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DEJA REVIEW™

Emergency Medicine

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To the love of my life Min Young for her support and patience with all things in life; the residents and attendings at The University of Pittsburgh Affiliated Residency in Emergency Medicine, who all have been excellent and continue to push me to better help others.
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## CHAPTER 1

# Introduction to Emergency Medicine

## EMERGENCY MEDICAL SERVICE

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What was the significance of the Highway Safety Act of 1966 to the development of Emergency Medical Service (EMS) in the United States?</td>
<td>This act authorized the Department of Transportation to provide funding for improvement of ambulance service and prehospital provider training, as well as the development of highway safety programs and EMS standards.</td>
</tr>
<tr>
<td>What important advancements in EMS occurred in First National Conference on EMS in 1969?</td>
<td>Development of a curriculum, certification process, and national register for the emergency medical technician (EMT)—ambulance.</td>
</tr>
<tr>
<td>In what year was EMT recognized as an occupational specialty by the Department of Labor?</td>
<td>1972</td>
</tr>
</tbody>
</table>
| What are the 15 elements of an EMS system as defined by the Emergency Medical Services Act of 1973? | 1. Personnel  
2. Training  
3. Communications  
4. Transportation  
5. Facilities  
6. Critical care units  
7. Public safety agencies  
8. Consumer participation  
9. Access to care  
10. Transfer to care |
11. Standardization of patient’s records
12. Public information and education
13. Independent review and evaluation
14. Disaster linkage
15. Mutual aid agreement

What are five types of EMS service systems?
1. Public Service (often provided by the fire department)
2. Hospital-based
3. “Third Service” model, usually a separate division of the local government
4. “Public Utility” model (a private ambulance company)
5. Volunteer model

What determines which service system is appropriate for a given community?
The type of EMS system depends on the needs and the resources of the community

What are the two general categories of care provided by EMS systems?
1. Basic life support (BLS)
2. Advanced cardiac life support (ACLS)

What are the three main methods of patient transport?
1. Ground transportation—ambulance
2. Rotary-wing transportation—helicopter
3. Fixed Wing transportation

What is the average cost of an ambulance transport?
$180–$600

Can a patient refuse EMS treatment and/or transport?
Yes. A competent, conscious adult patient may refuse treatment or transport, but he/she must be informed of risks when refusing

What are the four levels of EMS training and some specific skill sets?
First responder. Requires approximately 60 classroom hours of training: Initial scene and patient evaluation; Cardiopulmonary resuscitation (CPR); Basic airway skills; Hemorrhage control; Spinal immobilization
EMT—Basic. Requires 100 classroom hours as well as 10 clinical hours: Skills of the first responder; Triage and patient assessment; AED use; Assist patient in taking medications
EMT—Intermediate. Requires 300–400 hours of classroom and clinical training: Advanced patient assessment; Intravenous line placement; Manual defibrillation; Administration of a limited number of medications

EMT—Paramedic. Requires 1000–1200 hours of classroom training plus a clinical internship: Electrocardiogram (ECG) interpretation; Needle decompression of a tension pneumothorax; Needle and surgical cricothrotomy; Transthoracic cardiac pacing; Administration of selected medications

What advancement in communication significantly improved the public’s access to emergency medical services?
Answering, triaging, and prioritizing all calls; Alerting and dispatching the appropriate unit; Providing pre-arrival instructions

What are the responsibilities of the Emergency Medical Dispatcher?

Define unique characteristics of an EMS medical director.
The EMS medical director is a physician with a special interest in and knowledge of the patient care needs in the prehospital environment

What is the definition of a disaster?
A disaster is an incident that overwhelms the response capacities of a community. This occurs when the number or severity of patients presenting to the emergency medical response system in a given period of time overwhelms the available resources

What are the three phases of a disaster plan?
Activation. This includes the initial response by EMS and the organization of an incident command center

Implementation. Components include search and rescue, triage, and transport of patients

Recovery.

What are the two phases of a disaster operation?
1. Prehospital
2. Hospital
List the components of the prehospital phase of a disaster operation. Triage of patients; Scene control; Communications; Public health considerations

The second phase of a disaster operation focuses on hospital preparedness. List the six components that must be considered during this phase.

1. Development of a central control center
2. Activation of the disaster plan
3. Designation of treatment areas
4. Organization of documentation
5. Mobilization of security
6. Designation of waiting areas

What is the definition of triage?

Triage is the process of classifying injured patients into groups according to the priority for treatment. The goal is to do the most good for the largest number of potential survivors

Describe the four patient triage categories. Dead or unsalvageable.

Critical. These patients are severely injured but salvageable and require immediate medical attention

Serious. These patients have no immediate life-threatening injuries

Minor. Often these patients are referred to as the walking wounded

AIRWAY MANAGEMENT

<table>
<thead>
<tr>
<th>Sedative/Induction</th>
<th>Dosage</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etomidate</td>
<td>0.3–0.4 mg/kg</td>
<td>1 minute</td>
<td>3–6 minutes</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5–2 ug/kg</td>
<td>1 minute</td>
<td>30–60 minutes</td>
</tr>
<tr>
<td>Ketamine</td>
<td>1–2 mg/kg</td>
<td>1 minute</td>
<td>15 minutes</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.2–0.4 mg/kg</td>
<td>30 seconds</td>
<td>15–20 minutes</td>
</tr>
<tr>
<td>Propofol</td>
<td>1–2 mg/kg</td>
<td>30 seconds</td>
<td>3–5 minutes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neuromuscular Blocking Agents</th>
<th>Dosage</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>1–2 mg/kg IV</td>
<td>&lt;1 minute</td>
<td>3–5 minutes</td>
</tr>
<tr>
<td></td>
<td>2–4 mg/kg IM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.6–1.2 mg/kg</td>
<td>&lt;1 minute</td>
<td>&gt;30 minutes</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.1–0.2 mg/kg</td>
<td>2–3 minutes</td>
<td>30–60 minutes</td>
</tr>
</tbody>
</table>

IV, intravenous; IM, intramuscular.
What are some reasons a patient may need oxygenation; prevention/overcoming airway management? 

Oxygenation; Prevention/overcoming airway obstruction; Protection against aspiration; Assisted ventilation

What are some important things to do prior to any airway procedure (assuming patient is not in imminent danger)?

Inspect the patient’s mouth (teeth, palate, tongue, and oropharynx); Ask patient history if possible; Assess for possible cervical neck injury; Listen for any airway problems (i.e., stridor); Suction any secretions prior to procedures

List some important causes of airway obstruction.

Foreign bodies; Trauma (expanding hematoma); Infections (epiglottis); Congenital (enlarged airway)

What is the most feared complication of inability to secure an airway in a timely fashion?

Hypoxia; Brain damage

What are some important points for each of the following airway devices:

Oral airway

C-shaped rigid instrument placed into the mouth; Placed to create a patent airway; Used to prevent tongue from falling posterior; Used in patients with no gag reflex

Nasal airway

A nasopharyngeal tube placed into a nostril; Typically used to bypass obstructing tongue; Used in somnolent patient with a gag reflex

Bag-valve-mask (BVM)

The mainstay of airway management; Inflating bag with a nonrebreathing valve; Critical to airway management; Two-person use is optimal to avoid air leak

Esophageal tracheal combitube (ETC)

Plastic twin-lumen tube with inflatable cuffs; Placed blindly into the oropharynx; Commonly used as a backup airway to ETT; Typically used in the prehospital setting

Laryngeal mask airway (LMA)

Tubular oropharyngeal airway; Contains a distal laryngeal mask; Inserted blindly into the oropharynx; Commonly used as backup airway to ETT

Endotracheal tube (ETT)

Cuffed-tube placed into the larynx; Placed normally by direct laryngoscopy; Considered the gold standard
What is rapid sequence intubation (RSI)? The use of special drugs to rapidly sedate and paralyze a patient to allow ETT placement.

What is the primary reason for RSI? It allows optimal conditions to secure an airway.

What is a disadvantage of RSI? If unable to intubate, it can result in complete loss of airway control.

What is the most important thing to keep in mind while performing RSI? Always have a back-up airway ready! If ETT fails, the patient will not be able to breathe (paralytics still in effect).

What are the seven P’s of RSI? Prepare. Have different sized tubes and blades ready; Ensure cuff works; Have back-up airway ready (i.e., LMA). Preoxygenate. Preoxygenate for about 2—5 minutes; Hypoxia develops faster in children and pregnant women. Position. Flexion of lower neck; Extension at the atlantooccipital joint; This allows direct visualization of the larynx; Bad positioning common reason for failure. Premedicate (induction). Induce a deep level of unconsciousness; Agent depends on situation; Always premedicate prior to paralyzation. Paralyze. Administer neuromuscular blocking agent; Succinylcholine most preferred agent. Place ETT. Visualization of vocal cords is critical. Placement confirmation. Look for tube condensation; Bilateral chest rise; Auscultate stomach and lung; Capnography.

What is the Sellick’s maneuver? The application of cricoid pressure to help prevent aspiration as well as aid in direct visualization of vocal cords.

What are some reasons that succinylcholine is used most often in RSI? Rapid onset of action (<45 seconds); Brief duration of action (<7 minutes).

What are some adverse side effects to keep in mind about the use of succinylcholine? Increases intraocular/intracranial pressure; Avoid in hyperkalemic states (i.e., burns); In rare cases can cause malignant hyperthermia.
List some alternative paralytics that can be used if succinylcholine is contraindicated. Rocuronium (fast onset, but longer duration of action); Vecuronium (even longer duration of action)

What are some important points for the following alternative methods:

Cricothyrotomy

The primary surgical backup airway; Placement of trach/ETT through surgical incision in neck and cricoid membrane; Contraindicated in children < 8

Tracheotomy

Longer to perform then cricothyrotomy; Preferred in children; Also used in patients with tracheal injury

Digital intubation

Index/middle fingers to palpate epiglottis; Typically used in comatose patients; Success rate is lower then that of RSI

Retrograde intubation

Use of a guide wire via the cricoid; The guide wire guides the tube via the cord; Not commonly used in prehospital setting

**SHOCK**

What is the definition of shock? It is a clinical syndrome that is characterized by the body’s inability to meet the demands of tissue/organ perfusion resulting in decreased venous oxygen content and lactic acidosis

What is the initial step in management for any patient who presents with possible shock? Airway  
Breathing  
Circulation

What are four categories of shock? 1. Cardiogenic  
2. Hypovolemic  
3. Distributive  
4. Obstructive

What are some of the autonomic responses that occur with shock? Increase in heart rate (HR) and contractility of heart; Constriction of venous capacity vessels; Arteriolar vasoconstriction; Release of vasoactive hormones; Activation of renin-angiotensin system
What are some important vasoactive hormones that are released during a state of shock?
Dopamine; Norepinephrine; Epinephrine; Cortisol

What are two critical organs that the autonomic system tries to preserve blood flow to?
1. Brain
2. Heart

What are some common metabolic derangements that occur with shock?
Metabolic acidosis (lactic acidosis); Hyponatremia; Hyperkalemia; Prerenal azotemia

What are some important elements from the history that should be considered?
Medications (i.e., anaphylaxis); History of heart disease; History of volume depletion (i.e., emesis); Neurologic disease

What is an important way to assess shock as well as evaluate therapeutic intervention?
Hemodynamic monitoring

What are some components of hemodynamic monitoring?
ECG; Pulse OX; Central venous pressure (CVP)

Name some important early interventions to consider in a state of shock.
Airway control (intubate if necessary); Mechanical ventilation (decreases work of breathing); Aggressive fluid resuscitation; Ensure oxygen delivery (pressors if needed)

What is an important distinction to make in hypovolemic shock?
Hemorrhagic versus nonhemorrhagic

What are some important causes of nonhemorrhagic hypovolemia?
Burns; Gastrointestinal (GI) related such as emesis and diarrhea; Excessive urination (renal salt wasting)

What is the normal circulating volume of blood in a normal adult?
5 L

What portion of that is plasma and what is RBC?
3 L of plasma; 2 L of RBC

What is the hallmark response for each of the following categories of hemorrhage:

Class I (about 750 mL)
Usually no response in a healthy person

Class II (750–1500 mL)
Tachycardia and narrowed pulse pressure; Mild hypotension; Mild change in mental status

Class III (>1500 mL)
Tachycardia and pronounced hypotension; Decline in mental status; Peripheral hypoperfusion
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class IV (&gt;2 L)</td>
<td>Hemodynamic decompensation is common; Aggressive resuscitation is required</td>
</tr>
<tr>
<td>What is important to know about children and athletes who have acute hemorrhage?</td>
<td>They compensate very well (no tachycardia or hypotension), but can decompensate very fast soon after, without any warnings</td>
</tr>
<tr>
<td>What other etiologies of hypotension besides hemorrhage should be considered in the setting of trauma?</td>
<td>Myocardial infarction; Tension pneumothorax; Cardiac tamponade; Toxicologic involvement</td>
</tr>
<tr>
<td>What is the initial step in management that should be undertaken with acute hemorrhage?</td>
<td>Airway (ensure patent airway); Breathing (proper ventilation); Circulation (two large-bore IVs for fluids)</td>
</tr>
<tr>
<td>Why are two large-bore IVs more effective than long narrow IVs?</td>
<td>Infusion rate of fluids is much faster through short wide tubes</td>
</tr>
<tr>
<td>What are commonly used large-bore IVs?</td>
<td>13- or 14-gauge needles</td>
</tr>
<tr>
<td>What are some commonly used resuscitation fluids?</td>
<td>Isotonic crystalloids; Colloids; Blood</td>
</tr>
<tr>
<td>Name two commonly used isotonic crystalloid fluids used for resuscitation.</td>
<td>1. Normal saline (NS) 2. Lactated ringers (LR)</td>
</tr>
<tr>
<td>What are some concerns when large amounts of isotonic crystalloid fluids are used?</td>
<td>Increased neutrophil activation; LR may cause lactic acidosis; NS may cause hyperchloremic acidosis</td>
</tr>
<tr>
<td>What are the general guidelines for the infusion of blood?</td>
<td>Minimal response to 2–3 L of fluids; Obvious major loss of blood; Hematocrit of &lt; 16</td>
</tr>
<tr>
<td>What are some concerns whenever blood is given?</td>
<td>Transfusion reaction; Availability; Infections; Limited storage life</td>
</tr>
<tr>
<td>What are some types of blood given?</td>
<td>Whole blood; Packed red blood cells (PRBC); Fresh-frozen plasma (FFP); Platelets</td>
</tr>
<tr>
<td>What is a concern if too much fluid is given during a resuscitation?</td>
<td>Dilutional coagulopathy</td>
</tr>
<tr>
<td>What are some important things to know about sepsis?</td>
<td>50% mortality of those who develop shock; Gram -/+ often common cause of sepsis; Sepsis is more common in older adults</td>
</tr>
<tr>
<td>What are the most frequent sites of infection that can lead to sepsis?</td>
<td>Genitourinary tract; Abdomen; Lung</td>
</tr>
<tr>
<td>What are co-morbid conditions that can predispose one to sepsis?</td>
<td>Burns; Diabetes mellitus; Immunosuppressive agents</td>
</tr>
</tbody>
</table>
What is the definition of bacteremia? Presence of bacteria in the blood

Name the criteria of systemic inflammatory response syndrome (two or more must be met).
- Temperature: <36°C or >38°C;
- Tachycardia: >90 beats/min;
- Tachypnea: >20 breaths/min; WBC: >12000, <4000 or >10% bands

What is sepsis? Systemic response to infection that meet the criteria for systemic inflammatory response syndrome (SIRS)

What is septic shock? Hypotension with inadequate organ perfusion induced by sepsis with another metabolic dysfunction such as lactic acidosis

What are some possible clinical features of sepsis in the following organ system:

**Respiratory**
Adult respiratory distress system (ARDS); Pneumonia

**Cardiovascular**
Myocardial depression and tachycardia; Poor response to fluid administration

**Renal**
Acute renal failure due to renal ischemia; Oliguria

**Hepatic**
Cholestatic jaundice; Elevated liver function tests (LFTs); Elevated bilirubin

**Endocrine**
Hyperglycemia is common; Elevated cortisol and glucagon; Insulin resistance and decreased insulin

**Hematology**
Neutrophilia or neutropenia; Thrombocytopenia; Disseminated intravascular coagulation (DIC)

What are some key points in the management of sepsis?
- ABCs (aggressive fluid resuscitation);
- Not atypical for patients to require >6 L; Inotropes (i.e., DA) if not responsive to fluids; Empiric Abx is the cornerstone; Remove the source of infection

What is the definition of cardiogenic shock? Inadequate tissue perfusion due to decrease in cardiac output despite adequate circulating volume

What is the most common cause of cardiogenic shock? Myocardial infarction
What are some other causes of cardiogenic shock to be considered? Mechanical obstruction; Right ventricular infarct; Sepsis; Myocarditis

What are some clinical features of cardiogenic shock? Evidence of volume overload (i.e., rales), hypotension, mental status change, cool/clammy skin, diaphoresis, and jugular venous distension (JVD)

What are some important points for each of the diagnostic tests commonly used to evaluate cardiogenic shock:

**ECG**
Cornerstone test to diagnose ischemia; Can also detect arrhythmias, drug toxicity, and electrolyte derangements; Also to detect right ventricular infarct

**Chest X-ray (CXR)**
Commonly show pulmonary edema/effusion; R/O other disease states such as a dissection; Normal chest does not rule out shock

**Echocardiography**
Used to assess left-ventricular (LV) function; Color flow Doppler can assess mechanical cause such as valvular disease; Not typically used in the emergent setting

What are some laboratory tests to consider in cardiogenic shock? Cardiac enzyme; Brain natriuretic peptide (BNP); Arterial blood gas; Serum lactate

What is the definition of anaphylaxis? Severe hypersensitivity reaction with multisystem involvement that commonly include airway compromise and hypotension

What is a hypersensitivity reaction? Inappropriate immune response to an antigen

What is an anaphylactoid reaction? Reaction that presents similar to anaphylaxis, but is not IgE mediated and does not require prior sensitization

List some common causes of anaphylactoid reactions. Radiocontrast dye; Opiates; Muscular depolarizing agents

What are the top three causes of serious anaphylactic reactions? 1. Medication 2. Foods 3. Insects

What are the most common foods associated with serious allergic reactions? Nuts; Milk; Shellfish
What is the most common drug implicated in serious allergic reactions? Penicillin

What is the recurrence rate of anaphylaxis for penicillin upon re-exposure? Less than 25%

What is the cross-reactivity of penicillin allergy to cephalosporin? Less than 10%

What is the pathophysiology of anaphylaxis? Mast cell and basophil degranulation due to IgE cross-linking, direct activation, and complement activation

What are some clinical features of anaphylaxis? Diffuse urticaria, rhinorrhea, conjunctivitis, nausea, angioedema, airway compromise such as stridor, and hypotension

What is a general indicator of the severity of a anaphylactic reaction? Faster the onset of symptoms, typically more severe

How is anaphylaxis diagnosed? Clinically—special attention to airway and blood pressure

What is the mainstay in the treatment of suspected anaphylaxis? Epinephrine

What are some key points in the management of anaphylaxis? ABCs; Oxygen, IV fluids, and epinephrine; Decontamination

What are some commonly used agents for general allergic reactions? Antihistamines (e.g., diphenhydramine and ranitidine); Corticosteroids (e.g., methylprednisolone); Asthma medications (e.g., albuterol)

FLUIDS

What percent of the total body weight is comprised of water? 60%

Of the total body water, what percent makes up the intracellular compartment? 2/3

What makes up the extracellular compartment? Interstitial fluid and plasma

Define the following terms in regards to water regulation:

Osmosis Net movement of water across a selectively permeable membrane driven by a difference in solute concentrations on the two sides of the membrane
### Osmolality
- **Semipermeable membrane**: Allows passage of the solvent, but not solute such as cell membranes.

#### What is the normal serum osmolality?
- 280–295 mosm/L

#### Name some important solutes that contribute to serum osmolality?
- Chloride, sodium, bicarbonate, and glucose

#### What is the equation used to calculate the serum osmolality?
\[
2 [\text{Na}^+] + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8}
\]

#### What is the osmolal gap?
- Difference between the measured and calculated osmolality

#### List some causes of hyperosmolality.
- Uremia; Increase in serum sodium (no gap); Alcohol ingestion (methanol and ethylene glycol will cause an increase in osmole gap and anion gap acidosis); Ketoacidosis (small gap)

#### List a cause of hypo-osmolality.
- Decrease in serum sodium

#### How much water does an average human adult require each day?
- 2–4 L

#### What are the two categories of water loss?
1. Urinary loss (1–2 L/day)
2. Insensible loss (i.e., feces and skin)

#### Name two mechanisms by which the human body handles water?
1. Aldosterone
2. Antidiuretic hormone (ADH)

#### What are some things to know about ADH?
- Regulates serum osmolality; Acute volume depletion stimulates ADH; Increased serum osmolality stimulates ADH

#### Does aldosterone play a significant role in maintaining serum osmolality?
- No

### ELECTROLYTES

#### Hyponatremia
- **What is the serum sodium level in hyponatremia?** \([\text{Na}^+] <135 \text{ mEq/L}\)
- **What is the serum sodium level in severe hyponatremia?** \([\text{Na}^+] <120 \text{ mEq/L}\)
- **What are some clinical features of hyponatremia?** Headaches (HA), confusion, and seizures, but can be asymptomatic
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the most feared complication of Cerebral edema?</td>
<td>1. Syndrome of inappropriate ADH secretion (SIADH) 2. Decrease in effective circulating volume</td>
</tr>
<tr>
<td>Name the two causes of hyponatremia?</td>
<td>ADH</td>
</tr>
<tr>
<td>What is the primary hormone that regulates free water in the body?</td>
<td>Increase in osmolality; Decrease in circulating volume</td>
</tr>
<tr>
<td>What are some triggers that result in increased secretion of ADH?</td>
<td>Baro receptors in the carotid sinus</td>
</tr>
<tr>
<td>Name an area of the body that mediates ADH release in response to circulating volume?</td>
<td>True volume depletion (GI bleeding); Exercise-associated hyponatremia; Heart failure; Cirrhosis; Thiazide diuretics</td>
</tr>
<tr>
<td>Name some conditions that can result in a decrease in effective circulating volume that result in hyponatremia.</td>
<td>The carotid sinus will sense a reduced pressure from fall in cardiac output and increase ADH release</td>
</tr>
<tr>
<td>What is the mechanism by which hyponatremia occurs in patients with congestive heart failure (CHF) even though they may have a marked increase in plasma volume?</td>
<td>Peripheral vasodilation in cirrhosis that will result in decreased return of venous blood with a resultant drop in cardiac output</td>
</tr>
<tr>
<td>What is the mechanism by which hyponatremia occur in patients with cirrhosis?</td>
<td>Lung cancer; Drugs; Infections (i.e., brain abscess); Traumatic brain injury</td>
</tr>
<tr>
<td>What are some conditions that may be associated with SIADH?</td>
<td>1. Adrenal insufficiency 2. Hypothyroidism</td>
</tr>
<tr>
<td>Name two other conditions that hyponatremia can also occur in?</td>
<td>1. Primary polydipsia 2. Advanced renal failure</td>
</tr>
<tr>
<td>What are two disorders in which hyponatremia can occur despite normal/low ADH levels?</td>
<td>1. Primary polydipsia 2. Advanced renal failure</td>
</tr>
<tr>
<td>Name two causes of primary polydipsia?</td>
<td>1. Psychogenic 2. Hypothalmic lesions</td>
</tr>
<tr>
<td>Name a cause of hyponatremia with a high plasma osmolality.</td>
<td>Hyperglycemia</td>
</tr>
</tbody>
</table>
What are some elements to keep in mind in the history and physical of a patient with hyponatremia?

- History of fluid loss (i.e., diarrhea);
- Signs of edema (i.e., CHF or cirrhosis);
- Signs/symptoms suggestive of adrenal insufficiency or hypothyroidism; History that may point to SIADH such as small cell carcinoma.

What are three important laboratory tests to consider in differentiating hyponatremia?

1. Urine osmolality
2. Plasma osmolality
3. Urine sodium concentration

What is the plasma osmolality in most hyponatremic patients?

Reduced (<275–290)

What is the primary use of urine sodium concentration in elevating hyponatremia?

Helps to distinguish between effective volume depletion and other causes

Name one condition where a person may be hyponatremic, but have an elevated plasma osmolality?

Hyperglycemia

What does a urine osmolality of greater than 100 mosmol/kg typically indicate in patients with hyponatremia?

Inability to excrete free water (i.e., SIADH)

What does a urine osmolality of less than 100 mosm/kg typically indicate in patients with hyponatremia?

Primary polydipsia; Malnutrition

What are four things to consider when managing patients with hyponatremia?

1. Assessing risk of osmotic demyelination
2. Appropriate rate of correcting hyponatremia to avoid demyelination
3. Determine the best method to raise [Na⁺]
4. Estimate the sodium deficit if giving sodium

What can lead to the development of central pontine myelinolysis?

Rapid correction of severe hyponatremia

What are some clinical features of central pontine myelinolysis?

Dysphagia, dysarthria, quadriplegia, lethargy, coma, and possible death

At what rate should hyponatremia be corrected each day?

No more than 10 mEq/L per day
What are some indications for aggressive treatment of hyponatremia?  
Acute hyponatremia with severe neurologic symptoms such as seizures

### Hypernatremia

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the serum sodium level in hypernatremia?</td>
<td>[Na(^+)] &gt;145 mEq/L</td>
</tr>
<tr>
<td>What is the serum sodium level in severe hypernatremia?</td>
<td>[Na(^+)] &gt;155 mEq/L</td>
</tr>
<tr>
<td>What are some causes of hypernatremia in the following groups:</td>
<td></td>
</tr>
<tr>
<td>Sodium gain</td>
<td>Excessive saline/bicarb administration; Hypertonic dialysis; Hypertonic feedings</td>
</tr>
<tr>
<td>Water loss</td>
<td>Decreased water intake; Osmotic diuresis (i.e., diabetic ketoacidosis [DKA]); Diabetes insipidus (central and nephrogenic)</td>
</tr>
<tr>
<td>What is the most likely cause of hypernatremia in the emergency department?</td>
<td>Volume loss</td>
</tr>
<tr>
<td>What is the urine output of healthy hypovolemic patients?</td>
<td>Low urine output (&lt;20 mL/hr) and high urine osmolality (&gt;1000 mosmol/kg water)</td>
</tr>
<tr>
<td>What is diabetes insipidus (DI)?</td>
<td>Failure of peripheral or central ADH response</td>
</tr>
<tr>
<td>What are some characteristics of urine of patients with DI?</td>
<td>Low urine osmolality (200–300 mosmol/kg); Low urine sodium (60–100 mEq/kg)</td>
</tr>
<tr>
<td>What are some causes of central DI?</td>
<td>Pituitary surgery; Trauma; Neoplasm</td>
</tr>
<tr>
<td>What is the treatment for central DI?</td>
<td>Identify and correct underlying cause; Sodium restriction; May require vasopressin</td>
</tr>
<tr>
<td>What are some causes of peripheral DI?</td>
<td>Renal disease; Malnutrition; Hypokalemia</td>
</tr>
<tr>
<td>What is the treatment for peripheral DI?</td>
<td>Sodium restriction; May require dialysis</td>
</tr>
<tr>
<td>What are some clinical features of hypernatremia?</td>
<td>Altered mental status such as confusion, dehydration, and seizures</td>
</tr>
<tr>
<td>What is the cornerstone of treatment for hypernatremia due to volume-depletion?</td>
<td>Volume repletion</td>
</tr>
</tbody>
</table>
What is the formula to estimate body water deficit (BWD)?

BWD = TBW \times ([Na^+] / 140 - 1)

What is important to remember with volume-replacement for hypernatremia?

Avoid overly rapid correction due to potential for cerebral edema

**Hypokalemia**

What is the serum potassium level in hypokalemia?

\([K^+] < 3.5 \text{ mEq/L}\)

What is the serum potassium level in severe hypokalemia?

\([K^+] < 2.5 \text{ mEq/L}\)

What are some important causes of hypokalemia in the following conditions:

- **Renal**
  - Renal tubular acidosis, diuretics, Cushing’s syndrome, and hypomagnesemia

- **GI condition**
  - Emesis, starvation, diarrhea, laxative abuse, and colon cancer

- **Other**
  - Hypothyroidism, and intracellular shift

What are some clinical features of hypokalemia, especially if <2.5 mEq/L?

- Pronounced weakness, hyporeflexia, ileus, paralysis, and dysrhythmias

What are some characteristic ECG changes of hypokalemia?

- Flat T-waves, U-waves, ST depression, and prolonged QT interval

What is a concern if a patient with a history of CHF also has hypokalemia?

Potentiates digoxin toxicity

What are some key points in the management of patients who have chronic/subacute hypokalemia?

- Oral replacement of potassium preferred; Correction of any magnesium deficits

What are some key points in the management of patients with acute hypokalemia?

- Acute hypokalemia can be life-threatening; About 40 mEq will raise \([K^+]\) by 1 mEq/L; Give no more than 40 mEq over an hour

**Hyperkalemia**

What is the serum potassium level in hyperkalemia?

\([K^+] > 4.5 \text{ mEq/L}\)

What is the serum potassium level in severe hyperkalemia?

\([K^+] > 6.5 \text{ mEq/L}\)
What are some important causes of hyperkalemia in the following conditions:

- **Renal**
  - Renal failure, aldosterone insufficiency, postassium-sparing diuretics, Type IV renal tubular acidosis

- **Decreased cellular uptake**
  - Drugs (i.e., beta-blockers) and diabetic ketoacidosis

- **Increased potassium level**
  - Hemolysis, GI bleeding, and cellular breakdown such as trauma and rhabdomyolysis

What are some clinical features of hyperkalemia?

- Lethargy, weakness, hypotension, dysrhythmias, and paralysis

What are some ECG changes associated with the following degree of hyperkalemia?

<table>
<thead>
<tr>
<th>Hyperkalemia Level</th>
<th>ECG Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.5–6.5</td>
<td>Peaked/large amplitude T-waves</td>
</tr>
<tr>
<td>6.5–8.0</td>
<td>QRS widening</td>
</tr>
<tr>
<td></td>
<td>PR interval prolongation</td>
</tr>
<tr>
<td></td>
<td>P-wave flattening</td>
</tr>
<tr>
<td>&gt;8.0</td>
<td>Ventricular fibrillation</td>
</tr>
<tr>
<td></td>
<td>Sine wave appearance</td>
</tr>
</tbody>
</table>

What is an important consideration when treating hyperkalemia?

- Whether there any ECG changes

What are some treatment options for hyperkalemia?

<table>
<thead>
<tr>
<th>Treatment Method</th>
<th>Mechanism</th>
<th>Dose</th>
<th>Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuterol</td>
<td>Cellular shifting</td>
<td>10–20 mg (inhaler)</td>
<td>20–30 minutes</td>
</tr>
<tr>
<td>Insulin and glucose</td>
<td>Cellular shifting</td>
<td>15 units of insulin, 50 g of glucose</td>
<td>20–30 minutes</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Cellular shifting</td>
<td>1 mEq/kg IV</td>
<td>10 minutes</td>
</tr>
<tr>
<td>Kayexalate</td>
<td>Excretion</td>
<td>15–30 g PO</td>
<td>1–2 hours</td>
</tr>
<tr>
<td>Furosemide w/NS</td>
<td>Excretion</td>
<td>40 mg IV</td>
<td>1–2 hours</td>
</tr>
<tr>
<td>Hemodialysis</td>
<td>Excretion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>Membrane antagonism</td>
<td>10–30 cc IV</td>
<td>1–2 minutes</td>
</tr>
</tbody>
</table>
### Hypocalcemia

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the serum calcium level in hypocalcemia?</td>
<td>$[\text{Ca}^{2+}] &lt; 8.5 \text{ mg/dL}$</td>
</tr>
<tr>
<td>What is the serum calcium level in severe hypocalcemia?</td>
<td>$[\text{Ca}^{2+}] &lt; 7 \text{ mg/dL}$</td>
</tr>
<tr>
<td>What are some important causes of hypocalcemia?</td>
<td>Hypomagnesemia; Rhabdomyolysis; Hypoparathyroidism; Acute pancreatitis with fat necrosis; Vitamin D deficiency; Renal failure</td>
</tr>
<tr>
<td>What are some clinical features of hypocalcemia?</td>
<td>Typically symptomatic when $[\text{Ca}^{2+}] &lt; 6 \text{ mg/dL}$; HTN, paresthesias, carpopedal spasms, hyperreflexia, seizures</td>
</tr>
<tr>
<td>What is Chvostek’s sign?</td>
<td>Tapping of facial nerve that results in tetany</td>
</tr>
<tr>
<td>What is Trousseau’s sign?</td>
<td>Carpal spasm that may be elicited by occluding the brachial artery (i.e., BP cuff)</td>
</tr>
<tr>
<td>What are some key points in the management of hypocalcemia?</td>
<td>Identify and treat the underlying cause; CaCl$_2$ (10% solution) over 20 minutes if acutely symptomatic</td>
</tr>
</tbody>
</table>

### Hypercalcemia

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the serum calcium level in hypercalcemia?</td>
<td>$[\text{Ca}^{2+}] &gt; 10.5 \text{ mg/dL}$</td>
</tr>
<tr>
<td>What is the serum calcium level in severe hypercalcemia?</td>
<td>$[\text{Ca}^{2+}] &gt; 12 \text{ mg/dL}$</td>
</tr>
<tr>
<td>What are some important causes of hypocalcemia?</td>
<td>Malignancy; Vitamin D toxicity; Acute osteoporosis; Hyperparathyroidism; Sarcoidosis</td>
</tr>
<tr>
<td>What are some clinical features of hypercalcemia in the following systems:</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Nausea, emesis, abdominal pain, constipation, and anorexia</td>
</tr>
<tr>
<td>GU</td>
<td>Renal failure, nephrolithiasis, and polyuria</td>
</tr>
<tr>
<td>Chronic Villus Sampling (CVS)</td>
<td>Hypertension, dysrhythmias, and digitalis sensitivity</td>
</tr>
<tr>
<td>Central Nervous System (CNS)</td>
<td>Confusion, lethargy, weakness, and hyporeflexia</td>
</tr>
</tbody>
</table>
What are some ECG changes that can be seen with hypercalcemia?
What are some key points in the management of hypercalcemia?

Short QT interval, widening of the T-wave, and heart block
Identify and treat the underlying cause; Decrease bone reabsorption with bisphosphonates; Furosemide with normal saline (NS); Avoid thiazide diuretics

Hypomagnesemia

What is the serum magnesium level in hypomagnesemia?

What is the serum magnesium level in severe hypomagnesemia?

What are some important causes of hypomagnesemia?

Pancreatitis; Alcoholism; Malnutrition; Endocrine disorder (i.e., DKA)

What are some clinical features of hypomagnesemia?

Similar to hypocalcemia: hypotension, tetany, tremors, dysrhythmias, hypocalcemia, and hypokalemia

What are some ECG findings that can be seen in hypomagnesemia?

What are some key points in the management of hypomagnesemia?

Prolongation of PR and QT interval, ST depression, and wide QRS complex
Identify and treat the underlying cause; Check serum potassium and calcium; Magnesium sulfate replacement

Hypermagnesemia

What is the serum magnesium level in hypermagnesemia?

What is the serum magnesium level in severe hypermagnesemia?

What are some important causes of hypermagnesemia?

Renal failure; Iatrogenic; Adrenal insufficiency

What are some clinical features of hypermagnesemia?

Hyporeflexia, weakness, respiratory depression, hypotension, bradycardia, and systole in very high levels

What are some ECG findings that can be seen in hypermagnesemia?

Extreme ST elevation and T-waves along with prolonged PR and QT interval
What are some key points in the management of hypermagnesemia? Identify and treat the underlying cause; Dialysis for severe serum levels; Calcium gluconate for conduction problems

### Hypochloremia

What is the serum chloride level in hypochloremia? $[\text{Cl}^-] <100$ mEq/L

What is the serum chloride level is severe hypochloremia? $[\text{Cl}^-] <70$ mEq/L

What are some causes of hypochloremia? GI loss such as diarrhea and emesis; Hypokalemic alkalosis

What are some key points in the management of hypochloremia? Identify and treat the underlying cause; NaCl for severe hypochloremia or hypokalemic alkalosis

### Hyperchloremia

What is the serum chloride level in hyperchloremia? $[\text{Cl}^-] >110$ mEq/L

What is the serum chloride level is severe hyperchloremia? $[\text{Cl}^-] >120$ mEq/L

What are some causes of hyperchloremia? Bicarbonate loss; Dehydration

What are some key points in the management of hyperchloremia? NS for GI bicarbonate loss; Bicarbonate for renal bicarbonate loss

### Acid and Base Balance

Name three types of acid the body handles to maintain acid-base balance.
1. Exogenous acid
2. Abnormal metabolic pathway
3. Fixed acids

Name two organs that are crucial for maintaining acid-base balance.
1. Lungs
2. Kidneys

How much volatile acids does the lung excrete each day? 15,000 mg in the form of CO₂

How much nonvolatile acids does the kidney excrete each day? 70 mEq/L

What are three mechanisms by which the kidneys excrete nonvolatile acid?
1. Excretion with ammonia
2. Excretion with urinary buffers
3. Direct hydrogen excretion

What maintains regulation of hydrogen ion concentration on a minute-to-minute basis? Bicarbonate-carbonic acid system
What are important things to consider in the history of a patient who presents with abnormal acid-base status?

- Respiratory status;
- Volume status;
- Medication; Illicit drug use

What are some important laboratory tests to consider when evaluating an acid-base disturbance?

- Arterial blood gas;
- Electrolytes;
- Determination of an anion gap acidosis

Briefly give some causes for the following acid-base disturbance:

- **Respiratory acidosis**
  - Opioids; Respiratory failure; Sedative-hypnotics
- **Respiratory alkalosis**
  - Liver failure; Salicylates; Heart failure
- **Anion gap metabolic acidosis**
  - Hypoxia; Sepsis; Seizures
- **Normal anion gap acidosis**
  - Renal tubular acidosis; Elevated chloride
- **Metabolic alkalosis**
  - Volume depletion; Hyperaldosteronism

What else is important to consider in an acid-base disturbance?

- Existence of a mixed acid-base disturbance

Name two characteristic laboratory findings in metabolic acidosis?

1. pH <7.35
2. HCO₃⁻ <20 mEq/L

What is one of the most common cause of metabolic acidosis in the emergency setting?

- Lactic acidosis

What are some causes of normal anion gap acidosis (>Cl⁻)?

- Renal tubular acidosis; Diarrhea;
- Extensive fluid resuscitation; Adrenal insufficiency

What are some causes of an anion gap acidosis:

- Methanol
- Uremia
- DKA
- Paraldehyde
- Iron, Isoniazid
- Lactic acidosis
- Ethylene glycol
- Salicylates, strychnine

What is the treatment for metabolic acidosis?

- Identify and treat the underlying cause; Consider use of sodium bicarbonate if pH <7.1 or bicarbonate <5 mEq/L

Name two characteristic laboratory finding in metabolic alkalosis?

1. pH >7.45
2. HCO₃⁻ >26 mEq/L
How can one characterize metabolic alkalosis even further? Chloride-sensitive versus chloride-resistant
What are some causes of chloride-sensitive alkalosis? Diuretics; Emesis; Nasogastric suction
What is the treatment of choice for chloride-sensitive alkalosis? Normal saline
What are some causes of chloride-resistant alkalosis? Mineralocorticoid excess; Primary reninism; Chronic potassium depletion
What is the treatment of choice for chloride-resistant alkalosis? Correction of hypovolemia; Acetazolamide may help; Administer potassium as a chloride salt

Name two characteristic laboratory findings in respiratory acidosis? 1. pH < 7.40
2. CO₂ > 45 mm Hg;

What are some causes of respiratory acidosis? Neuromuscular disease; CNS depression; Chronic obstructive pulmonary disease (COPD)

How long before full renal compensation occurs? 48 hours of steady-state alteration

What are some key points in the treatment of respiratory acidosis? Identify and treat the underlying cause; Bronchodilators for COPD/bronchospasms; Assisting and increasing ventilation; Oxygen therapy (reduces pulmonary HTN); Drugs to reduce sedation

Name two characteristic laboratory findings in respiratory acidosis? 1. pH > 7.4
2. CO₂ < 35 mm Hg

What are some causes of respiratory alkalosis? CNS (i.e., anxiety); Drugs (i.e., salicylates); Hypoxemia

What are some key points in the treatment of respiratory alkalosis? Identify and treat the underlying cause; Respiratory alkalosis rarely life-threatening; Avoid rapid correction of PaCO₂
# Neurologic Emergencies

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## HEADACHES

### Cluster Headaches

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of a cluster headache (HA)?</td>
<td>Clustering of painful HA over a period of many weeks, peaks in about 5 minutes, and may last for an hour</td>
</tr>
<tr>
<td>What are some factors associated with cluster HAs?</td>
<td>Male gender; Smoking; EtOH use</td>
</tr>
</tbody>
</table>
| What are two types of cluster HAs?                                       | 1. Chronic—unsustained attacks  
2. Episodic—occurs in bouts                                                                                                         |
| What are some clinical features of cluster HAs?                          | Burning HA on unilateral side, lacrimation and flushing on affected side, and Horner’s syndrome                                           |
| What is Horner’s syndrome?                                               | Deficiency of sympathetic activity. The site of lesion to the sympathetic outflow is on the ipsilateral side of the symptoms           |
| What are some findings in Horner’s syndrome?                             | Ipsilateral ptosis; Anhidrosis; Miosis; Enophthalmos                                                                                        |
Name some commonly used medications for abortive therapy.
Sumatriptan; Dihydroergotamine; 100% oxygen

Name some commonly used medications for prophylactic treatment.
Beta-blockers; Tricyclic antidepressants; Calcium channel blockers

**Migraine**

What are some important things to know about migraines?
Severe headache that afflicts millions; Often preceded by sensory warning sign; Affects women more commonly than men

What are some of the mechanisms by which migraines occur?
Vascular structure involvement (constriction); Serotonergic involvement; Involvement of the trigeminal nerve

What are some features of auras?
They will often precede attacks of migraines; Often have visual phenomena; Can have motor/sensory disturbances; Present in about 15% of migraines

What are some factors that may provoke or exacerbate a migraine?
Physical activity; Changes in sleep cycle; Menstruation; Particular foods (i.e., chocolate)

What are some clinical features of migraines?
Pulsating, severe, unilateral headache often associated with nausea, emesis, and photophobia/phonophobia

What are some commonly used prophylactic medications for migraines?
Beta-blockers; Tricyclic antidepressants; Calcium channel blockers

What are some commonly used abortive therapy for migraines?
Sumatriptan (typically outpatient); Metoclopramide/prochlorperazine; Nonsteroidal anti-inflammatory drugs(NSAIDs); Dihydroergotamine

What are some other important diagnosis to consider?
Cluster HA; SAH; Tension HA

**Giant Cell Arteritis aka Temporal Arteritis**

What is giant cell arteritis (GCA)?
Inflammation of one or more branches of the external carotid artery

Name three branches of the carotid artery that are commonly affected by GCA.
1. Temporal artery
2. Posterior ciliary artery
3. Ophthalmic artery
What are some important things to know about GCA?
Rare before the age of 50; Mean age of onset is 70; Females commonly more affected

What is the most feared complication of GCA?
Irreversible blindness; Cerebral vascular accident (CVA)

What rheumatic condition is GCA commonly associated with?
Polymyalgia rheumatica

What are some clinical features of GCA?
Unilateral burning headache worse at night often accompanied with tender/pulseless temporal artery, scalp tenderness, jaw claudication, and decreased visual acuity

What is an important diagnostic laboratory test to obtain in GCA?
Erythrocyte sedimentation rate (ESR) (Between 50–100 mm/hr)

What study confirms the diagnosis of GCA?
Temporal artery biopsy

What are some key points in the management of GCA?
Treatment must be started immediately; High-dose prednisone; Temporal artery biopsy commonly done

Subarachnoid Hemorrhage

What are some important things to know about a subarachnoid hemorrhage (SAH)?
Accounts for about 10% of CVA; Ruptured saccular aneurysms common cause; Arteriovenous malformation (AVM) is another cause (less common)

What are other causes of SAH?
Illicit drug use (especially cocaine); Intracranial arterial dissections; Bleeding diathesis

What are important risk factors for the development of aneurysm formation, and hence hemorrhage?
Cigarette smoking; Moderate to heavy alcohol consumption; Hypertension; Family history of SAH; Antithrombotic therapy

What are some common clinical features of SAH?
Sudden, severe headache described as the “worst headache of my life,” commonly associated with brief loss of consciousness, seizure, nausea, vomiting, or meningismus

What is a sentinel headache?
Sudden and severe headache that often precedes a major SAH by 6–20 days: minor hemorrhage

What percentage of patients will manifest a sentinel headache prior to SAH?
Up to 50%
What are some complications to consider in SAH?
Seizures; Increase in intracranial pressure (ICP); Hyponatremia; Vasospasm—ischemia

How is SAH diagnosed?
Noncontrast head CT with or without lumbar puncture (LP) after CT of the head

What are some important points in the following diagnostic tests used for SAH:

Noncontrast head CT
Cornerstone for diagnosis of SAH; Sensitivity of head CT is highest early on; Less sensitive for minor bleeds

Lumbar puncture
Mandatory if there is a strong suspicion of SAH despite a normal head CT; Elevated opening pressure is classic; Elevated red blood cell (RBC) count

What is xanthochromia?
Pink or yellow tint that represents hemoglobin degradation products, commonly seen 2–4 hours after bleed

When is the optimal time to perform an LP to detect xanthochromia?
12 hours after onset of HA is optimal; Xanthochromia can last for up to 2 weeks

What are the three most common reasons to miss a diagnosis of SAH?
1. Failure to obtain a CT (know its limitations)
2. Failure to obtain an LP
3. Attribute HA to other causes like a migraine

What is the treatment objective in a patient with SAH?
Stabilization; Prevent rebleeding; Prevent vasospasms (i.e., nimodipine)

What are some key points in the management of SAH?
Airway breathing circulation(ABC); Urgent neurosurgical consultation; Slowly lower BP (i.e., labetalol); Treat for pain and emesis; Use nimodipine in consultation with neurosurgery

**SEIZURES**

What is the definition of a seizure?
Uncontrolled rhythmic electrical discharge within the brain that usually, but not always, results in characteristic abnormal movements of the body
What is the general incidence of recurrent seizures?

1–2%

What is typically the cause of primary seizures (epilepsy)?

Genetically determined, usually at a early age

What are some identifiable causes of seizures?

Intracranial mass; Vascular malformation; Infections; Toxicological (i.e., EtOH); Endocrine (hypoglycemia); Electrolyte

What are some elements in the history to obtain in a patient who presents with a seizure?

Whether the patient has a history of recurrent seizures; The circumstances that led to the seizure; Observed ictal behavior; Identify potential triggers (i.e., emotions); Loss of bladder/bowel function; Current medication

Define partial seizures.

Localized electrical discharge of the cerebral cortex

What are some important points for the following types of partial seizures?

- **Simple partial**
  - No alteration of consciousness;
  - Symptoms based on cortex affected;
  - Visual changes if occipital affected

- **Complex partial**
  - Consciousness is impaired; A simple partial with mentation affected;
  - Often due to discharge of temporal region

Define generalized seizures.

Global discharge of the cerebral hemisphere

What are some important points for the following types of complex seizures?

- **Absence (petit mal)**
  - Typically very brief, lasting only a few seconds; Loss of consciousness, but not postural tone; Will often continue unaware of event; Classically affects school-aged children

- **Atonic**
  - Less common type of seizure; Sudden loss of postural tone; May have brief loss of consciousness

- **Tonic**
  - Less common type of seizure; Prolonged contraction of the body; Often will be pale and flush

- **Clonic**
  - Less common type of seizure; Repetitive clonic jerks without tonic element
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoclonic</td>
<td>Less common type of seizure; Brief and shock-like movement of extremity; May affect entire body or just one limb</td>
</tr>
<tr>
<td>Tonic-clonic (grand mal)</td>
<td>Abrupt loss of consciousness; Typically starts with tonic (rigid) phase; Often clonic phase comes after tonic phase; Loss of bladder/bowel function is normal; Consciousness returns slowly</td>
</tr>
<tr>
<td>What are important elements in the physical exam to look for?</td>
<td>Evaluation of fractures and injuries; Particular attention to the head and spine; Vitals as well as glucose is important; A thorough neurological exam is crucial</td>
</tr>
<tr>
<td>What is Todd’s paralysis?</td>
<td>A focal neurological deficit that typically follows a simple-partial seizure that resolves within 48 hours</td>
</tr>
<tr>
<td>What are some other conditions that seizures can be mistaken for?</td>
<td>Syncope; Neuromuscular disorders; Migraines; Narcolepsy; Pseudoseizure</td>
</tr>
</tbody>
</table>
| What are four clinical features that help to distinguish seizures from other causes? | 1. Inability to recall attack  
2. Postictal confusion and lethargy  
3. Abrupt onset  
4. Purposeless movement                                                                 |
| What are some important diagnostic studies to consider in the evaluation of a seizure? | Anticonvulsant medication level; Glucose level very important; Chemistry for electrolyte imbalance; Complete blood count (CBC) for possible infection; Toxicology screen; Urinalysis; CT or MRI |
| What are some indications where imaging of the head is warranted?       | First time seizure (absence of fever); Seizure pattern that is different; New focal deficits; Recent head trauma; Use of any anticoagulants; Any suspicion of meningitis |
| What is the role of EEG in the evaluation of seizures?                  | Not typically utilized in the emergency department (ED); Typically done on outpatient basis; Can be used to classify seizure type |
| What are some key points in the management of seizures?                | Ensure intact ABCs; IV/O₂/monitor; Accurate diagnostic evaluation should be the first step                                          |
| What is typically done for actively seizing patients?                  | Management is expectant most of the time; Most seizures self-terminate within minutes; Medication for prolonged seizure; Gentle firm restraint should be used; Turn to side to avoid aspiration |
What is the most common reason that a person with a seizure disorder has a seizure?

Subtherapeutic anticonvulsant level

What is typically done for a patient with a seizure disorder who is therapeutic on anticonvulsant and still has a seizure?

If a single seizure, the focus is to identify precipitants that may have lowered the seizure threshold

Name some commonly used anticonvulsants.

Carbamazepine; Phenytoin/ Fosphenytoin; Valproic acid

What is the definition of status epilepticus (SE)?

Prolonged or clustered seizures that sometimes develop into non-stop seizures typically >30 minutes

What are some complications of SE?

Hypoxia; Hyperthermia; Acidosis; Permanent neural damage

What are some key points in the management of SE?

ABCs—protect the airway in particular; Any cause of seizure can cause SE; Initial laboratory tests should include glucose, toxicology test, etc.; Intubation may make it difficult to monitor SE

What are the three classes of medications used to treat SE?

1. Benzodiazepines
2. Phenytoin/Fosphenytoin
3. Phenobarbital

How successful is the combination of benzodiazepines and phenytoin in controlling seizures?

70–90%

What medication is commonly used in refractory cases of seizures?

Intravenous phenobarbital

What is a treatment option for refractory status epilepticus?

Endotracheal intubation, and EEG monitoring

MENINGITIS

Cerebrospinal Fluid (CSF)

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Viral</th>
<th>Bacterial</th>
<th>TB</th>
<th>Fungal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (mg/dL)</td>
<td>&lt;55</td>
<td>&lt;200</td>
<td>&gt;200</td>
<td>&gt;200</td>
<td>&gt;200</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>&gt;40</td>
<td>&gt;40</td>
<td>&lt;40</td>
<td>&lt;40</td>
<td>&lt;40</td>
</tr>
<tr>
<td>WBCs (µL)</td>
<td>&lt;5</td>
<td>&lt;1000</td>
<td>&gt;1000</td>
<td>&lt;1000</td>
<td>&lt;500</td>
</tr>
<tr>
<td>Gram stain</td>
<td>negative</td>
<td>negative</td>
<td>positive</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>Opening pressure (mm CSF)</td>
<td>&lt;170</td>
<td>~200</td>
<td>&gt;300</td>
<td>~200</td>
<td>~300</td>
</tr>
</tbody>
</table>

WBC, white blood cell
What is important to know about meningitis? One of the top 10 infectious causes of death; Causes over 100,000 deaths worldwide; Permanent neurologic deficits are common

What is meningitis? Inflammation of the leptomeninges

What are some common causes? Bacterial; Viral; Fungal; Tuberculosis

What is the mortality of a missed diagnosis of bacterial meningitis? 15–50%

What are the top three causes of meningitis: Depends on the age group

- In infants. *Streptococcus pneumoniae*; *Group B streptococcus infection*; *Escherichia Coli*

- In young adults. *S. pneumoniae*; *Nisseria meningitidis*; *Haemophilus influenza*

- In people over 60. *S. pneumoniae*; *Listeria monocytogenes*; *N. meningitidis/group B streptococcus infection*; *H. influenza*

- In nosocomial infections. *Pseudomonas aeruginosa*; Other gram negatives; *Staphylococcus aureus*

What are some risk factors for meningitis? Colonization of the nasopharynx; Bacteremia (endocarditis/UTI); Contiguous source (mastoid/sinus); Living in a dormitory or barracks

What are some host factors that can predispose to meningitis? Asplenia; HIV; Complement deficiency; Long-term steroid use

What is the classic triad of symptoms in meningitis? Fever; Nuchal rigidity; Mental status change

What are some other clinical features of meningitis? Headache; Significant photophobia; Nausea and vomiting; Seizures and focal neurological deficits; Rash

What is Kernig’s sign? Inability to extend patient’s knee due to pain when leg is flexed with hip at 90°

What is Brudzinski’s sign? Passive flexion of the patient’s neck causes flexion of both hips


Name two important diagnostic tests used to diagnose meningitis? 1. LP 2. Noncontrast head CT

What is the reason a noncontrast head CT is done prior to an LP? To rule out intracranial masses (elevated ICP)
What are some key points in the management of meningitis?
Empirical treatment should not be withheld for diagnostic tests; Always maintain a high index of suspicion; Do not wait for CT/LP to start treatment

What empiric treatment is commonly used?
Ceftriaxone; Ampicillin; Vancomycin; Acyclovir

When is chemoprophylaxis indicated?
High-risk contacts of patients with; N. meningitidis; H. influenzae type B

What is the drug of choice for chemoprophylaxis?
Rifampin

What role do steroids play in meningitis?
Should be given before or with the first dose of antibiotics, mostly beneficial in pneumococcal meningitis

CEREBRAL VASCULAR ACCIDENT

NIH Stroke Scale

<table>
<thead>
<tr>
<th>Category</th>
<th>Patient Response and Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.a. Level of consciousness:</td>
<td>0 Alert</td>
</tr>
<tr>
<td></td>
<td>1 Not alert, but arousable with minimal stimulation</td>
</tr>
<tr>
<td></td>
<td>2 Not alert, but requires repeated stimulation</td>
</tr>
<tr>
<td></td>
<td>3 Coma</td>
</tr>
<tr>
<td>1.b. Ask patient the month and age:</td>
<td>0 Answers both questions correctly</td>
</tr>
<tr>
<td></td>
<td>1 Answers one question correctly</td>
</tr>
<tr>
<td></td>
<td>2 Both questions answered incorrectly</td>
</tr>
<tr>
<td>1.c. Ask patient to open and close eyes and fist:</td>
<td>0 Obeys both correctly</td>
</tr>
<tr>
<td></td>
<td>1 Obeys one correctly</td>
</tr>
<tr>
<td></td>
<td>2 Does not obey either commands</td>
</tr>
<tr>
<td>2. Best gaze:</td>
<td>0 Normal</td>
</tr>
<tr>
<td></td>
<td>1 Partial gaze palsy</td>
</tr>
<tr>
<td></td>
<td>2 Forced deviation</td>
</tr>
<tr>
<td>3. Vision field testing:</td>
<td>0 No visual field loss</td>
</tr>
<tr>
<td></td>
<td>1 Partial hemianopia</td>
</tr>
<tr>
<td></td>
<td>2 Complete hemianopia</td>
</tr>
<tr>
<td></td>
<td>3 Bilateral hemianopia</td>
</tr>
</tbody>
</table>

(Continued)
What are some important things to note about the posterior circulation?

Originates from the vertebrobasilar arteries; Supplies 20% of cerebral blood flow; The following structures are supplied:

<table>
<thead>
<tr>
<th>Category</th>
<th>Patient Response and Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. Facial paresis:</td>
<td></td>
</tr>
<tr>
<td>5. Motor function—arm:</td>
<td></td>
</tr>
<tr>
<td>6. Motor function—leg:</td>
<td></td>
</tr>
<tr>
<td>7. Limb ataxia:</td>
<td></td>
</tr>
<tr>
<td>8. Sensory:</td>
<td></td>
</tr>
<tr>
<td>9. Best language:</td>
<td></td>
</tr>
<tr>
<td>10. Dysarthria:</td>
<td></td>
</tr>
<tr>
<td>11. Extinction and inattention:</td>
<td></td>
</tr>
</tbody>
</table>

NIH Stroke Scale (Continued)
Brainstem; Upper spinal cord; Medial portion of temporal lobe; Cerebellum; Thalamus; Occipital lobe

What are some important things to note about the anterior circulation?

Originates from the carotid arteries; Supplies 80% of cerebral blood flow; The following structures are supplied; Anterior portion of temporal lobe; Frontoparietal lobes; Optic nerve and retina

What is the circle of Willis?

Circle of arteries that supply the brain; creates redundancies in the cerebral circulation so if one vessel is blocked, blood flow from other vessels can maintain perfusion

Name some important causes of a CVA and some examples:

Ischemic stroke

Embolic
- Emboli from the heart (i.e., atrial fibrillation); Endocarditis;
- Plaques from large vessels (i.e., carotid)

Thrombotic
- Atherosclerosis; Sickle cell disease; Mycotic aneurysms;
- Hypercoagulable states; Vasculitis

Hemorrhagic stroke

Trauma; AV malformation; Bleeding disorders; Spontaneous rupture of berry aneurysm; Transformation of an ischemic stroke

List some important risk factors of a CVA.

Transient ischemic attack (TIA);
- Hypertension; Cardiac disease;
- Diabetes; Atherosclerosis;
- Erythrocytosis; Dyslipidemia

What are some other conditions that may mimic a stroke with respect to focal neurologic deficits?

Migraines; Hypoglycemia; Hepatic encephalopathy; Seizures

What is the definition of a TIA?

Blood supply to part of the brain is briefly interrupted, resulting in a transient stroke that lasts only a few minutes, but may persist up to 24 hours

What is the clinical significance of a TIA?

“Red flag” of an impending stroke in evolution
What are some clinical features for the following stroke syndrome based on the occluded vessel:

**Middle cerebral artery (MCA)**
- Contralateral hemiplegia/hemianesthesia; Upper extremity deficit more severe than lower extremity deficit; Gaze preference toward the affected side; Aphasia (dominant hemisphere affected); Constructional apraxia/agnosia (non-dominant hemisphere affected)

**Posterior cerebral artery (PCA)**
- Ipsilateral cranial nerve (CN) III nerve palsy; Contralateral homonymous hemianopsia, hemisensory loss, and hemiparesis

**Anterior cerebral artery (ACA)**
- Contralateral foot, leg, and arm paralysis; Lower extremity deficit more severe than upper extremity deficit; Frontal lobe disinhibition (i.e., abulia)

**Cerebellar infarct**
- Nausea, vomiting, ataxia, vertigo, lateralizing dysmetria, and nystagmus

**Basilar artery**
- Quadriplegia: severe bilateral signs; Coma; “Locked-in syndrome”—no motor function except upward gaze of eyes

---

**What is an important consideration in a stroke patient with a depressed level of consciousness?**
- Airway management (i.e., intubation)

**What is the NIH stroke scale?**
- Objective way to rapidly assess and determine the extent of neurologic deficits of a stroke patient and helps to determine if thrombolytics are needed

**Although hypertension is commonly associated with CVA, should it be treated in the ED?**
- Generally not—lowering the BP aggressively may worsen the stroke

**What is important to consider in the initial management of a patient who presents with a suspected stroke?**
- Determine if he/she is a candidate for lytics; Immediate CT scan (i.e., rule out bleeds); Establish onset of symptoms

**What are some important guidelines in determining if a patient is a candidate for thrombolytic therapy?**
- If symptom onset is within 3 hours; Significant neurologic deficit; Recommended blood pressure limits; No contraindications such as recent SAH
List some contraindications to the administration of thrombolytics in acute stroke?

- History of structural CNS disease;
- Systolic pressure >180 mm Hg;
- Significant head trauma in <3 months;
- History of intracranial hemorrhage;
- Recent trauma >6 weeks; Recent GI/GU bleeding

VERTIGO

<table>
<thead>
<tr>
<th>Central versus Peripheral Vertigo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central</td>
</tr>
<tr>
<td>Onset</td>
</tr>
<tr>
<td>Severity</td>
</tr>
<tr>
<td>Nystagmus</td>
</tr>
<tr>
<td>Auditory symptoms</td>
</tr>
<tr>
<td>Pattern</td>
</tr>
<tr>
<td>CNS symptoms</td>
</tr>
<tr>
<td>Prognosis</td>
</tr>
</tbody>
</table>

What is the definition of vertigo?

Sensation of movement of oneself or the surrounding area most often described as a feeling of spinning.

What is the pathophysiology of peripheral vertigo?

Disorders of the ear or CN VIII.

How much does peripheral vertigo account for all cases of vertigo?

85%.

What are some common clinical features of peripheral vertigo?

Sudden onset of intense sensation of intermittent disequilibrium, nausea and vomiting, hearing loss/tinnitus common; nystagmus common as well.

What are some important causes of peripheral vertigo?

Benign positional vertigo (BPV); Ototoxic drugs; Otitis media; Menière’s disease.

What is the Dix-Hallpike maneuver (i.e. Nylen Barany)?

Used to diagnose and treat BPV.
What are some key steps in the Dix-Hallpike maneuver?
Sit with patient’s legs extended on the examination table; Patient is brought rapidly from sitting to supine, head slightly extended below horizontal, then head is rotated to right and left quickly.

What are some treatments commonly used for peripheral vertigo?
Antihistamine; Antiemetics; Anticholinergics; Benzodiazepines in severe cases.

Despite intense symptoms of peripheral vertigo, do patients typically require admission?
No—usually can treat on outpatient basis, central vertigo is a different story.

What is the pathophysiology of central vertigo?
Commonly due to lesions of the cerebellum or brainstem.

What are some clinical features of central vertigo?
Mild, but constant disequilibrium that may present acutely, nausea/vomiting, vertical nystagmus, and often will have associated CNS symptoms.

What are some CNS symptoms that can be associated with central vertigo?
Lateralizing dysmetria, ataxia, dysarthria, scotomata, and blindness.

What are some important causes of central vertigo?
Multiple sclerosis; Cerebellar tumors; Brainstem infarct; Vertebrobasilar insufficiency.

What is the deposition of patients with central vertigo?
Often require admission for further evaluation.

PERIPHERAL NEUROLOGIC LESIONS

<table>
<thead>
<tr>
<th>Muscles and Motor Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper extremities</td>
</tr>
<tr>
<td>Deltoid</td>
</tr>
<tr>
<td>Biceps</td>
</tr>
<tr>
<td>Triceps</td>
</tr>
<tr>
<td>Lower extremities</td>
</tr>
<tr>
<td>Quadriceps</td>
</tr>
<tr>
<td>Iliopsoas</td>
</tr>
<tr>
<td>Gluteal</td>
</tr>
<tr>
<td>Anterior tibial</td>
</tr>
<tr>
<td>Reflexes</td>
</tr>
<tr>
<td>Supinator</td>
</tr>
<tr>
<td>Biceps</td>
</tr>
<tr>
<td>Triceps</td>
</tr>
</tbody>
</table>
# Myopathies and Myelopathies

## What are some defining features of myopathies?
- Proximal weakness (i.e., standing up);
- DTRs are typically intact; No alterations in sensation; Often have abnormal laboratory test results (i.e., CPK, sedimentation rate, and elevated WBC)

## What are some clinical features for the following types of common myopathies:

<table>
<thead>
<tr>
<th>Myopathy Type</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steroid myopathy</td>
<td>Long-term steroid use that is associated with muscle weakness and pain</td>
</tr>
<tr>
<td>Polymyositis</td>
<td>Acute inflammation often leads to proximal muscles weakness and pain; Often have elevated CPK; Patients can also have low-grade fever</td>
</tr>
<tr>
<td>Hypokalemic myopathy</td>
<td>Typically due to renal tubular acidosis; Often get proximal weakness as well; Consider toluene abuse as well as; Fanconi’s syndrome</td>
</tr>
</tbody>
</table>

## What are some clinical features for the following types of myelopathies:

<table>
<thead>
<tr>
<th>Myelopathy Type</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple sclerosis</td>
<td>Demyelinating disorder thought to be autoimmune in origin; Often have spinal cord involvement that results in upper motor neurons (UMN) signs and bladder/bowel dysfunction; Corticosteroids often used for exacerbations</td>
</tr>
<tr>
<td>Syringomyelia</td>
<td>Cyst forms within the spinal cord and over time destroys the center of the cord; Sensory disruption, especially in the hands; Can adversely affect sweating, sexual function, and bladder/bowel control</td>
</tr>
<tr>
<td>Epidural mass</td>
<td>Can be due to abscesses, metastatic tumor, and epidural hemorrhage; Commonly severe pain and signs of cord compression (i.e., sensory alterations)</td>
</tr>
<tr>
<td>Dorsal column disorders</td>
<td>Commonly due to B₁₂ deficiency or syphilis; Loss of position sense, vibration, and light touch</td>
</tr>
</tbody>
</table>
## Neuromuscular Junction

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the most common disorder of the neuromuscular junction?</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>What is myasthenia gravis?</td>
<td>Chronic autoimmune neuromuscular disease characterized by varying degrees of weakness of the skeletal muscles with no sensory involvement</td>
</tr>
<tr>
<td>What is the hallmark of myasthenia gravis?</td>
<td>Weakness that is typically first evident in the eyelids and extraocular muscles with generalized weakness of the limbs following</td>
</tr>
<tr>
<td>What are some ways that myasthenia gravis can be diagnosed?</td>
<td>Electromyogram; Serology (used with clinical picture); Edrophonium test</td>
</tr>
<tr>
<td>What are some treatment options in myasthenia gravis?</td>
<td>Pyridostigmine; Prednisone; IV gamma globulin, and may be thymectomy</td>
</tr>
<tr>
<td>What is the most important complication to consider in myasthenia gravis?</td>
<td>Respiratory failure (i.e., diaphragm)</td>
</tr>
<tr>
<td>What is myasthenic crisis?</td>
<td>Severe weakness from acquired myasthenia gravis (MG) that is severe enough to require intubation often due to dysfunctional deficiency of acetylcholine (ACh)</td>
</tr>
<tr>
<td>What is the treatment of choice for myasthenic crisis?</td>
<td>Intravenous immunoglobulin G; Plasmapheresis</td>
</tr>
<tr>
<td>What other crisis can also occur with myasthenia gravis?</td>
<td>Cholinergic crisis</td>
</tr>
<tr>
<td>How does cholinergic crisis commonly occur?</td>
<td>When too much acetylcholinesterase inhibitors are used that result in an excess of ACh are received</td>
</tr>
<tr>
<td>What are some clinical features of cholinergic crisis?</td>
<td>Often cholinergic with muscarinic effects such as excessive salivation and urination along with severe muscle weakness and possible respiratory failure</td>
</tr>
<tr>
<td>What treatment is commonly used for cholinergic crisis?</td>
<td>Atropine</td>
</tr>
<tr>
<td>What is Eaton-Lambert syndrome?</td>
<td>Presynaptic disorder of neuromuscular transmission defined by impaired release of acetylcholine (ACh) that causes proximal muscle weakness, depressed tendon reflexes, and autonomic changes</td>
</tr>
</tbody>
</table>
What does Eaton-Lambert syndrome have a high association with? Lung cancer
What complication should patients with Eaton-Lambert syndrome be monitored for? Respiratory failure (rare)
What are other important differentials to consider in patients with generalized weakness? Tick paralysis; Botulism
Amyotrophic lateral sclerosis (ALS); Organophosphate poisoning

### Neuropathies

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of a neuropathy?</td>
<td>Disorders of peripheral nerves</td>
</tr>
</tbody>
</table>
| What are the three types of nerves that make up the peripheral nervous system (PNS)? | 1. Motor nerves  
2. Sensory nerves  
3. Autonomic nerves |
| What are some clinical features of peripheral neuropathies?             | Mixed sensory/motor involvement typical; Reflexes usually absent; Impairment is typically symmetrical/distal |
| What disorders are commonly associated with peripheral neuropathies?   | Diabetes; Uremia; Cancer; Hypothyroidism; Tick paralysis; Guillain-Barré syndrome |
| What toxins are also commonly associated with peripheral neuropathies?  | Organophosphates; Tetanus; Heavy metals (i.e., lead); Ethanol |

### LOWER BACK PAIN

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important things to know about lower back pain (LBP)?</td>
<td>One of the most common ED complaints; Up to 80% have experienced LBP; LBP is more prevalent between the age of 20–40 years; LBP in elderly patients is more concerning</td>
</tr>
<tr>
<td>What are red flags in the history of a patient that presents with LBP?</td>
<td>Age &gt;50; History of cancer; Constitutional symptoms: fever, weight loss, etc.; Intravenous drug abuse (IVDA); Recent instrumentation; Incontinence; Neurological deficits</td>
</tr>
<tr>
<td>What are some findings on physical exam that is more concerning for serious pathology?</td>
<td>Positive straight leg raise; Neurological deficit; Any vertebral point tenderness</td>
</tr>
</tbody>
</table>
What are some clinical features of the following nerve root involvement:

L3/L4  
- Diminished or absent knee jerk;
- Weakness in the quadriceps;
- Anteromedial thigh and knee pain

L5  
- There is usually no reflex loss; Foot drop common

S1  
- Ankle jerk is often diminished or absent; There may be weakness of toe flexors; Leg pain is often worse than LBP

What is straight leg raising?
- Roots may be impinged upon stretching the nerve root causing pain

What are the three classifications that acute LBP can be placed into?
1. Symptoms referable to serious conditions
2. Sciatica
3. Nonspecific back pain

What is the most common cause of back pain?
- Strain of soft tissue elements in the back

What is sciatica?
- Pain radiating in a dermatomal distribution

What are some common causes of sciatica?
- Herniated disc; Tumor, infection, or hematoma compression; Spinal stenosis

How long does it typically take for nonspecific LBP to resolve?
- Within a month

What are some key points in the management of nonspecific lower back pain?
- Appropriate analgesia; Activity as tolerated; Muscle relaxants

What are some imaging tests to consider in LBP?
- Plain spinal films—concern of fracture; CT—superior for vertebral fractures; MRI—for emergent conditions

List some laboratory tests obtained for LBP that are possibly caused by an infection or tumor?
- CBC, ESR/C-reactive protein (CRP), and urinalysis

What are some important points to consider in each of the “can’t miss” diagnosis?

Metastasis  
- Often older then 50 with hx of cancer;
- Often >1 month of weight loss and LBP; Often requires a variety of imaging tests
Spinal epidural abscess  
Immunocompromised and IVDA at risk; Often have fever and local spine tenderness; Focal neurological deficit not uncommon; Broad-spectrum Abx/neurosurgery consult

Disc herniation  
Common in >30 years with progressive LBP; Sciatica and L4-L5 involvement common; Treat conservatively; Neurosurgery consult if evidence of cord compression

Vertebral fracture  
Often history of trauma or mets; Sudden onset of pain and neurological deficits; Imaging is important for further evaluation

Cauda equina syndrome  
Often in those with mets or hx of trauma; Incontinence/saddle paresthesias common; MRI test of choice; Neurological emergency

**SYNCOPE**

What is the definition of syncope?  
Abrupt/transient loss of consciousness associated with absence of postural tone, followed by a rapid and usually complete recovery

List important conditions that should be considered for each category:

- **Cardiovascular**  
Dysrhythmias; Obstruction (i.e., aortic stenosis); Myocardial infarction

- **Neurologic**  
Seizure; Subarachnoid hemorrhage; Posterior circulation infarct

- **Medication**  
Diuretics; Beta-blockers; Nitrates

- **Miscellaneous**  
Vasovagal; Carotid sinus hypersensitivity

What are important elements in the history to gather to help determine cause of syncope?  
Events prior to the episode; Any associated pain (HA/chest/abdominal pain); Diaphoresis and emesis; Exertion; Dyspnea
What are some findings to look for on physical exam?

Carotid bruits; Cardiac murmurs; Evidence of bleeding (i.e., GI bleed); Pulsatile abdominal mass; Adnexal tenderness (i.e. ectopic)

What is an important diagnosis to consider in the following scenario of a patient who presents with syncope and the following associated symptom:

A 21-year-old healthy male presents to the ED after passing out during soccer practice. Family history is significant for an uncle who died from sudden death at 27

Hypertrophic cardiomyopathy

A 31-year-old female presents to the ED after a syncopal episode while taking care of her kids. Her physical exam is significant only for right adnexal tenderness

Ectopic pregnancy

A 17-year-old female with no past medical history presents to the ED after passing out while giving blood at Red Cross. Observers noted she seemed diaphoretic and nauseous prior to passing out

Vasovagal

A 65-year-old male with history of hypertension, dyslipidemia, and CAD presents after passing out. His physical exam is significant for abdominal tenderness and bruits

Abdominal aortic aneurysm

A 24-year-old female is brought in by emergency medical service (EMS) when she was observed to pass out at the mall soon followed by rhythmic movements of her extremities. Physical exam is significant for lateral tongue bites

Seizure

A 62-year-old male is brought from home by his wife after he passed out. His history is only significant for HTN and DM. She mentioned he seemed diaphoretic prior to the event and also missed breakfast

Hypoglycemia

What are some considerations in the evaluation of syncope?

To separate benign from serious causes; A careful history and physical is paramount; Initial ECG is also the mainstay in evaluation
What is an important point to keep in mind about syncope? Although most cases of syncope are benign, syncope may be an initial symptom of something life-threatening such as AAA or SAH

What patients are often admitted for syncope? Elderly patients with many comorbidities; Syncope with worse HA, pelvic pain, etc.; Risk for fall and injury (typically elderly)

Which patients are typically safe to discharge? No evidence of structural heart defects

**CLINICAL VIGNETTES**

<table>
<thead>
<tr>
<th>Case Study</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>55-year-old-female with an hx of breast cancer presents with radicular pain of her legs, urinary retention, and lower back pain; PE: saddle anesthesia and absent ankle jerk reflexes</td>
<td>Cauda equine syndrome</td>
</tr>
<tr>
<td>71-year-old-female presents with a gradual decline in memory and having increasing difficulties with normal day-to-day routine, often getting lost when she walks back home; Head CT: diffuse cortical atrophy</td>
<td>Alzheimer’s disease</td>
</tr>
<tr>
<td>51-year-old-male with hx of heart disease presents with a sudden onset of left-sided extremity weakness that has not resolved; PE: flaccidity of left arms and leg along with Babinski (+) on left; head CT: normal</td>
<td>Right MCA cerebrovascular accident</td>
</tr>
<tr>
<td>16-year-old-male presents with a headache, nausea, and vomiting soon after being struck in the side of the head during a bar fight; head CT: lens-shaped, left-sided hyperdense mass near the temporal bone</td>
<td>Epidural hematoma</td>
</tr>
<tr>
<td>31-year-old-female with hx of gastroenteritis 1 week ago now presents with symmetric ascending weakness of her legs and paresthesias; PE: diminished reflexes; LP: above normal protein</td>
<td>Guillain-Barré syndrome</td>
</tr>
<tr>
<td>29-year-old-female presents with slow onset of paresthesias, diplopia, numbness of left upper extremity; MRI: discrete areas of periventricular demyelination</td>
<td>Multiple sclerosis (MS)</td>
</tr>
</tbody>
</table>
27-year-old-female with a long hx of headaches presents with a unilateral headache, nausea, vomiting, and photophobia that typically occurs during her menstrual period; head CT: normal

65-year-old-male with an hx of colon CA presents with an insidious onset of disequilibrium and dizziness that has been present for months; PE: vertical nystagmus and ataxia

72-year-old-male presents with loss of short-term memory, urinary incontinence, and dementia; PE: wide-based gait; head CT: massively dilated ventricular space

43-year-old-male with a long hx of alcohol abuse presents with psychosis and ataxia; brain MRI: mamillary body atrophy and diffuse cortical atrophy

12-year-old is referred to the ED from school due to frequent brief lapses of consciousness with slight limb jerking; PE: during exam, patient again has his brief lapse of consciousness with rapid eye blinking

23-year-old-female is brought in via EMS, per report, patient had loss of consciousness (LOC) with loss of postural control followed by tonic phase of contractions with clonic limb jerking; PE: patient in now awake, but minimally responsive

24-year-old-college student presents with a high-grade fever, headache, and neck stiffness; PE: Kernig’s sign and nuchal rigidity; LP: decreased glucose, increased protein, and high polymorphonuclear leukocytes

31-year-old-male presents with unilateral boring periorbital headache with periods of multiple headaches alternating with symptom-free intervals; PE: ipsilateral tearing and conjunctival injection

Migraine headache

Central vertigo

Normal pressure hydrocephalus

Wernicke’s encephalopathy

Absence seizure

Tonic-clonic seizure

Bacterial meningitis

Cluster headache
BASIC OPHTHALMOLOGY

What are the two chambers of the aqueous part of the eye called?
1. Anterior chamber
2. Posterior chamber

What is the jelly-like substance in the back part of the eyeball which provides shape and is relatively inert?
Vitreous humor

What are some components that make up the anterior segment of the eye?
Cornea; Conjunctiva; Anterior chamber; Lens; Iris; Ciliary body

What components make up the fundus of the eye?
Macula; Optic nerve; Retina

Please define the following forms:

Anisocoria
Unequal pupil size under equal lighting

Hyphema
Red blood cells in the anterior chamber

Hypopyon
White blood cells in the anterior chamber

Limbus
Circumferential border of the cornea and white sclera

Tonopen
Pen-shaped device to measure intraocular pressure

What are some important elements in the history that should be obtained in any general eye exam?
History of diabetes or hypertension; Use of contact lenses (i.e., extended wear); Past visual acuity; Occupation
What are eight components of the eye exam that should be obtained with all eye complaints?

1. Visual acuity
2. External eye
3. Pupils
4. Confrontation of visual fields
5. Extraocular movement
6. Fundus examination
7. Anterior segment
8. Intraocular pressure

TRAUMA OF THE EYE

Corneal Foreign Bodies

What is important to confirm during examination of the eye with regards to a foreign body?
Assess if superficial penetration versus full-thickness injury

What is the best way to assess foreign body depth in the emergency department (ED)?
Slit-lamp exam

Can a superficial corneal foreign body be removed in the ED?
Yes—under best magnification available

What are some key steps in the removal of a superficial corneal foreign body?
Instill topical anesthetics in both eyes; Use slit-lamp magnification; Can use a 30-gauge needle to remove or a moistened cotton-tipped applicator; Most superficial objects can be removed

What are some key steps in the removal of a full-thickness foreign body?
Do not remove in the ED—should be done by ophthalmology

What is an additional concern if a foreign body is metallic?
Metallic bodies can leave behind rust rings that are toxic to the cornea

Should rust rings be removed in the ED?
Can be removed with an ophthalmic burr, but only the superficial layer

A corneal abrasion will be present after foreign body removal, what are some treatments for it?
Antibiotic ointment; Cycloplegia; Referral to ophthalmology

Corneal Abrasions

What should always be done as part of an eye exam with corneal abrasions?
Check under the eyelids

What is usually done for conjunctival abrasions?
Erythromycin drops; Ensure no other ocular injuries
What are some clinical features of corneal abrasions?
Photophobia, tearing, and eye pain

What are two common causes of corneal abrasions?
1. Trauma
2. Use of contact lenses

What is typically a limiting factor to do a complete eye exam?
Patient is typically in extreme discomfort

What is an effective way to reduce pain?
Adequate cycloplegia

What is the optimal way to visualize corneal abrasions?
Fluorescein staining with cobalt-blue lighting

What is an effective long-acting cycloplegia for large or very painful abrasions?
Scopolamine

What are some key points in the management of corneal abrasions?
Adequate pain control with cycloplegias; Erythromycin drops; Abrasion typically heal without problems

What is a particular concern of corneal abrasions from contacts?
Pseudomonas infections

What other antibiotic ointment should be added if concerned about Pseudomonas infection?
Tobramycin or fluoroquinolone drops

Should patients be sent home with topical anesthetics for pain control?
No—can cause corneal toxicity if improperly dosed

Subconjunctival Hemorrhage

What is the mechanism by which a subconjunctival hemorrhage occurs?
Rupture of conjunctival vessels

What are some common causes of a subconjunctival hemorrhage?
Trauma; Hypertension; Sudden Valsalva (i.e., coughing)

What is the treatment of choice for a subconjunctival hemorrhage?
Nothing—will resolve in 1–2 weeks

Chemical Injuries

What is the most important point to remember about ocular chemical injuries?
True ocular emergency

What is considered a more devastating injury: acidic or alkali?
Alkali burns as they penetrate deeper

What are some common causes of alkali burns?
Ammonia; Lye; Industrial solvents
What is the immediate management of ocular chemical injuries?
Topical anesthetic; Placement of Morgan lens; Copious irrigation with 1–2 L of NS

When should the copious irrigation be stopped?
Once pH of the tears is near normal (7.5–8)

What are some long-term complications of chemical burns?
Symblepharon; Cataracts; Scarring/neovascularization of the cornea

When should patients be referred to ophthalmology?
Corneal clouding; Epithelial defect

Assuming there are no corneal clouding or anterior chamber findings, what is the general disposition?
Erythromycin drops; Cycloplegics for pain control; Ophthalmologic follow up within 2 days

Blunt Injuries

What is important to assess after blunt injury to the eye?
Vision and globe integrity

What is an important diagnosis to consider in any blunt trauma to the eye?
Ruptured globe

What are some clinical features of a ruptured globe?
Obvious full-thickness laceration, blindness, flat anterior chamber, irregular pupil, and hyphema

What are common causes of a ruptured globe?
Penetrating injuries (i.e., bullets); Blunt trauma

What is important not to do during an eye exam if a ruptured globe is suspected?
Checking intraocular pressure (IOP)

What are some key points in the management of a ruptured globe?
Avoid any pressure on the globe; Place a metal eye shield; Update tetanus status; Consider antibiotic use depending on object; Consultation with ophthalmology

What is a hyphema?
Blood in the anterior chamber of the eye

What are some common causes of hyphema?
Trauma (blunt or penetrating); Spontaneous (esp. sickle-cell disease)

What vessel is typically responsible for a hyphema?
Iris root vessel

What is also important to assess in a patient with a hyphema?
Any other associated trauma such as a ruptured globe
Why is it recommended to dilate the pupil? To avoid pupillary movement which may increase bleeding from an iris root vessel.

What is an important complication of hyphema? Increased IOP

What is the general disposition of patients with hyphema? Consultation with ophthalmology; Elevate patient’s head; Administer dilating agent (i.e., atropine); Treat significant IOP increase

How is increased IOP typically treated? Topical beta-blocker; Topical alpha-adrenergic agonists; (IV) Intravenous mannitol

What is an important diagnosis to consider in a patient with blunt trauma and inability to gaze upward? Orbital blowout fracture

What is the most frequent site of an orbital blowout fracture? Inferior-medial wall

How is the diagnosis of an orbital blowout fracture made? CT (axial and coronal scans)

What is the general disposition of an isolated orbital blowout fracture? Referral for surgery within 3–9 days

What are other important injuries to examine for with orbital blowout fractures? Hyphema; Abrasions; Traumatic iritis; Retinal detachment

### INFECTIONS OF THE EYE

#### Conjunctivitis

What is common element in the history of a patient with viral conjunctivitis? Preceding upper respiratory infection

What are some clinical features of viral conjunctivitis? May initially have one eye involvement with watery discharge, reddened conjunctiva, and often normal cornea

What is the primary reason the cornea should be stained? Avoid missing a herpes dendritic keratitis

What are some key points in the management of viral conjunctivitis? Typically self-limiting (1–3 weeks); Highly contagious; Naphcon-A for congestion/itching; Consider topical antibiotic in suspected bacterial conjunctivitis
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some clinical features of bacterial conjunctivitis?</td>
<td>Mucopurulent discharge, inflammation of eye, and often a history of exposure to someone with viral conjunctivitis</td>
</tr>
<tr>
<td>What should be done to avoid missing a corneal abrasion or ulcer?</td>
<td>Fluorescein staining</td>
</tr>
<tr>
<td>What is the treatment of choice for patients with bacterial conjunctivitis?</td>
<td>Broad-spectrum topical antibiotic</td>
</tr>
<tr>
<td>What is a special consideration for contact lens-wearing patients with bacterial conjunctivitis?</td>
<td><em>Pseudomonas</em> infection</td>
</tr>
<tr>
<td>What topical antibiotic should be used for contact lens-wearers with bacterial conjunctivitis?</td>
<td>Topical aminoglycoside or fluoroquinolone</td>
</tr>
<tr>
<td>What parts of the eye can be affected by herpes simplex virus (HSV)?</td>
<td>Conjunctiva; Cornea; Lids</td>
</tr>
<tr>
<td>What does fluorescein staining typically show with HSV involvement of the eye?</td>
<td>Linear branching pattern with terminal bulbs</td>
</tr>
<tr>
<td>What is an important concern with HSV keratitis?</td>
<td>Corneal scarring</td>
</tr>
<tr>
<td>What should be avoided with HSV keratitis?</td>
<td>Topical steroids</td>
</tr>
<tr>
<td>How is HSV keratitis commonly treated?</td>
<td>Viroptic drops (i.e., longer if cornea involved); Erythromycin drops to avoid secondary bacterial involvement</td>
</tr>
<tr>
<td>What is herpes zoster ophthalmicus (HZO)?</td>
<td>Shingles of CN V with involvement of eye</td>
</tr>
<tr>
<td>What is Hutchinson’s sign?</td>
<td>Cutaneous lesions of the tip of the nose</td>
</tr>
<tr>
<td>What are some clinical features of HZO?</td>
<td>Iritis with pain and photophobia with possible cutaneous lesions</td>
</tr>
<tr>
<td>How is HZO commonly treated?</td>
<td>Topical steroids for iritis; Topical cycloplegic agents for pain; Consider IV acyclovir; Distinguish from primary HSV infection</td>
</tr>
</tbody>
</table>

**Corneal Ulcer**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is a corneal ulcer?</td>
<td>Serious infection involving multiple layers of the cornea</td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>What is the pathophysiology of a corneal ulcer?</td>
<td>Break in the epithelial layer that allows bacteria to invade the corneal stroma</td>
</tr>
<tr>
<td>What are some causes of corneal epithelial break?</td>
<td>Trauma; Contact lenses; Incomplete lid closure</td>
</tr>
<tr>
<td>What are some clinical features of a corneal ulcer?</td>
<td>Eye pain, photophobia, tearing, and redness</td>
</tr>
<tr>
<td>What does a slit-lamp exam commonly reveal in a corneal ulcer?</td>
<td>Staining shows epithelial defect with underlying infiltrate as well as possible hypopyon</td>
</tr>
<tr>
<td>How are corneal ulcers commonly treated?</td>
<td>Topical aminoglycoside or fluoroquinolone; Topical cycloplegic for pain; Ophthalmology follow-up within 24 hours</td>
</tr>
<tr>
<td><strong>Periorbital/Orbital Cellulitis</strong></td>
<td></td>
</tr>
<tr>
<td>What is periorbital cellulitis?</td>
<td>Superficial cellulitis of the periorbital area</td>
</tr>
<tr>
<td>What are some clinical features of periorbital cellulitis?</td>
<td>Surrounding area of the eye (i.e., eyelid) is red, warm, and edematous with no involvement of the eye itself</td>
</tr>
<tr>
<td>What is the most common organism involved with periorbital cellulitis?</td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>What is the typical management for periorbital cellulitis without eye involvement?</td>
<td>Oral antibiotic is sufficient</td>
</tr>
<tr>
<td>What is a special concern of periorbital cellulitis in young children?</td>
<td>High risk of bacteremia and meningitis</td>
</tr>
<tr>
<td>What are some key points in the management of young children with periorbital cellulitis?</td>
<td>Full evaluation with Abx and blood cultures</td>
</tr>
<tr>
<td>What is orbital cellulitis?</td>
<td>Potentially life-threatening orbital infection that lies deep to the orbital septum</td>
</tr>
<tr>
<td>What are some common organisms to consider in orbital cellulitis?</td>
<td><em>S. aureus</em>; <em>Haemophilus influenzae</em> (in children); <em>Mucormycosis</em> (in immunocompromised patients)</td>
</tr>
<tr>
<td>What is the most common source of orbital cellulitis?</td>
<td>Paranasal sinus</td>
</tr>
<tr>
<td>What are some clinical features of orbital cellulitis?</td>
<td>Fever, pain, extraocular muscle (EOM) impairment, proptosis, decreased visual acuity</td>
</tr>
</tbody>
</table>
### Hordeolum

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is an external hordeolum (stye)?</td>
<td>Acute infection of an oil gland associated with an eyelash</td>
</tr>
<tr>
<td>What is the most common organism involved with a stye?</td>
<td><em>S. aureus</em></td>
</tr>
<tr>
<td>What is the typical appearance of a stye?</td>
<td>A small pustule at the lash line</td>
</tr>
<tr>
<td>What is treatment of a stye?</td>
<td>Warm compresses with erythromycin ointment</td>
</tr>
<tr>
<td>What is a internal hordeolum known as?</td>
<td>Chalazion</td>
</tr>
<tr>
<td>What is a chalazion?</td>
<td>Acute or chronic inflammation of the eyelid commonly due to blockage of an oil gland</td>
</tr>
<tr>
<td>What is the appearance of a chalazion?</td>
<td>Tender red lump at the lid, cystic mass can occur with recurrent chronic inflammation</td>
</tr>
<tr>
<td>What is the treatment of an acute chalazion?</td>
<td>Warm compresses with erythromycin ointment; Consider doxycycline if chronic inflammation</td>
</tr>
</tbody>
</table>

### ACUTE VISUAL LOSS

#### Central Retinal Artery Occlusion

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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</thead>
<tbody>
<tr>
<td>What vessel provides blood supply to the inner retina?</td>
<td>Central retinal artery from the ophthalmic artery</td>
</tr>
<tr>
<td>What are some clinical features of central retinal artery occlusion (CRAO)?</td>
<td>Sudden, painless, and profound monocular loss of vision</td>
</tr>
<tr>
<td>What is a possible warning symptom of a CRAO?</td>
<td>Amaurosis fugax</td>
</tr>
<tr>
<td>What is the definition of amaurosis fugax?</td>
<td>Loss of vision in one eye caused by a temporary lack of blood flow to the retina</td>
</tr>
<tr>
<td>What are some important causes of CRAO?</td>
<td>Giant cell arteritis; Embolus; Sickle-cell disease; Thrombosis; Trauma</td>
</tr>
<tr>
<td>What is the most common cause of CRAO?</td>
<td>Embolus (i.e., atrial fibrillation)</td>
</tr>
</tbody>
</table>
How long does it take before irreversible damage to the retina can occur? 60–90 minutes

What is the main focus in treatment of CRAO? Dislodging the embolus

What are some key points in the management if CRAO? Initiate treatment as rapidly as possible; Ocular massage (attempt to dislodge the embolus); Acetazolamide and topical beta-blockers; Immediate ophthalmology consultation

Central Retinal Vein Occlusion

What is typically the mechanism of central retinal vein occlusion (CRVO)? Thrombosis of the central retinal vein

Name some conditions that are associated with CRVO. Glaucma; Hypertension; Hypercoagulable disorders

What are some clinical features of CRVO? Acute, painless, and monocular involvement with variable vision loss

What is the typical funduscopic finding in CRVO? Diffuse retinal hemorrhage in all quadrants; Optic disc edema

What is the typical treatment option for CRVO? Ophthalmology consultation; May consider giving aspirin

Narrow-Angle Glaucoma

Is a history of glaucoma common in patients who present with narrow-angle glaucoma? No—patients will typically have an undiagnosed narrow anterior chamber angle

What is the mechanism by which aqueous humor is produced? Aqueous humor is produced in the ciliary body from the posterior chamber which flows through the pupil and into the anterior chamber, where it is reabsorbed

What is the pathophysiology of narrow-angle glaucoma? When the pupil becomes mid-dilated, the lens touches the iris leflet, blocking the flow of aqueous humor and causing an increase in IOP, causing the cornea to become edematous and distorted

What are some clinical features of narrow-angle glaucoma? Headache, eye ache, cloudy vision, nausea/vomiting, and increased IOP
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>How high can the IOP be in narrow-angle glaucoma?</td>
<td>Higher than 50 mm Hg</td>
</tr>
<tr>
<td>What is the typical finding on exam of the pupil?</td>
<td>Mid-dilated and nonreactive</td>
</tr>
<tr>
<td>What is the focus of treating narrow-angle glaucoma?</td>
<td>Quickly lowering IOP and decrease production of aqueous humor</td>
</tr>
<tr>
<td>What are some agents commonly used to suppress aqueous humor production?</td>
<td>Topical beta-blockers and alpha-agonists; Acetazolamide</td>
</tr>
<tr>
<td>What is another agent to consider that is effective in lowering IOP?</td>
<td>Mannitol</td>
</tr>
<tr>
<td>What agent is commonly used to constrict the pupil once the IOP has been reduced?</td>
<td>Pilocarpine (will not typically work during an acute attack)</td>
</tr>
<tr>
<td>What is the definitive treatment for narrow-angle glaucoma?</td>
<td>Peripheral laser iridectomy</td>
</tr>
</tbody>
</table>

**Optic Neuritis**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is optic neuritis?</td>
<td>Optic nerve dysfunction that is the most common cause of acute reduction of vision</td>
</tr>
<tr>
<td>What are some causes of optic neuritis?</td>
<td>Ischemia; Embolus; Nerve compression; Multiple sclerosis (Ms); Lupus</td>
</tr>
<tr>
<td>What are some clinical features of optic neuritis?</td>
<td>Rapid and painful reduction of visual acuity, but more commonly affects color vision and afferent pupillary defect</td>
</tr>
<tr>
<td>What test is useful to detect alteration in color vision?</td>
<td>Red Desaturation test</td>
</tr>
<tr>
<td>How is the Red Desaturation test carried out?</td>
<td>Have the patient look at a red object with each eye individually, the affected eye will often see the red object as pink or lighter</td>
</tr>
<tr>
<td>What is a possible finding on funduscopic exam of a patient with optic neuritis?</td>
<td>Optic disc is swollen (anterior neuritis)</td>
</tr>
<tr>
<td>In what case will the optic disc be normal during a funduscopic exam?</td>
<td>Retrobulbar neuritis</td>
</tr>
<tr>
<td>What is the typical disposition of patients with optic neuritis?</td>
<td>Discuss with ophthalmology on the use of steroids and follow-up</td>
</tr>
</tbody>
</table>
CLINICAL VIGNETTES

26-year-old male steel worker presents to the ED with right eye pain and blurring vision; eye exam: small metallic flecks in the cornea

Corneal foreign body

18-year-old female with no PMH presents with a 1 week history of tearing, photophobia, and left eye pain. She does have a history of sleeping with her contacts on; eye exam: fluorescein staining with cobalt-blue lighting

Corneal abrasions

23-year-old male with no PMH presents with a sudden onset of blood visible in the right eye, patient does not complain of any vision problems or pain, but is otherwise very concerned

Subconjunctival hemorrhage

41-year-old female chemist presents to the ED with recent history of lye splashing in her eyes. What is the most crucial aspect in management?

Copious irrigation

26-year-old male presents to the ED soon after being hit directly in his right eye with a baseball during a game. He now complains of pain and blindness; eye exam: irregular pupil, hyphema, and flat anterior chamber

Ruptured globe

3-year-old female is brought in by her mother due to concerns of bilateral red eyes with watery discharge. Significant history includes day care three times a week

Viral conjunctivitis

19-year-old college student with eye history of contact lens use presents with inflammation of her eyes along with mucopurulent discharge; eye exam: unremarkable for fluorescein staining

Bacterial conjunctivitis

55-year-old male presents with inflammation of the eyes with watery discharge for about 3 days; eye exam: linear branching pattern with fluorescein staining

HSV keratitis

21-year-old male with recent eye injury presents to the ED with left eye pain, photophobia, redness, and tearing for 2 days; eye exam: hypopyon and staining that shows epithelial defects

Corneal ulcers
61-year-old diabetic male presents with surrounding area of redness and edema around his left eye that is warm to the touch; eye exam: normal

24-year-old male presents to the ED due to concern of a small growth around his upper eyelash, but otherwise has no changes in vision; eye exam: remarkable for a small pustule at the lash line

61-year-old female with an Hx of comorbid disease (CAD), DM, atrial fibrillation (afib), and cerebral vascular accident (CVA) presents with sudden and painless loss of vision in her left eye

67-year-old female with history of DM presents with a pounding headache, cloudy vision, nausea, and eye pain soon after coming out from the movies; eye exam: mid-dilated and nonreactive pupil with IOP >50 mm Hg

Periorbital cellulitis

Stye

CRAO

Acute angle-closure glaucoma
# ENT and Dental Emergencies

## ACUTE OTITIS MEDIA

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important things to know about acute otitis media (AOM)?</td>
<td>Most frequent diagnosis in sick children; The highest incidence between 6–24 months of age; Often occur during winter/spring after an upper respiratory infection (URI)</td>
</tr>
<tr>
<td>What are some risk factors associated with the development of AOM?</td>
<td>Age; Day care; Second-hand smoke; Altered host defense</td>
</tr>
<tr>
<td>What is the pathogenesis of AOM?</td>
<td>Obstruction of the eustachian tube that results in a sterile effusion with aspiration of nasopharyngeal secretions into the middle ear that can result in acute infection</td>
</tr>
</tbody>
</table>
| What are the three most common bacterial pathogens involved in AOM?      | 1. *Streptococcus pneumoniae*  
2. *Haemophilus influenzae*  
3. *Moraxella catarrhalis*                                                                 |
| What are some clinical features of AOM?                                 | Examination of the ear often shows distortion of the tympanic membrane (TM), erythema, decreased motility of TM on pneumatic otoscopy, and fever |
| What are some complications to consider in otitis media if left untreated? | Hearing loss, TM perforation, mastoiditis, lateral sinus thrombosis, and meningitis                                                              |
| What is the most reliable sign of AOM?                                  | Decreased motility of the TM on pneumatic otoscopy                                                                                               |
| What is the first-line treatment for AOM?                               | Amoxicillin                                                                                                                                       |
| What are two other drugs to consider in penicillin-allergic patients?   | 1. Erythromycin  
2. Trimethoprim-sulfamethoxazole                                                                               |
### AOM (Acute Otitis Media)

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some key points in the management of AOM?</td>
<td>Local heat application for relief; Antibiotic for treatment; Return if AOM does not improve within 48 hours</td>
</tr>
<tr>
<td>What is the definition of bullous myringitis?</td>
<td>Inflammation of the TM with bullae that are present on the TM (typically more painful)</td>
</tr>
<tr>
<td>What agents are often associated with bullous myringitis?</td>
<td>Mycoplasma or viral infection</td>
</tr>
<tr>
<td>What is the treatment for bullous myringitis?</td>
<td>Macrolide antibiotics; Topical Auralgan for intact TM; ENT followup as needed</td>
</tr>
</tbody>
</table>

### OTITIS EXTERNA (SWIMMER’S EAR)

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of otitis externa?</td>
<td>Inflammation of the external auditory canal or auricle typically due to infection, allergic reaction, or dermal disease</td>
</tr>
<tr>
<td>What are some inherent defenses that contribute to protection against infection?</td>
<td>Hair follicles; Tragus and conchal cartilage; Cerumen</td>
</tr>
</tbody>
</table>
| What are the two most common organisms associated with otitis externa?   | 1. *Pseudomonas aeruginosa*  
2. *Staphylococcus aureus*                                                                |
| What are some risk factors that contribute to the development of otitis externa? | Warm, moist environment (i.e., swimming); Excessive cleaning; Devices that occlude the auditory canal |
| What are some clinical features of otitis externa?                      | Pain, itching, fullness of ear, redness or swelling of external ear, and cheesy or purulent green discharge                          |
| What are some features of severe cases of otitis externa?               | Complete obstruction of canal due to edema, auricular erythema, adenopathy, and fever                                                  |
| What are some key points in the management of otitis externa?           | Clean the canal thoroughly; Control pain; Topical agents in mild cases (i.e., Cortisporin); Antibiotic in more severe cases     |
| What is the definition of necrotizing otitis externa?                  | Serious complication of acute bacterial otitis externa where infection spreads from the skin to the soft tissue, cartilage, and bone of the temporal region and skull base |
| What population is more commonly affected by necrotizing otitis externa? | Elderly; Diabetic; Immunocompromised |
| What is the mortality rate of necrotizing otitis externa if left untreated? | Approaches up to 50% |
| What are some clinical features of necrotizing otitis externa? | Otorrhea, pain that is out of proportion to the exam, granulation tissue at the bony cartilaginous junction of the ear canal floor, and cranial nerve palsies |
| What are some key points in the management of necrotizing otitis externa? | Intravenous (IV) antibiotics; ENT consult; Possible surgical debridement; MRI/CT diagnostic test of choice to visualize complications if needed |

## ACUTE HEARING LOSS

| What are the three components of the ear? | 1. Outer ear: auricle and ear canal 2. Middle ear: TM and ossicles 3. Inner ear: cochlea and semicircular canals |
| How is hearing loss classified? | Conductive; Sensorineural; Mixed |
| What areas of the ear often result in conductive hearing loss if damaged? | External auditory canal; Tympanic membrane; Middle ear components (i.e., ossicles) |
| What are some important causes of conductive hearing loss? | Middle ear effusion; TM perforation; Otitis externa; Foreign body impaction |
| What areas of the ear often result in sensorineural hearing loss if damaged? | Cochlea; Auditory nerve; Inner ear |
| What are some important causes of sensorineural hearing loss? | Acoustic neuroma; Viral neuritis; Temporal bone fracture; Presbycusis |
| What are some common causes of bilateral sensorineural hearing loss? | Exposure to loud noise; Antibiotics (i.e., aminoglycosides); Nonsteroidal anti-inflammatory drugs (NSAIDs); Loop diuretics |
| What are important elements in the exam to evaluate acute hearing loss? | History of vertigo and tinnitus; Cranial nerve examination; Thorough otoscopic exam; CT if any suspicion of tumor |
### What two tests are useful to distinguish sensorineural from conductive hearing loss?

1. **Weber test**
2. **Rinne test**

### What is the Weber test?

- Tuning fork is struck and placed on the patient's forehead. The patient is asked to report in which ear the sound is heard loudest.

### In a patient with unilateral conductive hearing loss, in which ear would the sound be loudest in a Weber test?

- A patient would hear the tuning fork loudest in the affected ear.

### In a patient with unilateral sensorineural hearing loss, in which ear would the sound be loudest in a Weber test?

- A patient would hear the tuning fork loudest in the unaffected ear.

### How is a Rinne test done?

- This is achieved by placing a vibrating tuning fork (512 Hz) initially on the mastoid, then next to the ear and asking which sound is loudest.

### What are some possibilities with the Rinne test?

- In a normal ear, air conduction (AC) is better than bone conduction (BC); in conductive hearing loss, BC is better than AC; in sensorineural hearing loss, BC and AC are both equally depreciated, maintaining the relative difference of AC > BC.

### What are some key points in the management of acute hearing loss?

- Primarily depends on the cause; foreign body should be removed; offending medication should be discontinued; tumors require admission/consultation.

---

### NASAL

#### Nasal Trauma

<table>
<thead>
<tr>
<th>What is a common diagnosis in any nasal trauma?</th>
<th>Nasal fracture</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some clinical features of nasal fractures?</td>
<td>Deformity, nasal swelling, ecchymosis, tenderness, or crepitence</td>
</tr>
<tr>
<td>What role does x-ray play in the evaluation of uncomplicated nasal fractures?</td>
<td>Not commonly used</td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>What are some key points in the management of uncomplicated nasal fractures?</td>
<td>Early reduction if swelling is not severe; Delay reduction (2–3 days) if severe swelling; Reevaluation after edema has resolved</td>
</tr>
<tr>
<td>What are some examples of complicated nasal fractures?</td>
<td>Other facial fractures (i.e., orbital floor); Nasoethmoid fracture</td>
</tr>
<tr>
<td>What is the test of choice to further evaluate complicated nasal fractures?</td>
<td>CT</td>
</tr>
<tr>
<td>What are some possible indications for the use of prophylactic antibiotics?</td>
<td>Use of nasal packing; Laceration of nasal mucosa; Immunocompromised</td>
</tr>
<tr>
<td>What is another major complication of nasal trauma?</td>
<td>Septal hematoma</td>
</tr>
<tr>
<td>What are some clinical features of a septal hematoma?</td>
<td>Bluish-purple swelling of the nasal septum</td>
</tr>
<tr>
<td>What are some key points in the management of a septal hematoma?</td>
<td>Vertical incision of the hematoma; Pack the anterior nasal cavity; Antibiotic coverage (Staph coverage); ENT follow-up</td>
</tr>
<tr>
<td>What is the consequence of failure to drain a septal hematoma?</td>
<td>Avascular necrosis; Septal abscess</td>
</tr>
<tr>
<td>What is the common deformity that occurs due to avascular necrosis of the nasal septum?</td>
<td>Saddle-nose deformity</td>
</tr>
<tr>
<td>What can occur if the cribiform plate is fractured?</td>
<td>Cerebrospinal fluid (CSF) rhinorrhea</td>
</tr>
<tr>
<td>What is the timeline for when this can occur?</td>
<td>CSF rhinorrhea may not occur until weeks after the cribiform fracture</td>
</tr>
<tr>
<td>What is a common clinical scenario when this can occur?</td>
<td>Typically occurs in the setting of a facial trauma followed by clear nasal discharge that can be associated with anosmia and headache</td>
</tr>
<tr>
<td>What diagnostic test can be used to detect cribiform plate fracture?</td>
<td>Plain radiograph facial series</td>
</tr>
<tr>
<td>What are some things to do if one suspects CSF rhinorrhea?</td>
<td>Keep the patient upright; Avoid coughing/sneezing; Consult a neurosurgeon</td>
</tr>
<tr>
<td>What is the major concern of CSF rhinorrhea in regard to infections?</td>
<td>Meningitis</td>
</tr>
<tr>
<td>What role do antibiotics play in regard to CSF rhinorrhea?</td>
<td>Controversial—use in consultation with neurosurgery</td>
</tr>
</tbody>
</table>
### Nasal Foreign Bodies

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What age group do nasal foreign bodies occur in?</td>
<td>Children 2–4 years of age</td>
</tr>
<tr>
<td>What is the common clinical presentation of a child with a nasal foreign body?</td>
<td>Unilateral foul-smelling nasal discharge or persistent epistaxis</td>
</tr>
<tr>
<td>In many cases, can a history of an object being inserted into the nares be recalled?</td>
<td>No</td>
</tr>
<tr>
<td>How is the diagnosis of a nasal foreign body commonly made?</td>
<td>Inspection of nares with nasal speculum or otoscope</td>
</tr>
<tr>
<td>What are some commonly used methods to remove a nasal foreign body?</td>
<td>Forceps, wire loops, or right angle probes; Suction catheter; Positive pressure (i.e., blow via nose)</td>
</tr>
<tr>
<td>What is typically done if the foreign object cannot be removed?</td>
<td>ENT follow-up within 24 hours (most can be done as outpatient)</td>
</tr>
<tr>
<td>What are some indications for admission for immediate nasal foreign body removal?</td>
<td>Associated infections (i.e., facial cellulites); Sharp objects; Button batteries</td>
</tr>
</tbody>
</table>

### Epistaxis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is more common: anterior nosebleeds or posterior nosebleeds?</td>
<td>Anterior nosebleeds (90% of cases)</td>
</tr>
<tr>
<td>What is the most common source of anterior nosebleeds?</td>
<td>Kiesselbach’s plexus</td>
</tr>
<tr>
<td>What age group is commonly affected with anterior nosebleeds?</td>
<td>Children and young adults</td>
</tr>
<tr>
<td>What are some important causes of anterior nosebleeds to consider?</td>
<td>Foreign body; Trauma; Nose picking; Blood dyscrasias; Infections</td>
</tr>
<tr>
<td>What are some important elements in the history to consider with respect to anterior nosebleeds?</td>
<td>Recurrent; Onset; Duration; Medication; Illicit drug; Underlying medical problems</td>
</tr>
<tr>
<td>What are some important elements in the physical to focus on?</td>
<td>Vitals (i.e., orthostatics); Evidence of coagulopathy (i.e., bruising); Location (anterior versus posterior)</td>
</tr>
<tr>
<td>What simple thing can be done prior to further evaluation of nosebleeds?</td>
<td>Apply a topical vasoconstrictor/anesthetic; Pinch nose firmly and keep head forward</td>
</tr>
<tr>
<td>What are some commonly used methods to gain hemostatic control of anterior nosebleeds?</td>
<td>Silver nitrate sticks (cautery); Anterior nose packing; Piece of hemostatic material (i.e., Gelfoam)</td>
</tr>
</tbody>
</table>
What is the most common source of posterior nosebleeds? Sphenopalatine artery (arterial source); Woodruff’s plexus (venous source)

What age group is commonly affected with posterior nosebleeds? Elderly

What are some important causes of posterior nosebleeds to consider? Cancer; Coagulopathy

What are some key points in the management of posterior nosebleeds? Particular importance on airway; Posterior packing with premade posterior nasal-packing balloon; Admit with ENT consultation

How is posterior packing commonly done? Use gauze pack with an intranasal balloon device or Foley catheter

What are some important complications of epistaxis? Severe bleeding; Airway obstruction from bleeding; Sinusitis; AOM

ENT INFECTIONS

Pharyngitis

What is the definition of pharyngitis? Inflammation of the mucous membrane of the oropharynx with potential for airway compromise

What are some important causes of pharyngitis? Infections; Trauma (i.e., caustic ingestions); Irritant inhalant

What is the most common cause of pharyngitis? Viral infections

What are some viruses that are commonly implicated in pharyngitis? Epstein-Barr virus; Influenza virus; Parainfluenza virus; Adenovirus

What are some clinical features of infectious pharyngitis? Fever, sore throat, dysphagia, and cervical adenopathy

What are some clinical features of herpes simplex virus (HSV) pharyngitis? May present with features of infectious pharyngitis with grouped vesicles in the oropharynx that erode to form ulcers

What is the treatment for HSV pharyngitis? Acyclovir for immunocompromised patients, may benefit other patients (i.e., healthy)

What is the cause of infectious mononucleosis? Epstein-Barr virus
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What age group is commonly affected by infectious mononucleosis?</td>
<td>Young adults (10–26 years of age)</td>
</tr>
<tr>
<td>What are some clinical features of infectious mononucleosis?</td>
<td>Fever, sore throat, malaise, fatigue, and cervical adenopathy (esp. posterior) with exudative pharyngitis and hepatosplenomegaly</td>
</tr>
<tr>
<td>What is an important complication of infectious mononucleosis?</td>
<td>Splenic rupture</td>
</tr>
<tr>
<td>What is a common finding on a peripheral blood smear?</td>
<td>Lymphocytosis</td>
</tr>
<tr>
<td>What diagnostic test can be used to support the diagnosis of infectious mononucleosis?</td>
<td>Monospot test</td>
</tr>
<tr>
<td>What are some key points in the management of infectious mononucleosis?</td>
<td>Treatment is primarily supportive; Avoid contact sports for a month or so</td>
</tr>
<tr>
<td>List some indications for steroid use in infectious mononucleosis.</td>
<td>Neurologic complications (i.e., encephalitis); Airway compromise; Severe hemolytic anemia</td>
</tr>
<tr>
<td>What infectious organisms should be considered in a patient with infectious pharyngitis and a history of orogenital sex?</td>
<td>Gonorrhea</td>
</tr>
<tr>
<td>What is the significance of pharyngitis caused by gonorrhea in children?</td>
<td>Sexual abuse</td>
</tr>
<tr>
<td>What are some commonly used antibiotics for the treatment of pharyngitis caused by gonorrhea?</td>
<td>Ceftriaxone; Ofloxacin; Ciprofloxacin</td>
</tr>
<tr>
<td>What other organisms should be considered in pharyngitis caused by gonorrhea?</td>
<td>Chlamydia</td>
</tr>
<tr>
<td>What are two antibiotics commonly used to treat chlamydia?</td>
<td>1. Macrolides  2. Doxycycline</td>
</tr>
<tr>
<td>Is diphtheria a common cause of pharyngitis?</td>
<td>No—not with DPT immunizations, but can still occur for patients who did not receive DPT immunisation</td>
</tr>
<tr>
<td>Who are at risk for diphtheria?</td>
<td>Really young or old patients; DPT immunization not up-to-date; Developing countries</td>
</tr>
<tr>
<td>What is the organism responsible for diphtheria?</td>
<td>Corynebacterium diphtheriae</td>
</tr>
</tbody>
</table>
What is the pathophysiology of diphtheria?

Invasive infection that primarily affects the throat and nose causing tissue necrosis often producing the characteristic pseudomembrane in the posterior pharynx.

What are some clinical features of diphtheria?

Typically toxic-appearing with acute onset of fever, malaise, sore throat, and hoarse voice. PE: exudative pharyngitis with adherent pseudomembrane in the posterior pharynx and cervical adenopathy.

What are the systemic complications of diphtheria primarily due to?

Powerful exotoxin that primarily affects the cardiovascular system (CVS) and central nervous system (CNS).

What are some important complications of diphtheria?

Airway obstruction; Neuritis; Atrioventricular (AV) block; Myocarditis/endocarditis.

What are some common laboratory findings of diphtheria?

Positive culture on Loeffler’s media; Gram (+) rods with clubbing on swab; Complete blood count (CBC) showing thrombocytopenia.

What are some key points in the management of diphtheria?

Airway, breathing, circulation (ABC) (esp. airway); Respiratory isolation; Treatment aimed at bacteria and toxin; Consider tetanus and diphtheria (Td) booster in close contacts.

What is the typical medical treatment for a patient with diphtheria?

Diphtheria antitoxin; Penicillin or marcolide.

What is the most common cause of bacterial pharyngitis?

Group A beta-hemolytic Streptococcus.

Who are more commonly affected with Group A streptococcus?

Young adults during winter.

What is the Centor criteria?

Used to predict group A streptococcal (GAS) pharyngitis in adults, therefore help to guide use of Abx.

What are the four clinical features of the Centor criteria?

1. Fever
2. Absence of cough
3. Cervical lymphadenopathy
4. Tonsillar exudates

How is the Centor criteria used?

Used in conjunction with a rapid Streptococcus screen whether to treat for Group B streptococcus.
What are some commonly used antibiotics to treat GAS?

Penicillin; Azithromycin (for recurrent infections); First- and Second-generation cephalosporin

What role does the use of intramuscular (IM) dexamethasone play?

Often used for severe symptoms; Decreases severity of symptoms; Provides pain relief

What are some important complications of GAS?

Rheumatic fever; Glomerulonephritis; Pharyngeal space infections

Is the timely treatment of GAS enough to prevent the three mentioned complications?

All but glomerulonephritis

What is rheumatic fever?

Nonsuppurative complication of GAS, it is a serious inflammatory condition that can affect the heart, joints, nervous system, and skin. It most frequently occurs in children between the ages 6 and 16 years

What is the Jones criteria?

Used to help diagnose rheumatic fever in conjugation with laboratory findings

**Major.** Carditis; Polyarthritis; Subcutaneous nodules; Erythema marginatum; Chorea

**Minor.** History of rheumatic fever or heart disease; Fever; Arthralgias

What is the treatment of choice for rheumatic fever?

Penicillin; Steroids for carditis; NSAIDs for arthritis

What organism can produce pharyngitis in immunocompromised patients?

Fungi

List common fungal causes of pharyngitis.

Cryptococcus; Histoplasma; Candida

What groups are typically immunocompromised?

Diabetics; Chemotherapy recipients; Chronic steroid users; HIV-infected

What does the physical exam commonly reveal?

White/removable plaques on an erythematous base

What are two medications that can be used to treat fungal pharyngitis?

1. Nystatin swish and swallow
2. Systemic fluconazole

**Oral and Facial Infections**

What is the biggest concern of any abscess within the oral cavity?

Airway compromise
What is Ludwig's angina?
Progressive cellulitis of the floor of the mouth involving sublingual and submandibular space

What is a common cause of Ludwig’s angina?
Trauma or abscess to the posterior mandibular molars

Name the three potential spaces that the infection can tract to.
1. Sublingual space
2. Submandibular space
3. Submaxillary space

Name some commonly involved organisms in Ludwig’s angina.
Streptococcus; Staphylococcus; Anaerobic organisms (i.e., bacteroides)

What are some common clinical features of Ludwig’s angina?
Patient will often appear sick with odynophagia, dysphonia, dysphagia, drooling, trismus, massive swelling of the floor of the mouth, and an elevated tongue

What are the key points in the management of patients with Ludwig’s angina?
Airway management should be top priority; Immediate ENT consultation; Avoid putting the patient in a supine position; IV antibiotics (i.e., ampicillin-sulbactam); Admit to ICU

What are some organisms involved with a masticator space abscess?
Anaerobes; Streptococcus

What is the pathophysiology of how a masticator space abscess occurs?
Infection secondary to infection around third molar or extension from anterior space such as buccal space

What are some clinical features of a masticator space abscess?
Fever, trismus, and face swelling

What are some key points in the management of a masticator space abscess?
Careful attention to airway; Immediate ENT consultation; IV antibiotics (i.e., penicillin)

Name four potential spaces that can become infected in pharyngeal space infections.
1. Retropharyngeal space
2. Peritonsillar space
3. Peripharyngeal space
4. Prevertebral space

Where do retropharyngeal abscesses occur?
In the space posterior to the pharynx and anterior to the prevertebral fascia

In what age groups do retropharyngeal abscesses occur?
Most common in children <3 years of age

List the most common pathogens involved in retropharyngeal abscesses.
Anaerobes; Group A Streptococcus; S. aureus
What are some clinical features of retropharyngeal abscesses?

Patient will appear sick with fever, dysphagia, sore throat, swelling of neck, unilateral bulge of posterior pharynx wall, and stridor

What is the initial diagnostic test of choice for retropharyngeal abscesses?

Soft-tissue lateral film of neck

What are some findings of the lateral neck film that points to a retropharyngeal abscess?

Widening of the retropharyngeal space; Displacement of the larynx; Presence of air-fluid level in the space

What are some significant complications to keep in mind?

Airway obstruction; Invasion to adjacent structures; Sepsis; Aspiration

What are some key points in the management of retropharyngeal abscesses?

Careful attention to airway; Immediate ENT consultation for incision and drainage (I&D); IV antibiotics (i.e., ampicillin/sulbactum)

Where do prevertebral abscesses occur?

In the space anterior to the cervical spine and posterior to the prevertebral fascia

What are some clinical features of prevertebral abscesses?

Due to the very close proximity of the prevertebral space and retropharyngeal space, the clinical features are very similar to a retropharyngeal abscess

What distinguishing factor can help to distinguish one from the other?

Age (prevertebral abscesses more likely in older patients)

What is a common cause of prevertebral abscesses?

Cervical osteomyelitis

What is the initial diagnostic test of choice for prevertebral abscesses?

Lateral neck film

What are some findings of the lateral neck film that point to prevertebral abscesses?

Widening of the retropharyngeal space; Displacement of the larynx; Evidence of osteomyelitis of cervical spine

What three possible diagnostic tests can be used to confirm prevertebral abscesses?

1. CT
2. MRI
3. Cervical myelogram (not commonly used)

What are some key points in the management of prevertebral abscesses?

IV antibiotics; Neurosurgical consultation; Patient requires admission
Where do peritonsillar abscesses occur? Between the superior constrictor muscle and tonsillar capsule

What are peritonsillar abscesses commonly due to? Untreated tonsillitis

What age group are peritonsillar abscesses common in? Young adults

What are some organisms involved with peritonsillar abscesses? Usually polymicrobial

What are some clinical features of peritonsillar abscesses? Typically a history of sore throat and fever that becomes progressively worse and unilateral, can also have trismus, dysphagia, ear pain, tender cervical adenopathy, and deviated uvula to opposite side

What are some diagnostic studies that can help confirm the diagnosis of peritonsillar abscesses? CT; Ultrasound

What are some key points in the management of peritonsillar abscesses? ABC—ensure airway and hydration; IV antibiotics (i.e., penicillin); ENT consultation for I&D; Culture for pathogen; If uncomplicated can discharge with 24-hour follow-up

Where do peripharyngeal abscesses occur? Occur in the space lateral to the pharynx and medial to the masticator space

What are some common causes of peripharyngeal abscesses? Tonsillar infections; Dental infections

What are some clinical features of peripharyngeal abscess? Unilateral neck swelling, fever, neck pain, dysphagia, drooling, cervical adenopathy, and sore throat

What are some complication of peripharyngeal abscesses? Airway obstruction; Cranial nerve involvement; Erosion into carotids or jugular veins

What are some key points in the management of peripharyngeal abscesses? ABC—ensure intact airway; Admission for further care; ENT consultation; IV antibiotics

Facial Infections

What is the definition of sinusitis? Infection of the paranasal sinuses typically from a preceding URI
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
</table>
| Name four paranasal sinuses?                                            | 1. Maxillary  
2. Ethmoid  
3. Frontal  
4. Sphenoid                                                                                                                                 |
| What is the most commonly involved sinus in sinusitis?                  | Maxillary                                                                                                                                 |
| What is the pathophysiology of sinusitis?                              | Occlusion of the sinus ostia which is usually precipitated by a URI or allergic rhinitis, that results in a culture medium ideal for bacterial growth and infection |
| Name some pathogens typically involved in sinusitis?                    | *S. pneumoniae*; nontypeable *H. influenza*; *S. aureus*; *M. catarrhalis*                                                                 |
| What are some clinical features of sinusitis?                           | Nasal congestion, fever, purulent yellow-green discharge, headache, nasal congestion, tenderness over the affected sinus, and opacification of the sinus on transillumination |
| What are some diagnostic tests to consider in sinusitis?               | Diagnosis can typically be made on history and physical, but a CT of the sinuses can be done                                            |
| What are some key points in the management of sinusitis?               | Decongestants; Mucolytics; Analgesics; Antibiotics for severe cases or complications                                                    |
| What are some complications of sinusitis?                              | CNS involvement (i.e., meningitis, brain abscesses, etc.), cavernous sinus thrombosis, periorbital/orbital sinus, and surrounding abscess formation |
| What is the definition of mastoiditis?                                 | Infection of the mastoid air cells most commonly from AOM                                                                             |
| Name some pathogens typically involved in mastoiditis?                 | *S. pneumoniae*; nontypeable *H. influenza*; *S. aureus*                                                                                   |
| What are some clinical features of mastoiditis?                        | Posterior auricular tenderness, headache, hearing loss, otorrhea, and abnormal TM                                                       |
| What are some commonly used diagnostic studies to evaluate mastoiditis? | MRI; CT of temporal bone                                                                                                                |
| What are some complications of untreated mastoiditis?                 | CNS involvement (i.e., meningitis, brain abscesses, etc.), CN VII involvement, and labyrinthitis                                           |
What are some key points in the management of mastoiditis?

ENT consultation for possible debridment; IV antibiotics; Adequate pain control; Admission for further care

**DENTAL EMERGENCIES**

What cranial nerve (CN) provides primary sensation to the face?

Trigeminal nerve (CN V)  
Ophthalmic branch; Maxillary branch; Mandibular branch

What are branching nerves of the ophthalmic branch and the area they innervate:

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Area Innervated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasociliary nerve</td>
<td>Dorsal nose and cornea</td>
</tr>
<tr>
<td>Supraorbital nerve</td>
<td>Forehead and scalp</td>
</tr>
</tbody>
</table>

What are branching nerves of the maxillary branch and the area they innervate:

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Area Innervated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior alveolar nerves</td>
<td>Maxillary molar</td>
</tr>
<tr>
<td>Posterior</td>
<td>First and second bicuspid</td>
</tr>
<tr>
<td>Middle</td>
<td>Maxillary central, lateral, and cuspit teeth</td>
</tr>
<tr>
<td>Anterior</td>
<td></td>
</tr>
<tr>
<td>Nasopalatine and greater palatine nerves</td>
<td>Hard palate (along with gingiva)</td>
</tr>
<tr>
<td>Infraorbital nerve (with part of the superior alveolar nerve)</td>
<td>Midface, maxillary incisors, side of nose, upper lip, and lower eyelids</td>
</tr>
</tbody>
</table>

What are two commonly used local anesthetics to achieve oral anesthesia?

1. Lidocaine  
2. Marcaine (longer acting)

What are four nerves that are commonly blocked to achieve anesthesia?

1. Inferior alveolar nerve  
2. Posterior superior alveolar nerve  
3. Infraorbital nerve  
4. Supraorbital nerve

What type of infiltration is commonly used to achieve individual tooth anesthesia?

Supraperiosteal infiltrations

What are some important complications of performing nerve blocks in patients?

Vascular injury; Facial nerve damage (motor paralysis); Neural injury

What are the major portions of a tooth?

Root; Crown
What key structure keeps the tooth anchored into the alveolar bone?

Periodontal ligament

What are some key points in the initial management of an avulsed permanent tooth?

Hold tooth by crown and gently wash root; Place tooth back into socket; Do not brush root of the tooth; Immediate dental consultation

What is the key determinant of the viability of an avulsed tooth?

Time outside the socket

What is the reason why the root should not be brushed or wiped?

Preserving the periodontal ligament is vital

What are two other management points to consider?

1. Prophylactic antibiotics if indicated
2. Tetanus status

Are deciduous (primary) teeth typically placed back into the socket?

No—alveolar ankylosis may result

How are alveolar fractures typically noticed?

Panorex film or evident on exam

What are other possible dental injuries from alveolar fractures?

Avulsion or subluxation of tooth; Dental fractures

What is typically done for alveolar fractures?

Immediate dental consultation; Reduction and fixation (via wire); Antibiotics and tetanus when indicated

What are some important points and management for the following classification of tooth fractures:

**Ellis I**

Isolated enamel fracture; No pain; Elective treatment

**Ellis II**

Fracture of enamel; dentin exposed; Sensitive to temperature changes of hot/cold; Calcium hydroxide paste over dentin if <14 years of age; Dressing over tooth if >14 years of age; Dental follow-up in timely manner

**Ellis III**

Fracture of tooth with pulp exposure; Pink tinge may be seen on exam; This is a true dental emergency; Immediately consult a dentist and place wet cotton with dental or aluminum foil wrapped if there is a delay

What is typically done for dental caries?

Proper pain control and dentist referral
What is a complication of dental caries to consider?

Periapical abscess

What are some clinical features of a periapical abscess?

A fluctuant swelling, sharp/severe pain when tooth is percussed, and temperature sensitivity

What are some key points in the management of a periapical abscess?

I&D of the abscess; Antibiotic coverage (may or may not help); Dental referral

CLINICAL VIGNETTES

26-year-old with a recent URI presents with a fever, fatigue, and left ear pain, but is otherwise healthy; PE: left TM shows bullae and is erythematous in appearance

Bullous myringitis

18-year-old male with no past medical history (PMH) presents after being involved in a bar fight and complains of a bruise on his leg and some facial pain; PE: ecchymosis of left thigh and nasal swelling with tenderness and crepitence

Nasal fracture

31-year-old female presents with a 1-week history of sore throat with low-grade fevers and fatigue. Patient mentions her sore throat is getting progressively worse; PE: exudative pharyngitis with posterior cervical adenopathy along with left upper quadrant (LUQ) tenderness

Mononucleosis

3-year-old infant presents with low-grade fever, decreased appetite and mother mentions that he is tugging at his ear; PE: decreased mobility of TM on pneumatic otoscopy

Acute otitis media

61-year-old female who just recently finished her antibiotics for a UTI presents with bilateral hearing loss, but is otherwise healthy; PE: decreased hearing acuity and normal Rinne and Weber test

Sensorineural hearing loss secondary to antibiotic use

3-year-old female was brought in by mother due to purulent drainage from left nasal passage, but swears that the child did not place any objects in the nose; PE: general exam was unremarkable

Nasal foreign bodies
6-year-old male is brought in by mother for persistent nosebleeds, but is otherwise healthy with immunizations up-to-date; PE: child was actively picking his nose during the exam

13-year-old female with a sore throat 1 month ago now presents with fevers, joint pain, and what the mom notes as “weird movements”; PE: pain of joints with movement and subcutaneous nodules

19-year-old male presents with severe left ear pain and complains of decreased hearing with occasional purulent discharge; PE: TM could not be visualized due to the purulent discharge in the external canal

9-year-old female presents with 3-day history of low-grade fevers, sore throat, and fatigue but otherwise healthy; PE: cervical adenopathy with exudative pharyngitis

2-year-old female is brought in by mom for high fevers, sore throat, and some swelling of her neck; PE: sick-appearing child with unilateral bulge of posterior pharynx wall and stridor

67-year-old female present with epistaxis that began 2-hours earlier and has not stopped bleeding from conventional means; PE: epistaxis that is refractory to all methods that are used for anterior nosebleeds

43-year-old male with a recent history of AOM that was not treated presents otorrhea, pain around the ear, and a moderate headache; PE: tender posterior auricular area and distorted TM

24-year-old female presents with a history of nasal congestion, fever, purulent yellow-green discharge, and headache; PE: yellowish discharge from nose and tender maxillary sinus

- Anterior nosebleed
- Rheumatic fever
- Otitis externa
- Streptococcal pharyngitis
- Retropharyngeal abscesses
- Posterior nosebleed
- Mastoiditis
- Sinusitis
## Pulmonary Emergencies

### Pneumonia

**What are some important things to know about bacterial pneumonia?**

It accounts for about 10–15% of admissions; *Streptococcus pneumoniae* is the most common agent; Most common mechanism is aspiration

**Name some other important bacterial agents in bacterial pneumonia.**

*Pseudomonas aeruginosa; Hemophilus influenza; Staphylococcus aureus; Escherichia coli*

**Name some predisposing factors that increases susceptibility to bacterial pneumonia.**

Impaired immunity; Impaired gag reflex/mucociliary transport; Iatrogenic (i.e., endotracheal tube); Chest wall dysfunction

**What are some clinical features of bacterial pneumonia?**

Fever, chills, productive cough, purulent sputum, and pleuritic chest pain

**What are some common physical findings in a patient with bacterial pneumonia?**

Crackles, wheezes, dullness to percussion, egophony, and tactile fremitus

**Name the most likely organism for each of the following scenarios:**

- **Alcoholic who presents with fever, chills, and productive cough. Chest x-ray (CXR) shows lobar pneumonia**
  - *Klebsiella pneumoniae*

- **45-year-old male who has been in the ICU for 2 weeks on vent support develops fever and chills with productive green sputum**
  - *P. aeruginosa*
63-year-old male with a history of chronic obstructive pulmonary disease (COPD), DM, and debilitation presents with Shortness of breath (SOB), fever, and a Chest x-ray (CXR) that shows patchy infiltrates

36-year-old bird-breeder presents with a 3-day history of high fever, hacking cough, and severe headache

23-year-old farmer presents with a sudden onset of high fever, myalgias, and hacking cough. He mentions he often cleans at one of the slaughterhouses

41-year-old male presents with SOB, dyspnea, and productive cough recalls onset of symptoms after returning from a spa

37-year-old male who typically skins rabbits presents with high fever, SOB, and hemoptysis

19-year-old patient with AIDS and a cell count of <200 cells/mm³ presents with fever, nonproductive cough, and dyspnea

What are some diagnostic tests to consider in addition to the CXR?

Arterial blood gas (ABG); Sputum culture (typically for high-risk patients); Blood culture

What are some complications of bacterial pneumonia?

Abscess formation (esp. S. aureus); Sepsis; Empyema

Name some of the most common agents in the following age group:

Neonates

Group B streptococci, E. coli, and C. pneumoniae

Children (5 weeks to 18 years)

Respiratory syncytial virus (RSV), Mycoplasma pneumoniae, C. pneumoniae, and S. pneumoniae

Adults (18–40 years)

M. pneumoniae, C. pneumoniae, and S. pneumoniae

Adults (45 years and older)

S. pneumoniae, H. influenzae, anaerobes, and gram-negatives

What are some commonly used antibiotics in uncomplicated pneumonia?

Penicillin, macrolides, and doxycycline

H. influenza

Chlamydia psittaci

Coxiella burnetii

Legionella pneumophila

Francisella tularensis

Pneumocystis carinii

Arterial blood gas (ABG); Sputum culture (typically for high-risk patients); Blood culture

Abscess formation (esp. S. aureus); Sepsis; Empyema

Group B streptococci, E. coli, and C. pneumoniae

Respiratory syncytial virus (RSV), Mycoplasma pneumoniae, C. pneumoniae, and S. pneumoniae

M. pneumoniae, C. pneumoniae, and S. pneumoniae

S. pneumoniae, H. influenzae, anaerobes, and gram-negatives

Penicillin, macrolides, and doxycycline
What are some commonly used antibiotics for those with comorbidities? Fluoroquinolones

Name three common causes of atypical pneumonia? 1. M. pneumoniae 2. C. pneumoniae 3. L. pneumophilia

What are some clinical features of atypical pneumonia? Headache, fever, nonproductive cough, and myalgias

What are some important things to know about mycoplasma pneumonia? Most common cause of atypical pneumonia 1–3 weeks incubation; Most common in ages 4–40 years; CXR often show a reticulonodular pattern

What are some complications of mycoplasma pneumonia? Splenomegaly; Aseptic meningitis; Encephalitis; Respiratory failure

What is the preferred antibiotic? Erythromycin; Tetracycline or doxycycline are alternatives

Name the most likely organism for each of the following scenarios involving viral pneumonia and the preferred treatment:

- 34-year-old with a history of a kidney transplant presents with fever, cough, and a CXR showing interstitial infiltrates: Cytomegalovirus; Treatment (Tx): Ganciclovir or foscarnet
- 2-year-old child presents with a 4-day history of fever, chills, and coryza with a CXR that shows patchy infiltrates: RSV; Tx: Primarily supportive
- 21-year-old male presents with a 2-week history of fever, chills, and nonproductive cough during winter: Influenza virus; Tx Amantadine
- 34-year-old female presents with fever, headache, and myalgia. She primarily works with rodents and is from Arizona: Hantavirus; Tx: Supportive/ribavirin

ASTHMA

What is the definition of asthma? It is a chronic condition characterized by reversible airway constriction typically initiated by a variety of stimuli

What are some important things to know about asthma? More common in children and adolescents; Prevalence is increasing; Asthma-related morbidity is also increasing
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name some common triggers of asthma.</td>
<td>Allergens; Exercise; Medications; Cold exposure</td>
</tr>
<tr>
<td>What are some of the clinical features of asthma?</td>
<td>Dyspnea, cough, and wheezing</td>
</tr>
<tr>
<td>What are some important diagnostic tests to consider in asthma?</td>
<td>Pulmonary function tests (i.e., PEFR); ABG (if impending respiratory failure); CXR (more to rule out other conditions); ECG (if you suspect ischemia)</td>
</tr>
<tr>
<td>What are some key points in the management of asthma?</td>
<td>Ensure adequate oxygenation; Optimize lung function (i.e., medication); Identify the cause of exacerbation</td>
</tr>
</tbody>
</table>
| What are the three classes of drugs that are the mainstay for the treatment of asthma exacerbation? | 1. Beta-adrenergic (albuterol)  
2. Anticholinergic (ipratropium)  
3. Corticosteroids (methylprednisolone) |
| What role does noninvasive positive pressure ventilation (NPPV) play?    | Impending respiratory failure where the patient is able to cooperate                                                                  |
| What are some findings of impending respiratory failure?                | Use of accessory muscles; Cyanosis; Altered mental status (typically from hypercapnia); No breath sounds (no or very little airflow) |
| What procedure should be considered in the setting of impending respiratory failure? | Intubation                                                                                                                           |
| What are some other agents that can be considered when the mainstay treatment of asthma shows little improvement? | Magnesium sulfate; Heliox (helium to improve airflow); Terbutamine                                                                 |
| What are some important elements to consider when deciding to admit the patient? | Social supports; Recent hospitalizations and past intubations; Compliance with medication; Severity of the exacerbation               |

**CHRONIC OBSTRUCTIVE PULMONARY DISEASE**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are the disease elements that make up chronic obstructive pulmonary disease (COPD)?</td>
<td>Emphysema; Asthma; Chronic bronchitis</td>
</tr>
</tbody>
</table>
What are some important features for each of the following elements:

<table>
<thead>
<tr>
<th>Emphysema</th>
<th>Irreversible airway destruction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis</td>
<td>Airway hypersecretion</td>
</tr>
<tr>
<td>Asthma</td>
<td>Hyperactive airway and inflammation</td>
</tr>
</tbody>
</table>

What are some important risk factors for the development of COPD?

- Tobacco use (most common cause);
- Environmental pollution;
- Alpha1–antitrypsin deficiency;
- Cystic fibrosis

What are some clinical features of COPD?

- Dyspnea, cough, chest tightness, and occasional hemoptysis

What are some common causes of COPD exacerbation?

- Infections; Pulmonary embolism (PE); Congestive heart failure (CHF) exacerbation; Tobacco use

What are some of the common clinical features of the following COPD variant:

- **Chronic bronchitis (blue bloaters)**
  - Tend to be heavy set;
  - Normal chest diameter;
  - Productive wet cough

- **Emphysema (pink puffer)**
  - Tend to be thin;
  - Increased AP chest diameter;
  - Dyspneic

What are some possible findings on CXR?

- Increased AP diameter;
- Overinflation;
- Presence of bullae

What are some other diagnostic tests to consider in COPD?

- ABGs; ECG (for ischemia or dysrhythmias)

What two dysrhythmias are common in COPD?

1. Multifocal atrial tachycardia
2. Atrial fibrillation

What are some key points in the management of COPD?

- Oxygenation is the cornerstone;
- Beta-adrrenergic agonist;
- Anticholinergics; Corticosteroids use; Abx if signs of infection—purulent sputum

**HEMOPTYSIS**

What is the definition of hemoptysis?

- Coughing up of blood that originates from the tracheobronchial tree or pulmonary parenchyma
### PLEURAL EFFUSION AND EMPYEMA

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of a pleural effusion?</td>
<td>An abnormal accumulation of fluid in the pleural space</td>
</tr>
<tr>
<td>What are some of the characteristics of the following types of pleural effusion?</td>
<td></td>
</tr>
<tr>
<td><strong>Transudate</strong></td>
<td>Increase in hydrostatic pressure; Decreased oncotic pressure; CHF is the most common cause; Low protein infiltrate</td>
</tr>
<tr>
<td><strong>Exudate</strong></td>
<td>Lymphatic blockage; Typically due to malignancy and infection; High protein infiltrate</td>
</tr>
<tr>
<td>What are some clinical features of pleural effusion?</td>
<td>Pleuritic chest pain, cough, and SOB; PE: dullness to percussion and pleurisy</td>
</tr>
<tr>
<td>Which CXR view is more sensitive for detecting pleural effusion?</td>
<td>Lateral decubitus (as little as 5 mL seen)</td>
</tr>
<tr>
<td>What procedure is commonly used to analyze pleural effusions?</td>
<td>Thoracentesis</td>
</tr>
<tr>
<td>What are commonly used pleural fluid studies?</td>
<td>Gram stain and cultures; Pleural fluid lactate dehydrogenase (LDH) and protein; Serum LDH, protein, and glucose</td>
</tr>
</tbody>
</table>
Name some of the criteria used to support that a pleural effusion may be exudative?

- Pleural fluid LDH >200 IU/mL;
- Pleural fluid cholestrol >60 mg/dL;
- Pleural fluid protein/serum protein >0.5

Name some important causes of transudative pleural effusion?

- CHF;
- Low protein states (i.e., cirrhosis);
- Peritoneal dialysis

Name some important causes of exudative pleural effusion?

- Bacterial pneumonia (PNA);
- TB;
- Malignancy;
- Connective tissue disorder (i.e., Systemic lupus erythematosus [SLE])

What is the definition of empyema?

Collection of pus in the pleural space

What are some common causes of empyema?

- Infections (i.e., gram negatives);
- Aspiration PNA;
- Iatrogenic (i.e., chest tube)

What are some clinical features of empyema?

- Fever, chills, pleuritic chest pain,
- SOB, fatigue, and weight loss

What are some diagnostic tests used to diagnosis empyema?

- CXR;
- Thoracentesis

What are some complications of empyema?

- Loss of lung tissue;
- Bronchopleural fistula;
- Pleural adhesions

What are some key points in the management of empyema?

- Pleural drainage via chest tube;
- Broad spectrum Abx;
- Thoracoscopy (controversial)

**LUNG ABSCESS**

What is the definition of a lung abscess?

- It is a cavitation of the lung parenchyma due to central necrosis

What is the most common cause of a lung abscess?

- Aspiration

What class of bacteria are typically involved in a lung abscess?

- Typically mixed anaerobic and gram (-) bacteria

What are some clinical features of a lung abscess?

- Weakness, fever, SOB, pleuritic chest pain, putrid sputum, and hemoptysis

What are some important diagnostic tests to consider in a lung abscess?

- Complete blood count (CBC);
- CXR (shows cavitation);
- Sputum stain

What are some complications of a lung abscess?

- Empyema;
- Bronchopleural abscess;
- Chronic lung abscess
### TUBERCULOSIS

What are some key points in management of a lung abscess? | Abx therapy (Clindamycin preferred); Surgery if cause is a tumor or fistula
---|---

#### TUBERCULOSIS

**What are some important points to know about tuberculosis (TB)?**
The incidence of TB is rising (esp. in AIDS patients); Top cause of infectious death worldwide; Transmission is primarily respiratory

**What is the pathophysiology of infection from *M. tuberculosis***? 
Obligate aerobic rod (acid-fast staining) that is phagocytized by macrophages, but not killed and allowed to grow (albeit slowly)

**What is the primary determinant of whether the infection is contained or likely to spread?** 
Immune status (lifetime risk of activation is still 10% in the general population)

**What are some factors that are associated with an increase in reactivation?** 
DM; Immunocompromised (i.e., AIDS); Transplant recipient; Malignant disease

**What are some of the clinical features for each of the following TB states:**

<table>
<thead>
<tr>
<th>Primary</th>
<th>Asymptomatic in most patients; Positive TB test primary way to detect; Sometimes Ghon complex on CXR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secondary (reactivation)</td>
<td>Constitutional symptoms (i.e., weight loss); Productive cough; Hemoptysis; Up to 20% have extrapulmonary features</td>
</tr>
</tbody>
</table>

**Name the four most common sites of extrapulmonary involvement.**

1. CNS (TB meningitis)
2. Vertebral bodies (Pott’s disease)
3. Liver
4. Psoas muscle

**What are some important diagnostic tests to consider in TB?**

CXR; Sputum (acid-fast bacilli); Purified protein derivative (PPD) test

**What is the criteria for a positive PPD test?**

Less than 5-mm induration for immunocompromised patients (i.e., AIDS); Less than 10-mm induration for high-risk individuals (IV drug abusers and immigrants from high-risk areas); Less than 15-mm induration in healthy individuals
What is a common cause of a false negative PPD? Anergy
What is a common cause of a false positive PPD? Infection with a mycobacterium species such as *M. avium*
What are some key points in the management of TB? Isolation once TB is suspected; Multidrug therapy for more than 6 months; Baseline liver/kidney test and visual acuity
What are the main side effects for each of the drug used to treat TB (RIPE):
- **Rifampin** Orange-colored urine, tears, and saliva; Increase P450 activity
- **Isoniazid (INH)** Hepatitis; Neuropathy (give vitamin B6)
- **Pyrazinamide (PZA)** Hepatitis; Hyperuricemia; Arthralgias
- **Ethambutol** Optic neuritis; Rash

### SPONTANEOUS PNEUMOTHORAX

What is the definition of a spontaneous pneumothorax? It is collection of air into the pleural space (assuming that no trauma is involved)
What are some important things to know about each of the different type of spontaneous pneumothorax:
- **Primary spontaneous pneumothorax** Typically occurs in healthy individuals; Most have a history of smoking; Results from rupture of a bleb
- **Secondary spontaneous pneumothorax** Typically will have underlying lung disease; COPD and asthma most common cause; Usually patients are older than 45 years
What are some clinical features of spontaneous pneumothorax? Sudden onset of dyspnea and pleuritic chest pain. PE: decreased breath sounds and hyperresonance on the affected side
What is the diagnostic test of choice? CXR
What are the key points in the management of spontaneous pneumothorax? All patients should receive oxygen; Observation and serial CXR if small; If large/expanding it is equal to the chest tube
What is a feared complication of spontaneous pneumothorax?  
Tension pneumothorax

What are some clinical features of tension pneumothorax  
Hypotension, absent breath sounds on the affected side, jugular venous distension (JVD), and trachea deviation

What are some key points in the management of tension pneumothorax?  
Immediate chest decompression (14 gauge); Follow with chest tube placement

What are some more specifics of chest decompression in the management of tension pneumothorax?  
If any evidence of tension pneumothorax, immediate needle decompression should be done with a needle placed into the second and third intercostal space at the anterior axillary line followed by a chest tube in the fifth intercostal space in the mid-axillary line

CLINICAL VIGNETTES

10-year-old child with history of allergies presents with acute respiratory distress with a recent history of chronic coughing, but has otherwise been healthy; PE: tachypnea, intercostal retractions, and audible wheezing; CXR: hyperinflation of lung  
Bronchial asthma

63-year-old female with a long history of smoking comes in via Emergency medical services (EMS) in acute respiratory distress with a recent illness per report of family members; PE: hyperresonant chest, decreased breath sounds bilateral; CXR: hyperinflation of lung and small infiltrate of right lower lobe  
COPD exacerbation

81-year-old female with a long history of smoking presents with a 2-week history of worsening hemoptysis, but otherwise is healthy except for a 20-lb weight loss in a month period; CXR: a spiculated mass is seen on the left side  
Bronchogenic cancer

45-year-old male with an Hx of CHF presents with SOB and wet cough, but otherwise has been doing well; PE: dullness to percussion and pleurisy; lateral decubitus CXR: showed dependant fluid collection  
Pleural effusion
35-year-old alcoholic the ED for alcoholic intoxication presents with fever, hemoptysis, SOB, and purulent sputum who was recently seen in; PE: lung fields relatively clear; CXR: central cavitation

23-year-old lanky male with a smoking history presents with sudden onset of dyspnea and pleuritic chest pain, but is otherwise healthy; PE: an area of hyperresonance on the left side

31-year-old male with Hx of HIV presents with hemoptysis and recent weight loss along with a CD4+ count of <200; PE: cachetic appearance, is actively coughing, but otherwise unremarkable exam

56-year-old male with recent “flu” presents with a 2-day history of fever, chill, productive cough, and pleuritic chest pain; PE: ill-appearing patient, crackles, wheezes, and dullness to percussion of right lung

- Lung abscess
- Spontaneous pneumothorax
- Tuberculosis
- Community-acquired pneumonia
### ACUTE CORONARY SYNDROME

**What is acute coronary syndrome (ACS)?**

- It is a continuum of presentations of coronary artery disease where the symptoms are due to myocardial ischemia. The underlying cause of ACS is an imbalance between demand and supply of myocardial oxygen.

**What are the three clinical presentations that cover the ACS spectrum?**

1. Unstable angina
2. Non-ST-elevation MI (NSTEMI)
3. ST-elevation MI (STEMI)

**What are three non-modifiable risk factors associated with development of ACS?**

1. Gender
2. Age
3. Family history

**What are four modifiable risk factors associated with the development of ACS?**

1. Cholesterol
2. Hypertension
3. Diabetes
4. Smoking

**What are some clinical features of the following presentations of ACS:**

- **Stable angina**
  - Episodic pain that is transient and predictable, typically reproducible on exertion and improves with rest or use of nitro

- **Unstable angina**
  - New-onset angina that can be exertional or at rest, different from previous stable angina, increased frequency of attack or increased resistance to relief such as nitro
Myocardial infarction (MI): Substernal chest discomfort that lasts longer than 20 minutes, typically associated with nausea, vomiting, dyspnea, diaphoresis, and radiation to arms/jaw/back.

What are some clinical features of atypical MI? Vague chest discomfort/pressure, nausea and vomiting, short of breath, confusion, dizziness, abdominal pain, weakness, or syncope.

What population group can frequently present with atypical symptoms? Diabetics; Women; Elderly; Neurological dysfunction (i.e., cord injury).

What are three important elements in the patient’s presentation to consider in an MI? 1. History and physical 2. Cardiac enzymes 3. ECG.

What is the single most important diagnostic test to obtain in a patient with suspected MI? ECG (within 10 minutes of arrival).

What are other uses of the initial ECG? Screening other disease processes such as pulmonary embolism (PE) and pericarditis.

What are some important things to note about the use of an ECG? Initial ECG is diagnostic 50% of the time; Serial ECGs are more useful for evolving MI; Comparison with a previous ECG is important.

<table>
<thead>
<tr>
<th>ECG Infarct Region</th>
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<tbody>
<tr>
<td>I: Lateral</td>
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<tr>
<td>II: Inferior</td>
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<tr>
<td>III: Inferior</td>
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What are some ECG findings in a patient who presents with a STEMI? Inverted T-waves; Q-waves; ST segment elevation >1 mm in two or more contiguous leads; Left bundle branch block.

What are some common complications for the following infarction location: Inferior Increased vagal tone; Bradyarrhythmias are more common; High association with right ventricular wall infarct.
Greater risk of left ventricular (LV) dysfunction
Greater risk of LV dysfunction (CHF); Conduction abnormalities
Hypotension (preload dependent); Cardiogenic shock
Initial level cannot be used to exclude MI; Serial levels are more useful; Detection requires enough time/tissue death

<table>
<thead>
<tr>
<th>Cardiac Enzymes</th>
<th>Initial Elevation</th>
<th>Peak</th>
<th>Return to Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponins</td>
<td>2–6 hours</td>
<td>12–16 hours</td>
<td>5–14 days</td>
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<tr>
<td>CK-MB</td>
<td>4–6 hours</td>
<td>12–24 hours</td>
<td>2–3 days</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>2 hours</td>
<td>6–8 hours</td>
<td>3–4 days</td>
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What is MONA? It is the initial treatment for all patients with suspected ACS
Morphine
Oxygen
Nitro
Aspirin

What treatment within MONA is clearly shown to improve morbidity and mortality of ACS and should *always* be given (assuming no contraindications)? Aspirin

What else should be done for all patients with suspected ACS? IV-O₂-Monitor

What are some important points for the following treatments used in ACS:

**Aspirin (ASA)**
Antiplatelet medication; Should be given within 4 hours of chest pain onset; Clearly shown to improve outcome

**Glycoprotein IIb/IIIa inhibitors**
Abciximab, tirofiban, and eptifibatide; Platelet inhibitors; Used prior to percutaneous coronary intervention; Also indicated in some cases of NSTEMI
ADP-receptor inhibitors
Clopidogrel and ticlopidine; Also prevents platelets aggregation; Second-line if ASA cannot be used: Reduce risk of recurrence in patients with recent MI or stroke

Heparin
Antithrombin III inhibitor; Patients with ACS (UA/NSTEMI/ STEMI); Decrease reinfarction, deep vein thrombosis (DVT), LV thrombus; Adverse drug reactions include bleeding complications and heparin-induced thrombocytopenia

Beta-blockers
Improved outcome in acute MI; Should be given in acute MI assuming no contraindications; Should be given within 2–3 hours; Contraindications include high-degree heart block, bradycardia, severe CHF

Nitroglycerin
Decreases preload/dilates coronary arteries; Should be given in ischemic chest pain; Avoid if hypotensive and if on sildenafil

Morphine
Decreases anxiety, preload, and afterload; Should be given if pain persists after nitros; Can cause hypotension/decrease respiratory drive

What is the treatment for choice for STEMI?
Reperfusion therapy
“Door to balloon time” 90 minutes (PCI) or “door to lytics” 30 minutes

What are some commonly used thrombolytics in AMI?
Streptokinase (not commonly used); Tissue plasminogen activator; Tenecteplase

What is the most serious complication of lytics?
Intracranial hemorrhage (ICH)

Which is the preferred reperfusion modality?
PCI is associated with slightly better outcomes, lower incidence of reinfarction, and death

Should thrombolytics be withheld if PCI is anticipated?
They should not be withheld if transfer to a cath lab will be greater than 90 minutes despite better outcomes with PCI
### Congestive Heart Disease and Pulmonary Edema

#### What is the definition of congestive heart failure (CHF)?
A pathophysiologic state in which, at normal filling pressures, the heart is incapable of pumping a sufficient supply of blood to meet the metabolic demands of the body.

#### What are the four classifications commonly used in CHF:
- **Class I**: Not limited with normal physical activity by symptoms.
- **Class II**: Ordinary physical activity results in fatigue, dyspnea, or other symptoms.
- **Class III**: Marked limitation in normal physical activity.
- **Class IV**: Symptomatic at rest or with any physical activity.

#### How is congestive heart failure classified?
While many classification methods exist (high output vs. low; systolic vs. diastolic), a useful clinical construct is the distincton of left versus right heart failure.

#### What are other some clinical features of left ventricular failure?
Nocturnal angina, paroxysmal nocturnal dyspnea, orthopnea, fatigue, diaphoretic, and anxious. PE: rales/wheezes, S3 or S4 gallop, tachycardia and tachypnea, and pulsus alternans.

#### What are some common causes of left ventricular failure?
Ischemic heart disease (no. 1 cause); HTN; Valvular heart disease; Dilated cardiomyopathy.

#### What is cardiogenic pulmonary edema?
Acute presentation of left heart failure resulting from an imbalance in pulmonary vascular hydrostatic and oncotic forces and leading to transudation of fluid into the pulmonary interstitium.
What is the most common cause of right heart failure? Left heart failure

What are some other common causes of right heart failure? Pulmonary hypertension; Pulmonary embolism (PE); Chronic obstructive pulmonary disease (COPD); Right ventricular infarct

What are some common physical exam findings of right ventricular failure? Neck vein distension, ascites, dependent edema, and hepatojugular reflux

What are some precipitating factors of acute pulmonary edema? Myocardial ischemia; High sodium diet; Noncompliance with medications; Dysrhythmias; COPD (chronic cor pulmonale)

Name some common radiographic findings on a CXR in acute pulmonary edema? Generally an enlarged cardiac silhouette, pleural effusions, cephalization (vascular redistribution to upper lung fields), and bilateral perihilar infiltrates

Does a normal CXR exclude acute pulmonary edema? No—CXR findings may be delayed up to 12 hours after symptom onset

What is β-type natriuretic peptide (BNP)? Cardiac myocytes secrete BNP in response to the high atrial and ventricular filling pressures

How is BNP used clinically? Increasing use as a serum marker for CHF; Levels of <100 pg/ml reliably exclude acute CHF; High negative predictive value with low BNP

What is the single most important agent for the treatment of acute CHF? Oxygen

Patients with decompensated left heart failure frequently require assistance in maintaining adequate oxygenation/ventilation. What are the treatment options:

- **High-flow via nonrebreather mask**
  - Optimal option to deliver 100% oxygen; Used to maintain adequate oxygen saturation; Commonly used to avoid hypoxia

- **Noninvasive positive pressure ventilation**
  - Continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP) commonly used; Improves oxygenation and dyspnea; Early use helps avoid intubation
### Endotracheal intubation

Final pathway if other methods fail; Typically used for the following conditions:
- Cannot maintain PaO₂ above 60 mm Hg;
- Obtunded;
- Progressive increase in CO₂;
- Increasing acidosis

### What are some key points in the management of acute decompensated left heart failure?

- IV-O₂-monitor; NTG/furosemide/morphine first-line agents; Carefully monitor for hypotension as well

### DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

<table>
<thead>
<tr>
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<th>Answer</th>
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</thead>
<tbody>
<tr>
<td>What is Virchow’s triad?</td>
<td>Factors predisposing to vascular thrombosis with risk of pulmonary embolism: Hypercoagulability; Vessel wall injury; Venostasis</td>
</tr>
<tr>
<td>What is the biggest risk factor for the development of deep venous thrombosis (DVT)?</td>
<td>Prior history of DVT</td>
</tr>
<tr>
<td>List some other important risk factors for the development of a DVT.</td>
<td>Cancer; Pregnancy and postpartum; Recent trauma and surgery; Estrogen therapy; Obesity; Protein C and S deficiency</td>
</tr>
<tr>
<td>How does the number of risk factors effect the likelihood of a DVT?</td>
<td>Greater number of RFs = higher risk of DVT</td>
</tr>
<tr>
<td>What determines the clinical presentation of a DVT?</td>
<td>Degree of occlusion; Location of occlusion; Extent of collaterals</td>
</tr>
<tr>
<td>What are some common clinical features in a DVT?</td>
<td>Unilateral leg swelling, tenderness, edema, discoloration, palpable cord, and Homans’ sign</td>
</tr>
<tr>
<td>What is the most reliable finding on physical exam for a DVT?</td>
<td>Unilateral leg swelling with more than 3-cm difference from the other leg</td>
</tr>
<tr>
<td>Can a DVT be diagnosed by physical exam alone?</td>
<td>Due to variability of presentation, it cannot be used to exclude or make the diagnosis</td>
</tr>
</tbody>
</table>
What are some characteristics of commonly used ancillary testing for the diagnosis of a DVT:

**D-dimer assay**
Fibrin degradation product is with a DVT/PE; Other conditions raise it such as CA or recent surgery; More sensitive for proximal clots; A positive result may require further testing

**Duplex ultrasonography**
Initial diagnostic test in many cases; Ideal for patients who are pregnant, diabetic, or have a contrast allergy noninvasive; Highly sensitive/specific for proximal DVT; Less sensitive for deep vein, pelvic, and IVC thrombosis

**MRI**
MRI rarely used in the ED; Highly sensitive/specific for a DVT; Noninvasive but expensive; Can detect pelvic, renal, and calf thrombi; Useful for second/third trimester pregnancy

**Contrast venography**
Once the gold standard diagnostic test; Invasive/painful and requires contrast; Very high sensitivity/specificity

**What are the goals of treatment for a DVT?**
To prevent PE; Prevent post-phlebitic syndrome

**What are some commonly used anticoagulants for a DVT?**
Heparin; Low-molecular-weight heparin (LMWH); Warfarin

**What are some indications for the use of a Greenfield umbrella filter?**
Contraindication to anticoagulation; Urgent surgery (cannot anticoagulate prior); Anticoagulation has failed (still clotting)

**What are general indications for admission for patients with a DVT?**
Limited cardiopulmonary reserve; IV heparin use (contraindications to LMWH); Poor compliance with medications

**What is the epidemiology of a pulmonary embolism?**
Third most common cause of death in the United States; Most common preventable death in the hospital setting; Up to 1/3 of PEs are undiagnosed
What is a major source of a PE? Venous thrombi from lower extremities and pelvis

What are some other possible sources of a PE? Renal and ovarian veins; Paradoxical left-to-right shunts; Right side of heart

What are some risk factors for the development of a PE? The same as those for DVT

What is the most common symptom of a PE? Dyspnea

What are some common clinical features of a PE? Pleuritic chest pain, hemoptysis, cough, tachycardia, sweating, elevated temperature, and syncope/hypotension in massive PE

What is the classic triad of a PE? Pleuritic chest pain; Dyspnea; Hemoptysis

What are some commonly used screening tests for a PE? ABG; CXR; ECG; D-dimer

What are some functions of these screening tests? Excluding other disease processes; May support the diagnosis of a PE; Should not be used to rule out or rule in PE

What are some common findings in an arterial blood gas for a patient with PE? \( \text{PO}_2 < 80 \text{ mm Hg; Mild respiratory alkalosis; Elevated alveolar-arterial (A-a) gradient} \)

Does a normal A-a gradient, normal \( \text{PO}_2 \), and normal vital signs exclude a PE? No

What is the most common CXR finding in a patient with suspected PE? Normal CXR

What are some radiographic abnormalities that can be seen on CXR in a PE? Elevated hemidiaphragm; Atelectasis; Small pleural effusion

What is Hampton’s hump? Triangular density with a rounded apex that points toward the hilum representing pulmonary infarction

What is Westermark’s sign? Regional oligemia

How common are Hampton’s hump and Westermark’s sign? Rare—if present highly suggestive of PE

What are some common findings on ECG of a patient with a PE? Sinus tachycardia (most common finding); Evidence of right heart strain (S1, Q3, T3); Transient nonspecific ST-T wave changes
What are some characteristics of commonly used testing in PE:

Spiral CT Extensively used diagnostic test; Done within minutes/assess other possible disease processes; Also used if V/Q read as indeterminate; Disadvantage: contrast allergy, radiation, and risk of acute renal failure

Ventilation-Perfusion (V/Q) scan Commonly used as a screening test; Normal V/Q scan virtually excludes PE; Must also look at clinical probability; Typically either read as normal, indeterminate, or high probability; If indeterminate it implies further testing (i.e., CTA) may be required

Pulmonary angiography Considered the gold standard for the diagnosis of a PE; It is invasive, not available everywhere, and carries a small mortality risk; Complications more common in elderly patients

What are the treatment goals for a PE? Prevent recurrent PEs; Eliminate any thrombi in the pulmonary vasculature

What are some key points in the initial management of PE? IV-O₂-monitor; If present with shock: fluids/ inotrophic agent; Anticoagulation is the cornerstone of treatment

What are some commonly used anticoagulants for PE? Heparin; Low-molecular-weight heparin (LMWH)

What are two complications of heparin and LMWH? 1. Thrombocytopenia 2. Hemorrhage

What are two commonly used thrombolytics for PE? 1. Streptokinase 2. Tissue plasminogen activator (TPA)

What is an important indication for the use of thrombolytics in the setting of a PE? Hemodynamic instability

CARDIOMYOPATHIES

What is cardiomyopathy? Disease of the myocardium associated with cardiac dysfunction
What are some classifications of cardiomyopathies?  
Dilated cardiomyopathy;  
Hypertrophic cardiomyopathy;  
Restrictive cardiomyopathy

What diagnostic test is commonly used to evaluate cardiomyopathies?  
Echocardiographic evaluation

What is the definition of dilated cardiomyopathy?  
Dilatation and impaired contraction of one or both ventricles, affected patients have impaired systolic function and may or may not develop overt heart failure

What is commonly associated with dilated cardiomyopathy?  
Viral myocarditis

What are some other important causes of dilated cardiomyopathy?  
Idiopathic; Toxins (esp. ethanol/cocaine/lithium); Peripartum; Nutritional deficiencies (thiamine deficiency)

What are some clinical features of dilated cardiomyopathy?  
Signs and symptoms of right and left-sided heart failure such as exertional fatigue, dyspnea, JVD, orthopnea, and ascites

What are some common findings in the following diagnostic tests used for dilated cardiomyopathy:

ECG  
Poor R-wave progression; Atrial or ventricular enlargement; AV block; Atrial fibrillation most common dysrhythmia

CXR  
Cardiomegaly; Pulmonary venous congestion

Echocardiogram  
Decreased ejection fraction; Enlarged heart chambers; Mural thrombi; Abnormal ventricle contraction

What are the key points in the management of dilated cardiomyopathy?  
Alleviation of symptoms; Anticoagulation if mural thrombi or in afib

What are commonly used agents in alleviating symptoms of dilated cardiomyopathy?  
Diuretics, vasodilators, and digitalis

What is the definition of restrictive cardiomyopathy?  
Nondilated ventricles with impaired ventricular filling due to diastolic restriction
What are some important causes of restrictive cardiomyopathy?
- Amyloidosis
- Endomyocardial fibrosis
- Hemochromatosis
- Type II glycogen storage disease

What are some clinical features of restrictive cardiomyopathy?
- Similar to constrictive pericarditis: often will have symptoms of right-sided CHF with exercise intolerance being very common. PE: abnormal heart sounds (S3/S4 gallop), dependent edema, and rales/wheezes

What are some common findings in the following diagnostic tests in restrictive cardiomyopathy:

- **ECG**
  - Commonly show afib; Nonspecific ST-T wave changes; Low voltage

- **CXR**
  - Cardiomegaly can be seen; May initially show a normal heart

- **Echocardiogram**
  - Normal systolic function; Thickened wall; Atria size is greater than ventricle size

What are some points in the management of restrictive cardiomyopathy?
- Commonly use diuretics/digitalis for relief; Vasodilators may decease afterload; Diagnosis is confirmed with biopsy

What is the definition of hypertrophic cardiomyopathy?
- Left ventricular hypertrophy without dilation that often results in impaired diastolic relaxation and can result in decreased cardiac output

What is the most common cause of hypertrophic cardiomyopathy?
- 50% is autosomal dominant inherited

What is the most common presenting symptom of hypertrophic cardiomyopathy?
- Dyspnea on exertion

What are some other clinical features of hypertrophic cardiomyopathy?
- Syncope, dysrhythmias (afib most common), ischemic chest pain, and sudden death (esp. from ventricular fibrillation. PE: systolic ejection murmur especially with valsalva, rapid biphasic carotid pulse, and prominent A wave of neck veins
What are some common findings in the following diagnostic tests:

<table>
<thead>
<tr>
<th>Test</th>
<th>Findings</th>
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<tbody>
<tr>
<td>ECG</td>
<td>Afib and PVCs are common; Changes in anterior, inferior, or lateral leads; Left ventricular hypertrophy (LVH)</td>
</tr>
<tr>
<td>CXR</td>
<td>Typically normal</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>LVH especially with septal hypertrophy; Small left ventricular chamber</td>
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</table>

What are some components in the management of hypertrophic cardiomyopathy?

- Beta-blockers are the mainstay for symptom relief
- Calcium (channel blocker in select patients)
- Amiodarone for ventricular dysrhythmias
- Avoid inotropic agents
- Anticoagulation for afib

What treatment is reserved for severely symptomatic patients who fail medication?

- Septal myomectomy

ENDOCARDITIS

What is the definition of endocarditis?

- Localized infection of the endocardium that is typically characterized by vegetations

What is the pathophysiology of endocarditis?

- Any injury to the endocardium can result in platelet-fibrin complex that can be colonized by organisms such as bacteria or fungus

What are some risk factors of endocarditis?

- Prosthetic valves
- Intravenous drug abuse (IVDA)
- Any acquired or congenital valvular lesions
- Indwelling lines (i.e., shunts or catheters)
- Hemodialysis or peritoneal dialysis

Name two sites that commonly allow entry of bacteria in endocarditis.

1. Oral cavity
2. Genitourinary tract

What are some classes of organisms involved in endocarditis?

- Bacteria (most common)
- Fungi
- Viruses
- Rickettsiae

What are the top three cause of endocarditis in the following situations:

**IVDA or immunocompromised**

- Streptococcus species
- *S. aureus*
- Gram-negative bacteria
Normal valves

Prosthetic valves

What are some important things to know about right-sided endocarditis?

What are some important things to know about left-sided endocarditis?

What are some clinical features of endocarditis?

What are Janeway lesions?

What are Osler’s nodes?

What are some important diagnostic tests to consider in evaluating endocarditis?

Summarize the management of endocarditis?

What factors decide which antibiotic regiment to use?

Streptococcus viridans; S. aureus; Enterococci

Staphylococcus (coagulase negative); Streptococcus viridans; S. aureus

Commonly involves the tricuspid valve; Typically an acute presentation; Very common in IVDA; S. aureus most common agent

Commonly involves the mitral valve; More common in valvular defects; S. viridans and S. aureus most common

Typically nonspecific such as fever, fatigue, weight loss, neurologic complaints, and chest pain. PE: heart murmur, seeding to other sites such as lung (PNA), cutaneous signs (i.e., petechiae), and eye findings (i.e., conjunctival hemorrhages)

Nontender and small erythematous/hemorrhagic nodules in the palms or soles, which are pathognomonic of infective endocarditis. The pathology is due to a type III hypersensitivity reaction

Painful, red, raised lesions on the finger pulps that are indicative of subacute bacterial endocarditis (can be seen elsewhere such as systemic lupus erythematosus

Blood culture: positive in most cases; CBC; CXR; ESR/C-protein: often elevated; Echocardiography: often show vegetations

Empiric Abx typically after cultures drawn; Most patients are typically admitted

The stability of the patient; Resistance of the organism involved; Acuteness of the presentation
MYOCARDITIS

What is the definition of myocarditis?  
Inflammation of the muscles of the heart, often due to infection that is also often associated with acute pericarditis

Name some examples for the following causes of myocarditis:

- **Viruses (most common cause)**
  - Coxsackie A and B; Poliovirus; CMV

- **Bacteria**
  - N. meningitidis; Beta-hemolytic streptococcus; C. diptheriae

- **Parasites**
  - Chagas disease; Trichinosis; Toxoplasmosis

- **Drugs/Toxins**
  - Cocaine; Inhalants; Methyldopa

- **Systemic diseases**
  - Lupus; Kawasaki syndrome; Sarcoidosis

What are some clinical features of myocarditis?  
Highly variable depending on degree of cardiac involvement that can range from chest pain, signs of heart failure, to dysrhythmias and tachycardia. PE: S3/S4 gallop, pericardial friction rub (if pericarditis present), and various murmurs

What history is common to those who present with myocarditis?  
Preceding viral illness in many cases

What are some common findings for the following diagnostic studies that may be used to evaluate myocarditis:

- **ECG**
  - Any type of dysrhythmias may be present; Low-voltage QRS; Nonspecific ST-T wave changes

- **Echocardiography**
  - Dilated chambers; Focal wall motion abnormalities
<table>
<thead>
<tr>
<th><strong>What is the primary presentation of pericardial disease?</strong></th>
<th>The principal manifestations of pericardial disease are pericarditis and pericardial effusion</th>
</tr>
</thead>
</table>
| **What are the two most common causes of pericardial disease?** | 1. Infections (i.e., Coxsackie viruses A and B)  
2. Idiopathic |
| **List some other important causes of pericardial disease.** | Rheumatologic disease (i.e., lupus); Cancer (i.e., metastatic); Radiation; Cardiac injury (i.e., post MI); Medication (i.e., hydralazine) |
| **What are some clinical features of pericarditis?** | Sharp inspirational chest pain that is relieved when leaning forward, low-grade fever, and dyspnea |
| **What physical finding is pathognomonic for pericarditis?** | Pericardial friction rub |
| **What is the best way to elicit a pericardial friction rub?** | Sitting and leaning forward |
| **What is the most common ECG finding?** | Sinus tachycardia (dysrhythmias are rare) |
What are some common findings for the following diagnostic studies that may be used to evaluate pericarditis:

- **CBC**: Often show an elevated white count.
- **ESR/CRP**: Typically elevated due to inflammation.
- **Cardiac enzymes**: May be mildly elevated; Often increase in setting of myocarditis.
- **ECG**: Diffuse ST-segment elevation; Reciprocal ST segment depression in aVR and V1; PR segment depression; Diffuse T wave inversion—late finding.
- **CXR**: Typically normal; May show enlarged silhouette if pericardial effusion >200 mL.
- **Echocardiography**: Test of choice to evaluate effusion; Echo can also assess cardiac function; Can detect as little as 15 mL of effusion.

What are some key points in the management of pericarditis?

- Treat the underlying cause; Pain control with NSAIDs commonly used; Monitor for tamponade and tap if needed.

What are some guidelines to admit patients with pericarditis?

- Serious underlying cause (i.e., MI); Severe pain refractory to medication; Most can be managed on an outpatient basis.

What is the most serious complication of pericardial disease?

- Cardiac tamponade.

What is Beck's triad?

- Jugular venous distension (JVD); Hypotension; Muffled heart sounds.

What are some other common clinical features of cardiac tamponade?

- Dyspnea, narrow pulse pressure, pulse paradoxus, and tachycardia.

What are some common ECG findings in cardiac tamponade?

- Low QRS voltage; Total electrical alternans (beat-to-beat alternating pattern)—not always present.

What are some important differentials to consider in patients with JVD and hypotension?

- Cardiac tamponade; Tension pneumothorax; Massive pulmonary embolism.
What are some causes of tricuspid stenosis? 
Endocarditis secondary to IVDA; Rheumatic fever; Congenital tricuspid atresia; Carcinoid syndrome

What is important to note about tricuspid stenosis? 
Tricuspid stenosis often coexist with other valvular disease (i.e., mitral stenosis)

What are some common clinical features of tricuspid stenosis? 
Systemic venous congestion, fatigue, and dyspnea in some cases. PE: diastolic murmur, ascites, and JVD

What is the most common dysrhythmia associated with tricuspid stenosis? 
Atrial fibrillation

What are some common findings for the following diagnostic studies that may be used to evaluate tricuspid stenosis:

CXR 
May show an enlarged right atrium

ECG 
Tall and pointed P-waves; Afib if present

What are some key points in the management of tricuspid stenosis? 
Treat for afib (rate control/ anticoagulate); Antibiotic prophylaxis when indicated

Tricuspid Regurgitation

What are some causes of tricuspid regurgitation? 
Rheumatic fever; RV dilation due to pulmonary HTN; Infective endocarditis; Trauma

What are some common clinical features of tricuspid regurgitation? 
Dyspnea on exertion, fatigue, anorexia, peripheral edema, and JVD. PE: holosystolic murmur and palpable left ventricular heave
What are some common findings for the following diagnostic studies that may be used to evaluate tricuspid regurgitation:

**CXR**
May show an enlarged right atrium/ventricle; Pulmonary vasculature often normal

**ECG**
Right atrial and ventricular hypertrophy; Incomplete right RBBB; Afib if present

What are some key points in the management of tricuspid regurgitation?
Treat for afib (rate control/anticoagulate); Adequate control of fluid overload and failure symptoms; Surgical intervention for structural deformity

**Mitral Stenosis**

What are some causes of mitral stenosis?
Rheumatic fever (>90% of cases); Left atrial myxoma; Congenital

What are some clinical features of mitral stenosis?
Dyspnea on exertion, hemotysis, fatigue, othopnea, and palpitations. PE: early diastolic opening snap, palpable diastolic thrill, and loud S1

What are some common findings for the following diagnostic studies that may be used to evaluate tricuspid regurgitation:

**CXR**
Pulmonary congestion; Left atrial enlargement

**ECG**
P mitrale (left atrial enlargement); Afib if present

What are some key points in the management of mitral stenosis?
Treat for afib (rate control/anticoagulate); Diuretics for pulmonary congestion; Abx prophylaxis when indicated

**Chronic Mitral Regurgitation**

What are some causes of chronic mitral regurgitation?
Rheumatic fever; Connective tissue disorder; Mitral valve prolapse; Infective endocarditis
<table>
<thead>
<tr>
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<th>Answer</th>
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<tr>
<td>What are some clinical features of chronic mitral regurgitation?</td>
<td>Dyspnea on exertion and orthopnea, but even with severe MR, most are asymptomatic unless LV failure, pulmonary HTN, or afib. PE: S1 is diminished, S3/S4 gallop, and left parasternal heave</td>
</tr>
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<td>What are some common findings for the following diagnostic studies that may be used to evaluate chronic mitral regurgitation:</td>
<td></td>
</tr>
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<td><strong>CXR</strong></td>
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<tr>
<td></td>
<td>Treat for afib (rate control/anticoagulate); Adequate control of fluid overload and failure symptoms; Abx prophylaxis when indicated</td>
</tr>
<tr>
<td><strong>Acute Mitral Regurgitation</strong></td>
<td></td>
</tr>
<tr>
<td>What are some causes of acute mitral regurgitation?</td>
<td>Myocardial infarction; Trauma; Infective endocarditis</td>
</tr>
<tr>
<td>What structures associated with the mitral valve can be damaged?</td>
<td>Papillary muscle; Valve leaflet; Chordae tendineae</td>
</tr>
<tr>
<td>What are some clinical features of acute mitral regurgitation?</td>
<td>Dyspnea on exertion and orthopnea, but will often present as fulminant CHF and symptoms of the cause of the rupture (i.e. MI). PE: S1 is diminished, S3/S4 gallop, and left parasternal heave</td>
</tr>
<tr>
<td>What are some common findings for the following diagnostic studies that may be used to evaluate acute mitral regurgitation:</td>
<td></td>
</tr>
<tr>
<td><strong>CXR</strong></td>
<td>Often have a normal cardiac silhouette; Evidence of severe pulmonary edema</td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>Often show sinus tachycardia; May also show evidence of MI, if the cause</td>
</tr>
<tr>
<td>What are some key points in the management of mitral regurgitation?</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Oxygen and afterload reduction; Adequate control of fluid overload and failure symptoms; Emergent consult with CT surgery</td>
</tr>
</tbody>
</table>
## Mitral Valve Prolapse

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important things to know about mitral valve prolapse?</td>
<td>Most common valvular heart disease; More common in young females; Present in up to 10% of population</td>
</tr>
<tr>
<td>What are some causes of mitral valve prolapse?</td>
<td>Idiopathic; Associated with tissue connective disorder; Autosomal dominant congenital disorder</td>
</tr>
<tr>
<td>What are some clinical features of mitral valve prolapse?</td>
<td>Palpitations, syncope, chest pain, or can be asymptomatic. PE: high-pitched late systolic murmur or late systolic click</td>
</tr>
<tr>
<td>What are some complications to consider for mitral valve prolapse</td>
<td>Sudden death (very rare); CHF (due to severe regurgitation); Embolization</td>
</tr>
</tbody>
</table>
| What are some common findings for the following diagnostic studies that may be used to evaluate aortic stenosis: | **CXR**
Typically normal unless severe regurgitation

**ECG**
Typically normal; May show T-wave changes in inferior lead; May show QT prolongation

**What are some key points in the management of mitral valve prolapse?**
Abx prophylaxis when indicated (usually if with injury); Beta-blockers for chest pain/dysrhythmias; Anticoagulation for suspected embolization

## Aortic Stenosis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some causes of aortic stenosis?</td>
<td>Congenital bicuspid valve; Rheumatic heart disease; Calcific aortic disease</td>
</tr>
<tr>
<td>When do patients generally become symptomatic with aortic stenosis?</td>
<td>Most are asymptomatic until very late in the disease—valve opening decreases &lt;1 cm</td>
</tr>
<tr>
<td>What are some clinical features of aortic stenosis?</td>
<td>Syncope, chest pain, dyspnea on exertion, sudden death, and symptoms of heart failure PE: harsh systolic murmur (crescendo-decrescendo), narrow pulse pressure, and diminished carotid upstroke</td>
</tr>
</tbody>
</table>
### Aortic Stenosis

**What are some common findings for the following diagnostic studies that may be used to evaluate aortic stenosis?**

<table>
<thead>
<tr>
<th>Study</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>CXR</td>
<td>Aortic calcification; Left ventricular enlargement; Poststenotic dilatation of the aorta</td>
</tr>
<tr>
<td>ECG</td>
<td>Left ventricular hypertrophy; Left or right BBB</td>
</tr>
</tbody>
</table>

**What are some key points in the management of aortic stenosis?**

- Symptomatic patients referred for either valve replacement or valvuloplasty
- Admit patients in CHF
- Abx prophylaxis when indicated

### Chronic Aortic Regurgitation

**What are some causes of chronic aortic regurgitation?**

- Rheumatic heart disease
- Connective tissue disorder
- Bicuspid valve
- Infective endocarditis
- Tertiary syphilis

**What are some clinical features of chronic aortic regurgitation?**

- Dyspnea on exertion, orthopnea, fatigue and palpitations
- S1 is diminished, wide pulse pressure, high-pitched decrescendo blowing murmur, and displaced PMI
- Often have cardiomegaly
- Pulmonary root congestion
- Aortic root dilation

**What are some common findings for the following diagnostic studies that may be used to evaluate chronic aortic regurgitation?**

<table>
<thead>
<tr>
<th>Study</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>CXR</td>
<td>Often have cardiomegaly; Pulmonary root congestion; Aortic root dilation</td>
</tr>
<tr>
<td>ECG</td>
<td>Left ventricular hypertrophy; Sometimes an LBBB can be seen</td>
</tr>
</tbody>
</table>

**What are some key points in the management of chronic aortic regurgitation?**

- Adequate control of fluid overload and failure symptoms (treat as CHF)
- Abx prophylaxis when indicated

### Acute Aortic Regurgitation

**What are some causes of acute aortic regurgitation?**

- Aortic dissection
- Trauma
- Infective endocarditis
<table>
<thead>
<tr>
<th><strong>What are some clinical features of acute aortic regurgitation?</strong></th>
<th>Severe dyspnea on exertion, signs of heart failure, and chest pain. PE: low blood pressure, tachycardia, normal pulse pressure, midsystolic flow murmur, and low CO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What are some common findings for the following diagnostic studies that may be used to evaluate acute aortic regurgitation:</strong></td>
<td><strong>CXR</strong></td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>Often show sinus tachycardia; Left ventricle strain; Nonspecific ST-T wave change</td>
</tr>
<tr>
<td><strong>What are some key points in the management of acute aortic regurgitation?</strong></td>
<td>Determine cause and treat; Adequate control of fluid overload and failure symptoms; Emergent consult with CT surgery for valve replacement</td>
</tr>
</tbody>
</table>

### Prosthetic Valves

<table>
<thead>
<tr>
<th><strong>What are two types of prosthetic valves commonly used?</strong></th>
<th>1. Mechanical valves 2. Bioprosthetic valves (porcine or bovine)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What are some important points regarding mechanical valves?</strong></td>
<td>Typically made from carbon alloys; Most mechanical valves last 20–30 years and metallic noise can be heard; Life-long anticoagulation required; Greater hemolysis than tissue valves</td>
</tr>
<tr>
<td><strong>What are some important points regarding bioprosthetic valves?</strong></td>
<td>Can be human, bovine, or porcine tissue; Typically last &lt;10 years; Closure noise similar to native valves; Anticoagulation required in some situations; Less hemolysis than mechanical valves</td>
</tr>
<tr>
<td><strong>What is the most serious complication of prosthetic valves?</strong></td>
<td>Thromboembolic events</td>
</tr>
<tr>
<td><strong>What are some other complications of prosthetic valves?</strong></td>
<td>Structural failure; Bleeding; Embolization; Hemolytic anemia; Valvular obstruction (from thrombus)</td>
</tr>
</tbody>
</table>
# THORACIC AORTIC DISSECTION

What is the epidemiology of thoracic aortic dissection (TAD)?

- Males are affected more than females; Most patients affected are over 50 years; TAD are more common than AAA

What is the pathophysiology of TAD?

- Degeneration of the aortic media, or cystic medial necrosis, that leads to a tear in the aortic intima. Propagation of the dissection to various areas (i.e., coronary artery is the feared concern)

What are two factors that determine the rate of dissection propagation?

- 1. Blood pressure
- 2. Rate of ventricular contraction

What is the biggest risk factor for the development of TAD?

- Uncontrolled blood pressure

What are some other important risk factors of TAD?

- Connective tissue disorders—Marfan’s; Congenital heart disease; Turner’s syndrome; Infections (i.e., syphilis); Drugs that raise BP (i.e., cocaine); Trauma

What are the two major classification systems used to classify TAD based on location of dissection?

- 1. Stanford
- 2. DeBakey

What is the Stanford classification:

- Type A: Ascending aorta
- Type B: Descending aorta

What is the DeBakey classification:

- Type I: Ascending aorta and part distal aorta
- Type II: Ascending aorta only
- Type III: Descending aorta only
  - Subtype IIIA: Dissection above the diaphragm
  - Subtype IIIB: Dissection below the diaphragm

What is the mortality rate for untreated TAD once the dissection begins:

- 1 day: 33%
- 2 days: 50%
- 2 weeks: 75%
- 1 month: Approaches 90%
What is the character of chest pain in TAD?

Chest pain that is abrupt and maximal at onset, migrates as the dissection progresses that is typically described as tearing with radiation to jaw/arm/back.

What are some other clinical features of TAD based on the location of the dissection?

Abdominal pain (mesenteric ischemia); Flank pain/GU symptoms (< renal flow); CVA (dissection of carotid artery); MI (dissection of coronary artery); CHF; Syncope; Spinal cord deficits.

What are some important physical findings that help to establish the diagnosis of TAD?

Focal neurological deficits, a 20 mm Hg extremity BP difference, and unequal or absent pulses between extremities.

What is the clinical significance of a “silent” TAD?

“Silent” TAD is not that uncommon and must be distinguished from MI/CVA as the use of lytics would be disastrous.

What are some important points for the following initial tests that should be undertaken:

- **CXR**
  - Should be done immediately and upright; CXR will almost be abnormal in TAD; Mediastinal widening (>8 cm) common; Other common findings include loss of aortic knob, deviation of trachea, effusion, etc.

- **ECG**
  - Will be abnormal in most cases; Changes seen in MI is common; LV hypertrophy is common as well; Inferior wall MI most common pattern.

Name four studies that are commonly used to confirm the diagnosis of TAD?

1. MRI
2. Aortography
3. Transesophageal echocardiography
4. CT

What is the test of choice at most institutions as it is noninvasive, inexpensive, and fast?

TEE (CT is done in most cases when TEE cannot be done).

What is important initial management for any patient with suspected TAD?

Control of BP (i.e., nitroprusside); Control HR (i.e., beta-blocker); Avoid anticoagulants/lytics.
What are some key points in the treatment of TAD?

Immediate CT consultation; If hypotensive—small fluid bolus; Ascending dissection = surgery; Descending dissection = medical; Pain control with narcotics

ABDOMINAL AORTIC ANEURYSMS

What are some important things to know about abdominal aortic aneurysms (AAA)?

Involve all layers of the aorta; Most AAA occur below the renal arteries; Ruptured AAA is an emergency

What is the diameter of the aorta that is considered pathologic?

Diameter >3.0 cm is generally considered aneurysmal

What is the pathophysiology of AAA?

Aortic aneurysms are caused by a progressive weakening of the aortic wall which results in a dilatation. The aneurysm will grow progressively larger and eventually rupture if it is not diagnosed and treated

What are some risk factors for the development of AAA?

Age (most occur in >70 years); Male gender; History of smoking; Hypertension; Family history in first degree relatives; History of CAD or PVD

What are some clinical features of AAA rupture?

Classic presentation is sudden onset of severe abdominal, back, or flank pain that may be associated with syncope. Pain can radiate to the testicles/labia as well

What is the most common misdiagnosis of AAA rupture?

Kidney stone

What are some important physical findings that help to establish the diagnosis of AAA rupture?

A ruptured AAA will often have a tender pulsatile mass in the epigastric area, bruits, and signs of distal extremity ischemia

What are some important points for the following diagnostic tests commonly utilized for AAA:

Plain abdominal film

Not very accurate for AAA; May show aneurysmal calcification; Does not confirm/exclude diagnosis
### Ultrasonography

Can be utilized on unstable patients; Inexpensive, fast, sensitive; Can only detect aneurysm, not leaks; Limited by adipose tissue and gas.

### CT contrast with contrast

Very accurate and sensitive; Can also detect other abnormalities; Negative: IV contrast and long study test; Not to be used on unstable patients.

### What is the initial management for any patients with suspected AAA rupture?

IV O₂-monitor; Aggressive fluid resuscitation; Type and cross for 5–10 units; ECG; Immediate surgical consultation.

### HYPERTENSIVE URGENCIES AND EMERGENCIES

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of hypertensive urgency?</td>
<td>Severe hypertension (often defined by systolic blood pressure ≥180 mm Hg and/or diastolic blood pressure ≥120 mm Hg) without any evidence of end-organ damage</td>
</tr>
<tr>
<td>What is the most common cause of hypertensive urgency?</td>
<td>Nonadherence to antihypertensives</td>
</tr>
<tr>
<td>What is a consequence of aggressive blood pressure reduction?</td>
<td>CVA (due to fall below autoregulation)</td>
</tr>
<tr>
<td>What are some key points in the management of hypertensive urgency?</td>
<td>Slowly bring down the patient’s BP; Ensure patient’s compliance to medicines</td>
</tr>
<tr>
<td>What is the definition of hypertensive emergency?</td>
<td>Marked increase in blood pressure, generally ≥180/120 mm Hg, with evidence of end-organ damage</td>
</tr>
<tr>
<td>What is the pathophysiology of hypertensive emergency?</td>
<td>Initial response is arterial and arteriolar vasoconstriction, autoregulatory process both maintains tissue perfusion at a relatively constant level. Increasingly severe hypertension will result in failure of autoregulation</td>
</tr>
<tr>
<td>Name two characteristics of hypertension encephalopathy?</td>
<td>It is reversible and acute in onset</td>
</tr>
</tbody>
</table>
What are some clinical features of hypertensive encephalopathy?

Confusion, severe headache, focal neurologic deficits, or coma

What are two eye findings to look for with a patient who present with hepatic encephalopathy?

1. Papilledema
2. Hypertensive retinopathy

CLINICAL VIGNETTES

23-year-old female with no past medical history presents with unilateral left leg swelling soon after a trip to Mexico, but otherwise has been well; PE: unilateral swelling of left calf with a (+) Homans’ sign

Deep vein thrombosis

45-year-old male with a history of DM and HTN is brought over via EMS for altered mental status, confusion and minimal response, and they mention patient has a long history of poor medication compliance; PE: BP is taken at 240/180, with otherwise unremarkable PE

Hypertensive emergency

71-year-old female with Hx of HTN presents with syncope whenever she exerts herself, but otherwise no other complaints; PE: harsh systolic murmur (crescendo-decrescendo), narrow pulse pressure, and diminished carotid upstroke

Aortic stenosis

23-year-old male with Hx of IVDA presents with fever, fatigue, and weight loss for the past 2 weeks; PE: heart murmur, petechiae, and conjunctival hemorrhages

Infective endocarditis

18-year-old male with no PMH presents with a sudden syncopal episode during soccer practice, but otherwise is now feeling fine in the ED; PE: rapid biphasic carotid pulse and systolic ejection murmur; ECG: left ventricular hypertrophy

Hypertrophic cardiomyopathy

61-year-old male with Hx of DM and HTN presents with “chest pressure” for about 2 hours associated with dyspnea, diaphoresis, nausea, and radiation of pain to jaw; PE: unremarkable exam except patient is anxious; Labs: elevated cardiac enzymes; ECG: ST-depression in inferior leads (II, III, and aVL)

NSTEMI
55-year-old female with Hx of breast cancer presents with pleuritic chest pain and dyspnea on exertion for 3 days, but is otherwise stable; PE: tachycardia, but otherwise unremarkable exam; CXR: clear fields except for regional oligemia

51-year-old male with Hx of uncontrolled hypertension presents with tearing chest pain with radiation to the back that has been refractory to nitro; PE: 20 mm Hg extremity BP difference, and unequal or absent pulses between extremities

21-year-old female presents with CP that she describes as sharp and more painful on deep inspiration, but relieved when she leans forward; PE: friction heard on cardiac exam; ECG: depressed PR interval and diffuse ST segment elevation

73-year-old male with history of DM, HTN, and smoking presents with a sudden onset of abdomen pain with radiation to the left flank as well as his testicles; PE: pulsatile mass in the epigastric area as well as abdominal bruits

56-year-old male with Hx of DM and HTN presents with CP. Patient mentions that he previously had CP only when he did any moderate activity and was relieved by rest and his nitro, but now he gets his CP when he is at rest, but it still does not last more than 5 minutes or so

71-year-old female with no cardiac history presents as a transfer from an outside hospital for chest pain. She mentions that her CP started about 3 hours ago and says it is substernal associated with diaphoresis and nausea; PE: unremarkable; ECG 3-mm ST-elevations in V1–V4

31-year-old female who recently arrived from South America presents with chest pain and recalls that it started about a week after her cold; PE: S3/S4 gallop with a pericardial friction rub; ECG: low-voltage

Pulmonary embolism
Thoracic aortic dissection
Pericarditis
Aortic abdominal aneurysm
Unstable angina
Anterior STEMI
Myocarditis
ESOPHAGUS

Anatomy

What are some important anatomic points to know about the esophagus?
It is 25-cm in length; Upper third is striated muscle; Lower two-thirds is smooth muscle

What are the major nerves of the extrinsic nervous system that innervate the esophagus?
Vagus nerve; Sympathetic fibers; Spinal accessory nerve

What are the two major plexuses that are found within the esophagus?
1. Meissner’s plexus
2. Auerbach’s plexus

What are the three layers of the esophagus?
1. Inner mucosa
2. Submucosa
3. Muscle layer

What is the clinical significance of the lack of serosa?
Any compromise of the submucosa will lead to diffuse rapid mediastinitis

Dysphagia/Odynophagia

What is the definition of dysphagia?
It is a subjective experience that ranges from the inability to swallow to the sensation of food “stuck” in the esophagus

What is the definition of odynophagia?
The sensation of pain when swallowing
What are some important elements in the history to obtain in dysphagia?

Whether it is acute versus chronic; Dysphagia to food or liquids (or both); Intermittent versus progressive

What are some important points for the two categories of dysphagia:

**Transport dysphagia**
Problem typically lies at the esophagus; Often patient will complain of a sticky sensation; Commonly due to anatomical problems

**Transfer dysphagia**
Problem typically is at the oropharynx; Difficulty in transfer of foul bolus to esophagus; Commonly due to neuromuscular problems

List some common anatomical problems that may result in dysphagia.
Strictures (i.e., radiation injury); Malignancy; Webs; Diverticula

List some common neuromuscular problems that may result in dysphagia.
Achalsia; Spasms; Neurological insults (i.e., stroke)

What are some key points in the management of dysphagia?
Ensure the patient is stable; Often requires various tests (i.e., EGD, barium swallow, etc); A careful history is paramount

What is a common cause of odynophagia?
Esophagitis

What are some clinical features of odynophagia?
Pain on swallowing and chest pain (important to distinguish from cardiac pain)

What are two main causes of odynophagia?
1. Infectious
2. Inflammatory

List some examples that may cause infectious esophagitis?
Candida; Herpes simplex virus (HSV); Cytomegalovirus (CMV); Aphthous ulceration

List some examples that may cause inflammatory esophagitis?
Medication (i.e., nonsteroidal anti-inflammatory drugs [NSAIDs] and antibiotics); GERD

What are some key points in the management of esophagitis?
If chest pain, distinguish from cardiac origin; Monitor for bleed, perforation, and obstruction; Typically managed on an outpatient basis

---

**Gastroesophageal Reflux Disease**

What is the definition of gastroesophageal reflux disease (GERD)?
Reflex of stomach acid typically from transient relaxation of LES or a weak LES
### What are some complications of GERD?
- Esophageal erosions; Esophageal strictures; Barrett’s esophagus; Esophageal cancer

### Name some major causes of GERD?
- Decrease in esophageal motility (achalasia); Prolonged gastric emptying (obstruction); Transient decrease in LES tone (diet)

### What are some clinical features of GERD?
- Dysphagia, odynophagia, heartburn, asthma exacerbation, and presentation that may be similar to heart ischemic (squeezing pain, pain radiation, and nausea/vomiting)

### What are some things that may exacerbate GERD?
- Meals are often a major factor; Medication; Supine position

### What are some key points in the management of GERD?
- Avoid triggers (i.e., eating before bed); H₂-blockers and proton-pump inhibitors (PPI)

---

### Esophageal Perforation

<table>
<thead>
<tr>
<th>What are some causes of esophageal perforation?</th>
<th>Chest trauma; Iatrogenic (endoscopy); Swallowing (object); Sudden increase in intra-abdominal pressure such as emesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the most common cause of esophageal perforation?</td>
<td>Iatrogenic</td>
</tr>
<tr>
<td>What is Mallory-Weiss syndrome?</td>
<td>It is a partial thickness tear along the esophagus</td>
</tr>
<tr>
<td>What are some clinical features of Mallory-Weiss syndrome?</td>
<td>Mild self-limiting upper GI bleeding, dysphagia, and odynophagia</td>
</tr>
<tr>
<td>What are some risk factors for Mallory-Weiss syndrome?</td>
<td>Hiatal hernia; EtOH abuse; Esophagitis</td>
</tr>
<tr>
<td>What is Boerhaave’s syndrome?</td>
<td>It is perforation of the esophagus</td>
</tr>
<tr>
<td>What are some of the clinical features of Boerhaave’s syndrome?</td>
<td>Severe tearing chest pain that often radiates to the back/neck. PE: mediastinal crunch and epigastric tenderness</td>
</tr>
<tr>
<td>What are complications of Boerhaave’s syndrome?</td>
<td>Mediastinitis (high mortality); Sepsis</td>
</tr>
</tbody>
</table>
What are some diagnostic tests used in Boerhaave’s syndrome and their typical findings:

- **Chest x-ray (CXR)**
  - Widened mediastinum, left pneumothorax, left pleural effusion, and mediastinal emphysema

- **Esophagram (water-soluble contrast)**
  - Leakage of content into the mediastinal area

What are some key points in the management of esophageal rupture?

- Aggressive fluid resuscitation;
- Intravenous (IV) antibiotics;
- Surgical consult

---

**Swallowed Foreign Body**

What are some important things to know about a swallowed foreign body?

- 80% of complaints are in the pediatric population; Most ingestions do pass through the GI tract without intervention or problems; Typically 1500 die per year from object ingestion

What type of foreign bodies are most commonly swallowed by children?

- True foreign objects such as coins

What type of foreign bodies are most commonly swallowed by adults?

- Food impactions more common

What portion of the esophagus do most objects get lodged in children?

- Cricopharyngeal area

What portion of the esophagus do most objects get lodged in adults?

- Distal portion of the esophagus

What are some clinical features of a swallowed foreign body?

- Dysphagia, foreign body sensation, gagging, emesis, and possible respiratory distress

What is the diagnostic test of choice in a swallowed foreign body?

- Plain films with at least two views; Endoscopy (diagnostic and therapeutic); Esophagogram (if perforation is suspected)

What are three complications of foreign body impaction?

1. Obstruction
2. Esophageal perforation
3. Esophageal strictures

What is the probability that a foreign body will pass completely once past the gastroesophageal junction?

- 90%

What is typically done for proximal impactions of the esophagus?

- Removal of object via endoscopy
What are some commonly used medications to help with passage of distal esophagus impaction?  
- Nifedipine; Sublingual nitroglycerin  

About what percentage of foreign bodies that are lodged and cannot be removed require surgical intervention?  
- 1%  

What are some key points in the management of a swallowed foreign bodies?  
- Most can be managed expectantly; Lodged sharp objects mandate removal; Most foreign bodies are cleared in 2–3 days; Swallowed batteries also mandate removal  

### GASTROINTESTINAL BLEEDING

What are some important epidemiologic information about GI bleeding?  
- Common, but potentially life-threatening; Upper GI bleeding is more common in elderly males; Mortality rises with age  

What are some factors that are associated with high mortality in GI bleeding?  
- Advanced age; Coexisting organ disease; Hemodynamically unstable; Repeated hematemesis/hematochezia  

What defines upper GI bleeding?  
- Bleeding that is proximal to the ligament of Treitz  

What are some important things to know about each of the important causes of upper GI bleeding:

- **Gastric and esophageal varices**  
  - Commonly from portal hypertension; Very high rebleed rate and mortality rate; Comprise small number of upper GI bleeds  

- **Peptic ulcer disease**  
  - Includes gastric, and duodenal ulcers; Most common cause of upper GI bleed; Gastric ulcers higher rebleed rate than duodenal ulcers  

- **Mallory-Weiss syndrome**  
  - Longitudinal tear of esophagus; Classically hematemesis following retching; Seizures and coughing are risk factors  

What are some other less common causes of upper GI bleeding?  
- Arteriovenous malformation; Malignancy; Aortoenteric fistula
What is the most common cause of apparent lower GI bleeding?  
Upper GI bleeding

What is the most common cause of actual lower GI bleeding?  
Hemorrhoids

What are other some common causes of lower GI bleeding?  
Inflammatory (i.e., inflammatory bowel disease)  
Neoplasm (i.e., colon cancer)  
Other (i.e., hemorrhoids)  
Vascular (i.e., arteriovenous malformation [AVM])  
Anatomical (i.e., diverticulosis)

What are some important elements to obtain in a patient who presents with GI bleeding?  
Characterize the bleeding; Changes in bowel habits and weight loss; Retching and vomiting; History of medication (i.e., NSAIDs); Alcohol use; Ingestion of bismuth or iron

What are some elements on the physical exam to consider?  
Vitals (i.e., decreased pulse pressure); Stigmata of liver disease (i.e., jaundice); Abdominal examination; Rectal exam

What are some laboratory data to consider in GI bleeding?  
Type and cross-match blood; Complete blood count (CBC); Coagulation studies; Liver panel; Chem-7 (i.e., BUN can be elevated)

What are some diagnostic studies to consider in GI bleeding?  
CXR and abdominal films (low yield); Endoscopy (EGD) and colonoscopy; Scintigraphy

What is the most accurate test to perform in upper GI bleeding?  
EGD

Why is EGD evaluation useful in upper GI bleeding?  
Diagnostic and therapeutic such as band ligation of esophageal varices

What are some important management points with patients who present with GI bleeding?  
GI bleeding is potentially life-threatening; Immediate resuscitation (fluids and blood); Neogastric (NG) tube placement is important

What role does somatostatin or octreotide play in GI bleeding?  
Effective in reducing bleeding from esophageal varices and peptic ulcers, as effective as vasopressin without the adverse drug reaction
Should patients with upper GI bleeding be placed on a proton pump inhibitor? | Omeprazole shown to reduce rebleeding, need for surgery with PUD, and reduce transfusion requirement
---|---
What role does balloon tamponade play in GI bleeding? | Sengstaken-Blakemore tube can control variceal hemorrhage

**PEPTIC ULCER DISEASE**

<table>
<thead>
<tr>
<th>What is the definition of peptic ulcer disease (PUD)?</th>
<th>PUD is a chronic disease that is typically caused by defects in the mucosal barrier most commonly along the lesser curvature of the stomach and duodenum</th>
</tr>
</thead>
</table>
| What are the two most common causes of PUD? | 1. NSAIDs  
2. *Helicobacter pylori* |
| List some other predisposing factors for the development of ulcers. | Zollinger-Ellison syndrome; Cigarette smoking; Long-term steroid use; Stress |
| What are some important things to know about the following types of ulcers: | Gastric ulcers: Damage is from mucosal breakdown; *H. pylori* is found in over 75% of cases; Pain is typically shortly after eating  
Duodenal ulcers: Damage is usually from acid hypersecretion; *H. pylori* is found in over 90% of cases; Pain is typically 2–3 hours after meals  
Stress ulcers: Commonly due to acute trauma/CNS tumors; Usually located on fundus/body of stomach; Very common cause of gastric bleeding |
| What conditions are *H. pylori* usually implicated in? | PUD; Lymphoid hypertrophy; Adenocarcinoma of stomach; Gastric lymphoma |
| What are some common diagnostic methods used to identify *H. pylori*? | Serology; Endoscopy (i.e., rapid urea); Urea breath test |
| What are some clinical features of PUD? | Epigastric pain that is vague and described as “burning” often relieved by food |
What are some important diagnostic tests to consider in PUD?

Endoscopy; Barium-contrast x-ray

What are some key points in the management of uncomplicated PUD?

Avoid exacerbating factors (i.e., NSAIDs); Antacids—symptomatic relief; H₂-blockers (cimetidine); Proton pump inhibitors (omeprazole); Eradication of *H. pylori*

What is the importance in the eradication of *H. pylori*?

Reduces the recurrence of PUD; Reduces need for suppressive therapy

What are three drugs commonly used as triple therapy in the eradication of *H. pylori*?

1. Macrolide (clarithromycin)
2. Tetracycline
3. Omeprazole

What are some complications of PUD?

Bleeding; Perforation; Outlet obstruction

What are some key points in the management of the following complications of PUD:

Upper GI bleeding

Volume replacement and transfuse if needed; Administer PPI or H₂-blocker; Nasogastric (NG) tube drainage; GI or surgery consult in severe case

Perforation

Monitor for peritoneal signs; X-ray evidence of free air; IV fluids/ABx/NG tube drainage; Surgical consultation

Gastric outlet obstruction

Healed ulcer scar that blocks pyloric outlet; Endoscopy; Upright abdominal plain film; IV fluids/NG tube suction and admit

**APPENDICITIS**

What is the definition of appendicitis?

It is inflammation of the appendix due to obstruction of the outlet

What are some important things to know about appendicitis?

Most common cause of emergent surgery; Highest incidence in males 10–30 years of age
What is the pathophysiology of appendicitis? Obstruction of the lumen that leads to intraluminal distension, venous congestion, and eventually ischemia followed by perforation (bacterial invasion common)

What are some common causes of appendiceal obstruction? Fecalith—most common; Enlarged lymphoid follicles; Tumors; Adhesions

What are the most common symptoms of appendicitis? Abdominal pain (periumbilical then right lower quadrant); Anorexia; N/V (should occur after pain); Fever and chills

What are some common signs of appendicitis? Abdominal tenderness with/without rebound; Rosving’s sign (RLQ pain when pressing left lower quadrant [LLQ]); Psoas sign (Passive extension of right hip that causes RLQ pain); Obturator sign (Passive internal rotation of flexed hip causes RLQ pain); Cervical motion tenderness (seen in pelvic inflammatory disease)

How common is the classic migratory pain with associated symptoms in appendicitis? Found in up to 2/3 of patients with appendicitis

What is the concern in a patient with suspected appendicitis who has a sudden decrease in pain followed by a dramatic increase in pain? Perforation

What other conditions can appendicitis mimic? Nephrolithiasis; Pelvic inflammatory disease; Right upper quadrant pain in pregnant women; Ectopic pregnancy

What are some important diagnostic tests to consider in appendicitis? Pregnancy test—rule out ectopic pregnancy; Complete blood count (CBC)—elevated WBC is typical; Plain abdominal films—may show fecalith; CT with IV and rectal/oral contrast—first choice; Ultrasound (U/S)—useful for children/pregnant women

What are some key points in the management of appendicitis? NPO and IV fluids; Pain control; Early surgical consult if suspicion is high; If surgery—prophylactic antibiotics (Abx)
# GALLBLADDER DISEASE

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of cholecystitis?</td>
<td>Acute inflammation of the gallbladder that is commonly caused by obstruction at the neck of the gallbladder or cystic duct</td>
</tr>
<tr>
<td>What are some important things to know about cholecystitis?</td>
<td>It is more common in females; Most cases (&gt;90%) due to cystic stones; One of the most common indication for surgery</td>
</tr>
<tr>
<td>What are some risk factors for the development of cholelithiasis (hence cholecystitis)?</td>
<td>Obesity; Female; Rapid weight loss; Advanced age; Cystic fibrosis; Long-term TPN use</td>
</tr>
<tr>
<td>What are some important points for each of the following types of gallbladder disease:</td>
<td></td>
</tr>
<tr>
<td>Calculous cholecystitis</td>
<td>It is the most frequent variant; Most common cause of pancreatitis</td>
</tr>
<tr>
<td>Acalculous cholecystitis</td>
<td>It makes up about 5–10% of cases; More common in elderly, DM, and sepsis; Perforation and gangrene are more common</td>
</tr>
<tr>
<td>Ascending cholangitis</td>
<td>Extending infection into the liver; Charcot’s triad: fever/jaundice/RUQ pain; Reynold’s pentad: Charcot’s Triad plus shock/ΔMS; Requires rapid surgical intervention</td>
</tr>
<tr>
<td>Gallstone ileus</td>
<td>Uncommon cause of bowel obstruction; Gallstone erodes through the gallbladder and impacts in bowel near the cecum; More common in elderly females</td>
</tr>
<tr>
<td>Emphysematous cholecystitis</td>
<td>Rare infection of the gallbladder; Agents usually include anaerobes/gram (–)</td>
</tr>
<tr>
<td>What are some clinical features of cholecystitis?</td>
<td>RUQ pain, fatty food intolerance, gallstone risk factors, N/V, fever, and tachycardia</td>
</tr>
<tr>
<td>What is Murphy’s sign?</td>
<td>It is increase in pain and temporary cessation of breathing when direct pressure is applied to RUQ when the patient takes a deep breath</td>
</tr>
</tbody>
</table>
### What are some important diagnostic studies to consider in cholecystitis?

**CBC**—elevated WBC is typical; 
**Liver function test (LFT)**—enzymes and alkaline phosphate may be elevated; **Amylase/lipase**—increased if pancreatitis; **Abdominal plain films**: typically normal

### What are the typical findings in the following imaging modalities in the assessment of cholecystitis:

- **Ultrasound**: The study of choice where common findings include presence of gallstones, gallbladder wall thickening (>5 mm), pericholecystic fluid, and dilated common ducts.

- **Biliary scintiscanning (HIDA)**: Typically used if U/S results are indeterminate and clinical suspicion is high; Positive results typically show lack of isotopes in the gallbladder.

- **CT**: It is not any more sensitive or specific when compared to U/S and exposes patient to significant amount of radiation.

### What are some key points in the management of cholecystitis?

- **NPO/IV fluids/NG tube if needed**; **Broad-spectrum Abx**; **Surgical consult**; **Pain control**

### What are some general criteria for admission?

- Fever, significant abdominal pain, elevated WBC; Complications (i.e., ascending cholangitis); Cholecystectomy (usually within 72 hours)

---

### PANCREATITIS

**What is the definition of pancreatitis?**

It is acute inflammation of the pancreas

**What are the two most common causes of acute pancreatitis?**

1. Alcohol abuse 
2. Bile duct disease (gallstone)

**What are some other causes of acute pancreatitis?**

- Surgery; ERCP; Hyperlipidemia; Hypercalcemia
What are some clinical features of acute pancreatitis?
Epigastric pain typically after ingestion of EtOH or a fatty meal, N/V, low-grade fever, and tachycardia

What is Grey Turner's sign?
Bluish discoloration of the flank

What is Cullen's sign?
Bluish discoloration near the umbilicus

What do these two signs point to?
Although not common, they indicate the presence of hemorrhagic pancreatitis

What is chronic pancreatitis?
It is progressive, irreversible structural changes due to repeated bouts of acute pancreatitis commonly due to EtOH

Give some important features of the following diagnostic tests:

Amylase
Amylase is also found in other organs; 1.5 above upper limit points to pancreatitis

Lipase
More specific and as sensitive as amylase; Lipase is found primarily in the pancreas; It is reliable and inexpensive

CBC
Low hematocrit points to hemorrhagic pancreatitis; High WBC is common

What is Ranson's Criteria?
It is a set of prognostic factors that correlate with mortality based on the number of prognostic signs that are met

On admission
Age >55 years; Hyperglycemia >200 mg/dL; Leukocytosis >16,000 per mm³; LDH >350 IU/L; AST >250

After 48 hours
PO₂ <60 mm Hg; Calcium <8 mg/dL; Hct >10% drop; Base deficit >4 mEq/L; Sequestration >4 L of fluid; BUN >5 mg/dL

What are some complications of acute pancreatitis?
Abscess; Hemorrhagic; Fluid sequestration; Acute respiratory distress syndrome (ARDS)
### COLITIS AND ILEITIS

#### Crohn's Disease

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of crohn's disease (CD)?</td>
<td>Crohn’s disease is a chronic, recurrent inflammatory disease of the intestinal tract (primarily the ileum and colon)</td>
</tr>
<tr>
<td>What is the epidemiology of CD?</td>
<td>Greater incidence in whites between the age of 16–40, more likely to affect Jews and a positive family history in up to 20%</td>
</tr>
<tr>
<td>What are some clinical features of CD?</td>
<td>Recurrent abdominal pain, fever, and diarrhea with weight loss. RLQ that mimic appendicitis is also not uncommon</td>
</tr>
<tr>
<td>What are some extraintestinal manifestations of the following organ systems in CD?</td>
<td>Dermatology. Pyoderma gangrenosum; Erythema nodosum Ophthalmic. Iritis; Conjunctivitis; Uveitis Rheumatology. Ankylosing spondylitis; Arthritis Vascular. Arteritis; Thromboembolic disease; Vasculitis Hepatobiliary. Gallstones; Pericholangitis</td>
</tr>
<tr>
<td>What are some complications of CD?</td>
<td>Strictures; Perforation; Perianal complications; Abscess; Fistulas</td>
</tr>
<tr>
<td>What is the diagnostic test of choice for CD?</td>
<td>Colonoscopy with histological sample</td>
</tr>
<tr>
<td>What are some key points in the management of CD?</td>
<td>IV fluids and NG tube; Steroids to reduce inflammation; Azathioprine—steroid sparing; Metronidazole for perianal complications; Infliximab may help in severe cases</td>
</tr>
<tr>
<td>What are some indications for admission for CD?</td>
<td>Acute complications; Unable to keep PO; Severe exacerbation</td>
</tr>
</tbody>
</table>

#### Ulcerative colitis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of ulcerative colitis (UC)?</td>
<td>Chronic inflammatory disease of the colon that always has rectal involvement</td>
</tr>
</tbody>
</table>
### What is the pathophysiology of UC?
Mucosa/submucosa inflammation with sparing of the serosa with continuous involvement unlike Crohn’s disease

### What is the epidemiology of the UC?
Greater incidence in whites between the age of 16–40, more likely to affect Jews and a positive family history in up to 20%

### What are some clinical features of the following degrees of UC:

<table>
<thead>
<tr>
<th>Degree</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild disease</td>
<td>No systemic symptoms; Less than 4 bowel movements per day; Few extraintestinal symptoms</td>
</tr>
<tr>
<td>Severe disease</td>
<td>Systemic response (F/C, weight loss, etc.); Greater than 4 bowel movements per day; Extraintestinal symptoms</td>
</tr>
</tbody>
</table>

### What is the diagnostic test of choice for UC?
Colonoscopy

### What are some complications of UC?
Toxic megacolon (more common in UC); Perforation; Obstruction; Perianal abscess and fistulas; Colon carcinoma; Hemorrhage

### What are some key points in the management of mild/moderate attack of UC?
Sulfasalazine—mainstay therapy; Mesalamine/olsalazine—second line; Corticosteroid—supplement; Avoid antidiarrheal agents; Azathioprine/cyclosporine—if steroids fail

### What are some key points in the management of severe UC?
IV fluids and NG tube; Broad-spectrum Abx; Monitor for hemorrhage/toxic megacolon; Surgical consult

### Pseudomembranous enterocolitis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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</thead>
<tbody>
<tr>
<td>What is the definition of pseudomembranous enterocolitis?</td>
<td>Inflammatory bowel disease characterized by yellow exudative pseudomembranous plaque over necrotic colon</td>
</tr>
<tr>
<td>What is the pathogenic species responsible for pseudomembranous enterocolitis?</td>
<td><em>Clostridium difficile</em></td>
</tr>
<tr>
<td>What antibiotics are commonly associated with the proliferation of <em>C. difficile</em>?</td>
<td>Clindamycin; Ampicillin; Cephalosporins</td>
</tr>
</tbody>
</table>
What is the pathophysiology of *C. difficile* associated pseudomembranous enterocolitis in relation to Abx use? Abx use alters normal gut flora and allows *C. difficile* to propagate

What are some common clinical features of pseudomembranous enterocolitis? Profuse watery diarrhea with crampy abdominal pain, stool may have blood, and fever

What is the general time frame for the development of pseudomembranous enterocolitis after Abx use? Generally 7–10 days after Abx use, but can occur weeks after discontinuation

What is the diagnostic study of choice? *C. difficile* toxin in stool

What are some key points in the management of pseudomembranous enterocolitis? IV fluids and electrolyte balance; Discontinue the offending agent; Oral metronidazole is first-line; Oral vancomycin if metronidazole does not work

What role do antidiarrheal drugs play in management? None—they can worsen symptoms and increase likelihood of toxic megacolon

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**MESENTERIC ISCHEMIA**

What is the pathophysiology of mesenteric ischemia? Mesenteric arteries that do not deliver enough blood to the small or large intestine, typically due to sudden occlusion or decreased cardiac output (CO)

What are some important things to know about mesenteric ischemia? Commonly affects elderly with CVS disease; Mortality rate 50% once infarction occurs

What are some key points in the following causes of mesenteric ischemia:

- **Nonocclusive**
  - Typically due to reduction in CO (i.e., CHF); Account for up 25% of all cases; Commonly affects critically sick/elderly; Presentation is more subtle and insidious

- **Acute occlusion**
  - Typically due to embolization (i.e., afib); Accounts for the majority of cases; Common in severe atherosclerotic patients; Presentation is acute, sudden, and dramatic
Venous thrombosis

Typically due to hypercoagulable state; Often have history of deep vein thrombosis/Pulmonary embolism (DVT/PE)

What are some causes for the following causes of mesenteric ischemia:

Nonocclusive

Hypotension (i.e., sepsis); CHF; Hypovolemia

Acute occlusion

Recent MI; Atherosclerotic heart disease; Dysrhythmias (esp. afib)

Venous thrombosis

History of DVT/PE; Hypercoagulable

What are some clinical features of mesenteric ischemia?

Vague abdominal pain that is out of proportion early in the course, sudden severe pain if cause is acute, guaiac positive stool, N/V, and peritoneal signs late in the course if infarction occurs

What are some commonly used diagnostic tests?

CBC—often elevated white count; Arterial blood gas (ABG)—metabolic acidosis is common; Plain films—often normal; CT/US—not the first line choice; Lactate level

What is the diagnostic test of choice in suspected mesenteric ischemia?

Angiography

What are the general key points in the management of mesenteric ischemia?

IV fluids and NG tube for decompression; Broad-spectrum Abx; Look for underlying cause and correct them; Use of papaverine for diagnostic study

What are the indications for surgical intervention in mesenteric ischemia?

Necrotic bowel requiring resection; Revascularization; Evidence of perforation (peritoneal signs)

DIVERTICULAR DISEASE

What is the definition of a diverticula?

Sac-like herniations of the mucosa in the colon typically due to an increase in intra-luminal pressure often from lack of fiber
What are some important things to know about diverticular disease?

Direct correlation with incidence and age; High in patients who consume low fiber; Common cause of painless lower GI bleed

What are the two main complications of diverticular disease?

1. Diverticulosis
2. Diverticulitis

What are some clinical features of diverticulosis?

Hallmark is self-limiting painless rectal bleeding that is typically bright red or maroon, although a small percentage have massive lower GI bleed

What are some key points in the management of diverticulosis?

Ensure that there is no massive GI bleeding; Bleeding is typically self-limited; Diagnosis requires colonoscopy; Increase in fiber may reduce future attacks; Avoidance of seeds—not really proven

What is the definition of diverticulitis?

Microperforation of diverticula that result in an inflammatory response that is typically walled off by pericolic fat

What are some clinical features of diverticulitis?

LLQ pain present for a few days is the hallmark, N/V, diarrhea, and changes in urinary symptoms

What are some complications of diverticulitis?

Abscess formation; Fistula; Obstruction; Perforation

How commonly do patients with diverticulitis present with RLQ pain?

Less than 5%—more common in Asians

What other differential should be considered in those who present with RLQ pain?

Appendicitis

What are some commonly used diagnostic tests in diverticulitis?

CBC—leukocytosis; Ab plain film—examine for complications; CT—test of choice to evaluate extent of disease

What studies are contraindicated during an acute attack of diverticulitis?

Colonoscopy and contrast studies

What are some key points in the management of diverticulitis?

IV fluids and NPO; NG tube in suspected obstruction; Broad-spectrum Abx; Surgical consult if suspected complications
HERNIA

What is the definition of a hernia? It is the protrusion of any body part out of its natural cavity primarily due to inherent weaknesses (congenital) or acquired (surgery).

Define the following possibilities for a hernia:

- **Reducible**: The contents can be returned to its cavity.
- **Irreducible/Incarcerated**: Unable to reduce—no vascular compromise.
- **Strangulated**: Vascular compromise of herniation.

What are some important points for the following types of hernia:

- **Femoral hernia**: Protrudes below the inguinal ring; More common in females; High frequency of incarceration.
- **Direct inguinal hernia**: Directly via the floor of Hesselbach’s triangle; Incidence increases with age; Rarely incarcerates.
- **Indirect inguinal hernia**: Protrudes via the internal inguinal ring; More common in men; More common in younger population; High frequency of incarceration.
- **Umbilical hernia**: Represents a congenital defect in newborns; Most will close by 3 years of age; Rarely incarcerates.

What are some clinical features of a hernia? Palpable bulge that often can be detected on exam and can be sore when pressed, but rarely painful unless incarcerated.

What are some key points in the management of a hernia? If hernia is recent, can try to reduce; If suspected necrosis, do not reduce; Incarcerated = surgery consult; Strangulation = surgery and Abx; Reducible = consider elective surgery.
## Hemorrhoids

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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</thead>
<tbody>
<tr>
<td>What is the definition of a hemorrhoid?</td>
<td>Dilated internal or external hemorrhoidal venous plexus</td>
</tr>
<tr>
<td>What are some risk factors for the development of hemorrhoids?</td>
<td>Straining; Increase in portal pressure (i.e., cirrhosis); Constipation; Low fiber diet; Pregnancy</td>
</tr>
<tr>
<td>What are some important points for the following types of hemorrhoids:</td>
<td></td>
</tr>
<tr>
<td>Internal hemorrhoids</td>
<td>Originate above the dentate line; Relatively insensitive area—no/little pain; Rarely palpable—painless bleeding common; Visualized at 2, 5, and 9 o’clock positions</td>
</tr>
<tr>
<td>External hemorrhoids</td>
<td>Originate below the dentate line; In well-innervated area, often painful; Usually can be visualized</td>
</tr>
<tr>
<td>What are some common clinical features for the following types of hemorrhoids:</td>
<td></td>
</tr>
<tr>
<td>Internal hemorrhoids</td>
<td>Painless bright red blood per rectum, most common cause of lower GI bleed in younger population</td>
</tr>
<tr>
<td>External hemorrhoids</td>
<td>Tender palpable mass often due to thrombosis</td>
</tr>
<tr>
<td>What are some key points in the management in the following types of hemorrhoids:</td>
<td></td>
</tr>
<tr>
<td>Internal hemorrhoids</td>
<td>Often resolves on its own; Increase dietary fiber and fluids; Stool softeners, bulk laxatives, and sitz bath; Refractory bleeding = IV fluid/ packing/surgery</td>
</tr>
<tr>
<td>External hemorrhoids</td>
<td>Analgesics/sitz bath/stool softeners; Acute thrombosis: excision of clots</td>
</tr>
<tr>
<td>What are some indications for surgical intervention?</td>
<td>Refractory bleeding or pain; Incarceration/strangulation</td>
</tr>
</tbody>
</table>
## Anorectal Abscess

**What is the definition of an anorectal abscess?**
Abscess that typically develops in one of the potential spaces near the rectum/anus most often due to obstruction of glands at the base of the anal crypts.

**Name some potential spaces where an anorectal abscess can occur.**
Perianal, intersphincteric, and ischiorectal

**What are some other causes of an anorectal abscess?**
Inflammatory bowel disease; Radiation injury; Cancer; Trauma; TB

**What is a common complication of an anorectal abscess?**
Fistula formation

**What are some common clinical features of an anorectal abscess?**
Dull aching pain that is worse with bowel movements and relieved after, sometimes palpable mass on exam, fever, and obvious discomfort whenever patient sits

**What are some key points in the management of an anorectal abscess?**
Simple perianal abscess = ED drainage; Most require surgical intervention; Most individuals do not require Abx

**What are the indications for the use of Abx?**
DM, immunocompromised patient, and valvular heart disease

## Anal Fissure

**What is the definition of an anal fissure?**
Linear tears of the epithelium within the anal canal, typically due to recurrent diarrhea or passage of large hard stools

**What are some important points about an anal fissure?**
Majority are located in the posterior midline; Number 1 cause of painful rectal bleeding; IBD and TB are other causes

**What are some clinical features of an anal fissure?**
Severe pain with defecation, often with a history of constipation, and linear tear of the posterior midline on exam. Pain is so intense, patients often try to avoid defecation

**What are some key points in the management of an anal fissure?**
Symptomatic relief to allow healing; Analgesic topical, sitz bath, and dietary fiber; Refractory cases often require excision
What is the recurrence rate, even with treatment?  Up to 50%

### Fistula-In-Ano

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of a fistula-in-ano?</td>
<td>Abnormal communication between anus and the skin</td>
</tr>
<tr>
<td>What is fistula-in-ano often caused by?</td>
<td>Commonly from ischiorectal or perianal abscess</td>
</tr>
<tr>
<td>What conditions are fistula-in-ano associated with?</td>
<td>TB; Cancer; IBD</td>
</tr>
<tr>
<td>What are some of the clinical features of fistula-in-ano?</td>
<td>Persistent blood-stained purulent discharge or an abscess if it becomes blocked</td>
</tr>
<tr>
<td>What is the primary treatment for fistula-in-ano?</td>
<td>Surgical incision</td>
</tr>
</tbody>
</table>

### Anal Foreign Bodies

<table>
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<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>How do anal foreign bodies usually occur?</td>
<td>Placement of object into anus; Transit from GI foreign body</td>
</tr>
<tr>
<td>What important distinction must be made in regard to location of the foreign object?</td>
<td>Whether the object is below or above the rectosigmoid junction (difficult to visualize and remove if above)</td>
</tr>
<tr>
<td>What age group commonly present with anal foreign bodies?</td>
<td>20–30 years of age (anal eroticism)</td>
</tr>
<tr>
<td>What are some important points to know about anal foreign bodies?</td>
<td>Often present late due to embarrassment; Suspected in psychiatric patients with anal symptom; Attempted self-extraction = risk of perforation</td>
</tr>
<tr>
<td>What are some clinical features of anal foreign bodies?</td>
<td>Anal pain, bleeding, pruritus, and F/C with rigid abdomen in perforation</td>
</tr>
<tr>
<td>What are some commonly used diagnostic tests for anal foreign bodies?</td>
<td>Abdomen x-rays; Upright CXR if perforation suspected; Rigid proctosigmoidoscope</td>
</tr>
<tr>
<td>What are some key points in the management of anal foreign bodies?</td>
<td>ER removal if the object if low riding; Retractors, snares, forceps may be used; Serial observation after removal; Surgical consult if evidence of perforation</td>
</tr>
</tbody>
</table>
### Proctitis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of proctitis?</td>
<td>It is inflammation of the rectal mucosa within 15 cm of the dentate line that typically affect adult males</td>
</tr>
<tr>
<td>What are some clinical features of proctitis?</td>
<td>Passage of blood and mucus, tenesmus, and abdominal cramping</td>
</tr>
<tr>
<td>What are some common causes of proctitis?</td>
<td>Idiopathic; Infectious (HSV-1 and-2); Radiation; Ischemia</td>
</tr>
<tr>
<td>What is the diagnostic study of choice to evaluate proctitis?</td>
<td>Proctosigmoidoscopy</td>
</tr>
<tr>
<td>What are some complications of proctitis?</td>
<td>Fistula; Fissures; Strictures</td>
</tr>
<tr>
<td>What are some key points in the management of proctitis?</td>
<td>Analgesic relief; Abx if cause is infectious (i.e., HSV-1); Sitz bath for relief</td>
</tr>
</tbody>
</table>

### Rectal Prolapse

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>What is the definition of rectal prolapse?</td>
<td>It is when rectal mucosa or full-thickness rectal tissue slides outside the anal orifice</td>
</tr>
<tr>
<td>What is the pathophysiology of rectal prolapse?</td>
<td>Initially begins as an internal prolapse that progresses to an external prolapse outside the anal orifice</td>
</tr>
<tr>
<td>What are some common causes of rectal prolapse?</td>
<td>Straining (i.e., constipation); Weakness of the pelvic floor; Neurologic disorder</td>
</tr>
<tr>
<td>What two age groups are commonly affected with rectal prolapse?</td>
<td>1. Pediatric (up to 90% resolve on their own)</td>
</tr>
<tr>
<td></td>
<td>2. Elderly (most require corrective surgery)</td>
</tr>
<tr>
<td>What are some clinical features of rectal prolapse?</td>
<td>Fecal incontinence, painless mass on exam, and rectal bleeding</td>
</tr>
<tr>
<td>What are some complications of rectal prolapse?</td>
<td>Ulceration; Bleeding; Necrosis</td>
</tr>
<tr>
<td>What are some key points in the management of rectal prolapse?</td>
<td>Most rectal prolapses can be reduced; Emergent surgery if evidence of ischemia; Stool softeners if reduction is successful}</td>
</tr>
</tbody>
</table>
Pilonidal Sinus

What is the definition of a pilonidal sinus? Abscess that forms at the superior edge of the buttock in midline

What is pathophysiology for the development of a pilonidal sinus? Ingrowing hair that penetrates the skin and induces a foreign body reaction

What are some clinical features of a pilonidal sinus? Recurrent pain and purulent discharge

What are some key points in the management of pilonidal sinus? Incision and drainage of abscess; Surgical intervention for excision; Consider Abx if immunocompromised

DIARRHEA

What is the definition of diarrhea? Loose watery stools that occur more than three times per day that typically is self-limited, but can lead to dehydration and electrolyte imbalance

What are some important causes of diarrhea? Infection (bacterial/viral/parasitic); Food intolerance; Medication reaction; Intestinal disease (i.e., celiac disease); Functional bowel disorder (i.e., IBS)

List common parasite-induced diarrhea: Giardia lamblia; Entamoeba histolytica; Cryptosporidium; Necator americanus

What are some important things to know about viral-induced diarrhea? Causes the majority of all acute episodes; Norwalk and rotavirus most common; Peak during winter months; Adenovirus also common

What are some clinical features of viral-induced diarrhea? Low-grade fever, vomiting, diarrhea, mild abdominal cramping, and sometimes an upper respiratory infection (URI) prodrome beforehand

What are some common modes of transmission? Sick contact; Contaminated food

What are some key points in the management of viral-induced diarrhea? Treatment is supportive; Ensure adequate hydration; Typically self-limited
What are some important points to know about bacteria-induced diarrhea?

Accounts for about 25% of acute diarrhea; Classified as invasive or toxin producing

What are some examples of invasive bacteria?

Salmonella; Shigella; Vibrio; Campylobacter

What are some examples of toxin-producing bacteria?

Bacillus cereus; Staphylococcus aureus; Clostridium difficile

What does a wet mount of stool typically show?

Fecal leukocytes (typically + with bacteria); WBCs (use of methylene blue)

What are some important points and treatment for the following bacterial-induced diarrhea:

Vibrio cholera

Typically from contaminated water/seafood; Incubation about 5 days; Profuse watery diarrhea is the hallmark; Tx: IV hydration and Abx-fluoroquinolone

Vibrio parahemolyticus

Invasive bacteria—typically from bad seafood; Range from mild to explosive diarrhea; Tx: Supportive care; usually self-limiting

Staphylococcus aureus

Number 1 common cause of food-related diarrhea; Presentation from preformed toxins; Often in protein-rich food such as meat; Incubation in hours; Tx: Supportive; usually self-limiting

Escherichia coli serotype O157:H7

Common cause of hemorrhagic colitis; Often from contaminated beef and milk; Incubation in about a week; Diarrhea, vomiting, and severe abdominal pain; Associated with HUS; Tx: Supportive—typically a week to resolve

E. coli (enterotoxigenic)

Very common cause of traveler’s diarrhea; Often in contaminated food and water; Presents like V. cholera; Tx: Supportive; Abx can shorten course
**Shigella**

Includes *S. flexneri* and *S. dysenteriae*; Highly infectious and usually from fecal-oral; High-grade fever, bloody mucoid stool, and abdominal pain is common; Tx: Typically resolve in a week, highly infectious, and supportive care

**Salmonella**

Includes *S. typhi* and *S. typhimurium*; Often from contaminated food or pets; Immunocompromised patients most at risk; Variable presentation (i.e., typhoid fever); Tx: Mild cases supportive care; more severe cases may require Abx

**Campylobacter**

Very common cause of bacterial diarrhea; Often in food (poultry) and water; More common in the pediatric population; Incubation is about 4 days; Fever, HA, abdominal pain, and watery bloody stool; Tx: Abx in severe cases; Associated with HUS and Guillain-Barre syndrome

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### CLINICAL VIGNETTES

<table>
<thead>
<tr>
<th>Vignette</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>31-year-old male with long history of alcohol abuse presents with progressive difficulty in swallowing which was initially to foods only, but now to liquids; PE: unremarkable exam</td>
<td>Esophageal carcinoma</td>
</tr>
<tr>
<td>29-year-old female who recently finished her course of tetracycline presents with odynophagia, but is otherwise healthy; PE: unremarkable exam</td>
<td>Inflammatory esophagitis</td>
</tr>
<tr>
<td>4-year-old child is brought in by her mother due to recent onset of dysphagia and gagging, otherwise the child is healthy with no other complaints; PE: unremarkable exam, clear oropharynx</td>
<td>Swallowed foreign body</td>
</tr>
<tr>
<td>21-year-old male with no PMH presents with abdominal pain that was initially around the umbilicus, but now has progressed to the RLQ associated with nausea and vomiting after the onset of pain; PE: RLQ tenderness and (+) Rosving’s sign</td>
<td>Appendicitis</td>
</tr>
</tbody>
</table>
61-year-old elderly male with arthritis presents with epigastric pain that is often relieved by intake of food, but is otherwise healthy; PE: epigastric tenderness, but no rebound

45-year-old female with an Hx of recent ERCP presents with epigastric pain radiating to her back associated with nausea and emesis; PE: epigastric tenderness; Labs: elevated lipase

18-year-old female with a history of bulimia presents with chest pain with dysphagia that occurred soon after her bout of emesis; PE: unremarkable exam

34-year-old obese female presents with RUQ pain along with fever and nausea, patient has a history of gallstones; PE: fever, tachycardia, RUQ tenderness, and yellowish sclera on examination of eye; Labs: elevated alkaline phosphate and LFTs

81-year-old female with an Hx of HTN, afib, and DM presents with a sudden onset of diffuse abdominal pain along with nausea and vomiting; PE: pain out of proportion on exam, guaiac positive stool, and rebound; Labs: elevated lactate

24-year-old female presents with 2 days of lower GI bleeding and describes the toilet bowl as being bright red after each bowel movement, other then a history of constipation, patient is otherwise healthy; PE: unremarkable exam and guaiac positive stool

56-year-old male presents with LLQ pain with nausea, vomiting, and urinary changes for 2 days; PE: LLQ tenderness and no rebound on exam; Labs: elevated white count

31-year-old male with recent discharge from hospital now presents with diffuse watery diarrhea and crampy abdominal exam: PE: low-grade fever and mild tenderness of abdomen

31-year-old male with history of HIV presents with tenesmus, abdominal cramping, and passage of blood and mucus for 3 days; PE: tenderness on rectal exam

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# Acute Renal Failure

**What is the definition of acute renal failure (ARF)?**  
It is deterioration of renal function that results in accumulation of waste and loss of internal homeostasis.

**What are some key physiology points about the kidney?**  
Kidney receives 25% of the cardiac output; Outer medulla is susceptible to hypoxia; With decreased renal blood flow (RBF), increased susceptibility to toxins.

**What is the primary way to assess renal function?**  
Glomerular filtration rate (GFR) (via creatinine clearance).

**What are some important things to know about each in the following setting:**

- **Community-acquired ARF**  
The majority of cases are reversible; Mortality rate is less than 10%; The most common cause is hypovolemia (pre-renal).

- **Hospital-acquired ARF**  
Mortality rate can be higher than 50%; Many typically have other comorbidities; Most common cause is iatrogenic (intrinsic).

**What are three types of acute-renal failure?**  
1. Prerenal  
2. Renal  
3. Postrenal

**What are some important causes of prerenal azotemia?**  
Hypovolemia (i.e., diuretics/dehydration); Third space sequestration (i.e., pancreatitis); Sepsis; Decreased cardiac output.
What is the typical urine status in patients with prerenal azotemia? Oliguric; Avid reabsorption of sodium and water; BUN/creatinine (BUN/Crea) ratio of 20:1; U/A typically shows no evidence of damage; Fractional excretion of sodium <1%

What are some important causes of renal azotemia? Acute tubular necrosis; Thrombosis; Glomerular disease; Vascular disease; Acute interstitial nephritis

What are common causes of acute tubular necrosis (ATN)? Ischemia—most common; Pigments (i.e., myoglobin); Nephrototoxic agents

What are some common nephrotoxic agents? Contrast dye; Nonsteroidal anti-inflammatory drugs (NSAIDs); Angiotensin-converting enzyme inhibitors; Antibiotics (i.e., penicillin)

What is the typical urine status in patients with renal azotemia? Inability to concentrate urine (dilute); Have evidence of damage (i.e., casts); High urine sodium (>40 mEq/L)

What are some important causes of postrenal azotemia? Ureteral obstruction (i.e., stones); Bladder obstruction; Urethral obstruction (i.e., strictures)

What are some important tests to consider to differentiate the type of ARF? Urinalysis; Ultrasound; Postvoid residual urine; Urine and serum Na and creatinine; Urine osmolality; Urine eosinophil

What are some key points in treatment for each of the following causes of ARF:

**Prerenal**
- Rapid volume replacement; Find the cause of hypoperfusion and correct it;
- Initial fluid administration of isotonic saline is appropriate in most cases

**Renal**
- Increase the urine flow; If cause is a nephrotoxic agent, remove it;
- Maintaining balance of fluid / electrolytes; Dialysis if indicated

**Postrenal**
- Relieve obstruction; Catheter until obstruction is relieved

What are some important points for each of the following complications of ARF:

**Hypocalcemia**
- It is common in setting of ARF;
- Typically asymptomatic; Intravenous (IV) calcium chloride if symptomatic
| Hypermagnesium | It is common in setting of ARF; Typically asymptomatic |
| Hyperkalemia | Potentially the most life-threatening; Death due to cardiac dysrhythmias; Important to obtain serum K and ECG; Treat (IV glucose/insulin, bicarb, etc.) |
| Metabolic acidosis | It is also common in the setting of ARF; Typically asymptomatic |

**What are some indications of dialysis in the setting of ARF?**

Hyperkalemia; Uremia (i.e., encephalopathic); Creatinine >10 mg/dL or BUN >100 mg/dL; Clinically significant fluid overload/acidosis; Particular nephrotoxins (i.e., ethylene glycol)

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**CHRONIC RENAL FAILURE**

| What is the definition of chronic renal failure (CRF)? | The irreversible and gradual loss of renal function that results in inability to regulate homeostasis and concentrate urine |
| What are the two most common causes of CRF? | 1. Diabetes  
2. Hypertension |
| What are some other causes of CRF? | Glomerulonephritis; Polycystic kidney disease; Alport syndrome |
| What are some important things to know for each of the following stages of CRF: | |
| **Stage I** | Decreased renal function <50% GFR; At least 1/2 of renal function is gone; Homeostasis and excretion intact |
| **Stage II** | Renal insufficiency with 20–50% GFR; Mild anemia due to decreased erythropoietin (EPO); Mild azotemia |
| **Stage III** | Renal failure with 5–20% GFR; Severe anemia; Azotemia; Electrolyte imbalance (i.e., hyperkalemia) |
| **Stage IV** | Renal failure <5% GFR; Multiple organ system effects |
| What is the treatment for CFR? | Kidney transplant; Peritoneal dialysis; Hemodialysis (also for ARF) |
What are some complications associated with hemodialysis?

- Infection of vascular access;
- Thrombosis of the vascular access;
- Hemorrhage

### Nephrolithiasis

**What is the definition of nephrolithiasis?**

Supersaturation of a mineral within the ureters that result in urinary changes and ureter spasms

**What are some important things to know about nephrolithiasis?**

- More common in males between 20–45;
- There is a hereditary predisposition;
- Over 90% of stones <5 mm will pass;
- Recurrence can be as high as 50%

**What is the most common type of kidney stone?**

Calcium oxalate (about 75% of all stones)

**What are some possible causes of calcium stone?**

- Hyperparathyroidism;
- Sarcoidosis;
- Neoplasm

**What are some important things to note for each of the following stone types:**

<table>
<thead>
<tr>
<th>Stone Type</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Struvite stone</strong></td>
<td>After calcium stones, the next most common; Radiopaque; Associated with urea-splitting Proteus</td>
</tr>
<tr>
<td><strong>Uric acid stones</strong></td>
<td>Next common after calcium and struvite; Radiolucent; Common in patients with gout and leukemia</td>
</tr>
<tr>
<td><strong>Xanthine stones</strong></td>
<td>Rare; Radiopaque; Associated with methylxanthine/theophylline</td>
</tr>
<tr>
<td><strong>Cystine stones</strong></td>
<td>Radiopaque; Familial associated</td>
</tr>
</tbody>
</table>

**What are some clinical features of nephrolithiasis?**

- Unilateral flank pain that is often colicky, can also have pain in the back with radiation to the groin (labia/testicles), urinary symptoms (hematuria, dysuria, etc.), and nausea/vomiting (N/V)

**What is another important diagnosis to consider in a person who presents for the first time with flank pain and is elderly with history of uncontrolled HTN?**

- Abdominal aortic aneurysm

**What are some important laboratory tests to consider and common findings:**

<table>
<thead>
<tr>
<th>Test</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete blood count (CBC)</td>
<td>Usually normal</td>
</tr>
</tbody>
</table>
U/A

Hematuria (can be absent in up to 25%); Urinary pH > 7.6 (suspect *Proteus*).

Urine culture

Positive if infection is present.

BUN/Crea

To assess renal function.

**What are some important diagnostic tests to consider?**

CT: diagnostic study of choice; Intravenous pyelogram (IVP): for anatomical/functional assessment; Ultrasound (U/S): for pregnant women and children.

**What are some key points in the management of nephrolithiasis?**

Proper fluid hydration; Narcotic with ketorolac (optimal pain control); Antiemetic for sustained emesis.

**What are some common indications for admission of a patient with nephrolithiasis?**

Evidence of active infection (fever/pyuria); Inability to tolerate oral intake; Stone > 5 mm (unlikely to pass on its own); Renal insufficiency.

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**URINARY TRACT INFECTIONS**

**What is the definition of a urinary tract infection (UTI)?**

Presence of bacteria in the urinary system.

**What are some important things to know about UTI?**

One of the most common bacterial infections; 50% of women will have at least one UTI; Sexual activity increases risk of UTI.

**What are the three most common organisms associated with UTI?**

1. *Escherichia coli* (up to 80% of all UTIs)
2. *Chlamydia*
3. *Staphylococcus saprophyticus*

**What are some clinical features of UTI?**

Dysuria, urge to urinate, increased urination frequency, nocturia, and suprapubic heaviness (should not have systemic effects such as fever).

**What are some other differentials to consider in a woman who presents with UTI?**

Pelvic inflammatory disease (PID); Vulvovaginitis.

**What are some features of a complicated UTI?**

Resistant species; Male; Children or elderly; Pregnant female; Associated condition such as pyelonephritis; Underlying anatomical abnormality of the GU system.
List some methods used to collect a proper urine sample.

What are some common microscopic findings in a U/A of a patient with UTI?

When is it appropriate to obtain a urine culture?

What are some key points in the management of a UTI?

What is another important consideration in a patient who presents with a UTI?

What is the definition of pyelonephritis?

What are some risk factors for the development of pyelonephritis?

List the classification of pyelonephritis.

What are some clinical features of pyelonephritis?

What are some complications of pyelonephritis?

What are some key points in the management of pyelonephritis?

What are some indications for admissions in patients with pyelonephritis?

**MALE GENITAL PROBLEMS**

What are the three cylindrical bodies of the penis?

What is the primary blood supply of the penis?

What is the average size of the testis?
What are the two investing layers of the testis?
1. Tunica albuginea
2. Tunica vaginalis

What are some important components of the physical exam?
Visual inspection; Palpation of the scrotum for fluid; Milk the penis for discharge; Rectal exam (check prostate); Check for inguinal hernias

Common Genital Infections

What are some common organisms responsible for urethritis?
Gonorrhea and chlamydia (most common); Trichomonas vaginalis; Ureaplasma urealyticum

What are some clinical features of urethritis?
Discharge and dysuria, but can be asymptomatic

How is the diagnosis of urethritis usually made?
Gram stain

What is the treatment of choice?
Directed against gonorrhea (i.e., ceftriaxone); Directed against chlamydia (i.e., doxycycline); Metronidazole if suspected trich infection

What is orchitis?
Inflammation of the testis

What are some common causes of orchitis?
Systemic infections (i.e., mumps); Direct extension such as epididymitis

What are some clinical features of orchitis?
Testicular swelling and pain that typically does not include urinary symptoms

Does mumps-induced orchitis require treatment?
No—typically resolves

What is the key point in the management of orchitis?
Urology consultation; It is rare when compared to torsion/cancer

What are some common etiologic causes of acute bacterial prostatitis?
Usually gram (–) bacteria such as E. coli, Proteus, and Pseudomonas

What are some common clinical features of acute bacterial prostatitis?
Urinary symptoms (i.e., dysuria), pelvic/back pain, systemic signs of infection such as fever/chills; PE: swollen/tender prostate

What should one be careful not to do during a rectal exam?
Massaging the prostate

What will urinanalysis commonly show?
Evidence of cystitis
What are some key points in the management of acute bacterial prostatitis?

Antibiotic therapy; Analgesics, stool softeners, and hydration; Urology consultation if evidence of urinary retention

What are some common causes of penile ulcers?

Herpes simplex, chancroid, syphilis, and granuloma inguinale

How is the diagnosis of syphilis commonly made?

Positive VDRL or RPR confirmed by Treponema-specific tests

What is the antibiotic of choice for syphilis?

Penicillin, doxycycline, and tetracycline

What is the cause of a chancroid?

Haemophilus ducreyi

What is the antibiotic treatment of choice for a chancroid?

A macrolide (i.e., azithromycin)

What is the cause of granuloma inguinale?

C. granulomatus

What are some antibiotics commonly used to treat granuloma inguinale?

Doxycycline or trimethoprim-sulfamethoxazole (TMP-SMX)

What is Fournier’s gangrene?

Known as idiopathic scrotal gangrene

What are some common clinical features of Fournier’s gangrene?

Often febrile and toxic with a painful erythematous penis/scrotum

What groups are more commonly affected with Fournier’s gangrene?

Elderly; Diabetics; Immunocompromised people

What are some common etiologic causes of Fournier’s gangrene?

Typically mixed: E. coli, Streptococcus, Bacteroides fragilis, etc.

What are some key points in the management of Fournier’s gangrene?

Broad-spectrum antibiotics; Urologic consult for debridement; Supportive management

What is phimosis?

Inability to retract foreskin behind the glans

What is the most common cause of phimosis?

Chronic infection of the foreskin that results in scarring

What can be done if phimosis appears to be causing vascular compromise?

Dorsal slit of the foreskin and circumcision for definitive treatment

What is paraphimosis?

Inability to pull the foreskin over the glans

What is the primary concern of paraphimosis?

Vascular compromise

What can be done in an emergent situation if vascular compromise is evident?

Dorsal slit and circumcision for definitive treatment

What age group is epididymitis more common in?

Young adults
What are some clinical features of epididymitis? Gradual onset of unilateral testicular pain, dysuria, fever, and tenderness of epididymis on exam

What is Prehn’s sign? Relief of testicular pain by elevating it

What are some common etiologic causes of epididymitis? E. coli, Pseudomonas, and Chlamydia

What are some common diagnostic studies to consider in epididymitis? CBC; Urethral culture and gram stain; Urinanalysis

What are some key points in the management of epididymitis? Antibiotic coverage (i.e., Ciprofloxacin); Stool softeners; Analgesics with ice

What age groups are commonly affected with testicular torsion? Bimodal: neonates and 12–18 years of age

What are some important elements in the history of a patient who presents with testicular torsion? Recent physical exertion (i.e., sports/sex); History of testicular pain with relief after; History of cryptorchidism

What are some clinical features of testicular torsion? Acute onset of unilateral testicular pain often with nausea/vomiting; PE: affected testicles are high riding with loss of cremasteric reflex

What diagnosis can testicular torsion be confused with? Epididymitis

What is the diagnostic test of choice for testicular torsion? Color Doppler ultrasound

What are some key points in the management of testicular torsion? Urgent urology consult for surgery; Surgery within 6 hours: 80–100% salvage; Analgesics prior to surgery; Salvage rate is 20% after 10 hours and 0% after 24 hours

CLINICAL VIGNETTES

43-year-old female with PMH of afib Renal infarct presents with a sudden onset of left flank pain and hematuria Abdominal CT: wedge-shaped lesion of the left kidney

24-year-old female presents with dysuria and increased frequency of urination, patient is sexually active; PE: suprapubic tenderness; U/A: (+) nitrate and leukocyte esterase Urinary tract infection
31-year-old male presents with sudden onset of right flank pain along with nausea, vomiting, and hematuria; PE: right CVA tenderness and in severe pain; U/A: (+) blood; U/S: shows right hydronephrosis  
Nephrolithiasis

26-year-old male presents with hemoptysis, dark urine, and general fatigue for 3 days; PE: unremarkable exam; Labs: anti-GBM antibodies and urine that shows blood  
Goodpasture’s syndrome

81-year-old male with DM presents to the ER via EMS with fever and appears sick looking; PE: unremarkable except an erythematous penis that is very tender to the touch with evidence of a prior wound in the scrotum  
Fournier’s gangrene

14-year-old male with a recent history of sore throat presents with low-urine output and swelling of lower legs; PE: periorbital edema; Labs: elevated BUN/Crea and urine that shows blood  
Poststreptococcal glomerulonephritis

25-year-old female presents fever, chills, and left flank pain for about 2 days; PE: left CVA tenderness; U/A: (+) nitrate and leukocyte esterase  
Pyelonephritis

21-year-old male with no PMH presents with fever, dysuria, and pelvic/back pain; PE: remarkable for a tender and swollen prostate  
Bacterial prostatitis

82-year-old male with a long history of smoking presents with frank blood on urination, also with recent weight loss; PE: unremarkable exam; U/A: gross blood  
Bladder cancer

64-year-old male presents with a 2-week history of nocturia, urinary hesitance, and weak stream during urination, otherwise healthy; PE: rectal exam showed diffusely enlarged prostate; Labs: normal prostate-specific antigen (PSA)  
Benign prostatic hyperplasia (BPH)

18-year-old male with PMH of undescended testis presents with sudden onset of right testicular pain associated with nausea and vomiting; PE: tender/swollen right testicle with (–) cremasteric reflex  
Testicular torsion
61-year-old female with a history of long-standing hypertension and DM presents with altered mental status; Labs: significant for a potassium of 6, BUN of 99, creatinine of 7 with a GFR <5%

21-year-old male presents with a gradual onset of unilateral testicular pain, fever, and dysuria for about 4 days, patient does admit to having unprotected sex; PE: tenderness of the penis on exam that is relieved when raised

Chronic renal failure

Epididymitis
HYPOGLYCEMIA

What is the glucose blood level where manifestations of hypoglycemia typically occur?  
<50 mg/dL

What are some common clinical features of hypoglycemia?  
Tachycardia, tremulousness, diaphoresis, mental status change, seizures, focal neurologic deficits that can mimic a stroke

What are some elements of the history to obtain in a patient who is hypoglycemic?  
Medications; Medical problems; Fasting/fed state preceding

List some hormones released during hypoglycemia.  
Glucagon; Epinephrine; Growth hormone; Cortisol

List common causes of fasting hypoglycemia.  
Islet cell tumor; Myxedema; Adrenocortical insufficiency; Extrapancreatic tumor

List common causes of post-prandial hypoglycemia.  
Hyperinsulinism; Fructose intolerance; Galactemia

What is the most common cause of hypoglycemia in diabetics?  
Insulin and sulfonylureas

List other medications that commonly cause hypoglycemia.  
Beta-blockers; Ethanol; Salicylate; Cimetidine

What is Whipple’s triad?  
Diagnostic features of insulinoma of the pancreas:  
1. Symptoms and signs of hypoglycemia  
2. Blood sugar levels below 50 mg/dL  
3. Recovery from an attack following the administration of glucose
What are some key points in the management of hypoglycemia? Monitor glucose (glucometer often not reliable below 50 mg/dL); Intravenous (IV) D5W continuous drip; Intramuscular (IM) glucagon and IV dextrose if patient is unresponsive; If patient is awake, oral feeding is preferred

What are some indications to admit a patient who is hypoglycemic? Admit if overdose on insulin or oral hypoglycemics; Patients suspected of having fasting hypoglycemia for evaluation

### DIABETIC KETOACIDOSIS

What is the definition of diabetic ketoacidosis (DKA)? Diabetic ketoacidosis is a state of absolute or relative insulin deficiency aggravated by ensuing hyperglycemia, dehydration, and acidosis-producing derangements

What population is DKA primarily seen in? Predominately type 1 diabetics

What are some metabolic derangements that occur with DKA? Relative or absolute lack of insulin; Excessive stress hormones (i.e., cortisol); Overproduction of free fatty acids

What are three commonly seen ketone bodies? 1. Acetacetate 2. Beta-hydroxybutyrate 3. Acetone

Which ketone body is not measured in serum? Beta-hydroxybutyrate

List important precipitating factors of DKA? Infection (esp. PNA and UTI); Lack of insulin; Trauma; Surgery; Myocardial Infarction (MI) and cerebral vascular accident (CVA)

What are the clinical symptoms of DKA primarily due to? Volume depletion; Degree of hyperosmolality; Metabolic acidosis

What are some clinical features of DKA? Nausea, vomiting, and abdominal pain; partial motor seizures, visual changes, lethargy, obtundation, and coma; fruity breath and hyperventilation
### What are some commonly used diagnostic tests in DKA?
Complete blood count (CBC); Chem-7; Serum ketones; Calcium/magnesium/phosphorus; ECG—for changes in serum potassium

### What particular electrolyte is important to monitor in DKA?
Potassium

### What are some important confirmatory laboratory results in DKA?
- pH: <7.3; Bicarb: <10 mEq/L; Serum acetone: 2:1 ratio; Serum glucose: >350 mg/dL

### What are some key points in the management of DKA?
- ABCs and IV-O₂-monitor; Correct fluid losses, often require up to 5 L; IV infusion of low-dose insulin; Early potassium replacement; Consider use of bicarbonate (if pH <7.0)

### What are some complications that may occur when treating DKA?
- CHF from aggressive fluid resuscitation; Hypokalemia from not replacing potassium; Hypoglycemia from not monitoring glucose; Alkalosis from too much bicarbonate

### What is the primary cause of mortality in elderly patients with DKA?
Sepsis

### What is alcoholic ketoacidosis?
Accumulation of ketones in the blood, caused by excessive alcohol consumption and lack of food intake

### What are some clinical features of alcoholic ketoacidosis?
- Nausea, vomiting, and abdominal pain; partial motor seizures, visual changes, lethargy, obtundation, and coma

### What are some common laboratory findings in alcoholic ketoacidosis?
- High anion gap acidosis; Serum glucose—<200 mg/dL; Hypokalemia; Serum EtOH low or not present

### What are some key points in the management of alcoholic ketoacidosis?
- Large volume fluid replenishment; Early potassium replacement; Thiamine prior to glucose administration; Insulin typically not indicated

---

## THYROID

### Myxedema Coma (Hypothyroid)

### What is an important point to know about myxedema coma?
True emergency with up to 45% mortality
What is the normal physiologic mechanism of thyroid hormone production?
Hypothalamus—TRH; Anterior pituitary—thyroid-stimulating hormone (TSH); Thyroid—T3 and T4

What is primary hypothyroidism?
Intrinsic failure of the thyroid gland—most common cause

What are some common causes of primary hypothyroidism?
Partial thyroidectomy; Radioactive ablation; Autoimmune (i.e., Hashimoto’s thyroiditis); Iodine deficiency; Medications (i.e., lithium)

What is secondary hypothyroidism?
Hypothyroidism due to dysfunction of the pituitary or hypothalamus gland

What are some common causes of secondary hypothyroidism?
Pituitary tumor; Sarcoidosis; Sheehan’s syndrome

What are some common clinical features of hypothyroidism?
Cold-intolerance, hypoventilation, fatigue, constipation, weight-gain, memory loss, irregular menstruation, scaly skin, and muscle cramps

What is the definition of myxedema coma?
A rare and severe form of hypothyroidism typically due to undertreatment/undiagnosed

Who is commonly affected with myxedema?
Elderly women

What is the most common cause for the progression of myxedema to myxedema coma?
Physiological stressor (i.e., infections)

List some other common causes of myxedema coma?
Trauma; CHF; Medications (beta-blockers)

What are some important diagnostic tests to consider in the evaluation of myxedema coma?
TBG, TSH, and free T4; CBC (possible infection); Chem-7

What are some key points in the management of myxedema coma?
Supportive measures; Correction of electrolyte disturbances; Vasopressors for hypotension; Thyroxine IV pushed slowly; Hydrocortisone for adrenal insufficiency; Antibiotics for underlying infection; Search for underlying trigger

What is the disposition for patients who present with myxedema coma?
Generally admitted to ICU; Require endocrinologist consult
## Thyroid Storm

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What is the definition of hyperthyroidism?</strong></td>
<td>Elevated level of thyroid hormones can result in clinical manifestations ranging from mild to severely toxic with resultant morbidity and mortality for affected patients</td>
</tr>
<tr>
<td><strong>What is the most common form of hyperthyroidism?</strong></td>
<td>Grave’s disease</td>
</tr>
<tr>
<td><strong>What is Grave’s disease?</strong></td>
<td>Autoimmune condition in which autoantibodies are directed against the TSH receptor resulting in increase of thyroid hormone</td>
</tr>
<tr>
<td><strong>What are some common clinical features of hyperthyroidism?</strong></td>
<td>Heat intolerance, palpitations, fatigue, increased bowel movements, moist skin, insomnia, tremulous hands, and CNS hyperactivity</td>
</tr>
<tr>
<td><strong>What are some characteristic physical findings in a patient with hyperthyroidism?</strong></td>
<td>Exophthalmus, tachycardia, and palpable goiter</td>
</tr>
<tr>
<td><strong>What is thyroid storm?</strong></td>
<td>Thyroid storm is a decompensated state of thyroid hormone–induced severe hypermetabolism involving multiple systems</td>
</tr>
</tbody>
</table>
| **What are four diagnostic criteria used to diagnose thyroid storm?**    | 1. Temperature >37.8°C  
  2. Central nervous system (CNS) symptoms (i.e., obtundation)  
  3. Cardiovascular (tachycardia, dysrhythmias)  
  4. Gastrointestinal (i.e., diarrhea) |
| **What are some common triggers of thyroid storm?**                      | Infection; Grave’s disease; Trauma; MI                                                                                                                                                               |
| **What are some key points in the management of thyroid storm?**         | Supportive measures; Antithyroid medication (i.e., propylthiouracil); Iodine (suppresses release of T3/T4); Treat any other complications (i.e., afib); Glucocorticoids and propranolol often used |

## ADRENAL

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
</table>
| **What are the two major regions of the adrenal gland?**                | 1. Adrenal medulla  
  2. Adrenal cortex |
| **What major hormones does the adrenal medulla produce?**              | Catecholamines                   |
What three major hormones does the adrenal cortex produce? 1. Aldosterone 2. Glucocorticoid 3. Androgens

What are some common causes of primary adrenal insufficiency (Addison’s disease)? Infections (i.e., TB); Infiltrative (i.e., metastatic); Medications; Idiopathic atrophy

What is the name of the syndrome of bilateral adrenal hemorrhage secondary to meningococcemia? Waterhouse-Friderichsen syndrome

What are some common causes of secondary (pituitary dysfunction) adrenal insufficiency? Pituitary tumor; Head trauma; Infections; Infiltrative disease (i.e., sarcoidosis)

What is the most common cause of adrenal suppression? Iatrogenic steroid use (chronic)

What are some clinical features of adrenal insufficiency? Weak, fatigable, lethargic, postural hypotension/syncope secondary to aldosterone deficiency, nausea, abdominal pain, and emesis

What are characteristic laboratory findings in adrenal insufficiency? Hyponatremia and hyperkalemia; Hypoglycemia; Azotemia

What is adrenal crisis? Patients who have underlying chronic adrenal insufficiency and are exposed to any stress

What are some common stressors that can put a patient into adrenal crisis? Infections; Trauma; Surgery; Pregnancy

What are some common clinical features of adrenal crisis? Typically weak and very ill appearing, gastrointestinal (GI) affects (i.e., diarrhea), hypotension, delirium, and possible seizure

What are some key points in the management of adrenal crisis? IV fluids with dextrose; Glucocorticoid (dexamethasone)

CLINICAL VIGNETTES

28-year-old female presents with heat intolerance, fatigue, increased bowel movements, palpitations, and tremulous hands for months, but otherwise doing well; PE: exophthalmus, tachycardia, palpable goiter; Labs: increased TSH, reduced T4 and T3 levels

Grave’s disease
27-year-old male presents with weight loss, progressive weakness, nausea; PE: hyperpigmentation of skin; Labs: hyponatremia and hyperkalemia

41-year-old obese female presents with irregular menses, HTN, and increase in weight along with visual changes; PE: buffalo hump, hirsutism, and increase in BP; Labs: increased ACTH and suppression with high-dose dexamethasone test

21-year-old male presents with 2-week history of polyuria and polydipsia; U/A: urine osmolality <200 mosm/kg, hypernatremia, and urine specific gravity of <1.005

18-year-old male with history of type I diabetes mellitus presents with diffuse abdominal pain, nausea, and vomiting along with confusion; PE: shallow rapid breathing; Labs: glucose >300 and metabolic acidosis

34-year-old male is brought in by EMS for altered mental status and only knows that the patient is on sulfonylureas; PE: tachycardic, diaphoresis, and tremulousness

81-year-old female currently taking thyroid hormones presents via EMS with obtundation; PE: hypothermia, bradycardia, hypoventilation, cold non-pitting edema of legs; Labs: free T4 and T3 levels are low

Addison’s disease
Cushing’s disease
Diabetes insipidus
Diabetic ketoacidosis
Hypoglycemia
Myxedema coma
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## Hematology

### What is hemostasis?
It is an intrinsic balance between thrombosis and excessive bleeding.

### What are the major components of hemostasis?
- Platelets
- Vascular integrity
- Coagulation factors
- Fibrinolysis

### What are some components for bleeding disorders?
- Abnormal platelet function or count
- Missing factors in the coagulation cascade
- Excessive fibrinolysis
- Inflammation of blood vessel walls

### What are some important points for the following laboratory tests used to evaluate hemostasis:

<table>
<thead>
<tr>
<th>Test</th>
<th>Normal values and conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bleeding time (BT)</strong></td>
<td>Normal time 3–8 minutes; Measures integrity of platelet function; Increased BT in von Willebrand’s (vWF) disease and uremia; Aspirin and nonsteroidal anti-inflammatory drugs can affect BT</td>
</tr>
<tr>
<td><strong>Platelets</strong></td>
<td>Normal is 150,000–400,000 per mm$^3$; Decreased count: disseminated intravascular coagulation (DIC), uremia, idiopathic thrombocytopenic purpura (ITP), etc.; Increased count: consider malignancy; Less than 50,000 per mm$^3$: post-traumatic bleeding; Less than 20,000 per mm$^3$: life-threatening bleeding possible</td>
</tr>
<tr>
<td><strong>Prothrombin time</strong></td>
<td>Normal time is 10–12 seconds; Measures extrinsic (factor VII) pathway; Normal PTT but elevated</td>
</tr>
</tbody>
</table>
**Internationalized normalized ratio (INR)**

- PT: factor VII deficiency; Coumadin/Vitamin K/liver disease: < factor VII
- PT ratio: normal value 1; Monitor anticoagulation in Coumadin; INR 2–3 for most patients (i.e., afib); INR 2.5–3.5 for patients with mechanical valves

**Partial thromboplastin time (PTT)**

- Normal time is 25–35 seconds; Measures integrity of intrinsic pathway; Elevated in heparin use and hemophilia

---

### Bleeding Disorder

**What is one of the oldest hereditary bleeding disorders?**

- Hemophilia

**What are two types of hemophilia and their associated factor deficiency?**

1. Hemophilia A: lack of factor VIII
2. Hemophilia B: lack of factor IX

**What is the more prevalent form?**

- Hemophilia A

**What are some important elements in the bleeding history of hemophilia?**

- Hematomas; Hemarthrosis; Prolonged bleeding from dental procedures; Spontaneous hematuria; Epistaxis

**What are characteristic laboratory findings in hemophilia?**

- Prolonged PTT; Normal BT, PT, and platelets

**What is hemophilia A known as?**

- Classic hemophilia

**What are some important points to know about hemophilia A?**

- Sex-linked recessive disorder; Deficiency of factor VIII; Intracranial hemorrhage major cause of death

**What are some key points in the management of hemophilia A?**

- Infusion of Factor VIII; Desmopressin (DDAVP); Cryoprecipitate (not used often)

**What is hemophilia B known as?**

- Christmas Disease

**What are some important things to know about hemophilia B?**

- Sex-linked recessive disorder; Deficiency of factor IX; Comprises about 15% of all hemophilias

**What are some key points in the management of hemophilia B?**

- Factor IX concentrate; Fresh frozen plasma (FFP)

**What is vWF?**

- Autosomal dominant with either quantitative or qualitative disorder in vWF
What is the primary defect in each of the following forms of vWF disease:

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>A low level of the vWF factor; Mildest and most common form</td>
</tr>
<tr>
<td>Type II</td>
<td>Qualitative disorder of vWF</td>
</tr>
<tr>
<td>Type III</td>
<td>Virtual absence of vWF; Most serious, but rare form</td>
</tr>
</tbody>
</table>

What are some common clinical features of vWF disease?

- Mucocutaneous bleeding is the defining feature (i.e., epistaxis)—bleeding is milder then hemophilia A

What are classic laboratory findings in a patient with vWF disease?

- Increased BT; Increased PTT; Normal platelet count and function; Normal PT

What are treatment options available for vWF disease?

- FFP; Cryoprecipitate; DDAVP

### Platelet Disorders

What is the most common platelet dysfunction?

- Thrombocytopenia

What are some important causes of thrombocytopenia?

- Decreased platelet production;
- Increased platelet destruction;
- Increased splenic clearance

What are some examples for the following causes of thrombocytopenia?

- **Decreased platelet production.**
  - Aplastic anemia; Radiation; Myelofibrosis
- **Increased platelet destruction.**
  - Sepsis; Thrombotic thrombocytopenic purpura (TTP); HELLP syndrome

What are some clinical features of thrombocytopenia?

- Mucocutaneous bleeding (i.e., epistaxis), hematuria, menorrhagia, and GI bleeding

What are common laboratory findings in a patient with thrombocytopenia?

- Low platelets and increased BT; PT (INR) and PTT will be normal

What are a few indications for platelet transfusion in a patient with thrombocytopenia?

- Platelet <50,000 per mm³ and major bleeding; Platelet <20,000 per mm³

What is the most feared complication of thrombocytopenia?

- ICH

What is the most common hemorrhagic disease in the pediatric population?

- ITP
**What are some important things to know about ITP in children?**

Typically occurs in patients between 2 and 8 years of age; Generally self-limited and resolves in weeks; Often triggered by viral infections.

**What are some key points in the management of ITP in children?**

Treatment primarily supportive; Transfusion: major bleeding/platelet count; Consider use of steroids—dexamethasone.

**What age group typically manifests with ITP refractory to standard treatment of steroids?**

Females between 25 and 40 years of age.

**What are some treatment options for cases of ITP that are refractory to steroids?**

Platelet transfusion; Immunosuppressive drugs; Splenectomy.

**What is a platelet disorder that has a very high mortality rate if left untreated?**

TTP.

**What are some common clinical features of TTP?**

Thrombocytopenic purpura, hemolytic anemia, mental status change, fever, and renal disease.

**What is the treatment of choice for TTP?**

Fresh frozen plasma; Plasmapheresis.

**What are other treatment options to consider in TTP?**

Steroids; Splenectomy; Heparin.

**What treatment is generally avoided in TTP?**

Platelet transfusion.

**What is disseminated intravascular coagulation (DIC)?**

A life-threatening disorder that is a characterized by: Depletion of platelets; Depletion of coagulation factors; Small vessel occlusions; Fibrinolysis; Hemolytic anemia.

**What are some important causes of DIC?**

Sepsis; Trauma; Drug reactions; Snake bites; Cancer.

**What are some common clinical features of DIC?**

Bleeding, petechiae, thrombosis, and possible gangrenous changes.

**What are some classic laboratory findings in a patient with DIC?**

PT (INR) and PTT increased; Decreased platelet count; Decreased fibrinogen; Increased thrombin time; Increased D-dimer.

**What are some key points in the management of DIC?**

Important to find the underlying cause; Intravenous (IV) fluids; Packed red blood cells (PRBC) as needed; If serious hemorrhage—consider: FFP, platelets, and cryoprecipitate.
## Sickle Cell Disease

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important features of sickle cell disease?</td>
<td>Characterized by abnormal hemoglobin—HbS; High prevalence in African Americans</td>
</tr>
<tr>
<td>What are some characteristic features of HbS?</td>
<td>RBC sickling responsible for majority of symptom; Sickled RBCs are more easily hemolyzed; RBCs sensitive to hypoxia (i.e., sickling)</td>
</tr>
<tr>
<td>What is sickle cell trait?</td>
<td>Occurs when a child inherits HbS from one parent and HbA from another parent, so most RBCs will contain both types</td>
</tr>
<tr>
<td>What are some clinical features of sickle cell trait?</td>
<td>Generally asymptomatic; Spontaneous bleeding; Decreased ability to concentrate their urine; Laboratory evaluation is normal</td>
</tr>
<tr>
<td>What are some complications of sickle cell trait?</td>
<td>Splenic infarction; Vaso-occlusive crisis; Death</td>
</tr>
<tr>
<td>Are these complications common?</td>
<td>Rarely occur unless extreme hypoxia</td>
</tr>
<tr>
<td>What is sickle cell anemia?</td>
<td>HbS that is inherited from both parents where most RBCs have only HbS</td>
</tr>
<tr>
<td>What are some clinical features of sickle cell anemia?</td>
<td>Anemia; Jaundice; Hand-foot syndrome (swelling of foot/hand); Frequent infections; Vision problems</td>
</tr>
<tr>
<td>What infections are patients with sickle cell anemia more susceptible to?</td>
<td>Pneumonia; Meningitis; Sepsis; Osteomyelitis</td>
</tr>
<tr>
<td>What particular pathogens are those with sickle cell anemia more prone to?</td>
<td>Salmonella; <em>Haemophilus influenzae</em>; <em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td>What are some complications that occur with sickle cell anemia?</td>
<td>Aplastic crisis; Vaso-occlusive crisis (i.e., pain crisis); Acute chest syndrome; Cerebral vascular accident (CVA); Renal papillary necrosis; Priapism</td>
</tr>
<tr>
<td>What are some key points in the management of patients who present with sickle cell crisis?</td>
<td>Analgesics; IV hydration; Oxygen; Antibiotics if suspected infection</td>
</tr>
</tbody>
</table>
ONCOLOGY

Hypercalcemia (Secondary to Malignancy)

What is important to know about hypercalcemia secondary to malignancy?
Common life-threatening disorder associated with cancer

What are some important causes of hypercalcemia associated with malignancy?
Parathyroid hormone (PTH) (i.e., squamous cell lung carcinoma); Osteoclast-activating factor; Bone degradation (metastasis to bones)

What are some clinical features of hypercalcemia associated with malignancy?
Fatigue; Nausea and vomiting; Constipation; Back pain; Hypertension

What are some diagnostic studies to consider?
Calcium and phosphorus; Alkaline phosphatase; Chem-7 (chloride and potassium); Albumin; ECG (shorten QT interval); PTH

What is the mainstay treatment to quickly reduce ionized calcium?
IV normal saline and furosemide; Magnesium and potassium; Bisphosphonates (i.e., pamidronate); Steroids

Tumor Lysis Syndrome

What is tumor lysis syndrome (TLS)?
Constellation of metabolic disturbances that may be seen after initiation of cancer treatment

In what types of cancer does TLS occur?
Occurs in patients with rapidly proliferating, and treatment-responsive tumors

When is TLS commonly seen?
Most often is seen 48–72 hours after initiation of cancer treatment

What is the pathophysiology of TLS?
Rapid tumor cell turnover results in release of intracellular contents into the circulation which can inundate renal elimination

What are some common laboratory findings in patients who have TLS?
Hyperkalemia (first derangement); Hyperuricemia; Hyperphosphatemia (hypocalcemia)

What are some complications of TLS?
Dysrhythmias (hyperkalemia); Urate nephropathy; Acute renal failure; Neuromuscular instability; Metabolic acidosis
What is the most common cause of acute renal failure in the setting of TLS?
Hyperuricemia

What is the mainstay treatment for hyperuricemia?
Allopurinol; IV fluids; Alkalinization of urine

What are some key points in the management of TLS?
IV fluids; Hemodialysis in life-threatening situations; Serial chem-7 with calcium and phosphorus; Treat hyperuricemia

**Syndrome of Inappropriate ADH Syndrome**

<table>
<thead>
<tr>
<th>What malignancies are commonly associated with SiADH</th>
<th>Small cell lung cancer—most common; Brain; Prostate; Pancreas</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some diagnostic criteria of SIADH?</td>
<td>Hyponatremia (serum sodium &lt;135 mEq/L); Inappropriately concentrated urine; Clinical euvoeçemia</td>
</tr>
<tr>
<td>What are some common clinical features of SIADH?</td>
<td>Mental status change, weakness, dizziness, and seizures/coma in severe cases: severity is determined by rate of sodium loss</td>
</tr>
<tr>
<td>What are some diagnostic tests to obtain in SIADH?</td>
<td>Urinalysis, urine sodium/osmolality; Chem-7 and serum osmolality</td>
</tr>
<tr>
<td>What are some key points in the management of SIADH?</td>
<td>Water restriction; Furosemide; 3% saline given slowly for severe cases</td>
</tr>
</tbody>
</table>

**Spinal Cord Compression**

<table>
<thead>
<tr>
<th>What is spinal cord compression?</th>
<th>Spread of cancer to the spine and tissues around the spinal cord that may result in compression of the cord: oncologic emergency</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the initial presenting feature of spinal cord compression?</td>
<td>Back pain (commonly thoracic)</td>
</tr>
<tr>
<td>What are some other clinical features of spinal cord compression?</td>
<td>Sensory deficits; Lower extremity weakness/paralysis; Urinary incontinence; Urinary retention</td>
</tr>
<tr>
<td>What are some malignancies commonly associated with spinal cord compression?</td>
<td>Lung cancer (most common); Breast cancer; Prostate cancer; Multiple myeloma</td>
</tr>
<tr>
<td>What is the diagnostic test of choice for spinal cord compression?</td>
<td>MRI</td>
</tr>
</tbody>
</table>
What are some other diagnostic tests to consider? | Plain films; CT; Myelography (considerable complications)
---|---
What are some key points in the management of spinal cord compression? | Steroids to reduce edema and inflammation; Radiation and neurosurgical intervention

**Superior Vena Cava Syndrome**

| What are some important things to know about superior vena cava syndrome (SVCS)? | Gradual compression of the SVC; SVCS is associated chiefly with malignancy; Bronchogenic CA accounts for more than 80% of cases of SVCS
---|---
What are some other causes of SVCS aside from malignancy? | Thrombosis (central venous instrumentation); Infectious causes (i.e., tuberculosis/syphilis); Lymphoma
What are some clinical features of SVCS? | Venous distension of face/upper extremity, facial flushing, headache, JVD, cough, and dyspnea
What are some important diagnostic tests to consider in SVCS? | CXR: Mass or widened mediastinum; Thoracic CT: test of choice; Histological sample: important for therapy
What are some key points in the management of SVCS? | ABCs: rarely present acutely; Tissue diagnosis for palliative therapy; Elevation of head provides some relief

**Adrenal Insufficiency**

| What are two important hormones produced by the adrenal cortex? | 1. Aldosterone 2. Cortisol
---|---
What are some clinical features of adrenal insufficiency? | Patient is often hypotensive with dehydration and may present with vasomotor collapse as well as weakness
What are common laboratory findings in adrenal insufficiency? | Hyperkalemia; Hyponatremia; Hypoglycemia; Hypercalcemia
What are some common causes of adrenal insufficiency in the setting of malignancy? | Malignant melanoma; Breast cancer; Lung cancer; Chronic steroid withdrawal
## Malignant Pericardial Effusion

### What is the pathophysiology of malignant pericardial effusion?

Normally is lubricated by a very small amount of serous fluid, malignant involvement of the pericardium may be primary (less common) or secondary to spread from a nearby or distant focus of malignancy.

### What are some clinical features of patients with malignant pericardial effusion?

Often asymptomatic, but most common symptom is dyspnea, and can include cough, chest pain, and hypotension. PE: JVD, pulsus paradoxus, distant heart sound, and pericardial friction rub.

### Name some malignancies commonly associated with malignant pericardial effusion.

Leukemia; Breast cancer; Lung cancer; Melanoma

### What are commonly used diagnostic tests and possible findings.

- **ECG.** Low-voltage QRS complexes; Electrical alternans; ST-segment elevation and T-wave inversion
- **CXR.** Massive effusions = large cardiac shadow; Pleural effusion/mediastinal widening/mass
- **CT.** As little as 10 ml of pericardial fluid can be seen as a irregular contour of the cardiac silhouette
- **Echocardiography.** Test of choice (highly specific and sensitive); Used for guiding needle pericardiocentesis

### What are some key points in the management of malignant pericardial effusion?

Supportive care (IV fluids/inotrope if needed); Pericardiocentesis is definitive treatment; Tamponade can be the presenting symptom.
CLINICAL VIGNETTES

8-year-old male presents with a long history of intermittent epistaxis along with prolonged bleeding whenever he goes for any dental procedures, patient’s mother is now concerned since patient is having recent hematuria; Labs: prolonged PTT, but normal PT time as well as platelet count

Hemophilia A

21-year-old female with no known PMH presents to the ER with concern of nose bleeding that has become frequent; Labs: abnormal PTT and BT, but normal platelet count and PT

von Willebrand’s disease

6-year-old male is brought in by his mother due to concerns of episodes of sudden nose bleeding about a week after an upper respiratory illness (URI), patient is otherwise healthy; Labs: CBC otherwise unremarkable except for platelet count of 5,000

Idiopathic thrombocytopenic purpura

17-year-old female with a recent snake bite presents with hypotension, confusion, fever, and gingival bleeding; Labs: increased INR and PTT time, decreased platelets with decreased fibrinogen and increased thrombin time

DIC

23-year-old AA male with sickle cell disease presents with a recent cold and excruciating pain in his limbs; PE: unremarkable

Vaso-occlusive crisis

71-year-old female with a history of untreated squamous cell lung cancer presents with fatigue, constipation, and back pain; ECG shows shortened QT interval

Hypercalcemia

81-year-old male with recently treated cancer presents with weakness, flank pain, dysuria, and abdominal pain; Labs: elevated potassium, LDH, BUN/creatinine, and uric acid

Tumor lysis syndrome
65-year-old female with history of HTN, breast cancer, and CAD is concerned with recent onset of urinary retention and lower extremity weakness; PE: bilateral leg weakness

76-year-old male with history of bronchogenic cancer now presents with dyspnea and cough for about 1 month which is becoming more progressive; PE: obvious venous distension of face and JVD

56-year-old female with breast cancer in the past presents with chest pain and dyspnea for the past 2 weeks which is getting progressively worse; PE: distant heart sound, pulsus paradoxus, and JVD; ECG: low-voltage QRS complex

Hematology and Oncology Emergencies
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### INFLUENZA AND HERPES VIRUSES

#### Influenza Virus

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are influenza viruses?</td>
<td>Single-stranded RNA viruses that fall within the orthomyxovirus family with three types—A, B, and C</td>
</tr>
</tbody>
</table>
| Name two surface glycoproteins that are responsible for the pathogenicity of the influenza virus. | 1. Hemagglutinin (H)  
2. Neuraminidase (N)                                                                                                                                                                                                                                                                                                              |
| What is antigenic drift?                                                 | Minor mutations in the RNA genome that code for N or H molecule causing a change in antigenicity                                                                                                                                                                                                                                           |
| What is antigenic shift?                                                 | Occurs when a host is infected with two different influenza viruses, producing a new virus with little antigenic similarity to the old                                                                                                                                                                                                    |
| When does the flu generally occur in the United States?                 | The fall and spring                                                                                                                                                                                                                                                                                                                   |
| What are some clinical features of the flu?                             | Headache, fever, chills, myalgia and malaise often with rhinorhea, sore throat, enlarged cervical lymph nodes, and a dry cough                                                                                                                                                                                                              |
| What is the typical time course for the flu?                            | Fever that lasts for 2–4 days with rapid recovery, although cough and malaise may last longer                                                                                                                                                                                                                                               |
| What are some complications of an influenza infection?                  | Secondary bacterial pneumonia; Pneumonitis; Croup; Chronic obstructive pulmonary disease (COPD) exacerbation; Reye’s syndrome (if ASA used)                                                                                                                                                                                                  |
Name two medications currently approved for the treatment of influenza A.  
1. Rimantadine  
2. Amantadine

What are some points with regard to rimantadine and amantadine?  
Should be started within 48 hours of symptoms; Amantadine is renally cleared; Rimantadine is hepatically cleared

Name two medications approved for treatment of influenza A and B?  
Zanamivir and Oseltamivir

What is the flu vaccine?  
It is made annually and contains two strains of influenza A and one strain of influenza B

Which groups should receive the influenza vaccine?  
Anyone with cardiopulmonary disease; Immunocompromised patients; Healthcare workers; Patients over 65

Herpes Virus

What are some important facts about the herpes virus family?  
They are an ubiquitous group of DNA viruses; Ability to remain in a host as a lifelong latent infection that can reactivate; Commonly transmitted by close contact

What is the pathophysiology of herpes simplex virus (HSV) exposure?  
Infects and replicates in epithelial cells, causing lysis of the cell leading to an inflammatory response and the characteristic HSV rash

What is the general appearance of a HSV rash?  
Clusters of small, thin-walled vesicles on a erythematous base

What are some clinical features of oral HSV?  
Primarily caused by HSV-1, but can be caused by HSV-2 that range from asymptomatic to pharyngitis or gingivostomatis with fever and cervical adenopathy

How is oral HSV diagnosed?  
Typically made clinically, although viral cultures can be used (takes days)

What is the oral distribution of the lesions?  
All over the mouth

What is the recurrence rate of oral lesions?  
Vary from 60–90%, but recurrences tend to be milder

What are some triggers for an HSV recurrence?  
Stress; Trauma; Sunburn
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What role does acyclovir play in oral HSV?</td>
<td>Shown to shorten course if given within 72 hours of symptoms and can be used as prophylaxis in severe cases</td>
</tr>
<tr>
<td>What are some important points about genital herpes?</td>
<td>Majority caused by HSV-2; Recurrent lesions can cause intrauterine infections; C-section if active lesions are present during a pelvic exam</td>
</tr>
<tr>
<td>What is the most common manifestation of ocular HSV?</td>
<td>Ulcerative keratitis</td>
</tr>
<tr>
<td>What is the most feared complication of ocular HSV?</td>
<td>Recurrent infections leading to blindness</td>
</tr>
<tr>
<td>What are some clinical features of ocular HSV?</td>
<td>Herpetic vesicles on the conjunctiva or the lid margin and fluorescein staining that shows dendritic ulcerations</td>
</tr>
<tr>
<td>What are some key points in the management of ocular HSV?</td>
<td>Consultation with ophthalmology; Administration of IV acyclovir; Avoid the use topical steroids</td>
</tr>
<tr>
<td>What is one of the most common viral encephalitis?</td>
<td>HSV encephalitis (usually HSV-1)</td>
</tr>
<tr>
<td>What portion of the brain is typically involved?</td>
<td>Temporal lobes</td>
</tr>
<tr>
<td>What are some clinical features of HSV encephalitis?</td>
<td>Often a viral prodrome which may be followed by HA, fever, altered mental status, and even focal seizures</td>
</tr>
<tr>
<td>What does a lumbar puncture often show?</td>
<td>Nonspecific—elevated WBC count with an increase in mononuclear cells</td>
</tr>
<tr>
<td>What is the test of choice for diagnosing HSV encephalitis?</td>
<td>PCR</td>
</tr>
<tr>
<td>What is the treatment of choice for suspected HSV encephalitis?</td>
<td>Intravenous acyclovir</td>
</tr>
<tr>
<td>What is an HSV infection of the finger known as?</td>
<td>Herpetic whitlow</td>
</tr>
<tr>
<td>What is the concern of a patient who is immunocompromised with HSV?</td>
<td>Dissemination or severe HSV infection</td>
</tr>
<tr>
<td>What are some complications of HSV in an immunocompromised patient?</td>
<td>Proctitis, esophagitis, colitis, and pneumonitis</td>
</tr>
<tr>
<td>What is the cause of chickenpox?</td>
<td>Varicella-zoster virus (VZV)</td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>What is some important epidemiologic information about VZV?</td>
<td>Chickenpox is the primary infection; Zoster (shingles) is reactivation of VZV; Prior to vaccine, over 90% of primary infections occurred to those &lt;10 years</td>
</tr>
<tr>
<td>What is the dermatologic hallmark of chickenpox?</td>
<td>Skin lesions in various stages throughout the body</td>
</tr>
<tr>
<td>What are some clinical features of chickenpox?</td>
<td>Prodrome of fever, HA, and malaise followed by clear vesicles on an erythematous base which eventually scab over</td>
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<td>What are some serious complications of chickenpox?</td>
<td>Cerebellar ataxia; Pneumonitis; Encephalitis</td>
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<td>Who is oral acyclovir recommended for?</td>
<td>Patients older then 14 years of age; Patients on chronic ASA therapy</td>
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<td>Who should receive IV acyclovir?</td>
<td>Patients suffering from varicella encephalitis/pneumonitis</td>
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<td>What is herpes zoster (shingles)?</td>
<td>Reactivation of latent VZV infection with a lifetime incidence of 25%, especially in the elderly</td>
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<td>What are some clinical features of shingles?</td>
<td>Vesicular lesions similar to chickenpox in a single dermatome that may persist for up to a month</td>
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<td>What is herpes zoster ophthalmicus (HZO)?</td>
<td>Involvement of the ophthalmic branch of cranial nerve (CN) V which can threaten vision and also cause a lesion on the tip of the nose (Hutchinson’s sign)</td>
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<td>What is the most common complication of shingles?</td>
<td>Postherpetic neuralgia</td>
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<td>What are some clinical features of postherpetic neuralgia?</td>
<td>Severe pain and occasional involvement of the anterior horn cells leading to transient weakness</td>
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<td>What is the initial treatment for postherpetic neuralgia?</td>
<td>Systemic analgesia such as narcotics; Carbamazepine may work as a second-line treatment</td>
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<td>What is the role for the use of antivirals in shingles?</td>
<td>If used within 72 hours, may decrease the duration of the disease</td>
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<tr>
<td>What is the primary cause of infectious mononucleosis?</td>
<td>Epstein-Barr virus (EBV)</td>
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<td>How is EBV typically spread?</td>
<td>Close contact such as kissing, EBV cannot survive outside the host for long</td>
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<tr>
<td>What is the pathophysiology of infectious mononucleosis?</td>
<td>1–2 month incubation period where the EBV replicates in B lymphocytes resulting in the production of anti-EBV antibodies and heterophil antibodies</td>
</tr>
<tr>
<td>What are some clinical features of infectious mononucleosis?</td>
<td>Fever, HA, exudative pharyngitis, splenomegaly, atypical lymphocytosis, and bilateral cervical lymphadenopathy</td>
</tr>
<tr>
<td>What are some complications of infectious mononucleosis?</td>
<td>Splenic rupture; Thrombocytopenia; Autoimmune hemolytic anemia; Meningitis; Encephalitis</td>
</tr>
<tr>
<td>How is the diagnosis of EBV typically made?</td>
<td>Clinical features of EBV along with atypical lymphocytes and (+) monospot test are generally confirmatory</td>
</tr>
<tr>
<td>What are some epidemiologic features of CMV?</td>
<td>Ubiquitous virus found worldwide; Causes primary infection and often exists as a latent infection; Not easily spread by casual contact</td>
</tr>
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<td>What are some clinical features of CMV infection?</td>
<td>Often asymptomatic in healthy people, but can appear as flu-like symptoms such as fever, chills, and myalgia</td>
</tr>
<tr>
<td>When should CMV infection be suspected in healthy adults?</td>
<td>Mononucleosis-like illness, but heterophil antibody negative</td>
</tr>
<tr>
<td>What are some complications of CMV infection in healthy individuals?</td>
<td>Guillain-Barré syndrome; Hepatitis; Hemolytic anemia; Pneumonitis; Thrombocytopenia</td>
</tr>
<tr>
<td>In what population group can CMV be particularly devastating?</td>
<td>HIV; Transplant recipients</td>
</tr>
<tr>
<td>What is the most common CMV infection in patients with advanced HIV?</td>
<td>CMV retinitis</td>
</tr>
<tr>
<td>What are some CMV infections to consider in transplant recipients?</td>
<td>Hepatitis; Colitis; CNS disease</td>
</tr>
<tr>
<td>What is the most serious CMV infection in transplant patients?</td>
<td>CMV pneumonia</td>
</tr>
<tr>
<td>What are some ways in which transplant patients can contract CMV infection?</td>
<td>Blood products or transplant organ; Reactivation of latent infection</td>
</tr>
<tr>
<td>When does CMV infections most commonly occur in transplant recipients?</td>
<td>Within 3 months of transplantation</td>
</tr>
<tr>
<td>What are two medications are used in CMV infections?</td>
<td>1. Ganciclovir 2. Foscarnet</td>
</tr>
</tbody>
</table>
**HIV/AIDS**

What are some important points about the HIV virus?  
Cytopathic retrovirus of the lentivirus family; Very labile outside the body; Two major subtypes: HIV-1 and HIV-2; Selectively attacks CD4+ T-cells

What are some risk factors for the development of HIV infection?  
Intravenous drug use; Vertical transmission; Unprotected sex

What is the most common presentation of acute HIV infection?  
Fever, pharyngitis, fatigue, rash, and headache

Why is the diagnosis of HIV infection initially difficult?  
The nonspecific presentation, which often resembles flu-like symptoms

What is seroconversion?  
Detectable antibodies in response to HIV that usually occurs between 4–8 weeks, but can be delayed for up to a year

What is the average time frame from initial HIV infection to the development of AIDS?  
8–10 years

What are some conditions that may indicate AIDS?  
Kaposi’s sarcoma; Pneumocystis carinii pneumonia; Brain toxoplasmosis; Cryptococcosis; Mycobacterium avium complex; CD4+ T-cell count <200 cells/µL

What is the standard and most common way to diagnosis HIV infection?  
Detection of the antibodies to the virus by Western blot assay or ELISA

What are two useful things to know when a patient with HIV presents to the ED?  
1. CD4+ T-cell count  
2. HIV viral load

What are some numbers to keep in mind about CD4+ T-cell count and HIV viral load?  
CD4+ T-cell count of <200 and HIV viral load >50,000 is often associated with progression to AIDS-defining illness and an indication to start antiretrovirals

What are some differentials to keep in mind about HIV-infected patients who present with fever based on CD4+ T-cell count:  

- **CD4+ T-cell count >500**  
  Cause of fever similar to healthy patients who are non-immunocompromised

- **CD4+ T-cell count 200–500**  
  Early bacterial respiratory infection
CD4+ T-cell count <200

What is the most common cause of serious opportunistic viral disease in HIV-infected patients?

What is the most common cause of fever that is noninfectious in origin?

What is an important diagnosis to keep in mind about HIV-infected patients with a history of intravenous drug abuse (IVDA)?

Name the three most common causes of CNS disease in HIV-infected patients?

What are some clinical features that are indicative of CNS disease?

What should an ED evaluation include for HIV-infected patients who present with neurologic symptoms?

What is important to know about toxoplasmosis in patients with AIDS?

What is the treatment of choice for patients with suspected toxoplasmosis?

What should be given for HIV-infected patients who have positive toxoplasmosis antibodies and CD4+ T-cell count <100 cells/µL?

What are some presenting symptoms of cryptococcal CNS infection?

How is the diagnosis of cryptococcal CNS infection commonly made?

What is the preferred treatment for patients with cryptococcal CNS infection?

What are some other important CNS infections in consider?

What is the most frequent and serious ocular opportunistic infection of HIV-infected patients?

What is the treatment of choice for patients with CMV retinitis?
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important pulmonary infections to keep in mind with HIV-infected patients?</td>
<td>Bacterial pneumonia; CMV infection; TB; <em>Pneumocystis carinii</em> pneumonia (PCP); <em>C. neoformans</em>; Neoplasms</td>
</tr>
<tr>
<td>Which disease is the most serious complication and common cause of death in HIV-infected patients?</td>
<td><em>P. carinii</em> pneumonia</td>
</tr>
<tr>
<td>What are some clinical features of PCP?</td>
<td>Nonproductive cough, fever, and shortness of breath with diffuse interstitial infiltrates on CXR</td>
</tr>
<tr>
<td>What is the medication of choice for PCP?</td>
<td>TMP-SMX; Pentamidine isothionate</td>
</tr>
<tr>
<td>What are some clinical features of TB in HIV-infected patients?</td>
<td>Fever, hemoptysis, weight loss, night sweats, and anorexia</td>
</tr>
<tr>
<td>What is the CD4⁺ T-cell count where TB is more common?</td>
<td>CD4⁺ T-cell count 200–500 cells/µL</td>
</tr>
<tr>
<td>Does a negative PPD test in an HIV-infected patient rule out TB?</td>
<td>No—can be negative due to immunosuppression</td>
</tr>
<tr>
<td>What is a common treatment option for HIV-infected patients with TB?</td>
<td>INH and pyridoximine</td>
</tr>
<tr>
<td>What are some common oral/esophageal complaints in HIV-infected patients?</td>
<td>Oral candidiasis (most common); HSV; Oral hairy leukoplakia</td>
</tr>
<tr>
<td>What is the most frequent GI complaint in HIV-infected patients?</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>What are some common causes of diarrhea in HIV-infected patients?</td>
<td>Shigella; <em>Isospora belli</em>; <em>E. coli</em>; <em>Cryptosporidium</em></td>
</tr>
<tr>
<td>What are some common generalized cutaneous conditions in HIV-infected patients?</td>
<td>Seborrheic eczema; Pruritus; Xerosis</td>
</tr>
<tr>
<td>What is the appearance of Kaposi’s sarcoma?</td>
<td>Painless dark papules/nodules that do not blanch</td>
</tr>
<tr>
<td>What are some other important causes of skin lesions to consider?</td>
<td>HSV; Zoster; Scabies; Syphilis</td>
</tr>
<tr>
<td>What are some important treatment goals for HIV-infected patients?</td>
<td>Prolongation and improvement of life; Reduction of viral load; Improved CD4⁺ T-cell count; Maintain drug regimen with minimal ADR</td>
</tr>
</tbody>
</table>
SEXUALLY TRANSMITTED DISEASES

What are some important elements to establish when evaluating a patient for sexually transmitted diseases (STDs)?
- Pregnancy status;
- Sexual practice;
- Evaluate for sexual abuse;
- Evaluate for domestic violence

What infection commonly coexists with gonorrhea?
- *Chlamydia trachomatis*

What are some facts about chlamydial infections?
- Common cause of nongonococcal infection;
- Often asymptomatic in patients

What are some clinical features of chlamydial infections?
- Urethritis, dysuria, vaginal discharge, and proctitis

Name two important complications of chlamydial infections in females if left untreated.
- 1. Pelvic inflammatory disease (PID)
- 2. Infertility

Name some antibiotics commonly used to treat nongonococcal urethritis/cervicitis?
- Azithromycin; Doxycycline

What are some clinical features of gonococcal urethritis/cervicitis?
- Males tend to have dysuria and purulent penile discharge while females tend to have more nonspecific symptoms such as lower abdominal pain

What are some factors that contribute to complications of gonococcal infection?
- Poor detection method;
- Subclinical presentation (esp. females)

How common is disseminated gonococcal infection if left untreated?
- About 5%

What are some clinical features of disseminated gonococcal infection?
- Fever, malaise, skin lesions on an erythematous base, and asymmetric arthralgias

What is the standard for the diagnosis of gonococcal infection?
- Cervical or urethral culture

Name the antibiotics commonly used to treat gonococcal urethritis/cervicitis?
- Ceftriaxone or Ciprofloxacin

What is important to keep in mind about using fluoroquinolones for gonococcal infections?
- Increasing resistance in certain areas like California and Asia

Name five diseases that are characterized by genital lesions?
- 1. Syphilis
- 2. HSV
- 3. Lymphogranuloma venereum
- 4. Granuloma inguinale
- 5. Chancroid
What is the causative organism of syphilis? *Treponema pallidum*; The spirochete enters the body through the mucous membrane or non-intact skin

What are the three phases of syphilis and some important points regarding each phase:

<table>
<thead>
<tr>
<th>Phase</th>
<th>Hallmark</th>
<th>Incubation Period</th>
<th>Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>Painless chancre</td>
<td>About 3 weeks</td>
<td>Disappear after a month</td>
</tr>
<tr>
<td>Secondary</td>
<td>Nonspecific symptoms</td>
<td>Fever and malaise</td>
<td>Rash starts at the trunk and moves towards the palms/soles</td>
</tr>
<tr>
<td>Tertiary (latent)</td>
<td>CVS/CNS involvement is characteristic</td>
<td>Granulomatous lesions are common</td>
<td>Meningitis, dementia, tabes dorsalis, and thoracic aneurysm more likely</td>
</tr>
</tbody>
</table>

What are some methods for diagnosing syphilis? RPR, VDRL, and dark-field microscopy

What is the treatment of choice for syphilis? Penicillin; Doxycycline for penicillin allergy

What is the causative agent of chancroid? *Haemophilus ducreyi*

What are some clinical features of chancroid? Painful genital ulcer and lymphadenitis/abscess/periadenitis if left untreated

What are some other infections to consider in patients with chancroid? Syphilis, HSV, and HIV

How is the diagnosis of chancroid typically made? Typically made clinically

What are some antibiotics commonly used to treat chancroid? Azithromycin; Ceftriaxone; Ciprofloxacin

What is the causative agent of *C. trachomatis* lymphogranuloma venereum (LGV)? *C. trachomatis*

What are some clinical features of LGV? Painless primary chancre of short duration, lymphadenopathy, and systemic effects such as fever, arthralgias, and erythema nodosa

How is the diagnosis of LGV commonly made? Culture and serologic tests

What is the treatment of choice for LGV? Doxycycline
What is the causative agent of granuloma inguinale? 
**Calymmatobacterium granulomatis**

What are some clinical features of granuloma inguinale? 
Subcutaneous nodules on penis or labia/vulva area after incubation which progresses to a painless ulcerative lesion with a “beefy” appearance

How is the diagnosis of granuloma inguinale commonly made? 
Difficult to culture; visualization of Donovan bodies on tissue biopsy is characteristic

What is the treatment of choice for granuloma inguinale? 
Doxycycline and bactrim; Ciprofloxacin and azithromycin

What is the causative agent of genital warts? 
Human papillomavirus (HPV)

What is the typical appearance of HPV? 
Flesh-colored papules or cauliflower-like projection that is often painless

How is HPV commonly diagnosed? 
Often clinically, but can be done with PCR

What is an important long-term complication of HPV to consider? 
Cervical cancer

What HPV types are commonly associated with cervical cancer? 
HPV type 16 and 18

What are risk factors associated with acquisition of HPV? 
Increasing number of partners; Early age of first sexual intercourse

What is the primary reason for treatment of HPV? 
Removal of visible warts for cosmetic reasons

What are some treatment options for visible lesions of HPV? 
Cyrotherapy; Surgical removal; Podophyllin resin

Is there a vaccine for HPV? 
Yes—a quadrivalent vaccine that protects against HPV types 6, 11, 16, and 18. These four are responsible for 70% of cervical warts and 90% of genital warts

---

**MALARIA**

What is the primary vector for the transmission of malaria? 
**Anopheles** mosquito

Name four species that are responsible for malaria? 
1. *Plasmodium vivax*
2. *Plasmodium ovale*
3. *Plasmodium malariae*
4. *Plasmodium falciparum*
Which species of *Plasmodium* is the most deadly form of malaria?  
*Plasmodium falciparum*

Name some locations in the world where malaria transmission primarily occurs?  
Caribbean; Middle East; Central America; Indian subcontinent

Name two species of *Plasmodium* that can lie dormant for months and cause clinical relapse?  
1. *Plasmodium vivax*  
2. *Plasmodium ovale*

What form of the parasite is injected into the bloodstream when a mosquito takes its bloodmeal?  
Sporozoites

What form of the parasite invades the erythrocytes?  
Merozoites

What are some clinical features of malaria?  
Nonspecific: fever, HA, myalgia, and malaise; PE: splenomegaly and tender abdomen in acute infections

What are some common laboratory findings in malaria?  
Elevated ESR and LDH, mildly abnormal kidney and liver function

What is the clinical hallmark of malaria?  
Recurrent febrile paroxysm that corresponds to the hemolysis of infected erythrocytes

What are some complications of malaria if left untreated?  
Immune-mediated glomerulonephritis; Splenic enlargement or rupture; Hemolysis; Noncardiac pulmonary edema

What is cerebral malaria?  
Most common with infection from *Plasmodium falciparum*: coma, delirium, seizures, and somnolence with up to a 25% mortality

How is the diagnosis of malaria made?  
Giemsa-stained thick and thin blood smear—with the first smear being diagnostic in 90% of cases

What are two important questions to address when viewing a blood smear?  
1. Is *Plasmodium falciparum* responsible?  
2. Degree of parasitemia? (>3% is bad)

What is the drug of choice for the treatment of malaria due to any species aside from *Plasmodium falciparum*?  
Chloroquine

What drug is recommended for the dormant form of *Plasmodium vivax* and *Plasmodium ovale*?  
Primaquine

What can the use of primaquine in a patient with G6PD deficiency cause?  
Hemolytic anemia
### SOFT TISSUE INFECTIONS

#### Cellulitis

**What is the definition of cellulitis?**
Bacterial invasion of the skin that leads to a local soft tissue inflammatory reaction

**What groups does cellulitis more commonly occur in?**
Elderly; Immunocompromised patients; Diabetics; Peripheral vascular disease

**Name the two most common groups of bacteria that are involved with cellulitis.**
Streptococcus; Staphylococcus

**What organism is becoming more common as a cause of cellulitis, especially among team athletes, prison inmates, and military personnel?**
Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA)

**Name a common cause of cellulitis in children?**
*Haemophilus influenzae*

**What are some clinical features of cellulitis?**
Induration, pain, erythema, and warmth; PE: fever, leukocytosis, and lymphadenopathy as systemic involvement

**When is the use of soft-tissue radiography or ultrasound recommended?**
If a foreign body is involved as a cause

**What is *elephantiasis nostra***?
Recurrent attacks that can lead to dermal fibrosis, epidermal thickening, permanent swelling, and impairment of lymphatic drainage

**What are some treatment options for cellulitis in an otherwise healthy adult?**
Macrolide; Amoxicillin-clavulanate; Dicloxacillin

**What is an exception to outpatient treatment of simple cellulitis?**
Cellulitis of the head or neck where they should be admitted for IV antibiotics or immunocompromised patients with evidence of rapidly spreading cellulitis

**What is erysipelas?**
Superficial cellulitis with involvement of the lymphatic system
What organism is the most common cause of erysipelas?

Group A *Streptococcus*

What are some common ways that erysipelas occurs?

Ulcers; Infected dermatoses; Toe-web intertrigo

What are some clinical features of erysipelas?

Abrupt onset of symptoms that include high fever, chills, and nausea and with progression of the infection that leads to a shiny, red, and hot plaque; PE: bullae, purpura, and small areas of necrosis can be seen

What is a possible complication of erysipelas that should be considered?

Necrotizing fasciitis

What are some treatment options of erysipelas?

Penicillin G; Amoxicillin-clavulanate; Imipenem in severe cases; Marcolide for penicillin allergy

**Cutaneous Abscesses**

What are some factors that contribute to the skin’s protective function?

Lower pH of 3–5; Constant desquamation of epidermis; Skin continually shedding bacteria

Name some ways in which abscesses can develop.

Abrasions or lacerations; Puncture; Bites

How do abscesses typically start?

Local cellulitis

Name some organisms commonly involved with cutaneous abscesses?

Staphylococcal species; Streptococci; *Bacteroides*

What is a common site of abscesses when *Staphylococcus* species are involved?

Hair follicles

What is folliculitis?

Bacterial invasion of a hair follicle that causes inflammation

What is a deeper invasion of the soft tissue surrounding a hair follicle known as?

Furuncle (i.e., boil)

What is a carbuncle?

Several furuncles that coalesce to form a large area of infection that contains interconnecting sinus tracts

What is sufficient to treat most cases of folliculitis and boils?

Warm compresses

What are some clinical features of cutaneous abscesses?

Tenderness, erythema, and swelling with an area that may show induration and fluctuance

What are some clinical features that may
indicate systemic involvement?

What is typically done with cutaneous abscesses if it is fluctuant?

Are antibiotics commonly recommended along with I&D?

If the abscess is not fluctuant and an I&D cannot be done, what is recommended?

Of all the perirectal abscesses, which is the only one that can be drained safely in the ED?

When are antibiotics recommended in the case of cutaneous abscesses?

What is a particular concern in patients with underlying structural heart disease?

What are some high-risk cardiac conditions where prophylactic antibiotic coverage may be considered?

**GAS GANGRENE**

What agent is commonly implicated as a cause of gas gangrene?

Name two species of *Clostridium* that are identified as causing gas gangrene?

What is important to note about clostridial myonecrosis?

What is the primary pathophysiologic mechanism by which the *Clostridium* species cause myonecrosis?

In what environment does *Clostridium* species thrive?

Aside from direct inoculation from an open wound, name another route of entry?

lymphadenopathy

Incision and drainage (I&D)

I&D is sufficient in most cases

Treat with antibiotics as cellulitis

Perianal abscesses

Overlying cellulitis; Immunocompromised patients

Bacterial endocarditis

Prosthetic valves; Hypertrophic cardiomyopathy; History of bacterial endocarditis; Acquired valvular dysfunction

*Clostridium* species

1. *Clostridium perfringens* (80–90% of cases)
2. *Clostridium septicum*

It is a rapidly progressive and serious disease that threatens both life and limb and it is the deepest of the necrotizing soft tissue infections

Production of various exotoxin, α-toxin in particular, that causes a variety of problems such as tissue necrosis, cardiodepressant, and hemolysis

Anaerobic environments that can occur after injury

Hematogenous spread
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>In what group is hematogenous spread more common?</td>
<td>Immunocompromised patients</td>
</tr>
<tr>
<td>What is the incubation period of gas gangrene once inoculation occurs?</td>
<td>Around 3 days</td>
</tr>
<tr>
<td>What is the most common presentation of gas gangrene in the early stages?</td>
<td>Pain out of proportion to physical findings</td>
</tr>
<tr>
<td>What is the hallmark of gas gangrene?</td>
<td>Sepsis with gas production</td>
</tr>
<tr>
<td>What are some other clinical features of gas gangrene?</td>
<td>Low-grade fever, tachycardia, irritable, confused; PE: area may have edema with crepitance, brownish discoloration with a malodorous discharge</td>
</tr>
<tr>
<td>What can radiographic studies show in the case of gas gangrene?</td>
<td>Gas within the involved area</td>
</tr>
<tr>
<td>What are the four hallmarks for the treatment of gas gangrene:</td>
<td></td>
</tr>
<tr>
<td>Resuscitation</td>
<td>Aggressive fluid resuscitation; Avoid vasoconstrictors if possible</td>
</tr>
<tr>
<td>Surgical debridement</td>
<td>Mainstay for the treatment of gas gangrene; Early removal of the infected area is crucial; Debridement may range from fasciotomy to amputation</td>
</tr>
<tr>
<td>Antibiotic therapy</td>
<td>Includes penicillin; Ceftriaxone and macrolides as alternatives; Update tetanus status as indicated</td>
</tr>
<tr>
<td>Hyperbaric oxygen (HBO)</td>
<td>Initiated soon after debridement; Therapy consists of 100% oxygen at 3 atm of pressure for 90 minutes with three dives in the first 24 hours and 2 per day for 4–5 days</td>
</tr>
<tr>
<td>What is the most common cause of gas gangrene that is nonclostridial?</td>
<td>Mixed infections with both aerobic and anaerobic organisms</td>
</tr>
<tr>
<td>Does the presentation of nonclostridial gas gangrene differ much from one caused by clostridial species?</td>
<td>Not really</td>
</tr>
<tr>
<td>What are some species of bacteria involved with nonclostridial gas gangrene?</td>
<td>Enterococcus; Bacteroides; Bacillus; Staphylococcus</td>
</tr>
<tr>
<td>What are some treatment differences for nonclostridial gas gangrene when compared to clostridial gas gangrene?</td>
<td>Broad-spectrum antibiotic crucial; HBO still utilized</td>
</tr>
</tbody>
</table>
### Necrotizing Cellulitis/Fasciitis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is necrotizing cellulitis?</td>
<td>Superficial form of necrotizing soft tissue infection limited to the skin and subcutaneous fat</td>
</tr>
<tr>
<td>What are some conditions associated with necrotizing cellulitis?</td>
<td>Surgery; Trauma; Malignancy; Diabetes</td>
</tr>
<tr>
<td>What is the most common bacteria causing necrotizing cellulitis?</td>
<td>Clostridial species</td>
</tr>
<tr>
<td>What are some clinical features of necrotizing cellulitis?</td>
<td>Erythema and pain is the most common complaint; PE: may show blebs or vesicles</td>
</tr>
<tr>
<td>What are some key points in the management of necrotizing cellulitis?</td>
<td>Surgical debridement is crucial, but extensive soft tissue removal not needed; Broad-spectrum antibiotics</td>
</tr>
<tr>
<td>What is necrotizing fasciitis more commonly known as?</td>
<td>“Flesh-eating bacteria”</td>
</tr>
<tr>
<td>What is necrotizing fasciitis?</td>
<td>Widespread necrosis that commonly involves the fascia and subcutaneous tissue, but not underlying muscle as with myonecrosis</td>
</tr>
<tr>
<td>What are some major predisposing factors for necrotizing fasciitis?</td>
<td>Peripheral vascular disease; Diabetes; Intravenous drug use</td>
</tr>
<tr>
<td>What are two forms of necrotizing fasciitis?</td>
<td>1. One caused solely by group A streptococcus (GAS)</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Why does necrotizing fasciitis have the ability to spread so quickly?</td>
<td>Bacterial tissue toxins cause inflammation and thrombosis that leads to an environment favorable for bacterial growth and rapid spread along the fascial plane</td>
</tr>
<tr>
<td>What is the most common presenting complaint for patients with necrotizing fasciitis?</td>
<td>Pain out of proportion of the exam</td>
</tr>
<tr>
<td>What is indicated in all suspected cases of necrotizing fasciitis?</td>
<td>Early surgical consultation</td>
</tr>
<tr>
<td>What is the treatment of necrotizing fasciitis?</td>
<td>Similar to that of gas gangrene with focus on resuscitation, antibiotic use, surgical debridement, and HBO</td>
</tr>
</tbody>
</table>
What are some differences between GAS necrotizing fasciitis when compared to necrotizing fasciitis from mixed organisms?

While very similar in presentation and treatment, GAS necrotizing fasciitis tends to be more rapidly progressive with greater likelihood for bacteremia and TSS.

### TOXIC SHOCK SYNDROME

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the etiologic cause of toxic shock syndrome (TSS)?</td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>What are some risk groups of TSS?</td>
<td>Menstruating women; Postoperative staphylococcal wound; Persons who have undergone nasal surgery</td>
</tr>
<tr>
<td>What are some clinical features of TSS?</td>
<td>Sudden onset of fever, chills, vomiting, diarrhea, muscle aches and rash; Desquamation, particularly on the palms and soles can occur up to 2 weeks after onset</td>
</tr>
<tr>
<td>What is particularly worrisome about TSS?</td>
<td>Rapid progression to severe hypotension and multisystem dysfunction</td>
</tr>
<tr>
<td>What is the most crucial aspect in the management of TSS?</td>
<td>Aggressive management of circulatory shock</td>
</tr>
<tr>
<td>What are some key points in the management of TSS?</td>
<td>Identify and treat source of infection; Culture all sites; Remove all foreign bodies; Prompt antibiotic therapy</td>
</tr>
<tr>
<td>How is streptococcal toxic shock syndrome different from TSS caused by <em>S. aureus</em>?</td>
<td>More aggressive form of TSS that often develops in association with severe skin infection</td>
</tr>
<tr>
<td>What group of streptococcus is responsible for this form of TSS?</td>
<td>Group A</td>
</tr>
<tr>
<td>What are some clinical features of streptococcus TSS?</td>
<td>Similar to TSS caused by <em>S. aureus</em>, many will have signs of soft-tissue infection with pain</td>
</tr>
<tr>
<td>What are some key points in the management of TSS from streptococcus?</td>
<td>Aggressive exploration/debridement of soft-tissue infection; Early circulatory support; Prompt antibiotic therapy</td>
</tr>
</tbody>
</table>
OCCUPATIONAL POSTEXPOSURE PROPHYLAXIS

What are three infections that are commonly evaluated in an occupational postexposure such as needle sticks?

1. Human immunodeficiency virus (HIV)
2. Hepatitis B virus (HBV)
3. Hepatitis C virus (HCV)

Give some examples of potential infectious sources?

Contact with mucous membranes with infectious material; Percutaneous injury

Name some examples of potential infectious bodily fluids.

Blood; CSF; Semen; Amniotic fluid; Pleural fluid

What are some things to do when evaluating a patient who is exposed to potentially infectious material?

Obtain a thorough history that includes the circumstance, exposure type, etc.; Wash the wound with water and soap; Assess immune status of patient

Can HBV be transmitted by contact with environmental surfaces?

Yes—HBV can survive in dried blood

What is the risk of developing hepatitis if the blood source is HBsAg(+) and HBeAg(−)?

Less then 5%

What is the risk of developing hepatitis if the blood source is HBsAg(+) and HBeAg(+)?

About 25%

What are some factors to consider in the treatment of HBV?

HBV vaccination status of the patient; Immunity of the patient; HBV status of the source

What is the postexposure prophylaxis for HBV?

Hepatitis B immune globulin (HBIG); Vaccination series: Hepatitis B vaccine at the time of evaluation, at 1 month, and at 6 months

Is pregnancy a contraindication for HBV?

No

What is the risk of seroconversion from an HCV(+) source?

1–2%

What is the prophylaxis currently available for HCV?

None available

What is the probability of transmission of HIV from a single exposure in the following situations:

Vaginal intercourse 0.1–0.2%
Anal intercourse 0.1–4%

Percutaneous exposure 0.3%

What is a basic postexposure prophylaxis regiment for HIV exposure? Zidovudine; Lamivudine or Combivir

INFECTION DISEASE APPENDICES

Pregnancy Categories

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Generally acceptable. Controlled studies show no adverse effect to fetus</td>
</tr>
<tr>
<td>B</td>
<td>Use may be acceptable. Animal studies show no risk, but human studies not available</td>
</tr>
<tr>
<td>C</td>
<td>Use with caution only if the benefits outweigh the risk</td>
</tr>
<tr>
<td>D</td>
<td>Use only in life-threatening emergencies, possible risk to fetus</td>
</tr>
<tr>
<td>X</td>
<td>Do not use in pregnancy</td>
</tr>
<tr>
<td>NA</td>
<td>Information not available</td>
</tr>
</tbody>
</table>

Antibiotic Use in Pregnancy

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cephalosporins</td>
<td>Category B</td>
<td>generally safe</td>
</tr>
<tr>
<td>Penicillin</td>
<td>Category B</td>
<td>generally safe</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>Category B</td>
<td>generally safe</td>
</tr>
<tr>
<td>Quinolones</td>
<td>Category C</td>
<td>can cause arthropathy in the fetus</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>Category B</td>
<td>avoid in third trimester due to possible hemolytic anemia</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>Category D</td>
<td>can stain teeth and bone</td>
</tr>
<tr>
<td>Sulfonamides</td>
<td>Category B</td>
<td>in first and second trimester</td>
</tr>
<tr>
<td></td>
<td>Category D</td>
<td>can cause kernicterus</td>
</tr>
<tr>
<td>Clotrimazole</td>
<td>Category B</td>
<td>generally safe</td>
</tr>
</tbody>
</table>
CHAPTER 12

Pediatric Emergencies

HIGH-YIELD PEDIATRIC CHARTS

Vital Signs

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart Rate</th>
<th>Respirations</th>
<th>Systolic Blood Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>90–180</td>
<td>30–60</td>
<td>50–70</td>
</tr>
<tr>
<td>6 months</td>
<td>85–170</td>
<td>24–40</td>
<td>65–105</td>
</tr>
<tr>
<td>1 year</td>
<td>80–140</td>
<td>20–40</td>
<td>70–110</td>
</tr>
<tr>
<td>3 years</td>
<td>80–130</td>
<td>20–30</td>
<td>75–114</td>
</tr>
<tr>
<td>6 years</td>
<td>70–120</td>
<td>18–25</td>
<td>80–115</td>
</tr>
<tr>
<td>8 years</td>
<td>70–110</td>
<td>18–25</td>
<td>85–120</td>
</tr>
<tr>
<td>10 years</td>
<td>65–110</td>
<td>16–20</td>
<td>90–130</td>
</tr>
<tr>
<td>12 years</td>
<td>60–110</td>
<td>14–20</td>
<td>95–135</td>
</tr>
<tr>
<td>15 years</td>
<td>55–100</td>
<td>14–20</td>
<td>100–140</td>
</tr>
<tr>
<td>18 years</td>
<td>50–90</td>
<td>14–18</td>
<td>105–150</td>
</tr>
</tbody>
</table>

Temperature Conversion

<table>
<thead>
<tr>
<th></th>
<th>F</th>
<th>106</th>
<th>105</th>
<th>104</th>
<th>103</th>
<th>102</th>
<th>101</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>41.1</td>
<td>40.6</td>
<td>40</td>
<td>39.4</td>
<td>38.9</td>
<td>38.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>99</td>
<td>98.6</td>
<td>98</td>
<td>97</td>
<td>96</td>
<td>95</td>
</tr>
<tr>
<td>C</td>
<td>37.2</td>
<td>37</td>
<td>36.7</td>
<td>36.1</td>
<td>35.6</td>
<td>35</td>
<td></td>
</tr>
</tbody>
</table>
Name some important risk factors associated with cardiopulmonary arrest for each of the following categories:

**Fetal**
- Congenital infection; Acidosis;
- Prematurity or postmaturity; Thick meconium

**Maternal**
- Poor prenatal care; Illicit substance abuse; Premature rupture of membranes (PROM); Infections (i.e., HIV)

**Intrapartum**
- Placenta abruption/previa; Cord prolapse; Maternal shock; C-section

**What are some important things to know about pediatric intubations?**
- Pediatric intubation slightly differs from adult; Important to know the anatomic differences; Also know potential complications

---

### Seizures

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>0.1–0.3 mg/kg</td>
<td>IV/IO</td>
</tr>
<tr>
<td></td>
<td>0.5 mg/kg initially</td>
<td>Rectal</td>
</tr>
<tr>
<td></td>
<td>0.25 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>0.05–0.1 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td>Phenytobine</td>
<td>20 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>20 mg/kg</td>
<td>IV</td>
</tr>
</tbody>
</table>

IV, intravenous; IO, intraosseous.

### Rapid Sequence Intubation Protocol

<table>
<thead>
<tr>
<th>Premedicate:</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>&lt;10 kg 0.1 mg</td>
<td>IV/IO</td>
</tr>
<tr>
<td></td>
<td>&gt;10 kg 0.01 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1 mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sedation:</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etomidate</td>
<td>0.3 mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Paralysis:</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rocuronium</td>
<td>1 mg/kg</td>
</tr>
</tbody>
</table>
What is a Broselow tape?

Quick length-based reference for pediatric resuscitation that includes tube size and pediatric medication dosing.

What are some key things to know for each of the following anatomical variations:

Larynx

More anterior and superior than adults; Angle for intubation is more acute; Straight blade (Miller) is preferred; Infant tongue is larger relative to mouth size.

Trachea

Much shorter compared to adults; Intubation of right bronchus is more likely; Dislodgement of tube is more likely.

Cricoid ring

Narrowest region of the airway.

What is the formula used to calculate tracheal tube size for children?

Tracheal tube size = $4 + \frac{age}{4}$

What is the backup airway of choice in children less than 12 when intubation fails?

Percutaneous transtracheal ventilation.

What are some important points about percutaneous transtracheal ventilation?

Not a definite airway; Will progressively get hypercapnia; Typically useful for <1 hour.

What is the normal rate of breathing in each of the following age group:

- Neonates
  - About 50 breaths/minute
- Infants and children <8 years
  - 20 breaths/minute
- Children >12 years
  - 12 breaths/minute

What is an important point with regard to mechanical ventilation of children?

Make sure it is volume-limited and hyper-ventilation in the setting of acute herniation.

What are some key points in an infant who presents with complete airway obstruction due to a foreign body?

Avoid the Heimlich maneuver; Use back blows and chest compressions; Avoid blind finger sweeps.

What are the two preferred routes of vascular access?

Intravenous (IV) and intraosseous (IO).

What is the primary purpose of establishing IV/IO access in children?

Administration of medications; Fluid resuscitation; IO access is not usually effective for significant volume resuscitation.
What are some key points in vascular access?
The preferred site is the largest vein; Peripheral access should be attempted first; Central access can have significant complications.

What are some key points in IO access?
Typically performed if peripheral access fails; IO is easier and faster then central access; Anteromedial proximal tibia is the preferred site.

What is an important point about central access in children (typically <6 years)?
Should only be attempted if peripheral and IO access fails and by an experienced provider.

What is an alternative way to deliver medication?
Tracheal route.

What are some key points about tracheal medication administration?
Only useful for specific drugs; Typically use 2–3 × the amount of IV; Switch over to IV once available; Pharmokinetics are less reliable.

List commonly used medications that can be given though the tracheal route.
LEAN: Lidocaine, epinephrine, atropine, and naloxone.

What is a commonly used route for vascular access in newborns?
Cannulation of the umbilical artery.

Name some commonly used medications in pediatric resuscitation and their primary indications:

- **Epinephrine**
  - Used for primary arrests in children; Can be given IV/IO/tracheal route.

- **Atropine**
  - Not used in acute resuscitation; Can be used for increased vagal stimulation; Used as premedication prior to intubation.

- **Adenosine**
  - Used for supraventricular tachycardia (SVT) in pediatric patients.

- **Lidocaine**
  - Used to blunt ICP increase in intubation.

What are the two most common causes of cardiac arrest in children?
1. Respiratory arrest
2. Hypovolemic shock

What are two most common dysrhythmias in pediatric arrest?
1. Asystole
2. Bradyarrhythmias
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are the main categories of the APGAR score?</td>
<td>Activity (muscle movement)</td>
</tr>
<tr>
<td></td>
<td>Pulse</td>
</tr>
<tr>
<td></td>
<td>Grimace</td>
</tr>
<tr>
<td>What is the definition of the APGAR score?</td>
<td>It is a 0–10 point scale that is assigned to newborns at 1 and then 5 minutes that is used to evaluate the newborn and has prognostic functions as well</td>
</tr>
<tr>
<td>What are the key parameters to monitor in deciding if resuscitation should be done?</td>
<td>Respiratory status; Heart rate</td>
</tr>
<tr>
<td>List some important things to do during a neonatal resuscitation.</td>
<td>Suction airway of secretions; Maintain temperature; Supplemental oxygen; Cardiopulmonary resuscitation (CPR) (HR &lt;60 beats)</td>
</tr>
<tr>
<td>What is meconium?</td>
<td>Thick green substance that lines the intestines of the fetus and is not typically released as a bowel movement until the first few days of life</td>
</tr>
<tr>
<td>Is it possible for meconium to be released into amniotic fluid prior to delivery of fetus?</td>
<td>Yes—increased risk of aspiration</td>
</tr>
<tr>
<td>What is the feared complication of meconium aspiration?</td>
<td>Respiratory distress (esp. thick meconium); Meconium aspiration pneumonia (PNA)</td>
</tr>
<tr>
<td>In what circumstances can meconium be bad for the fetus/newborn?</td>
<td>Meconium found in amniotic fluid; Consistency of meconium is thick/green</td>
</tr>
<tr>
<td>What is the mortality rate if a newborn has aspirated thick meconium?</td>
<td>30–50%</td>
</tr>
<tr>
<td>What are some key points in the treatment of meconium staining?</td>
<td>Bulb suction mouth/nose during delivery; Meconium-stained fluid and respiratory depression = tracheal suctioning</td>
</tr>
<tr>
<td>What is esophageal atresia?</td>
<td>Birth defect where the esophagus is segmented and cut off</td>
</tr>
<tr>
<td>How common is esophageal atresia?</td>
<td>Occurs 1 in 4000 live births</td>
</tr>
</tbody>
</table>
What other congenital defect does esophageal atresia typically occur with? Tracheoesophageal fistula

What is the biggest risk factor for the development of esophageal atresia? Prematurity

What are some clinical features of esophageal atresia? Coughing/choking when feeding is attempted, recurrent aspiration PNA, and inability to pass a catheter into the stomach

What is the treatment for esophageal atresia? Surgical correction

What is the definition of necrotizing enterocolitis? Condition with varying degrees of intestinal necrosis most common in premature newborns with low birth weight

What are some risk factors associated with necrotizing enterocolitis? Prematurity; Infections; Hypertonic feeding solutions

When does necrotizing enterocolitis typically occur in the newborn? First 2 weeks of life

What is the most common physical finding in a newborn who presents with necrotizing enterocolitis? Abdominal distension with gastric retention

What are some other clinical features in a newborn with necrotizing enterocolitis? Bloody stools, bilious emesis, and abdominal wall redness and/or tenderness

What are some important clinical features of a newborn who may be septic? Bouts of apnea, temperature changes, lethargy, and metabolic acidosis

What are some complications of necrotizing enterocolitis? Necrosis of bowel; Perforation; Sepsis

What are some diagnostic tests to consider in a newborn with necrotizing enterocolitis? Plain films (classic finding is pneumatosis intestinalis); Cultures (stool, urine, blood, and CSF)

What are some key points in the management of a newborn with necrotizing enterocolitis? NPO and NG tube; IV fluids/Abx; Surgical consultation

What is an omphalocele? A defect in the umbilical wall with herniation of intestinal content covered in a peritoneal sac

What is important to know about omphalocele? Many of them are associated with other congenital defects

What is a gastroschisis? A defect in abdominal wall with herniation of intestinal content without peritoneal sac
What are some complications of omphalocele and gastroschisis?

Obstruction; Strangulation; Hypovolemia; Death

What are some key points in the management of omphalocele and gastroschisis?

NG tube for GI decompression; Do not attempt to reduce the mass; Cover in saline-soaked sterile gauze; IV fluids and Abx for prophylactic coverage; Surgical correction required

What is the definition of diaphragmatic herniation?

A congenital defect due to developmental failure of a portion of the diaphragm that allows herniation of stomach/intestines

Which are more common? Right-sided herniation or left-sided herniation?

Left-side far more common than right side

What is important to know about diaphragmatic herniation?

As with most congenital defects, there are typically other defects as well, with GI/GU abnormalities and congenital heart defects being fairly common

What are some clinical features of diaphragmatic herniation?

Symptoms referable to herniation of GI tract into the chest: emesis and respiratory distress as well as bowel sounds over the chest wall

What are some common radiographic findings in diaphragmatic herniation?

Displacement of mediastinal contents (heart); Loops of bowel in the chest; Lack of distinct diaphragmatic margin

What is an important consequence of diaphragmatic herniation on the lung?

Hypoplastic lung

What are some key points in the management of diaphragmatic herniation?

NG tube for GI decompression; NPO and IV fluids; Surgical correction

What are some different types of neonatal seizures?

Myoclonic; Tonic-clonic; Focal clonic

What is the most common cause of seizures in children?

Simple febrile seizure

What are some important things to know about febrile seizures?

Up to 5% of children are affected; Commonly occur between 3 months and 5 years

What are some clinical features of a febrile seizure?

Rapidly ascending fever; Generalized seizure less than 15 minutes duration; No focal neuro deficit

What is another important diagnosis to rule out in the setting of febrile seizure?

Meningitis
What important diagnostic test should be done for suspected meningitis? Lumbar puncture

What are some key points in the management of febrile seizures? Lower the fever; Treat the underlying cause of fever; Seizure prophylaxis is not recommended

What is another important seizure to consider in children? Generalized tonic-clonic seizure

What are some clinical features of a tonic-clonic seizure? Bilateral hemisphere involvement with motor involvement and alternations in consciousness

When should one consider use of a head CT? Focal neuro deficit; Signs of increased ICP; Suspected child abuse/head trauma

What are some key points in the management of tonic-clonic seizures? Most will terminate on their own; Benzodiazepines are the mainstay; Phenytoin and phenobarbital are second-line agents

What is the preferred benzodiazepine due to its long half-life and least effect on respiratory depression? Lorazepam

What are some important causes of neonatal seizures to consider? Hypoxia; Drug withdrawal; Electrolyte imbalance; Metabolic (i.e., hypoglycemia); CNS infections; Neoplasm

List the TORCHS infections. TOxoplasmosis Rubella Cytomegalovirus Herpes Syphilis

What are some key points in the management of neonatal seizures? Airway, breathing, circulation (ABCs); Correct easily reversible conditions (e.g., hypoxia); Monitor associated problems (e.g., acidosis); Anticonvulsant therapy

What is the drug of choice in the management of neonatal seizure? Phenobarbital

What are other classes of drugs used if phenobarbital fails? Benzodiazepine; Phenytoin

What are some reasons that benzodiazepine is not first-line treatment in neonates as opposed to adults? Profound respiratory depression; Displacement of bilirubin from albumin
## CONGENITAL HEART DISEASE

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>Is cyanosis ever normal in the newborn?</td>
<td>Yes, but only within the first (\frac{1}{2}) hour of life</td>
</tr>
<tr>
<td>What are the physical findings in a newborn with central cyanosis?</td>
<td>Bluish tongue, peripheral extremity, and mucous membrane</td>
</tr>
<tr>
<td>What is the amount of unsaturated Hgb in a newborn with central cyanosis?</td>
<td>Greater than 5 g</td>
</tr>
<tr>
<td>At what point is central cyanosis pathologic?</td>
<td>If it persists for greater than 30 minutes</td>
</tr>
<tr>
<td>What are some important causes of persistent central cyanosis in the newborn?</td>
<td>Primary lung disease; Cyanotic heart disease; Methemoglobinemia</td>
</tr>
<tr>
<td>What are the five “Ts” of cyanotic heart disease that result in central cyanosis due to right-to-left shunt?</td>
<td>Tetralogy of Fallot; Tricuspid atresia; Transposition of the great vessels; Truncus arteriosus; Total anomalous pulmonary venous return</td>
</tr>
<tr>
<td>What particular agent is important to maintain the patency of the ductus arteriosus in newborns with congenital heart defects such as transposition of the great vessels?</td>
<td>Prostaglandin E₃</td>
</tr>
<tr>
<td>What is a useful test to do to distinguish right-to-left shunts from other causes of central cyanosis such as sepsis?</td>
<td>Administer 100% oxygen and watch oxygen saturation, if it fails to improve, it points to a right-to-left shunt</td>
</tr>
<tr>
<td>What is the most common cyanotic congenital heart disease in children?</td>
<td>Tetralogy of Fallot</td>
</tr>
<tr>
<td>What are the four anatomical abnormalities in tetralogy of Fallot?</td>
<td>1. Pulmonary artery stenosis 2. Right ventricular hypertrophy 3. Ventricular septal defect (VSD) 4. Overriding aorta</td>
</tr>
<tr>
<td>What are some common findings for each diagnostic test used with tetralogy of Fallot:</td>
<td></td>
</tr>
<tr>
<td>ECG</td>
<td>Right ventricular hypertrophy; Right axis deviation</td>
</tr>
<tr>
<td>CXR</td>
<td>Decreased pulmonary vasculature; Boot-shaped heart</td>
</tr>
<tr>
<td>CBC</td>
<td>Compensatory polycythemia</td>
</tr>
</tbody>
</table>
What are two most common non-cyanotic congenital heart defects?

1. VSD
2. Aortic stenosis

What are some clinical features of aortic stenosis?

Typically not detected until later in life: congestive heart failure (CHF), chest pain (CP), and syncope

What are some complications of congenital aortic stenosis?

Sudden death (2° dysrhythmias); Endocarditis

What are some clinical features of VSD?

Determined by the size of VSD: ranges from asymptomatic to heart failure

What is the most common cause of CHF in neonates and children?

Congenital heart disease

What are some clinical features of CHF?

Rhonchi, rales, hepatomegaly, failure to thrive, and feeding difficulty

What are some other causes of CHF aside from congenital heart disease?

Sepsis; AV malformations; Severe anemia; Hypoplastic left heart syndrome; Infectious myocarditis

What are some key points in the management of CHF in neonates/children?

Search for and correct underlying cause; Supplemental oxygen; Use of digoxin and furosemide when needed

AIRWAY EMERGENCIES

Upper Airway

What is epiglottitis?

Epiglottitis is a life-threatening condition that occurs when the epiglottis—a small cartilage “lid” that covers the windpipe—swells, blocking the flow of air into the lungs

What is the most common cause of epiglottitis?

H. influenzae

What are some other causes of epiglottitis?

Burns from hot liquids; Direct trauma to throat; Various infections

What age group is epiglottitis most prevalent?

2–6 years of age

Is HIB epiglottitis common today?

No—since the introduction of HIB vaccine, it is not commonly seen. It is more common in immigrants and unvaccinated children

What are some clinical features of epiglottitis?

Typically will be ill-appearing, stridor and drooling with the child leaning forward is a classic picture
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What is the diagnostic test of choice in the evaluation of epiglottitis?</td>
<td>Lateral neck film—typically shows enlarged epiglottis</td>
</tr>
<tr>
<td>What are some key points in the management of evaluation of epiglottitis?</td>
<td>Ensuring intact airway is paramount; ENT should be consulted in severe cases; Low threshold for intubation; IV Abx (third generation cephalosporin common); Typically ICU admission for monitoring</td>
</tr>
<tr>
<td>What is croup?</td>
<td>Inflammation of the upper airway that leads to a cough that sounds like a seal bark, particularly when a child is crying</td>
</tr>
<tr>
<td>What is the most common cause of croup?</td>
<td>Viral (parainfluenza being most common)</td>
</tr>
<tr>
<td>What age group is croup most prevalent?</td>
<td>Around 2 years of age (in the fall-winter)</td>
</tr>
<tr>
<td>What are some clinical features of croup?</td>
<td>Bark-like cough worse at night is the hallmark, upper respiratory infection (URI) prodrome, stridor, and hoarseness with a low-grade fever</td>
</tr>
<tr>
<td>What role does a lateral neck film play?</td>
<td>To rule out epiglottitis (although rare)</td>
</tr>
<tr>
<td>What are some key points in the management of croup?</td>
<td>Typically resolves in a week; Abx not used—since viral most of the time; Cool mist and hydration; Steroids should be given to help resolve; Admit if refractory to tx (persistent stridor)</td>
</tr>
<tr>
<td>What role does racemic epinephrine aerosol play?</td>
<td>Used for children who have resting stridor and more severe respiratory distress</td>
</tr>
<tr>
<td>What is bacterial tracheitis?</td>
<td>Diffuse inflammatory process of the larynx, trachea, and bronchi with adherent or semiadherent mucopurulent membranes within the trachea</td>
</tr>
<tr>
<td>What are some clinical features of bacterial tracheitis?</td>
<td>Often will present as croup, but defining feature is that child will not respond to standard croup tx and will often get quite sick</td>
</tr>
<tr>
<td>What is the most common pathogen implicated in bacterial tracheitis?</td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>What are the key points in the management of bacterial tracheitis?</td>
<td>Ensure an intact airway; IV hydration and Abx directed against staph; ENT consult is usually recommended; Admit to ICU for monitoring</td>
</tr>
</tbody>
</table>
What is a retropharyngeal abscess?

Infection in one of the deep spaces of the neck with potential for airway compromise.

What are two ways in which the retropharyngeal space can become infected?

1. Direct inoculation via trauma
2. Spread from infection

What are some common infections that can lead to a retropharyngeal abscess?

URI, otitis, pharyngitis, and sinusitis

What age group is a retropharyngeal abscess most common?

6 months to 5 years of age

What are some complications of a retropharyngeal abscess?

Airway compromise the most important; Abscess rupture; Spread of infection (i.e., sepsis)

What are some common clinical features of a retropharyngeal abscess?

Typical picture is an ill-appearing child who is drooling and cannot tolerate PO and will often have a neck mass

What are some important diagnostic tests to consider in a retropharyngeal abscess?

Lateral neck film-retropharyngeal swelling; Make sure child is in inspiration during film; CXR-inspect for possible mediastinitis; CT is study of choice

What are some key points in the management of a retropharyngeal abscess?

ENT involvement for incision and drainage (I&D) of abscess; Abx; ICU monitoring in severe cases

What is the most common cause of accidental home death in young children?

Foreign body aspiration

What are some common clinical features in foreign body aspiration?

Stridor is common if the obstruction is higher; Respiratory wheezing if obstruction is lower; Suspect an impacted object in the airway if recurrent PNA

What is the most common location of foreign bodies?

Right mainstem bronchus

What are some common diagnostic tests used in foreign body aspiration?

CXR and MRI can be used to evaluate; Bronchoscopy is diagnostic and therapeutic

Lower Airway

What is bronchiolitis?

Acute infectious disease of the lower respiratory tract

What is the pathophysiology of bronchiolitis?

Narrowing of the bronchi/bronchioles typically due to inflammation of epithelial cells
What is the most common cause of bronchiolitis?
Viral-RSV most common

What is a common history element in most patients who present with bronchiolitis?
Sick contact or at day care

What age group is most commonly affected with bronchiolitis?
Infants of 2 months to 2 years

What are some clinical features of bronchiolitis?
Typically URI-like symptoms before progression to lower respiratory tract symptoms of wheezing, SOB, and possible cyanosis

What are common diagnostic findings in a CXR?
Patchy atelectasis; Hyperinflation of lungs; Air trapping

What airway disease do many children with bronchiolitis later develop?
Asthma

What are some key points in the management of bronchiolitis?
Supportive care; Ensure proper hydration; Abx not indicated—viral infection; A trial of bronchodilators may be warranted

What are some indications for admission for bronchiolitis?
Respiratory distress, extreme tachypnea; Hypoxia; Inability to take PO; Poor home care

What is the most common chronic disease of the pediatric population?
Asthma

What are some important points about asthma?
The prevalence is rising in the United States; Mortality from asthma is also rising; Accounts for large amount of ED visits

What is the definition of asthma?
It is a chronic inflammatory disorder characterized by increased responsiveness to a variety of stimuli that results in reversible airway constriction/obstruction

List important triggers for asthma exacerbation.
Any upper respiratory infection; Inhaled irritants (i.e., smoke); Medication; GERD; Cold environment; Exercise

What is the pathophysiology for asthma exacerbation?
Triggers that result in an IgE-mediated response that leads to inflammation and bronchial smooth muscle contraction, this eventually results in airway edema and movement of inflammatory cells. The end result is increased airway resistance
What are some clinical features of asthma exacerbation?
Respiratory distress, increased work of breathing, tachypnea, tachycardia, and in some, only a chronic cough.

Is it reassuring if no wheezes can be heard on exam of an asthmatic with exacerbation?
No—may represent total cessation of airflow.

What is PEFR?
Peak expiratory flow rate—typically measured before and after treatment to assess effectiveness.

What are some important points for each of the following categories of exacerbation:

- **Mild exacerbation**
  - Oxygen saturation above 95% on room air; PEFR >80%; Mild wheezing on exam; Able to speak in full sentences.

- **Moderate exacerbation**
  - Oxygen saturation in low 90s; PEFR 50–80%; Wheezing via expiratory phase; Difficulty in speaking.

- **Severe exacerbation**
  - Oxygen saturation <90% in room air; PEFR <50%; Typically using accessory muscles; Can only speak one or two words at a time.

What are risk factors associated with poor outcome in asthma exacerbation?
Prior intubation or ICU admission; Greater than three hospitalizations per year; Use or cessation of oral steroids; Significant comorbid disease (CAD); Lower socioeconomic status.

What are some key points in the management of asthma exacerbation?
ABCs—particular with O₂ administration; B₂-agonist is the mainstay treatment; Anticholinergic used in severe cases; Steroids.

What are some important points for each of the following used medications in asthma:

- **B₂-agonist**
The mainstay treatment: nebulizer or inhaler; Primary effect is on small airway; Albuterol most commonly used; IV use only in very sick patients.

- **Anticholinergic**
Ipratropium most commonly used agent; Primary effect is on large airways; Added to B₂-agonist in more severe cases; Atropine not used due to side effect profile.
### Steroids
- Shown to prevent progression and relapse; IV and oral equally effective; Should be continued on steroids once d/c

### Leukotriene modifiers
- Inflammatory mediators used in outpatient; No role in acute management of asthma

### Magnesium sulfate
- Has bronchodilator properties; Used in acute exacerbations as second-line treatment; Not particularly effective in mild exacerbations

### Ketamine
- Induction agent with mild bronchodilator effects; Recommended if intubation is to be done

### Heliox
- Mixture of helium-oxygen (80:20); Helps decrease work of breathing; May help in severe exacerbations

### What are some considerations for admission in a patient with asthma exacerbation?
- Failure to improve after treatment in the ED; Poor home care; History of ICU/intubation for asthma

### What are the general guidelines to safely discharge a patient from the ED?
- Good patient follow-up; 3–4 hours is usually enough to show improvement with medication

---

**PEDIATRIC GASTROINTESTINAL**

### Appendicitis

#### What are some clinical features of appendicitis?
- Typically have diffuse periumbilical pain that eventually leads to N/V and RLQ pain. Will often have a low-grade temperature as well

#### What are some commonly used diagnostic tests in appendicitis?
- Kidney-ureter-bladder (KUB): rarely shows a fecalith; CT with contrast: test of choice; U/S: operator-dependent

#### What are some findings in a child with perforation of the appendix?
- High-grade fever, high WBC, and symptoms over 2 days as well as diffuse abdominal pain and peritoneal signs

#### What are some key points in the management of appendicitis?
- IV fluids and NPO; Broad-spectrum Abx prior to surgery; Surgical consult
## Pyloric Stenosis

<table>
<thead>
<tr>
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<th>Answer</th>
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<tbody>
<tr>
<td>What is the definition of pyloric stenosis?</td>
<td>It is hypertrophy of the pylorus with gastric outlet obstruction.</td>
</tr>
<tr>
<td>What age group is commonly affected with pyloric stenosis?</td>
<td>Male newborns between 2–4 weeks.</td>
</tr>
<tr>
<td>What are some common clinical features of pyloric stenosis?</td>
<td>Nonbilious projectile vomiting is the hallmark with failure to thrive and sometimes a palpable right upper quadrant (RUQ) mass can be felt.</td>
</tr>
<tr>
<td>What are some tests used to diagnose pyloric stenosis?</td>
<td>U/S and upper GI series.</td>
</tr>
<tr>
<td>What are some key points in the management of pyloric stenosis?</td>
<td>NPO and IV fluids; Prompt surgical correction.</td>
</tr>
</tbody>
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## Incarcerated Hernia

<table>
<thead>
<tr>
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<th>Answer</th>
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<tbody>
<tr>
<td>What age group do incarcerated hernias typically occur in?</td>
<td>Under 1 year of age.</td>
</tr>
<tr>
<td>What are some clinical features of incarcerated hernias?</td>
<td>Emesis with a palpable scrotal/inguinal mass.</td>
</tr>
<tr>
<td>What other conditions are in the differential diagnosis for incarcerated hernia?</td>
<td>Hydrocele; Torsion of testicles; Undescended testis.</td>
</tr>
<tr>
<td>What are some key points in the management of incarcerated hernias?</td>
<td>Manual reduction, then outpatient surgery; If any evidence of ischemia = immediate surgical reduction.</td>
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## Intestinal Obstruction

<table>
<thead>
<tr>
<th>Question</th>
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<tbody>
<tr>
<td>What is the clinical hallmark of intestinal obstruction?</td>
<td>Emesis with abdominal pain and distension.</td>
</tr>
<tr>
<td>What are some important causes of intestinal obstruction?</td>
<td>Hernias; Intussusceptions; Congenital atresia.</td>
</tr>
<tr>
<td>What are some common findings on abdominal plain films?</td>
<td>Dilated loops of bowel with air-fluid levels.</td>
</tr>
<tr>
<td>What are some key points in the management of intestinal obstruction?</td>
<td>NPO, IV fluids, and NG tube; Surgical intervention required.</td>
</tr>
</tbody>
</table>
## Intussusception

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What are some important things to know about intussusception?</td>
<td>Number 1 common cause of obstruction in children; Most common age group: 3 months to 5 years; Ileocolic intussusception most common; More common in males</td>
</tr>
<tr>
<td>What are some clinical features of intussusception?</td>
<td>Emesis, colicky pain, and red jelly stools as well as possible mental status change. PE: may palpate a sausage-shaped mass</td>
</tr>
<tr>
<td>What is the most prominent feature of abdominal pain in intussusception?</td>
<td>Periods of intense abdominal pain followed by periods of no pain</td>
</tr>
<tr>
<td>What are some important points for each of the following diagnostic tests:</td>
<td></td>
</tr>
<tr>
<td>Abdominal plain films</td>
<td>May show abdominal mass in RUQ; May show dilated bowel with air-fluid levels; Free air in perforation</td>
</tr>
<tr>
<td>Barium enema/air-contrast</td>
<td>It the test of choice to detect intussusception; Therapeutic: reduces in most cases; BE may show coiled-spring appearance</td>
</tr>
<tr>
<td>What is the next step to be taken if BE or air-contrast fails to reduce the intussusception?</td>
<td>Surgical intervention</td>
</tr>
<tr>
<td>What is the recurrence rate after a successful BE or surgical reduction?</td>
<td>As high as 10% in the first 24 hours</td>
</tr>
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## Meckel’s Diverticulum

<table>
<thead>
<tr>
<th>Question</th>
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<tbody>
<tr>
<td>What is the definition of Meckel’s diverticulum?</td>
<td>A Meckel’s diverticulum is a remnant of structures within the fetal digestive tract that were not fully reabsorbed before birth and leads to a pouch with GI tissue</td>
</tr>
<tr>
<td>What remnant of tissue from the prenatal development of the digestive system is found in Meckel’s diverticulum?</td>
<td>Gastric tissue most common</td>
</tr>
<tr>
<td>What is the “rule of 2’s” in Meckel’s diverticulum?</td>
<td>Peak age of symptoms is 2 years of age; Affects 2% of the population; 2 inches in length; Two times more likely in males</td>
</tr>
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<td>Question</td>
<td>Answer</td>
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<td>--------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>What are some clinical features of Meckel’s diverticulum?</td>
<td>Painless bleeding from rectum, N/V, and sign of obstruction if a volvulus develops</td>
</tr>
<tr>
<td>What are some other considerations in an infant with painless bleeding?</td>
<td>Anal fissures; Juvenile polyps; Infection</td>
</tr>
</tbody>
</table>
| What are three complications of Meckel’s diverticulum?                  | 1. Inflammation that mimics appendicitis  
2. Bleeding—can be massive  
3. Obstruction—volvulus or intussusception                                                                                           |
| What is the diagnostic study of choice for Meckel’s diverticulum?       | Meckel’s isotope scanning                                                                                                                                                                       |
| What are some key points in the management of Meckel’s diverticulum?    | Remove if heavy bleeding or pain; Surgical intervention if sign of obstruction                                                           |

**Volvulus**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What is the definition of a volvulus?</td>
<td>A form of obstruction typically due to malrotation of the bowel during embryonic development</td>
</tr>
<tr>
<td>What age group is more commonly affected with a volvulus?</td>
<td>Greater than 90% present &lt;1 year of age</td>
</tr>
<tr>
<td>What are some clinical features of a volvulus?</td>
<td>Failure to thrive, anorexia, intermittent apnea, emesis (bilious) with abdominal distension</td>
</tr>
<tr>
<td>What is a feared complication of a volvulus if not promptly treated?</td>
<td>Gangrene with perforation</td>
</tr>
<tr>
<td>What are some key points in the management of a volvulus?</td>
<td>NPO, IV fluids, and NG tube; Surgical intervention</td>
</tr>
</tbody>
</table>

**Obstructive series**

- Gastric/duodenal distension (double bubble); Relative paucity of lower GI gas

**Upper GI contrast series**

- Study test of choice; “Bird-beak” obstruction at proximal duodenum

**Ultrasonography and CT scanning**

- Use is more as adjunctive tests; Definitive diagnosis rests on upper gastrointestinal (UGI) study

**What are some important points for each of the following diagnostic tests:**

- NPO, IV fluids, and NG tube; Surgical intervention
## INFECTIOUS DISEASE

### Bacteremia and Sepsis

- **What is the pathophysiology of fever?**
  - Typically due to exogenous substance (antigens/bacterial wall components) that result in the release of pyrogens that in turn result in PG production, this acts on the hypothalamus to raise the hypothalamic set point.

- **What are some common manifestations of a raised hypothalamic set point?**
  - Chills, shivering, peripheral vasoconstriction, and behavioral activities (using blankets) that result in elevation of body temperature.

- **What area of the hypothalamus regulates body temperature?**
  - Ventromedial preoptic area; Periventricular nucleus.

- **What are some common methods to measure temperature?**
  - Oral; Axillary; Rectal; Tympanic.

- **What method is the most accurate and thus should be used whenever possible?**
  - Rectal.

- **What are some risks for serious bacterial infection that may not be obvious in the pediatric population?**
  - Infants: rectal temp (>38°C) and leukocytosis; Neonates with hypothermia (<36°C); Fever with a low white count (<5k); Fever with a petechial rash.

- **What are some commonly used drugs to treat fevers?**
  - Ibuprofen; Acetaminophen.

- **What role does aspirin play in the treatment of fever from viral illnesses?**
  - Should be avoided due to association with Reye’s syndrome (it is effective and used commonly in some parts of the world).

- **What is Reye’s syndrome?**
  - It affects all organs of the body but is most harmful to the brain and the liver, causing an acute increase of pressure within the brain and, often, massive accumulations of fat in the liver and other organs.

- **What is the most common preceding factor?**
  - Viral illness (i.e., chicken pox).

- **What are some clinical features of Reye’s syndrome?**
  - Recurrent vomiting, listlessness, personality changes such as irritability or combativeness, disorientation or confusion, delirium, convulsions, and loss of consciousness.
<table>
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<tr>
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<th>Answer</th>
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<tbody>
<tr>
<td>What other conditions is Reye’s syndrome commonly mistaken for?</td>
<td>Meningitis, diabetes, drug overdose poisoning, and encephalitis</td>
</tr>
<tr>
<td>What is the most common cause of mortality in Reye’s syndrome?</td>
<td>Brain herniation from swelling</td>
</tr>
<tr>
<td>What is the treatment for Reye’s syndrome?</td>
<td>Treatment is primarily supportive with care focusing on reducing brain swelling</td>
</tr>
<tr>
<td>What is occult bacteremia?</td>
<td>It is fever with positive blood cultures in a child who does not have a major source of infection</td>
</tr>
<tr>
<td>What are the three most common organisms responsible for occult bacteremia?</td>
<td>1. <em>S. pneumoniae</em>—by far the most common 2. <em>N. meningitidis</em> 3. Salmonella species</td>
</tr>
<tr>
<td>What age group is most susceptible to infection?</td>
<td>Between 6 months and 2 years of age</td>
</tr>
<tr>
<td>What is the reason for this?</td>
<td>Infants &lt;6 months typically have maternal antibodies which decrease leaving infants more susceptible till the age of 2, when they eventually develop their own</td>
</tr>
<tr>
<td>What is the definition of sepsis?</td>
<td>It occurs when bacteria, which can originate in a child’s lungs, intestines, urinary tract, or gallbladder, make toxins that cause the body’s immune system to produce various cytokines that act on many targets in the body</td>
</tr>
<tr>
<td>What are the three most common organisms responsible for sepsis in the following age group:</td>
<td></td>
</tr>
<tr>
<td>Neonates</td>
<td><em>Group B Streptococcus; Listeria monocytogenes; E. coli</em></td>
</tr>
<tr>
<td>Infants</td>
<td><em>S. pneumoniae; H. influenzae; N. meningitidis</em></td>
</tr>
<tr>
<td>What are some clinical features of an infant who is septic?</td>
<td>Ill-appearing, lethargic, periods of apnea and bradycardia, failure to thrive, and often hypothermic (&lt;36ºC)</td>
</tr>
<tr>
<td>What is the standard workup for neonates/infants who may be septic?</td>
<td>CBC, blood cultures, U/A with urine cultures, stool cultures, CXR, and LP</td>
</tr>
</tbody>
</table>
### Meningitis

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of meningitis?</td>
<td>It is a serious CNS infection of the meninges with often devastating results in infants and young children if not treated early</td>
</tr>
<tr>
<td>What are two common sources of infections in meningitis?</td>
<td>1. Hematogenous spread—most common</td>
</tr>
<tr>
<td></td>
<td>2. Direct spread from a contiguous focus</td>
</tr>
<tr>
<td>Why is the diagnosis of meningitis more elusive in infants?</td>
<td>The classic signs/symptoms (stiff neck/HA/fever) are often not present</td>
</tr>
<tr>
<td>What are some clinical features of meningitis in infants (&lt;4 months)?</td>
<td>Lethargy, decreased oral intake, irritability, fever or hypothermia, seizure, and bulging fontanelle</td>
</tr>
<tr>
<td>What are the three most common organisms responsible for meningitis in the following age group:</td>
<td>Group B streptococcus; L. monocytogenes; E. coli</td>
</tr>
<tr>
<td>Neonates</td>
<td></td>
</tr>
<tr>
<td>Infants/young children</td>
<td>S. pneumoniae; H. influenzae; N. meningitidis</td>
</tr>
<tr>
<td>What are other important causes of meningitis to consider aside from bacteria?</td>
<td>Viral; Fungal; TB; Aseptic</td>
</tr>
<tr>
<td>What are some key points in the management of meningitis?</td>
<td>IV broad-spectrum Abx without delay; LP to diagnose and tailor Abx therapy; Antiviral tx if suspicious of herpes</td>
</tr>
<tr>
<td>What role do steroids play in the treatment of meningitis?</td>
<td>They may play a role in reducing neurologic sequelae if given early</td>
</tr>
</tbody>
</table>

### Otitis Media

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of otitis media?</td>
<td>Infection of the middle ear with acute onset, possible presence of middle ear effusion, and signs of middle ear inflammation</td>
</tr>
<tr>
<td>What is the pathophysiology of otitis media?</td>
<td>Obstruction of the eustachian tube that result in a sterile effusion with aspiration of nasopharyngeal secretions into the middle ear that result in acute infection</td>
</tr>
</tbody>
</table>
Why do otitis media occur more frequently in children? Infants and younger children have shorter and more horizontal eustachian tube then adults


What are some clinical features of otitis media? Exam of ear often show distortion of tympanic membrane (TM), erythema, decreased mobility of TM on pneumatic otoscopy, fever, poor feeding, and child pulling at ear

What are some complications to consider in otitis media if left untreated? Hearing loss, TM perforation, mastoiditis, lateral sinus thrombosis, and meningitis

What are the main Abx used to treat otitis media? Amoxicillin is the mainstay followed by TMP-SMX or macrolide as second-line treatment

When should the fever and symptoms begin to subside? Within a few days after Abx is started

**Pneumonia**

What age group is most commonly affected with pneumonia (PNA)? Incidence is greatest in 6–12 months of age

What is the primary mode in which PNA occurs? Typically from aspiration of infectious particles, such as from a preceding URI

What are some important elements in the history of a child with PNA? Comorbid conditions; Age; Sick contact (i.e., day care); Immunizations

What is the most common cause of PNA in children (not neonates)? Viruses—RSV being most common

What are some common bacterial pathogens that cause PNA in infants/children? Mycoplasma; *S. pneumoniae*; *C. trachomatis*; *H. influenzae*

What are some clinical features of PNA in infants/children? Often will have a preceding URI, cough, fever, and tachypnea are common

What are some important diagnostic studies to consider in PNA? Pulse ox (hypoxia), CBC and blood cultures are often ordered, CXR, and sputum stain

What is the more likely cause of PNA in which the CXR shows diffuse interstitial pattern? Viral; Chlamydial; Mycoplasma
What is the more likely cause of PNA in which the CXR shows lobar involvement? Bacterial

What are some key points in the management of PNA? Bacterial PNA require specific Abx coverage; Viral PNA typically require supportive care; Persistent PNA = possible foreign body aspiration in children

What are indications for admission in an infant/child who presents with PNA? Respiratory distress; Ill-appearing; PNA complications (i.e., empyema); Hypoxia; Outpatient Abx failure; Social reasons (poor care at home)

## Pertussis

What is the causative agent of pertussis (whooping cough)? *Bordetella pertussis*

What are some important things to know about pertussis? Highly infectious (via respiratory droplets); Incubation time is about 10 days; Mortality is highest in first few months

What group is commonly affected by pertussis? Nonimmunized children

Can adults who received vaccination against pertussis still develop it later in life? Yes—does not confer life-long immunity

What is the three-stage illness of pertussis:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catarrhal</td>
<td>URI prodrome that last for about 2 weeks; Highly infectious at this stage</td>
</tr>
<tr>
<td>Paroxysmal</td>
<td>Paroxysmal coughing spells; Emesis is common with the coughing; Can last up to 1 month</td>
</tr>
<tr>
<td>Convalescent</td>
<td>Residual cough that can last for months</td>
</tr>
</tbody>
</table>

What is the characteristic finding on CBC in a patient with pertussis? WBC that can be as high as 50k; Lymphocytosis is common

What are some commonly used tests to diagnose pertussis? Bordet-Gengou medium; PCR; Antibody staining

What is the Abx of choice to treat pertussis? Erythromycin (can also be given to close contacts of patients with pertussis)
What is the typical pertussis vaccine regiment? Before age 7, children should get five doses of the DTaP vaccine; These are usually given at 2, 4, 6, and 15–18 months of age and 4–6 years of age.

What are some complications of pertussis? PNA; Seizure; Brain death from hypoxia.

**Urinary Tract Infection**

**What are some important things to know about UTI in infants/children?** They are fairly common in the pediatric population; More common in males during infancy; Infants/children have few specific symptoms.

**What is the mechanism of UTI in infants/children?** Ascending infection from perineal contaminants is common, but hematogenous spread is more common in neonates.

**What is an important consideration in infants less than 1 year of age who have recurrent UTIs?** Structural problem in the GU tract; Vesicoureteral reflux.

**How common is urosepsis in infants 1–3 months in age?** 30%

**What are some clinical features of neonates with UTIs?** Irritability, emesis, diarrhea, poor oral intake, and possible septic (as children get older, their sx become more specific for UTI-dysuria and frequency).

**What is the most common pathogen in UTIs in this age group?** *E. coli*

**What are some possible causes of UTIs in male children?** Meatal stenosis; Phimosis; Paraphimosis.

**What are some complications of UTIs?** Pyelonephritis; Urosepsis; Renal scarring; Renal failure.

**What are three optimal ways to collect urine for a U/A?**
1. Midstream collection
2. Suprapubic aspiration
3. Bladder catheterization

**What are the typical U/A findings that suggest UTI?**
Pyuria: >10 WBCs/HPF; Bacteriuria: >100k CFU/mL

**What is another diagnostic test that should be obtained for females <3 years and males <1 year?** Urine culture

**What are the indications for Abx use?** Symptomatic with pyuria/bacteriuria; Any evidence of pyelonephritis.
What are commonly used Abx in the treatment of UTIs? | Trimethoprim-sulfamethoxazole (TMP/SMX); Amoxicillin; Third generation cephalosporins
---|---
What are commonly used radiographic studies to further evaluate UTIs? | Renal ultrasound; Voiding cystourethrography; IVP

## CHILD ABUSE

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answers</th>
</tr>
</thead>
</table>
| What are three common types of abuse in children? | 1. Sexual abuse  
2. Neglect  
3. Physical abuse |
| How common is sexual abuse in children? | Upto 25% of all females sexually abused; Upto 10% of all males sexually abused |
| In what percentage of sexual abuses is the perpetrator known to the victim? | Upto 90% (most often family/relatives) |
| What are some findings on physical exam that is suggestive of sexual abuse? | Vaginal discharge; Sexually transmitted disease; Scarring/tearing of the hymen; Anal fissures |
| Do sexually abused children always show evidence of abuse on exam? | No—up to 50% may present normally |
| What are important laboratory tests to conduct in a child who is sexually abused? | Culture for gonorrhea and chlamydia; Syphilis; HIV testing |
| Are health-care providers required to report sexual abuse? | Yes |
| What are some physical findings in a child who is suffering from neglect? | Poor hygiene, evidence of failure to thrive such as low weight for age, alopecia, and avoidance |
| What is an important consideration in a child who is suffering from neglect? | Suspect physical abuse |
| What are some important things to do if a child is suffering from neglect? | A skeletal survey for abuse; Report to the proper agencies; Child is typically admitted |
| What does the skeletal survey usually consist of? | AP and lateral views of skull/chest/pelvis/spine, and extremities |
| What are the most common causes of death in children who are physically abused? | Head and abdominal injury |
| What are red flags in a child’s history that should raise the suspicion of physical abuse? | Inconsistent history from caregivers; History that does not match PE; Pattern injuries such as choke marks; Bruises in certain areas like buttocks |
What are common injury patterns associated with physical abuse?
Posterior rib fractures; Cigarette burns; Skull fractures; Healing fractures that were not treated; Spiral fractures of extremities

What is shaken baby syndrome?
Type of inflicted traumatic brain injury that happens when a baby is violently shaken

What are some reasons why a baby is more susceptible to being shaken?
Weak neck; Proportionally larger head

What are the characteristic injuries that occur in shaken baby syndrome?
Subdural hematoma; Retinal hemorrhages/detachment; Spinal neck/cord damage; Fracture of ribs and bones

What are some clinical features of shaken baby syndrome?
Extreme irritability, lethargy, poor feeding, breathing problems, convulsions, vomiting, and pale or bluish skin

What age group is shaken baby syndrome most common in?
Typically infants

What are some important diagnostic tests to consider in suspected physical abuse?
CBC/coags—to assess for coagulopathy; Skeletal survey; Imaging studies such as CT or MRI

What is essential to do in all cases of suspected physical abuse?
Report to police and proper agencies; Must not allow child to go back home

CLINICAL VIGNETTES

A newborn is noticed to be apneic and choking whenever feeding is attempted for the past week, the newborn’s history is only significant for prematurity
Esophageal atresia

A 1-week-old newborn who was born premature is brought in the ER due to concerns of recent abdominal distension with bilious emesis; PE: abdominal tenderness
Necrotizing enterocolitis

A 5-week old is brought in due to periods of breathing difficulty as well as bouts of emesis for about a month; PE: remarkable for bowel sounds heard over the left anterior chest
Diaphragmatic herniation
A 1-year-old is brought in by his frantic mother due to a sudden onset of a generalized seizure that occurred about an hour ago; PE: low-grade temperature, but otherwise unremarkable PE

A 3-year-old ill-appearing female is brought in by her mother for high-fever, history is significant for recent immigration to the United States from China; PE: ill-appearing child leaning forward with drooling and stridor

A 4-year-old child presents with low-grade fever, HA, and decreased oral intake; PE: erythema and decreased motility of right tympanic membrane

A 2-year-old male is brought in with a 1-week history of a URI, now presents with a bark-like cough particularly worse at night; PE: child otherwise appears well despite the cough

An alarmed father brings in his 2-year-old son due to a sudden onset of wheezing, but is otherwise well; PE: unremarkable

An 8-year-old child with a long history of allergies is brought in by her mother due to difficulty in breathing soon after soccer practice; PE: bilateral wheezing

A 3-year-old female is brought in by her concerned mother who mentions that her child has intense periods of colicky abdominal pain with periods of no pain as well as red jelly stools

A 3-year-old female presents with a 2 day history of nausea, emesis, fever, and irritability; PE: diffuse abdominal pain; Labs: elevated WBC

A 3-year-old ill-appearing male with a recent history of sinusitis now presents with a high-grade fever and the inability to swallow; PE: Child is drooling and a small mass can be felt on the neck, lateral neck film: retropharyngeal swelling

A 3-week-old male is brought in by her mother with concerns of ability to keep...
any nutrition down, she mentions whenever the patient eats, he soon has projectile vomiting; PE: a nontender RUQ mass can be felt

A 2-year-old male is brought in with a 2-day history of abdominal pain and distension with the inability to tolerate any feedings; abdominal films: dilated loops of bowel with air-fluid levels

Intestinal obstruction

A 2-year-old male presents with painless bleeding with nausea and vomiting, but otherwise has no other medical problems; PE: unremarkable

Meckel’s diverticulum

A 3-month-old child is brought in by her mother with lethargy, irritability, fever, and decreased oral intake that has been ongoing for about 2 days; PE: bulging fontanelle

Meningitis

A 5-year-old male is brought in by her mother for a fall from his bed last night, his medical history is significant for three other fractures to various other areas of the body; PE: fracture of the left clavicle

Child abuse
# Normal Pregnancy

What are some physiologic changes that occur to each of the following system during normal pregnancy:

<table>
<thead>
<tr>
<th>System</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>Increase in tidal volume, minute ventilation, O₂ consumption, and respiratory rate along with a decrease in total lung capacity</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Increase in circulating volume, heart rate (HR), and cardiac output (CO) with a 20% decrease in BP during first trimester</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Gastroesophageal reflux disease (GERD) very common, cholestasis, hemorrhoids, and nausea/vomiting</td>
</tr>
<tr>
<td>Genitourinary</td>
<td>Increase in renal blood flow, glomerular filtration rate (GFR), kidney size, and urinary stasis; decrease in BUN/Crea</td>
</tr>
<tr>
<td>Hematology</td>
<td>Increase in plasma volume, decrease in hematocrit (Hct), decrease in White blood cell (WBC) counts, and increase in coagulation factors</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Increase in glucose level, progesterone, estrogen, T₃/T₄ (euthyroid), thyroid-binding globulin, and prolactin</td>
</tr>
</tbody>
</table>
What are some important points to know about human chorionic gonadotropin (hCG)?

- Detected as early as 9 days after fertilization; Doubles every 2 days early in pregnancy; Very low false negative rate (<1%); Peaks at about 10 weeks gestational age.

What are some conditions that can result in a positive pregnancy test?

- Intrauterine pregnancy; Ectopic pregnancy; Recent abortion; Trophoblastic disease; Germ cell tumors.

What are some common causes of very high levels of beta human chorionic gonadotrophin (β-hCG)?

- Multiple gestations; Advanced age; Ovarian cancer; Trophoblastic disease; Germ cell tumors.

## VAGINAL BLEEDING IN REPRODUCTIVE WOMEN (NONPREGNANT)

**Define the following types of vaginal bleeding?**

- Abnormal bleeding: Vaginal bleeding outside one’s regular cycle
- Dysfunctional uterine bleeding (DUB): Abnormal vaginal bleeding due to anovulation
- Menorrhagia: Excessive bleeding or cycles >7 days
- Metrorrhagia: Irregular vaginal bleeding
- Menometrorrhagia: Excessive irregular bleeding

**What are some important elements to gather in the history of anyone who presents with vaginal bleeding?**

- Menstrual history; Last menstrual period (LMP); Age of menarche; Any pattern of abnormal bleeding; Vaginal discharge; If they are pregnant (always do a pregnancy test).

**What are important elements to gather in the sexual history of a patient?**

- Number of sexual partners in the past; Contraception use and type; History of venereal disease (HIV, PID, Hep).
What are some important causes of vaginal bleeding to consider in reproductive females who are not pregnant?

- Pregnancy; Exogenous hormone use; Coagulopathy; Thyroid dysfunction; Polycystic ovary syndrome; Leiomyomas; Adenomyosis

What are some important causes of vaginal bleeding in menopausal women?

- Endocervical lesions; Endometrial cancer; Exogenous hormone use; Atrophic vaginitis

What are some important elements in the physical to perform?

- A thorough vaginal exam; Examine for possible GI or GU bleed

What are some key points in management of vaginal bleeding in reproductive non-pregnant women?

- Make sure patient is not unstable (bleeding); Rule out pregnancy; OCP are often effective to control bleeding; NSAIDs are also effective in management

PELVIC/ABDOMINAL PAIN IN NONPREGNANT WOMEN

What is the single most important test to do on a female who presents with pelvic/abdominal pain?

- Pregnancy test

What are some important points to know about each of the following causes of pelvic pain in nonpregnant women:

- **Adnexal torsion**
  - It is a surgical emergency; Often will have a history of cysts or tumors; Exercise or intercourse often precede pain; Often sudden onset of unilateral pelvic pain; U/S and early surgical consult is important

- **Ovarian cysts**
  - They may twist, bleed, or rupture; Sudden unilateral pelvic pain is common; Must distinguish from possible ectopic; U/S is an important diagnostic tool

- **Endometriosis**
  - Very common cause of cyclic pain; Most common in the third decade of life; Often due to ectopic endometrial tissue; Often can get a normal pelvic exam
Adenomyosis

Often present with dysmenorrhea; Most common in the fourth decade of life; Pelvic can show a symmetrical large uterus; Analgesic and hormonal tx often help

Leiomyomas (fibroids)

It is a smooth muscle tumor; Most common in fourth decade of life; Typically estrogen-growth responsive; U/S will often detect fibroids; Analgesic and hormonal tx often help as well

ECTOPIC PREGNANCY

What must be ruled out in any female who presents with pelvic/lower abdominal pain or syncope?

Ectopic pregnancy

What are some important points to know about ectopic pregnancy (EP)?

Leading cause of first-trimester death; Implantation of fertilized egg outside the uterus; Most EPs occur within the fallopian tube

What are some major risk factors for EP?

Pelvic inflammatory disease; Use of intrauterine device; History of tubal surgery; Exposure to diethylstilbestrol (DES) in utero

What is the classic triad for the clinical presentation of EP?

Pelvic pain, spotting, and amenorrhea

What are some clinical features of a ruptured EP?

Rebound tenderness, hypotensive, and adnexal mass

What are some less common clinical features of a ruptured EP?

Syncope, unexplained shock, tenesmus, or shoulder pain

What is the differential diagnosis for a suspected EP?

Ovarian rupture/torsion, abortion, and surgical abdomen

What is the single most important test to do on any female of child-bearing age?

Pregnancy test

How does a pregnancy test work?

Pregnancy tests rely on the detection of β-hCG, human chorionic gonadotropin is a hormone produced by the trophoblast

Qualitative pregnancy tests are positive at what level?

β-hCG is >20 mIU/mL in urine; β-hCG is >10 mIU/mL in serum
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is a concern of doing a urine pregnancy test?</td>
<td>Dilute urine can be false-negative, especially early in pregnancy</td>
</tr>
<tr>
<td>If the bedside urine pregnancy test is negative, but EP is still a consideration, what is the next step?</td>
<td>Quantitative serum test should be done</td>
</tr>
<tr>
<td>How is the definitive diagnosis of EP made?</td>
<td>Surgery; Visualization during laproscopy; Ultrasound</td>
</tr>
<tr>
<td>What is the primary purpose of U/S?</td>
<td>Determine if there is an intrauterine pregnancy (IUP)</td>
</tr>
<tr>
<td>If U/S shows an IUP, is EP now excluded?</td>
<td>No—should consider heterotopic pregnancy</td>
</tr>
<tr>
<td>What should be noted about transabdominal ultrasound (TA)?</td>
<td>Less invasive; Wider field of view and easier orientation; Requires a full bladder; Transvaginal if TA is not diagnostic</td>
</tr>
<tr>
<td>What are some findings on ultrasound that may be suggestive of an EP?</td>
<td>Echogenic adnexal mass; Free pelvic fluid</td>
</tr>
<tr>
<td>What is the discriminatory zone?</td>
<td>The level of β-hCG at which an IUP can be visualized by U/S</td>
</tr>
<tr>
<td>What is the discriminatory zone of TA U/S?</td>
<td>β-hCG &gt;6000 mIU/mL</td>
</tr>
<tr>
<td>What is the discriminatory zone of TV U/S?</td>
<td>β-hCG &gt;1500 mIU/mL</td>
</tr>
<tr>
<td>What is the preferred medical management for EP?</td>
<td>Methotrexate (MTX)</td>
</tr>
<tr>
<td>What is the mechanism of MTX?</td>
<td>Inhibits dihydrofolic acid reductase: Interferes with DNA synthesis, cellular respiration, and repair</td>
</tr>
<tr>
<td>What are some things to keep in mind about the use of methotrexate?</td>
<td>Surgical tx may be needed if MTX fails; MTX use should be in conjunction with close follow-up</td>
</tr>
<tr>
<td>What is the most common surgical method for EP?</td>
<td>Laparoscopic salpingostomy</td>
</tr>
<tr>
<td>What are the key points in the management of EP?</td>
<td>Patient should go to the OR if unstable; Medical approach is preferred to surgery; Alloimmunization can occur—give Rhogam</td>
</tr>
</tbody>
</table>

**EMERGENCIES DURING EARLY PREGNANCY**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some factors associated with pregnancy-related death?</td>
<td>Poor prenatal care; Unmarried; Advanced maternal age; Minority race</td>
</tr>
</tbody>
</table>
Name some leading causes of pregnancy-related death?
- Pulmonary embolism (PE)
- HTN (i.e., stroke)
- Hemorrhage

Name some common causes of first trimester bleeding?
- Abortion
- Ectopic pregnancy
- Gestational trophoblastic disease
- Cervical infection

**Abortion**

What is the definition of spontaneous abortion (SAB) or miscarriage?
The loss of pregnancy prior to 20 weeks or delivery of a fetus <500 g

What is the most common cause of SAB?
Chromosomal abnormalities

What are some risk factors associated with SAB?
- Poor prenatal care
- Advanced maternal care
- Infections

What are some clinical features of SAB?
Vaginal bleeding, cramping, and abdominal pain

What is the most common method of surgical evacuation in the first trimester?
Dilation and curettage (D&C)

What is the most common method of surgical evacuation in the second trimester?
Dilation and evacuation

Name the different types of abortion and their treatment:

- **Threatened abortion**
  - Vaginal bleeding with no cervical dilation; Tx: verify live fetus and bed rest

- **Inevitable abortion**
  - Vaginal bleeding with cervical dilation; No expulsion of products of conception (POC); Tx: surgical evacuation

- **Incomplete abortion**
  - Partial expulsion of POC; Tx: typically admit for D&C

- **Complete abortion**
  - Complete expulsion of POC; Tx: none

- **Missed abortion**
  - Death of fetus and retained POC; Tx: surgical evacuation of POC

**Gestational Trophoblastic Disease**

What is gestational trophoblastic disease (GTD)?
- Rare neoplasm of the trophoblastic cells that produce hCG
Name three types of hydatidiform moles for each description:

<table>
<thead>
<tr>
<th>Description</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karotype of product is 69XXY due to two sperms that fertilize egg, fetal parts are present</td>
<td>Incomplete mole</td>
</tr>
<tr>
<td>Karotype of product is 46XX due to sperm that fertilizes an egg with no DNA, no fetal parts</td>
<td>Complete mole</td>
</tr>
<tr>
<td>GTD that becomes malignant, penetrates the myometrium, and can potentially metastasize</td>
<td>Invasive mole</td>
</tr>
</tbody>
</table>

What are some clinical features of GTD?
- Vaginal bleeding, hyperemesis gravidarum, and HTN

What diagnostic abnormalities are typical of GTD?
- Very high hCG (>100,000), U/S that shows absence of fetal heart and “snowstorm” appearance

What are some key points in management of GTD?
- D&C and monitor hCG (should trend down); Also monitor for possible metastasize (rare); Most do well after removal

What is an important complication to consider in GTD?
- Choriocarcinoma

What are some key points in the management of choriocarcinoma?
- Chemotherapy that typically achieves almost 100% remission

**Hyperemesis Gravidarum**

What is hyperemesis gravidarum (HEG)?
- It is excessive nausea and vomiting that leads to dehydration/electrolyte imbalance

What are some important points to consider in HEG?
- It affects about 2% of all pregnancies; The presence of abdominal pain is unusual; Associated with weight loss and ketosis; Severe cases require admission

What is an important consideration for anyone who presents with HEG?
- Gestational trophoblastic disease

What are some key points in the management of HEG?
- NPO and IV fluids; Antiemetics; Refractory cases may require termination
**EMERGENCIES DURING LATER PREGNANCY**

### Hypertensive Emergencies

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>How is hypertension defined during pregnancy?</td>
<td>It is over 140/90 or a 20 mm Hg increase in systolic pressure or 10 mm Hg increase in diastolic pressure</td>
</tr>
</tbody>
</table>
| Name four types of hypertension that can occur during pregnancy?         | 1. Chronic hypertension  
2. Transient hypertension  
3. Preeclampsia  
4. Eclampsia |
| What are some risk factors that determine HTN in pregnancy?              | Multiple gestations; Nulliparity; Age >40; Obesity; GTD                  |
| What is the definition of preeclampsia?                                 | It is HTN after 20 weeks with proteinuria                                |
| What is believed to be the cause of preeclampsia?                       | Disturbed blood flow to the placenta                                    |
| What are important diagnostic tests used to diagnose preeclampsia and their typical findings: | More than 140/90 (even one reading merits a workup)                      |
| Blood pressure                                                          | Urine protein concentration of 0.1 g/L in two random collections or 0.3 g/day in a 24-hour collection |
| Urine protein collection                                                 | Headache, edema, abdominal pain, and visual disturbances               |
| What are some clinical features of preeclampsia?                        | Blood pressure of >160/110 and more than 5 g/day of protein in the urine |
| What is the definition of severe preeclampsia?                          | Thrombocytopenia and elevated liver function tests (LFTs)               |
| What are some other abnormal laboratory findings in severe preeclampsia? | Thrombocytopenia and elevated liver function tests (LFTs)               |
| What is the definition of eclampsia?                                    | It is essentially preeclampsia with the presence of seizures from 12 weeks to 1 month after delivery |
| What are the key points in the management of severe preeclampsia and eclampsia? | Magnesium sulfate for seizure prophylactic; Control HTN with methyldopa; Induce labor if fetus/mother unstable; Delivery is definitive cure |
**What is the definition of HELLP syndrome?**  
Hemolytic anemia, Elevated LFT, and Low Platelets

---

**Abruptio Placentae**

**What is the definition of abruptio placentae (placental abruption)?**  
It is separation of the placenta from the uterine wall

**What are some risk factors associated with placental abruption?**  
HTN; Trauma; Cocaine use; Advanced maternal age; Multiparity

**What are some clinical features of a placental abruption?**  
Third trimester bleeding, painful contractions, and fetal distress

**What are some complications of a placental abruption?**  
Fetal or maternal death; Disseminated intravascular coagulation (DIC); Hypovolemic shock

**How is placental abruption typically diagnosed?**  
U/S

**What are the key points in the management of a placental abruption?**  
Admit and resuscitation if in shock; C-section if fetus/mother unstable; Induction if stable; Rhogam is indicated

---

**Placentia Previa**

**What is the definition of placentia previa?**  
Implantation of the placenta over the cervical os (total, partial, or marginal)

**What are some risk factors for placentia previa?**  
Multiparity; Advanced maternal age; Smoking

**What are some clinical features of placentia previa?**  
Late pregnancy painless bleeding

**What is important to keep in mind during an exam?**  
Avoid a pelvic exam until an U/S is done

**What are some complications of placentia previa?**  
Preterm delivery; Hypovolemic shock

**What are the key points in the management of placentia previa?**  
Resuscitation if in shock; Rhogam when indicated; C/S if unstable or fetus is mature

---

**Premature Rupture of Membranes**

**What is the definition of premature rupture of membranes (PROM)?**  
Spontaneous rupture of membranes before labor. If it occurs preterm, it is PPROM
How is PROM diagnosed? Gush of fluid, positive pool ferning, or nitrazine test
What are some key points in the management of PROM? Induction of labor if failure to progress in 24 hours and Abx if chorioamnionitis is suspected (increased WBC count, fever, and uterine tenderness)

**Preterm Labor**

What is the definition of preterm labor (PTL)? Onset of labor prior to 37 weeks
What are some risk factors for PTL? PROM; Infection; Preeclampsia; Multiple gestations; Tobacco use
What is the most common cause of mortality in PTL? Lung immaturity
Name some commonly used tocolytics? Magnesium sulfate, indomethacin, and terbutaline
What are the purposes of tocolytics? Delay labor to allow administration of steroids for lung maturation
What are the key points in the management of PTL? Empiric Abx, hydration, tocolysis, and steroids if fetus less than 34 weeks

**EMERGENCIES DURING POSTPARTUM**

What are some important things to know about each emergency and their treatment:

**DVT/PE**
Leading cause of maternal death; Greatest risk is first few weeks after labor; Commonly will have SOB, CP, or shock; Tx: heparin or low molecular weight heparin (LMWH)

**Postpartum hemorrhage**
Related to one fourth of all postpartum deaths; Most occur within the first 24 hours; Consider uterine atony/rupture and inversion; Tx: if rupture = OR; atony = oxytocin; inversion = manual reduction
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postpartum infection</td>
<td>Most common postpartum complication; Fever, tenderness, and discharge (foul odor); Tx: drainage, debridement, and Abx</td>
</tr>
<tr>
<td>Peripartum cardiomyopathy</td>
<td>Present similar to CHF (DOE, cough, CP); Echo will show massively dilated chambers; Poor prognosis if no cause is found; Tx: diuretics and fluid restriction</td>
</tr>
<tr>
<td>Amniotic fluid embolus</td>
<td>Very acute onset and high mortality; Typically permanent neurological sequelae; Tx: supportive with high O₂ and monitor for DIC</td>
</tr>
</tbody>
</table>

## VULVOVAGINITIS

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the definition of vulvovaginitis?</td>
<td>It is inflammation of the vulva/vagina</td>
</tr>
<tr>
<td>What are some clinical features of vulvovaginitis?</td>
<td>Discharge, itching, and odor</td>
</tr>
<tr>
<td>What are some differentials to consider?</td>
<td>Infection; Foreign body; Allergic contact; Atrophic vaginitis</td>
</tr>
<tr>
<td>List some important points and treatment for each of the following:</td>
<td></td>
</tr>
<tr>
<td>Candida albicans</td>
<td>Dysuria, dyspareunia, and itching common; Wet prep of KOH to detect (shows hypae); Tx: topical-azole drugs or nystatin</td>
</tr>
<tr>
<td>Trichomonas vaginalis</td>
<td>High association with gonorrhea; Is almost always sexually transmitted; Associated with adverse outcomes in pregnancy; Slide prep will show teardrop trichomonads; Tx: metronidazole</td>
</tr>
<tr>
<td>Gardnerella vaginalis</td>
<td>Is almost always sexually transmitted; Commonly have malodorous discharge; Associated with PROM and endometritis; Tx: metronidazole</td>
</tr>
<tr>
<td>Genital herpes</td>
<td>Commonly caused by HSV-2 serotype; Neonatal infection can be devastating; Commonly have painful ulcers; Avoid normal delivery if active lesions; Tx: acyclovir or valacyclovir</td>
</tr>
</tbody>
</table>
### Foreign body
Very common in children and adolescents; Often have malodorous discharge; Children tend to insert tissues and objects; Adolescents tend to leave tampons; Can grow *E.coli/anaerobes* if left too long; Tx: remove object

### Contact vulvovaginitis
Contact dermatitis due to irritant (i.e., tights); Typically have erythema and edema; Commonly have superimposed infection; Tx: R/O infection, remove irritant, and steroids in severe cases

---

**PELVIC INFLAMMATORY DISEASE**

What is the definition of pelvic inflammatory disease (PID)?
It is a wide spectrum of infections of the upper female genital tract

What are the two most common causes of PID?
1. *Neisseria gonorrhoeae*
2. *Chlamydia trachomatis*

What is the pathophysiology of PID?
It is an infection that starts at the cervix and vagina and ascends up the genital tract

What are some immediate complications of PID?
Salpingitis; Endometritis; Tubo-ovarian abscess

What are some long-term complications of PID?
Infertility; Chronic pain; Ectopic pregnancy

Name some risk factors of PID.
Multiple sexual partners; History of STD; Frequent douching; Sexual abuse

What are some clinical features of PID?
Lower abdominal pain, vaginal bleeding or discharge, dyspareunia, but can also be asymptomatic

What is the minimum CDC criteria for the diagnosis of PID?
Cervical motion tenderness; Lower abdomen, adnexal, or uterine tenderness

What are some other diagnostic criteria for PID?
Fever; WBC >10,000/mm³; Elevated CRP or ESR; Cervical infection with *N. gonorrhoeae* or *C. trachomatis*

What are some common pelvic exam findings in PID?
Cervical motion, uterus, and adnexal tenderness

What is the name of the condition of RUQ tenderness and jaundice in the setting of PID?
Fitz-Hugh-Curtis Syndrome
What are some important points in the management of PID?

Rule out ectopic pregnancy; Cervical swab for culture and stain; Empiric treatment for gonorrhea/chlamydia; Patient education

What are some criteria for admission?

Ovarian abscess; Unable to tolerate PO; Peritonitis; Failed outpatient management

**CLINICAL VIGNETTES**

45-year-old G4P5 who just delivered twins followed by two whole placentas now has copious vaginal bleeding; PE: 800 cc blood in 5 minutes with boggy uterus

Uterine atony

19-year-old female with no PMH presents via EMS with a syncopal episode, patient has now regained consciousness and mentions she was treated for an STD 2 years ago; pelvic: cervical motion tenderness; Labs: positive pregnancy test

Ectopic pregnancy

37-year-old G2P1 at 10 weeks presents with severe nausea and emesis along with vaginal bleeding; pregnancy test: $\beta$-hCG >100,000 mIU/mL; U/S: no fetal activity and a snowstorm appearance

Gestational trophoblastic disease

67-year-old female with PMH of HTN, CAD, and DM presents with painless vaginal bleeding, but otherwise has no other associated symptoms such as dysuria or abdominal pain; vaginal exam: no cervical tenderness

Endometrial cancer

19-year-old G0P0 presents with a sudden onset of left sided pelvic pain soon after her basketball game, aside from a past history of an ovarian cyst, is otherwise healthy

Adnexal torsion

23-year-old G5P0 at 6 weeks presents with painless vaginal bleeding, but is otherwise healthy; pelvic: closed OS

Threatened abortion

41-year-old G2P1 at 21 weeks presents with headache as well as lower extremity swelling; PE: BP of 150/95, +1 lower extremity edema; Labs: significant proteinuria

Preeclampsia
6-year-old female is brought in by her mother for a vaginal malodorous discharge, but is otherwise healthy

Foreign body

34-year-old female in her third trimester presents after an MVC with vaginal bleeding along with painful vaginal contractions; fetal heart monitoring: late decelerations

Abruptio placenta

23-year-old female in her postpartum period presents with dyspnea and chest pain that she describes as sharp and worse on inspiration; PE: unremarkable

Pulmonary embolism
CHAPTER 14

Trauma

GENERAL APPROACH

What is the leading cause of death in people under the age of 45 in the United States?

Trauma: 50 million deaths occur each year, half of which require medical attention

Name the top three trauma-related deaths

1. Motor vehicle crashes (MVCs)
2. Falls
3. Burns and fire-related death

What are the three peak times for traumatic death and common causes of death for each:

First peak (immediate death)
Laceration of the great vessels; Airway obstruction; Massive head injury; High C-spine injury

Second peak (minutes–few hours)
Tension pneumothorax; Cardiac tamponade; Multiple injuries leading to hypovolemia; Ruptured spleen; Massive hemothorax

Third peak (days–weeks)
Sepsis; Pneumonia; Multiorgan failure

What constitutes the primary survey?

ABCDE: Airway, breathing circulation, disability (neuro), exposure

What is the single most important intervention to perform on all trauma patients at the scene?

Airway control with C-spine stabilization

What are some techniques to secure an airway on the field?

Endotracheal tube; Esophageal-tracheal combi tube; Laryngeal mask airway (LMA)

What is the procedure of choice to secure an airway on the field?

Endotracheal intubation

What is the most reliable method to confirm ET placement?

Visualization of the tube passing the cords
Although pediatric airway management is similar to adults, what are two differences?

1. Children <9 years; use uncuffed ET tube
2. Children <10 years; needle cric preferred over surgical cric

What surgical technique can one use if intubation fails?

Needle cricothyrotomy

What are some methods used to quickly assess volume status in trauma patients?

Skin color, capillary refill, pulse, mental status

What type of access should be done in any trauma patient?

Intravenous (IV) access with two large-bore IVs for rapid fluid infusion

What is the difference between colloid and crystalloid fluids?

Colloid: contains protein such as albumin and fresh-frozen plasma; Crystalloid: little or no protein such as normal saline (NS) or lactated ringers

Are there any advantages of colloids over crystalloid fluids?

Small amount of colloid can effect a large change in intravascular volume, crystalloids are just as effective/cheaper

What is the optimal fluid type and amount that should be used for initial resuscitation?

2 L of lactated ringers or normal saline

What is minimal amount of circulating volume loss to produce signs of shock?

30%

What is the first sign of hemorrhagic shock?

Tachycardia and cutaneous vasoconstriction

What is shock?

Shock is a state where the oxygen demands of the body are not met

What category of shock is most common in trauma?

Hypovolemic shock (hemorrhage)

What is the crystalloid to blood replacement ratio (mL)?

3:1

<table>
<thead>
<tr>
<th>Hemorrhagic Shock</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (mL)</td>
<td>0–750</td>
<td>750–1500</td>
<td>1500–2000</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>Blood volume loss (%)</td>
<td>0–15</td>
<td>15–30</td>
<td>30–40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>&lt;100</td>
<td>&gt;100</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Fluid replacement</td>
<td>Crystalloid</td>
<td>Crystalloid</td>
<td>Crystalloid and blood</td>
<td>Crystalloid and blood</td>
</tr>
<tr>
<td>Mental status</td>
<td>Anxious</td>
<td>Anxious</td>
<td>Confused</td>
<td>Lethargic</td>
</tr>
</tbody>
</table>
Name five potential spaces where life-threatening bleeding can occur?

1. Chest
2. Abdomen
3. Pelvis
4. Bilateral femoral fractures
5. External wounds

What clinical index is widely used to assess neurological function?

Glasgow Coma Score (GCS)

Name the three components of GCS

1. Eye opening
2. Verbal response
3. Motor response

What GCS score is indicative of severe neurological impairment?

8 or less—“eight and it’s too late”

Which component of the GCS has the highest prognostic factor?

Motor response

What are some examples of blunt trauma?

MVC, falls, assaults, and pedestrian-automobile accidents

What are some major factors determine severity of injury in an MVC?

Ejection from vehicle; Size and weight of vehicle; Location of victim in vehicle; Use of restraints; Direction of impact; Speed of car at impact

Do lateral impacts or frontal impacts carry a higher mortality in a MVC?

Lateral impacts

What is the mortality rate of a fall from 30 feet?

50%

What is the basic pattern of injury in falls where victims land on their feet?

Calcaneous fracture; Acetabular fracture; L1-L2 compression fracture

What are some examples of penetrating trauma?

Guns, knifes, arrows, swords

What are some major determinants of injury in gunshot wounds (GSW)?

Mass of projectile; Muzzle velocity; Location and trajectory of projectile

### HEAD INJURY

What is the most common cause of death from trauma?

Central nervous system (CNS) injury

What is the most common mechanism of injury?

MVC
What are the five layers of the scalp? Skin
Connective tissue
Aponeurosis
Loose areolar tissue
Pericranium

What is the thinnest region of the skull that is most vulnerable to injury? Temporal region

What are the three layers of meninges? 1. Dura mater
2. Arachnoid membrane
3. Pia mater

Name the regions of the brain. Cerebrum
Cerebellum
Brainstem
   Midbrain
   Pons
   Medulla

What portion of the brainstem controls the reticular activating system? Midbrain; Pons

What portion of the brainstem controls the cardiorespiratory system? Medulla

What is the Monroe-Kellie doctrine? The total volume in the intracranial compartment is constant

Why is this significant in head injury? The intracranial space does not tolerate increases in pressure very well such as tumors, bleeding, or brain swelling and has limited ability to compensate

<table>
<thead>
<tr>
<th>Intracranial Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>High</td>
</tr>
<tr>
<td>Severe</td>
</tr>
</tbody>
</table>

What is the threshold of intracranial pressure at which compression or ischemia can occur? 20 mmHg

What is the goal for the management of ICP? Maintaining ICP less then 20 mmHg and consider the placement of ventriculostomy catheter (can drain and monitor ICP)
What are some indications for ICP monitoring? GCS of less than 8 or abnormal CT suggest of ICP

What is the hallmark of brain injury? Altered level of consciousness

Which head-injured patients require a head CT? All but the most minor head-injured patients

### Traumatic Brain Injury

What is the most important evaluation to do in a person suspected of traumatic brain injury (TBI)? Serial GCS evaluation

What are the three categories of TBI and prevalence? 1. Mild: 80% 2. Moderate: 10% 3. Severe: 10%

**Categories of TBI:**
- **Mild TBI**
  - GCS of 13–15 with brief loss of consciousness (LOC)
- **Moderate TBI**
  - GCS of 9–12 and may be confused with possible focal neuro deficits
- **Severe TBI**
  - GCS of 8 or less: can have mortality up to 40% and most survivors have significant disabilities

What physical finding is indicative of TBI? LOC

What are some key points in the management of TBI? Rapidly diagnose any mass lesions followed by evacuation; Treat any extracranial lesions; Avoid any secondary brain injuries such a hypotension, hypoxity or hypoglycemia

**During a physical exam, what particular findings should one look for?** GCS, pupillary changes, extremity movement, and ability to answer questions

**What is the initial diagnostic test of choice in the setting of TBI?** Noncontrast CT of the head

**What are five key features to look for on head CT?**
- Blood
- Cistern
- Brain
- Ventricles
- Bone

**What is the period of risk highest for posttraumatic seizure?** First week after head trauma
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are the risk factors for a posttraumatic seizure?</td>
<td>Cortical contusions, subdural hematoma, penetrating head injury, epidural, and depressed skull fractures</td>
</tr>
<tr>
<td>Does anticonvulsant prophylaxis play a role?</td>
<td>Some recommend that phenytoin be given in the first week</td>
</tr>
<tr>
<td>What is the general deposition of those with mild head injury?</td>
<td>Most can be safely observed and discharged if normal neuro function</td>
</tr>
<tr>
<td>What role does serial neuroassessment have in mild head injury?</td>
<td>Patients with mild head injury can still develop posttraumatic intracerebral hematomas and brain swelling</td>
</tr>
<tr>
<td>What factors are considered when deciding if a patient with mild head injury can return to play sports?</td>
<td>If LOC and amnesia occurred</td>
</tr>
<tr>
<td>What is a major risk factor for sustaining head injuries?</td>
<td>History of head injuries</td>
</tr>
<tr>
<td>What is a cerebral concussion?</td>
<td>Head injury that typically results in brief loss of neurologic function such as LOC or amnesia</td>
</tr>
<tr>
<td>What are some other clinical features of a head concussion?</td>
<td>Nausea, vomiting, and confusion that often resolve rapidly</td>
</tr>
<tr>
<td>What is the typical finding on a head CT?</td>
<td>Usually normal</td>
</tr>
<tr>
<td>What is a cerebral contusion?</td>
<td>Similar to a concussion, but with more pronounced neurologic findings</td>
</tr>
<tr>
<td>What are some clinical features of a cerebral contusion?</td>
<td>More severe neurologic findings such as obtundation or coma</td>
</tr>
<tr>
<td>What regions of the brain are typically injured in a cerebral contusion?</td>
<td>Frontal and temporal regions</td>
</tr>
<tr>
<td>What are some findings on a head CT?</td>
<td>Lesions at the site of impact (coup contusion) and site opposite the impact (contrecoup contusion)</td>
</tr>
<tr>
<td>What is an important delayed complication of cerebral contusions?</td>
<td>Cerebral hematoma or edema</td>
</tr>
<tr>
<td>What are some key points in the management of cerebral contusions?</td>
<td>Typically admit for observation; Monitor for signs of greater intracranial pressure; If suspect complication, repeat head CT</td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>--------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>What is diffuse axonal injury (DAI)?</strong></td>
<td>Serious diffuse brain injury as a result of traumatic deceleration frequently causing a persistent vegetative state in patients</td>
</tr>
<tr>
<td><strong>What are some clinical features of DAI?</strong></td>
<td>Prolonged coma often with posturing and autonomic dysfunction (poor prognosis)</td>
</tr>
<tr>
<td><strong>What is the initial CT for patients who end up with DAI?</strong></td>
<td>Normal in most cases</td>
</tr>
<tr>
<td><strong>What are some later CT findings for DAI?</strong></td>
<td>Intraventricular hemorrhage; Hemorrhage within the corpus callosum; Small focal areas of low density</td>
</tr>
<tr>
<td><strong>What are some key points in the management of DAI?</strong></td>
<td>Admission with neurosurgery consultation</td>
</tr>
</tbody>
</table>

**Penetrating Head Injuries**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Distinguish between high-velocity and low-velocity injuries.</strong></td>
<td>High velocity: bullets; Low velocity: arrows and knives</td>
</tr>
<tr>
<td><strong>Is there a difference in prognosis between high- and low-velocity injuries?</strong></td>
<td>Yes: high-velocity projectiles carry a very high mortality</td>
</tr>
<tr>
<td><strong>Why are high-velocity injuries more destructive?</strong></td>
<td>Kinetic energy of the projectile destroys surrounding tissues</td>
</tr>
<tr>
<td><strong>What is the initial treatment for high-velocity injuries to the head?</strong></td>
<td>IV antibiotics and anticonvulsants</td>
</tr>
<tr>
<td><strong>Injury to which part of the brain carries the highest mortality?</strong></td>
<td>Basal ganglia, brainstem, and posterior fossa</td>
</tr>
<tr>
<td><strong>What is the primary factor that determines prognosis in low-velocity injuries?</strong></td>
<td>Location of the brain injury</td>
</tr>
<tr>
<td><strong>What is the initial management for a protruding object in the head such as knife or arrow?</strong></td>
<td>Leave it alone! The risk of hemorrhage mandates removal in the OR</td>
</tr>
</tbody>
</table>

**Skull Fractures**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Where do linear skull fractures most commonly occur?</strong></td>
<td>Temporal bone</td>
</tr>
<tr>
<td><strong>What is the most important complication to monitor in skull fractures?</strong></td>
<td>Intracranial hematoma</td>
</tr>
</tbody>
</table>
What are the treatment guidelines for the following types of skull fractures:

- **Open skull fractures**: Operative intervention
- **Depressed skull fractures**: Operative intervention to raise fragment
- **Linear skull fractures (nondepressed)**: None

**Is surgery generally required for depressed skull fractures?**

No

**When is surgery typically indicated in depressed skull fractures?**

Cerebrospinal (CSF) leak or cosmetic purposes

**What are the physical findings associated with basilar skull fractures?**

CSF leak (rhinorrhea/otorrhea); Periorbital ecchymosis (Raccoon’s eye); Hemotympanum; Retroauricular ecchymosis (Battle’s sign)

**Why are CSF leaks significant?**

Increased risk of meningitis

**Is there a role for prophylactic antibiotic use in CSF leak?**

It can actually increase mortality (can use in consultation with neurosurgery)

### Hemorrhage

**What is the most common artery involved in a epidural hematoma?**

Middle meningeal artery

**What is the classic clinical scenario for an epidural hematoma?**

Initially LOC followed by a lucid period then a coma (only in 1/3 of cases)

**What are some clinical features of an epidural hematoma?**

Mass effect on brain: contralateral hemiparesis with a fixed dilated pupil on the side of the hematoma

**What is the classic CT finding of an epidural hematoma?**

Biconvex lesion; Associated temporal/parietal skull fracture

**What are some key points in the management of an epidural hematoma?**

Immediate neurosurgical consultation; Often requires surgical decompression; Consider use of mannitol to decrease ICP

**What is the mechanism by which subdural hematomas occur?**

The bridging veins often tear resulting in intrinsic bleeding and mass effect

**What are some groups that are more susceptible to subdural hematomas?**

Alcoholics; Elderly (smaller brain volume)
What are some clinical features of a subdural hematoma?
Mass effect: range from headache to lethargy and coma

What is the classic CT finding of a subdural hematoma?
Crescent-shaped lesion

What are some key points in the management of a subdural hematoma?
Immediate neurosurgical intervention; Distinguish from chronic subdural, which may not require immediate surgery

---

**NECK TRAUMA**

Why are penetrating neck injuries so dangerous?
The high density of vascular, neurologic, and visceral structures

Name some important structures in the neck:
- **Vascular**
  - Carotid, jugular, vertebral, and great vessels
- **Nerves**
  - Vagus, phrenic, sympathetic trunk, and cranial nerve (CN) V
- **Others**
  - Esophagus, trachea, thoracic duct, and lung apices

What is the mortality rate of a missed neck injury?
10–15%

Which muscle of the neck, if not violated, can neck injuries be managed non-operatively?
Platysma

What is the first concern in any penetrating neck injury?
Airway injury

What are some factors that determine if a patient should be managed operatively or nonoperatively?
Stability, presence of hard signs, and location of the injury (zones)

What are some examples of hard signs?
Stridor, bleeding, and expanding hematoma

What are some soft signs?
Hoarseness, dysphonia, hemoptysis, dysphagia, and odynophagia

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<table>
<thead>
<tr>
<th>Three Zones of the Neck</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zone I</td>
</tr>
<tr>
<td>Zone II</td>
</tr>
<tr>
<td>Zone III</td>
</tr>
</tbody>
</table>
What mandates exploration? | Zone II injury with hard signs or an unstable patient
---|---
What is the standard diagnostic approach in a stable patient who has a neck injury? | Angiography, EGD/barium swallow, and tracheobronchoscopy
What are the three most common mechanisms of blunt injury to the neck? | 1. Direct impact (car/all-terrain vehicle)
2. Excessive flexion/extension
3. Compression (hanging)
What are some common causes of airway loss? | Expanding hematoma, thyroid fracture, tracheal fracture, and aspirations
What are some contraindications to orotracheal intubation in neck injury? | Obvious pharynx, larynx, tracheal, or facial injury
What are the clinical features of a missed esophageal injury? | Fever, tachycardia, and sepsis
What diagnostic test should be done in a patient who has an abnormal GCS with a normal CT in the setting of a neck injury? | Four-vessel angiogram

**BONY ORAL-MAXILLOFACIAL INJURY**

What potential injuries are associated with an oral-maxillofacial (OMF) injury? | Cervical injury
---|---
What is the first consideration when doing the primary survey? | Airway obstruction
What are some considerations in an oral-maxillofacial injury? | Search for life-threatening bleeding in the thoracic, abdominal, head, and extremities
What percentage of OMF injuries do mandibular fractures make-up? | 2/3
What is the most common mechanism of injury in mandibular injury? | Blunt trauma from assaults
What part of the mandible are most susceptible to injury? | Condylar, angle, and symphysis
How can airway obstruction occur in the setting of OMF injury | Dentures/avulsed teeth and aspiration of blood
What are the most common maxillofacial injuries that occur in blunt trauma? | Nasal and mandibular fractures
What is the most common physical finding of mandibular fractures? | Malocclusion of the teeth
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some physical finding of a mandibular fracture?</td>
<td>Malocclusion, trismus, pain, ecchymosis of the floor of the mouth, and deviation opening the mouth</td>
</tr>
<tr>
<td>What is important to remember about mandibular fractures?</td>
<td>Fracture in two or more places &gt;50%</td>
</tr>
<tr>
<td>What is the diagnostic test of choice for mandibular fractures?</td>
<td>Dental panoramic view (Panorex)</td>
</tr>
<tr>
<td>What are some key points in the management of mandibular fractures?</td>
<td>Consultation with ENT for reduction/fixation; Open fractures typically require antibiotics; Update tetanus status</td>
</tr>
<tr>
<td>What are some common causes of mandibular dislocations?</td>
<td>Excessive opening of mouth (i.e., laughing) and trauma</td>
</tr>
<tr>
<td>What are some clinical features of a mandibular dislocation?</td>
<td>Jaw displaced to unaffected side, difficulty talking/eating, and anterior open bite</td>
</tr>
<tr>
<td>What is commonly done for a mandibular dislocation?</td>
<td>Manual reduction</td>
</tr>
<tr>
<td>What is the main reason to obtain an x-ray evaluation?</td>
<td>Rule out fractures</td>
</tr>
<tr>
<td>What areas define the midface?</td>
<td>Orbital-zygomatic-maxillary complex</td>
</tr>
<tr>
<td>What is the typical mechanism of injury to the midface?</td>
<td>Blunt trauma from MVC and assault</td>
</tr>
<tr>
<td>What does mobility of the maxillary dentition indicate?</td>
<td>Maxillary fracture</td>
</tr>
<tr>
<td>What physical finding is most common in midface fractures?</td>
<td>Malocclusion</td>
</tr>
<tr>
<td>What physical maneuver can confirm a suspected midface fracture?</td>
<td>Grab anterior maxillary teeth and check for mobility of the hard palate</td>
</tr>
<tr>
<td>What specific exam should be done for any orbital/zygomatic complex?</td>
<td>Check pupils, globes, and visual acuity</td>
</tr>
<tr>
<td>What diagnosis is suspected when one finds a firm fixed point of limitation in gaze?</td>
<td>Entrapment of extraocular muscles</td>
</tr>
<tr>
<td>Do anterior or posterior epistaxis bleed more?</td>
<td>Posterior</td>
</tr>
<tr>
<td>What fractures are CSF leaks associated with?</td>
<td>Midfacial, frontal sinus, and basilar skull fractures</td>
</tr>
<tr>
<td>What is the radiographic test of choice for midface fractures?</td>
<td>CT scan with facial cut</td>
</tr>
</tbody>
</table>
When does osseous healing begin to occur? 7 days
What are the four stability points of the zygoma? 1. Frontal bone
2. Maxilla
3. Temporal bones
4. Frontozygomatic structure
What is the general physical finding in zygomaticomaxillary (ZMC) fractures? Depression at the site of trauma, pain on mandibular opening, or limited opening
What is the goal of the treatment of ZMC fractures? Surgical reduction without internal fixation
What is an orbital blowout fracture? Fractures of any of the orbital walls secondary to direct impact of the globe
What is the weakest section of the orbital complex? The medial wall and floor of the orbit
What are some clinical features of an orbital blowout fracture? Enophthalmos, upward gaze palsy, diplopia, pain on eye movement, and V2 parasthesia
What is the mechanism by which extraocular eye movement dysfunction occurs? Extraocular muscle entrapment
What is the radiographic test of choice? Modified-Waters view
What are some key points in the management of an orbital blowout fracture? Patients should get ophthalmology f/u; Persistent entrapment = surgery; Consider antibiotics if sinus involvement
What are maxillary fractures commonly due to? Direct trauma to the face (large force)
How are maxillary fractures commonly classified? LeFort I
LeFort II
LeFort III
Palate-facial
Pyramidal
Craniofacial
What are some clinical features of maxillary fractures? Midface mobility, malocclusion, CSF rhinorrhea, and soft-tissue swelling
What is the preferred imaging modality for maxillary fractures? CT
What are some key points in the management of maxillary fractures? ABCs; CT to delineate the extent of fracture; Antibiotics if sinus involvement
### SPINAL TRAUMA

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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</table>
| Name three common mechanisms of spinal cord injury (SCI).                | 1. MVC  
2. Violence  
3. Falls |
| What is the average age and gender of those who sustain spinal cord injury? | Males with an average age of 30 |
| What is the percentage of patients with SCI who also have other significant injuries? | 50% |
| What fraction of SCI involves the cervical spine?                       | 50% |
| What is the general treatment for spinal column injury?                  | Treatment centers on preventing further injury through fixation (internal or external) |
| Describe the general composition of the spinal column.                   | 7 cervical vertebrae, 12 thoracic vertebrae, 5 lumbar vertebrae, and 5 fused sacral vertebrae |
| Is the thoracic column flexible?                                         | No, it is relatively stiff due to the orientation of facets and interaction with ribs |
| Is the lumbar column flexible?                                           | Yes |
| Why is this important?                                                   | The point where the thoracic column and lumbar column meet creates a point where shear stress occurs making T12-L1 a site of common spinal trauma |
| What are the three main spinal cord pathways and what fibers are carried? | 1. Dorsal column pathway: position/vibration  
2. Spinothalamic pathway: pain/temperature  
3. Corticospinal pathway: movement |
| What is the “three columns of the spine” theory?                        | A way to visualize the biomechanical stability of the spine |
| Name the boundaries of the three columns of the spine:                  |  
- **Anterior column**  
  Anterior 2/3 vertebral body and anterior longitudinal ligament  
- **Middle column**  
  Posterior 1/3 of the vertebral body and posterior longitudinal ligament  
- **Posterior column**  
  Facets and posterior ligaments |
How many of the columns must be compromised in order for the spine to be considered unstable? 2 out of 3

What is the consequence of an unstable vertebral column? Spinal cord injury with possible paralysis

Does spinal column injury equate to spinal cord injury? Not necessarily

What are some examples of different types of mechanisms that can cause spinal injury? Axial loading; Hyperflexion/extension; Rotational injuries

What is complete spinal cord injury? Irreparable damage with no discernible motor, sensory, or electrical function

What is incomplete spinal cord injury? Some preservation of sensory and/or motor function

What are some examples of incomplete spinal cord injury:

- Posterior cord injury: Loss of vibration and position
- Anterior cord injury: Loss of bilateral motor, temperature, and pain
- Central cord injury: Loss of pain and temperature; Motor loss (arms > legs)
- Brown-séquard injury: Ipsilateral loss of position/vibration/motor; contralateral loss of pain/temperature

What presumption must be made with any tenderness along the spinal column? There is vertebral fracture and ligamentous injury

For which patient population should one have a higher index of suspicion for spinal injury? Elderly, children, patients with osteoporosis, and history of metastatic bone cancer

What is SCIWORA? Spinal cord injury without radiographic abnormality

Why is this more common in children? Elasticity of their ligaments

Why is this more common in the elderly? Underlying cervical stenosis

When should a cervical spine injury be suspected? High-speed MVC; Fall >15 feet; Any injury above the clavicle; Diving accidents; Electrical injury

What are the most commonly missed fractures in the cervical spine? C1-C2 and C7-T1
What is the Nexus criteria?  
It is a set of criteria that help to identify those patients with a low probability of injury to the cervical spine 

List the Nexus criteria.  
Normal alertness; Not intoxicated; No cervical midline tenderness; No focal neurologic deficits; No distracting injuries 

What are the three views recommended to assess cervical injury?  
1. Lateral  
2. AP  
3. Open mouth (odontoid view) 

Which view is commonly obtained?  
Lateral alone is adequate in 90% of cases 

True or False: As long as all cervical vertebrae are visualized, the film is adequate.  
False. C7-T1 must be visualized 

What are the ABCS of assessing lateral films?  
Alignment  
Bone  
Cartilage  
Soft tissue 

Alignment  
Anterior/posterior/spinolaminar lines 

Bones  
Check vertebral body heights 

Cartilage  
Intervertebral spaces and facets 

Soft tissue  
Look for soft tissue swelling, especially C2-C3 

When is a CT of the cervical spine indicated?  
Inadequate plain films; Fracture on films; Unconscious patients 

When is an MRI indicated?  
Neurological deficits 

What is a flexion-extension film useful for?  
A flexion-extension film is typically used to assess ligamentous injury 

What is a Jefferson fracture?  
Axial loading injury that results in a C1 burst fracture with C2 involvement 

What is an odontoid fracture?  
Type I  
Involves the tip of the dens of C2 

Type II  
Transverses the dens at the junction of the body of C2 

Type III  
Involves C2 vertebral body
Which odontoid fracture carries the worse prognosis? Type II

What is a clay shoveler’s fracture? Avulsion of the spinous process of C6 – T3 typically the result of flexion injury or direct trauma

What is a hangman’s fracture? Bipeduncular fracture of C2 due to excessive extension

What is the most common site of injury in the thoracolumbar injury? T12-L1

When are AP and lateral films indicated? If a patient complains of pain in the region or if the mechanism of injury is suggestive

When is a CT indicated? If there is a fracture noted on plain films, film is inadequate, or patient cannot respond

When is an MRI indicated? Neurological deficits

When is a compression fracture? Anterior vertebral body fracture

What is a burst fracture? Vertebral body is crushed in all directions

What is a chance fracture? Fracture due to excessive flexion such as an MVC where a seatbelt is used

What are some key points in the management of spinal injury? Protect the cord by stabilization; CT scan if plain films are indeterminate

THORACIC TRAUMA

What fraction of patients who sustain injury to the chest require thoracotomy? 10–25%

What findings are indicative of serious chest injury? JVD, subcutaneous emphysema, and tracheal deviation

If a patient with penetrating thoracic injury loses vital signs in the ED, what procedure is indicated? Emergent thoracotomy

If a patient with blunt thoracic injury loses vitals in the ED, would one still do a thoracotomy? No—the mortality rate approaches 100%

What are some primary indications for urgent thoracotomy or sternotomy? Massive hemothorax; Cardiac tamponade; Aortic tear; Esophageal disruption or perforation; Open pneumothorax
Name the six immediate life-threats associated with thoracic trauma.

1. Airway obstruction
2. Tension pneumothorax
3. Massive hemotherax
4. Open pneumothorax
5. Flail chest
6. Cardiac tamponade

What are the six potential life-threatening injuries to the thoracic region?

1. Blunt cardiac injury
2. Traumatic rupture of the aorta
3. Major tracheobronchial injury
4. Diaphragmatic injury
5. Esophageal perforation
6. Pulmonary contusion

**Open Pneumothorax**

<table>
<thead>
<tr>
<th>Question</th>
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<tbody>
<tr>
<td>What is the most common cause?</td>
<td>Penetrating injuries</td>
</tr>
<tr>
<td>What size is considered a large defect?</td>
<td>&gt;3 cm</td>
</tr>
<tr>
<td>What are some clinical features of an open pneumothorax?</td>
<td>Hypoxia; Hypoventilation; Tachypnea; Chest pain</td>
</tr>
<tr>
<td>Should the wound be fully closed with a dressing?</td>
<td>No! It can convert to tension pneumothorax</td>
</tr>
<tr>
<td>What is the standard treatment?</td>
<td>Tube thoracostomy on the affected side</td>
</tr>
</tbody>
</table>

**Tension Pneumothorax**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What is the pathogenesis of tension pneumothorax?</td>
<td>Air is able to enter, but not leave the pleural space</td>
</tr>
<tr>
<td>What are some clinical features of tension pneumothorax?</td>
<td>Decreased breath sound on one side; Tracheal deviation (late finding); Subcutaneous emphysema; Hypotension</td>
</tr>
<tr>
<td>What immediate action is required for tension pneumothorax?</td>
<td>Needle decompression followed by tube thoracostomy</td>
</tr>
<tr>
<td>Where do you insert the needle for needle decompression?</td>
<td>Second intercostal space mid-clavicular line or fifth intercostal space in anterior axillary line</td>
</tr>
<tr>
<td>What is the consequence of decompression?</td>
<td>Converts tension pneumothorax into simple pneumothorax</td>
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**Hemothorax**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What is a common cause of a hemothorax?</td>
<td>Damage to the primary or secondary pulmonary vessels</td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
<tr>
<td>How much blood can each hemothorax contain?</td>
<td>Upto 3 L</td>
</tr>
<tr>
<td>What will the chest x-ray show?</td>
<td>Total opacity of the affected side “white out”</td>
</tr>
<tr>
<td>How much fluid is required before an upright CXR can detect it?</td>
<td>200 mL</td>
</tr>
<tr>
<td>What are some clinical features of a hemothorax?</td>
<td>Dullness to percussion, diminished breath sounds, and decreased tactile fremitus</td>
</tr>
<tr>
<td>Do all hemothorax need surgical intervention?</td>
<td>No—most are self-limited</td>
</tr>
<tr>
<td>What are some indications for surgical intervention for a hemothorax?</td>
<td>Initial chest tube output is &gt;1500 mL; 50% hemothorax; Chest tube output is &gt;200 mL/hour over 4–6 hours</td>
</tr>
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</table>

**Flail Chest**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What are some clinical features of a flail chest?</td>
<td>Paradoxical movement of the flail segments with spontaneous breathing</td>
</tr>
<tr>
<td>What are some common radiographic findings in a patient with flail chest?</td>
<td>Two or more consecutive rib fractures with pulmonary contusions</td>
</tr>
<tr>
<td>What is the patient at high risk for?</td>
<td>Pneumothorax and hemothorax</td>
</tr>
<tr>
<td>What is the test of choice?</td>
<td>CXR (CT more accurate)</td>
</tr>
<tr>
<td>What are some key points in the management of a flail chest?</td>
<td>Low threshold for ET intubation; Pain control; Pulmonary physiotherapy</td>
</tr>
<tr>
<td>What are some indications to intubate?</td>
<td>PaCO₂ &gt;55 mmHg; Respiratory fatigue; PaO₂ &lt;60 mmHg</td>
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</table>

**Cardiac Tamponade**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What is cardiac tamponade?</td>
<td>Build-up of fluid in the pericardial space that obstructs effective cardiac pumping</td>
</tr>
<tr>
<td>What is the mechanism by which cardiac tamponade commonly occurs?</td>
<td>Penetrating injuries</td>
</tr>
<tr>
<td>What is the most common site of perforation that leads to cardiac tamponade?</td>
<td>Right atrium</td>
</tr>
<tr>
<td>What is Beck’s Triad?</td>
<td>Hypotension; Muffled heart sounds; Jugular venous distension (JVD)</td>
</tr>
<tr>
<td>How common does Beck’s triad present?</td>
<td>1/3 of cases</td>
</tr>
</tbody>
</table>
Traumatic Aortic Rupture

Where is the site where the aorta most commonly tears?
Ligamentum arteriosum

What is the most common mechanism by which a aortic rupture occurs?
Sudden deceleration (i.e., falls and MVCs)

About how many patients who sustain a traumatic aortic rupture die at the scene?
Up to 90%

What are some clinical features of a traumatic aortic ruptures?
Retrosternal pain; Pulse deficits; Dyspnea; Upper extremity hypertension with decreased femoral pulses

How is the diagnosis of a aortic rupture usually made?
History is very important, but an abnormal CXR along with confirmative studies can confirm the diagnosis

What are some findings on a CXR that may be suggestive of a aortic rupture?
Superior mediastinum widening, indistinct aortic knob, rib fractures, left hemothorax, and left apical pleural cap

What are two confirmative tests that can be used to help diagnose a aortic rupture?
1. CT
2. Transesophageal echocardiography (TEE)

What are some key points in the management of aortic ruptures?
Immediate surgical repair; Regulate BP to minimize tear

Blunt Cardiac Injury

How does a blunt cardiac injury (BCI) commonly occur?
Commonly occurs in a high-speed MVC where the chest strikes the steering wheel

What is the spectrum of BCIs?
Myocardial concussion; Myocardial contusion; Tamponade; Cardiac rupture

How do myocardial concussions occur?
Typically the heart will strike the chest wall with no permanent cell damage
What are some possible complications of myocardial concussions? Hypotension; Dysrhythmias
What are some key points in the management of a myocardial concussion? Most will resolve without treatment; ACLS for dysrhythmias (i.e., asystole)
What is a myocardial contusion? More forcible injury to the myocardium from impaction against the chest wall
What ventricle is more commonly injured in a myocardial contusion? Right ventricle
What are some commonly used tests to distinguish low-risk from high-risk patients? ECG; Echocardiography
What are some key points in the management of a myocardial contusion? Observation for low-risk patients (normal vitals, asymptomatic, etc.); Admit patients with conduction abnormalities

Pulmonary Contusion

What is a very common mechanism by which a pulmonary contusion occurs? Deceleration (MVCs or falls)
What is an important point to know about a pulmonary contusion? Most common potential lethal chest injury
What are some common clinical features of a pulmonary contusion? Dyspnea, tachycardia, tachypnea with chest wall tenderness
What are some common CXR findings in a pulmonary contusion? Typically show patchy alveolar infiltrate to consolidation, usually within 6 hours of injury
What are some potential complications of a pulmonary contusion? Pneumothorax; Pneumonia (most significant)
What are some key points in the management of a pulmonary contusion? Adequate ventilation to allow healing; Low-threshold for intubation; Liberal pain control to allow adequate breathing/coughing

Diaphragmatic Injury

Which side of the diaphragm is most injured in blunt trauma? Left, presumably due to an inherent weakness on that side
Which side of the diaphragm is most injured in penetrating trauma? Left, since most assailants are right-handed
What is the operative approach for diaphragmatic repair? Celiotomy
### Trauma

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td><strong>What are some sequela of a diaphragmatic rupture?</strong></td>
<td>Herniation of viscous that can result in SBO, incarceration, and compression of the heart/lungs (these can present years later)</td>
</tr>
<tr>
<td><strong>True or False: most diaphragmatic tears will spontaneously heal.</strong></td>
<td>False: most ruptures will require operative repair</td>
</tr>
</tbody>
</table>

### Abdominal Trauma

<table>
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<tr>
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<th>Answer</th>
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<tbody>
<tr>
<td><strong>How should any abdominal injury be divided into?</strong></td>
<td>Blunt versus penetrating trauma</td>
</tr>
<tr>
<td><strong>What are three common causes of blunt trauma?</strong></td>
<td>1. MCV</td>
</tr>
<tr>
<td></td>
<td>2. Falls</td>
</tr>
<tr>
<td></td>
<td>3. Assaults</td>
</tr>
<tr>
<td><strong>What are two common causes of penetrating trauma?</strong></td>
<td>1. Gunshot wounds</td>
</tr>
<tr>
<td></td>
<td>2. Knives</td>
</tr>
<tr>
<td><strong>Name three regions of the body to consider in abdominal trauma?</strong></td>
<td>1. Peritoneal cavity</td>
</tr>
<tr>
<td></td>
<td>2. Retroperitoneal cavity</td>
</tr>
<tr>
<td></td>
<td>3. Pelvis</td>
</tr>
<tr>
<td><strong>What is the general management for anyone who is hemodynamically unstable or has peritoneal signs?</strong></td>
<td>To the OR for laparotomy</td>
</tr>
<tr>
<td><strong>What are the goals of exploratory laparotomy?</strong></td>
<td>Immediate hemostatic control; Control any GI contamination; Operative repair</td>
</tr>
<tr>
<td><strong>What are some signs of hypotension?</strong></td>
<td>Tachycardia, obtundation, cool skin, poor capillary refill</td>
</tr>
<tr>
<td><strong>What are peritoneal signs caused by?</strong></td>
<td>Irritation to the peritoneal lining caused by leaking of blood, bile, or gastric juices</td>
</tr>
<tr>
<td><strong>What are some peritoneal signs?</strong></td>
<td>Guarding, rigid abdomen, or rebound tenderness</td>
</tr>
<tr>
<td><strong>What percentage with hemoperitoneum will have acute findings?</strong></td>
<td>80%</td>
</tr>
<tr>
<td><strong>What is the most important thing to do in a suspected abdominal injury with an initial benign exam?</strong></td>
<td>Serial abdominal exams</td>
</tr>
<tr>
<td><strong>What are other factors in a trauma situation that is associated with abdominal injury?</strong></td>
<td>Chest injury, pelvic fracture, hypotension, and lap belt contusion</td>
</tr>
<tr>
<td><strong>What is the most commonly injured solid organ?</strong></td>
<td>In blunt trauma: spleen; In penetrating trauma: liver</td>
</tr>
</tbody>
</table>
What is the most commonly injured hollow organ?

Small bowel

What are the three diagnostic tests to consider in any trauma to the abdomen?

1. Diagnostic peritoneal lavage (DPL)
2. Focused abdominal sonography for trauma (FAST exam)
3. CT

Diagnostic peritoneal lavage (DPL):

What is it

Catheter placement in the peritoneal cavity to see if there is any initial return of fluid. If nothing, place liter of warm saline and drain

Indications

Hemodynamically unstable with questionable abdominal injury

Accuracy

Sensitivity and specificity is 95%

Advantages

Fast, accurate, and inexpensive

Disadvantages

Invasive, nontherapeutic rate of 20%, inability to pick up retroperitoneal and isolated diaphragmatic injuries

Criteria for positive DPL

10 ml of gross blood; >100 k RBC/mm³; >500 WBC/mm³; Bacteria, bile, and food particles

FAST exam:

What is it

Use of sonography to rapidly detect hemoperitoneum

Indications

Hemodynamically unstable with questionable abdominal injury

Accuracy

Sensitivity and specificity is between 70–90% and poor at detecting solid organ damage

Advantages

Fast, accurate, and inexpensive

Disadvantages

Poor at detecting solid organ damage and small amounts of blood, requires training

CT:

What is it?

CT is used to evaluate solid organ injury and detect fluid/air in cavity

Indications?

Hemodynamically stable patients that require abdominal evaluation

Accuracy?

92–98%
Advantages? Noninvasive, evaluates solid organ injury, and evaluates retroperitoneal injuries
Disadvantages? Expensive, time, variable in detecting hollow viscus injury

Blunt Abdominal Injury

What is the first thing to assess in blunt trauma to the abdomen? ABC! Airway with proper ventilation and assess hemodynamic stability
What is the most common abdominal organ injured in blunt trauma? Spleen followed by liver
If the patient is unstable and has obvious peritoneal signs, what is the next step? Proceed directly to exploratory laprotomy
What is the test of choice in a stable patient with suspected abdomen injury? CT
What are the major forces involved with blunt trauma? Crushing, shearing, and stretching
Name the possible organ injury with the following:
- Right lower rib fracture: Liver and gallbladder
- Left lower rib fracture: Spleen and left kidney
- Epigastric contusion: Duodenum, pancreas, and mesentery
- Anterior pelvis fracture: Bladder and urethra

Penetrating Abdominal Injury

What percentage of those with GSW require operative repair? Up to 90%
What percentage of those with knife wounds require operative repair? 1/4
What abdominal organ is most commonly injured in penetrating injuries? Liver
Is CT useful in GSW? Exploratory laprotomy is diagnostic and therapeutic
What percentage of those with anterior stab wounds have peritoneal violation? 2/3
Of those with peritoneal violation, how many require operative management? 1/2
What are some indications for Ex Lap in a knife wound? Hemodynamically unstable, peritoneal signs, obvious evisceration
What is recommended in a stable patient with a knife wound? Local wound exploration

### GENITOURINARY TRAUMA

What is the cause of most genitourinary (GU) injuries? Blunt trauma
What is a key marker of GU injury? Hematuria
What are the possible locations of GU injury? Upper: kidney and ureter; Lower: bladder and urethra
What should be done with macroscopic hematuria? Further evaluation
What percentage of renal injuries will have no hematuria? 15%
Is initial return of blood on catheter placement concerning? No—is usually catheter-related
What should be done with microscopic hematuria? Further imaging if mechanism of injury is suggestive
What are some diagnostic tests utilized?
- **Urethrogram**
  In any suspected urethral injury
- **Cystogram**
  Important to fully inflate bladder to detect small injuries and done post-void
- **CT**
  Test of choice for renal trauma
- **US**
  Useful for detecting renal parenchyma injury
- **Intravenous pyelogram**
  Largely replaced by CT for staging
What percentage of renal injury is from blunt trauma? 80%
What percentage of those with blunt renal trauma will lose a kidney? 5%
What is the general management of those with renal trauma that is stable? Nonoperative management
What is the indication of operative management? Unstable, hilar/pedicle damage, and significant blood in urine
How common is post-injury hypertension? 15%
What is the cause of most bladder injury? Blunt trauma
What percentage of blunt trauma is extraperitoneal? 80%

What are the indications of a cystogram? Gross hematuria; Seatbelt contusions; Pelvic fractures

What are extraperitoneal injuries associated with? Fractures of superior and inferior pubic rami

What are intraperitoneal injuries associated with? Seatbelt injuries with a full bladder

What is the general treatment for bladder rupture? Ex Lap followed by primary repair

How are most extraperitoneal bladder injuries managed? Bladder drainage alone

What is the cause of most ureteral injury? Penetrating trauma

What are the diagnostic tests of choice? Intravenous pyelogram (IVP) and CT

What is the general treatment? Primary repair and stenting

What is the cause of most urethral injury? Blunt trauma

What are posterior urethral injuries associated with? Pelvic fracture

What are anterior urethral injuries associated with? Penetrating trauma

What is the diagnostic test of choice? Urethrogram

**ORTHOPEDIC TRAUMA**

What is a dislocation? Total loss of articulation contact

What is a subluxation? Partial loss of articular congruity

What is a fracture: Break (partial or complete) in continuity of the bone

- Open fracture Fracture that results in open communication
- Closed fracture Fracture with intact skin

What are some important descriptions for bone fractures? Pattern, morphology, location, open versus closed, and neurovascular status

Match the possible nerve injury:

- Anterior shoulder dislocation Axillary nerve injury
- Humeral shaft Radial nerve injury
Posterior hip dislocation
Proximal fibular fracture

What percentage of fractures are missed in those with multiple injuries?
10–15%

What are important components of the physical exam?
Inspection, palpation, range of motion, and neurovascular status

What is the initial diagnostic test of choice?
Plain films with at least two views, above and below the injury

What is the initial treatment in any fracture?
Reduction; Splint; Irrigate if open; Update tetanus status

Are antibiotics recommended in open fractures?
Yes

What is the purpose of splinting?
Immobilization to help control bleeding, pain, and prevent secondary injuries

Should open fractures be splinted?
Splint as they are

What is important to assess after splinting of open fractures?
Neurovascular status

What is the gold standard of splinting?
Plaster of paris

What is the mangled severity scoring system (MSSS)?
A scoring system to help guide whether a severely mangled limb should be salvaged versus amputated

What are the primary components of the MSSS?
Skeletal/soft tissue injury; Limb ischemia; Shock; Age

What is the most important factor when deciding amputation versus salvage?
Neurologic status

What is the primary issue in any open fracture?
Infection (osteomyelitis)

What is an important management issue in addition to antibiotics?
Adequate debridement

What is the initial treatment for open fractures?
Early irrigation; Early splinting

What is a typically antibiotic regiment?
First generation cephalosporin/aminoglycoside; Penicillin if the injury is barnyard related; Tetanus toxoid

Is operative management indicated for open fractures?
Yes—take to OR within 6–8 hours
Hip dislocations:
- Are anterior or posterior dislocations more common?  
  Posterior
- What is a common cause of posterior hip dislocations?  
  MVC
- What percentage of hip dislocations result in sciatic nerve injury?  
  10–15%
- What is the most concerning complication?  
  Avascular necrosis (AVN)
- What is done to avoid AVN?  
  Immediate reduction (closed or open)

Femoral neck/shaft fractures:
- What is a common cause of a femoral neck fracture in children/adults?  
  High-energy impacts (i.e., MVC)
- What is a common cause of a femoral neck fracture in elderly patients?  
  Low-energy impacts (i.e., falls)
- What is a particular concern?  
  AVN
- What is the typical treatment?  
  Open reduction internal fixation (ORIF)
- What is important to rule-out in femoral shaft fractures?  
  Femoral neck fractures
- What is the typical treatment for femoral shaft fractures?  
  Intramedullary nailing

Knee dislocations:
- What is a common cause of knee injury?  
  Any high-force impact
- How often is the popliteal artery injured?  
  20%
- What is typically done to assess the popliteal artery?  
  Arteriography
- What nerve injuries are typically associated with knee dislocations?  
  Tibial and peroneal nerve
- What is the initial management in knee dislocations?  
  Urgent reduction

Tibial shaft fractures:
- What is a common cause of tibial shaft fractures?  
  High-energy impacts (i.e., MVC)
- What syndrome are tibial fractures associated with?  
  Compartment syndrome
- What is the typical treatment for tibial shaft fractures?  
  ORIF
Pelvic fractures:

What is the primary concern in any pelvic fracture? Life-threatening bleeding

How many liters of blood can the pelvis accommodate? 5 L

What do pelvic fractures have a high association with? Head, thoracic, and abdomen trauma

What is the mortality rate of open pelvic fractures? 50%

What is the mortality rate of major vascular disruption secondary to pelvic fractures? 75% (it is rare)

What is the initial management in suspected pelvic fractures? External fixation of the pelvis

What type of physical exam is important to perform in a pelvic fracture? Detailed lower neurovascular exam

Hand trauma:

What is important to know about hand injuries? It is the most injured part of the body

What assumption must be made if there is a laceration, swelling, and ecchymosis? Neurovascular damage

What is the Allen test used for? To test patency of both the radial and ulnar artery

What is the function of the radial nerve? Extension of the wrist

What is the function of the median nerve? Flexion of the wrist and opposition of thumb

What is the function of the ulnar nerve? Assist in flexion of wrist

What is compartment syndrome? A significant increase in pressure within a confined space (fascia)

What is the common cause of compartment syndrome? Any injury that leads to swelling within a confined space

What percentages of compartment syndrome do fractures account for? 50%

What fractures are highly associated with compartment syndrome? Tibial fractures

What factors are associated with compartment syndrome? Reperfusion after 4–6 hours of swelling; Significant crush injury; Combined arterial and venous injury

What is a very common physical finding on exam? Pain out of proportion followed by paraesthesia
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some common signs on exam?</td>
<td>Swelling with pain on passive stretching</td>
</tr>
<tr>
<td>What is the first sign of compartment syndrome?</td>
<td>Loss of function</td>
</tr>
<tr>
<td>What is a late finding of compartment syndrome?</td>
<td>Loss of pulses</td>
</tr>
<tr>
<td>What is the primary treatment for compartment syndrome?</td>
<td>Fasciotomy</td>
</tr>
<tr>
<td>What is the typical pressure reading for fasciotomy?</td>
<td>Greater than 30 mm Hg or 20–30 mm Hg with symptoms</td>
</tr>
<tr>
<td>What is rhabdomyolysis?</td>
<td>It is any type of significant muscle injury that results in release of toxins</td>
</tr>
<tr>
<td>What is the most feared complication of rhabdomyolysis?</td>
<td>Kidney failure</td>
</tr>
<tr>
<td>What is the most sensitive marker for muscle damage?</td>
<td>Serum creatine phosphokinese (CPK)</td>
</tr>
<tr>
<td>What is the most common cause of rhabdomyolysis in trauma?</td>
<td>Anything that causes muscle death such as crush injuries</td>
</tr>
<tr>
<td>What is the most common cause of rhabdomyolysis in non-trauma situation?</td>
<td>Neuroleptic malignant syndrome; Malignant hyperthermia</td>
</tr>
<tr>
<td>What is the pathogenesis of rhabdomyolysis?</td>
<td>Fe: forms toxic oxygen metabolites; Myoglobin: forms casts to clog renal tubules</td>
</tr>
<tr>
<td>What is the primary objective in treatment?</td>
<td>Adequate fluids to ensure renal perfusion</td>
</tr>
<tr>
<td>What is another concern in rhabdomyolysis?</td>
<td>Hyperkalemia</td>
</tr>
<tr>
<td>What is the standard treatment to treat hyperkalemia?</td>
<td>Sodium bicarbonate and insulin to drive potassium into cells; Calcium to stabilize the heart; Kayexalate to bind potassium</td>
</tr>
<tr>
<td>What is the prognosis of rhabdomyolysis?</td>
<td>Generally good with most patients returning to baseline kidney function in 3–4 weeks</td>
</tr>
</tbody>
</table>

**TRAUMA IN PREGNANCY**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important points about trauma in pregnancy?</td>
<td>Most common cause of nonobstetric death; Fundamentally treating two patients; Management centers around mother; “What is good for the mother is good for the child”</td>
</tr>
</tbody>
</table>
### Important Caveats about Airway Management of Pregnant Trauma Patients
- Continuous 100% oxygen (esp. fetal Hb)
- Pulse oximetry monitoring
- RSI as required with normal medications
- Thoracostomy at third or fourth ICS

### Important Points in Circulatory Status
- Increased HR/low BP may reflect normal pregnancy, not shock
- Avoid supine position
- LR is preferred over NS
- Blood transfusion if failure to improve after 2 L of crystalloid

### Supine Hypotension Syndrome
- When the gravid uterus compresses the IVC, decreasing preload and CO when in supine position

### Optimal Position to Lay a Pregnant Trauma Patient
- Lateral decubitus position

### Obstetric Evaluation Components
- Uterine contractions
- Fetal heart rate (ensure between 120–160)
- Fundal height and tenderness
- Fetal movement
- Pelvic and rectal examination

### When is the Fetus Considered Viable?
- Gestational age >24 weeks

### Most Common Cause of Fetal Death Following Blunt Trauma
- Placental abruption

### Clinical Features of Placental Abruption
- Uterine tenderness, fetal distress, abdominal cramps, and signs of shock

### Most Important Preventative Measure in MVCs
- Properly worn seatbelts

## Clinical Vignettes

**18-year-old male is brought over by his parents due to concern for a head injury after a football game where the patient ran head first into another player, patient mentions he “blacked-out” but otherwise feels fine; PE: no focal neurologic deficits; CT of head: normal**
- Concussion

**81-year-old male with a history of afib was seen in the ED 3 days ago after falling and hitting his head, had a negative CT of the head at that time, but now is presenting with confusion; PE: unremarkable neuro exam; CT of head: now shows a crescent-shaped lesion**
- Subdural hematoma
19-year-old male who was at a diving competition is brought in by EMS in cervical precautions. Patient dove from a very high platform and mentions he could not extend his arms in time; Cervical films: C1 ring is fractured in multiple places

23-year-old female involved in a knife fight is being evaluated in the trauma bay and is currently complaining of dyspnea; PE: decreased breath sounds and hyper-resonance to percussion on the right chest

18-year-old female is emergently brought in by helicopter to the trauma bay to be evaluated for a gunshot wound to the chest, patient is intubated and suddenly becomes hypotensive; PE: jugular venous distension and muffled heart sound

You arrive at a scene involving a car accident, a patient was just extricated and is in obvious respiratory distress with suspected cervical spine injury. Is nasotracheal intubation the procedure of choice?

16-year-old male who was involved in a gang fight and hit squarely in the back with lead pipe is now complaining of back pain; PE: remarkable tenderness of his upper back; thoracic plain film: avulsion fracture of the spinous process of T2

57-year-old alcoholic male is brought into the ED by EMS after being knocked unconscious in a bar fight, patient was awake and demanding to go home, but now is unconscious; PE: unremarkable; CT of head: biconvex lesion near the temporal bone

61-year-old female is brought into the ED by paramedics after being extricated in a high-speed car collision, she is unconscious and unresponsive; PE: posturing; CT of head: widely scattered neuronal damage

34-year-old male is brought in by EMS from a high speed MVC where the patient was extricated and his side passenger was found dead; PE: fractured left femur and multiple scalp lacerations; CXR: fracture of the first rib and 9-cm superior mediastinum along with an indistinct aortic knob

Trauma

Jefferson fracture

Simple pneumothorax

Pericardial tamponade

No—orostracheal intubation is still the procedure of choice

Clay shoveler’s fracture

Epidural hematoma

Diffuse axonal injury (DAI)

Traumatic aortic rupture
41-year-old female is brought in by EMS after being hit by a car and was thrown 15-ft across the street, patient is currently hypotensive and unresponsive to fluids; FAST exam: blood in Morrison’s pouch

17-year-old female with no PMH is coming into the ED with an injury to her left eye. Patient mentions she was hit squarely in her left eye with a softball and now has double vision; PE: inability for the left eye to gaze upward; modified Waters view: air fluid level in maxillary sinus

8-year-old female is brought into the ED by her mother after being kicked in the chest by a horse at the ranch, the patient is having difficulties breathing and in significant pain; CXR: frank consolidation on the right lung

19-year-old male is brought into the ED by EMS after a diving accident where the patient dove head first and lost consciousness, patient is now in cervical precautions and is A&O x4; PE: clear fluid is slowly dripping down his left ear

67-year-old male with no PMH presents after an MVC where his chest hit the steering wheel and is coming into the ED with complaints of chest pain; PE: tenderness with palpation of the anterior chest wall; ECG: sinus tachycardia; Labs: normal cardiac enzymes

25-year-old male with a gunshot wound to the chest is currently being evaluated in the trauma bay when he suddenly becomes hypotensive and in respiratory distress with distended neck veins

Abdominal injury requiring laprotomy 15-ft

Orbital floor fracture

Pulmonary contusion

Basilar skull fracture

Myocardial concussion

Tension pneumothorax
What are some important causes of burns? Thermal; Chemical; Radiation; Electricity

What are some elements in the history to obtain in a patient who presents with burns? Any signs of respiratory distress?; Any toxic substance at the site of injury?; Did the burn occur within a closed space?

What is the “rule of nine”? It is used to estimate the body surface area burned, which guides treatment

<table>
<thead>
<tr>
<th>Body Part</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head and neck</td>
<td>9%</td>
</tr>
<tr>
<td>Each arm</td>
<td>9%</td>
</tr>
<tr>
<td>Anterior trunk</td>
<td>18%</td>
</tr>
<tr>
<td>Posterior trunk</td>
<td>18%</td>
</tr>
<tr>
<td>Each leg</td>
<td>18%</td>
</tr>
<tr>
<td>Perineum</td>
<td>1%</td>
</tr>
</tbody>
</table>

Can this be applied to infants and young children? No—they have proportionally larger heads

What are some clinical features to know for each of the following types of burns:

- **Superficial (First degree)**
  - Confined to superficial layer of skin; Erythema and pain, but no blisters; Sunburn most common cause; Heals in a week (does not scar)

- **Partial thickness (Second degree)**
  - Epidermal and top dermis involved; Blister formation is the hallmark; Thermal liquids most common cause; Heals in 2 weeks (some scarring)
Full thickness (Third degree)
Epidermal and full dermis involvement; Charred with leather appearance; Full skin and nerve permanently destroyed; Healing will only occur with grafting/surgery

Musculoskeletal (Fourth degree)
Involvement of muscle/fascia/bone; Necrosis is common; Melted metal is common cause; Debridement/amputation is common

What are some risk factors that makes a burn patient more predisposed to complications?
Immunocompromised; Extremes of ages; Associated head injury; Concomitant inhalation injury (i.e., CO)

What are some important basic management skills any bystander can utilize?
Remove patient from area; Also stop burning process; Apply a dry/clean/sterile dressing

What are some signs of an endangered airway in patient with thermal burn?
Respiratory problems (i.e., stridor); Carbonaceous sputum; Singed hair; Oropharyngeal swelling

What are some key points in the initial management of burn patients?
Very close monitoring of airway, breathing, circulation (ABCs); Low threshold for intubation; Aggressive fluid resuscitation in all but the most superficial of burns

What total body surface area (TBSA) will typically require aggressive fluid resuscitation?
TSBA >20%

What is the Parkland's formula?
Used to calculate the amount of fluid to give in the first 24 hours for moderate to severe burns

How is the Parkland’s formula used in the first 24 hours?
LR at 4 mL × kg × percentage burn with the half given over the first 8 hours and the rest given over 16 hours

What are some ways to measure fluid resuscitation?
Heart rate (<100 beats/min); Urinary output (0.5–1 mL/kg/hour); Mentation

What are some important complications to consider in burns?
Carbon monoxide and cyanide poisoning; Circumferential burns; Infections (late complication)
### What are some other management guidelines to remember with burns?
Morphine is commonly used for pain control; Prophylactic antibiotics for select patients; Tetanus prophylaxis; Contact burn centers for major burns; Escharotomy with circumferential burns

### Where are circumferential burns most dangerous?
Thoracic chest (compromise breathing); Extremities (compartment syndrome)

### List some important burn-care guidelines for minor burns?
Debride any lost tissue/broken blisters; Blisters on sole/palms can be left as is; Cool compresses for burn area; Remove all jewelry; Topical antibacterial agent; Discharge with pain medicine and follow-up

### What are some commonly used topical antibacterial agents for minor burns?
Bacitracin; Polymyxin B; Silver sulfadiazine

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### ELECTRICAL, LIGHTNING, AND CHEMICAL INJURIES

#### Electrical Injuries

<table>
<thead>
<tr>
<th>What is important to know about electrical injuries?</th>
<th>Leading cause of occupation-related death; It is more frequent in males between ages 20 and 40 years; Up to 45% of severe electrical injuries are fatal</th>
</tr>
</thead>
<tbody>
<tr>
<td>What types of electrical injuries are there?</td>
<td>Low voltage (&lt;1000 volts); High voltage (&gt;1000 volts); Lightning</td>
</tr>
<tr>
<td>What are some mechanisms of injuries due to electricity?</td>
<td>Direct effects of electrical current; Blunt injury (falls, muscle contractions, etc.); Conversion of electricity to thermal energy</td>
</tr>
<tr>
<td>What are some factors that contribute to the severity of electrical injuries?</td>
<td>Amount of current flowing via the body; Voltage; Resistance; Type of current (AC versus DC); Duration of currents</td>
</tr>
<tr>
<td>What are some features of exposure to AC currents?</td>
<td>Repetitive stimulation of muscles (spasms); Prolonged contact with electricity; AC current prevents self-release from source; Vfib most common dysrhythmia</td>
</tr>
</tbody>
</table>
What are some features of exposure to DC currents?

Single muscle spasms (typically thrown); Increased risk of trauma due to being thrown; Asystole most common dysrhythmia

What is the most common mechanism of injury in the following:

- **Low voltage**: Working on electrical circuits or appliances; Biting into cords (infants); Electrical weapons (taser)
- **High Voltage**: Conductive object contact with high voltage overhead lines
- **Lightning**: In open field or near a tall object

What is the most common cause of death in electrical injuries?

Cardiac arrhythmias; Respiratory arrest (paralysis of diaphragm)

What are some important baseline studies to consider?

CBC/Chem-7/Coag; ECG; U/A; Urine myoglobin; CK-MB; CPK

What are some other complications associated with electrical injuries?

Burns; Rhabdomyolysis; Myoglobinuria; Autonomic dysfunction; Vascular injuries; Cataracts

What are some key points in the management of electrical burns?

Electrical burns treated like thermal burns; Aggressive fluid replacement; Cardiac monitoring in severe injuries; Monitor for compartment syndrome; Also monitor for rhabdomyolysis (i.e., ARF); Tetanus prophylaxis

**Lightning Injuries**

What are some important points to know about lightning injuries?

High-intensity bursts of short duration; Direct current (up to 1.5–2 billion volts!); Rarely causes deep tissue burns; Fluid loss is rarely an issue

What are some common mechanisms by which lighting can cause injury?

Thermal burns; Blunt trauma from blast impact; Direct lightning strike; Lightning strikes nearby object

What are some common clinical features of a lightning strike?

Missing clothes/shoes, stunned, evidence of burns (not always), unconsciousness, headache, vision/hearing problems, and often have mild tachycardia/hypertension
What are important injuries to consider in the following organ systems:

**Central nervous system**
- Seizures; Loss of consciousness with amnesia; Peripheral nerve damage

**Cardiovascular system**
- Dysrhythmias (systole most common); Pericardial tamponade; Respiratory arrest

**Eyes and ears**
- Ruptured tympanic membrane is common; Corneal damage; Cataract formation

What are some important laboratory and diagnostic tests to consider?

- CBC/Chem-7/Coag; Cardiac enzymes; U/A; ECG; Cervical films for suspected spinal injury; CT for altered mental status

What are some key points in the management of lightning injuries?

- ABCs; Treat lightning burns like regular burns; Tetanus prophylaxis; Patients should be admitted with cardiac monitoring

---

**Chemical Injuries**

What are some important points for the following types of chemical burns:

**Acids**
- Acids are proton donors; Coagulation necrosis by denaturing proteins; Acid burns are typically more superficial

**Bases**
- Bases are proton acceptors; Severe injury (i.e., liquefaction necrosis); Bases tend to penetrate deeper into tissue

What are some factors that determine the severity of an acid/base burn?

- Length of contact of the agent; pH of the agent; Concentration of the agent; Volume of the agent

What are some commonly encountered acids?

- Hydrochloric acid; Sulfuric acid; Hydrofluoric acid

What are some commonly encountered bases?

- Sodium hydroxide; Ammonia; Sodium and calcium hypochlorite

What are some diagnostic tests to consider in chemical burns?

- Usually none in minor chemical burns; CBC/Chem-7/Coag in severe burns; Endoscopy for ingestions; CXR for ingestions as well
**NEAR-DROWNING**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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</thead>
<tbody>
<tr>
<td>What is near-drowning?</td>
<td>Survival after suffocation in a liquid medium</td>
</tr>
<tr>
<td>What are some important points to know about near-drowning?</td>
<td>Common cause of accidental death; Incidence highest in males between ages 1 and 5 years; Drowning is much more common in summer</td>
</tr>
<tr>
<td>What are some risk factors of near-drowning?</td>
<td>Inability to swim; Use of illicit drugs or alcohol; Poor adult supervision; Risk-taking behavior</td>
</tr>
<tr>
<td>What are some complications of near-drowning?</td>
<td>Hypothermia; Acute respiratory distress syndrome; Bradycardia; Hypoxia</td>
</tr>
<tr>
<td>What is important about hypothermia in the setting of near-drowning?</td>
<td>It has a neuroprotective effect which may allow prolonged resuscitation without permanent sequelae</td>
</tr>
<tr>
<td>What are some major pulmonary complications?</td>
<td>Surfactant washout; Adult respiratory distress syndrome (ARDS); Pulmonary edema</td>
</tr>
<tr>
<td>What are some major neurologic complications?</td>
<td>Cerebral edema; Hypoxia; Seizure</td>
</tr>
<tr>
<td>What are the most common arrhythmias?</td>
<td>Atrial fibrillation; Sinus bradycardia</td>
</tr>
<tr>
<td>What is the major factor in death due to drowning?</td>
<td>Cerebral hypoxia</td>
</tr>
<tr>
<td>What are some poor prognostic factors in near-drowning?</td>
<td>Submersion &gt;10 minutes; Time to CPR &gt;10 minutes; Water temp &gt;10 °C; GCS &lt;8; Resuscitation &gt;25 minutes</td>
</tr>
<tr>
<td>What are some pre-hospital management issues?</td>
<td>CPR; Possible cervical injury should be suspected; Pulses are difficult to palpate in hypothermia; Remove wet clothing; Consider various rewarming techniques</td>
</tr>
</tbody>
</table>
What are some in-hospital management issues?

- Treat organ-specific damage;
- Prevent secondary neurologic damage;
- Correct fluid/electrolyte imbalance;
- Permissive hypercapnia to avoid barotrauma

**HYPOTHERMIA**

What are the classifications of hypothermia?

- **Mild hypothermia**
  - Core temperature 32–35°C
- **Moderate hypothermia**
  - Core temperature 28–32°C
- **Severe hypothermia**
  - Core temperature below 28°C

What is the physiological response to hypothermia?

- Shivering; Increased adrenal activity; Increased thyroid activity; Peripheral vasoconstriction

Is a standard thermometer useful to measure the degree of hypothermia?

- No—cannot measure below 34.4°C

What is the most reliable method to measure temperature in hypothermia?

- Pulmonary artery probe

What are other less invasive methods to measure temperature in hypothermia?

- Rectal probe, tympanic membrane probe, and bladder probe

What are some causes of hypothermia?

- Environmental exposure; Malnutrition; Sepsis; Medications (i.e., general anesthetics); Hypothyroidism; Hypopituitarism

What are some clinical features of hypothermia:

- **Mild hypothermia**
  - Shivering; Hypertension; Confusion; Atrial fibrillation; Tachycardia

- **Moderate hypothermia**
  - Decreasing level of consciousness; Loss of shivering mechanism; Bradycardia; Cold diuresis; Dilated pupils

- **Severe hypothermia**
  - Coma; Oliguria; Asystole at <20°C; Pulmonary edema

What are some complications of hypothermia?

- Lactic acidosis; Bleeding diathesis; Rhabdomyolysis; Bladder atony; Frostbite
### Hypothermia

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are the characteristic ECG findings of hypothermia?</td>
<td>Prolongation of all intervals; Osborne wave (J-point elevation)</td>
</tr>
<tr>
<td>What are some examples of passive external rewarming?</td>
<td>Blankets; Humidified heated oxygen by mask; Removing wet clothing</td>
</tr>
<tr>
<td>In what degree of hypothermia is passive external rewarming used?</td>
<td>Mild hypothermia</td>
</tr>
<tr>
<td>What are some advantages of passive external rewarming?</td>
<td>Intense monitoring is not needed; Noninvasive</td>
</tr>
<tr>
<td>What is a disadvantage of passive external rewarming?</td>
<td>Slow process</td>
</tr>
<tr>
<td>What are some examples of active external rewarming?</td>
<td>Radiant heat; Electric heat blanket; Warm bath</td>
</tr>
<tr>
<td>At what degree of hypothermia is active external rewarming used?</td>
<td>Mild and moderate hypothermia</td>
</tr>
<tr>
<td>What are some advantages of active external rewarming?</td>
<td>Intense monitoring is not needed; Noninvasive; Can be combined with passive external rewarming</td>
</tr>
<tr>
<td>What is a disadvantage of active external rewarming?</td>
<td>May cause iatrogenic burns</td>
</tr>
<tr>
<td>What are some examples of active internal rewarming?</td>
<td>Warmed intravenous fluids; Peritoneal dialysis; Extracorporeal blood rewarming; Closed thoracic lavage</td>
</tr>
<tr>
<td>At what degree of hypothermia is active internal rewarming used?</td>
<td>Moderate and severe hypothermia</td>
</tr>
<tr>
<td>What are some advantages of active internal rewarming?</td>
<td>Fastest modality to raise core temperature; Most effective; Can be used if hemodynamically unstable</td>
</tr>
<tr>
<td>What is a disadvantage of active external rewarming?</td>
<td>Invasive; Intense monitoring</td>
</tr>
</tbody>
</table>

**Hypothermia**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is hyperthermia?</td>
<td>It is an elevation of core temperature above 37°C due to failure of thermoregulation</td>
</tr>
</tbody>
</table>
What are some important causes of hyperthermia? Heat stroke; Malignant hyperthermia; Neuroleptic malignant syndrome; Drugs (i.e., cocaine); Metabolic (i.e., DKA)

What are some risk factors for hyperthermia? Poor physical fitness; Obesity; Drug use; Dehydration

Describe the types of heat stroke

- Classic heatstroke
  Occurs commonly in elderly and the sick; Compromised thermoregulation; Cardiovascular and endocrine disorders

- Exertional heatstroke
  Common in young athletes; Typically massive exogenous heat; Exertional heat production

What are some common findings in the various types of hyperthermia:

- Heat exhaustion
  Mild hyperpyrexia; Nausea and vomiting; Signs of dehydration

- Heat stroke
  Temperature >105.8°F; Tachypnea; Rales; Excessive bleeding

- Malignant hyperthermia
  Muscle rigidity; Hypercarbia; Sinus tachycardia; Marked hyperthermia

- Neuroleptic malignant syndrome
  Altered mental status; Autonomic instability; Muscle rigidity; Hyperthermia

What are some complications of the various types of hyperthermia:

- Heat stroke
  Renal and hepatic failure; Acute respiratory distress syndrome; Disseminated intravascular coagulation; Seizures

- Malignant hyperthermia
  Rhabdomyolysis; Disseminated intravascular coagulation; Hypertension; Hyperkalemia

- Neuroleptic malignant syndrome
  Dysrhythmias; Pulmonary edema; Renal failure

What are the key points in management of hyperthermia?

- Lower core temperature to less then 38.8°C; Accurate core temperature measurements; Dantrolene in malignant hyperthermia; Bromocriptine for neuroleptic malignant syndrome (NMS); Treat metabolic derangements
When is the optimal time to begin cooling?
Immediately within the hour—golden hour

What are some techniques in cooling?
Immersion therapy (ice water bath); Evaporation (cool spray with fanning); Cold pack to axillary areas and groin

What are important points in patient education to avoid hyperthermia?
Lifestyle change (i.e., limit drug use); Caution when in hot weather; Ways to keep cool

ALTIMITUDE SICKNESS

What are some examples of altitude sickness?
Acute mountain sickness (AMS); High-altitude pulmonary edema; High-altitude cerebral edema

Define various heights of altitude:
- **High**: 8,000–12,000 feet
- **Very high**: 12,000–18,000 feet
- **Extremely high**: >18,000 feet

What are some important points to know about AMS?
Mild form of altitude sickness; It can occur at altitudes >6,000 feet; Three-fourth experience AMS at 10,000 feet

What are some clinical features of AMS?
Headache (most common); Dyspnea; nausea; Edema; Insomnia; Decreased urine output

What are some risk factors for AMS?
History of altitude sickness; Rate of ascent; Duration of stay at high altitude; Actual elevation

What is the most effective method to avoid AMS?
Slow ascent

What are some key points in the management of AMS?
Neve ascend with symptoms of AMS; Stop ascend or descend if symptomatic; Most cases are self-limiting; Low-flow oxygen

Name two commonly used drugs that help prevent AMS.
1. Acetazolamide
2. Ginkgo biloba

Name some other medications used for AMS?
Dexamethasone; Promethazine; Prochlorperazine
What are some methods to help prevent AMS prior to the ascend?

First camp at <8,000 feet; Avoid direct ascend >9,000 feet at one time; Well-hydration; Avoid narcotics, EtOH, and sleeping medicines; Pretreatment medication

What are some important points to know about in high-altitude pulmonary edema (HAPE)?

Major cause of death in altitude sickness; More common in ascents above 12,000 feet; Sudden presentation common; Children are more susceptible; More common in fit young climbers

What are some clinical features of HAPE?

Dry cough, dyspnea, fatigue, tachycardia, chest tightness, and periodic breathing

What are some key points in the management of HAPE?

Descend as soon as possible; Supplemental oxygen; Nifedipine prior to ascend; Descent in severe cases; Portable hyperbaric chamber use

What is high-altitude cerebral edema (HACE)?

Believed to be hypoxic-induced increase in cerebral blood flow along with decreased integrity of the blood-brain barrier

What are some clinical features of HACE?

Ataxia (most common); Decrease mental status; Papilledema; Retinal hemorrhage; Seizure; And rapid death from brain herniation is severe cases

What are some key points in its management?

Slow ascent whenever possible; When it occurs, immediate descent; Dexamethasone may be effective

DIVING INJURIES

What are some important points to know about diving injuries?

There are more then 1k diving injuries per year; Up to 10% of diving injuries are fatal

What are some specific elements to obtain in a diving history?

Activities prior to diving (esp. flying); Location (i.e., ocean); Dive times; Equipment used and gases breathed; Maximum depth, time spent, and rate of ascent; Dive problems
What are some complications associated with diving injuries? Hypothermia; Submersion injuries (drowning); Decompression sickness; Nitrogen narcosis; Barotrauma

What is the most common form of diving injury? Barotrauma

What is barotrauma? It is injury in air-filled spaces due to under-pressurization or over-pressurization during descent or ascent, respectively

Name some examples of barotrauma. Pulmonary barotraumas; Pneumomediastinum; Pneumothorax; Ear barotraumas

What is one of the most feared complications of diving? Air gas embolism (AGE)

What are two serious sequelae of AGE? 1. Myocardial infarction 2. Stroke

What are some clinical features of AGE? Dysrhythmia; Arrest; Change in mental status; Visual disturbances

What are some key points in the management of AGE? 100% oxygen; Recompression chamber; Ground transport to chamber

What is another feared complication of diving? Decompression sickness (DCS)

What is DCS? It is the release of bubbles from solution due to rapid reduction in pressure. Typically nitrogen bubbles are produced

What are two groups of DCS? 1. Type 1 DCS (musculoskeletal) 2. Type 2 DCS (neurologic)

What are some clinical features of Type 1 DCS? Pain to the arms or legs that ranges from mild discomfort to severe pain or may present as pruritus alone

What are some key points in the management of Type 1 DCS? Recompression; Watch for the progression to Type 2 DCS

What are three forms of Type 2 DCS? 1. Cerebral DCS (common in aviators) 2. Spinal DCS (common in divers) 3. Pulmonary DCS

What are some clinical features of cerebral DCS? Seizures, visual disturbances (blurry, diplopia, etc.), and hemiplegia

What are some clinical features of spinal DCS? Paresthesia, bladder paralysis, and incontinence
What are some clinical features of pulmonary DCS? Cough, dyspnea, and chest pain
What are some key points in the management of Type 2 DCS? Reduce size of bubbles via recompression 100% oxygen to wash out nitrogen; Admission for observation; Further recompression if new symptoms

BITEs

Cat Bites

What is the most common organism in cat bites? Pasteurella multocida
What are some other organisms that are commonly associated with cat bites? Fusobacterium; Staphylococcus; Actinomyces
What population demographics are most commonly bitten by cats? Elderly women (men are most commonly bitten by dogs)
What are some important elements in the history to obtain with regards to cat bites? Domestic or wild cat; Vaccine status; Tetanus status of patient
What are some important aspects of the physical to focus on? Neurovascular status; Bony injury; Tendon involvement; Joint space involvement; Foreign bodies
What are the essentials in the treatment of cat bites? Inspection of the wound; Debridement; Irrigation; Closure where indicated
What are some key points with respect to wound closure? Puncture wounds should be left open; Facial wounds can be closed primarily; Most other delayed primary closure
What are some indications for antibiotic prophylaxis? Immunocompromised patient; Hand wounds; Puncture wounds; Prosthetic valves
What are some common antibiotic regimens used? Ampicillin-clavulanate; TMP/SMX; Ciprofloxacin
What are two other important considerations? Rabies and tetanus status
What is cat-scratch disease? Regional lymphadenitis of the arms or legs that is typically unilateral and commonly affects children and adolescents
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What is the causative agent in cat-scratch disease?</td>
<td><em>Bartonella henselae</em></td>
</tr>
<tr>
<td>What is the typical incubation period?</td>
<td>Roughly 1 week</td>
</tr>
<tr>
<td>What is the treatment for cat-scratch disease?</td>
<td>Most cases are self-limited and may take months to resolve, severe cases may require antibiotics</td>
</tr>
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</table>

**Dog Bites**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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</thead>
<tbody>
<tr>
<td>What are some common pathogens involved with dog bites?</td>
<td><em>Pasteurella; Klebsiella; Streptococcus</em></td>
</tr>
<tr>
<td>Which organism can potentially cause a lethal infection in immunocompromised patients?</td>
<td><em>Capnocytophaga canimorsus</em></td>
</tr>
<tr>
<td>What are some clinical features of patients who may be infected with <em>Capnocytophaga canimorsus</em>?</td>
<td>Sepsis; Acute renal failure; Endocarditis</td>
</tr>
<tr>
<td>What are the key points in the management of recent lacerations (&lt;12 hours)?</td>
<td>Inspection; Debridement; Irrigation; Closure</td>
</tr>
<tr>
<td>What is typically done for dog bites that are older than 12 hours or on the hand?</td>
<td>Left open after irrigation then closed 3–5 days after (delayed primary closure)</td>
</tr>
<tr>
<td>What else is important to consider with dog bites?</td>
<td>Rabies; Tetanus</td>
</tr>
<tr>
<td>What are some indications for antibiotic prophylaxis with dog bites?</td>
<td>Same as cat bites</td>
</tr>
<tr>
<td>What are some commonly used antibiotic regiments?</td>
<td>Ampicillin-clavulanate; TMP/SMX; Ciprofloxacin</td>
</tr>
<tr>
<td>What are some indications for admission and use of IV antibiotics?</td>
<td>Injury to tendons, bones, and joints Systemic infections</td>
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**Human Bites**

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<tr>
<th>Question</th>
<th>Answer</th>
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<tbody>
<tr>
<td>What are the three most common organisms involved with human bites?</td>
<td><em>S. aureus; Streptococcus; Fusibacterium</em></td>
</tr>
<tr>
<td>What are some important points to keep in mind about the wound care of human bites?</td>
<td>Inspect the wound carefully (i.e., tooth); The surrounding skin cleansed thoroughly; X-ray hand to rule out fractures and fracture belt (FB)</td>
</tr>
</tbody>
</table>
### What particular area of the body should be left open initially?
- Hands

### What areas of the body can typically be sutured?
- Face, head, and neck

### What are some high-risk features where antibiotics may be indicated?
- Immunocompromised patient; Area with poor blood supply; Hand wounds

### What are some commonly used antibiotic regiments?
- Second or third-generation cephalosporin; Macrolide with clindamycin or TMP-SMX

### What are some indications for the use of IV antibiotics?
- Obvious infection (older wound); Tendon, bone, and joint space involvement; Patient showing signs of systemic infection

### What are some other issues to consider in human bites?
- Tetanus; HIV (although rarely transmissible)

### Snakes

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<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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| What are two families of snakes that account for the majority of venomous snake bites in the United States? | 1. Elapidae family (i.e., coral snakes)  
2. Crotalidae family (i.e., rattlesnake) |
| What are some important points in the field management of snake bites?  | Injured area should be immobilized and raised above the heart; Thoroughly clean the wound; Attempt to identify the snake; Immediate transportation to hospital |
| What are some characteristics of venomous rattlesnakes?                 | Triangular head; Elliptical pupils; Retractable fangs                   |
| What are some characteristics of venomous coral snakes?                | Their banding pattern “red and yellow; kill a fellow...red and black; friend of jack” |
| What are some methods not recommended for snake bites (were commonly used)? | Mechanical suction devices; Incision and oral suction; Tourniquet; Ice |
| About what percentage of venomous snake bites result in significant envenomation? | 3/4 |
| What are some complications of snake bites?                          | Coagulopathy; Thrombocytopenia; Local tissue damage; Neurotoxicity (i.e., respiratory depression) |
| What are some other issues to consider as with all bites?             | Update tetanus status; Snake venom is sterile |
What are some commonly encountered snakes within the *Crotalidae* family? Copperhead; Rattlesnakes; Cottonmouth

What are the most prominent clinical features of bites from within the *Crotalidae* family? Local tissue destruction and swelling; Thrombocytopenia; Coagulopathy

What is the treatment of choice for *Crotalidae* bites? Polyvalent *Crotalidae* ovine immune Fab (i.e., Crofab)

Should all patients with bites from snakes within the *Crotalidae* family be admitted? Patients with no signs of envenomations and normal laboratory values can usually be discharged after 10–12 hours

What is the most prominent clinical features of bites from within the *Elapidae* family? Neurotoxicity (i.e., total paralysis)

What are the earliest clinical features of envenomation from within the *Elapidae* family? Cranial nerve dysfunction; Mental status change

What is the most feared complication of bites from within the *Elapidae* family? Respiratory arrest

What is the treatment of choice for *Elapidae* envenomation? Horse serum based antivenin

What is the most frequent reaction for both antivenins? Serum sickness (delayed up to 2 weeks), but it is far more common in horse-based antivenin

How many hours can the effects of *Elapidae* envenomation be delayed up to? 12 hours

Should all patients with bites from within the *Elapidae* family be admitted? Yes

Rabies

What is rabies? It is a viral infection of the CNS with an incubation period of up to 2 months that is transmitted via the saliva of infected animals

What is the causative agent of rabies? Rhabdovirus

What is the most common source of rabies? Wildlife animals (i.e., bats), not very common in domestic animals

What wildlife animals are major reservoirs of rabies? Raccoon; Skunk
What are some clinical features in each of the following stage of rabies:

**Incubation**
- Incubation from 1–3 months; Can incubate for up to 7 years; Bites closer to brain progress faster

**Prodrome**
- Nonspecific flu-like symptoms; Ranges from a few days to a week; May get pain and pruritus at bite site

**Acute neurologic syndrome**
- Encephalitic or paralytic presentation; Lasts for 2–7 days

**Coma and death**
- Generalized flaccid paralysis; Respiratory and vascular collapse; Most die within 2 weeks once coma sets in

**How many patients with rabies who have not received the rabies vaccine survive?**
- Only one has ever survived

**What are some clinical features of encephalitic involvement?**
- Persistent fever; Painful pharyngeal or inspiratory spasms; Seizures; Hyperactivity

**What are some basic wound care management issues?**
- Thoroughly clean the wound; Tetanus prophylaxis if needed; Rabies vaccine as indicated

**What are things to know about vaccine selection for rabies?**
- Active immunization for bites from animals in a suspected group with HDCV; Passive immunization with IG for bites from animals with rabies with HRIG; Typically both will be used postexposure

**What should be done with the wild animal that bit the patient, if captured?**
- Sacrificed and tested

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**TETANUS**

**What is the pathogen responsible for tetanus?**
- *Clostridium tetani*

**Can the pathogen enter healthy tissue?**
- No—it requires an anaerobic environment such as a wound

**What is the pathophysiology of tetanus?**
- Toxins are released after the spores convert to vegetative forms in an anaerobic environment, which prevents release of inhibitory hormones (spinal cord) and results in generalized spasms
What are some important risk factors for the development of tetanus? Devitalized tissue; Any injury with inoculation of the spores; Coinfection with other bacteria; A foreign body

Although rare in developed countries, what are some high-risk groups for tetanus? Elderly; Intravenous drug abusers (IVDA); Patients with dental infections; Diabetic patients with infected ulcers

What is the incubation period for tetanus? Ranges from a few days to months

What are some forms of tetanus? Generalized—most common form; Neonatal; Local; Cephalic

What is the common presenting symptom of tetanus? Trismus (“lockjaw”)

What are some other clinical features of tetanus? Tonic and periodic muscular spasms that are generalized and often result in periods of apnea with no impairment of consciousness

What is local tetanus? Tonic/spasmic muscular contraction that is confined to one extremity or region that often progresses to generalized form

What is cephalic tetanus? Typically in patients with head injuries involvement of the cranial nerves, usually the facial nerve and often progress to generalized form

What is neonatal tetanus? Common cause of neonatal death in developing countries due to aseptic handling of umbilical stump which leads to spasms, seizures, and death

What are some other important diagnosis to consider in patients who present with generalized spasms? Infections (i.e., meningitis); Hypocalcemic tetany; Dystonic reactions to neuroleptics; Drug withdrawal (i.e., narcotics); Strychnine toxicity

What are some key points in the management of tetanus? ABCs—esp airway; Spasms can be managed with benzos; Surgical debridement for wounds; Give human tetanus immune globulin

What are some wounds that predispose to tetanus? Burns; Penetrating wound; Contaminated wounds
**INSECT BITES**

| What are the three major concerns for any insect bite or sting? | 1. Anaphylaxis  
2. Upper airway obstruction  
3. Toxic reactions from multiple stings |
<table>
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</thead>
<tbody>
<tr>
<td>What is the most common reaction from insect bites?</td>
<td>Local inflammatory reaction</td>
</tr>
<tr>
<td>What are some common insects that bite/sting humans?</td>
<td>Ants; Bees; Wasps; Spiders</td>
</tr>
</tbody>
</table>
| Name three insects that are commonly associated with systemic allergic reactions? | 1. Horseflies  
2. Blackflies  
3. Deerflies |

**Spider Bites**

<table>
<thead>
<tr>
<th>How many poisonous species of spiders are there in the United States?</th>
<th>Over 2500</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does this mean all spiders are dangerous?</td>
<td>No—most are either too small or are unable to penetrate skin</td>
</tr>
<tr>
<td>What are the most dangerous species of spiders in the United States?</td>
<td>Loxosceles species (Brown spiders); Latrodectus species (Black widow); Agelenidae and Atrax species</td>
</tr>
<tr>
<td>What type of spiders are becoming popular as pets and although rarely bite, have a bad reputation as being aggressive?</td>
<td>Tarantulas</td>
</tr>
<tr>
<td>Are Tarantula bites poisonous?</td>
<td>No—although they do have uticarial hair that can induce local reactions/anaphylaxis</td>
</tr>
</tbody>
</table>
| What are three reactions to spider bites? | 1. Local reactions  
2. Systemic reaction  
3. Allergic reaction |
| What are some clinical features of local reactions to spiders bites? | Commonly have fang markings with redness with no blisters if it is non-necrotizing and will last about a week |
What is the primary feature of necrotizing wounds from spiders bites?

Characterized initially by redness which expands upto 14 cm, followed by a blister which forms within a day and ulcers that can leave necrotic tissue

What are some species of spiders that characteristically cause necrotic lesions?

Loxosceles species (brown spiders); Chiracanthium

What are some clinical features of systemic reactions to spider bites?

Myalgias, fever, fatigue, and can rarely cause hemolysis and coagulopathy

What are some important things to know about black widow spiders?

Located in warm regions of the earth; The female is far more poisonous; The poison is a potent neurotoxin

What are some clinical features of a local reaction from a black widow spider bite?

Dull muscle crampings that often wax and wane, chest pain (due to radiation from upper extremity), rectal spasms, and can even mimic an acute abdomen

What are some clinical features of a systemic reaction to a black widow spider bite?

Primarily neurologic: hypertension, diffuse pain, tachycardia, profuse sweating, and difficulty in speaking

What is latrodectism?

It is a systemic reaction to black widow spider bites that results in nausea, emesis, and severe spasm that can result in death

What are some key points in the management of black widow spider bites?

Thoroughly clean wound with soap/water; Tetanus status update; Observation for 4 hours if a black widow; Narcotics for pain is the mainstay; Benzos for severe muscle spasms; Nitroprusside for severe hypertension

What role does antivenin play in the treatment of black widow spider bites?

Not commonly used—bites from black widows are rarely fatal

What are some indications for the use of antivenin in black widow spider bites?

Patients with refractory pain; Pregnant; Severe hypertension

What are some important things to know about brown spiders?

Live in human dwellings; Hide during the day in various spots (cracks); Distributed in the Midwest and south central region of the United States

What is viscerocutaneous loxoscelism?

It is a systemic response to a brown recluse spider bite that results in severe intravascular hemolytic syndrome
What is the feared complication of brown spider bites?  
Local tissue necrosis that may require surgical correction

What are some key points in the management of brown spider bites?  
Thoroughly clean wound with soap/water; Tetanus status updated; Use ice to help decrease inflammation; Consider use of Dapsone to treat local effects—although used historically

### Tick Bites

<table>
<thead>
<tr>
<th>What are some important tick-transmitted human diseases?</th>
<th>Lyme disease; Rocky Mountain spotted fever; Babesiosis; Ehrlichiae; Relapsing fever</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the infecting organism in Rocky Mountain spotted fever (RMSF)?</td>
<td><em>Rickettsia Rickettsii</em></td>
</tr>
<tr>
<td>What is the tick that commonly spreads <em>R. Rickettsii</em>?</td>
<td>Female <em>Dermacentor</em> tick</td>
</tr>
<tr>
<td>What is the incubation period of RMSF?</td>
<td>A couple of days to 2 weeks</td>
</tr>
<tr>
<td>What is the peak incidence of RMSF?</td>
<td>In the spring and summer with young children being most commonly infected</td>
</tr>
<tr>
<td>Is the disease most commonly reported in the rocky mountain region (i.e., Montana)?</td>
<td>No—more common in east region (i.e., Virginia)</td>
</tr>
<tr>
<td>What is clinical hallmark of RMSF?</td>
<td>Rash—an erythematous blanching rash with 2–4 mm macules that appear initially on the flexor surface of wrist/ankles that spreads to palm/soles, which then moves centrally</td>
</tr>
<tr>
<td>What are some other clinical features of RMSF?</td>
<td>Fever (usually high grade &gt;39°C), severe headaches, myalgias, and GI symptoms which are then typically followed by the hallmark rash</td>
</tr>
<tr>
<td>What is the diagnostic test of choice for RMSF?</td>
<td>It is a clinical diagnosis (fever, headache, and rash in spring/summer) and if you suspect, initiate treatment</td>
</tr>
<tr>
<td>Are there tests you can use to detect RMSF?</td>
<td>Serology (typically negative early on); Skin biopsy; Indirect fluorescent antibody</td>
</tr>
<tr>
<td>What is the most common cause of fatality in RMSF (although deaths are rare)?</td>
<td>Delayed treatment</td>
</tr>
</tbody>
</table>
List three antibiotics commonly used to treat RMSF?  

1. Doxycycline  
2. Tetracycline  
3. Chloramphenicol

What is the concern when using tetracycline antibiotics?  

Teeth staining in younger children

What are some important adverse reactions to keep in mind about chloramphenicol?  

Aplastic anemia; Bone marrow suppression; Gray baby syndrome

What are some indications for the use of chloramphenicol?  

Pregnancy; Children <8 years; Severe illness

What is the most common tick-borne illness in the United States?  

Lyme disease

What is the organism responsible for Lyme disease?  

*Borrelia burgdorferi*

What is the tick that harbors this organism?  

*Ixodes* tick

What is the peak incidence of Lyme disease?  

Spring and summer

What are the three phases of Lyme disease?  

1. Localized  
2. Dissemination  
3. Chronic infection

What is the hallmark of early localized disease?  

Erythema migrans (EM)

What are some other clinical features of localized disease?  

Usually occurs within a month, will develop fever, myalgia, headache, malaise, and fatigue with EM (can be absent in up to 20% of cases)

What are some clinical features of disseminated disease?  

Occur days-months after a tick bite that can be characterized by conjunctivitis, myocarditis, varying degrees of AV block, and neurologic abnormalities being the predominate feature

What are some common neurologic abnormalities of disseminated disease?  

Cranial neuropathy (Bell’s palsy common); Peripheral neuropathy; Meningoencephalitis

What are some clinical features of chronic disease?  

Occurs months-years after tick bite; where musculoskeletal complaints most common, peripheral neuropathy, encephalopathy, and neurocognitive dysfunction also can occur

What is the most important diagnostic test for Lyme disease?  

Clinical suspicion is the most important
What are some tests that can be done for Lyme disease?
Serology testing; CSF

What are some commonly used antibiotics for early Lyme disease?
Doxycycline; Amoxicillin; Clarithromycin

What is the treatment for severe CNS manifestations or carditis of Lyme disease?
Ceftriaxone or penicillin

**CLINICAL VIGNETTES**

24-year-old male who works as a cook presents with a burn to the left hand from spilling soup on it; PE: skin is red and painful with blisters
Second degree burn

32-year-old male is brought in unconscious by EMS from a frozen lake, patient is unresponsive and does not have evidence of falling via the lake; ECG: Osborn waves and prolongation of all intervals
Hypothermia

18-year-old healthy male complains of numbness, leg cramps, and paresthesias of lower extremities a few hours after hiking for about 4 hours via rivers; PE: unremarkable except for feet that are pale and insensitive to touch
Immersion foot (i.e., trench foot)

18-year-old male presents with complaints of pain on the back of his skin, he mentions he was tanning the day before; PE: skin on the back is red and tender to touch, but does not have blisters
First degree burn

76-year-old with a recent history of head surgery now comes to the ER complaining of facial spasms and inability to open her jaw, but otherwise no other complaints; PE: unremarkable except for trismus of the jaw
Cephalic tetanus

12-year-old male presents with a severe headache and high fever, patient mentions he developed these symptoms about a week ago with a rash developing yesterday; PE: erythematous blanching rash on flexor surface of wrists/ankles
Rocky Mountain spotted fever

Serving as the team physician on a mountain expedition, what is the most likely diagnosis based on the symptoms for each of the following members:
Four days into the ascend, a member complains of increasing dyspnea, fatigue, headache, cough; PE: rales and cyanosis

A few days into the ascend, a member is complaining of a bad headache, which is worse in the morning and has had trouble sleeping

A member is beginning to display odd behavior, seeing things that are not there and often acting confused

7-year-old child is brought in by her mother due to a dog bite to the hand about an hour ago. Patient is otherwise doing well with no other complaints; PE: normal exam of the hand; x-ray of hand: no foreign body or air; should you suture the hand?

42-year-old construction worker is brought in by a coworker in severe spasms of his entire body and screaming out in pain; PE: most noticeable for stiffness of the jaw (trismus) and evidence of an old puncture wound on his left foot

35-year-old male is brought into the ED via EMS for profuse sweating along with nausea while he was jogging at the beach several hours ago; PE: tachycardia, hypotension, normal temperature

34-year-old female is brought in by her husband with a week’s history of the “flu” but now is having periods of confusion and hallucinations. Husband mentions that the “flu” started soon after coming back from camping; PE: only remarkable for an old bite near the left calf

21-year-old female presents in distress with drooping of her eyelid and the corner of her mouth, she only recalls a brief bout of the “flu” and a funny rash a week after camping; PE: Bell’s palsy; ECG: first degree AV block

High-altitude pulmonary edema (HAPE)
Acute-mountain sickness (AMS)
High-altitude cerebral edema (HACE)
No—all hand wounds should be left open initially
Generalized tetanus
Heat exhaustion
Rabies (excitement phase)
Lyme disease
34-year-old diver comes in to the ED complaining of dizziness and extreme nausea whenever he stands up, he mentions this occurred after a dive where he underwent a rapid descent

17-year-old female presents with severe muscle cramps in her calves during track practice in hot and humid weather; PE: normal vitals

Barotitis interna
Heat cramps
GENERAL APPROACH

What is the first course of action for any patient who presents with suspected poisoning? Airway, breathing, circulation (ABC)

What are some other actions to take once ABCs have been established? O₂ saturation for hypoxia; Finger stick for glucose; Assess vitals; Accurate history

What two organ systems should the exam concentrate on? 1. Cardiovascular system (CVS) 2. Central nervous system (CNS)

Why concentrate on the CNS and CVS during the exam? The most lethal adverse affects of toxicological ingestion typically affect the CVS and CNS

What are some interventions for any poisoned person? Treatment is primarily supportive; Always consider other ingestions; Call poison center for recommendations

What is gastric decontamination? The use of various techniques to either remove the toxin or expedite passage through the GI tract to limit absorption


Are gastric decontamination methods routinely used in acute poisonings? No—while historically commonly used, gastric decontamination is now used in select cases
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<tbody>
<tr>
<td>What are important things to know about ipecac?</td>
<td>Derived from plant alkaloids; Single dose produces emesis in over 90% of patients; Emesis typically occurs around 30 minutes</td>
</tr>
<tr>
<td>What are some indications for the use of ipecac?</td>
<td>Considered where AC binds poorly to toxins; In acute ingestions (&lt;1 hour); If removal of small amount has significant impact on outcome; Patient should have intact gag reflex</td>
</tr>
<tr>
<td>What are some contraindications of ipecac?</td>
<td>Prior significant emesis; Avoid if unconsciousness/altered mental state; Nontoxic ingestions; Avoid if ingested caustic substances</td>
</tr>
<tr>
<td>What are some complications with the use of ipecac?</td>
<td>Aspiration; Lethargy; Mallory-Weiss tear; Intractable emesis</td>
</tr>
<tr>
<td>What is gastric lavage?</td>
<td>Orogastric lavage with a large-bore tube to lavage with adequate volumes until clear while removing any remaining toxins</td>
</tr>
<tr>
<td>What are some indications for the use of gastric lavage?</td>
<td>Consider if ingestion occurs within an hour; Preferred for patients who have no gag; Consider where a rapid deterioration is expected (i.e., TCAs)</td>
</tr>
<tr>
<td>What are some contraindications for the use of gastric lavage?</td>
<td>Any caustic ingestions; If drug is most likely not in the stomach; Any large foreign bodies or sharp objects</td>
</tr>
<tr>
<td>What are some complications of gastric lavage?</td>
<td>Aspiration; Esophageal/gastric perforation; Tension pneumothorax</td>
</tr>
<tr>
<td>What is activated charcoal (AC)?</td>
<td>Fine black powder produced by burning carbonaceous material that will result in a substance in a huge surface area to bind many substances</td>
</tr>
<tr>
<td>While AC will bind many substances, what are some substances that AC does not bind well?</td>
<td>Strong acids and bases; Metals (i.e., iron and lithium); Alcohols</td>
</tr>
<tr>
<td>What substances does multiple-dose activated charcoal (MDAC) prove effective in?</td>
<td>Theophylline; Digoxin; Phenytoin; Carbamazepine</td>
</tr>
<tr>
<td>What are some contraindications of AC?</td>
<td>Any perforation; Loss of airway reflex</td>
</tr>
<tr>
<td>What are some complications of AC?</td>
<td>Small bowel obstruction (very rare); Aspiration</td>
</tr>
</tbody>
</table>
What is whole bowel irrigation? Use of large volumes of fluid to cleanse the entire GI tract that will clear most matter (i.e., toxin) within a few hours.

What substance is commonly used in whole bowel irrigation? Polyethylene glycol (PEG).

What are some indications for whole bowel irrigation? Toxic substance not well absorbed by AC; Toxins with prolonged absorption; GI drug concealment.

What are some contraindications of whole bowel irrigation? Bowel obstruction and perforation; Hemodynamic instability; Evidence of no bowel activity.

What are some toxins where hemodialysis (HD) is commonly indicated in severe cases? MEAL Methanol; Ethylene glycol; Aspirin; Lithium.

What are some indications where HD should be considered? Sign of end-organ damage; Absolute level; Inability to metabolize.

What are some complications of HD? Blood loss; Hypotension; Coagulopathy from heparin; Decrease in platelets.

What is urinary alkalinization? A method of enhanced elimination by alkalinization of urine (via bicarb) to enhance ion trapping and elimination via urine.

What are some substances where urine alkalinization is indicated? Aspirin; Chlorpropamide; Methotrexate; Phenobarbital.

**OVER-THE-COUNTER DRUGS**

**Acetaminophen**

What are some important things to know about acetaminophen (APAP)? APAP is found in over 100 drug preparations; Leading cause of liver failure requiring transplantation; Leading drug involved in ingestion.

What is the normal metabolism of APAP? >90% conjugated to glucuronide/sulfate conjugates (eliminated by kidney after); 2% excreted by kidney unchanged; 5% oxidized to \( N \)-acetyl-\( \text{para} \)-benzoquinoneimine (NAPQI).

What is the primary toxic metabolite of APAP that is responsible for liver necrosis? NAPQI.
What is the body’s method to detoxify NAPQI under normal circumstances?

Glutathione binds to NAPQI preventing hepatocyte necrosis

What happens when there is an APAP overdose?

Conjugation and sulfation pathways are saturated which means more NAPQI is produced and overwhelms glutathione stores

What is the toxic dose of APAP in acute setting?

150 mg/kg (7.5 g) in an adult (24 hours)

What is the time course of APAP toxicity:

- **Phase 1 (0–24 hours)**: Anorexia, nausea, emesis, and elevated transaminases
- **Phase 2 (24–72 hours)**: Right upper quadrant (RUQ) pain, bilirubin and PT elevate, and transaminases begin to peak
- **Phase 3 (72–96 hours)**: Hepatic necrosis begins (may get encephalopathy, jaundice, and death)
- **Phase 4 (96 hours–2 weeks)**: Healing of liver if acute fulminant liver failure did not occur in phase 3

What is the Rumack-Matthew normogram?

Predicts the risk of toxicity assuming a one time ingestion with complete absorption

Based on the normogram, what is the cut-off level in deciding to treat or not?

150 mg/L (in the United States) at 4 hours

What are some limitations of applying the normogram to APAP overdose?

Does not apply to multiple ingestions; Not applicable to chronic ingestions; Typically will end up over treating

What is the antidote for APAP toxicity?

*N*-acetylcysteine (NAC)

What is the mechanism by which NAC works?

Precursor to cysteine then to glutathione; Enhance sulfation of APAP; Can act as free radical scavenger; Glutathione substitute

When is the optimal time to give NAC following APAP overdose?

Within 8 hours (100% protective)

How is NAC administered?

Oral; IV (if difficult to ingest due to smell/taste)

What are adverse reactions to IV NAC?

Anaphylactoid reaction; Hypotension and death (very rare); Elevates PT
What are some poor prognostic factors after APAP overdose?

pH <7.30; Creatinine >3.3; Grade III/IV encephalopathy

Salicylates

What are some of the therapeutic properties of aspirin (ASA)?
Antipyretic; Analgesic; Anti-inflammatory

What are some important things to know about ASA?
A significant source of poisoning; ASA can produce substantial toxicity/death; There are more than 200 products with ASA

What are some sources of ASA?
Oil of wintergreen; Arthritis/decongestants/cold preparations; Keratolytics; Pepto-Bismol

What is the toxic level of ingestion for acute ASA poisoning?
300-400 mg/kg produces serious toxicity; 100 mg/kg/day for over 2 days will produce chronic toxicity

<table>
<thead>
<tr>
<th>Serum Salicylate Level</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 mg/dL</td>
<td>Moderate toxicity</td>
</tr>
<tr>
<td>75 mg/dL</td>
<td>Severe toxicity</td>
</tr>
<tr>
<td>100 mg/dL</td>
<td>Potentially lethal</td>
</tr>
</tbody>
</table>

Do symptoms correlate well with serum levels?
Symptoms correlate better with CSF levels, treatment should be based on clinical picture

What are the two primary acid-base disturbances of ASA toxicity?
1. Respiratory alkalosis
2. Metabolic acidosis

What is the mechanism by which ASA toxicity occurs?
Uncouples ox-phos to produce fever; Stimulates respiratory drive for tachypnea; Directly causes metabolic acidosis; Acidosis will increase the Vd

What are some clinical features of acute ASA toxicity?
Primarily GI: nausea, vomiting, tinnitus, agitation, delirium, seizure, and coma

What are some clinical features of chronic ASA toxicity?
Nonspecific: altered mental status, lethargy, dehydration, and metabolic acidosis
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the primary way in which death occurs in ASA overdose?</td>
<td>CNS overstimulation (seizure/hyperthermia); Cardiovascular collapse; Pulmonary edema</td>
</tr>
<tr>
<td>What are some important diagnostic tests to consider in ASA overdose?</td>
<td>Serum ASA level (serial levels more useful); ABG (for acid-base disturbances); Potassium; Renal function</td>
</tr>
<tr>
<td>Is there any use for AC in ASA overdose?</td>
<td>Yes—AC binds ASA well</td>
</tr>
<tr>
<td>What are some key points in the management of ASA overdose?</td>
<td>ABCs is the first priority; Care is primarily supportive; Aggressive rehydration; Sodium bicarbonate for acidosis</td>
</tr>
</tbody>
</table>
What three organ systems are most affected by iron overdose (primarily from free-radical production)?

1. GI epithelium
2. Heart
3. Liver

What is the most common cause of death in iron overdose?

Circulatory shock

What are the four phases of iron toxicity:

Phase 1: GI (0–12 hours)
Direct injury to the GI mucosa: abdominal pain, diarrhea, emesis, hematemesis, etc.; Severity ranges from mild to shock

Phase 2: Latent (6–24 hours)
Period of apparent recovery. Patients in this phase are usually stable, but they are not asymptomatic. Risk of developing life-threatening hypovolemia and acidosis

Phase 3: Metabolic phase (24 hours–4 days)
Clinical manifestations of the metabolic phase include fever, pallor, cyanosis, jaundice, renal failure, lethargy, coma, shock, and bleeding. Potential for death is highest here

Phase 4: Delayed phase (2–8 weeks)
Characterized by late complications, usually intestinal scarring with GI obstruction

What are important serum iron concentrations to be aware of?

1. 50–150 µg/dL
2. 350 µg/dL
3. 500 µg/dL
4. >1000 µg/dL

Normal levels
Risk for toxicity
Significant toxicity likely
Considerable morbidity

What are some laboratory tests to consider?

CBC/Chem-7/Coags; ABG for moderate-severe cases; Iron studies (i.e., TIBC, Fe, etc.)

What role does an abdominal radiograph (KUB) play in iron toxicity?

While a KUB may be able to detect opacities (Fe) on film, its absence does not rule out ingestion

Is gastric decontamination effective with iron overdose?

Gastric lavage, ipecac, and AC relatively ineffective with iron ingestion

What is the antidote commonly used for iron toxicity?

Deferoxamine (DFO)
**What are some functions of DFO?**

Chelation of iron; DFO can remove iron bound to transferrin; DFO can also remove iron from cells

**When is the general serum iron level in which to administer DFO?**

Generally 500 ug/mL or greater

**What are some adverse reactions with administration of DFO?**

Acute renal failure; Septicemia from *Y. enterocolitica*; ARDS; Hypotension

### PRESCRIPTION MEDICATIONS

#### Anticoagulants

**What are the two main categories of anticoagulants and some examples of each:**

<table>
<thead>
<tr>
<th>Indanedione anticoagulants</th>
<th>Pindone; Diphacinone; Valone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydroxycoumarin anticoagulants</td>
<td>Brodifacoum; Warfarin; Fumarin</td>
</tr>
</tbody>
</table>

**What are some scenarios where overdose of anticoagulants can occur?**

Accidental ingestion by children; Drug interactions; Suicidal ingestion; Homicidal attempts (i.e., rat poison)

**What is the mechanism of action of warfarin?**

Inhibits the synthesis of vitamin K-dependent factors (II, VII, IX, X, and protein C and S), so that once the existing factors degrade, no more is made

**What are common sites of bleeding with anticoagulant overdose?**

GI tract and genitourinary tract; Epistaxis and hemoptysis can be common

**What is the most feared complication of anticoagulant overdose?**

Intracranial bleeding

**What are the typical abnormal labs with anticoagulants overdose?**

Elevated PT/PTT time; Platelets and LFT are usually normal

**What drugs interaction typically lead to excessive anticoagulation?**

Cimetidine, erythromycin, metronidazole, and ciprofloxacin typically lead to excess anticoagulation

**What are some distinguishing features of superwarfarins?**

Very long-acting anticoagulants; Half-life that exceed 3–4 months; Vitamin K therapy may require months; Typically only found in rat poison

**What are some key points in the management of accidental ingestion?**

If asymptomatic, typically observe; Coags/GI decontamination not needed; Advise to watch for any signs of bleeding
What are some key points in the management of intentional ingestion? Careful monitoring, especially if active bleeding; ABCs—active bleeding can obstruct airway; CBC and coag should be done serially; Know if patient needs to be anticoagulated.

What are some treatment options for a patient who is actively bleeding from anticoagulants? For severe bleeding: FFP or whole blood; Most other cases: vitamin K.

How often should PT be monitored? Initially every 6–8 hours, PT takes days to normalize.

What are some routes of vitamin K administration? Oral, IM, or IV.

What are adverse reactions of giving IV vitamin K? Anaphylactoid reaction (rare); Cerebral thrombosis.

What is the mechanism of action for unfractionated heparin (UFH)? Inhibits ATIII that results in prolonged PTT.

What are some adverse reactions of UFH? Heparin-induced thrombocytopenia (HIT); Hyperkalemia (inhibits aldosterone).

What are low-molecular-weight heparins (LMWH)? Derivatives of commercial heparin, LMWH inactivate factor Xa, but have a lesser effect on thrombin.


What are some advantages of LMWH over UFH? Longer duration of action; Laboratory monitoring is not necessary; They are much less likely to induce HIT; LMWH can be given outpatient.

What is the treatment of choice for heparin overdose? Discontinue heparin as it has a very short half-life. Protamine sulfate can be given for serious bleeding as a result of heparin or LMWH.

**Oral Hypoglycemics**

What are some important points regarding maintenance of plasma glucose levels? Normally maintained between 70–150 mg/dL; Glycogenolysis/gluconeogenesis help maintain normal levels; Adult liver has 70 grams of glycogen.
What is important to know about the brain and its use of glucose? Uses about 60% of glucose; First organ to be affected by hypoglycemia; Hypoglycemia will activate sympathetic axis

What are clinical features of hypoglycemia? Diaphoresis, tachycardia, tremor, altered mental status, seizure, coma, and rarely focal neurologic deficits that mimic TIAs

What is the general principle for initial treatment for all hypoglycemic agents? Give dextrose then feed the patient

Name some commonly used oral agents in the treatment of non-insulin-dependent diabetes mellitus (NIDDM)? Sulfonylurea; Alpha-glucosidase inhibitors; Thiazolidinediones Biguanides

What are some commonly used sulfonylureas? Glyburide; Glipizide; Tolazamide

What is the primary mechanism of action of sulfonylureas? They cause insulin release from remaining pancreatic cells via cell depolarization and also improve sensitivity to insulin

How soon can hypoglycemia occur after the ingestion of a sulfonylurea? Can vary anywhere from 30 minutes to many hours after ingestion. Administration of dextrose can mask hypoglycemia

What are some key points in the management of hypoglycemia secondary to sulfonylureas? Carbohydrate-rich meal for awake patients; 50% dextrose for patients with altered MS; Glucagon is not effective in hypoglycemia; Observe for at least 8–12 hours with frequent accuchecks

What are two agents available for refractory hypoglycemia secondary to sulfonylureas? 1. Diazoxide 2. Octreotide (main treatment)

What are some important things to know about diazoxide? Inhibits insulin secretion; Causes hypotension and hyponatremia; Can cause sodium and fluid retention

What are some important things to know about octreotide? Somatostatin analogue; More effective then diazoxide; Inhibits secretion of insulin; Generally very well tolerated

What are some key points in the management of hypoglycemia from sulfonylureas? A single ingestion by child = admission; Interactions may enhance toxicity; Any patients who present hypoglycemic = admission for observation
What are some other techniques to prevent absorption and enhance elimination? Activated charcoal

What is the mechanism of action of biguanides (i.e., metformin)? Decreases hepatic gluconeogenesis; Increases uptake of glucose; Increases utilization of glucose into lactate

What adverse effect is particularly important to monitor with metformin? Lactic acidosis

Who are at increased risk of lactic acidosis secondary to metformin use? Patients with impaired renal clearance

What is the treatment of lactic acidosis due to metformin use? Treatment is supportive with correction of acid-base disturbance and rehydration

Does metformin cause hypoglycemia? Rarely

### Cardiac Glycosides

<table>
<thead>
<tr>
<th>Onset</th>
<th>Time to Peak Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>1.5–6 hour</td>
</tr>
<tr>
<td>IV</td>
<td>5–20 minutes</td>
</tr>
</tbody>
</table>

What are cardiac glycosides? Drugs with a steroid ring, one-four sugars attached to them, and unsaturated lactone ring

What are the primary indications for digoxin? CHF; Control of rapid ventricular response from afib and aflutter

What are some sources of cardiac glycosides? Foxglove; *Bufo* toads; Milkweeds; Oleander

What is the mechanism of action of cardiac glycosides? Inhibit sodium-potassium exchange pump that will increase intracellular calcium

What are some effects of cardiac glycosides? Increased vagal tone; Increased automaticity; Increased contractility

What is the time course for toxicity to develop after an overdose? Drugs must first move into cells, symptoms generally do not occur for several hours
**What are some clinical features of acute overdose?**
Nausea and emesis are almost first symptoms with confusion and weakness, can also develop heart block/bradycardia

**What are some clinical features of chronic overdose?**
Anorexia, nausea, and emesis common with headaches, confusion, and lethargy

**Which patients commonly present with chronic digoxin toxicity?**
Commonly an elderly person with underlying heart disease who presents with nonspecific GI/neuro complaints (usually with precipitating factor such as dehydration)

**How does chronic toxicity commonly develop?**
Drug interactions that increase levels; Worsening renal function; Diuretics or infection that lead to dehydration

**What are the some possible ECG findings in digoxin overdose?**
Prolonged PR interval; Short Q-T; ST scooping and depression (esp. laterally); Decreased T-waves

**What is another feared complication of digoxin overdose?**
Life-threatening hyperkalemia

**What are some key points in the management of acute digoxin overdose?**
AC may be considered; HD is not effective; Follow potassium closely—treat accordingly; Avoid the use of calcium—greater arrhythmias; Treat any dysrhythmias accordingly, but avoid type IA/IC antiarrhythmics

**What is the standard treatment for acute digoxin overdose?**
Digoxin-specific Fab antibody fragment (Digibind)

**What are some indications for the use of digibind?**
Serious dysrhythmias; Bradycardia refractory to atropine; Hyperkalemia (>5.5)

## Beta-Blockers

**What are indications for the use of beta-blockers?**
Hypertension; Prevent reinfarction and s/p MI; Dysrhythmias; Glaucoma; Migraine headaches

**What are some important things to know about beta-blockers?**
There are many preparations; Agents may be selective or nonselective; With overdose, selectivity is loss

**What are some commonly used beta-blockers?**
Metoprolol; Carvedilol; Labetalol; Timolol
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the function of B₁ receptors?</td>
<td>Heart (increase HR/inotrophy/automaticity); Eye (increase aqueous humor production); Kidney (increase renin production)</td>
</tr>
<tr>
<td>What is the function of B₂ receptors?</td>
<td>Liver (gluconeogenesis); Smooth muscle relaxation; Skeletal muscle (glycogenolysis)</td>
</tr>
<tr>
<td>What is the function of B₃ receptors?</td>
<td>Adipose tissue (lipolysis)</td>
</tr>
<tr>
<td>How soon after ingestion of beta-blockers do patients manifest symptoms?</td>
<td>Usually within 6 hours</td>
</tr>
<tr>
<td>What are some important clinical manifestation based on systems?</td>
<td>Bradycardia, hypotension, CHF, QRS/QT prolongation (rare)</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td></td>
</tr>
<tr>
<td>Respiratory system</td>
<td>Apnea, respiratory depression, and bronchospasms</td>
</tr>
<tr>
<td>CNS</td>
<td>Seizure, delirium, and coma (mostly in the setting of hypotension)</td>
</tr>
<tr>
<td>Endocrine system</td>
<td>Children are particularly susceptible to hypoglycemia</td>
</tr>
<tr>
<td>What type of beta-blockers are considered the most dangerous?</td>
<td>Ones that are lipophilic, have sodium channel activity, and have potassium channel activity</td>
</tr>
<tr>
<td>Which beta-blocker causes a disproportionate amount of deaths?</td>
<td>Propranolol</td>
</tr>
<tr>
<td>What are some basic therapeutic measures for overdose with mild symptoms?</td>
<td>Supportive care with fluids; Cardiac monitoring</td>
</tr>
<tr>
<td>What are some key points in the management of beta-blocker overdose for moderate-severe sick patients?</td>
<td>ABCs; Cardiac monitoring/fluids/atropine; Glucagon is the drug of choice; Catecholamines in severe cases</td>
</tr>
<tr>
<td>What are indications for admission?</td>
<td>History of sustained-release overdose; Children should be admitted; Any symptoms/ECG changes within 6 hours</td>
</tr>
</tbody>
</table>

**Calcium Channel Blockers**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important features of calcium channel blockers (CCB)?</td>
<td>Block slow calcium channels in myocardium and vascular smooth muscle; Decrease myocardial inotrophy/conduction; Vasodilation in peripheral vasculature</td>
</tr>
</tbody>
</table>
What are some indications for the use of calcium channel blockers? Hypertension; Angina; Dysrhythmias; Migraines

What are the three most commonly used CCBs? 1. Diltiazem (benzothiazapine) 2. Verapamil (phenylalklamine) 3. Nifedipine (dihydropyridine)

How soon after ingestion do symptoms of CCBs overdose appear? Depending on the formulation, can range from the first hour to 24 hours

What is the mechanism of death in CCB overdose? Profound cardiogenic shock with peripheral vasodilation

What are some important clinical manifestation based on:

CVS Hypotension, dysrhythmias, bradycardia, and cardiogenic shock

Respiratory system ARDS

CNS Dizziness, seizures, altered MS, and stroke

Endocrine system Hyperglycemia

What are some key points in the management of CCB overdose? Prevent and correct hypotension; ABCs; AC; Bradydysrhythmias treated with advanced life support (ACLS)

What are important therapeutic maneuvers to reverse hypotension? IVF bolus; Calcium; Glucagon and catecholamines; High insulin therapy

Which patients can safely be discharged after CCB overdose? Typically those who show no symptoms or ECG changes after 6 hours and did not ingest any sustained-release formulation

PSYCHIATRIC MEDICATIONS

Selective Serotonin Reuptake Inhibitors

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Brand Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citaloprim</td>
<td>Celexa</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>Prozac</td>
</tr>
<tr>
<td>Fluvoxamine</td>
<td>Fluvax</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>Paxil</td>
</tr>
<tr>
<td>Sertraline</td>
<td>Zoloft</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>Effexor</td>
</tr>
</tbody>
</table>
### What are some important things to know about selective serotonin reuptake inhibitors (SSRIs)?

- SSRIs are generally safe; Designed to answer TCAs side effect profile

### What is the mechanism of action of SSRIs?

- They inhibit presynaptic neuronal reuptake of serotonin

### What are some clinical features of SSRIs overdose?

- Nausea, emesis, sedation, lethargy, and rarely seizures

### What is important to know about citalopram?

- Large overdoses can cause seizure and QT prolongation

### What are some key points in the management of SSRI overdose?

- Treatment is primarily supportive; Important to rule out other overdoses

### What are some general indications to medically clear a patient following SSRI overdose?

- Monitor for 6 hours and if no changes, can clear with exception of citalopram and buproprion

### What is serotonin syndrome?

- Excessive stimulation of serotonin receptors typically due to ingestion of serotonergic medication

### What are some mechanisms by which excessive serotonin can occur?

- Prevent breakdown of 5-HT; Enhance 5-HT release (i.e., ecstasy); Block reuptake (i.e., cocaine)

### What are some clinical features of serotonin syndrome?

- Mental status change, hyperreflexia, hyperthermia, agitation, myoclonus, and seizure

### What are some key points in the management of serotonin syndrome?

- Benzodiazepines, cooling, and hydration; Sedation/intubation in refractory cases

### Other Antidepressants

#### What are key points of drug overdose for the following antidepressants:

- **Trazadone and Nefazodone**: Inhibits reuptake of 5-HT; Overdose may cause sedation

- **Amoxapine**: Cyclic antidepressant; Works on DA and NE receptors; High incidence of seizures; Not associated with ECG abnormalities

- **Buproprion**: Prevents reuptake of DA and NE; Indicated for smoking cessation; Can cause seizures
Tricyclic Antidepressants

<table>
<thead>
<tr>
<th>Tricyclic Antidepressants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Imipramine</td>
</tr>
<tr>
<td>Amitriptyline</td>
</tr>
<tr>
<td>Desipramine</td>
</tr>
<tr>
<td>Nortriptyline</td>
</tr>
</tbody>
</table>

What are some important things to know about tricyclic antidepressants (TCAs)?
- Higher frequency of adverse effects;
- Have low therapeutic index;
- Significant sedative/anticholinergic effect

Why are TCAs fairly toxic in overdose?
Primarily due to their nonspecific blockage of reuptake of various neurotransmitters

What are some of the adverse effects when taken in overdose:

- **Anticholinergic**
  - Dry skin, hallucinations, delirium, hyperthermia, tachycardia, and mydriasis

- **Alpha-adrenergic blockage**
  - Peripheral vasodilation with hypotension

- **Sodium channel blockage**
  - Inhibit fast sodium channels (quinidine-like effect) with widened QRS complex

What is a useful diagnostic test to obtain to further evaluate TCA overdose?
ECG

What is the most sensitive indicator of toxicity on ECG?
QRS width

What is the QRS width where seizures and dysrhythmias may occur?
QRS width >120 msec

What are some key points in the management of TCA overdose?
ABCs with cardiac monitoring is crucial; AC should also be given <1 hour

What is the drug of choice for TCA overdose which manifest QRS widening?
Sodium bicarbonate

When is it generally safe to discharge patients from the ED after TCA overdose?
No signs of toxicity and continuous monitoring for 6 hours
Monoamine Oxidase Inhibitors

What are some important things to know about monoamine oxidase inhibitors (MAOIs)?

Were among the first class used for MDD; Were later largely replaced by TCAs; They now have limited indications for use.

What is the mechanism of action of MAOIs?

MAO is an enzyme that breaks down monoamines, so its inhibition will increase the concentration of NE, DA, and 5-HT.

What are some clinical features of MAOI overdose?

Tachycardia, hypertension, agitation, and diaphoresis; may get cardiovascular as well as neurological collapse in severe overdose.

How late can symptoms appear following an MAOI overdose?

Effects can be delayed for over 24 hours.

What are some late complications of MAOI overdose?

DIC, rhabdomyolysis, and pulmonary edema.

What are some key points in the management of MAOI overdose?

ABCs; Promptly treat severe hypertension; ACLS for dysrhythmias which may occur; Aggressive fluid bolus for hypotension.

Can patients be safely discharged after being asymptomatic for 6 hours?

MAOIs are the exception to the “6-hour rule” and should be monitored for at least 24 hours.

What other drugs can interact with MAOIs to produce toxicity?

Any sympathomimetic such as cocaine or dopamine can produce toxicity.

MAOIs are well-known to produce toxicity when ingested with “wine and cheese,” why?

These foods contain tyramine, which acts as an indirect sympathomimetic to precipitate toxicity.

What are some other foods that produce toxicity when ingested with MAOIs?

Aged meat; Soy sauce; Sauerkraut.

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Brand Name</th>
<th>Synonym</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isocarboxazid</td>
<td>Marplan</td>
<td></td>
</tr>
<tr>
<td>Phenelzine</td>
<td>Nardil</td>
<td></td>
</tr>
<tr>
<td>Selegiline</td>
<td>Deprenyl</td>
<td></td>
</tr>
<tr>
<td>Tranylcypromine</td>
<td>Parnate</td>
<td></td>
</tr>
</tbody>
</table>
### NEUROLEPTICS

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are neuroleptics?</td>
<td>Originally known as antipsychotics and tranquilizers, this class of drugs is commonly used for a variety of anxiety and psychotic states</td>
</tr>
<tr>
<td>What are some indications for the use of neuroleptics?</td>
<td>Psychosis; Delirium; Agitation; Nausea</td>
</tr>
<tr>
<td>What are some examples of positive symptoms and the receptor that mediates them?</td>
<td>Mediated primarily by central D&lt;sub&gt;2&lt;/sub&gt; receptor: Delusions; Thought disorders; Hallucinations</td>
</tr>
<tr>
<td>What are some examples of negative symptoms and the receptor that mediates them?</td>
<td>Mediated primarily by 5-HT&lt;sub&gt;2A&lt;/sub&gt; receptor: Apathy; Social withdrawal; Blunted effect</td>
</tr>
<tr>
<td>What are some adverse effects from normal use of neuroleptics?</td>
<td>More common with typical neuroleptics: Acute dystonia; Neuroleptic malignant syndrome; Glucose dysregulation</td>
</tr>
<tr>
<td>What are some common extrapyramidal symptoms seen with neuroleptics?</td>
<td>Akathisia; Parkinsonism; Dystonic reactions</td>
</tr>
<tr>
<td>What are some adverse reactions when taken in acute overdose?</td>
<td>Reduced seizure threshold; Hypotension/reflex tachycardia; Hyper- or hypothermia; CNS depression or coma (large doses); Quinidine-like effect</td>
</tr>
<tr>
<td>What are some key points in the management of neuroleptic overdose?</td>
<td>ABCs with IV access; Treat dystonia (i.e., diphenhydramine); Treat hypotension (i.e., fluids); Treat cardiotoxicity like TCAs (i.e., bicarb)</td>
</tr>
<tr>
<td>What are some commonly used medications to treat acute dystonic reactions?</td>
<td>Benztropine; Diphenhydramine; Diazepam</td>
</tr>
<tr>
<td>When can a patient be medically clear after a neuroleptic ingestion?</td>
<td>No signs and symptoms for 6 hours</td>
</tr>
<tr>
<td>What idiosyncratic reaction affects a small percentage of patients on neuroleptics that is potentially fatal?</td>
<td>Neuroleptic malignant syndrome (NMS)</td>
</tr>
<tr>
<td>What are some clinical features of NMS?</td>
<td>Autonomic instability (i.e., change in HR and BP), profound hyperthermia, mental status change, and rigidity</td>
</tr>
<tr>
<td>What are some key points in the management of NMS?</td>
<td>Rapid cooling (i.e., spray mist/ice); Use of benzos (paralytics if severe); Discontinue the offending agent</td>
</tr>
</tbody>
</table>
Lithium

What are interesting things to know about lithium?
Alkali metal with a long history of use; Used in the past for gout and CHF; Up to 90% will have some sign of toxicity

While the exact mechanism of lithium’s antimanic effects are not fully understood, what are some of its proposed mechanisms?
May substitute for sodium in neurons; Increase GABA transmission; Affect protein kinases (i.e., C and G)

What are some common preparations available?
Immediate release: 300 mg tiq or qid; Sustained release: 300 mg bid; Controlled release: 450 mg bid

What are some important pharmacokinetic properties of lithium?
95% of lithium is renally cleared; Lithium is absorbed preferentially to sodium; Any volume-depleted state will result in increased reabsorption of lithium

What are some side effects of lithium at therapeutic doses?
Fine tremors, polyuria, diabetes insipidus, weight gain, leukocytosis and cog-wheeling rigidity

What is an important question to ask when a patient presents with a question of lithium toxicity?
Acute versus chronic toxicity or is it acute on chronic

What are some clinical features of acute lithium toxicity?
Initial symptoms will be GI-related: nausea, emesis, and diarrhea followed by neurologic symptoms such as tremors, lethargy, and seizure or coma

Is acute lithium overdose directly cardiotoxic?
While ECG may show nonspecific T-wave changes, it is not directly cardiotoxic

What are some clinical features of chronic lithium toxicity?
Primarily neurologic: tremors, nystagmus, seizure, lethargy, and coma

What are some common causes of chronic toxicity?
Dehydration; Incorrect dosing; Renal insufficiency; Interaction with other drugs (i.e., NSAIDs)

<table>
<thead>
<tr>
<th>Therapeutic Level</th>
<th>Maintenance</th>
<th>0.5–0.8 mEq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute mania</td>
<td>0.7–1.2 mEq/L</td>
</tr>
</tbody>
</table>

Therapeutic Level

Maintenance 0.5–0.8 mEq/L
Acute mania 0.7–1.2 mEq/L
What are some long-term sequelae of lithium use? Personality changes; Memory deficits; Diabetes insipidus; Cerebellar dysfunction (i.e., ataxia)

What are some key points in the management of lithium toxicity? Follow lithium level; Chem-7 (esp. for renal function); Check for other drug interactions

What are some indications for the use of hemodialysis with lithium overdose? Renal failure (will not be able to clear lithium); Severe neurological symptoms

Why is it important to check lithium levels 6 hours after HD? Patients will get rebound lithium level as lithium redistributes from tissues

DRUGS OF ABUSE

Opioids

What is the definition of opioids? Natural and synthetic substances with morphine-like activity, opioids have analgesic and central nervous system depressant effects, as well as the potential to cause euphoria

What are endorphins? Endogenous peptides that produce pain relief (i.e., dynorphins/beta-endorphins)

What are some major opioid receptors found in the human body? Kappa, delta, and Mu

What is the primary opioid receptor that mediates euphoria/analgesia/respiratory depression? Mu

What are some other clinical features of opioid overdose? Most classic finding is miosis, altered mental status that can range from lethargy to coma, and respiratory depression

What is the most important adverse reaction to monitor with opioid overdose? Respiratory depression

What are some other adverse effects of opioid overdose? Noncardiogenic pulmonary edema (NCPE); Cardiotoxicity (i.e., pentazocine); Quinidine-like effect (i.e., QRS widening); Seizures (i.e., meperidine)

What is the mechanism of noncardiogenic pulmonary edema? Maybe involves loss of consciousness with respiratory depression and hypoxia
What is the agent of choice to reverse opioid overdose?  
Naloxone (Narcan)

Like alcohol withdrawal (i.e., life-threatening), is opioid withdrawal life-threatening as well?  
No

What are some indications for admission following opioid overdose?  
Anyone who requires a naloxone drip; Evidence of NCPE; Little improvement after naloxone; Life-threatening co-ingestion

Sedatives-Hypnotics

What is the definition of a sedative?  
Medication that reduces anxiety and induces relaxation

What is the definition of a hypnotic?  
Medication that induces sleep

Is there really a difference between the two?  
Not really, the two terms are used interchangeably

What are some examples of sedative-hypnotics?  
Benzodiazepines; Barbiturates; Buspirone; Zolpidem

What are some common indications for sedative-hypnotics?  
Anxiety; Seizures; Muscle spasms; Insomnia; Alcohol withdrawal

What is the mechanism of action of barbiturates?  
They enhance the activity of GABA receptors by increasing the duration by which chloride channels open as opposed to benzos, which increase the frequency of chloride channel opening

What are some key points in the management of sedative-hypnotics overdose?  
Airway support is crucial

Are barbiturates generally safer than benzodiazepines?  
No—benzodiazepines are generally safer as they produce less respiratory depression and minimal cardiac side effects

What are some clinical features of a benzodiazepine overdose?  
CNS effects ranging from sedation to coma and respiratory depression in large overdoses

What is the antidote of choice for benzodiazepine overdose?  
Flumazenil

What is the mechanism of action of flumazenil?  
Nonspecific competitive antagonist
Is it always safe to give flumazenil in a suspected benzodiazepine overdose?

No—particularly in multiple drug ingestions where benzos can have a seizure protective effect with drugs such as TCA or if the patient is on chronic use, as it may induce withdrawal.

Is benzodiazepine withdrawal dangerous?

Yes—it is similar to alcohol withdrawal (i.e., hyperthermia, hypertension, seizure, etc.) and can be potentially fatal.

Toxic Alcohols

Name the two alcohols that can be potentially fatal?

1. Methanol
2. Ethylene glycol

What is an important fact to note about these two alcohols?

All can increase the plasma osmolal gap; Methanol and ethylene glycol lead to high anion gap metabolic acidosis.

What are some characteristics of methanol?

Colorless clear flammable liquid that has a slight alcohol odor.

What are some common sources of methanol?

De-icing solutions; Shellac; Varnish; Windshield washer fluid.

What is the toxic dose of methanol?

Less than 1 mL/mg can lead to blindness or severe toxicity.

Methanol Levels

<table>
<thead>
<tr>
<th>Levels</th>
<th>Clinical Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20 mg/dL</td>
<td>Generally asymptomatic</td>
</tr>
<tr>
<td>&gt;50 mg/dL</td>
<td>Acidosis</td>
</tr>
<tr>
<td>&gt;100 mg/dL</td>
<td>Visual symptoms</td>
</tr>
<tr>
<td>&gt;150 mg/dL</td>
<td>Generally fatal</td>
</tr>
</tbody>
</table>

What is the major toxic metabolite of methanol?

Alcohol dehydrogenase metabolism to formaldehyde (causes metabolic acidosis) and formic acid (optic nerve toxin).

What are some clinical features of methanol toxicity?

Inebriation, nausea, abdominal pain, gastritis, and early visual disturbance such as blurriness and photophobia.

What are some severe symptoms of methanol toxicity?

Coma, seizure, blindness, hypotension, cardiac failure, and pulmonary edema.
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are important laboratory tests to obtain?</td>
<td>An osmolal gap and anion gap; Methanol level</td>
</tr>
<tr>
<td>What are some key points in the management of methanol toxicity?</td>
<td>ABCs; Aggressive early therapy is key, especially before the onset of symptoms; Sodium bicarbonate for acidosis; While ethanol can be given (and is effective) 4-methylpyrazole is commonly used; Folic acid may increase metabolism of formic acid</td>
</tr>
<tr>
<td>What is the mechanism of action of 4-methylpyrazole?</td>
<td>Inhibits alcohol dehydrogenase preventing the formation of toxic metabolites</td>
</tr>
<tr>
<td>When should hemodialysis be started?</td>
<td>High [methanol] &gt;50 mg/dL; Presence of metabolic acidosis; Severe symptoms such as visual changes</td>
</tr>
<tr>
<td>What are some common sources of isopropyl alcohol?</td>
<td>Nail-polish remover; Glues; Rubbing alcohol</td>
</tr>
<tr>
<td>What is the metabolism of isopropyl alcohol?</td>
<td>About 50% excreted in urine unchanged; The rest is converted to acetone</td>
</tr>
<tr>
<td>Is acetone dangerous?</td>
<td>It is not toxic, but can lead to a ketosis with no acidosis (hallmark of isopropyl alcohol)</td>
</tr>
<tr>
<td>How is acetone excreted?</td>
<td>Primarily through the kidney and lung</td>
</tr>
<tr>
<td>What is the typical lab finding in isopropyl alcohol?</td>
<td>Increased osmolal gap with no acidosis</td>
</tr>
<tr>
<td>What is the treatment for isopropyl alcohol toxicity?</td>
<td>Supportive care; Respiratory care</td>
</tr>
<tr>
<td>What are some common sources of ethylene glycol?</td>
<td>Brake fluid; Automobile coolant systems</td>
</tr>
<tr>
<td>What is the toxic dose of ethylene glycol?</td>
<td>&gt;15 mL/kg</td>
</tr>
<tr>
<td>What are the toxic metabolites of ethylene glycol?</td>
<td>Glycoaldehyde; Glycolic acid; Oxalate</td>
</tr>
<tr>
<td>What are some effects of oxalate?</td>
<td>Combines with calcium (calcium oxalate crystals) that damage the kidney and can also damage organs such as liver and brain, in addition, can cause hypocalcemia</td>
</tr>
<tr>
<td>What are some ECG findings associated with ethylene glycol?</td>
<td>ECG can show findings of hypocalcemia such as a prolonged QT or manifestations of hyperkalemia due to ARF</td>
</tr>
</tbody>
</table>
What is the typical lab finding in ethylene glycol toxicity? Elevated osmolal gap; Elevated anion gap acidosis

What are common urinary findings in ethylene glycol toxicity? Hematuria, proteinuria, and crystaluria, which is a diagnostic finding

Is gastric decontamination effective? Ipecac, cathartics, and gastric aspiration have little role here and AC poorly absorbs ethylene glycol

What are some key points in the management of ethylene glycol? Aggressive early therapy is key; Correct any acidosis immediately; While ethanol can be given (and is effective); Fomepizole is the standard of care now; Hemodialysis in severe cases

What are some indications of HD in ethylene glycol toxicity? Severe metabolic acidosis; Renal dysfunction (i.e., ARF); Levels >50 mg/dL

Cocaine

What is the mechanism of action of cocaine? Cocaine enhances monoamine neurotransmitter activity in the central and peripheral nervous systems by blocking the presynaptic reuptake pumps for these neurotransmitters

What is a secondary effect of cocaine that is unique among other stimulants? Blocks voltage-gated membrane sodium ion channels:

- Local anesthetic effects;
- Dysrhythmias

What are two forms of cocaine? 1. Base (form that can be smoked) 2. Salt (form that can be ingested or injected)

<table>
<thead>
<tr>
<th>Pharmacokinetics</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intravenous</td>
<td>seconds</td>
<td>15–30 minutes</td>
</tr>
<tr>
<td>Inhalation</td>
<td>seconds</td>
<td>15–30 minutes</td>
</tr>
<tr>
<td>Intranasal</td>
<td>20 minutes</td>
<td>1 hour</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>90 minutes</td>
<td>3 hours</td>
</tr>
</tbody>
</table>

What are some clinical features of acute intoxication? Euphoria, increased energy, alertness; decreased appetite, need for sleep, and fatigue
What are some adverse effects of cocaine intoxication? Panic attacks, paranoia, cocaine-induced psychosis, impaired judgment, and dysphoric mood

What are the effects of cocaine on specific organs:

CVS Increases heart rate, blood pressure, and systemic vascular resistance; cardiac arrhythmias, sudden death, and AMI; cardiomyopathy and myocarditis with chronic use

CNS Seizures, cerebral vasoconstriction, cerebrovascular disease, and stroke; acute dystonic reactions (i.e., akathisia)

Respiratory system Perforation of the nasal septum and chronic rhinitis from snorting; SOB, wheezing, pneumothorax, and pulmonary edema from smoking

What are some important causes of chest pain to consider in a patient who presents shortly after cocaine use? AMI (most likely); Pneumothorax; Aortic dissection; Pulmonary infarction

What is the drug of choice for acute cocaine toxicity? Benzodiazepines

What are some key points in the management of acute cocaine intoxication? Supportive care (i.e., ABCs); Liberal use of benzos; CT for any question of stroke; Cardiac workup if suspected AMI; Prevent/treat rhabdo and hyperthermia

What is particularly worrisome about intubating a patient with acute cocaine intoxication (i.e., having intractable seizures)? Can still have continued seizures that can lead to permanent brain damage (must have EEG monitoring in place)

What are some clinical features of cocaine withdrawal? Anhedonia, cocaine craving, anxiety, and depression (it is not life-threatening)

What is the difference between a body-stuffer and a body-packer?

Body-stuffer Swallow small packs to avoid police capture; Typically mild and transient adverse affects; Tx is observation and AC administration

Body-packer Smuggle large quantities of drugs (cocaine); Often swallow 100+ pre-packed drugs; Potentially fatal if they rupture; Surgical intervention if bags rupture/obstruct
### Phencyclidine

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some common street names for phencyclidine (PCP)?</td>
<td>Angel dust, crystal, peep, hog, and PCP</td>
</tr>
<tr>
<td>What are some available forms of PCP?</td>
<td>Powder, tablet, crystal, liquid, and capsule</td>
</tr>
<tr>
<td>What are some important things to know about PCP?</td>
<td>Frequently found as mixture in other drugs; Often produce brief dissociative reactions; Effects are often unpredictable (part of the appeal for many)</td>
</tr>
<tr>
<td>What is particularly important about the pharmacokinetics of PCP?</td>
<td>Well absorbed by any route</td>
</tr>
<tr>
<td>What is the clinical hallmark of PCP intoxication that allows it to be distinguished from other street drugs?</td>
<td>Vertical nystagmus</td>
</tr>
<tr>
<td>What are some clinical features of low to moderate PCP intake?</td>
<td>Confusion, ataxia, dysphoria, catatonic behavior, dystonia, violent behavior, and frank psychosis in rare cases</td>
</tr>
<tr>
<td>What are some adverse effects of high dose PCP intake?</td>
<td>Hypertension, seizure, and hyperthermia</td>
</tr>
<tr>
<td>What are some key points in the management of PCP?</td>
<td>Care is supportive (i.e., ABCs); Benzos for seizure and agitation; Reduce external stimuli; Physical/chemical restraint if violent; Severe HTN should be treated to avoid CVA; Prevent/treat rhabdo and hyperthermia</td>
</tr>
</tbody>
</table>

### Amphetamines

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are amphetamines?</td>
<td>Stimulant agents with sympathomimetic properties (like cocaine) that act on the CNS and PNS that stimulate both beta and alpha receptors</td>
</tr>
<tr>
<td>What are some common amphetamine derivatives?</td>
<td>Methamphetamine (i.e., crank, meth, glass); Methylphenidate (i.e., Ritalin); 3,4-Methylenedioxyamphetamine (i.e., Ecstasy)</td>
</tr>
<tr>
<td>What are some major routes of amphetamine administration?</td>
<td>Oral, intravenous, and inhalation</td>
</tr>
<tr>
<td>What are the two organ systems of concern with amphetamine intoxication?</td>
<td>1. CNS 2. CVS</td>
</tr>
</tbody>
</table>
What are some neurologic symptoms of amphetamine intoxication? Anxiety, aggression, seizure, delirium, euphoria, stroke, and cerebral edema

What are some cardiovascular symptoms of amphetamine intoxication? Tachycardia, hypertension, chest pain, dysrhythmias, AMI, and sudden death

What are some other complications of amphetamine intoxication? Renal failure, rhabdomyolysis, hyperthermia, anorexia, and complications associated with IVDA

What are some clinical features of amphetamine withdrawal? Anxiety, drug craving, irritability, insomnia, mood swing, and paranoia

What are some key points in the management of amphetamine intoxication? Primarily supportive (i.e., ABCs); Prevent/treat rhabdo and hyperthermia; Benzos for seizure and agitation

**Lysergic Acid Diethylamide**

What are some commonly used hallucinogens? Lysergic acid diethylamide (LSD); Psilocybin; Ketamine; Mushrooms; Mescaline

What is the mechanism of action of hallucinogens? Drugs that induce hallucinations, where a user perceives a sensory experience that is not actually there, although in many cases many drugs just distort sensory input (i.e., illusions)

Give some examples of common illusions produced by LSD? Trail: objects in visual field “leave a trail”; Feelings of depersonalization; Synesthesia: “see sound” or “hear colors”

What are some common clinical features of LSD intoxication? Altered perception is the hallmark along with hypertension, pupillary dilation, sweating, palpitations, blurred vision, incoordination and tremors

What is the hallmark of acute LSD intoxication? “Bad trip” where the user experiences fear, paranoia, feelings of depersonalization

What is the optimal way to handle a patient with a bad trip? Reassurance and “talking the patient down” until the drug wears off and consider use of benzos
**What are some long-term complications of LSD use?**

Primarily psychiatric: flashbacks (reliving the perceptual distortions), depression, psychosis, and personality change

**Is death from LSD common?**

LSD generally does not directly cause death, but indirectly via self-injury or depression/suicide

### METALS, CHEMICALS, AND GASES

#### General Information

**What is important to know about the acute toxicity of metals?**

Most metals bind to sulphydral groups of enzymes found throughout the body so have multisystem effects

**What are some common clinical features of acute toxicity of most metals:**

<table>
<thead>
<tr>
<th>System</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>The hallmark of acute metal toxicities: Nausea, emesis, and diarrhea</td>
</tr>
<tr>
<td>CVS</td>
<td>Can range from symptoms of volume depletion (i.e., tachycardia) to frank heart failure or dysrhythmias</td>
</tr>
<tr>
<td>Renal system</td>
<td>Loss of protein and amino acids in urine, can also get acute tubular necrosis</td>
</tr>
<tr>
<td>Nervous system</td>
<td>Peripheral neuropathy is common as well as altered mental status</td>
</tr>
</tbody>
</table>

**What are some clinical features of chronic toxicity of most metals:**

<table>
<thead>
<tr>
<th>System</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nervous system</td>
<td>CNS and PNS disturbances are more prominent than GI symptoms</td>
</tr>
<tr>
<td>Renal system</td>
<td>Varying degrees of renal insufficiency is usually noted</td>
</tr>
<tr>
<td>Hematology/Oncology</td>
<td>Anemias and neoplasm can be found</td>
</tr>
<tr>
<td>Dermatology</td>
<td>Rashes and colored lines of gums/nails often noted</td>
</tr>
</tbody>
</table>

**What are some important aspects of the evaluation to focus on with suspected exposure to metals?**

History, occupation, lifestyles, hobbies, use of herbal remedies, and travels

**What particular area of the exam should one focus on?**

Neurologic exam
What are some appropriate laboratory tests to obtain?
CBC with a peripheral smear; Chem-7 (assess renal function); Liver function tests; Urinalysis; Abdominal films; Blood and urine metal tests

### Arsenic

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What group is more likely to get arsenic (As) exposure?</td>
<td>Industrial workers</td>
</tr>
<tr>
<td>What are some important things to know about arsenic?</td>
<td>Over 1 million workers are exposed to As; Commonly found in pesticides/herbicides; Main route of exposure is inhalation; Also become exposed via smelting of ore</td>
</tr>
<tr>
<td>What are other common sources of As?</td>
<td>Shellfish; Combustion of fuel; Metal alloys/glass/ceramics</td>
</tr>
<tr>
<td>What are some forms of As?</td>
<td>Inorganic (arsenates, elemental arsenic); Organic (arsine)—generally nontoxic</td>
</tr>
<tr>
<td>Which form is generally more toxic?</td>
<td>Inorganic trivalent forms (i.e., arsenite)</td>
</tr>
<tr>
<td>What makes As particularly attractive as a poison?</td>
<td>Resembles sugar and tasteless</td>
</tr>
<tr>
<td>What are two primary routes of As exposure?</td>
<td>1. Inhalation&lt;br&gt;2. Ingestion</td>
</tr>
<tr>
<td>What is the primary mechanism by which As exerts its toxicities?</td>
<td>Uncouples oxidative phosphorylation; Inhibits mitochondrial enzymes; Binds to globin portion of hemoglobin</td>
</tr>
<tr>
<td>What are some clinical features of acute As due to inorganic salts?</td>
<td>Nausea, emesis, diarrhea, ECG changes, dysrhythmias, shock, hematuria, seizure, coma, bone marrow suppression, and peripheral neuropathies</td>
</tr>
<tr>
<td>What are some clinical features of chronic toxicity due to As?</td>
<td>Cirrhosis, hematopoietic malignancies, dermatitis, stocking-glove sensory neuropathy, and cancer</td>
</tr>
<tr>
<td>What are some methods to detect As?</td>
<td>Blood levels (&lt;5 mcg/L normal); Difficult to differentiate organic versus inorganic; Urine “spot” testing</td>
</tr>
<tr>
<td>What are some key points in the management of acute As toxicity?</td>
<td>Supportive care; Appropriate lab testing; Consider use of chelating agent</td>
</tr>
<tr>
<td>What are some chelating agents used?</td>
<td>Dimercaprol; D-penicillamine; Succimer</td>
</tr>
</tbody>
</table>
### Lead

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some functions of chelating agents?</td>
<td>Bind to metal to facilitate excretion; Deplete tissues of metals</td>
</tr>
<tr>
<td>What are the primary site of lead absorption in the body?</td>
<td>Bones (&gt;90% in adults compared to 75% in children)</td>
</tr>
<tr>
<td>What are the long-term cognitive deficits associated with elevated lead levels?</td>
<td>Learning, behavioral disorders, and decreased intelligence</td>
</tr>
<tr>
<td>How is lead typically absorbed in the body?</td>
<td>Lead initially attaches to red blood cells and then distributes to various locations such as the brain, kidney and bones</td>
</tr>
<tr>
<td>What are some clinical features of acute lead toxicity?</td>
<td>Abdominal pain, nausea, emesis, lethargy, fatigue, seizure, and coma</td>
</tr>
<tr>
<td>What are some clinical features of chronic lead toxicity?</td>
<td>Nephritis, peripheral neuropathy, myalgias, anemia, and motor weakness</td>
</tr>
<tr>
<td>What are some other diagnostic tests to consider?</td>
<td>X-ray fluorescence; Nerve conduction velocity testing; Neurobehavioral testing</td>
</tr>
<tr>
<td>What are the classic laboratory findings of lead poisoning?</td>
<td>Basophilic stippling; Anemia; Hemolysis</td>
</tr>
<tr>
<td>What is a normal lead level?</td>
<td>&lt;10 ug/dL</td>
</tr>
<tr>
<td>What are some key points in the management of lead poisoning?</td>
<td>Removal of lead source (i.e., strip paint); Chelating agents</td>
</tr>
<tr>
<td>What are some commonly used chelating agents?</td>
<td>EDTA; Succimer; Dimercaprol</td>
</tr>
<tr>
<td>What are some functions of chelating agents?</td>
<td>They bind inorganic metals and enhance excretion via the kidneys and GI tract; They can also deplete levels from soft tissues to be excreted</td>
</tr>
</tbody>
</table>
# Hydrocarbons

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some important things to know about hydrocarbons?</td>
<td>Common cause of mortality in children; Hydrocarbons are ubiquitous; Hydrocarbons commonly ingested/aspirated</td>
</tr>
<tr>
<td>What are some common sources of hydrocarbons?</td>
<td>Gasoline; Motor oils; Petroleum jelly; Laxatives; Solvents</td>
</tr>
</tbody>
</table>
| What are two primary routes of hydrocarbon toxicity?                    | 1. Ingestion  
2. Inhalation                                                                                                                                                                             |
| What are some hydrocarbons with systemic effects?                       | Aromatic hydrocarbons; Halogenated hydrocarbons                                                                                                                                                        |
| What are some clinical features of hydrocarbon ingestions?             | Drowsiness, seizures, coma, nausea, emesis, and in cases where there is aspiration of hydrocarbons, patients will exhibit respiratory involvement such as dyspnea, coughing, distress, and even hypoxia/cyanosis |
| How do most patients do after hydrocarbon ingestion?                    | Most are asymptomatic after ingestion                                                                                                                                                                |
| What is an important complication of hydrocarbon ingestion?             | Aspiration                                                                                                                                                                                           |
| What are some physical properties that predict the aspiration potential of hydrocarbons? | Greater volatility; Lesser viscosity; Surface tension                                                                                                                                                |
| What are some signs that aspiration may have occurred?                  | Typically patients will cough, gag, and exhibit dyspnea on exertion                                                                                                                               |
| What are some indications for patients with hydrocarbon ingestion of admission? | Symptomatic after 6 hours; Abnormal CXR suggestive of aspiration                                                                                                                                    |
| What does “sniffing,” “bagging,” or “huffing” imply?                   | Inhalation of volatile hydrocarbons with the intention of getting high                                                                                                                                  |
| What are some clinical features of inhaling hydrocarbons?              | Euphoria, agitation, seizure, stupor, and delusions                                                                                                                                                   |
| What is the most feared complication of inhaling halogenated hydrocarbons? | Sudden death (fatal dysrhythmias)                                                                                                                                                                     |
| What is the mechanism by which halogenated hydrocarbons can cause fatal dysrhythmias? | Heart is sensitized to circulating catecholamines, so any sudden increase in sympathetic response can cause fatal dysrhythmias                                                                           |
| What are some hydrocarbons that may cause thermal burns?               | Asphalt; Tar                                                                                                                                                                                          |
**What are some key points in the management of hydrocarbon toxicity?**

Supportive care is the mainstay (i.e., ABC); Monitor carefully for respiratory involvement; Avoid emetic agents (i.e., ipecac); AC is not particularly useful; Standard ACLS for dysrhythmias

---

**Methemoglobin**

**What is methemoglobin?**

Abnormal hemoglobin (Hg) that is in the ferric state (Fe³⁺) rather than the ferrous state (Fe²⁺) that renders it unable to accept oxygen or carbon dioxide

**What are some of the physiologic effects of methemoglobin on oxygen-carrying capacity?**

Reduces the oxygen-carrying capacity; Left shift of the dissociation curve

**What is the normal level of methemoglobin in a healthy adult?**

<1% of total hemoglobin

**What are the two primary mechanisms by which methemoglobin is eliminated?**

1. NADH electron donation of ferric to ferrous
2. NADPH (accounts for small portion)

**What are two common causes of congenital methemoglobinemia?**

1. NADH methemoglobin reductase deficiency
2. Hemoglobin M

**What is the most common cause of methemoglobinemia?**

Acquired methemoglobinemia

**What is the mechanism by which acquired methemoglobinemia occur?**

Commonly occurs due to drugs or toxins that oxidize ferrous iron

**List some common causes of acquired methemoglobinemia?**

Local anesthetics (most common cause); Nitrites; Sulfonamide; Dapsone

**What is the hallmark of methemoglobinemia?**

Cyanosis that fails to improve with high-flow oxygen

**What are some clinical features of methemoglobinemia?**

Largely dependent on level of methemoglobin: fatigue, anxiety, dizziness, tachycardia, mental status change, and dysrhythmias/acidosis at higher levels

**What is the methemoglobin level at which central cyanosis appears?**

Methemoglobin levels of 15%
Aside from persistent cyanosis, what are some other diagnostic clues of methemoglobinemia?

Chocolate brown appearance of blood on filter paper; Normal partial pressure of oxygen on ABG; MetHb level determined by cooximetry

What is the treatment of choice for methemoglobinemia?

Methylene blue

What is the mechanism of action of methylene blue?

Increases erythrocyte reduction of methemoglobin to oxyhemoglobin

What are some adverse reactions to methylene blue?

Hemolysis in G6PD deficiency; Methemoglobinemia at high doses; False low pulse ox readings

Carbon Monoxide

What are some important things to know about carbon monoxide (CO) poisoning?

Leading cause of poisoning in the United States; Majority of cases due to fires; Suicide contributes to a good portion of cases; CO is odorless and colorless

List some sources of CO.

Incomplete combustion of carbonaceous material (i.e., engine exhaust); Degradation of heme; Vertical transmission (maternal-to-fetal); Halogenated hydrocarbons

What is the pathophysiology of CO poisoning?

CO binds with Hb forming carboxyhemoglobin (COHb) that decreases oxygen content of blood and will also shift O2-Hb dissociation curve to the left (decrease oxygen delivery to tissue)

What two organ systems are most profoundly affected by CO poisoning?

1. CNS
2. CVS

<table>
<thead>
<tr>
<th>COHb Level</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–20%</td>
<td>Flu-like symptoms such as headache and nausea</td>
</tr>
<tr>
<td>20–30%</td>
<td>Severe headache, irritability, and impaired judgment</td>
</tr>
<tr>
<td>40–50%</td>
<td>Loss of consciousness and confusion</td>
</tr>
<tr>
<td>60–70%</td>
<td>Unconsciousness, cardiovascular collapse, seizure</td>
</tr>
<tr>
<td>&gt;80%</td>
<td>Rapidly fatal</td>
</tr>
</tbody>
</table>
What are some important points to know about COHb levels?

Smokers can have levels as high as 10%; Does not predict neurologic sequelae

What are some clinical features of CO poisoning in the following organ systems:

**CNS**

Headaches, dizziness, blurred vision, ataxia, seizure, coma, and even death

**CVS**

Signs of demand ischemia (i.e., chest pain), hypotension, and dysrhythmias

**Respiratory**

Pulmonary edema and ARDS

**Renal**

ARF (2° to rhabdomyolysis)

**Dermal**

Characteristic cherry-red color (more so after massive exposure and death)

What is an important neurologic complication after CO poisoning?

Delayed neurologic sequelae (DNS)

What is DNS?

Neurologic deterioration after a lucid period of around 2 weeks

What are some clinical features of DNS?

Ataxia, tremor, amnesia, memory impairment, paralysis, and dementia

When do the symptoms of DNS resolve?

Range from 1 month to 1 year depending on severity

What is the concern of the fetus with regards to CO poisoning?

Fetal Hb binds CO more avidly than maternal Hb, which can result in anoxic brain injury and death of the fetus

What are some key points in the management of CO poisoning?

Remove from source as soon as possible; Administer 100% O₂ immediately; Check COHb by co-oximetry; ABG/ECG when indicated; Hyperbaric oxygen when indicated

<table>
<thead>
<tr>
<th>FIO₂</th>
<th>COHb T₁/₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Room air</td>
<td>2–6 hours</td>
</tr>
<tr>
<td>100% at 1 atm</td>
<td>90 minutes</td>
</tr>
<tr>
<td>100% at 3 atm</td>
<td>30 minutes</td>
</tr>
</tbody>
</table>

What are some indications for the use of hyperbaric oxygen (HBO) in CO poisoning?

Evidence of end-organ damage (i.e., LOC); COHb levels >25%; COHb >15% for pregnant women/child

Persistent symptoms after 1 atm O₂
### Cyanide and Hydrogen Sulfide

#### What are some important sources of cyanide (CN)?
- Combustion of many types of material;
- Smoking;
- Food sources (i.e., amygdalin);
- Ingestion of cyanide salts (i.e., homicide)

#### What is the pathophysiology of CN toxicity?
- Inhibition of cytochrome oxidase (essential for oxidative phosphorylation) that results in cellular hypoxia leading to increased anaerobic metabolism (lactic acidosis)

#### Name three routes of exposure for CN.
1. Parental
2. Inhalation
3. Ingestion

#### What are some clinical features of acute CN toxicity?
- Headache, confusion, lethargy, hypotension, abdominal pain, nausea, vomiting, traditional cherry-red skin, and severe metabolic acidosis

#### When should one suspect CN toxicity?
- A fire victim with a coma and acidosis;
- Bitter almond odor;
- Unexplained coma/acidosis (i.e., in laboratory or industrial work)

#### What role does CN levels play in the acute management of cyanide?
- They cannot be obtained rapidly, so must use clinical judgement

#### What is a common laboratory finding in acute CN toxicity?
- Severe metabolic acidosis with greater anion gap

#### What is the initial management for patients with suspected CN toxicity?
- Supportive care (i.e., establish airway);
- Sodium bicarbonate for acidosis; Treat associated conditions (i.e., CO);
- Consider use of antidote

#### What is the antidote typically given for CN toxicity?
- Cyanide antidote kite
- Sodium nitrite; Sodium thiosulfate; Amyl nitrite pearls

#### What is the mechanism by which nitrite administration works?
- Induces a methemoglobinemia, for which CN has a greater affinity

#### What antihypertensive is known to contain CN?
- Nitroprusside

#### What other toxin produces effects similar to CN?
- Hydrogen sulfide

#### What are some sources of hydrogen sulfide?
- Natural sources (i.e., sulfur springs);
- Industrial sources; Decay of sulfur-containing products (i.e., fish)
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the pathophysiology of hydrogen sulfide?</td>
<td>Similar to CN, but binds to the same enzyme with greater affinity than CN and also causes mucous membrane irritation</td>
</tr>
<tr>
<td>What are some clinical features of hydrogen sulfide toxicity?</td>
<td>Hypoxia, irritation to areas such as eyes, throat, and nasal passage, and severe metabolic acidosis</td>
</tr>
<tr>
<td>When should the diagnosis of hydrogen sulfide be suspected?</td>
<td>Rapid loss of consciousness; Odor of rotten eggs; Rescue from an enclosed space; Multiple victims</td>
</tr>
<tr>
<td>What is a common laboratory finding in hydrogen sulfide poisoning?</td>
<td>Severe metabolic acidosis</td>
</tr>
<tr>
<td>What is the initial management in patients with suspected hydrogen sulfide toxicity?</td>
<td>Remove the patient from the source; Supportive care; Nitrite may be of some use; Consider HBO therapy</td>
</tr>
</tbody>
</table>

**Pesticides**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is a pesticide?</td>
<td>Agent commonly used to destroy or repel pests such as insects or rodents</td>
</tr>
<tr>
<td>What is the mechanism of organophosphate toxicity?</td>
<td>Bind to cholinesterases, especially acetylcholinesterases, preventing the breakdown of acetylcholine (ACh)</td>
</tr>
<tr>
<td>What is the mechanism of toxicity of organophosphates?</td>
<td>Cholinergic poisoning due to excessive accumulation of ACh</td>
</tr>
<tr>
<td>What are the clinical effects primarily due to?</td>
<td>Excessive ACh at the nicotinic receptors (autonomic ganglia and skeletal muscle) and muscarinic receptors</td>
</tr>
<tr>
<td>What are some factors that determine the clinical effects?</td>
<td>Route of exposure; Lipid solubility; Dose</td>
</tr>
</tbody>
</table>
| What is “SLUDGE” syndrome?                                            | Clinical effects due to excessive ACh at the muscarinic receptors  
  - Salivation  
  - Lacrimation  
  - Urination  
  - Diarrhea  
  - GI cramps  
  - Emesis                                                                                                                                                                                                                                                                  |
| What are some other clinical features of excessive muscarinic activation? | Bronchoconstriction, bronchorrhea, miosis, and bradycardia                                                                                                                                                                                                                                                                             |
What are some CNS effects of excessive ACh activity?
Agitation, confusion, coma, and seizure

What are the nicotinic effects of excessive ACh activity?
Fasciculations, muscle weakness, and paralysis

What is the initial management of organophosphate toxicity?
Supportive care; Decontamination of patient; Consider use of an antidote

What are two antidotes that can be used in organophosphate toxicity?
1. Atropine
2. Pralidoxime (2-PAM)

What is the mechanism of atropine?
Competitive inhibition of ACh only at muscarinic receptors

What is the endpoint of atropine therapy?
Drying of secretions

What is the mechanism of pralidoxime?
Regenerates organophosphate-bound acetylcholinesterase complex, regenerating its ability to metabolize ACh

## TOXICOLOGY SUPPLEMENT

<table>
<thead>
<tr>
<th>Toxin</th>
<th>Antidote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen</td>
<td>N-Acetylcysteine</td>
</tr>
<tr>
<td>Anticholinergics</td>
<td>Physostigmine</td>
</tr>
<tr>
<td>Arsenic</td>
<td>d-penicillamine/Dimercaprol</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Flumazenil</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>Glucagon</td>
</tr>
<tr>
<td>Black widow spider</td>
<td><em>Latrodectus</em> antivenin</td>
</tr>
<tr>
<td>Botulism</td>
<td><em>Botulinum</em> antitoxin</td>
</tr>
<tr>
<td>Brown recluse spider</td>
<td><em>Loxosceles</em> antivenin</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Glucagon and calcium</td>
</tr>
<tr>
<td>Coral snake bite</td>
<td><em>Elapid</em> antivenin</td>
</tr>
<tr>
<td>Cyanide</td>
<td>Amyl nitrite, sodium</td>
</tr>
<tr>
<td></td>
<td>nitrite, sodium</td>
</tr>
<tr>
<td></td>
<td>thiosulfate</td>
</tr>
<tr>
<td>Digitalis glycosides</td>
<td>Digoxin-specific FAB</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>Ethanol or fomepizole</td>
</tr>
<tr>
<td>Heparin</td>
<td>Protamine</td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
<td>Sodium nitrite</td>
</tr>
</tbody>
</table>
### Toxin Antidote

<table>
<thead>
<tr>
<th>Toxin</th>
<th>Antidote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoglycemic agents</td>
<td>Dextrose</td>
</tr>
<tr>
<td>Iron</td>
<td>Deferoxamine</td>
</tr>
<tr>
<td>Isoniazid</td>
<td>Pyridoxine (B6)</td>
</tr>
<tr>
<td>Lead</td>
<td>Dimercaprol</td>
</tr>
<tr>
<td>Methanol</td>
<td>Ethanol or fomepizole</td>
</tr>
<tr>
<td>Methemoglobin</td>
<td>Methylene blue</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>Leucovorin and folate</td>
</tr>
<tr>
<td>Opiates</td>
<td>Naloxone</td>
</tr>
<tr>
<td>Organophosphates</td>
<td>Atrophine</td>
</tr>
<tr>
<td>Rattlesnake bites</td>
<td><em>Crotalidae</em> antivenin (crofrib)</td>
</tr>
<tr>
<td>Tricyclics</td>
<td>Sodium bicarbonate</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Vitamin K</td>
</tr>
</tbody>
</table>

### (Continued)

<table>
<thead>
<tr>
<th>Toxin</th>
<th>Temp</th>
<th>HR</th>
<th>RR</th>
<th>BP</th>
<th>Pupil</th>
<th>Diaphoresis</th>
<th>MS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticholinergic</td>
<td>↑↑↑↑</td>
<td>+↑</td>
<td>−</td>
<td>−</td>
<td>↑</td>
<td>↓</td>
<td>Delirium</td>
</tr>
<tr>
<td>Cholinergic</td>
<td>−</td>
<td>+/−</td>
<td>+/−</td>
<td>+/−</td>
<td>+/−</td>
<td>↑</td>
<td>Normal</td>
</tr>
<tr>
<td>Sympathomimetic</td>
<td>↑↑↑</td>
<td>↑↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑↑↑↑</td>
<td>Agitated</td>
</tr>
<tr>
<td>Sedative-hypnotics or ethanol</td>
<td>↓↓↓↓</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>↓↓↓↓</td>
<td>Depressed</td>
</tr>
<tr>
<td>Opioids</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>−</td>
<td>Depressed</td>
</tr>
<tr>
<td>Withdrawal from opioids</td>
<td>–</td>
<td>↑</td>
<td>−</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>Normal anxious</td>
</tr>
<tr>
<td>Withdrawal from sedative-hypnotics or ethanol</td>
<td>↑↑↑↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑↑↑↑</td>
<td>↑↑↑↑</td>
<td>Agitated confused</td>
</tr>
</tbody>
</table>

334 Deja Review: Emergency Medicine
CHAPTER 17

Behavioral Emergencies

MEDICAL EVALUATION AND CLINICAL APPROACH

What are some important things to consider in the clinical approach to patients with psychiatric problems?

Is the patient a danger to self or others?; Are physical symptoms a manifestation of a psychiatric disorder?; Psychiatric disorders may be exacerbated by a physical condition; Patients may present with a medical problem caused by a psychiatric disorder

What are some features for each of the following triage categorization for psychiatric patients (as well as for all other patients):

Emergent
- Patient has active suicidal ideation;
- Patient has homicidal ideation;
- Acutely intoxicated;
- Life-threatening injury (i.e., myocardial infarction [MI]);
- Abnormal vital signs

Urgent
- Suicidal ideation;
- Agitation/anxiety;
- Incoherent patient

Nonurgent
- Does not meet criteria for the first two;
- Patient requests psychiatric help

What is a very important thing to keep in mind when evaluating a psychiatric patient?

All psychiatric patients should receive both a thorough psychiatric and medical evaluation

How do you deal with a patient who may have uncontrolled behavioral problems?

Restraints or seclusion

What are some warning signs that a patient with a psychiatric problem may require restraints?

Abrupt changes in behavior;
- Threatening violent behavior; Patient states in fear of losing control
What are some characteristics that a seclusion room should have? Safety foremost; Continuous observation; Low stimulation (i.e., low lights); Security staff

What are some key points for the following types of restraints used:

Verbal restraint Should be attempted in a calm approach; Encourage the patient to talk about any concerns and offer reassurance; Physical/chemical restraints may be needed

Physical restraint Commonly used for intoxicated, demented/delirious, and violent patients; Has minimal side effects and immediately reversible; Remove restraints when patient is not a danger to self or others

Chemical restraint Behavioral control once full evaluation done; Haldol and lorazepam drug of choice; Less intrusive than physical restraint; Benzodiazepines may worsen dementia and delirium

What are some characteristics of a patient presenting with a psychiatric disorder? Patient may regard behavior as normal; History of behavioral problems; Often will have normal vitals and laboratory test results; Can have hallucinations (i.e., auditory)

What are some medical conditions that may present as behavioral emergencies? Toxicological emergencies; Urinary tract infection; Drug withdrawal (i.e., EtOH); Myocardial infarction (MI); Diabetic ketoacidosis; Chronic renal disease; Thyroid dysfunction

What are some laboratory tests to consider in evaluation of a psychiatric patient? Glucose; Complete blood count (CBC) Urinalysis; Lytes (also calcium); Toxicology screen; Carboxyhemoglobin level

What are some elements in the medical history to consider when evaluating a patient with a psychiatric problem? Contact current and past primary doctors; Obtain all medical and psychiatric records; List of medications, especially sedatives/psych/pain medications; Always ask about alcohol and drug use

What are the key components of the mental status examination (MSE):

- Level of consciousness Alert; Fluctuating; Somnolent
**General appearance**
Overall appearance (i.e., hygiene); Movement: chores, tics, tremors, etc.; Activity level (i.e., agitation)

**Orientation**
Person, place, time, and event

**Memory**
Immediate, STM, and LTM; Three word recall

**Mood**
Stability; Quality (i.e., moody vs. anger)

**Speech**
Fluency, rate, and rhythm; Illogical versus logical

**Thought content**
Perception (i.e., hallucination); Bizarre thoughts; Delusions

**Insight and judgment**
History can usually infer this

**Cognitive function**
Ask to perform task such as spelling a word backward or serial 7s

---

**DEPRESSION AND SUICIDE**

**What are the symptoms of major depressive disorder?**
Five or more of the following symptoms for 2 weeks or greater:
- Anhedonia; Depressed mood;
- Fatigue; Sleep disturbance; Change in appetite; Inability to concentrate;
- Sense of worthlessness; Suicidal thoughts

**What are some important points in the history to consider when evaluating a patient with depression?**
Medications (i.e., beta-blockers); History of drug use; Neurologic conditions (i.e., CNS tumor); Endocrine conditions; Infectious disease (i.e., HIV); Any previous psychiatric history; Suicidal or homicidal ideation; Any recent life changes; Evaluation of social structure (i.e., family)

**What is the primary goal when evaluating a patient with depression?**
His/her potential for suicide

**What are some factors to consider when deciding to admit a patient with depression?**
Previous attempts at suicide; Social support; Younger or older males are more at risk; Plan and means to carry out suicide; Excessive use of drugs or alcohol
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are some discharge criteria to consider in a depressed patient?</td>
<td>If a support environment exists; Agrees to return if depression worsens; Not demented, delirious, or intoxicated; Close follow-up</td>
</tr>
<tr>
<td>Should antidepressants be started in the ED prior to discharge of the patient?</td>
<td>No—antidepressants take up to 4 weeks to work and will not acutely treat depression in the ED</td>
</tr>
<tr>
<td>How many people who attempt suicide are successful?</td>
<td>For every 20 attempts, 1 is successful</td>
</tr>
<tr>
<td>When do suicide attempts most commonly occur?</td>
<td>During a crisis marked by an acute personal loss</td>
</tr>
<tr>
<td>What are some common psychiatric illnesses associated with completed suicides?</td>
<td>Depression; Schizophrenia; Personality disorders; Panic disorders</td>
</tr>
<tr>
<td>What role does gender play in suicide?</td>
<td>Females attempt suicide three times more often; Males are successful three times more often</td>
</tr>
<tr>
<td>Does drug abuse play a role in suicide?</td>
<td>Yes—one fourth of successful suicides involve drugs and alcohol and up to half in adolescent suicides</td>
</tr>
<tr>
<td>List some risk factors associated with suicide attempts?</td>
<td>Underlying psychiatric illnesses; Age (rate highest in elderly); Chronic pain (i.e., cancer); Marital status (marriage is protective); Presence of lethal means; Family history</td>
</tr>
<tr>
<td>What is the most common cause of death in suicide in all age groups?</td>
<td>Firearms</td>
</tr>
<tr>
<td>What are some warning signs of suicide?</td>
<td>Recent life changes; Depression</td>
</tr>
<tr>
<td>Will asking a patient directly about suicide intent put ideas into his/her head?</td>
<td>No—one should always ask</td>
</tr>
<tr>
<td>What are key questions to ask a patient who expresses suicidal intention?</td>
<td>Ask if they are suicidal; Ask if they have a plan; Assess if they have the means</td>
</tr>
<tr>
<td>What is “silent suicide?”</td>
<td>Killing oneself slowly via nonviolent means such as not taking medication</td>
</tr>
<tr>
<td>What age group is “silent suicide” most common in?</td>
<td>Elderly</td>
</tr>
<tr>
<td>Can a suicidal patient leave American Medical Association (AMA)?</td>
<td>If they are found to be incompetent or dangerous, they cannot leave AMA</td>
</tr>
</tbody>
</table>
**ACUTE PSYCHOSIS**

What is the definition of acute psychosis?  
Break in reality often characterized by delusions, hallucinations, and disorganized speech/movement

Define the following terms:

**Hallucinations**  
False perception of a sensory modality that is not there with auditory stimuli being the most common

**Delusions**  
Fixed falsely held belief that is not accepted by a given cultural group and is held despite an evidence to the contrary

**Catatonia**  
Apparent detachment from the environment typically characterized by frozen rigid posture or violent agitation

What are some examples of negative symptoms?  
Poverty of speech; Loss of volition; Flat affect

What is a major psychiatric disorder that can present as an acute psychotic episode?  
Schizophrenia

What is the prevalence of schizophrenia in the general population?  
1% regardless of race or gender

When is the onset of schizophrenia?  
Commonly by late adolescence to early adulthood

What does the diagnosis of schizophrenia require?  
Severe impairment in the level of functioning; Duration of >6 months; At least two symptoms of acute psychosis for greater than a month; Exclusion of medical conditions as cause of symptoms

What are some features for each of the following psychiatric disorders that may present as acute psychosis:

**Schizoaffective disorder**  
Psychosis that is chronic; It is often associated with mood disorders; Psychotic features can occur without mood symptoms

**Schizophreniform disorder**  
Psychosis that lasts <6 months; Does not occur during a mood disorder
**Brief psychotic disorder**
Psychosis that lasts <1 month; Does not occur during a mood disorder

**Major depression with psychotic features**
Psychosis that occurs during a depressive episode

**What is the most important thing to do when evaluating a patient who is psychotic?**
Establishing safety

**What are some things to do to ensure safety when evaluating a patient with acute psychosis?**
Search for weapons; Use restraints if necessary; Avoid having the patient between you and an exit point

**Should all patients with acute psychosis be admitted?**
No, but patients who are a danger to others or themselves should probably be admitted

## MANIA

**What defines a manic episode?**
Three or more of the following for over 1 week:
- Impulsivity; Distractibility;
- Pressured speech; Grandiosity;
- Decreased need for sleep; Agitation;
- Flight of ideas

**Can a patient with a manic episode also have acute psychosis?**
Yes—often with paranoia or grandiosity

**What are some medical conditions that can cause mania?**
CNS tumors; Hyperthyroidism

**What are some medications/drugs that are known to cause mania?**
Phencyclidine; Steroids; EtOH; Psychostimulants

**What are some elements of the history to attain when evaluating a patient with mania?**
Current medications; History of illicit drug use; Any prior psychiatric history; Any homicidal or suicidal ideation; Any recent life stressors

**Are antimanic medications such as lithium or carbamazepine useful for an acute episode of mania?**
No—take days to weeks to take effect

**What class of drugs are useful for an acute episode of mania?**
Antipsychotic medications (i.e., haloperidol)

**What are some factors when deciding if a patient with acute mania should be admitted?**
Impulsivity leads to danger to self or others; Poor support structure; Active delusions that are dangerous
# PANIC ATTACKS

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<td>What are some clinical features of a panic attack?</td>
<td>Tremor; Shortness of breath; Paresthesias; Derealization; Chest pain; Tachycardia; Sense of impending doom</td>
</tr>
<tr>
<td>Are patients with panic disorder at increased risk of suicide?</td>
<td>Yes—up to 18 times more than the general population</td>
</tr>
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<td>Are patients who present with a panic attack just overreacting?</td>
<td>No—during a panic attack, the patient truly feels threatened and commonly needs reassurance</td>
</tr>
<tr>
<td>What are some medical conditions that may mimic a panic attack?</td>
<td>Asthma; Chronic obstructive pulmonary disease (COPD); Metabolic disturbances; Dysrhythmias; Hypoxia</td>
</tr>
<tr>
<td>What are some characteristics of a panic attack?</td>
<td>Typically begins suddenly; Lasts for about 15 minutes; Can occur without provocation</td>
</tr>
<tr>
<td>What are some elements of the history to attain when evaluating a patient with mania?</td>
<td>Current medications; Any prior psychiatric history; Excessive caffeine use; Any recent life stressors</td>
</tr>
<tr>
<td>What class of drugs are useful for the short-term management of a panic attack?</td>
<td>Benzodiazepines</td>
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<td>What is the most useful intervention for patients with a panic attack?</td>
<td>Reassurance and communication</td>
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# EATING DISORDERS

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<td>What are two eating disorders commonly seen in the emergency department?</td>
<td>1. Bulimia Nervosa (BN) 2. Anorexia Nervosa (AN)</td>
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<td>What is bulimia nervosa?</td>
<td>Chronic eating disorder that often waxes wanes, typically exacerbates during times of stress characterized by “binge and purge”</td>
</tr>
<tr>
<td>Describe the typical bulimic patient?</td>
<td>A normal-appearing female around the age of 18–24</td>
</tr>
<tr>
<td>How prevalent is bulimia?</td>
<td>5% of young adult females</td>
</tr>
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<td>What are some characteristic features of a binge?</td>
<td>Most patients with bulimia binge, that is characterized by excessive consumption of calories (up to 14,000 Kcal!), concealing from friends and family</td>
</tr>
</tbody>
</table>
Are bulimics typically underweight? No—often have normal weight

Is binge eating typically from hunger? Not necessarily—commonly described as a feeling of loss of control

What is purging? Inappropriate compensatory response to binging often characterized by self-induced emesis

What are some medical complications of bulimia:

- Ipecac use: Dermatomyositis; Cardiomyopathy
- Diuretic use: Electrolyte imbalance (i.e., hypokalemia); Dehydration
- Laxative use: Constipation; Hypokalemia; Dehydration
- Self-induced emesis: Electrolyte imbalance; Dental problems (i.e., erosions); Submandibular/parotid gland enlargement; May get esophageal tear or rupture

What are some clues during the history and physical exam that may point to bulimia?

- Loss of dental enamel; Unexplained hypokalemia; Large fluctuations in weight; Excessive exercise; Esophageal problems (i.e., bleeding)

What are some clues during the history and physical exam that may point to anorexia nervosa (AN)?

- Excessive exercise; Unexplained weight loss or growth problems; Activity or occupation (i.e., dancer)

What are some indications for admission for a patient who presents with bulimia?

- Metabolic complications (i.e., hypotension); Suicidal ideation; Persistent emesis

What is anorexia nervosa (AN)?

An eating disorder characterized by a preoccupying fear of obesity regardless of weight loss

What are four diagnostic criteria of AN?

1. Preoccupying fear of gaining weight
2. Weight loss >15% of ideal body weight
3. Amenorrhea greater than three consecutive cycles
4. Distorted body image

What is the mortality rate of AN at 10 years? Almost approaches 10%

What is characteristic of patients with AN who are in treatment?

Notorious for resistance to treatment and unmotivated

What are some clues during the history and physical exam that may point to AN?
What are some key points in the management of patients with AN?
Correct any underlying metabolic problems; Initial evaluation may require psychiatric involvement; Determine if outpatient treatment is possible

### DEMENTIA AND DELIRIUM

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<th>Patients with dementia or delirium often have impaired ability to recognize their condition and may be susceptible to injury</th>
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<td><strong>What is dementia?</strong></td>
<td>Progressive and global impairment of cognitive function without alteration in consciousness</td>
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<td><strong>What are some causes of irreversible dementia?</strong></td>
<td>Alzheimer’s disease; Vascular dementia (multi-infarct); Creutzfeldt-Jakob diseases; Parkinson’s disease</td>
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<tr>
<td><strong>What are some clinical features of dementia?</strong></td>
<td>Multiple cognitive deficits that include memory impairment along with either or some of the following: apraxia, aphasia, and agnosia</td>
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<td><strong>Is the onset of dementia typically acute?</strong></td>
<td>No—gradual onset with disturbances in recent memory that can be exacerbated by illnesses or certain medications</td>
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<tr>
<td><strong>What are some causes of reversible dementia?</strong></td>
<td>Medication; Metabolic disorders; Endocrine disorders; Depression (pseudodementia)</td>
</tr>
<tr>
<td><strong>What are some management points in the treatment of dementia?</strong></td>
<td>Eliminate medications that may exacerbate the condition; Identify and correct any underlying metabolic or endocrine disorder; If dementia is irreversible, consider medication that may slow the progression</td>
</tr>
<tr>
<td><strong>What are some clinical features of delirium?</strong></td>
<td>Acute onset with often diurnal fluctuation of symptoms, cognitive impairment, and reduced ability to focus and sustain attention</td>
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<tr>
<td><strong>What are some important causes of delirium to consider?</strong></td>
<td>Drugs and medications; Heavy metals; CNS injury; Infection; Metabolic disturbances</td>
</tr>
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<td><strong>What is the treatment of dementia?</strong></td>
<td>Identify and treat the underlying cause; Ensure the safety of a patient</td>
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INTOXICATION AND WITHDRAWAL

What is intoxication?
Ingestion of a drug or alcohol that often leads to impairment of judgment, perception, motor activity, and attention

What are some clinical features of intoxication?
Primarily manifests as impairment of judgment and motor activity with progression to delirium, coma, seizure, or even death with increasing amounts

How is the diagnosis of intoxication typically made?
Laboratory evaluation

What are some substances that cause psychostimulant intoxication?
Cocaine; Methamphetamine; Phenylpropanolamine

What are some clinical features of psychostimulant intoxication?
Can have paranoid psychotic excitation, may have signs of sympathetic response, and stereopathies (i.e., nail biting)

What medication class is useful for patient with psychostimulant intoxication?
Antipsychotics (i.e., haloperidol)

What are some key points in the management of patients with psychostimulant intoxication?
Ensure safety of patient (i.e., restraints); General supportive measures; Treatment of the intoxicating agent; Appropriate referral to psychiatry if needed

What are some clinical features of alcohol intoxication?
Confusion, ataxia, agitation, slurred speech, hallucinations, and possible violent paranoid ideation

What is an appropriate medication class if behavioral control is needed?
Antipsychotics

What is withdrawal?
Clinical syndrome that occurs with the cessation of a substance and can be reduced when the substance is taken again

What is the most commonly encountered withdrawal syndrome?
Alcohol

What are the clinical stages of alcohol withdrawal from the time of last drink:

6–24 hours
Hypertension, tachycardia, nausea, anxiety, and sleep disturbances
| **24–72 hours** | More severe autonomic disturbances and hallucinations and can take up to 6 days to resolve. Seizures can also occur during this time |
| | |
| **3–5 days** | Can progress to delirium tremens |
| **What is delirium tremens?** | Potentially fatal form of ethanol withdrawal |
| **What are some clinical features of delirium tremens?** | Autonomic instability, global confusion, tremors, incontinence, and hallucinations with a substantial mortality if left untreated |
| **What is the treatment of acute alcohol withdrawal?** | Establish supportive care; IV fluids along with thiamine, magnesium, and multivitamin; Generally avoid giving glucose before thiamine as this may precipitate Wernicke’s encephalopathy; Sedation with benzodiazepines is key |
| **What are some indications for a head CT in an alcoholic who has seizures?** | Focal seizures; Status epilepticus; New-onset seizure |

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### PSYCHOPHARMACOLOGY

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<th>What class of medications are commonly used for short-term control of anxiety and agitation?</th>
<th>Benzodiazepines</th>
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<td><strong>What are some indications for the use of benzodiazepines?</strong></td>
<td>Short-term management of anxiety; Control seizures; Alcohol withdrawal; Induce muscle relaxation</td>
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<td><strong>Name two benzodiazepines commonly used in the ED setting for psychiatric emergencies?</strong></td>
<td>1. Lorazepam</td>
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<td></td>
<td>2. Diazepam</td>
</tr>
<tr>
<td><strong>What are some side effects of benzodiazepines?</strong></td>
<td>Impairment of motor coordination; Respiratory depression; Ataxia at higher doses; Potential for addiction</td>
</tr>
<tr>
<td><strong>Name two benzodiazepines that have potential for abuse?</strong></td>
<td>1. Diazepam</td>
</tr>
<tr>
<td></td>
<td>2. Alprazolam</td>
</tr>
<tr>
<td><strong>Is it possible to die from benzodiazepine withdrawal?</strong></td>
<td>Yes</td>
</tr>
<tr>
<td><strong>What are some advantages of using lorazepam in the acute setting for behavioral emergencies?</strong></td>
<td>Minimal cardiovascular depression; Does not inhibit or induce cytochrome isoenzymes; No active metabolites</td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
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<tr>
<td>What is the primary concern of using high-dose benzodiazepines (especially IV route)?</td>
<td>Respiratory depression</td>
</tr>
<tr>
<td>Are overdoses of benzodiazepines commonly fatal?</td>
<td>No—unless concomitant ingestion with other sedatives such as alcohol</td>
</tr>
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<td>What are some indications of neuroleptics?</td>
<td>Reduces aggression; Reduces psychotic thinking; Helps relieve anxiety</td>
</tr>
<tr>
<td>What is the primary mechanism of action of neuroleptics?</td>
<td>Antagonizes dopamine receptors in the mesolimbic area within the CNS</td>
</tr>
<tr>
<td>What are some side effects of neuroleptics?</td>
<td>Reflex tachycardia; Orthostatic hypotension; Can lower seizure threshold</td>
</tr>
<tr>
<td>What are some characteristics of haloperidol that make it an ideal neuroleptic to use in the ED?</td>
<td>Minimal cardiovascular effects; Effective at reducing agitation; Minimal sedation; Rapid onset; Synergistic with benzodiazepines</td>
</tr>
<tr>
<td>What side effect is common with haloperidol?</td>
<td>Dystonic reactions</td>
</tr>
<tr>
<td>What are some characteristics of atypical neuroleptics?</td>
<td>Effective for psychotic patients who are refractory to typical neuroleptics; Effective for negative symptoms; Less likely to cause tardive dyskinesia, but more likely to cause akathisia</td>
</tr>
<tr>
<td>Give some examples of atypical neuroleptics?</td>
<td>Olanzapine; Quetiapine; Clozapine</td>
</tr>
</tbody>
</table>
| What are some examples of extrapyramidal symptoms seen with antipsychotics: | Parkinsonism: Commonly within the first month of use; Characterized by cogwheel rigidity, akinesia, masked facies, and bradykinesia; Reducing the dose can help symptoms  
Dystonias: Painful clonus of voluntary muscles; Typically involves the face and neck; Commonly within the first month of use; Treatment is with diphenhydramine or benztropine  
Akathisia: Internal sense of motor restlessness; Most common form involves pacing and an inability to sit still; Propranolol is the medication of choice  
What is neuroleptic malignant syndrome (NMS)? | Rare, but life-threatening, idiosyncratic reaction to a neuroleptic medication |
What are some clinical features of NMS? Characterized by fever, muscular rigidity, altered mental status, and autonomic dysfunction

Which types of neuroleptic are commonly associated with NMS? Although potent neuroleptics (i.e., haloperidol) are more commonly associated with NMS, all antipsychotic agents, typical or atypical, may precipitate the syndrome

What is the diagnostic criteria of NMS? High fever with severe muscle rigidity and two or more of the following:
- Change in mental status;
- Tachycardia; Tremor; Leukocytosis;
- Metabolic acidosis; Labile or high blood pressure; Elevated CPK

What is the treatment of NMS? Commonly requires an ICU setting; Stop all neuroleptics; Benzodiazepines are the mainstay

Are there any emergent indications for the use of antidepressants in the ED? No—they require weeks to take effect

What class of antidepressants were among the first to be used to treat depression? Tricyclic antidepressants (TCAs)

Name some examples of TCAs? Nortriptyline; Amitriptyline; Imipramine

What is particular to know about TCAs? Have a very low therapeutic index

What are some side effects of TCAs? Anticholinergic, orthostatic hypotension, increased seizure risk, and have various cardiac effects

What class of antidepressants have a high therapeutic index and largely replaced TCAs? Selective serotonin reuptake inhibitors known as SSRIs

What are some examples of SSRIs? Sertraline; Citalopram; Paroxetine

What are some indications of SSRIs? Depression; Anxiety; Posttraumatic stress disorders; Obsessive-compulsive disorders

What are some side effects of SSRIs? Generally mild; Notable drug interactions; Toxic in only very high doses

What is serotonin syndrome? It is an idiosyncratic reaction that can occur with interactions between serotonergic agents such as SSRIs
What are some clinical features of serotonin syndrome:

- **Gastrointestinal**: Nausea, emesis, and diarrhea
- **Central nervous system**: Hyperreflexia, tremor, and altered MS
- **Autonomic instability**: Hyperthermia, diaphoresis, and orthostasis

What is the treatment of serotonin syndrome? Primarily supportive

Name a class of antidepressants associated with hypertensive crisis with the ingestion of tyramine-containing foods? Monamine oxidase inhibitors (MAOIs)

What are some tyramine-containing foods? Aged cheese; Wine; Beer; Fava beans

What are some clinical features of hypertensive crisis? Hypertension, chest pain, severe headache, tachycardia, and diaphoresis

What is the treatment of choice for hypertensive crisis? Phentolamine
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