JUST THE FACTS IN EMERGENCY MEDICINE

David M. Cline
O. John Ma
Judith E. Tintinalli
Gabor D. Kelen
J. Stephan Stapczynski

- Essential information from Tintinalli's Study Guide
- Outline format, quick-reference tables, and figures
- Perfect for exam prep or a quick review
- Includes test-taking tips and strategies

American College of Emergency Physicians®

Just the Facts in
EMERGENCY MEDICINE

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PREFACE

In a crunch, when interviewing an eyewitness, Dragnet's Sgt. Joe Friday would implore, ''Just the facts, ma'am, just the facts.'' Our textbook, *Just the Facts in Emergency Medicine,* aims to provide just that for emergency physicians who are studying for either the written board (re)certification examination in emergency medicine or the in-training written examination.

This book has evolved from Judith Tintinalli's *Emergency Medicine: A Comprehensive Study Guide,* fifth edition, which has long been considered as a premier source for board certification preparation. Dr. Tintinalli's first edition of the *Study Guide,* published in 1978, was designed to cover the core content of emergency medicine for physicians preparing for the written board examination. Since then, along with the explosive growth in the field of emergency medicine, the *Study Guide* has been expanded to the point where it may be too voluminous to serve as a rapid review source. The other book that has evolved from the *Study Guide,* the *Companion Handbook,* was designed as a streamlined pocket reference guide for the practicing clinician and contains only the essential information that is pertinent to the clinical care of the patient in the emergency department.

Each chapter in *Just the Facts in Emergency Medicine* emphasizes the key points in the Epidemiology, Pathophysiology, Clinical Features, Diagnosis and Differential, and Emergency Department Care and Disposition of the disease entity. The bulleted outline for each factual item is designed to enhance its use as a rapid study aid.

We would like to express our deep appreciation to the *Just the Facts in Emergency Medicine* chapter authors for their commitment and hard work in helping to produce this textbook. We also are indebted to numerous individuals who assisted us with this project, in particular, we would like to thank Andrea Seils, Lester A. Sheinis, and Richard C. Ruzycka at McGraw-Hill. Finally, without the love and encouragement of our families, this book would not have been possible. DMC thanks his wife, Lisa, and his secretary, Nell; and OJM thanks Natasha, Gabrielle, Sabrina, Julius, Rebekah, and Elise.

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Just the Facts in
EMERGENCY MEDICINE

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Section 1
TEST PREPARATION AND PLANNING

1 FACTS ABOUT EMERGENCY MEDICINE BOARD EXAMS

David M. Cline

- The American Board of Emergency Medicine (ABEM) administers three written exams each year: the Certification Exam, the Recertification Exam, and the In-Training Exam. For the most up-to-date information concerning these exams, review the ABEM web site: *www.abem.org.*
- The American Board of Osteopathic Emergency Medicine (ABOEM) administers one certification examination per year.

ABEM WRITTEN CERTIFICATION EXAM

- The Certification exam is given each year in early November at several locations throughout the country; check for test site information at *www.abem.org.* This exam is usually given the day after the Recertification exam.
- The test consists of approximately 335 questions and lasts a total of 6 h and 15 min (1.1 min per question). There is a 60-min break for lunch.
- Of the test questions, 15 percent include a pictorial stimulus, generally during the first portion of the exam.
- The pass/fail criterion is 75 percent correct of those test items, which are included in the examination for the purpose of scoring.
- Typically, only two-thirds of the test is scored, with one-third of the test questions representing new trial content. These investigational questions are compared with standardized questions for reliability and may be included as scored items the following exam cycle. Typically, a question requires 2 years from the time of creation to use as a scored item.
- The pass rate for the Certification exam during the 1998 exam cycle was 91 percent for first-time takers with emergency medicine residency training and 73 percent for all others.
- Subject matter of the exam is based on the Emergency Medicine Core Content.[1]
- A percentage breakdown of the exam content compared to the chapters of this book is listed in Table 1-1. Although many of the questions are different, the content percentages are the same for all three ABEM written exams. *Just the Facts in Emergency Medicine* includes several chapters that include multiple topics, therefore our chapters do not precisely correlate to the exam question content areas.
- Compared to the Recertification exam, the Certification exam has more pathophysiology-based questions. Roughly 60 percent of the questions are management based, many of which require a diagnosis be made from the clinical description. There are 20 percent that are diagnosis based, and 10 percent are pathophysiology based. The remaining 10 percent of questions relate to administrative, emergency medical service (EMS), disaster medicine, and miscellaneous issues.
- Certification expires every 10 years.

ABEM WRITTEN RECERTIFICATION EXAM

- The Recertification exam is given each year in early November at several locations throughout the country, check for test site information at

TABLE 1-1 Percentage Distribution of Test Items by Core Content Category Compared to Chapter Listing of *Just the Facts in Emergency Medicine*

CONTENT AREA	WRITTEN EXAM PERCENTAGE DISTRIBUTION (%)	NUMBER OF CHAPTERS	*JUST THE FACTS IN EMERGENCY MEDICINE* CHAPTERS REPRESENTED
Abdominal and gastrointestinal disorders	7	15 (7.9%)	38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52
Cardiovascular disorders	11	15 (7.9%)	4, 5, 9, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 69, 81
Cutaneous disorders	1	2 (1.0%)	151, 152
Endocrine, metabolic, and nutritional disorders	6	8 (4.2%)	6, 78, 79, 82, 127, 128, 129, 130
Environmental disorders	2	11 (5.8%)	116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126
Head, ear, eye, nose, throat disorders	8	8 (4.2%)	16, 70, 83, 138, 147, 148, 149, 150
Hematologic disorders	2	7 (3.7%)	131, 132, 133, 134, 135, 136, 137
Immune system disorders	1	3 (1.6%)	11, 90, 97
Systemic infectious disorders	3	8 (4.2%)	8, 89, 91, 92, 93, 94, 95, 96
Musculoskeletal disorders (nontraumatic)	3	6 (3.2%)	175, 176, 177, 178, 179, 180
Nervous system disorders	5	9 (4.7%)	10, 139, 140, 141, 142, 143, 144, 145, 146
Obstetrics and disorders of pregnancy	2	4 (2.1%)	60, 61, 62, 63
Pediatric disorders	8	14 (7.4%)	67, 68, 71, 72, 75, 76, 77, 78, 79, 80, 84, 85, 86, 87
Psychobehavioral disorders	3	3 (1.6%)	181, 182, 183
Renal disorders	2	4 (2.1%)	53, 54, 57, 58
Thoracic-respiratory disorders	7	9 (4.7%)	32, 33, 34, 35, 36, 37, 73, 74, 161
Toxicology and clinical pharmacology	4	18 (9.5%)	98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115
Traumatic disorders	11	22 (11.6%)	7, 153, 154, 155, 156, 157, 158, 159, 160, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 172, 173, 174
Urogenital/gynecologic disorders	3	6 (3.2%)	55, 56, 59, 64, 65, 66
Administrative aspects of emergency medicine	2	3 (1.6%)	184, 185, 189
Emergency medical services/disaster medicine	3	1 (0.6%)	188
Clinical pharmacology	2	2.5 (1.3%)	4,* 12, 13, plus various chapters
Procedures/skills	4	10 (5.2%)	3, 14, 15, 17, 18, 19, 20, 21, 186, 187, plus various chapters

* Chapter content divided between two board exam content areas.

www.abem.org. This exam is usually given the day before the Certification exam.

- The test consists of approximately 310 questions and lasts a total of 5 h and 45 min (1.1 min per question). There is a 60-min break for lunch.
- Of the test questions, 15 percent include a pictorial stimulus, generally during the first portion of the exam.
- The pass/fail criterion for the Recertification exam is 75 percent correct of those test items, which are included in the examination for the purpose of scoring.
- Typically, only two-thirds of the test is scored, with one-third of the test questions representing new trial content. These investigational questions are compared with standardized questions for reliability and may be included as scored items the following exam cycle.
- The pass rate for the Recertification exam during the 1998 exam cycle was 95 percent.
- Subject matter of the exam is based on the Emergency Medicine Core Content.[1]
- A percentage breakdown of the exam content compared to the chapters of this book is listed in

Table 1-1. Although many of the questions are different, the content percentages are the same for all three ABEM written exams. *Just the Facts in Emergency Medicine* includes several chapters that include multiple topics, therefore our chapters do not precisely correlate to the exam question content areas.
- Compared to the Certification exam, the Recertification exam is more clinically based and has less pathophysiology-based questions.
- Recertification must be accomplished every 10 years to maintain ABEM Board Certification.

ABEM IN-TRAINING EXAM

- The In-Training exam is given to all emergency medicine residents each year in late February.
- The test consists of approximately 225 questions and lasts 4 h and 15 min (1.1 min per question), given as a single session.
- There is no pass/fail criterion; rather, residents are compared to other residents across the country at their same level of training. Scores for individual training programs are compared with other training programs across the country, and this information is provided to residency program directors.
- Subject matter of the exam is based on the Emergency Medicine Core Content.[1]
- The target at which all questions are aimed is the expected knowledge base and experience of an emergency medicine third-year resident.
- A percentage breakdown of the exam content compared to the chapters of this book is listed in Table 1-1. Although many of the questions are different, the content percentages are the same for all three ABEM written exams. *Just the Facts in Emergency Medicine* includes several chapters that include multiple topics, therefore our chapters do not precisely correlate to the exam question content areas.

AOBEM WRITTEN CERTIFICATION EXAM

- The certification exam is given the first week of February each year.
- Like the ABEM exams, the subject matter of the exam comes from the Emergency Medicine Core Content.[1] However, AOBEM adds some test items drawn from osteopathic principles and practice.
- The percentage breakdown of the exam may be different than that listed in Table 1-1.
- AOBEM, like ABEM, uses a preset passing score, but it is not currently published. Also, each exam contains nonscored test items that are in the process of evaluation and standardization.

REFERENCES

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2 TEST-TAKING TECHNIQUES

David M. Cline

- Excellent test performance requires both well-planned study methods and carefully applied test-taking skills.

STUDY TECHNIQUES

- Begin by setting a schedule to accomplish your study goals and objectives in the time remaining prior to the test. Allow time for reading this book, using a question-and-answer book to uncover any gaps in your knowledge base and your final review. Your schedule should be written and checked often to document your progress.
- Find a place to study that facilitates concentration, not distraction. Hettich found that a single place of study improved test performance.[1]
- Begin reading each chapter by glancing over the topic headings to get an overview of the material. Formulate questions in your mind such as:
  1. What etiologic information will help me to identify the disease?
  2. What pathophysiologic concepts will help to treat the disease?
  3. What clinical features will help me to identify the disease?

4. What criteria make the diagnosis of the disease?
5. What are the recommended treatments for the disease?

- Reading should be an active experience. Don't turn the exercise into a coloring contest with your highlighter. Write in the margins, circle, underline, and identify key points.
- Review your notes and key points at the end. If you find the material confusing or your understanding incomplete, you will need to go to other sources for additional information, such as the parent textbook for this review book: *Emergency Medicine: A Comprehensive Study Guide,* 5th ed.
- Last-minute cramming is an inefficient study method, taxes your energy level, and creates anxiety.[2]

PREPARATION IMMEDIATELY BEFORE THE TEST

- Get plenty of sleep the night before the test.
- Arrive at the test site well in advance of the start time to make sure you know where the exam room is located and become familiar with the surroundings.
- Check the temperature of the exam room so that you can anticipate proper attire. Dress comfortably.
- Schedule enough time to wake up, dress, and eat an unhurried breakfast.
- Eat an adequate but not heavy breakfast. Do the same for the lunch break.
- Bring a photo ID to identify yourself.
- Pencils are provided. No food (including candy) is allowed at the exam tables. Water is available in the room. If current policy holds, diplomates taking the recertification exam may find a snack table and coffee at the back of the room. However, you must consume these nourishments at the back tables and may not bring them with you to the table where you test.

TAKING THE TEST

- Listen carefully to the instructions given and read completely any written instructions.
- You have 1.1 min per question on the test. Make sure that at any given point you are keeping to schedule. For example, at the 1-h mark, you should have answered approximately 60 questions. However, the pictorial stimulus portion of the test is usually first, and these questions take more time than the remaining questions for most test takers.
- There is no penalty for guessing on this multiple-choice exam.
- Fill in the answer sheet as you go. Many authors recommend skipping the hard questions and returning to them at the end. This practice may leave you without time to revisit the unanswered questions. Skipping items also increases the chances that you will key the answer sheet incorrectly. Study proctors will not allow you extra time to correct or fill in your answer sheet.
- Carefully read the question stem and anticipate the answer before you read the options listed. If you see the choice you anticipate, that answer is most likely correct.
- Read all the answers to check for a more complete or better answer than the one you anticipate.
- Don't use excessive time on a single question that puzzles you. Simply make your best guess and move on. Make a note in the test booklet margin and return to the question at the end for further consideration.
- Remember that approximately one-third of the test is not scored (see Chap. 1). If you don't know the answer or find the question confusing, it may be a trial question. Don't loose your confidence or your momentum.
- Learn to identify the incorrect options quickly so that, if you are forced to guess, you have a better chance of being correct.
- On items that have "all of the above" as an option, if you are certain that two other answers are correct, you should choose "all of the above."
- Options that include broad generalizations are more likely to be incorrect.
- There is no evidence to support the idea that option "C" is more likely to be correct than others on ABEM exams.
- Use every minute of the test time. If you have time left over, review first the questions you have identified as difficult and then use the remaining time to reread the questions, looking for any misinterpretations that may have occurred the first time through.
- Contrary to popular opinion, your "first guess" is not more likely to be correct than a carefully considered reevaluation of the answer.[3] If, during the review process, you find a better answer to a question stem, do not hesitate to change your choice. You have a 57.8 percent chance of changing a wrong answer to a correct one, a 22.2 percent chance of changing a wrong answer to another

wrong answer, and only a 20.2 percent chance of changing a correct answer to an incorrect one.[3]

- Do not spend your lunch break discussing specific test questions with colleagues. This practice could disqualify you from the test, and it creates more anxiety, further limiting your performance in the afternoon. Remember, only two-thirds of the test is scored.
- Relax. The odds are in your favor. And now that you own this book, you have a concise means to review the practice of emergency medicine.

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Section 2

RESUSCITATIVE PROBLEMS AND TECHNIQUES

3 ADVANCED AIRWAY SUPPORT

Robert J. Vissers

INITIAL APPROACH

- Airway management takes priority over all other aspects of resuscitation.
- There are four main indications for invasive airway management: airway protection, ventilation, oxygenation, and facilitation of therapy.

PATHOPHYSIOLOGY

- The upper anatomic airway includes the oral and nasal cavities down to the larynx. The lower airway includes the trachea, bronchi, and lungs.
- Potentially difficult intubations can be predicted by the following:
  a. External features suggestive of difficulty, such as a beard, obesity, a short neck, a receding chin, and tracheostomy scars.
  b. Inability to open the mouth three finger breadths or a thyromental distance less than three finger breadths.
  c. A relatively large tongue for the oral cavity as estimated by the inability to visualize more than the base of the uvula in a cooperative patient opening the mouth in a sniffing position.[1]
  d. Evidence of upper airway obstruction (see Table 3-1).
  e. Lack of neck mobility. This should be assessed only in patients without a potential C-spine injury.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- All patients who require airway management should be on a cardiac monitor, receive pulse oximetry with oxygen, and have intravenous (IV) access.
- The method of airway management is dependent on the patient, the indications, and the perceived airway difficulty. Options for airway management include bag-valve-mask, tracheal intubation, alternative noninvasive airways, and surgical airways.
- Definitive airway management, if indicated on initial assessment, should not be delayed until the results for arterial blood gases are received.

TRACHEAL INTUBATION

- Tracheal intubation is the most common technique for definitive airway management.
- It is associated with a high success rate and a low complication rate and ensures airway protection, patency, and facilitation of ventilation and oxygenation.[2]
- Orotracheal intubation is associated with a higher success rate and lower complication rate than compared with nasotracheal intubation.[2]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Orotracheal intubation using rapid-sequence intubation (RSI) techniques is the preferred method for tracheal intubation.[2,3]
- A laryngoscope using a number 3 or 4 Macintosh blade or a number 3 Miller (straight) blade is

TABLE 3-1 Clinical Manifestations Associated with Acute Airway Obstruction

ETIOLOGY	MANIFESTATION
Vascular	Hematoma External hemorrhage Hypotension Hemoptysis
Laryngotracheal	Stridor Subcutaneous air (massive) Hoarseness Dysphonia Hemoptysis
Pharyngeal and/or hypopharyngeal	Subcutaneous air Hematemesis Dysphagia Sucking wound

sufficient for most adults, depending on size and intubator preference.

- An endotracheal tube with an internal diameter of 7.5 to 8.0 mm and 8.0 to 8.5 mm is appropriate for most adult females and males, respectively.
- Endotracheal tubes with high-volume, low-pressure cuffs are preferred for the prevention of aspiration and to avoid ischemia of the tracheal mucosa.[4]
- The tube ideally is placed 2 cm above the carina. From the corner of the mouth, this is approximately 23 cm in men and 21 cm in women.[5]
- Patient positioning is critical to successful intubation. Flexion of the lower neck with extension at the atlanto-occipital joint aligns the oropharyngeolaryngeal axes, allowing better glottic visualization.
- RSI involves the combined administration of an induction agent and a neuromuscular blocking agent to facilitate tracheal intubation.[2] The following steps are taken:
  a. Preparation of the patient and equipment and assessment of airway difficulty.
  b. Preoxygenation with 100% oxygen.
  c. Administration of pretreatment agents to blunt adverse responses to RSI in selected patients. The four most commonly used agents are lidocaine, opioids, defasciculating agents, and atropine.
  d. Administration of an induction agent (see Table 3-2).[6]
  e. Administration of neuromuscular blockade. Succinylcholine is the most common agent used because of its rapid onset and short duration of action.[2] Some adverse effects are unique

TABLE 3-2 Sedative Induction Agents

AGENT	DOSE	ONSET	DURATION	BENEFITS	CAVEATS
Thiopental	3–5 mg/kg	30–40 s	10–30 min	↓ ICP	↓ BP Laryngospasm
Methohexital	1 mg/kg	<1 min	5–7 min	↓ ICP Short duration	↓ BP Seizures Laryngospasm
Midazolam	0.1 mg/kg	1–2 min	20–30 min	Reversible Amnesic Anticonvulsant	Apnea No analgesia Highly variable dose
Ketamine	1–2 mg/kg	1 min	5 min	Bronchodilator "Dissociative" amnesia	↑ Secretions ↑ ICP Emergence phenomenon
Etomidate	0.3 mg/kg	<1 min	10–20 min	↓ ICP ↓ IOP Rare ↓ BP	Myoclonic excitation Vomiting No analgesia
Propofol	0.5–1.5 mg/kg	20–40 s	8–15 min	Antiemetic Anticonvulsant ↓ ICP	Apnea ↓ BP No analgesia
Haloperidol	5-mg aliquots	5–10 min	Variable	Rare ↓ BP	Titrate Dystonia
Droperidol	2.5-mg aliquots	5–10 min	Variable	Rare ↓ BP Antiemetic	Titrate Dystonia ↓ BP
Fentanyl	3–8 μg/kg	1–2 min	30–40 min	Reversible analgesia	Highly variable dose ICP—variable effects Chest wall rigidity

ABBREVIATIONS: BP = blood pressure; ICP = intracranial pressure; IOP = intraocular pressure.

TABLE 3-3 Succinylcholine

AGENT	ONSET	DURATION	BENEFITS
1.0–1.5 mg/kg	30–60 s	3–8 min	Rapid onset Short duration

COMPLICATIONS	
Bradyarrhythmias	Masseter spasm
Increased intragastric, intraocular, and intracranial pressure	Malignant hyperthermia
Hyperkalemia	Prolonged apnea with pseudocholinesterase deficiency
Fasciculation-induced musculoskeletal trauma	Histamine release
	Cardiac arrest

to depolarizing agents (see Table 3-3).[7] Nondepolarizing agents can be used, but all have a much longer duration of action and a generally slower onset (see Table 3-4).

f. Protection from passive reflux with cricoid pressure (Sellick's maneuver).
g. Insertion of the endotracheal tube.
h. Confirmation of tube placement.

- Tracheal placement of the tube must be confirmed by clinical measures: visualization of the tube passing through the cords, tube condensation, chest and epigastric auscultation, and chest wall expansion.
- Clinical confirmation can be falsely positive and must be supplemented with either end-tidal CO_2 detectors or esophageal detection devices.[8,9]
- Several methods are available to assist with difficult orotracheal intubation: digital intubation, a semirigid stylet (gum-elastic bougie), transillumination with a lighted stylet, fiberoptic-assisted intubation, and retrograde tracheal intubation.[10,11]
- A failed airway is defined as three consecutive unsuccessful attempts at intubation attempted by the most experienced operator.

NASOTRACHEAL INTUBATION

- Nasotracheal intubation may be indicated when laryngoscopy is predicted to be difficult or neuromuscular blockade is contraindicated.
- The nares should be sprayed with a topical vasoconstrictor and anesthetic.
- Tube size is generally 1.0 mm smaller than that used for an oral intubation.
- The tube is inserted in a spontaneously breathing patient ideally upon the initiation of inspiration.
- The optimal depth placement of a nasotracheal tube, measured at the nares, is 28 cm in men and 26 cm in women.[5]
- Nasotracheal intubation is associated with a lower success rate and a higher complication rate than is RSI-assisted orotracheal intubation.[2]

ALTERNATIVE NONINVASIVE AIRWAY TECHNIQUES

- The primary alternative to tracheal intubation is bag-valve-mask (BVM) ventilation.
- BVM provides ventilation and oxygenation but not airway protection from aspiration.
- The incidence of ''can't intubate, can't ventilate'' is estimated to be 1 : 1000 to 1 : 10,000 patients.
- Several airway rescue devices are available as alternatives to tracheal intubation.

TABLE 3-4 Nondepolarizing Neuromuscular Relaxants

AGENT	ADULT INTUBATING DOSE IV	ONSET	DURATION	COMPLICATIONS
Vecuronium (intermediate/long)	0.8–0.15 mg/kg 0.15–0.28 mg/kg (high-dose protocol)	2–4 min	25–40 min 60–120 min	Prolonged recovery time in obese or elderly or if there is hepatorenal dysfunction
Pancuronium (long)	0.1–0.15 mg/kg	3–5 min	80–100 min	Vagolytic tachyarrhythmias Prolonged recovery in elderly or if there is hepatorenal dysfunction
Doxacurium (long)	0.05–0.08 mg/kg	3–5 min	80–100 min	Prolonged block
Atracurium (intermediate)	0.4–0.6 mg/kg	2–3 min	25–45 min	Histamine release Hypotension Bronchospasm
Cisatracurium (intermediate)	0.15–0.20 mg/kg	2–3 min	50–60 min	Cardiovascular
Rocuronium (intermediate)	0.6–1.0 mg/kg	1–3 min	30–45 min	Tachycardia
Mivacurium (short)	0.15–0.20 mg/kg	2–3 min	10–20 min	Histamine release

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- BVM is most effective using the two-person technique, positioning similar to that for intubation, and with nasal or oral airways in place.[12]
- Esophageal airways are devices used primarily in the prehospital setting when orotracheal intubation is not an option. The devices are inserted blindly in apneic unconscious patients.[13]
- Types of esophageal airways include the esophageal obturator airway, the pharyngotracheal lumen airway, the esophageal tracheal combitube, and the tracheoesophageal airway.
- A laryngeal mask airway (LMA) can be placed blindly without manipulation of the patient's head.[14] An LMA does not protect against aspiration and should be considered a temporizing device in the emergency setting.

SURGICAL AIRWAY TECHNIQUES

- The most common indication for a surgical airway is failure to intubate and ventilate. This may be secondary to acute airway obstruction (see Table 3-1) or, rarely, a failed intubation in a paralyzed patient.
- The incidence has been reported to be as high as 2 percent; however, recent studies suggest a rate less than 1 percent.[2,15]
- Most emergency surgical airway techniques access the airway through the cricothyroid membrane in the midline between the cricoid cartilage and the thyroid cartilage, approximately one-third the distance from the manubrium to the mentum.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- An emergency cricothyrotomy requires a scalpel, a tracheal hook, and a dilator.
- Cricothyrotomy should be considered a blind technique.
- A #4 Shiley tracheal tube is an adequate size for the majority of adults.
- Complications include bleeding, creation of a false passage outside the trachea, injury to structures of the neck, and pneumothorax. Delayed voice changes and stenosis may occur.[15]
- Cricothyrotomy is contraindicated in patients younger than 12 years of age because of the small size of the membrane, and needle cricothyrotomy should be used in these patients.
- Needle cricothyrotomy utilizes a large-gauge needle to access the cricothyroid membrane. Oxygenation can be performed with a BVM or preferably with jet ventilation.
- Jet ventilation should be set at 50 psi for adults and 25 psi for children. Four seconds of expiration is allowed for each second of insufflation.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 14, ''Noninvasive Airway Management,'' by A. Michael Roman; Chap. 15, ''Tracheal Intubation and Mechanical Ventilation,'' by Daniel F. Danzl; and Chap. 16, ''Surgical Airway Management,'' by David R. Gens.

4 DYSRHYTHMIA MANAGEMENT AND CARDIOVASCULAR PHARMACOLOGY

David M. Cline

THE NORMAL CARDIAC CONDUCTING SYSTEM

- The heart consists of three types of specialized tissue: (a) pacemaker cells that undergo spontaneous depolarization and can initiate an electric impulse; (b) cells that conduct electrical waves more rapidly than other cardiac cells, causing a very rapid propagation of the electric impulse throughout the heart, and (c) contractile cells, which contract when electrically depolarized.
- The sinus node is the dominant cardiac pacemaker; blood supply is from the right coronary artery (in about 55 percent of individuals) or from the left circumflex artery (in the other 45 percent). The normal rate is 60 to 100 beats per minute.
- Normally, electric impulses from the atria can reach the ventricles only by passing through the atrioventricular (AV) node and infranodal conducting system.
- The AV node receives its blood supply from the right coronary artery in 90 percent of individuals and, in the other 10 percent, as it comes off the left circumflex artery. This accounts for the common occurrence of AV conduction disturbances with acute inferior myocardial infarctions.
- The AV node has two important electrophysiologic characteristics: it slows conduction velocity and has a long refractory period that allows time for atrial contraction to give an extra 10 percent ventricular filling. This ''atrial kick'' is most important for patients with ventricular failure. Electric impulses leave the inferior pole of the AV node through the bundle of His which consists of the rapidly conducting Purkinje cells. The bundle of His divides into the right and left bundle branches.

THE NORMAL ELECTROCARDIOGRAM

- In Fig. 4-1, depolarization starts on the left side of the ventricular septum and initially proceeds to the right; this is recorded as a small negative deflection in the recording electrode.
- Subsequent depolarization involves the free walls of both ventricles, and since the left side has a much larger mass, the net sum of electrical activity is directed toward the recording electrode and a tall, positive deflection is recorded.
- The P-QRS-T complex of the normal (electrocardiogram) ECG represents electrical activity over one cardiac cycle (Fig. 4-2).

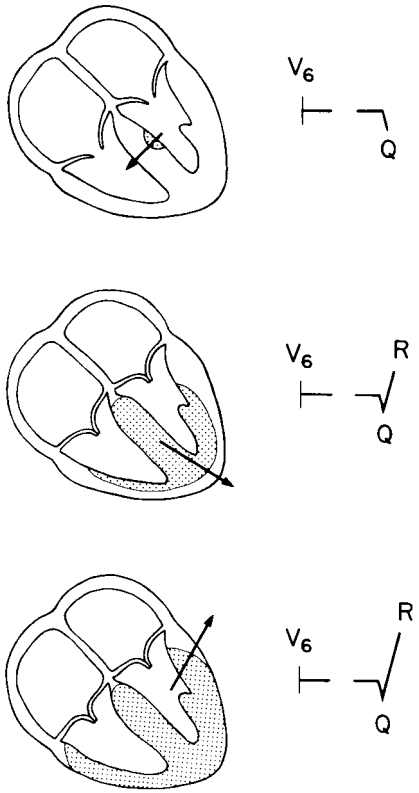

FIG. 4.1 Ventricular depolarization recorded in lead V_6.

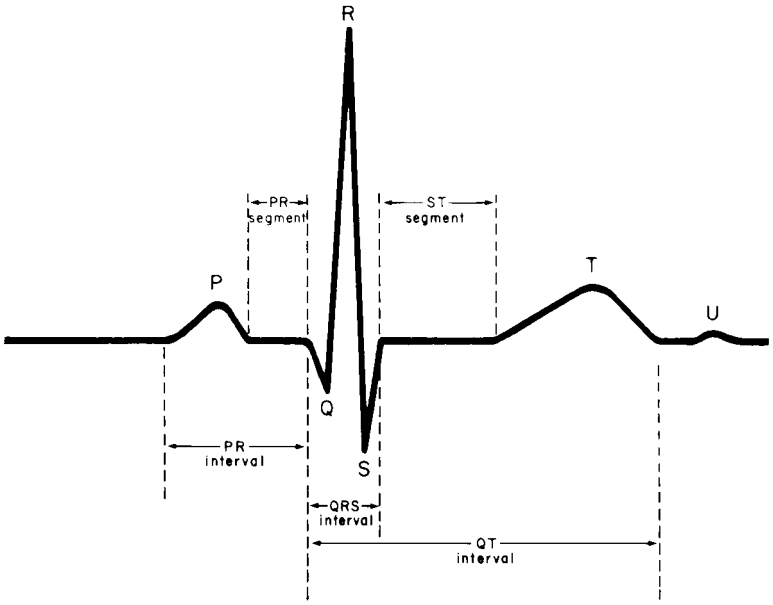

FIG. 4.2 Normal P-QRS-T ECG pattern.

- The P wave is caused by atrial depolarization. The QRS complex usually obscures atrial repolarization. The normal P-wave duration is less than 0.10 s (2.5 mm), and normal amplitude is less than 0.3 mV (3 mm).
- The PR interval is the time between the onset of depolarization in the atria and the onset of depolarization in the ventricles. It is commonly used as an estimation of AV nodal conduction time because the AV node is the most likely site for delay in conduction. For adults in sinus rhythm, the PR interval is 0.12 to 0.20 s (3 to 5 mm) at 25 mm/s.
- The QRS complex indicates ventricular depolarization. Despite the large amount of myocardium that must be depolarized, the specialized conducting system makes this a rapid process and the normal QRS duration is 0.06 to 0.10 s (1.5 to 2.5 mm). Any delay in intraventricular conduction results in a wide QRS.
- Ectopic impulses that originate below the bundle of His or that arrive prior to repolarization of the bundle branches also result in a widened QRS because they do not use the Purkinje network.
- While small negative initial deflections (Q waves) are normal, large Q waves can be due to an electrically unexcitable area just under the recording electrode. An abnormal Q wave has a width of 0.04 s or greater and a height one-third that of the QRS complex.
- The ST segment represents the plateau phase of ventricular depolarization. While the ST segment is usualy isoelectric, a small deviation, less than 0.1 mV (1 mm), is not always pathologic.
- The T wave is caused by ventricular repolarization. Depolarization is a rapid, near-simultaneous release of stored energy (like the release of a compressed spring); repolarization is a slow, asynchronous event where the metabolic machinery of each individual cell restores the transmembrane potential. Therefore, the T-wave duration is much longer and the amplitude much lower than those of the QRS complex.
- The QT interval represents ventricular depolarization and repolarization. While QT duration is commonly between 0.33 and 0.42 s, it does vary inversely with heart rate. The corrected interval is obtained by dividing the measured QT interval (in seconds) by the square root of the R-R interval (in seconds). The normal corrected QT interval is less than 0.47 s.
- The U wave may be seen as a normal component of the surface ECG. The classic explanation is that the U wave represents the delayed repolarization of the Purkinje network.

CARDIAC DYSRHYTHMIAS

MECHANISMS OF TACHYDYSRHYTHMIAS

- There are three accepted mechanisms for dysrhythmias: (a) increased automaticity in a normal or ectopic site, (b) reentry in a normal or accessory

pathway, and (c) after depolarizations causing triggered rhythms.

- An ectopic focus is an area of the heart, away from the normal sinus node pacemaker, that acquires independent pacemaker activity and usurps the pacemaking role.
- These ectopic pacemakers can be the result of (a) enhanced automaticity of subsidiary pacemaker cells (i.e., in the AV node or infranodal conducting system) or (b) abnormal automaticity of myocardial cells, which seldom possess pacemaking activity (i.e., Purkinje cells). Dysrhythmias due to an ectopic focus usually have a gradual onset (''warm-up period''). The termination is also gradual, as opposed to the abrupt onset and termination seen with reentry or triggered mechanisms.
- Reentry requires a temporary or permanent unidirectional block in one limb of a circuit and slower-than-normal conduction around the entire circuit. These conditions are secondary to disease, drugs, accessory pathways, or when tissue is stimulated during the partial refractory period (before full repolarization), as with premature depolarizations.
- As indicated in Fig. 4-3, the inciting impulse traveling in the normal downward direction encounters the two limbs, finds limb *a* blocked, and travels down limb *b*. Upon reaching the bottom portion of the circuit where the two limbs rejoin, the impulse can then travel retrograde up limb *a* and reach the upper connection of the circuit. Normally, conduction is so rapid that the impulse would encounter limb *b* still refractory to stimulation, and no further propagation would occur. However, if conduction around the circuit were slow enough, limb *b* would be able to conduct the impulse again in the antegrade direction.
- Reentry can occur around anatomically defined circuits, resulting in a regular rapid rhythm such as paroxysmal supraventricular tachycardia. Conversely, reentry can also occur in a disorganized and chaotic fashion through a syncytium of myocardial tissue—as seen, for example, in atrial or ventricular fibrillation.
- Triggered dysrhythmias are due to the oscillations of the transmembrane potential during or after repolarization (afterpotentials). Under ideal conditions of rate, afterpotentials reach threshold and trigger a complete depolarization (afterdepolarization). Once triggered, this process may be self-sustaining.
- The urgency with which tachydysrhythmias require treatment is guided by two considerations: (a) evidence of hypoperfusion (shock, altered mental status, anginal chest pain, or pulmonary edema) and (b) the potential to degenerate into a more serious dysrhythmia or cardiac arrest.

MECHANISMS OF BRADYDYSRHYTHMIAS

- Bradydysrhythmias can be caused by two mechanisms: depression of sinus nodal activity or conduction system blocks. In both situations, subsidiary pacemakers take over and pace the heart; and provided the pacemaker is located above the bifurcation of the bundle of His, the rate is generally adequate to maintain cardiac output.
- The need for emergent treatment of bradycardias is guided by two considerations: (a) evidence of hypoperfusion and (b) the potential to degenerate into a more profound bradycardia or ventricular asystole. In general, emergent treatment is not required, unless (a) the heart rate is below 50 and there is clinical evidence of hypoperfusion or (b) the bradycardia is due to structural disease of the infranodal conducting system (either transient or

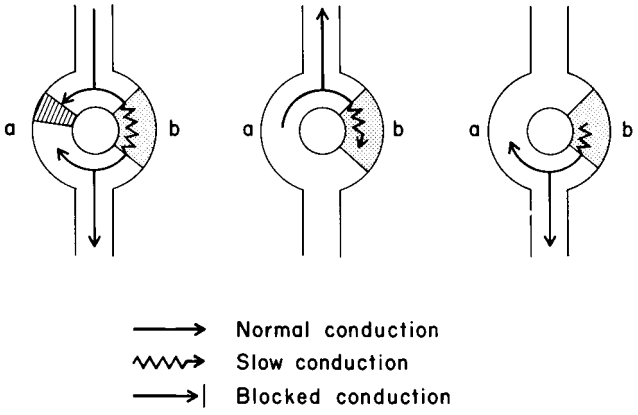

FIG. 4.3 Reentry circuit.

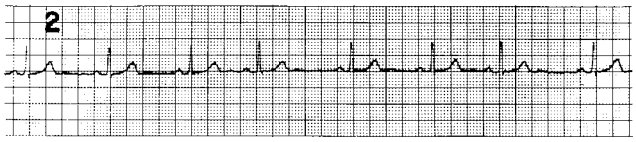

FIG. 4.4 Sinus dysrhythmia.

permanent) that has a risk of progressing to complete AV block.
- Three methods are currently available for emergent treatment of bradycardias: atropine, isoproterenol, and transcutaneous cardiac pacing.
- Internal pacing is the definitive treatment for progressive or persistent bradycardias. Emergent internal pacing is possible with the use of balloon-tipped flotation catheters, although, without fluoroscopic guidance, it is often technically difficult to achieve stable placement in a patient with low cardiac output.

SUPRAVENTRICULAR DYSRHYTHMIAS

SINUS DYSRHYTHMIA

Clinical Features
- Some variation in the sinus node discharge rate is common, but if the variation exceeds 0.12 s between the longest and shortest intervals, sinus dysrhythmia is present.
- The ECG characteristics of sinus dysrhythmia are (a) normal sinus P waves and PR intervals; (b) 1:1 AV conduction; and (c) variation of at least 0.12 s between the shortest and longest P-P interval (Fig. 4-4).
- Sinus dysrhythmias are primarily affected by respiration and are most commonly found in children and young adults, disappearing with advancing age.
- No treatment is required.

SINUS BRADYCARDIA

Clinical Features
- Sinus bradycardia occurs when the sinus node rate falls below 60.
- The ECG characteristics of sinus bradycardia are (a) normal sinus P waves and PR intervals, (b) 1:1 AV conduction, and (c) atrial rate below 60 (Fig. 4-5).

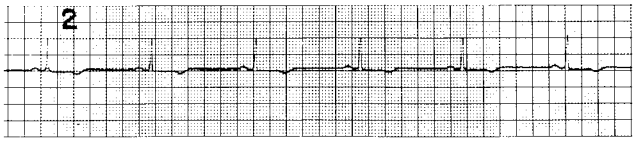

FIG. 4.5 Sinus bradycardia, rate 44.

- Sinus bradycardia represents a suppression of the sinus node discharge rate, usually in response to three categories of stimuli: (a) physiologic; (b) pharmacologic; and (c) pathologic (acute inferior myocardial infarction, increased intracranial pressure, carotid sinus hypersensitivity, hypothyroidism).

Emergency Department Care and Disposition
- Sinus bradycardia usually does not require specific treatment unless the heart rate is below 50 and there is evidence of hypoperfusion.
- Initial therapy should begin with atropine 0.5 to 1 mg IV and may be repeated up to 3 mg.
- External cardiac pacing can be used in the patient refractory to atropine.
- Epinephrine or dopamine drips may be used if external pacing is not available.
- Internal pacing is required in the patient with symptomatic recurrent or persistent sinus bradycardia.

SINUS TACHYCARDIA

Clinical Features
- The ECG characteristics of sinus tachycardia are (a) normal sinus P waves and PR intervals; (b) an atrial rate usually between 100 and 160; and (c) normally, 1:1 conduction between the atria and ventricles (although rapid rates can occur with AV blocks) (Fig. 4-6).
- Sinus tachycardia is in response to three categories of stimuli: (a) physiologic, (b) pharmacologic, or (c) pathologic (fever, hypoxia, anemia, hypovolemia, pulmonary embolism).
- In many of these conditions, the increased heart rate is an effort to increase cardiac output to match increased circulatory needs. The underlying condition should be diagnosed and treated.

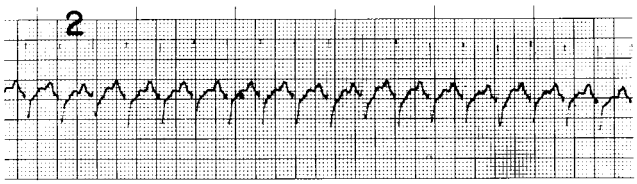

FIG. 4.6 Sinus tachycardia, rate 176.

PREMATURE ATRIAL CONTRACTIONS

Clinical Features

- The ECG characteristics of premature atrial contractions (PACs) are (a) ectopic P wave appears sooner (premature) than the next expected sinus beat; (b) the ectopic P wave has a different shape and direction; and (c) the ectopic P wave may or may not be conducted through the AV node (Fig. 4-7).
- Most PACs are conducted with typical QRS complexes, but some may be conducted aberrantly through the infranodal system. The sinus node is often depolarized and reset so that, while the interval following the PAC is often slightly longer than the previous cycle's length, the pause is less than fully compensatory.
- PACs are common in all ages and are often seen in the absence of heart disease.

Emergency Department Care and Disposition

- Any precipitating drugs (alcohol, tobacco, or coffee) or toxins should be discontinued.
- Underlying disorders should be treated (stress, fatigue).
- PACs that produce symptoms or initiate sustained tachycardias can be suppressed with various agents such as β-adrenergic antagonist, usually in consultation with a follow-up physician.

MULTIFOCAL ATRIAL TACHYCARDIA

Clinical Features

- Multifocal atrial tachycardia (MFAT) is caused by at least two different sites of atrial ectopy.
- The ECG characteristics of MFAT are (a) three or more differently shaped P waves; (b) varying PP, PR, and RR intervals; and (c) atrial rhythm usually between 100 and 180 (Fig. 4-8). MFAT can be confused with atrial flutter or fibrillation.
- MFAT is most often found in elderly patients with decompensated chronic lung disease, but it also may be found in patients with congestive heart failure or sepsis or may be caused by methylxanthine toxicity.

Emergency Department Care and Disposition

- Treatment is directed toward the underlying disorder.

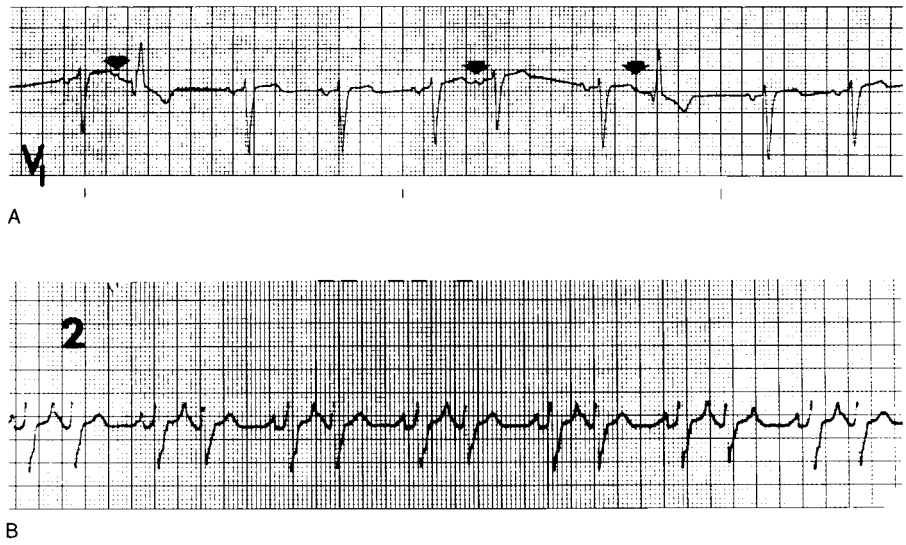

FIG. 4.7 Premature atrial contractions (PACs). Top: ectopic P′ waves (arrows). Bottom: atrial bigeminy.

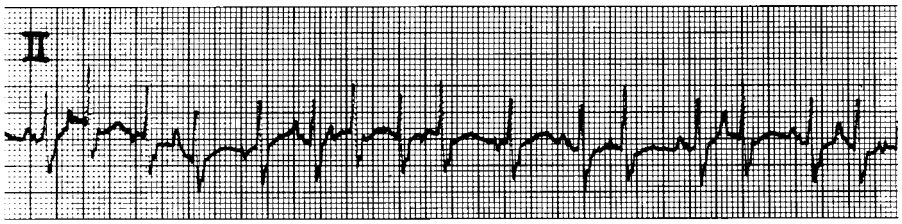

FIG. 4.8 Multifocal atrial tachycardia (MFAT).

- Specific antidysrhythmic treatment is uncommonly indicated.
- Magnesium sulfate 2 g IV over 60 s, followed by a constant infusion of 1 to 2 g/h, has been shown to decrease ectopy and convert MFAT to sinus rhythm in many patients.

ATRIAL FLUTTER

Clinical Features

- Atrial flutter is a rhythm that originates from a small area within the atria. The exact mechanism—whether reentry, automatic focus, or triggered dysrhythmia—is not known.
- ECG characteristics of atrial flutter are (a) regular atrial rate between 250 and 350 (most commonly 280 and 320); (b) sawtooth flutter waves directed superiorly and most visible in leads II, III, aV_F; and (c) AV block, usually 2:1, but occasionally greater or irregular (Fig. 4-9).
- Carotid sinus massage is a useful technique to slow the ventricular response, increase the AV block, and unmask flutter waves.
- Atrial flutter is most commonly seen in patients with ischemic heart disease or acute myocardial infarction. Less common causes include congestive cardiomyopathy, pulmonary embolus, myocarditis, blunt chest trauma, and, rarely, digoxin toxicity.
- Atrial flutter may be a transitional dysrhythmia between sinus rhythm and atrial fibrillation.
- Consider the need for anticoagulation prior to conversion to sinus rhythm.

Emergency Department Care

- Low-energy cardioversion (25 to 50 J) is very successful in converting more than 90 percent of cases of atrial flutter into sinus rhythm.
- If cardioversion is contraindicated, ventricular rate control can be achieved with diltiazem, 0.25 mg/kg IV over 2 min; may be repeated at 0.35 mg/kg.
- Intravenous esmolol will convert up to 60 percent of patients with new-onset atrial flutter to sinus rhythm.
- Ibutilide, 0.1 mg/kg IV up to 1 mg, over 10 min, has a high success rate for conversion of atrial flutter (and atrial fibrillation) to sinus rhythm. Because of the possibility of provoking torsades de pointes, do not administer ibutilide to patients with hypokalemia, prolonged QT on the ECG, or congestive heart failure.
- Alternatives include digoxin (0.5 mg IV), verapamil (5 to 10 mg IV), or procainamide (see ventricular tachycardia management for dosing guidelines).

ATRIAL FIBRILLATION

Clinical Features

- Atrial fibrillation occurs when there are multiple small areas of atrial myocardium continuously discharging and contracting.
- The ECG characteristics of atrial fibrillation are (a) fibrillatory waves of atrial activity, best seen in leads V_1, V_2, V_3, and aV_F; and (b) irregular

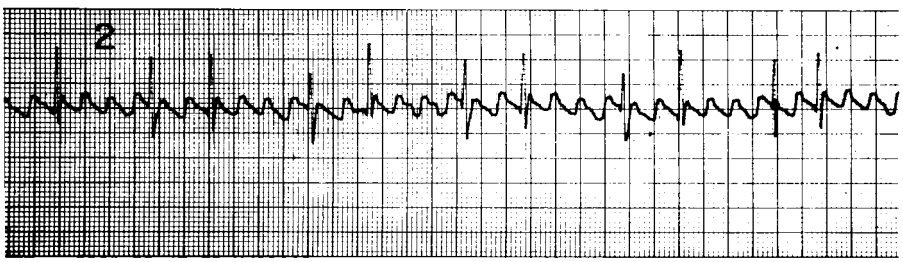

FIG. 4.9 Atrial flutter.

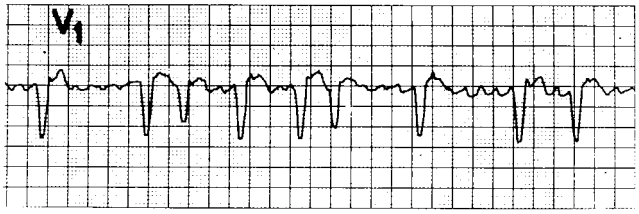

FIG. 4.10 Atrial fibrillation.

ventricular response, usually around 170 to 180 in patients with a healthy AV node (Fig. 4-10).
- Disease or drugs (especially digoxin) may reduce AV node conduction and markedly slow ventricular response.
- Predisposing factors for atrial fibrillation are increased atrial size and mass, increased vagal tone, and variation in refractory periods between different parts of atrial myocardium.
- Atrial fibrillation is usually found in association with four disorders: hypertension, ischemic heart disease, rheumatic heart disease, and thyrotoxicosis.
- In patients with left ventricular failure, left atrial contraction makes an important contribution to cardiac output. The loss of effective atrial contraction, as in atrial fibrillation, may produce heart failure in these patients.
- Conversion from chronic atrial fibrillation to sinus rhythm also carries up to a 1 to 2 percent risk of arterial embolism. Consider anticoagulation with heparin prior to conversion to sinus rhythm.

Emergency Department Care
- Atrial fibrillation with a rapid ventricular response and acute hemodynamic deterioration should be treated with synchronized cardioversion. Over 60 percent can be converted with 100 J, and over 80 percent with 200 J.
- Diltiazem 20 mg (0.25 mg/kg) IV over 2 min is extremely effective. An infusion of 10 mg/h is usually started after the initial dose to maintain control, and a second dose of 25 mg (0.35 mg/kg) can be given at 15 min if rate control is not achieved. Alternatives include digoxin (0.5 mg IV) and verapamil (5 to 10 mg IV).
- Once ventricular rate control has been achieved, chemical conversion can be considered with ibutilide (see comment earlier for atrial flutter), procainamide, or verapamil.

SUPRAVENTRICULAR TACHYCARDIA

Clinical Features
- Supraventricular tachycardia (SVT) is a regular, rapid rhythm that arises from either reentry or an ectopic pacemaker above the bifurcation of the bundle of His.
- The reentrant variety is clinically the most common (Fig. 4-11). These patients often present with acute, symptomatic episodes termed *paroxysmal supraventricular tachycardia* (*PSVT*).
- In patients with bypass tracts, reentry can occur in either direction. It usually occurs in a direction that goes down the AV node and up the bypass tract, producing a narrow QRS complex.
- Reentrant SVT can occur in a normal heart, or in

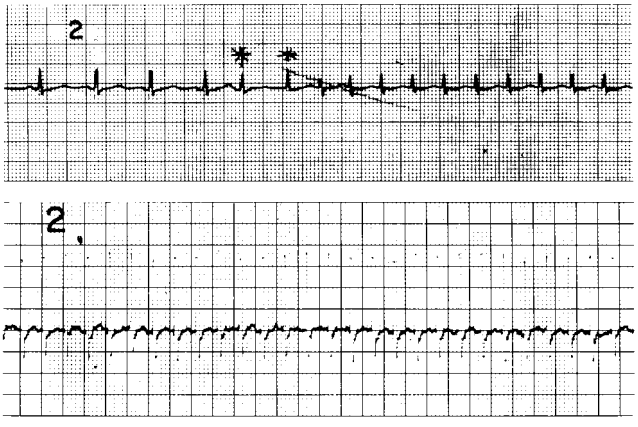

FIG. 4.11 Reentrant supraventricular tachycardia (SVT). Top: 2d (*) initiates run of PAT. Bottom: SVT, rate 286.

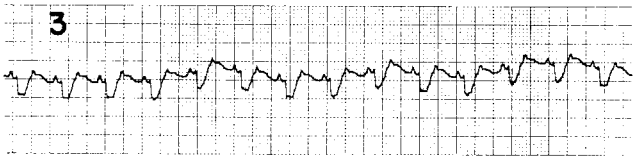

FIG. 4.12 Ectopic supraventricular tachycardia (SVT) with 2: AV conduction.

association with rheumatic heart disease, acute pericarditis, myocardial infarction, mitral valve prolapse, or one of the preexcitation syndromes.
- Ectopic SVT usually originates in the atria with an atrial rate of 100 to 250 (most commonly 140 to 200) (Fig. 4-12).
- Ectopic SVT may be seen in patients with acute myocardial infarction, chronic lung disease, pneumonia, alcoholic intoxication, and digoxin toxicity.

Emergency Department Care and Disposition

- The first attempt should be vagal maneuvers. These maneuvers can be done by themselves or after administration of drugs.
  a. Carotid sinus massage attempts to massage the carotid sinus and its baroreceptors against the transverse process of C6. Massage should be done for 10 s at a time, first on the side of the nondominant cerebral hemisphere, and should never be done simultaneously on both sides.
  b. Facial immersion in cold water for 6 to 7 s with the nostrils held closed (diving reflex). This maneuver is particularly effective in infants.
  c. The Valsalva maneuver done in the supine position appears to be the most effective vagal maneuver for the conversion of reentrant SVT. For maximal effectiveness, the strain phase must be adequate (usually at least 10 s).
- Adenosine, initially 6 mg rapid IV bolus. If there is no effect within 2 min, a second dose of 12 mg can be given. Fifty percent of patients experience distressing chest pain or flushing.
- Verapamil, 0.075 to 0.15 mg/kg (3 to 10 mg) IV over 15 to 60 s, with a repeat dose in 30 min, if necessary. Hypotension may occur but can be treated and/or prevented with calcium chloride, 4 mL of a 10% solution.
- Diltiazem, 20 mg (0.25 mg/kg) IV over 2 min.
- Further alternatives include esmolol (300 μg/kg/min), propranolol (0.5 to 1 mg IV), or digoxin (0.5 mg IV).
- Synchronized cardioversion should be done in any unstable patient with hypotension, pulmonary edema, or severe chest pain. The required dose is usually small, less than 50 J.

JUNCTIONAL RHYTHMS

Clinical Features

- If sinus node discharges slow or fail to reach the AV junction, junctional escape beats may occur, usually at a rate between 40 and 60, depending on the level of the pacemaker.
- Generally, junctional escape beats do not conduct retrograde into the atria, so a QRS complex without a P wave usually is seen (Fig. 4-13).
- Junctional escape beats may occur whenever there is a long enough pause in the impulses reaching the AV junction, such as in sinus bradycardia, slow phase of sinus dysrhythmia, AV block, or following premature beats.
- Sustained junctional escape rhythms may be seen with congestive heart failure, myocarditis, hyperkalemia, or digoxin toxicity. If the ventricular rate is too slow, myocardial or cerebral ischemia may develop.

Emergency Department Care and Disposition

- Isolated, infrequent junctional escape beats usually do not require specific treatment.
- If sustained junctional escape rhythms are producing symptoms, the underlying cause should be

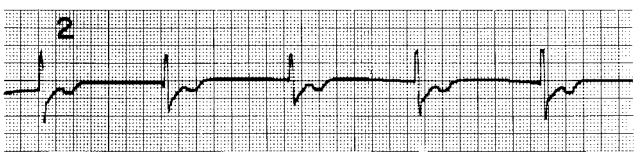

FIG. 4.13 Junctional escape rhythm, rate 42.

treated. Atropine can be used to accelerate temporarily the sinus node discharge rate and enhance AV nodal conduction.

VENTRICULAR DYSRHYTHMIAS

ABERRANT VERSUS VENTRICULAR TACHYDYSRHYTHMIAS

- In general, the majority of patients with wide complex tachycardia have ventricular tachycardia and should be approached as ventricular tachycardia, until proved otherwise.
- A preceding ectopic P wave is good evidence favoring aberrancy, although coincidental atrial and ventricular ectopic beats or retrograde conduction can occur. During a sustained run of tachycardia, AV dissociation favors a ventricular origin of the dysrhythmia.
- Postectopic pause: A fully compensatory pause is more likely after a ventricular beat, but exceptions occur.
- Fusion beats are good evidence for ventricular origin but, again, exceptions occur.
- A varying bundle branch block pattern suggests aberrancy.
- Coupling intervals are usually constant with ventricular ectopic beats, unless parasystole is present. Varying coupling intervals suggest aberrancy.
- Response to carotid sinus massage or other vagal maneuvers will slow conduction through the AV node and may abolish reentrant SVT and slow the ventricular response in other supraventricular tachydysrhythmias. These maneuvers have essentially no effect on ventricular dysrhythmias.
- A QRS duration of longer than 0.14 s is usually only found in ventricular ectopy or tachycardia.
- Historical criteria also have been found to be useful: a patient over 35 years old or history of myocardial infarction, congestive heart failure, or coronary artery bypass graft strongly suggest ventricular tachycardia in patients with wide complex tachycardia (WCT).

Emergency Department Care and Disposition

- As with ventricular tachycardia, start with lidocaine 1 to 1.5 mg/kg IV; may repeat up to 3 mg/kg.
- Adenosine 6 mg IV push may be tried prior to procainamide (see ventricular tachycardia management later for administration guidelines).

PREMATURE VENTRICULAR CONTRACTIONS

Clinical Features

- Premature ventricular contractions (PVCs) are due to impulses originating from single or multiple areas in the ventricles.
- The ECG characteristics of PVCs are (a) a premature and wide QRS complex; (b) no preceding P wave; (c) the ST segment and T wave of the PVC are directed opposite the major QRS deflection; (d) most PVCs do not affect the sinus node, so there is usually a fully compensatory postectopic pause, or the PVC may be interpolated between two sinus beats; (e) many PVCs have a fixed coupling interval (within 0.04 s) from the preceding sinus beat; and (f) many PVCs are conducted into the atria, producing a retrograde P wave (Fig. 4-14).
- PVCs are very common, occur in most patients with ischemic heart disease, and are universally found in patients with acute myocardial infarction. Other common causes of PVCs include digoxin toxicity, congestive heart failure, hypokalemia, alkalosis, hypoxia, and sympathomimetic drugs.

Emergency Department Care and Disposition

- Most acute patients with PVCs will respond to intravenous lidocaine (1 mg/kg IV), although some patients may require procainamide. Although single studies have suggested benefit, pooled data and meta-analysis find no reduction in mortality from either suppressive or prophylactic treatment of PVCs.

ACCELERATED IDIOVENTRICULAR RHYTHM

Clinical Features

- The ECG characteristics of accelerated idioventricular rhythm (AIVR) are (a) wide and regular QRS complexes; (b) rate between 40 and 100, often close to the preceding sinus rate; (c) most runs of short duration (3 to 30 beats); and (d) an AIVR often beginning with a fusion beat (Fig. 4-15).
- This condition is found most commonly with an acute myocardial infarction.

Emergency Department Care and Disposition

- Treatment is not necessary. On occasion, AIVR may be the only functioning pacemaker, and suppression with lidocaine can lead to cardiac asystole.

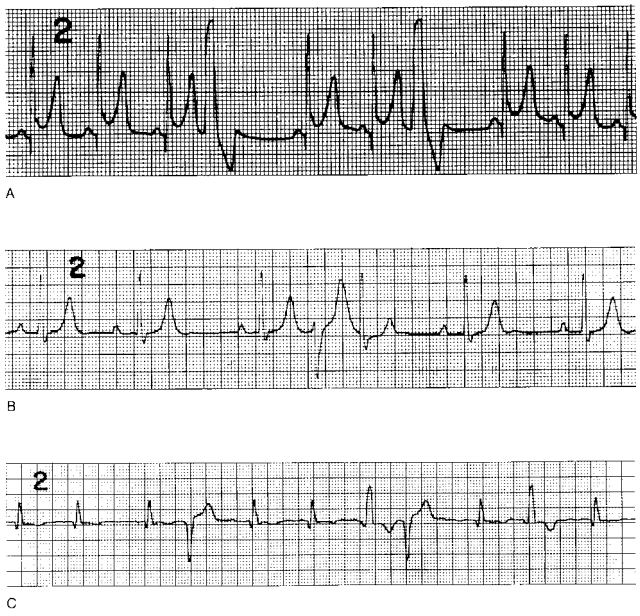

FIG. 4.14 Premature ventricular contractions (PVCs). Top: unifocal PVC. Center: interpolated PVC. Bottom: multifocal PVCs.

VENTRICULAR TACHYCARDIA

Clinical Features

- Ventricular tachycardia is the occurrence of 3 or more beats from a ventricular ectopic pacemaker at a rate greater than 100.
- The ECG characteristics of ventricular tachycardia are (a) wide QRS complexes; (b) rate greater than 100 (most commonly 150 to 200); (c) rhythm is usually regular, although there may be some beat-to-beat variation; and (d) QRS axis is usually constant (Fig. 4-16).
- The most common causes of ventricular tachycardia are ischemic heart disease and acute myocardial infarction. Ventricular tachycardia cannot be differentiated from SVT with aberrancy on the basis of clinical symptoms, blood pressure, or heart rate.
- Adenosine appears to cause little harm in patients with ventricular tachycardia and has potential merit for the treatment of wide QRS complex tachycardias.

Emergency Department Care and Disposition

- Unstable patients, or those in cardiac arrest, should be treated with synchronized cardioversion. Ventricular tachycardia can be converted with energies as low as 1 J, and over 90 percent can be converted with less than 10 J. Advanced Cardiac Life Support (ACLS) guidelines recommend that pulseless ventricular tachycardia be *defibrillated* (unsynchronized cardioversion) with 200 J. Another alternative for unstable patients is intravenous amiodarone. See treatment recommendations under ventricular fibrillation.
- Clinically stable patients should be treated with intravenous antidysrhythmics.
    a. Lidocaine 75 mg (1.0 to l.5 mg/kg) IV over 60 to 90 s, followed by a constant infusion at 1 to 4 mg/min (10 to 40 μg/kg/min). A repeat bolus dose of 50 mg lidocaine may be required during the first 20 min to avoid a subtherapeutic dip in serum level due to the early distribution phase.

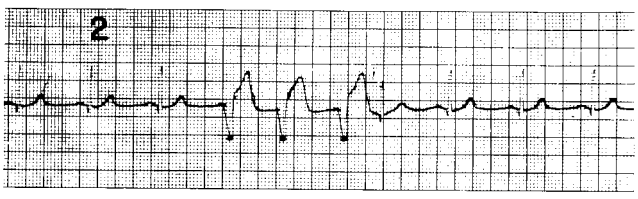

FIG. 4.15 Accelerated idioventricular rhythms (AIVR).

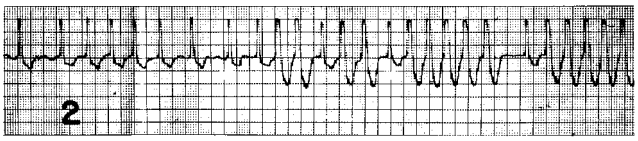

FIG. 4.16 Ventricular tachycardia.

b. Procainamide IV at less than 30 mg/min until the dysrhythmia converts, the total dose reaches 15 to 17 mg/kg in healthy patients (12 mg/kg in patients with congestive heart failure), or early signs of toxicity develop, with hypotension or QRS prolongation. The loading dose should be followed by a maintenance infusion of 2.8 mg/kg/h in normal subjects.
c. Bretylium 500 mg (5 to 10 mg/kg) IV over 10 min, followed by a constant infusion at 1 to 2 mg/min.

TORSADES DE POINTES

Clinical Features

- *Atypical ventricular tachycardia* (torsades de pointes, or twisting of the points) is where the QRS axis swings from a positive to negative direction in a single lead (Fig. 4-17).
- Drugs that further prolong repolarization—quinidine, disopyramide, procainamide, phenothiazines, tricyclic antidepressants—exacerbate this dysrhythmia.

Emergency Department Care and Disposition

- Reports have revealed that magnesium sulfate, 1 to 2 g IV over 60 to 90 s followed by an infusion of 1 to 2 g/h, is effective in abolishing torsades de pointes.
- To date, treatment for torsades de pointes consisted of accelerating the heart rate (thereby shortening ventricular repolarization) with isoproterenol (2 to 8 μg/min), while making arrangements for a ventricular pacemaker to overdrive the heart at rates of 90 to 120. Temporary pacing is the most effective and safest method to treat torsades de pointes and prevent its recurrence.

VENTRICULAR FIBRILLATION

Clinical Features

- Ventricular fibrillation is the totally disorganized depolarization and contraction of small areas of ventricular myocardium—there is no effective ventricular pumping activity. Ventricular fibrillation is never accompanied by a pulse or blood pressure.
- The ECG of ventricular fibrillation shows a fine-to-coarse zigzag pattern without discernible P waves or QRS complexes (Fig. 4-18).
- Ventricular fibrillation is most commonly seen in patients with severe ischemic heart disease, with or without an acute myocardial infarction.
- Primary ventricular fibrillation occurs suddenly, without preceding hemodynamic deterioration, whereas secondary ventricular fibrillation occurs after a prolonged period of left ventricular failure or circulatory shock.

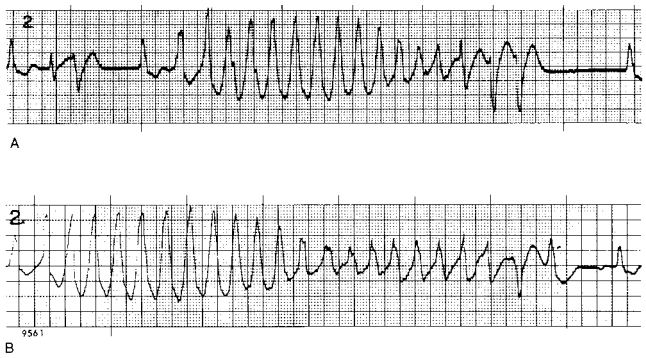

FIG. 4.17 Two examples of short runs of atypical ventricular tachycardia showing sinusoidal variation in amplitude and direction of the QRS complexes: ''Le torsades de pointes'' (twisting of the points). Note that the top example is initiated by a late-occurring PVC (lead II).

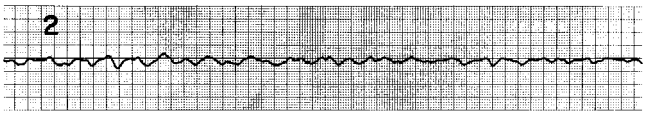

FIG. 4.18 Ventricular fibrillation.

Emergency Department Care and Disposition

- Current ACLS guidelines recommend immediate electrical defibrillation with 200 J. If ventricular fibrillation persists, defibrillation should be repeated immediately, with 200 to 300 J at the second attempt, and increased to 360 J at the third attempt.
- If the initial three attempts at defibrillation are unsuccessful, cardiopulmonary resuscitation (CPR) and intubation should be initiated; further electrical defibrillations should be done after the administration of various intravenous drugs, according to ACLS guidelines.
- Epinephrine in standard dose should be administered, 1 mg IV. If this is not successful, high-dose epinephrine may be given subsequently, 0.1 mg/kg. Repeat every 3 to 5 min.
- Defibrillation should be attempted after each drug administration, at 360 J, unless lower energy levels have been previously successful.
- Successive antidysrhythmics should then be administered with defibrillation attempted after each drug. The recommended sequence is lidocaine 1.5 mg/kg, bretylium 5 mg/kg, then consider magnesium 2 g IV, and procainamide (see dosing guidelines earlier).
- Amiodarone 150 mg over 10 min, followed by 1 mg/min for 6 h, may become a preferred treatment for ventricular fibrillation/ventricular tachycardia after lidocaine has failed.

CONDUCTION DISTURBANCES

ATRIOVENTRICULAR BLOCK

- First-degree AV block is characterized by a delay in AV conduction, manifested by a prolonged PR interval. First-degree AV block needs no treatment and will not be discussed further.
- Second-degree AV block is characterized by intermittent AV conduction—some atrial impulses reach the ventricles and others are blocked.
- Third-degree AV block is characterized by complete interruption in AV conduction.

SECOND-DEGREE MOBITZ I (WENCKEBACH) ATRIOVENTRICULAR BLOCK

Clinical Features

- With this block there is progressive prolongation of AV conduction (and the PR interval) until atrial impulse is completely blocked. Usually, only a single atrial impulse is blocked.
- After the dropped beat, the AV conduction returns to normal and the cycle usually repeats itself, with either the same conduction ratio (fixed ratio) or a different conduction ratio (variable ratio).
- The Wenckebach phenomenon has a seeming paradox. Even though the PR intervals progressively lengthen prior to the dropped beat, the increments by which they lengthen decrease with successive beats; this produces a progressive shortening of the R-R interval prior to the dropped beat (Fig. 4-19).
- This block is often transient and usually associated with an acute inferior myocardial infarction, digoxin toxicity, myocarditis, or is seen after cardiac surgery.

Emergency Department Care and Disposition

- Specific treatment is not necessary unless slow ventricular rates produce signs of hypoperfusion.
- Atropine, 0.5 mg, repeated every 5 min, as neces-

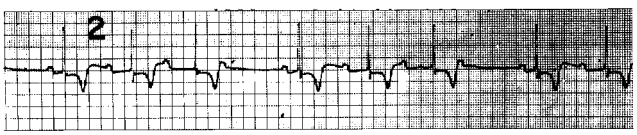

FIG. 4.19 Second-degree Mobitz I (Wenckebach) AV block 4:3 AV conduction.

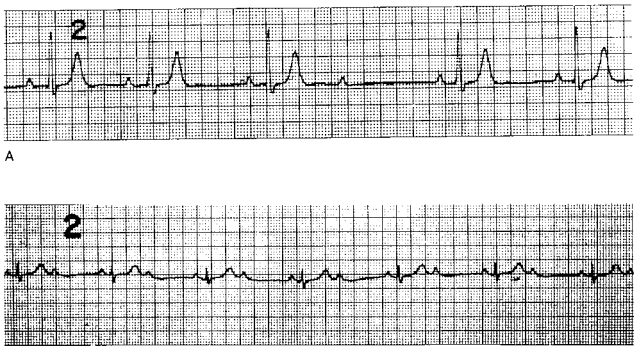

FIG. 4.20 Top: second-degree Mobitz II AV block. Bottom: second-degree AV block with 2:1 AV conduction.

sary, titrated to the desired effect or until the total dose reaches 3.0 mg.
- Although rarely needed, transcutaneous pacing may be used.

SECOND-DEGREE MOBITZ II ATRIOVENTRICULAR BLOCK

Clinical Features
- With this block, the PR interval remains constant before and after the nonconducted atrial beats (Fig. 4-20). One or more beats may be nonconducted at a single time.
- The QRS complexes are usually wide. When second-degree AV block occurs with a fixed conduction ratio of 2:1, it is not possible to differentiate between a Mobitz type I (Wenckebach) or Mobitz type II block.
- Type II blocks imply structural damage to the infranodal conducting system, are usually permanent, and may progress suddenly to complete heart block, especially in the setting of an acute myocardial infarction.

Emergency Department Care and Disposition
- Atropine (0.5 to 1 mg IV push, may repeat up to 3.0 mg total dose) should be the first drug used.
- Transcutaneous cardiac pacing is a useful modality in patients unresponsive to atropine.
- Most cases, especially in the setting of acute myocardial infarction, will require permanent transvenous cardiac pacing.

THIRD-DEGREE (COMPLETE) ATRIOVENTRICULAR BLOCK

Clinical Features
- In third-degree AV block, there is no AV conduction. The ventricles are paced by an escape pacemaker at a rate slower than the atrial rate (Fig. 4-21).
- When third-degree AV block occurs at the AV node, a junctional escape pacemaker takes over with a ventricular rate of 40 to 60 and, since the rhythm originates above the bifurcation of the bundle of His, the QRS complexes are narrow.
- When third-degree AV block occurs at the infranodal level, the ventricles are driven by a ventricular escape rhythm at a rate of less than 40. Third-degree AV block located in the bundle branch or Purkinje system invariably have escape rhythms with wide QRS complexes.
- Nodal third-degree AV block may develop in up to 8 percent of acute inferior myocardial infarctions where it is usually transient, although it may last for several days.
- Infranodal third-degree AV blocks indicate structural damage to the infranodal conducting system, as seen with an extensive acute anterior myocardial infarction. The ventricular escape pacemaker is usually inadequate to maintain cardiac output

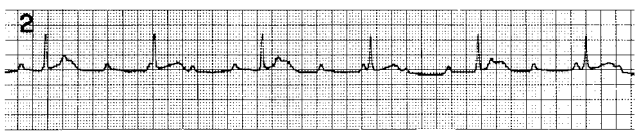

FIG. 4.21 Third-degree AV block.

and is unstable with periods of ventricular asystole.

Emergency Department Care and Disposition

- Third-degree AV blocks should be treated the same as second-degree Mobitz I AV blocks with atropine or ventricular demand pacemaker, as required. External cardiac pacing can be performed before transvenous pacemaker placement.

PRETERMINAL RHYTHMS

PULSELESS ELECTRICAL ACTIVITY

- Pulseless Electrical Activity (PEA) is the presence of electrical complexes without accompanying mechanical contraction of the heart.
- Potential causes should be diagnosed and treated: severe hypovolemia, cardiac tamponade, tension pneumothorax, massive pulmonary embolus, and rupture of the ventricular wall.
- Stabilizing treatment includes epinephrine 1 mg IV, followed by high-dose therapy of 0.1 mg/kg if the first dose is not successful. Repeat epinephrine every 3 to 5 min.
- Atropine 1 mg IV, up to 3 mg total, is also acceptable therapy if the electrical conduction is slow.

ASYSTOLE (CARDIAC STANDSTILL)

- Asystole is the complete absence of cardiac electrical activity.
- Treatment is the same as for pulseless electrical activity with the addition of transcutaneous pacing if the preceding measures fail (although this is rarely successful).

PREEXCITATION SYNDROMES

Clinical Features

- Preexcitation occurs when some portion of the ventricles is activated by an impulse from the atria sooner than would be expected if the impulse were transmitted down the normal conducting pathway.
- All forms of preexcitation are felt to be due to accessory tracts that bypass all or part of the normal conducting system, the most common form being Wolff-Parkinson-White syndrome (WPW; Fig. 4-22).
- There is a high incidence of tachydysrhythmias in patients with WPW—atria flutter (about 5 percent), atrial fibrillation (10 to 20 percent), and paroxysmal reentrant SVT (40 to 80 percent).

Emergency Department Care and Disposition

- Reentrant SVT (orthodromic, narrow QRS complex) in the WPW syndrome can be treated like other cases of reentrant SVT. Adenosine, 6 mg IV, or verapamil, 5 to 10 mg IV, are very successful at terminating this dysrhythmia in patients with WPW, but β-adrenergic blockers usually are ineffective.
- Tachycardia with a wide QRS complex is usually associated with a short refractory period in the

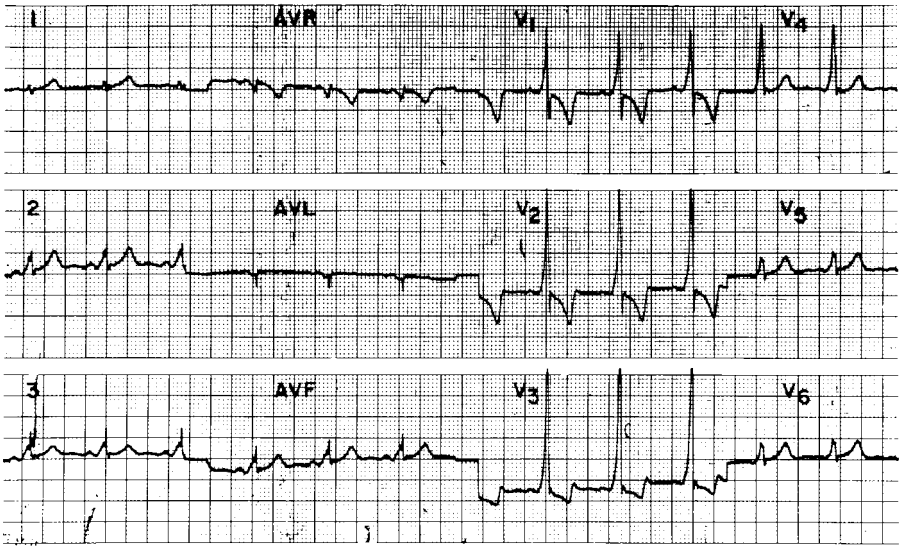

FIG. 4.22 Type A Wolff-Parkinson-White syndrome.

bypass tract; patients with this type of tachycardia are at risk for rapid ventricular rates and degeneration into ventricular fibrillation. Stable patients should be treated with intravenous procainamide and unstable patients should be cardioverted. β-adrenergic or calcium channel blockers (i.e., verapamil) should be avoided.
- Atrial flutter or fibrillation with a rapid ventricular response is best treated with cardioversion.

CARDIOVASCULAR PHARMACOLOGY

- The Vaughan-Williams classification of antidysrhythmics classifies drugs based on their ability to block sodium channels (class I), block calcium channels (class IV), block β-adrenergic receptors (class II), or prolong the refractory period (class III). Digoxin and adenosine do not fit into this scheme.

CLASS I ANTIDYSRHYTHMIC AGENTS

Procainamide
- Procainamide blocks sodium channels and depresses the speed of impulse conduction (phase 0) of the cardiac action potential.
- These effects directly depress myocardial conduction, suppress fibrillatory activity in the atria and ventricles, and prevent ectopic or reentrant dysrhythmias.
- Indications and dosing information for procainamide are listed in Table 4-1.
- Contraindications include complete AV heart block, second- or third-degree heart block, long QT intervals, and torsades de pointes.
- Lower doses (50 percent of standard doses) are necessary for patients with congestive heart failure, hypotensive states, and hepatic or renal failure.
- Adverse effects of procainamide include myocardial depression, prolongation of the QRS and QT interval, impairment of AV conduction, ventricular fibrillation, torsades de pointes, and hypotension.

Lidocaine
- Lidocaine preferentially depresses the automaticity (phase 4) of the distal conduction system of depolarized and ischemic tissue; it does not affect normal myocardium.
- Lidocaine is not effective against atrial dysrhythmias because it preferentially acts on the His-Purkinje and more distal conduction system.
- Indications and dosing information for lidocaine are listed in Table 4-1.
- Lidocaine is contraindicated in patients with known sensitivities to amide-type local anesthetics and those with high degrees of sinoatrial or AV block.
- Adverse effects from lidocaine usually occur when the drug is administered too rapidly in a conscious patient, when excessive doses are administered, or when a drug interaction potentiates toxicity.
- Symptoms of mild lidocaine toxicity that correlate with levels greater than 5 g/mL include slurred speech, drowsiness, confusion, nausea, vertigo, ataxia, tinnitus, paresthesias, and muscle twitching.
- An abrupt change in mental status is a classic symptom of lidocaine toxicity. Serious symptoms occurring at plasma levels greater than 9 g/mL may include psychosis, seizures, and respiratory depression, and high degrees of sinoatrial or atrioventricular (AV) block.

CLASS II ANTIDYSRHYTHMICS: BETA BLOCKERS

Propranolol
- In therapeutic doses, the major effect of propranolol is its β-adrenergic blocking activity. The drug blocks the effects of catecholamines on β receptors, inhibiting chronotropic, inotropic, and vasodilator responses to β-adrenergic stimulation.
- Propranolol slows the sinus rate, depresses AV conduction, decreases cardiac output, reduces blood pressure on exercise, and reduces both supine and standing blood pressures.
- Indications and dosing information for propranolol are listed in Table 4-1.
- The drug is generally not given to patients with asthma or allergic rhinitis and is contraindicated in those with sinus bradycardia or advanced sinoatrial or AV block. Propranolol should also not be used in patients with congestive heart failure or cardiogenic shock, unless these conditions are due to tachydysrhythmias.

Esmolol
- Esmolol prevents excessive adrenergic stimulation on the myocardium by selectively blocking the β_1 receptors, thus producing an increase in sinus cycle length, a prolongation of sinoatrial nodal recovery time, and a prolongation in conduction through the AV node.

TABLE 4-1 Summary of Dosing and Administration of Common Antidysrhythmic Agents*

AGENT	INDICATION	DOSE
Procainamide (Pronestyl)	Treatment of ventricular dysrhythmias and recurrent ventricular tachycardias refractory to lidocaine. Second-line therapy for pulseless VT/VF. Convert supraventricular dysrhythmias, particularly those associated with WPW.	20-mg/min IV infusion. Stop when: total dose of 17 mg/kg reached, QRS complex widens more than 50%, QT interval prolongation, dysrhythmia is controlled, or hypotension develops.
Lidocaine (Xylocaine)	VF/pulseless VT	1.5-mg/kg IV bolus, then 0.5-mg/kg bolus q5min as needed until 3 mg/kg total given. Follow with continuous infusion at 2 to 4 mg/min. Increase to 2 to 2.5 times the IV dose for ET administration. Mix with saline or sterile water for a total drug volume of 10 mL. Decrease the loading dose and maintenance by 50% in patients more than 70 years of age, those with CHF, liver disease, or impaired hepatic blood flow.
Atenolol (Tenormin)	Hypertension Angina AMI	5 mg IV over 5 min. Repeat in 10 min, then convert to PO medication.
Metoprolol (Lopressor)	Hypertension Angina AMI	5 mg IV over 5 min for three doses, then convert to 50 mg PO q12h.
Esmolol (Brevibloc)	Ventricular rate control of SVT	500-μg/kg bolus over 1 min, with a maintenance infusion of 50 μg/kg/min over 4 min. If inadequate clinical response, reload with 500-μg/kg bolus over 1 min followed by a maintenance infusion of 100 μg/kg/min. Continue the cycle of reloading with 500 μg/kg followed by an incremental increase of the maintenance infusion by 50 μg/kg/min until the desired heart rate is obtained. If hypotension occurs, hold the bolus and decrease the infusion by 50 μg/kg/min.
Propranolol (Inderal)	Life-threatening tachydysrhythmias	The dose is 0.5 mg to 1.0 mg IV up to 3 mg at a rate not exceeding 1.0 mg/min. Repeat dose in 2 min if necessary. Of note, esmolol is as effective in reducing heart rate; it has the advantage of a more rapid reversal of β blockade and less β_2 effects as compared with propranolol.
Labetalol (Normodyne, Trandate)	Hypertensive crisis Dissecting aneurysm Pregnancy-induced hypertension Pheochromocytoma	20 mg IV (0.25 mg/kg in an 80-kg patient) over 2 min. Double the dose until desired supine BP achieved or 300 mg cumulative dose is given. Or, 0.5 mg/min to 2 mg/min IV as a continuous infusion until desired response or 300 mg cumulative dose is given.

- Indications and dosing information for esmolol are listed in Table 4-1.
- The most common adverse effect associated with esmolol use is hypotension, which occurs in approximately 20 to 50 percent of patients being treated for SVT.

Labetalol

- Labetalol possesses membrane-stabilizing effects and thus has some antidysrhythmic action; however, the drug is often used as an antihypertensive agent because it blocks both α- and β-adrenergic receptors.
- Labetalol decreases heart rate, contractility, cardiac output, cardiac work, and total peripheral resistance.
- Indications and dosing information for labetalol are listed in Table 4-1.
- The most common adverse effect associated with labetalol use is orthostatic hypotension.
- Adverse central nervous system (CNS) effects that may occur include light-headedness, drowsiness, dizziness, fatigue, and lethargy.
- Avoid the use of IV labetalol in patients with risks for intracranial bleeding as a hypotensive episode can induce CNS herniation.

CLASS III ANTIARRHYTHMIC AGENTS

Amiodarone

- Amiodarone is classified as a class III antidysrhythmic; it has a complex pharmacodynamic pro-

TABLE 4-1 (*Continued*)

AGENT	INDICATION	DOSE
Amiodarone	Life-threatening ventricular tachydysrhythmias refractory to first-line agents	150 mg IV over 10 min, then 1 mg/min for 6 h, then 0.5 mg/min as maintenance. Re-bolus with 150 mg over 10 min for breakthrough VT or VF.
Bretylium	VF/pulseless VT Recurrent VT with a pulse resistant to first-line agents	1. Give 5 mg/kg by IV push followed by a 20-mL flush. 2. Defibrillate. Re-bolus with 10 mg/kg by IV push followed by a 20-mL flush. 3. Repeat step 2 q15 min until a maximal cumulative dose of 35 mg/kg is given.
Diltiazem (Cardizem)	Ventricular rate control of SVT Conversion for NSR of PSVT	1. Give 0.25 mg/kg of actual body weight (average adult dose is 20 mg) IV bolus over 2 min. 2. If inadequate response, then re-bolus with 0.35 mg/kg (average adult dose is 25 mg) over 2 min. 3. Give a continuous infusion at 5 to 15 mg/h to maintain reduced heart rate in patients with atrial fibrillation and/or flutter.
Verapamil (Isoptin, Calan)	Ventricular rate control of SVT Conversion of NSR of PSVT	Adults: 5 to 10 mg (0.075 to 0.15 mg/kg) IV over 2 to 3 min. A second dose may be given 15 to 30 min later as needed. Children 1 to 15 years: 0.1 to 0.3 mg/kg IV (usual dose range, 2 to 5 mg) over 2 to 3 min. A second dose may be given 15 to 30 min later as needed. Infants below 1 year: Do not use. There is an association with severe bradycardia, hypotension, and asystole.
Adenosine (Adenocard)	Convert PSVT into NSR Uncover atrial rhythm of a narrow complex tachycardia of unknown etiology	1. Give 6-mg IV bolus over 1 to 3 s followed by a 20-mL IV flush. 2. If no response, re-bolus wth 12 mg IV push followed by a 20-mL IV flush. 3. If no response, a third 12-mg IV bolus may be given followed by a 20-mL IV flush.
Magnesium ($MgSO_4$)	Torsades de pointes AMI and cardiac arrest with suspected hypomagnesemia Preeclampsia, eclampsia, and preterm labor	1 to 2 g IV in 100 mL D_5W over 1 to 2 min. 1 to 2 g IV in 100 mL D_5W over 20 min.

* ABBREVIATIONS: AMI = acute myocardial infarction; SVT = supraventricular tachycardia; VT/VF = ventricular tachycardia/ventricular fibrillation; CHF = congestive heart failure; ET = endotracheal; BP = blood pressure; NSR = normal sinus rhythm; PSVT = paroxysmal supraventricular tachycardia.

file that also includes class I, class II, and class IV properties.
- The antifibrillatory effect of amiodarone is caused by inhibition of potassium ion fluxes that normally occur during phases 2 and 3 of the cardiac cycle.
- Indications and dosing information are listed in Table 4-2.
- With parenteral use, hypotension is the most common side effect. Bradycardia may also occur.
- Amiodarone should not be used in patients with marked sinus bradycardia or second- and third-degree AV block unless emergent pacing is available.

BRETYLIUM

- Bretylium is a class III drug with a biphasic cardiovascular response. There is an appreciable increase in heart rate, blood pressure, and cardiac output after intravenous infusion. This effect lasts approximately 20 min.
- Next, there is a sympatholytic response with subsequent reduction in heart rate, blood pressure, and systemic vascular resistance.
- Bretylium prolongs the action potential duration and the effective refractory period in the ventricular myocardium.
- Indications and dosing information for bretylium are listed in Table 4-1.
- Postural hypotension is the most common adverse reaction and may occur within 15 to 30 min in as many as 60 percent of patients. If this occurs, the patient should be placed in a supine or Trendelenburg position and be resuscitated with crystalloid fluids.
- Bretylium should be avoided, if possible, in the

TABLE 4-2 Vasopressor Agents

AGENT	INDICATIONS AND DOSAGE	REMARKS
Epinephrine	Cardiac arrest (asystole, VF/pulseless VT), symptomatic bradycardia, PEA: IV: 1 mg (1:10,000) q3min ET: 2–2.5 mg (1:10,000) diluted with NSS to a volume of 10 mL Anaphylaxis, bronchospasm: SQ: 0.3 mg (1:1000) q20min Pressor and chronotropic agent IV: 1 μg/min titrated to desired effect (2 to 10 μg/min)	The use of high-dose epinephrine in cardiac arrest is neither recommended nor supported by the AHA. Alpha effects are responsible for increased coronary artery perfusion pressure, which may promote return of spontaneous circulation. β effects may precipitate myocardial ischemia.
Dopamine	Renal dose (dopaminergic effects): IV: 1–5 μg/kg/min infusion Cardiac dose (β_1 effects): IV: 5–10 μg/kg per minute infusion Vasopressor dose (α effects): IV: 10–20 μg/kg/min infusion	Renal dose improves renal blood flow. Cardiac doses exert positive inotropic and chronotropic effects. Indicated for the treatment for cardiogenic shock. Vasopressor doses cause vasoconstriction and cardiac stimulation. If shock is refractory to 20 μg/kg/min, add norepinephrine.
Dobutamine	Inotropic agent with little effect on SVR: IV: 2–20 μg/kg/min infusion	Good inotropy, weak chronotropy. Little α effects. Useful adjunct to dopamine for treating cardiogenic shock. May use alone for cardiac decompensation associated with normal or slightly low blood pressure.
Isoproterenol	Inotropy without any α effects: IV: 2–10 μg/min infusion	Used for bradycardia and heart block associated with a denervated heart (e.g., a transplanted heart) until pacing capabilities available. Increases myocardial oxygen consumption, which limits clinical usefulness in the adult population. Pediatric asthma.
Amrinone	Inotropy for CHF therapy: IV: 0.75 mg/kg over 3–5 min, then maintenance infusion at 5–10 μg/kg per minute	Positive inotropy with potent vasodilatation and increased stroke volume. Associated with thrombocytopenia Cardiac dysrhythmias more pronounced with milrinone therapy.
Phenylephrine	Vasopressor: IV: 40 to 60 μg/min, titrate until clinical response or maximum infusion of 180 μg/min reached	Pure alpha agonist, no β effects. α effects of epinephrine and norepinephrine are more potent. Avoid in cardiogenic shock.
Norepinephrine	Vasopressor: (septic shock, sympathectomy) IV: 0.5–1 μg/min initial infusion. Increase infusion until clinical response or infusion rate reaches 30 μg/min Standard adult dose is 2 to 12 μg/min	Powerful vasoconstrictor with some β_1 cardiac stimulatory effects. Unlike epinephrine, it lacks β_2 effects. Useful for dopamine-refractory septic shock. Associated with reflex bradycardia. High doses associated with cardiac irritability.

ABBREVIATIONS: VF = ventricular fibrillation; VT = ventricular tachycardia; AHA = American Heart Association; PEA = pulseless electrical activity; ET = endotracheal tube; NSS = normal saline solution; SVR = systemic vascular resistance; CHF = congestive heart failure.

setting of digoxin toxicity, since catecholamines are believed to exacerbate the toxic effects of digoxin.

CLASS IV ANTIDYSRHYTHMIC AGENTS: CALCIUM CHANNEL BLOCKERS

- Some studies suggest an increased risk of adverse cardiovascular events, particularly in the presence of left ventricular dysfunction;[1] other studies dispute these findings.[2] Currently, the American College of Cardiology/American Heart Association (ACC/AHA) Guidelines for the Management of Acute Myocardial Infarction do not recommend calcium channel blockers for routine use in acute myocardial infarction since β-adrenergic blocking agents are generally a more appropriate choice.
- However, if beta blockers are ineffective or contraindicated, verapamil or diltiazem may be given in patients with acute myocardial infarction without evidence of congestive heart failure, left ventricular dysfunction, or AV block.

VERAPAMIL

- Verapamil, a calcium channel blocking agent, is a class IV antidysrhythmic agent. In diseased tissue, verapamil decreases conduction velocity, prolongs the refractory period in the AV node, and decreases the discharge rate in the sinoatrial node.

- Verapamil interrupts the AV node reentrant pathway associated with PSVT, thus causing the myocardium to return to normal sinus rhythm.
- In addition, verapamil can slow ventricular response in patients with atrial fibrillation and/or flutter by its action on the AV node.
- Indications and dosing information for verapamil are listed in Table 4-1.
- Verapamil should be avoided in patients with WPW syndrome who present in atrial fibrillation or flutter since vertricular fibrillation may occur.[3]
- Pretreatment with calcium chloride may prevent serious adverse effects.
- Incidence of hypotension is 5 to 10 percent with IV administration and may rarely require treatment with IV calcium salts or vasopressors.
- Conduction disturbances, such as bradycardia, AV block, and bundle branch block, occur in approximately 2 percent or fewer of patients and usually respond to a dosage reduction or discontinuation of the drug.

Diltiazem

- Diltiazem slows AV nodal conduction time and prolongs AV nodal refractoriness.
- The ventricular rate is slowed in patients with a rapid ventricular response during atrial fibrillation or atrial flutter.
- PSVT is converted to normal sinus rhythm by interrupting the reentry circuit in AV nodal reentrant tachycardias and reciprocating tachycardias (e.g., WPW syndrome).
- Indications and dosing information are listed in Table 4-1.
- Cardiovascular adverse effects of diltiazem may include angina, bradycardia, asystole, congestive heart failure, AV block, bundle branch block, hypotension, and palpitations.

OTHER ANTIDYSRHYTHMIC AGENTS

Adenosine

- The positive inotropic, chronotropic, and dromotropic response of catecholamines depends on cyclic adenosine 5′-monophosphate (cyclic AMP). Adenosine exerts antiadrenergic effects by inhibiting the adenyl cyclase/cyclic AMP pathway.
- Adenosine terminates PSVT primarily via blockade of the AV node without altering conduction through accessory pathways, as is seen with the WPW syndrome. Reentrant SVTs not involving the AV node are not terminated by adenosine.
- Onset of action is within approximately 30 s, with a duration of 60 to 90 s. The drug is rapidly metabolized in the blood, with a half-life less than 7 s.
- Indications and dosing information for adenosine are listed in Table 4-1.
- When adverse effects occur due to adenosine, they are minor and well tolerated because they last less than 1 min due to the drug's short half-life. The most common are dyspnea, cough, syncope, vertigo, paresthesias, numbness, nausea, and metallic taste.
- Cardiovascular adverse effects may include facial flushing, headache, diaphoresis, palpitations, retrosternal chest pain, sinus bradydysrhythmias (i.e., bradycardia, sinus arrest, AV block), atrial tachydysrhythmias (i.e., atrial fibrillation or flutter), PVCs, and hypotension.

Magnesium

- Magnesium affects skeletal and smooth muscle contractility, vasomotor tone, and neuronal transmission directly via the Na^+, K^+-ATPase pump and indirectly via calcium blocking activity.
- It increases membrane potential, prolongs AV conduction, and increases the absolute refractory period.
- Indications and dosing information are listed in Table 4-1.
- Hypotension is the predominant adverse effect. Other signs of hypermagnesemia include flushing, sweating, CNS depression, depression of reflexes, flaccid paralysis, depression of cardiac function, circulatory collapse, hypothermia, and fatal respiratory paralysis.

VASOACTIVE DRUGS: VASOACTIVE AND INOTROPIC AGENTS

- Vasoactive drugs have two functions: (1) to improve cardiac perfusion pressure during cardiac arrest, and (2) to support the circulation during hemodynamic compromise.
- Epinephrine is the agent most frequently employed. Currently, no evidence exists to support the superiority of alternative agents although many have been tested in animal models.
- Adrenergic agents are divided into pure α agents (phenylephrine), mixed α and β agents (epinephrine, norepinephrine, dopamine), and pure β or primarily β agonists (isoproterenol, dobutamine).
- The α receptors are found primarily in blood vessels, where α stimulation causes vasoconstriction. The β agonists work primarily on the heart and promote increased heart rate, increased contractility, and increased myocardial oxygen consump-

tion. The β_2 receptors are found in smooth muscle of the bronchi, blood vessels, and uterus; stimulation causes bronchodilatation, vasodilatation, and uterine relaxation.
- Indications, dosing information, and guidelines for the use of the commonly used vasoactive drugs are listed in Table 4-2.

ATROPINE
- Atropine sulfate, an antimuscarinic agent, enhances sinus node automaticity and AV conduction by blocking vagal activity; thus it has been termed a *parasympatholytic* drug. It has anticholinergic properties.
- Atropine is indicated as the treatment of choice for increasing heart rate in hemodynamically unstable bradycardias (e.g., decreased heart rate with hypotension, altered mental status, escape beats, and chest pain).
- The dose of atropine for hemodynamically unstable bradycardias is 0.5 mg rapid IV push, repeated as necessary every 3 to 5 min until a desired heart rate is achieved. Bolus doses of 1 mg can be given for asystole and repeated once if necessary. A total dose of 3 mg (0.04 mg/kg) results in full vagolytic blockade in humans. Atropine can be administered by IV push, IM, and via the ET tube.
- Atropine is not indicated for bradycardia in hemodynamically stable patients. If administered, marked increases in heart rate can increase myocardial oxygen consumption, possibly inducing ischemia and precipitating ventricular tachydysrhythmias (ventricular tachycardia and ventricular fibrillation).

VASODILATOR AGENTS

NITROGLYCERIN
- Nitroglycerin is a direct vasodilator that induces venodilation at low doses (<100 mg/min) and arteriolar vasodilation at high doses (<200 mg/min). Coronary artery dilation occurs throughout the dosage range.
- Nitroglycerin is approved for the prophylaxis, treatment, and management of angina pectoris. Intravenous nitroglycerin is used to control hypertension associated with surgery and is also used in congestive heart failure associated with acute myocardial infarction.
- Nitroglycerin can be administered sublingually, lingually, intrabuccally, orally, topically, or by IV infusion.
- Sublingual tablets or sprays can be given every 5 min.
- Topical paste can be applied to the chest 1 to 2 in., as needed, every 4 to 8 h.
- Start IV infusion at 5 to 10 mg/min and titrate in increments of 5 to 10 mg/min to desired response. Most doses range between 50 and 200 mg/min.

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3. Strasberg B, Sagie A. Rechavia E, et al: Deleterious effects of intravenous verapamil in Wolff-Parkinson-White patients and atrial fibrillation. *Cardiovasc Drugs Ther* 2(6):801, 1989.

For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 24, ''Disturbances of Cardiac Rhythm and Conduction,'' by Edmund Bolton, and Chap. 25, ''Pharmacology of Antidysrhythmic and Vasoactive Medications,'' by Teresa M. Carlin.

5 RESUSCITATION OF CHILDREN AND NEONATES

David M. Cline

EPIDEMIOLOGY

- Children have very poor survival rates from cardiac arrest, because it is often associated with prolonged hypoxia or shock.[1, 2]
- Following a cardiac arrest, the survival rate without devastating neurologic sequelae in children is only 2 percent.[3]

PATHOPHYSIOLOGY

- Respiratory and cardiac arrest in children is most commonly due to primary respiratory conditions and shock.[4]

TABLE 5-1 Length-Based Equipment Chart

	PATIENT LENGTH, CM						
ITEM	54–70	70–85	85–95	95–107	107–124	124–138	138–155
ET tube size, mm	3.5	4.0	4.5	5.0	5.5	6.0	6.5
Lip-tip length, mm	10.5	12.0	13.5	15.0	16.5	18.0	19.5
Laryngoscope	1 straight	1 straight	2 straight	2 straight or curved	2 straight or curved	2–3 straight or curved	3 straight or curved
Suction catheter	8F	8–10F	10F	10F	10F	10F	12F
Stylet	6F	6F	6F	6F	14F	14F	14F
Oral airway	Infant/small child	Small child	Child	Child	Child/small adult	Child/adult	Medium adult
Bag-valve-mask	Infant	Child	Child	Child	Child	Child/adult	Adult
Oxygen mask	Newborn	Pediatric	Pediatric	Pediatric	Pediatric	Adult	Adult
Vascular access catheter/butterfly	22–24/23–25, intraosseous	20–22/24–25, intraosseous	18–22/21–23, intraosseous	18–22/21–23, intraosseous	18–20/21–23	18–20/21–22	16–20/18–21
Nasogastric tube	5–8F	8–10F	10F	10–12F	12–14F	14–18F	18F
Urinary catheter	5–8F	8–10F	10F	10–12F	10–12F	12F	12F
Chest tube	12–16F	16–20F	20–24F	20–24F	24–32F	28–32F	32–40F
Blood pressure cuff	Newborn/infant	Infant/child	Child	Child	Child	Child/adult	Adult

NOTE: Directions for use: (1) Measure patient length with centimeter tape; (2) Using measured length in centimeters, access appropriate equipment column.
SOURCE: Adapted from Luten RD, Wears RL, Broselow J, et al: Length-based endotracheal tube and emergency equipment in pediatrics. *Ann Emerg Med* 21:900, 1992.

- Because of age and size differences in children, drug dosages, compression and respiratory rates, and equipment sizes vary considerably (see Table 5-1).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

SECURING THE AIRWAY

- The airway in infants and children is smaller, variable in size, and more anterior than it is in the adult.
- Mild extension of the head (sniffing position) opens the airway. Chin lift or jaw thrust maneuvers may relieve obstruction of the airway related to the tongue.
- Oral airways are not commonly used in pediatrics but may be useful in patients whose airway cannot be maintained manually. Oral airways are inserted with a tongue blade as in adults.
- A bag-valve-mask system is commonly used for ventilation. Minimum volume for ventilation bags for infants and children is 450 mL. The tidal volume necessary to ventilate children is 10 to 15 mL/kg. In emergency situations, however, observation of chest rise and auscultation of breath sounds will ensure adequate ventilation.
- Endotracheal intubation is usually performed using a Miller (straight) blade with a properly sized tube.
- Resuscitation measuring tapes have been found to be more accurate than age-based formulas, which in turn are superior to the diameter of the fifth digit.[5,6]
- In the absence of resuscitation measuring tubes, the internal diameter of the tube should be the same size as the end of the patient's little finger. The formula 16 + age in years divided by 4 gives approximate tube size.
- Uncuffed tubes are used in children up to 7 to 8 years.
- Confirmation of endotracheal intubation is similar to that in adults: adequate chest rise, symmetric breath sounds, capnographic or capnometric reading,[7] improved oxygenation, and clinical improvement.
- The laryngeal mask airway (LMA) has been widely used in the pediatric population.[8] It has been found to be extremely useful in the management of difficult airways.
- Transtracheal jet ventilation allows ventilation

and oxygenation through a catheter. Ventilation is provided with short, intermittent bursts of oxygen. This requires high-pressure (50 psi) oxygen-delivery systems. The system of choice is a jet injector regulated by a flow meter attached to a wall or tank unit. A 1-s jet of oxygen followed by a 4-s expiratory phase achieves satisfactory ventilation.[9]

Rapid Sequence Induction

- Rapid sequence induction (RSI) is the administration of an intravenous anesthetic with a neuromuscular blocking agent to facilitate endotracheal intubation.[10]
- The patient should be preoxygenated with 100% oxygen.
- Lidocaine (1 mg/kg intravenously, IV) may be used in head trauma patients to prevent increased intracranial pressure (ICP).[11]
- Atropine (0.02 mg/kg, minimum dose 0.1 mg) may be used to prevent reflex bradycardia in children under 5 years old.
- Cricoid pressure should be applied before paralysis and maintained until intubation is accomplished.
- Induction of anesthesia is accomplished using one of several drug choices depending on the clinical situation and the experience of the physician.
- Sodium thiopental (3 to 5 mg/kg) is most commonly used. Advantages of thiopental include rapid onset of action, safe for use with increased ICP, and low cost. Disadvantages include histamine release, possible hypotension, and tissue necrosis if extravasated.
- Propofol (2 to 3 mg/kg) is a rapid-acting induction agent, which is safe for increased ICP. Disadvantages include pain on injection and cost.
- Ketamine (2 to 3 mg/kg) is a dissociative anesthetic, which increases heart rate and has bronchodilating effects. It has been used in trauma with hypotension and in patients with asthma. Disadvantages include increased airway secretions, increased ICP, emergence reactions, and possible laryngospasm.
- Midazolam (0.2 to 0.3 mg/kg) is a benzodiazepine which can be used for induction. One of the advantages is reversibility. Disadvantages include slower onset of action and possible cardiorespiratory depression.
- Neuromuscular blockade is accomplished by using succinylcholine, vecuronium, or rocuronium.
- Succinylcholine is a depolarizing blocking agent, which has a rapid onset (45 s) but short duration of action (3 to 5 min). Although producing reliable paralysis, it has several disadvantages. Hyperkalemia may occur when succinylcholine is used in patients with burns over 1 day old, spinal cord injuries, chronic immobilization, crush injuries with significant muscle injury, or conditions predisposing to hyperkalemia. It has been associated with hyperkalemic arrest in children with underlying but undiagnosed myopathies. Use of succinylcholine may also cause malignant hyperthermia in susceptible individuals, elevations in ICP and intraocular pressure, and bradycardia, particularly in infants (premedicate with atropine in children under 5 years to prevent this effect). Muscle fasciculations can be prevented by a defasciculating dose of a nondepolarizing agent before succinylcholine is given. The short duration of action of succinylcholine may be a particular advantage when a difficult airway is anticipated or when ongoing neurologic assessment is required.
- A fast-acting nondepolarizing agent, such as vecuronium or rocuronium, may be chosen with the knowledge that the onset of action is slower and duration of action is much longer than it is with succinylcholine.
- Rocuronium (0.9 to 1.2 mg/kg) is the fastest acting nondepolarizing agent with onset in 55 to 75 s. The duration of action is 30 to 60 min.
- Vecuronium (0.2 to 0.3 mg/kg) has an onset of 60 to 90 s and lasts 90 to 120 min.

Vascular Access

- Vascular access is done in the most rapid, least invasive manner possible; peripheral veins (arm, hand, or scalp) are tried first.
- Intraosseous access is a quick, safe route for resuscitation medications and may be tried next in the critically ill infant.
- Percutaneous access of the femoral vein or access of the saphenous vein through cutdown can also be used, but is more time consuming.
- Technique for insertion of the intraosseous line is as follows: the bone most commonly used is the proximal tibia. The anterior tibial tuberosity is palpated with the index finger, and the medial aspect of the tibia is grasped with the thumb. An imaginary line is drawn between the two, and the needle is inserted 1 cm distal to the midpoint of this line. A bone marrow needle is most commonly used; if a bone marrow needle is not available, an 18-gauge spinal needle can be used but is prone to bending. Using sterile technique, the needle is inserted in a slightly caudal direction until the needle punctures the cortex. The stylet is removed and marrow is aspirated to confirm placement. Fluids or drugs (including glucose, epinephrine, dopamine, anticonvulsants, and antibiotics) may

then be administered as they are through a normal IV line.

Fluids

- In shock, intravenous isotonic fluid (i.e., normal saline) boluses of 20 mL/kg should be given as rapidly as possible and should be repeated, depending on the clinical response.[4] (See Chap. 82 for more details.)
- If hypovolemia has been corrected and shock or hypotension still persists, a pressor agent should be considered.

Drugs

- The indications for resuscitation drugs are the same for children as they are in adults; however, epinephrine is considered the first-line drug prior to atropine for the treatment of bradycardia.
- Drug dose calculations are a problem particular to pediatrics (Table 5-1). Using a drug dosage chart or Broselow tape will reduce dosage errors. The Broselow tape is a length-based system for estimating the weight of children in emergency situations.[5] The tape has drug dosages, equipment sizes, and fluid volumes displayed according to patient size.
- The rule of sixes may be used to quickly calculate continuous drug infusions (such as dopamine, dobutamine, etc). The calculation is 6 mg $\times$ weight in kg, fill to 100 mL with D_5W. The infusion rate in milliliters per hour will equal the micrograms per kilogram per minute rate (i.e., an infusion running at 1 mL/h = 1 μg/kg/min or 5 mL/h = 5 μg/kg/min).
- Epinephrine is the only drug proved effective in cardiac arrest. It is indicated in pulseless arrest and in slow (bradycardia) rates that are hypoxia induced and unresponsive to oxygenation and ventilation.
- If the initial dose of epinephrine (0.01 mg/kg of a 1:10,000 concentration) is not effective in pulseless arrest, high-dose epinephrine is recommended (0.1 to 0.2 mg/kg of a 1:1000 concentration) subsequently.[4]
- Primary cardiac causes of bradycardia are rare and may be treated with atropine (0.02 mg/kg, minimum dose 0.1 mg) after adequate oxygenation and ventilation are ensured.
- The dose of endotracheal epinephrine for symptomatic bradycardia or pulseless cardiac arrest is 0.1 mg/kg, 1:1000 concentration every 3 to 5 min.
- Although the ideal endotracheal doses for other drugs have never been studied in children, current recommendations support the use of two to three times the IV dose.[4]
- Sodium bicarbonate is no longer recommended as a first-line resuscitation drug. It is recommended only after epinephrine administration has been ineffective or as guided by arterial blood gases.
- Calcium is not recommended in routine resuscitation, but may be useful in hyperkalemia, hypocalcemia, and calcium channel blocker overdose.

Dysrhythmias

- Dysrhythmias in infants and children are most often the result of respiratory insufficiency or arrest, not primary cardiac causes as in adults. Careful attention to oxygenation and ventilation are, therefore, cornerstones of dysrhythmia management in pediatrics.
- Ventricular fibrillation as a cause of cardiac arrest is rare in children and even more rare in infants.[12]
- The most common rhythm seen in pediatric arrest situations is bradycardia leading to asystole. Oxygenation and ventilation are often sufficient in this situation; epinephrine followed by atropine may be useful if unresponsive to ventilation.
- Chest compressions should be started when a child with bradycardia $<$60 beats per minute fails to respond to oxygenation and ventilation.
- Outside of the arrest situation, the most common dysrhythmia is supraventricular tachycardia (SVT). It presents with a narrow complex tachycardia with rates between 250 and 350 beats per minute. Adenosine (0.1 mg/kg), given through a well-functioning IV as close to the central circulation as possible followed by brisk saline flush, is the recommended treatment for stable SVT in children. Treatment of the unstable patient with SVT is synchronized cardioversion ($\frac{1}{4}$ to $\frac{1}{2}$ J/kg).
- It is sometimes difficult to distinguish between a fast sinus tachycardia and SVT. Small infants may have sinus tachycardia with rates above 200 beats per minute. Patients with sinus tachycardia may have a history of dehydration or shock; examination evidence of dehydration, fever, or pallor; and have a normally sized heart on chest x-ray.
- Infants with SVT often have a nonspecific history, an exam with rales, an enlarged liver, and may have an enlarged heart on x-ray.
- Transcutaneous pacing has not been associated with greatly improved survival rates, but it can be life saving if applied quickly in a child with sudden asystole or bradycardia. Adult patches should be used in children who weigh over 15 kg.[13]

Defibrillation and Cardioversion

- Ventricular fibrillation is rare in children but may be treated with defibrillation at 2 J/kg.[4] If this

attempt is unsuccessful; the energy is doubled to 4 J/kg.
- If two attempts at defibrillation at 4 J/kg are unsuccessful, epinephrine should be given and oxygenation and acid-base status should be reassessed.
- Cardioversion is used to treat unstable tachydysrhythmias at a dose of $\frac{1}{4}$ to $\frac{1}{2}$ J/kg.
- The largest paddles should be used, which still allow contact of the entire paddle with the chest wall. Electrode cream or paste is used to prevent burns. One paddle is placed on the right of the sternum at the second intercostal space and the other is placed at the left midclavicular line at the level of the xiphoid.

NEONATAL RESUSCITATION

- Most newborns do not require specific resuscitation after delivery, but about 6 percent of newborns require some form of life support in the delivery room.
- The first step in neonatal resuscitation is to maintain body temperature. The infant should be dried and placed in a radiant warmer.
- The airway should be cleared by suctioning the nose and mouth with a bulb syringe or a DeLee trap.
- Next, a 5- to 10-s examination should assess heart rate, respiratory effort, color, and activity. If the infant is apneic or the heart rate is slow (<100 beats per minute), positive pressure ventilation with bag-valve-mask and 100% oxygen should be administered. The rate should be 40 breaths per minute. In mildly depressed infants, a prompt improvement in heart rate and respiratory effort usually occur.
- If no improvement is noted after 30 s and the condition deteriorates, endotracheal intubation should be performed.
- If the heart rate is still below 50 beats per minute after intubation and assisted ventilation, cardiac massage should be started at 120 compressions per minute. Compressions and ventilations should be in a 3:1 ratio.
- If there is no improvement in heart rate following these efforts, drug therapy may be used. Most neonates respond to appropriate airway management, therefore, drug therapy is rarely needed.
- Vascular access may be obtained peripherally or via the umbilical vein. The most expedient procedure in the neonate is to place an umbilical catheter in the umbilical vein and advance to 10 to 12 cm.
- Medications useful in neonatal resuscitation include epinephrine, naloxone, isoproterenol, and bicarbonate.
- Epinephrine (0.01 mg/kg of 1:10,000 solution) may be used if the heart rate is still below 100 beats per minute after adequate ventilation.
- Naloxone (0.1 mg/kg IV) may be useful to reverse narcotic respiratory depression.
- Isoproterenol (0.05 to 0.1 μg/min) may be infused if epinephrine fails to raise the heart rate.
- Sodium bicarbonate (2 to 3 meq) may be given if there is a significant metabolic acidosis; this therapy should be guided by blood gases.

PREVENTION OF MECONIUM ASPIRATION

- Aspiration of meconium-stained amniotic fluid is associated with a high morbidity and mortality. With proper perinatal management, it is almost entirely preventable.
- If meconium is noted at the time of delivery, the nose, mouth, and pharynx of the infant should be suctioned with a DeLee trap prior to delivery of the infant's shoulders.
- Repeat suctioning of the airway should be performed with the infant under the radiant warmer prior to drying and stimulating the infant. This may be accomplished by visualizing the trachea with a laryngoscope and suctioning via an endotracheal tube. After suctioning, the infant should be dried and stimulated.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 9, ''Neonatal Resuscitation and Emergencies,'' by Eugene E. Cepeda and Seetha Shankaran; Chap. 10, ''Pediatric Cardiopulmonary Resuscitation,'' by William E. Hauda II; and Chap. 11, ''Pediatric Airway Management,'' by Marcie Rubin and Nicholas Sadovnikoff.

6 FLUIDS, ELECTROLYTES, AND ACID-BASE DISORDERS

David M. Cline

FLUIDS

- When altered, fluids and electrolytes should be corrected in the following order: (1) volume; (2) pH; (3) potassium, calcium, magnesium; and (4) sodium and chloride. Reestablishment of tissue perfusion often reequilibrates the fluid-electrolyte and acid-base balance.
- Because the osmolarity of normal saline (NS) matches that of the serum, it is an excellent fluid for volume replacement.
- Hypotonic fluids such as D_5W should never be used to replace volume.
- Lactated Ringer's solution is commonly used for surgical or trauma patients; however, only NS can be given in the same line with blood components.
- D_5.45 NS, with or without potassium, is given as a maintenance fluid.
- The more concentrated dextrose solutions, $D_{10}W$ and $D_{20}W$, are used for patients with compromised ability to mobilize glucose stores, such as those with hepatic failure, or as part of total parental nutrition (TPN) solutions.

CLINICAL ASSESSMENT OF VOLUME STATUS

- Volume loss and dehydration can be inferred by the patient history. Historical features include vomiting, diarrhea, fever, adverse working conditions, decreased fluid intake, chronic disease, altered level of consciousness, and reduced urine output.
- Tachycardia and hypotension are most commonly late signs of dehydration.
- On physical exam, you may find dry mucosa, shrunken tongue (excellent indicator), and decreased skin turgor. In infants and children, sunken fontanelles, decreased capillary refill, lack of tears, and decreased wet diapers are typical signs and symptoms of dehydration.
- Lethargy and coma are more ominous signs and may indicate a significant comorbid condition.
- Laboratory values are not reliable indicators of fluid status. Plasma and urine osmolarity are perhaps the most reliable measures of dehydration. Blood urea nitrogen (BUN), creatinine, hematocrit, and other chemistries are insensitive.
- Volume overload is a purely clinical diagnosis and presents with edema (central or peripheral), respiratory distress (pulmonary edema), and jugular venous distention (in congestive heart failure).
- The significant risk factors for volume overload are renal, cardiovascular, and liver disease. Blood pressure (BP) does not necessarily correlate with volume status alone; patients with volume overload can present with hypotension or hypertension.

MAINTENANCE FLUIDS

- Adults: D_5.45 NS at 75 to 125 mL/h + 20 meq/L of potassium chloride for an average adult (approximately 70 kg).

- Children: D_5.45 NS or D_{10}.45 NS, 100 mL/kg/d for the first 10 kg (of body weight) of above solution, 50 mL/kg/d for second 10 kg, and 20 mL/kg/d for every kilogram thereafter. (See Chap. 82 for further discussion of pediatric fluid management.)

ELECTROLYTE DISORDERS

- Correcting a single abnormality may not be the only intervention needed, as most electrolytes exist in equilibrium with others.
- Laboratory errors are common. Results should be double-checked when the clinical picture and the laboratory data conflict.
- Abnormalities should be corrected at the same rate they developed; however, slower correction is usually safe unless the condition warrants rapid and/or early intervention (i.e., hypoglycemia, hyperkalemia).
- Evaluation of electrolyte disorders frequently requires a comparison of the measured and calculated osmolarity (number of particles per liter of solution). To calculate osmolarity, measured serum values in meq/L are used:

$$\text{Osmolarity in mosm/L} = 2[Na^+] + \frac{\text{glucose}}{18} + \frac{\text{BUN}}{2.8} + \frac{\text{ETOH}}{4.6}$$

HYPONATREMIA ([Na^+] <135 meq/L)

CLINICAL FINDINGS

- The clinical manifestations of hyponatremia occur when the [Na^+] drops below 120 meq/L; they include abdominal pain, headache, agitation, hallucinations, cramps, confusion, lethargy, and seizures.

DIAGNOSIS AND DIFFERENTIAL

- The volume status is evaluated first, then the measured and calculated osmolarity. True hyponatremia presents with a reduced osmolarity.
- Factitious hyponatremia presents with a normal to high osmolarity. The most common cause is dilutional. It may be brought on by trauma, sepsis, cardiac failure, cirrhosis, or renal failure. Hyponatremia may also be factitious (false elevation in the measured sodium) due to hyperglycemia, elevated protein, or hyperlipidemia.
- Extracellular fluid (ECF) or volume status and urine sodium level can classify true hyponatremia (low osmolarity). The syndrome of inappropriate antidiuretic hormone (SIADH) is a diagnosis made by exclusion. Causes of hyponatremia are listed in Table 6-1.

TABLE 6-1 Causes of Hyponatremia

Hypotonic (true) hyponatremia (Posmol <275)
Hypovolemic hyponatremia
Extrarenal losses (urinary [Na^+] <20 meq/L)
Sweating, vomiting, diarrhea
Third-space sequestration (burns, peritonitis, pancreatitis)
Renal losses (urinary [Na^+] >20 meq/L)
Loop or osmotic diuretics
Aldosterone deficiency (Addison's disease)
Ketonuria
Salt-losing nephropathies; renal tubular acidosis
Osmotic diuresis (mannitol, hyperglycemia, hyperuricemia)
Euvolemic hyponatremia (urinary [Na^+] >20 meq/L)
Inappropriate ADH secretion (CNS, lung, or carcinoma disease)
Physical and emotional stress or pain
Myxedema, Addison's disease, Sheehan's syndrome
Drugs, water intoxication
Hypervolemic hyponatremia
Urinary [Na^+] >20 meq/L
Renal failure
Urinary [Na^+] <20 meq/L
Cirrhosis
Cardiac failure
Renal failure
Isotonic (pseudo) hyponatremia (Posmol 275–295)
Hyperproteinemia, hyperlipidemia, hyperglycemia
Hypertonic hyponatremia (Posmol >295)
Hyperglycemia, mannitol excess and glycerol use

ABBREVIATIONS: ADH = antidiuretic hormone; CNS = central nervous system.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The volume or perfusion deficit, if any, is corrected first, using normal saline.
- In stable normotensive patients, fluids are restricted (500 to 1500 mL of water daily).
- In severe hyponatremia ([Na^+] <120 meq/L) with central nervous system (CNS) changes, hypertonic saline, 3% NS (513 meq/L), is given at 25 to 100 mL/h. Concomitant use of furosemide in small doses of 20 to 40 mg has been shown to decrease the incidence of central pontine myelinolysis (CPM).[1]

- The sodium deficit can be calculated as follows:

  wt in kg × 0.6 × (140 − measured [Na^+])
  = sodium deficit in meq

- Complications of rapid correction include congestive heart failure (CHF) and CPM, which can cause alterations in consciousness, dysphagia, dysarthria, and paresis.

HYPERNATREMIA ([Na^+] >150 meq/L)

CLINICAL FEATURES

- The symptoms of hypernatremia usually begin when the osmolarity is greater than 350. Irritability and ataxia occur at osmolarities above 375. Lethargy, coma, and seizures present with osmolarities above 400.
- Brain hemorrhage can be seen in neonates after rapid infusion of $NaHCO_3$.
- An osmolarity increase of 2 percent sets off thirst to prevent hypernatremia. Morbidity and mortality are highest in infants and the elderly, who may be unable to respond to increased thirst.

DIAGNOSIS AND DIFFERENTIAL

- The most frequent cause of hypernatremia is a decrease in total body water due to decreased intake or excessive loss.
- Common causes are diarrhea, vomiting, hyperpyrexia, and excessive sweating.
- An important etiology of hypernatremia is *diabetes insipidus* (DI), which results from loss of hypotonic urine. It may be central (no ADH secreted) or nephrogenic (unresponsive to ADH).
- The causes of hypernatremia are listed in Table 6-2.

TABLE 6-2 Causes of Hypernatremia

Loss of water
Reduced water intake
Defective thirst
Unconsciousness
Inability to drink water
Lack of access to water
Water loss in excess of sodium
Vomiting, diarrhea
Sweating, fever
Dialysis
Drugs, hyperventilation
Diabetes insipidus, osmotic diuresis
Thyrotoxicosis
Severe burns
Gain of sodium
Increased intake
Hypertonic saline ingestion or infusion
Sodium bicarbonate administration
Renal salt retention (usually because of poor perfusion)

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treat any perfusion deficits with NS or lactated Ringer's (LR). Then, switch to 0.5 NS after a urine output of 0.5 mL/kg/h is reached. Avoid lowering the [Na^+] more than 10 meq/L/d. Monitor central venous pressure and pulmonary capillary wedge pressure.
- Use the formula below to calculate the total body water deficit. As a rule, each liter of water deficit causes the [Na^+] to increase 3 to 5 meq/L.

  Water deficit in liters
  = TBW (1 − measured [Na^+]/desired [Na^+])

- If no urine output is observed after NS/LR rehydration, rapidly switch to 0.5 NS: unload the body of the extra sodium by using a diuretic (i.e., furosemide, 20 to 40 mg IV).
- Central DI is treated using desmopressin (DDAVP).
- In children with a serum sodium level greater than 180 meq/L, consider peritoneal dialysis, using high glucose–low [Na^+] dialysate, which may be lifesaving.

HYPOKALEMIA ([K^+] <3.5 meq/L)

CLINICAL FEATURES

- The signs and symptoms of hypokalemia usually occur at levels below 2.5 meq/L and affect the following body systems: the central nervous system (CNS) (weakness, cramps, hyporeflexia), gastrointestinal (GI) system (ileus), cardiovascular system (dysrhythmias, worsening of digoxin toxicity, hypotension or hypertension, U waves, ST-segment depression, and prolonged QT interval), and renal system (metabolic alkalosis and worsening hepatic encephalopathy); last, glucose intolerance can also develop.

TABLE 6-3 Causes of Hypokalemia

Shift into the cell
Raising the pH of blood, β adrenergics
Administration of insulin and glucose
Reduced intake
Increased loss
Renal loss
Primary hyperaldosteronism, osmotic diuresis
Secondary hyperaldosteronism associated with diuretics, malignant hypertension, Bartter's syndrome, and renal artery stenosis
Miscellaneous
Licorice use
Use of chewing tobacco
Hypercalcemia
Liddle syndrome
Magnesium deficiency
Renal tubular acidosis
Acute myelocytic and monocytic leukemia
Drugs and toxins (PCN, lithium, L-dopa, theophylline)
GI loss (vomiting, diarrhea, fistulas)

ABBREVIATIONS: PCN = penicillin; GI = gastrointestinal.

DIAGNOSIS AND DIFFERENTIAL

- The most common cause is the use of loop diuretics. Table 6-3 lists the causes.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Replacement of $[K^+]$ at 20 meq/h will raise the $[K^+]$ by 0.25 meq/L.
- Administer 10 to 15 meq/h of potassium chloride (KCl) in 50 to 100 mL of dextrose in water (D_5W), piggyback into saline over 3 to 4 h.[2] In general, up to 10 meq/h of KCl can be given through a peripheral IV and up to 20 meq/h can be given through a central line. Add no more than 40 meq of KCl in 1 L of IV fluids. Patients should be monitored continuously for dysrhythmias.
- Oral replacement (in the awake asymptomatic patient) is rapid and safer than IV therapy. Use 20 to 40 meq/L of KCl or similar agent.

HYPERKALEMIA ($[K^+]$ >5.5 meq/L)

CLINICAL FEATURES

- The most concerning and serious manifestations of hyperkalemia are the cardiac effects. At levels of 6.5 to 7.5 meq/L, the electrocardiogram (ECG) shows peaked T waves (precordial leads), prolonged PR, and short QT intervals.
- At levels of 7.5 to 8.0 meq/L, the QRS widens and the P wave flattens.
- At levels above 8 meq/L, a sine-wave pattern, ventricular fibrillation, and heart blocks occur.
- Neuromuscular symptoms include weakness and paralysis. GI symptoms include vomiting, colic, and diarrhea.

DIAGNOSIS AND DIFFERENTIAL

- Beware of pseudohyperkalemia, which is caused by hemolysis after blood draws.
- Renal failure with oliguria is the most common cause of true hyperkalemia.
- Appropriate tests for management include an ECG, electrolytes, calcium, magnesium, arterial blood gases (ABG) (check for acidosis), urine analysis (UA), and a digoxin level in appropriate patients.
- Causes of hyperkalemia are listed in Table 6-4.

TABLE 6-4 Causes of Hyperkalemia

Factitious
Laboratory error
Pseudohyperkalemia: hemolysis, thrombocytosis, and leukocytosis
Metabolic acidemia (acute)
Increased intake into the plasma
Exogenous: diet, salt substitutes, low-sodium diet, and medications
Endogenous: hemolysis, GI bleeding, catabolic states, crush injury
Inadequate distal delivery of sodium and decreased distal tubular flow
Oliguric renal failure
Impaired renin-aldosterone axis
Addison's disease
Primary hypoaldosteronism
Other (heparin, β blockers, prostaglandin inhibitors, captopril)
Primary renal tubular potassium secretory defect
Sickle cell disease
Systemic lupus erythematosus
Postrenal transplantation
Obstructive uropathy
Inhibition of renal tubular secretion of potassium
Spironolactone
Digitalis
Abnormal potassium distribution
Insulin deficiency
Hypertonicity (hyperglycemia)
β-adrenergic blockers
Exercise
Succinylcholine
Digitalis

ABBREVIATION: GI = gastrointestinal.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Symptomatic patients are treated in a stepwise approach: stabilize the cardiac membrane with $CaCl_2$, and then shift the $[K^+]$ into the cell using glucose and insulin and/or bicarbonate. Finally, excrete the potassium using sodium polystyrene sulfonate (Kayexalate), diuretics, and dialysis in severe cases.
- For levels over 7.0 meq/L or if there are any ECG changes, give IV calcium chloride, 5 mL of a 10% solution; use caution in a digoxin-toxic patient (risk of dysrhythmias). The presence of digoxin toxicity with hyperkalemia is an indication for digoxin immune Fab (Digibind) therapy. See Chap. 106.
- For levels above 5.5 meq/L (especially in acidotic patients), give 1 to 2 ampules of sodium bicarbonate.
- Give 1 ampule of $D_{50}W$, with 10 U of regular insulin IV push (5 U in dialysis patients).
- Maintain diuresis with furosemide, 20 to 40 mg IV push.
- Kayexalate (PO or PR) 1 g binds 1 meq of $[K^+]$ over 10 min. Administer 15 to 25 g of Kayexalate PO with 50 mL of 20% sorbitol (sorbitol is used because Kayexalate is constipating). Per rectum, give 20 g in 200 mL 20% sorbitol over 30 min. Kayexalate can exacerbate CHF.
- In patients with acute renal failure, consult a nephrologist for emergent dialysis.
- Albuterol [by nebulization, 0.5 mL of a 5% solution (2.5 mg)], may also be used to lower $[K^+]$ (transient effect).

HYPOCALCEMIA ($[Ca^{2+}] < 8.5$ OR IONIZED LEVEL < 2.0)

CLINICAL FEATURES

- The signs and symptoms of hypocalcemia are usually seen with ionized $[Ca^{2+}]$ levels below 1.5. Clinically, patients have paresthesias, increased deep tendon reflexes (DTR), cramps, weakness, confusion, and seizures.
- Patients may also demonstrate Chvostek's sign (twitch of the corner of mouth on tapping with finger over cranial nerve VII at zygoma) or Trousseau's sign (more reliable: carpal spasm when the blood pressure cuff is left inflated at a pressure above the systolic BP for more than 3 min).
- If the patient is alkalotic, ionized calcium (physiologically active) may be very low, even with normal total calcium.
- In refractory CHF, $[Ca^{2+}]$ can be low.

DIAGNOSIS AND DIFFERENTIAL

- Common causes: shock, sepsis, renal failure, pancreatitis, drugs (cimetidine mostly), hypoparathyroidism, phosphate overload, vitamin D deficiency, fat embolism, strychnine poisoning, hypomagnesemia, and tetanus toxin.
- The ECG often shows prolonged QT.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- If the patient is asymptomatic, use oral calcium gluconate tablets, 1 to 4 g/d divided q6h, with or without vitamin D (calcitriol, 0.2 μg bid). Milk is not a good substitute (low $[Ca^{2+}]$).
- In more urgent situations with symptomatic patients, calcium gluconate or calcium chloride, 10 mL of a 10% solution, can be given over 10 min, slow IV.

HYPERCALCEMIA ($[Ca^{2+}] > 10.5$ OR IONIZED $[Ca^{2+}] > 2.7$ meq/L)

- Several factors affect the serum calcium level: parathyroid hormone (PTH) increases calcium and decreases phosphate; calcitonin and vitamin D metabolites decrease calcium.
- Decreased $[H^+]$ causes a decrease in ionized $[Ca^{2+}]$. Ionized $[Ca^{2+}]$ is the physiologically active form. Each rise in pH of 0.1 lowers $[Ca^{2+}]$ by 3 to 8 percent.
- A decrease in albumin causes a decrease in $[Ca^{2+}]$, but not in the ionized portion.
- Most cases of hypercalcemia are due to hyperparathyroidism or malignancies. One-third of the patients develop hypokalemia.

CLINICAL FEATURES

- Clinical signs and symptoms develop at levels above 12 mg/dL.
- A mnemonic to aid recall of common hypercalcemia symptoms is *stones* (renal calculi), *bones* (bone destruction secondary to malignancy), *psychic moans* (lethargy, weakness, fatigue, confu-

sion), and *abdominal groans* (abdominal pain, constipation, polyuria, polydipsia).

DIAGNOSIS AND DIFFERENTIAL

- On the ECG, you may see depressed ST segments, widened T waves, shortened QT intervals, and heart blocks. Levels above 20 meq/L can cause cardiac arrest.
- A mnemonic to aid recall of the common causes is *Pam P. Schmidt: p*arathyroid hormone, *A*ddison's disease, *m*ultiple myeloma, *P*aget's disease, *s*arcoidosis, *c*ancer, *h*yperthyroidism, *m*ilk-alkali syndrome, *i*mmobilization, excess vitamin *D,* and *t*hiazides.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergency treatment is important in the following conditions: a calcium level above 12 mg/dL, a symptomatic patient, a patient who cannot tolerate PO fluids, or a patient with abnormal renal function.
- Correct dehydration with normal saline, 5 to 10 L, may be required. Consider invasive monitoring.
- Administer furosemide, 40 mg, but do not exacerbate dehydration if present. Correct the concurrent hypokalemia or hypomagnesemia. Do not use thiazide diuretics (they worsen hypercalcemia).
- If above treatments are not effective, administer calcitonin 0.5 to 4 IU/kg IV over 24 h or IM divided every 6 h, along with hydrocortisone 25 to 100 mg IV every 6 h.

HYPOMAGNESEMIA

CLINICAL FINDINGS

- $[Mg^{2+}]$, $[K^{+}]$, and $[PO_4^{-}]$ move together intra- and extracellularly. Hypomagnesemia can present with CNS symptoms (depression, vertigo, ataxia, seizures, increased DTR, tetany) or cardiac symptoms (arrhythmias, prolonged QT and PR, worsening of digitalis effects).
- Also seen are anemia, hypotension, hypothermia, and dysphagia.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis should not be based on $[Mg^{2+}]$ levels, since total depletion can occur before any significant laboratory changes appear. It must therefore be suspected clinically.
- In the United States, the most common cause is alcoholism, followed by poor nutrition, cirrhosis, pancreatitis, correction of diabetic ketoacidosis (DKA), or excessive gastrointestinal losses.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- First correct volume deficit and any decreased potassium, calcium, or phosphate.
- If the patient is an alcoholic in delirium tremens (DTs) or pending DTs, administer 2 g magnesium sulfate in the first hour, then 6 g (in the first 24 h). Check DTR every 15 min. DTRs disappear when the serum magnesium level rises above 3.5 meq/L, at which time the magnesium infusion should be stopped.

HYPERMAGNESEMIA

CLINICAL FINDINGS

- Signs and symptoms manifest progressively; DTRs disappear with a serum magnesium level above 3.5 meq/L, muscle weakness at a level above 4 meq/L, hypotension at a level above 5 meq/L, and respiratory paralysis at a level above 8 meq/L.

DIAGNOSIS AND DIFFERENTIAL

- Hypermagnesemia is rare. Common causes are renal failure with concomitant ingestion of magnesium-containing preparations (antacids) and lithium ingestion. Serum levels are diagnostic. Suspect coexisting increased potassium and phosphate.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Rehydrate with normal saline and furosemide 20 to 40 mg IV (in absence of renal failure).
- Correct acidosis with ventilation and sodium bicarbonate 50 to 100 meq if needed.
- In symptomatic patients, 5 mL (10% solution) of CaCl IV antagonizes the magnesium effects.

ACID-BASE PROBLEMS

- Several conditions should alert the clinician to possible acid-base disorders: history of renal, endocrine, or psychiatric disorders (drug ingestion) or signs of acute disease: tachypnea, cyanosis, Kussmaul respiration, respiratory failure, shock, changes in mental status, vomiting, diarrhea, or other acute fluid losses.
- Acidosis is due to gain of acid or loss of alkali; causes may be metabolic (fall in serum $[HCO_3^-]$) or respiratory (rise in P_{CO_2}).
- Alkalosis is due to loss of acid or addition of base and is either metabolic (rise in serum $[HCO_3^-]$) or respiratory (fall in P_{CO_2}).
- The lungs and kidneys primarily maintain the acid-base balance.
- Metabolic disorders prompt an immediate compensatory change in ventilation, either venting CO_2 in cases of metabolic acidosis or retaining it in cases of metabolic alkalosis.
- The kidneys' response to metabolic disorders is to excrete hydrogen ion (with chloride) and recuperate $[HCO_3^-]$, a process that requires hours to days.
- The compensatory mechanisms of the lungs and kidney will return the pH toward but not to normal.
- In a mixed disorder, the pH, P_{CO_2}, and $[HCO_3^-]$ may be normal and the only clue to a metabolic acidosis is a widened anion gap.
- The most helpful formula to determine the expected fall in P_{CO_2} in response to a fall in bicarbonate is the following: P_{CO_2} falls by 1 mmHg for every 1 meq/dL fall in bicarbonate. This relationship holds true provided that the bicarbonate level is greater than 8 meq/dL.
- The most helpful formula to calculate the expected change in pH when P_{CO_2} changes is as follows: the change in $[H^+] = 0.8$ (change in P_{CO_2}). Thus, an increment of 10 mmHg in P_{CO_2} produces an 8-mmol increase in hydrogen ion concentration.
- Use as normals: pH = 7.4, HCO_3 = 24 mm/L, P_{CO_2} = 40 mmHg.
- If the pH indicates acidosis, the primary (or predominant) mechanism can be ascertained by examining the $[HCO_3^-]$ and P_{CO_2}.
- If the $[HCO_3^-]$ is low (implying a primary metabolic acidosis) then the anion gap (AG) should be examined and, if possible, compared with a known steady-state value.
- The AG is measured as follows: anion gap = $Na^+ - (Cl^- + HCO_3^-)$ = approximately 10 to 12 meq/L in the normal patient.
- If the AG is increased compared to the known previous value or is greater than 15, then by definition a wide-AG metabolic acidosis is present. If the AG is unchanged, then the disturbance is a nonwidened (sometimes termed unchanged-AG or hyperchloremic) metabolic acidosis.
- Next, examine whether the ventilatory response is appropriate. If the decrease in the P_{CO_2} equals the decrease in the $[HCO_3^-]$, there is appropriate respiratory compensation.
- If the decrease in the P_{CO_2} is greater than the decrease in the $[HCO_3^-]$, there is a concomitant respiratory alkalosis. If the decrease in the P_{CO_2} is less than the decrease in $[HCO_3^-]$, there is also a concomitant respiratory acidosis.
- If the P_{CO_2} is elevated (rather than the $[HCO_3^-]$ being decreased), the primary disturbance is respiratory acidosis. The next step is to figure out which type it is by examing the ratio of (the change in) $[H^+]$ to (the upward change in) the P_{CO_2}. If the ratio is 0.8, it is considered acute. If the ratio is 0.33, it is considered chronic.
- If the pH is greater than 7.45, the primary or predominant disturbance is a metabolic alkalosis.
- It is best to look at the $[HCO_3^-]$ first. If it is elevated, there is a primary metabolic alkalosis.
- If the P_{CO_2} is low, there is a primary respiratory alkalosis.

METABOLIC ACIDOSIS

- In considering metabolic acidosis, causes should be further divided into wide (elevated) and normal-AG acidosis. The term *anion gap* is misleading, because, in serum, there is no gap between total positive and negative ions; however, we commonly measure more positive ions than negative ions.

CLINICAL PRESENTATION

- No matter what the etiology, acidosis can cause nausea and vomiting, abdominal pain, change in sensorium, and tachypnea, sometimes a Kussmaul respiratory pattern.
- Acidosis also leads to decreased muscle strength and force of cardiac contraction, arterial vasodilation, venous vasoconstriction, and pulmonary hypertension.
- Patients may present with nonspecific complaints or shock.

TABLE 6-5 Causes of High-Anion-Gap Metabolic Acidosis

Lactic acidosis
Type A—Decrease in tissue oxygenation
Type B—No decrease in tissue oxygenation
Renal failure (acute or chronic)
Ketoacidosis
Diabetes
Alcoholism
Prolonged starvation (mild acidosis)
High-fat diet (mild acidosis)
Ingestion of toxic substances
Elevated osmolar gap
Methanol
Ethylene glycol
Normal osmolar gap
Salicylate
Paraldehyde
Cyanide

DIAGNOSIS AND DIFFERENTIAL

- Causes of metabolic acidosis can be divided into two main groups: (1) those associated with increased production of organic acids (increased-AG metabolic acidosis; see Table 6-5) and (2) those associated with a loss of bicarbonate or addition of chloride (normal-AG metabolic acidosis; see Table 6-6).
- A mnemonic to aid the recall of the causes of increased-AG metabolic acidosis is *a mud piles–* *a*lcohol, *m*ethanol, *u*remia, *D*KA, *p*araldehyde, *i*ron and *i*soniazid, *l*actic acidosis, *e*thylene glycol, *s*alicylates, and *s*tarvation.
- A mnemonic that can aid the recall of normal-AG metabolic acidosis is *used carp—u*reterostomy, *s*mall bowel fistulas, *e*xtra chloride, *d*iarrhea, *c*arbonic anhydrase inhibitors, *a*drenal insufficiency, *r*enal tubular acidosis, and *p*ancreatic fistula.

TABLE 6-6 Causes of Normal-Anion-Gap Metabolic Acidosis

With a tendency to hyperkalemia	With a tendency to hypokalemia
Subsiding DKA	Renal tubular acidosis type I
Early uremic acidosis	Renal tubular acidosis type II
Early obstructive uropathy	Acetazolamide therapy
Renal tubular acidosis type IV	Acute diarrhea (losses of HCO_3^- and K^+)
Hypoaldosteronism	Ureterosigmoidostomy
Potassium-sparing diuretics	

ABBREVIATIONS: DKA = diabetic ketoacidosis; HCO_3^- = bicarbonate; and K^+ = potassium.

TABLE 6-7 Indications for Bicarbonate Therapy in Metabolic Acidosis

INDICATION	RATIONALE
Severe hypobicarbonatemia (<4 meq/L)	Insufficient buffer concentrations may lead to extreme increases in acidemia with small increases in acidosis
Severe acidemia (pH < 7.20) with signs of shock or myocardial irritability that is not rapidly responsive to supportive measures	Therapy for the underlying cause of acidosis depends upon adequate organ perfusion
Severe hyperchloremic acidemia*	Lost bicarbonate must be regenerated by kidneys and liver, which may require days

* No specific definition by pH exists. The presence of serious hemodynamic insufficiency despite supportive care should guide the use of bicarbonate therapy for this indication.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Give supportive care by improving perfusion, administering fluids as needed, and improving oxygenation and ventilation.
- Correct the underlying problem. If the patient has ingested a toxin, lavage, administer activated charcoal, give the appropriate antidote, and perform dialysis as directed by the specific toxicology chapters in this handbook. If the patient is septic, perform cultures and administer antibiotics as directed by the appropriate chapters in this handbook. If the patient is in shock, administer fluids and vasopressors as directed by the appropriate chapters in Section 3 of this book. If the patient is in DKA, treat as directed in Chap. 125 with IV fluids and insulin.
- Indications for bicarbonate therapy are listed in Table 6-7.
- When bicarbonate is used, Adrogue and Madias[3] recommend administering 0.5 meq/kg bicarbonate for each meq/dL of desired rise in $[HCO_3^-]$. The goal is to restore adequate buffer capacity $[HCO_3^-]$ >8 meq/dL) or achieve clinical improvement in shock or dysrhythmias.
- Bicarbonate should be given as slowly as the clinical situation permits; 1.5 ampules of sodium bicarbonate in 500 mL D_5W produces a nearly isotonic solution for infusion.

METABOLIC ALKALOSIS

- The two most common causes of metabolic alkalosis are excessive diuresis (with loss of potassium,

hydrogen ion, and chloride) and excessive loss of gastric secretions (with loss of hydrogen ion and chloride).
- Other causes of hypokalemia should also be considered.

CLINICAL FEATURES

- Symptoms of the underlying disorder (usually fluid loss) dominate the clinical presentation, but general symptoms of metabolic alkalosis include muscular irritability, tachydysrhythmias, and impaired oxygen delivery.
- The diagnosis of metabolic alkalosis is made from laboratory studies revealing a bicarbonate level above 26 meq/L and a pH above 7.45.
- In most cases, there is also an associated hypokalemia and hypochloremia.
- The differential diagnosis includes dehydration, loss of gastric acid, excessive diuresis, administration of mineralocorticoids, increased intake of citrate or lactate, hypercapnia, hypokalemia, and severe hypoproteinemia.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Administer fluids in the form of NS in cases of dehydration.
- Administer potassium as KCl, not faster than 20 meq/h, unless serum potassium is above 5.0 meq/L.

RESPIRATORY ACIDOSIS

CLINICAL PRESENTATION

- Respiratory acidosis may be life-threatening and a precursor to respiratory arrest. The clinical picture is often dominated by the underlying disorder.
- Typically, respiratory acidosis depresses mental function, which may progressively slow the respiratory rate. Patients may be confused, somnolent, and eventually unconscious.
- Although patients are frequently hypoxic, in some disorders the fall in oxygen saturation may lag behind the elevation in P_{CO_2}. Pulse oximetry may be misleading, making arterial blood gases essential for the diagnosis.
- The differential diagnosis includes chronic obstructive pulmonary disease (COPD), drug overdose, CNS disease, chest wall disease, pleural disease, and trauma.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Increase ventilation. In many cases, this requires intubation. The hallmark indication for intubation in respiratory acidosis is depressed mental status. Only in opiate intoxication is it acceptable to await treatment of the underlying disorder (rapid administration of naloxone) before reversal of the hypoventilation.
- Treat the underlying disorder. Remember that high-flow oxygen therapy may lead to exacerbation of CO_2 narcosis in patients with COPD and CO_2 retention. Monitor these patients closely when administering oxygen and intubate if necessary.

RESPIRATORY ALKALOSIS

CLINICAL PRESENTATION

- Hyperventilation syndrome is a problematic diagnosis for the emergency physician, as a number of life-threatening disorders present with tachypnea and anxiety: asthma, pulmonary embolism, diabetic ketoacidosis, and others.
- Symptoms of respiratory alkalosis are often dominated by the primary disorder promoting the hyperventilation.
- Hyperventilation by virtue of the reduction of P_{CO_2}, however, lowers both cerebral and peripheral blood flow, causing distinct symptoms.
- Patients complain of dizziness; painful flexion of the wrists, fingers, ankles, and toes (carpal-pedal spasm); and, frequently, a chest pain described as tightness.
- The diagnosis of hyperventilation due to anxiety is a diagnosis of exclusion. Arterial blood gases can be used to rule out acidosis and hypoxia. (See Chap. 28, ''Pulmonary Embolism,'' for discussion of calculating the alveolar-arterial oxygen gradient.)
- Causes of respiratory alkalosis to consider include hypoxia, fever, hyperthyroidism, sympathomimetic therapy, aspirin overdose, progesterone therapy, liver disease, and anxiety.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treat the underlying cause. Only when more serious causes of hyperventilation are ruled out should you consider the treatment of anxiety. Anxiolytics, such as lorazepam 1 to 2 mg, IV or PO, may be helpful.
- Rebreathing into a paper bag can cause hypoxia; it is not recommended.[4, 5]

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Section 3
SHOCK

7 THERAPEUTIC APPROACH TO THE HYPOTENSIVE PATIENT

James L. Larson

EPIDEMIOLOGY

- More than 1 million cases of shock present to emergency departments every year.

PATHOPHYSIOLOGY

- Shock is defined as a circulatory insufficiency that creates an imbalance between tissue oxygen supply and demand.
- Shock is classified into four categories by etiology: (a) hypovolemic, (b) cardiogenic, (c) distributive (e.g., anaphylaxis), and (d) obstructive (extracardiac obstruction to blood flow).
- Mean arterial pressure (MAP) is equal to the cardiac output (CO) × systemic vascular resistance (SVR). When oxygen demand exceeds delivery, compensatory mechanisms attempt to maintain homeostasis. First, there is an increase in cardiac output. Next, the amount of oxygen extracted from hemoglobin increases. If the compensatory mechanisms are unable to meet oxygen demand, anaerobic metabolism occurs, resulting in the formation of lactic acid.

CLINICAL FEATURES

- The precipitating cause may be clinically obvious (e.g., trauma, anaphylaxis) or occult (e.g., adrenal insufficiency). The four main classes of shock are hypovolemic, cardiogenic, distributive, and obstructive.
- A targeted history of the presenting symptoms and previously existing conditions, including medication use, may reveal the cause of the shock.[1]
- Body temperature may be elevated, normal, or subnormal.
- Cardiovascular: Heart rate is usually elevated. Exceptions include paradoxical bradycardia in hemorrhagic shock, hypoglycemia, beta-blocker use, and cardiac disease. Blood pressure may initially be normal or elevated due to compensatory mechanisms, later falling when cardiovascular compensation fails. Neck veins may be distended or flattened, depending on the etiology of shock. Decreased coronary perfusion pressures can lead to ischemia, decreased ventricular compliance, and increased left ventricular diastolic pressure and pulmonary edema.
- Respiratory: Tachypnea, increased minute ventilation, and increased dead space are common. Bronchospasm, hypocapnia with progression to respiratory failure, and adult respiratory distress syndrome can be seen.
- Skin: Many skin findings are possible, including pale, dusky, clammy skin with cyanosis, sweating, altered temperature, and decreased capillary refill.
- Gastrointestinal: The low-flow state found in shock can produce ileus, GI bleeding, pancreatitis, acalculous cholecystitis, and mesenteric ischemia.
- Renal: Oliguria may result from a reduced glomerular filtration rate; however, a paradoxical polyuria can occur in sepsis, which may be confused with adequate hydration status.
- Metabolic: Respiratory alkalosis is the first acid-base abnormality, progressing to metabolic acidosis as shock continues. Blood sugar may be increased or decreased. Hyperkalemia is a potentially life-threatening metabolic abnormality.

DIAGNOSIS AND DIFFERENTIAL

- The presumed etiology of shock will determine the specific diagnostic measures to be employed.
- Commonly performed laboratory studies include complete blood count (CBC), platelet count, electrolytes, blood urea nitrogen (BUN), creatinine, glucose, prothrombin and partial thromboplastin times, and urinalysis. Other laboratory tests frequently employed include arterial blood gases (ABG), lactic acid, fibrinogen, fibrin split products, D-dimer, cortisol levels, hepatic function tests, and cerebrospinal fluid studies.
- Cultures of blood, urine, cerebrospinal fluid, and wounds are ordered as necessary.
- Common diagnostic tests ordered include radiographs (chest and abdominal), electrocardiograms, ultrasound or computed tomography (CT) scans (chest, head, abdomen, and pelvis), and echocardiograms.
- A pregnancy test should be performed in all females of childbearing age.
- Determination of the etiology of shock will guide therapy. Consider less common causes of shock when there is a lack of a response to initial therapy. These include cardiac tamponade, tension pneumothorax, adrenal insufficiency, toxic or allergic reactions, and occult bleeding. Occult bleeding can occur from a ruptured ectopic pregnancy or may stem from intraabdominal or pelvic sources.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The goal of the interventions is to restore adequate tissue perfusion and identify and treat the underlying etiology.
- Airway control, employing endotracheal intubation when necessary for respiratory distress or persistent shock.
- Supplemental high-flow oxygen.
- Early surgical consultation for internal bleeding. Most external hemorrhage can be controlled by direct compression.
- Adequate venous access. Large-bore peripheral intravenous catheters will usually allow adequate fluid resuscitation. Central venous access may be necessary for monitoring and employing some therapies, including pulmonary artery catheters, venous pacemakers, and long-term vasopressor therapy.
- Volume replacement. Isotonic, intravenous crystalloid fluids (0.9% NaCl, Ringer's lactate) are preferred for the initial resuscitation phase. Initial bolus volume is 20 to 40 mL/kg over 10 to 20 min. Blood is the ideal resuscitative fluid for hemorrhagic shock or in the presence of significant anemia. Fully cross-matched blood is preferred, but if more rapid intervention is required, type-specific or type O negative blood may be employed. The decision to use platelets or fresh-frozen plasma (FFP) should be based on evidence of impaired hemostasis and on frequent monitoring of coagulation parameters. Platelets are generally given if there is ongoing hemorrhage and the platelet count is 50,000 or less; FFP is indicated if the prothrombin time is prolonged more than 1.5 times.
- Vasopressors should be used if there is persistent hypotension after adequate volume resuscitation. American Heart Association recommendations based on blood pressure are dobutamine 2.0 to 20.0 μg/kg/min for systolic BP over 100 mmHg, dopamine 2.5 to 20.0 μg/kg/min for systolic BP 70 to 100 mmHg, and norepinephrine 0.5 to 30.0 μg/min for systolic BP under 70 mmHg.
- Acidosis should be treated with adequate ventilation and fluid resuscitation. Use of sodium bicarbonate (1 meq/kg) is controversial.[2] If it is used, it is given only in the setting of severe acidosis refractory to ventilation and fluid resuscitation.
- Early surgical or medical consultation for admission or transfer as indicated.

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8 SEPTIC SHOCK

James L. Larson

EPIDEMIOLOGY

- Mortality due to septic shock ranges from 20 to 80 percent, depending on the patient's premorbid state.[1]
- Sepsis is more common in older adults, with a mean age of 55 to 60 years.[1]
- Factors that predispose to gram-negative bacteremia include diabetes mellitus, lymphoproliferative disorders, cirrhosis of the liver, burns, invasive procedures or devices, and chemotherapy.[1]
- Factors that predispose to gram-positive bacteremia include vascular catheters,[1] indwelling mechanical devices, burns, and IV drug use.
- Fungemia most often occurs in immunocompromised patients.[2]

PATHOPHYSIOLOGY

- Sepsis starts as a focus of infection that results in either bloodstream invasion or a proliferation of organisms at the infected site. These organisms release exogenous toxins that can include endotoxins and exotoxins.[3–5]
- The host's reaction to these toxins results in the release of humoral defense mechanisms, including cytokines (tumor necrosis factor, interleukins), platelet activating factor, complement, kinins, and coagulation factors. These factors can have deleterious effects, including myocardial depression and vasodilation resulting in refractory hypotension and multiple organ system failure.

CLINICAL FEATURES

- Fever or hypothermia may be seen in sepsis. Hypothermia is more often seen in patients at the extremes of age and in immunocompromised patients.[6]
- Other vital-sign abnormalities include tachycardia, wide pulse pressure, tachypnea, and hypotension.[6]
- Mental status changes ranging from mild disorientation to coma are commonly seen.
- Ophthalmic manifestations include retinal hemorrhages, cotton-wool spots, and conjunctival petechiae.
- Cardiovascular manifestations initially include vasodilation, resulting in warm extremities.[7–9] Cardiac output is maintained early in sepsis through a compensatory tachycardia. As sepsis progresses, hypotension may occur. Patients in septic shock may demonstrate a diminished response to volume replacement.
- Respiratory symptoms include tachypnea and hypoxemia. Sepsis remains the most common condition associated with acute respiratory distress syndrome (ARDS). ARDS may occur within minutes to hours from the onset of sepsis.
- Renal manifestations include azotemia, oliguria, and active urinary sediment due to acute tubular necrosis.[10]
- Hepatic dysfunction is common. The most frequent presentation is cholestatic jaundice. Increases in transaminases, alkaline phosphatase, and bilirubin are often seen. Severe or prolonged hypotension may induce acute hepatic injury or ischemic bowel necrosis. Painless mucosal erosions may occur in the stomach and duodenum and cause upper GI bleeds.
- Skin findings may be present in sepsis. Local infections can be present from direct invasion into cutaneous tissues. Examples include cellulitis, erysipelas, and fasciitis. Hypotension and disseminated intravascular coagulation (DIC) can also produce skin changes, including acrocyanosis and necrosis of peripheral tissues. Infective endocarditis can produce microemboli, which cause skin changes.
- Hematologic changes include neutropenia, neutrophilia, thrombocytopenia, and DIC.[11] Neutropenia is associated with increased mortality. The hemoglobin and hematocrit are usually not affected unless the sepsis is prolonged or there is an associated GI bleed.
- Thrombocytopenia occurs in over 30 percent of patients with sepsis.[11] DIC is more often associated with gram-negative sepsis. Decompensated DIC presents with clinical bleeding and thrombosis. Laboratory studies can show thrombocytopenia, prolonged prothrombin time (PT) and partial prothromboplastin time (PTT), decreased fibrinogen level and antithrombin levels, and increased fibrin monomer, fibrin split values, and D-dimer values.
- Hyperglycemia may be the result of increased catecholamines, cortisol, and glucagon. Increased insulin resistance, decreased insulin production, and impaired utilization of insulin may further contribute to hyperglycemia.
- Arterial blood gas (ABG) studies in early sepsis may reveal hypoxemia and respiratory alkalosis. As perfusion worsens and glycolysis increases, a metabolic acidosis results.

DIAGNOSIS AND DIFFERENTIAL

- Septic shock should be suspected in any patient with a temperature of >38° or <36°C (>100.4° or <96.8°F), systolic blood pressure of <90 mmHg, and evidence of inadequate organ perfusion. Hypotension may not reverse with volume replacement.
- Clinical features may include mental obtundation, hyperventilation, hot or flushed skin, and a wide pulse pressure.
- Complete blood count (CBC), platelet count, DIC panel (PT, PTT, fibrinogen, D-dimer, and antithrombin concentration), electrolyte levels, liver function tests, renal function tests, ABG analysis, and urinalysis should be considered in a patient with suspected sepsis.
- Cultures of cerebrospinal fluid (CSF), sputum, blood, urine, and wounds should be obtained as indicated.
- Radiographs of suspected foci of infection (chest, abdomen, etc.) should be obtained.
- Ultrasonography or computed tomography (CT) scanning may help identify occult infections in the cranium, thorax, abdomen, and pelvis.
- Acute meningitis is the most common central nervous system infection associated with septic shock; in this case a lumbar puncture should be considered.[6] If meningitis is a significant consideration, empiric antibiotics should be given as soon as possible.
- Differential diagnosis should include noninfectious causes of shock, including hypovolemic, cardiogenic, neurogenic, and anaphylactic causes.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Aggressive airway management with high-flow oxygen and endotracheal intubation may be necessary.
- Rapid infusion of crystalloid IV fluid (Ringer's lactate or normal saline) at 500 mL (20 mL/kg in children) every 5 to 10 min; 4 to 6 L (60 mL/kg in children) may be necessary.[12] In addition to blood pressure, mental status, pulse, capillary refill, central venous pressure, pulmonary capillary wedge pressure, and urine output (>30 mL/h in adults, 1 mL/kg/h in children) can be monitored to evaluate therapy. If ongoing blood loss is suspected, blood replacement may be necessary.
- Dopamine 5 to 20 μg/kg/min, titrated to response, should be used if hypotension is refractory to IV fluid.[12]
- If blood pressure remains <70 mmHg despite preceding measures, a norepinephrine 8- to 12-μg/min loading dose and a 2- to 4-μg/min infusion to maintain mean arterial blood pressure of at least 60 mmHg should be started.[12]
- The source of infection must be removed if possible (remove indwelling catheters and incision and drainage of abscesses).
- Empiric antibiotic therapy. This measure is ideally begun after cultures are obtained, but administration should not be delayed. Dosages should be maximum allowed and given intravenously. When source is unknown, therapy should be effective against both gram-positive and gram-negative organisms. In adults, a third-generation cephalosporin (ceftriaxone 1 g IV, cefotaxime 2 g IV, or ceftazidime 2 g IV) or an antipseudomonal beta lactamase–susceptible penicillin can also be used. Addition of an aminoglycoside (gentamicin 2 mg/kg IV, tobramycin 2 mg/kg IV) to this regimen is recommended. In immunocompromised adults, ceftazidime 2 g IV, imipenum 750 mg IV, or meropenum 1 g IV alone is acceptable. If gram-positive infection is suspected (indwelling catheter or IV drug use), oxacillin 2 g IV or vancomycin 15 mg/kg IV should be added. If an anaerobic source is suspected (intraabdominal, genital tract, odontogenic, and necrotizing soft tissue infection), metronidazole 7.5 mg/kg IV or clindamycin 900 mg IV should additionally be administered. If *Legionella* is a potential source, erythromycin 500 mg IV should be added.
- Acidosis is treated with oxygen, ventilation, and IV fluid replacement. If acidosis is severe, administration of sodium bicarbonate 1 meq/kg IV is acceptable as directed by ABGs.
- DIC should be treated with fresh-frozen plasma 15 to 20 mL/kg initially to keep PT at 1.5 to 2 times normal and treated with platelet infusion to maintain serum concentration of 50 to 100,000.
- If adrenal insufficiency is suspected, glucocorticoid (Solu-Cortef 100 mg IV) should be administered.[13]

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9 CARDIOGENIC SHOCK

Rawle A. Seupaul

EPIDEMIOLOGY

- Cardiogenic shock is the most common cause of in-hospital mortality from acute myocardial infarction—accounting for 50,000 to 70,000 deaths per year.
- Approximately 5 to 7 percent of patients with acute myocardial infarction (AMI) will develop cardiogenic shock.
- Cardiogenic shock usually occurs early in the course of AMI—median time of 7 h.
- Risk factors for developing cardiogenic shock after AMI are advanced age, female gender, large MI, anterior wall MI, previous MI, previous congestive heart failure, multivessel disease, proximal left anterior descending artery occlusion, and diabetes mellitus.[1]
- With medical treatment alone, mortality from cardiogenic shock is high—70 to 90 percent.

PATHOPHYSIOLOGY

- Cardiogenic shock most commonly occurs secondary to left ventricular infarction involving approximately 40 percent of the left ventricular mass.
- Reduction in cardiac output leads to oliguria, hepatic failure, anaerobic metabolism, lactic acidosis, and hypoxia. These outcomes serve to further impair myocardial function.
- Multivessel disease, diastolic dysfunction, and dysrhythmias hasten the development of cardiogenic shock. The presence of these factors may produce shock with less than 40 percent left ventricular involvement.
- Compensatory mechanisms attempt to maximize cardiac output. Initially, sympathetic tone is increased, resulting in increased myocardial contractility. This can be visualized as compensatory hyperkinesis by echocardiography.
- Sympathetic activity activates the renin-angiotensin system. This results in arterial and venoconstriction as well as in an increased blood volume. The latter is accomplished by sodium and water resorption mediated by aldosterone.
- Right ventricular infarction accounts for approximately 3 to 4 percent of cases of cardiogenic shock. This is usually associated, however, with concomitant left ventricular dysfunction.
- Cardiogenic shock occurs when there is insufficient pumping ability of the heart to support the metabolic needs of the tissues.

CLINICAL FEATURES

- Cardiogenic shock almost always presents with hypotension (systolic blood pressure <90 mmHg).
- Tachycardia or bradycardia may be present. If excessive they should be treated appropriately.
- Patients may be cool, have clammy skin, and become oliguric.
- Diminished cerebral perfusion may lead to altered mentation.
- Left ventricular failure may result in tachypnea, rales, and frothy sputum.

- Valvular dysfunction and septal defects may be discernible by auscultating a murmur.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of cardiogenic shock should be suspected from the initial history and physical exam. Ancillary tests are, however, essential to confirm the diagnosis. These include (*a*) ECG consistent with AMI. Right-sided leads should be performed if posterior wall infarction is suspected. (*b*) Chest radiograph for evidence of congestive heart failure, abnormal mediastinum, and evaluation of the cardiac silhouette. (*c*) Two-dimensional transthoracic echocardiography done at the bedside can quickly evaluate regional hypokinesis, akinesis, or dyskinesis. (*d*) Laboratory studies including cardiac enzymes, coagulation parameters, serum lactate, and chemistries may also help establish the diagnosis.
- Disease processes to be considered in the differential diagnosis include aortic dissection, pulmonary embolism, pericardial tamponade, acute valvular insufficiency, hemorrhage, and sepsis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The patient should be stabilized, endotracheal intubation should be performed if necessary, intravenous access attained, high-flow oxygen provided, the patient placed on a monitor and pulse oximeter, and an ECG and rhythm strip obtained.
- The patient should bite and chew 160 to 325 mg of aspirin unless contraindicated by allergy.
- Rhythm disturbances, hypovolemia, hypoxemia, and electrolyte abnormalities should be identified early and treated accordingly.
- Intravenous nitroglycerin and/or morphine should be titrated for chest pain as well as hemodynamic parameters.
- If hypotension is present after adequate fluid resuscitation, dobutamine and/or dopamine should be considered for inotropic and pressor support.[2, 3]
- For preload and afterload reduction, the use of nitroglycerin or nitroprusside respectively may be indicated.
- An intraaortic balloon pump may be necessary for afterload reduction.
- Thrombolysis, percutaneous transluminal angioplasty, or emergent bypass surgery should be considered if available.
- Cardiology and/or thoracic surgery should be consulted early.

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10 NEUROGENIC SHOCK

Rawle A. Seupaul

EPIDEMIOLOGY

- Approximately 10,000 spinal cord injuries occur in the United States each year.[1]
- The majority of cases are due to blunt trauma (motor vehicle crash, fall, and sports), while penetrating trauma accounts for 10 to 15 percent of cases (gunshot and stab wounds).[2, 3]

PATHOPHYSIOLOGY

- Neurogenic shock occurs when an acute spinal cord injury disrupts sympathetic flow, resulting in hypotension and bradycardia.[2]
- Spinal shock is a distinct entity that refers to transient loss of spinal reflexes below the level of a complete or partial cord injury.[4]
- Primary cord injury reflects the initial changes caused by the traumatic event (compression, laceration, or stretching of the spinal cord).
- Secondary injury ensues over several days to weeks and is caused mostly by continued cord ischemia.[4, 5]

CLINICAL FEATURES

- Within the first 2 to 3 min, the initial cardiovascular response is hypertension, widened pulse pressure, and tachycardia.[2, 6]
- As sympathetic tone is lost, the patient will be hypotensive with warm, dry skin.[7]
- The inability to redirect blood from the periphery to the core may result in hypothermia.
- Most patients will be bradycardic secondary to overriding vagal tone.
- Any injury above T1 should disrupt the entire sympathetic chain. Injuries between T1 to L3 may result in partial sympathetic disruption; the lower the injury, the less effects on the sympathetic nervous system.
- The symptoms of neurogenic shock may last from 1 to 3 weeks.[7]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Diagnosing neurogenic shock is always one of exclusion. Other potential causes of hypotension must be ruled out and treated aggressively. Once the ABCs are addressed and the diagnosis of neurogenic shock is made, therapy is aimed at mitigating hypotension and bradycardia.
- Crystalloid should be infused with a goal mean arterial pressure above 70 mmHg. If inotropic support is necessary, the use of dobutamine or dopamine may be beneficial.[7, 8]
- For symptomatic bradycardia, atropine should be used. In patients who develop heart block or asystole, a pacemaker may be necessary.[6]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 31, ''Neurogenic Shock,'' by Brian Euerle and Thomas M. Scalea.

11 ANAPHYLAXIS AND ACUTE ALLERGIC REACTIONS

Damian F. McHugh

EPIDEMIOLOGY

- The spectrum of allergic reactions ranges from mild cutaneous symptoms to life-threatening anaphylaxis.
- Because of this disease spectrum, incidence and prevalence data are limited.
- Four fatalities per 10 million people are seen annually.[1]
- The faster the onset of symptoms, the more severe the reaction; half the fatalities occur within the first hour.[2]

PATHOPHYSIOLOGY

- The mechanism of allergic reactions is classically a type 1 hypersensitivity reaction, whereby allergen-induced IgE molecules cross-link on the surface of mast cells or basophils, causing degranulation and release of inflammatory mediators.
- Other reactions have been described, through complement activation,[3,4] by direct stimulation of

the mast cell, or by unknown mechanisms—the so-called anaphylactiod reactions.[3,5]
- Common causes are penicillin (especially intravenously, IV), aspirin/other nonsteroidals, ACE-inhibitors, trimethoprim-sulfamethoxazole, radiocontrast media, Hymenoptera stings, peanuts, shellfish, milk, eggs, monosodium glutamate, nitrites, and dyes.
- Idiopathic anaphylaxis is, by definition, of unknown cause.
- Perhaps surprisingly, anaphylaxis is not automatic on recurrent exposure; recurrence rates are 40 to 60 percent for insect stings, 20 to 40 percent for radiocontrast media, and 10 to 20 percent for penicillin.[5]
- Concurrent use of beta blockers is a risk for severe, prolonged anaphylaxis.

CLINICAL FEATURES

- Urticaria (hives) is a cutaneous IgE-mediated reaction yielding itchy red wheals of varying sizes that disappear promptly. Angioedema is a similar reaction with edema in the dermis, usually of the face and neck. By definition, anaphylaxis includes either respiratory compromise or cardiovascular collapse.
- Reactions can occur in seconds or be delayed over 1 h after allergen exposure. Reactions are ''biphasic,'' with further mediator release occurring up to 4 to 8 h later in up to 20 percent of cases.
- Respiratory symptoms are stridor, dyspnea, and wheezing.
- GI features are nausea, cramps, diarrhea, and vomiting.
- Pruritus and urticaria are the most common initial symptoms.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is made clinically. A history of exposure to an agent, followed by symptoms and signs as described earlier make the diagnosis of acute allergic reaction.
- No tests are diagnostic. Workup may focus on excluding other diagnoses or tests needed to stabilize the cardiorespiratory systems.
- Differential diagnosis includes vasovagal reaction, asthma, acute coronary ischemic syndromes/dysrhythmias, epiglottitis or foreign body, carcinoid, mastocytosis, or hereditary angioedema (treated with fresh-frozen plasma).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- A: *Airway.* Anticipate intubation earlier rather than later, especially in hoarse patients, or those with a ''lump in my throat.'' Edema may necessitate endotracheal tube selection 1 to 2 sizes smaller. A cricothyroidotomy kit should be open and ready before you intubate.
- B: *Breathing.* Administer high-flow oxygen as necessary. Treat bronchospasm with nebulized albuterol, 0.5 mL of a 5% solution in 3 mL saline.
- C: *Circulation.* Most patients, especially if hypotensive, need large volumes of crystalloid. If hypotension persists after 1 to 2 L of IV fluid, IV epinephrine is needed (see later). Consider colloid also.
- D: *Discontinue* the antigen exposure, for example, stop IV drug infusions or remove bee stingers.
- E: *Epinephrine.* If severe respiratory distress, laryngeal edema, or severe shock, IV epinephrine is indicated.[2] Put 0.1 mL of 1 : 1000 in 10 mL saline and infuse over 5 to 10 min. If no response, start an epinephrine infusion with 1 mg (1 mL of 1 : 1000) in 500 mL saline at 0.5 to 2 mL/min (1 to 4 μg/min) and titrate to effect. For less severe signs, give subcutaneous epinephrine 0.3 to 0.5 mL of 1 : 1000 every 5 to 10 min according to response. If repeated SC doses do not work, go to IV.
- F: *Further treatments.* Antihistamines are helpful: (H_1) blockers such as diphenhydramine 25 to 50 mg IV are helpful and (H_2) blockers such as ranitidine 50 mg can be helpful. Steroids only help control persistent or delayed allergic reactions. Severe cases can be given methylprednisolone 125 mg IV, with oral prednisone 60 mg for less severe cases.
- G: *Glucagon.* 1 to 2 mg every 5 min may be helpful for hypotension refractory to epinephrine and fluids in patients taking beta blockers.
- Observe for 1 h those patients with mild reactions, 6 h those patients who receive epinephrine, and admit all patients with severe reactions to the intensive care unit.
- Serious cases should be provided with Epi-Pens at discharge and instructed in how and when to use them.
- Discharge patients with prescriptions for antihistamines and prednisone that will cover 4 days.

- Referral of patients to an allergist for follow-up is good practice.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th, ed., see Chap. 30, ''Anaphylaxis and Acute Allergic Reactions,'' by Shaheed I. Koury and Lee U. Herfel.

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Section 4

ANALGESIA, ANESTHESIA, AND SEDATION

12 ACUTE PAIN MANAGEMENT AND CONSCIOUS SEDATION

Jim Edward Weber

- The majority of patients present to the emergency department (ED) with conditions associated with pain. However, inadequate analgesia and sedation continue to be problematic in this setting.
- Factors contributing to oligoanalgesia include a limited understanding of the related pharmacology, misunderstanding of the patient's perception of pain, and fear of serious side effects.[1]

PATHOPHYSIOLOGY

- Noxious stimuli are first registered peripherally by nociceptors, C fibers, A-σ fibers, and free nerve endings, resulting in the release of glutamate, substance P, neurokinin A, and calcitonin gene–related peptide within the spinal cord.[2]
- Pain is modulated at the level of the dorsal root ganglion, inhibitory interneurons, and ascending pain tracts.
- Cognitive interpretation, localization, and identification of pain occur at the level of the hypothalamus, thalamus, limbus, and reticular activating system.

CLINICAL FEATURES

- Physiologic responses to pain and anxiety include tachycardia, blood pressure elevation, tachypnea, diaphoresis, flushing or pallor, nausea, and muscle tension.
- Behavioral changes include facial expressions, posturing, crying, and vocalization.
- Competent patients who are awake and cooperative can often reliably localize pain and determine its quality and severity.[3]
- Patients who are less able to quantify and localize their pain are at risk for inadequate pain management. Patients at risk include those whose cultural background differs significantly from that of their providers, the elderly, children, patients with language barriers, those with psychosis, and the cognitively impaired.[4,5]
- Subjective impressions of pain are often incorrect. Therefore, pain is best assessed using a validated, age-appropriate, objective pain scale.[6,7]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of anxious patients or those in need of painful procedures should first begin with nonpharmacologic interventions. Examples include application of heat or cold, immobilization or elevation of injured extremities, relaxation, distraction, and guided imagery.
- Communication techniques include explanation and reassurance, with time given for questions and answers.
- With pediatric patients, discussion of the procedure just prior to the intervention may minimize anxiety. Parents should be included in the interventional process to provide comfort.
- Recalcitrant children will require restraints. Parents should not be included in the restraint process.
- If pharmacologic intervention using sedation and/

or analgesia is necessary, choice of the best agent should be guided by the route of delivery and the desired duration of effect.

Systemic Sedation and Analgesia

- Sedation is a pharmacologically controlled state of depressed consciousness. Light or conscious sedation allows for the maintenance of protective airway reflexes and appropriate response to verbal commands. Deep sedation produces marked depression of consciousness and may result in an unconscious state with or without protective reflexes. Analgesia refers to the interruption of the propagation of axonal action potentials without the production of intentional sedation.
- Agents providing conscious sedation often have a narrow therapeutic index and should therefore be given in small incremental doses, allowing adequate time for the development and assessment of peak effect. Constant reassessment is required.
- All patients undergoing systemic sedation or analgesia require continuous pulse oximetry, cardiac monitoring, and constant observation by a provider trained in airway management.
- Oxygen, suction, airway equipment, and resuscitation drugs should be immediately available.
- A baseline blood pressure, heart rate, respiratory rate, and level of consciousness should be assessed every 5 to 10 min.
- Precalculated doses of ''rescue'' or reversal agents should be at the bedside: naloxone, 0.1 mg/kg every 2 to 3 min, until desired effect for opiates; flumazenil, 0.01 to 0.02 mg/kg, with additional 0.005-mg doses to a maximum of 0.2 mg per dose and 1 mg total, for benzodiazepines.
- Flumazenil is indicated for reversal of respiratory depression during conscious sedation; routine use to awaken patients is not recommended.[8] In addition, due to the risk of seizures, it should not be used on patients with a history of chronic benzodiazepine or tricyclic antidepressant use.

Analgesia Nonopiates

- Nonopiate agents may be used for mild pain or as an adjunct for moderate pain in combination with codeine. Opiates are the analgesics of choice for moderate to severe pain.
- Acetaminophen has no anti-inflammatory or antiplatelet effects. Potential hepatotoxicity may occur in doses above 140 mg/kg/day in patients with normal kidney and liver function.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) include aspirin, naproxen, indomethacin, ibuprofen, and ketorolac. The safety and efficacy of ibuprofen have been established for children over 6 months of age. Advantages include no respiratory depression or sedation. NSAIDs have opiate dose–sparing effects. Potential side effects include platelet dysfunction, impaired coagulation, and gastrointestinal irritation and bleeding.
- Aspirin has anti-inflammatory, antipyretic, and platelet inhibitory effects. Aspirin use in children is discouraged because of the strong association with Reye's syndrome. Aspirin should also be avoided in children with varicella or influenza.

Opiates

- Morphine is a naturally occurring compound which peaks in 10 to 30 min and may produce analgesia for up to 6 h. The dose of morphine is 0.1 to 0.2 mg/kg and is commonly administered IV or IM. Side effects include respiratory depression (particularly in infants $<$3 months of age) and hypotension due to histamine release.
- Fentanyl is a synthetic narcotic that is 100 times more potent than morphine. IV administration results in an almost immediate onset of action and approximately 30-min duration. The dose of fentanyl is 2 to 3 μg/kg IV or IM, with additional doses titrated by 0.5 μg/kg until desired anesthesia is achieved. Oral transmucosal fentanyl lozenges (Oralet) are dosed 10 to 15 μg/kg and are useful for painful pediatric procedures.[9] Fentanyl does not release histamine and therefore rarely causes hypotension.[10] Administration over 3 to 5 min can minimize respiratory depression. Chest wall rigidity has been reported at higher doses; this may not reverse with naloxone. In such cases, neuromuscular blockade and intubation may be required.
- Meperidine is a semisynthetic opiate that has been in common use in the ED setting. Currently, its use for ED analgesia is discouraged for the following reasons: (1) significant histamine release, (2) production of a toxic metabolite that may cause seizures unantagonized by naloxone, and (3) the potential for a fatal reaction when inadvertently given with monoamine oxidase inhibitors.
- Hydromorphone is an alternative to morphine, with 1 mg equivalent to 5 mg of morphine. It has a more rapid onset (15 min) and shorter duration of action (2 to 3 h) than morphine.
- The Demerol-Phenergan-Thorazine (DPT) cocktail has previously been used for pediatric analgesia during longer procedures. Its use for ED analgesia is currently not recommended because of unreliable efficacy, the potential for respiratory depression, and an exceedingly long (7-h) half-life.[11]

Nitrous Oxide

- N_2O is classified as an analgesic with both euphoric and dissociative properties and minimal cardiac or respiratory effects.
- It has a fast onset—peak effects are reached within 1 to 2 min; it is short-acting—baseline arousal is reached within minutes of cessation of therapy.
- N_2O must be delivered with oxygen to avoid hypoxia, and a fail-safe scavenger system must be in place.
- Side effects include nausea and vomiting. Nitrous oxide is contraindicated in patients with altered mental status, head injury, suspected pneumothorax, chronic obstructive pulmonary disease (COPD), a perforated viscus, eye injuries, or with balloon-tipped catheters.
- N_2O has opioid-agonist properties and therefore should be used with extreme caution if combined with a sedative or opioid so as to avoid deep sedation or general anesthesia.[12]

Ketamine

- Ketamine is a dissociative agent with both analgesic and anesthetic properties. The dose of ketamine is 4 mg/kg when given PO, PR, or IM, with supplemental doses given at 2 mg/kg per dose. The IV dose is 1 to 2 mg/kg over 1 to 2 min, with supplemental doses given at 0.25 mg/kg. Atropine (0.01 mg/kg) is often coadministered to control hypersalivation.
- Ketamine is a direct myocardial depressant and vasodilator. However, its central nervous system (CNS) effects usually result in tachycardia and vasoconstriction. The pulmonary effects include bronchorrhea and bronchodilatation; respiratory depression is uncommon when given over 1 to 2 min.
- Ketamine has catecholamine-like properties. It should be avoided in the setting of head injury and hypotension. Ketamine may also cause laryngospasm.[13]
- Adults and older children may have unpleasant emergence reactions upon awakening. Midazolam has been shown to attenuate this experience, but caution must be taken to avoid respiratory depression.[14]
- Contraindications include age $\leq$ 3 months, history of airway instability or tracheal stenosis, procedures involving stimulation of the posterior pharynx, cardiovascular disease (hypertension and congestive heart failure), head injury, altered level of consciousness, CNS mass, hydrocephalus, history of seizures, glaucoma, acute globe injury, or psychosis.

Sedation

- Benzodiazepines (BNZs) are the sedative agents most commonly used for ED sedation.
- BNZs potentiate the effects of GABA, resulting in subsequent chloride influx, which produces the classic sedative, amnestic, anxiolytic, skeletal muscle–relaxant, and anticonvulsant effects.
- Midazolam is the most commonly used drug for ED conscious sedation. Advantages include rapid onset with short duration of action and excellent amnestic qualities. The adult dosage of midazolam is 0.25 to 1 mg every 3 to 5 min until sedation is achieved. Pediatric doses are 0.05 mg/kg to 0.1 mg/kg per dose every 3 to 5 min, with a maximum total dose of 0.2 mg/kg IV.
- Lower doses should be considered in elderly or intoxicated patients because of the risk of cardiovascular and respiratory depression.
- Barbiturates differ from BNZ in two important ways: (1) barbiturates can increase airway hyperreactivity and subsequent laryngospasm, thereby prohibiting their use in patients with underlying airway disease, and (2) barbiturates have a narrow therapeutic window, in which patients may rapidly progress from light sedation to general anesthesia. Hypotension is also common, particularly in hypovolemic patients.
- Methohexital and thiopental are classified as ultrashort-acting barbiturates. Methohexital (0.5 to 2 mg/kg) and thiopental (1 to 5 mg/kg) produce sedation within 1 to 2 min. Methohexital has also been successfully used to produce motionless sedation in children, for neuroimaging procedures, in doses of 25 mg/kg.
- Chloral hydrate (75 mg/kg) is a sedative without analgesic properties that has been used successfully in young children.[15] Respiratory depression is uncommon; however, deaths from airway obstruction have been reported. Major disadvantages include a long onset of action (30 to 60 min) and a prolonged duration of action (up to several hours).

Local and Regional Anesthesia

- Administered IV, by infiltration, and topically.
- Local anesthetics are divided into two classes, amides and esters. Lidocaine is the prototype amide and procaine the prototype ester. Bupivacaine is an amide anesthetic with a duration of action of 4 to 6 h and is preferred for prolonged procedures.
- Injection pain with lidocaine occurs because of the drug's acidic pH. Factors associated with decreased injection pain include buffering with bicarbonate, warming the medication prior to in-

jection, using smaller-gauge needles (27- to 30-gauge), and injecting the anesthetic slowly.
- The addition of epinephrine to lidocaine extends the length of anesthesia and slows systemic absorption. However, epinephrine decreases local perfusion and therefore cannot be used to anesthetize end organs (fingers, nose, penis, toes, and ears).
- Severe local anesthetic toxicity can lead to cardiovascular collapse, seizures, and death. The maximum dose of lidocaine is 4.5 mg/kg without epinephrine and 7 mg/kg with epinephrine.
- True allergic reactions to local anesthetics are rare and are usually due to the preservative para-aminobenzoic acid (PABA) in the case of esters and methylparaben in the case of amides. If a true allergy is suspected, the approach of choice is to use a preservative-free agent from the other class. Diphenhydramine is an additional alternative, despite having been shown to increase the pain of injection.
- Serious toxicity may result from inadvertent IV injection or infiltration of an excessive total dose. CNS complications include confusion, seizure, and coma; cardiac complications include myocardial depression and dysrhythmias.
- Several points are noteworthy in considering regional anesthesia: (1) the onset of anesthesia is delayed as compared with local anesthesia; (2) neurovascular status should always be performed prior to anesthesia; (3) epinephrine should not be used for digital blocks; and (4) aspiration should be performed prior to injection to avoid nerve injury and intravascular injection.
- The most common topical anesthetics for ED use are tetracaine, adrenaline cocaine, (TAC); lidocaine, epinephrine, tetracaine (LET); and lidocaine, prilocaine (EMLA). These preparations are advantageous because they obviate the need for injection and do not distort wound edges. Neither TAC nor LET should be used on mucous membranes or in end-arterial fields. EMLA, a cream, is reserved for use on intact skin.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 32, ''Acute Pain Management, Analgesia, and Anxiolysis in the Adult Patient,'' by Erica Liebelt and Nadine Levick; Chap. 33, ''Systemic Analgesia and Sedation for Painful Procedures,'' by David D. Nicolaou; and Chap. 130, ''Acute Pain Management and Sedation in Children,'' by Erica Liebelt and Nadine Levick.

13 MANAGEMENT OF PATIENTS WITH CHRONIC PAIN

David M. Cline

- Chronic pain is defined as a painful condition that lasts longer than 3 months.[1] Chronic pain can also be defined as pain that persists beyond the reasonable time for an injury to heal or a month beyond the usual course of an acute disease.

EPIDEMIOLOGY

- Chronic pain affects about one-third of the population at least once during a patient's lifetime, at a cost of 80 to 90 billion in health care payments and lawsuit settlements annually.
- Chronic pain may be caused by (*a*) a chronic pathologic process in the musculoskeletal or vascular system, (*b*) a chronic pathologic process in one of the organ systems, (*c*) a prolonged dysfunction in the peripheral or central nervous system, or (*d*) a psychological or environmental disorder.

PATHOPHYSIOLOGY

- The pathophysiology of chronic pain can be divided into three basic types. Nociceptive pain is associated with ongoing tissue damage. Neuropathic pain is associated with nervous system dysfunction in the absence of ongoing tissue damage. Finally, psychogenic pain has no identifiable cause.[2]

CLINICAL FEATURES

- Signs and symptoms of chronic pain syndromes are summarized in Table 13-1.
- ''Transformed migraine'' is a syndrome in which classic migraine headaches change over time and develop into a chronic pain syndrome. One cause of this change is frequent treatment with narcotics.[3]
- Fibromyalgia is classified by the American College of Rheumatology as the presence of 11 of 18 specific tender points, nonrestorative sleep, muscle stiffness, and generalized aching pain, with symptoms present longer than 3 months.[4]
- Risk factors for chronic back pain following an acute episode include male gender, advanced age, evidence of nonorganic disease, leg pain, prolonged initial episode, and significant disability at onset.[5]
- Previous recommendations for bed rest in the treatment of back pain have proved counterproductive.[6] Exercise programs have been found to be helpful in chronic low back pain.[7]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment with opiates frequently contributes to the psychopathologic aspects of the disease. Many pain specialists feel that they should not be used except for cancer pain.
- There are two essential points that affect the use of opioids in the emergency department (ED) on which there is agreement: (*a*) opioids should be used only in chronic pain if they enhance function at home and at work, and (*b*) a single practitioner should be the sole prescriber of narcotics or be aware of their administration by others.
- A previous narcotic addiction is a relative contraindication to the use of opioids in chronic pain.
- The management of chronic pain conditions is listed in Table 13-2.
- The need for long-standing treatment of chronic pain conditions may limit the safety of nonsteroidal anti-inflammatory drugs (NSAIDs). The newer cyclooxygenase-2 inhibitor types of NSAIDs, such as rofecoxib, 50 mg first dose, then 25 mg daily, may be an alternative for patients who cannot tolerate standard NSAIDs.
- Antidepressants are the most frequently used drugs for the management of chronic pain.[8] Often, effective pain control can be achieved at doses lower than typically required for relief of depression. When antidepressants are prescribed in the ED, a follow-up plan should be in place. The most common drug and dose is amitriptyline, 10 to 25 mg, 2 h prior to bedtime.
- Referral to the appropriate specialist is one of the most productive means to aid in the care of chronic pain patients who present to the ED. Chronic pain clinics have been successful at changing the lives of patients by eliminating opioid use, decreasing pain levels by one-third, and increasing work hours twofold.[9]

MANAGEMENT OF PATIENTS WITH DRUG-SEEKING BEHAVIOR

- Although it is known that approximately 10 percent of patients seeking treatment for drug addic-

TABLE 13-1 Signs and Symptoms of Chronic Pain Syndromes

DISORDER	PAIN SYMPTOMS	SIGNS
Myofascial headache	Constant dull pain, occasionally shooting pain	Trigger points on scalp, muscle tenderness, and tension
Transformed migraine	Initially migraine-like, becomes constant, dull; nausea, vomiting	Muscle tenderness and tension, normal neurologic examination
Fibromyalgia	Diffuse muscular pain, stiffness, fatigue, sleep disturbance	Diffuse muscle tenderness, >11 trigger points
Myofascial chest pain	Constant dull pain, occasionally shooting pain	Trigger points in area of pain
Myofascial back pain syndrome	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	Trigger points in area of pain, usually no muscle atrophy, poor ROM in involved muscle
Articular back pain	Constant or sharp pain exacerbated by movement	Local muscle spasm
Neurogenic back pain	Constant or intermittent, burning or aching, shooting or electric shocklike, may follow dermatome; leg pain > back pain	Possible muscle atrophy in area of pain, possible reflex changes
Complex regional pain type I (RSD)	Burning persistent pain, allodynia, associated with immobilization or disuse	Early: edema, warmth, local sweating Late: above alternates with cold, pale, cyanosis, eventually atrophic changes
Complex regional pain type II (causalgia)	Burning persistent pain, allodynia, associated with peripheral nerve injury	Early: edema, warmth, local sweating Late: above alternates with cold, pale, cyanosis, eventually atrophic changes
Postherpetic neuralgia	Allodynia, shooting, lancinating pain	Sensory changes in the involved dermatome
Phantom limb pain	Variable: aching, cramping, burning, squeezing, or tearing sensation	None

ABBREVIATIONS: ROM, range of motion; RSD, reflex sympathetic dystrophy.

TABLE 13-2 Management of Chronic Pain Syndromes

DISORDER	PRIMARY ED TREATMENT	SECONDARY TREATMENT*	POSSIBLE REFERRAL OUTCOME
Cancer pain	NSAIDs, opiates	Long-acting opiates	Optimization of medical therapy
Myofascial headache	NSAIDs, cyclobenzaprine	Antidepressants, phenothiazines	Trigger-point injections, optimization of medical therapy
Transformed migraine	NSAIDs, cyclobenzaprine	Antidepressants	Optimization of medical therapy, narcotic withdrawal
Fibromyalgia	NSAIDs	Antidepressants, exercise program	Optimization of medical therapy, dedicated exercise program
Myofascial chest pain	NSAIDs	Antidepressants	Trigger-point injections, optimization of medical therapy
Myofascial back pain syndrome	NSAIDs, stay active	Antidepressants	Trigger-point injections, optimization of medical therapy
Articular back pain	NSAIDs		Surgery, physical therapy
Neurogenic back pain	Acute: tapered solumedrol or prednisone	NSAIDs, muscle relaxants	Epidural steroids, surgery, exercise program
Complex regional pain types I and II (RSD and causalgia)	Acute: prednisone 60 mg/d × 4 days and taper to include 3 weeks of therapy	Chronic: Antidepressants, anticonvulsants	Sympathetic nerve blocks, TENS, spinal analgesia
Postherpetic neuralgia	Acute: simple analgesics	Chronic: antidepressants, capsaicin	Regional nerve blockade
Phantom limb pain	Simple analgesics	Antidepressants, anticonvulsants	TENS, sympathectomy

* If started in the ED, consultation and/or follow-up with pain specialist or personal physician recommended.
ABBREVIATIONS: NSAIDs, nonsteroidal anti-inflammatory drugs; RSD, reflex sympathetic dystrophy, TENS, transcutaneous electrical nerve stimulation.

tion identify a prescription drug as the principal drug of abuse,[10] there is no statistical documentation of the problem in the ED.

EPIDEMIOLOGY

- A study conducted in Portland found that drug-seeking patients presented to the ED 12.6 times per year, visited 4.1 different hospitals, and used 2.2 different aliases. Patients who were refused narcotics at one facility were successful in obtaining narcotics at another facility 93 percent of the time and were later successful at obtaining narcotics from the same facility 71 percent of the time.[11]

CLINICAL FEATURES

- Because of the spectrum of drug-seeking patients, the history given may be factual or fraudulent.
- Drug seekers may be demanding, intimidating, or flattering.
- In one study of the ED, the most common complaints of patients who were drug seeking were (in decreasing order): back pain, headache, extremity pain, and dental pain.[11]
- Many fraudulent techniques are used including ''lost'' prescriptions, ''impending'' surgery, factitious hematuria with a complaint of kidney stones, self-mutilation, and factitious injury.

DIAGNOSIS AND DIFFERENTIAL

- Drug-seeking behaviors can be divided into two groups: ''predictive'' and ''less predictive'' (Table 13-3). The behaviors listed under ''predictive'' are illegal in many states and form a solid basis to refuse narcotics to the patient.

TABLE 13-3 Characteristics of Drug-Seeking Behavior

Behaviors Predictive of Drug-Seeking Behavior*
Sells prescription drugs
Forges/alters prescriptions
Factitious illness, requests narcotics
Uses aliases to receive narcotics
Admits to illicit drug addiction
Conceals multiple physicians prescribing narcotics
Conceals multiple ED visits receiving narcotics
Less Predictive for Drug-Seeking Behavior
Admits to multiple doctors prescribing narcotics
Admits to multiple prescriptions for narcotics
Abusive when refused
Multiple drug allergies
Uses excessive flattery
From out of town
Asks for drugs by name

* Behaviors in this category are unlawful in many states.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of drug-seeking behavior is to refuse the controlled substance, consider the need for alternative medication or treatment, and consider referral for drug counseling.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 34, ''Management of Patients with Chronic Pain,'' by David M. Cline.

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Section 5
EMERGENCY WOUND MANAGEMENT

14 EVALUATING AND PREPARING WOUNDS

James F. Palombaro

EPIDEMIOLOGY

- Traumatic wounds account for more than 10 percent of all visits to emergency departments (EDs) in the United States.[1]
- The most frequently involved body locations are the face, scalp, fingers, and hands.[2–5]
- Children's wounds are more frequently linear, shorter, more likely to be located on the head, and more often caused by blunt trauma compared with wounds of adults.[6]

PATHOPHYSIOLOGY

- Acute traumatic wounds are caused by either shear, compressive, or tensile forces, which vertically separate the epithelium and dermis.[7]
- Shear forces produced by sharp objects that cut the skin with relatively low energy result in wounds with a straight edge and minimal cell damage or contamination; they heal with good results.
- A blunt object contacting the skin produces compressive and tensile forces. More energy is deposited from these forces, causing disruption of the microvasculature, devitalizing tissue, and creating an anaerobic environment, which supports bacterial proliferation.
- The tensile strength of a wounded area has 50 percent recovery by 40 days and nearly 100 percent recovery by 150 days after injury.
- Stages of wound healing: hemostasis, inflammation, epithelialization, angiogenesis, fibroplasia, wound contracture, and scar remolding.

CLINICAL FEATURES

- Wound repair has been traditionally divided into three categories: primary, secondary, and tertiary closure.
- Primary closure—healing by primary intention—is performed with suture, staples, or adhesives at the time of initial evaluation.
- Secondary closure—healing by secondary intention—the wound is allowed to granulate and fill in, with only cleaning and debridement as needed.
- Tertiary closure—delayed primary closure—the wound is initially cleaned, debrided, and observed for 4 to 5 days before closure.
- Assessing a wound's potential for infection must take into account the mechanism of injury as well as the exogenous and endogenous sources of bacteria.
- The density of bacteria is low over most of the body surface (trunk, upper arms, and legs).
- Moist areas and exposed anatomic areas (head, face, hands, and feet) harbor millions of bacteria.
- Bacteria reside on the most superficial skin layer; topically applied antiseptic agents provide sterility or near sterility, minimizing infection potential.
- Wounds contacting the oral cavity are heavily contaminated with facultative and anaerobic organisms.
- The most common foreign body in a wound is soil.
- Clay-contaminated soils and soils with large amounts of organic material have a high potential for infection.
- Sand and black dirt from highway surfaces have a low potential for infection.

TABLE 14-1 Wounds That Usually Require Consultation

Wounds involving the tarsal plate of the eyelid or lacrimal duct
Wounds involving an open fracture or joint space
Wounds associated with multiple trauma that need surgical admission
Wounds of the face that require extensive plastic reconstruction
Wounds associated with amputation
Wounds associated with loss of function
Wounds that involve tendons, nerves, or vessels
Wounds that involve a significant loss of epidermis

- Animal bite wounds pose a higher risk of infection.
- Wounds that usually require consultation are listed in Table 14-1.

EMERGENCY DEPARTMENT CARE

- Documentation of a wound should include location, size, shape, margins, and depth. When a limb is involved, the sensory, motor, tendon, and vascular integrity of the extremity should be documented.
- Use roentgenograms if any bony tenderness or instability surrounds the wound.
- Foreign bodies that are visible on x-ray include metal, glass, gravel, teeth, and bone larger than 1 mm.
- Foreign bodies not visible on x-ray include plastic, wood, and other organic material.
- Pain control should be provided prior to extensive wound exploration.
- Control of bleeding is necessary for proper wound evaluation and treatment. Direct pressure is usually effective; ligation of minor vessels, chemical means of hemostasis such as epinephrine, or the use of absorbable gelatin sponge (Gelfoam) or oxidized cellulose (Oxycel), may be required.
- Epinephrine should not be used in local anesthetic preparations for repairs involving end-capillary beds, such as fingers, toes, and the tip of the nose or the penis.
- Inspect wounds to their full depth for possible foreign bodies.
- If hair is the foreign body in the wound, it should be clipped and not shaved.[8,9] Shaving can cause an increase in infection.
- High-pressure irrigation will decrease bacterial count and helps remove foreign bodies, thus decreasing infection rate.[8,9]
- Saline solution is an adequate irrigant; there is no further benefit to the addition of povidone-iodine or hydrogen peroxide.[10]
- Wound soaking or scrubbing is not effective in cleaning contaminated wounds.[11]
- Removing devitalized tissue will decrease the risk of infection and will create sharp wound edges that are easier to repair.[8,9]
- Use of antibiotics on most wounds closed in the ED has not been shown to prevent wound infections.[8,9]
- If antibiotics are used, they should be started immediately and ideally prior to tissue manipulation in the ED.
- The most important step in the prevention of a wound infection is adequate irrigation and debridement.
- Tetanus prophylaxis in wound management has been developed by several public and professional organizations. The Centers for Disease Control and Prevention have published guidelines (see Chap. 91).[12]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 35, ''Evaluation of Wounds,'' by Louis J. Kroot, and Chap. 36, ''Wound Preparation,'' by Susan C. Stone and Wallace A. Carter.

15 METHODS FOR WOUND CLOSURE

James F. Palombaro

CLINICAL FEATURES

- Absorbable sutures degrade rapidly, losing all of their tensile strength within 60 days. Nonabsorbable sutures maintain their tensile strength longer than 60 days.
- All sutures compromise local tissue defenses and increase the potential for infection.
- Sutures tied too tightly impair blood flow and cause tissue necrosis of the wound edges.
- Sutures of natural fiber (silk) potentiate infection more than other nonabsorbable sutures and should be avoided in contaminated wounds.
- Synthetic monofilament sutures pose a lower risk of infection than does comparable multifilament material and is the recommended suture material for most percutaneous skin closures.
- Skin closure with staples is quick and economical, with the advantage of low tissue reactivity, leading to a low potential for infection.[1–4] Staples should be used for lacerations with regular skin edges, where the healing scar is not readily apparent (e.g., scalp). Staples should not be used for lacerations with irregular skin edges, since staples do not provide the same meticulous coaptation that can be achieved with sutures.
- Skin closure tapes work best on flat, dry, nonmobile surfaces where the wound edges fit together without tension. They are used as an alternative to sutures and staples and for additional support after suture and staple removal.[4]
- Taped wounds are more resistant to infection than sutured wounds.
- The skin tape should stay in place about as long as an equivalent suture and will spontaneously detach as the underlying epithelium exfoliates.
- Tissue adhesives close wounds by forming an adhesive layer on top of the intact epithelium.
- Never apply tissue adhesives within wounds due to their intense inflammatory reaction with subcutaneous tissue.
- Tissue adhesives should not be applied to mucous membranes, infected areas, joints, areas with dense hair (e.g., scalp), or in wounds exposed to body fluids. They also should not be applied alone on wound edges that are separated by more than 5 mm or longer than 5 cm.
- Tissue adhesives are most useful on wounds that close spontaneously, have clean or sharp edges, and are located on clean, nonmobile areas.
- Once tissue adhesives are applied, they should not be covered with ointment, bandage, or dressing. They should remain dry for 24 h, then they can be gently washed with plain water.

SUTURING TECHNIQUES

- Percutaneous sutures pass through both epidermal and dermal layers and are the most common sutures used in the ED.
- Percutaneous sutures should be placed to achieve eversion of wound edges. They should be used with straight, shallow lacerations only.
- Dermal, or subcuticular, sutures reapproximate the divided edges of the dermis without penetrating the epidermis. Occasionally these sutures and percutaneous sutures are used together in a layered closure.
- The following principles are used with deep, irregular wounds with uneven, unaligned, or gaping edges:
  1. Wounds where the edges cannot be brought together without excessive tension should have dermal sutures placed to partially close the gap.
  2. Adipose tissue beneath the skin should not be sutured, as obliteration of this potential dead space can increase the incidence of infection.
  3. When wound edges of different thickness are to be reunited, the needle should be passed through one side of the wound and then drawn out before reentry through the other side so

as to ensure that the needle is inserted at a comparable level.

4. Uneven edges can be aligned by first approximating the midportion of the wound with the initial suture. Subsequent sutures are placed in the middle of each half until the wound edges are aligned and closed.

- Continuous ''running'' sutures are best when linear wounds are being repaired. An advantage of this suture is that it accommodates to the developing edema of the wound edges during healing. However, a break in the suture may ruin the entire repair.
- Dermal (subcuticular) sutures can be used alone or as adjuncts to percutaneous sutures in wounds subject to strong skin tensions. If they are used alone, it is advisable to close the skin with surgical tape or wound adhesive for accurate approximation of the epidermis.
- Vertical mattress sutures are useful in areas of lax skin (the elbow and dorsum of the hand), where the wound edges tend to fold into the wound. They act as an all-in-one suture, avoiding the need for a layered closure.
- Horizontal mattress sutures are faster and better at eversion of skin edges than vertical mattress sutures. They are useful in areas of increased tension, such as fascia, joints, and callus skin.
- A purse-string suture is useful in reapproximating multiple flap tips and corner wounds. It is used in these areas in order to preserve the blood supply and minimize tissue destruction at the tips of the skin edges.
- The dog-ear maneuver is a technique used to handle excess tissue at one end of a wound. The wound is extended from the apex toward the long side in the form of a hockey stick. Then the triangular piece of excess skin is removed and the edges are sewn together.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 37, ''Methods for Wound Closure,'' by Julia E. Martin and Rob Herfel.

16 LACERATIONS TO THE FACE AND SCALP

David M. Cline

EPIDEMIOLOGY

- Each year, more than 12 million wounds are treated in emergency departments (EDs) across the United States.[1] The most cosmetically devastating are those that appear on the face.
- Anyone with facial trauma should be questioned about the possibility of domestic violence; if this is strongly suspected, appropriate authorities should be notified. Prompt identification and intervention are critical in preventing future injury.[2]

PATHOPHYSIOLOGY

- Facial and scalp wounds are most often caused by a combination of sharp and blunt mechanisms. It takes an average of 10 times fewer bacteria to cause an infection in a blunt wound than it would in a sharp wound.

SCALP AND FOREHEAD

ANATOMY

- The arterial supply to each side of the scalp involves three branches off the external carotid artery (occipital, superficial temporal, and posterior auricular arteries) and two branches from the internal carotid artery (supraorbital and supratrochlear arteries).[3]

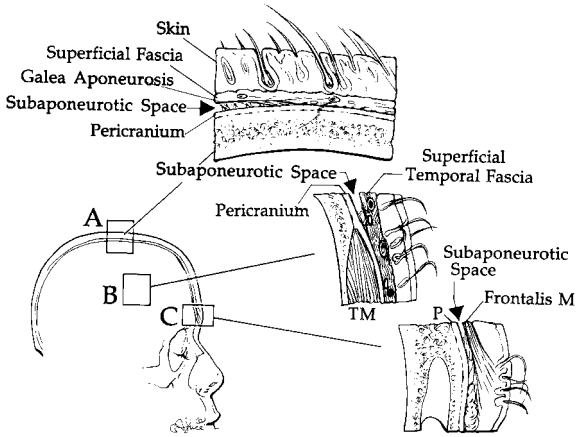

FIG. 16-1 The layers of the scalp and forehead.

- The scalp and forehead (which includes eyebrows) are parts of the same anatomic structure (Fig. 16-1).

EVALUATION

- Wounds that fall along the lines of skin tension have better cosmetic results. Skin tension lines are always perpendicular to the underlying muscles.

WOUND PREPARATION

- There are few data to support the belief that epinephrine reduces bleeding during wound repair. Conversely, the theoretical adverse effects of added epinephrine (increased risk of infection, ischemia of portions of the wound with poor circulation, and cardiovascular effects of epinephrine) are rarely an issue with facial and scalp lacerations.
- In nonbite, noncontaminated facial and scalp wounds presenting within 6 h, routine irrigation does not alter the rate of infection or subsequent cosmetic appearance after suture repair.[4]
- Eyebrows should never be clipped or shaved because their delicate contour and form are valuable landmarks for the meticulous reapproximation of the wound edges.

REPAIR OF SCALP LACERATIONS

- It is not necessary to shave the scalp prior to closure; shaving actually increases the likelihood of a wound infection and produces a less desirable cosmetic result in the short term.
- When the edges of a laceration of either the eyebrow or the scalp are devitalized, debridement is mandatory. When debriding these sites, the scalpel should cut an angle that is parallel to that of the hair follicles.
- Wound closure should be initiated first with approximation of the galea aponeurotica with buried, interrupted absorbable 4-0 sutures.
- The divided edges of muscle and fascia must also be closed with buried, interrupted, absorbable 4-0 synthetic sutures to prevent further development of depressed scars.
- The skin can be closed by staples or by simple interrupted nylon sutures (consider using sutures that are a different color than the patient's hair). Some authors recommend single-layer closure with 3-0 nylon sutures.
- The use of staples saves money[5] and is associated with a lower in section rate than the use of sutures for scalp laceration repair.[6]

REPAIR OF FOREHEAD LACERATIONS

- The epidermal layer can be closed with 6-0 nonabsorbable nylon in a simple, interrupted fashion; with wound closure strips over tincture of benzoin; or with tissue adhesive.[7,8]
- The skin edges of anatomic landmarks on the forehead should be approximated first with key stitches, using interrupted, nonabsorbable monofilament 5-0 synthetic sutures (Fig. 16-2).
- Accurate alignment of the eyebrow, transverse wrinkles of the forehead, and the hairline of the

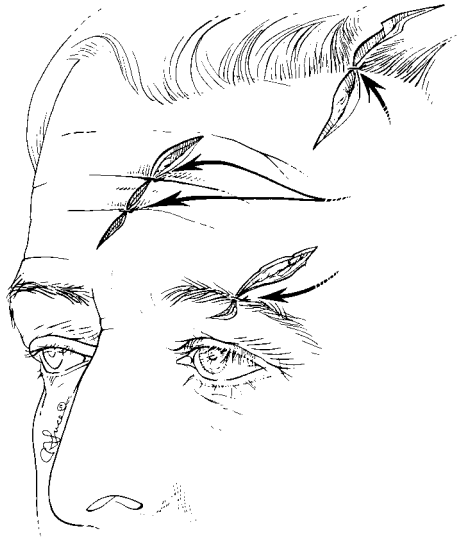

FIG. 16-2 Key stitches in the forehead.

scalp is essential. It may be necessary to have young patients raise their eyebrows to create wrinkles for accurate placement of the key stitches.

EYELIDS

- A complete exam of the eye's structure and function is essential. A search should be made for foreign bodies (see Chap. 147).
- The lid should be examined for involvement of the canthi and the lacrimal system or penetration through the tarsal plate or lid margin.
- The following wounds should be referred to an ophthalmologist: (*a*) those involving the inner surface of the lid, (*b*) those involving the lid margins, (*c*) those involving the lacrimal duct, (*d*) those associated with ptosis, and (*e*) those that extend into the tarsal plate.
- Failure to recognize and properly repair the lacrimal system can result in chronic tearing.
- Uncomplicated lid lacerations can be readily closed using nonabsorbable 6-0 suture. Tissue adhesive is contraindicated near to the eye.

NOSE

- Lacerations of the nose may be limited to skin or involve the deeper structures (sparse nasal musculature, cartilaginous framework, and nasal mucous membrane). They are repaired by accurate reapproximation of each tissue layer.
- Local anesthesia of the nose can be difficult because of the tightly adhering skin. Topical anesthesia may be successful with lidocaine, epinephrine, and tetracaine.
- When the laceration extends through all tissue layers, closure should begin with a marginal, nonabsorbable, monofilament 5-0 synthetic suture that aligns the skin surrounding the entrances of the nasal canals, to prevent malposition and notching of the alar rim.
- Traction upon the long, untied ends of the marginal suture approximates the wounds and aligns the anterior and posterior margins of the divided tissue layers.
- The mucous membrane should then be repaired with interrupted, braided, absorbable 5-0 synthetic sutures, with their knots buried in the tissue. The area is reirrigated gently from the outside.
- The cartilage may rarely need to be approximated with a minimal number of 5-0 absorbable sutures. In sharply demarked linear lacerations, closure of the overlying skin is usually sufficient.
- The cut edges of the skin, with its adherent musculature, are closed with interrupted, nonabsorbable, monofilament 6-0 synthetic sutures. Removal of the external sutures may take place in 3 to 5 days.
- Following any nasal injury, the septum should be inspected for hematoma formation using a nasal speculum. The presence of bluish swelling in the septum confirms the diagnosis of septal hematoma. Treatment for the hematoma is evacuation of the blood clot.
- Drainage of a small septal hematoma can be accomplished by aspiration of the blood clot through a #18 needle. A large hematoma should be drained through a horizontal incision at the base. Bilateral hematomas should be drained in the operating room by a specialist.
- Reaccumulation of blood can be prevented by nasal packing. Antibiotic treatment (penicillin) is recommended to prevent infection that may cause necrosis of cartilage.

LIPS

- Isolated intraoral lesions may not need to be sutured.
- Through-and-through lacerations that do not include the vermilion border can be closed in layers. The 5-0 absorbable suture should be used first for the mucosal surface; then reirrigation; and closure of the orbicularis oris muscle with 5-0 absorbable suture. The skin should be closed with 6-0 nylon suture or tissue adhesive.
- Repair of a complex lip laceration requires a three-layered closure (Fig. 16-3). Using skin hooks, traction is applied to align the anterior and

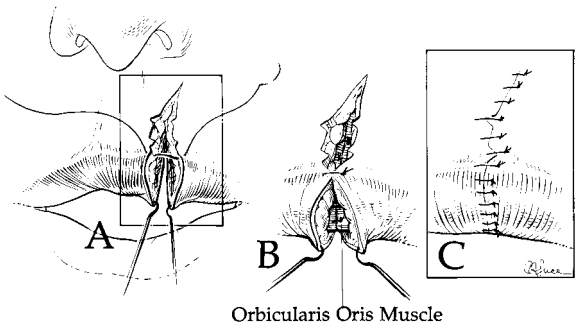

FIG. 16-3 Irregular-edged vertical laceration of the upper lip. **A.** Traction is applied to the lips, and closure of the wound is begun first at the vermillion-skip junction. **B.** The orbicularis oris muscle is then repaired with interrupted, absorbable 4-0 synthetic sutures. **C.** The irregular edges of the skin are then approximated.

posterior borders of the laceration. Closure of the wound should start at the vermilion-skin junction with a nonabsorbable, monofilament 6-0 synthetic suture. The orbicularis oris muscle is then repaired with interrupted, braided, absorbable 4-0 synthetic sutures. The vermilion-mucous membrane junction is approximated with a braided, absorbable 5-0 synthetic suture. The suture ligature's knot is buried in the subcutaneous tissue. The divided edges of the mucous membrane and vermilion are then closed using interrupted, braided, absorbable 5-0 synthetic sutures with a buried-knot construction.

- Skin edges of the laceration are usually jagged and irregular, but they can be fitted together as the pieces of a jigsaw puzzle using interrupted, nonabsorbable, monofilament 6-0 synthetic sutures with their knots formed on the surface of the skin.

CHEEKS AND FACE

- Facial lacerations are closed with 6-0 nonabsorbable; simple interrupted sutures and are removed after 5 days. Tissue adhesive is an alternative.
- Attention to anatomic structures including the facial nerve and parotid gland is necessary (see Fig. 16-4). If these structures are involved, operative repair is indicated.

EAR

- Superficial lacerations of the ear can be closed with 6-0 nylon suture.

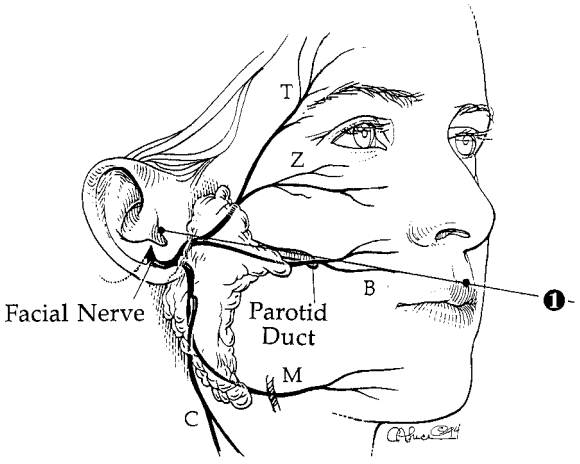

FIG. 16-4 Anatomic structures of the cheek. The course of the parotid duct is deep to a line drawn from the tragus of the ear to the midportion of the upper lip.

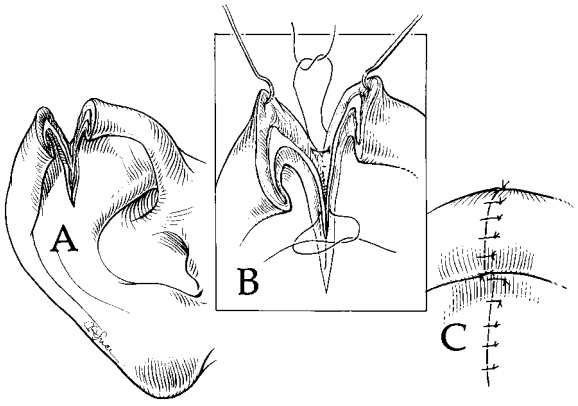

FIG. 16-5 A. Laceration through auricle. **B.** One or two interrupted, 6-0 coated nylon sutures will approximate divided edges of cartilage. **C.** Interrupted nonabsorbable 6-0 synthetic sutures approximate the skin edges.

- Exposed cartilage should be covered. Debridement of the skin is not advisable, since there is very little excess skin. In most through-and-through lacerations of the ear, the skin can be approximated and the underlying cartilage will be supported adequately (see Fig. 16-5).
- Following repair of simple lacerations, a small piece of nonadherent gauze may be applied over the laceration only and a pressure dressing applied. Gauze squares are placed behind the ear to apply pressure, and the head is wrapped circumferentially with gauze.
- Sutures should be removed in 5 days.
- An otolaryngologist or plastic surgeon should be consulted for more complex lacerations, ear avulsions, or auricular hematomas.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 38, "Lacerations to the Face and Scalp," by Wendy C. Coates.

17 FINGERTIP AND NAIL INJURIES

Martin J. Carey

EPIDEMIOLOGY

- The areas distal to the insertion of the extensor and flexor tendons are the most frequently injured parts of the hand.

PATHOPHYSIOLOGY

- Injuries may involve skin, pulp tissue, distal phalanx, or perionychium (nail, nail bed, and surrounding structures).
- See Fig. 17-1 for the anatomy of the perionychium.

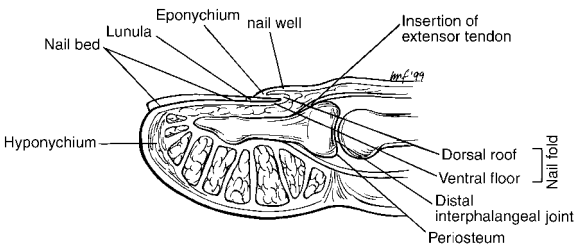

FIG. 17-1 Anatomy of the perionychium. [From Zook EG: The perionychium, in Green DP (ed): *Operative Hand Surgery,* 2d ed. New York, Churchill Livingstone, 1988, p 1332, with permission.]

CLINICAL FEATURES

- Most often injuries are isolated.
- Types of injury include closed crush, simple lacerations, open crush with or without partial amputation, and complete amputation.[1]
- Assess handedness, patient's occupation, number of digits injured, patient's age and gender, and tetanus prophylaxis status.[2]

DIAGNOSIS AND DIFFERENTIAL

- Always assess for other injuries.
- X-rays are frequently indicated.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Basic goals are to preserve finger length and cosmetic appearance, approach normal sensation and function, and heal in as rapid and uncomplicated manner as possible.
- Most injuries can be managed in the emergency department.
- Consultation with a plastic or hand surgeon is required with complex or extensive injuries, injuries requiring skin grafting, or those requiring technically demanding skills. Consultation with a specialist is also recommended if the hand is vital to the patient's career—for example, if the patient is a professional musician.
- All wounds are considered contaminated; scrupulous cleaning and irrigation are essential after adequate anesthesia, usually by means of a digital nerve block.
- Distal fingertip amputations with skin or pulp loss only are best managed conservatively, with serial dressing change only,[3] especially in children.[4]
- In cases with larger areas of skin loss (more than 1 cm^2) a skin graft, either using the severed tip itself or skin harvested from the hypothenar eminence, may be required.[1]
- Complications of the skin graft technique include decreased sensation of the fingertip, tenderness at the injury and graft site, poor cosmetic result, and hyperpigmentation in dark-skinned patients.
- Injuries with exposed bone are not amenable to skin grafting. Most of these injuries require specialist advice. If less than 0.5 mm of bone is exposed and the wound defect is small, the bone may be trimmed back and the wound left to heal by secondary intention. Injuries to the thumb or

index finger with exposed bone nearly always require specialist attention.
- Injuries to the nail bed require careful repair to reduce scar formation. They are associated with fractures of the distal phalanx in 50% of cases.
- Subungual hematomas require decompression by simple trephination of the nail plate.[5,6] Use of heated paper clip delays healing.[7] Use of nail drill, scalpel, or 18-gauge needle is recommended.
- Nail removal is needed if there is extensive crush injury, associated nail avulsion or surrounding nail fold disruption, or a displaced fracture of the distal phalanx on x-ray. The nail bed is inspected and repaired using 6 or 7/0 absorbable sutures. If the nail matrix is displaced from its anatomic position at the sulcus, the matrix should be carefully replaced and held in place with mattress sutures.
- If there is extensive injury to the nail bed with avulsed tissue, specialist consultation is required.[8]
- In children with fractures of the distal phalanx, the nail plate may come to lie upon the eponychium. In these cases, after careful cleaning and adequate anesthesia, the nail plate should be replaced under the proximal nail fold.
- Ring removal from all injured fingers is required. Swelling may require a ring to be cut off. If slower techniques are appropriate, simple lubrication may suffice. The string technique is an alternative method. String, umbilical tape, or 0-gauge silk may be used. The string is passed under the ring, then wrapped firmly around the finger from proximal to distal. The proximal end of the string is then gently pulled, and the ring advances down the finger.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 39, ''Fingertip and Nail Injuries,'' by Robert S. Chang and Wallace A. Carter.

18 LACERATIONS OF THE EXTREMITIES AND JOINTS

Martin J. Carey

EPIDEMIOLOGY

- More than 12 million traumatic lacerations are treated annually across the United States.[1]
- Injuries to the foot are common and may be devastating. Classic causes of foot injury include broken glass, lawn mowers, bicycle spokes, and high-pressure water hoses.[2–4]

PATHOPHYSIOLOGY

- Wounds may be caused by blunt or sharp mechanisms.
- Blunt objects tend to cause ragged wounds that are difficult to close. An underlying fracture is possible. These wounds are more susceptible to infection than wounds caused by sharp mechanisms.[5]
- Contamination of wounds by dirt, chemicals, or foreign bodies increases the risk of infection, slows healing, and may result in a less cosmetically attractive scar.

CLINICAL FEATURES

- A limited, injury-specific history is appropriate. Details of the time, mechanism, and exact position of the extremity at time of injury should be ascertained. The possibility of a foreign body, altered sensation, or weakness should be considered.
- The position, shape, size, and depth of the laceration should be accurately recorded.

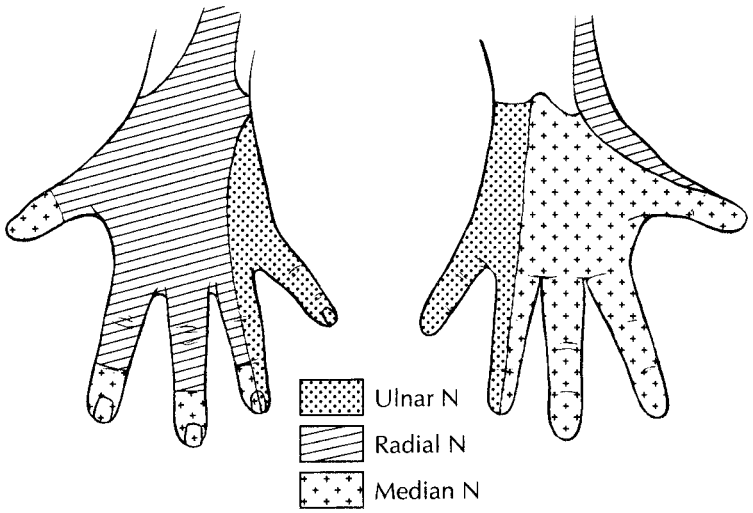

FIG. 18-1 Sensory innervation to the hand.

- Careful examination for distal sensory loss and blood supply is required (Figs. 18-1 and 18-2).
- The function of all tendons potentially injured by the laceration must be assessed and recorded.[6] Full motor function must also be assured (Tables 18-1 and 18-2).

DIAGNOSIS AND DIFFERENTIAL

- Laboratory studies are usually not indicated.
- Radiology is required if there is a possibility of fracture or of a radiopaque foreign body. All injuries caused by glass should be x-rayed.[6]

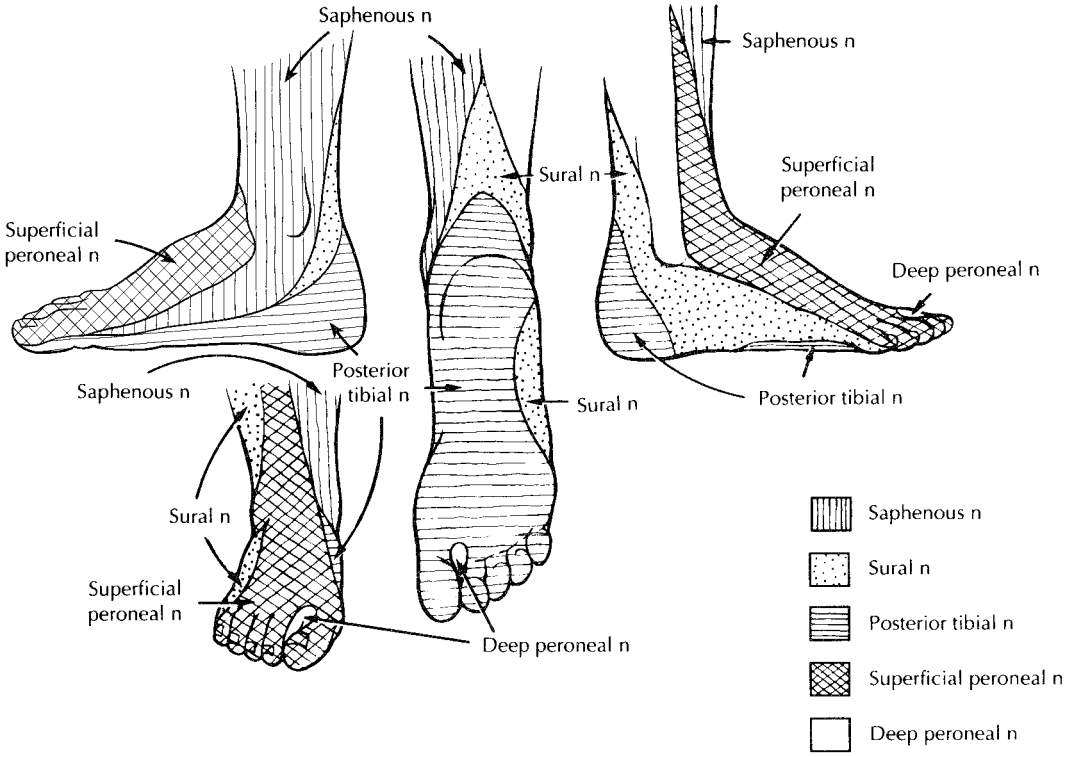

FIG. 18-2 Sensory innervation to the foot.

TABLE 18-1 Motor Function of Peripheral Nerves

NERVE	MOTOR FUNCTION
Radial	Wrist extension Digit extension
Ulnar	Finger abduction Finger adduction Thumb adduction
Median	Thumb flexion Thumb opposition Thumb abduction
Superficial peroneal	Foot eversion
Deep peroneal	Foot inversion Ankle dorsiflexion
Tibial	Ankle plantar flexion

- Suspected foreign bodies that are not radiopaque may be visualized by ultrasound, computed tomography, or magnetic resonance imaging.[7]
- Injuries over joints should also be evaluated for possible penetration of the joint capsule. If this is a consideration, radiography may reveal air in the joint. An alternative approach is to inject sterile saline, with or without a few drops of sterile fluorescein, into the joint, using a standard joint aspiration technique at a site separate from the laceration. Leakage of the solution from the wound indicates joint capsule injury.[8]

TABLE 18-2 Tendon Function of the Upper and Lower Extremities

TENDON	MOTOR FUNCTION
Flexor digitorum profundus	DIP joint flexion
Flexor digitorum superficialis	PIP joint flexion
Flexor carpi ulnaris	Flexion at wrist with ulnar deviation
Flexor carpi radialis	Flexion at wrist with radial deviation
Extensor carpi ulnaris	Extension at wrist with ulnar deviation
Extensor carpi radialis	Extension at wrist with radial deviation
Extensor digitorum communis	Extension of digits 2–5
Flexor pollicis longus	Thumb flexion
Extensor pollicis longus	Thumb extension at DIP
Extensor pollicis brevis	Thumb extension at MCP
Abductor pollicis longus	Thumb abduction
Extensor hallicis longus	Great toe extension with ankle inversion
Tibialis anterior	Ankle dorsiflexion and inversion
Achilles tendon	Ankle plantar flexion and inversion

ABBREVIATIONS: DIP, distal interphalangeal; PIP, proximal interphalangeal; MCP, metacarpophalangeal.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Attention to life-threatening injuries always takes priority. If necessary, a nonadherent dressing can be applied to non-life-threatening injuries and repair delayed until the patient is stabilized.
- Tetanus immunization status should always be considered. The elderly are at particular risk for being nonimmunized.[9]
- If the pattern of injury suggests assault, the possibility of abuse (child, spouse, or elder) should be considered. Patterns suggesting abuse are injuries over the midparts of long bones, injuries of various ages, or injuries that do not appear to be compatible with the mechanism stated.
- Injury over the wrist raises the possibility of a suicide attempt.
- Multiple parallel lacerations over the wrist may be repaired as noted in Fig. 18-3.
- Injuries to the palm may require a regional anesthetic—for example, a median or ulnar nerve block. Very careful exploration is mandatory. If no deep injury is suspected, the wound is closed,

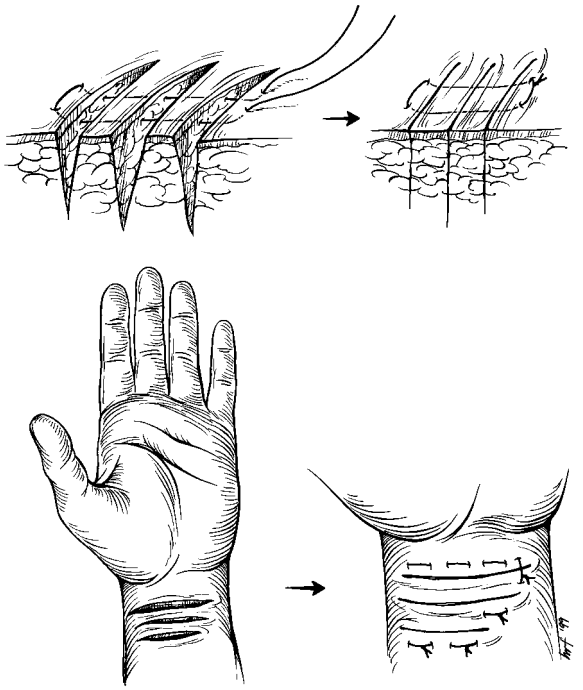

FIG. 18-3 Horizontal mattress sutures for multiple parallel lacerations.

paying particular attention to reopposing the skin creases accurately. Care should be taken to avoid using deep ''bites'' with the needle, as this risks injury to the underlying tendons or tendon sheaths.

- Deep injuries between the carpometacarpal joints and the distal creases of the wrist are considered to be in ''no man's land'' and should be referred to a specialist for exploration and repair.
- Injuries to flexor tendons must be referred to a specialist. The repair can be delayed. In these cases the wound should be cleaned, the skin repaired, the limb splinted in a position of function, and arrangements made for follow-up within 3 days with a hand surgeon.
- On the dorsum of the hand, lacerations over the metacarpophalangeal joint suggest a closed-fist injury (see Chap. 20).
- With the exception of the tendons to the thumb, experienced emergency physicians may repair extensor tendon injuries over the dorsum of the hand.[10] Otherwise the tendon injury should be discussed with a hand specialist. Usually a ''figure of eight'' knot is used, with a 4-0 nonabsorbable suture material. The limb is then splinted.
- Lacerations to the extensor tendons over the distal interphalangeal joint produce a mallet deformity, while lacerations over the proximal interphalangeal joint produce a boutonniere deformity. If the lacerations are open, they are surgically repaired; if closed, they are splinted for up to 6 weeks.[11]
- Lacerations to the lower limb are usually under more tension than upper limb injuries. These wounds are typically repaired in layers, with 4-0 absorbable material to the deep layers and 4-0 nonabsorbable to the skin. Deep sutures should be avoided in patients with diabetes or with stasis changes. In these patients, deep mattress sutures are a satisfactory alternative.
- The integrity of the Achilles tendon can be assessed by the Thompson test. The belly of the gastrocnemius is squeezed while the patient kneels on a chair. An intact Achilles tendon produces plantar flexion of the foot.
- Injuries to the foot are repaired in a similar manner to injuries elsewhere. Because of the high risk for infection, wounds older than 6 h at presentation should probably not be repaired primarily. If the wound is to be repaired, heavy, large needles are required to penetrate the thick dermis of the sole. The presence of foreign bodies in the foot should always be considered. They can be very difficult to find, and orthopedic assistance may be required.
- Hair strangulation is an unusual condition of infants. A strand of hair becomes wrapped, often many times, around the base of a toe. This results in vascular compromise and death of the digit if not identified. All strands must be removed in order save the toe. An incision along the extensor surface and down to the extensor tendon is a frequently used approach.
- Between 18 and 34 percent of foot lacerations become infected. Antibiotic prophylaxis should be considered.[2,12] Wounds caused while wading in fresh water are prone to infection with *Aeromonas.*[13] In these cases a fluoroquinolone antibiotic is required except in children, when trimethoprim-sulfamethoxazole is a better choice. *Aeromonas* should be considered in any rapidly progressive case of cellulitis in the foot after an injury.
- After repair, wounds should be kept clean and dry for 24 h. Sutures should be removed after 7 to 10 days with upper extremity injuries, 10 to 14 days for lower extremity injuries, and 14 days for wounds over joints. These times are extended by 2 to 3 days in older persons, as for them wound healing is delayed.
- Wounds should be rechecked after 48 h if they were heavily contaminated or if a complex repair was required.
- Patients should all be instructed in wound care and told to be alert for the signs of infection or compartment syndrome.

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19 SOFT TISSUE FOREIGN BODIES

Martin J. Carey

EPIDEMIOLOGY

- The potential for foreign bodies should be considered in all fresh wounds and in old wounds with evidence of infection or delayed healing.[1] However, only a small proportion of all wounds will contain a foreign body.

PATHOPHYSIOLOGY

- Foreign bodies in wounds produce an intensification of the normal inflammatory response to injury. This may result in delayed healing and destruction of surrounding soft tissue and bone.
- If the body fails to dissolve or extrude the foreign body, eventually it will be surrounded by a fibrous capsule, or granuloma. At this point inflammation will subside.[1]
- The degree of inflammation is related to the composition of the foreign body. Inert materials—glass, plastics, and most metals—may produce little inflammation, while vegetative matter will often produce an intense reaction. Some reactions may be secondary to volatile oils in the material—for example, with cedar wood, or to the presence of alkaloids, as in blackthorns. Some marine organisms have venom on their spines, and the introduction of these into the skin produces a severe reaction.[2]
- Infection is a common complication of foreign bodies.[3] Complications can include local wound infection, cellulitis, abscess formation, lymphangitis, tenosynovitis, bursitis, and osteomyelitis. Characteristically, these infections are resistant to therapy or show an initial response but then recur.

CLINICAL FEATURES

- History is very important. Objects that break, shatter, or splinter in the course of causing the injury are particularly likely to produce a foreign body. Penetrating injuries through clothing or shoes can thrust foreign material deep into the wound.
- Although not all foreign bodies are perceived by the patient, patients who state that they feel a foreign body need to be taken very seriously.
- Patients may present with a healed wound but with a sensation of something pricking or catching under the skin, or with pain produced on pressure over the wound. A foreign body must be suspected in these cases.
- Physical examination may produce obvious evidence of a foreign body, such as a palpable mass, a hard object under the skin, or something grating against an exploring forceps. Adequate lighting and effective hemostasis are vital when wounds are being explored for potential foreign bodies.

DIAGNOSIS AND DIFFERENTIAL

- Radiography is indicated if the potential foreign body is radiopaque.[4] If it is not, computed tomography, magnetic resonance imaging, or ultrasound may be required.
- ''Soft tissue'' radiography should identify 98 percent of radiopaque objects.[4–6] Projections in different planes will help in identifying the position of the foreign body.
- CT scanning is 100 times more sensitive at differentiating densities. However, it may not identify some thorns, spines, or certain types of wood.[7,8]
- Ultrasound is gaining favor in foreign body identification. Depending upon the experimental

method, sensitivity for the detection of foreign bodies is between 30 and 100 percent, while specificity is between 70 and 90 percent.[9–11] Variation is due to the size and sonographic nature of the foreign body, the presence of confounding objects, and the skill and experience of the operator. Ultrasound appears promising in guiding the retrieval of foreign bodies.[12]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Not all foreign bodies need to be removed at the initial emergency department visit. Examples may include bullets deep in muscle or small inert objects. However, their presence should be recorded.
- Vegetative material and heavily contaminated objects should always be removed, as should small particulate matter such as road grit, which can produce disfiguring wounds with tattooing.
- Long thin objects, like needles, may be difficult to locate. Incision over the midpoint of the needle may facilitate grasping the object in a hemostat and then pushing it back through the entrance hole. Sometimes, fluoroscopy may be required to find these foreign bodies.
- Splinters need to be carefully removed, as they can leave smaller pieces behind. If possible, the wound should be opened along its length and cleaned out after the splinter has been removed.
- Splinters under the nail are removed by taking a triangular piece of nail from over the splinter, so that the object can be grasped and carefully extracted.
- Cactus spines can be removed with forceps or utilizing an adhesive. Excision may be required if they are deeply imbedded.[13]
- Fishhooks are removed by a variety of methods. A loop of string looped around the hook is firmly and sharply tugged while pressure is placed downward on the shank of the hook. Alternatively, a needle is used to cover the barb of the hook and the hook is withdrawn through the skin. Alternatively, the hook is pushed out through the skin, the barb is cut off, and the hook is then pulled back. Whichever method is used, the skin around the hook should be anesthetized prior to the procedure.
- After foreign body removal, the wound is irrigated. If the wound is contaminated, the wound edges may have to be excised. If multiple radiopaque foreign bodies were removed, a repeat x-ray is indicated to ensure that all foreign bodies have been removed. In general, unless all foreign contamination can be removed, the wound should be left open and closed secondarily. Tetanus prophylaxis should be ensured. If a foreign body is left in place, the patient should be informed.
- If delayed removal is required, the patient should be ensured of the safety of the practice. Limb splinting is needed if the foreign body is over a joint. Antibiotics are given if the wound is infected.

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20 PUNCTURE WOUNDS AND ANIMAL BITES

Chris Melton

PUNCTURE WOUNDS

PATHOPHYSIOLOGY

- The most common sequela of puncture wounds is infection. Gram-positive organisms predominate, especially *Staphylococcus aureus.*
- If the wound is over a joint, it may result in a septic joint.
- Osteomyelitis may occur if the puncture involves bone. The most common organism is *Pseudomonas aeruginosa,* especially if the puncture occurs through the rubber sole of an athletic shoe.[1,2]
- Postpuncture wound infections may be secondary to a retained foreign body. These infections are frequently refractory to antibiotic therapy until the foreign body is removed.
- Postpuncture wound infections are more common in patients with decreased resistance to infection, including those with diabetes mellitus, peripheral vascular disease, and immunosuppression.[3]
- The following result in increased risk of postpuncture wound infection: more than 6 h from injury to presentation, larger wounds with deeper penetration, obvious contamination, occurrence outdoors, puncture through footwear, and puncture involving the forefoot.[4]
- Cellulitis is usually secondary to *Staph. aureus* and may be treated with dicloxacillin, a first-generation cephalosporin, or a fluoroquinolone.

CLINICAL FEATURES

- The wound must be assessed for damage to underlying structures, including tendons and distal nerves and vessels.
- The presence of foreign bodies must be excluded, including the use of radiographs of infected wounds and wounds suspicious for a retained foreign body (see Chap. 14).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Uncomplicated wounds and wounds less than 6 h old may be managed with irrigation and tetanus prophylaxis as indicated. Prophylactic antibiotics are not indicated in healthy, immunocompetent patients.
- Patients with diabetes mellitus, peripheral vascular disease, and immunosuppression may benefit from prophylactic antibiotics.[3]
- Plantar wounds, especially those located in the forefoot and those due to puncture through an athletic shoe, should receive prophylactic antibiotics.[5] Treatment should be for 5 to 7 days. A fluoroquinolone should be used in adults. In children and those in whom fluoroquinolones are contraindicated, cephalexin should be used.
- Wounds may present already infected. There may be cellulitis, abscess, chondritis, and osteomyelitis. Radiographs should be performed to evaluate for foreign bodies as well as gas in the soft tissues and osteomyelitis.
- If an abscess develops at the puncture site, incision and debridement are indicated and may point to the presence of a foreign body.
- If outpatient oral antibiotic therapy fails or there is a relapse, osteomyelitis or septic arthritis may have developed. Orthopedic consultation may be required for incision and debridement. After cultures are obtained, empiric therapy covering *Staphylococcus* and *Pseudomonas* should be initiated with nafcillin and ceftazidime as one possible regimen.
- Admission may be required for those patients with wound infections who have diabetes, peripheral vascular disease, immunosuppression, progressive cellulitis, or foreign bodies requiring operative removal.
- Outpatients should be rechecked in 48 h and advised to avoid weight bearing, to elevate the limb, and to soak the wound in warm water.

NEEDLE-STICK INJURIES

- Bacterial infection as well as infection with hepatitis and HIV are risks associated with needlestick injuries.

- Hospitals should have official protocols, designed by infectious disease specialists, for dealing with these injuries.

INJURIES DUE TO HIGH-PRESSURE INJECTION OF LIQUID

- These injuries result in severe inflammation; they cause severe pain with minimal swelling. The hand or foot is usually involved.[6]
- Parenteral analgesia is indicated; however, digital blocks should not be performed because of the risk of increasing tissue pressure and further compromising tissue perfusion.
- A hand specialist or orthopedist should be consulted for early surgical debridement.[7]

Bites and Scratches

HUMAN BITES

EPIDEMIOLOGY

- Human bites most commonly occur on the hands and upper extremities.
- Clenched-fist injuries (CFIs) are among the most serious types of human bite wounds.

CLINICAL FEATURES

- CFIs occur when teeth puncture the metacarpophalangeal joint region.[8]
- Physical examination should include a thorough evaluation of the underlying structures, particularly the extensor tendon.
- The tendon should be evaluated after local anesthesia with a digital block; this should be performed throughout the range of motion of the involved digit.
- Another potential complication is a bite involving the joint space.
- Radiographs should be performed to evaluate for fractures and foreign bodies.
- Other complications include cellulitis, lymphangitis, abscess, tenosynovitis, septic arthritis, and osteomyelitis.
- Common organisms involved include *Streptococcus viridans, Staphylococcus epidermidis, Staph. aureus,* as well as anaerobic bacteria including *Fusobacterium* and *Bacteroides.*
- Human bites that are not on the hand have rates of infection similar to nonhuman bite lacerations.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Wound irrigation as well as debridement of devitalized tissue should be performed.
- Hand wounds should be left open. Primary closure can be performed on wounds at other sites unless they are deemed at high risk for infection.
- Prophylactic antibiotics are indicated for hand bites and bites at other locations in high-risk patients such as those with HIV, diabetes mellitus, or immunosuppression. Amoxicillin/clavulanate or a fluoroquinolone may be used.
- CFI wounds should be left open. Prophylactic antibiotics should be initiated, and the hand should be immobilized and elevated for 24 h. The wound should be rechecked in 1 to 2 days.[8,9] A hand specialist should be consulted if there is tendon laceration, joint-space involvement, or bony abnormality.[10]
- Wounds that present already infected should be cultured and systemic antibiotics started. In healthy, reliable patients, a local cellulitis may be managed on an outpatient basis with close follow-up. More extensive cellulitis requires surgical consultation and parenteral antibiotics, including ampicillin/sulbactam or cefoxitin. Clindamycin plus ciprofloxacin may be used in penicillin-allergic patients.
- Tetanus immunization should be administered as indicated.

DOG BITES

CLINICAL FEATURES

- Radiographs should be obtained if there is evidence of infection, bony involvement, or suspicion of a foreign body.
- Infections are usually polymicrobial, including both aerobic and anaerobic bacteria, with *Staph. aureus, P. multocida, Eikenella corrodens, Actinomyces,* and *Bacteroides* being frequent isolates.
- *Capnocytophaga canimorsus* has been associated

with severe infections in immunocompromised patients, resulting in sepsis, disseminated intravascular coagulation (DIC), renal failure, and cardiopulmonary failure.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- All wounds should receive copious irrigation and debridement of devitalized tissues.
- Primary closure may be performed on all lacerations except those involving the hands and feet, which should be left open initially.
- Surgical exploration and repair may be needed for large lacerations.
- Puncture wounds, wounds involving the hands and feet, and wounds in high-risk patients should receive prophylactic antibiotics. Amoxicillin/clavulanate or clindamycin plus ciprofloxacin or clindamycin plus trimethoprim/sulfamethoxazole in children are appropriate choices.
- In high-risk patients—including those with asplenia, chronic alcohol use, or chronic lung disease—prophylactic antibiotics should be administered to cover *C. canimorsus.* Penicillin is the drug of choice for *C. canimorsus.* Cephalosporins, tetracyclines, and clindamycin are possible alternatives in the penicillin-allergic patient.
- Wounds infected at presentation should be cultured and antibiotics administered. Low-risk patients with local cellulitis may be treated as outpatients with oral antibiotics and close follow-up. If the infection develops within 24 h, *P. multocida* is usually the causative organism. The treatment of choice for *P. multocida* is penicillin, with alternatives including ciprofloxacin and trimethoprim/sulfamethoxazole. If the infection develops after 24 h, *Streptococcus* and/or *Staphylococcus* are usually the causative organisms and may be treated with dicloxacillin or a first-generation cephalosporin.
- Extensive infections require admission for parenteral antibiotics. Examples include lymphangitis, lymphadenitis, tenosynovitis, septic arthritis, and osteomyelitis. Radiographs should be obtained to evaluate for foreign bodies, bony injury or osteomyelitis, and soft tissue air. Wound cultures should be obtained, and irrigation and debridement may be required in the operating room. Initial antibiotic therapy should include ampicillin/sulbactam or clindamycin plus ciprofloxacin.
- Tetanus immunization should be administered as indicated.

CAT BITES

EPIDEMIOLOGY

- Approximately 80 percent of cat bites become infected because of the higher incidence of *P. multocida* in feline oral flora and because of the increased likelihood of puncture wounds.

CLINICAL FEATURES

- *P. multocida* is the most common pathogen in infected cat bites.[11,12]
- *P. multocida* develops rapidly and is often present at the time of the patient's presentation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- All wounds should receive copious irrigation and debridement of devitalized tissues.
- Primary closure can be performed except when the wound is less than 1 to 2 cm in size or is a puncture wound. In cosmetically significant wounds, delayed primary closure may be performed.
- Patients with punctures to the hand, immunocompromised patients, and patients with arthritis or prosthetic joints should receive prophylactic antibiotics. Antibiotic choices include amoxicillin/clavulanate, cefuroxime, or doxycycline. The duration of antibiotic therapy should be 3 to 5 days.
- For patients with cat bites who present with an infected wound, management is similar to that for dog bites. Penicillin is the antibiotic of choice for *P. multocida.*
- Tetanus immunization should be administered as indicated.

CAT-SCRATCH DISEASE

CLINICAL FEATURES

- This condition is characterized by persistent regional lymphadenopathy in an area that drains the site of a recent cat scratch or bite. The lymphadenopathy is frequently preceded by a pustule or erythematous papule at the site of the initial wound. Although it is usually mild and self-limiting, approximately 2 percent of patients may develop extension to the central nervous system, liver, spleen, bone, and skin.

- *Bartonella henselae* is thought to be the causative organism, although there is still some question as to the etiologic agent.

DIAGNOSIS

- Diagnosis is made by eliciting a history of cat exposure, typical lymphadenopathy, no other cause of lymphadenopathy, and a positive serologic test for *B. henselae.*[13,14]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Antibiotic therapy is not necessary for uncomplicated cases.
- Patients who are severely ill, those with complications, and patients who are immunocompromised should be treated with rifampin or trimethoprim/sulfamethoxazole.

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21 POSTREPAIR WOUND CARE

Chris Melton

DRESSINGS

- Dressings provide a moist environment, facilitating epithelialization, for the first 1 to 2 days.[1]
- Face and scalp lacerations may be dressed simply with a layer of antibiotic ointment. Wounds elsewhere are usually dressed.[2]
- Iodine solutions should not be used, as they may impair wound healing.
- A wound dressing has four layers: (1) nonadherent layer adjacent to the wound, (2) gauze to absorb drainage, (3) wrapping to hold the first two layers in place, and (4) tape or elastic bandage to secure the dressing.
- The reasons for wound dressings include (1) cleanliness, as the dressing absorbs exudate, (2) protection from external contamination, (3) camouflage to conceal the wound from view, (4) protection of the intact sutures, (5) prevention of excessive movement, and (6) satisfying the patient's expectation that the wound will be dressed.

DRESSING CHANGES

- Dressings should be changed to keep the wound clean and to remove exudate.

- A routine change at 24 h is recommended to evaluate the wound for infection, bleeding, and exudate. If minimal bleeding or exudate is present, a simpler dressing may be placed.

PAIN CONTROL

- If narcotic analgesia is required, it is generally not needed for more than 2 days.

ANTIBIOTIC PROPHYLAXIS

- The use of antibiotics has been recommended for the following types of wounds: (1) intraoral lacerations—penicillin, (2) complicated human bites—amoxicillin/clavulanate, (3) complicated dog bites—amoxicillin/clavulanate, (4) cat bites—amoxicillin/clavulanate, and (5) plantar puncture wounds, especially through rubber-soled shoes—ciprofloxacin and, for children, cephalexin.[3]

RECHECKS

- Rechecks should be scheduled for complicated wounds at risk for infection, for patients who may be immunosuppressed, and for those who may not understand signs of infection.

CLOSURE REMOVAL

- Timing for removal of cutaneous sutures and staples (see Table 21-1).

TABLE 21-1 Timing for Removal of Cutaneous Sutures and Staples

AREA	NUMBER OF DAYS
Face	4–5
Scalp	7–10
Trunk	10
Arm (surface)	10
Arm (joint)	10–14
Hand	10–14
Leg (surface)	10
Leg (joint)	10–14
Foot	14

- Facial sutures are removed early to avoid a ''railroad track'' appearance; adhesive strips may be needed for an additional 3 to 4 days.
- Sutures and staples in highly mobile areas may be needed for an additional 3 to 4 days.

INSTRUCTIONS FOR PATIENTS

1. *Washing:* Immersion or soaking should be avoided; however, scalp, face, and neck wounds can be washed in 8 to 24 h. Wounds in other areas may be washed in 12 to 24 h.
2. *Bleeding:* A small amount of bleeding is expected; however, more should merit a recheck.
3. *Infection:* The wound should be rechecked if there is increased pain, redness, purulent drainage, fever, or redness.
4. *Dehiscence:* The wound should be rechecked if the wound opens.
5. *Cosmesis:* Scar revision may be performed 6 to 9 months after the initial injury if there is not a desirable cosmetic outcome. Patients should be advised that the cosmetic outcome cannot be predicted based on the wound's appearance at the time of wound closure.[4]

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Section 6
CARDIOVASCULAR DISEASES

22 CHEST PAIN AND ISCHEMIC EQUIVALENTS

Thomas A. Rebbecchi

EPIDEMIOLOGY

- Chest pain accounts for 5 percent of all emergency department (ED) visits.
- Ischemic heart disease is the number 1 cause of death among adults in the United States (500,000 per year).

PATHOPHYSIOLOGY

- Chest pain can be visceral or somatic. Visceral pain originates from vessels and organs and is poorly described, such as a heaviness or aching. Somatic pain is dermatomal, from the parietal pleura or structures of the chest wall and is more exactly described.
- Ischemia is an imbalance of oxygen supply and demand.
- Coronary plaque formation occurs due to repetitive injury to the vessel wall. This leads to narrowing of the vessel.
- Angina pectoris is visceral pain due to lack of oxygen to myocytes. Anaerobic metabolism ensues; chemical mediators are released and pain results.
- Knowledge of coronary anatomy will help predict which coronary vessels are involved with an ischemic event (see Fig. 22-1).

CLINICAL FEATURES

- Typical ischemic chest pain is felt as a tightness, squeezing, crushing, or pressure-like feeling in the retrosternal/epigastric area. Radiation of pain to the jaw or arm is associated with a high risk of ischemia.[1] Elderly patients may have less definitive symptoms. Symptoms lasting less than 2 min or longer than 24 h are less likely to be ischemic. Atypical presentations are the rule and are more common in women than men.[2] Up to one-third of acute myocardial infarctions (AMIs) may be silent.[3]
- The 7 major cardiac risk factors are age, male sex, family history, cigarette use, hypertension, high cholesterol, and diabetes mellitus (DM). Cocaine use should be considered as a risk factor. These risk factors can only be used to predict coronary artery disease in a given population.
- In the ED patient with chest pain, risk factors are not predictive in females and only DM and family history are weakly predictive in males.[4]
- Stable angina is chest pain due to a fixed lesion precipitated by exertion, stress, cold, etc.
- Unstable angina is worsening of a stable situation, which puts patients at risk for MI.
- Variant Prinzmetal angina is coronary vasospasm usually at rest.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosing angina is usually based on historical information.
- The electrocardiogram (ECG) is the most important single test. Only 50 percent of patients will

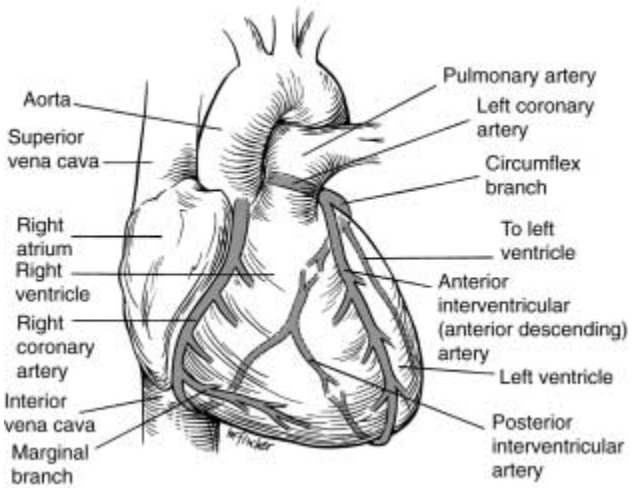

FIG. 22-1 Schematic diagram of the coronary arteries.

have a diagnostic ECG. Serial ECGs are imperative with continued chest pain.

- Serum cardiac markers are useful for ischemia and infarction. Creatine kinase (CK), specifically the MB fraction, measured over 24 h is the ''gold standard'' for myocardial infarction.[5] Other conditions can elevate this blood level (see Table 22-1).
- Troponin and myoglobin can also be used to determine cardiac cell injury (see Table 22-2).[6]
- Echocardiography can diagnose impaired wall function.
- Stress testing and nuclear imaging (Sestamibi) can be used in the ED to risk-stratify patients with chest pain.
- Chest x-ray can aid in the diagnosis of other noncardiac syndromes that may mimic ischemic chest pain (see Table 22-3).

TABLE 22-1 Common Conditions Associated with Elevated CK-MB Levels

PATIENT'S CONDITION OR PRECEDING EVENT
Unstable angina (intermediate coronary syndrome), Acute coronary ischemia
Inflammatory heart diseases
Cardiomyopathies
Circulatory failure and shock
Cardiac surgery
Cardiac trauma
Skeletal muscle trauma (severe)
Dermatomyositis, polymyositis
Myopathic disorders
Muscular dystrophy, especially Duchenne
Extreme exercise
Malignant hyperthermia
Reye's syndrome
Rhabdomyolysis of any cause
Delirium tremens
Ethanol poisoning (chronic)

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The patient should be placed on a cardiac monitor, oxygen should be administered, and IV access obtained. An ECG should be obtained as well.
- The patient should be treated aggressively if the clinical suspicion is high despite a nondiagnostic or normal ECG. The patient should be admitted to a high acuity setting in presence of an acute coronary syndrome.
- The combination of an ECG, clinical history, and

TABLE 22-2 Characteristics of Cardiac Markers

MARKER	MOLECULAR MASS (kDa)	ELEVATION (h)	PEAK	DURATION
Myoglobin	17,800	1–4	6 h	24 h
Myosin light chains	25,000	6–12	2–4 days	6–12 days
Cardiac troponin I	23,500	3–12	18 h	5–10 days
Cardiac troponin T	33,000	3–12	12 h	5–14 days
CK-MB	86,000	3–12	18–24 h	2 days
MB subforms	86,000	3–12	18 h	2 days
LDH	135,000	10	1–2 days	10–14 days
Glycogen phosphorylase BB	188,000	2–4	8 h	1–2 days
Myosin heavy chain	400,000	48	5–6 days	14 days

SOURCE: Adams JE III, Bodor GS, Davila-Roman VG, et al: Cardiac troponin I: A marker with high specificity for cardiac injury. *Circulation* 88:101, 1993,[6] with permission.

TABLE 22-3 Etiology of Nontraumatic Chest Pain

Cardiac causes
Coronary artery disease
Stable angina
Unstable angina
Variant angina
Acute myocardial infarction
Pericarditis
Valvular disease
Aortic stenosis
Subaortic stenosis
Mitral valve prolapse
Vascular causes
Aortic dissection
Pulmonary embolus
Pulmonary hypertension
Pulmonary causes
Pleural irritation from infection, inflammation, infiltration
Barotrauma from pneumothorax, pneumomediastinum
Tracheobronchitis
Musculoskeletal causes
Costochondritis
Intercostal muscle strain
Cervical thoracic spine problems
Gastrointestinal causes
Esophageal reflux/spasm
Mallory Weiss syndrome
Biliary colic
Dyspepsia
Pancreatitis
Miscellaneous causes
Herpes zoster
Chest wall tumors

cardiac markers can be used to risk-stratify the acuity of the admission.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 45, ''Approach to Chest Pain and Possible Myocardial Ischemia,'' by Gary B. Green and Peter M. Hill, and Chap. 47, ''Acute Coronary Syndromes: Unstable Angina, Myocardial Ischemia, and Infarction,'' by Judd E. Hollander.

23 SYNCOPE

Michael G. Mikhail

EPIDEMIOLOGY

- The elderly have the highest incidence of syncope, which accounts for 3 percent of emergency department (ED) visits each year.[1]
- Fifty percent of all patients will never have a definite etiology established.

PATHOPHYSIOLOGY

- The final common pathway of syncope is lack of vital nutrient delivery to the brainstem reticular activating system, leading to loss of consciousness and postural tone.
- The most common causes of syncope are cardiac dysrhythmia, vasovagal reflex, and orthostatic hypotension.[2,3]
- An inciting event causes a drop in cardiac output, which decreases oxygen and substrate delivery to the brain. The reclined posture and the response of autonomic autoregulation centers reestablish cerebral perfusion, leading to a spontaneous return of consciousness.
- In patients with reflex-mediated syncope, a stimulus produces an abnormal autonomic response, an increase in vagal tone. Hypotension with or without bradycardia ensues. Less commonly, the stimulus leads directly to vagal hyperactivity.

CLINICAL FEATURES

- The most common cause of syncope is reflex mediated, which leads to pronounced vagal tone with hypotension and/or bradycardia.
- The hallmark of vasovagal syncope is the prodrome of dizziness, nausea, diminished vision, pallor, and diaphoresis. This diagnosis requires an appropriate stimulus in combination with standing.
- Carotid sinus hypersensitivity, a form of reflex-mediated syncope, which results in asystole greater than 3 s and/or hypotension, is more common in men, the elderly, and among those with ischemic heart disease.
- Orthostatic syncope results from a sudden change in posture after prolonged recumbence and the inability to mount an adequate increase in heart rate and/or peripheral vascular resistance.
- Cardiac syncope is due to dysrhythmia or structural heart disease. Syncope from dysrhythmia is usually sudden with a brief prodrome of only seconds. Structural heart disease is usually unmasked as syncope during exertion or vasodilation. In the elderly this is most commonly due to aortic stenosis. In the young this is most commonly hypertrophic cardiomyopathy. Ten percent of patients with pulmonary embolism will present with syncope.[4]
- Less common causes of syncope include cerebrovascular disorders, subarachnoid hemorrhage, and subclavian steal syndrome.
- Multiple medications, such as beta blockers, calcium channel antagonists, and diuretics are frequent causes of syncope, especially in the elderly. The most commonly implicated medications include antihypertensives and antidepressants.[5]

DIAGNOSIS AND DIFFERENTIAL

- The most important tools in the workup of syncope are a good history, physical examination, and ECG.
- The history is aimed at identifying any high-risk features including age, structural heart disease, and prodromal events. Syncope without warning suggests a dysrhythmia; exertional syncope suggests outflow obstruction.
- The cardiac exam may uncover a murmur that would represent aortic stenosis or hypertrophic cardiomyopathy.
- An ECG may identify evidence of previous silent myocardial infarction or prolonged QT, or evidence of Wolff-Parkinson-White Syndrome.
- Selective laboratory testing of a hematocrit, pregnancy test, or electrolytes and glucose may reveal the etiology of syncope.
- Seizure is the most common disorder mistaken for syncope, which should be distinguished by the postictal phase.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The main goal of ED care is to identify those patients at risk for further medical problems. With a thorough history, physical exam, and ECG patients can be divided into three categories.
- If the diagnosis is established, then patients can be managed for the underlying cause. If the diagnosis is not established, then patients can be stratified as high-risk or non-high-risk.
- High-risk features suggesting risk for sudden cardiac death are age > 45, abnormal ECG, history of ventricular arrhythmia, and congestive heart failure.[6] Admission is directed at determining a possible structural or electrical cause of cardiac syncope.
- Non-high-risk patients are unlikely to have a cardiac etiology and therefore are appropriate for outpatient follow-up.[7,8]
- Worrisome or recurrent cases may benefit from further outpatient workup including an event monitor and tilt testing.
- Patients should also be advised not to drive, work at heights, or place themselves in danger in the event of another syncopal episode.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 46, "Syncope," by Barbara K. Blok.

24 MANAGEMENT OF MYOCARDIAL ISCHEMIA AND INFARCTION

Thomas A. Rebbecchi

EPIDEMIOLOGY

- Ischemic heart disease is the number 1 killer of adults in the United States. Identifying patients with acute coronary syndrome (ACS) is imperative but challenging.
- ACS is a spectrum of disease from stable angina to acute myocardial infarction (AMI).[1]

PATHOPHYSIOLOGY

- Coronary plaque forms on coronary vessel walls after repetitive injury. With plaque rupture, thrombogenic substances are exposed to platelets.
- Platelet adherence to plaque is stimulated by local collagen, macrophages, and sheer force. Platelet glycoprotein IIB/IIIA receptors cross-link fibrinogen as the common pathway of aggregation.
- Luminal occlusion occurs and cell death ensues due to lack of oxygen.
- AMI results in loss of myocardial function including injury to the conduction system leading to abnormal conduction, ectopy, and dysrhythmia. Left ventricular pump function, left ventricular end-diastolic volume, cardiac output, and stroke volume can all decrease. Left ventricular end-diastolic pressure increases.

CLINICAL FEATURES

- The physical examination of a patient with an ACS can range from normal to profound illness.
- Silent/atypical presentations of ischemia are common. Women and the elderly are more likely to present in this way.[2]
- Ischemic/anginal pain is similar to MI pain. MI pain usually resolves only with aggressive intervention, whereas anginal pain can resolve with time or rest.
- Myocardial cell death can lead to dysrhythmia and impaired ventricular function.
- Extent and location of myocardial loss determines prognosis and predicts complications. Twenty-five percent left ventricular loss leads to congestive heart failure; 40 percent left ventricular loss leads to shock. Right ventricular infarct leads to hypotension.
- Dysrhythmias are frequent. Premature ventricular contractions are universal among MIs. Anterior injury leads to tachydysrhythmia. Inferior injury leads to increased vagal tone, first-degree and Mobitz 1 blocks. Anterior/inferior injury leads to higher degree blocks.
- Of AMI patients 15 to 20 percent have some degree of congestive heart failure.
- Free-wall myocardial rupture account for 10 percent of AMI fatalities and occurs 1 to 5 days into the event.
- Interventricular wall rupture is signified by pain, shortness of breath (SOB), and a holosystolic murmur.
- Papillary muscle rupture occurs in 1 percent of all MIs 3 to 5 days into the event.
- Pericarditis is seen in up to 20 percent of all MIs 2 to 7 days after the event.

DIAGNOSIS AND DIFFERENTIAL

- The history is usually suggestive, but the ECG is the best single test available in the ED. In the setting of AMI, the ECG can range from normal (up to 5 percent) to distinct ST-segment elevation (see Table 24-1).

TABLE 24-1 Localization of MI Based on ECG Findings

V_2–V_4—anterior
II, III, aVF, V_5, V_6—inferior
V_1–V_3—anteroseptal
I, aVL, V_4–V_6—lateral
V_1–V_6—anterolateral
V_{4R}–V_{6R}—right ventricular (often associated with inferior)
Posterior MI has large R > .04 mm, R/S > 1, and ST depression in V_1 and V_2

ABBREVIATIONS: MI, myocardial infarction; ECG, electrocardiogram.

- With a nondiagnostic ECG, serum markers may be helpful in determining cardiac injury.
- Chest x-rays may be useful in determining other causes of ischemic-like pain.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of ACS and AMI require aggressive treatment and interventions ranging from oxygen therapy to thrombolytics. Several therapies are universal; others need to be individualized.
- The goal is to protect the myocardium by lowering oxygen demand and increasing oxygen delivery. Initial management includes intravenous (IV) access, oxygen, cardiac monitor, and ECG.
- Giving aspirin has been shown to reduce mortality by 20 percent.
- Nitroglycerin (NTG) will dilate coronary vessels to enhance oxygen delivery. Initially the sublingual route is appropriate. After 3 sublingual NTG tablets or sprays 3 to 5 min apart, IV NTG can be titrated for pain keeping the blood pressure within the normal range.
- Thrombolytic agents will disrupt a coronary clot if given within 12 h of onset of pain. These therapies are indicated for patients with symptoms consistent with AMI and have at least 1 mm ST-segment elevation in 2 contiguous ECG leads.[3] Streptokinase activates plasminogen, is antigenic, and is given as an infusion. Tissue plasminogen activator (tPA) is fibrin specific. Front-end (accelerated) loading is preferred and has been shown to be of greatest benefit.[4] Reteplase is a modified tPA given in bolus form. Hemorrhage is the major risk of these drugs. Fresh frozen plasma and cryoprecipitate are both needed to reverse the effects of thrombolytics (see Tables 24-2 and 24-3).
- Heparin is an anticoagulant that prevents clot propagation and is a recommended adjunct to thrombolytics. Dosing is based on weight. Heparin increases the risk of bleeding.

TABLE 24-2 Indications for Thrombolysis

Symptoms consistent with MI with onset <12h
ECG criteria: >1 mm ST elevation in 2 or more contiguous limb leads >2 mm ST elevation in 2 or more contiguous chest leads New left bundle branch block
No contraindications (see Table 24-3)
Absence of cardiogenic shock, unless mechanical reperfusion will be delayed > 60 min, then use tPA

ABBREVIATIONS: MI, myocardial infarction; ECG, electrocardiogram; tPa, tissue plasminogen activator.

TABLE 24-3 Contraindications for Thrombolysis

Absolute
Active or recent (<10 days) internal bleeding
Active bleeding
History of CVA < 2–6 months or any hemorrhagic CVA
Intracranial or intraspinous surgery or trauma <2 months
Intracranial or intraspinous neoplasm, aneurysm, AV malformation
Trauma or surgery at a noncompressible site <10 days
Suspected aortic dissection or pericarditis
Allergy to specific thrombolytic
Relative
Known bleeding diathesis
Severe uncontrolled HTN (SBP > 200 mmHg and/or DBP > 120 mmHg)
Active peptic ulcer disease
Cardiopulmonary resuscitation >10 min
Use of oral anticoagulants (PT > 15 s, INR > 2)
Hemorrhagic ophthalmic conditions
Ischemic or embolic CVA > 6 months
Uncontrolled HTN (SBP > 180 mmHg and/or DPB > 110 mmHg)
Puncture of noncompressible blood vessel <10 days
Significant trauma or major surgery >2 weeks but <2 months
Pregnancy

ABBREVIATIONS: CVA, cerebrovascular accident; AV, atrioventricular; HTN, hypertension; SBP, systolic blood pressure; DBP, diastolic blood pressure; PT, prothrombin time; INR, international normalized ratio.

- Primary (rescue) angioplasty can be used to open a vessel in place of thrombolytics or if thrombolytics are not effective. There is a lower incidence of reinfarction, ischemia, and bleeding. Complications include coronary artery dissection, platelet deposition, thrombus formation, and plaque hemorrhage.[5]
- Beta blockers lower mortality in AMI if given within 8 h. The optimal heart rate of 60 to 80 beats per minute will decrease the workload of the heart. There is a sustained reduction in mortality with use of beta blockers when compared with their absence.[6]
- Glycoprotein IIB/IIIA inhibitors stop platelet aggregation and are used to stabilize ACS and are used in conjunction with primary angioplasty. Given in the setting of non-Q-wave MI and refractory angina, the products have been shown to reduce death, AMI, and refractory ischemia in the short term.[7]
- Morphine reduces the pain of angina/MI, as well as reducing preload.
- Calcium channel blockers have antianginal, vasodilatory, and antihypertensive properties but have not been shown to reduce mortality rate after MI.[3,8]

- Management of cocaine-associated myocardial ischemia focuses on reversal of hypertension, vasoconstriction, tachycardia, and predisposition to thrombus formation.[9] ASA, NTG, heparin with an anxiolytic agent can be used to treat the ischemic effects of cocaine. Beta blockers should be avoided because they increase central nervous system toxicity and coronary vasospasm.[9] Thrombolytic therapy should be used with caution.
- All patients with ACS/AMI should be admitted to an intensive care setting and be evaluated by a cardiologist in an expeditious manner.

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25 HEART FAILURE AND PULMONARY EDEMA

David M. Cline

EPIDEMIOLOGY

- Mortality rates from heart failure are increasing,[1] and one-half of patients with severe heart failure (left ventricular ejection fraction less than 35 percent) die within 1 year of diagnosis.[2]

PATHOPHYSIOLOGY

- Three factors—contractility, preload, and afterload—determine ventricular stroke volume. Coupled with heart rate, stroke volume determines cardiac output.
- Low-output failure is due to an inherent problem in myocardial contraction.
- High-output failure occurs when functionally intact myocardium cannot meet excess systemic demands. The causes of high-output failure are relatively few and include anemia, thyrotoxicosis, large arteriovenous shunts, beriberi, and Paget's disease of the bone.
- Pulmonary edema or congestion is the cardinal manifestation of left-sided heart failure.
- Isolated right-sided failure may occur from such causes as right ventricular infarction or pulmonary embolism; however, the most common cause of right-sided failure is left-sided failure.[3]
- Once heart failure has developed, several neurohormonal compensatory mechanisms are initiated.[4] The reduction in blood flow to the kidneys results in increased stimulation of the renin-angiotensin-aldosterone axis and secretion of antidiuretic hormone. The end result is enhanced sodium and water retention by the kidneys, which leads to fluid overload and the clinical manifestations

of congestive heart failure (CHF). The increased adrenergic tone leads to arteriolar vasoconstriction, a significant rise in afterload, and finally to increase cardiac work.
- The most common precipitating factors of heart failure are (1) cardiac tachyarrhythmias, such as atrial fibrillation; (2) acute myocardial infarction or ischemia; (3) discontinuation of medications, such as diuretics; (4) increased sodium load; (5) drugs that impair myocardial function; and (6) physical overexertion.[5]

CLINICAL FEATURES

- Patients with acute pulmonary edema usually present with symptoms of left heart failure, such as severe respiratory distress, frothy pink or white sputum, moist pulmonary rales, and an S_3 or S_4. Patients frequently are tachycardic, have cardiac dysrhythmias such as atrial fibrillation or premature ventricular contractions (PVCs), and are hypertensive.
- The most common symptom of left-sided heart failure is breathlessness or dyspnea, particularly with exertion.[6]
- Other symptoms of left-sided heart failure include paroxysmal nocturnal dyspnea, orthopnea, nocturia, pulsus alternans, fatigue, and possibly altered mental status.[3]
- Patients with right-sided heart failure have dependent edema of the extremities and may have jugular venous distention, hepatic enlargement, and, less commonly, ascites.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of acute pulmonary edema is made with clinical findings and the chest x-ray.
- Chest x-ray may reveal cardiomegaly (cardiothoracic ratio greater than 0.5 on a posteroanterior film), vascular redistribution to the upper lung fields, Kerley B lines (short linear markings at the periphery of the lower lung fields), and pleural effusions.
- The diagnosis of right-sided heart failure is made clinically; but if the cause is left-sided heart failure, the heart will be enlarged on chest x-ray.
- The electrocardiogram (ECG) may reveal acute myocardial infarction, ischemia, or—if CHF is chronic—left ventricular hypertrophy (LVH), atrial enlargement, or conduction abnormalities.
- The differential diagnosis for acute pulmonary edema includes the common causes of acute respiratory distress: asthma, chronic obstructive pulmonary disease (COPD), pneumonia, allergic reactions, and other causes of respiratory failure.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Administer 100% oxygen by face mask to achieve an oxygen saturation of 95% by pulse oximetry. Consider immediate intubation for unconscious or visibly tiring patients.
- If hypoxia persists despite oxygen therapy, apply continuous positive airway pressure (CPAP) or biphasic positive airway pressure (BiPAP) via face mask.
- Administer 0.4 mg of nitroglycerin sublingually (may be repeated every 5 min), or, as a topical paste, 1 to 2 in. If the patient does not respond or the ECG shows ischemia, give nitroglycerin as an IV drip, 10 μg/min, and titrate.
- Administer a potent intravenous diuretic such as furosemide, 40 to 80 mg IV, or bumetanide (bumex), 0.5 to 1 mg IV. Furosemide is more efficacious when given with nitrates.[7] Electrolytes should be monitored, especially serum potassium.
- For patients with resistant hypertension or those who are not responding well to nitroglycerin, nitroprusside may be used, starting at 2.5 μg/kg/min and titrated.
- For hypotensive patients or patients in need of additional inotropic support, begin dopamine at 5 to 10 μg/kg/min and titrate to a systolic blood pressure of 90 to 100. Dobutamine can be given in combination with dopamine or as a single agent providing the patient is not in severe circulatory shock. Start dobutamine at 2.5 μg/kg/min and titrate to the desired response.
- Consider thrombolytic agents for heart failure caused by myocardial infarction.
- For acute mitral valve or aortic valve regurgitation, emergency surgery may be indicated.
- Treat coexisting dysrhythmias (see Chap. 4) or electrolyte disturbances (see Chap. 6), avoiding those therapies that impair the inotropic state of the heart.
- Morphine can be given (1 to 2 mg IV) and repeated as needed. Its use is controversial, however; it may cause respiratory depression and adds little to oxygen, diuretics, and nitrates.
- Digoxin acts too slowly to be of benefit in acute situations.
- Rotation of tourniquets does not reduce preload and should not be done.
- For anuric (dialysis) patients, sorbitol and phlebotomy may have some benefit, but dialysis is the

treatment of choice in patients who prove resistant to nitrates.
- Long-term treatment of CHF includes dietary salt reduction; chronic use of diuretics such as furosemide, 20 to 80 mg PO daily; afterload reducers such as captopril, 6.25 to 25 mg PO bid/tid; and digoxin 0.125 to 0.25 mg PO daily.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 49, ''Heart Failure and Pulmonary Edema,'' by Charles B. Cairns.

26 VALVULAR HEART DISEASE AND ENDOCARDITIS

David M. Cline

- Ninety percent of valvular disease is chronic, with decades between the onset of the structural abnormality and symptoms.
- Through chronic adaptation by dilation and hypertrophy, cardiac function can be preserved for years, which may delay the diagnosis for one to two decades until a murmur is detected on auscultation.
- The four heart valves prevent retrograde flow of blood during the cardiac cycle, allowing efficient ejection of blood with each contraction of the ventricles. The mitral valve has two cusps, while the other three heart valves normally have three cusps. The right and left papillary muscles promote effective closure of the tricuspid and mitral valves, respectively.

MITRAL STENOSIS

PATHOPHYSIOLOGY

- Despite its declining frequency, rheumatic heart disease is still the most common cause of mitral valve stenosis.
- The majority of patients eventually develop atrial fibrillation because of progressive dilation of the atria.

CLINICAL FEATURES

- As with all valvular diseases, exertional dyspnea is the most common presenting symptom (80 percent of patients with mitral stenosis present with dyspnea).
- Hemoptysis is the second most common presenting symptom and may be massive if a bronchial vein ruptures.
- Systemic emboli may occur and result in myocardial, kidney, central nervous system, or peripheral infarction.
- The classic murmur of mitral stenosis and associated signs are listed in Table 26-1.
- The electrocardiogram (ECG) may demonstrate notched or diphasic P waves and right axis deviation.
- On the chest radiography, straightening of the left heart border, indicating left atrial enlargement, is a typical early radiographic finding. Eventually, findings of pulmonary congestion are noted: redistribution of flow to the upper lung fields, Kerley B lines, and an increase in vascular markings.

MITRAL INCOMPETENCE

PATHOPHYSIOLOGY

- Infective endocarditis or myocardial infarction can cause acute rupture of the chordae tendineae

TABLE 26-1 Comparison of Heart Murmurs, Sounds, and Signs

VALVE DISORDER	MURMUR	HEART SOUNDS AND SIGNS
Mitral stenosis	Mid-diastolic rumble, crescendos into S_2	Loud snapping S_1, apical impulse is small and tapping due to underfilled left ventricle.
Mitral regurgitation	Acute: harsh apical systolic murmur that starts with S_1 and may end before S_2 Chronic: high pitched apical holosystolic murmur that radiates to the axilla	S_3 and S_4 may be heard.
Mitral valve prolapse	Click may be followed by a late systolic murmur that crescendos into S_2	Mid-systolic click; S_2 may be diminished by the late systolic murmur.
Aortic stenosis	Harsh systolic ejection murmur	Paradoxic splitting of S_2, S_3, and S_4 may be present; pulse of small amplitude; pulse has a slow rise and sustained peak.
Aortic regurgitation	High pitched blowing diastolic murmur immediately after S_2	S_3 may be present; wide pulse pressure.
IHSS	Harsh systolic crescendo-decrescendo best heart at the apex or left sternal border	No opening snap; apical impulse may be double; pulse has a brisk rise and double peak.

ABBREVIATION: IHSS, idiopathic hypertrophic subaortic stenosis.

or papillary muscles or cause perforation of the valve leaflets.
- Inferior myocardial infarction due to right coronary occlusion is the most common cause of ischemic mitral valve incompetence.
- Rheumatic heart disease is the most common cause of chronic mitral incompetence.
- An association has been found between the use of appetite suppressant drugs (fenfluramine and phentermine, or dexfenfluramine) and cardiac valve incompetence,[1] although this has been questioned.[2]
- Acute regurgitation into a noncompliant left atrium quickly elevates pressures and causes pulmonary edema. In contrast, in the chronic state the left atrium dilates so that left atrial pressure rises little, even with a large regurgitant flow.

CLINICAL FEATURES

- Acute mitral incompetence presents with dyspnea, tachycardia, and pulmonary edema.
- Intermittent mitral incompetence usually presents with acute episodes of respiratory distress due to pulmonary edema and can be asymptomatic between attacks.
- The ECG may show evidence of acute inferior wall infarction (more common than anterior wall infarction in this setting).
- On the chest radiography, acute mitral incompetence from papillary muscle rupture may result in a minimally enlarged left atrium and pulmonary edema, with less cardiac enlargement than expected.
- Chronic mitral incompetence may be tolerated for years or even decades. The first symptom is usually exertional dyspnea, sometimes prompted by atrial fibrillation. If patients are not anticoagulated, systemic emboli occur in 20 percent and are often asymptomatic.
- The classic murmur and signs of mitral incompetence are listed in Table 26-1.
- The ECG may demonstrate findings of left atrial and left ventricular hypertrophy (LVH). On chest radiography, chronic mitral incompetence produces left ventricular and atrial enlargement that is proportional to the severity of the regurgitant volume.

MITRAL VALVE PROLAPSE

PATHOPHYSIOLOGY

- The etiology of mitral valve prolapse (MVP), or the click-murmur syndrome, is not known but may be congenital. Mitral valve prolapse is the most common valvular heart disease in industrialized countries, affecting about 3 percent of the population.[3]
- Male sex, age over 45, and the presence of regurgitation, recognized clinically by a short diastolic murmur, places the patient in a high risk group for complications.[4]

CLINICAL FEATURES

- Most patients are asymptomatic (see Table 26-1). Symptoms include atypical chest pain, palpitations, fatigue, and dyspnea unrelated to exertion.

- There is an increased incidence of sudden death and dysrhythmias in patients with MVP. There is also an increased incidence of transient ischemic attacks under the age of 45.
- In patients with MVP without mitral regurgitation at rest, exercise provokes mitral regurgitation in 32 percent of patients and predicts a high risk for morbid events.[5]

AORTIC STENOSIS

PATHOPHYSIOLOGY

- Congenital heart disease is the most common cause of aortic stenosis, with the presence of a bicuspid valve accounting for 50 percent of cases.
- Rheumatic heart disease is the second most common cause, followed by degenerative heart disease or calcific aortic stenosis, which is the most common cause in patients over age 70.
- Blood flow into the aorta is obstructed, producing progressive LVH and low cardiac output.

CLINICAL FEATURES

- The classic triad is dyspnea, chest pain, and syncope.
- Dyspnea is usually the first symptom, followed by paroxysmal nocturnal dyspnea, syncope on exertion, angina, and myocardial infarction.
- The classic murmur and associated signs of aortic stenosis are listed in Table 26-1.
- Sudden death, usually from a dysrhythmia, occurs in 25 percent of patients.
- The ECG usually demonstrates criteria for LVH and, in 10 percent of patients, left or right bundle branch block.
- Early in the disease the chest radiograph is normal, but eventually LVH and findings of congestive heart failure are evident if the patient does not have valve replacement.

AORTIC INCOMPETENCE

PATHOPHYSIOLOGY

- In 20 percent of patients, the cause of aortic incompetence is acute in nature. Infective endocarditis accounts for the majority of acute cases; aortic dissection at the aortic root causes the remainder. In acute cases, a sudden increase in backflow of blood into the ventricle raises left ventricular end-diastolic pressure, which may cause acute heart failure.
- Rheumatic heart disease and congenital disease cause the majority of chronic cases.
- An association between the appetite-suppressant drugs (fenfluramine and phentermine or dexfenfluramine) has also been found for aortic incompetence.[2]
- In chronic disease, the ventricle progressively dilates to accommodate the regurgitant blood volume. Wide pulse pressures result from the fall in diastolic pressure, and marked peripheral vasodilation is seen.

CLINICAL FEATURES

- In acute disease, dyspnea is the most common presenting symptom, seen in 50 percent of patients. Many patients have acute pulmonary edema with pink frothy sputum. Patients may complain of fever and chills if endocarditis is the cause.
- Dissection of the ascending aorta typically produces a ''tearing'' chest pain that may radiate between the shoulder blades.
- Changes in the ECG may be seen with aortic dissection, including ischemia or findings of acute inferior myocardial infarction, suggesting involvement of the right coronary artery.
- The classic murmur and signs of aortic incompetence are listed in Table 26-1.
- In the acute state, the chest radiography demonstrates acute pulmonary edema with less cardiac enlargement than expected.
- In the chronic state, about one-third of patients will have palpitations associated with a large stroke volume and/or premature ventricular contractions. Frequently these sensations are noticed in bed.
- In the chronic state, signs include a wide pulse pressure with a prominent ventricular impulse, which may be manifested as head bobbing.
- ''Water hammer pulse'' may be noted; this is a peripheral pulse that has a quick rise in upstroke followed by a peripheral collapse.
- Other classic findings may include accentuated precordial apical thrust, pulsus biferiens, Duroziez sign (a to-and-fro femoral murmur), and Quincke pulse (capillary pulsations visible at the proximal nailbed, while pressure is applied at the tip).
- In chronic aortic incompetence, the ECG demonstrates LVH, and the chest radiograph shows LVH, aortic dilation, and possibly evidence of congestive heart failure.

HYPERTROPHIC CARDIOMYOPATHY (IDIOPATHIC HYPERTROPHIC SUBAORTIC STENOSIS)

- This disease is discussed in Chap. 27, ''Cardiomyopathies, Myocarditis, and Pericardial Disease,'' and is mentioned here only to aid in its differentiation from valvular aortic stenosis (see Table 26-1).

RIGHT-SIDED VALVULAR HEART DISEASE

PATHOPHYSIOLOGY

- Drug users with endocarditis due to aggressive organisms, such as *Staphylococcus aureus,* are the largest group of patients with isolated tricuspid disease.
- Rheumatic heart disease may affect more than one valve, and tricuspid disease is frequently seen in conjunction with left-sided valvular disease.
- The most common cause of pulmonary stenosis is congenital tetralogy of Fallot, which is usually corrected surgically in infancy.

CLINICAL FEATURES

- The most common presenting symptoms of right-sided valvular disease are dyspnea and orthopnea. Because of the organisms involved, patients presenting with tricuspid incompetence in association with endocarditis are acutely ill with sepsis.
- In tricuspid incompetence, the murmur is soft blowing, holosystolic, and best heard along the lower left sternal border. In tricuspid stenosis, the rumbling crescendo-decrescendo diastolic murmur occurs just prior to S_1. This murmur is best heard along the lower left sternal border.

DIAGNOSIS OF VALVULAR HEART DISEASE

- The ECG and chest radiograph may be of help, but neither is confirmatory. The suspected diagnosis should be confirmed by echocardiography and/or consultation with a cardiologist.

EMERGENCY DEPARTMENT CARE OF SYMPTOMATIC VALVULAR HEART DISEASE

- In cases of acute valvular incompetence caused by myocardial infarction, the infusion of thrombolytic therapy may reestablish blood flow to the papillary muscle, with restoration of function.[6] The alternative to thrombolytic therapy is coronary angioplasty.[7]
- The regurgitation of aortic and mitral incompetence may be lessened by reducing afterload. When the cause of mitral incompetence is myocardial ischemia, regurgitation can be lessened by treatment with nitrates.
- Pulmonary edema should be treated with oxygen, intubation for failing respiratory effort, diuretics, and nitrates if tolerated.
- Patients with aortic stenosis usually have normal-to-low blood pressure and do not tolerate afterload reducers. In contrast, patients with mitral incompetence or aortic incompetence can benefit from intravenous (IV) nitroprusside or nitroglycerin even with normal blood pressures.[8]
- The hypertension associated with aortic dissection should be controlled with IV nitroprusside and beta blockade with labetalol.
- In patients who do not respond to medical management, intraaortic balloon counterpulsation should be considered. However, this is contraindicated in wide-open aortic regurgitation.
- Rapid atrial fibrillation, which may precipitate symptoms in patients with silent valvular disease, should be rate-controlled with IV diltiazem or digoxin.
- Mitral stenosis is the most frequent valvular heart disease associated with hemoptysis, which can be severe enough to require blood transfusion and emergency surgery.
- In the event of embolization, anticoagulation should be undertaken with IV heparin as long as there is no evidence of central nervous system bleeding. Anticoagulation is especially needed in the setting of atrial fibrillation.
- Emergency surgery should be considered in all cases of acute symptomatic valvular disease.[9]
- Antibiotic prophylaxis for infective endocarditis is recommended during procedures that may produce bacteremia in patients at risk for developing endocarditis, and the American Heart Association guidelines, which were revised in 1997, should be followed (see Table 26-2).[10]
- Patients with aortic stenosis presenting with syncope on exertion should be considered for admission because of the critical limitation of blood flow that syncope usually heralds.

INFECTIVE ENDOCARDITIS

- Because of the declining frequency of rheumatic heart disease, the increasing number of cardiac

TABLE 26-2 Prophylaxis for Infective Endocarditis

PROCEDURE	STANDARD REGIMEN*	ALTERNATE REGIMEN
Dental procedure known to cause bleeding	Amoxicillin 2.0 g PO 1 h prior to the procedure, or Ampicillin 2.0 g IM or IV 30 min prior to procedure	Clindamycin 600 mg PO 1 h before procedure, or Cephalexin 2.0 g PO 1 h prior to the procedure, or Cefadroxil 2.0 g PO 1 h prior to the procedure, or Azithromycin 500 mg PO 1 h prior to the procedure, or Clarithromycin 500 mg PO 1 h prior to the procedure
Urethral catheterization if infection is present; urethral dilation	Ampicillin 2.0 g IV/IM plus gentamicin 1.5 mg/kg IV/IM (not to exceed 120 mg) 30 min before procedure followed by half the original dose of ampicillin 6 h later	Vancomycin 1.0 g IV over 1 h plus gentamicin 1.5 mg/kg IV/IM (not to exceed 120 mg), complete infusion within 30 min of starting procedure; for moderate risk patients, amoxicillin 2 g PO 1 h prior to procedure
Incision and drainage of infected tissue	Cefazolin 1.0 g IV/IM 30 min before procedure, or Cephalexin 2.0 g PO 1 h prior to the procedure, or Cefadroxil 2.0 g PO 1 h prior to the procedure	Vancomycin 1.0 g IV over 1 h plus gentamicin 1.5 mg/kg IV/IM (not to exceed 120 mg), complete infusion within 30 min of starting procedure

* Includes patients with prosthetic heart valves and others at high risk. Initial pediatric doses are as follows: amoxicillin, 50 mg/kg, ampicillin 50 mg/kg, cephalexin 50 mg/kg, cefadroxil 50 mg/kg, azithromycin 15 mg/kg, clarithromycin 15 mg/kg, clindamycin 20 mg/kg, gentamicin 2 mg/kg, and vancomycin 20 mg/kg. Pediatric doses should not exceed listed adult doses.

surgical procedures, and the increasing numbers of IV drug users, the nature of this disease has changed dramatically in the last 20 years.[11]

PATHOPHYSIOLOGY

- The cardiac valve leaflets are the portion of the heart most susceptible to infection because of their limited blood supply. Endocarditis can occur with normal valves but is more common with congenital and acquired valve disease and prosthetic valves.
- Bacteria and fungi gain entry to the circulation through various routes and settle on valvular tissue. A platelet-fibrin matrix forms, and further growth of the organisms forms a vegetation on the valve which makes the organisms inaccessible to normal cellular host defenses.
- Risk factors for infective endocarditis include congenital or acquired valvular heart disease, IV drug abuse, prosthetic valves, hemodialysis or peritoneal dialysis, indwelling venous catheters, postcardiac surgery, and calcific valve degeneration that occurs with increasing age.
- Rheumatic heart disease, although still important, is declining in frequency.
- Embolism of the vegetations is responsible for many of the clinical features of the disease.
- Left-sided disease (aortic and mitral involvement) is the most common, except in injecting drug users. The most common organisms include *Strep. viridans* (declining in frequency), *Staph. aureus* (increasing in frequency), *Enterococcus,* and fungal organisms.
- Cardiac failure is the most common cause of death in left-sided disease, but deaths due to neurologic complications are increasing.
- Right-sided disease is usually seen in IV drug abusers (60 percent) and is caused by *Staph. aureus* (75 percent), and *Streptococcus pneumonia* (20 percent), gram-negative organisms (4 percent), and fungal organisms.[12]
- Children with endocarditis most commonly have complex congenital heart disease (35 percent) or unrepaired ventricular septal defect (14 percent).[13]

CLINICAL FEATURES

- Acute left-sided disease presents with a picture of sepsis with or without cardiac failure. Typically, patients appear ill with fever, chills, and tachycardia and may have significant congestive failure symptoms such as dyspnea, frothy sputum, and chest pain.
- Neurologic symptoms secondary to aseptic meningoencephalitis and embolization of vegetations account for about one-third of emergency department presentations. These complications most commonly are mental status changes, hemiplegia, aphasia, ataxia, or severe headache. Monocular blindness can also occur.
- Patients with subacute left-sided disease present with recurrent intermittent fever and constitu-

tional symptoms such as malaise, anorexia, or weight loss.
- The majority of patients with left-sided subacute disease have a murmur of aortic or mitral regurgitation or a change in their previous murmur at the time of their admission to the hospital.
- Patients may have Roth spots, which are retinal hemorrhages with central clearing. Peripheral evidence of endocarditis includes Osler nodes, tender nodules on the tips of the toes and fingers, and Janeway lesions, nontender plaques on the soles of the feet and palms of the hands, and clubbing. Petechiae may be seen on the conjunctiva, hard palate, neck, and upper trunk. Splinter hemorrhages may be seen in the nails of the fingers or toes. Splenomegaly is noted in 25 percent of patients.
- Right-sided disease is usually acute and presents with fever and respiratory symptoms: cough, chest pain, hemoptysis, and dyspnea.
- Murmurs are detectable in fewer than 50 percent of patients with right-sided disease. The chest radiography may reveal pulmonary effusions and multiple pulmonary infiltrates of variable size and shape. Although meningitis coexists in only 5 percent of left-sided disease, bacterial meningitis is seen in up to 30 percent of patients with right-sided disease.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of endocarditis is based on positive blood cultures results and echocardiographic evidence of valvular injury or vegetations.
- Nonspecific laboratory findings that support the diagnosis of endocarditis include leukocytosis, elevated C-reactive proteins, positive rheumatoid factor, normocytic anemia, hematuria (25 to 50 percent), and pyuria.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Acute rupture of the mitral or aortic valve should be stabilized with afterload reducers such as sodium nitroprusside, with insertion of a Swan-Ganz catheter for monitoring therapy as soon as possible. Preparation for emergency surgery should be made for patients suspected of acute valvular rupture.[14]
- For acute infective endocarditis, a penicillinase-resistant penicillin, such as oxacillin 2 g q4h, should be given with an aminoglycoside, such as gentamicin 1 mg/kg up to 80 mg q8h, chosen on the basis of local patterns of susceptibility.
- In areas where there is a high incidence of methicillin-resistant *Staphylococcus* or in the case of a patient taking oral antibiotics already, vancomycin 1 g IV should be used in addition to an aminoglycoside.
- Patients with prosthetic valve endocarditis should be treated with antibiotics that cover *S. epidermidis,* usually vancomycin, 1 g IV, in addition to an aminoglycoside and rifampin.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 50, "Valvular Emergencies and Endocarditis," by David M. Cline.

27 CARDIOMYOPATHIES, MYOCARDITIS, AND PERICARDIAL DISEASE

David M. Cline

THE CARDIOMYOPATHIES

- Cardiomyopathies are the third most common form of heart disease in the United States and are the second most common cause of sudden death in the adolescent population.[1] It is a disease process that directly affects the cardiac structure and alters myocardial function.
- Four types are currently recognized: (*a*) dilated cardiomyopathy (DCM), (*b*) hypertrophied cardiomyopathy (HCM), (*c*) restrictive cardiomyopathy, and (*d*) dysrhythmic right ventricular cardiomyopathy.[2] Unclassified cardiomyopathy is an additional category including primary heart muscle disorders that do not fit into any of the above four groups.

DILATED CARDIOMYOPATHY

PATHOPHYSIOLOGY

- Dilation and compensatory hypertrophy of the myocardium result in depressed systolic function and pump failure, leading to low cardiac output.[3]
- Eighty percent of cases of DCM are idiopathic. Other etiologies include toxins (e.g., alcohol, cocaine, and antiretroviral drugs), infections (e.g., viral, bacterial, and fungal), collagen vascular disorders, hypersensitivity, peripartum, metabolic disorders (e.g., nutritional, endocrine, electrolyte disturbances), neuromuscular diseases, and genetic factors.
- Blacks and males have a 2.5-fold increased risk as compared with whites and females. The most common age at the time of diagnosis is 20 to 50 years.[3]

CLINICAL FEATURES

- Systolic pump failure leads to signs and symptoms of congestive heart failure (CHF), including dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea.
- Chest pain due to limited coronary vascular reserve may also be present.
- Mural thrombi can form from diminished ventricular contractile force, and there may be signs of peripheral embolization (e.g., flank pain, hematuria, and extremity cyanosis).
- Holosystolic murmur may be heard along the lower left sternal border or at the apex. Other findings include a summation group, an enlarged and pulsatile liver, bibasilar rales, and dependent edema.

DIAGNOSIS AND DIFFERENTIAL

- Chest x-ray usually shows an enlarged cardiac silhouette, biventricular enlargement, and pulmonary vascular congestion.
- The electrocardiogram (ECG) shows left ventricular hypertrophy, left atrial enlargement, Q or QS waves, and poor R-wave progression across the precordium. Atrial fibrillation and ventricular ectopy are frequently present.
- Echocardiography confirms the diagnosis and demonstrates ventricular enlargement, increased systolic and diastolic volumes, and a decreased ejection fraction.
- Differential diagnosis includes acute myocardial infarction, restrictive pericarditis, acute valvular disruption, sepsis, or any other condition that results in a state of low cardiac output.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with newly diagnosed, symptomatic DCM require admission to a monitored bed or intensive care unit.
- Intravenous diuretics (e.g., furosemide 40 mg intravenously) and digoxin (maximum dose 0.5 mg intravenously) can be administered. These drugs have symptomatic benefit but have not been shown to increase survival.
- Angiotensin converting enzyme (ACE) inhibitors (e.g., enalapril 1.25 mg intravenously every 6 h) and beta blockers (e.g., carvedilol 3.125 mg orally) can be administered. These drugs have been shown to improve survival in DCM with CHF.[4,5]

- Amiodarone (loaded 150 mg intravenously over 10 min, then 1mg/min for 6 h) for complex ventricular ectopy can be administered.[6]
- Anticoagulation should be considered to reduce formation of mural thrombus.

HYPERTROPHIC CARDIOMYOPATHY

PATHOPHYSIOLOGY

- This illness is characterized by asymmetrically increased left ventricular and/or right ventricular muscle mass involving the intraventricular septum without ventricular dilatation.[7]
- The result is abnormal compliance of the left ventricle leading to impaired diastolic relaxation and diastolic filling. Cardiac output is usually normal.
- Fifty percent of cases are hereditary.
- The prevalence is 1 in 500; the mortality rate is 1 percent but 4 to 6 percent in childhood and adolescence.

CLINICAL FEATURES

- Symptom severity progresses with age.
- Dyspnea on exertion is the most common symptom, followed by angina-like chest pain, palpitations, and syncope.[8]
- Patients may be aware of forceful ventricular contractions and call these palpitations.
- Physical exam may reveal a hyperdynamic apical impulse, a precordial lift, and a systolic ejection murmur best heard at the lower left sternal border or apex.
- The murmur may be increased with Valsalva maneuver or standing after squatting. The murmur can be decreased by squatting, forceful hand gripping, or passive leg elevation with the patient supine. (See Chap. 26 for contrasting murmurs.)

DIAGNOSIS AND DIFFERENTIAL

- The ECG demonstrates left ventricular hypertrophy in 30 percent of patients and left atrial enlargement in 25 to 50 percent. Large septal Q waves (greater than 0.3 mV) are present in 25 percent. Another ECG finding is upright T waves in those leads with QS or QR complexes (T-wave inversion in those leads would suggest ischemia).
- Chest x-ray is usually normal. Echocardiography is the diagnostic study of choice.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Symptoms of HCM may mimic ischemic heart disease; treatment of those symptoms is covered in Chap. 24. Otherwise, general supportive care is indicated. Beta blockers are the mainstay of treatment for patients with HCM and chest pain.[8]
- Patients should be discouraged from engaging in vigorous exercise.[9] Those with suspected HCM who have syncope should be hospitalized.

RESTRICTIVE CARDIOMYOPATHY

- This is one of the least common cardiomyopathies. In this form of the disease the ventricular volume and wall thickness are normal, but there is decreased diastolic volume of both ventricles.
- Most causes are idiopathic, but systemic disorders have been implicated, such as amyloidosis, sarcoidosis, hemochromatosis, scleroderma, carcinoid, hypereosinophilic syndrome, and endomyocardial fibrosis.[10]

CLINICAL FEATURES

- Symptoms of congestive heart failure (CHF) predominate, including dyspnea, orthopnea, and pedal edema. Chest pain is uncommon.
- Physical exam may reveal an S_3 or S_4 cardiac gallop, pulmonary rales, jugular venous distention, Kussmaul's sign (inspiratory jugular venous distension), hepatomegaly, pedal edema, and ascites.

DIAGNOSIS AND DIFFERENTIAL

- The chest x-ray may show signs of CHF without cardiomegaly.
- Nonspecific ECG changes or, in the case of amyloidosis or sarcoidosis, conduction disturbances and low-voltage QRS complexes may be seen.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment is symptom-directed, with the use of diuretics and ACE inhibitors.
- Corticosteroid therapy is indicated for sarcoidosis. Chelation is used for the treatment of hemochromatosis.

- Admission is determined by the severity of the symptoms and the availability of prompt subspeciality follow-up.

DYSRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

- This is the most rare form of cardiomyopathy, and the majority of patients present after an episode of near sudden death. All these patients require extensive workup and hospitalization.
- The ECG has the highest sensitivity and positive predictive value for the diagnosis.[11]

MYOCARDITIS

PATHOPHYSIOLOGY

- Inflammation of the myocardium may be the result of a systemic disorder or an infectious agent.
- Viral etiologies include coxsackie B, echovirus, influenza, parainfluenza, Epstein-Barr, and HIV. Bacterial causes include *Corynebacterium diptheriae, Neisseria meningitides, Mycoplasma pneumoniae,* and β-hemolytic streptococci.[12]
- Pericarditis frequently accompanies myocarditis.

CLINICAL FEATURES

- Systemic signs and symptoms predominate, including fever, tachycardia ''out of proportion'' to the fever, myalgias, headache, and rigors.
- Chest pain due to coexisting pericarditis is frequently present.
- A pericardial friction rub may be heard in patients with concomitant pericarditis.
- In severe cases, there may be symptoms of progressive heart failure (CHF, pulmonary rales, pedal edema, etc.).

DIAGNOSIS AND DIFFERENTIAL

- Nonspecific ECG changes, atrioventricular block, prolonged QRS duration, or ST-segment elevation (in the setting of associated pericarditis) are seen. Chest x-ray is normal.
- Cardiac enzymes may be elevated.[13]
- Differential diagnosis includes cardiac ischemia or infarction, valvular disease, and sepsis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Supportive care is the mainstay of treatment. If a bacterial cause is suspected, antibiotics are appropriate. Many patients have progressive CHF, therefore hospitalization in a monitored environment is usually indicated. (See Chap. 25 for management of CHF.)

PERICARDIAL DISEASE

ACUTE PERICARDITIS

PATHOPHYSIOLOGY

- Inflammation of the pericardium may be the result of viral infection (e.g., coxsackievirus, echovirus, HIV), bacterial infection (e.g., *Staphylococcus, Strep. pneumoniae,* β-hemolytic streptococci, *Mycobacterium tuberculosis*), fungal infection (e.g., *Histoplasma capsulatum*), malignancy (leukemia, lymphoma, melanoma, metastatic breast cancer), drugs (procainamide and hydralazine), radiation, connective tissue disease, uremia, myxedema, post–myocardial infarction (Dressler's syndrome). This condition may also be idiopathic.[14]

CLINICAL FEATURES

- The most common symptom is sudden or gradual onset of sharp or stabbing chest pain that radiates to the back, neck, left shoulder, arm, or trapezial ridge (especially distinguishing).
- The pain is typically aggravated by movement or inspiration and by lying supine. Sitting up and leaning forward reduces the pain.
- Associated symptoms include low-grade intermittent fever, dyspnea, and dysphagia.
- A transient, intermittent friction rub heard best at the lower left sternal border or apex is the most common physical finding.

DIAGNOSIS AND DIFFERENTIAL

- ECG changes come in stages. Initially there is ST-segment elevation in leads I, V_5, and V_6 with PR-segment depression in leads II, aV_F and V_4 through V_6. As the disease resolves, the ST segment nor-

malizes and T-wave amplitude decreases and inverts. In the final stage, the ECG returns to normal. It is difficult to distinguish these ECG changes from those of early repolarization.
- An ST-segment/T-wave amplitude ratio greater than 0.25 in leads I, V_5, or V_6 is indicative of acute pericarditis.[15]
- Pericarditis without other underlying cardiac disease does not typically produce dysrhythmias. Chest x-ray is usually normal but should be done to rule out other disease. Echocardiography is the best diagnostic test.
- Other tests that should be completed include complete blood cell count with differential, blood urea nitrogen and creatinine levels (to rule out uremia), streptococcal serology, appropriate viral serology, other serology (e.g., antinuclear and anti-DNA antibodies), thyroid function studies, erythrocyte sedimentation rate, and creatinine kinase levels with isoenzymes (to assess for myocarditis).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with idiopathic or presumed viral etiologies are treated as outpatients with nonsteroidal anti-inflammatory drugs (e.g., ibuprofen 400 to 600 mg orally four times daily) for 1 to 3 weeks.
- Patients should be treated for a specific cause if one is identified. Any patient with myocarditis or hemodynamic compromise should be admitted into a monitored environment.

NONTRAUMATIC CARDIAC TAMPONADE

PATHOPHYSIOLOGY

- Tamponade occurs when the pressure in the pericardial sac exceeds the normal filling pressure of the right ventricle, resulting in restricted filling and decreased cardiac output.
- Causes include metastatic malignancy, uremia, hemorrhage (excessive anticoagulation), idiopathic disorders, bacterial/tubercular disorders, chronic pericarditis, and others (e.g., systemic lupus erythematosus, postradiation, myxedema).[16]

CLINICAL FEATURES

- The most common complaints are dyspnea and decreased exercise tolerance. Other nonspecific symptoms include weight loss, pedal edema, and ascites.
- Physical findings include tachycardia, low systolic blood pressure, and a narrow pulse pressure. Pulsus paradoxus, neck vein distension, distant heart sounds, and right-upper-quadrant pain (due to hepatic congestion) may also be present. Pulmonary rales are usually absent.

DIAGNOSIS AND DIFFERENTIAL

- Low-voltage QRS complexes and ST-segment elevation with PR-segment depression may be present on the ECG. Chest x-ray is usually normal. An ECG is the diagnostic test of choice.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- An intravenous fluid bolus of 500 to 1000 mL of normal saline may temporarily improve the hemodynamics.
- Pericardiocentesis is both therapeutic and diagnostic. These patients require admission to an intensive care unit or monitored setting.

CONSTRICTIVE PERICARDITIS

PATHOPHYSIOLOGY

- Constriction occurs when fibrous thickening and loss of elasticity of the pericardium results in interference of diastolic filling. Cardiac trauma, pericardiotomy (open-heart surgery), intrapericardial hemorrhage, fungal or bacterial pericarditis, and uremic pericarditis are the most common causes.

CLINICAL FEATURES

- Symptoms develop gradually and mimic those of restrictive cardiomyopathy, including CHF, exertional dyspnea, and decreased exercise tolerance. Chest pain, orthopnea, and paroxysmal nocturnal dyspnea are uncommon.
- On physical exam, patients may have pedal edema, hepatomegaly, ascites, jugular venous distention, and Kussmaul's sign. A pericardial "knock" (an early diastolic sound) may be heard at the apex. There is usually no friction rub.

DIAGNOSIS AND DIFFERENTIAL

- The ECG is not usually helpful but may show low-voltage QRS complexes and inverted T waves.
- Pericardial calcification is seen in up to 50 percent of patients on the lateral chest x-ray.
- Doppler echocardiography, cardiac computed tomography, and magnetic resonance imaging are diagnostic.
- Other diseases that should be considered include acute pericarditis or myocarditis, exacerbation of chronic ventricular dysfunction, or a systemic process resulting in decreased cardiac performance (e.g., sepsis).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- General supportive care is the initial treatment. Symptomatic patients will require hospitalization and pericardiectomy.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 51, ''The Cardiomyopathies, Myocarditis, and Pericardial Disease,'' by James T. Niemann.

28 PULMONARY EMBOLISM

David M. Cline

EPIDEMIOLOGY

- Mortality for pulmonary embolism (PE) ranges from 2 to 10 percent in patients treated for PE and from 20 to 30 percent in those with unrecognized PE. More than 50 percent of fatal PE is unrecognized before autopsy.[1]
- Deep venous thrombosis (DVT) of the lower extremities proves to be the source of 80 to 90 percent of cases.[2]
- The majority of patients with PE will have at least one risk factor, with immobilization being the most prevalent.[3] Risk factors include congestive heart failure (CHF), acute myocardial infarction (MI), chronic obstructive pulmonary disease (COPD), pregnancy, prolonged immobilization, previous history of PE, history of DVT, marked

obesity, burns, malignancy, estrogen use and other hypercoagulable states, surgery in the last 3 months, or lower extremity trauma.

PATHOPHYSIOLOGY

- The pathophysiologic effects are caused by both mechanical obstruction of the pulmonary artery system and the release of vaso- and bronchoactive mediators. These mediators—prostaglandins, catecholamines, serotonin, and histamine—cause bronchoconstriction as well as vasoconstriction of the pulmonary artery.
- Vasoconstriction is the predominant pathophysiologic effect, leading to a ventilation/perfusion mismatch.
- PE tends to be multiple and bilateral, with the right lower lobe of the lung being the most commonly involved lung segment.

CLINICAL FEATURES

- Common symptoms, in decreasing order of frequency, include dyspnea, pleuritic chest pain, anxiety, cough, hemoptysis, sweats, nonpleuritic chest pain, and syncope.[4,5]
- Common signs, in decreasing order of frequency, include respirations >16/min, rales, pulse >100/min, temperature >37.8°C (100.4°F), phlebitis or DVT, cardiac gallop, diaphoresis, edema, and cyanosis.[4] Pleural friction rub and wheezes are infrequent signs of PE.
- The presence or absence of any symptom or sign does not confirm or exclude the diagnosis of pulmonary embolism. Chest pain (usually pleuritic) and dyspnea are the most common symptoms, and tachypnea (respirations >16/min) is the most common sign in the diagnosis of PE.
- Clinical evidence of DVT occurs in less than 50 percent of patients. However, up to 80 percent of patients with PE have positive venography.[2]
- Massive PE (5 percent of cases) presents with hypotension and hypoxia.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis can be excluded or confirmed only with more sophisticated tests, such as a ventilation/perfusion ($\dot{V}/\dot{Q}$) lung scan or pulmonary angiography.
- Hypoxia occurs in about 90 percent of patients with PE, but the Pa_{O_2} may be normal. While a Pa_{O_2} of 80 to 90 mmHg is 90 to 95 percent sensitive in identifying patients with PE, it is only 50 percent specific.[4]
- The presence of an increased alveolar-arterial (A-a) gradient has been reported to be more sensitive for PE but is normal in up to 25 percent of cases.[6] It is calculated with the following formula:

$$\text{A-a gradient} = (150 - 1.2\,[P_{CO_2}]) - Pa_{O_2}$$

- Compare the above value with the expected normal A-a gradient calculated with the formula A-a gradient = patient age/4) − 4. The A-a gradient is less reliable in the elderly.[7] Patients with an increased A-a gradient or hypoxia require further testing to confirm or reject the diagnosis of PE. A recent meta-analysis suggests that A-a gradient is unreliable as a screening test for PE.[8]
- A D-dimer level less than 500 U/mL has a negative predictive value of 87 to 97 percent for PE, depending on the assay method.[8] Clinicians should seek out second-generation tests. However, the D-dimer assay has a high incidence of false positives, up to 80 percent.
- The most common electrocardiographic (ECG) finding is nonspecific ST-T-wave changes. The classic $S_1Q_3T_3$ pattern on the ECG is highly suggestive of PE but is present in only 12 percent of patients.
- The chest x-ray may be normal in up to one-third of patients.[5] Infiltrate or atelectasis will appear in nearly 50 percent of patients. An elevated dome of one diaphragm is seen in 40 percent of patients, often with pleural effusion.[5] Hampton hump, a pleura-based, wedged-shaped infiltrate, is uncommon.
- The Westermark sign, relative oligemia distal to engorged pulmonary arteries, may be seen in patients with massive PE.
- A normal chest x-ray in the setting of dyspnea and hypoxemia without evidence of reactive airway disease is strongly suggestive of PE.[9]
- The $\dot{V}/\dot{Q}$ scan is 98 percent sensitive for PE but only 10 percent specific.[10] A high-probability scan is only 80 percent accurate in diagnosing PE, while a low-probability scan is only 20 percent accurate in excluding the disorder. The combination of a low-probability scan with a low clinical suspicion has a 96 percent predictive value of exclusion of PE, while a high-probability scan in the setting of high clinical suspicion has a 96 percent positive predictive value.[10]
- Pulmonary angiography is the ''gold standard'' for diagnosing PE and is a much more specific test than the $\dot{V}/\dot{Q}$ scan.[5] Angiography exposes pa-

tients—especially the elderly—to more potential complications.
- Disorders in the differential diagnosis include respiratory disorders, such as asthma, COPD, pneumonia, spontaneous pneumothorax, and pleurisy. Cardiac disorders that may mimic PE include MI and pericarditis. Musculoskeletal disorders that may mimic PE include muscle strain, rib fracture, costochondritis, and herpes zoster. Intraabdominal disorders that irritate the diaphragm or stimulate breathing may also present similarly to PE. Finally, hyperventilation syndrome may mimic PE; however, this is a diagnosis of exclusion.
- Spiral computed tomography (CT) scanning is an excellent confirmation test (experience may vary at different medical centers). Spiral CT is 93 to 98 percent specific for pulmonary embolism.[8,11]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of PE consists of initial stabilization, anticoagulation with heparin, and thrombolytic therapy in emergent cases.
- Administer oxygen.
- Crystalloid IV fluids should be given initially for hypotension.
- For hypotension in the absence of hypovolemia, dopamine can be started at 2 to 5 μg/kg/min and titrated to maintain a systolic blood pressure of 90 mmHg.
- Start heparin with an IV bolus of 10,000 to 20,000 U, followed by a continuous drip of 1000 U/h to be adjusted using the partial thromboplastin time, aiming for an international normalized ratio (INR) of two to three times normal. Contraindications to anticoagulation include active internal bleeding, uncontrolled severe hypertension, recent trauma, recent surgery, recent stroke, and intracranial or intraspinal neoplasm. Heparin can be used safely in the nonbleeding pregnant patient but must be discontinued prior to delivery. Heparin does not prevent the embolization of existing clots.
- Low-molecular-weight heparin has been shown to be safe and effective in the treatment of DVT and PE. Examples include enoxaparin 1 mg/kg SQ as the initial dose.
- For persistent hypotension despite medical management with the above measures, consider thrombolytic therapy. Tissue plasminogen activator (tPA), 50 to 100 mg IV over 2 to 6 h, has been recommended. Streptokinase can be given in a dose of 250,000 U IV over 30 min followed by a continuous IV infusion of 100,000 U/h for the next 12 to 24 h. Ideally, consultation with an intensivist should occur prior to starting thrombolytic therapy.
- For patients with contraindications to anticoagulation or thrombolytic therapy, a Greenfield filter is recommended.
- Further embolization and shock most commonly occur within 4 h of initial symptoms.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 52, ''Pulmonary Embolism,'' by Charles N. Schoenfeld.

29 HYPERTENSIVE EMERGENCIES

Jonathan A. Maisel

EPIDEMIOLOGY

- Hypertension is the fourth most prevalent chronic medical condition in the United States, affecting up to 24 percent of the general adult population.[1,2]
- The risk of developing serious cardiovascular, renal, or cerebrovascular disease increases with poorly controlled blood pressure.
- Nearly 75 percent of adult Americans with known hypertension have inadequate control of their blood pressure, and only one-half are compliant with prescribed medications.[2,3]

PATHOPHYSIOLOGY

- At the cellular level, postsynaptic α_1 and α_2 receptors are stimulated by norepinephrine released from presynaptic sympathetic nerve endings, leading to the release of intracellular calcium. Free calcium activates actin and myosin, resulting in smooth muscle contraction, increased peripheral vascular resistance, and an increase in blood pressure. Presynaptic α_2 receptors help limit this response via a negative-feedback loop.
- Hypertension develops: (*a*) as a result of alterations in the contractile properties of smooth muscle in arterial walls, or (*b*) as a response to failure of normal autoregulatory mechanisms within vascular beds of vital organs (i.e., heart, kidney, and brain).
- Long-standing, poorly controlled hypertension may damage target organs by injuring vascular beds. Endothelial injury leads to deposition of fibrin within vessel walls, and activation of mediators of coagulation and cell proliferation.[4] A recurrent cycle of vascular reactivity develops which leads to platelet aggregation and myointimal proliferation, and subsequent progressive narrowing of arterioles.
- Hypertension is associated with major cardiovascular risk factors such as smoking, hyperlipidemia, diabetes mellitus, age >60, gender (men and postmenopausal women), obesity, and a family history of cardiovascular disease.[3] Although no single cause of hypertension has been identified, a combination of factors such as these are believed to contribute to ''essential'' hypertension. Several specific causes do exist, with intrinsic renal and renovascular disease being the most prevalent of the known causes.
- Hypertensive emergencies in childhood, defined as systolic or diastolic blood pressure ≥95th percentile for age and sex, are most commonly caused by intrinsic renal or renovascular disease.

CLINICAL FEATURES

- Essential historical features include a prior history of hypertension; noncompliance with anti-hypertension medication; overall medication use, including over-the-counter and illicit drugs; and diet (especially products with sodium or tyramine).
- Any past medical history of cardiovascular, cerebrovascular, or renal disease; diabetes; hyperlipidemia; chronic obstructive pulmonary disease; or asthma; or a family history of hypertension or premature heart disease should be elicited.[3]
- Precipitating causes such as pregnancy, illicit drug use (i.e., cocaine and methamphetamine), monoamine oxidase inhibitors, and decongestants should be considered.
- Patients should be asked about central nervous system (CNS) symptoms (headache, visual changes, confusion, paresis, seizures), cardiovascular symptoms (chest pain, dyspnea, palpitations, pedal edema, tearing pain radiating to the back or abdomen), and renal symptoms (anuria, hematuria, edema).
- Blood pressure should be measured with an appropriately sized cuff (false elevations with small cuffs), at least twice if elevated, and in both arms and legs if substantially elevated.
- The physical exam should focus on target organ injury and its acuity, including mental status changes, focal neurologic deficits, funduscopic changes (hemorrhages, cotton-wool exudates, disk edema), and cardiovascular findings (carotid bruits, heart murmurs and gallops, asymmetric pulses—coarctation versus dissection, pulmonary rales, and pulsatile abdominal masses).[3]
- In the pregnant or postpartum patient, assessment should be made for hyperreflexia and peripheral edema, suggesting preeclampsia.
- Children present with nonspecific complaints such as a throbbing frontal headache or blurred vision. Physical findings are similar to those seen in adults.
- Pheochromocytoma is another common etiology in childhood, presenting with nervousness, palpitations, sweating, blurry vision, and skin flushing.

DIAGNOSIS AND DIFFERENTIAL

- Renal impairment may present as hematuria, proteinuria, red blood cell casts, or elevations in blood urea nitrogen (BUN), creatinine, and potassium levels.
- An electrocardiogram may reveal ST-T wave changes consistent with coronary ischemia, electrolyte abnormalities, strain, or left ventricular hypertrophy.
- A chest x-ray may help identify congestive heart failure, aortic dissection, or coarctation.
- In patients with neurologic compromise, a computed tomography scan of the head may reveal ischemic changes, edema, or blood.
- A urine or serum drug screen may identify illicit drug use.
- A pregnancy test should be done on all hypertensive women of childbearing age.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Though hypertension is defined as either a systolic blood pressure >140 mmHg or a diastolic blood pressure >90 mmHg, management depends more on the patient's clinical condition rather than absolute systolic or diastolic values.
- Classification of hypertension into four categories facilitates management:
  a. Hypertensive emergency: Elevated blood pressure associated with target organ (CNS, cardiac, renal) dysfunction. Requires immediate recognition and treatment.
  b. Hypertensive urgency: Elevated blood pressure associated with risk for imminent target organ dysfunction.
  c. Acute hypertensive episode: Systolic blood pressure >180 and diastolic blood pressure >110 without evolving or impending target organ dysfunction.
  d. Transient hypertension: Elevated blood pressure associated with another condition (e.g., anxiety, alcohol withdrawal, and cocaine abuse). Patients usually become normotensive once the precipitating event resolves.
- Patients with hypertensive emergencies require O_2 supplementation, cardiac monitoring, and intravenous access. Following attention to the ABCs of resuscitation, the treatment goal is to reduce the mean arterial pressure [diastolic blood pressure + 1/3 (systolic blood pressure − diastolic blood pressure)] by 20 to 25 percent over 30 to 60 min.
- For hypertensive encephalopathy, sodium nitroprusside should be used, beginning at 0.5 μg/kg/min and titrating to a maximum of 10 μg/kg/min. Rapid correction of blood pressure should be avoided to prevent cerebral ischemia secondary to hypoperfusion. Nitroprusside is a potent arteriolar and vasodilator, with an onset of action in seconds. An arterial line should be placed in order to closely monitor the blood pressure, and the solution and tubing should be wrapped in aluminum foil to prevent degradation by light. Hypotension is the most common complication of nitroprusside infusions. Cyanide toxicity is seen rarely after prolonged infusions.
- Labetalol is useful as a second line agent for hypertensive encephalopathy, providing a steady, consistent drop in blood pressure without diminishing cerebral blood flow or producing a reflex tachycardia. It is a competitive, selective α_1 blocker, and a competitive, nonselective β blocker, with the β-blocking action 4 to 8 times more potent than the α-blocking action. It has an onset of action in 5 to 10 min, and a duration of action of 8 h. Its use should be avoided in patients with asthma, chronic obstructive pulmonary disease, congestive heart failure, and heart block. The treatment should begin with incremental boluses of 20 to 40 mg intravenous (IV) and repeated every 10 min until the target blood pressure is achieved or a total dose of 300 mg is reached. Alternatively, after an initial bolus, a continuous infusion of 1 to 2 mg/min may be used, terminating the infusion when the target blood pressure has been achieved. Labetalol is also ideal for use in syndromes associated with excessive catecholamine stimulation.
- Hypertension associated with stroke is often a physiologic response to the stroke itself (to maintain adequate cerebral perfusion) and not its immediate cause. When the diastolic blood pressure is >140 mmHg, it may be slowly reduced by up to 20 percent using 5 mg increments of IV labetalol. The acute management of hypertension associated with intracranial hemorrhage is controversial.
- For hypertension associated with pulmonary edema, IV nitroglycerine or nitroprusside may be used. Nitroglycerine is both an arteriolar and venous dilator, with greater effect on the venous system, and an onset of action within minutes. Initial infusion should be at a rate of 5 to 20 μg/min, with 5 μg/min incremental increases every 5 min until symptoms improve or side effects (headache, hypotension, tachycardia) ensue.
- For hypertension associated with myocardial ischemia, IV nitroglycerine is first-line therapy. Because it is a better vasodilator of the coronary

vessels than nitroprusside, it is the drug of choice for severe hypertension complicating acute coronary ischemia or pulmonary edema.

- For hypertension associated with aortic dissection, reducing the blood pressure and ventricular ejection force may limit the extent of the dissection. Either labetalol alone, or a combination of nitroprusside and a beta blocker can be used. Esmolol, an ultra-short-acting β_1-selective adrenergic blocker, is very effective, achieving 90 percent of beta blockade within 5 min of an IV bolus of 0.5 mg/kg, followed by an infusion of 0.05 to 0.3 mg/kg/min. Propranolol and metoprolol are alternatives. Esmolol, as well as other beta blockers, should be avoided in patients with asthma, chronic obstructive pulmonary disease, and cocaine-induced cardiovascular complications (because of unopposed α-adrenergic effects).
- Worsening renal function in the setting of elevated blood pressure, manifested by elevation of BUN and creatinine levels, proteinuria, or the presence of red blood cells or red blood cell casts in the urine, is considered a hypertensive emergency. Nitroprusside is the preferred agent. Dialysis-dependent patients presenting with volume overload may require emergent dialysis if they present with uncontrolled hypertension and other evidence of end-organ dysfunction.
- Renovascular hypertension in children can be treated with diazoxide 1 to 3 mg/kg IV q5 to 15 min, labetalol 0.3 to 1 mg/kg IV q10 min, or captopril 0.5 to 1 mg/kg per 24 h PO in 3 to 4 divided doses.
- Treatment of pheochromocytoma requires surgical excision, managing the elevated blood pressure with an α-adrenergic blocker such as phentolamine.
- The treatment goal in hypertensive urgencies is the gradual reduction of blood pressure within 24 to 48 h by using oral antihypertensive agents. Useful agents include the following:
  a. Labetalol 200 to 400 mg PO, repeated every 2 to 3 h. Oral labetalol has an onset of action in 30 min and a duration of action of 6 to 12 h.
  b. Captopril, an angiotensin-converting enzyme inhibitor, has an onset of action in 15 to 30 min, a peak effect at 50 to 90 min, and a duration of effect of 4 to 6 h. A 25 mg oral dose is effective in refractory congestive heart failure and renovascular hypertension. Common side effects include rash, cough, and loss of taste, and rarely, life-threatening angioneurotic edema.
  c. Sublingual nitroglycerine in the form of spray, or 0.3 to 0.6 mg tablets, are the agents of choice for patients with angina or congestive heart failure. The hypotensive effect begins in minutes and can last several hours.
  d. Clonidine is a centrally acting, α_2-adrenergic agonist that decreases central sympathomimetic activity, lowering plasma catecholamine levels. Its onset of action is 30 to 60 min, with peak effect in 2 to 4 h. It is given as a dose of 0.1 mg hourly until the target blood pressure is reached, or a total of 0.7 mg has been given. A patient treated with clonidine in the emergency department (ED) does not need to be discharged on this drug. Because an adequate response may take up to 6 h, it is not a first-line agent.
  e. Nifedipine, a dihydropyridine Ca^+-channel antagonist, had been used commonly for hypertensive urgencies via oral and sublingual routes. Serious adverse reactions, such as acute coronary events and stroke, preclude recommending it for the urgent treatment of hypertension.[5]
- For nonemergent, nonurgent hypertension, there is no evidence of a beneficial effect of acute blood pressure reduction on long-term control or on the chronic effects of hypertension. These patients do not require acute intervention, but should be referred for timely follow-up. Should an oral agent be started in the ED, the choice should be based on coexisting conditions, if any. Diuretics, such as hydrochlorothiazide 25 mg/day, are first-line agents in the elderly, as well as for patients with renal disease and congestive heart failure. (Consider potassium supplementation.) Beta blockers, such as metoprolol 50 mg bid are first-line agents for patients with angina, or those postmyocardial infarction. Angiotensin-converting enzyme inhibitors, such as captopril 25 mg two to three times a day, can be used in patients with congestive heart failure or diabetes.
- For a discussion of hypertension associated with pregnancy, see Chap. 61.

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30 AORTIC DISSECTION AND ANEURYSMS

Suzanne M. Bertollo

ABDOMINAL AORTIC ANEURYSMS

EPIDEMIOLOGY

- Incidence of abdominal aortic aneurysms (AAA) increases with age; most patients are older than 60.
- Males are at increased risk.
- Other risk factors include connective tissue disease, Marfan syndrome, atherosclerotic risk factors (smoking, hypertension, hyperlipidemia, and diabetes) and a family history of aneurysm.

PATHOPHYSIOLOGY

- Destruction of the media of the aorta is a prominent feature in aneurysm pathogenesis with a reduction of elastin and collagen. Histologic examination reveals a thinned media and an intima that is infiltrated with atherosclerosis.
- Laplace's law [wall tension = (pressure x radius)/tensile force] dictates that as the aorta dilates, the force on the aortic wall increases, causing further aortic dilation. Rate of aneurysmal dilation is variable with larger aneurysms expanding more quickly. An average rate may be .25 to 0.5 cm per year.[1]

CLINICAL FEATURES

- Four clinical scenarios exist: acute rupture, aortoenteric fistula, chronic contained rupture, and AAA as an incidental finding.
- Acute leakage or rupture is rapidly fatal without intervention. Classic presentation is an older male with severe back or abdominal pain who presents with syncope or hypotension. On exam, such patients classically have a tender pulsatile abdominal mass, but this finding may be obscured by obesity. Femoral pulsations are typically normal.[2]
- Patients usually exhibit a variation of the classic presentation.[3] They may complain of unilateral flank or groin pain, hip pain, or abdominal pain localized to a specific quadrant. Abdominal tenderness may or may not be present. There may be signs of retroperitoneal hemorrhage such as periumbilical or flank ecchymosis or scrotal hematoma.
- Aortoenteric fistula presents as gastrointestinal bleeding. This is classically seen in a patient who has undergone prior aortic grafting.[4] These patients may present with a deceptively minor ''sentinel'' bleed or massive gastrointestinal hemorrhage.
- Chronic-contained rupture is uncommon but is seen when an AAA ruptures retroperitoneally with significant fibrosis that limits blood loss.[5] These patients may have pain for an extended time period and appear well.
- Asymptomatic AAAs may be found on physical exam or during unrelated radiologic evaluation. Those greater than 5 cm are at high risk of rupture.

DIAGNOSIS AND DIFFERENTIAL

- Variable presentations of aortic aneurysm present a diagnostic challenge. Diagnoses that might be considered are renal colic, musculoskeletal back pain, pancreatic disease or other intraabdominal processes (diverticulitis, cholecystitis, mesenteric ischemia, etc.), scrotal or testicular disorders, and disorders that cause gastrointestinal bleeding (varices, ulcers, tumors, etc.).
- Diagnostic studies are needed when the diagnosis of AAA is unclear. Though not the study of choice, plain films may reveal a calcified aorta in 65 percent of those with aneurysmal disease.[6] In the unstable patient, bedside abdominal ultrasound is very sensitive, reliably measuring aortic diameter and identifying an aneurysm[7] without the hazards of transporting a patient away from the emergency department. Computed tomogra-

phy scanning, however, is preferred in the stable patient as it better delineates the anatomic details of the aneurysm and any associated rupture.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The patient should be stabilized with supplemental oxygen, volume resuscitation with isotonic fluids, and/or blood transfusion via multiple large bore intravenous lines.
- For suspected rupturing AAA or aortoenteric fistula, immediate surgical consultation is warranted. No diagnostic testing should delay surgical repair.
- A vascular surgeon should be consulted for urgent repair of chronically contained ruptured AAAs. Admission to the intensive care unit should be sought.
- For incidentally discovered AAA, the patient can potentially be discharged home depending on aneurysm size and comorbid features. Consultation with a vascular surgeon for admission or close outpatient follow-up is usually adequate.

AORTIC DISSECTION

EPIDEMIOLOGY

- Most patients are over the age of 50 years with a history of hypertension.
- A second group of patients are younger than 50 years and have identifiable risk factors such as congenital heart disease, connective tissue disease, and pregnancy. Twenty-five to 30 percent of patients with Marfan syndrome develop dissection. Dissection may also be iatrogenic from cardiac catheterization or surgery.

PATHOGENESIS

- Aortic dissection occurs when the intima is violated, allowing blood to enter the media and dissect between the intimal and adventitial layers. Common sites for tear include the ascending aorta and the region of the ligamentum arteriosum.
- Dissections may extend proximally, distally, or both and are classified by two separate systems. The Stanford classification system considers any involvement of the ascending aorta a type A dissection and one restricted to the descending aorta a type B dissection. The DeBakey system classifies type I dissections as those that involve the ascending aorta, the arch, and the descending aorta. Type II involves only the ascending aorta and type III only the descending aorta.

CLINICAL FEATURES

- More than 90 percent of patients have abrupt onset of severe tearing chest or upper back pain. Accompanying nausea, vomiting, and diaphoresis are common.
- Clinical presentation depends on the location of the dissection with pain patterns often changing as the anatomic injury migrates.[8] Presentations include aortic valve insufficiency with or without pericardial tamponade, coronary artery occlusion with myocardial infarction, stroke symptoms with carotid involvement, or paraplegia with occlusion of vertebral blood supply. The dissection may progress distally causing abdominal or flank pain or limb ischemia.
- Physical exam findings also depend on location and progression of the dissection. A diastolic murmur or aortic insufficiency may be heard. Fifty percent of patients have decreased radial, femoral, or carotid pulses.[8] Hypertension and tachycardia are common, but hypotension may also be present.

DIAGNOSIS AND DIFFERENTIAL

- Ischemic end organ manifestations associated with dissections may confuse the differential diagnosis, which includes myocardial infarction, pericardial disease, pulmonary disorders, spinal cord injuries, and intraabdominal disorders. Rupture of the dissection back through the intima into the true lumen may cause a cessation of symptoms leading to false reassurance.
- Chest x-ray shows an abnormal aortic contour 90 percent of the time. The ''calcium sign'' may be present, with intimal calcium seen distant from the edge of the aortic contour.
- Computed tomography, angiography, and transesophageal echocardiography are all quite sensitive and specific. Their use varies by institution[9] and should be based on availability and patient stability, in conjunction with a vascular or thoracic surgeon.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with suspected aortic dissection require prompt radiographic confirmation of the diagnosis.
- Stabilization of the patient requires large-bore intravenous access, supplemental oxygen, and correction of hypotension with judicious fluid and/or blood product resuscitation.
- More commonly patients with dissection require antihypertensive treatment along with control of tachycardia to reduce shear force on the intimal flap of the aorta. This is generally accomplished with negative inotropes (esmolol, metoprolol, or propranolol) in conjunction with a vasodilator such as nitroprusside.
- Rapid consultation with a surgeon is mandatory. Dissection of the ascending aorta requires prompt surgical repair. Indications for repair of dissections involving only the descending aorta are controversial.[9]

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31 NONTRAUMATIC PERIPHERAL VASCULAR DISORDERS

David M. Cline

DEEP VENOUS THROMBOSIS

- Deep venous thrombosis (DVT) is a common potentially life-threatening condition with an estimated annual incidence of 5 to 20 million cases in the United States.

PATHOPHYSIOLOGY

- The formation of venous clots is related to at least one of Virchow's triad of factors: venous stasis, injury to the vessel wall, and a hypercoagulable state. Table 31-1 outlines the clinical risk factors predisposing to DVT, which can be remembered by the mnemonic *thrombosis.*
- Thrombi most commonly form at the venous cusps of deep veins in the lower extremities, where altered or static blood flow initiates clot formation.

CLINICAL FEATURES

- The classic features of DVT include swelling of the lower extremity, tenderness, pain, redness, in-

TABLE 31-1 Clinical Risk Factors for Deep Venous Thrombosis

T	Trauma
H	Hypercoagulable, hormone replacement
R	Recreational drugs (IV drugs)
O	Old (age >40)
M	Malignancy
B	Birth control pill, blood group A
O	Obesity/obstetrics
S	Surgery, smoking
I	Immobilization
S	Sickness

creased local warmth, and possibly low-grade fever.
- The clinical examination is unreliable for the detection or exclusion of DVT. Assessment of risk factors (Table 31-1) may be a stronger predictor whenever the diagnosis is entertained.
- One study showed that a single risk factor is associated with DVT in 24 percent of patients, while those with four or more risk factors are virtually certain to have the diagnosis established.[1]
- The constellation of pain, redness, swelling, warmth, and tenderness is present in less than one-half of patients with confirmed DVT. Swelling and tenderness in the involved extremity are the most common findings, occurring in 80 and 75 percent, respectively, of patients with DVT.
- Pain in the calf with forced dorsiflexion of the ankle and the leg straight (Hormans' sign) is not reliable for DVT.
- Symptomatic DVT will be in the popliteal or more proximal veins in more than 80 percent of cases.
- An isolated calf DVT will extend proximally only 20 percent of the time, usually within a week of presentation.[2] Unlike proximal DVT, nonextending calf DVT will rarely cause a pulmonary embolism.
- Uncommon presentations of DVT include phlegmasia cerulea dolens (painful blue inflammation) and phlegmasia alba dolens (''milk leg'').
- In phlegmasia cerulea dolens the patient presents with an extensively swollen, cyanotic leg from venous engorgement due to massive iliofemoral thrombosis. This high-grade obstruction can compromise perfusion to the foot from high compartment pressures and lead to venous gangrene. Petechiae and bullae may be present on the skin.
- Phlegmasia alba dolens is also due to massive iliofemoral thrombosis, but the patient's leg is pale or white secondary to associated arterial spasm.

DIAGNOSIS AND DIFFERENTIAL

- Less than one-third of patients with clinically suspected DVT are found to have the disease following objective investigation.[2]
- Venography has represented the historical ''gold standard'' for the detection of DVT. When contrast is seen throughout the deep venous system (not possible in 5 to 10 percent of tests), a venogram reliably excludes DVT.
- The most common test used to identify a DVT in North America is ultrasonography.
- A duplex scan with or without color flow is highly sensitive and specific for a proximal DVT (clot proximal to the popliteal veins). The positive predictive value of ultrasound is higher than impedance plethysmography (IPG) for DVTs (94 versus 83 percent, respectively).
- D-dimer fragments can be measured as an indicator of the presence or absence of DVT or pulmonary embolism.[3,4] Infections, surgery, trauma, cardiovascular disease, and cancer can elevate a D-dimer level. Despite a sensitivity less than 250 ng/mL of over 80 to 90 percent, a D-dimer level is useful only when it is low.[3]
- The combination of a normal IPG or ultrasound and low D-dimer level has a negative predictive value of about 99 percent for proximal DVT.[2]
- The primary objective in treating DVT is the prevention of pulmonary embolism. The mainstay of therapy is anticoagulation.
- In the setting of ultrasound-documented proximal DVT with other complications, hospital admission is appropriate.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Either low-molecular-weight heparin (LMWH) or unfractionated heparin (with weight-based dosing of a bolus of 80 U/kg followed by an infusion of 18 U/kg/h) may be used.[5] The available LMWHs include dalteparin, enoxaparin, or tinzaparin.[6] An example treatment regimen would be enoxaparin 1 mg/kg of lean body weight subcutaneously twice daily. When using unfractionated heparin, the goal is a PTT of 1.8 to 2.8 times normal.
- If anticoagulation is contraindicated, if a clot is extending proximally in spite of medical treatment, or if there is significant bleeding with the anticoagulants, consultation should be obtained for the placement of a Greenfield filter in the inferior vena cava.
- In the setting of ultrasound-documented proximal DVT, discharge to home on LMWH can be considered.[6] The patient should have few or no comorbid illnesses, be able to ambulate unassisted, have good social support at home, have a physician familiar with the use of LMWH who can follow up with the patient within 24 h, be able and willing to self-administer injections at home, and have no other reason for admission to the hospital. Warfarin therapy would then be initiated by the follow-up physician.
- In the setting of unilateral leg swelling and an ultrasound negative for venous thrombosis proximal to the popliteal fossa (presumed calf DVT), discharge with a follow-up ultrasound in 5 to 7 days is recommended.[7] Generally, no anticoagulation needs to be started except in very high risk

groups including those with previous proximal DVT or pulmonary embolus, poor ambulation, a known hypercoagulable state, or extensive cardiovascular comorbidity. With a known or presumed calf DVT, the risk of pulmonary embolus within 7 days after an initial negative ultrasound is near 0, even without anticoagulation.[2]

OCCLUSIVE ARTERIAL DISEASE

EPIDEMIOLOGY

- Intermittent claudication has a prevalence of between 1 and 7 percent for men above age 50, with symptomless disease existing in up to 25 percent of men scanned with noninvasive testing in this age group.[8]
- Symptoms of peripheral arterial disease increase with age and are two to four times more common in men than in women. The vast majority of these patients have a history of prolonged smoking.
- Given that atherosclerosis is the usual pathology in ischemic limb pain, it is not surprising that at least one-half of these patients have coronary or cerebrovascular disease.[8]

PATHOPHYSIOLOGY

- Acute limb ischemia results from a blood supply that is inadequate to meet tissue oxygen and nutrient requirements.
- Peripheral nerves and skeletal muscle are very sensitive to ischemia; in them, irreversible changes occur within 4 h of anoxia at room temperature.
- Nonembolic limb ischemia is secondary to atherosclerosis in the vast majority of patients.[9]
- An embolus is the commonest cause of an acute arterial occlusion in the limb and originates from the heart in 80 to 90 percent of cases of embolism. Atrial fibrillation and recent myocardial infarction are the two primary causes of mural thrombus within the heart.
- Other causes include thrombosis, inflammatory condition, low flow states, and arterial dissection.

CLINICAL FEATURES

- Patients with acute limb ischemia will exhibit one or more of the "six Ps": pain, pallor, polar (for cold), pulselessness, paraesthesias, and paralysis. A lack of one or more of these findings, however, does not exclude ischemia.
- Pain alone may be the earliest symptom.
- Complete arterial obstruction results in visible skin changes, with initial pallor that may be followed by blotchy, mottled areas of cyanosis and associated petechiae and blisters. Severe, steady pain in the involved extremity associated with decreased skin temperature is expected.
- Hypoesthesia or hyperesthesia due to ischemic neuropathy is typically an early finding, as is muscle weakness.
- An absent distal pulse is only so helpful. It may be an abrupt new sign of an occlusive clot or a long-standing finding of chronic vascular disease.
- Despite the generally held belief that limb salvage is possible with reperfusion within 4 to 6 h, tissue loss can occur with significantly shorter occlusion times.
- Disability and tissue loss are inevitable after 6 h of occlusion anoxic injury.
- Chronic peripheral arterial insufficiency is characterized by intermittent claudication, which may progress to intermittent ischemic pain at rest.
- Pain at rest typically localizes to the foot and is aggravated with leg elevation, improves with standing, and is poorly controlled with analgesics.[8] Shiny, hyperpigmented skin with hair loss and ulceration, thickened nails, muscle atrophy, vascular bruits, and poor pulses is a hallmark of chronic vascular disease.

DIAGNOSIS AND DIFFERENTIAL

- A thorough clinical evaluation is the most useful diagnostic tool for the assessment of occlusive arterial disease. A history of an abruptly ischemic limb in a patient with atrial fibrillation or recent myocardial infarction is highly suggestive of an embolus. Acute ischemia in the limb of a patient known to have advanced peripheral vascular disease is more likely due to thrombosis or a low cardiac output state.
- A hand-held Doppler can document the amplitude of flow or its absence when held over the dorsalis pedis, posterior tibial, popliteal, or femoral arteries in the lower limb and over the radial, ulnar, brachia, or axillary arteries in the arm.
- In consultation with a vascular surgeon and during the period of preoperative and/or medical management, an arteriogram can be done to confirm the diagnosis, define the vascular anatomy and perfusion, and guide aggressive management.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- When the diagnosis of acute limb ischemia is known or suspected, immediate intravenous hepa-

rinization should be started if no contraindications exist.[9]

- Prompt surgical embolectomy is the optimal therapy for an acute arterial embolism causing limb-threatening ischemia. Catheter embolectomy has been the choice technique for removal of clot ever since the development of the Fogarty balloon catheter in 1963.[9] It has reduced mortality from arterial emboli by 50 percent and need for amputation by 35 percent.
- Overall mortality from an arterial embolus is about 15 percent and is usually due to the underlying cardiovascular disease. The limb salvage rate ranges from 62 to 96 percent.[9]
- Intraarterial thrombolysis with streptokinase, urokinase, or tissue plasminogen activator (tPA) infused near or into the clot for a few hours to days is an alternative to surgery, with a rate of successful reperfusion of 50 to 85 percent.[9]
- Systemic thrombolysis has been compared with intraarterial lytic agents in randomized trials and has been shown to produce inferior results.[9]

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Section 7
PULMONARY EMERGENCIES

32 RESPIRATORY DISTRESS

Matthew T. Keadey

DYSPNEA

PATHOPHYSIOLOGY

- Dyspnea is a subjective feeling of difficult, labored, or uncomfortable breathing. It is a complex sensation, without a defined neural pathway, derived from many sources including mechanical, chemical, and vascular receptors.[1]
- Mechanical factors include a sense of skeletal muscle effort dependent on work of breathing and intraparenchymal stretch and irritant receptors in the lungs that respond to changes in compliance and edema.
- Chemoreceptors in the central medulla and the carotid body respond to changes in CO_2 and O_2, respectively. Receptors in the atrium(s) and pulmonary arteries also contribute in a poorly defined manner.
- Central and peripheral receptors send afferent neurons to the central nervous system, where the information is integrated in a complex manner.

CLINICAL FEATURES

- The patient may present with shortness of breath or breathlessness, tachypnea, tachycardia, use of accessory respiratory muscles, and stridor.
- The complaint of dyspnea must be rapidly evaluated, including abnormal vital signs and the primary survey [airway, breathing, circulation (ABCs)]. Airway obstruction, ineffective respiratory effort, and changes in mental status may necessitate rapid airway control and intervention.[2]
- Lesser degrees of dyspnea allow for a more detailed history and exam (Table 32-1).

DIAGNOSIS AND DIFFERENTIAL[3–6]

- Ancillary studies useful in determining a diagnosis include pulse oximetry and arterial blood gas analysis, but tests should be taken in light of work of breathing.
- A chest x-ray, electrocardiogram, peak flows, and a hemoglobin or hematocrit may also be useful.
- Other ancillary tests include spirometry/pulmonary function testing, cardiac stress testing, echocardiography, exercise testing, electromyography, ventilation/perfusion scan; pulmonary biopsy may also be useful in the appropriate setting.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The first priority is to recognize threats to life and aggressively support respiratory function. Supplemental oxygen is given to maintain Pa_{O_2} >60 mmHg (pulse oximeter >91 to 93 percent). Patients with chronic obstructive pulmonary disease (COPD) may tolerate a lower Pa_{O_2}.
- Further interventions include continuous positive airway pressure (CPAP) or biphasic positive airway pressure (BiPAP) ventilation, bag-valve-mask ventilation, and intubation with mechanical ventilation.
- All patients with an unclear cause of dyspnea and hypoxia require admission to a monitored bed.

TABLE 32-1 Common Causes of Dyspnea

AIRWAY	CARDIAC	LUNG PARENCHYMAL	PLEURAL AND CHEST WALL	VASCULAR	NEUROMUSCULAR	MISCELLANEOUS
Airway mass	Left ventricular failure	Asthma	Pneumothorax	Pulmonary embolism	Cerebrovascular accident	Anemia
Foreign body	Myocardial ischemia	COPD	Pleural effusion	Air embolism	Phrenic nerve paralysis	Metabolic acidosis
Angioedema	Pericarditis	Pneumonia	Pleural adhesions	Fat embolism	Guillain-Barré syndrome	Shock
Airway stenosis	Pericardial tamponade	Pulmonary edema	Chest wall injury	Amniotic embolism	Tick paralysis	Low cardiac output states
Bronchiectasis	Arrhythmia	Pulmonary contusion	Abdominal distention	Pulmonary hypertension	Botulism	Hypoxia
Tracheomalacia	Myocarditis	Atelectasis	Kyphoscoliosis	Venoocclusive disease	Neuropathy	Carbon monoxide poisoning
	Cardiomyopathy	Alveolitis	Pectus excavatum	Sickle cell disease	Myopathy	Methemoglobinemia
	Intracardiac shunt	Pulmonary fibrosis	Pregnancy	Vasculitis		Deconditioning
	Left ventricular outflow obstruction	Adult respiratory distress syndrome		Arteriovenous fistula		Fever
	Valvular disorder	Sarcoidosis				Hyperthyroidism
	Hypertensive crisis					Hypothyroidism
						Gastroesophageal reflux
						Psychogenic hyperventilation

HYPOXIA

PATHOPHYSIOLOGY

- Hypoxia is defined as the inadequate delivery of oxygen to the tissues and is caused by one of five distinct mechanisms. Hypoxia is arbitrarily defined as Pa_{O_2} <60 mmHg.
- *Hypoventilation:* Rising Pa_{CO_2} displaces oxygen from the alveolus, lowering the Pa_{O_2} and decreasing the O_2 diffusion gradient across the pulmonary membrane.
- *Right-to-left shunting:* Unoxygenated blood enters the systemic circulation. This may occur secondary to perfusion of underventilated lung or with congenital heart anomalies.
- *Ventilation/perfusion mismatch:* Results from regional alterations of ventilation or perfusion.
- *Diffusion impairment:* Caused by impairment of the alveolar blood barrier.
- *Low* Fi_{O_2}: The cause of high-altitude hypoxia.

CLINICAL FEATURES

- Signs and symptoms are nonspecific, ranging from tachycardia and tachypnea to central nervous system (CNS) manifestations such as agitation, seizures, and coma.
- At Pa_{O_2} <20 mmHg, there is a paradoxical depression of the respiratory drive.
- Dyspnea may or may not be present, and cyanosis is an insensitive indicator of Pa_{O_2} status.

DIAGNOSIS AND DIFFERENTIAL

- Pulse oximetry is a useful screening test, but arterial blood gas analysis defines the diagnosis.
- Similar ancillary tests used to determine causes of dyspnea might elucidate abnormalities leading to hypoxia.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Hypoxia is treated the same as dyspnea; support, identify, and aggressively treat underlying disorders, trying to maintain Pa_{O_2} >60 mmHg.
- All patients with persistent hypoxia require hospitalization until the abnormality is adequately addressed and stabilized. Frequent arterial blood samples may require an arterial line.

HYPERCAPNIA

PATHOPHYSIOLOGY

- Hypercapnia is defined as a Pa_{CO_2} >45 mmHg and is caused by hypoventilation. It is almost never caused by intrinsic lung disease or increased CO_2 production. Minute ventilation is dependent on respiratory rate and tidal volume; decreases in either will lead to hypoventilation. Disorders leading to hypoventilation and hypercapnia are varied, but their effect can always be traced to the minute ventilation relationship.
- *Alveolar ventilation* is less than minute ventilation; although this term is more appropriately used in describing ventilation, alveolar ventilation is impractical to measure. It is dependent on the tidal volume less the anatomic dead space and the respiratory rate. Dead space is the volume of air that must be inhaled to initially reach the alveolus and is made up of the large conducting airways.
- Both parameters in minute ventilation are controlled via efferent neuronal output from the chemoreceptor in the medulla.

CLINICAL FEATURES

- Signs and symptoms of hypercapnia are dependent on the rate and degree of elevation. Acute rises are associated with an increase in intracranial pressure, confusion, lethargy, seizures, and coma. On physical exam, asterixis may also be found.
- Acute changes to Pa_{CO_2} >100 mmHg may lead to cardiovascular collapse. In acute retention, for each 10-mmHg increase of Pa_{CO_2}, the pH will decrease 0.1 U.
- Chronic changes in Pa_{CO_2} may be well tolerated. To maintain a neutral milieu, the kidneys retain $[HCO_3^-]$. In the chronic setting, for every 10 mmHg of Pa_{CO_2} over 40 mmHg, $[HCO_3^-]$ increases 3.5 meq/L.

TABLE 32-2 Causes of Hypercapnia

Depressed central respiratory drive
Structural central nervous system disease
Sedating drugs
Exogenous toxins
Endogenous toxins
Thoracic cage disorders
Kyphoscoliosis
Extreme obesity
Neuromuscular diseases
Intrinsic lung disease associated with increased dead space
Chronic obstructive pulmonary disease

DIAGNOSIS AND DIFFERENTIAL

- Given clinical suspicion, the diagnosis will be confirmed on arterial blood gas analysis. See Table 32-2 for further differential diagnosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Hypercapnia is treated in the same manner as hypoxia: identify threats to life, evaluate and aggressively treat deficiencies in the ABCs. Identification of the underlying etiology will allow focused treatment. For example, a narcotic dose causing respiratory depression will respond to naloxone, while ineffective ventilation secondary to respiratory muscle weakness will respond only to assisted or mechanical ventilation.
- Supplemental oxygen should be given to maintain the level considered normal for the patient. Oxygen should not be withheld based on the worry of ''decreasing respiratory drive.'' Hypoxia will kill a patient, while only extreme hypercapnia will do the same.
- BiPAP or CPAP may be used as a bridge until a definitive diagnosis of hypercapnia and a treatment plan can be made, but it is never a long-term option. If all else fails, mechanical ventilation is indicated.
- Disposition depends on the underlying cause and frequently requires admission to a monitored bed.

WHEEZES

PATHOPHYSIOLOGY

- Wheezes are musical adventitious lung sounds produced by turbulent airflow through the central and distal airways.[7,8]

- While wheezes may occur in normal patients, they are more pronounced in obstructed airways. Airway obstruction is associated with bronchospasm, smooth muscle hypertrophy, increased secretions, and peribronchial inflammation.

CLINICAL FEATURES

- Wheezing usually occurs in asthma and other obstructive pulmonary diseases, but "not all that wheezes is asthma." A clinician must be savvy enough to recognize these other causes[9] (Table 32-3).
- In addition, not every obstructive pulmonary disease will cause wheezing. For example, the patient with severe asthma may have a quiet chest, not moving enough air to produce turbulent flow.
- One must judge the presence or absence of wheezes on the basis of the clinical situation.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is suspected in the proper clinical situation, and the patient improves with relief of obstruction. Relief may be judged by decreased work of breathing, improvement of bedside pulse oximetry, and decreased respiratory rate.
- Definitive diagnosis is confirmed by spirometric testing, but this is impractical at the bedside or during an acute exacerbation.
- A hand-held peak-flow meter is a useful clinical adjunct that can serve to gauge response to treatment. Any obtained value greater than 80 percent of predicted is considered normal. Results of this technique must be evaluated in the proper clinical situation and there must be an understanding of its limitations, including its dependence on effort and its usefulness in young children.
- Other ancillary studies include a chest x-ray and arterial blood gas analysis. However, in uncomplicated obstructive pulmonary disease, these studies may not be needed.

TABLE 32-3 Causes of Wheezing

Upper airway (more likely to be stridor, may have element of wheezing)
Angioedema: allergic, ACE inhibitor, idiopathic
Foreign body
Infection: croup, epiglottis, tracheitis
Lower airway
Asthma
Transient airway hyperreactivity (usually due to infection or irritation)
Bronchiolitis
Chronic obstructive pulmonary disease (COPD)
Foreign body
Cardiovascular
Cardiogenic pulmonary edema ("cardiac asthma")
Noncardiogenic pulmonary edema [adult respiratory distress syndrome (ARDS)]
Pulmonary embolus (rare)
Psychogenic

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial treatment is directed toward identifying threats to life and aggressively treating the underlying condition. Supplemental oxygen is given if the patient is hypoxic and, depending on the degree of obstruction, monitoring may be needed.
- Treatment of wheezing is initially directed at relieving bronchospasm by inhaled medications, including beta agonists and/or anticholinergic agents.
- Steroids are also used in the acute setting to reduce airway inflammation, but they are of no help in the acute setting. Other agents, but of unproven significance in the acute setting, include methylxanthine agents, magnesium, and parenteral beta agonists.
- Admission is required for those who have an oxygen requirement or have the potential for quick decompensation.
- If patients have failed treatment, mechanical ventilation may have to be instituted and other causes of wheezing considered.

CYANOSIS

PATHOPHYSIOLOGY

- Cyanosis is indicated by the bluish color of the skin and mucous membranes resulting from an increased amount of deoxyhemoglobin. The amount of oxyhemoglobin plays little role.
- Typically, 5 g/100 mL of deoxyhemoglobin must present for cyanosis to occur, but this is highly variable.[10]
- Various factors affect the presence or absence of cyanosis, including skin pigmentation and thickness, subcutaneous microcirculation, lighting, and ambient temperature.[11]

CLINICAL FEATURES

- The presence of cyanosis signals tissue hypoxia, but this is not always the case. The tongue is a

sensitive indicator of cyanosis, while the earlobes, conjunctiva, and nail beds are less reliable.
- Cyanosis may be central or peripheral. Central cyanosis is usually the result of unsaturated arterial blood or abnormal hemoglobin (e.g., methemoglobin). Peripheral cyanosis is caused by decreased peripheral circulation and clinical situations that lead to an increased arterial extraction of oxygen.

DIAGNOSIS AND DIFFERENTIAL

- The presence of cyanosis must be taken in context with the clinical situation. Arterial blood gas analysis will confirm the diagnosis. Other useful initial ancillary tests include a hematocrit, looking for anemia or polycythemia; a chest x-ray, an electrocardiogram, and tests for abnormal hemoglobin if clinically indicated. See Table 32-4 for differential diagnosis.
- Methemoglobinemia and carbon monoxide poisoning, although rare, must always be kept in mind in cases of cyanosis, since they will artificially alter peripheral pulse oximetry secondary to pigment formation in the blood.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Supplemental oxygen is supplied as appropriate. If the patient is unresponsive to supplemental O_2, poor perfusion, abnormal hemoglobin, or large right-to-left shunts may be present.
- Specific antidotes such as methylene blue (1 to 2 mg/kg IV) for methemoglobinemia should be used if signs of toxicity are present.

TABLE 32-4 Common Causes of Cyanosis

CENTRAL CYANOSIS	PERIPHERAL CYANOSIS
Hemoglobinopathies	Decreased cardiac output: shock
Methemoglobinemia: acquired, hereditary	Cold exposure
Sulfhemoglobinemia: acquired	Venous congestion
	Arterial thrombosis or embolus
Decreased arterial oxygen saturation	
Pulmonary etiologies: shunt, diffusion, $\dot{V}/\dot{Q}$ mismatch	
Hypoventilation	
High altitude	
Anatomic shunts	
Cardiac: VSD, ASD, TOF	
Intrapulmonary	

ABBREVIATIONS: VSD = ventricular septal defect; ASD = atrial septal defect; TOF = tetralogy of Fallot.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 58, ''Respiratory Distress,'' by J. Stephen Stapczynski.

33 PNEUMONIA AND BRONCHITIS

David M. Cline

PNEUMONIA

EPIDEMIOLOGY

- Community-acquired pneumonia (CAP) is a common medical problem that accounts for about 4

million visits to physicians and 600,000 adult hospitalizations per year.[1]
- There is an increasing frequency of atypical or opportunistic infections.[2,3] Atypical infections, infections in compromised hosts, and infections at the extremes of age may present with more subtle findings.[4] Older patients often present with a change in mental status and frequently do not manifest respiratory symptoms.

PATHOPHYSIOLOGY

- Pneumonia is an infection of the alveolar, or gas-exchange, portions of the lung.
- Bacterial pneumonia, with an intense inflammatory response, tends to cause a productive cough, whereas other atypical organisms do not lead to such an intense inflammatory response and may be associated with only a mild nonproductive cough.
- Pneumococcus is still the most common single agent, followed by viruses and atypical agents such as *Mycoplasma, Chlamydia,* and *Legionella.*

CLINICAL FEATURES

- Patients with bacterial pneumonia generally present with some combination of fever, dyspnea, cough, pleuritic chest pain, and sputum production[5] (see Table 33-1).
- Pneumococcus classically presents abruptly with fever, rigors, and rusty brown sputum.
- *Haemophilus influenzae* is more common in smokers and those at the extremes of age.
- *Staphylococcus aureus* frequently follows a viral respiratory illness, especially influenza or measles.
- Pneumonia caused by *Legionella* is spread by airborne, aerosolized water droplets rather than by person-to-person contact. This form of pneumonia presents, as do *Mycoplasma, Chlamydia,* and viral pneumonia, with fever, chills, malaise, dyspnea, and a nonproductive cough. *Legionella* also commonly causes gastrointestinal symptoms of anorexia, nausea, vomiting, and diarrhea. Mental status changes also may be present.
- The physical findings of pneumonia vary with the offending organisms and the type of pneumonia each one causes (see Table 33-1), although most are associated with some degree of tachypnea and tachycardia.
- Lobar pneumonias, such as those caused by pneumococcus and *Klebsiella,* exhibit signs of consolidation, including bronchial breath sounds, egophony, increased tactile and vocal fremitus, and dullness to percussion. A pleural friction rub and cyanosis may be present.
- Bronchopneumonias, such as those caused by *H. influenzae,* reveal rales and rhonchi on examination without signs of consolidation. A parapneumonic pleural effusion may occur in either setting; empyemas are most common with *S. aureus, Klebsiella,* and anaerobic infections.
- *Legionella,* which begins with findings of patchy bronchopneumonia and progresses to signs of frank consolidation, has other common signs, including a relative bradycardia and confusion.
- Interstitial pneumonias, such as those caused by viruses, *Mycoplasma,* and *Chlamydia,* may exhibit fine rales, rhonchi, or normal breath sounds. Bullous myringitis, when present in this setting, is pathognomonic for *Mycoplasma* infection.
- Clinical features of aspiration pneumonitis depend on the volume and pH of the aspirate, the presence of particulate matter in the aspirate, and bacterial contamination. Although acid aspiration results in the rapid onset of symptoms of tachypnea, tachycardia, and cyanosis and often progresses to frank pulmonary failure, most other cases of aspiration pneumonia progress more insidiously.[6]
- Physical signs develop over hours and include rales, rhonchi, wheezing, and copious frothy or bloody sputum. The right lower lobe is most commonly involved as a result of the anatomy of the tracheobronchial tree and gravity.[6]

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis includes acute tracheobronchitis; pulmonary embolus or infarction; exacerbation of chronic obstructive pulmonary disease (COPD); pulmonary vasculitides, including Goodpasture's disease and Wegener's granulomatosis; bronchiolitis obliterans; and endocarditis.
- The diagnosis of pneumonia is based on the presenting signs and symptoms, examination of the sputum, and chest radiography (see Table 33-1).
- Other tests include a white blood cell count with a differential count, pulse oximetric analysis, blood cultures, and pleural fluid examination. Arterial blood gas analysis may be performed in ill-appearing patients.
- If *Legionella* is being considered, serum chemistry studies and liver function tests should be performed, as hyponatremia, hypophosphatemia, and elevated liver enzyme levels are found commonly.

TABLE 33-1 Characteristics of Bacterial Pneumonia

ORGANISM	SYMPTOMS	SPUTUM	CHEST X-RAY	THERAPY
Streptococcus pneumoniae	Sudden onset, fever, rigors, pleuritic chest pain, productive cough, dyspnea	Rust-colored; gram-positive encapsulated diplococci	Lobar, occasionally patchy, occasional pleural effusion	Penicillin V 500 mg PO qid for 10 days or erythromycin 500 mg PO qid for 10 days *or* aqueous penicillin G 10–20 million units/d IV q 4–6 h *or* ceftriaxone 1 g IV qd
Group A streptococci	Abrupt onset, fever, chills, productive cough, pleuritic chest pain	Purulent, bloody; gram-positive cocci in chains and pairs	Patchy, multilobar large pleural effusion	See above
Haemophilus influenzae	Gradual onset, fever, dyspnea, pleuritic chest pain; especially in elderly and COPD	Short, tiny, gram-negative encapsulated coccobacilli	Patchy, frequently basilar, occasional pleural effusion	Ceftriaxone 1 g IV qd or cefuroxime 0.75–1.5 g IV q 8 h or amoxacillin clavulanate 875 mg PO bid for 10 days
Klebsiella pneumoniae	Sudden onset, rigors, dyspnea, chest pain, bloody sputum; especially in alcoholics or nursing home patients	Brown ''currant jelly''; thick, short, plump, gram-negative encapsulated paired coccobacilli	Upper lobes, bulging fissure sign, abscess formation	Cefazolin 0.5–1.0 g q 8 h IV *or* gentamicin 3–5 mg/kg/d divided q 8 h IV
Staphylococcus aureus	Gradual onset of productive cough, fever, dyspnea, especially just after viral illness	Purulent; gram-positive cocci in clusters	Patchy, multilobar; empyema, lung abscess	Oxacillin 8–12 g/d IV *or* vancomycin 500 mg IV q 6 h
Legionella pneumophila	Fever, chills, headache, malaise, dry cough, dyspnea, anorexia, diarrhea, nausea, vomiting	Few neutrophils and no predominant bacterial species	Multiple patchy nonsegmented infiltrates; progresses to consolidation, occasional cavitation and pleural effusion	Erythromycin 1g IV q 6 h ± rifampin 600 mg PO qd
Pseudomonas aeruginosa	Recently hospitalized, debilitated, or immunocompromised patient with fever, dyspnea, cough	Gram-negative coccobacilli	Patchy with frequent abscess formation	Tobramycin 3 mg/kg divided q 8 h IV and either piperacillin 100 mg/kg divided q 6 h IV or ceftazidime 50 mg/kg divided q 8 h IV
Chlamydia pneumoniae	Gradual onset, fever, dry cough, wheezing, occasionally sinus symptoms	Few neutrophils; organisms not visible	Patchy subsegmental infiltrates	Erythromycin 500 mg PO qid for 10 days or azithromycin 500 mg on day 1, then 250 mg qd for 4 more days or clarithromycin 500 mg PO bid for 10 days
Mycoplasma pneumoniae	Upper and lower respiratory tract symptoms, nonproductive cough, bullous myringitis, headache, malaise, fever	Few neutrophils; organisms not visible	Interstitial infiltrates (reticulonodular pattern), patchy densities, occasional consolidation	Same as for *Chlamydia pneumoniae* above
Anaerobic organisms	Gradual onset, putrid sputum, especially in alcoholics	Purulent; multiple neutrophils and mixed organisms	Consolidation of dependent portion of lung; abscess formation	Clindamycin 450–900 mg IV q 8 h or ticarcillin-clavulanate 3.1 g IV q 6 h

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Therapies directed against specific organisms are listed in Table 33-1, although empirical antibiotic coverage generally is recommended unless the clinical features and sputum Gram's stain strongly suggest a specific cause.[7,8]
- For outpatient management in otherwise healthy patients under 60 years old, erythromycin 500 mg daily for 10 to 14 days is an excellent choice for empirical therapy. Clarithromycin 500 mg twice a day for 10 days and azithromycin 500 mg on day 1 followed by 250 mg daily for 4 additional days are more expensive alternatives with fewer side effects and better compliance. Newer fluoro-

quinolones, such as levofloxacin 500 mg daily for 10 to 14 days, are also highly effective but are expensive and are restricted to patients over 18 years of age.[7,8]
- Hospital admission should be reserved for patients at the extremes of life, pregnant women, and patients with clinical signs of toxicity (i.e., tachycardia, tachypnea, hypoxemia, hypotension, and volume depletion) or serious comorbid conditions (e.g., renal failure, diabetes, and cardiac disease).[9,10]
- Patients who require admission generally also receive empirical antibiotic therapy. Recommended treatments include erythromycin 500 mg intravenously (IV) every 6 h, ceftriaxone 1 to 2 g IV daily, and levofloxacin 500 mg IV daily.
- Aspiration pneumonitides require a different therapeutic approach.[6] Witnessed aspirations should be treated with immediate tracheal suctioning, and the pH of the aspirate should be ascertained. Bronchoscopy is indicated for the removal of large particles and further clearing of the airways. Patients who require intubation also should be treated with positive end-expiratory pressure. Oxygen should be administered, but steroids and prophylactic antibiotics are of no value and should be withheld. For patients at risk of aspiration who present with signs and symptoms of infection, antibiotics are indicated. Appropriate choices include clindamycin 450 to 900 mg IV every 8 h and ticarcillin-clavulanate 3.1 g IV every 6 h.

BRONCHITIS

EPIDEMIOLOGY

- Acute bronchitis may occur in outbreaks as a respiratory virus spreads through a population or may be sporadic. It accounts for more than 7 million outpatient physician visits annually among patients older than age 18.

PATHOPHYSIOLOGY

- Acute bronchitis, an infection of the conducting airways of the lung, produces inflammation, exudate, and sometimes bronchospasm of the involved airways.
- The majority of cases of acute bronchitis are caused by viruses, including influenza A and B, adenovirus, parainfluenza virus, rhinovirus, respiratory syncytial virus (RSV), coxsackievirus A21, and, less commonly, measles virus, rubella virus, herpesviruses, and coronaviruses.[11]
- Adults who have contact with children may develop acute bronchitis and pneumonia from RSV.[12]
- Bacteria known to contribute to acute bronchitis include *Bordetella pertussis, Mycoplasma pneumoniae, Chlamydia pneumoniae,* and possibly *Streptococcus pneumoniae.*[13]

CLINICAL FEATURES

- The hallmark of acute bronchitis is cough, usually productive, in patients without evidence of pneumonia, sinusitis, or chronic pulmonary disease.[11]
- Sputum may be clear or colored, and the presence of colored sputum does not necessarily indicate a bacterial infection. Patients may complain of dyspnea or wheezing, usually caused by bronchospasm.

DIAGNOSIS AND DIFFERENTIAL

- Clinical diagnosis is appropriately made when the following findings are present: an acute cough for less than 1 week, no prior lung disease, normal arterial oxygenation, and no auscultatory abnormalities.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Nine randomized, double-blind, placebo-controlled trials were undertaken between 1966 and 1995 to determine antibiotic effectiveness in treating acute bronchitis.[14,15] Systematic review did not find statistical benefit for antibiotic treatment.
- There is some evidence that older adults and patients with underlying COPD benefit from antibiotic treatment for acute bronchitis.[15,16]
- There is evidence that bronchodilators are useful in treating acute bronchitis compared with placebo or erythromycin. Patients report decreased cough and a faster return to work when they are treated with oral or inhaled albuterol.[17,18]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 59, ''Bronchitis and Pneumonia,'' by Donald A. Moffa, Jr., and Charles L. Emerman; and Chap. 60, ''Aspiration Pneumonia, Lung Abscess, and Pleural Empyema,'' by Eric Anderson and Maxime Alix Gilles.

34 TUBERCULOSIS

David M. Cline

EPIDEMIOLOGY

- Tuberculosis (TB) causes 6 percent of all deaths worldwide.[1]
- The incidence of TB rose sharply in the United States between 1984 and 1992, driven by factors including rising rates of incarceration, human immunodeficiency virus (HIV) infection, drug-resistant TB strains, and immigration from areas with endemic TB.[2]
- Stronger TB control programs targeting high-risk groups have reversed this trend; since 1993, TB case rates have fallen steadily.

PATHOPHYSIOLOGY

- *Mycobacterium tuberculosis* is a slow-growing aerobic rod that has a unique, multilayered cell wall containing a variety of lipids that account for its acid-fast property.
- Transmission occurs through inhalation of droplet nuclei into the lungs. Persons with active tuberculosis who excrete stainable mycobacteria in saliva or sputum are the most infectious.[3]
- Survival of this organism is favored in areas of high oxygen content or blood flow, such as the apical and posterior segments of the upper lobe and the superior segment of the lower lobe of the lung, the renal cortex, the meninges, the epiphyses of long bones, and the vertebrae.[3]

CLINICAL FEATURES

- Primary TB infection is usually asymptomatic and noncontagious, presenting most frequently with

only a new positive reaction to TB skin testing. Some patients may, however, present with active pneumonitis or extrapulmonary disease. Immunocompromised patients are much more likely to develop rapidly progressive primary infections.
- The lifetime reactivation rate after primary TB infection is 5 to 10 percent. Rates are higher in the very young and the elderly as well as those with recent primary infection, major chronic diseases, or immune compromise. Most patients present subacutely with fever, cough, weight loss, fatigue, and night sweats.
- Most patients with active TB have pulmonary involvement characterized by constitutional symptoms and (usually productive) cough. Hemoptysis, pleuritic chest pain, and dyspnea may develop.
- Rales and rhonchi may be found, but the pulmonary exam is usually nondiagnostic.[3]
- Extrapulmonary TB develops in up to 15 percent of cases.[3] Lymphadenitis, with painless enlargement and possible draining sinuses, is the most common example.
- Pleural effusion may occur when a peripheral parenchymal focus or local lymph node ruptures. Pericarditis, with typical symptoms, may develop by extension of infection from local lymph nodes or pleura.
- TB peritonitis usually presents insidiously after extension from local lymph nodes.
- TB meningitis may follow hematogenous spread, presenting with fever, headache, meningeal signs, and/or cranial nerve deficits.
- Miliary TB is a multisystem disease caused by massive hematogenous dissemination. It is most common in immunocompromised hosts and children. Symptoms and findings may include fever, cough, weight loss, adenopathy, hepatosplenomegaly, and cytopenias.
- Extrapulmonary TB may also involve bone, joints, skin, kidneys, and adrenals.
- Immunocompromised patients, HIV patients in particular, are extremely susceptible to TB and far more likely to develop active infections with atypical presentations.[4] Disseminated extrapulmonary TB is also far more common in HIV patients and should be considered in the evaluation of nonpulmonary complaints as well.[3,4]
- Prior partially treated TB is the major risk factor for drug-resistant TB. It should be considered when TB is diagnosed, especially among those with suboptimal prior care, such as immigrants from endemic areas, prisoners, homeless persons, and drug users.
- Multidrug-resistant TB (MDR TB) is more common in HIV patients than the general population and has a high fatality rate in this group.[3,4]

DIAGNOSIS AND DIFFERENTIAL

- Consider the diagnosis of TB in any patient with respiratory or systemic complaints so as to facilitate early diagnosis, protect hospital staff, and make appropriate dispositions.
- Chest radiographs (CXRs) are the most useful diagnostic tool for active TB in the ED.[3] Classic findings in active primary TB are parenchymal infiltrates with or without adenopathy. Lesions may calcify.
- Reactivation TB typically presents with lesions in the upper lobes or superior segments of the lower lobes. Cavitation, calcification, scarring, atelectasis, and effusions may be seen.[3]
- Miliary TB may cause diffuse nodular infiltrates.
- Patients coinfected with HIV and TB are particularly likely to present with atypical or normal CXRs.[4]
- Acid-fast staining of sputum can detect mycobacteria in 60 percent of patients with pulmonary TB.[5] Atypical mycobacteria will yield false positives; many patients will have false negatives on a single sputum sample. Microscopy of nonsputum samples (e.g., pleural or cerebrospinal fluid) is less sensitive.
- Definitive cultures generally take weeks, but new genetic tests employing DNA probes or polymerase chain reaction (PCR) technology can confirm the diagnosis in days or hours.
- Intradermal skin testing with purified protein derivative (PPD) identifies most patients with prior or active TB infection. Results are read 48 to 72 h after placement, limiting the usefulness of this test for ED patients.
- Patients with HIV or other immunosuppressive conditions and patients with disseminated TB may be anergic.[6]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial therapy usually includes (four drugs) isoniazid (INH), rifampin, pyrazinamide, and either streptomycin or ethambutol for 2 months.[7] At least two drugs (usually INH and rifampin) are continued for 4 more months.[7]
- Patients with immune compromise or MDR TB may require more drugs for longer periods.

TABLE 34-1 Dosages and Common Side Effects of Some Drugs Used in TB

DRUG	DAILY DOSE (MAX.)	POTENTIAL SIDE EFFECTS
INH	Adult: 5 mg/kg (300 mg) Child: 10–20 mg/kg (300 mg) Route: PO	Hepatitis, neuritis, abdominal pain, acidosis, hypersensitivity drug interactions
Rifampin	Adult: 10 mg/kg (600 mg) Child: 10–20 mg/kg (600 mg) Route: PO	Hepatitis, thrombocytopenia, GI disturbance, fever, drug interactions
Pyrazinamide	Adult: 15–30 mg/kg (2 g) Child: same Route: PO	Hepatitis, rash, arthralgia, GI disturbance, hyperuricemia
Ethambutol	Adult: 15–25 mg/kg (2.5 g) Child: same Route: PO	Optic neuritis, headache, peripheral neuropathy, GI disturbance
Streptomycin	Adult: 15 mg/kg (1 g) Child: 20–30 mg/kg (1 g) Route: IM	8th cranial neuropathy, rash, renal failure, proteinuria
Ciprofloxacin	Adult: 750 mg bid Child: contraindicated Route: PO	Arthropathy, GI disturbance, CNS disturbance

- Table 34-1 summarizes usual initial daily drug doses and side effects.
- Persons with positive PPDs and no active TB disease should be evaluated for prophylactic treatment with INH to prevent reactivation TB.
- Patients with active TB who are discharged from the ED must have documented immediate referral to a physician or public health department for long-term treatment. Patients should be educated about home isolation, follow-up, and screening of household contacts.
- Admission is indicated for clinical instability, diagnostic uncertainty (such as a febrile HIV patient with pulmonary infiltrates), unreliable outpatient follow-up or compliance, and active known MDR TB. Admission to respiratory or ''droplet'' isolation is mandatory.
- ED staff should be trained to identify patients at risk for active TB as early as possible in their ED and prehospital course.[8] Patients with suspected TB should be masked or placed in respiratory isolation rooms. They should be transported wearing masks and admitted to respiratory isolation areas.
- Staff caring directly for patients with suspected TB should wear OSHA-approved respirators/masks. ED staff should receive regular PPD skin testing to detect new primary infections, rule out active disease, and consider INH prophylaxis.

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35 PNEUMOTHORAX

Rodney L. McCaskill

EPIDEMIOLOGY

- Spontaneous pneumothorax occurs primarily in male smokers with a large height-to-weight ratio.
- Primary spontaneous pneumothorax seems to result from bleb rupture.[1]

- Secondary pneumothorax occurs most often in patients with chronic obstructive pulmonary disease (COPD), but other underlying lung diseases such as asthma, cystic fibrosis, interstitial lung disease, cancer, and *Pneumocystis carinii* pneumonia have been implicated.[2]
- Iatrogenic pneumothorax occurs secondary to an invasive procedure such as placement of a subclavian line or nasogastric tube or positive-pressure ventilation and should always be ruled out by a postprocedure chest x-ray.
- Tension pneumothorax is caused by positive pressure in the pleural space, leading to decreased venous return, hypotension, and hypoxia.

PATHOPHYSIOLOGY

- Pneumothorax occurs when air enters the potential space between the parietal and visceral pleura, leading to partial lung collapse.[3]

CLINICAL FEATURES

- Symptoms resulting from a pneumothorax are directly related to its size, its rate of development, and the underlying lung disease.
- Acute onset pleuritic pain is found in 95 percent of patients.[4]
- Dyspnea occurs in 80 percent and predicts a large pneumothorax.[4]
- Decreased breath sounds on the affected side are present 85 percent of the time.[4]
- Only 5 percent have tachypnea over 24 breaths per minute.[4]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of tension pneumothorax is based on clinical features including hypoxia, hypotension, distended neck veins, displaced trachea, and unilaterally decreased breath sounds.
- The ''gold standard'' for diagnosis is an upright posteroanterior (PA) chest x-ray, but this is only 83 percent sensitive.
- Expiratory films have not been shown to be more effective in making the diagnosis.[5]
- Computed tomography (CT) may be more sensitive.
- The differential diagnosis includes costochondritis, angina, myocardial infarction (MI), pulmonary embolism (PE), pericarditis, pleurisy, and pneumonia.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Oxygen, 2 to 4 L by nasal cannula, helps increase resorption of pleural air.[6]
- In unstable patients (those with tension pneumothorax or pneumothorax with severe underlying lung disease), needle thoracostomy followed by tube thoracostomy should be performed before x-ray.
- Since pleural air is slowly resorbed, patients with small, spontaneous, asymptomatic pneumothoraces may be observed for 6 h and discharged if there is no enlargement on x-ray; however, 23 to 40 percent eventually require tube thoracostomy.[6]
- Small, asymptomatic pneumothoraces may be aspirated using a minicatheter and such patients discharged at 6 h if there is no recurrence.
- Tube thoracostomy is indicated for failed aspiration, complete lung collapse, recurrent pneumothorax, significant dyspnea, underlying lung disease, helicopter transport, general anesthesia, or mechanical ventilation.[7]

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36 HEMOPTYSIS

David F. M. Brown

EPIDEMIOLOGY

- Hemoptysis is defined as mild, less than 5 mL of blood in 24 h; moderate; or massive, more than 600 mL in 24 h or more than 100 mL for 3 days.[1]
- The most common causes are infection (including tuberculosis), neoplasm, and cardiovascular disease. No cause is found in 28 percent of cases.[2]
- Hemoptysis is found in all age groups, with a 60 : 40 male predominance.[3]

PATHOPHYSIOLOGY

- The lung has dual blood supply from the pulmonary and bronchial arteries. Bleeding may be from either source.
- The mechanism of bleeding is increased intravascular pressure, erosion by an inflammatory process into a blood vessel, or complication of a bleeding diathesis.
- Hemoptysis caused by increased intravascular pressure generally arises from a primary cardiac abnormality such as congestive heart failure or, less commonly, mitral stenosis.
- Hemoptysis caused by erosion most frequently is due to infection, malignancy, bronchiectasis, foreign-body aspiration, vasculitis, and pulmonary embolism.

CLINICAL FEATURES

- A history of underlying lung disease and tobacco use should be sought.
- The acute onset of fever, cough, and bloody sputum suggests pneumonia or bronchitis. A more indolent productive cough may indicate bronchitis or bronchiectasis. Dyspnea and pleuritic chest pain are hallmarks of pulmonary embolism. Fever, night sweats, and weight loss may reflect tuberculosis (TB) or malignancy. Chronic dyspnea and minor hemoptysis may indicate mitral stenosis or alveolar hemorrhage syndromes.
- The physical examination is aimed at assessing the severity of hemoptysis and the underlying disease process but is unreliable in localizing the site of bleeding.
- Common signs include fever and tachypnea. Hypotension is rare except in massive hemoptysis. Cardiac examination may reveal the diastolic rumble of mitral stenosis or a pronounced P2 suggestive of pulmonary embolus. Lung auscultation may reveal rales, wheezes, or focal consolidation. More frequently, the heart and lung examinations are normal.
- Careful inspection of the oral and nasal cavities is warranted to exclude an extrapulmonary source of bleeding.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis of hemoptysis includes infection (bronchitis, bronchiectasis, bacterial pneumonia, TB, fungal pneumonia, and lung abscess), neoplasms (bronchogenic carcinoma, metastatic carcinoma, and bronchial adenoma), cardiogenic causes (left ventricular failure, mitral stenosis), trauma, foreign body aspiration, pulmonary embolism, primary pulmonary hypertension, vasculitis, and bleeding diathesis.
- Basic testing should include pulse oximetry and chest radiography, although 20 to 30 percent of patients who present with hemoptysis have a normal chest x-ray.[2,4]
- A hematocrit and a blood bank sample should be obtained in patients with major hemoptysis. Other testing should be ordered as indicated by the clinical situation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial management focuses on the ABCs. Cardiac and pulse oximetry monitoring along with noninvasive blood pressure machines should be utilized. Large-bore intravenous (IV) lines should be placed.
- Supplemental oxygen should be administered to keep the oxygen saturation above 95%.
- IV crystalloid should be administered initially for hypotension. Packed red blood cells should be transfused as needed.
- Fresh-frozen plasma should be given to patients with coagulopathies; platelets should be administered to those with thrombocytopenia.
- Patients with ongoing massive hemoptysis should be placed in the decubitus position with the bleeding side down to minimize spilling of blood into the contralateral lung.
- Cough suppression with codeine (15 to 30 mg) or other opioids is indicated.

- Endotracheal intubation should be performed with a large tube (8.0 mm) for persistent hemoptysis and worsening respiratory status. This will optimize suctioning and permit bronchoscopy.
- Indications for admission include massive hemoptysis or minor hemoptysis whose underlying cause carries a high risk of proximate massive bleeding. Some underlying conditions may warrant admission regardless of the degree of bleeding.
- All admissions should include consultation with a pulmonologist or thoracic surgeon for help in making decisions regarding bronchoscopy, computed tomography scanning, or angiography for bronchial artery embolization.[5]
- Patients who are discharged should be treated for several days with cough suppressants, inhaled beta-agonist bronchodilators, and, if an infectious etiology is suspected, appropriate antibiotics. Close follow-up is warranted.

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37 ASTHMA AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

David L. Leader, Jr.

EPIDEMIOLOGY

- Asthma affects approximately 4 to 5 percent of the population in the United States.[1] In childhood, asthma is the most common chronic disease, with a prevalence of 5 to 10 percent.[2] Approximately 7 to 10 percent of the elderly are affected by asthma.[3]
- Chronic obstructive pulmonary disease (COPD) is rarely manifest in individuals below age 40, but it is very common in older individuals. Among those aged 55 to 85 years, the prevalence of COPD is approximately 10 percent.
- In the United States, COPD is the third most common cause of hospitalization, the fourth most common cause of death, and the only leading cause of death with an increasing incidence.[4]
- Among patients hospitalized for a COPD exacerbation, mortality is approximately 5 to 14 percent.[3,5]

PATHOPHYSIOLOGY

- In asthma, the pathophysiologic hallmark is a reduction in airway diameter caused by smooth muscle contraction, vascular congestion, bronchial wall edema, and thick secretions.
- During the acute-response phase of asthma, inflammatory mediators are released, causing an intense inflammatory reaction with resultant bronchoconstriction, vascular congestion, edema formation, increased production of mucus, and impaired mucociliary transport.[6]
- Although many stimuli have been noted to precipitate an increase in airway responsiveness, viral respiratory infections are the most common of the stimuli that cause acute exacerbation of asthma.[7]
- The physiologic consequences of airflow obstruction in asthma and COPD are demonstrated in increased airway resistance, decreased maximum expiratory flow rates, air trapping, increased airway pressures (resultant barotrauma and adverse hemodynamic effects), ventilation/perfusion imbalance (causing hypoxemia/hypercarbia), and increased work of breathing (causing respiratory muscle fatigue with ventilatory failure).
- An estimated 80 to 90 percent of the risk of developing COPD can be attributed to cigarette smoking. Factors predictive of COPD mortality include age of starting, total pack-years, and current smoking status.[8]
- $Alpha_1$-antitrypsin deficiency is the only proven genetic risk factor for COPD.
- The primary element in the pathophysiology of chronic airflow obstruction in COPD is impedance to airflow, especially expiratory airflow, due to increased resistance or decreased caliber throughout the small bronchi and bronchioles.

CLINICAL FEATURES

- Dyspnea, chest tightness, wheezing, and cough are frequent complaints of patients presenting with exacerbations of asthma or COPD.
- Physical exam findings include wheezing and a prolonged expiratory phase. Wheezing does not correlate with the degree of airflow obstruction, as a ''quiet chest'' indicates severe airflow obstruction.
- There are two dominant clinical forms of COPD: (1) pulmonary emphysema, due to abnormal permanent enlargement and destruction of the air spaces distal to terminal bronchioles, and (2) chronic bronchitis, a condition of excess mucus secretion in the bronchial tree, with a chronic productive cough occurring on most days for at least 3 months in the year for 2 consecutive years. Elements of both clinical forms are often present, although one predominates.
- Clinical features of COPD patients with severe attacks include sitting-up and forward posturing, pursed-lip exhalation, accessory muscle use, paradoxical respirations, and diaphoresis.
- A pulsus paradoxus above 20 mmHg is indicative of severe asthma/COPD.
- Tachypnea, cyanosis, agitation, apprehension, and hypertension demonstrate hypoxia.
- Signs of hypercapnia include confusion, tremor, plethora, stupor, hypopnea, and apnea.
- Indicators in patients who are at higher risk of respiratory failure with hypoxia and hypercarbia include attacks lasting more than several days, dependence on steroids, and prior attacks requiring intubation.

DIAGNOSIS AND DIFFERENTIAL

- Emergency department (ED) diagnosis of asthma or COPD is usually made clinically, with determination of the severity and any existing complications.
- The forced expiratory volume in 1 s (FEV_1) and the peak expiratory flow rate (PEFR) directly measure the degree of large airway obstruction.[9]
- Objective measurements of airflow obstruction, such as sequential peak expiratory flow rates, have been shown to be more accurate than clinical judgment in determining the severity of the attack and the response to therapy.
- Initial spirometry (PEFR—personal best of patient or percent predicted) and response to initial treatment can be used to predict the need for hospitalization with 86 percent sensitivity and 96 percent specificity.[10]
- Pulse oximetry is a useful and noninvasive means of assessing and monitoring oxygen saturation during treatment, but it does not aid in predicting clinical outcomes.[11]
- Testing of arterial blood gases (ABGs) should be reserved for sicker patients with severe exacerbations and those who are not responding to therapy. This can help to assess for hypoventilation with carbon dioxide retention and respiratory acidosis.
- A normal or slightly elevated P_{CO_2} in the setting of an acute asthmatic attack is an ominous finding if the patient is doing poorly. This is an indication of extreme airway obstruction and fatigue with possible onset of acute respiratory failure.
- A chest x-ray (CXR) should be obtained if there is clinical suspicion of a complication such as pneumothorax, pneumomediastinum, pneumonia, or other medical concern such as congestive heart failure (CHF), pleural effusion, or pulmonary neoplasia.
- X-ray findings in emphysematous disease may show signs of hyperaeration, such as increased anteroposterior (AP) diameter, flattened diaphragms, increased parenchymal lucency, and attenuation of arterial vascular shadows.
- Laboratory examinations are of limited value and should be used selectively.
- Findings on electrocardiography (ECG) in moderate to severe pulmonary disease may reveal right ventricular strain, abnormal P waves, or nonspecific ST-T-wave abnormalities; these may resolve with treatment. Arrhythmias can develop, including multifocal atrial tachycardia (MAT).
- The differential diagnosis of decompensated asthma and COPD includes CHF (''cardiac asthma''), upper airway obstruction or aspiration of a foreign body or gastric acid, pulmonary neoplasia, pleural effusions, interstitial lung diseases, pulmonary embolism, and exposure to asphyxiants.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with COPD often have more underlying illness than do asthmatics, but the therapy for acute bronchospasm and inflammation in each is similar.
- All patients should be placed on a cardiac monitor, pulse oximeter, and noninvasive blood pressure monitor; patients with moderate to severe attacks should have IV access.
- Administer oxygen. In COPD, the need for oxygen must be balanced against progressive hypercarbia and suppression of hypoxic ventilatory

drive.[12] Arterial saturation should be corrected to above 90 percent.

- Beta-adrenergic agonists are the drugs of choice to treat bronchospasm and should be used as first-line therapy in aerosolized or parenteral forms in critical settings. Albuterol and metaproterenol are the most $beta_2$-specific agents.[13] Subcutaneous terbutaline sulfate (0.25 to 0.5 mL) or epinephrine (1 : 1000) (0.1 to 0.3 mL) may also be administered.
- Steroids should be given immediately to patients with severe attacks as well as to those who are currently taking or have recently taken these drugs. Prednisone 60 to 180 mg/d is the optimal daily dose; IV methylprednisolone 60 to 125 mg may be used if the patient is unable to take oral medications. A 3- to 10-day daily course of steroids (prednisone 40 to 60 mg/d) should be given to discharged patients.
- Anticholinergics are useful adjuvants when given with other therapies; when they are used with beta agonists, the effects may be additive.[14] Ipratropium is the agent of choice (500 mg = 2.5 mL) and is available as a nebulized solution or metered-dose inhaler (MDI). A combined ipratropium and albuterol inhaler is available. The effects of ipratropium peak in 1 to 2 h and last 3 to 4 h. Dosages may be repeated every 1 to 4 h.
- The role of methylxanthines in the treatment of acute asthma and COPD has been seriously challenged.[13] Theophylline is no longer considered a first-line agent for acute asthma or COPD.
- Broad-spectrum antibiotics: All current guidelines recommend antibiotics for the treatment of bacterial respiratory infections, especially if there is evidence of infection (fever, CXR findings, and abnormal mucus production). Trimethoprim-sulfamethoxazole double strength, doxycycline, macrolides, cephalosporins, and newer fluoroquinolones may be used.
- Heliox: several studies have demonstrated that an 80 : 20 mixture of helium and oxygen (Heliox) can lower airway resistance and act as an adjunct in the treatment of very severe asthma exacerbation.[15]
- In selective cooperative patients, noninvasive positive-pressure ventilation (intermittent, continuous, or biphasic) may avert artificial ventilation when the patient begins to exhibit signs of acute ventilatory failure.[16]
- Assisted mechanical ventilation is indicated for inability to maintain oxygen saturation above 90% or severe hypercarbia associated with stupor, altered mental status, exhaustion, narcosis, or acidosis. Oral intubation is preferred, since larger endotracheal tubes that facilitate suctioning and ventilator weaning can be used.
- Criteria for admission in asthma patients are listed in Table 37-1 and for COPD patients in Table 37-2.
- In patients being discharged, continued treatment with $beta_2$ agonists and oral steroids is important. In addition, patient education and close medical follow-up is essential.

TABLE 37-1 Criteria for Hospital Admission in Acute Asthma

Emergency visit within the preceding 3 days
Failure of subjective improvement following treatment
Failure of posttreatment FEV_1 to increase by >500 mL, or absolute value <1.6 L
Failure of posttreatment PEFR to increase more than 15% above initial value, or absolute value <200 L/min, or PEFR <50% predicted
Change in mental status (lethargy, agitation, exhaustion, and confusion)
Failure of hypercarbia to resolve after treatment
Presence of pneumothorax

NOTE: Presence of any of these conditions warrants admission to the hospital.
ABBREVIATIONS: FEV_1 = forced expiratory volume; PEFR = peak expiratory flow rate.

TABLE 37-2 Criteria for Hospital Admissions in Acute Exacerbation of Chronic Obstructive Pulmonary Disease

One or more of the following: inability to walk between rooms, to sleep or eat due to dyspnea, or to manage at home without additional resources, which are not available
Prolonged or progressive symptoms prior to ED visit
Altered mental status
Worsening hypoxia, hypercarbia, or acidosis (pH < 7.30)
High-risk comorbid conditions or complications
Unresponsive new or worsening cor pulmonale
Planned invasive procedure that may worsen pulmonary function
Respiratory muscle fatigue

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Section 8
GASTROINTESTINAL EMERGENCIES

38 ACUTE ABDOMINAL PAIN

David M. Cline

EPIDEMIOLOGY

- Data from the U.S. National Center for Health Statistics indicate that abdominal pain was the single ''most frequently mentioned'' reason offered by patients for visiting the emergency department (ED) in 1996 (annual incidence is approximately 57 of 1000 adult ED visits.[1]
- Admission rates for abdominal pain vary markedly, ranging from 18 to 42 percent, with rates as high as 63 percent reported in patients over 65 years of age.[2]

PATHOPHYSIOLOGY

- Visceral abdominal pain is usually caused by stretching of fibers innervating the walls or capsules of hollow or solid organs, respectively. Less commonly, it is caused by early ischemia or inflammation.
- Foregut organs (stomach, duodenum, and biliary tract) produce pain in the epigastric region; midgut organs (most of the small bowel, appendix, and cecum) cause periumbilical pain; and hindgut organs (most of the colon, including the sigmoid) as well as the intraperitoneal portions of the genitourinary system tend to cause pain initially in the suprapubic or hypogastric area.
- Visceral pain is usually felt at the midline.
- Parietal or somatic abdominal pain is caused by irritation of fibers that innervate the parietal peritoneum, usually the portion covering the anterior abdominal wall.
- In contrast to visceral pain, parietal pain can be localized to the dermatome directly above the site of the painful stimulus. As the underlying disease process evolves, the symptoms of visceral pain give way to the signs of parietal pain, with tenderness and guarding. As localized peritonitis develops further, rigidity and ''rebound'' appear.
- Referred pain is felt at a location distant from the diseased organ.

CLINICAL FEATURES

- The principal characteristics of abdominal pain include location, quality, severity, onset, duration, aggravating and alleviating factors, and change in any of these variables over time.
- Associated symptoms should be sought: gastrointestinal, genitourinary, and gynecologic symptoms.
- Contrary to conventional teaching, absent or diminished bowel sounds provide little clinically useful information. This is supported by the observation that, in a series of 100 patients with operative confirmation of peritonitis due to perforation of peptic ulcer, about half were noted to have normal or increased bowel sounds.[3]
- Hyperactive/obstructive bowel sounds, although of limited value, are somewhat more helpful, as reflected by their presence in about half of 100 patients with small bowel obstruction (SBO), in contrast to only 5 to 10 percent of patients with 500 other surgical diagnoses. However, fully 25 percent of those with SBO had absent or diminished bowel sounds.[3]
- ''Rebound'' tenderness, often regarded as the

clinical criterion standard of peritonitis, has several important limitations. In patients with peritonitis, the combination of rigidity, referred tenderness, and especially ''cough pain''[4] usually provides sufficient diagnostic confirmation that little is gained by eliciting the unnecessary pain of rebound.[5]

- False-positive rebound tenderness occurs in about one patient in four without peritonitis,[5] perhaps because of a nonspecific startle response. Indeed, more recent work has led some authors to conclude that rebound tenderness, in contrast to cough pain, is of ''no predictive value.''[6]
- There is little evidence that rectal tenderness in patients with right-lower-quadrant (RLQ) pain provides any useful incremental information beyond what has already been obtained by other, less uncomfortable components of the physical examination.[7]

DIAGNOSIS AND DIFFERENTIAL

- Based upon three studies comprising a total of over 1800 patients, a white blood cell (WBC) count exceeding the threshold value of 10,000 to 11,000/mm^3 only doubled the odds of appendicitis, while a WBC below this cut the odds in half.[8–10]
- For acute cholecystitis, the likelihood ratios (LRs) of the WBC count are virtually identical to those seen in appendicitis and are of equally limited clinical value.[8–10]
- In one large, well-conducted series of nonspecific abdominal pain (NSAP), 18 percent (95 percent CI, 22 to 34 percent) of patients were reported to have WBC counts >10,500/mm^3.[11]
- Recent work has concluded that plain films continue to be markedly overutilized. One study concluded that restriction of the plain abdominal radiography (PAR) to patients with suspected obstruction, perforation, ischemia, peritonitis, or renal colic would have had no impact on management and the use of PARs would have been reduced by 80 percent.[12]
- It is clear that diagnostic error in adults with abdominal pain increases in proportion to age, ranging from a low of 20 percent if only young adults are considered to a high of 70 percent in the very elderly.[9,13]
- The most common causes of abdominal pain are listed in Table 38-1.
- Causes of abdominal pain stratified by age are listed in Table 38-2.

TABLE 38-1 Most Common Causes of Acute Abdominal Pain[8–10]

FINAL DIAGNOSIS	PROPORTION OF >10,000 PATIENTS	
Nonspecific abdominal pain	34%	
Appendicitis	28%	
Biliary tract disease	10%	
Bowel obstruction	4%	
Acute gynecologic disease	4%	
	Salpingitis	68%
	Ovarian cyst	21%
	Ectopic	6%
	Incomplete abortion	5%
	Subtotal, gynecologic	100%
Pancreatitis	3%	
Renal colic	3%	
Perforated peptic ulcer	3%	
Cancer	2%	
Diverticular disease	2%	
Other (≤1% each)	6%	

SPECIFIC DIAGNOSES

- Tests for specific diagnoses are discussed in the chapters that follow in this section. The exceptions are mesenteric ischemia and abdominal wall pain.
- The small bowel, which is supplied by the superior mesenteric artery, has a warm ischemia time of only 2 to 3 h.

TABLE 38-2 Causes of Acute Abdominal Pain Stratified by Age[8–10]

FINAL DIAGNOSIS	≥50 YEARS OLD (*N* = 2406)	<50 YEARS OLD (*N* = 6317)
Biliary tract disease	21%	6%
Nonspecific abdominal pain	16%	40%
Appendicitis	15%	32%
Bowel obstruction	12%	2%
Pancreatitis	7%	2%
Diverticular disease	6%	<0.1%
Cancer	4%	<0.1%
Hernia	3%	<0.1%
Vascular	2%	<0.1%
Acute gynecologic disease	<0.1%	4%
Other	13%	13%

- The clinical picture of mesenteric ischemia is characterized initially by poorly localized visceral abdominal pain without tenderness.
- Patients may become transiently better after a few hours of ischemia, at the time of onset of mucosal infarction, only to later develop peritoneal findings as full-thickness necrosis of the bowel wall becomes apparent.
- Timely diagnosis requires that an angiogram be obtained very early in the evolution of the pathologic process—so early, in fact, that it may seem clinically premature to order such an invasive test on an elderly patient who may not appear ill.[14]
- Computed tomography (CT) with contrast is 92 percent specific for mesenteric ischemia but only 71 percent sensitive.[14,15]
- A useful and underutilized test to diagnose abdominal wall pain is the situp test, also known as Carnett's test. Following identification of the site of maximum abdominal tenderness, the patient is asked to fold his or her arms across the chest and sit up halfway. The examiner maintains a finger on the tender area, and if palpation in the semisitting position produces the same or increased tenderness (Carnett's sign), the test is said to be positive for an abdominal wall syndrome.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The management of abdominal emergencies is discussed in the diagnosis-specific chapters that follow.
- The management of mesenteric ischemia is timely identification and aggressive surgical intervention. Survival is 30 percent or less.[16]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 68, "Acute Abdominal Pain," by E. John Gallagher, and Chap. 69, "Abdominal Pain in the Elderly," by Robert McNamara.

39 GASTROINTESTINAL BLEEDING

Mitchell C. Sokolosky

EPIDEMIOLOGY

- Acute upper GI bleeding has an annual incidence of 100 per 100,000.[1,2]

- Peptic ulcer disease accounts for 60 percent of all cases of upper GI bleeding, followed by erosive gastritis and esophagitis, esophageal and gastric varices, and Mallory-Weiss syndrome. The most common cause of apparent lower GI bleeding is upper GI bleeding.
- Lower GI bleeding has an annual incidence of 20 per 100,000.[3]
- Hemorrhoids are the most common cause of actual lower GI bleeding, followed by diverticular disease, arteriovenous malformations, inflammatory disease, and polyps.[4]
- Both upper and lower GI bleeding are more common in males and the elderly.
- Factors associated with a high morbidity rate are hemodynamic instability, repeated hematemesis or hematochezia, failure to clear with gastric lavage, age over 60, and coexistent organ system disease.

PATHOPHYSIOLOGY

- Upper GI bleeding is defined as that originating proximal to the ligament of Treitz.
- Irritative factors—such as alcohol, salicylates, and nonsteroidal anti-inflammatory agents—are predisposing factors for peptic ulcer disease, gastritis, and esophagitis.

CLINICAL FEATURES

- Most patients will volunteer complaints of hematemesis, hematochezia, or melena.
- Some will have more subtle presentations of hypotension, tachycardia, angina, syncope, weakness, and confusion.
- Hematemesis or coffee-ground emesis suggests a source proximal to the right colon.
- Hematochezia indicates a more distal colorectal lesion.
- Weight loss and changes in bowel habits are classic symptoms of malignancy.
- Vomiting and retching followed by hematemesis is suggestive of a Mallory-Weiss tear.
- A history of aortic graft should suggest the possibility of an aortoenteric fistula.
- A history of medication or alcohol use should be sought. This history may suggest peptic ulcer disease, gastritis, or esophageal varices.
- Hypotension and tachycardia suggest severe bleeding. Cool, clammy skin is an obvious sign of shock.
- Spider angiomata, palmar erythema, jaundice, and gynecomastia suggest underlying liver disease.
- A careful ear-nose-throat (ENT) exam can exclude swallowed blood as a source.
- A rectal exam is mandatory to detect the presence of blood, its appearance (bright red, maroon, or melanotic), and the presence of masses.
- Ingestion of iron or bismuth can simulate melena, and certain foods, such as beets, can simulate hematochezia; however, stool guaiac testing will be negative.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis may be obvious with the finding of hematemesis, hematochezia, or melena. Nasogastric tube placement and aspiration may detect occult upper GI bleeding.
- In patients with significant GI bleeding, the most important laboratory test is the type and crossmatch blood.
- Other important tests include a complete blood count, blood urea nitrogen (BUN), creatinine, electrolytes, glucose, coagulation studies, and liver function tests. The initial hematocrit level often will not reflect the actual amount of blood loss. Upper GI bleeding may elevate the BUN.
- Routine abdominal radiographs, including barium contrast studies, are of limited value in the emergency setting.
- Controversy in the literature remains as to whether scintigraphy, angiography, or colonoscopy, and in which order, should be the initial diagnostic procedure of choice in the evaluation of lower GI bleeding.[5–8]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Replacement of volume loss with crystalloids.
- The decision to start blood should be based on clinical factors (no improvement in perfusion after 2 L of crystalloids) rather than initial hematocrit.
- A nasogastric (NG) tube should be placed in all patients with significant bleeding. Concerns that NG tube passage may provoke bleeding in patients with varices are unwarranted. Room-temperature water is the preferred irrigant for gastric lavage.[9]
- Early therapeutic endoscopy, where available, should be considered the treatment of choice for significant upper GI bleeding. Esophageal varices can be endoscopically treated by either band liga-

tion or injection sclerotherapy. Endoscopic hemostasis (with injection sclerotherapy, electrocoagulation, heater probes, and lasers) has been used successfully in a variety of nonvariceal etiologies of upper GI bleeding.
- Infusions of somatostatin and octreotide have been shown to be effective in reducing bleeding from both varices and peptic ulcer disease. They should be considered useful adjuncts, either before endoscopy or when endoscopy is unsuccessful, contraindicated, or unavailable.
- Balloon tamponade with the Sengstaken-Blakemore tube or its variants can control documented variceal hemorrhage in 40 to 80 percent of patients.
- With patients who do not respond to medical therapy, and in whom endoscopic hemostasis—if available—fails, emergency surgical intervention is indicated.

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40 ESOPHAGEAL EMERGENCIES

Mitchell C. Sokolosky

EPIDEMIOLOGY

- Esophageal cancer has a prevalence of about 10,000 cases per year. Some 95 percent of esophageal neoplasms are squamous cell, with the remainder being adenocarcinoma. Men are affected more than women.
- Risk factors for squamous cell carcinoma include alcohol, smoking, achalasia, and previous caustic ingestion with lye.
- Barrett's esophagus predisposes to adenocarcinoma.[1] Barrett's esophagus is present in up to 10 percent of patients with gastroesophageal reflux disease (GERD).[2]
- The incidence of esophageal disease as the cause of chest pain in patients with normal coronary arteries ranges from 20 to 60 percent.[3]
- GERD affects up to 25 percent of adults, possibly more among the elderly.[4]
- Some 75 percent of all esophageal perforations are due to iatrogenic injuries.
- Boerhaave syndrome accounts for 10 to 15 percent of esophageal perforations, with trauma causing 10 percent of these.
- Among patients with chronic liver disease, 60 percent will develop varices.
- Of patients who develop varices, 25 to 30 percent experience hemorrhage.[5]
- Mortality in esophageal variceal bleeding is 40 percent.[5]
- Mallory-Weiss tears cause 5 to 15 percent of upper GI hemorrhage.

PATHOPHYSIOLOGY

- Dysphagia is defined as difficulty in swallowing. Two broad pathophysiologic groups represent an approach to patients with dysphagia: (1) transfer dysphagia (difficulty in initiating swallowing) and transport dysphagia (feeling of food getting

"stuck") and (2) obstructive disease (progressive symptoms, solids then liquids) and motor dysfunction (intermittent and variable symptoms).
- Transient relaxation of the lower esophageal/sphincter (LES) complex is a primary mechanism causing reflux.
- Prolonged gastric emptying, agents that decrease LES function, pressure, and impaired esophageal motility predispose to reflux.
- Esophagitis is a result of either an inflammatory (GERD, medications) or infectious (*Candida* most common) process.
- Esophageal perforation may be due to instrumentation, Boerhaave's syndrome, trauma, or foreign-body ingestion.
- Varices develop in patients with chronic liver disease in response to portal hypertension.
- Mallory-Weiss syndrome is from arterial bleeding from longitudinal mucosal lacerations of the distal esophagus/proximal stomach, thought to be due to a transient, large pressure gradient between the thorax and stomach and experienced maximally at the GE junction.

CLINICAL FEATURES

- Neoplasms are a common cause of transfer and transport dysphagia.
- Odynophagia is defined as painful swallowing (suggesting an inflammatory process).
- Esophageal stricture occurs as a result of scarring from GERD or other chronic inflammation. Symptoms may build over years and are often noted solely with solids.
- Schatzki's ring is the most common cause of intermittent dysphagia with solids.
- Esophageal webs are a component of Plummer-Vinson syndrome (along with iron deficiency anemia).
- Diverticula may be found throughout the esophagus and result in transfer dysfunction.
- Neuromuscular disorders typically result in misdirection of the bolus, with repeated swallowing attempts. Symptoms are often intermittent. Cerebrovascular accident (CVA) is the most common cause.
- Achalasia is a dysmotility disorder of unknown cause and the most common motility disorder producing dysphagia.
- Differentiating esophageal symptoms from acute ischemic coronary symptoms is often not possible in the emergency department (ED). Both types of pain may be accompanied by diaphoresis, pallor, and nausea and vomiting. Radiation of esophageal pain may be felt in either arm, the neck, the shoulders, or the back.
- Heartburn is the classic symptom of GERD, and chest discomfort may be the sole manifestation of the disease. The association of pain with meals, postural changes in pain, and relief of symptoms with antacids is more consistent with GERD.
- Esophagitis can cause prolonged periods of chest pain and odynophagia.
- Chest pain due to esophageal dysmotility often occurs at rest and is dull or colicky in nature.
- Pain due to esophageal perforation is classically described as acute, severe, unrelenting, and diffuse; it is reported in the chest, neck, and abdomen with radiation to the back and shoulders. Pain is often exacerbated by swallowing. Physical exam varies with the severity of the rupture and the elapsed time between the rupture and presentation. Abdominal rigidity with hypotension and fever often occur early.
- Mediastinal emphysema due to esophageal perforation takes time to develop. It is less commonly detected by examination or radiography in lower esophageal perforations, and its absence does not rule out perforation.[6] Hammon's crunch, caused by air in the mediastinum being moved by the beating heart, can sometimes be auscultated.
- Patients with bleeding varices and Mallory-Weiss syndrome usually present with an acute onset of upper GI bleeding. Less than half of patients with Mallory-Weiss tears will report a history of vomiting prior to hematemesis.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of the underlying pathology of dysphagia is most often made outside of the ED.
- Initial evaluation may include anteroposterior (AP) and lateral neck and chest x-rays.
- Direct laryngoscopy can identify structural lesions.
- Oropharyngeal dysphagia is best evaluated with videoesophagography.
- Traditional barium swallow can be used for evaluating transport dysphagia.
- Definitive diagnosis of esophageal cancer is made by endoscopy with biopsy.
- At a minimum, an electrocardiogram (ECG) and chest x-ray should be obtained in all patients with ambiguous presentations of chest pain.
- Outpatient workup options for chest pain of

esophageal origin may include an acid infusion test, esophagoscopy, and/or manometry.
- The diagnosis of advanced esophagitis is by endoscopy.
- The diagnosis of esophageal perforation is usually made by chest x-ray and contrast esophagography.
- Endoscopy, computed tomography (CT) of the chest, and thoracentesis can be useful adjuncts if esophagography (10 to 25 percent false-negative rate) is unrevealing.
- Endoscopy is both diagnostic and therapeutic for esophageal varices.

EMERGENCY DEPARTMENT CARE AND DISPOSTION

- Care of dysphagia and esophageal neoplasms in the ED is supportive.
- Definitive treatment occurs after discharge from the ED in consultation with a gastroenterologist.
- Obstructive causes of dysphagia—such as esophageal strictures, Schatzki's ring, and esophageal webs—are treated with dilatation.
- An antacid mixed with viscous lidocaine is often given initially in the ED to treat reflux.
- H_2 blockers or proton pump inhibitors are mainstays of therapy for reflux.
- Any patient with cardiac risk factors and pain of unclear origin should be strongly considered for cardiologic consultation and admission.
- Care of esophageal perforation in the ED includes resuscitation of shock, broad-spectrum parental antibiotics, and emergent surgical consultation.
- Bleeding esophageal varices are treated with airway control, resuscitation of shock, and emergent gastroenterology consultation for therapeutic endoscopy (sclerotherapy or ligation).
- Pharmacologic treatment with an intravenous vasopressin/nitroglycerin combination, somatostatin, or octreotide may be used.
- Balloon tamponade or surgical therapy is generally considered as a last resort.
- About 60 percent of variceal bleeding will resolve with supportive care alone.
- Initial treatment of Mallory-Weiss tears is supportive, as the vast majority of tears stop bleeding spontaneously.
- Ongoing hemorrhage can require treatment with electrocoagulation, sclerotherapy, and laser photocoagulation.
- Angiographic embolization or surgical intervention remains on option as well.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 71, ''Esophageal Emergencies,'' by Moss H. Mendelson.

41 SWALLOWED FOREIGN BODIES

Patricia Baines

EPIDEMIOLOGY

- Mortality for swallowed foreign bodies is approximately 1500 people per year.
- The pediatric population accounts for approximately 80 percent of all cases.
- Children most often ingest coins, toys, crayons, and ballpoint pen caps; adults are most likely to obstruct their esophagus with either meat or bones.[1]

PATHOPHYSIOLOGY

- Most objects pass spontaneously, 10 to 20 percent require some intervention, and only 1 percent require surgical treatment.[2]
- There are five common areas where objects lodge:

cricopharyngeal narrowing (C6 level), the most common site; thoracic inlet (T1 level); aortic arch (T4 level); tracheal bifurcation (T6 level); and hiatal narrowing (T10–11 level).
- Once an object passes the pylorus, it usually continues and is passed in the stool.
- Objects lodged in the esophagus can result in airway obstruction, stricture, or perforation with resulting mediastinitis, cardiac tamponade, paraesophageal abscess, or aortotracheoesophageal fistula.
- Perforation may be the due to direct mechanical erosion or chemical corrosion.

CLINICAL FEATURES

- Common symptoms in adults are retching or vomiting, dysphagia, choking, coughing, or aspiration.
- Common symptoms in children include refusal to eat, vomiting, gagging, choking, stridor, neck or throat pain, inability to swallow, increased salivation, and foreign body sensation in the chest.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis can frequently be made clinically by examining the nasopharynx and oropharynx. Examine subcutaneous tissue for air secondary to perforation.
- Laryngoscopy is indicated (direct or indirect) when patients complain of foreign body sensation.
- In children, a red throat, dysphagia, palatal abrasion, temperature elevation, anxiety, distress, and peritoneal signs are all findings suggestive of foreign body ingestion.
- Radiographs of the neck, chest, or abdomen should be performed.
- Consultation and endoscopy are recommended before initiation of any contrast study.
- In cases of suspected perforation, a water-soluble contrast agent (Gastrografin) should be used.
- Barium should be used if aspiration is possible.
- Perform serial exams with repeated x-rays to monitor the progress of the object.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

Food Impaction
- Use conservative management if patients can tolerate their own secretions. If the patient is unable to swallow fluids or if food does not pass within 12 h, intervention is necessary.
- Administer glucagon 1 mg intravenous (IV) to relax esophageal smooth muscle.
- Nifedipine 10 mg sublingual is used to reduce lower esophageal sphincter pressure.[3] Sublingual (SL) nitroglycerin (0.4 mg SL) can be used but may cause hypotension.
- Proteolytic enzymes such as papain (Adolph's meat tenderizer) are contraindicated due to risk of esophageal perforation.

Coin Ingestion
- Approximately 35 percent of children with coin ingestion will be asymptomatic.
- All children with suspected coin ingestion should have radiographs performed.
- Coins in the esophagus lie in the frontal plane with the flat side visible on the anteroposterior view. Coins in the trachea lie in the sagittal plane and are visible on end, on the anteroposterior view.
- Lodged coins require endoscopic removal.
- Foley catheter removal of ingested coins may be used if ingestion is less than 24 h. Aspiration may be a complication. Airway equipment for airway control must be immediately available.

Button Battery Ingestion
- A button battery ingestion is a true emergency because of the rapid action of the alkaline substance. Lithium cells are associated with a more adverse outcome.
- Esophageal burns can occur within 4 h, and perforation can occur within 6 h.
- Mercuric oxide cells contain heavy metals. Blood and urine mercury levels should be measured if the mercury cell opens within the gastrointestinal tract.
- Emergent endoscopic removal of a button battery should occur after radiographic documentation. Ipecac is contraindicated.[4]
- Foley catheter technique may be used if the battery has been lodged for less than 2 h.
- Button batteries that have passed the esophagus in an asymptomatic patient need not be retrieved. If the cell has not passed the pylorus within 48 h, then retrieval is necessary.
- Most batteries pass through the gastrointestinal tract within 48 to 72 h.
- Early surgical consult is mandatory for symptomatic patients with acute abdomen, tarry or bloody stools, fever, or persistent vomiting.
- Assistance with cell identification may be obtained by calling the National Button Battery Ingestion

Hotline (National Capital Poison Center, Washington, DC) at 202-625-3333.

Ingestion of Sharp Objects
- Objects longer than 5 cm and wider than 2 cm rarely pass the stomach.
- Open safety pins, razor blades, and other extremely pointed edges require removal before they pass from the stomach, because 15 to 35 percent will cause intestinal perforation, generally at the ileocecal valve.
- Children who have swallowed sharp objects should have radiographs and an examination.[5] Asymptomatic children can be followed with serial exams and x-rays.
- Children who are symptomatic or have swallowed a sewing needle require surgical consultation.

Cocaine Ingestion
- A condom packet can hold up to 5 g of cocaine.
- Rupture of one packet may be fatal.
- Surgery is the safest method of retrieval, although spontaneous passage may occur.

Foreign Body Retrieval
- Endoscopy is the procedure of choice for foreign body removal, except for cocaine.
- Consultation and possible admission are required with sharp or elongated objects, multiple foreign bodies, button batteries, evidence of perforation, a child with a nickel or quarter at the level of the cricopharyngeus muscle, airway compromise, or presence of foreign body for more than 24 h.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 72, "Swallowed Foreign Bodies," by Wade R. Gaasch and Robert A. Barish.

42 PEPTIC ULCER DISEASE AND GASTRITIS

Mark R. Hess

EPIDEMIOLOGY

- The great majority of peptic ulcers are directly related to infection with *Helicobacter pylori* or nonsteroidal anti-inflammatory drug (NSAID) use.[1,2] For white Americans below age 35, the rate of *H. pylori* infection is 10 percent, and this rate climbs to 80 percent by age 75. Black Americans have a higher infection rate than white Americans of 45 percent below age 25.[3]
- One out of 10 Americans over age 17 will have peptic ulcer disease (PUD) at some time.[4,5]

PATHOPHYSIOLOGY

- Acid and pepsin destroy gastric and duodenal mucosa and contribute to ulcer formation usually after *H. pylori* has broken down the protective mucous gel. *H. pylori* infection is present in 80 percent of gastric ulcers and 95 percent of duodenal ulcers.[6]
- *H. pylori* infection generally causes a chronic active form of gastritis, but development of PUD occurs in only 10 to 20 percent.[3,7] Eradication of *H. pylori* reduces recurrence rates by 65 percent in duodenal ulcers and by 40 percent in gastric ulcers.[6]
- Nonsteroidal anti-inflammatory drugs inhibit prostaglandin synthesis. This contributes to ulcer formation by lowering mucous and bicarbonate production as well as mucosal blood flow.[2]
- All ulcers generally respond to traditional therapy that inhibits acid production.
- Acute gastritis is generally caused by ischemia due to severe illness (burns, trauma, shock, etc.), direct toxic effects (NSAIDs, alcohol, etc.), or *H. pylori* infection.

CLINICAL FEATURES

- The most common symptom of PUD includes burning epigastric pain, 1 to 3 h after meals, classically awakening the patient at night and relieved with food, milk, or antacids. Acute gastritis may also present with nausea and vomiting, although the most common presentation is gastrointestinal (GI) bleeding (microscopic to gross blood).
- The only physical sign of PUD may be epigastric tenderness unless a complication has occurred. Complications may present with rigid abdomen (perforation), abdominal distention and vomiting (obstruction), or GI bleeding. Gastric ulcers may perforate posteriorly, causing pancreatitis, which presents with midback pain.

DIAGNOSIS AND DIFFERENTIAL

- Classic history with epigastric tenderness may suggest PUD, but a definitive diagnosis cannot be made clinically.[8] A definitive diagnosis is made by an upper GI series or endoscopy (with endoscopy having the highest yield).[1]
- The best emergency department (ED) test to detect *H. pylori* is a serologic study to detect IgG antibodies, which has a high sensitivity and specificity at a cost of $40 to $50 and a turnaround time that is less than 1 h. Antibodies do remain elevated for several years after eradication, however.
- Disorders to consider in differential diagnosis of PUD include gastritis, gastroesophageal reflux disease (GERD), pancreatitis, hepatitis, cholelithiasis, cardiac ischemia, abdominal aortic aneurysm (AAA), and gastroparesis.
- Ancillary tests to consider include a complete blood count to look for anemia in GI bleeding or abdominal aortic aneurysm leak and elevated white blood count in cholecystitis or pancreatitis; abdominal ultrasound for cholelithiasis and abdominal aortic aneurysm electrocardiogram and cardiac enzymes for cardiac ischemia; liver function tests for hepatitis and cholelithiasis; and amylase/lipase for pancreatitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The acute treatment of PUD in the ED consists of antacids to neutralize gastric hydrochloric acid.[1] Often this is combined with viscous lidocaine.
- H_2 receptor antagonists (H_2RAs) or proton-pump inhibitors are usually instituted for ongoing therapy to promote ulcer healing.[1,9] Proton-pump inhibitors will heal ulcers faster and may have an inhibitory effect against *H. pylori.*[1,9]
- Finally, if acute infection with *H. pylori* is found, antimicrobial and antisecretory therapy is instituted. Example regimen includes metronidazole (Flagyl) 500 mg bid, clarithromycin (Biaxin) 500 mg bid, and omeprazole (Prilosec) 20 mg bid for 10 days. This is followed by 10 more days of omeprazole 20 mg every day. This will not speed recovery but will help prevent recurrence.[6]
- All patients should receive discharge instructions including avoidance of NSAIDs, alcohol, tobacco, caffeine, and non-enteric-coated aspirin. Early follow-up should be sought for patients at high risk for cancer, including those with anorexia, dysphagia, anemia, and weight loss, or the elderly.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 73, "Peptic Ulcer Disease and Gastritis," by Matthew C. Gratton.

43 APPENDICITIS

David L. Leader, Jr.

EPIDEMIOLOGY

- The overall incidence or appendicitis is approximately 1 case per 1000 population per year. Some 6 percent of the population will experience appendicitis at some time in their lives. In the United States, an estimated 1 million hospital days annually can be attributed to acute appendicitis.[1]
- Acute appendicitis can be extremely difficult to diagnose, and misdiagnosis remains an important cause of successful malpractice claims against emergency physicians.[2]

PATHOPHYSIOLOGY

- Acute appendicitis develops from obstruction of the lumen of the appendix. Increased luminal pressure leads to vascular compromise, bacterial invasion, inflammatory response, and resultant tissue necrosis.
- Classically, appendicitis is associated with the migration of pain from the periumbilical area to the right lower quadrant. However there are many exceptions to this classic presentation; these are often due to the variability of the anatomic location of the appendix (i.e., retrocecal, retroileal, pelvic appendix, or gravid uterus causing displacement).[3]

CLINICAL FEATURES

- A summary of clinical examination operating characteristics for appendicitis are listed in Table 43-1.
- The primary and most reliable symptom in acute appendicitis is abdominal pain.
- Right-lower-quadrant pain is 81 percent sensitive and 53 percent specific for the diagnosis of acute appendicitis. Migration of the pain from an initial periumbilical site to the right lower quadrant is 64 percent sensitive and 82 percent specific for the diagnosis of acute appendicitis.[4]
- After the onset of vague abdominal pain, the classic triad of symptoms in appendicitis includes anorexia, nausea, and vomiting. Some 60 percent of patients with appendicitis will have some combination of these symptoms, but they are by themselves neither specific nor sensitive for appendicitis.[4]
- McBurney's point tenderness, Rovsing's sign, the psoas sign, the obturator sign, rectal exam tenderness, and rebound tenderness are all clinical exam findings that may be present.
- Fever in appendicitis is a relatively late finding

TABLE 43-1 Summary of Clinical Examination Operating Characteristics for Appendicitis*

PROCEDURE	SENSITIVITY	SPECIFICITY	LR + (95% CI)	LR − (95% CI)
Right-lower-quadrant pain	0.81	0.53	7.31–8.46†	0–0.28†
Rigidity	0.27	0.83	3.76 (2.96–4.78)	0.82 (0.79–0.85)
Migration	0.64	0.82	3.18 (2.41–4.21)	0.50 (0.42–0.59)
Pain before vomiting‡	1.00	0.64	2.76 (1.94–3.94)	NA
Psoas sign	0.16	0.95	2.38 (1.21–4.67)	0.90 (0.83–0.98)
Fever	0.67	0.79	1.94 (1.63–2.32)	0.58 (0.51–0.67)
Rebound tenderness test	0.63	0.69	1.10–6.30†	0–0.86†
Guarding	0.74	0.57	1.65–1.78†	0–0.54†
No similar pain previously	0.81	0.41	1.50 (1.36–1.66)	0.323 (0.246–0.424)
Rectal tenderness	0.41	0.77	0.83–5.34†	0.36–1.15†
Anorexia	0.68	0.36	1.27 (1.16–1.38)	0.64 (0.54–0.75)
Nausea	0.58	0.37	0.69–1.20†	0.70–0.84†
Vomiting	0.51	0.45	0.92 (0.82–1.04)	1.12 (0.95–1.33)

* LR + indicates the positive likelihood ratio with its 95% CI and LR−, the negative likelihood ratio with its 95% CI.
† In heterogeneous studies, the LRs are reported as ranges.
‡ Only one study on this is included in the meta-analysis.
SOURCE: From Wagner et al.,[4] with permission.

and rarely exceeds 39°C (102.2°F) unless rupture or other complications occur.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of appendicitis is primarily clinical. Factors that increase the likelihood of appendicitis, listed in decreasing order of importance, are right-lower-quadrant pain, rigidity, migration of pain to the right lower quadrant, pain before vomiting, a positive psoas sign, rebound tenderness, and guarding.
- If the diagnosis is unclear, additional studies such as a complete blood count, urinalysis, pregnancy test, and imaging studies should be considered.
- An elevation of the white blood count is sensitive but has a very low specificity for appendicitis.[5] The positive and negative predictive values of an elevated white blood cell (WBC) count in acute appendicitis are 92 and 50 percent, respectively.[6]
- Obtaining a urinalysis is important to rule out other diagnoses, such as urolithiasis or urinary tract infection; however, pyuria and hematuria can occur if an inflamed appendix overlies a ureter.[7]
- Pregnant and nonpregnant patients have an equal likelihood of developing appendicitis.[8]
- Plain radiographs of the abdomen are often abnormal but are not specific.[5] X-ray findings of possible acute appendicitis include appendiceal fecalith, appendiceal gas, localized paralytic ileus, blurred right psoas muscle, and free air.
- Ultrasonography has a high sensitivity but is limited in evaluating a ruptured appendix or an abnormally located (e.g., retrocecal) appendix.[9,10]
- Computed tomography (CT) is more sensitive than ultrasound (98 vs 87 percent), with comparable specificity (95 vs 97 percent), and may provide an alternative diagnosis.[11] CT findings suggesting acute appendicitis include pericecal inflammation, abscess, periappendiceal phlegmon, and fluid collections.
- In order to avoid premature surgical intervention or discharge of the patient with an uncertain diagnosis, patients with atypical presentations may be observed with serial abdominal examination.[12,13]
- Patients under the age of 6 and elderly patients have higher rates of misdiagnosis of appendicitis, leading to increased morbidity and mortality.
- Appendicitis is the most common extrauterine surgical emergency in pregnancy; if perforation and peritonitis occur, fetal mortality rates are high.[14]
- Patients with AIDS have an increased risk of complications from appendicitis because of delays in diagnosis due to their immunocompromised state.[15]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Before undergoing surgery, patients should have nothing by mouth and should have intravenous (IV) access, analgesia, and antibiotic therapy started.
- Narcotic analgesics are preferred, since they can be reversed by naloxone if needed. The dosage of morphine is 0.1 mg/kg.
- Antibiotics are most effective when given prior to surgery and should cover anaerobic flora, enterococci, and gram-negative intestinal flora.
- Recommended choices include metronidazole 15 mg/kg IV (up to 1 g), ampicillin 50 mg/kg IV (up to 2 g), gentamicin 1 mg/kg IV, or single-agent coverage with a second- or third-generation cephalosporin, such as cefoxitin, 20 to 40 mg/kg IV (up to 2 g).[16,17]
- If, after workup and surgical consultation, no precise diagnosis is obtained, patients should not be given any specific diagnostic label (e.g., nonspecific abdominal pain).
- Patients should be discharged with specific instructions to consult their primary care physician for close medial follow-up and to return if their condition worsens—if they develop increased pain, fever, or nausea.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., See Chap. 74, "Appendicitis," by Dennis J Fitzgerald and Arthur M. Pancioli.

44 INTESTINAL OBSTRUCTION

Roy L. Alson

EPIDEMIOLOGY

- Small bowel obstruction (SBO) is more common than large bowel obstruction (LBO).
- Intestinal obstruction is due to mechanical obstruction or functional (adynamic or paralytic ileus) with ileus being more common. Mechanical obstruction may be due to either intrinsic or extrinsic mechanisms.
- Adhesions following surgery are the most common cause of SBO.[1] Incarcerated inguinal hernias are the second most common cause of SBO. Other causes of bowel obstruction are listed in Table 44-1.
- Large bowel obstruction is most commonly due to neoplasm.[2] Fecal impaction is common in elderly and debilitated patients.
- Complications and mortality rise above 60 years of age.[1] Mortality also increases dramatically if corrective surgery is delayed beyond 24 h.[3]
- Ileus may be due to injury, infection, medications, or electrolyte abnormalities.

TABLE 44-1 Common Causes of Intestinal Obstruction

DUODENUM	SMALL BOWEL	COLON
Stenosis	Adhesions	Carcinoma
Foreign body (Bezoars)	Hernia	Fecal impaction
Stricture	Intussusception	Ulcerative colitis
Superior mesenteric artery syndrome	Lymphoma	Volvulus
	Stricture	Diverticulitis (stricture, abscess)
		Intussusception
		Pseudoobstruction

PATHOPHYSIOLOGY

- Blockage prevents passage of luminal contents and results in dilatation due to accumulation of gastric, biliary, and pancreatic secretions.
- With distention, intraluminal pressure rises, decreasing bowel wall blood flow. When pressure exceeds capillary pressure, absorption ceases and leakage of fluids (third-spacing) may occur. Microvascular changes may allow entry of gut flora into the circulation, resulting in bacteremia and sepsis. Necrosis and bowel perforation may follow.
- With obstruction, oral fluid intake stops and vomiting occurs. This fluid loss, coupled with the third space losses mentioned earlier, lead to hypovolemia and shock.[2]
- Closed-loop obstruction has a more rapid progression.

CLINICAL FEATURES

- Classic history includes vomiting, abdominal distention, and pain, with a past history of abdominal surgery or hernia.
- Abdominal pain is crampy and intermittent. Small bowel obstruction results in primarily periumbilical pain versus hypogastric pain for LBO.[1,4] Pain with ileus may be constant.

- Emesis is often bilious early and may be feculent with late SBO or with LBO.
- Early in the disease course, bowel sounds have ''high-pitched rushes,'' but this finding diminishes with time.
- The patient may have surgical scars, hernias (reducible?), or intraabdominal masses.
- Peritoneal signs suggest perforation.
- Clinical signs of dehydration and/or shock may be present (tachycardia or hypotension).
- Rectal exam may reveal impaction, occult blood, or carcinoma. Passage of stool does not rule out obstruction.
- Women may have palpable gynecologic neoplasms on pelvic exam.

DIAGNOSIS AND DIFFERENTIAL

- Radiographs help localize SBO versus LBO. Plicae circulares are linear densities that traverse the small bowel lumen. Haustrae in the large bowel do not extend fully across the lumen.
- Dilated loops of bowel on supine with stepladder air-fluid levels on upright film are diagnostic (see Fig. 44-1). Upright or decubitus film should be noted for free air suggesting perforation and for pneumonia or pleural effusions on the chest film.
- Laboratory tests include complete blood cell count, blood urea nitrogen levels, serum electrolyte levels, serum amylase level, and urinalysis. Liver function tests as well as cross-match and coagulation studies may also be needed.
- Leukocytosis with a left shift may suggest peritonitis, gangrene of the bowel, or an abscess.[2] Serum lactate may be useful in assessing presence of mesenteric vascular occlusion.
- As dehydration and shock develop, elevated urine specific gravity and metabolic acidosis may be seen along with hemoconcentration.
- Sigmoidoscopy or barium enema may be useful in localizing the site of LBO.
- Contrast-enhanced abdominal computed tomography has been advocated to identify partial versus complete bowel obstruction.[5]
- Pseudoobstruction (Ogilvie's syndrome) is most commonly seen in the low colonic region.[6] Intestinal motility is depressed (often due to tricyclic antidepressants or anticholinergic agents), resulting in large volumes of retained gas. Air-fluid levels are rarely seen on x-ray. Pseudoobstruction is treated by colonoscopy.

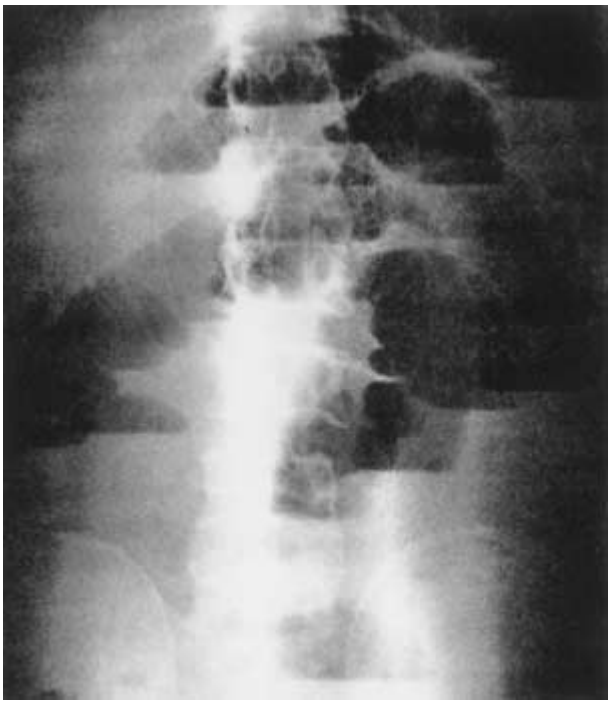

FIG 44-1 Upright film demonstrates multiple air-fluid levels and ''stepladder'' appearance. (From Harris JH, Harris WH: *The Radiology of Emergency Medicine,* 3d ed. Baltimore, Williams & Wilkins, 1993, p 843, with permission.)

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- With mechanical bowel obstruction, prompt surgical consultation is required.
- A nasogastric tube is used to decompress the bowel. Use of long intestinal tubes in the emergency department is not indicated.
- Fluid resuscitation should be started, using crystalloid replacement. Vital signs and urine output should be monitored to measure response to fluids.
- Appropriate antibiotic therapy (cefoxitin or similar agents) should be started.
- For adynamic ileus, conservative treatment including nasogastric decompression, fluid replacement, and observation is usually effective.

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For further reading in *Emergency Medicine, A Comprehensive Study Guide,* 5th ed., see Chap. 75, ''Intestinal Obstruction,'' by Salvator J. Vicario and Timothy G. Price.

45 HERNIA IN ADULTS AND CHILDREN

Maryanne W. Lindsay

- A hernia is an external or internal protrusion of a body part from its natural location.

EPIDEMIOLOGY

- Abdominal wall hernias occur in 6 locations: inguinal, femoral, umbilical, anterior abdominal, pelvic, or lumbar (see Fig. 45-1).
- Predisposing factors include prematurity, family history, genitourinary abnormalities, ascites, peritoneal dialysis, ventriculoperitoneal shunt, cystic fibrosis, lung disease, pregnancy, or wounds.
- Groin hernias occur more frequently in males. Indirect inguinal hernias in males are more common on the right side due to later passage of the right testis and have a bimodal incidence, with peaks in infants and in adults older than 40 years. Umbilical and femoral hernias are more common in females. Anterior abdominal wall hernias have a similar incidence in both genders.

PATHOPHYSIOLOGY

- Hernias occur in structural areas with inherent weakness, including penetration sites for extraperitoneal structures, areas lacking strong multilayer support, and wound sites (either surgical or traumatic).
- The specific hernia types include the following: (*a*) an indirect inguinal hernia passes through the inguinal canal, which is an internal ring defect lateral to the inferior epigastric vessels; (*b*) a direct inguinal hernia occurs primarily in adults and is an acquired defect through the external ring medial to the interior epigastric vessels; (*c*) a femoral hernia protrudes below the inguinal ligament in the femoral canal; (*d*) the umbilical hernia occurs in infants; (*e*) the epigastric hernia passes through the linea alba of the rectus sheath above the umbilicus; (*f*) the Spigelian hernia occurs at the site

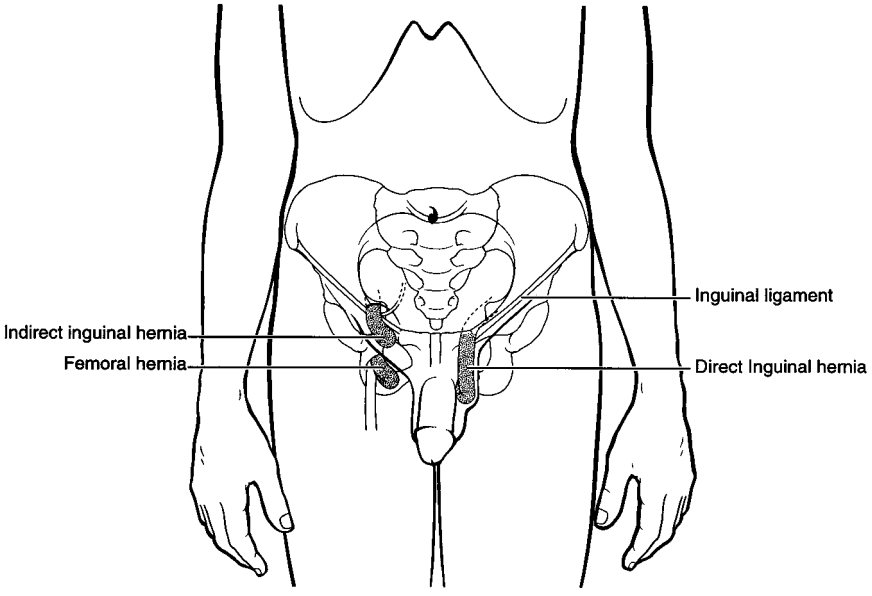

FIG. 45-1 Groin hernias.

of the semilunar or arcuate line, just lateral to the rectus muscle; (*g*) pelvic hernias are rare and pass through sciatic foramen; (*h*) lumbar hernias are extremely rare; and (*i*) incisional hernias occur through incision sites and are more likely with infection and obesity.
- Complicated hernia types include the following: (*a*) a sliding hernia includes a viscus, most frequently the colon, forming one wall of the herniation; and (*b*) a Richter hernia involves incarceration of a wall of hollow viscus.

CLINICAL FEATURES

- Symptoms may include pain, nausea, and vomiting, or possibly even clinical toxicity. Infants may exhibit irritability.
- Complications include the following: (*a*) inclusion of a viscus (a sliding hernia); (*b*) incarceration, or irreducibility; (*c*) vascular compromise of the incarcerated contents (strangulation); (*d*) bowel obstruction due to incarceration and local edema; (*e*) bowel perforation due to strangulation; and (*f*) gangrene, abscess formation, peritonitis, and sepsis due to ischemic bowel.

DIAGNOSIS AND DIFFERENTIAL

- Most of the previously described hernias are palpable on exam; however, the Spigelian hernia is frequently intraperitoneal and may not be detectable on physical exam.
- Radiographs are useful to exclude bowel obstruction or perforation.
- A groin hernia must be differentiated from a lymph node, vascular aneurysm, scrotal hydrocele, epididymitis, testicular torsion, undescended testis, or tumor.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Recently incarcerated hernias may be gently reduced in the emergency department. The patient may be discharged with outpatient surgical referral. Infants with inguinal hernias have a high risk of incarceration and should be referred for surgical repair within a few days after discovery.[1] In contrast, umbilical hernias rarely incarcerate.
- For cases suspected of strangulation and ischemic bowel, treatment should alternatively include broad-spectrum antibiotics, intravenous fluids, nasogastric decompression, and an immediate surgical consultation.

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46 ILEITIS, COLITIS, AND DIVERTICULITIS

David M. Cline

CROHN'S DISEASE

- Crohn's disease—also described as regional enteritis, terminal ileitis, and granulomatous ileocolitis—is an idiopathic gastrointestinal (GI) tract disease. Segmental involvement of any part of the GI tract from the mouth to the anus by a nonspecific granulomatous process characterizes the disease.

EPIDEMIOLOGY

- The peak incidence of Crohn's disease occurs in patients between 15 and 33 years of age with a secondary peak at age 55 to 60 years.
- The prevalence varies from 10 to 100 cases per 100,000 population and the incidence from 1 to 7 cases per year per 100,000 population in the United States. The incidence of Crohn's disease has been increasing over the past 20 years.[1]
- There is a 20 to 30 percent increased risk of Crohn's disease among women as compared to men. It is four times more common among Jews than non-Jews and is more common in whites than in blacks, Asians, or Native Americans.

PATHOPHYSIOLOGY

- The cause is still unknown.
- The most important pathologic feature of Crohn's disease is the involvement of all the layers of the bowel and extension into mesenteric lymph nodes. In addition the disease is discontinuous, with normal areas alternating with diseased areas.

CLINICAL FEATURES

- Abdominal pain, anorexia, diarrhea, and weight loss are present in up to 80 percent of cases, although the clinical course is variable and unpredictable.
- Patients commonly experience an insidious onset of recurring fever, abdominal pain, and diarrhea over several years without a definitive diagnosis.
- Many patients develop perianal fissures or fistulas, abscesses, or rectal prolapse. Fistulas occur between the ileum and sigmoid colon, the cecum, another ileal segment, or the skin. Abscesses are characterized as intraperitoneal, retroperitoneal, interloop, or intramesenteric.
- Growth retardation can be seen in children.[2]
- Obstruction, hemorrhage, and toxic megacolon also occur. Half of all cases of toxic megacolon occur in patients with Crohn's disease, frequently associated with massive GI bleeding.[3]
- Up to 30 percent of patients develop extraintestinal manifestations including arthritis, uveitis, or liver disease.
- Common hepatobiliary complications include gallstones, pericholangitis, and chronic active hepatitis.
- Some patients develop thromboembolic disease as a result of a hypercoagulable state; they have a 25 percent mortality rate.
- Malabsorption, malnutrition, and chronic anemia develop in long-standing disease, and the incidence of GI tract malignant neoplasm is triple that of the general population.

DIAGNOSIS AND DIFFERENTIAL

- The definitive diagnosis of Crohn's disease is usually established months or years after the onset of symptoms. Common misdiagnoses are appendicitis and pelvic inflammatory disease.
- A careful and detailed history for previous bowel symptoms that preceded acute presentation may provide clues to the correct diagnosis. The absence of true guarding or rebound is noted.
- Peritonitis and leukocytosis can be masked in patients taking glucocorticoids.
- The differential diagnosis of Crohn's disease includes lymphoma, ileocecal amebiasis, tuberculosis, Kaposi's sarcoma, *Campylobacter* enteritis, and *Yersinia* ileocolitis. Most of these are uncommon, and the latter two can be differentiated by stool cultures.
- Laboratory evaluation should include a complete blood count (CBC), chemistries, and blood bank testing when indicated.
- Plain abdominal radiography will identify obstruction and toxic megacolon, which may appear as a long, continuous segment of air-filled colon greater than 6 cm in diameter.
- Computerd tomography or ultrasound of the abdomen best identifies abscesses and fistulas.
- A definitive diagnosis is confirmed by an upper GI series, an air-contrast barium enema, and colonoscopy.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Sulfasalazine 3 to 4 g/d is effective for mild to moderate active Crohn's disease but has multiple toxic side effects, including GI and hypersensitivity reactions.
- Glucocorticoids (prednisone) 40 to 60 mg/d are reserved for severe small intestinal disease and ileocolitis.
- Immunosuppressive drugs, 6-mercaptopurine (1 to 1.5 mg/kg/d) or azathioprine (2 mg/kg/d), are used as steroid-sparing agents, in healing fistulas, and in patients with serious surgical contraindications.
- Metronidazole 10 to 20 mg/kg/d or ciprofloxacin 500 to 750 mg twice daily is useful in patients with perianal complications and fistulous disease.
- Diarrhea can be controlled by loperamide 4 to 16 mg/d, diphenoxylate 5 to 20 mg/d, and cholestyramine 4 g one to six times per day.
- Patients who should be admitted include those who demonstrate signs of fulminant colitis, peritonitis, obstruction, significant hemorrhage, severe dehydration, or electrolyte balance or those with less severe disease who fail outpatient management.
- Surgical intervention is indicated in patients with intestinal obstruction or hemorrhage, perforation, abscess or fistula formation, toxic megacolon, perianal disease, and sometimes in those who fail medical therapy.
- The recurrence rate after surgery is nearly 100 percent.

ULCERATIVE COLITIS

- Ulcerative colitis is an idiopathic chronic inflammatory and ulcerative disease of the colon and rectum characterized most often clinically by bloody diarrhea.

EPIDEMIOLOGY

- Ulcerative colitis is more prevalent in the United States and northern Europe.
- Peak incidence occurs in the second and third decades of life.
- The incidence of ulcerative colitis is about 10 cases per 100,000 and is increasing.[1]
- There is a slight predominance in men.

PATHOLOGY

- Ulcerative colitis involves primarily the mucosa and submucosa.
- Microscopically, the disease is characterized by mucosal inflammation with formation of crypt abscesses, epithelial necrosis, and mucosal ulceration.
- The rectosigmoid colon is involved in 95 percent of cases.

CLINICAL FEATURES

- Ulcerative colitis is commonly characterized by intermittent attacks of acute disease with complete remission between bouts.
- Patients with mild disease (60 percent) may present with constipation and rectal bleeding, fewer than four bowel movements per day, no systemic symptoms, and few extraintestinal manifestations.[4]
- Severe disease (15 percent) is associated with more than six bowel movements per day, weight loss, fever, tachycardia, anemia, and more frequent extraintestinal manifestations, including peripheral arthritis, ankylosing spondylitis, episcleritis, uveitis, pyoderma gangrenosum, and erythema nodosum.[4]
- Ninety percent of the mortality from ulcerative colitis occurs in patients with severe disease.[4]
- The most common complications are hemorrhagic blood loss and toxic megacolon.
- Mortality from perforation is 50 percent, but this is reduced to 10 percent if surgery is undertaken prior to perforation.[5]
- Abscess and fistula formation, which is much more common in patients with Crohn's disease, occurs in 20 percent of patients with ulcerative colitis.[6] Obstruction secondary to stricture formation and acute perforation are other complications.
- There is a 10- to 30-fold risk of developing colon carcinoma.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of ulcerative colitis may be considered with a history of abdominal cramps, diarrhea, and mucoid stools. Laboratory findings are nonspecific and may include leukocytosis, anemia, thrombocytosis, decreased serum albumin, abnormal liver function tests, and negative stool studies for ova, parasites, and enteric pathogens.
- Barium enema can confirm the diagnosis and defines the extent of colonic involvement, but colonoscopy is the most sensitive method. Rectal biopsy can exclude amebiasis and metaplasia.
- Rigid or fiberoptic proctosigmoidoscopic examination is abnormal in 95 percent of patients with ulcerative colitis and can be used in severely ill patients.
- The differential diagnosis includes infectious, ischemic, irradiation, pseudomembranous, and Crohn's colitis. When the disease is limited to the rectum, consider sexually acquired diseases such as rectal syphilis, gonococcal proctitis, lymphogranuloma venerum, and inflammation caused by herpes simplex virus, *Entamoeba histolytica, Shigella,* and *Campylobacter.*

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients who have not previously been treated with steroids respond best to adrenocorticotropic hormone (ACTH) 120 U/d.[7]
- Patients on steroids should receive hydrocortisone 300 mg/d, methylprednisolone 48 mg/d, or prednisone 60 mg/d.[7]
- Cyclosporine 4 mg/kg/d has been advocated for cases of fulminant colitis that have failed treatment with intravenous steroids.[8]
- Patients with significant gastrointestinal hemorrhage, toxic megacolon, and bowel perforation should be admitted with consultation to both a gastroenterologist and a surgeon.
- The majority of patients with mild and moderate disease can be treated as outpatients. Therapy

listed below should be discussed with a gastroenterologist, and close follow-up must be ensured.

1. Prednisone 40 to 60 mg/d is usually sufficient and can be adjusted depending on the severity of the disease. Once clinical remission is achieved, steroids should be slowly tapered and discontinued, as there is no evidence that maintenance dosages of steroids reduce the incidence of relapses.
2. Sulfasalazine 1.5 to 2 g/d is inferior to steroids in treating acute attacks and is most useful in maintenance therapy by reducing the recurrence rate.
3. Topical steroid preparations—such as beclomethasone, hydrocortisone, tixocortol, or budesonide—can be used acutely and to maintain remission.
4. Supportive measures include replenishment of iron stores, dietary elimination of lactose, and addition of bulking agents, such as psyllium (Metamucil). Antidiarrheal agents can precipitate toxic megacolon and should be avoided.

PSEUDOMEMBRANOUS COLITIS

- Pseudomembranous colitis is an inflammatory bowel disorder in which membrane-like yellowish plaques of exudate overlie and replace necrotic intestinal mucosa.

EPIDEMIOLOGY

- *Clostridium difficile* is a spore-forming obligate anaerobic bacillus that causes pseudomembranous colitis.
- The incidence of this disease has been increasing in recent years, coincident with the increased spectrum of antibiotics in use throughout the United States.
- Three different syndromes have been described: neonatal pseudomembranous enterocolitis, postoperative pseudomembranous enterocolitis, and antibiotic-associated pseudomembranous colitis.
- *C. difficile* is the most common enteric pathogen associated with nosocomial diarrhea.[9]

PATHOPHYSIOLOGY

- Hospitalized patients are colonized with *C. difficile* in 10 to 25 percent of cases.
- Broad-spectrum antibiotics—most notably clindamycin, cephalosporins, and ampicillin/amoxicillin—alter the gut flora in such a way that toxin-producing *C. difficile* can flourish within the colon, producing clinical manifestations of pseudomembranous colitis.
- Chemotherapeutic agents[10] and antiviral agents[11] have been implicated as well.

CLINICAL FEATURES

- Clinical manifestations can vary from frequent watery, mucoid stools to a toxic picture including profuse diarrhea, crampy abdominal pain, fever, leukocytosis, and dehydration.
- Examination of the stool may reveal fecal leukocytes. Toxic megacolon or colonic perforation occurs rarely.

DIAGNOSIS AND DIFFERENTIAL

- The disease typically begins 7 to 10 days after the institution of antibiotics, but the range is from a few days up to 8 weeks.
- The diagnosis is confirmed by the demonstration of *C. difficile* in the stool and by the detection of toxin in stool filtrates. Colonoscopy is not routinely needed to confirm the diagnosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of pseudomembranous colitis includes discontinuing antibiotic therapy, initiating intravenous fluid replacement, and correcting electrolyte abnormalities. This is effective without additional treatment in 25 percent of patients.
- Metronidazole 250 mg or vancomycin 125 to 250 mg PO four times daily is the treatment of choice in patients with mild to moderate disease who do not respond to supportive measures. Vancomycin should be reserved for patients who have not responded to or are intolerant of metronidazole and for pregnant patients.[12,13]
- Patients with severe diarrhea, those with a systemic response (fever, leukocytosis, severe abdominal pain), and those whose symptoms persist despite appropriate outpatient management must be hospitalized and should receive vancomycin 125 to 250 mg 4 times daily for 10 d. The symptoms usually resolve within a few days.
- Antidiarrheal agents may prolong or worsen symptoms and should be avoided.

DIVERTICULITIS

- Diverticulitis is an acute inflammatory process caused by bacterial proliferation within an existing colonic diverticulum.

EPIDEMIOLOGY

- Clinical diverticulitis occurs in 10 to 25 percent of patients with diverticulosis. One-third of the population will have acquired the disease by age 50, and two-thirds by age 85.[14]
- Only 2 to 4 percent of patients with diverticulitis are under the age of 40, but the younger age group tends to have a more virulent form of the disease, with frequent complications requiring earlier surgical intervention.[15]

PATHOPHYSIOLOGY

- A pathophysiologic mechanism to explain the development of diverticular disease is not apparent. It is still unresolved whether diverticular disease is a disorder of colonic motility, a colonic muscle abnormality, a connective tissue disorder, or a normal concomitant of aging. A low-residue diet has been implicated as a major factor in the pathogenesis of diverticular disease.

CLINICAL FEATURES

- The most common symptom is a steady, deep discomfort in the left lower quadrant of the abdomen. Other symptoms include tenesmus and changes in bowel habits, such as diarrhea or increasing constipation.
- The involved diverticulum can irritate the urinary tract and cause frequency, dysuria, or pyuria.
- If a fistula develops between the colon and the bladder, the patient may present with recurrent urinary tract infections or pneumaturia.
- Paralytic ileus with abdominal distention, nausea, and vomiting may develop secondary to intraabdominal irritation and peritonitis. Small bowel obstruction and perforation can also occur.
- Right-lower-quadrant pain, which may be indistinguishable from acute appendicitis, can occur with ascending colonic diverticular involvement and in patients with a redundant right-sided sigmoid colon.
- Physical examination frequently demonstrates a low-grade fever, but the temperature may be higher in patients with generalized peritonitis and in those with an abscess.
- The abdominal exam reveals localized tenderness, often with voluntary guarding and rebound tenderness. A fullness or mass may be appreciated over the affected area of colon.
- Twenty-five percent of patients demonstrate occult blood.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis includes appendicitis, peptic ulcer disease, pelvic inflammatory disease, endometriosis, ischemic colitis, aortic aneurysm, renal calculus, irritable bowel syndrome, lactate intolerance, colon carcinoma, intestinal lymphoma, Kaposi's sarcoma, sarcoidosis, collagen vascular disease, irradiation colitis or proctosigmoiditis, fecal impaction, foreign-body granuloma, and any bacterial, parasitic, or viral infectious cause.
- Laboratory studies should include routine screening blood tests, urinalysis, and an abdominal radiographic series.
- Leukocytosis is present in only 36 percent of patients with diverticulitis.
- The abdominal series may be normal or may demonstrate an associated ileus, partial small bowel obstruction, colonic obstruction, free air indicating bowel perforation, or extraluminal collections of air suggesting a walled-off abscess.
- Computed tomography of the abdomen is the diagnostic procedure of choice and may demonstrate presence of diverticulae, inflammation of pericolic fat, bowel wall thickening, or peridiverticular abscess.[16,17]
- Barium contrast studies can easily demonstrate diverticulae but are insensitive to the presence of diverticulitis.[17]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Inpatient treatment includes intravenous antibiotics, usually an aminoglycoside, such as gentamicin or tobramycin (1.5 mg/kg), and either metronidazole (500 mg) or clindamycin (300 to 600 mg) for aerobic and anaerobic organism coverage. Ticarcillin-clavulanic acid and imipenem have been used as alternate agents.
- The patient is placed on bowel rest, nothing by mouth is given, and intravenous fluids are administered. Nasogastric suction may be indicated in pa-

tients with bowel obstruction or adynamic ileus, and surgical consultation should be obtained.[18]

- Outpatient management is acceptable for patients with localized pain without signs and symptoms of local peritonitis or systemic infection. Treatment consists of bowel rest and broad-spectrum oral antibiotic therapy. Common agents effective against aerobic organisms include ampicillin (500 mg q6h), trimethoprim/sulfamethoxazole (2 tablets q12h), ciprofloxacin (500 mg q12h), or cephalexin (500 mg q6h). One of these medications is taken in combination with an agent effective against anaerobic organisms, such as metronidazole (500 mg q8h) or clindamycin (300 mg q6h). Patients should limit activity and maintain a liquid diet for 48 h. If symptoms improve, low-residue foods are added to the diet. Patients are advised to contact their physician or return to the emergency department if they develop increasing abdominal pain, fever, or malaise.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 77, "Ileitis, Colitis, and Diverticulitis," by Howard A. Werman, Hagop S. Mekhjian, and Douglas A. Rund.

47 ANORECTAL DISORDERS

Maryanne W. Lindsay

HEMORRHOIDS

EPIDEMIOLOGY

- Hemorrhoids are associated with constipation and straining at stool, pregnancy, obesity, chronic liver disease, and, rarely, tumors of the rectum and sigmoid colon.

PATHOPHYSIOLOGY

- Internal hemorrhoidal veins are located above the dentate line and drain into the portal venous system (see Fig. 47.1).

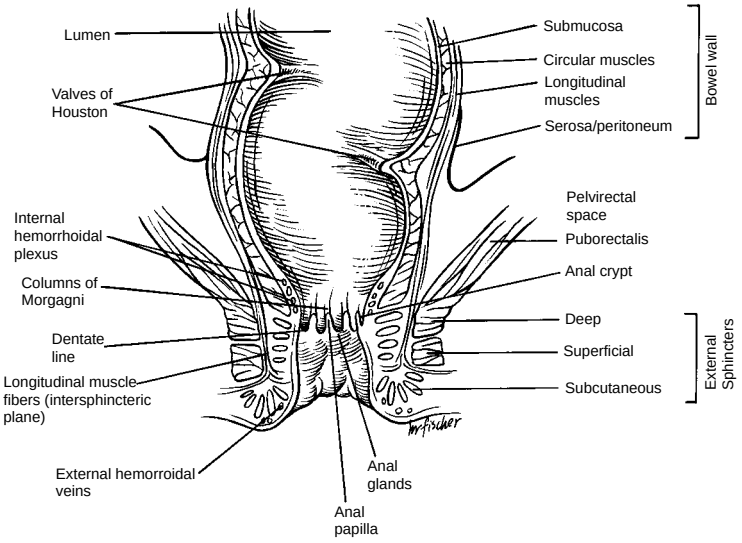

FIG. 47-1 Coronal section of the anorectum.

- External hemorrhoidal veins are located below the dentate line and drain through the pudendal and iliac venous systems.

CLINICAL FEATURES

- Internal hemorrhoids are only visible through an anoscope and cause painless, bright-red rectal bleeding with defecation. Constant locations are 2, 5, and 9 o'clock positions in a prone position.
- External hemorrhoids may be visualized on external exam and commonly cause pain and discomfort, most severe at the time of defecation.
- Thrombosis of external hemorrhoids is the usual cause of pain.
- Prolapse may occur with larger hemorrhoids, spontaneously reducing or requiring periodic manual reduction. Failure to reduce may lead to incarceration and even gangrene, requiring surgical intervention. Prolapse may cause mucous discharge and pruritus ani.
- The common complications of hemorrhoids are strangulation, thrombosis, and severe bleeding.

DIAGNOSIS AND DIFFERENTIAL

- Clinical signs cannot differentiate colonic lesions from hemorrhoids.[1]
- Other causes of rectal pain include malignancy, abscess, cryptitis, anal fissure, trauma, foreign bodies, and venereal proctitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- For most patients, treatment is nonsurgical and includes hot sitz baths and good hygiene, with the addition of bulk laxatives (psyllium seed compounds or gentle stool softeners) after the acute phase has subsided.
- Thrombosed external hemorrhoids require excision of the clots for relief. The thrombosed vein should be unroofed by an elliptical incision that allows evacuation of the multiloculated clots (see Fig. 47.2). Initial conservative treatment may be

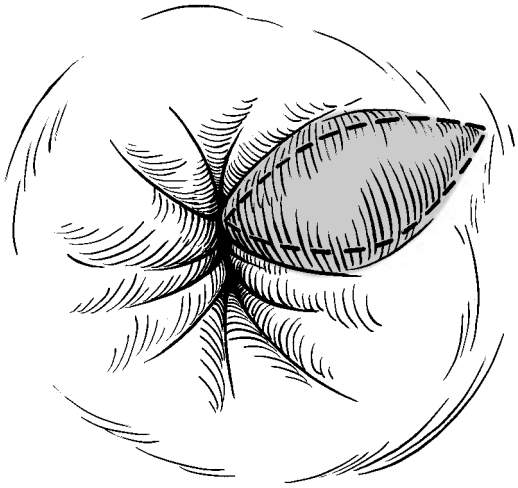

FIG. 47-2 Elliptical excision of thrombosed external hemorrhoid. (*From Goldberg SM et al: Essentials of Anorectal Surgery. Philadelphia, Lippincott, 1980, with permission.*)

tried for less severe cases that have been thrombosed for less than 48 h.
- Surgical referral and intervention are indicated for continued bleeding, intractable pain, incarceration, or strangulation. Rare complications of surgical repair include acute thrombosis and, for immunocompromised patients, potential pelvic sepsis.

CRYPTITIS

EPIDEMIOLOGY

- Cryptitis is associated with repetitive sphincter trauma, either secondary to spasm, recurrent diarrhea, or passage of large, hard stools.

PATHOPHYSIOLOGY

- The pleated columns of Morgagni, proximal to the dentate line, are connected at the base by small flaps of mucosa, the anal crypts, which are formed by the puckering action of the sphincter muscles and have the potential to become infected (cryptitis).

CLINICAL FEATURES

- Anal pain and itching, with or without bleeding, are the usual symptoms.
- The posterior crypts are most commonly involved and are tender, swollen, and nodular.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is confirmed by palpation or visualization by anoscopy.
- (See hemorrhoid section for differential of anal pain.) Cryptitis may coexist and lead to the development of fissures, fistulas, and abscess formation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Bulk laxatives, dietary fiber, hot sitz baths, and warm rectal irrigations will enhance healing. Surgical excision may be necessary in refractory cases.

FISSURE IN ANO

EPIDEMIOLOGY

- Anal fissures are the most common cause of painful rectal bleeding.
- An atypical anal fissure not located in the midline should alert the physician to other potentially life-threatening causes, including Crohn's disease, ulcerative colitis, carcinomas, lymphomas, syphilis, and tuberculosis.

PATHOPHYSIOLOGY

- Fissure in ano is the result of a linear tear from the dentate line through the sensitive anodermal tissue of the anal canal.

CLINICAL FEATURES

- Anal fissures most commonly occur in the posterior midline and are associated with severe, tearing pain during and immediately following defecation. In contrast to other anorectal disorders, the discomfort invariably subsides between bowel movements.
- Rectal examination may be limited secondary to severe pain; however, a characteristic sentinel pile, the result of swollen and hypertrophied papillae, may be visualized externally.

DIAGNOSIS AND DIFFERENTIAL

- (See hemorrhoid section for differential of anal pain.) Abscess and stricture formation may result from prolonged and severe fissure in ano.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Hot sitz baths and local analgesic and/or hydrocortisone-containing ointments will provide symptomatic relief and alleviate sphincter spasm.

ANORECTAL ABSCESSES

EPIDEMIOLOGY

- Abscess may develop from prolonged or severe fissure in ano. Specific diseases associated with

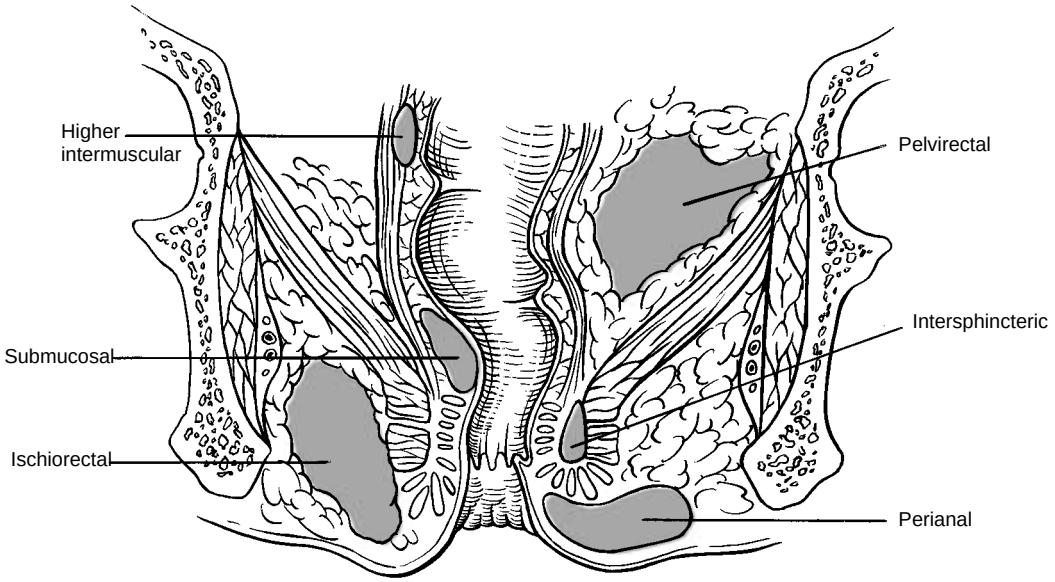

FIG. 47-3 Anatomic classification of common anorectal abscesses.

development of fistulous abscesses include gonococcal proctitis, carcinomas, Hodgkin's lymphoma, Crohn's disease, and tuberculosis.
- The most common abscess is the perianal abscess (see Fig. 47.3).

PATHOPHYSIOLOGY

- Abscesses originate in the anal crypts with gland obstruction and spread to involve the perianal, intersphincteric, ischiorectal, deep perianal, or supralevator (pelvirectal) spaces.

CLINICAL FEATURES

- The perianal abscess occurs midline posteriorly and may be palpated as a superficial, tender mass with or without fluctuance.
- A dull, aching, throbbing pain persists between bowel movements, but also increases with defecation. Fever and leukocytosis may be present.

DIAGNOSIS AND DIFFERENTIAL

- (See hemorrhoid section for differential of anal pain.) Deeper perirectal abscesses may be difficult to detect on physical exam only. Endorectal ultrasonography[2] may be useful to confirm diagnosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Most abscesses must be drained in the operating room by a surgeon, including all perirectal abscesses. Only simple perianal abscesses may be drained in the emergency department (ED); however, caution is still advised.
- Once a simple abscess has been adequately drained, antibiotics only need to be considered for patients whose immune system may be compromised.

PILONIDAL SINUS

EPIDEMIOLOGY

- Most commonly occurs before the fourth decade of life.
- Carcinoma is a rare complication of recurrent pilonidal sinus disease occurring more frequently in men.

PATHOPHYSIOLOGY

- A pilonidal sinus or cyst is formed by a classically chronic and recurring foreign-body granuloma reaction to an ingrown hair.

CLINICAL FEATURES

- A pilonidal sinus or cyst will occur in the midline, upper part of the natal cleft overlying the lower sacrum, and coccyx, causing pain, redness, and swelling.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Acute abscesses should be incised, drained, and packed in the ED.
- Once a simple abscess without complicating cellulitis has been adequately drained, antibiotics only need to be considered for patients whose immune system may be compromised.
- In order to prevent recurrence, definitive surgical excision of the entire pilonidal sinus system must be performed at least six weeks after all evidence of infection has resolved.

FISTULA IN ANO

EPIDEMIOLOGY

- A fistula most commonly results as a complication of a perianal or ischiorectal abscess; however, fistulas may be seen in association with ulcerative colitis, Crohn's disease, or tuberculosis.

PATHOPHYSIOLOGY

- A fistula is an abnormal tract connecting the anal canal with the skin.
- The Goodsall Rule purports that anterior-opening fistulas follow a direct course to the anal canal; however, posterior-opening fistulas may course through a more unpredictable and circuitous path.

CLINICAL FEATURES

- Malodorous and bloody discharge persists as long as the fistula remains open. Most cases involve recurrent blockages of the path, resulting in chronic repetitive abscess formation and subsequent spontaneous drainage.

DIAGNOSIS AND DIFFERENTIAL

- Abscess may be the presenting sign for fistula in ano.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Surgical excision is the definitive treatment.

RECTAL PROLAPSE/PROCIDENTIA

EPIDEMIOLOGY

- Prolapse of the rectal mucosa only is most commonly seen in children under the age of 2; however, it is also associated with third- and fourth-degree hemorrhoids in adults.
- Complete rectal prolapse (procidentia) occurs at the extremes of age and is most common in elderly women, with a higher incidence noted among women who have undergone hysterectomy.

PATHOPHYSIOLOGY

- The three classes of rectal prolapse are based upon anatomic differences: (a) prolapse of the rectal mucosa only; (b) prolapse of all three layers of the rectum; (c) intussusception or telescoping of the upper rectum through the lower rectum.
- The second and third classes of prolapse are due to both a laxity in the pelvic floor and a weakening of the sphincter musculature.

CLINICAL FEATURES

- Clinical features include the detection of a protruding mass, blood-stained mucus anal discharge, and fecal incontinence.

DIAGNOSIS AND DIFFERENTIAL

- The prolapsed rectum may be mistaken for hemorrhoids. A rectal tumor must be ruled out in adults.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Reduction may be accomplished after appropriate analgesia and sedation. If the tissue is ischemic, there is high risk of perforation and sepsis.
- Surgical correction is indicated; however, the patient may be discharged on stool softeners and

referred for outpatient proctosigmoidoscopy after successful reduction in the ED.

ANORECTAL TUMORS

EPIDEMIOLOGY

- The most common (80 percent) and most aggressive anorectal tumor is the anal canal tumor, located proximal to the dentate line and including the transitional zone of epithelium. Neoplasms that occur in this group include adenocarcinoma, malignant melanoma, and Kaposi's sarcoma.

CLINICAL FEATURES

- Patients present with nonspecific symptoms including sensation of a mass, pruritus, pain, and blood on the stool. Constipation, anorexia, and weight loss, narrowing of the stool caliber, and tenesmus eventually develop.
- An anal margin neoplasm will frequently present as an ulcer that fails to heal in a timely manner.

DIAGNOSIS AND DIFFERENTIAL

- Tumors may be misdiagnosed as hemorrhoids. Complications of anorectal tumors include rectal prolapse, prolonged blood loss, perirectal abscesses, or fistulas.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Referral for proctoscopic or sigmoidoscopic examination and biopsy is mandatory.

RECTAL FOREIGN BODIES

CLINICAL FEATURES

- The most common location for a rectal foreign body is in the ampulla.
- The most common complication of a rectal foreign body is perforation, which can result in overwhelming sepsis.

DIAGNOSIS AND DIFFERENTIAL

- A radiograph must be obtained to review the position, shape, and number of foreign bodies and to exclude the presence of free air due to rectal perforation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Most foreign bodies are low in the rectum and may be removed in the ED after local anesthesia to obtain sphincter relaxation.
- A surgeon or gastroenterologist should be consulted in cases with high risk of perforation or anticipated difficulty of removal. A broad-spectrum antibiotic should be administered.

PRURITUS ANI

EPIDEMIOLOGY

- Pruritus most commonly occurs during the fifth and sixth decades of life, primarily affecting men.
- Secondary pruritus ani may be due to anorectal disease, infection, diet, irritants, dermatologic conditions, or systemic diseases.

CLINICAL FEATURES

- Chronic pruritus ani may result in a thickened, depigmented appearance of the perianal skin.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Referral to a proctologist and/or dermatologist is usually necessary.
- Symptomatic treatments include sitz baths, zinc oxide ointment, and 1% hydrocortisone cream.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 78, "Anorectal Disorders," by James K. Bouzoukis.

48 VOMITING, DIARRHEA, AND CONSTIPATION

David M. Cline

VOMITING AND DIARRHEA

EPIDEMIOLOGY

- In the 1990s diarrhea accounted for less than 0.5 percent of all deaths in the United States.[1] Most diarrheal deaths occur in the elderly and the young.[1,2]
- Diarrhea is the second most common reason for work absenteeism and is estimated to cost $608 million in lost productivity per year.[3,4]
- From 1988 to 1992, a total of 2423 outbreaks of food-borne diseases in the United States were reported to the Centers for Disease Control and Prevention (CDC): 77,373 persons developed predominately diarrheal illness.[5] Most food-borne diseases are undiagnosed or unreported.
- The epidemiology of food-borne illnesses has evolved as new causative pathogens—such as *Escherichia coli* 0157:H7,[6] enteroinvasive *Klebsiella pneumoniae,*[6] and *Cyclospora cayetanensis*[7]—have been recognized. The expanding role of pathogens such as *Campylobacter jejuni, Listeria monocytogenes,* and *Yersinia enterocolitica,* previously unrecognized as causes of food-borne illness, has brought them to the forefront.[7]
- Nearly 80 percent of food-borne outbreaks in the United States from 1988 to 1992 occurred in cafeterias, restaurants, or delicatessens.[5]
- The most common pathogens causing food-borne illnesses are *Salmonella, Campylobacter, E. coli* 0157, and the Norwalk viruses.[8] Viral gastroenteritis—caused by the Norwalk viruses, rotaviruses, enteric adenoviruses, astroviruses, and calciviruses—accounts for the majority of cases of infectious diarrhea in the emergency department.

PATHOPHYSIOLOGY

- Vomiting is a complex, highly coordinated activity involving the gastrointestinal tract, the central nervous system, and the vestibular system.
- Three stages of vomiting have been described: nausea, retching, and emesis.[9] With nausea come hypersalivation and tachycardia. Retching occurs when the pylorus contracts and the fundus relaxes, thereby moving food to the gastric cardia. Finally, emesis occurs when the powerful abdominal muscles contract simultaneously and thus eject food or gastric secretions from the stomach.
- There are four basic mechanisms of diarrhea: increased intestinal secretion, decreased intestinal absorption, increased osmotic load, and abnormal intestinal motility.
- At a cellular level, intestinal absorption occurs through the villi, while secretion occurs though the crypts. Often, in diarrheal states, enterotoxins, inflammation, or ischemia damages the intestinal villi preferentially. As a result, diarrhea occurs because of diminished absorption by the intestinal villi and unopposed crypt secretion (crypts are more resilient after injury).[10]
- Direct invasion of the mucosal epithelial cells occurs with many food-borne pathogens, such as *Shigella, Salmonella,* enteroinvasive *E. coli, Campylobacter,* and *Vibrio parahaemolyticus.*[11] Intracellular multiplication of these organisms is followed by epithelial cell death.
- *Cytotoxins*—such as the Shiga toxin of *Shigella dysenteriae* or Shiga-like toxins produced by enterohemorrhagic *E. coli* O157:H7, enteropathogenic *E. coli,* and *V. parahaemolyticus*—also cause cellular membrane disruption and cell lysis.[11]
- *Vibrio cholera* and enterotoxic *E. coli* produce protein toxins that alter fluid and electrolyte transfer across epithelial cell membranes and produce large volumes of fluid that exceed the absorptive capacity of the colon. The resultant excessive diarrhea can lead to rapid dehydration.[11]

CLINICAL FEATURES

- Vomiting with blood can represent gastritis, peptic ulcer disease, or carcinoma. However, aggressive nonbloody vomiting followed by hematemesis is more consistent with a Mallory-Weiss tear.
- The presence of bile rules out gastric outlet obstruction, as from pyloric stenosis or strictures.
- An associated symptom such as fever would direct one to an infectious or inflammatory cause.
- Radiation of the pain to the chest suggests myocardial infarction or pneumonia.
- Radiation to the back can be seen with aortic aneurysm or dissection, pancreatitis, pyelonephritis, or renal colic.
- Headache with vomiting suggests increased intracranial pressure, as with subarachnoid hemorrhage or head injury.
- Vomiting in a pregnant patient is consistent with

hyperemesis gravidarum in the first trimester, but in the third trimester it can represent preeclampsia if accompanied by hypertension.
- Associated medical conditions are also useful in discerning the cause of vomiting: insulin use suggests ketoacidosis, peripheral vascular disease suggests mesenteric ischemia, previous surgery suggests intestinal obstruction, and medication use (e.g., lithium or digoxin) suggests toxicity.
- The physical examination in a vomiting patient includes a careful assessment of the gastrointestinal, pelvic, and genitourinary systems. In addition, assessment of hydration status is important.
- Other clues to specific causes for vomiting may come from the dermal exam (e.g. hyperpigmentation with Addison's disease) or pulmonary examination (e.g., clues of pneumonia).
- By definition, diarrhea represents a daily stool output of >200 g, but generally it refers to any increase in frequency or liquidity.[12] Other important historical factors include duration of illness and presence of blood.
- Acute diarrhea of less than 2 to 3 weeks' duration is more likely to represent a serious cause, such as infection, ischemia, intoxication, or inflammation.
- Associated factors—such as fever, pain, or type of food ingested—may help in the diagnosis of infectious gastroenteritis.
- Neurologic symptoms can be seen in certain diarrheal illnesses, such as seizure with shigellosis or theophylline toxicity or paresthesias with ciguatoxin.
- Details about the host can also better define the diagnosis. Malabsorption from pancreatic insufficiency or HIV-related bowel disorders need not be considered in a healthy host.
- History of foods ingested—such as meat, dairy products, seafood, or unpasteurized products—may isolate the vector and narrow the differential diagnosis for infectious diarrhea considerably (e.g., oysters suggest *Vibrio;* rice suggests *Bacillus cereus;* eggs suggest *Salmonella;* and meat suggests *Campylobacter, Staphylococcus, Yersinia, E. coli,* or *Clostridium*).
- Certain medications—particularly antibiotics, colchicine, lithium, and laxatives—can all contribute to diarrhea.
- Travel may predispose the patient to *E. coli* or *Giardia.* Social history—such as sexual preference, drug use, and occupation—may suggest such diagnoses as HIV-related illness or organophosphate poisoning.
- The physical examination usually concentrates initially on assessment of fluid status.
- Abdominal examination can narrow the differential diagnosis as well as reveal the need for surgical intervention. Even appendicitis can present with diarrhea in up to 20 percent of cases.
- Rectal examination can rule out impaction or presence of blood, the latter suggesting inflammation, infection, or mesenteric ischemia.

DIAGNOSIS AND DIFFERENTIAL

- A mnemonic to prompt the physician's recall of disease groupings causing vomiting and diarrhea is GASTROENTERITIS: gastrointestinal disease, appendicitis or aorta, specific disease (e.g., glaucoma), trauma, medications (Rx), obstetric-gynecologic disorders, endocrine disorders, neurologic disease, toxicology, environmental disorders, renal disease, infection, tumors, ischemia, and supratentorial.
- Etiologic agents for food-borne diseases are listed in Table 48-1.
- All women of childbearing age warrant a pregnancy test.
- In vomiting associated with abdominal pain, liver function tests, urinalysis, and lipase or amylase determinations may be useful.
- Electrolyte determinations and renal function tests are usually of benefit only in patients with severe dehydration or prolonged vomiting. In addition, they may confirm addisonian crisis with hyperkalemia and hyponatremia.
- The electrocardiogram and chest radiograph can be reserved for patients with suspected ischemia or pulmonary infection.
- An acute abdominal series can be used to confirm the presence of obstruction.
- The most specific tests in diarrheal illness all involve examination of the stool in the laboratory. Wright's stain for fecal leukocytes has an 82 percent sensitivity and 83 percent specificity for the presence of invasive bacterial pathogens.[13] Because of its poor sensitivity and the safety of antibiotics, even in noninvasive diarrhea, this test has lost its popularity.
- Instead, fecal blood testing may provide similar information at lower cost.
- A more expensive proposition, stool culture, also has poor sensitivity. It is therefore reserved for those patients with immunocompromise or persistent diarrhea or toxic patients with severe dehydration. In addition, it may be useful for patients involved in public health–sensitive occupations.
- In patients with chronic persistent diarrhea, an

TABLE 48-1 Etiologic Agents for Food-Borne Diseases and Usual Incubation Periods

1–6 h
Norwalk viruses
Astrovirus, calcivirus
Staphylococcus aureus
Bacillus cerus vomiting toxin
Ciguatoxin
Scombroid toxins
Paralytic or neurotoxic shellfish poisoning
Puffer fish, tetrodotoxin
Heavy metals
Monosodium glutamates
Short-acting mushroom toxins
6–24 h
Bacillus cereus diarrheal toxin
Clostridium perfringens
Vibrio parahaemolyticus
Long-acting mushroom toxins
24–48 h
Nontyphoidal *Salmonella*
Enterotoxigenic (ETEC)
Clostridium botulinum
Trichinella spp. intestinal phase
2–6 days
Shigella
Campylobacter
Escherichia coli O157:H7
Vibrio cholerae
Streptococcus group A
Yersinia enterocolitica
6–14 days
Cryptosporidium parvum
Salmonella typhi
Cyclospora
Giardia lamblia
>14 days
Hepatitis A
Brucella
Listeria monocytogenes invasive disease
Trichinella spp. systemic phase

SOURCE: From the CDC.[5]

examination for ova and parasites may be useful to rule out *Giardia* or *Cryptosporidium.*

- Although not extremely sensitive, assay for *Clostridium difficile* toxin may be useful in ill patients with antibiotic-associated diarrhea.
- Because of the low sensitivity and delay in results, laboratory testing in routine diarrheal cases is not indicated.
- In extremely dehydrated or toxic patients, electrolyte determinations and renal function tests may be useful.
- In an infant with bloody diarrhea, the presence of renal failure and anemia suggests hemolytic uremic syndrome, usually due to *E. coli* O157:H7.
- If toxicity is suspected, tests for levels for theophylline, lithium, or heavy metals will aid in the diagnosis.
- Radiographs are reserved for ruling out obstruction or pneumonia, particularly *Legionella.*

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Replacement of fluids can be intravenous (bolus 500 mL IV in adults, 10 to 20 mL/kg in children) with normal saline solution in seriously ill patients. Mildly dehydrated patients may tolerate an oral rehydrating solution containing sodium (at least 45 meq/L in children) as well as glucose to enhance fluid absorption. The World Health Organization advocates a mixture of 1 cup of orange juice, 4 tsp sugar, 1 tsp baking powder, and 3/4 tsp salt in 1 L of boiled water.[10] The goal is 50 to 100 mL/kg over the first 4 h.
- Nutritional supplementation should be started as soon as nausea and vomiting subside. Patients can quickly advance from clear liquids to solids, such as rice and bread. Patients may benefit from avoiding caffeine and sorbitol-containing products.
- Antibiotics are recommended for adult patients with severe or prolonged diarrhea.[14–16] In addition, they are indicated for travelers from tropical or third world countries. Although single-dose fluoroquinolones show some effectiveness, these antibiotics are usually given for 3 to 5 days: ciprofloxacin 500 mg PO bid, norfloxacin 400 mg PO bid, or ofloxacin 300 mg PO bid. Although inferior, trimethoprim/sulfamethoxazole (TMP/SMX), TMP 10 mg/SMX 50 mg/kg/d (maximum dose TMP 160 mg:SMX 800 mg) is indicated for children or nursing mothers if antibiotics are truly necessary. It should be noted that antibiotics are of questionable value in infectious diarrhea from *E. coli* O157:H7.
- Metronidazole 15 mg/kg PO divided tid for 5 days (maximum 1000 mg/d) is indicated for *C. difficile, Giardia,* or *Entamoeba* (treat for 10 days) infection. Antibiotics are especially indicated in patients or workers in the food industry or institutional settings, such as day care centers and nursing homes.
- Antidiarrheal agents, especially in combination with antibiotics, have been shown to shorten the course of diarrhea.[15,16] Loperamide is given 4 mg PO initially and then 2 mg PO after each diarrheal stool, maximum of 16 mg/d (for children over 2 years, 0.8 mg/kg/d is given, with one-third of the dose given initially and one-third of the dose after the next two diarrheal stools).
- Antiemetic agents are useful in actively vomiting

TABLE 48-2 Differential Diagnosis of Constipation

ACUTE OR SUBACUTE
Gastrointestinal: obstructing cancer, volvulus, stricture, hernia, adhesion, pelvic or abdominal masses
Medicinal: addition of new medicine (e.g., antipsychotic, anticholinergic, narcotic analgesic, antacids)
Environmental: change in defecation regimen (e.g., forced to use bedpan)
Exercise and diet: decrease in level of exercise, fiber intake, fluid intake
CHRONIC
Gastrointestinal: slowly growing tumor, colonic dysmotility, anal pathology
Medicinal: chronic laxative abuse, antipsychotics, anticholinergics, narcotic analgesics
Neurologic: neuropathy, Parkinson's disease, paraplegia, cerebral palsy
Endocrine: diabetes, hypothyroidism, hyperparathyroidism
Rheumatologic: scleroderma
Toxicologic: lead poisoning

patients with dehydration. Traditionally, promethazine 25 mg (12.5 mg in children over 2 years) intramuscularly (IM), IV, or rectally (PR) every 6 h is prescribed. Prochlorperazine 10 mg IV or IM or 25 mg PR or PO is particularly useful if vomiting is accompanied by headache. Finally, metochlorpramide 10 to 20 mg (1 mg/kg) is very useful for nausea and can be given in pregnancy (category B).
- Admission is dependent on toxic appearance, response to therapy, and preexistent medical conditions.

CONSTIPATION

EPIDEMIOLOGY

- Constipation is the most common digestive complaint in the United States and accounts for 2.5 million physician visits per year.[17]
- There is an age-related increase in the incidence of constipation, with 30 to 40 percent of adult patients over age 65 citing constipation as a problem.[18,19]

PATHOPHYSIOLOGY

- Fluid intake, fiber intake, exercise, medications, and medical condition affect gut motility.
- See Table 48-2 for a listing of causes of constipation.

CLINICAL FEATURES

- The usual definition for this disorder is the presence of hard stools that are difficult to pass.
- Acute onset implies obstruction until proven otherwise. Chronic constipation is less ominous and can be managed on an outpatient basis.
- Associated symptoms, such as vomiting and inability to pass flatus, confirm obstruction.
- Associated illnesses can help disclose the underlying diagnosis: cold intolerance (hypothyroidism), diverticulitis (inflammatory stricture), or nephrolithiasis (hyperparathyroidism).
- Physical examination should focus on detection of hernias or abdominal masses.
- Rectal examination will detect masses, such as fecal impaction, anal fissures, or fecal blood. The latter, accompanied by weight loss or decreasing stool caliber, may confirm colon carcinoma.
- The presence of ascites in postmenopausal women raises suspicion of ovarian or uterine carcinoma.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

- Directed testing in acute constipation, based on suspicion, can include a complete blood count (to rule out anemia), a thyroid panel (to rule out hypothyroidism), and electrolyte determinations (to rule out hypokalemia or hypercalcemia).
- Flat and erect abdominal films may be useful in confirming obstruction or assessing stool burden.
- Barium enema is diagnostic for such disorders as intussusception, Hirschsprung's disease, and volvulus.
- Ultrasound studies, particularly in pediatric cases, can discern the cause of obstruction, including pyloric stenosis and intussusception.
- The differential for constipation is listed in Table 48-2.
- Chronic constipation is a functional disorder that can be worked up on an outpatient basis. Nevertheless, complications of chronic constipation, such as fecal impaction and intestinal pseudoobstruction, will require either manual or colonoscopic intervention.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The most important prescription for functional constipation is a dietary and exercise regimen that includes fluids (1.5 L/d), fiber (10 g/d), and exercise. Fiber in the form of bran (1 cup/d) or psyllium

(Metamucil at 1 tsp tid) increases stool volume and gut motility.
- Medications can provide temporary relief for this chronic problem. Stimulants can be either given PO, as with anthraquinones (e.g., Peri-Colace 1 to 2 tablets PO at bedtime) or PR, as with bisacodyl (Dulcolax 10 mg PR tid in adults or children). In the absence of renal failure, magnesium (e.g., milk of magnesia 15 to 30 mL daily bid or magnesium citrate 200 mL once) is useful. In children mineral oil (15 to 30 mL per year of age daily or bid PO for up to 3 d) has been advocated.
- Enemas of soapsuds (1500 mL PR) or phosphate (e.g. Fleet's I U PR, 1 oz/10 kg in children) are generally reserved for severe cases or after fecal disimpaction.
- Fecal impaction should be removed manually using local anesthetic lubricant. In female patients, transvaginal pressure with the other hand may be helpful. An enema or suppositories to complete evacuation can follow.
- Early follow-up is indicated in patients with recent severe constipation or systemic symptoms, such as weight loss, anemia, or change in stool caliber. Patients with organic constipation from obstruction require hospitalization and surgical evaluation.
- Intestinal pseudoobstruction and sigmoid volvulus can sometimes be corrected colonoscopically.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 79, ''Vomiting, Diarrhea, and Constipation,'' by Annie Tewel Sadosty and Brian J. Browne.

49 JAUNDICE, HEPATIC DISORDERS, AND HEPATIC FAILURE

David M. Cline

JAUNDICE

PATHOPHYSIOLOGY

- Jaundice, a yellowish discoloration of the skin, sclerae, and mucous membranes, results from hyperbilirubinemia (breakdown of hemoglobin) and thus the deposition of bile pigments.[1]

TABLE 49-1 Causes of Jaundice

UNCONJUGATED	CONJUGATED
Hemolytic anemia	Intrahepatic
Hemoglobinopathy	Infectious
Transfusion reaction	Viral hepatitis
Gilbert disease	Leptospirosis
Crigler-Najjar syndrome	Infectious mononucleosis
Premature neonates	Toxins (drugs/chemicals)
Congestive heart failure	Familial
Sepsis	Rotor syndrome
	Dubin-Johnson syndrome
	Alcoholic liver disease
	Other
	Sarcoidosis
	Lymphoma
	Liver metastasis
	Cirrhosis
	Pregnancy
	Amyloidosis
	Extraheptic
	Gallstones
	Pancreatic tumors/cysts
	Cholangiosarcoma
	Bile duct stricture
	Sclerosing cholangitis

- It has many etiologies (Table 49-1).
- Hyperbilirubinemia can be divided into two types. The unconjugated form results from increased bilirubin production or a liver defect in its uptake or conjugation. The conjugated form occurs in the setting of intra- or extrahepatic cholestasis, resulting in decreased excretion of conjugated bilirubin.
- The total serum bilirubin should be elevated in a jaundiced patient. Clinically, jaundice usually becomes noticeable at a serum bilirubin level of 2.0 to 2.5 mg/dL and is often first seen in the sclera.
- An indirect fraction of serum bilirubin of 85 percent or higher is consistent with the uncojugated type, whereas a direct fraction of 30 percent or above indicates the conjugated form.
- Conjugated bilirubin is water-soluble and is detectable in the urine even with low serum levels.

CLINICAL FEATURES

- Jaundice is a symptom with a myriad of possible underlying causes.[2]
- Sudden onset of jaundice in a previously healthy young person together with a prodrome of fever, malaise, myalgias, and a tender, enlarged liver points to hepatitis (probably viral) as a likely cause.
- Heavy ethanol use suggests alcoholic hepatitis. In the setting of alcoholic liver disease and cirrhosis, jaundice usually develops gradually (discussed later).
- A family history of jaundice or a history of recurrent mild jaundice that spontaneously resolves usually accompanies inherited causes of jaundice, such as Gilbert syndrome.
- Cholecystitis in itself may not cause jaundice unless acute biliary obstruction is present, as with a gallstone retained in the common bile duct.
- Painless jaundice in an older patient classically suggests pancreatic or hepatobiliary malignancy.
- Patients with a known prior malignancy and a hard, nodular liver accompanied by jaundice are likely to be found to have liver metastases.
- Biliary tract scarring or strictures must always be suspected as a cause of jaundice in patients with a prior history of biliary tract surgery, pancreatitis, cholangitis, or inflammatory bowel disease.
- Hepatomegaly with jaundice, accompanied by pedal edema, jugular venous distention, and a gallop rhythm, suggests chronic heart failure.

DIAGNOSIS AND DIFFERENTIAL

- Initial laboratory tests that should be obtained in the workup of a jaundiced patient include serum bilirubin level (both total and direct fraction—indirect fraction can be deduced by simple subtraction), serum aminotransferases and alkaline phosphatase levels, urinalysis to check for bilirubin and urobilinogen, and a complete blood count (CBC).
- Additional laboratory tests may be indicated based on the clinical setting [serum amylase and lipase levels, prothrombin time (PT), electrolytes and glucose levels, blood urea nitrogen (BUN) and creatinine levels, viral hepatitis panels, drug levels, and pregnancy test].
- With normal liver enzyme levels, the jaundice is more likely to be caused by sepsis or systemic infection, inborn errors of metabolism, or pregnancy rather than by primary hepatic disease.
- With abnormally elevated liver enzymes, the pattern of abnormalities may suggest the etiology. If predominant, aminotransferase elevation suggests hepatocellular disease such as viral or toxic hepatitis or cirrhosis, whereas markedly elevated alkaline phosphatase levels (two to three times normal levels) and GGT points to intra- or extrahepatic obstruction (gallstones, stricture, and malignancy).
- A Coombs' test and hemoglobin electrophoresis may be useful if anemia is present, along with

normal liver aminotransferase levels (hemolysis and hemoglobinopathy).
- If clinical features and initial laboratory results reveal conjugated hyperbilirubinemia, ultrasound studies of the biliary tract, liver, and pancreas should be performed to rule out gallstones, dilated extrahepatic biliary ducts, or mass/tumor in the liver, pancreas, and portal region.
- A computed tomography scan may also be considered but is often more costly than ultrasound and not as sensitive for detection of gallstones in the gallbladder.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Jaundice by itself is not an adequate justification for hospital admission. In some situations discharge from the emergency department (ED) pending further outpatient workup may be appropriate if a patient is hemodynamically stable with new onset jaundice and has no evidence of liver failure or acute biliary obstruction and if appropriate laboratory studies have been ordered, timely follow-up is available, and the patient is reliable and has adequate social support.
- If extrahepatic biliary obstruction is suspected, surgical consultation should be obtained in the ED. For other possible admission indicators, see the remaining sections of this chapter as well as Chap. 50.

HEPATITIS

EPIDEMIOLOGY

- Chronic liver disease is currently the tenth leading cause of death among adults in the United States and accounts for 25,000 deaths yearly, or 1 percent of all deaths. The majority of end-stage liver disease (approximately 50 percent) is related to alcohol abuse. However, in recent decades, an increasing number of cases can be attributed to chronic viral hepatitis.[3–5]
- Currently, hepatitis C virus (HCV) infection is the most common of all blood-borne infections in the United States, with approximately 28,000 to 180,000 new cases yearly. An estimated 3.9 million (1.8 percent) Americans have been infected.
- HCV infection is often subclinical, with symptoms of chronic liver disease and cirrhosis delayed 1 to 2 decades. It is anticipated that the number of cases of chronic liver disease related to HCV will increase sharply in the next 10 to 20 years.[6,7]
- Effective vaccination against hepatitis B virus (HBV) has led to a decline in the prevalence of related disease in the general population. Still, there are an estimated 140,000 to 320,000 cases of HBV infection yearly, with 140 to 320 deaths due to acute infections.[8]
- The hepatitis D virus (HDV) is uncommon and described as a defective agent because infection depends on concomitant or preexisting chronic infection by HBV. This variety of infection is most commonly associated with intravenous drug use.[4,5]
- Hepatitis A virus (HAV) is commonly encountered by Americans, with 33 percent of the population having acquired immunity secondary to exposure.[4,9]

PATHOPHYSIOLOGY

- Common to essentially all causes of chronic liver disease is ongoing hepatocellular injury and death, with the progressive disruption of the functional microanatomy of the liver. Eventually, the metabolic function of the liver becomes both compromised and isolated, resulting in nutritional deficiencies, bleeding diatheses, and the accumulation of toxic metabolic wastes.
- Risk factors for viral hepatitis include male homosexuality, hemodialysis, intravenous drug abuse, ingestion of raw seafood, blood product transfusion, tattoos or body piercing, needle punctures, foreign travel, or close contact with an infected source patient.

CLINICAL FEATURES

- Viral hepatitis may range in severity from asymptomatic infection to fulminant hepatic failure to chronic cirrhosis.
- Symptomatic patients usually report a prodrome of sudden or insidious onset including anorexia, nausea, emesis, fatigue, malaise, and altered taste.
- Low-grade fever—accompanied by pharyngitis, coryza, and headache—may confuse the picture and lead to an initial misdiagnosis of upper respiratory infection or flulike illness.
- A few days of generalized pruritus and dark urine may precede the onset of gastrointestinal (GI) symptoms and jaundice. Malaise usually persists while the other prodromal symptoms resolve. Right-upper-quadrant pain with an enlarged, tender liver and splenomegaly may be found.

Many patients do not become clinically jaundiced, and most will recover gradually over the ensuing 3 to 4 months.
- Rarely, fulminant hepatic failure develops with a clinical picture of encephalopathy, coagulopathy, and rapidly worsening jaundice.
- Chronic persistent infection (usually with hepatitis B or C) can lead to the development of cirrhosis, with gradual jaundice, ascites, peripheral edema, and liver failure over a period of 10 to 20 years.
- Hepatitis A virus (HAV) is transmitted predominantly by the fecal-oral route and is commonly seen in Americans. Children and adolescents are more commonly affected, but often subclinically, whereas most adults are symptomatic, with a longer, more severe course. Symptom onset is often more abrupt than with other viruses. Epidemic outbreaks have been seen in children at day care centers, institutionalized patients, and patients exposed to a common-source case via contaminated food or water.
- Hepatitis B virus (HBV) is acquired primarily via a percutaneous exposure to infected blood or body fluids. Most cases are subclinical, without jaundice. Often symptom onset is insidious and in 5 to 10 percent of cases is preceded by a serum sickness–like syndrome with polyarthritis, proteinuria, and angioneurotic edema. Symptomatic patients usually have a more severe and protracted course than those with HAV.
- Chronic HBV infection occurs in 6 to 10 percent of patients who may go on to develop cirrhosis, end-stage liver disease (ESLD), and hepatocellular carcinoma.
- HCV is the most common of all blood-borne infections in the United States and may be contracted via parenteral, sexual, or perinatal contact. Most patients remain asymptomatic or have milder symptoms than those with HBV or HAV. Unfortunately, chronic HCV infection occurs in 85 percent of patients, the majority of whom remain subclinically infected. Up to 70 percent of chronic HCV cases progress to the development of chronic liver disease (cirrhosis and ESLD) and are at increased risk for hepatocellular carcinoma.
- HDV can coinfect only in the setting of acute or chronic HBV infection. Acute HDV superinfection of an HBV carrier, often in the setting of intravenous drug abuse, frequently leads to fulminant liver failure with a high mortality rate.
- Hepatitis E virus (HEV) is a small RNA virus and has been seen in sporadic water-borne outbreaks in Asia, India, Africa, Mexico, and the former Soviet Union. Imported cases of HEV have been reported in the United States.
- Non-A, non-B hepatitis is caused by other hepatotrophic viruses, which have yet to be characterized (formerly, HCV was the predominant virus in this group) and are transmitted via blood exposure, causing infection and a course similar to HCV.

DIAGNOSIS AND DIFFERENTIAL

- Establishment of a diagnosis of viral hepatitis depends primarily on liver function abnormalities coupled with the clinical picture. Serum transaminases (GGT, AST, ALT) should be checked—elevations are suggestive of hepatitis.[10,11]
- Values in the hundreds of units per liter are consistent with viral inflammation, but elevations into the thousands suggest hepatocellular necrosis, extensive liver injury, and more fulminant disease.
- In acute and chronic viral hepatitis, the AST:ALT ratio is usually <1 (whereas a ratio >2 is more suggestive of alcoholic hepatitis).
- Serum alkaline phosphatase level should also be determined—if elevated more than threefold above normal, cholestasis should be suspected. (A concurrently elevated GGT supports this.)
- Total serum bilirubin, along with its direct fraction (indirect fraction can be deduced by simple subtraction), may also be useful, since a conjugated (direct) fraction of 30 percent or higher is consistent with viral hepatitis.
- The magnitude of transaminase elevation is not a reliable marker of disease severity, but a persistent total bilirubin level >20 mg/dL or a PT prolonged by more than a few seconds indicates a poor prognosis (hence PT time should be checked).
- Serum electrolytes, BUN, and creatinine should be checked if there is clinical suspicion of volume depletion or electrolyte abnormalities.
- Abnormal mental status should prompt an immediate determination of serum glucose level, which may be low due to poor oral intake or hepatic failure. (Other causes of abnormal mental status—such as hypoxia, sepsis, intoxication, structural intracranial process, or encephalopathy—must also be considered.)
- A CBC may be useful, as an early transient neutropenia followed by a relative lymphocytosis with atypical forms is often seen with viral hepatitis. Anemia, if present, may be more suggestive of alcoholic hepatitis, decompensated cirrhosis, GI bleeding, or a hemolytic process.
- Serologic studies to determine the specific viral agent responsible may be ordered in the ED to facilitate the final diagnosis, but these results are

rarely immediately available and thus play no significant role in ED management.
- Important differential diagnoses include alcohol- or toxin-induced hepatitis, infectious mononucleosis, cholecystitis, ascending cholangitis, sarcoidosis, lymphoma, liver metastases, and pancreatic or biliary tumors.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Most patients can be successfully managed as outpatients with emphasis on rest, adequate oral intake, strict personal hygiene, and avoidance of hepatotoxins (ethanol and drugs). Follow-up arrangements should be made.
- Patients with any of the following should be admitted to the hospital: encephalopathy, PT prolonged by more than a few seconds, intractable vomiting, hypoglycemia, bilirubin >20 mg/dL, age >45 years, immunosuppression, or suspected toxin-induced hepatitis.
- Volume depletion and electrolyte imbalances should be corrected with intravenous crystalloid. Hypoglycemia should be initially treated with 1 ampule of 50% dextrose in water intravenously followed by the addition of dextrose to intravenous fluids and careful monitoring.
- Fulminant hepatic failure should warrant admission to the intensive care unit, with aggressive support of circulation and respiration, monitoring and treatment of increased intracranial pressure if present, correction of hypoglycemia and coagulopathy, administration of oral lactulose or neomycin, and a protein-restricted diet.[12,13] (See following section on treatment of cirrhosis.) Consultation with a hepatologist and liver transplant service are indicated.
- Glucocorticoid therapy has no value in acute viral hepatitis, even with fulminant hepatic failure, and should be avoided.

ALCOHOLIC LIVER DISEASE AND CIRRHOSIS

PATHOPHYSIOLOGY

- Three clinical syndromes best describe liver injury secondary to alcohol: hepatic steatosis (fatty liver), alcoholic hepatitis, and alcoholic cirrhosis. An enlarged, nontender or mildly tender liver from steatosis is usually seen in a relatively asymptomatic alcoholic patient.[14]
- The cirrhotic liver becomes increasingly resistant to blood flow. Portal hypertension results in splenomegaly and the development of gastroesophageal varices. Splenomegaly contributes to anemia and thrombocytopenia. Ascites develops secondary to portal hypertension and abnormalities in renal sodium and water excretion caused by diminished glomerular filtration rate (GFR) and elevations in both aldosterone and antidiuretic hormone.

CLINICAL FEATURES

- Alcoholic hepatitis is typically found in the chronic alcoholic who presents with a gradual onset of anorexia, nausea, fever, dark urine, jaundice, weight loss, abdominal pain, and generalized weakness.
- Physical exam reveals a tender, enlarged liver, low-grade fever, and icteric mucous membranes, sclera, or skin.
- Patients suffering from cirrhosis generally report a gradual deterioration in their health, with anorexia, muscle loss (often masked by edema or ascites), fatigue, nausea, emesis, diarrhea, and increasing abdominal girth (ascites). Low-grade intermittent or continuous fever may also be present, while hypothermia may be seen at end-stage disease.[3]
- Abdominal palpation may reveal a small, firm liver and possibly splenomegaly. Jaundice, pedal edema, ascites, and spider angiomata are also common.
- Hepatic encephalopathy, characterized by a fluctuating level of consciousness and confusion and possibly hyperreflexia, spasticity, and generalized seizures may also be present.
- Asterixis (''liver flap'') is characteristic but not specific for encephalopathy due to liver failure.
- Patients with cirrhosis often come to the ED because of worsening ascites or edema, complications such as GI or variceal bleeding (see Chap. 39, ''Gastrointestinal Bleeding''), encephalopathy, spontaneous bacterial peritonitis (abdominal pain), and various concurrent infections (urinary tract infection, pneumonia, etc.).

DIAGNOSIS AND DIFFERENTIAL

- Alcoholic hepatitis and cirrhosis may be diagnosed by their clinical features and laboratory findings. Laboratory studies that should be

checked include levels of serum transaminases (GGT, ALT, AST), serum alkaline phosphatase, total bilirubin (and its fractions), serum albumin, serum glucose and electrolytes, BUN, and creatinine, CBC, and PT.

- In the setting of alcoholic hepatitis, serum transaminases levels are usually elevated to a range of 2 to 10 times normal, with an AST:ALT ratio of >1.5 to 2.0 (AST production is stimulated by ethanol). With cirrhosis, transaminase levels are often only mildly elevated. Alkaline phosphatase and bilirubin levels are usually only mildly elevated with both alcoholic hepatitis and cirrhosis.
- Anemia, leukopenia, and thrombocytopenia are commonly seen in chronic ethanol abusers. If concurrent pancreatitis is suspected, serum lipase and amylase levels should be checked.
- Fever with or without leukocytosis in a chronic alcoholic warrants a chest x-ray to rule out pneumonia; cultures of blood, urine, and ascitic fluid; and a thorough search for other sources of sepsis (meningitis, cholecystitis, cellulitis, perirectal abscess, etc.).[15]
- Elevated serum ammonia level unfortunately does not correlate well with acute deterioration of liver function due to cirrhosis and, while it may be checked as a marker of encephalopathy, it cannot be used as an index of severity or response to treatment.
- Spontaneous bacterial peritonitis (SBP), the most common complication of cirrhotic ascites, should be suspected in any cirrhotic patient with fever, abdominal pain or tenderness, worsening ascites, subacute functional decline, or encephalopathy.[16] Other subtle clues to SBP include deteriorating renal function, hypothermia, and diarrhea.
- SBP may be confirmed through sampling of ascitic fluid by paracentesis, ideally under ultrasound guidance to minimize the risk of bowel puncture. Ascitic fluid should be tested for total protein and glucose levels, lactic (acid) dehydrogenase (LDH), Gram stain, and white blood cell count (WBC) with differential. A total WBC count >1000/mm^3 or polymorphonuclear (PMN) cell count >250/mm^3 is diagnostic for SBP. (A total WBC count >10,000/mm^3, total protein >1g/dL, glucose <50mg/dL, or elevated LDH points to the possibility of generalized peritonitis due to a local focus of infection—eg., cholecystitis or appendicitis.)
- Culture results from ascitic fluid are often negative, but placing 10 mL of ascitic fluid in a blood culture bottle may improve yield. Gram-negative Enterobacteriaceae (*E. coli, Klebsiella,* etc.) account for 63 percent of SBP, followed by the pneumococcus (15 percent) and the enterococcus (6 to 10 percent).
- Hepatorenal syndrome, a refractory form of acute renal failure that occurs in cirrhotic patients, may develop in the setting of sepsis, acute dehydration, overzealous diuresis, or high-volume paracentesis. The differential diagnosis includes other forms of hepatitis (drugs, toxins, etc.) as well as other causes of upper abdominal pain (cholecystitis, biliary colic, gastritis or peptic ulcer disease, pancreatitis, etc.).[17]
- Cirrhosis is often caused by ethanol or chronic viral hepatitis—uncommon causes include drugs or toxins, hemochromatosis, Wilson's disease, and primary (idiopathic) biliary cirrhosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

Alcoholic Hepatitis

- Hospital admission is required for all but the mildest of cases of alcoholic hepatitis.
- Fluid therapy with dextrose-containing intravenous fluids should be given, with the goal of maintaining adequate intravascular volume while avoiding fluid overload in the edematous or ascitic patient.
- Thiamine (100 mg) should always be given with initial intravenous fluids and dextrose. Vitamin supplements should also be given to any malnourished alcoholic.
- Correction of electrolyte abnormalities should be initiated (many alcoholic patients will require supplemental magnesium and potassium).
- Identified bacterial coinfections should be promptly treated with appropriate parenteral antibiotics and, pending culture results, broad-spectrum coverage should be initiated in any alcoholic with suspected sepsis.[18]

Cirrhosis and Liver Failure

- Abstinence from alcohol and other hepatotoxins (drugs, etc.) is essential for outpatient management. Adjunctive measures may include salt and water restriction, cautious diuretic use (spironolactone), protein-restricted diet, and therapeutic paracentesis for relief of abdominal distention.
- Emergency management often includes changing diuretic dosage, correction of fluid or electrolyte abnormalities, and blood transfusion for symptomatic anemia.

- New-onset or worsening encephalopathy warrants hospital admission. Management includes supplemental oxygen, support of perfusion, mechanical respiration as needed, and supplemental dextrose in intravenous fluids. Precipitating factors such as a coexisting infection, GI bleeding, or renal failure must be carefully investigated and aggressively treated.[19] Lactulose (30 mL) may be given orally, by nasogastric tube or by enema, up to three times a day until one or two soft stools per day are produced. Alternatively, neomycin may be given to help clear the gut of bacteria and nitrogenous products.[19]
- Cefotaxime 2 g intravenously followed by 1 to 2 g intravenously every 6 h is the current antibiotic regimen of choice for spontaneous bacterial peritonitis (alternatives include ticarcillin-clavulanate, piperacillin-tazobactam, ampicillin-sulbactam, or the quinolones).[18]
- Acute liver failure from any cause (with prolonged PT, hypoglycemia, coagulopathy, encephalopathy, marked jaundice, etc.) should warrant admission to the intensive care unit, aggressive treatment, and consultation with a hepatologist and transplant team if available.[12,13]
- Any cirrhotic patient whose clinical stability is in doubt should always be considered for admission. All patients with clearly decompensated cirrhosis, fever, hypothermia, or complications such as concurrent infection, SBP, GI bleeding, encephalopathy, and acute or worsening renal function should be admitted.

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50 CHOLECYSTITIS AND BILIARY COLIC

Nancy A. Wick

EPIDEMIOLOGY

- The classic patient with symptomatic gallstone disease is female, obese, and between 20 and 40 years old; however, all age groups are affected, especially those with diabetes and the elderly.[1]
- The four most common biliary tract emergencies caused by gallstones are biliary colic, cholecystitis, gallstone pancreatitis, and ascending cholangitis.
- Risk factors associated with gallstones and cholecystitis include increased age, female sex

and parity, obesity, familial tendency, oral contraceptives, clofibrate, Asian descent, chronic liver disease, sickle cell anemia, and hereditary spherocytosis.[2]

PATHOPHYSIOLOGY

- Gallstones are of three major types: cholesterol (70 percent), more frequently radiolucent; pigment (20 percent), more frequently radiopaque; and mixed (10 percent).
- Symptomatic cholelithiasis occurs when a stone migrates from the gallbladder into the biliary tract and causes obstruction, producing increased intraluminal pressure and distension of the hollow viscus, resulting in pain, nausea, and vomiting. Acute cholecystitis develops from persistent obstruction.
- Bacterial pathogens are present in 50 to 80 percent of patients with acute cholecystitis and include Enterobacteriaceae (70 percent, usually *Escherichia coli* and *Klebsiella* species), enterococci (15 percent), bacteroides (10 percent), *Clostridium* species (10 percent), and less commonly group D *Streptococcus* and *Staphylococcus* species.

CLINICAL FEATURES

- Common symptoms of acute biliary colic in decreasing order of frequency include right upper quadrant pain or epigastric pain; nausea and vomiting; pain referred to right shoulder or left upper back;[3] pain that is persistent, not colicky, and lasts for 2 to 6 h; association with meals (although *not* related to meals in one-third of patients[3,4]).
- Peak symptoms of acute biliary colic occur between 9 P.M. and 4 A.M.[4]
- Signs of acute biliary colic include right upper quadrant or epigastric tenderness without peritonitis, and sometimes volume depletion due to emesis.
- Symptoms and signs of acute biliary colic are easily confused with dyspepsia, peptic ulcer disease, gastritis, and esophageal reflux.[5,6]
- Symptoms of acute cholecystitis include right upper quadrant pain that lasts longer than 6 h, fever, chills, nausea, vomiting, and anorexia.
- Signs of acute cholecystitis include sharp, well-localized right upper quadrant tenderness, Murphy's sign (increased pain or inspiratory arrest during deep subcostal palpation of the right upper quadrant with inspiration, which is 97 percent sensitive for acute cholecystitis,[3] but only 48 percent sensitive in the elderly[7]), toxic appearance, fever, volume depletion, abdominal distention, and hypoactive bowel sounds.
- Gallstone pancreatitis is present in 30 to 70 percent of cases of acute pancreatitis, and symptoms include epigastric or diffuse abdominal pain that radiates to the back, nausea, emesis, and fever.
- Ascending cholangitis is a potentially life-threatening illness that presents with symptoms of fever, jaundice, and right upper quadrant pain (Charcot's triad, but all three features are present in only 25 percent), mental confusion, and shock.

DIAGNOSIS AND DIFFERENTIAL

- Laboratory studies that may aid diagnosis include white blood cell count (leukocytosis and left shift suggest cholecystitis but do not exclude it), serum bilirubin and alkaline phosphatase levels (normal or mild elevation), amylase and/or lipase levels (to exclude pancreatitis), urinalysis, electrolytes, blood urea nitrogen levels, and creatinine level, and pregnancy test in females of childbearing age.
- No single laboratory test or combination of tests has a sufficiently high sensitivity to detect cholecystitis.[8]
- Radiographic tests that may be helpful include plain abdominal films (but show gallstones only 10 to 20 percent of the time), chest x-rays to rule out pneumonia, and electrocardiograms in older patients to rule out myocardial infarction.
- Ultrasound of the hepatobiliary tract is the study of choice to diagnose biliary tract disease, with a sensitivity of 94 percent and specificity of 78 percent.[9]
- Differential diagnosis includes gastritis, peptic ulcer disease, hepatitis, pancreatitis, gastroesophageal reflux, renal colic, pyelonephritis, appendicitis, pneumonia, acute myocardial infarction, pelvic inflammatory disease, perihepatitis, and ectopic pregnancy.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Uncomplicated biliary colic will resolve spontaneously, while cholecystitis, pancreatitis, and cholangitis need hospital admission.
- Isotonic crystalloid intravenous (IV) fluids should be administered to correct volume deficits and electrolyte abnormalities. Perfusion should be supported in the patient with shock.
- Antiemetics (promethazine 12.5 to 25 mg IV) or

antispasmodics (glycopyrrolate 0.1 mg IV) should be given for vomiting.
- Meperidine (0.5 to 1.0 mg/kg IV or intramuscular, IM) is the preferred drug for analgesia because it causes less spasm of the sphincter of Oddi. Ketorolac (15 to 30 mg IV or 30 to 60 mg IM) is an alternative in patients without cholecystitis.
- Antibiotics should be given to any patient suspected of having cholecystitis. For nonseptic patients, a third-generation cephalosporin should be adequate (cefotaxime 1 to 2 g IV q 8 h, ceftazidime 1 to 2 g IV q 8 h, ceftizoxime 1 to 2 g IV q 8 to 12 h, or ceftriaxone 1 to 2 g IV q 12 to 24 h). For patients with sepsis, triple therapy with ampicillin (0.5 to 1.0 g IV q 6 h), gentamicin (3 mg/kg/day IV q 8 h), and clindamycin (1200 to 2700 mg/day divided into 2, 3, or 4 equal doses) should be given.
- Patients diagnosed with acute cholecystitis, gallstone pancreatitis, or cholangitis require immediate surgical consultation and hospital admission.
- Patients with uncomplicated biliary colic who are asymptomatic or improved with supportive treatment after 4 to 6 h and are able to maintain oral hydration can be discharged. Oral narcotic-acetaminophen analgesics may be prescribed for 24 to 48 h. Timely outpatient follow-up should be arranged. The patient should be instructed to return to the emergency department for fever, worsening abdominal pain, intractable vomiting, or another significant attack.

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51 PANCREATITIS

Robert J. Vissers

EPIDEMIOLOGY

- In the United States, cholelithiasis or alcohol abuse accounts for 90 percent of all cases of acute pancreatitis (AP).[1]
- The list of other factors associated with AP is extensive (see Table 51-1).
- The overall prevalence is estimated to be 0.5 percent.[1]
- About 5 to 10 percent of patients with acute pancreatitis develop complications or die.

PATHOPHYSIOLOGY

- The central cause is believed to be the intracellular activation of digestive enzymes and autodigestion of the pancreas.[2]
- Pancreatic digestion from activated proteolytic enzymes leads to edema, interstitial hemorrhage, vascular damage, coagulation, and cellular necrosis. Destruction of the pancreatic parenchyma causes a local inflammatory reaction that contributes to the vascular dilatation, permeability, and edema.
- AP also can cause a generalized systemic inflammatory response that may lead to shock, adult respiratory distress syndrome (ARDS), and multisystem organ failure.[3]

TABLE 51-1 Common Etiologic or Contributing Factors in Acute Pancreatitis

Ethanol ingestion
Biliary tract disease
Trauma, penetrating or blunt
Penetrating peptic ulcer
Following endoscopic procedures
Obstruction secondary to neoplasms, diverticula, and polyps
Metabolic disturbances
Hyperlipemia (Frederickson types I, IV, V)
Hypercalcemia
Diabetes mellitus, diabetic ketoacidosis
Uremia
Viral infections
Viral hepatitis
Infectious mononucleosis
Coxsackie group B
HIV
Pregnancy—any trimester, postpartum
Collagen vascular disease
Liver disease
Generalized infections
Drugs
Oral contraceptives
Azathioprine
Glucocorticoids
Tetracyclines
Isonazid
Indomethacin
Thiazides
Salicylates
Calcium
Warfarin

CLINICAL FEATURES

- The patient typically presents with moderate distress, a boring epigastric pain that radiates to the back, tachycardia, nausea and vomiting, and abdominal distention.[1]
- Cullen's sign, a bluish discoloration around the umbilicus, and Grey Turner's sign, a bluish discoloration in the left flank, are characteristic but rare signs of hemorrhagic pancreatitis.
- Complications may be life-threatening (see Table 51-2).
- Blood loss, refractory hypotension, and respiratory failure may accompany more severe forms.[4]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is made by a suggestive history and physical associated with elevated pancreatic enzymes.[5,6]

TABLE 51-2 Complications of Acute Pancreatitis

Pulmonary
Pleural effusions, usually left-sided
Atelectasis
Hypoxemia
Adult respiratory distress syndrome (>50% mortality)
Cardiovascular
Myocardial depression
Hemorrhage, hypovolemia, and myocardial depressant factor
Metabolic
Hypocalcemia
Hyperglycemia
Hyperlipidemia
Coagulopathy, disseminated intravascular coagulopathy
Other
Hemorrhage
Colonic perforation
Renal failure
Erythema-nodosum dermatitis
Arthritis
Pseudocyst
Abscess

- Amylase is found primarily in the pancreas and salivary glands; however, low levels also can be found in the fallopian tubes, ovaries, testes, adipose tissue, small bowel, lung, thyroid, skeletal muscle, and certain neoplasms, making this a relatively nonspecific test.[7] Amylase more than three times the upper limit of normal has a specificity of 75 percent and a sensitivity of 80 to 90 percent for AP.[6,8]
- Lipase is more specific than amylase for AP and is the preferred test. There is no benefit to ordering both amylase and lipase.[9]
- Leukocytosis may be present, and an elevated alkaline phosphatase suggests biliary disease.
- Hypotension, tachycardia > 130 beats per minute, P_{O_2} <60 mmHg, oliguria, increasing blood urea nitrogen (BUN) or creatinine, and hypocalcemia are indicators of a potentially complicated course[10] (see Table 51-3).

TABLE 51-3 Ranson Criteria for Predicting Mortality Risk from Acute Pancreatitis

ON ADMISSION	48 HOURS LATER
Age over 55	Change in HCT (falling) decreased more than 10 percent
Blood sugar >200 mg/dL	Rise in BUN over 5 mg/dL
WBC > 16,000/μL	↓ CA^{2+} below 8 mg/dL
SGOT > 250 Sigma-Fankel units/L	↓ Arterial P_{O_2} below 60 mmHg
	Rapid fluid sequestration over 6 L
LDH > 700 IU/L	Base deficit over 4 meq/L

ABBREVIATIONS: WBC = white blood cells; SGOT = serum glutamic oxaloacetic transaminase; LDH = lactic dehydrogenase; HCT = hematocrit; BUN = blood urea nitrogen.

- Plain radiographs of the abdomen are usually not helpful. Calcification is suggestive of chronic pancreatitis, and a sentinel loop or a colon-cutoff sign suggesting ileus may be present but is not diagnostic.[1]
- Ultrasonography is helpful in the identification of gallstones or dilatation of the biliary tree.
- Computed tomography is the study of choice for visualizing the pancreas, confirmation of inflammation, and identification of local complications. It cannot be used to rule out AP.[11]
- The differential diagnosis of AP includes left lower lobe pneumonia, rupture of a pseudocyst, gallbladder disease, peritonitis, peptic ulcer disease, small bowel obstruction, renal colic, a dissecting aortic aneurysm, diabetic ketoacidosis, and gastroenteritis.[2,4]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of AP primarily involves pancreatic rest (nothing taken orally), fluid resuscitation, pain control, and prevention of vomiting.[2,4]
- The mainstay of treatment for AP is fluid resuscitation with normal saline to maintain blood pressure and adequate urine output.[1]
- Pressors are indicated in patients with persistent hypotension despite adequate fluid resuscitation.
- Oxygen should be administered to maintain a pulse oximetry greater than 95 percent.
- A nasogastric tube may be used if a patient is distended with persistent vomiting; however, no studies have demonstrated that its presence alters the course of the illness.
- Supportive care includes adequate analgesia and the administration of antiemetics.[1]
- Urgent decompression by endoscopic sphincterotomy of the ampulla of Vater is indicated in persistent biliary obstruction.[4]
- Patients with mild pancreatitis, no evidence of systemic complications, and a low likelihood of biliary tract disease may be managed as outpatients if they are able to tolerate oral fluids and their pain is well controlled.
- Patients with significant systemic complications, shock, or extensive pancreatic necrosis require an intensive care setting.[12]

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52 COMPLICATIONS OF GENERAL AND UROLOGIC SURGICAL PROCEDURES

David M. Cline

- Fever, respiratory complications, genitourinary complaints, wound infections, vascular problems, and complications of drug therapy are some common postoperative disorders seen in the emergency department (ED). Most of these are discussed elsewhere in this book; certain specific problems are mentioned here.
- The causes of postoperative fever are listed as the five W's: *wind* (respiratory), *water* urinary tract infection (UTI), *wound, walking* (deep venous thrombosis, or DVT), and *wonder* drugs (pseudomembranous colitis, or PMC).[1]

CLINICAL FEATURES

- Fever in the first 24 h is usually due to atelectasis, necrotizing fasciitis, or clostridial infections.
- In the period from 24 to 72 h, pneumonia, atelectasis, intravenous catheter–related thrombophlebitis and infections are the major causes.
- UTIs are seen 3 to 5 days postoperatively.
- DVT typically occurs 5 days after the procedure, and wound infections generally manifest themselves 7 to l0 days after surgery.
- Antibiotic-induced PMC is seen 6 weeks after surgery.

Respiratory Complications

- Postoperative pain and inadequate clearance of secretions contribute to the development of atelectasis. Fever, tachypnea, tachycardia, and mild hypoxia are usually seen. Pneumonia may develop 24 to 96 h later.
- Hypoxia, tachycardia, a widened A-a gradient, and respiratory distress should point to the diagnosis of pulmonary embolism (see Chap. 28 for diagnosis and management).

Genitourinary Complications

- UTIs are more common after instrumentation of the urinary tract. Urinary retention occurs in 4 percent of all surgical patients and in 60 percent of patients after urethral surgery. It is more common in elderly males, especially after excessive fluid administration and spinal anesthesia. Lower abdominal pain, urgency, and inability to void should lead the clinician to suspect urinary retention.[2,3]
- Oliguria or anuria commonly results from volume depletion. Intrinsic factors such as acute tubular necrosis (ATN), drug nephrotoxicity, and postrenal obstructive uropathy may also lead to acute renal failure.

Wound Complications

- Hematomas result from inadequate hemostasis. Careful evaluation to rule out infections must be undertaken.
- Seromas are collections of clear fluid under the wound. Extremes of age, diabetes, poor nutrition, necrotic tissue, poor perfusion, foreign bodies, and wound hematomas contribute to the development of wound infections.
- Necrotizing fasciitis is characterized by extremely painful, erythematous, swollen, and warm areas without sharp margins. This staphylococcal infection spreads rapidly. Patients will exhibit marked systemic toxicity, and crepitance and bullae may be present.[4]
- Wound dehiscence can occur due to diabetes, poor nutrition, chronic steroid use, and inadequate or improper closure of the wound. Dehiscence of an abdominal wound may result in evisceration of abdominal organs.

Vascular Complications

- Superficial thrombophlebitis usually occurs in the upper extremities after intravenous catheter insertion or in the lower extremities because of stasis and varicosity of veins.
- DVT commonly occurs in the lower extremities. Swelling and pain of the calf are commonly encountered. (See Chap. 31 for diagnosis and management.)

Drug Therapy Complications

- Many drugs are known to cause fever and antibiotic-induced diarrhea.[5]
- PMC, a dreaded complication, is caused by *Clostridium difficile* toxin. Bloody, watery diarrhea, fever, and crampy abdominal pain are the usual complaints.

DIAGNOSIS AND DIFFERENTIAL

- Patients with suspected respiratory complications should have chest x-rays. They may reveal platelike or discoid atelectases, pneumonia, or pneumothorax. Pneumothorax occurs early after certain surgical procedures or catheter insertion; a chest x-ray will help confirm the diagnosis.
- Patients with oliguria or anuria should be evaluated for signs of hypovolemia or urinary retention.
- Diagnosis of PMC is established by demonstrating *C. difficile* cytotoxin in the stool. In 27 percent of the cases, however, the assay can be negative.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Always discuss patients and proposed treatments with the surgeon who initially cared for the patients.
- Although debilitated patients may need hospitalization, many patients with atelectasis can be treated as outpatients.
- Postoperative pneumonia is polymicrobial, and an antipseudomonal antibiotic with an aminoglycoside is usually recommended. Most patients with

UTI can be managed as outpatients with oral antibiotic therapy (see Chap. 55).
- Insertion of a Foley catheter and prompt drainage will alleviate urinary retention. There is no need to clamp the catheter periodically. Prophylactic antibiotics are reserved for patients who have had urinary tract instrumentation, those with prolonged retention, and those at risk for infection.
- Wound hematomas may require removal of some sutures and evacuation. Surgical consultation before treatment is appropriate. Seromas can be treated with needle aspiration and wound cultures. Admission may not always be necessary.
- Most wound infections can be treated with oral antibiotics (usually the surgeon's choice) unless there are systemic symptoms and signs. Perineal infections usually require admission and parenteral antibiotics. As surgical debridement and parenteral antibiotics are indicated for necrotizing fasciitis, physicians should start clindamycin 900 mg and penicillin G 6 million units intravenously.[6]
- Most patients with superficial thrombophlebitis can be treated with local heat and elevation of the affected area if there is no evidence of cellulitis or lymphangitis. Patients with suppurative thrombophlebitis—characterized by erythema, lymphangitis, fever, and severe pain—should be hospitalized and treated with excision of the affected vein.
- Fluid resuscitation, oral vancomycin, and metronidazole, orally or intravenously, are currently available treatment modalities for drug-induced PMC (see Chap. 48).

SPECIFIC CONSIDERATIONS

COMPLICATIONS OF BREAST SURGERY

- Wound infections, hematomas, pneumothorax, necrosis of the skin flaps, and lymphedema of the arms after mastectomy are common problems seen after breast surgery.
- Lymphedema of the arm occurs in 5 to 10 percent of patients. Elevation of the arm and minor restriction of activity will help to reduce swelling.

COMPLICATIONS OF GI SURGERY

Intestinal Obstruction

- Neuronal dysfunction following any surgery where the peritoneum is entered causes paralytic ileus. Following gastrointestional (GI) surgery, small bowel tone returns to normal within 24 h, gastric function within 2 days, and colonic function within 3 days.
- Prolonged ileus should alert the clinician to peritonitis, abscesses, hemoperitoneum, pneumonia, sepsis, and electrolyte imbalance. Clinical features include nausea, vomiting, obstipation, constipation, abdominal distention, and pain.
- Abdominal x-rays, complete blood count, electrolytes, blood urea nitrogen creatinine levels, and urinalysis should be obtained.
- Treatment of adynamic ileus consists of nasogastric suction, bowel rest, and hydration. Mechanical obstruction is usually due to adhesions and may require surgical intervention.

Nonobstructive Complications

- Intraabdominal abscesses are caused by preoperative contamination or postoperative anastomotic leaks. Diagnosis can be confirmed by computed tomography (CT) or ultrasonography. Surgical exploration, evacuation, and parenteral antibiotics will be required.
- Pancreatitis occurs especially after direct manipulation of the pancreatic duct. Patients typically have nausea, vomiting, abdominal pain, and leukocytosis. Lumbar pain, left pleural effusion, Turner's sign (discoloration of the flank), and Cullen's sign (periumbilical ecchymosis) may be present. Serum amylase and lipase levels are usually elevated. (See Chap. 51 for management.)
- Cholecystitis and biliary colic have been reported as postoperative complications. Elderly patients are more prone to develop acalculous cholecystitis.
- Fistulas, either internal or external, may result from direct bowel injury and require surgical consultation and hospitalization.
- Anastomotic leaks are especially devastating after esophageal or colon surgery. Esophageal leaks occur 10 days after the procedure. Dramatic presentation with shock, pneumothorax, and pleural effusion is usually seen.
- Dumping syndrome is noticed in gastric bypass procedures. It is due to the sudden influx of hyperosmolar chyle into the small intestine, resulting in fluid sequestration and hypovolemia. Patients experience nausea, vomiting, epigastric discomfort, palpitations, dizziness, and sometimes syncope.
- Alkaline reflux gastritis is caused by the reflux of bile into the stomach. Endoscopic evaluation will establish the diagnosis. Postvagotomy diarrhea and afferent loop syndrome are seen in some patients.
- Complications of percutaneous endoscopic gas-

trostomy (PEG) tubes include infections, hemorrhage, peritonitis, aspiration, wound dehiscence, sepsis, and obstruction of the tube.
- Complications arising from stomas are due to technical errors or to underlying disease such as Crohn's disease and cancer. Ischemia, necrosis, bleeding, hernia, and prolapse are sometimes seen.
- Colonoscopy may cause hemorrhage, perforation, retroperitoneal abscesses, pneumoscrotum, pneumothorax, volvulus, and infection.
- Complications of rectal surgery include urinary retention, constipation, prolapse, bleeding, and infections.
- Finally, tetanus has been known to occur following surgical wounds.[7, 8]

COMPLICATIONS OF UROLOGIC SURGERY

- Hematuria, urinary retention, strictures, and infections may occur following prostate surgery.[9,10]
- After extracorporeal shockwave lithotripsy, pain and hematuria persist for some time.[11] Suspected perineal hematomas from subcapsular renal hemorrhage can be confirmed by CT or ultrasonography. Ureteric reobstruction from stone fragments can be diagnosed by intravenous pyelography.
- Epididymitis, scrotal swelling, and hemorrhage are sometimes seen following vasectomies.[12]

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Section 9

RENAL AND GENITOURINARY DISORDERS

53 ACUTE RENAL FAILURE

David M. Cline

- Renal dysfunction and acute renal failure (ARF) present with a wide variety of manifestations, depending on the underlying etiology.
- Risk factors include cardiac disease, hypovolemia from any cause, vascular or thrombotic disorders, glomerular diseases, diseases affecting the renal tubules, use of nephrotoxic drugs, and a variety of anatomic problems of the genitourinary tract.

PATHOPHYSIOLOGY

- Regardless of the cause of ARF, reductions in renal blood flow (RBF) represent a common pathologic pathway for decreasing glomerular filtration rate (GFR).[1] This relationship is most clear in prerenal failure, defined by conditions with normal tubular and glomerular function, where GFR is depressed by compromised renal perfusion. Intrinsic renal failure occurs with diseases of the glomerulus, interstitium, or tubule associated with the release of renal vasoconstrictors.[2] Postobstructive renal failure initially produces an increase in tubular pressure decreasing the filtration driving force. This pressure gradient soon equalizes, and the maintenance of depressed GFR depends on vasoconstrictors.[3]
- Prerenal failure is the most common cause of ARF, accounting for 40 to 80 percent of all cases.[4] Prerenal failure is produced by conditions that decrease renal perfusion (Table 53-1). Besides being an independent cause of ARF, prerenal failure is a common precursor to ischemic and nephrotoxic causes of intrinsic renal failure.[5]
- The etiologies of intrinsic renal failure are subdivided anatomically into diseases of the tubules, interstitium, glomeruli, and vessels (Table 53-2). Intrinsic renal failure accounts for approximately 11 to 45 percent of all cases, depending on the population studied. For adults in a community hospital, prerenal failure accounted for 70 percent of ARF cases as compared with only 11 percent from intrinsic renal etiologies.[6] ARF has a different spectrum in the pediatric population: a higher incidence of intrinsic renal causes for ARF (45 percent) secondary to diseases such as glomerulonephritis and hemolytic-uremic syndrome.[7]
- Acute tubular necrosis (ATN) secondary to renal ischemia accounts for the majority of cases of intrinsic renal failure. Nephrotoxins are the second most common cause of ATN, accounting for approximately 25 percent.
- Postrenal failure accounts for 2 to 5 percent of all cases of ARF but has a significantly higher incidence in selected populations. In large surveys of elderly men for symptoms of urinary obstruction, prevalence between 20 and 35 percent has been estimated.[8]

CLINICAL FEATURES

- Reported mortality rates from ARF have remained the same before and after the advent of dialysis (40 to 90 percent).[9,10]
- Deterioration in renal function leads to excessive accumulation of nitrogenous waste products in the serum.
- ARF can be classified as oliguric (<400 mL urine

TABLE 53-1 Common Causes of Acute Renal Failure (ARF)

PRERENAL	RENAL	POSTRENAL
Decreased cardiac output Myocardial ischemia/infarction Valvular heart disease Cardiomyopathy Pericardial tamponade High-output failure	Vascular/ischemia Renal vasculature thrombosis, TTP, DIC, NSAIDs, severe hypertension, hemolytic-uremic syndrome	Penile lesions Phimosis Meatal stenosis Urethral stricture
Hypovolemia Blood loss/hemorrhagic shock Vomiting/diarrhea Diuretics Postobstructive diuresis Fluid sequestration Cirrhosis Pancreatitis Burns General anesthesia Septic shock	Glomerular Primary glomerular diseases (acute glomerulonephritis) or systemic disease with glomerular involvement (SLE, vasculitis, HSP, endocarditis)	Prostatic enlargement—BPH, cancer
	Tubulointerstitial Ischemic ATN, rhabdomyolysis, toxin induced tubular damage (aminoglycosides, radio contrast, solvents, heavy metals, ethylene glycol, myoglobin/hemoglobin), acute interstitial nephritis, infiltrative and autoimmune diseases, infectious agents	Upper urinary tract/ureteral diseases (usually requires bilateral involvement/obstruction) Calculi, tumors, blood clots Papillary necrosis Vesicoureteral reflux Stricture AAA Retroperitoneal fibrosis

ABBREVIATIONS: DIC = disseminated intravascular coagulation; NSAIDs = nonsteroidal anti-inflammatory agents; ATN = acute tubular necrosis; TTP = thrombotic thrombocytopenic purpura; SLE = systemic lupus erythematosus; HSP = Henoch-Schönlein purpura; BPH-benign prostatic hypertrophy; AAA = abdominal aortic aneurysm.

per 24 h) and nonoliguric (>400 mL/24 h). Oliguric renal failure has a higher mortality rate.[11]

- Patients usually have signs and symptoms of their underlying causative disorder but eventually develop stigmata of renal failure. Volume overload, hypertension, pulmonary edema, mental status changes or neurologic symptoms, nausea and vomiting, bone and joint problems, anemia, and increased susceptibility to infection (a leading cause of death) can occur as patients develop more chronic uremia.
- Approximately 20 to 60 percent of patients experiencing ARF will require dialysis;[12] the majority will recover, with only 25 percent requiring long-term dialysis.[13]

DIAGNOSIS AND DIFFERENTIAL

- Physical exam should assess vital signs and volume status, establish urinary tract patency and output, and search for signs of chemical intoxication, drug usage, muscle damage, or associated systemic diseases.
- Diagnostic studies include urinalysis, blood urea nitrogen (BUN) and creatinine levels, serum elec-

TABLE 53-2 Laboratory Studies Aiding in the Differential Diagnosis of Acute Renal Failure

TEST EMPLOYED	PRERENAL	RENAL*	POSTRENAL†
Urine sodium (meq/L)	< 20	> 40	>40
FE_{Na} (%)‡	< 1	> 1	> 1
Renal failure index (RFI)§	< 1	> 1	> 1
Urine osmolality (mosm/L)	> 500	< 350	< 350
Urine/serum creatinine ratio	> 40:1	< 20:1	< 20:1
Serum urea nitrogen/creatinine ratio	> 20:1	= 10:1	> 10:1

* FE_{Na} may be <1 in intrinsic renal failure patients with glomerulonephritis, hepatorenal syndrome, radio contrast acute tubular necrosis, myoglobinuric and hemoglobinuric acute renal failure, renal allograft rejection, and with certain drugs (captopril and nonsteroid anti-inflammatory agents).
† Can see indices similar to prerenal early in course of obstruction. With continued obstruction, tubular function is impaired and indices mimic those of renal causes.

‡ Fractional excretion of sodium (%) $= \dfrac{\text{Urine sodium/serum sodium}}{\text{Urine creatinine/serum creatinine}} \times 100$

§ $\text{RFI} = \dfrac{\text{Serum sodium}}{\text{Urine creatinine/serum creatinine}} \times 100$

trolyte levels, urinary sodium and creatinine levels, and urinary osmolality. Analysis of these tests allows most patients to be placed in either the prerenal, renal, or postrenal group.
- Fractional excretion of sodium and renal failure index can be calculated to help in this categorization[14] (Table 53-2).
- Normal urinary sediment may be seen in prerenal and postrenal failure, hemolytic-uremic syndrome, and thrombotic thrombocytopenic purpura.
- The presence of albumin may indicate glomerulonephritis or malignant hypertension.
- Granular casts are seen in ATN, glomerulonephritis, and interstitial nephritis. Albumin and red blood cell casts are found in both glomerulonephritis and malignant hypertension. White blood cell casts are seen in interstitial nephritis and pyelonephritis.
- Crystals can be present with renal calculi and certain drugs (sulfas, ethylene glycol, and radiologic contrast agents).
- Ultrasonography is the radiologic procedure of choice in most patients with acute renal failure when hydronephrosis is suspected.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial care of patients with ARF focuses on treating the underlying cause and correcting fluid and electrolyte derangement. Efforts should be made to prevent further renal damage and provide supportive care until renal function has recovered. (See Chap. 6 for treatment of electrolyte and acid-base disorders.)

Prerenal Failure
- Effective intravascular volume should be restored with isotonic fluids (normal saline and lactated Ringer's) at a rapid rate in appropriate patients.[15]
- If cardiac failure is causing prerenal azotemia, intravascular volume should be reduced (i.e., with diuretics) to improve cardiac output and renal perfusion.

Postrenal Failure
- Appropriate urinary drainage should be established. The exact procedure will vary depending on the level of obstruction.
- A Foley catheter should be placed to relieve obstruction caused by prostatic hypertrophy. Percutaneous nephrostomy may be required for ureteral occlusion until the patient's status is stabilized and definitive surgery to correct the obstruction can take place.
- For the acutely anuric patient, obstruction is the major consideration. If no urine is obtained on initial bladder catheterization, emergency urologic consultation should be considered.
- With chronic urinary retention, postobstructive diuresis may occur due to osmotic diuresis or tubular dysfunction. Patients may become suddenly hypovolemic and hypotensive. Urine output must be closely monitored, with appropriate fluid replacement.

Renal Failure
- Nephrotoxic agents (drugs, intravenous contrast) should be avoided. Diuretics (i.e., furosemide 20 to 80 mg intravenously) can occasionally augment diuresis and convert oliguric into nonoliguric renal failure.[16] Patients with nonoliguric ARF have improved recovery of renal function and lower mortality. (Volume must be restored in these patients before using diuretics.)
- Low-dose dopamine (1 to 5 μg/kg/min) may improve renal blood flow and urine output but does not improve recovery or lower mortality rates.[17] It is probably best used in ARF patients with congestive heart failure.
- Mannitol may be protective against myoglobinuric ARF in early rhabdomyolysis.
- Adequate circulating volume must be restored first.
- Renally excreted drugs (digoxin, magnesium, sedatives, and narcotics) should be used with caution since therapeutic doses may accumulate to excess and cause serious side effects. Fluid restriction (500 mL plus daily urine output) may be required.

Dialysis
- If treatment of the underlying cause fails to improve renal function, hemodialysis or peritoneal dialysis should be considered. Decisions about dialysis are usually made by the nephrology consultant.
- Dialysis is often initiated when the BUN level is >100 mg/dL or the serum creatinine level is >10 mg/dL.
- Patients with complications of ARF such as cardiac instability (due to metabolic acidosis and hyperkalemia), intractable volume overload, hyperkalemia, and uremia (i.e., encephalopathy, pericarditis, and bleeding diathesis) not easily corrected by other measures should be considered for emergency dialysis.

DISPOSITION

- Patients with new-onset ARF usually require hospital admission, often to an intensive care unit.
- Transferring patients to another institution should be considered if nephrology consultation and dialysis facilities are not available.

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54 EMERGENCIES IN DIALYSIS PATIENTS

David M. Cline

- Dialysis patients sustain multiple complications of their disease process and treatment. (See the appropriate chapters for discussion on the management of hypertension, heart failure, bleeding disorders, and electrolyte disorders.)

EPIDEMIOLOGY

- The 1996 annual data report of the United States Renal Data System (USRDS) noted there were 73,091 new cases of end-stage renal disease (ESRD) (incidence, 2.7 per 10,000), with 283,932 patients being treated for ESRD (prevalence, 10.4 per 10,000) during that year.[1]
- Diabetes mellitus is the most common disease causing ESRD, accounting for 32.5 percent of patients, followed by hypertension (24.5 percent), glomerulonephritis (17.7 percent), and cystic kidney disease (4.7 percent).[1]
- Cardiac causes account for approximately 50 percent of all cases of ESRD death.[2] Infectious causes of death occur in 25 percent of patients in the age group from 20 to 44 years.

PATHOPHYSIOLOGY

- The pathophysiology of renal failure can be categorized by three mechanisms: (1) excretory failure, or inability to excrete over 70 chemicals known to accumulate in renal failure, most notably urea; (2) biosynthetic failure, the loss of renal hormones vitamin D and erythropoietin; and (3) regu-

latory failure, the disruption of normal feedback mechanisms leading to the oversecretion of hormones that exacerbate uremia.

UREMIC PERICARDITIS

- The classic symptom is chest pain, which is partially relieved by sitting up and leaning forward.
- A pericardial friction rub may not be heard or may be heard intermittently. Low-grade fever and atrial arrhythmias (paroxysmal atrial tachycardia, atrial flutter–atrial fibrillation) are common as well.
- Echocardiography often demonstrates a pericardial effusion.
- The pericardial fluid may impede venous return, leading to congestive heart failure and hypotension.
- Treatment of simple pericarditis in uremic patients is intensive dialysis. Symptomatic tamponade is relieved by pericardiocentesis.

CARDIAC ARRHYTHMIAS AND CARDIAC ARREST

- The most common cause of cardiac arrest in uremic patients is hyperkalemia. The treatment should include administration of calcium gluconate (10 mL of 10% solution), followed by infusion of 50 mL of 50% glucose, along with 20 U of regular insulin, and infusion of 50 to 100 meq of intravenous sodium bicarbonate.
- Hemo- or peritoneal dialysis, using a lower concentration of K^+ in the dialysate, is the most effective way to reduce the potassium level and should be employed as soon as possible.

HYPOTENSION

- A sudden drop in blood pressure is a common complication during dialysis; if not promptly treated, it can lead to cardiac arrest. Subjective symptoms—such as muscle cramps, nausea, yawning, and mental confusion— may precede the actual hypotension in most patients but not in all.[3]
- Treatment consists of the rapid infusion of isotonic saline. Patients usually respond to 500 mL or less.
- In rare instances, the use of vasopressors may be required.
- Other causes of hypotension—including pericardial disease, sepsis, gastrointestinal (GI) bleeding, or cardiac dysfunction—should be ruled out.

NEUROLOGIC COMPLICATIONS

- Uremic encephalopathy presents with cognitive defects, memory loss, slurred speech, and asterixis. The progressive neurologic symptoms of uremia are the most common indications for initiating dialysis.
- Dialysis disequilibrium—demonstrated by symptoms of increased intracranial pressure, including nausea, vomiting, headache, and mental confusion—can develop soon after or within a few hours of a dialysis treatment. It is common after first dialysis, but can also occur on a rare occasion, even in patients treated with chronic dialysis.
- Therapy is mannitol 0.5 g/kg intravenously. Subdural hematoma is more common in hemodialysis patients and should be differentiated from the above disorders with a computed tomography scan.

GASTROINTESTINAL DISORDERS

- Upper GI bleeding may result from uremic gastritis, peptic ulcer disease, or excess anticoagulation. Management does not differ from that in nonuremic patients.
- Caution should be exercised, however, in using large doses of magnesium-containing antacids. Since magnesium is normally excreted by the kidney, abnormal levels can accumulate in the plasma of the uremic patient, leading to mental obtundation and respiratory depression.

PROBLEMS PECULIAR TO PERITONEAL DIALYSIS

- Infection of the peritoneal membrane is the most frequent and critical complication in patients receiving chronic peritoneal dialysis.[4]
- The symptoms may be subtle and include abdominal discomfort, pain during inflow, and fever. Physical examination may reveal abdominal tenderness, particularly around the catheter site, and decreased bowel sounds.
- Laboratory evaluation should include complete blood cell count and analysis of peritoneal fluid for cell count, Gram stain, protein, culture, and sensitivity. A bag of drained dialysate should be used for culture and analysis. A variety of microorganisms (bacterial, fungal, and parasitic) have been found after culturing the fluid from the peritoneal cavity of dialysis patients.
- The mainstay of therapy is the infusion of an ap-

propriate antimicrobial agent into the peritoneal cavity. Depending upon the results of Gram stain, usually cephalothin 500 mg/L is added to the dialysate. Alternatively vancomycin (1 g) is given, and possibly gentamicin (100 mg) is added. To avoid fibrin formation, 1000 U of heparin is added to the infusion.
- If a patient experiences recurrent bouts of peritonitis, tunnel infection, or intraabdominal abscess, the catheter should be changed.[5] Appropriate surgical drainage of intraabdominal abscess is also warranted to prevent relapse.

PROBLEMS RELATED TO VASCULAR ACCESS

- The most frequent complications associated with the external shunts are clotting and infection. When the shunt is acutely clotted, the vascular surgeon must be notified immediately.
- Declotting procedures using a Fogarty balloon catheter are normally accomplished by the surgeon in the operating suite. In some instances, however, instillation of urokinase, 5000 to 10,000 U, into the arterial and venous parts of the clotted shunt may dissolve the clot and prevent the need for further intervention.
- Infection of the cannula site is a significant problem in the hemodialysis patient. Coagulase-positive staphylococci and *Staphylococcus epidermidis* are frequently cultured from the exit site.[6]
- Physical examination may reveal local inflammation, tenderness over the cannula tips, and purulent drainage at the exit sites.
- Vancomycin, 1 g intravenously, is the drug of choice.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 89, ''Emergencies in Renal Failure and Dialysis Patients,'' by Richard Sinert.

55 URINARY TRACT INFECTIONS AND HEMATURIA

Kama Guluma

EPIDEMIOLOGY

- There are four groups of patients at risk for urinary tract infection (UTI): neonates, girls, young women, and older men.
- In neonates, UTI occurs with a male : female (M : F) incidence ratio of 1.5 : 1, often as part of a gram-negative sepsis syndrome.
- In preschool, the M : F ratio inverts, with girls being affected 10 times as often as boys. By school age, the incidence increases to 5 percent and is almost exclusively in girls.
- In young adult women, the incidence of UTI rises with commencement of sexual activity. Bacteriuria in men under age 50, in contrast, is rare and is typically associated with a sexually transmitted infection of the urethra or prostate.
- The incidence of UTI in postmenopausal women increases with age as, under the influence of decreased estrogen, *Escherichia coli* replaces vaginal lactobacilli. In males over the age of 50, the incidence of UTI approaches that of females dramatically due to the increasing prevalence of prostate hypertrophy and related instrumentation.
- Asymptomatic bacteriuria (ABU), defined as two successive urine cultures with more than 10^5 per milliliter of a single bacterial species in an asymptomatic patient, occurs in up to 30 percent of pregnant women, in up to 40 percent of female nursing-home residents, and commonly in patients with indwelling urinary catheters.

TABLE 55-1 Etiologic Agents in Uncomplicated Urinary Tract Infection

ORGANISM	INCIDENCE (%)
Escherichia coli	>80
Klebsiella sp. *Proteus* sp. *Enterobacter* sp. *Pseudomonas* sp.	5–20
Group D streptococci *Chlamydia trachomatis** *Staphylococcus saprophyticus**	<5

* Much more common in the ''dysuria-pyuria'' syndrome where sterile or low colony count culture results are obtained.

PATHOPHYSIOLOGY

- A thin film of urine remains in the functionally intact bladder after each void. Urinary pathogens, adhering to the uroepithelium with adhesins, fibrae, or pili, are removed from the film by mucosal production of organic acids. Incomplete bladder emptying renders this mechanism ineffective and is responsible for the increased frequency of UTI in patients with structural or neurogenic bladder outflow abnormalities.
- Ureteral valves restrict the majority of uncomplicated UTIs to the bladder. If ascending infection of the urinary tract occurs, renal defense mechanisms including local antibody secretion and complement activation are induced.
- In uncomplicated UTIs, the most common urinary pathogen is *E. coli* (see Table 55-1). Up to one-half of women with symptomatic UTI may have low-grade or early infection, usually with 10^2 to 10^4 colony-forming units (CFU) per mL of *E. coli, Staphylococcus saprophyticus,* or *Chlamydia trachomatis.* In complicated UTIs, i.e., in those occurring in patients with underlying urologic or neurologic dysfunction, *Pseudomonas* spp. and enterococci are likely pathogens.
- In young women, the risk of UTI is independently associated with recent sexual intercourse, recent use of a diaphragm with spermicide, and a history of UTI.[1,2] A ''milking action'' of the female urethra during intercourse can increase the concentration of bacteria in the bladder by up to a factor of 10.[1,2] The use of a spermicide enhances vaginal colonization with *E. coli.*[2]

CLINICAL FEATURES

- Urinary tract infections presenting to the emergency department (ED) are categorized into two major clinical syndromes: acute cystitis and acute pyelonephritis.
- Patients with acute cystitis, in which infection is localized to the bladder, will typically present with dysuria, frequency, and suprapubic discomfort. In the male, however, dysuria with a urethral discharge is more likely to represent urethritis.[3]
- Patients with acute pyelonephritis, in which infection has spread to the kidney, typically present with localized kidney pain, fever, chills, nausea, vomiting, malaise, and costovertebral angle tenderness, in addition to the preceding symptoms of cystitis.
- Subclinical pyelonephritis, a syndrome in which infection has spread to the kidneys without overt symptoms and signs beyond those of cystitis, may be clinically indistinguishable from acute cystitis without specialized diagnostic techniques. About 25 to 30 percent of patients diagnosed with acute cystitis are estimated to have subclinical pyelonephritis. Epidemiologic risk factors include lower socioeconomic status, pregnancy, structural urinary tract abnormality, history of UTI relapse after treatment, prior history of acute pyelonephritis, frequent UTIs, symptoms for more than 7 days, or diabetes or immunosuppressing infections.[4]

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis of UTI includes vulvovaginitis, cervicitis, mechanical or chemical urethritis, and urolithiasis. Factors that are independent predictors of a UTI in a patient with dysuria are advanced age, history of UTI, back pain, pyuria, hematuria, and bacteriuria.[3–5]
- A midstream urine specimen, or a sample from urethral catheterization if necessary, should be analyzed for nitrite and leukocyte esterase reactions and pyuria, bacteriuria, and hematuria.
- The urine nitrite reaction has a >90 percent specificity, but a low sensitivity (50 percent); a negative result does not rule out the diagnosis of UTI.[6]
- A positive urine leukocyte esterase reaction from pyuria is an indicator of UTI. Its sensitivity is 48 percent overall in ED patients,[6] but approaches 88 percent in symptomatic women with high levels of pyuria.
- Abnormal pyuria in women is defined as 2 to 5 leukocytes white blood cell count (WBC) per high-power field (HPF, or 400x) from a centrifuged specimen. In men, more than 1 to 2 WBC/HPF is significant.[3] False negatives can occur with

large, dilute urine volumes, self-medication, or renal obstruction.
- Bacteriuria may be absent in women with low-grade, noncoliform or chlamydial UTIs, but is a specific marker for detection of UTI. More than one bacteria per oil-power field (1000×) in an uncentrifuged specimen, or more than 15 per oil-power field in a centrifuged specimen, is significant and highly correlates with a culture result of $>10^5$ CFU/mL. False positives can occur with vaginal or fecal contamination.
- In a male with dysuria, a Gram's stain of urethral discharge may reveal evidence suggestive of a gonococcal or chlamydial urethritis or another sexually transmitted infection.
- Urine cultures should be sent in the following settings: acute pyelonephritis, patients needing to be hospitalized, patients with chronic indwelling urinary catheters, pregnant women, children, and adult males.[3–5]
- Elderly, diabetic, or severely ill patients with acute pyelonephritis that have been poorly responsive to therapy should undergo imaging with renal ultrasound or other modalities to evaluate for obstruction.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- For adult females with few prior episodes of UTI, brief duration of symptoms, no risk factors for subclinical pyelonephritis, and good available follow-up, a 3-day course of oral antibiotic therapy will suffice.[3–5,7] Options include cotrimoxazole, 1 double-strength tablet bid; amoxicillin/clavulanate 875/125 mg bid; ciprofloxacin 250 to 500 mg bid; ofloxacin 200 to 400 mg bid; nitrofurantoin macrocrystals 100 mg qid; and trimethoprim 200 mg bid.
- Adult females with risk factors for subclinical pyelonephritis should receive a 10- to 14-day course of one of the previously mentioned antibiotics. Patients with subclinical pyelonephritis respond poorly to a shorter course of therapy.[5]
- Adult males with lower UTI should also receive a 10-day course of antibiotics, once urethritis and prostatitis are ruled out. An underlying anatomic abnormality should be suspected and referral to a urologist made.
- Patients with signs of cervicitis, new sexual partners, sexual partners with urethritis, or pyuria without bacteriuria should be treated with doxycycline 100 mg bid for 10 days and cultured for gonococcus.
- Asymptomatic bacteriuria in pregnancy poses a special problem; if untreated, it may progress to symptomatic UTI or pyelonephritis, leading to subsequent complications including miscarriage. Treatment of ABU in pregnancy, with a 7-day course, is therefore unequivocally indicated.
- Adjunctive therapy can include adequate intake of fruit juices containing vitamin C to acidify the urine and enhance diuresis.[8]
- Patients with acute pyelonephritis are distinguished from those with cystitis by the clinical symptoms and signs described earlier. The decision to admit the patient with acute pyelonephritis is based on age, host factors, comorbidity, and response to initial emergency department interventions.
  a. Adequate intravenous (IV) fluid should be administered to dehydrated or vomiting patients.
  b. Cultures should be sent and a dose of an oral or IV antibiotic administered. Intravenous options include a fluoroquinolone, ampicillin plus gentamicin, third-generation cephalosporin, or an extended-spectrum penicillin plus β-lactamase inhibitor.[9]
  c. Of young patients who are able to take oral antibiotics, 80 to 90 percent respond to outpatient oral antibiotic therapy.[10,11] These patients can be discharged on a 10- to 14-day course of one of the oral regimens described earlier.
  d. Patients with intractable nausea and vomiting, unremitting fever, and loss of vasomotor tone should be admitted.
  e. Additional indications for admission involve factors associated with an unfavorable prognosis, such as old age, debility, renal calculi or obstruction, a history of recent hospitalization or instrumentation, diabetes mellitus, chronic nephropathy, sickle cell anemia, underlying carcinoma, or intercurrent cancer chemotherapy. In these cases antimicrobial coverage should be broadened and an antipseudomonal agent added.
- A relapse of UTI in less than 1 month after treatment usually represents a treatment failure.
- A cluster of more than three recurrences in 1 year suggests reinfection and should prompt a referral for a search for structural urologic abnormalities or underlying systemic disease.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 90, ''Urinary Tract Infections,'' by David S. Howes and William F. Young.

56 MALE GENITAL PROBLEMS

David M. Cline

TESTES AND EPIDIDYMIS

TESTES

- Testicular torsion must be the primary consideration in any male (of any age group) complaining of testicular pain. Pain usually occurs suddenly, is severe, and is felt in either the lower abdominal quadrant, the inguinal canal, or the testis.[1] The pain may be constant or intermittent but is not positional, since torsion is primarily an ischemic event.
- When the diagnosis is obvious, urologic consultation is indicated for exploration, since imaging tests can be too time-consuming. In indeterminate cases, color-flow duplex ultrasound and, less commonly, radionuclide imaging may be helpful.[2–4]
- The emergency department (ED) physician can attempt manual detorsion.[5] Most testes torse in a lateral to medial direction, so detorsion is performed in a medial to lateral direction, similar to the opening of a book.[6] The endpoint for successful detorsion is pain relief; urologic referral is still indicated.
- Torsion of the appendages is more common than testicular torsion but is not dangerous, since the appendix testis and appendix epididymis have no known function.[1] If the patient is seen early, diagnosis can be supported by the following: pain is most intense near the head of the epididymis or testis, there is an isolated tender nodule, or the pathognomonic blue-dot appearance of a cyanotic appendage is illuminated through thin prepubertal scrotal skin.
- If normal intratesticular blood flow can be demonstrated with color Doppler, immediate surgery is not necessary, since most appendages calcify or degenerate over 10 to 14 days and cause no harm. If the diagnosis cannot be assured, urologic exploration is needed to rule out testicular torsion.

EPIDIDYMITIS

- Epididymitis is characterized by a gradual onset of pain due to inflammation.
- Bacterial infection is the most common, with infecting agents dependent on the patient's age. In patients below 40 years of age, epididymitis is primarily due to sexually transmitted diseases (STDs).[7] Common urinary pathogens predominate in older men.
- Epididymitis causes lower abdominal, inguinal canal, scrotal, or testicular pain alone or in combination. Due to the inflammatory nature of the pain, patients with epididymitis may note transient pain relief in recumbency, when the scrotal contents are elevated.
- Initially, tenderness is well localized to the epididymis, but progression of inflammation results in the physical examination finding of a single large testicular mass (epididymo-orchitis) that is difficult to differentiate from testicular torsion or carcinoma. At this stage the patient may appear toxic and require admission for intravenous antibiotics (e.g., ceftriaxone 1 to 2 g every 12 h or trimethoprim-sulfamethoxazole 5 mg/kg trimethoprim component every 6 h), scrotal elevation and ice

application, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids for analgesia, and stool softeners.
- Outpatient treatment is an option in patients who do not appear toxic; urologic follow-up within a week is indicated. Oral antibiotic regimens should include 10 days of therapy with one of the following: doxycycline 100 mg bid or ofloxacin 300 mg bid for patients under age 40; for patients age 40 and older, trimethoprim-sulfamethoxazole, one double-strength tablet bid, or a quinolone, such as ciprofloxacin 500 mg bid.
- Orchitis in isolation is rare; it usually occurs with viral or syphilitic disease and is treated with disease-specific therapy, symptomatic support, and urologic follow-up.
- Testicular malignancy should be suspected in patients presenting with asymptomatic testicular mass, firmness, or induration. Ten percent of tumors present with pain due to hemorrhage within the tumor. Urgent urologic follow-up is indicated.

SCROTUM

- Scrotal abscesses may be localized to the scrotal wall or may arise from extensions of infections of intrascrotal contents (i.e., testis, epididymis, and bulbous urethra). A simple abscess of a hair follicle in the scrotal wall can be managed by incision and drainage; no antibiotics are required in immunocompetent patients.
- When a scrotal wall abscess is suspected of coming from an intrascrotal infection, ultrasound and retrograde urethrography may demonstrate pathology in the testis and/or epididymis and urethra, respectively. Definitive care of any complex abscess calls for a urology consultation.
- Fournier's gangrene is a polymicrobial infection of the perineal subcutaneous tissues.[8] Diabetic males are at highest risk. Prompt diagnosis is essential to prevent extensive tissue loss. Early surgical consultation is recommended for at-risk patients who present with scrotal, rectal, or genital pain. Aggressive fluid resuscitation with normal saline solution, broad-spectrum (i.e., gram-positive, gram-negative, and anaerobic) antibiotic coverage, surgical debridement, and hyperbaric oxygen are treatment mainstays.

PENIS

- Balanoposthitis is inflammation of the glans (balanitis) and foreskin (posthitis). Upon foreskin retraction, the glans and prepuce appear purulent, excoriated, malodorous, and tender. Treatment consists of cleansing with mild soap, assuring adequate dryness, application of antifungal creams (nystatin qid or clotrimazole bid), and urologic referral for follow-up and possible circumcision. An oral cephalosporin (e.g., cephalexin 500 mg qid) should be prescribed in cases of secondary bacterial infection.
- Phimosis is the inability to retract the foreskin proximally. Hemostatic dilation of the preputial ostium relieves the urinary retention until definitive dorsal slit or circumcision can be performed.
- Paraphimosis is the inability to reduce the proximal edematous foreskin distally over the glans.[9] Paraphimosis is a true urologic emergency because resulting glans edema and venous engorgement can progress to arterial compromise and gangrene. If surrounding tissue edema can be successfully compressed, as by wrapping the glans with 2 × 2-in. elastic bandages for 5 min, the foreskin may be reduced. Making several puncture wounds with a small (22- to 25-gauge) needle may help with expression of glans edema fluid.[10] Local anesthetic block of the penis is helpful if patients cannot tolerate the discomfort associated with edema compression and removal. If arterial compromise is suspected or has occurred, local infiltration of the constricting band with 1% plain lidocaine followed by superficial vertical incision of the band will decompress the glans and allow foreskin reduction.
- Entrapment injuries occur when various objects are wrapped around the penis. Such objects should be removed, and urethral integrity (retrograde urethrogram) and distal penile arterial blood supply (Doppler studies) should be confirmed when indicated.
- Penile fracture occurs when there is an acute tear of the penile tunical albuginea. The penis is acutely swollen, discolored, and tender in a patient with history of trauma during intercourse accompanied by a snapping sound. Urologic consultation is indicated.
- Peyronie's disease presents with patients noting a sudden or gradual onset of dorsal penile curvature with erections. Examination reveals a thickened plaque on the dorsal penile shaft. Reassurance and urologic follow-up are indicated.
- Priapism is a painful pathologic erection, which may be associated with urinary retention.[11] Infection and impotence are other complications. Regardless of etiology, the initial therapy for priapism is terbutaline 0.25 to 0.5 mg subcutaneously in the deltoid area.[12] Corporal aspiration and irri-

gation with either normal saline solution or an alpha-adrenergic antagonist is the next step and may have to be performed by the emergency physician when urologic consultation is not available. Even when emergency physicians provide stabilizing care, urologic consultation is indicated in all cases.

URETHRA

URETHRAL STRICTURE

- Urethral stricture is becoming more common due to rising incidence of sexually transmitted diseases (STDs). If a patient's bladder cannot be cannulated with a 14 or 16 Fr Foley or Coudé catheter, the differential diagnosis includes urethral stricture, voluntary external sphincter spasm, bladder-neck contracture, or benign prostatic hypertrophy.
- Retrograde urethrography can be performed to delineate the location and extent of urethral stricture. Endoscopy is necessary to confirm bladder neck contracture or define the extent of an obstructing prostate gland.
- Suspected voluntary external sphincter spasm can be overcome by holding the patient's penis upright and encouraging him to relax his perineum and breathe slowly during the procedure.
- After no more than three gentle attempts to pass a 12 Fr Coudé catheter into a urethra prepared with anesthetic lubricant, urology consultation should be obtained.
- In an emergency situation, suprapubic cystotomy can be performed. The infraumbilical and suprapubic area is prepped with povidone-iodine solution. A 25- to 27-gauge spinal needle is used to locate the bladder (ED ultrasound study can be useful at this point), followed by placement of the cystotomy using the Seldinger technique.[13]
- Urologic follow-up should occur within 48 h.

URETHRAL FOREIGN BODIES

- Urethral foreign bodies are associated with bloody urine and slow, painful urination.
- X-ray of the bladder and urethral areas may disclose a foreign body.
- Removal of the foreign body may be achieved with a gentle milking action; retrograde urethrography or endoscopy is required in such cases to confirm an intact urethra.
- Often, urologic consultation for endoscopy or open cystotomy is required for foreign-body removal.

URINARY RETENTION

- Urinary retention syndromes can range from overt retention to insidious overflow incontinence. A detailed history, including over-the-counter cold and diet aids, may reveal the cause of urinary retention.
- Men do not void as completely when sitting down, and infrequent ejaculation may lead to a secondary prostatic congestion and symptoms of outlet obstruction.
- An intact sensory examination, anal sphincter examination, and bulbocavernosus reflex test differentiate chronic outlet obstruction from the sensory or motor neurogenic bladder and spinal-cord compression.
- Physical examination should include search for meatal stenosis, palpation of urethral length for masses or fistulas consistent with urethral stricture disease or abscess formation, lower abdominal examination for palpation of suprapubic mass, and rectal examination to evaluate anal sphincter tone and prostate size and consistency. Most patients with bladder outlet obstruction are in distress, and passage of a urethral catheter alleviates both pain and urinary retention. Copious intraurethral lubrication including a topical anesthetic should be used, and a 16 Fr Coudé catheter is recommended if straight catheters fail. Be certain to pass the catheter to its fullest extent, obtaining free urine flow, before inflating the balloon.
- The catheter should be left indwelling and connected to a leg drainage bag. Belladonna and opium suppositories (one every 4 to 6 h) can be prescribed to alleviate the constant urge to void secondary to bladder spasm, which frequently accompanies an indwelling catheter.
- In patients whose bladder catheter will be left in longer than 5 to 7 days, prophylactic antibiotics (e.g., trimethoprim, 100 mg/day) should be instituted. Otherwise, antibiotics are indicated only if urinalysis is consistent with urinary tract infection.
- If urinary retention has been chronic, postobstructive diuresis may occur even in the presence of normal blood urea nitrogen and creatinine levels. In such patients, close monitoring of urinary output is indicated, and they should be observed for 4 to 6 h after catheterization.

- In all cases of urinary retention, urologic follow-up is indicated for a complete genitourinary evaluation.

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57 RENAL COLIC

Geetika Gupta

EPIDEMIOLOGY

- Stones are three times more common in males and usually occur in the third to fifth decades.[1]
- Children under 16 years of age constitute approximately 7 percent of all cases of renal stones, with a 1:1 sex distribution.[1,2]
- Overall incidence is around 12 percent.[1]
- There is an increased incidence from genetic predisposition and hereditary diseases (e.g., renal tubular acidosis, hyperparathyroidism, cystinuria).[2]
- Lifestyle factors augment stone growth. Increasing water intake results in a decreased incidence of calculi. Patients in mountainous, desert, or tropical regions and those in sedentary jobs suffer a higher frequency of stone disease. Medications such as protease inhibitors and diuretics have also shown an increase in prevalence.[1,3]

PATHOPHYSIOLOGY

- The precise cause of urinary stone formation is unknown.
- Approximately 75 percent of calculi are composed of calcium, occurring in conjunction with oxalate, phosphate, or a combination of both. Calcium excretion is elevated in conditions such as high dietary calcium intake, immobilization syndrome, or hyperparathyroidism. Oxalate excretion is enhanced in patients with inflammatory bowel disease and as a result of small bowel bypass surgery.[1]
- Some 10 percent of stones are made up of magnesium-ammonium-phosphate (struvite). These are associated with infection by urea-splitting bacteria and are the most common cause of staghorn calculi.[1]
- Uric acid causes 10 percent of uroliths, with cystine and other infrequent stones completing the remainder.[1]
- The majority (90 percent) of urinary calculi are radiopaque. Calcium phosphate and calcium oxalate stones have a density similar to that of bone.
- Common areas of impaction include the renal calyx, ureteropelvic junctions, and the ureterovesical junction (UVJ). The UVJ has the smallest diameter of the urinary tract and is the most common location for impacted stones.
- Common etiologies in pediatrics are metabolic abnormality (50 percent), urologic anomalies (20

percent), infection (15 percent), and immobilization syndrome (5 percent).[1]
- Approximately one-third of patients suffer recurrences within 1 year and 50 percent in 5 years.[1]

CLINICAL FEATURES

- Patients are asymptomatic until there is at least partial obstruction.
- Patients complain of the acute onset of severe pain, which can be associated with diaphoresis, nausea, and emesis. During extreme presentations, the patient is anxious, pacing or writhing, and may be unable to hold still or converse.
- Typically pain originates in either flank, radiating ipsilaterally and anteroinferiorly around the abdomen and toward the ipsilateral testicle or labia majora. The radiating pattern is the result of autonomic nerve fibers serving both the kidney and respective gonad. Anterior abdominal pain may radiate back toward the flank and is associated with midureteral stones. Stones near the bladder may cause urinary frequency and urgency.
- Extracorporeal shock wave lithotripsy (ESWL) fractures stones into small particles using focused sound waves. The resulting ''sludge'' is passed in the urine. When there are large fragments, an acute episode of renal colic occurs. The presentation is identical to that of de novo episodes of renal colic.

DIAGNOSIS AND DIFFERENTIAL

- All patients with suspected renal colic require a urinalysis. In 10 percent of the cases, urinary blood is absent.[4] Pyuria indicates the need for a thorough investigation to exclude infection.
- The kidney-ureter-bladder (KUB) radiograph's greatest utility is in the exclusion of other pathologies.
- The ''gold standard'' for diagnosis of renal colic has historically been the intravenous pyelogram (IVP). The IVP yields information regarding renal function as well as anatomic morphology. The first and most reliable indication of the presence of obstruction is a delay in the appearance of the nephrogram. Adjuncts to diagnosis include distention of the renal pelvis, calyceal distortion, dye extravasation, hydronephrosis, and visualization of the entire ureter.
- The sensitivity of an IVP is 64 to 90 percent and the specificity is 94 to 100 percent. A falsely negative IVP occurs infrequently when there is a radiolucent, partially obstructing stone.[5,6]
- Noncontrast helical computed tomography (CT) is the diagnostic procedure of choice in the emergency department (ED). The sensitivity is 95 to 97 percent and the specificity is 96 to 98 percent.[7,8] Advantages of CT include its speed and the fact that it avoids the risk of contrast allergy.[8]
- Positive findings on CT include changes in ureteral caliber, suspicious calcifications, stranding of perinephric fat, and dilation of the collecting system.
- Ultrasound (US) is reserved for patients unable to undergo an IVP or CT. US is not a functional test and provides anatomic information only. It is useful in the detection of hydronephrosis and larger stones (>5 mm) in the proximal and distal ureter.[9]
- A differential diagnosis includes a leaking abdominal aortic aneurysm, incarcerated hernia, epididymitis, testicular torsion, ectopic pregnancy, pyelonephritis, papillary necrosis (sickle cell disease, diabetes, nonsteroidal analgesic abuse, or infection), renal infarction, appendicitis, and musculoskeletal strain. A right ureteral stone can resemble cholecystitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Pain medication including narcotics and nonsteroidal anti-inflammatory drugs should not be delayed pending test results.
- In cases complicated by urinary tract infection (UTI), routine cultures of urine and blood are indicated and renal obstruction must be excluded. Antibiotics should be started promptly while the patient is in the ED. Appropriate intravenous antibiotics include an antipseudomonal cephalosporin, ticarcillin/clavulanate, antipseudomonal penicillin with an antipseudomonal aminoglycoside, a fluoroquinolone, or imipenem cilastatin.
- Hospitalization is required if the patient has an infection with concurrent obstruction, solitary kidney and complete obstruction, uncontrolled pain, or intractable emesis. Disposition should be discussed with a urologist in patients with a stone >6 mm, renal insufficiency, severe underlying disease, IVP with extravasation/complete obstruction, or failed outpatient management.[1]
- Stones with diameters less than 4 mm will pass in 75 percent of cases. Stones 4 to 6 mm in size pass around 50 percent of the time; only 10 percent of stones exceeding 6 mm pass spontaneously. Irreg-

ularly shaped stones with spicules and sharp edges will have a lower pass rate.[1]
- Rates of passage for stones found in the proximal, middle, and distal ureter are approximately 20, 50, and 70 percent, respectively, regardless of stone size.[10]
- Discharge is appropriate in patients with rounded stones <4 to 5 mm, in the absence of infection, and when pain is controlled by oral analgesics.
- Patients need to be counseled to return promptly for fever, vomiting, or uncontrolled pain, and they should receive a prescription for oral narcotics.
- Follow-up with a urologist should be arranged within 5 days.[11]
- All urine should be collected and strained for the identification of any passed stones. Patients whose stones pass in the emergency department require no further treatment.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 92, ''Urologic Stone Disease,'' by Joel Moll and William Franklin Peacock IV.

58 COMPLICATIONS OF UROLOGIC DEVICES

David M. Cline

COMPLICATIONS OF URINARY CATHETERS

- Infection is the most common complication of urinary catheters (10 to 30 percent);[1] management is discussed in Chap. 55.
- Minor traumatic complications of urinary catheters may require no therapy, while major complications (such as bladder perforation) require consultation with a urologist.

NONDRAINING CATHETER

- Obstruction is suggested if the catheter does not flush easily or there is no return of the irrigant. Obstruction of the catheter by blood clots often creates a situation in which the catheter is easily flushed but little or no irrigant is returned. If this occurs, the catheter can be replaced with a triple-lumen catheter so that the bladder can easily be irrigated. If, after clearing the bladder of all clots, evidence of continued bleeding is present, urologic consultation is recommended for possible cystoscopy.
- Some physicians advocate the use of single-lumen catheters to lavage the bladder, as the larger lumen may aid in the evacuation of larger clots.

NONDEFLATING RETENTION BALLOON

- If the obstruction is distal, the result of a crushed or defective valve, the catheter can be cut proximal to the defect. If this does not deflate the balloon, a lubricated guidewire can be introduced into the cut inflation channel in an attempt to clear the obstruction.

- The balloon can be ruptured within the bladder utilizing overinflation with sterile water. This procedure often requires 10 to 20 times the normal balloon volume.
- Urologic consultation may be required if simple measures are not successful.

COMPLICATIONS OF URETERAL STENTS

- Dysuria, urinary urgency, frequency, and abdominal and flank discomfort are common complaints in patients with ureteral stents.[2–4] The baseline discomfort in a functioning, well-positioned stent can range anywhere from minimal to debilitating. However, an abrupt change in the character, location, or intensity of the pain requires further evaluation for stent malposition or malfunction.
- Ureteral stents may remain in place for weeks to months and often function with no complication during the entire period. However, stents can often become encrusted with mineral deposits and may obstruct. Complete obstruction of urine flow is possible, although this tends to occur more often in patients with stents in place for long-term use. These patients may require urologic consultation and in some cases may require stent replacement.

UTI VERSUS STENT MIGRATION/ MALFUNCTION

- Changing abdominal or flank pain or bladder discomfort may be indicative of stent migration. X-ray examination is indicated with comparison to a previous film to evaluate stent position, and urologic consultation with further studies to evaluate stent position may eventually be necessary.
- When a urinary tract infection occurs in the presence of a stent, stent removal is not mandatory because most infections can be managed with outpatient antibiotics. If pyelonephritis or systemic infection is evident, however, then further evaluation and emergent intervention are indicated. Plain x-ray examination to check for stent migration and urologic consultation for evaluation of stent migration and malfunction are indicated as well as initiation of antibiotic therapy.

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Section 10
GYNECOLOGY AND OBSTETRICS

59 VAGINAL BLEEDING AND PELVIC PAIN IN THE NONPREGNANT PATIENT

Cherri D. Hobgood

ABNORMAL VAGINAL BLEEDING

EPIDEMIOLOGY

- Of women aged 20 to 69, up to 1 in 20 report abnormal vaginal bleeding.[1]
- Twenty-five percent of women between the ages of 30 and 49 will consult a physician for treatment of menorrhagia.[2]

PATHOPHYSIOLOGY

- The normal menstrual cycle is 28 days and has four phases: follicular, ovulatory, luteal or secretory, and menses (see Fig. 59-1[3]).
- Menopause is the result of ovarian burnout and occurs at the average age of 51.[4] The perimenopausal period is characterized by marked variation in the intermenstrual period and very high serum levels of FSH and LH as well as low levels of serum estrogen.
- Anovulatory cycles result from an imbalance of follicle degeneration and stimulation. In the presence of an estrogen steady state, the endometrium enters a prolonged proliferative phase and becomes hyperplastic. When the estrogen steady state is insufficient to meet the needs of the hyperplastic endometrium, a relative estrogen insufficiency occurs and the thickened endometrium sloughs. This hormonal pattern produces prolonged periods of amenorrhea with intermittent menorrhagia, which is characteristic of anovulatory cycles.

CLINICAL FEATURES

- Abnormal vaginal bleeding is defined as vaginal bleeding occurring outside the normal menstrual cycle.
- Menorrhagia is defined as menses >7 days or menstruation >60 mL or <21 day recurrence due to any cause.
- Metrorrhagia is defined as irregular vaginal bleeding outside the normal cycle.
- Menometrorrhagia is defined as excessive irregular vaginal bleeding.
- Dysfunctional uterine bleeding is defined as abnormal vaginal bleeding due to anovulation.
- Postcoital bleeding is defined as vaginal bleeding after intercourse, suggestive of cervical pathology.

DIAGNOSIS AND DIFFERENTIAL

- A thorough physical examination may reveal structural or traumatic causes of bleeding; this should include a complete abdominal and pelvic examination. Pregnancy must be excluded. Once the bleeding site is identified, a ranked differential may be formulated utilizing the following etiologies.
- In premenopausal women, bleeding may be due to any of the following causes: cervicitis, endometrial or cervical polyps, cervical or endometrial cancer, submucosal fibroids, local trauma, or retained foreign body.

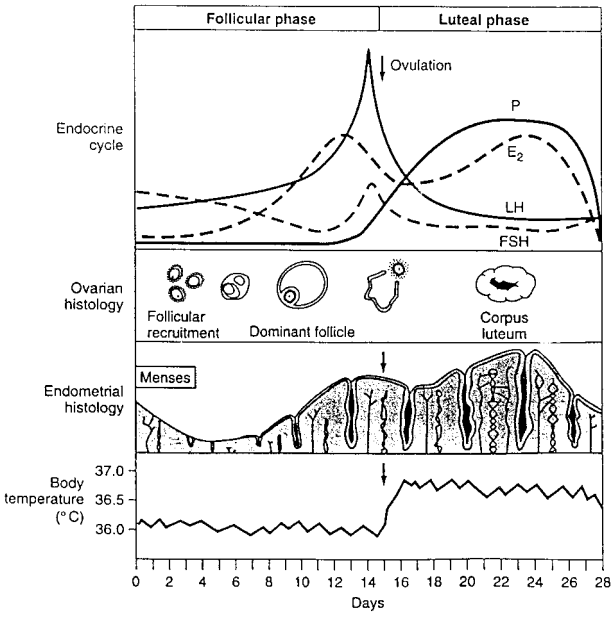

FIG. 59-1 The hormonal, ovarian, endometrial, and basal body temperature changes and relationships throughout the normal menstrual cycle. (From Carr and Wilson,[3] with permission).

- In postmenopausal women the most common causes of vaginal bleeding are exogenous estrogens, atrophic vaginitis, and endometritis, with each accounting for approximately 30 percent of cases. Endometrial cancer is less common and accounts for approximately 15 percent of cases.
- Anovulatory dysfunctional uterine bleeding is likely if the pelvic exam is normal. This is most common in perimenarcheal girls and perimenopausal women who present with prolonged menses or intermenstrual bleeding.
- Primary coagulation disorders—such as von Willebrand's disease, myeloproliferative disorders, and immunothrombocytopenia—are present in 19 percent of teens presenting with menorrhagia. Petechiae or other signs are frequently absent.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Most patients require no immediate intervention.
- Hemodynamically unstable patients will require resuscitation and a gynecology consult for possible dilation and curettage (D&C). Uterine packing should be avoided.
- Hemodynamically stable patients with anovulatory dysfunctional uterine bleeding can be managed with either of the following hormonal therapies:
  1. IV conjugated estrogens 25 mg or oral conjugated estrogens 2.5 mg PO qid. After bleeding subsides, add medroxyprogesterone 10 mg qd. Both medications should be continued for 7 to 10 days.
  2. Oral contraceptive pills: ethinyl estradiol 35 μg and norethindrone 1 mg—4 tablets for 7 days; or slow taper (ethinyl estradiol 35 μg and norethindrone 1 mg) 4 tablets for 2 days, then 3 tablets for 2 days, then 2 tablets for 2 days, then 1 tablet for 3 days.
  3. Progesterone therapy with medroxyprogesterone acetate (Provera) 10 mg/day for 10 days.
- Older patients in whom there is a concern for malignancy should not be started on hormonal therapy but must be referred to a gynecologist for possible endometrial biopsy.
- Teens presenting with menorrhagia should be evaluated with a complete blood count (CBC), coagulation studies, and a bleeding time.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) are useful as adjunctive therapy and serve to decrease both bleeding and painful cramping.

PELVIC PAIN

EPIDEMIOLOGY

- Of reproductive age women, 90 percent report some dysmenorrhea, 38 percent experience dyspareunia, and 39 percent report other pelvic pain.[5]
- Pelvic pain is most common in the 18- to 30-year-old woman. Prevalence does not vary by race, parity, or education.[5]
- Leiomyomas or fibroids are the most common pelvic tumors. They occur in 25 percent of white women and 50 percent of black women; they are frequently multiple. Of women with fibroids 30 percent have pelvic pain and bleeding.

PATHOPHYSIOLOGY

- Pelvic pain may arise from either gynecologic or nongynecologic conditions and may be referred to the back, buttocks, perineum, or legs.
- Visceral pain is colicky and caused by distention of a hollow viscus or stretching of a ligament. Pain of this type is produced by distention of the fallopian tube in ectopic pregnancy, uterine contractions in dysmenorrhea, or stretching of the round ligament with adhesions or in pregnancy.
- Peritoneal or somatic pain is sharp and localized

to the region of inflamed tissue. This pain type is seen in salpingitis, appendicitis, and endometritis. Generalized peritonitis may be seen with large degrees of inflammation—i.e., spillage of blood, pus, or gastrointestinal contents into the peritoneal cavity.

CLINICAL FEATURES

- Ovarian cysts are the most common noninfectious cause of acute pelvic pain. Ovarian cyst enlargement may be asymptomatic or may produce poorly localized visceral pain. When cyst leakage occurs, acute pain develops secondary to peritoneal irritation.
- Follicular cysts are the most common cyst type. Rupture produces the acute onset of sharp pain, which resolves over several days. If cysts are unruptured, regression occurs spontaneously over 1 to 3 months.
- Corpus luteum cysts are less common, and most resolve at the end of the menstrual cycle if pregnancy does not occur. Persistence of the corpus luteum cyst may cause unilateral pelvic pain and menstrual cycle abnormalities. Cyst rupture may cause the acute onset of sharp pain, peritoneal irritation, and bleeding mimicking rupture of an ectopic pregnancy.
- Ovarian torsion is rare. It occurs in the enlarged or abnormal ovary and tumors will be present in up to 50 percent of patients, usually benign dermoid tumors. The ovary twists on its pedicle, compromising its blood supply and subsequently undergoing necrosis. Torsion of tubal masses and pedunculated fibroids present in a similar manner.
- Mittelschmerz is physiologic midcycle pain at ovulation. It occurs on days 14 to 16 of the menstrual cycle. Pain is typically unilateral, mild to moderate, and may last a day or less. Vaginal spotting may occur.
- Dysmenorrhea is the most common cause of midcycle pain.
  1. Primary dysmenorrhea occurs most often in young girls just after menarche. The pain is crampy and may be associated with nausea, backache, and headache.
  2. Secondary dysmenorrhea occurs later in life and is associated with other gynecologic problems such as infection, fibroids, endometriosis, and adhesions.
- Endometriosis is the second most common cause of midcycle pain following dysmenorrhea. Symptoms include pelvic pain, usually at menses; dyspareunia; and dysmenorrhea.
- Leiomyomas or fibroids rarely produce acute pain, but if severe pain is present, torsion of a pedunculated fibroid should be considered. During pregnancy, a fibroid may produce severe pain when its blood supply is outstripped and degeneration occurs.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis of pelvic pain is extensive (Table 59-1).[6]
- A thorough physical examination should be performed, including a complete set of vital signs and an abdominal and gynecologic examination. The location and type of pain produced on exam as well as the presence or absence of masses or ab-

TABLE 59-1 Differential Diagnosis of Acute Pelvic Pain

GYNECOLOGIC DISEASE OR DYSFUNCTION
Acute pain
1. Complication of pregnancy
a. Ruptured ectopic pregnancy
b. Abortion, threatened or incomplete
c. Degeneration of a leiomyoma
2. Acute infections
a. Endometritis
b. Pelvic inflammatory disease (acute PID)
c. Tuboovarian abscess
3. Adnexal disorders
a. Hemorrhagic functional ovarian cyst
b. Torsion of adnexa
c. Twisted parovarian cyst
d. Rupture of functional or neoplastic ovarian cyst
Recurrent pelvic pain
1. Mittelschmerz (midcycle pain)
2. Primary dysmenorrhea
3. Secondary dysmenorrhea
Gastrointestinal
1. Gastroenteritis
2. Appendicitis
3. Bowel obstruction
4. Diverticulitis
5. Inflammatory bowel disease
6. Irritable bowel syndrome
Genitourinary
1. Cystitis
2. Pyelonephritis
3. Ureteral lithiasis
Musculoskeletal
1. Abdominal wall hematoma
2. Hernia
Other
1. Acute porphyria
2. Pelvic thrombophlebitis
3. Aneurysm
4. Abdominal angina

SOURCE: From Rapkin,[6] with permission.

normalities in the organs of reproduction will guide the formulation of the differential diagnosis.
- Laboratory evaluation should consist of pregnancy tests in all women of childbearing age and a complete blood count. If indicated by the history and physical examination, consideration should be given to coagulation studies and/or specific endocrine evaluations.
- Ultrasound is very useful in determining adnexal pathology, free fluid in the pelvis, and the thickness of the endometrium. Leiomyomas, ovarian cysts, hydrosalpinx, pelvic adhesions, tuboovarian abscesses, endometriosis, and ovarian carcinoma may all be visualized by this method.
- Computed tomography or magnetic resonance imaging is less useful than ultrasound in this setting; its value lies in the diagnosis of nongynecologic lesions and in cancer staging.
- Laparoscopy and/or laparotomy may be required in the diagnosis of pelvic pain if the etiology is uncertain or direct visualization of an ambiguous adnexal mass is required. It may also be required to make the final diagnosis in ovarian torsion and endometriosis.
- Chronic pelvic pain conditions are rarely diagnosed primarily in the ED; however, these pain syndromes are frequently associated with somatization disorders in women with a history of sexual abuse and/or physical assault[6] and should be considered in women presenting with these conditions.
- The diagnosis of mittelschmerz is clinical; more serious etiologies should be ruled out by physical examination and a pregnancy test. Extensive evaluation is unwarranted.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The emergency department (ED) treatment for the majority of patients with pelvic pain is analgesia and gynecologic follow-up. Leiomyomas, endometriosis, mittelschmerz, secondary dysmenorrhea, and chronic pelvic pain may all be treated in this manner.
- Ovarian cysts are treated primarily with analgesia and follow-up if unruptured. If the cyst has ruptured and the patient is hemodynamically stable, the same treatment protocol maybe used. If rupture of a corpus luteum cyst produces hemoperitoneum, surgical intervention may be required.
- Ovarian torsion requires surgical intervention for adnexal detorsion or removal of the abnormal ovary.
- Primary dysmenorrhea should be treated first with a trial of NSAIDs. The second-line therapy is oral contraceptives.

PREPUBERTAL CHILDREN

EPIDEMIOLOGY

- Of vaginal bleeding in prepubertal children, 21 percent of such cases are associated with precocious puberty, 54 percent are associated with other etiologies, and 24 percent are idiopathic.
- Ten years of age is the lower limit for menarche; the mean age in North America is 12.5 years. The average time required to establish ovulatory cycles is 2 years after menarche.
- Genital trauma represents 0.2 percent of all injuries in children younger than 15. The most common mechanisms of injury are bicycle accidents, straddle injuries, and falls. The labia majora is most commonly injured. The majority of injuries are superficial, with only 5 percent of children requiring surgical repair.[7]
- Imperforate hymen is found in 1 of 1000 term neonates. Transverse vaginal septum is found in 1 in 2000 to 1 in 84,000 women.
- Urethral prolapse occurs most frequently between the ages of 2 and 10; it is most common in black children.

PATHOPHYSIOLOGY

- In newborn females, the placental transfer of the maternal hormones estradiol and gonadotrophin is responsible for minor breast development and blood-tinged vaginal discharge. Normal neonates may experience uterine bleeding in the first 6 weeks of life secondary to maternal estrogen withdrawal.
- Secondary sex characteristics develop on average 2 years prior to menarche. Any variation of this is pathologic, and a specific etiology must be sought.

CLINICAL FEATURES

- Vaginitis is the most common cause of pelvic pain and bleeding in prepubertal children. Flora is generally *Staphylococcus epidermidis* and diptheroids, *Lactobacillus* is not found.
- Trauma to the perineum can produce ecchymoses and/or lacerations that may be associated with

injury to the vagina, urethra, and rectum. The hypoestrogenic skin of the vagina tears easily, and there is a significant risk of wall perforation with any penetrating injury to the vagina and rectum.
- Vaginal foreign bodies generally present with foul-smelling vaginal discharge, which may be bloody. Toilet paper is the most common foreign body.
- Congenital vaginal obstruction may be due to transverse vaginal septum or imperforate hymen. These typically present as abdominal or perineal masses or complaints of difficulty urinating. More severe cases may present with constipation, hydronephrosis, respiratory compromise, and lower extremity edema.
- Precocious puberty with or without menarche may occur in children aged 5 to 9. Premature menarche in prepubertal children without the development of secondary sexual characteristics may also occur.
- Urethral prolapse presents as a soft spongy mass 1 to 2 cm in diameter with a central dimple at the urethral meatus. Vaginal bleeding is the initial complaint in 90 percent of cases; 25 percent of cases present with dysuria or frequency.
- Seborrhea and psoriasis may present with bleeding after minor trauma. Lichen sclerosus appears as an hourglass-shaped depigmented area on the vulva and perineal and adjacent skin. The skin is atrophic and thin, with tiny ivory papules that coalesce. The patches are frequently dry and itchy.

DIAGNOSIS AND DIFFERENTIAL

- Prepubertal children presenting with vaginal bleeding require a thorough history as to the circumstances of bleeding, times of occurrence, associated symptoms including pain, and possible exposure to diethylstilbestrol (DES) or sexual abuse.
- The physical exam in prepubertal children should include a careful assessment of subtle signs of disease, injury, and/or abuse; the Tanner stages of sexual development should be noted. Speculum exam and vaginoabdominal exam should not be performed unless vaginal trauma or bleeding is present. If performed, anesthesia should be utilized. If a pelvic mass or foreign body is suspected, rectoabdominal examination with the child in the frog-leg position should be performed.
- Diagnoses of congenital vaginal malformations are made by careful examination of the perineum. The diagnosis may be unsuspected until the patient develops difficulty with urination or an abdominal or a perineal mass develops and prompts evaluation.
- Urethral prolapse may be differentiated from vaginal masses by observing the child urinate on a bedpan.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Care of traumatic injuries to the perineum should be based on the extent of injury. Hematomas can spread liberally and should be observed until expansion has stopped. All penetrating injuries require a careful vaginal and rectal examination. Any traumatic bleeding should be referred to a pediatric gynecologist for examination under anesthesia.
- Removal of vaginal foreign bodies may be attempted by irrigation of the vaginal vault with warm water or by milking hard objects from the vagina via the rectum. Failure to remove the object should prompt gynecologic consultation for removal under anesthesia.
- Congenital vaginal obstruction is treated surgically. The urgency of referral depends upon presenting symptoms. Urologic, fecal, or vascular compromise should prompt emergent referral.
- Precocious puberty and menarche as well as premature menarche require referral to a pediatrician after other serious causes of vaginal bleeding have been ruled out.
- Urethral prolapse is best treated with sitz baths and estrogen creams. If the mucosa is red or necrotic, surgical intervention may be required.
- Mild forms of lichen sclerosus may be treated with sitz baths and 1% hydrocortisone cream.

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60 ECTOPIC PREGNANCY

Karen A. Kinney

EPIDEMIOLOGY

- Ectopic pregnancy (EP) occurs in 2 percent of all pregnancies.[1]
- Ectopic pregnancy is more common in nonwhite women over the age of 35.[1]
- Ninety five percent of EPs occur in the fallopian tube. Other sites include the abdominal cavity, ovary, and cervix.
- Pelvic inflammatory disease is the most common risk factor. Other risk factors include tubal ligation and other surgical procedures of the fallopian tubes; previous EP; abortion; current use of an intrauterine device; peritubular adhesions from appendicitis or endometriosis; treatment with infertility drugs; and exposure to diethylstilbestrol.

PATHOPHYSIOLOGY

- Ectopic pregnancy is postulated to be caused by (a) mechanical or anatomic alterations in the tubal transport mechanism, or (b) functional/hormonal factors that alter the fertilized ovum.
- Tubal rupture is thought to be spontaneous, but trauma associated with coitus or a bimanual examination may precipitate tubal rupture. Tubal rupture may occur in the early weeks of an EP or as late as 16 weeks estimated gestational age.

CLINICAL FEATURES

- The classic triad is abdominal pain, a positive pregnancy test, and vaginal bleeding which is usually light.
- Abdominal pain occurs in 90 percent of patients presenting with EP. Vaginal bleeding occurs in 80 percent of these patients. Of the women with EP, 70 percent give a history of amenorrhea.[2]
- Vital signs may be normal or may indicate hemorrhagic shock. A relative bradycardia may be present in the patient with tubal rupture and hemorrhage.[2,3]
- Referred pain to the shoulder or upper abdomen may occur in the presence of hemoperitoneum causing diaphragmatic irritation.
- Physical findings are highly variable; from a normal pelvic exam to cervical motion tenderness; adnexal tenderness, with or without mass; and sometimes an enlarged uterus. The abdominal exam may be entirely normal, or there may be localized or diffuse tenderness. Peritoneal signs may or may not be present. Rarely, fetal heart sounds are audible.[2,3]

DIAGNOSIS AND DIFFERENTIAL

- A urine pregnancy test (UCG) should be performed immediately. A negative result rules out EP. A positive UCG or qualitative serum β human chorionic gonadotropin (β-HCG) implies a quantitative serum β-HCG level of $\geq$10 mLU/mL.[4]
- Pelvic ultrasound is the test of choice for identifying EP. If an intrauterine pregnancy (IUP) is identified, the chance of a coexisting EP is extremely rare in most patients. However, women who have been on fertility drugs, or who have undergone in vitro fertilization, or who have multiple risk factors for EP should have further evaluation.[5]
- Sonographic findings of an empty uterus with an adnexal mass with or without free abdominal fluid is highly suggestive of EP.[7]
- Sonographic findings of an empty uterus without free fluid or adnexal mass in the presence of a positive pregnancy test are considered indeterminate. These findings must be evaluated in context with the patient's serum quantitative β-HCG level.
- A serum quantitative β-HCG level >6000 with

an empty uterus seen on ultrasound is suggestive of EP.[6,7]

- A quantitative β-HCG level <1500 indicates that a pregnancy may be ectopic or intrauterine, but it is too small to be visualized by ultrasound. A repeat quantitative β-HCG test should be performed in 48 h in this case. Most normal IUPs will show at least a 66 percent increase in the β-HCG level in 48 h. An EP usually shows a slower rate of increase in the β-HCG level.[8,9]
- A serum quantitative β-HCG level between 1500 and 6000 may warrant dilation and curettage or laparoscopy by a consulted obstetrician-gynecologist to diagnose EP.[8,9]
- Disorders in the differential diagnosis of women of childbearing age presenting with abdominal pain include EP; appendicitis; inflammatory bowel disease; ovarian pathology, including cyst or torsion; pelvic inflammatory disease; endometriosis; sexual assault/trauma; urinary tract infection; or ureteral colic.
- Disorders in the differential diagnosis in women presenting with early pregnancy, abdominal pain, and vaginal bleeding include a normal IUP; EP; threatened, incomplete, or missed abortion; recent elective abortion; endometritis; molar pregnancy; or heterotopic pregnancy.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- For unstable patients, two large-bore intravenous lines should be initiated for rapid infusion of crystalloid and/or packed red blood cells to maintain an adequate blood pressure.
- For the unstable patient an immediate obstetric-gynecologic consult should be obtained, even before laboratory and diagnostic tests are complete.
- Blood should be drawn and the following laboratory studies ordered: complete blood cell count; blood typing and Rh factor determination; crossmatching for unstable patients; quantitative β-HCG level, if indicated; and serum electrolyte determinations.
- For the stable patient, the diagnostic evaluation should be continued. A reliable patient with a low quantitative β-HCG level and an indeterminate sonogram may be discharged from the emergency department with EP precautions and arranged follow-up in 2 days with obstetric-gynecologic reevaluation and a repeat quantitative β-HCG level.
- Definitive treatment determined by the obstetric-gynecologic consultant may include laparoscopy, dilation and curettage, or medical management with methotrexate.

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61 EMERGENCIES DURING PREGNANCY AND THE POSTPARTUM PERIOD

Cynthia Madden

- The leading causes of maternal death are pulmonary embolus (see Chap. 28), ectopic pregnancy

(see Chap. 60), pregnancy-induced hypertension, hemorrhage, and infection.

EMERGENCIES DURING THE FIRST HALF OF PREGNANCY

VAGINAL BLEEDING

- The differential diagnosis of vaginal bleeding during the first trimester should include abortion (most common cause), ectopic pregnancy (see Chap. 60), and gestational trophoblastic disease.
- Inevitable abortion will occur with vaginal bleeding and dilatation of the cervix.
- Incomplete abortion is defined as passage of parts of the products of conception and is more likely between 6 and 14 weeks of pregnancy. These patients require admission for dilatation and curettage.
- Threatened abortion is vaginal bleeding with a closed cervical os and benign physical examination.
- Complete abortion is passage of all fetal tissue before 20 weeks' conception.
- Missed abortion is fetal death at less than 20 weeks without passage of fetal tissue.
- Septic abortion is evidence of infection during any stage of abortion.
- A pelvic exam should be performed and a complete blood cell count (CBC) obtained, with blood typing and Rh factor determination, quantitative β-human chorionic gonadotropin (β-HCG), and urinalysis. Rh-negative women should receive 300 μg of Rh (D) immune globulin.
- Vaginal ultrasound should reveal a gestational sac in a normal pregnancy with a β-HCG >2000. Absence of a gestational sac with a β-HCG >2000 suggests spontaneous abortion or ectopic pregnancy.[1]
- Gestational trophoblastic disease is a neoplasm that arises in the trophoblastic cells of the placenta. The noninvasive form of the disease is the hydatidiform mole. Treatment is by suction curettage in the hospital, with subsequent monitoring of β-HCG levels.

NAUSEA AND VOMITING OF PREGNANCY

- Intractable nausea and vomiting without significant abdominal pain can cause hypokalemia or ketonemia (hyperemesis gravidum) and may result in a low birth weight infant.
- Diagnostic workup should include a CBC, electrolyte panel, and urinalysis. Treatment consists of rehydration with intravenous (IV) fluid 5% dextrose in normal saline solution or in lactated Ringer's, along with antiemetics.

EMERGENCIES DURING THE SECOND HALF OF PREGNANCY

VAGINAL BLEEDING DURING THE SECOND HALF OF PREGNANCY

- Common causes include abruptio placentae, placenta previa, and premature rupture of membranes. Pelvic speculum and digital examination should not be performed until ultrasound has definitively ruled out placenta previa as the cause of bleeding.
- Obtain emergent obstetrical consultation, CBC, type and cross-matching, disseminated intravascular coagulation profile, and electrolyte studies on all patients.
- Administer IV crystalloid fluid and/or packed red blood cells for hemodynamically unstable patients. RhoGam 300 μg should be given to Rh negative patients.

ABRUPTIO PLACENTAE

- Abruptio placentae is the premature separation of the placenta from the uterine wall.
- Risk factors include hypertension, advanced maternal age, multiparity, smoking, cocaine use, previous abruption, and abdominal trauma.
- Clinical features include vaginal bleeding, abdominal pain, and uterine tenderness.
- Emergency delivery may be needed to save the life of the fetus and/or mother.

PLACENTA PREVIA

- Placenta previa is the implantation of the placenta over the cervical os.
- Risk factors include multiparity and prior cesarean section.
- Clinical features are painless bright red vaginal bleeding. Diagnosis is made by ultrasound, not digital exam.

PREMATURE RUPTURE OF MEMBRANES (PROM)

- Premature rupture of membranes (PROM) is rupture of membranes prior to the onset of labor.

- Clinical presentation is a rush of fluid or continuous leakage of fluid from the vagina.[2]
- Diagnosis can be confirmed by identifying a pool of fluid in the posterior fornix with pH greater than 6.5 (dark blue on nitrazine paper) and ferning pattern on smear.
- Tests for chlamydia, gonorrhea, bacterial vaginosis, and group B streptococcus should be performed.
- Patients with suspected PROM should be admitted.

PRETERM LABOR

- Preterm labor is defined as labor prior to 37 weeks' gestation. It occurs in 10 percent of deliveries and is the leading cause of neonatal deaths.
- Risk factors include PROM, abruptio placentae, drug abuse, multiple gestation, polyhydramnios, cervical incompetence, uterine abnormalities, prior preterm labor, and infection.
- Clinical features include regular uterine contractions with cervical changes of effacement. The diagnosis is made by observation with external fetal monitoring and serial sterile speculum examinations.
- Emergency obstetrical consultation should be obtained for admission and for decision regarding tocolytics. If tocolytics are initiated, the mother should receive glucocorticoids to hasten fetal lung maturity.[3]
- The risk of group B streptococcus is higher in preterm infants—mothers should receive 5 million U penicillin G IV.[4]

HYPERTENSION, PREECLAMPSIA, AND RELATED DISORDERS

- Hypertension with pregnancy is associated with preeclampsia, eclampsia, HELLP (hemolytic anemia, elevated liver enzymes, and low platelets) syndrome, abruptio placentae, preterm birth, and low birth weight infants.
- Hypertension in pregnancy is defined as a blood pressure >140/90, a 20-mmHg rise in systolic blood pressure, or a 10-mmHg rise in diastolic blood pressure above the prepregnancy level.

PREECLAMPSIA

- Preeclampsia complicates 7 percent of pregnancies. Risk factors include primigravida and a family history of preeclampsia.
- Clinical presentation is hypertension, proteinuria, and edema.[5] Patients may present with headache, visual disturbances, or abdominal pain. Eclampsia is preeclampsia with seizures.
- HELLP syndrome presents usually with abdominal pain, and hypertension may not be present initially. Diagnosis is made by lab tests: schistocytes on peripheral smear, platelet count less than 150,000/μL, elevated AST (aspartate aminotransferase) and ALT (alanine aminotransferase) levels, and abnormal coagulation profile.
- Treat with $MgSO_4$ loading dose 4 to 6 g in 100 mL of fluid over 20 min, followed by maintenance infusion of 2 g/h to prevent seizures. Treat with hydralazine 2.5 mg initially, followed by 5 to 10 mg every 10 min IV or labetalol 20 mg IV initial bolus, with repeat boluses of 40 to 80 mg if needed to a maximum of 300 mg for blood pressure control.

EMERGENCIES DURING THE POSTPARTUM PERIOD

- Hemorrhage and infection are the most common postpartum complications presenting to the emergency department (ED). Postpartum preeclampsia or eclampsia, amniotic fluid embolism, and postpartum cardiomyopathy are rare but life-threatening complications.

HEMORRHAGE

- The differential diagnosis of hemorrhage includes uterine atony (most common), uterine rupture, laceration of the lower genital tract, retained placental tissue, uterine inversion, and coagulopathy.
- Diagnose by physical examination: the uterus is enlarged and ''doughy'' with uterine atony, a vaginal mass is suggestive of an inverted uterus. Bleeding in spite of good uterine tone and size may indicate retained products of conception.
- ED management consists of stabilization with crystalloid IV fluids and/or packed red blood cells if needed. Uterine atony is treated with oxytocin 20 U in 1 L of IV fluids at 200 mL/h. Minor lacerations can be repaired using local anesthetic. Extensive lacerations, retained products of conception, uterine inversion, or uterine rupture require emergency evaluation and operative treatment by the obstetrician.

INFECTION

- Postpartum endometritis infections are usually polymicrobial. Risk factors include obesity, diabetes, and hypertension.
- Clinical features include fever, malaise, lower abdominal pain, and foul-smelling lochia.
- Diagnosis is made by physical examination revealing uterine fundus tenderness, cervical motion tenderness, and purulent discharge. Laboratory tests include a CBC, urinalysis, and cervical cultures.
- Patients should be admitted for broad-spectrum antibiotic treatment, such as cefotaxime 1 to 2 g IV every 6 h or combination therapy with ampicillin 1 g IV q 6 h and gentamicin 1.5 mg/kg IV q 8 h.

MASTITIS

- Mastitis is cellulitis of the periglandular breast tissue. Treatment is with cephalexin 500 mg qid. Patients should continue nursing on the affected breast.

AMNIOTIC FLUID EMBOLISM

- Amniotic fluid embolism is a sudden, catastrophic illness of unknown cause with mortality rates of 60 to 80 percent. Clinical features include sudden cardiovascular collapse with hypoxemia. Care is supportive.[6]

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62 COMORBID DISEASES IN PREGNANCY

Cynthia Madden

- For information on hypertension in pregnancy, see Chap. 61. For information on pulmonary embolism, see Chap. 28.

DIABETES

- Those with diabeties are at increased risk for hypertensive diseases, preterm labor, spontaneous abortion, pyelonephritis, fetal demise, hypoglycemia, and diabetic ketoacidosis.
- Oral hypoglycemic agents are contraindicated. Insulin requirements increase throughout the pregnancy from 0.7 U/kg/d to 1.0 U/kg/d at term.
- Diabetic ketoacidosis and hypoglycemia are treated the same in pregnant and nonpregnant patients.

HYPERTHYROIDISM

- Hyperthyroidism in pregnancy can increase the risk of preeclampsia and neonatal morbidity. Propylthiouracil (PTU) is the treatment of choice.
- Thyroid storm presents with fever, volume depletion, and cardiac decompensation and has a high mortality rate. Propylthiouracil, along with sodium iodide and propranolol (unless cardiac failure is present), can control symptoms.

DYSRHYTHMIAS

- Dysrhythmias are encountered rarely in pregnancy.

- Lidocaine, digoxin, procainamide, and verapamil are safe in pregnancy.[1]
- Beta-blockers may be used acutely for control but not for long-term use.
- Cardioversion has not been shown to be harmful to the fetus. If anticoagulation is needed, heparin is the drug of choice.

THROMBOEMBOLISM

- The incidence of deep venous thrombosis in pregnancy ranges between 0.5 and 0.7 percent. Factors associated with increased risk include advanced maternal age, increasing parity, multiple gestation, operative delivery (13- to 16-fold increase compared to vaginal delivery), bed rest, obesity, and blood dyscrasias.
- Diagnosis may be made by impedance plethysmography and technetium-99m radionuclide venography. Ventilation and perfusion scanning can be performed in pregnancy. Iodine-125 fibrinogen scanning should not be used.
- Treatment of DVT and pulmanary embolism is with heparin; warfarin is contraindicated. (See Chap. 31.)

ASTHMA

- Clinical features, diagnosis, and management are similar in pregnant and nonpregnant patients. Clinical presentation includes cough, wheezing, and dyspnea.
- Peak expiratory flow rates are unchanged in pregnancy.[2] However, the normal P_{CO_2} on the arterial blood gas values is 27 to 32 with a normal pH of 7.40 to 7.45.
- Acute therapy includes β_2 agonists such as albuterol via nebulizer. Intravenous (IV) methylprednisolone and oral prednisone can be used in pregnancy. Epinephrine 0.3 mL (1:1000 dilution) can be given subcutaneously. Oxygen should be administered to maintain a P_{O_2} of >65 mmHg. Fetal monitoring should be done after 20 weeks.
- Decision making regarding intubation or admission is similar in pregnant and nonpregnant patients.

URINARY TRACT INFECTIONS

- Urinary tract infection is the most common bacterial infection during pregnancy.
- Simple cystitis may be treated with 7 to 10 days of nitrofurantoin, amoxicillin, or cephalexin.
- Patients with pyelonephritis should be admitted for IV antibiotics because of increased risk of preterm labor. Intravenous hydration and antibiotics—cefazolin, or ampicillin and gentamicin—should be used.
- Quinolones are contraindicated during pregnancy.

INFLAMMATORY BOWEL DISEASE

- The general treatment of the pregnant patient with inflammatory bowel disease is the same as that of the nonpregnant patient. Antidiarrheal drugs including codeine, opium, paregoric, and Lomotil may be safely used. Sulfasalazine, in combination with folic acid supplements, may also be used.

SICKLE CELL DISEASE

- Women with sickle cell disease are at higher risk for miscarriage, preterm labor, and vasoocclusive crises.
- Clinical features, evaluation, and treatment are similar in pregnant and nonpregnant patients. Management includes aggressive hydration and analgesic therapy. Narcotics should be used; nonsteroidal anti-inflammatory agents should be avoided after 32 weeks' gestation.
- Aplastic crises are rare but are associated with parvovirus infection and hydrops fetalis.

MIGRAINE

- Treatment includes acetaminophen and narcotics.
- Ergot alkaloids should not be used.

SEIZURE DISORDERS

- Management of a pregnant patient with a known seizure disorder is similar to a nonpregnant patient. Valproic acid is avoided because of an association with neural tube defects.
- Status epilepticus with prolonged maternal hyp-

TABLE 62-1 Drug Use in Pregnancy

DRUG	CATEGORY*	COMMENT
Antibiotics		
Cephalosporins	B	May use
Penicillins	B	May use
Erythromycin	B	Estolate salt contraindicated due to hepatotoxicity; otherwise may use
Azithromycin	B	May use
Nitrofurantoin	B	May use
Clindamycin	B	May use
Metronidazole	B	Should be avoided during first trimester
Isoniazid	C	May use when necessary
Ethambutol	B	May use
Antivirals		
Acyclovir	C	May use in life-threatening maternal illness
Antihypertensive agents		
Alpha-methyldopa	B	May use
Beta blockers	B, C	May use when necessary
Calcium channel blockers	C	May use when necessary
Prazosin	C	May use when necessary
Hydralazine	C	Widely used
Anticonvulsants		
All	C, D	Congenital malformations reported with all anticonvulsants, but benefits may outweigh risks; use of folic acid (1 mg/d) may help prevent teratogenesis; valproic acid has especially high risk of neural tube defects
Corticosteroids	C	May be used in pregnancy for serious maternal conditions; gestational diabetes may develop
Anticoagulants		
Heparin	C	Drug of choice for pregnant women requiring anticoagulation
Analgesics		
Acetaminophen	A	May use
Propoxyphene	C	Caution advised when used close to term; neonatal withdrawal may occur
Opiates	C	Caution advised when used close to term; neonatal withdrawal may occur; avoid aspirin combinations
Nonsteroidal anti-inflammatory drugs	B, C, D	May be used for short duration (48–72 h) and not at all after 32 weeks; ibuprofen widely used
Antiemetics		
Meclizine	B	May use
Dimenhydrinate	B	May use
Diphenhydramine	B	Avoid first trimester
Trimethobenzamide	C	Used widely
Phenothiazines	C	Used widely
Over-the-counter cold medications		
Pseudoephedrine	C	Topical sprays preferable
Phenylpropanolamine	C	Topical nasal sprays preferable
Vaccines		
Live vaccines (measles-mumps-rubella)	X	Contraindicated
Inactivated viral vaccines (rabies, hepatitis B, influenza)	C	May be given
Pneumococcal vaccine	C	May be given
Tetanus and diphtheria	C	May be given

* Categories: A = safe, human studies; B = presumed safe, animal studies; C = uncertain safety, animal studies show an adverse effect: D = unsafe, use may be justifiable in certain circumstances; X = contraindicated.

oxia and acidosis has a high mortality rate for the mother and infant and should be treated aggressively with early intubation and ventilation. The patient should be placed in the left lateral position to maximize placental oxygenation.

HIV INFECTION

- All pregnant HIV-infected women beyond 14 weeks' gestation should be on zidovudine therapy to reduce the risk of transmission to the fetus.[3]

- Patients with CD4 counts <200 should take prophylaxis for *Pneumocystis carinii.* Treatment of opportunistic infections is unchanged in pregnancy.

SUBSTANCE ABUSE

- Cocaine use is associated with increased incidence of fetal death in utero, placental abruption, preterm labor, premature rupture of membranes, spontaneous abortion, intrauterine growth restriction, and fetal cerebral infarcts. Treatment of toxicity is unchanged in pregnancy.
- Opiate withdrawal in pregnant women is treated with methadone or clonidine.
- Alcohol use contributes to increased rates of spontaneous abortion, low birth weight infants, preterm deliveries, and fetal alcohol syndrome. Acute withdrawal is treated with short-acting barbiturates.

DOMESTIC VIOLENCE

- Approximately 15 percent of pregnant women are victims of domestic violence.[4] They are at risk for placental abruption, uterine rupture, preterm labor, and fetal fractures.

DRUG USE DURING PREGNANCY

- Table 62-1 provides general recommendations regarding drug use during pregnancy.

DIAGNOSTIC IMAGING IN PREGNANCY

- Risk of radiation exposure varies with gestational age. The second- to the eighth-week postconception is the period of organogenesis. Neurologic development occurs between weeks 8 and 15.
- The threshold for human teratogenesis is 10 rad, and the fetus is most vulnerable at 8 to 15 weeks' gestation.
- Ultrasound, ventilation/perfusion scanning, and magnetic resonance imaging have not shown any teratogenic effects.

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63 EMERGENCY DELIVERY

David M. Cline

EVALUATING THE PREGNANT PATIENT

- Every patient presenting with signs of active labor should receive immediate monitoring of maternal vital signs and fetal heart rate. Maternal blood pressure should be monitored, and Doppler heart tones are helpful to confirm normal fetal heart rate (120 to 160 beats per minute). A persistently slow fetal heart rate (less than 100 beats per minute) is an indicator of fetal distress, and emergent obstetric consultation is necessary.
- False labor is characterized by irregular, brief contractions usually confined to the lower abdomen. These contractions, commonly called Braxton-Hicks contractions, are irregular in both intensity and duration.
- True labor is characterized by painful, regular contractions of steadily increasing intensity and duration, leading to progressive cervical dilatation. True labor typically begins in the fundal region and upper abdomen and radiates into the pelvis and lower back.
- Patients without vaginal bleeding should be examined both bimanually and with a sterile speculum. Patients presenting with vaginal bleeding should

initially be evaluated with ultrasound prior to any speculum or bimanual examination to rule out placenta previa.[1]
- If spontaneous rupture of membranes (SROM) is suspected, examination with a sterile speculum should be performed and digital exam avoided, as studies have shown an increased risk of infection after a single digital examination.[2]
- Determining whether membranes have ruptured is an important predictor of the likelihood of imminent labor as well as the potential for complications such as infection or cord prolapse.[3] SROM occurs during the course of active labor in most patients, although it may occur prior to the onset of labor in 10 percent of third-trimester patients.
- SROM typically occurs with a gush of clear or blood-tinged fluid. It can be confirmed by using nitrazine paper to test residual fluid in the fornix or vaginal vault while a sterile speculum examination is performed. Amniotic fluid has a pH of 7.0 to 7.4 and will turn nitrazine paper dark blue. Vaginal fluid typically has a pH of 4.5 to 5.5 and will make the nitrazine strip remain yellow.

PLACENTA PREVIA

- Placenta previa occurs when the placenta partially or completely overlies the internal cervical os. The presence of placenta previa should be suspected in any third-trimester patient presenting with painless vaginal bleeding, particularly bright red blood per vagina.
- If previa is suspected, an emergent ultrasound prior to speculum or bimanual examination is required.[4] If previa is present on ultrasound and the patient is actively laboring, no further examination should be performed and arrangements should be made for immediate transport to labor and delivery for cesarean section.

PLACENTAL ABRUPTION

- Abruptio placenta (or placental abruption) is the separation of the placenta from its implantation site prior to delivery.
- Placental abruption is classically characterized by vaginal bleeding, a ''rock-hard'' painful uterus, and fetal distress (decrease in fetal heart rate to $<$100 beats per minute).[5]
- Risk factors for abruption include maternal hypertension, smoking, cocaine use, and trauma.

EMERGENCY DELIVERY

- The use of routine episiotomy for a normal spontaneous vaginal delivery has been discouraged in recent years and increases the incidence of third- and fourth-degree lacerations at the time of delivery.[6,7]
- If an episiotomy is necessary, it may be performed as follows. A solution of 5 to 10 mL of 1% lidocaine is injected with a small-gauge needle into the posterior fourchette and perineum. While protecting the infant's head, a 2- to 3-cm cut is made with scissors to extend the vaginal opening. The incision must be supported with manual pressure from below, taking care not to allow the incision to extend into the rectum.
- Control of the delivery of the neonate is the major challenge. As the infant's head emerges from the introitus, the physician should support the perineum with a sterile towel placed along the inferior portion of the perineum with one hand while supporting the fetal head with the other. Mild counterpressure is exerted to prevent the rapid expulsion of the fetal head, which may lead to third- or fourth-degree perineal tears.
- As the infant's head presents, the left hand may be used to control the fetal chin while the right remains on the crown of the head, supporting the delivery. This controlled extension of the fetal head will aid in the atraumatic delivery. The mother is then asked to breathe through contractions rather than bearing down and attempting to push the baby out rapidly.
- Immediately following delivery of the infant's head, the infant's nose and mouth should be suctioned. This is particularly important in infants presenting with meconium, in order to prevent aspiration. A simple bulb will assist in the routine clearing of the infant's nose and mouth.
- After suctioning, the neck should be palpated for the presence of a nuchal cord. This is a common condition, found in 25 percent of all cephalad-presenting deliveries. If the cord is loose, it should be reduced over the infant's head; the delivery may then proceed as usual. If the cord is tightly wound, it may have to be clamped in the most accessible area by two clamps in close proximity and cut to allow delivery of the infant.
- After delivery of the head, the head will restitute, or turn to one side or the other. As the head rotates, the physician's hands are placed on either side of it, providing gentle downward traction to deliver the anterior shoulder. The physician's hand then gently guides the fetus upward, deliv-

ering the posterior shoulder and allowing the remainder of the infant to be delivered.
- It is useful to prepare for the delivery by placing the posterior (left) hand underneath the infant's axilla prior to delivering the rest of the body. The anterior hand may then be used to grasp the infant's ankles and ensure a firm grip.
- The infant is then loosely wrapped in a towel and stimulated as it is dried. The umbilical cord is double clamped and cut with sterile scissors; the infant is then further dried and warmed in an incubator, where postnatal care may be provided and Apgar scores calculated at 1 and 5 min after delivery. Scoring includes general color, tone, heart rate, respiratory effort, and reflexes.

COMPLICATIONS OF DELIVERY

CORD PROLAPSE

- In the event that the bimanual examination reveals a palpable, pulsating cord, the examiner's hand should not be removed but rather should be used to elevate the presenting fetal part to reduce compression of the cord.[8]
- Immediate obstetric assistance is then necessary, as a cesarean section is indicated. The examiner's hand should remain in the vagina, in order to prevent further compression of the cord by the fetal head, while the patient is transported and prepped for surgery.[9]

SHOULDER DYSTOCIA

- Shoulder dystocia is first recognized after the delivery of the fetal head, when routine downward traction is insufficient to deliver the anterior shoulder. After delivery of the infant's head, the head retracts tightly against the perineum (the ''turtle sign'').[10]
- Upon recognizing shoulder dystocia, the physician should suction the infant's nose and mouth and call for assistance to position the mother in the extreme lithotomy position, with legs sharply flexed up to the abdomen (the McRoberts maneuver) and held by the mother or an assistant.
- The bladder should be drained if this has not already been done. A generous episiotomy may also facilitate delivery. Next, an assistant should apply suprapubic pressure to disimpact the anterior shoulder from the pubic symphysis. It is important to remember never to apply fundal pressure, as this will further force the shoulder against the pelvic rim.[11]

BREECH PRESENTATION

- Breech presentations may be classified as frank, complete, incomplete, or footling. The frank and the complete breech presentations serve as a dilating wedge nearly as well as the fetal head, and delivery may proceed in an uncomplicated fashion.
- The main point in a frank or complete breech presentation is to allow the delivery to progress spontaneously. This lets the presenting portion of the fetus dilate the cervix maximally prior to the presentation of the fetal head. It is recommended that the examiner refrain from touching the fetus until the scapulae are visualized.
- Footling and incomplete breech positions are not considered safe for vaginal delivery because of the possibility of cord prolapse or incomplete dilatation of the cervix. In any breech delivery, immediate obstetric consultation should be requested.

POSTPARTUM CARE

- The placenta should be allowed to separate spontaneously, assisted with gentle traction. Aggressive traction on the cord risks uterine inversion, tearing of the cord, or disruption of the placenta, which can result in severe vaginal bleeding.[12]
- After removal of the placenta, the uterus should be gently massaged to promote contraction. Oxytocin (20 U in 1 L of 0.9 normal saline) is infused at a moderate rate to maintain uterine contraction.
- Episiotomy or laceration repair may be delayed until an experienced obstetrician is able to close the laceration and inspect the patient for fourth-degree (rectovaginal) tears.[13]

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64 VULVOVAGINITIS

David A. Krueger

EPIDEMIOLOGY

- Vulvovaginitis accounts for 10 million physician visits per year in the United States and is the most common gynecologic complaint in prepubertal girls.[1]
- Bacterial vaginosis (BV) is the most common cause of malodorous discharge and is seen almost exclusively in women who have been sexually active. BV is associated with preterm labor and premature rupture of membranes (PROM).[2]
- Candidal vaginitis will affect 75 percent of women at least once during their childbearing years.[3] Factors associated with increased rates of colonization include pregnancy, oral contraceptives, uncontrolled diabetes mellitus, and frequent visits to STD clinics. It is rare in premenarcheal girls and decreases incidence after menopause unless hormone replacement therapy is used.
- *Trichomonas vaginalis* affects 2 to 3 million women annually. The prevalence correlates with overall sexual activity.[4,5] It is associated with preterm delivery and PROM.[6,7] Some 70 percent of men and 85 percent of women who have intercourse with an infected partner develop *Trichomonas* infection.
- Genital herpes is sexually transmitted and is the most frequent cause of painful lesions of the lower genital tract in American women.

PATHOPHYSIOLOGY

- The pathophysiology of vulvovaginitis is related to inflammation of the vulva and vaginal tissues. Causes include infection, irritants and allergens, foreign bodies, and atrophy.
- In females of childbearing age, estrogen causes the development of a thick vaginal epithelium with glycogen stores that support the normal flora. The glycogen is converted by lactobacilli and acidogenic corynebacteria to lactic acid and acetic acid, which forms an acidic environment (pH 3.5 to 4.1) discouraging the growth of pathogenic bacteria.
- Causes of infectious vulvovaginitis include trichomoniasis, caused by *T. vaginalis;* bacterial vaginosis, caused by replacement of normal flora by overgrowth of both anaerobes and *Gardnerella vaginalis;* and candidiasis, usually caused by *Candida albicans.*
- Contact dermatitis results from exposure of vulvar epithelium and vaginal mucosa to chemical irritants or allergens. Secondary infections can occur.
- Foreign bodies left in place longer than 48 h can cause severe localized infections from *Escherichia coli,* anaerobes, or overgrowth of other vaginal flora.
- Atrophic vaginitis during menarche, pregnancy, lactation, and after menopause results from the lack of estrogen stimulation on the vaginal mucosa, resulting in loss of normal rugae, atrophy of squamous epithelium, and increase in vaginal pH.

CLINICAL FEATURES

- Bacterial vaginosis causes vaginal discharge and pruritus. Examination findings range from mild vaginal redness to a frothy gray-white discharge.
- Candidal vaginitis causes vaginal discharge, severe pruritus, dysuria, and dyspareunia. Examination reveals vulvar and vaginal erythema and edema and a thick ''cottage cheese'' discharge.
- *T. vaginalis* causes vaginal discharge, perineal irritation, dysuria, spotting, and pelvic pain. Examination reveals vaginal erythema and a frothy, malodorous discharge.
- Genital herpes causes painful, fluid-filled vesicles that progress to shallow-based ulcers. Local symptoms include dysuria and pelvic pain. Systemic symptoms such as fever, malaise, headache, and myalgias are common.
- Contact vulvovaginitis causes pruritus and a burning sensation. Examination reveals an edematous, erythematous vulvovaginal area.
- Vaginal foreign bodies can cause a bloody or foul-smelling discharge. Examination generally reveals the foreign body.
- Atrophic vaginitis causes vaginal soreness, dyspareunia, and occasional spotting or discharge. Examination reveals a thin, inflamed, and even ulcerated vaginal mucosa.

DIAGNOSIS AND DIFFERENTIAL

- A detailed gynecologic history should be obtained and a gynecologic exam should be performed.
- Microscopic evaluation of vaginal secretions using normal saline (demonstrating clue cells for BV and motile *T. vaginalis* for trichomoniasis) and 10% potassium hydroxide (demonstrating yeast or pseudohyphae for candidiasis and fishy odor for BV) will frequently provide the diagnosis.
- Secretions should be tested for pH using nitrazine paper. A pH greater than 4.5 is typical of BV or trichomoniasis. A pH less than 4.5 is typical of physiologic discharge or a fungal infection.
- According to the Centers for Disease Control and Prevention, bacterial vaginosis is diagnosed by three of the following: (1) discharge; (2) pH >4.5; (3) fishy odor when 10% KOH is added to the discharge (positive amine test result); and (4) clue cells, which are epithelial cells with clusters of bacilli stuck to the surface, seen on saline wet prep.[8]
- Candidal vaginitis is diagnosed microscopically by the presence of yeast buds and pseudohyphae. A 10% KOH solution will dissolve the epithelial cells, making the findings easier to see. Sensitivity is 80 percent.
- *T. vaginalis* is diagnosed microscopically by the presence of motile, pear-shaped, flagellated trichomonads that are slightly larger than leukocytes. Sensitivity is 40 to 80 percent.
- Genital herpes is diagnosed based on clinical suspicion and is confirmed by either culture or polymerase chain reaction of fluid obtained from the ulcer or vesicle.
- Contact vulvovaginitis is diagnosed by ruling out an infectious cause and identifying the offending agent.
- On wet preparation, atrophic vaginitis will show erythrocytes and increased polymorphonuclear leukocytes (PMNs) associated with small, round epithelial cells, which are immature squamous cells that have not been exposed to sufficient estrogen.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Bacterial vaginosis is treated with metronidazole 500 mg PO bid for 7 days or clindamycin 300 mg PO bid for 7 days. No treatment is necessary for male partners or asymptomatic women. If the patient is at high risk for preterm labor, treat with metronidazole 0.75%, one applicator intravaginally bid for 5 days, or use clindamycin.
- Candidal vaginitis is treated with clotrimazole 1% cream or miconazole 2% cream applied topically for 3 to 7 days. Alternative treatment is fluconazole 150 mg PO. Treatment of sexual partners is not necessary unless candidal balanitis is present.
- Genital herpes is treated by antiviral agents within 1 day of onset of symptoms to help control the symptoms and to accelerate healing of the lesions. Treatment is not curative and does not affect the frequency or severity of recurrences. Patients with severe disease may require hospitalization for IV therapy. Systemic analgesics may also be needed.
- Contact vulvovaginitis is treated by removal of the offending agent. Cool sitz baths and wet compresses of dilute boric acid or Burow's solution may provide some relief. Topical corticosteroids can also be used to relieve symptoms and promote healing.
- Vaginal foreign bodies require removal. No other therapy is necessary.
- Atrophic vaginitis is treated with topical vaginal estrogen. Nightly use of 1/2 to 1 applicator for 1 to 2 weeks should alleviate symptoms. Estrogen

should not be used if there is a history of cancer of any of the reproductive organs or postmenopausal bleeding. Referral to a gynecologist should be made.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 104, ''Vulvovaginitis,'' by Gloria J. Kuhn.

65 PELVIC INFLAMMATORY DISEASE

Laura R. Hopson

EPIDEMIOLOGY

- Pelvic inflammatory disease (PID) occurs at an annual rate of 10 to 20 cases per 1000 women of reproductive age.[1]
- Long-term sequelae occur in 25 percent. These include tubal factor infertility, ectopic pregnancy, and chronic pain.
- Risk factors include multiple sexual partners, history of other sexually transmitted diseases (STDs), substance abuse, frequent vaginal douching, young age, use of intrauterine devices (IUDs)—particularly in the first 4 months after insertion, use of oral contraceptives, and bacterial vaginosis.[2,3]
- Risk is reduced with use of barrier contraception and pregnancy; however, PID can occur during the first trimester and may cause fetal loss.[4,5]
- Disease severity is decreased after tubal ligation.[6]

PATHOPHYSIOLOGY

- PID represents an ascending infection from the lower genital tract.
- *Neisseria gonorrhoeae* and/or *Chlamydia trachomatis* are isolated from almost all cases.
- The infection is likely polymicrobial in 40 to 80 percent of cases.[7] Anaerobes, *Gardnerella vaginalis,* enteric gram-negative organisms, *Haemophilus influenzae, Streptococcus agalactiae, Mycoplasma hominis,* and *Urea urealyticum* have all been implicated.[8]
- Some 10 to 20 percent of cases of untreated gonorrheal or chlamydial cervicitis progress to PID.
- Tuboovarian abscess (TOA) is present in one-third of hospitalized women with PID.[9]
- Fitz-Hugh and Curtis syndrome is acute perihepatitis and focal peritonitis from direct or lymphangitic spread of infection.

CLINICAL FEATURES

- Lower abdominal pain is the most common presenting complaint.
- Other common symptoms include abnormal vaginal discharge, vaginal bleeding, postcoital bleeding, dyspareunia, irritative voiding symptoms, fever, malaise, nausea, and vomiting. PID may produce minimal symptoms.[9]
- Physical exam findings include lower abdominal tenderness, mucopurulent cervicitis, cervical motion tenderness, and bilateral adnexal tenderness.
- Asymmetric adnexal tenderness or an adnexal mass are signs of TOA.
- If there is associated right-upper-quadrant tenderness with jaundice, Fitz-Hugh and Curtis syndrome is likely.

DIAGNOSIS AND DIFFERENTIAL

- PID is a clinical diagnosis; see Table 65-1.
- Laboratory evaluation should include a pregnancy

TABLE 65-1 Diagnostic Criteria for Pelvic Inflammatory Disease

All major criteria below must be present:
Lower abdominal pain
Tenderness on lower abdominal examination
Cervical motion tenderness
Adnexal tenderness
In addition, one or more of the following criteria will enhance the specificity of the diagnosis:
Temperature >100.4°F (38°C)
Abnormal cervical or vaginal discharge
Laboratory evidence of *Chlamydia trachomatis* or *Neisseria gonorrhoeae*
Elevated erythrocyte sedimentation rate or C-reactive protein
White blood cell count >10,000/μL
The following definitive criteria are warranted in selective cases:
Positive transvaginal ultrasound, or other imaging technique, showing thickened, fluid-filled tubes with or without tubo-ovarian abscess or free pelvic fluid, *or*
Positive endometrial biopsy, *or*
Positive laparoscopy

SOURCE: Adapted from *MMWR* 47:79, 1998.

test, wet prep, and endocervical swabs for gonorrhea and chlamydia. A white blood cell count and erythrocyte sedimentation rate or C-reactive protein may also be considered.

- Transvaginal pelvic ultrasound is used to evaluate for TOA, which appears as a complex adnexal mass with multiple internal echoes.
- Endometrial biopsy, culdocentesis, and laparoscopy are additional diagnostic tests; they are not typically indicated in the emergency department.
- The differential diagnosis includes cervicitis, ectopic pregnancy, endometriosis, ovarian cyst, ovarian torsion, spontaneous abortion, septic abortion, cholecystitis, gastroenteritis, appendicitis, diverticulitis, pyelonephritis, and renal colic.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Analgesia and IV hydration should be given as needed.

TABLE 65-2 Outpatient Antibiotic Management of Pelvic Inflammatory Disease

Ceftriaxone 250 mg IM, *or*
Cefoxitin 2 g IM plus probenecid 1 mg PO, *or*
Other parenteral third-generation cephalosporin
plus
Doxycycline 100 mg PO bid × 14 d, *or*
Ofloxacin 400 mg PO bid × 14 d plus metronidazole 500 mg PO bid × 14 d

SOURCE: Adapted from *MMWR* 47:79, 1998.

TABLE 65-3 Inpatient Antibiotic Management of Pelvic Inflammatory Disease

Cefotetan 2 g IV q 12 h, *or*
Cefoxitin 2 g IV q 6 h
plus
Doxycycline 100 mg IV/PO q 12 h
or
Clindamycin 900 mg IV q 8 h
plus
Gentamicin loading dose IV/IM (2 mg/kg) and then maintenance 1.5 mg/kg q 8 h

SOURCE: Adapted from *MMWR* 47:79, 1998.

- Empiric broad-spectrum antibiotics[10] achieve an 84 to 98 percent microbiologic cure rate. See Tables 65-2 and 65-3 for treatment options as recommended by the Centers for Disease Control and Prevention.
- Long-term sequelae may be reduced if antibiotics are begun within 48 h of onset of symptoms.
- An IUD must be removed after antibiotics are started.
- Some 60 to 80 percent of TOAs resolve with antibiotics alone. The remainder may require drainage.
- Admission criteria include pregnancy, inability to exclude other surgical emergencies, immunosuppression, documented or suspected pelvic abscess, IUD in place, high fevers, severe nausea and vomiting, inability to comply with outpatient regimen, failed outpatient treatment, adolescence, or presence of significant fertility issues.[10]
- With outpatient treatment, patients should be followed up within 72 h to assess response to antibiotic therapy.
- Appropriate referrals for treatment of partners and HIV testing/counseling should be provided.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 105, ''Pelvic Inflammatory Disease,'' by Amy J. Behrman and Suzanne Moore Shepherd.

66 COMPLICATIONS OF GYNECOLOGIC PROCEDURES

David M. Cline

- The most common reasons for emergency department visits during the postoperative period following gynecologic procedures are pain, fever, and vaginal bleeding. A focused but thorough evaluation should be performed, including cervical cultures and bimanual examination. (Complications common to gynecologic and general surgery are covered in Chap. 52.)

COMMON COMPLICATIONS OF ENDOSCOPIC PROCEDURES

LAPAROSCOPY

- The incidence of major complications in the United States for laparoscopy may be as low as 0.22 percent.[1]
- In 1993, the American Association of Gynecologic Laparoscopists reported complications for 45,042 procedures as follows: hemorrhage, 1 percent; unintended laparotomy, 1 percent; blood transfusion for hemorrhage, 0.45 percent; and bowel or urinary tract injury 0.41 percent.[2]
- In 1996, the overall incidence of complications in major operative laparoscopy was reported as 10.4 percent.[3]
- The major complications associated with the use of the laparoscope are the following: (1) thermal injuries to the bowel; (2) bleeding at the site of tubal interruption or sharp dissection; and (3) rarely, ureteral or bladder injury, large bowel injury, and pelvic hematoma or abscess.
- Of these complications, the most serious and dreaded is that of thermal injury to the bowel. These patients generally appear 3 to 7 days postoperatively, depending upon the degree of necrosis, with signs and symptoms of peritonitis, including bilateral lower abdominal pain, fever, elevated white blood cell (WBC) count, and direct and rebound tenderness. X-rays may show an ileus or free air under the diaphragm. Although gas has been used to insufflate the abdomen, it should be absorbed totally within 3 postoperative days.
- Patients who have increasing pain after laparoscopy, either early or late, have a bowel injury until proved otherwise. If thermal injury is a serious consideration and cannot be distinguished from other causes of peritonitis, it is best to err on the side of early laparotomy.

HYSTEROSCOPY

- Complications of hysteroscopy occur in approximately 2 percent of cases, including the following: (1) reaction to the distending media, (2) uterine perforation, (3) cervical laceration, (4) anesthesia reaction, (5) intraabdominal organ injury, (6) infection, and (7) postoperative bleeding.[2]
- Postoperative bleeding will be the most likely cause of hospital revisit. After hemodynamic stabilization of the patient, the gynecologist can insert a pediatric Foley or balloon catheter to tamponade the bleeding.

MISCELLANEOUS COMPLICATIONS OF MAJOR GYNECOLOGIC PROCEDURES

CUFF CELLULITIS

- *Cuff cellulitis* is an infection of the contiguous retroperitoneal space immediately above the vaginal apex, including the surrounding soft tissue. It is

a common complication following both abdominal and vaginal hysterectomy.
- It usually produces a fever between postoperative days 3 and 5. These patients complain of fever and lower-quadrant pain. Pelvic tenderness and induration are prominent during the bimanual examination. A vaginal cuff abscess may be palpable.
- The treatment of choice is readmission, drainage, and intravenous antibiotics as determined by the gynecologist.

POSTCONIZATION BLEEDING

- The most common complication associated with major gynecologic procedures is bleeding. If delayed hemorrhage occurs, it usually occurs 7 days postoperatively. Bleeding following this procedure can be rapid and excessive. Visualization of the cervix is the key to controlling such bleeding.
- Application of Monsel's solution, if it is easily available, is a reasonable first step.
- Usually suturing of the bleeding arteriole is necessary. Quite often, the patient must be taken to the operating room for repair secondary to poor visualization.

INDUCED ABORTION

- Retained products of conception and a resulting endometritis are the most common complications.[4,5]
- Patients usually complain of excessive bleeding, fever, and abdominal pain 3 to 5 days postterminination, but they may not return with complaints for up to 2 weeks.
- Pelvic examination reveals a subinvoluted tender uterus with foul-smelling blood vaginally. An elevated WBC count is common.
- Treatment must include evacuation of intrauterine contents and intravenous antibiotic therapy. Triple antibiotic therapy (ampicillin, gentamicin, and clindamycin) is the standard; however, there is increasing evidence that ampicillin with sulbactam 3 g IV is equally effective.
- If the patient has pain, bleeding, or both—but no fever—missed ectopic pregnancy must be ruled out.

VESICOVAGINAL FISTULAS

- Vesicovaginal fistulas may occur after total vaginal hysterectomy. Patients return 10 to 14 days after surgery with watery vaginal discharge. Gynecologic consultation is necessary.

ASSISTED REPRODUCTIVE TECHNOLOGY

- Complications related to ultrasound-guided retrieval of oocytes are rare and include ovarian hyperstimulation syndrome, pelvic infections, intraperitoneal bleeding, and adnexal torsions.[6,7]
- Ovarian hyperstimulation syndrome can be a life-threatening complication of induced ovulation. The incidence in the moderate-to-severe form is 1 percent to 2 percent.
- In the mildest form, symptoms include abdominal distention, ovarian enlargement, and weight gain; in the most severe form, patients have massive third-spacing of fluids into the abdominal cavity, which can lead to ascites, electrolyte imbalances, pleural effusions, and hypovolemia.
- Abdominal and pelvic examinations are contraindicated due to extremely fragile ovaries that are at high risk of rupture or hemorrhage.
- Electrolyte studies, renal function tests, a complete blood count, coagulation studies, and blood for type and cross-match should be obtained. An electrocardiogram to evaluate potential hyperkalemic changes should also be obtained.
- The gynecologist should be consulted for admission.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 108, ''Complications of Gynecologic Procedures,'' by Michael A. Silverman and Karen J. Morrill Hardart.

Section 11
PEDIATRICS

67 FEVER

David M. Cline

EPIDEMIOLOGY

- Fever is the single most common complaint of children presenting to the emergency department and accounts for about 30 percent of all pediatric outpatient visits.

PATHOPHYSIOLOGY

- Fever is defined as a rise in deep body temperature associated with a resetting of the body's thermostat.[1] This thermostat is located in the preoptic region of the anterior hypothalamus, near the floor of the third ventricle.
- Exogenous fever-producing substances (pyrogens)—such as bacteria, bacterial endotoxin, antigen-antibody complexes, yeast, viruses, and etiocholanolone—may stimulate the formation and release of endogenous pyrogens.
- Endogenous pyrogens are produced by neutrophils, monocytes, hepatic Kupffer cells, splenic sinusoidal cells, alveolar macrophages, and peritoneal lining cells; they are believed to induce the synthesis of prostaglandins in the hypothalamus. Endogenous pyrogens include interleukin 1, interleukin 6, and tumor necrosis factor.[2]

CLINICAL FEATURES

- Current practice guidelines suggest that a temperature of 38°C (100.4°F) is a sufficient fever to warrant an evaluation.[3]
- In general, higher temperatures are associated with a higher incidence of bacteremia.[4]
- A retrospective study of hyperpyrexia reported that the incidence of meningitis was twice as high in children with fever above 41.1°C (105.9°F) as opposed to children with fever between 40.5° and 41.0°C (104.9° and 105.8°F).[5] Fever alone, however, is not a reliable indicator of the presence or absence of meningitis.

DIAGNOSIS AND DIFFERENTIAL

- **Infants up to 3 Months** Early studies suggested that infants under the age of 3 months are at high risk for serious life-threatening infection.[6,7]
- Clinical assessment of the severity of illness in a young febrile infant is problematic. Young infants lack social skills, such as the social smile, and lack the ability to interact with the examiner.[6]
- Febrile infants during the first 2 months of life appear to be at greater risk, as the incidence of bacteremia is 13 percent through 1 month and 10 percent during the second month of age.[8]
- A history of lethargy, irritability, or poor feeding suggests a serious infection.[9]
- Inconsolable crying or increased irritability during examination is frequently seen in infants with meningitis.[10]
- Cough or tachypnea with a respiratory rate over 40 might suggest a lower respiratory infection and the need for a chest x-ray.
- The absence of any diagnostic abnormalities on history or physical examination suggests the need for extensive laboratory tests to detect occult infection. These tests would include a complete blood count (CBC) and differential, blood culture, lumbar puncture, chest x-ray, urinalysis and cul-

ture, and a stool culture if there is a history of diarrhea.[10]

- Urinary tract infections (UTIs) are the most common bacterial infection in this age group. UTIs may not produce symptoms other than fever.[11,12] Urinalysis obtained via catheter and culture should be included routinely in the evaluation.
- The recognition of occult serious infection in the well-appearing young, febrile infant is problematic but certain characteristics may help to identify low-risk infants. No single variable can correctly identify these infants. Criteria include nontoxic appearance, white blood cell count (WBC) between 5000 and 15,000/mm^3, band count less than 1500/mm^3, no evidence of soft-tissue infection, normal urinalysis and stool with less than 5 white blood cells per high-power field in infants with diarrhea.[13,14] In low-risk patients, absence of these variables is usually (but not always) associated with the absence of serious illness (0.2 percent).[13,14]
- Some clinicians have recommended that these young febrile infants should receive antibiotic coverage with ceftriaxone (50 mg/kg) pending culture results (cefotaxime 50 mg/kg should be used if the child is less than 1 week old).[15] However, this management algorithm is no longer the standard of care.[16]
- The need for hospitalization in infants up to 3 months old presents another area of disagreement.[17] Some physicians hospitalize all febrile infants under 3 months of age, whereas others hospitalize only those under 1 month of age. Because the differentiation between a sick and a well infant is so difficult, all such febrile infants need extensive septic workups. The decision to use prophylactic antibiotics is not a substitute for a complete septic workup.

- **Infants 3 to 24 Months** Clinical judgment appears to be more reliable in the assessment of the older infant.[18] Characteristics to note are willingness to make eye contact, playfulness, response to noxious stimuli, alertness, and consolability.
- Pneumonia is commonly of viral etiology; however, it is appropriate to institute antibiotic therapy. See Chap. 73.
- Nuchal rigidity or Kernig's or Brudzinski's sign may not be apparent in the child under 2 years old. A bulging fontanelle, vomiting, irritability that increases when the infant is held, inconsolability, or a seizure may be the only signs suggestive of meningitis.[10]
- Up to 20 percent of children with petechiae will have bacteremia or meningitis, most frequently with *Neisseria meningitidis* or (less commonly) *Haemophilus influenzae.*[19,20]
- The organism most commonly causing bacteremia in this age group is *Streptococcus pneumoniae.* It is apparent that bacteremic patients do better if they receive antibiotics early. The blood culture appears to be useful for following a patient who may not be returning for periodic evaluations.
- Controlled trials investigating efficacy have demonstrated a reduction in the incidence of meningitis in bacteremic children treated with ceftriaxone (50 mg/kg) given twice, 24 h apart, as compared to those treated with oral or no antibiotics.[21] However, only 0.019 percent of children from 3 to 36 months of age with occult bacteremia develop meningitis, and widespread prophylactic antibiotics are not recommended.[16]
- Parenteral ceftriaxone should never be initiated without appropriate antecedent or coincident diagnostic studies.[21]

- **Older Febrile Children** Children over 3 years of age are easier to evaluate because they can specify their complaints. The risk of bacteremia appears lower in this age group.[22]
- Pneumonia in this age group may be caused by *Mycoplasma pneumoniae.* Rales may not be present early in the course, and bedside cold agglutinins may assist with diagnosis of *M. pneumoniae.* See Chap. 73 for treatment recommendations.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Although fever may provoke febrile seizures, fever is not known to produce any harmful effects in children. However, fever does cause the patient discomfort and as such should be treated. One can facilitate heat loss in a child using any combination of measures.
- Unwrapping a bundled child increases heat loss through radiation.[23]
- Drug dosage for ibuprofen is 5 to 10 mg/kg per dose at 6-h intervals (maximum dose, 600 mg), and dosage for acetaminophen is 10 to 15 mg/kg per dose at 4-h intervals.[24,25] Alternating these two drugs every 3 h in an effort to avoid the recrudescence of fever is common practice. Aspirin should not be used in children with chickenpox or with influenza-like illnesses due to its link with Reye's syndrome.
- All patients with positive blood cultures should be recalled for repeat evaluation. If clinically well

and afebrile, they should be instructed to complete the current course of therapy.
- However, any patient who remains febrile or does poorly, even if on oral antibiotics, should receive a complete septic evaluation (CBC, blood culture, lumbar puncture, chest film, urine culture), be hospitalized, and receive parenteral antibiotics.

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68 COMMON NEONATAL PROBLEMS

Lance Brown

NORMAL VEGETATIVE FUNCTIONS

- Bottle-fed infants generally take six to nine feedings per 24-h period, with a relatively stable pattern developing by the end of the first month of life. Breast-fed infants generally prefer feedings every 2 to 4 h.
- Infants typically lose 5 to 10 percent of their birth weight during the first 3 to 7 days of life. After that time, infants are expected to gain about 1 oz (20 to 30 g) per day during the first 3 months of life.
- The number, color, and consistency of stool vary

TABLE 68-1 Conditions Associated with Uncontrollable Crying, Irritability, and/or Lethargy in Neonates

Intestinal colic
Traumatic conditions
Battered child syndrome (fractures, burns, etc.)
Falls (e.g., skull or extremity fractures)
Open diaper pin
Strangulation of digit or penis
Corneal abrasion or foreign body
Infections
Meningitis
Generalized sepsis
Otitis media
Urinary tract infection
Gastroenteritis
Surgical
Incarcerated hernia (umbilical or inguinal)
Testicular torsion
Anal fissure
Improper feeding practices

in the same infant from day to day and certainly among infants. Normal breast-fed infants may go 5 to 7 days without stooling or have six to seven stools per day. Color has no significance unless blood is present.
- Respiratory rates in newborns can vary, with normal ranges from 30 to 60 breaths per minute. Periodic breathing with brief (less than 5 to 10 s) pauses in respiration may be normal.
- Normal newborns awaken at variable intervals that can range from about 20 min to 6 h. Neonates and young infants tend to have no day-night differentiation until about 3 months of age.

CRYING, IRRITABILITY, AND LETHARGY (INCONSOLABILITY)

- There are multiple causes of crying, irritability, and lethargy in infants (see Table 68-1). These causes range from the relatively benign to the life-threatening. True inconsolability represents a serious condition in the majority of infants.[1]

INTESTINAL COLIC

- Intestinal colic is the most common cause of excessive (but not inconsolable) crying. The cause is unknown. The incidence is about 13 percent of all neonates. The formal definition includes crying for 3 h per day or more for 3 days per week or more over a 3-week period. Intestinal colic seldom lasts beyond 3 months of age.

NONACCIDENTAL TRAUMA (CHILD ABUSE)

- A battered child may present with unexplained bruises at varying ages, skull fractures, intracranial injuries identifiable on computed tomography (CT) of the head, extremity fractures, cigarette burns, retinal hemorrhages, unexplained irritability, lethargy, or coma.

FEVER AND SEPSIS

- Fever in a neonate (28 days of age or younger) is defined by the history (temperature taken by parent) or the presence of a rectal temperature of 38°C (100.4°F) or more. Fever in a neonate must be taken seriously, and at this point in time the proper management includes a septic workup (complete blood count, urinalysis, blood culture, urine culture, lumbar puncture and analysis of cerebrospinal fluid, cerebrospinal fluid culture, chest x-ray if respiratory symptoms are present, and stool culture if diarrhea is present), the administration of parenteral antibiotics (ampicillin plus gentamicin or ampicillin plus cefotaxime), and admission. A well appearance on clinical examination and initial tests with results available in the emergency department cannot reliably rule out serious bacterial infection in a neonate.[2] See Table 68-2 for the signs and symptoms of neonatal sepsis.
- Neonates with fever have about twice the risk of having a serious bacterial infection compared with infants in the second month of life.
- Sepsis in neonates typically is grouped into ''early-onset'' disease occurring in the first 5 days of life and ''late-onset'' disease occurring after the first week of life. Risk factors for early-onset sepsis include maternal fever, prolonged rupture of membranes, and fetal distress. Late-onset sepsis typically develops somewhat more gradually and is associated more commonly with meningitis.
- Bacteria associated with neonatal sepsis include

TABLE 68-2 Signs and Symptoms of Neonatal Sepsis

Temperature	Fever, hypothermia
Central nervous system dysfunction	Lethargy, irritability, seizures
Respiratory distress	Apnea, tachypnea, grunting
Feeding disturbance	Vomiting, poor feeding, gastric distention, diarrhea
Jaundice	
Rashes	

group B streptococci, enteric organisms such as *Escherichia coli* and *Klebsiella, Haemophilus influenzae,* and *Listeria monocytogenes.*

GASTROINTESTINAL SYMPTOMS

SURGICAL LESIONS

- Surgically correctable abdominal emergencies in neonates are uncommon, may present with nonspecific symptomatology, and when suspected require prompt consultation with an experienced pediatric surgeon.
- The most common signs and symptoms are nonspecific and include irritability and crying, poor feeding, vomiting, constipation, and abdominal distention. Bilious vomiting is suggestive of malrotation with midgut volvulus and requires prompt consultation. A groin mass may represent an incarcerated hernia.

FEEDING DIFFICULTIES

- An emergency department visit may arise when there is a parental perception that an infant's food intake is inadequate. If the patient's weight gain is adequate and the infant appears satisfied after feeding, reassurance of the parents is appropriate. A successful trial of feeding in the emergency department can reassure parents, emergency department nurses, and physicians.
- When an underlying anatomic abnormality interferes with feeding or swallowing (e.g., esophageal stenosis, esophageal stricture, laryngeal clefts, compression of the esophagus or trachea by a double aortic arch), the infant typically has had trouble feeding since birth and usually presents with malnourishment and dehydration.
- Infants with a recent and true decrease in intake usually have an acute disease, most commonly an infection.[3]

REGURGITATION

- Regurgitation is due to reduced lower esophageal sphincter pressure and relatively increased intragastric pressure in neonates.
- Regurgitation is typically a self-limited condition, and if an infant is thriving and gaining weight appropriately, reassurance is appropriate.

VOMITING

- Vomiting is differentiated from regurgitation by forceful contraction of the diaphragm and abdominal muscles. Vomiting has a variety of causes and is rarely an isolated symptom.
- Vomiting from birth usually is due to an anatomic anomaly and usually prevents an infant from being discharged from the newborn nursery.
- Vomiting is a nonspecific but serious symptom in neonates. Etiologies are diverse and include increased intracranial pressure (e.g., shaken-baby syndrome), infections (e.g., urinary tract infections, sepsis, gastroenteritis), hepatobiliary disease (usually accompanied by jaundice), and inborn errors of metabolism (usually accompanied by hypoglycemia and metabolic acidosis).

DIARRHEA

- Although bacterial diarrhea is a cause of bloody diarrhea, it is rare in neonates. The most common causes of blood in the stool in infants less than 6 months of age are cow's milk intolerance and anal fissures. Breast-fed infants may have hem-positive stool from swallowed maternal blood as a result of bleeding nipples.
- Necrotizing enterocolitis may present as bloody diarrhea and usually presents with other signs of sepsis (e.g., jaundice, lethargy, fever, poor feeding, abdominal distention). Abdominal radiography may demonstrate pneumatosis intestinalis.
- Dehydrated neonates (and neonates with impending dehydration from rotavirus) should be admitted for rehydration.

ABDOMINAL DISTENTION

- Abdominal distention can be normal in a neonate and usually is due to lax abdominal muscles and relatively large intraabdominal organs. In general, if a neonate appears comfortable and is feeding well and the abdomen is soft, there is no need for concern.

CONSTIPATION

- Infrequent bowel movements do not necessarily mean that an infant is constipated. Stool patterns can be quite variable, and breast-fed infants may go a week without passing stool and then pass a normal stool.

- If an infant has never passed stool, the differential diagnosis includes intestinal stenosis and atresias, Hirschsprung's disease, and a meconium ileus or plug.
- Constipation that develops later in the first month of life suggests Hirschsprung's disease, hypothyroidism, or anal stenosis.

NOISY BREATHING AND STRIDOR

- Noisy breathing in a neonate is usually benign. Infectious causes of stridor seen commonly in older infants and young children (e.g., croup) are rare in neonates.
- Stridor in a neonate often is due to a congenital anomaly, with laryngomalacia being the most common. Other causes include webs, cysts, atresias, stenoses, clefts, and hemangiomas.

APNEA AND PERIODIC BREATHING

- Periodic breathing may be normal in neonates.
- Apnea is defined formally as a cessation of respiration for more than 10 to 20 s with or without bradycardia and cyanosis. Apnea generally signifies a critical illness, and prompt investigation (especially for sepsis) and admission for monitoring and therapy (including empirical antibiotics) should be initiated.

CYANOSIS AND BLUE SPELLS

- Many disorders may present with cyanosis, and differentiating them may present a diagnostic challenge. However, some symptom patterns may help differentiate various causes and assist in suggesting the correct diagnosis and course of action.
- Rapid, unlabored respirations and cyanosis suggest cyanotic heart disease with right-to-left shunting.
- Irregular, shallow breathing and cyanosis suggest sepsis, meningitis, cerebral edema, or intracranial hemorrhage.
- Labored breathing with grunting and retractions is suggestive of pulmonary disease such as pneumonia and bronchiolitis.
- All cyanotic neonates should be admitted to the hospital for monitoring, therapy, and further investigation.[4,5]

JAUNDICE

- There are multiple causes of jaundice, and the likelihood of these causes is based on the age at which the patient has the onset of jaundice.
- Jaundice that occurs within the first 24 h of life tends to be serious in nature and usually is addressed while the patient is in the newborn nursery.
- Jaundice that develops during the second or third day of life is usually physiological, and if the neonate is gaining weight, is feeding well, is not anemic, and does not have a bilirubin approaching 20 mg/dL, reassurance and close follow-up are appropriate.
- Jaundice that develops after the third day of life is generally serious. Causes include sepsis, congenital infections, congenital hemolytic anemias, breast-milk jaundice, and hypothyroidism. The workup of these infants usually includes a septic workup with a lumbar puncture, a peripheral blood smear, direct and total bilirubin levels, a reticulocyte count, and a Coombs test. Empirical antibiotics generally are administered when sepsis is suspected (see Table 68-2).

ORAL THRUSH

- Intraoral lesions caused by *Candida* are typically white and pasty, covering the tongue, lips, gingiva, and mucous membranes.
- The presence of oral thrush may prompt a visit to the emergency department because the parent notices ''something white'' in the mouth or because the discomfort of extensive lesions interferes with feeding.
- Treatment consists of the topical application of oral nystatin suspension.

SUDDEN INFANT DEATH SYNDROME

- When a neonate presents in full cardiopulmonary arrest, the myocardium generally has suffered severe hypoxic ischemic damage.
- Possible etiologies to consider are infection, trauma including child abuse, inborn errors of metabolism, and cardiac lesions dependent on a patent ductus arteriosus.
- It is exceedingly rare for a child presenting in asystolic arrest to have a return of spontaneous circulation.

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69 PEDIATRIC HEART DISEASE

Lance Brown

- This chapter covers three main presentations of pediatric heart disease as they present to the emergency department: cyanosis and shock, congestive heart failure, and complications of known congenital heart disease. Other cardiovascular topics, such as dysrhythmias (Chap. 4), syncope (Chap. 81), pediatric hypertension (Chap. 29), and myocarditis and pericarditis (Chap. 27), are covered in other chapters and are not discussed here.

EPIDEMIOLOGY

- Pediatric cardiac conditions are relatively rare. Congenital heart disease is a broad term that encompasses a multitude of anatomic abnormalities. Congenital heart disease is the most common form of pediatric heart disease and is present in only 8 cases per 1000 live births in all forms.[1] Acquired heart disease is less common and includes complications secondary to rheumatic fever (now quite uncommon), Kawasaki disease, severe chronic anemias, myocarditis, pericarditis, and endocarditis.

PATHOPHYSIOLOGY

- **CYANOSIS AND SHOCK** Five main conditions present with cyanosis: transposition of the great arteries, tetralogy of Fallot, truncus arteriosus, tricuspid atresia, and total anomalous venous return (all five start with ''T'').
- The anatomy of each of these conditions is different, but a few simple principles are common to all of them. Cyanosis is present and generally is due to an anatomic shunt with mixing of oxygenated and deoxygenated blood.

- **CONGESTIVE HEART FAILURE** Multiple causes of congestive heart failure are seen, and the likely etiology is based on the age of the patient at the time of presentation (see Table 69-1).
- The most common general cause of congestive heart failure in infants is an afterload increase that results from an anatomic abnormality. Less commonly, increases in preload or general volume overload are responsible for the heart failure. Older infants and children may have acquired causes of poor contractility (e.g., myocarditis) that lead to heart failure.

CLINICAL FEATURES

- **CYANOTIC HEART DISEASE** Tetralogy of Fallot may present with hypercyanotic ''tet spells'' characterized by episodes of paroxysmal dyspnea with labored respirations, cyanosis, and syncope.[2] Without prompt treatment, these events may progress to hypoxic seizures, cerebral thrombosis, and death.[2]

TABLE 69-1 Differential Diagnosis of Congestive Heart Failure Based on Age at Presentation

AGE	SPECTRUM	
1 min 1 h	Noncardiac origin: anemia, acidosis, hypoxia, hypoglycemia, hypocalcemia, sepsis	Acquired
1 day	PDA in premature infants	Congenital
1 week	HPLV	Congenital
2 weeks	Coarctation	Congenital
1 month	Ventricular septal defect	Congenital
3 months	Supraventricular tachycardia	Acquired
1 year	Myocarditis	Acquired
	Cardiomyopathy	Acquired
	Severe anemia	Acquired
10 years	Rheumatic fever	Acquired

ABBREVIATIONS: PDA = patent ductus arteriosus; HPLV = hypoplastic left ventricle.

- Transposition of the great vessels typically presents within the first week of life with dusky lips, tachypnea, and difficulty feeding.[3] The chest x-ray may show a normal cardiac silhouette, and typically there is no murmur.[3]
- In left ventricular outflow obstruction syndromes (e.g., hypoplastic left heart syndrome, tricuspid atresia, and critical coarctation of the aorta), the neonate initially can appear quite well. When these lesions are present, systemic perfusion relies on an open ductus arteriosus.[4] Upon closure of the ductus arteriosus, cardiac output falls, perfusion becomes negligible, and a state of profound cardiogenic shock ensues.[4]

- **Congestive Heart Failure** The presentation of congestive heart failure can be quite subtle and may include poor feeding, excessive diaphoresis (especially with feeding, a surrogate infantile ''stress test''), tachypnea, rales, rhonchi and/or wheezing, and hepatomegaly disproportionate to splenomegaly. Peripheral edema and jugular venous distention are not expected.

- **Children with Known Congenital Heart Disease** A child with a palliative surgical shunt may have shunt dysfunction with acute distress and increasing cyanosis.
- A pulmonary hypertensive crisis may ensue in young children with congenital heart disease (typically large ventricular septal defects) and pulmonary hypertension. Under stress such as a painful procedure, pulmonary vasospasm may occur, leading to cyanosis and lethargy.
- Young children on digoxin may experience digoxin toxicity, typically presenting with bradycardia.

DIAGNOSIS AND DIFFERENTIAL

- The goal in diagnosing pediatric heart disease in the emergency department is to place the patient's condition into a broad category such as cyanosis without shock, cyanosis with shock, shock without cyanosis, or congestive heart failure.
- The identification of the exact anatomic lesion is deferred to the inpatient workup, which often includes echocardiography.
- Infants who may have a lesion dependent on a patent ductus arteriosus (left ventricular outflow obstructions) can have a markedly improved clinical course and stabilization if prostaglandin E_1 is administered in a timely fashion.[3,4]
- A hyperoxia test should be performed. In this instance, 100% oxygen should be provided to the patient, and in general, patients with pulmonary conditions will have markedly improved oxygenation. If significant hypoxia persists, a cardiac abnormality should be strongly considered in a neonate in shock or respiratory distress.[1]

EMERGENCY DEPARTMENT MANAGEMENT AND CARE

- Attention to the ABCs and the administration of oxygen are the first priority. Cardiac and pulse oximetry monitoring and intravenous or intraosseous access should commence promptly.
- In children with known tetralogy of Fallot who appear to be having a ''tet spell,'' a trial of morphine sulfate at a dose of 0.2 mg/kg intramuscularly or subcutaneously is appropriate.[2]
- In neonates in severe cardiogenic shock, prostaglandin E_1 should be administered in an attempt to reopen the ductus arteriosus. In general, these infants should be intubated before the administration of the prostaglandin, as apnea is a prominent side effect.[3,4]
- When marked congestive heart failure is present, intravenous furosemide 1 to 2 mg/kg should be administered.[5]
- When congestive heart failure progresses to cardiogenic shock, dopamine or dobutamine may become necessary.[5]
- Prompt consultation with a pediatric cardiologist and transfer to a tertiary care center are appropriate when any of the conditions listed above is suspected.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 115, ''Pediatric Heart Disease,'' by C. James Corrall.

70 OTITIS AND PHARYNGITIS

David M. Cline

OTITIS MEDIA

- Otitis media (AOM), an infection of the middle ear, commonly affects infants and young children because of the relative immaturity of the upper respiratory tract, especially the eustachian tube.

EPIDEMIOLOGY

- Each year there are 24.5 million office visits and over 3.7 million emergency department visits for otitis media as well as indirect costs of $5.7 billion a year.[1–3]
- The incidence is higher in males, children who attend day care, children exposed to smoke, and those with a family history of otitis media.[1]
- *Streptococcus pneumoniae* is the most prevalent and most virulent cause, accounting for approximately 40 percent of infections.[4] *Haemophilus influenzae,* NT (nontypeable), and *Moraxella catarrhalis* account for another 40 percent and have a high rate of spontaneous resolution.[5] *Chlamydia pneumoniae* is more common in those less than 6 months of age, and *Staphylococcus aureus* is more common in those less than 6 weeks of age.[6]

PATHOPHYSIOLOGY

- Abnormal function of the eustachian tube appears to be the dominant factor in the pathogenesis of middle-ear disease. Both obstruction and abnormal patency play a role in eustachian tube dysfunction.

CLINICAL FEATURES

- The peak age is 6 to 18 months.[1] Symptoms include fever, poor feeding, irritability, vomiting, ear pulling, and earache.[7]
- Signs include a dull, bulging, immobile tympanic membrane (TM); loss of visualization of bony landmarks in the middle ear; air-fluid levels or bubbles in the middle ear; and bullae on the TM.[8,9] The light reflex is of no diagnostic value.[8]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on presenting symptoms and changes of the TM and middle ear. A red TM alone does not indicate the presence of an ear infection. Fever, prolonged crying, and viral infections can cause hyperemia of the TM.[8]
- Pneumatic otoscopy can be a helpful diagnostic tool; however, a retracted drum from any cause will demonstrate decreased mobility.[9]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment begins with the antibiotics listed in Table 70-1. Amoxicillin remains the first drug of choice despite the increasing incidence of penicillin-resistant *Strep. pneumoniae* and the predominance of β-lactamase-producing *H. influenzae* NT and *M. catarrhalis.*[1]
- An average of 20 percent of pneumococci have some degree of penicillin resistance; some geographic areas have rates as high as 40 percent. Approximately 50 percent of *H. influenzae* NT and >90 percent of *M. catarrhalis* are β-lactamase-producing strains.[1]
- Penicillin-resistant *Strep. pneumoniae* also exhibits resistance to erythromycin, trimethoprim-sulfamethoxazole, clindamycin, cefixime, ceftibuten, cefaclor, and loracarbef.[10–12] *Strep. pneumoniae* has rapidly developed significant resistance to macrolides (clarithromycin and azithromycin) (35 to 55 percent).[10–12]
- Pharmacokinetic studies suggest that higher doses of amoxicillin (80 to 100 mg/kg per day divided into two doses a day) are more active against moderately and highly resistant strains of *Strep. pneumoniae.*[13]
- Risk factors for drug-resistant *Strep. pneumoniae* (DRSP) include age <2 years, group day care, frequent AOM, frequent and/or recent antibiotics, and immunoincompetence.[6,13] High-dose amoxicillin (80 to 100 mg/kg per day) should be considered for children at risk.[13]
- Other antibiotics appropriate for DRSP include amoxicillin-clavulanate, cefpodoxime, and ceftriaxone.[1,7,12] Cefuroxime axetil exhibits moderate ac-

TABLE 70-1 Drug Treatment for Otitis Media

First-line antibiotics	Amoxicillin	45–60 mg/kg/d tid × 10 days
	Trimethoprim-sulfamethoxazole	8–10 mg trimethoprim/kg/d × 10 days
	Erythromycin/sulfisoxazole	50 mg erythromycin/kg/d × 10 days
Second-line antibiotics	Amoxicillin	80–100 mg/kg/d × 10 days
	Amoxicillin-clavulanate	45 mg/kg/d × 10 days
	Cefpodoxime	10 mg/kg/d (max 400) × 10 days
	Cefuroxime axetil	20 mg/kg/d × 10 days
	Azithromycin	10 mg/kg × 1 day, 5 mg/kg × 4 days
	Ceftriaxone	50 mg/kg/d × 1–3 doses
Analgesics	Auralgan otic	3–4 drops q4h
	Acetaminophen/codeine	0.5–1.0 mg codeine/kg/dose q4–6h
	Ibuprofen	10 mg/kg/dose q8h

tivity against intermediately resistant *Strep. pneumoniae.*[1,7,12]

- Infants less than 30 days of age with AOM are at risk for infection with group B *Streptococcus, Staph. aureus,* and gram-negative bacilli and should undergo evaluation and treatment for presumed sepsis.[8]
- Recurrent AOM is characterized as three or more episodes within 6 months or four or more within 12 months.[7]
- Persistent AOM occurs when the signs and symptoms of AOM do not improve after appropriate antibiotic therapy.[7]
- High-dose amoxicillin therapy or treatment with other antibiotics suitable for DRSP coverage should be considered for both recurrent and persistent AOM.[13]
- In using ceftriaxone for the presumed treatment of DRSP, one intramuscular 50 mg/kg dose may not suffice; children should be followed on a 24-h basis until the symptoms resolve.[14–16] As many as three injections may be necessary.[14–16]
- In uncomplicated AOM, symptoms resolve within 48 to 72 h; however, the middle-ear effusion may persist as long as 8 to 12 weeks. Routine follow-up is not necessary unless the symptoms persist or worsen.[4–7]

OTITIS MEDIA WITH EFFUSION (OME)

- OME is fluid in the middle ear without the associated signs and symptoms of an acute infection.[3] Chronic OME (duration >3 months) can result in significant hearing loss and language delay.[17]

CLINICAL FEATURES

- OME is characterized by a middle-ear effusion, distortion of bony landmarks, and decreased mobility of the TM.[8]
- There are no symptoms of acute infection such as fever, irritability, and otalgia.[8]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on the appearance of the TM in the absence of systemic symptoms. Audiometry is of limited value for diagnosis but is crucial to the evaluation of a hearing deficit.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of OME includes careful observation for resolution (the standard treatment of choice)[13,18] or ear, nose, and throat (ENT) referral and hearing evaluation for chronic OME.[19] There is no indication for antihistamines, decongestants, or steroids.[19]
- Antibiotics achieve resolution in only 14 percent of cases.[20]
- Bilateral myringotomy tubes may be required if the effusion does not resolve.

OTITIS EXTERNA

EPIDEMIOLOGY

- Otitis externa (OE) is an inflammatory process that involves the auricle, the external auditory

canal (EAC), and the surface of the TM. It is commonly caused by gram-negative enteric organisms, *Staphylococcus, Pseudomonas,* and fungi.[21,22]

PATHOPHYSIOLOGY

- Any compromise of the normal shape of the canal or the normal process of cerumen production can lead to OE caused by colonization and tissue invasion by pathogenic organisms.[21,22]

CLINICAL FEATURES

- Peak seasons for OE are spring and summer, and the peak age is 9 to 19 years. Symptoms include earache, itching, and fever.[21,22]
- Signs include erythema, edema of EAC, white exudate on EAC and TM, pain with motion of the tragus or auricle, and periauricular or cervical adenopathy.[21,22]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of OE is based on clinical signs and symptoms. A foreign body in the external canal should be excluded by carefully removing any debris that may be present.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The clinician should place a wick in the canal if significant edema obstructs the EAC.[22] Cortisporin otic solution, otic Domeboro, or propylene glycol solution can be used. Oral antibiotics are indicated if AOM or auricular cellulitis is present.[22]
- Follow-up should be advised if improvement does not occur within 48 h; otherwise, reevaluation at the end of treatment is sufficient.[22]
- Cultures of the EAC may identify unusual or resistant organisms. Patients with diabetes or other forms of immunoincompetence can develop malignant OE.[22]
- Malignant OE is characterized by systemic symptoms and auricular cellulitis. This condition can result in serious complications and requires hospitalization with intravenous antibiotics.[22]

PHARYNGITIS

EPIDEMIOLOGY

- It has been estimated that $300 million is spent annually on the diagnosis and treatment of pharyngitis.[23]

PATHOPHYSIOLOGY

- Etiologies include multiple viruses and bacteria, but only group A β-hemolytic streptococcus (GABHS), Epstein-Barr virus, and *Neisseria gonorrhoeae* require an accurate diagnosis.[23,24]
- The identification and treatment of GABHS pharyngitis are important in preventing the suppurative complications and sequelae of acute rheumatic fever.[23]

CLINICAL FEATURES

- Peak seasons for GABHS are late winter and early spring, and the peak age is 4 to 11 years. Symptoms (sudden onset) include sore throat, fever, headache, abdominal pain, enlarged anterior cervical nodes, palatal petechiae, and tonsillar hypertrophy.[25]
- With GABHS, there is absence of cough, coryza, laryngitis, stridor, conjunctivitis, and diarrhea.[25]
- A scarlatina-form rash associated with pharyngitis almost always is GABHS and is commonly referred to as scarlet fever. A diagnosis that is based on clinical findings alone has 50 to 70 percent accuracy at best.[26]
- Epstein-Barr virus (EBV) is a herpes virus and often presents much like streptococcal pharyngitis.[27] Common symptoms are fever, sore throat, and malaise. Cervical adenopathy may be prominent and often is posterior as well as anterior. Hepatosplenomegaly may be present.
- EBV should be suspected in a child with pharyngitis that is not responsive to antibiotic in the presence of a negative throat culture.[27,28]
- Gonococcal (GC) pharyngitis in children and nonsexually active adolescents should alert one to the possibility of child abuse.[29] GC pharyngitis tends to have a more benign clinical presentation than does GABHS pharyngitis.

TABLE 70-2 Treatment of GABHS and GC Pharyngitis

GABHS pharyngitis	
Penicillin V	1 g bid × 10 days >27 kg 500 mg bid × 10 days <27 kg
Amoxicillin	60 mg/kg/day tid × 10 days 750 mg qd × 10 days ≥5 years of age
Benzathine PCN	1.2 million U IM >27 kg; 600.000 U IM <27 kg
Erythromycin	E. estolate 20–40 mg/kg/day tid × 10 days E. ethylsuccinate 40–50 mg/kg/day tid × 10 days
Cephalexin	25–50 mg/kg/day bid × 10 days (500 bid adolescent)
Cefadroxil	30 mg/kg/day bid × 10 days
Azithromycin	12 mg/kg qd × 5 days
Gonococcal pharyngitis	
Ceftriaxone	125 mg IM <45 kg 250 mg IM >45 kg
Spectinomycin (PCN allergy) and erythromycin or doxycycline	40 mg/kg/IM × 7 days 40 mg/kg/day (<8 years old) 100 mg bid (>8 years old) × 7 days

DIAGNOSIS AND DIFFERENTIAL

- A definitive diagnosis of GABHS is made with the throat culture; however, this may not always be practical in the emergency department (ED) because of the time involved and potential problems with follow-up.
- Rapid antigen detection tests, if properly performed, have sensitivity and specificity close to those of a throat culture.[30,31]
- A negative rapid strep test does not exclude GABHS and should be verified with a throat culture. Other etiologies of pharyngitis to recognize are EBV (infectious mononucleosis) and *N. gonorrhea.*
- With EBV, the white blood cell count typically shows lymphocytosis with a preponderance of atypical lymphocytes. The diagnosis is confirmed with a positive heterophil antibody (mono spot).[32]
- The diagnosis of GC pharyngitis is made by culture on Thayer-Martin medium.[29] Vaginal, cervical, and rectal cultures also should be obtained if GC pharyngitis is suspected.[29]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- See Table 70-2 for antibiotic choices for GABHS and GC pharyngitis.[33,34] Antipyretics and sometimes analgesics are necessary during the first 48 to 72 h of treatment. Appropriate follow-up should be encouraged when there is treatment failure and for symptomatic contacts. Follow-up for suspected GC pharyngitis should include child sexual abuse and social service investigations.
- An increase in the number of treatment failures with penicillin has been reported.[35,36] The evidence does not support the abandonment of penicillin as a mainstay of treatment.[33]
- EBV is usually self-limited and requires only supportive treatment with antipyretics, fluids, and bed rest. Occasionally EBV is complicated by airway obstruction and can be treated effectively with prednisone 2.5 mg/kg per day tapered over 5 days or dexamethasone 1 mg/kg to a maximum of 10 mg and then 0.5 mg/kg every 6 h.[28]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 116, "Otitis and Pharyngitis in Children," by Kimberly S. Quayle, Susan Fuchs, and David M. Jaffe.

71 SKIN AND SOFT TISSUE INFECTIONS

David M. Cline

- This chapter discusses several common skin and soft tissue infections of childhood. Impetigo is discussed in Chap. 84.

CONJUNCTIVITIS

EPIDEMIOLOGY

- Conjunctivitis, the most common ocular infection of childhood, is usually a sporadic illness, but it may occur with epidemic periodicity with viral pathogens in the summer months.
- Although *Chlamydia trachomatis* is more common, *Neisseria gonorrhoeae* poses the greatest threat to the integrity of the eye in the neonate.
- Later in childhood, respiratory tract pathogens predominate, particularly untypable *Haemophilus* species.

PATHOPHYSIOLOGY

- Pathogens introduced into the conjunctival sac may proliferate and produce hyperemia and an inflammatory exudate. This exudate may be purulent, fibrinous, or serosanguineous. With certain organisms, corneal involvement (keratitis) may also occur.

CLINICAL FEATURES

- Older children with conjunctivitis may complain of photophobia, ocular pain, or the sensation of a foreign body in the eye, which is associated with crusting of the eyelids or conjunctival injection.
- Erythema and increased secretions characterize conjunctivitis, with intense redness and purulence being more common in the case of infectious rather than allergic causes.
- Allergic conjunctivitis is typically recurrent and seasonal and is accompanied by pruritus and sneezing.
- Fever and other systemic manifestations do not occur with isolated conjunctivitis.
- The duration of symptoms with infectious causes is often 2 to 4 days.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of infectious conjunctivitis depends on the clinical examination.
- A Gram stain should be performed in infants less than 1 month old or in confusing cases. It will show more than 5 white blood cells (WBCs) per high-power field and, in many cases, bacteria. The finding of gram-negative intracellular diplococci identifies *N. gonorrhoeae.*
- Conjunctival scrapings or cultures may be performed to diagnose *C. trachomatis* or other viral or bacterial pathogens.
- Fluorescein staining helps to identify the dendrites of herpes simplex.
- Conjunctivitis may be a manifestation of a systemic disorder, such as measles or Kawasaki's disease.
- Differential diagnosis of the red eye includes conjunctivitis, orbital and periorbital infection, retained foreign body, corneal abrasion, uveitis, and glaucoma.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment is directed at the most common causes of conjunctivitis based on the patient's age and examination findings as well as slit-lamp exam, fluorescein staining pattern, and Gram staining if indicated.
- Infants less than 1 month of age with exceptionally purulent conjunctivitis or gram-positive stain for *N. gonorrhoeae* should receive a single dose of ceftriaxone, 125 mg intramuscularly, hospital admission, or close follow-up the next day. Public health reporting and investigation are mandatory.[1]
- For infants under 3 months of age, treatment with erythromycin (50 mg/kg/d divided four times a day for 14 days) is instituted to treat *C. trachomatis* and prevent later development of the associated vertically transmitted pneumonia syndrome.
- Older children require only the instillation into the conjunctival sac of a topical antibiotic such as sulfacetamide.
- For herpes simplex infections, urgent consultation with an ophthalmologist is required. Topical and oral antiviral therapy—such as trifluridine, 1 drop nine times daily, and acyclovir—is indicated.
- Antihistamines: The administration of diphenhydramine (5 mg/kg/d divided every 4 to 6 h orally) or hydroxyzine (2 mg/kg/d divided every 6 h PO) may be useful for allergic conjunctivitis, along with eradication of exposure to offending agents.

SINUSITIS

- Sinusitis is an inflammation of the paranasal sinuses that may be secondary to infection and allergy; it may be acute, subacute, or chronic in time course.

EPIDEMIOLOGY

- The major pathogens in acute bacterial sinusitis in childhood are *Streptococcus pneumoniae, Moraxella catarrhalis,* and nontypable *Haemophilus influenzae.*[2]
- The incidence of *H. influenzae* sinusitis in children would be expected to decline with Hib vaccination.[3]

PATHOPHYSIOLOGY

- The ethmoid and maxillary sinuses are present at birth, but the frontal and sphenoid sinuses do not become aerated until 6 or 7 years of age.
- The sinuses are lined primarily by ciliated columnar epithelium and connect with the nasopharynx via narrow ostia.
- Resistance to infection depends on the patency of the ostia, the function of the ciliary mechanism, and the quality of the secretions.
- Obstruction of the ostia results either from mucosal swelling or, less commonly, mechanical obstruction. By far the most frequent offenders are viral upper respiratory infection and allergic inflammation.

CLINICAL FEATURES

- Two major types of sinusitis may be differentiated on clinical grounds: acute severe sinusitis and mild subacute sinusitis.
- Acute severe sinusitis is associated with elevated temperature, headaches, and localized swelling and tenderness or erythema in the facial area corresponding to the sinuses. Such localized findings are most often seen in older adolescents and adults.
- Mild subacute sinusitis is manifest in childhood as a protracted upper respiratory infection (URI), with a predominance of purulent nasal discharge and the absence of swelling. Rather than improving in 3 to 7 days, these children have persistent symptoms in excess of 2 weeks. Fever is infrequent. This latter type of sinusitis may be confused with the congestion of brief duration found with some URIs.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is made on clinical grounds without laboratory or radiographic studies. Transillumination of the maxillary or frontal sinuses is seldom helpful in children.
- Standard radiographs should be obtained for patients with uncertain clinical diagnoses and in cases of severe sinusitis. The most diagnostic finding is an air-fluid level or complete opacification of the sinus.
- Computed tomography (CT) is a more accurate and expensive tool for cases that fail to respond to standard therapy.
- Few other conditions masquerade as sinusitis, and the differential is limited, particularly in children.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- For acute severe disease, intravenous therapy is recommended: cefuroxime (100 mg/kg/d divided every 8 h) or ceftriaxone (75 mg/kg/d) or ampicillin-sulbactam (200 mg/kg/d of ampicillin divided every 8 h). Persistent disease demands ear, nose, and throat referral for surgical drainage.
- Mild subacute disease can be treated with amoxicillin (40 mg/kg/d orally divided three times a day). Persistent subacute disease can be treated with cefprozil (30 mg/kg/d orally divided three times a day) or erythromycin-sulfisoxazole (40 mg/kg/d of erythromycin orally divided four times a day).

CELLULITIS

- Cellulitis is an infection of the skin and subcutaneous tissues that extends below the dermis, differentiating it from impetigo.

EPIDEMIOLOGY

- It is a frequent infection in warm weather.
- Under normal circumstances, *Staphylococcus aureus, Streptococcus pyogenes,* and *H. influenzae* are the most commonly isolated organisms.
- Since the advent of effective conjugated vaccines against *H. influenzae,* such infections are rare in childhood but now more common in infants under the age of 6 months.

PATHOPHYSIOLOGY

- Cellulitis may occur either when a pathogen is directly inoculated into the subcutaneous tissue

or following an episode of bacteremia. The majority of infections involve local invasion after a breach in the integument.
- The organisms responsible are usually *Staphylococcus aureus* and *Streptococcus pyogenes.* In contradistinction, *H. influenzae* disseminates hematogenously.

CLINICAL FEATURES

- Cellulitis manifests a local inflammatory response at the site of infection, with erythema, warmth, and tenderness.
- Fever is unusual, except in severe cases, including those caused by *H. influenzae.*

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of cellulitis is made by inspection. Cellulitis must be differentiated from other causes of erythema and edema, including trauma, allergic reaction, and cold-induced lesions.
- Laboratory studies, including WBC concentration, blood culture, and, rarely, aspirate culture, are obtained in specific circumstances, to include immunocompromise, fever, severe local infection, facial involvement, and failure to respond to standard therapy.
- WBC count over 15,000 is more common in *H. influenzae* infections.[4,5]

EMERGENCY DEPARTMENT CARE

- For toxic patients with fever and leukocytosis or for facial involvement, intravenous therapy should be used: ampicillin-sulbactam (200 mg/kg/d of ampicillin divided every 8 h), cefuroxime (100 mg/kg/d divided every 8 h), or ceftriaxone (75 mg/kg/d).
- For nontoxic patients, dicloxacillin (50 to 100 mg/kg/d divided four times a day) or cephalexin (50 to 100 mg/kg/d divided four times a day) should be used.
- For immunocompromised patients, intravenous therapy should be used: oxacillin (150 mg/kg/d divided every 6 h) or cefazolin (100 mg/kg/d divided every 6 h) plus gentamicin (5 to 7.5 mg/kg/d divided every 8 h).
- Patients who fail to respond to reasonable outpatient antibiotic therapy must be further evaluated and considered for admission and intravenous antibiotic therapy. Other underlying conditions, such as diabetes or underlying immune compromise, must be sought.

PERIORBITAL/ORBITAL CELLULITIS

- Periorbital cellulitis is an inflammatory process of the tissues anterior to the orbital septum or within the orbit (orbital cellulitis).

EPIDEMIOLOGY

- *Staph. aureus* and *Strep. pneumoniae* are the principal etiologic agents. Orbital infections are most often due to *Staph. aureus,* particularly when puncture wounds are involved.
- Children under 3 years of age are more likely to be bacteremic, thus experiencing the highest incidence of periorbital cellulitis.
- Orbital cellulitis can occur at any age but is usually seen in children below 6 years of age.

PATHOPHYSIOLOGY

- Organisms reach the periorbital area either hematogenously or by direct extension from the ethmoid sinus. In the case of orbital disease, contiguous spread is most common.

CLINICAL FEATURES

- Orbital and periorbital cellulitis causes the periorbital area to appear red and swollen. Periorbital edema is usually more pronounced with preseptal infections.
- Proptosis or limitation of extraocular muscle function indicates orbital involvement.
- The eye is usually painful to touch but is nonpruritic.

DIAGNOSIS AND DIFFERENTIAL

- Allergic and traumatic causes for edema must be considered.
- Tumors and metabolic disease may cause swelling and discoloration, particularly thyrotoxicosis in adolescents and neuroblastoma in the young child.
- Leukocytosis occurs frequently with cellulitis and more often with bacteremic preseptal infections. Blood cultures in patients with leukocytosis are often positive.

- Computed tomography is performed when orbital involvement is suspected and may easily demonstrate an inflammatory mass or tumor.

EMERGENCY DEPARTMENT CARE

- Admission and treatment with intravenous antibiotics is indicated to prevent complications of meningitis and subperiosteal abscess. Antibiotic choices are the same as those listed earlier under cellulitis with facial involvement.
- Surgical drainage may be necessary with abscess formation.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 117, ''Skin and Soft Tissue Infections,'' by Richard Malley.

72 BACTEREMIA, SEPSIS, AND MENINGITIS IN CHILDREN

Lance Brown

BACTEREMIA

- The identification of bacteremia and the management of infants and young children with fever and no identifiable source of infection on initial presentation are areas of great controversy.

EPIDEMIOLOGY

- The risk of bacteremia in well-appearing children age 3 to 36 months with temperatures of 39°C or higher is 1.6 percent.[1] This rate has fallen significantly since the advent of the *Haemophilus influenzae* type b (Hib) immunization.
- Neonates with a temperature of 38°C or higher have a 5 percent risk of bacteremia and a 15 percent risk of a serious bacterial infection.[2]
- Children age 3 to 36 months with fever and a recognizable viral syndrome (including croup, varicella, bronchiolitis, and stomatitis) have been found to have an even lower risk of bacteremia (0 to 1.1 percent).[3]

PATHOPHYSIOLOGY

- Bacteremia is present when pathogenic bacteria are present in the blood. This is identified by the growth of a pathogenic bacteria in a blood culture (a ''positive'' blood culture). The term *occult bacteremia* is used when a patient presents without a clinically identifiable source of infection at the initial presentation but the blood culture is subsequently positive.
- Infants and young children are thought to be at increased risk for bacteremia because of their immature reticuloendothelial system. The likelihood of various organisms is age-dependent.
- Neonates are at risk for bacteremia and resultant sepsis from organisms acquired around the time of birth. These organisms include group B streptococci, *Escherichia coli, Listeria monocytogenes,* and enterococcus species. Risk factors include premature delivery, ruptured amniotic membranes more than 24 h before delivery, and maternal amnionitis.
- In older infants and children, *Streptococcus pneumoniae* accounts for more than 90 percent of occult bacteremia, with *Neisseria meningitidis,* group A streptococci, and salmonella responsible for the remainder. *Haemophilus influenzae* type b was a significant cause of bacteremia but has been nearly eliminated since vaccination against this organism began in the early 1990s.[4]

CLINICAL FEATURES

- By definition, occult bacteremia has only fever and a well appearance.
- The presence of croup, bronchiolitis, and uncomplicated varicella makes bacteremia very unlikely.[3]

- The presence of otitis media does not appear to change the risk of bacteremia.[5]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of bacteremia is made by blood culture, the results of which are not available during the initial emergency department visit.
- Other tests, such as complete blood count, erythrocyte sedimentation rate, and C-reactive protein, are neither sensitive nor specific.[6–8]
- A greater elevation in temperature correlates with a higher risk of bacteremia, but even with temperatures of 41°C or higher, most well-appearing children are not bacteremic.[9]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Neonates should undergo a septic workup that includes a complete blood count, blood culture, urinalysis, urine culture, and lumbar puncture; receive parenteral antibiotics (ampicillin and gentamicin or ampicillin and cefotaxime); and be admitted to the hospital.
- The treatment of well-appearing febrile infants and young children is very controversial. The current debate is between ''test minimizers'' and ''risk minimizers.''[10]

SEPSIS

EPIDEMIOLOGY

- Sepsis is an infectious inflammatory syndrome with clinical evidence of infection that may include focal infections and meningitis. Multiorgan failure and death may develop rapidly. The clinical situations in which sepsis may develop or be suspected are quite varied; therefore, the true incidence has not been well described.

PATHOPHYSIOLOGY

- The progression from bacteremia to sepsis is related to colonization with a bacterial pathogen (usually nasopharyngeal), invasion of the blood by encapsulated organisms, the release of inflammatory mediators, and failure of host defenses.

TABLE 72-1 Common Organisms Causing Sepsis in Infants and Young Children

AGE	ORGANISMS
0–1 month	Group B streptococci *Escherichia coli*
2 months–5 years	*Streptococcus pneumoniae* *Neisseria meningitidis* *Haemophilus influenzae* type b*
>5 years	*Neisseria meningitidis* Beta-hemolytic streptococci *Rickettsia rickettsii*†

* Marked decline in cases since the introduction of the Hib vaccine.
† Etiologic agent for Rocky Mountain spotted fever, which is seen in endemic areas after tick bites, with a summer and fall predominance.

- Risk factors include impaired splenic function, congenital metabolic disease, humoral or cellular immunodeficiency states, the presence of an indwelling foreign body (e.g., a central venous catheter), and obstruction to drainage of a body cavity.
- The likelihood of various pathogens as the etiologic agent for sepsis is age-dependent (see Table 72-1).

CLINICAL FEATURES

- Sepsis is a clinical diagnosis. The clinical findings of advanced sepsis are related to alteration in the functioning of end organs, including the brain, heart, blood vessels, lungs, kidneys, and skin.
- Sepsis may present early and subtly or late and obviously. Clinical deterioration may be very rapid.
- Neurologic symptoms include altered mental status with irritability, confusion, and lethargy. A history of poor feeding, a lack of spontaneous motor activity, and hypotonia are common.
- Fever is typical. Infants younger than 3 months of age may be hypothermic, a grave prognostic finding.
- Tachypnea and respiratory distress with retractions may develop as a result of hypoxia or metabolic acidosis.
- In early septic shock, the cardiovascular system responds with a resting tachycardia, warm distal extremities, and brisk capillary refill. In later stages of septic shock, circulatory collapse ensues with weak distal pulses, delayed capillary refill, and cool extremities. Hypotension is a very late, very ominous sign in young children.

- Skin findings may include petechiae that may progress to coalescent purpura, particularly in patients with meningococcal disease.
- Poor renal perfusion typically leads to oliguria and then anuria.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of sepsis is based on clinical appearance. A positive blood culture generally is expected but is not necessary for this clinical diagnosis.
- A child with a toxic appearance should be considered septic and should be treated appropriately with antibiotics promptly. However, in addition to infectious etiologies, the differential diagnosis of a septic-appearing infant or child includes toxicologic ingestion, cardiac disease (e.g., myocarditis), trauma (e.g., shaken-baby syndrome), and metabolic etiologies (e.g., previously unrecognized inborn errors of metabolism).
- The peripheral white blood cell count typically is elevated but may be normal. A low white blood cell count is characteristic of sepsis caused by *N. meningitidis.*

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of shock takes precedence over the diagnostic workup.
- The administration of high-flow oxygen, the initiation of cardiac monitoring, and the placement of intravenous or intraosseous access are important first steps.
- Endotracheal intubation may be required for respiratory failure.
- Fluid resuscitation with 20 mL/kg boluses of normal saline should be administered.
- Dopamine may be necessary to support perfusion after three to four fluid boluses.
- Hypoglycemia should be identified and treated.
- Broad-spectrum antibiotics should be administered as soon as access is available (and after the blood culture, if possible). The administration of antibiotics should not be delayed while awaiting for laboratory test or lumbar puncture results.
- Antibiotic selection is empirical and aged-based (see Table 72-2).

MENINGITIS

EPIDEMIOLOGY

- Since the advent of the *H. influenzae* type b (Hib) vaccine, the epidemiology of meningitis in the United States has changed dramatically. In 1986, the median age for all patients with meningitis was 15 months. In 1995, the median age was 25 years.[11] Meningitis has shifted from being predominantly a disease of infants and young children to being a disease predominantly of adults.

PATHOPHYSIOLOGY

- Typically, meningitis is a complication of primary bacteremia. It is thought that the products of bacterial multiplication alter the permeability of the blood-brain barrier and extend the infection to the brain and the surrounding cerebrospinal fluid spaces.
- Less commonly, meningitis may result from hematogenous spread from a distant primary focal infection, direct extension from an adjacent infection, or after cribriform plate or sinus fracture.
- The neurologic damage that sometimes follows meningitis is thought to result from direct in-

TABLE 72-2 Antibiotic Therapy for Sepsis and Meningitis

AGE	ANTIBIOTIC	DOSE
<1 month	Ampicillin and gentamicin* or ampicillin and cefotaxime	200–400 mg/kg/d divided q4–6h 7.5 mg/kg/d divided q8h 200–400 mg/kg/d divided q4–6h 200 mg/kg/d divided q6–8h
1–2 months	Ampicillin and gentamicin or ceftriaxone or cefotaxime	200–400 mg/kg/d divided q4–6h 7.5 mg/kg/d divided q8h 100 mg/kg/d divided q12–24h 200 mg/kg/d divided q6–8h
>2 months	Ceftriaxone or cefotaxime	100 mg/kg/d divided q12–24h 200 mg/kg/d divided q6–8h

* During the first week of life, reduce gentamicin dose to 5 mg/kg divided q12h.

flammatory effects, brain edema, increased intracranial pressure, decreased cerebral blood flow, and vascular thrombosis.
- Impaired splenic function and immunosuppression or immunodeficiency are associated with a relatively higher risk of meningitis.
- The bacterial agents responsible for meningitis vary with age. Group B streptococci, *E. coli,* and *L. monocytogenes* predominate in neonates. *Strep. pneumoniae* and *N. meningitidis* are most common in older infants and children.

CLINICAL FEATURES

- The presentation of meningitis is age-dependent.
- Neonates often present with nonspecific signs and symptoms. Symptoms may include decreased responsiveness, poor feeding, vomiting, fever (or normothermia or hypothermia), a bulging fontanelle, and apparent respiratory distress. Paradoxical irritability is present when an infant prefers lying still (resting the meninges) to being held or rocked.
- In infants outside the neonatal age range, generalized lethargy and a toxic appearance are typical. Nuchal rigidity generally is not appreciable until the patient reaches the toddler age group.
- Older children present more like adults, with headache, photophobia, neck stiffness, nausea, vomiting, and fever.
- *Neisseria meningitidis* meningitis may lead to a fulminant, rapid progression to shock and death over a period of hours.
- Seizures may present in as many as 25 percent of patients with bacterial meningitis and although usually generalized may be focal.[12]
- Pretreatment with oral antibiotics may mute the presenting symptoms and lead to a longer duration of symptoms before diagnosis.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of meningitis is made by analysis of cerebrospinal fluid (CSF) obtained from a lumbar puncture. A CSF leukocytosis with a preponderance of polymorphonucleocytes, a CSF protein greater than 100 mg/mL, and a CSF glucose level less than 50 percent of the blood glucose level are suggestive of a bacterial source of meningitis. A Gram's stain is considered 70 percent sensitive for identifying a causative bacterial agent.
- Other conditions that may present similarly to bacterial meningitis include sepsis without meningitis, intracranial mass lesions, aseptic meningitis, trauma, cardiac or respiratory failure, toxic ingestion, and metabolic abnormalities.
- If there is a CSF leukocytosis and the patient has previously been on antibiotics, bacterial antigen testing of the CSF may be critical to making an accurate diagnosis of partially treated meningitis.[13]
- Unusual organisms have a higher likelihood of causing meningitis in immunocompromised patients.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Critically ill children should be treated as was indicated in the section on sepsis, above.
- Rapid administration of antibiotics is critical to maximize the likelihood of a good neurologic outcome for the patient. In critically ill or toxic-appearing infants and children, antibiotic administration should not be delayed for computed tomographic (CT) scan of the head or lumbar puncture.
- The empirical antibiotic selection is based on the likely organism, which in turn is based on age. Doses are generally higher when meningitis is suspected to enhance drug penetration across the blood-brain barrier. Neonates should be given intravenous ampicillin and cefotaxime. Infants and children should be given intravenous cefotaxime or ceftriaxone. The use of vancomycin is somewhat controversial, but it should be given if cephalosporin-resistant pneumococcus is suspected in any patient outside the neonatal age group.[13,14]
- The use of steroids (dexamethasone) has been controversial, and their employment has decreased markedly because of the decreased incidence of *H. influenzae* type b. Steroids have been implicated in a worse neurologic outcome in patients with pneumococcal or meningococcal meningitis.[12]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 118, ''Bacteremia, Sepsis, and Meningitis in Children,'' by Peter Mellis.

73 PNEUMONIA IN CHILDREN

Lance Brown

EPIDEMIOLOGY

- Pneumonia is more common in early childhood than it is at any other age. The incidence of pneumonia decreases as a function of age (e.g., 40 per 1000 in preschool children and 9 per 1000 in 10-year-olds in North America).[1,2]
- Etiologic agents tend to have a seasonal variation. Parainfluenza virus tends to occur in the fall, respiratory syncytial virus (RSV) and bacteria in the winter, and influenza in the spring.
- Risk factors that increase the incidence or severity of pneumonia include prematurity, malnutrition, low socioeconomic status, passive exposure to smoke, and day care attendance.

PATHOPHYSIOLOGY

- Pneumonias occur when lung tissue becomes inflamed. This inflammation typically is due to aspirated virus or bacteria, but inhaled irritants also may cause pneumonia.
- Protective mechanisms against the development of pneumonia include nasal entrapment of aerosolized particles, mucus and ciliary movement in the upper respiratory tract, laryngeal reflexes and coughing, alveolar macrophages, the activation of complement and antibodies, and lymphatic drainage. Any derangement of these protective mechanisms leads to an increased risk for pneumonia.
- A viral upper respiratory tract infection often precedes bacterial pneumonia, and the coexistence of viral and bacterial pathogens has been seen in more than 50 percent of cases.[3,4]

CLINICAL FEATURES

- Clinical features are dependent primarily on the age of the patient. Other factors include the specific respiratory pathogen, the severity of the disease, immunosuppressive therapy, and any underlying illnesses.
- Infants with pneumonia typically present with a sepsis syndrome. The signs and symptoms are nonspecific and include fever or hypothermia, apnea, tachypnea, poor feeding, vomiting, diarrhea, lethargy, grunting, bradycardia, and shock.[5,6] Neonates are the only developmental group in which bacterial infections are more common than are viral infections.
- In infants younger than 2 years of age, tachypnea is sensitive for pneumonia but is not specific.[7] Examination findings include rales, wheezing, retractions, increased work of breathing, grunting, paradoxical breathing, and fever. Abdominal distention and poor feeding also may be present.[7,8]

TABLE 73-1 Common Organisms Causing Pediatric Pneumonia

AGE GROUP	ORGANISMS*
Newborn	Group B streptococci Gram-negative bacilli *Listeria monocytogenes* Herpes simplex Cytomegalovirus Rubella
0.5–4 months	Viruses *Chlamydia trachomatis* *Streptococcus pneumoniae* *Haemophilus influenzae*
4 months–4 years	*Staphylococcus aureus* Viruses *Streptococcus pneumoniae* *Haemophilus influenzae*
5–17 years	*Staphylococcus aureus* *Mycoplasma pneumoniae* Viruses *Streptococcus pneumoniae*

* Listed from top to bottom by greatest to lowest frequency of occurrence.

Posttussive vomiting may contribute to dehydration.

- In older children, the clinical presentation is more like that in adults. Classically, two presentations are seen: typical and atypical pneumonia. Typical pneumonia is characterized by the abrupt onset of fever, chills, pleuritic chest pain, localized findings on chest examination, and a toxic appearance. Sputum production may be seen in children older than 8 years of age. Atypical pneumonia is characterized by gradual onset, headache, malaise, nonproductive cough, low-grade fever, wheezing, rhinitis, conjunctivitis, pharyngitis, and rash. Although classically it was thought that bacterial agents cause typical pneumonia and viral agents cause atypical pneumonia, there is a significant overlap.[9]

DIAGNOSIS AND DIFFERENTIAL

- Several conditions may present similarly to pneumonia, including congestive heart failure, atelectasis, tumors, pulmonary congenital anomalies, aspiration pneumonitis, poor inspiration or technical difficulties with the chest x-ray, allergic alveolitis, chronic pulmonary diseases (e.g., cystic fibrosis), and congenital abnormalities such as pulmonary sequestration.
- Chest x-rays commonly are used to make the diagnosis of pneumonia. Consolidation on chest x-ray is considered a reliable sign of pneumonia.[10] Viral pneumonias tend to have diffuse interstitial infiltrates with hyperinflation, peribronchial thickening or cuffing, and areas of atelectasis. Bacterial pneumonias tend to have lobar or segmental infiltrates. However, there is an overlap, and identifying the etiologic agent by chest x-ray is only somewhat reliable (42 to 80 percent sensitive and 42 to 100 percent specific).[7,11,12]
- Blood cultures are positive in about 10 percent of children with proven bacterial pneumonia.[3,6,13]
- Sputum cultures may be diagnostic but are difficult to obtain in young children who are not intubated or do not have a tracheostomy.
- Nasopharyngeal or throat cultures may reveal the causative agent when chlamydia, pertussis, mycoplasma, or a viral pathogen is isolated. Rapid viral antigen tests are available for RSV and influenza. These tests do not play a role in identifying bacterial etiologies of pneumonia.
- Leukocytosis with a left shift is typical of bacterial pneumonia.[14]

TABLE 73-2 Antibiotic Therapy for Children with Pneumonia

AGE GROUP	INPATIENT THERAPY	OUTPATIENT THERAPY
0–1 month	Ampicillin and gentamicin or ampicillin and cefotaxime	N/A
1–3 months	Pneumonitis syndrome: erythromycin or clarithromycin	N/A
	Other: cefuroxime	N/A
3 months–5 years	Cefuroxime (consider adding erythromycin or clarithromycin)*	Amoxicillin, erythromycin, or clarithromycin
6–18 years	Erythromycin or clarithromycin (consider adding cefuroxime)*	Erythromycin, clarithromycin, or azithromycin
All ages	Add vancomycin if resistant *Streptococcus pneumoniae* is suspected	

* Add additional coverage in severely ill patients.

- The likelihood of various etiologic agents is age-dependent (see Table 73-1).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- General care of a pediatric patient with pneumonia includes assessment of and treatment for hypoxia, dehydration, and fever. In children with significant bronchospasm and wheezing, bronchodilators are suggested.
- Empirical antibiotic selection is based on the likely etiologic agents, which have a specific age distribution (see Table 73–2).
- Indications for admission include age less than 3 months, toxic appearance, respiratory distress, oxygen requirement, dehydration, vomiting, failed outpatient therapy, an immunocompromised state, and a noncompliant or unreliable caretaker. Admission to the pediatric intensive care unit should be considered for children with severe respiratory distress or impending respiratory failure.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 119, ''Viral and Bacterial Pneumonia in Children,'' by Kathleen Brown and Thomas E. Terndrup.

74 ASTHMA AND BRONCHIOLITIS

Jonathan L. Jones

ASTHMA

EPIDEMIOLOGY

- Asthma affects approximately 10 percent of the pediatric population.[1]
- The percentage of patients with adverse outcomes (intubation, need for cardiopulmonary resuscitation, and death) tripled between 1986 and 1993.
- Risk factors associated with development of asthma in children include low birth weight, family history of asthma, urban household, low income household, and race (children of African American, Asian, and Hispanic descent).[2,3]

PATHOPHYSIOLOGY

- Asthma is classified as extrinsic (IgE mediated), intrinsic (infection induced), and mixed (both IgE and infection induced).
- Allergens and irritants are the most common triggers of asthma in children above 2 years of age. Viral respiratory infections trigger asthma in those below age 2.
- Asthma is a two-stage process: (1) bronchocon-

TABLE 74-1 Risk Factors Associated with Asthma Death

Intubation for asthma
Two or more hospitalizations, three or more ED visits in past year
Hospitalization or ED visit in past month
Syncope or hypoxic seizure with asthma
Recent steroid use or dependence
Increased use of β_2 agonists
Poor access to health care and/or psychosocial problems

striction due to histamine and leukotriene release (early stage) and (2) airway mucosal edema with mucous plugging (late stage).
- Compensatory hyperventilation may cause a fall in $Paco_2$ and respiratory alkalosis. More severe obstruction and inadequate alveolar ventilation ultimately result in marked CO_2 retention, respiratory acidosis, and respiratory failure. Pseudonormalization of $Paco_2$ is therefore ominous.
- Pediatric asthma patients are at greater risk of respiratory failure than adult asthma patients because of anatomic differences. Young lung tissue lacks elastic recoil and is more prone to atelectasis. Airway walls are thicker and thus have greater narrowing with bronchoconstriction.[4] Risk factors for asthma-related death is listed in Table 74-1.

CLINICAL FEATURES

- Wheezing is the most common symptom of asthma.
- In cases of severe bronchospasm, auscultation may reveal only decreased breath sounds.
- Persistent nonproductive cough or exercise induced cough may be the result of bronchospasm.
- The amount of air movement, retractions, nasal flaring, and accessory muscle use usually reflect the severity of the asthma attack.
- Cyanosis, altered mental status, and somnolence may indicate respiratory failure. Bradycardia and shock herald impending cardiac arrest.

DIAGNOSIS AND DIFFERENTIAL

- Chest x-ray usually reveals hyperinflation and flattening of the diaphragm.
- Indications for chest x-ray in asthma include a first episode of wheezing, unilateral wheezing or rales, and fever.
- Measuring a peak expiratory flow rate may be useful in children over 4 years of age. Peak expiratory flow rate <50 percent of the predicted value indicates severe obstruction.
- Hypercarbia on arterial blood gas measurement may be the initial sign of respiratory failure.
- The most common cause of wheezing in infants and young children less than 3 years of age is bronchiolitis.
- Other causes of wheezing include bronchopulmonary dysplasia, congestive heart failure, gastroesophageal reflux, vascular rings, bronchial stenosis, mediastinal cysts, cystic fibrosis, pneumonia, and aspiration of foreign body.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Albuterol can be administered as episodic treatments at 0.15 mg/kg per dose q 20 min or as a continuous nebulization up to 0.5 mg/kg/h.
- Oxygen should be administered if oxygen saturation is below 94 percent.
- Steroids can prevent progression of an attack, decrease incidence of emergency department visits and hospitalization, and reduce rates of morbidity. Steroids may be given as prednisone or prednisolone 1 to 2 mg/kg per day and, if given for 5 days or less, need not be tapered.[5,6]
- Ipratropium should be considered for patients with severe distress or those who do not respond readily to albuterol alone.[7,8]
- Magnesium sulfate 50 to 75 mg/kg (maximum dose 2 g) intravenously (IV) over 20 min may benefit a subset of children with severe exacerbation.[9,10]
- Helium-oxygen (Heliox) may benefit children with severe exacerbation by decreasing airway resistance and work of breathing.[11]
- Intravenous fluids may be required in patients with status asthmaticus because of increased insensible water loss and decreased oral intake.
- If mechanical ventilation is required, low inflating pressures and long expiratory times may reduce the risk of barotrauma.
- Ketamine (1 to 2 mg/kg IV) is a useful induction agent for intubation due to its bronchodilating effects.

BRONCHIOLITIS

EPIDEMIOLOGY

- Bronchiolitis occurs typically during fall to early spring.

- Infants less than 2 years old are most commonly affected. The peak incidence in urban populations is 2 months of age.
- Young infants (under 2 months of age) and those with a history of prematurity, bronchopulmonary dysplasia, congenital heart disease, or immunosuppression are at increased risk of complicated courses of the disease.
- The infectious agent is highly contagious and is transmitted by direct contact with secretions and self-inoculation by contaminated hands via the eyes and nose.

PATHOPHYSIOLOGY

- Respiratory syncytial virus causes 50 to 70 percent of clinically significant bronchiolitis.[12]
- Non-respiratory syncytial virus bronchiolitis is caused by influenza virus, parainfluenza virus, echovirus, rhinovirus, mycoplasma pneumoniae, and chlamydia trachomatis.
- Mucous plugging results from necrosis of the respiratory epithelium and destruction of ciliated epithelial cells. This and submucosal edema lead to peripheral airway narrowing and variable obstruction.
- Increased airway resistance and decreased compliance result in increased work of breathing.

CLINICAL FEATURES

- Wheezing is the prominent clinical manifestation. Symptoms of upper respiratory infection will precede the respiratory distress.
- Most infants will have fever. Tachypnea, retractions, nasal flaring, and grunting may be present.
- Decreased breath sounds or absence of breath sounds signifies severe bronchoconstriction. Cyanosis and altered mental status are ominous signs of respiratory failure.

DIAGNOSIS AND DIFFERENTIAL

- Chest x-ray is recommended in all children with the first episode of wheezing. The chest x-ray may show hyperinflation and peribronchial cuffing.
- Pulmonary consolidation on the chest x-ray may reflect primary pneumonia or superinfection.
- Identification of respiratory syncytial virus can be made from nasal washings using fluorescent monoclonal antibody testing.
- Initial pulse oximetry reading is recommended in all children with respiratory distress with continuous pulse oximetry done in those with initial pulse oximetry reading <93 percent.
- Complete blood cell count and blood culture are generally not helpful.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Children with bronchiolitis may respond to an inhaled β agonist (albuterol 0.15 mg/kg per dose). If improvement occurs, treatments may be repeated as needed.
- Nebulized epinephrine (1 : 1000) 0.5 mL in 2.5 mL normal saline may be beneficial if albuterol fails. Epinephrine may be repeated every 2 h.[13]
- Helium-oxygen (heliox) should be considered for children with severe symptoms but should not be used in patients with an oxygen requirement >40 percent.[14]
- Dehydration from increased insensible water loss may require IV fluid therapy.
- Corticosteroids are not indicated in bronchiolitis unless there is a history of underlying reactive airway disease.[15]
- Indications for hospitalization include (1) apnea, (2) respiratory distress unresponsive to treatment, (3) hypoxia, and (4) vomiting and/or dehydration.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 120, ''Pediatric Asthma and Bronchiolitis,'' by Maybelle Kou and Thom A. Mayer.

75 SEIZURES AND STATUS EPILEPTICUS IN CHILDREN

David M. Cline

- Both the causes and the manifestations of seizure activity are numerous, ranging from benign to life-threatening.
- Although the majority of seizures are idiopathic in nature (e.g., epilepsy), risk factors include encephalitis, disorders of amino acid metabolism, structural abnormalities (e.g., hydrocephalus, microcephaly, and arteriovenous malformations), congenital infections, and neurocutaneous syndromes (e.g., tuberous sclerosis, neurofibromatosis, Sturge-Weber syndrome).
- Precipitants of seizures can include fever, sepsis, hypoglycemia, hypocalcemia, hypoxemia, hyper- or hyponatremia, hypotension, toxin or medication exposure, and head injury.

EPIDEMIOLOGY

- Approximately 2 percent of the U.S. population have some form of epilepsy.
- In children from birth to 9 years of age, the prevalence is 4.4 cases per 1000, and in those aged 10 to 19 years, the prevalence is 6.6 cases per 1000.
- Simple febrile convulsions constitute a separate category, with an incidence of 3 to 4 percent in children.

PATHOPHYSIOLOGY

- A seizure is an abnormal, sudden, and excessive electric discharge of neurons (gray matter) that propagates down the neuronal processes (white matter) to affect end organs in a clinically measurable fashion.

CLINICAL FEATURES

- Symptoms of seizure may include any of the following: loss of or alteration in consciousness, including behavioral changes and auditory or olfactory hallucinations; involuntary motor activity, including tonic or clonic contractions, spasms, or choreoathetoid movements; and incontinence.
- Signs could include alteration in consciousness or motor activity; autonomic dysfunction, such as mydriasis, diaphoresis, hypertension, tachypnea or apnea, tachycardia, and salivation; and postictal somnolence.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of seizure disorder is based primarily on history and physical examination, with laboratory studies (other than a bedside assay for glucose) obtained in a problem-focused manner.[1]
- In patients with breakthrough seizures or status epilepticus, determinations of drug levels in serum are useful for some antiepileptic agents (Table 75-1), while others, such as gabapentin, lamotrigine, topiramate, tiagabine, and vigabatrin may not

TABLE 75-1 Therapeutic Antiepileptic Drug Levels, μg/mL

DRUG	TOTAL	FREE
Phenytoin	10–22	1.0–2.2
Phenobarbital	15–20	NA
Carbamazepine	6–12	1.8–2.2
Primidone	5–12	NA
Valproic acid	50–130	10–25
Ethosuximide	50–100	NA

ABBREVIATION: NA = not applicable.

be immediately available or useful in guiding therapy.

- Serum chemistry studies (i.e., electrolytes, magnesium, calcium, creatinine, and blood urea nitrogen levels) are usually not indicated except in neonatal seizures, infantile spasms, febrile seizures that are complex in nature (with duration over 15 min, focal involvement, or several recurrences in 24 h), status epilepticus, or suspected metabolic or gastrointestinal disorders.[2]
- Serum ammonia, TORCH (toxoplasmosis, other agents, rubella, cytomegalovirus, herpes simplex) titers, and urine and serum amino acid screening may be useful in neonatal seizures.
- Blood gas analysis is indicated in neonatal seizures and status epilepticus.
- Cardiac monitoring is useful to assess the PR and QT intervals and the possibility of cardiac dysrhythmia as the precipitant of seizure.
- Magnetic resonance imaging is the preferred neuroimaging procedure for most cases of new-onset seizures, whereas cerebral ultrasound is useful in neonates, and immediate noncontrast computed tomography is indicated in cases of head trauma, nonfebrile status epilepticus, and focal seizures or focal neurologic signs.[2]
- Lumbar puncture should be performed in patients with neonatal seizure, infantile spasms, complex febrile seizures under 18 months of age, meningeal signs, or persistent alteration in consciousness.
- Emergent electroencephalographic (EEG) monitoring is indicated for neonatal seizures, nonconvulsive status epilepticus, and refractory status epilepticus, especially when a paralytic agent is used.
- It is important to differentiate true seizure activity from one of several nonepileptic paroxysmal disorders, such as neonatal jitteriness, hyperexplexia (startle disease), near-miss sudden death syndrome, breath-holding spells (of cyanotic or pallid types), hyperventilation, syncope, migraine, hysterical pseudoseizures, narcolepsy, cataplexy, night terrors, vertigo, Tourette's syndrome, chorea, or paroxysmal choreoathetosis, which are characterized by normal EEGs and are unresponsive to antiepileptic drugs.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial management should include (1) airway maintenance (supplemental oxygen, suctioning, airway opening, or intubation when necessary), (2) seizure termination, (3) correction of reversible causes, (4) initiation of appropriate diagnostic studies, and (5) arrangement of follow-up or admission, as appropriate.
- Termination of seizure activity is important to prevent irreversible pathologic changes and risk of persistent seizure disorder,[3] especially in the setting of status epilepticus, defined as one seizure greater than 20 min in duration or a series of seizures greater than 30 min without interictal awakening.[4] For this reason, seizures lasting greater than 10 min are treated as status epilepticus.

- **FIRST SEIZURE** Patients with prolonged or repetitive witnessed seizures, especially with concomitant neurologic deficit, are started on antiepileptic drugs. Although any antiepileptic agent may be used, the decision is based on side-effect profile, experience, and ease of administration.
- Carbamazepine 10 to 40 mg/kg/d in two to four daily doses, phenytoin 4 to 8 mg/kg/d in two to three daily doses, or phenobarbital 3 to 8 mg/kg/d in one to two daily doses is commonly used for partial seizures, and valproate 20 to 60 mg/kg/d in two to four daily doses is commonly used for generalized seizures.
- Felbamate 45 mg/kg/d in three daily doses or gabapentin 20 to 30 mg/kg/d in three daily doses is used for complex partial seizures.
- Ethosuximide 20 to 30 mg/kg/d in two to three daily doses, lamotrigine 5 to 15 mg/kg/d in one to two daily doses, or valproate is used for absence seizures (after confirmatory EEG).
- IV loading can be achieved with the IV form of valproate 10 to 30 mg/kg over 15 min or fosphenytoin 15 to 20 mg phenytoin equivalents (PE)/kg at 3 PE/kg/min, a phenytoin prodrug without infusion-related complications.

- **FEBRILE SEIZURE** Identification and treatment of the cause of fever is the primary goal of therapy

for febrile seizures.[5,6] Fever can be controlled by acetaminophen or ibuprofen and tepid water baths.
- Antiepileptic drug therapy with oral phenobarbital or valproate should be considered in patients at high risk of recurrence, such as those with an underlying neurologic deficit (e.g., cerebral palsy), complex (prolonged or focal) febrile seizures, repeated seizures in the same febrile illness, onset under 6 months of age, or more than three febrile seizures in 6 months.[7,8]

- **Neonatal Seizures** The cause of neonatal seizures should be investigated and treated aggressively in an intensive care setting.
- Persistent or uncertain cause of seizures should be treated with empiric IV pyridoxine (100 mg/d); hypoglycemia with 25% glucose solution 2 mL/kg IV or 10% glucose 3 mL/kg in neonates; hypocalcemia with calcium gluconate 4 mL/kg or 200 mg/kg of 5% solution IV and magnesium sulfate 0.2 mL/kg of 2% solution IV or 0.2 mL/kg of 50% solution intramuscularly (IM); and biotinidase deficiency with biotin 10 mg/d.
- The first-line agent is IV phenobarbital 20 mg/kg at 1 mg/kg/min followed by 3 to 4 mg/kg/d).[9]

- **Infantile Spasms** Therapy with adrenocorticotropic hormone (ACTH; or with clonazepam or valproate) is often started in the inpatient setting after specialty consultation. Glucose transporter defect syndrome [diagnosed by lumbar puncture (LP)] is treated with a ketogenic diet.

- **Head Trauma and Seizures** Immediate seizures following head trauma may require short-term treatment with fosphenytoin, especially following severe head injury.[10] Early and late post-traumatic seizures may require long-term antiepileptic therapy if recurrent.[11]

- **Status Epilepticus** Airway maintenance is of primary importance in status epilepticus because all therapeutic agents can result in respiratory depression.
- With IV access, lorazepam 0.1 mg/kg to a total of 8 mg, diazepam 0.2 to 0.5 mg/kg to a total of 2.6 mg/kg, or midazolam 0.2 mg/kg is the primary agents of choice.[12–14]
- Without IV access, alternatives include rectal, nasal, or IM midazolam 0.1 to 0.2 mg/kg, rectal diazepam 0.5 mg/kg; rectal valproic acid 60 mg/kg; or intraosseous (IO) infusion of lorazepam, diazepam, or midazolam (in similar dosages as IV).[15,16]
- Phenobarbital 20 to 30 mg/kg IV or IO repeated 10 mg/kg every 20 min to levels of 60 μg/mL should be started immediately after the primary agent, followed by fosphenytoin 20 mg PE/kg IV or IO if phenobarbital is ineffective.
- If seizures persist after fosphenytoin, consider continuous midazolam IV infusion 0.04 to 0.05 mg/kg/h or general anesthesia (along with continuous EEG monitoring) with pentobarbital 2 mg/kg bolus followed by 1 to 2 mg/kg/h IV infusion or inhalational agents.[12]
- Consider treatable causes such as hypoglycemia, hyponatremia, toxin exposure (e.g., iron, lead, carbon monoxide, salicylates, stimulants, etc.) or infections (e.g., meningoencephalitis or brain abscess). Specific toxicologic therapy (e.g., activated charcoal, hyperbaric oxygen, or chelation therapy) should be used where appropriate for suspected toxin exposure.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 121, "Seizures and Status Epilepticus in Children," by Michael A. Nigro.

76 VOMITING AND DIARRHEA IN CHILDREN

David M. Cline

EPIDEMIOLOGY

- In the United States, children younger than 3 years of age have 1.3 to 2.3 episodes of diarrhea each year. The prevalence is higher in children attending day care centers.
- Up to one-fifth of all acute-care outpatient visits to hospitals are by families with infants or children affected by acute gastroenteritis, and 9 percent of all hospitalizations of children younger than 5 years of age are for diarrhea.[1]
- Most enteric infections are self-limited, but excessive loss of water and electrolytes, resulting in clinical dehydration, may occur in 10 percent and is life-threatening in 1 percent.[2]
- Pathogenic viruses, bacteria, or parasites may be isolated from nearly 50 percent of children with diarrhea. Viral infection is the most common cause of acute diarrhea. Bacterial pathogens may be isolated in 1 to 4 percent of cases.
- Rotaviruses, Norwalk viruses, the enteric adenoviruses, calicivirus, and astroviruses are the most recognized viral pathogens that affect children. Rotavirus is most common and potentially lethal dehydration in 0.75 percent of children younger than 2 years of age.[3]
- The major bacterial enteropathogens in the United States are *Campylobacter jejuni, Shigella* species, *Salmonella* species, *Yersinia enterocolitica, Clostridium difficile, Aeromonas hydrophila,* and *Escherichia coli.*
- *Giardia lamblia* is a common cause of diarrhea in infants and young children in day care centers. As many as 50 percent of infected children may be asymptomatic.

PATHOPHYSIOLOGY

- Viral pathogens cause disease by tissue invasion and alteration of intestinal absorption of water and electrolytes.
- Bacterial pathogens cause diarrhea by the production of enterotoxins and cytotoxins and invasion of the mucosal absorptive surface.
- Dysentery occurs when bacteria invade the mucosa of the terminal ileum and colon, producing diarrhea with blood, mucus, or pus. Table 76-1 lists common causative agents, clinical features, and treatment for diarrhea in children.

CLINICAL FEATURES

- Evaluation of a child's state of hydration is most important. If possible, it is best to determine the degree of fluid loss by comparing the child's current weight to a recent previous weight.
- When objective measurements are not available, the state of hydration can be assessed by physical examination. Combinations of physical signs—including general ill appearance, capillary refill of longer than 3 s, dry mucous membranes, and absent tears—are good predictors. The presence of two or more signs predicts 5 percent or greater dehydration, whereas three or more signs predict 10 percent or greater dehydration.[4]
- Severe dehydration accompanied by lethargy, hypotension, and delayed capillary refill requires immediate administration of parenteral fluids. Although capillary refill may be affected by conditions other than dehydration, it should be considered a sign of significant dehydration until proven otherwise.[5]

TABLE 76-1 Common Agents, Clinical Features, and Treatment of Diarrhea

AGENT	CLINICAL FEATURES	TREATMENT
Viral		
Rotavirus	Watery diarrhea, winter, most common agent	Rehydration
Enteric adenovirus	Watery diarrhea, concurrent respiratory symptoms	Rehydration
Norwalk	Watery diarrhea, epidemic, fever, headache, myalgias	Rehydration
Bacterial		
Campylobacter jejuni	Fever, abdominal pain, watery or bloody diarrhea, may mimic appendicitis, animal reservoir	Rehydration Erythromycin
Shigella	Fever, abdominal pain, headache, mucoid diarrhea	TMP-SMX or ampicillin
Salmonella	Fever, bloody diarrhea, animal reservoir; antibiotics prolong the carrier state	TMP-SMX if complicated
Escherichia coli		
Enterotoxigenic	Watery diarrhea	TMP-SMX
Enterohemorrhagic	Dysentery, associated with HUS	Rehydration; check CBC, BUN, creatinine
Vibrio cholerae	Rice-water diarrhea	TMP-SMX
Yersinia enterocolitica	Fever, vomiting, diarrhea, abdominal pain; may mimic appendicitis	Rehydration
Clostridium difficile	Recent antibiotic use	Metronidazole
Staphylococcus aureus	Food poisoning	Rehydration
Parasitic		
Giardia lamblia	Diarrhea, flatulence; exposure to day care centers; mountain streams	Rehydration Metronidazole
Entamoeba histolytica	Bloody, mucoid stools; hepatic abscess	Metronidazole

DOSES: ampicillin 50 (mg/kg)/d divided qid; erythromycin 40 (mg/kg)/d divided qid; metronidazole 30 (mg/kg)/d divided bid; TMP-SMX based on 8–12 (mg/kg)/d of the TMP component divided bid.
ABBREVIATIONS: bid = twice a day; BUN = blood urea nitrogen; CBC = complete blood count; qid = four times a day; HUS = hemolytic-uremic syndrome; TMP-SMX = trimethoprin-sulfamethoxazole.

DIAGNOSIS AND DIFFERENTIAL

- The most important aspect of diagnosis is a thorough history and physical examination. Selective laboratory testing may be useful if enteric pathogens are suspected.
- Dehydration caused by diarrhea is usually isotonic, and serum electrolyte determinations are not necessary unless signs of severe dehydration are present.
- Protracted vomiting and/or diarrhea in infants and toddlers may cause hypoglycemia. Blood glucose determinations are useful in this setting.
- The fecal leukocyte test, sometimes used as a screening tool, has poor sensitivity.[6]
- A febrile child with abrupt onset of diarrhea occurring more than four times per day or with blood in the stool is more likely to have an illness caused by a bacterial pathogen and stool cultures are indicated.[7]
- Vomiting and diarrhea may also be a nonspecific presentation for other disease processes, such as otitis media, urinary tract infection, sepsis, malrotation, increased intracranial pressure, metabolic acidosis, and drug or toxin ingestion.
- Infants under 1 year of age are at risk for rapid dehydration and hypoglycemia.
- Bilious vomiting in an infant under 2 years of age is worrisome and considered a sign of intestinal obstruction until proven otherwise.
- Special attention should be given to those children who have chronically debilitating illnesses, high-risk social situations, or malnutrition, since they are at particular risk for rapid decompensation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- If vomiting is the prominent symptom:
  1. Since most cases are self-limited, oral rehydration is generally all that is necessary.[8,9] Vomiting

is not a contraindication to oral rehydration with glucose-electrolyte solutions. The key is to give small amounts of the solution frequently.
2. If oral rehydration is not possible or not tolerated by the patient, IV rehydration with normal saline may be necessary.
3. Antiemetics are controversial and generally not recommended.[10] If they are used, the physician should be aware of potential adverse effects associated with these drugs, such as dystonic reactions.

- If diarrhea is the prominent symptom:
  1. Children with mild diarrhea who are not dehydrated may continue routine feedings.[11]
  2. Children with moderate to severe dehydration should first receive adequate rehydration before resuming routine feedings. Food should be reinstated after the rehydration phase is completed and never delayed more that 24 h. There is no need to dilute formula, since over 80 percent of children with acute diarrhea can tolerate full-strength milk safely.[11]
  3. Dietary recommendations include a diet high is complex carbohydrates, lean meats, vegetables, fruits, and yogurt. Fatty foods and foods high in simple sugars should be avoided. The BRAT diet (bananas, rice cereal, applesauce, and toast) is discouraged, since it does not provide adequate energy sources.
  4. Antimotility drugs are not helpful and should not be used to treat acute diarrhea in children.[10,12]
  5. Antibiotics are considered if the diarrhea has persisted longer than 10 to 14 days or the patient has a significant fever, systemic symptoms, or blood or pus in the stool.[13] (See Table 76-1 for antibiotic recommendations.)
- All infants and children who appear toxic or have high-risk social situations, significant dehydration, altered mental status, inability to drink, bloody diarrhea, or laboratory evidence of hemolytic anemia, thrombocytopenia, azotemia, or elevated creatinine levels should be admitted.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 122, "Vomiting and Diarrhea in Children," by Christopher M. and Ronald D. Holmes.

77 PEDIATRIC ABDOMINAL EMERGENCIES

David M. Cline

EPIDEMIOLOGY

- The causes of abdominal pain vary with age. See Table 77-1 for a listing of causes stratified by age.

TABLE 77-1 Etiology of Abdominal Pain

UNDER 2 YEARS	6–11 YEARS
Appendicitis*	Appendicitis*
Colic (first 4 months)	Diabetic ketoacidosis
Congenital abnormalities*	Functional
Gastroenteritis	Gastroenteritis
Incarcerated hernia*	Henoch-Schönlein purpura
Intussusception*	Incarcerated hernia*
Malabsorption	Inflammatory bowel disease
Malrotation	Obstruction
Metabolic acidosis*	Peptic ulcer disease*
Obstruction	Pneumonia*
Sickle cell pain crisis	Renal stones
Toxins*	Sickle cell syndrome
Urinary tract infection	Streptococcal pharyngitis
Volvulus*	Torsion of ovary or testicle
	Toxins*
	Trauma*
	Urinary tract infection
2–5 YEARS	**OVER 11 YEARS**
Appendicitis	Appendicitis*
Diabetic ketoacidosis*	Cholecystitis
Gastroenteritis	Diabetic ketoacidosis*
Hemolytic uremic syndrome*	Dysmenorrhea
Henoch-Schönlein purpura	Ectopic pregnancy*
Incarcerated hernia*	Functional
Intussusception*	Gastroenteritis
Malabsorption	Incarcerated hernia*
Metabolic acidosis*	Inflammatory bowel disease
Obstruction	Obstruction
Pneumonia*	Pancreatitis
Sickle cell pain crisis	Peptic ulcer disease*
Toxins*	Pneumonia*
Trauma*	Pregnancy
Urinary tract infection	Renal stones
Volvulus*	Sickle cell syndrome
	Torsion of ovary or testicle
	Toxins*
	Trauma*
	Urinary tract infection

* Life-threatening causes of abdominal pain.

PATHOPHYSIOLOGY

- See Chap. 38 for a discussion of the pathophysiology of abdominal pain.

CLINICAL FEATURES

- Presenting signs and symptoms will vary with the child's age. The key gastrointestional signs and symptoms are pain, vomiting, diarrhea, constipation, bleeding, jaundice, and masses. These symptoms can be the result of a benign process or may indicate a life-threatening illness.
- The origin of abdominal pain may be extraabdominal, as with pneumonia or pharyngitis.[1,2]
- Pain in children less than 2 years of age usually manifests as fussiness, irritability, or lethargy. Pain may be peritonitic and exacerbated by motion or obstructive, spasmatic, and associated with restlessness. Pain of gastrointestinal (GI) origin is usually referred to the periumbilical area in children 2 to 6 years old.
- Associated symptoms or the presence of illness in other family members may be useful in arriving at a diagnosis.
- Vomiting and diarrhea are common in children. These symptoms may be the result of a benign process or indicate the presence of a life-threatening process (see Chap. 76). Bilious vomiting is frequently indicative of a serious process.
- Constipation may be functional or pathologic. The shape and girth of the abdomen, presence of bowel sounds or masses, and abnormalities in the anal area should be noted.
- GI bleeding can be from upper or lower sources.[3] Upper sources are vascular malformation, swallowed maternal blood, bleeding diathesis, foreign body, peptic ulcer disease, and Mallory-Weiss tear. Lower GI bleeding can be from fissures, intussusception, hemolytic uremic syndrome, swallowed maternal blood, vascular malformations, polyps, inflammatory bowel disease, or diverticulum. The cause of minimal to moderate amounts of blood in the stool is frequently never identified.
- Jaundice outside of infancy is usually an ominous sign.

DIAGNOSIS AND DIFFERENTIAL

- The likely etiologies of abdominal pain change with age. Table 77-1 lists common causes of abdominal pain seen in various age groups and identifies those that are potentially life-threatening.
- It is clinically useful to split the most serious causes of GI emergencies in the first year of life from older children. Common emergencies in the first year of life include malrotation of the gut, incarcerated hernia, intestinal obstruction, pyloric stenosis, and intussusception.
- Malrotation of the gut, although rare, can present with a volvulus, which can be life-threatening.[4] Presenting symptoms are usually bilious vomiting, abdominal distention, and streaks of blood in the stool. The vast majority of cases present within the first month of life. Distended loops of bowel overriding the liver on abdominal radiographs are suggestive of this diagnosis.
- The symptoms of incarcerated hernia include irritability, poor feeding, vomiting, and an inguinal or scrotal mass. The mass will not be detected unless the infant is totally undressed. The incidence of incarcerated hernia is highest in the first year of life. It is possible to manually reduce the

hernia on examination in most cases (see Chap. 45).
- Intestinal obstruction may be caused by atresia, stenosis, meconium ileus, malrotation, intussusception, volvulus, incarcerated hernia, imperforate anus, and Hirschsprung's disease. Presentation includes irritability, vomiting, and abdominal distention, followed by absence of bowel sounds.
- Pyloric stenosis usually presents with nonbilious projectile vomiting occurring just after feeding. It is most commonly seen in the second or third week of life. It is familial and male-predominant, with first-born males being particularly affected. Palpation of the pyloric mass, or ''olive,'' in the left upper quadrant is diagnostic. Ultrasound may also aid in the diagnosis if pyloric stenosis is suspected clinically and a mass is not palpated.
- Intussusception occurs when one portion of the gut telescopes into another. GI bleeding and edema give rise to bloody mucus-containing stools, producing the classic ''currant jelly'' stool.[5] The greatest incidence is between 3 months and 6 years of age. The classic presentation is sudden epigastric pain with pain-free intervals during which the examination can reveal the classic sausage-shaped mass in the right side of the abdomen. The presentation may involve mental status changes.[6,7] This mass is present in up to two-thirds of patients. A barium enema or insufflation can be both diagnostic and therapeutic, since the intussusception is reduced while doing the procedure in 80 percent of cases.[8]
- Common GI emergencies in children 2 years of age and older include appendicitis, bleeding, Meckel's diverticulum, colonic polyps, and foreign bodies.
- Appendicitis may present with the classic symptoms of pain, fever, and anorexia; however, presentation may be extremely varied, making the diagnosis quite challenging.[9] Guarding and rebound may or may not be found on examination, the temperature may be normal, the white blood cell count may be normal, the child may be asking for food and may not be anorexic, and associated gastroenteritis is fairly common.[10] Appendicitis is seen in children younger than 1 year, and the perforation rate is higher in this age group due to the difficulty of making the diagnosis and frequent confusion with gastroenteritis.
- GI bleeding can be caused by several sources. Upper GI bleeding usually results from peptic ulcer disease, gastritis, or varices. Lower GI bleeding can be due to infectious colitis, inflammatory bowel disease, coagulopathies, hemolytic-uremic syndrome, and Henoch-Schönlein purpura. A small amount of blood in the diaper is most likely related to anal fissure or ingested foodstuffs.
- Portal hypertension, although rare, is one of the common causes of major upper GI bleeding and is associated with congenital liver disease and biliary atresia.
- Colonic polyps can be single or multiple or may represent classic familial polyposis. They can give rise to painless bright red lower GI bleeding. A single polyp is most common and frequently is palpated by the mother or noticed as a mass protruding from the anus.
- Foreign bodies in the GI tract are frequently seen in young children (see Chap. 41). Laxatives are contraindicated. Any foreign body caught in the esophagus must be removed by esophagoscopy.
- Pancreatitis is increasing in incidence in childhood.[11] The most common cause is abdominal trauma followed by a postviral process or drugs and toxin exposure; it may also be idiopathic.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- If the child is critically ill, resuscitation efforts should begin immediately, and the examination can be done concurrently.
- Remove all clothing prior to examination. The examination should always include a rectal examination and testing of stool for occult blood.
- The most important laboratory studies are complete blood count with differential, urinalysis, and guaiac test for occult blood. Other tests should be guided by how ill-appearing the child is. Determinations of electrolyte and amylase levels and pregnancy test may be indicated.
- Chest and abdominal radiographs can be useful to diagnose pneumonia, obstruction, or ileus. Abdominal ultrasound is useful in assessment of pyloric stenosis, ectopic pregnancy, or appendicitis.[12] Abdominal computed tomography scan may be diagnostic with abdominal masses and appendicitis.[13]
- In some cases dehydration and electrolyte abnormalities may require correction with oral or intravenous rehydration.

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78 THE DIABETIC CHILD AND DIABETIC KETOACIDOSIS

Leslie McKinney

EPIDEMIOLOGY

- Insulin-dependent diabetes mellitus (IDDM) is the most common endocrine disorder of childhood.[1]
- Occurrence peaks in early to mid-puberty, and more cases are reported in the cooler months.[2]
- Diabetic ketoacidosis (DKA) is the single most common etiology of death in patients with diabetes under 24 years.[3]

PATHOPHYSIOLOGY

- Insulin-dependent diabetes mellitus is an autoimmune disease caused by destruction of insulin producing β cells of the islets of Langerhans in the pancreas.[4]
- Genetic predisposition exists for IDDM, although there is no single gene.
- Diabetic ketoacidosis is caused by insulin deficiency (Fig. 78-1). The resultant elevation of counterregulatory hormones (glucagon, cortisol, growth hormone, epinephrine, and norepinephrine) antagonize the effects of insulin and lead to increased glucose production.[2] Ensuing glucosuria causes an osmotic diuresis resulting in the loss of fluids and electrolytes. Dehydration, compensatory polydipsia, and hyperosmolality occur as a result of the fluid losses.
- The hormonal interplay of the lack of insulin and excess glucagon levels leads to increased production of ketone bodies from free fatty acids. This increased production of ketone bodies, primarily β-hydroxybutyrate and acetoacetate exceeds the capacity for peripheral utilization contributing to the development of metabolic acidosis and compensatory respiratory alkalosis. The presence of increased ketones and acidemia manifest as the classic fruity breath odor of ketosis.

CLINICAL FEATURES

- Insulin-dependent diabetes mellitus is typically characterized by polyuria, polydipsia, and polyphagia; however, other common complaints include failure to gain weight, weight loss, enuresis, anorexia, changes in vision, and school performance.
- Diabetic ketoacidosis should be considered in patients with hyperventilation, fruity breath odor of ketosis, dehydration, lethargy, hyperglycemia, vomiting, abdominal pain, or polyuria.

DIAGNOSIS AND DIFFERENTIAL

- Diabetic ketoacidosis is defined by hyperglycemia (blood glucose >250 mg/dl), ketonemia, and metabolic acidosis (pH <7.2 and plasma bicarbonate level <15 meq/L) associated with glucosuria and ketonuria.
- Laboratory tests required to manage and diagnose DKA include serum electrolytes, urinalysis, blood pH, and serum ketone determination.
- Sepsis, trauma, vomiting, noncompliance, and

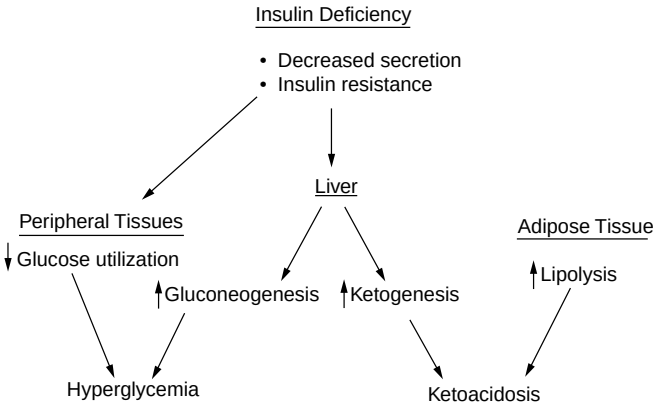

FIG. 78-1 Pathophysiology of diabetic ketoacidosis.

overall stress should be considered when the cause of DKA is not apparent.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of DKA consists of volume replacement, insulin therapy, correction of electrolyte abnormalities, and a search for a causative factor. Patients should be placed on a cardiac monitor, noninvasive blood pressure device, and pulse oximetry and intravenous lines should be established.
- Initially, hourly monitoring of electrolytes and pH is necessary.
- The total fluid deficit should be calculated by comparing the patient's presenting weight to a recent weight. If this is not available, a 10 percent (100 mL/kg) deficit should be assumed. Volume replacement using a normal saline infusion of 10 to 20 mL/kg over 1 to 2 h should be given initially to most patients.
- If evidence of shock is present consider a 20 mL/kg bolus of normal saline (NS). After initial stabilization is complete, the remaining fluid deficit should be replaced over 24 to 48 h using 0.45% NS, unless serum osmolality remains > 320 mosm/L. In this case, the NS should be continued until the osmolality approaches normal.
- Monitor glucose levels closely and begin 0.45% NS when blood glucose levels are between 300 to 250 mg/dL.
- A regular insulin infusion of 0.1 U/kg/h should be initiated as soon as a glucose level of >250 mg/dL is obtained. There is debate regarding an initial insulin bolus of 0.1 U/kg and most authorities begin with a continuous infusion. If the acidosis has not improved after 2 h of insulin therapy the insulin infusion should be increased to 0.15 to 0.2 U/kg/h. Both the insulin infusion and 0.45% NS should be continued until the acidosis is corrected.
- Restoration of sodium levels is accomplished by administration of NS and 0.45% NS fluid. Patients typically reveal sodium deficits of approximately 6 meq/kg. Also, the hyperglycemia and hyperlipidemia associated with DKA cause a falsely low serum sodium level. Serum sodium levels should be monitored closely as a decline of the sodium level is sometimes indicative of developing cerebral edema.
- Management of potassium abnormalities are critical to the care of DKA patients. Because of the shift of potassium to the extracellular space secondary to the acidosis of DKA, falsely elevated serum [K+] levels may be seen despite total body depletion. If the pH is 7.10 or less and the [K+] is normal or low, replacement therapy should begin immediately by adding 40 meq of [K+] to each liter of maintenance fluid. Doses as high as 60 meq/L should be considered if the potassium level is <3.0 meq/L. If the [K+] level is elevated (>6.0 meq/L) holding [K+] therapy until urine output is present and [K+] is correcting should be considered. One-half KCL and one-half KPO_4 should be used. Calcium levels should be monitored as excess phosphate can cause hypocalcemia.
- Bicarbonate therapy remains controversial and should be used only in life-threatening situations, such as cardiac dysrhythmias or dysfunction.
- A potentially fatal complication of DKA in children is development of cerebral edema. This typically occurs 6 to 10 h after initiating therapy and presents as mental status changes progressing to coma. Although the etiology of this complication is unknown, it is felt that several factors may contribute including overly aggressive fluid therapy,

rapid correction of blood glucose levels, bicarbonate therapy, and failure of the serum sodium level to increase with therapy. Treatment should include mannitol 1 to 2 g/kg, intracranial pressure monitoring, possible intubation with hyperventilation, and fluid restriction.
- Most of these patients will require admission to a pediatric intensive care unit. Consultation with the patient's primary care physician should be made early in the course of therapy.

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79 HYPOGLYCEMIA IN CHILDREN

Lance Brown

EPIDEMIOLOGY

- Hypoglycemia in children presenting to the emergency department (ED) is a relatively rare event. In one study, hypoglycemia had an incidence of only 6.54 cases per 100,000 pediatric ED visits.[1]
- With the exception of perinatal hypoglycemia, idiopathic ketotic hypoglycemia is by far the most common cause of hypoglycemia (58 percent of cases) in children presenting to the ED.[1]
- The most common drugs associated with clinically significant hypoglycemia in children are insulin, sulfonylurea-type medications, and ethanol.

PATHOPHYSIOLOGY

- Several factors make young children predisposed to hypoglycemia. These include a relatively high rate of glucose utilization, a higher basal metabolic rate, ongoing utilization of glucose for growth and development, greater degrees of physical activity, and relatively smaller glycogen stores.
- The brain is relatively larger in young children than it is in older children and adults. The brain is essentially dependent on glucose for its metabolism. In the fasted child, more than 80 percent of glucose utilization is by the brain.
- As glucose levels fall, a counterregulatory response is generated, which includes the release of glucagon, cortisol, growth hormone, and epinephrine. The release of these substances leads to a stimulation of gluconeogenesis. The clinical effects of the release of epinephrine is called the adrenergic response.

CLINICAL FEATURES

- Clinical features of hypoglycemia can be markedly varied, but can generally be divided into those due to neuroglycopenia and those due to the adrenergic response.
- Neurologic symptoms associated with hypoglycemia include confusion, ataxia, depressed consciousness, blurred vision, focal neurological deficits, and seizures.
- Symptoms of the adrenergic response include anxiety, tachycardia, perspiration, tremors, pallor, weakness, abdominal pain, and irritability.
- In neonates and infants the symptoms are usually less specific and more difficult to classify. These symptoms include poor feeding, jitteriness, emesis, ravenous hunger, lethargy, altered personality, repetitive colic-like symptoms, hypotonia, and hypothermia.
- Hypoglycemia often accompanies a critical illness (e.g., meningococcemia) and the features of that illness may dominate the clinical picture.

DIAGNOSIS AND DIFFERENTIAL

- The level at which one formally makes the diagnosis of hypoglycemia is controversial. It is generally accepted that a plasma glucose concentration of less than 60 mg/dL constitutes hypoglycemia in school-aged children, adolescents, and adults.[2] In the newborn and infant there is greater contro-

TABLE 79-1 Conditions Associated with Hypoglycemia in Infants and Children

PERINATAL PERIOD	INFANCY AND CHILDHOOD
Infant of a diabetic mother	Idiopathic ketotic hypoglycemia
Infection/sepsis	Infection/sepsis
Adrenal hemorrhage	Endocrinopathy
Congenital heart disease	Inborn errors of metabolism
Hypothermia	Hyperinsulinism
Hypoglycemia-inducing drug use by the mother	Drug induced (e.g., salicylates)
Maternal eclampsia	Factitious disorders
Fetal distress from any cause	Idiopathic

versy. In general, one should consider a plasma glucose of <30 mg/dL in the first 24 h of life to constitute hypoglycemia. For the remainder of the neonatal period, a plasma glucose level of <45 mg/dL is considered hypoglycemic.[2]
- Hypoglycemia is not a diagnosis per se, but represents an important clinical finding associated with many disorders, illnesses, and ingestions. A partial list of conditions associated with hypoglycemia in infants and children is provided in Table 79-1.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Although the treatment of hypoglycemia may seem straightforward (i.e., administer glucose), there is controversy as to how this is best accomplished. One nationally recognized course (Pediatric Advanced Life Support) recommends that 5 to 10 mL/kg bolus of $D_{10}W$ be administered intravenously or intraosseously to hypoglycemic neonates. Children with hypoglycemia should receive a bolus of 2 to 4 mL/kg of $D_{25}W$.[3] Adolescents typically receive the adult dose of 50 mL of $D_{50}W$. Other sources recommend smaller doses.
- The underlying illness or ingestion that is associated with the hypoglycemia should be investigated and treated appropriately.

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80 ALTERED MENTAL STATUS IN CHILDREN

Lance Brown

EPIDEMIOLOGY

- The etiologies of altered mental status (AMS) in children are quite varied. No epidemiologic data are available that capture all of the causes.

PATHOPHYSIOLOGY

- Alterations in mental status result from either depression of both cerebral cortices or localized abnormalities of the reticular activating system in the brainstem and midbrain.
- The pathologic conditions that result in AMS can be divided into three broad categories: supratentorial mass lesions, subtentorial mass lesions, and metabolic encephalopathy.[1,2]
- Supratentorial mass lesions cause AMS by compressing the brainstem and/or diencephalon. Focal motor abnormalities are often present from the onset of the alteration in consciousness. Neurologic dysfunction progresses from rostral to caudal with sequential failure of midbrain, pontine, and medullary function. Compromise by supratentorial lesions causes slow nystagmus toward and fast nystagmus away from a cold stimulus during caloric testing.
- Subtentorial mass lesions lead to dysfunction of the reticular activating system and prompt loss of consciousness. There is a discrete level of dysfunction. Cranial nerve abnormalities and an abnormality in respiratory pattern (e.g., Cheyne-Stokes respiration, neurogenic hyperventilation, ataxic breathing) are common. With brainstem injury,

asymmetric and/or fixed pupils are found. No eye movements occur despite cold stimuli to both auditory canals.

- Metabolic encephalopathy usually causes depressed consciousness before depressed motor signs. When motor signs are present, they are typically symmetric.[1,3] Respiratory function is involved relatively early, and abnormalities are often secondary to acid-base imbalance. Pupillary reflexes are generally preserved, but pupillary reactivity may be sluggish. The pupils are not usually fixed or asymmetric. However, pupillary reflexes may be absent in the setting of profound anoxia or toxicologic effects such as occurs with cholinergics, anticholinergics, opiates, and barbiturates.

CLINICAL FEATURES

- A long differential diagnosis list needs to be entertained when an infant or child presents with AMS. Historical data should focus on prodromal events leading to the change in consciousness, recent illnesses, infectious exposure, toxicologic exposure, and the likelihood of trauma and abuse. Detailed information should be obtained regarding antecedent fever, headaches, head tilt, abdominal pain, vomiting, diarrhea, gait disturbance, seizures, drug ingestion, palpitations, weakness, hematuria, weight loss, and rash. Developmental milestones, past medical history, immunization history, and family history should be assessed.

TABLE 80-1 AEIOU TIPS

A	**Alcohol.** Changes in mental status can occur with serum levels <100 mg/dL. Concurrent hypoglycemia is common. **Acid-base and metabolic.** Hypotonic and hypertonic dehydration. Hepatic dysfunction, inborn errors of metabolism, diabetic ketoacidosis, primary lung disease, and neurologic dysfunction causing hypercapnia. **Dysrhythmia (arrhythmia)/cardiogenic.** Stokes-Adams, supraventricular tachycardia, aortic stenosis, heart block.
E	**Encephalopathy.** Hypertensive encephalopathy can occur with diastolic pressures of 100–110 mmHg. Reye's syndrome. **Endocrinopathy.** AMS is rare as a presentation in this category. Addison's disease can present with AMS or psychosis. Thyrotoxicosis can present with ventricular dysrhythmias. Pheochromocytoma can present with hypertensive encephalopathy. **Electrolytes.** Hyponatremia becomes symptomatic around 120 meq/L. Hypernatremia and disorders of calcium, magnesium, and phosphorus can produce AMS.
I	**Insulin.** AMS from hyperglycemia is rare in children, but diabetic ketoacidosis is the most common cause. Hypoglycemia can be the result of many disorders. Irritability, confusion, seizures, and coma can occur with blood glucose levels <40 mg/dL. **Intussusception.** AMS may be the initial presenting symptom.
O	**Opiates.** Common household exposures are to Lomotil, Imodium, diphenoxylate, and dextromethorphan. Clonidine, an α agonist, can also produce similar symptoms.
U	**Uremia.** Encephalopathy occurs in over one-third of patients with chronic renal failure. Hemolytic uremic syndrome can also produce AMS in addition to abdominal pain. Thrombocytopenic purpura and hemolytic anemia can also cause AMS.
T	**Trauma.** Children with blunt trauma are more likely to develop cerebral edema than are adults. The child should be examined for signs of abuse particularly shaken baby syndrome with retinal hemorrhages. **Tumor.** Primary, metastatic, or meningeal leukemic infiltration. **Thermal.** Hypo- or hyperthermia.
I	**Infection.** One of the most common causes of AMS in children. Meningitis should be high on the differential list. **Intracerebral vascular disorders.** Subarachnoid, intracerebral, or intraventricular hemorrhages can be seen with trauma, ruptured aneurysm, or arteriovenous malformations. Venous thrombosis can follow severe dehydration or pyogenic infection of the mastoid, orbit, middle ear, or sinuses.
P	**Psychogenic.** Rare in the pediatric age group, characterized by decreased responsiveness with normal neurologic examination including oculovestibular reflexes. **Poisoning.** Drugs or toxins can be ingested by accident, through neglect or abuse, or in a suicide gesture.
S	**Seizure.** Generalized motor seizures are often associated with prolonged unresponsiveness in children. Seizure in a young febrile patient suggests intracranial infection.

- The physical examination should initially focus on cardiac and cerebral resuscitation. The objectives of the general examination are to identify probable infectious etiologies, trauma, specific toxidrome, or metabolic disease.[2,4]
- The neurologic examination should document the child's response to sensory input, motor activity, pupillary reactivity, oculovestibular reflexes, and respiratory pattern.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis for AMS in children is diverse and differs slightly from that for adults.[4,5] The familiar mnemonic AEIOU TIPS remains a useful tool in organizing diagnostic possibilities (Table 80-1).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Attention must be given to airway, breathing, and circulation from the outset.
- Oxygenation, fluid resuscitation, bedside glucose determination, control of body temperature, control of seizures with benzodiazepines (initially), and a restoration of the acid-base balance are required for all children with AMS.
- Most infants and children with AMS will require admission and extended observation.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 126, ''Altered Mental Status in Children,'' by Nancy Pook, Natalie Cullen, and Jonathan I. Singer.

81 SYNCOPE AND SUDDEN DEATH IN CHILDREN AND ADOLESCENTS

David M. Cline

EPIDEMIOLOGY

- Between 20 and 50 percent of adolescents will experience syncope by the age of 18.[1,2] This condition is transient and usually self-limited. However, serious cardiac disease is found in 25 percent of children referred to a cardiologist.[3]
- The rate of sudden unexpected death in children is 2.3 percent of all pediatric deaths.[4] Sudden cardiac death makes up about one-third of these cases.
- Except for trauma, sudden cardiac death is the most common cause of sports-related deaths,[5] more commonly associated with basketball, football, and track.[6]
- Other causes of sudden cardiac death in children are myocarditis, cardiomyopathy, congenital heart disease, and conduction disturbances.[7,8]
- Hypertrophic cardiomyopathy is the most common cause of sudden cardiac death in adolescents without known cardiac disease.[7]

PATHOPHYSIOLOGY

- Syncope is the temporary loss of consciousness from reversible disruption of cerebral functioning and usually involves inadequate cardiac output and cerebral hypoperfusion, resulting in a temporary loss of consciousness.
- Vascular syncope occurs when a stimulus causes venous pooling in the legs, leading to a decrease in ventricular preload with a compensatory increase in heart rate and myocardial contractility.
- Neurally mediated syncope (NMS) or reflex syncope occurs when receptors in the atria, ventricles, and pulmonary arteries sense a decrease in venous return and an efferent brainstem response via the vagal nerve causes bradycardia, hypotension, or both.
- Cardiac syncope occurs when there is an interruption of cardiac output from an intrinsic cardiac problem. These causes are divided into tachydysrhythmias, bradydysrhythmias, outflow obstruction, and myocardial dysfunction.
- Any event that causes sufficient cerebral hypoperfusion can lead to sudden death.

TABLE 81-1 Causes of Syncope in Children and Adolescents

Neurally mediated: most common cause of syncope in children
Orthostatic: light-headedness with standing
Situational: urination, defecation, coughing, and swallowing may precipitate
Familial dysautonomia
Cardiac dysrhythmias: events that usually start and end abruptly
Prolonged Q-T syndrome
Wolff-Parkinson-White syndrome
Sick sinus syndrome: associated with prior heart surgery
Supraventricular tachycardia
Atrioventricular block: most common in children with congenital heart disease
Pacemaker malfunction
Structural cardiac disease
Hypertrophic cardiomopathy: exertional syncope most common presentation, but infants can present with congestive heart failure and cyanosis; echocardiography necessary to confirm
Dilated cardiomyopathy: may be idiopathic, postmyocarditis, or with congenital heart disease
Congenital heart disease
Valvular diseases: aortic stenosis usually congenital defect, Ebstein's malformation, or mitral valve prolapse (which is not associated with increased risk of sudden death)
Dysrhythmogenic right ventricular dysplasia
Pulmonary hypertension: dyspnea on exertion, exercise intolerance, shortness of breath
Coronary artery abnormalities: aberrant left main artery causing external compression during physical exercise
Endocrine abnormalities: hyperthyroid, hyperglycemia, adrenal insufficiency
Medications and drugs: antihypertensives, tricyclic antidepressants, cocaine, diuretics, antidysrhythmics
Gastrointestinal disorders: reflux

CLINICAL FEATURES

- Syncope is the sudden onset of falling accompanied by a brief episode of loss of consciousness.
- Involuntary motor movements may occur with all types of syncopal episodes but are most common with seizures.[3]
- Two-thirds of children experience light-headedness or dizziness prior to the episode.[3]

TABLE 81-2 Risk Factors for Serious Causes of Syncope

Exertion preceding the event
History of cardiac disease in patient
Recurrent episodes
Recumbent episode
Family history of sudden death, cardiac disease, deafness
Chest pain, palpitations
Prolonged loss of consciousness
Medications that affect cardiac conduction

TABLE 81-3 Events Mistaken for Syncope

Basilar migraine: headache, loss of consciousness, neurologic symptoms
Seizure: loss of consciousness, simultaneous motor movements, prolonged recovery
Vertigo: no loss of consciousness, spinning or rotating sensation
Hysteria: no loss of consciousness, indifference to the event
Hypoglycemia: confusion, gradual onset associated with diaphoresis
Breath-holding spell: crying prior to the event, age 6–18 months old
Hyperventilation: severe hypocapnia can cause syncope

- There are many causes of syncope in children. Table 81-1 lists the most common causes of syncope by category.
- Neurally mediated syncope is the most common cause in children and includes vasovagal and neurocardiogenic syncope, reflex syncope, and simple fainting.
- Risk factors associated with serious causes of syncope are presented in Table 81-2.
- Events easily mistaken for syncope are presented in Table 81-3, along with common associated symptoms.

DIAGNOSIS AND DIFFERENTIAL

- No specific historical or clinical features reliably distinguish between vasovagal syncope and other causes.[3] However, a thorough history and physical examination can help to arouse suspicion for serious causes. Particular attention should be given to the cardiac examination.
- The most important step in the evaluation of children with syncope is a detailed history, including medications, drugs, fluid intake, and food.
- Syncope during exercise suggests a more serious cause. Many of the diseases that cause syncope also cause sudden death in children. Approximately 25 percent of children who suffer sudden death have a history of syncope.[4]
- If witnesses note that the patient appeared dead or cardiopulmonary resuscitation was performed, a search for serious pathology must be undertaken.[9]
- Cardiac dysrhythmia should be suspected if syncope is associated with fright, anger, surprise, or physical exertion.[10]
- The physical examination should include a complete cardiovascular, neurologic, and pulmonary examination. Any abnormalities noted in the car-

diovascular examination require an in-depth cardiac workup.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Laboratory assessment is guided by history, physical examination, and clinical suspicion. Routine laboratory studies are not needed if vasovagal syncope is clearly identified from the history. Those with worrisome associated symptoms should have a chemistry panel and hematocrit. A pregnancy test should also be done in females of childbearing age. Serum drug screening or alcohol level determination may also be useful if ingestion is suspected.
- A chest radiograph and electrocardiogram (ECG) may also be helpful if there is suspicion of pulmonary or cardiac causes.[3] The QT interval should be assessed.[11] Patients with hypertrophic cardiomyopathy or a prolonged QT interval should be referred to a cardiologist.
- An echocardiogram should be obtained in patients with known or suspected cardiac disease.
- If vasovagal syncope is diagnosed, only reassurance is needed.
- If no clear cause is found, the child may be discharged to be further evaluated and followed by the primary care physician unless there are cardiac risk factors or exercise-induced symptoms.
- Children with documented dysrhythmias should be admitted. Patients with a normal ECG but a history suggesting a dysrhythmic event are candidates for outpatient monitoring and cardiac workup.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 127, ''Syncope and Sudden Death,'' by William E. Hauda II and Thom A. Mayer.

82 FLUID AND ELECTROLYTE DISORDERS IN CHILDREN

Lance Brown

Fluid and electrolyte disturbances in infants and young children presenting to the emergency department (ED) occur most commonly in the setting of acute gastroenteritis. Inappropriate home remedies for vomiting and diarrheal illnesses also contribute significantly to fluid and electrolyte imbalances in infants and young children. (For more information on gastroenteritis see Chap. 76, ''Vomiting and Diarrhea in Children.'')

EPIDEMIOLOGY

- The incidence of fluid imbalance and electrolyte disturbances in children is unknown. However, there are about 3 million physician visits, 220,000 hospitalizations, and between 325 and 425 deaths each year in the United States due to gastroenteritis and the resultant fluid and electrolyte disturbances.[1]

PATHOPHYSIOLOGY

- During the first 2 years of life, there are tremendous caloric and water maintenance requirements. Rapidly growing infants need an enormous

amount of calories and water relative to their body weight. The relative daily free water turnover is 3 to 4 times that of an adult.
- Young infants are at risk for cardiovascular compromise from sudden fluid losses (e.g., vomiting and diarrhea). Factors contributing to this include extensive daily free water turnover, very large relative body surface area, insensible electrolyte free losses from the skin and respiratory tract (especially with fever), a relative inability to concentrate urine, and a relatively large percentage of total body water in the extracellular space.
- In an acute dehydrating illness, the extracellular space is disproportionately depleted. Sodium is the dominant extracellular cation. Dehydration is classified according to the relative balance between water and sodium. In general, dehydration can be classified as isotonic, hypernatremic, and hyponatremic.
- Isotonic dehydration is most common and results from a proportionately equal loss of sodium and water. The serum sodium will remain within the normal range (130 to 145 meq/L). The most common cause of isotonic dehydration is diarrhea.
- Hypernatremic dehydration results from a relatively greater loss of free water than sodium. The serum sodium is typically >150 meq/L. Hypernatremic dehydration typically occurs when a young patient with gastroenteritis is fed salt-rich solutions (e.g., inappropriately mixed formula, boiled skim milk, or chicken broth). Rapid rehydration can lead to an influx of water into brain cells and subsequent brain edema.
- Hyponatremic dehydration is characterized by a serum sodium <130 meq/L. Typically this state develops when acute fluid losses from vomiting and diarrhea are replaced with free water (e.g., tea or diluted formula). Hyponatremia may also occur in the setting of increased total body water relative to sodium (e.g., syndrome of inappropriate antidiuretic hormone, edema-forming states—nephrotic syndrome and cirrhosis, or psychogenic or infantile water intoxication). Conditions that lead to a rapid reduction in serum sodium negatively affect the central nervous system. Irritability, lethargy, and seizures are characteristically seen.
- Although rehydration is generally well tolerated, very rapid correction of profound hyponatremia may result in osmotic demyelination syndrome (central pontine myelinolysis).

CLINICAL FEATURES

- The clinical appearance of patients with dehydration and fluid and electrolyte disturbances depends primarily on the degree of dehydration.
- Because acute fluid (water) loss can be measured as lost weight (1 L water = 1 kg), the gold standard for assessing dehydration is the comparison of a very recent pre-illness weight with weight at presentation on the same scale. From this comparison

TABLE 82-1 Estimation of Dehydration

EXTENT OF DEHYDRATION	MILD	MODERATE	SEVERE
Weight loss			
Infants	5%	10%	15%
Children	3–4%	6–8%	10%
Pulse	Normal	Slightly increased	Very increased
Blood pressure	Normal	Normal to orthostatic, >10 mmHg change	Orthostatic to shock
Behavior	Normal	Irritable, more thirsty	Hyperirritable to lethargic
Thirst	Slight	Moderate	Intense
Mucous membranes*	Normal	Dry	Parched
Tears	Present	Decreased	Absent, sunken eyes
Anterior fontanelle	Normal	Normal to sunken	Sunken
External jugular vein	Visible when supine	Not visible except with supraclavicular pressure	Not visible even with supraclavicular pressure
Skin* (less useful in children >2 years of age)	Capillary refill <2 s	Slowed capillary refill, 2–4 s (decreased turgor)	Very delayed capillary refill (>4 s) and tenting; skin cool, acrocyanotic, or mottled*
Urine specific gravity	>1.020	>1.020; oliguria	Oliguria or anuria

* These signs are less prominent in patients who have hypernatremia.

a percentage dehydration (as represented by percentage weight loss) can be calculated. Unfortunately, this comparison is almost never available in the ED. However, physical examination has been shown to provide a reliable estimation of the degree of dehydration.[2] The dehydration state is classified as either mild, moderate, or severe (Table 82-1).

- An exception to this general pattern occurs in hypernatremic dehydration where fluid is drawn from the interstitial and intracellular spaces in the face of the increased serum osmolarity. This process protects the circulating blood volume. Peripheral perfusion and vital signs may be deceptively normal. The skin may reveal a characteristic doughy feel.

DIAGNOSIS AND DIFFERENTIAL

- In the absence of a reliable pre-illness comparison weight, the diagnosis of dehydration is based on historical data and physical exam findings. Laboratory data lend supporting evidence, help classify the type of dehydration (e.g., isotonic, hypernatremic, or hyponatremic), and identify related problems (e.g., renal failure, ketotic hypoglycemia, or diabetic ketoacidosis).
- The most common cause of dehydration and fluid and electrolyte imbalance in infants and young children is viral gastroenteritis. The most common enteropathogens identified in the United States are rotavirus and enteric adenoviruses.[3]
- Other important causes of fluid and electrolyte disturbances in children include burns, diabetic complications, inappropriate formula administration (mixed incorrectly), inappropriate feedings (e.g., extensive juice drinking, bottles of water offered to small infants, chicken broth, or boiled milk), diabetes inspidis, adrenal insufficiency, anorexia due to febrile illnesses, respiratory illnesses interfering with adequate oral intake, and cystic fibrosis.
- Pyloric stenosis has historically been identified with a hypochloremic metabolic alkalosis. However, with earlier identification of pyloric stenosis, this presentation is becoming increasingly uncommon.[4]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The management of fluid and electrolyte disturbances in infants and young children revolves around a few basic principles: (a) shock should be identified and treated, (b) causes that have a specific treatment (e.g., diabetic ketoacidosis, pyloric stenosis, or respiratory distress) should be identified and treated, and (c) appropriate fluids should be administered to replace maintenance fluids, fluids already lost, and ongoing fluid losses.
- Hypovolemic shock should be treated with 20 mL/kg boluses of intravenous (IV) (or intraosseous) isotonic crystalloid (e.g., 0.9% normal saline (NS) or lactated Ringer's solution) until improved mental status, vital signs, and peripheral perfusion are noted.
- Maintenance fluids are calculated as follows: for children $\leq$10 kg 100 mL/kg/day should be administered, for children 11 to 20 kg 1000 mL + 50 mL/kg for each kg >10 over 24 h should be administered, for children >20 kg 1500 mL + 20 mL/kg for each kg >20 over 24 h should be administered. Standard solutions for maintenance fluids are D_5 0.25NS for infants <1 year old and $D_5$0.5NS for older infants and children. Potassium chloride, 20 meq/L, is typically added after adequate urine output is established.
- Deficit fluids are determined from the clinical appearance and estimated percent dehydration (see Table 128-2 in *Emergency Medicine: A Comprehensive Study Guide*, 5th ed.). The calculations are performed in the following manner. If a patient weighs 15 kg on presentation and is estimated as 10 percent dehydrated, then it is estimated that 15 $\times$ 10 percent = 1.5 kg of water lost; 1.5 kg of water = 1.5 L of water. Therefore, 1500 mL is the estimated deficit. One-half of this total is administered during the first 8 h and the remaining half is given over the following 16 h. The hourly IV fluid rate is determined by the sum of maintenance and deficit fluid requirements for the patient.[5]
- Oral rehydration has been shown to be as effective as IV therapy for rehydrating infants and children. There is debate as to what the appropriate sodium content of the rehydration solution should be. The replacement is performed by administering 50 mL/kg orally over 4 h to mildly dehydrated patients and 100 mL/kg to moderately dehydrated patients.[6–9]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide*, 5th ed., see Chap. 128, "Fluid and Electrolyte Therapy," by William Ahrens.

83 UPPER RESPIRATORY EMERGENCIES

Jonathan L. Jones

STRIDOR

EPIDEMIOLOGY

- Diseases that cause upper respiratory tract (URT) obstruction account for a significant percentage of visits to the pediatric emergency department. Some diseases of the URT are common and quite benign, while others are much less common and are life-threatening.

PATHOPHYSIOLOGY

- Stridor is due to Venturi effects created by somewhat linear airflow through a variably collapsible tube, the airway. When one inhales, the relative pressure in the center of the tube becomes greater than that at its edges. The pressure differential leads to collapse of the airway walls.
- As one progresses from the supraglottic to the glottic and subglottic and finally the tracheal areas of the airway, there is an increase in physiologic support and therefore a decrease in the amount of collapse that occurs upon inspiration.[1]
- Stridor on inspiration is indicative of obstruction at or above the larynx. Biphasic stridor places the obstruction in the trachea. Expiratory stridor usually means that the obstruction is below the carina.
- Expiratory stridor, or wheeze, is common in distal airways, since intrathoracic pressure may become much greater than atmospheric pressure during expiration. The pressure differential creates high relative laminar flow through semicollapsible bronchi, resulting in wheezes.

CLINICAL FEATURES

- Hypoxia may be present without cyanosis. The presence of cyanosis is dependent on the hemoglobin level and the peripheral circulation. Cyanosis is an ominous sign.
- Tachypnea, chest retractions, and nasal flaring are the triad of labored respirations.
- Signs of labored respirations appear early in the course of the illness and worsen, thus serving as a prognostic sign as well as a diagnostic sign.
- The physical sign common to all URT obstructions is stridor.
- Tachypnea is not specific for respiratory tract disease. It can be seen in cardiac disorders and diseases that cause metabolic acidosis.
- Chest retractions and nasal flaring are more specific for respiratory tract disorders than is tachypnea.
- Grunting is a valuable diagnostic sign, as it localizes disease to the lower respiratory tract and correlates with disease severity.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis is made easier if one considers the age of the patient and the duration of symptoms.
- Children less than 6 months old with a long duration of symptoms characteristically have a congenital cause of stridor. Common causes are laryngomalacia and vocal cord paralysis.[2]

- Patients over 6 months old with a short duration of symptoms characteristically have an acquired cause of stridor (viral croup, epiglottitis, foreign-body aspiration, peritonsillar abscess, and retropharyngeal abscess).

EPIGLOTTITIS

CLINICAL FEATURES

- Epiglottitis is life-threatening and can occur at any age.
- Since the introduction of *Haemophilus influenzae* vaccine, the median age of presentation has been 7 years.[3] Most cases are due to gram-positive organisms.
- Classically there is an abrupt onset of high fever, sore throat, stridor, dysphagia, and drooling developing over 2 days.
- The presentation in older children and adults is much more subtle. The only complaint may be severe sore throat with or without stridor.

DIAGNOSIS AND DIFFERENTIAL

- Close monitoring during evaluation is important when epiglottitis is suspected.
- If total airway obstruction or apnea occurs, children with epiglottitis sometimes can be bagged effectively.
- Lateral neck x-rays must be taken with the neck extended and should be taken during inspiration. The epiglottis is normally tall and thin, but in patients with epiglottitis it is very swollen and appears squat and flat like a thumbprint.

EMERGENCY DEPARTMENT CARE

- Direct visualization of the epiglottis is safe and accurate when performed by clinicians skilled in difficult airway management. The objective of airway management is to prevent deterioration with sudden and total obstruction of the airway.
- Supportive therapy may include humidified oxygen and nebulized epinephrine.
- Choices for intravenous antibiotics include cefuroxime 50 mg/kg every 8 h intravenously (IV), cefotaxime 50 mg/kg every 8 h IV, and ceftriaxone 50 mg/kg every 24 h IV. Vancomycin may be added in regions with increased cephalosporin resistance. Steroids are not necessary but are used frequently.

VIRAL CROUP (LARYNGOTRACHEOBRONCHITIS)

CLINICAL FEATURES

- Viral croup is usually a benign, self-limited disease that causes marked edema and inflammation.
- The age range is 6 months to 3 years, and croup occurs mainly in the late fall and early winter.[4] The etiology is usually parainfluenza virus.
- The typical history is 2 to 3 days of upper respiratory infection (URI) with a gradually worsening cough, especially at night. The cough is barking in quality.
- Physical examination reveals stridor.
- Bacterial tracheitis (membranous laryngotracheobronchitis), a more severe form of croup, is usually caused by *Staphylococcus aureus.* Patients with bacterial tracheobronchitis have more respiratory distress than do patients with croup and may present similarly to those with epiglottitis. These patients usually need intubation and antibiotics.

DIAGNOSIS

- Croup usually can be diagnosed on clinical grounds. X-rays are not necessary in every patient.

EMERGENCY DEPARTMENT CARE

- Patients should be monitored with pulse oximetry and treated with cool mist and oxygen. Antibiotics are not needed.
- Steroids have been shown to be beneficial in treating croup.[5,6]
- Nebulized epinephrine can be used to treat severe cases. Patients should be monitored for at least 2 h for relapse after treatment; true rebound after epinephrine therapy (with stridor more severe than it was at presentation) is not common.[1]

FOREIGN-BODY ASPIRATION

CLINICAL FEATURES

- Ninety percent of foreign-body (FB) aspirations occur in children under 4 years of age.
- In children under 6 months of age, the cause is usually secondary to a sibling feeding the patient.
- Physical signs depend on the location of the FB and may include wheezing, crackles, tachypnea,

persistent pneumonia, stridor, coughing, and apnea.
- Most airway FBs are radiolucent.[7] Air trapping caused by an FB may lead to hyperinflation of the obstructed lung. A single negative x-ray does not rule out an FB.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of an airway FB usually consists of laryngoscopy or rigid bronchoscopy in the operating room under anesthesia.
- Antibiotics, steroids, oxygen, mist, and chest physiotherapy all may be necessary.

PERITONSILLAR ABSCESS

CLINICAL FEATURES

- Peritonsillar abscess in children most commonly presents in adolescents with an antecedent sore throat.
- The patients usually appear acutely ill with fevers, chills, dysphagia, trismus, drooling, and a muffled voice.
- The uvula is displaced away from the affected side. The involved tonsil is anteriorly and medially displaced.

DIAGNOSIS AND DIFFERENTIAL

- Careful visualization of the oral cavity can reliably rule out peritonsillar abscess in many cases.
- When uvular deviation, marked soft palate displacement, severe trismus, airway compromise, or localized areas of fluctuance are noted, the diagnosis of peritonsillar abscess can be made confidently and no imaging studies are required.
- Computed tomography (CT) or ultrasound imaging may be required in younger children.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The majority of patients with peritonsillar abscess are treated as outpatients with needle aspiration, antibiotics, and pain control.
- Antibiotic choices include ampicillin-sulbactam 40 mg/kg per day divided every 8 h and a third-generation cephalosporin.
- Definitive follow-up is essential.
- Formal incision and drainage in the operating room sometimes is necessary, especially in young or uncooperative patients.

RETROPHARYNGEAL ABSCESS

CLINICAL FEATURES

- Retropharyngeal abscess usually occurs in children age 6 months to 3 years.
- These patients usually appear toxic, presenting with fever, drooling, dysphagia, and inspiratory stridor.
- Dysphagia and refusal to feed occur before significant respiratory distress.

DIAGNOSIS AND DIFFERENTIAL

- CT of the neck is very helpful in establishing the diagnosis.[8]
- Lateral neck x-ray performed during inspiration may show a widened retropharyngeal space.
- Physical examination of the pharynx may show a retropharyngeal mass. Palpation of the mass could lead to rupture of the abscess.

EMERGENCY DEPARTMENT CARE

- The airway should be stabilized.
- Antibiotic choice is controversial since most retropharyngeal abscesses contain mixed flora. Single-agent treatment with ampicillin-sulbactam 40 mg/kg per day divided every 8 h may be best. Penicillin G is recommended by some.
- Consultation with an ear, nose, and throat specialist for operative incision and drainage is indicated.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 129, ''Upper Respiratory Emergencies,'' by Randolph J. Cordle and Nicholas C. Relich.

84 PEDIATRIC EXANTHEMS

Lance Brown

- Essential information to make the diagnosis of rash in a child includes the signs and symptoms which preceded or presented with the exanthem, immunization history, human and animal contacts, and environmental exposures.
- Pediatric exanthems can be broadly classified as bacterial, viral, rickettsial, and those of unclear etiology. (Rocky mountain spotted fever is discussed in Chap. 94.)

BACTERIAL INFECTIONS

BULLOUS IMPETIGO

- This exanthem typically occurs in infants and young children.
- Lesions are superficial, thin-walled bullae that characteristically occur on the extremities, rupture easily, leave a denuded base, dry to a shiny coating, and contain fluid that harbors staphylococci.
- Diagnosis is usually made by the appearance of the characteristic bullae.
- Treatment is with oral antistaphylococcal agents such as cephalexin or dicloxacillin and topical agents such as bacitracin.

IMPETIGO CONTAGIOSUM

- This exanthem is a superficial skin infection typically caused by group A beta hemolytic streptococci or *Staphylococcus aureus.*
- The lesions usually occur in small children, often in areas of insect bites or minor trauma. The lesions start as red macules and papules which then form vesicles and pustules. Rupture of the vesicles results in the formation of a golden crust.
- With the exception of lymphadenopathy, fever and systemic signs are rare.
- Most commonly affected areas include the face, neck, and extremities.
- Diagnosis is based on the appearance of the rash (see Fig. 84-1).
- Treatment consists of an oral cephalosporin (such as cephalexin), an oral macrolide (such as erythromycin), or an antistaphylococcal penicillin (dicloxacillin), wound cleaning, and topical bacitracin or mucopiricin.

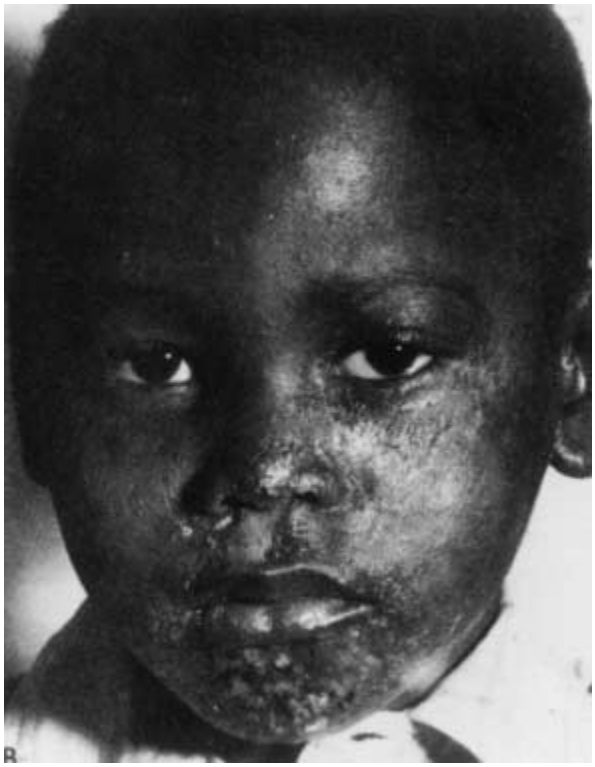

FIG. 84-1 Impetigo contagiosum. [From Marples RR, Leyden JL: Bacterial infections, section 1. Fundamental cutaneous microbiology, in Moschella SL, Hurley HJ (eds): *Dermatology.* Philadelphia, Saunders, 1985, vol 1, chap 11, pp 590–642, with permission.]

ERYSIPELAS

- Erysipelas is a cellulitis and lymphangitis of the skin due to group A beta-hemolytic streptococci.
- Fever, chills, malaise, headache, and vomiting are common.
- The face is the most common site and the lesion typically forms in the area of a skin wound or pimple.
- The rash starts as a red plaque which rapidly enlarges. Increased warmth to the touch, swelling, and a raised, sharply demarcated, indurated border are typical.
- Diagnosis is by history and the appearance of the rash.
- Treatment consists of intravenous antibiotics. Empirically, ceftriaxone or cefuroxime are effective. If the infection is confirmed to be streptococcal, and resistance is unlikely, penicillin G would be effective.

MYCOPLASMA INFECTIONS

- Rashes associated with mycoplasma infections typically occur in the setting of an acute respiratory illness in a school-aged child (5 to 19 years of age).
- Associated symptoms are typically fever, cough, sore throat, malaise, headache, chills, and rash.
- The rash is typically on the trunk and is red and maculopapular. Also seen is erythema multiforme and occasionally Stevens-Johnson syndrome.
- The treatment is a macrolide antibiotic (e.g., erythromycin).

SCARLET FEVER

- A distinctive rash is seen with scarlet fever. The etiologic agent is typically group A beta-hemolytic streptococci (group C streptococci has been implicated as well).
- Scarlet fever typically occurs in school-aged children and is diagnosed by the presence of exudative pharyngitis, fever, and the characteristic rash. Associated symptoms include sore throat, fever, headache, vomiting, and abdominal pain.
- The rash typically starts in the neck, groin, and axillae with accentuation at the flexural creases (Pastia's lines). The rash is red and punctate, blanches with pressure and has a rough sandpaper feel. In the early course of the illness the tongue has a white coating through which hypertrophic, red papillae project (the "white strawberry tongue"). Hemorrhagic spots may be seen on the soft palate. The rash typically develops 1 to 2 days after the illness onset. Facial flushing and circumoral palor are characteristic. Desquamation occurs with healing at about 2 weeks after the onset of symptoms.
- The diagnosis is generally made on clinical grounds. Throat culture typically reveals group A beta-hemolytic streptococci or group C streptococci.
- Treatment is with penicillin (or erythromycin in the penicillin-allergic patient). Antibiotic treatment shortens the course of the illness and reduces the incidence of rheumatic fever and nephritis.

STAPHYLOCOCCAL SCALDED-SKIN SYNDROME

- Staphylococcal scalded-skin syndrome is typically seen in neonates and infants.
- The syndrome is caused by a toxin produced by *Staphylococcus aureus* at a site distant to the affected skin. This is often the nose, pharynx, a wound, or an abscess.
- The toxin causes a separation at the granular layer of the epidermis leading to generalized confluent skin exfoliation. The illness begins with fever and irritability, then a diffuse erythroderma (sparing the palms, soles, and mucosa), then large thin-walled bullae appear. The skin layers at the edge of the bullae separate with gentle pressure (Nikolsky's sign). When the bullae rupture, large areas of denuded skin are exposed. The skin typically heals without scarring.
- Therapy includes parenteral antistaphylococcal antibiotics such as cefazolin or nafcillin, localized wound care, fluid and electrolyte management, thermal control, and eradication of any underlying focus of infection. Children with staphylococcal scalded skin syndrome are usually admitted to the hospital.

VIRAL INFECTIONS

ENTEROVIRUSES

- Enteroviruses are a group of viruses including coxsackieviruses and echoviruses that can produce a wide range of clinical presentations. These infections typically occur in the summer and early fall.
- One of the enterovirus infections that is both common and has distinctive features is hand-foot-and-mouth disease. The etiologic agent is coxsackie A

16. The patients typically become febrile at the outset. Characteristic oral lesions consist of small vesicles that quickly ulcerate. The skin lesions are initially red papules that progress to grayish vesicles on the palms, soles, and buttocks. The lesions heal without scarring in 7 to 10 days.
- Many enteroviral infections lack characteristic features. Clinical presentation of an enteroviral infection may include myocarditis, pericarditis, aseptic meningitis, orchitis, hepatitis, bronchitis, pneumonia, and a nonspecific illness with vomiting and myalgias.
- The rash of enteroviral infections may be macular, morbilliform, vesicular, petechial, purpuric, or scarlatiniform.
- Treatment typically focuses on hydration as oral lesions may be painful and inhibit adequate oral intake.

ERYTHEMA INFECTIOSUM

- Erythema infectiosum is a febrile illness, typically appearing in the spring, caused by parvovirus B19. School-aged children aged 5 to 15 years are most commonly affected.
- The rash typically starts as an abrupt onset, bright red rash on the cheeks, giving the so-called ''slapped-cheek appearance.'' The eyelids and chin are characteristically spared. Circumoral pallor is typical. This rash fades after 4 to 5 days.
- As the illness progresses, and 1 to 2 days after the facial rash appears, a nonpuritic erythematous macular or maculopapular rash appears on the trunk and limbs. This rash may last for one week. As the rash fades, central clearing of the lesions occurs leaving a lacy appearance to the rash. Palms and soles are rarely affected.
- This rash may recur intermittently in the weeks following the onset of illness. This may be exacerbated by sun exposure.
- Associated constitutional, respiratory, and gastrointestinal symptoms are common. There is no specific therapy.

MEASLES

- Due to immunizations, measles is no longer common. Local epidemics do occur. This myxovirus infection typically occurs in the winter and spring.
- The incubation period is 10 days. A 3-day prodrome of upper respiratory symptoms followed by malaise, fever, coryza, conjunctivitis, photophobia, and cough is typical. Ill appearance is expected.
- Just prior to the development of a rash, Koplik's spots—tiny white spots on the buccal mucosa—may be seen. These spots give a ''grains of sand'' appearance and are pathognomonic for measles.
- The rash develops 14 days after exposure. Initially a red, blanching, maculopapular rash develops. The rash progresses from the head to the feet. The rash rapidly coalesces on the face. The duration of the rash is about 1 week.
- As the rash resolves, a coppery brown discoloration may be seen and desquamation may occur.
- Measles is self-limited. Treatment is supportive.

INFECTIOUS MONONUCLEOSIS

- The etiologic agent for infectious mononucleosis is the Epstein-Barr virus. The disease primarily affects children and young adults.
- Systemic symptoms include fever, malaise, and sore throat. The pharynx is often inflamed with exudate present. Lymphadenopathy typically affects both anterior and posterior cervical chains.
- A generalized erythematous maculopapular rash with soft palate petechiae is seen in 5 percent of patients. Nearly all patients who are treated with ampicillin or other related penicillins (e.g., amoxicillin) develop an erythematous maculopapular rash.
- The Monospot test is less reliable in children under the age of 5 than it is in children over 5 years. Treatment is supportive. Of note is the splenic enlargement that occurs with infectious mononucleosis. If a child participates in contact sports or sustains an injury to the left upper quadrant of the abdomen, splenic rupture may occur.

RUBELLA

- Now quite rare due to immunizations, rubella can be seen in teenagers typically in the spring.
- The prodromal symptoms include fever, malaise, headache, sore throat, and upper respiratory tract symptoms.
- The rash develops as fine, irregular pink macules and papules on the face, which then spread to the neck, trunk, and arms in a centrifugal distribution. The rash coalesces on the face as the eruption reaches the lower extremities and then clears in the same order as it appeared.
- Lymphadenopathy typically involves the suboccipital and posterior auricular nodes. Treatment

is supportive. Of concern are the congenital fetal malformations that occur when a woman contracts rubella while pregnant.

VARICELLA (CHICKEN POX)

- Varicella typically occurs in children less than 10 years of age, but may occur in all ages. Varicella is usually more severe in adults and occurs most often in the winter.
- The etiologic agent is the varicella zoster virus. Patients are highly contagious from the prodrome phase of the illness until all lesions are crusted over.
- The rash is typically preceded by a prodrome of fever and upper respiratory tract infection symptoms. The rash starts as faint red macules on the head or trunk. Within the first day the lesions begin to vesiculate, giving the characteristic ''dewdrop on a rose petal'' appearance. Over the next few days, groups of lesions develop giving the appearance of simultaneous multiple stages of development (see Fig. 84-2). Over the next 1 to 2 weeks, the lesions become dry and crusted.
- Treatment is symptomatic (e.g., diphenhydramine for itching). In the immunocompromised patient, varicella zoster immune globulin and acyclovir may be considered.

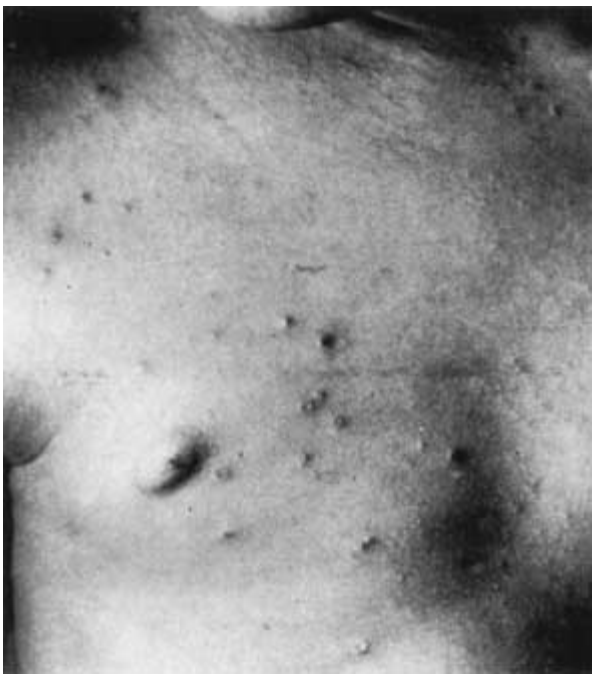

FIG. 84-2 Varicella. [From Burnett JW, Crutcher WA: Viral and rickettsial infections, in Moschella SL, Hurley HJ (eds): *Dermatology.* Philadelphia, Saunders, 1985, vol 1, chap 12, pp 673–738, with permission.]

ROSEOLA INFANTUM

- Roseola infantum is most common in children between 6 months and 3 years of age. It is caused by human herpes-virus 6.
- Initially starts with a high fever for 3 to 5 days. As the fever begins to resolve, blanching macular or maculopapular rose or pink discrete lesions develop. The most prominent location for the rash is the neck. Mucous membranes are not involved. The rash lasts 1 to 2 days and rapidly fades.

UNCLEAR ETIOLOGY

ERYTHEMA NODOSUM

- Erythema nodosum is an inflammatory exanthem that is associated with medications (e.g., oral contraceptives), sarcoidosis, inflammatory bowel disease, leukemia, vasculitis, tuberculosis, fungal diseases, and streptococcal infections.
- The lesions of erythema nodosum are distinctive tender nodules up to 5 cm in size on the shins and extensor prominences. The skin overlying the lesions is red, smooth, and shiny.
- Other symptoms include fever, arthralgias, myalgias, and fatigue.
- The lesions last several weeks. There is no known treatment except analgesia.

KAWASAKI DISEASE

- Kawasaki disease is a generalized vasculitis of unknown cause. The peak age of onset is 1 to 2 years.
- Diagnosis depends on the following clinical findings. The patient must have had a fever of at least 5 days duration. The illness must not be explained by another known disease process. Then, 4 of the following 5 criteria must be met: (a) bilateral conjunctivitis; (b) changes of the lips and oral mucosa (e.g., dry, red, fissured lips, strawberry tongue, or oropharyngeal edema); (c) changes of the extremities (e.g., erythema of palms and soles, edema of the hands and feet, or periungual desquamation); (d) Polymorphous rash; and (e) cervical lymphadenopathy.
- The rash usually consists of red, raised plaques. An erythrocyte sedimentation rate is often markedly elevated. There is an acute febrile phase for 1 to 2

weeks. Following this, desquamation of the hands and feet develops.
- Coronary artery aneurysms are seen in 20 percent of untreated patients and become clinically evident at about 1 month after the onset of illness. Sudden death occurs in 1 to 2 percent of untreated patients.
- Treatment consists of intravenous immunoglobulin and aspirin.

PITYRIASIS ROSEA

- Pityriasis rosea is characteristically seen in patients over 8 years of age and under 30 years of age during the spring and fall.
- It starts with a herald patch, one red lesion with a raised border on the trunk. Then 1 to 2 weeks later, a widespread eruption of pink maculopapular oval patches erupts on the trunk in a pattern following the ribs (the so-called ''Christmas tree distribution''). There may be mucosal involvement.
- Pityriasis rosea typically lasts 3 to 8 weeks. Testing for secondary syphilis is commonly done as secondary syphilis may look like pityriasis rosea.
- Treatment is symptomatic and includes antihistamines for itching.

For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 131, ''Pediatric Exanthems,'' by Michael S. Weinstock and Michael S. Catapano.

85 MUSCULOSKELETAL DISORDERS IN CHILDREN

David M. Cline

PATHOPHYSIOLOGY

- The long bones of children are generally less dense and more porous than the long bones of adults. The resulting increased compliance contributes to the tendency of children's long bones to respond to mechanical stress by bowing and buckling rather than fracturing through and through, as in adult fracture patterns.
- The periosteum of the diaphysis and the metaphysis is thicker in children and is continuous from the metaphysis to the epiphysis, surrounding and protecting the mechanically weaker physis. This physeal weakness is related to the reduced oxygen tension found in the hypertrophic zone of the physis, a location of frequent fractures within the physis.
- The ligaments of children are stronger and more compliant than those of adults.

CHILDHOOD PATTERNS OF INJURY

- The growth plate (physis) is the weakest point in children's long bones and a frequent site of fractures. The ligaments and periosteum are stronger than the physis, tolerating mechanical forces at the expense of physeal injury.
- The blood supply to the physis arises from the epiphysis, so separation of the physis from the epiphysis may be disastrous for future growth.
- The Salter-Harris classification is widely used to describe fractures involving the growth plate (Fig. 85-1).

TYPE I PHYSEAL FRACTURE

- In type I physeal fracture (6 percent of all physeal injuries), the epiphysis separates from the metaphysis. The reproductive cells of the physis stay with the epiphysis. There are no bony fragments. Bone growth is undisturbed.
- Diagnosis of this injury is suspected clinically in children with point tenderness over a growth plate.

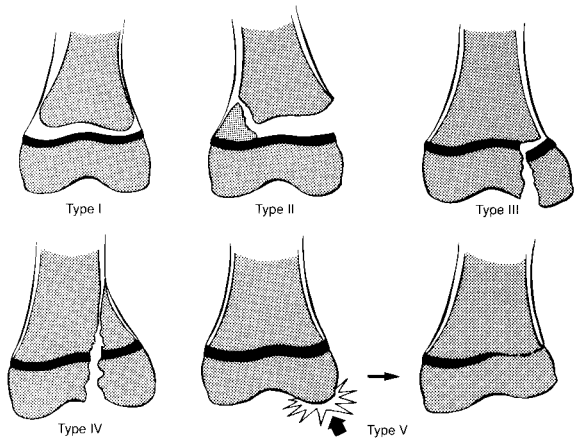

FIG. 85-1 Salter-Harris classification of physeal injuries. (Reproduced with permission from Tolo VT, Wood B: *Pediatric Orthopedics in Primary Care.* Baltimore, Williams & Wilkins, 1994.)

On x-ray, the only abnormality may be an associated joint effusion. There may be epiphyseal displacement from the metaphysis.
- Treatment consists of splint immobilization, ice, elevation, and referral.

TYPE II PHYSEAL FRACTURE

- Type II physeal fracture is the most common (75 percent) physeal fracture.
- The fracture goes through the physis and out through the metaphysis. The periosteum remains intact over the metaphyseal fragment but is torn on the opposite side. Growth is preserved since the physis remains with the epiphysis.
- Treatment is closed reduction with analgesia and sedation followed by cast immobilization.

TYPE III PHYSEAL FRACTURE

- The hallmark of type III physeal fracture is an intraarticular fracture of the epiphysis with the cleavage plane continuing along the physis. This injury usually involves the proximal or distal tibia and accounts for 8 percent of all physeal injuries.
- The prognosis for bone growth depends on the circulation to the epiphyseal bone fragment and is usually favorable.
- Reduction of the unstable fragment with anatomic alignment of the articular surface is critical. Open reduction is often required.

TYPE IV PHYSEAL FRACTURE

- The fracture line of type IV physeal fractures begins at the articular surface and extends through the epiphysis, physis, and metaphysis.
- This most often involves the distal humerus, accounting for 8 percent of all physeal injuries.
- Open reduction is required to reduce the risk of premature arrest of bone growth.

TYPE V PHYSEAL FRACTURE

- Type V physeal fracture is a rare (1 percent) pattern usually involving the knee or ankle. The physis is essentially crushed by severe compressive forces. There is no epiphyseal displacement.
- The diagnosis is often difficult. An initial diagnosis of sprain or type I injury may prove incorrect when later growth arrest occurs. X-rays may look normal or demonstrate focal narrowing of the epiphyseal plate. There is usually an associated joint effusion.
- Treatment consists of cast immobilization, avoidance of weight bearing, and close orthopedic follow-up in anticipation of focal arrest of bone growth.

TORUS FRACTURES

- Children's long bones are more compliant than those of adults and tend to bow and bend under forces that might fracture an adult's bone. Torus (also called cortical or buckle) fractures involve a bulging or buckling of the bony cortex, usually of the metaphysis.
- Patients have point tenderness over the fracture site and soft tissue swelling. Radiographs may be subtle but show cortical disruption.
- Torus fractures are not typically angulated, rotated, or displaced, so reduction is rarely necessary. Splinting or casting in a position of function for 3 to 4 weeks with orthopedic follow-up is recommended.

GREENSTICK FRACTURES

- In greenstick fractures, the cortex and periosteum are disrupted on one side of the bone but intact on the other.
- Treatment is closed reduction and immobilization.

PLASTIC DEFORMITIES

- Plastic deformities are seen in the forearm and lower leg in combination with a completed fracture in the companion bone. The diaphyseal cortex is deformed, but the periosteum is intact.

FRACTURES ASSOCIATED WITH CHILD ABUSE

- Certain injury patterns are consistently seen in abused children, particularly multiple fractures in various stages of healing.
- Twisting injuries create spiral fractures in long bones, highly specific for abuse in nonambulatory children. In ambulatory children, spiral fractures may occur from unintentional injury, the classic

example being the spiral fracture of the lower third of the tibia (toddler's fracture), but this can also be seen with abuse.
- The injury pattern most closely associated with abuse is the chip fracture of the metaphysis. The tight attachment of the periosteum to the metaphysis will cause avulsion of little chips of the bone with pulling. There is exuberant callus formation and periosteal new bone formation. With direct trauma, subperiosteal hemorrhage characteristically lifts the periosteum off the bone, where it appears as an opacified line.
- Fragmentation of the clavicle and acromion and separation of the costochondral junctions of the ribs are very suggestive of abuse.
- Bony injuries from shaking are similar to those from twisting but also include spinal compression fractures and other vertebral injuries.
- Distraction injuries to the long bones cause hemorrhagic separation of the distal metaphysis, creating a lucency proximal to the physis (bucket handle fracture).
- Squeezing injuries create rib fractures that are highly suggestive of abuse.

SELECTED PEDIATRIC ORTHOPEDIC PROBLEMS

CLAVICULAR FRACTURE

- Clavicular fracture is the most common fracture in children.
- Fractures may occur in the newborn during difficult deliveries. Babies may have nonuse of the arm. If the fracture was not initially appreciated, parents may notice a bony callus at 2 to 3 weeks of age.
- In older infants and children, the usual mechanism is a fall onto the outstretched arm or shoulder.
- Care of the patient with a clavicular fracture is directed toward pain control. Even if anatomic alignment is not achieved in the emergency department (ED), displaced fractures usually heal well, although patients may have a residual bump at the fracture site.
- ''Figure of eight'' shoulder abduction restraints have been the traditional treatment, but many patients have more pain with this device. Many orthopedists find a sling-and-swathe or shoulder immobilizer to be equally effective and less painful. Both devices should be worn day and night for 2 weeks, then during the day for another few weeks.

SUPRACONDYLAR FRACTURES

- The most common elbow fracture in childhood is the supracondylar fracture of the distal humerus. The fracture occurs when children fall on their outstretched arms.
- The close proximity of the brachial artery to the fracture predisposes the artery to injury. Subsequent arterial spasm or compression by casts may further compromise distal circulation. A forearm compartment syndrome, Volkmann's ischemic contracture, may occur.
- Symptoms include pain in the proximal forearm upon passive finger extension, stocking-glove anesthesia of the hand, and hard forearm swelling. Children complain of pain on passive elbow flexion and maintain the forearm pronated.
- Pulses may remain palpable at the wrist despite serious vascular impairment.
- Injuries to the ulnar, median, and radial nerves are common too, occurring in 5 to 10 percent of all supracondylar fractures.
- X-rays show the injury, but the findings may be subtle. A posterior fat-pad sign is indicative of intraarticular effusion and thus fracture. Normally, the anterior humeral line, a line drawn along the anterior distal humeral shaft, should bisect the posterior two-thirds of the capitellum on the lateral view. In subtle supracondylar fractures, the line often lies more anteriorly.
- Splinting of the elbow in extension is recommended. In cases of neurovascular compromise, immediate fracture reduction is indicated. If an ischemic forearm compartment is suspected after reduction, surgical decompression or arterial exploration may be indicated. Open reduction is often required.

RADIAL HEAD SUBLUXATION (''NURSEMAID'S ELBOW'')

- Subluxation of the radial head is a very common injury, seen most often in children between ages of 1 to 4. The typical history is that the child has been lifted up by an adult pulling on the child's hand or wrist. Sometimes there is a history of trauma and sometimes there is no event at all but the child refuses to use the arm.
- The patient holds the arm close to the body, flexed at the elbow with the forearm pronated. Gentle exam reveals no tenderness to direct palpation, but any attempt to supinate the forearm or move the elbow causes pain.
- If the history and exam are classic, radiographs

are not needed, but if the history is atypical or there is a point tenderness or sign of trauma, x-rays should be taken.
- To reduce the injury, one hand should be held over the child's radial head and the other hand should hold the child's hand. Then, simultaneously, the physician should press down on the radial head with the thumb while fully flexing the elbow and supinating the forearm. There may be a "click" with reduction. Usually the child will resume normal activity within 15 min if reduction is achieved. If the child is not better after a second reduction attempt, alternate diagnoses and radiographs should be considered.

SLIPPED CAPITAL FEMORAL EPIPHYSIS

- Slipped capital femoral epiphysis (SCFE) is more common in boys; peak incidence is between ages 12 and 15 in boys and between ages 10 and 13 in girls.
- With a chronic SCFE, children complain of dull pain in the groin, anteromedial thigh, and knee, which becomes worse with activity. With walking, the leg is externally rotated and the gait is antalgic. Hip flexion is restricted and accompanied by external rotation of the thigh.
- Acute SCFE is due to trauma or may occur in a patient with preexisting chronic SCFE. Patients are in great pain, with marked external rotation of the thigh and leg shortening. The hip should not be forced through full range of motion, as this may displace the epiphysis further.
- The differential includes septic arthritis, toxic synovitis, Legg-Calvé-Perthes disease, and other hip fractures.
- Children with SCFE are not febrile or toxic and have normal white blood cell (WBC) counts and erythrocyte sedimentation rates (ESRs).
- On x-ray, medial slips of the femoral epiphysis will be seen on anteroposterior (AP) views, while frog-leg views detect posterior slips. In the AP view, a line along the superior femoral neck should transect the lateral quarter of the femoral epiphysis, but not if the epiphysis is slipped.
- The management of SCFE is operative. The main long-term complication is avascular necrosis of the femoral head.

TRANSIENT TENOSYNOVITIS OF THE HIP

- Transient tenosynovitis is the most common cause of hip pain in children below age 10. The peak age is 3 to 6 years, with boys affected more than girls. The cause is unknown.
- Symptoms may be acute or gradual. Patients have pain in the hip, thigh, and knee and an antalgic gait. Pain limits the hip's range of motion. There may be a low-grade fever, and patients do not appear toxic.
- The WBC and ESR are usually normal. Radiographs of the hip are normal or show a mild-to-moderate effusion. The main concern is differentiation from septic arthritis, particularly if the patient is febrile, with elevation of WBC or ESR and effusion.
- Diagnostic arthrocentesis is required, either with fluoroscopic or ultrasound guidance or in the operating room. The fluid in transient tenosynovitis is a sterile clear transudate.
- Once septic arthritis and hip fracture have been ruled out, patients can be treated with crutches to avoid weight bearing, nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen 10 mg/kg, and close follow-up.

ACUTE SUPPURATIVE ARTHRITIS

- Septic arthritis occurs in all ages but especially in children under 3.
- The hip is most often affected, followed by the knee and elbow.
- The diagnosis is critical because, left untreated, purulent joint infection leads to total joint destruction. Bacteria access the joint hematogenously, by direct extension from adjacent osteomyelitis or from inoculation as in arthrocentesis or femoral venipuncture.
- The organisms vary with the children's ages (see Table 85-1). *Haemophilus influenzae* as the cause has diminished due to widespread vaccination.
- Although systemic symptoms can be subtle in the newborn, older children will appear ill, with high fever and irritability. The affected joint is very painful and shows warmth, swelling, and severe tenderness to palpation and movement.
- Children with hip or knee infection will limp or not walk at all. The child maintains the infected hip in flexion, abduction, and external rotation.
- X-rays show joint effusion, but this is nonspecific.
- The differential includes osteomyelitis, transient tenosynovitis, cellulitis, septic bursitis, acute pauciarticular juvenile rheumatoid arthritis (JRA), acute rheumatic fever, hemarthrosis, and SCFE.
- Distinguishing septic arthritis from osteomyelitis may be quite difficult. Osteomyelitis is more tender over the metaphysis, whereas septic arthri-

TABLE 85-1 Initial Antibiotic Therapy of Acute Suppurative Arthritis in Children

AGE	SUSPECTED ORGANISM	ANTIBIOTICS
Newborn (0–2 months)	*Staphylococcus* aureus	Methicillin or nafcillin*
	Group B *Streptococcus*	Ampicillin or penicillin and gentamicin
	Gram-negative bacilli	Cefotaxime/ceftriaxone
	Neisseria gonorrhoeae	Cefotaxime/ceftriaxone
	Unknown	Methicillin or nafcillin* and cefotaxime/ceftriaxone
Infant (2–36 months)	*Haemophilus influenzae*	Cefuroxime or cefotaxime/ceftriaxone
	Strep. species	Penicillin G
	Staph. aureus	Methicillin or nafcillin*
	Gram-negative bacilli	Cefotaxime/ceftriaxone
	Unknown	Methicillin or nafcillin* and cefotaxime/ceftriaxone
Child (>36 months)	*Staph. aureus*	Methicillin or nafcillin*
	Strep. species	Penicillin G, other β-lactams, clindamycin
	Gram-negative bacilli	Cefotaxime/ceftriaxone
	N. gonorrhoeae	Ceftriaxone or penicillin G
	Unknown	Methicillin or nafcillin* and cefotaxime/ceftriaxone

* Vancomycin if methicillinase-resistant *Staph. aureus* is suspected.

tis is more tender over the joint line. Joint motion is much more limited in septic arthritis.
- Prompt arthrocentesis is the key to diagnosis, either at the bedside or, in the case of the hip, in the operating room or under ultrasound. Synovial fluid shows WBCs and organisms.
- Prompt joint drainage is critical, either in the operating room in the case of the hip or arthroscopically or via arthrocentesis in more superficial joints. Suggested antibiotics are listed in Table 85-1.
- The prognosis depends on the length of time between symptoms and treatment, which joint is involved (worse for the hip), presence of associated osteomyelitis (worse), and the patient's age (worse for youngest children).

AVASCULAR NECROSIS SYNDROMES

LEGG CALVÉ-PERTHES DISEASE

- Legg Calvé-Perthes disease is essentially avascular necrosis of the femoral head with subchondral stress fracture. Collapse and flattening of the femoral head ensues, with potential of subluxation.
- The hip is painful, with limited range of motion, muscle spasm, and soft tissue contractures. Onset of symptoms is between ages 4 and 9. The disease is bilateral in 10 percent of patients. Children have a limp and chronic dull pain in the groin, thigh, and knee, which becomes worse with activity. Systemic symptoms are absent.
- Hip motion is restricted; there may be a flexion-abduction contracture and thigh muscle atrophy.
- Initial radiographs (in the first 1 to 3 months) show widening of the cartilage space in the affected hip and a diminished ossific nucleus of the femoral head. The second sign is subchondral stress fracture of the femoral head. The third finding is increased femoral head opacification. Finally, deformity of the femoral head occurs, with subluxation and protrusion of the femoral head from the acetabulum.
- Bone scan and magnetic resonance imaging are very helpful in making this diagnosis, showing bone abnormalities well before plain films would do so.
- The differential diagnosis includes toxic tenosynovitis, tuberculous arthritis, tumors, and bone dyscrasias.
- In the ED, the most important thing is to consider this chronic but potentially crippling condition. Nearly all children are hospitalized initially for traction.

OSGOOD-SCHLATTER DISEASE

- Osgood-Schlatter disease is a common syndrome that affects preteen boys more than girls. Repetitive stress on the tibial tuberosity by the quadriceps muscle initiates inflammation of the tibial tuberosity without avascular necrosis.

- Children have pain and tenderness over the anterior knee, this becomes worse with knee bending and better with rest.
- The patellar tendon is thick and tender, with the tibial tuberosity enlarged and indurated.
- X-rays show soft tissue swelling over the tuberosity and patellar tendon thickening without knee effusion. Normally, the ossification site at the tubercle at this age will be irregular, but the prominence of the tubercle is characteristic of Osgood-Schlatter disease.
- The disorder is self-limited. Acute symptoms improve after restriction of physical activities involving knee bending for 3 months. Crutches may be necessary, though a knee immobilizer or cylinder cast are only rarely needed. Exercises to stretch taut and hypertrophied quadriceps muscles are also helpful.

SELECTED PEDIATRIC RHEUMATOLOGIC PROBLEMS

HENOCH-SCHÖNLEIN PURPURA

- Henoch-Schönlein purpura (HSP) is a self-limited generalized leukocytoclastic vasculitis mediated by immune complexes.
- Palpable purpura, the classic vasculitic rash, appears on the trunk, buttocks, and legs.
- HSP also involves the glomeruli, with resulting hematuria and proteinuria.
- Involvement of the bowel wall causes, colicky abdominal pain and may lead to melena, hematochezia, or intussusception.
- A polymigratory periarticulitis occurs in most children.
- HSP is largely a clinical diagnosis; useful lab tests include urinalysis, complete blood cell count, tests of renal function, and sometimes tests for collagen vascular disease.
- Hospital admission is indicated when the diagnosis is in doubt, dehydration occurs, or when gastrointestinal or renal complications require close observation.
- Arthritis, when present as an isolated symptom, can be treated with salicylates.
- Chronic renal damage, sometimes requiring dialysis, occurs in 7 to 9 percent of children with HSP.

ACUTE RHEUMATIC FEVER

- Acute rheumatic fever (ARF) is an acute inflammatory multisystem illness affecting primarily school-age children. It is not common in the United States, but there have been recent epidemics.
- ARF is preceded by infection with certain strains of group A β-hemolytic streptococcus, which stimulates antibody production to host tissues. Children develop ARF 2 to 6 weeks after symptomatic or asymptomatic streptococcal pharyngitis.
- Arthritis, which occurs in most initial attacks, is migratory and polyarticular, primarily affecting the large joints.
- Carditis occurs in one-third of patients and can affect valves, muscle, and pericardium. Carditis confers greatest mortality and morbidity.
- Sydenham's chorea occurs in 10 percent of patients and may occur months after the initial infection. Manifestations include sudden, aimless, irregular movements and muscle weakness.
- The rash—erythema marginatum—is fleeting, faint, and serpiginous, usually accompanying carditis. Subcutaneous nodules, found on the extensor surfaces of extremities, are quite rare.
- Laboratory tests are used to confirm prior strep infection (throat culture and strep serology) or to assess carditis (electrocardiogram, chest x-ray, and echocardiogram).
- The differential includes JRA, septic arthritis, Kawasaki disease, leukemia, and other cardiomyopathies and vasculitides.
- Significant carditis is managed with prednisone 1 to 2 mg/kg/d initially with admission to the hospital. Arthritis is treated with high-dose aspirin (75 to 100 mg/kg/d) to start.
- All children with ARF are treated with penicillin (PCN) (or erythromycin if allergic): benzathine PCN 1.2 million U intramuscularly, procaine PCN G 600,000 U intramuscularly daily for 10 days, or oral PCN VK 25,000 to 50,000 U/kg/d divided four times a day for 10 days.

POSTSTREPTOCOCCAL REACTIVE ARTHRITIS

- Because of increased group A β-hemolytic strep infections, poststreptococcal reactive arthritis (PSRA) is also increasing. PSRA is a sterile inflammatory nonmigratory mono- or oligoarthritis occurring with infection—due to β-hemolytic strep and also *Staphylococcus* and *Salmonella*—at a distant site.
- Unlike ARF, PSRA is not associated with carditis and in general is a milder illness.
- PSRA is responsive to nonsteroidal anti-inflammatory drugs (NSAIDs).

JUVENILE RHEUMATOID ARTHRITIS

- The group of diseases comprising juvenile rheumatoid arthritis (JRA) all manifest chronic noninfectious synovitis and arthritis as well as systemic manifestations.
- Pauciarticular disease is the most common form, usually involving a single large joint such as the knee. Permanent joint damage occurs infrequently.
- Polyarticular disease occurs in one-third of cases. Both large and small joints are affected, and there may be progressive joint damage.
- Systemic JRA occurs in 20 percent of patients. This form is associated with high fevers and chills. Extraarticular manifestations are common, including a red macular coalescent rash, hepatosplenomegaly, and serositis. The arthritis in this form may progress to permanent joint damage.
- In the ED, lab tests focus mostly on excluding other diagnoses. Arthrocentesis may be necessary to exclude septic arthritis, particularly in pauciarticular disease.
- X-rays initially show joint effusions but are nonspecific. The diagnosis of JRA will likely not be made in the ED.
- Initial therapy for patients with an established diagnosis includes aspirin or another NSAID. Glucocorticoids are occasionally used—for example, for unresponsive uveitis or decompensated perior myocarditis.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 132, "Musculoskeletal Disorders in Children," by Richard A. Christoph.

86 SICKLE CELL ANEMIA IN CHILDREN

David M. Cline

- Sickle cell emergencies in children include vasoocclusive crises, hematologic crises, and infections. All children with sickle cell disease (SCD) who present with fever, pain, respiratory distress, or a change in neurologic function require a rapid and thorough emergency department (ED) evaluation.

EPIDEMIOLOGY

- SCD is the most common pediatric genetic condition encountered in emergency medicine.
- In the United States about 8 percent of the African-American population carries the hemoglobin (*HgbS*) gene and about 0.15 percent (approximately 1 in 500) of these persons are homozygous (*HgbSS*).[1]
- SCD is associated with a significant mortality rate; 20 to 30 percent of all deaths from SCD occur before age 5 years, with a mean age at death of 14 years.[2]
- The highest mortality rate occurs in children between 1 and 3 years of age, with sepsis being the leading cause of death.[2]
- Clinical effects of the disorder can begin in infancy but usually are not seen until 5 to 6 months of age because high levels of fetal hemoglobin are present after birth, and the β-hemoglobin subunit is not predominant until about 3 months of age.[1]

PATHOPHYSIOLOGY

- The genetic abnormality responsible for the sickling process is caused by a single amino acid substitution of valine for glutamic acid in the β subunit of the hemoglobin molecule.
- Affected red blood cells undergo repeated cycles of sickling and unsickling, with the HgbS strands polymerizing abnormally in response to deoxygenation. Without an attached O_2 molecule, they

tend to coalesce and stretch into long monofilaments, resulting in the distorted sickle shape of the red cell membrane. These irreversibly sickled cells diminish blood viscosity, causing hemolysis and obstructing the microcirculation (vasoocclusive phenomenon) of end-organ tissues.[1,3]

VASOOCCLUSIVE CRISES

- Vasoocclusive sickle episodes are due to intravascular sickling, which leads to tissue ischemia and infarction.[4] Bones, soft tissue, viscera, and the brain may all be affected.

PAIN CRISES

- **Clinical Features** The classic sickle cell pain crisis is characterized by episodes of acute pain that sometimes are triggered by stress, extremes of cold, dehydration, hypoxia, or infection.[4–6] Most episodes occur without an obvious cause.[6]
- Typically there are no physical findings except pain and perhaps local tenderness, swelling, and warmth.[7]
- It is rare for clinical symptoms of the disease to appear before 5 to 6 months of age. While young children tend to have pain in the limbs, older children may complain of pain in a variety of locations, including the abdomen and the lumbosacral area.

- **Diagnosis and Differential** Painful crises can be associated with low-grade fever and leukocytosis,[8] but temperatures higher than 38.3°C (101°F) are more likely to be due to an infectious cause than to tissue ischemia.[9]
- Vasoocclusive pain crises usually present in a stereotypical fashion. Atypical pain or new sites of pain warrant further investigation for infection or complications of sickle cell disease.[7] Osteomyelitis or septic arthritis should be considered in the differential diagnosis (see Chap. 85).
- Abdominal pain crises are common and are characterized by abrupt onset, lack of localization, and a recurrent nature.[7]
- It is important to determine whether the abdominal pain in SCD patients has changed substantially in character, quality, duration, severity, and associated symptoms. If such changes are present, infection or other related diagnoses, such as cholecystis, appendicitis, pancreatitis, hepatitis, perforated viscos, pelvic inflammatory disease, and other gynecologic pathology, should be considered and explored.[1,4]
- A complete blood count (CBC) should be obtained, looking for a drop in hematocrit. The white blood cell (WBC) count is typically 12,000 to 18,000 in children in a crisis.[10]
- Reticulocyte count is indicated with a mean value of 12 percent (range, 5 to 15 percent).[10]
- Chest x-rays should be obtained for patients with respiratory complaints.[8]

- **Emergency Department Care and Disposition** Aggressive hydration with oral fluids as tolerated or intravenous (IV) 5% dextrose (D_5) 0.25 normal saline solution (NS) or D_5 0.45 NS at 1 1/2 times the maintenance rate also is indicated.[11]
- Mild to moderate pain often can be managed with oral hydration and analgesics, such as narcotic-acetaminophen combinations and nonsteroidal anti-inflammatory drugs (NSAIDs).[6]
- Parenteral, long-acting narcotics, such as morphine 0.1 to 0.15 mg/kg IV and hydromorphone 0.015 mg/kg IV, are the next step.[12,13]
- Children should be admitted to the hospital if their pain is worsening, their oral fluid intake is inadequate, or they have not achieved adequate pain relief approximately 4 h after the initiation of IV analgesic medications and hydration.[11]

ACUTE CHEST SYNDROME

- Acute chest syndrome is believed to be attributable to a combination of pneumonia, pulmonary infarction, and pulmonary emboli from necrotic bone marrow.[14]
- It is a major cause of death in all patients with SCD, especially those over age 10 years.[2]

- **Clinical Features** Acute chest syndrome should be considered in all patients with SCD who present with complaints of chest pain, especially when it is associated with tachypnea, dyspnea, cough, and other symptoms of respiratory distress. Significant hypoxia and rapid deterioration to respiratory failure can occur.[7]

- **Diagnosis and Differential** Chest x-rays should be obtained but may be normal during the first 72 h. No specific laboratory abnormalities are typical of acute chest syndrome; however, a CBC, reticulocyte count, and blood culture should be obtained.[7]
- Noninvasive pulse oximetry should be instituted, and arterial blood gas analysis is indicated in

the presence of significant oxygen desaturation or respiratory distress. Whereas ventilation and perfusion scans may be useful if the diagnosis of pulmonary embolus is being entertained, pulmonary angiography should be avoided, since contrast material can cause more pulmonary sickling.[11]

- **Emergency Department Care and Disposition** All children in whom the diagnosis of acute chest syndrome is being considered should be monitored closely for changes in the work of breathing and oxygenation. Deterioration can be rapid.[14]
- Supplemental oxygen should be provided if respiratory distress is present or if oxygen saturation is persistently less than or equal to 94%.
- Adequate analgesia for chest pain should be provided (see above), as well as IV hydration with D_5 1/2 NS at 1 to 1 1/2 times maintenance.
- Potential underlying bacterial pneumonia should be treated with empirical antibiotic therapy such as ceftriaxone 75 mg/kg per day or cefotaxime 50 to 75 mg/kg per day divided every 8 h.[14]
- Simple red blood cell transfusion (10 to 15 mL red blood cells per kilogram) or exchange transfusion should be considered in children with severe anemia (hemoglobin level less than 5 g/dL) or rapidly worsening hypoxia.[15] Transfusion decisions should be made early and in consultation with a pediatric hematologist.[16]
- All children with suspected acute chest syndrome should be admitted to the hospital for further care.[14]

ACUTE CENTRAL NERVOUS SYSTEM EVENTS

- **Clinical Features** Acute central nervous system (CNS) crisis should be considered in any patient with SCD who presents with sudden-onset headache or neurologic changes, including hemiparesis, seizures, speech defects, sensory hearing loss, visual disturbances, transient ischemic attacks, dizziness, vertigo, cranial nerve palsies, paresthesias, and inexplicable coma.[17]

- **Diagnosis and Differential** When CNS vasoocclusion is suspected, computed tomography (CT) or magnetic resonance imaging (MRI) of the brain should be done.[18]
- A lumbar puncture is sometimes necessary to rule out subarachnoid hemorrhage.
- No specific hematologic changes are associated with CNS vasoocclusion; however, a CBC and a reticulocyte count should be obtained, and blood typing and screening should be ordered in case an exchange transfusion is necessary.[19]

- **Emergency Department Care and Disposition** Suspected CNS vasoocclusion necessitates immediate stabilization and careful monitoring.
- Once intracranial hemorrhage or infarction is confirmed, 1 1/2 to 2-volume exchange transfusion should be started as soon as possible in consultation with a pediatric hematologist.[18,19]
- A pediatric neurosurgeon should be consulted once intracranial bleeding has been confirmed. All children with diagnosed or suspected CNS vasoocclusion should be admitted to the pediatric intensive care unit for close monitoring and further care.[18]

PRIAPISM

- Priapism, a painful sustained erection in the absence of sexual stimulation, occurs when sickled cells accumulate in the corpora cavernosa.[20] It can affect all males with SCD regardless of age, and severe prolonged attacks can cause impotence.[20,21]
- Patients with priapism should receive IV hydration with D_5 0.45 NS at 1 1/2 to 2 times maintenance, appropriate analgesia, and bladder catheterization if they are unable to void spontaneously.[20,21]
- Treatment options include an oral α-adrenergic agonist (e.g., terbutaline or pseudoephedrine), intrapenile injection of vasodilators (e.g., hydralazine), and/or needle aspiration of the corpora cavernosa. Management and admission decisions should be made promptly in consultation with a urologist and a pediatric hematologist.[20,21]

HEMATOLOGIC CRISES

ACUTE SEQUESTRATION CRISES

- **Clinical Features and Diagnosis** Sequestration crises are the second most common cause of death in children with SCD under age 5 years.[2]
- The spleen of a young child with sickle cell disease can enlarge massively, trapping a considerable portion of the circulation blood volume. This condition can progress quickly to hypotension, shock, and death. Such crises often are preceded by a viral infection.[22]
- Classically, children present with sudden-onset left upper quadrant pain, pallor, and lethargy; a

markedly enlarged, tender, and firm spleen on abdominal examination; and signs of cardiovascular collapse, including hypotension and tachycardia.[23]
- A CBC reveals a profound anemia (hemoglobin drops to less than 6 g/dL or 3 g/dL lower than the patient's baseline level).[24]
- Minor episodes can occur with an insidious onset of abdominal pain, slowly progressive splenomegaly, and a more minor fall in the hemoglobin level (generally the hemoglobin level remains above 6 g/dL).
- Splenic sequestration crises may be characterized by thrombocytopenia and higher than normal reticulocyte counts.
- Less commonly, sequestration can occur in the liver. Clinical features include an enlarged and tender liver with associated hyperbilirubinemia, severe anemia, and an elevated reticulocyte count.[24] Cardiovascular collapse is rare in this condition.[24]

- **Emergency Department Care and Disposition** Early recognition and prompt initiation of treatment are the keys to successful management.
- The goal of treatment is to quickly expand the intravascular volume with the rapid infusion of large amounts of NS or albumin (starting with 20 mL/kg).[23]
- Transfusion with packed red blood cell or whole blood if available often is required and should be instituted immediately.[16,22] Even children with minor episodes should be admitted to the hospital.

APLASTIC EPISODES

- Potentially life-threatening aplastic episodes are precipitated primarily by viral infections but also can be caused by bacterial infections, folic acid deficiency, and bone-marrow-suppressive or toxic drugs.[7]
- These patients usually present with gradual onset of pallor, dyspnea, fatigue, and jaundice.[7]
- A CBC reveals an unusually low hematocrit (10 percent or lower) with decreased or absent reticulocytosis. White blood cell and platelet counts remain stable.
- Pain is not a hallmark of this crisis unless there is an associated vasoocclusive crisis.
- If anemia is severe, the patient should be admitted for red blood cell transfusion to avoid secondary cardiopulmonary complications.[7]

HEMOLYTIC CRISES

- Bacterial and viral infections in children with SCD can precipitate an increasing degree of active hemolysis.
- The onset is usually sudden. A CBC reveals a hemoglobin level decreased from baseline, with markedly increased reticulocytosis. Increased jaundice and pallor are noticed on physical examination, in addition to other signs and symptoms of the precipitating infection.
- Specific therapy rarely is required. Hematologic values return to normal as the infectious process resolves. Care should be directed toward treating the underlying infection. Close follow-up to monitor hemoglobin and reticulocyte count should be arranged at discharge.

INFECTIONS

- **Clinical Features** Children with SCD are functionally asplenic and have deficient antibody production and impaired phagocytosis.[9] Therefore, bacterial infections, especially with encapsulated organisms, pose a serious and potentially fatal threat to young children with SCD.[25]
- Since sepsis can be rapid, overwhelming, and fatal, particularly in children less than 5 years of age, all children with SCD and fever should be examined quickly and carefully and managed aggressively.

- **Diagnosis and Differential** A CBC, a reticulocyte count, and blood cultures should be obtained for all children with SCD and fever or a history of fever. Clinical signs and symptoms should direct the remainder of the workup, including a lumbar puncture as indicated.
- Knowledge regarding a child's immunization status (particularly whether he or she has been immunized against *Haemophilus influenzae* B and/or pneumococcus) and compliance with the child's home penicillin prophylaxis is helpful.

- **Emergency Department Care and Disposition** Children who are ill-appearing on presentation should be treated parenterally with an antibiotic with activity against *Streptococcus pneumoniae* and *Haemophilus influenzae* (e.g., ceftriaxone 50 mg/kg IV or intramuscular) before evaluation is complete and test results are available.[25]
- One should consider vancomycin if a patient is at high risk for penicillin-resistant pneumococcal infection.[25]
- One should manage septic shock aggressively with

IV fluids, vasoactive medications, and possible transfusion. The patient should be admitted to the hospital. Pediatric intensive care unit admission should be considered.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 133, "Sickle Cell Disease," by Peter J. Paganussi, Thom A. Mayer, and Maybelle Kou.

87 PEDIATRIC URINARY TRACT INFECTIONS

Lance Brown

EPIDEMIOLOGY

- Pediatric urinary tract infections (UTI) occur in 4 to 7 percent of febrile infants,[1] 2 percent of children 1 to 5 years of age, and up to 3 to 5 percent of school-aged girls.[2]

PATHOPHYSIOLOGY

- Urinary tract infection typically develops from retrograde contamination of the lower urinary tract with organisms from the perineum and periurethral area. In neonates, however, UTI typically develops after seeding of the renal parenchyma from hematogenous spread.
- Factors influencing the development of UTI in-

clude virulence of the pathogen, host immunity, congenital urinary tract abnormalities, vesicoureteral reflux, urolithiasis, poor hygiene, voluntary urinary retention, and constipation.[3–8]

CLINICAL FEATURES

- Clinical features vary markedly by age.
- Neonatal UTI may present with a septic-like appearance. Features may include fever, jaundice, poor feeding, irritability, and lethargy.
- Infants and young children typically present with gastrointestinal complaints that may include fever, abdominal pain, vomiting, and change in appetite.
- In older school-aged children over 8 years old, cystitis and urethritis (lower tract disease) typically present with urinary frequency, urgency, and dysuria. Pyelonephritis (upper tract disease) typically presents with fever, chills, back pain, vomiting, and dehydration.

DIAGNOSIS AND DIFFERENTIAL

- Urine culture is the gold standard in the diagnosis of UTI.[2] Of isolated organisms, *Escherichia coli* accounts for the vast majority of infections. Other important pathogens include *Klebsiella, Proteus,* and *Enterobacter* species. *Enterococcus* species, *Staphylococcus aureus,* and group B streptococci are the most common gram-positive organisms and are more common in neonates.
- Because urine culture results are not available to the emergency physician during the initial visit, urine chemical test strips and microscopic urinalysis are often employed to aid in the diagnosis of UTI.
- The leukocyte esterase portion of the test strip reacts to proteins released from the breakdown of white blood cells (WBC). The nitrate portion of the test strip reacts to nitrates converted to nitrites by gram-negative urinary pathogens. The sensitivities of a positive leukocyte esterase test or nitrite test for a positive urine culture are less than 50 percent, particularly in neonates and young infants who may not mount sufficient pyuria to have a positive test strip. Combining pyuria (>5 WBC per high power field) and bacteriuria on urinary microanalysis yields a sensitivity of 65 percent.
- Pyuria, hematuria, and proteinuria are commonly found in UTIs but are also present commonly in the absence of infection.
- The method of specimen collection impacts directly on how a urinary tract infection is defined. Bag specimens are inappropriate for culture because of a high degree of contamination.
- In infants and young children who are not yet toilet trained, bladder catheterization is the preferred method.[2] A positive urine culture is defined as $\geq 5 \times 10^4$ colony forming units (CFU)/mL of a single urinary pathogen.[9] Suprapubic aspiration, although invasive, is also an acceptable means of obtaining a cultured specimen. Growth of a urinary pathogen in any number from a suprapubic aspiration is considered a positive culture.[9]
- In toilet-trained children, a midstream clean catch specimen is preferred.[5] Symptomatic patients with $\geq 10^5$ CFU/mL of a single urinary pathogen are considered to have a positive urine culture.[9]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment and disposition depends on age of the patient and severity of the illness.
- Neonates and infants <2 or 3 months of age with fever and UTI are hospitalized and given intravenous (IV) antibiotics.
- Children over 3 months of age with fever and UTI complicated by vomiting, dehydration, any suspicion of sepsis, or inability to take oral antibiotics are hospitalized for IV antibiotics until they are afebrile and able to take oral medications.
- Children over 3 months of age with fever and uncomplicated UTI (can tolerate oral medication, are not dehydrated, are not immunocompromised, and appear well) may receive intramuscular or IV antibiotics in the emergency department (ED) and be started on oral antibiotics with close outpatient follow-up to arrange imaging studies.
- Children over 3 months of age with cystitis (afebrile UTI) are treated for 5 to 7 days with oral antibiotics with close outpatient follow-up.
- Adolescent girls with cystitis may be treated as adults with a 3-day oral antibiotic regimen.
- Parenteral antibiotics useful for treating UTIs include ampicillin/gentamicin in the neonate, and cefotaxime, ceftriaxone, or gentamicin in children over 3 months of age.
- Oral antibiotics include amoxicillin (be aware of emerging *E. coli* resistance), amoxicillin/clavulanate, trimethoprim-sulfamethoxazole, cephalexin, and cefixime.
- An important part of the follow-up of children with UTI is evaluation for vesicoureteral reflux, renal scarring, active renal infection, or other anatomic anomalies utilizing renal cortical scans,

voiding cystourethrogram, isotope cystogram, and renal ultrasound.[10–13] This testing is arranged as an outpatient or performed during hospitalization and not typically from the ED.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 136, "Pediatric Urinary Tract Infections," by Michael F. Altieri, Mary Camarca, and Thom A. Mayer.

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Section 12

INFECTIOUS DISEASES AND IMMUNOLOGY

88 SEXUALLY TRANSMITTED DISEASES

Gregory S. Hall

- This chapter covers the major sexually transmitted diseases in the United States with the exception of HIV (AIDS) (see Chap. 90). Vaginitis and pelvic inflammatory disease (PID) are covered separately in Chaps. 64 and 65, respectively. Granuloma inguinale (donovanosis) rarely occurs in the United States; readers are referred to the reference section for more information on this disease.[1–4]

CHLAMYDIAL INFECTIONS

- *Chlamydia trachomatis* is an obligate intracellular bacterium that can cause urethritis, epididymitis, and proctitis in men and urethritis, cervicitis, PID, and infertility in women.
- Asymptomatic infection in both sexes is common—patients with gonorrhea have a high incidence of coinfection with *Chlamydia.*
- The incubation period varies from 1 to 3 weeks, with symptoms including mild urinary burning, purulent or mucoid urethral discharge, vaginal discharge, vaginal irritation or pain, scrotal/epididymal pain with or without swelling, abdominal/pelvic pain, and even peritonitis.
- Diagnosis is usually achieved via mucosal swabs utilizing direct immunofluorescence, enzyme linked immunosorbent assays (ELISAs), or DNA probes for *Chlamydia.* Direct culture of the organism is possible but has a relatively low yield.
- Treatments of choice for uncomplicated chlamydial infections (not PID) include azithromycin 1 g PO single dose (considered safe in pregnancy) or doxycycline 100 mg PO bid for 7 days (see Table 88-1 for alternatives).
- Treatment should also be given for possible coinfection with gonorrhea.

GONOCOCCAL INFECTIONS

- *Neisseria gonorrhoeae,* a gram-negative diplococcus, causes urethritis, cervicitis, PID and infertility in women and urethritis, epididymitis, and prostatitis in men.
- Symptoms can include dysuria, purulent urethral discharge, vaginal discharge, scrotal/epididymal pain with or without swelling, abdominal/pelvic pain, and sometimes peritonitis.
- Rectal infection/proctitis and pharyngeal colonization (usually asymptomatic) can occur in both sexes.
- The incubation period ranges from 3 to 14 days; asymptomatic infection is common.
- Dissemination of gonorrhea occurs in 2 percent of patients (usually women) and presents with an initial febrile bacteremic stage with skin lesions (tender pustules on a red or hemorrhagic base) primarily on the extremities, tenosynovitis, and myalgias.
- During the second phase of disseminated gonococcal infection (GC) the initial symptoms subside and are followed by mono- or oligoarticular arthritis, with purulent synovial fluid.
- Diagnosis of uncomplicated gonorrhea is usually established via cervical or urethral swab with culture on a selective medium (sensitivity of 80 to 90 percent).
- Diagnosis of disseminated GC is often clinical—

TABLE 88-1 Antimicrobial Therapy for Sexually Transmitted Diseases

DISEASE	RECOMMENDED TREATMENT	ALTERNATIVE
Chlamydial infection	Azithromycin 1 g PO single dose *or* Doxycycline 100 mg PO bid for 7 d	Ofloxacin 300 mg PO bid for 7 d *or* Erythromycin 500 mg PO qid for 7 d
Gonococcal infections	Ceftriaxone 125 mg IM single dose *or* Cefixime 400 mg PO single dose *or* Ciprofloxacin 500 mg PO single dose *or* Ofloxacin 400 mg PO single dose	
Gonococcal, disseminated	Ceftriaxone 1 g IV daily for 7–10 d, or for 2–3 d, followed by cefixime 400 mg PO bid or ciprofloxacin 500 mg PO bid to complete 7–10 d total therapy	Ceftizoxime or cefotaxime, 1 g IV q 8 h for 2–3 d or until improved, followed by cefixime 400 mg PO bid or ciprofloxacin 500 mg PO bid to complete 7–10 d total therapy
Trichomoniasis	Metronidazole 2 g PO single dose	Metronidazole 500 mg PO bid for 7 d
Syphilis, first-degree, second-degree, early latent	Benzathine penicillin G 2.4 million U IM single dose	Doxycycline 100 mg PO bid for 14 d
Syphilis, late latent or unknown	Benzathine penicillin G 2.4 million U IM 3 doses 1 week apart	Doxycycline 100 mg PO bid for 28 d
Herpes simplex infections	Acyclovir 400 mg PO tid for 7–10 d *or* Valacyclovir 1 g PO bid for 7–10 d	Famciclovir 250 mg PO tid for 7–10 d
Chancroid	Azithromycin 1 g PO single dose *or* Ceftriaxone 250 mg IM single dose	Ciprofloxacin 500 mg PO bid for 3 d Erythromycin base 500 mg PO qid for 7 d
Lymphogranuloma venereum	Doxycycline 100 mg PO bid for 21 d	Erythromycin 500 mg PO qid for 21 d

ABBREVIATIONS: bid = twice a day; IM = intramuscular; IV = intravenous; PO = oral; q = every; qid = four times a day; tid = three times a day.
SOURCE: Adapted from Centers for Disease Control and Prevention: *MMWR* 47:RR-1, 1998.

culture of blood, skin lesions, or synovial fluid is positive in only 20 to 50 percent of patients (cultures of cervix, rectum, and pharynx may improve the yield).
- Effective therapy for uncomplicated gonorrhea (not PID) includes single-dose regimens of cefixime, ceftriaxone, or a fluoroquinolone (see Table 88-1).
- Disseminated gonorrhea is treated initially with parenteral ceftriaxone (up to 1 g/day).
- Treatment for possible coinfection with *Chlamydia* should also be given.

TRICHIMONAS INFECTION

- *Trichomonas vaginalis* is a flagellated protozoan that causes vaginitis with discharge, urethritis with dysuria, and occasionally abdominal pain.
- The majority of men infected with *Trichomonas* are asymptomatic, but some have dysuria. Women can also have asymptomatic infection.
- The incubation period ranges from 3 to 28 days, and diagnosis is made by microscopic exam of saline wet preparations of urethral or vaginal discharge or spun urine samples, revealing the classic flagellated motile parasites.
- Metronidazole (2 g PO as a single dose) is the treatment of choice for men and women, but it should be avoided in the first trimester of pregnancy. (Pregnant women may use clotrimazole 100-mg vaginal suppositories at bed time for 2 weeks.)

GENITAL WARTS

- Human papillomaviruses (HPV) are DNA viruses that are transmitted by direct contact and cause venereal or anogenital warts.

- The incubation period from contact to appearance of warts is usually 3 to 4 months.
- Venereal warts commonly occur at the urethra, frenulum, and coronal sulcus of the penis and in perianal regions in men; in women they are common at the posterior introitus and adjacent labia, in the vagina, and on the cervix. They often spread to other parts of the perineum (vulva and anus) as well.
- Diagnosis is often clinical (but may be confirmed by skin biopsy and histologic methods).
- Treatment is not usually attempted acutely in the ED setting. Most often it includes topical podophyllin or cryotherapy in a physician's office or outpatient setting.

SYPHILIS

- Syphilis is caused by the spirochete *Treponema pallidum,* which is transmitted through direct contact at mucous membranes or nonintact skin.
- Classically syphilis infection is divided into three stages: primary, secondary, and tertiary (or latent).
- Primary syphilis is characterized by a painless chancre or ulcer with indurated borders on the penis, vulva, or other areas of sexual contact; it appears approximately 3 weeks after acquisition. The primary chancre heals spontaneously over the next 3 to 6 weeks. Generally there are no constitutional symptoms in this stage.
- Secondary syphilis occurs 3 to 6 weeks after the primary chancre heals and includes lymphadenopathy and a nonpruritic, polymorphous rash (most often dull, red, and papular or maculopapular), which starts on the trunk or flexor surfaces of the extremities and spreads to involve the palms and soles.
- Constitutional symptoms are common during the secondary stage and may include fever, malaise, headache, and sore throat. Mucous membrane involvement with oral lesions and condyloma lata (wart-like growths) in the anogenital region may also occur.
- The secondary stage resolves spontaneously and may be followed years later by the tertiary (latent) stage of syphilis (though now uncommon in the United States).
- Specific features of tertiary syphilis include peripheral neuropathy (tabes dorsalis), meningitis, dementia, aortitis with aortic valve insufficiency, and thoracic aortic aneurysm formation.
- During the early stages, diagnosis may be made by dark-field microscopic identification of treponemes on a specimen obtained from the primary chancre or secondary oral or condylomata lesions.
- Serologic diagnosis may be made with nontreponemal tests (RPR or VDRL), which become positive about 14 days after the primary chancre appears (false-positive rate of 1 to 2 percent in general population) or with specific treponemal tests (FTA-ABS), which are more sensitive and specific but technically more difficulty to perform.
- Treatment: Syphilis in all of its stages remains uniformly sensitive to penicillin, the drug of choice. (See treatment regimens in Table 88-1.)

HERPES SIMPLEX INFECTIONS

- Herpes simplex virus type 2 (HSV-2) or less commonly type 1 (HSV-1) can cause genital infection via transmission through direct contact with mucosal surfaces or nonintact skin.
- Primary infections present after an incubation period of 8 to 16 days, with painful clusters of vesicular or pustular lesions on a red base. These then ulcerate and/or coalesce; they are often accompanied by inguinal adenopathy (80%).
- Additional symptoms common in primary genital herpes include fever, headache, myalgias, and dysuria. Urinary retention and aseptic meningitis can also occur.
- Left untreated, the primary illness lasts 2 to 3 weeks with complete healing of skin lesions, but the virus usually remains latent and is shed in urogenital tract secretions (even in asymptomatic patients). Recurrent or secondary infection, usually of milder and shorter duration, occurs in 60 to 90 percent of patients.
- Diagnosis is usually made on clinical grounds based on the characteristic appearance of the skin lesions, but the virus may be cultured from vesicular fluid (more reliable than the Tzanck prep exam of vesicular fluid for characteristic intranuclear inclusions).
- The treatment of choice for primary genital herpes is acyclovir or valacyclovir for 7 to 10 days. In those cases severe enough to require hospitalization, treatment with IV acyclovir 5 to 10 mg/kg body weight every 8 h may be given. (See Table 88-1 for specific treatment regimens.)
- Treatment for episodes of recurrent genital herpes is with a 5-day course of oral acyclovir, vala-

cyclovir, or famciclovir. If started at the onset of symptoms, this may reduce the severity and duration of the episode.

CHANCROID

- Chancroid—caused by *Haemophilus ducreyi,* a gram-negative bacillus—is more commonly seen in the tropics but has shown an increasing incidence in the United States.
- Following an incubation period of 3 to 10 days, a tender papule appears on the external genitalia. It then enlarges to form a painful, purulent ulcer with irregular edges (multiple ulcers may be present in 50 percent of cases).
- Painful inguinal adenopathy (usually unilateral) develops in about half of the untreated patients—sometimes forming a mass of matted lymph nodes (bubo) that may suppurate and drain spontaneously.
- Diagnosis is usually clinical (with care to exclude syphilis), but sometimes the organism can be cultured from the ulcer or bubo.
- Treatment regimens include erythromycin, ceftriaxone, or azithromycin (see Table 88-1 for regimens). Buboes may be aspirated to relieve pain from swelling but should not be excised.

LYMPHOGRANULOMA VENEREUM

- Lymphogranuloma venereum (LGV) is caused by several serotypes of *Chlamydia trachomatis,* which are endemic to some regions of the world but still uncommon in the United States.
- The primary lesion of LGV consists of a painless small papule or vesicle on the genitals that forms 5 to 21 days after exposure, often goes unnoticed, and heals spontaneously.
- With anal intercourse, primary LGV may present as painful mucopurulent or bloody proctitis.
- Several weeks to months after the primary lesion heals, painful inguinal adenopathy (usually unilateral) develops. The lymph nodes become matted together (bubo) and often suppurate and drain spontaneously.
- Diagnosis is achieved through serologic testing and culture of LGV from aspirated material taken from a bubo.
- Doxycycline 100 mg PO bid for 21 days is the treatment of choice (see Table 88-1).

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89 TOXIC SHOCK

Leslie McKinney

TOXIC SHOCK SYNDROME

EPIDEMIOLOGY

- Toxic shock syndrome (TSS) was, in the past, primarily associated with the use of tampons, although association with nasal packing has been noted recently.[1]
- The overall incidence of TSS and number of cases associated with the use of tampons have decreased dramatically over the past 20 years. One-third of TSS cases are seen in men, with a mortality rate 3.3 times that of menstruation-related TSS in women.

PATHOPHYSIOLOGY

- Colonization or infection with *Staphylococcus aureus* is associated with the majority of cases of TSS. Production of an exotoxin, toxic shock syndrome toxin (TSST-1), is believed to cause many of the symptoms associated with TSS via direct toxic effects or through release of secondary mediators.
- *Staph. aureus* strains that produce TSST-1 are re-

sponsible for 90% of cases related to menstruation; however, TSST-1 is present in less than half of the non-menstruation-related cases. Other enterotoxins with similar biochemical compositions have been identified, explaining the similar clinical presentations between menstrual and nonmenstrual TSS.[2]

- Certain vaginal conditions—such as neutral pH, increased temperature, and increased availability of oxygen and CO_2—occur during menses and with the use of tampons. These conditions allow increased production of TSST-1 by toxigenic strains of *Staph. aureus*.[3]
- Marked vasodilatation and movement of serum proteins and fluids from the intravascular to the extravascular space are witnessed. This causes hypotension, edema, and dehydration.

CLINICAL FEATURES

- TSS is characterized by high fever, hypotension, diffuse erythrodermatous rash described as painless ''sunburn,'' mucous membrane hyperemia, myalgias, headache, vomiting, diarrhea, and constitutional symptoms that rapidly progress to multisystem dysfunction.
- Hypotension or orthostasis is seen in all cases, and patients appear ill.
- Fever and chills are seen early in the course of TSS.
- The rash associated with TSS typically fades within 3 days and is followed by full-thickness desquamation.
- When TSS is associated with menstruation, women typically present between the third and fifth days of menses.

DIAGNOSIS AND DIFFERENTIAL

- Diagnostic criteria are listed in Table 89-1.
- When considering TSS, evaluation should include arterial blood gases (ABG), complete blood count (CBC) with differential, electrolytes including [Mg^{2+}] and [Ca^{2+}], coagulation panel, urine analysis (UA), and chest x-ray (CXR). Cultures of all potentially infectious sites should be obtained, and tampon removed if present.
- Other syndromes to consider in evaluating patients with the above findings include streptococcal toxic shock syndrome (STSS), Kawasaki disease, staphylococcal scalded-skin syndrome, Rocky Mountain spotted fever (RMSF), and septic shock.

TABLE 89-1 Diagnostic Criteria for Toxic Shock Syndrome

An illness with the following clinical manifestations:
Fever: temperature ≥38.9°C (≥102.0°F)
Rash: diffuse macular erythroderma
Desquamation: 1–2 weeks after onset of illness
Hypotension
Multisystem involvement (three or more of the following):
Gastrointestinal: vomiting or diarrhea at onset of illness
Muscular: severe myalgia or CPK elevation twice normal
Mucous membrane: vaginal, oropharyngeal, or conjunctival hyperemia
Renal: blood urea nitrogen or creatinine at least twice the upper limit of normal for laboratory or urinary sediment with pyuria (≥5 leukocytes per high-power field) in the absence of urinary tract infection
Hepatic: total bilirubin, alanine aminotransferase enzyme, or aspartate aminotransferase enzyme levels at least twice the upper limit of normal for laboratory
Hematologic: platelets <100,000/mL
Central nervous system: disorientation or alterations in consciousness

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of TSS consists of initial management of circulatory shock, use of antistaphylococcal β-lactamase stable antimicrobial agents, and search for a focus of infection. All patients should receive cardiac and noninvasive blood pressure monitoring pulse oximetry, oxygen, two intravenous lines, and a Foley catheter.
- Crystalloid IV fluids should be given initially for hypotension; consideration of a central venous pressure (CVP) or Swan-Ganz catheter may be necessary if there is no response to an initial fluid bolus of 1 to 2 L of normal saline. Large volumes of fluid may be required over the first 24 h.
- If there is no response to a fluid challenge, a dopamine infusion may be started at 3 μg/kg/min so as to maintain a systolic BP of 90 mmHg.
- Fresh-frozen plasma, packed red blood cells (PRBCs), or platelets may be given to correct any coagulation abnormalities.
- All potentially infected sites, including blood, must be cultured within 1 h of starting antibiotic therapy.
- Antistaphylococcal microbial therapy: Typically an antistaphylococcal penicillin is used, such as nafcillin or oxacillin in doses of 1 to 2 g IV every 4 h, or a cephalosporin with β-lactamase stability, such as cefazolin, 2 g every 6 h. In penicillin-allergic patients, clindamycin, vancomycin, or possibly cephalosporins may be used.
- Other considerations include the use of methylprednisolone and intravenous immunoglobulin in difficult cases.

- Patients are typically admitted to the intensive care unit (ICU).

STREPTOCOCCAL TOXIC SHOCK SYNDROME

EPIDEMIOLOGY

- Streptococcal toxic shock syndrome (STSS), initially defined in 1993, has increased in incidence over the past 10 years with 2000 to 3000 cases per year. STSS is very similar to TSS; however, it is associated with a soft tissue infection that is culture-positive for *Streptococcus pyogenes* or group A streptococci (GAS).
- Called the ''flesh-eating bacteria,'' GAS cause streptococcal necrotizing fasciitis and myositis, leading to mortality rates of 30 to 80 percent.[4]

PATHOPHYSIOLOGY

- Virulent streptococcal pyogenic exotoxins are produced by 90 percent of GAS isolates and are felt to be responsible for causing multisystem failure.[5]
- Infection may begin with minor local trauma not involving disruption of the skin. No portal of entry is identified in 50 percent of cases.[4]

CLINICAL FEATURES

- STSS typically presents with abrupt onset of pain preceding physical findings.[4] Generally there are some signs of soft tissue infection, most commonly affecting the extremities, although truncal signs are possible.
- Necrotizing fasciitis may develop rapidly and carries a poor prognosis.

TABLE 89-2 Diagnostic Criteria for Streptococcal Toxic Shock Syndrome

An illness with the following clinical manifestations:
Hypotension
Multiorgan involvement characterized by two or more of the following:
Renal impairment: creatinine level twice normal
Coagulopathy
Liver involvement: enzyme or bilirubin level twice normal
Acute respiratory distress syndrome
Generalized erythematous macular rash that may desquamate
Soft tissue necrosis, including necrotizing fasciitis or myositis, or gangrene

- Patients are usually febrile, hypotensive, and confused; they later develop multisystem organ dysfunction.
- Diagnostic criteria are listed in Table 89-2.

DIAGNOSIS AND DIFFERENTIAL

- When considering STSS, look for soft tissue infection and culture site. Laboratory evaluation includes CBC with differential, ABG, liver function tests (LFT), serum electrolytes, $[Mg^{2+}]$, $[Ca^{2+}]$, coagulation profile, blood cultures, CXR, UA.
- Consider TSS as well as other infections caused by GAS, *Clostridium perfringens,* and other bacteria, Kawasaki disease, RMSF, and septic shock.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Refer to above treatment plan for TSS and treat similarly, using aggressive fluid therapy and vasopressors as needed.
- Begin antistreptococcal microbial therapy with IV penicillin G 24 million U/day in divided doses, and IV clindamycin 900 mg q 8 h. Substitute erythromycin in penicillin-allergic patients.
- Immediate surgical consultation as debridement of wounds is mandatory.
- Intravenous immunoglobulin may be considered.
- Patients require admission to the ICU.

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90 HIV INFECTIONS AND AIDS

David M. Cline

EPIDEMIOLOGY

- Worldwide, the number of patients with HIV is continuing to grow dramatically, with current estimates of 30.6 million people living with HIV or AIDS.
- AIDS is now the leading cause of death among American men 25 to 44 years old and the third leading cause of death among women in the same age group.[1]
- Risk factors commonly associated with HIV infection include homosexuality or bisexuality, injected drug use, heterosexual exposure, receipt of a blood transfusion prior to 1985, and vertical and horizontal maternal-neonatal transmission.

PATHOPHYSIOLOGY

- HIV is a cytopathic retrovirus that kills infected cells. The viral genes are carried as a single-stranded RNA molecule within the viral particle. Following infection, the virus selectively attacks host cells involved in immune function, primarily CD4 T lymphocytes
- As a result of infection, immunologic abnormalities eventually occur, including lymphopenia, qualitative defects in CD4 T-lymphocyte function, and autoimmune phenomena. Profound defects in cellular immunity ultimately result in a variety of opportunistic infections and neoplasms.
- Transmission of HIV has been shown to occur via semen, vaginal secretions, blood or blood products, breast milk, and transplacental transmission in utero.

CLINICAL FEATURES

- The stages of HIV infection from initial acquisition to end-stage AIDS are described in Table 90-1. Acute HIV infection, essentially indistinguishable from a ''flulike'' illness, usually goes unrecognized but is reported to occur in 50 to 90 percent of patients.[2] The time from exposure to onset of symptoms is usually 2 to 4 weeks; the most common symptoms include fever, sore throat, fatigue, myalgias, and weight loss.
- Seroconversion, reflecting detectable antibody response to HIV, usually occurs 3 to 12 weeks after infection.
- This is followed by a long period of asymptomatic infection during which patients generally have no findings on physical examination except for possible persistent generalized lymphadenopathy.
- The mean incubation time from exposure to the development of AIDS is estimated at 8.23 years for adults and 1.97 years for children under age 5.

TABLE 90-1 Stages of HIV Infection

STAGE	CD4 CELL COUNT, CELLS/μL	CLINICAL MANIFESTATIONS
Acute infection	Normal	Mononucleosis-like syndrome
Asymptomatic infection	>500	Asymptomatic or persistent generalized lymphadenopathy, aseptic meningitis, myopathy, Guillain-Barré syndrome
Early symptomatic	200–500	Thrush, candidal esophagitis, bacterial pneumonia, herpes zoster, oral hairy leukoplakia, B-cell lymphoma, Hodgkin's disease, idiopathic thrombocytopenic purpura, Kaposi's sarcoma,* TB*
Late symptomatic infection (AIDS)	<200	Opportunistic infections and malignancies
End-stage disease	<50	Disseminated CMV or *M. avium* complex

* Frequently present in patients with CD4 cell counts >200; however, both are classified as AIDS indicator conditions.

- Early symptomatic infection is characterized by conditions that are more common and more severe in the presence of HIV infection but, by definition, are not AIDS indicator conditions. Examples include thrush, persistent vulvovaginal candidiasis, peripheral neuropathy, cervical dysplasia, recurrent herpes zoster infection, and idiopathic thrombocytopenic purpura.
- As the CD4 cell count drops below 200/μL, the frequency of opportunistic infections dramatically increases. AIDS is defined by the appearance of any indicator condition (Table 90-2) or a CD4 cell count of less than 200/μL.
- The average survival time following a diagnosis of AIDS has been estimated to be between 16 and 24 months.[3]
- Antiretroviral therapy and prophylaxis and treatment of opportunistic infections have been shown to delay the time to onset of complications and death in this group of patients.[4]
- Advanced HIV infection exists in patients with a CD4 cell count below 50/μL or clinical evidence of end-stage disease, including disseminated *Mycobacterium avium* complex or disseminated cytomegalovirus (CMV). Patients in this group have a median survival of 12 to 18 months.[3]

TABLE 90-2 AIDS-Defining Conditions

Esophageal candidiasis
Cryptococcosis
Cytomegalovirus retinitis
Herpes simplex virus
Kaposi's sarcoma
Brain lymphoma
Mycobacterium avium complex
Pnemocystis carinii pneumonia
Progressive multifocal leukoencephalopathy
Brain toxoplasmosis
HIV encephalopathy
HIV wasting syndrome
Disseminated histoplasmosis
Isosporiasis
Disseminated *Mycobacterium tuberculosis* disease
Recurrent *Salmonella* septicemia
CD4 count <200 cell/μL
Pulmonary tuberculosis
Recurrent bacterial pneumonia
Invasive cervical cancer

- **PULMONARY COMPLICATIONS** Pulmonary presentations are among the most common reasons for emergency department visits by HIV-infected patients.[5,6] Presenting complaints frequently are nonspecific and include cough, hemoptysis, shortness of breath, and chest pain.
- The most common causes of pulmonary abnormalities in HIV-infected patients include community-acquired bacterial pneumonia, *Pneumocystis carinii* pneumonia (PCP); infections due to *Mycobacterium tuberculosis* (MTB), CMV, *Cryptococcus neoformans,* and *Histoplasma capsulatum*; and neoplasms.
- Pulmonary radiographic findings are helpful in determining likely causes (Table 90-3).
- Approximately 70 percent of HIV-infected patients will acquire PCP at some time during their illness, and PCP is often the initial opportunistic infection that establishes the diagnosis of AIDS.
- This disease is the most frequent serious complication of HIV infection in the United States and the most common identifiable cause of death in patients with AIDS.
- The classic presenting symptoms of PCP are fever, cough (typically nonproductive), and shortness of

TABLE 90-3 Chest Radiographic Abnormalities: Differential Diagnosis in the AIDS Patient

FINDING	CAUSES
Diffuse interstitial infiltration	PCP CMV MTB MAI Histoplasmosis Coccidioidomycosis Lymphoid interstitial pneumonitis
Focal consolidation	Bacterial pneumonia *M. pneumoniae* PCP MTB MAI
Nodular lesions	Kaposi's sarcoma MTB MAI Fungal lesions Toxoplasmosis
Cavitary lesions	PCP MTB Bacterial infection Fungal infection
Adenopathy	Kaposi's sarcoma Lymphoma MTB Cryptococcosis

* ABBREVIATIONS: CMV = cytomegalovirus; MAI = *Mycobacterium avium intracellulare*; MTB = *Mycobacterium tuberculosis*; PCP = *Pneumocystis carinii* pneumonia.

breath (progressing from being present only with exertion to being present at rest).
- The incidence of tuberculosis (TB) in the AIDS population is estimated to be 200 to 500 times that in the general population.[7]
- Classic pulmonary manifestations of TB include cough with hemoptysis, night sweats, prolonged fevers, weight loss, and anorexia.
- Classic upper lobe involvement and cavitary lesions are less common, particularly among late-stage AIDS patients.[8] Negative purified protein derivative (PPD) TB test results are frequent among AIDS patients due to immunosuppression.
- Nonopportunistic bacterial pneumonias are the most common pulmonary infections in HIV-infected patients. Common pathogens include *Streptococcus pneumoniae, Haemophilus influenzae,* and *Staphylococcus aureus.* Productive cough, leukocytosis, and the presence of a focal infiltrate suggest bacterial pneumonia, especially in those with earlier-stage disease.

- **Neurologic Complications** Central nervous system (CNS) disease occurs in 90 percent of patients with AIDS, and 10 to 20 percent of HIV-infected patients initially present with CNS symptoms.
- AIDS dementia complex (also referred to as HIV encephalopathy or subacute encephalitis) is a progressive process commonly heralded by subtle impairment of recent memory and other cognitive deficits caused by direct HIV infection.
- Toxoplasmosis is the most common cause of focal encephalitis in patients with AIDS. Symptoms may include headache, fever, focal neurologic deficits, altered mental status, or seizures.
- On a noncontrast scan, toxoplasmosis typically appears as multiple subcortical lesions with a predilection for the basal ganglia.
- With contrast enhancement, toxoplasmosis lesions are ring-enhancing, with surrounding areas of edema.
- Cryptococcal CNS infection may be seen in up to 10 percent of AIDS patients and may cause either focal cerebral lesions or diffuse meningoencephalitis. The most common presenting signs are fever and headache, followed by nausea, altered mentation, and focal neurologic deficits.
- Other, less common CNS infections that should be considered in the presence of neurologic symptoms include bacterial meningitis, histoplasmosis (usually disseminated), CMV, progressive multifocal leukoencephalopathy, herpes simplex virus, neurosyphilis, and TB.

- **Gastrointestinal Complications** Approximately 50 percent of AIDS patients will present with gastrointestinal complaints at some time during their illness. The most frequent presenting symptoms include odynophagia, abdominal pain, bleeding, and diarrhea.
- Diarrhea is the most frequent gastrointestinal complaint and is estimated to occur in 50 to 90 percent of AIDS patients.
- Common causes include bacterial organisms such as *Shigella and Salmonella,* enteroadherent *Escherichia coli,* and *Campylobacter;* parasitic organisms such as *Giardia, Cryptosporidium,* and *Isospora belli;* CMV; *M. avium intracellulare;* and antibiotic therapy.[9]
- Oral candidiasis or thrush affects more than 80 percent of AIDS patients. The tongue and buccal mucosa are commonly involved; characteristically the plaques can easily be scraped from an erythematous base.
- Esophageal involvement may occur with *Candida,* herpes simplex, and CMV. Complaints of odynophagia or dysphagia are usually indicative of esophagitis and may be extremely debilitating.
- Hepatomegaly occurs in approximately 50 percent of AIDS patients. Elevation of alkaline phosphatase levels is frequently seen. Jaundice is rare. Coinfection with hepatitis B and C is common, especially among injected drug users.
- Anorectal disease is common in AIDS patients. Proctitis is characterized by painful defecation, rectal discharge, and tenesmus. Common causative organisms include *Neisseria gonorrhoeae, Chlamydia trachomatis, Treponema pallidum,* and herpes simplex virus.

- **Cutaneous Manifestations** Kaposi's sarcoma appears more often in homosexual men than in other risk groups. Clinically, it consists of painless, raised brown-black or purple papules and nodules that do not blanch. Common sites are the face, chest, genitals, and oral cavity; however, widespread dissemination involving internal organs may occur.
- Reactivation of varicella zoster virus is more common in patients with HIV infection and AIDS than in the general population.[10]

- **Constitutional Symptoms and Febrile Illnesses** Systemic symptoms—such as fever, weight loss, and malaise—are common in HIV-infected patients and account for the majority of HIV-related emergency department presentations.[11]
- Although fever may indicate any of a variety of

infections (including bacterial, fungal, viral, and protozoal pathogens), the most common causes in the HIV-infected population are HIV-related fever, non-Hodgkin's lymphoma, and systemic infections, such as *M. avium* complex, CMV, and infective endocarditis. Fever caused by HIV infection alone tends to occur in the afternoon or evening and is generally responsive to antipyretics.

- Disseminated *M. avium* complex is the most common opportunistic bacterial infection in AIDS patients, causing disseminated disease in up to 50 percent of patients at some time during their illness.
- Persistent fever and night sweats are typical. Associated symptoms include weight loss, diarrhea, malaise, and anorexia.
- CMV is the most common cause of serious opportunistic viral disease in HIV-infected patients. Disseminated disease commonly involves the gastrointestinal or pulmonary systems. The most important manifestation is retinitis.

- **Ophthalmologic Manifestations** Some 75 percent of patients with AIDS develop ocular complications.[12]
- CMV retinitis is the most frequent and serious ocular opportunistic infection and the leading cause of blindness in AIDS patients. The prevalence is estimated to be up to 40 percent.
- The presentation of CMV retinitis is variable. It may be asymptomatic early on but later causes changes in visual acuity, visual field cuts, photophobia, scotoma, or eye redness or pain.[13]

DIAGNOSIS AND DIFFERENTIAL

- The most common assay used to detect viral antibody is an enzyme-linked immunoassay (EIA) and a confirming Western blot test on EIA-positive specimens.
- EIA is approximately 99 percent specific and 98.5 percent sensitive; the Western blot test is nearly 100 percent sensitive and specific if performed under ideal laboratory circumstances.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The initial evaluation of HIV-infected and AIDS patients begins with a heightened awareness of the need for universal precautions. All blood and body fluid exposures should be considered infective. Respiratory isolation should be instituted for patients with suspected TB.
- All unstable patients should have airway management as indicated, oxygen, pulse oximetry, cardiac monitoring, and IV access. Shock, with its myriad causes, should be managed in standard fashion (see Chaps. 7 and 8).
- Seizures, altered mental status, gastrointestinal

TABLE 90-4 Treatment Recommendations for Common HIV-Related Infections

ORGAN SYSTEM	INFECTION	THERAPY
Systemic	*Mycobacterium avium*	Clarithromycin 500 mg PO bid plus ethambutol 15 mg/kg/d PO plus rifabutin 300 mg/kg/d PO
	CMV	Gancyclovir 5 mg/kg IV bid or foscarnet 60 mg/kg IV q8h
Pulmonary	PCP	TMP-SMX 15–20 TMP/kg/d and 75–100 mg SMX/kg/d PO or IV for 3 weeks or pentamidine 4 mg/kg/d IV or IM for 3 weeks; consider PO steroids for Pa_{O_2} <70 mmHg or A-a gradient >35
	MTB	Rifabutin 10 mg/kg/d PO plus pyrazinamide 15–30 mg/kg/d PO plus streptomycin 15 mg/kg/d IM
CNS	Toxoplasmosis	Pyrimethamine 50–100 mg/d PO plus sulfadiazine 4–8 mg/kg/d plus folinic acid 10 mg/d PO
	Cryptococcosis	Amphotericin B 0.7 mg/kg/d IV with or without flucystosine; maintenance therapy required
Ophthalmologic	CMV	Ganciclovir 5 mg/kg bid for 2 weeks; maintenance therapy required daily
Gastrointestinal	Candidiasis (thrush)	Clotrimazole 10 mg 5 times daily troches or nystatin 500 kU 5 times daily gargle
	Esophagitis	Fluconazole 100 mg/d PO
	Salmonellosis	Ciprofloxacin 500 mg bid PO for 2–4 weeks; maintenance therapy required
	Cryptosporidiosis	No known effective cure
Cutaneous	Herpes simplex	Acyclovir 1000 mg/d PO or acyclovir 5–10 mg/kg/d IV
	Herpes zoster	Acyclovir 4000 mg/d PO; IV therapy required for ocular involvement or dissemination
	Candida, Trichophyton	Cotrimazole, miconazole, or ketoconazole, topical therapy bid to tid for 3 weeks

Abbreviations: bid = two times per day; tid = three times per day; TMP-SMX = trimethoprim-sulfamethoxazole.

bleeding, and coma should be managed with standard protocols.
- Suspected bacterial sepsis and focal bacterial infections should be treated with standard antibiotics. Specific opportunistic infections should be treated with medications as listed in Table 90-4.
- The decision to admit an AIDS patient should be based on severity of illness with attention to the following: new presentation of fever of unknown origin, hypoxia worse than baseline or Pa_{O_2} below 60, suspected PCP, suspected TB, new CNS symptoms, intractable diarrhea, suspected CMV retinitis, herpes zoster ophthalmicus, or a patient incapable of self-care.

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91 TETANUS AND RABIES

David M. Cline

TETANUS

EPIDEMIOLOGY

- The current annual incidence of tetanus is 0.02 per 100,000 population.[1]
- In the United States, the majority of tetanus occurs in temperate areas, with the states of Texas, California, and Florida responsible for the greatest number of reported cases (34, 20, and 13, respectively).[2]
- Intravenous drug users, especially Hispanics in California, are at disproportionate risk of contracting the disease.[2]
- The majority of Americans over age 70 lack adequate immunity to tetanus.[3,4]

PATHOPHYSIOLOGY

- Tetanus is an acute, often fatal disease caused by wound contamination with *Clostridium tetani,* a motile, nonencapsulated anaerobic gram-positive rod.
- Any factor that lowers the local oxidation-reduction potential—such as the presence of crushed, devitalized tissue, a foreign body, or the development of suppuration—favors the development of the vegetative, toxin-producing form of *C. tetani.*[5]
- *Clostridium tetani* produces two exotoxins: tetanolysin, which appears to be clinically insignificant; and tetanospasmin, a potent neurotoxin that is responsible for all the clinical manifestations of tetanus.
- Tetanospasmin acts on the motor end plates of skeletal muscle, in the spinal cord, in the brain, and in the sympathetic nervous system.

CLINICAL FEATURES

- The clinical manifestations of tetanus are generalized muscular rigidity, violent muscular contractions, and instability of the autonomic nervous system.
- Wounds that become infected with toxin-producing *C. tetani* are most often puncture wounds[1] but vary in severity from deep lacerations to minor abrasions.[1,5]
- The incubation period of tetanus—that is, the period from initial inoculation to the onset of symptoms—can range from less than 24 h to longer than 1 month. The shorter the incubation period, the more severe the disease and the worse the prognosis for recovery.[6]
- Local tetanus is manifest by persistent rigidity of the muscles in close proximity to the site of injury and usually resolves after weeks to months without sequelae.
- Generalized tetanus is the most common form of the disease and frequently follows a puncture wound to the foot from a nail.[1]
- The most frequent presenting complaints of patients with generalized tetanus are pain and stiffness in the masseter muscles (lockjaw).[7] Nerves with short axons are affected initially; therefore, symptoms appear first in the facial muscles, with progression to the neck, trunk, and extremities.[7]
- Disturbances of the autonomic nervous system, generally a hypersympathetic state, occur during the second week of clinical tetanus and present as tachycardia, labile hypertension, profuse sweating, hyperpyrexia, and increased urinary excretion of catecholamines.[8]
- Cephalic tetanus follows injuries to the head or, occasionally, otitis media and results in dysfunction of the cranial nerves, most commonly the seventh.
- Neonatal tetanus occurs only if the mother is inadequately immunized. Most cases of neonatal tetanus arise from unsterile handling of the umbilical stump.[7]

DIAGNOSIS AND DIFFERENTIAL

- Tetanus is diagnosed solely on the basis of clinical evidence.
- Strychnine poisoning most closely mimics the clinical picture of generalized tetanus.
- Other diseases in the differential include dystonic reaction, hypocalcemic tetany, and rabies.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Human tetanus immune globulin (TIG) neutralizes circulating tetanospasmin and toxin in the wound but not toxin that is already fixed in the nervous system.
- Even though TIG does not ameliorate the clinical symptoms of tetanus, there is evidence that its administration significantly reduces mortality.[9]
- TIG should be administered intramuscularly opposite the site of tetanus toxoid administration. It should be given before wound debridement, since exotoxin may be released during wound manipulation.[10]
- Antibiotics, although of questionable utility in the treatment of tetanus, have traditionally been administered. Parenterally administered metronidazole should be considered the antibiotic of choice.[11] Penicillin, a centrally acting $GABA_A$ antagonist, may potentiate the effects of tetanospasmin; therefore, its use should be avoided.[6]
- The water-soluble agent midazolam is currently the preferred agent for producing muscle relaxation in patients with tetanus.[7]
- Succinylcholine is recommended for emergency airway control, while vecuronium is the neuromuscular blocking agent of choice for prolonged blockade because of its minimal cardiovascular side effects.[12]
- The combined alpha- and beta-adrenergic blocking agent labetalol has been successfully used to treat the manifestations of sympathetic hyperactivity in tetanus. However, several investigators have reported fatal cardiovascular complications in patients treated with beta-adrenergic blocking agents alone.[13,14]
- Magnesium sulfate inhibits the release of epinephrine and norepinephrine from the adrenal glands and adrenergic nerve terminals, eliminating the source of catecholamine excess in tetanus[15] and providing a rationale for its clinical use.[8]
- A summary of the guidelines for active tetanus immunization is presented in Table 91-1.[16–18]

RABIES

EPIDEMIOLOGY

- Rabies is primarily a disease of animals.[19]
- In 1996, a total of 49 states, the District of Columbia, and Puerto Rico reported 7124 cases of rabies in animals.[20] Wild animals accounted for almost 92 percent of the reported cases: raccoons (50.4

TABLE 91-1 Summary Guide to Tetanus Prophylaxis in Wound Management

	CLEAN, MINOR WOUNDS		ALL OTHER WOUNDS[a]	
HISTORY OF ADSORBED TETANUS TOXOID (DOSES)	Td[b] 0.5 mL IM	TIG, 250 U IM	Td[b] 0.5 mL IM	TIG, 250 U IM
Unknown or less than three	Yes[c]	No	Yes	Yes
Three or more[d]	No[e]	No	Yes[f]	No

[a] For example, wounds >6 h old, contaminated with soil, saliva, feces, or dirt; puncture or crush wounds; avulsions; wounds from missiles, burns, or frostbite.
[b] DPT for children <7 years of age (DT if pertussis vaccine is contraindicated); Td for persons >7 years of age.
[c] The primary immunization series should be completed. Three doses total are required, with the second dose given at least 4 weeks after the first and the third dose 6 months later.
[d] If only three doses of fluid toxoid have been received, then a fourth dose of *absorbed* toxoid should be given.
[e] Yes, if routine immunization schedule has lapsed in a child <7 years of age of if >10 years since last dose.
[f] Yes, if routine immunization schedule has lapsed in a child <7 years of age or if >5 years since last dose. Boosters more frequent than every 5 years may predispose to side effects.
ABBREVIATIONS: DTP = diphtheria-pertussis-tetanus; DT = diphtheria-tetanus toxoids; IM = intramuscular; Td = tetanus-diphtheria; TIG = tetanus immune globulin.
SOURCE: Adapted from the American College of Emergency Physicians,[16–18] with permission.

percent), skunks (23.2 percent), bats (10.4 percent), foxes (5.8 percent), and other wild animals including rodents and lagomorphs (2.1 percent). Rabid domestic animals included cats (3.7 percent), cattle (1.8 percent), dogs (1.6 percent), horses and mules (0.65 percent), sheep and goats (0.22 percent), and other animals such as ferrets (0.06 percent).

- Current epidemiologic patterns of rabies in the United States have been summarized as follows.[18] The annual reports of rabies in wildlife far exceed those of rabies in domestic animals; rabies variants in bats are associated with a disproportionate number of infections in humans (53 percent), although bats constitute only about 10 percent of all reported rabies cases in animals annually; most other cases of human rabies diagnosed in the United States are attributable to infections acquired in areas of enzootic canine rabies outside of the United States; most persons with a case of rabies that originated in the United States have no history of an animal bite.
- Bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, rabbits, hares, and other small rodents almost never require antirabies postexposure prophylaxis. However, this recommendation may change.

PATHOPHYSIOLOGY

- Once introduced, the initial infection and multiplication occur within local monocytes for the first 48 to 96 h.
- Subsequently, the virus spreads across the motor end plate and ascends and replicates along peripheral nervous axoplasm to the dorsal root ganglia, the spinal cord, and the central nervous system (CNS). Following CNS replication in the gray matter, the virus spreads outward by peripheral nerves to virtually all tissues and organ systems.

CLINICAL FEATURES

- The initial symptoms of human rabies are nonspecific and last 1 to 4 days: fever, malaise, headache, anorexia, nausea, sore throat, cough, and pain, and/or paresthesia at the bite site (80 percent).
- Subsequently, CNS involvement becomes apparent with restlessness and agitation, altered mental status, painful bulbar and peripheral muscular spasms, opisthotonos, and bulbar or focal motor paresis.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of rabies in the emergency department (ED) is clinical.
- A final diagnosis is made by postmortem analysis of brain tissue. Cerebrospinal fluid (CSF) and serum antibody titers should be sent to the lab. Elevated CSF protein and a mononuclear pleocytosis are also seen.
- The differential diagnosis includes viral or other infectious encephalitis, polio, tetanus, viral process, meningitis, brain abscess, septic cavernous sinus thrombosis, cholinergic poisoning, and the Landry-Guillain-Barré syndrome.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of rabies exposure consists of assessment of risk of rabies, public health and animal control notification, and if warranted, the administration of specific immunobiological products to protect against rabies.
- Local wound care includes debridement of devitalized tissue, if any; this is important in reducing the viral inoculum. Wounds of special concern should not be sutured, as this promotes rabies virus replication.[21]
- Minor bites by bats and awakening in a room with a bat have been associated with the development of rabies. For this reason, the Centers for Disease Control and Prevention (CDC) recommend rabies postexposure prophylaxis for all persons who have sustained a bite, scratch, or mucous membrane exposure to a bat unless the bat is available for testing and is negative for evidence of rabies.[22]
- The CDC recommends that a healthy dog, cat, or ferret that bites a person should be confined and observed for 10 days.[23]
- Human rabies immune globulin (HRIG) is administered only once at the outset of therapy. The dose is 20 IU/kg, with half the dose (based upon tissue volume constraints) infiltrated locally at the exposure site and the remainder administered intramuscularly.
- Human diploid cell vaccine (HDCV) for active immunization is available in two formulations of the same vaccine. The HDCV can be administered intramuscularly or intradermally. It is administered in five 1-mL doses on days 0, 3, 7, 14, and 28. The World Health Organization recommends a sixth dose on day 90, but this is not universally accepted.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 140, "Tetanus," by Donna L. Carden, and Chap. 141, "Rabies," by David J. Weber, David A. Wohl, and William A. Rutala.

92 MALARIA

Gregory S. Hall

- The growth in international travel has resulted in a recent increase in the number of cases of malaria seen in the United States; indeed, the worldwide incidence is also increasing. Malaria must be considered in any person with a history of travel to the tropics who presents with an unexplained febrile illness. The clinical symptoms are often nonspecific, so that a high index of clinical suspicion must be maintained to diagnose infection with *Plasmodium.*

EPIDEMIOLOGY

- Four species of the protozoan *Plasmodium—P. vivax, P. ovale, P. malariae,* and *P. falciparum*—infect humans via the bite of a carrier female anopheline mosquito.
- Malarial transmission is most prevalent in sub-Saharan Africa, large areas of Central and South America, the Caribbean (especially the Dominican Republic and Haiti), the Indian subcontinent, Southeast Asia, the Middle East, and Oceania (New Guinea, Solomon Islands, etc.).[1]
- More than half of all recent cases of malaria in the United States reported to the Centers for Disease Control and Prevention (CDC) in Atlanta (and the majority of *P. falciparum* cases) were acquired from travel to sub-Saharan Africa.[2]
- *Plasmodivm falciparum,* which is responsible for the highest mortality rate among malaria victims, has exhibited growing resistance to standard chloroquine therapy as well as newer drugs such as pyrimethamine/sulfadoxine (Fansidar).
- Chemotherapy-resistant *P. falciparum* is especially prevalent in Africa, tropical South America, Asia, and Oceania.[3]

PATHOPHYSIOLOGY

- Plasmodial sporozoites are injected into a host's bloodstream during the feeding of the female anopheline mosquito; they travel directly to the liver, where they invade hepatic parenchymal cells (exoerythrocytic stage). In the liver, the parasites undergo asexual reproduction, forming thousands of daughter merozoites, which—after an incubation period of 1 to several weeks—rupture their host hepatic cells and are released into the peripheral circulation.
- The merozoites then rapidly invade circulating erythrocytes, where they mature and take on various morphologic forms—early ring forms, trophozoites, and schizonts—which are masses of new merozoites (erythrocytic stage).
- Eventually the target red blood cell (RBC) lyses, releasing the merozoites to invade additional erythrocytes and continuing the infection. Such RBC lysis then often recurs at regular 2- to 3-day intervals, corresponding with the classic periodicity of symptoms. This cyclic feature may be absent in *P. falciparum* infection.
- With *P. vivax* or *P. ovale* infection, portions of the intrahepatic forms are not released, remain dormant for months, and can later activate, resulting in a clinical relapse.
- *Plasmodium* infection may also be acquired via transplacental transmission or infected blood during transfusion or by the sharing of IV needles among drug abusers.
- The classic febrile paroxysm of malaria results from hemolysis of infected RBCs and the resulting release of antigenic agents that activate macrophages and produce cytokines.
- Infected RBCs lose their flexibility and thus are prone to cause congestion and obstruction of the capillary microcirculation of various organs, resulting in sequestration of blood in the spleen and anoxic injury to the lungs, kidneys, brain, and other vital organs.
- Hemolysis is often high with *P. falciparum* infection because of its predilection for erythrocytes of all ages (while the other three *Plasmodium* species target young or old RBCs). The sequestration of RBCs accounts for the paucity of mature parasites sometimes seen on the peripheral blood smear in *P. falciparum* infection.
- Immunologic sequelae such as glomerulonephritis, nephrotic syndrome, thrombocytopenia, and polyclonal antibody stimulation may occur.

CLINICAL FEATURES

- The incubation period between infection and onset of clinical features ranges from 1 to 4 weeks, but partial chemoprophylaxis or incomplete immunity of the host can prolong the incubation period to months or even years.
- A recurring febrile paroxysm, the hallmark of malaria, occurs in conjunction with the typical 2- to 3-day cycle of RBC lysis by the merozoite forms.

- Most patients develop a nonspecific prodrome of malaise, myalgias, headache, low-grade fever, and chills[4]; in some cases there may be a prominence of chest pain, abdominal pain, nausea/emesis, diarrhea, or arthralgias, leading to misdiagnosis.
- Symptoms progress to cyclic episodes of high fever, severe rigors/chills, diaphoresis, orthostatic dizziness, and extreme weakness/prostration.
- Physical exam findings are nonspecific and may include high fever, tachycardia, tachypnea, pallor of skin or mucous membranes, prostration, and splenomegaly (common with all plasmodial forms).
- In *P. falciparum* infection, hepatomegaly, icterus, and peripheral edema often occur.
- Typical laboratory features include normochromic normocytic anemia, hemolysis, thrombocytopenia, and abnormal or low white blood cell (WBC) count. Hypoglycemia, hyponatremia, elevated blood urea nitrogen (BUN)/creatinine, elevated lactic dehydrogenase (LDH) and erythrocyte sedimentation rate (ESR), and mildly elevated liver function tests (LFTs) may be seen.
- Complications can occur rapidly and may include splenic rupture, glomerulonephritis (especially with *P. malariae*), cerebral malaria (somnolence, coma, delirium, seizures; mortality reaches 20 percent), noncardiogenic pulmonary edema, and metabolic derangements including lactic acidosis and severe hypoglycemia (the last two occur most often with *P. falciparum*).[5]
- ''Blackwater fever'' is a severe renal complication seen almost exclusively with *P. falciparum* infections; it presents with massive intravascular hemolysis, jaundice, hemoglobinemia, hemoglobinuria (black urine), and acute renal failure.

DIAGNOSIS AND DIFFERENTIAL

- A definitive diagnosis is achieved by identifying the plasmodial parasite within RBCs on Giemsa-stained thin and thick smears of peripheral blood.[2]
- In early infections, particularly with *P. falciparum,* initial attempts to detect the parasite on peripheral blood smears may prove unsuccessful; parasite load in the peripheral circulation varies over time and is highest during the clinical episodes of high fever and chills. Failure to detect the organism on initial smears is *not* an indication to withhold treatment if malaria is suspected.
- If the initial peripheral smear is negative, repeated smears should be examined at least twice daily for 3 days to fully exclude malaria as the diagnosis.
- Of paramount importance is the determination of which species of *Plasmodium* are present in the blood, since patients with *P. falciparum* should be hospitalized for treatment. (Mixed infections with multiple species of *Plasmodium* are uncommon—<1 percent of cases.)
- The differential diagnosis includes influenza, hepatitis, viral syndromes, and a wide variety of other infections.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The drug of choice for treatment of infection caused by *P. vivax, P. ovale,* and *P. malariae* is chloroquine (See Table 92-1).
- Chloroquine has no effect on dormant hepatic forms of *P. vivax* and *P. ovale;* thus additional treatment with primaquine is required to prevent relapse. (Primaquine must be avoided in patients with glucose 6-phosphate dehydrogenase deficiency due to the possibility of inducing hemolysis.)
- Indications for hospital admission include confirmed or suspected *P. falciparum* infection, parasitemia of >3 percent on peripheral smear, significant hemolysis, severe/chronic comorbid conditions that may be aggravated by high fever or hemolysis, infants and pregnant women, elderly patients, and those with apparent complications such as renal failure, cerebral malaria, pulmonary edema, lactic acidosis, hypoglycemia, etc.[6]
- Many patients can be managed adequately in the outpatient setting provided that adequate home care and close follow-up with repeated blood smears to measure treatment response are available.
- Unless the possibility of chloroquine resistance can be absolutely excluded based on geographic exposure history, it is best to assume the infection to be resistant and treat with a combination of quinine and doxycycline with or without pyrimethamine-sulfadoxine.
- Patients with high levels of parasitemia, complications of *P. falciparum,* or who are unable to tolerate oral medication should be treated with intravenous therapy—quinidine is the IV drug of choice. (Caution: both quinidine and quinine can cause severe hypoglycemia and myocardial depression—cardiac monitoring is required during administration.)

TABLE 92-1 Treatment Regimens for Malaria

CLINICAL SETTING	DRUG	DOSAGE GUIDELINES	
		ADULTS	CHILDREN
Uncomplicated infection with *Plasmodium vivax, P. ovale, P. malariae,* and chloroquine-sensitive *P. falciparum*	Chloroquine phosphate	1 g load (600 mg base), then 500 mg (300 mg base) in 6 h, then 500 mg (300 mg base) per day for 2 d (total dose 2.5 g)	10 mg/kg base to maximum of 600 mg load, then 5 mg/kg base in 6 h and 5 mg/kg base per day for 2 d
	plus		
	primaquine phosphate*	26.3 mg load (15 mg base) per day for 14 d upon completion of chloroquine therapy	0.3 mg/kg base for 14 d upon completion of chloroquine therapy
Uncomplicated infection with chloroquine-resistant *P. falciparum*	(a) Quinine sulfate	600–650 mg PO tid for 5–7 d	8.3 mg/kg PO tid for 5–7 d†
	plus		
	doxycycline	100 mg PO bid for 7 d	Contraindicated in children <8 years of age
	plus/minus		
	pyrimethamine-sulfadoxine‡	3 tablets (75 mg/1500 mg) PO single dose	Over 2 months old: >50 kg 3 tablets 30–50 kg 2 tablets 15–29 kg 1 tablet 10–14 kg ½ tablet 4–9 kg ¼ tablet
	OR		
	(b) Mefloquine	1250 mg PO single dose	1 tablet/10 kg PO single dose§
	plus		
	doxycycline¶	See above	See above
	or		
	Halofantrine#	500 mg 6 h apart for 3 doses (repeat again in 1 week)	8 mg/kg salt PO q6h for 3 doses (repeat again in 1 week)
Complicated infection with chloroquine-resistant *P. falciparum*	Quinidine gluconate	10 mg/kg load over 2 h, then 0.02 (mg/kg)/min continuous infusion until patient is stabilized and able to tolerate PO therapy (see above)	Same as adults**
	plus		
	doxycycline	100 mg IV q12h until tolerating PO therapy (see above)	Contraindicated in children <8 years of age

* Terminal treatment for *P. vivax* and *P. ovale* only.
† If unable to administer with doxycycline due to patient's age, extend treatment to full 10 d.
‡ Optional; of unlikely value if acquisition in area with pyrimethamine-sulfadoxine resistance.
§ Not formally approved yet by Food and Drug Administration in this setting.
¶ Optional; many experts feel comfortable with mefloquine alone.
# Halofantrine is not commercially available in the United States (contact SmithKline Beecham at 1-800-366-8900). It is becoming the drug of choice for self-treatment of presumptive malaria in Thai-Cambodian and Myanmar borders *if* access to medical care is not available. In these areas, treatment may need to be extended to 3 d instead of 1 d.
** Consult an expert in pediatric infectious disease immediately for guidance.
ABBREVIATIONS: bid = twice a day; IV = intravenous; PO = oral; q = every; tid = three times a day.

- Exchange transfusions have been lifesaving for some patients—those with >10 percent parasite load, pulmonary edema, cerebral malaria, or renal complications.
- Treatment with glucocorticoids for cerebral malaria has not been shown to be beneficial.[7]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 142, ''Malaria,'' by Jeffrey D. Band.

93 COMMON PARASITIC INFECTIONS

Joel L. Goldberg

CLINICAL FEATURES

- Parasitic diseases are rare in the United States. Generally, they are associated with international travelers, immigrants, and outdoor enthusiasts. Immunosuppressed individuals are also at risk for contracting some rare parasitic diseases.
- Most can be diagnosed by testing stool for ova and parasites. *Ascaris lumbricoides, Necator americanus, Ancylostoma duodenale,* and *Strongyloides stercoralis* larvae can be seen in sputum.
- Most helminth infections cause eosinophilia.
- See Table 93-1 for common symptoms.
- See Chap. 92 for discussion of malaria.

HELMINTHS

INTESTINAL NEMATODES

- Treat infections with mebendazole, albendazole, or pyrantel pamoate unless otherwise noted.

- ***ENTEROBIUS VERMICULARIS* (PINWORM)** Infection is caused by egg ingestion.
- Adult worms are very small (2 to 5 mm) and reside in the rectum.
- Adults lay eggs around the rectum, causing intense pruritus.
- Organisms can often be seen by direct examination of anus. The ''Scotch-tape test'' can be used to collect and observe eggs by microscopy.
- Infections are spread easily by close contact. Immediate family members should be treated.

- ***ASCARIS LUMBRICOIDES*** Infection is caused by egg ingestion.
- Adult worms are 25 to 35 cm in length and reside in the small bowel. Eggs are passed via feces.
- Chief symptoms can include pneumonitis caused by larval lung migration and intestinal obstruction caused by large adult parasitic loads.
- Large parasite burdens can cause intestinal obstruction.
- Visceral larva migrans is a related infection caused by the ingestion of eggs of related species that infect animals. Larvae hatch and encyst in muscle, causing chronic eosinophilia, hepatomegaly, or chronic nonspecific pulmonary disease.

- ***NECATOR AMERICANUS*, *ANCYLOSTOMA DUODENALE* (HOOKWORM)** Infection is acquired by larval migration through the skin (e.g., bare feet).

TABLE 93-1 Common Symptoms of Parasitic Disease

SYMPTOM	POSSIBLE CAUSE
Urticaria	*Ascaris, Strongyloides, Dracunculus, Trichinella, Fasciola*
Diarrhea	Hookworm, *Strongyloides, Trichuris, Trichinella Schistosoma, Fasciola, Fasciolopsis, Taenia, Hymenolepis, Entamoeba, Giardia, Dientamoeba, Balantidium, Leishmania donovani*
Abdominal pain	*Ascaris,* hookwarm, *Trichuris, Schistosoma, Entamoeba, Clonorchis, Fasciola, Taenia, Hymenolepis, Diphyllobothrium, Giardia*
Pruritus	*Enterobius, Trichuris,* filariae (*Onchocerca volvulus*), *Dientamoeba, Leishmania*
Nausea and vomiting	*Ascaris, Trichuris, Trichinella, Taenia, Entamoeba, Giardia, Leishmania*
Skin ulcers	*Dracunculus,* hookworm (*Ancylostoma duodenale*), *L. donovani, Trypanosoma*
Splenomegaly	*Babesia, Toxoplasma, Plasmodium* species
Intestinal obstruction	*Ascaris, Strongyloides,* fluke (*Fasciolopsis buski*), *Taenia, Diphyllobothrium*
Eosinophilia	*Strongyloides,* hookworm, *Trichuris, Dracunculus, Fasciola, Toxocara, Ascaris, Trichinella,* filariae (*W. bancrofti, B. malayi*), *Hymenolepsis. Schistosoma,* fluke (*P. westermani, C. sinensis, Fasciolopsis buski*), *Taenia*
Fever	*Ascaris, Toxocara,* hookworm, *Trichuris, Trichinella,* filariae (*W. bancrofti*), *Schistosoma* fluke (*C. sinensis*), *Fasciola, Entamoeba, Giardia, Trypanosoma, L. donovani, Babesia, Plasmodium* species
Hepatomegaly	*Trypanosoma L. donovani, Toxocara, Schistosoma* fluke (*C. sinensis, O. viverrini, Fasciola*), tapeworm (*Echinococcus*), *Plasmodium* species

- Obligate larval lung migration (pneumonitis) occurs before the organism matures on the intestinal mucosa, often causing anemia with large parasite loads.
- Cutaneous larva migrans is a related infection acquired when free-living animal (e.g., dog) hookworm larvae penetrate the skin and cause pruritus and rash.

- ***Strongyloides stercoralis*** Infection can be acquired through the ingestion of eggs or through larval skin penetration, often causing localized dermatitis and pneumonitis from migration through lung parenchyma.
- Infections can be quite large secondary to autoinfection, especially in immunosuppressed individuals.

- ***Trichuris trichiura*** **(Whipworm)** Adults (3 to 5 cm long), which reside in the rectum, are acquired by ingestion of eggs.
- Large infections can cause tenesmus, leading to rectal prolapse.

- ***Trichinella spiralis*** Infection occurs by ingestion of larvae encysted in pork, bear, or walrus meat. Larvae mature and reproduce on the intestinal mucosa. New larvae penetrate the mucosa and encyst in host striated muscle.
- Symptoms depend on parasite load and can include fever, periorbital edema, myalgia, and central nervous system (CNS) manifestations.
- Diagnoses are made by muscle biopsy or serologic testing. Treatment is largely symptomatic. Steroids may be of benefit.

BLOOD AND TISSUE NEMATODES—FILARIAE

- All are transmitted by an arthropod vector (usually fly or mosquito).
- The larval stages are found in the cutaneous body tissues or bloodstream and are microscopic.
- Most are treated by diethylcarbamazine or ivermectin.

- ***Wuchereria bancrofti, Brugia malayi*** Adult forms mature in lymph nodes (2 to 4 cm long) and cause lymphangitis, lymphadenitis, and lymphedema. This disease is known as elephantiasis.

- ***Loa Loa*** **(African Eye Worm)** Larvae are often seen migrating across conjunctivae.

- ***Onchocerca volvulus*** Larval migration occurs through ocular tissues and often results in blindness, and is known as river blindness.

- ***Dracunculus medinensis*** **(Fireworm)** Infection due to this tissue nematode is acquired through ingestion of copepods infected with larvae, usually by drinking contaminated water.
- Adults (up to 1 M in length) are found in the lower extremities, often with a small portion of the worm extruding through the skin so eggs can be passed into the environment.
- Treatment is by surgical removal or slowly winding the adult worm around a stick over a period of several days.

TREMATODES (FLUKES)

- Most forms are treated with praziquantel.
- Usually, with rare exceptions, eggs are passed in feces.

- ***Fasciolopsis buski*** **(Intestinal Fluke)** Infection is acquired by ingestion of metacercariae (larval form) on water chestnuts and bamboo shoots.
- Infection produces malabsorptive diarrhea.

- ***Clonorchis sinensis, Fasciola hepatica*** **(Liver Flukes)** *Clonorchis* infection is caused by ingestion of fish containing encysted metacercariae.
- *Fasciola* infection is acquired through ingestion of metacercariae on watercress.
- Both can cause hepatic symptomatology secondary to inflammation, biliary obstruction, or portal cirrhosis.
- Infection is associated with hepatocellular carcinoma.

- ***Paragonimus westermani*** **(Lung Fluke)** Infection is acquired through the ingestion of metacercariae encysted in crab.
- Adults are encapsulated in cystic structures adjacent to bronchi.
- Eggs may be seen in sputum or feces.

- ***Schistosoma mansoni, S. japonicum, S. haematobium*** **(Blood Flukes)** All have snails as intermediate hosts.
- Cercariae (larval form) are free-living in fresh water (where endemic) and directly penetrate the skin.
- Pathology is caused by inflammation induced by eggs.

- Adults of *S. mansoni* and *S. japonicum* reside in mesenteric veins. Eggs can cause hepatic cirrhosis and are usually passed in the stool.
- Adults of *S. haematobium* reside in the vesical, prostatic, and uterine plexuses. Eggs may be found in urine.

- ***Schistosomal dermatitis*** Caused by transient skin penetration of cercariae of other animal (e.g. birds).
- This disease is known as swimmer's itch.
- Symptoms are self-limited, usually requiring no treatment.

CESTODES (TAPEWORMS)

- Most are benign, and infections are caused by ingestion of encysted larvae [*Taenia saginata* (beef tapeworm), *Hymenolepis nana,* and *Dipylidium caninum*].
- Adult worms mature in the small bowel, and proglottids containing eggs are passed in the feces.
- Serious infections are caused by egg ingestion of certain species, leading to the development of cysts that can be life-threatening. These include *Taenia solium* (pork tapeworm), *Echinococcus granulosis, E. multilocularis,* and *E. vogeli.*
- *Taenia solium* is associated with cysticercosis and is often responsible for new-onset seizures in Mexican immigrants or travelers to Mexico.
- Infections are treated with praziquantel.
- Hydatid disease is caused by multilocular cysts of the *Echinococcus* genera and can be treated by albendazole and surgical removal.
- *Diphyllobothrium latum* infection by the adult tapeworm can cause pernicious anemia and is acquired through ingestion of larvae encysted in fish.

PROTOZOA

AMEBAS

- ***Entamoeba histolytica*** Infection is often responsible for dysentery. The infection is acquired through ingestion of cysts that are passed in stool. Liver abscesses can also be formed.
- Symptoms include diarrhea, cramps, vomiting, and malaise.
- Infections are treated with metronidazole.

- ***Naegleria fowleri*** Infection causes amebic meningoencephalitis.
- Infection is acquired when free-living forms penetrate nasal passages.
- This disease has been associated with swimming pools and hot springs.
- The infection is usually diagnosed at autopsy because it is rapidly fatal.

- ***Giardia lamblia*** The infection is usually acquired through ingestion of cysts found in contaminated water and is often seen in hikers and campers.
- Symptoms include diarrhea, abdominal distention, and flatus.
- The infection is treated with metronidazole.

- ***Trypanosoma cruzi*** The parasite is transmitted by the reduviid (kissing) bug, and is known as Chagas' disease.
- The parasite often infects soft tissues, leading to cardiomyopathy, megaesophagus, and megacolon.
- Infection is diagnosed by blood smear or xenodiagnosis.
- Ketoconazole may be an effective treatment.

- ***Leishmania* (Visceral and Cutaneous Leishmaniasis)** The parasite is transmitted by sandflies (*Phlebotomus* spp.).
- Hepatosplenomegaly is indicative of an infection known as kala-azar.
- The infection is treated by applying antimonial compounds topically or injecting them intravenously.

- ***Cryptosporidium parvum*** The parasite is usually found in contaminated water or in poorly treated urban water supplies.
- Infection can cause a self-limited diarrheal illness in healthy individuals but can also be life-threatening in the immunocompromised host.
- The treatment is supportive.

- ***Pneumocystis carinii*** Infection can cause severe pneumonia in the immunocompromised host.
- The infection is treated with trimethoprim-sulfamethoxazole and steroids.

- ***Toxoplasma gondii*** Domestic cats are reservoirs for infection.
- The parasite can be transmitted to the fetus transplacentally if mother has never been exposed to *T. gondii* before.
- Pregnant women should avoid contact with cats (most common domestic source).
- Infection is also a problem in the immunocompromised host and often presents with CNS mani-

festations such as confusion, seizures or encephalitis.
- Treatment with pyrimethamine and sulfonamides may be beneficial.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 143, ''Common Parasitic Infections,'' by Harold H. Osborn.

94 INFECTIONS FROM ANIMALS

Gregory S. Hall

- Zoonoses, or diseases transmitted from animal and arthropod vectors to humans, remain common and often underestimated in prevalence in North America. Contact with household pets (or their associated parasites), domesticated or wild animals and their infected tissues or secretions, and arthropods, especially ticks, are all sources of infections in humans.[1,2]
- Most zoonoses in the United States, including those spread by ticks, have their highest incidence in the spring and summer.[3] These diseases are easily mistaken for other nonspecific self-limited diseases, and many patients at risk fail to volunteer their exposure history (i.e., they cannot recall a tick bite).[4] This chapter focuses primarily on tick-borne infections and a few other entities. For information on rabies refer to Chap. 91.

LYME DISEASE

- This remains the leading vector-borne zoonosis in the United States. It is most prevalent in the Northeast but has been reported in all 48 continental states.[5]
- *Borrelia burgdorferi,* a spirochete, is the responsible organism and is transmitted to humans by *Ixodes* species ticks, with rabbits, rodents, and deer serving as host reservoir animals.
- Lyme disease is a multiorgan infection divided into three distinct stages, but not all patients suffer all stages, stages may overlap, and remissions between stages may occur.
- Erythema chronicum migrans (ECM), a skin lesion, is the hallmark of stage I. It occurs in 60 to 80 percent of cases and consists of an annular, erythematous skin plaque with central clearing that forms at the inoculation site 2 to 20 days after a tick bite. The primary pathophysiology of ECM is that of a vasculitis. ECM occurs in only 60 to 80 percent of cases.[6]
- Stage I (ECM lesion) may be accompanied by (in decreasing order of frequency) generalized malaise and fatigue, headache, fever, chills, stiff neck, arthralgias, and other constitutional symptoms—all of which, if left untreated, resolve spontaneously in 3 to 4 weeks.[4,7]
- Stage II corresponds to dissemination of the spirochete, resulting in multiple secondary annular skin lesions (ECM), fever, adenopathy, splenomegaly, and flulike constitutional symptoms.
- Some 10 percent of stage II patients develop neurologic disease—most often cranial neuritis (especially uni- or bilateral facial nerve palsy) or other peripheral neuropathies. Also, asymmetric oligoarticular arthritis (usually large joints, especially the knees) may develop. Occasionally first-, second-, or third-degree AV nodal heart block may develop.
- Stage III represents chronic persistent infection. It occurs years after the resolution of stage I and includes chronic intermittent migratory arthritis, myocarditis, encephalopathy, and axonal polyneuropathy.[8]
- Diagnosis is dependent initially on clinical features; a two step serologic test (enzyme immunoassay and Western blot) is used for confirmation. Culture of the organism is difficult and not widely available.
- Lyme disease responds well to antimicrobial therapy, especially if started early in the course of the infection. The treatment of choice for early Lyme disease is oral doxycycline 100 mg PO bid for 10 to 21 days. (Acceptable alternatives include

amoxacillin, cefuroxime, azithromycin, clarithromycin, ceftriaxone, or cefotaxime.)[5,9]
- Serious central nervous system (CNS) disease (meningitis, encephalitis, neuropathy), cardiac manifestations, or severe arthritis warrants hospital admission for supportive care and a 14- to 21-day course of IV ceftriaxone.
- Prophylactic treatment for tick bites is not generally recommended.

ROCKY MOUNTAIN SPOTTED FEVER

- Rocky Mountain spotted fever (RMSF) is caused by *Rickettsia rickettsii,* an obligate intracellular coccobacillus, carried by *Dermacentor* species. Ticks on deer, rodents, horses, cattle, cats, and dogs are the usual vectors.
- Transmission of RMSF to humans via tick bite occurs primarily (in 95 percent of cases) between April 1 and September 30, with the highest incidence in the mid-Atlantic states (cases have been reported in most continental states in the United States); two-thirds of all cases are reported in children <15 years old.
- RMSF is classically defined by a triad of fever, rash, and history of tick exposure, but only about 50 percent of afflicted patients can recall a tick bite, and rash may be absent in up to 17 percent (''spotless RMSF''—usually seen in African Americans, the elderly, and in severe or fatal cases).[10,11]
- The incubation period following a tick bite is usually 4 to 10 days and is followed by the abrupt or insidious onset of nonspecific symptoms such as fever, malaise, severe headache, myalgias, nausea/vomiting, diarrhea, anorexia, abdominal pain, and photophobia.
- Additional signs of symptoms may include lymphadenopathy, hepatosplenomegaly, conjunctivitis, confusion, meningismus, renal or respiratory failure, and myocarditis.
- Rash, the hallmark feature, usually begins during the first 2 weeks of illness. It is often maculopapular and typically begins on the extremities around the wrists and ankles (it often involves the palms/soles), and spreads centripetally to the trunk, usually sparing the face (it may become petechial and/or purpuric and rarely necrotic).
- Gastrointestinal symptoms, often prominent features, may precede the onset of rash and often lead to misdiagnosis of gastroenteritis or even acute abdomen.
- RMSF pneumonitis, a common and potentially fatal complication, presents with cough, dyspnea, pulmonary edema, and systemic hypoxia.[12]
- Serious neurologic involvement occurs in about one-quarter of cases, with confusion, stupor, ataxia, seizures, and coma.
- Untreated patients suffer up to 25 percent mortality. The clinical diagnosis must be presumed in to order to start early therapy, since serology to confirm a rise in antibody titer is not reliably positive until 6 to 10 days after onset of symptoms (diagnosis may also be confirmed by skin rash biopsy with immunofluorescent testing).[13]
- The differential diagnosis includes viral illness (measles, rubella, hepatitis, mononucleosis, encephalitis, enteroviral exanthem), gastroenteritis, acute abdomen, disseminated gonorrhea, meningitis (meningococcus), secondary syphilis, leptospirosis, typhoid fever, pneumonia, and streptococcal infection.
- Therapy for adults includes doxycycline 100 mg PO bid, tetracycline 500 mg PO qid, or chloramphenicol 50 to 75 mg/kg/d IV in four divided doses.[14]
- Therapy for children <45 kg (100 lb) includes doxycycline 4.4 mg/kg/d PO in two divided doses on day 1 followed by 2.2 mg/kg/d PO in two divided doses thereafter. Alternatives include tetracycline and IV chloramphenicol.
- Doxycycline has been used for short-course therapy in children without significant staining of teeth, but cosmetic risks must be balanced against the potentially serious side effects of chloramphenicol. The risk/benefits of either treatment should be discussed with the parents and the child's pediatrician, if possible, and informed consent should be obtained.
- Antimicrobial therapy for RMSF is given for 5 to 7 days or until the patient is afebrile and clinically improving for at least 48 h.
- Patients with nausea/vomiting or significant systemic disease should be admitted to the hospital for supportive care and IV antimicrobial therapy.

TICK PARALYSIS

- Tick paralysis, a relatively uncommon entity, may be fatal (aspiration or respiratory failure) if undiagnosed, yet it is easily cured by careful removal of the offending tick.
- This rare complication has been reported following bites of the dog tick (*Dermacentor variabilis*) and wood tick (*D. andersoni*) in the United States, with incidence highest in spring to late summer and children most commonly affected.

- Symptoms are believed to result from a neurologic venom secreted from the salivary glands of the female tick, which results in conduction blockade at the motor end plates of peripheral nerves.
- Clinical symptoms usually begin 4 to 7 days after attachment of the female tick, with an initial prodrome of malaise, irritability, restlessness, and paresthesias of hand or foot. Fever is usually absent.
- Symptoms progress to include a symmetric ascending flaccid paralysis (resembling Guillain-Barré syndrome), with eventual loss of deep tendon reflexes, dysphagia, involuntary eye movements, cranial nerve plasies, ataxia, and respiratory paralysis. (Sensation remains intact.)
- Diagnosis depends on locating the tick (often hidden in the scalp, under hair). The cerebrospinal fluid (CSF) remains normal.
- Prompt and careful removal of the tick along with supportive care (mechanical ventilation if needed) is curative. Most patients begin to recover within hours of tick removal, with complete recovery usually within 48 to 72 h.

TULAREMIA

- Tularemia (rabbit skinner's disease) is an infection caused by *Francisella tularensis,* a small gram-negative coccobacillus carried by *Dermacentor,* the *Amblyomma* species of ticks, and the deerfly. Principal animal host reservoirs include rabbits, hares, deer, muskrats, beaver, and dogs.[12]
- Tularemia has been widely reported in the continental United States but the highest incidence is in Arkansas, Missouri, and Oklahoma, with cases reported year round, but most cases appear in early winter (adults) and early summer (children).
- Transmission may occur via arthropod bite; animal bite; inoculation of skin, conjunctiva, or oral mucosa by blood or tissue from an infected animal host; and handling or ingestion of contaminated soil, grain, hay, or water. Several distinct clinical syndromes can occur, with clinical features that depend on the route of inoculation.
- The average incubation period following exposure is 3 to 5 days, after which there is a sudden onset of fever (which may persist for several days, remit briefly, then recur), chills, headache, anorexia, malaise, and fatigue. Additional symptoms that may occur include myalgias, cough, vomiting, abdominal pain, diarrhea, and pharyngitis.
- Ulceroglandular fever (the most common presentation) follows a tick or animal bite—a papule develops at the bite site and evolves into a tender necrotic ulcer with painful regional adenopathy. Glandular tularemia consists of tender regional adenopathy without a skin lesion.
- Other forms include oculoglandular tularemia—painful conjunctivitis with periauricular, submandibular, and cervical adenopathy; pharyngeal tularemia (ingestion of contaminated food/water)—exudative pharyngitis/tonsillitis; and tularemic pneumonitis (inhalation of organism)—productive cough, pleuritic chest pain, rales, consolidation, and pleuritic rub.
- Typhoidal tularemia (any form of transmission) includes multiorgan signs and symptoms—fever, headache, vomiting, diarrhea, myalgias, hepatosplenomegaly, cough, and pneumonitis.
- Clinical diagnosis rests on suggestive clinical features. Serologic [enzyme-linked immunosorbent assay (ELISA)] studies to determine acute and convalescent titers or culture of organism from blood, ulcers, lymph nodes, or sputum may be used to confirm the diagnosis. Other laboratory findings are nonspecific.
- Differential diagnosis includes pyogenic bacterial infection, syphilis, anthrax, plague, Q fever, psittacosis, typhoid, brucellosis, and rickettsial infection.
- Treatment is with streptomycin 7.5 to 10 mg/kg q12 h IM or IV (pediatric dose 30 to 40 mg/kg IM in two divided doses) or gentamicin 3 to 5 mg/kg/day IV in three divided doses. Inpatient therapy is given for 7 to 14 days.[15–17]

EHRLICHIOSIS

- A zoonotic disease with two clinical subtypes (human granulocytic and human monocytic) caused by *Ehrlichia* species, a small gram-negative coccobacillus that infects circulating leukocytes. The human monocytic form (*Ehrlichia chaffeensis*) predominates in the United States.[18]
- Transmission occurs via bite or exposure to ticks of the *Ixodes* and *Amblyomma* species. Animal host reservoirs include deer, dogs, and other mammals.
- The incubation period ranges from 1 to 21 days (median, 7 days) followed by onset of nonspecific symptoms such as high fever, headache, nausea/vomiting, malaise, abdominal pain, anorexia, and myalgias.
- In a minority of cases, a maculopapular or petechial rash (which may involve palms/soles) develops.
- Serious complications include renal or respiratory

failure, disseminated intravascular coagulopathy, cardiomegaly, and encephalitis.
- The diagnosis must rest on clinical features, but serology (antibody titers) can provide confirmation. Laboratory findings (most prominent on the fifth through seventh days of illness) include leukopenia, absolute lymphopenia, thrombocytopenia, and elevated serum transaminase and alkaline phosphatase levels (rarely, CSF pleocytosis is seen).
- Differential diagnosis includes rickettsial diseases (especially RMSF) and bacterial meningitis.
- The treatment of choice is doxycycline 100 mg PO or IV bid for 7 to 14 days. (Alternatives include tetracycline and chloramphenicol. There are no current recommendations for children or pregnancy.[18])

COLORADO TICK FEVER

- An acute viral illness caused by an RNA virus of the *Coltivirus* species, this infection is transmitted to humans via *Dermacentor* species ticks (animal reservoir hosts include deer, marmots, and porcupines), with most cases reported between late May and early July in the mountainous western regions of the United States.
- Symptoms begin suddenly 3 to 6 days following tick bite; they include fever, chills, severe headache, photophobia, nausea/vomiting, and myalgias. Lymphadenopathy, hepatosplenomegaly, and conjunctivitis may also be seen.
- Symptoms usually persist for 5 to 8 days and then spontaneously remit, but 3 days later up to 50 percent of patients develop a second phase that includes a transient generalized maculopapular or petechial rash. The secondary phase usually lasts for 2 to 4 days and resolves spontaneously.
- Diagnosis rests on clinical features but can be confirmed by fluorescent antibody staining of a patient's erythrocytes or mouse inoculation.[19]
- Differential diagnosis includes meningitis (bacterial or viral) and rickettsial infections (especially RMSF).
- Treatment consists mainly of supportive care. However, empiric treatment with antimicrobial therapy to cover bacterial meningitis and rickettsial infection is often used pending confirmation of the diagnosis.

HANTAVIRUS

- This viral zoonosis was identified in 1977. In North America, the etiologic agent is the *sin nombre* virus (member of Bunyaviridae family). To date at least 10 distinct serotypes have been identified, each with a specific rodent vector, geographic distribution, and clinical manifestation.[20]
- In the United States, the deer mouse is the primary vector, with transmission to humans accomplished via inhalation of dried particulate feces, contact with urine, or rodent bite.[21]
- Worldwide the majority of Hantavirus serotypes have a predilection for the kidney, with a clinical presentation of acute renal failure, thrombocytopenia, ocular abnormalities, and flulike symptoms.
- In the United States, the most common presentation is that of Hantavirus pulmonary syndrome—an initial flulike prodrome for 3 to 4 days followed by pulmonary edema, hypoxia, hypotension, tachycardia, dizziness, nausea/vomiting, thrombocytopenia, and metabolic acidosis. Cough is generally absent.[20–22]
- Diagnosis rests on clinical features plus history of exposure but may be confirmed by an immunofluorescent or immunoblot assay.[2] Differential diagnosis includes bacterial pneumonia, adult respiratory distress syndrome, and influenza.
- The Hantavirus pulmonary syndrome has a reported mortality rate of 50 to 70 percent. Treatment consists primarily of supportive care (especially oxygenation/ventilation) and possibly inhaled ribavirin.[20–22]

ANTHRAX

- This acute bacterial infection is caused by *Bacillus anthracis,* an aerobic gram-positive rod that forms central oval spores. Although it is very rare in North America, anthrax remains of concern partly because of its potential use as an agent of biological warfare or terrorism.
- In nature, the disease is most commonly seen in domestic livestock (cattle, sheep, horses, and goats) and wild herbivores. Human infection can result from inhalation of spores, inoculation of broken skin, arthropod bite (fleas), or ingestion of inadequately cooked infected meat.
- Symptoms depend on method of transmission. Inhaled or pneumonic anthrax is contracted via handling of unsterilized, imported animal hides or raw wool. Initially patients suffer a flulike illness that progresses over 3 to 4 days to include marked mediastinal and hilar edema (mediastinitis rather than true pneumonia) and respiratory failure. This condition is universally fatal.
- Cutaneous anthrax begins with a small red macule at the site of inoculation, which, over the course of a week, progresses through papular, vesicular,

or pustular forms to result in an ulcer with a black eschar and adjacent brawny edema (once fully developed, it may be painless). Spontaneous healing usually follows, but a small minority of untreated patients develop rapidly fatal bacteremia.
- Gastrointestinal anthrax exhibits variable symptoms: fever, nausea/vomiting, abdominal pain, bloody diarrhea, ascites, pharyngitis, and tonsillitis.
- Diagnosis may be established via Gram stain, direct fluorescent antibody stain, culture of skin lesions, or testing of sera for antibodies to the organism. Blood cultures may also be positive. Lab findings can include normal leukocyte counts (mild cases) or leukocytosis.
- Treatment includes either ciprofloxacin 750 mg PO bid or 400 mg IV or doxycycline 100 mg PO or IV bid. Therapy is given for 10 to 14 days. Alternatives include penicillin or erythromycin.[23]

PLAGUE

- Plague or *Yersinia pestis* is a gram-negative bacillus of the Enterobacteriaceae family and is endemic to the United States. It is most often found in rock squirrels and ground rodents of the Southwest but may also be carried by cats and dogs. The rodent flea is the primary vector.
- Transmission to humans occurs via the bite of a flea from an infected animal host or through ingestion of infected rodents, resulting in three clinical forms of human disease: (1) bubonic or suppurative (most common), (2) pneumonic, or (3) septicemic.
- The incubation period ranges from 2 to 7 days following exposure. Frequently an eschar develops at the bite site, followed by a painful, sometimes suppurative bubo (enlarged regional lymph nodes), often at the groin.
- Associated symptoms may include fever, headache, malaise, abdominal pain, nausea/vomiting, and bloody diarrhea.
- Some 10 to 20 percent of patients progress to develop secondary pneumonia with multilobar infiltrates, bloody sputum, and respiratory failure—this form is highly contagious and can be transmitted from person to person via aerosolized respiratory secretions (respiratory isolation is required).
- Subclinical disseminated intravascular coagulopathy (DIC) may also occur in a large number of patients—untreated bubonic plague may proceed to generalized sepsis, hypotension, and death.
- Diagnosis must depend on clinical features in a patient with possible contact with fleas or host animal—needle aspiration of a bubo with direct staining using Wayson's or Giemsa stain reveals bipolar ''safety pin''–shaped organisms. Fluorescent antibody staining of aspirate or antibody titers of acute and convalescent sera also confirms the diagnosis.
- Laboratory findings are nonspecific and may include leukocytosis, modest elevations of hepatic transaminases, and DIC.
- Therapy should begin immediately for any suspected case—treat as an inpatient with gentamicin 2.0 mg/kg IV loading dose, then 1.7 mg/kg IV q 8 h or streptomycin 1.0 g q 12 h IV or IM; therapy is continued for 10 to 14 days. Alternatives include a combination of tetracycline and an aminoglycoside or chloramphenicol.[24]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 145, ''Infections from Animals,'' by John T. Meredith.

95 SOFT TISSUE INFECTIONS

Chris Melton

GAS GANGRENE

Pathophysiology

- *Clostridium* species are the etiologic organisms, with *Clostridium perfringens* the most common isolate.[1]
- *Clostridium* produces exotoxins that cause cellular death, rapid progression, and systemic toxicity. Other effects secondary to tissue death include the release of myoglobin, creatine phosphokinase, and potassium. Bacteremia is rare.
- Mechanisms for infection with *Clostridium* include direct inoculation in open wounds and hematogenous spread in the immunocompromised.
- *Clostridium* thrives in contaminated wounds and wounds that offer an anaerobic environment.

CLINICAL FEATURES

- Also known as clostridial myonecrosis, gas gangrene presents with pain out of proportion to physical findings and a sense of heaviness in the affected part.
- Physical examination may reveal edema, brownish skin, bullae, malodorous discharge, and crepitance.
- Low-grade fever and tachycardia out of proportion to the fever are common findings.
- Delirium and irritability may be systemic manifestations of gas gangrene.

DIAGNOSIS AND DIFFERENTIAL

- Findings that may aid in confirming the diagnosis include gas in the soft tissues on plain radiographs, metabolic acidosis, leukocytosis, anemia, thrombocytopenia, myoglobinuria, and renal or hepatic dysfunction.
- The differential diagnosis includes other gas-forming infections such as necrotizing fasciitis, streptococcal myositis, acute streptococcal hemolytic gangrene, and crepitant cellulitis.
- Other causes of crepitance should be excluded, including pneumothorax, laryngeal or tracheal fracture, and pneumomediastinum.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The patient should be resuscitated with IV fluids as indicated. Packed red blood cells may be needed for resuscitation if there has been significant hemolysis.
- Vasoconstrictors should be avoided because of compromised perfusion in the affected part.
- Antibiotic therapy should be initiated, including penicillin G plus either vancomycin or a penicillinase-resistant penicillin such as nafcillin. If the patient is allergic to penicillin, clindamycin or metronidazole may be used.
- Tetanus prophylaxis should be administered as indicated.
- The patient should be admitted for surgical de-

bridement, hyperbaric oxygen therapy, and continued IV antibiotics.

CELLULITIS

PATHOPHYSIOLOGY

- Cellulitis results from soft tissue bacterial invasion, most commonly with *Staphylococcus* and *Streptococcus* in adults and *Haemophilus influenzae* in nonimmunized children.
- In patients with diabetes mellitus, Enterobacteriaceae and *Clostridium* should be considered as etiologic agents in addition to *Staph.* and *Strep.*
- Local inflammation occurs at the site of infection and is responsible for the clinical manifestations.[2] In patients who are immunosuppressed, systemic involvement including bacteremia, fever, and leukocytosis may occur.

CLINICAL FEATURES

- Features of cellulitis include localized tenderness, erythema, and induration.
- Cellulitis may progress to lymphangitis and lymphadenitis, which indicate a more severe infection.
- Bacteremia may develop, along with fever and chills.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is usually based on clinical findings.
- In patients with immune compromise or those with evidence of bacteremia, blood cultures and leukocyte counts are indicated.
- The differential diagnosis includes any erythematous skin condition.
- Cellulitis may be complicated by deep venous thrombosis. If there is evidence of venous obstruction, a venogram or Doppler study should be performed.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Simple cellulitis may be treated with an outpatient oral antibiotic such as dicloxacillin, a macrolide antibiotic, or amoxicillin/clavulanate.
- All patients should receive close follow-up to evaluate the patient's cellulitis and response to therapy.
- Patients with diabetes mellitus, alcoholism, immunosuppression, or evidence of systemic infection require admission for IV antibiotics. Choices include a first-generation cephalosporin such as cefazolin or a penicillinase-resistant penicillin such as nafcillin.
- In patients with diabetes, ceftriaxone may be used, while imipenem may be used in severe cases of cellulitis.[3]

ERYSIPELAS

PATHOPHYSIOLOGY

- Erysipelas is a superficial cellulitis with lymphatic involvement usually caused by group A streptococci.
- Inoculation occurs through a portal in the skin.
- Peripheral vascular disease is a significant risk factor for erysipelas.
- Most commonly the infection involves the lower extremities.[4]

CLINICAL FEATURES

- Erysipelas has an acute onset, with fever, chills, malaise, and nausea.
- A small area of erythema with a burning sensation then develops over the next 1 to 2 days.
- The sharply demarcated erythema is tense and painful.
- Lymphangitis and lymphadenitis commonly develop.
- Purpura, bullae, and necrosis may occur with the erythema.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is based primarily on physical findings.
- Differential diagnosis includes other types of local cellulitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- In nondiabetic patients, penicillin G may be used.
- Penicillinase-resistant penicillins such as nafcillin or a parenteral second- or third-generation cepha-

losporin should be used in patients with diabetes and those with facial involvement.
- Severe cases should be treated with imipenem.
- In patients allergic to penicillin, a macrolide may be used.
- Except for clearly mild cases of erysipelas, admission for IV antibiotics is required.

CUTANEOUS ABSCESSES

PATHOPHYSIOLOGY

- Cutaneous abscesses result from the breakdown of the cutaneous barrier and subsequent contamination with bacteria. The bacteria cause necrosis and liquefaction, followed by loculation and walling off to form the abscess.
- Staphylococcal species are most often the causative organisms.
- Streptococci may be the etiologic agent in tissues surrounding the oral and nasal mucosa.
- Intertriginous and perineal skin may become infected with *Escherichia coli, Proteus mirabilis,* and *Klebsiella* species.
- Axillary abscesses are frequently caused by *P. mirabilis.*
- Perirectal abscesses and abscesses in the genital region are frequently mixed anaerobic and aerobic species. *Bacteroides* is the most common anaerobe infecting these regions.
- Abscesses secondary to foreign bodies are usually caused by *Staph. aureus.*

CLINICAL FEATURES AND EMERGENCY DEPARTMENT CARE

- An abscess presents with an area of swelling, tenderness, and erythema. This area is usually localized and often fluctuant.
- Bartholin's gland abscess is a unilateral infection of the labia. *Neisseria gonorrhoeae* and *Chlamydia trachomatis* are common isolates from these abscesses. Treatment involves incision and drainage (I&D) along the vaginal mucosal surface of the abscess and then insertion of a Word catheter. Antibiotics are generally not needed unless a sexually transmitted disease is suspected.
- Hidradenitis suppurativa is a recurrent infection of the apocrine sweat glands, typically in the axilla and the groin. The most common isolate is *Staphylococcus,* although *Streptococcus* may also be present. These abscesses are typically multiple and in different stages of development. Treatment in the emergency department (ED) should include I&D of acute abscesses, antibiotics for cellulitis if present, and referral to a surgeon.
- Infected sebaceous cysts occur in sebaceous glands, which are located throughout the skin. I&D is the appropriate treatment, with wound recheck in 2 to 3 days in the ED or surgeon's office.
- Pilonidal abscess presents along the superior gluteal fold. I&D should be performed with iodoform gauze packing. The wound should be rechecked in 2 to 3 days, the packing removed, and the wound repacked. Antibiotics should be given if there is accompanying cellulitis. Surgical referral should be made for definitive treatment.
- Staphylococcal soft tissue abscess may present in several ways. Folliculitis occurs with bacterial invasion and subsequent inflammation of a hair follicle. Folliculitis can usually be treated with warm compresses. If deeper invasion occurs and surrounding soft tissues become infected, a furuncle (boil) is formed. Warm compresses usually promote spontaneous drainage. When several furuncles coalesce, they may form large interconnected sinus tracts and abscesses called a carbuncle. Carbuncles usually require surgical referral for wide excision.
- Conscious sedation should be considered in all patients who require I&D.
- In healthy, immunocompetent patients, antibiotics are not necessary unless there is secondary infection.
- If the patient is immunocompromised, the threshold for antibiotic use should be lowered.
- Patients with secondary cellulitis or systemic symptoms should be given antibiotics.
- Abscesses involving the face and hands should also be given antibiotics.
- Antibiotic choices include a first-generation cephalosporin such as cephalexin, clindamycin, or amoxicillin/clavulanate.
- Prophylactic antibiotics should be given to patients with structural cardiac abnormalities.

SPOROTRICHOSIS

PATHOPHYSIOLOGY

- This disease is caused by traumatic inoculation of the fungus *Sporothrix schenckii,* which is found on plants and in the soil.[5]

CLINICAL FEATURES

- The incubation period is 3 weeks.
- Three types of infection may present. The fixed cutaneous type presents as a crusted ulcer or a verrucous plaque at the site of inoculation. The local cutaneous type also is at the site of inoculation but presents as a subcutaneous nodule or pustule with or without surrounding erythema. The lymphocutaneous type (most common) presents as a painless nodule at the site of inoculation and develops subcutaneous nodules with migration through lymphatic channels.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on history and physical examination.
- Tissue biopsy cultures may yield a diagnosis but are of limited use in the ED.
- The differential diagnosis includes tuberculosis, tularemia, cat-scratch disease, leishmaniasis, nocardiosis, and staphylococcal lymphangitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Itraconazole for 3 to 6 months is effective in treating sporotrichosis.[6]
- If disseminated, sporotrichosis may be treated with IV amphotericin B.
- Most patients with cutaneous sporotrichosis can be treated on an outpatient basis.
- Patients with systemic symptoms should be admitted for possible treatment with amphotericin B.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 146, "Soft Tissue Infections," by Steven G. Folstad.

96 COMMON VIRAL INFECTIONS

David M. Cline

- Viral illnesses are among the most common complaints bringing people to an emergency department. This chapter focuses on the viral illness for which antiviral therapy has been developed. Treatment of primary herpes zoster and mononucleosis is discussed in Chap. 84, and genital herpes is discussed in Chap. 88. Treatment of HIV is covered in Chap. 90, and treatment of cytomegalovirus is discussed in Chap. 97.

INFLUENZA A AND B

EPIDEMIOLOGY

- In the United States, flu generally occurs from November to April. Influenza is spread by droplets generated by coughing. During epidemics, attack rates are in the 20 to 30 percent range; they may be as high as 50 percent during pandemics.[1]
- After exposure, the incubation period is usually about 2 days. Viral shedding (contagiousness) starts approximately 24 h before the onset of symptoms, rises to peak levels within 48 h, and then declines over the next 3 to 7 days.

PATHOPHYSIOLOGY

- Influenza viruses are single-stranded RNA viruses of the orthomyxovirus family.
- Following exposure, the virus enters the columnar cells of the respiratory tract epithelium. The invaded epithelial cells release large numbers of virions before cell death: thus, large numbers of viri-

ons are available for spread with respiratory secretions.

CLINICAL FEATURES

- Classic flu symptoms include fever of 38.6° to 39.8°C (101° to 103°F), with chills or rigor, headache, myalgia, and generalized malaise.
- Respiratory symptoms include dry cough, rhinorrhea, and sore throat, frequently with bilateral tender, enlarged cervical lymph nodes.
- Almost half of affected children have gastrointestinal symptoms, but these are unusual in adults.
- The fever generally lasts 2 to 4 days, followed by rapid recovery from most of the systemic symptoms.

DIAGNOSIS AND DIFFERENTIAL

- A clinical diagnosis of flu during a known outbreak has an accuracy of approximately 85 percent,[2,3] but bacteremia should also be considered in patients with rigor and myalgia.
- Newer rapid antigen tests are becoming available that may change the approach to flulike illnesses. One commercially available test requires a little more than 1 h to perform and lists a sensitivity of 50 to 70 percent, with a specificity of 93 to 100 percent.[4]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Two antiviral drugs—amantadine and rimantadine—have been available for the treatment of influenza A. For maximal effectiveness, both need to be started within 48 h of onset of symptoms and can reduce the duration of systemic symptoms by 1 to 2 days. The dose is 100 mg two times daily for 5 days for both drugs.
- Amantadine causes an increase in seizure activity in patients with a preexisting seizure disorder.
- Rimantadine has a significantly lower incidence of central nervous system (CNS) side effects than does amantadine but is seven times more expensive. Neither medicine should be used during pregnancy.
- A new medication, zanamavir, appears promising in clinical trials and has activity against both influenza A and B.[5] It was approved by the FDA in 1999.

HERPES SIMPLEX VIRUS 1

EPIDEMIOLOGY

- Transmission of herpes simplex virus (HSV) is via contact of infected secretions (saliva or genital) with mucous membranes or with open skin.

PATHOPHYSIOLOGY

- After exposure, the virus replicates locally in the epithelial cells, causing lysis of the infected cells and producing an inflammatory response. This response results in the characteristic rash of HSV, which is indistinguishable from the rash of varicella zoster virus (VZV).
- Following primary infection, the virus becomes latent in a sensory nerve ganglion.

CLINICAL FEATURES

- HSV-1 primarily causes oral lesions but may cause genital infection. The primary infection of HSV-1 is often mild or asymptomatic. The lesions are distributed throughout the mouth; they are raised, erythematous, and may not become vesicular.
- In children under age 5, HSV-1 may present as a pharyngitis or gingivostomatitis associated with fever and cervical lymphadenopathy.
- The primary lesions generally last 1 to 2 weeks.
- Recurrent oral lesions occur in 60 to 90 percent of infected individuals; they are usually milder than primary lesions and generally occur on the lower lip at the outer vermilion border. The recurrences are often triggered by local trauma, sunburn, or stress. The patient may have ''tingling'' prior to developing lesions. The lesions may begin as erythematous papules and then become vesicular.

DIAGNOSIS AND DIFFERENTIAL

- Viral cultures confirm the clinical diagnosis.
- The diagnosis is largely clinical.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Oral acyclovir has been shown to shorten the duration of symptoms in children if begun within the

first 72 h of symptoms. Treatment of recurrent oral herpes labialis with oral acyclovir 400 mg five times per day in adults shortens duration of symptoms.[6]
- Topical pencyclovir applied every 2 h for 4 days shortens duration of symptoms and has recently been approved for this indication.[7]

HERPES ZOSTER

PATHOPHYSIOLOGY

- Herpes zoster (shingles) is the reactivation of a latent herpes zoster virus infection. There is a lifetime incidence of almost 20 percent, with the majority of cases being among the elderly.

CLINICAL FEATURES

- The lesions of shingles are identical to those of chickenpox but are limited to a single dermatome in distribution. Thoracic and lumbar dermatomes are most common.
- The cranial nerves may be affected as well, with the potential complications of herpes zoster ophthalmicus[8] (HZO) and Ramsay Hunt syndrome[9] (symptoms similar to those of Bell's palsy).
- The disease begins with a prodrome of pain in the affected area for 1 to 3 days, followed by the outbreak of a maculopapular rash that quickly progresses to a vesicular rash. The course of the disease is usually around 2 weeks, but it may persist for a full month.
- Ocular involvement can be seen in the presence of only a slight rash on the forehead. HZO induces keratitis and may be followed by involvement of deeper structures. A dendriform corneal ulcer can often be identified with fluorescein staining.
- The most common complication of shingles is postherpetic neuralgia (PHN). PHN occurs in 10 to 20 percent of all patients after an episode of acute zoster but in up to 70 percent of patients aged 70 years or older. Treatment with antiviral medications decreases the duration of PHN.[10] It generally resolves in 1 to 2 months but may last longer than a year in some patients.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of herpes zoster in the normal host is aimed at decreasing the risk of PHN, as the antivirals have a clinically small but statistically significant effect on the duration of the acute disease.
- Treatment should begin as soon as possible—within 72 h of disease onset for maximal benefit.
- There is a suggestion that both famciclovir (500 mg two times a day for 7 days) and valacyclovir (1000 mg three times a day for 7 days) may be more effective than acyclovir (800 mg five times a day for 7 days), but this has not been shown to be clinically significant.[10]
- Initial treatment of patients with PHN is typically systemic analgesia, often narcotics. Patients should be referred back to their primary care provider, because first-line agents often fail and a trial of amitriptyline or carbamazepine may be tried as second-line therapy.
- HZO or suspected HZO mandates an ophthalmologic consultation due to the threat to vision.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 150, ''Common Viral Infections: Influenzaviruses and Herpesviruses,'' by Robert A. Brownstein.

97 THE TRANSPLANT PATIENT

David M. Cline

- Compromised response to infection and other side effects of immunosuppressive medication are common to all transplant recipients. Disorders specific to the transplanted organ are manifestations of acute rejection, surgical complications specific to the procedure performed, and altered physiology (most important in cardiac transplantation). Also, the management of routine injuries or illnesses may be complicated by the patient's immunosuppressed state of medication.

POSTTRANSPLANT INFECTIOUS COMPLICATIONS

- Predisposing factors to posttransplant infections include ongoing immunosuppression in all patients and the presence of diabetes mellitus, advanced age, obesity, and other host factors in some.
- Table 97-1 displays the broad array of potential infections and the time after transplant they are most apt to occur.[1]
- The most common infection in recipients of solid organs, especially in bone-marrow graft recipients, is cytomegalovirus (CMV). This infection may manifest with daily fever and malaise in its mildest form. Progressively more serious disease manifestations include leukopenia, hepatopathy (elevated transaminase enzymes), enteropathy (epigastric pain and diarrhea), and pneumonitis. Mortality associated with CMV pneumonitis exceeds 50 percent.
- Patients presenting with a febrile illness should have as part of their assessment a complete blood cell count, chest x-ray, and measurement of liver function. During active CMV infection, immunosuppression is maintained at the lowest possible level, and if liver, gut, or pulmonary involvement is documented, intravenous ganciclovir therapy, often in conjunction with immune globulin, is prescribed.

TABLE 97-1 Infectious Complications of Whole-Organ Transplantation

FIRST MONTH POSTTRANSPLANT
Bacterial
Wound infection (*Staph. aureus, S. epidermidis* gram-negative bacilli)
Pneumonia (gram-negative bacilli)
Urinary tract infection (gram-negative bacilli, enterococcus)
Line-related sepsis (*Staph. aureus, S. epidermidis,* gram-negative bacilli)
Intraabdominal infections (liver transplant)
Viral
HSV
Fungal
Candidal pharyngitis, esophagitis, cystitis
SECOND TO SIXTH MONTH POSTTRANSPLANT
Bacterial
Pneumonia: pneumococcal and other community acquired
Meningitis (*Listeria monocytogenes*)
Urinary tract infection
Nocardial infection
Listeriosis
Viral
Cytomegalovirus, EBV, HSV, varicella zoster
Adenovirus
Hepatitis A, B, C
Fungal
Aspergillosis
Candidal pharyngitis, esophagitis, cystitis
Other opportunistic infection
Pneumocystis carinii pneumonia, tuberculosis, toxoplasmosis
BEYOND SIXTH MONTH POSTTRANSPLANT
Bacterial
Pneumonia: pneumococcal and other community acquired
Urinary tract infection
Listeriosis
Viral
Cytomegalovirus chorioretinitis
Varicella zoster
Hepatitis C, B
Fungal
Cryptococcal
Other opportunistic infection
P. carinii pneumonia

ABBREVIATIONS: HSV = herpes simplex virus; EBV = Epstein-Barr virus.

- The initial presentation of a potentially life-threatening infectious illness may be quite subtle in transplant recipients. The transplant recipient receiving glucocorticoids may not mount an impressive febrile response.
- A nonproductive cough with little or no findings on physical examination may be the only clue to emerging *Pneumocystis carinii* pneumonia or CMV pneumonia. The threshold for obtaining chest x-rays for these patients should be low.

- Central nervous system (CNS) infections are more common in transplant recipients than in other patients. Common etiologies include *Listeria monocytogenes* and cryptococci. Complaints of recurrent headaches, therefore, with or without fever, should be investigated vigorously, first with a structural study to exclude a mass lesion (CNS lymphomas occur with increased frequency too), then with a lumbar puncture.
- A significant subset of renal transplant recipients have undergone intentional splenectomy to improve allograft survival. Although this procedure is no longer routinely practiced, these patients, like other postsplenectomy patients, are at particularly high risk for overwhelming sepsis caused by encapsulated bacteria such as pneumococci or meningococci.
- Liver transplant patients are especially susceptible to intraabdominal infections during the first postoperative month.
- Lung transplant patients are especially prone to pneumonia during the first 3 postoperative months.
- Cardiac transplant patients may develop mediastinitis during the first postoperative month.

MANAGEMENT OF INFECTION

- Drug choice, dose, and ultimate management should be accomplished in consultation with the transplant team.
- For skin and superficial wounds, probable offending organisms are gram-positive cocci, especially *Staphylococcus aureus,* and treatment should be with a penicillinase-resistant penicillin (e.g., nafcillin or oxacillin) or a first-generation cephalosporin (e.g., cefazolin).
- If there is a suspicion of methicillin-resistant organisms or sensitivity to β-lactams, vancomycin should be used.
- Nosocomial pneumonia is likely due to gram-negative organisms such as *Escherichia coli, Enterobacter,* or *Pseudomonas* and should be treated with a broad-spectrum antibiotic (e.g., cefoxitin, cefotetan, cefotaxime, ceftriaxone, and ceftazidime). Community-acquired pneumonia should be treated as such, with the proviso that opportunistic infection may also be present.
- Intraabdominal infection may be due to enterococci, gram-negative bacilli, or anaerobes and sometimes *Staph. aureus.* Triple coverage may be necessary empirically, with ampicillin or vancomycin plus an aminoglycoside to treat enterococci; a broad-spectrum penicillin or second- or third-generation cephalosporin to treat gram-negative organisms; and piperacillin, cefoxitin, cefotetan, clindamycin, or metronidazole to treat anaerobes. Penicillins with β-lactamase inhibitors (e.g., sulbactam and clavulanic acid) have broad coverage against gram-positive cocci, gram-negative bacilli, and anaerobes.
- Meningitis is frequently due to *L. monocytogenes,* and patients with suspected meningitis should be treated with a third-generation cephalosporin and ampicillin.
- The mainstay of fungal treatment has been amphotericin B. *Candida albicans* can be treated first with fluconazole.
- Viral therapy depends on the disease syndrome and the offending agent. CMV disease is treated with ganciclovir, with a dose of 5 mg/kg intravenously twice daily. Varicella and herpes simplex virus are typically treated with acyclovir, which has renal excretion; the dose must be adjusted for renal insufficiency. Epstein-Barr virus (EBV) is typically treated with a reduction in the immunosuppression regimen.
- Treatment of choice for *P. carinii* pneumonia is cotrimoxazole, with pentamidine reserved as an alternative therapy if cotrimoxazole is not tolerated. Toxoplasmosis is treated with pyrimethamine/sulfadiazine or clindamycin.
- Urinary tract infection, invasive gastroenteritis (due to *Salmonella, Campylobacter,* and *Listeria*) and diverticulitis can be treated with the usual antimicrobial agents.

COMPLICATIONS OF IMMUNOSUPPRESSIVE AGENTS

- Therapeutic immunosuppression is accompanied by a number of side effects and complications (Table 97-2). Combined toxicities can produce or worsen preexisting renal insufficiency, hypertension, and hyperglycemia.
- Elevated cyclosporine levels cause renal arteriolar constriction, which reduces glomerular blood flow and stimulates the renin-angiotensin system and raises blood pressure.
- Glucocorticoids promote renal salt and water retention, which further aggravate hypertension.
- A headache syndrome often indistinguishable from migraine is common in transplant recipients and usually develops within the first 2 months of immunosuppression. An important differential must include infectious causes and malignancy when headache first presents and usually requires a computed tomography (CT) scan of the head

TABLE 97-2 Drug Side Effects

Cyclosporine	
	Nephrotoxicity
	Neurotoxicity—tremors, seizures, headaches
	Hyperkalemia
	Hyperuricemia
	Hypertension
	Hypomagnesemia
	Anorexia
	Hyperbilirubinemia
	Glucose intolerance
	Cholestasis
	Gastric dysmotility
Prednisone	
	Cushing's syndrome
	Osteoporosis
	Adrenal suppression
	Hypertension
	Hyperglycemia
	Peptic ulcer disease
	Myopathy
	Poor wound healing
Azathioprine (Imuran)	
	Leukopenia
	Thrombocytopenia
	Cholestatic jaundice
	Alopecia

with subsequent biochemical analysis of cerebrospinal fluid.[2]

- Recently, the newer immunosuppressive agents tacrolimus and mycophenolate mofetil have been used in place of cyclosporine and azathioprine, respectively.[3,4] The most common side effects of tacrolimus are similar to those of cyclosporine. The most common side effects of mycophenolate mofetil are diarrhea, vomiting, leukopenia, and increased opportunistic infections, especially CMV.[5]
- Any illness that prevents transplant patients from taking or retaining their immunosuppressive therapy warrants hospital admission for intravenous therapy, preferably at a transplant center.

CARDIAC TRANSPLANTATION

- Transplantation results in a denervated heart that does not respond with centrally medicated tachycardia in response to stress or exercise but does respond to circulating catecholamines and increased preload.[6] Patients may complain of fatigue or shortness of breath with the onset of exercise, which resolves with continued exertion as an appropriate tachycardia develops.
- The donor heart is implanted with its sinus node intact to preserve normal atrioventricular conduction. The normal heart rate for a transplanted heart is 90 to 100 beats per minute.
- The technique of cardiac transplantation also results in the preservation of the recipient's sinus node at the superior cavoatrial junction. The atrial suture line renders the two sinus nodes electrically isolated from each other. Thus, electrocardiograms (ECGs) will frequently have two distinct P waves. The sinus node of the donor heart is easily identified by its constant 1:1 relationship to the QRS complex, whereas the native P wave marches through the donor heart rhythm independently.

CLINICAL FEATURES

- Because the heart is denervated, myocardial ischemia does not present with angina. Instead, recipients present with heart failure secondary to silent myocardial infarctions or with sudden death.
- Transplant recipients who present with new-onset shortness of breath, chest fullness, or symptoms of congestive heart failure (CHF) should be evaluated in routine fashion with ECG and serial cardiac enzyme levels for the presence of myocardial ischemia or infarction.
- Although most episodes of acute rejection are asymptomatic, symptoms can occur. The most common presenting symptoms are dysrhythmias and generalized fatigue. The development of either atrial or ventricular dysrhythmias in a cardiac transplant recipient (or CHF patient) must be assumed to be due to acute rejection until proven otherwise.
- In children, rejection may present with low-grade fever, fussiness, and poor feeding.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Consultation: Differentiating rejection from other acute illness in the transplant patient can be difficult. Treatment for rejection without biopsy confirmation is contraindicated except when the patient is hemodynamically unstable.
- Rejection: Management of acute rejection is 1 g of methylprednisolone intravenously, after consultation with a representative from the transplant center.
- Dysrhythmias: If patients are hemodynamically compromised by dysrhythmias, empiric therapy for rejection with methylprednisolone, 1 g intravenously, may be given after consultation. Atrial dysrhythmias may respond to treatment with di-

goxin or calcium channel blockers. Ventricular dysrhythmias may respond to lidocaine or other class I-C agents. Frequently dysrhythmias will be controlled only with antirejection therapy. Atropine has no effect on the denervated heart; isoproterenol is the drug of choice for bradydysrhythmias in these patients.[6]

- Hypotension: Low-output syndrome or hypotension should be treated with inotropic agents such as dopamine or dobutamine when specific treatment for rejection is instituted.
- Hospitalization: Transplant patients suspected of having rejection or acute illness should be hospitalized, preferably at the transplant center the patient is if stable for transfer.

LUNG TRANSPLANTATION

CLINICAL FEATURES

- Clinically, patients with rejection may have cough, chest tightness, fatigue, and fever (>0.5°C or 0.9°F above baseline).[7] Acute rejection may manifest with frightening rapidity, causing a severe decline in patient status in only a day.
- Isolated fever may be the only finding; in contrast, spirometry may show a 15% drop in FEV_1, and examination may reveal rales and adventitious sounds.
- Chest x-ray may demonstrate bilateral interstitial infiltrates, septal lines, and effusions. The chest x-ray may be normal, however, when rejection occurs late in the course. The longer period of time a patient is from transplant, the less classic a chest x-ray may appear for acute rejection.
- Infection, such as interstitial pneumonia, may present with a clinical picture similar to that of acute rejection. Diagnostically, bronchoscopy with transbronchial biopsy is usually needed not only to confirm rejection but to exclude infection.
- Two late complications of lung transplant are obliterative bronchiolitis and posttransplant lymphoproliferative disease (PTLD).[8,9] Obliterative bronchiolitis presents with episodes of recurrent bronchitis, small airway obliteration, wheezing, and, eventually, respiratory failure. PTLD is associated with Epstein-Barr virus and presents with painful lymphadenopathy and otitis media (due to tonsillar involvement), or it may present with malaise, fever, and myalgia.
- Evaluation of the lung-transplant patient should include chest x-ray, arterial blood gas analysis, complete blood cell count (CBC), serum electrolytes, creatinine, and magnesium levels; in some cases, it should also include a cyclosporine level.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Consultation: Communication should be made directly with the transplant center (often a nurse coordinator). Coordinators should have the patient's current medication doses, recent infection history, and knowledge of complications for which the patient may be at risk.
- Rejection: If clinically indicated (i.e., infection is excluded), methylprednisolone 500 to 1000 mg intravenously should be given. Patients who have a history of seizures associated with the administration of high-dose glucocorticoids will also need concurrent benzodiazepines to prevent further seizure episodes.
- Late complications: Obliterative bronchiolitis is treated with increased immunosuppression, whereas PTLD is treated with reduced immunosuppression. These decisions should be made by specialists from the transplant center.

RENAL TRANSPLANT

CLINICAL FEATURES

- Diagnosis and treatment of acute rejection are most critical. Without timely recognition and intervention, allograft function may deteriorate irreversibly in a few days.[10]
- Renal transplant recipients, when symptomatic from acute rejection, complain of vague tenderness over the allograft (in the left or right iliac fossa).
- Patients may also describe decreased urine output, rapid weight gain (from fluid retention), low-grade fever, and generalized malaise.[11]
- Physical examination may disclose worsening hypertension, allograft tenderness, and peripheral edema.
- The absence of these symptoms and signs, however, does not exclude the possibility of acute rejection.
- With improved methods of maintenance immunosuppression, the only clue may be an asymptomatic decline in renal function. Even a change in creatinine levels from 1.0 to 1.2 or 1.3 mg/dL may be important. When such changes in creatinine levels are reproducible, a careful workup consists of complete urinalysis, renal ultrasonography, and

a trough level of cyclosporine in addition to a careful history and examination. It is critical to interpret changes in renal function in the context of prior data (e.g., trends of recent serum creatinine levels, recent history of rejection, or other causes of allograft dysfunction).
- Evaluation should consider the multiple etiologies of decreased renal function in the renal transplant recipient. The two most common causes, apart from acute rejection causing an increase in creatinine, are volume contraction and cyclosporine-induced nephrotoxicity.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Consultation: Communication should be made directly with the transplant center (often a nurse coordinator). Coordinators should have the patient's current medication doses, recent infection history, and knowledge of complications for which the patient may be at risk.
- Rejection: Treatment of allograft rejection consists of high-dose glucocorticoids, typically methylprednisolone (250 to 500 mg intravenously).

LIVER TRANSPLANT

CLINICAL FEATURES

- Though frequently subtle in presentation, a syndrome of acute rejection includes fever, liver tenderness, lymphocytosis, eosinophilia, liver enzyme elevation, and a change in bile color or production.
- In the perioperative period, the differential diagnosis must include infection, acute biliary obstruction, and vascular insufficiency.
- Diagnosis can be made with certainty only by hepatic ultrasound and biopsy, which usually requires referral back to the transplant center for management and follow-up.
- Three possible surgical complications in liver transplant patients are biliary obstruction, biliary leakage, and hepatic artery thrombosis.[12]
- Biliary obstruction follows three typical presentations. The most common is intermittent episodes of fever and fluctuating liver function tests. The second is a gradual worsening of liver function tests without symptoms. Finally, obstruction may present as acute bacterial cholangitis with fever, chills, abdominal pain, jaundice, and bacteremia.
- It can be difficult to distinguish clinically between rejection, hepatic artery thrombosis, CMV infection, or a recurrence of a preexisting disease, especially hepatitis.
- Patients most often have peritoneal signs and fever, but these signs may be masked by concomitant use of steroids and immunosuppressive agents.
- Presentation is signaled by elevated PT and transaminase levels and little or no bile production, but this complication may also present as acute graft failure, liver abscess, unexplained sepsis, or a biliary tract problem (leak, obstruction, abscess, or breakdown of the anastomosis).
- If a biliary complication is suspected, all patients should have a CBC; serum chemistry levels; liver-function tests; amylase and lipase levels; cultures of blood, urine, bile, and ascites, if present; chest x-ray; and abdominal ultrasound.
- Ultrasound rules out the presence of fluid collections, screens for the presence of thrombosis of the hepatic artery or portal vein, and identifies any dilatation of the biliary tree.
- Biliary leakage is associated with 50 percent mortality. It occurs most frequently in the third or fourth postoperative week.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Consultation: Communication should be made directly with the transplant center (often a nurse coordinator). Coordinators should have the patient's current medication doses and recent infection history as well as knowledge of complications for which patients may be at risk.
- Rejection: Acute rejection is managed with high-dose glucocorticoid bolus.[13]
- Surgical complications are best managed at the transplant center. Biliary obstruction is managed with balloon dilatation, and all patients should receive broad-spectrum antibiotics against gram-negative and positive enteric organisms.[14] Biliary leakage is treated with reoperation, and hepatic artery thrombosis is treated with retransplantation.

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Section 13
TOXICOLOGY

98 GENERAL MANAGEMENT OF POISONED PATIENTS

Sandra L. Najarian

EPIDEMIOLOGY

- Poisonings are the third leading cause of death in the United States. The incidence has increased approximately 300 percent in recent years.
- The majority of exposures are ''accidental'' and preventable through increased awareness and education.

PATHOPHYSIOLOGY

- Poisons affect the body by inhibiting normal cellular function, changing normal organ function, or by changing the normal uptake or transport of substances into or within the organism.
- Routes of exposures include inhalation, insufflation, ingestion, injection, and cutaneous and mucous membrane exposure.

CLINICAL FEATURES

- Presentations are variable, requiring a detailed history and thorough head-to-toe examination with attention to vital signs, general appearance, skin, pupils, mucous membranes, heart, lung, gastrointestinal, and neurologic examinations.
- Toxidromes are a collection of signs and symptoms that are observed after exposure to a substance (Table 98-1).

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is made by history and physical examination; drug screens and other laboratory studies may be useful and often serve to confirm the diagnosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment includes initial stabilization, decontamination, elimination of the toxin, and administration of the antidote.
- The airway should be secured. In the obtunded patient, if gastric lavage is indicated, the patient should be intubated to protect the airway.
- Oxygen, naloxone, glucose, and thiamine should be administered for those patients found with altered mental status. Physical and chemical restraints (benzodiazepines or haloperidol) should be used for agitated patients.
- Surface decontamination consists of removing the patient from the toxic substance, undressing the patient completely, and washing the skin with copious amounts of water.
- Gastric decontamination includes gastric emptying, adsorption of the toxin in the gut, and irrigation of the bowel. Gastric lavage is recommended for patients presenting within 1 h of a potentially life-threatening ingestion.[1] Activated charcoal (1 g/kg) should be administered to bind any remaining toxin. Osmotic cathartics given with acti-

TABLE 98-1 Toxidromes

TOXIDROME	REPRESENTATIVE AGENT(S)	MOST COMMON FINDINGS	ADDITIONAL SIGNS AND SYMPTOMS	POTENTIAL INTERVENTIONS
Opioid	Heroin Morphine	CNS depression, miosis, respiratory depression	Hypothermia, bradycardia Death may result from respiratory arrest, pulmonary edema	Ventilation or naloxone
Sympathomimetic	Cocaine Amphetamine	Psychomotor agitation, mydriasis, diaphoresis, tachycardia, hypertension, hyperthermia	Seizures, rhabdomyolysis, myocardial infarction Death may result from seizures, cardiac arrest, hyperthermia	Cooling, sedation with benzodiazepines, hydration
Cholinergic	Organophosphate insecticides Carbamate insecticides	Salivation, lacrimation, diaphoresis, nausea, vomiting, urination, defecation, muscle fasciculations, weakness, bronchorrhea	Bradycardia, miosis/mydriasis, seizures, respiratory failure, paralysis Death may result from respiratory arrest secondary to paralysis and/or bronchorrhea, seizures	Airway protection and ventilaton, atropine, pralidoxime
Anticholinergic	Scopolamine Atropine	Altered mental status, mydriasis, dry/flushed skin, urinary retention, decreased bowel sounds, hyperthermia, dry mucous membranes	Seizures, dysrhythmias, rhabdomyolysis Death may result from hyperthermia and dysrhythmias	Physostigmine (if appropriate), sedation with benzodiazepines, cooling, supportive management
Salicylates	Aspirin Oil of wintergreen	Altered mental status, respiratory alkalosis, metabolic acidosis, tinnitus hyperpnea, tachycardia, diaphoresis, nausea, vomiting	Low-grade fever, ketonuria Death may result from pulmonary edema, cardiorespiratory arrest	MDAC, alkalinization of the urine with potassium repletion, hemodialysis, hydration
Hypoglycemia	Sulfonylureas Insulin	Altered mental status, diaphoresis, tachycardia, hypertension	Paralysis, slurring of speech, bizarre behavior, seizures Death may result from seizures, altered behavior	Glucose-containing solution intravenously, and oral feedings if able, frequent capillary blood for glucose measurement
Serotonin syndrome	Meperidine/dextromethorphan + MAOI, SSRI + TCA, SSRI/TCA/MAOI + amphetamine, SSRI overdose	Altered mental status, increased muscle tone, hyperreflexia, hyperthermia	"Wet dog shakes" (intermittent whole body tremor) Death may result from hyperthermia	Cooling, sedation with benzodiazepines, supportive management, theoretical benefit—cyproheptadine

ABBREVIATIONS: CNS = central nervous system; MDAC = multidose activated charcoal; MAOI = monoamine oxidase inhibitor; SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant.

vated charcoal reduce transit time through the gastrointestinal tract.[2, 3]

- Multidose activated charcoal administration is indicated after ingestions of toxins known to slow gut motility, toxins that have a slow release preparation, or toxins in a large quantity. Whole bowel irrigation with polyethylene glycol may be useful for eliminating sustained-release preparations, toxins not absorbed by activated charcoal, or packages of toxic drugs.[4]
- Specific antidotes should be administered once decontamination is underway. Elimination of certain toxins may be enhanced with methods such as urinary alkalization, hemoperfusion, or hemodialysis for specific toxins (Table 98-2).
- Psychiatric consultation is required for all intentional overdoses.

TABLE 98-2 Agents That Other Modalities May Increase Their Excretion

DRUGS	AGENTS WHERE URINARY ALKALIZATION IS COMMONLY CONSIDERED	AGENTS WHERE HEMODIALYSIS IS COMMONLY CONSIDERED
2-4-D (herbicide)	✓	
Phenobarbital	✓	
Chlorpropamide	✓	
Salicylates	✓	✓
Methanol	✓	✓
Ethylene glycol		✓
Lithium		✓
Theophylline*		✓

* Indicates also removed by hemoperfusion.

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99 ANTICHOLINERGIC TOXICITY

Mark B. Rogers

EPIDEMIOLOGY

- Anticholinergic toxicity is common because of frequent use of tricyclic antidepressants, phenothiazines, antihistamines, and antiparkinsonian drugs.
- Jimsonweed is the most common plant associated with anticholinergic toxicity.

PATHOPHYSIOLOGY

- The mechanism of action involves cholinergic blockade of either muscarinic receptors (primarily in the brain) or nicotinic receptors (from the spinal cord) or both.

CLINICAL FEATURES

- The clinical findings include mydriasis, hypotension or hypertension, absent bowel sounds, tachycardia, flushed skin, disorientation, urinary retention, hyperthermia, dry skin and mucous membranes, and auditory and visual hallucinations.
- Findings can be remembered using the mnemonic: Hot as Hades, Blind as a Bat, Dry as a Bone, Red as a Beet, and Mad as a Hatter.
- The most common electrocardiographic (ECG) finding is sinus tachycardia, but QRS prolongation, bundle branch blocks, atrioventricular dissociation, and atrial and ventricular tachycardias may be seen.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is clinical.
- With isolated anticholinergic toxicity, routine labs are usually normal and toxicology screening is of little value.
- In contrast, sympathomimetic toxicity and delirium tremens will show moist skin and active bowel sounds. Acute psychiatric disorders may have tachycardia and tachypnea, but the physical exam is otherwise unremarkable.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment is primarily supportive. Intravenous access and cardiac monitoring should be established. Gastric lavage may be useful within 1 h of ingestion. Oral activated charcoal (1 g/kg) may decrease absorption. Whole-bowel irrigation is recommended for jimsonweed ingestion up to 12 to 24 h after ingestion due to delayed gastric emptying of the seeds.
- Hyperthermia, hypertension, and seizures are treated conventionally, as necessary.
- Standard antidysrhythmics are usually effective, but class Ia agents should be avoided. Intravenous (IV) bicarbonate therapy may be effective for dysrhythmias, widened QRS complex, or hypotension due to sodium-channel blocking agents (e.g., cyclic antidepressants).
- For agitation, benzodiazepines should be administered. Phenothiazine use should be avoided.
- Physostigmine therapy (0.5 to 2.0 mg IV over 5 min) is controversial. It may be indicated only if conventional therapy fails to control life-threatening conditions of toxicity. Physostigmine may worsen the patient's condition and is contraindicated in cyclic antidepressant overdoses, cardiovascular or peripheral vascular disease, bronchospasm, intestinal obstruction, heart block, or bladder obstruction. Patients receiving physostigmine usually should be admitted for 24 h.

- With mild anticholinergic toxicity, the patient can be discharged after 6 h of observation with improvement.

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100 PSYCHOPHARMACOLOGIC AGENTS

Lance H. Hoffman

TRICYCLIC ANTIDEPRESSANTS

- Tricyclic antidepressants are associated with more drug-related deaths than any other prescription medication.
- Myocardial sodium channel antagonism leads to conduction abnormalities and decreased contractility.[1]
- Life-threatening ingestions usually require at least 10 mg/kg.
- Significant toxicity manifests within 6 h of the ingestion with hypotension, respiratory depression, cardiac dysrhythmias, dry mucosae, diminished bowel sounds, urinary retention, mental status changes, and seizures.
- An electrocardiogram showing a QRS interval >100 ms or right axis deviation of the terminal 40 ms >120° (R wave in AVR, S wave in I) is often indicative of an impending life-threatening complication.[2]
- Sodium bicarbonate should be used to treat QRS widening >100 ms, hypotension refractory to intravenous (IV) fluids, and ventricular dysrhythmias. Sodium bicarbonate should be dosed as 1 to 2 meq/kg IV followed by a continuous infusion of 2 to 3 ampules of sodium bicarbonate in 1 L of D_5W at a rate of 3 mL/kg/h. The potassium level should be monitored.
- Hypotension refractory to IV fluids and sodium bicarbonate therapy should be treated by titrating a norepinephrine infusion.[3]

NEWER ANTIDEPRESSANTS

TRAZODONE AND NEFAZODONE

- Acute toxicity includes central nervous system (CNS) depression, orthostatic hypotension, nausea, vomiting, abdominal pain, priapism, seizures, and, rarely, torsades de pointes.
- Patients with a trazodone or nefazodone overdose require IV access, continuous cardiac monitoring, and activated charcoal 1 g/kg orally (PO).
- Hypotension refractory to IV fluids should be supported with a norepinephrine infusion.

BUPROPION

- Toxicity begins manifesting near the maximum therapeutic dose of 450 mg/day.
- The hallmark of toxicity is generalized seizures, beginning an average of 4 h after the acute ingestion.
- Seizures should be treated with lorazepam or diazepam as first-line agents and phenobarbital as a second-line agent.

MIRTAZAPINE

- Mirtazapine is of limited toxicity in acute overdose. Manifestations include sinus tachycardia, mild hypertension, sedation, and confusion.[4]
- Patients with a mirtazapine overdose require IV access, continuous cardiac monitoring, and activated charcoal 1 g/kg PO.

SELECTIVE SEROTONIN REUPTAKE INHIBITORS

- Approximately 50 percent of adult patients and 75 percent of pediatric patients remain asymptomatic after an acute selective serotonin reuptake inhibitor (SSRI) overdose.

- Seizures and the serotonin syndrome are the most serious toxic effects.
- Citalopram can cause QRS widening and QT prolongation.[5]
- Patients with a SSRI overdose require IV access, continuous cardiac monitoring, and activated charcoal 1 g/kg PO.
- QRS widening >100 ms should be treated with sodium bicarbonate 1 to 2 meq/kg IV followed by a continuous infusion of 2 to 3 ampules of sodium bicarbonate in 1 L of D_5W at a rate of 3 mL/kg/h.

VENLAFAXINE

- Venlafaxine is a nonselective inhibitor of the reuptake of serotonin, norepinephrine, and dopamine that is used to treat depression.[6]
- Acute ingestions may result in tachycardia, hypertension or hypotension, diaphoresis, mydriasis, tremor, CNS depression, generalized seizures, QRS widening, and QT prolongation.
- Treatment includes IV access, continuous cardiac monitoring, activated charcoal 1 g/kg PO, lorazepam or diazepam for seizures, and sodium bicarbonate for a QRS interval >100 ms.

SEROTONIN SYNDROME

- The serotonin syndrome is a rare, idiosyncratic reaction caused by a drug or combination of drugs that increases central serotonin transmission.[7]
- Approximately 85 percent of cases occur at therapeutic drug levels.
- The serotonin syndrome is characterized by cognitive-behavioral, autonomic, and neuromuscular dysfunction, especially lower extremity muscle rigidity.
- Cyproheptadine 4 to 8 mg PO should be administered and repeated in 2 h, if no response is observed. If a response is observed, then cyproheptadine 4 mg PO should be continued every 6 h for the next 48 h.[7, 8]
- Benzodiazepines should be used for muscle rigidity and muscle pain.
- The patient should be monitored for acidosis and rhabdomyolysis.

MONOAMINE OXIDASE INHIBITORS

- Monoamine oxidase inhibitors (MAOIs) are used in the treatment of atypical depression or depression refractory to other agents.
- Monamine oxidase normally inactivates biogenic amines and decreases dietary absorption of biogenic amines such as tyramine, which is found in aged meats, cheeses, and red wine.
- Monamine oxidase inhibitor toxicity is achieved by three mechanisms: food interactions, drug interactions, and acute overdose.
- Ingestion of tyramine-containing foods results in a hyperadrenergic state within 90 min that lasts up to 6 h. This state may result in an acute myocardial infarction or intracranial hemorrhage.
- Drug interactions are varied and include the following: hyperadrenergic state with sympathomimetics, decreased clearance of opiates and sedative-hypnotics, hypoglycemia with sulfonylureas, and the serotonin syndrome when combined with serotonergic agents (e.g., meperidine and other antidepressants).
- Monoamine oxidase inhibitor toxicity of acute ingestion begins at doses <2 mg/kg. Ingestions of 4 to 6 mg/kg can be fatal. Acute toxicity manifests as a hyperadrenergic state, hemodynamic instability, seizures, and coma.
- All patients with MAOI toxicity require IV access, continuous cardiac monitoring, and supplemental oxygen.
- Only acute ingestions require gastrointestinal decontamination with gastric lavage and activated charcoal.
- Hypertension should be treated with phentolamine 2.5 to 5 mg IV every 10 min or a continuous, titrated infusion of sodium nitroprusside.
- Hypotension refractory to IV fluid boluses requires a continuous, titrated norepinephrine infusion.
- Bretylium and beta blockers are contraindicated secondary to catecholamine release and unopposed vasoconstriction associated with the respective agents.
- Benzodiazepines are useful for treating seizures and muscular rigidity. Nondepolarizing neuromuscular blockade or dantrolene 0.5 to 2.5 mg IV every 6 h may be needed if the benzodiazepines are ineffective.

ANTIPSYCHOTICS

- Therapeutic effects are due to dopamine receptor antagonism. Antipsychotic medications also antagonize $alpha_1$, adrenergic, muscarinic, and histaminergic receptors.
- Adverse effects include dystonic reactions, akathisia, tardive dyskinesia, and neuroleptic malignant syndrome.

- Acute overdose can cause QT-interval prolongation and ventricular dysrhythmias, seizures, depressed mental status, hypotension, and impaired thermoregulation.
- Piperidine phenothiazines (e.g., thioridazine) have the highest potential for ventricular dysrhythmias.[9–12]
- Class 1a antidysrhythmics (e.g., procainamide) and vasopressors with β-adrenergic activity (e.g., dopamine) are contraindicated.

LITHIUM

- The most common adverse effects of chronic lithium therapy are hand tremor, polyuria (nephrogenic diabetes insipidus), and rash.[13,14]
- Acute overdose results in prominent gastrointestinal disturbances such as vomiting and diarrhea, while chronic overdose results in prominent CNS disturbances such as seizures and coma. Both ingestions may cause cardiac conduction abnormalities and ventricular dysrhythmias.
- Activated charcoal does not bind lithium; however, it may still be useful in adsorbing coingested substances.
- Indications for hemodialysis are a lithium level >3.5 to 4.0, lithium level of 1.5 to 3.5 that is poorly responsive to 6 h of hydration with normal saline, increasing lithium levels with serial determinations, renal failure, and ingestion of a sustained-release preparation.[15]

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101 SEDATIVE-HYPNOTICS

Keith L. Mausner

BARBITURATES

EPIDEMIOLOGY

- Barbiturate abuse peaked in the 1970s, but has increased in the United States since 1990, especially among adolescents.[1–4]
- Barbiturates may be seen in conjunction with co-

caine or methamphetamine abuse, where they may be used to lessen the unpleasant extremes of the stimulant's effects.

PATHOPHYSIOLOGY

- Barbiturates are classified according to their duration of action: long-acting (barbital, phenobarbital, duration of action >6 h); intermediate-acting (amobarbital, duration of action 3 to 6 h); short-acting (pentobarbital, secobarbital, duration of action <3 h); and ultrashort-acting (thiopental, methohexital, duration of action 0.3 h).
- Long-acting barbiturates are weaker acids (lower pK_a values); in a basic medium they are largely ionized, and tissue permeability decreases. This is why forced alkaline diuresis is useful in treating long-acting barbiturate overdose. The intermediate-, short-, and ultrashort-acting barbiturates are stronger acids and are not affected by pH in this way; urinary alkalinization is not clinically useful.

CLINICAL FEATURES

- Drowsiness, disinhibition, ataxia, slurred speech, and confusion worsen with increasing dose and may progress to stupor, coma, or complete neurologic unresponsiveness.
- Respiratory depression and hypothermia are centrally mediated.
- Hypotension is due to decreased vascular tone and venous pooling.
- Pulse rate is not diagnostic, and pupil size and reactivity, nystagmus, and deep tendon reflexes are variable.
- Gastrointestinal motility is slowed, delaying gastric emptying.
- Hypoglycemia may occur.

DIAGNOSIS AND DIFFERENTIAL

- Barbiturate serum levels may establish the diagnosis and distinguish long- from short-acting barbiturates, since the treatment approach is different for each.
- Differential diagnosis includes intoxication with other sedative-hypnotic agents, alcohol, environmental hypothermia, and other causes of coma.
- Barbiturates are more likely to produce coma and myocardial depression than are benzodiazepines.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Cardiac monitoring and an intravenous (IV) line should be established.
- Fingerstick glucose determination is indicated for all patients with an altered level of consciousness, and administration of naloxone and thiamine should be considered.
- Laboratory studies, as clinically indicated, may include electrolytes; blood urea nitrogen; creatinine; complete blood cell count; toxicology screen, including acetaminophen to rule out coingestion; electrocardiogram; and chest x-ray. An arterial blood gas may be useful.
- Shock and hypotension should be treated with volume expansion using isotonic saline. In elderly patients, or those with congestive heart failure or renal failure, 200-mL boluses may be prudent. Dopamine or norepinephrine may be indicated if volume expansion is ineffective.[5]
- Activated charcoal (AC), 1 to 2 g/kg, should be administered to decrease absorption; the addition of a cathartic such as sorbitol may be beneficial. If there is risk of aspiration, the airway should be secured before AC administration. Multiple-dose AC every 4 h may decrease serum levels. There is no evidence of any benefit of gastric lavage over AC alone. Ipecac-induced emesis may be dangerous due to central nervous system (CNS) depression and risk of aspiration and should be avoided.
- Forced diuresis with saline and furosemide, titrating urine output to 4 to 6 mL/kg/h, is beneficial in phenobarbital poisoning.
- Urinary alkalinization promotes excretion of long-acting barbiturates. A sodium bicarbonate 1 to 2 meq/kg IV bolus should be administered, and then 100 to 150 meq bicarbonate should be added to 1 L D_5W and the drip rate adjusted to maintain an arterial pH of 7.45 to 7.50, urinary pH of 8.0, and urine output of 2 mL/kg/h. Serum potassium must remain at least 4.0 meq/L for alkalinization to be effective. Electrolytes should be checked every 2 to 4 h.
- Hemodialysis and hemoperfusion are indicated for patients who deteriorate despite aggressive supportive care.

- Monitoring and documentation of neurologic and vital signs improvement may allow patients with mild-to-moderate toxicity to be discharged to psychiatric care or home.
- Severe toxicity requires admission, and toxicology consultation is recommended.
- Barbiturate abstinence syndrome occurs with abrupt withdrawal in chronic users and produces minor symptoms within 24 h and major life-threatening manifestations in 2 to 8 days.
- Clinical findings are similar to alcohol withdrawal: anxiety, depression, insomnia, anorexia, nausea, vomiting, muscle twitching, abdominal cramping, and sweating. This may progress to psychosis, hallucinations, delirium, seizures, hyperthermia, and cardiovascular collapse.
- Aggressive supportive care should be instituted, and IV benzodiazepines or barbiturates should be administered, with subsequent tapering of dose.[6]

BENZODIAZEPINES

EPIDEMIOLOGY

- In 1996 there were 39,029 reported benzodiazepine toxic exposures.[7] There is a low mortality rate from isolated benzodiazepine ingestion.[8] However, mixed overdose results in high morbidity and mortality.

CLINICAL FEATURES

- The most significant effects are on the CNS, which include drowsiness, dizziness, slurred speech, confusion, and cognitive impairment. Headache, nausea, vomiting, chest pain, arthralgias, diarrhea, and incontinence also have been reported. Rare paradoxical reactions include rage and delirium.
- Respiratory depression and hypotension are more likely with parenteral administration or with coingestants.
- The elderly are more susceptible to adverse effects.
- Fatal isolated benzodiazepine ingestion is more likely with short-acting agents such as triazolam, alprazolam, or temazepam.

DIAGNOSIS AND DIFFERENTIAL

- Toxicology screening may help establish the diagnosis, but the laboratory may not screen for all available benzodiazepines. It is essential to know the laboratory's limitations.
- Serum benzodiazepine levels are not clinically useful in overdoses.
- The findings of benzodiazepine toxicity are nonspecific.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. As with barbiturates, many of the same initial principles apply for benzodiazepine toxicity management. (See first five entries in ''Emergency Department Care and Disposition,'' Barbiturates.)
- Flumazenil, a benzodiazepine antagonist, is *not* indicated for empiric administration in poisoned patients. There is a risk of seizures in mixed ingestions, especially involving tricyclic antidepressants, and in patients chronically on benzodiazepines or with underlying seizure disorders.[9] Flumazenil is also contraindicated in suspected elevated intracranial pressure or head injury.
- Flumazenil is primarily used to reverse the effects of benzodiazepines administered for diagnostic or therapeutic purposes (e.g., conscious sedation for procedures). Due to its short half-life (approximately 1 h), it is mainly effective with short-acting agents such as midazolam.
- Flumazenil is administered 0.2 mg IV every minute to response or a total dose of 3 mg.
- There is no role for forced diuresis or urinary alkalinization.

NONBENZODIAZEPINE SEDATIVE-HYPNOTICS

EPIDEMIOLOGY

- Despite rare clinical use, drugs such as ethchlorvynol, meprobamate, glutethimide, and methaqualone continue to be reported in toxic exposures. Newer drugs such as buspirone and zolpidem are prescribed commonly.
- Gamma-hydroxybutyrate (GHB) has no legitimate clinical use in the United States. Abroad, it is used as an anesthetic and in the treatment of narcolepsy and substance withdrawal. Gamma-hydroxybutyrate abuse is increasing, with 20 reported emergency department (ED) visits in 1992 and 629 in 1996. The majority of visits involved

males age 18 to 25 years. Gamma-hydroxybutyrate has also been implicated in substance-induced rape.[10, 11]

PATHOPHYSIOLOGY

- The nonbenzodiazepine sedative-hypnotics tend to be highly lipophilic and concentrate in the CNS, causing varying degrees of CNS depression.

CLINICAL FEATURES

Gamma-Hydroxybutyrate

- Effects are dose-dependent and range from euphoria, nystagmus, ataxia, and dizziness, to coma, respiratory depression, apnea, seizure-like activity, and bradycardia.
- Gamma-Hydroxybutyrate intoxication may produce sudden onset of aggressive behavior followed by drowsiness, dizziness, euphoria, or coma, with rapid reawakening and amnesia.

Chloral Hydrate

- Toxic doses produce severe CNS, respiratory, and cardiovascular depression.
- Resistant ventricular dysrhythmias are the leading cause of mortality.[12]
- Clues to ingestion include a combination of pear-like breath odor, hypotension, and dysrhythmias.
- Chloral hydrate is a gastrointestinal (GI) irritant, and overdose may be associated with GI bleeding.
- Chloral hydrate is radiopaque and abdominal radiographs may be useful in diagnosis.

Ethchlorvynol

- Central nervous system effects of overdose include nystagmus, lethargy, and prolonged coma.
- Hypothermia, hypotension, bradycardia, and noncardiogenic pulmonary edema may occur.
- A distinct vinyl-like breath odor may be detected.

Glutethimide

- Clinical manifestations of overdose are similar to barbiturate toxicity, except for the presence of prominent anticholinergic findings and a fluctuating, prolonged coma.[13]

Meprobamate

- Central nervous system manifestations of toxicity are similar to other sedative-hypnotics.[14]
- Hypotension is common in serious overdose.
- Seizures, cardiac dysrhythmias, and pulmonary edema have been reported.
- Prolonged fluctuating coma may occur secondary to continued absorption from GI concretions of the drug.
- Abstinence syndromes in individuals physically dependent on meprobamate can be severe and usually occur within 1 to 2 days of drug discontinuation.

Methaqualone

- Central nervous system, respiratory, and cardiovascular effects are similar to other sedative-hypnotics.
- Unlike other sedative-hypnotics, it causes hypertonicity, clonus, hyperreflexia, and muscle twitching.
- Methaqualone often impairs judgment and impulse control, increasing risk of trauma.[15]

Buspirone

- Buspirone is unrelated to the other sedative-hypnotics and does not appear to be addictive.[16]
- Overdoses of up to 3 g (150 times the average anxiolytic dose) have produced no lasting ill effects.
- Overdose produces drowsiness and dysphoria. Rare findings include hypotension, bradycardia, seizures, GI upset, dystonia, and priapism.
- Hypertension may be seen if buspirone is coadministered with monoamine oxidase inhibitors.

Zolpidem

- Zolpidem is used in the treatment of insomnia.
- Overdose may produce drowsiness, vomiting, and rarely coma and respiratory depression.[17]
- Flumazenil may reverse some of the effects of zolpidem. However, its use is not recommended in most overdose situations for the reasons outlined in the section on benzodiazepines.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. As with barbiturates and benzodiazepines, many of the same initial principles apply for nonbenzodiazepine sedative-hypnotic toxicity management. (See first five entries of ''Emergency Department Care and Disposition,'' Barbiturates.)
- Treatment for nonbenzodiazepine sedative-hypnotic toxicity is primarily supportive.

- Chloral hydrate-induced dysrhythmias may respond to lidocaine or β-blockers. Overdrive pacing may be necessary for ventricular tachycardia.
- β-adrenergic agents (epinephrine, isoproterenol, and dopamine) may worsen dysrhythmias. If a pressor is needed to treat hypotension, an α-acting agent such as norepinephrine should be used.
- Meprobamate tends to form GI concretions. Whole-bowel irrigation using 2 L/h polyethylene glycol (40 mL/kg/h in children) until rectal effluent is clear may be beneficial.
- Forced diuresis is not useful in nonbenzodiazepine sedative-hypnotic poisoning due to limited renal excretion.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 157, "Barbiturates," by Raquel M. Schears; Chap. 158, "Benzodiazepines," by George M. Bosse; and Chap. 159, "Nonbenzodiazepine Sedative-Hypnotics," by Raqueal M. Schears.

102 ALCOHOLS

Michael P. Kefer

- An understanding of the osmolal gap is important in discussing the toxicity of the common alcohols. The presence of an osmolal gap suggests the presence of a low-molecular-weight substance such as ethanol, isopropanol, methanol, or ethylene glycol.
- The osmolal gap = osm measured − osm calculated

normal osm gap < 10 mosm/L
osm measured = laboratory determination by freezing point depression
osm calculated = 2 (Na) + BUN/2.8 + glucose/18

ETHANOL

EPIDEMIOLOGY

- Ethanol is the most frequently used and abused intoxicant in the United States.
- Distilled spirits have ethanol volumes of 40 to 50% (80 to 100 proof), wines 10 to 20%, and beers 2 to 6%.
- Although ethanol may cause death directly from respiratory depression, morbidity and mortality are usually related to unanticipated injury from impaired cognitive function.

PATHOPHYSIOLOGY

- Ethanol is a central nervous system (CNS) depressant.
- The major site of absorption is in the proximal small bowel.
- Ethanol is metabolized in the liver by alcohol dehydrogenase, with a small portion excreted in the lungs and urine.
- On average, nondrinkers eliminate ethanol from the blood at a rate of 20 mg/dL/h and chronic drinkers at 30 mg/dL/h.

CLINICAL FEATURES

- Signs and symptoms of ethanol intoxication include slurred speech, disinhibited behavior, CNS depression, and altered coordination. A reflex tachycardia may be seen.
- Legally, a blood ethanol level of 80 to 100 mg/dL defines intoxication for operation of motor vehicles. Clinically, the level correlates poorly because of the development of tolerance. A level of 400 mg/dL may be lethal in the nondrinker, but in the alcoholic there may not even be signs of intoxication.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on clinical presentation with confirmation of ethanol intake, usually by obtaining a blood ethanol level.
- The differential diagnosis is wide when one considers other drugs that cause CNS depression such as benzodiazepines, barbiturates, narcotics, and other alcohols. Head injury or hypoglycemia may manifest identical to, or be clouded by, ethanol intoxication.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- A fingerstick glucose test should be obtained to exclude hypoglycemia. If intravenous (IV) fluids are administered, it should contain 5% dextrose, as these patients are often glycogen depleted.
- Thiamine should be administered.
- The patient should be observed until clinically sober. Any lack of improvement should be considered secondary to causes other than ethanol and managed accordingly.

ISOPROPANOL

EPIDEMIOLOGY

- Isopropanol is commonly found in rubbing alcohol, solvents, skin and hair products, paint thinners, and antifreeze. Its CNS depressant effects are double the potency and duration of ethanol.

PATHOPHYSIOLOGY

- Most isopropanol is absorbed within 30 min of oral ingestion.
- Isopropanol is metabolized in the liver to acetone. Acetone is further metabolized to acetate and formate, but not to a degree as to cause a significant metabolic acidosis.

CLINICAL FEATURES

- Isopropanol intoxication manifests similar to ethanol intoxication except the duration is longer and the CNS depressant effects are more profound.
- The smell of rubbing alcohol may be noted on the patient's breath.
- Severe poisoning is marked by the early onset of coma, respiratory depression, and hypotension. Hemorrhagic gastritis is a characteristic finding, which may cause nausea, vomiting, abdominal pain, and upper gastrointestinal (GI) bleeding. Less common complications include hepatic dysfunction, acute tubular necrosis, and rhabdomyolysis.
- Laboratory investigation reveals elevated ketones (from acetone) in the blood and urine, normal glucose levels, and an absent or minimal metabolic acidosis from acetate and formate formation. The osmolal gap is elevated.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on clinical presentation and laboratory findings of ketonemia and ketonuria and an osmolal gap with or without a minimal anion-gap metabolic acidosis. An elevated isopropanol level helps confirm the diagnosis.
- Isopropanol intoxication is characteristically distinguished from that of the other common alcohols by the presence of an elevated osmolal gap without a significant anion-gap metabolic acidosis and a negative ethanol level.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- General supportive measures should be instituted, which include IV fluids, evaluation of fingerstick glucose, and administration of thiamine. Administration of naloxone should be considered if the patient has an altered mental status. Patients with severe hemorrhagic gastritis may require blood transfusion.
- Laboratory evaluation includes measurement of serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose, isopropanol, and acetone.
- While charcoal does not bind alcohols, it should be administered if there is suspected coingestion of an adsorbable substance.
- Hemodialysis is indicated for refractory hypotension or when the predicted peak level of isopropanol is >400 mg/dL.
- Patients with mild ingestion who are asymptomatic after 6 to 8 h of observation can be discharged from the emergency department (ED).

METHANOL

EPIDEMIOLOGY

- Methanol is commonly found as a solvent in paint products, windshield wiper fluids, and antifreeze.

PATHOPHYSIOLOGY

- Methanol is metabolized in the liver by alcohol dehydrogenase to the toxic compounds formaldehyde and formic acid. Formic acid is converted to carbon dioxide in the presence of folate.
- Methanol accumulation results in a large osmolal gap.
- Formaldehyde accumulation in the retina causes edema and optic papillitis.
- Formic acid accumulation results in a high anion-gap metabolic acidosis.
- Methanol is a GI irritant and may cause pancreatitis.

CLINICAL FEATURES

- Symptoms may not appear for 12 to 18 h after ingestion, as the toxic metabolites must accumulate. The time of symptom onset is further delayed in the presence of ethanol, which inhibits methanol metabolism.
- Typical symptoms include CNS depression, visual disturbances (classically, the patient complains of looking at a snowstorm), nausea, vomiting, and abdominal pain.
- Typical signs are those of CNS depression, retinal edema, hyperemia of the optic disk, and abdominal tenderness.
- Laboratory evaluation reveals a high anion-gap metabolic acidosis with a high osmolal gap.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on the clinical presentation and laboratory findings of a high anion-gap metabolic acidosis. An elevated osmolal gap or methanol level helps confirm the diagnosis.
- The differential diagnosis includes others causes of an anion-gap metabolic acidosis recalled by the acronym MUDPILES (see Table 127-1).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment is based on preventing formation of the toxic metabolites and removing them from the body.
- General supportive measures should be instituted, which include IV fluids, evaluation of fingerstick glucose, and administration of thiamine. Administration of naloxone should be considered if the patient has an altered mental status.
- Laboratory evaluation includes measurement of serum electrolytes, BUN, creatinine, and glucose. If a serum methanol level can be directly measured, a serum osmolality is of little value.
- Charcoal does not bind the alcohols. It is administered if coingestion of an adsorbable substance is suspected.
- Fomepazole or ethanol is used to inhibit alcohol dehydrogenase. Fomepazole has 8000 times greater affinity for alcohol dehydrogenase, with fewer side effects compared to ethanol. Indications for initiating fomepazole or ethanol treatment include suspected methanol poisoning; the presence of an anion-gap metabolic acidosis and an osmolal gap; a methanol level >20 mg/dL; or any patient requiring dialysis.
- The dose of fomepazole is 15 mg/kg IV load followed by 20 mg/kg every 12 h for 4 doses.
- The treating dose of ethanol is 0.6 g/kg IV load followed by 0.11 g/kg/h continuous infusion, or 0.15 g/kg/h in the heavy drinker. The continuous infusion is adjusted accordingly to keep the blood

ethanol level at 100 to 150 mg/dL. If necessary, oral treatment with alcoholic beverages can be initiated. The amount of ethanol contained in these is calculated by grams ethanol = mL beverage × 0.9 × (proof/200). Ethanol treatment is continued until the methanol level is 0 and acidosis has resolved.
- Dialysis eliminates methanol and its toxic metabolites. Indications for dialysis are signs or symptoms of significant toxicity; methanol level >20 mg/dL; or the presence of an anion-gap metabolic acidosis. Peritoneal dialysis is considered only when hemodialysis is not available. Ethanol is dialyzable so the continuous infusion rate is doubled initially and readjusted accordingly, for a target level of 100 to 150 mg/dL.
- Vitamin therapy with folate 50 mg IV every 4 h is administered to drive the conversion of formic acid to carbon dioxide.
- Any asymptomatic patient with a history of significant ingestion should be admitted and treatment initiated because, as noted earlier, it may be 12 to 18 h before significant levels of toxic metabolites develop. Patients should be admitted to a facility capable of providing intensive care unit observation and hemodialysis.

ETHYLENE GLYCOL

EPIDEMIOLOGY

- Ethylene glycol is commonly used as an antifreeze and preservative and is found in polishes and detergents.

PATHOPHYSIOLOGY

- Ethylene glycol is metabolized in the liver by alcohol dehydrogenase to the toxic compound glycoaldehyde. This is further metabolized in the liver and kidneys to formic acid, glyoxylic acid, and oxalic acid. In the presence of thiamine or pyridoxine, glyoxylic acid is converted to nontoxic metabolites.
- Ethylene glycol accumulation results in a large osmolal gap.
- Acid metabolite accumulation results in a high anion-gap metabolic acidosis.
- Oxalic acid precipitates with calcium to form calcium oxalate crystals. These are found in the urine.

CLINICAL FEATURES

- Ethylene glycol poisoning often exhibits three distinct, clinical phases after ingestion due to formation of the toxic metabolites:
  1 to 12 h: CNS effects predominate. The patient appears drunk without the odor of ethanol on the breath.
  12 to 24 h: Cardiopulmonary effects predominate. Elevated blood pressure, heart rate, and respiratory rate are common. Congestive heart failure, respiratory distress syndrome, and shock may occur.
  24 to 72 h: Renal effects predominate. Flank pain and costovertebral angle tenderness are noted. Acute tubular necrosis with acute renal failure may occur.
- Calcium oxalate crystals precipitate and may result in hypocalcemia severe enough to cause tetany and prolongation of the QT interval.
- The funduscopic exam is normal, which helps to distinguish this from methanol toxicity.
- Laboratory evaluation reveals a high anion-gap metabolic acidosis with a high osmolal gap.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on the clinical presentation and laboratory findings of a high anion-gap metabolic acidosis. An elevated ethylene glycol level helps confirm the diagnosis.
- The differential diagnosis includes other causes of an anion-gap metabolic acidosis recalled by the acronym MUDPILES (see Table 127-1).
- Ethylene glycol poisoning differs from methanol poisoning in that visual disturbances and funduscopic abnormalities are absent and calcium oxalate crystalluria is present.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- For ethylene glycol, ED care and disposition is identical to that of methanol except for vitamin therapy. Instead of folate, pyridoxine 100 mg and thiamine 100 mg intramuscularly (IM) or IV should be administered; this drives the conversion of ethylene glycol to nontoxic metabolites.
- Intravenous fomepazole or ethanol treatment and dialysis are indicated for an ethylene glycol level >20 mg/dL.
- Calcium replacement may be necessary.
- Laboratory evaluation also should include a uri-

nalysis (to search for calcium oxalate crystals) and a serum calcium level. If a serum ethylene glycol level can be directly measured, a serum osmolality is of little value.

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103 DRUGS OF ABUSE

Joseph J. Randolph

OPIOIDS

EPIDEMIOLOGY

- The most commonly abused opioids are heroin and methadone.
- From 1990 to 1995, heroin-related emergency department (ED) visits more than doubled.

PATHOPHYSIOLOGY

- Opioids are agonists at the mu_1, mu_2, kappa, delta, sigma, and epsilon receptors in the central nervous system (CNS) and gastrointestinal (GI) tract. Specific binding within the locus ceruleus activates the ''pleasure pathway'' and results in dependence and craving.
- The metabolism of codeine, morphine, propoxyphene, oxycodone, meperidine, and methadone is mostly hepatic.

CLINICAL FEATURES

- Clinically, the classic triad of opioid toxicity is miosis, respiratory depression, and depressed mental status. Other features may include needle track marks, hypotension, ileus, nausea, vomiting, orthostatic hypotension, urinary retention, and noncardiogenic pulmonary edema.
- Histamine release results in urticaria and bronchospasm.
- Normal or even dilated pupils may be seen with meperidine, morphine, propoxyphene, pentazocine, and diphenoxylate.
- Meperidine may cause seizures that are unresponsive to naloxone therapy.
- The combination of meperidine or dextromethorphan with monoamine oxidase inhibitors can precipitate serotonin syndrome (hyperthermia, confusion, muscular rigidity, and hypotension or hypertension).
- Opioid withdrawal is characterized by flulike symptoms (myalgias, lacrimation, piloerection, yawning, abdominal cramps, vomiting, and diarrhea), anxiety, and insomnia.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of opioid overdose or withdrawal is clinical.
- Detection of opioids in the urine may aid diagnosis, but the results have a high false-negative rate and are not rapidly available.
- The differential diagnosis of opioid overdose includes toxic or depressant effects of clonidine, organophosphates and carbamates, phenothiazines, sedative-hypnotic agents, carbon monoxide, and gamma-hydroxybutyrate (GHB). Hypoglycemia, hypoxia, CNS infections, post-ictal states, and pontine hemorrhage also should be considered.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- If respiratory or mental status depression is present, a naloxone test dose of 0.2 to 0.5 mg intrave-

nously (IV), intramuscularly (IM), or per endotracheal tube should be administered, followed by doses of 2 mg IV, as needed. Fentanyl and propoxyphene require larger doses of naloxone for reversal effects.
- The half-life of naloxone is 1 h.
- A naloxone IV drip may be given if a longer-acting opiate has been ingested. To calculate the infusion and dose, the initial bolus that was successful in reversing respiratory depression should be established. Approximately two-thirds of that dose per hour should be administered by IV infusion.
- Noncardiac pulmonary edema may occur up to 24 h after serious heroin and methadone overdoses. Treatment is supportive, with oxygen and positive end-expiratory pressure.
- Criteria for admission include initial respiratory arrest, pulmonary edema, persistent respiratory depression, persistent hypotension, or ingestion of long-acting drugs (meperidine, methadone, and propoxyphene). Most overdoses can be managed by observation for 4 to 8 h.
- Treatment of opioid withdrawal is supportive. Clonidine 0.1 to 0.2 mg PO can be administered.

STIMULANTS, COCAINE, AND AMPHETAMINES

EPIDEMIOLOGY

- Cocaine use is highest among persons aged 18 to 25 years.
- Fatal injuries following cocaine use are a leading cause of death among young adults in New York City and in many other urban environments as well.

PATHOPHYSIOLOGY

- The water-soluble hydrochloride salt of cocaine is absorbed across all mucosal surfaces; thus, cocaine can be applied topically, swallowed, or injected intravenously. Both IV and inhalational routes produce a peak effect within 30 s to 2 min, with a duration of 15 to 30 min.
- Cocaine has been demonstrated to have quinidine-like effects on conduction, causing QRS widening and QTc prolongation. Thus, in large doses, cocaine may exert a direct toxic effect on myocardium, resulting in negative inotropy, wide complex dysrhythmia, bradycardia, and hypotension.
- Central effects are mediated through activation of the sympathetic nervous system. Sympathetic activation produces the characteristic findings of mydriasis, tachycardia, hypertension, and diaphoresis, which predisposes the user to dysrhythmias, seizures, and hyperthermia.

CLINICAL FEATURES

COCAINE
- Common presenting symptoms of cocaine toxicity include chest pain, abdominal pain, palpitations, shortness of breath, headache, paranoia, mania, agitation, and coma.
- Complications of cocaine use include myocardial, mesenteric, spinal cord, or cerebral ischemia; myocarditis; aortic dissection; seizures; stroke; pneumomediastinum; and rhabdomyolysis.
- Central nervous system complications of cocaine use include seizures, intracranial infarction or hemorrhage, spinal cord infarction, and cerebral vasculitis.
- Pulmonary complications include pulmonary hemorrhage, pneumothorax, pneumonitis, asthma, and pulmonary edema.
- Gastrointestinal complications include intestinal schema, bowel necrosis, ischemic colitis, splenic infarction, GI bleeding, and perforation.
- Renal complications include rhabdomyolysis with subsequent acute renal failure (ARF) and renal infarction.
- In pregnancy, cocaine use leads to spontaneous abortions, placental abruption, and fetal prematurity.

AMPHETAMINES
- Amphetamines include methamphetamine (speed), MDMA (ecstasy), methcathinone (ice), and the over-the-counter medications phenylpropanolamine and pseudoephedrine.
- Amphetamine intoxication presents with tachycardia, mydriasis, hypertension, hyperthermia, anxiety, restlessness, repetitive behavior, and paranoid psychosis.
- Complications of amphetamine toxicity include seizures and malignant hyperthermia.
- Withdrawal symptoms include lethargy, depression, increased appetite, abdominal cramps, diarrhea, headache, and chills.

DIAGNOSIS AND DIFFERENTIAL

- Vital signs assist in identifying patients intoxicated with cocaine or amphetamines. Adrenergic stimu-

lation leads to tachycardia, tachypnea, hypertension, and hyperthermia.
- In the absence of an adequate history, it may be difficult to distinguish this presentation from other conditions of catecholamine excess such as withdrawal from alcohol or sedative-hypnotics.
- Occult trauma and hypoglycemia must be excluded. Concomitant alcohol or drug use may alter the clinical presentation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of cocaine and amphetamine toxicity is similar. Patients need quiet rooms with dim lighting, continuous cardiac monitoring, IV access, and lorazepam 1 to 2 mg IV or diazepam 2.5 to 5 mg IV, titrated for sedation.
- The treatment of choice for severe hypertension is benzodiazepines; if ineffective, nitroprusside 0.5 to 10 μg/kg/min IV or phentolamine 5 mg IV should be administered.
- Beta blockers should not be administered because they can result in worsening hypertension secondary to unopposed alpha stimulation.
- Patients with seizures, hyperthermia, or suspected myocardial ischemia should be treated in the standard fashion.
- Admission criteria include unstable vital signs, toxic appearance, mental status changes, persistent chest pain, and electrocardiac (ECG) changes consistent with ischemia or intractable seizure activity.
- Body stuffers are patients who ingest a small amount of packaged drugs in order to destroy evidence. They require treatment with activated charcoal 1 g/kg and observation for 6 to 8 h.
- Body packers are individuals who ingest large amounts of well-packed drugs for smuggling. They require whole-bowel irrigation with PEG or GOLYTELY and admission until the packets have

TABLE 103-1 Characteristics of Hallucinogens

DRUG	CHEMICAL CLASSIFICATION	MECHANISM OF ACTION	TYPICAL DOSE	DURATION OF ACTION	CLINICAL FEATURES	COMPLICATIONS	SPECIFIC TREATMENT
LSD	Indole Alkylamine	5-HT_2 agonist	50–300 μg	8–12 h	Mydriasis, Sympathomimetic symptoms, Nausea, Muscle tension	Persistent psychosis, Hallucinogen-persisting perception disorder	Supportive, Benzodiazepines
Psilocybin	Indole Alkylamine	5-HT_2 agonist	5–100 mushrooms, 4–6 mg of psilocybin	4–6 h	Mydriasis, Sympathomimetic symptoms, Nausea	Seizures (rare), Hyperthermia (rare)	Supportive, Benzodiazepines
Mescaline	Phenyl-ethylamine	5-HT_2 agonist	3–12 "buttons," 200–500 mg of mescaline	6–12 h	Mydriasis, Abdominal pain, Vomiting, Dizziness, Sympathomimetic symptoms	Rare	Supportive, Benzodiazepines
MDMA ("Ectasy")	Phenyl-ethylamine	5-HT release	50–200 mg	4–6 h	Mydriasis, Sympathomimetic symptoms, Bruxism, Jaw tension, Ataxia	Dysrhythmias, Hypertension, Seizures, Hyperthermia, Rhabdomyolysis, DIC	Benzodiazepines, Hydration, Active cooling, ?Dantrolene,* ?5-HT antagonists†
Phencyclidine (PCP)	Piperidine Derivative	Glutamate agonist at NMDA receptor	1–9 mg	4–6 h	Miosis or midsize pupils, Nystagmus, Hypertension, Sympathomimetic, Anticholinergic, and Cholinergic symptoms	Coma, Seizures, Hyperthermia, Rhabdomyolysis, Hypertension, Hypoglycemia	Benzodiazepines, Hydration, Multiple-doses of activated charcoal, Active cooling, ?Dantrolene,* ?Alkalinize urine‡
Marijuana	Cannabinoid	Binds Cannabinoid receptor	5–15 mg of THC	2–4 h	Tachycardia, Conjunctival injection	Rare	Supportive, Benzodiazepines

* Dantrolene possibly indicated for severe hyperthermia.
† Experimental treatment.
‡ Urinary alkalinization for rhabdomyolysis.

passed. Signs of toxicity or bowel obstruction require surgical consultation.

HALLUCINOGENS

EPIDEMIOLOGY

- The ''classical'' hallucinogens include agents from the indole alkylamine (LSD, psilocybin) and phenylethylamine (mescaline) chemical families.
- In addition to these hallucinogens, MDMA (ecstasy), phencyclidine (PCP), anticholinergics, and marijuana possess the ability to alter sensory perceptions and may be placed in the broader category of hallucinogens.

CLINICAL FEATURES AND DIAGNOSIS AND DIFFERENTIAL

- Table 103-1 details the duration of action, clinical features, and complications for the various hallucinogens.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment for all hallucinogens is primarily supportive, with quiet surroundings and benzodiazepines as needed for agitation (Table 103-1). Droperidol 2.5 to 5.0 mg IV may be given for extreme agitation.
- Significant dysrhythmias should be treated using standard protocols.
- Severe hypertension may be treated with nitroprusside. The dose of nitroprusside starts at 0.5 μg/kg/min and can be titrated to a maximum of 10 μg/kg/min, as needed.
- Treatment of severe hyperthermia may require neuromuscular paralysis and endotracheal intubation.
- Multiple-dose activated charcoal every 6 h is recommended for acute PCP intoxication.
- Admission criteria include hyperthermia, severe hypertension, cardiovascular instability, seizures, rhabdomyolysis, or metabolic abnormalities.
- Asymptomatic patients may be discharged after an observation period. Patients with a reliable history of mild to moderate ingestions should be observed in the ED for 4 to 8 h.
- Patients with persistent psychotic symptoms require psychiatric evaluation or admission.

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104 ANALGESICS

Keith L. Mausner

SALICYLATES

EPIDEMIOLOGY

- In 1996, aspirin was involved in 25,281 toxic exposures, with over 12,000 treated at a health care facility. There were 48 deaths (0.2 percent of cases) attributed to salicylate toxicity.[1]
- Many over-the-counter medications [e.g., Pepto Bismol, oil of wintergreen (methyl salicylate), liniments used in vaporizers] contain large amounts of salicylates, and ingestion or application can lead to inadvertent salicylate toxicity.

PATHOPHYSIOLOGY

- Absorption of salicylate may be delayed or erratic. Peak serum levels may be significantly delayed, but toxic levels are usually apparent within 6 h. Peak levels from ingestion of enteric-coated or sustained-release aspirin have been reported from 10 to 60 h after ingestion.[2]
- Aspirin may form a gelatinous gastric mass, and large amounts of aspirin may remain in the stomach long after an overdose. In addition, aspirin has an inhibitory effect on gastric emptying.
- After absorption, aspirin is hydrolyzed to salicylate; toxicity depends on cellular salicylate concentration. At physiologic pH (7.40), essentially all salicylate molecules are ionized. A decrease in pH (acidosis) increases the proportion of nonionized salicylate. Nonionized salicylate molecules cross cell membranes, including the blood-brain barrier. Therefore, acidemia increases intracellular salicylate concentration. Mortality from salicylate toxicity correlates directly with brain salicylate concentration.[3]
- A urinary pH above 8.0 will ionize the salicylate in the urine and impair reabsorption of salicylate across the urinary tubules, resulting in enhanced urinary elimination.[4]
- Physiologic effects of salicylate include the following (*a*) Respiratory alkalosis is produced by initially increased respiratory rate through a direct effect on the medullary respiratory center.[5] (*b*) There is an increased catabolism and elevated carbon dioxide and heat production, increased glycolysis and demand for glucose, and production of organic acids including lactate, pyruvate, and ketoacids, leading to metabolic acidosis. (*c*) Salicylate toxicity produces a mixed acid-base disturbance: respiratory alkalosis, metabolic alkalosis (from volume contraction), and elevated anion-gap metabolic acidosis. (*d*) Salicylate affects central and peripheral glucose metabolism. Normoglycemia, hyperglycemia, or hypoglycemia may be seen in salicylate toxicity. Hypoglycemia in brain cells may occur despite normal serum glucose levels. (*e*) Salicylate's molecular structure is similar to that of vitamin K; large chronic doses may competitively inhibit vitamin K and cause hypoprothrombinemia.

CLINICAL FEATURES

- Clinical manifestations of toxicity depend on the dose, whether exposure is acute or chronic, and the patient's age.
- Acute ingestion of less than 150 mg/kg usually produces mild toxicity with nausea, vomiting, and gastrointestinal (GI) irritation.
- Acute ingestion of 150 to 300 mg/kg usually results in moderate toxicity with vomiting, hyperventilation, sweating, and tinnitus. In adults this typically corresponds to a salicylate level around 30 mg/dL.
- There is usually a mixed acid-base disturbance as noted earlier. However, coingestion of sedative drugs may impair the respiratory drive and result in respiratory acidosis.
- Ingestion of more than 300 mg/kg usually produces severe toxicity. Manifestations of severe toxicity include fever, neurologic dysfunction, renal failure, pulmonary edema, and adult respiratory distress syndrome (ARDS). Rarely, rhabdomyolysis, gastric perforation, and GI hemorrhage occur.
- Fatality is more likely with advanced age. Unconsciousness, fever, severe acidosis, seizures, and dysrhythmias are also associated with increased risk of mortality.
- In children, acute salicylate overdoses usually present within a few hours of ingestion. Children under age 4 tend to develop metabolic acidosis ($pH < 7.38$), whereas children over age 4 usually have mixed acid-base disturbances as in adults.
- Chronic salicylate toxicity is usually seen in elderly patients with underlying medical problems. It may present with hyperventilation, tremor, papilledema, agitation, paranoia, bizarre behavior, memory loss, confusion, and stupor. Chronic salicylism should be considered in any patient with unexplained nonfocal neurologic and behavioral abnormalities, especially with coexisting acid-base disturbance, tachypnea, or noncardiogenic pulmonary edema.
- In children, chronic (repeat dose) salicylate toxicity has higher morbidity and mortality than does acute toxicity.[6] Symptoms may not appear for several days, and there may be an underlying illness that triggered the salicylate administration. Chronic salicylism may be mistaken for an infectious process. Hyperventilation, volume depletion, acidosis, marked hypokalemia, and central nervous system (CNS) disturbances may be seen. Fever indicates a worse prognosis. Renal failure is a severe complication.

DIAGNOSIS AND DIFFERENTIAL

- Clinical status is the key to diagnosis and treatment.
- Salicylate levels should be interpreted with cau-

tion. Severe toxicity may be present despite a ''therapeutic'' or declining level. Use of the Done nomogram, which was developed to predict toxicity after acute ingestion within a known time frame, may be misleading and is no longer recommended.
- The differential diagnosis of salicylate toxicity includes theophylline toxicity, caffeine overdose, acute iron poisoning, Reye's syndrome, diabetic ketoacidosis, sepsis, and meningitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Cardiac monitoring and an intravenous (IV) line should be instituted.
- Bedside glucose determination is indicated for altered level of consciousness or seizures. Laboratory studies should include electrolytes, glucose, blood urea nitrogen (BUN), creatinine, complete blood cell count (CBC), prothrombin time (PT), salicylate level, acetaminophen level (to rule out coingestion), as well as an arterial blood gas analysis to determine acid-base status.
- Activated charcoal 1 g/kg should be administered to minimize absorption and hasten elimination. Multiple doses are probably not beneficial. Whole-bowel irrigation may be effective when toxicity is due to sustained-release or enteric-coated aspirin.
- Intravenous normal saline should be administered to patients with evidence of volume depletion. Except for the initial resuscitation, all subsequent fluids should contain at least 5% dextrose.
- Alkalinization of serum and urine enhances salicylate protein binding and urinary elimination. This may be accomplished with a second IV line concurrent with volume resuscitation. A bolus of 1 to 2 meq/kg of sodium bicarbonate should be administered. Then, 100 to 150 meq (2 to 3 ampules) of sodium bicarbonate should be added to 1 L D_5W and infused at 1.5 to 2.0 times the patient's maintenance rate. The infusion should be adjusted to maintain urine pH above 7.5. After urine output is established, 40 meq/L of potassium should be administered. Serum electrolyte levels and volume status should be monitored. Alkalinization decreases the serum potassium level. In addition, alkalinization cannot be maintained unless the serum potassium level is at least 4.0 meq/L.
- Hemodialysis is indicated for clinical deterioration despite supportive care and alkalinization, renal insufficiency or failure, severe acid-base disturbance, altered mental status, or ARDS.
- Hemorrhage, due to elevated PT, should be treated with fresh-frozen plasma and vitamin K.
- In significant ingestions, frequent clinical exams are indicated along with salicylate levels every 2 h until the peak occurs, then every 4 to 6 h until the level is nontoxic. In severe ingestions, hourly levels correlated with clinical status are indicated.
- Except with ingestion of enteric-coated or sustained-release formulations, patients may be discharged from the emergency department (ED) if there is progressive clinical improvement, no significant acid-base abnormality, and a decline in serial salicylate levels toward the therapeutic range.
- In intentional overdoses, psychiatric consultation or admission is indicated.
- In potentially large ingestions of enteric-coated or sustained-release salicylates, the patient should be admitted and observed for at least 24 h to ensure declining serial salicylate levels and improving clinical status.

ACETAMINOPHEN

EPIDEMIOLOGY

- Acetaminophen is the most popular over-the-counter analgesic in the United States. In 1996, acetaminophen accounted for 5 percent of all toxic exposures and for 11 percent of reported fatalities.[1]

PATHOPHYSIOLOGY

- Acetaminophen is rapidly absorbed from the GI tract. In overdose, peak serum levels usually occur within 2 h. However, delayed absorption may occur with acetaminophen-propoxyphene preparations and with a new preparation, Tylenol Extended Relief (Tylenol ER).
- Acetaminophen is mainly metabolized by the liver through sulfation and glucuronidation; only a small percentage (<5 percent) undergoes direct renal elimination.
- A small percent of acetaminophen is also oxidized by cytochrome P450 to a toxic metabolite, *n*-acetyl-*p*-benzoquinoneimine (NAPQI). NAPQI is detoxified by hepatic glutathione to a nontoxic compound that undergoes renal elimination. In acetaminophen overdose, hepatic glucuronidation and sulfation are quickly saturated, and a higher percentage of acetaminophen is metabolized by cytochrome P450 to NAPQI. When hepatic gluta-

thione stores are depleted to less than 30 percent of normal, NAPQI accumulates, and hepatic toxicity occurs. NAPQI causes hepatocellular injury and typically produces hepatic centrilobular necrosis.
- Patients with low glutathione stores (alcoholics and AIDS patients) and those with induced cytochrome P450 activity (alcoholics and individuals on anticonvulsant or antituberculosis drugs) are at greater risk of developing acetaminophen toxicity.
- *N*-acetylcysteine (NAC) is a specific antidote for acetaminophen. Among other actions, NAC inhibits binding of NAPQI to hepatic proteins, it may act as a glutathione precursor or substitute, and it may directly reduce NAPQI back to acetaminophen.

CLINICAL FEATURES

- Acute acetaminophen toxicity presents in four stages.
- Day 1: no symptoms, or such nonspecific symptoms as anorexia, nausea, vomiting, and malaise, are present.
- Days 2 to 3: nausea and vomiting may improve, but evidence of hepatotoxicity, such as right upper quadrant abdominal pain and tenderness with elevated transaminases and bilirubin, may be present.
- Days 3 to 4: progression to fulminant hepatic failure occurs, with lactic acidosis, coagulopathy, renal failure, and encephalopathy, as well as recurrent nausea and vomiting.
- Survivors of hepatic failure will begin to recover over the following several weeks with complete resolution of hepatic dysfunction.

DIAGNOSIS AND DIFFERENTIAL

- Acetaminophen toxicity may occur with acute ingestion of more than 140 mg/kg, or when more than 7.5 g are ingested by an adult in a 24-h period.
- Diagnosis of a significant ingestion depends on laboratory testing, since symptoms may initially be absent or nonspecific.
- An acetaminophen level should be considered in all patients presenting with any drug overdose, since acetaminophen is a common coingestant.[7]
- In a single large overdose, the Rumack-Matthew nomogram (Fig. 104-1) accurately predicts acetaminophen toxicity based on the serum acetaminophen level measured 4 to 24 h after the estimated time of ingestion.[8] The nomogram is not useful outside of the 4- to 24-h window. A 4-h level greater than 150 μg/dL is usually toxic. After 24 h, a detectable acetaminophen level or the presence of elevated transaminases may predict toxicity.

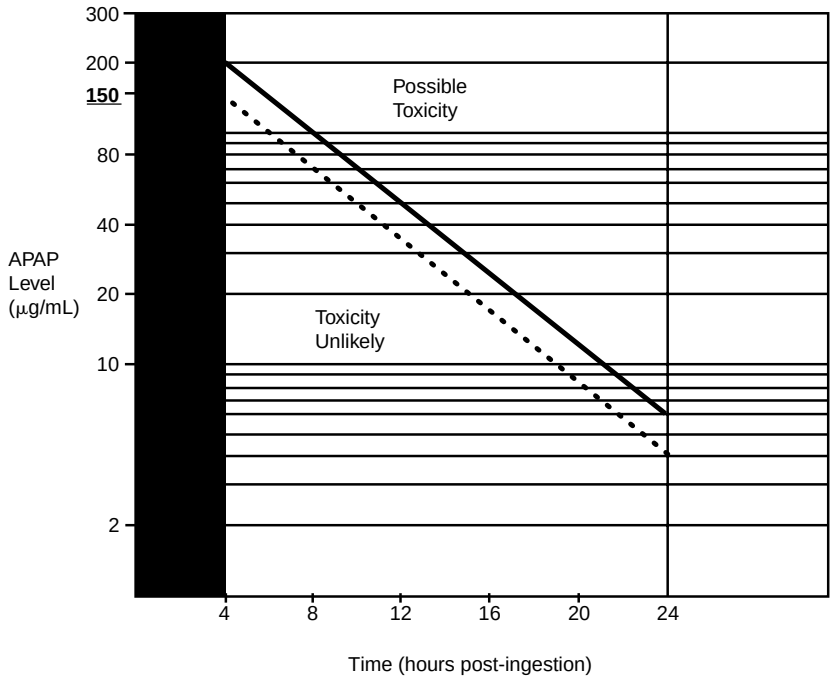

FIG. 104-1 Rumack-Matthew nomogram. *Abbreviation:* APAP, *N*-acetyl-*p*-aminophenol (acetaminophen). (From Rumack BH Matthew H: Acetaminophen poisoning and toxicity. *Pediatrics* 55:871, 1975, with permission.)

- Multiple ingestions over a period of time are problematic. One approach is to assume a single ingestion at the earliest possible point in time and use the Rumack-Matthew nomogram accordingly.
- Clinical experience with Tylenol ER ingestion is limited. Because serum peak levels may be delayed, one approach is to add 2 h to the time from ingestion. Thus, at 4-h postingestion, the nomogram should be interpreted as if it were 6-h postingestion.
- Differential diagnosis of acetaminophen toxicity includes viral and alcoholic hepatitis, other drug- or toxin-induced hepatitides, and hepatobiliary disease.
- Acute acetaminophen poisoning is often distinguished from other forms of hepatitis by its acute onset, rapid progression, and markedly elevated transaminase levels.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation.
- An acetaminophen level, drawn as soon as possible within 4 to 24 h of ingestion, will guide subsequent therapy and disposition. Other laboratory studies, including electrolytes, glucose, BUN, creatinine, transaminase levels, CBC, and PT should be drawn if clinically indicated or if the acetaminophen level falls in the toxic range on the Rumack-Matthew nomogram.
- Activated charcoal 1 g/kg is indicated for GI decontamination and in case of coingestion of other drugs.
- *N*-acetylcysteine (NAC) effectively prevents toxicity if administered within 8 h of ingestion, significantly reduces hepatotoxicity if administered within 24 h of ingestion, and may be of value even after 24 h.[9] If the acetaminophen level is not going to be available within 8 h of ingestion, NAC therapy should be initiated and continued if indicated based on the subsequent acetaminophen level.
- *N*-acetylcysteine is administered orally or by nasogastric tube as a 140-mg/kg loading dose, followed by 70 mg/kg every 4 h for 17 additional doses. It may be administered immediately after activated charcoal. There is no evidence that this decreases NAC's effectiveness, and NAC is safe in pregnancy.
- Nausea and vomiting during NAC therapy may be reduced by diluting NAC in a beverage and by administering antiemetics such as metoclopramide or ondansetron.
- Intravenous NAC therapy is not approved in the United States, but may be used in consultation with a toxicologist for patients unable to tolerate or receive oral therapy.[10]
- Treatment of fulminant hepatic failure includes NAC therapy, correction of coagulopathy and acidosis, treatment of cerebral edema, and early referral to a liver transplant center.
- Patients with nontoxic acetaminophen levels based on the Rumack-Matthew nomogram may be discharged from the ED if there is no evidence of other drug ingestion. In deliberate overdoses, psychiatric evaluation is indicated.
- All patients receiving NAC therapy should be admitted.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

EPIDEMIOLOGY

- Between 1992 and 1996 the American Association of Poison Control Centers[1] reported 26 deaths related to nonsteroidal anti-inflammatory drug (NSAID) overdose, compared with 213 for aspirin and 248 for acetaminophen.
- Nonsteroidal anti-inflammatory drug morbidity at therapeutic doses is far more significant than morbidity from overdoses.[11] In fact, NSAID-related GI bleeding is estimated to lead to 75,000 hospitalizations and 7500 deaths annually in the United States.[12] In addition, NSAIDs have been implicated in a significant proportion of drug-induced renal failure.

PATHOPHYSIOLOGY

- Nonsteroidal anti-inflammatory drugs inhibit the enzyme cyclooxygenase (COX), which produces prostaglandins from arachidonic acid. There are at least two forms of cyclooxygenase, COX I and COX II. COX I is probably responsible for most of the adverse effects of NSAIDs; work is underway to produce NSAIDs that selectively inhibit COX II.
- Nonsteroidal anti-inflammatory drugs are rapidly absorbed from the GI tract. Most NSAIDs undergo at least partial hepatic metabolism before elimination in the urine or feces.
- Plasma half-lives of NSAIDs range from 2 to 4 h

for ibuprofen to longer than 50 h for piroxicam and phenylbutazone.

CLINICAL FEATURES

- Phenylbutazone and naproxen may displace warfarin from plasma proteins, resulting in elevated PTs. Phenylbutazone also decreases the elimination of warfarin.
- Other NSAIDs do not interact in these ways with warfarin, but NSAID use is contraindicated with warfarin because NSAID platelet aggregation inhibition may significantly increase the risk of bleeding.
- The effectiveness of antihypertensives, including diuretics, α-adrenergic blockers, angiotensin-converting enzyme inhibitors, and β-adrenergic blockers may be decreased by NSAIDs.[13]
- The renal clearance of lithium and methotrexate may be inhibited by NSAIDs, and toxicity from these drugs may result.
- Chronic NSAID therapeutic use may produce significant toxicity. The most frequent problems are GI bleeding and renal insufficiency. Central nervous system effects may include headache, mental status changes, and aseptic meningitis.[14] Hepatic dysfunction may occur, especially in the elderly and in patients with autoimmune disease. Inhibition of platelet aggregation may lead to bleeding. Nonsteroidal anti-inflammatory drugs account for 10 percent of cutaneous drug reactions, ranging from benign rashes, to phototoxic reactions, to severe Stevens-Johnson syndrome and toxic epidermal necrolysis.[15,16] Fetal NSAID exposure may cause premature closure of the ductus arteriosus, oligohydramnios, renal dysfunction, necrotizing enterocolitis, and CNS hemorrhage.
- Acute NSAID overdose generally has low morbidity and usually becomes clinically apparent within 4 h of ingestion. Abdominal pain, nausea, and vomiting may occur. Central nervous system effects may include altered mental status, diplopia, nystagmus, headache, and rarely seizure. Renal failure may occur, with serum electrolyte abnormalities and volume overload.

DIAGNOSIS AND DIFFERENTIAL

- Manifestations of NSAID toxicity are nonspecific.
- The NSAID levels are not readily available and are not clinically useful in assessing toxicity.
- Acetaminophen and salicylate levels will exclude coingestion of these agents.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation.
- Laboratory evaluation should include electrolyte and glucose levels, renal and hepatic function, acetaminophen level, salicylate level, and CBC. Bedside glucose determination is indicated for altered mental status or seizures.
- Activated charcoal 1 g/kg is indicated for GI decontamination.
- Volume resuscitation, correction of acid-base and electrolyte disorders, and standard treatment of other complications such as seizures and renal failure should be performed as indicated.
- Patients with asymptomatic NSAID ingestions may be safely discharged from the ED after screening for coingestants and a 4- to 6-h observation period. Psychiatric evaluation is indicated for deliberate overdoses.

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105 XANTHINES

Mark B. Rogers

EPIDEMIOLOGY

- Theophylline and caffeine are the two most common xanthines.
- The elderly with concomitant medical problems are more susceptible to life-threatening toxicity after chronic overmedication than are younger patients after an acute overdose.

PATHOPHYSIOLOGY

- Theophylline's mechanism of action has not been entirely elucidated but includes inhibition of phosphodiesterase and antagonism of adenosine.
- Theophylline's elimination (85 to 90 percent) occurs via the hepatic cytochrome P450 system.
- Theophylline's half-life is increased by smoking cessation, disease states such as cirrhosis, congestive heat failure and chronic obstructive pulmonary disease, and medications such as cimetidine, quinolones, and erythromycin.

CLINICAL FEATURES

- Theophylline toxicity can cause life-threatening cardiac, neurologic, and metabolic abnormalities. Life-threatening effects may occur suddenly and before minor symptoms are evident.
- Caffeine produces many of the same toxic effects as theophylline.
- Cardiac side effects include sinus tachycardia, premature atrial contractions, atrial flutter, and atrial fibrillation. Premature ventricular contractions and ventricular tachycardia are seen particularly in elderly with chronic overdoses and levels at 40 to 60 μg/mL. Younger patients with acute ingestions may tolerate levels above 100 μg/mL.
- Neurologic side effects include agitation, headache, irritability, sleeplessness, tremors, and seizures.
- Metabolic side effects include an increase in catecholamine, glucose, free fatty acid, and insulin levels. Hypokalemia may occur and be exacerbated by β-agonist therapy.
- Gastrointestinal side effects include nausea and vomiting, and gastrointestinal bleeding and epigastric pain may occur.

DIAGNOSIS AND DIFFERENTIAL

- Therapeutic serum theophylline levels of 10 to 20 μg/mL may produce toxic effects.
- Caffeine toxicity is seen after 1-g doses in adults and 80 mg/kg in children.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of xanthine toxicity consists of initial stabilization, gastric decontamination and elimination, treatment of life-threatening toxic effects, and, in severe cases, hemoperfusion or dialysis. Patients should be observed on a cardiac monitor, noninvasive blood pressure device, and pulse oximeter. An intravenous (IV) line should be established.
- Gastric lavage should be considered for acute in-

gestions of toxic doses within 2 to 4 h. To enhance drug elimination, multiple doses of oral activated charcoal (1 g/kg), mixed with a cathartic such as sorbitol, should be administered every 2 to 4 h in the first 24 h.
- Ranitidine (50 mg IV) is useful for the nausea and vomiting associated with toxicity.
- Seizure activity can be treated with diazepam, phenobarbital, or phenytoin.
- Hypotension initially should be treated with IV isotonic crystalloid. In patients unresponsive to IV fluids and who have life-threatening dysrhythmias, medications with a β-blocker effect, such as labetalol or esmolol, may be administered cautiously. Diltiazem, lidocaine, and digoxin have been effective. Adenosine for supraventricular tachycardia may induce bronchospasm.
- In the absence of life-threatening effects (e.g., status epilepticus or ventricular dysrhythmias), hemodialysis or hemoperfusion is controversial.
- Patients with seizures or ventricular dysrhythmias should be monitored until levels normalize.
- With mild symptoms or levels below 25 μg/mL, patients do not require specific treatment or admission, but their dosing should be decreased or discontinued.
- Patients with levels above 30 μg/mL should be treated with oral activated charcoal and monitored for toxic side effects.

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106 CARDIAC MEDICATIONS

Joseph J. Randolph

DIGITALIS GLYCOSIDES

EPIDEMIOLOGY

- Digitalis preparations are used most commonly in the treatment of supraventricular tachydysrhythmias and congestive heart failure.
- It is found in plants such as foxglove, oleander, and lily of the valley.

PATHOPHYSIOLOGY

- Digitalis has a narrow therapeutic-toxic margin.
- Digitalis binds to a receptor site on the cardiac cell membrane, inactivating the Na-K adenosine triphosphatase (ATPase) pump and resulting in increased sarcoplasmic calcium and increased extracellular potassium.
- Digitalis increases vagal tone and decreases conduction through the atrioventricular (AV) node.

CLINICAL FEATURES

- Acute overdose leads to symptoms of nausea, vomiting, bradydysrhythmias, supraventricular dysrhythmias with AV block, and ventricular dysrhythmias.
- Chronic toxicity is more common in elderly patients taking diuretics and presents with gastrointestinal (GI) symptoms, weakness, visualization of yellow-green halos around objects, syncope, altered mental status, hallucinations, seizures, and ventricular dysrhythmias.
- Patients at increased risk for chronic toxicity are elderly patients and those with underlying conditions such as chronic obstructive pulmonary disease, heart or renal disease, or hypokalemia.
- Almost any cardiac dysrhythmia may be seen, but ventricular dysrhythmias occur more frequently in chronic poisonings.[1]

DIAGNOSIS AND DIFFERENTIAL

- Hyperkalemia may be seen in acute poisoning. The patient may have normal potassium or hypokalemia in chronic toxicity.

- The differential diagnosis includes overdose of calcium channel blockers, β-blockers, quinidine, procainamide, clonidine, organophosphates, or cardiotoxic plants such as rhododendron or yewberry.
- The therapeutic digoxin level is 0.5 to 2.0 ng/mL. Serum levels are most reliable when obtained 6 h after ingestion. Levels are normal to mildly elevated in chronic toxicity and are markedly elevated in acute toxicity.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Intravenous access and continuous cardiac monitoring should be secured.
- Activated charcoal should be administered at 1 g/kg, then 0.5 g/kg every 4 to 6 h.
- Atropine, 0.5 to 2.0 mg intravenously (IV), should be administered and cardiac pacing instituted for bradydysrhythmias.
- Ventricular dysrhythmias are treated with phenytoin 15 mg/kg IV, infused no faster than 25 mg/min; lidocaine 1 mg/kg IV; or magnesium sulfate 2 to 4 g IV. Cardioversion may induce refractory ventricular dysrhythmias and should be used as a last resort. The initial setting should be 10 to 25 W/s.
- Hyperkalemia is treated with glucose followed by insulin, sodium bicarbonate, potassium-binding resin, or hemodialysis. Calcium chloride should be avoided.

TABLE 106-1 Calculating Digoxin-Specific Fab Fragment Dosage

1. Calculate total body load
   Based on history of amount ingested:
   Total body load = amount ingested (mg) × 0.80 (bioavailability)
   Based on serum digoxin concentration:

$$\text{Total body load} = \frac{\text{serum digoxin level} \times 5.6\ \text{L/kg} \times \text{patient's weight (kg)}}{1000}$$

2. Calculate number of vials of digoxin-specific Fab fragments needed to neutralize the calculated total body load:
   It is assumed that an equimolar dose of Fab fragments is required for neutralization.
   One vial (40 mg) of Fab fragments binds 0.6 mg of digoxin.

$$\text{Number of vials required} = \frac{\text{Total body load}}{0.6}$$

A simple and accurate variation of the above calculations:

$$\text{Number of vials of Fab} = \frac{\text{serum digoxin level} \times \text{patient's weight (kg)}}{100}$$

- Digoxin-specific Fab is indicated for ventricular dysrhythmias, bradydysrhythmias with hypotension, and hyperkalemia greater than 5.5 meq/L (Table 106-1).
- Patients who are asymptomatic after 12 h of observation may be medically cleared. Patients with signs of toxicity or history of large overdose should be admitted to a monitored setting. Patients receiving Fab should be admitted to an intensive care unit.

β-BLOCKERS

EPIDEMIOLOGY

- β-blockers are used in the management of hypertension, tachydysrhythmias, and acute myocardial infarction. The majority of serious cases have resulted from ingestion of propranolol.[2,3]

PATHOPHYSIOLOGY

- β_1 stimulation increases force and rate of myocardial contraction. β_2 stimulation relaxes smooth muscle and stimulates glycogenolysis.
- Excessive β-blockade decreases inotropy and chronotropy.
- Labetalol has the potential to cause profound hypotension due to the combined α- and β-receptor antagonism.

CLINICAL FEATURES

- Toxicity presents with bradycardia, hypotension, congestive heart failure, depressed consciousness, and seizures.
- Systemic toxicity has been reported following instillation of ophthalmologic preparations.[4]
- QRS widening may occur with β-blockers that antagonize sodium channels, such as propranolol and others.
- Sotalol may cause QT prolongation, ventricular tachycardia, torsades de pointes, and ventricular fibrillation.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis includes overdose of calcium-channel blockers, centrally acting α-agonists, digoxin, organophosphates, plants such as

oleander and rhododendron, and Chinese herbal preparations containing cardiac glycosides.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- All patients require supportive care, continuous cardiac monitoring, and IV access.
- Activated charcoal 1 g/kg should be administered.
- Glucagon is a first-line agent, given as a bolus of 50 to 150 μg/kg IV and repeated as necessary or as a continuous infusion at 1 to 10 mg/L.[5,6] If glucagon is unavailable or ineffective, the next choice is a catecholamine.
- Dopamine or norepinephrine is the catecholamine of choice for refractory hypotension.
- Lidocaine, magnesium sulfate, isoproterenol, and overdrive pacing are used to treat sotalol-induced ventricular dysrhythmias.
- Patients who are symptom-free after 8 to 10 h with a normal repeat electrocardiogram (ECG) may be medically cleared. All others require admission to a monitored bed or intensive care unit.

CALCIUM CHANNEL BLOCKERS

Calcium channel blockers consist of phenylalkylamines (verapamil), benzothiapines (diltiazem), and dihydropyridines (nifedipine).

PATHOPHYSIOLOGY

- Calcium channel blockers bind to the calcium channel, causing it to favor the closed state and decreasing calcium entry during phase II repolarization.
- Verapamil is the most potent negative inotrope of all calcium channel blockers, causing more deaths than all other calcium channel blockers combined.[7]
- Sustained-release and second-generation dihydropyridines (nifedipine) can extend the duration of clinical toxicity and can lead to a delay in clinical manifestation of toxicity.[7]

CLINICAL FEATURES

- Cardiac manifestations include sinus bradycardia with hypotension, conduction disturbances, and complete sinus arrest with ventricular escape rhythms.
- Inadequate cerebral perfusion produces dizziness, lethargy, agitation, confusion, seizures, and hemiplegia.
- Other features include generalized weakness, metabolic acidosis with hyperglycemia, noncardiogenic pulmonary edema, hypokalemia, hyperkalemia, and hypercalcemia.
- Severe toxicity is recognized by a slow junctional rhythm, hypoxemia, lactic acidosis, and decreased left ventricular ejection fraction on echocardiography.

DIAGNOSIS AND DIFFERENTIAL

- Severe toxicity is recognized by slow junctional rhythm, hypoxemia, lactic acidosis, and decreased left ventricular ejection fraction on echocardiography.
- An arterial blood gas analysis and electrolytes should be drawn to determine acid-base status.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- All patients require supplemental oxygen, cardiac monitoring, and IV access.
- Activated charcoal 1 g/kg should be administered. Whole-bowel irrigation should be initiated for sustained-release preparations.[8]
- Hypotension should be treated with calcium chloride, starting with 1 g in 100 mL normal saline to run through a central venous line over 5 min, followed by 20 to 50 mg/kg/h.
- Glucagon is used for hypotension resistant to fluids or calcium chloride, starting at a dose of 0.1 mg/kg mixed in normal saline, followed by an infusion of 0.1 mg/kg/h.
- Dopamine, 1 to 20 μg/kg/h, should be used for persistent hypotension.[9]
- Rescue therapies for persistent hypotension include amrinone, 750 μg/kg IV, followed by an infusion of 1 to 20 μg/kg/h; insulin, 1.0 U/kg over the first hour followed by 0.5 U/kg/h, with coadministration of 20 to 30 g/h of glucose to maintain euglycemia; 4-aminopyridine, infused at 10 to 50 μg/kg/h; or cardiac pacing at 45 to 50 beats per minute.[10–15]
- Acidosis should be corrected to maintain arterial pH above 7.20 with hyperventilation or sodium bicarbonate.
- Patients who are asymptomatic with normal vital signs after trivial ingestions can be observed for

6 h and medically cleared. Patients who have taken a sustained-release preparation require longer observation, if without symptoms. All others require admission to a monitored bed or intensive care unit.

ANTIHYPERTENSIVES

PATHOPHYSIOLOGY

(See Table 106-2.)

TABLE 106-2 Specific Antihypertensive Medications

NAME OF DRUG	MECHANISM OF ACTION	THERAPEUTIC RANGE	LD_{50}	DIALYSIS	MAXIMUM TOLERATED EXPOSURE	THERAPEUTIC INTERVENTIONS AND COMMENTS
Hydrochlorothiazide	Inhibits reabsorption of Na^+ and Cl^- in the distal convoluted tubule of the kidney	12.5–100 mg qd (in divided doses)	10 g/kg in mice/rats	Partially	1 g	IV fluids, correct electrolytes, vasopressor (dopamine) if necessary
Furosemide	Decreases reabsorption of Na^+ and Cl^- in the loop of Henle	20–600 mg qd	1000 mg/kg in rats/dogs	No	Not established	IV fluids, correct electrolytes, vasopressor (dopamine) if necessary
Spironolactone	Specific antagonist of aldosterone	25–400 mg qd		Yes	Not established	IV fluids, correct electrolytes
Triamterene	Inhibits the reabsorption of sodium ions in exchange for potassium and hydrogen ions at the distal tubule	100–150 mg bid	350 mg/kg mice	Yes	Not established	IV fluids, correct electrolytes, vasopressor (dopamine) if necessary
Acetazolamide	Inhibits carbonic anhydrase in the kidney	Up to 100 mg qd	No deaths reported	Possibly	Not established	IV fluids, correct electrolytes, pH, vasopressor (dopamine) if necessary
Mannitol	Osmotic diuresis	0.5 g/kg to 2 g/kg	Not known	Yes	Not established	IV fluids, correct electrolytes, vasopressor (dopamine) if necessary
Clonidine	Central α_2 agonist	0.1–2.4 mg/day	Oral LD_{50} rats, 465 mg/kg	No	11.25 mg	IV fluids, vasopressors (dopamine), naloxone, Tolazoline
Captopril	Angiotensin-converting enzyme	6.25–150 mg tid	No deaths reported	Yes	7.5 g	IV fluids, vasopressors (dopamine), naloxone
Enalapril	Angiotensin-converting enzyme	5–40 mg qd	Oral LD_{50}, 200 mg/kg in mice/rats	Yes	300 mg	IV fluids, vasopressors (dopamine), naloxone
Methyldopa	Central inhibitory α-adrenergic receptors, false neurotransmission, and/or reduction of plasma renin activity	250–3000 mg qd	Oral LD_{50}, >1.5 g/kg in mice/rats	Yes	Not established	IV fluids, vasopressors (dopamine)
Hydralazine	Directly relaxes arteriolar smooth muscle	10–50 mg qid	Oral LD_{50}, 173 mg/kg in rats	No	Not established	IV fluids; vasopressors should be avoided secondary to dysrhythmias
Minoxidil	Direct peripheral vasodilator	5–100 mg/day	Oral LD_{50}, 1321–3492 mg/kg in rats	Partially	Not established	IV fluids, dopamine; epinephrine and norepinephrine should be avoided
Sodium nitroprusside	Relaxes arteriolar and venous smooth muscle	0.5–10 μg/kg/min	Oral LD_{50}, rabbits and dogs, 2.8 and 5.0 mg/kg	Yes, for thiocyanate toxicity	Unknown	Prolonged use can cause cyanide and/or thiocyanate toxicity; for cyanide toxicity, use cyanide antidote protocol
Prazosin	Arteriolar dilator, competitive blockade of postsynaptic α_1-adrenergic receptors	1 mg bid to 20 mg qd	Not known	No	200 mg	IV fluids, vasopressors (dopamine)

CLINICAL FEATURES

Thiazides and Loop Diuretics

- Toxicity of thiazides and loop diuretics involves two basic processes: volume contraction and electrolyte derangements.
- Symptoms include hypotension, tachycardia, and altered mental status.
- Common electrolyte derangements include hyponatremia, hypokalemia, hypocalcemia, hypomagnesemia, and hypochloremic metabolic alkalosis.

Potassium-Sparing Diuretics

- The most common medications of this subclass are spironolactone, triamterene, and amiloride.
- Volume depletion, hyperkalemia, hyponatremia, and hypochloremia are common manifestations of toxicity.

Carbonic Anhydrase Inhibitors

- An example of this subtype is acetazolamide, a sulfonamide whose adverse reactions include severe allergic reaction and Stevens-Johnson syndrome.
- Overdose with carbonic anhydrase inhibitors leads to volume depletion and electrolyte disturbances, as well as non-anion gap metabolic acidosis.

Osmotic Agents

- Toxicity can result in pulmonary edema, volume depletion, anaphylaxis, and acute renal failure.

Centrally Acting Agents

- Clonidine, guanabenz, and methyldopa are examples of centrally acting antihypertensive agents. Clonidine is also used for mitigation of the effects of opiate and ethanol withdrawal.
- Clonidine toxicity causes hypotension and bradycardia, leading to myocardial ischemia and congestive heart failure.
- Other findings include respiratory depression, hypothermia, mental status changes, seizures, and miosis.
- Guanabenz and methyldopa cause hypotension, bradycardia, and mental status changes.

Angiotensin-Mediated Antihypertensives

- Acute overdose of both angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists cause profound hypotension. Angiotensin II receptor antagonists also cause bradycardia.

Peripheral Vasodilators and Ganglionic Blockers

- These groups include prazosin, hydralazine, minoxidil, and sodium nitroprusside.
- Overdose of prazosin, hydralazine, and minoxidil may cause hypotension and tachycardia.
- Following minoxidil overdose, patients with renal insufficiency may develop fluid retention and pericardial effusion.
- Nitroprusside toxicity presents with hypotension and dysrhythmias.
- Thiocyanate and cyanide toxicity, as well as methemoglobinemia, have been reported after prolonged nitroprusside therapy.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

(See Table 106-2.)

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107 PHENYTOIN AND FOSPHENYTOIN

Mark B. Rogers

EPIDEMIOLOGY

- Most phenytoin-related deaths have been caused by rapid intravenous (IV) administration and hypersensitivity reactions.

PATHOPHYSIOLOGY

- Phenytoin works by blocking sodium channels in neurons.
- Peak levels occur anywhere from 3 to 12 h after a single oral dose.
- Phenytoin is extensively protein-bound (90 percent). The free, unbound form is active. Patients who have hypoalbuminemia (e.g., in cirrhosis, nephrosis, malnutrition, or burns) or take drugs that displace phenytoin from binding sites (e.g., salicylate, valproate, or sulfisoxazole) may exhibit toxic signs while in therapeutic range.
- Toxicity depends upon the duration of exposure, dosage taken, and, most importantly, route of administration.
- Life-threatening cardiovascular side effects, seen only with IV administration and caused by propylene glycol (the diluent in the IV preparation), are commonly related to the infusion rate.
- Fosphenytoin, a prodrug of phenytoin, is more soluble and less irritating to tissues. It is safe to administer intramuscularly (IM). When administered by IV route, it can cause pruritus and hypotension. The adverse and toxic effects of fosphenytoin are the same as phenytoin, except the toxic effects of propylene glycol do not exist.

CLINICAL FEATURES

- An acute oral overdose is typically dose-related and usually presents with nystagmus, nausea, vomiting, ataxia, dysarthria, choreoathetosis, opisthotonos, and central nervous system (CNS) depression or excitation.
- Central nervous system toxicity begins with horizontal nystagmus; however, vertical, bidirectional, or alternating nystagmus may occur with severe toxicity.
- Cardiovascular toxicity includes hypotension, bradycardia, conduction delays, ventricular tachycardia, ventricular fibrillation, and asystole.
- Electrocardiographic changes include increased PR interval, widened QRS interval, and altered ST-wave and T-wave segments.
- Significant soft tissue toxicity from IM injection of phenytoin can result in localized crystallization of the drug, hematoma, sterile abscess, and myonecrosis. Reported complications include skin and soft tissue necrosis, compartment syndrome, gangrene, and death.
- Hypersensitivity reactions usually occur within 1 to 6 weeks of initiation. They include systemic lupus erythematosus, erythema multiforme, toxic epidermal necrolysis, Stevens-Johnson syndrome, hepatitis, rhabdomyolysis, acute interstitial pneumonitis, disseminated intravascular coagulation, and renal failure.

- Phenytoin is teratogenic and should never be initiated in a pregnant patient without consulting a neurologist and obstetrician.

DIAGNOSIS AND DIFFERENTIAL

- A therapeutic level is between 10 and 20 μg/mL; however, some patients require levels above 20 μg/mL for seizure control.
- Toxicity generally correlates with increasing plasma levels.
- Almost any CNS-active drug, such as ethanol, carbamazepine, benzodiazepines, barbiturates, and lithium, can mimic phenytoin toxicity.
- Disease states that resemble phenytoin toxicity include hypoglycemia, Wernicke encephalopathy, and posterior fossa hemorrhage or tumor.
- Seizures caused by phenytoin toxicity are rare, and other causes should be investigated.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients should be placed on a cardiac monitor, noninvasive blood pressure device, and pulse oximeter. An IV line should be established. Supplemental oxygen should be administered.
- Hypotension should be treated with isotonic crystalloid and discontinuation of phenytoin infusion.
- For acute oral overdose, multiple doses of oral activated charcoal (1 g/kg), mixed with a cathartic such as sorbitol, should be given every 2 to 4 h in the first 24 h due to extended absorptive phase.
- Bradydysrhythmias may require atropine or cardiac pacing.
- Seizures may be treated with benzodiazepines or phenobarbital.
- Hemodialysis and hemoperfusion are of no benefit.
- Following oral ingestion, patients with serious complications (e.g., seizures, coma, ataxia, and altered mental status) should be admitted. With mild symptoms, the patient may be treated with activated charcoal and discharged home if not suicidal and repeat levels are normal.
- Patients with symptomatic chronic intoxication should be admitted for observation unless toxic effects are minimal and they are 8 to 12 h from their last dose.
- Following IV administration, patients with persistent or significant complications should be admitted; those with transient effects can be discharged home.

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108 IRON

O. John Ma

PATHOPHYSIOLOGY

- Vomiting and diarrhea from iron toxicity may lead to hypovolemia, which in turn may produce hypotension, tissue hypoperfusion, and metabolic acidosis.
- When transferrin's ability to combine with iron is exhausted, free iron becomes available to enter mitochondria, inhibiting oxidative phosphorylation, and producing metabolic acidosis.

CLINICAL FEATURES

- When determining the amount of iron ingested, elemental iron must be used in calculations. Ferrous sulfate, ferrous fumarate, and ferrous gluconate contain about 20 percent, 33 percent, and 12 percent elemental iron, respectively.
- Based on clinical findings, iron poisoning can be divided into five stages. It is imperative to

note that patients can die in any stage of iron poisoning.

- The first stage develops within the first few hours after the ingestion. The direct irritative effects of iron on the gastrointestinal (GI) tract produce abdominal pain, nausea, vomiting, and diarrhea. Hematemesis is not unusual. The absence of these symptoms within 6 h of ingestion essentially excludes a diagnosis of significant iron toxicity. Vomiting is most consistently associated with acute iron toxicity.[1–3]
- During the second stage, which may continue for up to 24 h following ingestion, the patient's GI symptoms may resolve, thereby giving a false sense of security despite toxic amounts of iron being absorbed into the body. Patients may not be symptomatic but will still appear ill and may have abnormal vital signs and evidence of poor tissue perfusion.
- The third stage may appear either early in poisonings or develop hours after the second stage. Shock and a metabolic acidosis develop. Iron-induced coagulopathy may worsen bleeding and hypovolemia. Hepatic dysfunction, heart failure, and renal failure also may occur.
- The fourth stage develops 2 to 5 days after ingestion. It manifests as elevation of aminotransferase and may progress to hepatic failure.
- The fifth stage, which occurs 4 to 6 weeks after ingestion, involves gastric outlet obstruction secondary to the corrosive effects of iron on the GI tract.

DIAGNOSIS AND DIFFERENTIAL

- Toxic effects have been reported following oral doses as low as 10 to 20 mg/kg elemental iron. Moderate toxicity occurs at doses of 20 to 60 mg/kg, and severe toxicity can be expected following doses of greater than 60 mg/kg.[4]
- It is crucial to note that the determination of a single serum iron level does not reflect what iron levels have been previously, what direction they are going, or the degree of iron toxicity in the tissues.
- Serum iron levels have limited use in directing management, since excess iron is toxic intracellularly and not in the blood. In general, serum iron levels between 300 to 500 μg/dL correlate with mild systemic toxicity and iron levels between 500 to 1000 μg/dL correlate with moderate systemic toxicity. Levels greater than 1000 μg/dL are associated with significant morbidity.[5,6] A single low serum level does not exclude the diagnosis of iron toxicity. The total iron-binding capacity is now thought to have little value in the assessment of iron-poisoned patients because it becomes falsely elevated in the presence of elevated serum iron levels or deferoxamine.[7]
- A plain radiograph of the kidneys, ureters, and bladder may reveal iron in the GI tract; however, many iron preparations are not routinely detected, so negative radiographs do not exclude iron ingestion.[3]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients who have remained asymptomatic for 6 h after ingestion of iron and who have a normal physical examination do not require medical treatment for iron toxicity. Patients whose symptoms resolve after a short period of time and who have normal vital signs usually have mild toxicity and require only supportive care.
- Patients who are symptomatic or demonstrate signs of hemodynamic instability after iron ingestion should be managed aggressively in the emergency department (ED).
- Clinically ill patients should receive intravenous (IV) crystalloid infusion to help correct hypovolemia and tissue hypoperfusion.
- Patients who present within 2 h of ingestion should undergo gastric lavage. Activated charcoal does not adsorb iron and its use is not recommended.
- Whole-bowel irrigation with polyethylene glycol solution has been demonstrated to be efficacious. Administration of 250 to 500 mL/h in children and 2 L/h in adults via nasogastric tube may clear the GI tract of iron pills before absorption occurs.
- Coagulopathy should be corrected with parenteral vitamin K_1 (5 to 25 mg) or fresh-frozen plasma (10 to 25 mL/kg in adults; 10 mL/kg in pediatric patients).
- Deferoxamine is a chelating agent that can remove iron from tissues and can remove free iron from plasma. Deferoxamine combines with iron to form water-soluble ferrioxamine, which is excreted in the urine. Deferoxamine is safe to administer to children and pregnant women.
- Patients with mild iron toxicity may be treated with deferoxamine 90 mg/kg intramuscularly, up to 1 g in children and 2 g in adults. The dose may be repeated every 4 to 6 h, as clinically indicated.
- For patients with more severe iron toxicity, the preferred route of deferoxamine administration is as an IV infusion. Since hypotension is the rate-

limiting factor for IV infusion, it is recommended to begin with a slow IV infusion at 5 mg/kg/h. The deferoxamine infusion rate can be increased to 15 mg/kg/h, as tolerated. It is recommended not to exceed a total daily dose of 6 to 8 g. In a clinically ill patient with a known acute ingestion of iron, deferoxamine therapy should be initiated without waiting for the serum iron level results.

- Determination for the efficacy of deferoxamine involves evaluating serial urine samples. As ferrioxamine is excreted, the urine changes to the classic *vin rose* appearance.
- Patients who remain asymptomatic after 6 h of observation and have a reliable history of an insignificant ingestion may be considered for discharge. Patients initially symptomatic who become asymptomatic should still be admitted since this may represent the second stage. Patients who receive deferoxamine therapy should be admitted to an intensive care setting. Coma or shock is associated with approximately a 10 percent mortality rate.

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109 HYDROCARBONS AND VOLATILE SUBSTANCES

Lance H. Hoffman

EPIDEMIOLOGY

- Approximately 3 to 10 percent of unintentional childhood poisonings in the United States are hydrocarbon ingestions.
- An estimated 3.5 to 10 percent of young people have sought intoxication by inhalation of volatile substances.[1]

PATHOPHYSIOLOGY

- Pulmonary toxicity manifests as a chemical pneumonitis caused by direct parenchymal injury and altered surfactant function, usually as a result of aspiration of a low-viscosity compound or inhalation of a high-volatility compound.
- Central nervous system toxicity is a result of a direct response to systemically absorbed hydrocarbon or from indirect hypoxia secondary to pulmonary toxicity.
- Metabolism of hydrocarbons to 2,5-hexanedione results in peripheral neuropathy from demyelinization and retrograde axonal degeneration.[2]
- Free-radical metabolism of halogenated hydrocarbons cause hepatocellular damage by lipid peroxidation.[3]
- Metabolism of methylene chloride results in the endogenous production of carbon monoxide extending after the exposure has ceased because of a release of methylene chloride from the tissues.[4]

CLINICAL FEATURES

- Pulmonary and cardiac toxicity are most common.
- Pulmonary toxicity includes a chemical pneumonitis, pneumomediastinum, pneumothorax, and pneumatocele characterized by dyspnea and coughing.
- Radiographic evidence of pulmonary toxicity is usually seen within 4 to 6 h of exposure.
- Central nervous system toxicity may manifest as giddiness, slurred speech, ataxia, hallucinations, seizures, lethargy, and coma.
- Cardiac toxicity includes ventricular dysrhythmias, decreased cardiac contractility, bradycardia, and atrioventricular heart blocks.

- Hepatocellular toxicity will cause elevated transaminase levels within a day and right upper quadrant abdominal pain and jaundice in an additional 1 to 2 days.
- Gasoline, kerosene, and tetrachlorethylene can cause hemolysis.[5]
- Carbon monoxide poisoning from methylene chloride metabolism may result in a metabolic acidosis.

DIAGNOSIS AND DIFFERENTIAL

- Laboratory studies valuable to the evaluation of hydrocarbon exposure are hemoglobin and hematocrit, blood urea nitrogen, serum creatinine, hepatic transaminase levels, serum bilirubin, serum haptoglobin, and arterial blood gas with a determination of carboxyhemoglobin.
- A chest radiograph may reveal evidence of pulmonary toxicity.
- A single radiographic view of the abdomen may show the presence of chlorinated hydrocarbons in the alimentary tract.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of hydrocarbon exposure should begin with hazardous materials decontamination, preferably in the out-of-hospital setting, to limit any further exposure to the patient or medical personnel.
- Endotracheal intubation should be considered to prevent aspiration or hypoxia.
- Continuous cardiac monitoring is necessary in all patients.
- Hypotension should be treated with intravenous crystalloid; vasopressors should be avoided since they may precipitate dysrhythmias.
- Tachydysrhythmias should be treated with lidocaine or beta-blockers.
- Gastrointestinal decontamination using gastric lavage and activated charcoal is only effective in treating enterally absorbed hydrocarbon ingestions. These include Camphor, Halogenated hydrocarbons, Aromatic hydrocarbons, Metals, Pesticides (CHAMP mnemonic), and wood distillates (e.g., turpentine and pine oil).
- Hyperbaric oxygen therapy should be considered in the treatment of carbon monoxide poisoning associated with methylene chloride exposure.

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110 CAUSTIC INGESTIONS

Joseph J. Randolph

EPIDEMIOLOGY

- The American Association of Poison Control Centers reports approximately 100,000 potentially caustic exposures in the United States annually.[1] Most exposures are unintentional, with a large portion occurring in children < age 6.
- Acids are present in solutions used in rust removal, photography, drain and toilet bowl cleaning, and fertilizers. Common acids include sulfuric acid, formic acid, nitric acid, phosphoric acid, acetic acid, chromic acid, and hydrofluoric acid.
- Alkalis are used in drain and oven cleaners, household bleach, and batteries. Common alkalis include sodium hydroxide, lithium hydroxide, ammonium hydroxide, and sodium hypochlorite (household bleach).
- Household bleach is the most common exposure, accounting for more than 50,000 exposures per year.
- The Eye Bank Association of America reports that 300 of the 1000 corneal transplants in the

United States in 1995 were secondary to eye injuries caused by chemicals.[2]

PATHOPHYSIOLOGY

- Alkali injuries can induce deep tissue injury from liquefaction necrosis. Initially, there is direct cellular destruction from contact with the alkali. This is followed by thrombosis of local microvasculature that leads to further tissue necrosis.
- Liquid alkali ingestions are characterized by esophageal injuries. Severe injuries to the pancreas, gallbladder, and small intestine after intentional ingestion have been reported.[3]
- Household liquid bleach is not corrosive to the esophagus, but ingestion may cause emesis secondary to gastric irritation or pulmonary irritation related to chlorine gas production in the stomach.[4]
- Injuries by strong acids produce coagulation necrosis. Tissue destruction and cell death result from eschar formation, which is believed to protect against deeper injury. It was previously thought that acids were esophagus sparing, with most tissue injury concentrated in the stomach, but one study demonstrated a similar incidence of gastric and esophageal injury.[5]
- Despite relatively less tissue destruction, strong acid ingestion results in a higher mortality rate than does strong alkali ingestion, probably as a result of systemic absorption of acid leading to metabolic acidosis, hemolysis, and renal failure.

CLINICAL FEATURES

- Acids are foul tasting and malodorous. Signs and symptoms of acid ingestions include hematemesis, melena, and gastric perforation with peritonitis. Gastric outlet obstruction is a late complication.
- Alkalis are relatively tasteless and odorless, resulting in presentation after larger ingestions. Alkali ingestions can present acutely with orofacial burns, drooling, vomiting, odynophagia, dyspnea, hoarseness, and stridor. Chest pain suggests esophageal perforation with mediastinitis. Immediate injury is followed in 2 to 3 days by tissue sloughing, with an increased risk of perforation at 5 to 14 days.
- Dermal exposures to acid and alkalis cause local irritation. One exception is hydrofluoric acid, which may cause extensive tissue penetration and life-threatening systemic absorption. Patients often present with benign-appearing wounds but complain of a tremendous amount of pain. Severe injuries may result in hypocalcemia, hypomagnesemia, hyperkalemia, acidosis, and ventricular dysrhythmias.

DIAGNOSIS AND DIFFERENTIAL

- Helpful laboratory tests include arterial blood gas analysis (in acid ingestions), complete blood cell count, liver profile, electrolytes, calcium, magnesium, blood urea nitrogen, creatinine, type and cross-match, and coagulation studies.
- An upright chest radiograph should be obtained to evaluate for aspiration, abdominal free air, and pneumomediastinum.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Oxygen should be administered, and the airway stabilized with early endotracheal intubation if there is stridor or drooling. Cricothyrotomy is indicated if there is extensive upper airway edema.
- For patients with chest pain or signs of peritonitis, urgent surgical consultation should be obtained.
- Insertion of a small nasogastric tube for significant acid ingestions should be considered but should be avoided in alkali ingestions due to increased risk of aspiration.
- Sodium bicarbonate should be administered for serum pH <7.1.
- For ocular exposures, copious irrigation with at least 2 to 3 L (up to 10 L) of normal saline should be administered and continued until the pH is 7.5 to 8.0 (pH should be measured 10 min after cessation of irrigation for greater accuracy).
- Hydrofluoric acid dermal exposures should be treated with copious irrigation of the skin. Benzalkonium chloride or calcium gluconate paste (Surgilube mixed with calcium gluconate powder) should be applied to the exposed skin. Pain relief is the end point for successful treatment. Other therapy includes intradermal calcium gluconate (not greater than 0.5 mL/cm^2). If the preceding measures fail, intraarterial calcium gluconate (10 mL of 10% calcium gluconate in 40 mL of normal saline over 4 h) is the next step. Oral ingestions should be treated with nasogastric aspiration and irrigation and oral magnesium citrate (300 mL).
- All patients with significant acid and alkali ingestions should be admitted. Early endoscopy (12 to 24 h) will determine the extent of injury in alkali ingestions. Delayed endoscopy increases the risk of perforation.

- Clinitest tablets used in urine ketone testing can cause extensive mucosal burns. Dilution with water or milk is recommended if no stridor or drooling is present.
- For button-battery ingestion, chest and abdominal radiographs help determine the location of the battery. A battery lodged in the esophagus requires endoscopy for removal, whereas one that has passed beyond the gastroesophageal junction can be followed with stool checks and serial abdominal radiographs.
- In cases of lime exposure and other caustic powders, patients should brush off the compound and remove contaminated clothing before irrigating.

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111 PESTICIDES

M. Chris Decker

EPIDEMIOLOGY

- In 1997, 85,225 pesticide exposures were reported to poison control centers, with approximately 50 percent of them in children <6 years of age.
- Many pesticides contain inactive ingredients such as petroleum distillates that are harmful as well.

INSECTICIDES

ORGANOPHOSPHATES

- Organophosphates bind irreversibly to cholinesterases in the nervous system, which leads to the accumulation of neurotransmitters at the nerve synapses and neuromuscular junctions.
- Onset of symptoms ranges from minutes to 24 h, depending on the amount, type of toxin, and route of exposure.
- Systemic signs are due to cholinergic excess as a result of inhibition of cholinesterase. Effects can be separated as muscarinic, nicotinic, central nervous system (CNS), or nonsystemic.
- Muscarinic overstimulation results in SLUDGE syndrome: *S*alivation, *L*acrimation, *U*rination, *D*efecation, *G*astrointestinal, and *E*mesis. Other muscarinic signs are bradycardia, bronchorrhea, and visual disturbances.
- Nicotine overactivity causes hypertension, tachycardia, muscle fasciculations, and paralysis.
- Central nervous system effects include headache and altered mental status.
- Nonsystemic signs include dermatitis, eye and mucous membrane irritation, and wheezing.
- Organophosphate-induced delayed neuropathy may occur 2 to 3 weeks after poisoning, with a flaccid paralysis of the lower limbs.

CARBAMATES

- Carbamates are structurally related to organophosphates. They transiently and reversibly inhibit the cholesterase enzyme. Carbamates produce a similar cholinergic toxidrome to that of organophosphates but of shorter duration and with less CNS symptomatology.

CHLORINATED HYDROCARBONS

- Hexachlorocyclohexane (Lindane) is used clinically to control lice. Chlorinated hydrocarbons toxicity ranges from dizziness, fatigue, malaise, headache, delirium, apprehension, tremulousness to seizures, coma, and death.
- Even with therapeutic use of hexachlorocyclohexane, children and the elderly are more at risk for developing CNS toxicity and seizures.

PYRETHRINS

- Pyrethrins block the sodium channel at the neuronal cell membrane, causing repetitive neuronal discharges. Pyrethrins most commonly cause hypersensitivity responses, which include bronchospasm and anaphylaxis. They may produce dermal, pulmonary, gastrointestinal (GI), and neurologic findings.

HERBICIDES

- Toxicity of herbicides, which are pesticides used to kill weeds, leads to a wide variety of symptoms generally based upon which organ system has been exposed.
- Chlorphenoxy compounds may cause tachycardia, dysrhythmias, and hypotension, and muscle toxicity manifested by muscle pain, fasciculations, and rhabdomyolysis.
- Common bipyridial herbicides are paraquat and diquat. Paraquat is especially toxic with caustic effects resulting in severe dermal, corneal, and mucous membrane burns, including the respiratory and GI epithelium. Cardiovascular collapse may occur early, especially in the case of large ingestions, and results in pulmonary edema, renal failure, hepatic necrosis, and multisystem organ failure. Metabolic acidosis is due to hypoxemia and multisystem organ failure.
- Urea-substituted compounds are much less toxic than other herbicides and generally cause few systemic effects other than methemoglobinemia.

RODENTICIDES

- Sodium monofluoroacetate, a commercial exterminator compound, is converted to a metabolite, fluorocitrate, which interferes with the Krebs cycle. Signs and symptoms of toxicity include nausea, lactic acidosis, respiratory depression, cardiovascular collapse, and altered mental status.
- Strychnine toxicity results from its competitive antagonism of the inhibitory neurotransmitter glycine at the postsynaptic spinal cord motor neuron. Signs and symptoms of strychnine toxicity include facial grimacing, muscle twitching, severe extensor spasms, and opisthotonos; it eventually may lead to medullary paralysis and death.
- Thallium sulfate is absorbed through the skin, by inhalation, and through the GI tract. Exposure to thallium sulfate initially causes GI hemorrhage followed by a latent period, in turn succeeded by the development of neurologic symptoms, respiratory failure, and dysrhythmias.
- Zinc phosphide ingestion results in the liberation of phosphine gas, which subsequently causes GI irritation, hepatocellular toxicity, direct pulmonary injury (if the gas is inhaled), cardiovascular collapse, altered mental status, seizures, and noncardiogenic pulmonary edema.
- Yellow phosphorous causes severe topical burns to areas of contact and also may cause jaundice, seizures, and cardiovascular collapse.
- ANTU exhibits primarily pulmonary effects with dyspnea, pleuritic chest pain, and noncardiogenic pulmonary edema, while cholecalciferol causes the typical symptoms of vitamin D excess.
- Red squill poisoning is a low-toxicity rodenticide that presents as severe GI distress and cardiac dysrhythmias.
- The most common low-toxicity agent poisoning occurs with superwarfarins and related compounds. Superwarfarins inhibit vitamin K–dependent clotting factors. Exposures most commonly come to attention on a delayed basis with symptoms of an unexplained coagulopathy.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of pesticide poisoning is made on the basis of the history and physical examination in the majority of cases.
- In the case of organophosphate poisoning, an assay of both serum and red blood cell cholinesterase activity can be obtained for diagnosis and to guide treatment, though results seldom become available for decision making in the emergency department.
- Nausea, vomiting, and cardiac dysrhythmia suggest red squill toxicity.
- In the case of superwarfarin ingestion, determination of the prothrombin time at 24 and 48 h is recommended.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The mainstay of treatment for pesticide exposure is identification of the specific agent involved and supportive monitoring and treatment.

TABLE 111-1 Pesticides and Specific Antidotes

PESTICIDE	ANTIDOTE	DOSING
Organophosphates	Atropine	0.5 mg/kg up to 2–4 mg IV q 5–15 min—consider IV infusion and titrate to effect (drying secretions)
	2-PAM	20–40 mg/kg up to 1 g IV—may repeat in 1–2 h, then every 6–8 h for 48 h
Carbamates	Atropine	As for organophosphates
	2-PAM	Use is controversial and may be contraindicated
Urea-substituted herbicides	Methylene blue	As for treatment of methemoglobinemia
Zinc phosphide	$NaHCO_3$	Used for intragastric alkalinization
Yellow phosphorous	K Permanganate or H_2O_2	Used for gastric lavage
Arsenic	Heavy metal chelators	As for heavy metal poisoning
Red squill	Antidysrhythmics, Fab fragments	As for digoxin toxicity
Superwarfarins	Vitamin K	Up to 20 mg IV, repeated and titrated to effect

ABBREVIATIONS: IV = intravenous; 2-PAM = pralidoxime.

- Symptomatic patients require attention to airway protection and ventilation with supplemental oxygen to maintain saturation to ≥95%. Tracheal intubation and mechanical ventilation with high oxygen concentrations may be necessary in severe poisoning. Maintenance of intravascular volume and urine output should be assured.
- Meticulous attention to patient decontamination (dermal, ocular, or GI) is important as is prevention of absorption by the patient and caretakers involved in patient care.
- Administration of a specific antidote may be appropriate for selected individual agents (Table 111-1).
- Pralidoxime (2-PAM) displaces organophosphates from the cholinesterases. It restores cholinesterase activity and detoxifies the remaining organophosphate molecules. Clinically, 2-PAM ameliorates the CNS, nicotinic, and muscarinic effects.
- Disposition depends upon the pesticide involved in the exposure. Asymptomatic patients with a history of contact with a pesticide may require decontamination and a 6- to 8-h observation period only. Close follow-up should be arranged for patients with exposure to rodenticides that produce symptoms on a delayed basis.
- A low threshold for admission should be maintained for patients with intentional ingestions. Any patient with a history of paraquat or diquat exposure should be admitted because of the extreme lethality of these compounds. Consideration for admission to the intensive care unit is an individual one based upon the specific toxin involved and the overall clinical picture of the patient.

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112 CARBON MONOXIDE AND CYANIDE

M. Chris Decker

CARBON MONOXIDE

EPIDEMIOLOGY

- Carbon monoxide (CO) is responsible for more morbidity and mortality than any other toxin.
- CO is formed from the incomplete combustion of fossil fuel or tobacco and as a metabolite of methylene chloride (paint remover).
- CO toxicity is more common in northern climates and during winter months.

PATHOPHYSIOLOGY

- CO—which binds to hemoglobin, myoglobin, and cytochromes P450 and AA3—competes with oxygen for binding sites and prevents oxygen utilization.
- CO binds to hemoglobin about 210 to 280 times more tenaciously than oxygen. The binding of CO to hemoglobin shifts the oxyhemoglobin dissociation curve to the left. Therefore, carboxyhemoglobin (COHb) holds on to oxygen at lower oxygen tensions.
- When CO binds to mitochondrial cytochromes, it stops the electron chain reaction and prevents oxidative phosphorylation.
- Poisoning of the myocardial myoglobin reduces cardiac contractility, cardiac output, and oxygen delivery.
- White blood cells adhere to CO-poisoned tissue. Upon reperfusion of those tissues, the white blood cells accelerate lipid peroxidation. This is termed reperfusion injury.
- The half-life of COHb is 320 min when a patient is breathing room air, 60 min when breathing 100% normobaric oxygen, and 23 min when breathing 100% hyperbaric oxygen at 2.8 atmospheres of pressure.

CLINICAL FEATURES

- High oxygen-extracting organs such as the brain and heart easily become dysfunctional from CO intoxication.
- The clinical picture at the site of poisoning often corresponds to the severity of poisoning and to on-scene COHb levels (Table 112-1).
- Symptoms and signs are worse in situations where neurologic and myocardial oxygen demand increases, such as trauma, burns, drug ingestion, and increased activity.
- Fetuses and neonates are particularly susceptible to the toxic effects of the gas due to the presence of fetal hemoglobin and an oxygen dissociation curve that is already shifted to the left. Children are frequently affected and make up almost 40 percent of patients treated with hyperbaric oxygen therapy.

DIAGNOSIS AND DIFFERENTIAL

- The primary key to the diagnosis is maintaining a high degree of clinical suspicion.
- The most useful laboratory test is the determination of the COHb level. Pulse oximetry may be normal in CO poisoning.
- Psychometric testing can detect subtle deficits in mental status and assess for indications for hyperbaric oxygen therapy.
- In cases of symptomatic exposure, an electrocardiogram (ECG) and cardiac enzyme determinations are suggested. Chest radiographs are generally obtained for fire victims, and other pulmonary function testing may be helpful as well.
- The differential diagnosis is extremely broad and includes a wide variety of toxins, infectious agents, and cardiac/pulmonary diseases as well as the host of causes for altered mental status. Particularly in colder months, patients with headache, nausea, weakness, fatigue, difficulty in concentrating, dizziness, chest pain, and abdominal pain must be evaluated with CO toxicity in mind.
- Victims of house fires with appropriate symptoms

TABLE 112-1 Symptoms and Signs at Various Carboxyhemoglobin Concentrations

COHb LEVEL(%)	SYMPTOMS AND SIGNS
0	Usually none
10	Frontal headache
20	Throbbing headache, dyspnea with exertion
30	Impaired judgment, nausea, dizziness, visual disturbances, fatigue
40	Confusion, syncope
50	Coma, seizures
60	Hypotension, respiratory failure
≥70	Death

and signs must be evaluated specifically for CO poisoning.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Cardiac monitoring and an IV line should be instituted. Oxygen (100%) should be administered through a tight-fitting mask.
- Table 112-2 outlines appropriate treatment guidelines for CO poisoning.
- Hyperbaric oxygen (HBO) therapy is indicated for severe poisoning based upon clinical findings and the COHb level. The goal of treatment is not only amelioration of the acute event but also to prevent delayed neuropsychiatric sequelae. HBO should be carefully considered, especially for patients at the extremes of age and in pregnancy.

CYANIDE

EPIDEMIOLOGY

- Cyanide is found in large amounts in certain nuts, plants, and fruit pits in the form of cyanogenic glycoside. Sodium nitroprusside contains cyanide.
- Acute cyanide poisonings occur in the following settings: (1) inadvertent occupational exposure (inhalation of hydrogen cyanide gas used in the production of solvents, enamels, paints, glues, wrinkle-resistant fabrics, herbicides, pesticides, and fertilizers and in electroplating); (2) inhalation of smoke from burning plastics in closed-space fires; (3) inadvertent, suicidal, or homicidal ingestion; (4) iatrogenic toxicity due to infusion of sodium nitroprusside; (5) ingestion of plant products containing cyanogenic glycosides.

PATHOPHYSIOLOGY

- Cyanide disrupts oxidative phosphorylation by binding to cytochrome A3 and blocks the ability of tissues to use oxygen, which leads to anaerobic metabolism. Anaerobic metabolism results in the accumulation of lactic acid and a metabolic acidosis.

CLINICAL FEATURES

- The most common modes of poisoning are inhalation, oral ingestion, and dermal contact. Absorption of cyanide gas is immediate. Ingestion of cyanide salts produces symptoms within minutes. Ingestion of cyanogenic compounds produces symptoms within hours.
- The hallmark of cyanide poisoning is apparent hypoxia without cyanosis.

TABLE 112-2 CO Poisoning Treatment Guidelines

Mild poisoning	Criteria	COHb levels <30% No symptoms or signs of impaired cardiovascular or neurologic function May have complaint of headache, nausea, vomiting
	Treatment	100% oxygen by tight-fitting nonrebreathing mask until COHb level remains <5% Admission for COHb level of >25% Admission for patients with underlying heart disease regardless of COHb level
Moderate poisoning	Criteria	COHb levels 30–40% No symptoms or signs of impaired cardiovascular or neurologic function
	Treatment	100% oxygen by tight-fitting nonrebreathing mask until COHb level remains <5% Cardiovascular status followed closely even in asymptomatic patients, consider ECG and cardiac enzymes Determination of acid-base status (will be corrected by high-flow oxygen) Admission for observation and cardiovascular monitoring
Severe poisoning	Criteria	COHb levels >40% *or* Cardiovascular or neurologic impairment at any COHb level
	Treatment	100% oxygen by tight-fitting nonrebreathing mask Cardiovascular function monitoring Determination of acid-base status Admission *or* Transfer to a HBO facility immediately if available or if no improvement in cardiovascular or neurologic function within 4 h

- Metabolic acidosis is prominent, with high lactate levels due to failed oxygen utilization.
- Awake patients complain of breathlessness and anxiety. In more severe cases, loss of consciousness (often with seizures) and tachydysrhythmias are apparent, which may proceed on to bradycardia and apnea and finally asystolic cardiac arrest.
- Other clues to cyanide toxicity are bright red retinal blood vessels, oral burns from ingestions, the smell of bitter almonds on the patient's breath, and high peripheral venous oxygen saturations (acyanosis).

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of cyanide toxicity should always be considered in the poisoned patient with profound metabolic acidosis. Further support for the diagnosis is any finding suggesting decreased oxygen utilization. Arterial blood gas assays can identify acid-base disturbances and the presence of an oxygen saturation gap, while serum lactate levels may provide additional supporting evidence.
- The differential diagnosis includes other cellular toxins such as carbon monoxide, hydrogen sulfide, and simple asphyxiants. In the setting of an ingestion, other possibilities are methanol, ethylene glycol, iron, and salicylates. Severe isoniazid or cocaine poisoning may mimic the effects of cyanide, causing severe metabolic acidosis and seizures.
- Iatrogenic thiocyanate toxicity may occur in a patient who is on nitroprusside and becomes encephalopathic or complains of tinnitus. Thiocyanate levels >100 mg/L support the diagnosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Cardiac monitoring and an IV line should be instituted. Those with altered mental status must be considered for IV glucose, thiamine, and naloxone administration.
- Gastric lavage and administration of activated charcoal are standard for cyanide ingestion; dermal contacts require skin decontamination, and inhalational exposures require removal from the source.
- Specific treatment with nitrite-thiosulfate antidote therapy in the form of a kit from Taylor Pharmaceuticals must be considered (Table 112-3). Asymptomatic patients or those with minimal symptoms should be observed and treated only if clinical deterioration is noted. Severely toxic patients with a clear history of exposure demand full and immediate treatment.
- Due to the potential side effects of hypotension and induction of methemoglobinemia, hypotensive acidotic patients without clear cyanide toxicity or with smoke inhalation are best served by administration of IV sodium thiosulfate only.

TABLE 112-3 Treatment of Cyanide Poisoning

CHILDREN

1. 100% oxygen
2. Administration of IV sodium nitrite and sodium thiosulfate:

Hb (g/100 mL)	3% $NaNO_2$ (mL/kg)	25% $Na_2S_2O_3$ (mL/kg)
7	0.19	1.65
8	0.22	1.65
9	0.25	1.65
10	0.27	1.65
11	0.30	1.65
12	0.33	1.65
13	0.36	1.65
14	0.39	1.65

3. May repeat once at half dose if symptoms persist.
4. Monitor methemoglobin to keep level less than 30%

ADULTS

1. 100% oxygen.
2. Amyl nitrite; crack and inhale 30 s/min.*
3. Sodium nitrite: 10 mL IV (10-mL ampule of 3% solution = 300 mg).
4. Sodium thiosulfate: 5 mL IV (50-mL ampule of 25% solution = 12.5 g).
5. May repeat once at half dose if symptoms persist.

* Administration of amyl nitrite is necessary only if venous access has not been obtained.

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113 HEAVY METALS

Lance H. Hoffman

LEAD

- Lead is the most common cause of chronic metal poisoning, affecting approximately 890,000 children, ages 1 to 5 years, with a blood lead level of 10 μg/dL or more.[1]
- Lead toxicity should be considered in patients with a combination of central nervous system (CNS) symptoms (e.g., delirium, seizures, coma, and memory deficit), abdominal symptoms (e.g., colicky pain, constipation, and diarrhea), or hematologic manifestations (e.g., hypoproliferative or hemolytic anemia).
- Serum lead levels >10 μg/dL are diagnostic of lead toxicity. Radiographic evidence of lead toxicity includes horizontal, metaphyseal bands on long bones, especially involving the knee, and radiopaque material in the alimentary tract.
- Chelation therapy is the mainstay of treatment in patients with encephalopathy or children with lead levels greater than 45 μg/dL. Dimercaprol (BAL) 3 to 5 mg/kg intramuscularly (IM) every 4 h and CaNa2-EDTA 1500 μg/m^2 every 24 h by continuous intravenous infusion beginning 4 h after the initial BAL dose are the standard agents. Radiopaque lead material in the alimentary tract requires whole-bowel irrigation for decontamination.
- Admission is indicated for all symptomatic patients, asymptomatic children with lead levels >45 μg/dL, and patients who would otherwise return to the environment of lead exposure.

ARSENIC

- Arsenic is the most common cause of acute metal poisoning and the second most common cause of chronic metal poisoning. It is found in agricultural chemicals and contaminated well water, and it is used in mining and smelting.
- Arsenic inhibits pyruvate dehydrogenase, interferes with the cellular uptake of glucose, and uncouples oxidative phosphorylation.[2]
- Acute arsenic toxicity results in nausea, vomiting, severe diarrhea, and hypotension a few hours after the exposure. Chronic arsenic toxicity presents as generalized weakness, malaise, morbilliform rash, and an ascending, stocking-glove sensory or motor peripheral neuropathy.
- Evaluation may reveal Mee lines (1 to 2 mm transverse, white lines on the nails), prolonged QT interval on electrocardiogram, and radiopaque arsenic in the alimentary tract.[3]
- Volume resuscitation is used to treat hypotension. Cardiac tachydysrhythmias are best treated with lidocaine and bretylium; class Ia, Ic, and III antidysrhythmics should be avoided since they may worsen QT prolongation.
- Chelation therapy with BAL 3 to 5 mg/kg IM every 4 h should be instituted in suspected arsenic toxicity. Whole-bowel irrigation is needed if arsenic is present in the alimentary tract on abdominal radiographs.

MERCURY

- Short-chained alkyl mercury compounds and elemental mercury predominantly affect the CNS,

producing erethism, which includes anxiety, depression, irritability, mania, sleep disturbances, shyness, and memory loss.[4] Tremor is also common.[5]
- Mercury salts spare the CNS, but cause a corrosive gastroenteritis resulting in abdominal pain and cardiovascular collapse with a high likelihood of acute tubular necrosis within a day of ingestion.
- All forms of mercury, except the short-chained alkyl mercury compounds, produce the immune-mediated condition in children called *acrodynia,* consisting of a generalized rash, irritability, hypotonia, and splenomegaly.
- Mercury inhalation produces a pneumonitis, acute respiratory distress syndrome, and progressive pulmonary fibrosis.[6]
- Although BAL is contraindicated in short-chained alkyl mercury compound toxicity because it may exacerbate CNS symptoms, it is the chelator of choice for mercury salts. Dimercaprol should be administered 3 to 5 mg/kg IM every 4 h, in addition to initial gastric decontamination.
- Dimercaptosuccinic acid is gaining favor as the treatment of choice for short-chained alkyl mercury compound toxicity.[7]

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114 HAZARDOUS MATERIALS EXPOSURE

Joseph J. Randolph

EPIDEMIOLOGY

- A hazardous material is any substance (chemical, nuclear, or biologic) that poses a risk to health, safety, property, or the environment.
- Eighty percent of events occur at fixed facilities, 20 percent are transportation related, and over 10 percent occur within hospitals and schools.[1]
- Sixty-five percent of fatalities result from associated trauma, 22 percent from burns, and 10 percent from respiratory compromise.[2]
- Most injuries and deaths are associated with exposure to chlorine, ammonia, nitrogen fertilizer, or hydrochloric acid. Other commonly involved chemicals include petroleum products, pesticides, corrosives, metals, and volatile organic compounds.[2]
- Data on involved chemicals are essential. Resources include regional poison centers, material safety data sheets, transportation-specific markings [Department of Transportation (DOT) placards, shipping papers], private agencies (CHEMTREC), government agencies [National Regulatory Commission, Center for Disease Control, Environmental Protection Agency (EPA), and ATSDR], computer databases (Poisindex, Safetydex, Tomes Plus, ToxNet), and the internet.[3–5]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Triage occurs outside the hospital where both urgency of care and adequacy of decontamination are assessed. Under no circumstances is a patient allowed into the hospital unless fully decontaminated.

- Level A attire (fully encapsulated chemical-resistant suit and self-contained breathing apparatus) is recommended by the EPA when the concentration or identity of toxins is unknown (most hazardous incidents).
- Medical stabilization prior to decontamination should be limited to opening the airway, cervical spine stabilization, oxygen administration, ventilatory assistance, and application of direct pressure to arterial bleeding.
- Decontamination is performed in three ''zones.'' The ''hot zone'' is the area at the scene or outside the hospital where patients with no prior decontamination are held. The ''warm zone'' is the area outside (or physically isolated from) the hospital where decontamination occurs. The ''cold zone'' is where fully decontaminated victims are transferred. There should be no movement of personnel between zones.
- Access to the hot and warm zones is restricted to personnel with suitable protective clothing (including, but not limited to, a chemical-resistant suit and self-contained breathing apparatus when the highest level of protection is needed).
- Removing all clothing and brushing away gross particulate matter begins decontamination. Whole-body irrigation is then initiated with copious amounts of water and mild soap or detergent, except in cases where water-reactive substances (lithium, sodium, potassium, calcium, lime, calcium carbide, and others) may be involved.
- The hands and face are generally the most contaminated; decontamination should begin at the head and work downward, taking care to avoid runoff onto other body parts. Decontamination should continue for at least 3 to 5 min. Patients should then be wrapped in clean blankets and transferred to the cold zone.

SPECIFIC MEDICAL MANAGEMENT

INHALED TOXINS

- This group includes gases, fumes, dusts, and aerosols, resulting in upper airway damage or pulmonary toxicity. Specific agents include phosgene, chlorine, ammonia, and riot control agents (mace and pepper spray).
- Oxygen and bronchodilators should be administered, along with examination of the upper airway for respiratory compromise. Patients should be intubated if they develop respiratory distress or airway edema.
- Riot control agents [including capsaicin (CS) used by law enforcement and mace (CN) sold for self-protection] result in self-limited irritation of exposed mucous membranes and skin.

NEUROTOXINS

- The most likely neurotoxins are the nerve agents. Five organophosphate compounds are recognized as nerve agents: tabun (GA), sarin (GB), soman (GD), GF, and VX.
- These agents inhibit acetylcholinesterase, resulting in build-up of acetylcholine at brain synapses (causing seizures and coma), motor endplates (causing weakness, paralysis, and respiratory insufficiency), and the autonomic nervous system (causing salivation, lacrimation, urination, diarrhea, bronchorrhea, and miosis).
- Treatment consists of complete decontamination, oxygen administration, administration of atropine 2 mg and pralidoxime (2-PAMCL) 600 mg intravenously (IV) or intramuscularly (IM), and supportive care.

DERMAL TOXINS

- Dermal toxins include alkalis (sodium hydroxide and cement), phenol, hydrofluoric acid, and vesicants [mustard (sulfur mustard; H; HD), Lewisite (L), and phosgene oxime (CX)]. These agents cause significant pulmonary toxicity and ocular toxicity.
- Skin decontamination with large volumes of water is the mainstay of treatment.
- Hydrofluoric acid burns result in dysrhythmias, seizures, local tissue destruction, and electrolyte abnormalities. Treatment consists of intravenous (IV) calcium or magnesium as well as topical calcium gluconate gel.
- Injection of calcium gluconate into the affected area at a maximum of 0.5 mL/cm^2 of tissue may be considered for intractable pain to neutralize the fluoride ion. Intraarterial calcium through a radial artery line has been recommended for digital burns.

OCULAR EXPOSURES

- Ocular exposures demand immediate irrigation with large volumes of water. Prehospital irrigation

for up to 20 min prior to transport (in stable patients) is recommended. Gross particulate matter should be brushed from around the eye, and contact lenses should be removed.
- Absence of pain may not indicate cessation of ocular damage, and irrigation should continue until ocular pH returns to 7.4.
- Visual acuity, fluorescein staining, and slit-lamp evaluation are indicated, with ophthalmologic consultation in all but the most trivial of exposures.

BIOLOGIC WEAPONS

- Biologic weapons include microbes (anthrax, plague, tularemia, Q fever, and viruses), mycotoxins (trichothecene), and bacterial toxins (ricin, staphylococcal enterotoxin B, botulinum, and shigella).
- Biologic agents used as weapons are almost invariably delivered by droplet (aerosol) spread, resulting in fulminant infectious complications after a variable incubation period.
- Anthrax spores are stable and easy to cultivate and have become an agent of choice among terrorist groups. After an incubation period of 1 to 6 days, infected patients develop fever, myalgia, cough, chest pain, and fatigue. Hemorrhagic meningitis and necrotizing hemorrhagic mediastinitis also are seen. Treatment involves IV ciprofloxacin or doxycycline.
- Botulism, the most potent toxin known, exerts its effects through entering presynaptic cholinergic neurons and blocking acetylcholine release. Following an incubation period of 24 to 36 h, bulbar palsies, diplopia, ptosis, mydriasis, and dysphagia develop. A classic descending, symmetric skeletal muscle paralysis ensues, followed by respiratory failure and death. The diagnosis is clinical, and treatment is directed primarily at providing respiratory support.
- Sodium hypochlorite 0.5% solution (household bleach diluted 1 : 9 with water) is effective at neutralizing most biohazard materials and should be used for patient decontamination.

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115 DYSHEMOGLOBINEMIAS

Alex G. Garza

METHEMOGLOBINEMIA

PATHOPHYSIOLOGY

- Methemoglobinemia is acquired when the normal mechanisms responsible for the elimination of methemoglobin are overwhelmed by an exogenous oxidant stress, such as a drug or chemical agent.
- At present, most cases of methemoglobinemia are due to phenazopyridine (Pyridium), benzocaine (topical anesthetic), and dapsone (antibiotic often used in HIV-related therapy).
- Methemoglobinemia can affect any age group but, due to an underdeveloped methemoglobin reduction mechanism, the prenatal and infant age groups are more susceptible. Another common cause of acquired infantile methemoglobinemia is gastroenteritis.

CLINICAL FEATURES

- The clinical suspicion of methemoglobinemia should be raised when the patient's pulse oximetry

approaches 85 percent, there is no response to supplemental oxygen, and brownish-blue skin and ''chocolate-brown'' blood discoloration are noted.
- Patients with normal hemoglobin concentrations do not develop clinically significant effects until the methemoglobin levels rise to about 15 percent of the total hemoglobin.
- Patients may seek evaluation for the profound cyanosis that occurs when the methemoglobin concentration reaches about 1.5 g/dL.
- At methemoglobin levels between 15 to 30 percent, symptoms such as anxiety, headache, weakness, and light-headedness develop, and patients may exhibit tachypnea and sinus tachycardia.
- Methemoglobin levels of 50 to 60 percent impair oxygen delivery to vital tissues, resulting in myocardial ischemia, dysrhythmias, depressed mental status (including coma), seizures, and lactic acidosis. Levels above 70 percent are largely incompatible with life.
- Anemic patients may not exhibit cyanosis until the methemoglobin level rises dramatically above 10 percent, because it is the absolute concentration, not the percentage of methemoglobin, that determines cyanosis. Anemic patients may likewise suffer significant symptoms at lower methemoglobin concentrations because the relative percentage of hemoglobin in the oxidized form is greater.
- Patients with preexisting diseases that impair oxygen delivery to red blood cells (e.g., chronic obstructive pulmonary disease and congestive heart failure) will manifest symptoms with less significant elevations of methemoglobin levels.
- Conditions that shift the oxyhemoglobin dissociation curve to the right, such as acidosis or elevated 2,3-DPG, may result in somewhat better toleration of methemoglobinemia.

DIAGNOSIS AND DIFFERENTIAL

- Pulse oximetry cannot distinguish oxyhemoglobin from methemoglobin. It may read an inappropriately normal value in patients with moderate methemoglobinemia, and it may trend toward 85 percent in patients with severe methemoglobinemia.
- Definitive identification of methemoglobinemia relies on co-oximetry.
- The oxygen saturation obtained from a conventional arterial blood gas analyzer also will be falsely normal because it is calculated from the dissolved oxygen tension, which may be appropriately normal.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with methemoglobinemia require optimal supportive measures to ensure oxygen delivery.
- The efficacy of gastric decontamination is limited due to the substantial time interval from exposure to development of methemoglobin. If an on-going source of exposure exists, a single dose of oral activated charcoal is indicated.
- Therapy with methylene blue is reserved for those patients with documented methemoglobinemia or a high clinical suspicion of the disease. Unstable patients should receive methylene blue, but may require blood transfusion or exchange transfusion for immediate enhancement of oxygen delivery. The initial dose of methylene blue is 1 to 2 mg/kg intravenously (IV), and its effect should be seen within 20 min. If necessary, repeat dosing of methylene blue is acceptable, but high doses (>7 mg/kg) may actually induce methemoglobin formation.
- Treatment failures occur in some groups, which include glucose-6-phosphate dehydrogenase (G6PD) deficiency and other enzyme deficiencies, and may occur with hemolysis.
- Patients who have been exposed to agents with long half-lives, such as dapsone, may require serial dosing of methylene blue.
- Patients with methemoglobinemia unresponsive to methylene blue therapy should be treated supportively. If clinically unstable, the use of blood transfusion or exchange transfusion is indicated.

SULFHEMOGLOBINEMIA

- Sulfhemoglobinemia is less common than methemoglobinemia. Although patients with sulfhemoglobinemia have a clinical presentation similar to that of methemoglobin, the disease process is substantially less concerning.
- The diagnosis is difficult to confirm, because standard co-oximetry does not differentiate sulfhemoglobin from methemoglobin.
- Sulfhemoglobin is not reduced by treatment with methylene blue, and generally patients require

only supportive care, although transfusions may be necessary for severe toxicity.

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Section 14
ENVIRONMENTAL INJURIES

116 FROSTBITE AND HYPOTHERMIA

Mark E. Hoffmann

EPIDEMIOLOGY

- In the United States, more than 700 people die from hypothermia each year; one-half of those who die are older than 65 years.[1]
- People at the extremes of age are at risk for developing hypothermia.
- Alcohol and drug-intoxicated persons, along with psychiatric patients, account for the majority of frostbite cases in the United States.[2]

PATHOPHYSIOLOGY

- Body temperature falls as a result of heat loss by conduction, convection, radiation, or evaporation.
- Heat conservation is controlled by the hypothalamus. Heat is conserved by shivering, peripheral vasoconstriction, and behavioral responses (dressing appropriately and seeking shelter).
- Exposure to a cold environment, depressed metabolic rate, central nervous system (CNS) dysfunction, sepsis, dermal disease, and drugs can lead to hypothermia.
- The initial excitatory response to hypothermia consists of a rise in heart rate, blood pressure, cardiac output, and vasoconstriction with shivering.
- Hypothermia impairs renal concentrating function leading to ''cold diuresis,'' impaired platelet function with bleeding, and a leftward shift of the oxyhemoglobin dissociation curve resulting in impaired oxygen release to the tissues.
- Local cold injury and frostbite occur when hypothermia causes increased blood viscosity, extracellular ice crystal formation, intracellular dehydration, and lysis. This occurs when freezing temperatures are reached.[3,4]

CLINICAL FEATURES

- Mild hypothermia, 32°C (89.6°F) to 35°C (95°F), presents with shivering, tachycardia, and elevated blood pressure.
- Shivering ceases and heart rate and blood pressure fall when core temperatures drop below 32°C (89.6°F). Mentation slows, and there is a loss of cough and gag reflexes. A ''cold diuresis'' ensues with resulting dehydration. Patients can have intravascular thrombosis and disseminated intravascular coagulation.
- The electrocardiogram may show Osborn J-waves in hypothermic patients. The cardiac rhythm progresses from tachycardia to bradycardia to atrial fibrillation with a slow ventricular rate to ventricular fibrillation and then to asystole as the core temperature falls.
- First-degree and second-degree frostbite are superficial injuries that present with edema, burning, erythema, and blistering.
- Third-degree and fourth-degree frostbite are deep injuries involving the skin, subcutaneous tissue (third-degree), and muscle/tendon/bone (fourth-degree). Patients present with cyanotic and insensate tissue that may have hemorrhagic blisters and skin necrosis. Later, this tissue appears mummified.[5]
- Frostnip is a less severe form of frostbite that resolves with rewarming and no tissue loss.

- Trench foot results from cooling of tissue in a wet environment at above-freezing temperatures over several hours to days. Long-term hyperhidrosis and cold insensitivity are common.
- Chilblains (pernio) presents with painful and inflamed skin lesions caused by chronic, intermittent exposure to damp, nonfreezing ambient temperatures.[6]
- Once affected by chilblains, frostnip, or frostbite, the body part involved becomes more susceptible to reinjury.

DIAGNOSIS AND DIFFERENTIAL

- Hypothermia is diagnosed when the core body temperature is below 35°C (95°F).
- Underlying disease states that may result in hypothermia, such as thyroid deficiency, CNS dysfunction, infection, sepsis, adrenal insufficiency, dermal disease, drug intoxication, and metabolic derangement, need to be evaluated and considered.
- Localized cold-related injuries are diagnosed by history and clinical exam.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Chilblains and trench foot should be managed with elevation, warming, and bandaging of the affected tissues. Nifedipine 20 mg tid, topical corticosteroids, and oral prednisone may be helpful.
- Rapid rewarming with circulating water at 42°C (107.6°F) for 10 to 30 min results in thawing of frostbitten extremities. Dry air rewarming may cause further tissue injury and should be avoided. Patients should receive narcotics, ibuprofen, and aloe vera. Penicillin G 500,000 U every 6 h for 48 h has been beneficial, according to some published protocols.[7]
- Patients with mild hypothermia may be warmed passively by removal from the cold environment and with the use of insulating blankets.
- Patients with more severe hypothermia should be placed on a pulse-oximeter or cardiac monitor, and a core temperature probe should be placed (rectal or esophageal).
- Attention should be placed on the ABCs and initial resuscitation. If there is no cardiovascular instability, active external warming may be applied (radiant heat, warmed blankets, warm water immersion, and heated objects) in conjunction with warmed intravenous fluids and warmed humidified oxygen.
- If cardiovascular instability is present, more aggressive active core rewarming is required (gastric, bladder, peritoneal, and pleural lavage). These lavage fluids should be heated to 42°C (107.6°F).[8] Ventricular fibrillation is usually refractory to defibrillation until a temperature of 30°C (86°F) is obtained, although three countershocks should be attempted.
- Rewarming through an extracorporeal circuit is the method of choice in the severely hypothermic patient in cardiac arrest.[9] When this equipment is not available, resuscitative thoracotomy with internal cardiac massage and mediastinal lavage is an acceptable alternative.
- All patients with more than isolated superficial frostbite or mild hypothermia should be admitted to the hospital. A patient should not be discharged unless they can return to a warm environment.

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117 HEAT EMERGENCIES

Mark E. Hoffmann

EPIDEMIOLOGY

- The death rate for heat-related conditions is highest among the extremes of age.
- Death rates increase from 1 death per million in people <40 years to approximately 5 deaths per million in the >85 year age group.[1]
- Children <4 years have a heat-related death rate of 0.3 per million; children >4 years have a heat-related death rate of 0.05 per million.[1]
- Heat-related illness and deaths are clearly related to high environmental temperature, and increased numbers have been seen in urban heat waves in the United States and elswhere.[2,3]

PATHOPHYSIOLOGY

- The pathophysiologic effects caused by heat-related injury result from the imbalance between heat production and heat loss. Through the four mechanisms of radiation, convection, conduction, and evaporation, the body is able to maintain a core temperature within a narrow range.
- Radiation, which is heat transferred by electromagnetic waves, is the primary mechanism of heat loss when the air temperature is lower than the body temperature. This is about 65 percent of cooling in such an environment.
- Convection is heat exchange between a surface and a medium, usually air. This accounts for 10 to 15 percent of cooling; however, when the ambient temperature around the body exceeds the body's temperature, convection can be a source of heat gain.
- Conduction, which is heat exchange between two surfaces in direct contact, accounts for only 2 percent of heat loss; however, in cases of water submersion, there is a 25-fold increase in heat exchange.
- Evaporation is the conversion of liquid to a gaseous phase at the expense of energy. Humans primarily disperse heat by sweating when the environment has a higher temperature than the body. Conditions of high humidity and dehydration can prevent effective evaporation.[4]

CLINICAL FEATURES

- Minor heat-related illness presents with heat edema, prickly heat, heat syncope, heat cramps, heat tetany, and heat exhaustion. The patient's mental status and neurologic exam remain intact.
- Heat edema is a self-limited process manifested by mild swelling of the hands and feet. It resolves within days to weeks.
- Prickly heat, or heat rash, is a pruritic, maculopapular, erythematous rash over clothed areas. It is an acute inflammation of the sweat ducts caused by blockage of the sweat pores by macerated stratum corneum.[5]
- Heat syncope is a variant of postural hypotension resulting from the cumulative effect of peripheral vasodilation, decreased vasomotor tone, and relative volume depletion.
- Heat cramps are painful, involuntary, spasmodic contractions of skeletal muscles, usually in the calves and legs. This results from deficiency of sodium, postassium, and fluid at the cellular level.
- Heat tetany is characterized by hyperventilation resulting in respiratory alkalosis, paresthesia, and carpopedal spasm.
- Heat exhaustion is an obscure syndrome characterized by nonspecific symptoms such as dizziness, weakness, malaise, light-headedness, fatigue, nausea, vomiting, headache, and myalgia. Clinical manifestations include syncope, orthostatic hypotension, sinus tachycardia, tachypnea, diaphroesis, and hyperthermia (up to 40°C or 104°F). There are no neurologic or mental status changes.
- Heat stroke patients exhibit signs and symptoms of heat exhaustion along with central nervous system (CNS) dysfunction (mental status changes or neurologic deficits) and temperatures above 40°C (104°F). Anhidrosis is classically described, but is not always present.

DIAGNOSIS AND DIFFERENTIAL

- Heat stroke should be considered in any patient with an elevated body temperature and altered mental status; heat exhaustion is a diagnosis of exclusion.
- The differential diagnosis includes infection (sepsis, meningitis, encephalitis, malaria, typhoid fe-

ver, and brain abscess), toxins [anticholinergics, phenothiazines, salicylates, phencyclidine (PCP), cocaine, amphetamines, and alcohol withdrawal], endocrine and metabolic emergencies (thyrotoxicosis and diabetic ketoacidosis), primary CNS disorders (status epilepticus, stroke, and intracranial hemorrhage), neuroleptic malignant syndrome, and malignant hyperthermia.
- Laboratory studies should include a complete blood cell count, electrolytes, blood urea nitrogen, creatinine levels, hepatic panel, coagulation studies, creatinine kinase, urinalysis, urine myoglobin, blood cultures, chest radiograph, arterial blood gas analysis, electrocardiogram, and pregnancy test.
- A computed tomography scan of the head and lumbar puncture should be considered in evaluating for CNS pathology.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of heat emergencies consists of initial stabilization, rapid cooling, and evaluation of underlying injuries or illnesses.
- ABCs should be assessed, and high-flow supplemental oxygen, cardiac monitoring, intravenous access, cycling blood pressure, pulse oximetry, and continuous core body temperature monitoring with a rectal probe should be provided.
- Patients should be intubated if mental status changes are significant and if they lack the ability to protect their airway or they have evidence of respiratory failure. Isotonic normal saline or lactated Ringer's should be given for volume depletion. Central venous pressure and urine output should be monitored.
- Evaporation cooling is the most efficient and practical means of cooling patients in the emergency department. Patients must be disrobed, sprayed with water, and placed in front of cooling fans.[6] Ice packs may cause shivering but can be applied to groin and axilla. Shivering should be treated with benzodiazepines or phenothiazines (chlorpromazine 25 mg intramuscularly). Active core cooling with cold gastric and peritoneal lavage or cardiopulmonary bypass are the most rapid cooling measures and should be reserved for cases that are recalcitrant to all other measures. Cooling should be discontinued after reaching 40°C to avoid ''overshoot hypothermia.''
- Patients with true heat stroke should be observed for further end-organ damage in an intensive care unit setting. Patients at the extremes of age or with underlying comorbid diseases who suffer heat exhaustion should be admitted. All other minor heat illnesses may be discharged home for outpatient follow-up.

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118 BITES AND STINGS

Alex G. Garza

HYMENOPTERA (BEES AND WASPS)

CLINICAL FEATURES

- Most of the allergic reactions reported each year occur from *Vespidae* (wasp, hornet, and yellow jacket) stings.
- The most common response to Hymenoptera venom consists of pain, slight erythema, edema, and pruritus at the sting site.
- A local reaction consists of marked and prolonged edema contiguous with the sting site. Although there are no systemic signs or symptoms, a severe

local reaction may involve one or more neighboring joints. When local reactions become increasingly severe, the likelihood of future systemic reactions appears to increase.
- Toxic reactions are nonantigenic responses to multiple stings. Symptoms of a toxic reaction may resemble anaphylaxis, but there is generally greater frequency of nausea, vomiting, and diarrhea while urticaria and bronchospasm are not present.
- Systemic or anaphylactic reactions are true allergic reactions that range from mild to fatal. In general the shorter the interval between the sting and the onset of symptoms, the more severe the reaction. Initial symptoms usually consist of itching eyes, urticaria, and cough. As the reaction progresses, patients may experience respiratory failure and cardiovascular collapse. The majority of reactions occur within the first 15 min and nearly all occur within 6 h. There is no correlation between the systemic reaction and the number of stings.
- Delayed reactions appear 10 to 14 days after a sting and consist of serum sickness–like signs and symptoms, including fever, malaise, headache, urticaria, and polyarthritis.[1,2]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The treatment of all Hymenoptera encounters is the same. Any bee stinger remaining in the patient should be removed immediately and the wound cleansed.
- Erythema and swelling seen in local reactions may be difficult to distinguish from cellulitis. As a general rule, infection is present in a minority of cases.
- For minor local reactions, oral antihistamines and analgesics may be the only treatment needed.
- More severe reactions—such as chest constriction, nausea, presyncope, or a change in mental status—require treatment with 1 : 1000 epinephrine SQ: 0.3 to 0.5 mL for an adult and 0.01 mL/kg for a child (0.3 mL maximum). Some patients may require a second epinephrine injection in 10 to 15 min.
- Parenteral H_1- and H_2-receptor antagonists (e.g., diphenhydramine and ranitidine, respectively) and steroids (e.g., methylprednisolone) should be rapidly administered.
- Bronchospasm responds to inhaled beta agonists (e.g., albuterol).
- Hypotension should be treated aggressively with crystalloid; dopamine and epinephrine infusions may be required.
- Patients with minor symptoms who respond well to conservative measures may be discharged after being monitored for several hours; severe reactions require admission.
- All patients with Hymenoptera reactions should be referred to an allergist for further evaluation.

ANTS (FORMICIDAE)

- Fire ants swarm during an attack, and each sting results in a papule that evolves to a sterile pustule over 6 to 24 h.
- Local necrosis and scarring as well as systemic reactions can occur.
- Treatment is the same as for Hymenoptera stings; appropriate referral should be made for desensitization therapy.[3]

ARACHNIDA (SPIDERS, SCABIES MITES, CHIGGERS, AND SCORPIONS)

BROWN RECLUSE SPIDER

- The bite of the brown recluse spider causes a mild erythematous lesion that may become firm and heal over several days to weeks. Occasionally a severe reaction with immediate pain, blister formation, and bluish discoloration may occur.
- These lesions often become necrotic over the next 2 to 4 days and form an eschar from 1 to 30 cm in diameter.
- Loxoscelism is a systemic reaction that may occur 1 to 2 days after envenomation. Symptoms include fever, chills, vomiting, arthralgias, myalgias, petechiae, and hemolysis; severe cases progress to seizure, renal failure, disseminated intravascular coagulation, and death.
- Treatment for the brown recluse spider's bite includes wound care, tetanus prophylaxis, analgesics, and dapsone. The roles of dapsone (50 to 200 mg/d) and hyperbaric oxygen have been challenged, but they may prevent some ongoing local necrosis.
- Surgery is reserved for lesions greater than 2 cm and is deferred for 2 to 3 weeks following the bite.
- Patients with systemic reactions and hemolysis must be hospitalized for consideration of blood transfusion and hemodialysis.[4,5]

BLACK WIDOW SPIDER

- The bite of the black widow spider is initially painful, and within 1 h, the patient may experience erythema (often target-shaped), swelling, and diffuse muscle cramps.
- Large muscle groups are involved, and painful cramping of the abdominal wall's musculature can mimic peritonitis. Severe pain may wax and wane for up to 3 days, but muscle weakness and spasm can persist for weeks to months.
- Serious acute complications include hypertension, respiratory failure, shock, and coma.
- Initial therapy includes local wound treatment and supportive care. Analgesics and benzodiazepines will relieve pain and cramping, and some patients may benefit from intravenous calcium gluconate, although controlled data are lacking.
- An antivenin derived form horse serum is effective for severe envenomation. If the patient tolerates placement of a standard cutaneous test dose, the usual intravenous dose is one to two vials over 30 min.[6]

SCABIES MITE

- The bites of *Sarcoptes scabiei* are concentrated in the web spaces between fingers and toes. Other common areas include the penis and the face and scalp in children. Transmission is usually by direct contact.
- The distinctive feature of scabies infestation is intense pruritus with "burrows." These white, threadlike channels form zigzag patterns with small gray spots at the closed end, where the parasite rests. Undisturbed burrows can be traced with a hand lens; the female mite is easily scraped out with a blade edge. Associated vesicles, papules, crusts, and eczematization may obscure the diagnosis.
- Adult treatment of scabies infestation consists of a thorough application of permethrin from the neck down; infants may require additional application to the scalp, temple, and forehead.
- Reapplication is necessary only if mites are found 2 weeks after successful therapy.

CHIGGERS

- Chiggers are tiny mite larvae that cause intense pruritus when they feed on host epidermal cells.
- Itching begins within a few hours, followed by a papule that enlarges to a nodule over the next 1 to 2 days. Single bites can also cause soft tissue edema, while infestation has been associated with fever and erythema multiforme.
- Children who have been sitting on lawns are prone to chigger lesions in the genital area.
- The diagnosis of chigger bites can be made on the basis of typical skin lesions in the context of a known outdoor exposure.
- Treatment consists of symptomatic relief with antihistamines; topical or oral steroids may be required in more severe cases. Annihilation of the mites requires lindane, permethrin, or crotamiton topical applications.

SCORPION

- Of all North American scorpions, only the bark scorpion (*Centruroides exilicauda*) of the western United States is capable of producing systemic toxicity.
- The venom of *C. exilicauda* causes immediate burning and stinging, although no local injury is visible. Systemic effects are infrequent and occur mainly at the extremes of patient age. Findings may include tachycardia, excessive secretions, roving eye movements, opisthotonos, and fasciculations.
- Treatment is supportive, including local wound care. Reassurance is also important, since many patients harbor misconceptions about the lethality of scorpion stings.
- Patients with pain in the absence of other toxic symptoms may be briefly observed before they are discharged home with analgesics. The application of ice often provides immediate relief of local pain. Muscle spasm and fasciculations respond promptly to benzodiazepines.[7]

FLEAS

- Flea bites are frequently found in zigzag lines, especially on the legs and in the waist area. The lesions have hemorrhagic puncta surrounded by erythematous and urticarial patches.
- Pruritus is intense; even after the lesions clear, dull red spots may persist.
- The main concern in the treatment of flea bites is the possibility of secondary infection. Children may develop impetigo as a complication. If secondary infection develops, topical or oral antibiotics may be needed.
- Oral antihistamines and starch baths at bedtime are recommended to relieve discomfort and pre-

vent scratching. For severe discomfort, application of a topical steroid cream or spray may be necessary.

LICE

- Body lice concentrate around the waist, shoulders, axillae, and neck. Pubic lice are spread by sexual contact.
- The lice and their eggs can often be found in the seams of clothing. Their bites produce red spots that progress to papules and wheals.
- These spots are so intensely pruritic that linear scratch marks are suggestive of infestation.
- Reactions to lice saliva and feces may cause fever, malaise, and lymphadenopathy.
- Permethrin is the primary treatment of body lice infestation. Treatment of hair infestation requires a thorough application of pyrethrin with piperonyl butoxide and mandatory reapplication in 10 days.
- Clothing, bedding, and personal articles must be sterilized in hot (>52°C or >126°F) water to prevent reinfestation.

KISSING BUG, PUSS CATERPILLAR, AND BLISTER BEETLE

KISSING BUG

- The *Triatoma* genus, commonly known as the kissing bug, is found mainly in the southeastern and Pacific Coast regions of the United States. These insects feed on blood and attack the exposed surface of a sleeping victim, commonly on the face.
- Bites are often multiple and result in wheals or hemorrhagic papules and bullae. Anaphylaxis commonly occurs in the sensitized individual.
- Treatment consist of local wound care and analgesics. Allergic reactions must be treated as previously outlined for Hymenoptera envenomation.

PUSS CATERPILLAR

- The puss caterpillar has stinging spines on its body that provoke immediate, intense, and rhythmic pain. Local edema and pruritus with vesicles, red blotches, and papules may follow.
- Infrequently, fever, muscle cramps, anxiety, and shock-like symptoms may occur. Lymphadenopathy with local desquamation may develop in a few days.
- Treatment consists of immediate spine removal with cellophane tape. Intravenous calcium gluconate, 10 mL of 10% solution, is effective in relieving pain. Mild cases may respond to an antihistamine.[8]

BLISTER BEETLE

- The only beetle of clinical significance for envenomation in humans is the blister beetle. Blister beetles are found throughout the United States and include beetles known as Spanish fly. When disturbed or crushed on the skin, they exude a vesicating agent called cantharidin that can penetrate the epidermis to produce irritation and blistering within a few hours of contact.
- If ingested, cantharidin can produce intense nausea, vomiting, diarrhea, and abdominal cramps. Initial contact with the beetle produces a burning, tingling sensation and a mild rash. Within a few hours, elongated vesicles and bullae develop from a few millimeters to several centimeters in diameter.
- Blebs erupt 2 to 5 h after contact and can be hemorrhagic and painful. A severe chemical conjunctivitis can occur if cantharidin contacts the eyes from contaminated hands.
- Treatment consists of protecting the bullae from secondary infection with occlusive dressings. Large bullae should be drained and antibiotic ointment applied. Application of steroid creams to blebs may be helpful.[9]

RATTLESNAKE

- There are approximately 8000 venomous snakebites each year in the United States, but only about 10 deaths result. The only venomous North American snakes are the pit viper (Crotalidae family; e.g., rattlesnake, copperhead, water moccasin, and massasauga) and coral snakes (Elapidae family).

CLINICAL FEATURES

- Pit vipers are identified by their two retractable fangs and by the heat-sensitive depressions located bilaterally between each eye and nostril.
- Crotalid venom is a complex enzyme mixture that causes local tissue injury, systemic vascular damage, hemolysis, fibrinolysis, and neuromuscular dysfunction, resulting in a combination of local and systemic effects.

- Crotalid venom quickly alters blood vessel permeability, leading to loss of plasma and blood into the surrounding tissue and causing hypovolemia. It also consumes fibrinogen and platelets, causing a coagulopathy.
- In some species, specific venom fractions block neuromuscular transmission, leading to ptosis, respiratory failure, and other neurologic effects.
- The effects of the envenomation depend on the size and species of the snake, the age and size of the victim, the time elapsed since the bite, and the characteristics of the bite itself.
- Bites that seem innocuous at first may rapidly become severe. The hallmark of pit viper envenomation is fang marks with local pain and swelling.
- The cardinal manifestations of crotalid venom poisoning are the presence of one or more fang marks, localized pain, and progressive edema extending from the bite site.
- In general, all affected patients experience swelling within 30 min, though some may take up to 12 h.[10–13]

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is made from clinical findings and corroborating laboratory findings.
- There are three classes of criteria that determine the severity of a rattlesnake bite: (1) degree of local injury (swelling, pain, and ecchymosis); (2) degree of systemic involvement (hypotension, tachycardia, and paresthesia); and (3) evolving coagulopathy [thrombocytopenia, elevated international normalized ratio (INR), and hypofibrinogenemia]. Abnormalities in any of these three areas indicate that envenomation has occurred. Conversely, the absence of any clinical findings after 8 to 12 h effectively rules out venom injection.
- The envenomation itself is graded on an evolving continuum. Minimal envenomation describes cases of local swelling, with no systemic signs or laboratory abnormalities.
- Moderate envenomation causes increased swelling that spreads from the site. These patients may also have systemic signs such as nausea, paresthesia, hypotension, and tachycardia. Coagulation parameters may be abnormal, but there is no significant bleeding.
- Severe envenomation causes extensive swelling, potentially life-threatening systemic signs (hypotension, altered mental status, and respiratory distress), and markedly abnormal coagulation parameters that may result in hemorrhage.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The patient should minimize physical activity, remain calm, and immobilize any bitten extremity in the neural position below the level of the heart.
- Incision of the wound is contraindicated, as are ice packs, tourniquets, and electric shocks.
- Intravenous access should be established. Laboratory studies such as complete blood count (CBC), INR, coagulation profile, urinalysis (UA), and blood typing should be obtained.
- Local wound care and tetanus immunization should be given, but prophylactic antibiotics and steroids have no proven benefit.
- Limb circumference at several sites above and below the wound should be checked every 30 min, and the border of advancing edema should be marked.
- Any patient with progressive local swelling, systemic effects, or coagulopathy should immediately receive equine-derived antivenin (Crotalidae) polyvalent.
- An intradermal skin test (0.03 mL of 1:10 antivenin) must be placed before the patient is treated; a 10-mm wheal within 30 min is considered positive. A positive skin test warrants a risk/benefit analysis before any antivenin is administered; these cases should be discussed with a toxicologist at once. The starting dose of antivenin is 10 vials IV. Severe cases require 20 vials. Dosing regimens are exactly the same for both children and adults, though the amount of fluid in which the antivenin is mixed will need to be adjusted accordingly.
- The antivenin package insert will guide administration, and the physician must be prepared to treat severe allergic and anaphylactic reactions. The endpoint of antivenin therapy is arrest of progressive symptoms and coagulopathy. Additional 10-vial doses of antivenin are repeated if the patient's condition worsens or if the coagulopathy increases.
- Compartment syndromes may occur secondary to envenomation. Pressures over 30 mmHg require limb elevation and mannitol (1 to 2 g/kg IV over 30 min) if no contraindications exist.
- Repeated dosing of antivenin is the most effective therapy for elevated compartment pressures. An additional 10 to 15 vials over 60 min should be given and the pressure reassessed. Persistently elevated pressure may require consultation for emergent fasciotomy.
- All patients with pit viper bites must be observed for at least 8 h. Patients with severe bites and

those receiving antivenin must be admitted to the intensive care unit.
- Patients with mild envenomation who have completed antivenin therapy may be admitted to the general ward.
- Patients with no evidence of envenomation after 8 to 12 h may be discharged.
- All patients who receive antivenin should also be counseled about serum sickness, since this occurs in nearly all patients at 7 to 14 days following therapy.

CORAL SNAKE

- North American coral snakes include the eastern, the Texas, and the Arizona coral snakes. All coral snakes are brightly colored, with black, red, and yellow rings. The red and yellow rings touch in coral snakes but are separated by black rings in nonpoisonous snakes, creating the well-known rhyme: ''Red on yellow, kill a fellow; red on black, venom lack.''
- Coral snake venom is primarily composed of neurotoxic compounds that do not cause marked local injury.
- Elapid bites produce primarily neurologic effects, including tremors, salivation, dysarthria, diplopia, and bulbar paralysis with ptosis, fixed and contracted pupils, dysphagia, dyspnea, and seizures. The immediate cause of death is paralysis of respiratory muscles.
- Signs and symptoms may be delayed up to 12 h.
- Patients should be admitted to the hospital for 24 to 48 h for observation.
- The effects of coral snake venom may develop hours after a bite and are not easily reversed. It is suggested that three vials of the antivenin (*Micrurus fulvius*) be administered to patients who have definitely been bitten because it may not be possible to prevent further effects or reverse effects that have already developed. The patient must be observed closely for signs of respiratory muscle weakness and hypoventilation. Prolonged ventilatory support may be required in severe cases.[14]

GILA MONSTER

- Gila monsters are slow-moving lizards that inhabit the desert in the southwestern United States. They possess venom as potent as that of the rattlesnake but lack the apparatus to inject it effectively.
- Gila monsters bite tenaciously and may be difficult to remove from the bitten extremity. Most bites result in local pain and swelling only, which worsens over several hours and then subsides over several more hours.
- Occasionally, a more severe syndrome of systemic toxicity develops, including weakness, light-headedness, paresthesia, and diaphoresis. Severe hypertension may occur, which also resolves over several hours.
- Treatment involves removal of the reptile from the bite site. The Gila monster will often loosen its grip when no longer suspended in midair. Standard local wound care is sufficient, and any teeth in the wound should be removed.

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119 TRAUMA AND ENVENOMATION FROM MARINE FAUNA

Keith L. Mausner

EPIDEMIOLOGY

- Exposure to hazardous marine fauna occurs primarily in tropical areas, but dangerous marine animals are encountered to a significant degree as far north as 50° N latitude.
- Contrary to common belief, shark attacks are infrequent; less than 100 attacks are reported annually worldwide, with 10 or fewer fatalities.[1]

CLINICAL FEATURES

- Coral cuts are the most common underwater injury. Local stinging pain, erythema, and pruritus may progress to cellulitis with ulceration, tissue sloughing, lymphangitis, and reactive bursitis.
- Marine animals reported in attacks include sharks, great barracudas, moray eels, giant groupers, sea lions, seals, crocodiles, alligators, needle fish, wahoos, piranhas, and triggerfish. Injuries include abrasions, puncture wounds, lacerations, and crush injuries.
- Ocean water contains many potentially pathogenic bacteria, including *Aeromonas hydrophila, Bacteroides fragilis, Chromobacterium violaceum, Clostridium perfringens, Erysipelothrix rhusopathiae, Escherichia coli, Mycobacterium marinum, Salmonella enteritidis, Staphylococcus aureus, Streptococcus* species, and *Vibrio* species.[2]
- *Vibrio vulnificus* and *V. parahaemolyticus* may cause severe cellulitis, myositis, or necrotizing fasciitis.
- *V. vulnificus* is also associated with sepsis in chronically ill patients, especially those with liver disease; it has 60 percent mortality.
- *Aeromonas hydrophila* can cause rapidly developing cellulitis or necrotizing myositis.
- The invertebrates include *five phyla:* Cnidaria, Porifera, Echinodermata, Annelida, and Mollusca.
- Cnidaria includes fire corals, Portuguese men-of war, jellyfish, sea nettles, and anemones. Most reactions are localized, with pain, erythema, and other cutaneous manifestations.[3] Anemones, jellyfish, and men-of-war may cause severe systemic reactions that occur in minutes to hours.[4]
- Porifera are sponges that produce allergic dermatitis. In severe cases erythema multiforme with systemic manifestations may occur.
- Echinodermata includes starfish, sea urchins, and sea cucumbers.[5]
- Sea urchin spines produce immediate pain, then erythema, myalgia, and local swelling. Severe envenomation may cause nausea, paresthesia, paralysis, abdominal pain, syncope, respiratory depression, and hypotension.
- Starfish spines cause pain, bleeding, and edema; in severe envenomation, nausea, vomiting, paresthesia, and paralysis may be seen.
- Sea cucumbers produce mild contact dermatitis, but eye exposure may result in a severe reaction.
- Annelida includes bristleworms, which embed bristles in the skin causing pain and erythema.[5]
- Mollusca includes cone shells and octopuses.[5]
- Mild cone shell envenomation is similar to a bee sting; severe reactions include paralysis and respiratory failure.
- Octopus bites may cause paresthesia, paralysis, and respiratory failure.
- Venomous spined vertebrates include the stingray, scorpionfish, catfish, weeverfish, surgeonfish, horned sharks, toadfish, ratfish, rabbitfish, stargazers, and leatherbacks.
- Stingray envenomation is the most common among the vertebrates.[5] The spine produces a puncture or laceration and may be retained in the wound, causing an intense local painful reaction. Systemic effects may include weakness, nausea, vomiting, diarrhea, syncope, seizures, paralysis, hypotension, and dysrhythmias.
- Scorpionfish envenomation may produce paralysis of skeletal, smooth, and cardiac muscle.
- Sea snakes are the most abundant venomous reptiles.[5] They are found in tropical and warm temperate areas of the Pacific and Indian Oceans. Sea snake venom contains a paralyzing neurotoxin and a myotoxin. Myalgia, ophthalmoplegia, ascending

paralysis, and respiratory failure may occur. Death is commonly due to respiratory failure.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway, breathing, circulation, treatment of life-threatening injuries, and correction of hypothermia take priority in the initial management.
- Wounds should be copiously irrigated and devitalized tissue debrided. Soft tissue radiographs may help locate foreign bodies. Most wounds should undergo delayed primary closure.
- Prophylactic antibiotics are not indicated for minor wounds in healthy patients.[6] Immunocompromised patients and those with grossly contaminated or extensive lacerations require antibiotics; in high-risk patients the first dose should be parenteral.
- Infected wounds may have retained foreign bodies. Antibiotic coverage should account for *Staphylococcus* and *Streptococcus* species.
- In ocean-related infections, *Vibrio* species should be covered with a third-generation cephalosporin, trimethoprim-sulfamethoxazole, doxycycline, a fluoroquinolone, an aminoglycoside, or chloramphenicol.[6]
- In Cnidaria envenomation, the wound should be rinsed with saline solution. Acetic acid (vinegar, 5%) or isopropyl alcohol (40 to 70%) inactivate the venom. The deactivated nematocyst should be removed by applying shaving cream or talcum powder and shaving with a razor. Corneal envenomation should be treated with topical steroids.
- Sponge-induced dermatitis should be treated with gentle drying of the skin and removal of spicules with adhesive tape. Acetic acid treatments 3 to 4 times a day for 10 to 30 min may be helpful.
- Echinodermata envenomation is treated by removing spines and with hot water immersion (45°C, or 113°F) for 30 to 90 min. Acetic acid or isopropyl alcohol may provide symptomatic relief in sea cucumber envenomation.
- In Annelida envenomation, the bristles should be removed with tape or forceps.
- With spined vertebrate envenomations, the area should be immersed in hot water, spines removed, and wound explored and debrided.
- With sea snake bites, the injured area should be kept immobilized and dependent. Application of local pressure with an elastic bandage may help sequester the venom. Antivenin is indicated for symptomatic patients and may be beneficial up to 36 h after envenomation. Hemodialysis may also be beneficial. If no symptoms develop 8 h after exposure, then envenomation did not occur.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 190, ''Trauma and Envenomations from Marine Fauna,'' by Paul S. Auerbach.

120 HIGH ALTITUDE MEDICAL PROBLEMS

Keith L. Mausner

EPIDEMIOLOGY

- The incidence of acute mountain sickness (AMS), as well as high altitude cerebral edema (HACE) and high altitude pulmonary edema (HAPE), is influenced primarily by the rapidity of ascent and sleeping altitude.
- An AMS incidence between 17 and 40 percent has been reported at resorts with altitudes between 2200 and 2700 m (7200 and 9000 ft).[1]
- The incidence of HAPE is much lower than that of AMS. HAPE has been reported in less than 1 in 10,000 skiers in Colorado. The incidence of HACE is lower than that of HAPE.

- Susceptibility to AMS is linked to a low hypoxic ventilatory response and low vital capacity; susceptible individuals are prone to recurrence on return to high altitude. Partially acclimatized individuals who live at intermediate altitudes of 1000 to 2000 m (3280 to 6560 ft) are less likely to develop AMS on ascent to higher altitude.
- Risk factors for development of HAPE include heavy exertion, rapid ascent, cold, excessive salt intake, use of sleeping medication, and a prior history of HAPE.

PATHOPHYSIOLOGY

- Acute mountain sickness is caused by hypobaric hypoxia, and HAPE and HACE can be viewed as extreme progression of the same pathophysiology.
- Hypoxemia increases cerebral blood flow and cerebral capillary hydrostatic pressure, resulting in fluid shifts and either mild cerebral edema in AMS or severe cerebral edema in HACE.[2] Hypoxemia also raises pulmonary artery pressure.
- Increased intracranial pressure elevates sympathetic nervous system activity, which in turn decreases the compliance of pulmonary arteries, promotes pulmonary venous constriction, and increases pulmonary capillary permeability. In addition, increased sympathetic nervous system activity is associated with decreased urine output, mediated by renin, angiotensin II, and aldosterone, as well as vasopressin. This leads to fluid retention and results in elevated capillary hydrostatic pressure in lung, brain, and peripheral tissues.[2]

CLINICAL FEATURES

- Acute mountain sickness is usually seen in unacclimated people making a rapid ascent to over 2000 m (6600 ft) above sea level.
- The earliest AMS symptoms are light-headedness and mild breathlessness. Other symptoms similar to a hangover may develop within 6 h after arrival at altitude, but may be delayed as long as 1 day. These include bifrontal headache, anorexia, nausea, weakness, and fatigue.
- Progression of AMS is indicated by worsening headache, vomiting, oliguria, dyspnea, and weakness. Postural hypotension and peripheral and facial edema may be seen. Localized pulmonary rales are noted in 20 percent of cases. Low-grade fever may also be seen. Funduscopy reveals tortuous and dilated veins; retinal hemorrhages are common at altitudes over 5000 m (16,400 ft).
- High altitude cerebral edema is an extreme progression of AMS and is usually associated with pulmonary edema. It presents with altered mental status, ataxia, stupor, and progression to coma. Focal neurologic signs such as third and sixth cranial nerve palsies may be present.
- High altitude pulmonary edema is the most lethal of the high altitude syndromes. Table 120-1 summarizes the classification, symptoms, and findings in the different stages of HAPE. Early recognition, descent, and treatment are essential to prevent progression.
- Chronic obstructive pulmonary edema patients may require supplemental O_2 or an increase in their usual O_2 flow rate.
- Patients with coronary artery disease do surprisingly well at high altitude, but may be at risk of early onset of angina during their first few days at high altitude. However, after acclimatization there may be no significant difference in the occurrence of angina compared with exertion at sea level.[3] There may be some risk of worsening of congestive heart failure at high altitudes.
- Pregnant long-term high altitude residents have an increased risk of hypertension, low-birth-weight infants, and neonatal jaundice, but no increase in pregnancy complications has been reported in visitors to high altitude who engage in reasonable activities.

DIAGNOSIS AND DIFFERENTIAL

- The differential diagnosis of the high altitude syndromes includes hypothermia, carbon monoxide poisoning, pulmonary or central nervous system infections, dehydration, and exhaustion.
- High altitude cerebral edema may be difficult to distinguish in the field from other high altitude neurologic syndromes.
- High altitude neurologic syndromes distinct from HACE include high altitude syncope, cerebrovascular spasm (migraine equivalent), cerebrovascular thrombosis, transient ischemic attack, and cerebral hemorrhage. Findings in these syndromes are usually more focal than in HACE.
- High altitude pulmonary edema must be distinguished from pulmonary embolus, cardiogenic pulmonary edema, and pneumonia. Low-grade fever is common in HAPE and may make it difficult to distinguish from pneumonia.
- A key to diagnosis of these syndromes is the clinical response to treatment.

TABLE 120-1 Severity Classification of HAPE

GRADE	SYMPTOMS	SIGNS	CHEST RADIOGRAPH
1, Mild	Dyspnea on exertion, dry cough, fatigue while moving uphill	Resting HR <100, resting RR <20, dusky nailbeds, localized rales, if any	Minor exudate involving less than one-fourth of one lung field
2, Moderate	Dyspnea, weakness, fatigue on level walking, raspy cough, headache, anorexia	HR 90–100, RR 16–30, cyanotic nailbeds, rales present, ataxia may be present	Some infiltrate involving 50% of one lung or smaller area of both lungs
3, Severe	Dyspnea at rest, productive cough, orthopnea, extreme weakness, stupor, coma, blood-tinged sputum	Bilateral rales, HR >110, RR >30, facial and nailbed cyanosis, ataxia	Bilateral infiltrates >50% each lung

SOURCE: Hultgren HN: High altitude pulmonary edema, in Staub NC (ed): *Lung Water and Solute Exchange.* New York, Marcel Dekker, 1978, pp 437–469.
ABBREVIATIONS: HR = heart rate; RR = respiratory rate.

FIELD AND EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Gradual ascent is effective at preventing AMS. A reasonable guideline for sea-level dwellers is to spend a night at 1500 to 2000 m (4920 to 6560 ft) before sleeping at altitudes above 2500 m (8200 ft). High altitude trekkers should allow 2 nights for each 1000-m (3280 ft) gain in sleeping altitude starting at 3000 m (9840 ft). Eating a high carbohydrate diet and avoiding overexertion, alcohol, and respiratory depressants may also help prevent AMS.
- Mild AMS usually improves or resolves in 12 to 36 h if further ascent is delayed, allowing acclimatization. Patients with mild AMS should not ascend to a higher sleeping elevation. Descent is indicated if symptoms persist or worsen. Immediate descent and treatment are indicated if there is a change in the level of consciousness, ataxia, or pulmonary edema. Descending 500 to 1000 m (1640 to 3280 ft) may provide prompt symptomatic relief.
- Oxygen relieves symptoms, and nocturnal low-flow O_2 (0.5 to 1 L/min) is helpful.
- Acetazolamide causes a bicarbonate diuresis, leading to a mild metabolic acidosis. This stimulates ventilation and pharmacologically produces an acclimatization response. It is effective in prophylaxis and treatment. Indications for acetazolamide are (a) prior history of altitude illness; (b) rapid ascent to over 3000 m (10,000 ft); (c) treatment of AMS; and (d) symptomatic periodic breathing during sleep at altitude. Adult dose is 125 mg twice a day, continued until symptoms resolve, or for 3 to 4 days as prophylaxis. It should be restarted if symptoms recur.[4]
- Dexamethasone [4 mg orally (PO), intramuscularly (IM), or intravenously (IV) every 6 h] is effective in moderate to severe AMS. Tapering of the dose over several days may be necessary to prevent rebound.
- Aspirin or acetaminophen may improve headache. Prochlorperazine (5 to 10 mg IM or IV) may help with nausea and vomiting. Diuretics may be useful for treating fluid retention, but should be used with caution to avoid intravascular volume depletion.
- High altitude cerebral edema mandates immediate descent or evacuation. Oxygen and dexamethasone (8 mg PO, IM, or IV, then 4 mg every 6 h) should be administered. Furosemide (40 to 80 mg) may help reduce brain edema. Endotracheal intubation and hyperventilation may be necessary. Arterial blood gases should be monitored to prevent excessive lowering of pCO_2 (below 25 to 30 mmHg), which may cause cerebral ischemia.
- High altitude pulmonary edema also mandates immediate descent. Oxygen may be life-saving if descent is delayed. Nifedipine (10 mg PO every 4 to 6 h, or 30 mg extended-release every 12 h), as well as morphine and furosemide, may be effective. An expiratory positive airway pressure mask may be useful in the field and, without supplemental O_2, can increase oxygen saturation by 10 to 20%.

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121 DYSBARISM

Keith L. Mausner

PATHOPHYSIOLOGY

- Dysbarism is commonly encountered in scuba divers and refers to complications associated with changes in environmental ambient pressure and with breathing compressed gases.
- Diving pathophysiology is largely explained by three gas laws.
- Boyle's law states that the volume of a gas is inversely proportional to its pressure at a constant temperature. This is the basic mechanism of barotrauma, which results when a diver is unable to equalize pressures in air-filled cavities with ambient environmental pressure.
- Dalton's law states that the total pressure exerted by a mixture of gases is equal to the sum of the partial pressures of the component gases.
- Henry's law states that the amount of gas dissolved in a fluid is proportional to the pressure of the gas with which it is in equilibrium.
- Decompression sickness occurs because increased ambient pressure as a scuba diver descends causes an increase in the partial pressure of the inspired nitrogen in the breathing air. Due to Henry's law, nitrogen dissolves and accumulates in tissues. If ascent is too rapid, nitrogen comes out of solution abruptly, leading to bubble formation.

CLINICAL FEATURES

- Barotrauma is the most common diving-related affliction.
- Middle-ear squeeze, or barotitis media, is the most frequent form of barotrauma and is due to eustachian tube dysfunction during descent. The diver complains of ear fullness or pain. If pressure is not equalized or the dive is not aborted, the eardrum may rupture, resulting in a sensation of escaping air bubbles from the ear, with nausea and vertigo.
- On physical examination, there may be blood around the ear and mouth, mild conductive hearing loss, and tympanic membrane hemorrhage or perforation.
- External-ear squeeze is less common and is due to occlusion of the external ear canal by cerumen, debris, or earplugs.
- Sinus squeeze most commonly affects the frontal and maxillary sinuses.
- Inner ear barotrauma is the most rare ear affliction and occurs after an overly forceful Valsalva maneuver, or with very rapid descent. Clinical findings include tinnitus, vertigo, sensorineural hearing loss, and a feeling of ear fullness, nausea, and vomiting.
- Barotrauma during ascent is due to expansion of gas in the body cavities.
- Alternobaric vertigo (ABV) can occur during ascent due to unbalanced vestibular stimulation from unequal middle ear pressures.
- Gastrointestinal barotrauma during ascent presents with abdominal fullness, colicky abdominal pain, belching, and flatulence. Symptoms usually resolve with venting of bowel gas during ascent.
- Pulmonary overpressurization syndrome (POPS) during ascent may result in mediastinal and subcutaneous emphysema. After the dive, there may be gradual onset of increasing hoarseness, neck fullness, substernal chest pain, dyspnea, and dysphagia. Severe cases may present with syncope or pneumothorax.
- Air embolism may occur with too rapid of an ascent. Gas bubbles may enter the systemic circulation from ruptured pulmonary veins and occlude distal circulation. Findings may include cardiac arrest and dysrhythmias, and the neurologic examination may be consistent with stroke affecting multiple areas of cerebral circulation. Multiplegias, sensory disturbances, confusion, vertigo, seizures, or aphasia may be seen.
- Decompression sickness (DCS) is not a form of barotrauma. It is due to gas bubble formation as nitrogen comes out of solution in blood and tissues if ascent is too rapid without adequate time for decompression.
- Clinical findings in DCS include aching joint pain and neurologic abnormalities such as bladder dysfunction and lower extremity paraplegia, paraparesis, and paresthesias. Chest pain, cough, dyspnea, pulmonary edema, and shock may be seen.

- Risk factors for DCS include advanced age, obesity, dehydration, recent alcohol intake, cold water diving, strenuous underwater exercise, and multiple repetitive dives.
- Nitrogen narcosis is due to the anesthetic effect of nitrogen, similar to alcohol, at elevated partial pressures. It resolves with ascent, but is a common cause of diving accidents and may result in amnesia of the circumstances related to the accident.

DIAGNOSIS AND DIFFERENTIAL

For descent:
- Squeeze syndromes are the most common maladies.
- Breathing gas problems, such as carbon monoxide poisoning or hypoxia, are more likely to present early during descent.
- During the ''at-depth'' phase of a dive, the most likely problems are mechanical trauma, encounters with marine fauna, and nitrogen narcosis.

For ascent:
- Barotrauma and ABV are most likely to occur.
- Severe DCS may become symptomatic during ascent.

After surfacing:
- Severe symptom onset within 10 min is an air embolism unless proven otherwise.
- Onset of symptoms after 10 min is DCS until proven otherwise. Most cases of DCS present 1 to 6 h after surfacing, but may be delayed up to 48 h.
- Mild POPS and other forms of barotrauma may also present hours after a dive.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway, breathing, circulation, and immediately life-threatening injuries are first priority. High flow oxygen should be administered, and hypothermia should be treated.
- If air embolism is suspected, the patient should be placed in a supine position. Trendelenburg and left lateral decubitus positions are no longer recommended because of concerns about interference with breathing and worsening cerebral edema.
- If air embolism or DCS is suspected, recompression-chamber therapy should be initiated as quickly as possible. Aeromedical transport should be at an altitude of less than 1000 ft (305m) or in an aircraft that can be pressurized to 1 atm. Most DCS patients are volume depleted; intravenous fluids should be administered, if not otherwise contraindicated.
- Patients with middle ear and other squeeze syndromes should stop diving until symptoms resolve. Decongestants and antihistamines may be helpful. Antibiotics, such as amoxicillin, are indicated if the tympanic membrane is ruptured, and diving is contraindicated until it has healed. Sinus squeeze should be treated similarly to middle ear squeeze. Antibiotics are usually indicated for frontal sinus squeeze. External ear squeeze is treated by keeping the canal dry; antibiotics should be administered if there is evidence of infection or tympanic membrane rupture.
- Inner ear barotrauma usually mandates otolaryngology consultation since surgical repair may be indicated; these patients should avoid straining and be at bed rest with the head elevated.
- Pulmonary overpressurization syndrome may require needle decompression and tube thoracostomy if pneumothorax is present. This syndrome usually resolves with rest and supplemental oxygen and rarely requires recompression therapy.

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122 NEAR DROWNING

Stefanie R. Seaman

EPIDEMIOLOGY

- Near drowning is the third leading cause of accidental death in the United States. Fresh water drowning is more common than saltwater.

- Children under the age of 4 make up a large number of deaths. The next group at risk is teenagers, followed by the elderly from bathtub drowning.

PATHOPHYSIOLOGY

- Death occurs from respiratory failure and ischemic neurologic injury after submersion.
- Hypoxia can occur from ''wet drowning,'' which consists of flooding of alveoli and impaired gas exchange. ''Dry drowning'' refers to laryngospasm and glottic closure.
- While saltwater and freshwater wash surfactant away, freshwater changes the surface tension properties of surfactant. This leads to atelectasis, ventilation and perfusion mismatch, and breakdown of the alveolar capillary membrane.
- Hypoxemia has been shown to occur with aspiration of 2.2 mL/kg of water. Noncardiogenic pulmonary edema occurs from direct pulmonary injury, surfactant loss, pulmonary contaminants, and cerebral hypoxia.
- Electrolyte abnormalities in near drownings are seldom significant. Rarely, hemolysis or disseminated intravascular coagulation occur.
- Poor perfusion and hypoxemia lead to metabolic acidosis.

CLINICAL FEATURES

- Respiratory failure and neurologic injury predominate.
- Respiratory insufficiency is evidenced by dyspnea, tachypnea, or accessory muscle use.
- On physical examination, there may be wheezing, rales, or rhonchi.
- Neurologic status may be impaired. Hypothermia is common and may be severe.

DIAGNOSIS AND DIFFERENTIAL

- Associated injuries should be sought, especially cervical spine injuries. The majority of spinal injuries are to the lower cervical spine after diving. Subtle signs in the evaluation may include paradoxical breathing, flaccidity, or unexplained hypotension or bradycardia.
- Essential tests include chest radiograph and arterial blood gas (ABG) analysis. Electrolytes, complete blood cell count, and renal function should be measured, as the clinical picture dictates.
- Precipitating events, such as those of a cardiovascular, neurologic, or metabolic (hypoglycemia) nature, should be considered.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway, ventilation, and oxygenation should be assessed first. Stabilization and evaluation of the cervical spine must be performed concurrently.
- All patients should receive supplemental oxygen. They should also have cardiac monitoring, an intravenous line, and continuous pulse oximetry.
- Intubation and mechanical ventilation with high flow oxygen (40 to 50 percent) are indicated for persistent hypoxia (Pa_{O_2} <60 mmHg in adults, Pa_{O_2} <80 mmHg in children). Positive end-expiratory pressure can assist mechanical ventilation.
- Following intubation, a nasogastric tube and Foley catheter should be inserted. Core body temperature should be monitored.
- Bronchospasm, seizures, hypothermia, and dysrhythmias should be treated, as necessary. There is no established role for steroids or antibiotics.
- Patients with mild-to-moderate hypoxemia that is corrected by supplemental oxygen should be admitted and monitored. Patients with minimal or no symptoms and a normal chest radiograph and ABG may be observed in the emergency department for several hours and discharged if stable.
- Survival and neurologic outcome is unpredictable. The need for cardiopulmonary resuscitation or cardiac medications and unreactive pupils indicate a poor prognosis.
- Up to 24 percent of children admitted after experiencing cardiac arrest survive with an intact neurologic status.

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123 THERMAL AND CHEMICAL BURNS

Alex G. Garza

THERMAL BURNS

EPIDEMIOLOGY

- Approximately 1.25 million patients come to the emergency department with burn injuries in the United States each year and about 50,000 are hospitalized.
- The risk of burns is highest in the 18- to 35-year-old age group. There is higher incidence of scalds from hot liquids in children 1 to 5 years of age and in the elderly than in any other age group.

PATHOPHYSIOLOGY

- Thermal injury results in a spectrum of local and systemic homeostatic derangements that contribute to burn shock. Fluid and electrolyte abnormalities seen in burn shock are largely the result of alteration of cell membrane potentials with intracellular flux of water and sodium and extracellular migration of potassium secondary to dysfunction of the sodium pump.
- In burns of greater than 60 percent of the body surface area (BSA), depression of cardiac output is frequently observed with lack of response to aggressive volume resuscitation.
- A significant metabolic acidosis may be present in early stages of a large burn injury.
- Hematologic derangements associated with massive thermal injury vary from an increase in hematocrit with increased blood viscosity during the early phase followed by anemia from erythrocyte extravasation and destruction. However, blood transfusions are infrequently required for patients with isolated burn injury.
- Although many factors may influence prognosis, the severity of the burn, presence of inhalation injury, associated injuries, patient's age, preexisting disease, and acute organ system failure are most important.
- The burn wound is described as having three zones: (a) the zone of coagulation, where tissue is irreversibly destroyed with thrombosis of blood vessels; (b) the zone of stasis, where there is stagnation of the microcirculation; and (c) the zone of hyperemia, where there is increased blood flow. The zone of stasis can become progressively more hypoxemic and ischemic if resuscitation is inadequate.

CLINICAL FEATURES

- Burns are defined by their size and depth. Burn size is quantified as a percentage of BSA involved.
- The most common method of approximating the percentage of BSA burned is the "rule of nines" (Fig. 123-1). A more precise estimation, especially in infants and children, is to use a Lund Browder burn diagram (Fig. 123-2). Smaller burns can be estimated by using the area of the back of the patient's hand as approximately 1 percent of the BSA.
- Burn depth has been historically described in degree: first, second, and third. However, classification of burn depth according to the need for surgical intervention has become the accepted ap-

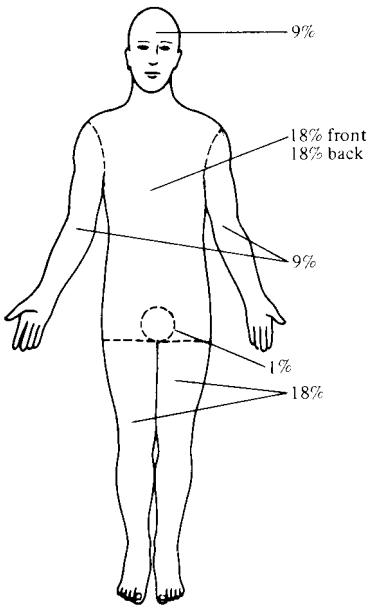
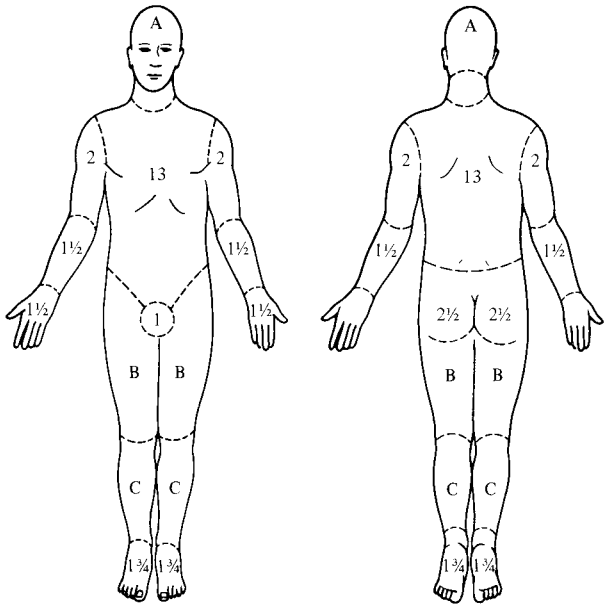


FIG. 123-1 Rule of nines to estimate percentage of burn.



Relative Percentages of Areas Affected by Growth (Age in Years)

	0	1	5	10	15	Adult
A: half of head	$9\frac{1}{2}$	$8\frac{1}{2}$	$6\frac{1}{2}$	$5\frac{1}{2}$	$4\frac{1}{2}$	$3\frac{1}{2}$
B: half of thigh	$2\frac{3}{4}$	$3\frac{1}{4}$	4	$4\frac{1}{4}$	$4\frac{1}{2}$	$4\frac{3}{4}$
C: half of leg	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{3}{4}$	3	$3\frac{1}{4}$	$3\frac{1}{2}$

Second degree _____ and

Third degree _____ =

Total percent burned ___

FIG. 123-2 Lund and Browder diagram to estimate percentage of pediatric burn.

proach in burn centers: superficial partial-thickness, deep partial-thickness, and full-thickness.

- Superficial partial-thickness burns have blistering exposed dermis that is red and moist with intact capillary refill, and they are very painful to touch. They heal in 14 to 21 days, and scarring is minimal.
- Deep partial-thickness burns extend into the deep dermis. The exposed dermis is white to yellow and does not blemish. Capillary refill and pain sensation are absent. Healing takes 3 weeks to 2 months, and scarring is common.
- Full-thickness burns involve the entire skin thickness. The skin is scarred, pale, painless, and leathery.
- Burns may also be associated with smoke inhalation injuries. Signs of pulmonary injury, which may have a delayed presentation for 12 to 24 h, include cough, wheezing, and respiratory distress. Thermal injury to the upper airway can occur and result in hoarseness, stridor, and rapidly occurring upper airway edema.
- Carbon monoxide poisoning should be suspected in all patients with smoke inhalation. Clinical signs include headache, vomiting, confusion, lethargy, and coma.

DIAGNOSIS AND DIFFERENTIAL

- Burns also can be diagnosed as major, moderate, or minor.
- Examples of major burns include full-thickness burns greater than 10 percent of the BSA or partial-thickness burns greater than 10 percent of the BSA. Burns involving the face, hands, feet, or perineum are also major.
- Minor burns include partial-thickness burns less than 10 percent of the BSA or full-thickness burns that are less than 2 percent of the BSA.
- Moderate burns are those not meeting criteria for either major or minor burns.
- With improvements in the treatment of burn shock and sepsis, inhalation injury has emerged as the main cause of mortality in the burn patient.
- The diagnosis of smoke inhalation is suggested by the history of a fire in an enclosed space. Physical examination signs include soot in the mouth or nose and carbonaceous sputum. The chest radiograph may be normal initially. Bronchoscopy may be helpful in determining the extent of injury.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Cardiac monitoring and two large-bore intravenous (IV) lines should be instituted. Oxygen 100% should be administered.
- If there are signs of airway compromise, the patient should be intubated. Indications for intubation includes full-thickness burns of the face or perioral region, circumferential neck burns, acute respiratory distress, stridor, progressive hoarseness or air hunger, respiratory depression or altered mental status, and supraglottic edema and inflammation on bronchoscopy or nasopharyngeal scope.
- Initial fluid resuscitation is based on the Parkland formula: 2 to 4 mL/kg/% BSA over 24 h. One-half the calculated amount is administered in the first 8 h from the time of injury, and the other one-half is administered over the subsequent 16 h. Lactated Ringer's solution is appropriate.
- Resuscitation should be monitored by assessment of the patient's urinary output (0.5 to 1.0 mL/kg/h) as well as other signs of perfusion.

- Important initial studies to be obtained include arterial blood gas analysis, carboxyhemoglobin level, complete blood cell count, and chest radiograph.
- Small burns should be covered with moist saline dressing and large burns with a sterile drape. Narcotic analgesia and a tetanus booster should be administered.
- Patients with circumferential deep burns of the limbs may develop compromise of the distal circulation. Distal pulses need to be monitored closely. If there is compromise to the circulation, escharotomy will be needed. The eschar needs to be incised on the midlateral side of the limb, allowing the fat to bulge through. This may be extended to the hand and fingers.
- If there are circumferential burns of the chest and neck, the eschar may cause mechanical restriction to ventilation. An escharotomy of the chest wall needs to be done to allow adequate ventilation. Incisions need to be made at the anterior axillary line from the level of the second rib to the level of the twelfth rib. These two incisions should be joined transversely so that the chest wall can expand.
- Criteria for transfer to a burn center are outlined in Table 123-1.
- Outpatient management of minor burns is appropriate. Blisters may be left intact or drained; the decision depends on size and location. Large blisters or those over very mobile joints should be debrided. Small blisters on nonmobile areas should be left intact. Burns should be cleansed and covered with a topical antibiotic (e.g., Silvadene or bacitracin).

TABLE 123-1 American Burn Association Criteria for Transfer to a Burn Unit

1. Partial- or full-thickness burns involving greater than 10% of body surface area (BSA) in patients under 10 or over 50 years of age.
2. Partial- or full-thickness burns of greater than 20% of BSA in other age groups.
3. Partial- or full-thickness burns with the threat of functional or cosmetic impairment that involve face, hands, feet, genitalia, perineum, or major joints.
4. Full-thickness burns of greater than 5% of BSA in any age group.
5. Electrical burns, including lightning injury.
6. Chemical burns with the threat of functional or cosmetic importance.
7. Inhalation injury with burns.
8. Circumferential burns of the extremities or chest.
9. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
10. Any burn patient with concomitant trauma, such as fracture.
11. Hospitals without qualified personnel or equipment for the care of children should transfer burned children to a burn center with these capabilities.

SOURCE: From American Burn Association: Hospital and prehospital resources for optimal care of patients with burn injury: Guidelines for development and operation of burn centers. *J Burn Care Rehab* 11:98, 1990, with permission.

CHEMICAL BURNS

EPIDEMIOLOGY

- Body sites most often burned by chemicals are the face, eyes, and extremities.
- The mortality rate for chemical burns is lower than it is for thermal burns, but wound healing and length of hospital stays are higher.

PATHOPHYSIOLOGY

- Acids or alkalis cause the majority of burns. Alkalis usually produce far more tissue damage than do acids.
- Acids in general cause coagulation necrosis with protein precipitation. The eschar limits spread of the agent.
- Alkalis produce liquefaction necrosis with loosening of material that allows deeper penetration of the unattached chemical into tissue.

CLINICAL FEATURES

- Clinical features of chemical burns depend on the agent, concentration, and duration of exposure. Superficial partial-thickness to full-thickness burns may result.
- An exception is hydrofluoric acid (HF), which rapidly penetrates intact skin and causes severe pain and progressive deep tissue damage. The involved skin may develop a blue-gray deep tissue damage with surrounding erythema. However, signs and symptoms may not develop until 12 to 24 h after exposure.
- Oxalic acid exposure may result in hypocalcemia and renal impairment.
- Since alkalis cause liquefaction necrosis, deep tissue destruction may result. Soft, gelatinous, brownish eschars may form. Wounds that initially appear superficial may progress to full-thickness burns.

- Pepper mace exposure causes mucous membrane, ocular, and upper airway irritation. Rarely, bronchospasm may occur.
- Chemical burns to the eyes result in redness, pain, and tearing. Corneal edema and ulceration may occur.
- Hypocalcemia, acidosis, hypotension, and renal and hepatic necrosis can occur, depending on the agents involved.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The first priority is to stop the burning process. Hydrotherapy is the cornerstone of the initial treatment for chemical burns. Copious irrigation is indicated for alkalis, acids, and pepper mace exposure. Dry chemical particles should be brushed away before irrigation. Treatment ideally should begin at the scene of the accident.
- For eye irrigation, 1 to 2 L normal saline should be used for a minimum of 1 h of continuous irrigation. Checking the conjunctival pH (normal 7.3 to 7.7) may be helpful in determining whether ocular burns need further irrigation. All significant ocular burns require an ophthalmology consult.
- Exceptions to irrigation include the elemental metals (sodium, lithium, and magnesium), which should be covered with mineral oil or extinguished with a class D fire extinguisher, and phenol, which should be decontaminated with PEG300, glycerol, or isopropyl alcohol.
- HF acid burns often require additional treatment. Calcium gluconate can be used topically if mixed with dimethyl sulfoxide. Subcutaneous and intradermal injections of a 5 to 10% solution into affected skin is recommended. A maximum dose of 0.5 mL of 10% calcium gluconate per square centimeter of burned skin is recommended.
- Cardiac monitoring and evaluation of electrolytes, renal functions, and calcium levels are indicated in significant HF, chromic, and oxalic acid burns.
- After initial specific measure, treatment should be as a thermal burn, with IV fluid replacement, analgesics, and tetanus prophylaxis.

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124 ELECTRICAL AND LIGHTNING INJURIES

Mark E. Hoffmann

EPIDEMIOLOGY

- There are an estimated 17,000 victims per year who require emergency treatment for electrical injuries. Lightning results in 1500 injuries and a 25 percent fatality rate per year.[1]
- Toddlers (household appliances and electrical cords), teenagers (risk-taking behavior), and those who work with electricity are the three largest risk groups for electrical injury.[2]
- Sports are associated with increased risk of lightning injury. Water sports account for the largest number of injuries and fatalities.[3]

PATHOPHYSIOLOGY

- Electrical current is the movement of electrical charge from one location to another; this flow is measured as amperes (A). Current flows when there is a potential difference between two locations; this is measured as volts (V). The intervening material resists the flow of electrical current; this is measured as ohms of resistance (R).

- Current can either be continuous in one direction (direct current, DC) or in alternating directions (alternating current, AC).
- The factors associated with severity of electrical injuries are current intensity, volts, type of current (AC or DC), tissue resistance, duration of contact, area of contact, current pathway through the body (vertical is more dangerous than horizontal), and environmental factors (water immersion).[4]
- Electrical energy is deposited into tissues, causing contact burns (entry and exit), thermal heating, flash burns, arc burns, flame thermal burns, blunt trauma, and prolonged muscular tetany.[5]
- Low-voltage AC currents will cause muscular tetany, and a person will actually continue to grasp the contact source and pull themselves closer, thus increasing the contact time.
- High-voltage AC and DC currents will cause a single violent muscular contraction, and the victim will tend to be thrown from the source of contact, thus increasing the risk of blunt trauma and blast injuries.
- Lightning is DC that imparts an instantaneous but extremely high-voltage discharge of electricity to the body.
- Lightning may cause injury by a direct strike (victim is directly struck), side flash (current flows over from another struck object), contact strike (a person touching a struck object), ground current (lightning passes through the ground and is transferred to a standing person), or step potential (ground strike passes up a person's leg and down through the other leg).

CLINICAL FEATURES

- Electricity causes damage by direct effects of current on cells and by thermal damage from the heat generated by the resistance of tissues.
- As current flows through the body, the greatest damage is sustained by the skin, nerves, blood vessels, and muscles. Skin injury does not correlate well with the underlying damage.
- The following conditions and injuries may be encountered: sudden death (ventricular fibrillation, asystole, respiratory arrest), dysrhythmias, myocardial damage, altered mental status, cerebral edema, central and peripheral neuropathy, thrombosis, disseminated intravascular coagulation, vessel rupture, pulmonary contusion, pneumothorax, tympanic membrane rupture, delayed labial artery rupture, corneal burns, cataracts, hepatic necrosis, pancreatic necrosis, hollow organ perforation, acute renal failure, myoglobinuria, lactic acidosis, hypokalemia, hypocalcemia, hyperglycemia, fractures, dislocations, compartment syndrome, skin burns, and fetal demise.[6–8]
- Myocardial infarction, pulmonary infarction, pneumonia, central and peripheral neuropathies, cognitive dysfunction, sleep disturbances, hearing loss, and cataracts may be delayed-presenting conditions of lightning injuries.
- Low-voltage AC tends to cause ventricular fibrillation, while high-voltage AC and DC currents result in asystole and respiratory arrest.

DIAGNOSIS AND DIFFERENTIAL

- Ruptured tympanic membranes or fernlike erythematous skin markings (Lichtenberg figures) should alert the physician to potential lightning injury.[9] A careful exam should assess neurologic status, otologic and ophthalmologic injuries, and blunt trauma.
- For patients who sustain a severe injury, diagnostic tests should include electrocardiogram (ECG), complete blood count, urinalysis, CK-MB level, electrolytes, blood urea nitrogen, creatinine, coagulation studies, liver panel, lipase, amylase, calcium, magnesium, and arterial blood gas analysis. Appropriate computed tomography (CT) scans and radiographs should be obtained for trauma patients.
- Common misdiagnoses include stroke, seizures, neurologic trauma, Stokes-Adams attacks, toxic ingestion, envenomation, myocardial infarction, dysrhythmia, and physical assault.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway, breathing, and circulation should be stabilized.
- Cervical spine immobilization should be instituted for any unwitnessed event or obtunded patient. Patients should be placed on 100% oxygen, cardiac monitor, pulse-oximetry, blood pressure monitoring, and have two large-bore intravenous (IV) lines established.
- Intravenous crystalloid fluid should be given with an initial bolus of 20 mL/kg.
- A Foley catheter should be placed, and a urine output of 1 mL/kg/h should be maintained.
- If evidence of rhabdomyolysis is present, urinary alkalization should be accomplished by giving 50

meq of sodium bicarbonate per liter of IV fluids. Blood pH should be maintained at 7.45, and the urine output at 2 mL/kg/h. Mannitol should be avoided in patients with thermal burns.
- Tetanus prophylaxis should be given.
- It is appropriate to consult a general surgeon if there is evidence of systemic or deep tissue injury.
- Patients with severe electrical injuries should be admitted to a regional burn or trauma center.
- Patients with brief low-intensity current exposure who are asymptomatic and have a normal ECG, urinalysis, and no evidence of significant burns or trauma may be discharged after 6 to 8 h of observation.[10]
- Admission criteria include high-voltage (>600 V) exposure, systemic injury, neurologic or vascular injury to an extremity or digit, deep burns, dysrhythmia or abnormal ECG, high-risk exposure, abuse or suicidal intent, associated injuries requiring admission, or the presence of comorbid diseases.

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125 RADIATION INJURIES

Keith L. Mausner

EPIDEMIOLOGY

- The United States Department of Energy Radiation Assistance Center/Training Site (REACTS) maintains a comprehensive record of radiation accidents worldwide. Since 1944, 403 radiation accidents have been recorded worldwide.
- The most commonly encountered radiation accident is one of high-dose local exposure, usually to the hands. Most of these accidents are related to inadvertent exposure to radiation from devices used to verify the integrity of metals and pipe welds.[1]

PATHOPHYSIOLOGY

- X-rays, gamma rays, neutrons, and alpha and beta particles are forms of ionizing radiation that damage tissue at the cellular level.
- Neutron radiation can also render matter radioactive; this would most likely be seen at nuclear power plants, weapons facilities, particle accelerators, or in a nuclear explosion.
- High-level ionizing radiation exposure may cause direct cell death; lower level exposure may interfere with cell division.
- Cells with high turnover rates, such as those of the hematopoietic, gastrointestinal, and reproductive systems, are more vulnerable than are slowly dividing cells of the central nervous system (CNS) and musculoskeletal system.
- The radian (rad) and gray (Gy) are units of absorbed radiation dose; they measure the amount of energy imparted by radiation as it passes through matter.
- The roentgen equivalent man (rem) equals the dose in rads multiplied by a factor that accounts for biological destructiveness; the sievert (Sv) has the same relation to the gray.

- A Geiger-Muller (GM) instrument detects gamma and x-rays and can also detect contamination with beta-particle-emitting matter; a special probe is required to detect alpha particles.
- Radiation dosage can be directly measured only if the victim was wearing a dosimeter at the time of exposure.

CLINICAL FEATURES

- Radiation exposure may involve either external or internal contamination, or external irradiation.
- External contamination can spread to the local environment, leading to internal contamination of the victim or others.
- Internal contamination occurs through inhalation, ingestion, or absorption through mucous membranes or abraded skin. An internally deposited radioisotope will irradiate local tissue until it decays to a stable isotope or is biologically eliminated.
- Beta radiation penetrates millimeters into tissue, and external contamination can inflict a dose to involved skin.
- Alpha radiation does not penetrate intact skin and is a significant hazard only when internalized.
- Iodine 131 is the predominant internal contaminant from nuclear accidents and weapons tests; exposure carries a significant risk of thyroid cancer or hypothyroidism.[2]
- In regard to external irradiation: (a) A quickly delivered dose causes more harm than a protracted exposure; (b) Time from exposure to symptom onset is inversely related to dose; (c) Acute radiation syndrome is most likely to be caused by whole-body gamma or x-ray irradiation; (d) In addition, doses of 8 to 9 Gy (800 to 900 rad) may cause pneumonitis, pulmonary fibrosis, and interstitial edema;[3] (e) Fetal injury generally occurs with exposures greater than 0.1 to 0.2 Gy (10 to 20 rad). However, fetal dosage above (500 millirad), especially between 8 to 15 weeks gestation, may increase the risk of CNS damage and growth defects.[4]
- Table 125-1 summarizes clinical syndromes associated with increasing doses.

DIAGNOSIS AND DIFFERENTIAL

- Nausea and vomiting are sensitive clinical indicators and are rarely seen with exposures under 1 Gy (100 rad).
- A lymphocyte count >1200 per milliliter indicates a good prognosis 24 h after exposure. A lymphocyte count <500 per milliliter predicts severe illness. If lymphocytes are depleted within 6 h, death is likely.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Hospitals should have an emergency department radiation accident protocol addressing prehospital care, notification of appropriate authorities, maintenance of appropriate supplies, and hazardous materials procedures, including isolation and decontamination.[5]
- Stable patients should be decontaminated prior to treatment. Critical patients should be triaged to an isolated area and resuscitated.
- The first priority remains addressing airway, breathing, circulation, and other potential life threats, including thermal burns and internal injuries.
- Table 125-2 summarizes common forms of contamination and their treatments.
- Treatment for acute radiation syndrome from whole-body irradiation is primarily supportive.

TABLE 125-1 Acute Radiation Syndrome

APPROXIMATE DOSE	ONSET OF PRODROME	DURATION OF LATENT PHASE	MANIFEST ILLNESS
>2 Gy (200 rad)	Within 2 days	1–3 weeks	Hematopoietic syndrome with pancytopenia, infection, and hemorrhage
>6 Gy (600 rad)	Within hours	<1 week	GI syndrome with dehydration, electrolyte abnormalities, GI bleeding, and fulminant enterocolitis
>30 Gy (3000 rad)	Within minutes	None	CV/CNS syndrome with refractory hypotension and circulatory collapse. Fatal within 24–72 h

ABBREVIATIONS: GI = gastrointestinal; CV = cardiovascular; CNS = central nervous system.

TABLE 125-2 Commonly Treated Forms of Internal Contamination

RADIONUCLIDE	PRIMARY ROUTE OF INTAKE	PRINCIPAL HAZARD	TREATMENT MECHANISM	AGENT	USUAL ADMINISTRATION*
I-131	Inhalation Ingestion Percutaneous absorption	Thyroid	Block thyroid uptake	KI	Oral: 390 mg a day for 7 to 14 days
Pu-239	Inhalation Ingestion Absorption through wounds	Bone Liver Lung	Chelation Increase excretion	DTPA	1 g/day for 5 days IV: 1 g in 250 mL NS or 5% dextrose in H_2O over 30 min Aerosol: 1 g in nebulizer; inhale over 15 to 20 min
H-3	Inhalation Ingestion Percutaneous absorption	Whole-body dose	Isotopic dilution Increase excretion	Water	Oral: 3–4 L a day for 2 weeks
Cs-137	Inhalation Ingestion	Whole-body dose	Mobilization Decrease GI uptake	Ferric Ferrocyanide (Prussian blue)	Oral: 1 g in 100–200 mL water tid for several days

ABBREVIATIONS: KI = potassium iodine; DTPA = diethylenetriamine; NS = normal saline; GI = gastrointestinal.
* Duration of therapy is based on dose estimations from radiochemical measurements of urine and fecal samples.

- Treatment of local radiation injury is supportive, with burn and surgical care as needed.

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126 POISONOUS PLANTS AND MUSHROOMS

Sandra L. Najarian

EPIDEMIOLOGY

- Mushrooms are one of the more common toxic exposures, with approximately 5 ingestions for every 100,000 people in 1996.[1]
- Amanita species is responsible for 95 percent of fatalities associated with mushrooms in the United States.
- Young children account for 70 to 80 percent of all plant-related exposures.

PATHOPHYSIOLOGY

- Various toxins found in plants and mushrooms produce effects ranging from mild gastrointestinal (GI) symptoms to organ failure and death.
- Mushrooms with psilocybin- and psilocin-containing toxins have neuroactive chemicals similar to lysergic acid diethylamide (LSD), causing hallucinogenic effects; they are often intentionally ingested for their mind-altering effects.
- Gyromitrin is a volatile, heat-labile toxin hydrolyzed in the stomach. It is converted to a free

radical in the liver, causing local hepatic necrosis and inhibiting the activity of the P450 system, glutathione, and other hepatic enzyme systems.[2]
- Amatoxins are absorbed in the intestines and enter the enterohepatic circulation; they bind to hepatocytes and inhibit formation of messenger RNA.[3]
- Ricin, a potent toxalbumin found in castor bean, produces severe cytotoxic effects in multiple organ systems.
- Cicutoxin, found in water hemlock, produces severe GI symptoms, followed by delirium, seizures, and death.
- Andromedotoxin, in *Rhododendron* species, produce a cholinergic syndrome.
- Amygdalin, found in the pits of peaches, apricots, pears, crab apples, and hydrangea, is metabolized to hydrocyanic acid and can lead to acute cyanide poisoning if ingested in sufficient quantities.
- Jimson weed contains atropine-like alkaloids that can cause an acute anticholinergic crisis.
- Urushiol, found in *Toxicodendron* species (poison ivy, oak, and sumac), produces a contact dermatitis in sensitized individuals.

CLINICAL FEATURES

- Dermatitis and GI complaints are the most common sequelae from plant-related exposures.
- Poisonous mushrooms can be divided into eight groups based on their clinical presentation and onset of symptoms (Table 126-1).
- The most commonly ingested toxic mushrooms are those that cause early-onset GI symptoms.
- Ingestion of *Gyromitra* and *Amanita* species of mushrooms produce delayed-onset of GI symptoms 6 to 48 h after ingestion. Manifestations of hepatic failure, intestinal necrosis, and renal failure develop 1 to 3 days after exposure.
- *Cortinarius* species of mushroom contain the nephrotoxic compounds, orellanine and orelline, which result in delayed-onset of GI symptoms 1 to 3 days after ingestion and delayed renal failure 3 to 20 days after ingestion.
- Consuming alcohol after ingestion of coprine-containing mushrooms will result in a disulfiram-like reaction. Facial flushing, nausea and vomiting, diaphoresis, palpitations, hypotension, and weakness can be observed 2 to 72 h after ingestion.

TABLE 126-1 Mushrooms: Symptoms, Toxicity, and Treatment

SYMPTOMS	MUSHROOMS	TOXICITY	TREATMENT
Gastrointestinal symptoms			
Onset <2 h	*Chlorophyllum molybdites* *Omphalotus illudens* *Cantharellus cibarius* *Amanita caesarea*	Nausea, vomiting, diarrhea (occasional bloody) Initial: nausea, vomiting, diarrhea	IV hydration Antiemetics IV hydration, glucose, monitor, AST, ALT, PT, PTT, bilirubin, BUN, creatinine
Onset 6–24 h	*Gyromitra esculenta*: fall season *Amanita phalloides, Amanita verna,* and *Amanita virosa*: spring season	Day 2: rise in AST, ALT Day 3: hepatic failure	For *Amanita:* activated charcoal Penicillin G 300,000–1,000,000 U/kg/day Silymarin 20–40 mg/kg/day Consider cimetidine 4–10 g/day Hyperbaric oxygen
Muscarinic (SLUDGE) syndrome Onset <30 min	*Inocybe* *Clitocybe*	Salivation, lacrimation, diarrhea, gastrointestinal distress, emesis	Supportive atropine 0.01 mg/kg repeated as needed for severe secretions
CNS excitement Onset <30 min	*Amanita muscaria* *Amanita pantherina*	Intoxication, dizziness, ataxia, visual disturbances, seizures, tachycardia, hypertension, warm dry skin, dry mouth, mydriasis (anticholinergic effects)	Supportive sedation with phenobarbital 30 mg IV or diazepam 2–5 mg IV as needed for adults
Hallucinations Onset <30 min	*Psilocybe* *Gymnopilus*	Visual hallucinations, ataxia	Supportive sedation with phenobarbital 0.5 mg/kg or, for adults, 30–60 mg IV, or diazepam 0.1 mg/kg or 5 mg IV for adults
Disulfiram 2–72 h after mushroom, and <30 min after alcohol	*Coprinus*	Headache, flushing, tachycardia, hyperventilation, shortness of breath, palpitations	Supportive IV hydration Propranolol for supraventricular tachycardia Norepinephrine for refractory hypotension

ABBREVIATIONS: ALT = alanine amino transferase; AST = aspartate amino transferase; BUN = blood urea nitrogen; CNS = central nervous system; PT = prothrombin time; PTT = partial thromboplastin time; SLUDGE syndrome = salivation, lacrimation, urination, defecation, gastrointestinal hypermotility, and emesis.

- Mushrooms with ibotenic acid and muscimol cause early-onset anticholinergic symptoms.
- *Inocybe* and *Clitocybe* species containing muscarine cause early-onset cholinergic and muscarinic effects, characterized by the SLUDGE syndrome (*S*alivation, *L*acrimation, *U*rination, *D*efecation, *G*astrointestinal, and *E*mesis).
- Consumption of psilocybin- and psilocin-containing mushrooms produce visual hallucinations and euphoria within 2 h of ingestion.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis of plant and mushroom poisoning is clinical, based on history of ingestion and onset of symptoms, and should be considered in patients at risk who present with gastroenteritis.
- If symptoms suggest cytotoxic mushroom poisoning, electrolytes, blood urea nitrogen, creatinine, liver enzymes, and coagulation studies should be obtained.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial treatment for plant-related and mushroom poisoning is supportive, with priority to airway management, ventilation, and fluid resuscitation.
- Activated charcoal should be administered to decontaminate the GI tract. Whole bowel irrigation is indicated for patients who may have ingested cytotoxic mushrooms and present within 24 h.
- High-dose penicillin therapy (0.3 to 1.0 million U/kg/day of penicillin G) is the most effective therapy for amatoxin poisoning; it blocks the uptake of amatoxin into the liver.[4] High-dose cimetadine (10 g/day) also has been found to be effective.[5] Hemodialysis and hemoperfusion, once thought to be standard of care, have limited use.
- Emergent liver transplant is indicated for patients with an aspartate aminotransferase level >2000 IU, grade 2 hepatic encephalopathy, and prothrombin time >50 s despite therapy.[6]
- High-dose pyridoxine (25 mg/kg) is recommended for patients presenting with neurologic symptoms associated with gyromitrin.
- Fluid and electrolyte replacement and hemodialysis are the mainstays of treatment for orellanine/orelline toxicity.
- Atropine should be administered to patients with severe muscarinic symptoms.
- Patients with potential amatoxin, gyromitrin, or orellanine/orelline poisoning, or those with refractory symptoms, require admission and monitoring for at least 48 h. All other patients who are asymptomatic after 4 to 6 h of treatment and observation can be discharged.

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Section 15
ENDOCRINE EMERGENCIES

127 DIABETIC EMERGENCIES

Michael P. Kefer

HYPOGLYCEMIA

EPIDEMIOLOGY

- Patients on insulin or oral hypoglycemic agents are especially at risk for hypoglycemia. Also at risk are those on beta blockers, barbiturates, or salicylates or patients with alcoholism, sepsis, adrenal insufficiency, or malnutrition.
- Newer oral hypoglycemic agents do not themselves cause hypoglycemia. These medications and their mechanism of action include the following:

  Metformin increases insulin effects and decreases glucose production. It rarely causes lactic acidosis as does its predecessor phenformin.

  Acarbose decreases the gastrointestinal absorption of carbohydrates.

  Troglitazone decreases insulin resistance and glucose production. Its use has been linked with hepatic failure.

PATHOPHYSIOLOGY

- Glucose is the main energy source of the brain. Severe hypoglycemia can cause brain damage and death.
- Blood glucose is dependent upon hormonal balance between insulin and the counterregulatory hormones epinephrine, glucagon, cortisol, and growth hormone. Excess insulin, either relative or absolute, will result in decreased glucose production and utilization.
- Glucose is supplied externally by food and internally by glycogenolysis and gluconeogenesis.

CLINICAL FEATURES

- Typical symptoms of hypoglycemia include sweating, shakiness, anxiety, nausea, dizziness, confusion, blurred vision, headache, and lethargy.
- Typical signs include diaphoresis, tachycardia, and almost any neurologic finding, from altered mental status or tremor to focal neurologic findings or seizure.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on a low blood glucose level in conjunction with the clinical features.
- The differential diagnosis is wide due to the nonspecific signs and symptoms manifested in patients with hypoglycemia. It can easily be misdiagnosed as a primary neurologic, psychiatric, or cardiovascular condition.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Glucose should be administered orally or intravenously as the condition warrants. Intravenous (IV) treatment begins with 1 amp of 50% dextrose. A continuous infusion of 5, 10, or 20% glucose solution to maintain a blood glucose level >100 mg/dL may be required.
- Hypoglycemia refractory to glucose administration may require hydrocortisone 100 mg IV or glucagon 1 mg IV.

- Glucagon 0.5 to 2.0 mg intramuscularly (IM) or subcutaneously may be required if IV access cannot be obtained.
- Diazoxide 300 mg IV may be required in sulfonylurea-induced hypoglycemia.
- Factors to be considered in determining disposition include the patient's response to treatment, etiology of hypoglycemia, existing comorbid conditions, and social situation.
- Most diabetics with insulin reactions respond rapidly to treatment. They can be discharged with instructions to continue oral intake of carbohydrates and to closely monitor their fingerstick glucose level. Those with prolonged or recurrent hypoglycemia, which can result from the sulfonylureas, require admission.

DIABETIC KETOACIDOSIS

EPIDEMIOLOGY

- Diabetic ketoacidosis (DKA) occurs predominantly in type 1 insulin-dependent diabetics but does occur in type 2 non-insulin-dependent diabetics.[1]
- New-onset diabetes mellitus presents as DKA in 25 percent of cases.
- Mortality occurs in 5 percent of the population, and it is higher in the elderly.[2,3]
- Diabetic ketoacidosis is precipitated by noncompliance with insulin therapy or any type of physiologic stress such as infection, stroke, myocardial infarction, trauma, or pregnancy.

PATHOPHYSIOLOGY

- Diabetic ketoacidosis results from a relative insulin deficiency and counterregulatory hormone excess resulting in cellular starvation.
- Insulin acts on the liver to promote glucose storage as glycogen, on adipose tissue to promote storage of triglycerides, and on skeletal muscle to promote protein synthesis. Although blood glucose levels are high, cells cannot use glucose as fuel in the absence of insulin.
- The counterregulatory hormones epinephrine, glucagon, cortisol, and growth hormone have the opposite effect of insulin. Glycogenolysis releases glucose stores. Proteolysis and lipolysis result in release of amino acids and glycerol, respectfully, for gluconeogenesis to synthesize more glucose.
- Free fatty acids are metabolized in the liver to the ketone bodies β-hydroxybutyrate, acetoacetate, and acetone. However, these also are unable to be used as fuel by cells in the absence of insulin.
- Hyperglycemia results in an osmotic diuresis with volume depletion and electrolyte loss.
- Ketonemia results in a high anion-gap metabolic acidosis with myocardial depression, vasodilation, and compensatory hyperpnea (Kussmaul's respiration).

CLINICAL FEATURES

- Clinical manifestations are directly related to the metabolic derangement caused by hyperglycemia and ketonemia.
- Typical symptoms include polyuria, polydipsia, dehydration, orthostasis, lethargy, weakness, nausea, vomiting, and abdominal pain. Acetone causes the characteristic fruity odor on the patient's breath.
- Typical signs include hypotension, tachycardia, Kussmaul's respiration, dry skin and mucous membranes, abdominal tenderness, and altered mental status.
- Blood glucose is elevated.
- Serum and urine ketones are elevated. β-hydroxybutyrate is the reduced form of acetoacetate. In DKA, reduction of acetoacetate to β-hydroxybutyrate is favored. As a result, in advanced cases, acetoacetate levels are low and β-hydroxybutyrate levels are high. If the nitroprusside test is used to detect serum or urine ketones, results may be falsely low or negative, since this test detects acetoacetate and not β-hydroxybutyrate.
- Sodium, chloride, calcium, phosphorus, and magnesium levels are low from osmotic diuresis.
- Pseudohyponatremia is common: for each 100 mg/dL increase in the glucose level, there is a 1.6 meq/L decrease in sodium.
- Serum potassium may be low (from osmotic diuresis and vomiting), normal, or high (from acidosis since acidosis drives potassium extracellularly). The patient who is acidotic with a normal or low potassium level has a marked depletion of total body potassium.
- Ketonemia results in an anion-gap metabolic acidosis $[Na^+ - (Cl^- + HCO_3^-) \geq 12 \pm 4$ meq/L].

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of DKA is based on the clinical presentation and a glucose level >250 mg/dL, bicarbonate level <20 meq/L (or pH < 7.3), and the presence of moderate ketonemia.[4]

TABLE 127-1 Differential Diagnosis of an Anion-Gap Metabolic Acidosis

Methanol
Uremia
Diabetic ketoacidosis
Paraldehyde
Iron, Isoniazid, Inhalants
Lactic acidosis
Ethanol, Ethylene glycol
Salicylates

- The differential diagnosis includes other causes of an anion-gap metabolic acidosis recalled by the acronym MUDPILES (see Table 127-1). Hypoglycemia and hyperosmolar hyperglycemic nonketotic syndrome also should be considered.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The goal of treatment is to correct hypovolemia, ketonemia, acidosis, and electrolyte abnormalities and treat the underlying cause.
- Resuscitation with isotonic fluid is the most important initial step to restore intravascular volume and tissue perfusion. Once intravascular volume is restored, hypotonic fluid is given to provide free water for intracellular volume replacement.
- Insulin is required to shut off ketosis and restore glucose utilization. Continuous IV infusion of insulin at 0.1 units/kg/h ensures a steady, reliable supply. A loading dose of 0.1 units/kg IV bolus is optional. If there is no response in the first hour, the insulin infusion rate is doubled. Hypoglycemia is corrected much more rapidly than ketoacidosis. Therefore, to prevent hypoglycemia, a glucose infusion should be initiated when the serum glucose approaches 250 mg/dL, and continued with the insulin drip until there is a resolution of the anion-gap.
- Basic laboratory investigation consists of determining serum glucose, electrolytes, blood urea nitrogen, creatinine, phosphorus, and magnesium levels, a complete blood cell count, urinalysis (and pregnancy test if indicated), electrocardiogram, and chest x-ray to evaluate the severity of DKA and to search for the underlying cause.
- Potassium replacement should begin as soon as urine output is adequate and the potassium level falls to <5.0 meq/L.
- Phosphorous replacement is recommended if the serum level is <1.0 mg/dL.
- Magnesium is replaced if serum levels are low.
- Bicarbonate replacement remains controversial and should not be routinely used.
- The serum glucose, anion-gap, potassium, and bicarbonate levels should be monitored hourly until recovery is well established.

HYPEROSMOLAR HYPERGLYCEMIC NONKETOTIC SYNDROME

EPIDEMIOLOGY

- The American Diabetes Association recommends this terminology for the condition previously referred to as *hyperglycemic nonketotic coma.*
- This condition is distinguished from DKA by the absence of ketonemia. Like DKA, it is a relatively common presentation of new-onset diabetes mellitus. Precipitating factors are the same as those for DKA (see earlier).

CLINICAL FEATURES

- Typically, the patient has a history of type 2 diabetes with preexisting heart or renal disease.
- Typical symptoms include weakness, altered mental status, polyuria, and polydipsia.
- Typical signs include dehydration, orthostasis, and altered mental status. Kussmaul's respiration and the presence of acetone on the breath are not present.
- Serum glucose is above 400 mg/dL and may be markedly elevated.
- Similar to DKA, sodium, potassium, chloride, calcium, phosphorus, and magnesium levels are low from osmotic diuresis. Pseudohyponatremia is common.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is based on the clinical and laboratory findings. It is differentiated from DKA by a greater degree of hyperglycemia, a lesser degree of acidosis, and the absence of ketones.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The goal of treatment is to correct hypovolemia and electrolyte abnormalities and treat the under-

lying cause. Basic laboratory investigation is the same as for DKA (see earlier) to evaluate the severity, search for the underlying cause, and to differentiate these two conditions from each other.
- Resuscitation with isotonic fluid is the most important initial step to restore intravascular volume and tissue perfusion. Once intravascular volume is restored, hypotonic fluid is given to provide free water for intracellular volume replacement.
- An insulin drip 0.1 units/kg/h can be initiated, but the patient may only require a bolus dose or two of insulin in conjunction with fluid therapy.
- Potassium, phosphorous, and magnesium levels are supplemented accordingly.
- The blood glucose and potassium levels should be monitored hourly until recovery is well established.[5–8]

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128 ALCOHOLIC KETOACIDOSIS

Michael P. Kefer

EPIDEMIOLOGY

- Alcoholic ketoacidosis (AKA) can occur in either first-time drinkers or chronic alcoholics.

PATHOPHYSIOLOGY

- Alcoholic ketoacidosis results from heavy ethanol intake, either acute or chronic, and minimal to no food intake. Glycogen stores become depleted, and insulin secretion is suppressed. To maintain a supply of glucose, the counterregulatory hormones glucagon, growth hormone, cortisol, and epinephrine are released. Fat and ethanol oxidation become the body's primary substrate for energy production resulting in the formation of the ketone bodies β-hydroxybutyrate, acetoacetate, and acetone. Acetone is rapidly excreted. Acetoacetate and β-hydroxybutyrate accumulate and result in a metabolic acidosis.
- β-hydroxybutyrate is the reduced form of acetoacetate. In AKA, the reduction of acetoacetate to β-hydroxybutyrate is favored. As a result, in advanced cases, acetoacetate levels are low and β-hydroxybutyrate levels are high. If the nitroprusside test is used to detect serum or urine ketones, results may be falsely low or negative, since this test only detects acetoacetate, and not β-hydroxybutyrate.

CLINICAL FEATURES

- Typical symptoms begin 2 to 3 days after the last ethanol intake and include nausea, vomiting, orthostasis, and abdominal pain.
- Typical signs include those of dehydration and nonspecific abdominal pain.
- The presentation of AKA may be confounded by other common conditions associated with ethanol use such as gastritis, hepatitis, pancreatitis, or ethanol withdrawal.

- Laboratory evaluation reveals an anion-gap metabolic acidosis [$Na^+ - (Cl^- + HCO_3^-) \geq 12 \pm 4$ meq/L], a low to mildly elevated blood glucose, and a low to absent blood ethanol. Serum and urine ketones are usually detected in significant amounts. However, as discussed earlier, if the redox state is such that most or all acetoacetate is reduced to β-hydroxybutyrate, the nitroprusside test used to detect the presence of ketones will be falsely low or negative.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is established in the patient with a recent history of ethanol consumption, decreased food intake, vomiting, abdominal pain, and laboratory findings of a high anion-gap metabolic acidosis, a low to mildly elevated glucose, and elevated serum or urine ketones.
- The differential diagnosis includes other causes of an anion-gap metabolic acidosis, recalled by the acronym MUDPILES (see ''Diabetic Emergencies,'' Table 127-1), as well as other causes of nausea, vomiting, and abdominal pain associated with ethanol use.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Intravascular volume should be restored with isotonic intravenous fluids.
- Glucose should be administered to stimulate insulin secretion and inhibit ketogenesis.
- Thiamine should be administered.
- Treatment should be continued until the acidosis is reversed and the patient can tolerate oral intake.[1–3]

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129 THYROID DISEASE EMERGENCIES

Stefanie R. Seaman

NORMAL THYROID STATE

- Thyroid hormone is released by thyroid-stimulating hormone (TSH) from the anterior pituitary. Regulation is by thyroid-releasing hormone (TRH) from the hypothalamus. Feedback occurs through the pituitary gland by thyroxine (T_4) and triiodothyronine (T_3) circulating levels in the blood.
- Thyroid hormone is reversibly bound to thyronine-binding globulin (TBG). Unbound hormone is biologically active, predominantly as T_4. T_3 is more active and deiodinated peripherally; it has a 1 day half-life versus 1 week for T_4.
- Thyroid hormone mediates cellular metabolism and protein synthesis.

HYPERTHYROIDISM

CLINICAL FEATURES

- Hyperthyroidism is uncommon under the age of 15, and it is 10 times more common in women.
- Graves' disease is the most common cause of hyperthyroidism followed by toxic multinodular and toxic nodular goiters. Graves' disease is common in the third and fourth decades of life. Clinical features include diffuse goiter, ophthalmopathy, and dermopathy.
- Causes of thyrotoxicosis are listed in Table 129-1.
- Symptoms of hyperthyroidism include heat intolerance, palpitations, weight loss, sweating, tremors, nervousness, weakness, and fatigue.
- Laboratory tests reveal an elevated free T_4 and low or undetectable TSH level. Occasionally, in Graves' disease, the T_4 may be normal and TSH decreased. T_3 levels should be checked to rule out T_3 toxicosis.

TABLE 129-1 Causes of Thyrotoxicosis

Primary hyperthyroidism
Graves' disease (toxic diffuse goiter)
Toxic multinodular goiter
Toxic nodular (adenoma) goiter
Iodine intake (jodbasedow disease)
Central hyperthyroidism
Pituitary adenoma
Thyroiditis
Subacute painful (de Quervain's disease)
Silent subacute
Postpartum
Radiation thyroiditis
Nonthyroidal disease
Ectopic thyroid tissue (struma ovarii)
Metastatic thyroid cancer
Drug induced
Lithium
Iodine (including radiographic contrast material)
Amiodarone
Excessive thyroid hormone ingestion (thyrotoxicosis facticia)

THYROID STORM

PATHOPHYSIOLOGY

- Thyroid storm is a life-threatening hypermetabolic state due to hyperthyroidism. It is most often seen in patients with unrecognized or poorly-treated Graves' disease. Mortality of thyroid storm is between 20 to 50 percent despite treatment.
- Precipitants of thyroid storm include infection, trauma, diabetic ketoacidosis, myocardial infarction, stroke, pulmonary embolism, surgery, withdrawal of thyroid medication, iodine administration, palpation of the thyroid gland, ingestion of thyroid hormone, and idiopathic (20 to 25 percent of cases).
- Generally, thyroid hormone levels do not differ between symptomatic, uncomplicated hyperthyroidism and thyroid storm.

CLINICAL FEATURES

- Signs and symptoms of thyroid storm include fever, tachycardia, diaphoresis, and emotional lability. There is central nervous system (CNS) disturbance in 90 percent of cases, which may manifest as confusion, delirium, seizures, and coma. Cardiovascular signs may include sinus tachycardia (out of proportion to fever), atrial fibrillation, and premature ventricular contractions. Gastrointestinal (GI) signs are diarrhea and hyperdefecation. Other signs include exophthalmos, increased pulse pressure, and palpable goiter.
- Apathetic thyrotoxicosis occurs in elderly patients. It is defined by a picture of lethargy, slowed mentation, apathetic facies, weight loss, proximal muscle weakness, atrial fibrillation, and, occasionally, congestive heart failure.

DIAGNOSIS AND DIFFERENTIAL

- Thyroid storm is a clinical diagnosis since no laboratory tests distinguish it from thyrotoxicosis. Diagnostic criteria include a temperature higher than 37.8°C (100.4°F); tachycardia out of proportion to fever; dysfunction of the CNS, cardiovascular or GI systems; and exaggerated peripheral manifestations of thyrotoxicosis. In this clinical setting, an elevated T_4 and a suppressed TSH confirms the diagnosis.
- The differential includes meningitis, sepsis, sympathomimetic ingestion, heat stroke, delirium tremens, malignant hyperthermia, malignant neuroleptic syndrome, hypothalamic stroke, pheochromocytoma, medication withdrawal, diabetic ketoacidosis, or hypoglycemia.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Oxygen, cardiac monitoring, and an intravenous (IV) line should be instituted.
- Fever should be treated with acetaminophen (aspirin can cause displacement of thyroid from TBG), cooling blankets, and ice packs. Dexamethasone 10 mg IV should be given for potential adrenal insufficiency that may occur with a hypermetabolic state.
- In order to produce blockade of peripheral hormone effects, propranolol 1 mg IV every 10 min to a total of 10 mg IV should be administered. Alternative therapy includes esmolol, guanethidine, or reserpine.
- The antithyroid drugs propylthiouracil (PTU) and methimazole act by blocking thyroid hormone synthesis. They must be given orally or by nasogastric tube. The initial loading dose of PTU is 600 to 1000 mg, followed by 200 to 250 mg every 4 h. Methimazole, 40 mg initially followed by 25 mg every 6 h, is an acceptable alternative.

- Propylthiouracil also decreases peripheral conversion of T_4 to T_3.
- Decreasing thyroid hormone release from stores is accomplished by administering iodine. Treatment should start 1 h after PTU administration. The dose is 5 drops of potassium iodide every 4 to 6 h or sodium iodide 0.5 to 1 g every 12 h by IV infusion. Iodine contrast material and Lugol's solution 8 to 10 drops PO every 6 h may be used as well. Lithium can be used in patients with a history of iodine allergy.
- Precipitating causes of thyroid storm should be identified and treated.
- In cases where clinical deterioration occurs despite appropriate therapy, direct removal by exchange transfusion, plasma transfusion, plasmapheresis, and charcoal plasma perfusion may prove successful.
- Patients with thyroid storm should be admitted into an intensive care unit setting.

HYPOTHYROIDISM

PATHOPHYSIOLOGY

- Hypothyroidism occurs with insufficient hormone production. Hypothyroidism occurs more frequently in women than in men.
- The most common etiologies are primary thyroid failure due to autoimmune disease (Hashimoto's thyroiditis); idiopathic, postablative surgery; or iodine deficiency. Postpartum thyroiditis occurs within 3 to 6 months postpartum in 2 to 16 percent of women.
- Secondary thyroiditis is due to pituitary tumors, infiltrative disease, or hemorrhage. Tertiary thyroiditis is due to hypothalamic disease.
- Medications that cause hypothyroidism include amiodarone (due to release of iodine during metabolism of the drug) and lithium (mimics iodine and inhibits thyroid hormone release).

CLINICAL FEATURES

- Symptoms of hypothyroidism include fatigue, weight gain, cold intolerance, depression, menstrual irregularities, constipation, joint pain, and muscle cramps.
- Signs of hypothyroidism include hoarseness; hypothermia; periorbital puffiness; delayed relaxation of ankle jerks; loss of outer one-third of eyebrow; cool, rough, dry skin; nonpitting edema; bradycardia; infertility; and peripheral neuropathy.

MYXEDEMA COMA

- Myxedema coma is a rare life-threatening expression of severe hypothyroidism. It is most common in the geriatric population.

CLINICAL FEATURES

- The clinical features of myxedema coma consist of all of the features of hypothyroidism plus hypothermia (in 80 percent of cases), altered mental status (coma is rare), delusions, psychosis (myxedema madness), hyponatremia, hypotension, bradycardia, and paralytic ileus or megacolon.
- Respiratory failure with hypoventilation, hypercapnea, and hypoxia is common.
- Cardiac findings include bradycardia, enlarged heart, and a low-voltage electrocardiogram.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of myxedema coma must be suspected based upon the clinical presentation and characteristic laboratory abnormalities.
- Laboratory tests may reveal low free T_4 and elevated TSH.
- Differential diagnosis includes coma secondary to respiratory failure, hyponatremia, hypothermia, congestive heart failure, stroke, or drug overdose.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway support and supplemental oxygen should be provided. All patients require dextrose-containing IV fluids, cardiac monitoring, Foley catheter, and nasogastric tube.
- Hypothermia and hyponatremia should treated in the standard fashion.
- Hydrocortisone 100 mg IV every 8 h should be administered.
- Thyroid hormone is the most specific and critical therapy for myxedema coma. Levothyroxine 300 to 500 μg IV should be administered by slow infusion, followed by 50 to 100 μg IV daily. Alternatively, L-triiodothyronine 25 μg IV or PO should be administered every 8 h.
- Patients with myxedema coma should be admitted to a monitored bed.

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130 ADRENAL INSUFFICIENCY AND ADRENAL CRISIS

Michael P. Kefer

PATHOPHYSIOLOGY

- Adrenal insufficiency results when the physiologic demand for glucocorticoids and mineralocorticoids exceeds the supply from the adrenal cortex.
- The hypothalamus secretes cortisol-releasing factor (CRF), which stimulates the pituitary gland to secrete adrenocorticotropic hormone (ACTH), which stimulates the adrenal cortex to secrete cortisol. Cortisol has negative feedback on the pituitary gland to inhibit ACTH and melanocyte-stimulating hormone (MSH) secretion.
- Cortisol is the major glucocorticoid. It has a major role in maintaining blood glucose levels by decreasing glucose uptake and stimulating proteolysis and lipolysis for gluconeogenesis. Cortisol is necessary for the proper function of catecholamines on cardiac muscle and arterioles. Cortisol also controls body water balance.
- Aldosterone is the major mineralocorticoid. The renin-angiotensin system and serum potassium regulate its secretion; ACTH has a minor effect.
- Adrenal insufficiency is described as primary, secondary, or tertiary based on whether it occurs at the level of the adrenal gland, pituitary, or hypothalamus, respectively.
- The most common cause of adrenal insufficiency is adrenal suppression from prolonged steroid use with subsequent steroid withdrawal. Other causes include autoimmune disorders, metastatic cancer, AIDS, sarcoidosis, and bilateral adrenal hemorrhage associated with meningococcemia (Waterhouse-Friderichsen syndrome) or heparin therapy.

CLINICAL FEATURES

- Primary adrenal insufficiency results from inadequate secretion of cortisol and aldosterone, and manifests as weakness, orthostasis, hypotension, anorexia, nausea, vomiting, and abdominal pain. Hyperpigmentation of both exposed and nonexposed skin and mucous membranes occurs as a result of uninhibited release of MSH in conjunction with ACTH. Typical laboratory abnormalities include hyponatremia, hyperkalemia, hypoglycemia, and prerenal azotemia.
- Secondary and tertiary adrenal insufficiency result from inadequate secretion of cortisol. Hypoglycemia is a prominent feature. Aldosterone secretion is not significantly affected because of regulation through the renin-angiotensin system. Therefore, the hyperpigmentation, hyponatremia, hyperkalemia, and volume depletion of primary adrenal insufficiency is not seen.
- Adrenal crisis is the extreme presentation of adrenal insufficiency, with shock and altered mental status as additional presenting features.

DIAGNOSIS AND DIFFERENTIAL

- Presentation with the clinical features described suggest the diagnosis. It may be confirmed in the emergency department by performing a screening cosyntropin (synthetic ACTH) stimulation test. An initial cortisol level is drawn followed by administration of cosyntropin 0.25 mg intramuscularly or intravenously (IV). After 30 to 60 min, a repeat cortisol level should be double the initial.

(Note there would be no response in primary adrenal insufficiency. The initial and repeat cortisol levels would be low. In secondary or tertiary adrenal insufficiency, the adrenal gland can still respond to ACTH so the initial level would be low with the repeat level elevated.)

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Outpatient treatment of primary adrenal insufficiency consists of prednisone for glucocorticoid replacement, fludrocortisone for mineralocorticoid replacement, and, in women, fluoxymesterone for estrogen replacement.
- Treatment of secondary and tertiary adrenal insufficiency is similar to primary adrenal insufficiency but does not require mineralocorticoid replacement, as aldosterone levels are not significantly affected with an intact renin-angiotensin system.
- Treatment of adrenal crisis consists of the following:
  1. Fluid resuscitation, with D5 normal saline initially.
  2. Hydrocortisone 100 mg IV bolus. (Dexamethasone 4 mg IV should be substituted if a cosyntropin stimulation test is performed so as not to give a false-positive test.)
  3. Additional hydrocortisone or vasopressors may be necessary in refractory cases.[1,2]

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Section 16

HEMATOLOGIC AND ONCOLOGIC EMERGENCIES

131 EVALUATION OF ANEMIA AND THE BLEEDING PATIENT

Sandra L. Najarian

PATHOPHYSIOLOGY

- Anemia is due to loss of red blood cells (RBCs) by hemorrhage, increased destruction of RBCs, or impaired production of RBCs (Table 131-1).
- Bleeding disorders from congenital or acquired abnormalities in the hemostatic system can result in excessive hemorrhage, excessive clot formation, or both.

CLINICAL FEATURES

- The severity of the anemia depends on the rate of development of anemia, extent of the anemia, and the ability of the cardiovascular system to compensate for the decreased oxygen-carrying capacity.
- Common symptoms of anemia include palpitations, dyspnea, dizziness, exertional intolerance, tinnitus, and feelings of postural faintness.
- Commons signs include pale conjunctiva, skin, and nail beds; tachycardia; hyperdynamic precordium; systolic murmurs; tachypnea at rest; and hypotension.
- Risk factors for underlying bleeding disorders include a family history of bleeding disorder, history of liver disease, and use of aspirin, nonsteroidal anti-inflammatory drugs, ethanol, warfarin, or certain antibiotics.
- Signs of platelet disorders include mucocutaneous bleeding (including petechiae, ecchymoses, purpura, and epistaxis), gastrointestinal or genitourinary bleeding, or heavy menstrual bleeding.
- Signs of coagulation factor deficiencies include delayed bleeding, hemarthrosis, or bleeding into potential spaces (e.g., retroperitoneum).

DIAGNOSIS AND DIFFERENTIAL

- Decreased RBC count, hemoglobin, and hematocrit are diagnostic of anemia. Hemoccult examination, complete blood cell count, reticulocyte count, review of RBC indices, and examination of peripheral blood smear are necessary for the initial evaluation of the patient with anemia (Table 131-2).
- Complete blood cell count, platelet count, prothrombin time, and partial thromboplastin time are necessary for the initial evaluation of the patient with a suspected bleeding disorder (Table 131-3).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent priorities remain airway, breathing, and circulation. Hemorrhage should be controlled with direct pressure. The treatment of anemia includes initial stabilization and investigation of the etiology of the anemia.
- Type- and cross-matched blood should be ordered if blood transfusion is anticipated. Packed RBCs should be transfused in symptomatic patients and those who are hemodynamically unstable.
- Indications for admission include patients with anemia and ongoing blood loss or evidence of tissue hypoxia and hemodynamic instability. He-

TABLE 131-1 Pathophysiologic Classification of Anemia

I. Loss of RBCs by hemorrhage
- As a result of acute or chronic blood loss.
- In the setting of acute blood loss, the bone marrow has not had sufficient time to increase erythropoiesis to replace the lost RBCs.
- In chronic blood loss, erythropoiesis may not be adequate to replace the lost RBCs.

II. Increased destruction of RBCs—hemolytic anemias
- Hereditary hemolytic anemias
- Acquired hemolytic anemias

III. Impaired production of RBCs
A. Hypochromic anemias
- The RBCs have a decreased amount of hemoglobin in each cell (hypochromic), and the cells typically are small (microcytic).
- Results from impaired hemoglobin synthesis.
- Examples are iron deficiency, anemia of chronic disease, thalassemias, sideroblastic anemias.

B. Aplastic/myelodysplastic anemias
- The RBCs are of normal size (normochromic) or large (macrocytic).
- Results from marrow stem cell failure.
- Caused by chemicals (including ethanol), radiation, infections (including HIV, human parvovirus B_{19}), chronic renal failure, marrow infiltration, myelodysplastic syndromes, idiopathic.

C. Megaloblastic anemias
- The RBCs are large (macrocytic).
- Results from impaired DNA synthesis.
- Caused by deficiency of vitamin B_{12} or folate, drugs (chemotherapeutics, HIV drugs)

ABBREVIATIONS: RBC = red blood cells; HIV = human immunodeficiency virus; DNA = deoxyribonucleic acid.

matology consultation is warranted in patients with suspected bleeding disorders and anemia of unclear etiology.

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TABLE 131-2 Initial Laboratory Evaluation of Anemia

TEST	INTERPRETATION	NORMAL VALUE	CLINICAL CORRELATION
RBC indices:			
MCV	Reflects average RBC size	80–95 fL	*Decreased MCV* (microcytosis)—chronic iron deficiency, thalassemia, anemia of chronic disease *Increased MCV* (macrocytosis)—decreased level of vitamin B_{12} or folate, chronic ethanol ingestion, chronic liver disease, reticulocytosis, phenytoin, HIV drugs
MCH	Reflects weight of hemoglobin in average RBC	28–32 pg	The MCH and MCHC do not provide much additional information for the classification of anemia.
MCHC	Reflects concentration of hemoglobin in average RBC	32–36%	
Reticulocyte count	These RBCs of intermediate maturity are an index of the production of mature RBCs by the bone marrow, reported as a percent of total RBCs	0.5–1.5%	*Decreased reticulocyte count* reflects impaired RBC production; seen with low levels of iron, vitamin B_{12}, folate, bone marrow failure *Elevated reticulocyte count* reflects accelerated erythropoiesis, the normal marrow response to anemia; seen with blood loss and hemolytic anemias
Peripheral blood smear	Used for the evaluation of: 1. Overall size of the RBCs; example: normocytic, microcytic, macrocytic 2. Amount of hemoglobin in the RBCs; example, hypochromic 3. Look for abnormal shapes such as sickled cells or schistocytes (evidence of hemolysis) 4. Examination of white blood cells and platelets		

ABBREVIATIONS: RBC = red blood cells; MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration.

TABLE 131-3 Tests of Hemostasis

SCREENING TESTS	NORMAL VALUE	MEASURES	CLINICAL CORRELATIONS
		PRIMARY HEMOSTASIS	
Platelet count	150,000–300,000/μL	Number of platelets per μL	Decresed platelet count (thrombocytopenia) Bleeding usually not a problem until platelet count <50,000/μL; high risk of spontaneous bleeding including CNS with count <10,000/μL. Causes Decreased production—viral infections (measles); marrow infiltration; drugs (thiazides, ethanol, estrogens, interferon-α) Increased destruction—viral infections (mumps, varicella, EBV, HIV); ITP, TTP, DIC, HUS; drugs (heparin, protamine) Splenic sequestration (hypersplenism, hypothermia) Loss of platelets (hemorrhage, hemodialysis, extracorporeal circulation) Pseudothrombocytopenia—platelets are clumped but not truly decreased in number; examine blood smear to recognize this Elevated platelet count (thrombocytosis)—commonly reactive to inflammation or malignancy, or in polycythemia vera; can be associated with hemorrhage or thrombosis
Bleeding time (BT)	2.5–10 min (template BT)	Interaction between platelets and the subendothelium	Prolonged BT caused by: Thrombocytopenia (platelet count <50,000/μL) Abnormal platelet function (vWD, ASA, NSAIDs, uremia, liver disease) Collagen abnormalities (congenital abnormality or prolonged use of steroids)
		SECONDARY HEMOSTASIS	
Prothrombin time (PT)	10–12 s, but laboratory variation	Extrinsic system and common pathway—factors VII, X, V, prothrombin, and fibrinogen	*Prolonged PT* most commonly caused by: Use of warfarin (inhibits vitamin K–dependent factors II, VII, IX, and X) Liver disease with decreased factor synthesis Antibiotics, some cephalosporins, (moxalactam, cefamandole, cefotaxime, cefoperazone) that inhibit vitamin K–dependent factors
Activated partial thromboplastin time (aPTT)	Depends on type of thromboplastin used; ''activated'' with Kaolin	Intrinsic system and common pathway including factors XII, XI, IX, VIII, X, V, prothrombin, and fibrinogen	*Prolongation of aPTT* most commonly caused by: Heparin therapy Factor deficiencies; factor levels have to be <30% of normal to cause prolongation **Note:** High doses of heparin or warfarin can cause prolongation of both the PT and aPTT due to their activity in the common pathway.
Thrombin clotting time (TCT)	10–12 s	Conversion of fibrinogen to fibrin monomer	*Prolonged TCT* caused by: Low fibrinogen level (DIC) Abnormal fibrinogen molecule (liver disease) Presence of heparin, FDPs, or a paraprotein (multiple myeloma); these interfere with the conversion Very high fibrinogen level (acute phase reactant)
''Mixes''	Variable	Performed when one or more of the above screening tests is prolonged; the patient's plasma (''abnormal'') is mixed with ''normal'' plasma and the screening test is repeated	*If the ''mix'' corrects* the screening test, one or more factor deficiencies are present. *If the ''mix'' does not correct the screening test,* an inhibitor is present.
		OTHER HEMOSTATIC TESTS	
Fibrin degradation products and D-dimer (evaluate fibrinolysis)	Variable	*FDPs* measure breakdown products from fibrinogen and fibrin monomer *D-Dimer* measures breakdown products of cross-linked fibrin	Levels of these are elevated in DIC, thrombosis, pulmonary embolus, liver disease.
Factor level assays	60–130% (0.60–1.30 units/mL)	Measures the percent activity of a specified factor compared to normal	Used to identify specific factor deficiencies and in therapeutic management of patients with deficiencies
Inhibitor screens	Variable	Verifies the presence or absence of antibodies directed against one or more of the coagulation factors	*Specific inhibitors*—directed against one coagulation factor, most commonly against factor VIII; can be in patients with congenital or acquired deficiency. *Nonspecific inhibitors*—directed against more than one of the coagulation factors; example is lupus-type anticoagulant

ABBREVIATIONS: ASA = aspirin; CNS = central nervous system; DIC = disseminated intravascular coagulation; EBV = Epstein-Barr virus; FDPs = fibrin degradation products; HIV = human immunodeficiency virus; HUS = hemolytic uremic syndrome; ITP = idiopathic thrombocytopenic purpura; NSAIDs = nonsteroidal anti-inflammatory drugs; TTP = thrombotic thrombocytopenic purpura; vWD = von Willebrand disease.

132 ACQUIRED BLEEDING DISORDERS

Kathleen F. Stevison

Bleeding disorders may be the result of platelet abnormalities, exogenous anticoagulants, coagulation factor deficiencies, drugs, or systemic illnesses.

BLEEDING DUE TO PLATELET ABNORMALITIES

- Acquired platelet abnormalities include both qualitative and quantitative platelet defects. Quantitative platelet disorders begin after levels drop below 50,000/μL and can be caused by decreased platelet production, increased platelet destruction, increased platelet loss, and splenic sequestration.
- Causes of decreased platelet production include marrow infiltration, aplastic anemia, drugs, and viral infections.
- Causes of increased platelet destruction include idiopathic thrombocytopenic purpura (ITP), thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome (HUS), and disseminated intravascular coagulation (DIC).
- Causes of increased platelet loss include hemorrhage and hemodialysis.
- Qualitative disorders result in excessive bleeding regardless of the number of available platelets; common causes include liver disease, drugs, antiplatelet antibodies, DIC, and uremia.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Platelet transfusion is warranted in all patients with a platelet count $<$10,000/μL, regardless of etiology.
- Patients with serious bleeding and platelet counts below 50,000/μL should receive transfusions. However, hematologic consultation should be obtained, as some conditions, such as DIC and TTP, may actually be worsened by platelet transfusion.

BLEEDING DUE TO WARFARIN USE OR VITAMIN K DEFICIENCY

- Vitamin K is a necessary coefficient in the production of factors II, VII, IX, and X as well as proteins C and S.
- Warfarin antagonizes vitamin K, resulting in therapeutic anticoagulation.
- Patients with liver disease, those with vitamin K deficiency due to poor nutrition or malabsorption, and patients taking warfarin are at increased risk of bleeding.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment depends on the seriousness of the bleeding. A prolonged prothrombin time (PT) with no active bleeding may require only observation and discontinuation of warfarin.
- Fresh-frozen plasma (FFP) rapidly replenishes coagulation factors and should be used to treat serious bleeding.
- Vitamin K (10 mg SQ or IM) may also be used to treat active bleeding, although it takes approximately 24 h to take effect and prevents anticoagulation with warfarin for about 2 weeks.

BLEEDING DUE TO HEPARIN USE OR THROMBOLYTIC THERAPY

- Bleeding is the most common side effect of the use of heparin or thrombolytic therapy. Major bleeding complications [defined as those requiring transfusion of packed red blood cells (PRBCs)] occur in approximately 1 to 2 percent of patients receiving heparin or thrombolytic agents.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Therapy should be discontinued immediately once significant bleeding is detected.
- Protamine, 1 mg IV for every 100 U of heparin administered in the previous 4 h, will reverse heparinization. Protamine is also effective against the low-molecular-weight heparin agents.
- Massive bleeding with thrombolytic agents requires cryoprecipitate (10 U IV), followed by FFP (2 U IV) if bleeding persists. Further treatment may include platelet transfusions and aminocaproic acid infusion for reversal. Hematology consultation is recommended.

BLEEDING IN LIVER DISEASE

- Multiple factors place these patients at risk for bleeding disorders; these include decreased coagu-

lation factor production, vitamin K deficiencies, thrombocytopenia (due to splenic sequestration after portal hypertension results in hypersplenism), and increased fibrinolysis (from a chronic, low-grade DIC state).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- All bleeding patients with liver disease should receive vitamin K (10 mg SQ or IM).
- Severe bleeding warrants FFP transfusion for replenishing coagulation factors and platelet transfusions for significant thrombocytopenia.
- Desmopressin (DDAVP, 0.3 μg/kg SQ or IV) may effectively lower bleeding times in some patients.

BLEEDING IN RENAL DISEASE

- The bleeding tendency exhibited by patients with renal disease is related to the degree and duration of uremia. Uremic degradation products, chronic anemia, platelet dysfunction, deficiency of coagulation factors, and thrombocytopenia all contribute to the bleeding disorder.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- PRBCs should be transfused to maintain a hematocrit between 26 and 30, which optimizes platelet function.
- Hemodialysis will transiently improve platelet function (1 to 2 days).
- DDAVP 0.3 μg/kg SQ or IV shortens bleeding time in the majority of patients.
- Conjugated estrogens, by an unknown mechanism, improve bleeding times in most patients.
- Platelet and cryoprecipitate transfusions are reserved for life-threatening bleeding only.

BLEEDING IN DISSEMINATED INTRAVASCULAR COAGULATION

- DIC results from the activation of both the coagulation and fibrinolytic systems. The most common trigger of DIC is the liberation of tissue factor from the extravascular space.
- The most common causes of DIC are in the clinical settings of infection, carcinoma, acute leukemia, trauma, shock, liver disease, pregnancy, vascular disease, envenomation, adult respiratory distress syndrome, and transfusion reactions.

CLINICAL FEATURES

- DIC results in both bleeding and thrombotic complications, although one usually predominates in an individual patient.
- Bleeding occurs in up to 75 percent of patients and typically affects the skin and mucous membranes. The skin may show signs of petechiae or ecchymoses. Bleeding from several sites, including venipuncture sites and surgical wounds, is common. GI, urinary tract, and CNS bleeding also may occur.
- Other patients show primarily thrombotic symptoms. Depending on the site of the thrombosis, the patient may exhibit focal ischemia of the extremities, mental status changes, oliguria, or symptoms of adult respiratory distress syndrome.
- Purpura fulminans develops when there is widespread thrombosis resulting in gangrene of the extremities and hemorrhagic infarction of the skin.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis depends on the clinical setting and characteristic laboratory abnormalities (see Table 132-1).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Hemodynamic stabilization should be provided through IV fluids or transfusion of PRBCs.
- The underlying illness should be treated.
- If bleeding predominates and the PT is elevated more than 2 s, replacement of coagulation factors is indicated. FFP is infused 2 U at a time. Cryoprecipitate is used to replace fibrinogen; it is typically infused 10 bags at a time. If the platelet count is less than 50,000/μL with active bleeding or less than 20,000/μL without bleeding, platelet transfusion should be initiated. All patients with bleeding due to DIC should also receive vitamin K (10 mg SQ or IM) and folate (1 mg IV).
- If thrombosis predominates, heparin therapy should be considered, although this is controversial. Heparin is most likely to provide benefit if the underlying medical condition is carcinoma, acute

TABLE 132-1 Laboratory Abnormalities Characteristic of Disseminated Intravascular Coagulation

STUDIES	RESULT
MOST USEFUL	
Prothrombin time	Prolonged
Platelet count[a]	Usually low
Fibrinogen level[b]	Low
HELPFUL	
Activated partial thromboplastin time	Usually prolonged
Thrombin clot time[c]	Prolonged
Fragmented red blood cells[d]	Should be present
FDPs and D-dimers[e]	Elevated
Specific factor analysis[f]	
Factor II	Low
Factor V	Low
Factor VII[g]	Low
Factor VIII[h]	Low, normal, high
Factor IX	Low (decreases later than other factors)
Factor X	Low

ABBREVIATION: FDP = fibrin degradation products.
[a] Platelet count usually low, most important that it is falling if it started at an elevated level.
[b] Fibrinogen level correlates best with bleeding complications: it is an acute phase reactant so it may actually start out at an elevated level: fibrinogen level <100 mg/dL correlates with severe DIC.
[c] Not a sensitive test, prolonged by many abnormalities.
[d] Fragmented red blood cells and schistocytes are not specific for DIC.
[e] Levels may be chronically elevated in patients with liver or renal disease.
[f] The factors in the extrinsic pathway are most affected (VII, X, V, and II).
[g] Factor VII is usually low early because it has the shortest half-life.
[h] Factor VIII is an acute phase reactant so its level may be normal, low, or elevated in DIC.

promyelocytic leukemia, or retained uterine products or if the patient exhibits signs of purpura fulminans.

BLEEDING DUE TO CIRCULATING ANTICOAGULANTS

PATHOPHYSIOLOGY

- Circulating anticoagulants are antibodies directed against one or more of the coagulation factors. The two most common circulating anticoagulants are factor VIII inhibitor (a specific inhibitor directed only against factor VIII) and lupus anticoagulant (a nonspecific inhibitor directed against several of the coagulation factors).

CLINICAL FEATURES

- Patients with factor VIII inhibitor present with massive spontaneous bruises, ecchymoses, and hematomas.
- Patients with the lupus anticoagulant may present with thromboses or recurrent fetal loss. Bleeding abnormalities are rare in patients with lupus anticoagulant.

DIAGNOSIS AND DIFFERENTIAL

- Laboratory studies in patients with factor VIII inhibitor reveal a normal PT, normal thrombin clot time (TCT), and a greatly prolonged aPTT that does not correct with ''mixing.'' A factor VIII–specific assay will show that the factor VIII activity is very low.
- Patients with lupus anticoagulant will have a normal or slightly prolonged PT, a normal TCT, and a moderately prolonged aPTT that also does not correct with mixing. Factor-specific assays will show a decrease in all factor levels.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with factor VIII inhibitor and active bleeding should be managed in conjunction with a hematologist. Treatment options include high doses of factor VIII concentrate, prothrombin complex concentrates, and recombinant factor VIIa.
- Patients with lupus anticoagulant and thrombosis should be treated with long-term anticoagulation with either warfarin (venous thrombosis) or aspirin (arterial thrombosis).

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133 HEMOPHILIAS AND VON WILLEBRAND'S DISEASE

John Sverha

HEMOPHILIAS

EPIDEMIOLOGY

- The most common hemophilias are due to the deficiency of either factor VIII (hemophilia A) or factor IX (hemophilia B).
- Patients with hemophilia B account for 85 percent of all hemophiliacs.
- Both hemophilia A and B are X-linked recessive disorders.
- The clinical classification of patients with hemophilia depends on the severity of their factor deficiency. Patients are classified as having mild disease if they have between 6 and 60 percent of normal factor VIII or IX activity, moderate disease if they have between 1 and 5 percent of normal factor activity, and severe disease if there is less than 1 percent of factor activity.

PATHOPHYSIOLOGY

- Deficiency of either factor VIII or factor IX results in diminished efficacy of the intrinsic coagulation pathway.

CLINICAL FEATURES

- Bleeding in hemophiliac patients is characterized by deep hematomas or hemarthroses that occur spontaneously or with minimal trauma. Bleeding may occur hours after the initial trauma.
- Central nervous system (CNS) bleeding may cause a headache or focal neurologic symptoms. Intracranial bleeding is a major cause of death in hemophiliacs.
- Spontaneous or traumatic bleeding into the neck or retroperitoneum can be life threatening.
- Compartment syndrome can occur with bleeding into fascial compartments of the extremities.

DIAGNOSIS AND DIFFERENTIAL

- Patients with hemophilia A or B typically have a prolonged partial thromboplastic time (PTT) if their factor activity level is less than 30 percent. Their prothrombin time (PT) and bleeding times are typically normal.
- The only way to distinguish hemophilia A and B is by specific factor activity assays for factors VIII and IX.
- About 10 percent of patients with hemophilia will develop an inhibitor, which is an antibody against the missing factor. The presence of an inhibitor is diagnosed when plasma from a patient with hemophilia is mixed 50–50 with plasma from a normal control and this mixture continues to show a prolonged PTT. The quantity of inhibitor present is measured by the Bethesda inhibitor assay (BIA) and is reported in BIA units.

EMERGENCY CARE DEPARTMENT AND DISPOSITION

- The management of bleeding in patients with hemophilia depends on (a) the type and severity of the hemophilia, (b) the presence or absence of inhibitor, and (c) the site and severity of the bleeding.
- Patients with symptoms of bleeding in the neck, retroperitoneum, or CNS should have immediate factor replacement followed by diagnostic testing.
- A 1-U/kg dose of factor VIII raises the factor VIII activity level by 2 percent. A 1-U/kg dose of factor IX raises the factor IX activity level by 1 percent.
- Life-threatening bleeding in patients with hemophilia A or hemophilia B requires an initial dose of 50 U/kg of either factor VIII or factor IX concentrate.
- Patients with inhibitor may require multiple factor infusions and possibly infusion of activated prothrombin complex concentrate.
- Desmopressin (DDAVP) can be used to raise factor VIII levels in patients with mild to moderate hemophilia A and no inhibitor.
- In circumstances of life-threatening bleeding where factor concentrate is not available or the type of hemophilia is unknown, fresh-frozen plasma should be administered. Each milliliter of

fresh-frozen plasma contains approximately 1 U of factor VIII and factor IX. It may be difficult to achieve desired factor activity levels due to volume constraints.
- Cryoprecipitate also may be used when factor concentrates are not available. However, cryoprecipitate contains factor VIII but not factor IX. It has the advantage of providing a more concentrated solution of factor VIII. Each milliliter of cryoprecipitate provides approximately 6 units of factor VIII.

VON WILLEBRAND'S DISEASE

EPIDEMIOLOGY

- Von Willebrand's disease (vWD) is the most common inherited bleeding disorder.
- One in 100 persons inherits a gene defective for von Willebrand's factor (vWF), but only 1 in 10,000 persons manifest a clinically significant bleeding disorder due to this abnormality.
- Von Willebrand's disease is usually inherited in an autosomal dominant pattern.

PATHOPHYSIOLOGY

- The normal function of vWF is to allow platelets to adhere to damaged endothelium and to carry factor VIII in the plasma.
- There are three major subtypes of vWD, and they vary tremendously in their clinical severity. Eighty percent of persons with vWD have type I disease that is mild. Less than 10 percent of persons with vWD have type III disease that results in severe bleeding similar to hemophilia.

CLINICAL FEATURES

- Bleeding in type I vWD tends to be mild and typically involves epistaxis, easy bruising, menorrhagia, bleeding after dental procedures, and gastrointestinal bleeding.
- Bleeding in type III vWD is severe and often involves development of spontaneous hemarthroses and hematomas similar to hemophilia.

DIAGNOSIS AND DIFFERENTIAL

- Patients with vWD typically have a normal PT, normal PTT, a prolonged bleeding time, low or normal factor VIII level, low or normal vWF antigen level, and low vWF activity level. Patients with severe vWD may have an elevated PTT.

EMERGENCY CARE DEPARTMENT AND DISPOSITION

- Treatment of bleeding in vWD depends on the type of vWD and the severity of the bleeding.
- Patients with type I vWD typically can be treated with DDAVP, which is administered intravenously, subcutaneously, or intranasally. Dental bleeding in patients with type I vWD also can be managed with aminocaproic acid.
- Patients with type II or type III vWD usually require transfusion of either vWF (in the form of cryoprecipitate) or factor VIII concentrate.

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134 HEMOLYTIC ANEMIAS

Sandra L. Najarian

HEREDITARY HEMOLYTIC ANEMIAS

EPIDEMIOLOGY

- Sickle cell disease (SCD) is inherited in an autosomal codominant pattern.
- Sickle cell trait, the most common variant of SCD, is found in 8 percent of the U.S. African-American population.
- Painful vasoocclusive crisis of SCD is the most common reason for emergency department (ED) visits. In children, 80 percent of vasoocclusive events are infection-related. In adults, the majority of crises are unexplained; however, up to one-third may be related to infection.
- Heterozygous hemoglobinopathies, such as hemoglobin sickle cell disease and sickle β-thalassemias, result from the inheritance of a sickle cell gene and an abnormal β-chain gene.
- Glucose-6-phosphate dehydrogenase (G6PD) deficiency, the most common human enzyme defect, is an X-linked disorder. In the United States, it affects 15 percent of African-American males.
- Hereditary spherocytosis (HS), a red blood cell (RBC) membrane defect, is the most prevalent hereditary hemolytic anemia among people of northern European descent.

PATHOPHYSIOLOGY

- Hemoglobinopathies are the result of an inherited abnormality of one or more hemoglobins.
- Hemoglobin S (HbS), the most common hemoglobin variant, is caused by a single point mutation on the β chain.
- Vasoocclusive crisis results from sludging of sickling RBCs in the microcirculation, which causes infarction.
- Acidosis, increased 2,3-diphosphoglycerate, vascular stasis, dehydration, low oxygen tension, and the presence of increased HbS can shift the oxygen dissociation curve to the right and promote increased sickling.
- Hydroxyurea helps to increase the concentration of hemoglobin F and decreases the sickling phenomenon.

CLINICAL FEATURES

- SCD is a chronic hemolytic anemia, and patients often have cardiopulmonary disease, such as flow murmurs, congestive heart failure, cardiomegaly, and cor pulmonale. Icterus, hepatomegaly, and lower extremity ulcerations are not uncommon.
- Musculoskeletal pain is the most prominent manifestation of vasoocclusive crisis in SCD. Other clinical presentations of vasoocclusive crisis include abdominal pain, hypoxia and other pulmonary complaints, priapism, swelling of the hands and feet (dactylitis), and infarction of the renal medulla, causing flank pain and hematuria.
- Patients with central nervous system (CNS) crisis, the only painless vasoocclusive crisis, may present with headaches, transient ischemic attack, seizures, coma, cerebral infarction (in children), and cerebral hemorrhage (in adults).
- Common precipitants of vasoocclusive crisis include cold exposure, dehydration, high altitude, and infections, particularly with encapsulated organisms such as *Haemophilus influenzae* or pneumococci.
- Hematologic crisis presents with weakness, dyspnea, fatigue, worsening congestive heart failure, or shock in the setting of an acute drop in hematocrit. Acute splenic sequestration and aplastic crises are the two types of hematologic crises.
- Patients with sickle cell trait have minimal to no complications; sickling is present only under conditions of extreme hypoxia.
- Patients with sickle cell–hemoglobin C disease have mild to moderate hemolytic anemia, mild reticulocytosis, and splenomegaly.
- Patients with sickle cell β-thalassemia vary in clinical presentation from mild hemolytic anemia to vasoocclusive crises.
- Infection, exposure to oxidant drugs, metabolic acidosis, and ingestion of fava beans can precipitate an acute hemolytic crisis in patients with G6PD deficiency.
- Patients with HS have mild hemolytic anemia, splenomegaly, and intermittent jaundice.

DIAGNOSIS AND DIFFERENTIAL

- SCD is usually diagnosed early in the patient's life. Presence of sickling RBCs on peripheral blood smear is diagnostic.
- Obtaining a complete blood cell count and reticulocyte count may be helpful in an acute SCD crisis. A reticulocyte count below the baseline of 5 to 15 percent may reflect aplastic crisis. Electrolytes,

liver enzymes, sedimentation rate, arterial blood gas, urinalysis, and radiographic studies should be obtained in the appropriate clinical presentation.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment for painful vasoocclusive crises of SCD includes IV hydration, analgesia, and supplemental oxygen. Patients who present with an infectious source should have the appropriate cultures obtained and be covered with broad-spectrum antibiotics.
- Admission criteria include pulmonary, neurologic, aplastic, or infectious crises; splenic sequestration; unremitting pain crisis; persistent nausea and vomiting; or uncertainty of diagnosis.

ACQUIRED HEMOLYTIC ANEMIAS

EPIDEMIOLOGY

- Warm antibody-mediated hemolytic anemia is more common in elderly female patients with underlying medical conditions. In children, it can occur after acute infections and immunizations.
- Cold antibody-mediated hemolytic anemia is more common in younger people, particularly after acute infections, as with *Mycoplasma pneumoniae.* It is also found in elderly patients with chronic disease.
- Drugs—such as α-methyldopa, penicillin, sulfa drugs, and quinidine—can cause an autoimmune hemolytic anemia.
- Thrombotic thrombocytopenic purpura (TTP) is more common in women between ages 10 and 60.
- Hemolytic uremic syndrome (HUS) is a disease of infancy and early childhood, with a peak incidence between 6 months and 4 years of age.

PATHOPHYSIOLOGY

- Acquired hemolytic anemias are due to autoimmune antibodies, fragmentation (microvascular and macrovascular), direct toxic effects, mechanical injury, or abnormal spleen function (Table 134-1).

CLINICAL FEATURES

- Presentations of antibody-mediated hemolytic anemias may range from mild to life-threatening anemia, splenomegaly, pulmonary edema, mental status changes, or venous thrombosis.

TABLE 134-1 Classification of Acquired Hemolytic Anemias

I. Autoimmune hemolytic anemia (antibody-mediated)
  A. Warm antibodies
  B. Cold antibodies
    1. Cold agglutinin disease
    2. Paroxysmal cold hemoglobinuria
  C. Drug-induced

II. Fragmentation hemolysis
  A. Microangiopathic hemolytic anemia (MAHA)
    1. Thrombotic thrombocytopenic purpura (TTP)/hemolytic uremic syndrome (HUS)
    2. Pregnancy-associated hemolysis (HELLP)
    3. Disseminated intravascular coagulation (DIC)
    4. Malignancy-associated hemolysis
    5. Hemolysis in vasculitis
    6. Hemolysis in malignant hypertension
  B. Macrovascular hemolysis
    1. Due to abnormal cardiac valves

III. Direct toxic effects causing hemolysis
  A. Infections
  B. Other toxins—bites, copper
  C. Drug-induced oxidative hemolysis—methemoglobinemia

IV. Mechanical damage causing hemolysis
  A. Heat denaturation
  B. March hemoglobinuria
  C. Cardiopulmonary bypass

V. Anemia due to abnormal splenic function (hypersplenism)

- Patients with TTP present with fever, neurologic changes, renal insufficiency, and hemorrhage.
- HUS is characterized by acute renal failure, microangiopathic hemolytic anemia (MAHA), fever, and thrombocytopenia.
- The HELLP syndrome is characterized by hemolysis, elevated liver enzymes, and low platelet counts in the presence of preeclampsia, eclampsia, or placental abruption.
- Oxidative hemolysis of RBCs results from methemoglobin-producing drugs, which include benzocaine, lidocaine, nitrates, nitrites, sulfonamides, phenacetin, sulfasalazine (Azulfidine), phenazopyridine (Pyridium), dapsone, and other antimalarials.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is based on clinical presentation and laboratory studies, including complete blood cell count, reticulocyte count, and peripheral blood smear (Table 134-2).
- The direct Coombs' test is positive in patients with immune-mediated hemolysis.
- Schistocytes are seen in fragmentation hemolysis. Spherocytes are evidence of warm antibody-mediated hemolysis and hereditary spherocytosis.

TABLE 134-2 Characteristics of Acquired Hemolytic Anemias*

	EVANS SYNDROME	TTP	HUS	DIC	HELLP
Autoimmune hemolytic anemia	Present	No	No	No	No
Microangiopathic hemolytic anemia (MAHA)	No	Prominent	Prominent	Often present	Present
Coombs test	Positive	Negative	Negative	Negative	Negative
Thrombocytopenia	Present	Prominent	Present	Present	Present
Renal abnormalities	No	Mild	Prominent	No	No
Neurologic abnormalities	No	Prominent	No or mild	No	No
Hepatic dysfunction	No	May have	May have	May have	Prominent
Fever	No	Present	Present	May have	No
Coagulation studies	Normal	Normal	Normal	Abnormal	Normal
Pregnancy-associated	No	Can be	Can be	Can be	Always

ABBREVIATIONS: TTP = thrombotic thrombocytopenic purpura; HUS = hemolytic uremic syndrome; DIC = disseminated intravascular coagulation; HELLP = hemolysis, elevated liver functions, low platelets.
* Disease descriptions here are based on presence of isolated disease without complications; individual patients often have other problems that make syndromes less readily identified.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment is directed at stabilization of vital signs and correction of the underlying disease process.
- The initial treatment for warm antibody-mediated hemolytic anemia is prednisone. Immunosuppressive therapy is used for refractory patients.
- Treatment for TTP includes plasma exchange transfusion, antiplatelet therapy with aspirin or dipyridamole, and prednisone or other immunosuppressive therapy. Platelet transfusion aggravates the thrombotic process and should be avoided.
- Treatment of HUS consists of hemodialysis for renal failure and supportive care.
- Treatment of HELLP syndrome consists of prompt delivery of the fetus and supportive care.

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135 BLOOD TRANSFUSIONS AND COMPONENT THERAPY

Keith L. Mausner

This chapter reviews blood and component therapy, complications of transfusions, emergency transfusion, massive transfusion, and blood administration.

WHOLE BLOOD

- Whole blood provides both volume and oxygen-carrying capacity. However, this is better achieved

using packed red blood cells (PRBCs) and crystalloid solution.
- One unit contains approximately 500 mL of blood plus a preservative-anticoagulant, usually citrate phosphate dextrose adenine.
- Among its disadvantages, whole blood has (1) low levels of clotting factors; (2) often, elevated levels of potassium, hydrogen ion, and ammonia; (3) a large number of antigens; (4) the potential for volume overload before needed components are replaced.

PACKED RED BLOOD CELLS

- One unit of PRBCs raises an adult patient's hemoglobin by approximately 1 g/dL or the hematocrit by 3 percent.
- Advantages over whole blood include reduced risk of volume overload; decreased infusion of citrate, ammonia, and organic acids; and decreased risk of alloimmunization because of exposure to fewer antigens.
- Major indications for PRBC infusion include (1) acute hemorrhage: blood loss greater than 25 to 30 percent of blood volume (1500 mL) in otherwise healthy adults usually requires PRBC transfusion to replace oxygen-carrying capacity and crystalloid infusion to replace volume, and (2) chronic anemia: transfusion may be indicated for symptomatic patients, patients with cardiopulmonary disease, and those with hemoglobin levels less than 7 g/dL.
- RBCs are available as leukocyte-poor, frozen, or washed.
- Leukocyte-poor RBCs have up to 85 percent of leukocytes removed. They are indicated for transplant recipients or candidates and patients with a history of febrile nonhemolytic transfusion reactions.
- Frozen RBCs are a source of rare blood types and provide reduced antigen exposure.
- Washed RBCs are indicated for patients with hypersensitivity reactions to plasma, for neonatal transfusions, and for those with paroxysmal nocturnal hemoglobinuria.

PLATELETS

- One unit contains about 4×10^{11} platelets in a volume of 250 to 350 mL.
- Platelets are usually transfused 6 U at a time, which raises the platelet count to about 50,000/μl.
- ABO- and Rh-compatible platelets are preferable.
- The platelet count should be checked at 1 and 24 h after infusion.
- Transfused platelets survive 3 to 5 days unless there is platelet consumption.
- Principles for platelet transfusion in adults are as follows: (1) If the platelet count is above 50,000/μl, bleeding from thrombocytopenia is unlikely unless there is platelet dysfunction. (2) The platelet count should be maintained above 50,000/μl in patients undergoing major surgery or with significant bleeding. (3) A platelet count between 10,000 and 50,000/μl increases the risk of bleeding from trauma or invasive procedures, and spontaneous bleeding may be seen in patients with platelet dysfunction (e.g., renal or liver disease). (4) A platelet count below 10,000/μl presents a high risk of spontaneous bleeding and preventative transfusion is indicated. (5) In immune thrombocytopenia, transfusion may have little effect due to platelet destruction.

FRESH-FROZEN PLASMA

- One bag of fresh-frozen plasma (FFP) has a volume of 200 to 250 mL, 1 U/mL of each coagulation factor, and 1 to 2 mg/mL of fibrinogen.
- FFP should be ABO-compatible.
- The usual initial dose is 8 to 10 mL/kg, or 2 to 4 bags of FFP.
- FFP is indicated for (1) acquired coagulopathy with active bleeding or before invasive procedures when there is greater than 1.5 times prolongation of the prothrombin time (PT) or activated partial thromboplastin time (aPTT) or a coagulation factor assay less than 25 percent of normal; (2) congenital isolated factor deficiencies when specific virally safe products are not available; (4) thrombotic thrombocytopenic purpura (TTP) patients undergoing plasma exchange; (5) patients receiving massive transfusion who develop coagulopathy and active bleeding; (6) antithrombin III deficiency when antithrombin III concentrate is not available.

CRYOPRECIPITATE

- Cryoprecipitate is derived from FFP.
- One bag of cryoprecipitate contains 80 to 100 U factor VIIIC, 80 U of von Willebrand factor, 200 to 300 mg of fibrinogen, 40 to 60 U of factor XIII, and variable amounts of fibronectin.
- The usual dose is 2 to 4 bags per 10 kg of body

weight, or 10 to 20 bags. ABO-compatible bags are preferable.
- Indications for cryoprecipitate include (1) fibrinogen level less than 100 mg/dL associated with disseminated intravascular coagulation (DIC) or congenital fibrinogen deficiency; (2) von Willebrand disease with active bleeding when desmopressin (DDAVP) is not effective and factor VIII concentrate containing von Willebrand factor is not available; (3) hemophilia A when virally safe (recombinant or monoclonal antibody purified) factor VIII concentrates are not available; (4) use as a fibrin glue surgical adhesive; (5) fibronectin replacement.

ALBUMIN

- Albumin is available as 5% and 25% solutions in saline.
- Albumin does not transmit viral diseases; however, its use is controversial and it is rarely infused.

IMMUNOGLOBULINS

- Indications for intravenous immunoglobulins (IVIg) include (1) treatment of primary and secondary immunodeficiency and (2) treatment of immune or inflammatory disorders including immune thrombocytopenia and Kawasaki syndrome.
- Several cases of hepatitis C have been documented from IVIg.

ANTITHROMBIN III

- Antithrombin III (ATIII) is a serum protein that inhibits coagulation factors, thrombin, and activated factors IX, X, XI, and XII.
- ATIII deficiency can be acquired or inherited.
- ATIII is indicated for prophylaxis of thrombosis or to treat thromboembolism in patients with congenital ATIII deficiency.

SPECIFIC FACTOR REPLACEMENT THERAPY

- Table 135-1 outlines therapy for congenital deficiencies of coagulation factors.

TABLE 135-1 Replacement Therapy for Congenital Factor Deficiencies

COAGULATION FACTOR	INCIDENCE*	REPLACEMENT THERAPY
Factor I (fibrinogen)	150 cases	Cryoprecipitate
Factor II (prothrombin)	>30 cases	FFP for minor bleeding episodes Prothrombin complex concentrate for major bleeding
Factor V	150 cases	FFP
Factor VII	150 cases	FFP for minor bleeding episodes Prothrombin complex concentrates for major bleeding Recombinant factor VIIA (experimental)
Factor VIII‡	1 in 10,000 males	Factor VIII concentrates (cryoprecipitate or FFP if not available) Desmopressin for those with mild hemophilia
von Willebrand disease	up to 1 in 100 persons	Desmopressin (or some factor VIII concentrates or cryoprecipitate)
Factor IX	1 in 30,000 males	Factor IX concentrates
Factor X	1 in 500,000	FFP for minor bleeding episodes Prothrombin complex concentrates for major bleeding
Factor XI†	3 in 10,000 Ashkenazi Jews 1 in 1,000,000 in general	FFP
Factor XII	Several hundred cases	Replacement not required
Factor XIII	>100 cases	FFP or cryoprecipitate

* Incidence as of 1998.
† Factor XI levels correlate poorly with bleeding complications; many patients have low levels, but no bleeding complications.

IMMEDIATE TRANSFUSION REACTIONS AND COMPLICATIONS

- Table 135-2 summarizes the types of immediate reactions as well as their recognition, management, and evaluation.
- Adverse reactions occur in up to 20 percent of transfusions and are usually mild.
- Transfusion reactions can be immediate or delayed.

TABLE 135-2 Acute Transfusion Reactions: Recognition, Management, Evaluation

REACTION TYPE	SIGNS AND SYMPTOMS	MANAGEMENT	EVALUATION
Acute intravascular hemolytic reaction	Fever, chills, low back pain, flushing dyspnea, tachycardia, shock, hemoglobinuria	Immediately stop transfusion IV hydration to maintain diuresis; diuretics may be necessary Cardiorespiratory support as indicated Can be life threatening	Retype and cross-match Direct and indirect Coombs tests CBC, creatinine, PT, aPTT Haptoglobin, indirect bilirubin, LDH, plasma, free hemoglobin Urine for hemoglobin
Acute extravascular hemolytic reaction	Often have low-grade fever but may be entirely asymptomatic	Stop transfusion Rarely causes clinical instability	Hemolytic workup as above to rule out the possibility of intravascular hemolysis
Febrile nonhemolytic transfusion reaction	Fever, chills	Stop transfusion Manage as in intravascular hemolytic reaction (above) because cannot initially distinguish between the two Can treat fever and chills with acetaminophen and meperidine Usually mild but can be life-threatening in patients with tenuous cardiopulmonary status Consider infectious workup	Hemolytic workup as above because initially cannot distinguish the etiology
Allergic reaction	If mild, urticaria, pruritus If severe, dyspnea, bronchospasm, hypotension, tachycardia, shock	Stop transfusion If mild reaction, can treat with diphenhydramine; if symptoms resolve, can restart transfusion If severe, may require cardiopulmonary support; do not restart transfusion	For mild symptoms that resolve with diphenhydramine, no further workup is necessary, although blood bank should be notified For severe reaction, do hemolytic workup as above because initially will be indistinguishable from a hemolytic reaction
Hypervolemic	Dyspnea, tachycardia, hypertension, headache, jugular venous distention, pulmonary rales, hypoxia	Stop transfusion or decrease rate to 1 mL/kg/h Diuresis Can be difficult to distinguish from a hemolytic reaction; if cannot distinguish, stop transfusion and treat as if intravascular hemolytic reaction	If clearly hypervolemic, no further evaluation is needed; CXR may be helpful If hemolytic reaction is a possibility, do hemolytic workup as above

ABBREVIATIONS: IV = intravenous; CBC = complete blood count; PT = prothrombin time; aPTT = activated partial thromboplastin time; LDH = lactate dehydrogenase; CXR = chest radiograph.

DELAYED TRANSFUSION REACTIONS

- There is a small risk of transmission of HIV, hepatitis B and C, cytomegalovirus, parvovirus, and human T-cell lymphotropic viruses I and II. Other rare pathogens include Epstein-Barr virus, syphilis, malaria, babesiosis, toxoplasmosis, and trypanosomiasis.
- Delayed hemolytic reactions can occur 7 to 10 days after transfusion.
- Hypothermia may occur from rapid transfusion of refrigerated blood.
- Noncardiogenic pulmonary edema may be caused by incompatible passively transferred leukocyte antibodies and usually occurs within 4 h of transfusion. Clinical findings are respiratory distress, fever, chills, tachycardia, and patchy infiltrates on chest x-ray without cardiomegaly and without evidence of fluid overload. Most cases resolve with supportive care.
- Electrolyte imbalance may occur. Citrate in preservative solution chelates calcium. Significant hypocalcemia is rare even in massive transfusion, because patients with normal hepatic function easily metabolize citrate into bicarbonate. Hypokalemia may occur with large transfusions due to metabolism of citrate to bicarbonate, which produces metabolic acidosis and drives potassium ions to the intracellular space. Hyperkalemia may occur in patients with renal failure or in neonates.
- Graft-versus-host disease occurs when nonirradiated lymphocytes are inadvertently transfused into an immunocompromised patient. This event is fatal in over 90 percent of cases.

EMERGENCY TRANSFUSIONS

- Type O or type-specific incompletely cross-matched blood may be lifesaving, but there is a risk of life-threatening transfusion reactions. Use should be limited to the early resuscitation of patients with severe hemorrhage without adequate response to crystalloid infusion.
- Before transfusing, blood should be obtained for baseline laboratory tests and type- and cross-matching.
- Type-specific blood can be available in 15 min and fully cross-matched blood in 30 to 60 min.
- Rh-negative blood is preferable when it is not fully cross-matched.
- Only PRBCs are available for emergency transfusion; plasma products contain too many antigens.

MASSIVE TRANSFUSION

- Massive transfusion is the replacement of a patient's blood volume within a 24-h period.
- Complications of massive blood transfusion include the following: (1) Bleeding may result from thrombocytopenia, platelet dysfunction, DIC, or coagulation factor deficiencies. Platelet transfusions are indicated for thrombocytopenia with bleeding. FFP is indicated for coagulopathy and bleeding. (2) Hypocalcemia may occur from citrate toxicity, which is more likely in patients with liver disease, neonates, and those receiving more than 5 U of whole blood. The QT interval is not a reliable indicator of hypocalcemia in this setting; an ionized calcium level is necessary. Hypocalcemia is treated with 5 to 10 mL of IV calcium gluconate slowly. (3) Hypothermia may occur, which is more likely when administering 3 U or more of blood rapidly.

BLOOD ADMINISTRATION

- A 16-gauge or larger IV catheter is preferred to prevent hemolysis and permit rapid infusion.
- Micropore filters should be used to remove microaggregates of platelets, fibrin, and leukocytes.
- Normal saline is the only crystalloid compatible with PRBCs.
- Warmed saline solution (39° to 43°C, or 102.2° to 109.4°F) may be given concurrently or a blood warmer used to prevent hypothermia. Blood will hemolyze if warmed to greater than 40°C (104°F).
- Rapid infusion may be facilitated with pressure infusion devices.
- Patients at risk for hypervolemia should receive each unit over 3 to 4 h.

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136 EXOGENOUS ANTICOAGULANTS AND ANTIPLATELET AGENTS

Kathleen F. Stevison

Arterial thrombi are composed primarily of platelets bound by thin fibrin strands. Venous thrombi are composed of red blood cells and large fibrin strands. Antithrombotic therapy must be component-directed. Antithrombotic agents are grouped by mechanism: anticoagulants that block clotting factors, antiplatelet drugs that interfere with platelet function, and fibrinolytic agents (or thrombolytic agents) that dissolve fibrin.

ANTITHROMBOTIC AGENTS

ORAL ANTICOAGULANT

- Warfarin inhibits vitamin K–dependent clotting factors (II, VII, IX, X).
- Dosing is guided by the international normalized ratio (INR), derived from the prothrombin time (PT); the desired INR is usually between 2.0 and 2.5. Onset of anticoagulation occurs after 3 to 4 days.
- Warfarin also affects proteins C and S, and for 24 to 36 h there may be a hypercoagulable effect; this is minimized by a starting dose of 5 mg/day. In situations where immediate anticoagulation is critical, a heparin product should be used until an adequate INR is achieved. Warfarin is contraindicated in pregnancy.

PARENTERAL ANTICOAGULANTS

- Unfractionated heparin forms a complex with antithrombin III (ATIII), which inhibits factors IXa and Xa.
- Body weight–based IV dosing is recommended, typically 70 to 80 U/kg IV bolus, followed by IV infusion at 15 to 18 U/kg/h. Therapy is monitored by the activated partial thromboplastin time (aPTT); the therapeutic range is 1.5 to 2.5 times the normal value.
- Low-molecular-weight (LMW) heparin fractions (enoxaparin, dalteparin, and ardeparin) are derived from unfractionated heparin. These agents are effective when administered SC once to twice daily. LMW heparin is used for both prophylaxis and treatment.
- Enoxaparin is FDA-approved for the prophylaxis of deep venous thrombosis (DVT) and for treatment of DVT with or without pulmonary embolism (PE), non-Q-wave myocardial infarction (MI), and unstable angina.
- For DVT, PE, and unstable angina, enoxaparin 1 mg/kg SC bid or dalteparin 100 IU/kg SC bid may be used.
- The heparins and danaparoid may be used in pregnancy.
- LMW heparins and danaparoid produce minimal elevation in prothrombin time (PT) or aPTT; laboratory monitoring is not routinely necessary except in renal failure, where anti-Xa activity can be measured.

PLATELET ACTIVATION BLOCKER

- Aspirin blocks the enzyme cyclooxygenase, which results in inhibition of platelet activation. The inhibitory effect is irreversible and lasts for the life span of the platelet (10 days).

PLATELET AGGREGATION BLOCKERS

- Platelet aggregation involves binding of fibrinogen to the platelet glycoprotein IIb-IIIa receptor.
- The platelet membrane–altering agents ticlopidine and clopidogrel render the receptor ineffective and block aggregation. These agents are generally used in patients who are intolerant of or have failed aspirin therapy. Glycoprotein IIB-IIIa inhibitors (abciximab, eptifibatide, and tirofiban) have been found beneficial in patients with acute MI and unstable angina and those undergoing percutaneous angioplasty.

FIBRINOLYTIC AGENTS

- Fibrinolytics work by activating plasminogen to plasmin, which then dissolves the fibrin in thrombi. Streptokinase (SK) is usually administered as 1.0 to 1.5 million U over 60 min.
- Anistreplase (APSAC) is derived from and has an effect similar to that of SK, but it can be administered as a slow bolus (typically 30 mg over 5 min).
- Tissue plasminogen activator (tPA), in theory, produces less systemic fibrinolysis and is more ''clot specific.'' In acute MI, a front-loaded regimen is commonly used: a 15-mg bolus, then 0.75 mg/kg over 30 min (maximum 50 mg), and then 0.50 mg/kg over 60 min (maximum 35 mg).
- Reteplase, a derivative of tPA, is administered as a double bolus (10-U bolus, repeated in 30 min). SK and APSAC are more antigenic than tPA or reteplase and therefore are more likely to produce allergic reactions.
- Though administered infrequently, urokinase is used for indwelling catheter–associated thrombosis.

INDICATIONS FOR ANTITHROMBOTIC THERAPY

ACUTE MYOCARDIAL INFARCTION

- Fibrinolytic therapy should be initiated within 30 min of patient arrival at the emergency department (ED). In the appropriate clinical setting, criteria for fibrinolytic therapy include (1) presentation within 12 h of symptom onset; (2) ST-segment elevation in two or more contiguous leads or new-

onset left bundle-branch block; and (3) absence of contraindications (Table 136-1).
- Angioplasty is preferred over fibrinolysis in cardiogenic shock.
- Rapid initiation of fibrinolytic therapy is more important than the specific agent used. However, tPA is the agent of choice with a history of the following: (1) allergy to SK or APSAC; (2) treatment with SK in the previous 6 months or with APSAC in the previous 12 months; (3) streptococcal infection in the previous 12 months; or (4) hemodynamic instability.
- Aspirin should be administered immediately. Unfractionated heparin should be administered to patients who have received tPA.

DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

- Treatment can be initiated with either unfractionated or LMW heparin.
- Selected patients may benefit from fibrinolytic therapy followed by heparin.

ISCHEMIC STROKE

- TPA may benefit some stroke patients if given within 3 h of symptom onset, although there is an increased risk of intracranial hemorrhage.
- Thrombolytic agents should be withheld from patients with rapidly improving symptoms, pretreatment hypertension (>185/110 mmHg), and signs or history of hemorrhagic stroke.

TABLE 136-1 Contraindications to Fibrinolytic Therapy

ABSOLUTE
Active or recent internal bleeding (≤14 d)
CVA <2–6 months or hemorrhagic CVA
Intracranial or intraspinal surgery or trauma <2 months
Intracranial or intraspinal neoplasm, aneurysm, or arteriovenous malformation
Known severe bleeding diathesis
On anticoagulants (warfarin, PT >15 s, heparin, increased aPTT)
Uncontrolled hypertension (i.e., blood pressure >185/100 mmHg)
Suspected aortic dissection or pericarditis
Pregnancy
RELATIVE
Active peptide ulcer disease
Cardiopulmonary resuscitation >10 min
Hemorrhagic ophthalmic conditions
Puncture of noncompressible vessel <10 d
Advanced age >75 years
Significant trauma or major surgery >2 weeks and <2 months
Advanced kidney or liver disease

Concurrent menses is *not* a contraindication
In ischemic CVA, symptoms >3 h, severe hemispheric stroke, platelets <100/μL, and glucose <50 or >400 mg/dL are additional contraindications.

COMPLICATIONS OF ANTITHROMBOTIC THERAPY

- Warfarin anticoagulation may be reversed by vitamin K_1, fresh-frozen plasma (FFP), and coagulation factor concentrates.
- Warfarin has many potential drug interactions, especially with antibiotics as well as drugs that affect the cytochrome P450 system; the most serious interactions can markedly increase the PT and lead to bleeding complications. Another complication of warfarin is skin necrosis, which primarily affects individuals with protein C deficiency.
- Heparin-associated bleeding is first treated by stopping the infusion. In severe cases protamine (1 mg IV per 100 U of heparin in previous 4 h) reverses the effect of heparin.
- LMW heparins cause less bleeding than unfractionated heparin. Reversal of LMW heparins by protamine is compound-specific; enoxaparin is only partially reversed.
- Heparin-induced thrombocytopenia (HIT) is a potentially deadly complication that affects 3 percent of patients on unfractionated heparin and fewer patients on LMW heparins. HIT is antibody-mediated, causing platelet activation, thrombocytopenia, and thrombosis; onset is usually 5 to 12 days into treatment. Heparin therapy is stopped as soon as HIT is recognized. Platelet counts usually recover in 4 to 6 days.
- Aspirin-related life-threatening GI bleeding is uncommon. Severe hemorrhage may respond to transfusion of functional platelets to increase the platelet count by 50,000/μL (6 U of platelets).
- Fibrinolytic therapy–related bleeding can be minimized by avoiding administration to patients with absolute contraindications. External bleeding can be controlled by local pressure. Major hemorrhage mandates replacement of coagulation factors (see Chap. 135). Intracranial hemorrhage requires rapid coagulation factor replacement and immediate neurosurgical consultation.

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137 EMERGENCY COMPLICATIONS OF MALIGNANCY

John Sverha

SPINAL CORD COMPRESSION

EPIDEMIOLOGY

- Spinal cord compression most often occurs as a complication of multiple myeloma, lymphoma, breast cancer, prostate cancer, and lung cancer.

PATHOPHYSIOLOGY

- Neurologic symptoms are caused by direct pressure on the spinal cord by a primary tumor or by metastases.

CLINICAL FEATURES

- Back pain is typically progressive over weeks.
- Neurologic symptoms include leg weakness or numbness and urinary retention.
- Physical examination may reveal vertebral percussion tenderness, decreased rectal tone, saddle anesthesia, and diminished lower extremity reflexes.

DIAGNOSIS AND DIFFERENTIAL

- All patients with back pain and a history of cancer should receive radiographs.
- Patients with signs or symptoms of cord compression require emergency magnetic resonance imaging scanning or computed tomography (CT) with myelography.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with symptoms of cord compression should receive immediate administration of dexamethasone 10 to 25 mg intravenously (IV).
- Consultation is required to determine need for radiation therapy or surgical decompression.

UPPER AIRWAY OBSTRUCTION

EPIDEMIOLOGY

- Upper airway obstruction is usually a late manifestation of a variety of tumors arising in the neck, oropharynx, or superior mediastinum.

PATHOPHYSIOLOGY

- Acute compromise often occurs when new bleeding, secretions, or infection obstructs an existing stricture.

CLINICAL FEATURES

- A change in voice often occurs in the weeks preceding the obstruction.
- The new onset of stridor indicates acute compromise.

DIAGNOSIS AND DIFFERENTIAL

- The presence of a foreign body or infection can produce symptoms similar to those of tumor expansion.
- Soft tissue views of the neck and fiberoptic laryngoscopy can be helpful.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The airway should be suctioned and supplemental oxygen administered. Heliox may be used as a temporizing measure.
- Patients with impending airway obstruction require immediate intervention to create a secure and patent airway. Ideally, this should be in the operating room after otolaryngology consultation. Otherwise, bedside orotracheal intubation or cricothyroidotomy should be performed.

MALIGNANT PERICARDIAL EFFUSION

EPIDEMIOLOGY

- Common causes of malignant pericardial effusion include breast carcinoma, lung carcinoma, and malignant melanoma. Pericardial effusions can also be caused by therapeutic irradiation and some chemotherapeutic agents.

PATHOPHYSIOLOGY

- The degree of cardiac dysfunction depends on the volume of the effusion and the speed of its accumulation. Sudden or large (>500 mL) effusions compress the right ventricle and reduce cardiac output.

CLINICAL FEATURES

- Classic features of tamponade include (a) hypotension and a narrowed pulse pressure, (b) jugular venous distention, (c) diminished heart sounds, (d) pulsus paradoxus >10 mmHg, (e) low QRS voltage or electrical alternans on electrocardiogram (ECG), and (f) cardiomegaly without congestive heart failure on chest radiograph.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis should be considered in any cancer patient with dyspnea or hypotension. Definitive diagnosis is obtained through echocardiography.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients in extremis should have emergency pericardiocentesis performed. Other patients with malignant pericardial effusions should have their care plan developed in consultation with an oncologist.

SUPERIOR VENA CAVA SYNDROME

EPIDEMIOLOGY

- Superior vena cava (SVC) syndrome is commonly associated with lung carcinoma, breast carcinoma, or lymphoma.

PATHOPHYSIOLOGY

- Superior vena cava syndrome may occur through tumor compression or invasion of the SVC. Superimposed thrombosis within the SVC often occurs as well.

CLINICAL FEATURES

- The onset is typically insidious, and patients may complain of headache, edema of the face or arms, or a vague sensation of head fullness. With disease progression, an increase in intracranial pressure (ICP) can cause confusion, seizure, or coma.
- Physical examination may reveal neck and upper chest vein distention, edema of the face or arms, facial telangiectasia, and sometimes a palpable supraclavicular mass. Papilledema indicates critically high ICP.

DIAGNOSIS AND DIFFERENTIAL

- Chest radiograph may reveal mediastinal widening or a lung mass. Definitive diagnosis is through contrast-enhanced chest CT scan or venography.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Administration of furosemide 40 mg IV and methylprednisolone 120 mg IV may be effective temporizing measures in patients with evidence of elevated ICP.
- Chemotherapy and radiation therapy should be initiated after the specific tumor type is identified.

HYPERCALCEMIA OF MALIGNANCY

EPIDEMIOLOGY

- Hypercalcemia of malignancy is typically associated with multiple myeloma, lung carcinoma, breast carcinoma, renal cell carcinoma, and lymphoma.

PATHOPHYSIOLOGY

- Hypercalcemia of malignancy is usually produced by osteolysis caused by bony metastases. Patients without bony metastases can develop hypercalcemia through the release of tumor-produced hormone-like substances. Squamous cell carcinoma of the lung is known to produce a parathyroid-like substance.

CLINICAL FEATURES

- Symptoms include nausea, constipation, abdominal pain, weakness, confusion, and coma. Hypercalcemia also causes a diuresis that results in dehydration.
- The QT interval on the ECG may shorten as the calcium level rises.

DIAGNOSIS AND DIFFERENTIAL

- Serum calcium determinations should consider the albumin level or preferably measure ionized calcium directly as this best correlates with symptoms. Patients tolerate greater degrees of hypercalcemia if it is gradual in onset.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- If significant symptoms are present or if calcium levels are >14 mg/dL, treatment with normal saline (NS) infusion (1 to 2 L) and furosemide diuresis (40 to 80 mg IV) is indicated. Other treatments such as phosphate, mithramycin, and prednisone are slower in onset and should be discussed with an oncologist before being initiated.

TUMOR LYSIS SYNDROME

EPIDEMIOLOGY

- Tumor lysis syndrome is most commonly seen after chemotherapy of hematologic malignancies, especially Burkitt's lymphoma.

PATHOPHYSIOLOGY

- Rapid destruction of tumor cells results in hyperkalemia, hyperuricemia, and hyperphosphatemia. Hypocalcemia develops secondary to hyperphosphatemia.

CLINICAL FEATURES

- Tumor lysis syndrome most commonly occurs 1 to 5 days after chemotherapy or radiation therapy. It is more common in patients with underlying renal insufficiency.
- Hyperkalemia can cause life-threatening dysrhythmias.
- Hyperuricemia and hyperphosphatemia can cause renal failure.
- Hypocalcemia can cause muscle cramps, confusion, and seizures.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Vigorous hydration, urinary alkalinization, and allopurinol administration can all be used to promote uric acid excretion.
- Emergency hemodialysis should be considered in the setting of serum potassium >6.0 meq/L, uric acid >10.0 mg/dL, phosphate >10 mg/dL, creatinine >10 mg/dL, or symptomatic hypocalcemia.

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE

EPIDEMIOLOGY

- Syndrome of inappropriate antidiuretic hormone (SIADH) is commonly associated with small cell lung carcinoma, primary and metastatic brain can-

cer, pancreatic adenocarcinoma, and prostate carcinoma.

PATHOPHYSIOLOGY

- Antidiuretic hormone is secreted by tumor cells in the absence of an appropriate physiologic stimulus. This results in the production of concentrated urine despite euvolemic hyponatremia.

CLINICAL FEATURES

- The symptoms of SIADH are those of hyponatremia. Depending on the degree of hyponatremia, the patient may demonstrate nausea, vomiting, weakness, confusion, seizures, and coma.

DIAGNOSIS AND DIFFERENTIAL

- The hallmarks of SIADH are hyponatremia, less than maximally dilute urine, and urine sodium concentration >30 meq/L in the setting of euvolemia.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Mild hyponatremia can be treated with water restriction.
- Patients with serum sodium levels <115 meq/L and central nervous system (CNS) signs and symptoms should be treated with hypertonic (3%) saline infusion. Care should be taken to correct sodium levels no faster than 1 meq/L/h to avoid central pontine myelinolysis.

HYPERVISCOSITY SYNDROME

EPIDEMIOLOGY

- Hyperviscosity syndrome is typically associated with Waldenström's macroglobulinemia (most common cause), multiple myeloma, cryoglobulinemia, various leukemias, and polycythemia vera.

PATHOPHYSIOLOGY

- Severe increases in serum proteins (typically immunoglobulins), red blood cell concentration, or white blood cell (WBC) concentration can cause a dangerous increase in blood viscosity.
- Increased blood viscosity can result in sludging, stasis, and a reduction in microcirculatory blood flow.

CLINICAL FEATURES

- Early symptoms include fatigue, headache, and somnolence.
- As viscosity increases, microthromboses may cause visual disturbances, deafness, seizures, stroke, and coma. Congestive heart failure and myocardial infarction have also been reported.

DIAGNOSIS AND DIFFERENTIAL

- Physical examination of the ocular fundi may reveal ''sausage-linked'' retinal vessels, hemorrhages, and exudates.
- Patients with hyperviscosity due to erythrocytosis typically have a hematocrit >60 percent. Those with hyperviscosity due to leukocytosis typically have WBC concentrations >100,000 cells per microliter.
- Patients with hyperviscosity due to increased serum proteins may show evidence of rouleau formation on the peripheral blood smear. Serum or urine protein electrophoresis is diagnostic.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Definitive treatment of symptomatic hyperviscosity due to increased serum proteins is emergency plasmapheresis. Temporizing measures include phlebotomy (2 U) and infusion of 1 to 2 L of NS.
- Definitive treatment of symptomatic hyperviscosity due to leukocytosis is leukapheresis. Symptomatic hyperviscosity caused by erythrocytosis is treated by phlebotomy (2 U) and infusion of 1 to 2 L of NS.

NEUTROPENIA AND INFECTION

PATHOPHYSIOLOGY

- Many chemotherapeutic agents cause myelosuppression and result in neutropenia days after their administration.

CLINICAL FEATURES

- Patients with neutropenia and fever often do not have focal symptoms.

DIAGNOSIS AND DIFFERENTIAL

- Patients with an absolute neutrophil count <500 cells per microliter and a fever >38.3°C (100.9°F) are at high risk for infection.
- Approximately two-thirds of cancer patients who are neutropenic with a fever will have a bacterial cause of their fever.
- A thorough physical exam including examination for possible cellulitis and perirectal abscess should be performed.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients should have blood and urine cultures obtained prior to antibiotic therapy.
- All patients with an absolute neutrophil count <500 cells and a fever >38.3°C (100.9°F) should have empiric antibiotic therapy initiated. Additional antibiotic coverage should be directed at any obvious sources of infection.
- Monotherapy with a third-generation cephalosporin such as ceftazidime or cefepime is considered adequate empiric antibiotic coverage. Vancomycin may be added on the basis of clinical suspicion or local institutional bacterial sensitivities.

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Section 17
NEUROLOGY

138 HEADACHE AND FACIAL PAIN

Philip B. Sharpless

EPIDEMIOLOGY

- Headaches are classified into the primary and secondary causes noted in Table 138-1.
- One study revealed that 3.8 percent of emergency department (ED) headache patients have serious or secondary pathology.
- Subarachnoid hemorrhage (SAH) represents about 1 percent of all nontraumatic headaches[1] and accounts for up to 25 percent of all sudden, severe headaches.[2]
- The prevalence of migraine is approximately 15 to 17 percent in women and 5 percent in men.[3]

PATHOPHYSIOLOGY

- Migraine auras are thought to be the result of a slowly spreading wave of neuronal hypoactivity with an associated secondary reduction in local blood flow. Since vascular territories are not respected, the cause of the aura is no longer considered primarily vasospastic.[4]
- The migraine headache is thought to result from sterile neurogenic inflammation of pain-sensitive intracranial structures (arteries, dura), and this promotes a secondary vasodilation. No consensus exists on the precise biochemical triggers that initiate the migraine.[4]

CLINICAL FEATURES

- Table 138-2 lists the findings on the patient's history and physical examination, which should alert the clinician to the possibility of a more serious secondary cause for the headache and prompt consideration of more extensive testing.
- Focal or nonfocal neurologic findings in the patient with a headache have a 39 percent predictive value for intracranial pathology.[1]
- Migraine headaches are typically gradual in onset, unilateral, and throbbing; they last 4 to 72 h, with frequent nausea and vomiting. The majority (80 percent) present without an aura. The aura in the remainder develops over 5 to 20 min, lasts no more than 60 min, and may consist of visual changes (scintillating scotomas, flashes) or other neurologic symptoms (focal weakness, paresthesias, vertigo, etc.).
- Tension headaches tend to be gradual in onset, bilateral, nonpulsating, and—unless very severe—without nausea and vomiting. Overlap with migrainous headache symptoms occurs.
- Cluster headaches are rare, occur primarily in men, and are typified by intense, unilateral, periorbital pain lasting 15 to 180 min. The headaches recur in ''clusters,'' often at the same time daily for weeks before remitting. Some combination of ipsilateral conjunctival injection, tearing, nasal congestion, or rhinorrhea is seen.
- Subarachnoid hemorrhage (SAH) is most commonly a sudden-onset, severe headache and is often described as ''the worst headache of my life.'' The headache may be global or unilateral but is frequently occipital and radiates to the neck and back.[5] Syncope, mental status changes, vomiting, meningismus, focal cranial nerve deficits (typically oculomotor nerve), or other neurologic deficits

TABLE 138-1 Primary and Secondary Causes of Headache

PRIMARY HEADACHE SYNDROMES
Migraine
Tension type
Cluster
SECONDARY CAUSES OF HEADACHE
Vascular
Subarachnoid hemorrhage
Intraparenchymal hemorrhage
Subdural or epidural hematoma
Ischemia (stroke, TIA)
Cavernous sinus thrombosis
Arteriovenous malformation
Temporal arteritis
Carotid or vertebral artery dissection
CNS infection
Meningitis (bacterial, viral, other)
Encephalitis
Cerebral abscess
Non-CNS infection
Focal or systemic
Sinusitis
Herpes zoster of face or scalp
Other CNS
Tumor (benign or malignant)
Pseudotumor cerebri
Ophthalmic
Glaucoma
Iritis
Optic neuritis
Drug-related and toxic or metabolic
Nitrates and nitrites
MAOI drugs
Chronic analgesic use and abuse
Hypoxia or high altitude
Hypercapnia
Hypoglycemia
Monosodium glutamate
Carbon monoxide poisoning
Alcohol withdrawal
Miscellaneous
Malignant hypertension
Preeclampsia
Pheochromocytoma
Fever
Post–lumbar puncture
Dental (referred)
Otic (referred)

ABBREVIATIONS: CNS, central nervous system; MAOI, monoamine oxidase inhibitor; TIA. transient ischemic attack.

may be present; however, almost half will have normal vital signs and exam.[6] Sentinel hemorrhages or warning bleeds occur in 30 to 60 percent of patients presenting with SAH. Subhyaloid (preretinal) hemorrhages may be seen.

- A family history of SAH increases the risk fourfold over the general population.[7]
- Post–lumbar puncture headache is due to persistent cerebrospinal fluid (CSF) leak through the dural puncture and develops 1 to 2 days after the lumbar puncture (LP). It is characterized by intense pain on standing with significant improvement when supine.
- Preeclampsia must be considered in the female patient with headache in the latter half of pregnancy or early postpartum period. Hypertension, proteinuria, and edema are frequent additional findings. Eclampsia increases the risk of intracranial bleed. Dural sinus thrombosis tends to occur in the early postpartum period.
- Meningitis often presents with fever, headache, meningismus, and photophobia. Sinusitis, influenza and other non–central nervous system infections may also present with fever and headache.
- Intraparenchymal hemorrhage produces headache in 55 percent of patients, and neurologic signs and symptoms are found in the vast majority.
- Subdural hematoma may present with headache, altered mental status, or focal neurologic abnormalities. There may be a history of recent or relatively remote trauma (weeks). Those at risk include the elderly, chronic alcoholics, and patients on anticoagulants or with bleeding diathesis.
- Temporal arteritis is a systemic panarteritis, which produces headache in 60 to 90 percent, usually in the temporal region with a tender temporal artery.

TABLE 138-2 High-Risk Findings in the Headache Patient

History	
Headache pattern	Severe, worst headache ever; significant change from prior headache
Headache onset	Sudden maximum severity at onset; new headache in the elderly
Associated symptoms	Syncope, altered mental status, neck pain, fever, seizure, focal neurologic complaints or visual disturbance
Other history	Medications (MAOIs, anticoagulants), cocaine, bleeding diathesis, carbon monoxide exposure, pregnancy, hypertension, HIV, malignancy, recent or remote trauma, ventricular-peritoneal shunt, polycystic renal disease
Family history	Subarachnoid hemorrhage
Physical examination	
Vital signs	Fever, marked hypertension
Head and neck	Papilledema, subhyaloid hemorrhage, absent ocular venous pulsations, corneal edema, neck stiffness, and temporal artery tenderness
Neurologic	Any focal or nonfocal neurologic finding

Table 138-3 Diagnosis of Migraine Headache

For a headache to be classified as a migraine headache, the following must be present: duration of 4-72 h (without treatment) and:

At least 2 of the following:
1. Unilateral position
2. Pulsating quality
3. Moderate or severe intensity (inhibits or prohibits daily activities)
4. Aggravation by walking stairs or similar routine physical activity

And at least one of the following:
1. Nausea, vomiting, or both
2. Photophobia and phonophobia

In addition, to be classified as a migraine with aura, the following must be satisfied:
1. One or more fully reversible aura symptoms indicating brain dysfunction
2. At least one aura symptom develops gradually over more than 4 min or two or more symptoms occur in succession
3. No single aura symptoms lasts more than 60 min
4. Headache follows aura with a free interval of less than 60 min

Almost all patients are over 50 years of age and have a sedimentation rate greater than 50. They may present with visual loss due to ischemic optic neuritis, jaw claudication, or symptoms of polymyalgia rheumatica.[9]

- Temporomandibular disorder (TMD) most often presents with pain localized to the temporomandibular joint (TMJ) and ear but may cause a more diffuse face and head pain. TMJ tenderness and palpable clicking are frequent findings, as is a history of bruxism.
- Trigeminal neuralgia (tic douloureux) produces paroxysms of brief lancinating pain on one side of the face. Trigger points on the cheek or gum are stimulated by light touch or chewing.

DIAGNOSIS AND DIFFERENTIAL

- Table 138-3 lists the diagnostic criteria for a migraine headache.
- Table 138-4 summarizes selected clinical features in the differential diagnosis of headache.
- Computed tomography (CT) without contrast is the test of choice for evaluating suspected SAH; new-generation scanners have a sensitivity greater than 93 percent in the first 24 h from symptom onset.[10] Sensitivity falls after 24 h.
- For the patient with suspected SAH and a normal head CT, a LP is considered necessary to assist in ruling out SAH. Xanthochromia detected by spectrophotometry in the cerebrospinal fluid

Table 138-4 Differential Diagnosis of the Patient with Headache

TYPE OF HEADACHE	HISTORY/PHYSICAL FINDINGS
Migraine headache	Young at onset; lasts longer than 60 min; unilateral, pulsating, throbbing; +/− visual aura; nausea and vomiting; precipitated by foods, drugs, alcohol, exercise or orgasm; + family history
Cluster headache	Onset in 20s; predominantly male; brief episodes or pain (45–60 min); orbital/retroorbital pain; periodic and seasonal (spring/autumn); nasal congestion and conjunctival injection/tearing associated; − family history
Tension-type headache	Onset at any age; dull, nagging, persistent pain; progressively worse throughout day
Subarachnoid headache	Sudden onset, ''worst headache ever,'' loss of consciousness, meningismus, vomiting, occipitonuchal location
Hypertensive headache	Throbbing, occipital
Meningitis	Entire head, fever, meningismus
Mass lesions	
Subdural hematoma	Depressed mental status, variable-quality headache
Epidural hematoma	History of trauma, consciousness wtih headache followed by unconsciousness; fracture across groove of middle meningeal artery
Brain tumor	Pain on awakening or with Valsalva; new headache associated with nausea and/or vomiting
Brain abscess	Findings similar to those of mass lesions, fever
Sinusitis	Stabbing or aching pain, worse by bending or coughing, decreased in supine position
Toxic/metabolic headache	Bicranial; headache remits after removal from offending agent/environment
Postconcussion headache	History of trauma within hours to days; vertigo, nausea, vomiting, mood alterations, concentration difficulty associated
Pseudotumor cerebri	Obese young female; irregular menstrual cycles/amenorrhea; papilledema
Acute glaucoma	Nausea, vomiting, orbital pain, edematous/cloudy cornea, midposition pupil, conjunctival injection, increased intraocular pressure

Table 138-5 Agents Used in the ED Management of Migraine Headache

AGENT	ROUTE	CONSIDERATIONS
Ergotamine	Inhalation, rectal	Contraindicated in coronary artery disease, hypertension, pregnancy
Chlorpromazine	0.1 mg/kg IV	May cause extrapyramidal effects, excellent antiemetic
Prochlorperazine	10 mg IV	May cause extrapyramidal effects, excellent antiemetic
Metoclopramide	10–20 mg IV	May cause extrapyramidal effects, excellent antiemetic
Dihydroergotamine	0.75–1.0 mg IV over 2 min	Contraindicated in coronary artery disease, hypertension, pregnancy
Sumatriptan	6 mg SQ	Contraindicated in coronary artery disease, hypertension, pregnancy
Ketorolac	60 mg IM	Moderately effective only

(CSF) supernatant is almost 100 percent sensitive if performed greater than 12 h after the onset of headache.[7]

- CT scan without contrast is the initial test of choice for the emergent evaluation of the patient with headache in whom a serious secondary cause is suspected.[8] However, CT with contrast or magnetic resonance imaging (MRI) may be required to detect small lesions.
- Suspected cases of meningitis require an LP for CSF evaluation. A head CT scan is not necessary before the LP if the patient displays a normal mental status and neurologic exam and no papilledema.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The medications used for treating migraines are listed in Table 138-5. Sumatriptan and dihydroergotamine (DHE) should not be administered together or in patients with hemiplegic or basilar migraines or cardiovascular disease.
- Cluster headaches frequently respond to high-flow oxygen and also to DHE or sumatriptan.
- Post–lumbar puncture headaches often respond to 1 L of IV crystalloid with 500 mg of caffeine given over 2 h. If necessary, an epidural blood patch using autologous blood will stop the leak.[11]
- For cases of suspected meningitis, antibiotics should be initiated after blood cultures and before the LP if there is any delay in obtaining the LP.[8]
- If temporal arteritis is suspected, then prednisone 60 mg PO daily should be prescribed and prompt outpatient ophthalmology follow-up arranged for temporal artery biopsy.[9]
- Trigeminal neuralgia frequently improves with carbamazepine.
- In those cases where intracranial pathology is identified or suspected, consultation and admission by the appropriate service (neurology, neurosurgery, internal medicine) is indicated.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 219, ''Headache and Facial Pain,'' by Michael Schull.

139 STROKE SYNDROMES

Stefanie R. Seaman

EPIDEMIOLOGY

- Stroke is the third leading cause of death and the leading cause of disability in the United States. The incidence of stroke doubles each decade after age 55.[1]

PATHOPHYSIOLOGY

- Stroke is the result of any process that causes disruption of blood flow to a particular part of the brain.
- There are two main mechanisms of stroke: (*1*) blood vessel occlusion leading to neuronal ischemia and death (80 to 85 percent of all strokes) and (*2*) blood vessel rupture leading to hemorrhage, direct cell trauma, mass effect, elevated intracranial pressure, and release of biochemical toxins (15 to 20 percent of all strokes).
- Ischemic strokes are most often caused by large-vessel thrombosis, although embolism or systemic hypoperfusion can also cause them. Causes of thrombosis include atherosclerotic disease, vasculitis, dissection, polycythemia, hypercoagulable states, and infectious diseases (such as HIV, syphilis, tuberculosis, and trichinosis).
- In ischemic strokes, injury occurs from ischemia, which deprives neurons of oxygen and substrate.
- With cessation of blood flow, cells die within minutes. Irreversible injury usually occurs at the center of the ischemic region while the periphery (the penumbra) has a degree of reversible injury. The damage also depends on the degree and duration of occlusion.
- Similar short-lived episodes, transient ischemic attacks (TIAs), often precede thrombotic strokes.
- Embolic strokes account for 20 percent of all strokes in the United States. Embolic strokes occur when intraluminal material travels and occludes a distal vessel.
- Common sources of emboli in embolic strokes are cardiac valve vegetations, mural thrombus (from atrial fibrillation, myocardial infarction, or dysrhythmias), paradoxical emboli (atrial septal defect, ventricular septal defect), and cardiac tumors (myxomas).
- Hemorrhagic strokes have a 30-day mortality of 30 to 50 percent, occur in a younger patient population than ischemic strokes, and are divided into intracerebral (ICH) and subarachnoid hemorrhages (SAH). Risk factors for an ICH include hypertension, older age, race (higher incidence in blacks and Asians), tobacco and alcohol abuse, and prior stroke.
- Bleeding diathesis, vascular malformations, and cocaine use can cause ICH.
- Most SAHs are due to rupture of a berry aneurysm and to arteriovenous malformations. In SAH, blood leaks from a cerebral vessel into the subarachnoid space, and this leak occurs at a higher systemic arterial pressure than that of an ICH, which occurs slowly at a lower pressure.

CLINICAL FEATURES

- History should include time of onset, concurrent symptoms, fluctuation of symptoms, thorough past medical history, family history, and recent trauma. The general physical examination should include a complete evaluation of the skin, fundi, heart, and lungs as well as listening for carotid and other vascular bruits.
- The neurologic examination recommended by the National Institutes of Health (NIH) is broken into six major areas: (*1*) level of consciousness, (*2*) visual assessment, (*3*) motor function, (*4*) cerebellar function, (*5*) sensation and neglect, and (*6*) cranial nerves.
- Integration of information from the history and physical examination allows the physician to determine the area of brain involvement. Specific stroke syndromes are listed in Table 139-1.
- Two special classes of patients are at risk for stroke. Over 10 percent of patients with sickle cell disease will present with stroke by age 20. Peripartum and postpartum (up to 6 weeks after birth), women have an increased incidence of both ischemic and hemorrhagic stroke.

TABLE 139-1 Stroke Syndromes

Ischemic stroke syndromes
Transient ischemic attack (TIA): resolves within 24 h (most within 30 min), 5–6% risk of stroke per year
Dominant hemispheric infarct: contralateral weakness/numbness, contralateral visual field cut, gaze preference, dysarthria, aphasia
Nondominant hemispheric infarct: contralateral weakness/numbness, visual field cut, constructional apraxia, dysarthria
Anterior cerebral artery infarct: contralateral weakness/numbness (leg more than arm), dyspraxia, speech perseveration, slow responses
Middle cerebral artery infarct: most common area involved; contralateral weakness/numbness (arm/face more than leg)
Posterior cerebral artery infarct: often goes unrecognized by patient, minimal motor involvement, light-touch and pinprick sensation significantly affected
Vertebrobasilar syndrome: dizziness, vertigo, diplopia, dysphagia, ataxia, cranial nerve palsies, bilateral limb weakness, crossed neurologic deficits
Basilar artery occlusion: quadriplegia, coma, locked-in syndrome
Cerebellar infarct: ''drop attack'' associated with vertigo, headache, nausea, vomiting, and/or neck pain, cranial nerve abnormalities
Lacunar infarct: pure motor or sensory deficits
Arterial dissection: often associated with severe trauma, headache, and neck pain hours to days prior to onset of neurologic symptoms
Hemorrhagic syndromes
Intracerebral hemorrhage: similar to cerebral infarction with lethargy, headache, nausea, vomiting, significant hypertension
Cerebellar hemorrhage: dizziness, vomiting, truncal ataxia, inability to walk, rapid progression to coma, herniation, and death
Subarachnoid hemorrhage: severe headache, vomiting, decreased level of consciousness

TABLE 139-2 Differential Diagnosis of Acute Stroke

Hypoglycemia
Postictal paralysis (Todd's paralysis)
Bell's palsy
Hypertensive encephalopathy
Epidural/subdural hematoma
Brain tumor/abscess
Complicated migraine
Encephalitis
Diabetic ketoacidosis
Hyperosmotic coma
Meningoencephalitis
Wernicke encephalopathy
Multiple sclerosis
Ménière's disease
Drug toxicity (lithium, phenytoin, carbamazepine)

DIAGNOSIS AND DIFFERENTIAL

- An emergent noncontrast computed tomography (CT) scan is necessary to distinguish ischemic from hemorrhagic stroke. CT may detect all regions of hemorrhage greater than 1 cm and up to 95 percent of all SAHs.
- Most ischemic strokes will not be visualized on CT up to 6 to 12 h, depending on the size.
- An electrocardiogram (ECG) will provide clues for any concurrent signs of myocardial ischemia. Atrial fibrillation and acute myocardial infarction are the cause of up to 60 percent of all cardioembolic strokes. Stroke occurrence is 2 to 5 percent within the first 4 weeks following acute myocardial infarction.
- Magnetic resonance imaging (MRI) can visualize ischemic infarcts earlier than CT and is more effective at visualizing posterior circulation strokes. MRI is less accurate at distinguishing ischemia from hemorrhage.
- Table 139-2 lists the differential diagnosis of patients with stroke syndromes.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients should receive supplemental oxygen, be placed on a cardiac monitor, and have an IV line established. Diagnostic tests that should be obtained immediately include a blood glucose determination, noncontrast head CT, and ECG.
- Other tests that may be helpful include laboratory tests (coagulation studies, toxic screen, cardiac enzymes), echocardiogram, and carotid duplex scanning. Emergency MRI should be considered if a dural sinus thrombosis or a lesion of the posterior circulation is considered.
- Patients with embolic stroke and minor deficits should be anticoagulated with heparin, as should patients with TIAs if they have known high-grade stenosis, a cardioembolic source, increasing frequency of TIAs (crescendo TIAs), or TIAs despite antiplatelet therapy. Heparin anticoagulation should be withheld for 3 to 4 days in patients with large cardioembolic strokes.
- Treatment for stable thrombotic stroke is supportive. Anticoagulation is not indicated. However, aspirin in a dose of 300 mg/day is beneficial.
- The NINDS trial showed that patients who received rt-PA for acute ischemic stroke within 3 h of symptom onset had a significantly lower morbidity. This study resulted in the FDA approving rt-PA for this indication in selected individuals (Table 139-3). Total dose of rt-PA is 0.9 mg/kg, with a maximum dose of 90 mg. Ten percent of the dose should be administered as an initial bolus, followed by an infusion of the remainder over 60 min. rt-PA is not indicated if the exact time of

TABLE 139-3 Criteria for Use of rt-PA in Acute Ischemic Stroke and Management of Patients Following Use of rt-PA

INCLUSION	EXCLUSION
Age 18 or over	Minor stroke syndromes
Clinical diagnosis of ischemic stroke	Rapidly improving neurologic signs
Well-established time of onset <3 h	Prior intracranial hemorrhage Blood glucose <50 or >400 Seizure at onset of stroke GI or GU bleeding within preceding 21 days Recent myocardial infarction Major surgery within 14 days Pretreatment SBP >185 or DBP >110 mmHg Previous stroke or head injury within 90 days Current use of oral anticoagulants Use of heparin within preceding 48 h Platelet count <100,000 Suspected aortic or vascular dissection or LP

ABBREVIATIONS: GI = gastrointestinal; GU = genitourinary; SBP = systolic blood pressure; DBP = diastolic blood pressure; LP = lumbar puncture.
Monitor arterial blood pressure during the first 24 h after starting treatment, every 15 min for 2 h after starting infusion, then every 30 min for 6 h, and then every 60 min for 24 h total.
If SBP is 180–230 mmHg or DBP is 105–120 mmHg for two or more readings 5–10 min apart:
- Give IV labetalol 10 mg over 1–2 min. The dose may be repeated or doubled every 10–20 min up to a total dose of 150 mg.
- Monitor blood pressure every 15 min during labetalol treatment and observe for hypotension.

If SBP >230 mmHg or if DBP is 121–140 mmHg for two or more readings 5–10 min apart:
- Give IV labetalol 10 mg over 1–2 min. The dose may be repeated or doubled every 10–20 min up to a total dose of 150 mg.
- Monitor blood pressure every 15 min during labetalol treatment and observe for hypotension.
- If no satisfactory response, infuse sodium nitroprusside (0.5–1.0 μg/kg/min); continuous arterial pressure monitoring advised.

If DBP >140 mmHg for two or more readings 5–10 min apart:
- Infuse sodium nitroprusside (0.5–1.0 μg/kg/min); continuous arterial pressure monitoring advised.

onset of symptoms cannot be ascertained. No aspirin or heparin therapy is given with the first 24 h of treatment. Admission to an intensive care unit (ICU) setting is recommended.

- Glucose-containing solutions are to be avoided because of increased neuronal damage in hyperglycemia. Only severe hypertension (SBP >220 or DBP >120 mmHg) should be treated. Hypotension should be treated with fluid therapy and vasopressors if needed.
- In patients with sickle cell anemia and ischemic stroke, immediate simple or exchange transfusion should be initiated to reduce Hb S concentration to below 30%. Use of hyperosmotic contrast solutions should be delayed until the Hb S concentration is below 30%.
- Early neurosurgical consultation is needed for patients with cerebellar infarction or hemorrhage.
- Patients with intracerebral hemorrhage and hypertension should have their blood pressure lowered only if their SBP is above 220 mmHg or their DBP is above 120 mmHg. Labetalol or nitroprusside are the agents of choice. Therapy to lower blood pressure should extend over 12 to 24 h. The desired endpoint is the prehemorrhage level of blood pressure, if known.
- To prevent rebleeding, patients with SAH should have their blood pressure maintained at prehemorrhage levels (if known) or the mean arterial pressure should be maintained at 110 mmHg. Nimodipine should be given to prevent vasospasm related to the SAH.
- Patients with new-onset strokes should be admitted to the hospital, as should patients with new-onset TIAs unless high-grade stenosis of the carotid arteries can be ruled out.[2–8]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 220, ''Stroke, Transient Ischemic Attack, and Other Central Focal Conditions,'' by Phillip A. Scott and William G. Barsan.

140 ALTERED MENTAL STATUS AND COMA

Philip B. Sharpless

A person's mental state is, in part, a function of the degree of wakefulness or arousal (awareness of one's environment and internal thoughts) and cognitive content, which includes verbal reasoning, calculation, abstraction, and perception. An altered mental state develops from processes affecting either or both of these components and includes delirium, dementia, and psychosis. Distinctions between these three conditions are found in Table 140-1.[1]

DELIRIUM

- Delirium, or acute confusional state, is an acute, reversible, and generalized disruption in behavior and cognition that is due to toxic or metabolic brain dysfunction.

EPIDEMIOLOGY

- At the time of admission, 11 to 24 percent of elderly patients have delirium.

PATHOPHYSIOLOGY

- Delirium is a generalized disorder of neuronal or neurotransmitter function, with disturbances in both arousal and cognition.
- Predisposing factors include old age, preexisting dementia, or other chronic central nervous system (CNS) diseases.

CLINICAL FEATURES

- Delirium is an acute state of confusion that develops and persists over hours to weeks. A reduced level of consciousness is manifest by an inability to sustain or shift attention. Abnormalities in cognition include disorientation and difficulties with learning and memory. Fluctuation in these difficulties over time is the hallmark.[1]
- Perceptual changes—including hallucinations (visual more often than auditory), illusions, and delusions—occur in up to 40 percent of cases.[1]
- Emotional disturbances and lability, as well as disruption of sleep-wake cycles with nocturnal agitation (''sundowning''), are frequent.[1]
- The variants of delirium are the hypoalert-hypoactive type, hyperalert-hyperactive type, and mixed type, which fluctuates between the former two.[1]

DIAGNOSIS AND DIFFERENTIAL

- The causes of delirium (Table 140-2) may be considered in four groups: primary cerebral disease, systemic disease with secondary central nervous system (CNS) effects, medications and toxins, and drug withdrawal.[1]
- History from family and friends—regarding the patient's baseline mental status, rapidity of change, and fluctuation in course—is the key to diagnosis.[2]
- History and physical examination aim at determining an underlying cause and should include a careful medication history, search for infection, and signs of cardiopulmonary, hepatic, or renal dysfunction, endocrinopathy, or focal neurologic disease.
- Baseline studies should include complete blood

TABLE 140-1 Features of Delirium, Dementia, and Psychiatric Psychosis

Characteristic	Delirium	Dementia	Psychiatric psychoses
Onset	Sudden	Insidious	Sudden
Course over 24 h	Fluctuating	Stable	Stable
Consciousness	Reduced or clouded	Alert	Alert
Attention	Disordered	Normal	May be disordered
Cognition	Disordered	Impaired	May be impaired
Orientation	Impaired	Often impaired	May be impaired
Hallucinations	Usually visual	Often absent	Usually auditory
Delusions	Transient, poorly organized	Usually absent	Sustained
Movements	Asterixis, tremor may be present	Often absent, it presents usually unrelated	Absent

SOURCE: Modified from Lipowski,[1] with permission.

cell count, electrolytes, calcium, liver enzymes, urinalysis, pulse oxymetry, electrocardiogram (ECG), and chest radiograph. Other studies that may be indicated are TSH, computed tomography (CT) of the head, cerebrospinal fluid analysis, and blood cultures.

TABLE 140-2 Causes of Delirium

Primary cerebral disease
- Subdural or other space-occupying lesion
- Stroke, transient ischemic attack
- Cerebral arteritis
- Nonconvulsive status epilepticus
- Meningitis, encephalitis
- Trauma

Systemic disease
- Infection: cystitis, pneumonia, etc.
- Acid-base disturbance
- Electrolyte disturbance
- Kidney or hepatic failure
- Endocrinopathy: thyroid, parathyroid, adrenal
- Cardiopulmonary disease
- Hypoxia
- Hypoglycemia, hyperglycemia
- Dehydration
- Severe anemia
- Hypertensive emergency
- Environmental—hypothermia, hyperthermia, heat stroke
- Vitamin deficiency

Medications/drugs (partial list)
- Alcohol
- Anticholinergics
- Antihistamines, cimetidine
- Digoxin, other cardiovascular drugs
- Narcotics
- Salicylism
- Psychotropic medicines: antidepressants, neuroleptics, sedatives, lithium

Drug withdrawal
- Alcohol
- Benzodiazepines
- Barbiturates

- Differential diagnosis includes (1) dementia, which is characterized by a more prolonged, nonfluctuating deterioration in cognition without a reduction in alertness, and (2) depression, which may resemble hypoalert-hypoactive delirium but is not associated with clouding of consciousness.[2]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The underlying cause should be identified and treated.
- Pharmacologic sedation may be used judiciously to control severe agitation; haloperidol and lorazepam are two reasonable choices. Lorazepam given parenterally, often in larger amounts, would be the treatment choice for delirium secondary to alcohol or sedative withdrawal.
- Admission for further evaluation and treatment is indicated unless a readily reversible cause is identified.[3]

DEMENTIA

- Dementia is a clinical syndrome characterized by gradual loss of cognitive function initially without change in wakefulness.

EPIDEMIOLOGY

- By age 60, some 1 percent of the U.S. population have dementia; by age 85, up to 50 percent may be affected.[4]

PATHOPHYSIOLOGY

- Up to 70 percent of dementia cases are due to Alzheimer's disease, a neurodegenerative disorder of unknown etiology.
- Vascular (multi-infarct) dementia accounts for 10 to 20 percent of cases.

CLINICAL FEATURES

- Patients may present with a history of gradual, progressive impairment of short-term memory with initial relative preservation of remote memory.
- In later stages, patients demonstrate decreased performance in social situations, lose direction, and ultimately lose all orientation and capacity for self-care.
- Affective symptoms, including depression and anxiety, are frequently present.
- Vascular dementia is often associated with a history of strokes and focal neurologic deficits, asymmetric reflexes, and extensor plantar responses.

DIAGNOSIS AND DIFFERENTIAL

- Table 140-3 presents the mnemonic for potentially treatable causes of dementia.
- Increased motor tone, cogwheel rigidity, and resting tremor may suggest Parkinson's disease.
- Normal-pressure hydrocephalus (NPH) should be suspected from a history of gait disturbance followed by incontinence and cognitive decline.[4]

TABLE 140-3 Mnemonic for Potentially Treatable/ Reversible Causes of Dementia

D	Drugs (anticholinergic, narcotic, sedatives, phenothiazines, others)
E	Electrolytes, eye or ear problems (partial blindness or deafness)
M	Metabolic disturbances (thyroid disease, hepatic failure, others)
E	Emotional (depression, schizophrenia)
N	Nutritional (B_{12}, folate deficiency, Wernicke-Korsakoff), Normal pressure hydrocephalus
T	Trauma, tumor (includes subdural hematoma)
I	Inflammation (SLE, others) Infections (chronic meningitis, syphilis, Lyme, HIV)
A	Alcohol*

* Chronic effects of alcohol are not easily reversible; however, with abstinence and proper nutrition, even severely affected (ex-) alcoholics may show improvement.
SOURCE: Modified from Tueth,[5] with permission.

- Practice guidelines by the American Academy of Neurology recommend that the diagnostic workup include a complete blood cell count, electrolytes, calcium, liver function, TSH, B_{12} level, syphilis serology, and neuroimaging with CT or magnetic resonance imaging (MRI). Other tests to consider include sedimentation rate, chest radiograph, folate level, HIV serology, and cerebrospinal fluid analysis.[6]

EMERGENCY CARE AND DISPOSITION

- Emergency care is directed at identifying the potentially treatable causes of dementia, such as subdural hematoma, NPH, hypothyroidism, neurosyphilis, etc.
- In the absence of a treatable cause, the disposition and management of the patient should be coordinated with the primary care physician with consideration of the patient's social support system.

COMA

- Coma is a state of unconsciousness in which the patient is unarousable and the eyes are closed.

PATHOPHYSIOLOGY

- Coma is produced from dysfunction of the brainstem reticular activating system (RAS) or diffuse dysfunction of both cerebral hemispheres.
- A general rule is that unless accompanied by mass effect, a unilateral cerebrocortical lesion does not cause coma.
- Supratentorial mass lesions resulting in coma may cause brainstem compression from herniation of tissue through the tentorium.
- The uncal herniation syndrome is due to the herniation of medial temporal lobe tissue through the tentorial notch. In the usual scenario, stretching of the ipsilateral oculomotor nerve occurs and causes, initially, a dilated, sluggishly reactive pupil on the same side as the CNS lesion. This may be seen prior to unresponsiveness in the patient. Further compression creates an oculomotor nerve palsy and a fixed, dilated pupil. Additionally, compression by the herniating tissue on the ipsilateral cerebral peduncle will cause contralateral weakness, while midbrain compression deepens the coma. Alternatively, shift of the brainstem across the midline may compress the contralateral cere-

bral peduncle against the tentorial edge and cause weakness ipsilateral to the CNS lesion.[7]
- The signs of uncal herniation are not reliable for localizing the CNS lesion; the contralateral oculomotor nerve is compromised first in up to 15 percent.[7]

CLINICAL FEATURES

- Coma resulting from diffuse CNS dysfunction is generally associated with symmetric, reactive pupils unless the toxin affects pupillary function. Hypoxia, anticholinergic-, and glutethimide-induced coma may present with dilated, nonreactive pupils.[8]
- Conversely, if the above insults and preexisting pupillary disease or cycloplegic use are excluded, the finding of nonreactive pupils suggests a structural lesion.[8]
- Narcotic overdose and pontine hemorrhage both cause pinpoint pupils that are minimally reactive.
- The eye movements of patients with mild metabolic coma are often roving and settle into a forward gaze as the coma deepens.
- A disconjugate gaze or conjugate deviation from midline suggests a structural lesion. Conjugate deviation occurs toward the side of a destructive cerebral hemispheric lesion and away from a brainstem (pontine) lesion.
- Tremor, asterixis, and multifocal myoclonus are frequently seen in metabolic encephalopathy and less commonly with structural brain lesions.[8]
- Focal weakness and asymmetric tone or reflexes strongly suggests a structural lesion but may on occasion be seen in a metabolic brain disease like hypoglycemia.[8]
- Uncal herniation usually presents with an ipsilateral dilated pupil followed by oculomotor palsy and contralateral weakness; however, ipsilateral weakness or contralateral ocular findings may occur. Progression of herniation results in bilateral fixed pupils and decerebrate rigidity.
- Expanding infratentorial lesions may present with rapid development of coma, decerebrate posturing, and loss of pupillary response.
- Increased intracranial pressure with or without lateralizing findings may cause reflex hypertension and bradycardia (Cushing reflex).

DIAGNOSIS AND DIFFERENTIAL

- The causes of coma (Table 140-4) are separated into two categories: (1) diffuse or metabolic brain dysfunction and (2) structural CNS lesions (both supratentorial and subtentorial subtypes).
- Abrupt onset suggests CNS bleed, brainstem stroke, seizure, or cardiopulmonary cause of anoxia.
- Gradual onset favors a metabolic cause or slowly expanding mass lesion (tumor, subdural hematoma).
- ''Locked-in'' syndrome caused by devastating brainstem damage outside the pontine-midbrain RAS can simulate a coma. The patient loses all ability to communicate and all voluntary movement except vertical eye movements yet maintains consciousness and understanding.[9]
- Psychogenic coma may be suspected from the historical circumstances, patient resistance to eye opening and abrupt closure on release, normal nystagmus with caloric testing, and preserved optokinetic nystagmus.

TABLE 140-4 Differential Diagnosis of Coma

COMA FROM CAUSES AFFECTING THE BRAIN DIFFUSELY
Encephalopathies
Hypoxic
Metabolic
Hypoglycemia
Diabetic ketoacidosis
Hyperosmolar state
Other electrolyte abnormalities
Organ system failure
Hepatic encephalopathy
Uremia/renal failure
Endocrine
Hypertensive encephalopathy
Toxins and drug reactions
CNS sedatives
Alcohol
Carbon monoxide, other inhalants
Neuroleptic malignant syndrome
Environmental causes—hypothermia, hyperthermia
Deficiency state: Wernicke's encephalopathy
COMA FROM PRIMARY CNS DISEASE OR TRAUMA
Direct CNS trauma
Vascular disease
Intraparenchymal hemorrhage
Hemispheric
Basal ganglia
Brainstem
Cerebellar
Infarction
Hemispheric
Brainstem
Subarachnoid hemorrhage
CNS infections
Neoplasms
Seizures
Nonconvulsive status epilepticus
Postictal state

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The initial treatment and evaluation are outlined in Table 140-5. Care involves stabilization of the airway, breathing, and circulatory status while searching for a treatable etiology or a readily reversible cause like hypoglycemia, hyperglycemia, hypoxia, or hypotension.
- Thiamine need not precede administration of dextrose in the acute setting of hypoglycemia.
- Unless a metabolic cause is clearly identified, a CT scan of the head is recommended, since exceptions to the general rules of differentiating structural from diffuse metabolic brain disease are frequent.
- Dexamethasone IV reduces edema associated with tumors; use and dosage should be discussed with the appropriate consultant.
- Treatment of clinically suspected or radiologically confirmed herniation is temporizing prior to definitive neurosurgical intervention. Treatment modalities such as mannitol (0.5 to 1.0 g/kg IV) or hyperventilation (the use of which has become more controversial) should be discussed with the neurosurgical consultant.[10]

TABLE 140-5 Management Steps for the Comatose Patient

I. History-utilize all resources

II. Initial assessment
  A. Primary survey
    1. Establish unresponsiveness/protect cervical spine
    2. *A*-manage airway, *B*-assess breathing, *C*-circulation
  B. Resuscitation/life-saving intervention
    1. Oxygen supplementation
    2. Establish intravenous access/draw initial blood sample
    3. Cardiac monitor
    4. Pulse oximetry monitor
    5. Thiamine: 100 mg IV (adults only)
    6. Glucose: 50 mL of 50% dextrose solution or glucose test
    7. Naloxone: administer 2 mg IV or SQ (or more)
  C. Secondary assessment
    1. Complete vital signs and general physical examination
    2. Neurologic examination
      a. Respiratory pattern
      b. Observation of posture and movements
      c. Verbal and motor response to stimulation
      d. Cranial nerve examination
      e. Reflexes
      f. Assignment to rating system/serial examinations
    3. Other monitoring
      a. Arterial blood gas analysis
      b. ECG monitor

III. Laboratory evaluation
  A. Routine labs: electrolytes, CBC, ABG
  B. Additional labs in selected patients
    1. COHgb, toxicologic screen, hepatic, CSF, thyroid, cortisol

IV. Radiologic evaluation tailored to patient. C-spine, CXR, cranial CT

V. Definitive care
  A. Supportive, monitoring
  B. Treatment
    1. Specific treatment if possible in emergency department
    2. Nonspecific treatment in selected cases
      a. Osmotic agents or loop diuretics
      b. Steroids
      c. Hyperventilation, head position
  C. Appropriate consultation

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 221, "Altered Mental Status and Coma," by J. Stephen Huff.

141 GAIT DISTURBANCES

Sandra L. Najarian

PATHOPHYSIOLOGY

- Gait disturbances are symptoms of underlying disease and not disease entities onto themselves.
- Ataxia is the failure to produce smooth, intentional movements.
- Systemic conditions, such as drug intoxication and metabolic disorders, and disorders of the central and peripheral nervous system are common etiologies of ataxia (Table 141-1).
- Disorders of the cerebellum cause motor ataxia. Some supratentorial lesions (internal capsule, thalamic nuclei, and frontal lobe) have been known to cause ataxia as well.[1,2]
- Disorders that affect proprioception and position sense cause sensory ataxia.
- Ataxia in children can be seen following immunizations, viral illnesses, or varicella, though it has been reported in the preeruptive phase of varicella[3] (Table 141-2).

TABLE 141-1 Common Etiologies of Acute Ataxia and Gait Disturbances

I. Systemic conditions
  A. Intoxications with diminished alertness
    1. Ethanol
    2. Sedative-hypnotics
  B. Intoxications with relatively preserved alertness (diminished alertness at higher levels)
    1. Phenytoin
    2. Carbamazepine
    3. Valproic acid
    4. Heavy metals—lead, organic mercurials
  C. Other metabolic disorders
    1. Hyponatremia
    2. Inborn errors of metabolism

II. Disorders predominantly of the nervous system
  A. Conditions affecting predominantly one region of the central nervous system
    1. Cerebellum
      a. Hemorrhage
      b. Infarction
      c. Degenerative changes
      d. Abscess
    2. Cortex
      a. Frontal tumor, hemorrhage, or trauma
      b. Hydrocephalus
    3. Subcortical
      a. Thalamic infarction or hemorrhage
      b. Parkinson's disease
    4. Spinal cord
      a. Cervical spondylosis
      b. Posterior column disorders
  B. Conditions affecting predominantly the peripheral nervous system
    1. Peripheral neuropathy
    2. Vestibulopathy

TABLE 141-2 Causes of Acute Ataxia in Children Roughly in Order of Frequency

CAUSE	EXAMPLE
Drug intoxication	Ethanol Isopropyl alcohol Phenytoin Carbamazepine Sedatives Lead, mercury Others
Acute viral infection, postinfectious inflammatory causes, and postimmunization	Varicella Coxsackievirus A and B Mycoplasma Echovirus
Neoplasm	Neuroblastoma Other central nervous system tumors
Trauma	Subdural or epidural posterior fossa hematoma
Congenital or hereditary	Pyruvate decarboxylase deficiency Friedreich's ataxia Hartnup's disease Others
Hydrocephalus	
Cerebellar abscess	
Labyrinthitis/vestibular neuronitis	
Meningoencephalitis	
Idiopathic	

SOURCE: Modified from Belcher RS: Preeruptive cerebellar ataxia in varicella. *Ann Emerg Med* 27:511, 1996; and Chutorian AM, Pavlakis SG: Acute ataxia, in Pellock JM, Myer EC (eds): *Neurologic Emergencies in Infancy and Childhood.* Boston, Butterworth-Heinemann, 1993, pp 208–219, with permission.

CLINICAL FEATURES

- A thorough neurologic evaluation, including cerebellar function, gait testing, and Romberg testing, is essential in evaluating ataxia.
- A positive Romberg test is suggestive of a sensory ataxia.
- In children presenting with ataxia, intoxications, ingestions, weakness, and musculoskeletal disorders must be ruled out.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is made based on history and physical examination.
- Neuroimaging is necessary if a mass lesion is suspected. Laboratory studies may be indicated if

anticonvulsant or heavy metal toxicity is suspected.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Admission is required for patients presenting with an acute gait disturbance over hours to days and for patients who are unable to care for themselves and may be unsafe at home.
- Patients presenting with symptoms over weeks to months may be referred for outpatient follow-up if they have a safe home environment.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 222, ''Ataxia and Gait Disturbances,'' by J. Stephen Huff.

142 VERTIGO AND DIZZINESS

Philip B. Sharpless

Dizziness is a common but nonspecific complaint; it may involve vertigo, presyncopal light-headedness, disequilibrium, or a nonspecific impairment of perceptual or mental clarity. Vertigo is the illusion of movement and a symptom of vestibular dysfunction; it may have a peripheral or central nervous system (CNS) cause.

PATHOPHYSIOLOGY

- Equilibrium and spatial orientation result from the processing within the CNS of three primary sensory inputs: proprioception, vision, and vestibular.
- The vestibular system consists of the otolithic organs (saccule and utricle) and three semicircular canals filled with endolymph. Maculae in the saccule and utricle contain embedded crystals (otoconia) and detect linear acceleration and head position. The cristae within the semicircular canals detect angular acceleration.
- Sudden, unilateral disturbance of the tonic bilateral vestibular input from either a destructive lesion (e.g., viral labyrinthitis) or a stimulatory lesion [e.g., benign paroxysmal peripheral vertigo (BPPV)] results in vertigo and nystagmus.
- A slowly evolving process like an acoustic neuroma may not cause vertigo because of CNS compensation.
- Nystagmus is the rhythmic movement of the eyes, which usually comprises fast and slow components. Nystagmus is described by the axis of movement (horizontal, vertical, rotary) and direction identified with the direction of the fast component.
- The slow phase of nystagmus points to the injured vestibular system.
- Nystagmus may change direction with eye or head position. This direction-hanging nystagmus suggests a central process or ingestion of alcohol or various anticonvulsants (phenytoin, carbamazepine, and phenobarbital). Peripheral lesions other than BPPV show unidirectional nystagmus.
- Bilateral vestibular damage produces oscillopsia and instability rather than vertigo.
- The generally accepted explanation for BPPV is the canalithiasis mechanism: free-floating debris (otoconia from the utricle) causes abnormal stimulation of the posterior semicircular canal with positional head changes.

CLINICAL FEATURES

- Vertigo is divided into peripheral and central causes. Peripheral causes are not frequently life-threatening, contrary to some central lesions.
- Peripheral vertigo implies dysfunction of the vestibular apparatus or eighth cranial nerve. Symptom onset is often acute and intense, with nausea and vomiting. Hearing loss and tinnitus may occur, but other neurologic signs are generally not expected.
- Peripheral vestibular nystagmus is unidirectional and horizontal (often with a torsional component).[1] The exception is BPPV, for which the nystagmus is torsional (often with a visible vertical component) and provoked when the patient is

placed in the supine, head-hanging position of the Dix-Hallpike maneuver.[2] The nystagmus reverses torsional direction on rapidly sitting up.
- Peripheral nystagmus and vertigo improve with visual fixation.
- Central vertigo involves lesions in the brainstem or cerebellum. Onset is often gradual, with vertigo and nausea less than expected for the observed nystagmus. Other neurologic signs and symptoms are usually present. However, sudden severe vertigo with vomiting is observed in cerebellar hemorrhage and strokes and may simulate peripheral vestibular neuritis.[1]
- Nystagmus associated with central vertigo may simulate peripheral nystagmus but is frequently purely vertical, horizontal, or rotary and is often direction-changing with change of gaze or head position.
- Central nystagmus does not improve with visual fixation.

DIAGNOSIS AND DIFFERENTIAL

- Most vertigo has a peripheral cause, and BPPV is one of the most common.[3] Positional changes like rolling over in bed provoke attacks that are brief, episodic, and without tinnitus or hearing loss. The Dix-Hallpike maneuver establishes the diagnosis when the vertigo and nystagmus show latency of onset (1 to 5 s), resolution in 5 to 40 s, and less intensity on repeat positioning.[4,5] Central causes of positional vertigo rarely show these features.[2]
- Ménière's disease is presumably caused by endolymphatic hydrops and episodic rupture of the membranous labyrinth. The disease is characterized by episodes of sudden vertigo, tinnitus, aural fullness, and sensorineural hearing loss typically lasting several hours but no more than 24 h.
- Vestibular neuritis is characterized by sudden, severe vertigo and vegetative symptoms without hearing loss. Recovery occurs over several days. Labyrinthitis includes sensorineural hearing loss. Presumably, both disorders are usually due to viral infections, but bacterial labyrinthitis may occur with otitis media, meningitis, or mastoiditis.
- Perilymphatic fistula at the round or oval window, caused by blunt or barotrauma, results in sudden vertigo and sensorineural hearing loss. Insufflation during otoscopy worsens symptoms (Hennebert sign).
- Tumors of the eighth cranial nerve and cerebellopontine angle most frequently present with hearing loss and unsteadiness. Vertigo, ipsilateral facial weakness, and cerebellar signs may also be seen.
- Herpes zoster oticus (Ramsay Hunt's syndrome) is associated with deafness, vertigo, facial nerve palsy, and grouped vesicles on the external auditory canal.
- Closed cranial trauma may produce deafness and vertigo by injury to the labyrinth or vestibular nerve due to fracture of the temporal bone. Displacement of otoconia may precipitate attacks of BPPV.
- Aminoglycoside antibiotics, loop diuretics, cisplatin, and vinblastine may cause irreversible hearing loss and vestibular dysfunction. Ataxia and oscillopsia are more common than vertigo.
- Cerebellar infarction or hemorrhage are potentially devastating causes of central vertigo. Vertigo, nausea, and vomiting may be sudden and severe or mild.[1] Truncal ataxia and abnormal gait on Romberg testing are frequently found. The nystagmus may change direction with change in direction of the gaze.
- Lateral medullary infarction (Wallenberg's syndrome) causes vertigo, ipsilateral facial numbness, dysphagia, dysarthria, diplopia, Horner's syndrome, and crossed sensory loss in the extremities.
- Vertebrobasilar insufficiency (VBI), which may cause brainstem transient ischemic attacks, can produce vertigo lasting minutes to hours. Other signs of posterior circulation ischemia are usually present. Head turning, by partially obstructing the ipsilateral vertebral artery, may provoke VBI if the opposite vertebral artery is stenotic.
- Multiple sclerosis (MS) frequently causes vertigo. Internuclear ophthalmoplegia consisting of defective adduction of one eye and nystagmus of the other abducting eye is a classic finding of MS.
- The aura of basilar migraines may include vertigo, visual loss, tinnitus, hearing loss, diplopia, dysarthria, ataxia, and other manifestations of VBI.[6] The aura develops over 5 to 20 min and may or may not be followed by the headache.
- Other causes of vertigo include brainstem, cerebellar, and fourth ventricular tumors, otosclerosis, Paget's disease, syphilis, and temporal arteritis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients with peripheral vertigo require symptomatic treatment with anticholinergics, antihistamines, and antiemetics (Table 142-1).
- Patients with BPPV often benefit from the canalith repositioning maneuver (Epley's maneu-

TABLE 142-1 Pharmacotherapy of Acute Peripheral Vertigo

Anticholinergics	Scopolamine	0.5 mg transdermal patch q 3–4 days (behind ear)
Antihistamines	Dimenhydrinate	50–100 mg IM or PO q4h
	Diphenhydramine	25–50 mg IM or PO q6h
	Cyclizine	50 mg PO q4h (not to exceed 200 mg/24h)
	Meclizine	25 mg PO q8–12h
Antiemetics	Hydroxyzine	25–50 mg q6h
	Promethazine	25–50 mg q6–8h
Benzodiazepines	Diazepam	2 mg PO q8–12h
	Clonazepam	0.5 mg q12h
Calcium antagonists	Cinnarizine	15 mg PO q8h
	Flunarizine	10 mg PO qd

ver),[7] which returns the otoconia to the utricle. This maneuver should not be performed on patients with cervical spondylosis.
- All patients with peripheral vertigo require primary care follow-up or ear-nose-throat (ENT) referral. Immediate ENT consultation is recommended for hearing loss and suspected bacterial labyrinthitis.
- Patients with central vertigo often require neuroimaging and specialty consultation. Posterior fossa hemorrhage requires immediate neurosurgical consultation. Ischemic cerebrovascular events generally require neurologic admission.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 223, ''Vertigo and Dizziness,'' by Brian Goldman.

143 SEIZURES AND STATUS EPILEPTICUS IN ADULTS

Mark E. Hoffmann

EPIDEMIOLOGY

- There are approximately 100,000 new seizure cases diagnosed in the United States each year.[1]
- Incidence rates are highest among people <20 years, with a second peak in incidence in those >60 years. The mortality rate for seizures is between 1 to 10 percent.[2]

PATHOPHYSIOLOGY

- A seizure is an episode of abnormal neurologic function caused by an abnormal electrical discharge of brain neurons.
- When this occurs in patients who are otherwise normal and in whom no evident cause for the event can be discerned, the seizures are referred to as primary.
- Seizures that occur as a consequence of some other identifiable neurologic condition are referred to as secondary.
- The mechanisms involved in generating clinical seizures appear to be multifactorial, requiring intense and prolonged neuronal electrical discharges and failure or inhibition of normal protective mechanisms.
- Structural abnormalities from insults (trauma, stroke, abscess, tumor, or bleeding) can act as epileptogenic foci. Factors such as medical noncompliance, fever, sleep deprivation, drugs and toxins, alcohol withdrawal, infection, pregnancy, hypertension, metabolic derangement, and infection can lower the seizure threshold.

CLINICAL FEATURES

- The two major groups of seizures are generalized and partial.[3]
- Generalized seizures always present with alteration in consciousness. Tonic-clonic seizures (grand mal) have a tonic phase with extensor muscle contraction and apnea followed by a rhythmic clonic phase. The recovery phase may have a postictal period. Absence seizures (petit mal) present with a confused and detached state with staring or eye fluttering. Other generalized seizures include myoclonic, tonic, clonic, and atonic (drop attacks).
- Partial seizures are subdivided into simple partial seizures (consciousness intact), which feature perceptual distortions, hallucinations, or focal involuntary motor activity, and complex partial seizures (consciousness altered), which feature automatism, visceral symptoms, hallucination, memory disturbances, and affective changes. Frequently, complex partial seizures are misdiagnosed as psychiatric problems.
- Partial seizures (simple partial or complex partial) may progress into a generalized seizure. This is referred to ''secondary generalization.''
- A transient focal deficit following a simple or complex partial seizure is referred to as Todd's paralysis and should resolve within 48 h.

DIAGNOSIS AND DIFFERENTIAL

- The history is extremely important (witnesses, onset, type of seizure activity, duration, aura, bowel or bladder losses, drug or alcohol abuse, and last medication doses).
- The physical exam should note any evidence of trauma (head, neck, posterior shoulder dislocations, or tongue biting), bowel or bladder incontinence, and mental status or neurologic exam deficits.
- Many episodic disturbances of neurologic function may be mistaken for seizures; these include syncope, migraines, movement disorders, narcolepsy, cataplexy, hyperventilation syndrome, and pseudoseizures.
- Secondary seizures may be caused by intracranial hemorrhage, head trauma, tumors, arteriovenous malformations, infections, metabolic disturbances (sodium, glucose, hypocalcemia, hypomagnesemia, hepatic failure, or uremia), toxins, drugs, withdrawal, eclampsia, hypertensive encephalopathy, and anoxic-ischemic injury.
- A serum glucose and anticonvulsant drug level may suffice for known seizure patients with a typical seizure event.
- Patients with a first-time seizure require more extensive studies, which include serum glucose, electrolytes, magnesium, calcium, blood urea nitrogen, creatinine, prolactin level, urinalysis, urine myoglobin, pregnancy test, toxicology screen, cerebrospinal fluid analysis, and computed tomography scan of the head.[4,5]
- An electroencephalogram (EEG) and magnetic resonance imaging may be scheduled on an outpatient or inpatient basis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway, breathing, and circulation should be stabilized.
- Cervical spine immobilization should be instituted for any unwitnessed event or obtunded patient. Patients should be placed on oxygen, cardiac monitor, pulse oximetry, blood pressure monitoring, and an intravenous (IV) line should be established.
- Patients who are actively seizing should be protected from injury and placed in a recovery position to prevent aspiration.
- Intubation should be considered for patients with prolonged seizures, those requiring gastrointestinal decontamination, and those being transferred to another facility.
- Thiamine 100 mg IV should be administered to patients with a history of alcohol abuse. A serum glucose level should be checked on all patients, and glucose should be administered to those who are hypoglycemic.
- Benzodiazepines are administered to control seizures (lorazepam 2 mg/min IV up to 0.1 mg/kg).
- Phenytoin 18 mg/kg IV can be loaded at a rate of 50 mg/min. If seizures continue, a second dose of 5 to 10 mg/kg IV may be administered. Alternatively, fosphenytoin 20 phenytoin equivalents (PE)/kg may be infused at 100 to 150 PE/min.
- Phenobarbital is a second-line anticonvulsant agent. The loading dose is 20 mg/kg infused at 50 mg/min. A second dose of 10 mg/kg may be given. Airway status and respiratory depression should be closely monitored.
- For seizures refractory to the preceding therapy, induction with midazolam, propofol, thiopental, or pentobarbital, in conjunction with succinylcho-

line, pancuronium, or vecuronium, may be used. Should neuromuscular blocking agents be used, continuous EEG monitoring is mandated.[6]
- Magnesium sulfate is used to treat eclampsia-induced seizures, starting with 4 to 6 g bolus IV, followed by a 1 to 2 g/h infusion. Definitive therapy is delivery of the fetus.
- Patients with an underlying seizure disorder who present with a seizure may be discharged after returning to their baseline mental status and their serum anticonvulsant level checked.
- The disposition of patients with a first-time seizure should be made in conjunction with the physician assuming follow-up care.
- Patients with status epilepticus, persistently altered mental status, underlying central nervous system infection or mass lesion, or clinically significant hypoxia, hypoglycemia, hyponatremia, or dysrhythmias should be admitted.
- All patients discharged should be advised to avoid driving, swimming, working at heights, or operating machinery.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 224, ''Seizures and Status Epilepticus in Adults,'' by Christina Catlett Viola.

144 ACUTE PERIPHERAL NEUROLOGIC LESIONS

Alex G. Garza

DISORDERS OF THE NEUROMUSCULAR JUNCTION

BOTULISM

- Botulism is caused by the *Clostridium botulinum* toxin and occurs is three forms: food-borne, wound, and infantile types.
- In the United States, the primary source is improperly prepared or stored food.
- Wound botulism should be considered in patients with open wounds, IV drug abuse, or symptoms of progressive, symmetric descending paralysis.
- Infantile botulism is usually caused by ingestion of spores, often honey, that produce a systemically absorbed toxin.
- Clinical features appear 1 to 2 d following ingestion and may be preceded by nausea, vomiting, and diarrhea. Early complaints commonly involve the optic or bulbar musculature and progress to descending weakness and respiratory insufficiency.
- Absent papillary light reflex is a diagnostic clue; the patient has normal mentation.
- Infants may present with poor sucking, listlessness, constipation, and weakness.
- Treatment includes respiratory support, gastrointestinal (GI) and wound decontamination, botulinum antitoxin (in consultation with an infectious disease specialist), and admission.

MYASTHENIA GRAVIS

This condition is discussed in Chap. 145.

ACUTE PERIPHERAL NEUROPATHIES

GUILLAIN-BARRÉ SYNDROME

- Guillain-Barré affects individuals of all ages and is the most common form of acute generalized neuropathy. There is usually an antecedent viral illness, commonly gastroenteritis.
- The patient may first notice numbness and tingling in the lower extremities, which is followed by as-

cending weakness of the legs, thighs, and upper extremities.
- In classic cases, there is symmetric extremity weakness, more pronounced in the legs. The hallmark physical examination finding is absent deep tendon reflexes.
- In all forms of the disease, there is potential for developing respiratory failure and lethal autonomic fluctuations.
- Lumbar puncture should be performed when acute disease is suspected. A classic laboratory finding is a high cerebrospinal fluid (CSF) protein with normal glucose and cell count.
- Differential diagnosis includes diphtheria, botulism, lead poisoning, tick paralysis, cord compression, and porphyria.
- No specific treatment is needed in the emergency department (ED). Patients should be admitted for monitoring, observation of airway status, and treatment.[1]

ACUTE INTERMITTENT PORPHYRIA

- Acute intermittent porphyria (AIP) is a rare autosomal dominant disorder involving the triad of weakness, psychosis, and abdominal pain. The three symptoms occasionally occur together, but in most cases they arise independently.
- Medications such as phenytoin, barbiturates, sulfonamides, and estrogens may trigger flares.
- The major neurologic findings include weakness and diminished reflexes, particularly in the legs.
- The key management issue is identification and discontinuation of the offending drug. Other treatment modalities include supportive care, glucose infusions, vitamin B_6 therapy, and hematin therapy.

MYOPATHIES

POLYMYOSITIS

- Polymyositis is an inflammatory myopathy that affects individuals over the age of 30, with a slight propensity for women.
- Patients present with complaints of proximal symmetric weakness. Some patients will present with rapidly evolving weakness, muscular pain, arthralgias, dysphagia, fever, and Raynaud's phenomenon.
- On physical examination, the patient may exhibit reduced proximal strength. Sensation and deep tendon reflexes are normal unless there is severe weakness.
- Patients may have an elevated sedimentation rate, creatinine phosphokinase, and leukocyte count.
- Admission is usually warranted for new cases in order to monitor the airway status and clinical progression.

DERMATOMYOSITIS

- Unlike polymyositis, dermatomyositis can affect children; in adults, it mostly affects women.
- The clinical manifestations are similar to those of polymyositis except for a violaceous rash that typically appears over the face and hands.
- The neurologic examination demonstrates a myopathic distribution of weakness without sensory or reflex abnormalities in most cases.
- The laboratory findings are also similar to those of polymyositis, with an elevated sedimentation rate and creatinine phosphokinase.
- Treatment is aimed at immunosuppression.[2]

ENTRAPMENT NEUROPATHIES

CARPAL TUNNEL SYNDROME

- Carpal tunnel syndrome is the most commonly seen entrapment neuropathy.
- Patients describe intermittent pain or numbness in the thumb and first two fingers.
- The symptoms can be reproduced by tapping on the median nerve (Tinel's sign) or compression of the nerve (Phalen's sign).
- When symptoms become long-standing or severe, weakness of the thenar musculature may develop.
- Wrist splints worn at night are useful in the conservative management of carpal tunnel syndrome. Patients should be referred to a hand surgeon for outpatient management.

ULNAR NERVE ENTRAPMENT

- Ulnar nerve entrapment usually occurs at the elbow, producing numbness to fifth digit and ulnar half of the ring finger.
- Weakness and wasting of the hypothenar muscles occur very late in the course.

DEEP PERONEAL NERVE ENTRAPMENT

- Entrapment of the deep peroneal nerve at the fibular head can cause footdrop and numbness of the web space between the great and second toes.
- This condition occurs in the setting of injury to the leg, rapid weight loss, or habitual crossing of the legs.
- Peroneal nerve entrapment should be confirmed by an outpatient electromyogram, which differentiates it from lumbar root disease or motor neuron disease.
- Almost all cases are treated conservatively and improve without specific therapy.

MERALGIA PARESTHETICA

- Meralgia paresthetica is entrapment of the lateral cutaneous nerve of the thigh.
- Patients describe numbness and dysesthesia of the lateral aspect of the upper leg.
- This occurs following weight loss or, notably, pelvic surgery or an obstetric procedure where the legs are abducted and flexed for prolonged periods of time.
- Tricyclic antidepressants are useful in the management of the dysesthesia associated with meralgia paresthetica.

PLEXOPATHIES

- Brachial neuritis is an acute condition that tends to affect younger individuals, with a slight male predominance. The cause is idiopathic, but cases have been reported following immunizations or viral infections.
- Patients report excruciating shoulder, back, or arm pain followed by weakness of the arm or shoulder girdle. In one-third of cases, it is bilateral.
- On physical examination, the patient has weakness in various distributions of the brachial plexus. The anterior interosseous nerve is also affected preferentially, causing inability to form a pincer with the index finger and thumb. Sensory abnormalities are found but are not as profound as the motor dysfunction. Reflexes are diminished in the affected limb.
- A chest radiograph assists in screening for mass lesions involving the brachial plexus.
- The management of brachial plexitis is supportive; no specific therapies have been shown to affect the course of the illness.

LUMBAR PLEXOPATHY

- Lumbar plexopathy occurs in diabetic patients who present with the acute onset of ipsilateral back pain, followed within days by progressive leg weakness.
- The examination reveals decreased leg strength in a variety of patterns reflecting impairment of plexus function with relatively normal symmetric sensation. There may be muscle wasting in affected limbs in long-standing disease. Bowel and bladder function are not affected.
- Radiographs and magnetic resonance imaging (MRI) of the lumbar spine are useful to screen for spine compression and degenerative or neoplastic disease.
- Patients with acute weakness from lumbar plexopathy should be admitted to the hospital for further evaluation and rehabilitation.

HIV-ASSOCIATED PERIPHERAL NEUROLOGIC DISEASE

- HIV infection, its complications, and its pharmacologic treatments are associated with a number of peripheral neurologic disorders.
- The most common of these, HIV neuropathy and drug-induced neuropathy, are chronic processes that do not cause sudden disability or symptoms.
- HIV-infected patients also have a higher rate of mononeuritis multiplex and inflammatory myopathy resembling polymyositis.
- Patients in the early stages of HIV infection have greater susceptibility to Guillain-Barré syndrome. The presentation is similar to that of the non-HIV-infected patient except that a CSF pleocytosis is seen commonly.
- In the latter stages of AIDS, patients may develop cytomegalovirus (CMV) radiculitis. These patients almost always have evidence of CMV infection elsewhere in the body and may have CMV retinitis.
- Patients with CMV radiculitis become acutely weak, with primarily lower extremity involvement, and may have variable degrees of bowel and bladder dysfunction. The physical examination shows primarily lower extremity weakness and hyporeflexia and decreased sensation in the lower extremities and groin. Rectal tone may be impaired.
- Lumbar puncture reveals a pleocytosis with predominantly polymorphonuclear cells and modestly increased protein. Imaging of these patients

is mandatory to rule out a mass lesion of the lower spine and nerve roots.
- The treatment of CMV radiculitis is IV gancyclovir, which should be initiated prior to definitive diagnosis.

BELL'S PALSY

- Bell's palsy is the most common cause of acute facial paralysis but is similar to other processes that are important to recognize.
- Patients with Bell's palsy complain of sudden facial weakness, difficulty with articulation, problems keeping an eye closed, or inability to keep food in the mouth on one side.
- Because the seventh cranial nerve also serves other functions, the patient may have variable degrees of eye dryness, metallic taste, and facial pain (commonly around the ear).
- The physical examination is notable for weakness of one side of the face, including the forehead, and no other focal neurologic findings. Occasionally, there may be slightly decreased sensation along the external acoustic meatus.
- Differential diagnosis considerations include stroke, Lyme disease, parotid tumors, middle ear lesions, cerebellopontine angle tumors, eighth-cranial-nerve lesions, HIV disease, and vascular disease.
- Stroke can lead to sudden facial weakness that involves only the lower face and also leads to neurologic involvement below the neck or other cranial neuropathies. If muscle strength is retained in the forehead and upper face, the lesion is most probably central (i.e., in the brainstem or above); this would exclude Bell's palsy, and CT scanning of the head would be indicated.
- The ear should be inspected carefully to rule out ulcerations caused by cranial herpes zoster activation (Ramsay-Hunt's syndrome), which should be treated with oral acyclovir.
- All patients with facial weakness should be screened for HIV risk factors, since seventh-nerve palsy can occur at the time of seroconversion.
- The treatment of Bell's palsy with steroids is controversial. More recent studies have suggested that steroids in combination with acyclovir lead to better outcomes. If the patient is seen more than a week after paresis began, steroids generally are not indicated.
- Eye care must be meticulous to avoid corneal abrasions. Patients should apply Lacrilube to the affected eye and patch the eye before sleeping.[3]

LYME DISEASE

- Lyme disease affects individuals exposed to the tick-borne pathogen *Borrelia burgdorfei.* Patients often, but not always, report prior tick exposure and have spent time in areas endemic to deer ticks.
- Although there are multiple neurologic manifestations, one of the most common sites of involvement is the peripheral nervous system.
- Initial manifestations of Lyme disease include arthralgias and fatigue.
- Neurologic complications ensue in the following weeks. A common neurologic sign of Lyme infection is seventh-nerve palsy. Lyme disease affects the peripheral nerves and the nerve roots.
- The patient may describe the acute or subacute progression of weakness and sensory loss, sometimes associated with radicular pain.
- On physical examination, apart from the seventh-nerve involvement, there may be weakness in the limbs. If there is localized radicular inflammation, there may well be a patchy myotomal pattern.
- Laboratory features suggestive of Lyme disease include serum and CSF Lyme antibodies. A CSF pleocytosis and increased protein with a normal glucose is the most common abnormality.[4,5]

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145 CHRONIC NEUROLOGIC DISORDERS

Mark B. Rogers

AMYOTROPHIC LATERAL SCLEROSIS

EPIDEMIOLOGY

- The typical age of onset for amyotrophic lateral sclerosis (ALS) is over age 50.

PATHOPHYSIOLOGY

- ALS is caused by both upper and lower motor neuron degeneration by an unknown etiology, which leads to rapidly progressive muscle wasting and weakness.

CLINICAL FEATURES

- Upper motor neuron dysfunction causes limb spasticity, hyperreflexia, and emotional lability.
- Lower motor neuron dysfunction causes limb muscle weakness, atrophy, fasciculations, dysarthria, dysphagia, and difficulty in mastication.
- Symptoms are often asymmetric and more prominent in the upper extremities.
- Patients may initially complain of cervical or back pain consistent with an acute compressive radiculopathy.
- Respiratory difficulty progresses to failure.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is clinical and is often previously established. Electromyography is useful.
- Other illnesses that should be considered include myasthenia gravis, diabetes mellitus, dysproteinemia, thyroid dysfunction, vitamin B_{12} deficiency, lead toxicity, vasculitis, and central nervous system (CNS) and spinal cord tumors.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergency care is required for acute respiratory failure, aspiration pneumonia, choking episodes, or trauma from falls.
- The treatment goal is to optimize pulmonary function through the use of nebulizer treatments, steroids, antibiotics, or endotracheal intubation.
- Patients with impending respiratory failure, pneumonia, inability to handle secretions, and disease progression requiring long-term care should be admitted.[1,2]

MULTIPLE SCLEROSIS

EPIDEMIOLOGY

- Three clinical courses are seen in multiple sclerosis (MS): relapsing and remitting (two-thirds of patients), relapsing and progressive, or chronically progressive.
- Peak age of onset is the third decade of life. Females are two to three times more likely to contract MS than are males.

PATHOPHYSIOLOGY

- Although the etiology of MS is unknown, it involves multifocal areas of CNS demyelination, causing motor, sensory, visual, and cerebellar dysfunction.

CLINICAL FEATURES

- Deficits are described by patients as a heaviness, weakness, stiffness, or numbness of an extremity. Lower extremity symptoms are usually more severe.
- Lhermitte's sign is an electric shock sensation, a vibration, or dysesthetic pain going down the back into the arms or legs that occurs with neck flexion.
- Decreased strength, increased tone, hyperreflexia, clonus, decreased proprioception, and reduced pain and temperature sense may be seen.
- Increases in body temperature may worsen symptoms (e.g., exercise, hot baths, or fever).
- Rarely, acute transverse myelitis may occur. Cerebellar lesions may cause intention tremor or ataxia. Brainstem lesions may cause vertigo. Cognitive and emotional problems are common (e.g., mood disorders or dementia).
- Optic neuritis, usually causing unilateral loss of central vision, is the first presenting symptom in up to 30 percent of cases and may cause an afferent pupillary defect (Marcus Gunn pupil). Retrobulbar or extraocular muscle pain usually precedes

visual loss. Fundoscopy is usually normal, but the disc may be pale.
- Acute bilateral internuclear ophthalmoplegia (INO), which causes abnormal adduction and horizontal nystagmus, is highly suggestive of MS.
- Dysautonomia causes vesicourethral, gastrointestinal, and sexual dysfunction.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is clinical and is suggested by two or more episodes lasting days to weeks and causing dysfunction that implicates different sites in the white matter.
- Magnetic resonance imaging (MRI) of the head may demonstrate lesions in the supratentorial white matter or periventricular areas.
- Cerebrospinal fluid protein and gamma-globulin levels are often elevated.
- The differential diagnosis includes systemic lupus erythematosus, Lyme disease, neurosyphilis, and HIV disease.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Those with severe motor or cerebellar dysfunction may be treated with steroids. A short-term (up to 5 days), high-dose course of pulsed methylprednisolone (250 mg IV every 6 h), followed by oral prednisone tapered over 2 to 3 weeks, may be beneficial.
- Fever must be reduced to minimize symptoms. A careful search for an infectious source should be performed. Acute cystitis and pyelonephritis are frequently the sources. Any infection associated with postvoid residuals greater than 100 mL requires intermittent catheterization.
- Admission is required for those at risk for further complications, respiratory compromise, depression with suicidal ideation, and those requiring IV antibiotics or steroids.[3,4]

MYASTHENIA GRAVIS

EPIDEMIOLOGY

- Peak age of onset for myasthenia gravis (MG) is in the second and third decades of life for females and in the seventh or eighth decade for males.

PATHOPHYSIOLOGY

- MG is an autoimmune disease caused by antibody destruction of the acetylcholine receptors (AChR) at the neuromuscular junction, which results in muscle weakness.

CLINICAL FEATURES

- Most MG patients have generalized weakness, specifically of the proximal extremities, neck extensors, and facial or bulbar muscles. There is usually no deficit in sensory, reflex, or cerebellar function.
- Ptosis and diplopia are the most common symptoms. Symptoms worsen as the day progresses or with muscle use (e.g., prolonged reading or chewing), and improve with rest.
- Myasthenic crisis is a life-threatening condition involving extreme weakness of the respiratory muscles that may progress to respiratory failure.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of MG is confirmed through the edrophonium (Tensilion) test, electromyogram, and serum testing for AChR antibodies.
- The differential includes Eaton-Lambert syndrome, drug-induced disorders (e.g., penicillamines, aminoglycosides, and procainamide), ALS, botulism, thyroid disorders, and other CNS disorders (intracranial mass lesions).

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- With myasthenic crisis, supplemental oxygen and aggressive airway management, including endotracheal intubation, should be considered. Depolarizing paralytic agents (i.e., succinylcholine) and long-acting nondepolarizing agents should be avoided.
- If the Tensilon test is positive, then neostigmine can be administered (0.5 to 2 mg IV or SC or 15 mg orally), which will be effective within 30 min and last for 4 h.
- Severe MG patients should receive high-dose steroid therapy (mandating a stay in the intensive care unit due to potential increased weakness) and possible plasmapheresis or IV immunoglobulin therapy.

- A neurologist should be consulted for disposition and admission.[5]

PARKINSON'S DISEASE

EPIDEMIOLOGY

- The average age of onset for Parkinson's disease is 55 to 60 years of age.

PATHOPHYSIOLOGY

- The etiology of Parkinson's disease is unknown, but patients have reduced dopaminergic receptors in the substantia nigra.

CLINICAL FEATURES

- Parkinson's disease presents with four classic signs: resting tremor, cogwheel rigidity, bradykinesia or akinesia, and impaired posture and equilibrium. Other signs include facial and postural changes, voice and speech abnormalities, depression, and fatigue.
- Initially, most patients complain of a unilateral resting arm tremor called ''pill rolling,'' which improves with intentional movement.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis is clinical and is most often previously established. No laboratory test or neuroimaging study is pathognomonic.
- ''Parkinsonism'' can result from street drugs, toxins, neuroleptic drugs, hydrocephalus, head trauma, and rare neurologic disorders.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Parkinson's disease patients may be on medications that increase central dopamine (e.g., levodopa, carbidopa, and amantadine), bind dopamine receptors (e.g., bromocriptine), and are anticholinergics (e.g., benztropine).
- Medication toxicity includes psychiatric or sleep disturbances, cardiac dysrhythmias, orthostatic hypotension, dyskinesias, and dystonia.
- With significant motor or psychiatric disturbances (e.g., hallucinations) or decreased drug efficacy a ''drug holiday'' for 1 week should be initiated.

POLIOMYELITIS AND POSTPOLIO SYNDROME

EPIDEMIOLOGY

- Poliomyelitis leads to paralysis in less than 5 percent of infected patients.

PATHOPHYSIOLOGY

- Poliomyelitis is caused by an enterovirus that produces paralysis via motor neuron destruction and muscle denervation and atrophy.[6]
- In developed countries, transmission is oral to oral; in developing countries, however, transmission is fecal to oral.

CLINICAL FEATURES

- Polio infection is asymptomatic in over 90 percent of cases.
- Most symptomatic patients have only a mild viral syndrome and no paralysis. Symptoms include fever, malaise, headache, sore throat, and GI symptoms.
- Spinal polio results in *asymmetric* proximal limb weakness and flaccidity, absent tendon reflexes, and fasciculations; sensory deficits are usually not seen. Maximal paralysis occurs within 5 days.
- Paralysis will resolve within the first year in nearly all patients.
- Other sequelae include bulbar polio (speech and swallowing dysfunction) and encephalitis.
- Postpolio syndrome is a recurrence of motor symptoms after a latent period of several decades. Symptoms may include muscle fatigue, joint pain, or weakness of new or previously affected muscle groups. These patients may have new bulbar, respiratory, or sleep difficulties.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis should be considered in any patient with an acute febrile illness, aseptic meningitis, and asymmetric flaccid paralysis.
- CSF may reveal an elevated white blood cell count (mostly neutrophils) and positive culture for poliovirus.

- Throat and rectal swabs are higher-yield tests.
- The diagnosis of postpolio syndrome is based on a previous history of paralytic polio with recovery and new symptoms.
- The differential includes Guillain-Barré syndrome, peripheral neuropathies (e.g., mononucleosis, Lyme disease, or porphyria), abnormal electrolyte levels, toxins, inflammatory myopathies, and other viruses (e.g., coxsackievirus, mumps, echo, and various enteroviruses).[7]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment is supportive.
- Disposition should be made in consultation with a neurologist.

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146 MENINGITIS, ENCEPHALITIS, AND BRAIN ABSCESS

O. John Ma

MENINGITIS

EPIDEMIOLOGY

- There are 25,000 cases of bacterial meningitis annually in the United States; two-thirds of these cases occur in children. The mortality rate is 25 percent in neonates, 5 percent in children beyond infancy, and 25 percent in adults.[1,2]
- There is an increasing prevalence of ceftriaxone- and penicillin-resistant *Streptococcus pneumoniae* strains in the community.[3]

PATHOPHYSIOLOGY

- Over two-thirds of bacterial meningitis cases are caused by *Strep. pneumoniae, Neisseria meningitidis,* and *Haemophilus influenzae.*[4]
- Infection begins with entrance of bacteria into the subarachnoid space, usually by upper airway inoculation, and is followed by dissemination into the bloodstream and invasion across the blood-brain barrier.[4] Direct inoculation is also possible from infection of parameningeal structures (e.g., otitis media, brain abscess, sinusitis), neurosurgery, and traumatic or congenital communications with the exterior.[5]
- The brain becomes edematous through several mechanisms: (1) There is reduced cerebrospinal fluid (CSF) drainage through interference with flow and absorption by arachnoid granulations. The increased quantity of CSF results in periventricular edema and hydrocephalus; and (2) There is disruption of the blood-brain barrier, which allows entry of protein and water. These mechanisms lead to ischemia as intracranial pressure exceeds cerebral perfusion pressure.[5]

CLINICAL FEATURES

- In classic and fulminant cases of bacterial meningitis, the patient presents with fever, headache, stiff neck, photophobia, and altered mental status.[4] Seizures may occur in up to 25 percent of cases.
- The presenting picture, however, may be less specific, particularly in the very young and elderly.

Confusion and fever may be signs of meningeal irritation in the elderly.
- Physical examination must include assessment for meningeal irritation with resistance to passive neck flexion, Brudzinski's sign (flexion of hips and knees in response to passive neck flexion), and Kernig's sign (contraction of hamstrings in response to knee extension while hip is flexed).
- The skin should be examined for the purpuric rash characteristic of meningococcemia. Paranasal sinuses should be percussed and ears examined for evidence of primary infection in those sites.
- Focal neurologic deficits, which are present in 25 percent of cases, should be documented. Fundi should be assessed for papilledema, indicating increased intracranial pressure.

DIAGNOSIS AND DIFFERENTIAL

- When the diagnosis of bacterial meningitis is entertained, performing a lumbar puncture (LP) is mandatory. At a minimum, CSF should be sent for Gram stain and culture, cell count, protein, and glucose. Typical CSF results for meningeal processes are listed in Table 146-1.
- Additional studies to be considered are latex agglutination or counterimmune electrophoresis for bacterial antigens in bacterial cases that may have been only partially treated, India ink and latex agglutination assay for fungal antigen in cryptococcal meningitis, acid-fast stain and culture for mycobacteria in tuberculous meningitis, *Borrelia* antibodies for possible Lyme disease, and viral cultures in suspected viral meningitis.
- Other laboratory tests should include a complete blood count, blood cultures, partial thromboplastin and prothrombin times, as well as serum glucose, sodium, and creatinine.
- The differential diagnosis includes subarachnoid hemorrhage, meningeal neoplasm, brain abscess, viral encephalitis, and cerebral toxoplasmosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emergent respiratory and hemodynamic support are given top priority.
- Upon presentation of the patient with suspected bacterial meningitis, LP should be performed expeditiously if the patient has no focal neurologic deficits or evidence of intracranial mass and coagulopathy on clinical grounds.
- Antibiotic therapy should be initiated as preparations for LP are made. Antibiotic therapy administered up to 2 h prior to LP will not decrease the diagnostic sensitivity if cerebrospinal fluid (CSF) bacterial antigen assays are obtained along with CSF cultures.[6]
- If the patient has focal neurologic deficits or papilledema, computed tomography (CT) of the head should be performed prior to LP in order to determine the possible risks for transtentorial or tonsillar herniation associated with LP. In these cases, antibiotic therapy must be initiated prior to patient transport to the radiology suite for CT scanning. Antibiotic therapy should always be initiated in the emergency department (ED) and never be delayed for CT scanning or other studies.
- Presently, the antibiotic therapy of choice is a third-generation cephalosporin, such as ceftriaxone or cefotaxime. A dose of 2 g IV should be administered and will cover the most common organisms (*Strep. pneumoniae, H. influenzae, N. meningitidis*). Additionally, it is recommended that ampicillin 2 g IV be administered to cover for *Listeria monocytogenes.* Vancomycin should

TABLE 146-1 Typical Spinal Fluid Results for Meningeal Processes

PARAMETER (NORMAL)	BACTERIAL	VIRAL	NEOPLASTIC	FUNGAL
OP (<170 mmCSF)	>300 mm	200 mm	200 mm	300 mm
WBC (<5 mononuclear)	>1000/μL	<1000/μL	<500/μL	<500/μL
% PMNs (0)	>80%	1–50%	1–50%	1–50%
Glucose (>40 mg/dL)	<40 mg/dL	>40 mg/dL	<40 mg/dL	<40 mg/dL
Protein (<50 mg/dL)	>200 mg/dL	<200 mg/dL	>200 mg/dL	>200 mg/dL
Gram stain (−)	+	−	−	−
Cytology (−)	−	−	+	+

ABBREVIATIONS: OP = opening pressure; PMNs = polymorphonuclear cells; and WBC = white blood cells.
SOURCE: From Greenlee,[7] with permission.

be added if *Strep. pneumoniae* resistance is possible. For the patient who is severely penicillin-allergic, the combination of chloramphenicol and trimethoprim-sulfamethoxazole is recommended.
- Steroid therapy (dexamethasone 0.15 mg/kg IV) is controversial in adults and, if initiated, should be given prior to the first dose of antibiotics.[8–10]
- Hypotonic fluids should be avoided. Serum sodium levels should be monitored to detect the syndrome of inappropriate antidiuretic hormone or cerebral salt-wasting. Hyperpyrexia should be treated with acetaminophen. Coagulopathy must be corrected using specific replacement therapies.
- Seizures and signs of marked intracranial pressure should be treated in the standard fashion.
- Viral meningitis without evidence of encephalitis can be managed on an outpatient basis provided that the patient is nontoxic in appearance, can tolerate oral fluids, and has reliable follow-up. However, it remains a diagnosis of exclusion; unless the diagnosis of viral meningitis is obvious, admission is warranted.

ENCEPHALITIS

EPIDEMIOLOGY

- The incidence of encephalitis is about one-tenth that of bacterial meningitis.
- Arboviruses can account for up to 50 percent of cases during epidemic outbreaks. The four most common arboviral encephalitides in the United States are the California encephalitis serogroup, St. Louis equine encephalitis, western equine encephalitis, and eastern equine encephalitis.
- Herpes simplex virus type 1 (HSV-1) is typically seen in older children and adults as a reactivation disease. Herpes simplex virus type 2 (HSV-2) is seen in neonates as a result of perinatal transmission.

PATHOPHYSIOLOGY

- In North America, viruses that cause encephalitis include arboviruses, lyssavirus (rabies), HSV-1, herpes zoster virus, and Epstein-Barr virus (EBV).
- The arboviruses are transmitted by mosquitoes and ticks. Rabies is transmitted by the bite of an infected animal. Impaired immune status plays a role in herpes zoster and cytomegalovirus (CMV) encephalitis.
- Neurologic dysfunction and damage are caused by disruption of neural cell functions by the virus and by the effects of the host's inflammatory responses. Gray matter is predominantly affected, resulting in cognitive and psychiatric signs, lethargy, and seizures.[11,12]

CLINICAL FEATURES

- Encephalitis should be considered in patients presenting with any or all of the following features: new psychiatric symptoms, cognitive deficits (aphasia, amnestic syndrome, acute confusional state), seizures, and movement disorders.
- Signs and symptoms of headache, photophobia, fever, and meningeal irritation may be present. Assessment for neurologic findings and cognitive deficits is crucial. Motor and sensory deficits are not typical.
- Encephalitides may show special regional trophisms. HSV involves limbic structures of the temporal and frontal lobes, with prominent psychiatric features, memory disturbance, and aphasia. Some arboviruses predominantly affect the basal ganglia, causing choreoathetosis and parkinsonism. Involvement of the brainstem nuclei leads to the hydrophobic choking characteristic of rabies encephalitis.[11,12]

DIAGNOSIS AND DIFFERENTIAL

- Emergency department (ED) diagnosis can be suggested by findings on magnetic resonance imaging (MRI) and LP. MRI not only excludes other potential lesions, such as brain abscess, but may display findings highly suggestive of HSV encephalitis if the medial temporal and inferior frontal gray matter is involved.
- On LP, findings of aseptic meningitis are typical.
- The differential diagnosis includes brain abscess; Lyme disease; subarachnoid hemorrhage; bacterial, tuberculous, fungal, or neoplastic meningitis; bacterial endocarditis; postinfectious encephalomyelitis; toxic or metabolic encephalopathies; and primary psychiatric disorders.[11,12]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The patient suspected of suffering from viral encephalitis should be admitted. Of the viruses causing encephalitis, only HSV has been shown by

clinical trial to be responsive to antiviral therapy. The agent of choice is acyclovir 10 mg/kg IV.
- Potential complications of encephalitis—seizures, disorders of sodium metabolism, increased intracranial pressure, and systemic consequences of a comatose state—should be handled in the standard fashion.

BRAIN ABSCESS

EPIDEMIOLOGY

- The incidence of brain abscess has progressively declined over the past century, reflecting the effect of antibiotics on predisposing conditions, such as otitis media.

PATHOPHYSIOLOGY

- A brain abscess is a focal pyogenic infection. It is composed of a central pus-filled cavity ringed by a layer of granulation tissue and an outer fibrous capsule.

CLINICAL FEATURES

- Since patients typically are not acutely toxic, the presenting features of brain abscess are nonspecific. For this reason, the initial diagnosis can be difficult in the ED.
- Presenting signs and symptoms include headache, neck stiffness, fever, vomiting, confusion, or obtundation. Meningeal signs and focal neurologic findings—such as hemiparesis, seizures, and papilledema—are present in less than half the cases.

DIAGNOSIS AND DIFFERENTIAL

- Classically, brain abscess can be diagnosed by a CT scan of the head with contrast, which demonstrates one or several thin, smoothly contoured rings of enhancement surrounding a low-density center that, in turn, is surrounded by white matter edema.
- LP is contraindicated when brain abscess is suspected and after the diagnosis has been established. Other studies like routine laboratory work and electroencephalography (EEG) are nonspecific. Blood cultures should be obtained.
- The differential diagnosis includes cerebrovascular disease, meningitis, brain neoplasm, subacute brain hemorrhage, and other focal brain infections, such as toxoplasmosis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Decisions on antibiotic therapy for brain abscess should depend on the likely source of the infection. In a suspected otogenic case, initial therapy should consist of a third-generation cephalosporin, such as ceftriaxone or cefotaxime, or trimethoprim-sulfamethoxazole with chloramphenicol or metronidazole.
- In a suspected sinogenic or odontogenic case, initial therapy should consist of high-dose penicillin with chloramphenicol or metronidazole.
- In a suspected cardiac case, initial therapy should consist of vancomycin with chloramphenicol or metronidazole.
- When communication with the exterior is suspected, as in penetrating trauma or after a neurosurgical procedure, initial therapy should consist of vancomycin or nafcillin.
- Ceftazidime should be added if gram-negative aerobes are suspected.
- Finally, in cases where no clear etiology exists, initial empiric therapy should consist of a third-generation cephalosporin and metronidazole.
- Neurosurgical consultation and admission are warranted, since many cases will require surgery for diagnosis, bacteriology and, often, definitive treatment.[13]

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Section 18

EYE, EAR, NOSE, THROAT, AND ORAL EMERGENCIES

147 OCULAR EMERGENCIES

Steven Go

INFECTIONS

STYE (EXTERNAL HORDEOLUM)

- A stye is an acute infection of an oil gland at the lid margin.
- Emergency department (ED) care of a stye includes warm compresses and erythromycin ointment for 7 to 10 days.

CHALAZION (INTERNAL HORDEOLUM)

- A chalazion is an acute or chronic infection of the meibomian gland.
- Emergency department care is the same as for a stye, plus a 14-day regimen of doxycycline for refractory cases.
- Persistent chalazions should be referred to an ophthalmologist for incision, curettage, and possible biopsy.

CONJUNCTIVITIS

- Bacterial conjunctivitis presents with eyelash matting, mucopurulent discharge, and conjunctival inflammation without corneal lesions. It is treated with topical antibiotic drops (infants: sulfacetamide 10%; adults: trimethoprim-polymyxin B drops, sulfacetamide 10% drops, tobramycin ointment, or erythromycin ointment. All are dosed tid or qid for 5 to 7 days.)
- *Neisseria gonorrhea* infections present with purulent discharge and may be diagnosed by Gram's stain (gram-negative intracellular diplococci). Emergency department care for *Neisseria gonorrhea* infections include culture, ceftriaxone 1 g intramuscularly (IM), tetracycline drops, frequent saline solution washes, and ophthalmologic consultation.
- Pseudomonas infections occur in contact lens wearers with conjunctivitis. Emergency department care should include fluoroquinolone or aminoglycoside drops. The worn contact lenses should be discarded and use of new contact lenses should not be resumed until the infection has completely cleared.
- Viral conjunctivitis is the most common type of conjunctivitis and is associated with concurrent upper respiratory infection (URI) symptoms, conjunctival injection, chemosis, and preauricular adenopathy. Epidemic keratoconjunctivitis (EKC) is a viral infection associated with greater pain, redness, photophobia, subepithelial corneal infiltrates, and a duration up to 3 weeks. Emergency department care of viral conjunctivitis includes empiric antibiotic drops, cool compresses, and naphazoline/pheniramine drops. Severe EKC may require topical steroids in consultation with an ophthalmologist.
- Allergic conjunctivitis is associated with a stringy white discharge, redness, chemosis, and pruritus and can follow an allergen exposure. Emergency department care of allergic conjunctivitis includes naphazoline/pheniramine drops, cool compresses, and avoidance of contact lens wearing during the episode.
- Neonatal conjunctivitis can be secondary to several organisms. Herpes (HSV-II) presents within the first 3 days of life and is treated with trifluridine

and occasionally parenteral acyclovir. *Neisseria gonorrhea* presents within the first 3 days of life and is treated with erythromycin ointment and parenteral ceftriaxone. *Chlamydia* and *Haemophilus* present 5 to 10 days postpartum and are treated with topical and oral erythromycin. Children with *Chlamydia* conjunctivitis are at risk for *Chlamydia* pneumonia, and infection with these two organisms requires ophthalmologic consultation. *Staphylococcus aureus* and *Streptococcus pneumoniae* present 5 to 10 days postpartum and are treated with oral erythromycin.

HERPES SIMPLEX VIRUS (HSV)

- Herpes infection can involve eyelids, conjunctiva, and cornea. It classically causes a dendritic epithelial defect. Ophthalmologic consultation should be obtained.
- Emergency department care depends on the site of infection: eyelids/conjunctiva involvement requires oral acyclovir or famciclovir plus trifluridine drops 5 times qd; keratitis involvement requires the same therapy as for eyelids/conjunctiva plus trifluridine drops 9 times qd and topical erythromycin.

HERPES ZOSTER OPHTHALMICUS (HZO)

- Herpes zoster ophthalmicus is shingles with a trigeminal distribution, ocular involvement, and, frequently, a concurrent iritis. A ''pseudodendrite'' (mucous corneal plaque without epithelial erosion) may be seen. Ophthalmologic consultation is mandatory.
- Emergency department care includes acyclovir therapy and topical erythromycin. Iritis may be treated with topical steroids and cycloplegics. Hospitalization and parenteral acyclovir may be required.
- Herpes zoster ophthalmicus must be differentiated from HSV because of the risks of topical steroid use in the latter.

PERIORBITAL CELLULITIS (PRESEPTAL CELLULITIS)

- Periorbital cellulitis presents with warm, indurated, and erythematous eyelids, without restriction of ocular motility, proptosis, painful eye movement, or impairment of pupillary function.
- Emergency department care in patients >5 years includes oral amoxicillin-clavulanate (40 mg/kg divided tid in children or 500 mg tid in adults). For toxic-appearing patients or children <5 years old, hospital admission for parenteral ceftriaxone and vancomycin may be required.
- Children <5 years also require a septic work-up because concurrent bacteremia and meningitis may be present.

ORBITAL CELLULITIS (POSTSEPTAL CELLULITIS)

- Orbital cellulitis should be suspected whenever signs and symptoms of periorbital cellulitis presents with fever, toxicity, proptosis, painful ocular motility, or limited ocular excursion.
- Emergent diagnosis with orbital and sinus thin-slice computed tomography (CT) scan without contrast is required. If this study is negative, a contrasted CT scan should be done, which may reveal a subperiosteal abscess.
- Ophthalmologic consultation and hospital admission for intravenous (IV) cefuroxime are required.

CORNEAL ULCER

- These infections of the corneal stroma present with pain, redness, and photophobia. Etiologies include dessication, trauma, direct invasion, and contact lens use.
- Slit-lamp exam reveals a staining corneal defect with a hazy infiltrate. A hypopyon may be seen.
- Emergency department care includes hourly topical ofloxacin or ciprofloxacin drops. Topical cycloplegia (1% cyclopentolate, 1 drop tid) helps relieve pain, but patching is contraindicated. Ophthalmology follow-up is required in 24 h.

TRAUMA

CORNEAL ABRASION

- Trauma may cause superficial or deep corneal abrasions that present with tearing, blepharospasm, and severe pain, which is relieved by a topical anesthetic. Instilled fluorescein will reveal dye uptake at the site of the defect.
- Emergency department care includes a cycloplegic, 1% cyclopentolate or 5% homatropine (one drop tid; both are contraindicated in narrow angle patients). Simple, clean abrasions are treated with topical erythromycin, tobramycin, or bacitracin-polymyxin ointment tid.

- Abrasions prone to infection are treated with ciprofloxacin, ofloxacin, or tobramycin drops every 4 h.
- Tetanus status should be updated on all patients with corneal abrasions.
- Studies suggest that patching does not facilitate abrasion healing and is also absolutely contraindicated in dirty abrasions and contact lens abrasions. Oral analgesics and topical cycloplegics are appropriate; however, topical anesthetics are strictly contraindicated.
- Intraocular foreign bodies should be suspected if a history compatible with penetrating injury is present.
- Ophthalmology follow-up is advised within 24 h for all corneal abrasions.

CONJUNCTIVAL FOREIGN BODIES

- When a corneal abrasion or a foreign body sensation is present, conjunctival foreign bodies should be sought with lid eversion. They may be removed with a moistened sterile swab.

CORNEAL FOREIGN BODIES

- Corneal foreign bodies may be removed with a fine needle tip, eye spud, or eye burr after applying a topical anesthetic. The resultant corneal defect should be treated as a corneal abrasion.
- Deep, corneal stoma foreign bodies or those in the central visual axis require ophthalmologic consultation for removal.
- Rust rings may be removed with an eye burr, although emergent removal is not required. Residual rust or deep stromal involvement requires ophthalmologic follow-up within 2 days.

LID LACERATIONS

- Damage to the eye and nasolacrimal system must be excluded in all eyelid and adnexal lacerations. Fluorescein instilled into the tear layer that appears in an adjacent laceration confirms injury to the nasolacrimal system.
- Suspected or proven nasolacrimal injuries, lid margin lacerations, levator mechanism lacerations, and all through-and-through lid lacerations require ophthalmology consultation.

SUBCONJUNCTIVAL HEMORRHAGE

- Disruption of conjunctival blood vessels may occur from trauma, sneezing, gagging, or the Valsalva maneuver, and will resolve spontaneously.
- When a dense, circumferential ''bloody chemosis'' is present, globe rupture must be excluded by exploration by an ophthalmologist.

TRAUMATIC IRITIS AND IRIDOCYCLITIS

- Inflammation of the iris or the ciliary body after trauma can present with photophobia, consensual pain, ciliary flush, and cell and flare in the anterior chamber.
- Emergency department care includes a cycloplegic, 1% cyclopentolate or 5% homatropine (1 drop tid). Topical steroids (prednisolone acetate 1%) may be given in consultation with an ophthalmologist, who should see the patient within 24 h.

TRAUMATIC MIOSIS AND MYDRIASIS

- Pupillary constriction (miosis), dilation (mydriasis), or triangular defects of the pupillary margin may result from blunt trauma and should be followed by an ophthalmologist.
- Pupillary abnormalities with a corneal abrasion should prompt a search for an intraocular foreign body.

IRIDODIALYSIS

- Blunt trauma can cause a separation of the iris at the ciliary body that creates a lentiform defect (''accessory pupil'') at the limbus. It is often associated with a hyphema. Emergent ophthalmologic consultation is required.

HYPHEMA

- Blood in the anterior chamber can occur spontaneously or following trauma. Rebleeding can occur 3 to 5 days following the initial injury and is associated with a high complication rate.
- Emergency department care includes placement of the patient in an upright position, topical atropine 1% (1 drop), topical prednisolone acetate 1% (1 drop), protection with a Fox shield, and ophthalmologic consultation. If the globe is intact

and intraocular pressure is >30 mmHg, timolol 0.5% (1 drop) and acetazolamide 500 mg PO or IV should be administered. If there is no response, mannitol 1 to 2 mg/kg IV should be added.
- Sickle cell patients tolerate hyphema poorly. Intraocular pressure should be kept at <24 mmHg, as stated earlier; however, the use of acetazolamide in these patients should be avoided.
- If the hyphema involves less than one-third of the anterior chamber, outpatient therapy may be considered after ophthalmologic consultation.

ORBITAL BLOWOUT FRACTURES

- The inferior and medial wall of the orbit may be fractured from blunt trauma. Physical exam signs include evidence of inferior rectus entrapment (diplopia on upward gaze) and subcutaneous emphysema, especially when sneezing or blowing the nose. Plain radiographs may show maxillary sinus clouding, an air-fluid level, or a teardrop-shaped opacity (''teardrop sign'').
- Isolated blowout fractures (with or without entrapment) require referral to a facial surgeon within 3 to 10 days, with concurrent ophthalmology referral.

PENETRATING OCULAR TRAUMA AND RUPTURED GLOBE

- These injuries may present with a teardrop-shaped pupil, bloody chemosis, extrusion of globe contents, hyphema, shallow anterior chamber, or significant reduction in visual acuity. It is especially important to suspect these injuries when a history of high-velocity small projectiles striking the eye is obtained. Fluorescein streaming (Seidel's test) may be present.
- Orbital thin-slice CT scan is the test of choice for identifying intraocular foreign bodies. Magnetic resonance imaging may be used unless the suspected foreign body is ferromagnetic.
- Emergency department care includes avoidance of any manipulation of the eye (e.g., checking intraocular pressure), protection with a Fox shield, tetanus status update, cefazolin 1 g IV therapy, and emergent ophthalmologic consultation.

CHEMICAL OCULAR TRAUMA

- All corrosive burns are managed similarly. Topical anesthetic and a Morgan lens should be used to immediately flush the eye with at least 2 L of normal saline. The irrigation should be continued until the pH is between 6 and 8 by litmus paper. The pH should be rechecked in 10 min to exclude further corrosive material leaching out from other tissues.
- If the cornea and anterior chamber is normal, erythromycin ointment qid should be administered and within 48 h ophthalmologic follow-up should be arranged.
- If a superficial epithelial defect or clouding is present, a topical cycloplegic, 1% cyclopentolate or 5% homatropine (1 drop tid) may be given for comfort. Erythromycin ointment should be instilled qid, tetanus status updated, and these patients referred to ophthalmology within 24 h.

CYANOACRYLATE GLUE REMOVAL

- Cyanoacrylate glue (Super-Glue, Krazy-Glue) readily adheres to the eyelids and corneal surface, but usually causes no permanent damage.
- Glue removal involves moistening the glue with erythromycin ointment and removing any easily removable pieces. Erythromycin ointment then should be applied into eye and eyelids 5 to 6 times a day. The patient should be referred to an ophthalmologist within 24 to 48 h.

ULTRAVIOLET KERATITIS (''WELDER'S FLASH'')

- Superficial punctate keratitis (SPK) can occur from tanning booths, welding flashes, unprotected eclipse viewing, and prolonged sun exposure. Patients present with pain, tearing, photophobia, and foreign-body sensation 6 to 12 h after the exposure.
- Slit-lamp examination with fluorescein staining reveals numerous superficial corneal punctate lesions, usually on the lower half of the cornea.
- Emergency department care is the same as for superficial corneal abrasions.

PAINFUL ACUTE VISUAL REDUCTION

ACUTE ANGLE CLOSURE GLAUCOMA

- Shallow anterior chambers predispose the patient to this condition and may be precipitated by pupillary dilating agents.
- Symptoms include cloudy vision, orbital pain,

headache, and gastrointestinal (GI) symptoms. Signs include ocular injection, corneal haziness, iritis, a minimally reactive or nonreactive middilated pupil, and increased intraocular pressure (40 to 70 mmHg).
- Multiple agents are used simultaneously in order to decrease aqueous production, facilitate aqueous outflow, and directly decrease intraocular pressure. Topical timolol 0.5% directly lowers intraocular pressure and may facilitate the action of pilocarpine; 1 drop should be used in the affected eye immediately. Pilocarpine 2% is a miotic that promotes aqueous outflow; 1 drop q 15 min should be used in the affected eye and 1 drop q 6 h on the contralateral side for prophylaxis. Apraclonidine 0.1% is an α-2 agonist that acts primarily by decreasing aqueous production. One drop should be placed in the affected eye immediately. Carbonic anhydrase inhibitors decrease aqueous formation; acetazolamide 500 mg IV should be used. Hyperosmotic agents also may be initiated. Oral regimens include glycerol 50%, 1 mL/kg, or 220 mL of isosorbide 45%. Alternatively, mannitol 20% (1 to 2 g/kg) may be given IV. All cases require immediate and concurrent ophthalmologic consultation.

OPTIC NEURITIS

- Inflammation of the optic nerve can be caused by infection, demyelination, and autoimmune disorders. It may present with reduction of vision (often with poor color perception), pain during extraocular movement, visual field cuts, and an afferent pupillary defect. Swelling of the optic disc may be seen in anterior optic neuritis.
- Diagnosis can be made with the red desaturation test (after staring at a bright red object with the normal eye only, the object may subsequently appear pink or light red in the affected eye).
- Emergency department care is controversial, and the use of IV steroids should be discussed with an ophthalmologist.

PAINLESS ACUTE VISUAL REDUCTION

CENTRAL RETINAL ARTERY OCCLUSION

- Central retinal artery occlusion may be caused by embolus, thrombosis, giant-cell arteritis, vasculitis, sickle cell disease, and trauma. It is often preceded by amaurosis fugax.
- The vision loss is a painless, graying or blurring of the visual field (''descending nightshade'') and may be complete or partial.
- An afferent pupillary defect is often present, and funduscopy classically reveals a pale fundus with narrowed arterioles with segmented flow (''boxcars'') and a bright red macula (''cherry red spot'').
- Emergency department care includes ocular massage (digital pressure for 15 s, followed by sudden release), topical beta blocker (timolol 0.5% 1 drop), acetazolamide 500 mg IV or PO, administration of 95 : 5 mixture of O_2 and CO_2 (Carbogen) for 10 min q h or paper bag rebreathing, and emergent ophthalmologic consultation.

CENTRAL RETINAL VEIN OCCLUSION

- Thrombosis of the central retinal vein causes painless, rapid monocular vision loss. Funduscopy classically reveals diffuse retinal hemorrhages, cotton wool spots, and optic disc edema. The contralateral optic nerve and fundus is normal.
- Emergency department care may include aspirin 60 to 325 mg qd, and all patients should be referred to an ophthalmologist within 24 h.

GIANT CELL ARTERITIS (TEMPORAL ARTERITIS)

- Giant cell arteritis is a systemic vasculitis that can cause ischemic optic neuropathy. Patients are usually over 50 years of age, female, and often have polymyalgia rheumatica.
- Symptoms and signs include headache, jaw claudication, myalgias, fatigue, fever, anorexia, temporal artery tenderness, and neurologic findings (including transient ischemic attacks and stroke). An afferent pupillary defect is often present. C-reactive protein and sedimentation rate is usually elevated (70 to 110).
- Emergency department care includes IV steroids and ophthalmologic consultation.

OTHER OCULAR EMERGENCIES

RETINAL INJURY

- Detachment of the retina can be acute or delayed. Typical symptoms include a ''flashing sensation'' and a visual field defect. Funduscopy may reveal the lesion. Emergent ophthalmologic consultation is required.

VITREOUS HEMORRHAGE

- Vitreous hemorrhage often presents with the patient perceiving ''floaters'' (clumps of red cells) in the visual field. Patients should avoid platelet inhibitors or straining and should be referred to an ophthalmologist.

EPISCLERITIS

- Inflammation of the junction of the conjunctiva and sclera presents with discomfort, localized hyperemia, and swelling. Emergency department care should be directed by ophthalmologic consultation, which may include topical decongestants, topical steroids, and oral nonsteroidal anti-inflammatory drugs.

UVEITIS AND IRITIS

- Inflammation of the iris, ciliary body, and choroid can be caused by trauma, infection, and autoimmune diseases. It typically presents with blurred vision, orbital aching, photophobia, consensual pain, and redness.
- Diagnosis is made when cell and/or flare is seen in the anterior chamber with the slit lamp.
- Emergency department care includes topical cycloplegics, topical steroids, and ophthalmologic consultation.

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148 EAR, NOSE, AND FACIAL DISORDERS

Burton Bentley II

OTOLOGIC EMERGENCIES

TINNITUS

- Tinnitus is the perception of sound without any external stimulation.
- Ototoxic agents cause 10 percent of tinnitus cases.[1] Commonly implicated drugs include aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics (particularly aminoglycosides), and chemotherapeutic agents.

HEARING LOSS

- Conductive deficits occur when sound waves are not conducted to the inner ear. Etiologies may include cerumen impaction, foreign body obstruction, external otitis, tympanic membrane (TM) perforation, tympanosclerosis, disruption of the ossicular chain, or middle ear fluid.
- Sensorineural deficits result from disruption of the neural pathway. Etiologies include viral neuronitis, acoustic neuroma, Ménière's disease, and autoimmune disorders.
- To perform the Weber's test, a tuning fork should be held on the central forehead while the patient notes which ear perceives a louder sound. In sensorineural hearing loss, sound lateralizes to the normal ear; in conductive loss, sound lateralizes to the abnormal ear.
- To perform the Rinne's test, a tuning fork should be held on the mastoid until the sound is inaudible. At this point, the tuning fork should be held near the ear canal where it should again be audible to a normal patient, but inaudible if there is a conductive deficit.

- Bilateral hearing loss (normal Weber and Rinne with decreased hearing) suggests noise or ototoxic exposure. Drugs causing ototoxicity include antibiotics (aminoglycosides, erythromycin, and vancomycin), NSAIDs, antimalarials, antineoplastics, and loop diuretics (furosemide and ethacrynic acid).

OTITIS EXTERNA

- Otitis externa (OE) and malignant otitis externa (MOE) represent the extremes of a spectrum progressing from dermatitis of the external auditory canal (EAC), to cellulitis, chondritis, and finally to osteomyelitis of the temporal bone and skull base.
- Otitis externa occurs frequently in swimmers; MOE occurs primarily in diabetics and immunocompromised patients. Most cases of OE are caused by *Pseudomonas aeruginosa* or *Staphylococcus aureus*;[2] 10 percent of cases are fungal. Malignant otitis externa should be suspected in any elderly, diabetic, or immunocompromised patient with OE, any patient with OE refractory to 2 to 3 weeks of treatment, or any patient with OE and pain out of proportion to the exam.
- Patients with OE have severe ear pain worsened by movement of the pinna or tragus. The EAC may be erythematous, suppurative, and clogged with debris. In MOE, the disease spreads to involve the periauricular structures. Malignant otitis externa also may be suggested by granulation tissue on the floor of the EAC. As MOE progresses, patients may develop trismus, fever, sepsis, cranial nerve palsies, meningitis, or brain abscess. Osteomyelitis in MOE may be confirmed by a nuclear bone scan or radiographic imaging.
- Otitis externa is treated with an antibiotic solution such as hydrocortisone and neomycin mixed with polymyxin B (Cortisporin Otic); if the TM is perforated or not visualized, the suspension form should be used. If the TM is not visualized, the patient should be treated empirically with additional oral antibiotics for possible concurrent otitis media (OM). Patients with MOE require hospital admission and treatment with an antipseudomonal antibiotic.

OTITIS MEDIA

- The most common bacterial pathogens in OM are *Streptococcus pneumoniae, Haemophilus influenzae,* and *Mycoplasma catarrhalis.* Signs and symptoms of OM include otalgia, fever, and an abnormal TM appearance; poor TM mobility on insufflation is the most sensitive sign of OM.
- Complications of OM may include TM perforation, cholesteatoma, hearing loss, mastoiditis, and brain abscess. Meningitis is the most common intracranial complication.
- In OM with effusion, an air-fluid level or bubbles may be seen.
- Bullous myringitis is a particularly painful variant of OM that may be caused by *Mycoplasma.*
- Patients with OM are treated with a 10-day course of antibiotics. First-line agents include amoxicillin, trimethoprim-sulfamethoxazole, or erythromycin-sulfisoxazole. Macrolides effective against *Mycoplasma* are the best choice for patients with bullous myringitis. Treatment failure after 2 to 3 days requires broader β-lactamase coverage with antibiotics such as amoxicillin-clavulanate or cefaclor. Otitis media with effusion is treated in the same manner, though additional prednisone may be beneficial.[3]

MASTOIDITIS AND LATERAL SINUS THROMBOSIS

- Mastoiditis is a serious complication of inadequately treated OM and occurs most often in the pediatric age group. Frequently, there is a history of OM, antibiotic use, persistent otalgia, or otorrhea. Clinical deterioration in the patient with OM should prompt concern for mastoiditis.
- Signs and symptoms of mastoiditis include mastoid tenderness and erythema, loss of the postauricular crease, inferolateral displacement of the pinna, local fluctuance, and TM abnormalities. A complete blood cell count may show a leukocytosis, and plain radiographs occasionally reveal mastoid opacification. Computed tomography (CT) imaging helps to confirm the diagnosis and to rule out other intracranial processes.
- Broad-spectrum antibiotics (e.g., cefuroxime[4]) and ear, nose, and throat (ENT) admission for surgical drainage are urgently required. Complications of mastoiditis may include facial palsies, extension of the abscess into the neck, meningitis, intracranial abscess, or septic thrombophlebitis.
- Lateral sinus thrombosis (LST) is a rare complication of OM that arises from extension of the infection into the lateral and sigmoid sinuses. Headache is the most common symptom, though some patients may have papilledema, vertigo, or a sixth nerve palsy.[5] Ear, nose, and throat consultation,

magnetic resonance imaging, and immediate antibiotic therapy are required. A combination of naficillin, ceftriaxone, and metronidazole is recommended.[5]

TRAUMA

- Improper treatment of ear hematomas may result in cartilage necrosis with a cosmetic deformity known as ''cauliflower ear.'' Immediate incision, drainage, and compressive dressings will relieve the hematoma and prevent reaccumulation.[6,7]

FOREIGN BODIES IN THE EAR

- Options for removal depend on the size and composition of the foreign body and may include irrigation, microforceps, hooked probes, suction-cup catheters, and cyanoacrylate glue on a probe tip.
- Live insects are particularly distressing to the patient and should be immediately immobilized with 2% lidocaine instilled into the ear canal.

TYMPANIC MEMBRANE PERFORATIONS

- Tympanic membrane perforation results from trauma, infection, or lightning and may cause slight pain and hearing loss. Vertigo and deafness indicate injury to the ossicles, labyrinth, or temporal bone and require urgent consultation. Ninety percent of TM perforations heal spontaneously. Antibiotics are required when there is coexistent OM, but are not useful in uncomplicated perforations.

OTHER FACIAL EMERGENCIES

MASTICATOR SPACE ABSCESS

- The masticator space consists of four contiguous spaces bounded by the muscles of mastication. Abscesses in this space often extend from infections in the buccal, submandibular, and sublingual areas.
- Signs and symptoms may include trismus, facial swelling, pain, erythema, fever, dysphagia, or sepsis. Masticator abscesses must be distinguished from parotitis. With simple parotitis, the patient generally has no trismus and the pain has a cyclical relation to eating.[8,9]
- Stable patients require antibiotics (e.g., penicillin, erythromycin, or clindamycin) and immediate follow-up. Patients with advanced symptoms require operative drainage.

LUDWIG'S ANGINA

- Ludwig's angina is an extensive bilateral cellulitis of the submandibular space that may evolve from infected lower molars. Signs and symptoms include fever, painful edema, restricted neck motion, drooling, trismus, dysphagia, dysphonia, and displacement of the tongue in a posterior and superior direction. Severe cases may have progressive respiratory distress with complete airway obstruction, involvement of the carotid artery and jugular vein, and mediastinitis.
- Direct laryngoscopy can provoke laryngospasm and should be avoided. Lateral neck radiographs are useful and frequently show airway narrowing, soft tissue swelling, and subcutaneous emphysema. Computed tomography scanning is diagnostic, though it is often impossible to perform in the distressed patient.
- Treatment consists of airway management, parenteral antibiotic, and operative drainage by an ENT specialist.

SALIVARY GLAND PROBLEMS

SIALOADENITIS

- Sialoadenitis refers to inflammation of the parotid, sublingual, or submandibular salivary glands.
- Mumps is one cause of painful parotid swelling in the pediatric age group. Symptoms include fever and malaise with parotid pain and swelling. Bilateral parotitis occurs in 70 percent of patients,[10] though there is no discharge from Stenson's duct. The diagnosis is clinical, and the treatment is symptomatic.
- Suppurative parotiditis sometimes occurs in people with a decreased flow of saliva. The parotid is swollen and tender with pus expressed at Stenson's duct. Twenty-five percent of cases are bilateral.[11] Progression is heralded by fever, trismus, and involvement of the face and neck. The diagnosis is strictly clinical.[10] Treatment consists of hydration, massage, local heat, sialogogues (e.g., lemon drops), and β-lactamase resistant antibiotics.

SIALOLITHIASIS

- Salivary calculi (i.e., sialoliths) present with unilateral pain and swelling, most commonly involving the submandibular glands. The stone is often palpable and visible on intraoral radiographs. Treatment consists of analgesics and sialogogues; antibiotics are given if an infection is present. Easily located calculi may be milked from the duct; all others require ENT referral.

NASAL EMERGENCIES AND SINUSITIS

EPISTAXIS

- Anterior epistaxis arises from the anterior nasal septum where the site of hemorrhage is often easily visualized. Posterior epistaxis arises from more posterior locations and usually requires endoscopic instruments for localization. Posterior epistaxis is suspected when an anterior source is not identified, bleeding occurs from both nares, or blood is seen draining into the posterior pharynx after anterior sources have been controlled.
- Anterior epistaxis may respond to simple direct pressure. Other options include topical vasoconstrictors, cautery, and nasal packing.
- Posterior epistaxis is treated with either a dehydrated posterior sponge pack or with a commercial balloon tamponade device. Patients with posterior packs require ENT consultation for possible hospital admission.
- All patients with nasal packing require antibiotic prophylaxis with antistaphylococcal medications to prevent sinusitis and toxic shock syndrome.

NASAL FRACTURES

- A nasal fracture is a clinical diagnosis that should be suspected in all cases of facial trauma. Suggestive findings include swelling, tenderness, crepitance, gross deformity, periorbital ecchymosis, epistaxis, and rhinorrhea. Radiographs are usually not indicted in the emergency department (ED), though they may be obtained at the follow-up appointment. Serious associated injuries must be ruled out.
- A simple, nondisplaced nasal fracture only requires supportive care. Ear, nose, and throat referral is not mandatory unless there is nasal congestion or cosmetic deformity after the swelling diminishes in 2 to 5 days.
- A septal hematoma appears as a collection of blood beneath the perichondrium of the nasal septum. If left untreated, a septal hematoma may cause abscess formation and avascular necrosis of the nasal septum. The treatment is local incision and drainage with placement of an anterior nasal pack.
- Fracture of the cribriform plate may result in cerebrospinal fluid (CSF) rhinorrhea and should be suspected in any patient with clear nasal drainage following facial trauma, even if the trauma occurred days to weeks earlier. Cerebrospinal fluid leakage may be suggested by bedside glucose reagent strip testing (a glucose level >30 mg/dL suggests CSF) or a positive ''halo'' test (a clear ''halo'' surrounds a central blood stain when a drop of bloody CSF is placed on a piece of filter paper). If a cribriform plate injury is suspected, then a CT scan and immediate neurosurgical consultation must be obtained.

NASAL FOREIGN BODIES

- Nasal foreign bodies should be suspected in any case of unilateral nasal obstruction, foul rhinorrhea, or persistent unilateral epistaxis.
- Removal may be facilitated by topical vasoconstrictor and anesthetic agents. Tools and techniques for removal include suction catheters, forceps, hooked probes, balloon-tipped catheters, and positive pressure applied by a puff of air to the patient's mouth.[12]

SINUSITIS

- Maxillary sinusitis presents with pain in the infraorbital area whereas frontal sinusitis causes pain in the supraorbital and lower forehead regions. Ethmoid sinusitis, which is especially serious in children because of its tendency to spread to the central nervous system, may produce a dull, aching sensation in the retroorbital area. Sphenoid sinusitis is uncommon and has vague signs and symptoms. Chronic sinusitis results in local discomfort and persistent, purulent exudate.
- Viral upper respiratory infections and allergic rhinitis are the most common precipitating factors.[13]
- The signs and symptoms of sinusitis are neither sensitive nor specific. They may include erythema, warmth, tenderness, swollen nasal mucosa, purulent discharge, and diminished transillumination. Radiographs may show sinus opacification, air-

fluid levels, or mucosal thickening of at least 6 mm, but they are generally not required in the ED.

- Treatment of sinusitis includes a brief course of topical nasal decongestants and a 10- to 21-day course of antibiotics (e.g., amoxicillin-clavulanate, cefuroxime, or trimethoprim-sulfamethoxazole).

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149 ORAL AND DENTAL EMERGENCIES

Burton Bentley II

ORAL PAIN

- Eruption of the primary teeth in infants and children may be associated with pain, low-grade fever, diarrhea, and refusal to eat.
- The most common cause of toothache is a carious tooth. Fluctuant oral abscesses from infected teeth require local incision and drainage, oral antibiotics, frequent saline rinses, and close follow-up.
- Periosteitis causes pain within 24 h of a tooth extraction; it responds well to analgesics.
- Alveolar osteitis (''dry socket'') causes severe pain and a foul odor 2 to 3 days after dental extraction. Treatment consists of socket irrigation, packing, antibiotics, and close follow-up.
- A periodontal abscess results from plaque and debris trapped between the tooth and gingiva. Most cases resolve with oral antibiotics, analgesics, and saline irrigation. Larger abscesses may require incision and drainage.
- Acute necrotizing ulcerative gingivitis (ANUG, or ''trench mouth'') is the only periodontal disease in which bacteria invade nonnecrotic tissue. It occurs mainly in HIV-positive adults, emotionally stressed patients, malnourished children, and patients with a prior history of ANUG. Signs and symptoms include regional lymphadenopathy, inflamed and tender gingiva, pseudomembrane formation, blunted and ulcerated interdental papillae, halitosis, fever, and malaise. Treatment requires oral metronidazole (Flagyl) and chlorhexidine oral rinses.[1]

SOFT TISSUE LESIONS OF THE ORAL CAVITY

- Oral candidiasis appears as white, curdlike plaques responsive to nystatin suspension or fluconazole. Risk factors include extremes of age, immunocompromised states, intraoral prosthetics, antibiotic use, and malnutrition.
- Aphthous stomatitis is a common pattern of mucosal ulceration triggered by cell-mediated immunity. The painful lesions resolve completely when treated with topical steroids.
- Herpes gingivostomatitis causes acute painful ulcerations of the gingiva and mucosal surfaces. A

prodrome of fever, lymphadenopathy, and tingling precedes the eruption of numerous vesicles, forming ulcerative lesions. The treatment is palliative, though early use of antiviral medications (e.g., acyclovir or Valacyclovir) may speed healing.
- Herpangina causes a self-limited illness of acute fever, sore throat, headache, and malaise followed by a diffuse vesicular eruption. The tiny vesicles rupture leaving ulcers on the soft palate, uvula, and tonsillar pillars; the buccal mucosa, tongue, and gingiva are spared.
- Hand, foot, and mouth disease is caused by Coxsackievirus infection and appears as vesicles on the soft palate, gingiva, tongue, and buccal mucosa; lesions may also appear on the fingers, toes, palms, soles, and buttocks. The vesicles rupture leaving painful ulcers surrounded by red halos. This self-limited exanthem lasts 5 to 8 days.

LESIONS OF THE TONGUE

- Erythema migrans (''geographic tongue'') is a common benign finding consisting of multiple, sharply circumscribed zones of erythema found predominantly on the tip and lateral borders of the tongue. The lesions are typically asymptomatic, but may cause a burning sensation. Symptomatic lesions respond to topical fluocinonide gel applied several times daily.[2]
- Black hairy tongue is a brown discoloration of unknown etiology that affects the dorsum of the tongue. Treatment consists of frequent tongue brushing and avoidance of tobacco, strong mouthwashes, and antibiotics. Symptomatic resolution is usually spontaneous.[3]
- Strawberry tongue is associated with *Streptococcus pyrogenes* infection as part of scarlet fever. The tongue appears white-coated with hyperemic papillae. Antibiotic treatment of the underlying infection leads to prompt resolution.

OROFACIAL INJURIES

DENTAL FRACTURES

- A painless dental injury may suggest neurovascular disruption and forewarn of tooth loss.
- Ellis class 1 fractures involve the enamel of the tooth. These injuries may be smoothed with an emery board or referred to a dentist for cosmetic repair.[4–6]
- Ellis class 2 fractures comprise 70 percent of tooth fractures. This fracture exposes the underlying pale yellow dentin and provokes thermal and air sensitivity. Incorrect management, particularly in children, increases the chance of infecting the dental pulp. The exposed dentin must be dried and covered with either a glass ionomer cement or calcium hydroxide paste (Dycal); dental follow-up is sought within 24 h. In patients <12 years, a visible blush of pulp under the dentin indicates that the pulp is at risk and should be treated as an Ellis class 3 fracture (see later).[4–6]
- Ellis class 3 fractures expose the dental pulp and are true dental emergencies. They are identified by the red blush of dentin or a drop of frank blood. If a dentist or maxillofacial surgeon is not immediately available, the injury should be treated as an Ellis class 2 and the patient should be sent for immediate follow-up. Topical anesthetics are contraindicated since they may cause sterile abscesses.[4–6]

SUBLUXED, INTRUDED, AND AVULSED TEETH

- Dental trauma may result in tooth loosening, termed *subluxation.* Blood in the gingival crevice is a subtle indicator of trauma. Minimally subluxed teeth heal well in 1 to 2 weeks if the patient maintains a soft diet. Grossly mobile teeth require stabilization by a dentist.
- Dental intrusion occurs when a tooth is forced below the gingiva. Intruded primary teeth are allowed to erupt for 6 weeks before considering repositioning. Intruded permanent teeth require surgical repositioning. Failure to diagnose dental intrusion may result in infection and cosmetic deformity.
- Complete tooth avulsion is a true emergency with a percentage point for successful reimplantation lost with each passing minute. If the missing tooth is not located, consider radiographs to rule out dental intrusion or aspiration. Primary (deciduous) teeth are not replaced since they may ankylose and cause facial deformity. Permanent teeth that have been avulsed for less than 3 h must be immediately reimplanted. Avulsed teeth in transported patients should be gently replaced in the socket. Other options, in descending order of preference, include placing the tooth in Hank's solution, saliva within the patient's mouth, a glass of milk, or wet gauze. Once in the emergency department, the tooth should be gently rinsed

without scrubbing and replaced into the irrigated socket.[7,8]

ORAL LACERATIONS

- Intraoral mucosal lacerations heal poorly and become infected if they are gaping open, though smaller lacerations (<1 cm) may be left alone. Treatment consists of debridement, irrigation, and closure with 4-0 chromic sutures.
- Laceration of the maxillary frenulum does not usually require repair.
- Lacerations of the lingual frenulum may require repair because of its vascularity.
- Tongue lacerations >1 cm should be closed with 4-0 silk or chromic sutures. Care must be taken to carefully approximate the wound edges in order to avoid cleft formation.
- Palate lacerations and puncture wounds may involve the hard or soft palate. Occult injuries to the retropharynx and neighboring vascular structures should be considered and ruled out. Gaping edges require loose approximation though most wounds may be left open to drain.
- Lip lacerations require meticulous closure. The subcutaneous tissues must be closed with absorbable sutures to reduce wound tension while the surface is closed with a 6-0 monofilament material. When the vermilion border has been lacerated, the first surface suture must be meticulously placed to align its edges, since failure to do so will lead to a noticeable cosmetic deformity.

HEMORRHAGE SECONDARY TO DENTAL EXTRACTION AND SURGERY

- Bleeding following dental extraction is usually controlled by direct pressure applied by biting on gauze. Negative pressure from smoking, spitting, and use of straws will increase the amount of hemorrhage.
- If bleeding persists, the socket should be suctioned free of clots and direct pressure should be tried again. Other hemostatic options include biting on a used tea bag, local infiltration of 2% lidocaine with epinephrine, and Gel-Foam packing. Bleeding following periodontal surgery requires immediate consultation with the periodontist since incorrect placement of periodontal packs can result in treatment failure.

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150 NECK AND UPPER AIRWAY DISORDERS

William R. Dennis, Jr.

PHARYNGITIS

- Infection of the pharynx may be caused by bacteria, viruses, or fungi. The most common bacteria causing pharyngitis are *Streptococcus, Mycoplasma, Chlamydia, Neisseria,* and *Corynebacterium.*
- Rhinovirus and adenovirus are the most common causes of pharyngitis.
- Group A beta-hemolytic streptococcus (GABHS) infections account for 15 percent of cases and may result in the nonsuppurative sequelae of acute rheumatic fever and poststreptococcal glomerulonephritis.[1] GABHS has its peak occurrence in the late winter and early spring.[2]
- Classic symptoms of GABHS infection include sore throat, painful swallowing, chills, fever, headache, nausea, vomiting, and abdominal pain.
- On physical examination, signs of GABHS infection demonstrate erythematous tonsils, pharyn-

geal exudate, and tender anterior cervical lymph nodes.
- The rapid antigen test or a throat culture may be used to guide therapy; however, empiric antibiotic therapy may be commenced when there is a high clinical suspicion of GABHS. Penicillin is the first-line drug of choice, while cephalosporins, macrolides, and clindamycin are appropriate alternatives.
- Pharyngitis can lead to suppurative complications, including cervical lymphadenitis, peritonsillar abscess, retropharyngeal abscess, sinusitis, and otitis media.

EPIGLOTTITIS

- Epiglottitis is an acute, life-threatening supraglottic infection occurring in all age groups but is traditionally reported to affect children between the ages of 2 and 8 years.[3]
- Epiglottitis can be caused by bacteria, fungi, and viruses. *Haemophilus influenzae* type b (Hib) is the most common cause. Since the introduction of the Hib vaccine, the incidence in children has been declining while the incidence in adults has been increasing.[4,5]
- Pediatric epiglottitis progresses over a 12- to 24-h period and may include fever, drooling, anxiety, sore throat, dysphagia, toxic appearance, and/or respiratory distress. Adult epiglottitis often presents with hours to days of dysphagia and throat pain out of proportion to the clinical examination findings.
- Movement of the trachea and thyroid cartilage is often quite painful and is a marker for supraglottic infection.
- In unstable patients, especially children, all diagnostic procedures are deferred until the patient is in the operating room with an otolaryngologist. Cooperative, stable adult patients should undergo indirect laryngoscopy.
- A soft tissue lateral neck radiograph may reveal an edematous epiglottis (''thumbprint'' sign) with ballooning of the hypopharynx and loss of the vallecula.[6]
- Treatment consists of oxygen, aggressive airway management, and antibiotics. Current antibiotic recommendations include cefuroxime, cefotaxime, or ceftriaxone as first-line drugs.[7] Aztreonam or chloramphenicol can be used in penicillin-allergic patients.
- The stable pediatric patient is treated with endotracheal intubation in the operating room. Stable adults can be treated in the intensive care unit without intubation. An endotracheal tube 0.5 to 1.0 smaller than standard should be used. If endotracheal intubation fails, an airway should be secured using cricothyrotomy in patients over 8 years of age and needle cricothyrotomy in those under 8 years.

PERITONSILLAR ABSCESS

- Peritonsillar abscess (PTA) is the most frequently occurring deep-space infection of the head and neck.[8,9] PTA is often preceded by a throat infection.
- Presenting symptoms may include fever, sore throat, drooling, muffled voice, trismus, dysphagia, otalgia, and foul breath. Classic physical examination findings include a unilaterally enlarged tonsil with swelling of the anterior tonsillar pillar and contralateral deviation of the uvula.
- The differential diagnosis of PTA includes cellulitis, mononucleosis, herpes simplex tonsillitis, retropharyngeal abscess, neoplasm, foreign body, and internal carotid artery aneurysm.
- In stable patients, needle aspiration of the PTA is both diagnostic and therapeutic and provides resolution of trismus and odynophagia.
- A contrast computed tomography (CT) scan of the neck is recommended when the results of needle aspiration are negative or retropharyngeal or parapharyngeal space process is suspected.
- Patients with successful drainage of PTA may be discharged home on broad-spectrum antibiotics (antistaphylococcal) such as amoxicillin-clavulanate. Otherwise, they must be taken to the operating room for adequate drainage and admitted for parenteral antibiotics such as ampicillin-sulbactam, clindamycin, or cefotaxime plus metronidazole.
- Complications of PTA include airway obstruction, rupture of the abscess with aspiration of contents, thrombophlebitis, ulceration of the large submaxillary arteries, epiglottitis, septicemia, endocarditis, retropharyngeal abscess, and mediastinitis.

RETROPHARYNGEAL ABSCESS

- Retropharyngeal abscess is an infection of the deep neck spaces resulting from suppuration and necrosis of the lymph nodes in those spaces. The retropharyngeal space is a connective tissue pocket that extends from the base of the skull to the level of the tracheal bifurcation. The two paramedial lymph nodes—which drain the naso-

pharynx, adenoids, and posterior nasal sinuses—are found here.
- The majority of cases occur in children less than 5 years of age. One-third of cases occur in children less than 6 months of age.[10] There is a higher incidence in children because of the prominence of several lymph nodes in the space. By 3 to 4 years of age, most of these lymph nodes atrophy and are no longer functional.[10]
- Presenting symptoms include fever, odynophagia, neck swelling, drooling, torticollis, meningismus, and stridor.
- The lateral neck radiograph frequently demonstrates prevertebral soft tissue swelling that exceeds one-half the width of the adjacent vertebral body. CT scan with contrast is useful in differentiating cellulitis from abscess and in defining the extent of the infection.
- Treatment consists of meticulous airway management, emergent otolaryngology consultation for operative drainage, and IV antibiotics (clindamycin combined with aminoglycoside, or penicillinase-resistant penicillin combined with a third-generation cephalosporin and metronidazole).[11]
- Complications include spread of infection to the mediastinum and upper airway asphyxia from direct pressure or sudden rupture of the abscess.[12]

PARAPHARYNGEAL ABSCESS

- The parapharyngeal space extends lateral to the pharynx from the base of the skull to the hyoid. Abscesses in this area may result from local infection, trauma, or dental procedures.
- Presenting complaints may include fever, pain on neck movement, sore throat, dysphagia, or drooling.
- Physical examination usually demonstrates cervical lymphadenopathy, pharyngitis, torticollis, and a bulging pharyngeal wall. Lateral neck radiographs may show retropharyngeal swelling, and CT scanning with contrast helps to confirm involvement of the parapharyngeal area.
- All patients require meticulous airway management, emergent otolaryngology consultation for drainage, and broad-spectrum IV antibiotics (ampicillin-sulbactam).

ACUTE UPPER AIRWAY OBSTRUCTION

- Airway foreign bodies (FBs) may present in a straightforward manner, or they may have an insidious presentation, such as progressive stridor or recurrent pneumonia.
- The most common FB associated with children is the coin, with food (peanuts and popcorn) a close second.[13] The most common FBs associated with adults are fishbones, dentures, meat, and meat bones.[14]
- Upper airway FBs may cause stridor and odynophagia with subsequent respiratory distress. Lower airway FBs, commonly in the right bronchial tree, may present as cough, wheezing, dyspnea, pneumonia, or respiratory distress.
- Stable patients should have direct laryngoscopy performed to look for the object. Soft tissue radiographs, including chest radiographs (posteroanterior and lateral) and endoscopy with bronchoscopy are other diagnostic adjuncts. Atelectasis, hyperinflation, and aerophagia are radiographic findings consistent with foreign-body aspiration.[15]
- Extreme care must be taken to avoid destabilizing a partial obstruction. Airway management must be a priority with surgical airway equipment immediately available. A double-lumen endotracheal tube may be needed to ventilate the unaffected lung.
- All suspected FB ingestions require immediate bronchoscopy.

LARYNGEAL TRAUMA

- Laryngeal injuries may result from blunt or penetrating trauma and are associated with significant morbidity and mortality. Patients may present with hoarseness, hemoptysis, dyspnea, dysphagia, aphonia, stridor, or respiratory distress. The earliest finding may be the subtle loss of voice.
- Physical examination signs include laryngeal swelling, tenderness, anterior neck contusion, altered laryngeal contour, tracheal deviation, or subcutaneous emphysema.
- Initial minor laryngeal injuries may progress due to edema and expanding hematomas.
- Emergent otolaryngology consultation is warranted for all patients with signs and symptoms consistent with laryngeal injury. Stable patients may be given humidified oxygen while waiting for consultation. The unstable patient requires emergent tracheostomy. This is performed with a vertical midline skin incision with passage of the tracheostomy tube at a level lower than usual (fourth or fifth tracheal ring) in order to avoid further damage to the larynx and surrounding structures. Retrograde intubation should not be attempted.
- Orotracheal intubation is controversial, as it may

cause iatrogenic injury as well as the loss of an already precarious airway.[16–18]

- Antibiotics (ampicillin-sulbactam) should be given when there is violation of the mucosa or subcutaneous emphysema.
- The diagnostic test of choice is flexible fiberoptic nasopharyngolaryngoscopy, but CT scanning may also be utilized. In the case of massive trauma, immediate tracheostomy and neck exploration will be required.

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Section 19
DISORDERS OF THE SKIN

151 DERMATOLOGIC EMERGENCIES

James Hassen, Jr.

EXFOLIATIVE DERMATITIS

- Exfoliative dermatitis is a cutaneous reaction to a drug, chemical, or underlying disease state where most or all of the skin surface is involved with a scaling erythema leading subsequently to exfoliation.
- Males are affected twice as often as females, and most patients are over the age of 40.
- Acute forms of exfoliative dermatitis are due to medications, contrast allergens, or cancer.
- Chronic forms of exfoliative dermatitis are usually due to underlying cutaneous disease.
- Patients usually complain of pain, pruritis, fever, and chills.
- Physical examination shows generalized warmth, erythroderma, scaling, and desiccation and exfoliation of the skin.
- The process usually begins on the face and upper trunk with progression to other skin surfaces.
- Widespread cutaneous vasodilation may result in high-output congestive heart failure, while disruption of the epidermis may result in significant electrolyte imbalances.
- Diagnosis of exfoliative dermatitis is confirmed by skin biopsy.
- Treatment includes securing the ABCs, for severe cases, and oral antihistamines, steroids, oatmeal baths, and bland lotions for dermatologic treatment.

ERYTHEMA MULTIFORME

- Erythema multiforme (EM) is an acute inflammatory skin disease that ranges from a mild papular eruption (EM minor) to a severe vesiculobullous form with mucous membrane involvement and systemic toxicity (Stevens-Johnson syndrome).
- Erythema multiforme is usually due to infection, drugs, malignancy, rheumatologic disorders, or pregnancy.
- The highest incidence affects adults (20 to 40 years of age), with males affected twice as often as females.
- Symptoms include malaise, arthralgias, fever, burning sensation, and pruritus.
- The target lesion is highly characteristic of EM. The erythematous papules appear symmetrically on the dorsum of the hands, feet, and the extensor surfaces of the extremities.
- Ocular involvement occurs in 10 percent of patients with EM minor and in 75 percent of patients with Stevens-Johnson syndrome.
- Treatment for EM minor includes topical steroids, analgesics, and antihistamines. Diphenhydramine and lidocaine rinses are useful for oral lesions. Burrow's solution is useful for blistered regions.
- For patients with extensive disease or systemic involvement, management of potential fluid, electrolyte, infectious, and thermoregulatory problems should be initiated. Patients should be admitted into an intensive care unit setting.

TOXIC EPIDERMAL NECROLYSIS

- Toxic epidermal necrolysis (TEN) is an explosive dermatosis characterized by tender

erythema, bullae formation, and subsequent exfoliation.
- The most common cause of TEN is medications; other etiologies include chemicals, infections, or immunologic factors.
- Sulfa-based drugs, penicillin, anticonvulsants, and nonsteroidal anti-inflammatory drugs (NSAIDs) are the most frequent medication triggers for TEN.
- Patients complain of malaise, myalgias, arthralgias, fever, and painful skin or upper respiratory symptoms.
- Physical examination reveals diffuse, tender erythema; mucous membrane involvement; areas of denuded skin with adjacent large bullous lesions; a positive Nikolsky's sign; and systemic toxicity.
- Diagnosis of TEN is confirmed by skin biopsy.
- Management of patients with TEN is best performed in a critical care setting such as a burn unit. Attention to adequate cardiorespiratory function is essential; correction of fluid, electrolyte, and infectious complications are early treatment considerations. Immediate dermatologic consultation is required.

TOXIC INFECTIOUS ERYTHEMAS

- Toxic Infectious Erythemas include toxic shock syndrome (TSS), streptococcal toxic shock syndrome (STSS), and staphylococcal scalded-skin syndrome (SSSS).
- The dermatologic hallmark of TSS is a nonpruritic, blanching macular erythroderma.
- The diagnosis of TSS requires the presence of all 4 major criteria and 3 or more indications of multisystem involvement.

  Major Criteria
  1. Fever: Temperature >102° F (38.9°C)
  2. Rash: Erythroderma (localized or diffuse) followed by peripheral desquamation
  3. Mucous membrane: Hyperemia of oral and vaginal mucosa and of conjunctiva
  4. Hypotension: History of dizziness, orthostatic changes, or hypotension

  Multisystem Manifestations
  1. Central nervous system: Altered mentation without focal neurologic signs
  2. Cardiovascular: Distributive shock; congestive heart failure; dysrhythmias
  3. Pulmonary: Adult respiratory distress syndrome
  4. Gastrointestinal: Vomiting and diarrhea
  5. Hepatic: Elevations in bilirubin, alkaline phosphatase, and the transaminases
  6. Renal: Blood urea nitrogen and/or creatinine elevations; abnormal urinary sediment; oliguria
  7. Hematologic: Thrombocytopenia or thrombocytosis; anemia; leukopenia or leukocytosis
  8. Musculoskeletal: Myalgias; arthralgias, rhabdomyolysis
  9. Metabolic: Hypocalcemia; hypophosphatemia
  10. Absence of other etiologic agent
- Streptococcal toxic shock syndrome presents with fever, hypotension, and skin infections (cellulitis, myositis, and fasciitis).
- The causative agent of STSS is *Streptococcus pyogenes.*
- Staphylococcal scalded-skin syndrome is divided into three stages: (a) initial and erythroderma, (b) exfoliative, and (c) desquamation and recovery.
- In SSSS, exotoxins released by bacteria cause acantholysis and intraepidermal cleavage of the skin.
- Staphylococcal scalded-skin syndrome occurs primarily in infants, young children, and those who are immunocompromised.
- Management of patients with TSS and STSS is dictated by the severity of their illness. As in any patient presenting in extremis, the emergency physician must perform a rapid, thorough review of the ABCs, ensuring a stable airway and ventilatory status, as well as an adequate hemodynamic state. The next step is to identify and remove any source of infection and administer broad-spectrum antibiotics.
- Management of the patient with SSSS includes fluid resuscitation and correction of electrolyte abnormalities, as well as identification and treatment of the source of the toxigenic *Staphylococcus* with the appropriate anti-staphylococcal antibiotic, preferably a penicillinase-resistant penicillin.

BULLOUS DISEASES

- Pemphigus vulgaris (PV) is a generalized mucocutaneous autoimmune blistering eruption characterized by intraepidermal acantholytic blistering.
- The primary lesions of PV are vesicles or bullae that vary in diameter from <1 cm to several centimeters, commonly first affecting the head, trunk, and mucous membranes. Nikolsky's sign is positive in PV. Mucous membranes are affected in 95 percent of PV patients.
- Bullous pemphigoid (BP) is characterized by tense blisters (up to 10 cm in diameter) arising from

either normal skin or from erythematous or urticarial plaques; frequent sites of involvement include intertriginous and flexural areas. The mucous membranes are involved in 40 percent of patients with BP.
- Bullous pemphigoid is a disease that usually affects the elderly, with the average age being 70.
- Diagnosis of both PV and BP is confirmed by skin biopsy.
- The cutaneous surfaces involved with blisters or eroded areas should be treated as burns with the application of *silver sulfadiazine cream* or antibiotic ointments with clean dressings; pain originating from oral lesions may be partially relieved with soothing mouth washes (1:1 mixture of diphenhydramine elixir with Mylanta) or with viscous lidocaine.
- Initial care of the patient with PV should be performed as an inpatient with early dermatologic consultation.

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152 OTHER DERMATOLOGIC DISORDERS

James Hassen, Jr.

PHOTOSENSITIVITY

- Patients with sunburn may present with minimal discomfort or in extreme pain with extensive blistering. A tender, warm erythema is seen in sun-exposed areas; vesiculation may result, representing a second-degree burn injury.
- Diagnosis is suspected in a patient who has been outdoors with significant ultraviolet (UV) light exposure. Sunburns are treated symptomatically with oral analgesics and burn wound care.
- Exogenous photosensitivity results from either the topical application or the ingestion of an agent that increases the skin's sensitivity to UV light. The topical photosensitizers usually result in a cutaneous eruption at the site of application once UV light is applied.
- Furocoumarins—lime juice, various fragrances, figs, celery, and parsnips—when topically applied are the most common group of agents causing photoeruptions; other topical photosensitizers include PABA esters and topical psoralens.
- The exogenous photoeruption is similar to a severe sunburn reaction, often with blistering. A linear appearance of the rash suggests an externally applied substance. The diagnosis is based on identifying the offending agent.
- Initial management is similar to that of the sunburn reaction, including the avoidance of the sun until the eruption has cleared. The causative agent should be discontinued.

CONTACT DERMATITIS

- Contact dermatitis may be a primary irritant reaction or an allergy-mediated event. Agents capable

of causing an aerosolized reaction include rhus (poison ivy and oak). Examples of directly applied agents include nickel, nail polishes, toothpaste, preservatives in cosmetics, contact lens solutions, and hair care products.
- Allergic contact dermatitis resulting from an aerosolized allergen presents with erythema or scaling, at times accompanied by blistering. The involvement is diffuse, with both upper and lower eyelids affected.
- Direct application of the allergen produces similar findings on the most sensitive skin areas, such as the eyelids.
- For therapy, corticosteroids (topical or oral depending upon the severity) are often required. Only low-potency topical corticosteroids (hydrocortisone 2.5%) should be used on the face. Often, extensive and severe periocular involvement requires oral prednisone. Oral antihistamines are also useful in reducing pruritus.

ALOPECIA

- The causative syndromes of hair loss include the nonscarring (secondary syphilis, alopecia areata, contact dermatitis, thyroid disorders, medication-related) and scarring (tinea capitis, zoster infection, discoid lupus, sarcoidosis, scleroderma, malignancy). Nonscarring alopecia may be reversible, while scarring alopecia is rarely reversible.
- Tinea capitis is a dermatophyte infection of the scalp and is most commonly seen in children. Areas of alopecia with broken hair shafts and peripheral scaling are noted; the alopecia is patchy and usually nonscarring.
- Diagnosis of tinea capitis is based on a potassium hydroxide (KOH) preparation or positive fungal culture. Certain types of dermatophytes fluoresce under Wood's lamp examination.
- Therapy for tinea capitis includes oral griseofulvin for 6 weeks as the first-line agent; topical treatment alone is not effective. Nizoral shampoo also is recommended.
- Alopecia areata presents with a patchy, nonscarring alopecia; loose round patches of hair are lost, leaving behind normal scalp. Any hair-bearing area may be affected, but the scalp is the most common site of involvement.
- Diagnosis of alopecia areata is based on clinical examination. Alopecia areata usually resolves spontaneously within 2 to 6 months.

TINEA INFECTIONS

- Tinea pedis is a fungal infection of the feet, also known as ''athlete's foot.'' Tinea pedis may present in several distinct forms.[1]
- The most common form is the interdigital presentation, manifested by maceration and scaling in the web spaces between the toes; ulcerations may be present in severe cases with secondary infection.
- The second type, which is the form seen in tinea manuum, is characterized by chronic, dry scaling with minimal inflammation on the palmar or plantar surfaces; it often extends to the medial and lateral aspects of the feet but not the dorsal surface.
- The third type of fungal infection presents as an acute, painful, pruritic vesicular eruption on the palms or soles; erythema is a prominent feature, while the nails and web spaces are usually spared.[2]
- Identification of fungal elements on a KOH preparation or with fungal culture may be required. Nonbullous tinea pedis and manuum can be treated with topical antifungal agents, such as clotrimazole, miconazole, ketoconazole, or econazole. Nail infections should also be treated with oral antifungal agents. Bullous tinea pedis requires oral antifungal therapy.
- Tinea cruris, a fungal infection of the groin commonly called ''jock itch,'' is very common in males. Erythema with a peripheral annular, scaly edge is seen; the rash extends onto the inner thighs and the buttocks and spares the penis and scrotum—a feature that is important in distinguishing tinea cruris from other eruptions in the groin, as most other eruptions will affect the scrotum.
- The diagnosis is established by KOH preparation. An antifungal cream such as clotrimazole, ketoconazole, or econazole is the initial treatment of choice.

CANDIDAL INTERTRIGO

- Candidal infections of the skin favor moist, occluded areas of the body. Although any skin fold may be involved, superficial candidal infections are commonly seen in the diaper area of infants, the vulva and groin of women, the glans penis (balanitis) in uncircumcised males, and the inframammary and pannus folds of obese patients. Antibiotic therapy, systemic corticosteroid ther-

apy, urinary or fecal incontinence, immunocompromised states, and obesity are predisposing factors.
- The typical presentation of candidal intertrigo is erythema and maceration with surrounding small erythematous papules or pustules; the satellite pustules are a characteristic finding in differentiating between candidal intertrigo and other inflammatory disorders affecting the skin folds.
- The rim of satellite pustules helps to distinguish candidal intertrigo from other eruptions of the skin folds. KOH preparation of the pustules may demonstrate short hyphae and spores.
- For treatment of candidal intertrigo, a topical antifungal cream such as clotrimazole, ketoconazole, or econazole should be applied. The addition of hydrocortisone 1% cream can speed symptomatic relief and healing.[3]

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Section 20
TRAUMA

153 INITIAL APPROACH TO THE TRAUMA PATIENT

William R. Dennis, Jr.

EPIDEMIOLOGY

- Injury is the fourth leading killer of Americans and the most common cause of death before the age of 45. Trauma causes more deaths among children and adolescents (ages 1 to 19) than all other diseases combined.

CLINICAL FEATURES

- Nonspecific signs such as tachycardia, tachypnea, or mild alterations in consciousness must be presumed to signify serious injury until proven otherwise, and they must be treated in a timely fashion.
- The mechanism of injury may suggest potential problems and these also should be pursued, especially in the patient without signs of significant trauma.

DIAGNOSIS AND DIFFERENTIAL

- The history should be obtained from the patient, witnesses, or prehospital providers and should include mechanism of injury, sites of injury, blood loss at the scene, degree of damage to any vehicles or types of weapons involved.
- Preexisting medical conditions and medications (e.g., steroids and beta blockers) need to be elicited to help understand the patient's physiologic response to injury.
- The primary survey (A-B-C-D-E), including the assessment of a complete set of vital signs, is initiated, characterized by the orderly identification and concomitant treatment of the most lethal injuries.
- Airway patency with cervical spine control and breathing should be assessed by means of examination of the head and neck for gag reflex, airway obstruction, tracheal deviation, quality of breath sounds, flail chest, crepitation, sucking chest wounds, and fractures of the sternum. Problems such as tension pneumothorax, hemothorax, pneumothorax, and misplacement of the endotracheal tube should be identified and immediately treated before preceding further.
- Circulatory status is evaluated via vital signs and cardiac monitoring. Sites of obvious bleeding, indications of shock, and signs of cardiac tamponade (Beck's triad of muffled heart tones, elevate venous pressure, and hypotension) should be identified and treated.
- Disability should be assessed by performing a brief neurologic examination, which includes assessing Glasgow Coma Scale, pupil size and reactivity, and motor function.
- Exposure of the patient should be performed by completely disrobing the patient and examining the total body surface area carefully for bruises, lacerations, impaled foreign bodies, and open fractures.
- The secondary survey is a rapid, but thorough, head-to-toe examination to identify all injuries and to establish priorities for care. Resuscitation and frequent monitoring of vital signs continue throughout this process.
- The scalp should be examined for bleeding, and the skull assessed for significant injury. The pupils should be rechecked, the face examined for frac-

tures, and the tympanic membranes visualized for hemotympanum. The neck, chest, abdomen, and pelvis should be examined thoroughly.
- Radiographs of the lateral cervical spine, chest, and pelvis should be ordered, as appropriate for the scenario.
- The genitourinary system should be evaluated by external inspection, a rectal exam, and a urethrogram for suspected urethral injury (meatal blood present, the prostate displaced). Otherwise, a urinary catheter should be placed and the urine obtained and checked for blood; a pregnancy test should be ordered for female patients of childbearing age. Vaginal blood on a bimanual exam is an indication for a speculum exam.
- The patients should be log-rolled, while maintaining cervical spine stabilization, for examination of the back. Extremities should be checked for soft tissue injury and fracture. A more thorough neurologic examination should be completed.
- After the secondary survey, laboratory studies and additional radiographic studies, such as cystogram, intravenous (IV) pyelogram, aortogram, or computed tomography (CT) scans, should be considered, as needed.
- Diagnostic peritoneal lavage is preferred over the CT scan to evaluate the hemodynamically unstable patient for intraabdominal bleeding. The Focused Abdominal Sonogram for Trauma (FAST) is used for the rapid bedside identification of free intraperitoneal fluid and includes examination for pericardial fluid collections.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- At the outset of the primary survey, airway patency is assured. A chin lift may initially help in opening the airway; suctioning may remove foreign material, blood, loose tissue, or avulsed teeth. Tracheal intubation using a rapid sequence technique is indicated for patients with altered mental status, including those with severe agitation, or for those who for any reason are unable to maintain an open airway on their own. In extensive facial trauma or failure of tracheal intubation, cricothyrotomy or another advanced airway technique must be done to secure the airway.
- During the evaluation of breathing, 100% oxygen is administered by mask or endotracheal tube. Suspected tension pneumothorax is treated immediately with needle decompression followed by tube thoracostomy. For large hemothoraces, consideration may be given to autotransfusion and immediate operative exploration for initial chest tube output of 1500 mL or more. The presence of a flail chest may require intubation, and sucking chest wounds require placement of an occlusive dressing followed by chest tube placement.
- Management of circulation requires placement of 2 large-bore peripheral IV lines. If this cannot be accomplished, central venous access should be obtained. If warranted, 2 L of warm crystalloid should be administered rapidly, followed by O-negative or type-specific blood, as required. External hemorrhage should be controlled with compression at the bleeding site.
- Patients with disability and evidence for intracranial injury on examination may benefit from tracheal intubation for airway protection. Exposure of the patient facilitates the remainder of the management providing that measures to prevent hypothermia are taken.
- Interventions are undertaken during the secondary survey to control problems as they are discovered. Reduction of fractures may prevent distal neurovascular compromise; all fractures should be splinted. A gastric tube should be inserted (orally in the setting of facial fractures) and a urinary catheter placed, if not contraindicated. Tetanus prophylaxis must be assured; an antibiotic such as cefotetan 2 g IV is indicated for possible ruptured abdominal viscus, vaginal, or rectal lacerations; open skeletal fractures should be treated with cephalexin 2 g IV or similar coverage with consideration given to the addition of more coverage for particularly contaminated injuries. Potential closed spinal cord injuries should be treated with methylprednisolone 30 mg/kg IV bolus over 15 min followed 45 min later by an infusion of 5.4 mg/kg/h for 24 to 48 h. Patients with pelvic fractures and signs of persistent hemorrhage may benefit from pelvic arteriography and embolization.
- Upon completion of the secondary survey, options for disposition of the patient are to move to the operating room, admit to the hospital, or transfer to another facility. Ideally, the trauma surgeon or surgeon on-call is present for the secondary survey and can at that point assume primary responsibility for the patient. If transfer is to be made, the resuscitating physician must relay all pertinent information to the accepting physician.

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154 PEDIATRIC TRAUMA

Joseph J. Randolph

EPIDEMIOLOGY

- Trauma is the most common cause of death and disability in children over 1 year of age and the second leading cause of ED visits after infectious disease.[1]
- Head injury is the most frequent cause of death.[2] Motor vehicle crashes are the leading mechanism of injury in children over 1 year of age.[2–4]

PATHOPHYSIOLOGY

- The priorities in pediatric trauma management are similar to those for adults; however, important differences in anatomy, physiology, and psychology dictate some modification to the evaluation and treatment of injured children.
- Nonaccidental trauma must also be considered in the evaluation of the pediatric patient.

CLINICAL FEATURES

- As obligate nose breathers, infants less than 6 months of age with facial trauma or bleeding into the nasopharynx will present with significant respiratory distress.
- A difference in the mechanics of breathing in children results in the early appearance of tachypnea and accessory muscle use in dyspneic patients. Nasal flaring, grunting, and retractions are also signs that should be noted.
- The physiology of shock in children causes tachycardia to be the most sensitive and earliest sign of volume loss; conversely, hypotension is a late and therefore ominous finding.
- Other important signs of hemorrhage are increased capillary refill time, decreased level of responsiveness, decreased urine output, narrowed pulse pressure, and decreased skin temperature.
- The ratio of surface area to mass is greater than in adults, putting pediatric patients at greater risk for hypothermia following injury.
- Signs of spinal cord injury may be subtle or transient, especially in the case of spinal cord injury without radiographic abnormality (SCIWORA). Spinal trauma is relatively uncommon in young children and is more commonly seen in adolescents.[5] Due to increased flexibility of the spine and spinal column in younger children, fractures and dislocations rarely occur with minor trauma.
- Up to 66 percent of spinal cord injuries in children have no radiographic abnormality and thus fall into the category of SCIWORA.[6]
- Over 50 percent of children with SCIWORA have a delayed onset of paralysis, ranging from hours to days. Many of these children have transient paresthesias, numbness, or weakness at the time of the injury or shortly thereafter.
- Sixty-seven percent of cervical spinal injuries in children under the age of 12 occur between the occiput and C2. By comparison, adolescents and adults more commonly experience injuries of the lower cervical spine.[5]
- In considering radiographs of the cervical spine, widening of the prevertebral soft tissues to 8 mm or more anterior to C2, or more than 75 percent of the adjacent vertebral body, is considered abnormal. In infants, however, this becomes less reliable.
- Since the scalp is richly vascularized, some children have developed shock due to significant blood loss, violating the dogma that ''head injuries do not cause shock.''
- Falls from heights less than 5 ft are associated with epidural bleeds in children under 5 years of age.[7] Although the presentation of syncope with the lucid interval has traditionally been associated with epidural hematomas, the presentation is more likely to point to a subdural hematoma, not because of greater association, but because subdural hematomas are much more common.
- Children, with their relatively compliant chest walls, may not show external evidence of serious intrathoracic trauma. In a multiply-injured child, death is 10 times more likely if chest trauma is present.[8]
- A rib fracture is a sensitive indicator of serious

underlying injury. The most common injury is pulmonary contusion, which may not be visible on the initial chest radiograph.[8]
- Elevated amylase levels are associated with injuries of both the pancreas and bowel.[9]
- The spleen, followed by the liver, is the most commonly injured abdominal organ in children. Handlebar injuries often cause isolated pancreatic trauma.[10]
- Pelvic fractures, particularly anterior ring fractures, are associated with urethral and bladder injury.
- The degree of hematuria correlates with the severity of injury in genitourinary trauma, although disruption of the renal pedicle may not be associated with hematuria.[11]

DIAGNOSIS AND DIFFERENTIAL

- The process of evaluating victims of trauma is the same for both children and adults; the primary and secondary surveys are completed in a systematic fashion.
- The imaging modality of choice for the evaluation of head injury is the computed tomography (CT) scan; indications for ordering this test include significant loss of consciousness, deteriorating level of consciousness, neurologic deficits, apparent skull fracture on physical examination, persistent nausea and vomiting, and seizure.
- A high clinical suspicion must be maintained for SCIWORA and high cervical spine injury in the younger child. Physical examination findings consistent with spinal cord injury or abnormalities on spine radiographs are strong indications for CT scanning.
- In the evaluation of abdominal injury in the pediatric patient, the physical examination has both a high false-positive and relatively high false-negative rate. Therefore, either CT scanning or diagnostic peritoneal lavage (primarily for hemodynamically unstable patients) is utilized frequently. CT scan is also indicated for patients with genitourinary trauma demonstrating as few as 20 red blood cells per high-power field.
- Cystourethrography is required for all patients with suspected injuries of the lower urinary tract.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway management in children can be particularly challenging. Anatomic differences responsible for this include a relatively larger tongue and more cephalad location of the larynx.
- All patients should initially be administered 100% oxygen. Suctioning, jaw thrust or chin lift maneuvers, and placement of either a nasal or an oral airway are other measures to be considered.
- The indications for endotracheal intubation are essentially the same as those for adults. The oral route for intubation is preferred; nasotracheal intubation should be avoided due to the cephalad location of the glottis and the propensity to traumatize the upper airway with this approach.
- In children less than 8 years of age, the narrowest portion of the airway is subglottic and a tube that fits through the vocal cords may not pass through this region. An endotracheal tube of appropriate size is selected by using the following formula:

  Internal diameter (in mm) = (16 + age of patient in years)/4

  Patients in this age range should have an uncuffed endotracheal tube placed.
- Rapid-sequence intubation is performed, using pretreatment with 100% oxygen, lidocaine at 1.0 mg/kg IV, and appropriate sedation (e.g., midazolam 0.1 mg/kg IV). Pharmacologic paralysis may be achieved by using either succinylcholine 1.0 to 1.5 mg/kg IV or a nondepolarizing paralytic agent (e.g., rocuronium at a dose of 1 mg/kg IV). Securing an airway in the setting of severe facial trauma may be achieved by transtracheal catheter ventilation. Cricothyrotomy is not recommended in children less than 5 years since identification of the cricothyroid membrane can be difficult and the cricoid cartilage is easily damaged.
- Prior to intubation, atropine at 0.02 mg/kg IV (minimum dose 0.1 mg, maximum dose 1.0 mg) should be administered to children younger than 6 years of age if succinylcholine will be used as the paralyzing agent.
- If IV access is not readily obtained, early placement of an intraosseous line should be performed. The femoral vein is the next easiest site because of the identifiable landmarks and the relative ease of this procedure compared with the placement of other central venous lines in children.
- Resuscitative fluids should be administered in 20-mL/kg boluses of crystalloid; if there is no improvement or deterioration occurs after an initial response, 10-mL/kg boluses of packed red blood cells or whole blood are indicated.
- Fluids should be warmed and used in conjunction with warming lights to prevent hypothermia.

- Burn patients should be resuscitated according to a standard burn formula such as the Parkland formula.
- Children tend to recover better from head injury than adults, but aggressive treatment of hypoxia and hypotension is important to facilitate a good outcome. Severe head injury should be treated with tracheal intubation, elevation of the head of the bed to 30 degrees, and maintaining the head and neck in neutral position. Intravenous mannitol at 0.5 to 1.0 g/kg and furosemide at 1.0 mg/kg may be useful in treating cerebral edema.
- Aggressive hyperventilation in head-injured children has been associated with worsened cerebral ischemia as compared with more moderate hyperventilation.[12] Aggressive hyperventilation should be reserved for children with signs of impending herniation.
- Prophylactic anticonvulsant therapy should be strongly considered in a head-injured child with a Glasgow Coma Scale score under 8, even if no seizures have yet occurred, because the risk of developing acute posttraumatic seizures is high and many of these children already have a high intracranial pressure that will increase further with a seizure.[13]
- In massive hemothorax, operative thoracotomy should be considered if the initial drainage is greater than 15 mL/kg or the chest tube output exceeds 4 mL/kg/h.
- Children with abdominal pain and an elevated serum amylase require an abdominal CT scan and should be hospitalized for observation even if the CT scan findings are normal.[14]
- Pediatric patients should be admitted to the hospital if they have sustained skull fractures or evidence of intracranial injury on CT scan, spinal trauma, significant chest trauma, abdominal trauma with evidence of internal organ injury, or significant burns.

TABLE 154-1 Indications for Transfer to a Pediatric Trauma Center

Mechanism of injury	Ejected from a motor vehicle
	Prolonged extrication
	Death of other occupant in motor vehicle
	Fall from greater distance than three times the child's height
Anatomic injury	Multiple severe trauma
	More than three long-bone fractures
	Spinal fractures or spinal cord injury
	Amputations
	Severe head or facial trauma
	Penetrating head, chest, or abdominal trauma

- Table 154-1 reviews the indications for transfer to a pediatric trauma center.

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155 GERIATRIC TRAUMA

O. John Ma

EPIDEMIOLOGY

- While persons over 65 years of age represent 12 percent of the population, they account for 36 percent of all ambulance transports, 25 percent of hospitalizations, and 25 percent of total trauma costs.[1]
- Approximately 28 percent of deaths due to accidental causes involve persons 65 years and older. The elderly have the highest population-based mortality rate of any age group.[1]

PATHOPHYSIOLOGY

- Chronologic age is the actual number of years an individual has lived. Physiologic age describes the actual functional capacity of a patient's organ systems in a physiologic sense.
- Comorbid disease states such as diabetes mellitus, coronary artery disease, renal disease, arthritis, and pulmonary disease can decrease the physiologic reserve of certain patients, which makes it more difficult for them to recover from a traumatic injury.[2,3]
- Physiologic reserve describes the various levels of functioning of patients' organ systems that allow them to compensate for traumatic derangement.[1]

CLINICAL FEATURES

- Falls are the most common accidental injury in patients over 75 years of age and the second most common injury in the 65 to 74 age group.[1] Falls are reported as the underlying cause of 9500 deaths each year in patients over the age of 65 years. In the >85-year-old age group, 20 percent of fatal falls occur in nursing homes.[4]
- Motor vehicle–related injuries rank as the leading mechanism of injury that brings elderly patients to a trauma center in the United States. Motor vehicle crashes are the most common mechanism for fatal incidents in elderly persons through 80 years of age.[1]
- The clinician should not be led into a false sense of security by ''normal'' vital signs. In one study of 15 patients initially considered to be hemodynamically ''stable,'' 8 had cardiac outputs less than 3.5 L/min and none had an adequate response to volume loading. Of 7 patients with a normal cardiac output, 5 had inadequate oxygen delivery.[5]
- There is progressive stiffening of the myocardium with age, which results in a decreased effectiveness of the pumping mechanism. A normal tachycardic response to pain, hypovolemia, or anxiety may be absent or blunted in the elderly trauma patient.[6] Medications such as beta blockers may mask tachycardia and hinder the evaluation of the elderly patient.
- Elderly persons suffer a much lower incidence of epidural hematomas than the general population. There is a higher incidence of subdural hematomas in elderly patients. As the brain mass decreases with advancing age, there is greater stretching and tension of the bridging veins that pass from the brain to the dural sinuses.[7]
- Severe thoracic injuries, such as hemopneumothorax, pulmonary contusion, flail chest, and cardiac contusion, can quickly lead to decompensation in elderly individuals whose baseline oxygenation status may already be diminished.
- Reduction in pulmonary compliance, total lung surface area, and mucociliary clearance of foreign material and bacteria result in an increased risk for elderly patients to develop nosocomial gram-negative pneumonia.[6]
- Hip fracture is the single most common diagnosis that leads to hospitalization in all age groups in the United States. Hip fractures occur primarily in four areas: intertrochanteric, transcervical, subcapital, and subtrochanteric. Intertrochanteric fractures are the most common, followed by transcervical fractures.[6] Emergency physicians must be aware that pelvic and long bone fractures are not infrequently the sole etiology for hypovolemia in elderly patients.
- The incidence of humeral head and surgical neck fractures in elderly patients are increased by falls on the outstretched hand or elbow.

DIAGNOSIS AND DIFFERENTIAL

- For older patients, the adhesions associated with previous abdominal surgical procedures may increase the risk of performing diagnostic peritoneal lavage in the emergency department.[1] For computed tomography (CT) scanning, it is important to ensure adequate hydration and baseline assessment of renal function prior to the contrast load for the CT scan. Some patients may be volume depleted due to medications, such as diuretics. This hypovolemia coupled with contrast adminis-

tration may exacerbate any underlying renal pathology.[1]
- For unstable patients, and especially those with multiple scars on the abdominal wall from previous procedures, the trauma ultrasound examination is the ideal diagnostic study to detect free intraperitoneal fluid.[8]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Prompt tracheal intubation and use of mechanical ventilation should be considered in patients with more severe injuries, respiratory rates >40 breaths per minute, or when the PaO_2 is <60 mmHg or $PaCO_2$ >50 mmHg.[9]
- Early invasive monitoring has been advocated to help physicians assess the hemodynamic status of the elderly. One study demonstrated that by reducing the time to invasive monitoring in elderly trauma patients from 5.5 h to 2.2 h, and thus recognizing and appropriately treating occult shock, the survival rate of their patients increased from 7 to 53 percent. Survival was improved because of enhanced oxygen delivery through the use of adequate volume loading and inotropic support.[5]
- During the initial resuscitative phase, crystalloid, while the primary option, should be administered judiciously since elderly patients with diminished cardiac compliance are more susceptible to volume overload. Strong consideration should be made for early and more liberal use of red blood cell transfusion.
- Among geriatric trauma patients who are hospitalized, the mortality rate has been reported to be between 15 and 30 percent. These figures far exceed the mortality rate of 4 to 8 percent found in younger patients.[1] In general, multiple organ failure and sepsis cause more deaths in elderly patients than they do in younger trauma victims.[10]
- Several markers for poor outcome in elderly trauma victims have been determined. Age >75 years, Glasgow Coma Scale score ≤7, presence of shock upon admission, severe head injury, and development of sepsis are associated with poor outcome and high mortality figures.[11]
- One study demonstrated that immediately after discharge, one-third of trauma survivors return to independent living, one-third return to dependent status but live at home, and one-third require nursing home facilities. Altogether, at long-term follow-up, 89 percent returned home after trauma and 57 percent returned to independent living.[12]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 245, "Geriatric Trauma," by O. John Ma and Daniel J. DeBehnke.

156 TRAUMA IN PREGNANCY

Stefanie R. Seaman

PHYSIOLOGIC CHANGES OF PREGNANCY AND PATHOPHYSIOLOGY

- By week 10, blood volume increases and red cell mass remains unchanged, leading to a physiologic anemia. Cardiac output and heart rate increase

in the second trimester. There is a subsequent decrease in blood pressure by 10 to 15 mmHg. With these changes, a pregnant woman may lose up to 30 to 35% of her circulating blood volume to demonstrate physiologic changes of shock.
- After 12 weeks, the uterus and bladder become intraabdominal organs, making both susceptible to injury.
- At 20 weeks, the expanding uterus begins to compress the inferior vena cava. This may cause decreased venous return and decreased cardiac output, leading to hypotension while the patient is in the supine position. The enlarged uterus may also cause engorgement of lower extremities and intraabdominal vessels, making the patient susceptible to retroperitoneal hemorrhage.
- After 20 weeks, tidal volume increases and residual volume and functional residual capacity decrease. Compensation to these changes results in respiratory alkalosis. Delayed gastric emptying increases the risk for potential aspiration.[1,2]

CLINICAL FEATURES

- Trauma during pregnancy is associated with risk of preterm labor, placental abruption, fetal-maternal hemorrhage, and pregnancy loss.
- Splenic injury is the leading cause of intraabdominal hemorrhage.
- Lower abdominal viscera are protected by the enlarging uterus. However, uterine irritability and preterm labor can develop.
- Upward displacement of intestines may result in complex injuries in penetrating trauma to the upper abdomen.
- Uterine rupture, most commonly seen during the second and third trimesters, is uncommon. It is diagnosed by loss of palpable uterine contour, ease of palpation of fetal parts, or radiologic evidence of abnormal fetal location. Uterine rupture is more likely to occur in the second and third trimesters. Fetal mortality is nearly 100 percent, while maternal mortality is less than 10 percent.
- Maternal death is the leading cause of fetal death.
- The second leading cause of fetal death is placental abruption, which presents with abdominal pain, vaginal bleeding, and uterine contractions. It may also lead to disseminated intravascular coagulation due to the introduction of placental products into the maternal circulation.
- Up to 12 weeks' gestation, the fetus is protected by the bony pelvis, making injury uncommon. Later in pregnancy, fetal injuries tend to involve the head.
- Fetal-maternal hemorrhage occurs in over 30 percent of cases of significant trauma and may result in Rh-isoimmunization of Rh-negative women. As little as 0.1 to 0.3 mL of fetal cells is needed to sensitize an Rh-negative woman. Fetal hemorrhage may also cause fetal hypovolemia, distress, and death.[3–5]

DIAGNOSIS AND DIFFERENTIAL

- Appropriate laboratory evaluation includes the complete blood cell count, blood type and Rh determination, and coagulation studies. The Kleihauer-Betke test on maternal blood is useful to quantify the degree of fetal-maternal hemorrhage.
- Intraabdominal injury may be detected using computed tomography (CT) of the abdomen, the trauma ultrasound exam, or diagnostic peritoneal lavage, which is performed using a supraumbilical approach.
- The indications for emergent laparotomy remain unchanged.
- While efforts should be made to limit radiographic studies to those that are clinically mandatory, studies should not be withheld out of concern for the fetus. Adverse fetal effects from radiation are greatest during the first 8 weeks of gestation and are negligible from doses less than 10 rad. Abdominal CT scan delivers between 2 and 5 rads. This can be reduced by decreasing the number of slices obtained. Standard trauma radiographs deliver substantially less than 1 rad.
- Fetal radiation exposure can be further limited by judicious shielding of the uterus. Magnetic resonance imaging and ventilation/perfusion scanning have not been associated with adverse fetal outcome.[6,7]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The best care for the fetus is proper resuscitation of the mother. Establishment of a patent airway, adequate ventilation, and large-bore vascular access are paramount.
- The airway should be secured and supplemental oxygen administered. Early passage of a nasogastric or orogastric tube decreases the risk of aspiration.
- Crystalloid IV fluids should be administered to treat hypovolemia. Vasopressors impair uterine blood flow and should be considered only after aggressive fluid resuscitation.

- The patient should be kept in the left lateral decubitus position, where feasible, to minimize hypotension due to compression of the inferior vena cava by the gravid uterus.
- Rh immune globulin (RhoGAM), 300 μg IM, should be administered to all Rh-negative patients beyond 12 weeks' gestation with abdominal trauma. One dose protects against 30 mL of fetal blood. The Kleihauer-Betke test an be used to determine the need for additional doses.
- Tetanus prophylaxis is safe to administer as needed.
- The use of tocolytic agents for increased uterine contractility should be individualized, as these drugs may interfere with the diagnosis of maternal and fetal injuries.
- The uterus should be assessed for tenderness or contractions and a sterile pelvic exam performed, inspecting for injuries or vaginal bleeding. Rupture of amnionic membranes is indicated by the presence of clear fluid of pH 7 in the vaginal canal that produces ''ferning'' when dried on a microscope slide.
- Fetal assessment starts with determination of the fetal heart rate. Fetal viability is directly related to the presence of fetal heart sounds. When these sounds are absent on patient arrival, resuscitation should be directed solely at the mother.
- The normal fetal heart rate is 120 to 160 per minute. Bradycardia suggests hypoxia, often due to maternal hypotension, hypothermia, respiratory compromise, or abruption. Tachycardia may result from hypoxia or hypovolemia. Bedside ultrasound can be used to determine fetal heart rate as well as gestational age, fetal activity, placental location, and amnionic fluid volume. Ultrasound has not been shown useful in diagnosing placental abruption or uterine rupture.
- External fetal monitoring should be initiated early. A minimum of 4 h of monitoring is predictive of immediate adverse outcome. After 20 weeks' gestation, the presence of more than eight contractions per hour is predictive of placental abruption. Beyond the viable gestational age of 23 weeks, fetal tachycardia, late decelerations, or lack of beat-to-beat variability may be indications for emergent cesarean section.
- Should the pregnant trauma patient die, perimortem cesarean section may be considered if fetal heart tones are detected on patient arrival and the gestation is determined to be beyond 23 weeks. Resuscitation of the mother should be continued during the procedure. Infant outcome is excellent when this operation is performed within 5 min of maternal death.
- Patients who display evidence of fetal distress or increased uterine irritability during the initial observation should be admitted.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 246, ''Trauma in Pregnancy,'' by Nelson Tang.

157 HEAD INJURY

Mark E. Hoffmann

EPIDEMIOLOGY

- Approximately 1.5 million people per year sustain a nonfatal traumatic brain injury (TBI)[1] and TBI accounts for 50 percent of all trauma-related deaths.
- Young men, the elderly, children, and alcoholics are at greater risk for TBI.[2,3]

PATHOPHYSIOLOGY

- Direct injury is caused immediately by the forces of an object striking the head or by a penetrating injury.
- Indirect injuries are from acceleration/deceleration forces that result in the movement of the brain inside the skull.
- Secondary injury occurs minutes to days after the event and may result in intracranial hemorrhage, cerebral edema, mass lesions, and increased intracranial pressure (ICP). Further brain injury may be prevented by treating hypoxia, anemia, hypotension, hyperglycemia, and hyperthermia.[4]
- Cerebral perfusion pressure (CPP) is the difference between the mean arterial pressure (MAP) and the ICP.[5] The elevation of the ICP and/or hypotension results in a depressed CPP and leads to further brain injury.
- Rapid rises in the ICP can lead to the ''Cushing reflex,'' characterized by hypertension, bradycardia, and respiratory irregularities. The Cushing reflex is seen uncommonly and usually in children.

CLINICAL FEATURES

- Out-of-hospital medical personnel often provide critical aspects of the history, including mechanism and time of injury, presence and length of unconsciousness, initial mental status, seizure activity, vomiting, verbalization, and movements of extremities.
- The Glasgow Coma Scale (GCS, Table 157-1), a numeric rating of the best eye/verbal/motor response, can be used to classify TBI as mild (GSC >13), moderate (GCS between 13 and 9), and severe (GCS <9) in the nonintubated and nonsedated patient.[6]
- The neurologic exam should note the patient's mental status, GCS, pupil size and reactivity, anisocoria, cranial nerve function, motor/sensory/brainstem function, deep tendon reflexes, and any decorticate or decerebrate posturing.
- Skull fractures that are linear and nondepressed with an intact scalp are common and do not require treatment; however, a computed tomography (CT) scan may be warranted if the fracture line crosses the middle meningeal artery or a major dural sinus. Depressed skull fractures should be elevated surgically. Basilar skull fractures may present with hemotympanum, periorbital ecchymosis (raccoon eyes), rhinorrhea, or retroauricular ecchymosis (Battle's sign).
- Concussion is a diffuse head injury, usually associated with transient loss of consciousness, that occurs immediately following blunt head trauma. Symptoms of amnesia and confusion are clinical hallmarks.
- Contusions and intracerebral hemorrhages are common in the frontal poles, the subfrontal cortex, and the anterior temporal lobes. Contusions may occur directly under the site of impact (coup lesion) or on the contralateral side (contrecoup lesion). Patients may demonstrate significant mental status changes or focal neurologic deficits. These

TABLE 157-1 The Glasgow Coma Scale for All Age Groups*

	4 YEARS TO ADULT	CHILD <4 YEARS	INFANT
EYE OPENING			
4	Spontaneous	Spontaneous	Spontaneous
3	To speech	To speech	To speech
2	To pain	To pain	To pain
1	No response	No response	No response
VERBAL RESPONSE			
5	Alert and oriented	Oriented, social, speaks, interacts	Coos, babbles
4	Disoriented conversation	Confused speech, disoriented, consolable, aware	Irritable cry
3	Speaking but nonsensical	Inappropriate words, inconsolable, unaware	Cries to pain
2	Moans or unintelligible sounds	Incomprehensible, agitated, restless, unaware	Moans to pain
1	No response	No response	No response
MOTOR RESPONSE			
6	Follows commands	Normal, spontaneous movements	Normal, spontaneous movements
5	Localizes pain	Localizes pain	Withdraws to touch
4	Movement or withdrawal to pain	Withdraws to pain	Withdraws to pain
3	Decorticate flexion	Decorticate flexion	Decorticate flexion
2	Decerebrate extension	Decerebrate extension	Decerebrate extension
1	No response	No response	No response
3–15			

* GCS reporting should be modified for intubated and paralyzed patients.

lesions may exert a mass effect that can result in the elevation of ICP and an increased risk of a herniation syndrome.

- Epidural hematomas are convex areas of extraaxial arterial bleeding between the dura and the skull. Approximately 80 percent of cases are associated with a skull fracture and a laceration of a meningeal artery, most commonly the middle meningeal artery. Patients may experience a ''lucid interval'' prior to deterioration.
- A subdural hematoma is a concave collection of venous blood between the dura and the arachnoid resulting from tears of the bridging veins that extend from the subarachnoid space to the dural venous sinuses. Patients with cortical atrophy, such as alcoholics and the elderly, are more susceptible to subdural hematoma formation when undergoing acceleration-deceleration forces during head trauma. After 2 weeks, patients are defined as having a chronic subdural hematoma, which appear hypodense on a CT scan.
- Subarachnoid hemorrhage results from the disruption of subarachnoid vessels and presents with blood in the cerebrospinal fluid. Patients may complain of headache, photophobia, and have mild meningeal signs.
- Diffuse or focally increased ICP can result in herniation of the brain at several locations.
- Transtentorial (uncal) herniation occurs when the uncus of the temporal lobe is forced through the tentorial hiatus causing compression of the ipsilateral third cranial nerve and the cerebral peduncle. This leads to a dilated ipsilateral pupil and contralateral hemiparesis.
- Cerebellotonsillar herniation through the foramen magnum occurs much less frequently. Medullary compression causes bradycardia, apnea, and death.
- Cingulate or subfalcial herniation occurs when part of the cerebral cortex is displaced underneath the falx cerebri into the opposite supratentorial space.
- Penetrating injury to the brain results from gunshot wounds and penetrating sharp objects. The degree of neurologic injury depends on the energy of the missile, whether the trajectory involves a single or multiple lobes or hemispheres of the brain, the amount of scatter of bone and metallic fragments, and whether a mass lesion is present.

DIAGNOSIS AND DIFFERENTIAL

- Approximately 5 percent of patients suffering a severe TBI have an associated cervical spine fracture. Cervical spine radiographs should be obtained on all patients with TBI who present with altered mental status, neck pain, intoxication, neurologic deficit, severe distracting injury, or mechanism of injury capable of producing cervical spine injury.
- All patients with moderate to severe TBI should undergo a CT scan of the head without contrast. Other indications for CT scan include mild TBI with failure to improve or deterioration, amnesia, loss of consciousness, vomiting, intoxication with failure to improve, posttraumatic seizures, coagulopathy, focal neurologic deficit, or suspected skull fracture over the meningeal artery or dural sinuses.[7]
- Skull radiographs are indicated for penetrating trauma to help localized foreign bodies or assess the degree of bone depression.
- Laboratory work for significant head injury patients should include type and cross-matching, complete blood cell count, electrolytes, glucose, arterial blood gas, directed toxicologic studies, prothrombin time, partial thromboplastin time, platelets, and disseminated intravascular coagulation panel.
- Occult trauma should be addressed by the history and physical examination. Approximately 60 percent of patients with TBI have associated major injuries. Further imaging and intervention should proceed when appropriate.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Oxygen, cardiac monitoring, and two intravenous (IV) lines should be secured. For patients with severe TBI, endotracheal intubation to protect the airway and prevent hypoxemia is the top priority. Orotracheal rapid sequence intubation should be utilized. When properly performed, it assists in preventing increased ICP and has a low complication rate. When performing rapid sequence intubation, it is imperative to provide adequate cervical spine immobilization and to use a sedation/induction agent.
- Hypotension can lead to depressed CPP. Restoration of adequate blood pressure is initially maintained by IV crystalloid fluid. Intravenous fluids should be administered cautiously to avoid cerebral edema. Hypotonic and glucose-containing solutions should be avoided. Hypotension is usually caused by the associated injuries, not the TBI.
- Initial management of increased ICP includes elevating the head of the patient's bed to 30°, provid-

ing adequate resuscitation to maintain a MAP of 90 mmHg, and maintaining adequate arterial oxygenation.[8] Administration of mannitol 0.25 to 1.0 g/kg IV should be considered. Hypoventilation should be avoided. Use of hyperventilation is controversial; it should be reserved as a last resort for decreasing the ICP. If used, hyperventilation should be implemented as a temporary measure, aiming to maintain a pCO_2 between 30 to 35 mmHg. The pCO_2 should be monitored closely.[9]

- For posttraumatic seizures, IV lorazepam or diazepam should be administered. Phenytoin at a loading dose of 18 mg/kg IV should be infused no faster than 50 mg/min.
- Patients with an initial GCS of 15 that is maintained, normal serial neurologic exams, and a normal CT scan may be discharged home. Those with a positive CT scan require neurosurgical consultation and admission. All patients who experience a head injury should be discharged home with a reliable companion who can observe the patient for at least 24 h, carry out appropriate discharge instructions, and follow the head injury sheet instructions.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 247, "Head Injury," by Thomas Kirsch, Salvatore Migliore, and Teresita Hogan.

158 SPINAL INJURIES

Mark E. Hoffmann

EPIDEMIOLOGY

- The incidence of traumatic spinal cord injuries (SCI) in the United States has been estimated at 30 cases per million population at risk.
- The mean age has been reported as 33.5 years, with a male-to-female predominance of 4 to 1.[1]
- Ninety percent of SCI are related to motor vehicle crashes.

PATHOPHYSIOLOGY

- The vertebral column serves as the central supporting structure for the head and trunk and provides protection for the spinal cord with 33 vertebrae.
- The vertebrae of the cervical, thoracic, and lumbar spine are stacked atop each other and are separated by intervertebral disks that cushion axial loads.
- There are 3 vertical columns that provide stability to the spine: the anterior column (anterior longitudinal ligament and the anterior half of the vertebral body), the middle column (posterior longitudinal ligament and the posterior half of the vertebral body), and the posterior column (the pedicles, lamina, spinous processes, and the posterior ligament complex).[2]
- Failure of 2 or more columns results in an unstable injury (radiographs may be without fractures in a pure ligamentous injury).
- The spinal cord is composed of three major tracts: the posterior columns (ipsilateral sensation and proprioception), the corticospinal tracts (ipsilateral motor fibers), and the spinothalamic tracts (contralateral pain and temperature).
- The lower nerve roots, inferior to the conus medullaris, form an array of nerves around the filum terminale; this is called the *cauda equina.*
- Various fractures, dislocations, blunt and penetrating injury patterns, and disk herniations may lead to SCI or nerve root impingement syndromes.

CLINICAL FEATURES

- Unstable bony injury may exist without actual SCI or nerve root trauma.
- Vertebral fractures may have localized pain on palpation of the injured spine, muscle spasms, splinting, and resistance to movement. Palpable crepitus, deformity, and step-off may also be present on examination of the midline.
- Paresthesias, dysesthesias, sensory disturbances, motor deficits, reflex abnormalities, and spinal shock may be present with bony fractures and SCI.
- Injury to the corticospinal tract produces an ipsilateral upper motor neuron lesion that results in increased deep tendon reflexes, spasticity, weakness, and a Babinski sign.
- Injury to the dorsal column, located in the posterior aspect of the spinal cord, results in loss of ipsilateral light touch sensation and proprioception.
- Injury to the spinothalamic tracts results in contralateral pain and temperature sensory losses. These fibers decussate in the anterior aspect of the spinal cord at the vertebral level.
- Injury to the nerve roots produces an ipsilateral lower motor neuron lesion and a radiculopathy that may result in decreased deep tendon reflexes, weakness, and sensory loss in that nerve distribution.
- Spinal shock is characterized by warm, pink, dry skin; adequate urine output; and relative bradycardia. Other signs of autonomic dysfunction may accompany spinal shock, such as ileus, urinary retention, fecal incontinence, and priapism.

DIAGNOSIS AND DIFFERENTIAL

- The history is useful in defining the mechanism of SCI, thus allowing the clinician to anticipate specific potential injury patterns.
- The physical examination should focus on complete palpation of the spine, testing the symmetry of reflexes, motor strength, pain sensation, and light touch and proprioception in each extremity.
- Rectal tone, perianal sensation and wink, and bulbocavernosus reflexes should be assessed.
- Plain film radiography of the traumatized portion of the spine is required when the following are present: (a) midline pain or bony tenderness, crepitus, or step-off; (b) neurologic deficit; (c) presence of distracting injuries; (d) altered mental status; (e) complaint of paresthesia or numbness.[3]
- Cervical spine radiographs require an anteroposterior view, a lateral view, and an odontoid view.
- A computed tomography (CT) scan with or without myelography or a magnetic resonance imaging (MRI) scan may be required to further evaluate the extent of the spinal injury.
- Once a bony abnormality is identified, a key component of the differential is the degree of stability associated with that particular type of injury.
- Fractures of the odontoid with rupture of the transverse atlantal ligament are extremely unstable.
- A Hangman's fracture is an unstable fracture of the pedicles of the posterior arch of C2 caused by extension and distraction injury.
- A Jefferson fracture is an axial load compression fracture of the anterior and posterior arches of C1 and is an unstable fracture.
- Extension ''teardrop'' fractures are unstable fractures where the anterior longitudinal ligament avulses the anterior-inferior corner of the vertebral body.
- Wedge or compression fractures may be unstable if there is a loss of greater than 50 percent of vertebral body height and failure of the posterior ligaments.
- Burst fractures result from axial loading and may be responsible for retropulsion of fragments causing spinal cord compression.
- Distraction fractures are associated with motor vehicle crashes; a severe and unstable variant is the Chance fracture with horizontal fracture from the spinous process through the vertebral body.
- Thoracolumbar fracture-dislocations are grossly unstable and have a significant incidence of associated SCI.
- For patients with obvious SCI, the differential includes complete lesions and a number of incomplete lesions and syndromes.
- Anterior cord syndromes involve the loss of motor function and pain and temperature sensation distal to the level of injury with preservation of light touch, vibration, and proprioception.[4]
- A central cord syndrome, associated with hyperextension injuries, presents with motor weakness more prominent in the arms than in the legs and with variable sensory loss.[5]
- The Brown-Sequard syndrome most often results from penetrating trauma and is caused by a hemisection of the spinal cord. There is loss of ipsilateral motor function, proprioception, light touch sensation, and loss of contralateral pain and temperature sensation.
- The cauda equina syndrome is less of a spinal cord lesion than it is a peripheral nerve injury, and it presents with variable motor and sensory loss in

the lower extremities, sciatica, bowel or bladder dysfunction, and "saddle anesthesia."
- In the pediatric patient, injuries due to child abuse and spinal cord injury without radiographic abnormality (SCIWORA) may be encountered.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Airway, breathing, and circulation should be stabilized.
- Cervical and complete spinal immobilization with long spine board and a hard cervical collar should be in place.[6] Patients should be placed on 100% oxygen, a cardiac monitor, pulse oximetry, and blood pressure monitoring, and have 2 large-bore intravenous (IV) lines established.
- If rapid sequence intubation is performed, then careful in-line cervical stabilization (not traction) should be applied.
- Strong consideration should be given to CT, ultrasound, or diagnostic peritoneal lavage to exclude the possibility of intraabdominal injury.[7]
- Hypotension resulting from spinal shock should be treated with IV crystalloid fluid and low dose dopamine at 5 to 10 μg/kg/min. A Foley catheter should be inserted to monitor urinary output.
- Closed SCIs should be treated with high-dose methylprednisolone, with a loading dose of 30 mg/kg over 15 min, followed 45 min later by an IV drip at 5.4 mg/kg/h for the next 23 h.[8]
- Removal of the patient from the long spine board within 2 h, with full spine precautions, is recommended to prevent skin breakdown and pressure sores.
- Stable patients may be further imaged with specific spinal radiographs, CT scans, or MRI.
- Neurosurgical or orthopedic consultation is required for clinically significant spinal fractures or SCI.
- Any patient with an unstable spine, nerve root compression, uncontrollable pain, or intestinal ileus should be admitted to the hospital.
- Patients with significant vertebral or spinal cord trauma should be managed at a regional trauma or spinal cord injury center.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 248, "Spinal Cord Injuries," by Bonny Baron and Thomas Scalea.

159 MAXILLOFACIAL TRAUMA

M. Chris Decker

EPIDEMIOLOGY

- The most common etiologies for facial fractures in the urban setting are assault and penetrating trauma.
- The most common etiologies for facial fractures in the community setting are motor vehicle crashes and sporting and recreational injuries.
- Approximately 30 percent of maxillofacial fractures in women are associated with sexual or domestic violence.[1]
- There is a strong association with facial trauma and domestic violence in the elderly.
- More than 50 percent of abused children sustain injuries to the head, face, mouth, or neck.[2]

PATHOPHYSIOLOGY

- The facial buttresses and bony arches are joined by suture lines that provide vertical and horizontal support for the face.

- Sutures linking the facial bones rupture in a predictable fashion during trauma.
- The most complex aspect of facial anatomy is the orbit, an elaborate structure comprising seven different bones: maxilla, zygoma, frontal, sphenoid, palatine, ethmoid, and lacrimal.
- The orbital foramina contains cranial nerves II, III, VI, and the branches of V.

CLINICAL FEATURES

- The mechanism of injury, any history of loss of consciousness, visual changes, diplopia, paresthesias, and malocclusion are essential components of the history.
- The physical examination should include the inspection and palpation of the following: the scalp, ears, auditory canals, tympanic membranes, mastoids, orbits, eyes, zygomas, maxilla, teeth, tongue, lips, mandible, and neck. The examination should include a complete sensorimotor evaluation of the face. Any facial tenderness, crepitus, and subcutaneous air should be noted.
- Approximately 90 percent of facial fractures are detected by palpation.[3]
- The degree of facial instability associated with the LeFort fractures should be assessed by grasping the maxillary arch (above the incisors) with one hand while stabilizing the forehead with the other (Fig. 159-1).
- The LeFort I is a transverse fracture through the maxilla, pterygoid plate, and nasal septum, resulting in a floating maxilla. Clinically, the hard palate and upper teeth move with stressing.
- The LeFort II is a pyramidal fracture of the central maxilla across the bridge of the nose. The nose, hard palate, and upper teeth move as a unit disjoined from the zygomas with stressing.
- The LeFort III, or craniofacial disjunction, involves the maxilla, nasal bones, ethmoid, and zygoma. The entire face moves with stressing.
- The eye examination should document visual acuity, pupil shape/size, alignment, and reactivity. A Marcus Gunn pupil (initial dilation with the swinging light test) suggests retinal or optic nerve injury. A teardrop pupil suggests globe rupture. Monocular diplopia may represent lens dislocation; binocular diplopia may represent entrapment of the inferior rectus or cranial nerve injury. The anterior chamber should be evaluated for the presence of a hyphema, and the cornea should be stained with fluorescein to identify abrasions.
- Facial sensation should be tested for anesthesia of the upper lip, nasal mucosa, lower lid, and maxillary teeth. Positive findings suggest infraorbital nerve injury.
- The mandible should be palpated for step-off, tenderness, crepitus, and instability.
- The mouth should be examined for lacerations, tooth fractures, malocclusion, tenderness, or anesthesia. Anesthesia to the dentition, lower lip, or chin may represent a mandibular fracture.
- Mastoid ecchymosis (Battle's sign), hemotympanum, periorbital ecchymosis (''raccoon eyes''), and cerebrospinal fluid (CSF) otorrhea are clinical signs of a basilar skull fracture.
- The nose should be assessed for septal hematoma and CSF rhinorrhea (halo/double-ring sign).
- The ear should be inspected for subperichondral hematoma.

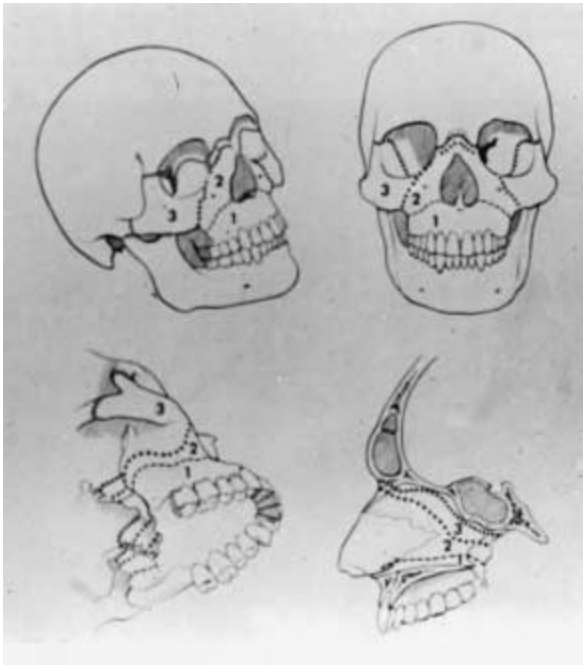

FIG. 159-1 Schematic of midfacial fracture lines: Le Fort I, II, and III. (Reprinted with permission from Dingman RO, Nativg P: *Surgery of Facial Fractures.* Philadelphia, Saunders, 1964, p 248.)

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of specific maxillofacial injuries is based on clinical findings, facial radiographs, and facial computed tomography (CT). Patient stability will dictate the timing and order of these imaging modalities.
- The following radiographs may be useful. The Waters view (occipital mental view) is the most valu-

able for midface fractures. The posteroanterior (PA or Caldwell) view best details the upper facial bones. The ''jug-handle'' (submental vertex) view is the best for evaluating the zygomatic arches. The Townes view is useful for mandibular rami and basilar skull fractures. Lateral radiographs can assess air-fluid levels in the ethmoid and sphenoid sinuses.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The major focus in prehospital care is airway management and spinal immobilization. Airway management and hemorrhage control are paramount in the emergency department (ED). Chin lift, jaw thrust, and oral suctioning without neck extension often restore patency.
- Severe mandibular fractures often cause posterior displacement of the tongue. The tongue should be pulled forward with a gauze pad, towel clip, or large suture to relieve any obstruction.[4]
- For endotracheal intubation, the oral route is preferred because of the risk of nasocranial intubation or severe epistaxis associated with nasotracheal intubation attempts. The use of neuromuscular blocking agents should be avoided if at all possible. Fiberoptic intubation, a Bullard intubating blade, and the laryngeal mask airway may be useful adjuncts with a difficult airway. If neuromuscular blocking agents are used, equipment for emergent cricothyrotomy should be at the bedside.
- The cervical spine should be cleared, either clinically or radiographically.
- Hemorrhage should be controlled with direct pressure; blind clamping should be avoided. Pharyngeal bleeding can be controlled with packing around the endotracheal tube. Severe epistaxis may be controlled with direct pressure and posterior nasal packing.
- Management of the airway, proper fluid resuscitation, and evaluation of associated head, chest, abdominal and spinal trauma should take precedence over facial radiographs.
- For reliable patients who have been cleared of serious injuries, radiographic evaluation may be performed on an outpatient basis.

CARE OF SPECIFIC FRACTURES

- Antibiotics—such as amoxicillin-clavulanate, trimethoprim-sulfamethoxazole, or a first-generation cephalosporin—should be administered to patients with sinus fractures or with nasal packing. Isolated sinus and frontal bone fractures can be managed on an outpatient basis. Inpatient management is warranted for sinus fractures of the posterior wall, depressed fractures, or intracranial injury.
- Orbital blowout fractures are the most common type of orbital fracture. A CT scan should be obtained to determine the surface area of injury. Indications for surgery include enophthalmos, persistent diplopia, and entrapment of the extraocular muscles.[5]
- Septal hematomas should be drained under local anesthesia, using a no. 11 blade, by incising along the inferior border of the hematoma. The nostril and septum should be packed and appropriate antibiotics prescribed.
- Zygomatic fractures: Patients with tripod fractures, which involve the infraorbital rim, a diastasis of the zygomatic-frontal suture, and disruption of the zygomatic-temporal junction at the arch require admission for open reduction and internal fixation. Those with fractures of the zygomatic arch can have elective outpatient elevation and repair.
- Open mandibular fractures require admission and IV antibiotics.
- Temporomandibular joint (TMJ) dislocation should be reduced with the physician standing behind the seated patient and pushing downward and backward on the posterior molar. Sedation and local anesthesia to the TMJ, lateral pterygoid, and masseter muscles may be necessary. A Barton bandage should be applied after reduction.
- Children under 6 years of age are more likely to have injury to the frontal bone, given its prominence. Maxillary fractures are uncommon in children due to the lack of maxillary sinus development. By age 12, the child's fracture pattern is the same as that of the adult.
- Early follow-up is important for pediatric facial fractures, given the rapid healing rates in children and the potential for asymmetric facial growth.

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 249, ''Maxillofacial Trauma,'' by Stephen Colucciello.

160 NECK TRAUMA

M. Chris Decker

EPIDEMIOLOGY

- The demographics of neck trauma patients are expected to mirror those of other trauma victims.
- Multiple injuries occur 44 to 52 percent of the time with penetrating trauma.[1–5]

PATHOPHYSIOLOGY

- The neck contains a high concentration of vascular, aerodigestive, and spinal structures in a relatively confined space.
- The Roon and Christensen anatomic classification divides the neck into three zones (Table 160-1). The at-risk structures located in zone 1 are the vertebral and proximal carotid arteries, major thoracic vessels, superior mediastinum, lungs, esophagus, trachea, thoracic duct, and spinal cord. The at-risk structures located in zone 2 are the carotid and vertebral arteries, jugular vein, esophagus, trachea, larynx, and the spinal cord. The at-risk structures located in zone 3 are the distal carotid and vertebral arteries, pharynx, and the spinal cord.
- The platysma is the most superficial structure beneath the skin and serves as an important planar landmark in evaluating penetrating neck injuries. Beneath the platysma is the deep cervical fascia and the fascial compartments that support the muscles, vessels, and viscera of the neck. The tight fascial compartments offer a tamponade effect, which helps limit potential for external bleeding from vascular injuries; however, bleeding within this confined space can result in extrinsic compression and airway compromise.

TABLE 160-1 Zones of the Neck

Zone I	Base of the neck to the cricoid cartilage
Zone II	Cricoid cartilage to the angle of the mandible
Zone III	Angle of the mandible to the base of the skull

CLINICAL FEATURES

- Presentations of neck injuries involve manifestations of vascular, aerodigestive, and neurologic symptoms and signs. All signs require diagnostic evaluation but hard signs are more often associated with significant injury (Table 160-2).
- Both blunt and penetrating laryngeal or pharyngeal trauma can cause dysphonia, stridor, hemoptysis, hematemesis, dysphagia, neck emphysema, and dyspnea progressing to respiratory arrest.
- Acute hemorrhage may be visible externally or can occur internally, leading to hematoma formation with tracheal deviation or bleeding into the pharynx. In both situations, tachycardia, hypotension, and other signs of shock indicate significant blood loss; airway compromise may result from the mass effect of an expanding hematoma.
- Neurologic symptoms and signs range from complaints of pain or paresthesias to hemiplegia, quadriplegia, and coma.
- Gastrointestinal injury initially may be asymptomatic, though patients may complain of dysphagia and hematemesis may be observed.
- Strangulation may cause petechiae of the skin

TABLE 160-2 Signs and Symptoms of Neck Injury

HARD SIGNS	SOFT SIGNS
Hypotension in Emergency Department	Hypotension in field
Active arterial bleeding	History of arterial bleeding
Diminished carotid pulse	Tracheal deviation
Expanding hematoma	Nonexpanding large hematoma
Thrill/bruit	Apical capping on chest x-ray
Lateralizing signs	Stridor
Hemothorax >1000 cc	Hoarseness
Air or bubbling in wound	Vocal cord paralysis
Hemoptysis	Subcutaneous emphysema
Hematemesis	Seventh cranial nerve injury
	Unexplained bradycardia (without CNS injury)

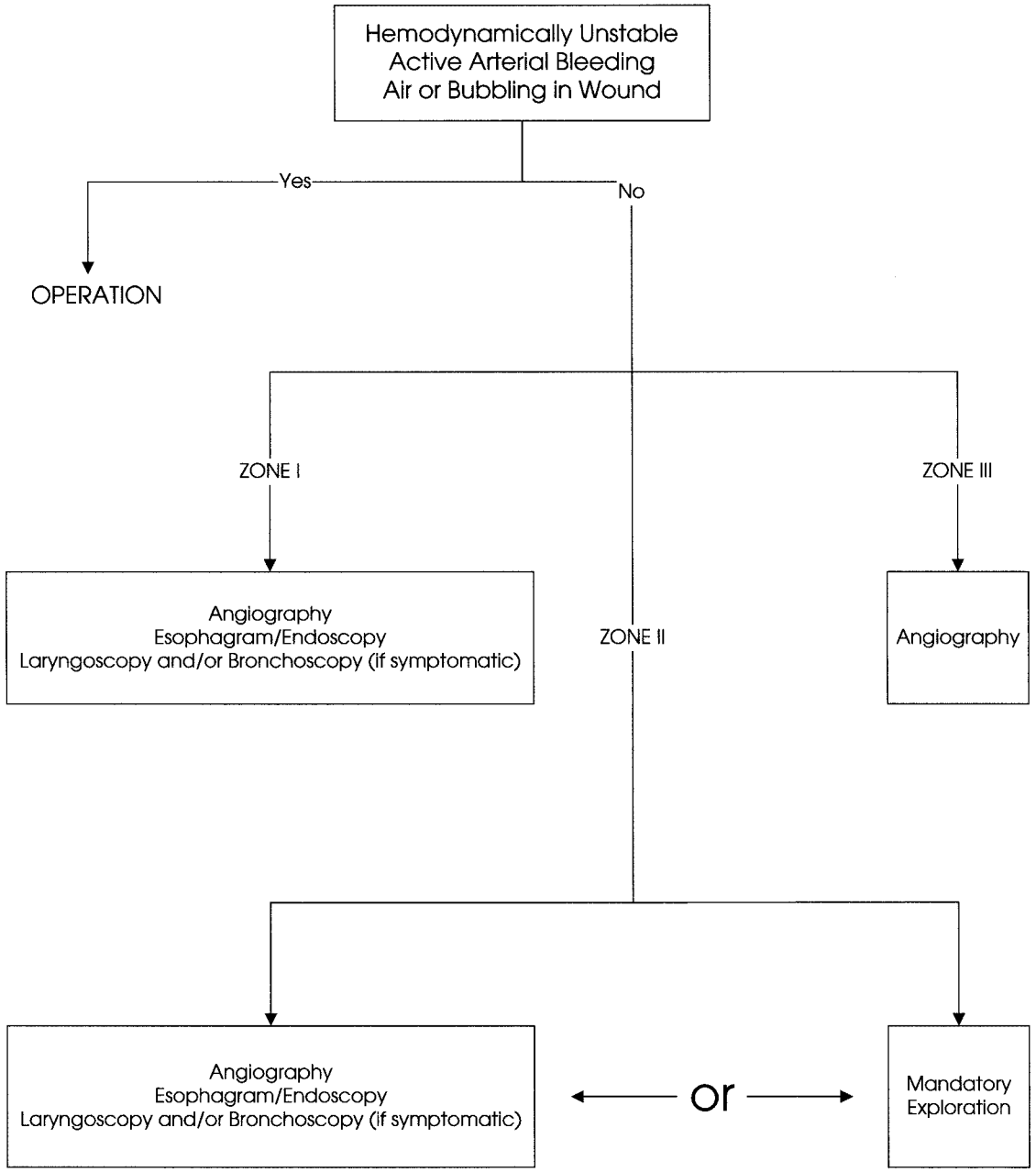

FIG. 160-1 Management of penetrating neck injury.

above the site of ligature and in the subconjunctivae.[6–9]

DIAGNOSIS AND DIFFERENTIAL

- Penetrating wounds are classified by the zone of injury and evaluated for possible violation of the platysma muscle. No further probing of deep wounds is warranted in the emergency department (ED); full exploration awaits surgical consultation and the capacity for proximal and distal vascular control in the operating room.
- Plain radiographs can identify cervical spine injury, the presence of any penetrating foreign body, air in the soft tissues, and soft tissue swelling. A chest radiograph is warranted for any suspected thoracic cavity penetration.
- Additional diagnostic procedures to be considered, in conjunction with surgical consultation, in-

clude arteriography or duplex sonography for suspected arterial injury, computed tomography (CT) scanning of the larynx or cervical spine, endoscopy of the airway and esophagus, or contrast studies of the esophagus.
- The differential diagnosis relates to the various structures at risk for injury. Airway injury may be encountered in cases involving blunt trauma as well as penetrating mechanisms of injury. Vascular injury is most common with penetrating trauma, although major vessel injury can occur due to blunt trauma and may simulate an acute stroke. Neurologic injuries include generalized brain ischemia (seen primarily with strangulation), spinal cord trauma, nerve root damage, and peripheral nerve damage. Cervical spine injury initially may present without neurologic deficit, but the spine can be cleared clinically in selected blunt trauma and gunshot wound victims. Gastrointestinal injuries are often occult and generally require evaluation by endoscopy or contrast radiography.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Hemodynamic and cardiac monitoring, IV access, and 100% oxygen with pulse oximetry are required initially for all patients.
- Airway management is made critical by the potential for direct injury and resulting potential for airway compromise. Tracheal intubation is indicated for patients unable to maintain airway patency secondary to structural disruption, edema, secretions, bleeding, enlarging hematoma, or impending respiratory arrest. In cases where oral or nasal intubation is not possible or is contraindicated, cricothyrotomy or transtracheal jet insufflation may be performed.
- The chest must be evaluated for pneumothorax and hemothorax secondary to vascular injury, primarily in the setting of penetrating trauma.
- External hemorrhage is controlled with direct pressure; blind clamping of bleeding vessels is contraindicated due to the complex vital anatomy compressed into a relatively small space and the danger of causing further injury with a misguided surgical instrument.
- Fluid resuscitation should begin with crystalloid, followed by blood products if needed.
- The cervical spine is secured and cleared clinically or radiographically, as appropriate.
- Penetrating wounds that do not violate the platysma muscle require only standard meticulous wound care and closure. After a period of observation, asymptomatic patients with these injuries can often be discharged home with close follow-up, presuming their medical condition otherwise makes this feasible.
- Wounds that violate the platysma muscle mandate surgical consultation. These patients are admitted for surgical exploration or for further diagnostic evaluation of any significant deep structure injury (Fig. 160-1).
- Patients with blunt neck trauma initially may present with subtle signs of injury and may develop significant symptoms on a delayed basis, particularly those with a strangulation mechanism. After a period of observation, asymptomatic patients may be discharged with close follow-up, although a low threshold for admission should be maintained.
- With blunt trauma, hoarseness, dysphagia, and dyspnea are indications for more extensive evaluation. Any initial symptoms of airway, vascular, or neurologic injury demand evaluation and stabilization along with urgent surgical consultation and admission.[10]

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For further reading in *Emergency Medicine: A Comprehensive Study Guide,* 5th ed., see Chap. 217, ''Penetrating and Blunt Neck Trauma,'' by Bonnie J. Baron.

161 THORACIC TRAUMA

Kent N. Hall

EPIDEMIOLOGY

- Thoracic trauma is directly responsible for 25 percent of trauma deaths.
- Patients with isolated chest trauma have a relatively low mortality of 5 percent.
- Only 5 to 15 percent of patients with chest trauma will require a thoracotomy.

PATHOPHYSIOLOGY

- Penetrating injuries routinely result in pneumothorax or hemothorax.
- Blunt trauma to the chest causes organ damage by compression, direct trauma, or acceleration/deceleration forces.

GENERAL PRINCIPLES AND CONDITIONS

- The initial step is to evaluate the patient's effort to breathe. No effort indicates a possible central nervous system problem, such as head trauma, drugs, or spinal cord injury.
- Significant effort signals an airway obstruction, most commonly a foreign body (including the tongue) in the hypopharynx, larynx, or trachea.
- If the patient is attempting to breath and the airway is clear, thoracic injuries (flail chest, hemopneumothorax, diaphragmatic injury or parenchymal lung damage) should be considered.
- In all cases of significant respiratory distress, the airway should be secured and adequate oxygenation and ventilation provided. Indications for ventilatory support are listed in Table 161-1.
- The most frequent symptoms associated with thoracic trauma are chest pain and shortness of breath. Physical examination begins with inspection of the chest wall, looking for open (''sucking'') chest wounds, flail segments, and contusions.

TABLE 161-1 Indications for Ventilatory Support

Impaired ventilation in spite of an open airway
Shock
Multiple injuries
Coma
Flail chest
Hypoxia (P_{O_2} <50 mmHg on room air)
Drainage of hemopneumothorax
Preexisting pulmonary disease
Respiratory rate >30 breaths per minute
Relief of chest wall pain
Multiple transfusions required
Elderly

- The neck is examined for the presence of distended neck veins, which are associated with pericardial tamponade, tension pneumothorax, air embolus, and cardiac failure. Swelling and cyanosis of the face and neck often signal a superior mediastinal injury resulting in superior vena cava blockage.
- Subcutaneous emphysema from a bronchial injury or pulmonary laceration can result in severe swelling of the face and neck. Palpation of the trachea to determine its normal position, of the chest to localize areas of tenderness or crepitation, and of the abdomen for the position of abdominal contents is important.
- Auscultation of the chest should be done systematically and thoroughly. The quality and equality of breath sounds should be documented. The presence of bowel sounds in the chest may be the first indication of a diaphragmatic injury. Inequality of breath sounds may suggest a pneumothorax, a hemothorax, or an improperly inserted endotracheal tube.
- Conditions that should be recognized and treated during the initial survey include tension pneumothorax, cardiac tamponade, massive hemothorax, open pneumothorax, and flail chest.

CHEST WALL INJURIES

CLINICAL FEATURES, DIAGNOSIS, AND DIFFERENTIAL

- Simple rib fractures should be suspected in the patient with point tenderness over a rib. The goal of evaluating these injuries is to look for complications, such as pneumothorax, pulmonary contusion, or major vascular injury.
- Suspicion of a pneumothorax that is not corroborated by chest x-ray might require inspiratory and expiratory radiographic views for detection.

- Pain from rib fractures can decrease ventilation, possibly resulting in atelectasis or pneumonia.
- Fractures of the first and second ribs not due to direct trauma may be associated with significant underlying injuries, including myocardial contusions, pulmonary contusions, bronchial tears, and major vascular injuries.
- Multiple rib fractures, especially the 9th, 10th and 11th, may be associated with intraabdominal injuries. Hypotension may indicate the presence of tension pneumothorax or hemothorax.
- Segmental fractures of three or more adjacent ribs produce a flail segment of the chest, which can increase the work of breathing.
- Flail chest is recognized by paradoxical movement of the segment during the respiratory cycle (outward during expiration, inward during inspiration).
- In the case of an open pneumothorax (''sucking chest wound''), the wound is often obvious. Open chest wounds indicate invasion into the pleural space and can act as one-way valves, potentially creating a tension pneumothorax.
- Traumatic asphyxia, caused by an inability to breathe due to added weight on the chest wall, results in subconjunctival hemorrhage or petechiae and vascular engorgement, edema, and cyanosis of the head, neck, and upper extremities.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Bleeding from chest wall injuries is best controlled by direct pressure. Probing of these wounds is not recommended.
- When subcutaneous emphysema is present, an underlying pneumothorax should be presumed. If the patient is to be intubated for any reason, a chest tube should be inserted.
- For rib fractures, adequate analgesia and pulmonary toilet are the mainstays of therapy. Patients with multiple rib fractures should be admitted for 24 to 48 h if they cannot cough and clear secretions, are elderly, or have preexisting pulmonary disease.
- Sternal fractures should alert the physician to the possible presence of underlying soft tissue injuries, especially of the heart and great vessels. Therapy for these fractures includes adequate analgesia and pulmonary toilet. Admission based solely on the presence of sternal fractures is controversial.
- For flail chest, management consists of stabilizing the flail segment, either externally by using sandbags or internally by endotracheal intubation and mechanical ventilation. Nonventilatory management includes adequate analgesia, chest physiotherapy, and restriction of IV fluids.
- Indications for ventilatory support of a patient with a flail chest are listed in Table 161-2. The patient with a flail chest should be suspected of having an underlying pulmonary contusion.
- Open (sucking) chest wounds should be covered with a sterile occlusive dressing while a chest tube is inserted simultaneously at a separate site. If a tension pneumothorax develops, the occlusive dressing should be removed until the chest tube is inserted.

TABLE 161-2 Indications for Endotracheal Intubation in the Presence of a Flail Segment

Presence of shock
Three or more associated injuries
Severe head injury
Comorbid pulmonary disease
Fracture of eight or more ribs
Age >65

LUNG INJURIES

CLINICAL FEATURES, DIAGNOSIS, AND DIFFERENTIAL

- Pulmonary contusions are usually seen as opacifications of the lung on chest radiograph within 6 h of injury.
- Pneumothorax is a collection of air in the pleural space. It does not usually cause significant symptoms unless a tension pneumothorax develops, the pneumothorax occupies more than 40 percent of a hemithorax, or the patient has preexisting shock or cardiopulmonary disease.
- A pneumothorax is readily seen on expiratory chest radiograph.
- Clinical signs and symptoms of a tension pneumothorax include dyspnea, hypoperfusion, distended neck veins, deviated trachea, and decreased or absent breath sounds on the affected side.
- Hemothorax should be considered in the severely traumatized patient with unilateral decreased breath sounds and dullness to percussion. Volumes of blood as low as 200 to 300 mL are usually visualized on upright chest radiograph. However, volumes in excess of 1 L of blood may be missed on supine chest radiograph because of its appearance as diffuse haziness without a distinct air-fluid level.
- Subcutaneous emphysema in the neck and a ''crunching'' sound during systole (Hamman sign)

should make the clinician suspect pneumomediastinum. The major significance of pneumomediastinum is the possibility of associated injuries to the larynx, trachea, major bronchi, pharynx, or esophagus.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of pulmonary contusions involves maintenance of adequate ventilation, with the use of mechanical ventilation and positive end-expiratory pressure (PEEP) to optimize ventilation/perfusion matching.
- Mechanical ventilation is often required if more than 28 percent of lung volume (estimated by chest radiograph) is involved. With severe unilateral lung injury, synchronous independent lung ventilation through a double-lumen endobronchial catheter prevents overinflation of the normal lung and provides better overall oxygenation.
- If a tension pneumothorax is suspected, a large bore needle or IV catheter (14 gauge) should be inserted in the second intercostal space at the midclavicular line for needle decompression. Emergent management should not be delayed while a chest radiograph is obtained. A chest tube can be inserted for definitive treatment later.
- If a hemothorax or nontension pneumothorax is suspected in a patient with severe respiratory distress, a chest tube should be inserted prior to obtaining a chest radiograph.
- Small pneumothoraces that have not expanded on serial chest radiographs taken 6 to 12 h apart do not usually require chest tube insertion. Admission of these patients for observation and serial examinations is important.
- An ''occult pneumothorax'' [one seen on computed tomography (CT) but not on plain radiograph] does not require chest tube insertion unless the patient is on a ventilator. Insertion of a small (24 or 28 Fr) chest tube is adequate if no hemothorax is present.
- A chest radiograph should be obtained in all patients after insertion of a chest tube. Persistent air leakage and failure of the lung to expand completely is an indication for thoracotomy.
- With a massive hemothorax, the blood should be evacuated with a large-bore (38 Fr or larger) chest tube. Prophylactic antibiotics, while controversial, have led to a clear reduction in pneumonia or empyema.
- Serial examinations of the chest, including chest radiographs, and monitoring of ongoing blood loss through the chest tube are important.
- The decision to perform a thoracotomy should be based on multiple factors. A conservative approach is to perform a thoracotomy if ongoing blood loss from the chest tube exceeds 600 mL/6 h. Also, if vital signs deteriorate as a large amount of blood is being evacuated from a chest tube, the chest tube should be clamped and the patient should undergo thoracotomy.
- While adequate ventilation is being ensured, restoration of adequate tissue perfusion should be achieved. Management of patients in shock includes the insertion of two large-bore IV catheters with rapid infusion of large volumes of crystalloid or blood.

TRACHEOBRONCHIAL INJURY

- Injuries to the lower trachea and bronchi are usually caused by severe deceleration forces.
- Common presenting signs and symptoms include dyspnea, hemoptysis, subcutaneous emphysema, Hamman's sign, and sternal tenderness.
- On chest radiograph, a large pneumothorax, pneumomediastinum, deep cervical emphysema, or endotracheal tube balloon that appears round all suggest tracheobronchial injury.
- Management includes assuring adequate ventilation and referral for immediate bronchoscopy to fully evaluate and treat the injury. Intrathoracic tracheal injury is usually associated with other intrathoracic injuries and is almost invariably fatal.
- Injuries of the cervical trachea usually occur at the junction of the trachea and cricoid cartilage and are caused by direct trauma, as from a steering wheel. Inspiratory stridor is common in these patients and indicates a 70 to 80 percent obstruction.
- Oral intubation, preferably over a bronchoscope, should be attempted.

DIAPHRAGMATIC INJURY

- The majority of diaphragmatic injuries are caused by penetrating trauma. Most series report that diaphragmatic injury associated with blunt injury is usually left-sided.
- An entrance wound in the abdomen with the missile located in the chest cavity should alert the physician of a probable injury to the diaphragm.
- In the setting of blunt trauma, any abnormality of the diaphragm or lower lung fields on chest

radiograph should make the clinician consider the possibility of diaphragmatic injury.

PENETRATING INJURY TO THE HEART

CLINICAL FEATURES, DIAGNOSIS, AND DIFFERENTIAL

- All patients with hypotension and penetrating chest injury anywhere near the heart should be considered as having a cardiac injury until proven otherwise. Patients without signs of life in the field are not considered candidates for resuscitation.
- Beck's triad (distended neck veins, hypotension, and muffled heart tones) suggests a pericardial tamponade, although most patients with this type of injury do not have distended neck veins until volume resuscitation has occurred.
- Chest radiographs are rarely helpful in diagnosing acute cardiac injury, and changes on electrocardiography (ECG) are usually nonspecific. Transthoracic echocardiography is a sensitive test for the detection of pericardial fluid. Transesophageal echocardiography (TEE) is a sensitive diagnostic tool, especially if the patient is already intubated.
- Pericardiocentesis has limited value in the evaluation of the patient with possible cardiac injury due to the high incidence of false-positive and false-negative aspirates. In the hemodynamically stable patient, when echocardiography is not available, a subxiphoid pericardial window can be performed.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Management of the patient with cardiac injury includes attention to the airway, assurance of breathing, and adequate fluid resuscitation. Two large-bore IV lines should be placed, with one flowing into the venous system draining into the inferior vena cava.
- Patients in shock who do not respond to adequate fluid resuscitation and who are suspected of having a cardiac injury should undergo emergent thoracotomy.
- The patient with penetrating thoracic trauma who loses vital signs just prior to arrival or in the ED may require emergent pericardiocentesis or ED thoracotomy.
- For ED thoracotomy, an incision is made at the fifth intercostal space on the affected side. The pericardium is opened vertically, with care to avoid the phrenic nerve. The heart, lung hilum, and aorta are inspected for injuries that can be repaired primarily.
- Patients with blunt traumatic arrest, penetrating abdominal or head injuries, or prolonged arrest times receive little if any benefit from ED thoracotomy.

BLUNT INJURY TO THE HEART

CLINICAL FEATURES, DIAGNOSIS, AND DIFFERENTIAL

- The most common mechanism of injury causing cardiac trauma is a deceleration injury, as with a high-speed motor vehicle crash. In addition, compression between the sternum and vertebrae, a sudden increase in intrathoracic pressure, abdominal compression forcing abdominal contents against the heart, or strenuous cardiac massage can all cause cardiac injury.
- Blunt trauma to the heart can result in multiple types of injuries, including rupture of an outer chamber wall, septal rupture, valvular injuries, direct myocardial injury, laceration of a coronary artery, or pericardial injury.
- *Blunt myocardial injury* (BMI) is the term currently in use to describe injuries previously termed myocardial concussions and myocardial contusions. The most common clinical features associated with a significant BMI are tachycardia out of proportion to blood loss, arrhythmias (especially premature ventricular contractions and atrial fibrillation), and conduction defects.
- Cardiac enzymes, including CPK-MB and the troponins, have been found to be nonspecific in making the diagnosis of significant BMI. Echocardiography does not seem to be very useful in evaluating the patient with suspected BMI, although it is the most widely used modality.
- BMI causes death very rarely, and the incidence of clinically significant dysrhythmias and other cardiac complications is low. Management of the patient with a significant BMI calls for the administration of supplemental oxygen and analgesics, treatment of significant cardiac dysrhythmias, and the administration of fluids or inotropic agents for hypotension.
- Cardiac rupture results in immediate death in 80 to 90 percent of cases. Patients with cardiac rupture who arrive at the hospital alive usually have a right atrial tear.
- Shock that is out of proportion to the degree of recognized injury and shock that persists despite

control of hemorrhage elsewhere as well as volume expansion should make one consider the possibility of cardiac rupture.
- Immediate left anterior thoracotomy may be lifesaving in these cases. Septal defects and valve injuries are rare after blunt trauma but should be considered if a murmur exists in the setting of possible cardiac damage.
- Signs of a ventricular septal defect include severe early hypoxemia with a relatively normal chest radiograph, heart murmur, and an injury pattern on ECG.
- Rupture of the aortic valve is the most common valvular lesion, followed by rupture of the papillary muscle or chordae tendineae of the mitral valve.

PERICARDIAL INJURY

- A pericardial effusion may develop acutely or over time. The rate of fluid collection influences the onset and severity of symptoms.
- Evidence of acute pericardial injury is usually seen on the ECG as diffuse ST-segment elevation.
- Most patients are asymptomatic, and no specific therapy is required. A tear of the parietal pericardium at the apex of the heart may result in sudden severe shock and cardiac arrest if the heart herniates through the hole.

POSTPERICARDIOTOMY SYNDROME

- This is seen in patients 2 to 4 weeks after heart surgery or trauma. Classically, patients will have chest pain, fever, and pleural or pericardial effusions. Friction rubs, arthralgia, and pulmonary infiltrates may also be seen.
- The ECG will often show diffuse ST–T-wave changes consistent with pericarditis.
- Management is symptomatic, with salicylates and rest often the only therapy required. Occasionally glucocorticoids are needed.

PENETRATING TRAUMA TO THE GREAT VESSELS OF THE CHEST

CLINICAL FEATURES, DIAGNOSIS, AND DIFFERENTIAL

- When a stab wound causes injury to the great vessels of the chest, survival is generally much higher than when such an injury is caused by a gunshot wound. Simple lacerations of the great vessels can lead to exsanguination, tamponade, hemothorax, air embolism, or development of an arteriovenous (AV) fistula or false aneurysm.
- In general, these wounds should not be probed. Assessment of bilateral upper extremity pulses for equality is important, as a large mediastinal hematoma may compress the subclavian vessels. The entire chest should be auscultated for bruits that may indicate a false aneurysm or AV fistula.
- Radiographic evaluation starts with a chest radiograph. In addition to evaluation for pneumothoraces, widening of the upper mediastinum may indicate injury to brachiocephalic vessels. CT scans are rarely performed immediately for penetrating wounds of the chest. However, in the stable patient, a CT scan can help localize hematomas adjacent to great vessels. The use of IV contrast helps further evaluate these structures and may demonstrate a vascular defect or false aneurysm. The major role of CT is as a screen for great vessel injury.
- Arteriograms are most helpful in identifying major intrathoracic vascular injuries within hematomas, especially those resulting from penetrating injury to the lower neck.
- Contrast swallow using meglumine diatrizoate (Gastrografin) may be performed on stable patients to evaluate the integrity of the esophagus. Endoscopy is sometimes used in hemodynamically stable patients with penetrating wounds of the chest or lower neck.
- Recently, use of TEE has been advocated, especially when the CT scan or aortogram are equivocal for injury to the aorta.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Early endotracheal intubation should be performed in patients with penetrating injuries to the thoracic inlet to avoid the problems associated with expanding hematomas distorting the airway.
- The patient in severe shock (systolic BP <60 mmHg) should have immediate surgery, with aggressive fluid resuscitation waiting until after major bleeding sites are controlled. If the systolic blood pressure (BP) is 60 to 90 mmHg, 2 to 3 L of crystalloid should be given rapidly. If the patient remains hypotensive, immediate surgery is required.
- If the patient did not have ''signs of life'' in the field, no resuscitative efforts are warranted. However, if the patient ''lost vital signs'' immediately

prior to arriving or in the ED, emergent ED thoracotomy is indicated.
- Bullets that enter great vessels can embolize to distant sites and should be sought by using multiple radiographs or fluoroscopy.[1–4]

BLUNT TRAUMA TO THE GREAT VESSELS OF THE CHEST

CLINICAL FEATURES, DIAGNOSIS, AND DIFFERENTIAL

- Some 90 percent of patients with injury to great vessels from blunt trauma who arrive at the hospital alive have an injury at the isthmus of the aorta (between the left subclavian artery and ligamentum arteriosum). Other common sites of injury are the innominate or left subclavian artery at their origins or a subclavian artery over the first rib.
- This injury can occur even when no external signs of trauma exist. Therefore, it should be suspected in any patient with a high-speed deceleration mechanism of injury or high-speed impact from the side. These patients complain primarily of their associated injuries. Retrosternal or interscapular pain, often described as a ''tearing'' sensation, may be the only initial indication.
- One-third of patients with blunt trauma to the aorta have no external evidence of thoracic injury. Findings that suggest an aortic injury include a difference in blood pressure or pulse amplitude between the upper and lower extremities, acute-onset upper extremity hypertension, or a harsh systolic murmur across the precordium or in the interscapular area.
- Findings associated with traumatic rupture of the aorta on plain chest radiograph are seen in Table 161-3. The most frequent radiologic finding is mediastinal widening. The best chest radiograph is an upright posteroanterior (PA) view taken from 72 in. with the patient leaning forward 10 to 15 degrees.
- The most specific radiographic sign of traumatic aortic rupture is deviation of the esophagus more than 1 to 2 cm to the right of the spinous process of T4. Up to one-third of patients with traumatic aortic rupture will have a normal chest radiograph initially.
- TEE is a highly sensitive diagnostic modality to evaluate for traumatic aortic rupture. It can be used at the bedside while the resuscitation is ongoing and yields results that are at least as good as those of aortography. It visualizes the aortic isthmus and descending aorta very well and defines the pericardial cavity, cardiac valves, and pulmonary veins as well as regional wall motion.
- Late-generation helical CT scans of the chest have been recommended as a tool to screen for traumatic aortic rupture in selected patients. Selection guidelines include patients with equivocal histories and equivocal radiographs who have a low probability for injury to the other great vessels, are hemodynamically stable, and are capable of tolerating two dye loads (one for the CT and one for the aortogram, if necessary).
- The presence of a mediastinal hematoma is an indication for aortography. Magnetic resonance imaging (MRI) cannot be recommended as a tool in the evaluation of patients with suspected traumatic aortic rupture, mostly because of the need for the patient to spend long intervals in an isolated setting. Aortography is the traditional ''gold standard'' for diagnosing aortic rupture.
- Injury to the ascending aorta usually results in immediate death. These injuries tend to occur within the pericardium and result in cardiac tamponade. If there is an associated valvular injury, a murmur of aortic insufficiency may be heard. The aortogram shows a pseudoaneurysm, possibly with aortic insufficiency.
- Injuries to the innominate artery are associated with rib fractures, flail chest, hemopneumothorax, fractured extremities, head injuries, facial fractures, and abdominal injuries. The diagnosis is difficult because there are no characteristic physical findings except for some decrease in the right radial or brachial pulse. Findings on chest radiograph are similar to those with traumatic rupture of the aorta except the mediastinal hematoma is usually higher and the esophagus is pushed to the left. Aortography is generally required for the diagnosis to be established.
- Subclavian artery injuries are most often caused by fractures to the first rib or clavicle. Absence of a radial pulse on the affected side is the most

TABLE 161-3 Chest Radiographic Findings Associated with Traumatic Rupture of the Aorta

Superior mediastinal widening (>8.0–8.5 cm)
Deviation of esophagus and/or trachea at T4
Obscuration of aortic knob and/or descending aorta
Displacement of left mainstem bronchus more than 40 degrees below horizontal
Obscuration of medial aspects of left upper lobe
Widening of the paratracheal stripe
Displacement of the paraspinal lines (either left or right)
Fracture of first or second rib
Apical cap

important sign. A pulsatile mass or bruit at the base of the neck is suggestive of this injury. Associated injury to the brachial plexus occurs in 60 percent of patients. A Horner's syndrome may occur on the affected side as well.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Sedatives, analgesics, vasodilators, and beta-adrenergic blockers may be required to control the patient's blood pressure.
- Insertion of a nasogastric tube is important, but this must be done with extreme care to avoid making the patient gag.
- Thoracotomy is the accepted standard of treatment. Delayed repair may be more appropriate in patients who are at extremely high operative risk or when conditions for surgery are not optimal.

ESOPHAGEAL AND THORACIC DUCT INJURIES

- Injuries to these structures are rare. If an esophageal injury is suspected, an esophagram should be performed. Most radiologists recommend use of meglumine diatrizoate (Gastrografin) because it causes less of an inflammatory reaction than barium. However, the false-negative rate with this contrast agent is as high as 25 percent. Flexible esophagoscopy can also be performed but carries a false-negative rate of 20 percent.
- Thoracic duct injuries result in a chylothorax in the right hemithorax.

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162 ABDOMINAL TRAUMA

O. John Ma

EPIDEMIOLOGY

- The most common cause of death and disability from nonintentional injury is the motor vehicle crash. Falls are the second leading cause of accidental death in the United States.

PATHOPHYSIOLOGY

- The injury pattern of blunt abdominal trauma is often diffuse. Blunt injuries involve a compression or crushing mechanism by energy transmission directly to the patient. If the compressive, shearing, or stretching forces exceed the tolerance limits of the tissue or organ, the tissues are disrupted.
- Injury also can result from movement of organs within the body. Some organs are rigidly fixed, whereas others are motile. Typical examples in the abdomen include mesenteric or small bowel injuries, particularly at the ligament of Treitz or at the junction of the distal small bowel and right colon.
- A gunshot may cause injury directly, as when the bullet itself strikes an organ, or secondarily, as when tissues are injured by missiles such as bone or bullet fragments or by energy transmission from the bullet.

CLINICAL FEATURES

Solid Visceral Injuries

- Injury to the solid organs cause morbidity and mortality primarily as a result of acute blood loss.
- The spleen is the most frequently injured organ in blunt abdominal trauma and injury to it is commonly associated with other intraabdominal injuries. The liver is also commonly injured in both blunt and penetrating injuries.

- Tachycardia, hypotension, and acute abdominal tenderness are the primary findings on physical examination. Kehr's sign, representing referred left shoulder pain, is a classic finding in splenic rupture. Lower left rib fractures should heighten clinical suspicion for splenic injury.
- It is important to note that patients with solid organ injury may occasionally present to the emergency department (ED) with minimal symptoms and nonspecific findings on physical examination. This is especially true when these are associated with distracting injuries, central nervous system (CNS) trauma, or intoxication.

Hollow Viscus Injuries

- These injuries produce symptoms by the combination of blood loss and peritoneal contamination. Perforation of the stomach, small bowel, or colon is accompanied by blood loss from a concomitant mesenteric injury.
- Gastrointestinal (GI) contamination will produce peritoneal signs over a period of time. Patients with head injury, distracting injuries, or intoxication may not exhibit peritoneal signs initially.
- Injuries to the small bowel and colon are most frequently the result of penetrating trauma. However, a deceleration injury can cause a bucket-handle tear of the mesentery or a blowout injury of the antimesenteric border.

Retroperitoneal Injuries

- Signs and symptoms of retroperitoneal injuries may be subtle or absent upon initial presentation to the ED. Duodenal injuries are most often associated with high-speed vertical or horizontal decelerating trauma. Clinical signs of duodenal injury are often slow to develop. Duodenal injuries may range in severity from an intramural hematoma to an extensive crush or laceration.
- Duodenal ruptures are usually contained within the retroperitoneum. They may present with abdominal pain, fever, nausea, and vomiting, although these may take hours to become clinically obvious.
- Pancreatic injury is most common with penetrating trauma. It also occurs after a severe crush injury. The classic case is a blow to the midepigastrium from a steering wheel or the handlebar of a bicycle.

Diaphragmatic Injuries

- The presentation of diaphragmatic injuries is often insidious. Only occasionally is the diagnosis obvious, when bowel sounds can be auscultated in the thoracic cavity.
- On chest radiograph, with herniation of abdominal contents into the thoracic cavity, the diagnosis is confirmed. In most cases, however, there is no herniation, and the only finding on the chest radiograph is blurring of the diaphragm or an effusion.
- This injury is most often diagnosed on the left.

DIAGNOSIS AND DIFFERENTIAL

Plain Radiographs

- For blunt trauma, routine use of plain abdominal radiographs is not a cost-effective or prudent method for evaluating the trauma patient. A chest radiograph is helpful in evaluating for herniated abdominal contents in the thoracic cavity and for evidence of free air under the diaphragm.
- An anteroposterior (AP) pelvic radiograph is important for identifying pelvic fractures, which can produce significant blood loss and may be associated with intraabdominal visceral injury.

Diagnostic Peritoneal Lavage

- Diagnostic peritoneal lavage (DPL) remains an excellent screening test for evaluating abdominal trauma. Its advantages include its sensitivity, availability, the relative speed with which it can be performed, and a low complication rate. Disadvantages include the potential for iatrogenic injury, its misapplication for evaluation of retroperitoneal injuries, and its lack of specificity.
- For blunt trauma, indications for DPL include (1) patients who are too hemodynamically unstable to leave the ED for computed tomography (CT) scanning or who have a physical examination that is unreliable secondary to drug intoxication or CNS injury and (2) unexplained hypotension in patients with an equivocal physical examination.
- In penetrating trauma, DPL should be performed when it is not clear that exploratory laparotomy will be required. DPL is useful in evaluating patients sustaining stab wounds where local wound exploration indicates that the superficial muscle fascia has been violated. Also, it may be useful in confirming a negative physical examination when tangential or lower chest wounds are involved.
- The DPL is considered positive if more than 10 mL of gross blood is aspirated immediately, the red blood cell count is $>100{,}000/\mu L$, the white blood cell count is $>500/\mu L$, bile is present, or vegetable matter is present.
- The only absolute contraindication to DPL is when surgical management is clearly indicated, in which case the DPL would delay patient transport to the operating room. Relative contraindications include patients with advanced hepatic dysfunc-

TABLE 162-1 Indications for Laparotomy

	BLUNT	PENETRATING
Absolute	Anterior abdominal injury and hypotension	Injury to abdomen, back and flank with hypotension
	Abdominal wall disruption	Abdominal tenderness
	Peritonitis	GI evisceration
	Free air on chest x-ray	Positive DPL
	CT diagnosed injury requiring surgery, i.e., pancreatic transection; duodenal rupture	(GSW) High suspicion for transabdominal trajectory
		CT diagnosed injury requiring surgery, i.e., ureter or pancreas
Relative	Positive DPL or FAST in stable patient	Positive local wound exploration (SW)
	Solid visceral injury in stable patient	
	Hemoperitoneum on CT without clear source	

ABBREVIATIONS: CT = computed tomography; DPL = diagnostic peritoneal lavage; FAST = focused abdominal sonography for trauma; GI = gastrointestinal; GSW = gunshot wound; SW = stab wound.

tion, severe coagulopathies, previous abdominal surgeries, or a gravid uterus.

ULTRASONOGRAPHY

- Recent literature has demonstrated that the focused abdominal sonography for trauma (FAST) examination, like DPL, is an accurate screening tool for abdominal trauma. Advantages of the FAST examination are that it is accurate, rapid, noninvasive, repeatable, and portable. Another advantage of the FAST examination is that it is capable of evaluating for free pericardial and pleural fluid.
- Disadvantages include its inability to determine the exact etiology of the free intraperitoneal fluid and that it is operator-dependent. Indications for FAST examination are the same as for DPL.

COMPUTED TOMOGRAPHY

- The abdominal CT scan has a greater specificity than DPL and ultrasonography, thus making it the initial diagnostic test of choice at most centers. Oral and IV contrast material should be given to provide optimal resolution.
- Advantages of the CT scan include its ability to locate intraabdominal lesions precisely before surgery, to evaluate the retroperitoneum, to identify injuries that may be managed nonoperatively, and noninvasiveness.
- The disadvantages of CT scanning are its expense, the time required to perform the study, the need to transport the trauma patient to the radiology suite, and the need for contrast agents.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Patients should be administered oxygen and have cardiac monitoring and two IV lines secured.
- For hypotensive abdominal trauma patients, resuscitation with IV crystalloid fluid is indicated. Transfusion with O-negative or type-specific packed red blood cells should be considered in addition to crystalloid resuscitation.
- Table 162-1 lists the indications for exploratory laparotomy. When a patient presents to the ED with an obvious high-velocity gunshot wound to the abdomen, DPL or the FAST exam should not be performed as it will only delay transport of the patient to the operating room. If organ evisceration is present, it should be covered with a moist, sterile dressing prior to surgery.
- For an equivocal stab wound to the abdomen, surgical consultation for local wound exploration is indicated. If the wound exploration demonstrates no violation of the anterior fascia, the patient can be discharged home safely.[1–5]

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163 FLANK AND BUTTOCK TRAUMA

William R. Dennis, Jr.

EPIDEMIOLOGY

- Penetrating trauma to the flank and buttock must be carefully evaluated in order to determine whether there is retroperitoneal injury that may mandate operative intervention.
- Penetrating injuries to the buttock are relatively uncommon. With appropriate management, the mortality is low; however, case reports indicate that there is a potential for missed injury to the bowel or major vessels if the clinician is not thorough in the evaluation.

PENETRATING TRAUMA TO THE FLANK

PATHOPHYSIOLOGY

- The flank is defined as the area between the anterior and posterior axillary lines, superiorly bordered by the sixth rib and inferiorly bordered by the iliac crest.
- A delay in diagnosis of duodenal, colonic, rectal, renal, pancreatic, or major vascular injuries may result in the late appearance of septic or hemorrhagic shock.

CLINICAL FEATURES

- The organs most commonly injured by penetrating trauma to this area include the liver, kidney, colon, duodenum, and pancreas.

DIAGNOSIS AND DIFFERENTIAL

- In the hemodynamically stable patient with no obvious visceral injury, adjunctive diagnostic tests such as diagnostic peritoneal lavage (DPL), computed tomography (CT) scan, or portable ultrasound are useful.
- Wound exploration is of limited value in penetrating flank trauma. Deep wound exploration often leads to further hemorrhage and tissue damage.
- Diagnostic peritoneal lavage is highly accurate for detecting intraperitoneal injuries, but poor for detecting retroperitoneal injuries. It may be indicated when injury to the diaphragm or hollow viscus is suspected.
- Computed tomography scanning is accurate in detecting intraperitoneal and retroperitoneal injuries. Oral, intravenous, and rectal contrast are necessary for an optimal study. Particular attention should be paid to the presence of intraperitoneal fluid and edema of the bowel wall. The latter may represent bowel perforation, although contrast leak may not necessarily be visible.[1]
- Portable ultrasonography is sensitive and specific for determining the presence of free intraperitoneal fluid which, in the unstable patient, is sufficient to recommend operative intervention.[2]
- A chest radiograph should be obtained to evaluate for intrathoracic involvement.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- A complete blood cell count (CBC), type and cross-match, urinalysis, and rectal examination should be performed on all patients.
- Selective management with early CT scanning is advocated since it results in fewer nontherapeutic laparotomies with no increase in untoward outcomes.[3] Selective management is also appropriate in stable patients with stab wounds. Exploratory laparotomy is indicated for patients with hemodynamic instability, evisceration, peritonitis, intraperitoneal free air, or transabdominal missile path. All patients should be admitted, with the exception of those patients with clearly defined superficial wounds.

PENETRATING TRAUMA TO THE BUTTOCK

PATHOPHYSIOLOGY

- Gunshot wounds to the buttocks have a greater potential for injury than stab wounds. The thick

musculature and fat over the buttocks normally protects the gastrointestinal, genitourinary, and neurologic systems from injury in all except the deepest stab wounds.
- A delay in the diagnosis of colonic or rectal injury will contribute to increased mortality and morbidity.

CLINICAL FEATURES

- If there is any concern of injury to the rectum because of blood noted on the rectal examination or because of the trajectory of the bullet, proctosigmoidoscopy should be performed.[4–6]
- Vascular injuries should be suspected when an enlarging hematoma, bruit, or change in peripheral pulses is present. Vascular injury to gluteal or internal iliac arteries has been reported from gluteal-penetrating wounds and may lead to profuse hemorrhage.[7,8]
- Neurologic injuries may present with paresthesias, sensory loss, or motor weakness.

DIAGNOSIS AND DIFFERENTIAL

- Laboratory studies include a CBC, type and crossmatch, and urinalysis. After the rectal examination, proctosigmoidoscopy should be performed when rectal and sigmoid injuries are clinically suspected.
- The presence of gross hematuria should be investigated with retrograde uretherogram and cystogram.
- Pelvic x-rays will reveal bony injury and suggest possible missile path. A CT scan of the pelvis may reveal colon, urinary tract, or vascular injury.
- Angiography is indicated when vascular injury is suspected.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- If a uretheral injury is suspected, a Foley catheter should not be inserted.
- Broad-spectrum antibiotics (e.g., Zosyn 3.375 g IV) are indicated for rectal injuries.
- Approximately 30 percent of patients who present with gunshot wounds to the buttocks require surgery. Indications for surgical intervention include peritonitis, hemodynamic instability, obvious signs of gastrointestinal bleeding, a positive finding with proctosigmoidoscopy, gross hematuria, entrance wound above the level of the greater trochanter, and a transpelvic bullet course.[4,5] Significant vascular or nerve injuries (sciatic or femoral nerves) require early operative intervention.

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164 GENITOURINARY TRAUMA

Gary M. Gaddis

EPIDEMIOLOGY

- Genitourinary injuries occur in 2 to 5 percent of adult trauma patients, with the vast majority due to blunt trauma.
- Over 80 percent of patients with renal injury have concurrent serious injuries, one-third of which are

life-threatening. Contusions account for 92 percent of renal injuries.
- Approximately 80 percent of urogenital injuries involve the kidney and 10 percent involve the bladder.
- About 10 percent of pediatric abdominal trauma victims will have renal system injury.[1,2]

PATHOPHYSIOLOGY

- A sudden deceleration mechanism from a fall or motor vehicle crash is associated with renal pedicle and renal vascular injuries.
- Blunt trauma is more commonly involved in renal contusions and fractures, intraperitoneal and extraperitoneal bladder ruptures, and lesions associated with pelvic fractures. These include bladder lacerations, bladder contusions, and posterior urethral injuries.
- Straddle injuries, instrumentation trauma, or kicks to the groin classically involve the anterior urethra (beyond the prostate) of males or the female urethra. Such injuries may injure the penis or the scrotum and its contents.
- Penile injuries include traumatic corpus cavernosum rupture from a forcible direct impact, bending of an erect penis, zipper entrapment, or self-inflicted and assault-related amputation or laceration.

CLINICAL FEATURES

- Lower rib, lower thoracic, or lumbar vertebral fractures may be associated with renal or ureteral injury. Flank ecchymosis or masses may suggest renal injury.
- Pelvic fractures and perineal straddle injuries should raise suspicion of bladder or urethral trauma.
- The following should suggest urethral injury: blood at the urethral meatus; penile, scrotal, or perineal hematoma; or a boggy or high-riding prostate on rectal exam. These findings are a contraindication for urethral catheterization in order to prevent converting a partial urethral laceration into a complete urethral transection.
- Female urethral injuries often accompany extensive pelvic fractures. Eighty percent of female urethral injuries present with vaginal bleeding.
- For women with vaginal bleeding, a speculum examination is required to evaluate for vaginal lacerations. Labial lacerations or hematomas mandate bimanual vaginal examination.
- Scrotal ecchymoses or lacerations may suggest testicular disruptions.
- Lacerations in the folds of the buttocks may denote open pelvic fractures. Perineal lacerations also may indicate open pelvic fractures. These lacerations should not be probed because disrupting a clot may precipitate profuse hemorrhage.

DIAGNOSIS AND DIFFERENTIAL

- Penetrating injury is likely to injure any nearby structure. Ureters are more likely to be injured by penetrating trauma and are seldom injured in blunt trauma. Extravasation of contrast on intravenous pyelogram (IVP) usually diagnoses ureteral injury; however, this is not infallible.[3]
- With penetrating trauma, there is no correlation between the degree of hematuria and injury severity.[4]
- In children, the degree of hematuria does seem to correlate with the degree of injury.[5] Pediatric patients with hematuria, even if hemodynamically stable, should undergo imaging studies if they have >50 red blood cells (RBCs) per high power field (hpf).[5] Some advocate that all pediatric patients with microscopic hematuria undergo imaging studies.[6,7]
- Hemodynamic instability mandates imaging studies to evaluate the cause of any hematuria. More life-threatening injuries take precedence over evaluation for genitourinary injuries.
- The dipstick evaluation for microscopic hematuria can be misleading due to myoglobinuria.
- The first voided urine can help localize the injury. Initial hematuria suggests injury to the urethra or prostate, whereas terminal hematuria suggests bladder neck trauma. Continuous hematuria may be due to bladder, ureteral, or renal injury.
- While there is no clinically validated upper limit of microscopic hematuria beyond which imaging studies are mandated, clinicians should strongly consider imaging blunt trauma patients with microscopic hematuria >50 RBCs/hpf.
- With blunt trauma, the degree of hematuria does not correlate with the degree of urinary tract injury. In adults, if hemodynamic compromise is absent, isolated microscopic hematuria is not likely to represent significant blunt injury. Only about 1 in 500 blunt trauma patients with microscopic hematuria has significant genitourinary injury.[8]
- Gross hematuria implies the need for a diagnostic imaging study that is chosen based on other findings. For instance, with a pelvic fracture, urethro-

TABLE 164-1 Grading of Renal Injuries

GRADE	INJURY
I	Contusion (microscopic or gross hematuria, with normal urologic study results) Subscapsular, nonexpanding hematoma without laceration
II	Parenchymal laceration <1.0 cm depth limited to cortex, no extravasation Nonexpanding hematoma, confined to retroperitoneum
III	Parenchymal laceration >1 cm depth with extravasation or collecting system rupture
IV	Laceration extending through to collecting system Vascular pedical injury, hemorrhage contained
V	Shattered kidney Avulsed hilum (devascularized kidney)

SOURCE: From Moore EE, Shackford SR, Pachter HL, et al: Organ injury scaling: Spleen, liver, kidney. *J Trauma* 29:1664, 1989, with permission.

graphy and cystography should be considered. With flank ecchymosis, computed tomography (CT) scaling of the abdomen to image the kidneys is often indicated.

- Abdominal CT scan is a useful diagnostic tool for evaluating hemodynamically stable patients with either microscopic or gross hematuria, and it may also identify associated abdominal injuries.
- Hemodynamically unstable patients should undergo a ''one shot'' IVP, either in the emergency department or the operating room.
- A systolic blood pressure <70 mmHg may cause the kidney to be poorly imaged and increases the risk for dye-induced nephrotoxicity. Renal injuries are graded by degree of injury (Table 164-1).
- Cystography or CT cystography is useful for evaluating bladder injury.
- Contrast should be administered from a position 60 cm above the bladder to approximate bladder pressures obtained during voiding and to decrease the likelihood of a false-negative test.
- Testicular ultrasound can be useful for determining the type and extent of testicular or scrotal injury.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Emphasis should be placed on securing the ABCs and identifying and stabilizing any life-threatening injuries. Laboratory studies should include type and cross-match, complete blood cell count, electrolytes, blood urea nitrogen, creatinine, coagulation studies, and urinalysis.
- Patients with grades 1 and 2 renal injuries should be admitted for observation. These injuries are managed nonoperatively, with follow-up urinalysis within 2 to 3 weeks. Isolated grades 3 and 4 renal injuries mandate admission; if managed nonoperatively, these require serial urinalysis and hematocrit determinations. Grade 5 renal injuries require operative management.
- Ureteral injuries, which are usually associated with penetrating trauma, require operative management.
- Bladder contusions are managed expectantly. Intraperitoneal bladder rupture and penetrating bladder injuries require operative management. Extraperitoneal bladder rupture is managed with catheter drainage.
- Partial posterior urethral lacerations may be managed by suprapubic drainage, while complete lacerations require surgery. Anterior urethral contusions can be managed expectantly. Partial anterior lacerations require an indwelling catheter or suprapubic cystostomy. Complete lacerations require end-to-end anastomosis. Urologic consultation for any urethral procedure is prudent to avoid converting partial to complete lacerations.
- Testicular contusions can be managed expectantly. Testicular rupture and penetrating trauma through the tunica vaginalis require operative exploration and repair.
- Penile lacerations and amputations require operative care. Penile fractures, due to corpus cavernosum rupture, require immediate surgery to drain clotted blood, repair the tunica albuginea, and repair any associated urethral injuries.

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165 PENETRATING TRAUMA TO THE EXTREMITIES

Gary M. Gaddis

EPIDEMIOLOGY

- Penetrating trauma causes 82 percent of all vascular injuries in the extremity. The majority, by a ratio of 4 : 1, are missile injuries.
- Since 1950, advances in wound care have decreased the rate of amputation from 50 to 5 percent for penetrating extremity wounds with vascular involvement.[1] However, 15 to 40 percent have long-term morbidity due to nerve damage, fractures, wound infections, open-joint injuries, and compartment syndromes.[2]

PATHOPHYSIOLOGY

- Tissue damage from missile injury depends on factors such as projectile shape, mass, composition, angle of impact, velocity, and flight characteristics (whether the missile travels sideways in flight).[3]
- Pressure waves accelerating radially away from the point of penetration (''temporary cavity'') displaces tissue and causes additional damage with missile wounds.[4]
- Other factors impacting amount of tissue damage include degree of comminuted fracture and displacement of bone, blood loss within a limb, and nerve or vascular injury.

CLINICAL FEATURES

- ''Hard signs'' of vascular injury, seen in fewer than 6 percent of cases, include absent or diminished distal pulses, obvious arterial bleeding, expanding or pulsatile hematoma, audible bruit, palpable thrill, or evidence of distal ischemia.
- ''Soft signs'' are much more common, and include small stable hematomas, injury to a nerve, unexplained hypotension, history of hemorrhage, proximity of the injury to a major vessel, or a complex bony fracture.
- Extremity color, temperature, and capillary refill may provide clues to subtle vascular injury, but are not completely reliable.
- A difference of >20 mmHg between blood pressures of the upper extremities is indicative of upper extremity arterial injury.
- Injuries to nerves are the most common cause of long-term morbidity. However, 70 percent of peripheral nerve injuries result in complete recovery within 6 months.
- Compartment syndrome is a potential complication of any penetrating injury to the extremities.

DIAGNOSIS AND DIFFERENTIAL

- Plain radiographs should be obtained in all cases of penetrating extremity trauma, including at least one joint above and one joint below the site of injury. Radiographs help detect bone fractures, foreign bodies, and joint space involvement.
- Foreign bodies, such as shotgun pellets, can enter an artery and embolize distally.
- Radiographic evidence of metal or gas in a joint is indicative of joint space involvement.
- Angiography can detail the extent, nature, and location of vascular injury. Angiography is most useful in patients with shotgun wounds, multiple or severe fractures, thoracic outlet wounds, extensive soft tissue injury, or significant underlying vascular disease.
- With the ''hard signs'' of vascular injury, preoperative angiography is indicated unless the patient is taken directly to the operating room.
- With the ''soft signs'' of vascular injury, 10 to 20 percent of angiograms are abnormal. Only 2 percent of these, however, will require surgical repair, and expectant management is appropriate.
- Duplex ultrasonography can image vessels with similar resolution, but with greater safety and speed than angiography. Ultrasonography is 96 percent accurate, but is very operator-dependent. Ultrasonography has not been tested in patients

with large open wounds or fractures, large hematomas, bulky dressings, or traction devices.[1,5,6]
- Capillary refill, taken alone, is an unreliable marker for vascular injury.
- Ankle brachial index (ABI) or wrist brachial index should be determined using doppler devices. These tests help diagnose occlusive injury, but do not detect nonocclusive injuries such as intimal flaps or pseudoaneurysm. The patient should have blood pressure evaluated in all four extremities while supine.
- An ABI measurement involves inflation of a standard adult blood pressure cuff just above the malleoli, monitoring blood flow over the anterior tibial artery. The cuff should be inflated to about 30 mmHg above the point at which flow is occluded. The ankle systolic pressure is the point at which flow is next heard, while slowly deflating the cuff 2 to 5 mmHg/s. The upper extremity blood pressure is determined similarly. Ankle brachial index equals ankle systolic blood pressure divided by the higher of the two upper extremity systolic blood pressures.
- An ABI >1.0 is normal. Values between 0.5 and 0.9 indicate injury to a single arterial segment, and values <0.5 indicate severe arterial injury or injury to multiple segments. For values between 0.9 and 1.0, the sensitivity and specificity of ABI testing vary greatly.[7,8]
- Preexisting peripheral vascular disease and hypothermia can adversely affect ABI accuracy.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Life-threatening injuries should be addressed first. Routine wound care and tetanus prophylaxis are performed, as needed.
- Fractures associated with penetrating trauma are treated as open fractures, with appropriate debridement and intravenous antibiotics administered.
- Direct pressure should be used to stop arterial bleeding. Clamping or ligating arteries should be avoided since they can injure accompanying nerves.
- Hard signs of arterial injury require either immediate surgical intervention or angiography and surgical consultation.
- Soft signs rarely denote need for immediate surgery, but should prompt in-patient observation.
- Wounds requiring exploration in the emergency department include those with suspected foreign bodies, ligamentous involvement, or minor venous bleeding.
- Wound exploration to control arterial or major venous bleeding should be reserved for the operating room.
- Orthopedic consultation is required when penetrating trauma causes joint space involvement.
- Patients without signs of vascular compromise, compartment syndrome, or significant soft tissue defect should be observed for 3 to 12 h; they may be discharged home with close follow-up if serial examinations are normal.

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Section 21
FRACTURES AND DISLOCATIONS

166 EARLY MANAGEMENT OF FRACTURES AND DISLOCATIONS

Michael P. Kefer

PATHOPHYSIOLOGY

- Bone fracture results in severing of the microscopic vessels crossing the fracture line, which cuts off blood supply to the involved fracture edges.
- Callus formation ensues and becomes progressively more mineralized.
- Necrotic edges of the fracture are gradually resorbed by osteoclasts. This explains why some occult fractures are not immediately detected on radiographs, but then appear several days later, after this resorption process is well established.
- Remodeling deposits new bone along the lines of stress. This process often lasts years.

CLINICAL FEATURES

- Knowing the precise mechanism of injury, listening carefully to the patient's symptoms, and performing a careful physical examination are important in diagnosing fracture or dislocation. Pain may be referred to an area distant from the injury (e.g., hip injury presenting as knee pain). If key aspects of the history and physical examination are not appreciated, all necessary radiographs may not be obtained and the diagnosis missed.
- Radiologic evaluation is based on the history and physical examination, not simply on where the patient reports pain. Radiographs of all long bone fractures should include the joints proximal and distal to the fracture to evaluate for coexistent injury. A negative radiograph does not exclude a fracture. This commonly occurs with scaphoid, radial head, or metatarsal shaft fractures. The emergency department (ED) diagnosis is often clinical and is not confirmed for 7 to 10 days, when enough bone resorption has occurred at the fracture site to detect a lucency on x-ray.
- An accurate description of the fracture to the consultant should include the following details:

  Open versus *closed*.

  Location: midshaft, junction of proximal and middle or middle and distal third, or distance from bone end; anatomic bony reference points should be used when applicable (e.g., humerus fracture just above the condyles is described as supracondylar, as opposed to distal humerus).

  Orientation of fracture line: transverse, spiral, oblique, comminuted, or segmental (single large segment of free-floating bone).

  Displacement: amount and direction the distal fragment is offset in relation to the proximal fragment.

  Separation: degree the two fragments have been pulled apart; unlike displacement, alignment is maintained.

  Shortening: reduction in bone length due to impaction or overriding fragments.

  Angulation: angle formed by the fracture segments; describe the degree and direction of deviation of the distal fragment.

  Rotation: degree the distal fragment is twisted on the axis of the proximal fragment.

  Fractures involving a joint: associated disruption

of proper joint alignment should be described as fracture-dislocation or fracture-subluxation; for intraarticular fracture, the percent of the joint surface involved should be described.
- Complications of fractures and dislocations include the following:

  Nerve or vascular injury may result from traction, compression, or laceration by the fracture or dislocation.

  Compartment syndrome that presents with the 5 classic signs of pain, pallor, paresthesias, pulselessness, and paralysis is well advanced. This diagnosis should be made presumptively, based on the character of pain, the earliest sign.

  Fat embolus may result from fracture of large bone, such as the femur.

  Long-term complications of fracture include malunion, nonunion, avascular necrosis, arthritis, and osteomyelitis.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Swelling should be controlled with application of cold packs and elevation. Analgesics should be administered as necessary. Any objects such as rings or watches that may constrict the injury as swelling progresses should be removed. The patient should have nothing by mouth if anesthesia will be required.
- Prompt reduction of fracture and dislocation is indicated to restore circulation to a pulseless distal extremity; alleviate pain; relieve tension on associated neurovascular structures; and minimize the risk of converting a closed fracture to an open fracture with a sharp, bony fragment.
- After reduction, the extremity should be immobilized and a radiograph obtained to confirm proper repositioning.
- Open fractures are treated immediately with prophylactic antibiotics to prevent osteomyelitis. A common regimen is a first-generation cephalosporin and an aminoglycoside. The patient's tetanus status should be confirmed. Irrigation and debridement in the operating room is indicated.
- Discharge instructions should emphasize keeping the injured extremity elevated above the level of the heart and seeking immediate reevaluation if increased swelling, cyanosis, pain, or decreased sensation develops.

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167 HAND AND WRIST INJURIES

Michael P. Kefer

ANATOMY AND EXAMINATION

- There are 27 bones in the hand: 14 phalanxes, 5 metacarpals, and 8 carpals.
- The intrinsic muscles of the hand are those that originate and insert within the hand. These are the thenar and hypothenar muscle groups, adductor pollicus, the interossei, and the lumbricals.
- Thenar muscles abduct, oppose, and flex the thumb and are innervated by the median nerve.
- Hypothenar muscles abduct, oppose, and flex the little finger and are innervated by the ulnar nerve.
- Adductor pollicus adducts the thumb and is innervated by the ulnar nerve.
- Interosseous muscles adduct and abduct the fingers from the midline and are innervated by the ulnar nerve.
- Lumbricals provide flexion and extension function to the digits. The two radial lumbricals are innervated by the median nerve. The two ulnar lumbricals are innervated by the ulnar nerve.
- Flexor digitorum superficialis inserts into the middle phalanxes and flexes all the joints it crosses. Function is tested when the patient flexes the prox-

imal interphalangeal (PIP) joint while the other fingers are held in extension.
- Flexor digitorum profundus inserts at the base of the distal phalanxes and flexes the distal interphalangeal (DIP) joint as well as all the other joints flexed by flexor digitorum superficialis. Function is tested when the patient flexes the DIP joint while the PIP and metacarpal-phalangeal (MCP) joints are held in extension.
- Extensor digitorum extends all the digits. Function is tested by having the patient hold the hand in the ''stop traffic'' position. This also tests radial nerve motor function.
- The radial nerve provides sensation to the dorsal radial aspect of the hand. None of the intrinsic muscles of the hand are innervated by the radial nerve.
- The ulnar nerve provides sensation to the fifth and ulnar one-half of the fourth digit, and motor function to the intrinsic hand muscles as discussed earlier.
- The median nerve provides sensation to the first, second, third, and radial half of the fourth digit, and motor function to the intrinsic hand muscles as discussed earlier.

MALLET FINGER

- This results from rupture of the extensor tendon at the DIP joint.
- Treatment, even if associated with a small avulsion fracture (less than 25 percent of the DIP joint surface), consists of splinting the finger in extension for 6 weeks.

BOUTONNIERE DEFORMITY

- This results from injury to the dorsal surface of the PIP that disrupts the extensor hood apparatus.
- Lateral bands of the extensor mechanism become flexors of the PIP joint and hyperextensors of the DIP joint.

GAMEKEEPER'S THUMB

- Gamekeeper's thumb results from forced radial abduction of the first metacarpal with injury to the ulnar collateral ligament. This is the most critical of the collateral ligament injuries since it affects pincer function.
- A partial tear of the ulnar collateral ligament is diagnosed when abduction stress causes the joint to open up to 20° more relative to the uninvolved side. Treatment consists of a thumb spica splint.
- A complete tear is diagnosed when abduction stress causes the joint to open greater than 20° more relative to the uninvolved side. Treatment consists of surgical repair.

DIGIT DISLOCATIONS

- Dorsal IP joint dislocations are reduced by traction, hyperextension, and dorsal pressure at the base of the dislocated phalanx. Splint should be placed in the position of function.
- Dorsal MP joint dislocations are reduced by placing the wrist in flexion and applying distal and dorsal pressure to the proximal phalanx. Splint should be placed in the position of function.
- Irreducible dislocations result from an entrapped avulsion fracture, tendon, or volar plate and require operative reduction.
- Volar IP and MP joint dislocations are rare.

PHALANX FRACTURE

- Distal phalanx fractures most commonly occur at the tuft and may be associated with subungual hematoma and nail bed laceration. Avulsion fractures of the base result in a mallet finger.
- Middle and proximal phalanx fractures are suggested if the fingertips of a closed hand do not point to the same spot on the wrist and the plane of the nail bed of the involved digit is not aligned with the others. Nondisplaced fractures are managed by splinting in the position of function. Displaced fractures are managed surgically.

METACARPAL FRACTURE

- Fourth or fifth metacarpal neck fracture (boxer's fracture) is the most common. Angulation of 30° in the fourth metacarpal or 50° in the fifth metacarpal can be tolerated. Ideally though, angulation $>20°$ should be reduced. An ulnar gutter splint is placed with the wrist extended 20° and the MP joint flexed 90°.
- Second or third metacarpal fractures with any angulation should be reduced.

- First metacarpal fractures are splinted in thumb spica. Bennett's fracture and Rolando's fracture are two fracture types with intraarticular involvement at the base of the metacarpal. Patients should be referred for surgical repair.

WRIST DISLOCATION

- Scapholunate dissociation presents as wrist tenderness at the dorsal radial aspect of the wrist. Radiographs reveal widening of the scapholunate joint >3 mm (the ''Terry Thomas'' sign). Treatment consists of a radial gutter splint and surgical referral.
- Lunate dislocation presents as generalized swelling and tenderness. Radiographs reveal the pathognomonic triangle shape of the lunate (the ''piece of pie'' sign). Lateral radiographs reveal the lunate displaced and tilted palmar (the ''spilled teacup'' sign). Emergent consult is indicated.
- Perilunate dislocation also presents as generalized swelling and tenderness. Lateral radiographs reveal the lunate is still aligned with the radius but the capitate is dislocated, usually dorsal to the radius. Emergent consult is indicated.

WRIST FRACTURES

- Scaphoid fracture is the most common carpal fracture. There is tenderness at the anatomic snuffbox. Radiographs are often negative, but the diagnosis is still made based on physical examination. A thumb spica splint is placed. The scaphoid is at risk for avascular necrosis.
- Triquetrum fracture results in tenderness dorsally, just distal to the ulnar styloid. Radiographs are often negative, but the diagnosis is still made based on a physical examination. A volar splint should be placed.
- Lunate fracture is the most serious of carpal fractures since the lunate occupies two-thirds of the articular surface of the radius. There is tenderness at the lunate fossa just distal to the rim of the radius, in line with the ray of the third metacarpal. Radiographs are often negative, but the diagnosis is still made based on a physical examination. A thumb spica splint should be placed. The lunate is at risk for avascular necrosis.
- Trapezium fracture results in tenderness at the apex of the anatomic snuffbox and base of the thenar eminence. A thumb spica splint should be placed.
- Pisiform fracture results in tenderness at its bony prominence at the base of the hypothenar eminence. The pisiform is a sesamoid bone within the flexor carpi ulnaris tendon. A volar splint with the wrist at 30° flexion and ulnar deviation is placed to relieve tension on the tendon.
- Hamate fracture commonly includes the hook. There is tenderness in the soft tissue of the radial aspect of the hypothenar eminence. A volar splint should be placed.
- Capitate fracture is usually associated with scaphoid fracture. A thumb spica splint should be placed. The capitate is at risk for avascular necrosis.
- Trapezoid fracture is rare. Radiographs are often negative. A thumb spica splint should be placed.
- Colles' fracture results in a dorsally displaced distal radius causing a ''dinner fork'' deformity on examination. Treatment consists of a hematoma block, placement of the hand in a finger trap, and closed reduction.
- Smith's fracture results in a volarly displaced distal radius causing a ''garden spade'' deformity. Treatment is the same as for a Colles' fracture.

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168 FOREARM AND ELBOW INJURIES

Sarah A. Wurster

ELBOW DISLOCATION

- Elbow dislocations are predominantly posterior and result from a fall on an outstretched hand.[1]
- Clinically, the patient presents with the elbow in 45 degrees of flexion. The olecranon is prominent posteriorly; however, this may be obscured by significant swelling.
- While the elbow deformity may resemble a displaced supracondylar fracture, the diagnosis of elbow dislocation is easily confirmed radiographically. On the lateral view, both the ulna and radius are displaced posteriorly. In the anteroposterior (AP) view, the ulna and radius have a normal relationship but may be displaced medially or laterally.
- Neurovascular complications occur in 8 to 21 percent of patients, with injuries to the brachial artery and ulnar nerve being the most common.
- After adequate sedation, reduction is accomplished by gentle traction on the wrist and forearm. An assistant applies countertraction on the arm. Distal traction is applied, while any medial or lateral displacement is corrected with the other hand. Downward pressure on the proximal forearm will help to disengage the coronoid process from the olecranon fossa. Distal traction is continued and the elbow is flexed. A palpable ''clunk'' is noted with successful reduction. Neurovascular status should be assessed before and after reduction.
- The elbow should be splinted in 90 degrees of flexion with a long arm posterior splint.
- Patients with instability in extension require immediate orthopedic referral.

ELBOW FRACTURES

- Fracture of the radial head is the most common fracture of the elbow. It usually results from a fall on an outstretched hand.
- Intercondylar fractures occur in adults, and any distal humeral fracture in an adult should initially be assumed to be intercondylar. Intercondylar fractures occur from a force directed against the elbow. Supracondylar fractures occur most often in children.
- Some 95 percent of these extraarticular fractures are displaced posteriorly.
- Fractures of the radial head result in lateral elbow pain and tenderness and inability to fully extend the elbow. Intercondylar elbow fractures typically present with significant swelling, tenderness, and limited mobility.
- On radiograph, in some undisplaced fractures of the radial head, the fracture line may not be visible and the posterior fat-pad sign may be the only evidence of injury. A careful evaluation for a fracture line separating the condyles from each other and the humerus, which distinguishes intercondylar fractures, should be made.
- Splint immobilization and orthopedic referral is appropriate for nondisplaced fractures. Minimally displaced fractures of the radial head may be treated with a sling and early range of motion.
- Displaced fractures and those with neurovascular compromise require immediate orthopedic consultation.

SUPRACONDYLAR FRACTURES

- Supracondylar fractures typically present with significant swelling, tenderness, and limited mobility. Most supracondylar fractures result from an extension force.
- Supracondylar fractures are often associated with injuries of the median nerve. The anterior interosseus nerve, a motor branch of the median nerve, is particularly prone to injury, resulting in the inability to flex the distal interphalangeal joint of the index finger and thumb interphalangeal joint.[2]
- The brachial artery may be injured, leading to Volkmann's ischemic contracture, which is muscle and nerve necrosis secondary to edema that reduces arterial and venous flow.[3]
- Signs of impending Volkmann's ischemia are forearm tenderness and pain with passive extension of fingers.
- Acute vascular injuries may present with a decreased or absent radial pulse and are most frequently due to transient vasospasm.
- In supracondylar fractures, the AP radiograph usually reveals a transverse fracture line, while the lateral view will show an oblique fracture line and displacement of the distal fragment proximally and posteriorly. The lateral radiograph may also reveal a posterior and anterior fat-pad sign.[4]
- Patients with supracondylar fractures should be admitted for observation of neurovascular status. Supracondylar fractures are often treated with closed reduction followed by pin fixation.

OLECRANON FRACTURES

- Fractures of the olecranon are common and usually result from direct trauma, such as a fall.
- Clinical findings of olecranon fractures include localized swelling and tenderness and limited range of motion. Associated ulnar nerve injuries may occur but are usually transient neuropathies.
- Some 32 percent of olecranon fractures are associated with other fractures, the most common being fractures of the radial head or neck.
- In olecranon fractures, any displacement (>2 mm) should be noted.
- Minimally displaced fractures (<2 mm) and nondisplaced fractures should be treated with splint immobilization and orthopedic referral. Open reduction and internal fixation (ORIF) is required for displaced fractures.

FRACTURES OF THE HUMERAL CONDYLE

- Lateral condylar fractures are more common than medial condylar fractures. The injury may be associated with ulnar nerve damage.
- Treatment for nondisplaced or minimally displaced (<2 mm) fractures is immobilization in a long arm splint. Displaced fractures require orthopedic consultation for ORIF.

LATERAL EPICONDYLITIS

- Patients with lateral epicondylitis, commonly known as ''tennis elbow,'' present with pain at the origin of extensors of the distal arm. The pain is increased with pronation of the forearm and dorsiflexion of the wrist against resistance.
- Treatment includes rest and nonsteroidal anti-inflammatory drugs (NSAIDs).

MEDIAL EPICONDYLITIS

- Medial epicondylitis, commonly known as ''golfer's'' or ''pitcher's elbow,'' is caused by overuse of flexor forearm muscles. This stresses and inflames the tendinous insertion at the medial epicondyle.
- Pronation or wrist flexion against resistance will increase the pain over the medial epicondyle.
- Treatment includes rest and NSAIDs.

BICEPS RUPTURE

- Some 97 percent of biceps ruptures are proximal, occurring in the long head of the biceps.
- Tendon rupture is usually the result of repetitive microtrauma with degenerative changes of the tendon. This injury frequently occurs between the fourth and sixth decades of life.
- Ruptures also are associated with chronic steroid use or steroid injections.

TRICEPS RUPTURE

- Triceps rupture is the least common of all tendon ruptures.
- Triceps rupture occurs most frequently in young males secondary to trauma. It is associated with a high percentage (80 percent) of avulsion fractures of the olecranon.
- Clinically, the patient is unable to extend the elbow.
- Treatment is surgical repair of the tendon.

FRACTURES OF THE RADIUS AND ULNA

- Fractures of both the radius and ulna occur most often from significant trauma, such as a motor vehicle crash or fall from a height.
- Fractures of both bones results in swelling, tenderness, and deformity of the forearm.
- Closed reduction is often adequate for both bone fractures in children. ORIF is usually required for displaced fractures in adults due to displacement and rotational deformity.
- Compartment syndrome is a potential complication.

ULNAR FRACTURES

- Isolated fracture of the ulna (''nightstick fracture'') often results from a direct blow to the forearm. Isolated fractures of the ulna present with swelling and tenderness over the fracture site.
- Fracture of the proximal ulnar shaft with radial head dislocation, a Monteggia fracture-dislocation, causes considerable pain and swelling at the elbow.
- Isolated ulnar fractures are considered displaced if there is more than 10 degrees of angulation or more than 50 percent displacement.
- In a Monteggia fracture, the proximal ulnar frac-

ture is clearly visible but the radial head dislocation may be overlooked. As a rule, the radial head normally aligns with the capitellum in all radiographic views of the elbow. The apex of the ulnar fracture points in the direction of the radial head dislocation.
- Nondisplaced fractures are immobilized in a long-arm cast and closely followed. Displaced fractures and a Monteggia fracture often require ORIF.

RADIAL FRACTURES

- A radial fracture is produced by a fall on the outstretched hand or by a direct blow.
- A fracture of the distal radial shaft with associated distal radioulnar joint dislocation, referred to as a Galeazzi fracture, will present with localized tenderness and swelling over the distal radius and wrist.
- On radiograph, the dislocation of the distal radioulnar joint seen with a Galeazzi fracture may be subtle. On the lateral view, the ulna will be displaced dorsally, while the AP view may show only a slightly increased radioulnar joint space.[5]
- Patients with nondisplaced isolated fractures may have the arm immobilized and be given orthopedic referral. ORIF is usually required for displaced and Galeazzi fractures.[6]

SUBLUXATION OF THE RADIAL HEAD ("NURSEMAID'S ELBOW")

- The peak age for subluxation of the radial head is between 1 and 4 years.
- The patient holds the injured arm in slight flexion and pronation. The neurovascular exam is normal.
- Reduction is accomplished by placing the physician's thumb on the radial head and the other hand on the patient's wrist. The physician fully supinates the patient's forearm and then fully flexes the elbow.
- Recurrent subluxations require orthopedic referral.

NEUROANATOMY OF THE FOREARM AND HAND

- The radial nerve travels over the lateral epicondyle and supplies muscles involved in wrist extension.
- The posterior interosseous nerve, a branch of the radial nerve, controls the muscles that extend the fingers and thumb.
- The remainder of the radial nerve is sensory and innervates the posterior aspect of the hand from the thumb to the radial half of the ring finger.
- The median nerve controls basic movements of the wrist and fingers and flexion and sensation on the volar surface of the hand from the thumb to the radial half of the ring finger.
- The recurrent branch of the median nerve supplies motor function to the thenar muscles of the thumb.
- A test of median nerve function is the ability to make the "OK" sign (anterior interosseus); adduction of the thumb (recurrent branch of the median nerve) and intact sensation on the radial side of the palm.
- The ulnar nerve innervates a few forearm muscles and controls the intrinsic muscles of the hand and provides sensation to the little finger.
- A test of ulnar nerve function is the ability to abduct the index finger against resistance and the presence of normal sensation on the ulnar side of the hand.

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169 SHOULDER AND HUMERUS INJURIES

Sarah A. Wurster

CLAVICLE FRACTURES

- Clavicle fractures account for 50 percent of significant shoulder girdle injuries and 5 percent of all fractures seen and treated in the emergency department.
- Clavicle fractures are the most common fracture in children.
- Eighty percent of clavicle fractures involve the middle one-third, 15 percent the distal one-third, and 5 percent the medial one-third.
- Simple immobilization with a sling is acceptable for most clavicle fractures.

STERNOCLAVICULAR DISLOCATION

- Anterior sternoclavicular dislocations are more common than posterior dislocations and result in a prominent medial clavicle that appears anterior to the sternum.
- Posterior dislocations of the sternoclavicular joint may result in impingement of superior mediastinal structures and are more difficult to diagnose.
- The differential diagnosis of sternoclavicular joint sprains should include septic arthritis, especially in intravenous IV drug users.

ACROMIOCLAVICULAR INJURIES

- A type I acromioclavicular injury is associated with a normal radiograph.
- A type II acromioclavicular injury reveals 25 to 50 percent elevation of the distal clavicle above the acromion on the radiograph.
- A type III acromioclavicular injury reveals 100 percent dislocation of the acromioclavicular joint and coracoclavicular space widening on the radiograph.
- Treatment of type I and II injuries includes rest, ice, analgesics, and immobilization with a sling, followed by early range-of-motion.
- Treatment of type III injuries is controversial, with some orthopedic surgeons opting for conservative treatment and others for surgical repair.

GLENOHUMERAL JOINT DISLOCATION

- The shoulder joint is the most common major joint that is dislocated. The most predominant type is the anterior dislocation (>98 percent). Posterior dislocations occur in <2 percent of cases.
- Rotator cuff injuries may accompany shoulder dislocations.
- The axillary nerve is the nerve most commonly injured with glenohumeral joint dislocations. Axillary nerve function is tested by pinprick sensation over the skin of the deltoid muscle.
- Associated bony injuries include fractures of anterior glenoid lip, greater tuberosity, coracoid, and acromion, and compression fractures of the humeral head (Hill-Sachs lesion).
- The most common complication of this injury is recurrent dislocation.
- The rare inferior dislocation (luxatio erecta) will present with the affected arm fully abducted, elbow flexed, and hand held above the patient's head. Complications of luxatio erecta include severe soft tissue injuries, fractures of the proximal humerus, and rotator cuff tears.
- Anteroposterior and lateral scapular (Y view) or axillary radiographs should be obtained before reduction.
- There are numerous methods for reducing anterior shoulder dislocations. Modified Hippocratic technique: This method uses traction–countertraction. The patient is supine with the arm abducted. A sheet is placed across the thorax of the patient and tied around the waist of the assistant. The physician gradually applies traction while the assistant provides countertraction.
- Milch technique: With the patient supine, the physician slowly abducts and externally rotates the arm to the overhead position. With the elbow fully extended traction is applied.
- External rotation technique: With the patient supine and the elbow at 90° flexion, the arm is slowly externally rotated. No traction is applied. This technique must be performed slowly and gently.

HUMERUS FRACTURES

- Proximal humerus fractures are typically seen in elderly patients with osteoporosis after a fall.

- Humeral shaft fractures usually occur in younger patients after direct or indirect trauma. Humeral shaft fractures most commonly occur in the middle third of the bone.
- The humerus is a common site of pathologic fractures, especially metastatic breast cancer.
- The axillary nerve is the most commonly injured nerve in proximal humerus fractures.
- The radial nerve is the most commonly injured nerve in humeral shaft fractures. This injury may result in wrist drop and altered sensation at the dorsal first web space. The incidence of associated radial nerve palsy ranges from 10 to 20 percent.
- An axillary artery injury is the most commonly associated vascular injury.
- The majority of uncomplicated humeral shaft fractures can be managed nonoperatively, usually by immobilization with an arm sling and close follow-up.

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170 INJURIES OF THE PELVIS, HIP, AND FEMUR

Craig E. Krausz

PELVIC FRACTURES

EPIDEMIOLOGY

- Pelvic fractures account for 3 percent of all skeletal fractures.[1]
- One-third of pelvic fractures are the result of industrial accidents or falls in the elderly.[2]

CLINICAL FEATURES

- Pelvic fractures should be suspected whenever there is trauma to the torso or a fall from a height.
- Pain, crepitus, or instability on palpation of the pelvis suggests a fracture. Perianal edema, pelvic edema, ecchymoses, lacerations, deformities, and hematomas over the inguinal ligament or the scrotum (Destot's sign) suggest pelvic fracture. The fracture line may be palpated on rectal examination (Earle's sign).
- Hypotension may be secondary to abdominal or thoracic injuries or to blood loss from disrupted pelvic bones or vessels.

DIAGNOSIS AND DIFFERENTIAL

- On radiograph, the anteroposterior (AP) pelvic radiograph is the most useful view; additional views include oblique hemipelvis, inlet (to evaluate AP displacement), and outlet views (to evaluate superoinferior displacement).
- Computed tomography (CT) is superior to plain radiography in assessing the posterior arch and the acetabulum and checking for associated hemorrhage.[3]
- Many classifications of pelvic fractures exist; the Young system is helpful because fractures are classified based on mechanism and directional forces (see Table 170-1). Four main patterns (suggested by the alignment of pubic rami fractures, pubic symphysis diastasis, and sacroiliac joint displacement) have been identified: (1) lateral compression (LC), which usually results from motor vehicle crashes—the mortality rate approaches 13 percent; (2) anteroposterior compression (APC), which usually results from a head-on motor vehi-

TABLE 170-1 Injury Classification Keys According to the Young System

CATEGORY	DISTINGUISHING CHARACTERISTICS
LC	Transverse fracture of pubic rami, ipsilateral or contralateral to posterior injury I—Sacral compression on side of impact II—Crescent (iliac wing) fracture on side of impact III—LC-I or LC-II injury on side of impact; contralateral open-book (APC) injury
APC	Symphyseal diastasis and/or longitudinal rami fractures I—Slight widening of pubic symphysis and/or anterior SI joint; stretched but intact anterior SI, sacrotuberous, and sacrospinous ligaments; intact posterior SI ligaments II—Widened anterior SI joint; disrupted anterior SI, sacrotuberous, and sacrospinous ligaments; intact posterior SI ligaments III—Complete SI joint disruption with lateral displacement; disrupted anterior SI, sacrotuberous, and sacrospinous ligaments; disrupted posterior SI ligaments
VS	Symphyseal diastasis or vertical displacement anteriorly and posteriorly, usually through the SI joint, occasionally through the iliac wing and/or sacrum
CM	Combination of other injury patterns. LC/VS being the most common

ABBREVIATIONS: APC = anteroposterior compression; CM = combination; LC = lateral compression; VS = vertical shear.

cle crash—the mortality rate approaches 25 percent; (3) vertical shear (VS), which usually results from a fall or jump from a height—the mortality rate approaches 25 percent; and (4) a combination (CM) of the preceding, with LC/VS being the most common.[4]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Standard protocols for the evaluation and stabilization of trauma patients should be initiated. Patients should receive supplemental oxygen, be placed on a cardiac monitor, and have two IV lines established.
- Blood should be sent for type and crossmatching. Since hemorrhage is the cause of death in 50 percent of patients with pelvic fractures, early use of blood products is indicated.[5]
- Early external fixation decreases complications, such as adult respiratory distress syndrome, and should be considered if there is evidence of continued blood loss with disruption of the posterior elements.[6]
- Angiography for embolization of pelvic vessels is indicated in 2 percent of pelvic fractures and approaches 100 percent efficacy.[7]
- Injuries of intraabdominal solid organs and other sources of blood loss should be considered. If diagnostic peritoneal lavage is performed, a supraumbilical approach should be taken to avoid disruption of a pelvic hematoma.
- Rectal exam (and bimanual pelvic exam for women) should be performed for rectal and gynecologic injuries. Rectal injuries are treated with irrigation, diverting colostomy, and antibiotics.
- If blood is found on pelvic exam, a speculum exam should be performed to evaluate for vaginal lacerations (which may occur with anterior pelvic fractures). Vaginal lacerations mandate operative debridement, irrigation, and IV antibiotics.
- Mortality has been shown to be reduced with early fixation and patient mobilization.[8–11]

STABLE PELVIC AVULSION FRACTURES

- Avulsion fracture of the anterior superior iliac spine (ASIS) occurs when a forceful contraction of the sartorius muscle causes separation of the ASIS. Symptoms include localized swelling and pain with thigh flexion and abduction.
- Avulsion fractures of the anterior inferior iliac spine (AIIS) occurs after forceful contraction of the rectus femoris muscle. Symptoms include groin pain and inability to flex at the hip. Radiography reveals downward displacement of the inferior iliac spine.
- Avulsion of the ischial tuberosity occurs in patients below age 20 to 25 when the hamstring forcefully contracts (jumping). Pain is present on sitting and thigh flexion. On rectal examination, there is tenderness on palpation of the tuberosity.
- Treatment of avulsion fractures is conservative, with rest in a position of comfort and use of crutches, with partial weight bearing followed by full weight bearing.[10]

STABLE FRACTURES INVOLVING A SINGLE PELVIC BONE

- The ischial bodies can be injured by a direct fall on the buttocks. Iliac wing (Duverney) fractures present with pain and swelling over the iliac wing. Intraabdominal injuries may coexist.[12]
- Sacral fractures may occur when large anteroposterior forces are applied to the pelvis. They may be difficult to diagnose on radiography; subtle ir-

regularity, buckling, or malalignment of the sacral foramina are suggestive; a lateral view may show displacement. A transverse fracture line at the level of the lower sacroiliac (SI) joint may be seen, along with irregularity, buckling, or sharp angulation of the foramina. Neurologic injury does not occur with fractures below S4. Sacral root injuries may be present in up to one-third of sacral fractures.[13,14] A bimanual rectal examination (one finger in the rectum, and a hand on the sacrum) may reveal crepitus.

- Coccygeal fractures result from a fall in a sitting position. The diagnosis is made clinically by rectal examination, which may reveal tenderness or crepitus. Treatment is symptomatic, with a soft doughnut cushion for sitting, ice, and analgesics.
- Treatment of simple fractures without neurologic injury is bed rest, stool softeners, and orthopedic follow-up. Complex fractures require orthopedic consult.

ACETABULAR FRACTURES

- Acetabular fractures account for 20 percent of pelvic injuries and usually occur secondary to motor vehicle crashes.
- The four anatomic sites of fracture—posterior, ilioischial column, transverse, and iliopubic column—are all associated with hip dislocations.
- The most common complication is a sciatic nerve injury.
- Early orthopedic consultation and hospital admission are indicated for patients with acetabular fractures.

Hip and Femur Injuries

HIP FRACTURES

- The incidence of hip fractures in the United States is 80 per 100,000 population.[15] The annual incidence increases with age and doubles for each decade after age 50. It is three to four times higher in women than in men.
- The affected leg is classically foreshortened and externally rotated. The position of the extremity, ecchymoses, deformity, and range of motion should be evaluated. Complications include infection, venous thromboembolism, avascular necrosis, and nonunion.
- On radiography, AP, lateral, and frog-leg views will evaluate the femur and acetabulum. Hip fractures are classified as intracapsular (femoral head and neck) or extracapsular (intertrochanteric and subtrochanteric). Intracapsular fractures may compromise blood supply to the femoral head and lead to avascular necrosis.
- Isolated fractures of the femoral head are most commonly associated with hip dislocations.[16] Femoral neck fractures are common in elderly patients with osteoporosis. The leg is shortened, abducted, and held in external rotation. There is a 90 percent incidence of avascular necrosis if the injury is left untreated and a 20 to 30 percent incidence of nonunion with displaced fractures.[7,16] Nondisplaced neck fractures are treated with pin fixation; displaced fractures are treated with open reduction or prosthesis placement.
- Stress fracture of the femur should be suspected if there is significant pain without radiographic abnormality. Radiographs should reveal a fracture, but a bone scan is more sensitive for subtle fractures. Stress fractures are treated conservatively, with a bone scan in 1 to 2 days or a follow-up radiograph in 10 to 14 days.
- Intertrochanteric fractures generally occur in the elderly after a fall or a motor vehicle crash. The extremity is markedly rotated externally and shortened. These fractures are classified as stable or unstable. Stable fractures are those in which the medial cortices of the femoral neck and the femoral fragments abut. Buck's traction may be applied until surgical fixation is performed. Overall mortality is 10 to 30 percent.[17]
- Subtrochanteric fractures may be seen in elderly osteoporotic patients and young patients after major trauma. Symptoms include pain, deformity, and swelling. Patients with this injury may present with hypotension secondary to blood loss into the soft tissue of the thigh. Immobilization with a traction apparatus is recommended, with eventual open reduction and internal fixation.[17]
- Fractures of the greater trochanter may occur in adults (true fracture) or children (avulsion of the apophysis). Pain is present on abduction and extension of the leg. Treatment is controversial and may include conservative treatment or operative fixation (based on the patient's age and displacement of the fragment).[7]
- Lesser trochanteric avulsions are most common in young athletes after avulsion secondary to a forceful contraction of the iliopsoas muscle. There is pain during flexion and internal rotation. If there is more than 2 cm of displacement, operative fixation with screws is recommended.

- Following trauma, significant hip pain with weight bearing, even in the presence of normal plain radiographs, suggests the possibility of an occult fracture, especially of the femoral neck or the acetabulum. If an occult fracture is suspected, close follow-up is needed for CT or magnetic resonance imaging (MRI). MRI is reliable in detecting occult fractures within 24 h of injury.[18–21]

HIP DISLOCATIONS

- Hip dislocations are most often the result of massive forces during trauma. Ninety percent are posterior and 10 percent are anterior.
- Both types are treated with early closed reduction (<6 h) in order to decrease the incidence of avascular necrosis.
- Posterior dislocations, which occur when a posterior force is applied to the flexed knee, may coexist with acetabular fractures. The leg is foreshortened and internally rotated and adducted. AP, lateral, and oblique views will evaluate the status of the acetabulum and the femoral head.
- Treatment of posterior dislocations includes early closed reduction using the Allis maneuver (hip flexion to 90 degrees, then internal and external rotation) or the Stimson maneuver (patient prone with the leg hanging over the edge of stretcher and application of gentle traction).
- Anterior dislocations occur during forced abduction. The leg is held in abduction and external rotation.
- Treatment of anterior dislocations includes early closed reduction with strong, in-line traction and flexing and externally rotating the leg, with abduction once the femoral head clears the acetabulum.

FRACTURES OF THE FEMORAL SHAFT

- Femoral shaft fractures typically occur when patients are involved in a motor vehicle crash.[22]
- Spiral midshaft femoral fractures can occur in toddlers who are running and trip in a twisting fashion. Midshaft femoral fractures in children are often a result of neglect or abuse.[23,24]
- Since the femur has a rich vascular supply and is surrounded by soft tissue, it can accommodate 1 L or more of blood, potentially contributing to hypotension and shock after a fracture.
- Distal neurovascular function should be thoroughly evaluated. Diagnosis is confirmed radiographically.
- Treatment involves immediate immobilization with Hare or Sager traction or a Thomas splint. Definitive repair is by operative fixation or, in children, traction. Open femoral fractures require early orthopedic consultation for copious irrigation and debridement in the operating room.[25]

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171 KNEE AND LEG INJURIES

Sarah A. Wurster

KNEE INJURIES

RADIOGRAPHIC EVALUATION

- Anteroposterior (AP), lateral, and oblique views are typically obtained for radiographic assessment of the knee.
- Fat-fluid levels may be identified on a lateral view of the knee, which is suggestive of intraarticular fracture.
- The sunrise view is the most useful view in evaluating for nondisplaced vertical or marginal fractures of the patella.[1,2]

OTTAWA KNEE RULES

- The Ottawa Knee Rules offer guidelines for obtaining radiographs of the knee. Radiographic evaluation is recommended if any of the following criteria apply: (1) patient >55 years old, (2) tenderness at the head of the fibula, (3) isolated patellar tenderness, (4) inability to flex the knee to 90 degrees, or (5) inability to transfer weight for four steps both immediately after the injury and in the emergency department (ED).

PATELLAR FRACTURES

- Fractures of the patella occur most often from a direct blow or from a fall on a flexed knee but may be caused by forceful contracture of the quadriceps muscle. Transverse patellar fractures are the most common, followed by stellate and comminuted fractures.
- Symptoms include pain and swelling over the patella. A palpable defect and tenderness are usually found on the patella. Patients with nondisplaced fractures may be ambulatory.
- Plain radiographs, including the sunrise view, confirm the diagnosis.
- Treatment depends on the type of fracture: minimally or nondisplaced fractures without disruption of the extensor mechanism of the knee are treated with a knee immobilizer and crutches, followed by 6 weeks in a long leg cast. Fractures that are displaced >3 mm or comminuted or that disrupt the extensor mechanism mandate early orthopedic referral for operative repair.

FEMORAL CONDYLE FRACTURES

- This injury often results from a fall from a height or a direct blow and accounts for 4 percent of femoral fractures.
- Signs and symptoms include pain, swelling, and deformity, occasionally with shortening and rotation. Associated neurovascular injuries may include the popliteal artery and the deep peroneal nerve (sensation in the web space between the first and second toes). The ipsilateral hip and quadriceps apparatus should also be fully evaluated for concomitant injuries.
- Orthopedic consultation is indicated. Nondisplaced fractures are treated with immobilization; displaced fractures are treated with open reduction and internal fixation (ORIF).

TIBIAL SPINE AND TUBEROSITY FRACTURES

- This injury is caused by anterior or posterior forces applied against a flexed knee and are often associated with avulsions of the cruciate ligament. Fracture of the anterior tibial spine is tenfold more common than fracture of the posterior tibial spine.
- On physical examination, the knee is swollen and tender and cannot be fully extended due to hemorrhagic joint effusion. There is a positive Lachman's test.
- Nondisplaced fractures are treated with knee immobilization in full extension. Displaced fractures often require ORIF.

TIBIAL PLATEAU FRACTURES

- This injury results from a direct blow or axial loading, which forces the femoral condyles onto the tibia. The lateral plateau is most commonly injured. The fracture occurs more frequently in the elderly and can be difficult to detect.
- Symptoms include pain and swelling of the knee and decreased range of motion. Injuries of the anterior cruciate and medial collateral ligaments are associated with fractures of the lateral plateau, whereas injuries of the posterior cruciate and lateral collateral ligaments are associated with fractures of the medial plateau.
- Radiographs may demonstrate a fracture or joint effusion with a fat-fluid level (lipohemarthrosis) on lateral view. Computed tomography (CT) is helpful in evaluating the fracture.[3]
- Treatment for nondepressed fractures is a long leg cast and non-weight-bearing activity. Depressed fractures are treated operatively with elevation of fragments.[3]

LIGAMENTOUS INJURIES

- Patients with these injuries present with pain and swelling at the knee. A hemarthrosis is common but may be absent if there is complete disruption of the joint capsule.[4]
- Injury to the anterior cruciate ligament (ACL) is the most common. ACL sprains may be associated with medial meniscal tears. Patients often describe hearing an audible ''pop'' associated with pain and swelling at the knee. The Lachman test (most sensitive) and pivot-shift test are sensitive for diagnosing ACL tears.
- If there is a demonstrated laxity of more than 1 cm without a firm end point as compared to the other knee, then there is a complete rupture of the medial or lateral collateral ligaments.
- Tears of the posterior cruciate ligament are rarer and often involve large posterior forces applied to the lower leg. A posterior drawer sign may be elicited but is not sensitive.
- Radiographs frequently reveal only a joint effusion.
- Treatment includes knee immobilization, weight bearing as tolerated, analgesics, and orthopedic follow-up. Arthrocentesis is beneficial only for symptomatic relief from tense hemarthroses that cause severe pain.

MENISCAL INJURIES

- Symptoms of meniscal injuries include painful locking of the knee, a popping or clicking sensation, or a sensation of the knee giving out. The medial meniscus is approximately twice as likely as the lateral meniscus to be injured.[5]
- Physical examination may reveal atrophy of the ipsilateral quadriceps muscle. The McMurray test or the grind test may be useful in making the diagnosis.
- Treatment includes knee immobilization, weight bearing as tolerated, analgesics, and orthopedic follow-up.

KNEE DISLOCATION

- Posterior knee dislocations are the most common form of knee dislocation.
- On physical examination, the knee is unstable; occasionally, the dislocation may have reduced spontaneously. A thorough neurovascular exam must be performed, since there is a high incidence of associated popliteal artery (50 percent incidence) and peroneal nerve (more common with posterior dislocations) injury with this dislocation.
- With the patient under conscious sedation, the dislocation should be reduced by applying longitudinal traction. The vascular status should be evaluated before and after reduction with an ankle-brachial index (ABI). If there is evidence of vascular insufficiency, an arteriogram should be performed. Some orthopedic surgeons advocate arteriography even if the ABI is normal.
- Emergent orthopedic consultation and admission are mandated for this injury.

PATELLAR DISLOCATION

- This injury most commonly presents with lateral patellar displacement after a twisting injury. A torn medial knee-joint capsule may be associated with the dislocation.
- Under conscious sedation, reduction is achieved by hyperextending the knee and flexing at the hip while sliding the patella medially back into place. After reduction, the knee should be immobilized in extension.
- Orthopedic follow-up should be arranged in 1 to 2 weeks. Recurrent dislocations of the patella occur in about 15 percent of patients.

PATELLAR TENDON RUPTURE

- This injury is more common in patients <40 years old who have a history of patellar tendonitis or steroid injections. It occurs after forceful contraction of the quadriceps muscle. The main symptom is pain inferior to the patella.
- On physical examination, there is a defect inferior to the patella, with the inability to extend the knee. The patella may be high-riding (patella alta) or low-riding (patella baja).
- Treatment requires knee immobilization and orthopedic consultation for operative repair in 7 to 10 days.

QUADRICEPS TENDON RUPTURE

- Quadriceps tendon rupture is more common in older individuals after sudden contraction of the quadriceps muscle (landing after a jump). Symptoms include sharp pain at the proximal knee on ambulating. If the tear is complete, the patient will be unable to extend the leg from knee flexion, although the ability to do a straight-leg raise may be maintained.
- There may be a palpable defect, with tenderness and swelling at the suprapatellar region, and the patella may migrate distally (patella baja).
- Partial tears are treated similarly to quadriceps muscle tears. A complete tear requires orthopedic consultation for operative repair.

PATELLAR TENDONITIS

- This condition, or ''jumper's knee,'' presents with pain over the patellar tendon when running up hills or standing from a sitting position.
- Treatment includes ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and quadriceps strengthening exercises.

CHONDROMALACIA PATELLAE

- This condition is caused by patellofemoral malalignment, which places lateral stress on the articular cartilage. It is most common in young, active women and presents with anterior knee pain that worsens with climbing stairs or rising from a sitting position.
- Diagnosis is assisted using the patellar compression test and the apprehension test.
- Treatment includes NSAIDs, rest, and quadriceps-strengthening exercises.

LEG INJURIES

FIBULAR FRACTURES

- This injury most commonly involves the distal fibula at the ankle. Isolated shaft fractures usually occur from direct trauma.
- Since the fibula is not a large, weight-bearing bone, the majority of fractures are treated with immobilization and orthopedic referral for casting.

TIBIAL SHAFT FRACTURES

- This injury presents with pain, swelling, and crepitus. A complete neurovascular evaluation is essential. Signs and symptoms of compartment syndrome should be evaluated.
- Plain radiographs confirm the diagnosis; radiographs of the knee and ankle should be included.
- Treatment depends on the location and the amount and displacement of bony fragments. Most fractures of the tibial shaft require urgent orthopedic evaluation. Indications for emergent operative repair include open fractures, the presence of vascular compromise, or compartment syndrome.[6]

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172 ANKLE AND FOOT INJURIES

Sarah A. Wurster

ANKLE INJURIES

OTTAWA ANKLE RULES FOR ANKLE AND MIDFOOT INJURIES

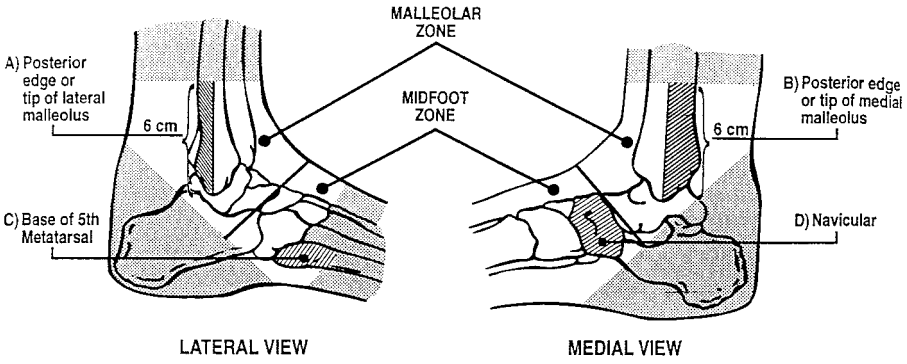
- The Ottawa Ankle Rules for ankle and midfoot injuries (Fig. 172-1) are simple guidelines that have been extensively validated in numerous clinical trials. When applied properly, they can help the emergency physician identify a subset of patients who can safely be treated without undergoing radiographic studies.[1–3]

ANKLE SPRAINS

- Most ankle sprains result from a twisting mechanism, with the vast majority due to an inversion mechanism. The ability to bear weight immediately after an injury with subsequent increase in pain and swelling as the patient continues to ambulate suggests a sprain rather than a fracture.
- On physical examination, significant findings include the absence of bony tenderness in the ankle, a normal neurovascular exam, and tenderness and soft tissue swelling over the involved ankle ligament. The most common sprain involves the anterior talofibular ligament.
- A positive anterior drawer test of the ankle is movement greater than 5 mm in comparison to the normal ankle. More than 10 degrees of movement with inversion or eversion in comparison to the other foot is a positive talar tilt test.
- As with all extremity injuries, the joints above and below the injury should be examined. Tenderness of the knee, the fibular head, or the proximal fibular shaft suggests a fibulotibial ligament tear or a Maissoneuve fracture.
- The Achilles tendon should also be examined. Rupture of the Achilles tendon occurs with forceful plantar flexion. Clinically, an Achilles tendon rupture is diagnosed when there is tenderness or a defect over the Achilles tendon and a positive Thompson's test (absence of plantar flexion of the foot when the calf is squeezed). Patients should be splinted in a neutral position and referred for prompt orthopedic follow-up.
- Most studies indicate that patients with ankle sprains who can bear weight easily, whether stable or unstable, should be treated with rest, ice, compression, and elevation (RICE) for 24 to 72 h. Patients who clearly have an unstable joint should be referred to an orthopedic surgeon and may benefit from complete immobilization with a posterior short-leg splint until follow-up.
- Isolated sprains of the deltoid ligament are rare. There is usually an associated fibular fracture or significant tear of the tibiofibular syndesmosis resulting from an eversion stress. The proximal fibula and fibular shaft should be carefully examined for evidence of a Maissoneuve fracture. If radiographs are negative, a significant tear of the syndesmosis should be suspected. Treatment is with RICE and early orthopedic referral.[4]

ANKLE FRACTURES

- Henderson's scheme for classifying ankle fractures is based on their radiographic appearance (unimalleolar, bimalleolar, or trimalleolar) and is adequate for emergency department (ED) treatment purposes. The lateral malleolus is the most commonly fractured site.
- All fractures of the ankle with the exception of fibular avulsion fractures require immobilization, either with casting alone or with surgical reduction and then casting. Isolated avulsion fractures of the distal tip of the fibula may be treated as stable sprains if minimally displaced, small (less than 3 mm), and without evidence of ligamentous instability.[5]
- Unimalleolar injuries may be treated with a posterior short leg splint. The patient should avoid



a) An ankle x-ray series is only required if:

There is any pain in malleolar zone and any of these findings:

i) bone tenderness at A
OR
ii) bone tenderness at B
OR
iii) inability to bear weight both immediately and in ED

b) A foot x-ray series is only required if:

There is any pain in midfoot zone and any of these findings:

i) bone tenderness at C
OR
ii) bone tenderness at D
OR
iii) inability to bear weight both immediately and in ED

FIG. 172-1 The Ottawa Ankle Rules for ankle and midfoot injuries.

weight bearing until orthopedic follow-up in 3 to 5 days.
- Bimalleolar and trimalleolar fractures usually require open reduction and internal fixation (ORIF).
- For open fractures, the initial antibiotic of choice is a first-generation cephalosporin (usually cefazolin, 1 g IV), or clindamycin (if the patient is allergic to cephalosporins). An aminoglycoside (gentamicin) also may be added if the wound is grossly contaminated.

ANKLE DISLOCATIONS

- Dislocations of the ankle joint usually occur with an associated fracture. Patients with fracture-dislocations of the ankle are at significant risk of neurovascular compromise.
- In cases where there is evidence of neurovascular compromise, the emergency physician should proceed with reduction of the injury as expeditiously as possible, without waiting for radiographs, in order to restore vascular integrity. This procedure is best accomplished with the emergency physician grasping the heel and foot with two hands and applying gentle but steady longitudinal traction while an assistant stabilizes the proximal leg. Radiographs may be completed once the reduction has been done and distal perfusion restored.
- The patient should be admitted to the hospital after orthopedic consultation.[6]

FOOT INJURIES

HINDFOOT INJURIES

- Fractures of the calcaneus can be caused by any axial load to the heel, such as a fall from a height. Calcaneal injuries are frequently associated with

spinal injuries and other lower extremity fractures, so a thorough physical examination is mandatory.
- Although some fractures of the calcaneus are clearly apparent on radiographs, others can be quite subtle. When a radiograph is unremarkable and a calcaneal fracture is still suspected, Boehler's angle—formed by the intersection of a straight line extending along the superior cortex of the body of the os calcis with a line extending from the dome to the anterior tubercle—should be measured on the lateral view of the foot. If the angle is less than 20 degrees, a fracture is likely.
- Comminuted calcaneal fractures are associated with a high incidence of compartment syndrome.
- Orthopedic consultation should be obtained for all calcaneal fractures.
- Talar fractures are uncommon because of the excessive forces required to fracture the bone. Talar fractures usually require ORIF and are frequently complicated by avascular necrosis.
- Peritalar or subtalar dislocations require immediate orthopedic consultation and urgent reduction.

MIDFOOT INJURIES

- Isolated fractures of the tarsal bones are uncommon and are usually treated conservatively.
- Fracture of the navicular bone is the most common fracture of the midfoot.
- When a fracture of the cuboid or cuneiforms is identified, an injury to the Lisfranc joint (tarsometatarsal complex) should be suspected. Injuries to this joint are rare and frequently missed in the ED.[7]
- A fracture of the base of the second metatarsal may be viewed as pathognomonic of a disruption of the ligamentous complex. This injury should be suspected when there is point tenderness over the midfoot or when there is laxity between the first and second metatarsals in a dorsoplanar direction.
- Diagnosis is made radiographically on the anteroposterior view when there is more than a 1-mm gap between the bases of the first and second metatarsals.
- Injuries to the Lisfranc joint may require open reduction or percutaneous pinning, and long-term morbidity may be significant.

FOREFOOT INJURIES

- Metatarsal fractures commonly are caused by a crush mechanism. Most nondisplaced fractures of the metatarsal shaft can be treated conservatively. Any fracture of the first metatarsal shaft must be treated with a period of no weight bearing. Displaced fractures of any of the metatarsal shafts are problematic, requiring avoidance of weight bearing and possibly surgical fixation.
- Fractures of the fifth metatarsal are the most common of the metatarsal fractures. Shaft fractures can usually be treated conservatively, as can the ''pseudo-Jones'' fracture (an avulsion from the proximal pole).
- The Jones fracture is a transverse fracture through the base of the fifth metatarsal 15 to 31 mm distal to the proximal part of the metatarsal. It is subject to complications much more frequently, including malunion or nonunion. The Jones fracture must be treated with a non-weight-bearing cast and close orthopedic follow-up.[8]

PHALANGEAL INJURIES

- Most nondisplaced phalangeal fractures can be treated conservatively, with ''buddy taping'' or a cast shoe.
- Dislocations and displaced fractures can be reduced by providing a digital block and applying manual traction, followed by buddy taping.

PUNCTURE WOUNDS

- These wounds carry the risk of retained foreign body, deep soft tissue infection, or osteomyelitis. Deep penetration increases the risk of damage to bone and tendons, and penetration through a rubber sole may increase the chance of infection with *Pseudomonas.*
- Radiographs may be useful in some cases; when normal, however, they do not exclude the possibility of bony injury or retained foreign body.
- The use of prophylactic antibiotics after a puncture wound is controversial and may be best reserved for patients who are immunocompromised, have peripheral vascular disease or diabetes, or have bone or tendon involvement.
- Patients who present with delayed puncture wounds complicated by infection should have the wound opened and irrigated, any foreign material removed and any devitalized tissue excised, and be started on antibiotic therapy.
- The antibiotic of choice is usually a first-generation cephalosporin (such as cephalexin), although a flouroquinolone may be added if *Pseudomonas* is suspected. Complicated foot infections, gunshot

wounds to the foot, and many lawn mower injuries require consultation for operative debridement.

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173 COMPARTMENT SYNDROMES

Stefanie R. Seaman

PATHOPHYSIOLOGY

- Compartment syndromes are caused by increased pressure within a closed tissue space that compromises blood flow to muscles and nerves.[1]
- Normal tissue pressure measures 0 to 10 mmHg. Capillary blood flow is compromised at pressures greater than 20 mmHg; muscle and nerves become at risk for necrosis at pressures of 30 to 40 mmHg or greater. Nerves are more sensitive than muscle because they are reliant on nutrient capillaries.
- Primarily, there are two causes that lead to compartment syndromes: (1) compression of a compartment or decreased compartment size and (2) volume increase within a closed compartment (secondary to hematoma and edema).

CLINICAL FEATURES

- Severe and constant pain of the involved muscle compartment is the hallmark clinical feature. Palpation of the affected compartment, active contraction, or passive stretching of muscles in the affected compartment(s) will exacerbate a conscious patient's pain.
- Muscle weakness (paralysis) and paresthesia occur when pressures affect neurologic function. This occurs at about the same time as pain. Of these, a sensory deficit is the most reliable finding.
- Pallor, coolness, and absent pulses appear late, usually after muscle necrosis has occurred.
- A thorough history is necessary, since this syndrome may occur in critically ill patients who may not be able to complain of pain.
- Any muscle mass enclosed by fascia is at risk for compartment syndrome. Table 173-1 demonstrates the symptomatology of acute compartment syndromes.[2]

DIAGNOSIS AND DIFFERENTIAL

- Patients who present with compression injuries, fractures, penetrating wounds, or hemorrhage should prompt a high index of suspicion.
- The mainstay of diagnosis is measuring compartment pressures. Compartment pressures can easily be measured in the emergency department with a Stryker STIC Monitor or ACE Intracompartmental Pressure Monitor. Using aseptic technique, an 18-gauge needle is inserted into the compartment and a small volume of sterile saline is injected. After 1-s, as resistance to flow is overcome, the self-contained pressure transducer will give a measurment in mmHg.
- Most muscle compartments normally have pressures of less than 10 mmHg, and such pressures are often normally near zero. The presence of an abnormally elevated pressure confirms the diagnosis.

TABLE 173-1 Symptomatology of Acute Compartment Syndromes[3]

Upper extremity	
Upper arm	
Anterior compartment	Pain on active and passive flexion and extension of the elbow
	Hypoesthesia in the distribution of the median, ulnar, and radial nerves
Posterior compartment	Pain on active and passive flexion and extension of the elbow
	Hypoesthesia over the dorsum of the hand
Forearm	
Volar compartment	Pain on active and passive flexion and extension of the fingers
	Hypoesthesia over the palm of the hand
Dorsal compartment	Pain on active and passive flexion and extension of the fingers
Hand	
Thenar and hypothenar compartments	Pain on thumb and little finger opposition
Interosseous compartments	Pain on abduction and adduction of the fingers
Lower extremity	
Gluteal compartments	Pain on active and passive flexion and extension of the hip
	Sciatic nerve paresthesias
Thigh compartments	Pain on active and passive flexion and extension of the knee
	Sciatic nerve paresthesias with posterior compartment involvement
Leg	
Anterior compartment	Pain on active and passive dorsiflexion and plantar flexion of the foot
	Hypoesthesia of the first web space
Lateral compartment	Pain on active and passive eversion and inversion of the foot
	Hypoesthesia of the first web space
Superficial posterior compartment	Pain on active and passive plantar flexion and dorsiflexion of the foot
	Hypoesthesia of the lateral foot
Deep posterior compartment	Pain on dorsiflexing the toes and everting the foot
	Hypoesthesia of the plantar surface of the foot

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Initial stabilization of injuries and measurement of compartment pressures dictate subsequent treatment and disposition.
- Pressures of 10 to 20 mmHg require reevaluation in 12 to 24 h, with serial measurements of compartment pressure in persistently symptomatic patients. Judgment must be exercised regarding whether to discharge patients based upon their likelihood to follow discharge instructions and return for close follow-up.
- Pressures greater than 20 mmHg can compromise capillary blood flow and require hospital admission or surgical consultation, as persistent pressures in this range can damage nerve and muscle.
- Pressures over 30 to 40 mmHg place nerve and muscle at risk for necrosis and are grounds for immediate fasciotomy. This is accomplished with a longitudinal incision of the skin and fascia to release the contents of the compartment. The goal of treatment is the avoidance of muscle necrosis, rhabdomyolysis, and nerve damage in the affected area(s).[4]

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174 RHABDOMYOLYSIS

Stefanie R. Seaman

PATHOPHYSIOLOGY

- Rhabdomyolysis is a syndrome comprising injury to skeletal muscle followed by muscle cell necrosis and release of intracellular contents. The common cellular event involves disruption of the Na^+-K^+ ATPase pump and calcium transport. The result is increased intracellular calcium and muscle cell necrosis.
- The most common causes of rhabdomyolysis are alcohol and drug abuse. Other causes include toxin ingestion, trauma, infection, strenuous physical activity, seizures, and heat-related illness.
- Alcohol causes rhabdomyolysis secondary to muscle compression and a direct toxic effect. Malnutrition and hypophosphatemia in alcoholics also increase the risk. Alcohol contributes to 20 percent of all cases of rhabdomyolysis.[1]
- Drugs of abuse implicated in acute rhabdomyolysis are cocaine, amphetamines, lysergic acid diethylamide (LSD), heroin, and phencyclidine (PCP).
- Medications that cause rhabdomyolysis include diuretics, narcotics, theophylline, corticosteroids, benzodiazepines, phenothiazines, and tricyclic antidepressants.
- Risk factors for rhabdomyolysis include poor physical conditioning, inadequate fluid intake, high ambient temperatures, and high humidity levels.[2]

CLINICAL FEATURES

- Symptoms of rhabdomyolysis include myalgias, stiffness, weakness, malaise, low-grade fever, and dark (tea-colored) urine. Brownish urine may be absent in mild cases.
- Nausea, vomiting, abdominal pain, palpitations, or mental status change may stem from uremic encephalopathy.
- Muscles may swell after rehydration with IV fluids. Postural muscles of the lower back, thighs, and calves are most commonly involved.
- Complications of rhabdomyolysis include acute renal failure, hyperuricemia, hyperkalemia, hyperphosphatemia (early), hypocalcemia (early), disseminated intravascular coagulation (DIC) with hypercalcemia and hypophosphatemia (late), and compartment syndrome with peripheral neuropathy.
- Rhabdomyolysis accounts for 5 to 8 percent of all cases of acute renal failure.[3–5] Factors contributing to acute renal failure are hypovolemia, acidosis, tubular obstruction, and the nephrotoxic effects of myoglobin. Neither the presence of myoglobin nor the degree of creatinine phosphokinase (CPK) elevation is predictive of which patients will develop acute renal failure.

DIAGNOSIS AND DIFFERENTIAL

- A fivefold or greater increase in the level of CPK is the hallmark for the diagnosis of rhabdomyolysis. CPK levels rise 2 to 12 h after injury and peak at 24 to 72 h. Levels decline at a rate of 40% daily.
- Muscle necrosis and breakdown releases myoglobin. Myoglobin spills in the urine after plasma levels exceed 1.5 mg/dL. Brownish urine occurs when urine myoglobin exceeds 100 mg/dL. Myoglobin contains heme and will test positive for occult blood. Myoglobin radioimmunoassays are only slightly more sensitive and are not required for the diagnosis.
- Laboratory studies that should be ordered include serum electrolytes, blood urea nitrogen (BUN), creatinine, calcium, phosphorus, uric acid levels, urinalysis, complete blood cell count, and DIC profile.
- Differential diagnosis includes sickle cell disease, toxin exposure, inflammatory myopathies, and infectious myalgias.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- The mainstay of treatment is IV rehydration with crystalloid to maintain a urinary output of at least 2 mL/kg/h. This should be continued for 24 to 72 h.
- Sodium bicarbonate (1 mmol/kg IV bolus) to maintain urinary pH above 6.5 has been recommended to decrease ferrihemate production.
- Furosemide 40 to 200 mg IV may be administered to assist in maintaining urinary output. Diuretics should be administered only after adequate volume replacement has been instituted.
- Electrolyte disorders, including hyperkalemia, should be treated in the standard fashion.

- Nephrology should be consulted if the patient develops early signs of acute renal failure.

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Section 22

MUSCULAR, LIGAMENTOUS, AND RHEUMATIC DISORDERS

175 CERVICAL, THORACIC, AND LUMBAR PAIN SYNDROMES

Gary M. Gaddis

EPIDEMIOLOGY

- Cervical disk herniations are 1½ times more common in males and are most common during the fourth decade.
- Spinal spondylosis and stenosis become more common as patients age.
- Thoracic compression fractures become more common with advanced age, especially in osteoporotic females.
- Thoracic and lumbar spinal fractures are most common at T10 to L2 from direct trauma or from hyperflexion injuries such as trunk flexion about a seat belt.[1]
- Lumbar pain is responsible for more work absenteeism than any illness except the common cold. Some 60 to 90 percent of persons will experience back pain at some time. The annual prevalence of low back pain in working adults may approach 50 percent.[2,3]
- Low back pain has disabled over 5 million individuals. In 85 percent of these, no definite source of the pain can be diagnosed.[4]

PATHOPHYSIOLOGY

- Segmental motor or sensory signs associated with a nerve root disorder are called *radiculopathies.* Signs and symptoms due to spinal cord disorders are called *myelopathies.*
- Pain can be generated from any innervated structure. Pain due to nerve irritation can be perceived locally and distally.
- Neck trauma may cause soft tissue hemorrhage or edema between any of the seven fascial planes of the neck, resulting in limited range of motion, pain, or swelling.
- Rear-end motor vehicle crashes tend to cause hyperextension of the neck, which explains associations with hyperextension dislocations, atlas fractures, extension teardrop fractures, posterior arch fractures, laminar fractures, or traumatic spondylolisthesis.
- Head-on motor vehicle crashes tend to cause hyperflexion neck injuries, such as anterior subluxations, unilateral or bilateral facet dislocations, vertebral compression fractures, and spinous process avulsions.
- Direct posterior cervical disc herniations can produce progressive myelopathies. Posterolateral herniations occur more commonly and produce cervical radiculopathy.
- Osteophytes and/or buckling of the ligamentum flavum can provoke cervical stenosis. Myelopathy due to stenosis becomes more frequent as the diameter of the spinal canal is reduced to less than 12 mm.
- The spinal canal and the canals for the paired segmental nerves are narrowest in the thoracic spine, so the thoracic spine is especially prone to compromise from compressing or space-occupying lesions.
- Thoracic vertebral compression fractures seldom cause neurologic compromise. Fractures may be due to direct trauma, osteoporosis, or hyperflexion injuries.
- Pathologic thoracic fractures associated with metastatic lesions are more likely to present with myelopathy and long tract signs.

- Since the spinal cord ends at the first or second lumbar vertebra in adults and the size of the spinal canal is larger in the lumbar than the thoracic region, the rate of neurologic compromise observed with lumbar bony injuries is decreased.

CLINICAL FEATURES

- Patients with neck pain often have associated stiffness and decreased range of motion. Generally, an identifiable inciting position or provocative maneuver can reproduce pain. Localized neck tenderness to palpation may be absent.
- Neck pain radiating in a dermatomal pattern suggests a cervical radiculopathy. Radiculopathies may present with neurogenic signs such as sensory abnormalities, weakness, muscle hypertonicity, reflex changes, or incoordination. Myelopathy may be suggested by sexual or sphincter dysfunctions.
- Neck extension and lateral flexion should exacerbate radicular neck pain. Flexion and distraction should relieve radicular neck pain.
- Signs and symptoms of cervical radiculopathy are summarized in Table 175-1.
- Thoracic pain syndromes may lack localized pain or tenderness of the spine. Lesions at the thoracic root typically cause pain worsened by reclining and improved by upright positioning.
- Facet syndrome is a degenerative process that causes 15 to 40 percent of chronic back pain. It is a diagnosis of exclusion and can be confirmed by relief with analgesic injection.[5]
- Osteoarthritis can cause localized stiffness, radicular pain, or spinal stenosis. Spinal stenosis is most common in the thoracic spine.
- Long tract signs such as hyperreflexia, an extensor toe sign (Babinski sign), urinary incontinence, or other neurologic deficit suggest that both intrinsic and extrinsic spinal cord pathology must be suspected.
- Herpes zoster neuralgia and diabetic radiculopathy may affect any spinal level. Pain from zoster may precede the development of rash. Diabetic neuropathy may cause radicular symptoms, with chest, abdominal, or hip pain.
- Lumbar radiculopathies are summarized in Table 175-2.
- A crossover straight-leg-raising sign (CSLR) is pain in the symptomatic leg elicited by elevation of the other leg. A CSLR sign is a stronger indication of nerve root compression than a straight-leg-raising (SLR) sign on the affected side. Head flexion should exacerbate pain when the leg is held where the SLR maneuver elicits the limit of pain tolerable to the patient. Patients with lumbar radiculopathy tend to lean backward to relieve tension on the nerve.[6]

DIAGNOSIS AND DIFFERENTIAL

- At any level, large spinal disk herniations, significant spinal stenosis, epidural hematomas, epidural abscesses, and epidural neoplasms can present

TABLE 175-1 Signs and Symptoms of Cervical Radiculopathy

DISK SPACE	CERVICAL ROOT	PAIN COMPLAINT	SENSORY ABNORMALITY	MOTOR WEAKNESS	ALTERED REFLEX
C1-C2	C1-C2	Neck, scalp	Scalp		
C4-C5	C5	Neck, shoulder, upper arm	Shoulder, thumb	Spinati, deltoid, biceps	Reduced biceps reflex
C5-C6	C6	Neck, shoulder, upper medial, scapular area, proximal forearm, thumb	Thumb and index finger, lateral forearm	Deltoid, biceps, pronator teres, wrist extensors	Reduced biceps and brachioradialis reflex
C6-C7	C7	Neck, posterior arm, dorsum proximal forearm, chest, medial $\frac{1}{3}$ scapula, middle finger	Middle finger, forearm	Triceps, pronator teres	Reduced triceps reflex
C7-T1	C8	Neck, posterior arm, medial proximal forearm, median inferior scapular border, medial hand, ring and little fingers	Ring and little fingers	Triceps, flexor carpi ulnaris, hand intrinsics	Reduced triceps reflex

TABLE 175-2 Symptoms and Signs of Lumbar Radiculopathies

DISK SPACE	NERVE ROOT	PAIN COMPLAINT	SENSORY CHANGE	MOTOR WEAKNESS	ALTERED REFLEX
L2-3	L3	Medial thigh, knee	Medial thigh, knee	Hip flexors	None
L3-4	L4	Medial lower leg	Medial lower leg	Quadriceps	Knee jerk
L4-5	L5	Anterior tibia, great toe	Medial foot	Extensor hallucis longus	Biceps femoris
L5-S1	S1	Calf, little toe	Lateral foot	Foot plantar flexors	Achilles

with similar neurologic findings of radiculopathy and/or myelopathy.

- At all spinal levels, computed tomography (CT) or magnetic resonance imaging (MRI) is useful in the workup of suspected radiculopathy or myelopathy, especially when compressive or neoplastic lesions are suspected.[7] Long tract signs such as hyperreflexia, Babinski's sign, or urinary incontinence imply that intrinsic or extrinsic spinal cord pathology must be suspected. Many causes of such spinal cord pathology require rapid diagnosis for optimal outcomes.
- Plain radiographs are useful for selected types of trauma, especially if the patient is elderly or if the mechanism of injury would lead to suspicion of fracture. Radiographs, however, are of little value with small or even large disk herniations.
- For suspected cervical lesions, range-of-motion testing, spine compression, and distraction techniques to assess for pain and radicular symptoms are critical. Thorough assessment of distal upper extremity pulses and a complete neurologic examination are important.
- History of IV drug use, infections of the skin or urinary tract, and immune suppression are risk factors for spinal infection. Fever is not reliably present with such infections. A complete blood cell count, urinalysis, and erythrocyte sedimentation rate can be useful in screening for infection in patients at risk for spinal infection.[6]
- Lumbosacral pain carries a long differential diagnosis, which includes degenerative problems of the spine, and potentially life-threatening problems remote from the spine itself, such as abdominal aneurysm. Spinal, neurologic, abdominal, vascular, and lower extremity evaluation should occur with lumbar pain syndromes.
- ''Red flag'' conditions as compiled by the Agency for Health Care Policy and Research for the development of guidelines for the evaluation of low back pain include unexpected anal sphincter laxity, perianal or perineal sensory loss, major lower extremity motor weakness, cauda equina syndrome (saddle anesthesia, bladder dysfunction of recent onset, progressive lower extremity neurologic deficit), a history suggestive of tumor or infection, or a history suggestive of fracture.[6,8]
- Lumbosacral radiographs are indicated in trauma patients and in the elderly but are not helpful for most cases involving a lumbar pain syndrome.
- Lower extremity pain exacerbated by ambulation can be characteristic of lumbar spinal stenosis or lower extremity arterial insufficiency. The following are associated with spinal stenosis: age greater than 65 years, absence of pain when seated, wide-based gait, and production of thigh pain with sustained lumbar extension of 30 s.[9] Arteriography, and CT, or MRI may be needed to differentiate the cause of pain exacerbated by ambulation.
- Waddell's nonorganic physical signs[10] can be used for challenging cases. These include perceived tenderness to superficial skin rolling, low back pain exacerbated by axial loading of the head, low back pain elicited by whole-body rotation, lack of pain with seated SLR but pain with a low angle of SLR, weakness or sensory loss in a nonanatomic distribution, and overreaction with pain behaviors.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Care of patients with significant spinal trauma is discussed in Chap. 158.
- Patients with a progressive neurologic deficit or with myelopathy should be admitted for an expedient workup. Some causes of extrinsic pain, such as epidural abscess or neoplastic spinal cord compression, must be diagnosed and treated quickly to preserve optimal neurologic function.
- Most patients can be managed as outpatients with conservative therapy, including NSAIDs, relative rest, cold and heat applications, and other supportive measures. Bed rest is reserved only for severe pain and should not exceed 2 days. Short-term

opioid treatment may be warranted for supplemental pain relief. Referral for follow-up evaluation and care is critical.[6]
- Close outpatient follow-up should be offered to monitor for appearance or progression of neurologic deficits and to monitor the patient's progress.

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176 SHOULDER PAIN

Gary M. Gaddis

EPIDEMIOLOGY

- Rotator cuff impingement injury is the most common cause of intrinsic shoulder pain. This injury continuum ranges from subacromial bursitis, through rotator cuff tendinitis, to partial and full thickness rotator cuff tears. Patients <25 years are most susceptible to subacromial bursitis. Patients <40 years are unlikely to have rotator cuff tears.
- Adhesive capsulitis (''frozen shoulder'') is most common in postmenopausal, diabetic women <70 years. It is only rarely associated with rotator cuff tears and is frequently associated with prior immobilization, trauma, or cervical disk disease.

PATHOPHYSIOLOGY

- The muscles of the rotator cuff (supraspinatus, infraspinatus, teres minor, and subscapularis) are dynamic stabilizers of the glenohumeral joint and provide much of the power for shoulder movement. The muscles must function within the coracoacromial arch, between the humeral head and the coracoid, acromion, and acromioclavicular ligament. They also function beneath the deltoid muscle and subacromial bursa. The rotator cuff is therefore prone to compression and impingement.
- The biceps tendon inserts on the glenoid labrum after passing between the subscapularis and supraspinatus tendons and assists with rotator cuff function. The long head of the biceps can become impinged due to its location. The tendon can become subluxed or dislocated out of the bicipital groove of the humerus or can rupture.
- Activities that cause repeated compression of these structures can cause impingement syndromes. The supraspinatus muscle or its tendon is the most commonly injured rotator cuff structure.
- Calcific tendinitis, associated with reversible calcium hydroxyapatite deposition within one or more rotator cuff tendons, is most common in the supraspinatus tendon.
- Adhesive capsulitis is associated with idiopathic fibrosis and scarring of the shoulder joint capsule.

CLINICAL FEATURES

- Calcific tendinitis causes pain with any motion of the shoulder. Osteoarthritis causes pain with activity and is relieved with rest.
- Decreased range of motion, crepitus, weakness, or atrophy of shoulder muscles may accompany various causes of shoulder pain, especially the more severe impingement syndromes.
- Neer's test involves compressing the rotator cuff and subacromial bursa as the examiner forcibly but smoothly fully abducts the straightened arm. Pain is associated with a positive test.
- Hawkins' test involves inward rotation of an arm previously placed in 90° of abduction and 90° of elbow flexion. Inward rotation of the arm across the front of the body compresses the rotator cuff and bursa between the coracoacromial ligament and the humeral head. Pain is associated with a positive test.
- Acute injuries to the rotator cuff generally involve acute traumatic forced hyperabduction or hyperextension of the shoulder.
- Calcific tendinitis causes sudden onset of shoulder pain, usually at rest, and is exacerbated by any shoulder motion. It is usually worse at night and coincides with resorption of the calcium deposit. The pain generally is self-limited after two weeks. Some patients have calcific deposits on shoulder radiographs long before they develop shoulder pain, and over 60 percent with calcifications never develop pain.
- Adhesive capsulitis often follows periods of immobilization of the shoulder and causes diffuse aching, especially at night, and limited passive and active range of motion. Pain is reproduced at the limits of motion, but not by palpation.
- Primary osteoarthritis is associated with degenerative disease in other joints.

DIAGNOSIS AND DIFFERENTIAL

- Subacromial bursitis usually occurs before age 25 and is commonly associated with positive impingement tests and tenderness at the lateral proximal humerus or in the subacromial space. Rotator cuff tendinitis is more common between ages 25 to 40 and involves signs of impingement, along with tenderness of the rotator cuff, and, often, demonstrable rotator cuff muscular weakness.
- Rotator cuff tears are more common after age 40. Tears may be partial or full thickness. Only about 10 percent are due to acute trauma. Commonly associated findings are muscular weakness, especially with abduction and external rotation, cuff tenderness, muscular atrophy, and impingement signs. Crepitus suggests more chronic injury.
- Osteoarthritis is often present in multiple joints, but is especially likely in a previously injured shoulder.
- Adhesive capsulitis is characterized by a generalized decreased range of motion, often after a period of immobilization.
- Radiographs are rarely diagnostic, but help detect abnormal calcifications with calcific bursitis, osteophytes or other arthritic changes, or subtle glenohumeral dislocations, which can be mistaken for adhesive capsulitis.
- Extrinsic causes of shoulder pain should be considered in the differential, and these include acute cardiac, pulmonary, aortic, and abdominal pathology. Also cervical spine radiculopathy, brachial plexus disorders, Pancoast's tumor, and axillary artery thrombosis must be considered in the evaluation of shoulder pain.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Reduction of pain and inflammation are the goals of emergency department care. This usually involves nonsteroidal anti-inflammatory drugs, "relative rest," and immobilization. Relative rest means avoidance of painful activities.
- A potential complication of local steroid injection (e.g., triamcinolone 20 to 40 mg) is tendon rupture.

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177 ACUTE DISORDERS OF THE JOINTS

Lance H. Hoffman

SEPTIC ARTHRITIS

- Bacterial infection of the joint space presents as a monarticular arthritis that can destroy the joint in a few hours to days. The patient may lack fever, chills, and malaise.
- While an elevated erythrocyte sedimentation rate is insensitive for septic arthritis in adults, it is 90 percent sensitive in children and infants with septic arthritis.[1,2] The white blood cell count lacks sensitivity and specificity in both adults and children with septic arthritis.
- Synovial fluid analysis usually reveals cloudy fluid with leukocytes >50,000 and cultures that are positive more than 50 percent of the time.
- Septic arthritis requires admission to the hospital for parenteral antibiotics, generally a combination of nafcillin and a third-generation cephalosporin, and orthopedic consultation for possible surgical drainage.

GONOCOCCAL ARTHRITIS

- Gonococcal arthritis is the most common cause of septic arthritis in adolescents and young adults and usually presents with fever, chills, and migratory arthralgias or tenosynovitis preceding a monarthritis.[3]
- Vesiculopustular lesions may be present distal to the involved joint.
- Synovial fluid cultures are often negative. However, cultures of the posterior pharynx, urethra, cervix, and rectum may increase the yield of isolating the organism.[3]

TRAUMATIC HEMARTHROSIS

- Hemarthrosis has a high association with intraarticular fracture and ligamentous injury.
- The synovial fluid aspirate may show fat droplets if an intraarticular fracture is present.
- Spontaneous hemarthrosis should prompt an investigation for a coagulopathy.

CRYSTAL-INDUCED SYNOVITIS

- Gout—uric acid crystal deposition—is the most common cause of inflammatory joint disease in men over the age of 40 years and typically affects the great toe, tarsal joints, or knee.[4]
- Up to 30 percent of patients with acute gout will have normal serum uric acid levels making this test of little utility in diagnosing gout.[4]
- Pseudogout—calcium pyrophosphate crystal deposition—typically affects the knee, wrist, ankle, or elbow.
- Synovial fluid analysis will reveal negative birefringent needle-shaped uric acid crystals in gout and weakly positive birefringent rhomboid calcium pyrophosphate crystals in pseudogout.
- Acute treatment is with indomethacin 50 mg PO tid for 3 to 5 days, adrenocorticotropic hormone 40 U intramuscularly (IM), or colchicine 0.6 mg PO q h until efficacy ensues or the patient experiences intolerable gastrointestinal side effects.

OSTEOARTHRITIS

- Osteoarthritis is a chronic, oftentimes symmetric, arthritis lacking constitutional symptoms, which is caused by destruction of the articular, hyaline cartilage.
- Radiographs may show joint space narrowing, sclerosis, or osteophyte formation.
- Acute pain is treated with nonsteroidal antiinflammatory drugs (NSAIDs) and resting the affected joint.

LYME ARTHRITIS

- Lyme arthritis is the result of a tick bite that infects the host with the spirochete *Borrelia burgdorferi.*
- Lyme arthritis manifests as a monarticular or symmetric oligoarticular arthritis, primarily affecting the large joints, with alternating periods of exacerbation and complete remission.
- Synovial fluid cultures are usually positive.
- Treatment of Lyme arthritis consists of 3 to 4

weeks of doxycycline, penicillin, amoxicillin, or erythromycin.

ACUTE RHEUMATIC FEVER

- Acute rheumatic fever is the result of an untreated group A β-hemolytic streptococcus infection that causes an immune-mediated migratory polyarthritis 3 to 4 weeks after the infection ensued.
- Diagnosis of acute rheumatic fever requires the presence of two major criteria (e.g., carditis, arthritis, chorea, erythema marginatum, and subcutaneous nodules) or one major and two minor criteria (e.g., fever, arthralgia, history of rheumatic fever, and elevated acute phase reactants).
- Treatment should include analgesics and penicillin or erythromycin.

REITER'S SYNDROME

- Reiter's syndrome is a seronegative spondyloarthropathy that manifests as an acute, asymmetric oligoarthritis with a predilection for the lower extremities that was preceded 2 to 6 weeks earlier by an infectious illness, usually urethritis or enteritis.
- The classic triad of urethritis, conjunctivitis, and arthritis is not mandatory for diagnosis.
- Nonsteroidal anti-inflammatory drugs should be used for the symptomatic treatment of joint pain.

ANKYLOSING SPONDYLITIS

- Ankylosing spondylitis is a seronegative spondyloarthropathy primarily affecting the spine and pelvis, which is characterized by morning stiffness, fatigue, and weakness.
- Ankylosing spondylitis is associated with HLA-B27 antigen positivity.
- Classic radiographic findings include sacroiliitis and squaring of the vertebral bodies (e.g., bamboo spine).
- Joint pain should be treated symptomatically with NSAIDs.

RHEUMATOID ARTHRITIS

- Rheumatoid arthritis is a chronic, symmetric, polyarticular synovial joint disease, pathologically characterized by synovial pannus formation. This disease is also associated with morning stiffness, depression, fatigue, and myalgias.
- The atlantoaxial joint may be involved in the disease process resulting in joint instability and the possibility of neurologic injury with minor trauma.
- Acute exacerbations of joint pain are treated with immobilization of the affected joint, NSAIDs, and corticosteroids.

VIRAL ARTHRITIS

- Common viral illnesses including rubella, hepatitis B, enteroviruses, adenoviruses, mumps, and Epstein-Barr virus can cause an acute, symmetric, polyarticular, immune-mediated arthritis.
- Treatment is supportive with NSAIDs.

BURSITIS

- Bursitis is an inflammatory process involving any bursae. It can be caused by infection, trauma, rheumatologic disorders, or crystal deposition or be idiopathic.
- Commonly affected bursae include the prepatellar bursa (e.g., carpet layer's knee) and the olecranon bursa (e.g., student's elbow).
- Septic and aseptic bursitis cannot reliably be differentiated by physical exam alone so aspiration of bursal fluid is required for cell count and differential, gram stain, and culture.[5–8]
- Treatment entails resting the affected joint, a compressive dressing, analgesics, and antistaphylococcal antibiotics (e.g., amoxicillin clavulonate, dicloxacillin, or cephalexin) for 10 to 14 days if there is evidence of infection.

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178 MUSCULOSKELETAL DISORDERS IN ADULTS

Michael P. Kefer

RHEUMATIC EMERGENCIES ASSOCIATED WITH RISK OF DEATH

RESPIRATORY SYSTEM

- Death may result from airway obstruction, respiratory muscle failure, or pulmonary tissue involvement.
- Relapsing polychondritis begins with abrupt onset of pain, redness, and swelling of the ears or nose. The tracheobronchial cartilage is involved in approximately 50 percent of cases and manifests as hoarseness and throat tenderness. Repeated attacks can lead to airway collapse. Patients should receive high-dose steroids and be admitted for observation.
- Rheumatoid arthritis (RA) may involve the cricoarytenoid joints causing dysphonia, hoarseness, or stridor. The joints may fix in a closed position mandating emergency tracheostomy.
- Dermatomyositis and polymyositis involving the respiratory muscles may lead to respiratory failure.
- Pulmonary hemorrhage occurs in Goodpasture's disease, systemic lupus erythematosus (SLE), Wegener's granulomatosis, and other vasculitic conditions.
- Pulmonary fibrosis occurs in ankylosing spondylitis and scleroderma.
- Pleural effusion occurs in RA and SLE.

HEART

- Pericarditis occurs in RA and SLE.
- Myocardial infarction occurs in Kawasaki's disease and polyarteritis nodosa.
- Pancarditis occurs in acute rheumatic fever.
- Valvular heart disease occurs in ankylosing spondylitis, relapsing polychondritis, and rheumatic fever. Involvement may extend into the conduction system.

ADRENAL GLANDS

- Glucocorticoids are commonly used for treatment of many rheumatic conditions. Any acute stress may result in adrenal insufficiency when the demand for glucocorticoids exceeds the supply. Stress-dose steroid therapy is indicated.

RHEUMATIC PRESENTATIONS ASSOCIATED WITH RISK OF MORBIDITY

CERVICAL SPINE AND SPINAL CORD

- Rheumatoid arthritis may result in ligamentous destruction of the transverse ligament of C2 causing symptoms of spinal cord compression.
- Ankylosing spondylitis may result in cervical spine inflexibility that predisposes to injury out of proportion to the mechanism.
- Anterior spinal artery syndrome may result from rheumatologic conditions that cause vasculitis, aortic dissection, or thromboembolism.

EYE

- Temporal arteritis should be considered in any patient >50 years who presents with new onset headache, visual changes, or jaw claudication. Laboratory evaluation reveals an elevated erythrocyte sedimentation rate (>50 mm/h), anemia, and elevated alkaline phosphatase. High-dose steroid therapy should be initiated immediately. Temporal artery biopsy for definitive diagnosis should be obtained within the next 7 days to be accurate.

- Dry eyes are a common manifestation of Sjögren's syndrome.
- Episcleritis occurs in RA. It is a self-limited, painless injection of the episcleral vessels.
- Scleritis also occurs in RA. It presents with marked ocular tenderness and a purple discoloration to the eye. The high risk of visual loss and scleral rupture mandate high-dose steroid therapy and emergent ophthalmologic consult.

HYPERTENSION

- Hypertension may result from any rheumatologic condition that affects the kidneys directly, as in SLE, or indirectly, secondary to nephrotoxic drugs used to treat the underlying condition.

KIDNEY

- Renal insult may be due to either the primary disease process, the drugs used to treat the disease, or both.
- Nephritis may result from SLE or Wegener's granulomatosis.
- Nephrotic syndrome may result from SLE and predisposes to renal vein thrombosis.
- Renal insufficiency may result from prostaglandin inhibition by nonsteroidal anti-inflammatory drugs or from rhabdomyolysis associated with advanced myositis.

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179 INFECTIOUS AND NONINFECTIOUS INFLAMMATORY CONDITIONS OF THE HAND

Mark E. Hoffmann

INFECTIOUS HAND CONDITIONS

EPIDEMIOLOGY

- A history of chronic illness or immunodeficiency increases the risk of hand infection by atypical pathogens.[1,2]

PATHOPHYSIOLOGY

- Three mechanisms are responsible for hand infections: superficial injury with extension, penetrating injury with deep seeding, and, rarely, hematogenous spread.[3]
- Hand infections spread along fascial planes and into adjacent compartments.
- The bacterial etiology of hand infections depends on the source of the offending inoculum. *Staphylococcus* and *Streptococcus* species, which colonize the skin, are the most common agents of infection.[4]
- Intravenous drug abusers typically present with an abscess or deep space infection secondary to *Staphylococcus aureus* by direct needle inoculation or by hematogenous spread from endocarditis.[3]
- Paronychia and felons are polymicrobial (mostly anaerobes) caused by local trauma and contamination with saliva related to chewing on the fingernails.
- Animal bites are related to the oral flora of the offender. Cat bites are associated with *Pasteurella multocida* and human bites with *Eikenella corrodens*.

CLINICAL FEATURES

- Cellulitis presents as a superficial infection with localized warmth, erythema, and edema. The assessment of deeper structures by inspection, palpation, and function testing is required to exclude the more serious deep space infections.
- The diagnosis of pyogenic flexor tenosynovitis is based on the following four criteria: (1) pain with passive extension, (2) tenderness over the flexor

tendon sheath, (3) a flexed position of the involved digit, and (4) symmetric swelling of the finger.
- Deep web space infections occur after penetrating injury and present with dorsal and volar swelling.
- Deep midpalmar space infections occur from spread of a flexor tenosynovitis or a penetrating wound to the palm. The infection involves the radial or ulnar bursa of the hand.
- Closed-fist injury is essentially a human bite wound to the metacarpophalangeal (MCP) joint of the hand sustained by striking another human on the teeth with a closed fist. Initial positioning of the hand (clenched fist/flexion of the MCP) during the examination is essential for identifying extensor tendon injuries. Infection rates are extremely high.
- Paronychia is a localized infection of the lateral nail fold. In advanced stages, a purulent fluid collection may be visualized beneath the nail.
- Felon is an infection of the pulp space of the fingertip. Pain results from distention by a purulent fluid collection within the fibrous septa of the finger pad.
- Herpetic whitlow is a viral infection of the fingertip involving intracutaneous vesicles. It presents in a similar fashion to a felon.

DIAGNOSIS AND DIFFERENTIAL

- Hand infections may have some overlap in specific entities. However, with a thorough history and a careful examination (inspection, palpation, sensorimotor testing, and a range-of-motion evaluation), specific entities may be delineated. Noninfectious hand conditions, including occult fractures, should be included in the differential.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Treatment of cellulitis consists of antibiotics (first-generation cephalosporin or antistaphylococcal penicillin), splinting in the position of function, elevation, and 24-h close follow-up care.[5] Vancomycin should be administered to patients who are IV drug abusers.
- Flexor tenosynovitis is a surgical emergency. Treatment consists of IV antibiotics (β-lactamase inhibitor or first-generation cephalosporin and a penicillin), splinting, elevation, and orthopedic consult. Ceftriaxone should be administered if *Neisseria gonorrhoeae* is suspected.
- Deep space infections are treated with IV antibiotics (β-lactamase inhibitor or first-generation cephalosporin and a penicillin), splinting, elevation, and orthopedic consult. Patients should be admitted.
- Closed-fist injuries are treated with IV antibiotics (β-lactamase inhibitor or first-generation cephalosporin and a penicillin), copious irrigation, splinting, elevation, and orthopedic consult for admission. Radiographs should be obtained to exclude fractures.
- Treatment of paronychia consists of incision and drainage with a no. 11 blade. After digital block, a lateral incision in the same plane as the nail (scalpel flush to the nail) may be made for a small paronychia. A direct incision over the greatest area of fluctuance also may be made. Partial nail removal may be required. Antibiotics (first-generation cephalosporin or antistaphylococcal penicillin), warm soaks, elevation, immobilization, and close follow-up are indicated.[6]
- Treatment of felon also consists of incision and drainage with a no. 11 blade after a digital block. A unilateral longitudinal approach just volar to the neurovascular bundle is most commonly used. The incision begins 5 mm distal to the distal interphalangeal crease and extends up to the fingertip. Antibiotics (first-generation cephalosporin or antistaphylococcal penicillin), a sterile packing with a cover dressing, splinting, elevation, and close follow-up should be arranged.
- Treatment of herpetic whitlow consists of protection with a dry dressing (to prevent autoinoculation and transmission), immobilization, and elevation. Antiviral agents such as acyclovir may shorten the duration.[7]

NONINFECTIOUS HAND CONDITIONS

PATHOPHYSIOLOGY

- Tendonitis and tenosynovitis are inflammatory states involving the flexor or extensor tendons of the hand; overuse and repetitive motion are usually involved.
- Trigger finger is a tenosynovitis in the flexor sheath of a digit with catching due to stenosis and fibrosis in the vicinity of the A1 pulley.
- De Quervain's tenosynovitis is a common inflammatory condition associated with overuse of the thumb (extensor pollicis brevis and abductor pollicis longus tendons).
- Carpal tunnel syndrome is a peripheral mononeuropathy that involves entrapment of the median

nerve in the carpal canal. Direct trauma, overuse, pregnancy, and congestive heart failure may cause swelling below the transverse carpal ligament that roofs the canal, resulting in the compression and partial compromise of the median nerve.
- Dupuytren's contracture is a poorly understood disorder resulting in fibrous changes of the subcutaneous tissues of the palm and volar aspects of the fingers.

CLINICAL FEATURES

- Tendonitis and tenosynovitis present with pain and swelling over the tendons. Palpation produces tenderness and active/passive movements result in worsened pain.
- Patients with a trigger finger may describe a sensation of locking or binding of the tendon after flexion. A painful snap may be experienced with unlocking.
- De Quervain's tenosynovitis usually presents with pain along the radial aspect of the wrist, which extends into the forearm. Finkelstein's test (pain elicited with passive stretch of the tendons by placing the thumb within the palm of the hand in conjunction with ulnar deviation) confirms the diagnosis.
- Carpal tunnel syndrome presents with pain and numbness of the palm in the distribution of the median nerve. Tinel's sign (dysesthesia produced by tapping over the volar aspect of the wrist) and Phalen's sign (paresthesia produced with maximal flexion at the wrist for 1 min) are supportive of the diagnosis.
- Dupuytren's contracture presents with firm longitudinal thickening and nodularity of the superficial tissues, which limit hand function and range of motion. Palpation of the distal palmar crease at the ring or small finger may identify nodules. The patient will usually have the classic flexion contracture.

DIAGNOSIS AND DIFFERENTIAL

- Most conditions are diagnosed clinically. When the suspicion of infectious etiology is high, antibiotic therapy and consultation should follow.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Tendonitis and tenosynovitis are treated with immobilization and nonsteroidal anti-inflammatory drugs (NSAIDs). Physicians may consider injecting triamcinolone 40 mg/mL mixed with 0.5% bupivacaine into the synovial sheath.
- Trigger finger is treated with steroid injections in the early stages, but surgical treatment is definitive.
- De Quervain's tenosynovitis is treated with NSAIDs and a thumb spica splint. Steroid injections may relieve the discomfort.
- Emergency care of carpal tunnel syndrome consists of a wrist splint and NSAIDs. Unresolving cases will require referral for elective surgery.
- Treatment of a Dupuytren's contracture requires referral to a hand surgeon.

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180 SOFT TISSUE PROBLEMS OF THE FOOT

Mark B. Rogers

TINEA PEDIS

- The most common form of tinea pedis is interdigital, usually a fissure between the fourth and fifth digits.
- The web space is often white, macerated, and

soggy owing to the presence of polymicrobial organisms (dermatophytes and bacteria). The lesions may be pruritic and painful.
- Other forms can affect the entire plantar surface, with scaling, erythema, and fissures.
- Topical imidazole antifungals (e.g., miconazole, econazole, ketoconazole, oxiconazole, sulconazole, and tioconazole) are the agents of choice and should be applied for 2 to 3 weeks.
- Alternatively, topical terbinafine or butenafine can be applied for 1 to 2 weeks.
- Oral antifungal therapy (e.g., itraconazole, fluconazole, and terbinafine) for 1 to 2 weeks can be used.[1,2]

ONYCHOMYCOSIS

- Dermatophyte fungi from surrounding skin cause the nail to appear opaque, discolored, and hyperkeratotic.
- High-risk patients include the elderly, diabetics, and immunocompromised.
- Oral antifungal agents (itraconazole, terbinafine, and fluconazole) are first-line treatment because topical agents are poorly absorbed.
- Treatment can be continuous (daily for 12 weeks) or, preferably, given as ''pulse dosing'' (daily for 1 week per month for 3 to 4 months).
- Adjunctive therapy may include surgical or chemical debridement of the nail matrix.[3,4]

ONYCHOCRYPTOSIS (INGROWN TOENAIL)

- Onychocryptosis occurs when part of the nail plate penetrates the nail sulcus, usually involving the medial or lateral toenail of the great toe.
- Patients with diabetes, arterial insufficiency, cellulitis, or necrosis are at risk for toe amputation.
- If infection is not present, elevation with a wisp of cotton between the nail plate and skin, daily foot soaks, and avoidance of pressure may be sufficient therapy.
- If granulation tissue or infection is present, partial removal of the nail and debridement are indicated with a wound check in 24 to 48 h.

BURSITIS

- Noninflammatory bursae are pressure-induced lesions over bony prominences.[5]
- Inflammatory bursae are due to gout, syphilis, or rheumatoid arthritis.
- Suppurative bursae are due to pyogenic organisms, usually from adjacent wounds. Nafcillin or oxacillin is the therapy of choice.
- Diagnosis and treatment depend on analysis of the aspirated bursal fluid. Fluid should be sent for cell count, crystal analysis, Gram stain, culture, and protein, glucose, and lactate levels.

PLANTAR FASCIITIS

- Plantar fasciitis is usually caused by overuse or arises in those unaccustomed to activity.
- Patients have point tenderness over the anteromedial calcaneus, which is worse on arising and after activity.
- Plantar fasciitis is usually self limited; the treatment includes rest, ice, and nonsteroidal antiinflammatory drugs (NSAIDs). Severe cases may require a short leg walking cast and podiatric referral.[6]

GANGLIONS

- A ganglion is a benign synovial cyst attached to a joint capsule or tendon sheath.
- The ganglion is often located at the anterolateral ankle. A firm, usually nontender cystic lesion is seen on exam.
- Treatment includes aspiration and injection of glucocorticoids; however, most ganglions require surgical excision.[7]

TENDON LESIONS

- Tenosynovitis or tendonitis usually arise from overuse. Treatment includes rest, ice, and NSAIDs. Tendon lesions should require orthopedic consultation due to their high complication rate.
- Rupture of the Achilles tendon presents with pain, a palpable defect in the area of the tendon, inability to stand on tiptoe, and absence of plantar flexion with squeezing of the calf (Thompson's sign). Treatment is surgical in the young and immobilization in equinus in older patients.
- Rupture of the anterior tibialis tendon, which is rare, results in a palpable defect and mild foot drop.
- Rupture of the posterior tibialis tendon occurs after the fourth decade and is usually chronic and

insidious. Findings include a flattened arch, a palpable defect, and inability to stand on tiptoe.
- Rupture of the flexor hallucis longus tendon presents with loss of plantar flexion of the great toe and must be surgically repaired in athletes.
- Disruption of the peroneal retinaculum occurs with a direct blow during dorsiflexion, causing pain and clicking behind the lateral malleolus as the tendon subluxes. Treatment is surgery.[8]

IMMERSION FOOT (TRENCH FOOT)

- Immersion foot results from prolonged exposure to a moist, nonfreezing (<65°F or <15°C), occlusive environment. It is classically seen in military recruits and the homeless.
- The foot initially becomes pale, pulseless, anesthetic, and immobile but not frozen. With rewarming, one sees hyperemia (lasting up to weeks) with severe burning pain and return of sensation. Edema, bullae, and hyperhidrosis may develop.
- Treatment is admission for bed rest, leg elevation, and air-drying. Normally, antibiotics are not indicated.[9]

FOOT ULCERS

- Ischemic ulcers are due to vascular compromise of larger vessels. The examination shows a cool foot, dependent rubor; pallor on elevation; atrophic, shiny skin; and diminished pulses. Treatment is vascular surgery.[10]
- Neuropathic ulcers are pressure ulcers due to poor sensation. The ulcers are well demarcated with surrounding callus-like material. The foot (in the absence of severe vascular disease) is normal except with regard to sensation. Treatment is relief of pressure and referral to a podiatrist.
- Diabetics may have both ischemic and neuropathic ulcers.[11]
- Infected ulcers require debridement, pressure relief via bed rest or total contact casting, and broad-spectrum IV antibiotics (e.g., ampicillin/sulbactam). Cultures of the drainage fluid and radiographs should be obtained. Vascular surgery consultation and admission are often warranted.
- Palpation of bone in an infected ulcer strongly correlates with osteomyelitis.[12]

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Section 23
PSYCHOSOCIAL DISORDERS

181 CLINICAL FEATURES OF BEHAVIORAL DISORDERS

Lance H. Hoffman

DEMENTIA

- Dementia is a pervasive disturbance in cognitive function, usually of gradual onset, that affects memory, abstract thinking, judgment, and personality.
- The first and second most common causes are Alzheimer's disease and multi-infarct dementia, respectively.
- Common causes of potentially reversible dementia include metabolic and endocrine disorders, polypharmacy, and depression.

DELIRIUM

- Delirium is an impairment of cognitive function characterized by difficulty maintaining attention and alertness (e.g., ''clouding of consciousness'') and sensory misperceptions.
- The onset of delirium tends to be acute and follow a course of fluctuating severity.
- Common causes of delirium are infections, electrolyte imbalances, toxic ingestions, and head injuries.

INTOXICATION

- Intoxication is an impairment of judgment, perception, attention, emotional control, or psychomotor activity resulting from the ingestion of an exogenous substance.

WITHDRAWAL

- Withdrawal is a substance-specific syndrome that occurs following cessation or reduction in use of a substance of abuse.

SCHIZOPHRENIA

- Schizophrenia is a psychotic disorder characterized by functional deterioration; hallucinations (usually auditory), delusions, disorganized speech, or catatonic behavior for at least one month; and the absence of a mood disorder.
- Schizophrenia is the most common psychotic disorder and usually begins in late adolescence or early adulthood.

BRIEF PSYCHOTIC DISORDER

- A brief psychotic disorder is a psychosis of less than 4 weeks duration that begins acutely following a traumatic life experience.

DELUSIONAL DISORDER

- Delusional disorder is characterized by the gradual development of persistent, nonbizarre delusions that do not impair daily functioning.
- Delusional disorder tends to begin in middle or late adulthood.

MAJOR DEPRESSION

- Major depression is a mood disorder that impairs functioning and is more common in women characterized by a persistent dysphoric mood and anhedonia of greater than 2 weeks duration.
- Additional symptoms experienced in major depression include feelings of self-reproach, feelings of hopelessness and worthlessness, loss of appetite, sleep disturbances, fatigue, and an inability to concentrate.
- Recurrent thoughts of death or suicide are common.

DYSTHYMIC DISORDER

- Dysthymic disorder is a chronic, less severe form of depression that does not impair daily functioning. It is characterized by a depressed mood that is present more days than not for at least 2 years.

BIPOLAR DISORDER

- Bipolar disorder is a mood disorder characterized by the episodic occurrence of mania with more frequent episodes of depression.
- Patients experiencing a manic episode are elated, energetic, and expansive, but may rapidly become argumentative or hostile if their goals are blocked or not achieved.
- Signs of mania include a decreased need for sleep, increased activity, pressured speech, and racing thoughts.

PANIC DISORDER

- Individuals with panic disorder experience recurrent episodes of intense anxiety accompanied by autonomic signs including palpitations, tachycardia, dyspnea, chest tightness, dizziness, diaphoresis, and tremulousness.[1]
- Panic attacks generally peak in approximately 10 min and last no more than 1 h.
- Panic disorder is more common in women and tends to manifest in late adolescence to the mid-30s.[1]
- Domestic violence, sexual abuse, or sexual assault are sometimes the source of the panic attacks.
- Effective treatment modalities include cognitive-behavioral therapy and pharmacotherapy with selective serotonin reuptake inhibitors, tricyclic antidepressants, monoamine oxidase inhibitors, or benzodiazepines.[2]

GENERALIZED ANXIETY DISORDER

- Individuals with generalized anxiety disorder experience chronic anxiety without discrete panic attacks.
- Symptoms include apprehensive worrying, muscle tension, insomnia, irritability, restlessness, and distractibility; and these must be present for more than 6 months in order to make the diagnosis.

SIMPLE PHOBIA

- A simple phobia is characterized by intense fear, recognized by the individual as being irrational and excessive, that is invoked by a specific stimulus (e.g., heights, insects, or enclosed spaces).

CONVERSION DISORDER

- Conversion disorder is a diagnosis of exclusion that involves a psychologically produced unconscious loss of physical function in response to a recent psychological stressor.
- Serious organic conditions are developed later in 25 to 50 percent of individuals with conversion disorder.[3,4]
- Physical disorders with nonspecific symptoms such as systemic lupus erythematosus, multiple sclerosis, polymyositis, Lyme disease, and drug toxicity should be considered.
- Patients should be reassured that no serious medical condition is present and that their symptoms will resolve.

SOMATIZATION DISORDER

- Somatization disorder is characterized by the presence of symptoms involving multiple organ systems that do not have an identifiable organic etiology.
- Somatization disorder tends to affect women more than men and often begins in late adolescence and early adulthood.
- These patients may have a history of having had

multiple invasive procedures that yielded normal results.

HYPOCHONDRIASIS

- Hypochondriasis is a preoccupation with the fear that an organic medical illness exists despite normal results of an appropriate medical evaluation and reassurance to the contrary.

PSYCHOGENIC AMNESIA

- Psychogenic amnesia is the temporary loss of memory for important personal information that cannot be attributed to an organic etiology. It often occurs in response to a recent psychological stressor.

PSYCHOGENIC FUGUE

- Psychogenic fugue is psychogenic amnesia accompanied by the individual assuming a new identity in a different geographic location from his or her home.

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182 ASSESSMENT AND STABILIZATION OF BEHAVIORAL DISORDERS

James Hassen, Jr.

ACUTE BEHAVIORAL DISORDERS

CLINICAL FEATURES

- The emergency department (ED) psychiatric assessment needs to determine if the patient: (a) is stable or unstable, (b) has a serious medical condition that is causing the abnormal behavior, (c) has a primarily psychiatric or functional cause for the change in behavior, (d) requires a psychiatric consultation, and (e) should be forcibly detained for evaluation.
- The emergency physician's goal is to distinguish organic from functional disorders.
- The medical-psychiatric history and physical examination are the most effective tools in the evaluation of behavioral disorder.
- Third-party accounts from family, friends, or coworkers are often the only source for obtaining historical information.
- History that should be obtained include: (a) review of systems, (b) description of previous level of functioning, (c) previous psychiatric illness and treatment, (d) history of medications and substance abuse, (e) exposure to toxins, and (f) stressors in the patient's life.
- The sudden onset of major change in behavior or mood usually results from an organic cause.
- A sudden change in behavior, especially in a patient over the age of 40, is a potentially important indicator of a new and correctable process.
- Mental status examination should include assessment of affect, orientation, language, memory, thought context, judgment, and perceptual abnormalities.
- Impaired language performance, including difficulty with speech, reading, writing, and word finding, commonly indicates a neurologic disorder.
- Patients with organic disease often have difficulty spelling backward or performing serial calculations.
- Visual hallucinations favor organic etiologies, while auditory hallucinations favor functional etiologies.
- The inability for a patient to fill in the numbers and hands to form the face of a clock (clock face test) indicates organic disease.

- Physical examination should include the evaluation of abnormal vital signs and the search for signs of trauma.

DIAGNOSIS AND DIFFERENTIAL

- Laboratory tests that should be considered include fingerstick serum glucose, urine and serum drug screens, pregnancy test, electrolytes, computed tomography scan of head, and cerebrospinal fluid analysis.
- Life-threatening disorders that must be ruled out in patients with acute changes in behavior include central nervous system (CNS) infections, intoxications, alcohol withdrawal, hypoglycemia, hypertensive encephalopathy, hypoxia, intracranial hemorrhage, unintentional poisoning, closed cranial trauma, seizure, and acute organ system failure.
- Bradycardia may indicate hypothyroidism, Stoke-Adams syndrome, elevated intracranial pressure, or cholinergic poisoning.
- Tachycardia may indicate hyperthyroidism, infection, heart failure, pulmonary embolism, alcohol withdrawal, anticholinergic toxicity, or sympathomimetic poisoning.
- Fever may indicate thyroid storm, vasculitis, alcohol withdrawal, sedative hypnotic withdrawal, or systemic infection.
- Hypothermia may indicate sepsis, hypoendocrine status, CNS dysfunction, or alcohol intoxication.
- Hypotension may indicate shock, Addison's disease, hypothyroidism, or medication side effect.
- Hypertension may indicate hypertensive encephalopathy or stimulant abuse.
- Tachypnea may indicate metabolic acidosis, pulmonary embolism, cardiac failure, or systemic infection.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Situations that require emergency stabilization involve patients stating that they are potentially or actually violent, suicidal, or developing rapidly progressive medical conditions causing disturbed behavior.
- Physical restraints may be needed to protect patients from harming themselves and others.
- Chemical restraint is indicated when behavior is dangerous despite physical restraints.
- Lorazepam is the agent of choice for control of agitated patients.
- Haloperidol and droperidol are most effective when agitation has psychiatric features.
- Decision to release patients from physical restraints should be made jointly by medical and nursing personnel on the basis of patients' behaviors.

SUICIDE

- The annual rate of suicide in the United States is 1 percent and accounts for 31,000 deaths.
- Those who complete suicide are more likely to be older, male, living alone, physically ill, depressed, schizophrenic, have a history of substance abuse, or have prior suicide attempts.
- Drug overdose accounts for the overwhelming majority of all suicide attempts.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- High-risk patients (those who display hopelessness, depression, and clear suicide intent) require immediate psychiatric hospitalization.
- Moderate-risk patients (those who display positive response to initial intervention and favorable social support) may be treated urgently in the outpatient setting.
- Low-risk patients (those who display suicide threats or minor attempts during an external crisis) may be managed on an outpatient basis once immediate follow-up has been arranged.
- Strict criteria must be followed before discharging a child or adolescent patient with suicidal ideation or behavior from the ED. These include the following: (a) the patient must not be imminently suicidal; (b) the patient must be medically stable; (c) the patient and parents agree to return to the ED if suicidal intent recurs; (d) the patient must not be intoxicated, delirious, or demented; (e) the patient must not have access to potentially lethal means for self-harm; (f) treatment of underlying psychiatric diagnoses has been arranged; (g) acute precipitants to the crisis have been addressed and attempts have been made to resolve them; (h) the physician believes that the patient and family will follow through with treat-

ment recommendations; and (i) the patient's caregivers and social supports are in agreement with the discharge plans.

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Section 24
ABUSE AND ASSAULT

183 CHILD AND ELDERLY ABUSE

Craig E. Krausz

CHILD ABUSE

EPIDEMIOLOGY

- Abused children 8 to 11 years of age frequently state that their abuse has been ongoing for years. The assailant is known in 90 percent of cases.[1]
- Two-thirds of victims of physical abuse are under the age of 3 years, and one-third of victims are under the age of 6 months.

CLINICAL FEATURES

- Abuse in infancy can result in the failure-to-thrive (FTT) syndrome; these children often present to the emergency department (ED) for other common problems, such as diaper rash or gastroenteritis.
- Physical manifestations of FTT include poor physical care and hygiene, little subcutaneous tissue, protruding ribs, loose skin over buttocks, and increased muscle tone.[2]
- The behavioral characteristics of FTT in these children include a wide-eyed and wary appearance, purposeful aversion to eye contact, irritability or fussiness, and assumption of a ''straphanger's position,'' with arms flexed at the elbows and extended over the shoulders.[3]
- Psychosocial dwarfs are children over the age of 2 to 3 years who have suffered neglect and present with the triad of short stature, a bizarre, voracious appetite, and a disturbed home situation. They are frequently hyperactive and have delayed or unintelligible speech.[4]
- In Munchausen's syndrome by proxy (MSBP), a parent induces or fabricates an illness in a child in order to secure for himself or herself prolonged contact with health care providers.[5]
- The most common complaints in MSBP are bleeding, seizures, altered mental status, apnea, diarrhea, vomiting, fever, rash, or multiple organ involvement; the patient's problems may be induced by forced administration of warfarin or ipecac.[6]
- Clinical features of sexual abuse are varied and many children present for genitourinary complaints such as vaginal discharge, vaginal bleeding, dysuria, urinary tract infections, or urethral discharge. Behavioral disturbances may include excessive masturbation, genital fondling or other sexually oriented or provocative behavior, encopresis, and regression.[7]
- Shaken-baby syndrome is caused by vigorous shaking or thrusting down onto a firm surface.[9]
- Clinical features suggestive of physical abuse include:
  1. Bruises, which may be observed over multiple areas, especially the low back, buttocks, thighs, ear pinna, cheeks, neck, ankles, wrists, corners of mouth, and lips.
  2. Handprints or marks of blunt objects.[8]
  3. Lacerations of the frenulum or the oral mucosa, which may be due to forced feeding. Trauma to the genital area in toddlers may be due to ''punishment'' during toilet training.
  4. Immersion burns have a ''glove-and-stocking'' appearance, with sharply demarcated margins.

5. Small, circumferential, scab-covered injuries are suggestive of cigarette burns.
6. Bruising around eyes, ears, and cheeks as well as swelling of the scalp.
7. Retinal hemorrhages, which are associated with intracranial hemorrhage.

DIAGNOSIS AND DIFFERENTIAL

- Histories that are conflicting, inconsistent, or changing with the nature or extent of injuries raise the suspicion of abuse.
- Any serious injury in children <5 years of age should be viewed with suspicion.
- Physicians must have a high level of suspicion for abuse with any anogenital complaints.
- Weight, length, and head circumference should be measured on FTT infants. Weight is affected more then length.[10] Weight gain during the hospitalization is the hallmark of environmental FTT.
- In MSBP, a parent (the mother 98 percent of the time) encourages more diagnostic tests and is uncharacteristically happy with a positive result. In addition, the patient will often present as a medically perplexing case and move from hospital to hospital.
- The diagnosis of sexual abuse can be confirmed by a careful genital and perianal exam. However, since the hymen varies based on age, measurements of the hymen are not reliable.[11–13] Hymeneal notch (concavities or clefts) at the 6 o'clock position is associated with penetrating trauma.[14,15]
- Children with suspected abuse should be evaluated with a complete blood cell count, coagulation studies, and a skeletal survey.
- Rarely, pathologic conditions such as leukemia, aplastic anemia, or osteogenesis imperfecta may mimic child abuse.
- Fractures indicative of inflicted injury include spiral fractures of long bones, metaphyseal chip fractures, multiple fractures at different stages of healing, fractures at unusual sites, and repeated fractures to the same site.
- The absence of physical examination findings does not preclude abuse.
- Abused children are frequently very compliant and submissive and do not resist painful procedures.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- A full social services assessment should be obtained.
- Infants with FTT and MSBP should be admitted.
- Medical care should be directed at physical findings and the nature of the injuries.
- Every state is required to report suspected child abuse cases. Failure to report can result in misdemeanor charges and fine or imprisonment.
- The final disposition of the child is dependent upon a court hearing.

ABUSE IN THE ELDERLY AND IMPAIRED

EPIDEMIOLOGY

- Elder abuse affects 3 to 4 percent of the elderly population.[16–20]

CLINICAL FEATURES

- The elder typically lives with the abuser, who is often dependent upon the elder for housing, financial support, and emotional support. Abuse can come when the caregiver is overwhelmed, frustrated, or resentful with the responsibilities involved in caring for a less than fully independent elder.
- The elder patient's cooperation may be difficult to obtain secondary to embarrassment, fear of abandonment, fear of retaliation, or fear of nursing home placement.
- Historical details that should be obtained in elder abuse include caregiver characteristics, family history of violence, patient isolation, caregiver and elder living together, recent stressful life events, elder characteristics and needs, and symptoms of victimization.

DIAGNOSIS AND DIAGNOSIS

- Indicators of potential elder abuse are that (1) elder is fearful of his or her companion; (2) there are conflicting accounts of the injury; (3) there is an absence of assistance from the caregiver; (4) the caregiver displays an attitude of indifference or anger toward the patient; (5) the caregiver is overly concerned with the costs; and (6) the caregiver opposes a private interaction between the patient and physician.
- The physical examination should note any signs of poor personal hygiene, inappropriate or soiled clothing, dehydration, malnutrition, worsening de-

cubitus ulcers, abrasions, burns, bruises, or sexually transmitted disease.
- Bruises on the upper arms bilaterally are consistent with shaking. Bruises on the inside part of arms and thighs are suggestive of intentional injury.

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Elder abuse should be considered in the differential diagnosis when a patient with frequent falls, dementia, dehydration or malnutrition is being evaluated.
- Intervention to prevent further abuse should involve consultation with social services and adult protective services.
- Admission is based upon the elder's medical problems or in order to protect the patient from the abuser.

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184 SEXUAL ASSAULT

Craig E. Krausz

EPIDEMIOLOGY

- Sexual assault accounts for 5 percent of all violent crimes.[1]
- One in 5 women will be raped during their lifetime,[1] and 12 percent of adolescent women have experienced some form of sexual abuse or assault.[2,3]
- Male sexual assault has a 2 to 4 percent incidence of reported cases.[4,5]

CLINICAL FEATURES

- A history must be obtained the purpose of which is to tactfully obtain data regarding the assault.

Essential historical points include the following: Who? (whether the assailant was known and the number of attackers); What happened? (injuries, penetration, ejaculation, foreign object, condom); When? (time of assault); Where? (vaginal, oral, or rectal penetration); Whether the patient douched, showered, or changed clothing since the attack).[3,6–8]
- The medical history should include the last menstrual period, birth control method used, last consensual intercourse, allergies and prior medical history, and prior sexual assault.[3,6–8]
- The physical examination should note bruises, lacerations, or other signs of trauma. Fifty percent of rape survivors have injuries outside the genital region.[3,6–8]
- Toluidine blue can aid in detecting subtle vulvar lacerations and appears as a linear blue stain.[3]

DIAGNOSIS AND DIFFERENTIAL

- Rape is not a medical diagnosis but a legal determination. It requires 3 elements: any degree of carnal knowledge; nonconsent (unless a minor, intoxicated, or mentally incompetent); compulsion or fear of great harm.[3,4,6–8]
- Informed consent is required prior to evidence collection.[3,7,8]
- Wood's lamp may reveal semen. Saliva, fingernail scrapings, hair samples, and blood samples should be collected. Vaginal swabs should be obtained, along with chlamydia and gonorrhea cultures. If indicated by history, rectal or buccal swabs for sperm should be collected.
- Courts have historically placed a high significance on presence of sperm.[9–11] Two to 3 h is the average time for loss of sperm motility, and nonmotile sperm may persist in vagina and rectum for 24 h. Seminal fluid is destroyed in the mouth within hours.[6,8]
- Additional forensic tests may include acid phosphatase, glycoprotein p30 and genetic typing (ABO antigens, peptidase A, phosphoglucomutase, and DNA).[3,8]

EMERGENCY DEPARTMENT CARE AND DISPOSITION

- Care of the rape victim includes management of any injuries, tetanus prophylaxis, counseling, and pregnancy and sexually transmitted disease prophylaxis.
- Pregnancy prophylaxis must be initiated within 72 h after the assault. Ovral (norgestrel plus tethinyl estradiol) 2 tablets initially and then 2 tablets 12 h later is recommended.[12–14] A negative pregnancy test must be documented prior to pregnancy prophylaxis.
- Sexually transmitted disease prophylaxis should be given for all sexual assault victims using the current Centers for Disease Control guidelines for gonorrhea, chlamydia, and trichomonas.[15] A baseline VDRL should be obtained.
- Counseling, testing, and prophylaxis for hepatitis B and HIV should be performed. The risk of contracting HIV is 0.008 to 0.032 infections per episode in unprotected anal intercourse and is 0.005 to 0.0015 infections per episode in unprotected vaginal intercourse. When prescribing post-exposure prophylaxis, clinicians must consider the likelihood of HIV exposure and the risks and benefits of anti-viral therapy.[16]

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Section 25
IMAGING

185 PRINCIPLES OF EMERGENCY DEPARTMENT USE OF COMPUTED TOMOGRAPHY AND MAGNETIC RESONANCE IMAGING

Craig E. Krausz

COMPUTED TOMOGRAPHY

- Spiral computed tomography (CT), a recent technologic advance, allows for continuous data collection in a spiral fashion.
- Spiral CT greatly decreases errors secondary to movement or breathing.
- The major advantages of spiral CT over conventional scanning are (1) rapid data acquisition, (2) less contrast material needed, (3) images that can be retrospectively reconstructed, (4) reduction in respiratory and cardiac motion artifacts, and (5) ability to produce high-quality three-dimensional and multiplanar reconstructions.
- The major disadvantages of spiral CT are (1) weight limitation (patients may not weigh more than 350 lb), (2) injection of contrast material must be timed precisely, and (3) children and uncooperative adults need sedation.[1]

GENERAL USES AND LIMITATIONS

- CT is the imaging study of choice for the evaluation of intracranial hemorrhage and lesions; intraabdominal pathology including the retroperitoneum; bony fractures of the face, cervical spine, and pelvis; and disorders of the mediastinum.[2]
- Spiral CT has become a primary imaging modality for evaluating appendicitis and ureteral calculi.[3]
- Areas that are poorly imaged with CT include the pituitary fossa and the posterior intracranial fossa. CT is not sensitive in differentiating the spinal cord or nerve roots from cerebrospinal fluid (CSF) unless contrast has been injected into the CSF space.

THE USE OF CONTRAST

- Contrast can be given orally, intravenously, rectally or intrathecally.
- Oral contrast ensures adequate contrast opacification and distention of the bowel, which enhances the appearance of the bowel wall.
- Water-soluble iodinated contrast should be used in trauma patients in order to avoid extravasation of barium agents.
- The administration of oral contrast takes approximately 2 h in a patient with a normal transit time if the entire bowel must be opacified.

MAGNETIC RESONANCE IMAGING

BASIC PRINCIPLES OF MRI

- Magnetic resonance imaging (MRI) has the following advantages over other imaging modalities: (1) it does not use ionized radiation; (2) it produces variable-thickness, two-dimensional slices in any orientation through the body part of inter-

est; and (3) it provides better contrast resolution and tissue discrimination than are achievable with plain radiographs and ultrasound.[4,5]

SAFETY AND CONSIDERATIONS

In a few cases, the large magnetic field can be a health hazard to the patient, necessitating the use of alternative diagnostic methods.

- Internal cardiac pacemakers may be converted to an abnormal asynchronous mode.
- Certain cerebral aneurysm clips may be affected, causing damage to the brain.
- Small steel slivers in the eyes of metal workers may enter the retina and cause damage.
- Life-support equipment may be affected.
- Cochlear implants can be damaged.
- Implantible cardiac defibrillators, neurostimulators, and bone growth stimulators may malfunction.
- The presence of a prosthetic heart valve is a relative contraindication.
- A complete MRI scan can take 30 to 60 min, which requires suspension of all motion.
- Some patients are claustrophobic and have difficulty with the exam.

APPLICATIONS OF MRI

- MRI of the brain and spinal cord provides superior images in diagnostic quality compared to CT.
- MRI has a major role in imaging the musculoskeletal system.[6] However, it is not indicated for acute fractures.
- MRI is preferred in the diagnosis of rotator cuff tears of the shoulder, internal derangement of the knee, tendon or soft tissue injury of the small joints, soft tissue injury of the spine, and posttraumatic avascular necrosis of any bone.
- MRI aids in the evaluation of sequelae of soft tissue musculoskeletal trauma, such as muscle tears, hematomas, and edema.[7,8]
- MRI is extremely sensitive in detecting metastatic disease in bone.

MRI SCANNING IN THE EMERGENT SETTING

- Three areas where MRI scanning is the procedure of choice include evaluation of (1) suspected spinal cord compression, (2) radiographically occult femoral intertrochanteric and neck fractures, and (3) the pituitary fossa and the posterior intracranial fossa.[9]
- Potential future indications for emergent MRI scanning include (1) aortic dissection, where MRI is superior to a contrast CT or transesophageal ultrasound in delineating an intimal flap; (2) evaluation of pulmonary embolism; and (3) pediatric fractures when there may be significant injury to unossified cartilage around open growth plates.

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186 PRINCIPLES OF EMERGENCY DEPARTMENT ULTRASONOGRAPHY

Craig E. Krausz

FUNDAMENTALS

- A perfect reflector of ultrasound waves appears white and is referred to as *hyperechoic.*
- A perfect transmitter of ultrasound waves appears dark and is referred to as *anechoic.*
- Orientation of the ultrasound image is as follows: (1) the skin-transducer interface is at the top of the image and (2) the marker on the transducer always points to the left side of the screen as viewed from the front.

PRIMARY INDICATIONS FOR EMERGENCY DEPARTMENT ULTRASONOGRAPHY

ABDOMINAL AORTIC ANEURYSM

- Ultrasound is as accurate as computed tomography (CT) in measuring the diameter of an abdominal aortic aneurysm.
- An ultrasound examination that images the aorta from the diaphragm to its distal bifurcation is extremely accurate in the evaluation for an abdominal aortic aneurysm. Any diameter greater than 3 cm is abnormal. Transverse images measured horizontally from outside wall to outside wall are the most reliable in accurately determining the true size of the aorta.
- The indications for performing ultrasonography of the aorta in the emergency department (ED) include hypotensive patients or elderly patients with unexplained back, flank, or abdominal pain.

RENAL COLIC

- The renal sinus appears as an echogenic stripe within the kidney and includes the collecting system. The renal cortex occupies the periphery of the kidney and has an echogenicity similar to that of the liver or spleen.
- Obstruction of urine outflow from a calculus will result in hydronephrosis, which appears as an anechoic fluid collection within the renal sinus. Hydronephrosis can be graded from mild, with minimal separation of the sinus echoes, to severe, manifest by extensive separation of the central echoes.
- To evaluate for hydronephrosis, both longitudinal and transverse images should be obtained of both kidneys.
- Renal cysts are thin-walled, round, anechoic structures that are typically located at the periphery of the kidney.
- Ureteral calculi are identified by ultrasound in only 19 percent of patients with documented stones.[1] Hydronephrosis is identified in 73 percent of patients with ureteral calculi. The calculus causing the obstruction most often lodges at the ureterovesicular junction, the ureteropelvic junction, or the pelvic brim.

GALLBLADDER DISEASE

- Ultrasound is the modality of choice in evaluating biliary disease.[2]
- Gallstones appear as bright, echogenic foci within the gallbladder and move with position.
- A sonographic Murphy's sign is positive when the point of maximal tenderness to transducer pressure is directly over the sonographically located gallbladder. A positive sonographic Murphy's sign in the presence of cholelithiasis is reported to have a 92 percent positive predictive value for symptomatic gallbladder disease.
- Gallbladder wall thickening, defined as proximal gallbladder wall thickness greater than 3 mm, occurs in 50 to 75 percent of patients with acute cholecystitis. Other ultrasound findings suggestive of biliary disease include gallbladder sludge and pericholecystic fluid.

FOCUSED ABDOMINAL SONOGRAPHY FOR TRAUMA

- The focused abdominal sonography for trauma (FAST) examination has an accuracy rate similar to that of diagnostic peritoneal lavage (DPL) for the detection of hemoperitoneum. The FAST examination has a sensitivity of 85 to 95 percent and a specificity of 96 to 100 percent; it has replaced DPL in many trauma centers.[3,4]
- The standard views on FAST examination[4] include (1) the subxiphoid view for the evaluation

of pericardial fluid; (2) Morison's pouch, the potential space between the right kidney and the liver; (3) splenorenal recess, the potential space between the left kidney and the spleen; and (4) the pouch of Douglas and rectovesicular space. In addition, the upper abdominal views are capable of evaluating the patient for hemothorax.[5]
- Hemodynamically unstable blunt trauma patients with a positive FAST examination for free intraperitoneal fluid should be taken to the operating room for exploratory laparotomy.
- The advantages of the FAST examination are that it is rapid, portable, accurate, repeatable, noninvasive, and inexpensive.

EVALUATION OF FIRST-TRIMESTER PREGNANCY

- In the ED, ultrasound detection of an intrauterine pregnancy greatly reduces the possibility of ectopic pregnancy. The incidence of heterotopic pregnancy (concurrent intrauterine and ectopic pregnancies) is less than 1 in 30,000.[6]
- When ED patients present with abdominal pain, adnexal mass, and vaginal bleeding, the incidence of ectopic pregnancy is greater than 10 percent.
- The current recommendation is that all first-trimester pregnant patients presenting to the ED with any abdominal or pelvic pain, vaginal bleeding, or risk factors for ectopic pregnancy should have an ultrasound evaluation.
- Pelvic ultrasound by emergency physicians has been shown to decrease the length of stay in the ED.[7]
- The earliest sonographic finding of a pregnancy is the gestational sac. This appears as a round or oval anechoic area within the uterus. True gestational sacs have two concentric echogenic rings surrounding the gestational sac (double decidual sign).
- Endovaginal scanning can detect a gestational sac as early as 4.5 weeks after the last menstrual period (LMP), while transabdominal scanning can detect a gestational sac at 5.5 to 6 weeks after the LMP. An intrauterine pregnancy should be detectable on endovaginal scanning if the β-HCG is greater than 2000 MIU/mL (termed the *discriminatory zone*).[8]
- Patients with a β-HCG greater than the discriminatory zone who do not have evidence of an intrauterine pregnancy on ultrasound are at high risk for an ectopic pregnancy; immediate obstetric consultation is indicated.

CARDIAC ULTRASONOGRAPHY

- The major applications for ED cardiac ultrasonography are in the evaluation of pulseless electrical activity, cardiac trauma, and pericardial tamponade. Key sonographic findings are pericardial fluid collections and myocardial wall activity.
- Pericardial effusions appear as echo-free areas within the pericardial sac. A small pericardial effusion (<100 mL) will occupy a dependent position, while a larger effusion (>300 mL) will present both anteriorly and posteriorly. Sonographic localization of the pericardial sac is the best approach for a pericardiocentesis.

MISCELLANEOUS EMERGENCY DEPARTMENT APPLICATIONS

- Compression ultrasound has been used by emergency physicians to diagnose deep venous thrombosis (DVT) in ED patients.[9] Compression ultrasound has a sensitivity and specificity of 95 percent in venographically proven DVT of the proximal leg.
- Ultrasound may guide the emergency physician in performing thoracentesis for small pleural effusions.
- Ultrasound may assist physicians in identifying small foreign bodies in soft tissue.[10]
- Ultrasound use in the placement of central venous catheters decreases failure rates and complications.[11]

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Section 26
ADMINISTRATION

187 EMERGENCY MEDICAL SERVICES

Lance H. Hoffman

GENERAL CONSIDERATIONS

- The National Highway Safety Act of 1966 authorized the United States Department of Transportation to fund ambulances, communications, and training programs for prehospital medical services.[1]
- On-line medical control is the direct medical communication of personnel from the hospital to the field personnel.
- Off-line medical control allows field personnel to function independently through the use of treatment protocols, quality assurance, and continuing education.
- Challenges faced by rural emergency medical systems include long distances, search and rescue, and the diminished likelihood of system activation secondary to the emergency inciting event not being witnessed.[2]

AIR MEDICAL TRANSPORT

- Air medical transport is warranted when patient care is dependent on time and distance considerations. Traumatic cardiac arrest does not warrant air medical transport since its use does not improve survival of these patients.[3]
- Advantages of air medical transport include faster transport (e.g., 125 to 175 mi/h), a lack of consideration for traffic or road conditions, and allowing local, and otherwise busy, emergency medical services to remain operational.
- Disadvantages of air medical transport include increases in weather sensitivity, expense, maintenance, continuing education for the crew and difficulty with in-flight patient assessment.

NEONATAL AND PEDIATRIC TRANSPORT

- Pediatric cases consist of 5 to 10 percent of an emergency medical system's volume, with trauma, respiratory emergencies, and seizures the most common complaints.[4]
- The ambient temperature has a profound effect on neonates and small children secondary to a large surface-to-body mass ratio, increased water vapor skin permeability, and a paucity of subcutaneous tissue.

DISASTER MEDICAL SERVICES

- The World Health Organization defines a disaster as a sudden ecological phenomenon of sufficient magnitude to require external assistance.[5]
- An external disaster is an event that occurs physically outside of the hospital. An internal disaster is an event that occurs physically within the hospital.[6] Both may coexist as in the case of a tornado that damages a hospital and the surrounding area.
- The Joint Commission on the Accreditation of Healthcare Organizations (JCAHO) requires that hospitals have a prearranged disaster plan and documentation of plan rehearsal twice yearly.[7]

JCAHO also requires provisions for the emergency treatment and decontamination of radioactively or chemically contaminated patients.[8,9]
- Key elements of a hospital's disaster plan include activation, assessment of hospital capacity, establishing a command center, communications, supplies, administrative and treatment areas, and training and drills.

TRIAGE

- Triage is the prioritization of care based on injury or illness severity, prognosis, and resource availability. Triage care should only consist of manual airway management and external hemorrhage control.
- Patients designated as ''red'' are given first priority for definitive treatment. These patients have life-threatening shock or hypoxia, but survival is likely with immediate care.
- Patients designated as ''yellow'' are given second priority for definitive treatment. These patients have systemic manifestations of their injuries, but will likely endure a 45 to 60 min delay to definitive treatment.
- Patients designated as ''green'' are given third priority for definitive treatment. These patients have only localized injuries that can wait several hours before receiving definitive care.
- Patients designated as ''black'' are considered dead in that their injuries are so severe that they have a poor chance of survival regardless of the level of care provided.

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188 EMERGENCY MEDICINE ADMINISTRATION

David M. Cline

NEGLIGENCE AND MEDICAL MALPRACTICE

- Negligence is defined as the failure to do something that a reasonable person, guided by those ordinary considerations that normally regulate human affairs, would do, or the doing of something that a reasonable and prudent person would not do.[1]
- The four components of negligence are duty, breach of duty, damages, and causation. The plaintiff (injured or complaining party) must prove that all four elements existed in order to find the defendant guilty of negligence.[2]
- Duty is considered a contract created by formation of a physician–patient relationship whereby the physician must act in accordance with ''standards of care'' to protect the patient from unreasonable risk.[2] In general, by contract with the hospital, emergency physicians (EPs) have a duty to see all

patients who present themselves to the emergency department to be seen.
- The standard of care is that which a similarly trained ''reasonable and prudent physician'' would exercise under similar circumstances.[2] The emergency physician is not required to exercise the highest degree of skill and care possible but must use the degree of skill and care ordinarily exercised by physicians within the same specialty.
- Breach of duty occurs if the physician with an established duty fails to act in accordance with these standards of care by commission or omission of a certain act.[2] Emergency physicians are held to a national standard of care for a specialist in emergency medicine.
- Damages encompass any actual loss, injury, or deterioration sustained by the plaintiff due to the breach of duty.[2] A plaintiff must prove that the damage occurred because of the physician's negligence.
- Legal causation theoretically consists of two branches: causation in fact and foreseeability.[2] Causation in fact means that ''an event A is the cause of another event B, if and only if B would not have occurred when and as it did but for event A.'' The concept of foreseeability is fulfilled if the patient's damages must be the foreseeable result of the defendant's substandard practice as compared with the standard of the reasonable physician. A bad result without proof of violation of the standard of care does not constitute negligence.

CONSENT

- Informed consent is considered ideal—the patient knows and understands the risks, benefits and consequences of accepting or refusing treatment.[3] Specific informed consent should be sought and obtained by the emergency physician whenever an invasive, risky, or complicated treatment or procedure is proposed. Examples include nonemergent thoracentesis, tube thoracostomies, paracentesis, and incision and drainage of a complex abscess.[4]
- Elements of informed consent include the following: (1) a concise statement of the patient's medical condition or problem; (2) an understandable statement of the nature and purpose of the proposed test, treatment, or procedure; (3) a description of the risks, consequences, and benefits of the proposed test, treatment, or procedure; (4) a statement regarding any viable alternatives to the test, treatment, or procedure; and (5) a statement regarding the patient's prognosis if the proposed test, treatment, or procedure is not given.[4]
- Express consent entails an awareness of the proposed care and an overt agreement (e.g., in oral or written form) to proceed. An example would be the patient who comes to the emergency department, requests assistance for a problem, and signs a registration form authorizing evaluation and treatment of the problem.[4]
- Implied consent is invoked if an emergency exists and the patient is incompetent (e.g., a minor or someone with an altered mental status). Simple procedures such as minor wound suturing, phlebotomy, injections, and peripheral IVs are allowed under express or implied consent.[4] An exception to this is testing for human immunodeficiency virus (HIV), which requires written informed consent.[5]
- Emergency consent bypasses normal consent standards due to the rapid need to treat a clinically ill patient. Implied consent is inferred by the patient's actions but without specific agreement. Emergency consent covers actions such as emergent intubation or placement of central lines in a critical patient when there is no other access.[4]
- Failure to obtain appropriate consent can leave the emergency physician vulnerable to a legal action based on battery (intentional, unauthorized touching).[4]

MINORS AND CONSENT

- The law always implies consent for treatment of a child in the event of an emergency. Parental consent is not needed; it is implied.[6]
- All states without a general consent statute for minors have provisions that specifically permit the physician to treat any minor for venereal disease.[6]
- Most states have treatment statutes for minors (usually 16 years or older) that enable them to consent for medical care. Many states also specifically permit treatment of minors for drug or alcohol problems, pregnancy, and psychiatric conditions.[6]
- ''Mature minor'' statutes vary from state to state but allow a minor (usually between 14 to 18 years of age) to give informed consent when he or she understands the risks and benefits of a treatment. This generally applies to treatments that do not pose a serious risk.[6]
- A parent with sole custody of a child has the legal right to provide consent for medical treatment. This permission should be obtained prior to treat-

ment whenever possible. On a practical basis, however, if a medical necessity exists and a delay could be deleterious, the EP may need to assume that a parent in possession of a child has the authority to provide consent.[6]

REFUSAL OF CONSENT AND PATIENTS LEAVING AGAINST MEDICAL ADVICE

- On general principle, adult patients may ethically and legally refuse treatment totally or in part.[7]
- A patient need not have a global decision-making ability to refuse treatment but rather enough for a given situation—that is, a relative decision-making capacity. Clinical circumstances require the use of the term *capacity,* whereas *competence* is a legal term, which can only be determined by a court ruling.
- Multiple components are required for a decision-making capacity. These include understanding the options, awareness of the consequences of each option, and appreciation of the costs and benefits of the options in relation to relatively stable values and preferences.[8,9]
- Informed refusal should be carefully documented on the chart of a patient who leaves against medical advice (AMA).[10] The following five issues can be problematic and should be addressed in the chart:
  1. Capacity: Document the patient's mental status. Ideally, a patient should be awake and alert, able to carry on a reasonable conversation, and should possess the mental ability to discuss the problem and act in his or her own interest.
  2. Discussion: Use and document clear terms that a layperson can understand; avoid euphemisms and technical jargon. If death is a possibility, say so.
  3. Offer of alternative treatment: Document whether alternative treatments are available and are offered.
  4. Family involvement: Document efforts to involve family or friends in the decision process. If the patient forbids family involvement, document this accordingly.
  5. Patient's signature: The physician is not legally protected if the patient signs a standard AMA form devoid of the other four elements. However, if a patient refuses to sign after an appropriate informed discussion, simply document the refusal to sign.

RESUSCITATION AND ''DO NOT RESUSCITATE'' ORDERS

- Current standards suggest that when the possibility exists that the brain is viable and there are no compelling medical or legal reasons to act otherwise, resuscitation should be initiated.[11]
- The current medical standard used to terminate resuscitations should be brain death or cardiovascular unresponsiveness. This principle is well founded in the standard references and well supported ethically.[12]
- Medically and ethically, it is important to remember that there is no obligation to deliver treatment that is futile.[13] When a person with a terminal illness is expected to die within a few hours or days, further aggressive diagnostic or therapeutic care would not benefit the patient and would be considered medically futile (and thus an ethical reason to withhold or cease resuscitation).[14]
- It is prudent to stabilize the patient first and then seek further clarification of his wishes, either from the patient directly or with the family or physician. Appropriate, ethical reasons to withhold or cease resuscitation include irreversible cessation of cardiac function, brain death, competent patient refusal, or an advance directive such as ''Do not resuscitate'' (DNR).[15]
- Even with a valid DNR order, conditions such as pain, infection, dehydration, and respiratory difficulty should be addressed. A patient with a DNR deserves respectful and compassionate care, which can maximize comfort and possibly improve the remaining quality of life.[15]

PHYSICIAN TELEPHONE ADVICE

- Even brief, seemingly straightforward advice is potentially a high-risk action when given over the telephone. A legally binding relationship (duty—the first element of a negligence tort) is established once advice is given.[10] Since one cannot see the patient and further information may not be forthcoming, an accurate assessment truly cannot be made.[10]
- It is acceptable, however, to give basic first aid advice if one includes a rejoinder to come immediately to the emergency department.[10]
- Medical facilities with formal telephone advice programs should use specific guidelines, track outcomes, provide close follow-up, and complete the calls with a patient reminder to come to the emergency department.[10]

COMPREHENSIVE OMNIBUS BUDGET RECONCILIATION ACT (COBRA)

- In 1986 Congress enacted the Comprehensive Omnibus Budget Reconciliation Act (COBRA) to combat widespread patient-dumping practices. The Emergency Medical Treatment and Active Labor Act (EMTALA) is the section of COBRA that applies to emergency departments.[16,17]
- According to COBRA regulations, a medically unstable patient can be transferred to another facility only if the transferring physician certifies that the transfer is medically necessary and the receiving facility agrees to accept the patient.[17]
- A patient with an illness or injury who presents to an emergency department (whose hospital has a Medicare contract) must receive a medical screening examination regardless of the ability to pay or of insurance coverage.[17]
- Next, the patient must be stabilized prior to transfer to another facility.[17]
- The patient must understand the risks and benefits and sign informed consent for the transfer.[17]

MEDICAL ETHICS

- There are five basic principles that should guide ethical decision making in medical practice.[18,19]
- Veracity is telling the truth. It forms the basis of maintaining an open health care provider–patient relationship and of keeping promises.
- Patient autonomy is based upon a person's right and freedom to make an informed choice about what will and will not be done; it also acknowledges the patient's right to privacy.
- Beneficence is the principle of doing good; it involves promoting the well-being of others and responding to those in need.
- Nonmaleficence is the principle of ''do no harm,'' which obliges the physician (or other health care provider) to protect others from danger, pain, and suffering. This concept stems from the Hippocratic oath as well as from other ancient medical traditions.
- Justice involves fairness, respect for human equality, and the equitable allocation of scarce resources.

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