitivity
  - Class II – enamel and dentin
    - Hot/cold sensitivity
    - Needs to be covered; follow up in 24 hours
  - Class III – enamel, dentin, and pulp
    - Pink tinge or drop of blood at fracture site
    - Immediate dental referral

Le Fort Fractures
- Le Fort I: palate-facial disjunction
  - Horizontal fracture of the maxilla
  - The upper alveolar ridge of the maxilla is mobile.
- Le Fort II: pyramidal disjunction
  - Fractures through nasal bones and infraorbital rim
  - Nose, hard palate, and upper teeth mobile as a unit
- Le Fort III: craniofacial disjunction
  - Fractures through zygomaticofrontal suture and frontal bone
  - Entire midface, including zygomas, is mobile
- Nonsymmetric Le Fort fractures are common
Mandibular Fractures
- The mandible is the second most commonly injured facial bone.
- The mandible, a ring structure, frequently fractures in two places (in >50% of cases).
- Most common sites: condyle (36%), body (21%), and angle (20%)
- Malocclusion of teeth and inability to bite down imply a mandibular fracture
- Plain x-ray films (Towne’s view) or Panorex films are usually adequate for diagnosis.
- Open mandibular fractures need antibiotics directed toward oral flora (penicillin or clindamycin is commonly used).

Orbital Fractures
- Fracture of the orbital floor is most common.
  - Pain and diplopia on upward gaze
  - Enophthalmos
  - Hypoesthesia of infraorbital nerve (ipsilateral cheek and lip)
  - Limited upward gaze (entrapment of inferior rectus muscle and fat) (see Image #63)
- Plain radiograph (Water’s view)
  - Air fluid level in maxillary sinus
  - Tear drop (soft tissue into maxillary sinus)
  - Disruption of floor
  - Orbital emphysema
  - Facial CT better defines fracture and associated injury
- Medial wall fracture
  - Epistaxis
  - Emphysema of lids or conjunctiva
  - Limitation of lateral gaze (rare, entrapment of medial rectus muscle)
- Radiograph
  - Unilateral clouding of ethmoid sinus
  - Orbital emphysema
- Treatment
  - 30% of patients with these fractures have associated ocular injuries
  - Decongestants and antibiotics
  - NO blowing their nose
  - Refer to ophthalmology (surgery is infrequently needed).

1.5 OPTHALMOLOGIC TRAUMA

Corneal Abrasions/Lacerations
- Signs of corneal perforation (full thickness)
  - Loss of anterior chamber depth
  - Tear-drop shaped pupil
  - Blood in anterior chamber
  - Seidel’s sign (leaking aqueous humor washes away the fluorescein stain, producing the appearance of a flowing stream)
- Full-thickness lacerations require operative repair.
• Superficial partial thickness (see Image #20)
  o Cycloplegic
  o Topical antibiotic

Corneal Burns
• Topical anesthetic and immediate copious irrigation (until pH is 6–8) are the initial treatment for any chemical burn.
  o Acids → coagulation necrosis, shallower burns
  o Alkalis → liquefaction necrosis, deeper burns, worse prognosis
  o Ultraviolet → UV keratitis
• Pain, photophobia, foreign body sensation 6 to 12 hours after exposure
• Fluorescein staining shows numerous punctate corneal “microdots”
• Use analgesics, cycloplegic, eye ointment, patch overnight

Eyelid Lacerations
• Simple lacerations may be repaired in the ED
• Full-thickness lacerations; those involving the lid margins, levator muscle, or canthal tendons; and those with possible lacrimal duct involvement need an ophthalmology consultation.

Hyphema (see Image #58)
• Elevate head of bed to assess blood level
• Consider co-morbid risks for increased bleeding
• Management is dictated by the grade; discuss with Ophthalmology
• Clinical grading system for traumatic hyphemas is preferred:
  o Grade 1 – layered blood occupying less than one third of the anterior chamber
  o Grade 2 – blood filling one third to one half of the anterior chamber
  o Grade 3 – layered blood filling one half to less than the total anterior chamber
  o Grade 4 – total clotted blood, often referred to as blackball or 8-ball hyphema

Lacrimal Duct Injuries
• Most frequently occur with naso-orbital fractures and soft-tissue lacerations
• Injury medial to the punctum suggests injury to the lacrimal duct.
• Repair prevents epiphora and dacryocystitis.

Penetrating Globe Injuries (see Image #73)
• Globe penetration is suggested by a penetrating mechanism, the presence of a foreign body, flat anterior chamber, hyphema, pupil defect, new vision loss, and difficult fundoscopic examination.
• “Waterfall” of aqueous fluid from the wound may wash away fluorescein (Seidel test).
• Consider radiograph or CT scan to evaluate for the presence of a radiopaque foreign body in or behind the globe.

Traumatic Iritis
• Pain upon pupillary motion after blunt trauma
• Direct and consensual photophobia
• Cell and flare on slit lamp examination
• Treat with topical cycloplegics, analgesics, outpatient referral
1.6 OTOLOGIC TRAUMA

Hematoma
- Auricular hematomas can result in necrosis of the underlying cartilage and “cauliflower ear” deformity.
- Hematomas should be incised and evacuated, with dressings secured for 24 hours, with a recheck at that time.

Perforated Tympanic Membrane
- Assess for secondary trauma.
- Most (68%) heal at 1 month; 94% at 3 months
- Strict dry ear precautions with ENT follow-up
- Evaluate hearing at 2 to 3 months if problems persist.

1.7 NECK TRAUMA

Laryngotracheal Injuries
- Commonly caused by a direct blow to the anterior neck
- Signs/symptoms: dysphonia, hoarseness, stridor, dyspnea, subcutaneous air, tracheal deviation
- Tracheal disruption commonly occurs at the cricoid area.
- Intubation options include over bronchoscope or immediate tracheostomy.
- Plain x-ray films plus CT scan are appropriate in stable patients.
- Combined esophageal and airway injuries occur in 20% to 50% of patients with penetrating laryngotracheal injuries.

Penetrating Neck Trauma
- 5% to 10 % of all traumatic injuries
- If the platysma muscle is violated, injury to deep structures should be suspected and surgical consultation is mandatory.
- Signs
  - Hemoptysis/hematemesis
  - Dyspnea
  - Dysphonia/dysphagia
  - Subcutaneous air
  - Focal neurologic deficits
  - Expanding hematoma
  - Severe active bleeding
  - Decreased radial pulse due to injury of subclavian artery
  - Vascular bruit or thrill
  - Airway obstruction
- Bleeding should be controlled by direct pressure or operative exploration.
  - Blind clamping is discouraged – risk of damage to other structures
- In stable patients, the diagnostic approach is based on three anatomic zones:
  - Zone 1
    - Sternal notch to cricoid cartilage
    - Angiography, bronchoscopy, endoscopy
o Zone 2 (see Image #21)
  ▪ Cricoid cartilage to angle of mandible
  ▪ Classically, surgical exploration is required.

o Zone 3
  ▪ Angle of mandible to base of skull
  ▪ Angiography, laryngoscopy

Vascular Injuries
  • Carotid artery
    o Most commonly injured artery in the neck (22%)
  • Vertebral artery (1.3%)
    o Significant injuries may be clinically occult, but signs include
      ▪ Expanding or pulsatile hematoma
      ▪ Bruit
      ▪ Pulse deficit
      ▪ Hemothorax
      ▪ Neurologic deficits (especially involving cranial nerves)
  • Horner’s syndrome has been associated with vascular injury.
  • Injuries may present as neurologic deficits similar to stroke; symptoms may develop weeks later.
  • Diagnosis by angiography or MRI/MRA
  • Medical management (anticoagulation, platelet inhibition) is usually adequate.

o Although it is counterintuitive to prescribe anticoagulation for a patient with a traumatic dissection, the purpose is to prevent a clot from forming at the area of injury to the endothelial lining. The dissection will most commonly heal itself without intervention.

1.8 CHEST TRAUMA

General
  • Thoracic trauma accounts for 25% of civilian trauma deaths.
  • Most chest injuries (85%–95%) do not require thoracotomy-operative repair.
  • Hypoxia is the most serious threat associated with chest trauma.
  • Penetrating chest injuries frequently produce pneumothorax, and 75% are associated with hemothorax.
  • Place subclavian lines on the side of the thoracic injury, unless vascular injury is suspected.

Aortic Dissection/Disruption
  • Associated with blunt trauma, high-speed deceleration mechanisms
  • Most common site (90%) is just distal to the left subclavian artery at the ligamentum arteriosum.
  • 80% die at scene; 50% of remainder die within 24 hours if not repaired
  • May have no external evidence of thoracic trauma (one third of patients)
  • Diagnosis
    o Symptoms
      ▪ Retrosternal or interscapular pain (25%)
      ▪ Dyspnea
Exam
- Frequently unrevealing
- Pulse deficits
- New harsh systolic murmur over the precordium
- Pseudocoarctation – isolated upper extremity hypertension with decreased or absent femoral pulses
- Voice changes or hoarseness without laryngeal injury
- Paraplegia

Chest radiograph (see Image #31)
- Superior mediastinal widening (8.0–8.5 cm) on upright PA (most common)
- Rightward deviation of esophagus/trachea at T4
- Obliterated or indistinct aortic knob
- Displaced left main bronchus >40 degrees below horizontal
- Widened paratracheal stripe or deviation to the right
- Widening of paraspinous stripes (left or right)
- Left apical pleural cap
- Up to one third have a normal initial chest radiograph.

Contrast enhanced dynamic CT scan
- Accurate
- Mediastinal hematoma

Transeosophageal echocardiography (TEE)
- Demonstrates lesion and hematoma

Angiography
- Gold standard, demonstrates lesion

Management
- Operative repair
- Immediate medical management with β-blockers (to reduce shear forces), then nitroprusside to maintain a systolic BP between 90 and 100 mm Hg while preparing for OR

Contusion
- Cardiac
  - Usually results from high-speed vehicular trauma
  - Signs/symptoms
    - Tachycardia, dysrhythmias, conduction delays, cardiogenic shock
    - Standard cardiac enzymes are nonspecific and not clinically helpful.
  - Echocardiography is poor as screening test, but helpful if cardiogenic shock or dysrhythmias are present.
  - Provide fluid/pressor support; treat dysrhythmias as needed.
- Pulmonary
  - Results from direct chest wall trauma
  - Signs/symptoms
    - Opacifications of lung are usually present on arrival, always within 6 hours after injury.
    - Arterial blood gas shows hypoxemia and widened A-a gradient.
CHAPTER 1 • Traumatic Disorders

- Treatment
  - Analgesics, good pulmonary toilet important to avoid atelectasis and pneumonia
  - Anticipate mechanical ventilation if
    - >28% of lung volume (by chest radiograph)
    - More than one lobe is involved
  - Utilize intermittent mandatory ventilation (IMV)
    - Lower tidal volumes (5–7 cc/kg)
    - PEEP to prevent alveolar collapse
    - Normal lung down (improves ventilation perfusion matching to injured lung)
  - Pneumonia is the most common and most significant complication.

Fracture
  - Clavicle
    - 5% of fractures
    - Most commonly fractured bone during childhood
    - Fractures of middle third are most common (80%) due to direct force to the lateral aspect of the shoulder.
    - Fractures of medial third (5%) occur due to direct blow to chest.
    - Fractures of lateral third (15%) occur due to blow on top of shoulder.
  - Ribs/flail chest
    - Lower rib fractures may be associated with abdominal injuries.
    - Analgesics and good pulmonary toilet are important to avoid atelectasis and pneumonia.
    - Flail chest (three or more adjacent ribs with segmental fractures)
      - Segment shows paradoxic motion during respiration.
      - Increases work of breathing, painful, loss of negative inspiratory pressure, underlying lung contusion
      - Treatment: pain control, intubation, positive-pressure ventilation
  - Sternum
    - Due to anterior blunt trauma, usually chest strikes steering wheel
    - Sternal fracture is more likely if restrained than unrestrained
    - Increased three-fold since the use of across-the-shoulder belts
    - Isolated sternal fractures are benign with low mortality (<1%)
    - Myocardial contusion in 1% to 6%
    - No association with aortic rupture
    - Mediastinal hematoma can lead to acute blood loss and compression of adjacent structures.
    - Lateral radiographic view of sternum is diagnostic.
    - CT shows associated mediastinal hematomas and injuries.

Hemothorax
  - Exam
    - Decreased breath sounds, dullness to percussion
    - Upright chest radiograph shows as little as 200 to 300 ml as blunting of costophrenic angles.
    - Supine chest radiograph shows diffuse haziness.
    - 25% have concomitant pneumothoraces (see Image #3)
    - Most are self-limited.
• Treatment
  o Large-bore (36+ Fr) chest tube
  o Indications to go the operating room
    ▪ Initial drainage of 1500 ml
    ▪ Bleeding or ongoing bleeding >200 ml/hr for 2 to 4 hr
    ▪ Persistent hypotension
    ▪ Persistent air leak or failure of lung to re-expand
  o Auto-transfusion of shed blood is possible with appropriate equipment.

Penetrating Chest Trauma
• Consider traumatic injury to lungs, heart, diaphragm, airway, vasculature
• Open thoracotomy
  o Penetrating trauma and loss of vital signs in emergency department
  o Incision at fifth intercostal space (ICS); open pericardium vertically, anterior to phrenic nerve

Pericardial Tamponade
• More common in penetrating trauma
• Beck’s triad: hypotension, jugular vein distension (JVD), muffled heart sounds
• Pulsus paradoxus may be present; electrical alternans is rarely seen in acute pericardial tamponade.
• Bedside ultrasound confirms the clinical diagnosis.
• Treatment: pericardiocentesis, thoracotomy

Pneumothorax
• Simple pneumothorax – collection of air in pleural space
  o Clinical exam is insensitive for detecting pneumothorax.
  o Chest radiographs PA and lateral are adequate.
  o CT scan may pick up additional small pneumothoraces.
  o SQ emphysema is commonly due to pneumothorax.
  o A small pneumothorax may be followed with serial chest films.
  o Use a large (36+ Fr) chest tube, because a larger tube will facilitate the drainage of the likely associated hemothorax.
• Tension pneumothorax
  o A large pneumothorax under pressure compresses the mediastinum and contralateral lung and increases intrathoracic pressure.
  o Clinical diagnosis (do not wait for chest radiograph to confirm)
    ▪ Dyspnea
    ▪ Hypotension
    ▪ Tachycardia (late: pulseless electrical activity [PEA])
    ▪ Tracheal deviation away from affected side
    ▪ Jugular venous distension
    ▪ Ipsilateral hyperresonance
    ▪ Ipsilateral absent breath sounds
  o Treatment: immediate decompression via needle or chest tube
1.9 ABDOMINAL TRAUMA

General
- Abdominal cavity extends from pubic symphysis to nipple line (fourth ICS) during exhalation
- Patients with significant mechanism should be evaluated for intra-abdominal injuries, as benign initial exams are found in 20% with injury.

Diaphragm
- Majority of diaphragmatic injuries are caused by penetrating trauma.
- Injuries are more commonly diagnosed on the left (80%), but this may be because the liver blocks the herniation of bowel contents on the right.
- Chest radiograph may reveal an effusion, blurred diaphragm, herniated viscera, or nasogastric tube positioned above the diaphragm.
- Due to diaphragmatic movement, consider abdominal injury from the fourth ICS anteriorly and the sixth ICS posterolaterally.

Hollow Viscus
- May be injured by blunt or penetrating mechanisms
- Involves hemorrhage and peritoneal contamination (irritation, infection)
- Parts of duodenum are retroperitoneal; therefore, symptoms may be delayed.

Penetrating Injuries
- Stab wounds most commonly involve the liver (40%), small bowel (30%), diaphragm (20%), and colon (15%).
- Local exploration of a stab wound in a patient without peritonitis or hypotension may be indicated, as up to 33% do not penetrate the peritoneum.
- Penetration through the anterior fascia increases the risk of intra-abdominal injury.
- Patients with gunshot wounds may also have concussive injuries, bullet fragments, and secondary projectiles (bone fragments); the small bowel (50%), colon (40%), liver (30%), and vascular structures (25%) are most commonly involved.

Retroperitoneum
- Significant hemorrhage (several liters) is possible with pelvic fracture.
- Signs and symptoms may be slow to develop after injury.
- Duodenal injury – commonly retroperitoneal rupture
Solid Organ
- Primary problem is hemorrhage.
- Most common are splenic injuries (40%–55%), liver injuries (35%–45%), and retroperitoneal hematoma (15%).
  - The liver is the most commonly injured organ in penetrating trauma.
  - The spleen is the most commonly injured organ in blunt trauma.
  - Pancreatic injuries usually result from penetrating trauma or a direct blow to the epigastrium (handlebar, steering wheel).
- The patient’s condition (e.g., the presence or absence of shock, comorbid medical conditions), not only the grade of organ injury, predicts the success of nonoperative management.

Vascular
- Active bleeding – as seen on clinical exam or by extravasation on CT or angiogram – must be addressed
- Angiography and embolization if the patient is stable and the injury is associated with blunt abdominal trauma
- Operation if the patient is unstable or if the injury was caused by penetrating abdominal trauma.

Diagnostic Imaging of Abdominal Trauma
- Routine plain abdominal radiographs are not indicated
- CT
  -Insensitive to hollow organ or diaphragm injury
  -Sensitive for retroperitoneum, solid organs, bones
  -CT cystourethrogram for gross hematuria
- Ultrasound
  -Focused Assessment with Sonography for Trauma (FAST): positive exam in unstable patient assists with rapid operative disposition (see Image #11)

1.10 PELVIC FRACTURE (SEE MUSCULOSKELETAL CHAPTER)

1.11 GENITOURINARY TRAUMA

General
- Approximately 80% of GU injuries involve kidney, 10% involve bladder
- Majority of GU injuries are from blunt trauma.
- Approximately 80% of patients with renal injuries have other serious injuries.
- CT scan, the imaging study of choice for upper genitourinary system, should be performed for the following:
  - Gross hematuria
  - Microhematuria plus hypotension or associated injuries
  - Rapid deceleration mechanism
  - Penetrating trauma with proximity to kidney
- Retrograde urethrogram and cystogram may be utilized to evaluate for injury involving the lower genitourinary system

Bladder Trauma
- Bladder injuries are often associated with pelvic fractures (70%–95%).
- Bladder rupture may produce intraperitoneal or retroperitoneal extravasation of urine.
- Imaging may be done by contrast urethrogram/cystogram or CT.
- Always get a post-void image to rule out hidden extravasation.
Urethral Trauma (see Image #70)

- Anterior injury
  - May be a straddle injury, iatrogenic (foreign body), a fractured penis, or the result of a fall or gunshot
  - Presents with hematuria
  - Evaluation by urethrogram
  - Risk of strictures, fistula

- Posterior
  - Pelvic fractures account for the majority of posterior urethral injuries.
  - Presents with blood at meatus, dysuria, scrotal hematoma
  - Risk of incontinence

External Genitalia (Testicular Trauma)

- Commonly a blunt or straddle injury
- Edema, ecchymosis, pain, hematuria
- Risks of hydrocele, abscess, infertility
- Assess with ultrasound, nuclear studies

Renal Trauma

- Associated with rapid deceleration, penetrating, compression trauma, lower rib fractures, and lumbar transverse process fractures
- Injuries may exist in the absence of hematuria.
- Penetrating/ureteral injury: consider IVP or CT
- Renal vascular injury: angiogram

1.12 INJURIES OF THE SPINE (SEE NEUROLOGY CHAPTER)

1.13 LOWER EXTREMITY BONE TRAUMA (SEE MUSCULOSKELETAL CHAPTER)

1.14 CUTANEOUS INJURIES

General

- Universal precautions and aseptic technique are essential.
- Gross decontamination followed by high-pressure saline irrigation
- Do not instill iodine-based agents into the wound.
- Use primary closure except for the following:
  - Puncture wounds
  - Grossly contaminated wounds
  - Delayed or infected wounds
- Suspect foreign bodies and explore thoroughly

Avulsions

- Affected areas less than 1x1 cm require general care.
- Large areas may require flap or graft assistance with closure.
Bite Wounds

- High risk for contamination
- Avoid closure if possible.
- Prophylactic antibiotics are generally used for human bites and bites on the hand.
  - *Staphylococcus* and *Streptococcus* are common.
  - Human bite – *Eikenella corrodens*
  - Cat bite – *Pasteurella multocida*
    - Causative organism in 50% to 80% of infected wounds
    - Infection often develops within 24 hours
  - Dog bite – *Capnocytophaga canimorsus*
- X-ray films are appropriate to evaluate for foreign bodies (especially broken teeth) as well as fractures.
- Consider rabies prophylaxis when a high-risk animal (raccoon, dog, bat) is involved; check current CDC recommendations.

Thermal Burns

- Epidemiology (annually in the United States)
  - 1 million burns
  - 600,000 ED visits
  - 5000 deaths
  - 45,000 burn center admissions
- Degree of burn
  - Superficial
    - Epidermis
    - Pain/erythema
  - Partial thickness
    - Epidermis and dermis
    - Blisters/moist and mottled skin
  - Full thickness
    - Epidermis, dermis, fat, muscle, and nerve
    - Charring, hard skin, little or no pain, pain at periphery
    - Intubate patients with inhalation injuries early
      - Burns of face or mouth
      - Singed facial hair
      - Sooty sputum
      - Hoarse voice or stridor
      - Cough or dyspnea
- Fluid resuscitation
  - Lactated Ringer’s is the standard fluid.
  - Parkland formula
    - 24-hour replacement volume = 4 ml/kg/% total body surface area burned
    - Give half within the first 8 hours after injury, and the other half over the next 16 hours.
  - Brooke formula
    - 24-hour replacement volume = 2 ml/kg/% total body surface area burned
    - Give half within first 8 hours after injury, and the other half over the next 16 hours.
• Estimate total burn area (second and third degree) by chart or estimation rule
  o “Rule of Nines” in adults
    ▪ Head – 9%
    ▪ Arms – 9%
    ▪ Legs – 18%
    ▪ Front – 18%
    ▪ Back – 18%
    ▪ Groin – 1%
    ▪ Palm of hand = 1% body surface area
  o Lund and Browder chart
    ▪ In children <1 year, the head is 18% and each leg is 14%.
    ▪ For each year, take 1% from head and add 0.5% to each leg.

• Circumferential burns to chest, neck, or limbs may require emergency escharotomy to allow ventilation and avoid compartment syndrome.

• Transfer to burn unit
  o >20% total body surface area burned in adults
  o >10% total body surface area burned in young (<10) or old (>55) patients
  o >5% full-thickness burn
  o High-voltage burn
  o Inhalation injury
  o Significant burn to face, eyes, ears, genitalia, hands, feet, or major joint
  o Significant associated trauma, but the burn causes the most concern

• Treatment
  o Cover burns with sterile dressings after application of topical antimicrobials.
  o 1% silver sulfadiazine – OK to apply over most of body (avoid on face/neck/ears); beware of sulfa allergy
  o Bacitracin or similar ointment – preferred on burns of face/neck/ears

Lacerations
• Dirty wounds may be treated by delayed primary closure in 96 hours.
• Large loss of tissue may require healing by secondary intention or grafting.

Puncture Wounds
• High risk for infection, usually skin flora (shoes may harbor Pseudomonas species)
• X-ray film/ultrasound for foreign body
• Do not close wound.

1.15 SOFT-TISSUE EXTREMITY INJURIES

Amputations/Replantation
• Store the amputated portion in moistened saline gauze placed in a bag placed on ice.
• Update tetanus; give antibiotics.
• Indications for replantation
  o All pediatric amputations
  o Single digits with amputation between the proximal and distal interphalangeal joints
  o Thumb
  o Multiple digits
  o Wrist and forearm
Compartment Syndromes

- Increasing internal pressure within a fixed space
- Signs/symptoms
  - Pain on passive range of motion is the first sign; pain out of proportion to physical findings
  - Pallor
  - Paresthesias
  - Poikilothermia
  - Pulselessness (late finding)
  - Paresis/paralysis (late finding)
- Requires immediate surgical consultation; fasciotomy

High-Pressure Injection

- Industrial sprayers and other high-pressure equipment may cause serious injuries with minimal exam findings.
- Usually involves the index finger of the non-dominant hand
- Single most important factor is type of material injected
  - Paint and paint thinner produce inflammatory response.
  - Grease is associated with a lower incidence of amputation.
- Hours after injury: painful, swollen, pale digit
- X-ray film may show air or other material dissecting along tissue planes.
- Treatment: administer analgesics, tetanus prophylaxis, antibiotics; elevate affected area; consult specialist for possible OR debridement
  - Avoid digital nerve blocks – can cause increasing edema and neurovascular damage

Injuries to Joints

- Knee
  - Interarticular injury should be suspected.
  - Evaluate for possible vascular compromise of the popliteal artery.
  - If presumed dislocation, consider angiography.
- Penetrating injury
  - Injury into the joint space has a high rate of infection.
  - Requires operative wash-out

Penetrating Soft-Tissue Injury

- 20% to 40% of gunshot wounds involve an extremity.
- Penetrating trauma causes about 80% of all extremity vascular injury.
- Hard signs of arterial injury demand surgical evaluation:
  - Absent distal pulses
  - Arterial bleeding
  - Expanding hematoma
  - Audible bruit
  - Palpable thrill
  - Distal ischemia
- Soft signs are of no value in the evaluation and do not predict injury.
• Arterial pressure index (API)
  o API >0.9 is normal.
  o API <0.9 is indicative of arterial injury but it cannot detect intimal flaps.
  o Injuries to the deep femoral artery cause no change in API.
• Injuries near the path of a vessel should be observed regardless of the lack of hard signs.

Periarticular Injury
• Maintain a high level of suspicion for interarticular injury.
• Evaluate through complete range of motion.

Sprains and Strains
• Assess muscular, ligamentous, and tendon injuries.
• Splint any area when concerned about associated occult fracture.
• Ice, elevation, rest, range of motion, and anti-inflammatory medications

Tendon Injuries
• Lacerations/transections
• Ruptures
  o Achilles tendon
    ▪ Thompson’s test: With knee flexed, squeeze calf muscles. The foot should plantar flex if the Achilles tendon is intact.
  o Patellar tendon
    ▪ Expect inability to extend the knee and a high-riding patella.

Vascular Injuries
• Assess hard signs of vascular involvement.
• Angiography reveals anatomic detail of injury and repair needs.
• Observation with arterial pressure index (API), ultrasound, and serial examination is recommended in equivocal cases.

1.16 PEDIATRIC TRAUMA

General
• Causes 50% of all pediatric deaths
• Trauma is leading cause of death between the ages of 1 and 19 years.
• Motor vehicle crash is the most common mechanism.
• Head injury is the most common cause of death.
• Approach to pediatric trauma (consider anatomic and physiologic issues)
  o Anatomic characteristics
    ▪ Large occiput, anterior larynx, narrow subglottic area
  o Physiologic characteristics
    ▪ Infants in respiratory distress are obligate nose breathers, have early tachypnea, and use accessory muscles.
    ▪ Infants and young children may have enough blood loss from a scalp wound to cause shock.
    ▪ Normal blood volume is 8% to 9% of body weight (80–90 ml/kg).
      □ Tachycardia is earliest sign of volume loss.
      □ Hypotension is a late sign.
CHAPTER 1 • Traumatic Disorders

- In infants and young children, pelvic organs are intra-abdominal and therefore more exposed to injury.
- Children are prone to hypothermia – ratio of body surface area to mass is high
- Flexible, non-ossified bony structures make SCIWORA (spinal cord injury without radiographic abnormality) more likely (two thirds of spinal injuries)
- Blunt chest trauma may transmit more force to internal organs, making pulmonary contusions more likely with little external evidence.

- Nonaccidental trauma should be considered if the mechanism of injury is inconsistent with the clinical findings or if the patient has retinal hemorrhages, fractures in multiple stages of healing, or specific injury patterns (stocking burns).
- In patients with difficult IV access, an intraosseous line should be placed.
- Resuscitation in hemorrhagic shock
  - Boluses of 20 ml/kg crystalloid, 10 ml/kg of packed red blood cells

1.17 TRAUMA IN THE ELDERLY

Epidemiology
- Most common mechanism of injury in patients >65 years old is a fall.
- Most common mechanism for fatalities among people between the ages 65 and 80 years is motor vehicle crash.
- Most common hip fracture is inter-trochanteric.
- 20% of geriatric C-spine fractures are odontoid fractures.
- The elderly develop more subdural hematomas because of brain atrophy, which stretches the bridging veins.

Mortality
- Elderly are more likely to die from injury.
  - Less physiologic reserve
  - Co-morbid medical conditions can interfere with recovery

Examination
- Medications and underlying conditions may interfere with evaluation of trauma.
  - Example: β-blockers, heart disease may prevent tachycardic response to shock
  - Example: dementia may prevent cooperation with neurologic examination
- The elderly are more likely to develop pneumonias and other complications after trauma.
- The IV contrast load may exacerbate underlying renal pathology
  - Poor baseline renal function
  - Volume depletion secondary to diuretics, trauma

1.18 TRAUMA IN PREGNANCY

General
- Blunt abdominal trauma (motor vehicle crash > falls > assault)
- Significant trauma occurs in up to 8% of all pregnancies

Physiologic Changes in Pregnancy
- First trimester – physiologic anemia: blood volume increases 45%, but red cell mass does not increase as much
- Second trimester – increased HR and decreased BP are normal
- Pulmonary changes include decreased residual volume and increased tidal volume: hyperventilation with respiratory alkalosis
- 12+ weeks – uterus is intra-abdominal and therefore more prone to injury
- 20+ weeks – uterus can compress the inferior vena cava (IVC), decreasing venous return and BP
Examination
- Indications for trauma related x-ray studies, medications, tetanus, and operations are unchanged.
- Assess fetal heart tones during secondary survey: 120 to 160 beats/min is normal
- Check mother’s Rh blood type; give RhoGam if Rh negative.
- In addition to maternal and fetal injuries, trauma may induce the following:
  o Uterine irritability
  o Preterm labor
  o Placental abruption (second most common cause of fetal death after maternal death)
  o Fetal-maternal hemorrhage (up to 30% of severe trauma cases)

Treatment
- Position patient on left side to avoid compression of IVC.
- Best treatment for fetus is optimal resuscitation of the mother.
- Consult OB/GYN for fetal monitoring after significant injuries to mother are addressed.

1.19 MULTISYSTEM TRAUMA

Blast Injury
- Explosives are a unique injury mechanism with multisystem effects.
- Distance from source and position in relation to buildings (which can reflect blast waves) are critical
- Four classes of blast injuries:
  o Primary (direct) blast injury
    ▪ Initial positive-pressure shock wave, followed by negative-pressure wave produce injury in air-filled (easily compressible) structures: tympanic membrane rupture (common), pulmonary contusions, gastrointestinal tract
  o Secondary blast injury
    ▪ Blunt and penetrating injuries from projectiles, flying debris, shrapnel
  o Tertiary blast injury
    ▪ Patient’s body falls or is propelled by blast and strikes object
  o Miscellaneous
    ▪ Dust inhalation, toxic gases, thermal burns, radiation
    ▪ Lung injury
      □ Pulmonary edema and hemorrhage
      □ Adult respiratory distress syndrome (ARDS) can occur up to 48 hours after a blast.
    ▪ Acute gas embolism; occlusion of vessels to the brain or spinal cord is most common
    ▪ Gastrointestinal tract
      □ Air-containing organs may rupture; colon is most commonly affected
      □ More common with underwater blast injuries
1.20 SHOCK (INADEQUATE TISSUE AND ORGAN PERFUSION)

Hemorrhagic Shock
- Induced hypovolemia secondary to blood loss is the most common cause of shock in the injured patient.
- Early manifestations include tachycardia and cutaneous vasoconstriction.
- A narrowed pulse pressure suggests significant blood loss and use of compensatory mechanisms (vasoconstriction and decreased preload).
- Normal adult blood volume is 7% of body weight, or 5 liters in a 70-kg adult (70 ml/kg)
- Amount of hemorrhage can be estimated based on patient's presentation:

<table>
<thead>
<tr>
<th>Class</th>
<th>Blood Loss</th>
<th>Adult Volume</th>
<th>Pulse</th>
<th>SBP</th>
<th>Pulse Pressure</th>
<th>Capillary Refill</th>
<th>Mental Status</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>&lt;15%</td>
<td>&lt;750 ml</td>
<td>&lt;100</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Fluids</td>
</tr>
<tr>
<td>II</td>
<td>15%–30%</td>
<td>750 ml–1.5 L</td>
<td>&gt;100</td>
<td>Normal</td>
<td>Narrowed</td>
<td>Delayed</td>
<td>+/- Anxious</td>
<td>Fluids</td>
</tr>
<tr>
<td>III</td>
<td>30%–40%</td>
<td>1.5 L–2.0 L</td>
<td>&gt;120</td>
<td>&lt;90</td>
<td>Narrowed</td>
<td>Delayed</td>
<td>Anxious</td>
<td>Fluids/Blood</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;40%</td>
<td>&gt;2.0 L</td>
<td>&gt;140</td>
<td>&lt;70</td>
<td>Very Narrow</td>
<td>Delayed</td>
<td>Depressed</td>
<td>Fluids/Blood</td>
</tr>
</tbody>
</table>

- Crystalloids (normal saline or lactated Ringer's) are the current resuscitation fluids of choice (warmed)
  - 3:1 ratio of crystalloid to blood loss
- Transfuse typed and cross-matched blood when able (Class II) or type-specific blood when needed (Class III)
- Transfuse type O when needed (Class IV)
  - Rh-positive blood for males, Rh-negative blood for females of childbearing age

Nonhemorrhagic Shock
- Tension pneumothorax
  - Develops when air enters the pleural space and a ball-valve mechanism prevents escape of air from the pleural cavity.
  - Pathophysiology: total lung collapse, increased intrathoracic cavity pressures, impaired venous return, and fall in cardiac output
  - Hallmarks: hypotension, absent breath sounds on affected side, trachea deviates away from affected side, and respiratory distress
- Spinal shock
  - Loss of neurologic function and autonomic tone below the level of a spinal cord lesion
  - Presents with flaccid paralysis with loss of sensation, deep tendon reflexes, and urinary incontinence accompanied by bradycardia, hypotension, hypothermia, and intestinal ileus
- Neurogenic hypotension
  - Secondary to spinal shock as a result of loss of peripheral vasomotor tone after spinal injury
  - Pathophysiology: sympathetic fibers exit spinal cord at T1 to L2/3; complete cord lesions can disrupt sympathetic tone, producing vasodilatation and unopposed vagal stimuli to heart.
  - Hallmarks: hypotension with normal heart rate or bradycardia, lack of vasoconstriction on exam ("warm shock")
  - Should not be considered the cause of hypotension unless the patient is flaccid and areflexic (reflex tachycardia and peripheral vasoconstriction are absent) and other causes of shock have been excluded
• Cardiac tamponade
  o Most commonly identified in penetrating thoracic trauma
  o Pathophysiology: pericardial effusion (blood) that increases intra-pericardial pressures to the point that filling of the heart is impaired
  o Hallmarks: hypotension, tachycardia, muffled heart sounds, engorged jugular veins, and resistance to fluid therapy

Assessment
• Response to fluids (vital signs, capillary refill, pulses)
  o Urine output
  o 0.5 ml/kg/hr in adults
  o 1 ml/kg/hr in kids
  o 2 ml/kg/hr in infants
  o Resolution of lactic acidosis
CHAPTER 2
Cardiovascular Disorders

James E. Colletti, MD, and Jeffrey A. Tabas, MD

2.1 AORTIC ANEURYSM

Definition
- Pathologic dilatation involving intima, media, and adventitia
- Aortic diameter >3 cm

Epidemiology
- Incidence increases with age
- May occur anywhere, most often infrarenal
- Associated with atherosclerosis
- First-degree relative with an abdominal aortic aneurysm increases risk 10- to 20-fold
- Most important factor for risk of rupture is size of aneurysm

Presentation
- Classic triad of pain, hypotension, and pulsatile mass
- Pain in the abdomen, back, or flank
- Often no symptoms until rupture occurs
- Rupture usually occurs into retroperitoneum (75%), which may allow the bleed to tamponade and temporarily stabilize blood pressure.
- May present with embolization from an intraluminal clot
- An enlarged abdominal aorta may or may not be appreciated on physical examination
- Abdominal bruits occur in 5% to 10% of patients
- Unequal femoral pulses (if there is extension into the iliofemoral vessels or if there is embolic disease)

Examples of Common Misdiagnoses
- Renal colic (most common)
- Musculoskeletal back pain
- Acute MI
- Gastritis
- Acute abdomen – pancreatitis, mesenteric ischemia, diverticulitis, cholecystitis, appendicitis, perforated viscus, bowel obstruction
CHAPTER 2 • Cardiovascular Disorders

Diagnostic Tests
• Abdominal radiograph
  o Neither specific nor sensitive
  o Abnormalities seen in up to two thirds of patients
• Ultrasound (see Image #37)
  o Very sensitive for detecting the presence of an aneurysm
  o Not reliable for demonstrating the presence of rupture
  o Bedside screening tool (may be used to help differentiate aortic aneurysm from renal colic)
• CT
  o Generally considered the gold standard for diagnosis
  o Almost 100% sensitive for aneurysm and rupture
  o Intravenous contrast is not essential for diagnosis of aneurysm, but it increases sensitivity for diagnosis of rupture.

Management
• Early surgical consultation is mandatory.
• Definitive management of a rupturing/ruptured aorta is surgical.
• Begin resuscitation, but do not delay surgery while resuscitating.
• Type and cross
• For asymptomatic aneurysms, there are two approaches:
  o Repair all aneurysms in patients with acceptable surgical risk.
  o Repair aneurysms that are >5 cm in diameter or are rapidly expanding over time.

2.2 AORTIC DISSECTION

Definition
• Tear of the intimal lining of the aorta
• Diagnosis is imperative, since the treatments used for myocardial ischemia or pulmonary embolus (i.e., anticoagulation or thrombolysis) are fatal if mistakenly used in this condition.
• Risk factors include hypertension, Marfan’s syndrome, pregnancy, coarctation of the aorta, bicuspid aortic valve, aortic stenosis, valvular disease, and syphilis (syphilis may cause aneurysmal dilatation or dissection, usually of the ascending aorta).

Presentation
• Consider dissection in any patient with any of the following:
  o Chest or other pain and a neurologic deficit
  o Pain across two body areas (e.g., chest, back, neck, abdomen, extremities)
  o Chest pain and acute renal failure
  o Chest pain and a new murmur

Symptoms
• Pain in 95% of patients
• Abrupt onset of pain in 85% of patients
• Severe or worst pain ever in 90% of patients
• Tearing or ripping pain in 50% of patients
• Pain in the chest in 75% of patients and/or back in 50% of patients
• Syncope in 10% of patients
Past Medical History
• Approximately 70% of patients have a history of hypertension.
• Cocaine use

Physical Findings
• Hypertension in 50% of patients
• Hypotension in 5% of patients
• Aortic insufficiency murmur occurs in 30% of patients with aortic dissection, characterized by a high-pitched blowing diastolic murmur heard immediately after S2; best heard in the right second or third intercostal parasternal area
• Pulse deficit in 15% of patients
• Significant blood pressure differences between the extremities (i.e., >20 mm Hg systolic blood pressure difference between the upper extremities or >30 mm Hg systolic blood pressure difference between the upper and lower extremities)

Diagnostic Tests
• Electrocardiogram (ECG)
  o Normal or nonspecific in 70% of cases
  o Q waves or ST abnormalities are noted in only 3% of patients
• Chest radiograph findings in aortic dissection
  o Wide mediastinum or abnormal aorta in 80% of cases
  o Intimal calcification separation
  o Irregular contour to the aorta
  o Normal in 10% to 15% of cases
  o Left-sided chest radiograph findings include pleural effusion, apical cap, or depressed left mainstem bronchus
  o Right-sided chest radiograph findings include a deviated trachea/NGT or elevation of the right mainstem bronchus
• CT scan with IV contrast
  o Sensitivity 94%/specificity 77%
  o Contraindications include impaired renal function and hemodynamic instability
• Transesophageal echocardiography (TEE)
  o Sensitivity 98%/specificity 83%
  o May be performed in the emergency department
  o Safer in the unstable patient
• MRI
  o Sensitivity 98%/specificity 98%
  o Advantage is that it does not require intravenous contrast.
  o Disadvantages are its cost and limited availability, and it is difficult to monitor a patient in the MRI scanner.
• Angiography
  o Sensitivity 77% to 85%/specificity 90% to 95%
  o Regarded as the gold standard
  o Advantages are that angiography shows anatomy and the extent of the dissection and allows assessment of aortic valves and branches.
  o Disadvantages are that it is difficult to perform and is expensive.
  o False negatives can occur secondary to thrombosis of the false lumen.
Management

- The goals of management are two-fold:
  o To reduce blood pressure
  o To decrease the rate of rise of arterial pressure (dP/dt) in an attempt to decrease the shearing stress on the aorta
- Heart rate and blood pressure goals:
  o Systolic blood pressure between 100 and 120 mm Hg
  o Heart rate between 50 and 60 beats/min
- β-Blockers
  o Should be initiated in any patient with a high suspicion for dissection
  o Esmolol is often preferred because it is easily titrated
  o Labetalol has the advantage of having both α- and β-blocking ability and therefore may be used as a single agent for heart rate and blood pressure control.
  o β-Blockers should be started prior to or in conjunction with vasodilator therapy such as sodium nitroprusside to lower the dP/dt.
  o With labetalol, additional nitroprusside may be necessary for blood pressure management.
- Calcium channel blockers
  o Use when β-blockers are contraindicated
  o Use calcium channel blockers with negative inotropic effect (diltiazem, verapamil)
  o Nifedipine is not recommended to treat an aortic dissection, as it has very little inotropic and chronotropic effects and may result in stimulation of sympathetic activity, thereby increasing the shear stress on the aorta.
- Nitroprusside
  o Can be used in conjunction with a β-blocker to maintain the systolic blood pressure between 100 and 120 mm Hg
- Type and cross

Classification of Aortic Dissection (Stanford Classification)

- Type A
  o Defined as any involvement of the ascending aorta
  o Therapy is emergent surgical repair.
- Type B
  o Defined as involvement only distal to takeoff of the left subclavian artery
  o Therapy is medical management (if possible) to diminish shear force; use negative inotropes and antihypertensives as described above.

2.3 DEEP VENOUS THROMBOSIS (DVT)

Risk Factors

- Postoperative state (pelvic/low abdominal surgery <6 months ago)
- Family history of thrombosis
- Cancer
- Prior thromboembolism
- Paralysis
- Lower extremity cast/immobility (extended travel)
- Congestive heart failure (CHF)
- Estrogen use/pregnancy
• Lower extremity or pelvic trauma
• Age over 40

Differential Diagnosis
• Cellulitis
• Ruptured Baker’s cyst
• Asymmetric swelling from venous stasis or lymphedema

Diagnostic Tests
• Ultrasound
  o 95% sensitive and specific
  o Does not evaluate calves well
  o Repeat exam in 5 to 7 days is recommended for moderate- to high-risk patients.
• D-Dimer
  o Measures the degradation products of circulating cross-linked fibrin
  o Useful to exclude DVT in low-risk patients

Management
• Heparin (unfractionated or low molecular weight) and coumadin until INR is therapeutic
• Inferior vena cava (IVC) filter if anticoagulation is contraindicated
• Isolated calf vein deep venous thrombosis – options are to initiate anticoagulation therapy or follow the patient with repeat ultrasounds

2.4 PULMONARY EMBOLISM

Background
• Mortality in untreated patients is as high as 35%.
• Mortality drops to 2.5% with treatment.
• The majority of pulmonary emboli originate in the lower extremity and pelvis.

Risk Factors
• Same as for deep vein thrombosis
• 20% of patients with pulmonary embolism (PE) have no known risk factors at the time of evaluation

Signs/Symptoms
• Dyspnea is the most common symptom.
• Tachypnea (≥20 breaths/min) is the most common sign.
• Pleuritic pain
• Hemoptysis
• Apprehension
• Hypoxia
• Tachycardia is seen in 30% to 50% of cases of pulmonary embolism.
Diagnostic Tests

- ECG
  - 40% have nonspecific ST/T-wave abnormalities
  - 30% demonstrate sinus tachycardia
  - Classic finding: S1Q3T3 (present in 10%–15% of cases) (Figure 2-1)

  ![Image of ECG](image)

  **Figure 2-1.** The classic electrocardiographic findings of pulmonary embolism. Note the arrows pointing to the S wave in lead I as well as the Q wave and the inverted T wave in lead III. *(Used with permission from Kevin Kilgore, MD.)*

  - T-wave inversion is especially common in the anteroseptal leads.

- Chest radiograph
  - May be normal
  - Atelectasis or parenchymal abnormality is the most common abnormality
  - Elevated hemidiaphragm
  - Pleural effusion (small)
  - Hampton’s hump – a wedge-shaped, pleural-based density
  - Westermark’s sign – distension of the vasculature proximal to the embolism, with loss of the vascular markings distally (rare)

- Arterial blood gas
  - Lacks significant predictive value

- D-Dimer
  - Measures the degradation products of circulating cross-linked fibrin
  - Useful to exclude pulmonary embolism in low-risk patients

- Ventilation perfusion (V/Q) scan
  - High clinical suspicion with a high-probability ventilation perfusion scan: 96% of patients with this scenario have PE
o Low clinical suspicion with a low-probability ventilation perfusion scan: ≤4% of patients with this scenario have PE
o Advantages of a ventilation perfusion scan are low radiation exposure and a high sensitivity (only 2% of patients with PE have a normal or near-normal result) (98% sensitive but only 10% specific)
o Indeterminate ventilation perfusion scans occur most commonly in patients with underlying chronic obstructive pulmonary disease (COPD) or pre-existing heart/lung disease
• Multi-detector CT angiography
  o 95% to 98% specificity
  o 85% to 90% sensitivity
  o Relative contraindications to multi-detector CT angiography are dye allergy and renal dysfunction.
• Conventional pulmonary angiography is now rarely performed.

Management
• Anticoagulation with IV unfractionated or low-molecular-weight heparin
• Indications for vena caval filters:
  o Recurrent thromboembolism despite adequate anticoagulation
  o Active bleeding or high risk for bleeding
  o History of heparin-induced thrombotic thrombocytopenia
• Thrombolysis indications:
  o Hemodynamic instability caused by PE
  o Massive iliofemoral venous thrombosis (phlegmasia cerulea dolens)

2.5 ANTIARRHYTHMIC MEDICATIONS

Class 1 – Na+ channel blockers (membrane stabilizing) work by slowing conduction through the conducting system as well as cardiac muscle
• Class 1A: quinidine, procainamide, disopyramide
• Class 1B: lidocaine, phenytoin
• Class 1C: flecainide, encaïnide, lorcaïnide, propafenone

Class 2 – β-Adrenergic blockers
• Propranolol, esmolol, acebutolol, nadolol, metoprolol

Class 3 – Antifibrillatory
• Bretylium, amiodarone (also has properties of other classes), ibutilide, sotalol

Class 4 – Calcium channel blockers
• Verapamil, diltiazem

Miscellaneous
• Adenosine
  o Drug interactions
    - The dose of adenosine should be doubled in patients on methylxanthines (caffeine and theophylline).
    - The dose of adenosine should be decreased in patients on dipyridamole, because adenosine potentiates dipyridamole’s effects.
    - The dose of adenosine should be decreased in patients on carbamazepine, because carbamazepine potentiates adenosine’s effects.
    - Contraindicated in asthmatics, because the use of adenosine in these patients may precipitate bronchospasm
2.6 ACUTE CORONARY SYNDROMES – MYOCARDIAL INFARCTION AND UNSTABLE ANGINA

Definition – Acute Myocardial Infarction (AMI)
- Rise and fall of serum cardiac markers plus one of the following:
  - Clinical history of ischemic-type chest discomfort
  - Changes on ECG (i.e., pathologic Q waves or changes of ischemia or infarction)
  - Coronary artery abnormality found during angioplasty

Definition – Angina
- Discomfort induced by exercise and relieved by rest or nitroglycerin

Definition – Unstable Angina (USA)
- Angina at rest (usually lasting >20 minutes)
- New-onset exertional angina within the past 2 months
- Increase in severity within the past 2 months

High-Risk Characteristics
- Advanced age, known coronary artery disease, diabetes
- Pain described like that associated with a previous MI or angina that is worse than usual
- Pain that is pressure-like or squeezing
- Radiation to neck, left shoulder, or left arm
- Electrocardiographic changes of ischemia not known to be old

Atypical Presentations
- One third of patients with acute coronary syndrome present without chest pain
- More often elderly, diabetic, or with a history of stroke or heart failure

Classic Cardiac Risk Factors
- Diabetes
- Smoking
- Hypertension
- Hyperlipidemia
- Premature family history
- While cardiac risk factors have been shown to predict the risk of developing ischemic heart disease in patients followed over many years, there is no evidence that they affect the assessment of risk for acute coronary syndrome for patients in the emergency department.

Cocaine
- Cocaine causes α-agonist–mediated vasospasm.
- Cocaine also predisposes the user to the development of premature coronary artery disease.
- Chronic cocaine use is associated with a seven-fold increased risk of early AMI.
  - Treatment is with benzodiazepines in addition to nitrates.
- β-Blockers should be avoided in acute cocaine intoxication since the unopposed α effect theoretically may worsen coronary vasospasm.
- If there is no ischemia on ECG and if symptoms resolve with treatment and observation, some would argue that discharge is safe.
- Patients with ischemia on ECG should be admitted.
Physical Exam
- Most helpful when it is positive for findings of decreased cardiac output:
  o Rales
  o Hypotension
  o S3
- New or worsening mitral regurgitation murmur secondary to acute papillary muscle dysfunction/rupture

Area of Myocardial Infarct Based on Electrocardiogram Leads (Table 2-1)

<table>
<thead>
<tr>
<th>ECG Leads</th>
<th>Area</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1–V2 (LAD)</td>
<td>Septum</td>
<td>Infranodal block Bundle branch block</td>
</tr>
<tr>
<td>V1–V4 (LAD)</td>
<td>Anterior wall of the left ventricle</td>
<td>Left ventricular dysfunction Congestive heart failure Bundle branch block Complete heart block</td>
</tr>
<tr>
<td>V5–V6, I, aVL (LCA)</td>
<td>Lateral left ventricle</td>
<td>Left ventricular dysfunction Atroventricular node block</td>
</tr>
<tr>
<td>II, III, aVF (RCA)</td>
<td>Inferior wall of the left ventricle Posterior wall of the left ventricle</td>
<td>Hypotension Nitroglycerin sensitivity Papillary muscle rupture</td>
</tr>
<tr>
<td>V4R (II, III, aVF) (RCA)</td>
<td>Right ventricle Inferior wall of the left ventricle Posterior wall of the left ventricle</td>
<td>Hypotension Supranodal and atrioventricular blocks Atrial fibrillation/flutter</td>
</tr>
<tr>
<td>V1–V3 (RCA or LCA) (a posterior MI will have ST depression in V1–V3 and ST elevation in leads V8 and V9)</td>
<td>Posterior wall of the left ventricle</td>
<td>Frequently occurs on coexisting inferior or lateral wall MI</td>
</tr>
</tbody>
</table>

Electrocardiographic Characteristics in Patients with AMI
- Diagnostic in 30% to 50%
- Nonspecific in 40% to 70%
- Normal in 1% to 10%

Assessment of the ECG for Ischemia
- First step in the assessment for ischemia is to examine for a wide QRS interval.
- Six causes of a wide QRS interval
  o Hyperkalemia
  o Medications
  o Paced rhythm
  o Ventricular rhythm
  o Bundle branch block
  o Wolf-Parkinson-White (WPW) syndrome
• Six causes of ST-segment elevation
  o Benign early repolarization (J-point elevation)
    ▪ Concave-upward ST segments
    ▪ Located in the anterior leads (V1–V4)
    ▪ Often associated with left ventricular hypertrophy
  o Acute MI
    ▪ Convex-upward or straightened ST segments
    ▪ Reciprocal ST segment depression
    ▪ ST elevation in at least two contiguous leads (Figure 2-2)

![ECG](image)

**Figure 2-2. Acute ST segment elevation MI. Note the ST segment elevation. (Used with permission from James E. Colletti, MD.)**

- T-wave inversions
- Evolution on serial ECGs
  o Pericarditis
    ▪ Typically concave-upward ST segments
    ▪ Often diffuse
    ▪ PR depression may also be seen
  o Left ventricular aneurysm
    ▪ ST-segment elevation, typically in precordial leads
    ▪ Evidence of a previous anterior infarct based on loss of R waves in the same leads
  o Variant or Prinzmetal's angina (vasospasm)
    ▪ ST changes characteristic of AMI that resolve with anti-anginal treatment
  o Bundle branch block
    ▪ QRS duration >0.12 seconds
    ▪ Right bundle branch block (RBBB) – terminal R wave in V1 and a wide sloping terminal S wave in I and V6
    ▪ Left bundle branch block (LBBB) – monophasic R wave in I and V6 and a wide sloping terminal S wave in V1
    ▪ ST segments are discordant with the terminal forces of the QRS complex.
• ST Depression
  o Ischemia – reversible angina, subendocardial infarction, posterior infarction, reciprocal changes
  o “Strain pattern” (left ventricular hypertrophy)
  o Digoxin effect
• T Waves
  o Tall, symmetric, peaked T waves are consistent with hyperkalemia or acute ischemia.
  o T waves that are inverted, deep, and symmetric cause concern about ischemia, pulmonary embolism, or an intracranial event.
• Cardiac Biomarkers (Table 2-2)
  
  **Table 2-2. Cardiac Enzymes: Rise, Peak, and Duration**

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Rise</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>1–2 hr</td>
<td>4–6 hr</td>
<td>24 hr</td>
</tr>
<tr>
<td>CPK-MB</td>
<td>3–4 hr</td>
<td>12–24 hr</td>
<td>2 days</td>
</tr>
<tr>
<td>Troponin</td>
<td>3–6 hr</td>
<td>12–24 hr</td>
<td>7 days</td>
</tr>
</tbody>
</table>

  • Management of Suspected Acute Coronary Syndrome (ACS)
  o Oxygen
  o Nitrates
    ▪ Coronary artery vasodilatation and preload/afterload reduction
    ▪ Response to nitroglycerin has no diagnostic value in the assessment of acute coronary syndrome.
    ▪ Recommended in patients with persistent ischemia, congestive heart failure, hypertension, or large anterior MI, although there is no proven mortality benefit
    ▪ Contraindications to the use of nitroglycerin include the following:
      □ Bradycardia (heart rate <50 beats/min)
      □ Tachycardia (heart rate >100 beats/min)
      □ Hypotension (systolic blood pressure <90 mm Hg or more than a 30-mm-Hg decrease from the patient’s baseline)
      □ Medications for erectile dysfunction – life-threatening hypotension can occur with the coadministration of nitrates (within 24 hours for sildenafil or 48 hours for tadalafil)
        — Hypotension should be supported with fluids and α-agonists.
    ▪ Use nitroglycerin with caution in patients with right ventricular infarct.
      □ Consider the diagnosis of right ventricular infarct in the patient presenting with an AMI in the presence of hypotension, jugular venous distension (JVD), and clear lungs.
      □ Right ventricular MI is present in one third of patients with inferior MI.
      □ Hypotension occurs secondary to decreased left ventricular preload.
      □ Treatment for right ventricular MI includes boluses of fluid.
      □ Cautious use of nitroglycerin and morphine in patients with right ventricular infarct, as preload-reducing medications can cause precipitous drops in blood pressure.
  o Morphine
    ▪ May be used in the setting of intractable pain despite maximal use of anti-anginal medications
    ▪ Class 1 recommendation in patients with ST-elevation myocardial infarction
    ▪ Increasing evidence suggests its use may be associated with worse outcomes.
    ▪ Its use has been downgraded in the 2007 ACC/AHA guidelines for the management of patients with unstable angina and non-ST-elevation myocardial infarction from class I (“it should be administered”) to class IIa (“it is reasonable to administer”).
Aspirin
- Give to everyone with suspected ACS and without a true aspirin allergy.
- Aspirin therapy results in a 23% reduction in 30-day mortality in patients with AMI as well as a 50% reduction in the rate of progression to AMI.
- In patients with a history of gastrointestinal bleeding, a proton-pump inhibitor should be prescribed concomitantly.
- Selective cyclooxygenase 2 inhibitors are associated with an increase in cardiovascular risk and should be discontinued in patients with ACS.
- Nonselective nonsteroidal anti-inflammatory use is also associated with increased adverse outcomes and, although a causal relationship has not been shown, discontinuation is recommended.

Clopidogrel
- Inhibits platelet aggregation
- May be administered to patients who are aspirin allergic or who have a history of major gastrointestinal intolerance to aspirin.
- In patients with a history of gastrointestinal bleeding, a proton-pump inhibitor should be prescribed concomitantly.
- According to the 2007 ACC/AHA guidelines for the management of patients with unstable angina and non-ST-elevation myocardial infarction, administer clopidogrel to patients in whom the following strategies have been selected:
  - Invasive strategy: antiplatelet therapy (with either clopidogrel or an intravenous glycoprotein IIb/IIIa inhibitor) in addition to aspirin should be initiated before diagnostic angiography.
  - Conservative strategy: clopidogrel should be added to aspirin and anticoagulant therapy as soon as possible after admission.
- According to the 2007 ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction, a loading dose of 300 mg of clopidogrel should be added to aspirin in patients younger than 75 years of age with STEMI, regardless of whether they undergo reperfusion.
- In patients taking clopidogrel in whom coronary artery bypass graft surgery is planned, clopidogrel should be held for at least 5 days unless the urgency for revascularization outweighs the risk of excess bleeding.
- Treatment should begin within the first 24 hours – need for immediate treatment in the ED is not well studied or clearly recommended.

β-Blockers
- According to the 2007 ACC/AHA guidelines for the management of patients with unstable angina and non-ST-elevation myocardial infarction as well as for the management of patients with ST-elevation myocardial infarction, a β-blocker should be administered orally in the first 24 hours in the absence of contraindications.
- Elimination of the recommendation for immediate intravenous β-blockade is based on data from the COMMIT trial, which demonstrated a lack of mortality benefit in a large group of primarily STEMI patients. There was a decreased rate of reinfarction and ventricular fibrillation but an increased risk of cardiogenic shock.
- Parenteral administration is recommended in STEMI patients with hypertension or continuing rest pain in the absence of risks for cardiogenic shock or other contraindications such as conduction blocks, bradycardia, or active reactive airway disease.
Risk factors for cardiogenic shock are listed below (the risk increases as the number of risk factors increases):
- Age >70 years
- Systolic blood pressure <120 mm Hg
- Sinus tachycardia >110 beats/min
- Bradycardia (heart rate <60 beats/min)
- Increased time since onset of symptoms
- JCAHO core measures currently measure β-blocker administration in the first 24 hours after hospital admission, not administration in the emergency department.

- **Heparin**
  - Weight-based dosing and use of a nomogram for dose adjustment improve outcomes.
  - Indications include the following:
    - Planned percutaneous coronary intervention (PCI), i.e., angioplasty or coronary stenting
    - Concurrent use of tissue plasminogen activator (tPA)
    - After streptokinase infusion is finished, if patient is at high risk for systemic emboli (large anterior MI, atrial fibrillation, known left ventricular thrombus)
    - High-risk patients with acute coronary syndrome (e.g., positive troponin, intractable ischemic pain, concurrent congestive heart failure, recent PCI or coronary artery bypass grafting with recurrent ischemia)

- **Low-molecular-weight heparin**
  - Eliminates need for laboratory monitoring
  - Lower rates of heparin-induced thrombocytopenia
  - Precautions include extremes of weight (<45 kg or >100 kg) and renal insufficiency

- **Fondaparinux and bivalirudin** are two new anticoagulants recommended as alternatives to unfractionated heparin and low-molecular-weight heparin
  - **Fondaparinux**
    - A synthetic factor Xa inhibitor
    - May have a decreased risk of serious bleeding and is therefore recommended for patients with increased risk of bleeding
    - Does not require laboratory monitoring
    - Cleared renally
    - Serum creatinine must be less than 3.0 to administer
    - Associated with an increased rate of catheter-associated thrombosis
  - **Bivalirudin** is the synthetic analog of hirudin (a direct thrombin inhibitor), which binds reversibly to thrombin and inhibits clot-bound thrombin

- **Glycoprotein IIb/IIIa inhibitors**
  - Block binding of fibrinogen at the glycoprotein IIb/IIIa platelet receptor site that normally causes aggregation
  - Most effective in patients receiving percutaneous coronary intervention
  - A glycoprotein IIb/IIIa inhibitor may be added in the patient with persistent or recurrent ischemic pain despite treatment or with contraindications to aspirin, nitrates, clopidogrel, anticoagulants, and β-blockers.
Reperfusion according to the 2007 ACC/AHA guidelines for management of patients with ST-elevation myocardial infarction

- STEMI patients who present to a hospital with PCI capacity should be treated with that intervention within 90 minutes after first medical contact.
- STEMI patients who present to a hospital without PCI capacity and who cannot be transferred to a PCI center and undergo that procedure within 90 minutes after first medical contact should be treated with fibrinolytic therapy within 30 minutes after hospital presentation unless there is a contraindication to thrombolytic therapy.

Thrombolytics

- Indications
  - ST elevation (>1 mm in two or more contiguous leads) or presumed new LBBB
  - Symptoms <12 hours, which are continuing (does not have to be chest pain)

- Absolute contraindications
  - Active internal bleeding (not including menses)
  - Suspected aortic dissection
  - Uncontrollable hypertension (>180/110 mm Hg)
  - History of hemorrhagic CVA
  - History of nonhemorrhagic CVA within the past year

- Relative contraindications
  - Presenting blood pressure >180/110 mm Hg
  - History of chronic severe hypertension
  - Pregnancy
  - Active peptic ulcer
  - Internal bleeding within the past 4 weeks
  - Trauma/surgery/CPR within the past 2 to 4 weeks
  - Noncompressible vascular punctures
  - Current use of anticoagulants in therapeutic doses (INR >2 or 3) or known bleeding diathesis
  - History of cerebral vascular accident or known intracerebral pathology not mentioned in contraindications
  - For streptokinase: prior exposure or allergic reaction

Percutaneous coronary intervention (PCI)

- Results in better outcomes than thrombolytics if delivered in a timely manner

- Indications for PCI
  - STEMI patients who present to a hospital with PCI capacity should be treated with that intervention within 90 minutes.
  - Patients who have undergone thrombolytic therapy and have the following:
    - Cardiogenic shock and are younger than 75 years of age
    - Severe congestive heart failure
    - Ventricular arrhythmias that are hemodynamically compromising
  - NSTEMI or unstable angina patients with refractory angina, hemodynamic instability, or electrical instability
Patients at increased risk of a clinical event (bad outcome)
  - Elevated troponin
  - New ST-segment depression
  - Signs or symptoms of heart failure
  - New or worsening mitral regurgitation
  - Hemodynamic instability
  - Sustained ventricular tachycardia
  - Percutaneous coronary intervention within the last 6 months
  - Prior coronary bypass graft surgery
  - A high score on a risk assessment tool, such as the Timi score, rated on presentation. The Timi risk score has seven risk indicators—age, coronary artery disease risk factors, known coronary artery disease, aspirin use in the past 7 days, angina in the past 24 hours, rising cardiac markers, ST deviation >0.5 mm. As the Timi score increases, so do composite endpoints such as mortality and recurrent MI or ischemia.
  - If thrombolytics are contraindicated
    - Urgent PCI is not indicated after thrombolytics if symptoms resolve.
  - ACE inhibitors
    - Recommended in all patients with AMI (especially those with congestive heart failure and systolic blood pressure >100 mm Hg)
    - Treatment should begin within the first 24 hours, but not acutely in the emergency department—immediate treatment may lead to increased cardiogenic shock, as with β-blockers.

• Complications
  - Accelerated idioventricular rhythm (AIVR)
    - Reperfusion arrhythmia
    - Ectopic ventricular rhythm with rate of 40 to 120 beats/min
    - Rhythm itself requires no treatment
    - Lidocaine is contraindicated, because suppression of accelerated idioventricular rhythm may lead to asystole.
  - Early complications of AMI
    - First-degree atrioventricular (1°AV block) or second-degree atrioventricular Mobitz type I (2°AV block Mobitz I, otherwise known as Wenckebach) – usually associated with inferior MI
    - Second-degree atrioventricular Mobitz type II or third-degree atrioventricular block (3°AV block) – usually associated with anterior MI; treat with pacemaker
  - Late complications of AMI
    - Dressler’s syndrome (post-MI pericarditis) occurs approximately 8 weeks after MI
      - Presents with a friction rub and pleuritic chest pain
      - Treatment is with nonsteroidal anti-inflammatory agents.
    - Papillary muscle dysfunction
      - Presents as acute myocardial infarction with associated hypotension, a new murmur of acute mitral regurgitation, and congestive heart failure
      - Associated with inferior-posterior MI
      - Therapy – afterload reduction
      - Diagnosis is confirmed via echocardiography or Swan-Ganz catheter
- Ventricular septal rupture
  - Presents as AMI with associated hypotension, a new murmur, and sudden onset pulmonary edema
  - Therapy
    - Dopamine to increase perfusion
    - Nitroprusside/nitroglycerin to decrease afterload
    - Intra-aortic balloon pump
  - Diagnosis is confirmed via echocardiography or Swan-Ganz catheter.
- Congestive heart failure

### 2.7 LEFT HEART FAILURE

**Causes**
- Coronary artery disease
- Hypertension
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Pericardial disease
- Valvular disease
- Myocarditis
- High output states
  - Anemia
  - Hyperthyroidism
  - Beri-beri
  - AV fistula
- Congenital heart disease

**Presentation**
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Peripheral edema

**Physical Exam**
- PMI displacement
- S3
- Mitral regurgitation murmur
- Rales
- Signs of right heart failure:
  - Jugular venous distension (JVD)
  - Edema
  - Hepatomegaly
Comparison of Clinical Presentation, Radiographic Findings, and Corresponding Pulmonary Arterial Wedge Pressure (PAWP) (Table 2-3)

Table 2-3. Comparison of the Progression of Findings in Congestive Heart Failure

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Radiographic</th>
<th>PAWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>Cephalization</td>
<td>12–18 mm Hg</td>
</tr>
<tr>
<td>Dry cough</td>
<td>Kerley B lines</td>
<td>18–25 mm Hg</td>
</tr>
<tr>
<td>Wet cough</td>
<td>Bat wing</td>
<td>&gt;25 mm Hg</td>
</tr>
</tbody>
</table>

Acute Management

- IV, O₂, monitor
- Nitrates, nitrates, and nitrates if blood pressure tolerates – preload and afterload reduction
- Diuretics (furosemide) – decrease intravascular volume
- Consider morphine – comfort and theoretical preload/afterload reduction
- Consider ACE inhibitors – afterload reduction
- Consider dobutamine, milrinone, or nesiritide for increased inotropy & afterload reduction in refractory cases

2.8 COR PULMONALE

Definition

Right heart hypertrophy or dilation caused by increased pulmonary vascular resistance

Causes

- Obstructive Lung Disease
  - Chronic obstructive pulmonary disease (COPD)
  - Asthma
- Restrictive Lung Disease
  - Kyphoscoliosis
  - Neuromuscular disease
  - Morbid obesity (+/- sleep apnea)
- Interstitial Lung Disease
  - Pulmonary fibrosis
  - Sarcoidosis
- Vascular Disease
  - Primary pulmonary hypertension
  - Pulmonary embolus
  - Vasculitis
- Cystic Fibrosis
- Bronchiectasis
- Other Causes of Right Heart Failure
  - Left heart failure (most common cause)
  - Pulmonary valve disease
  - Septal defects

Presentation – same as for signs of right heart failure

- JVD
- Edema
- Hepatomegaly
Diagnostic Tests

- ECG
  - Right atrial enlargement (P wave in lead II more than three boxes high)
  - Right bundle branch block
  - Right ventricular hypertrophy (large R wave in V1)
  - S1, Q3, T3 – S wave in lead I, Q wave in lead III, and inverted T wave in lead III (See Figure 2-1)
  - Chest radiograph – depends on underlying disease

Management

- Oxygen
- Other management depends on underlying disease

2.9 DILATED CARDIOMYOPATHY

Characteristics

- Accounts for up to 80% of cardiomyopathies
- Causes 25% of congestive heart failure
- Leading indication for cardiac transplant

Incidence

- 7.5 cases per 100,000 persons

Causes

- Idiopathic – 47% (idiopathic is the most common cause)
- Post-myocarditis – 12%
- Ischemic – 11%
- Peripartum – 5%
  - Occurs in the last trimester of pregnancy or during the first 6 months of the postpartum period
  - 50% demonstrate complete or near recovery in the first 6 months
- Hypertensive – end stage (“burned out”)
- Toxin-induced
  - Ethanol
  - Chemotherapeutic agents (doxorubicin, adriamycin)
  - Antiretroviral drugs (zidovudine, didanosine)
  - Phenothiazines
  - Cocaine
- Specific infections: viral (coxsackievirus, echovirus, cytomegalovirus, HIV), rickettsial, bacterial (diphtheria, rheumatic fever), mycobacterial, fungal, parasitic (toxoplasmosis, Chagas’ disease [may be the leading cause in South America])
- Collagen vascular disorders (scleroderma, systemic lupus erythematosus [SLE], dermatomyositis)
- Familial
- Miscellaneous
  - Amyloid
  - Nutritional deficiency (thiamine)
  - Electrolyte disturbances (hypophosphatemia, hypocalcemia)
  - Endocrine (hypothyroidism, Cushing’s disease, hyperthyroidism)
  - Muscular dystrophy, Friedreich’s ataxia
Characteristics
- Depressed systolic function and systolic pump failure
- Low cardiac output
- Increased end-systolic and end-diastolic volume and pressure

Presentation
- Often presents with symptoms of left sided heart failure (in 75%–85% of cases)
- May present with arrhythmias or thromboembolic events

Diagnostic Tests
- Chest radiograph – findings of cardiomegaly
- ECG – nonspecific (intraventricular conduction delays, bundle branch blocks, poor R-wave progression)
- Echocardiography
  - Diagnostic for dilated cardiomyopathy
  - Decreased left ventricular function (<45%), increased chamber size
- Cardiac catheterization – normal vessels in idiopathic dilated cardiomyopathy
- Endomyocardial biopsy is often used in evaluation.

Management
- Remove/manage offending factors (e.g., EtOH, uncontrolled hypertension)
- Diuretics (spironolactone)
- Vasodilators (nitrates)
- Angiotensin-converting enzyme inhibitors
- β-Blockers
- Digitalis

Prognosis
- Prognosis is poor: the 5-year mortality is as high as 75%.

2.10 HYPERTRPHIC CARDIOMYOPATHY (HCM)

Characteristics
- Left ventricular and/or right ventricular hypertrophy, which is often asymmetric
- Diastolic dysfunction occurs secondary to decreased compliance caused by hypertrophy
- Hypertrophic cardiomyopathy is the second most common cause of sudden death in adolescents.

Causes
- Idiopathic – 50%
- Familial – 50%

Presentation
- Symptoms often increase with age
- Dyspnea on exertion is the most frequent initial complaint
- Palpitations
- Chest pain
- Syncope
- Family history of sudden death
Physical exam
- S4
- Systolic ejection-type murmur at left sternal border or apex
- Murmur becomes louder with decreased left ventricular filling such as associated with standing, Valsalva maneuver, amyl nitrate, and β-agonists.
- Murmur becomes softer with increased left ventricular filling (e.g., squatting, isometric handgrip, passive leg elevation, and β-blockers)
- ECG with deep narrow q waves in lateral leads

Management
- β-Blockers or calcium channel blockers – slow heart rate and improve diastolic function
- Amiodarone – reduces ventricular arrhythmias associated with hypertrophic cardiomyopathy

2.11 PERICARDITIS

Pathophysiology
- The pericardium consists of a parietal and a visceral layer.
- Normally, 15 to 60 cc of fluid is contained in the space between the layers.
- Conditions that cause inflammation of the pericardium may result in accumulation of fluid in this space, resulting in the most feared complication, cardiac tamponade.

Causes
- Viral or idiopathic
- Postmyocardial (Dressler's syndrome)
- Neoplastic
- Infectious
- Uremic
- Radiation
- Connective tissue disorders – SLE, rheumatoid arthritis, Sjogren's syndrome, and others
- Traumatic

Patient History
- Sharp pain often of prolonged duration (days)
- Pleuritic component
- Worse when supine
- Improved when sitting forward
- Prodrome of fever and malaise is not uncommon

Clinical Findings – Pericardial Friction Rub
- Heard best in left lower sternal border using stethoscope's diaphragm
- Characteristically triphasic, but may have only two components

Electrocardiographic Findings
- Stage 1 – diffuse ST elevation, concave-upwards contour, PR depression (most commonly in leads II, aVF, and V4–V6)
- Stage 2 – ST segments become isoelectric and T waves flatten
- Stage 3 – symmetric T-wave inversion throughout the ECG
- Stage 4 - normalization
Management
- Evaluate and treat the underlying cause (e.g., hemodialysis for uremic pericarditis)
- Otherwise, treatment is with nonsteroidal anti-inflammatory agents.

2.12 PERICARDIAL TAMPONADE

Pathophysiology
- Fluid accumulates in the pericardial space (the space between the parietal and visceral layers) and impairs relaxation of the heart.
- Diastolic filling is impaired, resulting in decreased cardiac output and an increase in jugular venous pressure.
- Decreased cardiac output and left ventricular stroke volume result in a decline in systemic arterial pressure.
- Increased pressure on the right atrium results in increased central venous pressure (hypotension despite increased central venous pressure).

Findings Suggestive of Tamponade
- Tachycardia
- Hypotension
- Elevated jugular venous pressure
- Pulsus paradoxus – >10-mm decrease in systolic blood pressure during inspiration
- Beck’s triad – muffled heart sounds, elevated jugular venous pressure (JVP), and hypotension

Chest Radiograph
- Enlarged cardiac silhouette, especially without other findings of heart failure

Electrocardiogram
- Sinus tachycardia
- Low voltage – average height of QRS complex is <5 mm in limb leads or <10 mm in precordial leads
- Electrical alternans – alternating size and morphology of every other beat, caused by swinging of the heart in the fluid-filled pericardial sac (more common in chronic pericardial effusion that evolves into tamponade; rarely associated with acute pericardial tamponade)

Diagnosis
- Echocardiography is the study of choice – pericardial fluid and right atrial/right ventricular collapse during diastole (see Image #32 for a pericardial effusion)
- Right heart catheterization shows equalization of diastolic pressures in the right atrium, right ventricle, pulmonary artery, and pulmonary capillary wedge pressure

Management
- Volume resuscitation
- Pericardiocentesis when patient is unstable
- Intubate with caution since positive-pressure ventilation leads to preload reduction and may cause a precipitous drop in blood pressure
2.13 ENDOCARDITIS

Prophylaxis

- Conditions for which antimicrobial prophylaxis is recommended:
  - Prosthetic valves
  - Previous endocarditis
  - Cyanotic congenital heart disease that is unrepaired
  - During the first 6 months after repair of congenital heart disease with prosthetic material
  - Surgically constructed systemic pulmonic shunts or conduits

- Conditions that no longer require antimicrobial prophylaxis, according to the 2007 AHA guidelines:
  - Hypertrophic cardiomyopathy with latent or resting obstruction
  - Mitral valve prolapse with murmur or echocardiographic evidence of regurgitation

- Conditions that do not require antimicrobial prophylaxis:
  - Repaired ventricular septal defect
  - Previous coronary artery bypass grafting
  - Atrial septal defect
  - Patent ductus arteriosus beyond 6 months
  - Mitral valve prolapse without murmur or echocardiographic evidence of regurgitation
  - Previous Kawasaki's disease or rheumatic fever in the absence of valve dysfunction
  - Cardiac pacemakers or automatic implantable cardioverter defibrillators (AICDs)

- Procedures for which prophylaxis is recommended:
  - Dental (extractions, periodontal procedures, dental cleanings, orthodontic placement)
  - Respiratory (tonsillectomy, surgery on respiratory mucosa, rigid bronchoscopy, nasal packing)
  - Other (incision and drainage of abscesses)

- Procedures that no longer require prophylaxis, according to the 2007 AHA guidelines:
  - Any gastrointestinal procedure, including diagnostic colonoscopy and esophagogastroduodenoscopy
  - Any genitourinary procedures
  - Exception to the above is the patient with the highest risk cardiac conditions who has an established infection of the gastrointestinal or genitourinary tract and who may have enterococcal bacteremia

- Procedures that do not require prophylaxis:
  - Dental (filling dental cavities)
  - Respiratory (intubation, flexible bronchoscopy)
  - Other (cardiac catheterization)

- Antibiotic regimens
  - Amoxicillin, 2 grams orally 1 hour before procedure; the pediatric dose is 50 mg/kg
  - If the patient is unable to take oral medications, then ampicillin, 2 grams IV 30 minutes before the procedure; the pediatric dose is 50 mg/kg 30 minutes before the procedure
  - If the patient is allergic to penicillin and unable to take oral medications, then:
    - Cefazolin or ceftriaxone, 1 g IV or IM (children, 50 mg/kg)
    - Clindamycin, 600 mg IV or IM (children, 20 mg/kg)
  - If the patient is allergic to penicillin and able to take oral medications, then:
    - Clindamycin, 600 mg (children, 20 mg/kg) 1 hour before the procedure
    - Azithromycin, 500 mg (children, 15 mg/kg) 1 hour before the procedure
    - Clarithromycin, 500 mg (children, 15 mg/kg) 1 hour before the procedure
2.14 SUBACUTE BACTERIAL ENDOCARDITIS

May result from infection of a native valve that is abnormal as a result of the following:

- Rheumatic heart disease
- Congenital anomaly (bicuspid aortic valve or mitral valve prolapse)

Risks Factors

- Recent dental, genitourinary, or gastrointestinal procedures
- Intravenous drug use

Etiologies

- *Streptococcus* (especially *S. viridans*)
- *Staphylococcus*

Presentation

- Fever
- Night sweats
- Nonspecific symptoms such as malaise, headache, anorexia, or myalgia
- Heart murmur
- Petechiae (see Image #36)
- Splenomegaly
- Roth spots – retinal hemorrhages with central clearing
- Splinter hemorrhages – nonblanching, linear, brownish-red lesions in the nail beds perpendicular to the direction of growth of the nail (see Image #44)
- Janeway lesions – nontender plaques on soles and palms
- Osler’s nodes – tender nodules on tips of fingers and toes
- Hematuria
- Elevated erythrocyte sedimentation rate (ESR)

2.15 ACUTE BACTERIAL ENDOCARDITIS

Acute Infection of a Previously Normal Valve

- Most commonly involves the tricuspid valve, affecting the right side of the heart; often seen in intravenous drug users; in three fourths of cases, the causative organism is *Staphylococcus aureus*
- Acute bacterial endocarditis from *Staphylococcus aureus* presents in a more acute fashion than other causative agents, with fevers, heart failure, chest pain, and infiltrates on chest radiograph.
- Less than 35% of intravenous drug users and fewer than 20% of patients without a history of intravenous drug use with infective endocarditis have a murmur on initial exam.
- 50% of patients have left-sided endocarditis (left-sided endocarditis has a higher rate of cardiac failure and neurologic complications than right-sided endocarditis).
- *Streptococcus viridans* is the most common organism in left-sided endocarditis in patients with congenital valvular disease or mitral valve prolapse.
- There is an association between endocarditis from *Streptococcus bovis* and coexisting gastrointestinal malignancy.
- See below under Prosthetic Valve Endocarditis for empiric treatment regimens.
2.16 PROSTHETIC VALVE ENDOCARDITIS

Etiology
- Early prosthetic valve endocarditis has its onset within 60 days after surgery and is usually secondary to a nosocomial etiology such as the following:
  - *Staphylococcus epidermidis* (coagulase-negative)
  - Gram-negative rods
- Late prosthetic valve endocarditis has its onset beyond 60 days of surgery and is usually community acquired
- Late prosthetic valve endocarditis often results in valve failure and is secondary to the following organisms:
  - *Streptococcus* is the most common (accounting for 30% of cases)
  - *Staphylococcus epidermidis* (accounting for 29% of cases)
  - Gram-negative rods (accounting for 10% of cases)
  - *Staphylococcus aureus*

Complications
- Valvular destruction
- Valvular ring abscesses
- Conduction blocks
- Embolization to lungs, coronaries, brain, kidneys

Empiric Treatment Regimens
- Vancomycin plus gentamicin
- Ceftriaxone plus gentamicin

Diagnosis
- Positive blood cultures from two sites
- Echocardiographic evidence of endocarditis (e.g., an oscillating intracardiac mass located on a valve, an abscess, or a new partial dehiscence of a prosthetic valve)

2.17 HYPERTENSIVE EMERGENCY

Definition
- Elevated blood pressure with findings of acute end organ damage
- Evidence of acute end organ damage, not the absolute blood pressure, confirms the diagnosis of a hypertensive emergency. The classic example is preeclampsia or eclampsia, which both represent hypertensive emergencies and can occur without an extreme elevation of blood pressure.

Causes
- Idiopathic
- Medication rebound (especially clonidine)
- Stimulant use – cocaine, amphetamines
- Monoamine oxidase inhibitors (MAOI) reaction
- Pheochromocytoma
Presentation

- Papilledema
- Altered mental status/confusion/coma (reverses with control of pressure)
- Seizures
- Microangiopathic hemolytic anemia with thrombocytopenia
- Glomerulonephritis
- Hematuria and renal failure
- Congestive heart failure
- Cardiac ischemia
- Aortic dissection

Management

- Admission to ICU
- Treatment with intravenous antihypertensives (the management goal is a 30% reduction in mean arterial pressure in 30 minutes)
- Nitroprusside is regarded as the gold standard, but there are certain conditions in which an alternative is preferable.
- Antihypertensive management of hypertensive emergencies is summarized in Table 2-4, and antihypertensive agents are listed in Table 2-5.

Table 2-4. Hypertensive Emergencies and Antihypertensive Therapy

<table>
<thead>
<tr>
<th>Hypertensive Emergency</th>
<th>Antihypertensive Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preeclampsia</td>
<td>Labetalol, nicardipine, or hydralazine</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>Magnesium sulfate and hydralazine</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Phentolamine or nitroprusside and β-blockers (primarily α-blockade to control hypertension and subsequent β-blockade for the control of cardiac dysrhythmias)</td>
</tr>
<tr>
<td>Hypertensive encephalopathy</td>
<td>Nitroprusside</td>
</tr>
<tr>
<td>Intracranial hemorrhage</td>
<td>Labetalol, nitroprusside, or nicardipine</td>
</tr>
<tr>
<td>Acute pulmonary edema</td>
<td>Nitroglycerin, nitroprusside</td>
</tr>
<tr>
<td>Cardiac ischemia</td>
<td>Nitroglycerin, β-blockers</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>Nitroprusside, β-blockers</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>Nitroprusside</td>
</tr>
<tr>
<td>Antihypertensive</td>
<td>Mechanism of Action</td>
</tr>
<tr>
<td>------------------</td>
<td>---------------------</td>
</tr>
</tbody>
</table>
| Nitroprusside    | Potent vasodilator  | Mild reflex tachycardia  
Pulmonary and coronary shunting may occur and may worsen hypoxia and ischemia | Close blood pressure monitoring is required; consider placement of an arterial line and admission to the intensive care unit  
The intravenous bag should be wrapped in opaque material, as nitroprusside is unstable in ultraviolet light.  
Metabolized to thiocyanate and excreted by the kidneys.  
Cyanide is an intermediate metabolite. | Pregnancy |
| Labetalol        | Selective α- and β-blocker (ratio of α- to β-blockage is 1:3 to 1:7) | Orthostatic hypotension | Does not affect cerebral blood flow or renal function | Congestive heart failure  
Heart block  
Asthma  
Pheochromocytoma, as it may result in paradoxical hypertension |
| Esmolol          | Ultra-short-acting,  
selective β1-blocker |  | Useful to control the reflex tachycardia associated with vasodilating agents such as nitroprusside | Cocaine overdose  
Congestive heart failure  
Heart block  
Asthma  
Pheochromocytoma, as it may result in paradoxical hypertension |
| Nitroglycerin    | Vasodilator that acts mainly on the venous system  
Primarily reduces preload  
At high doses, reduces afterload | Hypotension, especially in patients with right ventricular dysfunction | Best utilized in patients with cardiac ischemia or pulmonary edema | Right ventricular dysfunction |
| Nicardipine      | Calcium channel blocker | Headache  
Flushing  
Tachycardia | Has been best studied in pregnant patients | Use with caution in patients with left ventricular failure and cirrhosis |
| Fenoldopam       | Peripheral dopamine-1 receptor agonist | Reflex tachycardia  
Flushing  
Headache | Hypotension occurs less commonly than with nitroprusside  
Offers a reasonable alternative to nitroprusside without concerns of light sensitivity and cyanide toxicity | |
2.18 HYPERTENSIVE URGENCY

Definition
- Elevated blood pressure (diastolic pressure >115 mm Hg) without associated acute end organ damage
- If end organ dysfunction exists, the challenge is to determine if it is acute or chronic.

Treatment
- Avoid acutely lowering the blood pressure in the emergency department.
- The goal is a gradual reduction in blood pressure with oral antihypertensive medications over a 24- to 48-hour period.
- Initiate antihypertensive agents and arrange for rapid follow-up (recheck in 1 or 2 days).

2.19 MITRAL STENOSIS

Overview
- Most common cause is rheumatic fever.
- The majority of patients develop atrial fibrillation secondary to atrial dilatation.

Presentation
- Exertional dyspnea is the most common presentation.
- Hemoptysis is the second most common presentation and may be massive if a bronchial vein ruptures.
- Paroxysmal nocturnal dyspnea
- Orthopnea

Clinical Findings
- Murmur
  - Mid-diastolic rumble with an opening snap
  - Loud S1
  - Heard best at the right of the apex
- Prominent a-wave in the neck
- Systemic blood pressure is typically in the normal or low range
- In the presence of pulmonary hypertension, auscultatory findings are less evident.

Electrocardiogram
- Left atrial enlargement – biphasic p-wave with terminal negative deflection in V1

Chest Radiograph
- Straightening of the left heart border, indicating left atrial enlargement

2.20 MITRAL REGURGITATION

Causes – Chronic
- Rheumatic heart disease is the most common cause.

Causes – Acute
- Infective endocarditis
- Myocardial infarction (may result in acute mitral regurgitation from rupture of the chordae tendineae or papillary muscles)
Clinical Features – Acute Mitral Regurgitation
- Acute mitral regurgitation presents with dyspnea, tachycardia, as well as pulmonary edema.
- Usually an S3 and S4 will be heard.
- The harsh apical systolic murmur starts with S1 and may end before S2.
- Patients may deteriorate quickly and develop cardiogenic shock or cardiac arrest.
- Intermittent mitral regurgitation may present with episodes of respiratory distress secondary to pulmonary edema as well as asymptomatic periods between attacks.

Clinical Features – Chronic Mitral Regurgitation
- Chronic mitral regurgitation can be tolerated for several years.
- The first symptom of chronic mitral regurgitation is usually exertional dyspnea (sometimes prompted by atrial fibrillation).

Exam
- Late systolic left parasternal lift
- High-pitched holosystolic murmur heard best in the fifth intercostal space and the mid left thorax, with radiation to the axilla
- The first heart sound is soft and often obscured by the murmur.

Electrocardiogram
- May demonstrate findings of left atrial and left ventricular hypertrophy

Complications
- Systemic emboli occur in approximately 20% of patients without anticoagulation.
- Endocarditis

2.21 MITRAL VALVE PROLAPSE

Epidemiology
- Mitral valve prolapse is the most common valvular heart disease in industrialized countries (affecting 3% of the population).

Causes – Congenital

Presentation
- Asymptomatic
- Atypical chest pain
- Palpitations
- Fatigue
- Dyspnea unrelated to exertion

Physical Findings
- Midsystolic click
- Second heart sound may be diminished by the late systolic murmur.

Prognosis
- Slightly increased incidence of sudden death and dysrhythmias

Management
- β-Blockers for chest pain
2.22 AORTIC STENOSIS

Causes
- Congenital heart disease is the most common cause of aortic stenosis (a bicuspid aortic valve is the most common congenital cause).
- Rheumatic heart disease is the second most common cause of aortic stenosis.
- Calcific degeneration of the cusps of the aortic valve (more common in patients older than 65 years of age)
- Bicuspid aortic valve is the predominant cause in patients younger than 65 years of age.

Clinical Features
- The classic triad of aortic stenosis is dyspnea, chest pain, and syncope.
- Symptoms appear late in the clinical course (the most common sign is a pulse of small amplitude).
- Pulsus parvus et tardus – pulse that is small and rises and falls slowly
- Narrowed pulse pressure – pulse pressure (the difference between the systolic and diastolic pressures) < 25 mm Hg
- Paradoxic splitting of S2, S3, and S4 is common
- Harsh systolic ejection murmur is best heard in the second right intercostal space and radiates to the right carotid artery
- Sudden death, usually from a dysrhythmia, occurs in 25% of cases

ECG
- Left ventricular hypertrophy is the most common electrocardiographic finding.
- Left or right bundle branch block in 10% of cases

Chest Radiograph
- Initially normal
- Late findings include signs of left ventricular hypertrophy and congestive heart failure.

Management
- Avoid the following:
  - Strenuous activity
  - Nitrates and diuretics (patients with aortic stenosis are dependent on adequate filling pressures)
  - Vasodilators (increase the gradient across the aortic valve and worsen left ventricular function)
  - Patients who are symptomatic should be referred for possible surgical repair or replacement.

Prognosis
- Expected survival is less than 5 years once angina or syncope develops.
- Expected survival is less than 2 years once heart failure develops.

2.23 AORTIC REGURGITATION

Causes
- Acute Aortic Regurgitation – 20% of cases
  - The majority of cases are caused by infective endocarditis.
  - The remainder of cases are caused by aortic dissection.
- Chronic Aortic Regurgitation
  - Rheumatic heart disease
  - Congenital
  - Syphilitic aortitis
  - Ankylosing spondylitis
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• Rheumatoid arthritis
• Reiter's syndrome – an inflammatory arthritis that presents with the classic triad of arthritis, urethritis, and conjunctivitis

• Presentation
  o Acute aortic regurgitation
    ▪ Dyspnea in 50%
    ▪ Acute pulmonary edema
    ▪ Fever in acute endocarditis
    ▪ High-pitched blowing diastolic murmur heard immediately after S2, best heard in the right second or third intercostal parasternal area

  o Chronic aortic regurgitation
    ▪ Palpitations occur in one third of patients, frequently noticed in bed (one of the earliest signs of chronic aortic regurgitation is an uncomfortable awareness of the heartbeat that is especially noticed while lying down)
    ▪ Stabbing chest pain
    ▪ Fatigue
    ▪ Dyspnea
    ▪ Two-thirds of patients have no symptoms for up to 20 years.
    ▪ Wide pulse pressure (pulse pressure is the numeric difference between the systolic and diastolic blood pressures; it is considered wide if the numeric difference is >40 mmHg) with a prominent ventricular impulse
    ▪ Head bobbing
    ▪ Water-hammer pulse – peripheral pulse that has a quick rise in upstroke followed by a peripheral collapse
    ▪ Accentuated precordial apical thrust
    ▪ Pulsus bisferiens (also known as bisferious pulse or biphasic pulse) – on palpation of the pulse, a double peak per cardiac cycle is appreciated
    ▪ Quincke’s pulse – capillary pulsations are visible at the proximal nailbed while pressure is applied at the tip

2.24 SYNCOPE

Definition
• Abrupt decrease in cerebral perfusion, resulting in a transient loss of consciousness/postural tone

Causes
• Vasomotor/vascular
  o Hypovolemia
  o Dehydration
  o Third-spacing
  o Excessive diuresis
  o Hemorrhage – intrabdominal, gastrointestinal, external
  o Postural orthostasis/autonomic insufficiency
    ▪ Vasovagal
  o Carotid sinus sensitivity
Cardiovascular

- Tachy dysrhythmias
  - Ventricular tachycardia
  - Ventricular fibrillation
  - Supraventricular tachycardia (including atrial fibrillation)

- Brady dysrhythmias
  - Asystole
  - Atrioventricular blocks
  - Sinus dysfunction
  - Pacemaker malfunction
  - Myocardial infarction/ischemia
  - Valvular disease (aortic stenosis)
  - Pulmonary embolus or hypertension
  - Congenital heart disease
  - Cardiac myxoma
  - Aortic dissection
  - Cardiomyopathies
  - Pericardial tamponade
  - Vertebrobasilar transient ischemic attack (TIA)
  - Carotid sinus hypersensitivity

Situational

- Post-tussive
- Micturition
- Defecation
- Postprandial
- Swallowing

Metabolic/Endocrine

- Hypoglycemia
- Addisonian crisis
- Hyponatremia
- Hypothyroid
- Pheochromocytoma

Neurologic

- Subarachnoid hemorrhage
- "Seizures" – seizures are to be distinguished from syncope

Medications That Cause Any of the Above
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2.25 TEMPORARY CARDIAC PACING

May be performed by a transcutaneous, transesophageal, or transvenous technique

Emergent Indications for Temporary Pacemaker Placement
• Bradycardia that results in hemodynamic compromise
• Bradycardia with malignant escape rhythms
• Overdrive pacing of refractory tachydysrhythmias
• Symptomatic bradycardia that fails to respond to medical therapy
• Early bradysystolic cardiac arrest (within the first 10–20 minutes)
• Third-degree atrioventricular block unresponsive to medical therapy

Indications for Prophylactic Temporary Pacemaker Placement
• AMI and Mobitz II
• AMI and third-degree atrioventricular block
• AMI and new bifascicular block

Transvenous Pacing
• The apex of the right ventricle is the ideal location of the catheter tip for temporary transvenous pacing.
• An indicator of successful pacemaker placement is ST elevation seen on the ECG during placement
• Most direct approach is the right internal jugular
• Settings
  o Power output set at two times the capture threshold
  o Output at maximum (20 mA) at a rate between 80 and 100 beats/min

2.26 CARDIOPULMONARY ARREST

Asystole
• Assessment
  o Check rhythm in two leads
  o Assess for evidence not to resuscitate (i.e., DNR order, rigor mortis)
• Treatment
  o CPR
  o Epinephrine – 1mg IV push every 3 to 5 minutes
  o Atropine – 1 mg IV every 3 to 5 minutes up to 0.04 mg/kg
  o Intubation

Pulseless Electrical Activity (PEA)
• Definition – rhythm is seen on the monitor but the patient does not have a detectable pulse
• Causes – 6 H’s and 5 T’s
  o Hypovolemia (overall the most common cause)
  o Hypoxia
  o Hydrogen ion – acidosis
  o Hyper/hypokalemia
  o Hypothermia
  o Hypoglycemia
  o Tablets (overdoses, especially digoxin, tricyclic antidepressant, calcium channel blocker)
o Tamponade
o Tension pneumothorax
o Thromboembolism (acute MI, pulmonary embolism)
o Trauma

• Management
  o Chest compressions – 5 cycles or 2 minutes after every drug
  o Search for treatable causes
  o Intubation
  o Epinephrine – 1 mg IV push every 3 to 5 minutes
  o Atropine – 1 mg IV every 3 to 5 minutes up to 0.04 mg/kg (if slow PEA)

Ventricular Fibrillation or Pulseless Ventricular Tachycardia

• Management
  o Chest compressions – 5 cycles or 2 minutes after every drug and defibrillation
  o If unwitnessed arrest or down time greater than 4 or 5 minutes, then give chest compressions for 5 cycles or 2 minutes.
  o Defibrillate once at maximum joules (360 J if monophasic defibrillator; 120–200 J if biphasic defibrillator, depending on the model)
  o After defibrillation, CPR should be resumed immediately
  o Escalating dosages of joules and stacked shocks are no longer recommended.
  o Intubate and confirm placement
  o Establish IV access
  o Epinephrine, 1 mg IVP every 3 to 5 minutes (or vasopressin, 40 U IV, one time)
  o Defibrillate
  o Medications – lidocaine, amiodarone, procainamide; magnesium for torsades de pointes
  o Defibrillate

2.27 NARROW COMPLEX TACHYCARDIAS
Defined by a heart rate >100 beats/min and a QRS width <0.12 sec

2.28 NARROW COMPLEX REGULAR TACHYCARDIA WITH THE PRESENCE OF P WAVES

Sinus Tachycardia (Figure 2-3)

Figure 2-3. Sinus tachycardia. Note the sinus rhythm >100 beats/min. (Used with permission from James E. Colletti, MD.)

• Definition – P waves are generated by the sinus node, which is confirmed by identification of the correct P-wave axis.
  o Will see upright P waves in lead II and inverted P waves in aVR on the ECG
  o The sinus beat conducts through the atrioventricular node and generates a ventricular complex (this is confirmed by a P wave preceding each QRS).
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- Causes – conditions that induce sympathetic excess
- Management – treat underlying condition

Atrial Flutter

- Diagnosis (Figure 2-4)

Figure 2-4. Atrial flutter. Note the regular narrow complex rhythm with ventricular rate of approximately 150 beats/min and the atrial rate of approximately 300 beats/min. The atrial beats ("flutter waves") classically are inverted and create a sawtooth pattern in the inferior leads.
(Used with permission from Amal Mattu, MD.)

- Regular narrow complex rhythm with an atrial rate of 250 to 350 beats/min
- Flutter waves with a sawtooth appearance, best seen in inferior leads and in leads V1–V2
- Often with 2:1 block, yielding heart rate of 150 beats/min

- Management
  - Unstable patient – cardioversion beginning at 100 J
  - Stable patient
    - Rate control – treat with medications that slow atrioventricular conduction such as β-blockers or calcium channel blockers
    - Digitalis can be used as a second-line agent in patients with mild tachycardia and preexisting congestive heart failure
    - Atrioventricular nodal blockers (β-blockers, calcium channel blockers, and digitalis) should be avoided if there is a suspected accessory pathway, because these agents primarily block the atrioventricular nodal conduction and therefore may enhance anterograde conduction along the accessory path, thereby permitting unbridled rapid ventricular response rates and precipitating ventricular fibrillation.
    - Rapid ventricular response rates (especially >200 beats/min) are a hint that an accessory pathway may be present, as normal atrioventricular nodal tissues rarely allow a ventricular response rate >150 to 165 beats/min
    - Class 1A agents (procainamide or quinidine) or cardioversion may be used to convert atrial flutter to sinus rhythm.
2.29 NARROW COMPLEX REGULAR TACHYCARDIA WITH THE ABSENCE OF P WAVES

Supraventricular Tachycardia (SVT) (Figure 2-5)

- Diagnosis – regular narrow complex tachycardia with a rate between 150 and 200 beats/min without the presence of P waves
- Management
  o Initial therapy
    - Vagal maneuvers (Valsalva or carotid sinus massage) – will terminate 20% to 25% of episodes of reentry SVT
    - Adenosine – must be given rapidly over 1 to 3 seconds through an IV line in a large (antecubital) vein, followed immediately by 20 mL saline flush and elevation of the arm; the initial dose of adenosine is 6 mg IV, followed by a second bolus of 12 mg IV if the rate does not convert in 1 to 2 minutes
  o Second-line agents – atrioventricular nodal blocking agents
    - Calcium channel blockers (diltiazem or verapamil)
    - β-Blockers
  o Third-line agents
    - Amiodarone may be utilized for narrow-complex tachycardias that have originated from a reentry mechanism (reentry SVT) in the event the rhythm remains uncontrolled by first-line and second-line agents (vagal maneuvers, adenosine, and atrioventricular nodal blockade).
    - Procainamide may be used for reentry SVT if the rhythm is uncontrolled by adenosine and vagal maneuvers.

Figure 2-5. Supraventricular tachycardia. Note the regular narrow complex tachycardia without P waves. (Used with permission from James E. Colletti, MD.)
2.30 NARROW COMPLEX IRREGULAR TACHYCARDIA WITH THE PRESENCE OF P WAVES

Multifocal Atrial Tachycardia (MAT) (Figure 2-6)

Figure 2-6. Multifocal atrial tachycardia. Note the arrows pointing to three distinct P-wave morphologies. (Used with permission from Kevin Kilgore, MD.)

- Diagnosis – at least three distinct P-wave morphologies with ventricular response >100 beats/min
- Causes – often associated with hypoxemia, chronic obstructive pulmonary disease (COPD)
- Management – treat underlying condition

2.31 NARROW COMPLEX IRREGULAR TACHYCARDIA WITH THE ABSENCE OF P WAVES

Atrial Fibrillation (Figure 2-7)

Figure 2-7. Atrial fibrillation. Note the narrow complex irregular tachycardia, with the absence of P waves. (Used with permission from Electrocardiography Lab, Mayo Clinic, Rochester, Minnesota.)

- Presentation
  - Palpitations, syncope, pre-syncope, shortness of breath, and weakness; or the patient may be asymptomatic
  - Symptoms arise from rapid ventricular rates.
  - Lack of organized contraction (“atrial kick”) contributes to symptoms in patients with significantly impaired ejection fraction
- Diagnosis
  - Narrow complex irregularly irregular tachycardia without distinct P waves
  - Acute causes
    - Alcohol intake
    - Myocarditis/pericarditis
    - Pulmonary embolism
    - Hyperthyroidism
    - Acute myocardial infarction
- Management – Unstable Patient
  - DC cardioversion beginning at 50 to 100 J is the treatment of choice.
- Management – Stable Patient
  - Rate control for normal cardiac function (ejection fraction >40% or congestive heart failure)
    - Calcium channel blockers
    - β-Blockers
  - Rate control for impaired heart (ejection fraction <40% or congestive heart failure)
    - Amiodarone
    - Diltiazem
    - Digoxin is not shown to benefit in the acute situation
• Rate control in the presence of Wolff-Parkinson-White (WPW) syndrome
  - Procainamide
  - Amiodarone (safety in WPW syndrome is questionable; recommended by ACLS, but literature suggests otherwise, as amiodarone also has atrioventricular nodal blocking properties)
  - Flecainide
  - Propafenone
  - Sotalol
  - Avoid atrioventricular nodal blocking agents, because they may increase conduction along accessory pathway, resulting in ventricular fibrillation.

• Conversion to sinus rhythm – should be attempted only if the patient is acutely unstable, if the duration of symptoms is <48 hours, or if the patient has been anticoagulated for 3 weeks before and will be anticoagulated for 4 weeks after cardioversion
  - Procainamide
  - Amiodarone
  - Ibutilide
  - Propafenone
  - Flecainide
  - DC cardioversion beginning at 50 to 100 J

2.32 WIDE COMPLEX TACHYARRHYTHMIAS

Definition
• Ventricular rate >100 beats/min and QRS duration >0.12 sec
• Differential diagnosis for a wide complex tachyarrhythmia includes the following:
  o Ventricular tachycardia
  o SVT with aberrancy or accessory pathway

Wolff-Parkinson-White Syndrome
• WPW syndrome (the most common form of pre-excitation) is characterized by the presence of an accessory pathway associated with various tachyarrhythmias.
• Patients with WPW syndrome have three main presentations of tachydysrhythmia: irregularly irregular, regular and wide, and regular and narrow.
  o Irregularly irregular
    - Caused by atrial fibrillation
    - QRS complexes vary in width and morphology.
    - QRS complexes appear narrow for beats conducted through atrioventricular node, wide for beats conducted through bypass tract.
    - DO NOT treat with atrioventricular nodal blocking agents.
  o Regular and narrow
    - Orthodromic reentry tachycardia
    - Conduction to the ventricles is through atrioventricular node and back up bypass tract to the atria.
    - Atrioventricular nodal blocking agents may be used.
  o Regular and wide
    - Antidromic reentry tachycardia
    - Conduction to the ventricles is down bypass tract and back up through the atrioventricular node to the atria.
• Resembles ventricular tachycardia
• Be careful not to miss ventricular tachycardia!
• DO NOT treat with atrioventricular nodal blocking agents.

• Diagnosis – Classic WPW syndrome presents with three electrocardiographic features (Figure 2-8):

![Figure 2-8. Wolff-Parkinson-White syndrome. Note the short PR interval (<0.12), the prolonged QRS (>0.10), and the slurred upstroke to QRS – the Delta wave. (Used with permission from James E. Colletti, MD.)](image)

  o Short PR interval (<0.12 sec)
  o Prolonged QRS (>0.10 sec)
  o Slurred upstroke to QRS (the delta wave)

• Management
  o WPW syndrome with atrial fibrillation or wide complexes
    • Stable: procainamide or amiodarone (questionable safety of amiodarone in WPW syndrome with atrial fibrillation, because, in addition to its antifibrillatory properties, amiodarone also has atrioventricular nodal blocking properties)
    • Unstable: cardioversion
    • Avoid medications that block the atrioventricular node but not the bypass tract (e.g., digoxin, calcium channel blockers, β-blockers, adenosine).
  o WPW syndrome with narrow complex, regular tachycardia
    • Adenosine is drug of choice – short half-life (<10 sec)
    • Procainamide or amiodarone is acceptable.

Ventricular Tachycardia
• Presentation – 80% to 85% of wide complex regular tachyarrhythmias seen in the emergency department are caused by ventricular tachycardia.
• Monomorphic Ventricular Tachycardia (Figure 2-9)

![Figure 2-9. Ventricular tachycardia. Note the wide complex regular tachyarrhythmias. (Used with permission from Electrocardiography Lab, Mayo Clinic, Rochester, Minnesota.)](image)
Monomorphic ventricular tachycardia appears as a regular pattern of morphologically consistent QRS complexes at a rate >120 beats/min, usually 150 to 200 beats/min.

- QRS width >0.12 sec
- Classic teaching is that a QRS width >0.14 sec suggests ventricular tachycardia as opposed to supraventricular tachycardia with aberrancy (this is not actually the case, as approximately 20% of patients with ventricular tachycardia have a QRS width <0.14 seconds; however, classic teaching holds true for the purposes of board review)
- Ventricular tachycardia cannot be differentiated from supraventricular tachycardia with aberrancy by clinical symptoms, blood pressure, or heart rate.

- Signs that ventricular tachycardia is present (as opposed to SVT with aberrancy) include the following:
  - Ventricular tachycardia is a form of atrioventricular dissociation.
    - The sinus node is depolarizing the atria in a normal manner at a rate that is either equal to or less than the ventricular rate; thus, sinus P waves may be seen between QRS complexes.
  - Fusion beat
    - QRS complex is of intermediate width and differing morphology compared with the other beats.
    - Occurs when a sinus beat is conducted to the ventricles through the atrioventricular node and joins (or fuses) with a ventricular beat originating from the abnormal ectopic focus
  - Capture beat
    - Narrow-appearing QRS complex
    - Occurs when an independent atrial impulse causes ventricular depolarization through the normal conducting system
  - Right-axis deviation or extreme right-axis deviation
  - Left-axis deviation

- Management: unstable
  - DC synchronized cardioversion

- Management: stable, preserved function
  - Amiodarone is recommended by the American Heart Association.
  - Procainamide and sotalol are acceptable alternatives.
  - Lidocaine can be used as well.

- Management: stable, poor ejection fraction (ejection fraction <40% or clinical signs of congestion heart failure)
  - Amiodarone, lidocaine, or DC cardioversion is acceptable.

- Polymorphic ventricular tachycardia
  - Characteristics – more than one QRS type
  - Torsades de pointes (Figure 2-10)
    - “Torsades de pointes” and “polymorphic ventricular tachycardia” are not synonymous terms.
    - All cases of torsades de pointes are a form of polymorphic ventricular tachycardia, but not all cases of polymorphic ventricular tachycardias are torsades de pointes.
    - Torsades de pointes is associated with a long QT interval, whereas polymorphic ventricular tachycardia is not.
    - Characterized by twisting, rotating complexes
    - Ventricular rate >200 beats/min
Associated with prolonged QT interval (a QT interval of 0.5 seconds or longer is indicative of an increased risk of torsades de pointes)

- Torsades de pointes is associated with the following:
  - Electrolyte abnormalities (hyperphosphatemia, hypomagnesemia, hypocalcemia)
  - Class 1A antiarrhythmics (procainamide, quinidine, and disopyramide)
  - Psychotropic agents (tricyclic antidepressants and phenothiazines)

Figure 2-10. Torsades de pointes. Note the twisting, rotating complexes with a ventricular rate >200 beats/min. (*Used with permission from James E. Colletti, MD.*)

- Management
  - Cardioversion
  - Magnesium sulfate (first-line therapy)
  - Isoproterenol
  - Overdrive pacing
  - The management of torsades de pointes differs from that of polymorphic ventricular tachycardia after conversion to sinus rhythm in that:
    - Patients with polymorphic ventricular tachycardia can be placed on an antidysrhythmic medication such as amiodarone, lidocaine, or procainamide.
    - Patients with torsades de pointes should be started on magnesium sulfate and cannot be placed on the above-listed antidysrhythmics because these drugs may prolong the QT interval.

2.33 BRADYARRHYTHMIAS

Bradyarrhythmias are secondary to a either a suppression of pacemaker or a conduction system block.

Sinus Bradycardia
- Sinus rhythm <60 beats/min (Figure 2-11)

Figure 2-11. Sinus bradycardia. Note the sinus rhythm <60 beats/min. (*Used with permission from James E. Colletti, MD.*)
Pathologic causes
- Hypothermia
- Medications (β-blockers, calcium channel blockers, parasympathomimetics, digitalis)
- Endocrine/metabolic (hypoadrenal state, hypothyroidism, hyperkalemia)
- Increased vagal tone (inferior wall MI, carotid sinus hypersensitivity)

First-Degree Atrioventricular Nodal Block (Figure 2-12)

![Figure 2-12. First-degree atrioventricular nodal block. Note the prolonged PR interval (>0.20 sec) without loss of impulse transmission. (Used with permission from James E. Colletti, MD.)]

- Characteristics
  - Prolonged PR interval (>0.20 sec) without loss of impulse transmission
  - Occurs normally in 1.6% of healthy adults
  - More common in elderly
  - No treatment needed

Second-Degree Atrioventricular Nodal Block
- Mobitz Type 1 (Wenckebach) (Figure 2-13)

![Figure 2-13. Mobitz Type 1 second-degree atrioventricular nodal block. Note the progressive PR lengthening until QRS is dropped. (Used with permission from Electrocardiography Lab, Mayo Clinic, Rochester, Minnesota.)]

- Mechanism – conduction dysfunction at atrioventricular node
- Diagnosis
  - Progressive PR lengthening until QRS is dropped (dropped beat)
  - Group beating (repeated groups of beats with pauses) of the QRS complexes may be observed.
- Causes
  - Inferior MI is the most common cause.
  - Medications – digitalis, β-blockers, or calcium channel blockers
Management
- Often unnecessary unless the patient is unstable
- Usually transient
- Usually responds to atropine
- Worsened by carotid massage and increased vagal tone

Prognosis – in the setting of inferior MI, this AV block is rarely the cause of adverse sequelae

Mobitz Type II Second-Degree Atroventricular Nodal Block (Figure 2-14)

Mechanism
- Block is below the level of the atroventricular node.
- Often caused by a diseased or ischemic His-Purkinje system

Diagnosis – dropped beats (intermittent conduction of sinus beats)

Causes
- Anteroseptal MI
- Cardiomyopathy

Management
- Atropine will have no benefit (theoretically could cause harm)
- Transcutaneous pacer availability
- Definitive pacemaker placement

Prognosis – often progresses to complete block in the setting of acute MI

Third-Degree (Complete) Atroventricular Nodal Block (Figure 2-15)

Mechanism
- Block is below the level of the atroventricular node.
- Often caused by a diseased or ischemic His-Purkinje system

Diagnosis – dropped beats (intermittent conduction of sinus beats)

Causes
- Anteroseptal MI
- Cardiomyopathy

Management
- Atropine will have no benefit (theoretically could cause harm)
- Transcutaneous pacer availability
- Definitive pacemaker placement

Prognosis – often progresses to complete block in the setting of acute MI
Mechanism
- Injury or damage to the cardiac conduction system so that no impulses are able to pass between the atrial and ventricles (complete block of the atrioventricular node)
- Absent conduction of all atrial impulses resulting in complete electrical and mechanical atrioventricular dissociation
- The complete block can occur at a few different areas:
  - Atrioventricular node (high or junctional nodal blocks)
  - Bundle of His
  - Bundle branches (low-nodal or infranodal block)
- Not all atrioventricular dissociation is complete heart block; for complete heart block to be present, the underlying supraventricular or junctional rhythm must be of a sufficient rate to overcome the action of any infranodal pacemakers.
  - Atrioventricular dissociation occurs when ventricular escape depolarization is faster than the atrial rate (rate is between 40 and 55 beats/min).
  - Third-degree atrioventricular block is present if the ventricular rate is slower than the atrial rate (rate is between 20 and 40 beats/min).

Diagnosis
- Atrioventricular dissociation – P-wave and QRS complexes are present but unrelated and are occurring at different rates.
- The duration of the QRS complex depends on the site of the escape rhythm pacemaker.
- Pacemakers above the bundle of His produce a narrow-complex rhythm (faster rate of 45–60 beats/min) and are responsive to atropine and isoproterenol
- Pacemakers at or below the bundle of His produce a wide-complex rhythm (slower rate of 30–45 beats/min) and are unaffected by atropine and isoproterenol.

Causes
- Ischemia
- Medications (digitalis, β-blockers, calcium channel blocker)

Management
- Transcutaneous pacer availability
- Definitive pacemaker placement
- Atropine
  - If there is clinical evidence of hypoperfusion, atropine can be administered, but third-degree heart block is best managed by transcutaneous pacing.
  - The concern regarding atropine use in third-degree heart block is that it can worsen conduction rates (but in practice, clinical deterioration is rare).
  - Pacemakers above the bundle of His respond to atropine and isoproterenol.
  - Pacemakers at or below the bundle of His are unaffected by atropine and isoproterenol.

2.34 CHANGES IN ACLS GUIDELINES (2005)
For Witnessed Ventricular Fibrillation/Pulseless Ventricular Tachycardia Cardiac Arrest
- Administer a single shock from a defibrillator at maximum joules
  - Monophasic defibrillator, 360 J
  - Biphasic defibrillator, 120 to 200 J
• Followed by 2 minutes of immediate chest compressions
• After each shock or medication, perform chest compression for at least 2 minutes before checking pulse or rhythm.

For Ventricular Fibrillation or Pulseless Ventricular Tachycardia Cardiac Arrest with an Unknown Downtime
• Perform 2 minutes of chest compressions before the first defibrillation.
• After giving two rescue breaths, lay rescuers should not check for signs of circulation before beginning chest compressions.
• Compressions – “Push hard, push fast” and with full chest recoil
  o Goal compression rate for adults is 100 compressions per minute.
  o The compressions-to-breaths ratio is 30:2 from infancy through adulthood
  o When two providers are present, a ratio of 15:2 can be used for children.

Basic Life Support (BLS) Skills
• The priority in managing cardiac arrest
• Care providers must minimize interruptions in chest compressions.

Therapeutic Hypothermia
• When the initial rhythm was ventricular fibrillation, unconscious adult patients with spontaneous return of circulation after out-of-hospital cardiac arrest should be cooled for 12 to 24 hours to 32° to 34°C (90–93°F).

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3.1 ABDOMINAL WALL

Hernia—a protrusion of any viscus from its proper cavity

- Incarcerated: hernia is irreducible
- Strangulated: vascular supply is compromised, producing ischemia or infarction of the involved viscus
  - Usually follows incarceration of the hernia as edema develops
  - Nausea, vomiting, and pain out of proportion are typical during ischemia; peritoneal findings develop with infarction
- Inguinal
  - Direct: through the floor of Hesselbach’s triangle; rarely incarcerates
  - Indirect: through the internal inguinal ring lateral to the inferior epigastric vessels
  - Most common type of hernia in males and females
  - Often incarcerates
- Femoral
  - Into femoral canal, below inguinal ligament and femoral vessels
  - Often incarcerates
  - More common in women
- Obturator
  - Through obturator foramen into medial thigh
  - Usually occurs in elderly women
- Ventral
  - Usually through anterior abdominal wall surgical site
  - Rarely incarcerates
- Umbilical
  - Usually in neonates, but closes by 3 years of age
  - Rarely incarcerates
3.2 ESOPHAGUS

Infectious Disorders

- **Candida Esophagitis**
  
  o Most common in immunosuppressed patients
  
  o Patients present with significant odynophagia; may also have dehydration
  
  o Initial therapy is a 10-day course of fluconazole; if symptoms persist, refer for endoscopy to rule out herpes and cytomegalovirus (CMV).

Inflammatory Disorders

- **Esophagitis and Gastroesophageal Reflux**
  
  o Reflux of stomach juices into esophagus
  
  o Retrosternal burning; radiates from epigastric area to throat, especially with recumbency; dyspepsia
  
  o Extra-esophageal manifestations (cough, hoarseness, wheezing) may occur with or without heartburn symptoms.
  
  o Usually relieved with antacids
  
  o Complications: Barrett's esophagitis (pre-malignant condition), stricture, bleeding, perforation
  
  o Initial recommended treatment: lifestyle modifications plus a 4- to 8-week course of an H2-blocker, followed by referral to GI specialist

- **Caustic Ingestions**
  
  o Acid: produces coagulation necrosis
  
  o Alkali: produces liquefaction necrosis; worse prognosis than acids
  
  o Complications: airway compromise (edema), perforation, stricture
  
  o Treatment: emergency diagnostic endoscopy if there is any suspicion of ingestion; no lavage, neutralization, induced emesis, or charcoal

Motor Abnormalities

- **Spasm**
  
  o Frequently mimics angina
  
  o May improve with nitrates or calcium channel blockers
  
  o Usually precipitated by foods, especially cold foods

- **Achalasia**
  
  o Lower two thirds of esophagus has no peristalsis.
  
  o Usual presentation is dysphagia, chest pain, esophageal spasms.
  
  o Treatment is aimed at reducing lower esophageal pressure with medications or surgery.

Structural Disorders

- **Esophageal Rupture**
  
  o Full-thickness perforation/rupture, usually left mid-thoracic esophagus
  
  o Classically caused by vigorous retching/vomiting (Boerhaave's syndrome), although this is NOT the most common cause of esophageal rupture: more commonly caused by iatrogenic rupture (e.g., endoscopy, dilation)
  
  o Generally associated with severe retching/vomiting OR recent upper GI procedure (e.g., endoscopy), followed by sudden chest and upper abdominal pain radiating to neck (Mackler's triad: chest pain, vomiting, subcutaneous emphysema)
  
  o Rupture produces chemical mediastinitis, followed by infectious mediastinitis and sepsis.
Chest film typically shows large left pleural effusion, pneumothorax/pneumomediastinum, and/or subcutaneous emphysema in neck.

Diagnosis is primarily clinical with chest film; can use Gastrograffin for esophageal swallow study; CT may also demonstrate subcutaneous and mediastinal air

Treatment involves broad-spectrum antibiotics and emergent surgery.

**Mallory-Weiss Syndrome**
- Partial-thickness tear usually at gastroesophageal junction, resulting in arteriolar bleeding
- Presentation often involves hematemesis (mild to profuse).
- Definitive diagnosis with endoscopy
- Supportive treatment in the ED
  - Consider IV vasopressin infusion until surgery if bleeding is refractory.

### Foreign Body
- **General**
  - Most common areas of obstruction
    - At the level of the cricopharyngeal muscle (C6) – children
    - At the level of the aortic arch (T4)
    - At the GE junction (T11) – adults
  - 80% to 90% of foreign bodies will pass once they reach the stomach.
- **Meat impaction**
  - More common in elderly due to underlying anatomic abnormalities; usually distal esophagus
  - Usually pass spontaneously with time and relaxation
  - Glucagon, 1 to 2 mg IV, may work by relaxing the lower esophageal sphincter (LES)
  - Nifedipine reduces esophageal tone, but its use is limited by side effects, including hypotension.
  - Meat tenderizer (papain) should be avoided because it increases the risk of perforation.
  - Refractory cases: endoscopy; barium swallow after passage allows evaluation for underlying pathology
- **Coins**
  - Coins in the esophagus orient in the frontal plane (“en face”) on PA chest film, whereas coins in the trachea orient sideways on PA chest film due the tracheal rings.
  - Coins in the upper esophagus should be removed endoscopically because of the risk of aspiration.
  - Coins in the lower esophagus can be observed for 12 to 24 hours and removed endoscopically if they remain present.
- **Sharp objects**
  - Should be removed even if they reach the stomach, as 15% to 35% will cause perforation
- **Large objects**
  - Objects larger than 2x5 cm will not likely pass through the stomach in children and therefore should be removed.
- **Button battery**
  - A true emergency if impacted in esophagus
    - Burns can occur within 4 hours.
    - Perforation can occur within 6 hours.
  - Emergent endoscopic removal should be performed if the battery is lodged in the esophagus.
    - If the battery passes into the stomach, the patient can be observed up to 48 hours to allow passage; if the battery has not passed the pylorus by that time or if any GI symptoms (GI bleed or abdominal pain) occur at any time, emergent endoscopic removal is required.
“Body Packers”—drug smugglers who ingest large amounts of carefully wrapped drugs
- Should be admitted for observation and whole bowel irrigation, because rupture of package is often deadly
- Endoscopy is generally avoided, because instrumentation may lead to rupture.

**Varices**
- Dilated venous vessels usually found in patients with portal hypertension (most frequent cause in the United States is alcoholic liver disease)
- Account for only 6% of all GI bleeds, but have high recurrence and mortality rate
- Initial treatment is primarily supportive—IV fluids, transfusions, consider Sengstaken-Blakemore tube
  - Should also consider
    - Octreotide bolus and infusion
    - Vasopressin infusion (less commonly used than octreotide): causes non-selective vasoconstriction; therefore, nitroglycerin infusion should also be given to prevent coronary ischemia
- Endoscopic therapy is up to 90% successful in controlling hemorrhage.
- Emergent transjugular intrahepatic portosystemic shunt (TIPS) should be considered if bleeding cannot be controlled endoscopically.
- Prevention strategy is sclerotherapy or band ligation; β-blockers reduce risk of rebleed.

### 3.3 LIVER

**Cirrhosis**
- **General**
  - Irreversible scarring of the liver from a number of causes results in portal hypertension and shunting of blood flow.
- **Presentation**
  - Classic stigmata include jaundice, spider angioma, palmar erythema, gynecomastia, ascites, pedal edema, testicular atrophy, and hepatic encephalopathy.
- **Complications**
  - **Encephalopathy**
    - Accumulation of toxic substances in the blood
    - Precipitants include infection, sedative medications, increase in nitrogen load to liver (dietary, GI bleeding), overzealous diuresis, hypoxia, and hypoglycemia.
    - Presentation includes altered mental status, asterixis, and elevated ammonia level.
    - Treatment is supportive; address the underlying cause (e.g., infection); oral lactulose and neomycin; admission
  - **Ascites**
    - Precipitants as for encephalopathy; also poor medication and dietary compliance; tends to be recurrent
    - Patients may present with spontaneous bacterial peritonitis—fever, ascites, abdominal pain, increased liver function tests; asymptomatic in 30%
      - Diagnosis with paracentesis cell count, culture
      - WBC count >500/mm³ with >250 PMNs/mm³ is suggestive; culture is definitive
      - *Escherichia coli* and *Streptococcus* species are most common.
      - Treat with broad-spectrum antibiotics (third-generation cephalosporin).
Hepatorenal syndrome
- Acute renal failure in the face of decompensated cirrhosis
- Caused by vasoconstriction and shunting of blood flow away from the renal cortex, producing azotemia
- Mortality rate approaches 100%.

Varices (see previous section on esophageal disorders)

Infectious/Inflammatory Disorders
- Hepatitis
  - Viral
    - Hepatitis A (HAV)
      - Caused by an RNA virus
      - Spread by fecal-oral route, person-to-person, or through ingestion of contaminated water/food (especially shellfish)
      - Incubation period, 15 to 50 days
      - Usually a mild course; no chronic carrier state or subsequent cirrhosis; fulminant hepatic failure is rare
      - If presentation is within 2 weeks after exposure, administer prophylaxis with immune globulin.
      - Treatment is primarily supportive; hospitalize if the patient has signs of encephalopathy, has INR >3, has bilirubin >20 mg/dl, or is immunosuppressed.
    - Hepatitis B (HBV)
      - Caused by a DNA virus
      - Spread by percutaneous or sexual exposure (blood, semen, saliva); perinatal transmission is possible
      - Incubation period >2 months (average, 3 months)
      - 5% to 10% will develop chronic hepatitis or a carrier state, which predisposes the patient to hepatocellular cancer. Risk of developing carrier state is 90% if the virus is contracted at birth.
      - Presentation may be mild or fulminant
      - Serologic markers
        - HBsAg = active/infective
        - HBsAB = immunity, positive 2 to 6 months after clearance of HBsAg
        - HBeAB IgM = recent infection, often in the “window” period, where it may be the only serologic marker of infection; positive 2 weeks after HBsAg becomes positive
        - HBeAB IgG = remote infection
        - HBeAg = ongoing viral replication, very high infectivity
      - Treatment of acute disease is supportive; chronic HBV may be treated with lamivudine or interferon.
      - HBIG and HBV vaccines are available.
    - Hepatitis C (HCV)
      - Caused by an RNA virus
      - Most common cause of viral hepatitis in the United States
      - Spread most commonly by percutaneous route, but also sexual exposure
      - Incubation period, 15 to 160 days
      - Acute illness is milder than for HBV, but chronic hepatitis develops in 50% to 85% of patients, many of whom will develop cirrhosis, some of whom may develop hepatocellular cancer
      - Treatment of acute disease is supportive, but some have suggested interferon and ribavirin therapy for chronic HCV infection.
      - No effective prophylaxis or vaccine
**Hepatitis D (delta, HDV)**
- Caused by a “defective” RNA virus; requires co-infection with HBV
- Superinfection with HBV has higher mortality, higher rate of chronic infection, and higher rate of cirrhosis than HBV infection alone.
- Spread primarily through percutaneous exposure
- No specific prophylaxis or vaccine, but vaccinating against HBV prevents HDV as well

**Hepatitis E (HEV)**
- Caused by an RNA virus
- Spread by fecal-oral route
- Incubation period, 15 to 60 days
- Clinical course is similar to that for HAV but associated with higher rates of fulminant liver failure and mortality, especially during pregnancy (20% mortality rate in the third trimester).
- No chronic or carrier state
- No vaccine or prophylaxis

**Cytomegalovirus (CMV)**
- Latent infection, reactivates with immunosuppression
- Most common opportunistic viral infection after liver transplant
- Treat with gancyclovir

**Chemical/toxin-induced**

**Alcoholic**
- The most common form of chemical hepatitis
- Patients may present with acute hepatic failure or cirrhosis.
- Symptoms include RUQ pain, fever, weakness, anorexia, nausea, jaundice, and dark urine.
- Signs include low fevers, hepatomegaly, jaundice, and typical stigmata of liver disease (spider angio­mata, palmar erythema, testicular atrophy, ascites, etc.).
- Lab results include pancytopenia; increased transaminases, 2 to 10 times normal (AST > ALT); INR elevation (>8 portends a bad prognosis); and hypoglycemia.
- Treatment is supportive (abstinence is key); manage GI bleeds and systolic blood pressure, vitamin K for coagulopathy, treat alcohol withdrawal, glucose supplementation

**Hepatotoxins that produce liver changes resembling viral hepatitis**
- Halothane
  - Toxicity caused by toxic metabolite and hypersensitivity reaction
  - Abrupt onset of fever, rash, pain, jaundice, eosinophilia 2 to 3 days after exposure to the anes­thetic
  - Fatality rate is 50%; no specific treatment—may require liver transplant
- Methyldopa
  - Toxicity caused by toxic metabolite and hypersensitivity reaction
  - Presentation includes abdominal pain, rash, arthralgias, lymphadenopathy, jaundice.
  - Chronic hepatitis and cirrhosis may develop.
- Isoniazid (INH)
  - Toxicity caused by toxic metabolite
  - Incidence increases with age; rare in young adults
  - Increased susceptibility in alcoholics and patients concurrently using rifampin or pyrazinamide
- Phenytoin
Hepatotoxins that produce cholestatic changes
- Anabolic steroids, oral contraceptives, chlorpropamide, chlorpromazine, erythromycin estolate, phenobarbital
- Presentation includes malaise, anorexia, nausea, and vomiting.
Hepatotoxins that produce massive hepatic necrosis
- Acetaminophen, carbon tetrachloride, mushrooms (see Toxicology chapter)

3.4 GALLBLADDER AND BILIARY TRACT

Cholangitis—purulent biliary tree infection
- Presentation
  - Charcot's triad of fever/chills, jaundice, and RUQ pain
  - Severe cases also have mental status changes and shock (Reynolds' pentad)
- Treatment
  - Early antibiotics and surgical consultation for drainage (surgical emergency)

Cholecystitis
- General Information
  - Inflammation of the gallbladder due to obstruction of the cystic duct or common bile duct (CBD) by a stone
  - Acalculous cholecystitis (5%–10% of cases) usually occurs in postoperative, burn, major trauma, diabetic, septic, post-partum, and elderly patients and in children.
    - Associated with biliary sludge
    - Increased susceptibility to gangrene and perforation
    - Requires early surgery or drainage; no "cooling off" period
- Presentation
  - RUQ pain radiating to right infra-scapular region of the back, accompanied by nausea and vomiting
  - Often follows large fatty meals
  - Classically, patients have fever and leukocytosis but these are often absent, especially in the elderly.
  - Elevations of bilirubin and alkaline phosphatase suggest a CBD stone but are not always present in cholecystitis.
  - Elevations in the results of other liver function tests are unreliable as indicators of cholecystitis.
- Diagnosis
  - Ultrasound is highly sensitive and specific.
    - Sonographic Murphy's sign, thickening of gallbladder wall, pericholecystic fluid, stones/sludge, dilated CBD
- Complications
  - Cholangitis, perforation, gangrene, abscess
- Treatment
  - IV antibiotics, antiemetics, IV fluids, and surgical consultation

Cholelithiasis
- General Information
  - Refers to the presence of biliary stones
  - Choledocholithiasis refers to the presence of a stone in the CBD.
  - Occurs in 30% of those over the age of 40
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• Presentation
  o Patients often report biliary colic symptoms—intermittent bouts of RUQ pain that is constant (not “colicky”), usually occurring 30 to 45 minutes after large fatty meals.
  o The pain is caused by contraction of the gallbladder, with transient obstruction of the cystic duct or CBD by the stone.

3.5 PANCREAS

Pancreatitis

• General Information
  o Alcohol is the most common cause of pancreatitis in the inner-city population; gallstone disease is the most common cause elsewhere; alcohol is the most common cause of chronic pancreatitis.
  o Other causes include drugs (e.g., thiazides), metabolic disorders (e.g., hyperlipidemias, hypercalcemia, uremia, diabetic ketoacidosis [DKA]), infections (e.g., mumps, mononucleosis), iatrogenic factors (e.g., recent surgery, endoscopic retrograde cholangiopancreatography [ERCP]), posterior penetrating peptic ulcers, penetrating and blunt trauma, and “exotic” exposures (e.g., scorpion bites).

• Presentation
  o Constant epigastric pain radiating to the back, nausea, vomiting, dehydration, upper abdominal tenderness with guarding.
  o Severe cases—hypotension; Grey-Turner sign (bluish discoloration of the flanks) and Cullen’s sign (bluish discoloration of the periumbilical area), caused by hemorrhagic pancreatitis with retroperitoneal blood; jaundice may occur with biliary obstruction.

• Diagnosis
  o Elevation of amylase and lipase levels (the lipase level is more specific and tends to parallel the clinical course).
  o Pancreatic calcifications may be seen with chronic pancreatitis on abdominal x-ray film.
  o Chest film may demonstrate left pleural effusion or ARDS seen with severe disease.
  o CT scan demonstrating edema is most reliable but may also show evidence of hemorrhage, abscess, or pseudocyst.

• Prognosis (Ranson’s Criteria)
  o On admission
    ▪ Age >55 years
    ▪ Glucose >200 mg/dL
    ▪ WBC >16,000/ml
    ▪ LDH >350 IU/L
    ▪ AST >250 SF units
  o After 48 hours
    ▪ \( pO_2 < 60 \) mm Hg (consider ARDS)
    ▪ Fall in Hct >10% (consider hemorrhagic pancreatitis)
    ▪ Base deficit >4 mEq/L
    ▪ Calcium <8 mg/dL
    ▪ Fluid sequestration >6 liters
    ▪ Rise in BUN >5 mg/dL
  o Mortality increases significantly with the number of positives.
    ▪ 0–2 criteria: <5% mortality
    ▪ >6 criteria: >80% mortality

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• Treatment
  o IV fluids, NPO (bowel rest), analgesics, antiemetics are routine
  o PRBCs in cases of hemorrhagic pancreatitis
  o Antibiotics indicated only in sepsis or concurrent biliary infections (e.g., cholangitis)
  o NG tube only for intractable vomiting

3.6 PERITONEUM

Spontaneous Bacterial Peritonitis—see section on liver disease/ascites

3.7 STOMACH

Peptic Ulcer Disease
• General Information
  o 20% occur in stomach, 80% in duodenum
  o Mucosal lesion resulting from inability to handle peptic acid load in a focal area
  o “Gastritis” refers to diffuse mucosal involvement
• Predisposing Factors
  o Use of NSAIDs, ASA, corticosteroids, tobacco, caffeine, stress
  o Helicobacter pylori infection has been identified as a direct cause in many cases, but treatment requires verification (empiric treatment in the ED is not currently recommended).
• Presentation
  o Stomach—epigastric burning classically occurs soon after meals
  o Duodenum—epigastric burning classically occurs a few hours after meals; penetrating duodenal ulcers may produce pain radiating to the back
• Complications
  o Hemorrhage
    ▪ Duodenal ulcers are the most common cause of upper GI bleeds.
  o Perforation
  o Scarring caused by ulcers can cause pyloric obstruction.
• Treatment
  o Antacids, H2 blockers, proton pump inhibitors (PPIs), mucosal surface protectants (sucralfate)
  o Antibiotics targeted against H. pylori in patients who test positive
  o Patients with bleeding ulcers should receive IV fluids and transfusions; consider IV proton pump inhibitors.

3.8 SMALL BOWEL

Inflammatory Disorders—Regional Enteritis/Crohn’s Disease/Terminal Ileitis
• General information
  o Can involve any area of the GI tract from mouth to anus
  o Pathology characterized by full-thickness bowel wall lesions, “skip” lesions, and appearance of “cobblestone” mucosa.
  o Family history in 10% to 15%
  o Most common in young adults; more common in Jews
• Presentation
  o Abdominal pain, pseudo-appendicitis, cramping, diarrhea, fever, rectal fistula or prolapse, and perianal abscesses; occult blood and fecal leukocytes are common
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• **Complications**
  o Intestinal: stricture and obstruction, GI bleed, perforation, GI malignancies of either small or large bowel
  o Extraintestinal: arthritis, uveitis, dermatologic disorders

• **Treatment**
  o Rule out obstruction
  o IV fluids, steroids, antidiarrheal agents (should not be used in patients with active colitis), antibiotics, sulfasalazine, azathioprine (can be steroid sparing)
  o Treatment should be administered in conjunction with a primary care physician or GI physician.
  o GI consultation and admission for patients who are dehydrated or who have significant electrolyte abnormality, severe exacerbations, or acute complications (listed above)

**Motor Abnormalities**

• **Obstruction**
  o Most common cause is the presence of adhesions; in patients without prior surgeries, most common cause is hernia
  o Presentation includes nausea, vomiting, abdominal distension, and diffuse abdominal pain.
  o Bowel sounds are hyperactive; diarrhea may occur early.
  o Plain films miss up to 30%; usually demonstrate dilated air-filled loops of small bowel and lack of air in large bowel; air–fluid levels may be present in small bowel
  o Treatment—early surgical consultation, IV fluids, NPO, NG decompression

• **Paralytic Ileus**
  o More common than mechanical obstruction
  o May be caused by medications, infections, electrolyte abnormalities, other GI or systemic stressors
  o Presentation is similar to that of mechanical obstruction, but bowel sounds are hypoactive.
  o Plain films generally show distended air-filled loops of small and large bowel.
  o Treatment—IV fluids, NPO, NG decompression, treat the underlying cause (e.g., infection)

**Vascular Insufficiency**

• **General Information**
  o Risk factors include atrial fibrillation, hypotensive states, low-flow states, hypercoagulable states (especially for venous thrombosis), peripheral vascular disease, digoxin (causes splanchnic vasoconstriction), and hemodialysis.
  o 50% of cases are caused by emboli to the superior mesenteric artery (SMA); remainder are related to mesenteric artery thrombus formation, mesenteric venous thrombosis, or nonocclusive ischemia.
  o Mortality <50% with early diagnosis and treatment, but >80% if diagnosis is delayed and infarction occurs

• **Presentation**
  o Classic presentation is sudden onset of abdominal pain (SMA embolus) with “pain out of proportion” to physical findings. One third have had a previous embolic event.
  o SMA thrombus causes “intestinal angina” with repeated episodes of post-prandial pain and long-term weight loss due to food aversion. Plaque rupture leads to acute event (similar to myocardial infarction).
  o Mesenteric venous thrombosis results in gradual onset of pain. Women >> men; half have a personal or family history of DVT/PE.
  o Nonocclusive mesenteric ischemia usually occurs in low-flow states (e.g., severe CHF or use of vasopressors), typically in ICU patients.
Early “benign” abdominal pain with vomiting and diarrhea may result in misdiagnosis (of gastroenteritis) or delayed diagnosis, especially in elderly patients.

Significant tenderness or peritoneal findings imply that infarction has already occurred.

**Diagnosis**

- Diagnosis should be considered early based on clinical presentation and risk factors.
- Patients usually have markedly elevated WBC count, elevated lactate, and elevated phosphate.
- Metabolic acidosis and heme-positive stools are late findings.
- Plain films are usually normal/nonspecific early on; later, may demonstrate bowel obstruction/ileus, thickening of the bowel wall (“thumbprinting”), gas within the bowel wall (“pneumatosis intestinalis”), air within the portal venous system.
- CT may demonstrate any of the above findings as well; multidetector CT angiography may yield results comparable to conventional angiography and appears promising as a new modality.
- Diagnostic gold standard is angiography and should be ordered early when the diagnosis is suspected. Early angiography is the only thing proven to reduce mortality.

**Treatment**

- Early surgical consultation at the first suspicion is mandatory.
- IV fluids, antibiotics, NPO, NG decompression, and Foley catheter (to monitor perfusion and in anticipation of surgery)
- Papaverine (intra-arterial vasodilator) can be infused during angiography and may be therapeutic. It is necessary to treat associated vasospasm even if surgical revascularization is successful.
- Intravenous heparin is used for mesenteric venous thrombosis if infarction and necrosis have not yet occurred. Should be considered for all causes of mesenteric ischemia, but the timing is controversial and should be discussed with the treating surgeon.
- The need for emergent surgery is based on the results of the angiogram and response to papaverine.

### Infectious Disorders

**Antibiotic-Associated Colitis (Pseudomembranous Colitis)**

- Caused by *Clostridium difficile* toxin, which can be identified in the stool.
- Most common in inpatients, postoperative patients, and nursing home patients.
- Usually begins within 6 to 10 days after starting antibiotics, but onset may be delayed as long as 6 weeks.
- Associated with crampy abdominal pain and watery (may be bloody) diarrhea.
- Treatment involves discontinuation of antibiotics, initiation of IV fluids and electrolyte replacement, and oral metronidazole or oral vancomycin.
- Avoid antimotility drugs (especially diphenoxylate), as they may worsen symptoms and predispose the patient to toxic megacolon.

**Bacterial—20% of cases of acute diarrhea**

- **Inflammatory (invasive)**
  - General information
    - Produce diarrhea by damaging cell membranes, leading to invasion, and producing an inflammatory response.
    - Primarily act on colon or distal small bowel.
    - Symptom onset tends to be more gradual and associated with constitutional symptoms (malaise, fevers, worse abdominal pain than with enterotoxin-producing bacteria).
Diarrhea is usually bloody or mucoid but of small volume.
Fecal leukocytes and occult (or gross) blood tests are usually positive.
Generally, this condition is self-limited, but severe cases are usually treated with antibiotics, especially quinolones (see specifics below).
There is significant controversy regarding the role of antimotility agents in acute diarrheal illnesses; however, on the board examination, antimotility agents should generally be avoided (exceptions are listed below).

**Salmonella**
- Primarily results from ingestion of contaminated eggs and poultry; transmission from person-to-person and animal-to-person (from pet turtles, iguanas, dogs, and cats) can occur
- Typically causes gastroenteritis but may also cause dysentery, typhoid fever, and carrier state
  - Typhoid (enteric) fever produces septicemia and a protracted course, including persistent fevers, relative bradycardia, cramps without much diarrhea, and skin lesions ("rose spots")
- Self-limited, but patients with severe disease or immunocompromised states should be treated with a fluoroquinolone (drug of choice) or TMP-SMX. Antibiotics are not indicated for uncomplicated, nontyphoidal Salmonella, as they may prolong the carrier state.
- Patients with typhoid fever (enteric fever) should be treated with ceftriaxone.

**Shigella**
- Fecal-oral transmission; can be spread by as few as 50 organisms
- High fevers, crampy pain, diarrhea (one-third develop dysentery [bloody mucoid stools]) develop 1 to 2 days after exposure. Tenesmus is classic.
- Classic presentation in a child is febrile seizure with diarrhea.
- Self-limited, but antibiotics (quinolones [avoid in children], TMP-SMX, ampicillin) are recommended in severe cases
- Complications include arthralgias, Reiter's syndrome (arthritis, urethritis, conjunctivitis), and hemolytic uremic syndrome (HUS).

**Campylobacter**
- The most common cause of bacterial diarrhea in the United States
- Primarily results from ingestion of contaminated poultry or direct contact with feces from infected animals; also may be transmitted sexually
- Presentation includes nausea, cramps, and diarrhea; abdominal tenderness may be localized, producing the appearance of a surgical abdomen, especially if in RLQ
- May produce dysentery
- Self-limited, but may be treated with a quinolone or erythromycin (drug of choice)
- Complications include Reiter's syndrome, HUS, Guillain-Barré syndrome

**Yersinia**
- Primarily results from ingestion of contaminated food or water; often there is a history of recent exposure to domestic, farm, or wild animals (direct transmission occurs)
- Presentation includes fevers, cramps, diarrhea; dysentery is common.
- May result in mesenteric adenitis or terminal ileitis, thus mimicking appendicitis ("pseudo-appendicitis")
- May result in polyarthritis or erythema nodosum
- Self-limited, but severe infections may be treated with a quinolone, TMP-SMX, or ceftriaxone
- Enterohemorrhagic *E. coli* (serotype O157:H7)
  - Produces a cytopathic toxin that invades and destroys the intestinal wall, causing a clinical syndrome similar to that induced by other invasive organisms
  - Usually caused by ingestion of undercooked beef or unpasteurized milk or person-person (fecal-oral)
  - Watery diarrhea occurs early, bloody diarrhea later, with nausea, vomiting, severe cramps, and low fevers (if any)
  - Hemolytic uremic syndrome (HUS) may develop in children, thrombotic thrombocytopenic purpura (TTP) in elderly
  - Antibiotic therapy is *not* recommended, as it may increase the chances of HUS or TTP, especially in children and elderly.

- *Clostridium difficile*
  - Produces a cytopathic toxin that invades and destroys the intestinal wall, causing a clinical syndrome similar to other invasive organisms
  - See above section on antibiotic-associated colitis.

- *Vibrio parahaemolyticus*
  - Primarily results from ingestion of shellfish and other raw or improperly prepared seafood (the leading cause of seafood-associated gastroenteritis in the United States)
  - Presentation includes nausea, vomiting, cramps, diarrhea; fever and dysentery may occur.
  - Self-limited, but quinolones or tetracyclines may be given in severe cases

  - Non-inflammatory (enterotoxin-producing)

- General information
  - These bacteria release a toxin that produces diarrhea by altering electrolyte transport in epithelial cells.
  - Toxin acts primarily on small bowel.
  - Symptom onset tends to be abrupt.
  - Large-volume, watery stools without blood, pus, or severe abdominal pain
  - Fecal leukocytes and occult blood tests are usually negative.

- *Staphylococcus aureus*
  - The most common cause of food poisoning
  - Release of a preformed enterotoxin after ingestion of protein-rich foods (eggs, mayonnaise-containing products) that are contaminated with the organism
  - Presentation includes rapid onset (within 4 to 6 hours after ingestion) of vomiting, cramps, and diarrhea.
  - Usually resolves within the first day

- *Bacillus cereus*
  - Primarily results from ingestion of contaminated fried rice
  - Rapid onset of nausea, vomiting, cramps within few hours; diarrhea is much less common
  - A diarrheal syndrome may occur later, usually the result of ingestion of contaminated meat or vegetables (classically meatballs).
  - Self-limited; resolves in 12 to 24 hours

- Enterotoxigenic *E. coli*
  - Most common cause worldwide of traveler's diarrhea
  - Fecal-oral transmission
  - Watery diarrhea (frequent, explosive) with cramps
Self-limited, though use of antibiotics (3-day course of ciprofloxacin) and loperamide (should not be continued for more than 2 days and should not be given to patients with fevers or bloody stools) shortens the duration.

Bismuth subsalicylate (Pepto-Bismol) may shorten the duration or serve as prophylaxis, but large doses are required.

- **Clostridium perfringens**
  - Primarily results from ingestion of contaminated meat and poultry
  - Live organism produces enterotoxin in the bowel, causing diarrhea and cramps.
  - Fever, headaches, chills may occur
  - Self-limited

- **Vibrio cholera**
  - Primarily results from the ingestion of contaminated water or shellfish; the enterotoxin-produces a diarrheal illness
  - Incubation period is 1 to 2 days.
  - Presentation includes profuse watery diarrhea ("rice-water stools") and abdominal distension; vomiting is less prominent.
  - Main complication is severe dehydration, including hypokalemia and hyperchloremic acidosis.
  - Treatment includes vigorous isotonic rehydration.
  - Antibiotics may be used—tetracycline, doxycycline, TMP-SMX; quinolones shorten the duration of illness and decrease excretion of the organism

- **Scombroid**
  - Primarily results from ingestion of heat-stable toxins produced by bacteria in dark-meat fish (e.g., mahi mahi, tuna, mackerel)
  - Usually associated with improper refrigeration or preservation of the fish
  - In many cases, a metallic or peppery taste was noted by the patient at the time of ingestion.
  - The bacteria produce histamine, which produces symptoms—facial flushing and redness, headache, abdominal cramps, vomiting, diarrhea, palpitations, bronchospasm.
  - Treatment includes H1 and H2 blockers, nebulized β-agonists for bronchospasm, and epinephrine if an anaphylactoid reaction has developed.

- **Ciguatera**
  - Primarily results from ingestion of a neurotoxin (ciguatoxin) that accumulates in certain carnivorous tropical fish (e.g., snapper, barracuda, grouper, jack, kingfish) that have consumed large quantities of a toxin-producing dinoflagellate
    - Most commonly occurs in the late spring and summer
    - Toxin is tasteless and heat stable.
  - Incubation is only a few hours.
  - Presentation includes vomiting, diarrhea, myalgias, and neurologic symptoms:
    - Weakness
    - Paresthesias
    - Reversal of hot and cold sensation (classic)
    - Sensation that teeth are falling out (!)
    - Dysesthesias (burning) in hands and feet
  - Neurologic symptoms may last for months and are exacerbated by alcohol.
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- Treatment includes IV fluids and antiemetics, analgesics.
  - Mannitol, 1 gm/kg IV, over 1 hour may help
- Abstinence from alcohol and seafood is recommended until all symptoms have resolved.

- **Aeromonas hydrophila**
  - Primarily results from ingestion of contaminated well water or spring water
  - Tends to affect immunocompromised patients and children more often
  - Presentation includes watery diarrhea, vomiting, and abdominal cramps lasting for weeks.
  - Treatment is best accomplished with antibiotics—quinolones, TMP-SMX, tetracycline, aminoglycosides.

- Parasitic
  - **Entamoeba histolytica**
    - Fecal-oral transmission from asymptomatic carriers who pass the cysts on
    - Presentation includes 1) asymptomatic carriage, 2) mild GI symptoms: abdominal cramps, diarrhea, flatulence, and 3) amebic dysentery: profuse bloody diarrhea, fevers, and severe cramps.
    - Extraintestinal manifestations can include abscesses, most commonly in liver but also in lung and brain and rarely in heart or kidney.
    - Diagnosis is based on finding trophozoites or cysts in stool or by sigmoidoscopy with rectal biopsy; serologic testing can also be done in symptomatic patients.
    - Treatment requires metronidazole (treats invasive disease) plus paromomycin (treats luminal disease).
    - Asymptomatic carriers are treated with iodoquinol (eradicates cyst stage).
  - **Giardia lamblia**
    - Most common intestinal parasite in the United States
    - Primarily results from ingestion of contaminated water, especially from wells and mountain streams; fecal-oral transmission of the cysts
    - Fecal-oral transmission during sexual practice is also commonly reported.
    - Associated with epidemics in child daycare centers and among travelers returning from Russia
    - Presentation includes watery/frothy foul-smelling diarrhea with associated upper GI symptoms (abdominal cramps, bloating, nausea, belching) exacerbated by eating.
    - Most patients are asymptomatic.
    - Diagnosis via ova and parasite testing, stool antigen, string test, or duodenal aspiration to look for trophozoites
    - Treatment of choice is metronidazole, furazolidone suspension, or tinidazole.
  - **Cryptosporidium**
    - Most common cause of chronic diarrhea in AIDS patients
    - Fecal-oral transmission of cysts, which are harbored in animals and humans
    - Primarily occurs in immunocompromised patients but can occur in children in daycare, animal handlers, and travelers (especially to Russia)
    - Presentation includes profuse watery diarrhea, abdominal cramps, nausea, and flatulence.
    - Generally self-limited, but immunocompromised patients can develop significant malabsorption that results in chronic wasting and even death
    - Diagnose by acid-fast stain of stool, demonstrating oocysts.
    - Treatment consists of rehydration, nitazoxanide or paromomycin, and antimotility agents.
      - Immunocompromised patients may get some benefit from these medications, but they are not curative.
• AIDS patients are best treated by stabilizing with the above medications then initiating highly active antiretroviral therapy (HAART).

  o *Isospora belli*
  - Fecal-oral transmission of the cysts
  - Primarily occurs in immunocompromised patients, causing a protracted illness; immunocompetent patients are usually asymptomatic
  - Presentation includes watery diarrhea, cramps, nausea, vomiting, and significant malabsorption, producing wasting.
  - Diagnose by identifying oocysts on acid-fast stain of stool.
  - Moderate eosinophilia is a clue to the diagnosis.
  - Treatment consists of TMP-SMX (long course); alternative is pyrimethamine.

  o *Enterobiasis* (pinworm)
  - The most common helminthic infection in the United States
  - Primarily results from ingestion of pinworm eggs
  - Eggs develop into adult worms in the large bowel, which migrate to the anus and deposit eggs at night.
  - Presentation includes nighttime pruritis ani.
  - Diagnose by using cellophane tape swab of the anus to identify eggs.
  - Treatment consists of mebendazole or pyrantel pamoate, with repeat treatment at 2 weeks; all close contacts should be treated as well.

  o *Necator americanus* (hookworm)
  - Affects one fourth of the world’s population
  - Primarily occurs when the larvae, harbored in feces-contaminated soil, contact and penetrate intact skin
  - Larvae enter the bloodstream, ascend the trachea, and descend the esophagus, migrate to bowels as adults, attach to mucosa, and feed on blood.
  - Presentation includes diarrhea, abdominal pain, fever, cough, weight loss; iron-deficient anemia and protein deficiency malnutrition may develop.
  - Diagnose by identifying ova in stool; eosinophilia is common.
  - Treatment consists of mebendazole, albendazole, or pyrantel pamoate.

• Viral—most common cause of acute diarrhea
  o *Rotavirus*
  - Mainly occurs in young children, <2 years old
  - Presentation includes nausea, vomiting, watery diarrhea, low fevers, and upper respiratory symptoms.
  - Diagnose with enzyme immunoassay (Rotazyme test).
  - Supportive treatment
  - Vaccine is now available.

  o *Norovirus* (previously known as Norwalk virus)
  - Primarily results from ingestion of contaminated foods, especially raw shellfish
  - Mainly occurs in older children and adults
  - Epidemics are common (daycare, cruises, households).
  - Presentation includes nausea, vomiting, watery diarrhea, cramps, low fevers. Vomiting is more common than diarrhea.
  - Supportive treatment
Enteric type adenovirus ("stomach flu")
- Nausea, vomiting, watery diarrhea associated with constitutional symptoms (low fever, malaise, myalgia)
- Self-limited

Inflammatory Disorders
- Acute Appendicitis
  - General information
    - The most common abdominal surgical emergency in the United States
    - Often caused by obstruction by an appendecolith, foreign matter (e.g., seeds), adhesions, etc.
    - May also occur as a result of lymphoid hyperplasia after viral infections
    - Highest incidence in patients 10 to 30 years of age, though the highest misdiagnosis rate is in infants and elderly, because of atypical presentations
  - Presentation
    - Classically begins with vague visceral, poorly localized periumbilical pain with unpleasant GI symptoms (anorexia, nausea) and low fever
    - RLQ pain develops over the course of 24 hours as peritonitis develops.
    - Atypical presentations include RUQ pain (especially in pregnant patients), right low back pain (retrocecal appendix), pelvic/adnexal pain (appendix near ovary), and dysuria (appendix near bladder).
    - Delayed diagnosis is typical in infants and elderly, because of atypical presentations, leading to perforation rates >50% in these groups and associated increased morbidity/mortality.
  - Diagnosis
    - Primarily a clinical diagnosis
    - WBC count elevation in 80%
    - CT is being used with increased frequency in cases of low to moderate clinical suspicion, with >90% sensitivity and specificity.
    - Ultrasound has much lower sensitivity and specificity but is often the primary imaging modality in pregnant women and children.
  - Treatment
    - Early surgical consultation, antibiotics, surgery
- Ulcerative Colitis
  - General information
    - Chronic inflammatory bowel disease of the colon and rectum with colitis, superficial ulcers, crypt abscesses
    - Inflammation is limited to the superficial layers of the bowel (mucosa and submucosa).
    - Unlike Crohn's disease, bowel involvement is continuous (no "skip areas"); rectal involvement is universal.
  - Presentation
    - Abdominal pain (usually lower) and cramps; diarrhea, frequently bloody; fever
    - Suspect this diagnosis in any young patient who presents with bloody diarrhea.
  - Complications
    - Intestinal
      - GI bleeding (massive bleed is uncommon), toxic megacolon, perforation, obstruction, perianal fistulas and abscesses, colorectal cancer (10- to 30-fold increase in risk)
      - Toxic megacolon is the complication that should cause the most concern, because of the associated marked colonic distension (>8 cm), abdominal distension, systemic toxicity, peritonitis, and high risk of perforation.
Extraintestinal

- Arthritis, ankylosing spondylitis, uveitis, erythema nodosum, pyoderma gangrenosum

**Treatment**
- In the absence of toxicity or complications, outpatient management can be done with corticosteroids, sulfasalazine, or 5-aminosalicylic enemas (for patients with ulcerative proctitis and left-sided colitis).
- Inpatient management with all of the above, as well as antibiotics and IV fluids
- Antimotility agents are not recommended, as they may predispose the patient to megacolon.

### Motor Abnormalities

- **Irritable Bowel**
  - Considered a functional disorder; symptoms worsen with stress
  - Presentation includes crampy abdominal pain with alternating diarrhea and constipation.
  - Nocturnal diarrhea is uncommon; if present, consider an alternative diagnosis.

- **Obstruction**
  - **General information**
    - Caused by tumors (65%), volvulus (15%; sigmoid more common than cecal), diverticulitis (15%)
    - Less common causes include ischemic/radiation colitis, inflammatory bowel disease (Crohn's disease, ulcerative colitis), and fecal impaction.
    - Large bowel obstruction near competent ileocecal valve is more prone to rupture than routine large or small bowel obstruction.
  - **Presentation**
    - Diffuse, poorly localized crampy abdominal pain and abdominal distension
    - Occult blood in stool suggests tumor or inflammatory bowel disease.
    - Nausea and vomiting are less common than in small bowel obstruction.
  - **Diagnosis**
    - Plain films show dilated loops of large bowel (>8 cm) or cecum (>6 cm).
    - When the ileocecal valve is competent, small bowel distension is absent.
  - **Treatment**
    - NG tube, NPO, surgical consultation
    - For sigmoid volvulus, sigmoidoscopy/rectal tube is usually successful but may require surgery for definitive treatment, because of the high rate of recurrence.
    - For cecal volvulus, colectomy is usually required.

### Structural Disorders

- **Aortoenteric Fistula**
  - Primarily occurs in patients who have had surgical repair of abdominal aortic aneurysm
  - Fistula develops between abdominal aorta and bowel, allowing transmission of blood from the aorta into the GI tract.
    - Produces GI bleed, often massive and with little pain
    - Usually lower GI bleed, but occasionally upper
  - Early clinical suspicion, prompt surgical consultation, and immediate surgery are mandatory.
  - Any amount of GI bleeding in a patient with a previous abdominal aortic aneurysm (AAA) repair is aortoenteric fistula until proven otherwise.
• Diverticular Disease
  o General information
    ▪ Diverticula are sac-like herniations of colonic mucosa, resulting from colonic muscular dysfunction.
    ▪ Diverticula form at the weaker points in the mucosa, generally where blood vessels penetrate the mucosa (thus the association with profuse GI bleeding in some patients).
    ▪ Most commonly occurs in sigmoid colon
    ▪ Associated with low-fiber, Western diet
    ▪ Two thirds of elderly patients have diverticular disease, 20% of whom become symptomatic.
  o Diverticulitis
    ▪ General information
      ▪ Painful inflammation resulting from fecal plugging of a diverticular neck, with subsequent bacterial invasion and proliferation
      ▪ The most frequent complication of diverticular disease
    ▪ Presentation
      ▪ Stool contains occult blood in 50% of cases, but massive lower GI bleeding is uncommon.
      ▪ Early presentation involves alternating constipation (primarily) and diarrhea (mild) accompanied by diffuse low abdominal pain (usually LLQ > RLQ), low fevers, normal or slightly elevated WBC count, and normal appetite.
      ▪ May cause urinary symptoms if inflammatory reaction is near the bladder
        – Commonly misdiagnosed as urinary tract infection (UTI) (can have WBCs in urine) or pelvic mass of gynecologic origin (if abscess is present)
      ▪ More advanced stages or abscess formation can be associated with anorexia, obstipation, high fevers, severe pain, perforation.
    ▪ Diagnosis
      ▪ Plain films may be useful to evaluate for complications (ileus, obstruction, free air).
      ▪ Abdominal CT scan with oral or rectal contrast can demonstrate evidence of diverticular disease as well as the presence of inflammatory reaction, abscess, or perforation.
        – Water-soluble contrast should be used in the event of a perforation.
    ▪ Treatment
      ▪ Mild disease can be treated on an outpatient basis with high-fiber diet, analgesics, antibiotics (to cover bowel flora), and early follow-up.
      ▪ Patients with more advanced disease, as well as immunocompromised patients and patients without follow-up, should be admitted and receive IV antibiotics, IV fluids, bowel rest (NPO), and analgesics.
      ▪ Surgical consultation should be obtained on admitted patients; up to 40% will require surgery.
  o Diverticulosis
    ▪ General information
      ▪ The most common cause of lower GI bleeding in adults
      ▪ The most common cause of massive rectal bleeding in elderly
      ▪ Bleeding usually stops spontaneously.
      ▪ Accompanying weight loss or mucus in stool suggests ulcerative colitis.
    ▪ Presentation
      ▪ Hematochezia
      ▪ Bleeding tends to be sudden, painless, and massive.
      ▪ Significant tenderness with lower GI bleeding suggests an alternative diagnosis.
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- Diagnosis
  - Definitive diagnosis with sigmoidoscopy, endoscopy, or mesenteric arteriography

- Treatment
  - Primarily supportive care; if bleeding exceeds transfusion ability, consider mesenteric arteriography for embolization, endoscopy with therapeutic measures, or surgery (hemicolecotomy)

- Volvulus
  - General information
    - Closed loop of the large bowel, resulting in obstruction
    - Accounts for approximately 10% to 20% of large bowel obstructions in the United States
    - May also result in mesenteric ischemia or infarction if vascular supply is compromised
  - Sigmoid volvulus
    - Accounts for two thirds of volvulus cases
    - Classically occurs in bed-ridden nursing home patients with chronic constipation
    - Chronic constipation leads to redundancy of bowel, especially sigmoid, and predisposes to volvulus
    - May result in gradual or sudden onset of abdominal pain with distension, nausea, vomiting, and obstipation
    - X-ray film demonstrates dilated twisted loop of distal bowel arising from left side of abdomen.
  - Cecal volvulus
    - Accounts for one third of volvulus cases
    - Classically occurs in otherwise healthy patients 20 to 40 years of age
    - Association with marathon runners
    - Results from embryologic defect (proximal portion of large bowel is not properly fixed to the posterior abdominal wall)
    - Typically results in sudden onset of abdominal pain with progressive distension, nausea, vomiting, and obstipation
    - X-ray film demonstrates dilated loop of proximal large bowel arising from right side of abdomen (“coffee bean”), markedly dilated cecum, possibly dilated loops of small bowel as well
  - Treatment
    - IV fluids, NG tube decompression, surgical consultation
    - If mesenteric ischemia or infarction is suspected, initiate early broad-spectrum antibiotics.
    - Sigmoid volvulus can be treated with reduction of the obstruction via rectal tube.
    - Cecal volvulus requires early surgical reduction.

3.10 RECTUM AND ANUS

Infectious Disorders
- Perirectal Abscess
  - Types include perianal, intersphincteric, supralevator, ischiorectal, and deep postanal
  - Usually result from obstruction of glands at the base of the anal crypts
  - May also be associated with inflammatory bowel disease, radiation therapy, STDs (e.g., lymphogranuloma venereum)
  - Infections are polymicrobial.
  - Presentation includes pain; constitutional symptoms may occur (low fevers, malaise); when proximate to the anal canal, painful bowel movements are common (digital rectal examination should be done to assess for anal involvement).
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- Treatment is incision and drainage (I&D); perianal abscesses may be drained in ED, others usually require I&D in the OR.
- Antibiotics are indicated if there is an accompanying cellulitis, if the patient is immunocompromised (HIV, diabetes, etc.), and if the patient has a history of valvular problems (endocarditis, prosthetic valve, etc.).

- Pilonidal Cyst and Abscess
  - Occurs at superior edge of buttock in midline
  - Men>>women; rare after age 40
  - An ingrowing hair penetrates the skin and induces a foreign body reaction, leading to the development of a sinus tract that becomes plugged and forms an abscess.
  - Treatment involves bedside I&D followed by (non-emergent) surgical excision of the sinus tract.

Structural Disorders
- Anal Fissure
  - The most common cause of painful rectal bleeding in both adults and children
  - Highly associated with constipation and passage of hard stools
  - Pain usually accompanies bowel movements; streaks of blood on stool
  - Usually occur in the posterior midline; if not, consider more serious pathology (e.g., inflammatory bowel disease)
  - Treatment consists of stool softeners and sitz baths; patient should be advised to use moist tissue when wiping after bowel movements until symptoms subside.

- Anal Fistula
  - Tract between anal canal and skin lined with granulation tissue, often resulting from a perirectal abscess, inflammatory bowel disease, cancer, or radiation therapy
  - Open tract results in persistent purulent discharge; clogged tract results in abscess.
  - Treatment involves I&D of abscess followed by definitive surgical excision of the tract.

- Foreign Body
  - If presentation includes abdominal pain, suspect perforation of bowel.
  - Complications include perforation, lower GI bleeding if mucosal tears occur, and embarrassment.
  - Plain films are helpful for localizing the foreign body, determining location and number, and assessing for perforation (look for free air).
    - Be aware that the sensitivity of plain films for detecting lower GI tract perforation is <50%.
    - If perforation is strongly suspected, consider CT scan.
  - Surgical consultation should be obtained in all but the most simple cases to aid in removal.
  - Following removal of the foreign body, patients should be observed for 6 to 12 hours.
    - Some suggest repeat plain films, CT scan, and/or sigmoidoscopy prior to discharge to rule out perforation or mucosal tears, which may occur during removal.

- Hemorrhoids
  - Associated with chronic constipation
  - Common cause of bright red blood per rectum (BRBPR), usually a mild amount
  - Bleeding tends to occur when straining during bowel movements.
  - Internal hemorrhoids (above the dentate line) are painful only when prolapsed.
  - External hemorrhoids become painful when thrombosed.
  - Abdominal pain should prompt consideration of an alternative diagnosis.
Treatment

- Manual reduction of prolapsed internal hemorrhoids; bulk laxatives, sitz baths, suppositories for pain/pruritis
- For thrombosed external hemorrhoids, incise and remove clot.
- Thrombosed internal hemorrhoids should never be incised in the ED.
- Surgery is indicated if bleeding continues, if strangulation/incarceration is present, or if the patient is experiencing intractable pain.

Rectal Prolapse

- Mostly occurs in the very young (always consider underlying pathology, including cystic fibrosis) and the elderly
- Presentation consists of the sensation of an anal mass (usually associated with minimal, if any, pain) following a Valsalva maneuver (vigorous coughing, bowel movement, heavy lifting, etc.).
- Concentric rings help differentiate rectal prolapse from prolapsed internal hemorrhoids.
- Treatment consists of manual reduction, followed by surgical consultation for surgical repair.
  - Patients should avoid further Valsalva maneuvers (e.g., no heavy lifting, use of stool softeners).
  - If vascular compromise is present (suspect if the patient has severe pain or if the prolapsed segment is pale), immediate surgical consultation is mandatory.
CHAPTER 4
Thoracic and Respiratory Disorders

Sara J. Levy, MD, and Joseph R. Lex, Jr., MD

4.1 ACUTE UPPER AIRWAY DISORDERS
Infections
- Croup (laryngotracheitis or laryngotracheobronchitis) (see Chapter 5, Pediatrics)
- Epiglottitis (for pediatric epiglottitis, see Chapter 5, Pediatrics)
  - General
    - Inflammation and edema of supraglottic structures
    - May spare epiglottis itself
    - Early presentations are often missed in adults.
    - Causative organism is rarely isolated; when cultures are positive, usually with Hemophilus influenzae type b
    - Other organisms include Group A β-hemolytic Streptococcus, Staphylococcus aureus, and Streptococcus pneumonia.
    - Noninfectious causes, e.g., thermal injury, are uncommon.
  - Presentation
    - Acute illness: severe sore throat, dysphagia, odynophagia, stridor, or dysphonia (voice changes) may be present
    - Upper respiratory infection (URI) prodrome typically 1 to 2 days (up to 7 days)
    - Patients may appear anxious or toxic.
    - High fever is common but is absent in 50% of cases.
    - Rapid progression in as little as several hours; assumption of sniffing position portends the need for an immediate and definitive airway
    - Tenderness may be present on the anterior neck near the hyoid or with manual lateral movement of the larynx.
  - Diagnosis
    - Classic epiglottitis is a clinical diagnosis: ongoing airway monitoring and management supersede the need for additional testing.
    - Any patient with respiratory distress should have the airway secured immediately, with a surgical airway if required.
    - Drooling and dysphonia are earlier signs; stridor and classic “respiratory distress” may not appear until just prior to complete airway obstruction.
Great caution with diabetics, immunocompromised individuals, and those with a rapidly progressive course

“Double setup”: direct laryngoscopy with equipment for cricothyroidotomy at bedside

All patients suspected of epiglottitis should undergo direct or indirect laryngoscopy; epiglottis may be normal but with severe supraglottic swelling; be prepared for surgical airway.

Epiglottis can be either red or pale with edema

Pharyngitis, uvulitis, tonsillitis, or peritonsillar abscess may coexist with epiglottitis.

Lateral neck x-ray film classically shows “thumbprint”-shaped epiglottis, dilated hypopharynx, and lack of air in the vallecula. (see Image #1)

Normal x-ray films do not rule out adult epiglottitis; if suspicion of this diagnosis remains, laryngoscopy is warranted.

Treatment

Ensuring continued airway patency is paramount; maintain patient upright with supplemental oxygen; do not agitate, transport, or reposition patient unnecessarily.

Respiratory failure, actual or impending, must be managed rapidly by experienced personnel; the proper endotracheal tube size may be smaller than predicted by patient's body size secondary to airway edema.

Oral laryngoscopy and nasopharyngeal flexible fiberoptic laryngoscopy are both options; they allow direct visualization of cords during intubation.

“Blind” nasopharyngeal intubation is contraindicated because it may cause obstruction.

If intubation is unsuccessful, an emergent surgical airway may be necessary; equipment should be at hand.

After the airway has been secured, additional tests, treatment, and supportive care may be performed.

Antibiotic coverage should include H. influenzae and other common pathogens; cultures should be drawn for later targeting of coverage.

Cefotaxime and ceftriaxone are first line; ampicillin-sulbactam, or trimethoprim-sulfamethoxazole are other options.

Patients without respiratory distress may be monitored carefully after laryngoscopy without a definitive airway.

If the patient has only mild swelling without drooling or stridor

Pertussis (“Whooping Cough”)

General

Caused by Bordetella pertussis, B. parapertussis; vaccine is included in standard childhood immunizations; immunity wanes 6 to 8 years after vaccine or 15 years after infection

Incidence is increasing; the condition is often unrecognized.

Presentation

Catarrhal phase – after 7 to 10 days of incubation, nonspecific upper respiratory symptoms with dry cough, usually beginning in the latter part of the 1- to 2-week duration of this phase

Paroxysmal phase – low-grade fever subsides, cough becomes more prominent, lasts 2 to 4 weeks

Paroxysms: a group of multiple short coughs followed by a single forceful inspiratory “whoop” are characteristic of pertussis

A minority of adults and neonates have this feature, which may be intermittently exhausting for the patient.

Convalescent phase – residual cough for weeks to months

Physical exam is often nonspecific; can include tussive sequelae
Complications

- Secondary bacterial or viral infection may occur.
- Neonates are at risk for bradycardia, hypotension, and apnea.
- Seizures, encephalopathy, and intracerebral hemorrhage occur rarely.

Diagnosis

- Cultures and even direct fluorescent antibody or PCR testing are often negative; these results are too delayed to have much utility in the ED.
- Lab studies are not very useful.
  - Significant lymphocytosis may occur, especially in infants.
  - Chest film is often nonspecific but helps rule out superinfection.

Treatment

- Supportive care: oxygen, suctioning, hydration
- Admit patients with severe symptoms or infectious or CNS complications.
  - Neonates should be admitted for apnea monitoring.
  - Children under 1 year of age, being only partially immunized, are at risk for greater morbidity as well.
- Antibiotics do not shorten the course of illness but do decrease infectivity. Postexposure prophylaxis should be given to all household contacts and any partially immunized contacts with significant exposure to the patient.
  - Erythromycin, azithromycin, and clarithromycin are acceptable choices.
  - Isolation for 3 weeks after onset of paroxysmal phase

Obstruction

- General
  - Many causes; the severity of respiratory distress ranges widely

- Presentation
  - Symptoms may include cough, wheezing, stridor, choking episodes, sore throat, and difficulty with feeding; patient may have retractions or tachypnea.
  - Important historical features include the chronicity, associated symptoms, progression, and any prior episodes or surgical procedures.
  - Stridor may be present with partial upper airway obstruction; its characteristics may help localize the obstruction (Table 4-1).
### Table 4-1. Identification of Airway Obstruction by Characteristics of Stridor

<table>
<thead>
<tr>
<th>Airway Region</th>
<th>Structures</th>
<th>Sound</th>
<th>Congenital Causes</th>
<th>Acquired Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasopharynx</td>
<td>Nose</td>
<td>Expiratory stridor</td>
<td>Micrognathia</td>
<td>Retropharyngeal abscess</td>
</tr>
<tr>
<td></td>
<td>Oropharynx</td>
<td>Sonorous</td>
<td>Treacher-Collins</td>
<td>Adenopathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gurgling</td>
<td>Pierre-Robin</td>
<td>Tonsillar hypertrophy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coarse</td>
<td>Macroglossia</td>
<td>Foreign body</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Muffled voice (&quot;hot potato&quot;)</td>
<td>Chonatal atresia</td>
<td></td>
</tr>
<tr>
<td>Supraglottic</td>
<td>Distal pharynx</td>
<td>Inspiratory stridor</td>
<td>Thyroglossal cyst</td>
<td>Croup</td>
</tr>
<tr>
<td></td>
<td>Epiglottis</td>
<td>High-pitched</td>
<td>Lingual thyroid</td>
<td>Epiglottitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hoarse voice</td>
<td></td>
<td>Foreign body</td>
</tr>
<tr>
<td>Glottic</td>
<td>Larynx</td>
<td>Biphasic stridor (due to</td>
<td>Laryngomalacia</td>
<td>Vocal cord paralysis</td>
</tr>
<tr>
<td></td>
<td>Vocal cords</td>
<td>respiratory changes in size of</td>
<td>Laryngeal web</td>
<td>Papillomas</td>
</tr>
<tr>
<td></td>
<td>Cricoic ring</td>
<td>glottic opening)</td>
<td></td>
<td>Foreign body</td>
</tr>
<tr>
<td>Subglottic</td>
<td>Trachea</td>
<td>Expiratory stridor</td>
<td>Subglottic/tracheal stenosis</td>
<td>Bacterial tracheitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>High-pitched</td>
<td>Tracheomalacia</td>
<td>Croup</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Vascular ring</td>
<td>Subglottic stenosis</td>
</tr>
</tbody>
</table>

### Causes

- Foreign bodies can cause any type of stridor (see Section 4.4, Foreign Body).
  - Most common locations are at the narrowest portion of the airway:
    - Adults: usually at vocal cords
    - Children: usually at cricoid cartilage
- Infections
- Mass/neoplasm
- Angioedema or hypersensitivity reactions
- Congenital abnormality
- Trauma or burns

### Diagnosis and Treatment

- Careful evaluation of respiratory distress and need for airway management
- Further diagnostics, tailored to the patient, may include the following:
  - Bedside fiberoptic nasopharyngoscopy
  - Rigid or fiberoptic bronchoscopy
  - CT, MRI, or esophagram
  - Plain AP/lateral x-ray films
- Specific treatment is based on location and type of obstruction.

### Tracheostomy and Complications

- **Device Anatomy**
  - Surgical opening between cartilaginous rings in trachea, with skin sutured to anterior tracheal wall
    - Cuffed tube used for first week; prevents aspiration
  - Standard 15-mm external connector, compatible with ventilator tubing and bag-valve devices
    - Pediatric sizes, 0.0 to 4.0 mm; adult sizes, 4.0 to 10.0 mm (inner diameter)
  - Obturator (solid) creates smooth tip distally for atraumatic insertion.
  - Inner cannula allows clearing without loss of patency.
• Most Common Complications
  o Accidental decannulation
    ▪ Occurs with coughing or neck extension
    ▪ To replace tube, hyperextend neck and gently reinsert the trach tube.
      ▪ This should be done with the obturator in place.
      ▪ Use endotracheal tube if no tracheostomy tube is available.
  o Obstruction
    ▪ Usually caused by mucus plugging, creating a ball-valve mechanism and air trapping
    ▪ Remove inner cannula (or entire tube if necessary), preoxygenate, then suction.
  o Infection of stomal skin, trachea, or bronchi
    ▪ Broad-spectrum antibiotic: cover for *S. Aureus*, *Pseudomonas*, and *Candida*
  o Bleeding
    ▪ Most common 1 to 3 weeks postoperatively
    ▪ Slow or minor bleeding: remove tube, control with silver nitrate or electrocautery
    ▪ Brisk bleeding: leave airway below bleeding site
  o Tracheo-innominate artery fistula
    ▪ Rare, life threatening
    ▪ Use local digital pressure or hyperinflate cuff and provide gentle tube traction.
    ▪ Immediate surgical subspecialty consult: this may be ENT or thoracic surgery (institution dependent)
  o Tracheal stenosis
    ▪ Late complication, prevented by change to cuffless tube 1 week postoperatively

4.2 DISORDERS OF PLEURA, MEDIASTINUM, AND CHEST WALL

Costochondritis
• General
  o Syndromes of inflammation of costal cartilage or sternal articulations
  o Pain is variably dull, sharp, or pleuritic
• Tietze syndrome
  o Fusiform swelling in one or more upper costal cartilages
• Xiphodynia
  o Sharp, pleuritic pain upon light palpation of xiphoid process
• Texidor’s twinge or precordial catch syndrome
  o Short episodes of lancinating pleuritic chest pain near cardiac apex
  o Individuals become predisposed to this condition by poor posture and inactivity.

Mediastinitis
• General
  o Most commonly related to esophageal perforation
  o Can spread from any head or neck infection (e.g., dental caries)
  o Infection descends through potential spaces
    ▪ Carotid sheath: from base of skull to aortic arch
    ▪ Prevertebral space: *posterior* to prevertebral fascia, down to T3
    ▪ “Danger space” (from skull base to diaphragm): *anterior* to prevertebral fascia, this retropharyngeal space continues down through superior into posterior mediastinum
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• Presentation
  o Commonly occurs in individuals with recent history of URI or dental infection.
  o Fever, chills, pleuritic chest pain, dyspnea, sore throat, neck swelling
  o Ill appearing, neck and face edema or crepitance
  o Consider if patient suddenly has septic or cardiovascular compromise after intubation.

• Diagnosis
  o Clinical suspicion
  o Neck x-ray films may show widening of prevertebral or retropharyngeal soft tissues.
  o Chest film may show diffuse mediastinal widening, pneumomediastinum, air in the soft tissues, mediastinal air-fluid levels, pneumothorax, or hydrothorax.
  o Suspicious x-ray findings should prompt CT scan.

• Treatment
  o Broad-spectrum antibiotics with anaerobic coverage
  o Early surgical consultation

Pleural Effusion

• General
  o Abnormal increase in fluid between visceral and parietal pleurae
  o Two types of effusions: transudates and exudates
    ▪ Transudates (low protein): caused by either an increase in hydrostatic pressure or a decrease in oncotic pressure in pulmonary vasculature
      □ Most commonly (90%) due to congestive heart failure (CHF)
      □ Also associated with pulmonary embolus, superior vena cava (SVC) syndrome, and hypoalbuminemia (cirrhosis, nephrotic syndrome)
      □ Mnemonic: transudates are plasma (both have two a's)
    ▪ Exudates (high protein): caused by defective lymphatic drainage or increased pulmonary capillary permeability
      □ Primary cause is parapneumonic; second most common cause is malignancy
      □ Other inflammatory causes include connective tissue disease, abdominal processes and infections, and uremia.
      □ Specific terms relating to exudative effusion:
        — Parapneumonic effusions – in association with pneumonia, bronchiectasis, or lung abscess
        — Loculated effusions – when inter-pleural adhesions prevent free flow

• Presentation
  o History points more to the underlying process than the effusion itself.
  o Physical exam will usually reveal findings consistent with underlying disease process, but may include decreased breath sounds, dullness to percussion, pleural rub, or decreased tactile fremitus.
  o Pulmonary embolism is the most common cause of pleural effusion and pleuritic chest pain in patients under 40 years old; dyspnea may be out of proportion to the size of the pleural effusion.

• Diagnosis
  o Chest film confirms the finding; it may be incidental to another diagnosis.
    ▪ Fluid may be visible in fissures or may be seen layering with a meniscus; if free-flowing, layering will vary with patient position
    ▪ Upright: blunting of costophrenic angle (with >250 mL of fluid)
Lateral: smaller effusions visible in posterior costophrenic gutter
- Supine: may reveal only diffuse haziness with larger effusions; apical capping may also be seen
- Lateral decubitus (in slight Trendelenburg): may reveal as little as 5 to 15 mL of fluid

- Ultrasound and CT of the thorax may be useful when plain films are equivocal or if the underlying process requires further imaging.
- Diagnostic thoracentesis permits pleural fluid analysis to define effusion using Light’s criteria or the serum-effusion albumin gradient (see below).
- Light’s criteria (98% sensitive)
  - Fluid is an exudate if
    - the pleural:serum protein ratio is >0.5 OR
    - the pleural:serum LDH ratio is >0.6 OR
    - the pleural LDH is >2/3 the upper limit of a normal serum LDH
- Transudate
  - Light’s criteria are less specific than sensitive.
  - If the (serum – effusion) albumin gradient >1.2 g/dL, a transudate is probable; therefore, Light’s criteria can be disregarded.
  - The utility of further serum tests varies by etiology.
- Exudate: further characterized by additional pleural fluid analysis:
  - Bacterial gram stain; also culture for bacteria, fungi, mycobacteria
  - pH (may reflect systemic acidosis)
    - <7.3: parapneumonic, malignant, rheumatoid, tuberculous
    - <7.0: empyema, esophageal rupture
  - Cell count with differential (normal <1000 WBC/mm³)
    - Neutrophilia: acute processes such as pneumonia, pulmonary embolus, tuberculous pleuritis
    - Mono/Lymphocytosis: more chronic malignancy, TB
  - Hematocrit (for bloody fluid)
    - Traumatic taps, trauma, neoplasm, and infarcts cause bloody fluid
    - If the hematocrit of the pleural fluid is >50% of the peripheral blood hematocrit, the effusion is a hemothorax.
  - Amylase: high in pancreatic disease, esophageal rupture, 10% of malignancies
  - Parapneumonic effusions: LDH high (>1000), glucose low (<40)
  - Cytology may provide diagnosis of cancer in malignant effusion.

- Treatment
  - ED intervention is based on etiology of effusion and patient condition.
    - Transudates require treatment directed at underlying etiology.
    - Exudates need further diagnostic work-up, usually as inpatients.
  - Thoracentesis is done in the ED when necessary to diagnose rapidly fatal conditions or to stabilize the patient’s cardiovascular or respiratory status; otherwise, this procedure is often deferred to the inpatient phase of care.
    - Serous effusions: 10 to 12 French thoracentesis catheter
    - Purulent/bloody collections: 24 to 28 French chest tube
    - In CHF: if very asymmetric or with a fever or pleuritic pain
    - Parapneumonic effusion: diagnostic, as up to 10% are empyema
• Empyema: chest tube for drainage and prevention of loculation
  □ Lociations may require local streptokinase or urokinase.
• Hemothorax: chest tube for source tamponade by lung reexpansion; also quantifies hemorrhage
  □ Thoracotomy considered if >200 mL/hr
• Symptomatic patients with a known diagnosis of recurrent malignant effusion may not require admission after palliative therapeutic thoracentesis; they may benefit from pleurodesis or a pleuroperitoneal shunt to prevent recurrence.
  o Transient hypoxia may occur after drainage due to V/Q mismatch, but unless large volumes are removed, reexpansion pulmonary edema is rare, as is hypotension in a patient who is not already volume depleted.
  o Empiric broad-spectrum antibiotics (if a bacterial cause is suspected)
    □ Clindamycin and third-generation cephalosporin, e.g., ceftriaxone
  o Consider intrapleural thrombolytics for loculated effusions

Pleuritis (“Pleurisy”)
• General
  o Defined as inflammation of the pleura
  o Symptom with a wide variety of causes – viral disease, pneumonia, trauma to parietal pleura, pulmonary embolism, chronic inflammatory disease
  o May have associated exudative effusion, which can lead to adhesions
• Presentation
  o Symptoms are related to the underlying process; may include “stabbing” chest pain that is often “pleuritic,” i.e., worse on inspiration or coughing
  o Viral pleuritis is preceded by viral prodrome: fever, sore throat, and other mild constitutional or upper respiratory symptoms.
  o Pleural friction rub may be present on physical exam, though this is not a sensitive finding.
• Treatment
  o Analgesia; treat any underlying disease; encourage good pulmonary toilet

Pneumomediastinum
• General
  o Gas in mediastinal tissues
• Types
  o Spontaneous
    □ Can occur in association with asthma
    □ Can be associated with exertion, Valsalva, Heimlich, seizure, childbirth, inhaled drugs, intubation, endoscopy
    □ Benign clinical course, resolves spontaneously
  o Secondary
    □ Esophageal perforation or rupture, as in Boerhaave’s syndrome
    □ Trauma (blunt or penetrating)
    □ As a result of pulmonary barotrauma
• Presentation
  o Chest pain, dyspnea, throat/neck pain, dysphonia, dysphagia, physical exam reveals subcutaneous air in neck and face, often with crepitance
    □ A crunch may be heard with each heartbeat (“Hamman’s sign”)
• Complications
  o Infection, pneumothorax, tension pneumothorax
  o The presence of mediastinal air, in the absence of cardiopulmonary compromise, is not life threatening in itself but mandates a search for the cause.

• Diagnosis
  o Suspect and test based on history and physical
  o Chest film (see Image #57)
    ▪ Mediastinal air, in vertical radiolucent lines at heart border or aorta
    ▪ Subcutaneous emphysema
    ▪ Associated pneumothorax or other injuries or clues to the cause
  o Other studies may be needed to evaluate the patient, including Gastrografin or barium swallow, CT scan, endoscopy

• Treatment
  o Supportive, while searching for and managing coexisting conditions
  o Airway management, other interventions for acute disease (e.g., pneumothorax) where indicated

Pneumothorax (see also Chapter 1, Traumatic Disorders)

• General
  o Air between visceral and parietal pleura, enlarging this potential space
    ▪ Caused by a defect between intrapleural space (normally at negative pressure) and proximal or distal airways
    ▪ Communication between intrapleural space and a space with positive pressure causes air to accumulate until the pressures equalize or the defect is closed.

• Simple
  o Pneumothorax causes collapse of the ipsilateral lung, with resultant decrease in lung capacities; shunting with VQ mismatch may cause hypoxemia.

• Tension
  o Alveolar-pleural defect, acting as a one-way valve, leads to progressive accumulation of air in the intrapleural space with each inhalation.
  o Increases intrapleural pressure and shifts the mediastinal contents away from the involved side, eventually compressing the contralateral lung, causing hypoxia
  o High intrapleural pressures and mediastinal shift can significantly impair venous return, leading to cardiovascular collapse and death.

• Causes
  o Traumatic – due to blunt or penetrating trauma
  o Iatrogenic – usually also due to penetrating trauma, e.g., a procedure
  o Spontaneous – without a precipitant: two thirds are primary, one third are secondary; usually a subpleural bulla ("bleb") is responsible; pneumothorax may be more likely, with risk factors weakening the wall of this structure
    ▪ Primary (no lung disease) – more common with risk factors:
      □ Male, smoker, tall, change in ambient pressure, Marfan's syndrome
    ▪ Secondary (history of lung disease, usually COPD)
      □ Other risks: Pneumocystis jiroveci pneumonia (formerly Pneumocystis carinii pneumonia [PCP]), malignancy, asthma, cystic fibrosis, lung abscess, TB, interstitial lung disease
• Presentation
  o Sudden onset of unilateral chest pain (may be pleuritic or dull), dyspnea, cough; presentation may be delayed by days, and symptoms may resolve even prior to complete resorption of the intrapleural air
  o Sinus tachycardia is common; diminished breath sounds with hyperresonance to percussion or asymmetric chest excursion may be seen; absence of these features does not rule out the diagnosis
  o Tension pneumothorax causes tachycardia, hypoxia, jugular venous distension, hypotension (late), and then tracheal deviation to the contralateral side (a rare, preterminal finding)
  o Patients with underlying lung disease present differently
    ▪ With lower reserve, they are more likely to be symptomatic.
    ▪ Lung exam at baseline may have features that overlap with those typical of pneumothorax.

• Diagnosis
  o Clinical suspicion of the diagnosis should prompt imaging.
  o Chest film classically reveals a thin pleural line, parallel to the contour of the chest wall, separating tissue with apparent lung markings from a peripherally radiolucent stripe. (see Image #3 for an example of a hemo/pneumothorax)
    ▪ Size is graded as small, moderate, large, or total
    ▪ Tension pneumothorax should be diagnosed and treated clinically, prior to imaging. It would appear as a total pneumothorax with tracheal and mediastinal deviation to the opposite side.
  o Other causes of dyspnea or chest pain, such as pulmonary embolism, may present similarly, but with no pneumothorax on plain film.

• Treatment
  o Depends on clinical status of patient, but generally is treated by tube thoracostomy, which brings the visceral and parietal pleurae back together by removing the intrapleural air keeping them apart.
    ▪ Other options that may be appropriate include observation, catheter aspiration, video-assisted thorascopic surgery, even thoracotomy.
    ▪ Considerations include size of pneumothorax and air leak, severity of symptoms, underlying comorbidities or pulmonary disease, recurrent pneumothorax, and reliability for follow up.
  o Observation – if small (<20%) spontaneous pneumothorax in a young and healthy patient.
    ▪ Intrinsic reabsorption occurs at 1% to 2% per day; increases to 4% to 8% a day with use of 100% oxygen
    ▪ If patient is reliable, at least 6 hours of observation and a repeat chest film help ensure that the pneumothorax has not increased in size prior to discharge; good access to EMS and prompt 24-hour follow up are essential for these patients.
    ▪ Air travel and underwater diving must be avoided until complete resolution occurs.
  o Simple catheter aspiration – may be used in young and healthy patients with a first episode of a moderate (>20%) pneumothorax
    ▪ Less invasive but not always successful; requires reevaluation prior to discharge to rule out recurrence or worsening
  o Tube thoracostomy – widely used, either primarily or when more conservative management has failed; the treatment of choice when a pleural effusion is present, patient is in respiratory distress, has tension pneumothorax, or may require positive-pressure ventilation
    ▪ Small (7–14 French) tubes for primary spontaneous pneumothorax
      □ Better tolerated but have a higher risk for malfunction
    ▪ Standard (20–28 French) for secondary spontaneous pneumothorax
    ▪ Large (>28 French) for detectable pleural fluid or anticipated positive-pressure ventilation
Tube is then connected to a water-seal device (or one-way “Heimlich” or “flutter” valve)
- Suction does not generally improve the rate of resolution.
- Hospital admission is almost always required.

Sufficient suspicion of tension pneumothorax is an indication for immediate decompression of the intrapleural space.
- Tube thoracostomy is still the definitive treatment.
- Needle decompression is temporizing but usually more rapidly available and can be performed while preparing the chest tube.
- A hiss of air escaping confirms the diagnosis with the placement of either a needle or chest tube.

Complications of tube thoracostomy
- Malposition, infection, prolonged pain, obstruction, and, rarely, reexpansion pulmonary edema or hypotension following rapid removal of large volumes of intrapleural air
- Referral for prevention of recurrence of secondary spontaneous pneumothorax or recurrent primary spontaneous pneumothorax, as well as for enthusiasts of flying, diving, and other high-risk activities
- Pleurodesis and resection of bullae are two strategies.

4.3 NONCARDIOGENIC PULMONARY EDEMA

General
- Also known as acute (adult) respiratory distress syndrome
- Caused by increased permeability of the alveolar-capillary membrane
  - Not from increased hydrostatic pressures, as in cardiogenic form
- Interstitial edema \(\rightarrow\) shunting and reduced lung compliance
- Inflammatory process \(\rightarrow\) microvascular membrane damage \(\rightarrow\) further increases in permeability

Presentation
- Seen 2 to 24 hours after a variety of insults:
  - Systemic sepsis/systemic inflammatory response syndrome/septic shock (most common)
  - Aspiration (second most common) or near-drowning
  - Trauma, multiple transfusions, or fat emboli syndrome
  - Inhalation injuries (toxins, smoke)
  - Drugs (tricyclic antidepressants, narcotics, salicylates, sedative-hypnotics)
  - Neurogenic causes
  - High-altitude pulmonary edema
- Symptoms include tachypnea, dyspnea, hypoxia
- Fine scattered rales on physical exam

Diagnosis and Treatment
- Suspect in appropriate scenario and hypoxemia refractory to increased FiO\(_2\)
- Defined as:
  - \(\text{PaO}_2/\text{FiO}_2\) ratio <200
    - Often requiring mechanical ventilation
  - Chest film: bilateral alveolar infiltrates, but normal cardiac silhouette
  - No clinical evidence of CHF, fluid overload, or chronic lung disease
- Supportive but aggressive treatment:
  - Intubation and mechanical ventilation may be required.
Consider PEEP to improve alveolar recruitment.
- Keep peak airway pressures <35 cm H₂O
- Reduced tidal volumes (5–7 cc/kg) with permissive hypercapnia improve outcome.
- Pulmonary artery pressure monitoring can be useful for fluid management.
- Steroids are not helpful; they may increase the risk of later infection.

### 4.4 OBSTRUCTIVE AND RESTRICTIVE LUNG DISEASE

#### Asthma/Reactive Airway Disease

**General**
- Chronic inflammatory disease of airways resulting in episodic, reversible airway obstruction
  - Mediated by inflammatory cascade with numerous effects
    - Bronchial hyperresponsiveness (hyperreactivity) to triggers
    - Vascular congestion, bronchial wall edema, thick secretions
  - Early response (may resolve within 60 minutes)
    - Release of preformed histamine leads to bronchial smooth muscle constriction, airway edema, wheezing, airflow obstruction
  - Late response (4–6 hours after exposure to trigger)
    - Caused by cytokines (prostaglandins and leukotrienes): prolonged bronchospasm

- Increased risk for death from asthma occurs with the following:
  - History of sudden severe exacerbations
  - Prior intubation or ICU admission for asthma
  - Two or more admissions for asthma in the past year
  - Three or more ED visits for asthma in the past year
  - Admission or ED visit for asthma in the past month
  - Use of more than two short-acting β₂-agonist MDI canisters per month
  - Current use of systemic corticosteroids or recent withdrawal
  - Comorbidities (cardiovascular disease, serious psychiatric disease)
  - Psychosocial problems, illicit drug use (inhaled cocaine or heroin)

**Subtypes**
- Pediatric asthma is discussed in Chapter 5, Pediatrics.
- Aspirin-exacerbated respiratory disease
  - Induced by aspirin or NSAIDs, via change in balance of mediators
  - Anti-leukotriene drugs may be of additional use in these patients.
- Menstruation-associated asthma
  - One third or more of premenstrual asthmatic women have decreased peak expiratory flow rates and may have more frequent exacerbations.

**Presentation**

**History**
- Onset, triggers, severity, comorbidities, medications, and exacerbating drugs (ASA, NSAIDs, β-blockers, ACE inhibitors)
- Known asthmatics should be questioned about their asthma history.
  - Risk factors for death (see above)
  - Comparison of episode with past episode severity
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- Symptoms and physical findings are quite variable:
  - Cough (may be the only symptom; can be productive), wheezing, dyspnea, and chest tightness; nocturnal worsening is common
    - Intensity of wheezing varies with the velocity and turbulence of air flow. With extremely poor air movement, wheezing may be inaudible: a "silent chest."
  - Hyperresonance to percussion, decreased breath sounds, wheezing or prolonged expiratory phase
  - Accessory muscle use, retractions, paradoxic respirations
  - Abnormal vital signs support the diagnosis of severe asthma, but normal values do not rule it out:
    - Respiratory rate correlates poorly unless >40 breaths/min
    - Tachycardia, even >120 beats/min, may be present, but not always
    - Pulsus paradoxus (inspiratory fall in systolic BP) of >10 mm Hg is another specific, insensitive finding
  - Lethargy, exhaustion, or confusion heralds impending respiratory failure; cyanosis is uncommon due to respiratory alkalosis, which causes left-shift of the oxyhemoglobin dissociation curve.
  - Restlessness and agitation are common but unreliable indicators of hypoxia or hypercapnia.

• Diagnosis
  - Severity is most reliably assessed with objective measurements.
  - Forced expiratory volume in 1 second (FEV1) or peak expiratory flow rate should be measured and evaluated for progress, ideally against the patient's "personal best" or, alternatively, as a percentage of that predicted by age (<85 yr), sex, and height.
  - Other testing
    - Arterial blood gas rarely adds to proper decision making:
      - Hypoxia is unusual and ruled out by pulse oximetry.
      - Can be useful to assess for hypoventilation (as shown by PaCO₂ >42 mm Hg) or respiratory alkalosis.
      - Side-stream capnography appears useful.
    - Due to inflammation or demargination, the WBC count is often elevated; only values above 20,000/mm³ should increase concern for infection.
    - B-type natriuretic peptide may help evaluate the contribution of CHF in older patients.
    - Measure theophylline levels in patients taking it.
    - Chest films are usually not required, but they can be used to evaluate the patient for complications or coexisting disease (pneumothorax, pneumonia).

• Treatment
  - Inhaled β-agonists (albuterol or levalbuterol)
    - Mainstay of treatment for most exacerbations
  - Subcutaneous β-agonists (epinephrine or terbutaline)
    - For critical patients who are unable to inhale sufficient dosing of medication; more prominent side effect profile
  - Long-acting β-agonists (LABA) can be started but they have little effect in the ED.
  - Intravenous epinephrine in severe, life-threatening asthma (2–10 mL of 1:10,000 solution given IV over 5 minutes followed by an epinephrine drip if the patient responds)
  - Corticosteroids should be given early in a moderate or severe acute asthma episode and to patients with incomplete response to initial treatment, those taking oral or inhaled corticosteroids, and those who have relapsed from a recent exacerbation.
Onset of effect in hours; IV and PO are equivalent routes
  - Oral course for 3 to 10 days
- Inhaled anticholinergics (ipratropium bromide, tiotropium) block secretions and bronchoconstriction; use with initial doses of inhaled β-agonists
- Maximal effect at 30 to 120 minutes
- Magnesium sulfate IV – may be helpful; mixed data; optimal dose is unclear
  - 2 to 3 g IV over 20 minutes or as fast as 1 gm/min if needed while inhaled treatments continue
- Theophylline/aminophylline – narrow therapeutic window, large side effect profile, many hepatic enzyme drug interactions; patients may be taking this chronically, but its use in the ED is not recommended
- Treatments with insufficient evidence: leukotriene modifiers (zafirlukast, montelukast), heliox, ketamine, continuous or bilevel positive airway pressure; none is currently standard for asthma therapy in the ED
- Intubation is occasionally required for impending or actual respiratory failure in severe asthma.
  - Continue corticosteroids, β-agonists, anticholinergics
  - Permissive hypercapnia (tolerating a higher PaCO₂)
    - Permits oxygenation and ventilation; but minimizes breath stacking, barotrauma, and hypotension
    - High FiO₂, tidal volumes 6 to 8 mL/kg, lower ventilation rates, and high inspiratory flow rate
    - Sodium bicarbonate IV to keep pH >7.2
  - Ketamine or inhaled anesthetics (halothane, isoflurane) may be useful as bronchodilators in addition to the usual maximal treatment.

Bronchitis
- General
  - Infection of conducting airways of lung, with inflammation, exudates, sometimes bronchospasm; majority are caused by viruses
  - Chronic bronchitis – defined as the presence of a productive cough for 3 months out of each of 2 consecutive years; it may occur without airflow limitation; in those whom airflow obstruction occurs, considered COPD
- Presentation
  - Cough is the hallmark: usually productive, but patients lack evidence of pneumonia, sinusitis, or chronic pulmonary disease; color of sputum is not predictive of bacterial infection
- Diagnosis
  - Acute cough less than 1 week, no prior lung disease, normal oxygenation, no auscultatory abnormalities; chest film, cultures, CBC or other blood testing is unnecessary
  - In absence of fever, tachycardia, tachypnea, or lung exam findings: radiographic infiltrate in <1%
- Treatment
  - Supportive treatment for this self-limited disease; no benefit to antibiotics
    - Bronchodilators may be useful.
  - Patients with COPD and older adults may benefit from antibiotics.

Bronchiolitis
- General
  - Most common lower respiratory infection in children
  - Inflammation leads to mucus, edema, and sloughed debris causing distal airway obstruction
  - Children ≤2 years old are most commonly symptomatic (peak is 2–6 months); seasonal disease is prominent in colder months
    - Risk factors: prematurity, daycare, secondhand smoke
o Majority caused by respiratory syncytial virus (RSV), which can also cause pneumonia
  ▪ Parainfluenza is the second most common cause (fall and spring)
  ▪ Also adenovirus, rhinovirus, influenza, *Mycoplasma*

• Presentation
  o Prodrome of 3 to 5 days of nasal congestion and cough is common.
    ▪ May be fussy, not eating well, febrile
  o Respiratory distress with subcostal retractions, tachypnea, tachycardia
    ▪ Wheezing and coarse breath sounds are commonly heard.
    ▪ Generally alert and playful; lethargy and irritability should raise concern for hypoxia, hypercarbia, or a more serious disease
    ▪ Severe distress: grunting, nasal flaring, suprasternal retractions

• Complications
  o Viral pneumonia or bacterial otitis media may coexist.
  o The vast majority of healthy infants do well.
  o Complication rates are higher in patients with congenital heart disease, preexisting pulmonary disease, prematurity, or immunodeficiency.

• Diagnosis
  o Clinical diagnosis with serial exams is sufficient in most cases
    ▪ Recognize early respiratory failure, risk factors for apnea
    ▪ Pulse oximetry <95% predicts severe bronchiolitis
  o Chest film is useful if the patient is deteriorating or has prolonged or severe disease.
    ▪ Hyperinflation; increased markings; infiltrate and atelectasis may be difficult to distinguish; study may also be normal.
  o Laboratory: CBC not useful; nasopharyngeal RSV test, while not routine, can be useful for placing the patient in appropriate isolation if patient is to be admitted

• Treatment
  o Inhaled β-agonists (albuterol or epinephrine): routine use is controversial; a trial of therapy may be done, and if the patient improves, it may be continued
    ▪ If discharged, prescribe nebulized albuterol (not epinephrine) for responders only.
  o Steroids are controversial; they may be helpful in severe disease requiring intubation.
  o Antibiotics are unnecessary.
  o Nasopharyngeal suctioning for patient comfort and ease of breathing
  o Patients at high risk for severe disease or apnea (premature [<34 weeks], underlying cardiopulmonary disease) warrant admission, as do patients who will not feed adequately.
    ▪ Patients with SaO₂ <95% or tachypnea >70 breaths/min or who are <3 months old are also at higher risk.
  o Close follow-up is important; parents should be informed of signs of worsening distress and resulting need for immediate return to the ED.

**Bronchopulmonary Dysplasia (BPD)**

• General
  o Common in premature and low-birth-weight infants; also known as “chronic lung disease of infancy”; its severity is variable
  o Immature lungs have high resistance and low compliance and may have bronchospasm in response to pneumonia.
Patients with severe disease may be on long-term diuretics.

- Cor pulmonale is a frequent complication for neonates with oxygen requirements.
  - Must differentiate from pneumonia

- Immunizations are essential to decreasing morbidity from pneumonia: not only against influenza and pneumococcus, also monthly RSV vaccinations

### Chronic Obstructive Pulmonary Disease

- **General**
  - Worldwide and in the United States, fourth leading cause of death
  - Airflow limitation that is not fully reversible
    - Has associated inflammatory response causing progressive disease
  - Spectrum of disease with components of chronic bronchitis, emphysema, bronchiectasis
    - Dyspnea on exertion, chronic productive cough, hemoptysis
  - Usually due to smoking; minority caused by \( \alpha_1 \)-antitrypsin deficiency

- **Presentation**
  - Patients usually have components from both clusters of the disease spectrum:
    - Bronchitic: prominent cough, coarse crackles from uncleared secretions, chronic respiratory failure (\( CO_2 \) retention and hypoxia), plethora and cyanosis, chronic jugular venous distention (JVD), cor pulmonale
    - Emphysematous: anxiety, dyspnea, tachypnea, using pursed-lips to prolong exhalation (auto-PEEP), hyperinflated thorax, impaired diaphragmatic motion, diminished breath sounds throughout, hyperresonance to percussion, faint end-expiratory rhonchi
  - Acute exacerbation = increased dyspnea, often accompanied by an increase in sputum volume or purulence
    - Commonly triggered by viral infection or other irritants
  - Fatal progression of severe exacerbation: increasing work of breathing, increasing muscle \( CO_2 \) production to become greater than that liberated by the increase in minute ventilation, overall \( CO_2 \) retention and respiratory acidosis, hypercapnea, confusion, stupor, hypopnea, apnea

- **Diagnosis**
  - Chief complaint has a broad differential of cardiac, pulmonary diseases
  - Evaluate for evident bacterial infection; consider possible comorbidities
    - Pneumonia, pneumothorax, pulmonary embolism
    - Right- and/or left-sided CHF, visceral congestion
      - Most common disease to be misdiagnosed as COPD is cardiogenic pulmonary edema, which presents with dyspnea and wheezing (a.k.a. cardiac asthma)
  - Pulse oximetry: detect life-threatening hypoxemia
    - Assess trends in degree of hypoxemia, home oxygen dependence
  - Arterial blood gases (ABG) are of little use, particularly when baseline ABG is unavailable.
    - Consider if concern for hypercapnia or respiratory acidosis
    - In severe exacerbations, can help show severity of respiratory failure, lack of response to therapy in consideration of ICU admission
  - Chest film: primarily to identify any other cause of the patient’s symptoms, which may be treatable in parallel with the COPD exacerbation:
    - Pneumothorax, consolidation (pneumonia, or postobstructive atelectasis due to tumor or mucous plugging), CHF, effusions
    - Chronic findings: hyperinflated lungs, flattened diaphragms, decreased vascular markings OR normal inflation, increased vascular markings, enlarged heart. Bullae may mimic pneumothorax.
Pulmonary function tests are of limited use (less reversible obstruction than in asthma).
- Patients tend to respond to treatment in the reversible component only.
- Sputum culture is useful only if particular agents are suspected.
  - Diagnostic of *Legionella*, TB, PCP, fungus
- ECG
  - Continuous monitoring for dysrhythmias and response to therapy
  - Criteria for right ventricular hypertrophy (RVH) suggest cor pulmonale (not sensitive or specific)
- Theophylline level, in patients who take it as outpatients
- Blood testing
  - Nonspecific elevation in WBC count is seen
  - B-type natriuretic peptide (BNP) may be useful: rules out CHF with high sensitivity (but less specificity)
    - In cor pulmonale, RV strain is another cause of increased BNP.

### Treatment
- Oxygen
  - Its use remains controversial, but the risks of hypoxemia must be considered alongside those of reducing ventilation by removing the hypoxic drive.
  - However, do not allow severe hypoxemia to persist; consider a low, titrated dose of oxygen if hypoxemia exists.
    - Myocardial and tissue ischemia with metabolic acidosis and muscular fatigue will also be detrimental to the patient.
  - It may be reasonable to provide oxygen sufficient to keep $\text{PaO}_2 > 60$ mm Hg or $\text{SaO}_2 > 90\%$.
    - Monitor for any inappropriate drop in respiratory rate
- Bronchodilators: inhaled $\beta$-agonist with an anticholinergic given together, simultaneously (e.g., albuterol with ipratropium)
  - First-line therapy (though bronchospasm is not the cause of exacerbation)
  - $\text{SaO}_2$ may initially drop (transient worsening in V/Q mismatch)
  - Subcutaneous terbutaline or epinephrine – with great caution only
- Corticosteroids: short (1- to 2-week) course; degree of response varies among individuals
- Antibiotics: consider in exacerbation, especially if evidence of infection is present (fever, change in mucus production, abnormal radiograph)
  - Macrolide, third-generation cephalosporin, TMP-SMX, or "respiratory" fluoroquinolone (moxifloxacin, gatifloxacin, levofloxacin)
- Theophylline: use in ED is, at best, controversial
- Assisted ventilation: decisions per clinical judgment
  - Invasive (endotracheal) mechanical ventilation may be required for patients whose severe exacerbations are not responsive to initial treatment but who are not candidates for noninvasive positive-pressure ventilation (see below).
    - Permissive hypercapnia with gradual correction of $\text{PaCO}_2$
  - Noninvasive positive-pressure ventilation can help avoid intubation and assists ventilation in cooperative patients with moderate to severe respiratory failure or respiratory muscle fatigue, with hypercarbia or some degree of hypoxemia; contraindicated by abnormal mental status, hemodynamic instability, inappropriate anatomy, aspiration risk, or imminent respiratory arrest; monitor for deterioration with need for invasive ventilation
Continuous positive airway pressure (CPAP) at 5 to 10 cm H₂O
- Bilevel positive airway pressure
  - Inspiratory positive airway pressure at 8 to 10 cm H₂O
  - Expiratory positive airway pressure at 2 to 4 cm H₂O
- Beware: auto-PEEP, hypotension, barotrauma, respiratory alkalosis

Cystic Fibrosis
- General
  - Genetic defect leading to abnormal chloride conductance, poor ciliary function, thickened mucus, chronic inflammatory state, and predisposition toward bacterial infection of the respiratory tract
- Features
  - Findings may include wheezing, productive cough, purulent sputum, dyspnea, nasal polyps, chronic sinusitis, hemoptysis, pneumothorax
  - Chest film: diffusely increased markings with peribronchial thickening, bronchiectasis, emphysema, and, frequently, infiltrates
  - Prone to repeated episodes of pneumonia, particularly *Pseudomonas aeruginosa* or *S. aureus*; antibiotic coverage should include those organisms
    - *Burkholderia cepacia* is another important pathogen.
    - Also at high risk for nonbacterial pneumonia
    - Respiratory isolation from other high-risk patients to prevent new colonization or spread of infection
    - Excellent pulmonary toilet is crucial
      - Acute treatment with bronchodilators and inhaled N-acetylcysteine (as mucolytic) is often helpful.

Environmental/Industrial Exposure
- General
  - Silicosis: common and deadly; until recently the most serious occupational disease; seen in those working in sandblasting, mining, tunneling, foundries, gun-flint industry, sandstone, granite, pottery, metal grinding, and manufacture of abrasive soaps
  - Asbestosis: lung cancer, mesothelioma
  - Farmer's lung: first phase is transient IgE-mediated syndrome with bronchoconstrictive wheezing; second phase is IgG-mediated vasculitis, which can be acute, subacute, or chronic

Foreign Body
- General
  - Children more than adults; adults more likely to aspirate non-food items
    - 75% of aspirations are in children <9 years old (with a peak at 2 years)
    - In adults, peak incidence is in the sixth decade.
  - 3,000 deaths from asphyxiation due to foreign bodies annually in the United States
    - Morbidity from anoxic brain injury
  - Wide variety of items, with a few leading objects:
    - In children, primary culprit = coin (followed by food, especially peanuts and popcorn)
    - In adults: fishbones, dentures, meat, meat bones
    - Aspiration of nuts, hot dogs, candy, or grapes is commonly fatal.
    - Some people often hold pins, needles, or nails in their mouths and thus risk aspiration of those objects.
    - Other items found include jewelry and small toys.
Mechanisms include ingestion, aspiration, and penetrating trauma.

Acutely altered mental status or baseline neurologic deficit increases risk.

See Chapter 7 (Head, Ear, Eye, Nose, and Throat Disorders) for more information on foreign bodies.

Presentation

Variable: chronic nonspecific complaints to acute hypoxic obstruction

- Usually suspected on the basis of a thorough and accurate history

Delayed presentation is common; half may come in after days to weeks

- Patient may not recall recent trauma or blast injury as pertinent.
- Dislodged teeth can be aspirated, as can dental hardware.

Suspect in all children with choking, stridor, wheezing, or new cough

- Esophageal foreign bodies can cause stridor and even partial tracheal obstruction due to the soft posterior tracheal wall in children.

Consider with chronic cough or wheezing that fails to respond to medical management, especially in a child with a history of reactive airway disease.

Often caused by swallowing a food bolus exceeding the esophageal diameter, which then lodges in the hypopharynx or trachea.

- The “penetration syndrome”—a choking sensation with coughing, wheezing, dyspnea—occurs in half of aspirations.
- Patients may have noisy breathing, inspiratory stridor, vomiting, even some hemoptysis.
- Coughing may actually impact the item proximally in the subglottic area; partial or complete posttussive symptom resolution does not exclude the possibility of foreign body.
  - Dyspnea and odynophagia are typical at this level.

Alternately, the delayed presentation may be that of an infection:

- Sequelae include retropharyngeal abscess, recurrent pneumonias
- Or a foreign body presentation may mimic croup, epiglottitis

Physical exam may reveal coughing or audible wheezing with a degree of respiratory distress, even cyanosis; may have stridor, hoarseness, or fever

- Oropharyngeal exam may help visualize a proximal foreign body; look for fractured or missing teeth and dental prostheses.
- Indirect or direct laryngoscopy or nasopharyngoscopy may be performed if it will not cause further airway compromise.
  - Definitive airway management tools should be bedside.
- Palpation of the trachea may elicit a “thud” by pushing the item against the wall. Tracheal auscultation may reveal abnormal sounds.
- Breath sounds may be absent on one side; wheezing may be localized or seem generalized; air trapping (due to one-way-valve function of the object) may cause hyperresonance.

Diagnosis

- Utilize the history to obtain clues as to the nature and location of the object.
  - With a good history, maintain suspicion even if the exam is normal.
  - Determine radiopacity (most swallowed objects are not radiopaque)
- Assess stability of the patient and the safety of additional studies.
  - For example, is the patient able to manage the airway while in radiology?
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- X-ray films
  - PA/lateral films of chest (vs. bilateral decubitus films in infants) and neck
  - May help locate the foreign body; but with a highly suspicious history, they cannot rule one out
  - Direct identification is rare. Most objects are detected indirectly:
    - Narrowing of subglottic space from embedded object
    - Prevertebral swelling, soft tissue air
    - Most common abnormalities are changes in inspiration/expiration
      - Early – asymmetric inflation with air trapping: ipsilateral flat diaphragm, mediastinal shift away from obstructing object with expiration (if complete, leads to atelectasis)
      - Late – bronchiectasis, bronchial stenosis
  - In the trachea, a coin faces laterally and is seen lying sideways on a frontal chest x-ray film; in the esophagus, a coin is coronal and circular on a frontal chest film (see Image #4 for an example of an esophageal coin)
    - SAFE: Sideways=Airway, Frontal=Esophagus
- CT, MRI, fluoroscopy may be useful
  - CT can show radiolucent objects with subtle density differences.
  - MRI is particularly useful for nuts because of their high fat content.
  - Fluoroscopy use has declined for this application.
- Bronchoscopy and microlaryngoscopy are definitive.

- Treatment
  - Immediate airway management as indicated. Options include the following:
    - Promote expulsion via cough or other techniques:
      - Stable patients who are oxygenating adequately and are not in respiratory distress may be initially observed in the position of comfort as they attempt to cough up the item.
        - Observe closely for onset of complete obstruction: ineffective cough, cyanosis, poor air movement
      - Back blows and chest thrusts (infants)
      - “Heimlich” abdominal thrusts (children >1 year and adults)
      - No blind finger sweep (may convert partial to complete obstruction or push the object distally)
    - Laryngoscopy with forceps retrieval under direct visualization
    - Definitive airway control via intubation may follow
      - If the object prevents intubation, a surgical airway should be created if ventilation proves to be impossible as well.
      - Intubation (especially if into mainstem bronchus) may push the obstructing object distally. This does allow oxygenation of at least the opposite lung once the tube is withdrawn.
  - Once the object is removed, recovery is usually rapid.
    - Beyond the oropharynx, a foreign body is generally removed most safely and quickly by a subspecialist.
      - Risks include bleeding, trauma, and the inadvertent dropping or distal displacement of the object
      - Caution if impaled – hemorrhage may follow removal
      - Early bronchoscopy, when indicated, decreases morbidity
        - Fiberoptic is diagnostic; rigid is also therapeutic.
        - Thoracotomy may be required if bronchoscopy fails.
  - Patients should be observed for sequelae after removal, and the patient or caretaker should be counseled on prevention of future episodes, as appropriate.
4.5 PHYSICAL AND CHEMICAL IRRITANTS/INSULTS

Pneumoconiosis
• Progressive pulmonary fibrosis and dyspnea secondary to occupational dust inhalation; treatment is based on symptoms.
• See also Environmental/Industrial Exposure (above)

Toxic Effects of Gases, Fumes, Vapors (see also 12.1, Inhaled Toxins)
• Types of Inhalants
  o Asphyxiants
    ▪ Any gas can cause simple asphyxia if it replaces enough oxygen.
      □ Simple asphyxiants are usually inert; may not cause symptoms unless they cause $FiO_2$ to decrease below 21%.
    ▪ Symptoms are those of hypoxia:
      □ $FiO_2$ 15% – autonomic nervous system stimulation (tachycardia, tachypnea, dyspnea) and cerebral hypoxia (ataxia, dizziness, incoordination, confusion)
      □ $FiO_2$ 10% – lethargy from cerebral edema
      □ $FiO_2$ <6% – unlikely to be compatible with life
    ▪ Majority are occupational exposures; removal from exposure leads to rapid improvement; residual symptoms suggest sequelae of ischemia.
      □ Those at risk for these complications should be monitored.
      □ Otherwise, supportive oxygenation should be sufficient.
      □ Identification of gas by trained team for on-site control.
    ▪ Carbon dioxide, nitrogen, helium, methane, nitrous oxide
  o Irritants
    ▪ Dissolve on respiratory mucus, causing an inflammatory response
    ▪ Products are usually acid or alkaline; may be free radicals
    ▪ Water solubility determines syndrome of effects (see Table 4-2).
### Table 4-2. Respiratory Irritants and Their Effects

<table>
<thead>
<tr>
<th>Solubility</th>
<th>High</th>
<th>Intermediate</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affected Area</td>
<td>Eyes and upper airway, immediate tearing, nasal burning, cough, pungent odor</td>
<td>Massive exposure: upper airway effects</td>
<td>Distal airways</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Smaller exposure: lower airway effects (may be delayed)</td>
<td>Lack of offensive odor permits higher incidence of prolonged exposure</td>
</tr>
<tr>
<td>Examples</td>
<td>Ammonia: corrosive gas; forms ammonium hydroxide on contact, causing bronchospasm, pulmonary edema. Supportive treatment.</td>
<td>Chlorine: green-yellow gas, acrid odor. Forms acids and oxidants on contact, causing irritation, inflammation, and edema in eyes, upper airway. Symptoms are cough, hoarseness, pulmonary edema. Supportive treatment.</td>
<td>Phosgene: gas with density four times that of air, forms white cloud with odor of newly mown hay. Forms CO₂ and HCl in alveoli, causing diffuse capillary leak and pulmonary edema. Observe for 24 hours, even if asymptomatic. Identification of sequelae is more important than identification of the toxin.</td>
</tr>
<tr>
<td></td>
<td>Capsaicin, &quot;mace,&quot; or &quot;pepper spray&quot;: local transient noxious effects, though fatalities from pulmonary edema have occurred with significant inhalations</td>
<td>Nitrogen oxides: silo gas, fire combustion, industrial sources. Slow conversion to nitric acid in alveoli: initial dyspnea and flu-like symptoms with transient improvement prior to worsening symptoms, heralding pulmonary edema 24 to 72 hours after exposure.</td>
<td></td>
</tr>
<tr>
<td>Evaluation and Treatment</td>
<td>Symptoms last a few hours. Decontaminate eyes using irrigation with copious normal saline, skin with soap and water. Supportive treatment, may be discharged after improved.</td>
<td>Observe for several hours prior to discharge. Steroids may prevent bronchiolitis obliterans.</td>
<td>Supportive care: Remove from source, give oxygen, inhaled bronchodilators for wheezing, early intubation for upper airway edema. Serial evaluation of oxygenation and ventilation. Observe at least several hours, consider admission as delayed sequelae are common.</td>
</tr>
<tr>
<td>Complications</td>
<td>Significant exposure can cause laryngospasm, laryngeal edema, bronchospasm, or pulmonary edema.</td>
<td>Delayed pulmonary edema</td>
<td>After a 2- to 24-hour latency, pulmonary endothelial injury occurs. Mild at onset, may progress to acute respiratory distress syndrome over the next 24 to 36 hours. Upper airway swelling may lead to delayed upper airway compromise.</td>
</tr>
</tbody>
</table>

### Smoke

- **General**
  - Heat capacity of air increases with steam or soot.
    - Thermal damage is more probable.
  - Carbonaceous particles with adsorbed toxins
    - Damage through inflammation, acid, free radicals
    - Substances in smoke depend on fuel of fire: consider carbon monoxide, cyanide toxicity
- **Symptoms of smoke inhalation:** cough, dyspnea, bronchospasm, stridor
  - Delayed laryngeal injury from heat and irritation
  - Singed nasal hairs, sooty sputum suggest exposure
Diagnostics
- Consider chest film: hyperinflation, edema
- Co-oximetry is more reliable than arterial blood gas and pulse oximetry.
- Lactate concentration >10 with acidosis suggests cyanide toxicity.

Treatment
- Frequent re-evaluation and management of the airway
  - Direct visualization is most accurate.
  - Early intubation prior to decompensation
- Supportive care, oxygenation
- Bronchodilators are frequently used; no evidence of benefit
- Steroids are not indicated; may be harmful
- Treat carbon monoxide and cyanide toxicities if present.
- Consider admission to ICU or burn center if
  - Concern for respiratory or airway compromise
  - Significant exposure (closed space, sooty sputum)

Poisons and toxins (see Chapter 12, Toxicologic Disorders)
- Nerve agents: acetylcholinesterase inhibitors (with acetylcholine buildup) cause seizures, coma, respiratory depression, apnea, fasciculations with progression to weakness and paralysis; also tachycardia, hypertension, diaphoresis, miosis, lacrimation, salivaition, bronchorrhea, bronchospasm, bradycardia, vomiting, diarrhea, urination
  - Cholinergic syndrome (SLUDGE: salivation, lacrimation, urination, diarrhea, gastrointestinal cramping, and emesis): key pulmonary symptoms are weakness, respiratory depression, apnea, bronchorrhea, bronchospasm; airway issues include salivation and vomiting
  - Absorbed via all routes: high contamination risk to providers
  - Treatment: decontamination first, then oxygenation; succinylcholine will have a prolonged effect
    - Antidotes: atropine until secretions are dried; pralidoxime (2-PAM) will reverse weakness, fasciculations, neuromuscular findings (variable effectiveness with different nerve agents)

4.6 PULMONARY EMBOLISM/INFARCT

Septic Emboli
- General
  - May be from internal jugular vein septic thrombophlebitis caused by a pharyngeal infection that spread to the parapharyngeal space
    - Fusobacterium or MRSA (in patients who are intravenous drug abusers)
  - Consider if risk factors and physical findings raise concern for endocarditis

Venous Thromboembolism
- Deep Vein Thrombosis (DVT)
  - General
    - Superficial venous system: greater and short saphenous, perforating veins
    - Deep venous system: calf veins (anterior, posterior tibial, peroneal veins) join at knee to form the popliteal vein, becoming the femoral vein
    - Both "superficial" and "deep" femoral veins are deep veins.
      - These join to form the "common" femoral and then the external iliac vein.
    - Considered "proximal" DVT if popliteal or higher
Presentation
- Early symptoms: mild cramping, sense of fullness, pain
- Signs: unilateral swelling, edema, erythema, warmth, tenderness of extremity, dilation of superficial veins, palpable venous "cord"
  - Homan's sign is of no utility.

Diagnosis
- Determination of pretest probability (risk stratification)
  - Clinical gestalt or objective scoring (e.g., Wells criteria)
- Venous duplex ultrasonography (95% sensitive and specific for proximal DVT) is sufficient to rule out DVT in low-risk patients.
  - Higher risk requires D-dimer or a repeat duplex in 1 week.
- D-dimer is sensitive enough to rule out DVT (in low-risk patients)
  - False positives abound, as D-dimer is elevated in any condition causing fibrin deposition, including pregnancy, malignancy, advanced age, prolonged bed rest, recent surgery, infection, inflammation, new indwelling catheters, stroke, and MI.
- MRI can image pelvic veins and inferior vena cava (unlike duplex) but is limited by cost and availability.

Treatment
- Anticoagulation — in the absence of contraindications: heparin followed by warfarin (continue anticoagulation for 3 months)
  - Unfractionated heparin and low-molecular-weight heparin (LMWH) are equally effective.
  - Select patients may be discharged with LMWH and warfarin if appropriate education and follow-up are given.
- Patients who are not candidates for anticoagulation and those who have recurrent clots despite anticoagulation should be considered for caval interruption (e.g., an inferior vena cava filter)
- Treatment for isolated calf or saphenous vein thrombosis (in an otherwise young and healthy patient) is controversial: 25% of thrombi may still propagate proximally; it is unclear if optimal therapy is anticoagulation versus antiplatelet agent with follow-up duplex

Pulmonary Embolism (PE)

General
- Usually caused by emboli from a lower extremity (or upper) DVT
  - Vasoactive and inflammatory mediator release
    - Obstruction or vasoconstriction may lead to infarct.
  - V/Q (ventilation/perfusion) mismatch causes dyspnea
- Highly variable presentations can make this a difficult diagnosis.
  - 40% lack identifiable classic risk factors

Presentation
- The risk factor categories outlined by Virchow are still useful:
  - Stasis — bed rest, hospitalized, immobilized, stroke, spinal cord injury, prolonged travel, obesity, recent surgery
  - Hypercoagulability — malignancy, estrogen therapy, pregnancy, deficiency of protein C, protein S, or antithrombin III
  - Venous injury — trauma (especially pelvic/lower extremity), burn
  - Smoking is not an independent risk factor for venous thromboembolism.
- Possible symptoms include dyspnea (90%), chest pain (only 70%, classically pleuritic), hemoptysis, weakness, dizziness, syncope or near-syncope, extremity discomfort, or malaise
- Tachypnea (>16 breaths/min) is useful to recognize; half of patients are tachycardic; physical exam can be variable and may reveal a patient in no distress or anxious and diaphoretic; signs of DVT may or may not be apparent
- Massive PE can cause acute right heart failure (jugular vein distention [JVD] with clear lung fields, loud P2)
- Rales, wheezing, even low-grade fever can occur

**Diagnosis**
- The presence of otherwise unexplained signs and symptoms should prompt consideration of the diagnosis (and further testing).
- Selection of testing depends on pretest probability
  - Clinical experience and subjective assessment (gestalt)
  - Objective decision rule score or flow algorithm
  - These are highly dependent on resources available.
    - Specific to institution or practice environment
- D-Dimer accuracy varies by assay; generally rules out DVT/PE for low-probability patients only. A positive result indicates further testing, usually imaging (V/Q scan or CT angiography).
- Chest film abnormalities, while common, are usually not specific to PE; its most useful function is to support alternative diagnoses (pneumonia, pneumothorax). Unilateral basilar atelectasis increases the probability of PE.
  - Hampton's hump – if infarct has been present for 3 or more days, may (rarely) see pleural-based, wedge-shaped infiltrate
  - Westermark’s sign – unilateral lung oligemia distal to clot is a (rarely seen) sign of a large PE
- Similarly, ECG is most useful for identifying alternate diagnoses.
  - Changes caused by PE are usually related to pulmonary hypertension: tachycardia, symmetric anterior T-wave inversion, S1Q3T3 pattern (S-wave in lead I, Q-wave in lead III, and an inverted T-wave in lead III), or new RBBB.
- V/Q scan
  - “Low probability (normal)” scan with low pretest probability rules out PE with 96% negative predictive value.
  - “High probability” scan in high pretest probability diagnoses PE with 96% positive-predictive value.
  - Unfortunately, “moderate” or “indeterminate” results are very common and mandate further imaging (usually CT angiography) for definitive results:
    - 98% sensitive and 10% specific
    - Low specificity is a major flaw of V/Q.
    - Specificity is decreased by airspace disease.
- Helical CT pulmonary angiography is increasingly available:
  - Sensitivity and specificity are around 90% but depend on scanner/image quality; extremely high pretest probability with a negative or poor-quality CTA may need to be pursued with V/Q or formal angiography.
  - Some protocols include CT venography (delayed imaging of the leg veins following the single contrast bolus given for CTA of the thorax) to evaluate for DVT, with equivalent results to duplex ultrasonography.
CTA can also, like chest film and ECG, provide a more likely diagnosis that significantly lowers the likelihood of PE.

- Arterial blood gas levels may be normal; the alveolar-arterial gradient is normal in 25% of patients.
- Hypoxemia is common, but $\text{PaO}_2$ of 80 to 90 mm Hg is more sensitive (95%) than specific (<50%); suspect PE if $\text{PaO}_2$ is <70% and not explained by another diagnosis.
- Formal pulmonary angiogram is still the "gold standard."
- Echocardiography, MRI/MRA – no clear role

TREATMENT

- Anticoagulation
  - Unfractionated heparin and low-molecular-weight heparin have equivalent effect and are safe in the absence of contraindication to anticoagulation.
  - No evidence for starting prior to imaging in patients with high pretest probability; benefit may outweigh risk

- Thrombolytic therapy
  - Remains controversial but likely beneficial to administer in absence of contraindications if patient is at high risk for death, shock, or respiratory failure within a week:
    - Predictors include hypotension, tachycardia greater than systolic BP, high B-type natriuretic peptide or troponin I, hypoxia on room air, echocardiography showing RV failure.
    - A cardiology or cardiac surgery consult is prudent, when available, prior to thrombolysis in patients who are not in extremis.
    - Patients with PE having a major (i.e., intracranial) contraindication to anticoagulation should have an inferior vena cava filter placed urgently.
  - Surgical embolectomy may even be a possibility for patients with known right heart thrombus or who are not good candidates for thrombolysis; interventional radiology may alternatively perform catheter thrombectomy.
  - Hypotension (without pulmonary edema) should be treated aggressively with IV crystalloid; consider prompt thrombolysis.

PROGNOSIS

- Most patients treated for PE with heparin begin to improve by the next day and usually have an almost-total recovery:
  - In-hospital mortality for those without hypotension is 10%.
  - Up to 20% of survivors have permanent residual dyspnea.
- The clinical course of obstructive PE is often unpredictable. Patients may be intermittently unstable due to variable RV outflow obstruction by clot or new embolization, arrhythmias, or worsening respiratory failure; progression of RBBB is ominous.
- Cardiac arrest (PEA, asystole) often follows

4.7 PULMONARY INFECTIONS

Lung Abscess

- General
  - Initial aspiration event introduces bacteria into dependent lung
  - Pneumonitis and inflammation ensue, initiating localized suppuration and necrosis, leading to eventual cavitation.
  - Risk factors are alcohol abuse, aspiration, poor dental hygiene

- Prognosis
  - Most patients treated for PE with heparin begin to improve by the next day and usually have an almost-total recovery:
    - In-hospital mortality for those without hypotension is 10%.
    - Up to 20% of survivors have permanent residual dyspnea.
  - The clinical course of obstructive PE is often unpredictable. Patients may be intermittently unstable due to variable RV outflow obstruction by clot or new embolization, arrhythmias, or worsening respiratory failure; progression of RBBB is ominous.
  - Cardiac arrest (PEA, asystole) often follows
Presentation
- Cavitation usually occurs 1 to 2 weeks after initial aspiration event.
- Fever, chest pain, cough, tachypnea, dyspnea, weight loss, malaise, sweats
  - Putrid expectoration in >40% of patients

Diagnosis
- Chest film is diagnostic, showing inflammatory infiltrate with one or more cavities containing air-fluid levels (see Image #8)

Treatment
- Conservative medical management, antibiotics covering for anaerobes and aerobes

Pneumonia (also see Chapter 5, Pediatrics)

General
- Community-acquired pneumonia is the sixth leading overall cause of death.
- Risk factors for developing pneumonia or having more severe disease
  - Immunosuppression (via medication or recent/chronic comorbidities), impaired mucociliary clearance, bacteremia, nasogastric or endotracheal intubation (and other aspiration risks)
- ED focus is on diagnosis of pneumonia and identification of any key factors that influence the type of pneumonia likely to have developed:
  - Host factors, symptoms, setting, and type of possible exposures
  - These affect antibiotic choice, disposition, and prognosis.
  - The actual etiologic agent can be identified in less than two thirds of cases, even with an extensive inpatient workup.

Presentation
- Commonly: dyspnea, fever, cough with purulent sputum
  - If the lower lobe is involved, the patient may have low back or abdominal pain.
- Elderly: complaints may be nonspecific; with decline in baseline functional status, the patient becomes confused; in severe cases, the patient presents in frank sepsis.
- Infants: fever, irritability, tachypnea, tachycardia, intercostal retractions, nasal flaring, grunting; may not have cough
- Physical exam may reveal inspiratory rales (alveolar fluid), bronchial breath sounds (consolidation), dullness and decreased breath sounds (effusion), rhonchi and wheezing (bronchial congestion); tachypnea, tachycardia, fever

Diagnosis is based on chest film findings.
- As with history and risk factors, the patterns listed below imply a causative agent but not with certainty. Suggestive but nonspecific clues:
  - Segmental or subsegmental infiltration and air bronchograms – pyogenic bacterial
  - Lobar – *S. pneumoniae* or *Klebsiella*
  - Dense lobar infiltrate with bulging fissure – *Klebsiella*
  - Fluffy or patchy infiltrates – wide variety of agents can cause this pattern: *Chlamydia, Mycoplasma, Legionella*, viruses, fungi
  - Interstitial pattern – usually *Mycoplasma*, virus, or PCP
  - Tiny nodules throughout lung fields – miliary pattern seen with granulomatous diseases (e.g., TB or fungal)
  - Dependent areas (superior segment of lower lobe or posterior segment of upper lobe) – aspiration
Peripheral – hematogenous spread (e.g., *S. aureus*)
- Apical infiltrate – be concerned about TB
- Associated features seen on chest film may also be clues
  - Infiltrate with hilar or mediastinal adenopathy – TB, fungal disease, or related to lung mass
    - Hilar adenopathy without infiltrate: atypicals
  - Cavitation – anaerobes, aerobic gram-negative rod, *S. aureus*, fungal disease, TB, or malignancy
  - Abscess – *S. aureus*, Klebsiella
  - Pneumatoceles or pneumothorax – PCP
  - Pleural effusion – many, including bacterial agents, *Chlamydia*, *Legionella*, TB; if anaerobics, at risk for empyema
- Cavitation- anaerobes, aerobic gram-negative rod, *S. aureus*, fungal disease, TB, or malignancy
- Abscess – *S. aureus*, Klebsiella
- Pneumatoceles or pneumothorax – PCP
- Pleural effusion – many, including bacterial agents, *Chlamydia*, *Legionella*, TB; if anaerobics, at risk for empyema
- Radiographic features are not specific for making distinctions between “typical” and “atypical”: either can show a dense infiltrate or diffuse interstitial infiltrates; immunocompromised patients commonly have unusual appearances for a given causative agent.
- The absence of radiographic evidence for pneumonia does not exclude its existence; clinical pneumonia should be treated, and repeat x-ray films often later reveal supportive evidence.
- Blood culture and Gram stain or sputum culture are not usually positive, and their results rarely alter therapy; however, these tests may be considered for patients with severe disease.
- Thoracentesis with pleural fluid analysis should be done for patients with parapneumonic effusions (see Section 4.2).
- Lab – an elevated (or normal) WBC count is not sensitive or specific for pneumonia or its subtypes; a decreased WBC is a clue to immune status; evaluation of certain patients may include assessment of renal, hepatic, or acid-base status.
- Pulse oximetry – on any patient suspected to have pneumonia
- Viral tests – rapid antigen tests for RSV, *influenza*, CMV are available but of limited utility; more specific than sensitive

**Treatment**
- Symptomatic treatment with antipyretics, analgesics; patients with COPD or asthma may benefit from bronchodilators and steroids.
- Replenish intravascular volume as indicated; treat for sepsis if present.
- Isolation
  - If the patient has risk factors or symptoms of TB or was exposed to TB in the past - initiate prior to chest film or other evaluation
  - Isolate those with influenza, varicella, plague
- Antibiotics
  - Community-acquired pneumonia, outpatient – macrolides, doxycycline, or third-generation “respiratory” fluoroquinolone (moxifloxacin, gatifloxacin, levofloxacin) for 7 to 10 days
    - If the patient is older than 60 or immunocompromised, use fluoroquinolone alone or a macrolide combined with “extended spectrum” cephalosporin.
  - If the patient is hospitalized, administer a second- or third-generation cephalosporin or penicillin plus a β-lactamase inhibitor, usually with macrolide; can also give fluoroquinolone as monotherapy
    - Early administration of antibiotics decreases mortality and days in hospital
- Disposition
  - Optimal decision is based on good clinical judgment with the assistance of a decision rule.
    - Prospectively validated to predict mortality risk
Aspiration Pneumonia
- Infectious condition resulting from inhalation of inflammatory oral or gastric contents into the lungs
- Aspiration risk is higher with stroke, dysphagia, tube feeding, transient or permanently decreased level of consciousness, alcohol or drug intoxication, seizure, anesthesia, advanced age, and supine position.
- Severity of symptoms is related to the volume of material aspirated, amount of bacterial contamination, and pH of aspirated material.
- Aspiration pneumonia should be differentiated from the mere event of aspiration, which itself can cause pneumonia-like symptoms and signs (fever; leukocytosis; productive cough; localized rales, wheezes, or rhonchi; tachypnea; tachycardia; and even infiltrates that resemble bacterial pneumonia).
  - RLL is the most common location of pneumonia if upright/sitting; any lobe if supine
- Healthy patients who are not hypoxic and have no infiltrate may be observed.
- Elderly or chronically ill patients who have signs/symptoms of infection should be treated:
  - Clindamycin, cefoxitin, ticarcillin-clavulanate, pipercillin-tazobactam
- If the patient has a fever of new onset, an expanding infiltrate 36 hours after the event, or unexplained worsening in clinical status, treat for pneumonia:
  - Usually polymicrobial with anaerobes: *Peptostreptococcus, Bacteroides, Fusobacterium*
  - Empiric antibiotics for aspiration: no proven benefit

Atypical Pneumonia
- *Mycoplasma and Chlamydia* are common causes of community-acquired pneumonia in healthy patients under the age of 40; together they cause up to 15% of cases
- Artificial distinction from “typical,” impossible to distinguish with certainty on clinical grounds alone
  - Atypical nature of pneumonia is NOT predicted by:
    - Gradual onset, viral prodrome, absence of rigors, nonproductive cough, lower fever, absence of pleurisy, absence of consolidation, low WBC count
- *Mycoplasma pneumoniae*
  - Subacute cough (usually nonproductive), sore throat, headache, retrosternal chest pain, pharyngeal erythema, cervical lymphadenopathy, scattered rales and rhonchi
  - Chest film – patchy infiltrates, hilar adenopathy, pleural effusions
  - May be associated with bullous myringitis (a finding that is neither sensitive nor specific), rash, arthralgia, hematologic abnormalities (and, rarely, renal failure)
- *Chlamydia pneumoniae*
  - In young adults: mild subacute cough, sore throat, fever; may have rales/rhonchi, bronchitis, wheezing, sinusitis
  - Chest film – patchy subsegmental infiltrate (more often seen in elderly)
- *Legionella* – has been outbreak-related, via water supply, but may also cause up to 20% of cases of community-acquired pneumonia; regionally variable; risk factors include cigarette smoking, COPD, transplant/immunosuppression; disease may be mild, but can cause multisystem organ failure with acute respiratory distress syndrome
  - Severe illness with malaise, lethargy, high fever, dry cough, chest pain; may have altered mental status
  - GI symptoms (abdominal pain, vomiting, diarrhea) are common
  - Chest film: patchy infiltrate, hilar adenopathy, pleural effusions
- Atypicals are not sensitive to β-lactams; use doxycycline, macrolide, or third-generation fluoroquinolone
Bacterial
- **S. pneumoniae** - most common cause of community-acquired pneumonia in adults
  - Even more common in severe disease
    - Asplenic, sickle cell disease, immunosuppressed, multiple myeloma, agammaglobulinemia: risk for fulminant pneumococcal bacteremia, sepsis, and death
  - Classic presentation: “sudden shaking chill” preceding high fever and rusty sputum, chest pain; may be preceded by URI
  - Chest film – lobar pneumonia with parapneumonic effusion
  - Risk factors for likely penicillin resistance: extremes of age, day care, immunosuppression, alcoholism, cancer
    - Vancomycin, imipenem, or third-generation fluoroquinolone
- **H. influenzae** – second most common cause of community-acquired pneumonia, especially in alcoholics and patients with COPD, sickle cell disease, and those with malnutrition, malignancy, or diabetes
  - Usually gradual onset of a low-grade fever, productive cough
  - Can have sudden onset of chest pain, dyspnea, productive cough
  - Chest film – pleural effusions; multilobar infiltrates are common
- **S. aureus** (including MRSA) incidence is rising; can be necrotizing and cavitary; often follows influenza epidemics
  - Typically insidious onset with low-grade fever, productive cough, dyspnea
  - IV drug abusers are at high risk, even for bilateral involvement (septic emboli from tricuspid endocarditis)
  - Chest film – extensive disease with empyema, pleural effusions, multiple infiltrates
- **Moraxella (Branhamella) catarrhalis** – especially in COPD
- **Klebsiella pneumoniae** is unusual; it tends to be seen in elderly or diabetic patients and in those with some aspiration risk, e.g., alcoholics
  - Acute severe disease with fever, rigors, chest pain
    - Classic “currant jelly” sputum (infection can be necrotizing and hemorrhagic)
    - Abscess, empyema, bacteremia, mortality are common
  - Chest film – bulging fissure below an upper lobar consolidation
- **Pseudomonas aeruginosa**: more in inpatients, intubated patients, nursing home residents
  - Severe illness with cyanosis, confusion, rash, green sputum
- **Chlamydia trachomatis**: transmitted perinatally to neonates; pneumonia typically presents between 3 and 19 weeks of age with congestion and cough; can cause apnea or hypoxia
  - Half of patients have conjunctivitis or abnormal middle ear exam; some retractions, inspiratory crackles are possible
  - Interstitial infiltrates are bilateral and diffuse; treatment is with erythromycin and supportive care.

Fungal
- **Histoplasma capsulatum** (Mississippi/Ohio River valleys), **Blastomyces dermatitides** (slightly wider area), and **Coccidioides immitis** (desert Southwest); regionally present in soil
  - Acute or chronic pneumonia as well as asymptomatic granulomas and hilar adenopathy are possible results.
- **PCP** (see Image #55)
  - Caused by a fungal organism, which has been renamed *Pneumocystis jiroveci*; still may be abbreviated PCP (*Pneumocystis pneumonia*)
  - Seen in AIDS patients and other immunocompromised hosts
  - Subacute presentation with fatigue, dyspnea, dry cough, chest pain, fever
• Viral
  o Often preceded by URI symptoms; onset may be insidious
  o Cough more likely to be nonproductive, without chest pain
  o Exam may show mild, scattered rhonchi; consolidation is rare
  o Infants, young children: RSV and parainfluenza (in cooler months)
  o Adults: influenza is the predominant cause of viral pneumonia, striking mostly in the elderly
  o CMV affects primarily the immunosuppressed (e.g., transplant patients)
  o Varicella-zoster (chicken pox) — causes severe pneumonia in adults
  o Severe acute respiratory syndrome (SARS) — recently identified virus from southeast Asia, with a 15% mortality rate (even higher in the elderly)

• Unusual Causes
  o Often associated with indirect contact with an animal vector or its contaminated surroundings:
    - Coxiella burnetii (cattle, sheep, cats: Q fever)
    - Rhodococcus equi (horses)
    - Bordetella bronchiseptica (dogs: kennel cough)
    - Yersinia pestis (rodents and fleas: plague)
    - Hantaviruses (rodents)
    - Francisella tularensis (rabbits: tularemia)
    - Chlamydia psittaci (birds: psittacosis)

• Special Patient Populations
  o Community-acquired pneumonia in healthy patients (including military recruits) is usually viral, mycoplasmal, or pneumococcal.
  o COPD
    - Often colonized with S. pneumoniae or H. influenzae
    - More aerobic gram-negative rods, less pneumococcal disease
    - If the patient uses steroids frequently, nonbacterial infections may be present and mimic bacterial pneumonia:
      - Aspergillus fumigatus, Strongyloides stercoralis, Mycobacterium tuberculosis
  o Diabetes
    - Higher risk of bacteremia, mortality
    - More sequelae with gram-negative rods (bacteremia, empyema, necrosis)
    - Preexisting neuropathy and/or nephropathy: avoid aminoglycosides
  o Pregnancy
    - Pneumonia is a common non-obstetric cause of maternal mortality. Infection may cause preterm delivery/fetal loss.
    - Aspiration during labor and delivery
    - Varicella pneumonia — cough, dyspnea, pleuritic chest pain, hemoptysis a few days after onset of fever, rash, malaise
      - Chest film shows miliary or nodular infiltrates
      - May progress to acute respiratory distress syndrome; risk of fetal morbidity with varicella zoster virus
    - Penicillins, cephalosporins, and erythromycin (not estolate) are safe during pregnancy.
Nursing home patients
- 30% of all nursing home infections, with 50% mortality
- Nonimmunized individuals are subject to influenza outbreaks.
- Aspiration is common; gram-negative rods are common.
- Recent hospitalization increases risk of Enterobacteriaceae, Pseudomonas, S. aureus.

HIV
- Many patients are unaware of their HIV status or unwilling to volunteer risk factors; consider potential for positive HIV status
- AIDS-defining illnesses include recurrent bacterial pneumonia, pulmonary TB, and opportunistic pneumonia.
- If the CD4 count is >800, opportunistic pneumonia is uncommon.
  - Pneumococcus and H. influenzae remain top etiologies.
  - Chest film may not have the usual appearance for these pathogens.
- If the CD4 count is 250 to 500, consider M. avium complex, M. tuberculosis, Cryptococcus neoformans, and H. capsulatum.
  - Cryptococcus chest film: diffuse or localized infiltrates, cavitation, nodules, miliary disease, lymphadenopathy, or pleural effusions
- If the CD4 count is <200, Pneumocystis pneumonia is possible.
  - Usually subacute presentation with nonproductive cough, dyspnea on exertion, weight loss, fever
    - Fever is absent in 20% of patients.
  - Tachypnea, tachycardia, and hypoxemia are common.
    - Look for desaturation on exertion if history is unclear.
  - Chest film: bilateral interstitial infiltrates are classic, but not universal.
  - LDH is often quite high.

Transplant/immunosuppressed
- Usual pathogen list plus CMV, varicella, HSV, and gram-negative rods
- Fungi: Candida, H. capsulatum, Pneumocystis

Pulmonary Tuberculosis
- General
  - Infection with Mycobacterium tuberculosis (a slow-growing aerobic rod) is transmitted by respiratory droplets from a cough or a sneeze or by talking.
  - Immunocompetent hosts form granulomas as an inflammatory response, walling off the infection into latency. These may progress to caseation necrosis and calcification. Immunocompromised patients have early hematogenous spread, or “reactivation,” if immunocompromise develops or worsens after initial infection occurs.
  - Activation can occur on exposure or after years of dormancy.
  - 5% of the US population and one third of the world population is infected
- Predisposing Factors
  - High risk of exposure or active disease: occupational, immigrants from countries where TB is endemic (Asia, Africa, Latin America), homelessness, incarceration, having close contact with potentially infected persons (halfway house, nursing home), immunosuppression (diabetes, dialysis, malignancy, malnutrition, but especially HIV), alcohol or drug abuse
  - Macrophages are unable to destroy the bacillus or keep it contained.
• Presentation
  o Initial infection is usually asymptomatic in healthy people.
    ▪ May have mild fever/malaise due to immune response
    ▪ Up to 10% of those who convert to a positive purified protein derivative (PPD) skin test will get active TB.
      □ Half (5%) in 2 years: "acute primary tuberculosis"
      □ Half (5%) later: "reactivation tuberculosis"
  o Among patients who are HIV and PPD positive: high frequency of active disease
    ▪ Nearly 40% rate of acute primary TB within 6 months
    ▪ Up to 10% per year thereafter will have reactivation TB
  o "Postprimary" TB is active or chronic disease in a patient known to be previously infected; reinfection and reactivation are indistinguishable.
  o Symptoms may be minimal initially; systemic reaction causes constitutional symptoms (anorexia, weight loss, fatigue, malaise, weakness, headache, chills, fever with "night sweats")
  o Variably productive cough is the most common symptom, followed by the constitutional symptoms listed above; chest pain, dyspnea, or hemoptysis can also occur.
    ▪ Up to two thirds of ED patients with active TB did not have a pulmonary chief complaint, and one third may have no cough at all.
  o Hemoptysis indicates severe lung involvement (caseous sloughing or endobronchial erosion, causing blood vessel rupture)
  o Spontaneous pneumothorax from peripheral lung erosion
  o Extrapulmonary TB may be present and symptomatic as well.
    ▪ Bones, nodes, adrenals, joints, GI/GU tract, meninges, pericardium
  o Atypical presentations are common in elderly, infants, immunocompromised
    ▪ Extrapulmonary disease, lower/mid-lung bronchopneumonia
  o Physical exam is usually abnormal, often revealing an infiltrate or consolidation; general appearance is suggestive: pallor or cachexia
• Complications
  o Pneumothorax, empyema, endobronchial spread (causing bronchiectasis or bronchial stenosis), superinfection (e.g., aspergilloma or “fungus ball”), hemoptysis (may be massive), pericarditis, other extrapulmonary disease
• Diagnosis
  o Risk factor screening is crucial to the diagnosis.
    ▪ Consider in any patient with cough and risk factors
  o Chest film is the most useful test.
    ▪ A normal radiograph has a high negative predictive value (NPV): falsely negative in only 1% of immunocompetent individuals, but up to 15% in HIV-positive patients.
      □ Classic finding is upper lobe cavitation or infiltrates
      □ Immunosuppressed: adenopathy, atypical infiltrates
    ▪ Only serial exams can distinguish active from inactive disease.
  ▪ Primary TB
    ▪ Infiltrates can be in any lobe, usually single
    ▪ Associated with hilar/mediastinal lymphadenopathy
      □ Usually unilateral, with infiltrate
      □ May be massive in young children, with atelectasis
Pleural effusion, moderate to large
- Miliary (diffuse) 1- to 3-mm noncalcified nodules
  - Bilateral, may be more prominent in basilar regions
- Tuberculoma: nodular parenchymal lesion
  - If calcified, termed “Ghon focus”

**Postprimary TB**
- Upper lung infiltrate or consolidation
  - Upper lobe or superior segment of lower lobe
  - Variable size; may show cavitation
  - High infectivity, complication rate
- May progress to multiple lobe involvement
- Bilateral upper lobe disease: high positive predictive value (PPV) for TB
- Fibroproductive lesion is characteristic of granulomatous disease: irregular, angular, stranding toward hilum, having one or more calcified nodules
  - Progresses to scarring, anatomic distortion
- Endobronchial spread – 5- to 10-mm nodules clustered in dependent portion of lung. May consolidate: “galloping consumption”

- Sputum studies, serially obtained, establish the diagnosis.
  - Direct microscopy of acid-fast bacilli (AFB) stained smear – results become available in 24 hours
    -Insensitive if few organisms are present in sample
    -More sensitive if severe or cavitary disease is present
- Culture confirms diagnosis
  - Was a 3- to 8-week delay – now as little as 7 to 14 days
  - More sensitive than microscopy
  - Characterize susceptibility to drugs
- DNA (PCR) or RNA (MTDT) amplification may provide results in hours, but sensitivity is much lower if AFB smear was negative.
- Nebulizer-induced sputum or gastric aspirate may be required.
  - Negative pressure room only; ensure person performing test is maximally protected from acquired infection
- Fiberoptic bronchoscopy/bronchoalveolar lavage may be needed.

- Tuberculin skin testing (PPD) – negative in 20% of patients with active disease!
  - Subcutaneous 0.1 mL, induration measured at 48 to 72 hours, positive if:
    - >5 mm (HIV, close TB contact, chest film consistent with TB)
    - >10 mm (health care worker, others “at risk” or <4 years old)
    - >15 mm (all others: healthy, no risk factors)
  - False negatives can occur due to poor immune responses
  - Useful in detecting latent disease, at least 3 to 8 weeks after exposure
  - Cross-reactivity with nontuberculous mycobacteria

- Laboratory tests are nonspecific: may reveal anemia, high ESR, hyponatremia
Treatment

- Immediate isolation at first suspicion of the disease.
- Massive hemoptysis (>600 mL/24 hr) is the most emergent presentation
  - Prevent aspiration by intubation with large tube (8.0) to permit emergent bronchoscopy; to protect the unaffected lung, the patient may be positioned with the bleeding lung dependent, or intubation may be done selecting the mainstem of the unaffected lung.
  - Bronchoscopy, possibly surgery or embolization
- Active TB requires isolation and a multi-drug therapy regimen – preferably directly observed therapy (DOT), as noncompliance is common
  - If these can be accomplished with appropriate arrangements and good follow-up, admission is not necessarily mandated.
  - Generally, these patients are admitted for monitoring of response to treatment, side effects, complications, and compliance. Admission is clearly indicated for the elderly, HIV positive, and otherwise ill patients.
  - A minimum of 2 weeks of therapy is required before the patient is considered noninfectious.
- HIV patients
  - At increased risk of drug interactions, depending on their HAART regimen
  - Treatment lightens the HIV burden
  - First-line therapy is RIPE (rifampin, INH, pyrazinamide, ethambutol); pyridoxine instead of pyrazinamide in pregnancy
  - Second-line therapy may include amikacin or a fluoroquinolone if the TB is known to be multidrug-resistant.
- Latent TB
  - Active disease must first be ruled out, using clinical evaluation and a chest film.
  - INH treatment must last 6 to 9 months.
  - If HIV positive, rifampin and pyrazinamide for at least 2 months

4.8 TUMORS

Oncologic Emergencies

- General
  - Oncologic emergencies may arise in two subgroups of ED patients:
    - Those diagnosed with malignancy, often with ongoing treatment
    - Those with complaints suggestive of a new diagnosis
- Examples
  - Local effects
    - Pericardial effusion with tamponade
    - Spinal cord compression, high intracranial pressure
    - Pathologic fractures
    - Upper airway obstruction
    - Superior vena cava syndrome
  - Metabolic effects
    - SIADH, hyponatremia
    - Hypercalcemia
Hematologic effects
- DVT/PE
- Fever, neutropenia, infection
- Hyperviscosity syndrome

Treatment effects
- Acute tumor lysis syndrome
- Side effects: pain, nausea, vomiting

Breast
- General
  - Delayed diagnosis is unfortunately still common and contributes to this important source of morbidity and mortality in the United States

Symptoms of Breast Malignancy
- Mass, abnormal nipple discharge, Paget's disease, palpable axillary nodes
- Inflammatory breast cancer: peau d'orange skin, nipple retraction, warmth, tenderness; may initially appear to be a cellulitis or abscess, and must be carefully differentiated; surgical consultation when in doubt
- If concern exists for cancer, refer to primary care or breast specialist for bilateral mammography and/or biopsy.

Pulmonary
- General
  - Most common cause of cancer death in the United States for men and women

Types
- Non small-cell
  - Adenocarcinoma, squamous cell, large cell
- Small cell
- Carcinoid
- Rare
  - Lymphoma, melanoma, sarcoma, histiocytoma

Diagnosis
- Radiographic abnormalities may appear prior to symptoms.
  - As incidental findings on radiographs or CT
  - Common example: solitary pulmonary nodule
- Symptoms indicate late disease.
- Symptoms that should prompt suspicion of lung malignancy in high-risk patients include the following:
  - Local
    - New cough, change in chronic cough, hemoptysis, unilateral wheezing, atelectasis, postobstructive pneumonia, cavitation, lung abscess
  - Invasion
    - Chest pain, pleural effusion with dyspnea, pericardial tamponade, SVC syndrome, hoarseness
    - Horner's syndrome – unilateral ptosis, miosis, anhidrosis
    - Pancoast syndrome – apical lung tumor may involve brachial plexus, with shoulder, arm pain, possibly Horner's syndrome
  - Metastases
    - Brain, bone, adrenals, liver are common sites.
    - Supraclavicular and cervical lymphadenopathy
• Paraneoplastic syndromes
  • Systemic: weight loss, fever, anorexia
  • Cutaneous: clubbing
  • Hematologic: hypercoagulable states, anemia from marrow replacement
  • Endocrine/metabolic:
    — ACTH: hypokalemia
    — PTHrp: hypercalcemia from PTH-related-peptide secretion
    — SIADH: hyponatremia
    — Eaton-Lambert syndrome (myasthenic)
# CHAPTER 5

## Pediatrics

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## 5.1 ABDOMINAL DISORDERS

### Differential Diagnosis of Pediatric Abdominal Pain (Table 5-1)

Table 5-1. Differential Diagnosis of Pediatric Abdominal Pain

<table>
<thead>
<tr>
<th>Infancy</th>
<th>Childhood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incarcerated hernia</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>Intussusception</td>
<td>Constipation</td>
</tr>
<tr>
<td>Pyloric stenosis</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td>Hirschsprung disease</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Colic</td>
<td>Gallbladder disease</td>
</tr>
<tr>
<td>Malrotation with volvulus</td>
<td>Ulcers</td>
</tr>
<tr>
<td>Perforation</td>
<td>UTI</td>
</tr>
<tr>
<td>Necrotizing enterocolitis</td>
<td>Hemolytic uremic syndrome</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>Henoch-Schoenlein purpura</td>
</tr>
<tr>
<td></td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td></td>
<td>Renal stone</td>
</tr>
<tr>
<td></td>
<td>Mesenteric adenitis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Adolescence</th>
<th>Extra-Abdominal Causes of GI Distress</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ectopic pregnancy</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>Hemolytic uremic syndrome</td>
</tr>
<tr>
<td>PID</td>
<td>Henoch-Schoenlein purpura</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Biliary disease</td>
<td>Pharyngitis, especially streptococcal</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>Abdominal migraine</td>
</tr>
<tr>
<td>Testicular torsion</td>
<td>Abdominal epilepsy</td>
</tr>
<tr>
<td>Henoch-Schoenlein purpura</td>
<td>Ingestions (especially iron)</td>
</tr>
<tr>
<td>Epididymitis</td>
<td>Black widow spider bite</td>
</tr>
<tr>
<td>Renal stone</td>
<td></td>
</tr>
<tr>
<td>Pancreatitis</td>
<td></td>
</tr>
<tr>
<td>Mittelschmerz</td>
<td></td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
</tr>
<tr>
<td>Ovarian torsion/cyst</td>
<td></td>
</tr>
<tr>
<td>Peptic ulcer disease (PUD)</td>
<td></td>
</tr>
</tbody>
</table>
Pyloric Stenosis

- **General**
  - Narrowing of the pyloric canal due to muscle hypertrophy
  - 1/250 live births; more common in Caucasians, rare in Asians
  - Usual onset of symptoms is during the third to fifth week of life
  - Male-to-female ratio is 4:1

- **Presentation**
  - Initially presents with occasional vomiting after meals and then progresses to vomiting after each meal; can result in classic "projectile" vomiting
  - The baby is hungry and will re-feed immediately after vomiting.
  - Weight loss may be seen as a result of continued symptoms.
  - Constipation can occur as a result of fluid loss and resultant dehydration.
  - Palpable "olive" in RUQ; passing an NG tube before trying to palpate will decompress the stomach
  - Peristaltic waves from left to right are seen after feeding.

- **Diagnosis**
  - **Labs**
    - Metabolic alkalosis: hypochloremic, hypokalemic early in the course; if the patient is markedly dehydrated, then acidosis occurs
  - **Ultrasound**
    - Normal pyloric wall thickness is <2 mm; with pyloric stenosis, the wall thickness is ≥4 mm and the canal length is elongated to ≥14 mm.
  - **Upper GI**
    - Shows the "string sign" – contrast going through the stenotic and elongated channel; false positives can result from pyloric spasm
  - **NG aspirate**
    - >5 cc of gastric volume after 3 to 4 hours of NPO status; specificity is 94%, with an accuracy of 96%

- **Treatment**
  - IV hydration with correction of electrolyte abnormalities
  - Surgical treatment – incision of the pylorus (pyloromyotomy)

Intussusception

- **General**
  - Prolapse of one part of the intestine into the lumen of an immediately adjacent distal part; most common location is the ileo-cecal junction
  - Age: most common between 3 months and 5 years of age; children younger than 2 years of age typically do not have a lead point; 60% of all cases occur in the first year of life, with peak incidence in children 6 months to 11 months of age

- **Presentation**
  - Classic triad: seen in 20% to 40% of patients
    - Intermittent colicky abdominal pain: 50% to 90% of cases
    - Vomiting: 60% to 90%
    - "Currant jelly" stools (diarrhea containing mucous and blood): 21% to 60%
  - Right upper quadrant (RUQ) mass (may also be in right lower quadrant [RLQ])
  - Colicky pain: typically the child will draw the knees up in pain, cry for 4 or 5 minutes and then look better between episodes; the child gradually becomes more irritable, with vomiting that may be bilious
• Mental status changes: initially irritable and then progresses to lethargy; lethargy is frequently the main presenting complaint, thought to be from endogenous opioid production
• Stools are highly variable; currant jelly is a late finding; occult blood seen in 75% of cases of intussusception with non-bloody stools; therefore, a rectal examination is imperative
• Low-grade fever can be present.
• Vomiting without diarrhea or fever is suspicious for intussusception.

• Diagnosis
  • Abdominal films: may be normal early in the course, but later evidence of obstruction can be seen, such as air-fluid levels, paucity of air, and dilated loops of small bowel; 89% of cases have a paucity of or abnormality in intestinal gas (particularly in the RLQ). (see Image #30)
  • Ultrasound: classic finding is “target” or “donut” sign, which is a single hypoechoic ring with a hyperechoic center; “pseudokidney” sign is superimposed hypoechoic and hyperechoic rings consisting of edematous walls of intestine and compressed mucosal layers
• Enema
  ▪ Barium: has been the gold standard and is still performed at many institutions
  ▪ Air: advantages of air versus barium
    □ Less radiation exposure
    □ Less expensive
    □ Higher success rate
    □ Easier administration
    □ If perforation does occur, air is safer for the peritoneum
  ▪ Contraindications: perforation, hypovolemic shock, peritonitis
  ▪ Recurrence
    □ 80% to 90% are reduced successfully; remainder need to go to the OR
    □ After successful reduction, 5% to 10% recur, usually within 24 hours.

• Treatment
  • Call pediatric surgeon before performing definitive treatment.

Malrotation with Midgut Volvulus
• General
  • Abnormal fixation of bowel mesentery (Ladd bands), which can lead to twisting of loop of bowel around mesenteric attachments
  • Age: usually occurs in the first months of life, but can occur anytime; mortality rate with volvulus is as high as 15%
  • Gender: male to female ratio is 2:1
  • 75% of malrotations develop volvulus, most within the first month of life
• Presentation
  • Sudden onset of bilious vomiting
  • Abdominal distension
  • Constant pain
  • Hematochezia is a late sign.
  • Jaundice
  • Shock
Diagnosis
- Abdominal films: classic “double bubble sign”—overall there is a paucity of gas, with two air bubbles—one in the duodenum and one in the stomach
- Upper GI: this is still the gold standard; the small intestine is rotated to the right side of the abdomen, with narrowing of contrast at the site of obstruction; “cork-screw” or “apple core” sign is seen, which is the spiraling of the small intestine around the superior mesenteric artery
  - However, time should not be taken to perform this study prior to a surgical consultation; the surgeon should be contacted immediately and the decision to obtain an upper GI or other study can be made by the consultant; any delay in management can lead to ischemic complications
- Ultrasound: may show a distended, fluid-filled duodenum and dilated loops of small bowel to the right of the spinal column

Treatment
- Rehydrate aggressively.
- Place NG tube.
- Start antibiotics: ampicillin, gentamicin, and metronidazole or clindamycin
- Contact pediatric surgeon immediately.

Appendicitis
- General
  - Most common surgical cause of abdominal pain in children
  - 4/1000 children; peak incidence is 9 to 12 years of age
  - Approximately 6% of the population will develop appendicitis.
  - Male to female ratio is 2:1.
- Presentation
  - Anorexia and vomiting
  - Fever
  - Diarrhea is present in 10% of patients
  - Pain is initially periumbilical, progressing to involve the RLQ
  - Tenderness over McBurney’s point; Rovsing’s sign, psoas sign, obturator sign
  - Peritonitis is caused by perforation (occurs in 15%–40% of cases; 90% rate of perforation in patients under 2 years old)
- Differential Diagnosis
  - Gastroenteritis: most common misdiagnosis; *Yersinia* is known as the great imitator and can cause RLQ pain; also consider mesenteric adenitis, lymphoma, urinary tract infection (UTI), constipation
- Diagnosis
  - Laboratory data: >96% of patients have an elevated WBC count (>10,000/mm³) or a left-shifted differential with >75% neutrophils; also elevated c-reactive protein (CRP)
  - Abdominal films: fecalith is seen in only 10% of patients; other findings—loss of psoas shadow, lumbar spine scoliosis, localized ileus in RLQ
  - Barium enema: non-filling of the appendix with contrast and resultant non-visualization; 10% to 30% of normal appendices will not fill, giving false-positive results
Ultrasound: the inflamed appendix is not compressible and measures >6 mm in diameter; fecalith may be present; peri-appendiceal fluid collection may be evidence of early perforation
- Sensitivity and specificity are operator dependent; sensitivity, specificity, and overall accuracy up to 95% in experienced hands
- Helical CT: can show inflamed appendix, fecalith, abscess, stranding of peri-appendiceal fat, which indicates an inflammatory process
- Treatment: IV rehydration, initiate antibiotics if perforation is suspected; appendectomy
  - Consider triple antibiotics (ampicillin, gentamicin, and metronidazole or clindamycin) or cefoxitin; consider meropenem if perforation

Gastroenteritis/Enterocolitis
- General
  - Etiology: viral, 60%; bacterial, 20%; parasitic, 5%; remainder, idiopathic
  - Rotavirus accounts for 30% to 60% of all cases of severe diarrhea in children between the ages of 3 and 15 months of age; bloody diarrhea is uncommon; fever and vomiting precede the onset of diarrhea
  - Norwalk virus is responsible for 40% of cases of diarrhea in older children and adults in schools and camps
- Presentation
  - Dehydration (Table 5-2)
  
  **Table 5-2. Signs and Symptoms of Dehydration**

<table>
<thead>
<tr>
<th>Clinical Findings</th>
<th>Mild ≤5%</th>
<th>Moderate 10%</th>
<th>Severe ≥15%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental status</td>
<td>Alert</td>
<td>Irritable</td>
<td>Lethargic</td>
</tr>
<tr>
<td>Tears</td>
<td>Present</td>
<td>Decreased</td>
<td>Absent</td>
</tr>
<tr>
<td>Mucous membranes</td>
<td>Moist</td>
<td>Dry</td>
<td>Very dry</td>
</tr>
<tr>
<td>Urine output</td>
<td>Normal</td>
<td>Oliguric</td>
<td>Anuric</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>Normal</td>
<td>Normal-rapid</td>
<td>Rapid</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Normal</td>
<td>Normal-rapid</td>
<td>Rapid</td>
</tr>
<tr>
<td>Fontanelle</td>
<td>Normal</td>
<td>Flat</td>
<td>Sunken</td>
</tr>
<tr>
<td>Eyes</td>
<td>Normal</td>
<td>Sunken</td>
<td>Glassy</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>&lt;2 sec</td>
<td>2–3 sec</td>
<td>&gt;3 sec</td>
</tr>
</tbody>
</table>

- Stool can vary from watery to grossly bloody
- Fever: more likely to be bacterial if temperature is >40°C
- Seizures with *Shigella*
  - *E. coli* 0157:H7 (enterohemorrhagic *E. coli*)
    - Hallmark is visible bloody stool, abdominal pain, and absence of fevers
- Diagnosis
  - Complete blood count (CBC): *Shigella* gives a normal or low WBC count but a marked left shift.
  - Electrolytes help to determine degree and type of dehydration; bicarbonate level <17 mEq/L is present in up to 94% of patients with >10% loss of body weight
  - Stool studies: fecal leukocytes, culture, Rotazyme, ova, and parasites
- Treatment
  - Oral rehydration therapy, with quick return to formula or breast milk; BRAT (bananas, rice, applesauce, toast) diet is no longer recommended
    - Consider NG tube placement for oral rehydration.
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- IV hydration
- Admit for dehydration >5%, poor social situation
- Selected antimicrobial therapy (Table 5-3)

Table 5-3. Antimicrobial Therapy for Specific Causes of Gastroenteritis/Enterocolitis

<table>
<thead>
<tr>
<th>Organism</th>
<th>Antibiotic</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Shigella</em></td>
<td>Trimethoprim/sulfamethoxazole or azithromycin</td>
</tr>
<tr>
<td><em>Campylobacter</em></td>
<td>Erythromycin or azithromycin</td>
</tr>
<tr>
<td><em>C. difficile</em></td>
<td>Oral vancomycin or metronidazole</td>
</tr>
<tr>
<td><em>Salmonella</em></td>
<td>Antibiotics generally not needed in mild disease (also thought to prolong carrier state), but may need IV antibiotics (ceftriaxone) in severe disease, immunocompromised patients, sickle cell, or patients younger than 4 months of age</td>
</tr>
<tr>
<td><em>E. coli 0157:H7</em></td>
<td>If suspected, avoid treatment with antibiotics until cultures return, as antibiotics may enhance toxin release and increase rate of hemolytic-uremic syndrome</td>
</tr>
</tbody>
</table>

- Focus is on oral hydration.
- AAP does not recommend antidiarrheals or antiemetics
  - Antiemetics are generally not needed, but oral ondansetron (0.15 mg/kg) is becoming more popular as supportive care.
  - Promethazine: FDA black-box warning against use in children under 2 years of age due to fatal respiratory depression

Gastrointestinal Bleeding (Table 5-4)

Table 5-4. Causes of Gastrointestinal Bleeding in Pediatric Age Groups

<table>
<thead>
<tr>
<th>Age</th>
<th>Upper GI</th>
<th>Lower GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1 month</td>
<td>Idiopathic</td>
<td>Anal fissure (most common)</td>
</tr>
<tr>
<td></td>
<td>Esophagitis</td>
<td>Upper GI bleed</td>
</tr>
<tr>
<td></td>
<td>Maternal blood</td>
<td>Volvulus</td>
</tr>
<tr>
<td></td>
<td>Blood dyscrasias</td>
<td>Necrotizing enterocolitis</td>
</tr>
<tr>
<td></td>
<td>Arteriovenous malformation</td>
<td>Swallowed maternal blood</td>
</tr>
<tr>
<td></td>
<td>Stress ulcer</td>
<td>Infectious colitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Milk allergy</td>
</tr>
<tr>
<td>1 month–1yr</td>
<td>Esophagitis</td>
<td>Anal fissure</td>
</tr>
<tr>
<td></td>
<td>Mallory-Weiss</td>
<td>Intussusception</td>
</tr>
<tr>
<td></td>
<td>Stress ulcer</td>
<td>Volvulus</td>
</tr>
<tr>
<td></td>
<td>Arteriovenous malformation</td>
<td>Meckel’s diverticulum</td>
</tr>
<tr>
<td></td>
<td>Stress ulcer</td>
<td>Infectious colitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Milk allergy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pseudomembranous colitis</td>
</tr>
<tr>
<td>1–12 years</td>
<td>Esophagitis</td>
<td>Anal fissure</td>
</tr>
<tr>
<td></td>
<td>Mallory-Weiss</td>
<td>Intussusception</td>
</tr>
<tr>
<td></td>
<td>Stress ulcer</td>
<td>Volvulus</td>
</tr>
<tr>
<td></td>
<td>Foreign body</td>
<td>Meckel’s</td>
</tr>
<tr>
<td></td>
<td>Stress ulcer</td>
<td>Infectious colitis</td>
</tr>
<tr>
<td></td>
<td>Esophageal varices</td>
<td>Henoch-Schonlein purpura</td>
</tr>
<tr>
<td></td>
<td>PUD</td>
<td>Hemolytic-uremic syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Polyps</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pseudomembranous colitis</td>
</tr>
</tbody>
</table>
Meckel's Diverticulum

- General
  - Most common congenital abnormality of the small intestine
  - Remnant of the omphalomesenteric duct that connected the embryo's gut to the yolk sac
  - Rule of 2's: found in 2% of the population, 45% of symptomatic patients are younger than 2 years of age, 2 cm wide, 2 cm long, and 2 feet from the ileocecal valve
  - Symptomatic patients are more likely to be male.

- Presentation
  - Isolated rectal bleeding is common in patients younger than 5 years of age; classic presentation is painless GI bleeding
  - Abdominal pain, distension, vomiting if obstruction has occurred
  - May mimic appendicitis
  - May be the cause of intussusception
  - Heterotopic gastric tissue may be present and result in bleeding from a peptic ulcer within the diverticulum or in the adjacent ileum

- Differential diagnosis
  - Peptic ulcer disease, polyps, tumors, intussusception, volvulus, appendicitis

- Diagnosis
  - Abdominal film may show signs of obstruction.
  - Meckel's scan: injection of technetium-pertechnetate IV; the test relies on the presence of gastric mucosa, which has an affinity for the radionucleotide; 95% accuracy rate for detecting the presence of gastric mucosa within the diverticula
  - Arteriography can detect the site of active bleeding.

- Treatment
  - Large-bore IV
  - NPO/NG tube
  - Transfusion may be necessary.
  - Start antibiotics if peritoneal signs are present.
  - Diverticulotomy versus more extensive resection if there is irreversible ischemia

Hirschsprung's Disease/Aganglionic Megacolon

- General
  - The absence of intramural ganglion cells in the rectum, extending to the sigmoid colon in 77% of patients and involving the entire colon in 15% of patients
  - 1/5,000 births; no ethnic predilection; male to female ratio is 4:1

- Presentation
  - Suspect the diagnosis if there is no passage of meconium stool in the newborn within the first 24 to 48 hours
  - Vomiting
  - Abdominal distension
  - Chronic constipation
  - Toxic megacolon
  - Do not do rectal examination: it interferes with the barium enema by giving a false negative
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• Diagnosis
  o Abdominal films: may show signs of obstruction
  o Barium enema: cone-shaped transition zone with dilated segment of proximal colon
  o Rectal manometry: shows paradoxic contraction of the internal anal sphincter
  o Rectal biopsy: definitive diagnosis; reveals the lack of ganglion cells in the rectal submucosa
• Treatment
  o IV hydration in the child with enterocolitis
  o Surgical repair: decompressing colostomy followed by closure at 1 year of age versus a one-step repair
    ▪ Complications of surgery
      □ Perineal abscess formation
      □ Enterocolitis in up to 9% of patients; symptoms are bloody stools, fever, abdominal distension, elevated WBC count

Colic/Formula Intolerance
• General
  o Classically defined as unexplained paroxysmal crying for more than 3 hours for more than 3 days in more than 3 weeks in an otherwise healthy child
  o Affects 10% to 30% of infants; typically occurs in the late afternoon and evening; usually starts in the third week of life and resolves by 3 months of age
• Differential Diagnosis
  o Do not assume the patient has colic and forget to look for other causes of crying: infection, obstruction, hernia, tourniquet syndrome, corneal abrasion, torsion, fracture, abuse
• Diagnosis
  o History is usually classic; physical examination is normal
  o This is a diagnosis of exclusion.
• Treatment
  o Reassure the parents that the infant is not medically sick.
  o The parent can try rocking the baby, going for a drive, swaddling the baby, or giving the baby a pacifier; make sure that the parents take a break from the crying; at times, formula changes can be tried as well as simethicone; the patient usually self-improves by 3 months of age.

5.2 RENAL/GENITOURINARY DISORDERS

Testicular Torsion
• General
  o Most involve "bell clapper" deformity, i.e., the testis lacks normal attachment to tunica vaginalis
  o Peak incidence, 13 years
  o Salvage rate: 80% to 100% within 6 hours, <10% after 24 hours
• Presentation
  o Abrupt onset scrotal pain; can be inguinal/abdominal pain
  o Nausea and vomiting
  o Absent cremasteric reflex; horizontal lie
• Diagnosis
  o Usually clinical
  o Urinalysis: pyuria does not rule out torsion
  o Ultrasound with color flow Doppler
Management
- Surgical exploration
- Manual detorsion as a temporizing measure may be attempted (opening book technique)

Paraphimosis
- Inability to reduce foreskin over glans penis
- Management
  - Circumferential compression of the glans and penis
  - Needle fluid aspiration of foreskin
  - Urologic consultation

Balanoposthitis
- Inflammation of glans penis (balano) and/or foreskin (posthitis), usually from infection
- Management: hygiene/sitz baths; 0.5% hydrocortisone cream sparingly; oral antibiotics if cellulitis; urologic referral
- Consider diabetes in patients with recurrent candidal balanoposthitis

Hemolytic Uremic Syndrome (HUS)
- General
  - Most common in infants and children younger than 5 years of age. Most commonly associated with verotoxin-producing \textit{E. coli} 0157:H7 (up to 10%)
- Presentation
  - Severe abdominal cramping
  - Watery diarrhea followed by grossly bloody stools
  - Emesis and symptoms of upper respiratory infection (URI) may be also be present; this stage is followed by acute renal failure, petechiae, GI bleeding, and CNS symptoms such as irritability, seizures, hemiparesis, or coma.
  - Hypertension is seen in 40% to 50% of patients.
- Diagnosis
  - CBC shows microangiopathic hemolytic anemia and thrombocytopenia (platelet count <50,000/mm$^3$).
- Treatment
  - Early peritoneal dialysis for severely affected patients, rehydration, treatment of hyperkalemia, transfusion of packed red blood cells, platelet transfusion for active bleeding or counts below 20,000/mm$^3$
  - HTN can be treated with nifedipine, labetolol, captopril, or hydralazine.
  - Treat seizures with benzodiazepines and phenytoin.

5.3 CARDIOVASCULAR DISORDERS

Congenital Heart Disease (CHD)
- General
  - 8 to 10 cases per 1000 live births
  - Neonatal circulation
    - First breath: pulmonary vascular resistance decreases, with resultant increase in pulmonary blood flow; peripheral vascular resistance continues to decrease and the right ventricle reaches adult pressures by day 10 of life; when the umbilical cord is clamped, systemic vascular resistance increases, resulting in an increase in left ventricular afterload and an increase in left atrial pressure; this normally leads to physiologic closure of the foramen ovale, ductus arteriosus, umbilical arteries, and umbilical vein
Patent ductus arteriosus (PDA) dependent lesions: usually have a sudden onset and present in the first week of life with cyanosis and shock when the ductus closes.

Categories
- Blue baby: cyanotic heart disease with right-to-left shunt
- Mottled baby: outflow tract obstruction with shock
- Pink baby: congestive heart failure with left-to-right shunt

Causes of cyanotic congenital heart disease (The “Terrible T’s”)
- Tetralogy of Fallot (6%-10%)
- Transposition of the great arteries (3%-5%)
- Tricuspid atresia (1%-2%)
- Truncus arteriosus (1%)
- Total anomalous pulmonary venous return (1%)
- Pulmonary atresia (<1%)
- Hypoplastic left heart (<1%)

Presentation
- Difficulty feeding, sweating with feeds, failure to thrive, tachypnea
- Cardiac cyanosis worsens with crying, whereas pulmonary cyanosis usually improves
- Sudden onset of lethargy, pallor, or central cyanosis
  - Central cyanosis: tongue, conjunctivae, and body are cyanotic
  - Peripheral cyanosis: tongue and conjunctivae are pink
- Tachypnea, retractions, grunting
- Poor perfusion
- Murmur is present in most cyanotic patients with CHD; single S2 is also common.
- Hepatomegaly may be seen.
- Weakened/absent femoral pulses – coarctation; check four extremity blood pressures and simultaneous pre- and post-ductal saturations (right hand and either foot)
- Hyperoxia test: 100% for 10 minutes, then ABG, PaO₂ <150 mm Hg or O₂ saturations <75% suggest cyanotic CHD; expect at least a 10% increase in the O₂ saturation if a pulmonary process is present

Treatment
- ABC’s: it may not be necessary to intubate, because the patient should not be brought up to 100% oxygen saturation; however, the use of PGE1 may require intubation because of the risk of apnea.
- If you suspect a ductal-dependent lesion, start PGE1 infusion (prostaglandin E1): 0.05 to 0.1 μg/kg/min IV; usually see improvement in 15 minutes; side effects are apnea, tachycardia, fevers, and hypotension
- If the patient has congestive heart failure, administer furosemide, 1 mg/kg; consider morphine, dobutamine, and dopamine
- In a child this ill, you cannot rule out sepsis; therefore, start antibiotics and send cultures.
- Admit to NICU or PICU

Kawasaki Disease (see Section 5.10, Rheumatologic Disorders)

ENDOCRINE/METABOLIC DISORDERS

Congenital Adrenal Hyperplasia
- General
  - 1/10,000 to 1/15,000 live births; higher in Eskimos (up to 1/300)
  - Deficiency in one of five enzymes involved in the production of cortisol
Most common is 21-hydroxylase deficiency, seen in 90% to 95%: simple virilizing (1/3) versus salt wasting (2/3)

- Presentation
  - Vomiting
  - Dehydration
  - Fever may be present.
  - Circulatory collapse during the first 2 weeks of life, unresponsive to IV fluids
  - Females may have enlarged clitoris and fusion of the labial folds
  - Males are more prone to missed diagnosis; may have a small phallus
  - Some children have hyperpigmentation due to the increased melanin production that is concurrent with increased ACTH level.
  - Hyponatremia, hyperkalemia, hypoglycemia
  - May see arrhythmias caused by hyperkalemia and acidosis
  - Patient may have seizures as a result of hypoglycemia.

- Treatment
  - Volume repletion with NS, then switch to maintenance fluid of D5 0.9% NS at 100 to 125 ml/kg/day
  - Replace cortisol with hydrocortisone, 25 mg IV, then 25 to 50 mg/m2/day, divided every 6 to 8 hours
  - Usually hyperkalemia responds to fluid replacement; however, severe hyperkalemia should be treated with 10% calcium gluconate (100 mg/kg); sodium bicarbonate, 1 mEq/kg; insulin, 0.1 unit/kg, with 10% dextrose, 2 to 4 ml/kg; or sodium polystyrene sulfonate
  - Monitor glucose; these patients may be hypoglycemic
  - Prior to administering hydrocortisone, draw a red-topped tube of blood for 17-hydroxyprogesterone, dehydroepiandosterone, androstenedione, testosterone

**Glucose Replacement**

- Normal blood glucose: >30 mg/dl in infants, >40 mg/dl in children
- Treatment of hypoglycemia: glucose, 0.25 to 1 gm/kg
  - Neonates: D10, 2 to 10 cc/kg (4 ml/kg = 0.5 gm/kg)
  - Infants and young children: D25, 2 to 4 ml/kg (2 ml/kg = 0.5 gm/kg)
  - Glucagon: 0.1 to 0.2 mg/kg IM if known insulin excess
  - D10 in newborns and small infants to avoid vein damage and intracranial hemorrhage

**5.5 NEUROLOGIC DISORDERS**

Seizures

- General
  - Definitions
    - Seizure: paroxysmal electrical discharge of neurons, resulting in either behavioral or motor change
    - Epilepsy: two or more unprovoked seizures not immediately preceded by fever, trauma, head injury, or chemical imbalance
    - Status epilepticus: classic definition is any seizure persisting more than 30 minutes or more than one seizure without return of consciousness; however, recent authors recommend that status epilepticus be redefined as any single seizure lasting longer than 5 minutes or any two seizures between which normal cognitive function is not regained
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- Incidence
  - 3.5% of all children will experience a seizure by age 15
  - 2% to 5% of children between the ages of 6 months and 5 years have febrile seizures
  - 1% of children develop epilepsy
    - 75% idiopathic
    - 5% CNS infection
    - 13% cerebral palsy
    - 3% head trauma

- Etiology (Table 5.5)

Table 5-5. Causes of Seizures

<table>
<thead>
<tr>
<th>Infections</th>
<th>Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meningitis/encephalitis</td>
<td>Epidural/subdural</td>
</tr>
<tr>
<td>Brain abscess</td>
<td>Intracerebral</td>
</tr>
<tr>
<td>Shigella gastroenteritis</td>
<td>Post-traumatic</td>
</tr>
<tr>
<td>Cysterciosis</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Inborn Errors of Metabolism</th>
<th>Tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological</td>
<td>Metabolic</td>
</tr>
<tr>
<td>Breath-holding spell</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Hypomagnesemia</td>
</tr>
<tr>
<td>Hypoxic/Ischemic</td>
<td>Hypophosphatemia</td>
</tr>
<tr>
<td></td>
<td>Hypocalcemia/hypercalcemia</td>
</tr>
</tbody>
</table>

Ingestions (PLASTIC – mnemonic for substances that can cause seizures)

<table>
<thead>
<tr>
<th>P</th>
<th>L</th>
<th>A</th>
<th>S</th>
<th>T</th>
<th>I</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propoxyphene</td>
<td>Lead</td>
<td>Antihistamine</td>
<td>Salicylates</td>
<td>TCAs</td>
<td>Insulin</td>
<td>Cocaine</td>
</tr>
<tr>
<td>PCP</td>
<td>Lithium</td>
<td>Anticholinergic</td>
<td>Strychnine</td>
<td>Tegretol</td>
<td>Inderal</td>
<td>Caffeine</td>
</tr>
<tr>
<td>Pesticides</td>
<td>Lidocaine</td>
<td>Antidepressant</td>
<td>Serotonin agonists</td>
<td>Theophylline</td>
<td>INH</td>
<td>Camphor</td>
</tr>
<tr>
<td>Phenothiazines</td>
<td>Lindane</td>
<td></td>
<td>Sympathomimetics</td>
<td></td>
<td>Industrial acids</td>
<td>CO</td>
</tr>
</tbody>
</table>


- Differential diagnosis
  - Syncope
  - Breath-holding spells
  - Migraines
  - Night terrors
  - Psychiatric disturbances/pseudoseizures

- Important history
  - Known epileptic versus first-time seizure
  - Change in seizure type, pattern, frequency, or duration
  - Medication history
  - Fever/stiff neck/rash
  - Drugs/alcohol
  - Head trauma
  - Sleep cycle history
  - Medical history: coagulopathy, diabetes, cardiac, immunosuppression
  - Last menstrual cycle
Status Epilepticus

• Treatment
  - ABC's
  - Measure glucose, chem 7, calcium, magnesium levels
  - Treat hypoglycemia
  - Treat hyponatremia <120 mEq/L with 5 ml/kg of 3% saline over 10 to 15 minutes
    - Should raise sodium by 5 mEq/L
  - Obtain drug levels, toxicology screen in cases of suspected ingestion
  - Medications
    - Lorazepam: onset, 1 to 2 minutes; duration of action up to 13 to 15 hours
      - Dose: 0.05 to 0.1 mg/kg IV; rectal dose, 0.5 mg/kg
    - Diazepam: onset, <1 minute; duration of action, 15 to 20 minutes
      - Dose: 0.1 to 0.3 mg/kg IV; rectal dose, 0.5 mg/kg
    - Midazolam: onset, 1 to 2 minutes, duration of action, 15 to 30 minutes
      - Dose: 0.1 mg/kg IV, 0.2 mg/kg IM
      - Drip: loading dose of 0.15 mg/kg then maintenance of 0.75 to 10 μg/kg/min
    - Phenytoin
      - Load 15 to 20 mg/kg IV, no faster than 1 mg/kg/min
      - Peak CNS levels in 10 to 30 minutes
    - Fosphenytoin
      - Load 15 to 20 mg/kg; can give 3 mg/kg/min
      - Peak IV level, 7 minutes; IM, 30 minutes
    - Phenobarbital
      - Load 15 to 20 mg/kg IV, no faster than 1 mg/kg/min
      - Peak CNS levels reached in 20 to 60 minutes
    - Pentobarbital
      - Load 10 to 20 mg/kg over 1 hour, then infuse 0.5 to 2 mg/kg/hr
    - Valproic acid
      - 10 to 30 mg/kg IV bolus over 15 minutes has been successful for both generalized tonic-clonic status and non-convulsive status epilepticus
  - Newborn seizures
    - Acyclovir (20 mg/kg per dose every 8 hours) if WBC count is increased and protein is elevated without organisms on CSF examination; xanthochromia; focal neurologic findings; or maternal history of herpes, vesicular rash, pneumonitis, or hepatitis
    - Consider calcium gluconate 10%, 100 to 300 mg/kg IV, if seizures are refractory.
    - Consider pyridoxine, 100 mg IV, if seizures are refractory.
  - Intubation and ventilatory support
  - Treatment pearls
    - Diazepam is considered a second-line drug after lorazepam (more respiratory depression, less efficacy in status with diazepam)
    - Anticonvulsant duration: diazepam <30 min, lorazepam >4 to 6 hours
    - Phenobarbital levels: 1 mg/kg raises level by approximately 1
Febrile Seizures

- General
  - Incidence, 2% to 5%
  - Risk increases to 10% to 30% with parent or sibling with a history of febrile seizures
  - Recurrence rate of 25% to 30%
    - 50% of recurrences occur within the first 6 months and 75% within the first year
- Simple Febrile Seizures
  - Brief duration of <15 minutes
  - Occurs only once in a 24-hour period
  - Generalized seizure without focal findings
  - No evidence of CNS infection
  - Age range: 6 months to 5 years
- Complex Febrile Seizures
  - Seizure duration > 15 minutes
  - Focal seizure
  - More than 1 seizure in a 24-hour period
- Risk Factors for Recurrence
  - Age <12 months with initial seizure
  - Lower temperature with initial seizure (<39°C)
  - Complex seizure
  - Parent or sibling with history of febrile seizures
- Diagnosis
  - No need for routine CT or MRI in cases of simple febrile seizures
  - Fever evaluation based on patient age: consider urinalysis (UA) and culture, CBC and blood culture, chest film, lumbar puncture (LP) as per standard evaluation for febrile illness
  - No need to routinely measure electrolyte or calcium and magnesium levels
  - ACEP recommendations regarding the performance of a lumbar puncture after a first febrile seizure:
    - LP should be strongly considered in infants <18 months of age with febrile seizure if any of the following exists:
      - History of irritability, decreased feeding, lethargy
      - Abnormal appearance or mental status in initial assessment of child after postictal state
      - Any physical examination signs of meningitis (bulging fontanelle, Kernig/Brudzinski signs, photophobia, severe headache)
      - Any complex features
      - Any slow postictal clearing of mentation
      - Pretreatment with antibiotics
    - If these factors are absent, then LP can be safely deferred.

Infant Botulism
- Presentation
  - Ptosis
  - Constipation
  - Loss of developmental milestones
  - "Floppy child"
• Treatment
  o Supportive
  o Trial of antitoxin, but this is usually not effective because of minimal amounts of circulating toxin
  o Antibiotics also are not effective, but if they are used, avoid aminoglycosides.

5.6 ORTHOPEDICS

Physeal Injuries
• Occurs at growth plate (physis)
• Up to one third of all pediatric fractures involve physis; usually upper extremities
• Peak incidence, 11 to 13 years of age
• Salter-Harris classification
  o Type 1 (6%): involves separation of metaphysis from the epiphysis through zone of provisional calcification; SH1 may be suspected on clinical grounds alone; may not be radiographically evident if the epiphysis is not displaced; visible radiographically only if the physis is widened or distorted or if the epiphysis is displaced
  o Type 2 (75%): most common; involves metaphysis; carries good prognosis
  o Type 3 (10%): epiphysis, intra-articular; anatomic position must be re-established to restore normal joint mechanics and prevent growth arrest or chronic disability (see Image #23)
  o Type 4 (10%): contiguous fracture through epiphysis, physis and metaphysis, intra-articular
  o Type 5 (1%): crush injury due to axial compression of germinal growth plate; difficult to see on radiograph, can confuse with Salter 1; base diagnosis on radiograph and if mechanism of injury suggests an axial compression along the long axis of the bone; can use comparison views if needed
• Distal radius is the most common site of injury (30%–60%)
• Ligaments tend to be stronger than physis: physeal separation/fracture is more common than sprain

Elbow Fractures
• Supracondylar most common (60%) (see Image #33)
• Posterior fat pad/large anterior fat pad alone is highly predictive of elbow fracture.
  o Assume nondisplaced (Type 1) supracondylar fracture in children; occult radial head fracture in adult
• Remember sequence of ossification centers:
  o CRITO: Capitellum, Radial head, Internal epicondyle, Trochlea, Olecranon, External epicondyle
    ▪ Generally seen at 2, 4, 6, 8, 10, and 12 years, respectively
    ▪ The ages at which these changes are seen radiographically are highly variable, but the sequence is always the same.

Slipped Capital Femoral Epiphysis
• General
  o Medial slip of the femoral epiphysis; associated with obesity and puberty
  o May present acutely as a result of trauma or can be chronic
  o Peak incidence is 12 to 16 years of age in boys and 10 to 14 years in girls
  o Bilateral in 10% to 25%
• Presentation
  o Pain in the groin, thigh, and knee; usually gradual but can be acute
  o Lower limb is held in external rotation; restricted full flexion
  o Limb shortening and proximal thigh muscle atrophy
Diagnosis
- Obtain AP and frog leg views of the hip: medial slip of the femoral epiphysis is seen on AP hip film; in the AP view, a line across the lateral (superior) aspect of the femoral neck should transect the lateral portion of the femoral epiphysis (Klein's line) (see Image #66)
- Treatment
  - Orthopedic consultation and admission; operative reduction and fixation to avoid avascular necrosis

Legg-Calve-Perthes Disease
- General
  - Avascular necrosis of the femoral head; incidence is 1/1,200 to 1/12,500 of children
  - 4 to 9 years of age in 80% (age range, 3–12 years); bilateral in 10% to 20%
    - Age presentation of Legg-Calve-Perthes disease is younger than for slipped capital femoral epiphysis
- Presentation
  - Limp and pain for weeks to months; pain is referred to the groin and knee; pain is relieved by rest; thigh muscle atrophy may also be seen
- Diagnosis
  - Radiograph of the hip shows widening of the cartilage space and smaller femoral head; increased opacification of the femoral head may also be seen; MRI and bone scan can also be helpful for diagnosis
- Treatment
  - Orthopedic consultation for traction and future therapy, including surgery

Osgood-Schlatter Disease
- Seen in early adolescence; boys are more commonly affected
- Repetitive injury from inflammation of the tibial tubercle apophysis; partial or complete avulsion of the tibial tubercle can occur
- Symptoms include localized swelling, pain, and tenderness over the tibial tubercle.
- Treatment: rest, NSAIDs, knee immobilization for 2 to 4 weeks for severe symptoms; avulsion may require surgery

Transient Synovitis of the Hip
- General
  - Peak age: 3 to 6 years of age; right hip is more commonly affected than the left
- Presentation
  - Symptoms may be gradual or acute
  - Preceding viral syndrome (URI)
  - Fever, usually low grade
  - Pain on palpation of the anterior hip
  - Decreased range of hip motion
- Diagnosis
  - Diagnosis of exclusion
  - WBC count and ESR/CRP are usually normal; if elevated, consider joint aspiration to rule out septic arthritis
  - Hip radiographs may show a mild hip effusion
- Treatment
  - NSAIDs and close follow-up
5.7 EARS, NOSE, AND THROAT

Acute Otitis Media
- General
  - Definition
    - Acute onset of symptoms
    - Presence of middle ear effusion, as indicated by one of the following:
      - Bulging tympanic membrane (TM)
      - Limited/absent TM mobility
      - Air/fluid level behind TM
      - Otorrhea
    - Signs/symptoms of middle ear inflammation
      - Distinct TM erythema or
      - Distinct otalgia
  - Treatment
      - Observation without antibiotics is an option if
        - Age > 2 years
        - Age 6 months to 2 years with uncertain diagnosis and nonsevere symptoms (T < 39.0°C, mild otalgia)
      - Observation for 48 to 72 hours is an option only if follow-up is assured
      - If the above criteria are not met, then treat with antibiotics.
      - Initial antibiotics
        - Amoxicillin, 80 to 90 mg/kg/day in most cases
        - Cefdinir, cefuroxime, cefpodoxime, and macrolides are alternatives
        - Ceftriaxone, 50 mg/kg x 1 dose
      - Treatment failures
        - Amoxicillin/clavulanate
        - Clindamycin and ceftriaxone x 3 doses are alternatives

Epiglottitis
- General
  - Etiology
    - *H. influenza* type B incidence is decreasing due to vaccine
    - Reported cases of *S. pneumonia*, *S. aureus*, and group A β-hemolytic streptococci
  - Wide age range: newborns to adults; average pediatric age, 3 to 7 years
- Presentation
  - Several hours of fever and sore throat with rapid progression
  - Irritability, lethargy, drooling
  - Viral prodrome is usually absent
  - Dysphagia
  - Severe stridor is usually absent
  - Tripod/sniffing position
• Diagnosis
  o Usually clinically suspected
  o Lateral neck radiograph
    ▪ Classic “thumb” sign (see Image #1)
    ▪ Do not send patient out of department for films
  o Confirmed by direct visualization in the operating room
  o 70% to 90% of blood cultures yield the offending organism
  o WBC count will be elevated with bandemia.

• Treatment
  o Keep patient in position of comfort
  o Mobilize OR team
  o Start IV in OR
  o If emergent intubation is required, use 0.5- to 1.0-mm smaller endotracheal tube than predicted.
  o Third-generation cephalosporins are antibiotics of choice.
  o Steroids are not indicated.

Croup (Laryngotracheobronchitis)

• General
  o Parainfluenza type 1 is the most common cause; also RSV, adenovirus, influenza A
  o 6 months to 6 years; peak incidence at 2 years

• Presentation
  o 1- to 3-day history of URI
  o Hoarse voice and barking cough
  o Fever
  o Stridor: in severe cases can be inspiratory and expiratory

• Diagnosis
  o Radiographic confirmation (rarely needed, consider to exclude other causes)
    ▪ Lateral neck film findings
      ▪ Hypopharyngeal overdistension
      ▪ Subglottic narrowing
      ▪ Normal epiglottis
    ▪ Frontal (AP) neck film findings
      ▪ Classic “steeple sign” in the subglottic region

• Treatment
  o Cool mist humidification is without proven benefit but has little downside.
  o Racemic epinephrine via nebulizer is indicated in moderate/severe croup
    ▪ 0.25 cc of a 2.25% solution in 2 ml NS for patients <6 months
    ▪ 0.5 cc of a 2.25% solution in 2 ml NS in older patients
    ▪ Observation for 2 to 4 hours after administration
    ▪ Steroids should be given to all patients who are given racemic epinephrine.
  o Dexamethasone should be administered to all patients with croup, including those with mild disease.
    ▪ 25 times more potent than hydrocortisone
    ▪ Dose: 0.15 to 0.6 mg/kg IM, PO, IV (max 16 mg)
    ▪ Long biologic half-life (up to 54 hours), so a single dose is usually sufficient
o Admission criteria
  • Stridor at rest, despite above interventions
  • Incomplete response to racemic epinephrine
  • Multiple doses of racemic epinephrine are required.
  • Persistent respiratory distress
  • Dehydration
  • Poor social situation

**Bacterial Tracheitis**

- **General**
  - Etiology: *S. aureus* is most common; also *S. pneumonia*, group A β-hemolytic streptococci, *H. influenza, M. catarrhalis*, mixed flora with anaerobes

- **Presentation**
  - Presents in two ways:
    - Initial mild/moderate croup-like symptoms for days followed by sudden worsening
    - Previously well with acute onset of symptoms and airway obstruction
  - Preceding URI
  - High fever
  - Stridor
  - Retractions, wheezing may be present
  - Dysphagia
  - No improvement or deterioration with standard croup interventions

- **Diagnosis**
  - Clinical suspicion: child appears toxic
  - Radiographs: subglottic narrowing similar to croup
  - Blood cultures usually not positive, but positive tracheal aspirate

- **Treatment**
  - Emergent intubation is usually necessary; management similar to that for epiglottitis
  - IV antibiotics: nafcillin, 100 to 150 mg/kg/day divided q6h + ceftriaxone, 50 mg/kg/day, until cultures provide more information on susceptibility

**Gingivostomatitis**

- **General**
  - Etiology: herpes simplex virus (HSV), Coxsackie virus, *Candida albicans*
    - Vesicles and ulcerations are more common in viral causes.
    - Involvement of the anterior portion of the mouth and lip is more common with HSV.
    - Only soft palate/tonsillar pillar involvement suggests Coxsackie virus (inspect the palms and soles for hand-foot-mouth disease)
    - White plaques indicate *Candida*

- **Presentation**
  - Fever (usually present before the oral lesions, and patients often are evaluated as “fever without a source”)
  - Pain
  - Decreased oral intake
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- Treatment
  - Supportive with appropriate pain control
    - “Magic mouthwash” (diphenhydramine, aluminum/magnesium hydroxide, lidocaine 1%) or equivalent; be careful about sending parents home with too much solution, due to possible lidocaine toxicity
  - Nystatin suspension or fluconazole if treating Candida
  - Dehydrated patients may require in-house therapy with IV fluids.

5.8 RESPIRATORY DISORDERS

Pneumonia
- Causes vary by age (Table 5-6)

Table 5-6. Causes of Pneumonia

<table>
<thead>
<tr>
<th></th>
<th>Bacterial</th>
<th>Viral</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>Group B streptococci</td>
<td>Respiratory syncytial virus</td>
<td>Chlamydia</td>
</tr>
<tr>
<td></td>
<td>E. coli</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Listeria</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pseudomonas</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Klebsiella</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 weeks–3 months</td>
<td><em>S. pneumonia</em></td>
<td>Respiratory syncytial virus</td>
<td>Chlamydia</td>
</tr>
<tr>
<td></td>
<td><em>S. aureus</em></td>
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<td></td>
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<tr>
<td></td>
<td>Pertussis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Group A/B streptococci</td>
<td>Parainfluenza</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>H. influenzae</em> (type/non-type)</td>
<td>Adenovirus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Influenza</td>
<td></td>
</tr>
<tr>
<td>4 months–4 years</td>
<td><em>S. pneumonia</em></td>
<td>Respiratory syncytial virus</td>
<td>Mycoplasma</td>
</tr>
<tr>
<td></td>
<td><em>S. aureus</em></td>
<td></td>
<td></td>
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<td><em>H. influenzae</em> (type/non-type)</td>
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<tr>
<td></td>
<td>Group A streptococci</td>
<td>Adenovirus</td>
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</tr>
<tr>
<td></td>
<td>Pertussis</td>
<td>Influenza</td>
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<td></td>
<td></td>
<td>Enterovirus</td>
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<td></td>
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<td>Rhinovirus</td>
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<td>≥5 years</td>
<td><em>S. pneumonia</em></td>
<td>Parainfluenza</td>
<td>Mycoplasma</td>
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<td></td>
<td><em>H. influenzae</em></td>
<td></td>
<td>Chlamydia pneumoniae</td>
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<tr>
<td></td>
<td>Group A streptococci</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- 60% to 90% viral overall
- Presentation
  - Tachypnea
    - Most reliable sign in children
  - Cough
  - Grunting, flaring, retractions
  - Fevers
  - Vomiting, poor feeding, irritability, lethargy in infant/toddlers
- Treatment
  - Age less than 3 months: admission with IV ampicillin and cefotaxime (also consider erythromycin for afebrile pneumonias due to Chlamydia or suspected pertussis)
  - 3 months to 4 years: amoxicillin, 80 to 100 mg/kg/day PO, or cefuroxime, amoxicillin/clavulanate, macrolide, trimethoprim-sulfamethoxazole; IV medications include cefuroxime, cefotaxime, and ceftriaxone
Children ≥5 years of age: macrolide (azithromycin, erythromycin, clarithromycin) to cover for *Mycoplasma pneumoniae*; consider doxycycline if >8 years of age. Admit all patients with suspected *S. aureus* pneumonia.

**Chlamydial pneumonia**
- Young infants, usually afebrile
- Classic cough is staccato like and may be associated with post-tussive emesis
- Up to half of patients have concurrent or had previous eye infection with *Chlamydia*.
- Hyperinflation or increased interstitial markings on chest film
- Treatment is with oral erythromycin or sulfonamide.

**Pertussis**
- General
  - *Bordetella pertussis*: gram-negative organism
  - Transmitted by respiratory droplets
  - Incubation period: 6 to 20 days
  - Stages (Table 5-7)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Duration</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catarrhal</td>
<td>1–2 weeks</td>
<td>Lacrimation, rhinorrhea, mild cough, low-grade fever to 101°F</td>
</tr>
<tr>
<td>Paroxysmal</td>
<td>2–4 weeks</td>
<td>Paroxysmal cough, apnea, cyanosis, emesis, neurologic and respiratory symptoms</td>
</tr>
<tr>
<td>Convalescent</td>
<td>1–4 weeks</td>
<td>Cough may persist up to 6 months</td>
</tr>
</tbody>
</table>

**Diagnosis**
- WBC count >15,000 with lymphocytosis
- Fluorescent antibody staining
- Nasopharyngeal culture

**Treatment**
- IV hydration may be necessary
- Supplemental oxygen
- Erythromycin, 50 mg/kg/day, eliminates organisms from nasopharynx in 3 or 4 days
- May use TMP/SMX, 8 mg/kg/day, if erythromycin is not tolerated
- Antibiotic therapy does not shorten the paroxysmal stage.
- Treat household contacts with a 14-day course of erythromycin.
- Admission criteria
  - History of apnea
  - Inability to tolerate fluids
  - Poor social situation or unreliable parents
  - Age <6 months, history of prematurity
  - Persistent O₂ requirement
Asthma

- General
  - Mucosal edema, bronchospasm, increased secretions
  - Cough, wheezing, dyspnea
  - Three categories
    - Symptoms in the presence of URI
    - Continued wheezing after 3 years of age
    - Those associated with atopic disease
- Differential diagnosis of wheezing
  - Asthma, bronchiolitis, pneumonia, CHF, PE, anaphylaxis/allergic reaction, cystic fibrosis, laryngotracheomalacia, tracheoesophageal fistula, bronchopulmonary dysplasia, mediastinal masses, vascular anomalies, foreign body aspiration
- Treatment
  - ABCs
  - Beta agonists are the cornerstone of management
    - Albuterol
    - Levalbuterol gives no significant advantage over standard albuterol
  - Epinephrine, subcutaneous, 0.01 ml/kg of 1:1000 concentration (max 0.5 ml)
    - May use IV if in extremis: 0.1 ml/kg of 1:10,000 concentration; use 1/10th to 1/3rd of code dose
  - Steroids: 2 mg/kg/day of prednisolone, prednisone, methylprednisolone
    - Decadron (0.6 mg/kg), single-dose or two-dose therapy has also been effective
  - Anticholinergics: ipratropium bromide
  - Magnesium sulfate: 50 to 75 mg/kg up to 2 grams over 20 to 30 minutes
  - Intubation: use ketamine for sedation, 2 mg/kg (reduces bronchospasm)
- Admission criteria
  - Persistent respiratory distress
  - Hypoxia
  - Poor response to treatment
  - Concurrent pneumonia
  - Multiple visits for same episode
  - Poor parental compliance or poor social situation
  - Inability to tolerate fluids
  - Hypercapnia or normal CO₂ on blood gas indicates distress
  - Low threshold for patients with history of intubation

5.9 BACTEREMIA AND SEPSIS

- General
  - Common pathogens by age
    - Neonates
      - Group B streptococci, Escherichia coli, Listeria monocytogenes, Enterococcus species
    - 30 to 90 days
      - As above, also Streptococcus pneumoniae, Neisseria meningitidis, hemophilus influenza B, group A streptococci, Salmonella species, Staphylococcus aureus
• 3 to 36 months
  □ *Streptococcus pneumoniae*, *Neisseria meningitidis*, hemophilus influenza B, group A streptococci, *Escherichia coli*, *Salmonella* species, *Staphylococcus aureus*

• Fever without source
  o Fever definition
    ▫ ≥38.0°C (100.4°F) rectal if <3 months
    ▫ ≥39.0°C (102.2°F) rectal if ≥3 months
  o Serious bacterial infection (SBI)
    ▫ Bacteremia
    ▫ Bacterial meningitis, pneumonias, gastroenteritis
    ▫ Urinary tract infection
      □ 7.5% rate in febrile infants <2 months
    ▫ Cellulitis
    ▫ Osteomyelitis
    ▫ Septic arthritis
  o Under 3 months of age
    ▫ SBI up to 7%
      □ Bacteremia or bacterial meningitis, 2% to 3%
    ▫ Up to 13% under 29 days old
    ▫ Otitis media not considered source at this age
  o 3 to 36 months of age
    ▫ Before *Haemophilus influenzae* type b (HIB) immunization, the SBI prevalence is up to 5%
    ▫ After HIB immunization, SBI prevalence <2%
    ▫ Post-Pneumovax <1%
  o Occult pneumococcal bacteremia
    ▫ 10% to 25% complication rate, of which 3% to 6% develop meningitis
### Evaluation based on age (Table 5-8)

#### Table 5-8. Evaluation of Children with Fever

<table>
<thead>
<tr>
<th>Age</th>
<th>Temperature</th>
<th>Work-Up</th>
<th>ED Treatment</th>
<th>Disposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–28 days</td>
<td>≥38.0 R</td>
<td>Full septic W/U</td>
<td>Ampicillin and cefotaxime ± acyclovir*</td>
<td>Admit</td>
</tr>
<tr>
<td>28 days–2 months</td>
<td>≥38.0 R</td>
<td>Full septic W/U</td>
<td>Ceftriaxone</td>
<td>Admit or D/C if low risk</td>
</tr>
<tr>
<td>2–3 months</td>
<td>≥38.0 R</td>
<td>CBC, Bl Cx, UA, Urine Cx ± LP, CXR, and stool ex</td>
<td>± Ceftriaxone</td>
<td>Next-day follow-up</td>
</tr>
<tr>
<td>3 months–3 years</td>
<td>&gt;39.0 R</td>
<td>UA and Ur Cx in girls &lt;2 years old or Ur Cx in boys &lt;6 months old or &lt;12 months if uncircumcised</td>
<td>± Ceftriaxone</td>
<td>D/C if W/U is negative</td>
</tr>
<tr>
<td></td>
<td>and without source</td>
<td></td>
<td></td>
<td>if no LP</td>
</tr>
</tbody>
</table>

*Consider acyclovir, 20 mg/kg per dose q8h, if the patient has CSF pleocytosis or elevated protein, vesicular lesions, focal neurologic findings/seizures, hepatitis, pneumonia, or a maternal history of herpes.

R, rectal temperature (°F); W/U, workup; CBC, complete blood count; Bl Cx, blood culture; UA, urinalysis; LP, lumbar puncture; CXR, chest x-ray film; stool cx, stool culture; D/C, discharge; Ur Cx, urine culture.

- Lab screening and empiric antibiotics may not be necessary in the highly febrile, well-appearing 6- to 36-month-old child who has received the primary pneumococcal conjugate vaccine series.
  - CBC and blood cultures are being de-emphasized.

### 5.10 RHEUMATOLOGIC DISORDERS

#### Kawasaki Disease

- **General**
  - Also called mucocutaneous lymph node syndrome
  - A generalized vasculitis of unclear etiology, which involves the coronary arteries and the small- to medium-sized arteries; toxins from *S. aureus* and *Streptococcus pyogenes* may produce superantigen toxins
  - One of the most common childhood vasculitides: up to 5,000 children are affected annually in the United States; male-to-female ratio is 1.5:1; peak age is 1 to 2 years of age; 80% of patients present before 4 years of age
  - Most common cause of acquired heart disease in the United States

- **Presentation**
  - Fever for at least 5 days plus at least four of the following:
    - Bilateral non-exudative conjunctivitis
    - Changes of the lips and oral mucosa (fissured lips, strawberry tongue)
    - Changes in the extremities (erythema of the palms and soles, edema, periungual desquamation)
    - Polymorphous rash
    - Cervical adenopathy (1.5 cm in diameter or greater)
Incomplete Kawasaki disease, with only two or three of the features listed above, requires close follow-up and possible treatment.

Acute phase lasts 7 to 14 days and is followed by the subacute phase (2–4 weeks).

- **Complications**
  - Coronary artery aneurysms
    - 15% to 25% without treatment
    - Up to 4% despite treatment
    - More common in infants, because they can present with fever only and none of the classic features
  - MI, CHF, peripheral artery occlusion, hydrops of gallbladder, arthritis, aseptic meningitis

- **Diagnosis**
  - Criteria above; ESR/CRP and WBC count are usually elevated, sterile pyuria (due to urethritis), elevated liver enzymes, thrombocytosis after 1 week; check ECG and echocardiogram

- **Treatment**
  - ASA, 80 to 100 mg/kg/day until day 14, then 3 to 5 mg/kg/day until platelet count returns to normal
  - Single infusion of IVIG, 2 g/kg over 10 hours, is effective

**Henoch-Schönlein Purpura**

- **General**
  - IgA-mediated vasculitis involving the small vessels of the skin, GI and renal tracts, and musculoskeletal system
  - Most common acute vasculitis in children

- **Presentation:** “ARENA”
  - A = Abdominal pain (bloody stools, intussusception)
  - R = Rash, purpuric, classically “palpable purpura” (see Image #14)
    - Rash of some form occurs in all cases.
    - Presenting symptom in >50%
    - Starts in lower extremities – ankles/feet and buttocks
      - Face, trunk, palms/soles are usually spared
    - Typically no itching
  - E = Edema
  - N = Nephritis
  - A = Arthralgias

- **Diagnostics**
  - No pathognomonic tests
  - Hemoglobin, platelets usually normal
  - ESR normal or mild elevation
  - WBC count frequently elevated
  - Positive anti-streptolysin-O (ASO) or group A β-hemolytic streptococcal (GABHS) throat culture is common
  - Elevated BUN/Cr or significant hematuria → glomerulonephritis

- **Treatment**
  - Arthralgias can be treated with NSAIDs
  - Renal/GI or joint involvement sometimes treated with steroids and IVIG
  - Admit patients with severe symptoms, hypertension, or renal involvement.
5.11 SKIN AND SOFT TISSUE INFECTIONS

Orbital Cellulitis
- General
  o Infection of the orbit is considered an ophthalmologic emergency.
  o Most common cause is bacteria spread by direct extension from the sinuses (*S. pneumonia, H. influenza, M. catarrhalis*, anaerobes) or from the skin (*Staph* and *Strep*).
- Presentation
  o Decrease in visual acuity, globe movement, and proptosis are the hallmark symptoms.
  o Fever, swollen eye lid, red eyes
  o Patient usually appears ill.
- Diagnosis
  o CBC and blood culture
  o Orbital CT
- Treatment/disposition
  o Admit for IV antibiotics (third-generation cephalosporin ± vancomycin or nafcillin ± clindamycin).

Periorbital Cellulitis
- General
  o May develop after a break in the skin from trauma, insect bites, or chickenpox.
  o *H. influenza* is the causative agent in unimmunized patients.
  o *Strep* and *Staph* are more common in immunized patients.
- Presentation
  o Swelling and erythema of the periorbital region
  o Eye movement is not affected
  o No proptosis
  o Warmth
- Diagnosis
  o Patients with marked swelling may need CT.
- Treatment
  o IV antibiotics, as for orbital cellulitis.
  o Older children who have no signs of toxicity may be managed on an outpatient basis if good follow-up is assured.

Impetigo (see Image #74)
- Superficial bacterial infection of the skin due to *S. aureus* and group A streptococci; usually no associated systemic symptoms except local lymphadenopathy.
- Erythematous papules with transient small vesicles.
- The hallmark is honey-crusted lesions, the characteristic site being between the upper lip and nose.
- Glomerulonephritis is rare.
- Topical antibiotics (mupirocin) or an oral course of either cephalaxin, penicillin, or erythromycin; combination therapy is not necessary.
Erythema Infectiosum (Fifth Disease)

- **General**
  - Caused by Parvovirus B19
  - Usually respiratory transmission; also maternal-fetal
  - Incubation period is 4 to 20 days; patients are contagious for few days before and after rash

- **Presentation**
  - Low-grade fever in 15% to 30% of patients
  - Classic rash
    - Slapped cheeks on face
    - Lacy rash on arms, trunk
    - Rash recurs with heat, sunlight
    - Adults and teens can develop arthralgia and arthritis

- **Complications**: aplastic crisis in hemolytic disease (sickle cell), fetal hydrops in pregnancy

- **Treatment**: supportive, isolate pregnant women

Roseola (Exanthem Subitum)

- **General**
  - Caused by human herpes virus 6
  - Incubation, 5 to 15 days
  - Common infection in children 6 months to 2 years of age

- **Presentation**
  - High fever for 3 to 5 days
  - Febrile seizures are possible.
  - Maculopapular rash develops after defervescence.

- **Treatment**
  - Antipyretics

Varicella

- **General**
  - Macules, papules, and vesicles that develop and spread over 24 hours (see Image #76)
  - Rash starts on trunk, then to face and extremities
  - Highly contagious until crusted

- **Complications**
  - Cellulitis
  - Pneumonia
  - Encephalitis: seizures, coma (early)
    - Cerebellitis: benign ataxia (late)
    - Reye syndrome (especially in association with aspirin use)

- **Treatment**
  - Antipruritics
  - Antipyretics: use acetaminophen
  - Consider acyclovir (20 mg/kg/dose; max 800 mg/dose) QID for 5 days; must be started within 24 hours after onset of symptoms, which is often not possible because the rash starts after the prodrome; should be given to children at risk if exact exposure has been documented
Varicella zoster immune globulin (VZIG) for children at high risk for development of severe disease; must be given within 96 hours after exposure (48 hours preferred); dose is 125 U/10 kg IM, max 625 U

Prevention with the varicella vaccine, given between 12 and 18 months of age
  - After 13 years, two doses are needed to be effective.

Scarlet Fever
- Commonly associated with group A β-hemolytic streptococci
- Presentation
  - Sand paper rash first noted in skin folds such as the axillae, groin, and antecubital areas (Pastia's lines)
  - Circumoral pallor
  - Rash usually develops 12 to 48 hours after the onset of sore throat, fever, and chills; may be accentuated by heat; and can last 4 or 5 days
  - Desquamation occurs over the next 2 weeks, especially on the hands and feet
- Treatment
  - Penicillin VK (25–50 mg/kg/day QID) or erythromycin (20–50 mg/kg/day TID or QID)
  - No school until 24 hours after starting the antibiotics

Staphylococcal Scalded Skin Syndrome
- Usually seen in children younger than 5 years of age
- Presentation
  - Irritability when skin is touched
  - Fever
  - Generalized skin erythema followed by bullae formation and skin desquamation (see image #17)
  - The skin can be rubbed off in layers with lateral pressure – Nikolsky's sign
  - Mucous membranes are not involved
- Treatment
  - Aggressive IV hydration
  - All but the most mildly affected children should be admitted for IV cefazolin or nafcillin.
  - All newborns should be admitted, even if symptoms are minimal.

Candida
- Oral candidiasis
  - Causes inflammation of the tongue, palate, and buccal mucosa
  - White plaques cannot be wiped off the mucous membranes.
  - Rare in newborns; more common in children over 2 months of age
  - Treat with oral nystatin suspension (100,000 units/ml), 1 ml in each side of the mouth QID
- Cutaneous candidiasis
  - Erupts in the moist, warm areas of the body, axillae, neck folds, and diaper area
  - Satellite lesions are common along the edge of the eruption
  - Treatment is with topical antifungal agents (nystatin, clotrimazole); if possible, avoid combination preparations that have steroids, as they can cause thinning of the skin.
Herpangina (see Image #48)
- Vesicular stomatitis in the posterior pharynx
- High fever and sore throat, with drooling
- Recover in 4 to 6 days
- Symptomatic therapy

Hand-foot-mouth disease
- Coxsackie virus
- Typically in children younger than 10 years of age
- Fever
- Vesicular rash in mouth, hands, and feet (palms and soles are included)
- Treatment is symptomatic.

Tinea Capitus
- *Trichophyton tonsurans* (most common, 90%) transmitted from person to person via fomites (e.g., barber's razor)
- Presentation
  - Alopecia
  - “Black dot” appearance on scalp
  - Kerion (swollen boggy abscess of scalp)
- Differential diagnosis
  - Alopecia areata
  - Trichotillomania
  - Traction alopecia
- Diagnosis
  - Clinical or, if unclear, may send culture of scraping from hair roots
- Treatment: must be oral
  - Griseofulvin, 20 mg/kg/day for 6 weeks; given with a fatty meal
  - Selenium sulfide shampoo should be used twice a week for the first 2 weeks of oral treatment to prevent the spread of spores.
  - Prednisone may be added at 1 mg/kg/d for 5 days in the treatment of a severe kerion.
  - Discharge home with follow-up with primary care physician at the end of treatment.
    - Routine liver function tests do not need to be done for treatment with griseofulvin.

5.12 PSYCHIATRIC DISORDERS

Abuse
- General
  - Child abuse is broadly defined as maltreatment of a child by parents, guardians, or other caregivers.
  - May take the form of physical, sexual, or emotional abuse
  - Denial of nutrition, medical care, or a safe environment can also be considered abuse.
• Physical abuse
  o Red flags
    ▪ Child claims to have been injured.
    ▪ No history at all is offered.
    ▪ History of inflicted injury
    ▪ History changes over time or different caretakers give different histories
    ▪ Serious injury is blamed on another child
    ▪ Child is developmentally incapable of acting as described
    ▪ History provided is inconsistent with injuries suffered
    ▪ Delay in seeking medical care
  o Clinical indicators
    ▪ A lack of physical findings does not exclude abuse.
    ▪ Lethargy, poor feeding, colic, bulging fontanelle, apnea, seizures
    ▪ Retinal hemorrhages beyond the neonatal period
    ▪ Injury does not match history
    ▪ Multiple injuries of various types and ages
    ▪ Pathognomonic injuries: loop marks, cigarette burns, immersion burns, fractures of posterior ribs, metaphyseal or “bucket handle” fractures, spiral femur fractures in non-weight bearing infants, retinal hemorrhages
  o Characteristic fractures of abuse
    ▪ High specificity
      □ Fractures of metaphyseal corner (“bucket-handle”), posterior rib, sternum, long-bone shaft in non-weight-bearing age
    ▪ Medium specificity
      □ Complex skull fractures, vertebral body fractures, multiple fractures of different ages
    ▪ Low specificity
      □ Long-bone shaft fracture in weight-bearing age, linear skull fracture
  o Shaken baby syndrome
    ▪ Subdural hematoma
    ▪ Retinal hemorrhage
    ▪ Long-bone fracture
    ▪ Minimal signs of external trauma
  o Management of suspected physical abuse
    ▪ Laboratory studies
      □ Platelet count, PT, PTT, if contusions or hematomas are present
      □ Other labs as indicated
    ▪ Radiographic studies
      □ Complete skeletal survey indicated for children <2 years of age with evidence of abuse
      □ Other x-ray films as clinically indicated
    ▪ Call the Child Protection Team.
• Sexual abuse
  o More commonly committed by family or household members than by strangers
  o Historical components suggestive of sexual abuse
    ▪ Inappropriate knowledge of adult sexual behavior
    ▪ Compulsive masturbation
    ▪ Excessive sexual curiosity
    ▪ Sleep disturbances
    ▪ Aggressive behavior
    ▪ Running away
    ▪ Suicide attempt
    ▪ Abrupt behavioral change
    ▪ Diminished school performance
    ▪ Abdominal pain
    ▪ Sexually provocative behavior and promiscuity
  o Physical exam findings suggestive of sexual abuse
    ▪ Genital injury
    ▪ Rectal injury
    ▪ Vaginal/urethral discharge
    ▪ Vaginal/rectal pain or bleeding
    ▪ Pregnancy
    ▪ Evidence of physical abuse
    ▪ Sexually transmitted diseases (gonorrhea, syphilis, chlamydia) are definitive of sexual abuse in children and infants
  o Management
    ▪ Laboratory studies
      □ Cultures for *Neisseria gonorrhea* from the oropharynx, rectum, vagina/cervix should be obtained for girls and from the oropharynx, rectum, and urethra for boys as indicated; DNA probes are not acceptable.
      □ *Chlamydia* cultures should be obtained from the vagina/cervix or urethra; DNA probes are not acceptable.
      □ Urinalysis and pregnancy tests should be obtained as indicated.
      □ A vaginal wet mount for *Trichomonas* is indicated for vaginal discharge.
    ▪ Unless medically indicated, the physician should not perform a pelvic exam; instead, the genital exam and cultures can be deferred to the examiner from the Child Protection Team.
    ▪ Call the Child Protection Team.

5.13 SUDDEN INFANT DEATH SYNDROME/APARENT LIFE-THREATENING EVENT

Definitions
• SIDS: sudden death of an infant younger than 1 year of age, which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of death scene, and review of the clinical history
• Apnea: absence of respirations for 20 seconds or any length of time if associated with a decrease in heart rate, hypotonia, or change in color (pallor or cyanosis)
Apparent life-threatening event (ALTE): characterized by apnea, color change (cyanosis or pallor), marked change in muscle tone, choking or gagging
- Usual age is 2 to 3 months, but can occur at any time
- Unknown association with SIDS
- Differential: seizure, gastroesophageal reflux, bronchiolitis, pertussis, choking, URI, head injury, breath holding

Evaluation
- CBC
- Electrolytes
- Blood, urine, stool, and CSF cultures and respiratory syncytial virus testing in infants born prematurely and patients with congenital heart disease
- Consider a lumbar puncture if <2 months of age
- Pertussis and Chlamydia cultures should be obtained if clinically suspected
- Chest radiographs and, if upper airway obstruction is suspected, anteroposterior and lateral soft-tissue radiographs of the neck
- ECG
- Tox screen
- Head CT should be obtained in patients with an altered level of consciousness, abnormal muscle tone, focal neurologic findings, or retinal hemorrhages.

Disposition
- Admit all children who meet the criteria for an ALTE, despite a normal workup and physical examination

**5.14 PEDIATRIC RESUSCITATION**

**CPR**
- Two-person CPR, 30:2 compressions to ventilation
- Health care provider or trained individual, 15:2
- Once an advanced airway is placed, do not give more than 8 to 10 rescue breaths per minute.
- Avoid cessation of compressions.

**Fluids**
- Newborns: 10 ml/kg NS
- Older children: 20 ml/kg NS
- 10 ml/kg PRBCs

**Drugs**
- Epinephrine: 0.1ml/kg of 1:10,000 (or 0.01 mg/kg) IV
  - Use the 1:1,000 form for ETT drug dosing (except neonates)
  - PALS has “de-emphasized” high-dose epinephrine IV
- Atropine: 0.02 mg/kg, minimum dose is 0.1 mg
  - The only PALS indication is symptomatic bradycardia
  - Not used for neonatal resuscitation
- Lidocaine: 1 mg/kg
- Adenosine for SVT: 0.1 mg/kg (maximum 6 mg), repeat dose 0.2 mg/kg (maximum 12 mg)
  - Must be given as centrally as possible, since adenosine has a very short half-life
• Amiodarone: 5 mg/kg bolus for pulseless VT and VF; 5 mg/kg over 20 to 60 minutes for VT with pulse or SVT
• Cardioversion: start with 0.5-1 J/kg for unstable SVT or VT with a pulse; 2 J/kg for subsequent doses
• Defibrillation: 2 J/kg followed by 2 minutes or 5 cycles of CPR; if no response, the subsequent voltage is 4 J/kg for VF and pulseless VT
  o Shocks must no longer be stacked, if there is no change in rhythm, then proceed to epinephrine, start IV, intubate, and initiate CPR; alternate epinephrine and amiodarone; consider lidocaine; if there a concern about torsades de pointes, give magnesium

Supraventricular Tachycardia (SVT)
• Stable
  o Vagal maneuvers
    □ Apply ice to face in infants
    □ Blowing through occluded straw in children
  o Adenosine: 0.1 mg/kg rapid IV push (max 6 mg)
    □ Double the dose if the first is not effective (max 12 mg)
  o Consider alternative medications
    □ Amiodarone, 5mg/kg over 20 to 60 minutes, or
    □ Procainamide, 15 mg/kg over 30 to 60 minutes
  o Verapamil should not be used in infants.
• Unstable
  o Cardioversion: 0.5 to 1 J/kg first dose, 2 J/kg subsequent doses

Tachycardia with Poor Perfusion Algorithm
• Narrow complex (≤0.08 sec)
  o If heart rate >220 beats/min in infants or 180 beats/min in children, probable SVT
    □ Consider vagal maneuvers
    □ If IV/IO access has been established:
      □ Adenosine via rapid IV push, 0.1 mg/kg IV/IO (max 6 mg first dose)
      □ Adenosine via rapid IV push, 0.2 mg/kg (max 12 mg), can repeat x 1
    OR
    □ Consider cardioversion
      □ 0.5 to 1 J/kg first dose
      □ 2 J/kg subsequent doses
      □ If possible, sedate prior to cardioversion
• Wide complex tachycardia (>0.08 sec)
  o Probable ventricular tachycardia
  o Immediate cardioversion
    □ 0.5 to 1 J/kg first dose
    □ 2 J/kg subsequent doses
  o Consider alternative medications
    □ Amiodarone, 5 mg/kg IV over 20 to 60 minutes, or
    □ Procainamide, 15 mg/kg IV over 30 to 60 minutes, or
    □ Lidocaine, 1 mg/kg IV bolus
Bradycardia Algorithm

- If patient has cardiorespiratory compromise and heart rate <60 beats/min despite oxygenation/ventilation
  - Chest compressions, 100/min
  - Epinephrine, 0.01 mg/kg (1:10,000; 0.1 ml/kg) IV/IO, repeat every 3 to 5 min
    - If ETT has been placed, 0.1 mg/kg (1:1,000; 0.1 ml/kg)
  - Atropine, 0.02 mg/kg IV/IO (minimum dose, 0.1 mg)
    - Can repeat x 1
  - Consider dopamine or epinephrine drip
    - Dopamine, 5 to 20 micrograms/kg/min
    - Epinephrine, 0.1 to 1 micrograms/kg/min
  - Consider cardiac pacing
- Identify and treat possible causes
  - Hypoxemia
  - Hypothermia
  - Head injury
  - Heart block
  - Toxins/poisons/drugs

PEA/Asystole Algorithm

- Verify lead placement
- CPR/chest compressions
- Intubation
- IV access
- Medications
  - Epinephrine, 0.01 mg/kg (1:10,000; 0.1 ml/kg) IV/IO
    - If ETT has been placed, 0.1 mg/kg (1:1,000; 0.1 ml/kg)
- Identify and treat causes
  - Hypoxemia
  - Hypovolemia
  - Hypothermia
  - Hyperkalemia, hypokalemia, and metabolic disorders
  - Tamponade
  - Tension pneumothorax
  - Toxins/poisons/drugs
  - Thromboembolism
Pulseless V-tach/V-fib Algorithm

- **CPR**
- **Defibrillation** 2 J/kg, 2 minutes of CPR, epinephrine, defibrillate 4 J/kg
- **Medications**
  - Epinephrine, 0.01 mg/kg (1:10,000; 0.1 ml/kg) IV/IO every 3 to 5 minutes
    - If ETT has been placed, 0.1 mg/kg (1:1,000; 0.1 ml/kg)
  - **Antiarrhythmics**
    - Amiodarone, 5 mg/kg bolus IV/IO, or
    - Lidocaine, 1 mg/kg bolus IV/IO, or
    - Magnesium, 25 to 50 mg/kg IV/IO, for torsades de pointes or hypomagnesemia (max 2 grams)
  - Defibrillate 4 J/kg every 3 to 5 min

5.15 NEONATAL RESUSCITATION

- Position, suction
- Tactile stimulation
- Keep baby warm, dry
- Heart rate is the most important factor
  - If >100 beats/min, okay
  - If <100 beats/min, give positive-pressure ventilation (PPV)
  - If <60 beats/min despite stimulation, PPV, and O₂, start CPR and give epinephrine
- Bradycardia reflects hypoperfusion/hypoxia.
- Atropine is not used for neonatal resuscitation.
- Epinephrine: always use 1:10,000
  - 0.01 mg/kg IV/IO/UVC
  - 0.02 to 0.03 mg/kg ETT
- Umbilical vein can be used for fluids and medications.
- Give glucose as 2 to 4 ml/kg of 10% dextrose
- No need to suction meconium at perineum
- May resuscitate initially with room air; however, move quickly to 100% oxygen if there is no response
- CPR ratio of compressions to ventilations is 3:1
CHAPTER 6
Procedures and Skills

Joel M. Schofer, MD, and Matthew Robinson, MD

6.1 AIRWAY TECHNIQUES

Airway Adjuncts
- Oropharyngeal and Nasopharyngeal Airways
  - Utilized to prevent the tongue from falling back against the posterior pharynx and obstructing the airway.
  - Oral airway is contraindicated in conscious patients with intact reflexes, because it stimulates gagging, retching, and vomiting.
  - Nasopharyngeal airway is contraindicated in patients with nasal, facial, and basilar skull fractures.
- Laryngeal Mask Airway (LMA)
  - Indicated as a rescue device in patients with failed intubation
  - Ventilation success rates approach 100%.
  - Intubation through an LMA is successful in 80% of patients.
  - Not a definitive airway since it does not offer complete airway protection
- Combitube
  - Indicated as a rescue device in patients with failed intubation
  - Contraindicated in awake patients with airway-protective reflexes, upper airway obstruction, caustic ingestions, and known esophageal disease
  - Complications include airway hematomas, esophageal perforations, and ischemic mucosal damage from high cuff pressures.
- Lighted Stylet
  - Blind technique is indicated when intubation by direct laryngoscopy is difficult or unattainable.
  - Not indicated in the “can't intubate, can't ventilate” patient, owing to time requirement
  - Patient obesity and operator's inexperience are the most common causes of technique failure.
- Fiberoptic Intubation
  - Indicated in patients with predicted difficult intubation
  - Relatively contraindicated when excessive blood or secretions are present

Cricothyrotomy
- Indicated in “can't intubate, can't ventilate” patients and those with upper airway obstruction
- Contraindicated when endotracheal intubation can be easily achieved and in patients with tracheal transection or laryngeal fracture
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- Relatively contraindicated in children <8 years old (due to difficulty finding landmarks and small diameter of cricoid cartilage) and in patients with bleeding diathesis or massive neck edema
- Most common complications are bleeding, incorrect tube placement, and prolonged procedure time.

**Intubation**

- Nasotracheal
  - Indicated in the spontaneously breathing patient with an identified difficult airway and predicted difficult rapid sequence intubation
  - Contraindicated in apneic patients; cases of increased intracranial pressure (ICP); combative patients; and in patients with severe facial trauma and suspected basilar skull fracture or significant craniofacial anatomic disturbances, coagulopathy, or upper airway obstruction

- Orotracheal
  - Indicated in any clinical situation in which a definitive airway is necessary
  - Tracheal tube size for infants and children is calculated using the following formula:
    \[
    \text{Tube size} = \frac{(4 + \text{age in years})}{4}
    \]
  - Cuffed or uncuffed tubes are now recommended for infants and toddlers.
  - Tracheal tube depth in children is estimated by the following formula:
    \[
    \text{Depth (cm)} = \frac{\text{age in years}}{2} + 12 \text{ or } 3 \times \text{tracheal tube size}
    \]
  - In-line cervical stabilization is required for intubation in any patient with suspected cervical spine trauma.
  - End-tidal CO₂ detection using colorimetric capnometry is standard for detecting proper endotracheal tube (ETT) placement. Direct visualization of ETT passage through the cords is unreliable.
  - Color change should occur within two breaths but may be delayed for up to six breaths. Lack of immediate color change raises the suspicion of esophageal or supraglottic positioning. ETT placement should be verified through direct visualization or reintubation.

- Rapid Sequence Intubation (RSI)
  - Contraindication to RSI is the predicted difficult airway, in which bag-mask ventilation is predicted to be unsuccessful
  - Seven discrete steps
    - Preparation: patient positioning, equipment check, and medications readied
    - Preoxygenation: establishment of an oxygen reservoir in the lungs using 100% oxygen for 3 minutes. Alternatively, eight vital capacity breaths with 100% oxygen may be used. Time for desaturation to <90% in a preoxygenated, healthy 70-kg adult is 8 minutes. Healthy children, moderately ill patients, pregnant women, and obese patients desaturate much more rapidly.
    - Pretreatment
      - Lidocaine (1.5 mg/kg IV): mitigates reflex bronchospasm in patients with reactive airway disease and blunts the rise in ICP in patients with head injury
      - Fentanyl (3 µg/kg IV): attenuates reflex sympathetic response to laryngoscopy. Indicated for patients with elevated ICP and those who might be adversely affected by increased BP (intracranial hemorrhage, aneurysm, heart disease). May induce hypotension.
      - Atropine (0.02 mg/kg, minimum dose 0.1 mg): indicated for all children <12 years old to prevent succinylcholine-induced or laryngoscopy-induced bradycardia
      - Defasciculating agents: attenuate succinylcholine-induced elevation in ICP when given 1 or 2 minutes prior to succinylcholine. Appropriate dose is 10% of normal paralyzing dose of any competitive neuromuscular blocking agent.
• Paralysis with induction
  - Thiopental: potent vasodilator and myocardial depressant; do not use in hypotensive patients
  - Etomidate: most hemodynamically stable induction agent, with the exception of ketamine
  - Ketamine: commonly used in asthmatic patients because of stimulation of catecholamine release and bronchodilating effects
  - Propofol: produces vasodilatation and myocardial depression and is associated with hypotension
  - Succinycholine: adverse effects include fasciculations, hyperkalemia, bradycardia, malignant hyperthermia, masseter spasm
  - Nondepolarizing agents: delayed time to paralysis when compared to succinylcholine. Pancuronium causes tachycardia.

• Protection and positioning
  - Sellick’s maneuver (cricoid pressure) should be initiated when the patient is losing consciousness and maintained until ETI placement.

• Placement and proof
  - End-tidal CO₂ detection using colorimetric capnometry is standard to confirm tracheal placement.
  - Chest radiography to confirm proper depth of ETI

• Postintubation management
  - Treat underlying condition

Mechanical Ventilation
- Volume-cycled ventilation: delivers constant minute ventilation.
  - May result in high peak airway pressures and barotrauma.
- Pressure-cycled ventilation: volume of air delivered with each ventilation depends on pulmonary and thoracic compliance.
  - Advantage is decreased risk of barotrauma.
  - Disadvantage is that minute ventilation changes as pulmonary compliance changes.
- Control mode: ventilator delivers preset volume once triggered, regardless of patient effort.
- Support mode: provides inspiratory assistance.
  - Requires adequate respiratory drive.
- Mechanical ventilation results in decreased cardiac output due to decreased venous return.
- PEEP decreases venous return by increasing intrathoracic pressure and increases oxygenation in pulmonary edema by shifting lung water from the alveoli to the perivascular space.
- Low tidal volumes (6 ml/kg) in ARDS reduce mortality compared with high tidal volume ventilation.
- Permissive hypercapnea may be used in patients who would otherwise require high airway pressures and tidal volumes.
  - Not indicated in patients with elevated ICP, CVA, or cardiovascular instability.

Percutaneous Transtracheal Ventilation
- Safe and effective for maintaining oxygenation for prolonged periods when large-diameter catheters (>3 mm internal diameter) and intermittent high-pressure administration of oxygen are used.
- Surgical airway of choice in children <8 years old. May be used in any age group; however, cricothyrotomy is the preferred surgical airway in adults.
- Indicated in patients who cannot be intubated.
- Contraindicated when endotracheal intubation is easily performed and in patients with tracheal transection and significant cricolaryngeal trauma.
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- Requires a high-pressure oxygen source capable of delivering oxygen at 50 psi. Oxygenation is supplied by delivering 1-second bursts of oxygen for insufflation, with recommended rates of 20 bursts per minute for adults and 30 bursts per minute for children.

- Complications include barotrauma, pneumothorax, pneumomediastinum, esophageal perforation, obstruction (due to, for example, kinking of the tube or the presence of blood), and reflex coughing with ventilation.

6.2 ANESTHESIA

Local

- Anesthetics and Allergic Reactions
  - Ester-type agents: procaine, chloroprocaine, cocaine, benzocaine, and tetracaine
  - Amide-type agents: lidocaine, mepivacaine, prilocaine, bupivacaine, and etidocaine
  - Many allergic reactions are not from the anesthetic but from additives present in multi-dose vials.
  - All the esters have only one “i” in their name, while the amides have two; if a patient is allergic to an agent in one class, it is safe to use a preservative-free agent from the other class or diphenhydramine for local anesthesia.

- Anesthetic Toxicity
  - Most toxic reactions are caused by inadvertent intravenous injection; the earliest sign of toxicity is central nervous stimulation causing numbness of the tongue, light-headedness, tinnitus, visual disturbances, muscle twitching, convulsions, coma, and apnea.
  - Bupivacaine: known for causing cardiac toxicity; usually manifested as supraventricular and ventricular arrhythmias
  - Benzocaine: can precipitate methemoglobinemia in usual doses
  - The maximum dose and duration of action of commonly used anesthetics are presented in Table 6-1.

Table 6-1. Maximum Dose and Duration of Action of Commonly Used Anesthetics

<table>
<thead>
<tr>
<th>Name</th>
<th>Maximum Dose</th>
<th>Duration of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>4.5 mg/kg</td>
<td>1–2 hours</td>
</tr>
<tr>
<td>With epinephrine</td>
<td>7 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>2 mg/kg</td>
<td>4–8 hours</td>
</tr>
</tbody>
</table>

- To calculate the concentration in mg/mL from the percentage, simply move the decimal point one place to the right (for example, 1% lidocaine is 10 mg/mL).
- 1 mL of 1:1,000 lidocaine contains 1 mg; 1 mL of 1:10,000 lidocaine contains 0.1 mg

- Regional Nerve Block
  - Vasospasm and resultant ischemia from anesthetic solutions containing epinephrine are rare but can be treated with phentolamine administered by local infiltration or regional intra-arterial injection.

Sedation – Analgesia for Procedures

- Ketamine
  - Associated with unpleasant hallucinatory emergence reactions in adults; can be prevented by the co-administration of benzodiazepines and/or maintenance of a quiet atmosphere before, during, and after the procedure
  - Provides analgesia and maintains airway-protective reflexes
  - Hypersalivation can be prevented by co-administration of atropine or glycopyrrolate.
• Midazolam
  o Reversed with flumazenil
  o Can cause paradoxic reactions (hyperexcitability) in children
  o Incidence of respiratory depression increases in the presence of alcohol or opiates.

• Thiopental/Methohexital
  o Can cause hypotension when given IV, so must be used with caution in patients with volume depletion or cardiovascular compromise

• Etomidate
  o Associated with transient adrenal suppression of uncertain clinical significance

• Fentanyl
  o Associated with chest wall rigidity at doses higher than usually used for procedural analgesia; can be reversed with naloxone or positive-pressure ventilation

6.3 DIAGNOSTIC PROCEDURES

Arthrocentesis
• Usually indicated to diagnose septic or crystal-induced arthritis or determine if a laceration communicates with a joint
• Contraindicated if the tissues overlying the puncture site are infected, such as in cellulitis or an abscess
• Synovial fluid is usually sent for a cell count with differential, crystal analysis, gram staining, culture and sensitivity analysis, and glucose measurement.
  o A positive gram stain is immediately diagnostic of septic arthritis.
  o A WBC count >50,000/mm³ is highly suggestive of a septic joint.
  o Acute gout demonstrates needle-like crystals with negative birefringence.
  o Pseudogout shows rhomboid crystals with positive birefringence.

Bedside Ultrasonography (US)
• High-density tissues (bone) appear highly echogenic (white) while fluid is anechoic (black).
• Sound waves travel best through substances with tightly packed molecules; therefore, air and air-filled structures are poorly imaged by US, but fluids conduct sound well and facilitate imaging by US.
• Lower frequency US probes provide low-resolution pictures of deeper structures, such as the heart or aorta, while higher frequency probes provide high-resolution images of superficial structures, such as veins and subcutaneous tissues.

Cystourethrogram
• Indicated in trauma patients with signs of lower urinary tract injuries, including blood at the urethral meatus, abnormal position of the prostate on rectal examination, gross hematuria, perineal ecchymosis, or scrotal hematoma
• Extravasation of contrast material from a urethral disruption appears as a flame-like density outside the urethral contour; any contrast present in the bladder is diagnostic of a partial urethral injury, while a complete injury would not allow any contrast into the bladder.
• Extravasation of contrast material from a bladder injury appears as flame-like areas of contrast in the pelvis and projecting lateral to the bladder (extraperitoneal injury) or contrast filling the paracolic gutters and outlining intraperitoneal structures, especially bowel, spleen, or liver (intraperitoneal injury).
Lumbar Puncture (LP)

- Contraindicated if the puncture site is infected
- Also contraindicated if there are signs of increased intracranial pressure from a space-occupying lesion, such as hemiparesis or a unilateral third cranial nerve palsy with altered consciousness (uncal herniation); in these cases, a brain CT scan should be obtained prior to LP
- Most common complication is post-LP headache, occurring in 5% to 40% of patients; classically begins 1 to 3 days after the LP as a headache with sitting or standing that is relieved by lying down; treated with bed rest and hydration; resistant cases often respond to intravenous caffeine or a blood patch.
- May cause spinal epidural hematomas in patients with abnormal clotting mechanisms or bleeding diatheses, such as those on anticoagulants or with thrombocytopenia
- Interpretation of cerebrospinal fluid (CSF) analysis
  - Xanthochromia, indicated by a yellow-orange color of the supernatant of centrifuged CSF, is a sign of RBC breakdown products from subarachnoid hemorrhage (SAH); traumatic LP does not cause xanthochromia.
  - WBC counts higher than 5 cells/µL are pathologic in normal adults and should raise suspicion of infection; neonates may have up to 32 cells/µL, and infants up to 8 weeks old may have 22 cells/µL.
    - Viral meningitis is classically associated with WBC counts of 10 to 1000 cells/µL with a lymphocytic/mononuclear predominance.
    - Bacterial meningitis is associated with WBC counts of 500 to 20,000 cells/µL and a predominance of neutrophils, but these characteristics are variable and can be altered early in the course of disease or by antibiotics.
  - RBC counts over 10 cell/µL are abnormal; elevated RBC counts may be caused by traumatic LP, herpes simplex virus (HSV) meningoencephalitis, or intracranial hemorrhage; in traumatic LP, the RBC count should decrease from tube 1 to tube 3.
  - An abnormal gram stain may indicate the pathogen and guide therapy.
    - Gram-negative intracellular or extracellular diplococci are consistent with Neisseria meningitidis.
    - Small gram-negative bacilli may indicate Haemophilus influenzae.
    - Gram-positive cocci indicate Streptococcus pneumoniae, other Streptococcus species, or Staphylococcus.
  - CSF cultures are unaffected by antibiotic administration for 2 to 3 hours, but LP should be performed before or concurrent with antibiotics if possible.
  - Polymerase chain reaction is the preferred test for HSV meningoencephalitis.
  - India ink stains are abnormal 50% of the time in cryptococcal meningitis, and CSF cultures and cryptococcal polysaccharide capsular antigens are almost always abnormal.

Nasogastric Tube

- Safe to place in the presence of esophageal varices
- Contraindicated in patients with facial fractures with cribriform plate injuries; relatively contraindicated in patients with coagulopathy; beware of esophageal perforation in patients with alkali ingestions and esophageal injury.

Paracentesis

- Indicated to relieve tense ascites, in patients with new-onset ascites, and to rule out spontaneous bacterial peritonitis
- Low risk of hematoma if done in patients with coagulopathy; fresh frozen plasma and platelets are not normally indicated prior to the procedure.
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Pericardiocentesis
- Indicated to relieve pericardial tamponade; thoracotomy is preferred in patients with traumatic arrest associated with tamponade
- Removal of even small amounts of fluid can dramatically improve blood pressure and cardiac output.
- ECG-guided or blind techniques may be used in emergencies, but ultrasound or fluoroscopic guidance is preferred if available.
  - ECG guidance will demonstrate a wide QRS complex; ST elevation ("current of injury") will be displayed when the needle is advanced too far and is touching the epicardium; the needle should be withdrawn slightly until the "current of injury" disappears.
- Complications include failure to obtain pericardial fluid, myocardial or coronary artery laceration, hemopericardium, pneumothorax, air embolism, and dysrhythmia.

Thoracentesis
- Indicated to evaluate the cause of a pleural effusion or relieve dyspnea associated with a large pleural effusion
- Removal of more than 1 L of fluid is not recommended, to avoid reexpansion pulmonary edema, which causes dyspnea, tachypnea, tachycardia, cough, and frothy sputum.
- Most common complication is pneumothorax.

Tonometry
- Measures intraocular pressure (IOP)
- Indicated to diagnose acute angle-closure glaucoma and in patients with hyphema, who often have acute rises in IOP
- Absolutely contraindicated if a ruptured globe is suspected, as it may extrude intraocular contents

6.4 GENITAL/URINARY

Bladder Catheterization
- Foley Catheter
  - Indicated in acute urinary retention or obstruction and to monitor urine output
  - Absolutely contraindicated in trauma patients with suspected urethral injury (blood at the urethral meatus, abnormal or high-riding prostate on exam, or penile/scrotal/perineal hematoma); obtain a retrograde urethrogram prior to placement
- Suprapubic
  - Indicated in any patient who requires urethral catheterization but in whom a catheter cannot be passed; usually men with urethral stricture or prostatic disease and trauma patients with urethral disruption

6.5 HEAD AND NECK

Control of Epistaxis
- Anterior Packing
  - Controls bleeding through direct pressure and protects region from additional trauma and desiccation
  - Commercial packing or gauze packing is applied in an accordion fashion. Bilateral nasal packing is usually required to obtain adequate compression. Leave packing in place for 48 to 72 hours.
  - Coat nasal packing with antibiotic ointment and place patients on oral antibiotics to prevent sinusitis (caused by obstruction of paranasal sinuses and nasolacrimal ducts) and toxic shock syndrome.
Cautery
- Indicated for small, anterior, identified sources of bleeding
- Most commonly performed with chemical cautery using silver nitrate. Electrocautery is also used but requires more caution due to more rapid tissue penetration and increased risk for septal perforation.
- Silver nitrate will not cauterize an actively bleeding source. Cauterize peripherally to centrally and superiorly to inferiorly. Contact with blood renders the sticks ineffective.
- Contact with silver nitrate should be less than 15 seconds because of the risk of septal perforation. Bilateral use of silver nitrate is contraindicated because of the risk of septal necrosis through interruption of the septal blood supply.

Posterior Packing/Balloon Placement
- Posterior epistaxis requires direct pressure on the branches of the sphenopalatine artery. Pressure is applied with a posterior gauze pack or balloon pack.
- Foley catheters may be used and placed by inflating the balloon with 10 to 15 ml of saline and pulling traction against the middle turbinate.
- A decrease in the PaO₂ and increase in the PCO₂ (10 mm Hg) may occur with posterior packing and is caused by a nasopulmonary reflex.
- Additional complications include nasal tissue necrosis caused by improperly placed packs, asphyxiation and aspiration of packs, toxic shock syndrome, and sinusitis. Antibiotic prophylaxis is required, as with anterior nasal packs.
- Remove packing in 2 to 5 days. Removal earlier than 48 hours increases the risk of rebleeding, and later removal increases the risk of nasal tissue necrosis.

Laryngoscopy
- Indirect laryngoscopy requires the patient to be placed in the sniffing position and the tongue to be manually retracted.
- Complications of fiberoptic laryngoscopy include traumatic abrasions, bleeding, and laryngospasm.

Needle Aspiration of Peritonsillar Abscess (PTA)
- Indicated in cooperative, adult patients with an initial presentation of PTA
- Average cure rate of 80% with aspiration and antibiotics
- The superior tonsillar pole is the most common location for an abscess; initial aspiration is performed in this area.
- The carotid artery is located 2.5 cm posterior and lateral to the tonsil; aspiration or laceration of the carotid artery is the most feared complication.
- Negative aspirates from posteriorly located abscesses lead to misdiagnosis as peritonsillar cellulitis.

Removal of Rust Ring (see Image #54)
- Can be removed immediately with foreign body using a rotating burr or spud
- Removal may be delayed for 24 to 48 hours, allowing removal as a solid tissue plug after the ring has killed the surrounding epithelium

Tooth Replacement
- Reimplant as soon as possible. Best prognosis if time out of socket is less than 20 minutes. All periodontal ligament cells die after 60 minutes.
- Tooth should be stored in Hank's solution, milk, saline, or saliva during transport. Hank's solution and milk increase the viability of periodontal ligament to >3 hours.
HEMODYNAMIC TECHNIQUES

6.6 Arterial Catheter Insertion

- Indicated for direct arterial blood sampling, for continuous blood-pressure monitoring, or if indirect blood pressure monitoring is impossible or failing (severe burns, morbid obesity)
- Relatively contraindicated in patients on anticoagulants, with coagulopathies, or post-thrombolysis; radial and femoral arteries are preferred due to ease of compression.

Central Venous Access

- Indicated for the following:
  o Central venous pressure monitoring
  o Volume loading (8 French introducing catheter)
  o Emergency venous access
  o Routine venous access (drug abusers, major burn patients, obese patients)
  o Infusion of hyperalimentation or of irritating or vasoactive solutions
  o Placement of pulmonary artery catheter or transvenous pacemaker
  o Hemodialysis
- Should be placed under sterile conditions unless in extreme conditions, such as in patients in cardiac arrest
- Post-procedure imaging is indicated in all approaches except femoral to verify catheter position and assess for complications.
- Air embolism is associated with all approaches; treated by placing the patient in the left lateral decubitus position to relieve right ventricular outflow obstruction by the trapped air

Femoral
- Contraindicated in patients with suspected injury to the groin, iliac vessels, or inferior vena cava
- Associated with a higher rate of infectious complications and deep venous thrombosis

Jugular
- Less risk of pneumothorax than in infraclavicular subclavian technique
- Associated with hematoma formation, often from carotid artery puncture; treated with direct pressure

Subclavian
- Infraclavicular approach is associated with the highest incidence of pneumothorax.
- To be performed on the same side as a chest wound if subclavian vessel or superior vena cava injury is not suspected; avoids possibility of bilateral pneumothoraces
- To be performed on opposite side if injury to the subclavian vessels is suspected
- If superior vena cava injury is suspected, a subclavian catheter should not be placed; secure subdiaphragmatic access
- Not recommended in children under 2 years of age

Umbilical
- Umbilical vein catheterization is used to attain emergency vascular access in newborn resuscitations
- Vein remains patent for approximately 1 week after birth
- Vein is a single vessel usually located at the 12 o'clock position, with a thin wall and large lumen (the paired umbilical arteries have a smaller lumen and thicker walls)
Intraosseous Infusion
- Recommended in pediatric emergencies (usually cardiac arrest) when venous access is not immediately available
- Can be used to infuse nearly every medication and type of fluid
- Preferred site of insertion is the broad, flat, anteromedial surface of the proximal tibia, followed by the distal femur and tibia.
- Signs of a successful insertion into the marrow cavity include aspiration of blood and bone marrow, the ability of the needle to stand upright without support, and an easy infusion of fluids without signs of extravasation.

Peripheral Venous Cutdown
- Usually done in the greater saphenous vein due to its superficial and predictable location; found 1 cm anterior to the medial malleolus

OTHER TECHNIQUES

Excision of Thrombosed Hemorrhoids
- Thrombosed hemorrhoid diagnosed by finding of a large, firm nodule that often has a bluish discoloration.
- Excision is performed using an elliptical incision directed radially from the anal orifice and unroofing the clot.
- Linear incisions should not be used, because of the risk of premature closure and increased risk of infection and recurrence.

Foreign Body Removal
- Plain films can detect almost all glass foreign bodies larger than 1 mm in soft tissue. Metallic objects are easily visualized on plain films, with the exception of aluminum, which is more radiolucent.
- Wooden splinters require complete removal and may require incision along their entire length for removal.
- Nematocysts from coelenterates (jellyfish, man-of-war) should be removed by scraping with a hard-edged surface (knife, credit card) after inactivation with vinegar.
- Remove cactus spines using a depilatory or gel mask applied over the region.
- Remove imbedded hair tourniquets by cutting across with a No. 11 blade.
- Alkaline button batteries require early removal from nasal passages because of the risk of septal perforation.
- Sigmoidoscopy and anoscopy should be performed after removal of a foreign body from the rectum to evaluate for perforation.
- Insertion of a Foley catheter or endotracheal tube past glass foreign bodies in the rectum aids in removal by disrupting the vacuum created by the object.

Gastric Lavage
- Indicated for recent ingestions (<1–2 hours) of potentially life-threatening toxins when removal of small amounts is expected to be beneficial.
- Contraindicated in unprotected airways, strong alkali ingestions, esophageal strictures, and hydrocarbon ingestions.
- Patient should be placed in the left lateral decubitus position for lavage, which decreases passage of stomach contents into the duodenum.
- Lavage should be carried out only after gastric contents have been removed.
- Complications include esophageal tears, perforations, and pulmonary aspiration or injury.

Gastrostomy Tube Replacement
- Contraction of the stoma begins within 4 to 6 hours after tube removal.
- Placement is verified with infusion of water-soluble contrast (Gastrografin) through the tube, followed by plain radiography.
Incision/Drainage
- Superficial abscesses that can be anesthetized adequately and do not involve major neurovascular structures may be drained in the emergency department.
- Local anesthesia may be difficult because of low tissue pH, thin skin over the dome of the abscess, and patients' poor tolerance of additional skin distention.
- Conform the incision to natural skin creases and extend it the entire length of the abscess cavity.
- Decortication of the abscess cavity using blunt hemostats is recommended. Use of a gloved finger is discouraged because of the risk posed by sharp foreign bodies that could lie within the cavity.
- Packing changes are recommended every 24 to 48 hours and should be continued until healthy granulation tissue forms and drainage tract is present.
- For facial abscesses, remove the initial packing after 24 hours.

Pain Management (See Section 6.2, Anesthesia)

Physical Restraints
- Indications include protecting the patient, medical staff, or bystanders from harm; facilitating the assessment and treatment of disoriented and impaired patients; facilitating medically necessary procedures; and preventing elopement of patients who are potentially suicidal or homicidal.
- Use chemical restraints as an adjunct to physical restraints.
- Complications include patient and staff injuries. All patients in restraints should be placed on monitors.

Sexual Assault Examination
- Ideally performed at a designated rape center using standardized protocols and examination kits for evidence collection. Formal consent is mandatory due to legal implications.
- Medical stabilization takes precedence over any forensic investigation. Objective history and physical examination provide a medical and forensic evaluation of the patient. History should include a brief history of the event and a gynecologic and medical history.
- All clothing should be placed separately in paper bags to allow any fluids to be appropriately preserved.
- Areas of external trauma should be meticulously noted and photographed.
- Toluidine dye applied to the vaginal walls is highly sensitive for the detection of otherwise nonvisible lacerations.
- Bedside wet prep of fluids may be performed to look for sperm motility and is useful for documenting recent intercourse. Sperm remain motile in the cervix for up to 5 days and in the vagina for 6 to 12 hours.
- STD and pregnancy prophylaxis is recommended as follows:
  - Gonorrhea: ceftriaxone, 125 mg IM, or spectinomycin, 2 gm IM, or oral ciprofloxacin, 500 mg
  - Chlamydia: azithromycin, 1 gm PO, or doxycycline, 100 mg PO BID for 7 days, or tetracycline, 500 mg PO QID x 7 days
  - Trichomonas: metronidazole, 2 gm oral single dose
  - Hepatitis B: postexposure hepatitis B vaccination without immunoglobulin administered to patients not previously immunized
  - HIV: post-exposure prophylaxis may be offered in high-risk cases
  - Emergency contraception: offered to patients with negative pregnancy tests at the time of examination

Trephination, Nails
- Performed within 24 to 48 hours after injury
- Prepare patient with surgical scrub of finger and digital block if needed.
- Trephination may be performed with a portable hot-wire electrocautery, a heated paperclip, or 18-gauge needle.
- Multiple holes or large single hole recommended to allow continued drainage.
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Wound Closure Techniques
- Wound adhesive: indicated for linear, superficial lacerations in low-tension areas; no significant difference in cosmesis
- Staples: no significant difference in cosmesis, infection, or healing for scalp lacerations
- Fast-absorbing gut: no significant difference in cosmesis; loses tensile strength in 4 to 5 days; no removal required
- Suture: braided suture has higher tissue reactivity and risk of infection than monofilament

Wound Management
- Soaking in saline or Betadine is not recommended; does not decrease the risk of infection
- Irrigation at 20 to 40 psi significantly decreases risk of infection; may be achieved using a 35-cc syringe and 19-gauge needle
- Irrigation with tap water decreases the risk of infection.
- Suturing of facial bite wounds is recommended.
- Delayed primary closure may be used on highly contaminated wounds, with wet-to-dry dressings placed for 4 or 5 days prior to closure.

6.8 RESUSCITATION

Cardiopulmonary Resuscitation (CPR)
- Activate EMS system first, unless you are the lone rescuer of a likely asphyxial cardiac arrest, such as a child whose collapse was not witnessed.
- Chest compressions are recommended at a rate of 100 per minute in all victims except newborns.
- Ventilation (bagging) rate is 12/minute
- In all one-person CPR and adult two-person CPR, a compression-to-ventilation ratio of 30:2 is recommended until the endotracheal tube is secured. A compression-to-ventilation ratio of 15:2 is recommended for two-person CPR in infants and children.
- Each rescue breath should be delivered over 1 second and should make the chest rise.
- Immediate defibrillation for all individuals >1 year of age with a witnessed collapse and an automatic external defibrillator on scene.
- When attempting defibrillation, all rescuers should deliver one shock followed by immediate CPR beginning with chest compression, with recheck of the rhythm after 5 cycles of CPR (2 minutes). Defibrillation with a biphasic manual defibrillator should start at a dose of 120 to 200 J, with the second dose at the same or higher level.
- The initial dose for attempted defibrillation in infants and children with a monophasic or biphasic defibrillator is 2 J/kg for the initial dose and 4 J/kg for subsequent doses.

Neonatal Resuscitation
- Approach focuses almost entirely on respiratory management.
- Drying, warming, positioning, and stimulating the infant are usually sufficient for resuscitation.
- The presence of meconium no longer requires tracheal suctioning with an ETT prior to proceeding to the other steps in resuscitation, but it may be considered at several steps in neonatal resuscitation.
- In the absence of meconium, suction the mouth first and then the nose.
- Bag-valve-mask ventilation is used if stimulation does not initiate respiration.
- Chest compressions are indicated for patients with a heart rate <60 beats/min despite oxygen and adequate ventilation.
- Epinephrine is indicated for patients in asystole or with a heart rate <60 beats/min despite ventilation and chest compressions.
Naloxone, 0.1 mg, may be used in opioid-induced respiratory depression. Its use may precipitate opioid withdrawal seizures in infants.

6.9 SKELETAL PROCEDURES

Fracture/Dislocation Immobilization Technique
- Upper extremity double sugar-tong splint: immobilization of elbow and distal forearm.
- Upper extremity volar splint: immobilization of hand and wrist injuries.
- Thumb spica splint: immobilization of scaphoid, lunate, thumb fractures, and de Quervain tenosynovitis.
- Ulnar gutter splint: immobilization of the fourth and fifth metacarpals.
- Radial gutter splint: immobilization of the second and third metacarpals.
- Sling and swathe shoulder immobilizer: immobilization for proximal humerus fractures, shoulder dislocations, acromioclavicular separations, clavicle fractures.
- Figure-of-eight clavicle strap: no benefit over sling; may promote nonunion and increased fracture deformity; may predispose the patient to axillary vein thrombosis.
- Posterior knee splint: immobilization of fractures and injuries to knee.
- Posterior ankle splint: Immobilization of distal tibia/fibula fractures, reduced ankle dislocations and tarsal and metatarsal fractures; may add anterior splint, creating a bivalve splint for better immobilization.

Fracture/Dislocation Reduction Techniques
- Shoulder Dislocation
  - Most commonly dislocated joint.
  - Anterior dislocation is most common, with posterior dislocations accounting for <2%.
  - Anterior reduction techniques
    - Hippocratic technique: counter-traction by placement of foot in axilla on affected side; not recommended.
    - Kocher technique: leverage, adduction, internal rotation; not recommended.
    - Stimson maneuver: hanging arm with weight strapped to wrist; highly effective but difficult to set up.
    - Scapular manipulation: inferior tip of scapula is pushed medially and dorsally; no reported complications; >90% efficacy.
    - External rotation method: external rotation of adducted arm; >80% success rate.
    - Hennepin technique: slow external rotation, followed by abduction until head lies within the bed of the elbow; arm is then lowered onto the chest.
    - Milch technique: abduction, external rotation and traction; 70% to 90% success rate.
- Elbow Dislocations
  - Second most commonly dislocated joint in adults; most common dislocation in pediatric patients.
  - Posterior dislocation is more common.
  - Reduction is accomplished by placing the elbow in 90° of flexion and in slight supination while providing distal traction at the forearm with posterior pressure on the elbow.
  - Delayed neurovascular compromise of the ulnar or median nerve and the brachial artery is the most common complication.
CHAPTER 6 • Procedures and Skills

• Hip Dislocations
  o Posterior dislocation in 85% of cases
  o Reduction with Stimson technique of placing patient in prone position and applying downward pressure with hip, knee, and ankle flexed
  o The Allis technique places the patient in the supine position, with traction placed on the hip in 90° of flexion.

• Knee Dislocations
  o Complications include peroneal nerve injury and popliteal artery injury (20%)

Spine Immobilization Techniques

• Full spinal immobilization requires the application of a cervical collar, full rigid spine board, and foam padding or towel rolls placed alongside the cervical spine
• Sternal-occipital-mandibular-immobilization (SOMI) brace: additional support over Philadelphia collar; moderate resistance against flexion but inadequate for preventing extension due to weak occiput support
• HALO brace: very effective at limiting movement in all planes; complications include pin site infection, CSF leakage, patient intolerance

6.10 THORACIC

Cardiac Pacing

• Cutaneous
  o Indicated in symptomatic bradycardia unresponsive to atropine (sinus arrest, second- and third-degree AV block, atrial fibrillation with slow ventricular response, and asystole). Also indicated as overdrive pacing in supraventricular tachycardias and prolonged QT arrhythmias (torsade de pointes)
  o Useful in asystole only if initiated early (<10 minutes after onset)
  o Major complication is failure to recognize the presence of underlying ventricular fibrillation.

• Transvenous
  o Indications are the same as those for transcutaneous pacing.
  o Relatively contraindicated in hypothermic patients, because of the risk of inducing ventricular fibrillation
  o Right internal jugular and left subclavian veins have the most direct anatomic access to the right ventricle and highest rates of proper placement.
  o May be placed blindly or through electrocardiographic guidance using the pacemaker as a sensing lead
  o P wave and QRS complex are negative and the p wave is larger in the high right atrium.
  o P wave becomes smaller and upright with a normal QRS in the low right atrium
  o A current of injury with ST segment elevation is seen when the catheter tip reaches the ventricular wall.
  o Pacing amperage should be two to three times the threshold (level at which pacing is lost)
  o Complications include ectopy and dysrhythmias, misplacement of the catheter, and ventricular or pericardial perforation.
  o Coronary sinus placement is suggested by a right bundle branch block pattern or a posteriorly pointing catheter tip on lateral chest film.
  o Ventricular or pericardial perforation is suggested by chest pain, catheter tip outside the cardiac silhouette on chest film, or loss of the ability to properly sense.
Defibrillation/Cardioversion

- Cardioversion is indicated in patients with unstable reentrant tachycardia (hypotension, chest pain, pulmonary edema) or to electively restore sinus rhythm.
- Pacemakers and implantable cardiac defibrillators should be at least 10 cm from direct contact with paddles. Anterior-posterior pad placement is preferred.
- Complications include chest wall burns (alleviated by applying adequate conductive gel), dysrhythmias, hypoxia, and pulmonary edema.
- Dysrhythmias following high-dose shocks include VF, VT, bradycardia, AV block, and asystole.
- VF is caused by electrical discharge when large T waves are present and sensed as R waves. Change sensing leads if large T waves are noted.

Thoracostomy

- Indicated treatment of pneumothorax, hemothorax, and hemopneumothorax; prophylactically placed in patients with penetrating trauma receiving positive-pressure ventilation in the absence of radiographic abnormality
- Relative contraindications include multiple adhesions or blebs, bleeding dyscrasia, need for open thoracotomy
- Most commonly placed in fourth or fifth interspace in the midaxillary line; may also be placed in anterior second intercostal space or posterior midaxillary line
- Elevate the head of the bed 30° to 60° to decrease the risk of diaphragmatic, spleen, or liver injury
- Complications include local infection, bleeding, tube misplacement, organ injury, reexpansion pulmonary edema, air leaks, and tube blockage

Thoracotomy

- Indicated for patients with penetrating trauma with witnessed loss of vital signs in the prehospital or hospital setting and short transport time
- Selective use in patients who have sustained exsanguinating abdominal injuries
- Rarely used in cardiopulmonary arrest resulting from blunt trauma, because of poor survival rate; considered in blunt trauma with witnessed loss of vital signs at the trauma center
- Left anterior thoracotomy, the most common approach, is used when the site of injury is unknown.
- Extend the incision through the sternum into the right chest for a bilateral anterior thoracotomy when exposure is inadequate or when right-sided injuries are identified.
- Open the pericardium longitudinally anterior to the phrenic nerve.
- Place a nasogastric tube to aid in identification of the esophagus and prevent esophageal cross-clamping.
- Cross-clamp the pulmonary hilum to control bleeding from pulmonary lacerations.
- Air embolism is suggested by the presence of air bubbles in the coronary vessels and is treated by ventricular aspiration.
- The overall survival rate is 8%. Survival rate is approximately 10% in penetrating trauma and 1.5% in blunt trauma.
- The survival rate is best among patients with penetrating cardiac injuries (15%).
CHAPTER 7

Head, Ear, Eye, Nose, and Throat Disorders

James E. Colletti, MD, and Dale P. Woolridge, MD, PhD

7.1 COMMON DISORDERS OF THE EXTERNAL, MIDDLE, AND INNER EAR

External Ear

- Auricular Hematoma
  - Occurs after a shearing force is applied to the external ear
  - Commonly occurs in wrestlers
  - Management
    - Complete evacuation of the subperichondrial hematoma followed by application of a compression dressing
    - Application of a topical antibiotic ointment and oral anti-staphylococcal antibiotics
    - Resessment in 24 hours to check for reaccumulation of blood and the need for repeat drainage

- Mastoiditis
  - A complication of otitis media
  - Presentation
    - Occurs 1 to 2 weeks after otitis media
    - Fever
    - Pain is almost universally present.
    - Tender mastoid: may be red, swollen
  - Complications
    - Lateral sinus thrombosis
    - Meningitis
    - Osteitis
    - Subperiosteal abscess
    - Facial nerve palsies
    - Extension into the neck (often referred to as Bezold abscess)
  - Diagnostic test: CT scan
  - Management
    - IV antibiotics (semisynthetic pencillin in conjunction with chloramphenicol or a third-generation cephalosporin)
    - Emergent drainage by otolaryngology
Foreign Bodies
- Any living foreign body should be drowned (to kill it) by either viscous lidocaine or a 2% lidocaine solution before attempting removal.
- In addition to drowning the living foreign body, lidocaine also acts as a topical anesthetic.

Acute Diffuse Otitis Externa (OE) (Swimmer's Ear)
- Presentation - ear pain worsened by movement of the pinna with an erythematous edematous external auditory canal
- Organisms
  - *Pseudomonas aeruginosa*
  - *Staphylococcus aureus*
- Management
  - Analgesia
  - Cleansing of the external ear canal
  - 2% acetic acid solution
  - Topical antimicrobials and, in the setting of edema, a topical steroid can be utilized (a mixture of polymyxin B, neomycin, and hydrocortisone is often used).
  - Ophthalmic preparations have a higher pH than the otic solutions and therefore may be better tolerated by patients than the more acidic otic solutions.
  - If perforation of the tympanic membrane is suspected, antibiotic drops should be instituted.
  - If hydrocortisone/polymyxin/neomycin otic is chosen, the suspension, not the solution, should be used, as the solution is toxic to the middle ear.

Malignant Otitis Externa (MOE)
- Etiology
  - An infection of the external auditory canal, which is life-threatening
  - It may extend to the skull base.
  - The typical or classic patient with malignant otitis externa is an elderly diabetic who is debilitated or immunocompromised.
- Presentation
  - Fever
  - Excruciating ear pain
  - Friable granulation tissue in the auditory canal
  - Pinna/periauricular edema and/or erythema (see Image #50)
  - Cranial nerve palsy
    - Cranial nerve involvement is a serious sign.
    - Cranial nerve VII is usually the first affected cranial nerve.
    - Cranial nerves IX, X, and XI may also be involved, signifying more extensive disease.
    - Trismus indicates masseter or temporomandibular joint involvement.
- Organism: *Pseudomonas aeruginosa* is the most common organism.
- Diagnosis
  - A high index of suspicion for malignant otitis externa should be maintained in elderly, diabetic, and immunocompromised patients presenting with otitis externa and in patients presenting with persistent otitis externa despite 2 to 3 weeks of appropriate therapy.
Management
- Hospitalization for parenteral antibiotics
- Otolaryngologic consultation

Bullous Myringitis
- Presentation
  - Otalgia (ear pain)
  - Presence of bulla on the tympanic membrane and deep external auditory canal
  - Typically occurs after an upper respiratory tract infection
  - Several pathogens have been implicated as the cause of bullous myringitis, including *Chlamydia psittaci* and *Mycoplasma pneumoniae*.

- Management - erythromycin or tetracycline

Middle Ear
- Perforated Tympanic Membrane
  - May occur from penetrating or blunt trauma as well as blast injuries with changes in air or water pressure
  - Most tears are small and heal spontaneously.
  - Oral antibiotic if associated with acute otitis media
  - Otolaryngologic referral for the following:
    - Associated facial nerve palsy, hearing loss, or vertigo
    - Perforations that fail to heal
    - Perforations in the posterosuperior quadrant
    - Perforations from penetrating trauma

- Otitis Media (OM)
  - Presentation
    - Disease mainly of infancy and childhood
    - Impaired mobility of the tympanic membrane on pneumatic otoscopy
    - The clinician should assess the facial nerve because of its proximity to the middle ear.
  - Organisms
    - *Streptococcus pneumoniae*
    - *Haemophilus influenzae*
    - *Moraxella catarrhalis*
  - Management
    - Treatment of choice is a 7- to 10-day course of one of the following:
      - Amoxicillin, 80 to 90 mg/kg/day, for the majority of patients
      - Amoxicillin-clavulanate (for patients with severe otalgia or fever higher than 39°C)
      - Alternatives to penicillin for penicillin-allergic patients: second-generation cephalosporin, macrolide, trimethoprim-sulfamethoxazole, or erythromycin plus sulfisoxazole
    - According to the 2004 guidelines issued by the American Academy of Pediatrics, children with otitis media should be treated with antibiotics unless
      - They are over 2 years of age
      - They are between 6 months and 2 years with an uncertain diagnosis and symptoms that are non-severe (temperature <39.0°C, mild otalgia)
    - In the presence of one of the above criteria, observation is an option.
    - Observation for 48 to 72 hours is an option only if close follow-up can be ensured.
o Complications
  - Mastoiditis
  - Facial nerve palsies
  - Intracranial extension, i.e., meningitis (the most common intracranial complication of otitis media)

External/Middle/Inner Ear
  • Peripheral Vertigo
    o Presentation
      - Acute onset
      - Intense spinning sensation aggravated by positional changes
      - Nausea, vomiting, and diaphoresis
      - Hearing loss and tinnitus
      - Fatigable horizontal nystagmus
      - Positive Dix-Hallpike test
    o Etiology
      - External ear foreign body
      - Otitis media
      - Ototoxic drugs
      - Vestibular neuronitis
      - Benign positional vertigo
      - Labyrinthitis
      - Meniere's disease – recurring, abrupt episodes of vertigo, hearing loss, and tinnitus that last from minutes to hours; typical in 40 to 60 year olds
      - Acoustic neuroma
    o Treatment
      - Typically relieved by antihistamines (meclizine, diphenhydramine), antiemetics (promethazine, prochlorperazine), or benzodiazepines (diazepam)
  • Sudden hearing loss
    o The majority of cases of severe hearing loss (a sensorineural loss occurring over 3 days) are idiopathic.
    o Causes of sudden hearing loss include viral infections (most typically mumps), vascular abnormalities, trauma, metabolic disturbances, and ototoxic medications.
    o The most common cause of hearing loss in a previously healthy patient is cerumen impaction.
    o Rinne test – compares air conduction to bone conduction
      - Normal – air conduction is greater than bone conduction
      - Conduction deficit – bone conduction is greater than air conduction
      - Sensorineural deficit – air conduction is greater than bone conduction (same as normal)
    o Weber test – sound lateralization
      - A tuning fork is placed in the middle of the forehead; under normal conditions, the sound is heard equally in both ears.
      - Sound lateralizes in hearing loss
        - In sensorineural loss, sound lateralizes to the good ear.
        - In conduction loss, sound lateralizes to the impaired ear.
    o Management
      - If possible, identify the cause of the loss.
      - If a cause cannot be identified, obtain an emergency otolaryngologic consult.
7.2 Ocular Emergencies

External Eye

- Disorders of the Lid
  - Lid lacerations for which ophthalmology referral is warranted:
    - Lacerations of the lid margin
    - Lacerations involving the lacrimal canalicular system, canthal tendons, or levator muscle
    - Lacerations with a significant loss of tissue
    - Lacerations through the orbital septum (fat appearing in laceration)
  - Blepharitis
    - Inflammation of the margins of the lid secondary to staphylococcal infection or seborrheic dermatitis
    - Presentation: scaly, greasy lid margins that may be “red rimmed”
    - Management: baby shampoo and topical antibiotics
  - Stye (external hordeolum)
    - Acute staphylococcal infection of an oil gland
    - Management
      - Warm compresses
      - Erythromycin ophthalmic ointment twice a day for 7 to 10 days
  - Chalazion
    - Chronic eyelid inflammation that occurs from blockage of the meibomian oil glands (located in the tarsal plate)
    - Presentation: a nontender lump in the lid’s midportion
    - Management
      - Erythromycin ophthalmic ointment applied to the lid margin four times a day

- Disorders of the Conjunctiva and Cornea (Trauma)
  - Corneal abrasion
    - Presentation
      - Eye discomfort
      - Photophobia
      - Tearing
      - Corneal epithelial defects (best seen by a cobalt blue light after fluorescein staining, see Image #20)
    - Abrasions associated with contact lens use are prone to corneal ulceration and Pseudomonas infection
    - Management
      - Eyelid eversion and inspection for foreign bodies (one should suspect a foreign body under the eyelid if multiple vertical, linear abrasions are visualized on fluorescein examination)
      - To achieve cycloplegia, administer one drop of cyclopentolate, 1%, or homatropine, 5%. The dose can be repeated every 6 to 8 hours as needed for pain.
      - Refer the patient to ophthalmology or re-examine the next day.
      - Administer tetanus toxoid.
      - For abrasions not related to contact lens wear:
        - Erythromycin ophthalmic ointment
        - The eye can be patched or not patched.
      - If the abrasion is related to contact lens wear:
        - Tobramycin ophthalmic ointment four times a day
        - Do not patch
If the abrasion is from an organic source:
- Erythromycin ophthalmic ointment four times a day
- Do not patch, because patching increases the risk of infection.

**Corneal ulcer**
- **Etiology**
  - Bell's palsy can lead to corneal desiccation and sloughing of the epithelium, which allows bacteria to gain access to the underlying stroma and create an ulcer.
  - Trauma can cause a break in the cornea's epithelial layer and inoculation of the cornea.
  - Wearing soft contact lenses is a risk factor for development of a corneal ulcer (higher incidence associated with extended-wear lenses and sleeping with the lens in place).
- **Presentation**
  - Painful, red eye, with tearing
  - Photophobia may be present.
  - White corneal opacity
  - Hypopyon may be present on slit-lamp examination.
- **Management**
  - Administer a fluoroquinolone (i.e., ciprofloxacin or ofloxacin).
  - Do not patch the eye, because patching increases the risk of infection from *Pseudomonas*.
- **Complication – corneal perforation**

**Corneal foreign bodies**
- **Presentation:** a high-velocity ocular impact (e.g., hammering, grinding, or sanding) suggests a penetrating injury
- **Management**
  - After instillation of a topical anesthetic, attempt removal under slit-lamp magnification using a 30- to 25-gauge needle or a moistened cotton-tipped applicator to remove the foreign body.
  - Rust rings may be caused by foreign bodies containing iron. (see Image #54)
    - Removal may be attempted by using a spud or a burr drill.
    - Do not attempt to remove rust rings in the visual axis (pupil); such a procedure risks significant scarring that will affect vision.

**Ruptured globe** (see Image #73)
- **Presentation**
  - Teardrop-shaped pupil
  - Shallow anterior chamber
  - Hyphema
  - Chemosis
  - Decrease in visual acuity
  - A lid laceration may be accompanied by a globe laceration.
  - Seidel test – extruding aqueous humor causes fluorescein to stream
- **Management**
  - When a ruptured globe is suspected, measuring intraocular pressure is contraindicated.
  - Protect the affected eye by application of a Fox shield (a protective metal eye shield).
  - Check tetanus status and administer tetanus toxoid if indicated.
  - Tell the patient to take nothing by mouth.
  - Administer parenteral cephalosporin, analgesics, antiemetics.
Consult ophthalmology for surgical repair.

To exclude an intraorbital foreign body, obtain a Water’s view radiograph and/or orbital CT scans.

- Chemical ocular injury
  - Alkali ocular injury results in liquefaction necrosis and is generally worse than an acid burn.
  - Acid ocular injury results in coagulation necrosis.
  - Management
    - Immediate copious irrigation with at least 1 to 2 L of saline, which may be facilitated by application of topical anesthesia and placement of a Morgan lens to enhance delivery of the fluid directly to the corneal surface.
    - Irrigation should continue until pH is neutral (7.5–8).

- Cyanoacrylate ("Crazy Glue")
  - Management: Apply erythromycin to the eye and on the surface of the eyelids.

- Disorders of the Conjunctiva and Cornea (Infectious)
  - Bacterial conjunctivitis
    - Presentation
      - Mucopurulent discharge
      - Conjunctival inflammation
      - Eyelid mattering, especially in the morning
      - Foreign body sensation
    - Management
      - Warm compresses
      - Topical ophthalmic antibiotics (i.e., sulfacetamine 10%, trimethoprim sulfate and polymixin B, erythromycin, tobramycin). Neomycin should be avoided because it is associated hypersensitivity reactions.
      - Soft contact lens wearers need to be covered for *Pseudomonas* with a fluoroquinolone (ciprofloxacin, ofloxacin) or an aminoglycoside (tobramycin).
  - Viral conjunctivitis – the most common cause of conjunctivitis
    - Presentation
      - Preauricular node
      - Watery discharge
      - Chemosis – erythematous and edematous conjunctiva
      - Clear cornea
      - Fluorescein dye may demonstrate occasional punctuate staining, seen on slit-lamp examination as multiple tiny dots of stain uptake.
    - Management
      - Fluorescein staining of the cornea should be performed to exclude the corneal dendrite of herpes simplex keratitis.
      - Naphazoline may be used for conjunctival congestion and itching.
      - Follow up with ophthalmology.
      - Cool compresses may be applied.
  - Epidemic keratoconjunctivitis – a highly contagious infection caused by an adenovirus
    - Presentation
      - Chemosis
      - Corneal opacities
Photophobia
Tender preauricular lymphadenopathy
Protracted course

Neonatal conjunctivitis (ophthalmia neonatorum) – can be either chemical or infectious
- Chemical neonatal conjunctivitis occurs on the first or second day of life
- Gonorrheal conjunctivitis (secondary to *Neisseria gonorrhoea*)
  - Presentation
    - Occurs between the third and fifth days of life
    - Occurs in newborns and sexually active adults
    - May result in corneal perforation
    - An important diagnostic clue is a copious purulent discharge.
  - Management
    - IM or IV ceftriaxone
    - Topical polymyxin B, bacitracin
    - Cover for *Chlamydia*
    - Ocular irrigation with saline
    - Ophthalmology consultation and hospital admission
    - Adults – treat the mother of the neonate and her sexual partner
- Chlamydial conjunctivitis
  - The leading cause of preventable blindness worldwide
  - Trachoma – chronic follicular keratoconjunctivitis with neovascularization of the cornea, which may result in blindness; caused by *Chlamydia trachomatis*
  - Presentation
    - Occurs on the 5th to 14th day of life
    - Occurs in newborns and sexually active adults
    - When a newborn presents with conjunctivitis and concomitant pneumonia, consideration should be given to chlamydial infection.
  - Management - systemic and topical antibiotic therapy
    - Oral erythromycin (doxycycline or tetracycline may be used in adults only)
    - Topical erythromycin
- Herpes simplex keratitis – ocular herpes simplex virus
  - Presentation
    - Eye pain
    - Foreign body sensation
    - Photophobia
    - Tearing
    - Red eye
    - Decreased corneal sensation
    - Dendritic pattern over the cornea on fluorescein stain (see Image #9)
  - Management
    - Topical antiviral drops ( trifluridine: five times a day if only the lids and conjunctiva are involved, but nine times a day if the cornea is involved) plus an oral acyclovir derivative
    - Topical cycloplegic (cyclopentolate)
Avoid steroids
Ophthalmology consultation

Herpes zoster ophthalmicus – shingles in distribution of the trigeminal nerve with ocular involvement
- Presentation
  - Ocular pain
  - Vesicular eruption on the nose (known as Hutchinson’s sign) indicates involvement of the nasociliary nerve and is associated with an ocular lesion
  - The cornea may demonstrate a poorly staining mucous plaque without epithelial erosion (known as a pseudodendrite).
  - Slit lamp examination may demonstrate associated iritis.

Management
- Acyclovir
- Broad-spectrum topical antibiotic to prevent a secondary infection
- Emergent ophthalmology consultation
- Topical steroid for associated iritis only if fluorescein staining is negative for a corneal epithelial defect

Disorders of the Lacrimal Sac
- Dacryocystitis – lacrimal sac infection usually caused by *Staphylococcus aureus* secondary to nasolacrimal duct obstruction
- Presentation
  - Erythematous swollen area below the medial canthus (lacrimal sac)
  - Pressure applied over the lacrimal sac results in a purulent discharge
- Management
  - Oral antibiotics (amoxicillin/clavulanate)
  - Warm compresses
  - Ophthalmologic referral

Anterior Pole of the Eye
- Acute Iritis
  - Presentation
    - Painful red eye (deep ache unrelieved by topical anesthetic)
    - Consensual photophobia
    - Blurred vision
    - Miosis with ciliary flush
    - Decreased visual acuity
    - Cell and flare of the anterior chamber
  - Etiology
    - Trauma
    - Infection
    - Autoimmune: rheumatoid disease, ankylosing spondylitis, Reiter’s syndrome, inflammatory bowel disease
  - Management
    - Long-acting cycloplegic (homatropine)
    - Topical steroids in consultation with ophthalmologist
  - Complications
    - Glaucoma (via anterior/posterior synechiae)
    - Decreased visual acuity (lens thickening and cataract)
Hyphema (see Image #58)

- Presentation
  - Blurry vision
  - Dull eye pain
  - Photophobia following trauma

- Etiology
  - Traumatic hyphemas most commonly occur secondary to bleeding from a ruptured iris root vessel
  - Sickle cell disease is associated with spontaneous hyphemas.

- Management
  - Elevate the head of the bed
  - Instill atropine drops to dilate the pupil to decrease constriction and dilation of the iris (pupillary play)
  - Avoid activities that increase eye movement (e.g., reading and watching television)
  - Apply a metal Fox shield
  - Analgesics as needed but avoid aspirin and nonsteroidal anti-inflammatory agents
  - Antiemetics (to prevent increasing intraocular pressure)
  - In patients with sickle cell disease, avoid carbonic anhydrase inhibitors.
    - Carbonic anhydrase inhibitors lower the pH of the anterior chamber.
    - A decrease in pH results in an increase of red blood cell sickling.
    - Sickled red blood cells are less flexible and clog the outflow of aqueous humor through the trabecular meshwork, resulting in an increase in intraocular pressure.
    - In sickle cell patients, use mannitol instead of carbonic anhydrase inhibitors for intraocular pressure higher than 24 mm Hg
  - Ophthalmology consultation
  - Measure intraocular pressure (only if the globe is intact)
  - Individuals with hyphemas occupying one third or less of the anterior chamber can be followed closely as outpatients as long as follow-up can be arranged to assess for complications

- Complications
  - Rebleeding is the most common complication; it usually occurs 2 to 5 days after the initial bleed.
  - Acute and chronic glaucoma
  - Corneal blood staining
  - Optic atrophy
  - Anterior and posterior synechiae formation

Visual Reduction/Loss

- Painful Visual Reduction/Loss (Anterior Pole)
  - Acute angle-closure glaucoma
    - Presentation
      - The eye is red and painful with a hazy “steamy” cornea.
      - The pupil is mid-dilated and nonreactive.
      - Visual acuity is decreased.
      - Increased intraocular pressure – pressures of 50 mm Hg and higher are not uncommon (normal intraocular pressure is 10–21 mm Hg).
      - Often precipitated by pupillary dilation (dimly lit room, e.g., a movie theater)
      - May also present as headache, nausea, and abdominal pain
- Pathophysiology
  - Aqueous humor is produced in the ciliary body and then enters the posterior chamber.
  - From the posterior chamber, the aqueous humor moves to the anterior chamber.
  - In the anterior chamber, the aqueous humor exits via the trabecular meshwork of the anterior chamber angle to enter Schlemm's canal.
  - In acute angle-closure glaucoma during pupillary dilation, the iris is displaced forward and abuts the lens, which obliterates the angle between the cornea and the iris, thereby obstructing the trabecular meshwork.
  - This obstruction leads to an accumulation of aqueous humor, thereby increasing intraocular pressure.

- Management
  - Identify mid-dilated, nonreactive pupil with increased intraocular pressure
  - Administer topical pilocarpine, 1% to 2%, one drop four times a day, after intraocular pressure is below 40 mm Hg (reopens the angle and thereby increases the flow of aqueous humor).
  - Acetazolamide, a carbonic anhydrase inhibitor, 500 mg IV or PO (decreases aqueous humor production by the ciliary body)
  - Topical β-blocker (timolol [Timoptic], 0.5%), one drop (decreases aqueous humor production)
  - Mannitol, 1 to 2 g/kg IV
  - Recheck intraocular pressure on an hourly basis
  - Emergent ophthalmology consultation

- Painful Visual Reduction/Loss (Posterior Pole)
  - Optic neuritis
    - Presentation
      - Among patients 20 to 40 years of age, optic neuritis is the most common cause of acute reduction of vision related to optic nerve dysfunction
      - Dull ocular pain aggravated by eye movements
      - Optic disc appears swollen and hypervascular.
      - Women are more frequently affected.
      - Classic first presentation of multiple sclerosis
    - Diagnosis
      - Red desaturation test may be useful in diagnosing optic neuritis.
      - The red desaturation test is performed by having the patient look with one eye at an object that is dark red and then looking with the opposite eye to determine if the object is the same color.
      - The affected eye will see the dark red object as a lighter shade of red or as pink.
    - Management
      - Check visual acuity
      - Use of corticosteroids is controversial; discuss with ophthalmologist or neurologist as observational treatment may be preferred over steroid therapy

- Painless Visual Reduction/Loss
  - Central retinal artery occlusion (CRAO)
    - Presentation
      - Manifests as abrupt, profound, painless, unilateral visual loss
      - Funduscopic examination – A pale retina with a “cherry-red spot” (the intact choroidal circulation) can be seen through the macula.
      - Afferent pupillary defect is a common associated finding.
Management
- Treatment should begin immediately because the retina sustains irreversible damage within 90 minutes after total occlusion.
- Emergent ophthalmology consult
- Patients with CRAO rarely respond to therapy, but because vision loss can be profound, attempts to restore retinal blood flow should begin immediately by all of the means listed below:
  - Ocular massage, done in a cyclic fashion. Steady firm pressure is applied to the globe for approximately 10 to 15 seconds, followed by a sudden release of pressure. Then the application of pressure should be repeated.
  - Administer a carbonic anhydrase inhibitor such as acetazolamide 500 mg IV or PO or a topical beta-blocker (timolol, 0.5%, one drop) to decrease intraocular pressure.
  - Carbon dioxide (paper bag rebreathing) or carbogen (95% oxygen, 5% carbon dioxide) inhalation, which increases pCO₂ and thereby produces vasodilation.
  - Anterior chamber paracentesis may decompress the eye and dislodge the clot.

Central retinal vein occlusion (CRVO)
- Presentation
  - Painless, monocular, and abrupt loss of vision
  - On funduscopic examination, the optic disk is edematous and there are diffuse retinal hemorrhages ("blood-and-thunder" fundus).
  - In CRVO, the contralateral funduscopic exam is normal appearing, as opposed to papilledema, in which optic disc edema is bilateral.
  - CRVO has diffuse retinal hemorrhages, whereas optic neuritis does not.
- Treatment
  - Ophthalmology consultation to confirm the diagnosis, estimate the degree of ischemia, and minimize neovascularization
  - No specific therapy is currently available, but administration of aspirin, 60 to 325 mg/day PO, may be considered.

Giant-cell arteritis (GCA) (temporal arteritis) – systemic vasculitis of the medium and large arteries
- Presentation
  - Usually a disease of the elderly; the vast majority of patients are over 50 years of age
  - There is a strong association with polymyalgia rheumatica.
  - Symptoms may include headache, jaw claudication, rapidly progressive decreasing vision, myalgias, and tenderness of the temporal artery.
  - Thirty-three percent of people with giant cell arteritis have associated neurologic symptoms (i.e., transient ischemic attacks or cerebral vascular accident).
  - The majority of cases confirmed by biopsy are associated with an elevated erythrocyte sedimentation rate (ESR) in the range of 70 to 110 mm/hr.
- Management
  - Admission to the hospital and initiation of high-dose intravenous steroids
  - Initiation of steroids should not be delayed while waiting for confirmation from the temporal artery biopsy
Retinal detachment

- Presentation
  - Painless loss of vision
  - Flashing lights or floaters
  - Sensation of a curtain lowering over the field of vision
  - Examination of the retina demonstrates a hazy gray retina with whitish folds
  - Associated with diabetic retinopathy, trauma, Marfan's disease, sickle cell disease, nearsightedness (myopia), pre-eclampsia, premature birth

- Management
  - Patient should remain on bed rest
  - Emergent ophthalmology consultation

Neuro-Ophthalmology

- Papilledema (see Image #29)
  - Presentation
    - An indicator of increased intracranial pressure (may be from an intracranial mass, subarachnoid hemorrhage, hydrocephalus, malignant hypertension, meningitis, encephalitis, or pseudotumor cerebri)
    - Before papilledema occurs, the earliest fundoscopic indicator of increased ICP is the loss of venous pulsations.
    - Papilledema appears as bilateral optic nerve edema with blurring of the disc margin.

- Posterior Communicating Artery (PCA) Aneurysm
  - Presentation
    - Loss of the third cranial nerve results in a dilated unreactive pupil, ptosis, and limited extraocular movements.
    - Acute palsy of the third cranial nerve associated with pupillary involvement (ipsilateral dilation of the pupil) should raise suspicion for a posterior communicating artery aneurysm.
    - A pupil-sparing lesion may be a sign of hypertensive or diabetic vascular disease, but a normal pupil with third nerve palsy does not exclude a posterior communicating artery aneurysm.
    - May be associated with a headache
  - Diagnostic tests: Neuroimaging with an emergent CT or MRI to exclude space-occupying lesions, followed by a cerebral angiogram or magnetic resonance angiography (MRA)
  - Management
    - Immediate emergent neurosurgery consultation
    - Blood pressure control

Orbit

- Preseptal Cellulitis (Periorbital Cellulitis) – infection confined to the superficial tissues anterior to the orbital septum (see Image #72)
  - Organisms
    - *Staphylococcus aureus* (most common)
    - *Streptococcus*
    - *Haemophilus influenzae*
  - Presentation
    - Most commonly occurs in children younger than 3 years of age
    - Eyelid erythema, edema, warmth, and tenderness
Conjunctival injection
Normal visual acuity, extraocular movement (extraocular movement is nonpainful), and pupillary findings

Management
Mild cases of preseptal cellulitis in older children should be treated on an outpatient basis with a broad-spectrum antibiotic and close follow-up.
In more extensive cases and in younger children (younger than 5 years of age), strong consideration should be given to drawing blood for culture and hospitalization for intravenous antibiotics.

Orbital Cellulitis (Postseptal Cellulitis) – usually occurs in children; the most common source is orbital extension of sinusitis (especially ethmoid sinusitis).

Presentation
Chemosis
Ocular pain and limitation of extraocular movements are features of orbital, not periorbital, cellulitis.
Proptosis
Decreased visual acuity
Increased intraocular pressure

Organisms
Staphylococcus aureus is the most common pathogen.
Streptococcus pneumoniae
Haemophilus influenzae

Diagnostic tests: CT scan of orbits and paranasal sinuses with contrast to identify subperiosteal abscess

Management
Intravenous antibiotics (cefuroxime, penicillin and nafcillin, or chloramphenicol and nafcillin; for patients who are allergic to penicillin, vancomycin or a cephalosporin can be used)
Hospital admission
Emergent ophthalmology consultation

Complications
Subperiosteal abscess
Cavernous sinus thrombosis
Cranial nerve involvement
Osteomyelitis
Meningitis/sepsis
Loss of vision

7.3 NASAL EMERGENCIES AND SINUSITIS

Nasal Fracture
Individuals with nasal bone fractures should be referred to a plastic surgeon or otolaryngologist for re-evaluation within 5 to 7 days after emergency department treatment (after edema subsides).

Complications
Nasoseptal hematoma, which can progress to an abscess or avascular necrosis, which can then progress to a “saddle-nose” deformity
Other associated fractures (e.g., orbital floor fractures, sinus fractures, or cribriform plate fractures)
Fracture of the cribriform plate of the ethmoid bone
Cribriform plate fractures are associated with a leak of cerebrospinal fluid (CSF) through torn meninges.

A CSF leak should be suspected in a patient with clear nasal discharge after facial trauma.

To evaluate for cerebrospinal fluid rhinorrhea, place a drop of the nasal discharge on a piece of filter paper and then observe for a clear region surrounding a central stain of blood.

Epistaxis

- Incidence increases during the winter months, secondary to abrupt temperature changes and exposure to dry heat
- Anterior Epistaxis
  - More common in young patients and children
  - Accounts for the majority (90%) of cases of epistaxis
  - Kiesselbach plexus is the most common site of origin for anterior epistaxis.
  - Severe but uncommon causes of recurrent anterior epistaxis include
    - Hereditary hemorrhagic telangiectasia
    - Osler-Weber-Rendu disease
  - Diagnostic tests: laboratory investigations are not routinely necessary
  - Management
    - Evacuate all blood and clots from the nose.
    - Administer a topical vasoconstrictor and an anesthetic.
    - If the bleeding source is easily visualized, cautery can be attempted with silver nitrate in the emergency department and electrocautery later, usually by otolaryngology.
    - Anterior nasal packing can be performed with the use of nasal tampons, sponges, or petrolatum-impregnated gauze.
    - Insertion of nasal packing is facilitated by coating the tampon with a water-soluble antibiotic ointment (which delays tampon expansion until it is in place).
    - Patients who have nasal packing in place should be started on antibiotics to prevent sinusitis.
    - Follow-up in 2 to 3 days should be arranged, and nasal packing should be removed at that time.
    - If a hemostatic material such as gelfoam is used to control the bleeding, it may be left in place to be absorbed by the body.
  - Complications
    - Sinusitis
    - Toxic shock syndrome
    - Packing becomes dislodged
    - Septal necrosis
- Posterior Epistaxis
  - More common in older patients.
  - The most common arterial source of posterior epistaxis is the sphenopalatine artery, and the most common venous source is Woodruff's plexus.
  - Causes include systemic disorders such as
    - Atherosclerotic nasal arteries
    - Coagulopathy
  - Management
    - Patients with posterior epistaxis are at risk for morbidity and mortality; therefore, laboratory evaluation is often appropriate (complete blood count, coagulation panel, as well as blood type and screening).
Posterior nasal packing requires hospitalization, because patients are at risk for hypoxia, hypercarbia, bradycardia, dysrhythmias, and coronary ischemia.

Nasal Foreign Bodies
- **Presentation**
  - Observed commonly in children (a common presentation is a child with a unilateral foul-smelling nasal discharge)
  - May present with a unilateral sensation of an obstructed nares, persistent epistaxis, or persistent foul-smelling rhinorrhea despite appropriate antibiotic treatment
- **Management**
  - Several techniques have been described to remove a nasal foreign body:
    - **Positive-pressure technique** can be done by either the patient or the caregiver
      - The patient should blow his/her nose while simultaneously occluding the unobstructed nostril.
      - The caregiver blows air into the mouth of the child while occluding the nostril that does not contain the foreign body.
    - Removal by a suction catheter
    - Use of alligator forceps to grab and remove the foreign body
    - Passing either a Fogarty catheter or a pediatric Foley past the object and then inflating the balloon, followed by removal of the catheter and the obstructing object

Sinusitis
- **Major cause** is obstructed drainage at the osteomeatal complex.
- **Acute sinusitis** is sinus inflammation for less than 3 weeks, whereas **chronic sinusitis** is sinus inflammation of more than 3 weeks’ duration.
- **Presentation**
  - Maxillary toothache
  - Mucopurulent or colored nasal discharge
  - Poor response to use of nasal decongestants or antihistamines
  - Abnormal sinus transillumination
- **Organisms**
  - *Streptococcus pneumoniae*
  - *Haemophilus influenzae*
  - *Moraxella catarrhalis*
  - *Pseudomonas* is the predominant pathogen in individuals with cystic fibrosis and HIV (70% of HIV-positive patients develop sinusitis).
- **Diagnostic tests:** CT is not routinely indicated in the emergency department setting.
- **Management**
  - Symptomatic treatment (decongestants, analgesics) is used for symptoms that have been present fewer than 7 days.
  - **Antibiotics**
    - First line: amoxicillin (high dose) or trimethoprim-sulfamethoxazole (for the penicillin-allergic patient)
    - Second line (use if there is failure to improve after 7 days of therapy): amoxicillin-clavulanate, second-generation cephalosporins, fluoroquinolones, or a macrolide
  - For patients with HIV or cystic fibrosis presenting with sinusitis, clindamycin and ciprofloxacin may be administered to provide *Pseudomonas* and anaerobic coverage.
• Complications – cellulitis
  o Facial
  o Periorbital
  o Orbital – usually a complication of ethmoid sinusitis
  o Pott’s puffy tumor – abscess that occurs from anterior erosion of a frontal sinusitis
  o Subdural empyema
  o Meningitis
  o Brain abscess
  o Cavernous sinus thrombosis – presents with proptosis, eye lid edema, chemosis, palsies of cranial nerves III through VI, mental status changes, fever

7.4 OROPHARYNX/THROAT

Dental Emergencies/General
• Temporomandibular Joint (TMJ) Syndrome
  o Patients usually complain of dull, unilateral pain in the region of the TMJ, which worsens throughout the day.
  o Radiographs are not helpful.
  o Treatment includes external heat, soft diet, analgesics (usually NSAIDs), and a muscle relaxant (diazepam), with referral to a dentist specializing in TMJ disorders.

Dental Emergencies/Trauma
• Ellis Class I Fracture
  o Fracture of only the tooth’s enamel without pain or temperature sensitivity
  o Emergent treatment is not indicated.

• Ellis Class II Fracture
  o Exposure of dentin, which is creamy yellow, compared with the whiter enamel
  o Identified by sensitivity to hot, cold, and air
  o Management involves covering the exposed dentin in attempt to decrease pulpal contamination.
    • Ellis II fractures are more serious in children, because there is not much dentin to protect the pulp. Therefore, exposed dentin in children should be covered with a calcium hydroxide paste and with foil or an enamel-bonded plastic.
    • Adults should have a dressing applied for comfort.

• Ellis Class III Fracture
  o Exposure of the pulp (pink tinge or drop of blood) associated with severe pain
  o Management – An Ellis III fracture represents a true dental emergency; it therefore requires immediate attention by a dentist or an oral/maxillofacial surgeon.

• Avulsed Tooth
  o Permanent teeth that are avulsed should be reimplanted immediately into the socket.
  o Each minute of reimplantation delay is equivalent to a 1% decrease in the likelihood of tooth survival.
  o Transport media include saliva, milk, and Hank’s solution.
  o Do not replace a child’s primary teeth, as replacement may result in alveolar ankylosis.

Dental Emergencies/Infections
• Periapical Abscess
  o An infection of the tooth’s apical aspect
  o Common cause of severe dental pain
• Acute Necrotizing Ulcerative Gingivostomatitis (ANUG) (Trench Mouth or Vincent Disease)
  o Caused by spirochetes and *Fusobacterium* species
  o Presentation
    ▪ Localized swelling, pain, erythema, and ulceration of the gingiva (interdental papillae), which is covered by a grayish pseudomembrane that, when removed, results in gingival bleeding
    ▪ Patients’ breath is extremely foul smelling. They may complain of having a metallic taste, and the teeth may be mobile.
    ▪ Fever and malaise may also be present.
  o Management
    ▪ Saline irrigation
    ▪ Antibiotics – penicillin or metronidazole
    ▪ Chlorhexidine rinses
    ▪ Systemic and local analgesics to permit dental hygiene care
    ▪ Dental referral
• Periosteitis
  o Periosteitis can be distinguished from postextraction alveolar osteitis, as the pain of periosteitis emerges within the first 24 hours after a dental extraction and responds well to oral analgesia.
• Postextraction Alveolar Osteitis (Dry Socket)
  o Typically begins 3 or 4 days after extraction
  o Occurs secondary to premature dislodgment or dissolution of the healing clot from the socket, with associated localized osteomyelitis
  o Management
    ▪ Nerve block
    ▪ Socket irrigation
    ▪ Placement of packing consisting of iodoform gauze saturated with a medicated dental paste or eugenol (oil of cloves) provides almost instant relief.
    ▪ Dental referral within 24 hours

Diseases of the Oral Soft Tissue
• Ludwig’s Angina (see Image #10)
  o Bilateral infection of the submental, sublingual, and submandibular spaces
  o Stems from an odontogenic infection
  o Combination of aerobic (*Streptococcus* spp.) and anaerobic infection
  o Clinical Presentation
    ▪ Brawny induration of submandibular region
    ▪ Odynophagia, trismus, dysphonia, and dysphagia
    ▪ Symptoms may progress to airway compromise.
    ▪ Predisposing factors include alcoholism, diabetes, and systemic lupus erythematosus.
  o Management
    ▪ Be prepared for difficult airway management.
    ▪ Otolaryngology consult
    ▪ Parenteral antibiotics (penicillin/metronidazole, cefoxitin, clindamycin, or extended spectrum penicillin)
    ▪ ICU admission
Diseases of the Salivary Glands

- **Suppurative Parotitis**
  - **Presentation**
    - Occurs in debilitated, dehydrated, or postoperative patients
    - Predisposing conditions:
      - Abnormalities of the salivary duct (i.e., stricture)
      - Any medication or therapy that decreases salivary flow (e.g., antihistamines, phenothiazines, radiation therapy)
    - Parotid gland is tender, erythematous, and swollen.
    - The patient may also complain of fever and trismus.
    - A purulent discharge can be expressed from Stenson’s duct.
    - Usual bacteria are *S. aureus* mixed with anaerobes
  - **Diagnostic tests:** The diagnosis is mainly a clinical one, but if a purulent discharge is expressed from Stenson’s duct, it should be sent for culture.
  - **Management**
    - Hydration
    - Massage
    - Application of local heat
    - Sialogogues (e.g., lemon drops or other tart hard candy)
    - Antibiotics (amoxicillin-clavulanate or ampicillin-sulbactam; alternatives are clindamycin alone or a combination of vancomycin and metronidazole)

- **Sialolithiasis**
  - **Presentation**
    - The majority (more than 80%) of salivary calculi are found in the submandibular gland; most of the remainder of salivary stones occur in the parotid gland.
    - Presents with pain, tenderness, and swelling of the salivary gland, which is aggravated by stimulation of gland secretion (e.g., eating)
  - **Diagnostic tests**
    - Intraoral radiographs will find the majority (more than 90%) of calculi
  - **Management**
    - Analgesic
    - Moist heat
    - Antibiotics if there is concurrent infection
    - Massage
    - Sialogogues (e.g., lemon drops or other tart candy)

Disorders of the Neck and Upper Airway

- **Trauma**
  - **Laryngotracheal trauma**
    - Injuries from laryngotracheal trauma include the following:
      - Tears of the mucosa
      - Fracture of the thyroid, hyoid, or cricoid
      - Disruption of the vocal cords
      - Separation of the trachea from the larynx
CHAPTER 7 • Head, Ear, Eye, Nose, and Throat Disorders

- Presentation
  - Pain in the larynx
  - Hoarseness
  - Changes in voice to inability to talk
  - Dysphagia
  - Stridor
  - Subcutaneous emphysema

- Diagnostic tests
  - Soft tissue of the neck
  - Computed tomography
  - Fiberoptic bronchoscopy

- Management
  - The major issue is airway management.
    - Avoid use of paralytic agents.
    - Surgical exploration with formal tracheostomy
    - Intubation is controversial in the setting of laryngeal trauma.
  - Hospital admission
  - Otolaryngologic consultation
  - Voice rest
  - Humidified air
  - Prophylactic antibiotics

- Infectious
  - Pharyngitis
    - Epidemiology
      - Viruses are the most common cause.
      - Other casual organisms are bacteria, fungi, and parasites.
      - Viral pharyngitis cannot reliably be distinguished from bacterial based on clinical findings.
      - The incidence of group A β-hemolytic Streptococcus (GABHS) pharyngitis peaks in the late winter and early spring.
      - Occurs most commonly between the ages of 3 and 20
    - Presentation of GABHS pharyngitis
      - The incubation period lasts 3 to 5 days.
      - Abrupt onset of throat pain, pain on swallowing, anterior cervical adenopathy that is tender to palpation, fever, and chills
      - Commonly associated symptoms are headache, nausea, and vomiting.
      - Palatal petechiae usually accompany marked erythema of the throat and tonsils.
      - The CDC lists four criteria for diagnosis of GABHS: 1) tonsillar exudate, 2) tender anterior cervical lymphadenopathy, 3) absence of cough, and 4) history of fever.
    - Management
      - Warm salt water gargles
      - Intravenous fluids should be administered to patients who are unable to tolerate oral fluids or are dehydrated.
      - Penicillin is the drug of choice for pharyngitis secondary to GABHS because of its narrow spectrum, efficacy, and low cost.
For penicillin-allergic patients, erythromycin is a suitable alternative.

Complications
- Suppurative: peritonsillar abscess, retropharyngeal abscess, suppurative cervical lymphadenitis, deep-space neck infections
- Glomerulonephritis (not preventable with antibiotic therapy; related to specific strains of GABHS)
- Rheumatic fever – Appropriate antibiotic treatment within 9 days of infection will prevent rheumatic fever.

Epiglottitis
- Presentation
  - Epiglottitis is a life-threatening disease caused by *Haemophilus influenzae* type b (Hib).
  - Historically, epiglottis was a childhood disease, but since the introduction of the Hib vaccine, the incidence of epiglottitis in children has declined.
  - Adults with epiglottitis present with a disproportionately sore throat and inspiratory stridor.
  - Patients may appear apprehensive and toxic, sitting in the tripod position to maintain their airway or lying supine. They may be unable to control their oral secretions and therefore may be drooling.
  - A physical exam marker for supraglottic infection is pain on movement of the upper trachea or thyroid cartilage.
- Diagnostic tests: Soft-tissue lateral neck radiograph (should be done with a portable unit) may reveal obliteration of the vallecula, enlarged and thumb-shaped epiglottis, edema of the aryepiglottic folds, as well as ballooning of the hypopharynx with air (see Image #1)
- Management
  - Be vigilant and never leave the patient unattended or allow him/her to leave the emergency department. Patients with epiglottitis can experience an unpredictable and sudden obstruction of the airway.
  - Prepare for a potential surgical airway.
  - Obtain immediate otolaryngologic consultation to establish a definitive airway in the operating room.
  - Antibiotics against *Haemophilus influenzae* type b (cefuroxime, cefotaxime, or ceftriaxone) should be initiated.

Bacterial tracheitis
- Presentation
  - Occurs in children younger than 3 years of age
  - Patients initially have a few days of croup-like symptoms that progress to a toxic appearance, stridor, a high fever, and copious thick purulent secretion.
- Organisms
  - *Staphylococcus aureus*
  - *Streptococcus pneumoniae*
  - *Haemophilus influenzae*
  - *Moraxella catarrhalis*
- Diagnostic tests: Soft-tissue lateral neck radiograph may demonstrate subglottic tracheal narrowing and a ragged tracheal border.
- Management
  - Obtain immediate otolaryngologic consultation for intubation and bronchoscopy in the operating room.
  - Vancomycin and a third-generation cephalosporin
Supplemental humidified oxygen and monitoring
Intravenous fluids

Peritonsillar abscess (PTA) is the most common deep-space infection of the head and neck.

Presentation
- Occurs in patients in their twenties and thirties
- Patients present febrile, drooling, with a “muffled hot-potato voice,” complaining of a sore throat, odynophagia, dysphagia, malaise, and otalgia.
- On physical exam, there is marked swelling of the pharyngeal pillar, with the uvula and soft palate pushed to the contralateral side.
- Needle aspiration aids the clinician in distinguishing a peritonsillar abscess from peritonsillar cellulitis.
- Needle aspiration is performed as follows:
  - Obtain adequate anesthesia.
  - Direct an 18-gauge needle medially and superiorly within the abscess cavity no more than 1 cm deep.

Management
- Most patients can be treated on an outpatient basis with needle aspiration, antibiotics, and oral analgesics.
- If needle aspiration fails, incision and drainage or tonsillectomy can be performed.
- A child with a peritonsillar abscess should be admitted to the hospital, receive intravenous hydration and antibiotics, and undergo removal of the abscessed tonsil under general anesthesia.
- Antibiotics
  - The drug of choice is high-dose penicillin.
  - For penicillin-allergic patients, erythromycin may be used.
  - Other potential regimens include cefoxitin, ampicillin/sulbactam, clindamycin, or a combination of metronidazole or rifampin with penicillin.

Retropharyngeal abscess
- The retropharyngeal space is a potential space anterior to the prevertebral fascia that extends from the skull base to the level of the tracheal bifurcation.
- In a retropharyngeal abscess, the lymph nodes contained in the retropharyngeal space become supplicative.
- The lymph nodes in the retropharyngeal space atrophy after 4 to 6 years of age.

Presentation
- Mainly a pediatric entity occurring between 6 months and 5 years of age
- The majority of children are less than a year old; almost one third of cases occur in children less than 6 months old.
- History of a puncture wound to the hypopharynx (e.g., running with a pen in the mouth and falling)
- Sore throat, fever, drooling, dysphagia, stridor, and a muffled voice
- Physical exam reveals the posterior pharynx to be edematous and erythematous with associated tender cervical adenopathy.

Organisms
- Group A Streptococcus
- Anaerobic organisms
- Staphylococcus aureus
• Diagnostic tests
  - Inspiratory lateral soft-tissue radiograph of the neck taken in moderate extension demonstrates an increased width of the soft tissues anterior to the vertebrae.
  - CT
    - Should be obtained if there is suspicion on radiographs
    - A CT scan with intravenous contrast is regarded as the gold standard for diagnosing deep neck space infections, as it aids in distinguishing between abscess formation and cellulitis as well as determining the extent of disease.

• Management
  - Immediate otolaryngologic consultation for transoral or transcranial incision and drainage in the operating room
  - Hospital admission
  - Maintain vigilance over the airway for signs of obstruction.
  - Intravenous hydration
  - Antibiotics effective against normal oropharyngeal flora, *Staphylococcus aureus*, and *Bacteroides* should be used. Reasonable choices: ampicillin/sulbactam, ticarcillin/clavulanate, piperacillin/tazobactam, cefoxitin, clindamycin, or a combination of metronidazole and high-dose penicillin.

• Complications
  - Mediastinitis
  - Airway obstruction
  - Empyema
  - Aspiration pneumonia
  - Erosion into the carotid artery; internal jugular vein thrombosis

• Miscellaneous
  - Posttonsillectomy bleeding
    - Presentation
      - Occurs between postoperative days 5 and 10
      - More common in adolescents
    - Management
      - Airway control – in cases of massive hemorrhage, intubation may be necessary for airway protection
      - Obtain hemostasis
        - Application of direct pressure using ringed forceps
        - May use gauze impregnated with 1:1000 epinephrine and 1% lidocaine
      - Otolaryngology consultation

Angioedema (see Image #67)
• Allergic Angioedema
  - IgE-mediated reaction that results in swelling of the subcutaneous and submucosal tissues
  - Presentation: nonpruritic, well-demarcated edema of the face and neck
  - Management
    - Airway management is crucial.
      - Intubation – can be nasotracheal or endotracheal (may be difficult secondary to oral obstruction)
      - Surgical
• Epinephrine
  □ Early laryngeal edema – 0.3 to 0.5 ml 1:1,000 IM
  □ Late laryngeal edema – 10 ml of 1:100,000 IV over 10 minutes
• Antihistamines (diphenhydramine, 50 mg IM or IV)
• Steroids (methylprednisolone, 125 mg IV)
• H2 receptor blocker (cimetidine, 300 mg IV, or ranitidine, 50 mg IV)
• Allergy referral

• Hereditary Angioedema (HAE)
  o Autosomal dominant inheritance
  o Caused by a C1 esterase inhibitor deficiency
  o Presentation: face and airway edema, abdominal pain, nausea, and vomiting
  o Management
    □ Airway management is crucial.
    □ Epinephrine, antihistamines, and steroids are often tried, but benefit has not been reported in the literature.
    □ C1 esterase inhibitor replacement
      □ C1 esterase inhibitor concentrate
      □ Fresh-frozen plasma

• Angioedema from Angiotensin-Converting Enzyme (ACE) Inhibitor Antihypertensives
  o The pathophysiology is believed to be secondary to an accumulation of bradykinin and angiotensin I, which can lead to angioedema.
  o Presentation: predilection of edema to the lips, laryngeal soft tissues, and the tongue
  o Management
    □ Airway management
      □ Intubation – can be nasotracheal or endotracheal (may be difficult secondary to oral obstruction)
      □ Surgical
    □ Epinephrine, antihistamines, and steroids are often tried but benefit has not been reported in the literature.
    □ The patient should be taken off ACE inhibitors immediately and is never to take them again.

• Uvulitis – edema of the uvula
  o Etiology
    □ Infectious
    □ Angioedema
    □ Idiopathic
  o Presentation
    □ Sore throat, sensation of a foreign body
    □ If secondary to an infectious etiology, fever may be present
    □ The uvula appears erythematous and edematous and is tender.
    □ If secondary to angioedema, the uvula may appear pale and edematous and the patient may have associated allergic symptoms (wheezing, pruritus, and urticaria)
  o Management
    □ The majority of cases are self-limiting.
    □ If secondary to angioedema, it is treated as angioedema (see above).
If secondary to infection, antibiotics should be initiated depending on the suspected primary source of infection.

Disorders of the Mouth

- Infectious
  - Oral thrush
    - Most common in newborns
    - Immunosuppressive illness such as AIDS should be considered when oral thrush is present in an adult who does not wear dentures or is not on antibiotics.
    - Presentation
      - White flaky plaques on an erythematous base located on the buccal mucosa, gingiva, tongue, tonsil, or palate
    - Organism – *Candida albicans*
    - Management
      - Oral nystatin suspension (100,000 U/mL) for infants
      - Clotrimazole troches for adults
      - If topical treatment fails, then oral ketoconazole or fluconazole can be used.
  - Aphthous stomatitis
    - Presentation – 2- to 3-mm ulcers on the oral mucosa with a white center that typically spontaneously resolves in 10 to 14 days
    - Management
      - Supportive care: pain management with “magic mouthwash” (a liquid mixture of diphenhydramine, lidocaine, and aluminum/magnesium hydroxide)
      - Topical steroids (betamethasone syrup or a mouth rinse of 0.01% dexamethasone elixir) may be tried as well.
      - Oral antibiotics if secondary infection occurs
  - Herpes gingivostomatitis
    - Mainly a disease of small children and the immunosuppressed
    - Presentation
      - Fever, malaise, cervical lymphadenopathy, throat and mouth pain
      - Vesicular and ulcerative lesions on the lips and tongue
    - Organism – herpes simplex virus
    - Management
      - Supportive care: maintain hydration, pain management with “magic mouthwash” (a liquid mixture of diphenhydramine, lidocaine, and aluminum/magnesium hydroxide)
      - Acyclovir should be considered for immune suppressed patients.

- Facial Trauma
  - Mandibular fractures
    - Presentation
      - Jaw pain, deformity, abnormal motility, and deviation to the fractured side upon opening
      - Dental malocclusion
      - Intraoral laceration suggests open mandibular fracture (check between teeth).
      - Ecchymosis of the floor of the mouth
      - Mental nerve anesthesia
      - Multiple fractures occur in more than 50% of patients
Most common fracture sites: condyle, body, and angle

Major concerns:
- Patency of airway
- Possibility of cervical spine injury
- Open fractures: admission, IV antibiotics (penicillin or clindamycin)

**Mandibular dislocation**
- May result from yawning, laughing, vomiting, or trauma
- Presentation
  - Jaw pain
  - Difficult speaking or swallowing
  - Malocclusion
  - A bilateral dislocation presents with an anterior open bite.
  - In a unilateral dislocation, the mandible deviates away from the dislocation, in the direction of the unaffected side.
- Diagnostic tests: Panoramic radiographs of the mandible should be obtained in the setting of trauma.
- Management
  - Analgesia
    - Short-acting muscle relaxant to reduce muscular spasm (midazolam)
  - Manual reduction
    - The physician’s thumbs wrapped in gauze are placed over the lower molars as far back as possible.
    - The clinician’s fingers are curved beneath the angle and body of the mandible.
    - Downward and backward pressure is applied.

**Maxillary fractures**
- Presentation: tenderness, deformity, crepitus, mid-face mobility
- Associated injuries:
  - Trigeminal and facial nerves
  - Entrapment of extraocular muscles
  - CSF rhinorrhea
    - Major concerns: patency of airway and possibility of cervical spine injury

**Frontal sinus fracture**
- Fractures through the anterior wall of the frontal sinus often continue through the posterior wall.
- When a fracture of the posterior wall of the frontal sinus is present, a leak of cerebrospinal fluid should be assumed.
- Fractures through the posterior wall may be associated with a cranial injury or a dural tear.
- Presentation
  - Forehead depression
  - Subcutaneous air is indicative of sinus disruption
  - May be an associated laceration
  - Numbness of the supraorbital (forehead) nerve distribution indicates the possibility of an occult fracture.
  - A thorough nasal speculum examination should be performed to evaluate for a cerebrospinal fluid leak or blood located high in the nasal cavity.
Diagnostic tests
- Caldwell view of the face
- CT to evaluate the integrity of the posterior wall of the frontal sinus and for associated brain injury

Management
- Antibiotics that cover skin and sinus flora (erythromycin, trimethoprim-sulfamethoxazole, amoxicillin-clavulanate, cephalosporins)
- Epistaxis control
- Analgesia
- Fracture through the frontal sinus, even when minimally displaced, requires elevation for cosmetic reasons.
- Fractures of only the anterior table can be managed conservatively, with a referral to ENT in 1 or 2 days for elevation for cosmetic reasons.
- Fractures that involve the posterior table require immediate neurosurgical involvement.

Complications
- Involvement of the posterior table can lead to mucopyocele or epidural empyema
- Cerebrospinal fluid leaks may present in a delayed fashion.

Le Fort I
- Maxilla fracture at the level of the nasal fossa
- Mobility of the hard palate and alveolar ridge

Le Fort II
- Pyramidal fracture involving maxilla, nasal fossa, and medial orbits
- Mobility of the nose and upper dental arch

Le Fort III
- Craniofacial disjunction involving zygoma, nasal bone, across the orbits, ethmoid bones, and cranial base
- Associated with cerebrospinal fluid rhinorrhea
- Mobility of the entire face

Zygomatic Fractures
- Zygomatic arch fractures
  - Flatness of cheek, bony defect, and painful mandibular movement
  - Best visualized on a submental-vertex view
- Zygomatic-maxillary complex fractures ("tripod fracture")
  - Fracture at the zygomatic arch, zygomaticofrontal suture, and the infraorbital foramen (may also include fracture of the lateral wall of the maxillary sinus)
  - Presentation
    - Flattening of the cheek
    - Periorbital swelling and ecchymosis
    - Anesthesia in infraorbital nerve distribution
    - Diplopia
  - Diagnostic tests: Waters and submental vertex radiographs
• Orbital
  o Orbital blowout (floor) fracture (most common)
    ▪ The most common orbital fracture
  ▪ Presentation
    □ Pain
    □ Diplopia on upward gaze (may indicate entrapment of the inferior rectus muscle) (see Image #58)
    □ Inability to look up or limited upward gaze (may indicate entrapment of inferior rectus muscle)
    □ Enophthalmos
    □ Infraorbital anesthesia (anesthesia of the ipsilateral cheek, upper lip, and maxillary teeth)
    □ Step-off deformity over the intraorbital rim
  ▪ Diagnostic tests
    □ Water's view
      – Air/fluid level in maxillary sinus
      – Hanging tear drop (soft tissue into maxillary sinus)
      – Orbital emphysema
    □ Facial CT defines fracture and associated injury
  ▪ Management
    □ If subcutaneous emphysema is present, give antibiotics against sinus pathogens.
    □ If the fracture extends into the sinus, the patient should not blow his/her nose.
    □ Ophthalmology referral

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CHAPTER 8
Systemic Infectious Disorders

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8.1 BACTERIAL

Meningococcemia

- General Information
  - Organism: *Neisseria meningitidis*
    - Aerobic, gram-negative diplococcus
    - Releases an endotoxin that causes fulminant sepsis
    - Groups B and C are the most common in the United States.
  - Peak incidence during the winter months
  - Factors associated with a high risk for developing sepsis:
    - Chronic alcohol abuse
    - Asplenia
    - Recent respiratory illness
    - Complement deficiency
    - Corticosteroid use
  - Overall mortality is approximately 10%.

- Clinical Presentation
  - Can range from a mild febrile illness to fulminant sepsis
  - Meningitis
    - Gradual onset of symptoms over 24 hours
    - Symptoms are fever, headache, photophobia, and vomiting.
    - 20% of patients present with seizures
    - Over 50% have a rash (see below)
  - Meningococcemia
    - Signs and symptoms include fever, lethargy, cyanosis, hypoventilation or hyperventilation, and poor peripheral perfusion.
    - Petechial rash (see Image #36)
      - Begins on the ankles, wrists, and axilla
      - Can progress to involve any body surface
      - Typically spares the palms and soles
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- Purpura (see Images #35A and 35B)
  - Distinct from petechiae
  - Purpura fulminans is usually seen in children and is associated with disseminated intravascular coagulation (DIC).
- Waterhouse-Friderichsen syndrome
  - Bilateral adrenal hemorrhage
  - Seen in 10% to 20% of patients
  - Characterized by rapid deterioration with circulatory collapse and shock
- Complications
  - Cardiac
    - Myocarditis resulting in congestive heart failure
    - Conduction abnormalities
  - Acute renal failure
  - DIC
  - Acute respiratory failure
  - Septic arthritis
- Diagnosis
  - Initial diagnosis is made on clinical grounds.
  - Confirmed by finding organism in blood or in cerebrospinal, synovial, pleural, or pericardial fluid
  - Characteristic cerebrospinal fluid (CSF) findings:
    - Elevated opening pressure
    - Elevated protein
    - Low glucose concentration
    - Elevated white blood cell (WBC) count with a left shift
    - Gram stain with gram-negative diplococci
- Treatment
  - Supportive care with airway management, IV fluids, vasopressor medication if needed, and correction of electrolyte disturbances
  - Antibiotics should be given as soon as the diagnosis is suspected.
    - Drug of choice remains penicillin G IV every 2 to 4 hours.
    - Acceptable alternatives are cefotaxime, ceftriaxone, and chloramphenicol.
  - Prophylaxis is indicated for household, intimate, nursery school, and day care center contacts; also indicated for health care workers with intimate exposure (those performing endotracheal intubation)
    - Antibiotics
      - Rifampin: 600 mg every 12 hours for 4 doses OR
      - Ciprofloxacin: 500 mg as a single dose
    - Vaccination
      - Quadrivalent vaccine for groups A, C, Y, and W-135
      - Indicated as an adjunct to prophylaxis in epidemics and close contacts in sporadic cases

Staphylococcal Toxic Shock Syndrome (TSS)
- General Information
  - Organism: *Staphylococcus aureus*
  - Infection is caused by toxigenic strains of *S. aureus*.
    - TSS toxin 1 and endotoxin B are the most common toxins.
Menstruation remains the most common setting for TSS, although non-menstrual causes are known (superinfection of burns, surgical sites, and varicella or staphylococcal respiratory infections).

**Clinical Presentation**
- Initially patients present with prodromal symptoms: fever, myalgias, nausea, vomiting, and diarrhea
- Rash
  - Diffuse, nonpruritic, blanching erythroderma
  - After 1 week, the rash begins to desquamate, especially over the face, trunk, and extremities.
  - Eventually, full-thickness desquamation of the palms, fingers, and soles occurs.
- Central nervous system (CNS) symptoms may include confusion, agitation, combativeness, or somnolence.
- Gastrointestinal symptoms include abdominal pain and hepatomegaly.
- Mucosa inflammation is characterized by vaginal, oropharyngeal, or conjunctival hyperemia.

**Complications**
- Acute renal failure
- Acute respiratory distress syndrome (ARDS)
- DIC
- Cardiovascular collapse
- Less common complications include rhabdomyolysis, seizures, pancreatitis, pericarditis, and cardiomyopathy

**Diagnosis**
- Diagnosis does not require a positive culture for *S. aureus*.
- Patients must have the following:
  - Fever, rash, desquamation, hypotension, AND
  - At least three major organ systems involved (gastrointestinal [GI], musculoskeletal, mucous membranes, renal, hepatic, hematologic, or CNS)
- Laboratory studies are nonspecific; they may demonstrate leukocytosis or leukopenia, anemia, thrombocytopenia, elevated blood urea nitrogen (BUN)/creatinine, hyperbilirubinemia, or elevated aminotransferases.

**Treatment**
- Supportive care with IV fluids, vasopressor medications if needed, and removal of the source (tampon, nasal pack, incision and drainage [I&I] of abscess)
- Antibiotics of choice are penicillin + clindamycin
- Consider intravenous immunoglobulin (IVIG) in patients who do not respond to antibiotics, fluids, and vasopressors.

**Streptococcal Toxic Shock Syndrome (Strep TSS)**

**General Information**
- Organism: group A streptococcus
  - Produces streptococcal pyrogenic exotoxins A and B
- Classically associated with soft-tissue infections (necrotizing fasciitis), pneumonia, peritonitis, and osteomyelitis
- Cases in children typically follow chickenpox
- Mortality is much higher than TSS, ranging from 30% to 80%

**Clinical Presentation**
- Hyperthermia or hypothermia
- Tachycardia
- Skin findings can include erythema, bullae, and extreme tenderness out of proportion to appearance.
• Complications
  o Similar to TSS (see above)

• Diagnosis
  o Requires a positive culture for group A streptococcus PLUS
  o Hypotension PLUS
  o Signs of multiple organ involvement (GI, renal, hepatic, musculoskeletal, pulmonary, hematologic)

• Treatment
  o Early surgical consultation for debridement
  o Antibiotics of choice are a combination of penicillin and clindamycin
  o Acceptable alternative regimens include a combination of ceftriaxone and clindamycin OR erythromycin alone

Necrotizing Fasciitis
• General Information
  o Two types exist
    ▪ Type 1 is polymicrobial (non-group A streptococcus plus anaerobes) and is typically located on the abdomen or perineum (Fournier's gangrene, see Image #2).
    ▪ Type 2 is caused by group A streptococcus and is typically located on the extremities.

• Clinical Presentation
  o Fever
  o Wound
    ▪ Abrupt onset of erythema and edema
    ▪ Pain out of proportion to physical exam findings
    ▪ Serosanguinous discharge may be present.

• Diagnosis
  o Initially a clinical diagnosis
  o X-ray film may show gas (this is not a consistent finding).
  o MRI can aid in differentiating necrotizing fasciitis from cellulitis; however, imaging should not delay initiation of treatment.

• Treatment
  o Supportive care with aggressive IV fluids + vasopressors if needed
  o Urgent surgical debridement is required.
  o Broad-spectrum antibiotics against *S. aureus*, *Streptococcus*, gram-negative organisms, and anaerobes should be given.

Gas Gangrene (see Image #5)
• General Information
  o Organisms
    ▪ Clostridial species are the most common organisms.
      ▪ Spore-forming, anaerobic gram-positive organisms
      ▪ Produce a toxin that destroys muscle
    ▪ Non-clostridial gas gangrene is typically polymicrobial, involving *Escherichia coli*, *Klebsiella*, *Enterobacter*, *Peptostreptococcus*, *Peptococcus*, and *Bacillus fragilis*.
  o Infection typically occurs in traumatized skin or necrotic tissue.
  o Abrupt onset and progression of symptoms over 6 to 24 hours
• Clinical Presentation
  o Pain is the earliest symptom.
  o Fever and tachycardia (out of proportion to fever)
  o Initially, the wound will be tense from edema; eventually, vesicles or dark fluid-filled vesicles or bullae form; wound is malodorous.
  o Crepitus is not a reliable finding.
• Diagnosis
  o Diagnosis is clinical.
• Treatment
  o Supportive care with IV fluids + vasopressors if needed
  o Urgent widespread surgical debridement
  o Antibiotics regimens include penicillin + clindamycin OR ceftriaxone + clindamycin
  o Consider hyperbaric oxygen therapy.

**Sepsis**

• General Information
  o Definitions
    • Systemic Inflammatory Response Syndrome (SIRS)
      □ At least two of the following:
        — Temperature >38°C or <36°C
        — Respiratory rate >20 breaths/min
        — Heart rate >90 beats/min
        — WBC count >12,000 cells/µL OR <4,000 cells/µL OR >10% bands
    • Sepsis: two or more SIRS criteria in the setting of an identified, or presumed, infection
    • Severe Sepsis: sepsis with one or more signs of organ dysfunction (see below)
    • Septic Shock: severe sepsis with hypotension that is refractory to fluid resuscitation
  o Pneumonia, abdominal infection, and urogenital infection are the leading causes of sepsis.
• Clinical Presentation
  o Widely variable and dependent on patient co-morbidities, source of infection, and organ systems involved
  o Vital signs are abnormal and can demonstrate hyperthermia or hypothermia, tachycardia, tachypnea, hypoxia, or hypotension.
  o Skin initially is flushed, warm, and well perfused; in late stages, the skin appears mottled and cyanotic.
  o CNS dysfunction is manifested by lethargy or altered mental status.
  o Renal dysfunction is indicated by oliguria.
• Diagnosis
  o Diagnostic studies are performed to determine the source of infection and the severity of disease.
  o Routine laboratory studies may reveal the following:
    □ CBC: leukocytosis or leukopenia, bandemia, thrombocytopenia or thrombocytosis, or anemia
    □ Chemistries: acute renal failure, low bicarbonate, elevated aminotransferases, elevated bilirubin, and elevated lactate
    □ Coagulation profile: DIC (elevated PT, elevated aPTT, decreased fibrinogen, and elevated fibrin split products)
Radiologic studies are used to determine the source of infection:
- Chest film to exclude pulmonary source and/or ARDS
- Soft tissue x-ray films if a necrotizing infection is suspected
- CT (abdomen, head, extremity) depending on clinical presentation
- Ultrasound if pelvic or gallbladder pathology is suspected

Microbiology
- Blood, sputum, and urine cultures
- Where applicable, CSF cultures and tissue cultures

Treatment
- Early airway management to maintain adequate oxygenation
- Aggressive circulatory resuscitation
  - Goals
    - Mean arterial pressure (MAP) >65 mm Hg
    - Urine output >0.5 to 1.0 cc/kg/hr
    - Central venous pressure >8 mm Hg
    - Central venous oxygen saturation >70%
    - Decreasing lactate concentration
    - Fluid resuscitation: may require 6 to 10 liters
    - Vasopressor medication should be started if MAP is not >65 mm Hg after a 40-cc/kg fluid bolus.
- Antibiotics must be given early; the initial antibiotic should be broad spectrum and based on patient co-morbidities, history of infections, and current suspected source.
- Source control with drainage of abscess/fluid collections, if present

Syphilis (The Great Imitator)
- General Information
  - Organism: *Treponema pallidum* (spirochete)
  - Transmission occurs when moist skin contacts an infected area.
- Clinical Presentation
  - Primary syphilis
    - Chancre (see Image #15)
      - Occurs at the site of inoculation
      - Begins as a papule and then ulcerates
      - Typically a single lesion that is painless
      - Resolves spontaneously over 2 to 6 weeks
    - Lymphadenopathy may be seen in proximity to the lesion.
    - Absence of systemic symptoms
  - Secondary syphilis
    - Constitutional symptoms include fever, fatigue, myalgia, headache, and arthralgia.
    - Rash
      - Begins on the trunk as a fine macular rash, then spreads outward to the extremities (involves the palms and soles)
      - Becomes papulosquamous and slightly annular
    - Additional skin lesions may include condyloma lata or mucous patches (seen on the tongue).
    - Resolves spontaneously if not treated
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• Tertiary syphilis
  ▪ Occurs after 3–4 years of untreated infection
  ▪ Characterized by vascular (thoracic artery aneurysm) and CNS (meningitis, peripheral neuropathies) involvement

• Diagnosis
  o In patients with primary or secondary syphilis, rapid diagnosis can be obtained by demonstrating the spirochete in darkfield examination of scrapings of lesions.
  o Serologic testing
    ▪ Nontreponemal (VDRL, RPR)
      ▪ Becomes positive about 2 weeks after chancre
      ▪ Measures nonspecific antibodies
      ▪ Antibody titers are followed during treatment
    ▪ Treponemal (FTA-ABS)
      ▪ Measures specific antibodies to T. pallidum
      ▪ Main value is to confirm a nontreponemal test

• Treatment
  o Primary or secondary syphilis
    ▪ Penicillin G, 2.4 million units IM as a single dose
  o Tertiary syphilis
    ▪ Penicillin G, 2.4 million units IM for 3 doses separated by 1 week

Tuberculosis (TB)

• General Information
  o Organisms
    ▪ Mycobacterium bovis: drinking milk from diseased cows
    ▪ M. africanum: rare cause
    ▪ M. tuberculosis: the major causative agent of TB worldwide
  o Organism is an intracellular, aerobic, non-spore-forming bacillus.
  o Transmitted via respiratory droplets
  o Risk of transmission is greatest with airway and cavitary disease

• Clinical Presentation
  o Primary infection of TB is most often asymptomatic.
  o Reactivation is responsible for most clinical manifestations of TB.
  o Constitutional signs and symptoms include fever, night sweats, anorexia, weight loss, fatigue, malaise, and lymphadenopathy.
  o Pulmonary involvement is manifested by cough, hemoptysis (mild), dyspnea, and pleuritic chest pain.
Extrapulmonary TB is more common in infants, the elderly, and the immunocompromised; accounts for approximately 15% of new cases of TB in the United States each year; can present in any number of systems or structures:

- Lymph: lymphadenitis (scrofula)
- Spine: Pott's disease
- Joints: hip, knee, ankle, elbow, shoulders
- Renal tuberculoma: sterile pyuria is classic
- GU: epididymitis, prostatitis, orchitis
- CNS: tuberculous meningitis
- GI: peritonitis, perirectal abscess

Complications
- Complications of pulmonary TB include empyema, spontaneous pneumothorax, massive hemoptysis, and superinfection with organisms such as *Aspergillus*.
- Cardiac complications include pericarditis and pericardial effusion.

Diagnosis
- Routine lab studies are not helpful.
- PPD is of limited value in the ED for diagnosing active disease.
- Chest film is the most useful study in diagnosing pulmonary TB.
  - Primary TB
    - Infiltrate can occur in any lobe.
    - Mediastinal or hilar lymphadenopathy is present.
    - Can also see a pleural effusion
  - Reactivation TB
    - Upper lobe disease with or without cavitation
    - Infiltrates can also be seen in the upper segment of the lower lobe.
    - Immunocompromised patients often have atypical infiltrates, are less likely to have cavitation, and may have only mediastinal or hilar lymphadenopathy.
    - 21% of patients with AIDS have a normal chest film
- Sputum
  - Acid-fast bacillus (AFB) smear is the most rapid test available (a negative test does not rule out disease).
  - Culture is the traditional method of confirming the diagnosis (may take several weeks to detect a positive result).

Treatment
- Pulmonary TB
  - Preferred regimen consists of isoniazid (INH), rifampin, pyrazinamide, and ethambutol.
  - Side effects of INH include hepatitis, peripheral neuropathy, and seizures.
  - Rifampin can cause orange discoloration of bodily fluids.
  - Side effects of pyrazinamide include hepatotoxicity and polyarthritis.
  - Ethambutol can cause retrobulbar neuritis (red-green color blindness); patients need ophthalmologic evaluation.
- Extrapulmonary TB
  - Antimicrobial therapy is the same as for pulmonary TB but for a longer duration.
  - Corticosteroids can be used in pericardial and CNS disease.
Atypical Mycobacterial Infections

- *Mycobacterium avium* complex
  - Most often seen in immunocompromised patients (HIV/AIDS)
  - Clinically presents with weight loss, chronic diarrhea, malaise, and anorexia
  - Diagnosis is made with acid-fast stain of bodily fluids or through isolation of organism in blood.
  - Treatment
    - Regimen of choice is azithromycin and ethambutol.
    - The combination of clarithromycin and ethambutol is also acceptable.
    - Treatment does not eradicate organism; AIDS patients with CD4 <50 cells/mm³ require prophylaxis with azithromycin.

- *Mycobacterium kansasii*
  - Second most common cause of nontuberculous pulmonary disease
  - Occurs in severely immunocompromised patients
  - The clinical presentation includes fever, cough, night sweats, weight loss, and lymphadenopathy.
  - Radiographically, the disease is usually unilateral and most often seen as multiple cavities of varying size in the upper lobes.
  - Diagnosis is made by isolation of the organism in pulmonary secretions or any other sterile bodily fluid.
  - Treatment consists of INH, ethambutol, and rifampin.

- *Mycobacterium leprae*
  - Leprosy is a slowly progressive disease caused by *M. leprae*.
  - Endemic in subtropical areas (India, Ethiopia, Brazil)
  - Clinical hallmark is hypopigmented, or reddish, skin lesions with loss of sensation.
  - Also causes thickening of peripheral nerves with loss of sensation (ulnar and peroneal nerves are most commonly affected)
  - Diagnosis is made with an AFB skin smear and culture.
  - Treatment consists of dapsone and rifampin.

Diphtheria

- General Information
  - Organism: *Corynebacterium diphtheria*
    - Unencapsulated, club-shaped, gram-positive bacillus
    - Produces an exotoxin that causes the clinical features
  - Infection typically involves either the respiratory tract or skin
  - Endemic in Africa, South and Central America, Asia, and Eastern Europe

- Clinical Presentation
  - Upper respiratory tract symptoms
    - Sore throat
    - Dysphagia
    - Change in voice
    - Diphtheric membrane
      - Seen in more than 50% of patients
      - Begins with localized erythema that progresses to a thick *grayish membrane* with sharply defined borders
      - The membrane is adherent and will cause hemorrhage if forcibly removed.
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- Cutaneous symptoms
  - Skin lesion typically has an ulcer with a grayish membrane.
  - Cutaneous diphtheria generally does not have associated systemic toxicity.

- Complications
  - Airway tract obstruction – the most common cause of death
  - Cardiac complications can include myocarditis, endocarditis, congestive heart failure (CHF), and conduction abnormalities.
  - Polynuertis is a recognized complication manifested by proximal muscles weakness; can produce respiratory failure secondary to muscle paralysis

- Diagnosis
  - Routine laboratory tests are generally nonspecific and may reveal thrombocytopenia, leukocytosis, or proteinuria.
  - Cultures are the most common method of diagnosis
    - Cutaneous – obtain culture from sample of skin lesion
    - Respiratory tract – obtain throat or nasopharyngeal swab
  - Polymerase chain reaction (PCR) can be used to test for the exotoxin.

- Treatment
  - Respiratory diphtheria
    - Place in respiratory isolation
    - Antitoxin – obtained from the Centers for Disease Control (CDC)
    - Antibiotics – 14 days of treatment with erythromycin (IV or PO) OR penicillin (IV or IM)
  - Cutaneous diphtheria
    - Cleanse the skin lesion.
    - Antibiotics – oral erythromycin or penicillin
    - Antitoxin is controversial.
  - Post-exposure prophylaxis
    - Immunized >5 years ago – diphtheria toxoid booster
    - Unimmunized patients should be observed closely and should receive active immunization PLUS erythromycin or penicillin PO for 7 days.

Pertussis

- General Information
  - Organisms: *Bordetella pertussis* and *Bordetella parapertussis*
    - Non-motile, gram-negative coccobacilli
    - Produces several toxins responsible for clinical symptoms
  - Transmitted via airborne respiratory droplets
  - Superinfection with pneumonia is the leading cause of death.

- Clinical Presentation
  - *Catarrhal* phase lasts 1 to 2 weeks and is characterized by fever, cough, rhinorrhea, conjunctival injection, and anorexia; infants may present with apneic episodes, cyanotic spells, or bradycardia.
  - *Paroxysmal* phase lasts 2 to 4 weeks and is characterized by paroxysms of cough; paroxysms can occur up to 50 times per day and are associated with the “inspiratory whoop.”
  - *Convalescent* phase lasts several weeks to months and is characterized by a residual cough.
• Diagnosis
  o CBC, often with WBC count ranging from 25,000 to 50,000 cells/mm³ with lymphocytosis
  o Diagnosis is typically based on cultures of nasopharyngeal swabs.

• Treatment
  o Supportive care with oxygen, hydration, and avoidance of respiratory irritants
  o Antibiotic of choice is erythromycin for 14 days.
  o Vaccination – routine booster currently not recommended
  o Erythromycin prophylaxis should be prescribed for close contacts.

Tetanus
• General Information
  o Organism: *Clostridium tetani*
    ▪ Non-invasive, anaerobic, spore-forming, gram-positive rod
    ▪ Requires portal of entry to cause infection (wound)
    ▪ Produces exotoxins that are responsible for clinical features
    ▪ Tetanospasmin – binds to motor end plate and interferes with release of inhibitory neurotransmitters, leading to excessive uncontrolled muscle spasm

• Clinical Presentation
  o Four types: generalized, cephalic, localized, neonatal
  o Generalized tetanus
    ▪ Most common and most severe
    ▪ Trismus is most often the presenting symptom.
    ▪ Additional symptoms include weakness, myalgia, dysphagia, hydrophobia, risus sardonicus, and opisthotonos.
  o Cephalic tetanus (rare)
    ▪ Characterized by trismus and cranial nerve palsies
    ▪ Most common cranial nerve (CN) involved is VII
    ▪ Other CNs affected include III, IV, IX, X, and XII.
  o Localized tetanus
    ▪ Muscle spasm at the site of inoculation
  o Neonatal tetanus
    ▪ Typically occurs in underdeveloped countries as a result of the use of contaminated material to cut umbilical cord
    ▪ Symptoms include irritability and poor feeding.

• Complications
  o Acute respiratory failure: the most common cause of death
  o Autonomic dysfunction: hypertension, arrhythmias
  o Musculoskeletal: subluxations and fractures caused by forceful muscle contractions, rhabdomyolysis
  o Renal failure
  o GI: ileus, peptic ulcers, intestinal perforation

• Diagnosis
  o The diagnosis is based on clinical findings.
  o Wound cultures are rarely useful, as only one third are positive.
It is important to consider the differential diagnosis of tetanus:

- ENT disorders – peritonsillar/retropharyngeal abscess
- Mandibular dislocation
- Dystonic reactions
- Rabies
- Strychnine poisoning – trismus is a late finding; muscle rigidity alternates with relaxation

**Treatment**

- Supportive care, with particular attention to airway protection
- Muscle spasms should be treated with benzodiazepines; non-depolarizing neuromuscular blockers may be needed.
- Autonomic dysfunction:
  - Sympathetic overactivity – treat with combined α- and β-blocker (labetolol)
  - Bradycardias – treat with temporary pacing
- Wound care is essential and should include debridement and cleansing.
- Antitoxin/immunization
  - Administer both tetanus toxoid and human tetanus immunoglobulin.
  - Human tetanus immunoglobulin (TIG)
    - Does not treat symptoms but neutralizes any circulating toxin or toxin at the site of inoculation
    - Should be given early and at a different site from tetanus toxoid
    - No value in injecting directly into wound site
- Antibiotics of choice are metronidazole, penicillin G, or doxycycline.

**Tetanus Prophylaxis**

- Unimmunized adults should receive the primary series of vaccinations.
- Clean minor wounds.
  - If the primary series has been completed, with the last booster received >10 years ago, give tetanus, diphtheria vaccine (Td).
  - If it is not known if the primary series was received or completed, give Td.
- All other wounds
  - If the primary series was completed, with the last booster received >5 years ago, give Td.
  - If it is not known if the primary series was received or completed, give Td and TIG.

**Botulism**

- **General Information**
  - Organism: *Clostridium botulinum*
    - Anaerobic, gram-positive, rod-shaped organism
    - Secretes a potent neurotoxin that blocks pre-synaptic acetylcholine release
  - Exists primarily in three forms: food-borne, infant, and wound
  - Infant botulism is the most common form.
  - Wound botulism is increasing because of the use of black tar heroin.
  - Primary cause of death is respiratory failure.
- **Clinical Presentation**
  - Characterized by cranial nerve palsies; parasympathetic blockade; and a symmetric, descending muscle paralysis
Food-borne botulism
- Nonspecific symptoms: weakness, lethargy, and nausea
- Cranial nerve palsies: diplopia, dysphagia, and dysarthria
- Neuromuscular: symmetric descending weakness (including muscles of respiration)
- Parasympathetic blockade: postural hypotension, dilated/fixed pupils, dry mouth, constipation, urinary retention

Infant botulism
- Peak incidence between 2 and 4 months of age
- Caused by the ingestion of spores that produce toxin
  - Honey and corn syrup implicated as sources
- Constipation is typically the presenting symptom.
- Infants also have generalized weakness, hypotonia, weak suck, and poor feeding.

Wound botulism
- The toxin is generated at the wound site (longer incubation).
- GI symptoms are typically absent.

**Diagnosis**
- Initially a clinical diagnosis
- Diagnosis is ultimately confirmed by isolation of toxin from blood, stool, or wound or from a sample of ingested food.

**Treatment**
- Supportive care with particular attention to airway management
  - Predicted vital capacity <30% indicates need for intubation
- Antitoxin should be given as soon as possible; one vial is given IV and one vial is given IM; not recommended for infant botulism
- Antibiotics are not recommended, as they may cause toxin release.

8.2 BIOLOGIC WEAPONS

**Anthrax**
- **General Information**
  - **Organism:** *Bacillus anthracis*
    - Gram-positive, spore-forming bacillus
    - Releases toxins that are responsible for clinical symptoms (protective antigen, edema factor, and lethal factor)
  - Disease occurs when spores are inhaled, ingested, or inoculated into the skin.
  - Classically divided into inhalational (most lethal), cutaneous, and gastrointestinal anthrax
- **Clinical Presentation**
  - **Inhalational anthrax**
    - Illness begins with flu-like symptoms of fever, malaise, and non-productive cough.
    - Abrupt deterioration occurs over 24 to 48 hours, with symptoms of dyspnea, stridor, and cardiovascular collapse.
    - Death usually occurs within 72 hours.
Cutaneous anthrax ("wool sorter’s disease")
- Spores are inoculated into the skin through an open wound or abrasion.
- Initial skin lesion is an erythematous papule that quickly progresses to a large vesicle with significant surrounding edema.
- After 1 week, the lesion ruptures to form a black eschar.

Gastrointestinal anthrax
- Infection begins through ingestion of contaminated meat.
- Patients may present with fever, nausea, vomiting, and abdominal pain (secondary to mesenteric lymphadenitis).
- As the illness progresses, patients develop hematemesis, ascites, and hematochezia.

Complications
- Inhalational anthrax can cause hemorrhagic pleural effusions, respiratory failure, and hemorrhagic mediastinitis.
- Cutaneous anthrax can disseminate, resulting in fulminant disease.

Diagnosis
- Inhalational anthrax
  - Initial diagnosis is made on clinical grounds.
  - Chest film demonstrates widened mediastinum and hilar adenopathy.
  - Sputum gram stain and culture and blood cultures are not helpful until late in the disease course.
  - Anthrax can be confirmed with PCR of pleural fluid, detection of antibody to protective antigen, or immunohistochemical testing of biopsy specimens.

- Cutaneous anthrax
  - Primarily a clinical diagnosis
  - Diagnosis is established by isolation of the organism from a culture of the lesion or biopsy.

Treatment
- Cutaneous anthrax without systemic toxicity can be treated with oral ciprofloxacin, doxycycline, or amoxicillin for 7 to 10 days.
- Inhalational, gastrointestinal, or cutaneous anthrax with systemic toxicity should be treated with ciprofloxacin, doxycycline, or penicillin G IV until toxicity resolves, then oral treatment with the same antibiotic for an additional 60 days OR until the patient receives three doses of vaccine.
- Vaccination: anthrax vaccine given on days 0, 14, and 28.
- Close contacts should be given oral ciprofloxacin or doxycycline for 60 days or until the patient receives all three doses of vaccine.

Plague
- General Information
  - Organism: *Yersinia pestis*
    - A gram-negative bacillus
  - Endemic in the western half of the United States
  - Exists in three forms: pneumonic, bubonic, and septicemic plague
  - Animal reservoir: dogs, cats, and rodents
  - Transmitted via the bite of an infected flea or inhalation
  - Pneumonic plague is most pertinent to bioterrorism
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• Clinical Presentation
  o Pneumonic plague
    ▪ Initial symptoms are nonspecific and include the abrupt onset of fever, chills, myalgias, arthralgias, and headache.
    ▪ Within 24 hours, fulminant disease develops and includes pneumonia (usually lobar), respiratory failure, circulatory collapse, DIC, and acral gangrene (most notable on the fingers, toes, and nose).
  o Bubonic plague
    ▪ Occurs when organism is inoculated directly into skin
    ▪ Clinically characterized by large, painful lymph nodes ("buboes") located predominantly in the groin, axilla, or cervical region
    ▪ Nonspecific symptoms such as fever, chills, and malaise may also be seen.
    ▪ In 50% of patients, the organism disseminates, causing either pneumonic or septicemic plague.
  o Septicemic plague is characterized by DIC, cardiovascular collapse, coma, and death.

• Diagnosis
  o Initial diagnosis is made on clinical grounds since laboratory tests require several days to complete.
  o Diagnosis is confirmed using cultures of the sputum, blood, CSF, or lymph node aspirate.

• Treatment
  o All patients should be placed in respiratory isolation, as pneumonic plague can be transmitted from human to human.
  o Antibiotics are the same for all three forms and should initially be given IV; streptomycin, gentamicin, doxycycline, ciprofloxacin, and chloramphenicol are acceptable choices.
  o Post-exposure prophylaxis should be provided for 7 to 10 days; acceptable antibiotics include oral tetracycline, doxycycline, ciprofloxacin, and chloramphenicol.

Smallpox

• General Information
  o Organism: Variola virus
  o The organism is spread via aerosol droplets and is highly infectious.
  o Classically divided into variola major, variola minor, hemorrhagic smallpox, and malignant smallpox

• Clinical Presentation
  o After inhalation, the virus migrates to the lymph nodes and, after several days, migrates to the spleen, liver, and other lymphoid tissue.
  o 7 to 10 days later, viremia occurs, characterized by fever, chills, headache, and malaise
  o The characteristic rash of smallpox occurs after the viremia and begins on the face and forearms; initially maculopapular, it becomes vesicular, and eventually pustular; all lesions are of the same stage and age

• Diagnosis
  o Initial diagnosis is based on clinical findings.
  o Can be confirmed with examination of the vesicular fluid or scabs

• Treatment
  o No effective therapy currently exists.
  o There is currently no role for vaccinia immunoglobulin (VIG) in patients infected with smallpox.
  o A susceptible population should be vaccinated within 3 days after exposure; susceptible persons should also receive VIG.
Tularemia

• General Information
  o Organism: *Francisella tularensis*
  o Animal reservoirs include ticks, lagomorphs (rabbits), and rodents.
  o In the United States, infection is most often transmitted via the bite of an infected deer tick, a Lone Star tick, or a dog tick.
  o Patients at most risk are hunters, trappers, campers, butchers, and laboratory workers.
  o Has recently gained attention as a possible biologic weapon because infection via inhalation of dust or water aerosol can occur

• Clinical Presentation
  o The clinical presentation depends on the site of inoculation.
    - Ulceroglandular is the most common; an erythematous papule that ulcerates over 2 to 3 days is noted at the site of the bite; regional lymphadenopathy develops and nodes may rupture and suppurate
    - Glandular tularemia is characterized by lymphadenopathy without associated skin lesions
    - Typhoidal tularemia accounts for approximately 10% of cases; characterized by fever, chills, abdominal pain, diarrhea, and weight loss
    - Pulmonary tularemia is characterized by fever, chills, non-productive cough, dyspnea, malaise, and fatigue.

• Complications
  o Pericarditis
  o Endocarditis
  o Meningitis
  o Appendicitis
  o Osteomyelitis

• Diagnosis
  o Based on clinical findings and a fourfold rise in antibody titers
  o Aspiration of lymph nodes is not recommended, given the transmission risk to health care personnel.

• Treatment
  o Antibiotic of choice is streptomycin; gentamicin, tetracycline, chloramphenicol, and imipenem are less effective but acceptable alternatives.
  o Post-exposure prophylaxis – doxycycline for 14 days

8.3 FUNGAL INFECTIONS

Mucormycosis

• General Information
  o Mucormycosis is caused by a variety of fungal organisms, of which the *Rhizopus* species are the most common.
  o Infection is typically seen only in the immunocompromised.
  o Patients at greatest risk are those with diabetes, steroid use, organ transplants, neutropenia, or hematologic malignancies.
  o Routes of infection are inhalation, ingestion, and direct inoculation.
  o Once infection begins, the fungal hyphae invade blood vessels, producing tissue infarction, necrosis, and thrombosis.
Clinical Presentation
- Clinical presentation depends on the site of infection.
  - Rhinocerebral disease (most common) is characterized by fever, headache, facial pain, and nasal stuffiness that progresses to a black discharge; orbital and facial swelling may also be seen.
  - Pulmonary involvement manifests as fever, cough, hemoptysis, and dyspnea.
  - Cutaneous infection is characterized by cellulitis that progresses to necrosis with black eschar formation.
  - Gastrointestinal infection can occur and is manifested by abdominal pain, nausea, vomiting, and hematochezia.

Complications
- Rhinocerebral disease can produce significant complications from direct extension of infection; meningitis, cavernous sinus thrombosis, brain abscess, and epidural or subdural empyema can be seen.
- Gastrointestinal infection can be complicated by perforation leading to peritonitis.

Diagnosis
- Initially, the diagnosis is based on clinical findings.
- Diagnosis is confirmed with a biopsy of the affected tissue; staining of the sample reveals broad, irregular, non-septate, right-angled, branching hyphae.

Treatment
- Prompt surgical consultation for debridement
- Antifungal therapy with amphotericin B

Aspergillus

General Information
- Most cases of aspergillosis in humans are caused by *Aspergillus fumigatus*.
- Invasive infection most often occurs in the immunocompromised.

Clinical Presentation
- There are four types of clinical presentation:
  - Allergic bronchopulmonary aspergillosis (ABPA)
    - Typically found in asthmatics, those on chronic steroids, and patients with cystic fibrosis
    - Symptoms include fever, cough, hemoptysis, wheezing, and pulmonary consolidation not responsive to antibiotic therapy.
  - Aspergilloma
    - Typically an asymptomatic cavitary lesion in patients with sarcoidosis, TB, or pneumocystis carinii pneumonia (PCP)
  - Chronic necrotizing *Aspergillus* pneumonia
    - Occurs in alcoholics and patients with COPD
    - Clinically presents as fever, night sweats, weight loss, and pneumonia unresponsive to antibiotics over months
  - Invasive aspergillosis
    - Mortality ranges from 30% to 95%
    - Typically occurs in bone marrow transplant recipients and individuals with leukemia or lymphoma
    - Clinically presents with fever, cough, dyspnea, chest pain, and hemoptysis

Complications are primarily related to invasive pulmonary disease and include massive, life-threatening hemoptysis and respiratory failure.
**Diagnosis**
- Requires isolation of the organism in tissue samples; hallmark is the isolation of hyphae that branch at 45° angles

**Treatment**
- Invasive aspergillosis: voriconazole or caspofungin
- Aspergilloma: itraconazole and surgical consultation for resection
- Allergic bronchopulmonary aspergillosis: corticosteroids
- Chronic necrotizing *Aspergillus* pneumonia: IV voriconazole, caspofungin, or amphotericin B

**Histoplasmosis**
- **General Information**
  - Organism: *Histoplasma capsulatum*
  - Endemic in the Ohio, Missouri, and Mississippi River valleys
  - The fungus grows well in damp soil enriched by bird droppings.
  - Infection is acquired via inhalation of conidia and mycelial fragments from contaminated soil.
- **Clinical Presentation**
  - The majority of patients are asymptomatic.
  - Nonspecific symptoms of infection are flu-like and include fever, chills, malaise, myalgia, and headache.
  - Pulmonary disease presents with cough, dyspnea, hypoxia, and hemoptysis (usually mild); effusions and ARDS can be seen in severe disease.
- **Complications**
  - Disseminated disease can result in cardiac (pericarditis, tamponade, endocarditis, CHF), CNS (altered mental status, meningitis, seizures), GI (diarrhea), and ocular (blindness) complications
- **Diagnosis**
  - Blood and sputum cultures have a variable yield.
  - Urine and serum antigen assays have sensitivities ranging from 50% to 90%.
  - Serology is the most useful test, demonstrating a rise in antibody titers.
- **Treatment**
  - In immunocompetent patients, the infection is usually self-limited and does not require therapy.
  - Administer ketoconazole, itraconazole, or amphotericin B to immunocompromised patients or immunocompetent patients with systemic complications.

**Coccidioidomycosis**
- **General Information**
  - Organism: *Coccidioides immitis*
  - Endemic in Arizona, Nevada, New Mexico, the western half of Texas, and south central California
  - Peak incidence of infection occurs during the dry summer months
  - Infection is acquired through inhalation of arthrospores.
  - Human-to-human transmission does not occur.
- **Clinical Presentation**
  - The majority of patients are asymptomatic.
  - In symptomatic patients, the most common symptoms are fever, cough, dyspnea, fatigue, and arthralgia.
Patients who develop disseminated disease can have
- Skin involvement, as demonstrated by maculopapular lesions, erythema nodosum, or erythema multiforme
- Pulmonary involvement with pleural effusions or empyema
- Hepatomegaly and/or splenomegaly
- Synovitis or osteomyelitis
- CNS infection with meningitis, resulting in hydrocephalus

- Diagnosis
  - Isolation of the organism from sputum establishes the diagnosis.
  - Special serology studies must be used for definitive diagnosis in patients with negative sputum cultures.

- Treatment
  - The majority of patients have self-limited disease and do not require treatment.
  - In patients with systemic involvement, oral antifungal therapy with fluconazole, itraconazole, or ketoconazole is indicated.
  - Amphotericin B is indicated for refractory disease.

**Blastomycosis**

- General Information
  - Organism: *Blastomyces dermatitidis*
  - Endemic in the central and southeastern parts of the United States
  - Infection is acquired via inhalation of conidial forms of the fungus.
  - Once inhaled, the conidial form transforms to the yeast phase and can disseminate via the blood or lymphatics to other organs.

- Clinical Presentation
  - Infection begins with a flu-like illness consisting of fever, chills, myalgia, headache, and a non-productive cough.
  - Pulmonary involvement is manifested by a productive cough, dyspnea, hypoxia, tachypnea, and diffuse pulmonary infiltrates.
  - Disseminated disease is indicated by skin lesions (sharply demarcated papules on the face, neck, and extremities that ulcerate), lytic bone lesions, and GU symptoms (prostatitis).

- Diagnosis
  - Staining of sputum or skin lesions reveals the characteristic broad-based, budding yeast.
  - Culture of sputum confirms the diagnosis.
  - Serologic studies are not useful in blastomycosis.

- Treatment
  - Patients with severe pulmonary or systemic involvement should be treated with amphotericin B or itraconazole.

### 8.4 PROTOZOA/PARASITIC INFECTIONS

**Protozoa**

- Malaria
  - General information
    - Organism: *Plasmodium* species (*P. falciparum, P. vivax, P. ovale, and P. malariae*)
      - *P. falciparum* is responsible for most severe disease.
      - *P. vivax* and *P. ovale* are responsible for recrudescent disease.
Vector is the female *Anopheles* mosquito.

- Endemic in Asia, Africa, Central America, and South America
- Life cycle: sporozoites invade hepatic cells and multiply → hepatic cells lyse and release merozoites → merozoites invade RBCs and transform to trophozoites → trophozoites feed on hemoglobin and transform to schizonts → schizonts divide into more merozoites that are released upon RBC lysis

**Clinical presentation**
- Initially, the patient has nonspecific symptoms of fever, chills, headache, nausea, lethargy, and upper respiratory symptoms.
- Infection with *P. falciparum* can further present with
  - Cerebral malaria: altered mental status, seizures
  - Severe anemia
  - Noncardiogenic pulmonary edema
  - Renal failure
  - DIC

**Diagnosis**
- Thick and thin peripheral blood smears demonstrating organism

**Treatment**
- *P. falciparum*
  - Resistance to chloroquine is increasing
  - Quinine + doxycycline are drugs of choice
  - Quinine + clindamycin OR mefloquine are acceptable alternatives
  - Rapid infusions of intravenous quinine can cause hypoglycemia.
- *P. vivax, P. ovale,* and *P. malariae*
  - Chloroquine is the drug of choice.
  - Primaquine is used to eradicate hepatic phases of *P. vivax* and *P. ovale.*
  - Patients should be checked for G6PD deficiency before starting primaquine.
- Patients with >3% parasitemia should be admitted to an ICU.

**Amebiasis**

**General information**
- Organism: *Entamoeba histolytica*
- Infection occurs through ingestion of cysts contained in contaminated food or drink
- Organism colonizes the cecum and colon

**Clinical presentation**
- Amebic dysentery is characterized by fever, tenesmus, abdominal pain, and watery stool mixed with blood and mucus.
- Hepatic abscesses present with fever, weight loss, anorexia, right upper quadrant pain; typically patients do not have jaundice.
- CNS disease is rare but can present with brain abscesses or meningoencephalitis.
- Sympathetic pleural effusions can also be seen as a result of either direct pulmonary infection or hematogenous seeding.

**Diagnosis**
- Stool examination for mobile trophozoites
- In patients with CNS disease, biopsy of tissue is more specific.
o Treatment
  ▪ Antibiotic of choice is metronidazole.

Giardiasis
  • General Information
    o Organism: *Giardia lamblia*
    o Most common intestinal parasite in the United States
    o Infection occurs through ingestion of cysts (e.g., a camper who drinks unfiltered mountain spring water or day-care-center outbreaks)
    o Organism colonizes the duodenum and jejunum
  • Clinical Presentation
    o Abdominal cramping
    o Flatulence
    o Explosive malodorous diarrhea
    o Weight loss
  • Diagnosis
    o Stool examination for cysts and trophozoites
    o Can also do direct immunofluorescence or ELISA of stool
  • Treatment
    o Antibiotic of choice is metronidazole.

Trypanosomiasis
  • General Information
    o Organisms
      ▪ *Trypanosoma cruzi*: Chagas’ disease
      ▪ *T. gambiense* and *T. rhodesiense*: African sleeping sickness
    o Vectors
      ▪ Reduviid bug ("kissing bug"): Chagas’ disease
      ▪ *Glossina* (tsetse fly): African sleeping sickness
    o Regions
      ▪ Chagas’ disease: Central and South America
      ▪ African sleeping sickness: East and West Africa
  • Clinical Presentation
    o Nonspecific symptoms include fever, malaise, lethargy, headache, and posterior cervical lymphadenopathy.
    o Chagas’ disease
      ▪ Chagoma is a nodular swelling at the site of the bite. It is most often noted in the periorbital region and causes facial swelling.
      ▪ Cardiac manifestations of Chagas’ disease include conduction abnormalities (atrial and ventricular arrhythmias, bundle branch block, complete heart block) and CHF.
      ▪ GI symptoms include hepatosplenomegaly and elevated aminotransferase levels.
    o African sleeping sickness presents with extreme fatigue and lethargy. Many patients also have psychiatric symptoms.
  • Diagnosis
    o Demonstration of motile trypomastigotes in peripheral blood smear, CSF, or lymph node aspirates
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• Treatment
  o Drug of choice for *T. cruzi* is nifurtimox.
  o *T. gambiense* and *T. rhodesiense* are treated with suramin sodium.

Nematodes
• Trichinosis
  o General information
    ■ Organism: *Trichinella spiralis*
    ■ Infection occurs through ingestion of infected pork.
    ■ Primary lesions are in striated muscle.
  o Clinical presentation
    ■ Can be asymptomatic
    ■ Symptomatic patients can present with myalgia, anorexia, nausea, and vomiting.
  o Complications
    ■ Myocarditis
    ■ Pneumonia
    ■ Meningitis
    ■ Encephalitis
    ■ Seizures
  o Diagnosis
    ■ Diagnosis can be made with biopsy of affected muscle or serology.
    ■ Examination of stool is not helpful in patients without gastrointestinal symptoms.
  o Treatment
    ■ Drug of choice is mebendazole.
    ■ Corticosteroids may be required in severe disease (CNS involvement).

• *Strongyloides*
  o General information
    ■ Organism: *Strongyloides stercoralis*
    ■ Worms penetrate the skin and enter small cutaneous venules.
    ■ Organism resides in the small intestine.
    ■ Infection is clinically significant in the immunocompromised.
  o Clinical presentation
    ■ Skin demonstrates dermatitis at the entry point.
    ■ GI involvement is indicated by abdominal pain, weight loss, and persistent diarrhea.
    ■ Pulmonary disease presents with cough, dyspnea, and respiratory failure in patients with massive infection.
  o Complications
    ■ Respiratory failure
    ■ CNS disease (pyogenic meningitis and encephalitis)
  o Diagnosis
    ■ Confirmed with demonstration of larvae in stool.
  o Treatment
    ■ Drug of choice is thiabendazole.
    ■ Ivermectin is an acceptable alternative.
- **Hookworm**
  - General information
    - Organism: *Necator americanus*
    - Major cause of anemia worldwide
    - Worms penetrate the skin (usually the feet) and enter small venules.
    - Eventually, adult worms penetrate intestinal mucosa and feed there.
  - Clinical presentation
    - Classic presentation is iron-deficiency anemia due to blood loss.
  - Diagnosis
    - Identification of ova in the stool
  - Treatment
    - Drug of choice is mebendazole.
    - Pyrantel pamoate is an acceptable alternative.

- **Whipworm**
  - General information
    - Organism: *Trichuris trichiura*
    - Seen in warm climates (tropics, southern United States)
    - Worms penetrate through skin and reside in the cecum.
  - Clinical presentation
    - Fever
    - Abdominal pain
    - Anorexia
    - Bloody diarrhea
    - Iron-deficiency anemia
  - Diagnosis
    - Demonstration of ova in the stool
  - Treatment
    - Drug of choice is mebendazole.
    - Albendazole is an acceptable alternative.

- **Pinworm**
  - General information
    - Organism: *Enterobius vermicularis*
    - Infection acquired through ingestion of eggs
    - Organism resides in ileum, cecum, appendix, and ascending colon.
  - Clinical presentation
    - Intense perianal pruritus, which becomes worse at night due to female worm migrating to the anus to deposit eggs
  - Diagnosis
    - Primarily clinical
    - Confirmed by finding worms on the anal verge
    - “Scotch tape test” occasionally picks up eggs from the anal region
  - Treatment
    - Drugs of choice are mebendazole and pyrantel pamoate.
Ascariasis
  o General information
    ▪ Organism: *Ascaris lumbricoides*
    ▪ Infection is acquired through ingestion of larval eggs.
    ▪ Larvae penetrate small intestine, gain entry to venules, and migrate to the lungs.
  o Clinical presentation
    ▪ Pulmonary signs and symptoms include a non-productive cough, wheezing, rales, and infiltrative disease on chest film.
    ▪ Gastrointestinal symptoms include abdominal pain and anorexia.
    ▪ Children can present with a partial small bowel obstruction.
  o Diagnosis
    ▪ Identification of eggs or adult worms in the stool
  o Treatment
    ▪ Drugs of choice are mebendazole and pyrantel pamoate.

Elephantiasis
  o General information
    ▪ Organism: *Wuchereria bancrofti*
    ▪ Majority of cases are seen in Asia, Africa, and South America.
    ▪ Bite of infected mosquitoes introduce worm into the bloodstream.
    ▪ Worms migrate to the lymphatic system, where they induce an inflammatory reaction.
  o Clinical presentation
    ▪ The characteristic presentation is massive peripheral edema due to mechanical obstruction of the lymphatic system.
    ▪ Patients may also have pulmonary involvement (tropical eosinophilic pneumonia) manifested by fever, malaise, dyspnea, and nocturnal wheezing.
    ▪ Chest film demonstrates nodular infiltrates.
  o Diagnosis
    ▪ Demonstration of microfilariae in thick peripheral blood smears
  o Treatment
    ▪ Drug of choice is diethylcarbamazine.
    ▪ Surgical treatment is rarely helpful in relieving the obstruction.

Cestodes
  • Cysticercosis
    o General information
      ▪ Organisms
        ▪ *Taenia solium*: pork tapeworm
        ▪ *Taenia saginata*: beef tapeworm
      ▪ Infection is acquired through ingestion of food/drink contaminated with eggs.
      ▪ Organism resides in small bowel and is tropic for muscle and CNS tissue.
    o Clinical presentation
      ▪ Patients may be asymptomatic.
      ▪ Symptomatic patients present with nausea, vomiting, abdominal pain, headache, and seizures.
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- Fish Tapeworm
  - General information
    - Organism: *Diphyllobothrium latum*
    - Infection occurs through ingestion of raw fish that contain larva.
    - Organism resides in small intestine.
  - Clinical presentation
    - Classic presentation is pernicious anemia (organism competes for absorption of vitamin B12).
  - Diagnosis
    - Demonstration of ova in stool
  - Treatment
    - Drug of choice is praziquantel.

Trematodes
- Schistosomiasis
  - General information
    - Organisms are *Schistosoma haematobium*, *Schistosoma japonicum*, and *Schistosoma mansoni*.
    - Adult worm resides in the venous system.
  - Clinical presentation
    - “Katayama fever” is characteristic of schistosomiasis, with typical features of fever, cough, and diaphoresis.
    - Hepatosplenomegaly and portal hypertension are seen with GI involvement.
    - Involvement of the genitourinary system, primarily with *S. haematobium*, causes hematuria and obstructive hydrourerter.
    - Pulmonary disease is demonstrated with diffuse pulmonary nodules on chest film; pulmonary hypertension with cor pulmonale caused by venous obstruction by the worm can be seen.
  - Diagnosis
    - Demonstration of eggs in the stool
  - Treatment
    - Drug of choice is praziquantel.

8.5 TICK-BORNE ILLNESSES

Rocky Mountain Spotted Fever (RMSF)
- General Information
  - Organism: *Rickettsia rickettsii*
  - Obligate intracellular bacterium that initially invades the vascular endothelial cells
  - Tick vector is the *Dermacentor* tick.
  - Most prevalent in the southeastern United States
  - Associated with significant morbidity and mortality
Clinical Presentation

- Nonspecific symptoms include fever, lethargy, and myalgia (especially in large muscle groups).
- Cutaneous manifestations
  - Characteristic rash
  - Begins around the 4th febrile day
  - Skin lesions initially begin as pink, irregular macules on the wrists, ankles, palms, soles, and forearms. (see Image #42)
  - Over 6 to 12 hours, the rash spreads centripetally and becomes palpable and non-blanching.
  - Approximately 10% of patients do not present with the characteristic rash.
- Cardiac manifestations include tachycardia, atrial fibrillation, first-degree atrioventricular block (AVB), and paroxysmal atrial tachycardia.
- Pulmonary manifestations of RMSF include interstitial pneumonitis, pulmonary edema, and pleural effusions.
- Neurologic presentations include meningitis, thrombovasculitis (seizures), ataxia, and aphasia.
- Gastrointestinal symptoms are nausea, vomiting, and abdominal pain (caused by myositis of the abdominal wall muscles).

Diagnosis

- Routine laboratory tests may show hyponatremia, thrombocytopenia, and/or anemia.
- Serology demonstrates a fourfold rise in antibody titers between acute and convalescent samples (takes several weeks).
- Skin biopsy is the most rapid method of confirmation; requires a punch biopsy from the center of a lesion.
- Isolation of organism in blood or tissue samples is rarely attempted.

Treatment

- Antibiotic therapy should be initiated at the appearance of the rash.
  - Drugs of choice
    - Tetracycline: 2 gm/day PO OR
    - Doxycycline: 100 mg PO BID OR
    - Chloramphenicol: 50 mg/kg/day PO or IV
  - Treat for 7 to 10 days or until the patient is afebrile for at least 48 hours.
  - Avoid sulfonamides, as they may worsen the primary infection.
- Steroids are controversial and not routinely recommended.
- Isolation is not required.

Lyme Disease

- General Information
  - The most common vector-borne disease in the United States
  - Organism: *Borrelia burgdorferi* (spirochete)
    - Tropic for skin, CNS, and synovial tissue
  - Tick vector: *Ixodes* species
    - *Ixodes scapularis* – Northeast and Midwest
    - *Ixodes pacificus* – West
  - Peak incidence between May and August
  - Less than one third of affected individuals recall tick bite
• Clinical Features
  o Early localized Lyme disease – incubation period approximately 1 week
    ▪ Erythema migrans (EM, see Image #40)
      □ Most characteristic clinical manifestation
      □ Present in approximately 90% of patients
      □ Common locations of rash are groin, popliteal fossa, gluteal folds, axillary folds, and ear lobes
      □ Begins as erythematous papule/macule and spreads gradually (1 to 2 cm/day)
      □ Can appear oval, round, triangular, elongated
      □ Central clearing not always present
      □ Can have secondary lesions that are smaller and typically spare the palms/soles
    ▪ Constitutional symptoms are common and include malaise, fatigue, lethargy, headache, and low-grade fever
    ▪ Patients may also have GI symptoms of anorexia, fatigue, vomiting, and RUQ tenderness.
    ▪ Arthralgia and myalgia are also common.
  o Early disseminated Lyme disease – approximately 4 weeks after onset of EM
    ▪ Neurologic manifestations (seen in 15% of untreated patients) consist of meningoencephalitis (most common), cranial nerve palsies (CN VII most common), and peripheral neuropathies.
    ▪ Cardiac symptoms occur in 4% to 10% of untreated patients and include AVB of varying degrees, myopericarditis, tachydysrhythmias, and ventricular impairment.
    ▪ Monoarticular or oligoarticular arthritis typically affects large joints such as the knee.
    ▪ Ophthalmic signs and symptoms can occur during this stage and include conjunctivitis, keratitis, choroiditis, optic neuritis, and retinal detachment.
  o Late Lyme disease
    ▪ Arthritis that is exacerbating and remitting over years
    ▪ Neurologic manifestations include chronic encephalopathy (memory disturbance, depression, paranoia) and sensory polyradiculoneuropathy.
    ▪ Acrodermatitis chronica atrophicans can be found on the distal extremities at the site of the bite.
• Diagnosis
  o Primarily based on clinical and epidemiologic features
  o Routine lab studies are nonspecific and generally not helpful.
  o Cultures (blood, CSF, synovial) have low yield.
  o PCR and urine antigen testing have not been clinically validated.
  o Serologic testing most practical
    ▪ IgM – peaks between 3 and 6 weeks after onset
    ▪ IgG – detectable 2 months after onset
    ▪ ELISA has a sensitivity of approximately 90%
• Treatment
  o Asymptomatic tick bite
    ▪ Routine antibiotic therapy is not indicated, even in endemic areas.
    ▪ Most important is patient education regarding signs and symptoms
  o Early localized disease in men, nonpregnant and nonlactating women, and children >8 years is treated with oral doxycycline for 14 to 21 days
  o Pregnant women and children <8 years with early localized disease are given amoxicillin.
Treatment of early disseminated disease depends on manifestations.

- **Neurologic**
  - Isolated CN VII palsy with normal CSF can be treated with doxycycline or amoxicillin for 30 days.
  - Patients with meningoencephalitis, peripheral neuropathies, or cranial nerve palsies other than CN VII should receive ceftriaxone IV for 28 days.
- Patients with cardiac manifestations should be given ceftriaxone IV for 28 days; temporary pacing may be required for high-grade block.

Treatment of late disease remains controversial and includes various regimens of ceftriaxone or doxycycline; this stage is typically resistant to therapy.

**Ehrlichiosis**

- **General Information**
  - Two forms of ehrlichiosis exist in the United States: human monocytic ehrlichiosis (HME) and human granulocytic ehrlichiosis (HGE).
  - Organism is a gram-negative, obligate, intracellular rickettsia-like, coccobacilli.
    - **HME:** *Ehrlichia chaffeensis*
    - **HGE:** *Ehrlichia phagocytophila* and *Ehrlichia equi*
  - Tick vector
    - **HME:** *Amblyomma americanum*
    - **HGE:** *Ixodes scapularis* and *Ixodes pacificus*
  - Peak incidence is from June through August.

- **Clinical Presentation**
  - Nonspecific symptoms include fever, headache, malaise, myalgia, nausea, and vomiting.
  - Meningitis, pancarditis, renal failure, and DIC have also been reported.

- **Diagnosis**
  - Primarily based on clinical presentation
  - Labs demonstrate leukopenia, thrombocytopenia, and elevated liver function tests.
  - Peripheral smear may demonstrate morulae inside leukocytes.
  - Most common method of diagnosis is a fourfold increase in the titer of IgG antibody on immunofluorescence staining.

- **Treatment**
  - Doxycycline for 7 to 14 days.

**Babesiosis**

- **General Information**
  - Organism: *Babesia microti* and *Babesia equi*
    - Intraerythrocytic protozoan infection
  - Tick vector: *Ixodes species*
  - Peak incidence is between May and August.
  - Illness is more severe in patients with splenectomies.

- **Clinical Presentation**
  - Nonspecific symptoms are fever, headache, fatigue, myalgia, anorexia, and nausea.

- **Diagnosis**
  - Examination of thick and thin peripheral smear reveals intraerythrocytic ring forms.
    - Parasites in a budding tetrad formation: “Maltese cross”
Lab test results demonstrate anemia, evidence of hemolysis (elevated bilirubin, elevated LDH), liver dysfunction, and renal failure.

Serologic testing and PCR are also available.

- **Treatment**
  - Patients who have had a splenectomy require oral quinine and IV clindamycin for 7 to 10 days.
  - Patients who have not undergone splenectomy generally recover with supportive therapy.

### Colorado Tick Fever

- **General Information**
  - Endemic to the Rocky Mountain area at altitudes >4,000 feet
  - Organism: Orbivirus species
  - Tick vector: Dermacentor andersoni

- **Clinical Presentation**
  - Nonspecific symptoms are flu-like and include fever, headache, myalgias, anorexia, nausea, and a maculopapular rash (rare).
  - Fever follows a characteristic “biphasic” course over several days.

- **Diagnosis**
  - Primarily a clinical diagnosis
  - Confirmed with serologic testing of acute and convalescent titers
  - Lab tests demonstrate leukopenia and thrombocytopenia.

- **Treatment**
  - Supportive, as virtually all patients recover without sequelae

### Tick Paralysis

- **General Information**
  - Organism: 43 species of ticks found to cause tick paralysis
  - Pathophysiology
    - Secretion of a neurotoxin that results in acute cerebellar ataxia and/or ascending paralysis
    - Neurotoxin thought to block release of acetylcholine at the neuromuscular junction
  - Most commonly occurs in children during the spring and summer
  - Differential includes Guillain-Barré syndrome, Eaton-Lambert syndrome, myasthenia gravis, botulism, diphtheria, and polio.

- **Clinical Presentation**
  - An acute ascending paralysis characterized by loss of deep tendon reflexes; can progress rapidly to involve the bulbar muscles and cause respiratory failure

- **Diagnosis** is based on clinical findings.

- **Treatment** is simply to remove the tick (improvement seen in hours).

### 8.6 VIRAL

#### Influenza

- **General Information**
  - Three types: A, B, and C
  - Type A is associated with epidemics and has the highest mortality.
  - The very young and old are at highest risk of morbidity and mortality.
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- **Clinical Presentation**
  - The clinical presentation comprises constitutional symptoms: fever, myalgia, headache, coryza, and a non-productive cough.

- **Diagnosis**
  - Diagnosis of influenza is initially based on clinical findings.
  - Viral culture is the gold standard but is not helpful in the ED.
  - Can confirm the diagnosis via serology or isolation of virus from nasal secretions

- **Treatment**
  - Primarily supportive measures (avoid aspirin in children and adolescents – Reye syndrome)
  - Neuraminidase inhibitors can be given.
    - Zanamavir and oseltamivir are approved for influenza A and B.
    - These medications should be started within 48 hours.
    - Adjust dose for impaired creatinine clearance.
  - Amantadine is approved for influenza A; however, it is less effective than the neuraminidase inhibitors and has significant side effects.

**Parainfluenza**

- **General Information**
  - Infection is acquired through contact with respiratory secretions.
  - Typically occurs during the fall and spring months
  - Most common cause of croup in children (parainfluenza type 1)

- **Clinical Presentation**
  - The clinical presentation is mainly nonspecific symptoms that include fever, cough, and rhinorrhea.

- **Diagnosis**
  - The diagnosis of parainfluenza is based on clinical findings.
  - If serologies are sent, there is a fourfold rise in antibody titer between acute and convalescent serum samples.

- **Treatment**
  - Treatment of parainfluenza is supportive care. For additional information on the treatment of croup, see Chapter 5, Pediatrics.

**Hantavirus**

- **General Information**
  - Infection occurs through inhalation of aerosols contaminated with rodent urine or feces.
  - Animal reservoir is the deer mouse.
  - Seen more commonly in the southwestern United States
  - Death occurs from decreased cardiac output and circulatory failure.

- **Clinical Presentation**
  - Initial symptoms are nonspecific: fever, malaise, and myalgia.
  - Pulmonary symptoms follow the initial prodrome and include tachypnea, hypoxia, and respiratory distress; pulmonary involvement can rapidly progress to ARDS.

- **Diagnosis**
  - The diagnosis must initially be made clinically.
  - Lab tests may reveal nonspecific findings of thrombocytopenia, hemoconcentration, and renal failure.
  - Chest film will demonstrate bilateral interstitial infiltrates.
  - PCR can demonstrate hantavirus-specific antibodies.
There is no specific therapy for hantavirus infection.
Treatment is primarily supportive, with attention to respiratory status and oxygenation.

Herpes Simplex

- General Information
  - Viruses: Herpes simplex 1 and 2 (HSV-1 and HSV-2)
  - Both can infect any mucous membrane; however:
    - HSV-1 is typically associated with oral lesions
    - HSV-2 is typically associated with genital lesions
  - Virus invades and replicates in epithelial cells of abraded skin or mucous membranes.
  - Reactivation of disease can be triggered by emotional stress, infection, local trauma, and sunlight.

- Clinical Presentation
  - Characteristic lesions are thin-walled vesicles on a mildly erythematous base.
  - Oropharyngeal lesions are usually seen at the vermillion border and can be associated with pharyngitis and gingivostomatitis.
  - Patients may present with ocular involvement, as evidenced by conjunctivitis, blepharitis, and corneal opacities.
  - Herpetic whitlow is a lesion on the distal pulp space of a digit.
  - Genitourinary
    - Men: lesions typically located on the glans or shaft of penis (see Image #19)
    - Women: lesions seen on the vulva, perineum, vagina, or cervix
    - Urethral involvement may present as acute urinary retention.
  - Encephalitis
    - Most common acute, non-epidemic encephalitis in the United States
    - Typically caused by HSV-1
    - Symptoms include headache, lethargy, confusion, and psychiatric disturbances.
    - The virus typically localizes to the temporal lobe.

- Diagnosis
  - Diagnosis is based on clinical findings.
  - If obtained, a Tzanck preparation demonstrates multinucleated giant cells with intranuclear inclusions.
  - Definitive diagnosis of HSV encephalitis requires a biopsy.

- Treatment
  - Oral or genital herpes can be treated with acyclovir, valacyclovir, or famciclovir, along with analgesics.
  - HSV encephalitis should be treated promptly with IV acyclovir.

Varicella Zoster

- General Information
  - Virus: human herpes virus 3
  - Causes both chickenpox and herpes zoster

- Clinical Presentation
  - Chickenpox usually occurs in children <9 years of age.
  - Characterized by the sudden onset of fever and malaise followed by a characteristic rash: initially maculopapular then vesicular; lesions can be located anywhere and appear in crops in a variety of stages (see Image #76)
Zoster is a reactivation infection of virus that is dormant in a dorsal root ganglion; characteristic rash is multiple vesicles on an erythematous base; usually unilateral and appears along a sensory dermatome; may be preceded by hypesthesia or tingling (see Image #26).

Immunocompromised patients can have severe disease involving multiple dermatomes.

Zoster involving the ophthalmic branch of the trigeminal nerve is an ocular emergency.

- Look for Hutchinson's sign: lesion on the tip of the nose

Diagnosis
- Diagnosis is based primarily on clinical findings.
- Tzanck preparation will demonstrate multinucleated giant cells.
- ELISA can be performed on skin scrapings.

Treatment
- Chickenpox
  - Supportive
  - Avoid aspirin: linked to development of Reye syndrome
  - Acyclovir can be given but is not very effective.

- Zoster
  - Pain control
  - Acyclovir, famciclovir, and valacyclovir can be given.
  - Patients with disseminated disease or ocular involvement should be given IV acyclovir.

Infectious Mononucleosis
- General Information
  - Virus: Human herpes virus 4 (Epstein-Barr virus)
  - Infects and transforms B lymphocytes
  - Implicated in the pathogenesis of Burkitt’s lymphoma and nasopharyngeal carcinoma
  - Most common among high school and college students
  - Transmitted via the oropharyngeal route, most often by kissing

- Clinical Presentation
  - The clinical presentation is characterized by fever, exudative pharyngitis, fatigue, and malaise.
  - Posterior cervical adenopathy is a classic association.
  - Gastrointestinal manifestations include abdominal pain, hepatitis, and splenomegaly.
  - Some patients present with CNS involvement manifested as meningitis, encephalitis, transverse myelitis, and Guillain-Barré syndrome.

- Diagnosis
  - CBC reveals lymphocytosis with atypical lymphocytes.
  - Look for elevation of heterophile antibodies (Monospot test).

- Treatment
  - Supportive
  - Avoidance of contact sports, given risk of splenic rupture

Cytomegalovirus (CMV)
- General Information
  - Virus: human herpes virus 5
  - Infection is clinically similar to infectious mononucleosis (heterophile negative).
Severe infections occur in neonates and in immunocompromised patients (those with HIV/AIDS and transplant recipients).

**Clinical Presentation**
- Immunocompetent patients present with fever, lymphadenopathy, and pharyngitis.
- Neonates can present with lethargy, petechial rash, hepatosplenomegaly, seizures, and jaundice.
- Infection can be severe in immunocompromised patients; the presentation includes colitis, esophagitis, pneumonitis, retinitis, and polyradiculopathy (ascending weakness with loss of deep tendon reflexes).

**Diagnosis**
- CBC demonstrates lymphocytosis with atypical lymphocytes.
- Definitive diagnosis depends on virus isolation or rise in antibody titers.

**Treatment**
- Immunocompetent patients are treated supportively, as the illness is self-limited, lasting 2 to 4 weeks.
- Neonates and immunocompromised patients should receive ganciclovir, cidofovir, or foscarnet; IVIG may be required in certain conditions (pneumonitis).

**Human Immunodeficiency Virus (HIV)**

**General Information**
- HIV is a retrovirus that belongs to the lentivirus subfamily.
- There are two major subtypes: HIV-1 (the predominant subtype) and HIV-2 (rare in the United States).
- HIV selectively attacks T4 helper cells, macrophages, and monocytes.
- Over the past several years, the greatest percentage increase in HIV/AIDS cases has occurred among heterosexual women, children, and minority populations.
- Primary risk factors for HIV include male homosexuality, bisexuality, IV drug use, heterosexual exposure to a partner with risk factors, receipt of a blood transfusion prior to 1985; transmission from mother to fetus is also common.

**Clinical Presentation**
- The clinical presentation of HIV depends on the degree of immunosuppression and the organ involved.
- Acute retroviral syndrome
  - Occurs during the first few weeks following infection
  - Symptoms include fever, fatigue, adenopathy, pharyngitis, diarrhea, and diffuse lymphadenopathy.
  - Symptoms typically subside over 1 to 3 weeks.
  - Most patients do not seek medical attention.
- Pulmonary involvement
  - Bacterial infections with *S. pneumoniae, H. influenzae*, and *Pseudomonas* are the most common pulmonary infections among AIDS patients.
  - *Pneumocystis jiroveci* (formerly *P. carinii*)
    - One of the most common opportunistic infections
    - Patients typically present with a non-productive cough, low-grade fever, and dyspnea that worsens with exertion.
    - Chest film can be normal or demonstrate bilateral “ground-glass” opacities. (see Image #55)
    - Elevated LDH is common.
    - Diagnosis can be made using indirect immunofluorescent staining of sputum or bronchoalveolar lavage samples.
    - Treat with trimethoprim-sulfamethoxazole for 21 days.
Acceptable alternatives include pentamidine, dapsone, clindamycin + primaquine, or atovaquone. Prednisone is recommended for patients with a PaO₂ < 70 mm Hg or an A-a gradient > 35.

Neurologic involvement

- Cryptococcus neoformans
  - A fungal infection of the CNS that is most common in patients with CD4 counts < 100 cells/mm³
  - The most common initial symptoms are headache and low-grade fever; less frequent symptoms are visual changes, seizures, nausea, vomiting, and cranial nerve deficits.
  - CT scan of the head is usually normal.
  - Definitive diagnosis is made with lumbar puncture, which demonstrates elevated opening pressure with a mononuclear pleocytosis; CSF or serum cryptococcal antigen can be used for presumptive diagnosis.
  - Depending on the clinical presentation, patients may be treated with oral fluconazole or IV amphotericin.

- Toxoplasma gondii
  - The most common cause of focal intracranial mass lesions in patients with AIDS
  - Typically occurs in patients with CD4 counts < 100 cells/mm³
  - Symptoms may include headache, fever, altered mental status, seizures, and focal neurologic deficits.
  - Diagnosis is most often made by the demonstration of multiple subcortical ring-enhancing lesions (the differential diagnosis includes CNS lymphoma, cerebral tuberculosis, fungal infection, CMV infection, Kaposi's sarcoma, and microhemorrhage). (see Image #68)
  - Serologic testing is not helpful in the ED.
  - Suspected toxoplasmosis is treated with pyrimethamine and sulfadiazine with folinic acid.
  - Acceptable alternatives include clindamycin, azithromycin, atovaquone, and doxycycline.
  - Steroids are indicated for significant mass effect.

Gastrointestinal involvement

- Oropharyngeal candidiasis
  - Affects more than 80% of AIDS patients
  - Candida albicans is the most common causative fungal organism.
  - Symptoms include dysphagia, burning, and soreness of the tongue.
  - Exam reveals characteristic whitish, lacy plaques that are easily scraped off an erythematous base.
  - Microscopic exam can confirm the diagnosis.
  - Patients can be treated with clotrimazole troches or fluconazole.

- Esophagitis
  - Can be caused by Candida, HSV, or CMV
  - Candida is the most common cause.
  - Patients present with odynophagia, dysphagia, and chest pain.
  - Diagnosis can be made with endoscopy, viral cultures, fungal stains, or, occasionally, biopsy.
  - Treatment depends on the etiology.

- Diarrhea
  - The most common GI complaint in AIDS patients
  - Can be caused by a number of bacterial, viral, and fungal infections. In addition, diarrhea caused by medication side effects is common.
Cryptosporidium
- A common cause of diarrhea in AIDS patients
- Diagnosis is made by AFB staining of stool, serology, or ELISA.
- Treatment is with paromomycin or azithromycin for 4 weeks.

Isospora
- Opportunistic organism that produces watery diarrhea
- Diagnosis made by stool samples or ELISA
- Treatment is with TMP-SMX for up to 21 days.

Ocular involvement
- CMV retinitis
  - The most common cause of blindness in AIDS patients
  - Infection can be asymptomatic or can present as blurred vision, changes in visual acuity, floaters, scotoma, or eye pain.
  - Characteristic fundoscopic finding is perivascular fluffy white retinal lesions.
  - Patients should be treated with ganciclovir along with highly active antiretroviral therapy (HAART).

Adverse drug reactions
- A number of drug reactions are important to recognize in AIDS patients:
  - Didanosine – pancreatitis
  - Indinavir – nephrolithiasis
  - INH – hepatitis
  - TMP-SMX – hepatotoxicity, rash, hypokalemia
  - Ritonavir – parasthesias
  - Dapsone – hepatitis
  - Pentamidine – hypoglycemia or hyperglycemia

Rabies
- General Information
  - Organism: a neurotropic rhabdovirus of the Lyssavirus genus
  - Dogs are the most commonly infected animal worldwide.
  - In the United States, the principal animal reservoirs are raccoons, skunks, foxes, and bats.
  - Small rodents and lagomorphs are unlikely to carry the virus.
  - Infection is acquired when the virus is inoculated into bite wounds, open cuts or sores, or mucous membranes.
  - The virus replicates in muscle cells near the bite and then ascends peripheral nerves to the CNS (12 to 100 mm/day).

- Clinical Presentation
  - Infection begins with a nonspecific prodrome of fever, headache, rhinorrhea, sore throat, myalgias, and nausea.
  - Paresthesias at the bite site may be the first neurologic presentation.
  - Neurologic manifestations occur in two forms: the “furious” stage and the “dumb” stage
    - The furious stage is characterized by agitation, hydrophobia, irritability, and hyperexcitability; patients are usually tachycardic, tachypneic, and febrile; hallucinations, seizures, ataxia, and aerophobia may be seen.
    - The dumb stage presents predominantly with extremity weakness; consciousness usually remains intact.
  - Regardless of the stage, coma and death occur after about 1 week.
Diagnosis
- Brain biopsy is the definitive method for diagnosis.
- Isolation of viral RNA, viral antigen, or antibody from peripheral nerve samples can also be used.

Treatment
- No specific or effective rabies treatment exists
- Once manifested, patients usually die within 3 to 10 days.
- The most important treatment is postexposure prophylaxis.
- Postexposure prophylaxis must take into account the exposure, the biting animal, and the incident.
  - Any bite from a raccoon, skunk, fox, bat, or coyote is considered high risk.
  - If a bat is found in the room of a sleeping individual, assume a bite has occurred.
  - Wound care is essential to treatment; scrub wound edges with soap and water.
  - Human rabies immunoglobulin (HRIG), 20 IU/kg, should be given, infiltrating as much as possible into and around the bite site.
  - Human diploid cell vaccine (HDCV), 1 cc IM, is given on days 0, 3, 7, 14, and 28.

Adenovirus
- General Information
  - Adenoviruses 8, 19, and 37 cause epidemic keratoconjunctivitis.
- Clinical Presentation
  - The clinical presentation consists of fever and upper respiratory symptoms (rhinorrhea, sore throat, cough, conjunctivitis).
- Diagnosis
  - Based on clinical findings
- Treatment
  - Supportive care with antipyretics, fluids, decongestants

Rotavirus
- General Information
  - Spread via the fecal-oral route
  - Most common in the winter months
  - Infects epithelial cells of small intestine, resulting in secretory diarrhea
  - Can be fatal in children as a result of severe dehydration
- Clinical Presentation
  - Classic symptoms include fever, headache, myalgias, nausea, vomiting, and watery diarrhea
- Diagnosis
  - Direct visualization of the virus in a stool sample
  - Immunoassays for rotavirus antigen can be performed.
- Treatment
  - Supportive therapy, with emphasis on maintaining hydration

Arboviruses
- General Information
  - Several types exist
    - Alphaviruses: Eastern equine encephalitis (EEE), Western equine encephalitis (WEE)
    - Flaviviruses: St. Louis encephalitis, West-Nile encephalitis, Dengue fever
    - Bunyaviruses: California encephalitis
Transmitted via arthropod vector: most often a mosquito

Clinical Presentation
- Patients present with fever and signs of encephalitis (headache, altered mental status, seizures, or coma).

Diagnosis
- Confirmed by isolation of the virus or rise in antibody titers

Treatment
- Entirely supportive

**Rubella (German Measles)**

General Information
- Infection is acquired via respiratory secretions.
- Highly communicable from 1 week before to approximately 4 days after rash onset
- Congenital rubella syndrome: hearing loss, cataracts, retinopathy, mental retardation, cardiac abnormalities, premature delivery, fetal death

Clinical Presentation
- Characterized by fever, malaise, headache, lymphadenopathy (postauricular, cervical, occipital) and rash; rash is maculopapular and appears similar to rubeola and roseola

Diagnosis
- Diagnosis is based on clinical findings.

Treatment
- Supportive care, with avoidance of women of childbearing age

**Mumps**

General Information
- Occurs most commonly in the winter and spring months
- Typically communicable 1 week before to 10 days after the onset of parotitis

Clinical Presentation
- Fever
- Parotid swelling
  - Can be unilateral or bilateral
  - Nonsuppurative
  - Occasionally associated with trismus
- Epididymoorchitis
  - Can be unilateral or bilateral
- Meningitis (rare)
  - CSF with low glucose concentration and lymphocytic pleocytosis

Complications
- Transverse myelitis
- Guillain-Barré syndrome
- Pancreatitis
- Myocarditis
- Deafness

Diagnosis
- Clinical: parotid swelling + constitutional symptoms in the setting of an exposure
• Treatment
  o Supportive care with analgesics and antipyretics
  o Close contacts who are unimmunized should be vaccinated

Rubeola (Measles)
• General Information
  o Infection acquired via respiratory secretions
  o Highly communicable but not teratogenic
• Clinical Presentation
  o Classic triad: cough, coryza, and conjunctivitis (precede rash by 2 to 4 days)
  o Rash
    ▪ Maculopapular rash that begins on the head
    ▪ Progresses downward over the body over a period of 3 days
  o Koplik's spots are grayish spots on an erythematous base located on lateral buccal mucosa.
• Complications
  o Pneumonitis
  o Encephalitis (subacute sclerosing panencephalitis)
• Diagnosis
  o Diagnosis is based on clinical findings.
• Treatment
  o Supportive care
  o Infants <1 year of age or immunocompromised children should receive immune globulin within 6 days after exposure.
9.1 CRANIAL NERVE DISORDERS

Bell's Palsy

- General
  - Defined as an idiopathic peripheral facial nerve paralysis
  - Incidence: about 25 per 100,000; male=female; young adults (age 20–35) may have a higher incidence

- Symptoms
  - Typical: unilateral (rarely bilateral) upper and lower facial paralysis
  - Pain behind the ear
  - Hyperacusis (due to paralysis of the stapedius muscle)
  - Decrease or change in taste of the anterior two thirds of the tongue on the affected side
  - Change in tearing or salivation
  - Crocodile tears: lacrimation on chewing or eating
  - Should not include cranial nerve V symptoms (i.e., no sensory defects) but may have jaw or ear pain


- Localization of lesion is critical.
  - Central lesions (not consistent with Bell’s palsy) will spare forehead due to cross-innervations, but the patient may have other cranial nerve deficits.
  - Peripheral lesions (such as Bell’s): hemifacial motor weakness

- Treatment
  - Pharmacologic treatment if within 7 days of onset of symptoms
    - Prednisone, 1 mg/kg, for 7 to 10 days
    - Acyclovir, 400 to 800 mg, 5 times a day for 10 days
    - Patching eye and eye drops are indicated for patients unable to close eye; prevents corneal ulceration
  - Refer to ENT

- Prognosis
  - 98% recover at least partial function
  - About 85% recover completely
  - Recovery usually begins in 1 to 4 weeks and may take more than 3 months to be completed.
Neurologic Disorders

- Prognosis is poorer in patients with more proximal lesions, complete facial paralysis, or hypertension and in older patients (>55 years).

Trigeminal Neuralgia

- General
  - Brief repeated unilateral stabs of pain in one or more of the trigeminal nerve distributions
  - Incidence is 5 per 100,000, with most idiopathic cases occurring after 50 years of age. Occurs from compression of nerve root by vein, artery, or other mass, which results in local demyelination and symptoms

- Symptoms
  - Electric shock or stabbing pain lasting several seconds
  - Usually in V2 or V3 distribution
  - Facial spasm may also occur.
  - Diagnosis is usually clinical.

- Treatment
  - Carbamazepine, 100 mg BID, with eventual increase to TID is preferred.
  - Baclofen, gabapentin, phenytoin, lamotrigine, and valproate are alternatives.
  - Refractory cases may require surgery.

9.2 Demyelinating Disorders

Multiple Sclerosis

- General
  - Most common serious neurologic disease in young adults (between 30 and 50 years of age)
  - 2.5:1 female:male ratio, 2:1 caucasian:non-caucasian ratio
  - Dysfunction of myelination slows nerve conduction
  - Plaques are characteristic lesions and represent focal myelin destruction
  - Size and number are highly variable

- Presentation
  - Most typical: recurrent focal neurologic deficits
  - Half of patients present with a single, isolated neurologic deficit.
  - Optic neuritis is most common initial symptom — perceptual changes (including color changes) and vision loss.
  - Extraocular muscle deficits also common — diplopia
  - Fatigue is seen in the majority of patients; it may be severe.
  - Deficits usually increase with exercise and high ambient temperatures.
  - Highly variable course: most have "relapsing and remitting" form with full recovery between exacerbations; 15% have progressive forms with permanent deficits
  - Exacerbations develop over hours to days and persist for weeks to months.
  - Depression is very common.

- Diagnosis
  - A "great imitator," as symptoms may be vague or mimic numerous other neurologic abnormalities
  - Definitive diagnosis requires at least two episodes of focal neurologic dysfunction and therefore is rarely made in the ED.
  - Multiple white matter lesions on brain MRI
  - CSF shows increased protein (IgG) and modest WBC pleocytosis
  - Differential diagnosis: Lyme disease, stroke, space-occupying lesions (tumor/clot), HIV, neurosyphilis, lupus
Treatment
- Acute exacerbations are treated with methylprednisolone, 250-500 mg IV, every 12 hours for 3 to 7 days
- Interferon and immunosuppressives may be employed for chronic management.

9.3 HEADACHE

Tension-Type Headache
- Synonyms include tension, muscle contraction, stress, ordinary, and psychogenic headaches
- Most common type of headache
- Occurs in all age groups
- Usually presents with a tight, band-like discomfort that is nonpulsating and dull; usually mild and short in duration
- Often worse later in the day
- Not associated with photophobia, phonophobia, nausea, or vomiting
- Treatment: analgesics (acetaminophen, NSAIDS); narcotics are rarely indicated

Hypertensive Headache
- Hypertension is usually not the cause of a headache unless the diastolic blood pressure is >130 mm Hg.
- Diffuse, worse in the morning, and decreases over the course of the day
- Blurred vision, confusion, drowsiness, or vomiting may occur and should prompt consideration of more severe conditions, such as hypertensive encephalopathy.
- May be considered a form of hypertensive encephalopathy, though most would not start IV antihypertensive drips if headache is the sole manifestation
- Treatment: prudent blood pressure control

Mass Lesions
- Brain tumors
  - Primary tumor usually if less than 50 years old; metastasis is usual cause if over 50
  - Usually pressure-like pain
  - May be present for prolonged periods of time; often worse in the morning
  - Classic triad of sleep disturbances, severe pain, and nausea/vomiting seen in only one third of cases
  - Diagnosed with CT scan or MRI; contrast may aid in differentiation of cause with CT
  - Treatment with neurosurgical consult; steroids are useful if intracranial pressure is increased; anticonvulsants if seizures
- Chronic subdural hemorrhages
  - May produce a diffuse or focal headache of constant nature
  - Far more frequent in the elderly and alcoholics; caused by atrophy and the associated stretching of bridging veins
  - Associated with confusion, memory loss, weakness, seizures

Temporal Arteritis
- Synonyms: giant cell arteritis, granulomatous arteritis
- Caused by inflammation of branches of the external carotid artery
- Usually seen in the elderly
- Strongly associated with polymyalgia rheumatica (seen in half of cases)
- Risk of sudden, permanent vision loss mandates aggressive management
• **Presentation**
  - Sudden onset of severe unilateral or bilateral headaches
  - Jaw claudication
  - Temporal artery tenderness is found in less than half of cases
  - Erythrocyte sedimentation rate is usually >50 mm/hr and often >100 mm/hr
• **Treatment:** must be initiated empirically if diagnosis is strongly suspected
  - Prednisone, 60 to 120 mg/day
  - Prompt temporal artery biopsy
  - Usually managed as outpatient, but reliable follow-up is imperative

**Vascular Headache Syndromes**
• **Migraine headache**
  - Typical onset in puberty
  - More common in women; associated with menstruation
  - Majority of patients have family history of migraines
  - Auras: nonspecific or focal neurologic event preceding headache onset
    - Only 15% of patients have associated aura ("classic migraines") while the remainder have no aura ("common migraines")
    - Usually a visual disturbance; less commonly, transient aphasia or focal deficit
    - May be caused by vasoconstriction of cerebral arteries, but the exact mechanism of action has not been determined
  - Headache is usually unilateral, may progress to bilateral, lasts 4 to 72 hours
  - Diagnosis: requires one of the following to be present:
    - Nausea and/or vomiting
    - Photophobia and phonophobia
  - Treatment
    - NSAIDS or oral narcotics for mild episodes
    - Prochlorperazine, 10 mg IV, is often used for more severe episodes with or without diphenhydramine
    - Sumatriptan, 6 mg SQ, is useful if given early in the episode; effective within 2 hours in 80% of patients; contraindicated for patients with coronary artery disease or poorly controlled hypertension
    - Dihydroergotamine, ketorolac, and corticosteroids are other potential treatments.
    - IV/IM narcotics offer nonspecific analgesia and sedation; often effective but to be used only in patients who do not respond to initial migraine-specific therapies
  - **Cluster Headache**
    - Much more common in men; onset in 20s
    - Excruciating unilateral boring pain behind or around the eye
    - Associated with conjunctival injection, tearing, nasal congestion or rhinorrhea, facial flushing; no aura
    - Lasts minutes to hours
    - Occurs daily or several times a day for several days in a row, episodes then typically do not recur for 6 to 12 months
  - **Treatment**
    - Most migraine therapies are effective, especially sumatriptan and dihydroergotamine.
    - Oxygen, 7 to 10 L/min, is usually rapidly effective.
    - Intranasal cocaine or lidocaine is advocated by some.
Headache Following Lumbar Puncture
- Most common complication of lumbar puncture, occurring in up to 40% of patients
- Described as bilateral throbbing headache worsened by upright positioning
- Nausea, vomiting, blurred vision, and neck stiffness may also be present.
- Occurrence is decreased with the use of smaller diameter needles, entering bevel up when patient is in lateral decubitus position, and taking the minimal amount of fluid.
- Treatment
  - Most resolve with bedrest, hydration, and simple analgesics (aspirin, acetaminophen, oral narcotics).
  - Caffeine, 500 mg IV, is often effective as well.
  - Severe headache lasting more than 24 hours may require an epidural blood patch.

Carotid/Vertebral Artery Dissection
- Often presents with a sudden onset of head or neck pain; may or may not have focal neurologic defects; usually neurologic findings occur in the first few hours
- Pathophysiology
  - Often caused by minor trauma or sudden neck movement (neck torsion, coughing, or minor falls)
  - Causes an intramural hemorrhage in the vessel wall
  - Thrombus forms later and can precipitate a stroke.
- Carotid dissection
  - Often causes a severe throbbing unilateral headache
  - Headache may also be similar to previous headaches.
  - Severe retro-orbital pain without cluster headache may be suggestive.
  - Patients develop ipsilateral partial Horner’s syndrome (miosis and ptosis with preserved facial diaphoresis) and contralateral hemispheric findings, including paralysis, neglect, and aphasia.
- Vertebral artery dissection
  - Presents as severe unilateral posterior headache
  - Neurologic findings include vertigo, ataxia, diplopia, hemiparesis, and unilateral facial weakness
- Diagnosis and treatment
  - Head CT is often obtained first but is normal in many patients.
  - MRI or angiography is often required for definitive diagnosis.
  - Treatment is stroke prevention with anticoagulation and antiplatelet therapy.

9.4 INFECTION

Encephalitis
- Viral infection of brain parenchyma
- Often with neurologic AND meningeal symptoms
- Causes: herpes simplex virus, Epstein-Barr virus, herpes zoster virus, rabies
- Clinical findings
  - New psychiatric symptoms: behavior and personality changes are most common
  - Fever
  - Cognitive defects
  - Seizures
  - Movement disorders
  - Headache and photophobia
  - Lethargy and altered level of consciousness
Abscesses

Brain
- Focal pyogenic infection of the parenchyma
- Uncommon event (3/100,000 admissions for headache)

Clinical presentation:
- Typically well appearing
- 50% with fever
- 33% with focal neurologic deficit
- Most with headache
- Best diagnosed using CT with contrast

Treatment
- Antibiotics based on suspected organism
- Cefotaxime or ceftriaxone with metronidazole for sinus or otogenic infections
- Traumatic or post-surgical abscesses need coverage for methicillin-resistant *Staphylococcus aureus*
- Cover for tuberculosis, parasitic, or fungal cause if patient is at risk for these infections.
- Neurosurgical consultation

Epidural
- Most commonly seen in intravenous drug abusers
- Hematogenous spread to epidural space

Clinical findings
- Back pain
- Fever
- Percussion tenderness
- Weakness or paralysis of extremity may be present

Diagnosis
- There may be leukocytosis on CBC, but it is often normal.
- The erythrocyte sedimentation rate (ESR) is usually >20 mm/hr in these patients.
- Plain films are usually normal.
- Definitive diagnosis is made with MRI.

Treatment
- Antibiotics are chosen based on suspected organism.
- Neurosurgical drainage

Meningitis
- Primary goal is recognition and early treatment.
- Infection leads to inflammation, edema, and ischemia.

Clinical findings
- Fever, headache, stiff neck, photophobia
- 25% of patients with meningitis have seizures
- Meningeal irritation findings (at least one is seen in 50% of cases)
  - Brudzinski’s sign (flexion of hips and knees in response to passive neck flexion)
  - Kernig’s sign (contraction of hamstrings in response to knee extension while hip is flexed)
• Types of meningitis
  
  o Bacterial
    - High mortality and neurologic sequelae without antibiotic treatment
    - These patients often appear toxic.
    -Characteristic lumbar puncture findings include
      - WBC count >1,000/μL
      - >80% polymorphonuclear cells
      - Glucose <40 mg/dl
      - Protein >200 mg/dl
      - Gram stain is positive in 80%.
    - Common pathogens and antibiotic therapy are listed in Table 9-1.

  
  Table 9-1. Common Bacterial Causes of Meningitis and Appropriate Empiric Antibiotic Therapy

<table>
<thead>
<tr>
<th>Patient Population</th>
<th>Common Pathogens</th>
<th>Initial Empiric IV Antibiotic Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>Group B strep, <em>Listeria</em>, gram-negative bacilli</td>
<td>Ampicillin, 50 mg/kg, with cefotaxime, 50 mg/kg, or gentamicin, 2.5 mg/kg</td>
</tr>
<tr>
<td>Infants (1–3 months)</td>
<td>Group B strep, <em>Listeria</em>, <em>H. influenza</em>, <em>S. pneumoniae</em>, <em>N. meningitidis</em></td>
<td>Cefotaxime, 50 mg/kg, or ceftriaxone, 100 mg/kg; add ampicillin, 50 mg/kg, up to 8 weeks of age; add vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>3 months–18 years</td>
<td><em>H. influenza</em> (decreased due to vaccine), <em>S. pneumoniae</em>, <em>N. meningitidis</em></td>
<td>Ceftriaxone, 100 mg/kg IV (max 2 g), plus vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>18–50 years</td>
<td><em>S. pneumoniae</em>, <em>N. meningitidis</em></td>
<td>Ceftriaxone, 2 g IV, plus vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>&gt;50 years, alcoholic, or immunosuppressed</td>
<td><em>S. pneumoniae</em>, <em>N. meningitidis</em>, <em>Listeria</em>, aerobic gram-negative bacilli</td>
<td>Ceftriaxone, 2 g IV, plus ampicillin, 2 g IV, plus vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
</tbody>
</table>

  - Corticosteroids are recommended before or concurrently with antibiotics; thought to decrease cerebral edema and lessen neurologic sequelae
  - Immunocompromised patients are at increased risk for multiple pathogens (*S. pneumoniae*, *N. meningitidis*, *Listeria*, aerobic gram-negative bacilli) and should be treated with vancomycin, 25 mg/kg, ampicillin, 2 g IV q4h, and ceftazidime, 2 g IV q8h.

  o Viral
    - Often has a benign, self-limited course
    - Common pathogens are arbovirus, enteroviruses, and herpes simplex virus.
    - Patients with herpes simplex appear toxic and may be treated with acyclovir.
    - In all other cases, treatment is supportive.
    - Lumbar puncture findings include the following:
      - WBC count <1,000/μL
      - Polymorphonuclear cells, 1% to 50%
      - Glucose >40 mg/dl
      - Protein <200 mg/dl
      - Gram stain is negative.
    - In early cases, patients with viral meningitis may be indistinguishable from those with bacterial infections and therefore often receive antibiotics.
9.5 NEUROMUSCULAR DISORDERS

Guillain-Barré Syndrome

- Acute inflammatory demyelinating polyneuropathy
- May follow upper respiratory or gastrointestinal infection
- Early signs
  - Paresthesias in extremities
  - Progressive, ascending, symmetric muscle weakness
  - Hyporeflexia with or without sensory or autonomic symptoms
- Respiratory muscles are involved later, as are cranial nerves.
- Onset is rapid—hours to a few days
- Diagnosis is made on clinical grounds and CSF analysis for elevated protein.
- Treatment involves intravenous immune serum globulin, plasma exchange, and supportive care with special attention to respiratory support.
- Miller-Fischer variant presents with ataxia, ophthalmoplegia, and areflexia with relatively little weakness; patients with this condition are at decreased risk for respiratory compromise.

Myasthenia Gravis

- General
  - Rare autoimmune disease
  - Antibodies to acetylcholine (ACh) receptors of neuromuscular junction
  - Onset in young adulthood with female predominance
  - Very strong association with thymomas
- Presentation
  - Muscle weakness that improves with rest
  - Usually affects extraocular muscles initially, with ptosis and diplopia
  - When generalized, affects proximal more than distal muscles
  - Symptoms precipitated by viral illness, immunizations, menses, surgery
  - May present with respiratory insufficiency (myasthenic crisis)
- Diagnosis
  - Classic association of ocular, cranial nerve, and proximal muscle weakness that worsens with exercise (sufficient for provisional diagnosis)
  - Myasthenic crisis: edrophonium test is both diagnostic and therapeutic
    - Edrophonium inhibits acetylcholinesterase, which normally degrades ACh at the neuromuscular junction.
    - Edrophonium test dose (1–2 mg IV) will relieve myasthenic weakness in seconds and lasts <10 minutes.
    - If test is positive (symptoms abate), a higher dose of edrophonium can be given for longer effect; neostigmine or pyridostigmine can be given for long-term therapy.
    - Prepare for intubation prior to giving edrophonium, as a negative test may exacerbate respiratory insufficiency.
    - Atropine at bedside for test because edrophonium can cause bradycardia, hypotension, and bronchospasm
- Cholinergic crisis: may mimic myasthenic crisis
  - Seen in overdose or overtreatment with anticholinergic medication for myasthenia
  - Due to acetylcholine excess, causing weakness and respiratory failure; unlike myasthenic crisis, SLUDGE symptoms are also seen (salivation, lacrimation, urination, defecation, GI symptoms such as diarrhea, emesis)
  - Edrophonium test will be negative (no improvement in weakness) and is likely to exacerbate symptoms.
• Differential diagnosis
  o Botulism: weakness starting with bulbar and ocular muscles. Unlike myasthenia gravis, deep tendon reflexes are absent.
  o Familial periodic paralysis associated with hyperkalemia or, more commonly, hypokalemia
  o Tick paralysis: ascending weakness and paralysis produced by neurotoxin from feeding tick; resolves promptly with tick removal
  o Guillain-Barré syndrome: as described above

Parkinson's Disease
• General
  o Neurodegenerative disorder caused by the loss of dopaminergic neurons in the substantia nigra
  o Typical onset after 60 years of age
  o Cause is generally idiopathic
• Physical findings
  o Tremor usually begins in one upper extremity and may be intermittent.
  o Initial symptoms are nonspecific and include fatigue and sleep disturbance.
  o As it progresses, the most common findings are resting tremor, rigidity, and bradykinesia.
  o The tremor has a frequency of 3 to 5 Hz and usually manifests as a pin-rolling motion of the thumb and forefinger.
  o Rigidity may be smooth (lead pipe) or oscillating (cogwheeling).
• Management
  o No laboratory abnormalities are specific to Parkinson's disease.
  o CT and MRI appear normal in these patients.
  o Levodopa combined with a peripheral decarboxylase inhibitor is first-line treatment.
  o Dopamine agonists may also be used, but they cause sleepiness and hallucinations.

Peripheral Neuropathy
• Toxic and other neuropathies
  o Tetanus (see Chapter 8, Systemic Infectious Disorders)
  o Botulism (see Chapter 8, Systemic Infectious Disorders)
  o Tick paralysis
    ▪ Symptoms are almost identical to those of Guillain-Barré syndrome.
    ▪ Caused by toxin from an engorged tick
    ▪ Treatment is removal of the tick, which resolves symptoms.

Poisoning
• Metallic compounds
  o Combined central and peripheral nervous system defects
  o Common causes are arsenic (insecticides, rat poison), iron (industry), lead, and mercury.
• Organic compounds
  o Wide variety cause neuropathies
  o Ethanol – slow progressive neuropathies
Metabolic
- Diabetes – acute hyperglycemia can cause acute neuropathic changes such as cranial nerve VI deficits
- Hepatic failure, renal failure, myxedema, and porphyria have been associated with acute neuropathies

9.6 PSEUDOTUMOR CEREBRI OR IDIOPATHIC INTRACRANIAL HYPERTENSION
- Typical patient: young obese female with irregular menses or amenorrhea
- Nonspecific headache with visual complaints
- Papilledema on fundoscopic exam (see Image #29)
- CT shows slit-like ventricles with no mass effect
- Lumbar puncture opening pressure >20 cm H₂O
- Treatment options include acetazolamide with or without furosemide, repeated lumbar punctures, or, in severe cases, a ventriculoperitoneal shunt or optic nerve fenestration.

9.7 HYDROCEPHALUS
General
- Disturbance in the formation, flow, or resorption of cerebrospinal fluid (CSF)
- If untreated, leads to increased intracranial pressure, neurologic sequelae, and possibly death if herniation or compression on respiratory centers occurs

Types
- Normal pressure – enlarged ventricles, normal CSF opening pressure
- Obstructive – flow of CSF is obstructed
- Other types are caused by overproduction or defective absorption of CSF

Clinical Features
- Headaches – more prominent in the morning
- Nausea/vomiting
- Blurred vision
- Unsteady gait related to limb or truncal ataxia
- In normal pressure hydrocephalus, there is a recognized triad of gait disturbance, dementia presenting as recent memory impairment, and urinary incontinence.

Management
- CT of the head
- Lumbar puncture to assess opening pressure
- Neurosurgical consultation
- Ventricular-peritoneal shunt is most commonly placed as definitive treatment.

9.8 DEMENTIA
General
- Gradually progressive deterioration of cognitive function
- Many different causes
- Most are irreversible
- Criteria for dementia include
  - Memory impairment (difficulty recalling old or learning new information)
  - One of the following: aphasia, agnosia, apraxia, disturbance in executive functioning
  - Deficits cause an impairment of previous level of functioning
Etiologies

• Primary causes
  o Alzheimer's disease
  o Pick's disease
  o Subcortical dementia of the thalamus
  o Lewy body dementia

• Secondary causes
  o Drugs – sedative hypnotics, antidepressants, lithium, anticholinergics, anticonvulsants
  o Exposures – heavy metals or carbon monoxide
  o Endocrine abnormalities – hypothyroidism, hyperthyroidism, parathyroid disease, Cushing's syndrome
  o Nutritional deficiency – folate, B12, thiamine, niacin
  o Intracranial infection, especially in immunocompromised patients who develop toxoplasmosis, cytomegalovirus, or cryptococcal meningitis
  o Depression
  o Normal pressure hydrocephalus
  o Hypertension (vascular dementia)

Clinical Features

• Insidious cognitive dysfunction
• Nonspecific – irritability, insomnia, somatic complaints, and memory loss
• Cognitive deficits become more pronounced as disease progresses.
• Memory impairment, language deficits, and errors in judgment
• Often the patient is brought in by family or friends after recognized worsening mental status
• In depression, the onset of symptoms tends to be more acute; patients emphasize the difficulty, and most have a history of psychiatric illness.

Evaluation

• All patients should have baseline screening for reversible causes of dementia.
• Labs: CBC, electrolytes, liver function, renal function, thyroid function, tests for syphilis and HIV
• CT of the head
• Some patients require drug and heavy metal screens.
• Lumbar puncture may be required if infection is suspected.

Management

• The most common treatable causes of dementia are depression, normal pressure hydrocephalus, intracranial mass, and medications.
• For primary dementia, tacrine and donepezil are first-line agents, but they do not halt the disease process.
• These patients may also require treatment for agitation or sleep disturbance.
  o Haloperidol is useful for treating agitation.
  o Temazepam can be used to treat sleep disturbances.
• Vascular dementia should be treated with blood pressure control.
Neuroleptic Malignant Syndrome

- General
  - Characterized by severe extrapyramidal dysfunction (muscle rigidity), altered mental status, hyperthermia, and autonomic disturbances following use of neuroleptic drugs
  - Very rare: <0.2% of patients exposed to neuroleptics develop this syndrome
  - Mechanism via dopamine receptor blockage in the striatum and hypothalamus
  - Develops over a period of 24 to 72 hours; can last 5 to 10 days even after oral neuroleptics are discontinued
  - Predisposing factors
    - Use of long-acting depot neuroleptic drugs
    - Use of antipsychotics, especially haloperidol; reported in anti-Parkinsonian medication withdrawal and with recreational drug use
    - Onset is not related to the duration of exposure to neuroleptic or to toxic overdoses (idiosyncratic reaction)

- Clinical features
  - Hyperthermia: temperatures to 41°C (106°F) and higher
  - Autonomic system instability: diaphoresis, BP instability, tachycardia, cardiac dysrhythmias
  - Hypertonicity of skeletal muscles: generalized “lead pipe” increase in tone
  - Alteration in level of consciousness
  - 10% mortality from respiratory failure, cardiac arrest, renal failure

- Laboratory abnormalities
  - Most are nonspecific – neuroleptic malignant syndrome is a diagnosis of exclusion
  - Leukocytosis: 15,000 to 30,000 WBCs with or without a left shift
  - Increased SGOT (AST), SGPT (ALT), LDH, and alkaline phosphatase
  - Increased CPK: may exceed 16,000 IU/L
    - Reflects rhabdomyolysis from intense, sustained muscle contractions
    - May result in acute renal failure
  - Lumbar puncture: normal or nonspecific

- Differential diagnosis
  - Heat stroke: does not include rigidity
  - Lethal catatonia: due to sustained manic agitation
  - Malignant hyperthermia: occurs after exposure to certain depolarizing muscle relaxants or gas anesthetics
  - Drug interactions with MAO inhibitors
  - Anticholinergic syndrome: dry flushed skin, dry mouth, dilated pupils, decreased bowel sounds, urinary retention, increased temperature, confusion, and disorientation
  - Serotonin syndrome: hyperthermia, agitation, hyperreflexia more prominent in lower extremities, increased muscle tone from increased serotonin neurotransmission due to serotonin agonist medications (SSRI, MAO inhibitors, tricyclic antidepressants, meperidine)

- Treatment – no controlled studies regarding treatment of neuroleptic malignant syndrome
  - Muscle relaxants
    - Benzodiazepines
    - Often need supranormal doses
  - Reversal of hyperthermia
    - Rapid cooling
    - Decrease heat production by muscles by administering a muscle relaxant
Treatment of rhabdomyolysis
- IV fluids to ensure good urine output
- Consider urinary alkalinization
Bromocriptine
- Dopamine agonist
- Reverses muscle rigidity over days

Dystonic Reaction
- General
  - Characterized by intermittent spasmodic contraction of muscles in the face, neck, trunk, and extremities
  - A recognized reaction to a long list of medications; most common are antipsychotics, antidepressants, and antiemetics
  - Reaction is usually idiosyncratic.
  - Occurs within 48 hours after drug administration in 50% and within the first 5 days in 90% of patients
- Physical findings
  - Oculogyric crisis – deviation of eyes in all directions
  - Trismus
  - Forced jaw opening
  - Grimacing
  - Lordosis
  - Torticollis
  - Mental status is normal.
- Treatment
  - Drugs with anticholinergic properties (benztropine, diphenhydramine)
  - Second-line treatment involves benzodiazepines

9.10 SEIZURE DISORDER

Focal Seizures
- Simple: consciousness unimpaired
  - Simple motor seizures
  - Simple sensory seizures
  - Autonomic seizures
- Complex: consciousness impaired
  - Psychomotor seizures: may mimic psychiatric syndromes or have bizarre symptoms; commonly originate in temporal lobe
  - Partial with secondary generalization (e.g., Jacksonian)
  - May present as a generalized seizure; however, history of focal seizure onset points to underlying structural abnormality (such as CVA or trauma) rather than idiopathic epilepsy

Generalized Seizures
- Grand mal (tonic-clonic)
  - May or may not have aura
  - Tonic phase progresses to clonus (60–90 sec)
  - Relaxation of all muscle groups (including sphincters)
  - Postictal period may last hours
Immediate medication is not usually necessary. Most seizures last 2 to 5 minutes and are benign, self-limiting events.

Phenytoin may be given as a loading dose in the emergency department. Full load is 20 mg/kg IV at no more than 50 mg/min. It is not indicated in alcohol withdrawal seizures.

Fosphenytoin may be given instead at a dose of 15 to 20 PE/kg at 100 to 150 PE/min; it may also be given IM.

Phenobarbital may be used for febrile seizures, but no treatment is required for simple febrile seizures.

**Petit mal (absence)**

- Younger school-aged children (onset between 5 and 12 years of age)
- Onset is rare before 3 years or after puberty.
- Typical attack has sudden onset without aura and lasts 3 to 20 seconds.
- May have 20 to hundreds of seizures per day
- Child is unaware, maintains postural tone, abruptly halts preceding activity, blinks eyes, often has slight rhythmic movements of hands, lips
- Seizure suddenly ends and previous activity continues.
- No postictal state
- Characteristic EEG abnormality (three per second spike-and-wave pattern)
- Complex absence – more complicated movements and occasionally incontinence
- Most children with absence seizure are otherwise normal; 50% go on to develop tonic clonic seizure.

**Minor motor**

- All have in common the characteristic feature of myoclonus (i.e., sudden, involuntary jerks of a single muscle group)
- When onset is in infancy, they are called “infantile spasms.”
- Akinetic seizure – sudden loss of tone of a muscle group
- Very frequently associated with mental retardation
- May go on to develop generalized tonic-clonic seizure

**Eclampsia**

**General**

- A complication of pregnancy occurring after the 20th week of gestation
- Occurs most commonly in antepartum period, although it can occur postpartum
- Progresses from preeclampsia
- Clinical picture
  - Preeclampsia is hypertension and proteinuria during pregnancy with or without edema.
  - When seizures occur, the process has progressed to eclampsia.
- Clinical findings (all may not be present)
  - Sustained systolic blood pressure >160 mm Hg or diastolic >110 mm Hg
  - Tachycardia
  - Hyperreflexia
  - Clonus
  - Visual disturbances
  - Generalized edema
• Management
  o Always consider diagnosis in a pregnant patient with seizure
  o Treat seizure with magnesium sulfate at a loading dose 6 g IV followed by 2 g IV/hr
  o Monitor for signs of hypermagnesemia after seizure termination: loss of reflexes followed by respiratory depression
  o After seizure termination, if diastolic blood pressure is still >105 mm Hg, treat with antihypertensive (hydralazine is most widely used)
  o Complete blood count, liver function tests, and BUN/creatinine to assess organ damage

Status Epilepticus
• General
  o Prolonged seizure lasting more than 30 minutes or repeated seizures within that time (does not return to normal alert state between seizures).
  o Incidence: 60,000 to 100,000 Americans, involves 1.3% to 16% of those with epilepsy
  o Mortality: 6% to 30%; usually due to hypoventilation, arrhythmias, or acidosis
  o Etiology
    ▪ Adults: 72% known, 28% unknown
    ▪ Children: 50% unknown
    ▪ Patients often have no history of seizure
  o Precipitating factors
    ▪ Withdrawal from anticonvulsants is the primary cause.
    ▪ Alcohol withdrawal
    ▪ Metabolic imbalance (hypoglycemia, Ca++, Mg++, patients with renal failure)
    ▪ Acute CNS insult: trauma, CVA, infection
    ▪ Drug intoxication
  o May divide into two groups:
    ▪ Patients with previous seizure disorder – 50% to 80% have subtherapeutic anticonvulsant levels, up to 20% are in alcohol withdrawal
    ▪ No previous seizure disorder – common causes include stroke, alcohol withdrawal, tumor, cardiac arrest, eclampsia, and infection
  o Subclinical (nonconvulsive) status: electrical seizure activity occurs in brain without associated motor activity
    ▪ May result from medical therapy for seizure (patient is paralyzed but seizure continues)
    ▪ May masquerade as prolonged postictal state
    ▪ Complicates up to 25% of cases of status
    ▪ If suspected, EEG is needed for diagnosis
• Goals of management
  o Maintain cardiorespiratory status/oxygenation (ABCs)
  o Termination of electrical/clinical seizure activity in less than 30 to 60 minutes because neuronal damage occurs with prolonged seizures
  o Correct precipitant cause
  o Prevent recurrent seizure
• Initial treatment
  o Assess respiratory status – may need ET intubation, maintain cardiovascular system
  o IV access for glucose/anticonvulsants
• Check levels of any antiseizure medications
• Prevent self injury (loosen tight clothing)
• Brief history: previous seizure/current medications
• Labs: electrolytes, BUN, glucose, Ca++, toxicology screen, ABG, therapeutic drug levels
• Drug therapy
  o First line: 80% to 90% of patients will respond to benzodiazepines, but they are usually combined with an antiepileptic medication (phenytoin or fosphenytoin)
    ▪ Diazepam, 0.15 to 0.3 mg/kg (10 mg max)
    ▪ Lorazepam, 0.05 to 0.15 mg/kg: slower onset but half-life = 15 hours; therefore, decreased risk of repeat seizures
    ▪ Phenytoin, 20 mg/kg (rate no more than 50 mg/min, patient must be on cardiac monitor)
      □ If patient has a history of recent withdrawal from phenytoin, administration of this alone without benzodiazepines may be enough to stop seizure.
      □ History of head trauma – usually give phenytoin alone to decrease the sedative effects of benzodiazepines
    ▪ Fosphenytoin is the water-soluble prodrug of phenytoin
      □ Can achieve free phenytoin therapeutic level faster than phenytoin
      □ Safer, can be given more rapidly, and can be given intramuscularly
  o Second line
    ▪ Phenobarbital: up to 20 mg/kg at 50 mg/min (need not give full load if seizure stops)
      □ 15 to 20 minutes to take effect
      □ 95% of patients respond after second-line therapy
      □ Generally, patients should be mechanically ventilated.
    ▪ Propofol
      □ Give IV loading dose of 1 to 3 mg/kg followed by infusion of 1 to 15 mg/kg/hr.
      □ May work synergistically with benzodiazepine
      □ Patients must be mechanically ventilated for treatment.
      □ Monitor patient for hypotension.
  o Third line: general anesthesia, barbiturate coma
• Common mistakes in treating status
  o Frequent small doses of benzodiazepines: need large IV doses of 1 to 2 mg per minute to a maximum of 10 mg
  o Using PO or IM treatments instead of the preferred IV route
  o Not beginning maintenance therapy (e.g., phenytoin or phenobarbital) once seizure is aborted
• Preventing recurrent seizures once stopped
  o Check levels after load
  o Begin maintenance
  o Treat the cause of the seizure if it is known, e.g., infection or metabolic abnormality
• Systemic complications
  o Cardiopulmonary compromise – hypoxia, aspiration, arrhythmias, pulmonary edema
  o Disseminated intravascular coagulation (DIC)
  o Hyperkalemia
  o Rhabdomyolysis/myoglobinuria – may lead to acute renal failure
9.11 SPINAL CORD DISORDERS

Compression

- General
  - Result from trauma, intervertebral disk herniation, primary or metastatic tumors
  - Lesions localized by neurologic exam
  - Emergent neurosurgical consultation, CT/MRI needed
  - Incidence of new cervical spinal cord injuries: about 50 per 1 million population per year
  - Typical patient is a young man (as all trauma patients); mechanisms of injury include MVA (41%), fall (13%), recreation (5%), penetrating injury (9%).
  - Prevention is more effective and valuable than any details of post-injury management.

- Spinal cord injuries
  - Incomplete injuries
    - Central cord syndrome
      - Most common incomplete injury
      - Older patients with degenerative joint disease and thickened ligamentum flavum with hyperextension injury
      - Upper extremity weakness>>>lower extremity weakness
      - Most will recover considerably
    - Anterior cord syndrome
      - Mechanism: forced flexion resulting in cord contusion or injury to the anterior spinal artery
      - Dense paralysis and loss of sense of pinprick and temperature
      - Preservation of touch, position, and vibration
      - Early surgery is associated with partial recovery
    - Brown-Séquard syndrome
      - Hemisection of the cord
      - Usually associated with penetrating injury (bony fragments from blunt injury may also occasionally produce this picture)
      - Ipsilateral paralysis and loss of position, vibration, and touch with contralateral loss of pinprick and temperature
  - Complete spinal cord injuries
    - Defined as total loss of motor power and sensation distal to the injury
    - Functional recovery is rare if loss persists longer than 24 hours
    - Look for evidence of sacral sparing, including sphincter tone, perianal sensation, and flexor toe movement, since these indicate a partial lesion only
    - Mimicked by spinal shock, which results from concussive injury to the spinal cord and results in total neurologic dysfunction distal to injury; this lasts less than 24 hours
    - End of spinal shock is marked by the return of bulbocavernosus reflex.
    - Absence of this reflex indicates spinal shock and prevents assessment of patient's overall prognosis.
  - Cauda equina injuries
Classic presentation
- Low back pain
- Radicular pain in legs (sciatica)
- Saddle anesthesia
- Bilateral lower extremity weakness
- Urinary retention is the most sensitive finding, but overflow incontinence may be present
- Fecal incontinence
- Absent ankle jerk reflex
- May present as sciatica initially

Cause
- Commonly caused by lumbar disk central prolapse; most common at L4-L5
- Tumors
- Atrial venous malformation

Pain is constant and worse with movement; may be similar to sciatica

Radiology
- Plain radiographs are usually negative in disc disease
- MRI is the preferred study, but CT myelography may be useful if MRI is not available.

Treatment – urgent neurosurgical consultation and decompression is required

Prognosis
- Best predicted by urinary sphincter involvement
- Sensory abnormalities: worse prognosis
- Motor abnormalities: better prognosis

Peripheral Nerve Injuries
- Associated primarily with penetrating trauma
- Deficit depends on affected nerve
- Close follow-up is suggested; healing takes weeks to months

Cervical Spine Injuries
- Jefferson fracture (C1)
  - Blow-out fracture of the ring of C1 from vertical compression
  - Diagnosed on odontoid view or on CT
  - Extremely unstable
- Odontoid fracture (C2)
  - Fracture of the odontoid process of C2
  - Unstable C1-C2 complex
  - Mechanism is flexion with shearing force
  - Seen on odontoid view
  - Also seen as creased prevertebral space on lateral x-ray film
- Hangman’s fracture (C2)
  - Bilateral fracture of the posterior elements of C2 (pedicles)
  - Cord damage is usually minor
    - Canal is wide at C2
    - Bilateral nature of the fracture usually decompresses the cord
• Mechanism is extreme hyperextension, usually in MVA
  • Seen on the lateral view
  • Unstable

• Lower C-spine injuries
  • Injury depends on the mechanism of applied force
  • Neurologic deficits are common – canal is narrower, especially at C3-C4
  • Most common injury (by far) is at C5, followed by C4 and C6
  • Clayshoveler’s fracture – C6, C7, T1 spinous process avulsion; stable fracture (see Image #61)

• Dislocations
  • Unilateral facet
    • Flexion/rotation injury
    • Lateral x-ray film shows 25% to 33% anterior displacement of one vertebral body on the next lower one
  • Bilateral facet
    • Total ligamentous disruption from high-force flexion injuries
    • Lateral x-ray film shows >50% anterior displacement of one vertebral body on the next lower one

• Ligamentous injury
  • Neck pain without concomitant fracture
  • Loss of normal lordosis
  • Prevertebral soft tissue swelling
  • Diagnosis
    • Plain radiographs may show soft tissue edema but are too insensitive to be used for this diagnosis.
    • MRI
    • Flexion/extension views – usefulness in acute setting is controversial; if done, should be in a subacute setting

Thoracolumbar Spine Injury
• Most occur at T9-T10
• Wedge/compression fracture (see Image #22)
  • Mechanism: axial loading with flexion
  • Acutely stable
  • Neurologic damage uncommon, treatment is symptomatic
  • >50% loss of anterior vertebral height may be associated with ileus and significant pain
• Burst fracture
  • Mechanism: axial loading
  • Vertebral endplate fractures with explosion of the body
  • May cause cord damage
  • Diagnosis with CT
• Distraction/seatbelt fracture
  • Associated with abdominal injuries
  • Failure of the spine in its posterior ligamentous and bony components
  • Chance distraction fracture: bony, disc, and/or ligamentous injury to upper lumbar spine, most commonly L2
• Fracture/dislocation
  • Mechanism: flexion rotation injury
  • Usually associated with cord damage
Sacral and Coccygeal Injury
- Neurologic findings are rare.
- Injuries tend to be from direct blows.
- Isolated coccygeal injuries are diagnosed by rectal exam and do not generally require radiographs.

General Management of Spinal Cord Injuries
- Methylprednisolone (30 mg/kg, followed by an infusion of 5.4 mg/kg/hr) for patients over age 13 with acute spinal cord injuries. This is continued for 24 hours if started within 3 hours after injury and for 48 hours if started between 3 and 8 hours after injury.
  - This is treatment is still controversial, as data have not clearly shown a benefit.
  - Patients who receive this treatment are also at increased risk for pulmonary and gastrointestinal infection and avascular necrosis.
  - Treatment is usually based on institutional protocols.
- Obtain proper imaging
- Transfer patient to a trauma or spinal cord center once other life-threatening injuries have been managed.

9.12 STROKE

Hemorrhagic Stroke
- Subarachnoid hemorrhage
  - Sudden onset ("thunderclap") of symptoms with rapid progression, caused by rupture of a cerebral aneurysm or arteriovenous malformation
  - Excruciating headache ("worst headache of life")
  - Nuchal rigidity, photophobia may be present from meningeal irritation
  - Warning or "sentinel" headaches are experienced by up to half of patients, caused by antecedent blood leakage or aneurysmal dilatation
  - Usually <50 years of age
  - No lateralizing symptoms
  - Associated with polycystic kidneys and aortic coarctation
  - Treatment
    - Urgent neurosurgical consultation
    - Opioids for pain control
    - Consider intubation for altered level of consciousness
    - Treat with nimodipine, 60 mg orally, to lessen chances of vasospasm and ischemic stroke
- Intracerebral hemorrhage
  - Focal neurologic deficits; may be clinically indistinguishable from occlusive strokes
  - Usually >50 years of age with atherosclerotic risk factors, particularly hypertension
  - Pontine lesions: pinpoint pupils, decerebrate posturing, coma
  - Cerebellar: vomiting, dizziness, cranial nerve VI palsy, brainstem compression
  - Treatment
    - Blood pressure treatment is recommended for systolic blood pressure >180 mm Hg and for diastolic pressure >105 mm Hg.
    - Nitroprusside is the recommended agent.
    - Lasix and mannitol may be used to lower intracranial pressure, but only in consultation with a neurologist/neurosurgeon.
    - Consider seizure prophylaxis with phenytoin.
May require intubation, owing to altered level of consciousness.
Reverse coagulopathies, which may worsen bleeding

Ischemic Stroke
- Account for >80% of strokes
- Usually present with obvious focal deficits that develop over minutes to hours
- Thrombotic strokes – usually caused by atherosclerosis
- Embolic strokes – usually cardiogenic (MI, valvular heart disease, atrial fibrillation, cardiomyopathy): hypokinetic chambers allow formation of clot, which embolizes to brain via carotids. Less commonly due to septic emboli from endocarditis or air or fat embolism.

Treatment
- Blood pressure control
  - Nonthrombolytic patients:
    - Diastolic >140 mm Hg - nitroprusside drip; aim for 10-20% decrease in diastolic blood pressure
    - Systolic >220 mm Hg, diastolic >120 mm Hg, MAP >130 mm Hg - labetalol 10-20 mg IV (nitroprusside, enalapril are alternatives)
  - Thrombolytic patients: 1 to 2 inches of nitropaste or 1 or 2 doses of 10 to 20 mg of labetalol for:
    - Systolic >185 mm Hg or
    - Diastolic >110 mm Hg
    - If more aggressive measures are needed to keep blood pressure below 185/110 mm Hg, then thrombolytics should not be used.
- Thrombolytics
  - Current recommendations state that thrombolytics must be given within 3 hours after onset of stroke.
  - Give alteplase (tPA), 0.9 mg/kg, up to 90 mg, with 10% as bolus and the rest infused over 60 minutes.
  - Although thrombolytics have been shown to resolve stroke symptoms, complication of serious intracranial hemorrhage can occur as well as bleeding at other sites.
- Inclusion criteria
  - Age >18 years
  - Well-established time of onset <180 minutes before treatment will begin
  - Clinical diagnosis of stroke with measurable neurologic defect
- Exclusion criteria
  - Evidence of hemorrhage on head CT
  - Minor or resolving stroke symptoms
  - Active internal bleeding (GI bleed within the past 21 days)
  - Known bleeding diathesis
  - Within 3 months after previous stroke, serious head trauma, or intracranial surgery
  - Within 14 days after major surgery or serious trauma
  - History of aneurysm or atrial venous malformation
  - Recent myocardial infarction
  - Witnessed seizure at stroke onset
  - Lumbar puncture within 7 days
  - Systolic pressure >185 mm Hg or diastolic pressure >110 mm Hg at time of treatment
Transient Ischemic Attack

- Neurologic deficit that has complete resolution within 24 hours; most last less than 1 hour
- Warning sign for future ischemic stroke, as 10% of patients with a TIA develop a stroke within 3 months; 5% of TIA patients develop a stroke within 2 days
- Patients with TIAs usually warrant hospital admission due to high risk of short-term complications; however, TIA scoring systems exist to guide disposition and differentiate low-risk from high-risk presentations.
- In some institutions, extensive workups (including carotid Doppler, MRA or CT angiogram, and echocardiogram) are available in the ED or expeditiously as an outpatient, and discharge with close follow-up can be considered.
- Discuss the initiation of antiplatelet agents (aspirin, clopidogrel) with the neurologist.

Stroke Syndromes

- Anterior cerebral artery stroke: contralateral sensorimotor deficits greater in the leg than in the arm. Personality changes may be seen with frontal lobe lesions.
- Middle cerebral artery stroke: sensorimotor deficits of contralateral face, arm, and leg; arm deficits greater than leg deficits. Receptive and expressive aphasias seen when stroke involves dominant hemisphere (usually the left hemisphere).
- Posterior circulation: crossed deficits (e.g., weakness in left side of face, right arm, right leg). Symptoms may include cranial nerve or cerebellar deficits (dizziness, nausea, vomiting, nystagmus), which are frequently subtle.
- Lacunar infarcts: small, deep infarcts seen in hypertensive and diabetic patients
  - Account for 20% of all strokes
  - Rapid onset of symptoms, most often an isolated pure motor or pure sensory defect, with no further progression
  - No language deficits or alteration in consciousness
  - Commonly occur in pons, internal capsule, basal ganglia, thalamus

9.13 HEAD TRAUMA

General

- Half of all trauma deaths are caused by head injury.
- Motor vehicle crashes account for 75% of serious head injuries.
- Head trauma is the primary injury among traumatically injured teens and young adults; the incidence peaks again among the elderly.
- Severity of injury
  - Minor injury: Glasgow Coma Scale (GCS) score of 13 to 15 (80% of patients). Many practitioners consider a GCS score of 14 or 15 to indicate minor injury, with a score of 13 considered moderate. Less than 1% of patients with minor brain injury have significant intracranial lesions.
  - Moderate injury: GCS score of 9 to 12 (10% of patients); 20% mortality
  - Severe injury: GCS score of 3 to 8 (10% of patients); 40% mortality; 60% in this category have multisystem injuries

Specific Injuries

- Scalp lacerations
  - May bleed copiously and can result in hypotension or exsanguination if not controlled
  - Large vessels are located between galea and dermis
  - Need to repair galea if it was lacerated
**Skull fractures**
- Simple linear – usually significant only if level of consciousness is depressed or fracture line crosses groove of middle meningeal artery
- Basilar – generally a clinical diagnosis confirmed with CT scan; raccoon eyes, hemotypanum, Battle's sign (see Image #52); antibiotics controversial with CSF leak; neurosurgical consultation
- Depressed – usually clinically apparent, requires neurosurgical evaluation

**Concussion** – “minor” head trauma
- Transient loss of consciousness (<5 min)
- Normal neuro exam and level of consciousness in ED
- Associated with postconcussive syndrome lasting up to 6 months
  - Insomnia, headache, personality change, memory loss, inattentiveness, depression
  - May have objective findings on neuropsychiatric tests
  - Requires follow-up

**Contusion**
- Cortical injury often associated with acute subdural hematoma
- CT: hemorrhage surrounded by edema
- Caused by coup and contrecoup injuries
- Should be referred to neurosurgery and admitted for observation (for increasing ICP)

**Intracerebral (parenchymal) hemorrhage** (see Image #53)
- Similar to contusion but deeper in brain
- In combination with contusion, a common cause of increased ICP and herniation

**Diffuse axonal injury**
- Most common cause of coma in head-injured patients
- Caused by shearing (inertial) forces in brain during sudden deceleration
- Intracranial pressure is always increased; CT may show diffuse edema
- Carries poor prognosis

**Epidural hematoma**
- Rare (<2% of serious head injury)
- Arterial bleeding under high pressure
- Characteristic lenticular (football shaped) hemorrhage on CT (see Image #39)
- Skull fracture in 90%
- Classic presentation is blow to temple, loss of consciousness, then a “lucid interval” followed by rapid progression of coma with increased ICP and lateralizing signs of uncal herniation
- Needs immediate surgery, prognosis is very good if diagnosis is made

**Subdural hematoma**
- Six times more common than epidural
- Bleeding largely from bridging veins and sinuses, under lower pressure
- CT shows crescent-shaped hemorrhage lining inside of skull (see Image #41)
- Often associated with extensive brain injury
- Accounts for high mortality even if evacuated early (about 30%)
- Mortality is over 60% if repair is delayed
- Acute subdural hematomas are symptomatic within 24 hours after trauma; patients present with altered level of consciousness and may have pupil inequality or motor deficits from brain edema.
b Chronic subdural hematomas become symptomatic 2 weeks or more after trauma
- May present with subtle signs and symptoms
- Some patients demonstrate unilateral weakness.
- Most patients cannot recall the trauma or report only a mild injury.
- Chronic subdural hematomas may appear isodense or hypodense to brain parenchyma.

- Penetrating injury
  - Stab wounds and impaled objects are uncommon.
  - Significance is related to focal area and vessels involved and contamination of brain
  - Gunshot wounds are more common; inflict massive blast effect, which results in high intracranial pressure, early herniation, poor outcome
  - Patients with penetrating injuries presenting in coma have almost universally poor/lethal outcome.

Management
- ABCs with C-spine immobilization – assume a concomitant cervical spine injury
- Intubate obtunded head-injured patients, optimizing intracranial pressure with pCO₂ of 30 to 35 mm Hg.
- Intubation considerations in head trauma
  - Lidocaine, 1.5 mg/kg IV – supposedly blunts the increased intracranial pressure that occurs with intubation and suppresses cough reflex
  - Pancuronium, 1 mg, or vecuronium, 1 mg IV (10% of the paralyzing dose), given prior to administration of succinylcholine decreases fasciculations and may reduce intracranial pressure; do only if time permits, as its benefit is not established
- CT is most useful for patients with depressed mental status or focal deficits.
- Intracranial pressure control
  - Mannitol, 1 gm/kg, causes osmotic diuresis and decreases intracranial pressure; may make hypovolemia worse
  - Diuretics – use cautiously if at all
  - Steroids – of no use in acute head injury
- Neurosurgical consultation

9.14 TUMOR-RELATED EMERGENCIES

General
- Of all patients with cancer, 15% to 20% will have neurologic complications.
- Neurologic complaints are occasionally the presenting symptom in systemic cancer.

Cerebral Herniation
- Occurs from an increase in intracranial pressure, which causes a shift in intracranial contents
- Brain parenchyma is forced through the tentorial opening and the foramen magnum.
- This can be caused by an expanding mass lesion or an acute hemorrhage from the tumor site.
  - Primary brain tumors account for half of all intracranial tumors.
  - Metastasis most commonly from breast, lung, colon, kidney, and testicular cancer and malignant melanoma.
- Herniation syndromes
  - Uncal herniation
    - Lateral mass displaces temporal lobe, compressing the upper brainstem
    - Rapid loss of consciousness, unilateral pupil dilation, and ipsilateral hemiparesis
o Central herniation
  ▪ Occurs from slowly expanding multifocal lesions
  ▪ Causes downward shift on pons
  ▪ Slow loss of consciousness, small reactive pupils, and Cheyne-Stokes respirations
  ▪ No focal signs

o Tonsillar herniation
  ▪ Large posterior fossa mass pushes the cerebellar tonsils through the foramen magnum
  ▪ Compresses medulla
  ▪ Causes rapid loss of consciousness, vomiting, hiccups, occipital headache, meningismus, and change in respiratory pattern

• Management
  o Rapid stabilization is required.
  o Intubation for altered consciousness
  o Consider hyperventilation to pCO2 of 25 to 30 mm Hg to decrease intracranial pressure.
  o Give mannitol, 1 g/kg IV.
  o Dexamethasone, 12 to 24 mg IV, has not been shown to improve outcomes but is sometimes given to decrease edema.
  o CT of the head should be obtained as soon as the patient is stable.
  o Neurosurgical consultation

Seizures
• Can be the result of intracranial mass as well as toxic or metabolic complications caused by tumor.
• When this cause is suspected, obtain electrolytes, glucose, CBC, calcium, magnesium, BUN, coagulation studies, liver function tests, CT of the head, and lumbar puncture if infection is suspected.
• Treat underlying cause.
• May give a loading dose of phenytoin

Epidural Cord Compression
• General
  o Caused by metastasis from lung, lymphoma, breast, or prostate
  o Lymphoma extends from the intervertebral lymph nodes.
  o Other cancers compress the spinal cord after invading the vertebral body.
• Physical findings
  o Back pain, either local or radicular
  o May be acute or insidious
  o Pain may increase with spinal percussion or straight leg raise at the site of the tumor.
  o May present with weakness or sensory findings
  o Some patients present with inability to walk.
• Management
  o Plain films may show evidence of metastasis in vertebral bodies.
  o Abnormal plain films should be followed by MRI.
  o Initiate treatment with dexamethasone to reduce cord edema.
  o Obtain radiation oncology consult for possible radiation treatment.
CNS Infection

- Cancer patients have decreased immune response.
- Treat with antibiotic coverage for immunocompromised patient, as previously described.
- Neutropenic patients may not have WBCs in the CSF.
CHAPTER 10
Obstetric and Gynecologic Disorders

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10.1 INFECTIONS

Chancroid
• *Haemophilus ducreyi*: gram-negative bacillus
• Incubation period, 2 to 5 days
• Vesiculopustular lesion develops, followed by a painful ulcer
• Distinguished from the nonpainful lesion of primary syphilis
• Tender unilateral fluctuant adenopathy (a bubo)
• Diagnosis: clinical
• Treatment: ceftriaxone, 250 mg IM x 1 dose; erythromycin, 500 mg PO QID x 7 days; azithromycin, 1 g PO x 1 dose; ciprofloxacin, 500 mg PO BID x 3 days

Condyloma Acuminatum
• Human papilloma virus
• Common name: venereal/genital warts
• Rule out other sexually transmitted diseases (STDs)
• Increased risk of cervical carcinoma
• Rectal, penile, and perineal lesions
• Occurs in soft vegetating clusters
• Treatment is usually done by primary care physician because long-term follow-up is needed to assess response.

Genital Herpes
• 70% to 95% of genital lesions are caused by HSV type II
• Causes fever, malaise, headache, myalgia, and adenopathy
• Primary lesion appears at 2 to 8 days (shallow, painful vesicles and ulcers)
• Diagnosis: antibody titer, culture, antigen testing of lesion
• Multinucleated giant cells are seen on Tzanck smear.
• Treatment shortens the duration of pain and systemic symptoms.
• Treatment for first episode: acyclovir, 200 mg PO 5x/day x 7 to 10 days or 400 mg PO TID x 7 to 10 days; famciclovir, 250 mg PO TID x 7 to 10 days; or valacyclovir, 1 g PO BID x 7 to 10 days
• Treatment for recurrent episodes: acyclovir, 200 mg PO 5x/day, or 400 mg PO TID x 5 days, or 800 mg PO BID x 5 days; famciclovir 125 mg PO BID x 5 days; or valacyclovir, 500 mg PO BID x 5 days or 1 g PO QD x 5 days
• Prophylaxis with antiviral agents decreases recurrences.
• Complications: hepatitis, meningitis, encephalitis, urinary retention (sacral root ganglia or urethral involvement)

**Lymphogranuloma Venereum (LGV)**

*Chlamydia trachomatis*

• Incubation: 3 days to 3 weeks
• Shallow, painless vesicles or ulcers with initial infection
• Painful inguinal nodes emerge 7 to 30 days later (unilateral in 70% of cases).
• Diagnosis: clinical or via serology to confirm
• Treatment: doxycycline, 100 mg PO BID x 21 days; erythromycin, 500 mg PO QID x 21 days
• Partners need treatment as well.

**Pelvic Inflammatory Disease (PID)**

*Neisseria gonorrhoea* and *Chlamydia trachomatis* are the most common causes, but other organisms that ascend from the cervix/vagina may cause PID as well.

• Risk factors: prior PID, IUD (only during first month of use), adolescence, multiple partners, smoking, menses
• Findings suggestive of PID
  - Lower abdominal pain
  - Tenderness on palpation of lower abdomen
  - Cervical motion tenderness
  - Adnexal tenderness
• The presence of one or more of the following criteria enhances the specificity of the diagnosis:
  - Temperature >38°C (100.4°F)
  - Vaginal/cervical discharge
  - Elevated ESR or C-reactive protein
  - Laboratory evidence of gonorrhea or chlamydial infection
  - White blood cells on a wet mount of vaginal secretions
• Admit if any of the following conditions is present:
  - Tubo-ovarian abscess (TOA)
  - Pregnancy
  - Peritoneal signs
  - IUD (because of increased rate of adnexal inflammatory masses)
  - Sepsis
• Complications: ectopic pregnancy, adhesions, TOA, infertility
• Fitz-Hugh-Curtis syndrome
  - Bacterial perihepatitis
  - Causes RUQ and shoulder pain
  - Produces violin strings, which are adhesions around the liver
• Treatment regimens (*2007 CDC update: fluoroquinolones are no longer recommended for treatment of gonococcal infections*)
  - Parenteral
    - Cefotetan or cefoxitin, 2 g IV, PLUS doxycycline, 100 mg IV/PO
    - Clindamycin, 900 mg IV, PLUS gentamicin, IV/IM (various doses acceptable)
    - Ampicillin/sulbactam, 3 g IV, PLUS doxycycline, 100 mg IV/PO
Syphilis

- *Treponema pallidum* (spirochete)
- Primary syphilis: painless ulcer (chancre, see Image #15); resolves spontaneously; serology can be negative
- Secondary syphilis: rash (maculopapular) on palms and soles, fever, arthralgia, condyloma latum; painless lymphadenopathy
- Tertiary syphilis
  - The progressive inflammatory stage of tertiary syphilis can mimic many medical conditions, particularly of the neurologic and cardiovascular systems.
  - Scarring of the tunica media leads to syphilitic aortitis and narrowed coronaries with nervous system involvement in 40% of patients with cardiovascular syphilis.
- Positive dark field microscopy for primary and secondary lesions
- Positive serology: VDRL, RPR, FTA-ABS
- Treatment
  - Benzathine penicillin is strongly preferred (there are various treatment regimens based on the stage of the syphilis).
  - Pencillin-allergic patients should receive appropriate testing and desensitization prior to treatment with penicillin.
  - Alternative, but second-line, treatment regimens include doxycycline, ceftriaxone, and azithromycin.

*T. vaginalis*

- Grey-yellow, frothy, malodorous vaginal discharge
- Positive wet mount with visible, motile organism
- “Strawberry” cervix on exam
- Treatment: metronidazole, 2 g PO x 1 dose or 500 mg PO BID x 7 days
- Treat partner.

*Gardnerella Vaginosis*

- Gram-negative rod, facultative anaerobe
- Copious vaginal discharge; fishy odor, positive sniff test
- Presence of “clue cells” (vaginal epithelial cells with adherent bacteria)
- pH >4.5
- Treatment: metronidazole, 2 g po x 1 dose, 500 mg PO BID x 7 days, or 5 g intravaginal x 5 days; clindamycin, 300 mg PO BID x 7 days or 5 g intravaginally QHS x 7 days

*Candida Vaginitis*

- Part of the normal vaginal flora
- Risk factors for infection
  - Diabetes mellitus
  - Use of oral contraceptives
  - Recent antibiotics
• "Cottage cheese," thick, white, non-odorous vaginal discharge
• KOH wet mount shows hyphae and spores
• Vaginal pH is normal (4.0–4.5)
• Oral treatment: fluconazole, 150 mg PO x 1 dose; itraconazole, 200 mg PO BID x 1 day
• Intravaginal treatment: multiple regimens and forms of treatment are available with clotrimazole, miconazole, nystatin, and terconazole

10.2 OVARIAN DISEASE

Ovarian Cyst
• A follicular cyst forms during the first 2 weeks of the menstrual cycle, then an ovum is released.
• A corpus luteum cyst forms during the last 2 weeks of the menstrual cycle.
• Complications of ovarian cysts include ovarian torsion and cyst rupture.
• Cysts can cause abdominal pain and vomiting.
• Physical examination may demonstrate tender adnexa with or without mass or cervical motion tenderness.
• MUST RULE OUT ECTOPIC PREGNANCY
• Diagnosis: ultrasound may show cysts or pelvic free fluid; laparoscopy sometimes is required
• Treatment: supportive care with analgesics and gynecology follow-up if stable; gynecology consult for significant hemorrhage or peritoneal signs, as operative intervention may be necessary

Ovarian or Adnexal Torsion
• Ovary twists on pedicle
• Associated with tumors or cysts
• Causes severe abdominal/pelvic pain that is constant and unilateral.
• Nausea and vomiting are often present.
• The patient is usually afebrile.
• Vaginal bleeding is uncommon.
• Exam: unilateral abdominal tenderness, rebound tenderness, or detection of a mass
• Diagnosis: ultrasound will show ovarian cyst or mass; color flow Doppler can be diagnostic, showing decreased flow to the ovary; laparoscopy may be necessary if ultrasound is indeterminate
• Treatment: gynecology consultation for operative treatment

Ovarian Tumor
• Peak incidence: 60 to 70 years of age
• Second most common gynecologic malignancy
• Highest mortality among gynecologic malignancies
• Often asymptomatic
• Exam: fixed unilateral adnexal mass
• Diagnosis: pelvic ultrasound, CT scan, and laparoscopy can identify an ovarian mass
• Treatment: gynecology referral

10.3 UTERINE DISEASE

Endometriosis
• Caused by endometrial tissue found outside the uterus, most commonly on the ovaries, fallopian tubes, bladder, and abdominal cavity
• Causes constant pelvic pain associated with menses, dyspareunia, hypermenorrhea, and infertility
Exam: adherent uterus, adnexal induration, pelvic tenderness

Diagnosis: ultrasound may show an endometrioma, but laparoscopy is required for definitive diagnosis

Treatment: analgesics and long-term treatment with hormonal therapy or surgery by a gynecologist

Complications: bleeding, pain, and infertility

Uterine Tumor
- Most common uterine malignancy is endometrial carcinoma.
- Peak incidence: 60 to 70 years of age
- Risk factors: continuous estrogen, obesity, diabetes, and hypertension
- Pap smear detects only 20%
- Symptoms include abnormal bleeding and painless uterine enlargement.
- Diagnosis: D&C or uterine/endometrial biopsy
- SUSPECT IN POSTMENOPAUSAL WOMEN WITH BLEEDING; ENSURE THEY RECEIVE GYNECOLOGIC FOLLOW-UP.

Dysfunctional Uterine Bleeding
- Irregular, excessive bleeding
- Common at the extremes of reproductive age
- An anovulatory menstrual cycle is the most common cause, leading to continuous estrogen stimulation of the endometrium.
- Measure hCG level to rule out pregnancy-related bleeding
- Treatment
  - Estrogen therapy: oral conjugated estrogen, 10 mg/d for 5 to 10 days → referral to a gynecologist
  - Oral contraceptives: ethinyl estradiol/norethindrone, 1 mg (4 tabs for 7 days) → referral to a gynecologist
  - Progesterone: medroxyprogesterone acetate, 10 mg/day for 10 days → referral to a gynecologist
  - All women older than 35 require an endometrial biopsy to rule out endometrial carcinoma.

Uterine Prolapse
- Herniation of the uterus into the vagina
- Common in postmenopausal, multiparous women
- Presents with pelvic, rectal, or inguinal pain or “heaviness”
- Examination shows a firm, mobile vaginal mass
- Treated with stool softeners, pessaries, and surgery

10.4 PREGNANCY

Common Terminology
- Gravid (G): number of times pregnant
- Parity (P): subdivided into the following:
  - “T”: number of term gestations
  - “P”: number of preterm deliveries/gestations
  - “A”: number of abortions (spontaneous or voluntary)
  - “L”: number of living offspring

Confirmation of Pregnancy
- Clinical suggestion
  - Any female patient of childbearing age is pregnant until proven otherwise.
  - Missed menses, previous abnormal menses
o Fatigue, nausea, vomiting, anorexia
o Urinary frequency
o Uterine size
  - Size of an orange at 6 to 8 weeks
  - Reaches the symphysis pubis at 10 weeks
  - Reaches the umbilicus at 20 weeks
  - After the uterus grows beyond the umbilicus, measure the size of the uterus from the symphysis pubis. Each centimeter is equal to 1 week of pregnancy.

- Laboratory testing
  o Enzyme-linked immunoassay (ELISA) is the most frequently used method to detect the beta subunit of hCG.
  o Urine testing
    - Sensitive at hCG levels of 20 mIU
    - Detectable 2 or 3 days after implantation (9–11 days after ovulation)
  o Serum testing
    - Qualitative – sensitive at hCG levels of 10 mIU
    - Quantitative – sensitive at hCG levels of 5 mIU
      - Beta hCG increases at least 66% every 48 hours for the first 8 weeks
      - Peak levels: 50,000 to 100,000
  o Serial measurements of quantitative hCG are used to confirm healthy growth – NOT TO DATE PREGNANCY
    - 15% of women with normal intrauterine pregnancies have abnormal rises
    - 30% to 40% of women with initially normal rises miscarry
    - 15% to 20% of women with initial normal rises have ectopic pregnancies
    - Abnormally high levels of hCG (>100,000 mIU/mL) may be indicative of a molar pregnancy or multiple gestations.
- Imaging, ultrasonography
  o Transabdominal
    - Gestational sac is visible at 5 to 6 weeks of pregnancy.
    - Fetal pole and fetal heart activity are visible at 7 to 8 weeks of pregnancy.
Transvaginal
  ▪ Greater resolution
  ▪ Optimized if the bladder is empty
  ▪ Landmarks are seen 1 week earlier than with transabdominal ultrasound.

Physiologic Changes of Pregnancy

- Vital signs
  - HR: increases 15 to 20 beats/min
  - BP: systolic, 5- to 10-mm-Hg decrease; diastolic, 10- to 15-mm-Hg decrease
  - RR: unchanged

- Cardiovascular
  - Increased: cardiac output (30%-50%) and blood volume (the plasma increases more than the RBCs, so the hematocrit decreases)
  - Decreased: systemic vascular resistance
  - Venal caval compression occurs in 10% to 15% of women who lie flat on their back.

- Pulmonary
  - Increased: tidal volume (30%-40%)
  - Decreased: functional residual capacity (FRC)
  - Unchanged: vital capacity
  - Respiratory alkalosis in third trimester

- Renal
  - Increased: glomerular filtration rate
  - Decreased: creatinine, BUN

- Coagulation
  - Increased: factors VII, VIII, IX, X
  - Unchanged: bleeding time, PT, PTT
  - Thromboembolism is the primary cause of maternal mortality.

- Hematologic
  - “Normal leukocytosis” as high as 15,000 cells/μL

- Gastrointestinal
  - Decreased: gastric tone/mobility, gallbladder emptying

Hyperemesis Gravidarum

- Definition: vomiting severe enough to cause weight loss, dehydration, starvation ketoacidosis, ketonuria, and alkalosis from loss of HCl, hypokalemia
- Incidence: peaks at 8 to 12 weeks of pregnancy
- Symptoms usually resolve by 16 weeks of pregnancy
- Increased incidence with molar pregnancy and multiple gestations
- Diagnosis: clinical + assess ketonuria by urinalysis
- Management
  - IV hydration with 5% glucose LR or NS
  - Antiemetics
  - Correct electrolytes
  - Dietary advice: eat small, light, frequent meals
  - Vitamin B6 (pyridoxime) supplementation may be helpful
CHAPTER 10 • Obstetric and Gynecologic Disorders

First-Trimester Bleeding

• General
  o Occurs in 20% to 25% of all pregnancies
  o 50% of pregnancies will proceed normally, 50% will end in miscarriage
  o 50% incidence of chromosomal anomalies in abortuses

• Differential diagnosis
  o Implantation bleeding: minimal bleeding at the time of the first missed period
  o Abortion: termination before 20 weeks (<12 weeks in 80%)
    ▪ Threatened: uterine bleeding without cervical dilation or effacement
    ▪ Inevitable: uterine bleeding with cervical dilation but without placental or fetal passage through the cervix
    ▪ Incomplete: passage of some but not all tissue through the cervix; usually occurs between 6 and 14 weeks
    ▪ Complete: spontaneous expulsion of all fetal and placental tissue before 20 weeks
  o Blighted ovum: ultrasonographic visualization of a gestational sac without a fetus after more than 7.5 weeks of gestation
  o Missed abortion: dead fetus retained in uterus >5 weeks after death (increased risk for coagulopathy)
  o Septic abortion: any type of abortion accompanied by uterine infection
    ▪ 1% to 2% of spontaneous abortions; fever, leukocytosis, uterine tenderness

• Management
  o Ultrasound
    ▪ Rule out ectopic pregnancy; assess gestational sac and fetal heart activity
  o Threatened abortion
    ▪ Expectant home management, pelvic rest, counseling
  o Inevitable and incomplete abortion
    ▪ Consider CBC, type and screen, or a coagulation panel
    ▪ Obstetric consultation for surgery or oxytocin therapy
  o Missed abortion
    ▪ Requires gynecologic management for evacuation
    ▪ Consider PT/PTT
  o Septic abortion
    ▪ CBC, UA, blood/uterine cultures
      ▪ IV antibiotics with broad-spectrum coverage
      ▪ Surgical evacuation
  o Rh-immune globulin
    ▪ 75% of pregnant women have transplacental hemorrhage
    ▪ 2% to 3% risk of isoimmunization in Rh-negative mothers in first trimester
    ▪ Current recommendation: All Rh-negative unimmunized women with antepartum bleeding should receive 300 micrograms of RhoGAM IM (50 micrograms if in first trimester).

Ectopic Pregnancy

• General
  o 1.5% of pregnancies
  o Incidence of heterotopic pregnancy (both an ectopic and intrauterine pregnancy) is 1/4,000 to 30,000 in the general population (1% to 8% in patients with in vitro fertilization)
  o 10% maternal mortality
o Missed on first office visit 50% of the time and on first ED visit 36% of the time
o Dizziness and syncope are uncommon.
o 90% have pelvic or abdominal pain or vaginal bleeding

- Risk factors
  o Previous ectopic
  o Current intrauterine device (IUD)
  o History of pelvic inflammatory disease (PID)
  o Abdominal or pelvic surgery
  o Infertility
  o Tubal ligation

- Serum hCG
  o Quantitative hCG levels are non-diagnostic themselves (low, normal, or high levels can be present).
  o Plateauing or very low levels are more common.
  o Appropriate hCG levels DO NOT rule out ectopic pregnancy.
  o Low hCG levels should not be dismissed as an early IUP.
  o Women with hCG levels above the discriminatory zones listed below and without an IUP visualized by ultrasonography should be considered to have an ectopic pregnancy until proven otherwise.
  o Discriminatory zones
    - 1,500 to 2,000 mIU/ml for transvaginal ultrasonography
    - 6,500 mIU/ml for transabdominal ultrasonography

- Diagnosis
  o Definitive: ectopic fetal heart activity and ectopic fetal pole
  o Suggestive: adnexal mass without IUP or cul-de-sac fluid without IUP (see Image #18)
  o Indeterminate: no intrauterine findings, single gestational sac, multiple intrauterine echoes

- Differential diagnosis (appropriate hCG level without visualization of IUP)
  o Ectopic pregnancy
  o Early IUP
  o Completed spontaneous abortion (hCG levels take approximately 2–4 weeks to return to normal after a miscarriage)

- Culdocentesis (has been almost completely replaced by ultrasound)
  o Indications: unstable patient who cannot tolerate time for ultrasound or if ultrasound is not available; to determine if intraperitoneal hemorrhage is present
  o Interpretation of culdocentesis findings
    - Fluid is present in 85% of ruptured and 65% of nonruptured ectopic pregnancies
    - Negative: clear fluid
    - Positive: non-clotting blood
    - Indeterminate: dry tap
    - A hematocrit of <12 in the aspirated fluid suggests a corpus luteum cyst rupture

- Management
  o Unstable – ABCs; immediate gynecologic consultation for surgery
  o Stable – if low degree of suspicion may be followed as outpatient with serial quantitative hCGs and precautions for immediate reevaluation
  o Nonruptured ectopics less than 4 cm with hCG < 3,500 mIU/ml may be treated with methotrexate therapy; otherwise the treatment is surgical
Molar Pregnancy

- Placental proliferation without fetal tissue
- Mimics incomplete or threatened abortion
- Painless vaginal bleeding
- Abnormally high hCG level
- Increased incidence of hyperemesis gravidarum
- Increased incidence of preeclampsia
- Uterine size greater than expected for gestational age
- Diagnosis: on ultrasound, a “snowstorm”-like pattern
- Increased risk of choriocarcinoma

Second- and Third-Trimester Bleeding

- General
  - Frequency: 4%
- Differential diagnosis
  - Placenta previa (20%)
  - Placenta abruptio (30%)
  - Cervical or vaginal lesions or tumors
  - Preterm labor
  - Incompetent cervix
  - Molar pregnancy
- General principle
  - Sterile speculum exam is CONTRAINDIATED until placenta previa is ruled out.
  - Painless third-trimester bleeding = placenta previa (or molar pregnancy)

Placenta Previa

- General
  - Definition: implantation of the placenta over the internal cervical os
  - Three types: total, partial, marginal
  - Frequency: 1 in 250 live births
- Mechanism
  - Separation of placenta from lower uterine segment as uterine thinning occurs; as bleeding continues, further separation occurs
- Presentation
  - PAINLESS bright red vaginal bleeding during the third trimester
- Diagnosis
  - Ultrasound (preferably in OR environment)
- Management
  - Depends on gestational age of fetus (lung maturity)
  - Observation versus C-section
  - ABCs, two large-bore IV lines, type and cross for a minimum of 2 units of packed red blood cells
  - Consider tocolysis
  - Painful third-trimester bleeding typically indicates placental abruption.
Placenta Abruptio

- General
  o Definition: separation of normally implanted placenta from decidua basalis of the uterus at more than 20 weeks' gestation and prior to birth
  o Frequency: 1 in 85 to 200 live births
  o Vaginal bleeding may or may not be present.
  o If hemorrhage evolves away from the internal cervical os, abdominal pain and shock may be the only presenting findings.
- Risk Factors
  o Multiparity
  o Increased maternal age
  o History of abruption
  o Preeclampsia
  o Trauma
  o Smoking
  o Cocaine use
- Mechanism
  o Small arterial vessels in decidua rupture
  o Bleeding also from fetal or placental vessels
  o Accumulation of blood increases separation of placenta
- Management
  o Depends on stage
  o Minimal
    - Vaginal bleeding is present, separation is minimal, no concealed hemorrhage
    - Hospitalize; deliver if term
  o Moderate
    - Concealed hemorrhage, uterine tenderness, fetal distress, maternal compromise is not yet present
    - Deliver
  o Severe
    - Extensive concealed hemorrhage, uterine tenderness, maternal compromise with coagulopathy, and potential fetal death
    - Maternal stabilization and delivery

Medical Complications of Pregnancy

- Hypertension
  o General
    - Incidence, 5% to 7%
  o Definitions
    - Chronic hypertension is present before pregnancy and lasts >6 weeks postpartum.
    - Chronic hypertension with superimposed pregnancy-induced hypertension (PIH) – preexisting hypertension with an exacerbation during pregnancy
    - Transient hypertension – transient elevation during pregnancy
Pregnancy-induced hypertension

- Predisposing factors
  - Extremes of age
  - Primiparas (<20 years of age are at greatest risk)
  - Patient history/family history
  - Hypertension
  - Diabetes mellitus
  - Renal, vascular, or connective tissue diseases
  - Molar pregnancies
  - Multiple gestation

Preeclampsia

- Definition: hypertension >20 weeks + edema and/or proteinuria

<table>
<thead>
<tr>
<th>Mild</th>
<th>Severe</th>
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<tbody>
<tr>
<td>Rise in systolic BP by 30 mm Hg</td>
<td>Systolic BP &gt;160 mm Hg</td>
</tr>
<tr>
<td>Rise in diastolic BP by 15 mm Hg</td>
<td>Diastolic BP &gt;110 mm Hg</td>
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<tr>
<td>Pressure ≥140/90</td>
<td>End organ damage:</td>
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<tr>
<td></td>
<td>Proteinuria</td>
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<td>Oliguria</td>
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<td>Epigastric pain</td>
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<td>Cerebral or visual disturbances</td>
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<td>Pulmonary edema</td>
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- HELLP (Hemolysis, Elevated Liver enzymes, Low Platelets)
  - Presentation
    - Severe RUQ or epigastric pain; nausea/vomiting
    - Platelet count <100,000
    - Hemolysis – burr cells and schistocytes (normal PT, PTT, fibrinogen)
    - LDH >600 IU
    - Bilirubin >1.2 mg/dl
    - SGOT (AST) >72 IU/L
    - Increased maternal mortality due to hepatic or splenic hemorrhage

- Management of severe preeclampsia
  - Hospitalization
  - ABCs, check glucose, IV, fetal monitoring, left lateral decubitus positioning
  - Labs: CBC with platelet count, renal function studies, LFTs, coagulation profile, magnesium
  - Head CT: used to look for intracranial abnormalities when there is no history of pregnancy-induced hypertension or in patients unresponsive to therapy
  - Do NOT use diuretics to control edema or blood pressure (decreases intravascular volume).
  - Magnesium therapy
    - Membrane stabilizer and vasodilator
    - Seizure prophylaxis is indicated in severe preeclampsia.
      - Loading dose: 4 to 6 g IV over 20 min in 100 ml of fluid
      - Maintenance: 2 g/hr (range, 1–3 g)
      - IM dosing is not recommended but can be used if close observation is not possible.
      - Therapeutic level: 4 to 6 mEq/L
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Eclampsia

- **Definition:** preeclampsia + seizure or coma
- **Premonitory symptoms**
  - Headache (80%)
  - Generalized edema (50%) + proteinuria
  - Visual changes (40%)
  - Abdominal pain with nausea (20%)
- **Signs and symptoms**
  - SBP >160 mm Hg and/or DBP >100 mm Hg
  - Hyperreflexia
  - RUQ/epigastric pain
- **Laboratory**
  - Glucose
  - CBC (hemolytic anemia, thrombocytopenia – HELLP)
  - CHEM-7 (renal function)
  - UA (proteinuria)
  - LFTs (potential hepatocellular injury – HELLP)
  - Coagulation profile (PT/PTT, fibrin split products, fibrinogen)
- **Management**
  - General: ABCs, control blood pressure, anticonvulsants, delivery
  - Anticonvulsant therapy
    - Loading dose: magnesium sulfate, 2 to 6 grams of 20% solution over 2 to 4 minutes
    - Maintenance: 2 g/hr (range, 1–3 g/hr)
    - Seizures are almost always controlled with adequate doses of magnesium.
  - Observe for toxicity (see above)
  - Unresponsive – conventional anticonvulsants (benzodiazepines and/or phenytoin)
  - Antihypertensive
    - Hydralazine: 5 mg IV loading, then 5 to 10 mg IV q20 min, maximum 30 mg IV
    - Labetalol: 20 mg IV, slow infusion, repeat 20 mg doses q10 min
  - Radiologic evaluation: head CT is usually indicated for patients with severe headache

- **Side effects**
  - Flushing
  - Warmth
  - Headache
  - Blurred vision
- **Toxicity**
  - Hypotension
  - Respiratory depression/apnea
  - Hyporeflexia
  - Altered mental status
  - Decreased urinary output
  - Cardiac arrest
  - Antidote: calcium gluconate 10%, 10 ml over 3 minutes
Pyelonephritis

- Presentation
  - Fever, dysuria, frequency, urgency, flank pain, chills, rigors, vomiting, anorexia, dehydration

- Management
  - Admit
  - Labs: urine and blood cultures
  - Intravenous antibiotics (cephalosporins, aminoglycosides)
  - For women at >20 weeks' gestation, monitor on labor unit

Asthma

- Incidence: 4%

- Management
  - Treat the same as nonpregnant patients
  - Inhaled β₂-agonists and corticosteroids are the mainstays of treatment.
  - Rule out concomitant respiratory infections

Pneumonia

- Pathophysiology: alterations in immune function (decrease in cell-mediated cytotoxicity by lymphocytes and decrease in the number of helper T lymphocytes)
- Pathogens: Streptococcus pneumoniae, Haemophilus influenza, varicella, TB, viruses (more virulent during pregnancy)
- Morbidity: potential fetal risk with high fever, hypoxemia
- Management: oxygen, hydration, antipyretics, antibiotics
- Special concerns
  - Varicella pneumonia: more severe in pregnancy; mortality 10% to 35%; hospitalize pregnant women with varicella and cough; consider IV acyclovir
  - Influenza: with symptoms >5 days, suspect bacterial superinfection. Consider anti-influenza antivirals (all pregnancy class C): amantadine; oseltamivir; rimantadine; zanamivir

Thromboembolic Disease

- Causes 16% of obstetric mortality
- In patients with a history of DVT and/or PE, prophylactic heparin is recommended after 34 weeks of pregnancy
- Pathophysiology: hypercoagulable state due to increased coagulation levels and venous stasis
- Clinical findings are generally unreliable (DVT and PE)
- Diagnostics
  - D-Dimer is of questionable value because pregnancy can make it positive.
  - Doppler ultrasonography: diagnostic study of choice (confirm abnormal study with patient placed in left lateral decubitus position)
  - Venography: gold standard but rarely performed today
  - Technetium-labeled V/Q
  - Helical CT: more sensitive than V/Q and less fetal radiation exposure, but studies in pregnant patients are lacking
- Treatment
  - Heparin: fetal risks—fetal osteoporosis, thrombocytopenia, prematurity, miscarriage
  - Coumadin: CONTRAINDICATED (miscarriage, fetal malformation)
o Enoxaparin: recent studies have demonstrated safety and efficacy of low-molecular-weight heparin, including lower risk of osteoporosis and thrombocytopenia

Gynecologic Complications of Pregnancy

• Abdominal pain
  o Ovarian cyst rupture
    ▪ Pathophysiology: corpus luteum is a physiologic cyst that originates in the Graafian follicle, is supported by hCG, and produces progesterone to support pregnancy during first 6 to 7 weeks
    ▪ Presentation:
      □ Rupture occurs in first trimester
      □ Similar to ruptured ectopic pregnancy
      □ Abrupt onset of unilateral abdominal/pelvic pain
      □ Symptoms are precipitated by intercourse or other activity.
      □ Pelvic exam shows unilateral adnexal tenderness (usually without a palpable mass)
    ▪ Diagnosis
      □ Ultrasound may show fluid in the cul-de-sac
      □ Culdocentesis is rarely performed, but a hematocrit <12% in the aspirated fluid is unlikely to be from an ectopic pregnancy
    ▪ Conservative management with analgesics, although surgery may be needed in severe cases of hemoperitoneum
  o Ovarian torsion
    ▪ Pathophysiology: corpus luteum cyst predisposes to torsion
    ▪ Occurs during the first two trimesters
    ▪ Presents with the abrupt onset of unilateral abdominal/pelvic pain with nausea
    ▪ Exam shows an enlarged, palpable adnexa; peritoneal signs are variably present
    ▪ Diagnosis
      □ Ultrasound may show a cystic, solid, or complex adnexal mass or abnormal adnexal blood flow
      □ Laparoscopy is required to make a definitive diagnosis.
      □ 70% are misdiagnosed preoperatively
  o Premature rupture of membranes
    ▪ Morbidity: increased intrauterine infection if delivery occurs >24 hours after membrane rupture
    ▪ History: color and odor of fluid
    ▪ Diagnosis
      □ Evaluate fluid by sterile speculum exam.
      □ Amniotic fluid turns nitrazine paper blue and develops "fern-like" crystals visible under the microscope as it dries.
    ▪ Management: OB consultation
  o Premature labor
    ▪ Definition: labor at <37 weeks' gestation
    ▪ Management
      □ Rule out infections
      □ OB consultation
      □ Tocolysis: β-agonist, terbutaline, 0.25 to 0.5 mg SQ q2h (with consultation)
Sexually transmitted diseases

- Bacterial vaginosis
  - Incidence: 15% to 20% of pregnancies
  - Morbidity: amnionitis, premature rupture of membranes, prematurity, postpartum infection
  - Treatment: pregnant women with symptomatic bacterial vaginosis should be treated with metronidazole, 250 mg PO TID x 7 days (avoid in the first trimester), or clindamycin, 300 mg PO BID x 7 days

- *Candida albicans*
  - Incidence: increased due to high estrogen levels
  - Treatment: clotrimazole or miconazole, 200 mg intravaginally QHS x 7 days

- Trichomoniasis
  - Incidence: rarely aggressive in pregnancy
  - Treatment: treat those who are symptomatic with 2 grams of metronidazole PO x 1 dose (defer treatment until after the first trimester)

- *Chlamydia trachomatis*
  - Screen during pregnancy
  - Morbidity: preterm labor, postpartum endometritis, neonatal conjunctivitis, pneumonia
  - Treatment: azithromycin, 1 g PO x 1 dose; erythromycin, 500 mg PO QID x 7 days; or amoxicillin, 500 mg PO TID x 7 days

- Herpes simplex
  - Treatment: culture new lesions to identify patients at risk for perinatal transmission
  - Oral acyclovir as in nonpregnant patients (pregnancy class C)

- *Neisseria*
  - PID is rare in pregnancy but may develop during the first trimester.
  - There is a higher risk of disseminated infection in pregnancy.
  - Morbidity: a third-trimester complication is gonococcal ophthalmia
  - Treatment is unchanged by pregnancy. Treat for *Chlamydia* as well.

Surgical Complications of Pregnancy

- Appendicitis
  - General
    - Incidence: same as in nonpregnant patients
    - Most common surgical emergency in pregnancy
    - Outcomes are worse due to delays in diagnosis and an increased perforation rate of approximately 30%
    - Unperforated appendicitis is associated with 5% perinatal mortality; perforated appendicitis is associated with 27% perinatal mortality
  - Pathophysiology
    - Appendix is displaced clockwise into the right upper quadrant after the third month of pregnancy.
  - Presentation
    - During the first half of pregnancy, it presents similar to appendicitis in nonpregnant patients.
    - Anorexia (60%–70%)
    - Nausea or vomiting
    - Right-sided pain is the most constant finding but is less reliable in later stages of pregnancy.
    - May lack fever, tachycardia
• Diagnosis
  • Leukocytosis is nonspecific and variable.
  • Urinalysis: absence of bacteria with pyuria can help distinguish appendicitis from pyelonephritis
  • Ultrasound-guided compression technique is highly specific but poorly sensitive for appendicitis.
  • Abdominal CT delivers a large amount of radiation (>5 rads) to the fetus and is used sparingly in pregnancy.

• Management
  • ABCs, NPO, IV hydration, fetal monitoring
  • Early surgical and obstetric consultation
  • Admission for serial evaluations if the diagnosis is unclear; surgery if appendicitis strongly suspected

• Cholecystitis
  • General
    • Incidence: slight increase during pregnancy (4%)
    • 50% symptomatic, 20% of these have major complications
  • Pathophysiology
    • Increased gallbladder volume combined with less efficient contraction
  • Presentation
    • Similar to nonpregnant state
  • Diagnosis
    • Ultrasound (93% sensitive)
  • Management
    • Most patients are managed conservatively.
    • Fetal loss with surgery is approximately 5%
    • With secondary pancreatitis, there is a 50% fetal loss rate.
    • ABCs, IV hydration, analgesics, and antibiotics

Trauma During Pregnancy
• General
  • Most frequent cause of nonobstetric maternal death (motor vehicle crashes, falls, penetrating injury)
  • Trauma scoring systems do not reliably predict outcome in pregnant women.
  • Outcome in trauma is good if immediate catastrophes do not occur.
  • Continuous fetal monitoring (4 hours for immediate adverse outcomes) predicts:
    • Abruptio
    • Fetal death
    • Preterm delivery
    • PROM

• Anatomic changes
  • Displacement of gravid uterus off the IVC may increase cardiac output 25%, so place all pregnant trauma patients at >20 weeks’ gestation in the left lateral decubitus position.
  • Increased risk for aspiration
Cephalad displacement of abdominal contents
- Protective effect in blunt abdominal trauma
- More intestinal injury in upper abdominal penetrating injury
- Bladder is more susceptible to injury
- Uterus increases in size and blood flow and is more susceptible to massive blood loss when injured.

Evaluation and management principles
- Position in left lateral decubitus position, as described above
- Hypervolemia allows 30% to 35% blood loss before signs of hypovolemia occur.
- Vasopressors decrease uterine blood flow (ephedrine is the preferred vasopressor).
- Pelvic exam is mandatory; check for ferning and use nitrazine paper (amniotic fluid turns it blue).
- Use the supraumbilical approach in an open diagnostic peritoneal lavage.
- **MATERNAL STABILIZATION IS THE MOST IMPORTANT FACTOR IN DETERMINING FETAL SURVIVAL.**

Specific injuries
- Seat belt injuries
  - Three-point systems reduce the risk of maternal and fetal injury.
- Pelvic fracture
  - Increased risk of:
    - Hemorrhage
    - Lacerations (urethra, bladder, ureter, vagina)
    - Fat embolism
    - Lumbar plexus injury
    - Fetal skull fracture
  - C-section rate is only 5% to 10%, as there is no automatic contraindication to vaginal delivery.
- Intra-abdominal injuries
  - Most common injuries: splenic rupture, kidney injury, liver laceration
  - Uterine rupture is rare, because it requires a tremendous amount of force.
- Placental abruption
  - Minor trauma: 1%-5% incidence
  - Major trauma: 20%-50% incidence
  - Presents with vaginal bleeding, uterine irritability, contractions
- Feto-maternal hemorrhage
  - UNIVERSAL in trauma
  - All Rh-negative mothers must receive RhoGAM.
  - Kleihauer-Betke test: Identifies and quantifies fetal-maternal hemorrhage based on observation of fetal nucleated RBCs in maternal blood. Most labs screen for feto-maternal hemorrhage of 5 ml or more.

Cardiac Arrest and Post-Mortem Cesarean Section
- If there is no response to ACLS following maternal arrest, thoracotomy and open chest massage may improve maternal and fetal outcome.
- Timely emergency C-section has been shown to improve venous return and cardiac output in the mother. It is not a maneuver simply to save the fetus.
- Vertical, classic C-section should be initiated after 4 minutes of maternal resuscitation, with the goal of having the child delivered by 5 minutes after the arrest.
- Obtain neonatal consultation, if possible.
CHAPTER 10 • Obstetric and Gynecologic Disorders

- Improved fetal survival if:
  - Gestational age >28 weeks
  - Interval between death and delivery
    - <5 minutes: Excellent
    - 5–10 minutes: Good
    - 10–15 minutes: Fair
    - 15–20 minutes: Poor
    - >20 minutes: Unlikely

Drugs That are Safe in Pregnancy
- Antimicrobials
  - Penicillins, cephalosporins
  - Erythromycins except estolate
  - Imidazoles and nystatin
  - Clindamycin
  - INH, ethambutol
  - Nitrofurantoin and sulfonamides, except in third trimester (hemolysis and increased risk of kernicterus in newborn)
  - CONTRAINDICATED: tetracyclines, streptomycin, tobramycin, fluoroquinolones
- Vaccines
  - Hepatitis B immune globulin, tetanus immune globulin, rabies
  - CONTRAINDICATED: live attenuated vaccines (measles, mumps, varicella, rubella, smallpox)
- Antiemetics
  - Metoclopramide and ondansetron are the only class B medications; the remainder are class C, but the risk of starvation and ketosis from vomiting and dehydration outweigh the risk of using antiemetics, so they are commonly used.
- Analgesics
  - Acetaminophen
  - Nonsteroidals and ASA should be avoided in the third trimester.
- Anticoagulants
  - Heparin
  - Enoxaparin
  - CONTRAINDICATED: coumadin
- Anti-hypertensives
  - Methyldopa, β-blockers, calcium channel blockers
- Asthma
  - β-Agonists, terbutaline, corticosteroids, theophylline
  - Contraindicated while breastfeeding
  - Barbiturates, laxatives, ephedrine, lithium, cytotoxic drugs

Ionizing Radiation in Pregnancy
- American College of Radiology: no single diagnostic test results in a radiation dose that threatens the well-being of the embryo; however, cumulative doses may enter the harmful range.
  - Threshold for teratogenesis = 10 rads
  - Fetal vulnerability peaks at 8 to 15 weeks’ gestation
Emergent and Eminent Deliveries

- **Principles**
  - Always better to deliver newborn in delivery room
  - Better to transport a mom than an infant

- **Maternal transport to regional center**
  - **Indications**
    - Preterm labor
    - Premature rupture of membranes
  - **Conditions that must be met prior to transport**
    - Arrest dilatation with tocolytics
    - Fetal and maternal cardiovascular stability

- **Management (if obstetric facilities are unavailable)**
  - **ABCs, oxygen, IV fluid bolus**
  - **Tocolytics**
    - **Indications**
      - Gestational age 20 to 36 weeks
      - Regular contractions
      - Cervical dilation of 4 cm or less
    - **Contraindications**
      - Age <20 weeks
      - Unknown lung maturity
      - Uncorrected fetal distress or fetal death
      - Obstetric complication requiring delivery
      - Chorioamnionitis
  - **β-Adrenergic agents**
    - Terbutaline: 0.25 mg SQ
    - Magnesium sulfate: dose of 4 to 8 g IV followed by 1 to 3 g/hr
    - Calcium channel blocker

- **Preparation for emergency delivery**
  - **High risk**
    - Indigent mother
    - Multiple gestation
    - Prematurity
    - Meconium
  - **General**
    - Assess anyone arriving in the ED with active contractions with bimanual exam.
    - Examine for cervical dilation, effacement, presenting part, absence of cord.
    - Manage vaginal bleeding.
    - Suspicion of rupture of membranes mandates a STERILE speculum exam.
    - Vitals, ABCs, fetal monitoring
    - IV, CBC, type and screen
    - No pushing unless fully dilated
• Liberal use of episiotomy
• Be prepared for neonatal resuscitation.
• Fetal bradycardia: oxygen, fluid bolus, place mother in left lateral decubitus position

- Eminent deliveries
  - “Crowning” = ED delivery is imminent and indicated
  - Consultation: OB and pediatrics
  - Maternal preparation: IV and fetal heart monitoring
  - Have neonatal resuscitation equipment ready
    - Infant warmer
    - IV, umbilical, and IO access equipment
    - Neonatal airway
    - Resuscitation medications

- “Routine” delivery of newborn
  - Vaginal exam
    - Effacement: process of cervical thinning
    - Dilation: estimation of the diameter of cervical os (10 cm is full dilation)
    - Station: level of presenting part in relation to the ischial spines
      - Above spines = -1, -2, -3 cm, and floating
      - Below spines = +1, +2, +3 cm
  - Basic principles
    - Controlled delivery
    - Stretch and support perineum with contraction
    - Episiotomy: if there is no time for anesthetic, make the incision during maximal perineal stretch, when it will cause the mother the least pain
      - Cut midline, protecting the baby’s head with gloved finger
    - Control the head’s emergence.
    - Face will be down and the head will turn lateral
    - When face is exposed:
      - Check neck for nuchal umbilical cord (25% of deliveries)
      - If nuchal cord present and loose, lift and slip it over the baby’s head.
      - If unsuccessful, double clamp and cut.
    - Delivery of anterior shoulder: downward traction
    - Posterior shoulder: upward traction
    - The body will emerge quickly, so it is important to hold the neonate about the neck and support the body.
    - When the body is delivered, double clamp and cut the cord.
    - Examine for the presence of three vessels (two arteries, one vein).
    - A two-vessel cord may be indicative of other congenital anomalies.
    - Stimulate the neonate by drying, warming, and supplying supplemental oxygen if needed.
Assessment of the Newborn

Apgar Score: assessment of newborn at 1 and 5 minutes

<table>
<thead>
<tr>
<th>Sign</th>
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<th>1</th>
<th>2</th>
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<tbody>
<tr>
<td>Heart rate</td>
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<td>&lt;100</td>
<td>&gt;100</td>
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<tr>
<td>Respiratory effort</td>
<td>None</td>
<td>Slow, irregular</td>
<td>Cry</td>
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<tr>
<td>Muscle tone</td>
<td>Limp</td>
<td>Flexion</td>
<td>Active</td>
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<tr>
<td>Reflex irritability</td>
<td>None</td>
<td>Grimace</td>
<td>Cough or sneeze</td>
</tr>
<tr>
<td>Color</td>
<td>Blue or pale</td>
<td>Blue extremities</td>
<td>Completely pink</td>
</tr>
</tbody>
</table>

Intrapartum Emergencies

- Arrest of normal labor
  - Etiology
    - Uterine malfunction
    - Cephalopelvic disproportion (CPD): progressive cervical dilation with no change in station
    - Fetal malposition
  - Management
    - These circumstances require emergent obstetric intervention; operative intervention is often needed.

- Shoulder dystocia
  - Definition
    - Impaction of anterior shoulder behind the pubic symphysis (after delivery of the head in vertex presentations)
  - Incidence
    - 0.15% deliveries; 1.7% of infants >4,000 grams
  - Risks
    - Fetal death, brachial plexus injury
  - Clinical
    - Dystocia should be expected when the head retracts after presentation ("turtle sign")
  - Maneuvers
    - Maternal positioning: perineum at the end of table, maximal flexion of the hips
    - Episiotomy
    - Suprapubic pressure (not fundal pressure)
    - Rotation of the posterior shoulder: apply pressure to posterior scapula upward and anteriorly
    - Wood's screw: rotation of shoulders until posterior shoulder passes beneath the symphysis and is delivered as an anterior shoulder
    - Intentional fracture of the anterior clavicle or humerus
    - Zavenelli maneuver: if general anesthesia and C-section delivery are available—flex fetal head and push into vagina as C-section is performed.

- Prolapsed umbilical cord
  - Incidence with various presenting parts
    - Cephalic: 0.4%
    - Frank breech: 0.5%
    - Footling breech: 15% to 18%
    - Complete breech: 4% to 6%
Etiology
- Usually occurs at the same time as rupture of the membranes
- Occurs when presenting part does not completely fill the lower uterine segment

Management
- If obstetric consultation is unavailable, consider tocolytics to maintain placental blood flow.
- Exert manual pressure to lift and maintain the presenting part away from the prolapsed cord.
- Positioning: deep Trendelenburg or knee to chest

Abnormal presentations

Determination of presenting part
- Examiner’s fingers travel from symphysis to maternal sacrum, determining course of sagittal suture
  - Vertex
    - Anterior fontanelle: diamond shaped
    - Posterior fontanelle: triangular
  - Face: chin palpated
  - Breech: sacrum
  - Incomplete breech: foot or knee

Face presentation
- Hyperextension of the head, occiput in contact with the fetal back, chin is presenting
- Incidence: 0.17%
- Delivery: impossible vaginally unless the chin rotates anteriorly

Breech presentation
- Classification
  - Frank (65%): hips flexed, knees extended
  - Complete (10%): hips flexed, knees flexed
  - Incomplete (25%): one or both hips extended, foot presenting
- Incidence: 3% to 4% (single deliveries)
- Complications
  - Increased perinatal morbidity and mortality
  - Low birth weight
  - Growth retardation
  - Prolapsed cord
  - Placenta previa
  - Anomalies
  - Increased operative interventions (C-section)
  - Perinatal mortality three to four times that of a normal delivery

Delivery
- Successively larger and less compressible parts need to be delivered.
- If cephalopelvic disproportion is not diagnosed, the prognosis is grave.
- Intracranial hemorrhage is the most frequent cause of death in breech delivery.
- Three types
  - Spontaneous breech: spontaneous delivery
  - Partial breech extraction: spontaneous delivery to the umbilicus
  - Total breech extraction: indicated only if there is a definitive diagnosis of fetal distress and cesarean delivery cannot be accomplished promptly
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• Technique: total breech extraction of incomplete breech
  • Grab the feet and apply gentle traction until the feet are pulled through the vulva.
  • Wide episiotomy
  • Gently apply downward traction as higher portions of the legs and thighs are grasped.
  • After the buttocks emerge, the fetal back usually rotates anteriorly.
  • Apply gentle traction, with fingers on the sacrum.
  • Induce lateral rotation as the scapulas are delivered, leading to delivery of the shoulder.
  • Head delivery

• Technique: delivery of frank breech
  • Episiotomy
  • Allow a frank breech to deliver as far as possible.
  • Apply traction on both hips.

Postpartum Management
• Placenta delivery
  • General: allow spontaneous separation
  • Signs: gush of blood and lengthening of cord (within 5 minutes after delivery)
  • Brandt-Andrews maneuver: apply gentle traction to the cord as well as pressure between the uterine fundus and symphysis
• Management of uterus
  • Use firm massage to express clots and stimulate contraction.
  • Oxytocin
    • Administer only after delivery of placenta
    • Oxytocin dose: 20 units/l liter saline run at 10 cc/min
  • Methylergonovine dose: 0.2 mg IM (contraindicated in patients with hypertension and preeclampsia)
• Uterine inversion
  • Incidence: 1 in 4,000 to 5,000 deliveries
  • Etiology: usually traction on the cord of an adherent placenta
  • Diagnosis: visualization of fundal wall near the cervical os; no palpable uterine fundus
  • Presentation: hemorrhage and shock
  • Management
    • Hemodynamic support
    • Immediate repositioning of uterus, gradual manual replacement
    • General anesthesia and laparotomy may be necessary for repositioning.
• Postpartum hemorrhage
  • Etiology: uterine atony is most common cause
  • Definition: >500 cc of maternal blood loss during the first 24 hours
  • Other causes
    • Lacerations (vagina, cervix, perineum)
    • Retained placental products
    • Coagulation disorders
    • Uterine rupture
    • Uterine inversion
Management
- ABCs, crystalloids, blood products
- Repair lacerations
- Pitocin, 20 mg in 1 liter of normal saline administered at 10 cc/min
- Methylergonovine, 0.2 mg IM or IV (avoid if hypertensive)

Amniotic fluid embolism
- General
  - Amniotic fluid in the maternal circulation
  - High mortality rate
  - Most common during labor
  - Rare (but leading cause of death in induced abortions and miscarriages)
  - May occur after amniocentesis or abruptio placenta following trauma
- Pathophysiology
  - Fluid is released into the maternal circulation during uterine contraction or manipulation
  - Profound immunologic response is triggered
  - Two-phase reaction
    - 0 to 30 minutes: cardiopulmonary collapse due to vasospasm and plugging
    - >30 minutes: left ventricular dysfunction, acute respiratory distress syndrome (ARDS), coagulopathy
- Complications
  - Maternal mortality 50% in first hour, 80% at 4 to 5 hours
  - Fetal death rate is high
- Differential diagnosis
  - Pulmonary embolism
  - Pyelonephritis with sepsis
  - ARDS
- Management
  - Admit to the ICU
  - ABCs, fluid resuscitation
  - Ventilatory support
  - Ionotropes
  - Manage coagulopathy

Postpartum Infection
- Endometritis
  - General
    - Any woman presenting to the ED after delivery with increasing abdominal pain and/or fever should be evaluated for endometritis.
  - Incidence
    - 3% with vaginal delivery
    - 15% to 30% with C-section
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- Pathophysiology
  - Ascending vaginal flora
  - Adherence of bacteria to decidua
  - May progress to myometrium and parametrium
  - Polymicrobial: gram-negative aerobes; group A, B, D streptococci, *Staphylococcus*, anaerobes, *Chlamydia*, *Mycoplasma hominis*, *Ureaplasma urealyticum*

- Timing
  - Infection within 48 hours: group A and B streptococci, *Staphylococcus*, *Clostridium*
  - If infection occurs later, consider atypical organisms.

- Presentation
  - History: fever, chills, malaise, lower abdominal pain, dysuria, foul-smelling lochia
  - Group A streptococci may be odorless.
  - Exam: purulent vaginal/cervical discharge, cervical motion tenderness, uterine tenderness
  - Ultrasound may show retained products of conception.

- Management
  - Lab: CBC, urine and blood cultures
  - Admit
  - Administer broad-spectrum antibiotics, with acceptable combinations being clindamycin/gentamicin, penicillin/β-lactamase inhibitor, imipenem/cilastatin

- Mastitis
  - General
    - Cellulitis of periglandular breast tissue
    - Most common in lactating women
    - Often occurs during the second or third postpartum week
  - Pathophysiology
    - *Staphylococcus*, *Streptococcus*, *H. influenzae*
  - Presentation
    - Fever, chills, myalgia, breast pain, breast erythema/induration
  - Management
    - Outpatient treatment is appropriate if infection is not extensive and patient is not septic appearing.
    - Cultures are unnecessary.
    - The mother should be instructed to continue to nurse on the side of infection (it will not harm infant, and it helps clear the infection).
    - β-Lactamase-resistant penicillin or first-generation cephalosporin
    - Abscess formation (rare) requires incision and drainage.

10.5 SEXUAL ASSAULT

Management
- Treat as a trauma patient with immediate assessment for serious, traumatic injury.
- After serious injuries are addressed, a detailed examination with specific protocols must be followed to preserve evidence and is best done at a specialized center if available.
- Obtain baseline hCG, HIV, syphilis, and GC/Chlamydia cultures.
STD Prophylaxis
- Ceftriaxone, 125 mg IM x 1 dose, or azithromycin, 2 g PO x 1 dose, for gonorrhea prophylaxis
- Azithromycin, 1 g PO x 1 dose; doxycycline, 100 mg PO BID x 7 days; or erythromycin, 500 mg PO QID x 7 days for Chlamydia prophylaxis
- Metronidazole, 2 g PO x 1 dose, for Trichomonas prophylaxis
- Hepatitis B vaccine if unvaccinated (hepatitis B immunoglobulin is needed only if the offender is known to have acute hepatitis B and the victim is not vaccinated)
- HIV prophylaxis is not generally recommended.

Pregnancy Prophylaxis
- Mestranol/norethindrone, 2 tablets, repeat in 12 hours
- Initiate up to 72 hours after assault
- 98% to 99% effective if initiated in a timely fashion
CHAPTER 11
Hematologic/Oncologic and Immune System Disorders

Steven B. Kailes, MD, MPH, Timothy Costello, MD, and Usamah Mossallam, MD

11.1 BLOOD COMPONENT THERAPY

Transfusion Complications

- **Febrile Reaction**
  - Will see at least a 1°C rise (1.6°F) in temperature within 1 hour after initiation of transfusion
  - Cause: antileukocyte/antiplatelet antibodies to donor cells
  - Treatment in first-time transfusion (12.5% chance of recurrence in future transfusions)
    - Work up as possible hemolytic transfusion reaction.
    - Stop transfusion.
    - Give steroids and antipyretics.
    - If recurrent, condition is self-limited and may be treated with analgesics/antipyretics and antihistamines; washed or frozen red blood cells (RBCs) must be used in the future.

- **Hemolytic Reaction**
  - Acute intravascular hemolytic transfusion reaction
    - Risk is 1 in 300,000 to 700,000 units secondary to ABO incompatibility
    - Symptoms: immediate onset of fever, chills, nausea/vomiting, myalgias, headache, and burning at infusion site
    - May have chest pain and restriction, severe joint and lower back pain, disseminated intravascular coagulation, and possibly shock
    - Treatment: stop transfusion immediately
      - Replace tubing
      - Expeditious administration of isotonic IV therapy
      - Diuretics (e.g., furosemide) to maintain urine output of 1 to 2 ml/kg/hr
      - Corticosteroids
  - Delayed extravascular transfusion reaction
    - Symptoms: fever, anemia, and jaundice days to weeks after transfusion
    - Cause: non-ABO erythrocyte antigen response
    - Treatment: No specific therapy is typically required because the hemolysis is rarely significant, owing to its extravascular nature; however, additional transfusions with antigen-negative units may be required, depending on the severity of the resultant anemia.
- Monitoring parameters: The following lab tests should be ordered initially and then followed on an outpatient basis: haptoglobin, lactate dehydrogenase, bilirubin, direct anti-globulin test, and antibody screen.
- Disposition depends on the patient’s clinical status. Patients are usually asymptomatic.

- IgA-Mediated Reaction
  - Anaphylactic response to IgA (1:20,000 transfusions)
    - Symptoms: afebrile, hypotensive, bronchospasm, and GI complaints
    - Cause: genetic IgA deficiency in host, leading to anti-IgA antibody (either IgE or rarely IgG) reaction to donor IgA
    - Treatment: antihistamines, epinephrine, and corticosteroids, along with possible ventilatory and circulatory support
    - Ensure future transfusions with washed RBCs and plasma components from IgA-deficient source.
  - Urticarial response to IgA
    - Symptoms: urticarial rash during transfusion without other signs or symptoms of anaphylaxis
    - Cause: allergic response to donor plasma proteins (as in anaphylactic response, described above)
    - Treatment: antihistamines and corticosteroids; continue transfusion at a slower rate
    - Ensure future transfusions with washed RBCs and plasma components from IgA-deficient source.

- Sepsis Secondary to Bacterial Contamination of Transfused Blood Components
  - Rare occurrence (0.1% of all transfusions)
  - Second leading cause of transfusion-related mortality
  - Symptoms may mimic those of acute intravascular transfusion reaction (typically developing within 1 hour after initiation of transfusion)
  - Treatment: discontinue transfusion, start broad-spectrum antibiotics, and initiate additional supportive care measures

- Transfusion-Related Acute Lung Injury (TRALI)
  - Caused by donor white blood cell antibodies reacting with host white blood cells
  - Third leading cause of transfusion-related mortality (approximately 13% of all deaths)
  - Clinically identical to adult respiratory distress syndrome; typically presents within 6 hours after transfusion
  - Treatment: stop transfusion and provide respiratory support as needed

- Disease Transmission: Screening and Risks
  - According to the CDC in October 2006 – each unit of transfused blood is thoroughly screened for the following:
    - HIV
    - HTLV-1
    - Hepatitis B and C
    - *Treponema pallidum* (syphilis)
  - Risks of disease transmission per unit of blood transfused
    - HIV – 1 in 1.4 to 2.4 million
      - Expected to continue to decrease significantly in the future as a result of nucleic acid testing (NAT)
    - Hepatitis B – 1 in 58,000 to 149,000
    - Hepatitis C – 1 in 872,000 to 1.7 million (continues to decrease with NAT)
    - HTLV-1 – 1 in 641,000 transfused units
    - Unquantified risk of transmission of other viral and prion diseases such as Creutzfeldt-Jakob disease, cytomegalovirus, Lyme disease, Epstein-Barr virus, and herpes simplex viruses
• Bacterial infection/sepsis – 1 in 1 million units infected with *Yersinia* or *Pseudomonas* (organisms able to multiply in low-temperature storage conditions)

**Massive Transfusions**
- Replacement of volume equivalent to patient’s normal blood volume (typically 75 ml/kg) within a 24-hour period (or >10 units over a few hours)
- Most Common Complications
  - Coagulation deficits: due to dilutional effects, resulting in platelet dysfunction, clotting factor deficiencies (factors V and VIII), and DIC
    - Treatment: platelet concentrate, FFP, and cryoprecipitate
  - Metabolic alkalosis and hypocalcemia from citrate toxicity
    - High levels of citrate will be infused during a massive transfusion because blood is anticoagulated with both sodium citrate and citric acid.
    - After transfusion, as the citrate is metabolized, bicarbonate is produced (23 mEq of bicarbonate per unit of blood), which can lead to clinically significant metabolic alkalosis with resultant hypokalemia (especially with a rapid transfusion in a patient with underlying renal disease).
    - Hypocalcemia results as high levels of citrate bind to ionized calcium, causing the free calcium concentration to plummet.
      - Clinical signs: circumoral tingling, tremor, QT prolongation, dysrhythmias, tetany, and hypotension
      - Replace with calcium gluconate for abnormal ionized calcium or electrocardiographic changes.
  - Hypothermia: increases risk for ventricular dysrhythmias and decreased cardiac output
    - Generally seen with rapid infusion of 3 or more units of cold blood
    - Prevent by use of blood warmer (37°C)/warmed saline
  - Hypervolemia
    - Rapid volume expansion resulting in overload/CHF (especially in patients with diminished cardiovascular reserve)
    - Treatment includes slowing the infusion rate and administering diuretics.

**Component Therapy**
- Whole Blood
  - Rarely available, owing to instability (clotting factors disintegrate after 72 hours) and high levels of donor antigens
  - Needed by less than 10% of transfusion recipients
  - Replaced by component therapy
- Packed Red Blood Cells (PRBCs)
  - Centrifuged whole blood with >80% of plasma removed
  - Indicated for shock not responsive to intravenous fluids, known blood loss >750 mL, hemoglobin <6 g/dL, or symptomatic anemia not amenable to nutritional correction
  - 1 unit of PRBCs – boosts hemoglobin by 1 gm/dL or hematocrit by 3% in adults
  - Each mL/kg of PRBCs transfused in a child typically raises hematocrit by 1%
- Frozen RBCs
  - The process of freezing blood destroys all components except RBCs and a few leukocytes.
  - Reserved for transplant patients, because of its expense
- Washed RBCs
  - Indicated for patients with hypersensitivity reaction to plasma (usually IgA deficient)
• Leukocyte-poor blood (>70% of leukocytes removed)
  o Indicated for
    ▪ Transplant patients
    ▪ Patients with previous transfusion reactions

• Platelets
  o Limited shelf life of 5 days
  o ABO and Rh compatibility checks required
  o Each “six pack of platelets” raises count by 30,000 (5,000 per unit)
  o No definitive guidelines exist for the decision to transfuse platelets. However, platelet transfusion is generally indicated for the patients described as follows:
    ▪ Those with spontaneous bleeding (platelet count <10,000–20,000)
    ▪ Those with platelet count <50,000 with oozing or planned procedure
    ▪ Trauma patients with platelet counts ≤75,000 (some use up to 100,000)
  o Standard administration for adults – 6 to 10 units (approximately 1 pack [unit] of platelets per 10 kg); children – 1 unit/10 kg

• Fresh Frozen Plasma
  o Fresh plasma – plasma used within 6 hours after collection
  o Frozen plasma – plasma kept at −18°C
  o Contains 1 unit of each coagulation factor per milliliter except platelets
  o No cellular components; lasts up to 1 year
  o Indicated for the following:
    ▪ Specific coagulopathies caused by factor deficiencies if the particular individual factor is not available
    ▪ Reversal of coagulopathy from warfarin
    ▪ With massive transfusions (dilutional factor deficiency)
  o Typical dose: 10 to 15 mL/kg (three to four 250-mL bags for a 70-kg person)
  o Warfarin reversal – typically requires only half the dose (5–8 mL/kg)

• Cryoprecipitate
  o Cold-precipitable protein fraction from thawed FFP
  o Contains factor VIIIc, von Willebrand factor, factor IX, fibrinogen, factor XIII, and fibronectin
  o Typical dose: 2 to 4 bags/10 kg (10–20 bags for adults)
  o Indications
    ▪ Used to correct a deficiency of coagulation factor VIII (hemophilia A and von Willebrand syndrome), factor XIII, or fibrinogen
    ▪ Factor VIII concentrate is preferred in hemophilia A, because of the large volume of cryoprecipitate needed and increased risk of infection transmission.

• Albumin
  o Available in 5% and 25% solutions in saline
  o Hyperosmolar solution with oncotic effect five times the same volume of plasma
  o Indications
    ▪ Maintain oncotic pressure to provide for plasma volume expansion and maintenance of cardiac output in shock.
    ▪ In 2004, a randomized controlled study of intensive care unit patients (published in *NEJM*) demonstrated no difference in clinical outcomes or 28-day mortality when comparing use of albumin with saline solutions for fluid resuscitation.
• Principal replacement fluid for therapeutic plasma exchange and/or plasmapheresis

• Wait time for blood products
  o Type-specific (ABO and Rh): 5 to 10 minutes
  o Type and screen (screens only against common antibodies): 30 minutes
  o Type and crossmatch: 45 minutes

• Universal donor: Type O
  o Absence of A and B antigens prevents agglutination and hemolysis from anti-A or B antibodies
  o Type-O–negative blood products (prevent possible Rh immunization) are reserved for the following:
    ▪ Females of child-bearing age
    ▪ Pregnant females
  o Type-O–positive blood products are recommended for most other patient groups
    ▪ Except individuals who have received previous or are receiving multiple transfusions

11.2 RED BLOOD CELL DISORDERS

Anemia
• Definition: reduced concentration of RBCs
  o Emergent
    ▪ Most commonly caused by blood loss
    ▪ Tachycardic, decreased BP, thirsty, altered mental status, and decreased urine output
    ▪ Underlying health status is an important variable for tolerance of or compensation for the condition.
  o Non-emergent
    ▪ Generally can be managed on an outpatient basis
    ▪ Fatigue, irritability, headaches, dyspnea, decreased exercise tolerance, decreased libido
  o Ancillary evaluation
    ▪ CBC with indices and peripheral smear
    ▪ Coagulation studies
    ▪ BUN, creatinine, electrolytes may reveal related underlying disease
  o Differential diagnosis
    ▪ Decreased RBC production – thalassemias, iron deficiency, chronic disease, vitamin B12 or folate deficiency, primary bone marrow involvement ( aplastic)
    ▪ Increased RBC destruction, i.e., hemolysis
    ▪ Blood loss, i.e., hemorrhage
    ▪ Functional, i.e., impaired hemoglobin function, as seen in carbon monoxide poisoning or methemoglobinemia
  o Indices used to help differentiate types of anemia (generally of only limited usefulness in the ED)
    ▪ Mean corpuscular volume (MCV) – indicates RBC size
    ▪ Mean corpuscular hemoglobin (MCH) – incorporates RBC size and hemoglobin (Hb) concentration (least helpful)
    ▪ Mean corpuscular hemoglobin concentration (MCHC) index – a measure of the concentration of Hb
    ▪ Red blood cell distribution width (RDW) – a measure of the homogeneity of the RBCs, used to differentiate iron-deficiency from thalassemia
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Microcytic Anemias

- Additional Labs: reticulocyte count, serum iron, iron-binding capacity, ferritin
- Iron Deficiency
  - Most common anemia in women of childbearing age (caused by menses)
  - Microcytosis occurs only after iron stores are depleted.
  - Search for occult blood loss
  - Therapy is oral iron replacement, with improvement in as little as 24 hours, but can be started by the patient's primary care manager (PCM).
  - Can cause nausea, vomiting, constipation, and blackened stools

- Thalassemias
  - Genetic autosomal defect affecting synthesis of globin chains (α, β, δ, γ); many variations of thalassemia can occur
    - Normal Hb (HbA) has two α and two β chains (α₂β₂)
    - Fetal Hb (HbF) is α₂γ₂
  - Decreased globin synthesis results in ineffective erythropoiesis.
    - Normally, erythropoiesis fails to produce RBCs 10% to 20% of the time, resulting in intramarrow hemolysis; this failure rate can be two to three times higher in thalassemia.
  - Thalassemia major (homozygous β chains) – mostly in Mediterranean populations
    - Severe anemia, hepatosplenomegaly, jaundice, and premature death
    - Transfusion dependent; people die of iron deposition in tissues
    - Treatment: supportive and iron-chelation therapy
  - Thalassemia minor (heterozygous β chains)
    - Mild, microcytic hypochromic anemia with target cells on peripheral smear
    - Usually no treatment is necessary.
  - α-Thalassemia – more commonly seen in Asians and African-Americans
    - Spectrum from asymptomatic carrier to prenatal death
    - Microcytic hypochromic anemia with target cells and basophilic stippling; diagnosis with Hb electrophoresis
    - Treatment: depends on severity, but includes blood transfusions and iron-chelation therapy

- Sideroblastic Anemia
  - Defect in porphyrin synthesis, impairing Hb production, resulting in increased serum iron and ferritin and saturated transferrin
  - May be an idiopathic, sex-linked hereditary form (rare) or can be secondarily caused by toxins (e.g., lead, alcohol abuse, INH), infection, or other diseases
  - Mild to moderate anemia results, often seen in elderly patients
  - May see pallor and splenomegaly, along with vitamin B6 (pyridoxine) deficiency
  - Treatment: trial of pyridoxine, transfusion, and iron-chelation may be necessary
  - Considered a pre-leukemic state; 20% develop acute myelogenous leukemia

- Anemia of Chronic Disease
  - Very common; can be normocytic, normochromic
  - Not responsive to iron therapy
  - Search for occult blood loss; however, often caused by cancer, inflammation, infection, or uremia
  - Treatment is usually unnecessary; patients rarely have hematocrit <25% to 30%
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Macrocytic Anemia

- Megaloblastic Anemia
  - Altered DNA synthesis caused by vitamin B12 and/or folate deficiency, with resultant pancytopenia
    - Anemia may not be seen until body stores are used up, which may take months (folate) to years (vitamin B12).
    - May be caused by antimetabolites (chemotherapy) or rare inherited disorders
    - Vitamin B12 deficiency can develop neurologic features (weakness, lower extremity spasticity and altered reflexes, depression, irritability).
    - On peripheral smear, large oval RBCs and hypersegmented polymorphonuclear neutrophils may be seen.
    - Therapy is specific to cause.

- Other Macrocytic Anemias
  - Seen in liver disease (alcoholism), hypothyroidism

Normochromic and Normocytic Anemias

- Aplastic Anemia
  - Decreased production resulting from marrow stem cell failure; normal indices; low reticulocyte count
  - 50% of cases are caused by drugs or chemicals (e.g., chloramphenicol)
  - Also seen with radiation, infection (including HIV), renal failure, marrow infiltration (e.g., leukemia, tumor), myelodysplastic syndromes, idiopathic conditions
  - Treatment: remove cause (if possible) or treat underlying disorder; may need bone marrow transplant

Hemolytic Anemias

- Intravascular Hemolysis
  - Generally an acute process: free Hb is transported to the liver for conversion to bilirubin, conjugation, and excretion
    - When this system is overwhelmed, free Hb is seen in the blood (pink serum) and unconjugated bilirubin is increased.
  - May be mild (mechanical hemolysis); if severe (see transfusion reaction), prostration, fever, jaundice, abdominal and back pain, acute renal failure, and oliguria are present
    - Schizocytes on smear; decreased haptoglobin due to degradation after binding with Hb (also low in liver disease), increased LDH, positive Coombs test (positive direct Coombs means there are antibodies on the RBC surface; positive indirect test means there are serum antibodies)
  - Paroxysmal nocturnal hemoglobinemia – rare stem cell defect; blood cells are sensitive to complement. Transfusion can be life threatening (RBC lysis caused by donor complement) unless washed packed RBCs are used.

- Extravascular Hemolysis
  - Occurs in spleen or in bone marrow (as with increased ineffective erythropoiesis seen in thalassemia)
  - Primary hyperactivity of the reticuloendothelial system's removal of older or damaged RBCs
    - Antibody mediated
    - RBC membrane defects may also increase the activity of the reticuloendothelial system
  - Usually mild to moderate, with mild jaundice, splenomegaly; variable presentation based on severity
    - Spherocytes are seen on smear, decreased haptoglobin, increased LDH, increased bilirubin (both conjugated and unconjugated)
  - Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency
    - Most common human enzyme defect (15% of African-American males have at least a mild form; 10% of the world population, especially Greeks, Sicilians, Arabs)
G6PD is an important enzyme in the glycolytic pathway of RBC energy production. It is involved in the production of glutathione, which is necessary to prevent oxidant injury to hemoglobin (which clumps in the cells as Heinz bodies) and cell membrane destruction.

Acute (within 24–48 hours) hemolytic crises incited by infection, oxidant drugs (e.g., sulfa, phenazopyridine, antimalarials, salicylates), metabolic acidosis (like DKA), and ingestion of fava beans may be either intravascular or extravascular and is dose related.

Treatment: volume and RBC support as necessary; prevention is essential.

Other Hemoglobinopathies

Sickle Cell Disease and Trait

Autosomal dominant abnormal allele for Hb β-chains, causing interlocking Hb chains when deoxygenated, resulting in sickled cells (less deformable), sludging, and increased viscosity.

- Sickle cell anemia → homozygous HbSS; most severe if >75% of hemoglobin is HbS
- Sickle cell trait – heterozygous HbAS
  - Usually asymptomatic; genetic counseling is useful

Diagnosis and laboratory findings

- Usually patient knows and reports diagnosis
- Blood smear shows irreversibly sickled RBCs (HbSS or double heterozygotes but NOT HbSA [trait])
- Sickle prep is positive in all forms
- Anemia and elevated reticulocyte count with chronic compensated hemolysis (extravascular)
- Increased platelets and white blood cells (even without infection) due to bone marrow activation

Increased risk of infection with encapsulated organisms (Pneumococcus, H. influenzae) and Salmonella osteomyelitis

- Secondary to functional asplenia and other factors
- Get a white blood cell count and blood cultures (and/or other appropriate cultures) in febrile patients.

Types of sickle cell crises

Vaso-occlusive crisis (painful crisis): crescendo pain over hours to days in joints, chest, back, abdomen, extremities; priapism may also occur
- Precipitating factors: infection, dehydration, cold exposure, trauma; labs are not helpful in making diagnosis
- Pain from tissue ischemia secondary to effect of sickled cells
  - May mimic acute abdomen, PE, and other painful problems; helpful to limit workup if presentation is similar to previous pain crises

Hemolytic crisis: exacerbated hemolysis with precipitous fall in hematocrit and increased jaundice

Aplastic crisis
- Bone marrow failure with marked fall in hematocrit and reticulocyte count
- Usually precipitated by infection (parvovirus) or folate deficiency
- Suspect if Hb level falls 2 g/dL or more from baseline, with low reticulocyte count (<2%)

Sequestration crisis
- Almost exclusively in children
- Pancytopenia with sudden painful enlargement of liver and spleen due to intrasplenic sickling and obstruction
- May be in shock
Acute chest syndrome
- A leading cause of death (25% of premature deaths in sickle cell disease)
- Fever, cough, chest pain, dyspnea
- Pneumonia, thrombosis (pulmonary infarct), or thromboembolism (fat emboli from bone infarct), acute pulmonary hypertension

- Treatment: supportive care, including the following:
  - Hydration such as 5% dextrose in half-normal saline (D5 ½ NS) at 150 to 200 mL/hr, but be aware of the potential to develop CHF
  - Oxygen, 2 to 4 mL/min via nasal cannula
  - Analgesics, including NSAIDs and narcotics, are helpful. Patients discharged home may need a prescription for 4 to 6 days worth of analgesia. Your institution may or may not have standard protocols for treating sickle cell disease pain crises.
  - Anti-sickling agents such as hydroxyurea stimulate HbF production but are NOT useful for acute conditions.
  - Consider transfusion (if hematocrit <18%); exchange transfusion therapy may be done under the care of a hematologist.
  - Antibiotics as necessary for potential infections

Polycythemia
- Primary disorder of stem cells; elevated absolute red cell mass (hemoglobin >18 g/dl, hematocrit >56%) due to uncontrolled red cell production
  - Can also see increased white cell (myeloid) and platelet (megakaryocytic) production
  - Secondary causes, due to increased erythropoietin levels and/or tissue hypoxia, will be associated with normal WBC and platelet counts, as these cells lines are not affected by erythropoietin. Causes include the following:
    - Tumors of uterine, CNS, renal, or hepatic origin
    - Hypoxia from CHF, high altitude, carboxyhemoglobin (smokers), and pulmonary disease
    - “Apparent polycythemia” occurs in patients who are dehydrated, as a result of decreased plasma volume
  - Disease is usually self limiting and “burns out” over approximately 20 years.
    - 10% develop poorly responsive acute leukemia
- Symptoms are often insidious: related to hyperviscosity impairing microcirculation tissue flow
  - Related to poor oxygen delivery
    - Headache, dizziness, vertigo, tinnitus, visual disturbances, angina, intermittent claudication
  - Bleeding complications due to platelet dysfunction
    - Epistaxis, gum bleeding, ecchymoses, GI bleeding
  - Thrombosis
    - CVA, MI, DVT, hepatic vein thrombosis (acute Budd-Chiari syndrome)
  - Pruritis due to increased histamine release from increased granulocytes, exacerbated by warm bath or shower.
- Physical Findings
  - Plethora (characteristic skin color or ruddy complexion, on face, palms, and nail beds as well as mucosa and conjunctiva)
  - Venous engorgement
  - Splenomegaly is present in 75% at time of diagnosis; hepatomegaly in about 30%
  - CHF findings may be present.
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- Lab studies
  - RBCs are usually normochromic, normocytic.
  - WBC count is elevated (>12,000/µl) in about 60% of patients (mainly neutrophils with a few immature cells; mild basophilia in 60% of patients)
  - Platelet count is 400,000 to 800,000/µl in about 50%
  - Release of potassium into serum from increased platelets during in vitro coagulation may cause pseudohyperkalemia while plasma potassium is normal.
  - PT/aPTT - may be artifactually prolonged because of erythrocytosis (quantity of plasma collected will be low in relation to anticoagulant in tube)

- Treatment
  - Slow phlebotomy as necessary to keep Hct <55%; replace blood with normal saline
    - If hematocrit >70%, phlebotomize twice a week to reduce Hct to ~40%.
    - If the patient has severe plethora with altered mentation or vascular compromise, phlebotomize 500 ml whole blood daily.
  - Low-dose aspirin to prevent thromboses

11.3 WHITE BLOOD CELL (WBC) DISORDERS

Leukemia
- Most patients with leukemia present with fever, chills, and other flu-like symptoms. Variable presentations include the following:
  - Weakness and fatigue
  - Frequent infections
  - Loss of appetite and/or weight
  - Swollen or tender lymph nodes, liver, or spleen
  - Easy bleeding or bruising, petechiae (see Image #36), swollen or bleeding gums
  - Sweating, especially at night
  - Bone or joint pain

- Acute Lymphocytic Leukemia
  - Malignancy of greatest incidence in children <15 years
  - Leukostasis may be present.

- Chronic Lymphocytic Leukemia
  - For patients over 50, most common leukemia; a B-cell disorder
  - Hematopoesis is common, along with anemia, neutropenia, and thrombocytopenia.

- Chronic Myeloid Leukemia
  - Least common leukemia; a stem cell disorder from the Philadelphia chromosome
  - WBC counts >50,000, but with anemia; leukostasis may cause end-organ injuries
  - Blast crisis
    - Evolving disease with an abrupt presentation of blast cell counts >50,000 with new fevers, new anemia, and/or splenomegaly disproportionate to height of the WBC, increasing immaturity of the myeloid series in the bone marrow
• Leukemoid Reaction
  o Systemic reaction that resembles leukemia but that is actually caused by other conditions (e.g., infection or cancer)

• Leukopenia
  o Abnormally low white cell count (<4,000) usually resulting from reduced neutrophil count (neutropenia, granulocytopenia)
  o Neutropenia is the most clinically significant leukopenia; the absolute neutrophil count (ANC) is calculated by the following formula:
    \[
    \text{Absolute Neutrophil Count} = (\% \text{ bands} + \% \text{ polysegmented}) \times \text{WBC}
    \]
    - Mild neutropenia (ANC 1000–1500 cells/mm³)
    - Moderate neutropenia (ANC 500–1000 cells/mm³)
    - Severe neutropenia (ANC <500 cells/mm³) can cause death in few days, owing to susceptibility to overwhelming infection.
  o Agranulocytosis
    - Neutropenia so severe that the body can no longer effectively confer protection
    - The patient becomes predisposed to infection (usually caused by drugs, infection, or an autoimmune process).
  o Clinical manifestations of neutropenia
    - Infections: necrotizing lesions of gingiva, oral floor, buccal mucosa, pharynx (agranulocytic angina); also in vagina, skin, anus, GI
    - Deep undermined ulcers with gray-green-black necrotic membranes
    - Physical signs may be minimized secondary to lack of effective inflammatory response or purulence
  o Usually caused by decreased marrow production, increased cell destruction, or tissue pooling of circulating neutrophils
  o Treatment: for severe neutropenia, obtain hematology consultation early, as well as the following:
    - Remove inciting agent/problem if possible (e.g., medication)
    - Pan-culture the patient, institute basic isolation techniques, and begin antibiotic therapy (usually broad-spectrum antibiotics unless a definite source is identified)
    - Early admission
    - In consultation with a hematologist, production of neutrophils can be stimulated by recombinant human granulopoietic factors (GM-CSF, G-CSF)
  o Patients with mild to moderate neutropenia may be treated as outpatients if:
    - An identifiable, reversible source is found
    - No significant clinical findings are present
    - Follow-up is assured, preferably with the patient’s PCM or other specialist as indicated.

Multiple Myeloma
• Malignancy of Plasma Cells in the Bone Marrow
  o Clinical picture varies; characteristic features include bone destruction with bone pain, elevated levels of paraprotein in serum and Bence Jones proteins in the urine, anemia, elevated serum calcium, and impaired renal function
  o ~70% present with pain of varying intensity, often in the lower back or ribs; general malaise and vague complaints are often seen, but weight loss is not common
o Neutropenia and hypogammaglobulinemia increase the likelihood of infections (pneumococcal pneumonia is the “classic” infection).

o Hypercalcemia, present in 30% of patients at diagnosis, causes tiredness, thirst, and nausea; precipitation of calcium salts can result in deterioration of kidney function.

o Hyperviscosity, resulting from high myeloma protein levels, can cause problems such as ecchymoses, nose bleed, hazy vision, headache, gastrointestinal bleeding, sleepiness, and a variety of ischemic neurologic symptoms caused by reduced blood and oxygen supply.

• Lymphomas
  o Tumors of B or T cells
  o Two types: non-Hodgkin’s lymphoma (NHL) and Hodgkin’s disease (HD)
    ▪ NHL is the third most common childhood malignancy.
    ▪ NHL (>55,000 cases per year in the US) is more common than HD (~7,100 cases per year in the US)
    ▪ Diagnosis: symptoms are often not specific
      □ Swollen lymph nodes
        — HD often presents with a painless, enlarged lymph node (especially axillary, supraclavicular, or cervical)
        — NHL usually involves the mediastinal or abdominal lymph nodes (less commonly superficial nodes)
      □ Lack of energy, weight loss
      □ May have dyspnea or superior vena cava syndrome
      □ GI pain or symptoms
      □ Fever, night sweats, or unexplained itching
  o Initial evaluation of patient’s symptoms (e.g., chest film for dyspnea or CT scan for abdominal pain)
  o Diagnosis is often an unexpected finding.
  o In most cases, further workup and staging are done on an outpatient basis.
  o Treatment depends on severity of symptoms and associated complications.
  o Disposition may be determined after discussion with the patient’s PCM or consultants.

• Pancytopenia
  o Stem cell defect, hypoplastic anemia
  o Bone marrow failure: patient presents with low blood counts
    ▪ Neutropenia places patients at risk for serious infection.
    ▪ Bleeding and infections bring them to ED
  o History: weakness and fatigue from anemia can develop slowly
    ▪ Note exposure to toxins, drugs, environmental hazards, and recent viral infections, such as hepatitis
  o Physical
    ▪ In severe anemia: pallor and/or signs of congestive heart failure
    ▪ Bruising (ecchymoses or petechiae [see Image #36]) in skin, gum bleeding, and nosebleeds are frequently associated with thrombocytopenia.
    ▪ Fever, cellulitis, pneumonia, and sepsis can be complications of severe neutropenia.
  o Expect complications with WBC count <500/µl, platelets <20,000/µl, or anemia with reticulocyte count <1%
11.4 HEMOSTASIS DISORDERS

Overview

- Normal hemostasis is a complex physiologic response leading to arrest of abnormal blood flow.
- When a vessel is damaged, the subendothelial matrix is exposed, leading to platelet aggregation at the site.
- Platelets and fibrinogen form an occlusive plug, which serves as a matrix for reactions of the coagulation cascade.
- Extrinsic and intrinsic pathways activate factor X, which converts prothrombin to thrombin.
  - Tissue factor (at the injured vessel site) or thromboplastin activates the extrinsic pathway.
    - Responsible for initiating the clotting cascade
    - Measured by prothrombin time (PT)
      - Includes activity of factors V, VII, and X; fibrinogen; and prothrombin
  - Surface factors activate the intrinsic pathway
    - Responsible for sustaining the clotting cascade, as tissue factor exists in limited amounts and is rapidly inactivated
    - Measured by partial thromboplastin time (PTT)
      - Includes activity of all factors except VII and XIII
    - Thrombin cleaves fibrinogen to fibrin, forming an insoluble clot.
    - Fibrin gets cross-linked, and the plug is anchored by clot retraction.
- This system is a delicate balance. Problems occur when the normal regulatory factors are in excess or deficient or when other controls are unbalanced, i.e., the fibrinolytic system, proteins C and S, antithrombin III.

Coagulation Defects, Acquired

- Warfarin Overdose
  - Competitive inhibition of vitamin K for liver synthesis of factors II, VII, IX, and X
  - Therapeutic dose leads to an INR of 2.0 to 3.0 (PT 1.5 to 2.5 times normal) with normal or minimally prolonged PTT.
  - Anticoagulant effect is increased by drugs that displace protein-bound coumarin derivatives (e.g., phenylbutazone, clofibrate, indomethacin) or inhibit its breakdown (e.g., allopurinol, nortriptyline, chloramphenicol).
  - Treatment
    - Fresh frozen plasma for life-threatening bleeding
      - Effect is immediate and short-lived; start with 4 to 6 units
    - Vitamin K1 (phytonadione, mephyton), 1 to 25 mg orally or IM, decreases PT over 8 to 24 hours
    - Vitamin K can be given IV with a faster effect (4–12 hours), but there is a risk of anaphylaxis and death (rare, but the incidence is hard to quantify), so this route is reserved for severe hemorrhage caused by coagulopathy secondary to vitamin K deficiency
- Heparin Overdose
  - Binds anti-thrombin III (ATIII), inhibiting thrombin and activated factors IX, X, XI, XII
  - Treatment
    - Very short half-life; discontinuing heparin is usually sufficient
    - For patients with severe overdose, protamine sulfate, 1 mg per 100 units heparin, can be used
      - In very high doses, protamine can inhibit coagulation.
- Liver Disease
  - Multifactorial in origin
  - Nearly all components of the clotting cascade are synthesized in the liver.
Coagulation Defects, Hemophilias
- Hemophilia A (Classic Hemophilia)
  - Factor VIII $\rightarrow$ normal amount but abnormal function; normal PT but prolonged PTT (affects intrinsic pathway)
  - Severity of disease is proportional to factor activity:
    - Severe: under 1% activity $\rightarrow$ spontaneous bleeding
    - Moderate: 1% to 5% activity $\rightarrow$ bleeding follows trauma or surgery, occasional spontaneous bleeding
    - Mild: >5% activity $\rightarrow$ occasionally hemorrhage after tonsillectomy, dental extractions, heavy menses
  - Major morbidity: joint bleeds (90% of bleeds into joints and muscle)
  - Major mortality: AIDS (transfusion related), strokes; mucosal and GI bleeds are RARE
  - Trauma patients: late bleeding can occur up to 3 days after, but usually by 8 hours
    - Admit and observe if significant trauma may have occurred, if the patient has deep lacerations or hematomas in vulnerable areas (e.g., eye, neck), or if the patient has a head injury.
  - Patients unresponsive to home therapy may be seen in the emergency department. Believe them when they say they are bleeding, and start therapy.
  - Treatment
    - Principles: believe the patient and treat early; local measures include ice, compression, and splinting; consult hematologist
      - Treat all potential head injuries to avoid intracranial bleeds.
    - Factor VIII is now the most commonly used concentrate.
      - Prepared in different strengths and derived from pooled, paid, screened donors
      - Higher cost (very expensive), but less volume is required and the product has a longer shelf life than cryoprecipitate and FFP
      - Special preparation techniques reduce infection rate; disease transmission rate is ZERO.
      - Monoclonal-antibody–purified recombinant factor VIII is available in limited quantities.
      - Dosing of factor VIII concentrate is dependent on the severity of the potential bleeding.
        - Potential for minor bleeding (hemarthrosis, hematuria): 12.5 u/kg
        - Potential for severe bleeding (major surgery, trauma, head injury): 50 u/kg
    - Cryoprecipitate: 80 to 100 units of factor VIII per single donor bag + 250 mg fibrinogen
      - Now used mostly if factor VIII concentrates are not available
    - Desmopressin acetate (DDAVP), a synthetic analogue of vasopressin
      - Drug of choice for acute bleeding or prophylaxis in mild/moderate hemophilia A and Type I von Willebrand's disease
      - Can rapidly stimulate a rise in functional factor VIII by 3- to-5 fold (onset in 30 minutes; peak 90 to 120 minutes)
      - Dose: 0.3 mcg/kg/dose IV
    - Fresh frozen plasma (FFP): one unit of factor VIII per milliliter of FFP, so volume overload is a limiting factor
- von Willebrand's Disease
  - Most common genetic bleeding disorder
Von Willebrand's factor (vWF) acts as bridge between platelets and exposed endothelium, serves as basis for hemostatic plug; also complexes with and stabilizes factor VIII (VIII-vWF), protecting it from rapid clearance. Lab findings: normal PT, usually a normal PTT (prolonged PTT in 25%), prolonged bleeding time, platelet count is normal but platelet function is decreased (due to decreased adhesion from lack of factor VIII/vWF complex). Variable manifestations, generally milder than hemophilia A; GI, mucosal, and cutaneous bleeds are more common. Treatment: “Humate-P” factor VIII concentrate or cryoprecipitate (most factor VIII concentrates, except Humate P, lack adequate vWF activity for serious bleeding). FFP may be used if Humate-P and cryoprecipitate are not available. DDAVP (desmopressin) can be used, but usually after hematology consultation. Humophilia B (Christmas Disease) Sex-linked factor IX deficiency (normal antigen level, abnormal procoagulant activity); prolonged PTT, normal PT. Clinical presentation: same as hemophilia A. Treatment: Purified factor IX concentrate or recombinant factor IX. Prothrombin complex factor concentrate containing factors II, VII, IX, and X. This complex carries a high risk of hepatitis, HIV transmission, and thrombosis. FFP can be used but has a higher infection rate than recombinant factor IX. Disseminated Intravascular Coagulation (DIC) Simultaneous unregulated activity of coagulation and fibrinolytic pathways → “consumptive coagulopathy.” Caused by infection, obstetric pathology, trauma, malignancy, drugs, transfusion, snake bites. May result in diffuse bleeding (loss of platelets, clotting factors, and fibrinolysis), ischemia (small vessel obstruction by fibrin), and anemia (hemolysis). Diagnosis: a clinical presentation consistent with DIC combined with the following lab abnormalities: Fibrin split products (FSP) are elevated in >95% of patients with DIC (test may not be available in the ED). D-dimer is more specific, but less sensitive, for DIC. Prolonged PT, PTT, and thrombin time (TT); decreased platelets; and decreased fibrinogen may be seen. Fragmented RBCs on peripheral smear. Treatment: Treat underlying cause and provide general supportive care (this alone may be sufficient). Further care depends on the clinical picture: If the patient is bleeding, packed RBC transfusion and platelet transfusion will be needed. Replace coagulation factors with FFP and cryoprecipitate (if the patient is in hemorrhagic shock). If thrombosis and fibrin deposition predominate, use heparin at low doses (300–500 units/hour IV) or use low-molecular-weight heparin. The outcome of septic shock (with or without DIC) is improved with the use of activated protein C (drotrecogin alfa). Immune Thrombocytopenic Purpura (ITP) (old name – idiopathic thrombocytopenic purpura) IgG antiplatelet antibody; increased clearance of platelets by reticuloendothelial system.
Two forms

Acute
- Usually seen in childhood (2 to 6 years) after a viral prodrome
- Platelet count may fall to <20,000/mm^3
- Self-limited (-90% spontaneous remission in 6–12 months)
- Steroids and splenectomy are rarely needed.
- If needed for severe bleeding, purified human immunoglobulin given intravenously, 1 gm/kg, can suppress the activity of the anti-platelet antibody

Chronic
- Usually seen in adults; female: male ratio is 3:1
- Insidious onset (no prodrome)
- Variable course and severity (waxes and wanes)
- Other than bleeding abnormalities (petechiae [see Image #36], purpura, low platelet count), physical exam is normal (splenomegaly is rare).
- Platelet counts range from 30,000 to 100,000/mm^3
- Consider corticosteroids and splenectomy; immunosuppression may be needed.
- Life-threatening bleeds should be treated with IV immunoglobulin, corticosteroids, and platelet transfusion.

Thrombotic Thrombocytopenic Purpura (TTP)
- Intravascular and subendothelial fibrin and platelet deposition in capillaries and arterioles
- Cause is usually idiopathic, but some medications can be associated with it
- Can occur at any age, but most patients are 10 to 40 years old; women are more often affected than men
- Presentation is variable—only ~40% exhibit the entire classic pentad:
  - Thrombocytopenia – usually severe (10,000–50,000/mm^3)
  - Microangiopathic hemolytic anemia (Hb <7 g/dL, HCT <20%)
  - Fluctuating neurologic symptoms (strokes, altered mental status, seizures, coma)
  - Renal disease that is usually mild, with hematuria and proteinuria; acute renal failure can occur
  - Fever
- Diagnosis is challenging because there are no specific clinical or laboratory diagnostic criteria.
- Treatment
  - Massive plasma exchange with FFP
    - Usually in combination with steroids and anti-platelet drugs, such as aspirin (response rate ~80%)
    - Untreated results in mortality rate of ~80% to 90% within 1 to 3 months
    - Aggressive treatment results in mortality rate of 15% to 20%
    - Avoid platelet transfusion unless life-threatening bleed, as it may cause new thrombi

Hemolytic-Uremic Syndrome (HUS)
- Acute renal failure, microangiopathic hemolytic anemia (MAHA), fever, and thrombocytopenia
  - Similar to TTP but without the CNS involvement and with more severe renal involvement
- One of the most common causes of acute renal failure in children
- Often follows prodromal infectious disease
  - Usually diarrhea (90%) or upper respiratory illness (10%)
  - Antimotility drugs for diarrhea may increase risk
  - Most commonly associated diarrheal illness is caused by *E. coli* serotype 0157:H7; toxin produces the effects that are seen
o Usually age 6 months to 4 years; mortality 5% to 15%; worse prognosis in older children and adults

o Treatment
  ▪ Younger children need supportive care with fluid restriction and admission.
    □ Antibiotics may worsen condition/outcome.
    □ Transfuse RBCs or platelets as needed.
    □ Dialysis may be necessary (in up to 50% of cases).
  ▪ Older children and adults should be managed similar to those with TTP.

• Heparin-Induced Thrombocytopenia
  o Immune mediated; usually occurs during the first week of treatment
  ▪ Can be delayed from 2 weeks to 1 month after heparin treatment
  o Thromboses may develop, with sequelae dependent on thrombus location
  o Either thrombocytopenia or a 50% decrease in platelet count will be seen.
  o Treat with lepirudin or argatroban (both are direct thrombin inhibitors).

• Others Drugs Causing Inactivation of Platelets
  o Most common: quinidine/quinine, sulfonamides, gold salts, phenytoin, aspirin
  o Less common: amrinone, chronic ethanol, indomethacin, valproic acid, heroin
  o Thrombocytopenia develops within 24 hours after drug use (platelets may be below 10,000/mm³) secondary to the development of antiplatelet antibodies.

  o Treatment
    ▪ Remove offending agent; spontaneous recovery over 1 week
    ▪ Tapering dose of prednisone
    ▪ Treat severe hemorrhage with DDAVP and platelet transfusions

11.5 ONCOLOGIC EMERGENCIES

Local Tumor Compression

• Acute Spinal Cord Compression
  o Incidence: <5% of oncology patients
  o Common causes are breast and lung cancer, lymphoma
  o Progressive weakness → pain → paralysis (lower extremity weakness, decreased sensation, urinary retention)
  o Treatment: dexamethasone, radiation therapy, surgery

• Brain Herniation
  o Caused by primary or metastatic tumors, especially lung, breast, kidney, and colon
  o Three types
    ▪ UncaI herniation: brain stem compression from lateral mass in temporal lobe
      □ Ipsilateral paralysis and unilateral dilated pupil
    ▪ Central herniation: from multiple locations, downward pressure on upper brainstem (pons)
      □ Depressed level of consciousness, Cheyne-Stokes respirations, small reactive pupils, no focal signs
    ▪ Tonsillar herniation: lower brain stem (medulla) compression from mass in cerebellar tonsil(s)
      □ Vomiting, occipital headache, altered mental status
  o Treatment
    ▪ Intubate to protect airway, and elevate the head of bed
      □ Hyperventilation is used very selectively
• IV mannitol and steroids
• Surgery versus medical management (antibiotics, chemotherapy, or radiation therapy), depending on nature of intracranial mass

• Upper Airway Obstruction
  o Insidious onset of upper airway growths
  o Common causes: laryngeal and thyroid cancer, lymphoma
  o Treatment: establish definitive airway if stability is in question

• Malignant Pericardial Effusion with Tamponade
  o Symptoms depend on speed of onset
  o Symptoms include sudden shortness of breath, chest pain, hypotension, anxious feeling
  o Causes include malignant melanoma; lymphoma; lung, breast, and ovarian cancer; pericarditis secondary to radiation therapy
  o Signs include pulsus paradoxus >10 mm Hg, low-voltage ECG +/- electrical alternans, cardiomegaly on chest film
    - Echocardiogram is often the quickest, easiest test (see Image #32)
  o Treatment: if unstable, emergent pericardiocentesis of as little as 50 mL may be enough to temporize the condition (send fluid for analysis)
    - Otherwise, discuss with oncologist

• Superior Vena Cava Syndrome
  o Obstruction to venous flow from upper body → head congestion, face and neck fullness
    - Facial plethora, papilledema (see Image #29), palpable supraclavicular mass
    - May have dyspnea, cough, difficulty swallowing
  o Most common: lung cancer (small cell and squamous cell), lymphoma, breast cancer, testicular cancer
    - Can be caused by nonmalignant processes
  o Treatment: general approach emphasizes chemotherapy with potential intraluminal stent placement
    - Elevate the head of the bed.
    - Diuretics for symptomatic relief
    - Steroids if respiratory compromise; otherwise, steroids have little clinical benefit
    - Radiation therapy
    - Usually life-threatening only if there is concurrent tracheal compression

Biochemical Derangement and System Collapse in Oncology Patients
• Fever
  o Considered “significant” if a single oral temperature is ≥38.3°C (101°F) or if temperature is ≥38°C (100.4°F) for more than 1 hour
  o May be caused by malignancy or therapy; the majority of time, it is related to infection secondary to immunosuppression
  o Risk of infection raised with granulocytopenia, ≤1000 cells/mm³
  o Granulocytopenia results in minimal or lack of “traditional” signs of inflammation (e.g., rubor, calor)
  o Workup and treatment
    - Look closely for subtle markers of infection.
    - Obtain a CBC with differential, PT/PTT, urinalysis, basic chemistry
    - Pan-culture with urine and blood cultures (aerobic, anaerobic, fungal)
      - Draw at least one set of blood cultures from an indwelling catheter (if present)
• Chest film is often standard, but is really necessary only if the patient has respiratory symptoms or signs
• Treat early with broad-spectrum antibiotics, possibly including antifungal coverage

• Acute Tumor Lysis Syndrome
  o During the first week of chemotherapy or radiation treatment, especially with rapidly growing malignancies such as leukemia and lymphoma
  o Hyperuricemia (from cell breakdown), hyperphosphatemia with secondary hypocalcemia, and hyperkalemia may be seen.
    • Acute renal failure, dysrhythmias (VFib or VTach), and neuromuscular problems (hypocalcemia) may also develop.
  o Treatment
    • Hyperuricemia: hydration, prn diuretics, urine alkalinization, allopurinol
    • Delay further chemotherapy and radiation therapy if possible.
    • Hemodialysis may facilitate correction of metabolic derangements.

• Hypercalcemia of Malignancy
  o High levels affect heart, brain, muscle
  o Common causes: renal cell, lung, and breast cancer; leukemia; multiple myeloma, especially if there is bone involvement
  o Manifestations: nausea, vomiting, anorexia, constipation, various CNS changes (especially decreased mental status), shortened QT interval
  o Severity depends on rapidity of onset, not the absolute level
  o Treatment
    • Saline (hydration) and furosemide (facilitates excretion)
      • Monitor other electrolytes (especially K⁺), cardiac monitoring
    • Rapid infusion of inorganic phosphate lowers calcium rapidly, but causes metastatic tissue calcification and death
    • Corticosteroids used for long-term control, along with oral bisphosphonates (for example, pamidronate)

• Syndrome of Inappropriate Antidiuretic Hormone (SIADH)
  o Hyponatremia and high urine sodium with euvolemia
    • Urine osmolarity > serum osmolarity
  o Causes
    • Include malignancies of the brain (primary and metastatic), lung (small cell), pancreas, prostate
    • Pulmonary processes and infections
    • Drugs such as vasopressin, diuretics, narcotics, selective serotonin reuptake inhibitors (SSRIs)
  o Anorexia, confusion, nausea, vomiting, weakness; seizures if severe
  o Na⁺ concentration <115 mEq/L may be life threatening
  o Treatment: correct serum sodium concentration slowly to avoid cerebral demyelination and central pontine myelinolysis
    • Fluid restriction to 500 to 750 mL/day
    • Diuretics (for example, furosemide) and hypertonic saline (3% NaCl) may be needed for severe hyponatremia (seizures, coma)

• Hyperviscosity Syndrome
  o Sludging, with a reduction in microcirculatory perfusion
  o Common causes are leukocytosis; polycythemia; and elevated serum proteins (most common) as in multiple myeloma, Waldenström's macroglobulinemia, and chronic myelogenous leukemia (CML)
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- Manifestations: fatigue, headache, anorexia, somnolence, deafness, blindness, strokes, seizures
  - Classic triad comprises bleeding, changes in vision, and neurologic disturbances.
- Treatment
  - Perform a 2-unit phlebotomy and replace the volume removed with saline infusion; obtain oncology consult for chemotherapy.

- Adrenocortical Insufficiency with Shock
  - Vasomotor collapse due to adrenal malfunction
  - Common oncologic causes: lung and breast cancers, melanoma, retroperitoneal malignancies
    - Can also result from nononcologic causes, such as previous glucocorticoid administration (most common), infection, hypothalamic or pituitary insufficiency
    - The rare adrenal hemorrhage may be caused by trauma (including birth and seizures), anticoagulation, or overwhelming septicemia (the Waterhouse-Friderichsen syndrome).
  - Manifestations: fever, dehydration, shock, collapse
    - Mild hypoglycemia, hyponatremia, hyperkalemia, and eosinophilia may be seen.
  - Treatment: emergent dosing with hydrocortisone, 100 mg IV every 6 to 8 hours

11.6 IMMUNOLOGY

Collagen Vascular Disease

- Raynaud’s Disease
  - Vasospastic disorder without identifiable underlying cause
  - Female: male ratio is 5:1
  - Diagnostic criteria with 95% specificity
    - Precipitated by environmental or psychological stressors
    - Bilateral distribution
    - Significant gangrene is absent
    - No identifiable underlying or associated disease (Raynaud’s phenomenon)
    - Occurs for >2 years
  - Presentation: classically triphasic
    - Vasospasm causes alteration in blood flow to palmar and digital arteries/arterioles
    - Fingers change from white (pallor due to cessation of blood flow) to blue (cyanosis from initial reperfusion with relaxation of spasm) to red (reactive hyperemia as full blood flow is restored)
    - Associated pain and paresthesias
    - Generally self limited, without histologic changes in the vessel walls
  - Treatment
    - Reassurance (and re-warming if necessary)
    - Education and continued PCM follow-up

- Reiter’s Syndrome—“Can’t see, can’t pee, can’t climb a tree”
  - Reactive arthritis in genetically susceptible individuals (HLA-B27 involvement) that presents 1 to 3 weeks after the infections listed below:
    - *Chlamydia trachomatis* infection of the GU tract
    - *Salmonella/Shigella/Yersinia/Campylobacter* infection of the GI tract
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Presentation
- Early
  - Urethritis or urinary symptoms
  - Diarrhea or enteritis
  - Conjunctivitis – may progress to uveitis or corneal ulceration
  - Shallow painful ulcers of oral mucosa and glans penis
  - Digital edema (“sausage” finger and toes)
- Late
  - Musculoskeletal symptoms – low back pain with diminished truncal flexion and Achilles tendonitis
  - Sterile inflammation of synovial fluid
  - Enthesopathic changes – inflammation at sites of tendon insertions and resultant erosion of surrounding bone
    - Interphalangeal (IP) joint of great toe and sacroiliac (SI) joint are most commonly involved.

Treatment
- Early resolution can be aided by tetracycline therapy in patients with chlamydial infections.
- Supportive care is paramount.
- Nonsteroidal anti-inflammatory medications are recommended.
  - Indomethacin is preferred – up to 200 mg/day

Prognosis
- Symptoms may become recurrent, with possible progression to ankylosing spondylitis and aortic insufficiency.

Rheumatoid Arthritis
- Chronic systemic inflammatory disease related to the following:
  - Immune complex formation
    - Subsequent influx of polymorphonuclear cells (PMNs) into synovial cavity of the joint spaces, which release lysosomal enzymes, eliciting a severe inflammatory reaction
  - Erosion of articular cartilage and bone
- Peak incidence during the fourth to sixth decades; female: male ratio, 2.5:1
- Diagnostic criteria (four of seven required):
  - Morning stiffness lasting for more than 1 hour
  - Symmetric, additive pattern of joint involvement
  - Polyarticular involvement (more than three joints)
  - Articular involvement of the hands (sparing the distal interphalangeal [DIP] joints)
  - Positive serum rheumatoid factor
  - Subcutaneous rheumatoid nodules
  - Radiographic evidence of rheumatoid arthritis
- Acute presentation
  - Warm, tender, and swollen joints
  - May appear similar to a viral arthropathy
  - Tenosynovitis, acute pericarditis, and knee effusions are possible.
Subacute or chronic presentation

- Long-term changes are more likely to be present:
  - Swelling of the metacarpophalangeal (MP) and proximal interphalangeal (PIP) joints and ulnar deviation
  - Swan neck and boutonniere deformities
  - Diminished dorsiflexion at the wrist
  - Long-standing disease – risk for degeneration of transverse ligament and possible atlanto-axial instability
    - Minor trauma can lead to neurologic sequelae → caution during intubation to avoid cord damage

Possible concomitant remote organ system involvement

- Cardiac – carditis, pericarditis
- Pulmonary – pleuritis, intrapulmonary nodules, interstitial fibrosis
- Hepatic – hepatitis
- Ocular – scleritis, episcleritis, keratoconjunctivitis sicca in 25% of patients (ocular discharge and foreign body sensation with dry eye)
- Vascular – vasculitis
- Integumentary – subcutaneous nodules
- Felty syndrome – rheumatoid arthritis (RA), neutropenia, splenomegaly

Diagnostic approach

- Exclude acute causes – septic/infectious arthritis; bursitis/tendonitis, osteoarthritis
- Polyarthralgia (especially lasting >6 weeks):
  - CBC – normochromic, normocytic anemia (80%)
  - Erythrocyte sedimentation rate, C-reactive protein, and rheumatoid factor – sensitive, but not specific, tests
  - Arthrocentesis – imperative!
    - Evaluate synovial fluid for crystals and purulent material.
    - Perform gram stain and culture.
    - Anticipate a cell count of 4,000 to 50,000 with predominance (75%) of PMNs
    - Low glucose
  - Radiographic studies – MRI (as a part of the outpatient workup) is beneficial in demonstrating early evidence of uniform joint space narrowing

Treatment

- Initial ED treatment:
  - Rest affected joint via splinting
    - Upper extremity – 3 weeks
    - Lower extremity – 8 weeks
- Suppression of inflammation
  - “Bridge concept” – stepwise approach involving the following:
    - Salicylate or NSAID therapy
    - Glucocorticoids (prednisone, 5–7.5 mg QD)
    - Slow-acting anti-rheumatic drugs (SAARDS) – methotrexate, gold, hydroxychloroquine, infliximab) – are an important part of adjunctive outpatient therapy
• Scleroderma (Systemic Sclerosis)
  o Systemic autoimmune connective tissue disease of unknown etiology
  o Excessive production and deposition of Type I + III collagen
  o Characterized by progressive inflammation and fibrosis of the skin, multiple organ systems, and vasculature
  o CREST syndrome = systemic sclerosis
    ▪ C – Calcinosis
    ▪ R – Raynaud’s phenomenon
    ▪ E – Esophageal dysmotility
    ▪ S – Sclerodactyly
    ▪ T – Telangiectasia
  o Organ systems involved:
    ▪ Integumentary – thin epidermis anchored to lower layers by collagen deposition
      ▪ Skin appears tight, with loss of skin creases and mobility.
      ▪ Skin pigmentation changes and atrophy occur late.
    ▪ GI tract – atrophy of GI smooth muscle leads to the following:
      ▪ Lower esophageal sphincter incompetence and esophageal dysmotility
        ▪ Resultant reflux, dysphagia, and delayed gastric emptying
      ▪ Small bowel dysmotility causing diarrhea
      ▪ Colonic dysmotility causing constipation
    ▪ Pulmonary – Diffuse fibrotic changes throughout lungs, with possible pulmonary hypertension
    ▪ Cardiac – Fibrosis of conduction system, leading to abnormal conduction and dysrhythmias, fibrinous pericarditis causing congestive heart failure
    ▪ Renal – >50% have intimal hyperplasia of interlobular arteries, fibrinoid necrosis of afferent arterioles, and glomerular basement membrane thickening
      ▪ May lead to renal failure and/or malignant hypertension
    ▪ Musculoskeletal – inflammatory joint pathology similar to that seen in rheumatoid arthritis
  o Appropriate evaluation
    ▪ CT scan and PFTs to evaluate for pulmonary fibrosis
    ▪ Echo – evaluate for pericardial effusions (see Image #32) and monitor for pulmonary hypertension
    ▪ Esophagogastroduodenoscopy (EGD) – evaluate degree of esophageal dysmotility
    ▪ Studies performed in the emergency department are chosen depending on the severity of presentation.
      ▪ It is likely that PFTs and EGD can be done on an outpatient basis by a PCM, rheumatologist, or consultant.
  o Conservative treatment
    ▪ D-Penicillamine to treat skin changes
    ▪ Moisturizers and anti-histamines for pruritis
    ▪ Proton pump inhibitors (PPIs) and H2 blockers for GERD symptoms
    ▪ ACE-I to prevent hypertension and renal crisis
    ▪ NSAIDs for arthralgias
    ▪ Rheumatology consult
    ▪ Avoidance of high-dose vitamin C (stimulates collagen formation)
    ▪ Emergency department management (depends on presentation) should include referral to rheumatologist and symptomatic management.
      ▪ Patient will likely require at least skin care, NSAIDs, and PPIs
Systemic Lupus Erythematosus (SLE)

- Autoimmune disease of multiple organ systems causing systemic complications (including renal dysfunction and neurologic sequelae)
- Mechanism of disease – complex interaction of genetic, environmental (sunlight), and hormonal factors (estrogen), which leads to an abnormal cellular and humoral response of polyclonal B cells and an exaggerated production of autoantibodies
  - Autoantibodies can produce immune complex formation, or they can cause disease in a specific organ system via direct tissue binding interactions

- Diagnostic criteria – at least 4 of 11 criteria for the diagnosis of SLE:
  - Malar rash
  - Discoid rash
  - Photosensitivity
  - Oral ulcers
  - Arthritis
  - Serositis (pleuritis or pericarditis)
  - Renal disorders (persistent proteinuria or cellular casts)
  - Neurologic disorder (seizure or psychosis)
  - Hematologic disorder (hemolytic anemia, leukopenia, lymphopenia, thrombocytopenia)
  - Immunologic disorder (Anti-ds DNA Ab, Anti-Sm Ab, antiphospholipid Ab)
  - Antinuclear Ab (ANA)

- Clinical features
  - Rheumatologic
    - Arthralgias, fatigue, and myalgias are prevalent symptoms.
    - Inflammatory pattern similar to RA (except that SLE is non-erosive and non-deforming), with symmetric involvement of PIP and MP joints
  - Dermatologic
    - Malar rash (fixed erythema of face, sparing nasolabial folds) documented in up to 40% of SLE patients
    - Discoid lupus (erythematous plaques to head and neck) seen in 25%
    - Shallow oral ulcers of mucous membranes may also be present
  - Renal
    - Nephritis in 50% of cases
    - Asymptomatic until progression to frank nephritic syndrome or renal failure
  - Neurologic
    - Seizures are the most common CNS manifestation (70%).
    - Strokes are also common due to association with antiphospholipid syndrome.
    - Lupus cerebritis/frank psychosis
    - Migraine headache
    - Peripheral neuropathy
  - Cardiovascular
    - Pericarditis is a common manifestation.
    - Vasculitis with digital infarcts and splinter hemorrhages (see Image #44)
    - Systemic hypertension due to lupus nephritis
    - Increased risk of coronary artery disease
Pulmonary
- Exudative pleural effusions and pleurisy are common findings.
- Hemoptysis may occur secondary to pulmonary hemorrhage or infarcts.
- Pulmonary fibrosis and pulmonary hypertension may also occur.

Diagnostic approach
- Check ANA (higher titers increase positive predictive value)
  - Anti-ds DNA and anti-Smith antibodies have increased specificity for SLE.
- Check urinalysis and serum creatinine for disease flare or worsening nephritis.
- Remain suspicious for underlying infection, especially in patients with low-grade fever and those who are receiving immunosuppressive therapy.
- Emergency department diagnostic workup should depend on severity of disease process and whether previous lupus diagnosis has been made.

Treatment
- Symptomatic therapy
  - Avoidance of stress and fatigue
  - Avoidance of prolonged direct sunlight exposure (in conjunction with recommendation for sunscreen use)
  - Discontinue oral contraceptive pills (OCPs) or use low-estrogen formulations only.
  - Acetaminophen and NSAIDs for arthralgias, pleurisy, pericarditis
    - Avoid overuse of NSAIDs to prevent worsening of likely underlying nephritis
- Corticosteroid therapy
  - Minor disease – prednisone, 0.5 mg/kg in a single daily dose
  - Major disease – prednisone, 1 mg/kg in a single daily dose
- Anti-malarial agents and immunosuppressive agents (MTX, azathioprine, etc.) may be required on an outpatient or inpatient basis, in conjunction with internal medicine and rheumatology consultation.

Vasculitis
- Disease process causing inflammatory changes and destruction of blood vessels
- Suspected stepwise pathophysiology:
  - Immune complex deposition
  - Activation of complement system
  - Attraction of PMNs and release of lysosomal enzymes
  - Vessel wall damage and deterioration
- Large vessel vasculitis
  - Temporal (Giant cell) arteritis (see Chapter 7, Head, Ear, Eye, Nose, and Throat Disorders)
- Medium vessel vasculitis
  - Polyarteritis nodosa
    - Acute inflammation and necrosis of medium vessels
    - Specific to nervous system and GI tract
    - pANCA (-)
      - Differentiated from microscopic polyangitis (MPA), which occurs in nerve, glomerular, and lung tissue
      - MPA is pANCA (+)
    - Must first exclude hepatitis B and C
      - Produce similar clinical pictures but require completely different treatment
Early prodrome (fever, malaise, arthralgia, myalgia)
- Progresses to the following:
  - Peripheral neuropathy
  - Ischemic bowel changes
  - Secondary hypertension from renal artery inflammatory changes
- Diagnosis based on recognition of clinical pattern and biopsy of arterial segment
- Treatment – corticosteroids, high-dose immunosuppressant therapy

Wegener's granulomatosis
- Necrotizing granulomatous vasculitis involving the lungs, kidneys, and medium-sized vessels
- Rare disease, often seen in men after age 45
- Disease progression:
  - Upper respiratory symptoms (sinusitis, otitis, and ulceration of the nasal cavity) are initial findings.
  - Lower respiratory symptoms (cough, dyspnea, hemoptysis, pulmonary infiltrates, tracheal stenosis) develop next.
  - Glomerulonephritis and ocular/skin involvement (granulomatous deposition) are late findings.
- cANCA (+) – highly sensitive and specific for diagnosis
- Definitive diagnosis is made with lung biopsy.
- Diagnosis should prompt admission because of the high mortality rate (most patients with this condition die within a year as a result of complications related to renal disease).
  - Initial diagnosis – inpatient admission for treatment with combination of cyclophosphamide and IV corticosteroids
  - Flare or complications of renal disease – inpatient admission for at least IV corticosteroids

Small vessel vasculitis
- Hypersensitivity vasculitis
  - Small vessel (typically venules) vasculitis caused by immune complex deposition from inflammatory antigen (drugs and infectious organisms)
  - Typical offending agents – penicillins, sulfa drugs, non-steroidal anti-inflammatory drugs (NSAIDs), streptokinase
  - Symptoms
    - Skin lesions – flat, erythematous, purpuric papules or purpura that progress to bullae
    - Commonly found on lower extremities with associated edema
- Henoch-Schoenlein purpura (see Chapter 5, Pediatrics)
- Nodular vasculitis/panniculitis
  - Hypersensitivity vasculitis of the vessels of the subcutaneous layers of the skin
  - Seen more often in women in the third decade
  - Subcutaneous nodules
    - Erythema nodosum – erythematous nodules on both shins (blue hue during resolution, see Image #38)
    - Panniculitis – nodules on calves
  - Self-limited process; look for underlying cause of vasculitis
11.7 ANAPHYLACTIC/ANAPHYLACTOID REACTIONS

Classic Immunopathology of Anaphylaxis
- Immune-mediated injury that results in immediate-type hypersensitivity reaction
- Sensitization to allergen causes production of IgE antibodies
- IgE Ab fixation causes upregulation of antigen receptors.
- Re-exposure to antigen causes a massive IgE-induced release of mediators from mast cells and basophils.
- Mediator release causes a myriad of reactions (typically in organ systems rich in mast cells/basophils)
  - Increased mucous secretions in pulmonary and GI tracts
  - Increased bronchial smooth muscle tone (airway compromise)
  - Decreased vascular smooth muscle tone (shock)
  - Increased capillary permeability (shock)
  - Mediator-induced coagulation deficits – fibrinolysis/DIC

Anaphylactoid Reaction
- Clinically Similar to Anaphylaxis
  - Antigen exposure leads to direct degranulation of mast cells and basophils, independent of IgE
  - Most common inciting agents:
    - Parenteral antibiotics (especially penicillins)
    - Latex
    - Hymenoptera stings
    - Food allergens (most notably peanuts and shellfish)
    - IV contrast (especially older hyperosmolar contrast agents)

Clinical Signs and Symptoms of Anaphylaxis
- Dermatologic Manifestations
  - Usually the first clinical sign of anaphylaxis
  - Sensation of generalized warmth
  - Tingling of face, mouth, chest, palms/soles, or exposure site
  - Pruritis (nearly universal), with generalized flushing and urticaria
  - Angioedema (see Image #67) is possible.
- Pulmonary Manifestations
  - Upper respiratory tract
    - Nasal congestion, sneezing, cough
    - Laryngeal edema and oropharyngeal angioedema (see Image #67) causing respiratory distress
  - Lower respiratory tract
    - Chest tightness, dyspnea, and wheezing from bronchospasm caused by smooth muscle contraction
- Cardiovascular Symptoms
  - Light-headedness or syncope
    - Manifestations of hypotension or dysrhythmias (also from hypoxia)
- Ocular Symptoms
  - Ocular itching
  - Increased lacrimation
  - Conjunctival injection
• Gastrointestinal Symptoms
  o Nausea, vomiting, and diarrhea
  o Cramping abdominal pain
  o Tenesmus
  o Hypersalivation

Physical Exam Findings
• Vital Signs
  o Tachypnea
  o Tachycardia
  o Hypotension

• General
  o Anxiety
  o Decreased responsiveness with severe disease

• Head and Neck
  o Laryngeal stridor
  o Hypersalivation
  o Hoarseness
  o Oropharyngeal angioedema (see Image #67)

• Pulmonary
  o Wheezing and reduced airflow
    ▪ From lower airway bronchoconstriction
  o Complete airway obstruction
    ▪ Most common cause of death due to anaphylaxis
    ▪ Stridor and hoarse voice
    ▪ Wheezing secondary to bronchospasm
    ▪ Progressive edema and increased bronchospasm
  o Impending airway compromise
    ▪ Lack of ability to speak
    ▪ Decreased wheezing

• Dermatologic
  o Urticaria
    ▪ Erythematous raised plaques that blanche
    ▪ Common finding
  o Angioedema – typically seen in lips, palms, soles, and genitalia (see Image #67)

• Cardiovascular
  o Circulatory collapse from hypotension due to increased vascular permeability or decreased tone
  o Dysrhythmias are common.
    ▪ Sinus tachycardia
    ▪ PAC/PVC
    ▪ Atrial fibrillation
Management of Anaphylaxis

- Emergency Management
  - Removal of offending or triggering agent
  - O₂
  - Cardiac monitoring
  - Intravenous fluids (large-bore IV line with crystalloid)
  - Suctioning of upper airway secretions
  - Assessment of airway patency
    - Jaw thrust/chin lift
    - Racemic epinephrine while awaiting definitive airway
    - Early intubation due to high likelihood of rapidly progressive edematous changes to airway
      - Endotracheal intubation with rapid-sequence induction
      - Failure followed by surgical airway (standard cricothyrotomy)
- Hypotension – typically caused by vasodilation and capillary fluid leakage
  - Typical treatment
    - Fluid resuscitation
    - Epinephrine therapy
      - Subcutaneous route – appropriate for mild symptoms
      - Intramuscular route – recommended for more severe disease causing concern; this method of delivery is rapid and predictable
      - Intravenous route – for emergent crisis
    - H1 blockers (e.g., diphenhydramine) and H2 blockers (e.g., ranitidine) used for anti-histaminic effects
- Refractory Hypotension
  - Treatment
    - Large volumes of IVFs
    - Repeated doses of epinephrine
      - Constant IV infusion as needed
    - Addition of other pressors with α-adrenergic activity
      - Norepinephrine
      - Dopamine
- Drug Therapy
  - Epinephrine – first drug of choice in treatment of anaphylaxis
    - Combined α- and β-adrenergic agonist actions
      - α-Adrenergic agonist effect
        - Increases peripheral vascular resistance
        - Reverses peripheral vasodilation, vascular permeability
        - Improves systemic hypotension
      - β-Adrenergic agonist effect
        - Bronchodilation
        - Positive inotropic/chronotropic cardiac activity
        - Increases cAMP (inhibits further mediator release)
    - Dangers of epinephrine therapy
      - Increased α activity can cause hypertensive crisis
      - Increased β activity can cause myocardial ischemia
CHAPTER II

• Hematologic/Oncologic and Immune System Disorders

Dosage/administration

- 0.01 mL/kg of 1:1,000 solution given SC or IM, depending on clinical picture
  - Maximum of 0.5 mL of 1:1,000 (0.5 mg)
  - 0.1 to 0.2 mL given directly at site of antigenic exposure (if identified)

- Severe symptoms
  - IV infusion of 10 mL of 1:100,000 aqueous epinephrine over 10 minutes (10 mcg/min bolus or 100 mcg total)
  - Continuous infusion at 1 mcg/min for adults (if needed after initial IV infusion)
  - Alternately, use 0.1 mcg/kg/min in children and infants, increasing by 0.1 mcg/kg/min to a maximum of 1.5 mcg/kg/min, as needed
  - If no IV access, aside from SC or IM, can give IV dose via either intraosseus or endotracheal route

Antihistamines

- Competitively block the action of circulating histamines at target tissue cell receptors
  - Do NOT reduce mediator release or affect leukotrienes
  - H-1 blockers
    - Diphenhydramine is most commonly used.
      > Typical dose is 50 mg every 4 to 6 hours
      > Loading dose of 1 to 2 mg/kg to a maximum of 100 mg IV may be used in severe cases.
  - Concomitant use of H-2 blockers
    - Helps to inhibit effects of histamine on myocardial and peripheral vascular tissue

Corticosteroids

- Acute utility is limited by lengthy onset of action (4–6 hr)
- Helps prevent the biphasic nature of anaphylaxis
- Recurrence of cutaneous and systemic symptoms, after initial resolution

Glucagon therapy

- Useful in patients on β-blockers
- Positive inotropism by increasing cAMP synthesis via a non-adrenergic mechanism
- Initial dosing
  - Adults – 1 mg IV/IM/SC as dictated by severity of clinical presentation
    - More severe reactions may require IV infusion of 1 to 5 mg/hr
  - Children – 0.5 mg IV/IM/SC

Inhaled β-agonists

- Augments epinephrine’s β-agonist effects, especially with refractory bronchospasm
  - Continuous nebulization of β-agonists may be required.

Inhaled anti-cholinergic therapy

- Ipratropium bromide
  - Acts to decrease mediator release by decreasing cGMP
11.8 ANGIOEDEMA (see Image #67)

- Subcutaneous and dermal vascular changes (arteriolar dilation and venule leak) caused by vasoactive mediators
- Common causes
  - Generally a hypersensitivity IgE-mediated allergic reaction
    - Serum sickness, complement pathway involvement, and direct mast cell stimulation can also cause angioedema
  - Variety of contributing allergens and factors
    - Usual suspects
      - IV contrast and dextran
      - Angiotensin-converting enzyme inhibitors, aspirin, nonsteroidal anti-inflammatory drugs, opiates
      - Insect venom and foods
      - Trauma
- Should be evaluated and treated similarly to anaphylactic reaction (depending on severity)
  - First-line therapy for stable symptoms – antihistamines (H1 and H2)
- Hereditary angioedema
  - Autosomal dominant process caused by C1 esterase deficiency
    - Symptoms
      - Edematous changes to airway, face, or extremities
      - Abdominal pain and enteritis
    - Refractory to normal-dose epinephrine, anti-histamine, and steroid therapy
    - Treatment
      - Airway management
      - FFP (contains C1 inhibitor)
      - Judicious use of high-dose epinephrine

11.9 SARCOIDOSIS

Diffuse Granulomatous Disorder of Unknown Etiology
- Non-caseating granulomas are a response to an exaggerated immune reaction.
- Rare disease with an estimated prevalence of 15 cases per 100,000 persons
  - Majority present between 10 and 40 years of age (70%-90%)
- Pulmonary involvement – >90% of patients have lung involvement
  - Classic chest film findings demonstrate bilateral hilar adenopathy and reticular opacities.
  - 50% of these cases are found incidentally on chest film
- Onset can be acute or chronic, with a multitude of presenting symptoms
  - Most common symptoms: cough, dyspnea, chest pain, ocular and dermatologic lesions
- Spontaneous remission seen in up to 60% of cases
- Workup includes chest film, CBC, chemistries, and LFTs; may also perform an LP for CSF studies and consider assessment of cardiac markers
- Treatment is predominantly nonemergent, performed in outpatient setting
  - Supportive
  - Oral steroids for 2 to 3 months
  - Nonsteroidal anti-inflammatory drugs as needed
  - Consult rheumatology, other specialties as indicated
11.10 RHEUMATIC FEVER

General
- A sequela of group A β-hemolytic streptococci infection
  - Thought to be caused by antibodies cross-reacting with certain tissues (such as heart, joints, CNS)
- Greatest risk in children 4 to 18 years of age
  - Latent period of 1 to 5 weeks after pharyngitis
- A leading cause of death in children when antibiotic therapy is not readily available, as in developing nations and in the United States prior to 1950

Diagnosis
- Modified Jones criteria
  - Major criteria
    - Carditis (33% of cases)
    - Pancyteritis
    - Sydenham's chorea
      - Random, rapid, purposeless movements of the upper extremities and face
    - Erythema marginatum (<10% of cases)
      - Nonpruritic, painless, transitory pink rings on the trunk and upper extremities
    - Subcutaneous nodules (<10% of cases)
  - Minor criteria:
    - Arthralgias (60%-75% of cases)
    - Fever (usually only during initial 2 weeks of illness)
    - Increased erythrocyte sedimentation rate or C-reactive protein
    - Prolonged P-R interval on ECG
- Diagnosis is made if there is evidence of recent group A streptococci infection and if one major and two minor or two major manifestations are present, as listed above.
  - However, a presumptive diagnosis can be made if only one major or three minor criteria are present.
  - Within 4 to 6 weeks after infection, anti-streptococcal antibody titers can be detected.
  - Patients with carditis may have a heart murmur (most commonly mitral valve regurgitation), pericardial effusion (see Image #32), and CHF.

Treatment
- Prevention of illness is paramount strategy.
- Treat streptococcal pharyngitis with either oral penicillin for 10 days or a single intramuscular dose of benzathine penicillin.
  - Patients diagnosed with acute rheumatic fever will also need anti-streptococcal prophylaxis for up to 5 years with penicillin or erythromycin.
- Salicylates or NSAIDs for arthritis and mild carditis
- For moderate to severe carditis with CHF, use corticosteroids.
- Control the presentation of chorea with benzodiazepines or haloperidol.
11.11 TRANSPLANT-RELATED PROBLEMS

General
- Pain is an unreliable indicator, as organs lack innervation.
- Usual inflammatory patterns may be lacking secondary to chronic immunosuppressive agents.
  - Thus, the clinician must look for subtle symptoms and signs of illness or disease and have a very high index of suspicion and a broad evaluation/search for problems.
- Involve local transplantation specialists early in the workup for consultation and disposition decisions.
- Admission rates are high.

Complications – Generally Involve Infection, Rejection, or Drug Toxicity
- Infections After Transplantation
  - The first month
    - The usual postoperative causes and nosocomial infections predominate.
  - One to six months
    - Viruses such as cytomegalovirus (CMV), hepatitis B and C, and Epstein-Barr virus (EBV)
      - CMV (primary or reactivation of latent infection) is common and affects multiple organ systems, often insidiously presenting as pneumonitis.
        - Often fatal
        - Can also precipitate graft rejection
        - Treat with ganciclovir and CMV-specific immunoglobulin
      - EBV’s presentation is similar to that of CMV; they often coexist.
  - Six months and beyond – problems are divided into three groups: healthy transplant, chronic viral infection, chronic rejection
    - Healthy transplant
    - Slightly elevated risk of the usual community-acquired infections
    - Chronic viral infection
      - Usually caused by CMV, EBV, or hepatitis B or C
      - May develop the typical long-term sequelae of these viruses (e.g., liver disease with hepatitis or malignancy)
      - The combination of chronic infection and immunosuppression can yield other progressive diseases, such as CMV retinitis.
      - In primary varicella-zoster virus (VZV) infection: patients who are receiving immunosuppressive therapy and who were not previously exposed to the virus are susceptible to rapid dissemination and more significant illness, such as encephalitis, pneumonia, and hepatitis.
        - Therefore, admit VZV-seronegative patients exposed to VZV and treat them with intravenous VZV immune globulin.
      - Shingles, or reactivation of latent VZV, usually does not diseminate (see Image #26).
        - These patients should be considered for admission and treatment with IV acyclovir.
Patients in a chronic rejection state are at the highest risk for significant opportunistic infection because of their chronic, aggressive immunosuppression.

- **Fungal infections** include invasive candidiasis, aspergillosis, cryptococcosis, coccidioidomycosis, histoplasmosis, and blastomycosis.
  - Often present with respiratory illness, including focal or miliary pulmonary infiltrates
  - May also see upper respiratory tract illness (e.g., sinusitis) or gastrointestinal complaints
  - Treat with IV amphotericin B with care, because of its potential renal toxicity

- **Bacterial infections**
  - Can disseminate from initial infection to bacteremia or meningitis
  - Often begins as gastrointestinal illness, such as acute diverticulitis or invasive diarrheal illness from *Salmonella* or *Listeria*
  - Subacute pulmonary infections from *Nocardia asteroides* can occur.

- **Parasitic infections**
  - Subacute pneumonia secondary to *Pneumocystis jiroveci* is often seen as a co-infection with CMV.
    - May be prevented with prophylactic low-dose trimethoprim-sulfamethoxazole (TMP-SMX)
    - Treat infection with IV TMP-SMX and corticosteroids.
  - Toxoplasmosis may be reactivated (lies dormant in tissues), resulting in disseminated disease such as myocarditis, encephalitis, or brain abscess.
    - Treat for 4 weeks with IV sulfadiazine and pyrimethamine.
  - Hyperinfection from *Strongyloides stercoralis* can result in necrotizing hemorrhagic enterocolitis and hemorrhagic pneumonia.
    - May see resultant gram-negative bacteremia and meningitis

**Graft Rejection**

- Hyperacute rejection occurs during the perioperative period
  - Now rare, owing to careful donor-recipient matching
- Acute rejection occurs during the first months after transplant.
  - Will see signs of transplant organ insufficiency and general constitutional symptoms
  - Treatment is adjusting the patient’s immunosuppressive therapy.
- Chronic rejection occurs over a course of years.
  - Will see a gradual decline in function of the transplanted organ

**Drug Toxicity**

- **Cyclosporine** is a mainstay of transplant therapy.
  - Dose-related nephrotoxicity, which is additive to other nephrotoxic agents
  - May also induce renal-artery vasospasm and subsequent hypertension
  - Can cause hyperuricemia or worsen hyperlipidemia
  - Metabolism via cytochrome P-450 system; therefore, cyclosporine levels will be affected by drugs that inhibit or enhance this mechanism
Azathioprine
- Inhibits DNA and RNA synthesis, suppressing lymphocytes
- Acts as a bone marrow toxin with dose-related neutropenia
  - Goal is usually a WBC count of 4,000 to 6,000/mm³
- Causes hepatic dysfunction and GI illness

Corticosteroids
- Long-term sequelae include adrenal suppression, osteoporosis, cataracts, myopathy, and avascular necrosis
- Acute withdrawal can lead to an Addisonian crisis

Mycophenolate mofetil
- Similar action as azathioprine, but with a lower side-effect profile
- Often see gastrointestinal complaints, leukopenia, and thrombocytopenia

Antilymphocyte monoclonal antibody preparations
- Short courses of agents like OKT3 and antithymocyte globulin are used to reverse allograft rejection.
- Increased risk for opportunistic infection
- During administration, patients may experience headaches, fever, chills, hypotension, or pulmonary edema (if overhydrated).

Tacrolimus
- Actually a macrolide agent; has similar mechanism as cyclosporine
  - Thus, macrolide antibiotics should not be prescribed to these patients, because of potential toxicity.
- Used as either primary or rescue therapy for graft rejections
- Can cause nephrotoxicity or neurotoxicity, hyperglycemia, hyperkalemia, anorexia, nausea, and diarrhea
12.1 TOXIDROMES

Sympathomimetic Toxidrome
- Catecholamine excess
- Symptoms: increased heart rate (HR), increased blood pressure (BP), diaphoresis, dilated pupils, agitation, seizures, altered mental status, hyperthermia, piloerection, cardiac dysrhythmias
- Causes: cocaine, amphetamines, theophylline, over-the-counter (OTC) decongestants, caffeine
- Mimics: hypoglycemia, alcohol withdrawal, benzodiazepine withdrawal
- Treatment: benzodiazepines, hydration, cooling

Anticholinergic Toxidrome
- "Mad as a hatter, hot as a hare, dry as a bone, blind as a bat, and red as a beet"
- Symptoms: increased HR, possibly increased BP, dry flushed skin, dilated pupils, agitation, hyperthermia, hallucinations, seizures, urinary retention, sedation, coma
- Urinary retention and lack of diaphoresis distinguish this from sympathomimetic toxidrome
- Causes: antihistamines, jimsonweed, atropine, tricyclic antidepressants (TCAs), antiparkinsonian agents, some antipsychotics, skeletal muscle relaxants, certain mushrooms
- Treatment: supportive care with benzodiazepines, physostigmine (except TCAs), sodium bicarbonate for wide complex tachydysrhythmias

Cholinergic Toxidrome
- SLUDGE (salivation, lacrimation, urination, defecation, GI upset, emesis) plus “killer B’s” of bradycardia, bronchospasm, bronchorrhea
- DUMBBELS (diarrhea, urination, miosis/muscle weakness and fasciculations, bradycardia, bronchorrhea, emesis, lacrimation, salivation)
- Acetylcholinesterase inhibition leads to excess acetylcholine at four systems:
  - Parasympathetic system (muscarinic): leads to SLUDGE and bradycardia
  - Sympathetic (due to acetylcholine nicotinic receptors in sympathetic ganglia and adrenal medulla)
  - Skeletal muscle (nicotinic)
    - Muscle fasciculations, weakness, respiratory muscle paralysis leading to respiratory failure
  - Brain: leads to delirium, seizures, confusion

CHAPTER 12
Toxicologic Disorders

Nima Majlesi, DO, and Richard D. Shih, MD
TOXICOLOGIC DISORDERS

12.1 Causes: organophosphates, insecticides, physostigmine, pilocarpine, mushrooms, nerve agents

12.2 Treatment: secure airway/ventilation

- Muscarinic symptoms – atropine in incremental doses until airway secretions dry
- Nicotinic symptoms – pralidoxime (2-PAM)

Cholinesterase levels are not helpful in acute management.

Opioid Toxidrome

12.3 Symptoms: triad of miosis, altered mental status, respiratory depression

Causes: heroin, morphine, codeine, diphenoxylate, fentanyl

Treatment: naloxone, 0.05 to 0.1 mg IV, with incremental doses as needed until respiratory rate ≥12

Withdrawal: similar to sympathomimetic toxidrome, with diarrhea, abdominal cramps, piloerection, and yawning

Sedative/Hypnotic Toxidrome

12.4 Symptoms: mental status depression (spectrum from drowsiness to coma), ataxia, respiratory depression with large overdoses

Causes: benzodiazepines, barbiturates, γ-hydroxybutyrate (GHB)

Treatment: supportive care with airway management, flumazenil if known benzodiazepine overdose as single agent (controversial area due to risk of provoking seizures with flumazenil use in patients on chronic benzodiazepines)

Extrapyramidal Toxidrome

12.5 Symptoms: oculogyric crisis, rigidity, torticollis, trismus, dysphagia, dystonia, akathisia, neuroleptic malignant syndrome, tardive dyskinesia

Causes: haloperidol, phenothiazines, metoclopramide

Treatment: diphenhydramine, 1 to 2 mg/kg IV every 6 hours PRN; benztropine, 0.05 mg/kg IV every 12 hours PRN; benzodiazepines

12.2 GASTRIC EMPTYING

General

- Role is diminishing (controversial area)
- Induced emesis
- Gastric lavage
- Activated charcoal
- Cathartics
- Whole bowel irrigation

Induced Emesis (Ipecac)

- Role of this modality is decreasing dramatically (no longer recommended to be kept in the home by the American Academy of Pediatrics or for ED use by the American Academy of Clinical Toxicology)
- Effective drug removal with emesis decreases with delayed presentations (>30 min post-ingestion)
- Indications: no definite indications, but there are situations where this is more likely to be used:
  - Home therapy, especially with prolonged transport to medical care
  - Pediatrics
  - Toxins that do not bind well to charcoal
• Contraindications
  o Patients with loss of airway protection (or risk of loss)
  o Active or previous vomiting
  o Altered mental status
  o Seizure
  o Age <6 months
  o Certain ingestions, including caustics, TCA, hydrocarbons

Gastric Lavage
• Role of this modality is decreasing
• Labor-intensive procedure
  o 36 to 40 F orogastric tube for adults
  o Maneuvers to prevent aspiration (left lateral decubitus, Trendelenburg position, assistant to suction secretions, possible airway protection)
  o Indication: recent ingestion (<1 hour prior to arrival) of a potentially life-threatening agent such as a calcium channel blocker or TCA
  o Contraindications: large pills that do not fit through tube holes, nontoxic or mild severity ingestions, caustics, hydrocarbons

Activated Charcoal (AC)
• Recommended form of GI decontamination in most cases, if the procedure is done at all. The American Academy of Clinical Toxicology recommends against routine administration of AC, but states that AC may be considered if the patient ingested a toxic dose of a charcoal-adsorbed poison within the preceding hour.
• Mechanism of action
  o Direct adsorption
  o Adsorption of toxin from enterohepatic circulation
  o “Gut dialysis” – concentration gradient allowing free drug movement into gut
• Toxins that are poorly bound to charcoal:
  o Iron
  o Lithium
  o Heavy metals
  o Hydrocarbons
  o Caustics
  o Alcohols
• Indications for multiple-dose activated charcoal (MDAC)
  o Theophylline
  o Sustained-release drugs
  o Phenobarbital
  o Salicylates
  o Carbamazepine
Cathartics
- Role of this modality is decreasing
- Examples: magnesium citrate, sorbitol
- Not proven effective
- Avoid multi-dosing of cathartics, especially in association with MDAC (can cause dehydration, electrolyte abnormalities)
- Contraindications
  - Airway compromise
  - Ileus
  - Profuse diarrhea
  - Renal failure

Whole Bowel Irrigation
- “Ultimate cathartic:” rapid flushing of the GI tract with polyethylene glycol
- Labor intensive
- Relative indications
  - Agent not bound by activated charcoal
  - Sustained-release product
  - Body packer/stuffer
  - Patient becoming seriously toxic despite maximal treatment

12.3 TOXICOLOGIC GAPS

Anion Gap (AG)
- Useful for differential diagnosis in metabolic acidosis
- \[ AG = [\text{Na}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-]) \]
- Normal value = 8-16
- Mnemonic for causes of AG metabolic acidosis = MUD PILES/CAT
  - Methanol
  - Uremia
  - Diabetic ketoacidosis (DKA)
  - Paraldehyde
  - Isoniazid/iron
  - Lactic acid
  - Ethylene glycol
  - Salicylates
  - Cyanide/carbon monoxide
  - Alcoholic ketoacidosis (AKA)/alcohol intoxication
  - Toluene

Osmolar Gap (OG)
- Useful for evaluating possible toxic alcohol poisoning
- \[ OG = (\text{measured serum osmolality}) - (\text{calculated serum osmolality}) \]
- Calculated serum osmolality = \( 2 [\text{Na}^+] + [\text{glucose}]/18 + [\text{BUN}]/2.8 + [\text{ethanol}]/4.6 \)
- Normal level <10
• Absence of elevated OG dose not eliminate the possibility of toxic alcohol ingestion (lab variation or delayed presentation may have resulted in metabolism of osmotically active parent alcohol)
• Causes of elevated OG
  o Toxic alcohols (methanol, ethylene glycol, isopropanol)
  o Other ingestions: mannitol, propylene glycol, glycerol, and ethyl ether
  o Non-toxicologic causes of an elevated OG include hyperlipidemia, hyperproteinemia, DKA, alcoholic ketoacidosis, multiple organ system failure, chronic renal failure

12.4 SPECIFIC AGENTS

Acetaminophen
• Hepatic toxin
• Toxic metabolite (NAPQI) formed via P450 pathway
• Toxic ingestion: >150 mg/kg (>7.5 g in adult)
• Rumack-Matthew nomogram: assess risk of toxicity in acute single ingestion with serum level and known time of ingestion
• Initially, the patient can be entirely asymptomatic.
• Determining the toxicity risk associated with chronic ingestions and multiple ingestions is difficult.
• Acetaminophen toxicity may be greater at smaller doses in patients with chronic alcohol abuse.
• Antidote: N-acetylcysteine (NAC)
  o Oral NAC: loading dose of 140 mg/kg followed by 70 mg/kg every 4 hours
  o IV NAC: increased cost and increased risk of anaphylactoid reactions
  o Most beneficial if given within 8 hours; however, still efficacious after this initial period
  o Short-course NAC therapy is being increasingly utilized (<72 hours)

Alcohols (Ethanol, Ethylene Glycol, Isopropanol, and Methanol)
• Ethanol
  o Sedative/hypnotic toxidrome
  o Withdrawal: four syndromes on a continuum of severity and timing
    ▪ Alcoholic withdrawal tremulousness: sympathomimetic
    ▪ Alcoholic hallucinosis: typically visual and persecutory in nature
    ▪ Alcohol withdrawal seizures
    ▪ Delirium tremens: mental status change, autonomic instability, hyperthermia
  o Treatment: high dose of lipophilic benzodiazepines such as diazepam and midazolam with incrementally increasing doses as required for symptoms of withdrawal ("symptom trigger therapy")
  o Wernicke's encephalopathy (triad): caused by thiamine deficiency (a.k.a. dry beriberi)
    ▪ Manifestations
      □ Mental status change (confusion)
      □ Ophthalmoplegia: The most common ocular palsy is cranial nerve VI
      □ Ataxic gait
    ▪ Treatment: thiamine replacement
  o Korsakoff's psychosis
    ▪ Anterograde and retrograde amnesia
    ▪ Confabulation
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- Disulfiram reaction
  - Disulfiram, metronidazole, sulfonylureas, mushrooms (Coprinus), some cephalosporins
  - Acetaldehyde dehydrogenase enzyme inhibited
  - Acetaldehyde accumulates
  - Flushing, vomiting, headache

- Ethylene Glycol (EG)
  - Antifreeze
    - (+) anion gap, (+) osmol gap, (-) ketones
    - EG is metabolized to toxic metabolites: glycolic acid, oxalic acid, and others
    - Urine: may fluoresce under Wood’s lamp (test is neither sensitive nor specific)
    - Treatment: fomepizole (4-MP), ethanol, and/or hemodialysis
    - 4-MP and ethanol serve as competitive inhibitors for alcohol dehydrogenase, thus blocking EG’s metabolism to toxic metabolites.
    - Dialysis indications: renal insufficiency, EG level >25 mg/dL, severe acidosis

- Isopropanol
  - Rubbing alcohol
    - (-) anion gap, (+) osmol gap, (+) ketones
    - Gastritis, CNS depression, hypotension (severe cases)
    - Converted to acetone (strong acetone smell)
    - Treatment: supportive care with hemodialysis if indicated
    - Less toxic than ethylene glycol and methanol
    - Indications for hemodialysis (rare): refractory hypotension, serum levels >400 to 500 mg/dL

- Methanol
  - Windshield washer fluid, Sterno, shellacs
    - (+) anion gap (severe), (+) osmol gap, (-) ketones
    - Visual symptoms: “snowstorm” blurry vision, optic disc hyperemia
    - Toxic metabolite: formic acid
    - Treatment: fomepizole (4-MP), ethanol, and/or hemodialysis
    - 4-MP and ethanol serve as competitive inhibitors for alcohol dehydrogenase, thus blocking methanol’s metabolism to toxic metabolites.
    - Dialysis indications: renal insufficiency, methanol level >25 mg/dL, severe acidosis, visual symptoms

Anticoagulants
- Warfarin, superwarfarin (rat poison)
- Block vitamin-K–dependent activation of clotting factors II, VII, IX, X
- PT prolonged within 24 to 48 hours
- Treatment
  - No or mild bleeding: oral vitamin K; IV vitamin K carries risk of anaphylactoid reactions
  - Severe bleeding: fresh frozen plasma; can consider activated factor VII

Anticonvulsants
- Carbamazepine
  - Triad
    - Dizziness
    - Ataxia
    - Nystagmus
Toxicologic Disorders

• Severe overdose: cyclic coma, seizures, dysrhythmias (QRS widening due to structural similarities to TCAs)
  o Treatment: multiple-dose activated charcoal, hemoperfusion (rare), bicarbonate if QRS widened (rare)

• Phenytoin
  o Ataxia, nystagmus, slurred speech, cerebellar signs
  o Many drug interactions: may be the cause of elevated level
  o Dysrhythmias are rare except when rapidly administered IV, due to the drug’s vehicle (propylene glycol).
  o Treatment: supportive care, multiple-dose activated charcoal may increase drug clearance

Antidepressants

• Monoamine oxidase inhibitors (MAOI)
  o Block the breakdown of catecholamines (epinephrine, norepinephrine, serotonin)
  o May be asymptomatic initially
  o Severe overdose: catecholamine excess/sympathomimetic toxidrome, hypertensive crisis
  o Drug interactions
    ▪ Food: tyramine reaction (avoid aged meats/cheeses, alcohol, fava beans, alcohol)
    ▪ Serotonin syndrome (see below)
  o Treatment
    ▪ Aggressive management of hyperthermia with external cooling and possibly neuromuscular blockade with nondepolarizing agent
    ▪ Benzodiazepines as needed to control seizures
    ▪ Maintenance of hemodynamics with short-acting agents, as vital signs are often labile
    ▪ No β-adrenergic blockade
    ▪ Cyproheptadine can be considered in serotonin syndrome

• Selective Serotonin Reuptake Inhibitors (SSRI)
  o Inhibit presynaptic serotonin reuptake
  o Minimal or no cardiac effects (drug dependent)
  o Acute overdose: mild mental status depression, rarely life threatening
  o P450 metabolized (drug interactions)
  o Serotonin syndrome: idiosyncratic reaction caused by a drug or combination of drugs that increase serotonin neurotransmitter activity
    ▪ Increased muscle tone/rigidity; greater in lower than upper extremities
    ▪ Hyperthermia, autonomic dysfunction
    ▪ Culprit agents: meperidine, MAOI, dextromethorphan, SSRI, TCA
    ▪ Treatment options: see section on monoamine oxidase inhibitors, above

• Tricyclic Antidepressants (TCA)
  o Quinidine-like effects, type Ia antiarrhythmic effects, QRS widening
  o Anticholinergic (antimuscarinic effects only), GABA inhibition, peripheral α-blockade, decreased cardiac contractility
  o Main toxicity is cardiovascular (arrhythmias and hypotension) and seizures
  o Treatment
    ▪ Sodium bicarbonate for widened QRS (>100 mg); increases serum pH and decreases cardiac toxicity
    ▪ Multiple-dose activated charcoal
    ▪ 3% saline for severe cases
    ▪ Norepinephrine for hypotension refractory to IV fluids
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- Type Ia, Ic, and III anti-arrhythmics are contraindicated, as are β-blockers and calcium channel blockers.
- Flumazenil is contraindicated.

β-Blockers
- Bradycardia and hypotension are the most important effects; hypoglycemia and depressed mental status may occur
- Treatment
  - Atropine (unlikely to be effective)
  - Glucagon
  - Calcium
  - Pacemaker for refractory bradycardia
  - Pressors (epinephrine)
  - Insulin/glucose as a pressor agent for recalcitrant hypotension (controversial)
  - Intra-aortic balloon pump

Calcium Channel Blockers
- Bradycardia, AV blocks, and hypotension are the most important consequences of overdose.
- Treatment
  - Atropine (unlikely to be effective)
  - Calcium (calcium chloride if central venous access is in place; otherwise, calcium gluconate)
  - Glucagon
  - Pacemaker (may improve HR, but unlikely to improve BP)
  - Pressors (norepinephrine or dopamine)
  - Insulin/glucose as a pressor agent for recalcitrant hypotension
  - Intraaortic balloon pump

Carbon Monoxide (CO)
- Binds to hemoglobin, myoglobin, cytochrome, and other sites
- Acute effects predominantly caused by hypoxia from hemoglobin binding and CO binding to cytochromes in oxidative phosphorylation
- CO-Hb half-life
  - Room air: 5 hours
  - 100% oxygen: 1.5 hours
  - Hyperbaric oxygen (HBO): 0.5 hours
- Falsely high readings with pulse oximetry (device interprets COHb the same as oxyhemoglobin)
- Children are at greater risk for acute toxicity, but their long-term outcomes may be better.
- Fetal Hb has higher CO affinity; therefore, the fetus is at higher risk
- Indications for hyperbaric oxygen therapy
  - Carboxyhemoglobin level >25%
  - Mental status change, seizure, coma
  - Dysrhythmia, cardiac ischemia, history of syncope
  - Hypotension
  - Pregnancy with COHb >20% or signs of fetal distress
  - Abnormal cerebellar examination
Caustics
- Acids: coagulation necrosis; gastric burns more common, but esophageal burns can occur
- Alkali: liquefactive necrosis; oral and proximal esophageal burns are more common
- Airway at risk: consider prophylactic intubation
- The absence of oral burns does not exclude significant GI injury.
- The presence of two or three symptoms, including stridor, vomiting, and drooling, may be predictive of the degree of injury.
- Treatment
  - Dilution – not recommended
  - Neutralization – not recommended
  - Steroids – controversial
  - Surgery for signs of perforation (free air, peritonitis, etc.)
  - Supportive/nutritional support
  - Endoscopy to assess level of injury

Clonidine
- Centrally acting $\alpha_2$-agonist
- $\alpha_2$-Receptors are presynaptic and decrease CNS sympathetic output.
- Mental status change (usually sedation), hypotension, miosis, bradycardia, respiratory depression
- Can mimic opioid overdose
- Treatment
  - Naloxone may reverse mental status depression (high dose, 0.1 mg/kg)
  - Pressors (norepinephrine or phenylephrine)

Cyanide (CN)
- Chemical plants, labs, jewelry making, fires (burning plastic), amygdalin (apricot pits), prolonged nitroprusside infusions
- Inhalational toxin primarily, but GI and dermal exposures are possible
- Bitter almond smell
- CN binds to cytochrome oxidase, the enzyme responsible for the final step of oxidative phosphorylation. Disruption of cellular energy production results, which leads to cellular hypoxia and lactic acidosis.
- Treatment: CN antidote kit
  - Amyl nitrite pearls (inhaled)
  - Sodium nitrite IV
  - Sodium thiosulfate IV
- Nitrite administration is designed to produce methemoglobin.
- (Met-Hb): $-CN + Met-Hb \rightarrow$ cyano-Met-Hb (non-toxic)

Digitalis
- Plant sources: Digitalis purpurea (foxglove), oleander
- Inhibits sodium-potassium ATPase pump
- Symptoms: nausea/vomiting, visual color aberrations (especially yellow-green), cardiac dysrhythmias
- Dysrhythmias: paroxysmal atrial tachycardia with block, bidirectional VT, slow atrial fibrillation, junctional tachycardia, VF, AV blocks, and many others
- Most common dysrhythmias: PVCs
• Poor prognosis: potassium >5 mEq/L (acute ingestions)

• Treatment
  o Atropine (for bradycardia and high-degree AV block)
  o Pacemaker (for bradycardia and high-degree AV block) – may increase risk of dysrhythmia due to ventricular irritability
  o Digoxin-specific Fab fragments – treatment of choice
  o Indications for digoxin immune Fab
    ▪ Malignant dysrhythmias
    ▪ Potassium >5.0 mEq/L
    ▪ Digoxin level >15 ng/mL acutely or >10 ng/mL 6 hours after ingestion
  o Digoxin immune Fab dosing:
    ▪ In average acute ingestion administer 10 vials over 30 minutes
    ▪ In cardiac arrest administer 20 vials by IV bolus
    ▪ In average chronic toxicity administer 4-6 vials
    ▪ Consult the package insert for dosing based on amount ingested or digoxin levels
  o Calcium administration is contraindicated.

Drugs of Abuse
• Amphetamines
  o Sympathomimetic toxidrome: increased HR, increased BP, diaphoresis, dilated pupils, agitation, CNS excitation, hyperthermia
  o Treatment: sedation (benzodiazepines), cooling measures if the patient is hyperthermic
  o Hallucinogenic amphetamines: MDMA (Ecstasy), MDA, MDEA
    ▪ Hyperthermia, hyponatremia (with MDMA)

• Cocaine
  o Sympathomimetic toxidrome: increased HR, increased BP, diaphoresis, dilated pupils, agitation, CNS excitation, hyperthermia
  o Treatment: sedation (benzodiazepines), cooling measures if the patient is hyperthermic
  o Cocaine chest pain
    ▪ Hypertensive crisis: avoid β-blockers, which may cause unopposed α-effect and worsen vasoconstriction/ischemia/hypertension

• Hallucinogens
  o LSD, mescaline, psilocybin mushrooms
  o Serotonin-like agents (agonist at 5-HT22 receptors)
  o “Bad trip”: anxiety, paranoia, agitation, hallucinations, psychosis
  o Treatment
    ▪ Quiet environment, reassurance
    ▪ Sedation with benzodiazepines if extremely agitated
    ▪ Haloperidol – second-line agent

• Phencyclidine (PCP)
  o Acute effects produce dissociative anesthesia similar to ketamine.
  o Upon awakening, patients develop emergence phenomenon, which causes sympathomimetic symptoms.
  o Rotary nystagmus
  o Agitation, violent/bizarre behavior, seizure, hyperthermia, elevated creatine kinase
Treatment: sedation (benzodiazepines), haloperidol

Do not acidify the urine—acid urine pH does not assist much in drug elimination and may promote myoglobin precipitation in the renal tubules

- Opioids
  - Triad: miosis, altered mental status, respiratory depression
  - Heroin, morphine, codeine, diphenoxylate, fentanyl
  - Treatment: naloxone (clinical duration of action ~30 minutes)
  - Synthetic opioids may require large-dose naloxone (repeated 1- to 2-mg doses): fentanyl, methadone, propoxyphene, pentazocine
  - Severe overdose: acute lung injury/ARDS

- Hydrocarbons
  - Aspiration pneumonitis after ingestion is common due to several characteristics of hydrocarbons:
    - Low viscosity
    - High volatility
    - Low surface tension
  - Gastric emptying
    - Do not use ipecac
    - Lavage only if hydrocarbon has another dangerous toxin associated with it (CHAMP):
      - Camphor
      - Halogenated hydrocarbons
      - Aromatic hydrocarbons
      - Metal-containing hydrocarbons
      - Pesticide-containing hydrocarbons
  - Hydrocarbon aspiration: antibiotics and steroids are not useful
  - Medically clear ingestions if the patient is asymptomatic and has a normal chest film at 6 hours.
  - Inhalational hydrocarbon
    - "Bagging" (pouring a hydrocarbon into a plastic bag and inhaling), "huffing" (inhaling through a cloth soaked with a hydrocarbon), "sniffing" (inhaling directly from an open container of a hydrocarbon)
    - Recreational abuse
    - Sudden sniffer's death: ventricular arrhythmia caused by cardiac sensitization by endogenous catecholamines
  - Lindane
    - Chlorinated hydrocarbon
    - Mental status change and seizures
    - Toxicity can occur from large cutaneous exposure (especially in children)

Hydrogen Fluoride (HF)
- Jewelers, metal cleaning products, glass etching, petrochemical industry
- Toxin binds strongly to Ca²⁺ and Mg²⁺
- Cutaneous exposures cause severe pain with minimal or no apparent burn injury.
- Large cutaneous exposures and ingestions can cause systemic toxicity: hypocalcemia, hypomagnesemia, arrhythmias, hyperkalemia
- Treatment: calcium gluconate (NOT calcium chloride!) gel applied topically, intradermal infiltration, or arterial infusion
Iron (Fe)
- Ferrous sulfate (20% elemental Fe), ferrous fumarate (33% elemental Fe), ferrous gluconate (12% elemental Fe)
- Toxic dose is calculated based on elemental iron content: >20 mg/kg; severe toxicity at >60 mg/kg elemental iron
- Iron toxicity
  - Local GI toxicity: abdominal pain, diarrhea, vomiting, GI bleeding
  - Vasodilation: hypotension
  - Uncoupling of oxidative phosphorylation leads to shock and lactic acidosis (wide anion gap metabolic acidosis)
  - Coagulopathy, hepatic injury, renal failure in severe toxicity
  - Pyloric scarring, possibly leading to delayed gastric outlet obstruction
- KUB: pills are radiopaque
- TIBC is falsely elevated and not useful in acute poisoning; follow serum iron levels instead
- Treatment
  - Activated charcoal does not bind iron.
  - Whole bowel irrigation
  - Deferoxamine chelation
- “Vin rose colored urine”: the urine from toxic patients will take on a “red wine” appearance after deferoxamine chelation

Isoniazid (INH)
- Inhibits production of GABA via vitamin B6 depletion
- Seizures, coma, acidosis
- Must consider in cases of refractory seizures
- Antidote: pyridoxine (vitamin B6)

Lithium
- GI symptoms (nausea/vomiting/diarrhea), tremor, ataxia, hyperreflexia, dehydration due to diabetes insipidus
- Severe toxicity manifests as encephalopathy and seizures
- Excretion is almost entirely renal – impaired if patient is dehydrated
- Treatment
  - Fluid hydration with normal saline (to correct dehydration and facilitate renal excretion of lithium)
  - Hemodialysis
- Indications for dialysis
  - Renal failure
  - Inability to fluid hydrate (i.e., CHF, pulmonary edema)
  - CNS toxicity (altered mental status, coma, or seizures)
  - Symptomatic patients with lithium level >4 mEq/L in acute overdose

Methemoglobinemia
- Toxins that have oxidative properties induce this condition: nitrites, nitrates, dapsone, phenazopyridine, benzocaine
- Normal Fe²⁺ in deoxyhemoglobin is oxidized to Fe³⁺, converting Hb to Met-Hb
- Met-Hb does not bind to oxygen and results in decreased oxygen content in blood
- Met-Hb has a blue color and causes a cyanosis-like appearance.
- Chocolate brown blood
• Pulse oximetry is not reliable (reads ≈85%); need co-oximetry analysis
• Treatment: methylene blue (promotes conversion of Met-Hb to normal Hb) – but should be used with caution in G6PD-deficient individuals

Neuroleptics
• Haloperidol, droperidol, chlorpromazine
• Extrapyramidal symptoms (EPS)
  o Dystonic reaction – torticollis, trismus, oculogyric crisis
  o Akathisia – anxiety and restlessness
  o Tardive dyskinesia – choreoathetoid movements of face or tongue
  o Neuroleptic malignant syndrome (NMS)
• Treatment: diphenhydramine, benztropine
• NMS: rare cause of hyperthermia associated with muscle rigidity and autonomic dysfunction
  o Treatment
    • Cooling measures and hydration
    • Muscle relaxants (benzodiazepines)
    • Dantrolene can be used (not a specific antidote in this case)

Oral Hypoglycemics
• May have delayed onset of hypoglycemia several hours after ingestion
• Concern is with sulfonylureas and meglitinides (nateglinide, repaglinide)
• May cause prolonged hypoglycemia
• All exposures (even if asymptomatic) need to be observed for at least 24 hours
• If hypoglycemia develops, treatment is glucose and octreotide

Salicylates
• Aspirin, oil of wintergreen (methyl salicylate), bismuth subsalicylate
• Respiratory alkalosis (from central respiratory stimulation) and metabolic acidosis (due to uncoupling of oxidative phosphorylation) and metabolic alkalosis (from volume contraction)
• Anion gap acidosis
• Acute overdose: nausea/vomiting, tinnitus, respiratory alkalosis, metabolic acidosis, cerebral edema, seizures
• Can cause acute lung injury/ARDS
• Uncouples oxidative phosphorylation
• (+) Urine ferric chloride reaction: will turn purple
• (+) Urine ketones in severe cases
• The Done nomogram is no longer used.
• Chronic poisoning: patient is often more toxic at same serum level compared with acute poisoning
• Treatment
  o Urinary alkalinization to a pH >7.5 with bicarbonate to increase urinary excretion of salicylate via “ion trapping”
  o K+ administration to correct losses once urine output is adequate in order to allow urinary alkalinization
Hemodialysis

- Indications:
  - Renal failure
  - Pulmonary edema
  - Coma, altered mental status, or seizure
  - Salicylate level >100 mg/dL in acute poisoning
  - Severe cardiac toxicity
  - Severe acidosis
  - Rising salicylate levels despite urinary alkalinization

- Oil of wintergreen contains very large amounts of salicylate and is extremely toxic in small doses.

Sedative Hypnotics

- General
  - Triad: mental status depression (spectrum from drowsiness to coma), normal vital signs, respiratory depression with large doses
  - Treatment: supportive care

- Barbiturates
  - Pentobarbital and secobarbital: short acting, hepatic metabolism and high lipid solubility, not dialyzable
  - Phenobarbital: long acting, 25% renal excretion, weak acid enabling ion trapping for enhanced renal elimination (urine alkalinization), can be dialyzed (rarely done)
  - Severe overdose: cutaneous bullae (“barb blisters”) – not specific for barbiturate toxicity; likely pressure-related lesions on dependent body parts due to coma

- Benzodiazepines (BZDs)
  - Flumazenil use is controversial because its half-life is shorter than that of most BZDs, possibility of unknown polydrug ingestion (TCAs), and ability to manage BZD toxicity adequately with supportive care alone
  - Contraindications to flumazenil use
    - Co-ingestion of TCAs, INH, MAO-I, bupropion, cocaine, lithium, or any other drug known to lower the seizure threshold (may precipitate seizures that are difficult to control)
    - History of seizures
    - High risk for benzodiazepine withdrawal

- Chloral Hydrate
  - Additive effects when taken with alcohol (“Mickey Finn”)
  - Overdose: can cause cardiac sensitization and ventricular tachycardia

- Methaqualone
  - Seizures, GI bleeding

- γ-Hydroxybutyric Acid (GHB)
  - Used as a sports supplement and as a recreational and general anesthetic and for chemical submission (“date rape” drug)
  - GABA receptor agonist
  - Clinical manifestations: hallucinations, disorientation, miosis, lethargy, stupor, and coma
  - Hypoventilation is the most common cause of death.
  - Violent arousal is common, with self-extubation
  - Treatment: supportive care
Strychnine
- Competitive antagonist of glycine, a postsynaptic inhibitory neurotransmitter of the spinal cord
- Rapid onset of muscle twitching, extensor muscle spasms, opisthotonos, trismus, facial grimacing
- Treatment: muscle relaxation (benzodiazepines, paralytics), supportive care; watch for rhabdomyolysis

Theophylline
- Sympathomimetic toxidrome
- GI effects (severe vomiting), tachycardia, CNS (seizures), cardiac (dysrythmias – PAC, MAT, AF, PVC, VT), hypokalemia (due to excess catecholamines)
- Chronic ingestions: more severe toxicity at lower serum levels
- Treatment
  - Antiemetics (high-dose metoclopramide, ondansetron)
  - Multiple-dose activated charcoal
  - Hemoperfusion/hemodialysis
- Indications for hemoperfusion/hemodialysis
  - Severe toxicity (malignant dysrhythmia, seizure)
  - Acute overdose: theophylline level >100 µg/mL
  - Chronic overdose: theophylline level >60 µg/mL

Mushroom Poisoning
- Difficult to manage because of the prevalence of mixed mushroom ingestions
- Send gastric contents for identification.
- Most ingestions with early onset (<2 hours after ingestion) of symptoms have a benign course.
- The three most toxic classes have delayed onset (>6 hours after ingestion) of initial GI symptoms.
  - Orellanine (*Cortinarius* spp.)
    - Cause delayed renal toxicity
    - Hemodialysis and possibly renal transplantation
  - *Gyromitra* species – resemble a brain in appearance
    - Hepatic toxicity and refractory seizures due to monomethyl hydrazine (similar to INH toxicity)
    - Benzodiazepines and pyridoxine
  - Cyclopeptides (Amatoxin) – *Amanita phalloides* is a common example
    - Responsible for most mushroom deaths in the United States
    - Hepatotoxicity – coagulopathy, encephalopathy
    - Treat with activated charcoal (MDAC), IV penicillin G, and N-acetylcysteine
13.1 HYPOGLYCEMIA

Symptoms
- Diaphoresis and tremulousness
- Tachycardia
- Confusion and altered level of consciousness
- Seizure
- Symptoms may be masked by β-blocker therapy.

Etiologies
- Common etiologies include oral hypoglycemic agents and insulin.
- Post-prandial hypoglycemia may be an early marker of new-onset diabetes.
- Other medications that can cause hypoglycemia include barbiturates, salicylates, and alcohol.
- Endocrine disorders that can cause hypoglycemia include hypothyroidism, adrenal insufficiency, and rarely, an insulinoma.
- Other causes include liver disease (due to decreased glycogen stores), pituitary insufficiency, starvation, and chronic renal failure.

Pathophysiology
- Maintenance of normal serum glucose depends on the counterregulatory hormones: epinephrine and norepinephrine, glucagon, growth hormone, and glucocorticoids.

Treatment
- Adults: 1 g/kg D50 (50% dextrose in water) given IV.
- The usual starting dose is an "amp" of D50 (50 grams).
- Can be given as a continuous infusion of D5 or D10.
- Glucose can also be given orally.
- Glucagon in a dose of 1 or 2 mg can also be given IM or IV to diabetics and to patients with no intravenous access.
- Warning: Glucagon may not be effective in patients with depleted glycogen stores, such as alcoholics and patients with liver disease.
- Consider administration of steroids if adrenal insufficiency is suspected.
Octreotide: can be given subcutaneously in a dose of 50 micrograms every 12 hours for two doses
  - Decreases insulin secretion from the pancreas
  - Considered a good antidote for oral hypoglycemic agent overdose
- Children: the intravenous route should be used when oral treatment is not an option
- Neonates: 5 to 10 cc/kg D10 (D25 and D50 are hyperosmolar and may cause phlebitis and tissue necrosis)
- Infants and children: 2 to 4 cc/kg D25
- Establish a maintenance drip after initial treatment.

Disposition
- Depends on many factors, including comorbid diseases, the cause of the hypoglycemic episode, the presence of liver or kidney disease, the patient's social situation, acute ingestion
- Admit the patient if the patient ingested or is currently taking oral hypoglycemic agents
- A single oral hypoglycemic tablet ingested by a child can produce life-threatening hypoglycemia.

13.2 DIABETIC KETOACIDOSIS (DKA)

Causes
- Lack of insulin (noncompliance)
- Infection
- Myocardial infarction/ischemia
- Surgery
- Pregnancy
- Trauma
- Various endocrine diseases such as hyperthyroidism

Clinical Presentation
- Common complication of diabetes; may be the first manifestation of the disease in as many as 30% of patients
- Initially, only polydipsia and polyuria may be evident.
- Development of anion-gap metabolic acidosis and compensatory respiratory response

Treatment
- Summary: IV fluids (IVFs), insulin, and potassium
- Serum glucose, potassium, and other electrolytes should be monitored every few hours.
- IVFs (in large volumes); 1 to 2 liters in the first few hours; normal saline for initial fluid resuscitation
  - Many patients with DKA will have a 6- to 8-liter volume depletion.
  - Can switch to ½ NS after several liters of NS have been given
  - Add glucose to the IVFs when the serum glucose concentration falls below 200 to 250 mg/dL.
- Insulin: an initial bolus is not indicated
  - Start a continuous infusion at a dose of 0.1 unit/kg/hr.
- Potassium: start replacement early.
  - If a patient with DKA arrests during therapy, consider that the serum potassium concentration has fallen precipitously.
  - Start replacing potassium during the initial resuscitation if the patient is already hypokalemic.
  - Add potassium to the IVF if the serum potassium level is normal during the early phases of treatment (after urine output begins), and follow with serial potassium measurements if the patient was initially hyperkalemic.
- Phosphate: typically not indicated unless the serum level is <1 mg/dL.
- Bicarbonate: currently no indication for bicarbonate unless the pH is very low (<7.0) or if the patient has concomitant lactic acidosis and refractory hypotension.
Pathophysiology
- Underlying mechanism behind DKA is lack of insulin (either secretion or production) and excess circulating counterregulatory hormones.
- This results in hyperglycemia, anion-gap metabolic acidosis, and ketonemia.
- All three of these result in significant volume depletion and, potentially, shock. See diagram below.

**Testing for ketones**
- The three primary ketones are acetoacetate, β-hydroxybutyrate, and acetone.
- Current serum and urine assays detect acetoacetate only.
- In a volume-depleted patient, the equilibrium of these products shifts toward the undetectable form of β-hydroxybutyrate.
- As therapy is instituted and the patient's volume status improves, more acetoacetate is produced and the ketone test becomes more positive.

13.3 HYPEROSMOLAR NON-KETOTIC COMA

**Causes**
- Infection (one of the most common precipitants), pneumonia, urinary tract infection (UTI), pyelonephritis, sepsis
- Myocardial infarction or ischemia: get an ECG early
- Stroke
- Renal failure
- Head injury (subdural hematoma)

**Pathophysiology**
- Hyperglycemia and hyperosmolality without acidosis
- Patients usually have enough endogenous insulin to prevent the development of ketosis.
- It takes much lower levels of insulin to shut off ketogenesis than to increase cellular uptake of glucose.
- Enough stress, however, can overwhelm the system and actually lead to DKA.
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Presentation
• Typically an older patient who presents with lethargy and evidence of volume depletion
• Serum glucose tends to be much higher (>1000 mg/dL) than in patients with DKA
• Pearl: may look exactly like a cerebrovascular accident

Laboratory
• Serum glucose concentration is usually >600 mg/dL.
• Serum osmolality is usually >350 Osm/kg. There are two ways to determine this value:
  o Calculated osmolality derived from equation below
  o Machine-derived serum osmolality in the laboratory
  o Any difference in the calculated versus machine-derived osmolality might indicate the presence of alcohol or some other substance.
• Absence of ketones (unless DKA has developed)
• Normal pH
• Serum osmolality – expect to use this equation on a toxicology question:
  \[ 2 \times [\text{Na}] + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8} \]

Treatment
• Search for the inciting event: MI, infection, stroke
• IVF: start with NS (especially if unstable/shock) regardless of serum Na concentration
• Add glucose to the IVF when the serum glucose concentration is <200 to 250 mg/dL.
• The average fluid deficit in this disorder is in the range of 8 to 12 liters.
• Insulin drip: 0.1 units/kg/hour while monitoring glucose and electrolytes every 1 to 2 hours
• Potassium replacement

13.4 ALCOHOLIC KETOACIDOSIS

Occurs secondary to heavy ethanol use and decreased food intake, which leads to increased production of ketoacids

Laboratory
• Usually shows an anion-gap metabolic acidosis
• Blood glucose concentration is usually not very elevated (usually <250 mg/dL).
• Blood alcohol level is usually low or undetectable.

Treatment
• Administer IV fluid containing both normal saline and glucose (to shut off ketone production): D5 NS or D5 ½ NS
• Insulin is NOT indicated.
• Give thiamine, 100 mg, with glucose administration to malnourished patients; metabolism of glucose uses thiamine, which may result in Wernicke's encephalopathy (confusion, ataxia, nystagmus, and ophthalmoplegia) if the patient is already thiamine deficient
13.5 LACTIC ACIDOSIS

Lactate is the normal product of anaerobic metabolism.

The diagnosis is usually made when the serum lactate is \( >1.5 \) mEq/L in association with an anion-gap metabolic acidosis.

Differential diagnosis of an elevated anion gap and some associated pearls (mnemonic CATMUDPILES):

- CO (by generation of lactic acid), cyanide poisoning
- Alcoholic ketoacidosis
- Toluene (the “glue sniffers”; causes an anion-gap metabolic acidosis and cerebellar ataxia)
- Methanol: consider if the patient has visual complaints. Anion gap is typically very high.
- Uremia
- Diabetic ketoacidosis (hyperglycemia, ketonemia, and anion-gap metabolic acidosis)
- Phenformin (no longer used in the United States; newer metformin has been associated with anion gap metabolic acidosis)
- INH and iron
- Lactic acidosis (multiple causes)
- Ethylene glycol (calcium oxalate stones on urinalysis, mouth lights up with Woods lamp)
- Salicylates

There are multiple causes of lactic acidosis. Here are the ones you should keep in mind:

- Type A: seen with inadequate tissue perfusion, alcoholic ketoacidosis (AKA), sepsis, and septic shock
- Type B1: medical disorders, such as renal failure, liver failure, lymphoma, seizures, infections, Reye’s syndrome, underlying thiamine deficiency
- Type B2: drugs and toxins, such as ethanol (most common cause), methanol, epinephrine and other catecholamines, and metformin; consider this one in a diabetic on multiple medications who presents with unexplained lactic acidosis
- Type-B3: inborn errors of metabolism

Treatment of Lactic Acidosis

- Involves first identifying and treating the inciting event
- Traditionally, intravenous bicarbonate (sodium bicarbonate) has been recommended.
- Although the use of bicarbonate is still somewhat controversial, it has been shown to improve serum pH and increase vascular responsiveness to vasopressors.

13.6 HYPOTHYROIDISM (MYXEDEMA COMA)

Myxedema coma is a rare, life-threatening form of hypothyroidism.

It usually occurs in patients with untreated or undiagnosed hypothyroidism.

Pathophysiology

- Occurs when T3 and T4 production is reduced or when the pituitary decreases its production of TSH (thyroid-stimulating hormone)
- All organs are dependent on circulating thyroid hormones.
- Deficiency of T3 and T4 leads to a generalized slowing of all bodily functions
Causes of Hypothyroidism
- Primary thyroid failure (autoimmune thyroid disease): Hashimoto's thyroiditis
- Prior treatment of hyperthyroidism with radiation or surgical ablation
- Rare: iodine deficiency, antithyroid drugs (e.g., lithium)
- Secondary hypothyroidism: pituitary disease, postpartum pituitary necrosis (Sheehan's syndrome), and sarcoidosis

Causes of Myxedema Coma (Severe Hypothyroidism)
- The key inciting event is a stressful condition.
- Common stressors include the following:
  - Congestive heart failure
  - Infections (pulmonary infections are the most common precipitants)
  - Cold exposure
  - Medications (particularly phenothiazines, narcotics, β-blockers, and sedatives)
  - Trauma
  - Stroke
  - Metabolic derangements such as hypoglycemia, hypoxia, and hyponatremia

Clinical Presentation of Myxedema Coma
- A patient with known hypothyroidism (or undiagnosed) presents with unexplained hypothermia, hypotension, and bradycardia.
  - Hypoxemia and hypercapnia are common.
  - Look for a thyroidectomy scar and ask about a history of thyroid disease.
  - Ask about symptoms of hypothyroidism.
  - Things to look for:
    - Bradycardia (may also see low-voltage ECG). One of the classic electrocardiographic findings associated with hypothyroidism is diffuse T-wave inversions in almost all leads.
    - Cardiomegaly on chest film, since many of these patients have pericardial effusions caused by hypothyroidism.
    - Thin eyebrow hair and scant body hair
    - Puffy eyes
    - Non-pitting edema
    - Classic association: hypothyroidism and carpal tunnel syndrome

Lab Findings
- Hyponatremia, elevated CPK and LDH
- May see hypocalcemia in patients who have undergone thyroidectomy
- Elevated WBC count with possible left shift
- Samples should be sent for measurement of TSH and free T4, but the results are typically unavailable during the ED evaluation.

Treatment
- Supportive measures: IVF, glucose, antibiotics for infection
- Treatment of hyponatremia
- Thyroid hormone replacement: the drug of choice for myxedema coma is intravenous thyroxine in a dose of 400 to 500 micrograms followed by 50 to 100 mcg/day, depending on free T4 levels
- Patients should be given empiric hydrocortisone in a dose of ~100 mg q8h to prevent adrenal failure (should be given before thyroid replacement)
13.7 HYPERTHYROIDISM (THYROID STORM)

Pathophysiology

- Undiagnosed or under treated hyperthyroidism + a stressful event lead to severely increased levels of circulating thyroid hormone and clinical symptoms of organ hyperfunction.

Causes

- Thyroid storm is usually precipitated by a stressful event and usually occurs in someone with Grave's disease (hyperthyroidism caused by anti-TSH receptor antibodies)
  - Infections, particularly pulmonary infections
  - Overtreatment with thyroid hormone replacement therapy
  - Administration of radioactive iodide
  - Radiointrogen agents
  - Overdose on thyroxine
  - Stroke
  - Pulmonary embolism

Clinical Presentation

- Fever, mental status changes, tachycardia, and a widened pulse pressure
- Patient may appear to be more of a psychiatric case than medical.
- Development of diarrhea in a patient with hyperthyroidism may be the signal of impending storm.
- Other physical examination findings may include a palpable goiter, exophthalmos, stare/lid lag, or warm, velvety palms.
- Most patients will be febrile and have evidence of CNS dysfunction.
- Differential diagnosis includes meningitis/encephalitis, toxidromes, heat stroke, and sepsis.

Pearl: Many patients present with atrial fibrillation and may have signs/symptoms of congestive heart failure.

Treatment

- Based on clinical suspicion, as the results of thyroid function studies are often unavailable to help guide decisions
- Supportive care (airway support/oxygen, IV if not in CHF, fever-control measures)
- Treatment of possible atrial fibrillation
- IV glucocorticoids may help prevent adrenal hyperfunction and subsequent crisis. Most endocrinologists recommend steroids. Dexamethasone is reasonable, as it also has the effect of decreasing the conversion of T4 to T3.
- Antithyroid therapy
  - Propylthiouracil (PTU) and methimazole both block the synthesis of new thyroid hormone but not the release of already formed T4 and T3.
  - PTU has the advantage of inhibiting peripheral conversion of T4 to T3.
  - Doses: PTU (only oral), 600 to 1000 mg initially, followed by 100 to 300 q4–6h. Methimazole is given once a day in a dose of 30 to 60 mg after an initial 90- to 120-mg oral load.
  - Iodide: inhibits release of stored (pre-formed) hormone.
    - Should be given at least 1 hour after antithyroid medications have been given to prevent iodide from being incorporated into new thyroid hormone.
    - Can be given as a saturated solution of iodine (SSKI) or sodium iodide
  - β-Blockers: decrease the stimulation of adrenergic receptors that cause tachycardia and other sympathetic hyperactivity symptoms.
    - Propranolol can be given in a dose of 1 mg every 10 to 15 minutes.
    - Has the advantage of being able to inhibit peripheral conversion of T4 to T3
Drugs to avoid (may worsen the hyperthyroid state): aspirin (increases T4 and T3), sedatives, and atropine

Apathetic Thyrotoxicosis
- A form of hyperthyroidism in which the typical hyperkinetic manifestations are lacking
- Patients may present with depression, lethargy, weakness, and/or excessive weight loss.
- Some patients present only with findings of atrial fibrillation and CHF.

13.8 ADRENAL INSUFFICIENCY/CRISIS

Pathophysiology
- Three zones of the adrenal cortex (glomerulosa, fasciculata, and reticularis).
- The cortex makes glucocorticoids and mineralocorticoids.
- The major glucocorticoid, cortisol, is released when ACTH made in the pituitary is released and stimulates the adrenal cortex to make and release it.
- The major mineralocorticoid is aldosterone.
- The medulla makes catecholamines (epinephrine and norepinephrine).
- Adrenal insufficiency results when the amount of circulating cortisol is reduced.

Causes
- Primary adrenal failure (chronic autoimmune adrenal insufficiency is called Addison’s disease)
- May be idiopathic (diabetes, Grave’s disease, pernicious anemia)
- Secondary to infectious/infiltrative disorders such as TB, fungal infections, sarcoidosis, AIDS/CMV adrenalitis, hemochromatosis, and metastatic cancer
- The most common cause overall is autoimmune adrenalitis.
- Secondary adrenal insufficiency may be caused by pituitary disease (tumor, infarction).
- One of the most common causes of adrenal insufficiency is iatrogenic adrenal suppression from chronic steroid use.

Clinical Presentation
- Patients with adrenal insufficiency from whatever cause may present with adrenal crisis if subjected to a significant stressor.
- Patients with adrenal crisis appear very ill, with hypotension, tachycardia, and unresponsiveness to intravenous fluids.
- Patients may initially be orthostatic and develop shock.
- Gastrointestinal symptoms are frequently present (nausea, vomiting, and abdominal pain), as is fever.
- Hypoglycemia is characteristic.
- The classic electrolyte picture consists of hyponatremia and hyperkalemia.
- In its chronic form, adrenal insufficiency may lead to clinical manifestations of weakness, lethargy, and/or fatigue.
- Anorexia, nausea, and vomiting are quite common. Patients frequently present with volume depletion and hyperkalemia.
- Cutaneous findings are common and include hyperpigmented skin, especially in crease marks (hands) and at pressure points.
- Patients may present with hyponatremia, hyperkalemia, and hypoglycemia.
Treatment of Adrenal Crisis
- Intravenous fluids: D5 normal saline initially to replace glucose and volume depletion. Patients may be several liters negative.
- Serum glucose should be measured frequently.
- Glucocorticoid therapy with hydrocortisone (has more of an effect on vascular tone than dexamethasone) in a dose of 100 mg IVP then 100 mg q6-8h
- Glucocorticoids are necessary to maintain vascular tone.

Treatment of Addison's disease (Chronic Adrenal Failure)
- Glucocorticoid therapy with oral hydrocortisone or prednisone (each one given BID)
- Mineralocorticoid therapy with fludrocortisone. Dose should be reduced if hypertension develops.

13.9 ACID-BASE DISORDERS
Method of Calculation for the Boards
- Calculate the anion gap immediately if given enough values to calculate it.
- Also, calculate the expected serum osmolality if given the measured serum osmolality. A gap >10 suggests things such as alcohol intoxication.
- Causes of an osmol gap: ethanol, methanol, ethylene glycol, isopropanol, acetone, glycerol, mannitol, uremia
- Note: a normal gap (<10) does not rule out the presence of toxic alcohol.
- First, look at the ABG and electrolytes together, then determine what disorder is present.
- Second, calculate the anion gap. Is an anion-gap or non-anion-gap acidosis present?
- Use Winter's formula to determine if the CO₂ has responded to the metabolic acidosis. If the PCO₂ is greater than expected from this formula, then there is an anion gap metabolic acidosis and a respiratory acidosis (patient with lactic acidosis and CNS depression). If the PCO₂ is less than expected, respiratory alkalosis is present (patient with DKA and hypoxia).

Winters formula = 1.5 X HCO₃⁻ + 8 ± 2 = expected PCO₂ after compensation

- Then look at the delta HCO₃⁻ and delta gap: for a pure anion-gap metabolic acidosis, the HCO₃⁻ will fall as much as the anion gap rises (1:1 ratio)
- If the HCO₃⁻ doesn't fall as much as the gap rose, then a mixed metabolic acidosis and metabolic alkalosis are present (an example would be a DKA patient with significant vomiting).
- If the HCO₃⁻ falls more than the gap rises, then there is a mixed anion-gap and non-anion gap metabolic acidosis (an example would be a patient with a preexisting renal tubular acidosis who develops lactic acidosis).
- Causes of a decreased anion gap
  o Hypoalbuminemia: Each 1 mg/dL of albumin contributes to 2.5 to 3 of the anion gap. So, if the albumin is 4, a normal gap is -12. If a patient has a serum albumin of 2, their normal anion gap is 6; so if they present with a gap of 12, they have anion-gap metabolic acidosis.
  o Multiple myeloma
  o Nephrotic syndrome
  o Bromide ingestion

Metabolic Acidosis (pH <7.35 and HCO₃⁻ <20 mEq/L)
- Metabolic acidosis may lead to severe hypotension, especially if serum pH is <7.10.
- Patients may present with hemodynamic collapse and may be refractory to vasopressor therapy unless the pH is partially corrected with bicarbonate administration (somewhat controversial).
Anion gap (see section on lactic acidosis): treat the underlying cause
  - One of the most common causes in clinical practice is lactic acidosis
- Non-anion gap
  - GI losses: diarrhea or ureteroenterostomy
  - Renal losses: renal tubular acidosis (RTA), adrenal insufficiency, post-hypocapnia

Metabolic Alkalosis (pH >7.45 and \( \text{HCO}_3^- \) >24 mEq/L)
- The most common causes include vomiting, nasogastric tube suction (both remove chloride from the gut and cause the kidneys to retain \( \text{HCO}_3^- \)), diuretics causing contraction alkalosis, and adrenocortical hormone excess.
- The classic presentation of Cushing's syndrome is hypokalemic, metabolic alkalosis.
- Two types:
  - Chloride sensitive
    - Chloride-sensitive causes, such as diuretic-induced, respond to chloride administration (normal saline).
    - The alkalosis will reverse with saline (NaCl) and K replacement.
  - Chloride resistant
    - Chloride-resistant alkalosis (saline unresponsive) can be caused by mineralocorticoid excess, such as in Cushing's syndrome.
    - Does not usually reverse with saline alone
    - Treat with potassium.

Respiratory Acidosis (pH <7.35 and PCO\(_2\) >45 mm Hg)
- Multiple causes, including inadequate ventilation (from coma, sedation, head or chest trauma, or neuromuscular disorders)
- Full renal compensation usually takes a few days.

Respiratory Alkalosis (pH >7.45 and PCO\(_2\) <35)
- Multiple causes, including early sepsis (may be the first sign of impending hemodynamic compromise), CNS disorders, anxiety, pulmonary embolism, hypoxic conditions
- Treat the underlying cause.

13.10 ELECTROLYTE DISORDERS

Hyponatremia (Serum Sodium <135 mEq/L)
- The most common electrolyte abnormality
- Categorized as hypovolemic, hypervolemic, or euvolemic hyponatremia (in other words, what is the patient's volume status?)
  - Hypovolemic hyponatremia
    - Caused by entities that induce excessive sodium loss, e.g., vomiting, diarrhea, diuretics, and adrenal insufficiency
    - Measurement of the urinary sodium concentration allows categorization of hyponatremia as due to renal loss (diuretics, osmotic diuresis) or extrarenal loss (vomiting, diarrhea, or excessive sweating).
      - >20 mEq/L indicates a renal cause
      - <10 mEq/L indicates a nonrenal cause
  - Hypervolemic hyponatremia
    - Causes include congestive heart failure, cirrhosis, and the nephrotic syndrome.
• Euvolemic (normovolemic) hyponatremia
  ▪ Common causes include hypothyroidism, cortisol deficiency, and stable renal disease
  ▪ Other possible causes are psychogenic polydipsia and the syndrome of inappropriate antidiuretic hormone secretion (SIADH)

Pseudohyponatremia
• Conditions that may cause a falsely low serum sodium include multiple myeloma, hyperlipidemia, and hyperglycemia.
• Any condition that leads to markedly elevated serum proteins, lipids, and or glucose may cause pseudohyponatremia.

Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)
• Causes
  o The list of entities that can cause this syndrome is extensive.
  o Consider the common ones: lung cancer and CNS lesions (brain abscess, meningitis)
• Diagnosis
  o One of exclusion: lab values typically show a urine osmolality less dilute than expected (>150)
  o In a patient with a low serum sodium concentration, you would expect the urine to maximally dilute, <100.
  o The urine sodium concentration will be >20 mEq/L.
  o Very common clues to the diagnosis include low blood urea nitrogen (BUN) and uric acid levels.
  o Thyroid, adrenal, and renal disease should be ruled out before this diagnosis is made.
  o Signs/symptoms: headache, confusion, seizures. Patients may be asymptomatic.
• Treatment
  o Hypovolemic
    ▪ Isotonic saline (regardless of the cause)
  o Euvolemic (the volume status of the patient is deemed to be normal)
    ▪ Treatment depends on symptoms.
    ▪ Patients with few if any symptoms may be treated with fluid restriction.
    ▪ Patients with symptoms may be treated with 3% saline (hypertonic saline) and furosemide (loop diuretics get rid of free H₂O).
  o Hypervolemic
    ▪ Patients are typically edematous.
    ▪ “Ineffective” volume leads to salt and water retention with excess water>excess sodium, hence hyponatremia.
    ▪ The underlying disorder should be treated.
    ▪ CHF: diuretics and afterload reduction; may require fluid restriction
  o Treatment with 3% saline (hypertonic saline) is indicated for severe symptoms such as seizures and coma. As a rule of thumb, slowly developing hyponatremia should be treated slowly, and rapidly developing hyponatremia should be treated quickly. Too-rapid correction of hyponatremia has been associated with the development of osmotic demyelination syndrome, a devastating neurologic disorder. The usual (safe) correction rate for hyponatremia >24 hours is no more than 0.6 mEq/L/hr or 12 mEq/L/hr for the first 24 hours.
Hypernatremia (Serum Sodium >145 mEq/L)

- Causes
  - Conditions that lead to reduced water intake (abnormal thirst, osmotic diuresis, or inability to gain access to free water) or increased output
  - Conditions that cause total sodium gain (3% saline administration, hypertonic dialysis, and excessive bicarbonate administration)
  - Diabetes insipidus (DI): condition in which there is depleted central ADH or renal resistance to the action of ADH. ADH stimulates the kidneys to retain free H₂O. DI can be central or nephrogenic in etiology. Usually does not occur unless there is no access to free water.

- Signs/Symptoms: Altered mental status ± seizures

- Treatment: should be based on the rate of development of hypernatremia and patient symptoms
  - The free water deficit should be calculated:
    \[ \text{Volume of H}_2\text{O in liters} = 0.6 \times \text{weight in kg} \times (\text{sodium}/140 - 1) \]
  - Correct free water deficit slowly over 48 hours
  - Initial volume resuscitation with isotonic saline for volume-depleted patients, despite the severity of the hypernatremia. Followed by a more hypotonic fluid such as D5W or ½ NS.
  - Too-rapid correction of hypernatremia may lead to cerebral edema. Chronic hyponatremia causes neurons to generate idiogenic osmoles, which attempt to counteract the hypernatremia.
  - Serum sodium concentration should not be lowered more than 2 to 3 mEq/L over the first 4 to 6 hours.

Hypokalemia (Serum K <3.5 mEq/L)

- Causes
  - GI conditions: emesis, gastric suction, chronic diarrhea, villous adenoma of the colon, laxative abuse
  - Renal disease: renal tubular acidosis and post-obstructive diuresis (both cause metabolic acidosis); diuretics and Cushing's syndrome (both cause metabolic alkalosis)
  - Familial periodic paralysis (consider if a young, healthy patient presents with severe weakness and hypokalemia)
  - β-Agonists and insulin both shift K intracellular and may lead to hypokalemia.
  - Hypomagnesemia (consider, especially if refractory hypokalemia)

- Signs/Symptoms: weakness, hyporeflexia, paralysis
  - ECG: U-waves, flattened T waves (prolonged QT and ST depression when severe), ventricular arrhythmias (e.g., premature ventricular contractions)

- Treatment
  - Acute hypokalemia (may be life-threatening) requires intravenous therapy. As a rule of thumb, estimate 100 mEq K needed to raise serum K by 1 mEq/L.
  - Chronic hypokalemia: patients may have as much as a 200 mEq/L deficit in K for each mEq/L below 3.5

Hyperkalemia (Serum K >5 mEq/L)

- Causes
  - Common causes include renal failure, ingestion of potassium-containing salt substitutes, medications (such as ACE inhibitors, potassium-sparing diuretics, NSAIDS, TMP/SMX), adrenal insufficiency, hemolysis, type 4 renal tubular acidosis (nonanion gap metabolic acidosis and hyperkalemia in a diabetic), rhabdomyolysis, and conditions/medications that cause transcellular shift of K (DKA, β-blockers, digoxin)
  - A dialysis patient in cardiac arrest is hyperkalemic until proven otherwise.
• Signs/Symptoms: may have lethargy or weakness, which progresses to paralysis and areflexia
• ECG: tall, peaked T waves, wide QRS, prolonged PR interval, flattened P wave, sine wave pattern.
• Treatment: depends on the level of serum K and whether or not electrocardiographic findings are present.
  o Calcium gluconate or chloride (calcium chloride has ~ 3X the amount of Ca) IV for cardiac membrane stabilization; does not alter serum K levels
  o Sodium bicarbonate IV causes intracellular shift of K (works if patient is acidemic)
  o Insulin + glucose given IV causes intracellular shift of K
  o Kayexalate (oral) causes Na/K exchange in the bowel; is essentially an oral sodium load, so use with caution in severely volume-overloaded patients; not a potent hypokalemic agent
  o Albuterol (nice trick to use if no IV access is available): give 5 to 10 mg as a nebulizer
  o Dialysis

Hypocalcemia (Serum Ca <8.6 mEq/L)
• Causes
  o Hypomagnesemia (can lead to refractory hypocalcemia if not recognized and treated). Mg is required for parathyroid hormone and calcium homeostasis.
  o Rhabdomyolysis
  o Pancreatitis (with fat necrosis [fat binds Ca])
  o Hypoparathyroidism (common after thyroidectomy [transient or permanent])
  o Drugs: phenytoin, cimetidine, foscarnet
  o Any form of alkalosis (increase in pH decreases free or ionized Ca)
  o Massive blood transfusions (Ca binds to citrate); usually requires several units
  o In patients with LOW ALBUMIN, serum Ca is ~50% bound to albumin and is in equilibrium with free or ionized Ca. Anything that decreases albumin can decrease the total serum Ca concentration, but the concentration of the ionized form will be normal.
  o To correct serum calcium for hypoalbuminemia:

    \[
    \text{measured serum Ca} + [(4 - \text{patient's albumin}) \times 0.8] = \text{corrected serum Ca}
    \]

• Signs/Symptoms
  o Cardiac: prolonged QT, dysrhythmias, hypertension
  o Neuromuscular: perioral and peripheral parathesias, Chvostek's and Trousseau's sign (carpopedal spasm), hyperreflexia, and/or seizures
• Treatment
  o Identify and treat the underlying cause.
  o Symptomatic patients require Ca replacement with IV therapy: Ca gluconate, 10 cc of a 10% solution over 15 to 20 minutes.
  o Less severe cases can be treated with oral calcium.

Hypercalcemia
• Causes
  o Most common causes include hyperparathyroidism, cancer (metastatic cancer to bone, lymphoma), sarcoidosis, multiple myeloma, immobilization, Addison's disease, vitamin D toxicity, and hyperthyroidism.
  o Cancers that metastasize to bone include breast, lung, thyroid, renal, and prostate.
• Signs/Symptoms: “stones, moans, groans, and bones”
  o Renal: kidney stones, may cause renal failure, causes nephrogenic diabetes insipidus (presents with polyuria)
  o GI: anorexia, vomiting, abdominal pain, a rare cause of pancreatitis
  o Cardiac: hypertension, dysrhythmias, short QT interval, digoxin sensitivity
• Treatment: depends on the cause
  o Start with hydration (normal saline).
  o Can use a loop diuretic such as furosemide but not until the patient is adequately volume loaded
  o Steroids, especially if lymphoma or sarcoidosis is suspected
  o Bisphosphonates (pamidronate, etidronate, zoledronic acid); newer ones being developed; typically given in consultation with the patient’s oncologist

Hypomagnesemia
• Causes
  o Inadequate intake or absorption (malnutrition), especially alcoholism, pancreatitis, endocrine disorders such as DKA, hyperparathyroidism, and hyperaldosteronism
  o Malabsorption diseases such as chronic diarrhea, fistulas, and abdominal radiation
• Signs/Symptoms
  o Cardiac: may cause hypotension and dysrhythmias. Electrocardiographic findings include prolongation of the PR and QT intervals, wide QRS complex, ST depression
  o Neuromuscular symptoms: lethargy, carpopedal spasm, hyporeflexia, tetany
  o May lead to signs and symptoms of hypokalemia and hypocalcemia; adequate magnesium levels are required to maintain normal serum K and Ca
• Treatment
  o Rule out low K and Ca levels
  o For severe symptoms, give IV magnesium sulfate, 1 to 4 grams (depending on the initial serum Mg concentration)
  o Patients with malnutrition or alcoholism may be severely magnesium depleted.

Hypermagnesemia
• Causes
  o Renal failure
  o Iatrogenic (patients with preeclampsia or eclampsia given IV magnesium)
  o Adrenal insufficiency
• Signs/Symptoms
  o Neuromuscular: weakness, hyporeflexia, coma, respiratory failure, lethargy
  o Cardiac: prolonged PR and QT intervals; hypotension (Mg >10–12 mEq/L); may cause bradycardia, AV block, and asystole
• Treatment
  o Calcium IV (antagonizes the effect of Mg)
  o Can also use loop diuretics and/or dialysis
Hypophosphatemia
- Causes: malabsorption, malnutrition, diuretics, hyperglycemia, alkalotic states (intracellular shift)
- Signs/Symptoms
  - Vary widely depending on the phosphorus level
  - Phosphate is needed for ATP generation, so patients may present with hypotension, CHF, respiratory depression, weakness, myalgias
  - Hypophosphatemia causes rhabdomyolysis (as does hypokalemia), hemolysis, and WBC and platelet dysfunction
- Treatment
  - Can give oral phosphate (as potassium acid phosphate) for mild hypophosphatemia or IV Na or K phosphate for moderate to severe symptoms
  - In DKA, most would agree with phosphate replacement if the phosphate level is <1 mEq/L.

Hyperphosphatemia
- Causes
  - Renal failure
  - Rhabdomyolysis
  - Hemolysis
  - Thyrotoxicosis
  - Exogenous causes (laxatives and enemas)
  - Tumor lysis syndrome (metabolic syndrome characterized by hyperphosphatemia, hyperkalemia, hypocalcemia, hyperuricemia, and renal failure in patients receiving chemotherapy)
- Signs/Symptoms: progressive neuromuscular irritability, tetany, hypotension, CHF, bradycardia, dysrhythmias
- Treatment: hemodialysis or aluminum-containing phosphate binders (aluminum hydroxide)
CHAPTER 14
Environmental Disorders

Glenn K. Geeting, MD

14.1 BITES AND ENVENOMATIONS

Insects
- Hymenoptera
  - Bees, fire ants, hornets, wasps
  - Venom has multiple components (acid phosphatase, hyaluronidase, phospholipase A, others)
  - Erythema, pain, local swelling
  - Systemic toxicity is worse with multiple stings (Africanized bees, fire ants).
  - Can get anaphylaxis or serum sickness
  - Treatment for local symptoms includes ice, antihistamines, and analgesics.
- Tick-borne diseases
  - Babesiosis
    - *Ixodes* tick: protozoan parasite
    - Splenectomy is a risk factor.
    - Malaria-like illness: hemolysis, elevated liver function tests (LFTs)
    - 20% have concurrent Lyme infection
  - Colorado tick fever
    - *Dermacentor* tick in Rocky Mountain states transmits an RNA virus
    - Fever, petechial rash (see Image #36), transient leukopenia
  - Ehrlichiosis
    - *Ixodes* and *Amblyomma* (lone star tick): *Rickettsia*-like intracellular coccobacillus
    - Fever, myalgias, thrombocytopenia, leukopenia, liver dysfunction
    - Treatment with doxycycline for 7 to 14 days
  - Lyme disease
    - *Ixodes* tick: spirochete *Borrelia burgdorferi*
    - Erythema migrans rash (see Image #40), arthritis, meningoencephalitis, cranial neuropathies (especially facial nerve palsy), myocarditis with atrioventricular blocks.
    - Diagnosis is made by serology.
    - Treatment is with doxycycline, cephalosporin, or macrolide.
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- Rocky Mountain spotted fever
  - *Dermacentor* species (wood and dog ticks) are present in all 48 states: *Rickettsia rickettsii*
  - Triad of fever, petechial rash (see Image #42), and tick bite
  - Diagnosis by skin biopsy; serologic tests are not useful in acute illness
  - Treatment: doxycycline, even in pediatric patients
- Tularemia
  - *Dermacentor* and *Amblyomma* ticks: Gram-negative coccobacillus
  - Rabbit host
  - Causes ulceroglandular or pulmonary disease
  - Diagnosis by serology
  - Treatment: streptomycin
- Tick paralysis
  - Salivary gland toxin secreted by *Dermacentor* and *Ixodes* ticks
  - Causes ascending flaccid paralysis, ataxia, and loss of deep tendon reflexes
  - 10% mortality rate
  - Treatment: remove the tick, supportive care
- Relapsing fever
  - Caused by spirochete *Borrelia*
  - Causes febrile illness that resolves and then recurs after 7 days
  - Diagnosis: malaria smears
  - Treatment: tetracycline or erythromycin

Marine Organisms

- Bony fishes
  - Stonefish, lionfish, turkeyfish, scorpionfish, zebrafish
  - Toxin ejected through dorsal fins or spine
  - Heat-labile toxin: prostaglandin B2, thromboxane E2, others
  - Severe local burning, pain, swelling, nausea, vomiting, cardiopulmonary dysfunction
  - Treatment: hot water, analgesics, x-ray for foreign body
- Stingrays
  - Toxin ejected through barbed spines of the tail
  - The stinger is often broken off and must be removed from the wound.
  - Heat-labile toxin: phosphodiesterase, serotonin, others
  - Pain out of proportion to exam
  - Possible local wound infection with *Vibrio* species
  - Treatment: hot water, analgesics, x-ray for foreign body, consider antibiotics
- Coelenterata
  - Jellyfish, sea anemones, stinging corals
  - Nematocysts: venom sacs with spring-loaded stinging mechanism
  - Venom: complex mixture (bradykinin, phosphodiesterases, fibrinolysin, others)
  - Severe burning sensation and erythema
  - Treatment: remove intact nematocysts (rinse with sea water or vinegar, scrape off), antihistamines, analgesics
  - Box jellyfish (*Chironex fleckeri*): most deadly of stinging marine species, Australian waters, use antivenin/verapamil IV
• Sea urchin
  o Toxin-coated spines often break off
  o Heat-labile toxin
  o Local pain, burning, and erythema
  o Treatment: hot water, analgesics, remove spines

Snakes
• Crotalidae (pit vipers): rattlesnakes, copperheads (found in United States)
  o Identification: triangular head, elliptical pupil, single row of caudal plates, heat-sensing pit between the eye and nostril
  o Symptoms can range from local toxicity (pain, burning, numbness, edema) to systemic toxicity (metallic taste, nausea/vomiting, thrombocytopenia, DIC, cardiovascular collapse)
  o Envenomation can be graded from 0 (minimal) to 4 (severe)
  o Intravenous or proximal bites are more rapidly fatal
  o Treatment
    ▪ Do NOT perform incision and suction.
    ▪ Local compression and immobilization slow systemic effects.
    ▪ Consider antivenin for moderate to severe (Grades 2–4) envenomations
      □ Be alert for anaphylaxis whenever horse serum is used; consider pretreatment with epinephrine

• Coral snake (and other exotic Elapidae like the cobra)
  o Identification of a coral snake versus a harmless king snake
    ▪ “Red on yellow kill a fellow; red on black venom lack.”
  o Neuromuscular toxin with minimal local toxicity
  o Ptosis is common and often the first outward sign of envenomation.
  o Usual cause of death is respiratory failure.
  o Treatment: local wound care, antivenin

Spiders
• Black widow (Latrodectus)
  o Large, black spider with red hourglass on ventral abdomen
  o Toxin: latrotoxin and other neuroactive agents
  o Symptoms: pinprick bite followed by dull, crampy pain, muscular rigidity that can mimic a surgical abdomen, hypertension, rare cardiovascular effects
  o Treatment: wound care, analgesia, muscle relaxants (benzodiazepines or dantrolene), nitroprusside for severe hypertension
    ▪ Antivenin (horse serum) for severe envenomation, pregnant women, children, and elderly
  o Admit pregnant women, children, those with symptoms of moderate envenomation, preexisting cardiovascular disease, or hypertension

• Brown recluse (Loxosceles)
  o Dark violin shape on dorsal abdomen
  o Painless bite with delayed symptoms of bull’s eye rash that can progress to a large ulcerated lesion (see Image #13)
  o Treatment: local wound care, dapsone, hyperbaric oxygen, analgesics
Scorpions
- *Centruroides exilicauda*, "bark scorpion" in Arizona has a neurotoxic venom that is particularly dangerous
  - The sting site has heightened sensitivity.
- *Tityus trinitatis* stings typically cause pancreatitis.
- Treatment
  - Goat-derived antivenin is available for severe *C. exilicauda* envenomations.
  - Benzodiazepines may help myoclonus.
  - Atropine may be used for hypersalivation and bradycardia.
  - Nitroprusside or prazosin for hypertension
  - Avoid narcotics and barbiturates, which may increase toxicity

14.2 DYSBARISM

Gas Laws
- Boyle's law: \( PV = K \)
  - As the pressure increases (descent), the volume decreases
  - “Squeeze” syndromes relate to this change
- Dalton's law: \( P(\text{total}) = P(\text{oxygen}) + P(\text{nitrogen}) + P(x) \)
  - The total pressure of the mixture of gases is equal to the sum of the partial pressures of the gases.
  - The partial pressure of a gas changes as the pressure changes despite no change in the absolute concentration.
  - Air is about 78% nitrogen and 21% oxygen; used for most recreational diving
- Henry's law: \( C(x) = P(x) \times \text{Solubility} \)
  - The concentration of dissolved gas is proportional to the pressure of the gas.
  - Gas under pressure dissolves in fluid. If the pressure is relieved quickly, it comes out of solution as bubbles.

Barotrauma
- Middle ear barotrauma ("ear squeeze")
  - Gas volume decreases with descent (Boyle's law).
  - A blocked Eustachian tube with the inability to equalize middle ear pressure causes traction on the tympanic membrane and conductive hearing loss.
  - Complications include tympanic membrane rupture; cold water entry can cause severe vertigo and facial nerve palsy (rare)
  - Prevent with decongestants and equalizing inner ear pressure by swallowing on a closed glottis or performing Valsalva maneuver while plugging nose
- External ear barotrauma
  - Squeeze when external canal is blocked by cerumen, ear plugs, or a tight hood
- Inner ear barotrauma
  - Rare rupture of the inner ear round window during descent
  - Triad: tinnitus, neural deafness, and vertigo (same as Meniere's); also nystagmus and ataxia
  - Treatment: ENT or neurology referral, supportive care
- Sinus barotrauma
  - Squeeze of sinus caused by blocked sinus passages: sinusitis, allergies, polyps
  - Treatment: analgesics, decongestants
• Facial barotrauma
  o Squeeze in space occupied by face mask can cause facial pain and edema, conjunctival edema, and subconjunctival hemorrhages
  o Prevented by diver exhaling through nose while descending

Nitrogen Narcosis ("Rapture of the Deep")
• With increasing pressures (descent), dissolved nitrogen enters tissues and the CNS
• Below ~100 ft, affected people act drunk: euphoria, confusion, loss of judgment (associated with diving accidents)
• At depths >120 feet, mixed gases are recommended (rather than compressed air).
• Resolves with ascent

Pulmonary Over-Pressurization Syndrome (POPS), Pulmonary Barotrauma
• General
  o Occurs as diver ascends without exhaling
  o Increased volume in the lungs can force air across the alveolar membrane or cause the alveoli to burst.
  o Divers with asthma have a two-fold risk of POPS
  o Example: at a 33-ft depth (2 atmospheres pressure), a diver takes a full breath. If she does not exhale while ascending, her lung volume is double at the surface (1 atmosphere pressure), per Boyle’s law.
• Arterial gas embolus
  o Second leading cause of death among sports divers
  o Caused by air being forced across the alveolar membrane during ascent
  o Highest risk in the last 10 feet of ascent
  o Air bubbles in pulmonary venous circulation proceed through the left side of the heart and may embolize systemically (coronary or cerebral arteries)
  o Most common symptoms: altered consciousness, headache, dizziness, convulsions, and visual changes
• Pneumothorax
  o Air dissests across the visceral pleura
  o Tension pneumothorax is rare.
• Pneumomediastinum (see Image #57)
  o Air dissests into the pulmonary interstitium and can travel into the neck, mediastinum, and pericardium.
  o Can cause subcutaneous crepitus, neck fullness, change in voice quality
  o Not life-threatening unless massive
• Alveolar hemorrhage
  o Hemoptysis, chest pain, and dyspnea
  o Can look like Pulmonary Type II decompression syndrome (DCS) (see below); begins during ascent; the cause is alveolar rupture rather than nitrogen bubble dissolution

Decompression Syndrome (DCS)
• General
  o Clinical illness caused by the formation of small bubbles of nitrogen gas in the blood or tissues
  o Henry’s law: gas (nitrogen) under pressure dissolves in fluid. If the pressure is relieved too quickly, it will come out of solution as bubbles.
  o Signs and symptoms depend on where the bubbles form and deposit.
  o A patent foramen ovale or other right-to-left shunt increases susceptibility to and severity of DCS.
  o Risk factors include age, obesity, fatigue, exertion, dehydration, fever, cold temperatures, diving at high altitudes, and flying after diving.
• Type I DCS, “The Bends,” Caisson’s Disease
  o Painful joints (typically elbow and shoulder), skin bubbling, muscular pain
  o Cutis marmorata (skin marbling) results from venous stasis and causes pruritus and erythema.

• Type II DCS
  o Symptoms beyond joints: usually ear, CNS, or lungs
  o Inner ear DCS, “The Staggers”
    ▪ Gas bubbles in the inner ear cause severe vertigo, ataxia, tinnitus, and nystagmus.
  o CNS DCS
    ▪ Spinal DCS (more common): limb paresthesias or pain, weakness, dermatomal sensory symptoms, incontinence, priapism
    ▪ Cerebral DCS: headache, visual symptoms, behavioral changes, mental status change, stroke symptoms
  o Pulmonary DCS, “The Chokes”
    ▪ Microbubble emboli to the lungs cause cough, dyspnea, chest pain.
    ▪ Differential diagnosis includes pulmonary embolus and arterial gas emboli (see above).

Recompression (Hyperbaric) Therapy
• Therapy of choice for decompression syndrome and arterial gas embolism
  o NOT for barotrauma, nitrogen narcosis, oxygen toxicity, pneumothorax, or pneumomediastinum
• Treatment prior to recompression: 100% oxygen (speeds nitrogen washout), IV fluids
• Low threshold for treatment, even in delayed presentations
• Ground therapy to the chamber is preferred; if air transportation is necessary, maintain aircraft cabin pressure at 1000 ft
• Consult U.S. Navy Diving Manual treatment tables
• No flying for 3 to 7 days after treatment of Type I DCS and for 4 weeks after treatment of Type II DCS

14.3 HIGH-ALTITUDE ILLNESS

General
• Risk factors
  o Rate of ascent
  o Sleeping altitude
  o Final altitude reached
  o Duration of stay at altitude
  o Individual susceptibility (difficult to predict)
• Definitions

<table>
<thead>
<tr>
<th>Altitude</th>
<th>Definition</th>
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</thead>
<tbody>
<tr>
<td>Moderate altitude</td>
<td>8,000–10,000 ft</td>
</tr>
<tr>
<td>High altitude</td>
<td>10,000–18,000 ft</td>
</tr>
<tr>
<td>Extreme altitude</td>
<td>&gt;18,000 ft</td>
</tr>
<tr>
<td></td>
<td>Rapid ascent may cause illness</td>
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<tr>
<td></td>
<td>Desaturation occurs</td>
</tr>
<tr>
<td></td>
<td>Acclimatization is not usually possible</td>
</tr>
</tbody>
</table>

• Barometric pressure decreases logarithmically as altitude increases.
• Partial pressure of oxygen decreases as the total pressure decreases (Dalton’s law).
• The steep portion of the oxygen-hemoglobin dissociation curve indicates that a relatively small change in altitude can create a significant change in oxygenation.
Acclimatization
- Gradual, physiologic changes in response to hypoxia can restore tissue oxygen pressures.
  - Increased minute ventilation
  - Renal bicarbonate excretion compensates for the respiratory alkalosis.
  - Increased catecholamine level increases cardiac output.
  - Hematopoiesis – increased RBC count and hemoglobin
- Has allowed some climbers to ascend to extreme altitude without supplemental oxygen
- Cardiac, pulmonary, hematologic, and genetic factors influence this process.

Acute Mountain Sickness (AMS)
- Rarely occurs below moderate altitude (8,000 ft)
- Symptoms generally develop several hours after reaching altitude and peak at 24 to 48 hours
- "Like a hangover" – headache, nausea, anorexia, insomnia, dizziness, fatigue
- Alcohol or sedatives may bring on or exacerbate AMS by causing hypoventilation, which prevents compensatory hyperventilation
- Prevention
  - Gradual ascent to allow acclimatization, high-carbohydrate diet, avoid alcohol and smoking
  - Acetazolamide (carbonic anhydrase inhibitor) prophylaxis induces renal bicarbonate diuresis, causing a metabolic acidosis, which increases ventilation
- Treatment
  - Mild cases are usually self limited and require no treatment other than discontinuing ascent and rest.
  - For moderate cases, consider acetazolamide, supplemental oxygen, antiemetics, acetaminophen.
  - If severe or persistent: descend, consider dexamethasone

High-Altitude Cerebral Edema (HACE)
- Least common but most severe high-altitude illness
- Rare below 12,000 feet
- HACE usually presents after 2 or 3 days of AMS
- Cerebral edema and increased intracranial pressure (ICP)
- Global CNS alteration: confusion, seizures, mental status change, retinal hemorrhages
- Patients generally also have severe AMS (headache, fatigue, vomiting) and HAPE (cough, dyspnea) (see below).
- Treatment
  - Descent (most important)
  - Supplemental therapy (while descending): high-flow oxygen, dexamethasone
  - If severe, consider diuretics, intubation, and hyperventilation to control ICP
  - Portable hyperbaric chamber (Gamow bag) simulates descent of 5,000 feet by adding 2 psi of pressure

High-Altitude Pulmonary Edema (HAPE)
- Most common fatal manifestation of severe high-altitude illness; rare below 10,000 feet
- Symptoms develop slowly over 2 to 4 days after arrival at altitude
- Severe dyspnea on exertion, severe fatigue, dry cough
- Dyspnea at rest warns of a serious pulmonary problem
- Usually associated with AMS symptoms and may rapidly lead to HACE
- Chest film shows patchy alveolar infiltrates that are bilateral in advanced cases
- Can be difficult to distinguish from pneumonia, as both can present with fever and dyspnea
  - Treat the same way; add antibiotics for suspected pneumonia
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- Treatment
  - Descent (most important) of 2,000 to 3,000 feet markedly improves symptoms
  - Rest, warmth, high-flow oxygen
  - Furosemide, nifedipine (pulmonary vasodilator), and morphine
  - Gamow hyperbaric bag

14.4 SUBMERSION INCIDENTS

Drowning
- Terminology
  - Drowning = death due to submersion
  - Submersion = survivor (previously termed near-drowning)
- Second leading cause of death in children
- Two age peaks:
  - Toddlers: swimming pools, bathtubs, large buckets
  - 16 to 20 years: 80% males, often involves alcohol, reckless behavior, associated trauma (spinal precautions), seizures
- Freshwater (pool) drownings are more common than those in saltwater.
- Adverse survival factors: duration of immersion >5 min (most important), associated trauma, associated illnesses, age <3 years, no bystander CPR, hypothermia
  - Salt versus fresh water: no survival difference
- “Dry drownings,” thought to be caused by laryngospasm, are probably deaths from other causes.
- Patients can have delayed deterioration with development of ARDS.

Cold Water Immersion
- Rapidly leads to hypothermia, especially in children
- Hypothermia is a bad prognostic sign.
- Occasional cases of survival from extended (>30 min) cold water immersion
  - Diving reflex shunts blood to the heart and brain.
- Cold water immersion syndrome: syncope resulting from cardiac dysrhythmias on sudden contact with very cold water
  - May be linked to long Q-T syndrome

14.5 TEMPERATURE-RELATED ILLNESS

General
- Heat-transfer mechanisms
  - Radiation: heat loss/gain by electromagnetic waves (main mechanism of heat loss in cool environments, ~65%)
  - Evaporation: lose heat as sweat turns into gas (dominant mechanism for heat loss in hot conditions)
  - Conduction: transfer of heat by direct contact with another object (cold water immersion)
  - Convection: heat loss to circulating air (wind chill)

Heat Illness
- General
  - Acclimatization: physiologic adaptations after repeated exposure to heat
    - Takes 1 to 2 weeks to develop
    - Plasma volume expansion
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Risk factors
- Extremes of age
- Exertion: 20-fold increase in heat production with exercise
- Dehydration
- Occlusive clothing
- Cardiac disease (need to increase cardiac output to facilitate heat loss)
- Medications: use of β-blockers, anticholinergics, diuretics
- Alcohol or street drug use (cocaine, amphetamines, MDMA, ecstasy, PCP)
- Mental status abnormalities

Heat exhaustion, “heat prostration”
- May progress to heat stroke if not treated
- Malaise, fatigue, headache, dehydration
- Similar to heat stroke without CNS dysfunction, liver dysfunction
- Primarily a problem of volume depletion
- Treatment: cool environment, oral electrolyte solution, consider IV fluids
- Replace free water deficits over 48 hours, as rapid correction is associated with seizures and cerebral edema

Heat stroke
- Core temperature >40.5°C (105°F) with multisystem dysfunction
- Indicates failure of thermoregulatory mechanisms
- Neurologic dysfunction is the hallmark: cerebral edema, mental status change, seizures, delirium, bizarre behavior
- Muscular injury: rhabdomyolysis
- Hepatic injury with elevated transaminases: if not present, makes the diagnosis of heat stroke doubtful
- Diarrhea: from splanchnic vasoconstriction
- Classic heat stroke versus exertional heat stroke
  - Classic: heat wave, nonexertional, elderly patients on medication, minimal sweating, rhabdomyolysis is rare
  - Exertional: young, active, profuse sweating, rhabdomyolysis is common
- Treatment: rapid cooling is the key
  - Cool mist with fans
  - Cold water immersion
  - Ice packs in groin, axillae
  - Cooling blanket
  - Peritoneal or gastric lavage with cold fluids
  - Extracorporeal cooling
  - Muscle relaxants/paralytics for shivering
  - NO antipyretics: ineffective in environmental hyperthermia
  - Stop cooling measures at 40°C (104°F)

Cold Illness
- General
  - Risk factors: age, alcohol or street drug use, altered mental status
  - Unlike heat adaptation (acclimatization), the body has limited cold adaptation.
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- Frostbite
  - In cold stress, peripheral vasoconstriction limits heat loss ("life vs. limb")
  - Occurs when tissue temperature drops below 0°C, causing ice crystal formation and microvascular thrombosis
  - Numbness, sensory deficit, "chunk of wood" sensation
  - Rewarming and refreezing worsen prognosis (do not do field rewarming).
  - Prognosis correlates directly with time frozen.
  - Expect significant post-treatment sequelae: paresthesias, pain, thermal sensitivity, atrophy, edema, etc.
  - Treatment: warm with water at 40 to 42°C (35–40°C may be better tolerated)
    - Rewarming is painful and may require IV analgesics.
    - Premature termination of thawing is a common error.
    - Management of blisters (controversial)
      - Debride only broken blisters
      - Aspirate rather than debride intact blisters
      - Hemorrhagic blebs reflect subdermal damage, less favorable prognosis
      - Conservative surgical debridement after weeks of observation for line of demarcation
    - Prediction of tissue loss is not possible immediately after injury.

- Frostnip, chillblains, pernio: reversible, transient numbness that resolves with warming
  - Intermittent exposure to subfreezing temperatures
  - Painful "cold sore" skin lesions of hands, feet, pretibial areas
  - Supportive care, nifedipine, steroids

- Immersion foot, trench foot
  - Prolonged exposure to wet and cold environment at non-freezing temperatures
  - Develops over days
  - Neurovascular damage in the absence of ice crystals

- Hypothermia
  - Mild hypothermia <35°C
    - Vasoconstriction, shivering, thermogenic increase in basal metabolism
  - Severe hypothermia <32°C
    - Consider hypoglycemia, hypovolemia, and overdose if there is tachycardia disproportionate to the temperature.
  - Cardiovascular:
    - Initial tachycardia followed by bradycardia (profound with severe hypothermia)
    - Osborne J wave: dome or hump after the QRS complex
    - Myocardium is irritable and prone to arrhythmias.
    - Most common arrhythmias: bradycardia and atrial fibrillation
    - Below 32°C, expect an irritable myocardium; asystole and ventricular fibrillation can occur spontaneously below 25°C.
  - CNS: depression with progressive slowing of EEG as temperature decreases
  - Renal: cold diuresis produces a large amount of dilute urine and dehydration
  - Respiratory: initial increase in respiratory rate followed by progressive slowing in minute volume as temperature decreases
  - Core temperature afterdrop: warming vasodilates the cold periphery and brings cold blood with lactic acidosis to core
Cold-induced disseminated intravascular coagulation: clotting factors are temperature dependent

Treatment
- Fluid resuscitation with D5 normal saline at 40 to 42°C
- Rewarming
  - Passive external: warm blankets
  - Active rewarming is indicated for cardiovascular instability, temperature <32°C, failure to rewarm by passive means, endocrine insufficiency, vasodilation
    - Active external: humidified warm oxygen, radiant source, forced air warming systems (Bair Hugger)
    - Active core: warm NG/Foley/colonic lavage, warm peritoneal lavage, warm chest tube lavage, extracorporeal warming
- Advanced Life Support considerations
  - Contraindicated: atropine, epinephrine, high-dose dopamine
  - Try defibrillation, but do not shock multiple times; rewarm and continue CPR
  - Defibrillation is rarely successful below 30°C
  - Most hypothermia-induced arrhythmias convert spontaneously during rewarming.

14.6 RADIATION ILLNESS

General

<table>
<thead>
<tr>
<th>Type of Radiation</th>
<th>Usual Source</th>
<th>Penetration</th>
</tr>
</thead>
<tbody>
<tr>
<td>X-rays</td>
<td>X-ray machines or accelerators</td>
<td>Deep</td>
</tr>
<tr>
<td>Gamma rays</td>
<td>Radioisotopes</td>
<td>Deep</td>
</tr>
<tr>
<td>Neutron</td>
<td>Fallout, nuclear reactors, critical assemblies, or accelerators</td>
<td>Deep</td>
</tr>
<tr>
<td>Beta</td>
<td>Radioisotopes</td>
<td>Shallow (&lt;8 mm)</td>
</tr>
<tr>
<td>Alpha</td>
<td>Plutonium decay</td>
<td>Very shallow</td>
</tr>
</tbody>
</table>

- Terms
  - Irradiated – exposed to ionizing radiation; not radioactive or dangerous to care providers
  - Contaminated – radioactive particulate matter (alpha and beta particles) covering exposed surfaces; decontamination and precautions are effective in protecting care providers
  - Incorporation – radioactive material is ingested, inhaled, or absorbed through an open wound

- Triage: Initial symptoms and signs are the most reliable indicator of the radiation dose received and the patient’s prognosis.
  - Survival probable group – no or mild symptoms, <2 Gray
  - Survival possible group – initial nausea and vomiting are brief with later hematopoietic changes, 2 to 10 Gray
  - Survival improbable group – fulminating vomiting and diarrhea, >10 Gray

- Syndromes
  - Gastrointestinal syndrome – nausea, vomiting, >1 Gray
    - High fever and bloody diarrhea are ominous signs.
  - CNS syndrome – headache, altered mental status, >20 Gray
  - Hematopoietic syndrome – 2 days to 4 weeks, pancytopenia from marrow suppression

- The LD50 for radiation is 4.5 Gray with proper medical care, 3.5 Gray in mass casualty scenarios
  - 10 Gray is probably the maximum survivable dose with optimal care (including bone marrow transplant)
• Absolute lymphocyte count (ALC) is the best prognostic indicator in the first 48 hours.
  o ALC <500 correlates to very severe exposure (>4 Gray) and poor prognosis.
• A “dirty bomb” would likely cause more panic than actual radiation injuries.
• Treatment
  o Decontamination is best done at the scene with removal of clothing and cleansing with soap and water
  o Contaminated clothing, irritants, and body fluids should be kept in sealed containers.
  o Radiation skin burns are generally treated like thermal burns.
  o Wounds should be irrigated until monitors indicate contamination is removed; consider debridement for continued high readings.
  o Wounds after exposures >1 Gray should be closed primarily.
    ▪ Necessary surgery should be performed immediately because delays may be complicated by fluid shifts and marrow suppression.
  o Therapy is generally supportive.
  o Ingestion or inhalation of radioactive material may warrant administration of blocking (iodine) or chelating agents.
CHAPTER 15
Musculoskeletal Disorders

Kevin G. Rodgers, MD, and Kohei Hasegawa, MD

We would like to recognize Dr. Laurence Raney for his contributions from original handouts and illustrations 1 and 5-11, inclusive, which were incorporated into Chapter 15, Musculoskeletal Disorders.

15.1 BONY ABNORMALITIES

Osteonecrosis

- General Information
  - Bony infarction caused by disruption of the blood supply
  - Causes: idiopathic (20%) or associated with trauma, steroid use, alcoholism, sickle cell disease, dysbarism, chronic pancreatitis, collagen vascular disease, or renal transplantation

- Primary Osteonecrosis of the Hip
  - Most common anatomic site
  - More common in men (mean age of 37)
  - Presentation: hip/thigh/knee pain/stiffness and no history of trauma
    - Occurs bilaterally in ~50%
  - Treatment: protected weight bearing, NSAIDs, and orthopedic follow-up

- Primary Osteonecrosis of the Knee
  - Most common in elderly females
  - Etiology is unknown.
  - Presentation: acute knee pain, especially at night, and tenderness over the medial-lateral femoral epicondyle
  - Diagnosis: x-ray films are usually normal; MRI is diagnostic.

- Secondary Osteonecrosis of the Humerus
  - Commonly seen in sickle cell disease

- Traumatic Osteonecrosis of the Proximal Femur (Hip)
  - Occurs following femoral neck fractures or hip dislocation
    - 10% to 20% incidence with optimal treatment; up to 40% incidence overall
    - Preventing this is the primary reason for emergent reduction of these injuries.

- Traumatic Osteonecrosis of the Scaphoid
  - Seen in 3% of scaphoid fractures (non-union in 5%–10%); leads to bony collapse and severe arthritis
  - Since 15% of scaphoid fractures are NOT seen on initial plain radiographs, any tenderness in the anatomic snuffbox following wrist trauma dictates immobilization in a thumb spica splint for 10 to 14 days, followed by reimaging to look for callus formation.
    - Although costly, a normal bone scan at 96 hours following injury can accurately rule out fracture as well.
• Traumatic Osteonecrosis of the Lunate (Kienbock’s Disease)
  o Seen in 20% of lunate fractures
  o Presentation: chronic wrist pain, often without recall of a traumatic episode
  o Diagnosis: lunate is shrunken and sclerotic on radiographs
  o Treatment: initial immobilization and operative repair or prosthetic replacement

• Pediatric Osteonecrosis of the Proximal Femoral Epiphysis (Legg-Calve-Perthes Disease)
  o Occurs most commonly in boys (three to five times the incidence in girls) ages 3 to 12 (peak ages are 5 to 7)
  o Discovery is often made after a traumatic event, but the condition is not caused by the trauma
  o May occur as a complication of slipped capital femoral epiphysis (SCFE, see Image #66)
  o Presentation: chronic hip/knee pain and limp; bilateral in 20%
  o Diagnosis: MRI is the best modality for evaluation
  o Treatment: orthopedic consultation; keep non-weightbearing

Osteomyelitis
• General Information
  o Osseous infection of non-articular bone
  o Classified as acute (<6 weeks) or chronic
  o Three mechanisms of contamination: 1) hematogenous spread (pediatric and adult vertebral), 2) seeding from a contiguous source of infection, or 3) direct inoculation of the bone (open fracture, iatrogenic)
• Predisposing Factors
  o Associated with diabetes mellitus, sickle cell disease, AIDS and other immunocompromised states, alcoholism, intravenous drug abuse, chronic steroid use, pre-existing joint disease, and indwelling hardware/prosthesis
• Etiology
  o Staphylococcus aureus is the primary cause in all age groups except neonates, among whom group B Streptococcus is the leading cause.
  o Consider these specific associations:
    ▪ Children: Kingella kingae is now the primary pathogen since the widespread use of the H. influenza vaccination
    ▪ Elderly: gram-negative organisms
    ▪ Sickle cell and asplenic individuals: encapsulated organisms, especially Salmonella
    ▪ Patients with foot puncture wounds, IV drug abuse, or in-dwelling hardware: Pseudomonas
    ▪ Prosthetic devices: Staphylococcus epidermidis
    ▪ Fractures with exposure to fresh water: think Aeromonas hydrophila
    ▪ Dog and cat bites involving bone: Pasteurella multocida
    ▪ Pott’s disease: TB of the spine/vertebral body, especially in immunocompromised, homeless, or “shelter” patients
    ▪ “Fight bites”: Eikenella corrodens (see Image #27)
    ▪ Hospitalized, immunocompromised, chronically debilitated patients: fungal infections cause <1% of osteomyelitis (Candida and Aspergillus spread hematogenously in adults; blastomycosis/cryptococcus/aspergillosis involving the bone in disseminated disease)
• Presentation
  o Bony pain and focal tenderness without recent trauma in a patient with a predisposition (see above), with or without localized erythema, warmth, and swelling
  o Children may refuse to use the involved limb.
  o Systemic symptoms: fever, weight loss, night sweats, fatigue, malaise, and anorexia may or may not be present
• Signs: cover the spectrum from none (normal exam) to general signs of inflammation to overlying skin lesions such as ulcerations to palpable bony sequestrum with draining sinuses

• Diagnosis
  o Lab studies: generally not helpful although the ESR is elevated in 90%; blood cultures are helpful in cases of hematogenous spread
  o Plain films
    ▪ During the first 7 to 10 days, <33% have evidence (lucent lytic areas of cortical bone destruction or periosteal reaction)
    ▪ At 28 days after the onset of symptoms, 90% have abnormal plain radiographs.
  o Bone scans can detect osteomyelitis 48 to 72 hours after the onset of infection.
  o MRI scan with gadolinium contrast provides better anatomic resolution and is comparable to bone scanning for the detection of osteomyelitis.
  o Needle aspiration or bony resection with cultures can also make the diagnosis.

• Special Cases
  o Osteomyelitis in children: usually acute via hematogenous spread involving the long bones 80% of the time; treated with antibiotics alone
  o Brodie’s abscess: a subacute form of osteomyelitis involving the femoral or tibial metaphysis around the knee; caused by S. aureus, Proteus, or Pseudomonas

• Treatment
  o Focused antibiotic therapy
    ▪ As always, directed by cultures and the clinical presentation
    ▪ Initial selection is aimed at treating the most common organism (S. aureus) with a first-generation cephalosporin, nafcillin, or clindamycin
  o Surgical debridement
  o Hyperbaric oxygen therapy may be useful, especially in cases of chronic osteomyelitis.

• Complications
  o The most worrisome complication of vertebral osteomyelitis is spinal epidural abscess.
  o Other complications include septicemia, toxic shock, brain abscess, meningitis, spinal cord compression, pathologic fractures, and pneumonia.

Tumors
• General Information
  o Primary bone tumors are uncommon.
  o Metastatic disease is the most common bone tumor, primarily from breast, lung, and prostate cancer (especially spinal lesions) plus thyroid and kidney carcinoma, multiple myeloma, lymphoma, and leukemia.

• Classic/Benign Tumors of the Bone
  o Osteoma
    ▪ Small flat growths of cortical bone, believed to be localized exaggerations of intramembranous ossification.
    ▪ The skull is the most common site, especially the sinuses, but osteomas are occasionally seen in long bones and/or ribs.
  o Bone island
    ▪ Appears as an asymptomatic, radiodense nodule of compact cortical bone with little clinical significance, as it is benign
    ▪ Typically found in the pelvis, proximal femur, and ribs
CHAPTER 15 • Musculoskeletal Disorders

- Non-ossifying fibroma
  - Appears as a localized defect in bone growth
  - Appear as a fibrous cortical defect (<2 cm in diameter) in the metaphysis of the distal femur, proximal and distal tibia, proximal and distal fibula
  - Especially in children (30%-40% of children have one) after the age of 2; they are mostly seen around 10 years of age and begin to involute around age 14
  - These lesions are rare in adults.

- Osteoid osteoma
  - Common benign tumor of the vertebral column
  - Presentation: back pain at night relieved by NSAIDs
  - Treatment: excision

- Paget's Disease (Osteitis Deformans)
  - An inflammatory condition of the skeleton in which there is rapid, chaotic bone resorption followed by excessive bone formation. The result is enlarged but weakened and highly vascularized bone that is painful, is easily deformed, and fractures with minimal trauma.
  - Presentation: frequently asymptomatic, but patients can have bone pain; cranial and vertebral involvement can cause neurologic deficits
  - Lab studies: alkaline phosphatase is usually elevated.
  - Imaging studies: x-ray films typically show “punched-out” lesions

15.2 DISORDERS OF THE SPINE

Disc Disorders
- General Information
  - Repeated trauma and stress to the nucleus pulposis may result in extrusion of its contents through the annulus fibrosis, most commonly in the posterolateral aspect. This extrusion may compress the exiting spinal nerve, causing
    - Radicular pain (burning, sharp, electric-shock-like pain)
    - Numbness in the dermatome involved
    - Muscle weakness in the muscles supplied by the impinged spinal nerve
  - Rarely, the extrusion is central/directly posterior, causing cord compression (see cauda equina syndrome) and myelopathy.
  - The most common site of disc herniation is the lumbar spine, followed by the cervical spine; disc herniation is rare in the thoracic spine.

- Cervical Spine
  - Disorders in this area, most commonly at C4–C7, occur at a rate of 5.5/100,000 among individuals aged 35 to 55.
  - Presentation
    - Chronic neck pain and radicular shoulder/arm pain; pain is worsened by hyperextension, flexion to affected side, or Valsalva maneuver.
    - There may be a history of antecedent trauma.
    - Spurling's sign (pain worsened by rotation to affected side, ipsilateral flexion, and optional axial loading) is helpful if positive but lacks sensitivity. Discomfort is often relieved by opposite motion, manual distraction of neck, or raising the arm on the affected side.
  - Plain radiographs are of little utility in the ED.
• Lumbar Spine
  o Most common site for disc herniation
  o The herniation is usually lateral (rarely central), with 95% occurring at L4–L5 or L5–S1 disc level, affecting
    the L5 and S1 nerve root, respectively.
    ▪ 10% have herniation at more than one level
  o Presentation
    ▪ Acute or acute on chronic back pain that radiates down the leg on the affected side in a radicular fashion
    ▪ Paresthesias/numbness should occur along the affected dermatome, and muscular weakness should cor­
      respond to the affected nerve root:
        ▪ L3=hip extension
        ▪ L4=quadriceps weakness
        ▪ L5=weak foot or great toe dorsiflexion
        ▪ S1=weak plantar flexion or toe walking
        ▪ Straight leg raise (SLR) testing should elicit or worsen this radicular pain (pain in the back or but­
          tocks or pain not following a distinct dermatome elicited with a SLR constitutes a NEGATIVE sign
          for nerve root impingement).
  o Diagnosis
    ▪ Plain films are indicated only in patients with direct trauma and bony tenderness; in patients with a his­
      tory of malignancy, severe osteoporosis, IVDA, fever, immunocompromise or prolonged symptoms; and
      patients at the extremes of age.
    ▪ MRI is the best test to diagnose lumbar disc herniation.

Inflammatory Spondylopathies
  • General Information
    o The seronegative spondylopathies share the characteristics of SI joint involvement, peripheral inflammatory
      arthropathy, absence of rheumatoid factor, pathologic changes around the ligamentous and tendinous inser­
      tions, and a genetic component related to the HLA-B27 marker.
    o This group includes the following:
      ▪ Ankylosing spondylitis
      ▪ Reactive arthritis including Reiter’s syndrome (arthritis, iritis, urethritis, oral ulcerations)
      ▪ Psoriatic arthritis
      ▪ Arthropathy of inflammatory bowel disease
  • Ankylosing Spondylitis
    o Defined by chronic and progressive inflammatory changes and new bone formation at attachment of tendons
      and ligaments to bone (called enthesopathy)
    o Hallmark is SI joint involvement with variable spinal involvement; 20% to 30% have large joint involvement.
    o White males predominate.
    o Onset: insidious between the ages of 20 and 40
    o Presentation: low back or subgluteal pain, worse at night and better with activity. Other complaints include
      morning stiffness, wakening to "walk off stiffness," worse with rest, loss of motion in all planes of spinal mo­
      tion ("ramrod spine")
      ▪ Extra-articular manifestation: uveitis is most common
    o X-ray films: sclerosis of SI joint, squaring of spinal vertebrae, and ossification of annulus fibrosis ("bamboo
      spine")
    o Treatment: NSAIDs and physical therapy
• Reiter's Syndrome
  o Reactive arthritis that occurs in genetically susceptible individuals after infection with *Chlamydia trachomatis* in the GU tract or with *Salmonella*, *Shigella*, *Yersinia*, or *Campylobacter* in the GI tract
  o Presentation: The arthritis is typically polyarticular and asymmetric, involving the weight-bearing joints of the lower extremity, including the heel.
  o Lab studies
    ▪ Synovial fluid examination is inflammatory with a predominance of polymorphonuclear cells.
    ▪ Rheumatoid factor and ANA are typically negative.
  o Treatment: NSAIDs and a course of a tetracycline for patients whose initial illness was urethritis.

Low Back Pain (LBP)
• General Information
  o Annual incidence in working adults is as high as 50%, with a prevalence of 15% to 20%.
  o Most cases of LBP have a muscular or ligamentous etiology and resolve spontaneously.
  o Conservative treatment with rest, back exercises, and NSAIDs is the norm.
  o Degree of evaluation is primarily related to the possibility of other causes of the pain (e.g., AAA) (Tables 15-1 and 15-2).

<table>
<thead>
<tr>
<th>Table 15-1. Common History and Physical Examination “Red Flags” for Low Back Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient History</strong></td>
</tr>
<tr>
<td>• Recent significant trauma</td>
</tr>
<tr>
<td>• Recent mild trauma in patients older than 50 years of age</td>
</tr>
<tr>
<td>• History of prolonged steroid use</td>
</tr>
<tr>
<td>• History of osteoporosis</td>
</tr>
<tr>
<td>• Patients over 70 years of age</td>
</tr>
<tr>
<td>• Syncope</td>
</tr>
<tr>
<td>• History of cancer</td>
</tr>
<tr>
<td>• History of recent infection</td>
</tr>
<tr>
<td>• Fever</td>
</tr>
<tr>
<td>• Intravenous drug use</td>
</tr>
<tr>
<td>• Immunocompromised state</td>
</tr>
<tr>
<td>• Pain is worse at rest</td>
</tr>
<tr>
<td>• Unexplained weight loss</td>
</tr>
<tr>
<td>• Acute onset of back and/or flank pain</td>
</tr>
<tr>
<td>• Diaphoresis or nausea associated with pain</td>
</tr>
<tr>
<td><strong>Physical Examination</strong></td>
</tr>
<tr>
<td>• Abnormal vital signs – hypotension, tachycardia</td>
</tr>
<tr>
<td>• Unequal blood pressure readings in the upper extremities</td>
</tr>
<tr>
<td>• Pulsatile abdominal mass</td>
</tr>
<tr>
<td>• Pulse deficit or circulatory compromise of the lower extremities</td>
</tr>
<tr>
<td>• Loss of rectal sphincter tone, urinary retention, or focal lower extremity weakness</td>
</tr>
<tr>
<td>• Focal back pain with fever</td>
</tr>
<tr>
<td>Critical Conditions</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Vascular</td>
</tr>
<tr>
<td>Abdominal</td>
</tr>
<tr>
<td>Infections</td>
</tr>
<tr>
<td>Mechanical</td>
</tr>
<tr>
<td>Mechanical</td>
</tr>
<tr>
<td>Mechanical</td>
</tr>
</tbody>
</table>
### Table 15-2. Differential Diagnosis of Low Back Pain, continued

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>History</th>
<th>PE Findings</th>
<th>Ancillary Testing</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emergent Conditions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infectious</td>
<td>Vertebral osteomyelitis</td>
<td>Similar group at risk as for epidural abscess. Onset may be insidious. Back pain, tenderness, and stiffness may precede neurologic findings by a significant amount of time.</td>
<td>CBC, blood cultures generally low yield. Plain films are diagnostic in 80%-95% of cases, but MRI is more accurate and detailed.</td>
<td>Biopsy may be necessary for diagnosis. <em>S. aureus</em> is common.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fever and other constitutional symptoms. Localized body tenderness of two adjacent vertebrae.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Immune</strong></td>
<td>Transverse myelitis</td>
<td>Back pain and neurologic deficits. Almost 50% of patients worsen maximally in 24 hours.</td>
<td>Partial/total loss of sensory, motor, autonomic, and sphincter function below the level of the lesion. Leg weakness is more common; arm involvement is rare. Bladder (bowel) control is involved in most patients.</td>
<td>Goal is to rule out mass lesion compressing the cord. Thought to be of autoimmune origin. MRI is the imaging modality of choice. Contrast CT and CT myelogram may be obtained. May be associated with multiple sclerosis, SLE, sarcoidosis. Also associated with Lyme disease, Epstein-Barr virus, and following other viral (herpes, enterovirus) or bacterial (TB, syphilis) infections.</td>
</tr>
<tr>
<td><strong>Mechanical</strong></td>
<td>Back pain with neurologic deficits</td>
<td>Most patients recall an atraumatic mechanism (lifting, twisting). Common complaints are stiffness, tenderness, decreased range of motion.</td>
<td>Positive straight leg raise test Muscular weakness Potential for sensory deficits Absent or diminished deep tendon reflexes</td>
<td>Selective use of plain films. CT or MRI performed for complete assessment. Search for “red flags” to rule out serious underlying disease.</td>
</tr>
<tr>
<td></td>
<td>Intervertebral disk herniation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Spinal stenosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Spinal fractures w/o cord impingement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Malignancy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sciatic with potential of nerve root compression</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
• **Cauda Equina Syndrome**
  
  o **Causes:** central herniated nucleus pulposus (HNP), abscess, hematoma, lumbar spondylosis/spondylolisthesis or stenosis with nerve compression
  
  o **Presentation:** chronic dull aching pain in saddle distribution, involving the perineum and sacral area. 90% have some degree of urinary retention (may present as overflow urinary incontinence) with or without fecal incontinence and decreased rectal tone.
  
  o **Management:** emergent MRI followed by emergent decompression laminectomy

• **Spinal Stenosis**
  
  o **Causes:** narrowing of canal, usually from degenerative joint disease (DJD)
  
  o **Presentation**
    - Lower extremity pain exacerbated by walking, especially uphill, and relieved by sitting (easily confused with vascular claudication) or leaning forward (e.g., with a shopping cart in the grocery store)
    - Symptoms can be reproduced with spinal extension and relieved with spinal flexion (differentiates spinal stenosis from vascular claudication).
  
  o **Diagnosis:** aided by CT or MRI

• **Spinal Epidural Abscess**
  
  o **Infectious process confined to adipose tissue of the dorsal epidural space**
  
  o **Predisposing factors:** DM, IVDA, surgery or instrumentation to area (steroid injection), alcoholism, and immunosuppression
  
  o **Etiology:** *S. aureus* is most common.
  
  o **Presentation:** backache progressing to local back tenderness with fever, sweats, and rigor, progressing to neurologic compromise. 10% present with encephalopathy secondary to local extension of the infection.
  
  o **Diagnosis:** MRI is the diagnostic modality of choice.
  
  o **Treatment:** immediate neurosurgical consultation for decompression; IV vancomycin and a third-generation cephalosporin

• **Spondyloysis**
  
  o **A defect in the pars interarticularis; unclear if congenital or traumatic**
  
  o **Not treated unless slippage occurs**

• **Spondylolisthesis**
  
  o **Slippage of one vertebra on another in association with spondyloysis**
  
  o **Causes:** combination of factors, including trauma
  
  o **Stages classified by percentage of slip**
    - I, <25%
    - II, 25% to 50%
    - III, 50% to 75%
    - IV, >75%
  
  o **Greater slip leads to increased likelihood of neurologic compromise**
15.3 JOINT ABNORMALITIES

Arthritis

- General Information
  - The differential diagnosis based on the pattern of involved joints is summarized in Table 15-3.

Table 15-3. Joint Pattern and Differential Diagnosis of Arthritis

<table>
<thead>
<tr>
<th>Number of Joints</th>
<th>Differential Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Monoarthritis</td>
<td>Trauma-induced arthritis</td>
</tr>
<tr>
<td></td>
<td>Infection/septic arthritis</td>
</tr>
<tr>
<td></td>
<td>Crystal-induced (gout, pseudogout)</td>
</tr>
<tr>
<td></td>
<td>Osteoarthritis (acute)</td>
</tr>
<tr>
<td></td>
<td>Lyme disease</td>
</tr>
<tr>
<td></td>
<td>Avascular necrosis</td>
</tr>
<tr>
<td></td>
<td>Tumor</td>
</tr>
<tr>
<td>2–3 = Oligoarthritis</td>
<td>Lyme disease</td>
</tr>
<tr>
<td></td>
<td>Reiter's syndrome</td>
</tr>
<tr>
<td></td>
<td>Ankylosing spondylitis</td>
</tr>
<tr>
<td></td>
<td>Gonococcal arthritis</td>
</tr>
<tr>
<td></td>
<td>Rheumatic fever</td>
</tr>
<tr>
<td>&gt;3 = Polyarthritis</td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>Systemic lupus erythematosus</td>
</tr>
<tr>
<td></td>
<td>Viral arthritis</td>
</tr>
<tr>
<td></td>
<td>Osteoarthritis (chronic)</td>
</tr>
<tr>
<td>Migratory joint pattern</td>
<td>Gonococcal arthritis</td>
</tr>
<tr>
<td></td>
<td>Acute rheumatic fever</td>
</tr>
<tr>
<td></td>
<td>Lyme disease</td>
</tr>
<tr>
<td></td>
<td>Viral arthritis</td>
</tr>
<tr>
<td></td>
<td>SLE</td>
</tr>
</tbody>
</table>

- Age and gender patterns
  - Septic arthritis is an important consideration in infants and children.
  - Sexual activity increases the likelihood of GC and Reiter's syndrome secondary to chlamydial infection.
  - Crystal arthropathies affect middle-aged individuals.
  - Gout is the most common inflammatory joint disease in men over 40.
  - Osteoarthritis generally occurs in people older than 60.
  - Rheumatoid arthritis emerges earlier, with the prevalence among women 3 or 4 times higher than among men.
- Septic Arthritis
  - Three mechanisms of contamination: 1) hematogenous spread of bacteria, 2) migration of bacteria from a focus contiguous to a joint, 3) direct inoculation of bacteria into the joint
  - Presentation: Constitutional symptoms are NOT always present but MUST be considered in any patient with monoarticular arthritis.
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- Diagnosis: The joint must be tapped. Joint fluid analysis results are categorized in Table 15-4.

Table 15-4. Joint Fluid Findings in Various Causes of Arthritis

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Noninflammatory</th>
<th>Inflammatory</th>
<th>Septic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clarity</td>
<td>Transparent</td>
<td>Transparent</td>
<td>Cloudy</td>
<td>Cloudy</td>
</tr>
<tr>
<td>Color</td>
<td>Clear</td>
<td>Yellow</td>
<td>Yellow</td>
<td>Yellow</td>
</tr>
<tr>
<td>WBC/ml</td>
<td>&lt;200</td>
<td>&lt;200–2000</td>
<td>200–50,000</td>
<td>&gt;50,000</td>
</tr>
<tr>
<td>PMNs (%)</td>
<td>&lt;25</td>
<td>&lt;25</td>
<td>&gt;50%</td>
<td>&gt;50%</td>
</tr>
<tr>
<td>Culture</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>&gt;50% positive</td>
</tr>
<tr>
<td>Crystals</td>
<td>None</td>
<td>None</td>
<td>Osteoarthritis, trauma, rheumatic fever</td>
<td>None</td>
</tr>
<tr>
<td>Associated conditions</td>
<td>Osteoarthritis, trauma, rheumatic fever</td>
<td>Gout, pseudogout, spondyloarthropathies, RA, Lyme disease, SLE</td>
<td>Nongonococcal or gonococcal septic arthritis</td>
<td></td>
</tr>
</tbody>
</table>

Note that the WBC counts and PMN percentages vary widely. Early in septic arthritis, they may be lower than the values listed.

- Treatment: If tap is nondiagnostic or positive, admit the patient for IV antibiotics, and obtain orthopedic consult. Etiologic agents and the appropriate antibiotics are listed in Table 15-5.

Table 15-5. Antimicrobial Selections for Septic Arthritis Based on Bacterial Cause

<table>
<thead>
<tr>
<th>Patient Age/Condition</th>
<th>Usual Organisms</th>
<th>Antimicrobial Selections</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates and infants</td>
<td><em>Staphylococcus</em>, gram-negative bacteria, group B <em>Streptococcus</em>, <em>Candida</em></td>
<td>Penicillinase-resistant penicillin (PRP) plus third-generation cephalosporin (3-Ceph); PRP plus anti-pseudomonal aminoglycoside (APAG)</td>
</tr>
<tr>
<td>Children</td>
<td><em>Staphylococcus</em>, <em>Haemophilus influenzae</em></td>
<td>PRP plus 3-Ceph</td>
</tr>
<tr>
<td>Older children and healthy adults</td>
<td><em>Staphylococcus</em>, gonococcus, <em>Streptococcus</em></td>
<td>PRP alone; 3-Ceph alone; vancomycin plus 3-Ceph; penicillin plus aminoglycoside; penicillin plus 3-Ceph</td>
</tr>
<tr>
<td>Involvement of the foot</td>
<td><em>Staphylococcus</em>, <em>Pseudomonas</em></td>
<td>PRP plus ceftazidime; PRP plus APAG</td>
</tr>
<tr>
<td>Intravenous drug users</td>
<td><em>Staphylococcus</em>, gram-negative bacilli</td>
<td>PRP plus APAG; PRP plus fluoroquinolone</td>
</tr>
<tr>
<td>Sickle-cell patients</td>
<td><em>Salmonella</em></td>
<td>PRP plus 3-Ceph; fluoroquinolone alone</td>
</tr>
</tbody>
</table>

*If MRSA is prevalent, add vancomycin (or substitute it for PRP) in all of the above regimens.

- Gonococcal Arthritis
  - Most common cause of septic arthritis in adolescents and young adults
  - Presentation: prodrome of migratory arthritis and tenosynovitis—then settles in one or more joints. Look for vesiculopustular lesions, especially on the palm and fingers.
  - Diagnosis: Synovial fluid cultures are frequently negative (needs to be plated immediately on chocolate agar). Add swabs from pharynx, urethra, rectum, and cervix to increase yield.
  - Treatment: Admit for parenteral therapy until clinical improvement is seen, then PO for another 7 to 10 days, with IV ceftriaxone every 8 hours, supplemented with oral doxycycline.
• Crystal Arthropathies
  o General Information
    ▪ Typically involve older men with precipitating factors, e.g., trauma, surgery, significant illness, change in medications
    ▪ Predilection: Lower extremities, but the condition shows no predilection for any joint, even though the first MTP joint is “classic” for gout
    ▪ Diagnosis: fluid aspiration and seeing typical crystals WITHIN phagocytes
    ▪ First-line treatment: NSAIDs, typically indomethacin, with significant pain relief being seen within 2 hours
  o Gout
    ▪ Pathology: uric acid, needle-shaped crystals with bright negative birefringence – “pretty young girls” mnemonic = parallel, yellow (appears light blue when perpendicular), gout
    ▪ Presentation: typical attack comes on over a few hours; skin overlying joint is typically sensitive/tender
      ▪ Classic symptom: sheet causes pain
    ▪ Diagnosis
      ▪ Serum uric acid levels are not diagnostic – can be normal during an attack
      ▪ Crystals are NOT always seen in the joint fluid.
    ▪ Treatment: Consider colchicines acutely (0.6 mg/hr until relief or emergence of the side effects of nausea or diarrhea) followed by NSAIDs +/- long-term treatment with allopurinol or probenecid.
  o Pseudogout
    ▪ Pathology: calcium pyrophosphate – rhomboid shaped with weak positive birefringence, blue when parallel and yellow when perpendicular
    ▪ Presentation: attack comes on more slowly, over days
    ▪ Treatment: the same as gout
• Osteoarthritis
  o Progressive deterioration of articular cartilage
  o Distinguished from other types of arthritis by lack of systemic symptoms and/or lack of other organ involvement
  o Presentation: distal interphalangeal (DIP) joint involvement is classic, with Heberden's nodes; involvement of the proximal interphalangeal (PIP) joint is seen less frequently, with Bouchard's nodes
  o Treatment: Acetaminophen is the first choice, followed by NSAIDs for short periods to manage exacerbations; no need for steroids
• Osteochondritis Dissecans
  o A rare disorder with unknown cause
  o A segment of articular cartilage and underlying bone becomes detached
  o Typically seen in the femoral condyles (medial-lateral), the patella, the dome of the talus, and the medial epicondyle of the humerus (“little league” elbow)
  o Seen in children and young adults, with a predilection for males
  o Presentation: intermittent joint pain, stiffness, locking
  o Treatment: conservative with rest and rehabilitation; if a loose body has formed, it must be removed
CHAPTER 15 • Musculoskeletal Disorders

15.4 MUSCLE ABNORMALITIES

Myalgia/Myositis

- Myositis Ossificans
  - Inappropriate ossification, usually of muscle
  - Usually follows trauma (66%), but one third of cases have no known cause
  - Presentation: a painful, hard, swollen area in the traumatized muscle, which is sometimes red and warm
  - Diagnosis: a calcific mass in the muscle on radiographs
  - Treatment: nonurgent orthopedic consultation, as the mass may require excision

Rhabdomyolysis

- Causes
  - Alcohol, drugs (statins, cocaine, amphetamines, other sympathomimetics), trauma/crush injury, compartment syndrome (cause or complication), infection, strenuous exertion, seizures, heat-related conditions, metabolic myopathies, hypoxia, electrolyte disorders; also seen in 10% of electrocutions and may be seen with reperfusion of an ischemic limb
  - Alcohol is the cause or contributing factor in 20% of cases.

- Pathophysiology
  - Terminal event is damage to sarcolemma, with loss of normal pump functions, causing elevated intracellular calcium and subsequent necrosis.

- Presentation
  - Typically includes weakness, stiffness, myalgias, malaise, fever, and brown urine
  - In severe cases, patients may exhibit nausea, vomiting, abdominal pain, and tachycardia.
  - Some patients may be seen with NO symptoms or findings. The diagnosis must be suspected based on history and lab results.

- Diagnosis
  - Suspect in patients with urine positive for blood but no RBCs
  - Elevated creatine kinase (CK) (five times normal) is the most sensitive test. CK rises for 24 to 36 hours and then falls at constant rate of approximately 39% per day. If not, suspect ongoing damage.
  - Myoglobin may return to normal within 1 to 6 hours. A normal myoglobin level does NOT exclude rhabdomyolysis because of its lack of sensitivity.

- Treatment
  - Saline infusion initially supplemented with urine alkalinization, mannitol, and dialysis
    - Initial boluses to restore hydration and maintain urine output at 2 to 3 ml/kg/hr
    - Alkalinization of the urine (add 1 or 2 amps of sodium bicarbonate to 1 L 0.45 NS), with a goal of maintaining output at 2 to 3 ml/kg/hr output and pH >6.5, may aid clearance of myoglobin.
    - Mannitol, 1 gm/kg over 30 minutes, and/or furosemide
    - Watch electrolytes – hyperkalemia, hypocalcemia, hyperphosphatemia

- Complications
  - Worst complication is acute renal failure (ARF) (rhabdomyolysis is the cause of 5%–15% of cases of ARF); may require dialysis
15.5 OVERUSE SYNDROME

Bursitis
- Causes: overuse or direct trauma
- Presentation: pain, swelling, +/- warmth over extensor surface of elbow, the lateral surface of the shoulder, and the prepatellar surface of the knee
- Diagnosis: Straightforward bursitis must be distinguished from septic or crystalline bursitis through aspiration of bursal fluid (avoid aspiration if the patient might have overlying cellulitis).
- Treatment: Avoid further trauma, administer NSAIDs, and apply compression.
  - If septic, 14 days of oral antibiotic (first-generation cephalosporin)
  - If not septic, refer for possible steroid injection

Chondromalacia Patellae
- Seen in young active women
- Presentation: knee pain, especially when going up steps, due to chronic overuse syndrome involving the patellar cartilage. Patellar grind and apprehension tests are positive.
- Treatment: NSAIDs and quad strengthening

Epicondylitis
- Lateral Epicondylitis
  - Associated with inflammation at the insertion of the extensor carpi radialis brevis
  - Predisposing factors: also known as tennis elbow (secondary to poor technique) and associated with hammering
  - Treatment: rest, NSAIDS, improved technique (virtually 100% eliminated by two-hand backhand), and compression apparatus
- Medial Epicondylitis
  - Predisposing factors: also known as golfer’s elbow; this syndrome is also seen in racquet sports and pitching
  - Presentation: severe pain over the medial epicondyle; two thirds of patients have concomitant cubital tunnel syndrome
  - Treatment: NSAIDs, rest, and compression, with activity resumed after 6 weeks

Rotator Cuff Injuries
- Shoulder pain is the most common presenting complaint in patients over 40
- Overuse injury is more common than traumatic
- Examination should evaluate the four muscles of the rotator cuff, the SITS group (the first three insert at the greater tuberosity):
  - Supraspinatus (abduction, most commonly injured)
  - Infraspinatus (external rotation)
  - Teres minor (external rotation)
  - Subscapularis (internal rotation)
- Presentation
  - Weakness to external rotation and abduction
  - Neer’s test (forced flexion of the shoulder while it is maximally internally rotated)
  - Hawkins’ test (flex the shoulder to 90 degrees and forcibly internally rotate the shoulder)
  - The “drop arm” test (patient is unable to hold the arm up when abducted to 90°)
• Treatment
  o 50% of patients do well with conservative treatment consisting of rest/modification of activity (do not immobilize), NSAIDs, ice, ROM and strengthening exercises
  o If conservative treatment fails, follow-up referral to orthopedics for possible surgical repair

Synovitis/Tendinitis/Tenosynovitis
• Patellar Tendonitis
  o Predisposing factors: referred to as “jumper’s knee” in runners, basketball and volleyball players, and high jumpers
  o Presentation: a tender, swollen tendon with pain that is worse going from sitting to standing or climbing
  o Treatment: conservative treatment, with rest, ice, and NSAIDs; avoid injecting steroids, as this may lead to tendon rupture

• DeQuervain’s Tenosynovitis
  o Tenosynovitis of extensor pollicis brevis and abductor pollicis at the wrist
  o Presentation: Finkelstein’s test (grasping the thumb in the fist and ulnar deviating the wrist produces pain)
  o Treatment: Splint for 3 weeks; daily ROM exercises; NSAIDs for 10 to 14 days; if not improving, refer to orthopedics for steroid injection into sheath

• Trigger Finger
  o Causes: scarring from synovitis
  o Treatment: steroid injection and specialty referral

• Dupuytren’s Contracture
  o Fibroplastic changes in tissues of palm, with nodular thickening and contracture starting on the ulnar side (small finger)
  o Treatment: specialty referral for surgical removal

Nerve Compression Syndrome—caused by compression, repetitive friction, or stretching of the nerve
• Ulnar Nerve Compression
  o General information
    ▪ Numbness or weakness in the ring and small fingers
    ▪ Initially treated with rest, NSAIDs, +/- splinting; if not improving, the patient may need decompression surgery
  o Cubital tunnel syndrome
    ▪ Entrapment of the ulnar nerve at the elbow
    ▪ Predisposing factors: golf or pitching
    ▪ Presentation: Positive Tinel’s sign at the elbow and Phalen’s sign with the elbow flexed. Patients may also have thumb adduction weakness. Look for Froment’s sign—Ask the patient to hold a paper between the thumb and index finger proximal phalanx. A need to bend the thumb IP joint represents weakness.
  o Guyon’s Canal (Ulnar Tunnel) Syndrome
    ▪ Associated with direct compression of the ulnar nerve as it enters the hand (e.g., cyclists gripping the handlebars)
    ▪ Presentation: The results of Phalen’s and Tinel’s tests are similar to those associated with carpal tunnel syndrome, except the symptoms/findings are in the ulnar distribution.
    ▪ Rare fixed deficits result in ulnar claw hand
Medial Nerve Compression
  - Carpal tunnel syndrome
    - The most common compression syndrome typically occurring in patients 40 to 60 years old (females > males 3:6:1)
    - Presentation
      - Burning and tingling in hand and fingers, which may extend up the arm
      - Decreased sensation in the palm and volar fingertips
      - Chronic, severe disease results in thenar wasting
      - Phalen's and Tinel's signs are not reliable in the absence of other findings.
    - Treatment: rest, NSAIDs, avoidance of precipitating activities, and use of wrist cock-up splints; referral for consideration of further diagnostics/surgical decompression

15.6 SOFT TISSUE INFECTIONS

Necrotizing Fasciitis
  - Spectrum of disease, beginning with cellulitis and eventually involving the skin, SQ tissue, and fascia
  - Causes: penetrating trauma, extension from deep soft tissue infection (e.g., perirectal abscess), recent surgery
  - Etiology: mixed anaerobe and aerobes (non-group A streptococci plus anaerobes versus group A β-hemolytic streptococci)
  - Predisposing factors: diabetes, poor circulation, immunocompromise, trauma
  - Presentation
    - Red, moderately tender skin (pain out of proportion without history of trauma or injury is an early finding), +/- blisters and areas of necrosis, severe swelling, +/- palpable gas
    - Systemic toxicity is moderate to severe.
  - Treatment: emergent wide excision, antibiotics, and possibly hyperbaric oxygen (HBO) therapy
    - Antibiotic choice is guided by gram stain findings or location, indicating the likely organism.
    - Start with penicillin, an aminoglycoside, and clindamycin.
    - Consider vancomycin if MRSA is suspected.
  - Prognosis: despite treatment, mortality is 35%

Myonecrosis
  - Similar to necrotizing fasciitis, except involvement now includes muscle
  - Causes and predisposing factors: similar to those of necrotizing fasciitis
  - Etiology: Organisms are similar to those that cause necrotizing fasciitis, with the addition of Clostridium.
  - Presentation: The involved area appears blanched, with massive swelling and frank necrosis/gangrene. Gas is common. Pain is severe, as is systemic toxicity.
  - Treatment: like necrotizing fasciitis, but usually wider (radical) excision.
  - Prognosis: mortality >25%

Gangrene
  - Fournier's Syndrome (see Image #2)
    - Insidious necrotizing subcutaneous infection of the perineum, resulting in acute dermal gangrene; commonly seen in men aged 20 to 50
    - Etiology: aerobic (E. coli) and anaerobic (B. fragilis) bacteria (commonly of the distal colon)
    - Treatment: consists of resuscitation, broad-spectrum antimicrobials, and surgical debridement
    - Prognosis: Mortality is as high as 35%.
• Meleney's Synergistic Gangrene
  o Superficial and deep fasciitis with thrombosis of subcutaneous vessels and gangrene of the tissues; usually secondary to a laceration or surgical wound
  o Etiology: group A streptococci or staphylococci

• Clostridial Cellulitis
  o Gas-producing, anaerobic suprainfection of previously traumatized or necrotic tissue that spreads along intrafascial planes
  o Treatment: penicillin or tetracycline

Hand Infections
• Etiology
  o Staphylococcus and Streptococcus are most common overall.
  o Consider S. aureus if the patient is an IVDA.
  o Paronychia and felon are frequently caused by mouth organisms, including anaerobes.

• Paronychia
  o Commonly seen in nail biters, this infection of the nail complex may present as cellulitis or a small abscess
  o Etiology: mouth organisms, including anaerobes
  o Treatment
    - If not fluctuant, try warm soaks and antibiotics (first-generation cephalosporin)
    - If pus/fluctuance, treatment is I&D; antibiotics are not indicated if there are only minimal signs of surrounding cellulitis

• Felon (see Image #56)
  o Infection involving the pulp space of the volar distal phalynx (often the extension of an untreated paronychia)
  o Etiology: commonly caused by S. aureus but also mouth organisms
  o Presentation: volar swelling, redness, pain
  o Treatment
    - Most can be treated with a lateral incision, avoiding the digital nerve and artery. Pack the wound, prescribe a first-generation cephalosporin, and refer the patient to a hand surgeon.

Tenosynovitis
• Flexor Tenosynovitis
  o Infection of the tendon sheath is a surgical emergency.
  o Causes: puncture wound, animal bite, or "fight bite" (see Image #27)
  o Etiology: Staphylococcus is most common.
  o Presentation: classic signs of Kanavel include symmetric swelling of finger, pain on passive extension, tenderness over tendon sheath, and finger held in a flexion posture
  o Treatment: Initiate antibiotics (nafcillin, oxacillin, or cefazolin) and call a hand surgeon for I&D.

15.7 ORTHOPEDIC TRAUMA

Initial Evaluation and Management
• Immediately assess any orthopedic injury for the following critical features:
  o Neurovascular compromise
  o Compartment syndrome
  o Dislocation
  o Risk of infection (high with open fractures)
• Immediately reduce and stabilize unstable/compromised fractures; administer antibiotics, tetanus/TIG, and analgesics as indicated; obtain immediate consultation for critical complications; remove any constricting objects; and elevate and ice as possible.

• Order appropriate radiographs based on H&P and mechanism, always considering the joint above and below the injury.
  o Use well-validated decision rules (Ottawa Ankle Rules, Nexus criteria) when possible to avoid excessive radiation exposure, especially in children and pregnant women.
  o Do not accept inadequate studies.

• Immobilize all fractures (including occult fractures with normal radiographs) to prevent further injuries, preferentially with plaster splints or bi-valved casts to reduce the incidence of compartment syndrome.

• Consider specialized imaging techniques in patients with
  o Complicated fractures
  o Neurovascular compromise
  o Negative plain films with a high index of suspicion or gross function deficit such as being unable to bear weight

• Traumatic hemarthroses carry a high incidence of ligamentous and/or bony injury.
  o Aspirate significant hemarthroses, then ice, rest, analgesics, and orthopedic follow-up
  o Consider a bleeding disorder in a patient with spontaneous hemarthrosis and treat appropriately (do not aspirate).

Salter-Harris Pediatric Fractures (Figure 15-1)

Figure 15-1. Salter-Harris pediatric fractures.

• Types and percentage occurrences are listed in Table 15-6. (See Image #23 for a Salter-Harris III fracture.)

Table 15-6. Salter-Harris Pediatric Fractures: Types and Percentage Occurrence

<table>
<thead>
<tr>
<th>Type</th>
<th>Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>6%</td>
</tr>
<tr>
<td>II</td>
<td>75%</td>
</tr>
<tr>
<td>III</td>
<td>&lt;10%</td>
</tr>
<tr>
<td>IV</td>
<td>10%</td>
</tr>
<tr>
<td>V</td>
<td>1%</td>
</tr>
</tbody>
</table>

• Type I fractures may present with pain and swelling over the physeal plate, with normal radiographs. Although Type I fractures are at low risk for growth plate injury and associated morbidity. Prudent therapy involves immobilization and reexamination in 7 to 10 days.
Orthopedic Emergencies – must be assessed and treated immediately
- Open fractures – infection risk
- Dislocations (shoulder>patella>elbow)
- Neurovascular compromise
- Compartment syndrome

### 15.8 HAND AND DIGITS INJURIES

#### Neuroanatomy (Figure 15-2)

**Digital Nerve**
- Sensory: provides sensation for the digit, as measured by two-point discrimination (should be <5 mm)
- Anatomically located two thirds of the finger depth from the dorsal surface on each side of the digit
- Always repaired if lacerated proximal to the PIP joint or proximal to the DIP joint on the radial side of the index finger, the ulnar side of the small finger, or proximal to the thumb IP joint

**Median Nerve**
- Sensory: provides volar sensation to palm; thumb; and the index, the long, and radial side of the ring finger
- Motor: flexes wrist, fingers, and thumb; opposes the thumb; and flexes the elbow

**Ulnar Nerve**
- Sensory: sensation to ulnar (medial) side of hand
- Motor: abduction and adduction of fingers and flexion of small finger distal interphalangeal (DIP) joint

**Radial Nerve**
- Sensory: radial (lateral) forearm and hand
- Motor: extends wrist/fingers/thumb and abducts thumb

#### Tendon Injuries

**General Information**
- Initial exam should view the tendon in a bloodless, well-anesthetized field over its full range of motion to avoid missing “hidden” lacerations
- Flexor tendon injuries are more complicated and associated with stronger muscles, thus requiring more complex/stronger repairs. Patients with this type of injury should be referred to a hand/plastic surgeon for repair after appropriate wound care.

![Figure 15-2. Sensory innervation of the hand.](image-url)
Extensor tendon lacerations proximal to the metacarpophalangeal joints may be repaired using a buried figure-of-eight stitch after appropriate wound care +/- antibiotics.

- Mallet Finger (Figure 15-3, see Image #62)

![Figure 15-3. Mallet finger.](image)

- Loss of the extensor tendon at the base of the distal phalanx, resulting from an avulsion fracture at the base of phalanx or a tendon tear or laceration
- Treatment: Splint in extension and orthopedic follow-up.
- Complications: chronic untreated can lead to Swan-neck deformity

- Boutonniere Deformity (Figure 15-4.)

![Figure 15-4. Boutonniere deformity.](image)

- Avulsion fracture/tendon tear/laceration of the central extensor mechanism, with volar displacement of lateral components
- Treatment: splint in extension and orthopedic follow-up

Ligamentous/Dislocation Injuries

- Distal Interphalangeal (DIP) Joint
  - Uncommon type of dislocation; usually dorsal
  - Treatment: easily relocated unless the volar plate becomes interposed, which then requires open reduction

- Proximal Interphalangeal (PIP) Joint
  - Common site of dislocation; typically dorsal and lateral, with ulnar >> radial (six times more)
  - Treatment: Check strength and motion after reduction to ensure volar plate stability; consult if abnormal or unstable (possible ORIF)

- Metacarpophalangeal (MCP) Joint
  - Less common than PIP joint injuries
  - Subluxations appear hyperextended.
    - Reduce with dorsal pressure and flexed wrist
  - Complete dislocations usually have volar plate interposed
    - Require operation
• Carpometacarpal (CMC) Joint
  o Uncommon, frequently caused by motor vehicle crash (MVC)
  o Presentation: usually dorsal and commonly associated with a fracture
  o Treatment: requires orthopedic consult

Thumb Injuries
• Interphalangeal (IP) Joint
  o Rare, usually open, requiring ORIF; otherwise, same as the DIP joint of other digits

• Metacarpophalangeal (MCP) Joint
  o Hyperextension injury with dorsal displacement and volar plate disruption
  o Treatment: Reduce with pressure on the base of the proximal phalanx.

• Gamekeeper’s – Skier’s
  o Disruption of ulnar collateral ligament, usually at insertion on proximal phalanx
  o Diagnosis: Test in extension and 30° flexed.
  o Treatment: Those with >40° radial deviation need consultation, usually surgical repair within 1 week.

Fractures
• Distal Phalanx
  o 15% to 30% of all hand fractures
  o Presentation: frequently associated with subungual hematomas
  o Treatment: Treat as soft tissue injury.
    ▪ No evidence to support removal of the nail and nail bed repair, especially if nail is anatomically aligned
    ▪ Splint for contact protection.

• Middle and Proximal Phalanx
  o Treatment
    ▪ Stable fractures: Splint and refer to orthopedics (Figure 15-5).

  Figure 15-5. BOH (blade of the hoe) position for splinting wrist.

  ▪ Unstable fractures: frequently require ORIF

• Metacarpals II–V
  o General information
    ▪ Second and third have limited mobility, requiring anatomic reduction (maximum 10–20° of angulation)
    ▪ Fourth and fifth have 15 to 20° of AP motion and thus are more forgiving (maximum 30–40° of angulation)
  o Metacarpal head
    ▪ Mechanism: direct blow or crash
    ▪ Consider fight bite (see Image #27)
    ▪ Treatment: Splint and refer to orthopedics.
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- Metacarpal neck
  - Boxer’s type
  - Treatment: splint
    - Angulations of 15°, 15°, 20°, and 40° are tolerated (or second through fifth, respectively)
    - Check for rotational deformity – NONE tolerated

- Metacarpal shaft
  - Mechanism: direct blow or rotational injuries
  - Treatment: Surgical fixation is common, owing to rotational deformity or shortening.

- Metacarpal base
  - Mechanism: direct blow or axial load
  - Presentation
    - Associated carpal fractures are common.
    - With fourth and fifth metacarpal bases, look for ulnar motor paralysis; check abduction of small finger
  - Treatment: Splint and refer for orthopedic follow-up.

- Thumb metacarpal
  - Bennett’s fracture (simple fracture with dislocation) and Rolando fracture (comminuted)
  - Mechanism: Both are induced by axial load on thumb.
  - Both are intra-articular fractures at the base of the metacarpal.
  - Treatment: require thumb spica and surgical referral

Fight Bite (see Image #27)
- Assume with all lacerations over dorsum of MCP joints and inquire about possible salivary exposure.
- Perform careful inspection in a bloodless field, looking for tendon injury or joint involvement.
- Treatment: typically requires consultation with a hand surgeon; irrigate, dress open, apply antibiotics, splint, and bring back in 2 days for recheck

15.9 WRIST INJURIES

General Information
- Children are more inclined to sustain physis injuries.
- Fall on outstretched hand (FOOSH) is the most common, with injury based on age.
- Landmarks to check on the AP x-ray film (Figure 15-6)

![Figure 15-6. AP x-ray view of the wrist.](image-url)
• Check alignment of 3-C’s, 15-25° ulnar inclination of the radius, radial styloid protrudes 8 to 18 mm beyond the ulna

• Landmarks to check on the lateral x-ray film (Figure 15-7)

![Figure 15-7. Lateral x-ray view of the wrist.](image)

• FIRST - be sure is true lateral
  - Superimposition of the four ulnar metacarpals
  - Superimposition of the proximal pole of the scaphoid on the lunate and triquetrum
  - The radial styloid centered over its distal articular surface

• Check alignment of 3-C’s

• Anatomic Issues
  - Lister’s tubercle - small dorsal bony prominence on the distal radius that the ext. pollicis longus wraps around; just ulnar to the snuff box
  - Just distal to Lister’s tubercle – scapholunate joint
  - Ulnar to Lister’s tubercle, slight depression – radioulnar joint
  - Proximal carpal row forms mobile link for wrist, which is potentially unstable

Carpal Dislocations – by increasing force

• Scapholunate
  - Most common ligamentous injury
  - Diagnosis: Terry Thomas sign (see Image #65), dorsal intercalated segment instability (DISI) pattern (Figure 15-8)

![Figure 15-8. Scapholunate dislocation.](image)

• Treatment: radial gutter, orthopedic follow-up
• Triquetral-Lunate
  o Less common
  o Diagnosis: volar intercalated segment instability (VISI) pattern (Figure 15-9), may see widening of triquetra-lunate space

![Figure 15-9. Triquetral-Lunate dislocation.](image)

• Perilunate
  o Diagnosis: radiographically, 3-C's are disrupted, both AP and lateral (see Image #7)
    ▪ AP – capitolunate joint is obliterated and scaphoid is shortened
    ▪ Lateral – capitate is pushed behind lunate
  o Frequently associated with carpal fractures
    ▪ Usually scaphoid and capitate (named by adding "trans" to the fractured bone, e.g., transscaphoid perilunate dislocation)
  o Treatment: immediate orthopedic evaluation

• Lunate
  o Diagnosis
    ▪ Radiographically, 3-C's are disrupted, both AP and lateral
    ▪ "Piece of pie" on AP – pathognomonic
    ▪ Lateral view shows lunate pushed off radius; described as "spilled teacup"
  o Treatment: immediate orthopedic evaluation

Carpal Fractures (in order of occurrence)

• General information
  o 7% to 10% of wrist injuries
  o Frequently missed

• Scaphoid
  o Most common fracture
  o Presentation: snuffbox and axial load tenderness
  o Imaging studies: scaphoid views on x-ray film- miss 25%
    ▪ Two thirds of these fractures are across the waist>proximal>distal
    ▪ Considered unstable if >1 mm distraction, rotation, angulation, shortening, or carpal instability pattern
  o Treatment
    ▪ Stable or occult = short arm thumb spica splint and orthopedic follow-up
    ▪ Unstable = orthopedic consult, long arm thumb spica splint
Complications: high risk of avascular necrosis (about 3%) and non-union due to disruption of distal blood supply; requires careful and repeated radiographic evaluation and possibly bone scan or MRI

- Triquetrum
  - Dorsal avulsion (second most common) associated with hyperextension shear or resisted twisting motion
  - Presentation: dorsal tenderness distal to the ulnar styloid
  - Imaging studies: most commonly seen as a bone fleck on the lateral radiograph
  - Body fracture: associated with direct blow to bone or associated with lunate/perilunate dislocations; best radiographic view is AP
  - Treatment: splint for 6 weeks

- Lunate
  - Third most common
  - Diagnosis: rarely an isolated fracture but difficult to see on x-ray film; maintain a high index of suspicion in patients with mid dorsal wrist tenderness
  - Treatment: thumb spica splint and orthopedic follow-up are necessary because of the risk of avascular necrosis
    - Kienböck's disease is seen in about 20% of cases

- Trapezium
  - Mechanism: seen with direct blow to thumb or dorsi-radial flexion
  - Diagnosis: seen radiographically on the oblique view; clinically presents as tenderness at the base of the thumb
  - Treatment
    - Non-displaced fractures are treated with a thumb spica splint.
    - Displaced fractures require ORIF.

- Pisiform
  - Flexor carpi ulnaris (FCU) sesamoid is fractured through falls on the hypothenar eminence.
  - Must exclude ulnar nerve and artery damage (wall of Guyon's canal)
  - Imaging study: The best radiographic image is carpal tunnel view (beware of multiple ossification centers).
  - Treatment: splint wrist in 30° flexion and ulnar deviation (relaxes the FCU)

Other Fractures
- Distal radius is the most commonly fractured bone of the wrist.
- Colles' Fracture
  - Most common fracture in adults over 50
  - Mechanism: FOOSH
  - Presentation: clinically shows dorsal displacement (“dinner fork” deformity)
    - Risk for median nerve palsy presenting as palmar numbness
    - Associated ulnar styloid fracture is common
  - Treatment: usually repaired with closed reduction; displaced intra-articular fracture, severely comminuted fracture, or displacement of the radio-ulnar joint requires ORIF
- Smith's Fracture
  - Volarly displaced or reverse Colles' fracture
  - Mechanism: direct blow or fall onto the back of the hand
  - Presentation: clinically appears as a “garden spade”
    - Watch for median nerve and flexor tendon injuries
  - Treatment: usually amenable to closed reduction
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- Barton's Fracture
  - Rim fracture of distal radius, with dorsal or volar displacement; often associated with dislocation of carpal bones
  - Treatment
    - If minimally displaced and/or <50% of the articular surface is involved, splint and follow-up with orthopedics.
    - More displaced fractures or involvement of >50% of the articular surface requires immediate orthopedic consult.

- Radial Styloid Process Fracture
  - Mechanism: axial load on radial side of hand, which shears the styloid process
  - Pathophysiologically important because the styloid process is the major attachment point for carpal ligaments.
  - Diagnosis: primarily seen on AP view
  - Treatment: may require open fixation

- Ulnar Styloid Process Fracture
  - Mechanism: forced radial deviation, dorsiflexion, or rotary stress
  - Can disrupt the distal radioulnar joint – wrist may click

- Radioulnar Disruption (see also Galeazzi fracture [below])
  - Rare as an isolated injury but missed 50% of time
  - Presentation: painful wrist, clicks, and weakness; ulnar head may be prominent, but this finding is subtle
  - Diagnosis
    - Lateral film makes the diagnosis – be sure to get a good lateral view
    - Consider CT for any doubtful cases
  - Prognosis: associated with a high recurrence rate

15.10 ELBOW AND FOREARM INJURIES

Radiographic Issues
- Fat Pad
  - Anterior fat pad is normal in a third to half of patients.
  - Posterior fat pad is never normal but requires an intact capsule.
    - Can be seen with occult fracture, gout, bursitis, infection

- Normal Lines
  - Radial head always points to capitellum
    - If not, a dislocation is present
  - Anterior humeral line should bisect the middle third of the capitellum

Elbow Dislocations
- Third most common major joint injury; posterior is the most common type
- Complications: 8% to 21% have neurovascular compromise
  - Ulnar nerve is most often affected
- Presentation
  - Usually presents with elbow held at 45° and prominent olecranon
  - Check brachial artery and ulnar, radial, and median nerves before and after manipulation.
  - Absent radial pulse, open dislocation, and other significant injuries are associated with an increased likelihood of arterial injury.
• Treatment: Reduce with wrist traction and downward pressure on forearm; splint at 90°, with neurovascular re-check in 24 hours

Radial Head Subluxation – “Nursemaid’s” Elbow
• Peak incidence between 1 and 4 years of age; rare after 7
• Mechanism: traction on pronated forearm with elbow extended
• Presentation: loss of function (not using a flexed and pronated arm) and no evidence of trauma
• Imaging study: x-ray films are not necessary
• Treatment: supinate and flex the elbow or hyperpronate the forearm to reduce the dislocation, feeling a pop at the radial head upon reduction

Fractures
• Intercondylar
  o More common in adults than children
  o Assume this injury in an adult with any distal humerus fracture
  o Mechanism: direct force to elbow, driving olecranon up
  o Presentation: associated with severe soft-tissue injuries
  o Diagnosis: Look for intercondylar fracture line.
  o Treatment: Patients with severe swelling or dislocation should be admitted to the hospital to be monitored for neurovascular function.
• Supracondylar (see Image #33)
  o Extra-articular, more common in children
  o 95% show posterior displacement of the distal fragment
  o Mechanism: hyperextension
  o Complications
    ▪ Forearm compartment syndrome in 7%
    ▪ High incidence of anterior interosseous nerve injury – test flexion of index finger distal interphalangeal joint and thumb interphalangeal joint.
  o Treatment: Patients with severe swelling or displacement should be admitted to the hospital for monitoring of neurovascular function.
• Humeral Condyle
  o Fracture of both condyles is rare; lateral is more common than medial
  o Treatment
    ▪ Minimal displacements (<2 mm) can be treated with a splint, with the elbow flexed; wrist pronated and dorsiflexed for lateral condyle, flexed for medial condyle
    ▪ Displaced fractures are treated operatively.
• Trochlea and Capitellum
  o Both injuries are very rare.
  o Treatment
    ▪ Non-displaced: splint and refer to orthopedics
    ▪ Displaced: ORIF
• Coronoid
  o Rare, usually associated with dislocation
  o Treatment: immediate orthopedic consult
• Olecranon
  o Common
  o Mechanism: usually a direct blow
  o Types: avulsions, oblique, transverse intra-articular, comminuted
  o Presentation
    - 32% are associated with other fractures, with radial head and neck being most common
    - Watch for radial nerve injury – usually transient palsy
  o Treatment
    - Nondisplaced (<2 mm in both flexion and extension): splint at 45°, orthopedic follow-up
    - Displaced: ORIF

• Radial Head
  o Most common elbow fracture
  o Mechanism: FOOSH
  o Check for distal radioulnar joint tenderness at wrist – may indicate radioulnar joint disruption and interosseous ligament tear (Essex-Lopresti fracture)
  o Diagnosis: radial head capitellum view
  o Treatment: consider aspiration of the joint to improve ROM; sling for comfort; PT for ROM
    - Orthopedic follow-up if severely comminuted or displaced

• Radius
  o Isolated fractures
    - RARE – look for Galeazzi fracture
    - Treatment: splint and follow-up
  o Galeazzi fracture
    - Radius (usually distal) fracture with disrupted radioulnar joint (Figure 15-10)

![GRUM Image](image)

Figure 15-10. GRUM - mnemonic for Galeazzi and Monteggia fractures.
  - Complications: ulnar nerve injury
  - Treatment: ORIF

• Ulna
  o Isolated = nightstick fracture
    - Treatment
      - Displaced: >10° angulation or >50% width displaced – obtain orthopedic consultation
      - Nondisplaced: splint and follow-up
  o Monteggia fracture (see Image #28)
    - Ulnar fracture: usually involves the proximal third and is usually displaced, with radial head dislocation
    - Complications: radial nerve, radial head fracture; often open and requires ORIF

• Both Bones
  o Mechanism: high energy; usually caused by MVC, falls from height, or a direct blow
  o Presentation: often displaced and open
  o Treatment: obtain orthopedic consultation – ORIF
Biceps Rupture
- Anatomy: origin on coracoid process (short head) and glenoid labrum (long head); inserts in bicipital tuberosity of radius; innervated by the median nerve
- 97% of ruptures are proximal, in long head, with distal being rare
- Causes: tendon rupture caused by repetitive micro-trauma
- Presentation: seen in the fourth to sixth decades, usually after a long history of tendonitis pain, with a sudden pop and an obvious defect
- Imaging studies: x-ray films are needed to rule out avulsion fracture
- Treatment: orthopedic consult within 72 hours with immobilization in a sling and analgesia
  - The young and athletes get surgical repair.
  - Everyone else is managed conservatively.

Triceps Rupture
- Least common tendon rupture
- Typically at the common tendon insertion on the olecranon
- Usually occurs in young men secondary to trauma; >80% are associated with fractures
- Treatment: requires surgical repair

15.11 SHOULDER INJURIES

Anatomy
- Axillary nerve
  - Sensory: sensation over lateral deltoid
  - Motor: deltoids/shoulder abduction

Sternoclavicular Dislocations
- Anterior
  - Much more common than a posterior dislocation; less potential for harm
  - Tendency to be unstable and to recur
  - Treatment: frequently need surgery
- Posterior
  - Less common than anterior dislocations; potentially serious, owing to underlying structures
    - Carefully search for underlying structure damage
  - Treatment: orthopedic consult

Clavicle Injuries
- Very common – 5% of all fractures
- Most common fracture of childhood, with half occurring by age 7
- Presentation
  - Middle third, 80%
  - Distal third, 15%
  - Medial third, 5%
- Treatment: simple sling is adequate in most cases
  - For severe comminuted or open fractures, consult orthopedics.

Scapula
- Fractures are rare (<1% of fractures); most common in men between 25 and 40 years of age
- Mechanism: MVC, falls, direct blow to scapula
Complications: >80% associated with injury to lung, thoracic cage, shoulder girdle (rib fracture is most common)
- Treatment: most are treated nonsurgically – sling and analgesics

Rotator Cuff
- Injury after shoulder dislocation is common and frequently missed.
- Presentation: pain/inability to abduct and/or externally rotate
- Treatment: sling, analgesic, and orthopedic referral
- See more in Section 15.5, Overuse Syndrome

Acromioclavicular Joint
- Common injury, usually sustained by young males
- Mechanism: fall/blow to point of shoulder
- Grades I to III are common; rest are uncommon
  - Grade I - tender AC joint without deformity
  - Grade II - tender AC joint with slight upward deformity (intact coracoclavicular ligament)
  - Grade III - large upward deformity (torn coracoclavicular ligament)
- Treatment
  - I and II - sling and analgesics, early ROM
  - III - add orthopedic consult

Shoulder Dislocations
- Anterior, 98%
  - Mechanism: abduction, extension, and external rotation
  - Reduction techniques
    - Multiple, with 70% to 90% success rate for all
    - Facilitated by IV analgesia and/or sedation
    - Standard technique - gentle, steady external rotation of shoulder with elbow flexed; if no reduction after 90 degrees of external rotation (hand in same plane as torso) then abduct the arm while maintaining this external rotation
    - Scapular manipulation - stabilize torso and apply forward traction to injured shoulder with weights or assistant while adducting inferior tip of scapula
    - Traction/countertraction - apply axial traction to arm while applying countertraction with sheet wrapped through axilla and across chest
    - Hennipen technique - patient supine with elbow flexed at 90 degrees; apply gentle, slow external rotation of arm followed by slow elevation of arm above head
    - Hanging weight technique (Stimson technique) - place patient prone and hang affected arm over edge of bed with 5-10 pound weight attached to wrist
  - Complications
    - Recurrence
      - <20 years of age at first dislocation, >90% recurrence
      - >40 years of age at first dislocation, 14% recurrence
    - Axillary nerve is injured in 10% to 25% of cases.
    - Rotator cuff injury is frequent.
    - Less common complications are avascular necrosis and adhesive capsulitis.
    - Vascular injury is rare; when it does occur, it is usually in an elderly patient and involves the axillary artery.
    - Hill-Sachs lesion – posterolateral defect; seen laterally on internal rotation view
- Bankart lesion – glenoid rim fracture/avulsion; not usually seen on x-ray film
- Greater tuberosity fracture is seen in about 15% (see below).
- Posterior, 2%
  - Mechanism: forced internal rotation and adduction
  - Seizures, electrocution, or direct anterior trauma to shoulder
  - Frequently missed
  - Presentation: arm is internally rotated and adducted; won't allow external rotation
  - Plain radiography - may appear normal on AP view or demonstrate loss of elliptical overlap pattern between glenoid and humeral head; axillary or Y-view is essential to make diagnosis
  - Complications: Neurovascular and rotator cuff injuries are less common than in anterior dislocations.

Proximal Humerus Fractures
- Relatively common, 5% of all fractures
- Presentation: typically in elderly people after a fall
- Associated injuries: especially associated with significantly angled surgical neck fractures
  - Axillary nerve – test skin over deltoid, deltoid function
  - Axillary artery
  - Brachial plexus
  - Anatomic neck fractures are associated with avascular necrosis.
  - Greater tuberosity – anterior shoulder dislocation, rotator cuff injury
  - Lesser tuberosity – posterior shoulder dislocation
- Classification (Neer – based on number of significantly displaced parts)
  - One part (80%) = any number of fracture lines, no significant displacement
  - Two parts, 10%
  - More than two, 10%
  - Significant displacement = >1 cm distraction or >45° angulation between pieces
- Treatment: emergent orthopedic consultation for any significant displacement or multipart fracture, otherwise sling/analgesia and early orthopedic referral

Humeral Shaft Fractures
- Bimodal distribution, peaks in third (active young men) and seventh (osteoporotic women) decades
- The middle third of the bone is most commonly involved.
- Complications
  - Radial nerve injury is common, occurring in 10% to 20% of patients with this fracture (wrist drop, decreased sensation in the dorsal first web space)
  - Brachial artery and vein injuries
- Treatment: sugar tong, orthopedic referral

15.12 SPINE INJURIES

Epidemiology
- Mechanism: 90% blunt trauma (MVC 41%), followed by GSW, falls, sports
- 10% associated with another fracture
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Anatomy
* See Table 15-7.

Table 15-7. Dermatomes and Myotomes of Spinal Levels

<table>
<thead>
<tr>
<th>Motor</th>
<th>Sensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1, C2</td>
<td>Neck and scalp</td>
</tr>
<tr>
<td>C3, C4</td>
<td>Diaphragm, deltoid, biceps, biceps reflex</td>
</tr>
<tr>
<td>C5</td>
<td>Shoulder</td>
</tr>
<tr>
<td>C6</td>
<td>Deltoid, biceps, wrist extensors, biceps and brachioradialis reflexes</td>
</tr>
<tr>
<td>C7</td>
<td>Triceps, triceps reflex</td>
</tr>
<tr>
<td>C8</td>
<td>Small finger abduction, triceps, triceps reflex</td>
</tr>
<tr>
<td>T4</td>
<td>Nipples</td>
</tr>
<tr>
<td>T10</td>
<td>Umbilicus</td>
</tr>
<tr>
<td>L1</td>
<td>Inguinal area</td>
</tr>
<tr>
<td>L4</td>
<td>Quadriceps, patellar tendon reflex</td>
</tr>
<tr>
<td>L5</td>
<td>Dorsiflexors of ankle</td>
</tr>
<tr>
<td>S1</td>
<td>Plantar flexors</td>
</tr>
<tr>
<td>S2, S3, S4</td>
<td>Anal sphincter</td>
</tr>
<tr>
<td>S4, S5</td>
<td>Anus</td>
</tr>
</tbody>
</table>

Cervical
* Radiography
  o NEXUS (National Emergency X-ray Utilization Study) criteria mandate that cervical spine imaging be obtained if any of the conditions below are present - “NSAID”
    * N – neurological deficit
    * S – spinal tenderness (midline), NOT ”neck pain”
    * A – altered mental status
    * I – intoxication
    * D – distracting injury
  o “ABCs” method for reading cervical spine x-rays
    * Alignment
    * Bones
    * Cartilage (intervertebral spaces)
    * Spaces
  o Lateral view (finds 70-80% of injuries)
    * First assessment is whether it is an adequate film (can you see C7/T1?)
    * Prevertebral space: up to 6 mm at C2, up to 22 mm at C6 (14 mm in children) is normal; any abnormal space is consistent with hematoma from a fracture and further imaging is required
    * Predental space: <3 mm in adults or <5 mm in children is normal; any abnormal values mandate further imaging to exclude fracture or ligamentous injury
    * Distinguishing pseudosubluxation at C2 from true subluxation
      * Draw a line from the anterior cortical margin of the C1 spinous process to the anterior cortical margin of the C3 spinous process.
      * If this line crosses the anterior cortical margin of the C2 spinous process or is within 2 mm of doing so, then pseudosubluxation is present and the injury is not a true subluxation.
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- AP view
  - Not as helpful
  - Look at alignment of articular masses and spinous processes.

- Odontoid view
  - Dens centered and bisected by central incisors if true AP, lateral masses of C1 and C2 are aligned
  - Classification
    - Type I fracture of the tip of the dens is stable
    - Type II fracture at the base of the dens and Type III fracture into the body of the dens are unstable
    - Jefferson fracture – "ring fracture" of C1

- Flexion/extension views
  - Show soft tissue disruption and ligamentous injury
  - NEXUS study failed to show the utility of these films; rarely useful

- CT scan
  - Increased sensitivity for fracture compared with plain radiographs

- MRI
  - Best for soft tissue injuries and evaluation of the cord and spinal roots

- Stable and Unstable Injuries (Table 15-8)

<table>
<thead>
<tr>
<th>Stable</th>
<th>Unstable (require immediate consultation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior subluxation</td>
<td>Jefferson's fracture</td>
</tr>
<tr>
<td>Clay shoveler's fracture</td>
<td>Bilateral facet dislocation, Burst fractures</td>
</tr>
<tr>
<td>Posterior arch C1 fracture</td>
<td>Odontoid type II and III fractures</td>
</tr>
<tr>
<td>Unilateral facet dislocation</td>
<td>Any fracture/dislocation</td>
</tr>
<tr>
<td>Wedge fracture</td>
<td>Hangman's fracture</td>
</tr>
<tr>
<td></td>
<td>Teardrop fractures</td>
</tr>
</tbody>
</table>

- Anterior subluxation
  - Mechanism: hyperflexion
  - Fanning of posterior elements
  - Diagnosis
    - Plain films may be normal.
    - Flexion extension shows >11° deviation
  - Treatment: stable – hard collar and follow-up

- Unilateral facet dislocation
  - Mechanism: flexion/rotation
  - Imaging studies
    - AP view – spinous processes not aligned
    - Lateral view – <50% anterior displacement
  - Treatment: considered stable, BUT immediate consult

- Bilateral facet dislocation
  - Mechanism: hyperflexion injury
  - >50% anterior displacement
  - High incidence of cord injury
  - Treatment: unstable, immediate consultation
o Simple wedge fracture
  ▪ Mechanism: compression, flexion
  ▪ Anterior elements only – stable
  ▪ Posterior involvement may be unstable – consult

o Clay shoveler's (see Image #61)
  ▪ Avulsion injury to spinous process of a lower cervical vertebra
  ▪ Stable

o Teardrop fracture
  ▪ Mechanism: extreme flexion, breaks off anteroinferior body
  ▪ Complications: cord compromise – usually anterior cord syndrome
  ▪ Treatment: unstable, immediate consultation

o Burst fractures
  ▪ Mechanism: axial compression
  ▪ Diagnosis: seen on lateral view, delineated on CT scan
  ▪ Treatment: unstable, immediate consultation

o Hangman's fracture
  ▪ Mechanism: hyperextension
  ▪ Posterior elements of C2
  ▪ Treatment: unstable, immediate consultation

o Jefferson's fracture
  ▪ Mechanism: axial load injury – burst of C1
  ▪ Diagnosis: odontoid view – displacement of lateral masses >7 mm
  ▪ Treatment: unstable, immediate consultation

o Atlanto-occipital dislocation
  ▪ Mechanism: uncertain
  ▪ Frequently fatal
  ▪ Treatment: unstable, immediate consultation

o Transverse ligament rupture
  ▪ Mechanism: direct blow to skull
  ▪ Treatment: unstable, immediate consultation

o Odontoid fractures
  ▪ 7% to 14% of all cervical fractures
  ▪ Frequently associated with other cervical fractures
    ▪ One third of all C1 fractures have associated odontoid fracture.
  ▪ 18% to 25% are associated with neurologic compromise
  ▪ Classification
    ▪ Type I – tip, spares transverse ligament, stable
    ▪ Type II – junction of dens and body, most common, unstable
    ▪ Type III – body of C2, unstable

Thoracolumbar (see Image #22)
  ▪ Liberal use of CT
  ▪ Unstable fractures: burst, Chance, and translational
    ▪ Stability of wedge fracture depends on integrity of posterior elements
15.13 PELVIC, HIP, AND FEMUR INJURIES

Pelvic Fractures

- General Information
  - 3% of all fractures
  - Frequent cause of death in blunt trauma because of associated injuries and hemorrhage
  - Assessment
    - Look for perineal/pelvic hematomas; Destot’s sign is the presence of a hematoma above the inguinal ligament or over the scrotum.
    - Look for associated internal injuries: rectal, vaginal, GU, and even diaphragmatic injuries.
  - Treatment: external fixation and/or embolization
  - Prognosis: vertical shear injuries have the worst prognosis; 75% of these cause severe hemorrhage

- Fractures of the Pelvic Ring
  - Lateral compression, 50%
    - Mechanism: MVC side impact, auto vs pedestrian
    - Prognosis: 13% mortality
  - AP compression ("open book fracture"), 25% (see Image #64)
    - Mechanism: head-on MVC
    - Treatment: greatest benefit from external fixation (decreases pelvic volume)
    - Prognosis: 25% mortality
  - Vertical shear, 5%
    - Mechanism: fall from height
    - Prognosis: 25% mortality
  - Combination, 20%
    - Malgaigne fracture = double ring fracture
      - Classically, both pubic rami and oblique fracture through sacrum with vertical displacement
      - Cause: vertical shear is the most common mechanism, but all three mechanisms can cause this fracture
      - Complications: 20% GU injury, 38% visceral injury

- Avulsions/Isolated Single-Bone Fractures
  - General information
    - Presentation: local pain, swelling, and tenderness
    - Treatment: conservative treatment – NSAIDs, rest, crutches partial or NWB, follow-up in 1 or 2 weeks
  - Anterior superior iliac spine (ASIS)
    - Adolescents
    - Mechanism: forceful contracture of sartorius
  - Anterior inferior iliac spine (AIIS)
    - Mechanism: forceful contraction of rectus femoris
    - Presentation: pain in groin, difficulty walking
  - Ischial tuberosity
    - Adolescents
    - Mechanism: forceful contracture of hamstrings (e.g., kicking)
    - Presentation
      - Pain on sitting or with hips flexed with extended knees
      - Rectal exam – tuberosity is tender
o Isolated ramus (pubis or ischium) fracture
  ■ Mechanism: elderly patient, fall on buttocks
  ■ Treatment: analgesics, crutches

o Ischium body fracture
  ■ Rare
  ■ Mechanism: violent fall on buttocks
  ■ Presentation: local pain, pain with hamstring movement
  ■ Treatment: analgesics, rest, donut cushion, follow-up

o Iliac wing (Duverney) fracture
  ■ Mechanism: direct trauma, usually compressive
  ■ Presentation: extreme pain on walking, Trendelenburg’s sign
  ■ Complications: rare associated injuries, but frequent severe abdominal pain
  ■ Treatment: conservative treatment, usually outpatient

o Sacrum
  ■ 4% to 5% of all fractures
  ■ Transverse fractures are associated with major pelvic trauma.
  ■ Diagnosis: sometimes difficult to diagnose with plain radiographs, CT scan if question of fracture
  ■ Treatment: neurosurgical/orthopedic consultation in all sacral fractures; conservative treatment is frequently employed if no neurological involvement

o Coccyx
  ■ Women > men
  ■ Presentation: fall onto buttocks
  ■ Diagnosis: no radiographs needed - rectal exam demonstrates coccygeal tenderness and makes the diagnosis
  ■ Treatment: analgesics, donut cushion, follow-up as needed

* Acetabular Fractures

o General information
  ■ 20% of all pelvic fractures
  ■ Mechanism: usually associated with MVC and other fractures
  ■ Treatment: prompt orthopedic evaluation.
  ■ Complications: sciatic nerve injury is common

o Four types, all associated with dislocation:
  ■ Posterior
    □ Mechanism: direct trauma to flexed knee and hip
    □ Diagnosis: AP view – posterior fracture with posterior dislocation
    □ Complications: sciatic nerve injury (40%), femur fracture
  ■ Ischial column
    □ Mechanism: axial force to knee with thigh adducted and flexed
    □ Diagnosis: x-ray film – medially displaced fragment with central dislocation
    □ Complications: sciatic nerve injury is common
  ■ Transverse
    □ Mechanism: lateral to medial force
    □ Diagnosis: x-ray film – fracture with central displacement
• Iliopubic column
  - Mechanism: lateral to medial force with femur externally rotated
  - Diagnosis: x-ray film – marked external rotation, anterior lip fracture

**Hip Fractures**

- **General Information**
  - Incidence: 80 per 100,000; doubles for each decade after 50
  - Two to three times more common in women and white > black
  - Prognosis: mortality 15% to 35% within first year; of the survivors, 20% to 50% will not walk again
  - Classification: femoral head and neck, trochanteric, subtrochanteric, and intertrochanteric
  - Medical emergency – can bleed to death; all need prompt consultation

- **Femoral Head**
  - Infrequent, associated with dislocations

- **Femoral Neck**
  - Older people, rare in young
  - Mechanism: 90% from falls
  - Diagnosis: high suspicion with negative plain film; consider CT or MRI

- **Trochanteric**
  - Greater
    - Mechanism
      - Usually caused by avulsion of gluteus medius; typically 7 to 17 years old, epiphyseal separation
      - Direct trauma in adults
    - Presentation: local pain and tenderness, worse with abduction and extension, limp
    - Treatment
      - Conservative treatment if distraction <1 cm, orthopedic follow-up
      - Prompt referral for distraction >1 cm
  - Lesser
    - Mechanism: forceful contraction of iliopsoas; dancing or gymnastics
    - Children and young athletic adults
    - Presentation: pain with flexion and internal rotation
    - Treatment
      - Bed rest and gradual return to activity
      - ORIF for displacement >2 cm

- **Intertrochanteric**
  - Women, elderly
  - Mechanism: falls, occasionally MVC
  - Presentation: pain and swelling; leg is shortened and externally rotated
  - Treatment: admit for ORIF
  - Prognosis: mortality 10% to 30%

- **Subtrochanteric**
  - Bimodal epidemiology: falling in 40 to 60 year olds; young people with major trauma
  - Complications: large amount of blood loss
  - Treatment: immediate orthopedic consultation
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Hip Dislocations

- Anterior, 10%
  - Mechanism: majority from MVC
  - Presentation: hip abducted; leg is externally rotated and flexed
  - Complications: neurovascular injury is rare
  - Treatment: sedation and strong in-line traction with flexion and internal rotation

- Posterior, 90%
  - Mechanism: anterior force to flexed knee
  - Presentation: leg is shortened, internally rotated, slightly flexed, and adducted.
  - Imaging studies: Judet view for acetabular fracture
  - Complications: sciatic nerve injury in about 10%
  - Treatment: heavy sedation, traction at 90°, gentle internal/external rotation

- Central is rare

Femoral Fractures

- Usually occur in men during the most active part of life
- Complications
  - May lose a liter of blood or more
  - Nerve damage and nonunion are rare
- Pathologic fractures are usually associated with breast, lung, or prostate cancer.

15.14 KNEE INJURIES

Fractures

- Knee Rules
  - Only 6% of patients with knee trauma have a fracture.
  - Ottawa vs Pittsburgh knee rules
    - Pittsburgh rule states that a radiograph is necessary only if the patient fell or sustained blunt trauma to the knee and either of two conditions is present:
      - Age younger than 12 or older than 50
      - Inability to walk four full weight-bearing steps in the ED
    - Pittsburgh rule is just as sensitive, more specific, and simpler than Ottawa knee rule
  - Patella
    - Mechanism: direct blow or violent contracture of quadriceps
    - Transverse fracture most common (50%-80%)
    - Treatment
      - If able to actively extend without displacement, knee immobilizer and follow-up
      - If not, orthopedic consultation
    - Always consider bipartite patella instead of fracture; characteristics of bipartite patella are listed below:
      - Bilateral 80% of the time (comparison views are helpful)
      - Smaller part is usually superiolateral
      - Cortices of bony fragments are intact without visible fracture
  - Femoral Condyles
    - 4% of femur fractures
    - Classification: supra-, intra- and condylar fractures
Complications
- Neurovascular problems are uncommon, but check pulses and first web space sensation
- Look for other injuries

• Tibial Spine
  - Mechanism: direct force to upper tibia – anterior or posterior
  - Anterior spine is 10X more commonly injured than posterior spine
  - Presentation: pain, swelling
  - Treatment: knee immobilizer, NWB, timely orthopedic follow-up (usually requires surgery)

• Tibial Tuberosity
  - Mechanism: force to flexed knee with quads contracted
  - Diagnosis: lateral view
  - Treatment: knee immobilizer, NWB, orthopedic follow-up

• Tibial Plateau
  - More common in elderly
  - Presentation: severe pain and unable to bear weight
  - Diagnosis: CT scan may help make the diagnosis as these fractures can be subtle on plain radiographs
  - Complications: post-traumatic arthritis (most common), ligamentous injury in one third, neurovascular injury with severe/unstable injuries, deep peroneal nerve injury, and DVT
  - Treatment
    - Single plateau non-displaced – splint, NWB, orthopedic follow-up
    - Others – orthopedic consultation

Ligamentous Injury
- Mechanism: lateral force is the most common cause
- The anterior cruciate ligament (ACL) is most commonly injured (and is the most common serious ligament injury in adults).
- The medial collateral ligament is the most commonly injured ligament in children.
- Presentation of ACL injury
  - Hemarthrosis is common; 75% of all knee hemarthroses are caused by ACL injury
  - MAY have no swelling, even with complete disruption
  - Loud pop
  - Lachman test is the most sensitive for ACL injury – 20° flexion, femur stabilized, tibia pulled
- Imaging studies: x-ray film - can see Segond fracture (lateral avulsion of the tibial plateau) or avulsion of the tibial spine, but usually there is no osseous injury
- Treatment
  - Minor/stable – immobilizer and follow-up
  - Major/unstable – immediate orthopedic consultation

Meniscal Injury
- Isolated or with ligament – ACL/meniscus common
- Medial meniscus injuries occur twice as often as lateral meniscus injuries; 80% of injuries occur at the peripheral, posterior aspect of the meniscus
- Presentation
  - Suspect with popping, clicking, LOCKING, swelling after exercise
  - Diagnostic maneuvers are limited – 50% positive
• Diagnosis: MRI is the preferred diagnostic study
• Treatment: NSAIDs and follow-up

Knee Dislocation
• Associated with major ligamentous disruptions
• Posterior is most common
• Presentation: often reduces spontaneously
• Complications
  o 50% associated with popliteal artery injury
  o Peroneal nerve injury is common
• Treatment: immediate orthopedic referral; vascular consultation for diminished pulse

Patella Dislocation
• Mechanism: twisting on extended knee, dislocating the patella laterally
• Women > men
• Treatment: gentle reduction (to reduce the risk of osteochondral fracture during reduction) with or without sedation
  o Post-reduction x-ray film – include sunrise view to rule out osteochondral fracture

Quadriceps Tendon and Patellar Ligament Rupture
• Presentation
  o Patellar tendon rupture usually occurs in people younger than 40 with a history of chronic knee pain and repetitive injury
  o Quadriceps tendon rupture usually occurs in people older than 40 without prior symptoms

15.15 LEG INJURIES

Compartments (Figure 15-11) (Table 15-9) – see compartment syndrome below

Figure 15-11. Compartments of the leg (A=artery, N=nerve, T=tibia, F=fibula).
Table 15-9. Compartments of the Leg and Their Structures

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Anterior (anterior tibial artery and deep peroneal nerve)</th>
<th>Lateral (superficial peroneal nerve)</th>
<th>Deep Posterior (posterior tibial artery and nerve)</th>
<th>Superficial Posterior (sural cutaneous nerve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor</td>
<td>Dorsiflexion of foot and toes</td>
<td>Foot eversion</td>
<td>Toe flexors</td>
<td>Ankle flexors</td>
</tr>
<tr>
<td>Sensation</td>
<td>First web space</td>
<td>Dorsolateral foot</td>
<td>Sole</td>
<td>Lateral heel</td>
</tr>
<tr>
<td>Vascular</td>
<td>Dorsalis pedis pulse</td>
<td></td>
<td>Posterior tibialis pulse</td>
<td></td>
</tr>
</tbody>
</table>

Fibula Fractures (Isolated)
- Mechanism: direct blow
- Complications: suspect peroneal nerve damage - check for dorsolateral foot sensation
- Treatment: minimal treatment needed

Tibia Fractures
- Mechanism: higher energy required for fracture; worse prognosis
- Fractures of the distal third of the tibia have the highest incidence of complications

Achilles Tendon Rupture
- Most commonly occurs 2-6 cm above tendinous insertion on calcaneus where the blood supply is the poorest
- Predisposing Factors
  - Participating in sports without prior conditioning
  - Systemic lupus erythematosus
  - Rheumatoid arthritis
  - Previous local steroid injections
- Diagnosis: Thompson test fails to demonstrate plantar flexion when the calf is squeezed
- Treatment: splint with foot in plantar flexion (equinus), outpatient orthopedic consultation

Gastrocnemius Rupture
- Most commonly occurs at the medial head
- Predisposing Factors: sports with advanced age, inadequate stretching, previous injury
- Differential Diagnosis: ruptured Baker’s cysts, DVT

Osgood-Schlatter’s Disease
- Occurs in athletic teenagers, same incidence in males and females
- Partial separation of tibial tuberosity
- One fourth are bilateral

15.16 ANKLE INJURIES

Epidemiology
- Common ED problem
- Majority of patients are < 40 years of age; equal incidence in males and females

Ottawa Ankle Rules
- Decision rule used to decide if ankle and/or foot radiographs are necessary
- Order ankle radiographs if there is pain in the malleolar region and one of the following conditions is met:
  - The patient cannot bear weight for four steps both at the time of injury and at the time of evaluation in the ED
  - Tenderness over the posterior edge of the distal 6 cm or the tip of either malleolus
• Add foot radiographs to the ankle series if there is pain in the midfoot region and one of the following conditions is met:
  o The patient cannot bear weight for four steps both at the time of injury and at the time of evaluation in the ED
  o Tenderness is present at the base of the fifth metatarsal
  o Tenderness is present over the navicular bone

Ligamentous Injuries
• Lateral Complex – anterior talofibular, posterior talofibular, calcaneofibular ligaments
  o 90% of ankle injuries; most injuries are minor
  o Of these, 90% involve the anterior talofibular ligament
  o Assess all injuries for ligamentous stability vs instability
  o Treatment
    ■ Stable injuries - rest, ice, compression, and elevation (RICE); crutches and ACE wrap, ankle brace, or other support (no casting) as needed
    ■ Unstable - as above for stable injuries but consider splinting and outpatient orthopedic referral

• Medial Complex – deltoid ligament
  o Isolated injury here is rare
  o Complications
    ■ Suspect Maisonneuve fracture (proximal fibular fracture, see Image #71) - examine fibula for tenderness
    ■ Suspect significant syndesmosis injury with proximal swelling and pain

• Tibiofibular Syndesmosis (High Ankle Sprain)
  o Prolonged recovery time
  o Diagnosis: Squeeze test (compression of the distal tibia and fibula together causes pain)
  o Treatment: RICE therapy, analgesics and immobilization as needed, outpatient referral to orthopedics

• Peroneal Tendon Dislocation
  o Usually a skiing injury
  o Misdiagnosed as lateral ankle sprain
  o Presentation: pain and ecchymosis at the lateral ankle, no pain anteriorly, patient may feel a “pop” in the area of injury
  o Treatment: surgical repair

Fractures and Dislocations
• Classification
  o Medial, lateral, and posterior malleoli
  o Single, bimalleolar, or trimalleolar
  o Stable or unstable
  o Associated ligamentous injury
  o Above and below tibial plafond
• Maisonneuve Fracture (see Image #71)
  o Proximal fibula fracture associated with a medial malleolus fracture and/or deltoid/syndesmotic ligamentous injury
  o Prolonged recovery
  o Treatment: immobilization, non-weight bearing, and outpatient orthopedic referral
• Osteochondral Fracture
  o Commonly missed in ED
  o Diagnosis: mortise view or CT scan
• Os Trigonum
  o Accessory ossicle behind talus, seen in 2% to 14% of population
  o Can be confused with a fracture
  o Presentation: occasionally associated with chronic pain in this area

15.17 FOOT INJURIES

Anatomic Issues

• Three compartments, divided by Chopart’s and Lisfranc’s joints
• Blood supply tenuous
• First metatarsal bears twice the weight of any other
• Base of second metatarsal is keystone to Lisfranc’s joint

Hindfoot

• Calcaneus Fracture
  o Mechanism: axial load to heel
  o Diagnosis: lateral and calcaneal views (see Image #43)
    • Boehler’s angle: think of it as the “roof angle” of the calcaneus; less than 20° indicates possible fracture
  o Complications
    • Look for other fractures
    • Rule of 10s
      □ 10% are bilateral
      □ 10% have concomitant lumbar compression fractures
      □ 10% have concomitant tibial plateau fractures
  o Treatment: bulky compression dressing, orthopedic consultation
• Talus Fracture
  o Rare
  o Mechanism: excessive dorsiflexion from an MVC, snowboarding, or basketball injuries
  o Treatment
    • Minor avulsions are treated with splinting and orthopedic follow-up.
    • Major fractures are associated with avascular necrosis and therefore need immediate orthopedic referral.

Midfoot

• General Information
  o Isolated fractures are rare and hard to see.
  o Navicular is the most commonly fractured bone.
  o Cuboid or cuneiform fractures should raise suspicion of Lisfranc’s fracture
  o Any midfoot radiograph on the boards is showing either a Lisfranc injury or a fifth metatarsal fracture.
• Lisfranc’s fracture (see Image #60)
  o NOT uncommon, missed in up to 20% of cases!
  o Cause: usually MVC
  o Presentation: majority associated with fractures – metatarsal or tarsal
    • Fracture at the base of the second metatarsal is pathognomonic.
  o Treatment: emergent orthopedic consultation – ORIF is common
Forefoot

- General Information
  - First, fourth, and fifth metatarsals are mobile; second and third are fixed.
  - Stress fractures (AKA March fractures) occur most commonly in the second and third metatarsals.
  - Phalangeal fractures are common and are usually handled conservatively.
  - Nondisplaced fractures of the first through fourth metatarsals are splinted, with outpatient orthopedic follow-up.
  - Fifth metatarsal fractures and all displaced fractures should undergo closed reduction if possible (if reduction is unsatisfactory, operative repair may be required), splinting, NWB, and referral to orthopedics.

- Fifth Metatarsal Base (Figure 15-12): Jones vs. Avulsion

![](image)

Figure 15-12. Fractures of the fifth metatarsal base.

- Don’t confuse!
- Most common fractures of foot
- Avulsion fractures (dancer’s fracture) - >90% of fractures in this area
  - Frequently extra-articular
  - Treatment: cast boot or splint for 2 to 3 weeks
    - Tend to do very well with conservative treatment
- Jones fracture, 4%
  - Acute transverse fracture of proximal metaphyseal-diaphyseal area
    - Specifically 15 to 31 mm distal to joint
  - Prognosis: high incidence of nonunion, chronic pain
  - More proximal injuries do worse
  - Treatment
    - If non-displaced, treat acutely with NWB cast for 6 to 8 weeks; two thirds heal
    - Displaced fractures require early orthopedic consultation and ORIF.
- Stress fractures, 3%
  - Diagnosis
    - Similarly located to Jones (slightly more distal), but with pre-existing history of pain
    - Should see some sclerosis at border or evidence of new bone formation
- Prognosis: frequently does not heal
- Treatment: Refer to orthopedics.
  - Normal findings that simulate fractures
    - Normal apophysis: in 9 to 14 year olds, parallels shaft, cortical border without irregularity, does not enter joint
    - Os peroneum: sesamoid bone in peroneus longus, near insertion on fifth metatarsal, 15% of population, cortical border without irregularity, more rounded

15.18 COMPARTMENT SYNDROME

Pathophysiology
- Normal capillary pressures are <10 mm Hg
- Circulatory compromise begins at compartmental pressures of 20 mm Hg
- First nerves then muscles are compromised between 30 and 40 mm Hg
- Can still have arteriolar and arterial flow

Compartments
- Virtually any muscle surrounded by heavy fascia
- Lower leg, forearm, and foot are the most commonly involved, with the primary site being the anterior compart­ment of the lower leg.

Presentation
- Pain out of proportion and away from the actual site of the injury.
- Diagnosis and management: When in doubt, measure compartmental pressure.
  - <15 mm Hg: normal
  - 15 to 20 mm Hg: potentially problematic and patient dependent
  - 20 to 30 mm Hg: consult orthopedics, potential for observation and repeat measurements
  - >30 mm Hg: fasciotomy
16.1 ADDICTIVE BEHAVIOR

Alcohol and Drug Dependence
• Direct or indirect cause of a large number of emergency department visits
• Associated with the following:
  o Chronic medical conditions, e.g., pancreatitis, HIV infection, cirrhosis, coronary artery disease
  o Suicidal and homicidal behavior; violent crime
  o Blunt and penetrating trauma
  o Child abuse
• Definition of Dependence
  o Tolerance to a drug
  o Withdrawal from a substance produces symptoms; use of a substance to avoid withdrawal
  o Inability to control substance use
  o Increasing amount and/or duration of substance use beyond what is intended
  o Spending large amounts of time obtaining or recovering from substance’s effects
  o Impaired occupational, social, or recreational activities as a result of substance use

Eating Disorders
• Anorexia Nervosa
  o Defined by the following:
    ▪ Refusal to maintain normal body weight
    ▪ Distortion of body image
    ▪ Fear of gaining weight
    ▪ Amenorrhea for three consecutive menstrual cycles
  o Body weight <85% predicted for height and weight
  o Subtypes
    ▪ Restrictive – food intake is severely limited
    ▪ Binging-purging – excessive food intake followed by induced emesis, laxative, or diuretic abuse or excessive exercise
Complications consistent with nutritional deficiencies and purging:
- Electrolyte imbalances
- Pancytopenia
- Endocrine derangements
- Bradycardia
- Cardiac dysrhythmias
- Prolonged QT interval
- Esophagitis and gastritis
- Hypothermia

Bulimia Nervosa
- Defined by excessive food intake followed by purging via emesis, laxatives, or diuretics or by excessive exercise
- Patients feel a lack of control over eating
- Body weight is normal or above normal
- Less pronounced complications than with anorexia nervosa plus the following:
  - Parotid gland enlargement
  - Aspiration
  - Pneumomediastinum
  - Periodontal disease

Substance Abuse
- Alcohol
  - Intoxication
    - CNS depression, disinhibition, slurred speech, ataxia, nystagmus, aggressiveness, risk-taking behavior, hypothermia, coma, death by aspiration, and respiratory depression
    - Metabolic clearance estimated at \(-20 \text{ mg/dl/hr}\)
    - Treatment
      - Manage airway, give thiamine to prevent Wernicke's encephalopathy, give dextrose if hypoglycemic (preferably after thiamine administration)
      - Glucagon may not be effective for hypoglycemia because it works on endogenous glycogen stores; alcoholics have depleted glycogen stores as a result of malnutrition
  - Withdrawal
    - Spectrum of symptoms after cessation of, or decrease in, usual alcohol consumption
    - Minor withdrawal
      - 6 to 24 hours after ethanol level falls
      - Lasts 2 to 7 days
      - Nausea, anorexia, tremor, tachycardia, hypertension, insomnia, anxiety, sleep disturbance, hyperreflexia
    - Major withdrawal
      - 24 hours to 5 days after ethanol level falls
      - More pronounced symptoms of minor withdrawal, plus hyperthermia, seizure, auditory and visual hallucinations with otherwise clear sensorium
    - Delirium tremens (DTs)
      - 48 hours to 1 week after ethanol levels fall
      - More pronounced symptoms than minor and major withdrawal, plus profound confusion, agitation, frightening visual hallucinations, mydriasis
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- Treatment
  - Minor withdrawal – give fluids and vitamins, correct electrolyte abnormalities (hypomagnesemia and hypokalemia are common)
    - Ideal fluids contain glucose (to reverse ketosis) and sodium (for volume), e.g., D5NS or D5LR
  - Alcohol withdrawal seizures – unlike other seizure etiologies, benzodiazepines are effective for prophylaxis against recurrent seizures
  - DTs – use benzodiazepines (lorazepam, diazepam), supportive care, and airway management

- Hallucinogens
  - Lysergic acid diethylamide (LSD), psilocybin, N,N-dimethyltryptamine (DMT), mescaline, methamphetamine, 3,4-methylenedioxymethamphetamine (MDMA, Ecstasy), ketamine, peyote
  - Intoxication
    - Acute psychotic behavior, dilated pupils, tachycardia, tachypnea, non-focal neurologic exam
    - MDMA – hyperthermia, hyponatremia, seizure, stupor, death
    - Treatment – place in quiet, non-threatening environment; use chemical (benzodiazepine) or, less often, physical restraint as needed; supportive care for MDMA-related effects
  - Withdrawal – no withdrawal state requiring treatment

- Opioids
  - Intoxication
    - Miosis, respiratory depression, CNS depression
    - Non-cardiogenic pulmonary edema, nausea, and vomiting
    - Treatment – give naloxone for opiate reversal; protect airway
  - Withdrawal
    - Dilated pupils, tachypnea, CNS excitation
    - Cramping abdominal pain, nausea, vomiting, diarrhea, piloerection, yawning, lacrimation, rhinorrhea, myalgias
  - Treatment – provide symptomatic and supportive care when necessary

- Phencyclidine (PCP)
  - Can be used alone (inhaled, ingested, or injected) or in combination with marijuana or cocaine
  - Intoxication
    - Altered and fluctuating mental status, hypoglycemia, violent behavior, psychosis, staring spells, rotatory nystagmus, hypertension, tachycardia, hyperthermia, rhabdomyolysis, dystonia, seizures, coma
    - Treatment – give supportive care; use chemical and physical restraints as needed (benzodiazepines for seizures, haloperidol for agitation)
  - Withdrawal – no withdrawal state requiring treatment

- Sedatives/Hypnotics/Anxiolytics
  - Barbiturates
    - Intoxication
      - Ataxia, nystagmus, slurred speech, altered level of consciousness, respiratory depression, reflexes ranging from decreased to flaccid, hypotension, non-cardiogenic pulmonary edema, hypothermia, delayed gastric emptying, vesicles and bullae at contact surfaces (“barbiturate blisters”)
    - Treatment – manage airway; supportive care; use charcoal for recent ingestions if patient is able to cooperate and the risk for aspiration is minimal
Withdrawal
- Delirium, hallucinations, tremor, seizure
- Treatment – benzodiazepines and supportive care

Benzodiazepines
- Intoxication
  - Slurred speech, drowsiness, ataxia, nystagmus, altered level of consciousness, respiratory depression, hypotension, hypotonia
  - Treatment – provide supportive care, manage airway, consider flumazenil use only if isolated overdose in a nonhabituated user
    - Flumazenil use in a chronic benzodiazepine user may be associated with intractable seizures
- Withdrawal
  - Insomnia, tachycardia, diaphoresis, tremor, anxiety, delirium, seizures
  - Treatment – can use long-acting benzodiazepines with taper over the course of weeks

Flunitrazepam (Rohypnol) – intoxication
- Disinhibition, anterograde amnesia, CNS depression
- Treatment – give supportive care as needed

γ-Hydroxybutyrate (GHB) and its prodrug, γ-butyrolactone (GBL)
- Intoxication
  - Dose dependent
  - Emesis, disinhibition, amnesia, hypotonia, CNS depression with alternating agitation when attempting intubation, respiratory depression, miosis
  - All symptoms resolve rapidly (GHB has a short half-life).
  - Treatment – provide supportive care; manage airway
- Withdrawal
  - Seen after constant ingestion for extended periods of time
  - Anxiety, tremor, insomnia, delirium, psychosis, auditory and visual hallucinations, autonomic instability
  - Treatment – use high-dose benzodiazepines; use barbiturates if benzodiazepines are ineffective

Chloral hydrate – intoxication
- Synergistic with ethanol (AKA Mickey Finn)
- Drowsiness, ataxia, incoordination, “pear-like” odor on patient’s breath, miosis, flaccidity
- Hypoventilation, hypothermia, hypotension, hepatic dysfunction, emesis, gastrointestinal irritation/bleeding, cardiac dysrhythmias
- Treatment – provide cardiopulmonary support, β-blockers for dysrhythmia/ectopy (avoid epinephrine, lidocaine, norepinephrine, flumazenil, naloxone)

Buspirone
- Intoxication – minimal CNS depression
- Withdrawal – no known withdrawal state

Zolpidem and zaleplon – intoxication
- Drowsiness, vomiting, rarely respiratory compromise and coma
- Treatment – supportive care
Over-the-counter sleep aids
- Contain diphenhydramine and doxylamine
- Intoxication
  - Somnolence, psychotic behavior, agitation, seizures, rhabdomyolysis, anticholinergic effects, prolonged QRS interval
  - Treatment – supportive care only if mild to moderate toxicity; give sodium bicarbonate for QRS prolongation, physostigmine for severe anticholinergic toxicity

Cocaine and Other Sympathomimetics
- Cocaine
  - Intoxication
    - Tachycardia, agitation, anorexia, mydriasis, diaphoresis, hyperthermia, severe hypertension, delirium, psychosis, seizures, choreiform movements, rhabdomyolysis, cardiac dysrhythmias, myocardial infarction, intracerebral hemorrhage
    - Fever/hyperthermia is generally considered the greatest predictor of mortality among the various vital sign abnormalities.
    - Treatment – use benzodiazepines (diazepam) for agitation and most other effects of cocaine intoxication; rapidly cool if hyperthermic; administer intravenous nitroprusside, nitroglycerin, and phentolamine for hypertensive emergencies and sodium bicarbonate (or lidocaine for those who fail bicarbonate therapy) for dysrhythmias
    - β-Blockade is contraindicated in cocaine-intoxicated patients due to the risk of unopposed α-adrenergic vasoconstriction.
  - Cocaine washout syndrome ("cocaine crash")
    - Secondary to catecholamine depletion after binge cocaine use
    - Self-limited psychomotor retardation, exhaustion, suicidal ideation
    - Treatment – provide supportive care
  - Withdrawal
    - Apathy, depression, lethargy, anxiety, sleep disorders
    - Treatment – supportive care if necessary

Other sympathomimetics
- Amphetamines, methamphetamine, ephedrine, khat, methcathinone
- Symptoms and treatments are similar to those of cocaine.

16.2 AFFECTIVE AND THOUGHT DISORDERS

Bipolar Disorder – alternating periods of extremes of mood
- Bipolar Type I – manic and depressive periods
  - Mania – minimum of 5 days or hospitalization required for the following:
    - Expansive or irritable mood
    - Grandiosity
    - Reckless behavior (spending sprees, promiscuity)
    - Decreased need for sleep
    - Pressured speech
    - Distractibility
  - Depression – see below
Bipolar Type II – hypomanic and depressive periods
  o Hypomania
    - Minimum 4 days' duration without need for hospitalization
    - Same symptoms as for mania, but with lesser severity and impairment in function, stress on relationships, etc.
  o Depression – see below
  o Patients have no history of manic episodes
• Significant morbidity and mortality – roughly 10% of bipolar patients eventually commit suicide
• Treatment
  o May require antipsychotics, benzodiazepines for agitation
  o Mood stabilizers – use lithium, valproic acid, carbamazepine for long-term control, with adjunctive antidepressants

Major Depression
• Depressed mood or loss of interest in previously pleasurable activities
• Increased or decreased psychomotor activity
• Neurovegetative symptoms
  o Sleep: insomnia or hypersomnia
  o Appetite: increased or decreased appetite
  o Libido: decreased sexual interest
• Cognitive impairment
  o Difficulty concentrating
  o Feelings of guilt, worthlessness, hopelessness, helplessness
• Symptoms last for at least 2 weeks
• Suicide
  o More women attempt suicide; more men complete it
  o Majority of attempts from ingestion; majority of completions from firearms
  o Untreated depressive illness carries a 15% lifetime suicide risk
  o Risk factors
    - White men >65 years; women >60 years
    - Unmarried, unemployed, living alone
    - Recent personal loss or crisis
    - Psychiatric disorder and/or substance abuse history
    - Prior suicide attempt
    - Chronic illness or pain
    - Positive family history of suicide
    - Firearm in home
    - Hopelessness and anhedonia
• Treatment – admit if psychotic or suicidal; otherwise, refer for outpatient follow-up

Adjustment Disorders – disabling reaction to a stressful situation or event
• Reaction to the event is greater than expected from the stressor, with resultant significant impairment in social and occupational functioning.
• Occurs within 3 months of and lasts less than 6 months after end of the stressor exposure.
• Can manifest symptoms of anxiety, depression, or conduct problems (e.g., substance abuse)
Grief Reaction/Bereavement

• Normal grief
  o Symptoms include somatic complaints, insomnia, sadness
  o Classic stages of grief (not necessarily in this order)
    ▪ Denial/disbelief
    ▪ Anger/awareness
    ▪ Bargaining
    ▪ Depression
    ▪ Acceptance
  o Duration of grief heavily influenced by culture

• Complicated grief
  o Difficult to distinguish from major depressive disorder
  o Grief/bereavement differs from major depression in the following ways:
    ▪ Suicidal tendency – desire to have died with the loved one
    ▪ Worthlessness and feelings of guilt about things done or not done for the deceased
    ▪ Hearing the voice or seeing images of the deceased
  o Includes severe psychomotor retardation

Acute Psychosis

• Brief Psychotic Disorder
  o Psychotic symptoms (see schizophrenia section for symptom elaboration)
    ▪ Hallucinations
    ▪ Delusions
    ▪ Disorganized speech or behavior
  o Can be in response to acute stressor or postpartum reaction
  o Symptoms last from 1 day to 1 month

• Schizophreniform Disorder
  o Same symptoms as for brief psychotic disorder
  o Symptoms last longer than 1 month and less than 6 months

• Other Causes of Acute Psychosis
  o Drugs of abuse – ethanol, amphetamines, marijuana, cocaine, hallucinogens, opioids, PCP, sedative-hypnotics
    (see above for specific acute intoxication and withdrawal states)
  o Prescribed medications – anxiolytics, antibiotics (isoniazid, rifampin), anticonvulsants (phenytoin, phenobarbital), antidepressants (tricyclics), cardiovascular drugs (digitalis, propranolol, captopril), other drugs (antihistamines, corticosteroids)
  o Medical disorders
    ▪ Metabolic – hypercalcemia, hypoxia, hypercarbia, hypoglycemia, hyponatremia
    ▪ Inflammatory – systemic lupus erythematosus (SLE), sarcoidosis, temporal arteritis
    ▪ Organ failure – hepatic encephalopathy, uremia
    ▪ Neurologic – encephalitis, dementia, multiple sclerosis, neoplasm, normal-pressure hydrocephalus, Parkinson’s disease, Pick’s disease, Wilson’s disease, cerebrovascular disease
    ▪ Endocrine – Cushing’s disease, Addison’s disease, hypopituitarism, parathyroid disease, Sydenham’s chorea, postpartum psychosis
    ▪ Nutritional deficiencies – niacin, thiamine, folate, vitamin B12
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Schizophrenia – 6 months of positive and negative symptoms

• Positive Symptoms
  o Delusions
    ▪ Fixed, false beliefs not amenable to argument
    ▪ Often persecutory, religious, somatic
  o Hallucinations
    ▪ Perceived sensory experience that does not exist
    ▪ Most often auditory; may be visual, gustatory, olfactory, tactile
  o Disorganized speech and behavior
    ▪ Loosening of associations, tangential and rambling speech
    ▪ Disheveled appearance

• Negative Symptoms
  o Poverty of speech
  o Flat affect and social/emotional withdrawal
  o Avolition (inability to perform goal-directed activities)

• Must assess for side effects from neuroleptic use (see toxicology chapter for treatments), as well as suicidality and homicidality
  o Dystonic reactions – contraction of truncal, limb, neck (torticollis), jaw, eye muscles (oculogyric crisis)
  o Tardive dyskinesia – repetitive, purposeless, involuntary movements, tongue fasciculations
  o Akathisia – motor restlessness
  o Neuroleptic malignant syndrome – fever, rigidity, decreased level of consciousness, autonomic instability, rhabdomyolysis

• Treatment – neuroleptics, antipsychotics, benzodiazepines for agitation

16.3 NEUROTIC (ANXIETY) DISORDERS – FEAR OR TENSION OUT OF PROPORTION TO EXTERNAL STIMULI CAUSING FUNCTIONAL IMPAIRMENT

Generalized Anxiety Disorder

• Intense, unrealistic worry and anxiety that are difficult to control
• Associated fatigue, irritability, insomnia, difficulty concentrating, restlessness
• Symptoms for >6 months

Panic Disorder

• Apprehension, intense fear, sense of dread or impending doom
  o Episodes are discrete, unprovoked, sudden in onset
  o Symptoms peak within minutes, resolve within an hour
• Autonomic arousal – palpitations, tremor, tachycardia, diaphoresis, chest pain, dyspnea, dizziness
• With or without agoraphobia – anxiety about or avoidance of open or public places from which escape/assistance not available
• Treatment
  o Use selective serotonin reuptake inhibitors (SSRIs) as first line, then tricyclic antidepressants (TCAs) for symptom control/prevention
  o Benzodiazepines for acute symptoms and for short-term use (1–2 weeks) as necessary
Obsessive-Compulsive Disorder
- Obsessions - intrusive thoughts, images, or impulses
- Compulsions - repetitive rituals or behaviors that decrease anxiety associated with the obsessions (e.g., handwashing, counting)

Phobias - irrational fear of an object or situation, which the patient recognizes as inappropriate
- Specific phobia - examples include airplanes, snakes, blood
- Social phobia - fear of humiliating oneself (e.g., public speaking)

Post-traumatic Stress Disorder
- Reaction to severe, life-threatening traumatic event (war, rape, witnessed homicide or trauma)
- Increased arousal and avoidance of similar situations
- Intrusive memories, nightmares, flashbacks, detachment from others

16.4 ORGANIC PSYCHOSES

Chronic Organic Psychotic Conditions
- Alcoholic Psychoses
  - Wernicke’s encephalopathy
    - Rapid onset of delirium, ataxia, oculomotor dysfunction (ophthalmoplegia and nystagmus)
    - Secondary to thiamine deficiency
    - Mortality is 10% to 20% from comorbid alcohol-related diseases (infection, hepatic failure)
    - Treatment - replete thiamine early, correct hypomagnesemia
    - May be precipitated by treatment with dextrose before thiamine in chronic alcohol-abusing patients
  - Korsakoff’s psychosis
    - Anterograde and retrograde memory impairment, confabulation
    - Present both with and independent of Wernicke’s encephalopathy
    - Low likelihood of recovery despite thiamine replacement
- Drug Psychoses - see substance dependence section and toxicology chapter

Delirium
- Global cognitive impairment
- Disturbed level of consciousness and awareness
- Sensory misperceptions (visual, olfactory, tactile hallucinations)
- Autonomic abnormalities often present (fever, tachycardia, hypertension)
- Poor short-term memory
- Fluctuating course over hours or days
- Sudden onset
- Multiple etiologies - infectious, vascular, intoxication or withdrawal states, metabolic, trauma, CNS disease
- Treatment
  - Treat underlying cause
  - Provide pharmacologic sedation (haloperidol, benzodiazepines) as necessary

Dementia
- Characterized by disturbed abstract thought, judgment, personality, and higher cortical functions
- Impaired short- and long-term memory
- Intact consciousness and perception
- Insidious onset
• Gradual course
• Reversible causes of dementia
  o Medication side-effects/polypharmacy/chemical intoxication
  o Pseudodementia - dementia symptoms caused by depression
  o Normal pressure hydrocephalus - ataxia, confusion, urinary incontinence
  o Space-occupying lesions

16.5 PATTERNS OF VIOLENCE/ABUSE/NEGLECT

Domestic
• Child Abuse
  o 1 million cases annually of suspected physical, sexual, emotional child abuse and neglect in the United States
  o Higher incidence associated with
    ■ Parental substance abuse or other mental illness
    ■ Socioeconomic stressors
    ■ Domestic violence in home
    ■ Isolation of parent
  o History
    ■ Incompatible with injuries
    ■ Unexplained or poorly explained injuries
    ■ Delay in seeking treatment
  o Abusive injuries - physical clues
    ■ Trunk, upper arms, upper legs, neck and face and perineal area - usually well protected in accidental injuries
    ■ Injuries in various stages of healing
    ■ Head injury
      a Leading cause of fatal injury from abuse
      D Shaken baby syndrome
        — Violent shaking causes coup-contrecoup injury from rapid deceleration
        — Children less than 1 year old
        — May have no external signs of head trauma
        — Subdural or subarachnoid bleeds, diffuse axonal injury, retinal hemorrhages, secondary cerebral edema, anterior and posterior rib fractures
    ■ Abdominal injury
      a Second leading cause of fatal injury from abuse
      D Usually secondary to blunt trauma
      D Liver or splenic lacerations, duodenal hematoma, pancreatitis, perforation, obstruction
    ■ Skeletal injury — suspicious fractures
      D Spiral fractures of long bones caused by twisting
      D Metaphyseal fractures
        — From tugging on an extremity
        — Bucket-handle or corner chip injury
      a Rib (posterior) and scapula fractures
      D Spinous process fractures
Skull fractures (other than parietal fractures)
   - Depressed
   - Comminuted
   - Crossing suture lines

Cutaneous injury

Burns
   - Stocking-glove pattern
   - Anogenital region
   - Immersion burns: usually second degree
   - Patterned burns (curling iron, iron)
   - Cigarette burns

Bruising
   - Patterned bruises of inflicting instrument
   - Colors of bruising progress from purple to green to yellow to brown over time
   - Note: ambulatory children are likely to have accident-related bruising over bony prominences
       (shins, forehead)

   - Human bite marks

Child sexual abuse

Indications
   - Contact with genitalia; penetrating and non-penetrating injury
   - Oral palate injuries - bruising, petechiae
   - Hymen tears, petechiae, hematomas, vaginal tears, vaginal discharge
   - Anal lacerations, abnormal anal tone

Treatment
   - After stabilization, initiate institutional protocol for workup (e.g., CBC, coagulation studies, urinalysis, liver function studies, radiographic skeletal survey, CT head, ophthalmologic examination for retinal injury, photographs of external injuries) and treatment of abuse, including appropriate consultants and child protective services

   - Sexual abuse - may need to give antibiotics for prophylaxis against sexually transmitted diseases

Intimate Partner Abuse

Physical, sexual, emotional, and economic abuse

Highest rates in women 16 to 34 years old, but heterosexual and homosexual males are also victims

- 2% to 4% of women's emergency department visits are related to intimate partner abuse

- Increased risk of violence against women if partner has any of the following:
  - Ethanol or substance abuse
  - Unstable employment
  - Past arrest
  - Low education level
  - History of abuse as a child

Murder rates by intimate partner violence

- 33% of murdered women and 4% of murdered men are killed by their intimate partners
- Majority of deaths by guns
- Men kill partners when women attempt to leave.
- Women kill partners in self-defense or retribution.
o Presentations
  ■ Direct physical injury
    D Head
    D Face
    D Neck
  n Areas covered by clothing
  ■ Anxiety and depression
  ■ Pregnancy complications
  ■ Chronic pain - abdominal, pelvic, head, neck
  ■ Exacerbations of chronic illnesses

o All women who present with signs/symptoms suggestive of domestic abuse must be screened; the questions should cover abuse against dependents.
  ■ Assess immediate risk.
  ■ Ensure patient that help is available,
o Disposition
  ■ Refer to shelter/safe house if patient does not require admission.
  ■ Provide instructions to ensure safety should the patient want to return home.

• Elder abuse
  o Neglect (most common); physical, psychological, and verbal battery; financial abuse
  o Contributed to by isolation resulting from physical illness, disability, and mental illness
  o Two thirds of victims are women, the majority are Caucasian, almost half are clinically depressed,
o Most perpetrators are family members,
o Physical findings
  ■ Injuries in various stages of evolution
  ■ Delay in seeking treatment
  ■ Injuries inconsistent with history
  ■ Contradictory explanations given by patient and caregiver
  ■ Lab findings indicating underdosage or overdosage of medication
  ■ Bruises, welts, lacerations, rope marks, burns, genital infections
  ■ Dehydration and/or malnutrition
  ■ Decubitus ulcers, poor hygiene
  ■ Signs of emotional withdrawal, depression, agitation, or infantile behavior

o Screening for elder abuse is imperative; reporting is mandatory.
o Immediate care
  ■ Treat physical symptoms.
  ■ Ensure patient safety.
  ■ **Obtain a court protective order if necessary.**
  ■ Set up support services for caregivers,
o Disposition
  ■ Safe home placement
  ■ Discharge home if patient is competent and refuses intervention
Homicidal Risk
- Hostile behavior, verbal aggressiveness, and statements about violent intent must be taken seriously.
- Tarasoff versus Regents of the University of California, II (1976) - duty to protect a third party when it is believed there is a risk of serious bodily harm and when the potential victim is reasonably identifiable
- Inform law enforcement or notify third party directly of potential harm
- Patient may need to be committed involuntarily if there is a concern about homicidality.

Sexual Assault
- Majority of victims are female; mean age, 20 years old
- Assailant is usually known to victim.
- Lifetime estimates are 1 in 4 women, 1 in 7 men
- Usual route of abuse is penile-vaginal intercourse, followed by digital-vaginal penetration, then oral-genital contact and anal intercourse
- Injuries
  - Genital injuries - worse in adolescents than adults
  - Non-genital injuries - extremities, head, and neck
  - Psychological - counseling is paramount
- Emergency department considerations
  - Provide psychological support.
  - Refer to appropriate resources, including sexual assault counselor,
  - Treat physical injuries.
  - Collect legal evidence while preserving chain of custody.
    - Document all collection, handling, transfer, and storage of evidence, including evidence location.
    - Transfer of evidence must be documented with dates, times, and signatures of the people who are transferring and accepting items.
  - Document pertinent history with thorough physical exam,
  - Prevent/screen for sexually transmitted diseases, hepatitis, pregnancy, HIV

Staff/Patient Safety
- "4% to 8% of patients presenting to the emergency department carry weapons
- Predictors of patients who may become violent:
  - Male
  - History of violence
  - Drug or alcohol abuse
- Signs of impending violence
  - Insistent, progressively louder speech, threatening comments
  - Tense posture, motor restlessness, clenching fists, throwing objects
- Use of physical restraints - for violent patients in whom verbal techniques have not worked to prevent harm to self or others
- Emergency department methods for reducing violence
  - Security personnel visible to patients
  - Metal detectors
  - Patient searches - legal if conducted in a non-discriminatory manner
  - Alarm systems - allow rapid response by security personnel
  - Limited access to emergency department - one or two entrances, especially during evening hours
16.6 PERSONALITY DISORDERS - SET OF PERVERSIVE MALADAPTIVE TRAITS CAUSING DISTRESS AND IMPAIRED FUNCTIONING

Antisocial Personality
- Males > Females
- Long-standing disregard for and violation of others’ rights
  - Criminal behavior, assault
  - Lying
- Irritable, aggressive, impulsive, deceitful, irresponsible
- The person lacks remorse or is unable to empathize.
- High levels of substance abuse

Borderline Personality
- Females > Males
- Unstable, intense interpersonal relationships, moods, and self-image
- Recurrent suicidal gestures, threats, self-mutilation
- Chronic feelings of emptiness
- Splitting
  - Inability to feel ambivalently about others
  - People are characterized as “all good” or “all bad”
- Impulsivity with spending, sex, gifts

Histrionic Personality
- Females > Males
- Excessive emotion and attention-seeking
- Inappropriately seductive and provocative behavior
- Shallow expression of emotion, speech lacks detail

Narcissistic Personality
- Grandiose, self-important, requires excessive admiration
- Entitled and arrogant

Paranoid Personality
- Unwarranted suspicion and mistrust of others without basis
- Believes benign remarks to be threatening
- Bears grudges; guarded and suspicious

16.7 FACTITIOUS DISORDERS AND MALINGERING - SYMPTOMS ARE INTENTIONALLY PRODUCED OR FEIGNED

Factitious Disorders - no clear external incentives are present
- Motivation is to assume sick role
- Patients do not have voluntary control over their actions
- Females > Males
  - Munchausen’s syndrome - severe form of factitious disorder
    - Males > females
    - Dramatic presentation of severe illness using medical jargon
    - Symptoms may change as workup proves negative
    - Extensive history of admissions, surgeries in multiple cities and hospitals
Munchausen’s by proxy - production of disease in a child by parent or caregiver
- Perpetrator assumes sick role through child
- Almost all perpetrators are mothers
- Form of child abuse - contact appropriate child welfare services to protect victim

Malingering - external incentives outside sick role (financial gain, evading the law, avoiding work, seeking shelter)
- Patients have control over their actions.
- Symptoms do not correlate with objective findings.
- History of antisocial behavior is often present.
- Patients tend to be noncompliant with prior treatment and uncooperative with evaluation.
- Drug-seeking behavior - symptoms feigned to obtain medication
  - Patients are manipulative, demanding
  - Resist non-pharmacologic treatment recommendations
  - May report allergies to multiple non-narcotic agents
  - Utilize multiple physicians

16.8 PSYCHOSOMATIC DISORDERS - SYMPTOMS ARE UNCONSCIOUS, UNINTENTIONALLY PRODUCED

Hypochondriasis - preoccupation with fear of serious illness despite appropriate evaluation and reassurance
- Minor symptoms are exaggerated.
- Normal sensations are misinterpreted.
- “Doctor shopping” - multiple medical encounters for the same complaints
- Equal incidence among men and women

Hysteria/Conversion - sudden-onset loss of physical functioning, usually neurologic, without anatomic or physiologic etiology
- Chief complaint of motor (tremor, seizure, paralysis, incoordination) or sensory (anesthesia, blindness) dysfunction
- Often temporally related to psychosocial stressor
- A diagnosis of exclusion
CHAPTER 17
Renal and Urogenital Disorders

Bernard L. Lopez, MD, MS, and Matthew Brooks, MD

17.1. ACUTE AND CHRONIC RENAL FAILURE

General
- Definition
  - Deterioration of renal function over hours or days (acute) or longer (chronic)
  - Results in accumulation of metabolic waste products
  - 50% reduction in creatinine clearance or 50% increase in serum creatinine
- Classification
  - Pre-renal (40%-80%)
  - Renal (25%)
  - Post-renal (2%-5%)

Pre-renal
- The most common cause of acute renal failure (ARF); caused by a reduction in renal blood flow, which leads to acute reduction of glomerular filtration rate (GFR) and renal cellular ischemia
- Causes of reduction in renal blood flow
  - Volume loss: hemorrhage, GI loss, diuresis
  - Fluid sequestration: cirrhosis, nephrotic syndrome, burns, septic shock
  - Renal artery disease: bilateral stenosis, thrombosis, embolism

Intrinsic Renal
- Intrinsic diseases of the glomerulus, interstitium, or tubule, with acute tubular necrosis (ATN) accounting for the majority
- Glomerular disease (acute glomerulonephritis): systemic lupus erythematosus (SLE), Henoch-Schönlein purpura, Wegener’s granulomatosis, Goodpasture’s syndrome
- Tubular diseases: ischemia, nephrotoxins (aminoglycosides, contrast media, methotrexate, heavy metals), myoglobinuria
- Vascular diseases: scleroderma, thrombotic thrombocytopenic purpura, hemolytic-uremic syndrome, malignant hypertension (HTN), polyarteritis nodosum
Post-renal

- Results from obstruction to urinary outflow with increased back pressure on kidneys
- Causes include benign prostatic hypertrophy (BPH), neurogenic bladder, calculi, blood clots, cancer, urethral strictures.

Clinical Features of Renal Failure

- Decreased urine output
- Thirst, dizziness, weakness
- Hematuria, edema, hypertension (nephritic syndrome)
- Fever, rash, arthralgia (interstitial nephritis)
- Flank pain, inability to void, overflow incontinence (BPH)
- Other symptoms directly related to conditions causing acute renal failure

Diagnosis

- Urine output
  o Anuria (<100 ml/day)
  o Oliguria (<400 ml/day)
  o Non-oliguria (>400 ml/day) (50% to 60% are non-oliguric)
- Urinalysis
  o Normal (pre-renal ARF and some causes of post-renal ARF, such as BPH)
  o Heme positive on dipstick without RBCs on microscopy (rhabdomyolysis)
  o Proteins (glomerulonephritis, nephritic syndrome, nephrotic syndrome)
  o Granular casts (ATN, interstitial nephritis)
  o WBC casts (pyelonephritis, interstitial nephritis)
  o RBC casts (glomerulonephritis)
  o Crystals (stones)
  o Urine specific gravity: >1.018 favors pre-renal, <1.012 favors ATN
  o Urine sodium (mEq/L): <20 favors pre-renal, >40 favors ATN
- Serum chemistry
  o Elevated creatinine: <1.0 mg/dl is normal; 2.0 mg/dl equals an approximate 50% reduction in GFR
  o Elevated blood urea nitrogen (BUN): suggestive of acute renal failure, but there are other causes of high BUN (GI bleeding, use of steroids or tetracycline)
  o BUN/Cr ratio >20 is typically associated with pre-renal causes of ARF
  o Hyperkalemia
  o Hyponatremia
  o Hypocalcemia
  o Hyperphosphatemia
  o Hyperuricemia
  o Metabolic acidosis
- Renal imaging studies
  o Ultrasound: non-invasive, lower cost, no contrast; allows identification of hydronephrosis (see Image #12), Doppler analysis of renal blood flow
o Radionuclide scanning
  ■ 90% sensitivity for high-grade obstruction
  ■ No nephrotoxic risk
  ■ Not reliable for visualization of the site of obstruction

o CT
  ■ Defines anatomy of obstruction
  ■ Most useful for stones

o MRI
  ■ 100% sensitive, 96% specific for obstruction
  ■ Helps to assess renal function without contrast
  ■ Not easily available from ED

Complications of Renal Failure
  • Uremia
  o Affects multiple organ systems
  o Problem occurs with the buildup of nitrogenous waste, derangements in vitamin D, and parathyroid hormone metabolism
  o Pericarditis with or without pericardial effusion
    ■ Effusion may be serous or hemorrhagic
    ■ Occurs in 6% to 10% of patients with end-stage renal disease (ESRD) prior to dialysis
    ■ Presents with pleuritic chest pain with or without friction rub
    ■ Treat with intensive dialysis for 10 to 14 days with NSAIDs
    ■ Pericardiocentesis may be necessary if evidence of tamponade is found,
  o Uremic encephalopathy
    ■ Variable presentation, including memory loss, impaired concentration, delusions, or lethargy
    ■ On exam, the patient may exhibit myoclonic jerks or twitches, fasciculations, asterixis, tetany, or seizures.
    ■ Patient may also present with peripheral neuropathies.
    ■ The exact mechanism is unknown.
    ■ Treat by correcting metabolic derangements and giving anticonvulsants for seizures.
    ■ Patients should also have CT scan to rule out intracranial hemorrhage,
  o Anemia
    ■ Caused by erythropoietin deficiency
    ■ May also be caused by vitamin deficiencies or iron deficiency
    ■ Anemia is normocytic, normochromic, hypoproliferative.
  o Bleeding diathesis
    ■ Platelet dysfunction occurs with uremia.
    ■ Patients have increased bleeding time.
    ■ The gastrointestinal tract is the most common site.
    ■ Improves with dialysis
  o Hyperkalemia
    ■ Causes potentially life-threatening dysrhythmias
    ■ Obtain ECG if suspected and treat if signs of hyperkalemia are noted (peaked T waves, widened QRS, sine wave pattern)
    ■ Potassium level of 6 mEq/L should be considered potentially lethal.
- Treat with calcium gluconate, which counteracts hyperkalemia.
- Insulin with glucose, bicarbonate, and albuterol transiently lowers potassium
- Dialysis and kayexalate remove excess potassium.

- Volume overload results in pulmonary edema and effusion,
- Decreased cellular immunity increases the predisposition to infection.

**Indications for Dialysis**

- Severe metabolic acidosis
- Hyperkalemia (>6.5 mEq/L)
- Pericarditis
- Uremic encephalopathy
- Severe volume overload
- Acute toxicity from dialyzable agents (ethylene glycol, aspirin, methanol, lithium)

**Hematuria**

- Causes
  - Most common: kidney stones, UTIs, bladder and kidney carcinoma, and trauma
  - In older populations, carcinoma becomes more common and prostatic hypertrophy becomes more prevalent,
  - Multisystem diseases like SLE or Goodpasture’s syndrome
  - Coagulopathy, infarction, polycystic kidney disease, tuberculosis, or vascular malformation

- Clinical Features
  - Costovertebral angle tenderness may indicate tumor, calculi, or pyelonephritis,
  - Palpable enlarged kidney indicates polycystic kidneys or malignancy,
  - Prostate may be enlarged or tender in prostatic hypertrophy or prostatitis,
  - In women, a vaginal source should be ruled out.
  - A new heart murmur or new-onset atrial fibrillation may indicate a renal embolism.

- Management
  - Check BUN and creatinine to assess underlying renal function.
  - Urinalysis to look for red cell casts indicating intrinsic renal disease or pyuria indicating an infectious source
  - CT is indicated if the history suggests renal colic or disorders of the upper urinary tract,
  - Patients who have no other symptoms and who do not have azotemia or anemia may be monitored as outpa-
    tients.

### 17.2 COMPLICATIONS OF HEMODIALYSIS

**Air Embolism**

- Typically presents as acute dyspnea, chest tightness, and loss of consciousness
- Can progress to cardiac arrest

**Treatment**

- Place patient in left lateral decubitus position,
- Clamp venous lines
- Percutaneous aspiration of bubbles from right ventricle
- IV steroids
- Hyperbaric chamber
Hypotension
• Most frequent complication (10%-13% incidence)
• Excessive ultrafiltration of fluid is the main cause.
• Pre-dialysis volume deficit (vomiting, diarrhea, sepsis, GI bleeding) is a contributing factor.
• Autonomic dysfunction, especially in diabetics
• Antihypertensives can block sympathetic tone.
• Myocardial ischemia and cardiac dysrhythmias

Dialysis Disequilibrium Syndrome (DDS)
• A result of cerebral edema caused by excessive clearance of solutes during dialysis (temporary brain osmolality>blood osmolality)
• Nausea, vomiting, and hypertension
• Can lead to seizures, coma, and death
• Treatment consists of stopping dialysis and administering IV mannitol.

Anemia
• Erythropoietin deficiency
• Leads to left ventricular dilation and hypertrophy
• Patients should be on erythropoietin therapies monitored by their primary care physician or nephrologist; blood transfusion if symptomatic

Vascular Access Complications
• Thrombosis
  o Loss of thrill/bruit
  o Can be treated within 24 hours by means of angiographic clot removal, angioplasty, thrombolytics
• Infection
  o More common in vascular grafts than in fistulas
  o Most common organism is Staphylococcus aureus
  o Fever, high WBC count, and hypotension are common findings,
  o Classic signs such as edema, redness, warmth and discharge are often not present.
• Bleeding
  o Can be from aneurysm, anastomotic leak, or excessive anticoagulation
  o Should be controlled initially with gentle pressure for 10 minutes, and patient should be observed for 1 to 2 hours.
  o Vascular surgeon should be consulted if bleeding cannot be controlled,
  o Protamine sulfate should be given to reverse the effects of heparin,
  o DDAVP (desmopressin) can be given as an adjunct.
• Aneurysms
  o True aneurysms are rare (<4%).
  o Most are asymptomatic, rarely rupture
  o Pain and impingement neuropathy are rare clinical symptoms.

Peritoneal Dialysis
• General
  o Peritoneal surface serves as the blood-dialysate interface.
  o Continuous ambulatory peritoneal dialysis (CAPD) consists of four daily exchanges using 2 L of dialysate solution.
o Approximately 10 L fluid is drained over 24 hours (net negative 2 L/day).

- Complications of Peritoneal Dialysis
  - Most common: peritonitis
  - Incidence is one episode per year per person
  - Mortality rate is 2.5% to 12.5%
  - Clinical signs are abdominal pain, fever, and rebound tenderness,
  - Peritoneal fluid is cloudy, WBC count >100, >50% PMN
  - Gram stain is positive in 10% to 40% of cases.
  - \(S.\) \textit{epidermidis} is the most common (40%) organism isolated; others include \(S.\) \textit{aureus} (10%), streptococci (15%-20%), gram-negative organisms (15%-20%), anaerobes (5%), and fungi (5%).
  - Infection around a catheter is much less frequently caused by \(S.\) \textit{aureus} and \textit{Pseudomonas}.

- Treatment of CAPD-Associated Peritonitis
  - Patients who are nontoxic can be discharged to home.
  - Vancomycin, 1 g IV, then 30-50 mg/kg intraperitoneal dose with each 2-L exchange
  - Gentamicin should be added to dialysate fluid for gram-negative rods seen on gram stain,
  - Use both vancomycin and gentamicin if the gram stain is negative,
  - Treatment should continue for 10 days after negative culture.

17.3 GLOMERULAR DISORDERS

Acute Glomerulonephritis

- Pathophysiology
  - Proliferation of glomerular tissue
  - Secondary to inflammation triggered by some immunologic mechanism

- Causes of Acute Glomerulonephritis
  - Post-infectious: Group A P-hemolytic streptococci are most common; other causative organisms are staphylococci, \textit{Mycoplasm}, \textit{Salmonella}, \textit{Brucella}.

- Clinical Features of Post-Streptococcal Glomerulonephritis
  - Most cases occur in the age group of 5 to 15 years
  - Only 10% of cases occur in patients above the age of 40.
  - Male to female ratio of 2:1
  - Symptom onset is abrupt.
  - Latent period of 1 to 3 weeks post-streptococcal infection; may occur as late as 6 weeks after infection
  - Nonspecific symptoms include weakness, fever, abdominal pain, and malaise,
  - Hypertension, edema, hematuria, proteinuria, and impaired renal function are typical,
  - Gross hematuria is reported in 30% of pediatric patients.

- Diagnostic Tests
  - \textbf{CBC may show dilutional anemia.}
  - Electrolytes, BUN, and creatinine may reveal renal insufficiency.
  - Urinalysis reveals dark urine, specific gravity >1.020, proteinuria, and microscopic hematuria with RBCs and red cell casts
  - Other tests may include quantitative antistreptolysin titer (increased in 60%-80% of patients with antecedent throat but not skin strep infection) and blood, throat, and skin cultures for strep
• Treatment
  o Post-streptococcal: oral antibiotics (penicillin) to eradicate streptococcal infection if still present (early antibiotic therapy does not affect the development of post-streptococcal glomerulonephritis)
  o Patients presenting with renal failure, symptomatic hypertension, or nephrotic syndrome should be admitted.

Nephrotic Syndrome
• Pathophysiology
  o Proteinuria caused by abnormalities in glomerular basement membrane,
  o Hypoalbuminemia occurs secondary to increased filtration,
  o Edema results from the following:
    ■ Decreased oncotic pressure from low albumin
    ■ Increased sodium retention in collecting tubules secondary to increased aldosterone secretion
  o Hyperlipidemia and lipiduria occur.
• Causes
  o Minimal change disease
    ■ 90% of patients with nephrotic syndrome are under age 10
    ■ Idiopathic in many cases
    ■ Associated with NSAID use
  11 Associated with malignancy, especially Hodgkin’s disease
  o Focal segmental glomerulosclerosis
    ■ Most common cause in adults
    ■ Idiopathic
    ■ Associated with HIV and heroin use
• Clinical Presentation
  o Dependent edema with proteinuria
  o Nausea and vomiting may be present if the GI tract is edematous,
  o Intravascular hypovolemia from severe hypoalbuminemia
  o May progress to ARF
  o Increased incidence of thromboembolic events and infection
• Diagnosis
  o Heavy proteinuria (>3.5 gm/24 hr)
  o Biopsy needed (except in patients younger than 10 years, due to higher prevalence of minimal change disease)
• Treatment
  o Most treatment is supportive.
  o Glucocorticoid therapy is recommended for minimal change disease,
  o Sodium restriction and loop diuretics for edema
  o Angiotensin-converting enzyme inhibitor (ACEI) for proteinuria or hypertension

17.4 INFECTION

Pyelonephritis
• Clinical Features
  o Fever and shaking chills
  o Flank pain and tenderness
  o Nausea/vomiting
o Dysuria
o White blood cells in clumps or casts; bacteriuria

• Risk Factors
  o Pregnancy
  o Recurrent UTI (three or more UTIs in past year)
  o Diabetes or other immunocompromised state
  o Structural abnormality of urinary tract
  o Instrumentation of urinary tract
  o Neurogenic bladder
  o Obstruction caused by stone or enlarged prostate

• Complications
  o Acute papillary necrosis
  o Perinephric abscess
  o Septic shock

• Treatment
  o Outpatient management is acceptable for mild to moderate cases in young healthy patients who can take oral medication and fluids.
  o Inpatient management if the patient has signs of systemic toxicity such hypotension or tachycardia, is pregnant, is older, has comorbid conditions, is vomiting, or has urologic abnormalities.

• Pharmacotherapy
  o First-line agent: fluoroquinolone, second or third generation, PO or IV
  o Second-line agents
    - TMP/SMX: if bacterial sensitivities are known
    - Ampicillin + gentamicin for newborns and young infants being treated as inpatients
  o Alternatives
    - Third-generation cephalosporin (if first- and second-line agents cannot be used)
    - Amoxicillin/clavulanate after bacterial sensitivities are known

**Urinary Tract Infections**

• Epidemiology
  o UTI is defined as significant bacteriuria with clinical symptoms,
  o 20% of women are affected at some point in their lives.
  o The five groups at increased risk are neonates, girls after infancy, sexually active young women, pregnant women, and males > 50 years of age.
  o In the neonatal period, more males are affected than females (1.5:1).
  o 2% of pre-school age children are affected,
  o 5% of school-age girls are affected,
  o Bacteriuria is rare in males younger than age 50.
  o Dysuria and urinary frequency in younger males are more likely from STDs,
  o 40% of elderly women in nursing homes have chronic bacteriuria.

• Bacteriology
  o 80% to 90% of UTIs are caused by *E. coli*.
  o Acute urethral syndrome (dysuria with $10^2$—$10^4$ CFU/ml) is caused by *Chlamydia trachomatis* or *S. saprophyticus*. 
o Asymptomatic bacteriuria (>10^5 CFU/ml of one bacterial species on two urine cultures in a patient without symptoms) is common in pregnant women, nursing home patients, and patients with indwelling catheters, 
  o *Pseudomonas* or enterococcus can be a cause of “complicated” UTI.  
  o *Klebsiella, Proteus*, and *Pseudomonas* combined cause 20% of UTIs.  

- Pathophysiology  
  o Urine is a good medium for bacteria.  
  o Healthy bladder mucosa inhibits bacterial growth by producing organic acids that lower the urine pH.  
  o Lack of estrogen in older women causes change in vaginal flora (loss of lactobacilli), which encourages colonization by *E. coli*.  
  o The concentration of bacteria in the bladder may increase after sexual intercourse; therefore, prompt emptying of the bladder minimizes the risk of UTI.  

- UTI are categorized into three clinical syndromes:  
  o Simple UTI (acute cystitis)  
  o Subclinical pyelonephritis (not clinically distinguishable from acute cystitis)  
  o Acute pyelonephritis  

- Predisposing factors for complicated UTI  
  o Diabetes  
  o Pregnancy  
  o Obstruction caused by stones  
  o Urinary tract structural abnormality  

- Clinical features  
  o Dysuria, frequency, lower abdominal pain  
  o Fever, chills, nausea, and vomiting suggest acute pyelonephritis, not cystitis,  
  o Flank pain and tenderness can be referred pain or manifestations of pyelonephritis,  
  o Dysuria and urethral discharge in males suggest STD.  

- Diagnosis  
  o Presence of >10 WBCs/hpf and bacteria in a urine specimen  
  o Urine nitrite has high specificity (>90%) but low sensitivity  
  o Leukocyte esterase has low sensitivity (48%) for the presence of pyuria.  

- Indications for urine culture  
  o All pregnant females, children, and adult males  
  o Pyelonephritis  
  o Hospitalized patients  
  o Nursing home patients  
  o Patients with recurrent UTI  
  o Patient not responding to treatment  
  o Immunocompromised states  

- Treatment  
  o 3-day treatment for uncomplicated cystitis in nonpregnant women  
    - TMP/SMX or ciprofloxacin for 3 days is adequate  
    - Nitrofurantoin requires 5 days of treatment  
  o 7- to 10-day therapy is recommended for the following:  
    - Symptoms longer than a week  
    - Recurrent UTIs
Diabetes
- Patients older than 65 years
- Pregnant women (nitrofurantoin is a safe alternative to ciprofloxacin and TMP/SMX in pregnancy)
- Pyelonephritis (treatment lasts up to 14 days)

Urethritis
- Purulent or mucopurulent discharge from the urethra
- Diagnosis is based on clinical findings.
- Patients complain of dysuria and pruritis in addition to discharge.
- *N. gonorrhoeae* and *C. trachomatis* are the most common causes.
- Herpes simplex virus (HSV), *Ureaplasma urealyticum*, and *Trichomonas vaginalis* are less frequent causes.
- Obtain urethral cultures for *N. gonorrhoeae* and *C. trachomatis*.
- Treat patients with ceftriaxone, 125 mg IM, PLUS azithromycin, 1 g PO, or doxycycline, 100 mg PO bid X 7 days.
- If *Trichomonas* is a cause, treat with metronidazole.

17.5 MALE GENITAL CONDITIONS

Genital Lesions
- Genital Ulcers
  - Causes
    - Herpes simplex virus
      - D Most common cause of genital ulceration in the United States
      - D Multiple small painful vesicles with erythematous base (see Image #19)
      - D Many patients have recurrent episodes.
    - Syphilis
      - D Caused by *Treponema pallidum*
      - 1-2 Indurated chancre with clean edges; often painless (see Image #15)
      - RPR and VDRL serology (nontreponemal tests) are sensitive but not specific.
      - D Treponemes can be visualized by darkfield microscopy.
    - Chancroid
      - D Caused by *Haemophilus ducreyi*
      - D Ulcer has ragged edges and gray or yellow exudate.
      - D May have multiple ulcers; usually is very painful
    - Behcet's disease
      - D Vasculitis resulting in recurrent mucocutaneous ulcers
      - D Oral aphthous ulcers
      - D Genital aphthous ulcers
      - D Uveitis
      - D Occasional neurologic symptoms
  - Workup
    - Serologic tests such as RPR for syphilis
    - Darkfield microscopy if available
    - Viral culture for HSV
    - Screen for associated STDs, e.g., gonorrhea and *Chlamydia*
Treatment

- Patients with HSV infection should receive acyclovir to decrease severity and time course of disease.
- Primary syphilis is treated with penicillin G IM.
- A single dose of azithromycin or ceftriaxone is effective for chancroid.

Genital Warts

- Caused by human papillomavirus
- Results from sexual contact
- Presents as multiple painless warts
- Warts are variable and range from smooth flattened papules to a verrucous papilliform appearance,
- May be removed by the following:
  - Chemicals such as podophyllin or trichloroacetic acid
  - Immune modulation with intralesional interferons
  - Excision with cryotherapy or laser therapy

Hernias (see Chapter 3, Abdominal and Gastrointestinal Disorders)

Inflammation/Infection

- Balanitis
  - General
    - Inflammation of the glans penis
    - Balanoposthitis: involvement of the prepuce
    - Most common complication is phimosis
  - Clinical features
    - Inability to retract prepuce if phimosis is present
    - Discharge around the glans penis
    - Tenderness
    - Erythema and edema
  - Causes of balanitis
    - Infection is the primary cause, although contact dermatitis, trauma, or chemical irritation may play a role.
    - Lack of aeration and irritation caused by the presence of smegma lead to inflammation.
    - Diabetes is the most common predisposing condition.
    - Poor personal hygiene
    - Morbid obesity
  - Bacteriology
    - Candida, most commonly in diabetics
    - Anaerobes
    - Group B streptococci
    - Gardnerella
    - Trichomonas
    - Borrelia vincenti
  - Treatment
    - Daily retraction of foreskin; wash with warm water to cleanse the penis
    - Topical clotrimazole
    - Obtain culture from discharge and treat with appropriate antibiotic; consider first-generation cephalosporin empirically.
    - Circumcision electively
- Epididymitis
  o Etiology
    - Most common cause is bacterial infection.
    - In patients younger than 35, usually STD: gonorrhea or Chlamydia
    - In patients older than 35, usually coliforms
    - Coliforms are also seen occasionally in infants with congenital urologic anomalies,
  o Presentation
    - Onset is usually more gradual than that of torsion.
    - Pain occurs in the lower abdomen, inguinal canal, scrotum, and testicle—alone or in combination
    - Voiding or urethral symptoms are reported by some patients,
  o Physical exam
    - Early: nodularity and tenderness of affected epididymis, which normally sits like an apostrophe on the superoposterior testis
    - Later: sulcus, which is normally palpable between the epididymis and testis, becomes obliterated, and a large scrotal mass develops (epididymo-orchitis), which is very hard to differentiate from torsion and cancer
    - At this stage, the patient may appear toxic and require IV antibiotics,
  o Laboratory
    - CBC is not helpful but may show leukocytosis.
    - Urinalysis is helpful only if positive for WBCs, which are present in about half of cases; urine culture is needed in children and older patients
    - Consider culture of urethra for GC/ Chlamydia.
  o Management
    - Antibiotics
      - Presumed STD: ceftriaxone, 250 mg IM, then doxycycline, 100 mg BID x 10 days
      - Presumed coliform: TMP-SMX BID for 10 to 14 days; alternatively, ciprofloxacin, 500 mg BID x 10 to 14 days
      - Analgesics with NSAID plus narcotics prn
      - Scrotal elevation and support ± bedrest to minimize pain
      - Urology referral is recommended for all patients.
      - Admit for high fever, signs of toxicity
      - Abscess formation may complicate epididymo-orchitis.
      - Scrotal ultrasound may help clarify the diagnosis,
  o Common pitfalls
    - Hot, tender, swollen scrotum may represent torsion, not epididymo-orchitis.
    - Most common misdiagnosis of testicular tumor is epididymitis.
- Orchitis
  o General
    - Acute infection involving the testis
    - Rare without initial epididymitis
    - Most frequent bacterial organisms are E. coli, Klebsiella, and Pseudomonas
    - Viral orchitis occurs in 20% to 30% of postpubertal boys with mumps
o Clinical features
  ■ Bacterial orchitis presents with fever and an acutely tender, swollen testicle, pyuria, and leukocytosis.
  ■ Viral orchitis presents with testicular swelling 4 to 6 days after the initial parotid infection, with resolution in 4 to 5 days.
  ■ Viral orchitis is unilateral in 70% of cases,

o Management
  ■ Obtain urinalysis, urine culture, and blood culture.
  ■ Exclude the diagnosis of torsion.
  ■ Treat bacterial orchitis with antibiotics directed against *E. coli, Klebsiella, Pseudomonas*, staphylococci, and streptococci - ceftriaxone, 250 mg IM, and either doxycycline or a fluoroquinolone for 10 days
  ■ Treat with NSAIDs for pain and scrotal elevation for comfort.
  ■ Viral orchitis requires supportive treatment only.

- Fournier’s Gangrene (see Image #2)

  o Polymicrobial infection
  ■ *E. coli* is the predominant aerobic organism; *Bacteroides fragilis* is the main anaerobe; other organisms are *Proteus, Staphylococcus, Enterococcus*, aerobic and anaerobic streptococci, *Pseudomonas*, and *Clostridia*.

  o Risk factors for the development of Fournier’s gangrene are listed below:
    ■ Diabetes
    ■ Advanced age
    ■ Alcoholism
    ■ Malignancy
    ■ Perianal, ischiorectal, and perirectal abscesses
    ■ Urinary extravasation from urethral strictures
    ■ Instrumentation
    ■ Chronic steroid use
    ■ HIV

  o Incidence
    ■ Relatively uncommon
    ■ Most cases occur between the ages of 30 and 60

  o Mortality/morbidity
    ■ Wide range reported (4% to 75%)

o Clinical findings
  ■ Most cases begin with an insidious onset of pain or pruritis
  ■ Pain out of proportion to physical findings
  ■ Swelling and redness follow pain
  ■ Fever and chills
  ■ Skin edema, induration, blisters, gangrene, sloughing
  ■ Foul odor due to anaerobic infection

o Diagnosis
  ■ Primarily clinical
  ■ Plain x-ray films may reveal gas in the tissue planes.
  ■ Absence of gas in tissue does not exclude the diagnosis.
  ■ CT scan to define the extent of the disease
  ■ Ultrasound showing subcutaneous gas in the scrotal wall
o Treatment
  ■ Wide surgical debridement
  ■ Immediate urologic consult is mandatory.
  ■ Early intravenous broad-spectrum antibiotics - penicillin/clindamycin combination, ceftriaxone, imipenem, or meropenem
  ■ Hyperbaric oxygen
    □ Used as an adjunctive treatment
    □ Useful when *Clostridial myonecrosis* is present
    □ Postulated to reduce systemic toxicity, prevent extension of the disease, inhibit bacterial growth
    □ Effectiveness is currently debated in the literature.

• Acute Prostatitis
  o Acute bacterial prostatitis is the most common presentation of prostatitis in the ED.
    o Risk factors
      ■ Diabetes
      ■ Dialysis
      ■ Immunocompromised state
      ■ Urethral instrumentation
    o Sexual transmission of bacteria is common; infection can be spread through hematogenous, lymphatic, and contiguous mechanisms,
    o Clinical symptoms
      ■ Fever, chills, malaise
      ■ Low back pain, lower abdominal pain, perineal and rectal pain, scrotal and penile pain
      ■ Urethral discharge, pain on ejaculation, dysuria
      ■ Prostate gland on exam is boggy, nodular, tender, and hot to touch. At times, the prostate may feel normal.
    o Bacteriology
      ■ 80% *E. coli*
      ■ *Neisseria gonorrhoea* and *Chlamydia trachomatis* are common causes of prostatitis in males under the age of 35.
    o Laboratory tests
      ■ Complete blood count and blood cultures in patients who appear toxic or septic
      ■ Urinalysis, urine culture
    o Treatment
      ■ Acutely ill patients should be admitted.
      ■ Acute obstruction is relieved by the suprapubic route (urology consult is recommended) to avoid urethral catheterization.
      ■ **Fluoroquinolones or TMP/SMX for 30 days as outpatient therapy; ceftriaxone or a fluoroquinolone as inpatient options**
      ■ In patients <35 years old, typical STD regimen of ceftriaxone and doxycycline may be used
  o Complications
    ■ Chronic prostatitis
    ■ Urinary retention
    ■ Abscess formation, which is especially prevalent in people with HIV
    ■ Cystitis and ascending urinary tract infection
o Prognosis: good with aggressive antibiotic treatment

**Structural**

- Phimosis
  o Phimosis is the inability to retract the foreskin over the glans penis,
  o Incidence is 1% of males over the age of 16
  o May be congenital
  o Acquired phimosis is caused by recurrent balanitis.
  o Definitive treatment is circumcision, but topical steroids may provide successful nonsurgical treatment.
- Paraphimosis
  o Inability to reposition the proximally retracted prepuce into its usual location over the glans penis,
  o Edema, pain, tenderness, and erythema
  o Constriction by the tight band of retracted foreskin may cause severe edema of the glans penis, leading to necrosis,
  o ED treatment
    - Manual reduction under penile block with plain lidocaine
    - Dorsal slit of the constricting band to facilitate reduction of the foreskin
    - Elective circumcision to prevent recurrence
- Priapism
  o Persistent, painful, ’nonsexually stimulated erection
  o Cause
    - Idiopathic
    - Sickle cell disease (most common cause in children)
    - Spinal cord injury
    - Self-injected medication for impotency
    - Leukemia
    - Thalassemia
    - Street drug usage (Ecstasy, cocaine, marijuana, alcohol)
  o Pathophysiology
    - Affects only the corpora cavernosa
    - Corpus spongiosum remains flaccid
    - Arterial (high flow) priapism is caused by penetrating trauma or blunt perineal injury; priapism induced by this mechanism is usually not painful
    - Veno-occlusive priapism is caused by sickle cell disease, leukemia, and low-flow states from medications/drugs
  o Treatment
    - External compression; ice packs are frequently unsuccessful
    - Terbutaline orally (5 mg followed by another 5 mg 15 minutes later)
    - Treatment of choice: terbutaline injected subcutaneously (0.5 mg, up to three injections q 20-30 min) in deltoid region
    - Hydration and exchange transfusion for priapism related to sickle cell disease
    - Intracavernous injection of dilute phenylephrine (10 mg [1 ml] in 500 cc of NS). Inject 10 to 20 cc into the corpora cavernosa after penile block with plain lidocaine; 20 to 30 cc of venous blood may need to be aspirated initially. Penis should be dressed with elastic bandage to help drain the blood.
    - Surgical procedure may be required if the above procedure fails.
• Prostatic Hypertrophy
  o Epidemiology
    ■ Most common in men over 50
    ■ There is some evidence of hyperplasia in 80% of men over 80.
  o Presentation
    ■ Frequency of urination
    ■ Hesitancy
    ■ Nocturia
    ■ Weak urinary stream
    ■ Gradual and progressive onset of symptoms
  o Differential
    ■ Bladder cancer
    ■ Bladder stone
    ■ Urethral stricture
    ■ Prostate cancer
    ■ Neurogenic bladder
  o Diagnosis
    ■ Digital rectal exam to palpate nodules and prostate size
    ■ Urinalysis for the presence of blood or leukocytes
    ■ Creatinine to assess for renal insufficiency
    ■ Post-void residuals
    ■ Prostate-specific antigen (PSA) may be slightly elevated in patients with benign prostatic hypertrophy (BPH); main purpose is screening for prostate cancer
  o Treatment
    ■ Referral to urologist
    ■ (Xj-Adrenergic antagonist, e.g., terazosin and tamsulosin
    ■ Surgical reduction (transurethral resection of the prostate [TURP]) may be necessary.
• Testicular torsion
  o Pathophysiology: testis twists on the spermatic cord and blood flow is interrupted
    ■ If untreated, ischemia leads to testicular necrosis.
    ■ Twisting is normally prevented by fixation of the posterior aspect of the testicle to the scrotal wall. Some males are born without this fixation (bell-clapper deformity), predisposing them to torsion.
  o Epidemiology
    ■ Peak incidence occurs around puberty, but the condition can occur at any age.
    ■ Often occurs in sleep or after strenuous activity
  o Presentation
    ■ Classic history is acute onset of unilateral testicular pain and scrotal swelling.
    ■ Pain may radiate to inguinal canal and abdomen.
    ■ Nausea and vomiting are common.
    ■ Voiding symptoms are rare.
    ■ Many patients have a history of similar pain that resolved on its own.
    ■ A history of trauma does not exclude torsion.
o Physical examination

- A kink may be felt in the spermatic cord, and the epididymis can be palpated anteriorly.
- Testis is very tender, but this is a nonspecific finding.
- High-riding testis with horizontal lie—a very suggestive finding
- Late findings: scrotal edema and discoloration
- Cremasteric reflex is absent (preserved cremasteric reflex is very uncommon in torsion).
- Prehn’s sign—elevation of testis relieves pain in epididymitis but not torsion—is not reliable

o Laboratory

- Urinalysis is typically normal.
- Mild leukocytosis is common,

o Management

- Rapid, tentative diagnosis should be made on the basis of the history and physical (H&P)
- Immediate urologic consultation on suspicion of diagnosis
- Time counts here: shorter ischemia time, better salvage rate. Detorsion within 6 hours is associated with a viable testis.
- Adjunctive diagnostic tools:
  - Technetium nuclear scan - ischemic testis does not “light up”; good sensitivity/specificity, but this is a time-consuming test that may cause delay in treatment; can be falsely negative if spontaneous detorsion occurs before imaging study
  - Color Doppler US - can determine specific anatomy of blood flow; may give more info than nuclear study about nearby structures/alternative diagnosis
  - It is not necessary to obtain such studies when there is a high clinical suspicion for torsion; definitive surgical exploration is paramount.
- Manual detorsion - a temporizing technique while awaiting surgery. After analgesia, twist outward, toward ipsilateral thigh (like opening a book) since most testes torse medially. Turn until pain is relieved. If pain increases, try other direction.
- Surgical exploration and bilateral orchiopexy, since predisposing anatomic defect is often bilateral

• Torsion of the Appendix Testis

o Pathophysiology

- Appendix testis is a remnant of the mullerian duct.
- Torsion of appendix testis is much more common than torsion of appendix epididymis.
- Located at superior pole of testis
- Twists on pedunculated base and becomes ischemic and painful

o Presentation

- Peak ages, 3 to 13 years; rare after age 20
- In prepubertal boys, appendix torsion is more common than testicular torsion.
- Pain is less severe than that associated with testicular torsion,

o Physical exam

- Early on, an isolated tender nodule can be palpated at the superior pole the of testis, laterally.
- When the scrotal skin is stretched, a pathognomonic “blue dot sign” (gangrenous appendix) can sometimes be visualized.
- May see reactive hydrocele
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- Management
  - When the diagnosis is certain, analgesia and bed rest, as most appendages will auto-amputate over 1 to 2 weeks
  - Surgery for unremitting pain or if testicular torsion cannot be excluded
- Hair Tourniquet Syndrome
  - Acute strangulation of the glans penis in a young child by a hair or thread
  - May present as acute swelling, erythema, or pain in the penis
  - Nonverbal children may present as inconsolable or irritable,
  - Physical exam shows a edematous, tender penis with circumferential constriction,
  - The hair may not be visible.
  - Treat by cutting the hair or applying a hair removal agent,
  - Urgent urologic consultation is warranted if treatment fails

17.6 NEPHRITIS: HEMOLYTIC UREMIC SYNDROME (see Chapter 5, Pediatrics)

17.7 STRUCTURAL DISORDERS

Nephrolithiasis
- Epidemiology
  - Generally occurs in adults ages 20 to 50; male:female ratio, 3:1
  - Stone composition
    - Most common: calcium oxalate or mixed calcium oxalate and phosphate
    - Less common
      - Struvite = magnesium-ammonium phosphate: associated with chronic infections with urea-splitting bacteria, e.g., *Proteus/Klebsiella/Pseudomonas*, and alkaline pH
      - Uric acid - may follow institution of uricosuric drugs for gout
  - About 90% of stones smaller than 5 mm pass spontaneously,
  - Stones 5 mm or greater are unlikely to pass spontaneously.
  - About 40% of patients with a symptomatic stone will never have a second episode.
- Pathogenesis
  - Urine gets supersaturated with a particular mineral in the context of
    - Decreased urine volume from poor oral intake or excess extra-urinary fluid losses
    - Excess excretion of a mineral in urine, as in
      - Hypercalciuria, which occurs with large dietary calcium load or secondary to hypercalcemia, which can be caused by hyperparathyroidism or sarcoidosis
      - Hyperoxaluria, which occurs with large dietary load (certain summer vegetables) or in association with diseases of the small bowel (Crohn’s)
  - Some stone formers have reduced urinary concentration of natural inhibitors of stone formation,
  - Stones are formed in the renal collecting system and pass into the ureter.
- Clinical Features
  - Classic presentation: abrupt onset of flank pain, which radiates to abdomen, groin, testicle, or labia
  - Location of pain correlates with stone location,
  - Nausea and vomiting are common.
  - Physical exam: patient is typically in agony, cannot find a comfortable position
    - Fever is atypical and should raise suspicion for proximal infection or another diagnosis.
- Abdomen is typically nontender; bowel sounds may be diminished and distension noted, as ileus accompanies some renal colic.
- Important to listen for bruits and palpate for pulsatile masses to consider the possibility of an abdominal aortic aneurysm (AAA).
- Costovertebral angle tenderness is inconsistent,

**Urinalysis**
- Expect to see hematuria; 15% of patients with urolithiasis have no hematuria (complete unilateral ureteral obstruction).
- WBCs and bacteria raise the question of infection, either complicating the renal colic or as the primary diagnosis.
- pH >7.6 raises suspicion for struvite stone/urea-splitting organisms,

**Blood tests in the ED**
- Electrolytes - usually normal, but very low K and bicarb suggest renal tubular acidosis (type 1), which predisposes to stones
- BUN/Cr - should be normal in uncomplicated renal colic
- Calcium - easy to obtain; quick screen for systemic illnesses associated with stone formation (e.g., hyperparathyroidism)

- **Diagnosis:** vascular catastrophe: leaking AAA or iliac aneurysm - you must always rule this out!
- **Complications**
  - **Obstruction** - Complete ureteral obstruction can begin to cause irreversible loss of kidney function after 5 to 14 days.
  - **Infection** - Infection proximal to a suspected ureter blocked by a stone puts patient at risk for septic shock and requires emergent urologic consultation for prompt drainage of the system.

**Imaging Procedures**
- **Plain films:** role in ED diagnosis is controversial
  - Advantages: easy to obtain, 90% of stones are radiopaque, identification of a large stone prompts ED urologic consultation (since it is more likely to require instrumentation), may give information about alternative diagnosis (e.g., bowel obstruction, free air)
  - Disadvantages: calcifications on KUB are only presumptively stones (cannot be sure they are in urinary tract), results may not affect ED management when high suspicion already exists
- **Intravenous pyelogram (IVP):** used less now due to availability of CT
  - Useful to diagnose urinary calculi, estimate size, evaluate renal function, and identify extravasation of dye (rare event from stones)
  - Without a bowel prep, small stones can be missed on IVP.
  - Contraindicated in patients with known radiocontrast allergy or renal insufficiency
- **Ultrasound**
  - Great for assessing renal size and obstruction of collecting system
  - Very good for identifying stone in renal pelvis, but misses a fair number of nonobstructing ureteral stones
  - May gain useful information about alternative diagnoses
- **CT scan**
  - Provides better anatomic definition than US; can help with alternative diagnosis
  - No contrast needed in spiral CT; great for patients with contrast allergy
  - Spiral CT takes only a few minutes to perform.
  - No information on kidney function
o Choice and timing of imaging study depend on
  ■ Clinical certainty about diagnosis
    D For a young adult in whom one is not concerned about life-threatening diagnoses, an outpatient study is acceptable.
    D Alternatively, when the presentation is atypical (e.g., no hematuria, or some abdominal tenderness), an imaging study during the ED visit is essential,
  ■ Concern about more serious diagnosis: patients with risk factors for AAA, who present with renal colic
  ■ Concern about obstruction with infection: fever, toxicity, or pyuria/bacteriuria

• Management
  o Analgesia with a non-steroidal anti-inflammatory drug, an opioid, or both
  o Parenteral ketorolac may be required because of vomiting,
  o NSAIDs are first-line treatment, but opioids are often needed as a supplement.
  o Hydration with IV fluids to restore any volume depletion; benefits of acute increases in urine output are controversial
  o Antiemetics for nausea and vomiting
  o α-Blocking agents such as tamsulosin can be used to relax the ureteral musculature and facilitate stone passage.

• Indications for Admission
  o Absolute
    ■ Intractable nausea and vomiting
    ■ Intractable pain
    ■ Obstruction with infection
    ■ Extravasation of IVP or CT contrast dye (suggests obstruction that caused rupture into perinephric tissue)
  o Relative
    ■ Stone 5 mm or larger
    ■ High-grade obstruction
    ■ Solitary kidney
    ■ Deteriorating renal function

**Polycystic Kidney Disease**
• One of the most common inherited renal disorders
• The most frequent genetic cause of renal failure
• Accounts for 10% of patients on dialysis
• Clinical symptoms include pain, nocturia, hypertension, hematuria, palpable masses in the flanks, and symptoms related to poor renal function.
• Renal ultrasound is the most useful imaging study.
• Treatment is directed to control hypertension, urinary tract infections, and renal failure. Surgical decompression of large cysts and infected cysts is effective for pain relief in over 60% of patients.
• People with polycystic kidney disease (PKD) are at increased risk for cerebral aneurysm: 4% to 10% in the PKD population; may rise to 18% to 26% if family history is positive for it.
Acute Urinary Retention

- Acute inability to pass urine
- Causes
  o Most common: prostatic hypertrophy in an elderly man
  o Foreign body constriction
  o Phimosis and paraphimosis
  o Tumor
  o Stricture
  o Calculus
  o Spinal cord syndrome
  o Early manifestation of multiple sclerosis or tabes dorsalis
  o Medication (anticholinergics and tricyclic antidepressants)
- Clinical Features
  o Urinary retention symptoms: hesitancy, straining to void, nocturia, dribbling stream
  o History of catheterization, prostate surgery, stricture dilation, or genitourinary carcinoma
  o If infection is also present, patient will have dysuria and increased frequency.
- Management
  o Check BUN and creatinine to assess renal function.
  o Urinalysis should be obtained; hematuria suggests a calculi, tumor, or infection,
  o Imaging studies such as CT should be reserved for patients with systemic toxicity or infection,
  o Attempt to relieve urinary retention by passing a urinary catheter,
  o A coude catheter should be used if a standard catheter does not work,
  o Use lidocaine jelly to anesthetize and lubricate during the procedure,
  o If a coude catheter fails, then urologic consultation should be sought.
  o If urgent decompression is needed and consultant is unavailable, then attempt suprapubic percutaneous bladder aspiration.
  o Gradual decompression is recommended, since patients can have hypotension as a complication after drainage.
  o All patients with acute urinary retention require urologic consultation or immediate referral,
  o Patients may be discharged with an indwelling catheter.

17.8 THE RENAL TRANSPLANT PATIENT

Evaluation of a Renal Transplant Patient in the ED

- The most common complaint is fever.
- Temperature may be masked by steroids, uremia, or hyperglycemia.
- Risk of infection in a transplant patient is related to the state of immunosuppression and the time since transplant surgery.
- Rejection is less likely beyond 6 months after transplant.
- Infection in a transplant patient may be a new infection or reactivation of an old infection.

Transplant Rejection

- Transplant failure is one of the most common causes of ARF.
- Rejection is related to T-cell activation, which in turn stimulates antibody production against the transplanted kidney.
**Classification**

- Hyperacute rejection occurs immediately in the operating room secondary to unrecognized ABO incompatibility or HLA mismatch.
- Acute rejection occurs within 3 months after transplantation. Incidence is 30% in recipients of cadaveric kidney, 27% in those who received live donor kidney.

**Signs of Acute Rejection**

- Decreased urine output
- Elevated BP
- Rise in creatinine level
- Mild leukocytosis
- Fever, graft swelling, pain, and tenderness may be seen in a severe rejection episode.

**Chronic rejection** occurs 1 year after transplantation due to immunologic and nonimmunologic factors (hypertension, medication toxicity); clinically evidenced by progressive loss of renal function.

**Infection in Transplant Patients**

**General**

- Most common cause of morbidity and mortality for transplant patients during the first year
- Most common infections are mucocutaneous, urinary tract, and respiratory.
- During the first month after transplant, patients are at risk for the infections most commonly acquired after any surgery: pneumonia, UTI, wound infection, and line infection
  - Fever during this time should raise clinical suspicion for perinephric abscess or local surgical infection,
- During the second through the sixth month, transplant recipients have an increased risk for opportunistic infections, owing to immunosuppression.

**Viral Infections**

- Cytomegalovirus
  - Accounts for two thirds of febrile episodes in the first 6 months
  - Transmission occurs from the donor.
  - Present with fever, myalgia, arthagia, and lymphadenopathy
  - Virus is detected by polymerase chain reaction (PCR)
  - Treat patients with ganciclovir,
- Epstein-Barr virus
  - May present as mononucleosis syndrome or as posttransplant lymphoproliferative disease (PTLD)
  - PTLD is an activation of B cells with expansion in lymphoid tissue
  - Presents as fever, hepatotoxicity, pulmonary infiltrates, and GI bleeding
  - Treatment of PTLD often requires excision, radiation, antiviral, and chemotherapy,
- Varicella (see Image #76)
  - Reactivation increases due to immunosuppression.
  - Some patients have not been exposed to the virus but have primary infection, which may progress to encephalitis.
  - Treat with acyclovir.
• Bacterial Infections
  o Urinary tract infection
    ■ Responsible for 60% of cases of gram-negative bacteremia in transplant patients
    ■ Similar organisms to nontransplant UTI
    ■ Gram-positive organisms are present in the postoperative period.
    ■ *Pneumocystis, Listeria, and Nocardia* infections can occur due to immunosuppression.
    ■ Often progress to urosepsis and pyelonephritis
    ■ Treat with fluoroquinolones initially for 10 to 14 days.
    ■ Patients should be on long-term prophylaxis.

17.9 TESTICULAR MALIGNANCY

General
• Most common malignancy in young men (average age, 32)
• Most are seminomas.
• Spread by lymphatic system

Clinical Features
• Present as an asymptomatic mass, firmness, or induration
• If there is acute hemorrhage into the area, the patient may have acute pain.
• Acute hemorrhage causes pain by expanding on the nonpliable tunica albuginea.
• Metastatic disease should be suspected with unexplained supraclavicular lymphadenopathy, abdominal mass, or acute nonproductive cough from metastasis.

Management
• Doppler ultrasonography is often the diagnostic modality of choice.
• Chest film or CT may be useful if metastasis is suspected.
• As with all malignancy, there is also increased concern for pulmonary embolism in patients with chest pain or dyspnea.
CHAPTER 18
Cutaneous Disorders

Glenn K Geeting, MD

18.1 CANCERS OF THE SKIN

Basal Cell Carcinoma
- Can be nodular, ulcerating, sclerosing, superficial, or pigmented
- Commonly seen in midface and behind ears
- No acute treatment necessary; refer to dermatologist or primary care physician for evaluation

Kaposi Sarcoma
- Painless, raised brown-black or purple papules and nodules that do not blanch
- Face, chest, genitals, and mouth are common locations
- Diagnostic for HIV/AIDS; second most common manifestation of AIDS
- No acute treatment necessary

Melanoma
- Lesion characteristics suspicious for melanoma:
  - Asymmetry
  - Irregular border
  - Multiple colors
  - Diameter greater than 5 mm
  - Evolution (i.e., change) or elevation (raised lesion)
- Early detection leads to improved cure rates; refer if suspicious

Squamous Cell Carcinoma
- More common in men, patients older than 55, and light-skinned individuals
- 90% cure rate if treated early
- No acute treatment is necessary; refer to dermatologist or primary care physician for evaluation

18.2 DECUBITUS ULCER
- External compression of dermis and hypodermis, leading to ischemic tissue damage and necrosis
- Risk factors: inadequate nursing care, diminished sensation/immobility, hypotension, fecal or urinary incontinence, presence of fracture, hypoalbuminemia, poor nutritional status
  - Stage I: superficial, redness that does not subside after pressure is relieved
  - Stage II: damage to the epidermis, no deeper than the dermis
CHAPTER 18 • Cutaneous Disorders

- Stage III: deep necrosis, crateriform ulceration with full-thickness skin loss; damage or necrosis extends to, but not through, fascia
- Stage IV: full-thickness ulceration with extensive damage to muscle, bone, or supporting structures
- Management
  - Stage I and II: topical antibiotics under moist sterile gauze, address contributing risk factors
  - Stage III and IV: surgical
- Complications: osteomyelitis, sepsis with resistant organisms (vancomycin-resistant enterococci [VRE], meticillin-resistant Staphylococcus aureus [MRSA])

18.3 DERMATITIS

Atopic Dermatitis (Eczema)
- Pruritus leads to rubbing and scratching, causing lichenification (hyperplasia)
- Diagnosis: based on clinical findings of thickened inflammatory papular lesions
  - In children and adults, commonly in the antecubital and popliteal flexion areas
  - In infants, common on the cheeks, extensor surfaces, and diaper area
- Associated with allergic rhinitis, asthma
- Treatment focuses on control of inflammation, dryness, and pruritus
  - Topical corticosteroid ointments are primary therapy, but can lead to atrophy, tachyphylaxis, or hypertrichosis
    - Avoid fluorinated corticosteroids on the face
    - Lubricate with petroleum jelly or Eucerin cream to prevent dryness
    - Antihistamines reduce pruritus

Contact Dermatitis (see Image #77)
- Generic term applied to acute or chronic inflammatory reactions to substances that come in contact with the skin
- Allergic skin reaction causes an antigen that elicits a type IV (cell-mediated or delayed) hypersensitivity reaction
- Common allergens include clothing, jewelry (nickel), soaps, cosmetics, hair treatments, plants (especially Rhus [poison ivy and poison oak]), medications, rubber
- Prevention: wear protective clothing; if contact occurs, wash with water or weak neutralizing solution; use barrier creams
- Topical treatment: gauze soaked in Burows’s solution, change every 2 to 3 hours
- Class I topical corticosteroid preparations may be effective if nonexudative, nonbullous
- Oral or parenteral antihistamines
- Systemic corticosteroids if severe (cannot function or sleep) for exudative lesions; long taper of at least 10 to 14 days, 21 days for poison ivy

Psoriasis
- Bilateral, often symmetric; frequently spares exposed areas; favors elbows, knees, scalp, and intertriginous areas; face involvement is uncommon (see Image #78)
- Guttate psoriasis: disseminated small lesions without predilection of site
- Treatment
  - Soak in water and remove scales
  - Apply topical fluorinated glucocorticoid ointment to wet skin (betamethasone valerate, fluocinolone acetonide, betamethasone propionate)
  - Cover with occlusive dressing and leave in place overnight
  - Long-term treatment by dermatologist or primary care physician

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Sebaceous Cyst (wen, infundibular cyst, epidermal cyst)
- Most common cutaneous cyst; origin is epidermis or hair follicle epithelium: cystic enclosure fills with keratin and lipid-rich debris; infection and rupture are common
- Young to middle-aged adults
- Treatment: drainage by incision, usually with packing

Seborrheic Dermatitis
- “Cradle cap” in infants
- Yellowish-red or gray-white skin, often with “greasy” or white dry scaling macules and papules of varying size (5-20 mm) (see Image #75)
- Treat with frequent shampooing with shampoos containing selenium, salicylic acid, tar, or sulfur; topical antifungals (ketoconazole) or corticosteroids (fluocinolone) also useful

18.4 INFECTIONS

Bacteria
- Abscess: *Staphylococcus aureus* (increasingly antibiotic resistant)
  - Treatment: incision and drainage; antibiotic only if associated cellulitis
- Hidradenitis Suppurativa: apocrine sweat glands of axilla in women, groin in men; can lead to chronic draining fistulae and cicatrical formations
  - Consider early surgical referral for obliteration of the involved area
- Cellulitis and Erysipelas
  - Acute, spreading infections of dermal and subcutaneous tissues
  - Signs include red, hot, tender skin, often at site of bacterial entry; erysipelas is generally well demarcated
  - Most common organisms: group A P-hemolytic streptococci or *S. aureus*
  - Outpatient management: cephalaxin, dicloxacillin, macrolides, amoxicillin-clavulanate
    - For suspected MRSA, consider trimethoprim/sulfamethoxazole ± rifampin, clindamycin, or doxycycline
  - Consider admission for immunocompromise, systemic symptoms, extremes of age, edema, diabetes, or failure of outpatient therapy
  - For facial cellulitis in an ill, unimmunized child, consider *Haemophilus influenza*
- Impetigo and Ecthyma
  - Transient superficial small vesicles or pustules that rupture; may leave erosions or golden-yellow crust (often seen, not pathognomonic) (see Image #74)
  - Treatment: topical mupirocin is highly effective against group A streptococci and *S. aureus* (including MRSA)
  - Systemic antimicrobials: penicillin, dicloxacillin, erythromycin; consider doxycycline/minocycline, clindamycin, or trimethoprim/sulfamethoxazole for suspected MRSA
  - Poststreptococcal glomerulonephritis is a possible complication.
- Necrotizing Infections
  - Type 1 is polymicrobial and involves non-group A streptococci plus anaerobes, typically found on the abdomen and perineum
  - Type 2, group A streptococcal gangrene that involves the extremities
    - Numbness or deep pain out of proportion to the examination
    - Cellulitis that turns dusky blue; bullae or vesicles appear, filled with yellow or red-black fluid; rapidly spreads along fascial planes, leading to necrotic slough, “dish water” appearance
    - Systemic symptoms are common: fever, hypotension, tachycardia
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- Radiographs reveal gas in the tissues in about half of cases (see Image #5); MRI is probably the best diagnostic test and helps delineate the extent of disease
- Fournier’s gangrene: polymicrobial, rapidly spreading soft-tissue infection of the perineum, associated with diabetes (see Image #2)
- Ecthyma gangrenosum
  - *Pseudomonas aeruginosa* infection producing tense, white, shiny vesicles with erythematous halos; becomes hemorrhagic and then ulcerative with necrotic centers
  - Commonly occurs on the buttocks and extremities
  - Associated with immune compromise and/or neutropenia
- Treatment includes resuscitation, early extensive surgical debridement, high-dose broad-spectrum antibiotics

**Fungus**

- **Candidiasis (moniliasis):** *Candida albicans*, others
  - Diagnosis: potassium hydroxide (KOH) preparation shows hyphae and pseudohyphae
  - Intertrigo: in moist skin folds, under breasts, in groin, between fingers and toes, typically with satellite lesions
    - Treatment: keep dry; topical zinc oxide or clotrimazole
  - Diaper dermatitis
    - Treatment: frequent diaper changes, keep dry, topical zinc oxide or antifungal cream
  - Thrush: white pharyngeal exudates with surrounding erythema
    - Common in infants and the immunocompromised (diabetics, people with HIV/AIDS, corticosteroid users)
    - Treatment: nystatin, 100,000 units; swish and swallow QID
  - Consider oral therapy for patients that don’t respond to topical treatments: fluconazole, itraconazole, ketoconazole

- **Tinea Infections**
  - Diagnosis: potassium hydroxide (KOH), microscopic identification of septated, tubelike structures and spores
  - Tinea pedis (feet), manus (hands), cruris (groin)
    - Treatment: keep dry; topical imidazoles (clotrimazole, miconazole, ketoconazole, econazole) or terbinafine; consider oral itraconazole for severe or refractory disease
  - Tinea capitis (scalp)
    - Kerion: indurated, boggy, inflammatory plaque of scalp with pustules
    - Treatment: oral griseofulvin for 8 weeks (possible transaminase elevation, disulfiram reaction); nizoral shampoo reduces contagiousness
  - Onychomycosis (nails)
    - Treatment: oral itraconazole, fluconazole, or terbinafine; topical agents are ineffective

**Parasites**

- **Pediculosis: head lice, body lice, pubic lice**
  - Pruritic
  - Nits are white dots attached to the bases of hair shafts,
  - Treatment
    - Wash and dry hair, then apply permethrin.
    - Lindane 1% shampoo only for treatment failures; can cause seizures, aplastic anemia in children
    - Wash underclothing and sheets in hot water at the time of treatment
    - Treat sexual partners; no need to treat uninfested household members
- Evaluate at 1 week and re-treat if symptomatic or if lice/eggs are found.
- Watch for secondary infections.

- Scabies: mite *Sarcoptes scabiei*
  - Extremely pruritic, worse at night
  - Look especially in interdigital web spaces, flexion areas, and groin, (see Image #16)
  - Almost never affects above the clavicles, except in infants
  - Norwegian scabies creates widespread, severe crusting
  - Diagnosis: presence of burrows or scabies prep (shave lesion, use magnification)
  - Treatment
    - Permethrin 5% applied overnight and repeated in a week
    - Wash underwear and bed sheets in hot water.
    - Treat family and close personal contacts.
    - Pruritis may persist for several weeks despite successful eradication.
    - Watch for secondary infections.

**Virus**

- Aphthous Ulcer (“canker sore”): painful shallow ulcer covered with fibrin; adenopathy is common
  - Differential: Beh’ets disease, herpes
  - Treatment: symptomatic; some relief from topical steroids and analgesics

- Erythema Infectiosum (see Chapter 5, Pediatrics)
- Herpes Simplex (see Chapter 8, Systemic Infectious Disorders)
- Herpes Zoster (see Chapter 8, Systemic Infectious Disorders)

- Human Papillomavirus
  - Verruca vulgaris: common warts; verruca plantaris: plantar warts; verruca plana: flat warts; epidermodysplasia verruciformis
  - Treatment usually conservative, since spontaneous remission is the rule
  - Aggressive treatment if symptomatic (e.g., plantar, genital): podofilox 0.5% gel or solution applied BID x 3 days, then no therapy x 4 days, then repeat cycle up to 4 times; cryotherapy by primary care physician

- Measles
  - Koplik’s spots: small, irregular, bright red spots with bluish white centers on buccal mucosa
  - Maculopapular erythematous lesions spread from forehead and neck downward
  - Encephalitis in 0.1%, carries a 15% mortality

- Molluscum Contagiosum
  - Poxvirus causing umbilicated papules (see Image #25)
  - Normal host: cosmetic problem, spontaneous remission
  - Disseminated disease common in HIV patients

- Varicella: chickenpox (see Chapter 5, Pediatrics, and Chapter 8, Systemic Infectious Disorders)

**18.5 MACULOPAPULAR LESIONS**

**Erythema Multiforme**

- More than half are idiopathic.
  - Implicated drugs: sulfonamides, phenytoin, barbiturates, phenylbutazone, penicillin, allopurinol
  - Infection: herpes simplex virus (HSV), hepatitis, influenza A, fungus, bacteria

- Accompanied by malaise, fever, myalgias, arthralgias; pruritus or burning can occur before skin lesions develop
**Chapter 18 • Cutaneous Disorders**

- "Multiforme" lesions are most common (symmetric erythematous macules, papules, vesicles, or bullae), "target" most familiar, commonly involving the palms and soles (see Image #49)
- Systemic steroids give symptomatic relief but are unproven in changing duration or outcome.
- If extensive disease, toxic patient, and/or mucous membranes involved, hospitalize patient, optimally in an ICU or a burn unit.

**Erythema Nodosum**
- Inflammatory/immunologic reaction causing painful nodules, usually on the anterior shins (see Image #38)
- Multiple and diverse etiologies, often idiopathic
  - Infectious: tuberculosis (rare, generally seen in childhood), coccidiomycosis, histoplasmosis, P-hemolytic streptococci, *Yersinia*, lymphogranuloma venerium (LGV)
  - Drugs: sulfonamides, oral contraceptives
  - Miscellaneous: sarcoid (common), ulcerative colitis, Behcet’s disease, idiopathic (40%)
- If not known, symptomatic relief: bed rest, leg elevation, and NSAIDs
- Usually resolves in 3 to 8 weeks

**Henoch-Schönlein Purpura (see Chapter 5, Pediatrics)**

**Purpura Fulminans**
- Fever, shock, multiorgan failure, and rapidly developing hemorrhagic skin necrosis
- Dermal vascular thrombosis resulting from vascular collapse and disseminated intravascular coagulation (DIC)

**Urticaria and Angioedema**
- Urticaria = wheals = hives: transient, blanching, edematous papules and plaques, usually pruritic
  - Cutaneous IgE-mediated reaction to an allergen
  - Treatment
    - **Remove the offending agent**
    - Mild/moderate: antihistamines ± steroids
    - Severe (anaphylaxis): add subcutaneous, intramuscular, or intravenous epinephrine
    - Consider an H2 receptor blocker if severe or refractory
    - Refer severe, recurrent, or refractory cases to an allergy specialist
- Angioedema: edematous area involving dermis and subcutaneous tissue (see Image #67)
  - Angioedema of tongue, lips, and face in 0.1% to 0.2% of patients taking ACE inhibitors can occur years after initiation of therapy
  - Treatment: supportive, with special attention to the airway; usual anti-allergy medications (as above for urticaria) are not proven to be beneficial but are often used; remove causative agent if known

**Pityriasis Rosea**
- Multiple pink or pigmented oval papules or plaques 1 to 2 cm in diameter on the trunk and proximal extremities, which parallel the ribs in a “Christmas tree pattern” (see Image #51)
- Look for a herald patch that preceded the diffuse rash by a week.
- Self-limited, resolves in 3 to 8 weeks
18.6 VESICULAR/BULLOUS LESIONS (TABLE 18-1)

- Nikolsky’s Sign: epidermis dislodged by gentle finger stroke near blister or pressure on blister causes lateral extension of subdermal fluid; seen in pemphigus vulgaris, staphylococcal scalded skin syndrome, erythema multiforme major, and toxic epidermal necrolysis (TEN)

**Bullous Pemphigoid**
- Average age 65; chronic course lasting 1.5 to 5 years
- Less severe disease than pemphigus vulgaris
- Tense blisters up to 10 cm lead to ulceration
- Intertriginous and flexural areas, fewer oral lesions than pemphigus vulgaris
- Commonly treated with anti-inflammatory agents (corticosteroids, tetracyclines, dapsone) and immunosuppressants (azathioprine, methotrexate, mycophenolate mofetil, cyclophosphamide)

**Pemphigus Vulgaris**
- Autoimmune, often fatal if untreated; 40 to 60 years old
- Large vesicles or bullae of the skin and mucous membranes rupture after several days, leaving painful, denuded areas
  - Heals slowly, prone to infection
- Treatment: high-dose glucocorticoids, other immunosuppression: mycophenolate mofetil, azathioprine, cyclophosphamide, plasmapheresis, methotrexate
- Consider admission to a burn unit or an ICU in severe disease; consult dermatology

**Staphylococcal Scalded Skin Syndrome** (see Chapter 5, Pediatrics)

**Erythema Multiforme Major**
- Younger patients, typically 10 to 30 years old
- Erythema multiforme with bullae, mucosal lesions, and multisystem involvement (GI, respiratory, GU, renal)
- Bullae form from typical EM target lesions; distributed peripherally
- Less than 10% of total body surface area
- Caused by drugs (sulfonamides, allopurinol, carbamazepine, hydantoins, aminopenicillins), HSV, hepatitis viruses, influenza A, fungi (rare), strep (rare), collagen-vascular disorders (rheumatoid arthritis, systemic lupus erythematosus [SLE], dermatomyositis, polyarteritis nodosa)
- 50% idiopathic
- Patients may appear toxic
- Death from dehydration or infection (0-15%)
- Treatment: rehydration, steroids

**Toxic Epidermal Necrolysis (TEN, Stevens-Johnson Syndrome)**
- Severe variant of erythema multiforme
- -80% of cases are drug related: sulfa drugs, allopurinol, aspirin, carbamazepine, hydantoins, NSAIDs
  - Has also been associated with vaccination and lymphoma
- Skin is tender and red with extensive cutaneous and mucosal exfoliation (see Image #46)
- Prodrome: fever, flu-like symptoms 1 to 3 days prior to emergence of mucocutaneous lesions
- Eye involvement: conjunctival burning or itching may progress to permanent injury; involve ophthalmology
• Treatment is supportive.
  o High mortality (30%-40%): requires aggressive hydration and prevention of infection in an ICU or a burn unit

Table 18-1. Characteristics of Vesicular/Bullous Lesions

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Age</th>
<th>Defining Signs</th>
<th>Mucous Membrane Involvement</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bullous pemphigoid</td>
<td>Elderly (average 65)</td>
<td>Chronic course, large bullae of intertriginous and flexor surfaces</td>
<td>In 10%-25%</td>
<td>Low, complicated by comorbidities</td>
</tr>
<tr>
<td>Pemphigus vulgaris</td>
<td>40-60 years old</td>
<td>Diffuse tense bullae, systemic symptoms</td>
<td>In 50%-70%</td>
<td>5%-15%, generally fatal if untreated</td>
</tr>
<tr>
<td>Staphylococcal scalded skin syndrome</td>
<td>Infants and children</td>
<td>Erythroderma followed by exfoliation, then bullae leaving scalded-appearing skin; heals in 7-10 days</td>
<td>No</td>
<td>Low</td>
</tr>
<tr>
<td>Erythema multiforme major</td>
<td>10-30 years old</td>
<td>Target lesions progress to bullae; limited mucous membrane involvement; &lt;10% of TBSA</td>
<td>Limited</td>
<td>Low</td>
</tr>
<tr>
<td>Toxic epidermal necrolysis</td>
<td>Adults</td>
<td>Widespread bullae, systemic symptoms, mucous membrane involvement</td>
<td>Severe</td>
<td>30%-40%</td>
</tr>
</tbody>
</table>

18.7 PETECHIAL LESIONS
• Red or purple spots that do not blanch, because of small subcutaneous hemorrhages (see Image #36)
• May indicate severe illness

Meningococcal Sepsis (see Images #35a and b)
• Neisseria meningitidis, gram-negative diplococcus
• Can progress rapidly to death
• Overall mortality rate is 10%; 20% to 60% in septicemia without meningitis
• Fever, irritability, headache, vomiting, meningeal irritation
• 60% have the classic petechial rash that appears first on ankles, wrists, axillae, and pressure points; typically spares palms, soles, and head

Disseminated Gonorrhea
• Fever and migratory polyarthralgia commonly accompany skin lesions.
• Characteristic rash: involves periarticular areas of the distal extremities
  o Erythematous or hemorrhagic papules evolve into pustules and vesicles with an erythematous halo
  o Can resemble the rash of meningococcemia
• Cultures of the lesions are typically negative
• Treatment: third-generation cephalosporin, spectinomycin, or quinolone
• Hospitalization is recommended for patients with an uncertain diagnosis, septic arthritis, meningitis, or endocarditis.
Rocky Mountain Spotted Fever (RMSF)
- Tick-borne systemic infection of *Rickettsia rickettsii*
  - Only 60% to 70% of patients with RMSF give a history of a tick bite.
- Found throughout the Americas, beyond the Rocky Mountain states
- Typically presents with abrupt onset of headache, vomiting, high fever, and malaise
- The classic rash starts about 3 days after the fever, (see Image #42)
  - Erythematous macules that blanch with pressure, starting on the wrists and ankles and spreading to the trunk and face over hours and progressing to petechial or hemorrhagic lesions
  - Lesions of the palms and soles are particularly characteristic,
  - Rocky Mountain spotless fever: rash is absent in 4% to 16% of patients
- Treatment with doxycycline or chloramphenical should begin immediately, before confirmatory testing

Other Bacterial Causes of Petechiae
- Endocarditis (see Image #44), toxic shock syndrome, scarlet fever

Viral Causes of Petechiae
- Hemorrhagic fevers, including dengue and Ebola

Noninfectious Petechiae
- Forceful vomiting or coughing can cause facial petechiae.
- Thrombocytopenia: idiopathic thrombocytopenic purpura (ITP), thrombotic thrombocytopenic purpura (TTP), leukemia
- Vasculitis: polyarteritis nodosa, Henoch-Schoenlein purpura, Kawasaki disease, drug reactions
CHAPTER 19
Prehospital and Emergency Medical Services
David E. Manthey, MD, and Henderson McGinnis, MD

19.1 ADMINISTRATION, MANAGEMENT, AND OPERATIONS

Goals of Emergency Medical Services (EMS)
- Provide medically trained personnel to patients as quickly as possible
  - First responders trained in automatic external defibrillation (AED) to provide immediate treatment of cardiac arrest
  - Best outcome if cardiopulmonary resuscitation (CPR) begins within 4 minutes and Advanced Cardiac Life Support (ACLS) within 8 minutes after arrest
- Prevent additional injury
- Rapid transport
- Advance notification

EMS System Designs
- Fire Service - EMS operate as part of fire department
- Third Service - emergency medical response is a freestanding public service (separate from fire/police)
- Public Utility - private company contracts to provide EMS
- Volunteer - common in smaller communities that cannot afford paid personnel
- Combined Public/Private - first response, public; transport, private

Planning (EMS System Demands)
- One ambulance response per day for every 10,000 people in area served
  - Increase if the population is elderly, technology dependent, or medically underserved
- System status management
  - Peak load staffing - staff when most EMS calls come in

Prevention
- Safety and injury prevention
- Vehicle safety and protective equipment
- Poison prevention

Manpower Statistics
- 75% of emergency medical technicians (EMTs) are volunteers (highest in rural areas)
- When paid, salaries account for 50% of budget
Communications

- The 911 Access System
  - Universal number allows uniform activation of police/fire/EMS
  - 93% of population covered
  - Enhanced 911 (E-911) system displays address and phone number of caller, facilitating response; 95% of covered population
- Dispatch
  - Dispatchers receive emergency calls and send most appropriate resources,
  - Priority dispatch
    - Templates guide dispatcher questions, aid in assigning EMS resources, and enable dispatchers to give pre-arrival instructions to callers.
  - Computer-aided dispatch (CAD)
    - Computers are used to assign calls, assign EMS resources, and make unit notifications.
- Notification
  - Communications with hospital
  - Information about patient and expected resources needed
  - On-line medical direction

Coordinated EMS System

- Will match anticipated patient needs with the most appropriate receiving hospital:
  - Trauma centers (designations vary by state)
    - Level I - full capabilities (ED), all specialty services, emergency surgery 24 hours
    - Level II - full capabilities (ED), some specialty services, emergency surgery 24 hours
    - Level III - full capabilities (ED), without specialty services, emergency surgery on call backup
    - Level IV - minimal capabilities
  - Cardiac centers - personnel and facilities to provide emergent cardiac catheterization
  - Stroke centers - personnel and facilities to provide acute stroke management and rehabilitation
  - Burn centers
  - Psychiatric centers
  - Toxicology centers

Prehospital Triage

- Determining if patient needs a specialized center (e.g., a trauma center)
  - Over-triage (taking a low-risk patient to a trauma center)
    - Increases trauma center volume and dilutes resources
  - Under-triage (taking a seriously ill patient to a non-trauma center)
    - Patient is without the resources and expertise of a trauma center
- Scoring systems
  - Anatomic - based on site of injury or organ injured
  - Physiologic - based on vital signs and physiologic patterns
  - Mechanism of injury - based on amount, type, and delivery of force
  - Combined
  - Prehospital medical care providers “injury severity perception” is as good as scoring systems
19.2 CREDENTIALING OF PREHOSPITAL CARE PROVIDERS

Tides and Requirements
- Vary from state to state
- Based on training curriculum developed by DOT

Responder Levels and Capabilities
- First Responder
  - Training: 40-60 hours
  - Capabilities: First aid, CPR, spinal immobilization
- Emergency Medical Technician (EMT) - Basic
  - Training: -100 hours
  - Capabilities: first aid, CPR, immobilization, extrication, ambulance operations, insertion of Combitube, use of automatic external defibrillator, assist patients in self-administration of medications
- EMT - Intermediate (various names are used)
  - Training: EMT Basic + 30 additional hours + adequate clinical hours
  - Capabilities: patient assessment, IV access, defibrillation, basic ECG interpretation
- EMT - Paramedic
  - Training: EMT Basic + 30 additional hours + 230 clinical hours + field internship
  - Capabilities: ACLS and other medications, ECG interpretations, intubation, defibrillation/cardioversion

Staffing
- Varies from state to state
- Usually two EMT-Bs or an EMT-B with higher level EMT

19.3 DIRECT PATIENT CARE

Prehospital Care Is Directed Toward the Stabilization of Critically 111 and Injured Patients.
- 3% of EMS patients are emergent/critical
- 15% are urgent
- 80% are non-urgent
- 5% to 10% are pediatric cases

Medical Control
- Medical Director
  - The physician medical director of each EMS system has the primary responsibility and authority to provide medical oversight.
  - EMS care providers work under the supervision of a physician. Depending on the state, they may not be under the license of a physician,
  - Liaison between EMS and medical community
- Direct(On-line) Medical Control (Concurrent)
  - Direction given by physician in person at scene or remotely (by radio)
    - Adds an average of 8 minutes on scene
    - Rarely deviates from devised protocols (4%)
    - More common in cardiac (78%) than trauma (35%) cases
o An on-scene (bystander) physician who wishes to direct the care given by EMS personnel must be able to provide proof of identity and licensure.
  ■ If treatment is in line with protocols, physician need not accompany patient to hospital or assume responsibility.
  ■ If on-scene physician accepts medical control from on-line physician, he/she accepts full medical/legal responsibility and must accompany patient to hospital.
• Indirect (Off-line) Medical Control
  o Prospective
    ■ Direction given through protocols/standing orders and ongoing education of providers
  o Retrospective
    ■ Quality assurance/improvement surveillance of ambulance runs
    ■ EMS training when deficiencies are noted

**Infectious Disease Risk**
• Utilize universal precautions with all patients
  o Exposure to blood borne pathogens greatest with trauma victims
• Diseases
  o AIDS
  o Hepatitis B and C
  o Tuberculosis
  o Bacterial meningitis
• Ryan White Act gives EMS personnel the right to obtain blood from source patient after an exposure.

**Aeromedical EMS**
• Selected patients with time-critical illnesses and injuries may be transported.
• Effectiveness is not clearly established, but early studies showed improvement.
• Commission on Accreditation of Medical Transport Systems (CAMTS)
  o Established in 1990
  o Voluntary process
• Rotary Wing (Helicopters)
  o Primary advantages
    ■ Reduced transport time
    ■ Overcome geographic obstacles
    ■ Level of care often higher
    ■ Higher level of on-line medical control
  o Limitations of helicopters
    ■ Limited range
    ■ **Care is difficult due to cramped, loud setting**
    ■ Weather dependent (can operate only under clear conditions)
    ■ Expensive (average charge, $4,500)
      □ Fixed cost is about $1.5 to $2 million per year, with each run adding a minimum $1,000 in cost
    ■ High risk
    ■ In urban settings, there is often little or no time saved
      □ More effective if transport is 20 miles or more
o Medical issues
  ■ Atmospheric pressure drop causes expansion of air-filled spaces.
  • Chest tube for pneumothoraces
  ■ Airway should be secured before transport if borderline
  ■ Combative patients
• Fixed-wing medical transport services have utility for long-range inter-facility transfers,
  o Better safety record
  o Wider range of weather conditions for operation
  o Less expensive for distance flown
  o Increased mileage range
  o Increased crew and equipment capacity
  o Longer time to facility
  o Limited to airports

19.4 MULTI-CASUALTY INCIDENTS (MCIS)
• MCIs frequently (not always) represent “disaster” situations.
• Disaster = needs of the casualties exceed available resources
  o Due to sheer numbers
  o Due to the level of care
  o Due to loss of infrastructure/assets

Mass Gathering
• Gathering of more than 1,000 people in one place for the same reason
• Problems
  o Potential for increased number of injuries
  o Physical barriers to get to patients
  o Climatic issues or terrorist attack
  o Need for HAZMAT and law enforcement

Disaster Classification
• Based on ability of area to meet the needs for response
  o Level I - local medical response adequate
  o Level II - regional/adjacent community support required
  o Level III - state/federal support required (declared by governor or president)

Prehospital Disaster Response
• First Responders
  o Police department
    ■ Scene control and security
    ■ Establish communications
  o Fire department
    ■ Fire control
    ■ Hazardous material control
      □ Inner perimeter, which no EMS personnel should breach for own safety
      ■ Extrication of victims from hazardous positions
o EMS
  ■ Staging of site
  ■ Triage of patients
  ■ Decontamination
  ■ Medical care
  ■ Transport

• Command Post
  o Incident command system
    ■ Scaleable
    ■ Task oriented
  o Uphill, upwind, up water from disaster to prevent contamination
  o Medical command by physician
  o Central control for
    ■ All communications
      ◦ Dual methods for all responding agencies
      ◦ Preplanned modes/frequencies/use
    ■ EMS staging and transport
    ■ Hospital resources
      ◦ Level of care available
      ◦ Availability of beds
    ■ Additional fire, police, EMS support
    ■ Other
      ◦ Waste disposal
      ◦ Water/food

• Triage
  o Definition: classification of patients into treatment priorities
    ■ A fluid, ongoing process
  o Guiding principle: do the greatest good for the greatest number
  o Primary
    ■ Field triage assigning patients to class (usually color coded)

<table>
<thead>
<tr>
<th>Triage Category</th>
<th>Color Code</th>
<th>Treatment Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td>Red</td>
<td>Critical</td>
</tr>
<tr>
<td>Level 2</td>
<td>Yellow</td>
<td>Priority</td>
</tr>
<tr>
<td>Level 3</td>
<td>Green</td>
<td>Delayed (“walking wounded”)</td>
</tr>
<tr>
<td>Level 4</td>
<td>Black</td>
<td>Dead or expectant</td>
</tr>
</tbody>
</table>

■ The only treatment provided is opening airways and controlling hemorrhage.
■ In a disaster setting, resources and time are not given to any patient in cardiac arrest or deemed unsalvageable with currently available resources.
■ Usually done by EMS personnel
  o Secondary/tertiary
    ■ Re-triage occurs at staging area (casualty collection) and at hospital
Hospital Disaster Response

- Joint Commission Requirements
  - Written disaster plan
    - Must integrate with local disaster plan
  - Drills twice a year
  - Incident command system
- Command Center
  - In house
  - Communications with scene command center or community emergency operations center (EOC)
  - Activation of plan
    - Recruitment of in-house personnel to appropriate staging areas
    - System for calling in backup
    - Rapid clearing of treatment areas
- Treatment Areas
  - Receiving
    - Triage
    - Decontamination
  - Critical care area
    - Often the emergency department
    - Level 1 critical patients
    - Rapid stabilization and clearance to OR/ICU or intermediate care area
  - Intermediate area
    - Level 2 priority patients
    - Continued reassessment and re-triage of these patients
  - Delayed treatment
    - Level 3 patients
  - Palliative care/morgue
    - Pain control for unsalvageable patients
- Other
  - Public relations to provide accurate information to family members
  - Waiting areas for family
  - Re-supply/staging areas for EMS/police
- Documentation
  - Master list of all casualties arriving from scene
  - Pre-prepared patient chart sets are preferred
    - Usual registration process cannot handle disaster situation and limits care
      - Each chart is pre-labeled with disaster number.
    - Medical record
      - Limited to critical issues and treatments
      - Stays with patient
    - Expected lab and radiograph request sheets
    - Identification bands
• Security
  o Security of institution
  o Control of press/family members/others
  o Direct traffic
    ■ Ambulances
    ■ Family members

Debriefing
• Review of process for improvement
• Stress management
• Recuperation of equipment and personnel

19.5 PERFORMANCE IMPROVEMENT/PROTOCOL DEVELOPMENT
• A quality assurance or performance improvement system is required of EMS systems.
• EMS medical director is responsible for reviewing medical performance,
  o Based on measurable benchmarks
• Protocol Development
  o EMS medical director is responsible for developing and updating medical protocols to authorize and guide medical care.
  o Some systems rely heavily on on-line medical control, while others rely heavily on standing orders/protocols.
    Many systems fall along that spectrum,
  o Special protocols, equipment, and training are needed for pediatric patients.
    ■ EMS for Children (EMSC) programs have been developed in many areas to address these issues.
    ■ The benefit of intubation by paramedics over bag-valve-mask ventilation in the pediatric population has been questioned.

19.6 OTHER ISSUES

Refusal of Care
• 50% of legal cases against EMS providers are related to failure or refusal to treat or transport
• A capable, lucid patient may refuse care and transport after being informed of possible consequences,
  o Must be of sound mind
  o Must be able to understand the nature of the treatment being offered
  o Must be able to understand alternatives to transport/care
  o Must be able to understand the potential consequences of refusal
• The decision to not transport should be made in conjunction with an on-line physician.
• An incompetent or impaired patient should NOT be allowed to refuse care. The police can be utilized to restrain the individual.
• If there is a question about the patients competence (possible intoxication, for example), EMS providers should err in favor of the patient’s well-being.
  o Act in the patient’s best interest
  o Act within established protocols
  o Appropriately document
Malpractice Risk
- 1 claim per 24,000 EMS calls
- Liability cases continue to rise in number and amount
- 30% to 90% are related to non-transport or delayed transport

Automatic External Defibrillators
- Proven role of defibrillation in improving outcomes after cardiac arrest
- Faster delivery, better outcome
- Technology to analyze dysrhythmias
- Semi-automated, as the push of button is required to discharge electrical shock
- Varies by state as to level of training and skill (CPR, EMT) required
CHAPTER 20

Rapid Board Review

CHAPTER 1

Traumatic Disorders

- Class III shock is the first class in which there is a drop in the systolic blood pressure below 90 mm Hg.
- The hallmark of neurogenic shock is hypotension with a normal heart rate or bradycardia and a lack of vasoconstriction on examination.
- 90% of aortic disruptions caused by blunt trauma occur just distal to the left subclavian artery at the ligamentum arteriosum.
- 80% of diaphragmatic injuries occur on the left side.
- The most commonly injured abdominal organ in stab wounds is the liver. The most commonly injured abdominal organ in blunt trauma is the spleen.
- Industrial sprayers and other high-pressure equipment may cause serious injuries with minimal examination findings.
- The most common traumatic cerebral bleed is subarachnoid hemorrhage.
- The most common cause of coma following head injury is diffuse axonal injury.
- Intubate all trauma patients with a Glasgow Coma Scale (GCS) score <8.
- Uncal herniation—the most common type of herniation—causes compression of the oculomotor nerve and an ipsilateral fixed and dilated pupil.
- Cerebral perfusion pressure = mean arterial pressure - intracerebral pressure
- On CT scan, a subdural hematoma (see Image #41) is crescent shaped and an epidural hematoma (see Image #39) is biconvex (lens or football shaped).
- The classic presentation of an epidural hematoma follows a blow to the temple with loss of consciousness, then a “lucid interval,” followed by rapid progression of coma with increased ICP and lateralizing signs of uncal herniation.
- The classic mechanism producing a Jefferson fracture, or burst fracture of C1, is an axial load. A Hangmans fracture involving the posterior elements of C2 is produced by severe hyperextension.
- Infective organisms specific to various bites are listed below:
  o Human bite - *Eikenella corrodens*
  o Cat bite - *Pasteurella multocida*
  o Dog bite - *Capnocytophaga canimorsus*
CHAPTER 2

Cardiovascular Disorders

- In patients with wide-complex Wolff-Parkinson-White (WPW) syndrome, avoid medications that block the AV node but not the bypass tract, such as digoxin and calcium channel blockers. Procainamide and cardioversion are safe in these patients.
- Suspect right ventricular infarcts in patients with inferior myocardial infarction. Treat them with intravenous fluids and avoid nitroglycerin, because it frequently precipitates hypotension.
- Contraindications to thrombolytic therapy:
  o Absolute contraindications
    - Active internal bleeding or a known bleeding disorder
    - Suspected aortic dissection or pericarditis
    - Uncontrolled hypertension with a systolic blood pressure of 180/110 mm Hg despite therapy
    - History of hemorrhagic CVA
    - History of non-hemorrhagic CVA within the past year
  o Relative contraindications
    - History of chronic severe hypertension or presenting blood pressure > 180/110 mm Hg
    - Active peptic ulcer disease
    - Pregnancy
    - Internal bleeding within the past 4 weeks
    - Noncompressible vascular puncture
    - Trauma surgery or CPR within the past 2 to 4 weeks
    - Use of anticoagulation that is in therapeutic range or known bleeding diathesis
    - History of CVA or known intracerebral pathology
    - For streptokinase/anistreplase: previous exposure or allergic reaction
- Accelerated idioventricular rhythm is an ectopic rhythm of ventricular origin at a rate between 40 and 100 beats/min, which occurs after administration of thrombolytic therapy. It does not require treatment.
- Avoid P-blockers in patients with cocaine-induced chest pain and pheochromocytomas, because of the possibility of unopposed a stimulation and the resultant hypertension.
- The best approach for placement of an emergency transvenous pacer is through the right internal jugular vein. ST elevation noted on the monitor indicates successful pacemaker placement in the apex of the right ventricle.
- The classic chest radiographic findings in a patient with pulmonary embolism include Hamptons hump and Westermarks sign:
  o Hamptons hump is a triangular, pleural-based density with a rounded apex that points in the direction of the hilum.
  o Westermarks sign is dilatation of the pulmonary vasculature proximal to the embolus, associated with regional oligemia (decreased vascular markings) distally.
- The echocardiographic findings of pericardial tamponade include the following:
  o A large pericardial effusion (see Image #32)
  o Diastolic collapse of the right ventricle and the right atrium
  o Swinging motion of the heart in the pericardial effusion
- The electrocardiographic findings in pericardial tamponade include electrical alternans, tachycardia, and low voltage.
- Torsades de pointes is treated by cardioversion in unstable patients and with magnesium or overdrive pacing in stable patients.
• The murmur of hypertrophic cardiomyopathy is a harsh crescendo-decrescendo murmur at the left sternal border. It is increased by maneuvers that decrease left ventricular filling (Valsalva, standing) and decreased by maneuvers that increase left ventricular filling (squatting, hand grip, or Trendelenburg position).
• The most common cause of left-sided endocarditis is *Streptococcus viridans*. The most common cause of right-sided endocarditis is *Staphylococcus aureus* from IV drug abuse.
• Strongly suspect abdominal aortic aneurysm in any man over the age of 50 with hypertension and abdominal/flank pain with or without hematuria.
• Suspect thoracic aortic dissection in patients with chest pain and neurologic findings. Treat them with P-blockers and nitroprusside. Type A dissections (ascending) are managed with surgery; type B dissections (descending) are managed medically unless a major branch vessel is affected.
• Physical exam findings in endocarditis:
  o Janeway lesions - nontender plaques on the palms and soles
  o Osier's node - a tender nodule that appears on the tips of the fingers or toes
  o Roth spot - a retinal hemorrhage with central clearing

CHAPTER 3
Abdominal and Gastrointestinal Disorders
• Abdominal pain associated with atrial fibrillation, severe CHF, cardiomyopathy, digoxin use, or vasopressor use suggests mesenteric ischemia.
• Upper GI procedures are the most common cause of esophageal rupture and are now more common than the classic Boerhaave's syndrome (vigorous retching/vomiting). Suspect Boerhaave's syndrome in the alcoholic with vomiting and chest pain or a left pleural effusion.
• Charcot's triad of fever, jaundice, and right upper quadrant pain suggests ascending cholangitis, which should be treated with parenteral antibiotics and surgical consultation.
• Button batteries lodged in the esophagus should be removed emergently. Those in the stomach can be followed clinically up to 48 hours. If the battery has not passed through the pylorus by that time or if any GI symptoms develop at any time, the button should be removed endoscopically.
• Spontaneous bacterial peritonitis is strongly suggested when the ascitic fluid has >500 WBCs/mm³ with >250 PMNs/mm³.
• Co-infection with hepatitis B and hepatitis D is associated with a higher mortality, higher rate of chronic infection, and higher rate of cirrhosis than hepatitis B infection alone.
• 50% to 85% of patients infected with hepatitis C develop chronic hepatitis, which predisposes them to cirrhosis and hepatocellular cancer.
• The most common cause of pancreatitis is choledolithiasis followed by alcohol.
• Crohn's disease is characterized by involvement of all layers of the bowel wall, causing fistulas and abscesses. It can "skip" parts of the GI tract, but can involve any portion of it, and spares the rectum. In contrast, ulcerative colitis affects only the mucosa, with continuous involvement of the GI tract. It is usually limited to the colon and extends to the rectum.
• Common associations in diarrhea:
  o Pet turtle or iguana, sickle cell anemia, splenectomy, after eating poultry or eggs - fecal WBCs are present
    - *Salmonella*
  o After eating poultry or meat - fecal WBCs are not present - *Clostridium perfringens*
  o Recent antibiotics - *Clostridium difficile*
  o After eating potato salad or mayonnaise - *Staphylococcus aureus*
o After eating fried rice - *Bacillus cereus*

o After eating raw oysters - *Vibrio cholera*

o After drinking from natural water sources - *Giardia lamblia*

o AIDS patient - *Isospora* or *Cryptosporidium*

- *Yersinia* infection may cause terminal ileitis that mimics appendicitis.
- *E. coli* 0157:H7 is associated with the development of hemolytic uremic syndrome in children and thrombotic thrombocytopenic purpura in the elderly.
- *Cryptosporidium* is the most common cause of chronic diarrhea in patients with AIDS.
- Patients with GI bleeding and a history of abdominal aortic aneurysm repair have an aortoenteric fistula until proven otherwise.
- Diverticulosis is the most common cause of lower GI bleeding in adults. The bleeding is usually painless and is often massive.

**CHAPTER 4**

**Thoracic and Respiratory Disorders**

- Tracheal deviation or hemodynamic symptoms in the context of a possible tension pneumothorax should prompt immediate needle decompression, followed by insertion of a chest tube. “Obtain a chest film prior to decompression” is never the correct answer on a test question discussing the management of tension pneumothorax.
- The superficial femoral vein is part of the “deep” division; a clot in this vessel is considered deep vein thrombosis (DVT).
- A negative D-dimer is not adequate to rule out pulmonary embolism in a moderate- or high-risk patient.
- A low-probability VQ scan rules out PE only if the pretest probability of PE was low.
- Children with epiglottitis often present in the tripod position, with drooling, stridor, and a toxic appearance. Patients suspected of epiglottitis should undergo direct or indirect laryngoscopy. Remember that a normal x-ray film of the soft tissue of neck does not rule out the diagnosis.
- “Red Flags” in asthma (risk factors for death):
  o Systemic steroids (recent withdrawal or current use)
  o History of exacerbations that were sudden or severe
  o History of exacerbations requiring ICU admission (with or without intubation) or medical floor admissions (>2 in the past year)
  o MDI use (>2 canisters per month)
  o ED visits (>3 in the past year)
  o Comorbid medical, psychiatric, or substance abuse problems
- Toxic irritants usually can be managed with supportive treatment. Exceptions include phosgene and nitrogen oxides, which both can cause delayed pulmonary edema. It is prudent to consider admission for observation.
- Aspiration is not an indication for prophylactic antibiotics; supportive care is appropriate unless the patient shows clinical signs or symptoms of pneumonia.
- Consider the diagnosis of active TB in homeless people, alcoholics, HIV-positive (or otherwise immunosuppressed) individuals, previously incarcerated people, as well as immigrants from (or recent travelers to) countries with high rates of TB; not all will have a pulmonary complaint.
- Ventilator management
  o Acute respiratory distress syndrome or noncardiogenic pulmonary edema is best treated with low tidal volumes (6-8 mL/kg) and adequate PEEP,
  o Asthma is best treated with low tidal volumes, a low ventilation rate, and permissive hypercapnia if required.
• The risk factors for severe bronchiolitis or apnea include age <3 months, respiratory rate >70 breaths/min, congenital cardiopulmonary disease, prematurity (<34 weeks gestation), immunodeficiency, and SaO₂ <95%.
• An aspirated radiolucent foreign body is detectable by air trapping on the affected side, seen when comparing bilateral decubitus films or paired inspiratory and forced-expiratory films.
• Always assume hypercapnia is present in patients with COPD exacerbation and mental status changes.
• Lung abscesses are associated with alcoholism and periodontal disease, produce cavities with air-fluid levels on chest film (see Image #8), and should be treated with antibiotics that cover both aerobes and anaerobes.
• Classic pneumonia associations:
  o After influenza - *Staphylococcus aureus*
  o During pregnancy - varicella
  o With abdominal pain, vomiting/diarrhea, abnormal liver function tests (LFTs), or hyponatremia - *Legionella pneumophila*
  o With bullous myringitis - *Mycoplasma*

**CHAPTER 5**

**Pediatrics**

• Currant jelly stools are a late finding in patients with intussusception. Complaints of vomiting without diarrhea and a low-grade fever, with associated lethargy and/or colicky episodes of pain, should raise suspicion for intussusception.
• Cyanosis that does not respond to oxygen may indicate a congenital heart lesion. Remember the five “terrible Ts” of cyanotic congenital heart disease: tetralogy of Fallot, transposition of the great arteries, tricuspid atresia, truncus arteriosus, and total anomalous pulmonary venous return.
• Neonates with CHF and left-to-right shunting (ventriculoseptal defect, patent ductus arteriosus [PDA], endocardial cushion defect) often present with poor, diaphoretic feeding.
• PDA-dependent lesions usually have a sudden onset and present in the first week of life with cyanosis and shock when the ductus closes. They are treated with a prostaglandin E1 infusion.
• Hyponatremia, hyperkalemia, and hypoglycemia are seen in patients with congenital adrenal hyperplasia. Adrenal crisis should be considered in any newborn with circulatory collapse within the first few weeks of life that is unresponsive to intravenous fluids. Many patients with this condition have ambiguous genitalia. Treatment includes hydrocortisone IV and therapy for hyperkalemia.
• A simple febrile seizure lasts less than 15 minutes, is generalized in nature, lacks clinical findings of meningitis, occurs between the ages 6 months and 5 years, and occurs only once in a 24-hour period.
• Fever in the newborn is defined as a rectal temperature ≥38°C (100.4°F). All patients less than 28 days of age, regardless of a source of fever, should undergo a full septic workup, including lumbar puncture, and should be admitted for IV antibiotics.
• Knee pain in an adolescent or obese patient may be referred from the hip, so consider the diagnosis of slipped capital femoral epiphysis (SCFE) (see Image #66).
• Gingivostomatitis in the anterior mouth is associated with herpes simplex virus infection; posterior involvement suggests Coxsackievirus infection.
• Erythema infectiosum (fifth disease), caused by parvovirus B19, gives patients a “slapped cheek” appearance. It can cause aplastic crisis in patients with sickle cell disease.
• Roseola, caused by human herpes virus 6, produces a high fever for 3 to 5 days, followed by defervescence and a rash.
• Suspect child abuse in the patient whose history is inconsistent with motor development for age and in a patient
with changing histories. Signs of abuse include metaphyseal corner (“bucket-handle”) fractures, posterior rib frac-
tures, subdural hematomas, retinal hemorrhages, and fractures/bruising in different stages of healing.
• Kawasaki’s disease is treated with ASA and IVIG. It is defined by fever for 5 days or longer and the presence of
four of the following five conditions:
  o Bilateral non-exudative conjunctivitis
  o Changes of the lips and oral mucosa (fissured lips, strawberry tongue)
  o Changes in the extremities (erythema of the palms and soles, edema, periungual desquamation)
  o Polymorphous rash
  o Cervical adenopathy (>1.5 cm in diameter)
• Meckel’s diverticulum classically presents with painless GI bleeding and follows the rule of “2’s”:
  o Found in 2% of the population, 45% of symptomatic patients are less than 2 years of age, 2 cm wide, 2 cm
    long, and 2 feet from the ileocecal valve

CHAPTER 6

Procedures and Skills
• Cricothyrotomy is contraindicated in children less than 8 years old.
• Tracheal tube size in infants and children is calculated using the following formula: tube size = 4 + (age in years/4).
• End-tidal CO₂ detection using colorimetric capnometry is the standard to confirm tracheal placement of an endo-
  tracheal tube.
• The ester local anesthetics only have one “i” in their name, while the amides have two. If a patient is allergic to
  an agent in one class, it is safe to use a preservative-free agent from the other class or diphenhydramine for local
  anesthesia.
• Benzocaine can precipitate methemoglobinemia in usual doses.
• 1 mL of 1:1,000 lidocaine contains 1 mg; 1 mL of 1:10,000 lidocaine contains 0.1 mg.
• The maximum dose of lidocaine for local anesthesia is 4.5 mg/kg without epinephrine and 7 mg/kg with epineph-
  rine.
• Acute gout demonstrates needle-like crystals with negative birefringence; pseudogout shows rhomboid crystals
  with positive birefringence.
• Foley catheter placement is absolutely contraindicated in trauma patients with suspected urethral injury (blood at
  the urethral meatus, abnormal or high-riding prostate on exam, or penile/scrotal/perineal hematoma). Obtain a
  retrograde urethrogram prior to placement.
• Xanthochromia occurs when the supernatant of centrifuged CSF is yellow-orange. It is a sign of RBC breakdown
  products from subarachnoid hemorrhage.
• Coat nasal packing with antibiotic ointment and place patients on oral antibiotics to prevent sinusitis (due to
  obstruction of paranasal sinuses and nasolacrimal ducts) and toxic shock syndrome.
• Avulsed teeth should be stored in Hank’s solution, milk, saline, or saliva during transport. Hank’s solution and
  milk increase the viability of the periodontal ligament to >3 hours.

CHAPTER 7

Head, Ear, Eye, Nose, and Throat Disorders
• Patients with cavernous sinus thrombosis are toxic appearing with elevated temperature, proptosis, eyelid edema,
  chemosis, cranial nerve III-VI palsies, as well as engorgement of the fundus. Management involves ENT consulta-
  tion, antibiotics, and admission.
• Malignant otitis externa (see Image #50) is a life-threatening infection of the external auditory canal (the most common organism is *Pseudomonas aeruginosa*) that presents in elderly diabetic, debilitated, or immune compromised patients.
• Ludwig’s angina (see Image #10) is a progressive cellulitis of the floor of the mouth, which involves the bilateral submandibular glands and can produce massive swelling and lead to airway obstruction.
• Persistent foul-smelling rhinorrhea despite appropriate antibiotic treatment in a young child is a nasal foreign body until proven otherwise.
• Corneal burns
  o Acids con JOHN necrosis, shallower burns
  o Alkalis lizards necrosis, deeper burns
• Fluorescein staining may show a “waterfall” of unstained aqueous fluid from the puncture site in penetrating globe trauma; also known as a positive Seidel test.
• A key distinguishing feature between orbital cellulitis and peri-orbital cellulitis (see Image #72) is that orbital cellulitis presents with painful extraocular movement and proptosis, which are not associated with peri-orbital cellulitis.
• Contact lens wearers who present with evidence of conjunctivitis should be treated with fluoroquinolone such as ciprofloxacin or an aminoglycoside such as tobramycin to cover for *Pseudomonas*.
• Herpes simplex keratitis presents with decreased corneal sensation and a dendritic pattern over the cornea (see Image #9) on fluorescein staining. Steroids should not be administered.
• Herpes zoster ophthalmicus can present with Hutchinson’s sign, a vesicular eruption on the nose, which indicates nasociliary nerve involvement.
• Retinal detachment presents as flashing lights, spider webs, and a curtain falling across the visual field.
• The classic funduscopic appearance of central retinal vein occlusion is a “blood and thunder” fundus.
• The classic funduscopic appearance of central retinal artery occlusion is a cherry red spot and a pale retina.
• Temporal or giant cell arteritis is associated with headaches, fever, fatigue, anemia, visual changes, and a high ESR. It is treated with high-dose steroids to avoid blindness.

CHAPTER 8

**Systemic Infectious Disorders**

• Botulism is a descending paralysis, starting with bulbar palsies, diplopia, ptosis, and dysarthria. Guillain-Barre syndrome, in contrast, is an ascending paralysis.
• Staphylococcal toxic shock syndrome is caused by colonization or infection with *Staphylococcus aureus*. It is a multisystem disease with fever, rash with desquamation, and hypotension. It is treated with IV fluids, anti-staphylococcal antibiotics, and vasopressors, if needed.
• The chest film of a patient with primary TB can demonstrate an infiltrate in any lobe.
• The most common cause of death from diphtheria is airway obstruction.
• The classic rash of Rocky Mountain spotted fever (see Image #42) begins on the distal extremities and moves centrally.
• The rash of varicella (see Image #76) has lesions in different stages of evolution. The rash of smallpox has lesions that are all in the same stage of evolution.
• The three stages of Lyme disease are listed below:
  o Stage I (early localized disease) - erythema migrans rash (see Image #40), flu-like syndrome
  o Stage II (early disseminated) - neurologic symptoms (meningoencephalitis, Bells’ palsy), cardiac disease (AV block), mono/oligoarthritis, ophthalmologic disease
  o Stage III (late disseminated) - chronic arthritis, encephalopathy

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• If the patient has signs and symptoms of Lyme disease but has leukopenia, thrombocytopenia, or elevated liver function tests (LFTs), suspect erlichiosis.
• Tetanus causes muscular spasms and contractions (trismus, risus sardonicus, opisthotonus). In strychnine poisoning, trismus is rare and muscle spasms alternate with relaxation.
• Human tetanus immunoglobulin should be given at a site different from where the tetanus toxoid is administered. There is no benefit from injecting it into the wound. In contrast, human rabies immunoglobulin should be injected directly into the wound.
• Toxoplasmosis (see Image #68) is the most common cause of focal intracranial mass lesions in patients with AIDS.
• Cytomegalovirus (CMV) retinitis is the most common cause of blindness in patients with AIDS.
• Consider opportunistic infections in HIV patients with a CD4 count <200 cells/cc.
• Common side effects of medications used in patients with HIV/AIDS:
  o Didanosine: pancreatitis
  o Indinavir: nephrolithiasis
  o Isoniazid: hepatitis
  o Trimethoprim-sulfamethoxazole: hypokalemia, Stevens-Johnson syndrome (see Image #46)
  o Ritonavir: parasthesias
  o Pentamidine: hyperglycemia or hypoglycemia
  o Dapsone: hepatitis

CHAPTER 9

Nervous System Disorders
• Subarachnoid hemorrhage is associated with polycystic kidney disease and aortic coarctation.
• Bell’s palsy (cranial nerve VII palsy) affects the upper and lower facial muscles, but central lesions (not consistent with Bell’s palsy) spare the forehead as a result of cross-innervations.
• Common headache associations:
  o Tension - worsens throughout the day
  o Migraine - young women, aura in 15%, nausea/vomiting
  o Cluster - young man, orbital, occurs in clusters with periods of relapse between episodes
  o Subarachnoid hemorrhage - sudden, severe, “worst headache of life”
  o Meningitis - fever, meningismus
  o Tumor - worse in the morning
  o Pseudotumor cerebri - obese young female, papilledema
  o Glaucoma - vomiting, orbital pain, cloudy cornea, midposition pupil
• Subarachnoid hemorrhage is treated with oral nimodipine to prevent vasospasm and ischemic stroke.
• Myasthenia gravis (MG) is caused by antibodies to the acetylcholine receptors of the neuromuscular junction. It commonly occurs in young females and causes ptosis, diplopia, and proximal muscle weakness. Edrophonium, 1 to 2 mg IV, resolves the symptoms of MG but can cause respiratory arrest or bradycardia in patients with cholinergic crisis; therefore, have airway equipment and atropine at the bedside.
• The infant form of botulism occurs in infants who eat raw honey containing C. botulinum spores and is characterized by constipation, lethargy, failure to thrive, paralysis, and death if not treated.
• Neuroleptic malignant syndrome is characterized by severe extrapyramidal dysfunction, altered mental status, hyperthermia, and autonomic disturbances following neuroleptic use with an elevated CPK. Treat with sodium dantrolene and bromocriptine.
- Epidural abscesses are common in IV drug users and manifest as fever, back pain, and percussive tenderness. MRI is the preferred imaging modality. Treat with antibiotics and neurosurgical consultation.
- Raccoon's eyes, Battle's sign (see Image #52), and CSF otorhea/rhinorhea are all signs of a basilar skull fracture.
- In central cord syndrome, upper extremity weakness is much greater than lower extremity weakness.
- In anterior cord syndrome, there is loss of motor function and pain/temperature sensation distal to the lesion, with preservation of vibration, position, and touch.
- Spinal shock produces complete loss of reflexes and paralysis.
- Brown-Séquard syndrome (cord hemisection) presents with ipsilateral paralysis and loss of position, vibration, and touch, with contralateral loss of pinprick and temperature sensation.
- Thrombolytics for ischemic stroke are indicated only if they can be administered within 180 minutes after the known onset of the neurologic deficit. If the patient awoke with the symptoms, then the 180-minute timer starts when he/she went to bed without symptoms. Fibrinolytics are contraindicated if aggressive antihypertensive therapy (more than 1 or 2 doses of up to 20 mg of labetalol or more than 2 inches of nitroglycerin paste) is required to lower the blood pressure below 185/110 mm Hg. tPA is given at a dose of 0.9 mg/kg, up to a maximum of 90 mg, with 10% given as a bolus and the remainder infused over 60 minutes.
- Pathogens that cause meningitis in various age groups and the corresponding empiric antibiotic therapy are listed below:

<table>
<thead>
<tr>
<th>Patient Population</th>
<th>Common Pathogens</th>
<th>Initial Empiric TV Antibiotic Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>Group B streptococci, <em>Listeria</em>, gram-negative bacilli</td>
<td>Ampicillin, 50 mg/kg, with cefotaxime, 50 mg/kg, or gentamicin, 2.5 mg/kg</td>
</tr>
<tr>
<td>Infants (1 to 3 months)</td>
<td>Group B strep, <em>Listeria</em>, <em>H. influenza</em>, <em>S. pneumoniae</em>, <em>N. meningitidis</em></td>
<td>Cefotaxime, 50 mg/kg, or ceftriaxone, 100 mg/ kg; add vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>3 months to 18 years</td>
<td><em>H. influenza</em> (decreased due to vaccine), <em>S. pneumoniae</em>, <em>N. meningitidis</em></td>
<td>Ceftriaxone, 100 mg/kg IV (max 2 g), plus vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>18 to 50 years</td>
<td><em>S. pneumoniae</em>, <em>N. meningitidis</em></td>
<td>Ceftriaxone, 2 g IV, plus vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>&gt;50 years, alcoholics, or immunosuppressed patients</td>
<td><em>S. pneumoniae</em>, <em>N. meningitidis</em>, <em>Listeria</em>, aerobic gram-negative bacilli</td>
<td>Ceftriaxone, 2 g IV, plus ampicillin, 2 g IV, plus vancomycin for resistant <em>S. pneumoniae</em></td>
</tr>
</tbody>
</table>

CHAPTER 10

Obstetrics and Gynecology
- Postmenopausal women with vaginal bleeding must have close follow-up to exclude endometrial carcinoma.
- Thromboembolism is the primary cause of maternal mortality in the United States.
- For the treatment of thromboembolic disease in pregnant women, heparin is the drug of choice because coumadin is contraindicated.
- Preeclampsia is characterized by hypertension, headache, visual disturbances, edema, and abdominal pain in a patient >20 weeks pregnant. HELLP syndrome is a form of preeclampsia characterized by hemolysis, elevated liver enzymes, and low platelets. Eclampsia is preeclampsia combined with seizures, which usually responds to magnesium and antihypertensive therapy, with delivery as the ultimate definitive treatment.
- Vena caval compression occurs in 10% to 15% of pregnant women who lie supine. Place pregnant trauma and critically ill patients in the left lateral recumbent position to relieve compression and improve venous return to the heart.
- The sterile speculum exam is contraindicated until placenta previa has been ruled out with ultrasound in women with painless third-trimester vaginal bleeding.
The appendix is displaced counterclockwise into the right upper quadrant after the third month of gestation. Appendicitis may present as right upper quadrant pain.

Maternal stabilization is the most important factor in determining fetal survival.

Any third-trimester pregnant patient with trauma and vaginal bleeding should be presumed to have placental abruption. Transabdominal ultrasound is insensitive for placental abruption.

An intrauterine pregnancy (IUP) can be visualized by transvaginal ultrasound when the quantitative P-hCG level is between 1500 and 2000 IU/ml and by transabdominal ultrasound when the level is greater than 6500 IU/ml. If an IUP is not visualized and the quantitative HCG concentration is above these discriminatory zones, ectopic pregnancy should be assumed until proven otherwise.

Rh-negative unimmunized women with antepartum vaginal bleeding in pregnancy should receive 300 micrograms of RhoGAM IM (50 micrograms if in first trimester).

Fitz-Hugh-Curtis syndrome presents in patients with pelvic inflammatory disease as RUQ pain and jaundice.

Causes of vulvovaginitis and associations:
- Trichomonas vaginalis - grey/yellow frothy malodorous discharge; treated with metronidazole or clindamycin
- Gardnerella vaginalis - “fishy” odor, clue cells; treated with metronidazole or clindamycin
- Candida albicans - white “cottage cheese” discharge; treated with topical or oral antifungals

CHAPTER 11

Hematologic and Immune System Disorders

- Transfusing 1 unit of packed red blood cells increases the hemoglobin by 1 g/dL and hematocrit by 3%. One unit of platelets increases the platelet count by 5,000 to 10,000/pL.
- Warfarin inhibits the vitamin-K-dependent factors II, VII, EX, and X. FFP provides immediate but short-lived reversal of coagulopathies, while vitamin K provides definitive, long-term reversal.
- Initial treatment of a sickle cell crisis includes hydration, oxygen, pain control (avoid meperidine, because its metabolite can cause seizures), and supportive care. If the patient is febrile, consider antibiotics, because the risk of infection is increased. The various crises are listed below:
  - Hemolytic - drop in hematocrit with jaundice
  - Aplastic - drop in hematocrit and reticulocyte count, caused by parvovirus
  - Sequestration — pancytopenia in children with hepatosplenomegaly
- Hemophilia A patients have abnormal function of factor VIII, with a normal PT and prolonged aPTT. Recombinant factor VIII is administered for bleeding as described below:
  - Minor bleed (hemarthrosis, hematuria) - 12.5 U/kg
  - Major bleeds (intracranial, major trauma or surgery) - 50 U/kg
If the potential exists for a severe bleed, factor replacement should begin before diagnostic imaging, such as a head CT in a patient with suspected intracranial hemorrhage.
- G6PD deficiency—the most common human enzyme defect—presents in association with acute hemolytic crises incited by infection, administration of oxidant drugs (e.g., sulfa, phenazopyridine, antimalarials, salicylates), metabolic acidosis (such as DKA), and ingestion of fava beans.
- Thrombotic thrombocytopenic purpura (TTP) presents classically with thrombocytopenia, hemolytic anemia, fluctuating neurologic changes, renal disease, and fever. It is treated with plasma exchange with FFP, steroids, and antiplatelet drugs (e.g., aspirin).
- Von Willebrand’s disease is the most common genetic bleeding disorder. It is characterized by a normal platelet count with abnormal function (prolonged bleeding time). It can be treated with DDAVP, FFP, cryoprecipitate, or “Humate-P” factor VIII concentrate.
• Oncologic emergencies and their treatments:
  o Spinal cord compression — dexamethasone, radiation, surgery
  o Upper airway compression - establish definitive airway
  o Pericardial tamponade - pericardiocentesis
  o Superior vena cava syndrome - diuretics, radiation, vascular stenting
  o Hypercalcemia - IV saline, furosemide
  o SIADH - fluid restriction, diuretics, hypertonic saline (coma/seizures)
  o Hyperviscosity - IV saline, phlebotomy
  o Adrenocortical insufficiency with shock - hydrocortisone IV

• Oncology associations:
  o Malignancy and constipation - hypercalcemia
  o Malignancy and fever - neutropenia or sepsis
  o Malignancy and hypotension/JVD/SOB - tamponade or superior vena cava syndrome
  o Malignancy and hypoglycemia - adrenal insufficiency
  o Malignancy and hyponatremia - adrenal insufficiency or SIADH
  o Malignancy and back pain - cord compression

• The hyponatremia associated with SIADH should be corrected slowly to avoid central pontine myelinolysis and cerebral demyelination.

• Adrenocortical insufficiency is caused by previous steroid therapy, tumors, infection, hypothalamic-pituitary insufficiency, and adrenal hemorrhage. It presents as vasomotor collapse and is treated with hydrocortisone, 100 mg IV q6-8h.

• The classic triad of Reiter’s syndrome consists of arthritis, conjunctivitis, and non-gonococcal urethritis triggered by Chlamydia or a gastrointestinal infection.

• Anaphylaxis
  o Most common cause of death is airway obstruction.
  o Classic anaphylaxis is IgE mediated while anaphylactoid reactions are non-IgE mediated,
  o Causes hypotension, vasodilation, and capillary leak
  o Primary treatment is epinephrine.
  o Other treatments include albuterol (bronchospasm), diphenhydramine, H2-blocker (ranitidine), and corticosteroids.
  o Patients on P-blockers are resistant to epinephrine and need glucagon.
  o Refractory hypotension is treated with an epinephrine, a norepinephrine, or a dopamine drip.

• Rheumatic fever is diagnosed based on modified Jones criteria:
  o Major: carditis, polyarthritis, Sydenham’s chorea, erythema marginatum, subcutaneous nodules
  o Minor: arthralgias, fever, increased erythrocyte sedimentation rate or C-reactive protein, prolonged P-R interval on ECG

Two major criteria or one major and two minor criteria must be present to make the diagnosis.

CHAPTER 12

Toxicologic Disorders

• Cyanide smells like bitter almonds; hydrogen sulfide smells like rotten eggs.

• Anytime a toxicology patient has a tachycardic ECG, look for signs of tricyclic antidepressant (TCA) toxicity:
  large S in I and aVL, R >3 mm in AVR, wide QRS complex, sinus tachycardia, long QT interval.
Paroxysmal atrial tachycardia with variable AV block is digoxin toxicity until proven otherwise.

Every toxicology case on the boards that has an ECG associated with it is either TCA or digoxin toxicity.

Charcoal is not useful for lithium, alkalis/acid, iron, and other heavy metals.

Drugs for which hemodialysis may be effective can be remembered with the mnemonic STUMBLE: salicylates, theophylline, methanol, barbiturates, lithium, ethylene glycol. “U” stands for “uremia” and serves as a reminder that patients who are uremic or in renal failure may also need dialysis to help eliminate the drug.

Whole bowel irrigation is useful for lithium, iron, heavy metals, sustained-release drugs, and body stuffers.

Do not lavage caustic (acid and alkali) ingestions.

Serum alkalinization is useful for TCA toxicity, with a goal blood pH of 7.5. Urine alkalinization is useful for salicylates, chlorpropamide, and barbiturates, with a goal urine pH >7.5.

The combination of anion gap metabolic acidosis with respiratory alkalosis is aspirin toxicity until proven otherwise. Aspirin can also cause hyperthermia, hypoglycemia, and hypokalemia.

Ingestion of ethylene glycol causes anion gap acidosis and an osmolar gap. It is treated with ethanol or fomepizole.

Ingestion of methanol causes anion gap acidosis and an osmolar gap. The primary manifestations are visual symptoms with optic disc hyperemia secondary to formic acid. It is treated with fomepizole, ethanol, or dialysis.

CHAPTER 13

Endocrine, Metabolic, and Nutritional Disorders

- Hypoglycemic patients taking oral hypoglycemic medications should be admitted for observation.
- Isopropyl alcohol ingestion usually presents with ketosis without acidosis.
- The classic presentation of Wernicke's encephalopathy is confusion, ataxia, ophthalmoplegia, and nystagmus.
- The classic triad of myxedema coma is hypotension, hypothermia, and bradycardia.
- The classic electrolyte abnormalities of adrenal insufficiency are hyponatremia and hyperkalemia.
- Lactic acidosis is the most common cause of anion-gap metabolic acidosis.
- The anion gap = Na - [HC03 + Cl] (normal is <10).
- Dialysis patients in cardiac arrest are hyperkalemic until proven otherwise.
- If a patient is hypokalemic, consider hypomagnesemia as the cause.
- The treatment of a hyponatremic patient who has had a seizure or who has mental status changes is administration of 3% hypertonic saline.

CHAPTER 14

Environmental Disorders

- Lyme disease (spirochete Borrelia burgdorferi) is tick-borne; often manifests with a target or erythema migrans rash (see Image #40); and can later cause facial nerve palsy, meningoencephalitis, and myocarditis.
- The key treatment for tick paralysis is to remove the tick.
- Bony fishes, stingrays, and sea urchins inject a heat-labile toxin that can be deactivated with hot water, but beware of foreign bodies.
- Jellyfish nematocysts should be removed using salt water or vinegar; using fresh water may cause them to activate.
- Antivenin is indicated for any coral snake bite and any moderate to severe pit viper bite; beware of anaphylaxis when horse serum antivenin is used.
- Black widow spider bites cause muscular rigidity that can be mistaken for a surgical abdomen and require treatment with benzodiazepines and antivenin.
- The symptoms of barotrauma can include joint pain or mimic stroke or pulmonary embolus; treatment is with 100% oxygen followed by hyperbaric oxygen.
For high-altitude illness such as pulmonary edema or cerebral edema, the primary management is to decrease altitude; milder acute mountain sickness can be managed while the patient acclimatizes.

The primary management for heat stroke is to lower the core temperature to 40°C (104°F); acetaminophen is ineffective. Consider heat stroke in any patient with altered mental status and a fever, especially an athlete who collapses.

Severe hypothermia creates Osborne J-waves and dysrhythmias, which generally resolve during rewarming; atropine, epinephrine, and high-dose dopamine are contraindicated; defibrillate once if indicated.

An absolute lymphocyte count <500/mcL correlates with severe radiation exposure and a poor prognosis.

CHAPTER 15

Musculoskeletal Disorders

Osteomyelitis is most commonly caused by *Staphylococcus aureus*. On the board exam, the following are common associations with causative agents: sickle cell disease and *Salmonella*, foot puncture wounds and *Pseudomonas*, and dog/cat bites and *Pasteurella*.

The most common infective agent in septic arthritis is *Staphylococcus aureus*. If it is migratory, think gonorrhea.

Gonorrhea, rheumatoid arthritis, Lyme disease, and Reiter’s syndrome cause oligoarticular arthritis, while lupus, viral infections, and rheumatoid arthritis cause polyarthritis (more than three joints involved).

Rhabdomyolysis classically shows urinalysis positive for hemoglobin with no red cells on microscopy and a total CPK more than five times the upper limit of normal. It is treated with a saline infusion initially, supplemented with urine alkalinization, mannitol, and dialysis.

The scaphoid is the most commonly fractured carpal bone.

Scapholunate dislocation is present when there is a gap greater than 3 mm between the scaphoid and lunate (Terry Thomas sign, see Image #65).

GRUM is a mnemonic to remember the difference between a Galeazzi and Monteggia fracture - a Galeazzi fracture is a fracture of the radius, and a Monteggia fracture (see Image #28) is a fracture of the ulna.

On a lateral radiograph of the elbow, a large anterior fat pad (sail sign) or any visible posterior fat pad is consistent with a radial head fracture in adults and a supracondylar fracture in children.

Posterior shoulder dislocations are caused by falls, seizures, and electric shocks.

A Lisfranc fracture is a disruption of the tarsal-metatarsal joint, commonly with a fracture at the base of the second metatarsal (see Image #60).

A nursemaid’s elbow, or radial head subluxation, is caused by a distracting force applied to an immature elbow—often a pulling motion applied to a child’s arm.

Legg-Calve-Perthes disease is avascular necrosis of the femoral head and commonly occurs in prepubertal children. In contrast, a slipped capital femoral epiphysis (see Image #66) commonly occurs in obese pubertal children and may present as knee rather than hip pain.

CHAPTER 16

Psychobehavioral Disorders

Flumazenil use in a patient on chronic benzodiazepine therapy may cause intractable seizures.

P-Blockade is contraindicated in cocaine-intoxicated patients, because of the risk of unopposed a-adrenergic vasoconstriction.

Organic causes of new-onset psychosis must be ruled out before a psychiatric diagnosis is made.

In chronic alcohol abusers, thiamine should be administered before glucose so as not to precipitate Wernicke’s encephalopathy.
• Retinal hemorrhages in a child younger than 1 year of age are indicative of shaken baby syndrome.
• Alcohol withdrawal can occur in patients whose blood alcohol levels are elevated but lower than usual.
• Delirium can be differentiated from dementia by its sudden onset, fluctuating course, disturbed level of consciousness and awareness, and hallucinations.
• When a patient expresses homicidal intent, the physician has a duty to report the information to authorities or third parties.
• Chain of custody must be maintained when collecting legal evidence in sexual assault cases.
• Suicide risk factors include Caucasian race; elderly, unmarried, unemployed men; women with recent personal loss; and anyone with a history of chronic illness, prior suicide attempt, psychiatric or substance abuse history, a positive family history, and a firearm in the home.

CHAPTER 17

Renal and Urogenital Disorders
• The most common cause of acute intrinsic renal failure is acute tubular necrosis.
• If a child presents with hypertension, edema, proteinuria, and hematuria, consider acute post-streptococcal glomerulonephritis.
• E. coli is the most common organism causing urinary tract infection.
• Hyperkalemia should be strongly considered in all patients with end-stage renal disease.
• Non-contrast spiral CT is the “gold standard” for detection of urolithiasis, with 95% sensitivity and specificity.
• Consider testicular torsion in any male with lower abdominal pain.
• Acute epididymitis in men older than 35 years is likely caused by E. coli.
• Fournier’s gangrene (see Image #2) is polymicrobial, with E. coli being the most predominant organism.
• The treatment of irreducible paraphimosis is with a dorsal slit of the constricting band.
• Dialysis disequilibrium syndrome is a result of excessive solute clearance and can lead to seizures, coma, and death.
• The treatment of priapism includes external compression, subcutaneous terbutaline, and intracavernous injection of dilute phenylephrine.
• With torsion of the testicular appendage, the “blue dot sign” (gangrenous appendix) can sometimes be visualized when the scrotal skin is stretched; this sign is pathognomonic.

CHAPTER 18

Cutaneous Disorders
• Kaposi’s sarcoma presents as nonblanching, raised brown-black nodules and is diagnostic of AIDS.
• The treatment of severe toxicodendron dermatitis (poison ivy, oak, or sumac) should involve a 2-3 week prednisone taper.
• For facial cellulitis in an ill, unimmunized child, consider Haemophilus influenza.
• Severe, necrotizing infections often present with pain out of proportion to the exam and require aggressive management with early surgical consultation.
• Severe tinea capitis can create a kerion (a boggy, indurated plaque) that is best managed with a 6- to 8-week course of griseofulvin.
• Scabies creates a pruritic rash with burrows (see Image #16). It spares the head and neck and is best managed with permethrin for the patient and close personal contacts.
• Koplik’s spots are small, irregular, bright red spots with bluish white centers on the buccal mucosa. They are diagnostic of measles.
• Erythema multiforme (see Image #49) and toxic epidermal necrolysis often create a target lesion with central clearing.
• Pityriasis rosea may be identified by a large initial “herald patch” and “Christmas tree” pattern (see Image #51); it generally resolves in 3 to 8 weeks without treatment.
• Nikolsky’s sign—the sloughing off of normal skin with lateral pressure—may indicate severe disease, such as pemphigus vulgaris or toxic epidermal necrolysis, that requires inpatient management similar to the management of a severe burn.
• Petechial lesions are red or purple subcutaneous hemorrhages that do not blanch (see Image #36). They may indicate severe illness such as meningococcal sepsis (see Images #3 5A and 35B), disseminated gonorrhea, or Rocky Mountain spotted fever (see Image #42).

CHAPTER 19

Emergency Medical Services
• The best outcomes in cardiac arrest occur if CPR is started within 4 minutes after arrest and ACLS-level care within 8 minutes after arrest.
• An on-scene (bystander) physician who wishes to direct care by EMS must be able to provide proof of identity and licensure.
• The primary advantage of helicopter transport is reduced transport time.
• A disaster is present when the needs of the casualties exceed available resources.
• Triage—the classification of patients into treatment priorities—is a fluid, ongoing process.
• The guiding principle of triage in a disaster situation is to do the greatest good for the greatest number of patients.
• The four categories of triage are critical, priority, delayed, and expectant.
• An incapable or impaired patient should NOT be allowed to refuse care. Police officers can be used to restrain such an individual.
CHAPTER 21
Practice Test

Elizabeth A. Gray, MD (Visual Stimuli Questions)

This practice test is modeled after the American Board of Emergency Medicine in-training examination, also known as the “in-service exam.” Like the in-service exam, it consists of 225 multiple-choice questions, including 25 that involve visual stimuli. Readers wishing to simulate the in-service exam should allow a maximum of 4.5 hours to complete the test. These questions were developed by the chapter authors and edited by Joseph R. Lex, Jr., MD. The questions utilizing visual stimuli were developed by Elizabeth A. Gray, MD.

For the first 25 questions, please refer to Images 1 through 25 in Chapter 22, Visual Images.

1. For the past 12 hours, a previously healthy, full-term, fully immunized 14-month-old girl has had a fever, difficulty breathing, and drooling. Her parents tell you that she refuses to eat or drink. She is resting quietly in her father’s arm with her chin draped over his shoulder, drooling profusely, and making a soft high-pitched noise with inspiration. Vital signs: heart rate, 162 beats/min; respiratory rate, 30 breaths/min; rectal temperature, 102.1°F; SaO₂, 97% on room air. You order a lateral neck radiograph, which is shown in Image 1, Chapter 22. Your next most important step is to:
   A. Obtain IV access and blood cultures, then start broad-spectrum antibiotics.
   B. Perform emergent needle cricothyrotomy in the emergency department.
   C. Administer nebulized racemic epinephrine.
   D. Obtain consultation for urgent intubation in the operating room.
   E. Administer nebulized albuterol.

2. A 36-year-old man complains of several days of severe perineal pain and erythema. Image 2, Chapter 22, illustrates what you see on his genital examination. The organism most likely responsible for this condition is:
   A. Candida albicans.
   B. Treponema pallidum.
   C. Neisseria gonorrhoeae.
   D. Cryptosporidium.
   E. Bacteroides fragilis.
3. A 19-year-old woman is brought to your emergency department after a motor vehicle crash in which she was the unrestrained driver. She is awake, alert, and speaking clearly but complains of chest pain and shortness of breath. Vital signs: heart rate, 105 beats/min; blood pressure, 99/63 mm Hg; respiratory rate, 24 breaths/min; SaO₂, 96% on room air. The patient's chest radiograph is shown in Image 3, Chapter 22. You should now:
A. Perform endotracheal intubation.
B. Perform tube thoracostomy with a 24 French chest tube.
C. Do a needle thoracostomy in the second intercostal space.
D. Arrange for 6 hours of emergency department observation with serial chest films.
E. Place an intravenous catheter and run a bolus of 2,000 ml isotonic fluid.

4. You are evaluating a 22-month-old boy whose mother suspects he swallowed a coin 2 hours ago after his 4-year-old sister gave him some spare change. The child is asymptomatic and is breathing normally. His radiograph is shown as Image 4, Chapter 22. You should now:
A. Order a lateral radiograph to determine whether the coin is in the trachea or esophagus before you attempt any intervention.
B. Order a barium swallow study to determine whether there is esophageal perforation before you attempt any intervention.
C. Attempt to retrieve the object by passing a Foley catheter distal to it, inflating the balloon, and withdrawing the catheter.
D. Consult gastroenterology for urgent endoscopy to remove the object.
E. Reassure the mother that the coin will pass and no intervention is necessary.

5. A 33-year-old man complains of severe leg pain, but denies trauma. Vital signs: heart rate, 133 beats/min; blood pressure, 72/35 mm Hg; respiratory rate, 24 breaths/min; temperature, 101°F. His radiograph is shown in Image 5, Chapter 22. Your next step is to:
A. Administer broad-spectrum antibiotics.
B. Administer low-molecular-weight heparin.
C. Administer vasopressor agents.
D. Arrange for hyperbaric oxygen therapy.
E. Perform incision and drainage in the emergency department.

6. A 57-year-old woman is brought in by family for evaluation after she experienced her first seizure. Her medical history is unremarkable, and she felt fine prior to the seizure. Her noncontrast head CT scan is shown in Image 6, Chapter 22. The historical feature most likely to explain the CT finding and the seizure is:
A. Enlarging breast mass over several months.
B. Consumption of pork in a developing country.
C. Exposure to cat feces.
D. Dental surgery 6 weeks ago.
E. Fall with loss of consciousness 3 months ago.
7. A 24-year-old man complains of severe wrist pain after falling while playing ice hockey. His radiograph, shown in Image 7, Chapter 22, shows a:
   A. Scaphoid fracture.
   B. Scapho-lunate dissociation.
   C. Distal radioulnar joint dislocation.
   D. Lunate dislocation.
   E. Perilunate dislocation.

8. A 32-year-old woman complains of 10 to 12 days of pleuritic chest pain, productive cough, and low-grade fever. Vital signs: temperature, 100.2°F; heart rate, 88 beats/min; respiratory rate, 18 breaths/min; blood pressure, 108/72 mm Hg. Other than undergoing surgery for correction of scoliosis several years earlier, she has an unremarkable medical history. Her chest film is shown as Image 8, Chapter 22. The historical or physical finding most pertinent to this radiographic finding is:
   A. Car travel over 3,000 miles 2 weeks ago.
   B. Smoking 2 packs of cigarettes per day for 15 years.
   C. Severe gingivitis.
   D. Employment as a prison health care worker.
   E. Family history of alpha-1-antitrypsin deficiency.

9. A 27-year-old woman complains of eye pain that has lasted 2 days. She wears contact lenses. After instilling fluorescein, you examine her eye, which is shown in Image 9, Chapter 22. She requires therapy with:
   A. Topical acyclovir drops.
   B. Intravenous acyclovir.
   C. Topical ciprofloxacin drops.
   D. Topical prednisolone drops.
   E. Intravenous methylprednisolone.

10. A 14-year-old boy complains of fever, neck swelling, and painful swallowing. His neck examination is shown in Image 10, Chapter 22. The most worrisome life-threatening complication of this condition is:
    A. Mediastinitis.
    B. Asphyxiation resulting from sudden airway loss.
    C. Bacteremia and overwhelming sepsis.
    D. Pulmonary abscess.
    E. Thyroxine (T4) and catecholamine excess.

11. A 36-year-old woman is brought by paramedics from a motor vehicle crash, where she had to be extricated from the back seat. She is diaphoretic and confused and has undetectable peripheral pulses. Vital signs: heart rate, 130 beats/min; blood pressure, 65/30 mm Hg; respiratory rate, 28 breaths/min. During the primary survey, you intubate the patient and begin fluid resuscitation through two large-bore peripheral IV lines. You then perform a bedside ultrasound exam; Image 11, Chapter 22, gives you one view. Your next step is to:
    A. Order a CT scan of the chest, abdomen, and pelvis.
    B. Arrange for pelvic angiography to be done by interventional radiology.
    C. Obtain an oral contrast study to rule out visceral rupture.
    D. Arrange for the patient to be taken to the operating room for emergent laparotomy.
    E. Perform emergent pericardiocentesis.
12. A 23-year-old woman at an estimated 29 weeks gestational age complains of severe right lower quadrant pain of 7 hours duration. She has some mild nausea but no fever. She has not had any hematuria or vaginal bleeding. You perform ultrasound, with the most diagnostic image shown in Image 12, Chapter 22. These findings:
   A. Mandate urgent urologic consultation.
   B. Exclude the possibility of acute appendicitis.
   C. Confirm obstructive urolithiasis as the cause of the symptoms.
   D. May be normal in pregnancy.
   E. Mandate noncontrast CT scan as a follow-up imaging procedure.

13. A 12-year-old boy was bitten on the leg by a spider 6 days ago while playing in an abandoned shed. The bite was mildly painful at first, and over several hours erythema developed at the bite site. Now the area looks as shown in Image 13, Chapter 22. Your next step is:
   A. Urgent surgical excision of the necrotic area.
   B. Antivenin therapy.
   C. Antibiotics for the surrounding cellulitis.
   D. Benzodiazepines and opioids for pain management.
   E. Immersion of the affected extremity in hot water.

14. A 9-year-old girl has had a non-blanching rash (shown in Image 14, Chapter 22) for the past 12 hours. In addition, she complains of generalized abdominal pain, knee pain, and ankle pain. She is well appearing, is afebrile, and has normal vital signs. The lab abnormality you would most likely expect is:
   A. Thrombocytopenia.
   B. Elevated lipase.
   C. Prolonged PTT.
   D. Positive antinuclear antibody titers.
   E. Hematuria.

15 A 22-year-old man complains of a painless lesion at the base of the penile shaft (Image 15, Chapter 22), which has been present for 12 days. The treatment of choice is:
   A. Benzathine penicillin, G 2.4 million units IM in a single dose.
   B. Acyclovir, 400 mg PO TID for 10 days.
   C. Azithromycin, 1 g PO in a single dose.
   D. Podofilox, 0.5% solution applied to lesion BID for 3 days.
   E. Ceftriaxone, 250 mg IM in a single dose.

16. A 19-year-old man complains of an itchy rash on both hands, as shown in Image 16, Chapter 22. The most appropriate treatment is:
   A. Selenium sulfide shampoo to scalp and affected areas daily.
   B. Clotrimazole cream, 1%, topically BID.
   C. Hydrocortisone cream, 1%, topically BID.
   D. Fluocinonide ointment, 0.05%, topically BID.
   E. Permethrin cream, 5%, to the entire body except the head, then washed off in 8 to 12 hours.
17. A 4-year-old girl has had 2 days of a tender erythematous rash, as shown in Image 17, Chapter 22. Her mucous membranes are normal, but lateral pressure on the skin lesions causes wrinkling and peeling of the skin, even in areas not affected by the erythema. The patient has not taken any medications within the past 10 days. The most likely cause of her rash is:
   A. Autoantibodies to cell adhesion proteins.
   B. A sulfonamide antibiotic taken as a 10-day course completed 2 months ago.
   C. A bacterial exotoxin.
   D. Cutaneous diphtheroids.
   E. Parvovirus B19.

18. A 25-year-old woman, G3P1 at 7 weeks gestational age estimated by last menstrual period, complains of severe left lower quadrant pain that started suddenly 2 hours ago. She saw her primary care provider 5 days ago for painless vaginal bleeding and had missed her prior period 2 weeks before; a qualitative urine hCG was positive. She has had no further bleeding since then and no pain until today. Her quantitative hCG today is 2,175. The results of a pelvic ultrasound are shown in Image 18, Chapter 22. The most likely cause of the patient's symptoms is:
   A. Normal intrauterine pregnancy with round ligament pain.
   B. Ruptured ectopic pregnancy.
   C. Ruptured ovarian cyst.
   D. Molar pregnancy.
   E. Blighted ovum.

19. A 29-year-old man complains of painful genital lesions, as shown in Image 19, Chapter 22. The agent most likely responsible for these findings is:
   A. Treponema pallidum.
   B. Haemophilus ducreyi.
   C. Chlamydia trachomatis.
   D. Calymmatobacterium granulomatis.
   E. Herpes simplex virus type 1.

20. A 19-year-old woman developed sudden intense right eye pain as she was attempting to insert her contact lenses. Image 20, Chapter 22, shows her eye after fluorescein staining. You know you must treat her with an antipseudomonal topical antibiotic and:
   A. Topical prednisolone drops.
   B. Topical cycloplegic drops.
   C. Patching the eye.
   D. Topical acyclovir drops.
   E. Topical timolol drops.

21. A 28-year-old man is brought to the ED by police after being shot in the neck (Image 21, Chapter 22). Since there is a defect in the platysma, the next step in management is:
   A. Closure of the soft tissue defect in the emergency department.
   B. Wound probing to determine the trajectory of the missile.
   C. Angiography of the neck vessels.
   D. Esophogram and esophagoscopy.
   E. Surgical exploration of the wound in the operating room.
22. A 22-year-old man was the restrained back seat passenger in a motor vehicle crash. He was extricated by EMS personnel, placed on a backboard with spinal precautions, and brought to the emergency department complaining of low back pain. The lumbar spine lateral radiograph is shown in Image 22, Chapter 22. The finding most commonly associated with this injury is:
   A. Paralysis of both lower extremities.
   B. Anesthesia in the L1 dermatome.
   C. Saddle anesthesia.
   D. Abdominal tenderness.
   E. Urinary retention.

23. A 9-year-old girl fell off her skateboard and sustained a distal radius fracture. Image 23, Chapter 22, shows her injury. What type of Salter-Harris fracture is this?
   A. Type II, with low potential for growth arrest
   B. Type II, with high potential for growth arrest
   C. Type III, with some potential for growth arrest
   D. Type IV, with low potential for growth arrest
   E. Type IV, with high potential for growth arrest

24. A 66-year-old man complains of a fast heart rate. His ECG shows only sinus tachycardia at a rate of 118 beats/min. Because of his appearance, as shown in Image 24, Chapter 22, you decide the test most likely to demonstrate the cause of his tachycardia is:
   A. Complete blood count (CBC).
   B. Urine drug screen.
   C. Thyroid-stimulating hormone (TSH).
   D. Measurement of intraocular pressures.
   E. Serum digoxin level.

25. A 7-year-old boy has multiple pearly flesh-colored papules (Image 25, Chapter 22), which have increased in number over the past 5 days. Some of these lesions have a small central depression. He denies itching and is otherwise asymptomatic and afebrile. The causative virus is:
   A. Molluscum contagiosum virus.
   B. Variola.
   C. Varicella-zoster.
   D. Herpes simplex virus.
   E. Vaccinia.

The remainder of the questions will not require the images in Chapter 22.

26. A 60-year-old woman who recently returned from Europe presents with a mildly swollen and painful right leg. Vital signs: heart rate, 90 beats/min; blood pressure, 120/90 mm Hg; respiratory rate, 14 breaths/min; SaO₂, 97%. She is afebrile. The most appropriate next step in management is to:
   A. Administer antibiotics.
   B. Begin warfarin therapy.
   C. Obtain a lower extremity ultrasound.
   D. Obtain lower extremity venography.
   E. Order a d-dimer and, if positive, begin therapy.
27. You are seeing a 3-month-old boy for mild croup. As you prepare to discharge him, his mother asks you about a “bulge” in his umbilicus. He is asymptomatic and has been eating and drinking normally without change in his usual bowel habits. You should:
   A. Recommend his pediatrician refer him to a surgeon for elective repair.
   B. Cancel the discharge and admit him to the inpatient pediatrics team.
   C. Reassure the mother that this condition usually resolves on its own.
   D. Obtain general surgery consultation in the ED.
   E. Perform a flat and upright plain radiograph to assess for obstruction.

28. A trauma patient is brought to the emergency department by an EMS unit. During your secondary survey, the pupil exam is notable for an ipsilateral fixed, dilated pupil. This is most likely caused by a(n):
   A. Frontal contusion.
   B. Cerebellotonsillar herniation.
   C. Occipital contusion.
   D. Uncal herniation.
   E. Transtentorial herniation.

29. A 25-year-old man with a history of asthma is brought to the emergency department by ambulance. He is agitated, leaning forward, shaking the stretcher rails with his hands, and occasionally yelling in short bursts, “Let me go.” The EMTs tell you that he became increasingly agitated during transport and would not tolerate nebulizer treatments. Vital signs: heart rate, 135 beats/min, SaO2, 89% on room air (he keeps removing the nebulizer mask). He is still uncooperative, and his agitation is decreasing. He is not tachypneic but is using accessory muscles, and you are able to quickly listen to his chest. The only breath sounds you hear are minimal wheezing, equal in all lung fields. Your next intervention should be:
   A. Intravenous or intramuscular haloperidol, 5 mg, and lorazepam, 2 mg.
   B. Intubation and mechanical ventilation.
   C. Subcutaneous terbutaline or epinephrine.
   D. Intravenous methylprednisolone, 125 mg.
   E. Continued nebulized albuterol and ipratropium.

30. You have intubated the patient described in Question 29, and you confirm tube placement with examination, end-tidal CO2 detection, and a chest film. You order intravenous steroids and nebulized beta-agonist treatments to be given through the tubing. You are called into the next room for a newly arrived cardiac arrest patient; when you return, you see that the new respiratory therapist has chosen the initial ventilator settings for your asthma patient. He is still paralyzed, but the ventilator alarm is sounding because of high plateau pressures. After you listen to verify that the patient has equal breath sounds, you can reduce his risk of barotrauma by:
   A. Drawing an arterial blood gas and altering the settings based on the results.
   B. Decreasing the I:E ratio.
   C. Ordering lorazepam, 2 mg IV.
   D. Increasing the number of breaths per minute.
   E. Decreasing the inspiratory flow rate.
31. A previously healthy 6-year-old girl has had 2 days of grossly bloody diarrhea and diffuse crampy abdominal pain with no fever or vomiting. She has normal vital signs and appears nontoxic and well hydrated. You find minimal diffuse abdominal tenderness. Blood tests are normal and a stool specimen is collected and sent for culture. You should now:

A. Discharge her home on no antibiotics, with close follow-up of the stool culture.
B. Start trimethoprim/sulfamethoxazole.
C. Start azithromycin.
D. Start metronidazole.
E. Admit to the hospital for intravenous antibiotics.

32. You are repairing a laceration on a patient with a lidocaine allergy. A local anesthetic that would be safe to use in this patient is:

A. Mepivacaine.
B. Prilocaine.
C. Procaine.
D. Bupivacaine.
E. Etidocaine.

33. A 16-year-old boy complains of ear pain. He says that he was wrestling and was hit in the ear with an elbow. You note that his left ear is red and warm, and the auricle has areas of ecchymosis and edema. There is a significant amount of tenderness to palpation. The most correct statement regarding his treatment is:

A. Following treatment, his condition is self-limiting and requires no follow-up.
B. Following treatment, he should return in 24 hours for re-assessment.
C. He requires a STAT head CT scan. Bruising of the auricle is a hallmark of underlying intracranial injury.
D. Incision, drainage, and wound packing are the treatment of choice. Antibiotics are rarely needed in the management of an auricular hematoma.
E. Application of a loose, non-occlusive dressing along with 24-hour follow-up is the treatment of choice.

34. Lacunar cerebrovascular infarcts are:

A. Usually caused by an acute hypertensive event.
B. Caused by infarction of large arteries.
C. Primarily located in the cerebellum.
D. Associated with pure motor or sensory deficits.
E. Known to frequently cause changes in level of consciousness.
35. A 56-year-old man has altered mental status. His daughter, who does not live with him, found him this morning lying in bed disoriented. She spoke with him on the phone 2 days ago and he told her that he had a headache and some nausea. The daughter also tells you that he has a history of chronic alcohol abuse. His surgical history is significant for a splenectomy following a motor vehicle collision 15 years ago. He has no allergies and is taking no medications. There is no known history of recent travel or outdoor activities. Vital signs: temperature, 102.1 °F; heart rate, 123 beats/min; blood pressure, 77/43 mm Hg; respiratory rate, 22 breaths/min; pulse oximetry reading, 95% on room air. He looks lethargic and is oriented only to self. His cardiopulmonary and neurologic exams are normal except for the tachycardia and altered mental status. He has a petechial rash that involves the ankles, wrists, and axilla, but not the palms or soles. The head CT scan is read as normal. After confirming an adequate platelet count, you perform a lumbar puncture and order intravenous antibiotics. After 3 liters of intravenous normal saline, repeat vital signs are as follows: blood pressure, 76/44 mm Hg; heart rate, 115 beats/min. The most likely explanation for his persistent hypotension is:

A. Acute myocardial infarction.
B. Bilateral adrenal hemorrhage.
C. Cardiac tamponade.
D. Gastrointestinal hemorrhage.
E. Pulmonary embolism.

36. A 35-year-old man who recently had a viral upper respiratory tract infection presents with sharp pleuritic chest pain that is worse when he is supine and improved when he sits forward. Vital signs: heart rate, 114 beats/min; blood pressure, 140/90 mm Hg; respiratory rate, 20 breaths/min; SaO₂, 95%. He is afebrile. On exam you hear a pericardial friction rub. An ECG demonstrates diffuse ST segment elevation with PR depression. His basic metabolic panel is within normal limits. The most appropriate therapy includes:

A. Aspirin, nitroglycerin, and morphine.
B. Thrombolytic therapy.
C. Morphine.
D. Nonsteroidal anti-inflammatory agents.
E. Antibiotics.

37. A 33-year-old man ingests lye in an attempt to commit suicide. In the ED, you see that he has obvious oral burns and hear stridor. After you secure his airway, you should:

A. Instill activated charcoal to absorb any remaining lye.
B. Place a nasogastric tube to decompress the stomach.
C. Neutralize the lye with a dilute solution of acid.
D. Arrange endoscopy to assess for perforation.
E. Administer empiric broad-spectrum antibiotics in case there is perforation.

38. What is the cerebral perfusion pressure if the mean arterial pressure is 75 mm Hg and the intracranial pressure is 18 mm Hg?

A. 93 mm Hg
B. 75 mm Hg
C. 57 mm Hg
D. 39 mm Hg
E. 18 mm Hg
39. A 17-year-old football player collapsed at practice 1 hour ago. He was initially confused but is now awake with normal mental status. Vital signs: oral temperature, 100.6°F; heart rate, 98 beats/min; respiratory rate, 22 breaths/min; blood pressure, 102/55 mm Hg. His physical examination, including neurologic evaluation, is completely normal. Complete blood count, basic metabolic panel, and liver function studies are normal but his total creatinine phosphokinase is 5,500 IU/L.

Your next order is:

A. Administer intravenous normal saline at a rate to produce a urine output of 2 to 3 ml/kg/hour.
B. Administer 1 gram/kg of intravenous mannitol and consult the nephrologist for emergent dialysis.
C. Administer 1 gram/kg of oral sodium polystyrene sulfonate for impending hyperkalemia.
D. Alkalinize the urine using intravenous sodium bicarbonate to maintain the urine pH at 7.0.
E. Confirm the diagnosis by sending a urine sample for myoglobin testing.

40. A 50-year-old woman has a temperature of 104°F. She is oriented to person and place only and follows commands inconsistently. Her exam is notable for rigidity in all four extremities. She takes fluphenazine decanoate for schizophrenia. The serum laboratory value most consistent with this clinical picture is:

A. Elevated thyroid-stimulating hormone (TSH).
B. Elevated platelets.
C. Elevated creatine kinase (CK).
D. Decreased white blood cell (WBC) count.
E. Decreased potassium.

41. A 15-year-old male presents with a 4-hour history of right lower abdominal and groin pain associated with vomiting. His right testicle is exquisitely tender, riding high in the scrotum, and demonstrates an epididymal head that is palpable anteriorly. Your next step is to:

A. Obtain immediate urologic consultation and attempt manual testicular detorsion.
B. Consult urology after a color Doppler testicular ultrasound is performed.
C. Order a technetium nuclear scan.
D. Await the results of urinalysis and a complete blood count.
E. Send a urinalysis and culture and treat the patient for epididymitis.

42. A 30-year-old man complains of weakness, which started in his legs but has moved to his arms over the past few days. He now has a hard time walking. On his exam, you find moderate weakness that is definitely worse in his lower extremities; he is also ataxic and has decreased deep tendon reflexes. You also note an *Ixodes* tick on his back. The most important part of his management is to:

A. Perform a head CT.
B. Remove the tick.
C. Perform a brain MRI.
D. Request a neurology consult.
E. Perform a lumbar puncture.
43. A fire rescue crew brings a 55-year-old man with mental status changes to the emergency department. The medics tell you his fingerstick glucose measurement was 30 mg/dL. You give 1 ampule of D50 with a subsequent increase in his glucose to 92 mg/dL and mental status improvement. The patient is a diabetic and admits that he has been depressed lately. His medications include extended-release metoprolol, glyburide, and aspirin. His physical examination shows normal vital signs and no significant abnormalities. After 1 hour in the emergency department, he is again confused; a repeat fingerstick glucose measurement is 38 mg/dL. You should now consider treatment with:
   A. Intramuscular glucagon.
   B. Intravenous glucose.
   C. Normal saline.
   D. Octreotide.
   E. Oral glucose.

44. A 69-year-old man with a lower GI bleed has a hemoglobin of 6 g/dL. You order the patient to be transfused with 2 units of packed red blood cells. Thirty minutes into the transfusion, the nurse notes that the patient's temperature has risen to 38.6°C. You should now:
   A. Stop the transfusion.
   B. Continue the transfusion at a slower rate.
   C. Speed up the transfusion so it can finish more quickly.
   D. Change the filter in the intravenous tubing and resume the transfusion.
   E. Change the intravenous tubing.

45. A 30-year-old man tells you he took a “bottle of acetaminophen” 12 hours ago to try to kill himself. Vital signs and physical examination are normal. You should:
   A. Obtain a 12-hour acetaminophen level and start intravenous N-acetylcysteine only if the level is greater than 150 mg/dL.
   B. Obtain a 12-hour acetaminophen level and start intravenous N-acetylcysteine if the level is greater than 37.5 mg/dL.
   C. Obtain a 12-hour acetaminophen level and liver function tests, and start intravenous N-acetylcysteine only if acetaminophen and transaminitis are present.
   D. Start intravenous N-acetylcysteine prior to obtaining an acetaminophen level or laboratory values.
   E. Give the patient activated charcoal and call a transplant center for transfer.

46. A 23-year-old woman who began her last menstrual period 8 weeks ago now complains of pelvic pain and vaginal bleeding. Vital signs: blood pressure, 80/60 mm Hg; heart rate, 135 beats/min. A bedside pregnancy test is positive. The most appropriate immediate intervention is:
   A. Consult OB/GYN immediately.
   B. Order a complete blood count and quantitative HCG.
   C. Send the patient to radiology for a pelvic ultrasound.
   D. Order a type and screen in case the patient needs blood products.
   E. Infuse 2 liters of normal saline and consult OB/GYN if her vital signs do not stabilize.
47. A 33-year-old woman complains of left leg pain. She tells you that it started about 5 hours ago just above her knee, but it has now spread to her entire thigh. She denies any trauma. She tells you that she feels warm and is a “little lightheaded.” She has no significant medical history, takes no medications, and has no allergies. She is employed as a nursing technician at a local nursing home. Vital signs: temperature, 101.1°F; blood pressure, 109/67 mm Hg; heart rate, 113 beats/min; respiratory rate, 22 breaths/min. Her left leg is red just proximal to the knee; she has full range of motion and you can find no effusion. Her proximal thigh is edematous and very tender to minimal palpation but is not erythematous. There is no crepitance, serosanguinous discharge, or bullae. An x-ray film is negative for acute fracture or gas. The most important step in the ED management of this patient is:
   A. Analgesics.
   B. Broad-spectrum antibiotics.
   C. Intravenous fluids.
   D. Emergent surgical consultation.
   E. Tetanus toxoid.

48. A 76-year-old man had sudden onset of left face, arm, and leg weakness 1 hour prior to arrival. Your examination reveals left hemiplegia. His only significant history is of hypertension and a bleeding ulcer, for which he was discharged from the hospital 2 weeks ago. A CT scan done 30 minutes after arrival shows early changes consistent with an ischemic right temporal stroke. Blood pressure at this time is 170/88 mm Hg and his neurologic exam is unchanged. The appropriate course of treatment is:
   A. Intravenous tPA
   B. Supportive care
   C. Intravenous heparin
   D. Nitroprusside to lower blood pressure
   E. Emergent MRI

49. A 3-year-old girl has a chief complaint of fever and ear pain. Her father tells you that she has had frequent ear infections. On exam you note an ill-appearing girl whose left ear is sticking out from her head. She has exquisite pain when you apply traction to the auricle, and the area behind the ear is quite red. You suspect mastoiditis. To confirm your diagnosis, the imaging modality of choice is:
   A. None.
   B. MRI.
   C. CT scan.
   D. Ultrasound.
   E. Lateral radiographs of the skull.

50. A 45-year-old woman was clearing brush around her home a few days ago. Yesterday she noticed a rash with lines of macules and pustules that initially appeared on both forearms and hands. Today she complains of significant eyelid and facial edema. You begin treatment with:
   A. Prednisone, 50 mg daily for 5 days.
   B. Topical calamine lotion and diphenhydramine, 50 mg orally every 6 hours as needed.
   C. Beclomethasone, 1% cream applied twice daily until the rash clears.
   D. Hospital admission, initiating an intravenous third-generation cephalosporin.
   E. A 2-week prednisone taper.
51. A 42-year-old G2P1 woman at 9 weeks estimated gestational age complains of 6 days of left leg swelling and calf pain, mild intermittent chest pain, and worsening breathlessness with exertion. She recently flew back from Japan, where she had gone on a business trip. She denies significant past medical problems. Vital signs: heart rate, 115 beats/min; respiratory rate, 28 breaths/min; blood pressure, 110/70 mm Hg; temperature, 97.8°F; SaO₂, 93%. She is in no acute distress but has a swollen left leg up to her knee. There is no warmth and minimal erythema, and there are no palpable cords. You suspect that she has deep vein thrombosis. The most appropriate diagnostic study to perform is:
   A. D-dimer testing.
   B. V/Q scan.
   C. CT pulmonary angiogram.
   D. Left lower extremity Doppler ultrasonography.
   E. Venography.

52. A 43-year-old man with a history of asthma and diabetes complains of a “regular” headache, generalized weakness, fatigue, dyspnea on exertion, and a mild, occasionally productive, cough for the last month. He used up his last albuterol MDI but did not get relief from his symptoms, which include intermittent sweats and chills. He denies leg swelling, and he says, “I must be eating less. I noticed my pants are looser than usual.” He is employed as a corrections officer in the local jail. He smoked a pack a day for 10 years before quitting but does not use drugs or alcohol. Vital signs: temperature, 99.1°F; heart rate, 106 beats/min; respiratory rate, 20 breaths/min; SaO₂, 95% on room air. His fingerstick glucose concentration is 120 mg/dL. A chest film shows a right middle lobe infiltrate, which corresponds to the crackles heard on his lung exam, but he was not wheezing. Optimal management would be to:
   A. Discharge the patient with a prescription for antibiotics.
   B. Order nebulizer treatments and steroids and write a new prescription for his MDI.
   C. Order antibiotics and a CT scan of the head, and perform a lumbar puncture.
   D. Initiate respiratory isolation, order antibiotics, and admit the patient.
   E. Explain to the patient that he does not need antibiotics for this viral syndrome.

53. A 15-month-old boy has had 2 days of lethargy and non-bilious vomiting. There is no history of fevers or diarrhea, although his mother states that he appears to have episodes of crying and drawing his knees up to his chest. He is afebrile and a well-hydrated infant with no nuchal rigidity. His abdomen is not tender. The rectal examination shows brown stool that is positive for occult blood. The most likely diagnosis is:
   A. Meningitis.
   B. Bacterial gastroenteritis.
   C. Appendicitis.
   D. Intussusception.
   E. Malrotation with mid-gut volvulus.

54. A 16-year-old boy complains of sore throat. Your lab does not offer a rapid test for group A beta-hemolytic streptococcus (GAS). You know that antibiotics will not help a patient with a viral infection but may offer some benefit in a patient with GAS pharyngitis. Your colleague suggests you use the CDC (Centor) criteria to help determine whether this is a streptococcal infection. These criteria are:
   A. Absence of cough, palatal petechiae, tonsillar exudates, and tender anterior cervical lymphadenopathy.
   B. Tonsillar exudates, tender anterior cervical lymphadenopathy, absence of rhinorrhea, and palatal petechiae.
   C. Absence of cough, history of fever, palatal petechiae, and strawberry tongue.
   D. History of fever, palatal petechiae, strawberry tongue, and submandibular lymphadenopathy.
   E. Tonsillar exudates, tender anterior cervical lymphadenopathy, absence of cough, and history of fever.
55. The patient in Question 54 returns 10 days after you treat him with an intramuscular penicillin injection. He now complains of worse pain and difficulty swallowing his saliva. He has a characteristic “hot potato” muffled voice. When you examine his oropharynx, you note asymmetry, with the uvula deviated to the right. You strongly suspect a peritonsilar abscess (PTA). Choose the true statement:
   A. PTA is the second most common deep-space infection of the head and neck.
   B. Absence of purulent return by needle aspiration excludes PTA and confirms the diagnosis of peritonsilar cellulitis.
   C. All patients with PTA must be admitted for IV antibiotics and surgical drainage.
   D. Most patients with PTA can be treated as an outpatient with needle aspiration, antibiotics, and oral analgesics.
   E. Intravenous penicillin is the drug of choice for PTA.

56. The medication that is first-line therapy in a seizing child in the ED is:
   A. Lorazepam.
   B. Phenobarbital.
   C. Phenytoin.
   D. Pyridoxine.
   E. Valproic acid.

57. A patient presents to the ED after sustaining a dental injury while biking. You do not suspect a head injury, but he is having dental hot/cold sensitivity and has evidence of enamel and dentin injury. The most appropriate management is:
   A. Nothing—no acute care is needed.
   B. Cover the dental fracture and obtain a dental referral within 24 hours.
   C. Obtain immediate dental referral.
   D. Arrange dental referral within 1 week.
   E. Order imaging for possible fracture.

58. A 25-year-old woman is ejected from the back of a motorcycle at high speed. She was not wearing a helmet. In addition to treating her multiple other injuries, you obtain a CT scan of the face. The radiologist’s CT read describes “a transmaxillary fracture that involves the pterygoid processes bilaterally... The orbits are intact bilaterally... also shows a comminuted nasal bone fracture.” The best description for this fracture is:
   A. LeFort I fracture.
   B. LeFort II fracture.
   C. LeFort III fracture.
   D. LeFort IV fracture.
   E. Non-LeFort type fracture.

59. A 55-year-old man has severe hematemesis. Vital signs: blood pressure, 95/50 mm Hg; heart rate, 110 beats/min. You find a jaundiced male with ascites, caput medusa, and palmar erythema. While the gastroenterologist is driving to the hospital, your initial therapy should include:
   A. Intravenous vasopressin infusion.
   B. Octreotide bolus and infusion.
   C. Intravenous antibiotics and ice water gastric lavage.
   D. Intravenous propranolol.
   E. Careful placement of a Sengstaken-Blakemore tube.
60. A 5-week-old has a 2-day history of tactile fevers without cough, vomiting, or diarrhea. She is otherwise feeding well and vigorous. She is not toxic in appearance and has a rectal temperature of 38.4°C. The most appropriate diagnostic workup is:
   A. No diagnostic laboratory tests are needed.
   B. Urinalysis and urine culture only.
   C. CBC and blood culture only.
   D. Urinalysis, urine culture, CBC, and blood culture only.
   E. Urinalysis, urine culture, CBC, blood culture, and cerebrospinal fluid studies.

61. A 56-year-old man has several large lacerations requiring wound repair. He weighs 100 kg. The maximum dose of lidocaine you can use is:
   A. 7 ml of 1:1,000 lidocaine.
   B. 70 ml of 1:10,000 lidocaine.
   C. 450 ml of 1:1,000 lidocaine.
   D. 4.5 ml of 1:1,000 lidocaine.
   E. 450 ml of 1:10,000 lidocaine.

62. A 35-year-old woman presents with exertional dyspnea and hemoptysis. Vital signs: heart rate, 90 beats/min; blood pressure, 140/80 mm Hg; respiratory rate, 18 breaths/min; SaO₂, 99%. She is afebrile. You hear a mid-diastolic rumble with an opening snap loudest just right of the apex. An ECG shows a biphasic p-wave with terminal negative deflection in V1. The most common cause of her symptoms is:
   A. Rheumatic fever.
   B. Congenital heart disease.
   C. Pulmonary embolism.
   D. Infective endocarditis.
   E. Aortic dissection.

63. You are the first responder to an explosion at a bus station. There are hundreds of people walking around the site. Some of them have obvious injuries and some of them just look dazed. You examine a patient with a small laceration on his arm. He should be put in triage category:
   A. Level 1 (Red)
   B. Level 2 (Yellow)
   C. Level 3 (Green)
   D. Level 4 (Black)
   E. Level 5 (Blue)

64. Regarding stingray injuries:
   A. Foreign bodies are rare.
   B. The wound is typically numb.
   C. Hot water is often therapeutic.
   D. Opiate analgesics are contraindicated.
   E. Use vinegar or salt water to remove the active nematocysts.
65. A 40 year-old diabetic woman is brought to the emergency department by family members for evaluation of altered mental status. She has not been taking her insulin because she says she can't afford it. Her fingerstick glucose reading at triage is “high.” She has been “sleeping a lot” and has not been eating or drinking the past 4 days. Vital signs: heart rate, 140 beats/min; blood pressure, 88/40 mm Hg; respiratory rate, 40 breaths/min; temperature, 99.8°F; SaO₂, 97% on room air. She appears to be severely volume depleted. Her cardiac and respiratory examination is normal except for tachycardia and tachypnea, but her abdominal examination shows diffuse tenderness. You draw blood and insert an intravenous line. You give 2 liters of normal saline in the first hour and start an intravenous insulin infusion at 8 units/hr for presumed diabetic ketoacidosis. About 30 minutes later, she has a cardiac arrest and cannot be resuscitated. The most likely cause of the cardiac arrest is:

A. Hyperglycemia.
B. Hyperkalemia.
C. Hypokalemia.
D. Hypernatremia.
E. Myocardial infarction.

66. A 22-year-old woman with sickle cell disease complains of chest pain, cough, and fever. She says that her pain feels like her usual vaso-occlusive crisis pain and requests meperidine. The initial management of her condition should include:

A. Intramuscular pain medication, oral hydration, and discharge to home.
B. Exchange transfusion.
C. Initiation of intravenous hydration, parenteral opioids, and empiric antibiotics.
D. Confirmation of the diagnosis of sickle cell disease by sickle prep and hemoglobin electrophoresis.
E. CT scanning of the chest.

67. The most common infective agent causing non-sexually transmitted bacterial epididymitis in men over age 35 years is:

A. *Chlamydia trachomatis*.
B. Coliform organisms or *Pseudomonas* species.
C. Diplococci.
D. Gram-negative coccobacilli.
E. Gram-positive cocci.

68. A 2-year-old girl swallowed one of grandma's glyburide tablets about 1 hour ago. She has no symptoms; her bedside glucose reading is 120 mg/dL. Vital signs are normal. You should:

A. Observe for 6 hours and, if asymptomatic, discharge home to follow-up with her pediatrician in the morning.
B. Feed the child a meal and discharge home, since one glyburide tablet is a therapeutic dose and will not cause any toxicity.
C. Admit for 24 hours of observation.
D. Admit for child protective services to determine safety of home environment.
E. Place the child on dextrose infusion and observe for any signs of hypoglycemia over the next 6 hours.
69. A 25-year-old man complains of chest pain after a night of intranasal cocaine use. Vitals signs: heart rate, 130 beats/min, blood pressure, 190/90 mm Hg. An ECG shows sinus tachycardia with ST elevation in leads V5-6 and reciprocal changes in leads II, III, and AVF. A potentially harmful therapy for this patient is:
   A. Thrombolytics.
   B. Phentolamine.
   C. Heparin.
   D. Propranolol.
   E. Nitroglycerin.

70. A 56-year-old man complains of severe low back pain, buttock numbness, and lower extremity numbness and weakness for the last 6 hours. You are worried that he may have cauda equina syndrome. The element in the history or physical that would worry you most is:
   A. Fecal incontinence.
   B. Lower extremity motor deficits.
   C. Lower extremity sensory deficits.
   D. Saddle anesthesia.
   E. Urinary retention.

71. A 5-year-old boy fell on his outstretched hand and is complaining of elbow pain. The most likely fracture is:
   A. Radial head.
   B. Olecranon.
   C. Supracondylar.
   D. Lateral condyle.
   E. Medial condyle.

72. You and the on-call ophthalmologist are discussing a patient who was assaulted about the face. After describing her extraocular and visual findings, you mention that the patient has a hyphema that occupies one third to one half of her anterior chamber. Which grade hyphema is this?
   A. Grade 1
   B. Grade 2
   C. Grade 3
   D. Grade 4
   E. Grade 5

73. An 84-year-old woman has an acute ischemic stroke. She presents within 30 minutes after symptom onset; a head CT scan shows only atrophy. She has no contraindication to fibrinolytic therapy, as described in the National Institute of Neurologic Disorders and Stroke criteria. She weighs 50 kg, so you choose to treat her with rtPA using a:
   A. 10-mg bolus, 50-mg infusion over 30 minutes
   B. 10-mg bolus, 90-mg infusion over 60 minutes
   C. 100-mg bolus
   D. 4.5-mg bolus, 40.5-mg infusion over 60 minutes
   E. 50-mg bolus
74. A surgical resident is sent to the ED after she is accidentally stuck with a hollow needle in the operating room. Previous hepatitis serologies done on the source patient showed him to be HbsAg positive, HBeAg positive, and HbsAB negative. The resident’s immunizations are up to date, but recent titers showed her to be a non-responder to the hepatitis B vaccine. Appropriate treatment should involve:

A. Reassurance only.
B. Hepatitis B immunoglobulin (HBIG) only.
C. Revaccination against hepatitis B.
D. Revaccination and HBIG.
E. Interferon therapy.

75. A 23-year-old woman underwent cesarean section 1 week ago and now complains of worsening abdominal pain and fevers to 101°F. She has no urinary symptoms. A brown vaginal discharge has been unchanged since her c-section. Vital signs: heart rate, 120 beats/min; temperature, 101.4°F. She has suprapubic tenderness without guarding or peritoneal signs. There is no sign of infection at the incision site. On pelvic examination, you note uterine tenderness and purulent discharge from her cervix. Her white blood count is 13,000/ml with a left shift; urinalysis gives normal results, except for 5 to 10 white blood cells with 5 to 10 epithelial cells. Her pregnancy test is positive. The next most appropriate step is:

A. Order a pelvic ultrasound, administer intravenous antibiotics, and admit her to the obstetric service.
B. Treat her with intravenous antibiotics for pelvic inflammatory disease and then discharge her home on oral antibiotics with outpatient follow-up.
C. Give her 2 grams of azithromycin and discharge her home.
D. Give her ceftriaxone, 125 mg IM, and then discharge her home on 10 days of oral doxycycline.
E. Provide analgesia and discharge her home with supportive care and primary care follow-up.

76. A 65-year-old man with diabetes complains of experiencing severe groin pain for the last 24 hours. Vital signs: temperature, 98.6°F; heart rate, 95 beats/min; respiratory rate, 18 breaths/min; blood pressure, 145/90 mm Hg. He looks anxious and uncomfortable. On physical examination, you find erythema and tenderness of his scrotum and groin with minimal swelling. Your plan is to:

A. Write a prescription for a topical antifungal.
B. Initiate broad-spectrum antibiotics and consult urology.
C. Observe him in the ED until morning.
D. Start a 2-week oral prednisone taper and arrange follow-up with his primary doctor within the next 2 days.
E. Consult dermatology.

77. A 42-year-old immigrant from Cambodia complains of having fever, cough, and shortness of breath for about 4 weeks. He has been having night sweats and has lost about 10 kg over the past 2 months. A chest film shows a right upper lobe infiltrate. You admit the patient to a respiratory isolation room with the presumed diagnosis of tuberculosis. He is started on appropriate medications for tuberculosis and discharged home after 6 days. Two weeks later, he returns to the emergency department, reporting blurred vision and difficulty differentiating colors. The medication most likely to be causing his symptoms is:

A. Ethambutol.
B. Isoniazid.
C. Pyrazinamide.
D. Rifampin.
E. Streptomycin.
78. 55-year-old man with a history of hypertension and smoking complains of crushing retrosternal chest pressure, shortness of breath, and sweating. Vital signs: heart rate, 120 beats/min; blood pressure, 160/90 mm Hg; respiratory rate, 20 breaths/min; SaO₂, 97%. He is afebrile. An ECG shows significant ST-segment elevation in leads V1 through V4. You want to give him a fibrinolytic agent, knowing that an absolute contraindication is:
   A. Major surgery 3 weeks ago.
   B. Active internal bleeding.
   C. Active peptic ulcer disease.
   D. History of chronic severe hypertension.
   E. Current use of anticoagulants.

79. A 53-year-old diabetic man has a severe sore throat that started today, after 2 days of rhinorrhea and a dry cough. His temperature is 102.1°F. He is anxious because he has never had a sore throat this severe, and he states that his voice sounds different than it usually does. He complains of dysphonia, dysphagia, and odynophagia, but he is not using accessory muscles and he does not have stridor. He is able to swallow but prefers not to. There is no swelling on external neck exam, but he does have tenderness when you move his hyoid laterally. His pharynx and tonsils are mildly erythematous but lack exudates or fluctuance. The first action for ideal management of this patient is:
   A. Penicillin and salt water gargles.
   B. Immediate cricothyroidotomy.
   C. Rapid-sequence endotracheal intubation.
   D. Fiberoptic nasopharyngeal laryngoscopy.
   E. Reassurance and analgesic medication.

80. A woman brings her 6-month-old boy to the emergency department, stating that he has been listless for the past 2 days. The baby’s vital signs are within normal limits. You note that he is lethargic and has bruises in various stages of healing. His mother denies any history of trauma. You strongly suspect your patient has been physically abused. While awaiting a CT scan of the patient’s head, you perform a thorough physical exam. The physical finding that most strongly suggests shaken baby syndrome is:
   A. Sunken fontanelles.
   B. Retinal hemorrhages.
   C. Perforated tympanic membrane.
   D. Palatal petechiae.
   E. Cotton wool spots.

81. A 19-year-old woman with a history of asthma complains of 2 days of runny nose and wheezing after running out of her albuterol inhaler. She is 32 weeks pregnant. Vital signs: heart rate, 120 beats/min; respiratory rate, 24 breaths/min; blood pressure, 105/60 mm Hg; temperature, 99.9°F; SaO₂, 94%. She is in mild respiratory distress with diffuse bilateral wheezing, poor aeration, and mild substernal retractions. The asthma therapy that is contraindicated because of her pregnancy is:
   A. Corticosteroids.
   B. Albuterol.
   C. Subcutaneous epinephrine.
   D. Subcutaneous terbutaline.
   E. None of the above.
82. After sustaining a direct blow to his anterior neck while playing basketball, a patient now has rapidly developing dysphonia, stridor, hoarseness, and dyspnea. Appropriate medical management should begin with:
   A. Intravenous steroids.
   B. Obtaining a soft tissue neck CT scan.
   C. Performing bronchoscopic intubation.
   D. Performing emergent cricothyroidotomy.
   E. Obtaining bedside plain films.

83. An 8-year-old boy has a closed angulated radius and ulna fracture requiring reduction. You choose to use intravenous ketamine as your sedation agent. You know that ketamine:
   A. Is not associated with hypersalivation.
   B. Has analgesic properties.
   C. Is associated with the loss of protective airway reflexes.
   D. Frequently produces unpleasant emergence reactions in children.
   E. Can be reversed by the administration of flumazenil.

84. A third-year medical student returns from Africa, where she did a rotation in tropical medicine. She complains of fatigue, vomiting, and diarrhea. You note that she is mildly icteric. The type of hepatitis she probably has:
   A. Often progresses to fulminant hepatic failure.
   B. Is spread via the parenteral route, so she must have been stuck by a needle in her lab.
   C. Can be effectively prevented with appropriate post-exposure prophylaxis.
   D. Could cause significant mortality if she was 8 months pregnant.
   E. Should be aggressively treated with interferon.

85. A patient with a hydrocele usually complains of:
   A. Dysuria, urgency, and frequency.
   B. Fever, chills, and nausea or vomiting.
   C. A painless, enlarged scrotum.
   D. Sudden urinary retention.
   E. A mass that increases when he lies down and decreases when he stands up.

86. A 51-year-old man complains of fever, malaise, shortness of breath, and a non-productive cough, which started abruptly about 24 hours ago. Over the past 4 hours, his shortness of breath is much worse and he has developed generalized abdominal pain. His medical history is significant for hypertension and hyperlipidemia, but he takes no medicines and has no allergies to medications. There is no history of travel; he works for a governmental agency in a distribution warehouse. On physical exam, he appears ill. Vital signs: temperature, 102.3°F; heart rate, 129 beats/min; blood pressure, 89/53 mm Hg; respiratory rate, 26 breaths/min; pulse oximetry reading, 91% on room air. He has diffuse rhonchi. You also find mild abdominal tenderness without localization or peritoneal signs. The remainder of his physical exam, including skin, is normal. You place the patient on supplemental oxygen and give intravenous fluids. A chest film shows an enlarged mediastinum with hilar lymphadenopathy but no definite infiltrative disease. The most appropriate antibiotic for this patient is:
   A. Azithromycin.
   B. Ceftriaxone.
   C. Ciprofloxacin.
   D. Erythromycin.
   E. Vancomycin.
87. A 55-year-old woman complains of coughing up blood. Over the past 2 weeks, she has had a low-grade fever, a non-productive cough, and some shortness of breath. Her primary care physician evaluated her and prescribed a 5-day course of azithromycin, but she felt no better. Earlier today she started coughing up blood-tinged sputum. She currently feels more short of breath. Her medical history is significant for hypertension and sarcoidosis. She is currently taking hydrochlorothiazide and prednisone. She has no allergies and is not a smoker. There is no recent travel or known infectious exposure. She reports that a PPD test done by her primary care physician 6 months ago was negative. On physical exam, she appears to be in no acute distress. Vital signs: blood pressure, 144/78 mm Hg; heart rate, 79 beats/min; respiratory rate, 18 breaths/min; temperature, 99.2°F; pulse oximetry reading, 95% on room air. Her physical exam is significant only for faint, bilateral wheezing. A chest film shows a small cavitary lesion in the left upper lobe, which was also noted on a chest film obtained 6 months earlier. The organism most likely responsible for the patient’s hemoptysis is:

A. Aspergillus.
B. Haemophilus influenza.
C. Mycoplasma pneumoniae.
D. Staphylococcus aureus.
E. Streptococcus pneumoniae.

88. A 30-year-old man with a history of Wolff-Parkinson-White syndrome (WPW) syndrome presents with an irregularly irregular wide complex rhythm. The medication most appropriate to treat him is:

A. Digoxin.
B. A calcium channel blocker.
C. Adenosine.
D. Procainamide.
E. A beta-blocker.

89. A 70-year-old man has a large hemorrhagic stroke. His blood pressure is 240/160 mm Hg, even after receiving supplemental oxygen and comfort measures in the 2 hours you have been caring for him. You should manage his blood pressure by giving him:

A. Oral labetalol.
B. Intravenous nitroprusside.
C. More time.
D. Sublingual nifedipine.
E. A transdermal clonidine patch.

90. A 27-year-old man tells you that he has a recurrent kidney stone. His vital signs and urinalysis are normal. When you are evaluating the patient, he appears uncomfortable and is writhing on the bed, but the nurses observe that he rests comfortably after you leave. He requests a specific pain medication with a specific dose. His triage note lists multiple allergies to non-narcotic analgesics. The personality disorder most frequently associated with his behavioral disorder is:

A. Antisocial.
B. Borderline.
C. Histrionic.
D. Schizoid.
E. Narcissistic.
91. A 32-year-old G3P2 woman at 37 weeks' estimated gestational age complains of painless vaginal bleeding and abdominal cramping, which she says is heavier than a normal menstrual period and seems to be increasing. She denies trauma. She is in no acute distress and her vital signs are normal. Your next step is to:
   A. Perform a sterile bimanual examination.
   B. Consult an obstetrician to perform a stat pelvic ultrasound.
   C. Perform a sterile speculum examination to look for evidence of amniotic fluid rupture.
   D. Admit her to labor and delivery for observation.
   E. Consult an obstetrician to perform a stat c-section.

92. A diagnostic criterion for Kawasaki disease (mucocutaneous lymph node syndrome) is:
   A. Leukocytosis.
   B. Conjunctivitis.
   C. Scaling groin rash.
   D. Preauricular adenopathy.
   E. Thrombocytopenia.

93. An EMS unit transports a patient with both blunt and penetrating neck injury. His exam findings are notable for subcutaneous air, active bleeding, dysphonia, no neurologic deficits, and an intact airway. There is a puncture wound just superolateral to the cricoid cartilage. The diagnostic approach to this stable patient would be:
   A. Endoscopy.
   B. Bronchoscopy.
   C. Angiography.
   D. Surgical exploration.
   E. Direct laryngoscopy.

94. While hiking in Pennsylvania, a young man was bitten by a snake. He came to your emergency department and brought the snake with him. The snake has bands with the color pattern yellow/black/red/black/yellow. Your most important action is to:
   A. Perform local wound care and reassure the patient.
   B. Administer lorazepam, 1 mg intravenously.
   C. Order a complete blood count, coagulation studies, and basic metabolic panel.
   D. Apply a tourniquet to the involved extremity.
   E. Administer antivenin.

95. A 27-year-old man complains of fever, chills, headache, and profound lethargy. He states these symptoms began approximately 5 days ago after he returned from a vacation to visit family in Nigeria. He denies prior similar symptoms. He has no medical conditions, takes no medications, and has no allergies. He is a non-smoker and drinks occasionally. His family history is significant for diabetes. Vital signs: temperature, 103.1°F; blood pressure, 112/77 mm Hg; heart rate, 95 beats/min; respiratory rate, 18 breaths/min; pulse oximetry reading, 98% on room air. His physical exam, including skin and neurologic exam, is normal. The study most likely to diagnose the patient's condition is:
   A. Blood cultures.
   B. Chest film.
   C. Complete blood count.
   D. Lumbar puncture.
   E. Thick and thin peripheral blood smears.
96. You pick up the chart of a patient with the chief complaint of “coughing up blood.” The patient is HIV positive and you are concerned about tuberculosis. You know that:
   A. In massive hemoptysis, placing a patient on his/her side with the lesion in a dependent position is harmful.
   B. Sputum that is non-bloody is extremely unlikely to be associated with TB.
   C. All radiographic findings of TB are in the apices of the lungs.
   D. In HIV-positive patients with active pulmonary TB, the chest film is more likely to be normal if the CD4 count is very low.
   E. When massive hemoptysis becomes fatal, the cause of death is usually exsanguination.

97. An 81-year-old woman is sent from a nursing home with symptoms of classic acute cholecystitis—right upper quadrant tenderness, fever, and vomiting. Emergency bedside ultrasound shows a positive sonographic Murphy’s sign, but no gallstones. Appropriate management would involve:
   A. Emergency surgery.
   B. Admission to the surgical service for a “cooling off” period on antibiotics.
   C. Oral antibiotics and discharge back to the nursing facility.
   D. CT scan of the abdomen to look for a stone impacted near the ileocecal valve.
   E. Mesenteric angiography.

98. The recommended treatment for Bell’s palsy is:
   A. Acyclovir, 400 mg five times per day for 10 days.
   B. Cold packs.
   C. Prednisone, 1 mg/kg/day for 5 days, and then tapered over the next 5 days, and acyclovir, 400 mg five times per day for 10 days.
   D. Prednisone, 1 mg/kg/day for 5 days, with or without taper.
   E. Surgical ablation.

99. An EMS unit transports an elderly demented nursing home patient to the emergency department for evaluation of new left-sided weakness. He has a percutaneous endoscopic gastrostomy (PEG) tube, which was placed for dysphagia related to a prior ischemic stroke, and he was given a bolus feeding just before transport. An EMT reports that while being transported supine, the patient vomited tube-feeding solution, and subsequently had an episode of coughing. He tells you, “I think he aspirated.” The patient is sitting up, alert and conversant. He is confused but follows most commands. He coughs occasionally. In addition to evaluating the chief complaint of new left-sided weakness, your response to this aspiration event is to order:
   A. Antibiotics to cover gram-negative bacteria only.
   B. Antibiotics to cover anaerobic bacteria only.
   C. Antibiotics to cover gram-negative bacteria and anaerobes.
   D. Supportive care only.
   E. Medications to permit intubation so you can protect his airway.
100. A 50-year-old woman with a history of ovarian cancer presents with pleuritic chest pain, dyspnea, hemoptysis, and swelling of her right leg. She recently traveled to China. Vital signs: heart rate, 130 beats/min; blood pressure, 120/90 mm Hg; respiratory rate, 24 breaths/min; SaO₂, 92%. She is afebrile. You strongly suspect she has a deep venous thrombosis with pulmonary embolism. Choose the correct statement regarding her suspected thromboembolism:

A. Being younger than 40 years of age is a risk factor for thromboembolism.
B. Beta-blocker therapy should be initiated.
C. History of prior thromboembolism is a risk factor for recurrent thromboembolism.
D. Results of arterial blood gas analysis will be diagnostic.
E. Hyperlipidemia is a risk factor for thromboembolism.

101. On your way home from an evening shift, you stop at the scene of a motor vehicle crash as paramedics are taking a patient out of the car. The patient becomes pulseless and apneic. You properly identify yourself to the crew as an emergency physician and you:

A. Intubate the patient.
B. Perform needle decompression of the chest.
C. Perform needle cricothyroidotomy.
D. Offer assistance.
E. Perform thoracotomy.

102. A 50-year-old man with a known history of alcoholism presents with altered mental status. Family members state that he has been on a recent binge and has not been eating food. He takes no medications and has no known medical problems. On physical examination, the patient is disheveled and in mild distress. Vital signs: heart rate, 120 beats/min; blood pressure, 130/78 mm Hg; respiratory rate, 28 breaths/min. The rest of the examination is unremarkable. Laboratory findings include a serum glucose concentration of 250 mg/dL and an anion gap of 22. Serum ketones are present. The most important treatment is:

A. 0.45% saline.
B. D5 normal saline.
C. Folate.
D. Insulin.
E. Normal saline.

103. A 30-year-old woman with a history of asthma, who required intubation during ED management 6 months ago, returns to your ED for a second visit. This is her fourth ED visit in the past year. She is on day 2 of a steroid course and has had no resolution of her symptoms, despite use of her albuterol inhaler every 4 hours around the clock. She is afebrile, wheezing, and in mild respiratory distress. Her chest film from this second visit is normal. After two nebulizer treatments in the ED, she tells you "I'm just not getting any better." You tell her:

A. "I'm going to extend your course of prednisone by 5 more days."
B. "I'm going to recommend you stay in the hospital."
C. "I'm going to prescribe an antibiotic."
D. "I'm going to recommend using your inhaler every 2 hours at home."
E. "I'm going to prescribe you a long-acting beta-agonist pill."
104. A restrained passenger involved in a rear-impact MVC presents to the ED complaining of lateral neck pain. Your exam is notable for a small hematoma, a bruit, and subtle unilateral ptosis. After you confirm your suspected diagnosis, appropriate management includes:
   A. Surgical repair.
   B. No intervention.
   C. Embolectomy.
   D. Anticoagulation.
   E. Intravascular thrombolysis.

105. The most consistent finding in patients with cauda equina syndrome is:
   A. Absent anal wink.
   B. Absent bulbocavernosus reflex.
   C. Low back pain.
   D. Urinary incontinence.
   E. Urinary retention.

106. A 19-year-old man complains of several weeks of worsening abdominal pain associated with low-grade fevers and diarrhea. He has also noted joint pain. You can palpate a tender mass in his right lower quadrant. This is likely his first presentation of:
   A. Ulcerative colitis.
   B. Crohn's disease.
   C. Chronic appendicitis.
   D. Meckel's diverticulum.
   E. *Giardia intestinalis* infection.

107. A 48 year-old diabetic man has had a painful, red, swollen right knee for 2 days, accompanied by fever. He denies trauma. You perform diagnostic arthrocentesis, knowing the finding most consistent with septic arthritis is:
   A. White blood cell count of 47,000/ml with 68% polymorphonuclear white blood cells.
   B. Needle-like crystals.
   C. Rhomboid crystals.
   D. Gram-positive cocci on gram stain.
   E. White blood cell count of 78,000/ml with 52% polymorphonuclear white blood cells.

108. A 55-year-old woman initially complained of a severe headache, which has now progressed to altered mental status. Vital signs: heart rate, 100 beats/min; blood pressure, 190/110 mm Hg; respiratory rate, 20 breaths/min; *SaO₂*, 97%. She is afebrile. Physical exam is significant for papilledema and somnolence. Her creatinine level is elevated at 3.0 mg/dL. Her head CT is negative for blood, mass, or shift. Her condition is best described as:
   A. Hypertensive headache.
   B. Hypertensive emergency.
   C. Transient hypertension.
   D. Meningitis.
   E. Thoracic dissection.
109. A 33-year-old man from North Carolina complains of having fevers, lethargy, headache, and muscle aches for about 48 hours. He has no significant medical or surgical history. He currently takes no medicines and has no allergies. He has no history of recent travel. He works at a local fast food restaurant and has not had any known contact with sick people. Vital signs: temperature, 100.9°F; heart rate, 102 beats/min; blood pressure, 122/88 mm Hg; respiratory rate, 18 breaths/min. He has no neck stiffness and his neurologic exam is normal. You find some pink, irregular macules on his wrists, palms, soles, and forearms. He tells you the rash was not present 6 hours ago. The most appropriate antibiotic for this patient is:
   A. Ampicillin.
   B. Ceftriaxone.
   C. Cephazolin.
   D. Tetracycline.
   E. Vancomycin.

110. A 1-week-old boy has been vomiting and not feeding well. He had an unremarkable birth and prenatal history. Vital signs: rectal temperature, 37.2°C; heart rate, 195 beats/min; blood pressure, 60/35 mm Hg; respiratory rate, 70 breaths/min; SaO₂, 97%. He is notably listless with dry mucus membranes, clear lungs, no cardiac murmur, and palpable femoral pulses. Blood tests show a normal CBC but he has a sodium concentration of 120 mEq/L, a potassium concentration of 6.2 mEq/L, and a glucose concentration of 35 mg/dL. You give an intravenous glucose bolus and normal saline bolus of 40 ml/kg, and the resultant blood pressure is 65/40 mm Hg. After starting empiric antibiotics, your next step is:
   A. Start intravenous epinephrine infusion.
   B. Administer phenobarbital, 20 mg/kg intravenously over 15 minutes.
   C. Obtain a stat bedside echocardiogram.
   D. Administer hydrocortisone, 25 mg intravenously.
   E. Start intravenous prostaglandin E infusion.

111. A trauma patient is being flown to your facility after being intubated at a community hospital. The patient was an ejected passenger in a head-on collision. His Glasgow Coma Scale score is 8. The flight team reports having increasing difficulty with bagging the patient, decreased breath sounds unilaterally, increasing tachycardia, and decreasing blood pressure. You advise them to:
   A. Increase the tidal volume.
   B. Perform needle decompression.
   C. Give a bolus of intravenous fluid.
   D. Increase PEEP.
   E. Fly faster but make no other changes at this time.

112. A 17-year-old boy fell off his skateboard and struck his face on the sidewalk. He hands you some tooth fragments as he complains of mouth pain. You note that his medial incisors are fractured obliquely and there is a small amount of blood at the fracture line. This describes:
   A. Ellis class I fracture.
   B. Ellis class II fracture.
   C. Ellis class III fracture.
   D. Ellis class IV fracture.
   E. Ellis class V fracture.
113. A 75-year-old man with a history of atrial fibrillation had a sudden onset of abdominal pain, diarrhea, and vomiting. He is ill appearing and complains of excruciating pain, but does not have peritoneal signs on examination. The therapy most likely to reduce the likelihood of his death is:
   A. Intravenous heparin.
   B. Emergent surgery.
   C. Intra-arterial papaverine.
   D. Emergent angiography.
   E. Rate control with digoxin.

114. A 62-year-old man with a history of hypertension, diabetes, and hyperlipidemia complains of retrosternal chest pressure associated with shortness of breath, diaphoresis, nausea, and vomiting. Vital signs: heart rate, 104 beats/min; blood pressure, 140/90 mm Hg; respiratory rate, 18 breaths/min; \( SaO_2 \), 98%. He is afebrile. When you listen to his lungs, they are clear. The patient has no jugular venous distention or lower extremity edema. An ECG shows ST-segment elevation in leads I, aVL, V5, and V6, which represents infarction of the:
   A. Anterior myocardium.
   B. Septal myocardium.
   C. Lateral myocardium.
   D. Posterior myocardium.
   E. Inferior myocardium.

115. A 15-year-old boy with Type A hemophilia fell off of a horse several hours ago. He denies any head injury, saying he landed on his buttocks and back. He complains of mild pain in his back and lower pelvic region. Your initial evaluation is negative for any significant trauma. You should now:
   A. Admit the patient for observation.
   B. Discharge him to home.
   C. Refer him to hematology for same-day evaluation.
   D. Initiate workup to ascertain the degree of severity of the hemophilia.
   E. Begin treatment with 25 units/kg of recombinant factor VIII.

116. A 55-year-old man took a lot of pills in an attempt to commit suicide. Paramedics report several bottles at the scene, but they did not bring any of them with them. The patient has vomited several times. Arterial blood gas analysis gives the following results: pH, 7.46; \( \text{paCO}_2 \), 20; \( \text{paO}_2 \), 60; \( \text{HCO}_3^- \), 17. The patient's wife states he is "not acting himself" and didn't recognize her. A chest film shows bilateral infiltrates. Your next most appropriate action is:
   A. Administer a dose of activated charcoal and start whole bowel irrigation.
   B. Administer intravenous N-acetylcysteine, as the patient has classic signs of massive acetaminophen overdose.
   C. Administer fomepizole and order measurement of serum osmolality.
   D. Administer deferoxamine and admit to ICU.
   E. Call nephrology and request stat hemodialysis.
117. A 62-year-old woman complains of vaginal bleeding. Her last period was 9 years ago. Her vital signs are normal and her physical examination is unremarkable except for mild bleeding from her cervical os. A complete blood count and coagulation studies are normal. You arrange close follow-up with the gynecology service because you are concerned she needs:
   A. Pelvic ultrasonography.
   B. Hysterectomy.
   C. A pap smear.
   D. Laparoscopic surgery.
   E. An endometrial biopsy.

118. Several patients arrive by EMS units from a multi-vehicle injury accident. The first patient has no signs of external trauma and is complaining of middle to upper thoracic back pain. His chest radiograph is normal, but his femoral pulses seem somewhat diminished despite being hypertensive. This scenario causes concern for:
   A. Pelvic fracture.
   B. Musculoskeletal back pain.
   C. Acute dissection.
   D. Aortic rupture.
   E. Cardiac contusion.

119. A 72-year-old obese man with a history of hypertension had an acute onset of right flank pain about 1 hour prior to arrival. His blood pressure is 80/50 mm Hg and his heart rate is 120 beats/min. You suspect he may have a ruptured aortic aneurysm. You would maximize bedside ultrasound imaging by:
   A. Using a high-frequency ultrasound probe in a patient with copious bowel gas.
   B. Using a low-frequency ultrasound probe in a patient with copious bowel gas.
   C. Using a high-frequency ultrasound probe in a patient with minimal bowel gas.
   D. Using a low-frequency ultrasound probe in a patient with minimal bowel gas.
   E. Going straight to computerized tomography, as the aorta is poorly imaged with ultrasound no matter the probe used or patient’s condition.

120. A 50-year-old man has developed Clostridium difficile diarrhea after being treated with clindamycin for a dental infection. The best treatment will be:
   A. Intravenous hydration and control of diarrhea with diphenoxylate/atropine.
   B. Oral metronidazole.
   C. Intravenous vancomycin.
   D. Surgical colectomy.
   E. Watchful waiting.
121. A 49-year-old male smoker complains of a dry cough. He works as a plumber, is HIV negative, and does not drink alcohol. His wife tells you that he has had a cough and diarrhea for the past week, and also reports he has been somewhat confused the past few days. Vitals signs: temperature, 103°F; heart rate, 120 beats/min; respiratory rate, 26 breaths/min; blood pressure, 136/80 mm Hg; SaO₂, 88% on room air. He looks dehydrated. A chest film confirms an infiltrate. In addition to giving antibiotics to cover the usual etiologic agents of community-acquired pneumonia (i.e., pneumococci, *H. influenzae*), you should also choose an antibiotic that will treat:

A. Legionella.
B. *Pneumocystis jiroveci*.
C. *Klebsiella pneumoniae*.
D. Varicella zoster.
E. *Pseudomonas aeruginosa*.

122. Guillain-Barré syndrome is the most common cause of demyelinating polyneuropathies. It is usually recognized through the findings of progressive symmetric:

A. Distal motor weakness and hyperreflexia.
B. Lower extremity weakness more than upper extremity weakness and hyperreflexia.
C. Proximal and distal musculature weakness worse in the lower extremities and hyporeflexia.
D. Proximal motor weakness and hyperreflexia.
E. Upper extremity weakness more than lower extremity weakness and hyporeflexia.

123. A 35-year-old man with a history of hypertension, diabetes, and hyperlipidemia; a strong family history of MI; and a 30-pack-year history of tobacco use presents with crushing retrosternal chest pressure accompanied by shortness of breath and diaphoresis, which began 15 minutes prior to presentation. He is pale and diaphoretic. Vital signs: heart rate, 80 beats/min; blood pressure, 130/80 mm Hg; respiratory rate, 16 breaths/min; SaO₂, 99%. He is afebrile. An ECG shows normal sinus rhythm and no acute injury pattern. His first set of cardiac biomarkers, drawn on admission to the emergency department, is negative. A chest radiograph is normal. You call the admitting team to admit the patient, but they refuse “because he is too young to be having acute coronary syndrome and, besides, his troponin is negative.” Your next best course of action is to:

A. Discharge the patient to home.
B. Wait for shift change and sign the patient out to one of your colleagues.
C. Insist that for the safety of the patient he must be admitted for evaluation of acute coronary syndrome despite the initial negative workup.
D. Initiate a workup for pulmonary embolism with a d-dimer and, if positive, a chest CT/pulmonary angiogram.
E. Initiate antibiotics for pneumonia and discharge to home.

124. A 28-year-old intoxicated man was an unrestrained passenger who was ejected from an automobile after it crashed with a stationary object. You notice blood at his urethral meatus and a perineal hematoma. His cystourethrogram shows a normal urethra and a flame-like density lateral to the expected location of the bladder, but is otherwise normal. This finding probably represents:

A. Complete urethral injury.
B. Partial urethral injury.
C. Intraperitoneal bladder injury.
D. Extraperitoneal bladder injury.
E. A normal variant.
125. Given a stable patient with a suspected myocardial contusion, what is the best management approach?
   A. Observation and monitoring
   B. Echocardiography
   C. Serial biomarkers
   D. Discharge
   E. Serial radiographs

126. You are told by EMS that you will be receiving a patient who was exposed to an irritating gas that smelled like moldy or newly mown hay. When he arrives, he has tearing of both eyes, is gagging and coughing intermittently, and is wheezing loudly but is not currently in subjective respiratory distress. Because you recognize this as a phosgene exposure, you know your management will include:
   A. Observation for 24 hours.
   B. Nebulized bronchodilators prior to discharge, then PRN at home.
   C. 80% inhaled nitric oxide (INO).
   D. Nebulized methylene blue.
   E. Nebulized lidocaine prior to discharge.

127. A 36-year-old man complains of retrosternal chest pain beginning 1 hour after smoking crack cocaine. He is afebrile, with a heart rate of 113 beats/min, blood pressure of 140/90 mm Hg, respiratory rate of 18 breaths/min, and SaO₂ of 98% on 2 liters via nasal cannula. An ECG shows nonspecific S-T and T wave changes. He looks anxious and is diaphoretic and tachycardic. His lungs are clear. His stool is guaiac negative. A chest film is normal. The treatment that is contraindicated in this patient is:
   A. Aspirin.
   B. Sublingual nitroglycerin.
   C. Heparin.
   D. Metoprolol.
   E. Morphine sulfate.

128. A 35-year-old woman complains of diffuse abdominal pain and nausea. She denies vomiting, fever, or change in urinary or bowel habits. Earlier today a dark-colored spider bit her on the ankle. Vital signs: temperature, 37.5°C; heart rate, 95 beats/min; respiratory rate, 18 breaths/min; blood pressure, 165/95 mm Hg. Her abdomen is rigid and diffusely tender. You should now:
   A. Give benzodiazepines and seek black widow antivenin.
   B. Discharge her home with oral pain medications as long as she is not pregnant.
   C. Consult general surgery and prepare her for the operating room.
   D. Administer intravenous labetalol for her hypertension.
   E. Administer broad-spectrum antibiotics and corticosteroids.
129. A 27-year-old woman complains of frequent nosebleeds. You notice petechiae on her arm where the blood pressure cuff was inflated, and the patient mentions that she had heavier than usual menses last week. She denies fevers or headaches. She had taken trimethoprim/sulfamethoxazole for a urinary tract infection. A complete blood count shows a platelet count of 4,000/μL. The most appropriate management for this condition is:
   A. Platelet transfusion.
   B. Intravenous immunoglobulin (IVIG).
   C. Outpatient hematology evaluation.
   D. Admission for observation and supportive care.
   E. Plasma exchange with fresh frozen plasma (FFP).

130. A 29-year-old cowboy complains of lower abdomen and leg pain, which occurred when a bull fell on him during a local rodeo. Vital signs (after 2 liters of crystalloid and 4 units of PRBCs): heart rate, 118 beats/min; respiratory rate, 20 breaths/min; blood pressure, 88/54 mm Hg. He has no signs of head or chest trauma and his Focused Abdominal Sonogram for Trauma (FAST) exam is normal. His pelvis is unstable on the right and you have applied a Hare-type traction splint for a closed right femur fracture. The only finding on abdominopelvic CT scan is an open-book/vertical shear pelvic injury on the right with associated hematoma. You should now:
   A. Apply an external fixation belt and continue resuscitation in the ICU.
   B. Consult a general surgeon for immediate exploratory laparotomy.
   C. Consult an interventional radiologist for arterial embolization.
   D. Consult an orthopedic surgeon to apply an external fixation device.
   E. Consult an orthopedic surgeon for open reduction-internal fixation.

131. A 75-year-old man complains of weakness. Vital signs: heart rate, 45 beats/min; blood pressure, 110/50 mm Hg. An ECG shows a Type II second-degree heart block. His medications include diltiazem, digoxin, furosemide, and warfarin. You want to avoid giving this patient:
   A. Glucagon.
   B. Atropine.
   C. Digoxin fab.
   D. Calcium.
   E. Dopamine.

132. A 26-year-old obese G1P0 at 36 weeks’ estimated gestational age complains that her hands and feet have become edematous over the past 1 to 2 weeks. She just arrived in the United States from Mexico to live with her relatives, who are going to help her take care of her newborn. She has received no prenatal care. Her medical history is unremarkable. Vitals signs: blood pressure, 160/110 mm Hg; otherwise normal. Fetal heart rate is 144 beats/min. A comprehensive laboratory evaluation is normal, except for 4+ proteinuria on a urine dipstick. The definitive treatment for her condition is:
   A. Delivery.
   B. Labetalol.
   C. Magnesium.
   D. Hydralazine.
   E. Diuretic therapy.
133. A 44-year-old woman complains of left facial droop and decreased sensation along her entire left face, which she noted on awakening this morning. She also remembers a peculiar rash under her arm about 1 month ago that resolved without treatment. She denies any relevant medical history. She takes over-the-counter NSAIDs for occasional myalgia. There is no family history of early stroke or atherosclerotic vascular disease. She is afebrile, and her blood pressure is 155/78 mm Hg, her heart rate is 88 beats/min, and her respiratory rate is 15 breaths/min. She has a left facial droop that involves both the forehead and lower face. The remainder of her exam, including skin exam, is normal. Of the abnormalities listed, the one most likely to be found on her electrocardiogram is:

A. Atrial fibrillation.
B. First-degree atrioventricular block.
C. Left bundle branch block.
D. Right bundle branch block.
E. Sinus tachycardia.

134. A 4-year-old girl has high fevers and diarrhea. As you are examining her, she seizes. The likely cause of her diarrhea is:

A. Campylobacter.
B. Shigella.
C. Yersinia.
D. Staphylococcus aureus toxin.
E. Enterohemorrhagic E. coli.

135. A patient arrives at the ED in cardiac arrest. You notice a dialysis shunt in the left arm and a Medic Alert bracelet reading, “Renal Failure: Dialysis Patient.” In addition to the standard ACLS medications, you should give an early dose of:

A. Calcium.
B. Diphenhydramine.
C. Magnesium.
D. Mannitol.
E. Phosphorus.

136. EMS brings you a 60-year-old woman who is confused. She takes atenolol, levothyroxine, and atorvastatin. According to family members, she has had a cough and fever for a week. She is disheveled and confused. Vital signs: heart rate, 40 beats/min; blood pressure, 80/40 mm Hg; rectal temperature, 85°F. Cardiac examination shows bradycardia without murmurs. Lung examination is significant for rales at the left base. Deep tendon reflexes are absent. You decide to initiate therapy. The most crucial medication to start in the ED is:

A. Decadron.
B. Hydrocortisone.
C. Hypertonic saline.
D. Solumedrol.
E. Thyroxine.
137. A mother brings you her 4-year-old boy after she found him in the bathroom with an open bottle of drain cleaner. He has vomited several times and now appears to have stridor. Vital signs: heart rate, 120 beats/min; respiratory rate, 30 breaths/min; blood pressure, 100/70 mm Hg; SaO₂, 98% on room air. You should now:

A. Call a gastroenterologist for endoscopic evaluation of the esophagus.
B. Call an otolaryngologist for evaluation of upper airway edema and possible early intubation.
C. Administer a dose of activated charcoal.
D. Administer a dose of steroids for prevention of strictures.
E. Administer intravenous antibiotics for prophylaxis against mediastinitis and admit to the PICU.

138. A 50-year-old man complains of a sudden tearing chest pain, which started about 45 minutes ago and radiates to his back. Vital signs: heart rate, 130 beats/min; blood pressure, 190/110 mm Hg; respiratory rate, 18 breaths/min; SaO₂, 99%. On physical examination, you hear a high-pitched blowing diastolic murmur immediately after S2 near the right upper sternal border. The initial ECG shows only nonspecific ST-T wave changes. A chest radiograph is pending, but based on this information you decide to administer:

A. 325 mg of aspirin by mouth; sublingual nitroglycerin, 0.4 mg; and, if he is not pain free after 3 doses, begin a nitroglycerin drip.
B. Unfractionated heparin, 60 IU/kg (maximum 4,000 units), as an intravenous bolus followed by 12 IU/kg/hr (maximum 1,000 units/hr).
C. Intravenous hydralazine to control blood pressure.
D. An intravenous fibrinolytic agent.
E. Intravenous esmolol and sodium nitroprusside.

139. Choose the true statement concerning pyloric stenosis.

A. It occurs more frequently in first-born females.
B. The usual onset of symptoms is during the third to fifth week of life.
C. Electrolyte abnormalities include hyperchloremic hypokalemia.
D. Vomiting is projectile and bilious.
E. Plain radiographs are the study of choice.

140. Which statement is true concerning the administration of edrophonium, used to help differentiate myasthenic crisis from cholinergic crisis?

A. A maximum dose of 5 mg can be given intravenously.
B. An intravenous test dose of 1 to 2 mg followed by muscle fasciculations and respiratory depression demonstrates an allergic reaction, so further edrophonium administration is contraindicated.
C. Dramatic improvement of symptoms within 90 seconds is proof of cholinergic crisis.
D. The test is “graded” by measuring the distance between the upper eyelid and lower eyelid in the most severely affected eye before and after intravenous edrophonium.
E. Edrophonium is a short-acting acetylcholinesterase stimulator.
141. A patient with blunt trauma arrives after transfer from an outside ED about 8 hours after a vehicular crash. The patient is intubated, has nearly complete whiteout of her left lung field, has a widened A-a gradient, and has serial worsening blood gases despite adjustments in oxygen, PEEP, and volumes. What type of positioning may be of value to improve oxygenation?
   A. Prone.
   B. Reverse Trendelenburg.
   C. Normal lung up.
   D. Normal lung down.
   E. Trendelenburg.

142. A family of four presents with large-volume, watery stools several hours after eating potato salad at an outdoor picnic. Which of the following is true?
   A. Antibiotics are useful to rapidly kill the responsible organism.
   B. This family's symptoms will likely last 1 or 2 weeks.
   C. There is a high likelihood of the children developing hemolytic uremic syndrome.
   D. Symptoms are caused by ingestion of a pre-formed toxin.
   E. Antibiotic use may increase the risk of developing a carrier state.

143. A paramedic from the local EMS agency contacts you by radio in the ED to give a patient report. The patient was a restrained driver who lost control of his vehicle and struck a tree. The patient has no obvious injuries but claims amnesia for the event. His vitals are stable, with a blood pressure of 130/80 mm Hg and a heart rate of 78 beats/min. There is an odor of alcohol on his breath. He states that he does not want to be taken to the hospital and that he wants to go home. The paramedic asks what they should do. You advise them to:
   A. Allow the patient to refuse care.
   B. Contact a friend or family member to pick up the patient.
   C. Encourage the patient to seek medical treatment on his own.
   D. Contact local law enforcement for assistance in transporting the patient.
   E. Transport the patient to his home.

144. Which of the following best describes the antiarrhythmic classification of lidocaine?
   A. Class 1A
   B. Class 1B
   C. Class 2
   D. Class 3
   E. Class 4
145. A 55-year-old man with a history of hypertension, cardiomyopathy, and chronic ethanol abuse has altered mental status. His wife reports that he became confused and “unsteady on his feet” that morning. Vital signs are normal. Bedside finger-stick glucose reading is 129. The patient is confused, with a GCS score of 14. His HEENT exam is significant for nystagmus and bilateral lateral rectus palsy. His gait is ataxic, and he has dysdiakinesia. There is no other focal neurologic deficit. A CT scan of the head shows no acute intracranial abnormality. Routine labs are significant for macrocytic anemia and hemoglobin of 9.8 mg/dL. The nutritional problem most likely causing his symptoms is deficiency of:
   A. Niacin.
   B. Cobalamin.
   C. Thiamine.
   D. Iron.
   E. Folate.

146. A patient sustained a direct blunt force to the top of his shoulder and is complaining of pain. He has symmetric chest rise and no chest pain. The injury you are most likely to find is:
   A. Medial third clavicle fracture.
   B. Sternal fracture.
   C. Flail chest.
   D. Lateral third clavicle fracture.
   E. Chest wall contusion.

147. A 43-year-old woman is having the “worst headache of her life,” which started suddenly more than 8 hours ago. Her head CT is normal, so you perform a lumbar puncture (LP) to rule out subarachnoid hemorrhage (SAH). The finding most suggestive of SAH is:
   A. Red blood cell (RBC) count of 7 cells/μL in Tube 1.
   B. Clear supernatant after centrifugation of the cerebrospinal fluid (CSF).
   C. RBC count of 20,000 cells/μL in Tube 1 and 120 cells/μL in Tube 3.
   D. Xanthochromia.
   E. White blood cell count of 15 cells/μL.

148. A young man complains of diarrhea and vomiting. He also notes that the cold soda he drank in the waiting room felt hot to the touch. He is likely suffering from:
   A. Ciguatera poisoning.
   B. Scombroid poisoning.
   C. *Aeromonas hydrophila* infection.
   D. *Vibrio parahaemolyticus* infection.
   E. *Entamoeba histolytica* infection.

149. A 56-year-old man presents to the ED with acute onset of chest pain and shortness of breath. One hour earlier he was released from the dialysis center after undergoing his usual 2-hour dialysis session. He presents with a blood pressure of 60 mm Hg palpable and has a depressed level of consciousness. The monitor shows narrow complex tachycardia of 150 beats/min. You suspect that the patient may have sustained an air embolism. You should immediately:
   A. Cardiovert the patient.
   B. Place the patient supine and in the left lateral decubitus position.
   C. Initiate intravenous norepinephrine.
   D. Administer oxygen if needed to maintain the oxygen saturation above 96%.
   E. Activate the cardiac catheterization lab.
150. A 30-year-old man complains of severe right knee pain that started an hour after multiple sport scuba dives to explore a wreck at 100 feet of depth. His pain is worse with movement. You see no sign of injury. The most effective management of this condition is:
   A. Benzodiazepines to relieve the muscle spasm.
   B. Aspirin to reduce platelet adherence.
   C. Hyperbaric recompression.
   D. Four liters of oxygen by nasal cannula.
   E. Arthrocentesis.

151. Family members tell you that a 68-year-old man has been "acting funny" and not taking his medicines. He is disheveled, unkempt, and agitated. Vital signs: heart rate, 146 beats/min and irregular; blood pressure, 180/80 mm Hg; temperature, 100.9°F. His skin is warm and sweaty, especially the palms and soles. Neck exam shows an enlarged thyroid gland with a bruit. Cardiac exam shows an irregular tachycardic rhythm. You initiate treatment in the emergency department, choosing as your first drug:
   A. Iodide.
   B. Metoprolol.
   C. Propylthiouracil.
   D. Solumedrol.
   E. Thyroxine.

152. A 69-year-old man with a history of lung cancer complains of shortness of breath, chest tightness, and feeling "like I'm going to die." Vital signs: blood pressure, 88/40 mm Hg; heart rate, 112 beats/min; respiratory rate, 18 breaths/min; pulse oximetry reading, 96% on room air. You notice jugular venous distension, decreased breath sounds in the left base, and distant heart sounds. You expect this patient's electrocardiogram to show:
   A. Sinus tachycardia with a right heart strain pattern.
   B. ST segment elevation in leads II, III, and AVF.
   C. Low voltage across the precordial leads.
   D. Nonspecific ST-T changes.
   E. Prolongation of the QT interval.

153. A 16-year-old boy is hallucinating. His friend tells you he was smoking a plant to get high. He is tachycardic and has large pupils and dry, flushed skin. The most likely cause of the toxicity is:
   A. Jimsonweed.
   B. Heroin.
   C. LSD.
   D. MDMA.
   E. Marijuana.

154. A first-year medical student comes to see you because she has severe nausea and has been vomiting. You determine that she is pregnant. Because this is her first pregnancy, she asks about the physiologic changes that might occur. You tell her to expect:
   A. Increased blood pressure.
   B. Increased heart rate.
   C. Decreased urination.
   D. Increased gastric emptying.
   E. No change in cardiac output.
155. A 30-year-old man presents with syncope. He has a history of dyspnea on exertion and a family history of sudden death during exertion. You hear a harsh mid-systolic murmur at the left sternal border. You can make the murmur louder by having the patient:
   A. Squat.
   B. Elevate his legs.
   C. Do an isometric handgrip.
   D. Perform a Valsalva maneuver.
   E. Take an oral dose of metoprolol and reexamining his heart sounds in 60 minutes.

156. A 6-year-old boy is brought to the ED by his mother for evaluation of a 6x6 cm boggy, indurated, pustular scalp lesion that has progressed over the past 2 weeks. Alopecia and tenderness are apparent. Wood's lamp examination shows yellow-green fluorescence. You initiate treatment with:
   A. Oral cephalexin for 2 weeks.
   B. Intravenous ceftriaxone and arrange admission for incision and drainage under general anesthesia.
   C. Oral griseofulvin for 6 to 8 weeks.
   D. A 10-day oral prednisone taper.
   E. An antifungal shampoo with rinses twice daily in sterile water.

157. A patient assaulted about the chest with a baseball bat is brought to the ED by an EMS unit. He has dyspnea, decreased breath sounds on the right, and mildly increased oxygen requirement. A supine chest radiograph shows right-sided haziness but no pneumothorax. A chest tube is placed with return of blood. An indication for operative management is:
   A. Initial drainage of 1000 ml.
   B. Persistent hypotension.
   C. Lack of air leak.
   D. Ongoing bleeding of 150 ml/hr for 3 hours.
   E. No change in chest radiograph.

158. A contraindication to the placement of a nasogastric tube is:
   A. History of esophageal varices.
   B. Prior placement within 30 days.
   C. Known gastric ulcers.
   D. Alkali ingestion.
   E. Clopidogrel use.

159. A 16-year-old boy complains of bloody diarrhea. Emergently performed flexible sigmoidoscopy shows him to have continuous, superficial ulcerations of the sigmoid colon and rectum. You should counsel him that:
   A. He will likely experience massive lower GI bleeding at some point.
   B. His risk of developing a small bowel cancer is greatly increased.
   C. His diarrhea is best treated with rest and loperamide.
   D. It would be unusual to experience extra-intestinal manifestations.
   E. Systemic corticosteroids may be helpful during exacerbations.
160. A 19-year-old man has an open left femur fracture following ejection from a motor vehicle crash, which occurred in a rural area 6 hours ago. Exam reveals a large wound to the mid-thigh with exposed bone and soil contamination. Distal pulses are present but weak. Appropriate antibiotic coverage includes:

A. Ciprofloxacin or levofloxacin alone.
B. First-generation cephalosporin plus an aminoglycoside.
C. First-generation cephalosporin plus an aminoglycoside plus penicillin.
D. Second-generation cephalosporin alone.
E. Third-generation cephalosporin alone.

161. A 65-year-old woman with a COPD exacerbation is signed out to you at shift change. She was placed on noninvasive positive-pressure ventilation 15 minutes ago for moderate respiratory distress. When you evaluate her, she denies feeling better or worse and is anxious about having to be admitted to the hospital. She is tolerating the mask well, and you plan to reevaluate her frequently. In the meantime, the best intervention for this patient is to:

A. Administer a benzodiazepine.
B. Administer magnesium.
C. Continue NIPPV.
D. Administer a loading dose of theophylline.
E. Intubate her.

162. A 30-year-old man with a history of tuberculosis complains of dyspnea. Vital signs: heart rate, 120 beats/min; blood pressure, 70/50 mm Hg; respiratory rate, 30 breaths/min; $\text{Sa}\text{O}_2$, 90%. His physical exam is notable for elevated jugular venous distention and muffled heart tones. A portable chest radiograph demonstrates an enlarged cardiac silhouette. An ECG shows low voltage and electrical alternans. After you initiate volume resuscitation and oxygen, your next step should be to:

A. Start a dopamine drip.
B. Perform rapid-sequence intubation.
C. Perform a needle thoracostomy.
D. Place bilateral chest tubes.
E. Perform pericardiocentesis.

163. A 19-year-old man is in cardiac arrest after being stabbed just to the left of the lower part of his sternum. Bedside ultrasound confirms a large pericardial effusion. You know that:

A. You have to remove at least 150 $\text{ml}$ of pericardial fluid to improve his symptoms.
B. Pericardiocentesis is the preferred procedure to relieve the tamponade.
C. Blind pericardiocentesis should never be used in emergency situations.
D. ECG-guided pericardiocentesis will demonstrate a wide QRS-complex with ST elevation (“current of injury”) when the needle is touching the pericardium.
E. Thoracotomy is preferred over pericardiocentesis in this patient.

164. Which of the following scenarios is an indication for an open ED thoracotomy?

A. Penetrating trauma and loss of vital signs in the ED.
B. Blunt arrest with ongoing CPR.
C. GSW to chest and loss of vital signs at the scene.
D. Blunt arrest with loss of vital signs >5 minutes from ED.
E. Penetrating trauma with loss of vital signs >5 minutes from ED.
165. Classic features of botulism include:
   A. Ascending paralysis.
   B. Bowel and bladder incontinence.
   C. Difficulty swallowing.
   D. Pinpoint pupils.
   E. Sore throat.

166. A 61-year-old man with a history of hypertension, diabetes, and remote abdominal aortic aneurysm repair presents obtunded and in shock with hematochezia. He should emergently undergo:
   A. Mesenteric angiography.
   B. Upper endoscopy.
   C. Colonoscopy.
   D. Exploratory laparotomy.
   E. Tagged red blood cell scan.

167. A 6-year-old boy fell 15 feet out of a window onto the driveway below. His GCS score is 8 and he has a left temporal hematoma. Because of pooled secretions in his oropharynx, you decide to intubate him. What tube size do you choose and to what depth do you place it?
   A. 5.0 cuffed ETT placed at a depth of 15 cm.
   B. 6.0 cuffed or uncuffed ETT placed at a depth of 18 cm.
   C. 5.5 uncuffed ETT placed at a depth of 20 cm.
   D. 5.5 cuffed or uncuffed ETT placed at a depth of 15 cm.
   E. 6.5 cuffed or uncuffed ETT placed at a depth of 15 cm.

168. A 30-year-old woman presents with an abscess that requires incision and drainage. She has a history of repaired ventricular septal defect. During childhood, she was given a dose of penicillin and developed a rash. The best course of action prior to abscess incision and drainage is:
   A. Endocarditis prophylaxis with 2 grams of oral amoxicillin 1 hour prior to I&D.
   B. Endocarditis prophylaxis with clindamycin, 600 mg, 1 hour prior to I&D.
   C. Endocarditis prophylaxis with azithromycin, 500 mg, 1 hour prior to I&D.
   D. Endocarditis prophylaxis with ampicillin, 2 grams IV, 30 minutes prior to I&D.
   E. Incision and drainage without endocarditis prophylaxis.

169. A 31-year-old man, active and otherwise healthy, complains of 3 days of constant anterior chest pain, which is pleuritic and exacerbated by his dry cough of 3 days. He admits to some increased fatigue, which he attributes to “getting out of shape already,” as he has been using crutches for the past week while awaiting surgical repair of his left Achilles tendon, which he ruptured while playing basketball. He denies fever and URI symptoms other than the cough. His lungs are clear and the toes of his left foot show some edema, which the patient says has worsened since the cast was placed. An ECG shows sinus tachycardia at 110 beats/min without LVH, and a chest film is normal. The triage nurse ordered a d-dimer before the patient was brought to the treatment area, and the result was negative. Your next move is to:
   A. Pursue risk stratification for consideration of coronary artery disease.
   B. Order a CT scan of the thorax with contrast.
   C. Discharge the patient with an NSAID and diagnosis of muscle strain from crutch use.
   D. Prescribe an antitussive medication and give instructions for a viral URI.
   E. Prescribe an albuterol MDI and incentive spirometer for atelectasis.
170. A 55-year-old man is brought to an emergency department in Arizona by an EMS unit for evaluation of shortness of breath. Over the past 2 days, he noted fever, malaise, and generalized muscle aches. Shortly before calling paramedics, he developed shortness of breath, which had progressed rapidly over several hours. He has a history of hypertension and takes lisinopril. He is not allergic to any medications. He is not a smoker. He is in moderate respiratory distress with use of accessory muscles. Vital signs: respiratory rate, 32 breaths/min; pulse oximetry reading, 86% on room air; heart rate, 122 beats/min; blood pressure, 89/44 mm Hg. He has diffuse rhonchi in both lungs, but the remainder of his exam is normal. A chest film shows bilateral pulmonary infiltrates consistent with acute respiratory distress syndrome. The most likely reservoir for this systemic infection is the:
   A. Anopheles mosquito.
   B. Deer mouse.
   C. Dermacentor tick.
   D. Ixodes tick.
   E. Hookworm.

171. An EMS unit transports a patient with a penetrating lower chest wall injury. While evaluating the patient, you recognize distant heart tones, mild jugular venous distension, and pulsus paradoxus. Given the current BP of 104/86 mm Hg and the known history of hypertension, you act on your clinical diagnosis by:
   A. Ordering a chest radiograph.
   B. Performing pericardiocentesis.
   C. Performing thoracostomy.
   D. Ordering an ECG.
   E. Ordering a CT scan of the chest.

172. A 13-year-old boy has had right hip and knee pain for 3 weeks. He denies trauma, fevers, or chills. His vital signs are normal. He is holding his right hip in mild external rotation. The most likely diagnosis is:
   A. Transient synovitis of the hip.
   B. Slipped capital femoral epiphysis (SCFE).
   C. Legg-Calve-Perthes (LCP) disease.
   D. Septic arthritis.
   E. Hip avulsion fracture.

173. A 64-year-old man complains of right-sided headache and sudden vision loss in his right eye. Visual acuity is 20/200 in the right eye and 20/40 in the left eye. Extraocular movements are normal. His right pupil is non-reactive to light. You instill a topical anesthetic, but there is no relief of pain. The most likely diagnosis is:
   A. Acute angle-closure glaucoma.
   B. Acute iritis/uveitis.
   C. Giant cell arteritis.
   D. Central retinal vein occlusion.
   E. Optic neuritis.
174. A 62-year-old woman complains of lower abdominal pain and constipation. She has a low-grade fever, and you find a tender mass in the left lower quadrant. You suspect she has diverticulitis. Choose the true statement about diverticulitis.
   A. All patients with diverticulitis should be hospitalized.
   B. The finding of pyuria should change your primary diagnosis from diverticulitis to cystitis.
   C. Abscess formation is never seen in diverticulitis.
   D. Hospitalized patients frequently require surgery.
   E. Colonoscopy is the test of choice to make the diagnosis.

175. A 24-year-old man with a history of intravenous drug use complains of fevers, night sweats, chest discomfort, malaise, diffuse nonlocalizing headache, and anorexia. Vital signs: heart rate, 120 beats/min; blood pressure, 150/100 mm Hg; respiratory rate, 20 breaths/min; SaO₂, 99%; temperature, 103.4°F. His cardiac exam is normal except for tachycardia. His lungs are clear to auscultation. He does not have any meningeal signs. When ordering antibiotics, you must cover the most likely organism causing endocarditis, which is:
   A. *Staphylococcus epidermidis*.
   B. Gram-negative rods.
   C. *Streptococcus viridans*.
   D. *Staphylococcus aureus*.
   E. *Pseudomonas aeruginosa*.

176. A useful method for treating epistaxis of undetermined source would be:
   A. Silver nitrate application to any anterior actively bleeding sources.
   B. Icepacks across the bridge of the nose.
   C. Icepacks on the back of the neck.
   D. Bilateral electrocautery or chemical cautery of identified actively bleeding sources.
   E. Placement of a posterior balloon pack and prophylactic systemic antibiotics.

177. A small traumatic pneumothorax after blunt trauma in a stable patient would be best managed by:
   A. Chest tube placement.
   B. Discharge.
   C. Nasal cannula oxygen and repeat imaging.
   D. Facemask oxygen and repeat imaging.
   E. Needle decompression.

178. An 85-year-old man with COPD and obstructive sleep apnea has had a newly productive cough for 1 week, with increased dyspnea and fatigue. He does not use home oxygen. His oxygen saturation on room air is currently 70%. You note that he is leaning forward over his knees, with increased work of breathing and prolonged expirations. He is alert and afebrile; his chest film shows no changes from the one taken last month. The treatment most likely to help decrease the morbidity related to his hospital stay is:
   A. Noninvasive positive-pressure ventilation (NIPPV).
   B. Ipratropium or another quaternary ammonium compound, such as tiotropium.
   C. Oxygen and aspirin or another antiplatelet agent.
   D. Albuterol.
   E. Intubation with mechanical ventilation.
179. Your hospital's infection control nurse notifies you that the infant you admitted yesterday with meningitis is growing meningococci from both blood and cerebrospinal fluid cultures. You know that prophylaxis with rifampin should be mandatory for:

A. Personnel at the daycare center where the child recently spent 6 hours.
B. Emergency department nursing personnel.
C. Paramedics who performed mouth-to-mouth resuscitation prior to intubating the child.
D. The resident who performed the spinal tap.
E. The physician who did the history and physical examination.

180. An elderly nursing home patient is sent to the ED for evaluation of abdominal distention. Symptoms have progressed over several weeks, and the patient now is markedly distended with vomiting and diffuse tenderness. Plain radiographs reveal a "bent inner tube" pattern to the bowel gas. This condition:

A. Can initially be treated nonoperatively.
B. Occurs most frequently in 20 to 40 year olds.
C. Recurs infrequently.
D. Usually presents with more acute onset of symptoms.
E. Requires antibiotics in all cases.

181. A 22-year-old man brings you his incisors soaking in a glass of milk. They were knocked out by a baseball about 90 minutes ago. Choose the correct statement about the treatment of this patient:

A. Re-implantation will be unsuccessful because all periodontal ligament cells die after 60 minutes.
B. You should scrub the tooth clean prior to attempting re-implantation.
C. You should attempt re-implantation because storage in milk extends the life of periodontal ligament cells up to 3 hours.
D. You should store the tooth in saline and refer the patient to an oral surgeon.
E. You should wash the tooth with dilute povidone-iodine prior to re-implantation.

182. A 65-year-old woman arrives by ambulance complaining of acute onset of shortness of breath. Vital signs: heart rate, 120 beats/min; blood pressure, 140/100 mm Hg; respiratory rate, 24 breaths/min; SaO₂, 93%. She is afebrile. On physical examination, you note jugular venous distension and lower extremity edema. Two large-bore IV lines have been placed, and the patient is receiving supplemental oxygen. She has been placed on a cardiac monitor and pulse oximeter. The best next steps in management include:

A. Furosemide and nitrates.
B. Broad-spectrum antibiotics.
C. Heparin.
D. Albuterol and steroids.
E. Nonsteroidal anti-inflammatory agents.

183. A 13-year-old schoolgirl has a swollen right eye. She is having her school picture taken next week and asks that you fix the swelling. You see a raised nodule in her upper eyelid, which is not tender or red. Her visual acuity is normal, and her conjunctiva is not injected. The most likely diagnosis is:

A. Hordeolum.
B. Chalazion.
C. Stye.
D. Blepharitis.
E. Conjunctivitis.
184. The chest radiograph of a trauma patient with stab wounds to the abdomen demonstrates pleural effusion and an NG tube above the diaphragm. This probably indicates a:
   A. Pleural contusion.
   B. Hiatal hernia.
   C. Hollow viscous injury.
   D. Liver laceration.
   E. Diaphragm disruption.

185. An 11-year-old boy weighing 30 kg is brought to the ED in cardiopulmonary arrest by a local fire rescue unit. The initial dose of intravenous epinephrine should be:
   A. 0.03 mg.
   B. 0.1 mg.
   C. 0.3 mg.
   D. 1 mg.
   E. 3 mg.

186. In the emergency department, it is safe to perform incision and drainage on:
   A. An ischiorectal abscess.
   B. A deep postanal abscess.
   C. A supravelator abscess.
   D. A perianal abscess.
   E. An intersphincteric abscess.

187. The most common type of elder abuse is:
   A. Verbal.
   B. Financial.
   C. Psychological.
   D. Neglect.
   E. Physical.

188. You are just about to reach the 12,000-foot summit of Mount Trepidation when your trekking buddy complains of fatigue, a dry cough, and dyspnea on exertion. You take your stethoscope out of your backpack and listen to his lungs; you hear bilateral rales. You should:
   A. Give him some acetazolamide, wait 15 minutes, and continue ascending the mountain.
   B. Stop for a few minutes to breathe some supplemental oxygen in order to adjust to the altitude.
   C. Have him hold his breath for 30 seconds in order to equilibrate his oxyhemoglobin-carboxyhemoglobin dissociation curve.
   D. Immediately descend.
   E. Administer an albuterol inhaler.
189. A 30-year-old woman complains of nausea and vomiting. Her blood pressure is 70/40 mm Hg. She has a history of asthma and has been to your ED frequently for treatment. You give her 3 liters of normal saline, but her blood pressure does not increase. The most likely cause of her symptoms is:
   A. Adrenal failure.
   B. Asthma.
   C. Gastroenteritis.
   D. Invasive fungal infection.
   E. Sepsis.

190. A 22-year-old woman is transported by an EMS unit after she dove into the pool at a local motel from a second-floor balcony. She complains of neck pain and body numbness. She can shrug her shoulders and flex her arm at the elbow, but she cannot extend her arm at the elbow. In her hand, she has sensation only in her thumb. This exam corresponds to a lesion below the level of:
   A. C4.
   B. C5.
   C. C6.
   D. C7.
   E. C8.

191. A 56-year-old woman with metastatic breast cancer complains of nausea, vomiting, poor appetite, constipation, and confusion. The most appropriate treatment option initially is:
   A. Rapid infusion of inorganic phosphate.
   B. Administration of furosemide after intravenous hydration with saline.
   C. Strict water restriction.
   D. Free water replacement.
   E. 3% hypertonic saline infusion.

192. You would expect a patient with an opioid overdose to have:
   A. Large pupils.
   B. Acute abdominal pain.
   C. Decreased respirations.
   D. Dry mucous membranes.
   E. Normal mental status.

193. A 65-year-old man with a history of hypertension and diabetes complains of chest heaviness, shortness of breath, sweating, and vomiting. His medications include hydrochlorothiazide, insulin, and sildenafil citrate, all of which he has taken in the past 6 hours. Vitals signs: heart rate, 80 beats/min; blood pressure, 140/100 mm Hg; respiratory rate, 16 breaths/min; SaO₂, 99%. The medication you must avoid in his treatment is:
   A. Oxygen.
   B. Metoprolol.
   C. Morphine.
   D. Nitroglycerin.
   E. Aspirin.
194. An 82-year-old woman who is obviously short of breath arrives by ambulance from her home. You recognize her, as you treated her last week when she tripped and fell on the sidewalk while walking her dog. At that time, you thoroughly evaluated her and diagnosed minor chest wall abrasions and contusions. Vital signs: temperature, 100.6°F; heart rate, 112 beats/min; respiratory rate, 28 breaths/min; blood pressure, 168/104 mm Hg; SpO₂, 93% on supplemental oxygen. Her neck veins are flat, her trachea is midline, and she has diminished breath sounds on the left. She is splinting when she takes a deep breath, but you note no asymmetry. Heart sounds are normal, other than the tachycardia. It is likely that, despite your careful evaluation, you missed a:
   A. Myocardial contusion.
   B. Hemopneumothorax.
   C. Rib fracture.
   D. Splenic laceration.
   E. Flail chest.

195. A 76-year-old man with a history of hypertension and kidney stones presents with right flank pain radiating to the groin. Urine dipstick analysis reveals the presence of blood, leukocyte esterase, and nitrates. His physical examination reveals mild mid-epigastric tenderness, and his blood pressure is 90/60 mm Hg with a pulse of 110 beats/min. Your next step should be to:
   A. Perform a bedside aortic ultrasound.
   B. Administer intravenous antibiotics.
   C. Administer intravenous ketorolac for presumed renal colic.
   D. Request a CBC, liver function tests, and lipase measurement and await the results.
   E. Initiate early goal-directed sepsis therapy in response to the patient’s hypotension and evidence of urinary tract infection.

196. A non-cyanotic cause of congenital heart disease is:
   A. Transposition of the great arteries.
   B. Tetralogy of Fallot.
   C. Patent ductus arteriosus.
   D. Tricuspid atresia.
   E. Truncus arteriosus.

197. A young teenager is transported by EMS after falling off his bicycle and landing on the handlebars. He was wearing a helmet and did not lose consciousness. He is complaining of abdominal and back pain and is tender in his epigastrium. He has a normal neurologic exam. CT imaging shows no solid organ injuries or free fluid. You should be most worried about:
   A. Delayed liver injury.
   B. Splenic sequestration.
   C. Mesenteric ischemia.
   D. Pancreatic injury.
   E. Retroperitoneal bleed.
198. A 9-month-old girl had a seizure lasting about 2 minutes. Her mother tells you the child's eyes rolled back, followed by generalized convulsions with a short post-ictal state. She previously had a fever for 1 day but was otherwise acting and eating well. She has never had a seizure before. Physical examination is unremarkable except for a rectal temperature of 38.4°C. The most appropriate set of diagnostic studies to obtain in the ED is:

A. Bedside glucose only.
B. Serum electrolytes.
C. CBC, serum electrolytes, blood and urine cultures, and lumbar puncture.
D. Lumbar puncture only.
E. CT scan of the head and lumbar puncture.

199. Which of the following is true regarding anal fissures?

A. They are most commonly midline.
B. Diarrhea is a predisposing factor to their development.
C. Antibiotics increase the cure rate.
D. They are often a source of painless rectal bleeding.
E. All require surgical follow-up for definitive treatment.

200. A 67-year-old otherwise healthy woman presents after a syncopal event. She admits to dyspnea and chest pain with exertion. Her review of systems is otherwise negative. Vital signs: heart rate, 80 beats/min; blood pressure, 120/100 mm Hg; respiratory rate, 16 breaths/min; \( \text{SaO}_2 \), 99%. Her physical exam is significant for a harsh systolic ejection murmur best heard in the second right intercostal space, with radiation to the right carotid artery. You also hear a split S2. Her ECG demonstrates left ventricular hypertrophy but is otherwise normal. Her initial troponin and d-dimer are negative. You know that:

A. Appropriate management includes the administration of nitrates and diuretics.
B. She should be sent for an immediate treadmill stress test.
C. Her expected survival is less than 5 years.
D. She should be sent for an emergent diagnostic chest CT scan to rule out pulmonary embolism and thoracic aorta dissection.
E. She may be discharged to home.

201. A paramedic gives a patient a medication as part of a standing order or protocol without speaking to medical control. Choose the correct statement about medical control:

A. Using a protocol is an example of direct medical control.
B. Speaking with the medical director is an example of indirect medical control.
C. Speaking with a physician at the scene is an example of indirect medical control.
D. Standing orders are an example of direct medical control.
E. Standing orders are an example of indirect medical control.
202. At 6:30 a.m., a 54-year-old man with a history of smoking arrives in your emergency department saying that he woke up feeling very short of breath. He denies chest pain or fevers, but has had rhinorrhea and a productive cough for the past 4 days, and he felt even more breathless after he made breakfast this morning. His respiratory rate is 24 breaths/min, and he has mild to moderate difficulty breathing. He has flat neck veins, and no S3 or leg swelling; his lungs have fine scattered wheezing. A chest film shows flattened diaphragms but is otherwise unremarkable. After nebulized beta-agonist treatment, the next drug you administer should be:
A. An ACE inhibitor.
B. A diuretic.
C. A nitrate.
D. A steroid.
E. An antiplatelet agent.

203. A multi-system trauma patient is being evaluated in your ED. Clinical appearance and radiographic images confirm an open-book pelvic fracture. The next step is:
A. CT imaging.
B. Arteriogram.
C. Pelvic compression.
D. Central line placement.
E. Surgical intervention.

204. The most appropriate initial antibiotic for a 6-year-old with suspected pneumonia is:
A. Amoxicillin.
B. Azithromycin.
C. Cefuroxime.
D. Doxycycline.
E. Trimethoprim-sulfamethoxazole.

205. A 22-year-old man complains of abdominal pain. His abdominal exam shows some suprapubic rigidity. His oral temperature is 101°F and his lab studies show mild leukocytosis. Plain films show no free air, but you note a rectal foreign body. Which of the following is true regarding management?
A. The foreign body will likely pass on its own.
B. If you can successfully remove the foreign body, you can safely discharge the patient since there is no free air on the radiograph.
C. Further questioning may only embarrass the patient and is not warranted.
D. CT scan should be performed.
E. Opioid analgesia should be withheld.
206. A 45-year-old welder complains of eye pain. He tells you that earlier today he was working on a framing rig, where he was welding and grinding iron while lying on his back. Toward the end of the day, he noticed some left eye pain, which has gotten worse over several hours. His right eye is unremarkable, but his left eye is tightly closed with tearing. He gets significant relief from a topical anesthetic. A visual acuity test shows 20/20 vision in each eye. His eyelids are unremarkable, but the left conjunctiva is injected. His pupils react appropriately. His most likely diagnosis is:

A. Blepharitis.
B. Acute iritis.
C. Ocular foreign body with corneal abrasion.
D. Optic neuritis.
E. Acute angle-closure glaucoma.

207. A 58-year-old man with lung cancer presents 7 months after his most recent radiation treatment with progressive dyspnea and dry cough for 1 month. He has right-sided pleuritic chest pain but no extremity swelling. Vital signs: oral temperature, 98.9°F; heart rate, 95 beats/min; respiratory rate, 26 breaths/min; blood pressure, 136/78 mm Hg; SaO₂, 93%. The test result that would most decrease your estimated likelihood of pulmonary embolism is:

A. An ECG without S1-Q3-T3 pattern.
B. Negative Homan's sign.
C. Chest film showing a small infiltrate in the right middle lobe.
D. Chest film showing 60% pleural effusion on the right.
E. Arterial blood gas showing a PaCO₂ of 30.

208. A 70-year-old man with a history of atherosclerosis presents with a sudden onset of abdominal pain radiating to his left flank. Vital signs: heart rate, 140 beats/min; blood pressure, 90/60 mm Hg; respiratory rate, 18 breaths/min; SaO₂, 93%. He is afebrile. His abdomen is obese, making palpation very difficult. The department ultrasound machine has a sign on it that says "broken." You should now:

A. Obtain a stat non-contrast abdominal CT scan.
B. Obtain a contrast abdominal CT scan after full oral preparation.
C. Obtain an abdominal radiograph.
D. Administer ketorolac and obtain urinalysis; if hematuria is present, then discharge the patient home with urology follow-up.
E. Obtain immediate vascular surgery consultation.

209. A 2-week-old girl is found to have a blood glucose reading of 20 mg/dL. The correct glucose replacement fluid is:

A. Dextrose, 5%, 2 ml/kg.
B. Dextrose, 10%, 4 ml/kg.
C. Dextrose, 25%, 2 ml/kg.
D. Dextrose, 50%, 1 ml/kg.
E. Lactated Ringer's solution, 20 ml/kg.
210. A 15-month-old boy has a 1-day history of fever (rectal temperature of 39.8°C). He has been clutching his left ear. He has never had an ear infection before. You see a bulging red tympanic membrane with limited mobility with insufflation. The most appropriate treatment is:
   A. Observation only with follow-up in 2 days.
   B. Observation, with a wait-and-see prescription (WASP) for oral antibiotics, with instructions to fill the prescription and administer the antibiotics if symptoms don't improve in 1 day.
   C. Amoxicillin, 40 mg/kg/day.
   D. Amoxicillin, 80 mg/kg/day.
   E. Amoxicillin/clavulanate, 40 mg/kg/day.

211. A patient is brought to the ED by an EMS unit, intubated, after blunt chest and abdominal trauma. He remains hypotensive despite administration of 2,000 mL of intravenous fluids by the prehospital crew. Exam is notable for a distended abdomen, cool skin, and faint distal pulses. Secondary survey shows no obvious fractures, and a pelvic radiograph is negative. A FAST study is positive for fluid in Morrison's pouch and in the splenorenal view. Given the persistent hypotension despite intravenous fluids in the ED, the patient requires:
   A. Serial FAST.
   B. CT imaging.
   C. Angiography.
   D. Surgical intervention.
   E. Diagnostic peritoneal lavage.

212. A 56-year-old man with history of arthritis complains of a painful, swollen left knee. He denies trauma, but says he thinks he had a fever. You perform arthrocentesis and send the fluid for evaluation. The WBC count is 30,000/ml, with 70% polymorphonuclear cells; his glucose concentration is 15 mg/dl; and the Gram stain is negative. This is most consistent with:
   A. Osteoarthritis.
   B. Psoriatic arthritis.
   C. Rheumatoid arthritis.
   D. Septic arthritis.
   E. Viral arthritis.

213. Which of the following is true regarding esophageal foreign bodies?
   A. Foreign bodies in children usually lodge at the lower esophageal sphincter.
   B. Coins in the esophagus are usually seen in the sagittal plane on frontal radiographs.
   C. Button batteries need to be removed emergently, even if they have passed into the stomach.
   D. Endoscopy is the best method to retrieve sharp, elongated objects.
   E. Glucagon is helpful for objects impacted at the crossover of the aortic arch.

214. A 65-year-old woman complains of chest pain and vomiting. On exam you note that she has subcutaneous emphysema in her neck. The most frequent cause of this syndrome is:
   A. Iatrogenic instrumentation.
   B. Trauma.
   C. Caustic ingestion.
   D. Weightlifting.
   E. Retching.
215. A 5-month-old girl has a rectal temperature of 39.6°C but is vigorous and feeding well without vomiting. She was born full term without complications and has no other medical problems. Urinalysis reveals 35 WBCs/hpf but a negative leukocyte esterase test. The most appropriate management is:
   A. Hospitalize and administer intravenous antibiotics.
   B. Hospitalize; perform full septic workup, including lumbar puncture; and administer intravenous antibiotics.
   C. Hospitalize but do not administer antibiotics, pending urine culture results.
   D. Administer parenteral antibiotics in ED and discharge on oral antibiotics, pending urine culture results.
   E. Discharge home and do not administer antibiotics, pending urine culture results.

216. A local BLS unit brings you a 19-year-old woman who was found somnolent in the back seat of a parked car. Her breath sounds are clear, but she has snoring respirations. She moans to noxious stimuli. She has no external signs of trauma, and one of the nurses recognizes her as a heroin user who has been seen at several local hospitals after overdose. Her respiratory rate is 6 breaths/min, and her oxygen saturation is 94% on room air, but her temperature, blood pressure, and heart rate are normal. You give her 0.4 mg of intravenous naloxone and she becomes more alert. About 10 minutes later, she becomes dyspneic and tachypneic and starts to cough. Her lung exam now shows diffuse crackles. She becomes increasingly hypoxic and requires intubation. The chest film shows the endotracheal tube at an appropriate depth, a normal-sized heart, bilateral fluffy infiltrates, and no pneumothorax. The most important feature(s) to include in this patient's treatment is (are):
   A. Steroids and high peak pressures.
   B. Trimethoprim/sulfamethoxazole and high-dose steroids.
   C. Tidal volume of 6 mL/kg.
   D. IV fluids and emergent transthoracic echocardiography.
   E. Aggressive suctioning and broad-spectrum antibiotics.

217. A 35-year-old woman with a history of pulmonary embolism complains of pleuritic chest pain, shortness of breath, and coughing up blood. She has recently returned from a trip to Russia. Vital signs: heart rate, 120 beats/min; blood pressure, 120/60 mm Hg; respiratory rate, 28 breaths/min; SaO₂, 93%. She is afebrile. You order a CT pulmonary angiogram, which shows a large central pulmonary embolism. As you are explaining the results of the CT scan to the patient, she becomes unresponsive. Her cardiac monitor shows a slow, regular heart beat but you find no pulses. In addition to standard ACLS protocol, you should also:
   A. Give unfractionated heparin, 60 IU/kg (maximum 4,000 units), as an IV bolus, followed by 12 IU/kg/hr (maximum 1,000 units/hr).
   B. Administer 325 mg of aspirin by rectum.
   C. Perform a needle thoracostomy.
   D. Administer a thrombolytic agent.
   E. Perform immediate pericardiocentesis.
218. A 14-year-old swimmer complains of worsening right ear pain. Last night she had difficulty sleeping. You find an edematous, red external auditory canal that is full of fluid and debris. She experiences a lot of pain when you apply traction to her pinna. You suspect she has acute otitis externa. Choose the correct statement regarding acute otitis externa:

A. The most common causative organisms are *S. pneumoniae* and *S. aureus*.
B. Avoid antibiotic suspensions if you suspect perforation of the tympanic membrane.
C. A 2% acetic acid solution can be used to clean the external ear canal but is inadequate to treat acute infection.
D. The most common causative organisms are *P. aeruginosa* and *S. aureus*.
E. Ophthalmic preparations should not be used for otitis externa because they have a higher pH than otic solutions and cause more pain.

219. A 45-year-old man is brought by ambulance from a restaurant with the feeling that he has a piece of steak stuck in his throat. He is hemodynamically stable and his airway is patent. Of the therapies listed below, the one most likely to be effective is:

A. Nifedipine.
B. Meat tenderizer.
C. Glucagon.
D. Diazepam.
E. Nitroglycerin.

220. Which of the following statements about supraventricular tachycardia (SVT) in infants and children is true?

A. Adenosine is the drug of choice for stable patients.
B. Initial energy level for synchronized cardioversion is 2 joules/kg.
C. It never causes hemodynamic instability.
D. Rates usually are less than 200 beats/min.
E. Wide QRS complex tachycardia commonly is supraventricular in origin.

221. You have five patients, each of whom has right middle lobe pneumonia. None of them has been hospitalized recently. Which one requires antibiotic coverage beyond the common combination of a macrolide with a third-generation cephalosporin?

A. The 84-year-old man who has isolated chronic hemiplegia and lives with his son.
B. The 28-year-old asthmatic with an exacerbation requiring admission.
C. The 36-year-old HIV-positive patient with a recent CD4 of 500.
D. The 72-year-old COPD patient who has a temperature of 101.1°F.
E. The 49-year-old diabetic with alcoholism and severe periodontal disease.

222. A 50-year-old man with uncontrolled hypertension complains of sudden-onset severe tearing chest pain radiating to his back. He has no history of similar symptoms. The most likely finding on the chest radiograph of this patient would be:

A. Normal.
B. Wide mediastinum or abnormal aortic contour.
C. Atelectasis.
D. Elevated hemidiaphragm.
E. Hampton's hump.
223. You would expect a patient with a cocaine overdose to have:

A. Large pupils.
B. Acute abdominal pain.
C. Decreased respirations.
D. Dry skin.
E. Low blood pressure.

224. A 24-year-old woman complains of malaise, headache, mild sore throat, and an itchy rash. A single 3-cm oval, red patch appeared on her mid back about a week ago. Now she has a diffuse rash consisting of 1-cm pink, symmetric lesions that spare the palms and soles and appear to be elongated in the direction of the lines of skin tension. Your recommendations include:

A. Intravenous antibiotics, steroids, and lumbar puncture.
B. Fluorinated corticosteroids applied twice daily to all facial lesions.
C. Counseling that this may take more than a month to resolve.
D. Admission for intravenous ceftriaxone.
E. Dermatology appointment for a biopsy.

225. A 50-year-old man is seizing on arrival at the ED. He has a history of psychiatric disease and has reportedly been drinking "a lot of water." You give 200 mL of 3% saline solution and the seizure stops. The factor most likely to lead to the osmotic demyelination syndrome is:

A. Initial sodium concentration.
B. Rapidity of fluid administration.
C. Serum osmolality.
D. Type of seizure.
E. Volume of fluid.
Practice Test Answers

1. The correct answer is D. The lateral neck radiograph (Image 1) shows “thumbprinting” of the edematous epiglottis and, when combined with the clinical vignette, strongly suggests the diagnosis of acute epiglottitis. Epiglottitis typically occurs with a rapid onset of symptoms, unlike bacterial tracheitis, which typically follows several days of antecedent upper respiratory infection symptoms. In this case, the next step in management should be obtaining emergent otolaryngology consultation for emergent intubation in the operating room by the most experienced intubator available, with a double setup for immediate surgical airway placement if direct laryngoscopy fails. Obtaining IV access and blood cultures is a reasonable step, but these interventions are secondary in importance to securing the airway before the edema progresses further. You should agitate the child as little as possible to prevent crying and precipitating worsening dynamic airway obstruction. Defer painful or anxiety-producing procedures until the airway is secured. There is no indication for emergent surgical airway in the emergency department unless the child abruptly loses her airway prior to transport to the operating room. Nebulized racemic epinephrine is the agent of choice for severe croup, but it is unlikely to help a patient with epiglottitis. Nebulized albuterol treats lower airway obstruction such as bronchospasm in asthma but is less valuable for upper airway emergencies.

2. The correct answer is E. Image 2 shows perirectal swelling that is probably an abscess that has progressed to erythema of the entire perineum and scrotum. The symptom of severe pain, frequently out of proportion to exam, and the photograph suggest that this infection is Fournier’s gangrene. This type of infection is typically polymicrobial, with the most common anaerobe being Bacteroides fragilis and the most common aerobic organism being E. coli. Other common organisms cultured from patients with Fournier’s gangrene include Staphylococcus aureus, Enterococcus, aerobic and anaerobic Streptococcus spp., Proteus, and Klebsiella. Since these infections are polymicrobial, broad-spectrum antibiotic coverage is required, being certain to include coverage for gram-positive, gram-negative, and anaerobic organisms. Candidal infections do not typically cause severe pain or abscess formation. There are no lesions suggestive of syphilis, and Treponema pallidum does not cause diffuse erythema and edema. Gonococcal infections cause cutaneous findings that tend to be maculopapular or petechial and remote from the site of genital infection. Cryptosporidium infection causes diarrheal disease but not local perirectal cutaneous abnormalities.

3. The correct answer is E. The patient is speaking clearly, has a normal mental status, and has adequate oxygen saturation, so you have no indications at this time to perform emergent endotracheal intubation. The radiograph (Image 3) shows a combined hemothorax and pneumothorax, so you should perform tube thoracostomy with a 36 or 40 French chest tube (a simple pneumothorax can be managed with a 24 or 28 French chest tube). She has no clinical or radiographic evidence of tension pneumothorax, so emergent needle thoracostomy is not required at this time. With a pneumothorax of this size (involving apex to diaphragm on the chest film, >1 cm away from the chest wall), observation is inadequate therapy. The hemothorax mandates tube thoracostomy. Of the options listed, the next appropriate intervention is IV catheter placement with fluid resuscitation.
4. The correct answer is D. A lateral radiograph is unnecessary because coins will lie in the coronal plane if lodged in the esophagus (as in Image 4) or in the sagittal plane if lodged in the trachea (due to lack of cartilage in the posterior part of the trachea). Esophageal perforation is unlikely to have occurred in 2 hours, and only water-soluble contrast should be used if perforation is suspected. The coin is lodged at the level of the cricopharyngeus (C6, the most common site) or the thoracic inlet (T1). Since this radiograph was taken 2 hours after the object was swallowed, it is probably stuck and spontaneous passage is unlikely. The method of choice to remove the object is endoscopy, since this technique directly visualizes the object.

5. The correct answer is A. The radiograph shows gas tracking along fascial planes in the musculature. Gas gangrene may be caused by clostridial species (*Clostridium perfringens* most commonly) or nonclostridial mixed infections of aerobic and anaerobic organisms (*Enterococcus, Staphylococcus, E. Coli, Klebsiella, Bacteroides, Proteus, alpha Streptococcus*). Your most important management considerations are vigorous fluid resuscitation, broad-spectrum antibiotics, and early surgical debridement. There is no indication for heparin use, and, in fact, coagulopathy may complicate these infections. You should avoid vasopressor agents, as they might decrease perfusion in ischemic vascular beds. Hyperbaric oxygen therapy is a useful adjunctive therapy but is secondary to early antibiotics, fluid resuscitation, and surgical therapy. Areas of myonecrosis may be extensive. Involved tissue should be explored and debrided in the operating room, not the emergency department.

6. The correct answer is B. Since the CT was done without contrast, the finding suggests that the lesions are high CT density (e.g., acute accumulations of blood or calcium). In this case, the calcifications have resulted from neurocysticercosis, a long-term sequela of infection with *Tenia solium*, the pork tapeworm. Outside the United States, neurocysticercosis is a common cause of adult-onset seizures. An enlarging breast mass might suggest breast cancer with risk for metastases to the brain, but metastases typically appear hypodense on noncontrast CT and require either contrast head CT or MRI for better characterization. Exposure to cat feces would be a risk factor for cerebral toxoplasmosis, but again these lesions are typically hypodense on noncontrast head CT and are seen better on either contrast head CT or MRI. Recent dental surgery might suggest a risk for odontogenic brain abscess but would be unlikely to result in multiple lesions and the site would appear hypodense on noncontrast head CT. A older adult who fell several months before ED presentation could have chronic subdural hematoma, but this lesion would not result in intraparenchymal radiographic abnormalities and would be isodense to brain on a noncontrast head CT.

7. The correct answer is E. The wrist radiograph shows a perilunate dislocation, which is best seen on the lateral view. The lunate remains in contact with the radius but the capitate is dislocated posterior to the lunate. In a scapho-lunate dissociation, the PA view is most helpful because the scapholunate joint space is widened to more than 3 mm (the “Terry Thomas sign,” named after the British comedian who had a gap between his teeth). In a distal radioulnar joint (DRUJ) dislocation, the PA view shows narrowing of the DRUJ joint space. In a lunate dislocation, the PA view demonstrates a triangular-appearing lunate (“piece of pie sign”) and the lateral view shows the lunate spilling into the palm (“spilled teacup sign”) with loss of lunate relationship to the radius. Scaphoid fracture is not seen on this radiograph, but it still may be present but radiographically occult.
8. The correct answer is C. The chest film shows a pulmonary cavitary lesion with an air-fluid level in the left mid-lung field medially. While the differential diagnosis of a pulmonary cavitary lesion includes bacterial causes of abscess, fungal causes of pulmonary abscess, tuberculosis, neoplastic processes, and autoimmune disease such as sarcoidosis, the acute nature of this process suggests an infectious cause, specifically pulmonary abscess. Lung abscess in the immunocompetent patient is most commonly secondary to aspiration of anaerobic oral flora, particularly associated with gingivitis. Long-distance car or airline travel would place the patient at risk for pulmonary embolism, but this problem is not associated with cavitary lesions. A history of smoking would place the patient at risk for pulmonary embolism, spontaneous pneumothorax, and neoplastic disease. Employment as a prison health care worker might place the patient at somewhat higher risk for tuberculosis, but cavitary lesions from tuberculosis tend to be located in the apices of the upper lobe or superior segment of the lower lobe and air-fluid levels are uncommon. Alpha-1-antitrypsin deficiency can lead to early-onset COPD with resultant blebs on chest radiograph, but these areas of lucency lack air-fluid levels.

9. The correct answer is A. The lesion represents true dendrites associated with corneal infection with herpes simplex virus and requires topical acyclovir nine times a day with urgent ophthalmology follow-up. Topical ciprofloxacin is appropriate for bacterial conjunctivitis in the contact lens wearer. Topical steroids such as prednisolone should be avoided in herpes simplex virus keratitis but have a role in treating iritis. Intravenous medications are not indicated.

10. The correct answer is B. The photograph shows Ludwig's angina, an aggressive soft tissue infection of the submento-submandibular spaces, which is typically odontogenic in origin. The edema can progress very rapidly, causing sudden loss of airway patency, so early endotracheal intubation (usually fiberoptic due to the technical difficulty of direct laryngoscopy in these patients) is indicated. Mediastinitis, bacteremia/sepsis, and pulmonary abscess are all known complications of this process but are less common causes of mortality. Thyroxin and catecholamine excess would be the expected result of hyperthyroidism, which may present with neck pain, fever, and odynophagia if it is associated with acute thyroiditis; however, the degree of submandibular erythema seen in the photograph would not accompany thyroiditis.

11. The correct answer is D. The image shows free fluid in the hepatorenal recess (Morrison's pouch), which in the context of trauma can be assumed to be blood. The question describes a patient in shock, which mandates immediate intervention for hemorrhage control. With free blood in the peritoneal cavity, control is best achieved by diagnostic and therapeutic laparotomy. The patient is too unstable to undergo CT scanning. Pelvic fractures more commonly result in retroperitoneal bleeding than intraperitoneal bleeding, and the former is not visible on the FAST (Focused Assessment with Sonography for Trauma) exam. The patient is too unstable for oral contrast studies, and the presence of a perforated viscus should be determined during laparotomy for this patient. The image provides no data on the condition of the pericardium.

12. The correct answer is D. The ultrasound shows the presence of hydronephrosis, which may be physiologic during late pregnancy—resulting from fetal compression of the ureter. This finding does not mandate urgent urologic consultation and is not specific for obstructive urolithiasis, nor does it exclude the possibility of appendicitis. The finding of hydronephrosis alone does not require follow-up CT scan, although evaluation of the patient's right lower quadrant pain might require further imaging procedures.
13. The correct answer is C. The image shows the necrotic wound characteristic of envenomation by a *Loxosceles* spider, most commonly the brown recluse spider in the United States. Surgical debridement should be delayed for several weeks until the margins are clear, if indeed it is considered at all. While antivenin for this *Loxosceles* does exist, it is not commercially available in the United States and is not indicated for treatment. Antibiotics are indicated for any symptoms of infection that accompany the bite, which may be considered in this case as the area of surrounding erythema may indicate concurrent cellulitis. Antivenin therapy, opioids, and benzodiazepines form the backbone of treatment for envenomation by *Latrodectus* (black widow) spiders. Immersion of envenomated extremities in hot water is indicated only for heat-labile toxins, such as those elaborated by many marine animals.

14. The correct answer is E. The image and clinical picture together suggest the diagnosis of Henoch-Schönlein purpura (HSP). The rash is usually limited to the lower extremities and buttocks. Hematuria, gross or microscopic, is a common associated finding in HSP. Thrombocytopenia from idiopathic thrombocytopenic purpura (ITP) and thrombotic thrombocytopenic purpura (TTP) may cause other petechial/purpuric rashes, but HSP is typically associated with mild thrombocytosis. The pancreas is not typically involved in HSP, so the lipase level should be normal. There are no coagulation defects reflected in abnormal prothrombin or partial thromboplastin times (PT/PTT) in patients with HSP. Positive antinuclear antibody titers (ANA) would be seen in systemic lupus erythematosus (SLE) or possibly in juvenile rheumatoid arthritis (JRA) but are not elevated in HSP.

15. The correct answer is A. The lesion is the chancre of primary syphilis, and the treatment of choice is single-dose IM penicillin. Acyclovir is the treatment for herpes infections, which are clinically recognized as crops of shallow genital ulcers. Azithromycin or ceftriaxone may be used to treat chancroid, which is a painful genital ulcer. Podofilox is the treatment for genital warts.

16. The correct answer is E. The linear lesion is a burrow caused by the scabies mite, which is treated with application of permethrin cream from the neck down. The web space involvement is also highly characteristic of scabies. Selenium sulfide is used to treat seborrheic dermatitis and tinea versicolor. Clotrimazole is a topical antifungal agent for cutaneous fungal infections. Hydrocortisone is a low-potency topical steroid, and fluocinonide is a high-potency (fluorinated) topical steroid. Both are used to treat a variety of inflammatory dermatoses; the choice depends on severity of the condition and skin thickness at the site of involvement.

17. The correct answer is C. The image shows generalized erythroderma, a sunburn-like appearance, which can be associated with staphylococcal scalded skin syndrome (SSSS), toxic epidermal necrolysis (TEN), toxic shock syndrome, and Hodgkin’s disease. The lateral pressure on the skin causing wrinkling and peeling is Nikolsky’s sign, which occurs in both TEN and SSSS as well as pemphigus vulgaris. The presence of Nikolsky’s sign on areas of skin unaffected by erythroderma, as well as the patient’s young age, are highly suggestive of staphylococcal scalded skin syndrome. SSSS is caused by an exotoxin elaborated by some, but not all, strains of *Staphylococcus aureus*. Pemphigus vulgaris is caused by antibodies to cell adhesion molecules (desmogleins—the same target affected by *Staphylococcus aureus* exotoxins) but is not associated with erythroderma. Medications are a frequent cause of TEN, but a medicine taken 2 months ago is unlikely to result in dermatologic manifestations so far removed from ingestion. Nikolsky’s sign in TEN is typically positive only in areas affected by the erythroderma; mucous membrane involvement is much more common (over 90% of cases). Cutaneous diphtheroids are a cause of erythrasma, a chronic dermatosis that gets its name from the coral red fluorescence of cutaneous lesions but does not result in erythroderma. Parvovirus B19 causes erythema infectiosum (fifth disease), which may result in erythematous cheeks (“slapped cheek” appearance) but not generalized erythroderma or Nikolsky’s sign.
18. The correct answer is B. The image shows an empty uterus with complex free fluid in the pelvis, which is most consistent with a ruptured ectopic pregnancy. At 7 weeks, if the dates are correct, fetal structures, including cardiac activity, should be present on ultrasound. Even if the dates are incorrect, with a quantitative hCG over 2,000, at least a gestational sac should be observable by ultrasound. The empty uterus makes normal pregnancy and blighted ovum unlikely. Ruptured ovarian cyst may be hemorrhagic and could cause complex pelvic free fluid, but with an hCG at this level, some structures should be present in the uterus. A molar pregnancy causes a “snowstorm” appearance in the uterus and markedly elevated hCG levels.

19. The correct answer is E. The presence of multiple small painful vesicles that quickly unroof and become erosions strongly suggests genital herpes. While herpes simplex virus type 2 more commonly causes genital infections, lesions caused by herpes simplex virus type 1 are identical in appearance and may be caused by oral-genital contact. Treponema pallidum is the spirochete that causes syphilis, which presents as a painless chancre that is usually single. Hemophilus ducreyi causes chancroid, characterized by painful ulcerations 1 to 2 cm in diameter. Chlamydia trachomatis causes lymphogranuloma venereum, a disease in which the primary chancre is painless and rarely noticed by the patient. Calymmatobacterium granulomatis causes donovanosis, in which the lesions are painless highly vascular ulcers.

20. The correct answer is B. Cycloplegia prevents ciliary spasm and enhances pain control for large (>2 mm) corneal abrasions, such as shown here. Topical steroids or beta-blockers are not indicated. Because of the risk of Pseudomonas infection, patching the eye should be avoided in contact lens wearers with corneal abrasions. Topical acyclovir drops are indicated only for herpetic corneal infections. No characteristic dendritic lesions are seen in this patient.

21. The correct answer is E. The image shows a wound to zone II of the neck. Zone I extends from the thoracic inlet to the cricoid cartilage. Zone II extends from the cricoid cartilage to the angle of the mandible. Zone III extends from the angle of the mandible to the base of the skull. There is some controversy about the management of zone II injuries in terms of whether all wounds that violate the platysma should be explored in the operating room or observed following negative angiography and esophageal studies. If a nonoperative approach is elected, both vascular and esophageal studies must be completed, as well as bronchoscopy if any airway injury is suspected. Neck wounds should never be probed in the emergency department if the platysma has been violated, because an underlying hematoma might be destabilized.

22. The correct answer is D. The image shows an L1 compression fracture. The posterior elements of L1 are difficult to visualize on this film, but the clinical vignette combined with the image suggests the possibility of a Chance fracture, which typically occurs at the thoracolumbar junction via a hyperflexion mechanism (as around a lap belt with rapid deceleration in a motor vehicle crash). The anterior column is compressed, and the middle and posterior columns are distracted. Many of these injuries are initially misdiagnosed as simple compression fractures, so a Chance fracture should be sought with CT if the mechanism of injury is appropriate. Chance fractures are only rarely associated with neurologic findings, but they are frequently associated with intra-abdominal injuries such as splenic and liver lacerations, pancreatic trauma, and small bowel injuries, so the patient may have significant abdominal tenderness on exam and should be imaged with a CT scan of the abdomen.

23. The correct answer is C. The fracture involves only the epiphysis and physis, so this is a Salter-Harris III injury. The Salter-Harris classification of pediatric fractures ranges from grade I (physeal injury only, no growth problems) to grade V (severe crush injury to the physis with growth arrest likely). Salter-Harris II fractures (fracture line through physis and metaphysis) are the most common fractures involving the growth plate. The likelihood of growth arrest increases with higher grades of injury.
24. The correct answer is C. The image demonstrates exophthalmos, which is suggestive (but not pathognomonic) of ophthalmic disease associated with hyperthyroidism secondary to Graves disease. There is no periorbital edema or erythema to suggest periorbital/orbital cellulitis. Although several drugs of abuse cause tachycardia, none causes exophthalmos. High intraocular pressures (glaucoma) may cause tachycardia secondary to pain but do not cause exophthalmos. Digoxin toxicity may cause a variety of tachydysrhythmias and bradydysrythmias, might affect color vision perception, but does not cause exophthalmos.

25. The correct answer is A. Molluscum contagiosum is caused by a poxvirus (as are smallpox [variola virus] and vaccinia [vaccinia virus]). It is common in school-aged children. Lesions will remit without treatment after several weeks or months. It is characterized by multiple umbilicated papules from 1 to 5 mm in size. It may be transmitted sexually or by direct contact (such as on wrestling mats) and then spreads by autoinoculation. Lesions of smallpox may also be umbilicated but are preceded by a prodrome of fever, myalgias, and headache. Varicella-zoster lesions typically are in multiple stages (macule, papule, vesicle, crust) at different sites on the body and do not typically umbilicate. Herpes simplex lesions are clear vesicles that unroof to become shallow erosions and then crust over. Vaccinia lesions progress from papule to vesicle to pustule to scab, but patients with vaccinia at sites distant from the typical immunization location (over the deltoid) usually have systemic signs of illness.

26. The correct answer is C. It is reasonable to initiate anticoagulation therapy in a patient with a high probability of deep venous thrombosis. However, warfarin should not be initiated alone, because it can result in a transient hypercoagulable state that will promote embolization and propagation of the thrombus. Duplex ultrasound is noninvasive and is approximately 95% sensitive and specific. Venography is painful and invasive and may result in an iatrogenic venous thrombus. Several disorders (infection, trauma, recent surgery, pregnancy, and malignancy) can cause the D-dimer level to be elevated; therefore, further testing with ultrasound should follow an elevated D-dimer level.

27. The correct answer is C. This child has an asymptomatic umbilical hernia. These generally do not incarcerate and usually close by 3 years of age. No special therapy is required.

28. The correct answer is D. In general, an isolated frontal or occipital contusion should not cause the ipsilateral findings described. A cerebellotonsillar or transtentorial herniation causes bilateral fixed, pinpoint pupils.

29. The correct answer is B. Hypoxia is a late finding in severe exacerbations of asthma. This patient is in respiratory failure. He has already demonstrated that he will not tolerate inhaled treatments (choice E). Medicating his agitation (choice A) does not treat its cause. Steroids (choice D) should be given, but this patient cannot wait for them to take effect. Subcutaneous beta-agonists (choice C) may be useful when nebulized beta-agonists are not tolerated, but they do not have instant onset. There is no replacement for direct respiratory support when it is immediately required, as in this case.
30. The correct answer is B. Barotrauma can occur with high pressures (plateau over 30 mm). When you decrease the I:E ratio, you increase the fraction of a breath cycle allotted for exhalation. This allows complete exhalation, which prevents breath stacking. You can disconnect the endotracheal tube from the ventilator and manually assist an exhalation by gently compressing the thorax; this both assesses the degree of breath stacking and temporarily decompresses the accumulated pressure. Increasing the inspiratory flow rate (not decreasing it, as in choice E) also extends the proportion of time in a breath cycle that allows exhalation, although an excessive rate will lead to high peak pressures. The number of breaths per minute may need to be decreased (even to <10) to allow adequate exhalation; tidal volumes should be set at 6 mL/kg for the same reason. While pressures fall into the safe range, the patient's PaCO₂ may rise due to decreased minute ventilation. This should be tolerated within small deviations of pH; it is called “permissive hypercapnia” because hypercapnia is allowed to persist in order to decrease barotrauma risk. An arterial blood gas (choice A) should be ordered to monitor the pH but will not be the best single reference for reducing barotrauma. Ventilator dysynchrony can occur if the patient is “fighting” or “bucking” the ventilator. Benzodiazepine sedation (choice C) is helpful to prevent this, but a paralyzed patient (who should also receive sedation) cannot produce this cause of high pressure.

31. The correct answer is A. This patient may have enterohemorrhagic *E. coli* (0157:H7), which is characterized by grossly bloody stools and abdominal pain with the absence of fever. Empiric antibiotics should be avoided in nontoxic children with bloody diarrhea without fevers until cultures return, as antibiotics may enhance toxin release and increase the likelihood of hemolytic-uremic syndrome with *E. coli* 0157:H7.

32. The correct answer is C. Lidocaine is an amide local anesthetic, most easily identified by the fact that all amide local anesthetics have two of the letter “i” in their generic names. Procaine is an ester anesthetic and should not cause an allergic reaction in a patient allergic to amide anesthetics.

33. The correct answer is B. This is an auricular hematoma, which should be drained in the ED. Auricular hematomas should be re-assessed in 24 hours to check for reaccumulation of blood and the need for repeat drainage. Failure to drain the hematoma can result in deformity of the auricle.

34. The correct answer is D. Lacunar infarcts cause pure motor or sensory deficits as a result of the infarction of small penetrating arteries. They are commonly associated with chronic hypertension. Lesions are primarily located in the pons and the basal ganglia. They do not affect level of consciousness.

35. The correct answer is B. This patient has meningococcemia. He is lethargic with altered mental status and is febrile and hypotensive. In addition, he has the characteristic peripheral petechial rash of meningococcemia. Chronic alcohol abuse and splenectomy place him at great risk for this life-threatening infection. Additional risk factors for meningococcemia include a recent respiratory illness, complement deficiency, and corticosteroid use. Ten to twenty percent of patients with meningococcemia develop bilateral adrenal hemorrhage, also known as Waterhouse-Friderichsen syndrome, which is characterized by rapid deterioration with circulatory collapse and shock. The clinical clue to acute adrenal insufficiency is the lack of blood pressure improvement despite adequate volume resuscitation.

36. The correct answer is D. Appropriate therapy for pericarditis comprises nonsteroidal anti-inflammatory agents, hydration, and reassurance. Aspirin, nitroglycerin, and morphine would be appropriate for chest pain secondary to acute coronary syndrome. Thrombolytics would be appropriate for an ST-segment elevation MI and antibiotics for pneumonia.

37. The correct answer is D. The first three answers are contraindicated in any significant caustic ingestion. Endoscopy should be performed to assess the degree of damage and to evaluate for perforation. Antibiotics are indicated only if perforation is confirmed, either radiographically or by endoscopy. They should not be administered routinely to every patient with a caustic ingestion.
38. The correct answer is C. Cerebral perfusion pressure equals the mean arterial pressure minus the intracranial pressure.

39. The correct answer is A. Patients with creatinine phosphokinase elevations in excess of five times the reference range with appropriate clinical presentations should be suspected of having rhabdomyolysis. Urine tests for myoglobin (heme-positive dipstick without RBCs) are insensitive and will miss up to 50% of patients with rhabdomyolysis. Acute renal failure develops in 30% to 40% of patients with rhabdomyolysis. Predictors for the development of renal failure include severe dehydration, peak CK level more than 6,000 IU/L, sepsis, hyperkalemia, hyperphosphatemia, and hypoalbuminemia. The primary treatment of rhabdomyolysis is saline diuresis to maintain a urine output of 2 to 3 cc/kg/hr. Once established, this may be augmented by urinary alkalinization (clinical evidence lacking), mannitol administration to enhance renal perfusion, or a loop diuretic such as furosemide to enhance urinary output.

40. The correct answer is C. Neuroleptic malignant syndrome (NMS) is characterized by hyperthermia, autonomic instability, muscle rigidity, decreased level of consciousness, and rhabdomyolysis (leading to an elevated CK). It is a life-threatening side effect of both typical and atypical neuroleptic medications. NMS can also cause coagulopathies, liver and kidney dysfunction, respiratory failure, and gastrointestinal bleeding. Treatment consists of supportive measures, fever reduction, IV fluids for rehydration, and administration of dantrolene, a direct-acting muscle relaxant.

41. The correct answer is A. In obvious cases of testicular torsion, emergent urologic consultation and surgical exploration are recommended. While awaiting transportation of the patient to the operating room, the emergency physician should attempt manual detorsion of the affected testis.

42. The correct answer is B. A salivary gland toxin from *Dermacentor* and *Ixodes* ticks causes tick paralysis. There is a typical ascending paralysis, ataxia, and loss of deep tendon reflexes. The mortality rate is approximately 10%. The primary treatment is to remove the tick.

43. The correct answer is D. This patient is hypoglycemic and is taking an oral hypoglycemic agent. His depression should prompt consideration of an intentional overdose of glyburide. Octreotide decreases insulin secretion from the pancreas and has been found useful in patients who overdose on oral hypoglycemic agents. This agent, administered in a dose of 50 to 100 micrograms subcutaneously every 6 to 12 hours, is effective at sustaining serum glucose levels.

44. The correct answer is A. The patient is having an acute febrile reaction, probably from an antibody response to the donor cells. You must stop the transfusion and initiate a transfusion reaction workup to ensure this is not the beginning of a more severe hemolytic transfusion reaction. Steroids and antipyretics are indicated to relieve the symptoms and stop the antibody response. Continuation of the transfusion could potentially endanger this patient, as it is difficult to distinguish a febrile reaction from hemolytic reaction early in the reaction.

45. The correct answer is D. When a patient is a late presenter, usually defined as after 8 hours, intravenous N-acetylcysteine should be started based on history alone. The infusion can always be stopped if the level comes back below the nomogram. In this case, a level warranting concern would be above 37.5 mg/dL.

46. The correct answer is A. This patient’s presentation is highly indicative of a ruptured ectopic pregnancy. OB/GYN consultation should be initiated immediately, before confirmatory laboratory and radiologic test results are available.

47. The correct answer is D. This patient has necrotizing fasciitis. Two types exist: type 1 and type 2. This patient has type 2 necrotizing fasciitis, which is typically caused by group A streptococci and is often located on the extremities. It is characterized by the abrupt onset of erythema, edema, and pain out of proportion to examination findings. Initial radiographs may be misleading, as gas is an inconsistent finding. The most important step in the management of patients with necrotizing fasciitis is surgical consultation—patients require urgent debridement.
48. The correct answer is B. This patient has one contraindication to thrombolytic therapy for stroke in that he had a bleeding ulcer within 21 days before presentation. Otherwise, he met the criteria: CT diagnosis of an ischemic stroke within 3 hours after the onset of symptoms, an acceptable blood pressure, and persistent neurologic deficits. Other exclusion criteria include cerebral hemorrhage; resolving symptoms; bleeding diathesis; stroke, significant head trauma, or intracranial surgery within the preceding 3 months; active internal bleeding; or major trauma/major surgery within the past 14 days. In light of the contraindication, supportive care is the recommended therapy.

49. The correct answer is C. Mastoiditis may at times be difficult to identify clinically. CT scan is the imaging modality of choice to radiographically confirm mastoiditis.

50. The correct answer is E. Severe *Toxicodendron* dermatitis (exposure to poison ivy, oak, and sumac) is often best treated with a 2-week taper of oral steroids. A shorter course may be followed by a recurrence of the rash. Topical steroids have limited effectiveness. Facial, genital, or widespread involvement is an indication for systemic therapy.

51. The correct answer is D. Doppler ultrasonography is the diagnostic study of choice in pregnant patients with suspected deep venous thrombosis; it is highly sensitive and specific and does not expose the fetus to radiation. V/Q scanning and CT pulmonary angiograms are both diagnostic considerations but are less desirable because they expose the fetus to radiation. Venography is the gold standard test but is invasive, involves fetal radiation exposure, and is rarely performed, because of improvements in CT technology. D-dimer testing is unreliable during pregnancy because the pregnant state itself can make the D-dimer abnormal and lead to false-positive studies.

52. The correct answer is D. This patient complains of a number of nonspecific constitutional symptoms and has important risk factors for tuberculosis (working in a jail and having an immunocompromising condition). A significant percentage of patients diagnosed with active pulmonary TB lack pulmonary complaints, and fewer than one third have a pulmonary chief complaint. However, the physical exam is usually abnormal. This man's infiltrate could be related to TB and he should be isolated until it is ruled out. Answers A, B, C, and E do not address his need for isolation.

53. The correct answer is D. This presentation is most characteristic of intussusception. This disease frequently presents with lethargy as the chief complaint, thought to be related to endogenous endorphin release with intussuscepting bowel. Currant jelly stools are typically a late finding, whereas occult blood is frequently found in stools that do not appear to be bloody.

54. The correct answer is E. The CDC criteria for the clinical diagnosis of group-A β-hemolytic streptococcus are tonsillar exudates, tender anterior cervical lymphadenopathy, absence of cough, and a history of fever. The consensus opinion is to treat someone who meets three or four of those criteria.

55. The correct answer is D. Most patients with peritonsillar abscess can be treated on an outpatient basis with needle aspiration, antibiotics, and oral analgesics. If needle aspiration fails, incision and drainage or tonsillectomy can be performed.

56. The correct answer is A. Benzodiazepines, particularly lorazepam, are considered the first-line antiepileptic drug of choice in active seizures and status epilepticus in the pediatric population. Pyridoxine can be considered for refractory seizures in newborns.

57. The correct answer is B. Given the information provided, the patient has an Ellis Class II injury. This requires covering the injury site and subsequent dental referral within 24 hours. If the injury included the pulp, immediate referral would be warranted.
58. The correct answer is A. LeFort I fracture is a transmaxillary fracture that runs between the maxillary floor and the orbital floor. It does not involve the orbits. LeFort type II and III both involve the orbits. The comminuted nasal fracture in this case is a separate injury.

59. The correct answer is B. Both octreotide and vasopressin can be used to help control acute variceal bleeding; however, vasopressin is less selective and often causes marked coronary vasoconstriction as well. Therefore, octreotide is a better choice. Antibiotics are appropriate and have been shown to decrease the rate of rebleeding, but they are not the initial therapy. Propranolol is more helpful long term to prevent bleeding from developing. Esophageal balloon tamponade devices may be required, depending on the severity of the bleeding and availability of endoscopic therapy, but a trial of octreotide, volume resuscitation, and blood transfusion should be tried first.

60. The correct answer is E. In any infant under 2 months of age with a rectal temperature ≥38.0°C, a full septic workup should be performed, including urine, blood, and CSF studies. A chest radiograph or stool studies should also be obtained if the patient has respiratory symptoms or diarrhea.

61. The correct answer is C. The maximum dose of lidocaine is 4.5 mg/kg. Lidocaine concentrations are 1 mg/ml for the 1:1,000 concentration and 0.1 mg/ml for the 1:10,000 concentration. So 450 ml of 1:1,000 lidocaine would be 450 mg, or 4.5 mg/kg, and is the maximum safe dose in this patient.

62. The correct answer is A. This patient has mitral stenosis, the most common cause of which is rheumatic fever. Congenital heart disease is the most common cause of aortic stenosis. Infective endocarditis is a cause of acute mitral regurgitation and the most common cause of acute aortic regurgitation. Aortic dissection accounts for the remaining causes of acute aortic regurgitation.

63. The correct answer is C. The patient has a minor injury and should be assigned to a delayed treatment group (the “walking wounded”).

64. The correct answer is C. The stingray stinger often breaks off, and a radiograph is required to look for the foreign body. The sting typically produces pain out of proportion to the physical exam. Stingrays produce a heat-labile toxin, so hot water may be therapeutic. Pain control with opiates may be necessary. Nematocysts are the agent of a jellyfish sting. Fresh water may activate them and therefore should be avoided.

65. The correct answer is C. This patient had diabetic ketoacidosis (DKA). While intravenous fluids and an insulin infusion were appropriate treatments, the patient’s serum potassium was not known at the start of therapy. Both insulin therapy and improvements in serum pH (brought about by hydration and subsequent improvements in tissue perfusion) lower serum potassium by causing transcellular shift. This patient was likely hypokalemic from the very beginning. The insulin drip probably caused a precipitous drop in serum potassium levels, which led to cardiac arrest.

66. The correct answer is C. Chest pain, cough, and fever in a patient with sickle cell disease may be pneumonia or acute chest syndrome. Initial treatment should be with oxygen therapy, intravenous hydration, and broad-spectrum antibiotics. It would be incorrect to send this potentially very ill patient home. Exchange transfusions are indicated for acute hemolytic and aplastic crises.

67. The correct answer is B. The most common cause of nonsexually transmitted bacterial epididymitis in men over age 35 years old is infection with coliform organisms or Pseudomonas species. Gram-positive cocci are also important pathogens. This age group distinction is important not only from the standpoint of therapy but also because bacterial epididymitis in men over age 35 years is commonly associated with underlying urologic pathology.
68. The correct answer is C. Sulfonylurea exposures are long-acting medications that can cause serious morbidity and mortality in children. Any child with possible exposure should be observed for a minimum of 24 hours. If hypoglycemia is present after exposure, the child should quickly receive a bolus of dextrose followed by octreotide subcutaneously every 6 hours for 24 hours. A continuous infusion of dextrose should be discouraged, as glucose may perpetuate the continued release of insulin by the sulfonylurea-poisoned β-islet cell.

69. The correct answer is D. Though all of these medications have various risks associated with them, they all, except for propranolol, can be considered useful for ST-elevation myocardial infarction in the presence of cocaine. Any beta-blocker should be avoided in a patient with signs of acute cocaine exposure, because of the unopposed alpha agonism that may ensue. This may lead to vasoconstriction of coronary arteries and possible worsening ischemia.

70. The correct answer is E. Cauda equina syndrome is a result of sudden compression of multiple lumbar and sacral nerve roots, typically caused by massive central disc herniation. It is also associated with abscess, hematoma, malignancy, and trauma. Although the presentation may be subtle, it typically includes back pain, saddle anesthesia, fecal incontinence, urinary retention followed by overflow incontinence, and multilevel sensory/motor radiculopathies. Exam reveals saddle anesthesia, sensory deficits over the buttocks and upper thighs, lower extremity weakness, and—the most sensitive finding—urinary retention (post-void residual >100–200 ml). Treatment is immediate surgical or interventional decompression.

71. The correct answer is C. Supracondylar fractures account for up to 60% of elbow fractures in children and are typically the result of a hyperextension mechanism. Radial head fractures are uncommon in children and more common in adults. Olecranon fractures are usually the result of direct elbow trauma after falls. Lateral and medial condylar fractures represent a smaller percentage of pediatric elbow fractures.

72. The correct answer is B. Grade 1 hyphemas fill less than one third of the anterior chamber. Grade 2 hyphemas fill one third to one half. Grade 3 hyphemas fill more than half without completely filling it. Grade 4 hyphemas completely fill the anterior chamber ("eight ball" hyphemas). Grade 5 does not exist.

73. The correct answer is D. The total dose of rtPA for treating acute ischemic stroke is 0.9 mg/kg, with a maximum dose of 90 mg. Ten percent of the dose is administered as a bolus, with the remaining amount infused over 60 minutes. Blood pressure should be measured and neurologic checks performed every 15 minutes for 2 hours after starting the infusion.

74. The correct answer is D. The source patient has hepatitis B and his positive e antigen connotes increased infectivity. The resident did not respond to hepatitis B vaccine, so she is essentially non-immune. She should undergo another vaccination series and be treated with HBIG to give temporary passive immunity.

75. The correct answer is A. Any woman presenting after delivery with increasing abdominal pain and fever should be evaluated for endometritis. Endometritis is found in 3% of women after vaginal delivery and in 15% to 30% after cesarean section. A pelvic ultrasound should be done to evaluate for retained products of conception. The patient should be given broad-spectrum antibiotics and should be admitted.

76. The correct answer is B. Fournier's gangrene is a polymicrobial, synergistic, necrotizing infection of the perineal subcutaneous fascia. It has a mortality rate around 20%. Patients with this severe infection are generally immunocompromised, typically have diabetes, and often present with pain out of proportion to the findings on examination. Broad-spectrum antibiotics and prompt surgical consultation are vital.
77. The correct answer is A. It is important to know the side effects of medications used to treat tuberculosis. Isoniazid, used in the treatment of TB as well as in patients with a positive PPD test, can cause hepatitis, peripheral neuropathy, and seizures. Rifampin causes an orange discoloration of bodily fluids; patients should be warned of this side effect. Pyrazinamide can cause polyarthralgias and hepatotoxicity. The classic side effect of ethambutol is retrobulbar neuritis, which typically presents as red-green blindness.

78. The correct answer is B. Other absolute contraindications include suspected aortic dissection, uncontrollable hypertension (>180/110 mm Hg), or history of a hemorrhagic cerebral vascular accident at any time. The other choices are all relative contraindications.

79. The correct answer is D. This patient does not meet criteria for treatment of streptococcal pharyngitis (answer A). Advanced airway equipment should be at the bedside of a patient with suspected epiglottitis, in the event that a surgical airway (answer B) becomes necessary during the course of examination of the epiglottis and supraglottic structures or an attempt at intubation. This patient does not have stridor or respiratory distress, and intubation may not ultimately be necessary but he does need to undergo laryngoscopy. Fiberoptic laryngoscopy (NPL, answer D) is ideal but direct laryngoscopy is also sufficient, though a topical anesthetic and sedation may be required. Paralysis as part of RSI (answer C) should be avoided, as complete relaxation of the pharyngeal muscles may cause complete airway obstruction if significant swelling is present. Blind nasal intubation is similarly contraindicated, as it may cause obstruction rather than bypassing it. Answer E may be sufficient and appropriate after the patient’s airway has been evaluated and epiglottitis has been ruled out. If epiglottitis is diagnosed but the patient has no respiratory distress, s/he may be admitted and observed without intubation but should be watched carefully in an ICU setting so the airway can be managed rapidly should the condition suddenly worsen. In addition to airway management, patients with epiglottitis should also receive antibiotics.

80. The correct answer is B. Shaken baby syndrome occurs in children under 1 year of age. It is caused by a coup-contrecoup injury caused by shaking or other trauma. Infants with this injury may have no external signs of head trauma. CT scan of the head demonstrates subdural or subarachnoid hemorrhage, diffuse axonal injury, and cerebral edema. Funduscopic examination shows retinal hemorrhages. Patients with shaken baby syndrome can also have anterior and posterior rib fractures.

81. The correct answer is E. Pregnant asthmatic patients are treated in the same manner as non-pregnant asthmatics. Untreated asthma is a greater threat to the unborn fetus than that posed by any asthma treatments.

82. The correct answer is C. There is no indication for emergent steroids from direct blunt trauma causing laryngotracheal injury. The patient may require further imaging but requires a secure airway prior to this assessment. Bronchoscopic intubation and tracheostomy are the best airway options. Cricothyroidotomy is not recommended given the location of the blunt injury.

83. The correct answer is B. Ketamine has analgesic properties. It is associated with hypersalivation, which can be prevented by the administration of glycopyrrolate or atropine. Airway reflexes are generally preserved. Emergence reactions occur most commonly in adults. Ketamine has no reversal agent.

84. The correct answer is D. Hepatitis E is a RNA virus that is rarely seen in the United States in non-travelers, but is an enormous health problem in developing countries. It is similar to hepatitis A in that it is transmitted via the fecal-oral route, rarely progresses to fulminant hepatic failure, and has no chronic carrier state. However, it is important to remember that the mortality rate for women in their third trimester of pregnancy is nearly 20%.
85. **The correct answer is C.** A hydrocele is a collection of fluid that accumulates in the tunica vaginalis. Communicating hydroceles form when the upper processus vaginalis fails to obliterate and there is an open tract between the peritoneum and the scrotum. The tract is closed in noncommunicating hydroceles. Most hydroceles are right sided. They may be present at birth but are usually painless and may resolve spontaneously by 18 months of age. Examination with transillumination will reveal enlargement of the scrotum. Asymptomatic patients can be discharged with urologic follow-up.

86. **The correct answer is C.** This patient has inhalational anthrax. He initially had the prodromal symptoms of fever, malaise, and a nonproductive cough. He has had rapid progression of symptoms and now exhibits cardiovascular collapse. His chest film demonstrates the classic abnormality of an enlarged mediastinum resulting from hemorrhagic mediastinitis. Immediate antibiotic therapy is warranted. Acceptable antibiotics for the treatment of anthrax include ciprofloxacin, doxycycline, and penicillin G.

87. **The correct answer is A.** This patient's sarcoidosis, steroid use, and pre-existing cavitary lesion place her at risk of infection with *Aspergillus*. This fungal infection commonly invades cavitary lesions in immunocompromised patients, such as those with a history of TB, *Pneumocystis jiroveci*, or sarcoidosis. The typical chest film appearance is a cavitary lesion, with a fungus ball noted within the cavitation. Life-threatening hemoptysis is a complication of this infection. Patients require anti-fungal therapy and pulmonary consultation.

88. **The correct answer is D.** Stable Wolff-Parkinson-White syndrome with atrial fibrillation is managed with procainamide. Medications that block the atroioventricular node but not the bypass tract are contraindicated (e.g., digoxin, calcium channel blockers, beta-blockers, and adenosine).

89. **The correct answer is B.** Nitroprusside is the agent most commonly recommended because one can obtain a rapid and consistent lowering of the blood pressure to the desired level and adjustments can be made rapidly. It has rapid onset, it can be titrated, and it has no effect on mental status. This patient requires emergent blood pressure control and cannot simply wait. Sublingual nifedipine can cause a precipitous drop in pressure. The transdermal clonidine patch is not effective for acute blood pressure control.

90. **The correct answer is A.** This patient is malingering, which is frequently associated with antisocial personality disorder. Malingering patients often will not cooperate with physical or other diagnostic evaluation, are noncompliant with prior treatment, and have symptoms that do not correlate with objective findings. In malingering, external incentives outside the sick role exist, e.g., financial gain, seeking shelter, evading the law, obtaining pain medications. Drug-seeking patients tend to be manipulative and demanding, to resist non-pharmacologic treatment recommendations, and to "doctor shop," and they may report allergies to multiple non-narcotic agents.

91. **The correct answer is B.** Painless third-trimester bleeding is highly suspicious for placenta previa. Digital examination is contraindicated until this diagnosis is ruled out. A stat pelvic ultrasound should be performed to look for evidence of placenta previa.

92. **The correct answer is B.** The diagnostic criteria for Kawasaki disease is fever of at least 5 days duration with the presence of at least four of the following: bilateral conjunctivitis, changes of the lips and oral mucosa such as strawberry tongue, changes of the extremities such as erythema or edema, polymorphous rash, and cervical lymphadenopathy. Leukocytosis, thrombocytosis, and elevation of other acute-phase reactants are frequently seen but are not part of the diagnostic criteria for Kawasaki disease.
93. The correct answer is D. Classically, Zone 2 penetrating neck traumas are managed with surgical exploration. Zone 1 and 3 injuries are often evaluated with angiography and possibly with subsequent endoscopy or bronchoscopy. Direct laryngoscopy would not likely add any information given the location of this patient's injury.

94. The correct answer is A. "Red on yellow kill a fellow; red on black venom lack." The venomous coral snake has a pattern in which red is surrounded by bands of yellow. Ptosis is an early sign of envenomation, and respiratory failure is the most common cause of death induced by this neuromuscular toxin. Antivenin is indicated in any coral snake bite. The nonvenomous king and scarlet snakes have bands of black surrounding the red areas.

95. The correct answer is E. Given the recent travel to an endemic region, along with fever and profound lethargy, this patient likely has malaria. Malaria is caused by the Plasmodium species, of which P. falciparum causes the most severe disease. Malaria is endemic in Asia, Africa, Central America, and South America. The diagnosis is made by thick and thin peripheral blood smears that demonstrate the organism. Treatment depends on the degree of parasitemia and the likelihood of chloroquine resistance. P. falciparum resistance to chloroquine is increasing, such that quinine plus doxycycline are the drugs of choice for this species.

96. The correct answer is D. Patients with HN may have "false-negative" chest films. This is increasingly common with falling CD4 counts and progression toward AIDS. Although TB can cause massive hemoptysis (>600 mL/24 hr), fewer than 10% of patients with active pulmonary TB have any hemoptysis (answer B). Answer A is incorrect, as this positioning may be beneficial, as is selective intubation to ventilate and protect the unaffected lung. As the volume of the bronchial tree is relatively small, death by hemoptysis is death by drowning, not exsanguination (answer E). TB has a wide variety of radiographic appearances, can be seen in any lobe (usually a single lobe), and is not exclusively in the apices (answer C), although apical disease is certainly suspicious for TB.

97. The correct answer is A. This patient has acalculous cholecystitis, an uncommon entity seen mostly in elderly patients, in children, or in individuals of any age after major illnesses, traumas, or burns. Unlike acute calculous cholecystitis, a "cooling off" period is not recommended. The increased risk of complications mandates early surgery or drainage. Oral antibiotics would be insufficient treatment. While gallstone ileus is responsible for about one quarter of all small bowel obstructions (SBOs) in women older than 64, it presents like an SBO, not like cholecystitis. Mesenteric angiography is not warranted here.

98. The correct answer is C. The treatment of Bell's palsy consists of prednisone for anti-inflammatory effect at 60 mg PO daily or 1 mg/kg/day for 5 days and then tapered over the next 5 days, along with acyclovir, 400 mg five times per day for 10 days and follow-up with either ENT or neurologic consultation. Acyclovir or prednisone alone is not considered as effective as the combination. The prognosis is generally good for total recovery, but patients with total paralysis are at increased risk of long-term or permanent paralysis and should be seen in follow-up within 2 to 3 days. Patients with incomplete paralysis should be instructed to return if the weakness becomes total paralysis.

99. The correct answer is D. Aspiration should be treated with supportive care: supplemental oxygen, nebulized beta-agonists, and possibly chest physiotherapy. Not all aspiration events lead to aspiration pneumonia, and antibiotics should not be prescribed prophylactically after an aspiration event. This patient has a GCS score of 14 and does not need definitive airway protection at this time.
100. **The correct answer is C.** Age over 40 is associated with an increased risk of thromboembolism. Risk factors for thromboembolism include a postoperative state, family history of thrombosis, history of cancer, prolonged immobility, estrogen use, pregnancy, lower extremity or pelvic trauma, and age over 40. An arterial blood gas lacks significant predictive value. Hyperlipidemia is a risk factor for acute coronary syndrome as are smoking, hypertension, and premature family history.

101. **The correct answer is D.** You have to properly identify yourself as a physician and prove that you are licensed. You should offer assistance to the crew and then treat the patient as appropriate. If you want to deviate from the EMS system's protocols, then you may need to contact the medical director.

102. **The correct answer is B.** This patient has alcoholic ketoacidosis. Treatment must include intravenous fluids that contain glucose. Glucose will "turn off" ketogenesis.

103. **The correct answer is B.** This patient is in respiratory distress and has several risk factors for death from asthma, so she should be admitted for close observation. The other options do not address her risk of further clinical decompensation. Steroid course prolongation (answer A) does not have a role here, as she has not even finished the course prescribed before, although recent withdrawal of steroids is a risk factor for death, and so is current use. This patient's asthma may have been precipitated by a viral URI, as is common, but antibiotics (answer C) are not indicated. Other medical, psychiatric, and substance-using comorbidities also increase the risk for asthma-related death. These include a history of prior exacerbations that have been sudden and severe, prior ICU admission (with or without intubation), use of more than two beta-agonist MDI canisters per month (consider this when contemplating answer D), and either two or more admissions in the past year or three or more ED visits in the past year. For patients with chronic persistent, poorly controlled asthma, a daily controller medication may be indicated (for example, inhaled steroid such as fluticasone, possibly in combination with a long-acting beta agonist such as salmeterol); however, these are not indicated as therapy for acute exacerbations. Oral (pill) beta-agonists are not indicated here (answer E).

104. **The correct answer is D.** This patient has a carotid artery dissection. While it may seem counterintuitive to prescribe anticoagulation for a dissection, it helps prevent clot progression at the site of endothelial injury. The dissection will most likely heal without intervention.

105. **The correct answer is E.** The cauda equina ("horse's tail") is the name given to the lumbar and sacral nerve roots that continue on within the dural sac caudal to the conus medullaris. The cause of cauda equina syndrome is usually a ruptured, midline intervertebral disk, most commonly occurring at the L4–L5 level. Tumors and other compressive masses may cause the syndrome as well. Patients generally present with progressive symptoms of fecal or urinary incontinence, impotence, distal motor weakness, and sensory loss in a saddle distribution. Muscle stretch reflexes may also be reduced. The presence of urinary retention is the single most consistent finding, with a sensitivity of 90%. Low back pain may or may not be present.

106. **The correct answer is B.** Crohn's disease often affects the terminal ileum and presents as "pseudo-appendicitis" with a tender, inflammatory mass palpable in the right lower quadrant. Extra-intestinal manifestations such as arthritis or uveitis are common.

107. **The correct answer is D.** While a synovial white blood cell count >50,000 cells/mm³ is strongly suggestive of septic arthritis, a positive Gram stain is diagnostic. Needle-like uric acid crystals are consistent with gout; rhomboid calcium pyrophosphate crystals are consistent with pseudogout.
108. The correct answer is B. A hypertensive emergency is defined as elevated blood pressure with evidence of acute end organ damage. Hypertensive urgency is elevated blood pressure, usually >115 mm Hg diastolic, without findings of end organ damage or dysfunction. Transient hypertension may be a response to pain or anxiety.

109. The correct answer is D. This is presumed Rocky Mountain Spotted Fever (RMSF). RMSF is most prevalent in the southeastern United States. This patient has the classic peripheral petechial rash that starts on the wrists, forearms, ankles, and soles and spreads centripetally over 6 to 12 hours. Antibiotic therapy should be started at the appearance of the rash. The antibiotics of choice for RMSF are tetracycline, doxycycline, and chloramphenicol.

110. The correct answer is D. This patient most likely has adrenal crisis from congenital adrenal hyperplasia, as evidenced by his presentation, laboratory abnormalities, and unresponsiveness to IV fluid administration. Sepsis can mimic adrenal crisis, so empiric antibiotics are appropriate for a patient with this presentation. Congenital heart disease such as coarctation of the aorta is unlikely given the lack of murmur and normal femoral pulses.

111. The correct answer is B. The clinical scenario suggests the development of tension pneumothorax, which may have resulted from subclinical pneumothorax related to the positive-pressure ventilation or to the flight itself.

112. The correct answer is C. Ellis class III dental fractures result in exposure of the pulp. Since the pulp is the only vascular component of the tooth, a pink tinge or drop of blood is typically seen when it is exposed. Class I fractures involve only the enamel and do not usually cause pain or temperature sensitivity. Class II fractures involve exposed dentin that is creamy yellow; the patient experiences pain and temperature sensitivity. Class IV and V fractures do not exist.

113. The correct answer is D. This patient has the classic triad for acute mesenteric ischemia: abdominal pain out of proportion to examination, gut emptying, and underlying cardiac disease (especially atrial fibrillation). The only therapy proven to reduce mortality in acute mesenteric ischemia is emergent angiography before the onset of peritoneal signs. Angiography should not be delayed for other less useful imaging. Heparin is indicated after discussion of its timing with the treating surgeon. Surgery will almost assuredly be required to revascularize, and intra-arterial papaverine will be required to treat the associated vasospasm, but neither is proven to reduce mortality, especially if the diagnosis is delayed. Digoxin may worsen splanchnic vasoconstriction and should be avoided.

114. The correct answer is C. ST-segment elevation in leads I and aVL as well as V5 and V6 are consistent with a lateral infarct. An anterior injury pattern is seen in leads V1 through V4. ST elevation in leads V1 and V2 is consistent with a septal injury, while elevation in II, III, and aVF is representative of an inferior infarction. A posterior infarct can be recognized by ST depression in leads V1 and V2.

115. The correct answer is A. Hemophilia patients can develop delayed bleeding 8 to 72 hours after an injury. The degree of severity of the hemophilia dictates the initial dose of factor replacement but should not change the decision to observe for late bleeding.

116. The correct answer is E. This patient has signs and symptoms consistent with salicylate toxicity. The patient's arterial blood gas shows mixed respiratory alkalosis and metabolic acidosis. The patient also has evidence of acute lung injury as well as alteration of mental status. This patient requires immediate nephrology consultation and hemodialysis.

117. The correct answer is E. Vaginal bleeding in all postmenopausal women requires close follow-up to rule out the possibility of endometrial carcinoma. This is most commonly diagnosed with an endometrial biopsy.
118. The correct answer is C. The presentation of an acute traumatic aortic dissection can be variable, with only 25% of patients describing the classic tearing intrascapular pain. The exam is frequently unrevealing but may include pulse deficits, precordial murmur, pseudocoarctation, voice changes, and paraplegia. The chest radiograph is normal in almost 30% of presentations.

119. The correct answer is D. High-frequency ultrasound probes best show superficial structures, while low-frequency probes show deeper structures, such as the aorta. Air-filled structures, such as bowel, impair imaging with ultrasound.

120. The correct answer is B. Discontinuation of the offending antibiotic and treatment with oral metronidazole remain the mainstay of treatment for C. difficile infection. Oral vancomycin would be a second-line agent. Antimotility agents may predispose the patient to toxic megacolon and should be avoided. Colectomy is reserved for severe cases unresponsive to standard treatment or for complications such as perforation of toxic megacolon.

121. The correct answer is A. Legionella often presents with high fevers and dry cough and classically causes the extrapulmonary symptoms of diarrhea and mental status changes. It may cause mild disease, or it may progress to ARDS. It may be responsible for up to 1 in 5 cases of community-acquired pneumonia (CAP), and although it is not transmitted from person to person, outbreaks have been traced to indoor water sources. The antibiotic selected should be a macrolide, doxycycline, or a respiratory fluoroquinolone; beta-lactams are not effective, but empiric treatment may be added to cover the other possibilities. Pneumocystis infection (choice B) is associated with HIV-positive patients and is considered an AIDS-defining illness. Klebsiella infection (choice C) produces "currant jelly" sputum and is found in those with risk factors for aspiration, such as the elderly, alcoholics, and diabetics with some degree of gastroparesis. Varicella pneumonia (choice D) can be a severe disease, particularly in adults, and is treated with acyclovir. Its radiographic appearance is usually that of diffuse nodules. Pseudomonas (choice E) pneumonia is unusual outside the nursing home or hospital-acquired settings and classically produces foul green sputum.

122. The correct answer is C. The majority of patients seek treatment days to weeks after resolution of a respiratory or gastrointestinal illness and present in the ED with progressive, symmetric weakness of proximal and distal musculature. Signs and symptoms are worse in the lower extremities and are associated with diminution or loss of deep tendon reflexes, variable sensory findings, and sparing of the anal sphincter. Urinary retention from autonomic dysfunction may occur, contributing to a clinical picture easily mistaken for a spinal cord lesion or conus medullaris syndrome.

123. The correct answer is C. A physician's clinical suspicion is an accurate determinant of coronary artery disease. Patients of any age with an appropriate story and risk factors for coronary disease need a complete workup to rule out acute coronary syndrome, including admission to the hospital for serial ECGs and biomarkers. The initial ECG is nondiagnostic in more than 50% of patients eventually diagnosed with acute coronary syndrome. Troponin rises in approximately 3 to 6 hours, peaks in 12 to 24 hours, and stays elevated for 7 days.

124. The correct answer is D. The extravasation of contrast material from a urethral disruption appears as a flame-like density outside the urethral contour. Any contrast present in the bladder is diagnostic of a partial urethral injury; a complete injury would not allow any contrast into the bladder. The extravasation of contrast material from an extraperitoneal bladder injury appears as flame-like areas of contrast in the pelvis, projecting lateral to the bladder. Intraperitoneal bladder injuries fill the paracolic gutters and outline intraperitoneal structures, especially the bowel, spleen, or liver. Extravasation of contrast is never a normal finding on a cystourethrogram.
125. The correct answer is A. Patients with traumatic cardiac contusions are at risk of developing dysrhythmias, tachycardia, conduction delays, and cardiogenic shock. In the absence of shock or evidence of ischemic injury, an echocardiogram is a poor screening test. Serial biomarkers are nonspecific and not clinically helpful in the absence of other findings or complaints.

126. The correct answer is A. Most inhaled irritants require just supportive treatment (including supplemental oxygen) or nebulized bronchodilators (answers B and E) if wheezing is present. However, phosgene and other low-solubility irritants (such as nitrogen oxides) can cause distal airway complications and even delayed pulmonary edema, so it is prudent to admit these patients for observation if the exposure is deemed significant or if prolonged symptoms are expected. Methylene blue (answer D) and INO (answer C) have no role in the treatment of this exposure.

127. The correct answer is D. This patient likely has an acute coronary syndrome caused by cocaine abuse. β-Blockade for acute coronary syndrome is contraindicated in patients intoxicated with cocaine, because of the potential for unopposed α-adrenergic coronary vasoconstriction.

128. The correct answer is A. A black widow spider (Latrodectus) bite is associated initially with a pinprick sensation followed by muscle rigidity that can imitate an acute surgical abdomen. Treatment consists of wound care, analgesia, muscle relaxants (usually benzodiazepines), and nitroprusside for severe hypertension. Antivenin is administered to pregnant women, children, the elderly, and those with severe envenomations. Antibiotics and corticosteroids have no role in the management of Latrodectus envenomation.

129. The correct answer is D. This patient’s thrombocytopenia is probably either drug induced (trimethoprim/sulfamethoxazole) or idiopathic. Platelet transfusions for ITP are only going to worsen the degree of the thrombocytopenia. The platelet count is too low for safe outpatient evaluation. IVIG is a reasonable option for ITP, but only after a complete hematologic evaluation. Plasma exchange with FFP is indicated for the treatment of TTP.

130. The correct answer is C. Unstable pelvic ring fractures (combination open book/posterior vertical shear > posterior vertical shear alone > open book alone) are associated with significant hemorrhage and mortality up to 50% in those with shock. Initial management includes resuscitation with intravenous crystalloid fluid and blood, application of an external compression belt, and a search for associated injuries to other intra-abdominal structures, especially the aorta, spleen, liver, urethra, bladder, rectum, vagina, and diaphragm. For ongoing shock resulting from unstable pelvic fracture alone (in patients with negative FAST, “arterial blush” or large retroperitoneal/pelvic hematoma on CT scan, the need for more than 4 units PRBCs in 24 hours), interventional radiology with arterial embolization is the treatment of choice.

131. The correct answer is D. All of the answers can be an antidote for toxicologic-induced bradycardias. However, in a patient known to be taking digoxin, calcium should be avoided, as there is theoretical danger of cardiac tetany (also known as stone heart). All of the other antidotes would be considered useful.

132. The correct answer is A. The only definitive treatment for pre-eclampsia is delivery. Antihypertensive medications are only temporizing measures until delivery can be performed safely. Diuretics should not be used for edema or hypertension, as they can cause unsafe decreases in intravascular volume.
133. The correct answer is B. This patient has early disseminated Lyme disease. The rash she reported 1 month ago was likely erythema migrans, the first manifestation of Lyme disease. If untreated, the rash eventually resolves. Disseminated Lyme disease typically begins 4 weeks after the onset of the rash. The central nervous system and the cardiovascular system are primarily involved in this second stage of Lyme disease. A peripheral cranial nerve VII palsy is the most common neurologic manifestation of early disseminated Lyme disease, and atrioventricular blocks (AVB) of varying degrees are the most common cardiac manifestation. Temporary pacing may be required for high-grade AVB.

134. The correct answer is B. Febrile seizures plus diarrhea in a child is classic for shigellosis.

135. The correct answer is A. A patient with chronic renal failure who is in cardiac arrest should be assumed to have hyperkalemia and treated accordingly while the usual resuscitative measures are taken. The most rapidly effective treatment for hyperkalemia is intravenous calcium, which transiently reverses the cardiac manifestations of hyperkalemia without altering the serum potassium level or the total-body potassium concentration.

136. The correct answer is E. This patient has myxedema coma. Although treatment with intravenous steroids will be required to prevent adrenal failure, treatment with thyroid hormone replacement must be initiated as soon as possible. In general, 400 to 500 micrograms of intravenous thyroxine need to be given in the ED.

137. The correct answer is B. This child appears to have been exposed to a caustic. As with almost any problem in emergency medicine, securing the airway is the most crucial first step. This child has stridor, which is an ominous indicator of upper airway edema. Evaluation of the vocal cords and upper airway should take priority over the other injuries that may exist. Intravenous steroids may have a role in decreasing upper airway edema but have an undecided benefit for mitigating esophageal injury.

138. The correct answer is E. The goal of management for thoracic aortic dissection are two fold: reducing blood pressure and decreasing the rate of rise of arterial pressure and shearing stress on the aorta. Sodium nitroprusside reduces blood pressure by acting as a potent vasodilator. When used as a sole agent, it can result in reflex tachycardia. A beta-blocker should be administered along with sodium nitroprusside to prevent reflex tachycardia. It is important to correctly diagnose an aortic dissection, because the treatment for myocardial ischemia or infarction as well as for pulmonary embolism is lethal in a patient with a thoracic dissection.

139. The correct answer is B. Pyloric stenosis is more common in first-born males. Hypochloremic hypokalemia is caused by vomiting, which is non-bilious, since emesis material originates proximal to the pylorus. Ultrasound or an upper GI would be the study of choice.

140. The correct answer is D. The edrophonium test is performed by measuring the distance from the upper to the lower eyelid in the most severely affected eye before and after intravenous administration of the short-acting acetylcholinesterase inhibitor edrophonium. An IV test dose of 1 to 2 mg is given first. Muscle fasciculations and respiratory depression within a few minutes suggest that the muscle weakness is related to a cholinergic crisis; in this case, further edrophonium administration is contraindicated. If no adverse reaction is found, and the patient does not dramatically improve in 30 to 90 seconds, a second dose of 3 mg is given. If there is still no response, a final dose of 5 mg is given for a total maximum dosage of 10 mg in order to demonstrate benefit in the face of a presumed myasthenic crisis.

141. The correct answer is D. This patient has substantial blunt injuries and developing pulmonary contusions. Placing this patient with the normal lung down may allow improved ventilation-perfusion matching.
142. The correct answer is D. The short incubation period and exposure to mayonnaise-containing food is classic for diarrhea from the ingestion of the preformed toxin of *Staphylococcus aureus*. Antibiotics are not useful, there is no carrier state, and symptoms last only 1 day on average. Hemolytic uremic syndrome is associated enterohemorrhagic *E. coli*.

143. The correct answer is D. If the patient is not capable of understanding the risks and benefits of treatment or if he is impaired in any way, then he is not able to refuse treatment. This is a situation where you should get law enforcement to assist with transport to the hospital for evaluation. The EMS crew may need to restrain the patient for transport to the hospital.

144. The correct answer is B. Class 1 anti-arrhythmic medications are sodium channel blockers that work by decreasing conduction through the conduction system and the cardiac muscle. Class1A agents are medications such as quinidine and procainamide. Class 1B agents are medications such as lidocaine. Class 2 agents are beta-blockers. Class 3 agents are antifibrillatory agents (e.g., bretylium). Class 4 agents are calcium channel blockers.

145. The correct answer is C. Wernicke's encephalopathy is caused by thiamine deficiency and is characterized by a rapid onset of delirium, ataxia, and oculomotor dysfunction, such as ophthalmoplegia and nystagmus. The most common cause of Wernicke's encephalopathy is chronic ethanol abuse, but it can also be the result of long-term malnutrition from other disease states (e.g., AIDS) or have iatrogenic causes. It is associated with high mortality, mostly from other alcohol-related diseases. Treatment includes repletion of thiamine and other electrolyte abnormalities as well as supportive care.

146. The correct answer is D. Lateral third clavicular fractures are associated with blunt trauma to the top of the shoulder. Medial third clavicle fractures are frequently associated with direct force to the lateral aspect of the shoulder. This patient lacks the common findings of flail chest, chest wall contusions, and sternal fractures.

147. The correct answer is D. RBC counts over 10 cell/µL are abnormal. Elevated RBC counts may be caused by traumatic LP, herpes simplex virus (HSV) meningoencephalitis, or intracranial hemorrhage. In traumatic LP, the RBC count should decrease from tube 1 to tube 3. Xanthochromia occurs when the supernatant of centrifuged CSF is yellow-orange and is a sign of RBC breakdown products from subarachnoid hemorrhage. Traumatic LP does not cause xanthochromia. WBC counts over 5 cells/µL are pathologic in normal adults and should raise suspicion of infection.

148. The correct answer is A. The combination of vomiting, diarrhea, and neurologic symptoms, especially hot-cold reversal, is classic for ingestion of a ciguatoxin.

149. The correct answer is B. This patient has air embolism from his recent dialysis session. Patients will present with chest pain and shortness of breath or may present in cardiac arrest. The patient should be placed supine and in the left lateral decubitus position in an attempt to have the air embolus settle in the right atrium. Other suggested treatments include percutaneous aspiration of the right ventricle, intravenous steroids, and hyperbaric oxygen therapy.

150. The correct answer is C. The best treatment for Type I decompression sickness ("the bends") is hyperbaric oxygen to wash out retained nitrogen. Aspirin may be helpful but is not the primary management. Prior to hyperbaric treatment, 100% oxygen should be administered.

151. The correct answer is C. Patients with thyroid storm require several medications to control the disease. As a rule of thumb, patients need to be treated with antithyroid medications, like propylthiouracil, before iodide therapy is initiated. Administration of iodide before antithyroid medications may worsen the disease.
152. The correct answer is C. A patient with a history of lung cancer who presents with the findings described in the question is most likely suffering from a pericardial effusion that is at the cusp of tamponade. Typical ECG findings for a significant pericardial effusion include low voltage and electrical alternans. The patient described is less likely to be suffering from an acute pulmonary embolism or a myocardial infarction.

153. The correct answer is A. The patient presents with anticholinergic poisoning, associated with the jimsonweed plant.

154. The correct answer is B. Pregnancy induces numerous physical changes. These include increased heart rate, cardiac output, blood volume, tidal volume, and glomerular filtration rate and decreased blood pressure and gastric and gallbladder emptying.

155. The correct answer is D. This patient has hypertrophic cardiomyopathy (HCM). The murmur of HCM increases with decreased left ventricular filling (e.g., standing, Valsalva, beta-agonists, amyl nitrate) and decreases with increased left ventricular filling (squatting, passive leg elevation, beta-blockade, and isometric handgrip).

156. The correct answer is C. A kerion is a fungal scalp infection that presents as a significantly boggy, indurated lesion. The most common treatment is oral griseofulvin for 6 to 8 weeks. Griseofulvin has been known to cause transaminase elevations and a disulfiram reaction. A long course of other oral antifungals, such as itraconazole, terbinafine, and fluconazole, may be considered.

157. The correct answer is B. Initial drainage of more than 1500 mL, bleeding >200 mL/hr for 3 or 4 hours, persistent air leak, and persistent hypotension are all indications for operative management of this hemothorax.

158. The correct answer is D. Nasogastric tubes are safe to place in the presence of esophageal varices. They are absolutely contraindicated in patients with facial fractures with cribriform plate injuries. They are relatively contraindicated in patients with coagulopathy, alkali ingestions, and esophageal injury.

159. The correct answer is E. This patient has ulcerative colitis (UC). Patients with UC often have bloody diarrhea, but massive gastrointestinal hemorrhage is rare. As in Crohn's disease, extra-intestinal manifestations are often seen. Patients are at greatly increased risk for colorectal (not small bowel) malignancy and should be screened frequently. Exacerbations should be treated with corticosteroids, not antimotility agents, which may increase the risk of toxic megacolon.

160. The correct answer is C. Open fractures are often complicated by the development of wound infections and osteomyelitis. Several grading schemes have been developed, relating the degree of tissue damage, contamination, and blood supply to the typical bacteria associated with subsequent infections and hence the indicated antibiotic coverage:

- Grade I/II: minimal tissue damage and contamination, neurovascularly intact = skin flora, Staphylococcus and Streptococcus → first-generation cephalosporin
- Grade III/IIIa: significant tissue damage and/or contamination = Staphylococcus and Streptococcus plus gram-negative bacteria → first-generation cephalosporin plus aminoglycoside
- Grade IV/IIIb/c: significant tissue damage and/or contamination and vascular injury/no pulse = Staphylococcus and Streptococcus plus gram-negative bacteria plus Clostridia → first-generation cephalosporin plus aminoglycoside plus PCN.
161. The correct answer is C. Since the patient continues to tolerate noninvasive ventilation and does not appear to be clinically worsening, it is appropriate to reevaluate her after she continues her trial of this therapy. Clinical evaluation and monitoring are usually sufficient, although an arterial blood gas may be useful to identify a trend if it is otherwise unclear how the patient is responding to therapy. Benzodiazepines (answer A) are not the best choice, as many of them can cause respiratory depression. Magnesium (answer B), while potentially useful as adjunct therapy in asthma, is not indicated for COPD exacerbations. Theophylline (answer D) does not have a role in acute ED therapy, though in patients who may be taking it chronically, it is prudent to ensure the drug is at a therapeutic (and not supratherapeutic, and possibly toxic) level. Answer E, intubation, is not yet indicated here.

162. The correct answer is E. The presentation is consistent with acute pericardial tamponade with the presence of Beck’s triade (jugular venous distension, hypotension, and muffled heart tones), an enlarged cardiac silhouette on the chest radiograph, and an ECG with low voltage and electrical alternans. Management of pericardial tamponade includes volume resuscitation, oxygen, and pericardiocentesis if the patient is unstable. Dopamine would serve only as a temporizing measure. Intubation should be performed with caution in pericardial tamponade, as positive-pressure ventilation can result in a reduction of preload, leading to a precipitous decline in blood pressure. Needle thoracostomy is used to treat tension pneumothorax.

163. The correct answer is E. Thoracotomy is preferred over pericardiocentesis in traumatic arrests associated with pericardial tamponade. In pericardial tamponade, the removal of even small amounts of fluid can dramatically improve blood pressure and cardiac output. When performing pericardiocentesis, blind techniques may be used in emergencies. ECG guidance will demonstrate a wide QRS complex with ST elevation (“current of injury”) when the needle is advanced too far and is touching the epicardium. The needle should be withdrawn slightly until the “current of injury” disappears.

164. The correct answer is A. Penetrating trauma and loss of vital signs in the ED is the only clear indication for ED thoracotomy.

165. The correct answer is C. The classic feature of botulism is descending symmetric paralysis. Dysphagia, diplopia, and dysarthria occur early. The toxin inhibits cholinergic output, leading to constipation and urinary retention. There is no pain. Pupils are often dilated and nonreactive.

166. The correct answer is D. A patient with a history of AAA repair and any degree of gastrointestinal bleeding should be presumed to have an aorto-enteric fistula until proven otherwise. Workup should proceed similar to that for a patient with suspected ruptured AAA. If unstable, the patient should undergo emergent surgery.

167. The correct answer is D. Cuffed or uncuffed ETTs are now recommended for infants and toddlers. Tracheal tube size is estimated by the formula \(4 + \frac{\text{age in years}}{4}\). Tracheal tube depth may be estimated by using either the formula \(3 \times \text{tracheal tube size}\) or \(\left(\frac{\text{age in years}}{3}\right) + 12\).

168. The correct answer is E. A patient with a history of a repaired ventricular septal defect does not require endocarditis prophylaxis. Prophylaxis is indicated for patients with prosthetic heart valves (including bioprosthetic and homograft valves), a history of previous infective endocarditis, and unrepaired cyanotic congenital heart disease and those presenting within the first 6 months after repair of congenital heart disease completely repaired with prosthetic material. Amoxicillin is the primary antibiotic for most patients. If the patient is unable to take oral medications, ampicillin may be used. Clindamycin and azithromycin are used in penicillin-allergic patients.
169. **The correct answer is B.** This patient is in the moderate- or high-risk category for pulmonary embolism (PE), so a definitive diagnosis should be pursued. A d-dimer should not have been ordered, as a normal value does not exclude PE with sufficient sensitivity. He has had recent immobilization with increased swelling, which raises suspicion for a DVT in that extremity, he is tachycardic, and there is not a more likely diagnosis that ties together all his symptoms. While a muscle strain (answer C) is possible, it does not explain the cough. Answer D similarly does not adequately explain all his symptoms. This patient is not prohibitively young to have coronary artery disease (answer A), but this cause of his pain is unlikely given his presentation. Answer E does not address his risk of PE.

170. **The correct answer is B.** This patient is infected with *Hantavirus*. It is seen more commonly in the southwestern United States. The animal reservoir is the deer mouse. Infection is acquired through inhalation of rodent urine or feces. The initial prodrome is fever, myalgia, followed rapidly by tachypnea, hypoxia, and fulminant respiratory failure. The chest film shows bilateral infiltrates and ARDS. Treatment is supportive, with attention to respiratory status and oxygenation.

171. **The correct answer is B.** Beck’s triad of hypotension, muffled heart sounds, and JVD is present. The history, along with these findings, suggests pericardial tamponade. Bedside US would be invaluable to assist with this diagnosis and with the procedure as well. Electrical alternans is rarely seen in patients with acute tamponade.

172. **The correct answer is B.** SCFE and LCP have similar presenting symptoms; however, the age of usual presentation is different. SCFE usually presents between 10 and 15 years of age, whereas LCP usually presents under 10 years of age. Transient synovitis usually presents under 5 years of age. This presentation is not consistent with septic arthritis or hip avulsion fracture.

173. **The correct answer is C.** Giant cell arteritis is usually a disease of the elderly, with the vast majority of patients over 50 years of age. Symptoms may include headache, jaw claudication, rapidly progressive decreasing vision, myalgias, and temporal artery tenderness.

174. **The correct answer is D.** Diverticulitis is a common disease with a spectrum that ranges from mild, which can be treated on an outpatient basis, to abscess formation with frank perforation and sepsis. Pyuria is a common finding if the area of inflammation is near the bladder or ureters, and this should not dissuade the astute clinician from making the correct diagnosis. The test of choice is a CT scan. Nearly 40% of patients sick enough to be hospitalized will require surgery.

175. **The correct answer is D.** The majority of cases of infective endocarditis in intravenous drug users are secondary to *Staphylococcus aureus*. Less than 35% of intravenous drug users have a murmur present on the initial cardiac exam. *Staphylococcus epidermidis* and gram-negative rods can be the cause of prosthetic valve endocarditis. *Streptococcus viridans* is the most common organism in left-sided endocarditis in patients with congenital valvular disease or mitral valve prolapse.

176. **The correct answer is E.** Silver nitrate is ineffective on actively bleeding sources, and bilateral use of cautery is contraindicated due to the risk of septal necrosis from interruption of the vascular supply to the nasal septum. Bilateral nasal packing is usually required to provide adequate compression. Packing should be coated with antibiotic ointment and left in place for 48 to 72 hours to allow adequate time for healing. Anterior and posterior packing requires use of prophylactic oral antibiotics. All patients should be monitored closely when posterior packs are placed, because the nasopulmonary reflex produces a drop in the PaO₂ and an elevation in the PCO₂. Admission should be strongly considered.

177. **The correct answer is D.** High-flow oxygenation increases the rate of pneumothorax resorption. Repeat imaging in 4 hours to assess for expansion or decline in pneumothorax assists with further disposition decisions. Patients who become symptomatic require further care and monitoring.
178. The correct answer is A. This patient is alert and could benefit greatly from NIPPV, which would decrease his work of breathing, oxygen consumption, and CO₂ production while improving his oxygenation and ventilation. For patients who are not alert or who fail a trial of NIPPV and require intubation (answer E), it is important to titrate the ventilator to a near-physiologic pH, but not to decrease the paCO₂ to a “normal” level, as the baseline levels in COPD patients are often chronically high and are accompanied by appropriate chronic compensatory metabolic alkalosis. Overcorrection beyond baseline causes concomitant respiratory alkalosis, which can lead to fatal arrhythmias. This can also lead to prolonged ventilator weaning with the attendant risks of nosocomial infections, among other dangers. Intubation may ultimately be required, but the morbidity and mortality associated with ventilator dependence, particularly for COPD patients, are significant. All efforts to manage the patient without intubation should be undertaken in the meantime. Nebulized beta-agonists (answer D) and anticholinergics (answer B) may help with the portion of dyspnea that is caused by bronchospasm and bronchorrhea, as these may be present to some degree in many patients. It is certainly prudent to consider a trial of these medications, and they can usually be run through the tubing for the NIPPV mask. However, COPD, by definition, is a disease characterized by irreversible obstructive lung disease, and it is unlikely that someone with such a severe exacerbation of COPD would be “turned around” with the addition of the anticholinergic medication. Steroids can also help decrease the overlying acute inflammation that accompanies exacerbations. Supplemental oxygen is important if the patient is significantly hypoxic, but antiplatelet agents (answer C) are not indicated here.

179. The correct answer is C. Health care workers are not at any increased risk for meningitis and do not require prophylaxis unless they have had direct mucosal contact with the patient's secretions, as might occur during mouth-to-mouth resuscitation, endotracheal intubation, or nasotracheal suctioning.

180. The correct answer is A. This is the typical presentation of sigmoid volvulus, which usually occurs in debilitated elderly patients with a history of chronic constipation. Cecal volvulus occurs in younger patients and presents more like a small-bowel obstruction with acute symptoms. Sigmoid volvulus can be decompressed via rectal tube or sigmoidoscope, but often recurs. Cecal volvulus must be treated operatively. Antibiotics should be reserved for cases of suspected infarction, perforation, or ischemia.

181. The correct answer is C. Re-implantation is most successful if accomplished within 20 minutes. Periodontal ligament cells die after 60 minutes. Placing the tooth in milk increases the viability of the tooth to more than 3 hours. Scrubbing of the tooth or placing the tooth in disinfectants is contraindicated due to the trauma and toxicity to the fragile periodontal ligament cells.

182. The correct answer is A. Therapy for congestive heart failure includes nitrates to reduce preload and diuretics to decrease intravascular volume if the patient is determined to have total body fluid overload. ACE inhibitors can reduce afterload. CPAP or BiPAP therapy should be strongly considered.

183. The correct answer is B. A chalazion, a nontender lump in the lid's midportion, results from chronic eyelid inflammation that occurs from blockage of the meibomian oil glands (located in the tarsal plate). In contrast, a sty or hordeolum is located along the lid margin and is caused by acute staphylococcal infection of an oil gland.

184. The correct answer is E. Classically, diaphragmatic injuries are associated with an effusion, blurred diaphragm, and herniated viscera or a nasogastric tube above the diaphragm. A pleural contusion would account only for the effusion. A liver laceration would not account for these findings.

185. The correct answer is C. The weight-based dose of epinephrine for cardiopulmonary arrest is 0.01 mg/kg (0.1 ml of 1:10,000 solution). High-dose epinephrine (0.1 mg/kg) has been de-emphasized and is no longer recommended in standard pediatric arrest algorithms.
186. The correct answer is D. Perianal abscesses can be incised and drained safely in the ED. The other mentioned abscesses should undergo operative therapy.

187. The correct answer is D. Reporting elder abuse is mandatory. While all types of mental and physical abuse can occur to the elderly, the most common type of elder abuse is neglect. Victims show signs of dehydration and malnutrition. They also tend to have decubitus ulcers, poor hygiene, and lab findings that indicate medication noncompliance. Family members perpetrate the majority of elder abuse; most victims are caucasian women.

188. The correct answer is D. Your buddy has symptoms typical of high-altitude pulmonary edema (HAPE), which is the most common fatal manifestation of high-altitude illness. Pneumonia can be indistinguishable from HAPE, so antibiotics could be considered. The primary treatment is to have the patient descend 2,000 to 3,000 feet or use of a hyperbaric (Gamow) bag.

189. The correct answer is A. This patient has recently been treated in the ED for an asthma exacerbation and presumably prescribed steroids. Since her blood pressure does not respond to fluids, she likely has adrenal failure induced by chronic steroid administration.

190. The correct answer is C. Although spinal shock, vascular injuries, and incomplete spinal lesions may alter or complicate a neurologic exam following spinal cord injury, it is very useful to document the initial level of intact neurologic function and follow this exam with serial exams to detect progression of involvement. For cervical injuries, here are the sensory/motor functional examinations:

- C4: sensation to the suprasternal notch/spontaneous breathing
- C5: sensation to the clavicle/shoulder shrugging
- C6: sensation to the thumb/elbow flexion
- C7: sensation to the index finger/elbow extension
- C8–T1: sensation to the small finger/finger flexion

191. The correct answer is B. This constellation of symptoms is typical of hypercalcemia. Initial management is intravenous hydration followed by furosemide diuresis. This is then followed by bisphosphonate administration to further normalize the calcium level. Treatment of the underlying malignancy is ultimately the best treatment option. Inorganic phosphate infusion, although effective, will lead to metastatic calcium deposition and death. The other options are used in the treatment of hyponatremia or hypernatremia.

192. The correct answer is C. The opioid toxidrome causes the triad of altered mental status, respiratory depression, and miosis.

193. The correct answer is D. Medications for erectile dysfunction (PDE4 inhibitors) have been associated with life-threatening hypotension when nitrates are taken within 24 hours after their use. Other contraindications to the administration of nitrates include bradycardia, tachycardia, and hypotension. Nitrates should be used with caution in the setting of a right ventricular infarction.
194. The correct answer is C. Rigidity of the chest wall related to osteoarthritis and osteoporosis makes chest wall injuries more common in the elderly, after even relatively minor trauma. Rib fractures are the most common chest injuries in these patients. Because elderly individuals have less pulmonary reserve, they are more prone to respiratory insufficiency and development of pneumonia and respiratory failure. The most common complications of chest trauma in the elderly are atelectasis, pneumonia, and acute respiratory distress syndrome (ARDS). Myocardial contusion and hemopneumothorax (answers A and B) would not present like this. Splenic laceration (answer C) would likely present with hypotension, and flail chest (answer E) would present with asynchronous chest wall movement.

195. The correct answer is A. While a medical history of renal colic coupled with hematuria and flank pain radiating to the groin may suggest renal colic, the first consideration should be the possibility of a rupturing aortic aneurysm. His history of hypertension suggests the possibility of peripheral vascular disease. The red and white blood cells in the urine may be secondary to ureteral irritation from an abdominal hematoma.

196. The correct answer is C. Remember the “Terrible T’s” of cyanotic congenital heart disease: total anomalous pulmonary venous return in addition to the four entities presented as answers A, B, D, and E. Patent ductus arteriosus in isolation does not cause cyanosis.

197. The correct answer is D. Given the mechanism of injury (blunt force to the epigastrium) and the CT findings, the pain in the epigastrium is quite likely associated with a pancreatic injury. Mesenteric ischemia and splenic sequestration are not likely given the patient's age and his lack of relevant medical history.

198. The correct answer is A. This presentation is consistent with a first simple febrile seizure. Aside from a glucose level, routine electrolytes are not needed, and fever evaluation should be based on standard evaluation of a febrile illness based on age and possible source. In a well appearing 9-month-old who has been acting and eating well and has not been on antibiotics, lumbar puncture and/or brain imaging is not needed.

199. The correct answer is A. Anal fissures are extremely common and cause painful rectal bleeding. Hard stools and constipation predispose individuals to their formation. Antibiotics are not indicated unless there are signs of infection or abscess formation. Most heal with conservative care and do not require surgery. Anal fissures that are not in the midline require workup for inflammatory bowel disease.

200. The correct answer is C. Based on the case vignette, the most likely cause of the syncopal event is aortic stenosis. The classic triad indicative of aortic stenosis is dyspnea, angina, and syncope. Symptoms often appear late in the disease course. Patients with aortic stenosis may have a narrowed pulse pressure. The cardiac exam may demonstrate a harsh systolic ejection murmur best heard in the second right intercostal space with radiation to the right carotid artery. The S2 is paradoxically split. Strenuous activity, nitrates, and diuretics should be avoided. Patients who are symptomatic should be referred for possible surgical repair or replacement.

201. The correct answer is E. Direct or online medical control is through direct communication with the medical command. Indirect medical control is prospective and retrospective and is accomplished with protocols or standing orders, training, and QA/QI.

202. The correct answer is D. This man is having a COPD exacerbation. Bronchodilators and steroids are the mainstays of treatment. Antibiotics may also be indicated. Answer E would be appropriate in suspected acute coronary syndrome. The other answers (A, B, C) would be appropriate in a CHF exacerbation, but this patient has no signs of volume overload or cardiomegaly. Had this patient been in more significant respiratory distress, NIPPV could have helped decrease the morbidity and mortality associated with his hospital stay if it helped him avoid intubation.
203. The correct answer is C. Circumferential pelvic compression is a standard component of the acute stabilization of patients with pelvic fractures. By decreasing the intra-pelvic volume, the potential blood loss is reduced. If the patient becomes unstable, an arteriogram may be indicated.

204. The correct answer is B. After 4 or 5 years of age, a macrolide such as azithromycin should be used for pneumonia to cover for *Mycoplasma pneumonia*. Under 4 years of age, mycoplasma is uncommon, and high-dose amoxicillin is the antibiotic of choice. Amoxicillin, cefuroxime, and trimethoprim-sulfamethoxazole do not cover *Mycoplasma pneumonia*. Doxycycline can cause staining of primary teeth and is generally not recommended for patients under 8 years of age.

205. The correct answer is D. This patient exhibits signs of perforation with rigidity, fever, and leukocytosis. Plain radiographs are less than 50% sensitive for lower GI tract perforation, so CT scanning should be performed if the index of suspicion is high. Rectal foreign bodies need to be removed. While most rectal foreign bodies are inserted for sexual gratification, they may be also used in cases of sexual assault, so the patient should be questioned about this. Opioid analgesia will likely be required for removal, as may procedural sedation or general anesthesia.

206. The correct answer is C. Patients with acute iritis, blepharitis, optic neuritis, and acute angle glaucoma typically do not get total pain relief with the simple administration of a topical anesthetic.

207. The correct answer is D. The large effusion provides a very good alternate diagnosis (large malignant effusion) for the cause of this man's dyspnea and hypoxia. A small infiltrate (answer C) does not rule out PE; in fact, in the absence of a good alternate diagnosis to explain the symptoms and signs, a subtle finding like this may cause a patient to move into the moderate- or high-risk category. Chest films in patients with PE are usually abnormal in some way, but often with nonspecific findings. The S1-Q3-T3 pattern (answer A) is quite insensitive for PE, and the Homan's sign (answer B) is not sufficiently sensitive or specific to be of use in diagnosing DVT. Decreased PaCO₂ (answer E) is consistent with tachypnea but does not differentiate its many potential causes.

208. The correct answer is E. This patient's presentation raises concern for a ruptured abdominal aortic aneurysm; you should immediately consult vascular surgery. The patient is too unstable to go for a CT scan. Plain abdominal radiographs are neither sensitive nor specific for the diagnosis. An elderly patient with a sudden onset of abdominal pain with radiation to the flank should be assumed to have a ruptured abdominal aortic aneurysm, not a kidney stone.

209. The correct answer is B. Treatment of hypoglycemia is with glucose, 0.25 to 1 gm/kg. Using a mid-level dose of 0.5 gm/kg equates to dextrose 10%, 4 ml/kg; dextrose 25%, 2 ml/kg; or dextrose 50%, 1 ml/kg. Dextrose 10% solution is used in neonates and small infants to avoid vein damage and risk of intracranial hemorrhage. Dextrose 25% is used in older infants and children, while dextrose 50% is used in adolescents and adults.

210. The correct answer is D. This patient has acute otitis media. Observation with or without a wait-and-see prescription is an option only for patients under 2 years of age if the symptoms are not severe (temperature <39.0°C and/or mild otalgia) and follow-up is ensured. Otherwise, treatment is with high-dose amoxicillin because of the high rate of penicillin-resistant *Streptococcus pneumoniae*. Amoxicillin/clavulanate is usually reserved for patients in whom amoxicillin treatment has failed or who have recurrent infections.

211. The correct answer is D. This patient is hypotensive with a positive FAST exam. CT imaging, angiography (given the negative radiograph), and DPL are not indicated, as he remains unstable despite intravenous fluids. He requires surgical intervention immediately.
212. The correct answer is C. Synovial fluid from a patient with rheumatoid arthritis classically has an elevated WBC count (8,000–50,000/ml) and a low glucose level. The other possible choices are not associated with the same synovial fluid findings. Patients with septic arthritis have positive Gram stain and a WBC count greater than 50,000/ml. Patients with osteoarthritis and viral arthritis should not have a low glucose level.

213. The correct answer is D. Foreign bodies in children usually lodge at the cricopharyngeal muscle, whereas, in adults, the lower esophageal sphincter is more likely. Coins in the esophagus are seen “en face” on frontal radiographs. Button batteries in the esophagus need to be removed emergently but can be watched if they have passed into the stomach. Endoscopy is the best method for removal in general, especially if the object is sharp or elongated. Glucagon is of utility only if the object is at the lower esophageal sphincter.

214. The correct answer is A. This patient has Mackler’s triad of chest pain, vomiting, and subcutaneous emphysema, which is seen in esophageal rupture. The most common cause by far is iatrogenic (for instance, after dilation of esophageal strictures). Esophageal rupture may also be the result of Boerhaave’s syndrome after retching, increased intrathoracic pressure from weightlifting or labor, or trauma.

215. The correct answer is D. This patient has pyuria and presumed acute pyelonephritis and thus should be treated with antibiotics, pending culture results. In neonates and infants with pyelonephritis, leukocyte esterase or even pyuria may be falsely negative due to their limited leukocytic response. Urine cultures are mandatory in suspected cases of urinary tract infection in this age group regardless of urinalysis results. In patients older than 3 months of age who appear well without co-morbidities and with good follow-up, outpatient management with oral antibiotics after IV or intramuscular antibiotic administration is recommended. Cerebrospinal fluid studies are generally not needed in non-toxic infants older than 3 months.

216. The correct answer is C. Noncardiogenic pulmonary edema (NCPE) is characterized by a PaO₂:FiO₂ ratio (P:F) <200 and classically appears on a chest film as bilateral fluffy infiltrates without cardiomegaly. NCPE can be precipitated by opiates and a variety of other insults, including trauma, high-altitude pulmonary edema (HAPE), aspiration/near-drowning, inhaled toxins, smoke, systemic inflammatory response syndrome (SIRS), sepsis, and TCA or salicylate overdose. Optimal treatment includes tidal volumes of 5 to 7 mL/kg, peak pressures <35 mm Hg, and adequate PEEP as part of the strategy for ventilator management. Steroids (answers A and B) may be harmful. Aspiration is part of the differential in those at risk (who should then receive supportive treatment), but this acute presentation is not consistent with aspiration pneumonia, so antibiotics (answers B and E) are not indicated. Answer D (and antibiotics) might be indicated if this patient with a known history of IV drug abuse had presented with a history, signs, or symptoms consistent with septic emboli from tricuspid valve endocarditis.

217. The correct answer is D. A thrombolytic agent is indicated for a patient who is hemodynamically unstable from pulmonary embolism. Anticoagulation with heparin is part of the therapy for pulmonary embolism but is not the single best answer in the setting of hemodynamic instability. Pericardiocentesis would be performed for cardiac tamponade and a needle thoracostomy for a tension pneumothorax.

218. The correct answer is D. The most common organisms causing otitis externa are *P. aeruginosa* and *S. aureus*.

219. The correct answer is C. Glucagon is helpful in approximately 50% of meat impactions at the lower esophageal sphincter. Nitroglycerin and nifedipine are limited by their side effects and are less efficacious than glucagon. Benzodiazepines are ineffective. Meat tenderizer (papain) has fallen out of favor after it was shown that it increased the risk of esophageal necrosis and subsequent perforation. Endoscopy is often needed in refractory cases. All patients with meat impactions require follow-up to assess for underlying esophageal pathology.
220. The correct answer is A. Cardioversion starts with 0.5 to 1 joule/kg, and SVT rates are usually greater than 240 beats/min.

221. The correct answer is E. Individuals at high risk for aspiration pneumonia are alcoholics, diabetics, those with bad periodontal disease, and people with dysphagia. Anaerobic and Gram-negative agents should be covered with antibiotic selection when aspiration pneumonia is suspected. Patient A has no dysphagia and does not live in a nursing home. Patients B and D have comorbid pulmonary disease but have not been hospitalized recently. Patient C is at higher risk than healthy people for community-acquired pneumonia (CAP), but his CD4 count is well above 200, so *Pneumocystis carinii* pneumonia (PCP) would not normally be considered.

222. The correct answer is B. The patient presents with classic symptoms of aortic dissection. Aortic dissection is commonly missed because it often presents atypically. Eighty percent of patients with aortic dissection have a chest radiograph showing wide mediastinum or abnormal aortic contour. However, these findings can be missed if concern for aortic dissection is not relayed to the radiologist. In addition, given that 10% to 15% of patients with aortic dissection have a normal chest radiograph, the patient with a history that causes concern should receive further evaluation with CT angiography or echocardiography. Nonspecific atelectasis, Hampton's hump, and an elevated hemidiaphragm are sometimes seen in patients with pulmonary embolism.

223. The correct answer is A. Cocaine toxicity causes a sympathomimetic toxidrome, including hypertension, tachycardia, diaphoresis, and mydriatic pupils.

224. The correct answer is C. Pityriasis rosea is a mild inflammatory reaction that typically affects patients between 10 and 35 years of age. A typical description notes a "herald patch" followed a week later with a diffuse, symmetric rash in a "Christmas tree" pattern. Testing for syphilis is wise. Management is symptomatic, with resolution expected in 3 to 8 weeks. Recent articles indicate that oral erythromycin may shorten the course. Fluorinated corticosteroids are not recommended on the face.

225. The correct answer is B. This patient has had a seizure secondary to severe hyponatremia, as evidenced by the excessive water intake. Osmotic demyelination syndrome, of which central pontine myelinolysis is a variant, is the most dreaded complication of treating this disorder. It can be prevented by not raising the serum sodium concentration by more than 0.5 mEq/hr (or 12 mEq/24 hr). Hypertonic saline is the treatment of choice for hyponatremic seizures.
CHAPTER 22

Visual Images

Images Editor: Andreas Alfer, BSN, RN

The images contained in this chapter are representative of the types of images encountered on the American Board of Emergency Medicine Qualifying Examination (commonly referred to as the “written board examination”) and In-Training Examinations (commonly referred to as “inservice examinations”). They are referenced in the appropriate portions of Chapter 21 (Practice Test) and all other individual chapters. We thank all of the contributors to this chapter (listed below each image) for allowing us to utilize these images.

Image 1

Elizabeth Gray, MD
Image 68 Mark Silverberg, MD FACEP MMB
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This book is intended for
- residents taking the annual in-service examination
- graduating residents preparing for the qualifying exam, formerly the “written boards”
- physicians undergoing recertification
- medical students planning to match in emergency medicine

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