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*The views expressed in this publication are those of the authors and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.
ACKNOWLEDGMENTS

Practicing medicine is like having a front row seat at the play of life. If that is true, and I believe it is, emergency medicine is like being back stage. This book is for the dedicated men and women who are often back stage making life and death decisions without a net knowing that the expectation is that they get it right the first time, every time, without blinking.

Siobhan, Jake, Gaby, Finn, for whom I exist, thank you for tolerating my passion of academic medicine.

RICHARD D. ZANE, MD

To all those from whom I continue to gain wisdom—my teachers, my colleagues, my students, and perhaps foremost, my patients. And to Devorah, Harry, Jake, and Judah, whose support means the world to me.

JOSHUA M. KOSOWSKY, MD
The practice of emergency medicine, like all disciplines, is changing and evolving. More than ever, the care of our patients depends upon having accurate, actionable, and accessible information in real time. Now in its fourth edition, *Pocket Emergency Medicine* remains the essential, go-to reference for busy clinicians on the front lines of emergency care. Unlike traditional texts, *Pocket Emergency Medicine* is designed to be used at the bedside, organized around presenting conditions and mirroring the thought process of clinicians: from history and physical exam to differential diagnosis testing; from testing and therapeutics to disposition. Clinical pearls and updates in medical practice are highlighted throughout the text.

This book was written by four dedicated emergency medicine residents from the University of Colorado and Harvard University and edited by senior faculty; the text has been updated and referenced in exacting detail, while retaining the fundamental ease of use so cherished by busy providers. We hope our readers find this edition of *Pocket Emergency Medicine* to be a valuable tool in their daily practice.

Richard D. Zane, MD  
Joshua M. Kosowsky, MD
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CHEST PAIN

Approach
• Immediate: All nontrivial CP get IV access, O₂, cardiac monitoring, ECG, CXR
  • Compare all ECGs to prior, repeat q15–20min if high suspicion for ACS; consider R-sided +/- posterior ECG if high suspicion (see Electrocardiography section)
  • History: Obtain thorough pain HPI (position, quality, radiation, severity, timing, associated sx, alleviating & exacerbating factors), cardiac risk factors (eg, for CAD, aortic dz, PE, etc.), prior cardiac testing (timing & results of last stress test, catheterization, echo) & prior cardiac events/procedures (eg, myocardial infarction [MI], CABG, valve repair, etc.)
  • Empiric tx: ASA 325 mg (if considering ACS & low suspicion for AoD), NTG for pain (unless R-sided ischemia, hypotension, PDE-inh)
  • Risk stratify for dxs being considered: ACS (TIMI, GRACE, or PURSUIT), PE (Well's), AoD (Aortic Dissection Detection risk score)

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Etiologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>ACS (UA/NSTEMI, STEMI), Prinzmetal's/cocaine-induced angina, myocarditis, pericarditis, cardiac tamponade, constrictive pericarditis, CHF/acute pulmonary edema, post-MI cx</td>
</tr>
<tr>
<td>Vascular</td>
<td>PE, AoD, thoracic aortic aneurysm, pulmonary HTN</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>PNA, PTX, pleural effusion/empyema, pleuritis, pulmonary infarct</td>
</tr>
<tr>
<td>GI</td>
<td>GERD, esophageal spasm, Mallory–Weiss tear, Boerhaave syndrome, PUD, biliary dz, pancreatitis</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>MSK strain/contusion, costochondritis, OA/radiculopathy</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Herpes zoster, anxiety, sickle cell chest crisis</td>
</tr>
</tbody>
</table>
**ELECTROCARDIOGRAPHY**

**Approach**
- Always check: correct pt, date, lead placement; calibration (mV, paper speed)
- Rate, rhythm, axis
- Waves (P, Q, R, T, U waves) & segments (PR, QRS, QT intervals, & ST segment)
- Conduction & bundle blocks
- Atrial enlargement, ventricular hypertrophy
- Ischemia/infarction
- Miscellaneous (stigmata of electrolyte abx, syncope, tox, PMs, PE, etc.)

### Orientation: ECG Calibration and Standardization

<table>
<thead>
<tr>
<th>Voltage calibration</th>
<th>• Standard ECG voltage is usually set w/ a calibration box encompassing 2 large vertical squares (10 mm tall) &amp; is equal to 1 mV (10 mm/mV): 1 small vertical box = 0.1 mV</th>
</tr>
</thead>
</table>
| Paper recording speed | • Standard ECG paper speed if usually set at 25 mm/s:  
  - Large horizontal box (5 mm wide) = 200 ms (0.2 s)  
  - Small horizontal box (1 mm wide) = 40 ms (0.04 s) |

### Determining Heart Rate \((nl = 60–100\) bpm\)

| Quick approach | • Count the number of bold vertical lines b/w adjacent R waves:  
  - 0 = 300 bpm, 1 = 150 bpm, 2 = 100 bpm, 3 = 75 bpm, 4 = 60 bpm, 5 = 50 bpm. |
| Mathematical approach | • Multiply the number of QRS complexes on the ECG by 6 (at a standard paper speed of 25 mm/s), each ECG records 10 s of activity. |

### Determining Rhythm (see also section on *Dysrhythmia*)

- Determining the heart’s rhythm is a complex process that requires synthesis of other features of ECG interpretation (esp rate, axis, intervals, & waves/segments)
- Key questions to help narrow the DDx of dysrhythmias include:
  1. Is the rate slow (eg, bradydysrhythmia) or fast (eg, tachydysrhythmia)?
  2. Is the QRS narrow (eg, SVT) or wide (eg, aberrancy, ventricular, electrolyte d/o)?
  3. Is the rhythm regular (eg, AFL, SVT, VT) or irregular (eg, AF, AFL w/ variable block, MAT, polymorphic VT)?
  4. Are P waves present? (If absent: AF vs. nodal/ventricular etiology)
5. Is every P wave followed by a QRS & every QRS preceded by a P wave?
6. For select tachydysrhythmias, is there response to vagal maneuvers or adenosine?

| **Determining Axis** (nl QRS axis = −30° to +90°) |
| --- | --- | --- |
| **Type** | **Definition** | **Causes** |
| L axis deviation | QRS b/w −30° & −90°  
- Lead I: Positive  
- Lead II: Negative | LVH, LBBB, inferior MI, LAFB, ventricular pre-excitation w/ posteroseptal accessory pathway (WPW) |
| R axis deviation | QRS b/w +90° & +180°  
- Lead I: Negative  
- aVF: Positive | RVH, lateral MI, LPFB, ventricular pre-excitation w/ free wall accessory pathway (WPW), COPD, dextrocardia |
| Extreme axis deviation | QRS b/w +180° & −90°  
(−QRS lead I, −QRS aVF) | Ventricular tachycardia, Hyperkalemia, apical MI, RVH |

| **ECG Waveforms and Segments** |
| --- | --- |
| **Type** | **Definition** |
| P wave | Represents atrial depolarization (1st half represents predominant R atrial depolarization & 2nd half L atrial depolarization); best seen in leads II & V1  
- NI: Duration <0.12 s, Amplitude ≤0.2 mV (frontal) or ≤0.1 mV (transverse), axis upright in I, II, aVF, & V2–V4 & inverted in aVR  
- Can be absent (AF, SVT), aberrant shape (AT, MAT, AFL) |
| PR interval | Represents time b/w onset of atrial depolarization (start of P) & onset of ventricular depolarization (start of QRS); isoelectric region represents conduction w/ AV node, bundle of His, bundle branches, & Purkinje fibers  
- NI: duration normally 0.12–0.20 s (120–200 ms) |
| Q wave | Defined as any initial negative deflection; represents onset of ventricular depolarization (specifically: L to R depolarization of septum)  
- NI: Small Qw can be ni in all leads EXCEPT V1, V2, V3; Large Q waves can be ni variant in Lead III & aVR  
- Pathologic Q waves: Any Qw V1–V3, Qw >0.04 s (1 mm) & ≥0.2 mV (2 mm); or any Qw >25% of QRS complex |
| R wave | Defined as any positive deflection w/ QRS; normally, Rw should become greater than Sw ~V3–V4 (called R-wave progression [RWP])  
- Pathology suggested by poor RWP (LVH, LBBB, LAFB, ant-MI, WPW, COPD, infiltrative d/o, etc.), early RWP/dominant Rw in |
| QRS complex | • Represents ventricular depolarization (1st half: septum & RV; 2nd half: LV)  
  • Nl duration 0.06–0.11 s (60–110 ms) measured in lead w/ widest QRS complex (see Causes of Abnl Interval Duration)  
  • Pathology suggested by prolongation (see Causes of Prolonged QRS below) or low voltage (R + S <0.5 mV in limb leads or <0.1 mV precordial; suggests presence of fluid [pericardial/pleural effusion], air [COPD], or excess fat/tissue [obesity, infiltrative CMP, myxedema]) |
| ST segment | • Represents plateau from end of ventricular depolarization (end of S) to start of repolarization (beginning of T); jxn of QRS & ST called J point  
  • Normally isoelectric w/ TP segment  
  • Pathology suggested by ST elevation (≥0.2 mV contiguous precordial leads, ≥0.1 mV limb leads, & ≥0.5 mV in R-sided & posterior leads) or depression (horizontal or downsloping depression ≥0.05 mV in 2 contiguous leads) (see Causes of ST Elevation) |
| T wave | • Represents ventricular repolarization  
  • Normally smooth & round morphology, positive in all leads except aVR; may be biphasic in V1/V2; amplitude generally 2/3 that of the R wave  
  • Pathology suggested by Tw inversions in I, II, aVL, V2–V6 (BBBs, LVH w/ strain pattern, Wellens’ sign, myocardial ischemia, myopericarditis, cardiac contusion, MVP, SAH, hypokalemia, digoxin effect) or peaked-Tw morphology (hyperkalemia, early myocardial ischemia) |
| U wave | • Small wave following T wave; represents prolonged repolarization of mid-myocardial layer cells “M cells”  
  • Nl amplitude <1.5 mm tall or ~10% on T-wave amplitude  
  • Pathology suggested by prominent Uw (hypokalemia/hypocalcemia, sinus bradycardia, LVH, MVP, hyperthyroid, etc.) |
| QT interval | • Measured from start of QRS complex to end of T wave; represents duration of electrical activation & recovery of ventricle  
  • Nl duration 390–450 ms in men; 390–460 ms in women (see Causes of Abnl Interval Duration) |

### Normal Intervals & Causes of Abnormal Interval Duration

<table>
<thead>
<tr>
<th>Type</th>
<th>Normal</th>
<th>Shortened</th>
<th>Prolonged</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR</td>
<td>120–200 ms</td>
<td>↑ sympathetic tone, 1° AVB, meds (digoxin,</td>
<td></td>
</tr>
</tbody>
</table>
### Conduction Delays

<table>
<thead>
<tr>
<th>Category</th>
<th>Definition</th>
</tr>
</thead>
</table>
| **RBBB** | 1. QRS duration ≥120 ms (≥100–120 ms = “incomplete” RBBB)  
2. Late intrinsicoid (R-wave peak time >0.05 s) M-shaped QRS (rsr', rSr', rSR') in V1–V2 ("rabbit ears")  
3. Early intrinsicoid, broad terminal slurred S wave in I, V5–V6  
Causes: AMI, Right-heart strain (PE, pHTN), myopericarditis, CMP, endomyocardial fibrosis, Chagas dz, CHD (ASD, VSD, ToF) |
| **LBBB** | 1. QRS ≥120 ms (≥100–120 ms = “incomplete” RBBB)  
2. Wide, notched R wave & absent Q wave in V5–V6, I, aVL  
3. Late intrinsicoid (R-wave peak time >0.06 s) in V5–V6  
4. Wide S wave in V1 w/ rS or QS complex  
Causes: Anterior AMI, LVH, CMP, hyperkalemia, digoxin tox |
| **LAFB** | 1. QRS duration ≤120 ms  
2. LAD (usually ≥−60°) |
3. QR pattern in I & aVL
4. rS pattern in II, III, & aVF
5. Late intrinsicon (R-wave peak time >0.045 s) in aVL
6. Increased QRS voltage in limb leads

Causes: Acute or remote MI, AS, OSA, CMP, endomyocardial fibrosis, Chagas dz, CHD

LPFB

1. QRS duration ≤120 ms
2. RAD (usually ≥+120°) w/o e/o RVH
3. rS pattern in I & aVL
4. QR pattern in II, III, & aVF
5. Late intrinsicon (R-wave peak time >0.045 s) in aVF

Causes: Acute cor pulmonale, CAD Lenègre’s dz, CMP, endomyocardial fibrosis, Chagas dz, hyperkalemia

Bifascicular block

2 of RBB, LAFB, & LPFB; can be complete or incomplete

Trifascicular block

All 3 of RBBB, LAFB, & LPFB; can be complete or incomplete (ie, incomplete trifascicular block can present w/ fixed block of both fascicles w/ e/o delayed conduction in remaining fascicle as in a 1° or 2° AVB)

Intraventricular conduction delay

1. QRS duration >110 ms
2. Typical waveforms of RBBB & LBBB not present

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial Abnormality and Ventricular Hypertrophy</td>
<td></td>
</tr>
<tr>
<td>Type</td>
<td>Definition</td>
</tr>
<tr>
<td>RAE (P pulmonale)</td>
<td>1. P ≥0.15 mV in V1/V2</td>
</tr>
<tr>
<td></td>
<td>2. P ≥0.25 mV in II or aVF</td>
</tr>
<tr>
<td></td>
<td>3. P-wave duration &lt;0.12 s</td>
</tr>
<tr>
<td></td>
<td>4. P-wave axis (&gt;75°–90°)</td>
</tr>
<tr>
<td></td>
<td>Causes: TR, PS; pHTN (eg, ILD, COPD, CHF); ASD, VSD</td>
</tr>
<tr>
<td>LAE</td>
<td>1. Terminal negative P wave in V1 &gt;0.04 s &amp; &gt;0.01 mV</td>
</tr>
<tr>
<td></td>
<td>2. Duration b/w peaks in P wave notches &gt;0.04 s (in II)</td>
</tr>
<tr>
<td></td>
<td>3. P-wave duration &gt;0.12 s</td>
</tr>
<tr>
<td></td>
<td>Causes: MS/MR, AS; CHF; HTN, HOCM</td>
</tr>
<tr>
<td>RVH</td>
<td>1. Right atrial enlargement</td>
</tr>
<tr>
<td></td>
<td>2. Right-axis deviation</td>
</tr>
<tr>
<td></td>
<td>3. S wave in I + Q wave in III</td>
</tr>
<tr>
<td></td>
<td>4. R in V1 &gt;0.7 mV or S in V5 or V6 &gt;0.7 mV</td>
</tr>
<tr>
<td></td>
<td>5. QR complex V1 or rR’ in V1 w/ R’ &gt;1 mV (w/ QRS duration</td>
</tr>
</tbody>
</table>
LVH

Sokolow–Lyon criteria:
- S wave in V1 + R wave in V5 or V6 ≥3.50 mV (sens 22%, spec 100%)
- R wave in aVL >0.9 (F) or >1.1 mV (M) (sens 11%, spec 100%)

Cornell voltage criteria:
- R wave aVL + S wave V3 >2 mV (women), >2.8 mV (men)
  (sens 42%, spec 96%)

Causes & Morphologies of ST Elevation *(NEJM 2003;349;2128–2135)*

<table>
<thead>
<tr>
<th>Differential</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>STEMI</td>
<td>Upward convex; coronary distribution; large T waves</td>
</tr>
<tr>
<td>Prinzmetal’s angina</td>
<td>As above (STEMI), but transient due to coronary spasm etiology</td>
</tr>
<tr>
<td>Myo/pericarditis</td>
<td>Upward concave; diffuse (can be regional); +/- PR depression</td>
</tr>
<tr>
<td>Massive PE</td>
<td>Inferior &amp; anteroseptal leads</td>
</tr>
<tr>
<td>LV aneurysm</td>
<td>Concave or convex; precordium common; +/- pathologic Q waves; smaller T waves compared to STEMI</td>
</tr>
<tr>
<td>LBBB</td>
<td>Concave, usually discordant w/ QRS</td>
</tr>
<tr>
<td>LVH</td>
<td>Concave, other features of LVH present</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td>Seen w/ other features of hyperkalemia</td>
</tr>
<tr>
<td>Brugada</td>
<td>Usually incomplete RBBB, RAD, rSR′ &amp; downsloping STE V1, V2</td>
</tr>
<tr>
<td>nl (esp young men)</td>
<td>Concave, seen in healthy young men, most marked in V2</td>
</tr>
<tr>
<td>Early repolarization</td>
<td>Most marked at V4, notching at J point; tall upright T waves present</td>
</tr>
<tr>
<td>Cardioversion</td>
<td>Seen 1–2 min after DCCV; can be markedly elevated</td>
</tr>
</tbody>
</table>

**ACUTE CORONARY SYNDROME**

**Overview**
- Approach to patient w/ angina sx: See section on *Chest Pain*
- **Chronic Stable Angina:** Substernal chest discomfort (pain, tightness, pressure) of less than 10-min duration, provoked by exertion or stress & alleviated by rest or NTG, & nonprogressive (ie, stable) over long
periods of time (see table; compare to unstable angina [UA])

- Chronic angina should be a dx of exclusion in ED (after reviewing recent stress or cath results), as pts often present to EDs b/c sx are worse in some capacity

<table>
<thead>
<tr>
<th>Canadian Cardiovascular Society (CCS) Grading of Angina Pectoris</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grade</strong></td>
</tr>
<tr>
<td>Grade I</td>
</tr>
<tr>
<td>Grade II</td>
</tr>
<tr>
<td>Grade III</td>
</tr>
<tr>
<td>Grade IV</td>
</tr>
</tbody>
</table>

- **Acute Coronary Syndrome:** Clinical spectrum of conditions ranging from UA through MI (NSTEMI & STEMI); due to vulnerable or high-risk plaque undergoing disruption of the fibrous cap causing thrombogenesis & ultimate imbalance b/w myocardial O₂ supply & demand (eg, tissue ischemia)

- **Myocardial Infarction** (see *Universal Definition*): death of myocardial cells due to myocardial tissue hypoxia, acutely causing release of intracellular cardiac biomarkers
- Once diagnosed, important to consider subtype & etiology (see tables below)
  - MI DDx is broad: not always 2/2 acute plaque rupture (see table below)
  - Elevated troponin not always MI: consider nonischemic etiologies (see table below)

<table>
<thead>
<tr>
<th>Universal Definition of Myocardial Infarction Classification System (JACC 2012;60(16):1581)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Summary of Criteria for Acute MI</strong></td>
</tr>
<tr>
<td>Detection of a rise &amp;/or fall of cardiac biomarker values (preferably cTn) w/ at least one value above the 99th percentile upper reference limit w/ at least one of the following:</td>
</tr>
</tbody>
</table>
**Symptoms:** Sxs of ischemia  
**ECG:** New or presumed new significant ST-T changes, LBBB, Qw  
**Imaging:** e/o new loss of viable myocardium or new regional wall motion abx  
**Pathology:** Identification of an intracoronary thrombus by angiography or autopsy

<table>
<thead>
<tr>
<th>Criteria for Prior MI</th>
</tr>
</thead>
</table>
| **ECG:** Pathologic Q waves w/ or w/o sxs in the absence of nonischemic causes  
**Imaging:** e/o loss of viable myocardium in the absence of nonischemic causes  
**Pathology:** Pathologic findings of a prior MI |

<table>
<thead>
<tr>
<th>Universal Classification of MI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type 1</strong></td>
</tr>
<tr>
<td><strong>Type 2</strong></td>
</tr>
<tr>
<td><strong>Type 3</strong></td>
</tr>
<tr>
<td><strong>Type 4a</strong></td>
</tr>
<tr>
<td><strong>Type 4b</strong></td>
</tr>
<tr>
<td><strong>Type 5</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differential for MI and Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes</strong></td>
</tr>
<tr>
<td><strong>Ischemic injury</strong></td>
</tr>
<tr>
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<tr>
<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td><strong>Nonischemic injury</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>History</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Typical symptoms of angina: Substernal pressure, pain, or tightness; often radiating to neck, jaw, or arm(s); precipitated by exertion &amp; relieved w/ rest or NTG</td>
</tr>
</tbody>
</table>
- Associated sx: dyspnea, diaphoresis, N/V, palpitations, LH
- Up to 23% of AMIs lack typical anginal sx (AJC 1973;32:1)
- Concerning features: new, at rest, or crescendo (frequency, severity, duration, ↓ threshold)

| Value of Specific Symptoms in Diagnosis of AMI (JAMA 2005;294:2623) |
|---------------------------------|--------------------|----------------|----------------|
| **Pain Descriptor**             | **LR (95% CI)**    | **Pain Descriptor** | **LR (95% CI)** |
| **Increased Likelihood of AMI** |                    | **Decreased Likelihood of AMI** |                    |
| Radiation: R arm/shoulder       | 4.7 (1.9–12)       | Described as pleuritic | 0.2 (0.1–0.3)    |
| Radiation: B/L arms/shoulders   | 4.1 (2.5–6.5)      | Described as positional | 0.3 (0.2–0.5)   |
| Exertional                      | 2.4 (1.5–3.8)      | Described as sharp   | 0.3 (0.2–0.5)   |
| Radiation to L arm              | 2.3 (1.7–3.1)      | Reproducible w/ palpation | 0.3 (0.2–0.4) |
| A/w diaphoresis                 | 2 (1.9–2.2)        | Inframammary location | 0.8 (0.7–0.9)  |
| A/w N/V                         | 1.9 (1.7–2.3)      | Nonexertional        | 0.8 (0.6–0.9)   |
| Worse w/ previous angina or similar to previous MI | 1.8 (1.6–2) | |
| Described as pressure           | 1.3 (1.2–1.5)      |                  |                |

**Physical Exam**
- Can be unremarkable unless c/b hypotension, heart block/arrhythmia, pulm edema
- Helpful for assessing for other causes of chest pain: bilateral UE BPs (AoD), lung exam (CHF, PTX, PNA), abdominal exam (biliary & pancreatic etiologies), chest wall ttp

**Evaluation**
- **ECG**: always check w/i 10 min, if sx change, at 6–12 h; always compare w/ baseline; if pain persists or changes present, always repeat q15–20min; always consider posterior ECG (leads V7–V9) in pts w/ non-dx initial ECG to r/o L circumflex STEMI
- Acute ischemia changes: ↑ or ↓ in ST or new TWI in anatomic distribution, new LBBB
- Old ischemic changes: Qw or PRWP (indicates presence of CAD even if no known hx)
- Sgarbossa criteria: Used to identify STEMI in the presence of old LBBB (see table)
Anatomic Distribution of ECG Findings Associated with AMI

<table>
<thead>
<tr>
<th>Anatomic Area</th>
<th>ECG Leads</th>
<th>Coronary Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal</td>
<td>V1–V2</td>
<td>Proximal LAD&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Anterior</td>
<td>V3–V4</td>
<td>LAD</td>
</tr>
<tr>
<td>Apical</td>
<td>V5–V6</td>
<td>Distal LAD, LCx, or RCA</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5–V6</td>
<td>LCx</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>aVR</td>
<td>L main CA</td>
</tr>
<tr>
<td>Inferior&lt;sup&gt;2&lt;/sup&gt;</td>
<td>II, III, aVF</td>
<td>RCA (~85%), LCx (~15%)</td>
</tr>
<tr>
<td>RV</td>
<td>V1–V2 &amp; V4R (most sens)</td>
<td>Proximal RCA</td>
</tr>
<tr>
<td>Posterior</td>
<td>ST depression V1–V2</td>
<td>RCA or LCx (obtain posterior leads)</td>
</tr>
</tbody>
</table>

<sup>1</sup>Wellen’s syndrome: Biphasic T waves in V2–V3; specific for critical prox LAD lesion

<sup>2</sup>Always obtain R-sided leads in inferior STEMI to evaluate for RV infarc

Sgarbossa Criteria for Identifying AMI in Presence of Old LBBB

<table>
<thead>
<tr>
<th>Criteria &amp; Points</th>
<th>Sens (%)</th>
<th>Spec (%)</th>
<th>Pos LR</th>
<th>Neg LR</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 pts: ≥1 mm STE concordant w/ QRS</td>
<td>73</td>
<td>92</td>
<td>9.5</td>
<td>0.3</td>
</tr>
<tr>
<td>3 pts: ≥1 mm STD in V1–V3</td>
<td>25</td>
<td>96</td>
<td>6.6</td>
<td>0.8</td>
</tr>
<tr>
<td>2 pts: ≥5 mm discordant w/ QRS</td>
<td>31</td>
<td>92</td>
<td>3.6</td>
<td>0.8</td>
</tr>
</tbody>
</table>


- Cardiac biomarkers: Troponin (I or T) preferred over CK-MB
  - Troponin: longer duration (↑ Sens) & higher specificity
  - CK-MB: only useful in addition to Tn if c/f new event w/i 1 wk from prior event in which +Tn (eg, return visit after recent PCI, MI, CABG, etc.)
    - Cardiac index: CI = (CK – MB/CK) × 100. CI <3 suggests skeletal source, CI 3–5 → indeterminate, CI >5 suggests cardiac source
  - Serial biomarker testing if signs/sx ACS: Perform repeat troponin at 3–6 h after arrival, & at 6 h (+/- 12 h) if intermediate- or high-suspicion of ACS; if positive, continue measuring until levels peak & downtrend (J Am Coll Cardiol 2014;64(24):e139–228)
  - If initial Tn positive (eg, CKD), Δ Tn > +20% suggests new myocardial
injury (if no AKI)

- Non-MI causes of elevated biomarkers: myopericarditis; drug toxicity; acute neurologic diseases (eg, ICH); myocardial contusion; myocardial O₂ supply-demand mismatch 2/2 tachyarrhythmia, CHF, HTN, hypotension, PE, sepsis, burns, respiratory failure

- **Special note on novel high-sensitivity troponin I assays:** HS TnI assays can detect TnI levels far earlier, but may also detect nonnecrosis processes (eg, nl apoptosis), & thus can even be positive even in some healthy individuals

- Single- & serial-HS-Tn protocols under investigation: Prelim studies suggest very high Sens at 0 h (99.6–100%) & 3 h (*Am Heart J* 2016;181:16–25; *Int J Cardiol* 2013;168(4):3896–3901); potentially helpful for ruling out AMI quickly

- Due to lower Spec, Δ in serial HS-Tn may have greater clinical significance than elevation itself for ACS (*J Am Coll Cardiol* 2014;64(24):e139–e228), though absolute elevations may have prognostic value (*J Am Heart Assoc* 2014;3(1):e000403)

### Characteristics of Cardiac Enzymes*

<table>
<thead>
<tr>
<th>Cardiac Enzyme</th>
<th>Initial Elevation</th>
<th>Peak</th>
<th>Return to Baseline</th>
<th>Sens @ 8 h (%)</th>
<th>Sens @ 12 h (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK-MB</td>
<td>4–6 h</td>
<td>18 h</td>
<td>2–3 d</td>
<td>91</td>
<td>93–95</td>
</tr>
<tr>
<td>Troponin I</td>
<td>3–12 h</td>
<td>24 h</td>
<td>1–2 wk</td>
<td>90</td>
<td>95–100</td>
</tr>
</tbody>
</table>

*A single set of cardiac enzymes cannot r/o MI (multiple sets cannot r/o ischemia w/o infarction)*

- **Other labs:** Chem 7, CBC, coags, T/S (if intervention planned), tox (if cocaine suspected)

- **CXR:** Useful to r/o other causes of CP; check lungs, cardiac silhouette, mediastinum

- **Transthoracic echo:** If ECG is not interpretable (prior LBBB, paced) & suspicion for ACS is high, can obtain TTE to assess for regional wall motion abnormalities; +WMA in pt w/ ongoing CP may suggest benefit from earlier PCI (*J Am Coll Cardiol* 2014;64(24):e139–e228)

- **Risk-stratification testing:** See section on *Risk Stratification Testing*

- Coronary CTA, exercise stress testing, stress echocardiography, nuclear stress testing
Treatment

- Give ASA if considering ACS & no CIs (50–70% drop in D/MI for UA/NSTEMI \((NEJM\) 1988;319(17):1105–1111); 23% drop in death in STEMI \((Lancet\) 1988;2(8607):349–360)
- **Chronic stable angina:** ASA (NNT = 50 in pts w/ known or suspected CAD), BP control, moderate- to high-intensity statin supported by mx RCTs \((NEJM\) 2016;374:1167–1176; \(Lancet\) 2009;373(9678):1849–1860)
- **ACS:** See \textit{UA/NSTEMI & STEMI} for details

Disposition

- Admit all STEMI, NSTEMI, & UA (see \textit{UA/NSTEMI & STEMI} for details)
- For patients w/ nondiagnostic hx, ECG, & biomarkers: Risk-stratify w/ HEART score
- HEART (score ≤ 3) > TIMI & GRACE in predicting major adverse cardiac events w/i 30 d (Sens 99%, NPV 98%) \((Int J Cardiol\) 2016;221:759–764; \(Int J Cardiol\) 2017;227:65–661)

\textbf{HEART Score for Chest Pain Patients in the ED} \((Neth Heart J\) 2008;16(6):191–196)
<table>
<thead>
<tr>
<th>History</th>
<th>Troponin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highly suspicious</td>
<td>≥3× NI limit</td>
</tr>
<tr>
<td>Moderate suspicious</td>
<td>&gt;1 to &lt;3× NI limit</td>
</tr>
<tr>
<td>Slightly/nonsuspicious</td>
<td>≤ NI limit</td>
</tr>
</tbody>
</table>

**ECG**

<table>
<thead>
<tr>
<th>ECG</th>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Significant ST depressions</td>
<td>≥3 risk factors**</td>
</tr>
<tr>
<td>Nonspecific repolarization</td>
<td>1 or 2 risk factors</td>
</tr>
<tr>
<td>NI</td>
<td>No risk factors</td>
</tr>
</tbody>
</table>

**Age**

<table>
<thead>
<tr>
<th>Age</th>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥65 y</td>
<td>2 pts</td>
</tr>
<tr>
<td>&gt;45 to &lt;65 y</td>
<td>1 pt</td>
</tr>
<tr>
<td>≤45 y</td>
<td>0 pts</td>
</tr>
</tbody>
</table>

**Total Score, Prognostic Value, and Disposition**

<table>
<thead>
<tr>
<th>Score</th>
<th>Prognostic Value</th>
<th>Disposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–3</td>
<td>2.5% MACE over next 6 wk</td>
<td>Discharge home w/ f/u</td>
</tr>
<tr>
<td>4–6</td>
<td>20.3% MACE over next 6 wk</td>
<td>Observation &amp; risk-stratification testing</td>
</tr>
<tr>
<td>7–10</td>
<td>72.7% MACE over next 6 wk</td>
<td>Admit for early catheterization</td>
</tr>
</tbody>
</table>

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**STEMI: Overview & Treatment**

**Definition**

- Acute complete occlusion of coronary artery (usually proximal) due to unstable thrombus, causing transmural ischemia & myocardial necrosis; characterized by angina usually at rest usually >30 min, ECG e/o ST elevations (see below for criteria), & +troponin

**ECG Criteria:** ≥0.2 mV precordial leads, ≥0.1 mV limb leads, & ≥0.5 mV in R-sided & posterior leads in at least 2 contiguous leads

**Treatment Approach**

- Initiate early medical therapies (ASA, heparin, nitrates prn, O₂ prn, analgesia)
Antithrombotic/adjunctive therapy should not delay transfer for pPCI

- Reperfusion: Immediate decision regarding availability of 1° PCI (see below)
- pPCI or transfer to PCI-capable hospital preferred for all pts **except** if time b/w 1st medical contact (FMC) & pPCI is expected >120 min
- Fibrinolysis may be preferred if delay to pPCI expected >120 min: after 120 min, no benefit of tfx to PCI-capable facility (Circulation 2011;124:2512–2521)
- Goal time from FMC to lysis: <30 min
- If FMC-to-pPCI time expected >120 min, consider: (J Am Coll Cardiol 2009;54(23):2205–2241)
  - Known CI to lysis (see below): pPCI preferred
  - Delay from sx onset (>3 h): pPCI preferred (lytics have ↓ efficacy w/ ↑ delays)
  - High-risk patient (shock, Killip class ≥3): pPCI preferred
  - Dx of STEMI in doubt (eg, AoD w/ RCA dissection): pPCI preferred
- If planning PCI, call cardiology/PCI lab as early as possible (potentially even before the pt arrives in the ED—if reliable pre-notification by EMS)
- If transferring for PCI, call for tfx early & ensure their door-to-balloon time is <90 min

Monitor & treat complications (eg, CHB, cardiogenic shock, pulm edema, arrhythmias)

### Adjunctive Medical Therapies (Fibrinolytics or pPCI)

- **Analgesia:** Morphine formerly used widely but may carry increased risk of adverse outcomes; use opioids only if absolutely needed (Am Heart J 2005;149(6):1043–1049)

- **O₂ supplementation:** No e/o benefit & may cause harm, possibly 2/2 free radical formation; use only in hypoxic pts w/ O₂ saturation <90% (Cochrane 2013;8:CD007160)

- **Nitrates:** No proven long-term mortality benefit, but may ameliorate sx; typical dose 0.4 mg SL q5min × 3; CI w/ ↓ BP, RV infarct, PD-inh w/i 24–48 h (Cochrane 2009;4:CD006743)

- **Anti-plt tx:** Always give ASA (162–325 mg PO/PR), 23% ↓ in death c/w placebo (Lancet 1988;2:349); additional benefit from other anti-plt agents (see table); all patients should be administered additional anti-plt agents either in the ED or cath lab
- **Antithrombotic tx:** See table for recommended regimens
- **Beta-blockers:** Early IV BB ↓ VT/VF & reinfarction acutely & ↑ LVEF in long term, but also ↑ acute cardiogenic shock (esp if >70 y/o, SBP <120 mmHg, HR >110 bpm); give oral BB w/i 24 h of STEMI; consider IV BB acutely if no CI or ongoing ischemia (*Int J Cardiol* 2013;168(2):915–921; 2017;228:295–302. COMMIT/CCS-2, *Lancet* 2005;366:1622)
- Other: Often started as inpatients include oral BBs, statins, ACE inh/ARBs

<table>
<thead>
<tr>
<th>P2Y$_{12}$ receptor inh (loading dose)</th>
<th><strong>Clopidogrel:</strong> 300 mg for pts ≤75 y/o, 75 mg for pts &gt;75 y/o; ↑ artery patency, ↓ MACE if give w/ ASA; consider deferring decision to cardiology if potential need for CABG (CLARITY-TIMI 28, <em>NEJM</em> 2005;352:1179; COMMIT, <em>Lancet</em> 2005;366:1607)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antithrombotic tx</td>
<td><strong>UFH:</strong> IV bolus 60 U/kg (max 4,000 U) then gtt at 12 U/kg/h (max 1,000 U), maintain aPTT ~50–70 s x 48 h or until revasc</td>
</tr>
<tr>
<td>Enoxaparin: If ≤75 y/o, 30 mg IV bolus, then 15 min later, 1 mg/kg SC q12h; if &gt;75 y/o, no bolus, 0.75 mg/kg SC q12h; if CrCl &lt;30 mL/min, 1 mg/kg q24h; continue 8 d or until revasc; no mortality diff c/w UFH, may ↓ recurrent MI &amp; need for urgent revasc, but also ↑ bleeding (<em>NEJM</em> 2006;354:1477–1488)</td>
<td></td>
</tr>
<tr>
<td><strong>Fondaparinux:</strong> Initial 2.5 mg IV, then 2.5 mg SC the following day; continue × 8 d or until revasc; especially useful if hx of HIT; CI if CrCl &lt;30; may ↓ mortality w/o ↑ bleeding c/w UFH (<em>JAMA</em> 2006;295(13):1519–1530)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Adjunctive Therapies for PCI in STEMI</strong> (<em>Circulation</em> 2013;127(4):529–555)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P2Y$_{12}$ receptor inh (loading dose)</td>
</tr>
<tr>
<td><strong>Prasugrel:</strong> 60 mg load; mild ↓ ischemic cx but ↑ bleeding c/w clopidogrel; best if young &amp; no need for surgery w/i 1 y; avoid if hx of CVA/TIA (<em>NEJM</em> 2007;357:2001; TRITON-TIMI, <em>Lancet</em> 2009;373:732)</td>
</tr>
<tr>
<td><strong>Ticagrelor:</strong> 180 mg load; mild ↓ mortality, MI, stroke c/w clopidogrel, but ↑ nonprocedural bleeding (eg, ICH) (<em>NEJM</em> 2009;361:1045–1057)</td>
</tr>
<tr>
<td>Antithrombotic tx</td>
</tr>
</tbody>
</table>
**Bivalirudin:** 0.75 mg/kg IV bolus, then 1.75 mg/kg/h infusion w/ or w/o UFH; preferred over UFH w/ GP IIb/IIIa inh in pts at high risk of bleeding; useful if hx of HIT

NOTE: ACCF/AHA Guideline for Mgmt of STEMI recommends GIIb/IIIa inh (Class IIa recommendation) in selected patients, though often performed in cath lab. Options include: Abciximab 0.25 mg/kg bolus, then 0.125 mcg/kg/min (max 10 mcg/min); Tirofiban (high-bolus dose): 25 mcg/kg IV bolus, then 0.15 mcg/kg/min, ↓ by 50% in CKD; Eptifibatide (double bolus): 180 mcg/kg IV bolus, then 2 mcg/kg/min; a 2nd 180 mcg/kg bolus given 10 min after 1st bolus, ↓ by 50% in CKD, avoid in dialysis pts.

## Fibrinolysis

- **Indications:** STEMI AND sx onset <12 h prior AND time b/w 1st medical contact & pPCI >120 min; may consider up to 24 h after sx onset if persistent sx, ongoing STE, rising troponin, hemodynamic instability, & pPCI unavailable
- **Goal:** Door-to-needle time should be ≤30 min
- **Benefits:** ~20% ↓ mortality in anterior MI or new LBBB; 10% ↓ mortality in IMI
- **Risks:** ICH (<1%), high-risk groups include elderly (~2% if >75 y), women, low weight
- Fibrin-specific lytic (front-loaded tPA) 14% ↓ mortality c/w SK (1% abs Δ; GUSTO, *NEJM* 1993;329:673) although ↑ ICH (0.7% vs. 0.5%); 3rd-generation bolus lytics easier to administer, but no more safe or efficacious

<table>
<thead>
<tr>
<th>Absolute CIs</th>
<th>Relative CIs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial neoplasm, aneurysm, AVM</td>
<td>Any known active intracranial path not listed w/ absolute contraindications</td>
</tr>
<tr>
<td>H/o intracranial hemorrhage</td>
<td>H/o iCVA &gt;3 mo prior</td>
</tr>
<tr>
<td>H/o intracranial/spinal surgery w/ 2 mo</td>
<td>Active PUD, pregnancy, or dementia</td>
</tr>
<tr>
<td>H/o i-CVA/closed head trauma w/ 3 mo</td>
<td>Current use of anticoagulants</td>
</tr>
<tr>
<td>Active internal bleeding or bleeding d/o</td>
<td>H/o trauma or major surgery w/ 3 wk</td>
</tr>
<tr>
<td>Suspected aortic dissection</td>
<td>H/o recent internal bleeding w/ 2–4 wk</td>
</tr>
<tr>
<td>Severe HTN (unresponsive to IV tx)</td>
<td>H/o severe poorly controlled HTN, or SBP &gt;180 or DBP &gt;110 on presentation</td>
</tr>
<tr>
<td><em>If considering using streptokinase: prior streptokinase use w/ 6 mo</em></td>
<td>Traumatic or prolonged CPR (&gt;10 min)</td>
</tr>
<tr>
<td></td>
<td>Noncompressible vascular punctures</td>
</tr>
</tbody>
</table>
Fibrinolytic Agents for STEMI *(Circulation 2013;127(4):529–555)*

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosing</th>
<th>Patency, 90 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tenecteplase (TNK-tPA)</td>
<td>Single IV weight-based bolus: &lt;60 kg (30 mg), 60–69 kg (35 mg), 70–79 kg (40 mg), 80–89 kg (45 mg), ≥90 kg (50 mg)</td>
<td>85%</td>
</tr>
<tr>
<td>Reteplase (rPA)</td>
<td>10 U + 10 U IV bolus given 30 min apart</td>
<td>84%</td>
</tr>
<tr>
<td>Alteplase (tPA)</td>
<td>Bolus 15 mg, infusion 0.75 mg/kg for 30 min (max 50 mg), then 0.5 mg/kg (max 35 mg) over 60 min; total dose not to exceed 100 mg</td>
<td>73–84%</td>
</tr>
</tbody>
</table>

**Indications for Transfer for PCI After Fibrinolysis**

- Cardiogenic shock or severe acute HF: Immediate tfx regardless of time from sx onset (can tfx even >48 h after MI); ↓ 6-mo mortality w/ immediate tfx; *(NEJM 1999;341:625–634)*
- Any pt: As part of an invasive strategy in stable pts after successful fibrinolysis; ideally PCI performed >3 h & <24 h after fibrinolysis; greatest benefit in high-risk pts
  - Routine angio ± PCI w/i 24 h of successful lysis: ↓ D/MI/Revasc *(Lancet 2004;364:1045)*

**Primary PCI *(NEJM 2007;356:47)*

- **Indications:** STEMI sx onset <12 h prior, STEMI sx onset >12 h if CI to fibrinolytics, or presence of severe acute HF, or cardiogenic shock; ongoing ischemia 12–24 h
- **Goal:** door-to-balloon <90 min by skilled operator at high-volume center
- **Benefits:** 27% ↓ death, 65% ↓ re-MI, 54% ↓ stroke, 95% ↓ ICH c/w lysis *(Lancet 2003;361:13)*
- PCI w/i 3 h of lytics in stable pts (w/o e/o failed re-perfusion) may cause harm *(Lancet 2006;367:569; Lancet 2006;367:579; FINESSE, NEJM 2008;358:2205)*

**Disposition**

- If no PCI available: transfer to PCI-capable center regardless of decision to use lytics
- If PCI available: admit to cath lab → CCU/cardiology
**Definition**

- **Pathogenesis:** Nonocclusive coronary thrombus on pre-existing plaque, causing dynamic & progressive obstruction, inflammation, & ischemia
  - Coronary lesion can be located proximally or distally for UA, usually distal for NSTEMI
- **Unstable Angina:** Any angina that is new-onset (if CCS III-IV severity), occurring at rest (if >20 min), or crescendo in nature (frequency, severity, duration, or more easily triggered), but *lacking* both ST elevations & positive troponin
- **NSTEMI:** Similar as UA, but characterized by positive troponin
- **ECG Criteria:** None; can have nl ECG, territorial STD, TWI, or NSSTW changes

**Treatment Approach**

- Initiate early medical therapies (ASA, heparin, nitrates prn, O₂ prn, analgesia)
- Determine risk using TIMI or GRACE risk scores to guide early vs. delayed timing of angiography (see section below) *(Eur Heart J 2005;26(9):865–872)*
- Monitor & treat complications (eg, CHB, cardiogenic shock, pulm edema, arrhythmias)

### GRACE Risk Score for UA/NSTEMI *(BMJ 2006;333(7578):1091)*

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>0 pts</th>
<th>25 pts</th>
<th>58 pts</th>
<th>80–89</th>
<th>91 pts</th>
<th>40–49</th>
<th>70–79</th>
<th>75 pts</th>
<th>≥90</th>
<th>100 pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>40–49</td>
<td>60–69</td>
<td>58 pts</td>
<td>80–89</td>
<td>91 pts</td>
</tr>
<tr>
<td>30–39</td>
<td>8 pts</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>50–59</td>
<td>70–79</td>
<td>75 pts</td>
<td>≥90</td>
<td>100 pts</td>
</tr>
<tr>
<td>≥90</td>
<td></td>
<td></td>
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<td></td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Heart Rate (bpm)</th>
<th>0 pts</th>
<th>24 pts</th>
<th>≥200</th>
<th>46 pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤50</td>
<td></td>
<td>70–89</td>
<td>9 pts</td>
<td></td>
</tr>
<tr>
<td>50–69</td>
<td>3 pts</td>
<td>110–149</td>
<td>15 pts</td>
<td>38 pts</td>
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<table>
<thead>
<tr>
<th>Systolic Blood Pressure (mmHg)</th>
<th>58 pts</th>
<th>24 pts</th>
<th>≥200</th>
<th>0 pts</th>
</tr>
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<tbody>
<tr>
<td>≤80</td>
<td>100–119</td>
<td>140–159</td>
<td>160–199</td>
<td>10 pts</td>
</tr>
<tr>
<td>80–99</td>
<td></td>
<td>120–139</td>
<td>140–159</td>
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<table>
<thead>
<tr>
<th>Killip Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (No heart failure)</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>II (Crackles in lower lung fields)</td>
</tr>
</tbody>
</table>

**Serum Creatinine Level (mg/dL)**

<table>
<thead>
<tr>
<th>Level</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–0.38</td>
<td>1 pt</td>
</tr>
<tr>
<td>0.80–1.19</td>
<td>7 pts</td>
</tr>
<tr>
<td>1.59–1.90</td>
<td>13 pts</td>
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<tr>
<td>≥4</td>
<td>28 pts</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Level</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.39–0.79</td>
<td>4 pts</td>
</tr>
<tr>
<td>1.20–1.58</td>
<td>10 pts</td>
</tr>
<tr>
<td>2.0–3.99</td>
<td>21 pts</td>
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**Cardiac Arrest at admx**

<table>
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<tr>
<th>Occurrence</th>
<th>Points</th>
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<tbody>
<tr>
<td>Yes</td>
<td>0 pts</td>
</tr>
<tr>
<td>No</td>
<td>39 pts</td>
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**ST-Segment Deviation**

<table>
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<tr>
<th>Severity</th>
<th>Points</th>
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<tr>
<td>Yes</td>
<td>0 pts</td>
</tr>
<tr>
<td>No</td>
<td>28 pts</td>
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**Troponin Elevation**

<table>
<thead>
<tr>
<th>Presence</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>0 pts</td>
</tr>
<tr>
<td>No</td>
<td>14 pts</td>
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**Risk Classification and Prognosis**

<table>
<thead>
<tr>
<th>Total score</th>
<th>Risk level</th>
<th>In-hospital death</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤100</td>
<td>Low risk</td>
<td>&lt;1%</td>
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<tr>
<td>101–170</td>
<td>Medium risk</td>
<td>1–9%</td>
</tr>
<tr>
<td>≥171</td>
<td>High risk</td>
<td>&gt;9%</td>
</tr>
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</table>

**TIMI Risk Score for UA/NSTEMI** *(JAMA 2000;284:825)*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥65 y</td>
<td>1 pt</td>
</tr>
<tr>
<td>≥3 RFs for CAD</td>
<td>1 pt</td>
</tr>
<tr>
<td>Known CAD (stenosis ≥50%)</td>
<td>1 pt</td>
</tr>
<tr>
<td>ASA use in past 7 d</td>
<td>1 pt</td>
</tr>
</tbody>
</table>

**Risk Classification and Risk of Death/MI/Urgent Revascularization w/i 14 d**

<table>
<thead>
<tr>
<th>Total score</th>
<th>14d Risk: D/MI/Urg Revasc (%)</th>
<th>Total score</th>
<th>14d Risk: D/MI/Urg Revasc (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1 pt</td>
<td>5</td>
<td>4 pts</td>
<td>20</td>
</tr>
<tr>
<td>2 pts</td>
<td>8</td>
<td>5 pts</td>
<td>26</td>
</tr>
<tr>
<td>3 pts</td>
<td>13</td>
<td>6–7 pts</td>
<td>41</td>
</tr>
</tbody>
</table>

**Adjunctive Medical Therapies**

- **Analgesia**: Morphine formerly used widely but may carry increased risk of adverse outcomes; use opioids only if absolutely needed *(Am Heart J 2005;149(6):1043–1049)*
- **O₂ supplementation**: No e/o benefit & may cause harm, possibly 2/2 free radical formation; use only in hypoxic pts w/ O₂ saturation <90% *(Cochrane 2013;8:CD007160)*
Nitrates: No proven long-term mortality benefit, but may ameliorate sx; typical dose 0.4 mg SL q5min × 3, continuous gtt if CP not improved w/SL (titrate until CP free; once CP free, titrate off); CI w/ ↓ BP, RV infarct, PD-inh w/i 24–48 h (Cochrane 2009;4:CD006743)

Anti-plt tx: Always give ASA (162–325 mg PO/PR), 23% ↓ in death c/w placebo (Lancet 1988;2:349); additional benefit from other anti-plt agents (see table)

If allergic to ASA: Clopidogrel 300–600 mg load (regardless of PCI approach)

If early invasive approach: Once decision made to proceed to PCI made, give Clopidogrel (600 mg), Ticagrelor (180 mg), or GP IIb/IIIa inh (eptifibatide IV, tirofiban IV) in addition to ASA; discuss w/ cardiology, may have greatest value if expected delay to PCI; further agents (P2Y12 inh, GPI) may have benefit when given peri-PCI in cath lab (J Am Coll Cardiol 2013;61(23):e179–e347)

If conservative approach: If PCI uncertain or not planned, give Clopidogrel (300 mg or 600 mg) or Ticagrelor (180 mg) in addition to ASA; reasonable to consider addition of GP IIb/IIIa inh (eptifibatide IV, tirofiban IV) unless low risk score &/or high bleeding risk (J Am Coll Cardiol 2013;61(23):e179–e347)

Inconclusive benefit from ED administration (vs. cath lab/inpatient unit) of DAPT in either early invasive or conservative approach, though generally earlier tx is better if UA/NSTEMI dx certain & low bleeding risk

Antithrombotic tx: See table for recommended regimens; continue until angiography (early invasive approach) or 48 h (conservative approach, if stress results indicate no need for angiography); hold anticoagulation if on warfarin until INR <2.0; anticoagulants have short-term benefit (UFH ↓ mortality 33–56% at 2–12 wk), but long-term benefit unclear, as dz process resumes once anticoagulation discontinued

Beta-blockers: Early IV BB ↓ VT/VF & reinfarction acutely & ↑ LVEF in long term, but also ↑ acute cardiogenic shock (esp if >70 y/o, SBP <120 mmHg, HR >110 bpm); give oral BB w/i 24 h of UA/NSTEMI; IV not routinely indicated (Int J Cardiol 2013;168(2):915–921. Int J Cardiol 2017;228:295–302. COMMIT/CCS-2, Lancet 2005;366:1622)

Other: Often started as inpatients include oral BBs, statins, ACE inh/ARBs
Antithrombotic Therapy in UA/NSTEMI

<table>
<thead>
<tr>
<th>Early invasive approach</th>
<th>UFH: IV bolus 60 U/kg (max 4,000 U) then gtt at 12 U/kg/h (max 1,000 U), maintain aPTT ~50–70 s x 48 h or until revasc</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Enoxaparin</strong>: If &lt;75 y/o, 30 mg IV bolus, then 15 min later, 1 mg/kg SC q12h; if &gt;75 y/o, no bolus, 0.75 mg/kg SC q12h; if CrCl &lt;30 mL/min, 1 mg/kg q24h; mild ↓ in nonfatal MI c/w UFH</td>
</tr>
<tr>
<td></td>
<td><strong>Bivalirudin</strong>: 0.75 mg/kg IV bolus, then 1.75 mg/kg/h infusion w/o UFH; preferred over UFH w/ GP IIb/IIIa inh in pts at high risk of bleeding</td>
</tr>
<tr>
<td>Conservative approach</td>
<td>UFH: IV bolus 60 U/kg (max 4,000 U) then gtt at 12 U/kg/h (max 1,000 U), maintain aPTT ~50–70 s x 48 h or until revasc</td>
</tr>
<tr>
<td></td>
<td><strong>Enoxaparin</strong>: If &lt;75 y/o, 30 mg IV bolus, then 15 min later, 1 mg/kg SC q12h; if &gt;75 y/o, no bolus, 0.75 mg/kg SC q12h; if CrCl &lt;30 mL/min, 1 mg/kg q24h</td>
</tr>
<tr>
<td></td>
<td><strong>Fondaparinux</strong>: Initial 2.5 mg IV, then 2.5 mg SC the following day; preferred if hx of HIT or ↑ bleeding risk</td>
</tr>
</tbody>
</table>

J Am Coll Cardiol 2013;61(23):e179–e347; 64(24):e139–e228

**Early Invasive vs. Conservative Approach** *(J Am Coll Cardiol)*

- Ultimately, approach decided by interventional cardiology based on multiple factors: risk score, procedural risks, recent angiography results, clinical stability & sx, individual pt goals, etc.
- **Early invasive approach**: Routine angiography w/i 72 h, urgency based on presentation:
  - *Immediate-invasive (PCI < 2 h)*: Any HD instability, VT/VF, HF/MVR, refractory angina
  - *Routine-invasive (PCI w/i 12–48 h)*: High-risk scores (TIMI ≥ 3, GRACE > 140), rising troponin, or new STD on ECG
  - *Delayed-invasive (PCI 25–72 h)*: Medium-risk (TIMI ≥ 2, GRACE 109–140) or high-risk scores (w/o rising troponin or STD); +/- hx of PCI w/i 6 mo, prior CABG, CKD, ↓ EF
- **Conservative (“selective invasive”) approach**: Best for initially stabilized pts w/o high-risk scores, ongoing symptoms, arrhythmias, heart failure; 2/2 marginal benefit but ↑ risks of early invasive approach *(Cochrane 2016;26(5):CD004815)*
  - Medical therapies (see above) for 48 h minimum, pre-discharge stress test
- Angiography only if recurrent ischemia, arrhythmias, heart failure, positive stress test
- Early invasive approach: In meta-analysis, no ↓ all-cause mortality/nonfatal MI, may ↓ risk of MI, refractory angina, & rehosp at 6–12 mo c/w conservative approach; however, also ↑ bleeding risk & procedure-related MI (Cochrane 2016;26(5):CD004815)
- Higher-risk pts benefit most from earlier angiography (J Am Coll Cardiol 2013;61(23):e179–e347; TIMACS, NEJM 2009;360:2165–2175) as reflected in guidelines above

**Disposition**
- Admission to CCU/cardiology based on risk, clinical stability, arrhythmia risk
- If UA, low-risk score, –Tn, nondiagnostic ECG: consider admitting to a CP/observation unit for serial troponin testing & stress testing; admx if recurrent sx, Δ ECG, +Tn

**Guidelines:** J Am Coll Cardiol 2013;61(23):e179–e347; 2014;64(24):e139–e228.

| Angiography Selection & Timing in UA/NSTEMI (J Am Coll Cardiol 2014;64(24):e139–e228) |
|---------------------------------|---------------------------------|
| Immediate (<2 h) invasive       | Hemodynamic instability         |
|                                 | Sustained VT or VF              |
|                                 | Signs or sx of HF or new or worsening mitral valve regurg |
|                                 | Refractory angina               |
| Selective (med tx, PCI prn)     | Low-risk score (eg, TIMI 0–1, GRACE <109) |
| invasive                        | Low-risk Troponin-negative female pts |
|                                 | Patient or clinician preference in absence of high-risk features |
| Early (<24 h) invasive          | None of above, but high risk (TIMI ≥ 3, GRACE score > 140) |
|                                 | Temporal change in Troponin     |
|                                 | New or presumably new STD on ECG |
| Delayed (25–72 h) invasive      | None of above but DM            |
|                                 | Renal insufficiency (GFR <60 mL/min) |
|                                 | Reduced LV systolic function (EF <0.40) |
|                                 | Early postinfarction angina     |
|                                 | PCI w/i 6 mo                    |
|                                 | Prior CABG                      |
|                                 | Medium-risk score (TIMI 2, GRACE 109–140) |

**RISK STRATIFICATION TESTING**
**Approach**

- **Definition:** Noninvasive eval for obstructive CAD in low-risk pts w/ acute CP/sx c/f ACS
  - Result usually qualitative ("positive" vs. "negative") for ischemia
- **Indications:** dx obstructive CAD, assess Δ clinical status in pt w/ known obstructive CAD, localize ischemia in pts w/ known symptomatic obstructive CAD
- **Contraindications:** severe acute illness, AMI w/i 48 h, high-risk UA, alternative critical dx (PE, AoD, myopericarditis, acute decompensated CHF, arrhythmias, severe AoS)
- Low-risk (HEART 0–3, TIMI 0–1, GRACE <109) pts may be safely discharged w/o stress testing if close f/u for outpatient stress testing can be arranged
- **ED stress testing in low-risk pts is low yield & high-cost** *(Am J Cardiol 2015;116(2):204–207)*
- 6-mo risk of MACE is low & may be unchanged regardless of whether pt receives stress in ED *(Int J Cardiol 2017;227:656–661; Crit Pathw Cardiol 2016;15(4):145–151)*

**Exercise Treadmill Testing**

- Patient runs on treadmill; monitoring includes ECG, symptoms, Δ hemodynamics (HR, BP)
  - "Diagnostic" test requires pt to achieve min of 85% of predicted HR (pHR = 220 − age)
- **Test characteristics:** 68% Sens, 77% Spec *(NEJM 2011;344(24):1840–1845)*
- **Benefits:** Lowest cost of risk stratification tests
- **Downsides:** requires nl resting ECG; ↓ Sens if low-risk pt; ↓ Sens & Spec in women; ↓ Sens if anti-ischemic drugs not d/c-ed (d/c BBs, digoxin, vasodilators, anti-HTN drugs ∼ 2 d prior to testing if possible)
- Duke Treadmill Score: weighted index of treadmill time, ECG chgs, induced angina sx
  - DTS = Duration of exercise in min − (5 * max STD in mm) − (4 * angina index)
  - Angina index: No angina (AI = 0), nonlimiting angina (AI = 1), limiting angina (AI = 2)

<table>
<thead>
<tr>
<th>Prognostic Value of Duke Treadmill Score in Exercise Stress Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DTS Risk</strong></td>
</tr>
</tbody>
</table>
### Pharmacologic Stress w/ Nuclear SPECT Imaging

- Ischemia induced by pharmacologic agents (dobutamine, adenosine, dipyridamole); radio-labeled tracers (e.g., sestamibi) enter myocardial cells & reflect regional perfusion; ↓ tracer uptake during stress that resolves w/ time suggests viable area of tissue ischemia; fixed defect suggest existing infarct
- **Note:** nuclear imaging can be performed after physical exercise as well
- **Test characteristics:** Adenosine SPECT Sens 90%, Spec 75%; dipyridamole SPECT Sens 89%, Spec 65%; dobutamine SPECT Sens 82%, Spec 75% (*Am Heart J* 2001;142(6):934–944)
- **Benefits:** Can use if abnl baseline ECG or unable to exercise; can localize ischemia
- **Downsides:** More expensive than exercise stress; ↓ Sens & Spec in women; ↓ Sens if anti-ischemic drugs not d/c-ed (d/c BBs, digoxin, vasodilators, anti-HTN drugs ∼2 dys prior to testing if possible)

### Pharmacologic/Exercise Stress w/ Echo Imaging

- Ischemia induced by pharmacologic agents (dobutamine, adenosine, dipyridamole) or exercise; echocardiography performed to assess for regional WMAs compared to rest
- **Test characteristics:** Adenosine Echo Sens 72%, Spec 91%; dipyridamole Echo Sens 70%, Spec 93%; dobutamine Echo Sens 80%, Spec 84% (*Am Heart J* 2001;142(6):934–944)
- **Benefits:** Can use if abnl baseline ECG or unable to exercise; can localize ischemia; can provide information re LVEF & valvular fxn
- **Downsides:** More expensive than exercise stress; ↓ Sens & Spec in women; ↓ Sens if anti-ischemic drugs not d/c-ed (d/c BBs, digoxin,
Coronary Computed Tomographic Angiography

- CT angiography of coronary arteries; images timed in conjunction w/ HR; assesses CAD burden & severity based on CA calcification; does not assess myocardial perfusion
- **Test characteristics:** Sens 85–99%, Spec 64–97%, NPV >95% (*Eur Heart J* 2016;37(30)2397–2405)
- **Benefits:** ↓ LOS & ↓ costs c/w conventional stress testing; especially useful for low-risk pts or intermediate-risk & nl serial ECGs/biomarkers, can evaluate global & regional LV fxn (*Circulation* 2006;114:1761; *JACC* 2006;48:1475; *NEJM* 2012;366(15):393; *NEJM* 2012;367(4):299)
- **Downsides:** Increased risk of downstream testing (2/2 ↓ Spec & detection of incidental findings), radiation exposure, requires relative bradycardia (often requires βB)
  - Radiation: 3× more radiation than ETT or stress echo, but equivalent to nuclear stress; more important to avoid in young pts & women; newer protocols are being designed to minimize radiation exposure (*Eur Heart J* 2016;37(30):2397–2405)
- Combination of single negative conventional troponin & negative coronary CTA has equivalent risk of 28 d MACE c/w conventional stress (*Eur Heart J* 2016;37(30):2397–2405)

**Disposition**

- Inadequate quality study: discuss case w/ cardiology
- Adequate quality + Neg result + Low-risk: d/c w/ f/u
- Adequate quality + Neg result + Int-risk: discuss case w/ cardiology, likely d/c w/ f/u
- Adequate quality + Pos result: discuss w/ cardiology, admit
- For adequate study w/ high-risk test results, consider coronary angiography, ± admission depending on clinical presentation

**Pearls**

- False-positives: Positive risk-stratification testing in a pt who presented to ED w/ CP does not necessarily mean CP was 2/2 by CAD; esp if low pre-TP & other causes possible
- False-negatives: Negative risk-stratification testing in a pt who presented to ED w/ CP does not necessarily mean CP was not 2/2 by CAD; esp if high pre-TP
CARDIAC CATHETERIZATION

Overview

- **Indications:** ACS (see STEMI & UA/NSTEMI above for timing of PCI); high-risk stress test result OR indeterminate-risk stress test result & high PreTP for obstructive CAD; ongoing angina despite tx; r/o CAD in pts w/ CP suspected from nonatherosclerotic etiology (ie, spasm) or systolic dysfxn suspected of nonischemic etiology (ie, ni-CMP); after ROSC in pts w/ cardiac arrest (see below for criteria)
  - Postarrest PCI recommended if STEMI &/or absence of mx unfavorable features (unwitnessed arrest, no bystander CPR, initial non-VF rhythm, >30 min to ROSC, ongoing CPR, noncardiac / traumatic arrest, pH <7.2, lactate >7, age >85, ESRD); decision individualized for each case *(J Am Coll Cardiol 2015;66(1):62–73)*

- Types of percutaneous coronary interventions:
  - Balloon angioplasty: Effective but ↑ risk of CA dissection & restenosis 2/2 remodeling
  - Bare metal stent: ↓ restenosis, repeat revasc, & MACE c/w BA, but may ↑ MI (most periprocedural), no Δ D *(Am Heart J 2006;151(3):682–689)*; requires DAPT × 4 wk & lifelong ASA thereafter
  - Drug-eluting stent: ↓ restenosis & repeat revasc c/w BMS, but no Δ D/MI over 6 y *(NEJM 2016;375;1242–1252)*; requires DAPT × 1 y & lifelong ASA thereafter

Post-PCI Complications

- **Bleeding (femoral access site):** Apply pressure, reverse/stop anticoag
- **Bleeding (retroperitoneum):** May c/o back pain, ±Hct drop, ↓ BP, ↑ HR; obtain abd/pelvic CT (I–); reverse/stop anticoag, consult IR/surgery
- **Vascular damage (pseudoaneurysm):** Pain, expanding mass, systolic bruit; obtain US; tx w/ manual compression, ± thrombin injection/surgery
- **Vascular damage (AV fistula):** May p/w sx 2/2 ↓ perfusion to LE (2/2 emboli, dissection, thrombus), continuous bruit, ↓ distal pulses; obtain US ± angiogram; consult card &/or surgery for repair (percutaneous or operative)
- **Renal failure:** Usually 2/2 contrast, occurs w/i 24 h, peaks 3–5 d
Stent thrombosis: P/w acute CP & STE; consult cards/cath lab, for urgent catheterization; may be more common in BMS than DES (JACC Cardiovasc Interv 2015;8(12):1552–1562); commonly 2/2 underexpanded stent, dissection, or d/c anti-plt Rx (JAMA 2005;293:2126). Stent stenosis: P/w subacute or chronic return of prior anginal sx months after PCI (but 10% p/w ACS); occurs 2° postprocedure remodeling, not atherosclerosis; despite advances, occurs still >10% cases (BA > BMS > DES) (BMJ 2015;351:h5392).

POST-MI COMPLICATIONS

Immediate Complications

- **LV systolic dysfunction/cardiogenic shock**: Common in L-sided (esp anterior) AMI; Dx w/ JVD, CXR or BSUS (B/L B-lines, ↓ EF); Tx w/ O₂ for hypoxia, ↓ preload (NTG SL → gtt), ↓ afterload (nitroprusside; IV ACE-I if Cl; avoid hydral 2/2 reflex ↑ HR), inotropy PRN (norepi > dopamine 2/2 fewer arrhythmias [NEJM 2010;362(9):779–789]; ± dobutamine esp if SVR high), diuretics, minimize PEEP (if intubated), emergent reperfusion (lytics/pPCI), may need IABP in cath lab (Lancet 2000;356(9231):749–756).

- **RV systolic dysfunction/cardiogenic shock**: Common in RV AMI; Dx w/ R-sided leads, BSUS (few B-lines, ↑ RV:LV ratio, dilated IVC); Tx w/ O₂ for hypoxia, ↑ preload (IVF until e/o nonfluid responsive; ongoing IVF may aggravate), ↓ PVR (bronchodilators, inh NO or prostacyclins), inotropy PRN (milrinone > norepi), minimize PEEP/TV (if intubated), emergent reperfusion (lytics/pPCI) (J Am Coll Cardiol 2010;56(18):1435–1446).

- **Tachyarrhythmias (eg, VT/VF, AF)**: Ischemia causes re-entry circuits in myocardium; place defibrillation pads on pt immediately on arrival; If unstable, tx per ACLS; If stable VT, tx w/ IV BB / membrane stabilization (amiodarone, metoprolol), check & replete lytes; emergent reperfusion (lytics/pPCI).

- **Bradyarrhythmias (eg, Heart block)**: Heart block can be 2/2 strong vagal tone (1° AVNB) &/or ischemia to AV node (1°–3° AVNB); place pacer pads on pt immediately on arrival; Tx w/ IVF (for BP), atropine, TC/TV pacing if unstable; emergent reperfusion (lytics/pPCI).

Early Complications
Infarct expansion, re-infarction, postinfarction ischemia: Usually w/i 4 d of MI; can present similarly to initial MI but diagnostics subtler: Δ ECG (2/2 nl evolution from prior MI vs. new ischemia), +Tn (may be ↓ from prior MI; ↑ suggests new ischemia), +CK-MB (suggests new ischemia); tx as w/ ACS; discuss w/ cardiology; depending on prior mgmt (pPCI vs. lytics; BA vs. BMS/DES), may need pPCI

Ventricular wall rupture: Usually w/i 2–7 d after MI; RFs include ↑ age, female, anterior infarct, ↑ wall strain (↑ HR, ↑ afterload); occur at jxn of nl tissue & infarct

Free wall rupture: Rapid bleeding into pericardium causing s/sx of tamponade; Tx w/ IVF/blood; emergent pericardiocentesis & cardiac surgery; mortality >90%

Pseudoaneurysm: Bleeding contained w/i myocardial wall; may p/w arrhythmias, heart failure, systemic embolization, or be asx & dx’ed only on imaging; once identified, c/s cardiology & cardiac surgery

Septal rupture: May be asx or p/w sx of L → R shunt & ↓ L-sided CO (angina, shock, pulm edema); new pansystolic murmur; dx by echo; tx w/ urgent surgical closure

Papillary muscle rupture: Usually w/i 7 d of MI; frequency i-MI & p-MI > a-MI; p/w sx of acute pulmonary edema, pansystolic murmur; BSUS differentiate from post-MI VSD; tx w/ ↓ preload & afterload (nitroprusside), diuretics, O₂, IABP, emergent surgical repair

Pericarditis: Usually w/i 7 d of MI; most common w/ a-MI; p/w low-grade fever, chest pain, friction rub; ECG w/ diffuse STE w/o reciprocal chgs; BSUS ± pericardial effusion; tx w/ NSAIDs; NOTE: early pericarditis is distinct from Dressler’s syndrome (below)

Delayed Complications

Left ventricular aneurysm: Suspect if ECG w/ persistent STE post-MI; can p/w HF, embolic sx, arrhythmias; dx w/ echo; c/s cardiology (reperfusion), cardiac surgery

Left ventricular thrombus: Most common in a-MI; RFs include ↓ EF, severe MVR, LV aneurysm (eg, slow, nonlaminar flow); tx w/ anticoagulation

Dressler’s syndrome: Usually 2–10 wk after MI; presumed autoimmune-mediated; p/w fever, chest pain, pleurisy; BSUS w/ pericardial & pleural effusions; self-limited w/ NSAIDs
PRINZMЕTAL’S (VARIANT) ANGINA

Overview

- **Definition:** Distinct syndrome of ischemic CP classically occurring at rest 2/2 focal coronary artery spasm, & a/w transient STE; exact etiology unknown
- Most vasospasm occurs in areas of pre-existing stenosis
- Can be a/w infarction, arrhythmia, & sudden cardiac death; consider in all pts w/ healthy SCD, particularly if arrest occurred in morning or cold settings

History

- Often young (35–50 y/o), F > M, tobacco use, EtOH use; PMH/FHx migraine, Raynaud’s, pericarditis, MV prolapse; may have no known cardiac hx but CAD not uncommon
- Sxs include substernal pressure radiating to jaw & arm; can respond to NTG; often occur midnight to early AM (↑ vagal tone), or after hyperventilation or cold
- May be a/w marked diurnal variation in exercise tolerance (↓ tolerance in AM, ↑ in PM)

Evaluation

- EKG reveals transient territorial STE & reciprocal ST Δs; may induce a variety of conduction disturbances or arrhythmias
- Stress testing may induce no ST Δs, STDs, or STEs, or STEs may be seen during recovery phase of stress testing
- Dx definitively w/ angiography & provocative intracoronary ACH &/or ergot derivative (>90% Sens, >90% Spec; even better if combined); noninvasive approach w/ hyperventilation & exercise (65% Sens, >90% Spec) *(J Am Coll Cardiol 2013;63(2):103–109)*

Treatment

- High-dose CCB (nifedipine, verapamil, diltiazem), nitrates (SL prn); d/c smoking

Disposition

- Admit, given risk of MI & arrhythmia during acute episodes
COCAIN-INDUCED ANGINA

Overview
- Definition: Anginal sx occurring after cocaine use, 2/2 ↑ myocardial O₂ demand (↑ HR, ↑ afterload, ↑ contractility & end-systolic wall stress) & ↓ O₂ supply (vasoconstriction); generally not 2/2 acute thrombosis, though cocaine a/w premature CAD/ACS
- Overall incidence of cocaine-associated MI is 0.7–6% of those presenting w/ CP after cocaine (Acad Emerg Med 2000;36:469; COCHPA, Acad Emerg Med 1994;1:330)
- Can be c/b arrhythmia & heart failure (~90% occur w/i 12 h of presentation)

History
- CP that may be a/w dyspnea, anxiety, palpitations, diaphoresis, dizziness, or nausea
- Sxs typically occur w/i 3 h of ingestion, but cocaine metabolites may persist up to 24 h to cause delayed or recurrent vasoconstriction
- RF for cocaine-induced MI: Male gender, current smoker, non-white

Evaluation
- Similar to ACS (see above)
- Maintain high index of suspicion for aortic dissection as well
- Urine toxicology: Usually detects cocaine metabolite benzoylecgonine (urine t₁/₂ of 6–8 h) up to 24–48 h (range 16–66 h); however, chronic cocaine users may have detectable levels for weeks after last use

Treatment
- Given risk of MI, tx similarly to ACS (see ACS, “Adjunctive Medical Therapies”)
  - ASA, analgesia PRN, O₂ PRN, NTG PRN, antithrombotic tx all as per ACS guidelines
  - Avoid BB given risk of unopposed α-adrenergic effect (↑ CA vasospasm, ↑ BP)
  - IV Benzodiazepines (↓ central stimulatory effects of cocaine)
  - IV Anti-HTN (NTG, sodium nitroprusside, phentolamine; avoid BB)
  - If STEMI: pPCI preferred over lytics 2/2 ↑ ICH risk after cocaine
VT/VF immediately after cocaine is 2/2 local anesthetic (Na channel) effect & may respond to sodium bicarbonate tx in addition to standard therapies

Disposition

- Admit: If +Tn, ongoing CP, persistent unstable VS
- EDOU: If sx & VS controlled, -Tn, nonischemic ECG; no difference in 30-d outcomes if pts w/ & w/o stress-testing, consider if CAD RFs & poor f/u (Circulation 2008;117:1897–1907)
- Provide drug-abuse counseling to all pts prior to d/c

DVT AND PULMONARY EMBOLISM

Overview

- Definition: In situ thrombosis of LE/UE deep veins, often provoked by stasis/turbulence, hypercoagulability, endothelial injury (Virchow’s triad)
- RFs: Hypercoagulable state (cancer, pregnancy, OCPs, APLAS); recent surgery or trauma; prolonged immobilization; venous outlet obstruction; excess extremity use (eg, sports, occupation; for UE DVT); increased age; obesity; FHx of DVT/PE
- Lower-extremity DVT: Comprise 90% of DVTs; but as many as ~50% may be isolated distal DVT of the calf (only require tx if severe sx or propagating; see below)
- Upper-extremity DVT: Comprise the minority (10%) of DVTs; c/w LE DVT, ↓ risk of PE (6% vs. 15–32%), ↓ risk of recurrence (2–5% vs. 10%); can be 1° (20%) or 2° (80%) (NEJM 2011;364(9):861–869; Circulation 2012;126;768–773)
  - Primary: thoracic outlet compression of SC vein (eg, ribs, clavicle), microtrauma to SC vein from repeat UE movements (often young, athletes or occupation), idiopathic
  - Secondary: catheter-associated, cancer-associated (hypercoag, compression) surgery (immobilization, endothelial trauma), systemic hypercoag state (preg, etc.)
  - Management based on location (proximal vs. distal), depth (deep vs. superficial)
History & Physical Exam

- HX: May have unilateral discomfort, swelling, paresthesia, weakness, erythema, warmth
  - Always ask about RFs; obtain ROS to assess for s/sx of concurrent PE
- EX: Physical exam notoriously insensitive for DVT (*JAMA* 1998;279:1094–1099)

Evaluation

- Use Well’s score to determine pre-TP of DVT (see table)

<table>
<thead>
<tr>
<th>Well's Criteria for DVT</th>
<th>D-dimer Sensitivity (%)</th>
<th>Prevalence of DVT (%)</th>
<th>NPV of D-dimer (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active cancer (tx ongoing or w/i 6 mo or palliative)</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paralysis, paresis, or recent immobilization of LE</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recently bedridden &gt;3 d or major surgery w/i 4 wk</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Localized tenderness along deep venous system</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entire leg swelling</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calf swelling by &gt;3 cm c/w asx (10 cm below tibial tuberosity)</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pitting edema (greater in symptomatic leg)</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collateral superficial veins (nonvaricose)</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previously documented DVT</td>
<td>1 pt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alternative dx as likely or greater than that of DVT</td>
<td>−2 pts</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RFs not incorporated into score but a/w inc risk of DVT include FHx of DVT (>2 1st-degree relatives), hospitalization w/i 6 mo; erythema.

- UE DVT: Compression US (Sens 97%, Spec 96%); if negative US but high pre-TP, obtain serial US or D-dimer (Sens 100%, Spec 14%) (*NEJM* 2011;364(9):861–869)
- LE DVT (initial eval): lab & imaging directed based on pre-TP (see
Algorithms below)

- **D-dimer:** Assays include enzyme-linked immunofluorescence assays (Sens 96%, Spec 46%), microplate ELISA (Sens 94%, Spec 53%), immunoturbidimetric (Sens 93%, 53%), whole-blood assay (Sens 83%, Spec 71%), & quantitative latex agglutination assays (Sens 95%, 53%) (Chest 2012;141(2Suppl):e351S–418S)

- **Lower-extremity ultrasound (two types: Proximal compression, Whole-leg)**
  - **WL-US:** Superior to PC-US alone in detection of DVT (mostly distal)
  - Increased chance of dx isolated distal DVT of unclear significance (see Tx)
  - **PC-US:** should be used w/ D-dimer to increase sensitivity (see Algorithm)
  - If PC-US neg & D-dimer pos, repeat PC-US in 1 wk
  - 3-mo rate of PE after negative WL-US: 0.3% (low pre-TP), 0.8% (mod pre-TP), 2.5% (high pre-TP) (Chest 2012;141(2Suppl):e351S–418S)
  - No difference c/w combined PC-US + D-dimer (& prn repeat PC-US): No difference in 3-mo risk of PE (0.6%) (Chest 2012;141(2Suppl):e351S–418S)

- **CT venography:** Highest sensitivity but risk of radiation & contrast; use selectively if mod or high pre-TP, positive PC-US & D-dimer but unable to obtain WL-US or repeat PC-US in 1 wk (Chest 2012;141(2Suppl):e351S–418S)

- **LE DVT (recurrent):** Risk of false-positives (2/2 scarring, postthrombotic syndrome) high
  - Recommended approach: Combined D-dimer (usually nl w/i 3 mo of starting tx for DVT) & PC-US; if PC-US neg or undiagnostic, repeat in 1 wk (if unable: CTV, MRV)
Figure 1.2 Diagnosis and Treatment of DVT, Low Pre-Test Probability. (Chest 2012;141(2)(suppl):e351S)
Figure 1.3 Diagnosis and Treatment of DVT, Mod Pre-Test Probability. (A) Starting with D-dimer assessment, and (B) Starting with ultrasonography (Chest 2012;141(2) (suppl):e351S)
Treatment

- UE DVT: Anticoagulation × 3–6 mo, though comparative data lacking on specific regimens
  - Cather-associated DVT: Catheter removal only indicated if catheter malfxn or infxn, no further need for catheter, or strong CIs to systemic A/C (NEJM 2011;364(9):861–869)
  - Isolated basilic/cephalic vein thrombosis: very low risk of PE, no A/C required
- LE DVT: Anticoagulation × 3–6 mo unless strong CIs (Chest 2012;141(2) (Suppl):e419S–e494S)
  - Anticoagulation regimen should be selected based on comorbidities, ability to take PO medications, patient preference (monitoring, etc.), risks of bleeding:
    - SC LMWH (1 mg/kg BID; renally dose): slight ↓ risk of death, recurrence, major bleeding c/w UFH; preferred w/ malignancy; relative CIs include CKD & obesity
    - SC Fondaparinux (5 mg QD [<50 kg], 7.5 mg QD [50–100 kg], 10 mg QD [>100 kg]; renally dose): Similar risk of death, recurrence,
major bleeding c/w LMWH; preferred w/ hx of HIT
- IV UFH (80 U/kg bolus, 18 U/kg/h gtt): As above, may be preferred over LMWH if CKD/ESRD; risk of HIT higher than LMWH
- PO Warfarin (INR 2.0–3.0): Bridge w/ LMWH/Fondaparinux until INR therapeutic
- PO Rivaroxaban (15 mg BID x 3 wk, 20 mg QD thereafter)
- PO Apixaban (10 mg BID x 7 d, 5 mg BID thereafter)
- Duration of treatment generally depends if provoked (3 mo) or nonprovoked (6 mo if no bleeding risk, 3 mo if bleeding risk)
- If strong contraindications to A/C: SVC filter until bleeding risk resolves
- Isolated distal DVT: Tx as above if severe sx or e/o extension on repeat U/S (1–2 wk)

**Complications**

- **Phlegmasia alba dolens:** Emergent complication; P/w swollen white leg 2/2 extensive DVT obstructing collaterals (but not involving them), largely impeding arterial inflow; Tx w/ IV Heparin (in case need for surgery), +/- catheter-directed thrombolytic tx

- **Phlegmasia cerulea dolens:** Emergent complication; P/w severely swollen cyanotic leg 2/2 extensive DVT including thrombosis of collaterals & capillary beds, fully impeding arterial flow, causing massive fluid sequestration in affected limb (2/2 hydrostatic pressure), circulatory shock, death (20–40% cases); Tx w/ IV Heparin (in case need for surgery), catheter-directed thrombolytic tx, aspiration thrombectomy, or open surgical thrombectomy

---

**PULMONARY EMBOLISM**

**Overview**

- Definition: embolization of systemic venous thrombus into pulmonary arterial system
- Diff from amniotic fluid embolism (RF: peripartum) & fat embolism (RF: long bone fx)
- RFs: See section on *DVT* above; Major identifiable RFs include recent surgery (OR 21.0), trauma (OR 12.7), immobility (hosp or nursing home) (OR 8.0), cancer (OR 4.1–6.5), paraplegia (OR 3.0), estrogen tx (OR 3.0) (*JAMA* 2003;290(21):2849–2858)
**Approach**

- IV access (if PE), ECG, O₂ prn, Monitor, CXR to r/o alternative dx
- If HD stable: diagnostic tests depending on pre-TP
- If unstable, consider empiric antithrombotic tx ± lysis if potential benefit > bleeding risk

**History & Physical Exam** *(Chest 1991;100:598; Am J Card 1991;68:1723)*

- HX: Dyspnea (73%), pleuritic CP (66%), cough (37%), syncope, ↓ BP, PEA
- Assess PreTP: May use PERC (to decide whether any testing is necessary) or Wells criteria (to decide whether D-dimer is sufficient w/u)
- EX: Unexplained ↑ HR, ↑ RR, ↓ SpO₂, fever, JVD

<table>
<thead>
<tr>
<th>PERC Criteria for Pts w/ Low Risk of PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥ 50</td>
</tr>
<tr>
<td>Recent trauma or surgery or hosp w/i 4 wk</td>
</tr>
<tr>
<td>HR ≥ 100</td>
</tr>
<tr>
<td>Hemoptysis</td>
</tr>
<tr>
<td>O₂ Sat on room air &lt;95%</td>
</tr>
<tr>
<td>Exogenous estrogen</td>
</tr>
<tr>
<td>Prior hx of DVT/PE</td>
</tr>
<tr>
<td>Unilateral leg swelling</td>
</tr>
</tbody>
</table>

**Using the PERC Criteria:** If any of above criteria present, PE cannot be r/o PE w/o additional dx tests. If all criteria negative, PE unlikely (Sens 97.4%, Spec 21.9%). *(Thromb Haemost 2008;6:772)*

<table>
<thead>
<tr>
<th>Wells Criteria for PE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Criteria</strong></td>
</tr>
<tr>
<td>Clinical signs &amp; sx of DVT (OR 5.8)</td>
</tr>
<tr>
<td>Tachycardia &gt;100 bpm (OR 3.0)</td>
</tr>
<tr>
<td>Immobilization or surgery w/i 4 wk (OR 2.5)</td>
</tr>
<tr>
<td>Previous DVT/PE (OR 2.5)</td>
</tr>
<tr>
<td>Hemoptysis (OR 2.4)</td>
</tr>
<tr>
<td>Malignancy (OR 2.30)</td>
</tr>
<tr>
<td>PE more likely than alternative dx (OR 4.6)</td>
</tr>
</tbody>
</table>

**Cut-off for “PE unlikely” (sum of points)**  
- Original: ≤4 pts  
- Modified: ≤2 pts  
- Simplified: ≤1 pt

**Rates of PE Based on Score**

<table>
<thead>
<tr>
<th></th>
<th>Original</th>
<th>Modified</th>
<th>Simplified</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>


Rates of PE Based on “PE Unlikely” Score & Negative D-dimer

<table>
<thead>
<tr>
<th>Score Range</th>
<th>Original (%)</th>
<th>Modified (%)</th>
<th>Simplified (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2 pts</td>
<td>11.5%*</td>
<td>11.0%*</td>
<td></td>
</tr>
<tr>
<td>&gt;2 pts</td>
<td>37.3%**</td>
<td>35.8%**</td>
<td></td>
</tr>
</tbody>
</table>

*Order D-dimer to r/o out PE.

Revised & Simplified Geneva Score for PE

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Revised</th>
<th>Simplified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;65</td>
<td>1 pt</td>
<td>1 pt</td>
</tr>
<tr>
<td>Previous DVT/PE</td>
<td>3 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>Surgery (under GA) or fx (LE) w/i 1 mo</td>
<td>2 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>Active malignancy or cure &lt;1 y</td>
<td>2 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>Unilateral LE pain</td>
<td>3 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>2 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>HR 75–94</td>
<td>3 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>HR ≥ 95</td>
<td>5 pts</td>
<td>1 pt</td>
</tr>
<tr>
<td>Pain w/ LE deep vein palpation &amp; unilateral edema</td>
<td>4 pts</td>
<td>1 pt</td>
</tr>
</tbody>
</table>

Cut-off for “PE unlikely” (sum of points) NA ≤2 pts

Rates of PE Using Simplified Geneva Score

<table>
<thead>
<tr>
<th>Score Range</th>
<th>≤1 pt (%)</th>
<th>2–4 pts (%)</th>
<th>5–7 pts (%)</th>
<th>0–2 pts (%)</th>
<th>3–7 pts (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate of PE</td>
<td>8</td>
<td>29</td>
<td>64</td>
<td>13</td>
<td>42</td>
</tr>
<tr>
<td>Rate of PE if D-dimer neg</td>
<td>1</td>
<td>3</td>
<td>12</td>
<td>1–3**</td>
<td>5–14***</td>
</tr>
</tbody>
</table>

*Order D-dimer in “PE Unlikely” to r/o PE.
**Range of rates includes both high- & low-sensitivity D-dimer.
***Order imaging (eg, CTA, V/Q) in “PE Likely” to r/o PE. Arch Intern Med 2008;168(19):2131–2136.

Evaluation

- ECG (Sinus tach, S1Q3T3 not sens/spec, diffuse TWI), CBC, PT/PTT, Cr
- CXR: R/o other dx; “classic” PE findings (Hampton’s hump, Westermark’s sign) not Sens/Spec
Patients w/ low clinical gestalt for PE & negative PERC score may not need D-dimer
- Combined low gestalt & PERC negative: Sens 97.4%, Spec 21.9%
- If unable to r/o by PERC criteria, compute Well’s score (or modified Geneva)
- Know your hospital’s D-dimer test characteristics; wide variation among assays
- False-positive D-dimer: Pregnancy, trauma, infection, malignancy, inflammatory conditions, surgery, ↑ age, SCD, AF, ACS, CVA, acute UGIB, DIC
- If “PE likely” by Well’s score or D-dimer positive, obtain additional testing:
  - Bedside Ultrasound: Echo for RV dilatation (RV:LV >1) or dysfxn (hypokinesis, paradoxical septal wall motion, McConnell’s sign) can suggest dx but not r/o (Sens 50%, Spec 98%, PPV 88%, NPV 88%) (Ann Emerg Med 2014;63(1):16–24); combined thoracic & LE ultrasound can reduce need for CTA by dx’ing DVT or suggesting alternative dx (Chest 2014;145(4):818–823)
  - CT angiography (CTA Sens 83%, CTA/CTV Sens 90%, Spec 95%): (NEJM 2006;354:2317)
    - May miss small/subsegmental PEs (of uncertain clinical significance if asx & no further clot burden on LENIs); If negative for PE but suspicion is high, consider additional test (D-dimer, US, pulmonary angiogram); requires dye load (relative CI if CrCl <50 NEJM 2006;354:379)
  - V/Q scan (if CI to CTA): Requires nl baseline CXR; tx if results are high-probability; 2/3 of all cases may result in low/intermediate probability
  - MR angiography (MRA Sens 78%, Spec 99%; MRA/MRV Sens 92%, Spec 96%): Use in pts w/ CI to CTA; high proportion of studies limited qual (Ann Intern Med 2010;152(7):434–443)
  - Pulmonary angiogram: Gold standard, though rarely used
- Risk stratify: ↑ HR, ↓ BP, ↓ SpO₂, CTA RV/LV dimension >0.9, ↑ Tn or BNP, echo e/o RV dysfxn, D-dimer >4,000 all predict bad outcomes

Treatment
Supportive: O₂, IV fluids for ↓ BP (preload dep)

Anticoagulation regimen should be selected based on comorbidities, ability to take PO medications, patient preference (monitoring, etc.), risks of bleeding:
- SC LMWH (1 mg/kg BID; renally dose): slight ↓ risk of death, recurrence, major bleeding c/w UFH; preferred w/ malignancy; relative CIs include CKD & obesity
- SC Fondaparinux (5 mg QD [<50kg], 7.5 mg QD [50–100 kg], 10 mg QD [>100 kg]; renally dose): Similar risk of death, recurrence, major bleeding c/w LMWH; preferred w/ hx of HIT
- IV UFH (80 U/kg bolus, 18 U/kg/h gtt): As above, may be preferred over LMWH if CKD/ESRD; risk of HIT higher than LMWH
- PO Warfarin (INR 2.0–3.0): Bridge w/ LMWH/Fondaparinux until INR therapeutic
- PO Rivaroxaban (15 mg BID × 3 wk, 20 mg QD thereafter)
- PO Apixaban (10 mg BID × 7 d, 5 mg BID thereafter)
- IV thrombolysis (tPA: 100 mg over 2 h): Indicated if massive PE / HD instability (SBP <90 mmHg), HD unstable & high suspicion of PE, or submassive PE w/ high risk of hypotension (e/o significant pHTN or RV dysfxn)
- Submassive PE: tPA + UFH ↓ mortality & deterioration c/w UFH alone (NEJM 2002;347:1143–1150)
- Consider lytics in unexplained PEA arrest if possibly 2/2 massive PE
- Catheter or surgical thrombectomy (PE): For pts w/ HD instability & massive PE if (1) CI to lysis, (2) failed lysis w/ tPA, or (3) experienced center & +RV dysfxn. Consult cardiac surgery; improved outcomes c/w UFH alone (Circulation 2014;129:479–486)
- IVC filter: When a/c fails or CI; no long-term mortality benefit (NEJM 1998;338:338:409).

Disposition
- HD stable, few comorbidities, no e/o RV strain: Observation Unit for A/C, LENIs, echo
- HD stable, comorbidities, e/o RV strain: Admit, tele floor
- HD unstable, mx comorbidities, e/o RV strain: Admit, ICU
Overview

- Heart failure: Any chronic state in which the heart’s ability to pump blood with normal efficiency to meet the body’s metabolic demands is impaired; either 2/2 ↓ systolic function (reduced EF: HFrEF) or ↓ diastolic relaxation (preserved EF: HFpEF); can be primarily L-sided, R-sided, or biventricular; although a chronic illness, characterized by intermittent decompensation 2/2 volume disequilibrium

- Manifestations of decompensated L-sided HF: pulmonary edema (↑ PCWP 2/2 hydrostatic forces), pleural effusions (↑ PCWP 2/2 hydrostatic forces), atrial arrhythmias (↑ atrial size), systemic hypoperfusion (HFrEF: ↑ LV end-diastolic volume → ↓ contractility [once over Frank–Starling curve] → ↓ EF; HFpEF: ↓ LV end-diastolic volume 2/2 impaired relaxation → ↓ EF)

- Manifestations of decompensated R-sided HF: pleural effusions (↑ systemic venous pressure → ↑ thoracic duct / lymphatic pressure → ↓ absorption of nl pleural fluid), peripheral edema (↑ systemic venous pressure), liver dysfxn (congestion)

- Acute decompensations can occur 2/2 many causes (see table)

<table>
<thead>
<tr>
<th>Common Precipitants of Acute Decompensated Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medication change or nonadherence*</td>
</tr>
<tr>
<td>Dietary indiscretion (↑ Na)</td>
</tr>
<tr>
<td>Myocardial infarct/ischemia</td>
</tr>
<tr>
<td>Tachyarrhythmia (eg, AF)</td>
</tr>
<tr>
<td>COPD/PE (↑ RH pressures)</td>
</tr>
<tr>
<td>Renal failure (↑ volume)</td>
</tr>
<tr>
<td>Hypertensive crisis (↑ afterload)</td>
</tr>
<tr>
<td>Overdoses (βB, CCB) or toxins (EtOH)</td>
</tr>
<tr>
<td>Myopericarditis, endocarditis</td>
</tr>
<tr>
<td>Sepsis</td>
</tr>
<tr>
<td>Valvular heart dz (see table at end of section)</td>
</tr>
<tr>
<td>Structural heart dz (see table at end of section)</td>
</tr>
</tbody>
</table>

*Especially diuretics, anti-HTN, or rate-controlling agents; however, review any med changes, as may have pharmacokinetic properties that affect cardiac meds

Approach

- Initiate immediately: IV access, O₂ PRN, ECG, Monitor, CXR
- If e/o severe respiratory distress, early CPAP/BiPAP, NTG gtt (unless ↓ BP)
- Early use of bedside U/S (thoracic & echo) can reduce time to dx

History & Physical Exam

- HX: SOB/DOE, CP, cough (clear → pink sputum), orthopnea/PND,
LE/abd swelling
- Always ask: timing (acuity), severity (fxnal capacity), behavioral chgs (sleeping upright), chg in home O₂, frequency of wt monitoring & any change from dry wt
- Always assess for possible precipitants (see table above)
- EX: ↑ BP, ↑ HR, ↑ RR, cardiac dysrhythmia; +S₃ (HFrEF) +S₄ (HFpEF); Rales or ↓ BS, wheeze (L-sided); Leg edema, JVD, ↑ liver size, +hepatojugular reflex (R-sided)

<table>
<thead>
<tr>
<th>Acute Decompensated HF: Value of Specific Hx Components (JAMA 2005;294:1944)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Historical Factor</td>
</tr>
<tr>
<td>H/o heart failure</td>
</tr>
<tr>
<td>H/o MI</td>
</tr>
<tr>
<td>PND</td>
</tr>
<tr>
<td>Orthopnea</td>
</tr>
</tbody>
</table>

**Historical Features w/ Minimal Diagnostic Utility**
H/o CAD, HLD, DM, HTN, COPD, smoking. Sxs of edema, cough, fatigue, & weight gain.

<table>
<thead>
<tr>
<th>Acute Decompensated HF: Value of Specific PEx Components (JAMA 2005;294:1944)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase Likelihood of ADHF</td>
</tr>
<tr>
<td>Exam Factor</td>
</tr>
<tr>
<td>S₃ on auscultation</td>
</tr>
<tr>
<td>JVD</td>
</tr>
<tr>
<td>Rales</td>
</tr>
<tr>
<td>Any murmur</td>
</tr>
<tr>
<td>LE edema</td>
</tr>
</tbody>
</table>

**Exam Features w/ Minimal Diagnostic Utility**
Abdominojugular reflex, SBP <100 mmHg or >150 mmHg, wheezing, ascites

**Evaluation**
- Acute decompensated HF is primarily a clinical dx, aided by lab &
imaging evaluations

▶ ECG: L-atrial enlargement, LVH, tachyarrhythmia, ischemia, e/o old infarction(s)
▶ Labs: CBC, lytes, Cr, troponin, LFTs, VBG, BNP/NT-proBNP (see below)
▶ Bedside thoracic U/S (Sens 94%, Spec 92%): >3 B-lines/field in 2+ fields bilaterally (Acad Emerg Med 2014;21(8):843–852); operator-dependent (some studies w/ Sens as low as 60%), but w/ skilled operator may be superior to CXR in dx of L-sided HF (Chest 2015;148(1):202–210); BSUS also used to eval for other dx:
   - Pleural effusions (2/2 CHF or other dx)
   - Focal B-lines (eg, 2/2 PNA, infarct > asymmetric pulm edema)
   - Reduced EF & pericardial effusion
   - IVC inspiratory collapsibility: <50% collapsibility w/ inspiration suggests volume overload; cannot be used if pt on PPV (Am J Emerg Med 2015;33(5):653–657)
▶ CXR (Sens 70%, Spec 82%): Pulm edema, pl effusion, ↑ heart size (Chest 2015;148(1):202–210)
▶ BNP (>100 ng/L), NT-proBNP (>300 ng/L): Imp to compare w/ dry weight BNP if hx of CHF; levels correlate w/ dz severity (NYHA) of underlying CHF (NEJM 2002;347:161–167)
   - False-negatives: Obesity (Int J Cardiol 2014;176(3):611–617)
   - False-positives: Large PE, cor pulmonale, ESRD, AMI
   - NT-proBNP may also be elevated w/ ↑ age; higher cut-offs suggested (>900 ng/mL if over 50 y)

| Pooled Sensitivity & Specificity of BNP/NT-proBNP for Acute Decompensated HF |
|-------------------------|-------|-------|-------------------------|-------|-------|
| BNP Level              | Sens (%) | Spec (%) | NT-proBNP Level         | Sens (%) | Spec (%) |
| ≤100 ng/L              | 95     | 63     | ≤300 ng/L               | 99     | 43     |
| 100–500 ng/L           | 85     | 86     | 300–1800 pg/ml          | 90     | 76     |
| ≥500 ng/L              | 35     | 78     | ≥1800 ng/L              | 67     | 72     |


**Treatment**

▶ **Diuresis (↓ volume):** Patients w/ refractory edema have impaired PO absorption & may need IV diuresis; give 2× home dose in IV form (see
Conversions below; give home nonloop diuretics (eg, metolazone) for sequential nephron blockade (*NEJM* 2010;362(3):228–238)

- Conversions: Furosemide:Torsemide:Bumetanide 40:10:1; Furosemide (PO:IV) 2:1; Torsemide (PO:IV) 1:1; Bumetanide (PO:IV) 1:1
- If allergy to furosemide/torsemide/bumetanide, can use ethacrynic acid

- **Nitrates (↓ preload):** Nitrates (0.4 mg SL or 10–300 mcg/min IV): Caution in pts w/ AS → ↓ BP 2° preload dep; nitroprusside if NTG ineffective; nesiritide may ↑ Cr/mortality compared to noninotropinic tx (*JAMA* 2005;293:1900)
- **Positive Pressure Ventilation:** CPAP/BiPAP for ↓ SaO₂ (if no CIs); ↓ mortality, ↓ need for intubation (*JAMA* 2005;294:3124; *Lancet* 2006;367:1155); Intubate profound AMS, resp failure
- **Inotropes:** Cardiogenic shock (see section on *Shock*)
- **Other:** Positioning (sit up > supine), Foley may be necessary to assess ins/outs, IABP/LVAD (severe cardiogenic shock)

**Disposition**

- Mild exacerbation, benign etiology (ie, dietary indiscretion), & close f/u: Discharge after discussion w/ cardiologist; may ↑ diuretic for a few days
- Selected HF pts can be managed by a rapid tx protocol in the Observation Unit w/ fewer bed days & similar readmission rates to admitted pts (*Acad Emerg Med* 2013;20(6):554)
- Most pts require admission/Δs to tx regimen before d/c home: Cardiology/Tele
- All pts on PPV or severe resp distress: ICU

<table>
<thead>
<tr>
<th>Structural Causes of Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dilated CMP</strong></td>
</tr>
<tr>
<td><strong>Pathophys:</strong> Ventricular dilatation → ↓ contractility → ↑ EDV → ↓ EF</td>
</tr>
<tr>
<td><strong>Causes:</strong> Idiopathic, familial, ischemia, valvular, infxn (Chagas), EtOH, cocaine, autoimmune</td>
</tr>
<tr>
<td><strong>Presentation:</strong> L or R HF sx; embolic events; arrhythmia</td>
</tr>
<tr>
<td><strong>Evaluation:</strong> ECG (PRWP, Qw, BBB, AF), CXR (↑ heart size), Echo (LV dilatation, ↓ EF, LV ± RV HK)</td>
</tr>
<tr>
<td><strong>Treatment:</strong> See standard HF tx below</td>
</tr>
<tr>
<td><strong>Pearl:</strong> Always consider in chronic EtOH users w/ SOB</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Hypertrophic CMP</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophys:</strong> LV outflow tract obstruction, worse if ↓ EDV → ↓ EF</td>
</tr>
<tr>
<td><strong>Causes:</strong> 50% are familial; asymmetric septal hypertrophy (eg, 2/2</td>
</tr>
</tbody>
</table>
### HOCM

**Presentation:** SOB/angina; arrhythmias; sudden death  
**Evaluation:** Systolic crescendo/decrescendo murmur; ECG (LVH, septal Qw), CXR (↑ heart size), Echo (↑ septal thickness)  
**Treatment:** βB, CCB (verapamil)  
**Pearl:** Avoid diuretics/preload reduction (inc PPV), digoxin, exercise

### Restrictive CMP

**Pathophys:** ↓ compliance → ↓ EDV → ↓ EF  
**Causes:** Amyloidosis, sarcoidosis, hemochromatosis, XRT, cancer  
**Presentation:** R > L HF; embolic events; poor response to diuretics  
**Evaluation:** ↑ JVP, S3, S4, ECG (low voltage), CXR (pulm edema w/o ↑ heart size), Echo (symmetric wall thickening, LAE/RAE)  
**Treatment:** Treat underlying cause, gentle diuresis

### Constrictive pericarditis

**Pathophys:** ↓ compliance → ↓ EDV → ↓ EF  
**Causes:** Postviral, XRT, TB, Postcardiac surgery, idiopathic  
**Presentation:** R > L HF  
**Evaluation:** ↑ JVP, pericardial knock; Echo (septal bounce)  
**Treatment:** Diuresis, pericardiectomy

### Valvular Heart Disease

#### Aortic Stenosis

**Causes:** Calcification (age >70 y), bicuspid valve, rheumatic heart dz  
**Presentation:** Angina, syncope, CHF  
**Exam:** Midsystolic, crescendo–decrescendo @ RUSB  
**Eval:** Echo (transvalvular velocity, EF, AVA)  
**Acute tx:** ↓ Afterload; minimize ↓ preload & negative inotropy; if severe acute HF decompensation 2/2 critical AS, c/s cardiac surg for consideration of urgent AVR  
**Pearl:** Indications for AVR (if sx) include:  
\[ \text{V}_{\text{max}} \geq 4 \text{ m/s; V}_{\text{max}} < 4 \text{ m/s + EF} < 50\% + \text{AVA} \leq 1.0 \text{ cm}^2; \text{ or } V_{\text{max}} < 4 \text{ m/s + AVA} \leq 0.6 \text{ cm}^2 \]  
(NEJM 2014;372:744–756)

#### Aortic Regurg

**Causes:** Rheumatic heart dz, bicuspid valve, endocarditis, HTN  
**Presentation:** Acute or chronic CHF  
**Exam:** Diastolic decrescendo murmur, wide pulse pressure  
**Eval:** Echo: Severity of AI → width of regurgitant jet  
**Acute tx:** ↓ Afterload (nifedipine, ACEi); vasodilators ± dobutamine; if severe & unstable, c/s cardiac surg for consideration of urgent AVR  
**Pearl:** Limited data on mortality benefit of AVR; indications for AVR mostly based on sx severity (NYHA III/IV)  
(NEJM 2004;351:1539–1546)

#### Mitral Stenosis

**Causes:** Rheumatic heart dz  
**Presentation:** Pulmonary edema, AF, Emboli
Exam: Diastolic murmur, opening snap
Eval: ECG (LAE), Echo (valve area, pressure gradients)
Acute tx: Careful diuresis, βBs; however percutaneous balloon valvuloplasty (PBV) & MVR have best outcomes
Pearl: Most have sx if MVA <1 cm²; MVR/PBV based on sx (NYHA III/IV sx [+/- NYHA II]); PBV favorable c/w MVR, but 10–40% may have delayed re-stenosis & require repeat (Lancet 2009;374:1271–1283)

Mitrail Regurg

Causes: MVP, endocarditis, rheumatic heart dz, ruptured chordae, papillary muscle dysfxn
Presentation: Pulmonary edema
Exam: Blowing holosystolic murmur
Eval: ECG (LAE), Echo (width of regurgitant jet)
Acute tx: Vasodilators (ACEi), βB; however, MVR is only intervention w/ proven outcome benefit (Lancet 2009;373:1382–1394)

AORTIC DISSECTION

Overview

• Definition: Any extent of tearing of the aortic tunica intima that enables blood to enter into & traverse the aortic wall b/w tunica intima & tunica media layers
  • Intramural “false lumen” can obstruct nl flow in true aortic lumen, including critical vascular branches (esp carotid, celiac, sup/inf mesenteric, renal, & spinal arteries)
  • Can also manifest w/ penetrating ulcer, pseudoaneurysm, & traumatic rupture
• Classification of dissection impacts management & prognosis (see table)

<table>
<thead>
<tr>
<th>Stanford Type</th>
<th>% of Cases</th>
<th>Anatomical Distribution</th>
<th>Organs at Risk</th>
<th>Prognosis w/ Medical Tx</th>
<th>Prognosis w/ Surgical Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type A</td>
<td>62%</td>
<td>Ascending aorta +/- descending</td>
<td>Brain Coronary Art Spinal cord Abd/kidneys Legs</td>
<td>58%</td>
<td>26%</td>
</tr>
<tr>
<td>Type B</td>
<td>38%</td>
<td>Descending aorta w/o</td>
<td>Spinal cord</td>
<td>10.7%</td>
<td>31%</td>
</tr>
</tbody>
</table>

Prognosis for Aortic Dissection (IRAD, JAMA 2000;283:897)
### Approach

- Immediate IV, ECG, pCXR, Analgesia, BP control (if HTN)
- Consult cardiothoracic surgery early, esp for type A dissection if clinically suspected
- Attention to extent of dissection, size of T/F lumens, involvement of branches, presence of periaortic/mediastinal hematoma or pleural effusion

### Risk Factors for Aortic Dissection *(Circulation 2010;121:e266)*

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Associated Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>‡ Aortic wall stress</td>
<td>Hypertension, cocaine/stimulant use, extreme valsala (eg, power lifting), blunt trauma/deceleration injury, aortic coarctation, pheochromocytoma</td>
</tr>
<tr>
<td>Vulnerability of Ao wall</td>
<td>Genetic disorders (Ehlers–Danlos, Marfan, Turner, Loeys–Dietz, Noonan syndromes, congenital bicuspid valve, familial dissection), inflammatory vasculitides (SLE, GCA, Behçet's), infectious vasculitides (syphilis, TB)</td>
</tr>
<tr>
<td>Iatrogenic wall injury</td>
<td>Cardiac/valve surgery, IABP use, aortic cannulation, cath</td>
</tr>
<tr>
<td>Other</td>
<td>Male, &gt;50 y/o, pregnancy, PCKD, chronic steroids, immunosupp</td>
</tr>
</tbody>
</table>

### History & Physical Exam

- Individual elements of hx in isolation notoriously insensitive &/or nonspecific (see table)
- HX: Abrupt onset & often worst-ever CP (ascending), interscapular back pain (descending), or neck pain; often maximal at onset, ripping/tearing in quality, & can migrate; can be a/w syncope, neurologic deficits
  - Note that up to 10% of pts may not p/w pain
  - As for RFs & consider in all blunt trauma pts w/ CP or back pain
- EX: Check for murmur, B/L UE BP asymmetry >20 mmHg († Sens, but ominous finding), pulse deficit (27% of pts), neurologic deficits including Horner syndrome, abd pain +/- guaiac exam (+ result can suggest bowel ischemia), flank pain

### Aortic Dissection: Frequency of History, Exam, & CXR Findings

<table>
<thead>
<tr>
<th>Component</th>
<th>Overall (%)</th>
<th>Type A (%)</th>
<th>Type B (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
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</tbody>
</table>
Aortic Dissection: Sensitivity of Components of History

<table>
<thead>
<tr>
<th>Component</th>
<th>Sens (95% CI)</th>
<th>Sens (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hx of HTN</td>
<td>64% (54–72)</td>
<td>Hx of Marfan syndrome</td>
</tr>
<tr>
<td>Any pain</td>
<td>90% (85–94)</td>
<td>Back pain</td>
</tr>
<tr>
<td>Chest pain</td>
<td>67% (56–77)</td>
<td>Abdominal pain</td>
</tr>
<tr>
<td>Anterior chest pain</td>
<td>57% (48–66)</td>
<td>Syncope</td>
</tr>
<tr>
<td>Posterior chest pain</td>
<td>32% (24–40)</td>
<td></td>
</tr>
<tr>
<td>Severe pain</td>
<td>90% (88–92)</td>
<td>Ripping/Tearing pain</td>
</tr>
<tr>
<td>Sudden-onset pain</td>
<td>84% (80–89)</td>
<td>Migrating pain</td>
</tr>
</tbody>
</table>

**Evaluation**

- ECG: Assess for inf-STEMI (Type A dissection can involve RCA; ~4–8% of thoracic dissections will present w/ signs of STEMI), LVH (e/o chronic HTN)
- In pts w/ inferior STEMI, consider Type A dissection always
- Labs: Type & Cross, CBC, Lytes, Cr (↑ w/ renal ischemia), Troponin,
Lactate (↑ w/ any ischemia, ↑↑ suggests abd viscera ischemia), PT/PTT

- CXR: may be nl in 20%; characteristic findings include wide mediastinum, abnl aortic knob, L apical cap, trachea shift → R, depressed L bronchus, L pl effusion
- Combined use of D-dimer & Aortic Detection (ADD) Risk Score: Early data support combined use of ADD & D-dimer; ADD <1 & neg D-dimer can r/o AoD (Sens 100%, NPV 100%); ADD 1 & neg D-dimer also very high Sens (98.7%) & NPV (99.2%), & likely improved further if CXR nl (60% of pts w/ AoD have wide mediastinum) (see table)
  - D-dimer not appropriate in pts w/ ADD 2–3, given ↓ Sens & ↓↓ Spec
- Bedside cardiac US: Limited data suggest high diagnostic utility (Sens 88%, Spec 94%), esp in conjunction w/ ADD 0 (Sens 96%, Spec 98%); positive study includes any of following findings: intimal flap, intramural hematoma, ascending Ao dilatation, AV insuff, pericardial effusion; operator-dependent (Intern Emerg Med 2014;9(6):665–670)
  - May be most useful in low-risk pts w/ chronically elevated D-dimer (eg, cancer, age)
- Definitive diagnostic modalities: TEE (Sens 98%, Spec 95%), CTA (Sens 100%, Spec 98%), MRI (Sens 98%, Spec 98%) (Arch Intern Med 2006;166:1350–1356)

### Aortic Dissection Detection (ADD) Risk Score

<table>
<thead>
<tr>
<th>High Risk Conditions</th>
<th>High Risk Pain Features</th>
<th>High Risk Exam Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marfan syndrome</td>
<td>Chest, back, or abd pain described as:</td>
<td>Evidence of perfusion deficit (pulse deficit, SBP differential)</td>
</tr>
<tr>
<td>FHx aortic dz</td>
<td>Abrupt in onset</td>
<td>Focal neuro deficit in conjunction w/ pain</td>
</tr>
<tr>
<td>Known AoV dz</td>
<td>Severe in intensity</td>
<td>Hypotension/shock</td>
</tr>
<tr>
<td>Recent aortic manipulation</td>
<td>Ripping or tearing</td>
<td></td>
</tr>
<tr>
<td>Known thoracic AA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Test Characteristics of ADD & Combined Approach Using ADD + D-Dimer

<table>
<thead>
<tr>
<th>No. ADD High Risk Categories Present</th>
<th>ADD Alone*</th>
<th>ADD Combined w/ D-Dimer**</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sens (%)</td>
<td>Sens (%)</td>
</tr>
<tr>
<td>0 (Low risk)</td>
<td>95.7*</td>
<td>100</td>
</tr>
<tr>
<td>1 (Int risk)</td>
<td>63.5</td>
<td>98.7</td>
</tr>
<tr>
<td>2–3 (High risk)</td>
<td>40.8</td>
<td>97.5</td>
</tr>
</tbody>
</table>

*Note that half (48.6%) of low-risk pts w/ AoD in derivation data had widened mediastinum on CXR. Circulation 2011;123:2213–18.
**Int J Cardiol 2014;175:78–82.**

### Diagnostic Characteristics of Advanced Imaging for AD (Arch Intern Med 2006;166:1350)

<table>
<thead>
<tr>
<th>Imaging Study</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>+LR</th>
<th>−LR</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEE</td>
<td>98% (95–99%)</td>
<td>95% (92–97%)</td>
<td>14.1 (6–33)</td>
<td>0.04 (0.02–0.08)</td>
</tr>
<tr>
<td>CTA</td>
<td>100% (96–100%)</td>
<td>98% (87–99%)</td>
<td>14 (4.2–46)</td>
<td>0.02 (0.01–0.11)</td>
</tr>
<tr>
<td>MRI</td>
<td>98% (95–99%)</td>
<td>98% (95–100)</td>
<td>24 (11–57)</td>
<td>0.05 (0.03–0.10)</td>
</tr>
</tbody>
</table>

### Treatment (Lancet 2008;372:55–66)
- In general, surgical tx preferred for Type A, medical tx for Type B
- Tx revolves around close BP & HR control; Goal HR 60–80, SBP 100–120
  - First-line: IV BB gtt preferred to bolus (esmolol, labetalol)
  - Second-line (CI to BB, need for further control): IV CCB gtt (eg, nicardipine, diltiazem)
  - If refractory HTN/tachy: Vasodilator (nitroprusside)
  - A-line for close monitoring (pref RUE or farthest from false lumen)
- Analgesia: Short-acting narcotics preferred in case of hemodynamic changes
- Urgent surgical consultation should be obtained (cardiac surgery for Type A, vascular surgery for Type B) for all pts diagnosed w/ thoracic aortic dissection regardless of the location as soon as the Dx is made or suspected
  - Type A: Evaluate for emergent surgical repair (1–2% mortality/h in 1st 24 h)
  - Type B: Manage medically w/ consideration for endovascular repair (esp if e/o malperfusion, enlarging aneurysm, inability to control BP/sx)

### Disposition
- All patients w/ acute aortic dissection are admitted to ICU (+/− via OR)

---

**THORACIC AORTIC ANEURYSM**

### Overview
- NL aortic diameter ↑ w/ age, sex (M > F), body surface area, imaging
modality

- **Thoracic Aortic Aneurysm:** Permanent localized of aortic wall dilatation involving all 3 layers (tunica intima, tunica media, tunica externa) & reaching $1.5 \times \text{nl aortic diameter}$; dilatation b/w $>1$ & $<1.5 \times \text{nl dilatation}$ referred to as ectatic.

- **Thoracic Aortic Pseudoaneurysm:** See TAA, but involves $<3$ aortic wall layers

  - Can occur at the aortic root (annular aortic ectasia) &/or ascending aorta (50%), descending aorta (40%), aortic arch (10%), or thoracoabdominal aorta (10%)
  - Up to $\sim25\%$ of pts w/ TAA may also have an AAA
  - Most TAAs are caused by degenerative dz resulting in dilation of the aorta
  - RFs: See section on *Aortic Dissection* (see above)

- Complications vary based on diameter; average rate of expansion $0.10–0.42 \text{ cm/y}$

<table>
<thead>
<tr>
<th>Aortic Size</th>
<th>$&gt;3.5 \text{ cm (%)}$</th>
<th>$&gt;4 \text{ cm (%)}$</th>
<th>$&gt;5 \text{ cm (%)}$</th>
<th>$&gt;6 \text{ cm (%)}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rupture</td>
<td>0</td>
<td>0.3</td>
<td>1.7</td>
<td>3.6</td>
</tr>
<tr>
<td>Dissection</td>
<td>2.2</td>
<td>1.5</td>
<td>2.5</td>
<td>3.7</td>
</tr>
<tr>
<td>Death</td>
<td>5.9</td>
<td>4.6</td>
<td>4.8</td>
<td>10.8</td>
</tr>
<tr>
<td>Any of above</td>
<td>7.2</td>
<td>5.3</td>
<td>6.5</td>
<td>14.1</td>
</tr>
</tbody>
</table>

### History, Physical Exam, & Evaluation

- **HX:** Often discovered incidentally on imaging; sx can vary widely:
  - Compressive sx: Hoarseness (compression of recurrent laryngeal nerve), stridor (compression of trachea/bronchi), dyspnea (lung compression), dysphagia (esophageal compression), plethora/edema (SVC compression)
  - Heart failure sx: May occur 2/2 aortic regurgitation
  - Embolization of atherosclerotic debris w/ end-organ sx may occur
  - May lead to dissection (see *Aortic Dissection* section) or rupture

- **EX:** May have nl exam; see Aortic Dissection section exam above

- **Imaging:** CTA (Good Sens, quick, noninvasive); MRI (best for Ao Root); TTE (limited for eval of Ao Root or descending TA); TEE (Better than TTE for Ao Root & descending TA)
Treatment

- Risk factor modification: Lipid profile optimization, smoking cessation, BP control (BB, ACEi), avoid intense exercise or valsalva
- Urgent open vs. endovascular repair as indicated (see table)

### Indications for Urgent Cardiac Surgical Consultation *(Circulation 2010;121:e266)*

- Asymptomatic pts w/ degenerative TAAs, chronic aortic dissection, intramural hematoma, penetrating atherosclerotic ulcer, mycotic aneurysm, or pseudoaneurysm for whom the ascending aorta or aortic sinus diameter is ≥5.5 cm
- Pts w/ Marfan syndrome or other genetically mediated disorders (see above) for whom the ascending aorta or aortic sinus diameter is 4–5 cm
- Pts who have a growth rate of more than 0.5 cm/y in an aorta that is <5.5 cm
- Pts w/ sxs suggestive of expansion of TAA

Disposition

- Admit: Patients meeting indications for urgent repair, symptomatic patients
- Discharge (w/ vascular/cardiac surgery f/u): Pts w/ large but asx (ie, incidental) TAA
- Discharge (w/ PCP f/u for surgery referral): Pts w/ small & asx (ie, incidental) TAA
- All discharged pts: DC w/ RF modification (eg, improved BP control) & serial monitoring

### ACUTE PERICARDITIS

**Definition** *(NEJM 2014;371(25):2410–2416)*

- Acute inflammatory dz of the pericardium due to a variety of causes:
  - Idiopathic (80% in developed nations)—Presumed post-viral
  - Infectious (TB, fungal, less likely staph/strep)
  - Post-MI (Dressler’s)
  - Systemic dz (cancer, connective tissue d/o, myxedema, uremia)
  - Trauma or treatment (postsurgical, XRT, posttraumatic)
- Dx requires the absence of more likely cause of CP (eg, ACS, etc.) & ≥2 of the following:
  1. Characteristic CP (see below)
  2. Pericardial friction rub (high pitched, scratch sound heard best at left sternal border)
3. Suggestive ECG findings (see below)
4. New or worsening pericardial effusion
   - Can be relapsing in 10–30%: Incessant (d/c of tx or attempts to wean cause relapse in <6 wk) or intermittent (symptom-free intervals >6 wk, but recurs)
   - Can be a/w pericardial effusion w/ or w/o tamponade, or can be constrictive

**History & Physical Exam**
- HX: Characteristic CP—Sudden onset, retrosternal, pleuritic, positional (better w/ leaning forward or upright); pain can radiate to neck, arms, shoulders similar to ACS
  - Ask about recent viral illness
  - May have low-grade fever, SOB, dysphagia
- EX: Friction rub (high pitched, scratch sound heard best at LLSB apex), ↑ HR, ↑ RR, nl BP

**Evaluation**
- ECG: findings occur in 4 stages (see table), generally characterized by diffuse STE & PR depressions, though subtle PR depressions may be only sign
  - Assess for electrical alternans (suggests pericardial tamponade, see next section)

<table>
<thead>
<tr>
<th>Stages of ECG Changes in Pericarditis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage 1</strong></td>
</tr>
<tr>
<td><strong>Stage 2</strong></td>
</tr>
<tr>
<td><strong>Stage 3</strong></td>
</tr>
<tr>
<td><strong>Stage 4</strong></td>
</tr>
</tbody>
</table>

- Labs: CBC, BUN/Cr (r/o uremia), LFTs, ESR/CRP (↑ CRP in 75%), cardiac enzymes (as much as 1/3 cases a/w myocarditis) (*NEJM* 2014;371(25):2410–2416)
  - Further testing unnecessary unless WBC >13 k, T >38.5 F, or comorbidities or hx suggests specific underlying cause; PRN TSH, serologies (infxn, inflam)
- CXR: r/o other dx; can see cardiomegaly if >250 cc pericardial effusion
- Bedside echo: Assess for (1) pericardial effusion, (2) tamponade
physiology (late diastolic collapse of RA, persistence of RA collapse >1/3 cardiac cycle, early diastolic collapse of RV, collapse of LA, dilated IVC w/ <50% respiratory collapse)

- Although not routinely indicated, CT & MRI can help make dx (pericardial thickening)

**Treatment**

- Pharmacologic tx is mainstay:
  - NSAIDs: Ibuprofen (600–800 mg q6h–q8h), Indomethacin (25–50 mg q8h), aspirin (2–4 g qd in divided doses) x 1–2 wk; Give w/ PPI for gastric protection
  - ASA preferred among NSAIDs in early post-MI period
  - Colchicine (0.5 mg QD if ≤70 kg; 0.5 mg BID if >70 kg): Used in conjunction w/ NSAIDs; c/w placebo, ↓ risk of recurrence & persistent sx at 72 h by 50% (NEJM 2013;369:1522–1528)
  - Use cautiously w/ CKD, hepatobiliary dz, bleeding dyscrasias, GI motility d/o
  - In conjunction w/ NSAIDs, usually improves sx w/i 1–3 d
  - Steroids (prednisone 1 mg/kg/d w/ slow taper after 2–4 wk): First-line for autoimmune or uremic etiologies, or those who fail NSAID or colchicine therapy; may ↑ risk of recurrence (COPE, Circulation 2005;112:2012).
  - Optimal duration of tx unclear: 3 mo course recommended (NEJM 2013;369:1522–1528)
  - Tx underlying condition PRN (abx, dialysis, chemo, etc.)
  - Pericardiocentesis indicated for purulent (postsurgical, TB, etc.) or neoplastic pericarditis
  - Cardiology consult: If tamponade/echo is being considered
  - CT surgery consult: Recent cardiac surgery or if pericardial window needed

**Disposition**

- 85% of pts can be discharged home
- Admit anyone w/ HD abnlty, myocarditis, uremia, large effusion
Overview

- Definition: A life-threatening state in which intrapericardial pressure (2/2 fluid, blood, pus) > RVEDP → ↓ LV preload → ↓ LVEDP → equilibration of L & R heart pressures → ↓ CO
- Tamponade more related to rate of fluid accumulation than volume of fluid
- Can be caused by blood (Type A AoD, post-MI free wall rupture, postsurgical, trauma), pus (TB, postsurgical), or fluid (myxedema, uremia, malignancy, SLE, XRT)

History & Physical Exam

- HX: If atraumatic, can p/w progressive SOB/DOE, orthopnea, PND, CP, LH, AMS, weakness; Traumatic usually w/ gross penetrating wound or blunt aortic injury
- EX: ↑ HR, ↑ RR, Beck’s triad (↓ BP, distended neck veins, muffled heart sounds), narrow pulse pressure, pulsus paradoxus (see below)

### Performing Pulsus Paradoxus Test: Assessing the Reversed Bernheim Effect

- Using a sphygmomanometer, inflate the cuff to 20 mmHg above systolic pressure, then deflate until the 1st Korotkoff sound is heard, which you should only hear during expiration. Record this number. Next, deflate the cuff until Korotkoff sounds are heard equally during both inspiration & expiration. Subtract this number from the 1st.
- If the difference b/w these 2 numbers is >10 mmHg, the pt has a pulsus paradoxus of a magnitude equal to that difference
- DDx: Cardiac tamponade, severe asthma/COPD, PE, constrictive pericarditis

Evaluation

- ECG: Low voltage, electrical alternans, ± signs of pericarditis
- CXR: Globular heart, but may be nl if rapid accumulation (eg, trauma)
- Bedside Echo: Can confirm dx; effusion (can be variable size) w/ septal shift, late diastolic collapse of RA, persistence of RA collapse >1/3 cardiac cycle, early diastolic collapse of RV, collapse of LA, dilated IVC w/ <50% respiratory collapse
- Pericardial fluid: If atraumatic, consider sending fluid culture & Gram stain, BUN, Cr, ANA, RF, malignancy screen/cytology

Treatment

- IVF Bolus: Preload dependent state; ↑ preload to RV causes ↑ RVEDP > intrapericardial pressure → ↑ LV preload → ↑ CO
- Preload is purely temporizing to pericardiocentesis; ultimately, w/
excess preload pts will develop pulm edema & hypoxia; any need for PPV must be avoided at all costs given profound effect on ↓ preload
• Pericardiocentesis: cardiac tamponade w/ HD compromise requires urgent drainage (bedside if unstable; preferred in OR if time)

Disposition
• Admit all patients w/ cardiac tamponade. If drained effectively & stable, can be admx to tele floor (ie, cardiology). If admitted while awaiting drainage, ICU.

MYOCARDITIS

Overview
• Definition: Acute lymphocytic inflammatory dz of the myocardium of varying severity ranging from subclinical dz to fulminant systolic failure & death
• Frequently a/w viral infections (coxsackie, enterovirus, adenovirus), Chagas dz, toxins/meds (cocaine, lithium, doxorubicin), SLE, scleroderma

History & Physical Exam
• HX: Dyspnea (72%), CP (32%), arrhythmias (18%); May have systemic sxns including fever, arthralgia, malaise; Can present similar to HFrEF
• EX: Ranges from subtle signs of systolic dysfxn (crackles, LE edema) to fulminant respiratory failure (JVD, tachypnea, dec BS, LE edema), arrhythmia, or cardiac arrest

Evaluation
• ECG: Sinus tach, STE/STD/NSSTW Δs, VT/VF, heart block, Δs of pericarditis (see above)
• CXR: ↑ cardiac size
• Labs: Cardiac enzymes (Troponin > CKMB; 34% Sens, 89% Spec; ↑ Sens w/ ↑ extent of dz), BNP, CBC w/diff (can see eosinophilia), ↑ ESR/CRP (NEJM 2009;360:1526–1538)
• Cardiac MRI: Useful for establishing definitive dx &/or planning bx

Treatment
• Largely supportive; treat CHF, cardiogenic shock, or arrhythmias
Overview

- Definition (syncope): Loss of consciousness & postural tone arising from an abrupt drop in cerebral perfusion w/ spontaneous recovery.
- Definition (pre-syncope, near-syncope): As above, but sx resolve before complete LOC or loss of tone; may experience AMS & weakness before return to nl
- Objective in ED is to distinguish from other causes of sudden LOC, & differentiate benign etiologies from those requiring further eval or tx (see table)

### Common or Concerning Causes of Syncope

<table>
<thead>
<tr>
<th>Primary Cardiac Etiologies</th>
<th>Mechanism</th>
<th>HX</th>
<th>DX</th>
<th>TX</th>
<th>DISPO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachydysrhythmia</td>
<td>↑ HR (eg, VT, AF, AT, SVT, WPW), ↓ LVEDV, ↓ CO;</td>
<td>May be unheralded or prodrome of LH, CP, palp, diaphoresis, nausea, SOB;</td>
<td>ECG, Tele, o/p cardiac monitor;</td>
<td>rhythm-specific;</td>
<td>Admx</td>
</tr>
<tr>
<td>Bradydysrhythmia</td>
<td>↓ HR (eg, SSS, BB, CCB, Heart block esp 3°), ↓ CO;</td>
<td>May be unheralded or prodrome of LH, CP, weakness, diaphoresis, nausea, SOB;</td>
<td>ECG, Tele, o/p cardiac monitor;</td>
<td>rhythm-specific;</td>
<td>Admx, may need PPM</td>
</tr>
<tr>
<td>Valvular Heart dz (usually AoS)</td>
<td>↓ Preload w/ fixed severe AS, ↓ CO;</td>
<td>May be unheralded, often a/w position (standing), dehydration, dysrhythmia (↓ CO), can have chronic DOE/known AoS;</td>
<td>Murmur, echo;</td>
<td>Optimize preload, AVR;</td>
<td>Admx (see Valvular Ht Dz table)</td>
</tr>
<tr>
<td>HFrEF (eg, post-MI)</td>
<td>↓ EF (esp if ↑ neg inotropic med);</td>
<td>Weakness, DOE, PND/orthopnea, recent MI or hx HF, med Δs;</td>
<td>Echo;</td>
<td>HF optimization (↓ afterload, ↓ preload if not hypovolemic, +/- ↑ inotropy);</td>
<td>Admx for med optimization. (see CHF section)</td>
</tr>
<tr>
<td>HOCM</td>
<td>↑ HR, ↓ EDV, ↓ SV 2/2 outflow obstruction (can also ↑ risk of VT/VF);</td>
<td>Often a/w exercise (↑ HR), missed meds (↑ HR), or dehydration (↓ preload);</td>
<td>Echo;</td>
<td>BB, CCB, ↑ Preload;</td>
<td>Admx, may need AICD (see Cardiomyopathy table)</td>
</tr>
<tr>
<td>Tamponade</td>
<td>↑ intrapericardial pressures &gt; RV filling pressure, ↓ L-sided filling pressures, ↓ CO;</td>
<td>Progressive weakness, SOB, DOE, orthopnea, PND, +/- CP;</td>
<td>Echo;</td>
<td>↑ preload,</td>
<td></td>
</tr>
</tbody>
</table>

- Syncope: Loss of consciousness & postural tone arising from an abrupt drop in cerebral perfusion w/ spontaneous recovery.
- Definition (pre-syncope, near-syncope): As above, but sx resolve before complete LOC or loss of tone; may experience AMS & weakness before return to nl.
- Objective in ED is to distinguish from other causes of sudden LOC, & differentiate benign etiologies from those requiring further eval or tx (see table).
### Primary Vascular Etiologies

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mechanism</th>
<th>HX</th>
<th>DX</th>
<th>TX</th>
<th>DISPO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulm Embolism</td>
<td>↑ PA obstruction, ↓ L-sided preload, ↓ CO; <strong>HX:</strong> May be unheralded, or sudden SOB, CP, sense of doom; <strong>DX:</strong> Risk stratify, then: D-dimer or CTA or V/Q; <strong>TX:</strong> Lysis vs. anticoagulation; <strong>DISPO:</strong> Admx (if cause of syncope) (see PE section)</td>
<td></td>
<td></td>
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<tr>
<td>Pulm HTN</td>
<td>↓ LV preload 2/2 any ↑ PVR; <strong>HX:</strong> Often a/w exertion, PMH of IPH, CTD, MS/MR, COPD; <strong>DX:</strong> ECG (RAE, RBBB, RVH), CXR (enlarged pulm vasc, RA, RV), BNP, echo (↑ RSVP, PR/TR), cardiology c/s ± right-heart cath; <strong>TX:</strong> O2 (↑ hypoxic vasoconstriction), diuresis, ↑ inotropy (digoxin, dobutamine), +/- inh NO if decomp, prostacyclins, PDE5 inh, discuss w/ cardiology; <strong>DISPO:</strong> Admx</td>
<td></td>
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<tr>
<td>AoD (Type A&gt;B)</td>
<td>False lumen ↓ carotid inflow, OR tamponade present; <strong>HX:</strong> Sudden CP, back pain; <strong>DX:</strong> Echo, CTA; <strong>TX:</strong> Emergent cardiac surgery (Type A); <strong>DISPO:</strong> Admx (see Aortic Dissection section)</td>
<td></td>
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<tr>
<td>TAA/AAA</td>
<td>Sudden expansion, contained leak, or rupture of AA; <strong>HX:</strong> Sudden but not always severe CP, back pain, flank pain, abd pain; <strong>DX:</strong> Abd U/S (AAA), CTA; <strong>TX:</strong> Optimize BP/HR, Emergent vasc surgery c/s; <strong>DISPO:</strong> Vascular c/s, Admx</td>
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</tr>
<tr>
<td>Subclavian (SCA) steal syndrome</td>
<td>Sudden ↓ SBP or ↑ SCA (eg, UE movement) overlying chronic prox SCA stenosis → retrograde vert artery flow ipsilaterally, ↓ postcirculation perfusion; <strong>HX:</strong> Can be a/w movements of affected UE, dehydration, med Δs, sometimes also w/ vertigo; <strong>DX:</strong> B/L SBP Δ &gt;45 mmHg, Asymmetric pulses, CXR (1st rib), Duplex U/S, CTA, MRA; <strong>TX:</strong> Open or endovascular surgery; <strong>DISPO:</strong> Vascular c/s, Admx</td>
<td></td>
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<tr>
<td>Carotid stenosis</td>
<td>↓ SBP (any cause) w/ chronic o/w asx carotid stenosis can ↓ cerebrovascular perfusion (if impaired autoreg), ↓ CPP &amp; syncope; <strong>HX:</strong> May be unheralded, often a/w position (standing), dehydration, dysrhythmia (↓ CO); <strong>DX:</strong> Duplex U/S; <strong>TX:</strong> Optimize BP, HR, +/- o/p CEA; <strong>DISPO:</strong> Admx</td>
<td></td>
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<tr>
<td>Vertebrobasilar insufficiency</td>
<td>↓ SBP (any cause) w/ chronic VB stenosis (eg, CAD) can ↓ cerebrovascular perfusion (if impaired autoreg), ↓ CPP &amp; syncope; <strong>HX:</strong> May be unheralded, often a/w position (standing), dehydration, dysrhythmia (↓ CO), a/w dizziness/vertigo, dysarthria, ataxia, vision chg; <strong>DX:</strong> CTA, MRA, Neuro c/s; <strong>TX:</strong> Med mgmt of atherosclerosis, rarely surgery; <strong>ADMX:</strong> Admx</td>
<td></td>
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</tbody>
</table>

### Non-Cardiovascular Etiologies

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mechanism</th>
<th>HX</th>
<th>DX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasovagal</td>
<td>↑ vagal tone a/w emotional or physiologic stressor; <strong>HX:</strong> Common emotional precipitants inc sight of blood, sudden</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
emotional shock; physiologic stressors inc fatigue, long standing, warmth, n/v, coughing, swallowing, micturition, defecation; **DX**: Clinical dx, ↓ HR (sinus brady) & BP during event; **TX**: None needed; **DISPO**: Home

<table>
<thead>
<tr>
<th>Carotid sinus hypersensitivity</th>
<th><strong>Mechanism</strong>: ↑ vagal tone after mechanical pressure on carotid sinus; <strong>HX</strong>: often after shaving, head turning; <strong>TX</strong>: None indicated; <strong>DX</strong>: Clinical; <strong>DISPO</strong>: Home</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Orthostatic hypotension</th>
<th><strong>Mechanism</strong>: ↓ vascular compliance → ↓ SBP w/ position chgs; <strong>HX</strong>: Often elderly (stiff vessels), can be a/w GI bleed, ectopic preg; <strong>DX</strong>: CBC, imaging if c/f underlying condition, orthostatic VS ↓ Sens (sx w/ standing may be more helpful &amp; Sens than VS); <strong>TX</strong>: IVF, +/- blood if e/o ongoing losses; <strong>DISPO</strong>: Varies depending on if underlying condition identified; if none found &amp; pt stable gait, can dc home w/ FU</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Autonomic dysfxn</th>
<th><strong>Mechanism</strong>: Impaired fxn of autonomic nervous system; <strong>HX</strong>: May be a/w dysfxn of other autonomic fxns (GI, bladder, sweating), may have hx of DM, EtOH, HIV, SLE, Neuro dz; check med Δs; often hx of similar episodes in the past; <strong>DX</strong>: Tilt table testing, c/s neurology <strong>TX</strong>: Tx underlying condition, salt tabs, +/- midodrine (discuss w/ cardiology &amp; neurology); <strong>DISPO</strong>: Admx; can dc home w/ close o/p f/u if low-risk pt &amp; low-frequency events</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Medications</th>
<th>Common medications (new or ↑ dose) a/w syncope: vasodilators (α-blockers, nitrates, ACEI/ARB, CCB, hydralazine, phenothiazines, antidepressants), diuretics, negative chronotropes (BB, CCB), antiarrhythmics (class IA, IC, III), psychoactive meds (antipsych, TCAs, barbs, benzos), substances (EtOH)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Syncope mimics</th>
<th>Seizure,* TIA/stroke,* ICH,* migraine*</th>
</tr>
</thead>
</table>

*Can mimic syncope, but not considered true syncopal events. (modified from: NEJM 2002;347:878; JACC 2006;47:473)

### History & Physical Exam

- **HPI**: Always ask about preceding activity (inc. posture), precipitants, prodromal sx (weakness, LH, diaphoresis, visual chgs), duration (<5 s suggests cardiac; >5 s suggests vasovagal), assoc sx (CP, palp, focal neuro deficits, HA, abd pain, nausea)
- Differentiate from seizure: C/w seizure, syncope typically more abrupt, shorter duration, quicker return to nl (seconds–minutes), no tongue biting or incontinence, lack of rigidity; note syncope commonly can occur w/ slow & irregular myoclonic jerking mistaken as seizure/convulsive activity.
- **ROS**, **PMH** (cardiac dz), **meds**, & **FHx** (sudden cardiac death) are very
**Important**
- High-risk hx: Older age, structural heart dz, h/o CAD
- Lower-risk hx: Young, healthy, nonexertional, no hx or e/o cardiac dz, no FHx SCD
- EX: Guided by hx; evaluate neuro exam (inc stability w/ standing/gait), murmurs, carotid bruits, abd exam +/- guaiac

**Evaluation**
- ECG in all pts: Evaluate for stigmata of malignant dysrhythmia (HOCM, ARVD, Brugada syndrome, prolonged QTc, pre-excitation syndrome, coronary artery abnormalities)

<table>
<thead>
<tr>
<th>Cardiac Dz</th>
<th>ECG Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brugada syndrome <em>(Circulation 2005;111:659)</em></td>
<td>- Type I: Coved ST-segment elevation ≥2 mm followed by a negative T wave in &gt;1 R precordial lead (V1–V3)</td>
</tr>
<tr>
<td></td>
<td>- Type II: STE w/ saddle-back appearance w/ a high takeoff STE ≥2 mm, a trough displaying STE ≥1 mm, &amp; then either a + or biphasic T wave</td>
</tr>
<tr>
<td></td>
<td>- Type III: Either saddleback or coved appearance w/ STE &lt;1 mm</td>
</tr>
<tr>
<td></td>
<td>- Other: Prolonged QT, P wave, PR interval, QRS</td>
</tr>
<tr>
<td>HOCM <em>(Am J Emerg Med 2007;25:72)</em></td>
<td>- Characteristic findings of LVH (see ECG section)</td>
</tr>
<tr>
<td></td>
<td>- Deep narrow Q waves in inferior (II, II, aVF) &amp; lateral (I aVL, V5, V6) leads in pts w/ septal hypertrophy</td>
</tr>
<tr>
<td></td>
<td>- Deep inverted T waves in mid &amp; lateral precordial leads in pts w/ isolated apical hypertrophy</td>
</tr>
<tr>
<td>Arrhythmogenic R ventricular dysplasia <em>(Am J Med 2004;117:685)</em></td>
<td>- Epsilon waves (small amplitude deflections at transition of QRS &amp; ST segment) in R precordial leads</td>
</tr>
<tr>
<td></td>
<td>- Prolonged QRS complex to &gt;110 ms in V1–V3 w/o RBBB</td>
</tr>
<tr>
<td></td>
<td>- Inverted T waves n V1–V3 in absence of RBBB</td>
</tr>
<tr>
<td></td>
<td>- Reduced R-wave amplitude</td>
</tr>
<tr>
<td>Long QT syndrome <em>(Circulation 1995;92:2929; Circulation 2000;102:2849)</em></td>
<td>- Prolongation of QT interval, usually &gt;500 ms</td>
</tr>
<tr>
<td></td>
<td>- LQT1 has a broad T wave, LQT2 has small &amp;/or notched T wave, LQT3 has unusually long onset T wave</td>
</tr>
<tr>
<td>Pre-excitation syndrome *(WPW) <em>(Am Heart J 1930;6:685)</em></td>
<td>- Short PR interval</td>
</tr>
<tr>
<td></td>
<td>- Slurred upstroke of QRS complex (delta wave)</td>
</tr>
<tr>
<td></td>
<td>- Increased QRS duration</td>
</tr>
</tbody>
</table>

- Labs & imaging: All guided by history/exam & specific dx’s being considered; consider CBC, electrolytes (+/- Hcg) in most pts; however,
obvious vasovagal syncope in a young o/w healthy male may not require any labs at all
- Consider cardiac markers, UA, stool guaiac, head CT in elderly
- Any pt w/ ICD who has syncope should have their ICD interrogated by an appropriate specialist given the high likelihood of malignant dysrhythmia in such pts, which was likely the initial indication for ICD placement prophylactically.

Disposition *(Ann Emerg Med 1997;29:4)*
- Home if low-risk cardiac features: (1) Age <45, (2) nl ECG, (3) nl exam. Consider outpatient f/u.
- Admit if high-risk cardiac features: (1) Age (unknown age threshold, but continuous variable), (2) h/o cardiac dz (esp e/o heart failure or structural heart dz), (3) one or more Criteria of San Francisco Syncope rule
- Other if diagnosed or suspected life-threatening diseases (eg, MI, aortic dissection, GI bleed), acute neurologic abnlty (eg, stroke, sz), ± for congenital heart dz, FHx sudden death, exertional syncope in pt w/o obvious cause

Decision Rules in Evaluation of Syncope
- Note at present no single clinical decision rule should outweigh clinical judgment

<table>
<thead>
<tr>
<th>San Francisco Syncope Rule</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Features (“CHESS”)</strong></td>
</tr>
<tr>
<td>CHF (past or present)</td>
</tr>
<tr>
<td>Hct &lt;30%</td>
</tr>
<tr>
<td>ECG abnl (new change or nonsinus)</td>
</tr>
</tbody>
</table>

Using SFSR to Guide Disposition Decisions
- If any of above features present, admit patient.
- Predicts risk of serious outcome (mortality, MI, arrhythmia, PE, CVA, SAH, significant hemorrhage, return to ED) w/i 7 d; Sens 86% (CI 83–89%), spec 49% (CI 48–41%) *(Ann Emerg Med 2010;56(4):362)*
- If none of above features present, consider d/c. Note that at publication of this book, pooled studies on SFSR have revealed a considerable population of pts w/ serious outcomes that do not have any of the five SFSR clinical features; however, all of these patients were admitted for other reasons. Combination of clinical gestalt & SFSR may have higher Sens & NPV than SFSR alone.
**OESIL Score (Osservatorio Epidemiologico sulla Sincope nel Lazio)**

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;65 y</td>
<td>1</td>
</tr>
<tr>
<td>Syncope w/o prodrome</td>
<td>1</td>
</tr>
<tr>
<td>H/o Cardiovascular dz: Clinical or lab dx of any form of structural heart dz (ischemic, valvular, 1° myocardial dz, CHF, PAD, TIA/CVA)</td>
<td>1</td>
</tr>
<tr>
<td>Abnl ECG: Abnl rhythm (AF/AFL, SVT, MAT, frequent or repetitive PATs/PVCs, sustained or nonsustained VT, paced rhythms), AV or interventricular conduction d/o (CHB, Mobitz I or II AVB, BBB, IVCD), LVH, RVH, left-axis deviation, definitive or possible e/o prior MI</td>
<td>1</td>
</tr>
</tbody>
</table>

**Prognostication Based on OESIL Score**

<table>
<thead>
<tr>
<th>Points</th>
<th>All-Cause Mortality w/i 12 mo of ED Visit (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>0.8</td>
</tr>
<tr>
<td>2</td>
<td>19.6</td>
</tr>
<tr>
<td>3</td>
<td>34.7</td>
</tr>
<tr>
<td>4</td>
<td>57.1</td>
</tr>
</tbody>
</table>

**Notes:**
- Sens 95% (CI 88–98%), spec 31% (CI 29–34%) (Ann Emerg Med 2010;56(4):362)
- Best at long-term outcomes, poor w/ short term
- Not rigorously externally validated compared to other syncope scores; derived & validated in Italian community ED settings.

---

**Boston Syncope Criteria for Predicting Adverse Event or Critical Interventions**

<table>
<thead>
<tr>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic symptoms</td>
</tr>
<tr>
<td>Signs of conduction dz</td>
</tr>
<tr>
<td>H/o underlying cardiac dz</td>
</tr>
<tr>
<td>Family Hx (1st degree)</td>
</tr>
<tr>
<td>Persistent (&gt;15 min) abnl</td>
</tr>
</tbody>
</table>
VS w/o need for intervention

Volume depletion | GIB by hx or hemocult, Hct <30%, Dehydration not corrected in ED by treating EP discretion

1° CNS event | (eg, SAH, CVA/TIA)

- Predicts critical intervention (PM/ICD placement, PCI, surgery, blood transfusion, CPR, alteration in antidysrhythmic therapy, endoscopy w/ intervention, or correction of carotid artery stenosis) or an adverse outcome (death, PE, CVA, severe infection/sepsis, ventricular/atrial dysrhythmia, ICH, hemorrhage, AMI, cardiac arrest, or other life-threatening sequelae) w/i 30 d
- Authors recommend admission for any + finding
- Diagnostic utility of any + finding: Sens 97% (CI 93–100%), spec 62% (CI 56–69%)
- Has not been externally validated


**HYPERTENSION AND HYPERTENSIVE EMERGENCIES**

**Approach**
- Must differentiate chronic elevations in BP from an acute elevation
- Must differentiate transient elevations (ie, from anxiety or pain) from other causes
- Search for life-threatening causes of elevations in BP, including e/o end-organ damage (see HTN emergency)

<table>
<thead>
<tr>
<th>Differentiation for Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>Cardiovascular</td>
</tr>
<tr>
<td>Renal</td>
</tr>
<tr>
<td>Endocrine</td>
</tr>
</tbody>
</table>

**Definition** *(JAMA 2003;289:2560)*
-HTN: SBP ≥140 or DBP ≥90
-HTN urgency: SBP ≥180 or DBP ≥110 w/ no acute organ damage; this term is also referred to as “hypertensive crisis” & has largely fallen out of favor
-HTN emergency: Elevated BP w/ acute organ damage (cardiac, CNS, renal)

**History**
- H/o CAD, CHF, TIA, stroke, peripheral a. dz, renal insufficiency, meds (sympathomimetics, cocaine, amphetamines), med noncompliance

**Evaluation**
- Check BP in both arms, check cuff/cuff size
- In ED pts w/ asymptomatic markedly elevated BP, routine screening for acute target organ injury (ie, serum Cr, US, ECG) is not required
- In select pt populations (ie, those w/ poor f/u), screening for an elevated Cr level may identify kidney injury that affects disposition

**Treatment**
- Goal BP <140/90 mmHg; if DM or renal dz goal is <130/80 mmHg
- Tx HTN results in 50% ↓ CHF, 40% ↓ stroke, 20–25% ↓ MI *(Lancet 2000;356:1955)*
- In pts w/ asymptomatic markedly elevated BP (ie, ≥180/≥110), routine ED medical intervention is not required
- In selected pt populations (ie, those w/ poor f/u), EPs may treat markedly elevated BP in the ED &/or initiate therapy for long-term control
  - For initiation of long-term therapy, it may be reasonable to start a thiazide-type diuretic for most pts, but may consider ACEI, ARB, BB, CCB, or combination *(Hypertension 2003:42:1206)*
  - In this situation, consider HCTZ 12.5–50 mg QD or Chlorthalidone 12.5–25 mg QD. Chlorthalidone may be superior to HCTZ *(MRFIT, Circulation 1990;82(5):1616; SHEP, JAMA 1991;265;265(24):3255; ALLHAT, JAMA 2002;288(23):2981)*

<table>
<thead>
<tr>
<th>Antihypertensive Medications for Specific Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dz</strong></td>
</tr>
<tr>
<td>Cardiac ischemia</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
### Hypertensive Emergency

#### Approach
- Look for e/o acute end-organ damage
- Neurologic: Encephalopathy, hemorrhagic or ischemic stroke, papilledema
- Cardiac: ACS, CHF, aortic dissection
- Renal: ARF
- Other: Preeclampsia–eclampsia

#### History
- Look for precipitants: Progression of essential HTN, medication noncompliance, rebound HTN (clonidine), worsening renal dz, pheochromocytoma, Cushing drug use (cocaine, amphetamines, MAOIs + tyramine), cerebral injury
- CP, dyspnea, HA, blurry vision, confusion, oliguria, hematuria

#### Findings

<table>
<thead>
<tr>
<th>CHF</th>
<th>NTG</th>
<th>10–200 mcg/min IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICH, HTN encephalopathy</td>
<td>Nitroprusside, Labetalol</td>
<td>0.3–10 mcg/kg/min IV, up to 300 mg</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>Esmolol + nitroprusside, or labetalol alone</td>
<td>Esmolol: Bolus 0.25–0.5 mg/kg over 1–2 min, then 10–200 mcg/kg/min gtt; see above for nitroprusside &amp; labetalol</td>
</tr>
<tr>
<td>Renal artery stenosis</td>
<td>ACEI or ARBs</td>
<td>Captopril 25 mg PO BID, Losartan 50 mg PO QD</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Phenoxybenzamine, Phentolamine</td>
<td>10 mg PO BID, 5 mg IV during HTN crisis</td>
</tr>
<tr>
<td>Preeclampsia–eclampsia</td>
<td>Magnesium, Hydralazine</td>
<td>1–4 g IV over 2–4 min, 10 mg IV</td>
</tr>
</tbody>
</table>

### Disposition
- Asymptomatic pts may be d/c home w/ PCP f/u

### Pearls
- HTN in the ED is often a/w anxiety/pain. Always re√ BP once pt is calm & pain free
- Tx of pts w/ asymptomatic HTN in the ED is not necessary if outpatient f/u is available
- In neonates, suspect renovascular dz, coarctation of the aorta, or kidney malformation
Assess MS, e/o papilledema, visual acuity

**Evaluation**
- BUN/Cr, lytes, CBC, UA, ECG (e/o LVH), CXR, cardiac enzymes (if ischemia suspected), head CT (if ICH suspected)

**Treatment**
- ↓ MAP by 25% w/i 1–2 h using IV meds, then f/u w/ PO version
- Avoid tx HTN during acute stroke unless pt is getting lysed, has extreme HTN (>220/110), aortic dissection, active ischemia, or CHF (Stroke 2003;34:1056)
- Treat by underlying cause as noted above

**Disposition**
- True hypertensive emergencies require ICU admission for BP monitoring


---

**HYPOTENSION AND SHOCK**

**Approach**
- ABCs: Always address airway/breathing prior to circulation
- Differentiate hypotension from shock

**Definition**
- Hypotension: BP below pt’s baseline, often defined as SBP <90 mmHg
- Shock: Insufficient perfusion pressures for organs’ metabolic needs

<table>
<thead>
<tr>
<th>Differential for Hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Shock</td>
</tr>
<tr>
<td></td>
</tr>
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<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
Peripheral vasodilation (ie, distributive) \hspace{1cm} Septic shock, anaphylactic shock, neurogenic shock

| Hypotension* | Adrenal insufficiency, medications (eg, nitrates, narcotics, antihypertensives), orthostatic hypotension, neurocardiogenic syncope, pregnancy, hypoglycemia, pseudohypotension (ie, inaccurate measurement, faulty BP cuff) |

*Some causes of hypotension can lead to shock.

**History**
- AMS, CP, SOB

**Findings**
- ↓ BP, ↑ HR, hypoxia, ↑ RR, UOP <1 mL/kg/h

**Evaluation**
- CBC, Chem 7, PT/PTT, cardiac markers, LFTs, blood gas, lactate, T/S, stool guaiac, ECG e/o ischemia
- POC ultrasonography: RUSH (Rapid Ultrasound in Shock, Emerg Med Clin N Am 2010;28:29) protocol incorporates a 3-part bedside physiologic assessment simplified as:
  - **the pump** (POC cardiac US to assess for pericardial effusion, global LV contractility, relative size of LV to RV)
  - **the tank** (POC IVC US to assess respiratory dynamics of IVC & volume status, as well as lung, pl & abdominal US to assess for pathology that could alter vascular volume; ie, PTX, pl effusion, free intra-abdominal fluid)
  - **the pipes** (POC thoracic & abdominal aortic US to assess for AD/AAA & LE compression US to assess for DVT)


<table>
<thead>
<tr>
<th>RUSH Evaluation</th>
<th>Hypovolemic Shock</th>
<th>Cardiogenic Shock</th>
<th>Obstructive Shock</th>
<th>Distributive Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pump</td>
<td>Hypercontractile heart Small chambers</td>
<td>Hypocontractile heart Dilated heart</td>
<td>Hypercontractile heart Pericardial eff Cardiac tampon RV strain Cardiac thromb</td>
<td>Hypercontractile heart (early) Hypocontractile heart (late)</td>
</tr>
</tbody>
</table>
### Treatment

- **Priority should be to obtain adequate IV access.** If peripheral large-bore IVs cannot be placed in timely manner, consider IO (humeral/tibial/sternal) or stat central venous access w/ large internal diameter catheters (ie, cordis).
- **Priority should be to restore hemodynamics before time-consuming diagnostic w/u:**
  - 1–2 L of isotonic crystalloid infusion as rapid as possible (ie, on pressure bag if indicated)
  - Consider stat uncrossmatched blood in life-threatening hemorrhage; consider using rapid infuser device; consider permissive hypotension in hemorrhagic shock
  - Consider peripheral vasoactive agents if persistently hypotensive after IVF bolus as bridge to obtaining central venous access

### Vasoactive Agents and Dosing *(Emerg Med Clin N Am 2008;26:759)*

<table>
<thead>
<tr>
<th>Vasoactive Agent</th>
<th>Primary Receptor Activity</th>
<th>Relative Effects</th>
<th>Typical IV Dosing</th>
<th>Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenylephrine</td>
<td>α1 +++</td>
<td>↑ SVR ↓ HR</td>
<td>20–200 mcg/min</td>
<td>Reflex bradycardia</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>α1 ++++ α2 +++ β1 +++ β2 0(+)</td>
<td>↑ HR ↑ SV ↑ SVR</td>
<td>1–40 mcg/min</td>
<td>Tachydysrhythmia</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>α1 ++++ α2 +++(+) 1 +++ β2 0(+)</td>
<td>↑↑↑ HR ↑↑↑ SV ↑↑↑ SVR Brchdilate</td>
<td>1–20 mcg/min</td>
<td>Tachydysrhythmia Splanchnic ischemia Acute MI</td>
</tr>
<tr>
<td>Dopamine</td>
<td>α2+, β1+, β2+, D++ α1/2+, β1++, β2+, D++</td>
<td>Natriuresis ↑↑ HR ↑↑ SV ↑↑ SVR</td>
<td>Dose dependent: 1–5 mcg/kg/min 5–10 mcg/kg/m 10–20 mcg/kg/m</td>
<td>Tachydysrhythmia</td>
</tr>
<tr>
<td>α1 (++), α2+, β1++, β2+, D++</td>
<td>Vasopressin</td>
<td>V1 receptor</td>
<td>↑ SVR ↓ HR</td>
<td>0.01–0.03 U/min</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
<td>------------------</td>
</tr>
<tr>
<td>α1 0(+) α2 0(+) β1 +++ β2 +++</td>
<td>Dobutamine</td>
<td>↑↑ HR ↑↑ SV ↓ SVR</td>
<td>2–20 mcg/kg/min</td>
<td>Tachydysrhythmia HypoTN Acute MI</td>
</tr>
<tr>
<td>PDE inhibition</td>
<td>Milrinone</td>
<td>↑ HR ↑↑ SV ↓ SVR</td>
<td>0.25–0.75 mcg/kg/min</td>
<td>Tachydysrhythmia HypoTN Acute MI</td>
</tr>
</tbody>
</table>

- Use MS, UOP, & MAP as early e/o adequate end-organ perfusion

**Pearls**
- Not all hypotension is clinically significant. Use clinical context, pt’s baseline BP, & check the BP cuff.
- Pulses provide a marker of baseline SBP, but may overestimate the absolute value (*BMJ* 2000;321:673)

<table>
<thead>
<tr>
<th>Pulse Present</th>
<th>Minimum SBP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radial artery</td>
<td>80</td>
</tr>
<tr>
<td>Femoral artery</td>
<td>70</td>
</tr>
<tr>
<td>Carotid</td>
<td>60</td>
</tr>
</tbody>
</table>

**HYPOVOLEMIC SHOCK**

**Approach**
- Dehydration is a Dx of exclusion; consider other etiologies (hemorrhage, ectopic pregnancy, etc.)

**Definition**
- Intravascular volume depletion → ↓ perfusion, most commonly 2° blood loss
**Differential for Hypovolemic Shock**

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhage</td>
<td>Trauma (internal, external), GI bleed, ruptured AAA</td>
</tr>
<tr>
<td>Other</td>
<td>Dehydration, ectopic pregnancy, placenta previa, placental abruption</td>
</tr>
</tbody>
</table>

**History**
- Trauma, melena, hematochezia, hematemesis, ↓ PO intake

**Findings**
- E/o trauma, guaiac + stool, pelvic exam

**Evaluation**
- As above +UA/HCG, FAST (blood in abdomen or chest); consider CT chest/abd/pelvis, pelvic US, type/screen

**Treatment**
- **Identify/treat cause**, IV fluid bolus; consider PRBCs; **consult** immediately for life-threatening disorders requiring definitive tx (surgery, GI, OB/Gyn)

**Disposition**
- Admit vs. OR

---

**CARDIOGENIC SHOCK**

**Approach**
- Consider intubation early, look for & **treat underlying cause**

**Definition**
- ↓ CO + nl intravascular volume → ↓ systolic contractility + ↑ diastolic filling

<table>
<thead>
<tr>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACS, myocarditis, dysrhythmia, valvular failure, severe CMP, cardiac contusion, pulmonary HTN</td>
</tr>
</tbody>
</table>

**Findings**
- ↑ HR, ↓ BP, ↑ RR, hypoxia, pulmonary rales, S3, S4
Evaluation
- CBC, Chem 7, Ca, Mg, PO₄, ECG, CXR, stat echo (systolic/diastolic dysfxn, papillary muscle rupture, ventricular wall rupture, VSD, pericardial effusion, R heart strain)

Treatment
- Treat underlying dz, IV fluids (if ↓ intravascular volume)
- Dopamine: ↑ myocardial contractility & BP, but ↑ O₂ demand
- Dobutamine: ↑ HR & inotropy, less O₂ demand, but causes vasodilation (best if not tachycardic or severely hypotensive)
- Central venous catheter: Consider for CVP monitoring, administration of pressors
- Cardiology consult
- Other: Thrombolytics, IABP, ventricular assist device

Disposition
- Admit to ICU

SEPTIC SHOCK

Approach (NEJM 2006;355:1699)
- Identify & treat early → best outcomes when treated w/i 6 h
- Look for source of infection

Definition
- Sepsis = SIRS (severe inflammatory response syndrome) + source infection
- SIRS: ≥2 of the following: Temp ≥38°C or ≤36°C, HR ≥ 90, RR ≥ 20, WBC (≥12,000, ≤4,000, or >10% bands)
- Severe sepsis: Sepsis + sepsis-induced hypoperfusion (hypotension persisting after initial fluid challenge or blood lactate >4 mmol/L) or organ dysfxn (see organ dysfxn variables below)
- Septic shock: Severe sepsis + ↓ BP despite adequate fluid resuscitation

Common Causes of Sepsis
<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>PNA, empyema</td>
</tr>
<tr>
<td>Abdominal</td>
<td>Peritonitis, abscess, cholangitis</td>
</tr>
<tr>
<td>Skin</td>
<td>Cellulitis, fasciitis</td>
</tr>
<tr>
<td>Renal</td>
<td>Pyelonephritis</td>
</tr>
<tr>
<td>CNS</td>
<td>Meningitis, brain abscess</td>
</tr>
</tbody>
</table>

### Evaluation

- CBC w/ diff, Chem 10, LFTs, lactate, blood (×2)/urine/sputum culture, PT/PTT, cardiac markers, VBG, CXR; consider CT brain/LP, CT chest &/or abdomen, RUQ US based on pt
- Consider 1,3 beta-D-glucan assay & galactomannan assay if available & invasive candidiasis is in the DDx as cause of infection

### Diagnostic Criteria for Sepsis

#### Inflammatory variables:
- Leukocytosis (WBC >12,000 μL⁻¹)
- Leukopenia (WBC <4,000 μL⁻¹)
- nl WBC w/ >10% immature cells (band forms)
- Plasma CRP >2 SD above nl
- Plasma procalcitonin >2 SD above nl

#### Organ dysfxn variables:
- Arterial hypoxemia (PaO₂/FiO₂ <300)
- Acute oliguria (UOP <0.5 mL/kg/h for at least 2 h despite fluid resuscitation)
- Cr increase >0.5 mg/dL
- Coagulation abx (INR >1.5 or aPTT >60 s)
- Ileus
- TTP (plt count <100,000 μL⁻¹)
- Hyperbilirubinemia (plasma Tbili >4 mg/dL)

#### Tissue perfusion variables:
- Hyperlactemia (>1 mmol/L)
- Decreased cap refill/mottling

### Treatment

- **EGDT** *(NEJM 2001;345:1368)*; ↓ mortality/hospital stay in 1 study, though no prospective validation study
- Protocolized quantitative resuscitation of pts w/ sepsis-induced hypoperfusion. **Goals during the 1st 6 h:**
  - **CVP 8–12 mmHg** → crystalloid (NS or LR) is the initial fluid of choice;
    *initial fluid challenge* 30 cc/kg; consider albumin when pts require
substantial IVFs
- Central venous access should be obtained as soon as practical
- **MAP ≥65 mmHg** → use of vasopressors, whereby:
  - Norepinephrine is 1st choice
  - Epinephrine (added to or substituting NE) when additional agent needed
  - Vasopressin 0.03 U/min can be added to NE to raise MAP or decrease NE dose
  - Dopamine as alternative to NE only in highly selected pts (low-risk arrhythmia)
  - Phenylephrine not recommended except special circumstances
  - Arterial catheter should be placed as soon as practical
- **UOP ≥0.5 mL/kg/h**
  - Foley catheter should be placed as soon as practical for I/O monitoring
- **ScvO$_2$ or mixed venous O$_2$ saturation 70% or 65%, respectively**
  - Trial of dobutamine up to 20 mcg/kg/min should be given or added to vasoactive agent in the presence of myocardial dysfxn (elevated filling pressure/low CO) or ongoing signs of hypoperfusion, despite CVP & MAP goals (ScvO$_2$ <70%)
  - Abx: Broad spectrum, given prior to drawing cultures (cover gram+, gram−, anaerobes; consider double coverage for pseudomonas)
  - Start abx w/i 1 h of recognition, regardless of whether source is known
  - Source control
  - **Hydrocortisone:** Consider hydrocortisone use in pts w/ severe sepsis refractory to IV fluids & pressors; corticotropin test + routine steroid use → no benefit & possibly harm (*NEJM* 2008;358:111)
  - **Blood products:** PRBCs to target Hgb 7–9 g/dL; Plts if <10,000 μL$^{-1}$ w/o bleed, <20,000 μL$^{-1}$ w/ risk of bleeding, <50,000 μL$^{-1}$ for active bleeding, surgery, procedure
  - **Oxygenation/ventilation:** Supplemental O$_2$; consider need for intubation early; if intubated use VTs of 6 cc/kg predicted BW (*NEJM* 2000;342:1301) use of sedation/paralytics → ↓ O$_2$ consumption
  - **Glucose control:** q1–2h measurements; initiate protocolized blood glucose management when 2 consecutive measurements >180 mg/dL to a target <180 mg/dL
  - **Renal replacement therapy:** Use continuous therapies (ie, CVVH) to
facilitate managing fluid balance in HD unstable pts

- **Activated protein C**: Use is controversial; ↓ mortality in severe sepsis based on 1 phase 3 trial *(NEJM 2001;344:699; Crit Care Med 2003;31:12)*, but ↑ bleeding, ↑ cost, & no benefit in less sick (APACHE II <25) populations *(NEJM 2005;353:1332)*; recently taken off market

### Disposition

- Admit


---

**NEUROGENIC SHOCK**

### Approach

- Cervical spine injury → risk of apnea, may require intubation; evaluate according to ATLS

### Definition

- Transection of the spinal cord → disruption of sympathetic pathways → loss of vascular sympathetic tone → vasodilation (typically cervical or high thoracic lesions)

### History

- Trauma w/ severe injury to the spinal cord

### Findings

- ↓ HR, ↓ BP, anesthesia, paralysis below an spec dermatome; saddle anesthesia, ↓ rectal tone, areflexia, Horner syndrome, absent bulbocavernosus reflex, priapism (unopposed PNS stimulation)

### Evaluation

- CT spine (esp cervical, thoracic); consider CT head, chest, abd/pelvis if h/o trauma

### Treatment

- C-spine immobilization: Aspen or Philadelphia collar for prolonged immobilization
- Strict log-roll precautions
- IV fluids: Prior to starting pressors
- Vasopressors: Dopamine, norepinephrine, phenylephrine
- Consult neurosurgery immediately

**Disposition**
- Admit

**Pearl**
- Any trauma pt w/ hypotension should be suspected of having hemorrhagic shock until proven o/w, thus neurogenic shock should be treated if suspected but should not be the 1° DDx in the hypotensive trauma pt.

## DYSRHYTHMIA

### Approach
- Follow ACLS protocols for anyone unstable or symptomatic (CP, SOB, AMS, abnl VS)

<table>
<thead>
<tr>
<th>Differential</th>
<th>Type</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradycardia</td>
<td></td>
<td>Sinus bradycardia, SA node block/escape rhythm, sick node dysfxn, AV blocks (2nd-&amp; 3rd-degree AV block)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tachycardia</th>
<th>Regular</th>
<th>Narrow-complex Sinus tachycardia, SVT (AVNRT, AVRT), AT, AFL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wide-complex</td>
<td>Ventricular tachycardia, SVT w/ aberrancy, SVT w/ pre-excitation (eg, WPW), tachycardia w/ PM</td>
</tr>
<tr>
<td></td>
<td>Irregular</td>
<td>Narrow-complex AF, AFL w/ variable AV block, multifocal atrial tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wide-complex AF w/ aberrancy, polymorphic VT</td>
</tr>
</tbody>
</table>

## BRADYCARDIA

### Approach
Follow ACLS protocols for anyone unstable or severely symptomatic (CP, SOB, AMS)
Anticipate need for external/transvenous pacing & cardiology consult early
Always obtain ECG & rhythm strip
Medication hx is crucial
In children, be highly suspicious of toxic ingestion
In neonates, consider congenital cardiac dz

Definition
HR <60 in an adult, <80 in a child <15 y/o, <100 in an infant <1 y/o.
Caused by depressed function of the SA node or conduction system block/delay.

Sinus Bradycardia (NEJM 2000;342:703)

History
Fatigue, syncope/presyncope, DOE, medication hx (esp βBs)

Differential
Physiologic (athletic young adults), medications (nodal agents), hypothyroidism, ↑ vagal tone (including inferior MI), hypothermia, ↑ ICP

Evaluation
ECG (HR <60 in adults, nl PR intervals, P wave preceding each QRS), rhythm strip

Treatment
Asymptomatic bradycardia does not require tx. Tx only if symptomatic or life-threatening cause is suspected w/ atropine &/or pacing.

Disposition
Admit anyone who is symptomatic

SA Node Block/Escape Rhythm

History
Same as for sinus bradycardia

Differential
Same as for sinus bradycardia. Also a/w ↑ K, ↑ vagal tone.

Evaluation
ECG (absent atrial depolarization & missing P waves), rhythm strip, lytes, consider TSH, cardiac markers
Treatment
- Asymptomatic bradycardia does not require tx. Tx only if symptomatic or life-threatening cause is suspected.

Disposition
- Admit anyone who is symptomatic

Sinus Node Dysfunction (Sick Sinus Syndrome/Tachy–Brady Syndrome)

Definition
- Sinus node dysfxn includes a series of ECG abnormalities characterized by failure to generate appropriate cardiac potentials from the sinus node
- In sick sinus syndrome, there are frequent long sinus pauses that may degenerate to absent atrial depolarization for a period of time before the resumption of regular cardiac conduction (sinus arrest)
- In tachy–brady syndrome, episodes of sinus bradycardia or sinus arrest are interspersed w/ episodes of supraventricular tachycardia (often AF)

History
- Syncope, presyncope, fatigue, weakness, DOE, palpitations
- Typically observed in 70–80 y/o, suggesting age-related degeneration

Differential
- Consider other life-threatening arrhythmias

Evaluation
- ECG (frequent sinus pauses, bradycardia/tachycardia rhythms); consider electrolytes, cardiac markers, CBC; Holter or event monitoring

Treatment
- Acute tx only for symptomatic or life-threatening arrhythmia; ultimately may require combination of rate control for tachycardia & PPM for bradycardia

Disposition
- Admit anyone who is symptomatic for permanent PM placement
- If minimal or no sxs are present, d/c home w/ close f/u
Definition

- These occur when conduction from the atria to the AV node & into the His bundle is disrupted
- These blocks can anatomically be located above, w/i, or below the His bundle
- Classified as 1st-degree, 2nd-degree Mobitz I (Wenckebach), 2nd-degree Mobitz II, & 3rd-degree blocks based on characteristic ECG patterns:

<table>
<thead>
<tr>
<th>Classification</th>
<th>ECG Findings</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>1° AV block</td>
<td>• Prolonged PR interval &gt;0.2 s, nl QRS</td>
<td>• vagal tone, MI, age-related degeneration, drugs (BB, CCB, digoxin), infection, endocarditis</td>
</tr>
</tbody>
</table>
| 2° AV block Mobitz type I | • Progressive ↑ PR interval w/ RR interval shortening until QRS dropped  
|                          | • Appears as grouped beats                                                   | • vagal tone, inferior MI, age-related degeneration, drugs (BB, CCB, digoxin), infection, endocarditis |
| 2° AV block Mobitz type II | • Stable PR & RR interval w/ occasional dropped QRS  
|                            | • Can be regular (2:1) or irregular                                           | • Age-related degeneration, anteroseptal MI                                  |
| 3° AV block             | • Complete AV dissociation                                                  | MI (IMI w/ AV node ischemia or anteroseptal MI w/ H-P ischemia), age-related degeneration, drugs (BB, CCB, digoxin), infection, endocarditis, myocarditis, RF, congenital |

Approach

- Differentiate 1st, 2nd Mobitz I (Wenckebach), 2nd Mobitz II, & 3rd-degree blocks
- 2° Mobitz II & 3° blocks are never nl → look for underlying cardiac dz
- In children, be highly suspicious of toxic ingestion
- In neonates, consider congenital cardiac dz
• Determine (1) rate, (2) wide or narrow QRS, (3) rhythm regular or irregular, (4) P waves present or absent, (5) every P wave followed by QRS & every QRS preceded by P

History
• 1°: Asymptomatic, incidental finding on ECG
• 2° Mobitz I (Wenckebach): Often asymptomatic; irregular heartbeat, fatigue
• 2° Mobitz II: May be asymptomatic; presyncope/syncope, fatigue, DOE
• 3°: Usually symptomatic; presyncope/syncope, fatigue, weakness, DOE

Findings
• See above

Evaluation
• ECG & rhythm strip
• 2° Mobitz II & 3°: Labs in anticipation of PPM placement

Treatment
• 1° & 2° Mobitz I: No tx generally necessary
• 2° Mobitz II & 3°:
  • Continuous tele monitoring
  • Symptomatic pts require transcutaneous &/or transvenous pacing; if HD unstable, consider a beta-adrenergic agent (dopamine, epinephrine, or isoproterenol) as bridge to pacing. Dopamine has been demonstrated to have equivalent survival outcomes & adverse events to transcutaneous pacing (*PrePACE*, *Resuscitation* 2008;76(3):341)
  • Treat active cardiac ischemia
  • Consult cardiology

Disposition
• Pts w/ 1° & 2° Mobitz I: D/c home w/ f/u
• Pts w/ 2° Mobitz II & 3°: Admit all to Tele ward for cardiology consult & PPM

Pearls
• Avoid atropine for reversal of AV block as this can worsen conduction
• Have transcutaneous pacer attached & ready for use in high-risk pts
• Mobitz II is concerning b/c risk of progression to 3°
TACHYCARDIA/PALPITATIONS

Approach
- Follow ACLS protocols for anyone unstable or severely symptomatic (CP, SOB, AMS)
- Anticipate need for intubation & defibrillator early
- Always obtain ECG & rhythm strip
- Determine (1) rate, (2) wide or narrow QRS, (3) rhythm regular or irregular

<table>
<thead>
<tr>
<th>Causes of Wide Complex Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tx</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
</tr>
<tr>
<td>SVT w/ *aberrancy (ie, BBB)</td>
</tr>
<tr>
<td>SVT w/ pre-excitation</td>
</tr>
<tr>
<td>Tachycardia + PM</td>
</tr>
</tbody>
</table>

*Causes of aberrancy: Bundle branch blocks (fixed, rate-related, Ashman's phenomenon), accessory pathways (ie, WPW), meds (ie, class Ia/Ic antiarrhythmics, TCAs), pseudo-STEMI, PM, hyperkalemia, hypothermia, cardiomyopathies, channelopathies.

Supraventricular Tachycardia

Approach
- Differentiate type based on ECG, rhythm strip, & response to adenosine/vagal maneuvers (see below)

Definition
- Rhythm arises above the ventricles (either atrium or AV jxn) w/ narrow QRS unless pre-excitation or aberrant conduction

History
- H/o pulmonary or cardiac dz → AT, MAT, AFL, AF, NPJT; o/w health adult → AVNRT, AVRT
- Gradual onset → ST, AT; abrupt onset → AVNRT, AVRT

Evaluation
- Consider CBC, TSH, tox screen, though in most cases, ECG/rhythm strip is sufficient
### SVT Pathophysologies

<table>
<thead>
<tr>
<th>Type of SVT</th>
<th>Pathophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial</td>
<td></td>
</tr>
<tr>
<td>ST</td>
<td>Pain, fever, anxiety, hypovolemia, PE, medication, anemia, hyperthyroid</td>
</tr>
<tr>
<td>AT</td>
<td>Originates in atria but not SA node; a/w COPD, CAD, EtOH, digoxin</td>
</tr>
<tr>
<td>MAT</td>
<td>Originates in atria at ≥3 separate sites</td>
</tr>
<tr>
<td>AFL</td>
<td>Atrial macroreentry, typically R atrium</td>
</tr>
<tr>
<td>AF</td>
<td>Multiple irregular atrial impulses typically from pulmonary veins</td>
</tr>
<tr>
<td>AV jxn</td>
<td></td>
</tr>
<tr>
<td>AVNRT</td>
<td>Re-entrant pathway w/i AVN</td>
</tr>
<tr>
<td>AVRT</td>
<td>Re-entrant pathway using AVN + accessory pathway b/w atria &amp; ventricles</td>
</tr>
<tr>
<td>NPJT</td>
<td>Originates at AV jxn, a/w myo/endocarditis, cardiac surgery, IMI, digoxin</td>
</tr>
</tbody>
</table>


### Diagnosis by ECG, Vagal Maneuvers, and Adenosine (*NEJM* 2006;354:1039)

| Rate         | ST: Typically <150 bpm  
|--------------|-------------------------|
|              | AFL: Typically 150 bpm (2:1 AV block)  
|              | AVNRT/AVRT: Typically >150 bpm |
| Rhythm       | Irregular → AF, MAT |
| P wave       | UPRIGHT before QRS: ST, AT, MAT  
|              | Retrograde AFTER QRS: AVNRT (w/ QRS), AVRT (after QRS)  
|              | FIBRILLATION or no P wave → AF  
|              | SAWTOOTH appearance → AFL |
| Vagal/adenosine | Slows rate w/ ↑ AV block: ST, AT, MAT |
| Response     | Terminates rhythm or no response: AVNRT, AVRT  
|              | “Unmasks” sawtooth waves ↑ AV block → AFL |

### Treatment
- Cardiovert any unstable rhythm
- ST: Treat underlying condition
- AT/MAT: Treat underlying condition; consider AV nodal blocker
- AF/AFL: CCB, βB, dig, antiarrhythmic (amiodarone, lidocaine)
- AVNRT/AVRT: Vagal maneuvers, adenosine, CCB preferable to βB → avoid adenosine/nodal agents if e/o pre-excitation (see WPW below)
NPJT: CCB, βB, amiodarone

**Disposition**
- Most pts w/ ST, AVNRT, AVRT can be d/c home once rhythm is controlled if → asymptomatic & no acute underlying condition. Admission for other rhythms is variable, but often necessary due to underlying condition.
- Consult cardiology for any pt w/ unstable SVT & those difficult to control w/ standard tx

**Pearl**
- MAT is often misdiagnosed as AF. Look closely at P wave morphology.

**Atrial Fibrillation and Atrial Flutter**

**Definition** *(JACC 2006;48:e149)*
- AF is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activity w/ consequent deterioration of mechanical function
- Can be 1st episode or recurrent (≥2 episodes) as well as paroxysmal (self-limited), persistent (>7 d), permanent (>1 y) &/or cardioversion has failed
- Valvular → rheumatic heart dz, or postvalve surgery
- Lone AF → <60 y/o & no e/o cardiac dz or HTN

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other</td>
<td>Idiopathic (50%)</td>
</tr>
<tr>
<td>Cardiac</td>
<td>CHF, peri/myo/endocarditis, MI/ischemia, s/p cardiac surgery, HTN</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>COPD, PNA, PE</td>
</tr>
<tr>
<td>Endo</td>
<td>Hyperthyroid, stress, infection, postop</td>
</tr>
<tr>
<td>Drugs</td>
<td>EtOH &quot;holiday heart syndrome&quot;, cocaine, amphetamines, sympathomimetics, caffeine</td>
</tr>
</tbody>
</table>

**History**
- Abrupt vs. gradual onset (palpitations, DOE, fatigue presyncope/syncope, CP); recent illness, drug & alcohol use

**Findings**
- Irregularly irregular pulse; may be regular w/ AFL
Evaluation

- ECG, CBC, lytes, Ca, Mg, PO4; CXR
- Consider cardiac markers (if active CAD is suspected); TSH, dig level if appropriate; echo (LA size, thrombus, valves, LV fxn)
- Consider outpatient Holter in pts w/ suggestive hx who arrive in NSR
- ECG in AF: Replacement of consistent P waves by rapid oscillating or fibrillatory waves that vary in amplitude, shape, & timing, a/w irregular, frequently rapid ventricular response rate
- ECG in AFL: Atrial rate 250–350 bpm w/ ventricular response rate typically 150 bpm presence of “sawtooth” flutter (“F”) waves. Can be **typical** (spiky V1, negative in II, III, aVF, V5–V6) or **atypical** (appearance other than typical). F waves revealed via adenosine or vagal maneuvers. Most commonly **2:1 or 4:1** conduction.

Treatment

- Main objectives: Rate control, prevention of thromboembolism, & correction of rhythm
- When deciding on management strategies in the ED, several things to consider include:
  - a. Is the pt stable or unstable?
  - b. Is this 1st episode or recurrent episode, & is this part of a paroxysmal, persistent, or permanent duration paradigm?
  - c. If 1st-episode or paroxysmal, how long have sx been present (ie, <48 h)?
  - d. What is the pt's stroke risk?
  - e. Does the pt have a cardiologist/PCP w/ whom you can make joint decision or poor f/u?

**Rate control vs. rhythm control**: Numerous studies have sought to answer this question, but bottom line is that there appears to be no difference in symptomatic improvement, CHF, thromboembolic cx, severe bleeding, or mortality when comparing the 2 strategies (PIAF, *Lancet* 2000;356:1789; AFFIRM, *NEJM* 2002;347:1825; STAF, *J Am Coll Cardiol* 2003;41:1690; HOT CAFÉ, *CHEST* 2004;126:476); however, rhythm control seems to be a/w increased rates of hospitalization & adverse medication effects (PIAF, *Lancet* 2000;356:1789; AFFIRM, *NEJM* 2002;347:1825)

*Note: majority of these studies included pts w/ persistent AF, thus may not be generalizable to ED pt presenting w/ 1st episode or paroxysmal AF

- Suggested initial tx algorithms (Adapted from guidelines: *Can J Cardiol*)
Figure 1.5 Unstable patient with afib. (Note: Mean energy level for successful cardioversion 50 J biphasic and 200 J monophasic [Am J Cardiol 2004;93:1495–1499]. There may be higher first-shock success for DVVC if initial energy used 200 J vs.100 J [BEST-AF, Heart 2008;94:884–887]).
Figure 1.6 Stable patient, first episode or paroxysmal afib. Note: These patients may undergo cardioversion without anticoagulation, however, consider delaying DCCV and anticoagulate for 3 wk if high risk of stroke (ie, mechanical valve, RHD, recent CVA/TIA).

Figure 1.7 Stable patient, persistent or permanent afib.
• **Rate control**: βB or nondihydropyridine CCBs recommended as 1st-line therapy for rate control. CCB, however, should be avoided in pts w/ ADHF & AF.

• Digoxin can be added to therapy w/ βB or CCB in pts whose HR is not controlled

• Dronedarone may be added for additional rate control w/ uncontrolled ventricular rate despite above therapy

• IV administration of digoxin or amiodarone is recommended to control HR in pts w/ AF & HF

• Amiodarone for rate control should be reserved for exceptional cases in which other means are not feasible or insufficient

• IV procainamide, disopyramide, ibutilide, or amiodarone may be considered for HD stable pts w/ AF involving conduction over an accessory pathway. In this situation, IV CCB or digoxin should be avoided as they may paradoxically accelerate the ventricular response.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Initial Dose</th>
<th>Mant Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>βB</td>
<td>Metoprolol</td>
<td>2.5–5 mg IV bolus q5min × 3</td>
</tr>
<tr>
<td></td>
<td>Esmolol</td>
<td>500 mcg/kg IV q4min × 3</td>
</tr>
<tr>
<td></td>
<td>Propranolol</td>
<td>0.15 mg/kg IV q5min × 5</td>
</tr>
<tr>
<td>CCB</td>
<td>Diltiazem</td>
<td>0.25 mg/kg IV × 1; may repeat 0.25–0.35 mg/kg IV after 15 min</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>0.075–0.15 mg/kg IV; may repeat dose after 15–30 min</td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.25 mg IV q2h, up to 1.5 mg</td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>150 mg IV over 10 min</td>
<td>0.5–1 mg/min IV</td>
</tr>
</tbody>
</table>

• **Direct current cardioversion**: Recommended dose 150–200 J biphasic waveform

• Mean energy level for successful cardioversion 50 J biphasic & 200 J monophasic (Am J Cardiol 2004;93:1495). There may be higher 1st-shock success for DVVC if initial energy used 200 J vs. 100 J (BEST-AF,
Pretreatment w/ amiodarone, flecainide, ibutilide, propafenone, or sotalol can be used to enhance the success of DCCV & prevent recurrent AF.

**Pharmacologic cardioversion:** Administration of flecainide, dofetilide, propafenone, or ibutilide is recommended for pharmacologic cardioversion.

Procainamide has been shown to be effective in ED population w/ 58.3% cardioversion, w/ 91.7% success rate if followed by DCCV in nonresponders *(CMEJ 2010;12(3):181)*.

Amiodarone is a reasonable option, but digoxin & sotalol may be harmful for cardioversion & are not recommended.

βB or nondihydropyridine CCBs should be given before administering class I antiarrhythmic agents.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Initial Dose</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class Ia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procainamide</td>
<td>15–17 mg/kg IV over 60 min</td>
<td>• Preferred w/ WPW&lt;br&gt;• May cause hypotension</td>
</tr>
<tr>
<td>Class Ic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propafenone</td>
<td>450 mg PO (&lt;70 kg)&lt;br&gt;600 mg PO (&gt;70 kg)&lt;br&gt;2 mg/kg IV</td>
<td>• May cause hypotension, bradycardia&lt;br&gt;• CI in pts w/ ischemic HD, LV dysfxn, structurally abnl heart</td>
</tr>
<tr>
<td>Flecainide</td>
<td>200 mg PO (&lt;70 kg)&lt;br&gt;300 mg PO (&gt;70 kg)&lt;br&gt;2 mg/kg IV</td>
<td>• May cause hypotension, bradycardia&lt;br&gt;• ↓ dose in renal insuff&lt;br&gt;• CI in pts w/ ischemic HD, LV dysfxn, structurally abnl heart</td>
</tr>
<tr>
<td>Class III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dofetilide</td>
<td>0.5 mg PO (eGFR &gt;60)&lt;br&gt;0.25 mg PO (eGFR 40–60)&lt;br&gt;0.125 mg PO (GFR 20–40)&lt;br&gt;CI if eGFR &lt;20</td>
<td>• 2–3% risk torsades de pointes; CI long QT, bradycardia&lt;br&gt;• Require hospitalization for initiation given QTc prolongation</td>
</tr>
<tr>
<td>Ibutilide</td>
<td>1 mg over 10 min (&gt;60 kg)&lt;br&gt;0.01 mg/kg, 10 min (&lt;60 kg)&lt;br&gt;<em>May repeat once at same dose if doesn’t terminate</em></td>
<td>• CI in pts w/ hypokalemia, prolonged QTc, torsades de pointes</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>5–7 mg/kg IV over 30 min, then 1.2–1.8 g/day continuous IV infusion</td>
<td>• SE: Hepatotoxicity, hypothyroid, thyrotoxicosis, pneumonitis, pulmonary fibrosis, corneal</td>
</tr>
</tbody>
</table>
Anticoagulation: All pts w/ AF or AFL (paroxysmal, persistent, or permanent) should be stratified using a predictive index for stroke (ie, CHADS$_2$ or CHA$_2$DS$_2$-VASc) & for the risk of bleeding (ie, HAS-BLED) & most pts should receive anticoagulation.

- Pts w/ very low risk of CVA (CHADS$_2$ = 0) should receive ASA 81–325 mg/d
- Pts w/ low risk of CVA (CHADS$_2$ = 1) should receive oral anticoagulation w/ either warfarin or dabigatran, but ASA is reasonable for some pts
- Pts w/ mod risk of CVA (CHADS$_2$ ≥ 2) should receive oral anticoagulation w/ either warfarin or dabigatran
- Most pts should receive dabigatran 150 mg PO BID preferable to warfarin when anticoagulation indicated
- Newer effective oral anticoagulants include rivaroxaban 20 mg QD (ROCKET-AF, NEJM 2011;365:883) & apixaban 5 mg PO BID (ARISTOTLE, NEJM 2011;365:981)
- Anticoagulation not recommended for pts w/ lone AF

<table>
<thead>
<tr>
<th>CHADS$_2$ Risk Criteria</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHF</td>
<td>1</td>
</tr>
<tr>
<td>HTN</td>
<td>1</td>
</tr>
<tr>
<td>Age &gt;75 y/o</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke or TIA (prior)</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHADS$_2$ Score</th>
<th>Adjusted Stroke Rate%/y (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.9% (1.2–3)</td>
</tr>
<tr>
<td>1</td>
<td>2.8% (2–3.8)</td>
</tr>
<tr>
<td>2</td>
<td>4% (3.1–5.1)</td>
</tr>
<tr>
<td>3</td>
<td>5.9% (4.6–7.3)</td>
</tr>
<tr>
<td>4</td>
<td>8.5% (6.3–11.1)</td>
</tr>
<tr>
<td>5</td>
<td>12.5% (8.2–17.5)</td>
</tr>
</tbody>
</table>
### HAS-BLED Score for Estimating Major Bleeding Risk in Patients with AF *(CHEST 2010;138:1093)*

<table>
<thead>
<tr>
<th>Clinical Characteristic</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>HTN (SBP &gt;160 mmHg)</td>
<td>1</td>
</tr>
<tr>
<td>Abnl renal or liver fxn (1 pt each)**</td>
<td>1 or 2</td>
</tr>
<tr>
<td>Stroke</td>
<td>1</td>
</tr>
<tr>
<td>Bleeding</td>
<td>1</td>
</tr>
<tr>
<td>Labile INRs</td>
<td>1</td>
</tr>
<tr>
<td>Elderly (age &gt;65 y/o)</td>
<td>1</td>
</tr>
<tr>
<td>Drugs or EtOH use (1 pt each)****</td>
<td>1 or 2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HAS-BLED Score</th>
<th>Major Bleed%/y*</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.13%</td>
</tr>
<tr>
<td>1</td>
<td>1.02%</td>
</tr>
<tr>
<td>2</td>
<td>1.88%</td>
</tr>
<tr>
<td>3</td>
<td>3.74%</td>
</tr>
<tr>
<td>4</td>
<td>8.70%</td>
</tr>
<tr>
<td>5</td>
<td>12.50%</td>
</tr>
<tr>
<td>6–9</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

*Major bleeding defined as any bleeding requiring hospitalization &/or causing a decrease in Hgb of >2 g/L &/or requiring blood transfusion that was not a hemorrhagic stroke.

**Abnl renal fxn defined as chronic dialysis, renal transplant, or Cr >200 μmol/L (2.3 mg/dL). Abnl liver fxn defined as chronic hepatic dz (ie, cirrhosis), Tbili >2× ULN, in association w/ AST/ALT. ALP >3× ULN.

***Drugs included anti-plt agents & NSAIDs.

### Disposition

- **Home:** Pts who convert to sinus, or are rate controlled, & anticoagulated if necessary
- **All discharged pts should get close PCP or cardiology f/u**
- **EDOU:** Depending on local clinical protocols
- **Admit:** Pts w/ acute underlying illness, ongoing sx, or poor rate control
Pearls

- Risk of stroke is similar in all forms of AF/AFL (recurrent paroxysmal, persistent, & permanent AF, & AFL)
- Spontaneous cardioversion occurs w/i 24 h in 50–67% acute AF
- 5–8% elderly have recurrent AF

Pre-excitation

Definition

- **Accessory pathway**: A bypass tract that conducts impulses b/w atria
- **Wolff–Parkinson–White**: Accessory conduction pathway evident on resting ECG
- **Orthodromic AVRT**: Impulse travels down AV node (fast), then conducts retrograde, up the accessory pathway (slowly) → thus narrow-complex QRS
- **Antidromic AVRT**: Impulse travels down the accessory pathway (slowly), then conducts retrograde, up the AV node (fast) → thus wide-complex QRS

Evaluation

- ECG & rhythm strip
- Orthodromic AVRT: Narrow complex tachycardia
- Antidromic AVRT: WCT

Treatment

- AVRT: Vagal maneuvers, βBs, CCB
- AF/AFL w/ pre-excitation → cardiology consult, DC cardiovert, or use procainamide; βB & CCB are ineffective & can precipitate VF

**Ventricular Tachycardia**

**Approach**
- Determine if pt is stable or unstable → use ACLS protocol for any pt w/ unstable VT
- Differentiate VT from nonsustained VT (NSVT), & other causes of WCT (see above)
- Differentiate monomorphid from polymorphid VT

**Definition**
- NSVT: VT lasting <30 s
- SVT w/ aberrancy: VT look-alike b/c abnl conduction → WCT. Caused by fixed BBB, rate-related BBB, or accessory pathway
- Torsades de pointes: Polymorphid VT + prolonged QT

**Causes**
- **Monomorphid, structurally abnl heart:** Prior MI, CMP, arrhythmogenic RV dysplasia
- **Monomorphid, structurally nl heart:** Idiopathic VT
- **Polymorphid:** Ischemia, CMP, torsades de pointes, Brugada syndrome (see below)

**History**
- Palpitations, lightheadedness, CP, SOB, nausea, syncope, unresponsiveness; PMH: CAD, CMP, multiple CAD RFs, & FHx sudden death all ↑ risk VT

**Evaluation**
- ECG, rhythm strip, lytes, Ca, Mg, PO₄, cardiac markers; CXR; digoxin level if appropriate

**Treatment**
- **Unstable VT:** ACLS protocol
- **Stable VT:** Use either:
  - Lidocaine: 100 mg IV load, then 1–4 mg/min
  - Amiodarone: 150 mg IV load, then 1 mg/min
- **Polymorphid VT:** Magnesium 2–4 g IV bolus
- **Other:** Replete electrolytes (Ca, Mg, PO₄); treat coincident ischemia if present
Disposition

- Admit to cardiac step-down unit or cardiac ICU

Pearls

- Assume all WCT to be ventricular unless proven o/w
- Best clinical predictors that WCT is VT → prior MI, CHF, LV dysfxn *(Am J Med 1998;84:53)*

| Brugada Criteria for WCT Suggesting VT *(Circulation 1991;83:1649)* |
|-------------------------|----------------------------------|
| **Criterion**           | **ECG Appearance**               |
| AV dissociation         | Independent P waves, capture/fusion beats |
| Wide QRS                | RBBB type: >140 ms  LBBB type: >160 ms |
| Extreme axis deviation  |                                  |
| Atypical QRS morphology for BBB | –QRS b/w +180° & –90° (–QRS lead I, & –QRS aVF)  RBBB type: Absence of tall R’ in V1, r/S ratio <1 in V6  LBBB type: Onset to nadir >60–100 ms in V1, Q wave in V6 |
| Concordance             | Of QRS in precordial leads w/ same pattern & direction |

Brugada Syndrome

Definition

- Incomplete RBBB w/ STE V1–V3 caused by alteration of the myocyte Na channel, a/w VT & sudden cardiac death

History

- Classically young, o/w healthy male, FHx sudden D; sx: Presyncope, syncope, cardiac arrest

Evaluation

- ECG, electrolytes, Ca, Mg, PO₄

Treatment

- Tele; electrophysiology consult

Disposition

- If incidental finding, refer to cardiology for f/u. O/w, admit to Tele bed for EP study, possible ICD placement.

PACEMAKER AND AICD MALFUNCTION
Definition

- **PM**: Intracardiac device used for significant AV block &/or sinus node dysfxn
- **AICD**: Intracardiac device for the termination of VF/VT, & prevention of sudden cardiac death for pts s/p VF/unstable VT arrest, persistent EF ≤30–35%, Brugada, or long QT syndrome \( (Circulation\ 2007;115:1170;\ NEJM\ 1997;337:1576) \)
- Biventricular pacing (cardiac resynchronization therapy): RA, RV, & coronary sinus leads → synchronize RV & LV function → ↓ CHF sxs & hospitalization, ↑ survival \( (NEJM\ 2004;350:2140;\ 2005;352:1539) \)

Approach

- Obtain an ECG & rhythm strip immediately
- Obtain the make & model of the device (most pts have a card, o/w obtain AP CXR → magnify device to obtain model number → internet search for type)
- Common PM codes: DDD (dual chamber paced, sensed, & response to sensed beat) & VVI (dual chamber paced, sensed, & inhibitory response to sensed beat)

Evaluation

- Magnet placed over device
- **PM**: Inhibits sensing, paces at fixed rate regardless of intrinsic cardiac activity
- **AICD**: Inhibits further firing, though not bradycardic pacing

<table>
<thead>
<tr>
<th>Pacemaker Malfunction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
</tr>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Failure of output → no pacer spike despite indication to pace</td>
</tr>
<tr>
<td>Failure to capture → pacing spikes <em>not</em> followed w/ depolarization</td>
</tr>
<tr>
<td>Oversensing → pacer spike despite <em>no</em> indication to pace</td>
</tr>
<tr>
<td>Undersensing → <em>no</em> pacer</td>
</tr>
<tr>
<td>PM-mediated tachycardia</td>
</tr>
</tbody>
</table>

### History
- Lightheadedness, palpitations, syncope

### Findings
- ↑, ↓, &/or irregular HR, ↓ BP

### Evaluation
- ECG, CXR (to visualize device & leads)

### Treatment
- Transcutaneous pacing: For unstable pt
- **MAGNET: FOR PM-mediated tachycardia:** Magnet over the PM → paces @ 80 bpm OR OVERSENSING

### Disposition
- Consult EP or device rep. for interrogation & reprogramming; to cath lab for lead/battery replacement

---

### AICD Firing *(JAMA 2006;296:2839; NEJM 2003;349:1836; JACC 2006;48:1064)*

<table>
<thead>
<tr>
<th>Pt Sxs</th>
<th>AICD Interrogation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt-sensed AICD* firing</td>
<td>No firing</td>
</tr>
<tr>
<td></td>
<td>Inappropriate firing</td>
</tr>
<tr>
<td></td>
<td>Appropriate firing</td>
</tr>
</tbody>
</table>

*AICDs can also malfunction like PMs (see PM section above).*

### History
- **AICD firing:** Sudden jolt of pain
- **Premonitory sxs:** Palpitations, LH, dyspnea, CP
- **Precipitants:** Exercise, illness, noncompliance w/ antiarrhythmics, new meds

### Evaluation
- ECG (ischemia, ↑ QT), CBC, Chem 7, cardiac markers, CXR
**Treatment**
- Treat 1°-illness, follow ACLS protocol for arrhythmia

**Disposition**
- Consult EP or device rep. for interrogation & reprogramming
- No firing (nl interrogation despite sxs): Look for other cause of sxs → d/c home
- Inappropriate firing (based on interrogation): Treat underlying condition; reprogram if necessary
- Appropriate firing (based on interrogation): Admit to Tele unit or CCU
  - Look for precipitants: VT, abnl electrolytes, ↑ QT, ischemia, medication noncompliance or abuse

**Pearl**
- If make/model # of device unknown, magnification of PA CXR will reveal device-sp code in small print.
Pneumonia

Definitions  *(Clin Inf Dis 2016;63(5):575–582)*

- Community-Acquired PNA (CAP): Occurs out of hosp or w/i 48 h of admx; no HCAP factors
- Healthcare-Associated PNA (HCAP): a/w hosp admx (2+ d) w/i last 90 d; residence in long-term care or nursing home; immunosuppression; family member w/ MDR organism; or any of the following w/i 30 d: IV abx, HD, home wound care
- Hospital-/ Vent-Acquired PNA (HAP/VAP): Occurs >48 h after hosp admx or intubation

History

- Typical CAP (eg, *Strep, Klebsiella, Haemophilus*): Fever/chills, SOB, CP, cough, sputum
- Atypical CAP (eg, *Mycoplasma*): Low-grade fever, mild/mod SOB, CP, dry cough, GI sx
- Influenza: Fevers/chills, myalgias, malaise, HA, sore throat, dry cough
- Legionella: Severe PNA in elderly; a/w hyponatremia, GI sx
- Ask about risk factors for special organisms:
  - TB: Homeless, HIV+/immunosuppressed, IVDA, incarceration, travel to endemic region; present w/ blood-tinged sputum, night sweats, fevers, weight loss
  - PCP: Poorly controlled HIV (CD4 <200, Lymph <1 k); presents w/ subacute tachypnea
  - MDR organisms: IV abx w/i 90 d, chronic HD, immunosuppression, recent influenza (risk for MRSA), CF/bronchiectasis (*pseudomonas*), asplenia

Physical Exam

- Fever, tachycardia, tachypnea, hypoxia, rales, decreased breath sounds
- PNA less likely w/ nl VS & clear lungs, except in elderly, infants, immunosuppressed

- Who needs a CXR? Reserve for pts needing hospitalization, those w/ abnl VS or PEx, age extremes, concerning comorbidities, poor outpt f/u, high morbidity if PNA not detected
- CXR: Focal consolidation (typical); diffuse interstitial pattern (atypical); bat-wing pattern (PCP); hilar adenopathy, calcified or cavitary apical lesions (TB)
- CBC, Chem 7 +/- lactate (if suspecting sepsis), ABG & LDH (if PCP), BCx (if cavitary, parapneumonic eff., immunosupp/leukopenia, asplenia, liver dz, hx ETOH, failed outpt abx; ICU admx), Sputum cx (if cavitary, parapneumonic eff., hx severe lung dz, failed outpt abx, ICU admx), Influenza (in epidemics), Pneumococcal UAT (if asplenia, liver dz, immunosupp/leukopenia, parapneumonic eff.; Se 50–80%, Sp 90%), Legionella UAT (consider if recent travel, elderly, hypoNa, parapneumonic eff.; Se 80–95%, Sp 99%)

<table>
<thead>
<tr>
<th>Scenario/Etiology</th>
<th>Empiric Treatment Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CAP, outpt</strong></td>
<td>Healthy &amp; no recent abx w/ 90 d: macrolide or doxycycline Comorbidities or recent abx: resp. fluoroquinolone OR (macrolide + [amoxicillin or amoxicillin/clav. or 2nd-gen cephalosporin])</td>
</tr>
<tr>
<td><strong>CAP, inpt</strong></td>
<td>Resp fluoroquinolone OR (macrolide + [ampicillin or 3rd-gen cephalosporin]) Consider pseudomonas &amp; MRSA coverage if severe (eg, ICU). Legionella is covered by macrolide or fluoroquinolone.</td>
</tr>
<tr>
<td><strong>MDR risk factors</strong></td>
<td>Vancomycin + (antipseudomonal PCN or 3rd-gen cephalosporin or carbapenem). Refer to local antibiogram.</td>
</tr>
<tr>
<td><strong>Suspect PCP</strong></td>
<td>PaO₂ &gt;70: TMP-SMX DS 2 Tabs PO q8h OR (TMP 5 mg/kg PO TID + dapsone 100 mg PO QD) OR (clindamycin + primaquine) OR atovaquone. PaO₂ &lt;70: (TMP-SMX [15 mg of TMP component/kg] PO/IV q8h or [clindamycin + primaquine] or pentamidine) + (prednisone [40 mg BID] or methylprednisolone [40–60 IV Q6H] x 21 d); NNT for early steroids is 9 (Cochrane 2006;19;(3):CD006150).</td>
</tr>
<tr>
<td><strong>Aspiration PNA</strong></td>
<td>3rd-gen cephalosporin OR fluoroquinolone ± (clindamycin or metronidazole). If sick, b-lactam/b-lactamase inhibitor.</td>
</tr>
<tr>
<td><strong>Influenza A &amp; B</strong></td>
<td>Oseltamivir (75 mg PO BID × 7 d), zanamivir Tx only reduces sx by 1 d (must be given w/ the 48 h of onset) Tx if critically ill, extremes of age, lung dz (inc. asthma), morbid obesity, immunosuppressed, pregnant</td>
</tr>
</tbody>
</table>

Influenza A & B (BMJ 2009;339:b5106)
Suspect TB
INH 5 mg/kg PO QD + Vit B₆ 25–50 mg PO QD (neuropathy) + rifampin 10 mg/kg (max 600 mg) PO QD + pyrazinamide 15–30 mg/kg (max 2 g) PO QD + ethambutol 15–25 mg/kg (max 2.5 g) PO QD; ensure infectious dz f/u
Airborne precautions, negative pressure room

Disposition
- CAP/HCAP: See PNA Severity Index Score & CURB-65 (below), unless need for IV abx
- PCP: Inpt unless S_pO₂ >95% w/o desaturation on exertion
- TB: Inpt, report to Dept of Health

### Pneumonia Severity Index *(NEJM 1997;336)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Points Assigned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td>If male: (+age); if female, (+age – 10); nursing home resident (+10)</td>
</tr>
<tr>
<td>Comorbidities</td>
<td>Neoplastic dz (+30); liver dz (+20); CHF (+10); cerebrovascular dz (+10); renal dz (+10)</td>
</tr>
<tr>
<td>Physical exam</td>
<td>AMS (+20); HR ≥125 (+20); RR &gt;30 (+20); SBP &lt;90 (+15); temp &lt;35°C or ≥40°C (+10)</td>
</tr>
<tr>
<td>Lab &amp; radiographic findings</td>
<td>pH &lt;7.35 (+30); BUN ≥30 mg/dL (9 mmol/L) (+20); Na &lt;130 (+20); glucose ≥250 mg/dL (14 mmol/L) (+10); HCT &lt;30 (+10); PaO₂ &lt;60 (+10); pl eff. (+10)</td>
</tr>
</tbody>
</table>

### PORT Score (Recommended Triage and Prognosis) Calculated from PSI

<table>
<thead>
<tr>
<th>Class</th>
<th>Score</th>
<th>Mortality (%)</th>
<th>Disposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>&lt;50</td>
<td>&lt;1</td>
<td>Outpt</td>
</tr>
<tr>
<td>II</td>
<td>≤70</td>
<td>&lt;1</td>
<td>Outpt</td>
</tr>
<tr>
<td>III</td>
<td>71–90</td>
<td>2.8</td>
<td>Outpt/inpt (clinical judgment)</td>
</tr>
<tr>
<td>IV</td>
<td>91–130</td>
<td>8.2</td>
<td>Inpt</td>
</tr>
<tr>
<td>V</td>
<td>&gt;130</td>
<td>29.2</td>
<td>ICU</td>
</tr>
</tbody>
</table>

### CURB-65 Score *(Thorax 2003;58(5):377)*

1 point each
- Confusion, Urea >20 mg/dL, RR >30, SBP <90, DBP <60, age >65

Score < 2
- Low risk, consider outpt tx; Mortality 0.7% (if 0), 3.2% (if 1)

Score = 2
- Short inpt hospitalization or close outpt supervision; Mortality 3%

Score > 2
- Hospitalize, consider ICU; Mortality 17% (if 3), 41.5% (if 4), 57% (if 5)
**Pearls**

- Special considerations: IVDU/endocarditis (multifocal PNA, esp b/l), malignancy (postobstructive PNA), postinfluenza (MRSA PNA), recent elective surgery (asp PNA risk increases w/ duration of anesthesia, periop NG tube, age [Arch Surg 1998;133(2):194–198])
- In severe PNA, high-flow NC (vs. NIPPV) reduces 30 d mortality, & may reduce need for intubation (esp. if PaO₂:FiO₂ < 200) (NEJM 2015;372(23):2185–2196)
- Consider social factors if discharging pt w/ PNA (eg, f/u, ability to comply w/ regimen)

**Acute Bronchitis**

**Etiology**

- Most commonly viral: parainfluenza, adenovirus, rhinovirus, coronavirus, RSV, influenza
- Atypical bacteria ~5% of cases (*chlamydia p.*, *mycoplasma*, B. *pertussis* esp. in epidemics)

**History**

- Cough >5 d (dry or wet), low-grade fever, myalgias, wheezing, often after URI sxs
- Consider pertussis: posttussive emesis, whoop, duration >1 wk (JAMA 2010;304(8):890)
- All-cause median duration of cough is 18 d; pertussis once called “100-day cough”

**Physical Exam**

- Fever uncommon (consider influenza or PNA); may have chest wall tenderness from muscle strain; lungs often clear but up to 40% have bronchospasm/wheeze

**Evaluation**

- CXR nl or bronchial wall thickening; mild leukocytosis
- Labs/CXR not routinely needed: Reserve for abnl VS, extremes of age, comorbidities

**Treatment**

- Supportive care, antipyretics, antitussive (e.g., Tessalon Perles 100 mg TID)
- No good evidence for or against OTC expectorants, decongestants, or antihistamines (Cochrane Database Syst Rev 2012;8:CD001831)
If wheezy or hx asthma: bronchodilator (albuterol MDI 2 puffs QID), can consider inhaled corticosteroids x 7 d (though no major data to support)
• Abx not routinely indicated *(Cochrane Database Syst Rev 2012;CD000245)*
  • Abx reduce duration of sx by <1 day *(NEJM 2006;355(20):2125–2130)*
  • Reserved for elderly, significant comorbidities, high suspicion for pertussis
  • Pertussis: Azithromycin 500 mg Day 1 then 250 mg QD × 4 d OR doxycycline 100 mg BID × 7 d; Abx limit transmission but minimal effect on sx duration (unless in first wk
• See PNA section for influenza tx guidelines

**Disposition**
• Discharge home w/ PCP f/u as needed; pts will likely recover in 2–3 wk

---

### DYSPNEA (SHORTNESS OF BREATH)

**Definition**

• Difficult or labored breathing (acute or progressive) often due to primary pulm or cardiovascular etiologies, but carries broad ddx (e.g., endo, heme, tox, neuromuscular)
• Always assess for respiratory distress: RR >24 or <8, tripoding, accessory muscle use, unable to speak in full sentences, altered mental status (AMS), abnl chest movement

<table>
<thead>
<tr>
<th>Approach to the Patient</th>
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</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
</tr>
<tr>
<td>Common or Severe Cardiac Etiologies</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td>Arrhythmia</td>
</tr>
<tr>
<td>Supraventricular</td>
</tr>
<tr>
<td>Nonsustained VT</td>
</tr>
<tr>
<td>Valve dz</td>
</tr>
<tr>
<td>Left heart (AV, MV)</td>
</tr>
<tr>
<td>Right heart (PV, TV)</td>
</tr>
<tr>
<td>Common or Severe Upper Airway Etiologies</td>
</tr>
<tr>
<td>Airway obstruction</td>
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<tr>
<td>-------------------------</td>
</tr>
<tr>
<td>• FB aspiration</td>
</tr>
<tr>
<td>• Epiglottitis</td>
</tr>
<tr>
<td>• Croup (pedi)</td>
</tr>
<tr>
<td>• Angioedema</td>
</tr>
<tr>
<td>• Abscess/hematoma</td>
</tr>
<tr>
<td><strong>Common or Severe Pulmonary Etiologies</strong></td>
</tr>
<tr>
<td>Pulm. edema (2/2 CHF, left-sided valve dz, myocarditis, constrictive pericarditis, tamponade)</td>
</tr>
<tr>
<td>PTX (spontaneous [esp tall/thin or emphysema], traumatic, barotrauma [scuba, inh drugs])</td>
</tr>
<tr>
<td>Pulm embolism</td>
</tr>
<tr>
<td>Obstructive lung dz</td>
</tr>
<tr>
<td>• Asthma</td>
</tr>
<tr>
<td>• COPD</td>
</tr>
<tr>
<td>• Bronchospasm (bronchitis, anaphylaxis)</td>
</tr>
<tr>
<td>• Tracheomalacia (premature infants)</td>
</tr>
<tr>
<td>PI eff. (2/2 PNA, HF, cancer, cirrhosis, rarely other causes)</td>
</tr>
<tr>
<td>PNA</td>
</tr>
<tr>
<td>Pneumonitis (drugs, XRT, environment exposure)</td>
</tr>
<tr>
<td>Malignancy</td>
</tr>
<tr>
<td>Pathophysiology</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>Metabolic/endocrine</td>
</tr>
<tr>
<td>Hematologic</td>
</tr>
<tr>
<td>Toxins</td>
</tr>
<tr>
<td>Mechanical</td>
</tr>
<tr>
<td>Neuromuscular</td>
</tr>
<tr>
<td>Psychogenic</td>
</tr>
</tbody>
</table>

**Asthma**

**Definition**
- Chronic recurrent inflammatory disorder w/ airway hyperresponsiveness, bronchospasm, & reversible airway obstruction

**Clinical Features**
- Progressive wheezing, dyspnea, chest tightness, cough (esp nocturnal)
- Always assess sx frequency, severity, duration, home txs;
  - Evaluate for triggers: Cold air, exercise, URI, stress, allergens, meds (NSAIDs, βBs), respiratory irritants (perfumes, smoke, detergents, dander, dust)
- Assess asthma hx: Past txs, baseline PEFR, no. ED visits/yr, admx/yr, prior intubations
**Figure 2.1** Treatment algorithm. From NHBLI Expert Panel Report 3, 2007. NIH Pub no. 08–4051.

**Physical Exam**
- Tachypnea, tachycardia, inspiratory/expiratory wheezes, prolonged expiration, decreased or no air movement, use of accessory muscles, tripoding, cyanosis

**Evaluation**
- CXR: Avoid in routine exacerbations; order to r/o PNA/PTX, elderly,
comorbidities
› PEFR: Compare to pt's baseline if he/she is aware. Varies by age, gender, & height. Average adult female: 300–470; adult male: 400–660.
› ABGs are not routinely indicated to assess for severity, but normocarbia in severe asthma may be a sign of “tiring out,” impending respiratory failure.

Pearls
› MDI w/ spacer as effective as nebulizers (but harder for ill pts) (NEJM 2010;363(8):755–764)
› Medium-dose inh budesonide Rx (21 d × 600–1200 mcg/d) at d/c in addition to PO corticosteroids decreases 21 d relapse by 48% (NEJM 2010;363(8):755–764)

Chronic Obstructive Pulmonary Disease (COPD)
Definition
› Progressive incompletely reversible airflow obstruction, w/ impaired gas exchange, usually w/ smoking hx. Formal dx needs PFTs (postbronchodilator FEV$_1$/FVC <70% predicted).

Mild (FEV$_1$ ≥ 80%), Mod (FEV$_1$ 50–80%), Sev (FEV$_1$ 30–50%), Very sev (FEV$_1$ < 30%)
History
› Cough (worse than baseline), increased sputum (purulence & volume), dyspnea, wheeze
› Precipitants: Cold weather (inc incidence in winter months), infxn (viral > bacterial), cardiopulmonary dz, PE (16% of acute exacerbations; Chest 2016), med changes

Physical Exam
› Chronic bronchitis (“Blue Bloater”): Cough w/ inc. sputum production; cyanotic, plethoric, not in overt resp distress; scattered rhonchi & rales
› Emphysema (“Pink Puffer”): Thin, anxious, dyspneic, tachypneic; noncyanotic, tripoding, pursed-lip exhalation (for auto-PEEP), diminished breath sounds

Evaluation
› ECG for associated dysrhythmia (AF or MAT), cor pulmonale (P pulmonale: Big P in II)
› CXR to r/o PNA, PTX, eff., edema, malignancy
› Consider CTA Chest (2/3 of PEs in COPD are segmental or larger; Chest
Blood gas to evaluate pH & PaCO₂, BMP & CBC (esp if admitted)
- If chronic resp acidosis present, compare PaCO₂ w/expected (calculated from HCO₃⁻)
- Influenza (if epidemic), sputum cx (if severe COPD & PNA)
- Exacerbation severity: No resp failure (RR 20–30, no access muscles, no AMS, hypoxia FiO₂ <40%, nl PaCO₂), Non–life-threatening acute resp failure (RR >30, +access muscles, no AMS, FiO₂ <40%, PaCO₂ 50–60), Life-threatening acute resp failure (same, but +AMS, FiO₂ >40%, PaCO₂ >60 or pH ≤7.25) (GOLD 2017 Report)

Treatment (GOLD 2017 Report)
- Titrate supplemental O₂ (goal S_pO₂ 88–92%): Chronic hypoxemia inc. risk of O₂-induced hypoventilation (Crit Care 2012;16(5):323); ~2× mortality w/ high-flow O₂ (BMJ 2010;342:c5462)
- Albuterol (short-acting β-agonist): 2.5–5 mg neb q30min × 3, then q4h OR MDI w/ spacer
- Neb vs MDI (mild cases): no diff in sustained FEV₁ or admx (Cochrane 2016;(8):CD011826)
- Ipratropium bromide (anticholinergic): 0.5 mg nebulized q30min × 3 doses, then q4h (synergistic effect w/ albuterol, so give together)
- Steroids: Prednisone 40 mg PO QD (5–7 d) OR methylprednisolone (for severely ill pts)
- Abx recommended if increased sputum purulence & either increased SOB or sputum volume, OR life-threatening acute resp distress
  - Abx dec mortality 12% (NNT 8) & tx failure 31% (NNT 3) (Cochrane 2006; (2):CD004403)
  - Choice based on RFs (age >65, FEV₁ <50%, recent abx, heart dz); duration 5–7 d
  - Outpt w/o RFs: Macrolide, amoxicillin, doxycycline, or TMP/SMX
  - Outpt w/ RFs: Fluoroquinolone or amoxicillin/clavulanate
  - Inpt: Fluoroquinolone (esp if pseudomonas RF) OR (3rd-gen cephalosporin + Macrolide)
- Positive Pressure Ventilation (PPV):
  - Noninvasive PPV (BiPAP): resp acidosis, severe SOB or fatigue; watch for PTX w/PPV
  - Decreases mortality by 50% (NNT 8), intubation by 60% (NNT 3), tx
failure by 50%, & hosp LOS by >3 d compared to usual care (Cochrane 2004;(3):CD004104)

- Invasive (intubation): Not tolerating BiPAP, impending resp failure, CV instability, AMS

**Disposition**
- Home: Mild sx, ambulatory $S_pO_2 > 90\%$, <Q4H bronchodilators, outpt f/u, home support
- Early f/u dec. mortality; 20% pts not back at prior baseline by 2 mo (GOLD 2017 Report)
- Admx: Incomplete tx response, sig. below baseline, mx comorbidities, severe COPD / freq. exacerbations, elderly, poor home support (Am J Respir Crit Care Med 2013;187(4):347)

**Acute Respiratory Distress Syndrome (ARDS)**

**Berlin Definition** (JAMA 2012;307(23):2526–2533)
- Acute (sx < 1 wk) diffuse inflammatory lung injury, characterized by vascular leak, edema, & diffuse alveolar damage; imaging w/ b/l opacities, not fully 2/2 cardiac failure or fluid overload; $PaO_2:FiO_2$ 200–300 (mild), 100–200 (mod), <300 (sev) w/ PEEP ≥ 5 cm H$_2$O

**Pathophysiology**
- Impaired gas exchange, poor compliance (stiff lungs), intrapulmonary shunt

**Etiology**
- Direct lung injury: PNA, aspiration, near-drowning, hydrocarbons, inhalational injury, embolism (thrombotic, fat, air, amniotic)
- Systemic: Sepsis, shock, DIC, trauma, burns, transfusion, pancreatitis, meds

**Clinical Features**
- Rapid progressive dyspnea (<1 wk), cyanosis, crackles, & eventual respiratory failure

**Evaluation**
- Dx requires ABG ($PaO_2:FiO_2 < 300$) & CXR w/ bilateral pulm edema
- May need TTE to r/o cardiac etiology, bronchoscopy to r/o diffuse alveolar hemorrhage

**Treatment**
- Supportive, focus on treating the underlying condition
Minimize barotrauma: low TV (<6 mg/kg), keep $P_{Plat} <30$
Avoid hyperoxia: wean FiO$_2$, maintain high PEEP to keep alveoli open.
Avoid excess fluids: (CVP goal 4–6 cm if CVC present) (NEJM 2006;354(24):2564–2575)
Excess volume initially may negate any subsequent benefit from conservative fluid management in ICU (Crit Care Med 2016;44(4):782–789)
Refractory hypoxia: Best PEEP trial, paralysis, inh prostacyclin, prone positioning, ECMO
No consensus on role of steroids; most meta-analyses show no mortality benefit

Upper Airway Obstruction/Foreign Body (FB)

History
- Acute FB aspiration: May be witnessed but often hx is unclear in adults
  - RFs: Extremes of age, neuro disorders, syncope, szs, alcohol or sedative abuse
  - DDx: angioedema, infectious etiology (eg, epiglottitis), soft tissue abscess/hematoma
- Subacute (eg, malignancy, expanding goiter): Often a delayed Dx (eg, wheezing unresponsive to bronchodilators)

Physical Exam
- General appearance: May arrive cyanotic & in respiratory arrest if total obstruction
- In breathing pt, respiratory exam depends on degree & location of obstruction: dec air movement, stridor, wheezing, secretion intolerance. Do not underestimate pt distress.

Evaluation
- CXR, XR neck rarely shows FB. Diagnostic & therapeutic flex bronchoscopy is standard.

Treatment
- If still breathing: Airway equipment, including cricothyrotomy kit, at bedside. Prepare for transfer to OR to remove FB in a controlled environment (bronchoscopy or DL).
- If not breathing: Attempt direct laryngoscopic visualization & removal of FB w/ forceps. If unsuccessful, perform surgical airway.
If FB moves inferior to vocal cords but still occluding, push object into 1 lung by pressure from Ambu bag/ETT; once intubated, position ETT to ventilate contralateral lung.

**Disposition**
- Flex bronchoscopy successful in 90% cases (*Respir Care* 2015;60(10):1438–1448)
- If object is safely removed & pt stable, can discharge home

---

**HEMOPTYSIS**

**Definition**
- Expectoration of blood or blood-stained sputum from below the vocal cords
- “Massive” hemoptysis: no defined volume (generally >500 cc/d or >100 cc/h), but any volume inhibiting breathing should be treated similarly; high mortality 2/2 asphyxiation

- Bronchial arteries (high pressure) > pulm arteries (low pressure) > alveoli

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulm</td>
<td>COPD, CF, bronchiectasis, pulm HTN, PE, AVM, lung trauma</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Pulm edema (eg, 2/2 CHF, mitral valve pathology)</td>
</tr>
<tr>
<td>Infectious</td>
<td>Acute bronchitis (#1 cause), PNA, TB, abscess, fungal infxn</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Malignancy (primary or met), carcinoid</td>
</tr>
<tr>
<td>Autoimmune</td>
<td>Goodpasture’s (anti-BM), granulomatosis polyangiitis (ANCA+)</td>
</tr>
<tr>
<td>Other</td>
<td>Recent instrumentation, tracheoarterial fistula (recent thoracic/vasc surgery), FB aspiration, inh cocaine, Osler–Weber–Rendu (telangiectasias), spontaneous (coagulopathy),</td>
</tr>
</tbody>
</table>

**Approach to Patient**

**History**
- Onset (sudden vs. progressive); quantity of blood; differentiate from GI or ENT source
- ROS: Fever, SOB, CP, weight loss, epistaxis (granulomatosis
polyangiitis, coagulopathy)

- Identify hx or RFs for COPD, PE, TB, CHF, cancer, autoimmune dz, coagulopathy

**Physical Exam**

- Assess airway first, if compromised, proceed directly to stabilizing airway
- Lungs: May show signs of COPD, PNA, edema
- Cardiac: for signs of CHF or valve dz
- Skin: Evaluate for evidence of bleeding &/or telangiectasias

**Evaluation**

- Labs: CBC, PT, PTT; type & screen. Consider AFB, BNP, D-dimer, UA (Goodpasture, granulomatosis polyangiitis) based on clinical scenario
- Imaging: CXR if unstable; chest CT if stable (much more helpful); ± bronchoscopy

**Treatment**

- Airway: HOB >45°; lean to side of bleeding (if known), suction, supplemental O₂ prn
  - If intubation necessary: double suction, large-bore ETT (consider advancing ETT into unaffected lung; double-lumen ETT if skilled operator), urgent bronchoscopy
- Definitive management: Minor hemoptysis can usually be managed conservatively, but if massive requires bronchoscopy or IR embolization, surgical resection if all else fails

**Disposition**

- Healthy, minimal bleeding: Get CXR; if negative: Home, outpt f/u
- High-risk pt, minor bleeding: Get CT, consider admit for observation, bronchoscopy
- Massive: ICU, consult pulmonology, interventional radiology, thoracic surgery
### Approach

- Assess nature of pain: Location, acute or chronic, constant or intermittent, relation to eating, associated sx$s$ such as fever, nausea, vomiting, dysuria, change in bowel habits
- Always ask about previous abd surgeries
- Labs depend on presentation. Consider CBC, BMP, UA, LFTs, lipase, hCG, lactate
- In the elderly, low threshold to evaluate for AAA w/ bedside US & ACS w/ EKG

<table>
<thead>
<tr>
<th>Location</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>RUQ</td>
<td>Cholelithiasis, acute cholecystitis, cholangitis, acute hepatitis, perforated duodenal ulcer, RLL pneumonia, pulmonary embolism (PE)</td>
</tr>
<tr>
<td>LUQ</td>
<td>Gastritis/PUD, splenic enlargement/rupture/infarction, LLL pneumonia, PE</td>
</tr>
<tr>
<td>Epigastric</td>
<td>Gastritis/PUD, pancreatitis, MI, myocarditis (see Cardiology), GERD</td>
</tr>
<tr>
<td>Lower Quadrants</td>
<td>Ruptured ectopic pregnancy, ovarian cyst/torsion, PID/TOA, endometriosis, kidney stone, incarcerated/strangulated hernia</td>
</tr>
<tr>
<td>RLQ</td>
<td>Appendicitis, Meckel's diverticulum, psoas abscess</td>
</tr>
<tr>
<td>LLQ</td>
<td>Diverticulitis</td>
</tr>
<tr>
<td>Diffuse</td>
<td>Early appendicitis, mesenteric ischemia, gastroenteritis, peritonitis, AAA, SBO, large bowel obstruction/volvulus, spontaneous bacterial peritonitis, IBD, colitis, DKA, sickle cell crisis, irritable bowel syndrome, anaphylaxis, colon ischemia, constipation</td>
</tr>
</tbody>
</table>

**Right Upper Quadrant Pain**
# Biliary Etiologies

<table>
<thead>
<tr>
<th>Dx</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholelithiasis</td>
<td>The presence of stones in the gallbladder</td>
</tr>
<tr>
<td>Biliary Colic</td>
<td>Intermittent obstruction of cystic duct or ampulla of Vater</td>
</tr>
<tr>
<td>Choledocholithiasis</td>
<td>Full obstruction of the CBD by a stone</td>
</tr>
<tr>
<td>Cholecystitis</td>
<td>Acute inflammation of the gallbladder due to obstruction in cystic duct, often a stone</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>Infection of the CBD, 80% caused by stone</td>
</tr>
</tbody>
</table>

## Cholelithiasis

### Presentation
- Intermittent epigastric &/or RUQ pain, +N/V, a/w fatty meals
- Pain may radiate around to the back or to the R scapula
- In biliary colic, sxs generally resolve completely in b/w episodes (minutes to hours)
- Mild RUQ tenderness but no fever or Murphy’s sign
- In choledocholithiasis & cholecystitis, sxs will become constant

### Evaluation
- Nl labs in biliary colic
- RUQ U/S spec/sens is 90–95% for stones.

### Treatment
- NSAIDs, opiate analgesics, antiemetics; elective surgical management

### Disposition
- If pain controlled, d/c home w/ surgery f/u to consider cholecystectomy
- If persistent pain, consider posisibility of impacted stone in GB neck/impending cholecystitis

### Pearls
- Acutely, biliary colic presents w/ diffuse upper abd pain before localizing to the RUQ
- RFs for gallstones include female gender, increasing age & parity, & obesity

## Choledocholithiasis

### Presentation
Biliary colic that becomes constant; late presentation may be a/w jaundice
Mild RUQ tenderness but no fever or Murphy's sign

Evaluation
- Obstructive LFT pattern, U/S shows dilated CBD >6 mm

Treatment
- ERCP-guided stone removal +/- cholecystectomy

Disposition
- Admit medicine. Usually initially managed by GI.

Cholecystitis

Presentation
- Persistent RUQ pain w/ N/V; may be accompanied by fever
- In elderly, delayed presentation w/ fever & poorly localized abd pain
- RUQ tenderness; Murphy's sign (arrest of inspiration w/ RUQ palpation), or Sonographic Murphy’s sign (pain w/ palpation of visualized gallbladder w/ U/S probe); fever

Evaluation
- CBC (elevated WBC ± left shift), LFTs (may be elevated but are often nl), RUQ US: The presence of stones, thickened gallbladder wall (>3 mm), & pericholecystic fluid has a PPV of >90%, but early presentation may lack US findings
- HIDA scan: May be considered if US is equivocal; high sens/spec for GB duct obstruction

Treatment
- 2nd- or 3rd-generation cephalosporin (E. coli, Enterococcus, Klebsiella) broaden coverage if septic
- Surgical consult for cholecystectomy; may do percutaneous drain if poor surgical candidate

Disposition
- Admit for surgical management

Cholangitis

Presentation
- Charcot’s triad: RUQ pain, jaundice, fever (present in 70% of pts)
- Reynold's pentad: Charcot’s triad +shock & MS changes (present in 15% of pts)
Evaluation
· Labs: ↑ WBC, ↑ LFTs, ↑ alk phos, positive blood cultures
· US/CT not very sens; can be suggestive
· ERCP is diagnostic & can be therapeutic if obstructing stone is found

Treatment
· Broad-spectrum abx for gram-negative enterics (eg, *E. coli*, *Enterobacter*, *Pseudomonas*): Piperacillin/tazobactam OR ampicillin/sulbactam OR ticarcillin/clavulanate OR ertapenem OR metronidazole + (ceftriaxone OR ciprofloxacin)

Disposition
· Admission to medicine for IV abx ± ERCP w/ surgery consultation

Pearls
· 80% pts respond w/ conservative mgmt & abx w/ elective biliary drainage
· 20% require urgent ERCP biliary decompression, percutaneous drainage, or surgery
· 5% mortality

---

**Epigastric Pain**

Pancreatitis

Etiology
· Alcohol (25–30%), gallstones (40–70%), idiopathic, hypertriglyceridemia (TG >1000), hypercalcemia, drugs (thiazides, furosemide, sulfa, ACE-I, protease inhibitors, estrogen, acetaminophen, steroids), obstructive tumors, infection (EBV, CMV, HIV, HAV, HBV, coxsackievirus, mumps, rubella, echovirus), trauma, post-ERCP, ischemic

Presentation
· Epigastric pain radiating through to the back, nausea, vomiting
· Often h/o previous pancreatitis, alcohol abuse, gallstones
· A/w smoking, type 2 diabetes mellitus
· May be ill appearing, tachycardic, epigastric ttp, guarding, ↓ bowel sounds (adynamic ileus)

Evaluation
Increased lipase >3× nl (amylase is not specific)
If severe: ↑ WBC, ↑ BUN (>20 or rising), ↑ HCT (>44% or rising), ↑ creatinine
CT scan: 100% spec but low sens. Not required; should be obtained only to r/o cx (acute fluid collection, pseudocyst, necrosis, abscess), esp after 24–48 h if no improvement
Abd U/S: Used to evaluate for gallstones, CBD dilatation, or pseudocyst
CXR: Pleural effusions & pulmonary infiltrates are a/w severe dz

Treatment
Aggressive IV fluids (LR preferred); NPO initially, but early enteral nutrition if tolerated
IV analgesia, antiemetics
Prophylactic abx have unclear benefit; may use for severe necrotizing pancreatitis
Delayed cholecystectomy for gallstone pancreatitis
IR drainage for persistent or infected fluid collection,

Disposition
Admission for supportive care if severe or not tolerating PO
Atlanta criteria: In mild dz, there is absence of organ failure & local cxs, which are present in severe dz. Organ failure defined as GI bleeding, shock, PaO₂ ≤60%, creatinine ≥2.

LOWER QUADRANT/PELVIC PAIN

Appendicitis
(Lancet. 2015;386:1278)

History
Classically, dull vague periumbilical pain → migrates to RLQ, localizes & becomes sharp
Nausea, vomiting, anorexia, fever
Greatest at 10–30 y of age but can occur at any time

Physical Findings
RLQ (McBurney's point) tenderness, localized rebound, & guarding
Psoas sign: Pain w/ active flexion against resistance or passive extension of the right leg
Obturator sign: Pain w/ internal rotation of the flexed right hip
- Rovsing sign: RLQ pain w/ palpation of the LLQ

**Evaluation**
- Labs: Leukocytosis (not sens or spec); cannot r/o w/ nl WBC. Check hCG.
- US: Less sens than CT but high spec. Consider esp in children young (thin) adults
- Abd CT (92% sens)—secondary signs of appendicitis (eg, fat stranding) less visible in thin pts
- MRI is a useful modality in pregnancy
- Alvarado score uses signs, sx’s & lab values to place pts in low risk (1–4 points), intermediate risk (5–6) & high risk (7–10) groups. High sens/low spec.
- In cases w/ strong clinical e/o appendicitis & low suspicion of alternate etiology, it may be reasonable to proceed to OR w/o imaging

| Alvarado Score for Acute Appendicitis |
|-------------------------------|-----------------|
| RLQ tenderness                    | +2              |
| Elevated temp >99.1               | +1              |
| Rebound tenderness                | +1              |
| Migration of pain to RLQ          | +1              |
| Anorexia                         | +1              |
| Nausea or vomiting                | +1              |
| Leukocytosis >10 K                | +2              |
| Leukocyte left shift              | +1              |

**Management**
- Abx: Cefoxitin, cefotetan, fluoroquinolone/metronidazole, OR piperacillin–tazobactam
- Admission to surgical service. Traditionally surgically removed; treatment w/ abx alone a/w high readmission rate (25–30%) for surgery w/i 1 year

**Pearl**
- Pts at extremes of age are more likely to have atypical presentations & present w/ perforated appendicitis.

**Hernia**
*(NEJM. 2015;372:756)*
**Definition**

- Defect in the abd wall that allows protrusion of abd contents
- Incarcerated hernia: Cannot be reduced
- Strangulated hernia: Incarcerated hernia w/ vascular compromise (ischemia)

**History**

- Bulging mass in abd wall (eg, umbilical, epigastric), inguinal region, or scrotum, or inner thigh (femoral); worse w/ increased intraabdominal pressure
- Inguinal hernias are either direct or indirect; medial or lateral to the inferior epigastric vessels, respectively

**Physical Findings**

- Bulge &/or palpable defect in abd wall or groin
- Strangulated: Tenderness, fever, skin discoloration, or associated peritonitis

**Evaluation**

- If concern for strangulated hernia, consider CBC, lactate, pre-op labs
- CT scan required if concern for strangulated hernia

**Management**

- Attempt reduction w/ generous analgesia/anxiolysis, pt in Trendelenburg
- If easily reduced, d/c w/ analgesic, stool softener, & surgery f/u
- If not reducible or if strangulated, consult surgery for operative intervention

**Pearl**

- Be cautious about reducing a hernia that has been irreducible by the pt for more than 12 h & is difficult to reduce in the ED b/c bowel may be compromised.

**Diverticulitis**

*(BMJ. 2006;332:271)*

**Definition**

- Inflammation of (colonic) diverticulum
- Complicated diverticulitis: Associated perforation, obstruction, abscess, or fistula

**Presentation**
LLQ pain, fever, nausea, change in bowel habits, urinary sxs
Mild LLQ tenderness, 50% of pts have heme-positive stool
Complicated may have peritonitis, septic shock

Evaluation
- Clinical Dx if mild sxs & typical presentation
- Labs: Increased WBC
- CT to confirm dx or if concern for complicated diverticulitis. May see pericolonic stranding, abscess or contained free air if micro perforation

Treatment
- Mild: PO metronidazole + (cipro or TMP-SMX) for 7–10 days
- Severe: NPO, IV fluids, IV ampicillin–sulbactam OR piperacillin–tazobactam OR ceftriaxone/metronidazole OR quinolone/metronidazole OR carbapenem
- Most complicated diverticulitis can be managed medically +/- IR drainage
- Surgery is required if medical therapy fails, large free air is present, or for large abscess that can’t be drained percutaneously. Elective surgery may be recommended for & recurrent dz (≥2 episodes)

Disposition
- If mild, d/c w/ abx, antiemetic, analgesia & PCP or general surgery f/u. If severe, admit.

Pearl
- Consider diverticulitis in older pts w/ urinary sxs but unremarkable or equivocal urine sediment

DIFFUSE PAIN

Abdominal Aortic Aneurysm

Definition
- Dilation of the abd aorta (true aneurysm, involves all layers of the vessel wall).

History
- Older pt w/ low back pain, abd pain, or flank pain (may mimic renal colic), syncope/hypotension
**Physical Findings**
- Pulsatile mass (often not present), early satiety due to duodenal compression
- Ruptured/leaking AAA: Hypotension, abd tenderness, decreased femoral pulses, mottling

**Evaluation**
- Abd CT if hemodynamically stable
- Bedside US may reveal enlarged aorta & free fluid

**Treatment**
- Stable, nonruptured: Surgical or endovascular repair required if >5.5 cm (1%/y risk of rupture if >5 cm) or rapidly growing; usually arranged as outpt
- Ruptured/leaking: Immediate surgical repair, allow permissive hypotension (SBP 90s)

**Disposition**
- Direct to OR/IR if unstable

**Pearls**
- Larger the AAA the greater the risk for rupture
- Rupture into RP can temporarily tamponade, intraperitoneal rupture is rapidly fatal, can also rupture into GI tract (aortoabdominal fistula)
- RFs: Smoking, HTN, hyperlipidemia, CAD, PVD, age ≥65 y, male (5×), FH
- 50% mortality for AAA if ruptured at presentation

**Small Bowel Obstruction**
*(Acad Emerg Med. 2013:20:528)*

**Definition**
- Mechanical obstruction of nl intestinal transit leading to proximal bowel dilation

**History**
- Diffuse, colicky abd pain, nausea/vomiting, abd distension, h/o abd surgeries/prior obstructions/hernia, obstipation (not passing gas)

**Physical Findings**
- Diffuse abd tenderness, distension, high-pitched bowel sounds

**Evaluation**
- Supine & upright abd x-rays (~75% sens): Multiple air–fluid levels, >3
cm small bowel dilation, more than 3 mm small bowel wall thickening
- Bedside US (~90% sens): >2.5-cm dilated loops of bowel, back & forth peristalsis
- Abd CT (~87% sens) can be diagnostic & used to characterize the obstruction (level, severity, cause)

**Treatment**
- NPO, bowel rest, gastric decompression w/ NGT placement
- IV fluids, analgesia, antiemetics
- Surgical consultation—most cases managed conservatively

**Disposition**
- Admission
- Direct to OR if high risk (e.g., closed-loop obstruction, impending perforation, e/o bowel ischemia)

**Large Bowel Obstruction/Volvulus**

**Definition**
- Mechanical obstruction of the large bowel usually caused by cancer (most commonly), volvulus (twisting of the large bowel on itself), intussusception, fecal impaction

**History**
- Insidious onset of diffuse, colicky abd pain, distention, constipation, N/V

**Physical Findings**
- Diffuse abd tenderness, distension, bowel sounds present early

**Evaluation**
- Supine & upright abd x-rays: Dilated large bowel (84% sens), but cannot identify underlying cause
- Abd CT: Can be helpful to distinguish from pseuo-obstruction

**Treatment**
- IV fluids & correction of electrolyte abnormalities
- NGT for proximal decompression
- Surgical consultation for likely operative reduction (particularly for cecal volvulus)

**Disposition**
- Surgical admission

**Pearls**
Sigmoid volvulus most common in ill, debilitated, elderly pts, or pts w/ psychiatric/neurologic disorders
Cecal volvulus common in young adults, classically marathon runners

Perforated Viscus
(Surgical Clin North Am. 2014;94:471)

Definition
- Perforation of hollow viscus leading to abd free air, intraluminal spillage

History
- Acute onset, severe abd pain, worse w/ movement
- May be consequence of bowel obstruction, diverticulitis, cancer, or other primary GI pathology

Physical Findings
- Acute peritonitis: Rigidity, tap tenderness, rebound, hypotension, sepsis

Evaluation
- Supine & upright abd x-rays: May show pneumoperitoneum
- Abd CT: Definitive study but not required for operative management

Treatment
- Immediate surgical consult
- Broad spectrum abx to cover polymicrobial infection (enteric GNR, GPC, anaerobes)

Disposition
- Surgical admission

Pearl
- Findings may be masked in pts who are elderly or chronically immunosuppressed

Mesenteric Ischemia

Definition
- Insufficient perfusion to the intestine
- Etiologies: arterial embolism (40–50%, typically SMA), arterial thrombosis (25–30%, a/w severe atherosclerosis), nonocclusive mesenteric ischemia (20%, low cardiac output state), mesenteric venous thrombosis (10–15%, a/w clotting disorders)

History
- RFs: Age >60, recent MI, AF, vascular dz (coronary, peripheral), CHF (↓
forward flow)
- May have h/o prior abd angina: Postprandial pain, food aversion
- Acute presentation w/ abd pain, anorexia, vomiting, bloody stools

**Physical Findings**
- Ill appearing, pain out of proportion to exam, tachycardia, fever, occult blood in stools. Late signs include peritonitis, shock.

**Evaluation**
- Early surgical eval
- Labs: May see ↑ WBC, ↑ HCT, AG acidosis, ↑ lactate, ↑ amylase, ↑ LDH
- Abd x-ray: NI prior to infarction, “thumbprinting” of the intestinal mucosa later
- Abd CT: Colonic dilation, bowel wall thickening, pneumatosis of the bowel wall
- CT angiography: More sens than CT alone

**Treatment**
- IV fluids
- Broad spectrum abx
- Surgical consultation
- Anticoagulation for venous thrombosis & embolic dz
- IR for thrombolysis or embolectomy
- OR for resection of dead/nonviable gut

**Disposition**
- Surgical admission vs. IR/OR

**Pearl**
- 20–70% morality; improved if dx made prior to infarct

**Colon Ischemia (Ischemic Colitis)** *(Curr Gastroenterol Rep. 2015;17:45)*

**Definition**
- Nonocclusive microvascular dz of the colon, secondary to hypoperfusion & reperfusion injury

**History**
- Crampy abd pain over segment of colon involved (typically left), blood in stool, diarrhea, recent surgery, or illness

**Physical Findings**
- Tenderness over affected colon usually mild, peritoneal findings suggest perforation

**Evaluation**
- Labs: WBC, BUN, creatinine, LDH may be high but all nonspecific
- Abdominal CT: Nonspecific mesenteric fat stranding, bowel wall thickening, abnormal colon wall enhancement

**Treatment**
- Supportive care, bowel rest, hydration, pain management, abx for severe dz

**Spontaneous Bacterial Peritonitis**

**Definition**
- Infection of the ascitic fluid in pts w/ severe chronic liver dz

**History**
- Fever, abd pain, new or worsening ascites, hepatic encephalopathy

**Physical Findings**
- Stigmata of liver failure, diffuse abd pain, ascites

**Evaluation**
- Labs: ↑ Bili, ↓ platelets increase likelihood of dz. Coags, platelets prior to paracentesis
- Paracentesis: >250 PMN, blood:ascites pH gradient >0.1, culture

**Treatment**
- Abx: Cefotaxime 2 g IV OR levofoxacin 750 mg IV. Carbapenem if nosocomial, recent abx or long-term ppx abx.
- Albumin 1.5 g/kg at Dx & 1 g/kg for 3 d shows survival benefit

**Disposition**
- Medical admission

**Pearls**
- Caused by bacteria that translocate from gut. 70% GNR (*E. coli*, *Klebsiella*), 30% GPC (*S. pneumoniae*, *Enterococcus*)
- Occurs in 20% of cirrhotics
- Clinical signs unreliable; have low threshold for paracentesis in admitted pt w/ ascites. Delayed paracentesis >12 h a/w higher mortality. (*Am J Gastroenterology*. 2014;109:1436)
INFLAMMATORY BOWEL DISEASE (ULCERATIVE COLITIS AND CROHN'S DISEASE)

(Lancet. 2007;369:1641)

Definition

- Ulcerative colitis (UC): Inflammation of the colonic mucosa
- Crohn's dz (CD): Transmural inflammation of the GI tract

<table>
<thead>
<tr>
<th>Inflammatory Bowel Disorder (Ulcerative Colitis and Crohn's Disease)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ulcerative Colitis</strong></td>
</tr>
<tr>
<td><strong>Clinical features</strong></td>
</tr>
<tr>
<td>Fever, bloody diarrhea, tenesmus, urgency, painful BMs</td>
</tr>
<tr>
<td><strong>GI involvement</strong></td>
</tr>
<tr>
<td>Exclusively colon (mostly rectal), continuous lesions limited to submucosa, friable mucosa; irregular, shallow ulcers; pseudopolyps; crypt abscesses; loss of haustral markings</td>
</tr>
<tr>
<td><strong>GI cx</strong></td>
</tr>
<tr>
<td>Toxic megacolon (&gt;8 cm, usually transverse colon), colon cancer</td>
</tr>
</tbody>
</table>

History

- Women > men typically presents in 2nd or 3rd decade, weight loss, vomiting, abd pain/diarrhea (grossly bloody in UC) that flares w/ emotional stress, infections, withdrawal from steroids

Physical Findings

- Diffuse abd tenderness (focal RLQ tenderness in CD), heme-positive stools 20% of pts have extraintestinal sxs, perianal dz (seen in CD); fissures, fistulas, abscess

<table>
<thead>
<tr>
<th>Common Extraintestinal Features</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Arthritic</strong></td>
</tr>
<tr>
<td><strong>Intraabdominal</strong></td>
</tr>
<tr>
<td><strong>Dermatologic</strong></td>
</tr>
<tr>
<td><strong>Ophthalmic</strong></td>
</tr>
</tbody>
</table>

Evaluation

• Labs: Low HCT (from chronic blood loss), increased WBC, hypokalemia (from diarrhea)
• Plain abd x-ray: If perforation, obstruction, or toxic megacolon suspected
• Abd CT: May r/o cx (eg, abscess, obstruction, fistula)
• Outpt colonoscopy: If Dx not known & once acute flare resolved

Treatment
• IV fluids, bowel rest, surgical consult, steroids, ± 5 ASA agents (mesalamine, sulfasalazine)

Disposition
• Admit for severe dz or acute cx

NAUSEA AND VOMITING

(Emeg Med Clin North Am. 2011;29:211)

Approach
• Common sxs of many dz processes (eg, intra-abd dz, metabolic derangements, toxic ingestions, neurologic dz)
• Careful attention to ROS, PMH, previous abd surgeries
• Labs: Consider CBC, BMP, UA, LFTs, lipase, hCG
• Treat underlying cause: Antiemetics (eg, ondansetron, promethazine), IVF if not taking PO

<table>
<thead>
<tr>
<th>Nausea &amp; Vomiting Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Abdominal/GU</strong></td>
</tr>
<tr>
<td>Obstruction (gastric outlet, small bowel, large bowel)</td>
</tr>
<tr>
<td>Infections (appendicitis, cholecystitis, pyelonephritis)</td>
</tr>
<tr>
<td>Gastroenteritis, food poisoning</td>
</tr>
<tr>
<td>Gastritis/ulcers</td>
</tr>
</tbody>
</table>
**Gastroenteritis**

- **Definition:** Irritation of the GI tract causing vomiting & diarrhea usually caused by infections (viruses, bacteria, bacterial toxins, parasites) or due to medications or diet
- **History:** Vomiting & diarrhea, crampy abd pain, ±fever
- **Physical Findings:** Nl exam or mild diffuse abd ttp, tachycardia, dehydration
- **Evaluation:** Consider BMP if clinical concern for significant electrolyte derangement. Stool culture if systemically ill, fever, recent abx, exposure to treatable pathogen.
- **Management:** Supportive care, antiemetics. IVF if not taking PO. Home when tolerating PO. Abx & antimotility agents generally not indicated
- **Pearl:** Viral & bacterial toxins (food poisoning) are most common, typically resolve w/o tx in 48H

### GASTROINTESTINAL BLEED

(Emeg Med Clinics North Am. 2016;34:309)

**Approach**

- Hemodynamically unstable pts should get 2 large-bore IVs (14–18 gauge), early transfusion of PRBC as well as FFP & Vit K if impaired coagulation
- ROS, PMH, previous GIB, alcohol use, liver dz
- Labs: CBC, BMP, LFTs, lipase, coagulation studies, lactate, type & screen. BUN/Cr ratio >30 indicates upper GI source

<table>
<thead>
<tr>
<th>GI Bleed Differential Location</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>UGB (bleeding proximal to the ligament of Treitz)</td>
<td>PUD, gastritis, variceal bleed (esophageal &amp; gastric), Mallory–Weiss tear, aortoenteric fistula, gastric cancer</td>
</tr>
</tbody>
</table>
**UPPER GI BLEED**

**Approach**
- Glasgow–Blatchford score was designed to predict need for transfusion or urgent endoscopy. A score of zero identifies low-risk pts who can safely be discharged w/ outpt f/u (*JAMA* 2012;307:1072; *Lancet* 2000;356:1318).

<table>
<thead>
<tr>
<th>Criteria for Glasgow–Blatchford Score of 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
</tr>
<tr>
<td>Systolic BP</td>
</tr>
<tr>
<td>Heart rate</td>
</tr>
<tr>
<td>BUN</td>
</tr>
<tr>
<td>No melena, syncope, heart failure, or liver dz</td>
</tr>
</tbody>
</table>

**Bleeding Peptic Ulcer Disease (PUD) or Gastritis**
(*NEJM*. 2016;374:2367)

**Definition**
- Inflammation or ulceration of the stomach or duodenal lining caused primarily by *H. pylori* infection, NSAIDs, (15–30%), alcohol

**History**
- Bloody or coffee ground emesis; dark, tarry stool

**Physical Findings**
- Epigastric tenderness, melena or heme-positive stool

**Evaluation**
- Labs: CBC, LFTs, coagulation panel, elevated BUN; *H. pylori* serology (90% sens)
- NG tube not routinely indicated

**Treatment**
- IVF resuscitation, PRBC if Hgb <7 or hypotensive, IV proton pump inhibitor ↓ need for endoscopic therapy but does not ↓ bleeding or mortality
Emergent EGD if hemodynamically unstable

Disposition
- If ongoing bleeding, Blatchford >0, high risk: Admit for EGD

Variceal Bleeds
(Hepatology. 2007;46:922)

History
- Bright red hematemesis, diffuse abd pain, nausea, h/o portal hypertension

Physical Findings
- Stigmata of liver failure (jaundice, spider angiomas, ascites, caput medusae), Ill-appearing hypotension, tachycardia, melena

Evaluation
- Labs: CBC, LFTs, coagulation panel, type & cross

Treatment
- Place 2 large bore IVs, initiate IV fluid resuscitation, PRBC if Hgb <7 or active bleeding
- Octreotide bolus & drip; IV PPI
- Antibiotic prophylaxis (ceftriaxone or levofloxacin) increases survival
- Emergent EGD if hemodynamically unstable, may need emergent TIPS if still bleeding
- Balloon tamponade w/ Minnesota or Blakemore tube if exsanguinating (after intubation)

Disposition
- Usually ICU admission, pts can decompensate quickly

Mallory–Weiss Tear

Definition
- Tears in the mucosal membrane of the distal esophagus caused by vomiting. A/w heavy alcohol use.

History
- Specks of bright red blood in emesis or mild hematemesis after forceful retching

Physical Findings
- Most have no physical findings, mild tachycardia

Evaluation
- Upright CXR if hemodynamically unstable to evaluate for subcutaneous or mediastinal air for Boerhaave syndrome (complete esophageal rupture)

**Treatment**
- Antiemetics, PO challenge

**Disposition**
- D/c w/ outpt EGD

**Pearl**
- Boerhaave syndrome can result from emesis but usually pts are ill-appearing w/ shock & require surgical management. Consider water-soluble swallow study if high suspicion.

**Aortoenteric Fistula**

**Definition**
- Fistula b/w the aorta & GI tract, most commonly in duodenum

**History**
- H/o AAA, aortic graft (usually >5 y), may have sentinel bleed or large-volume GIB

**Physical Findings**
- Rapid GIB, hemodynamic collapse

**Evaluation**
- CBC, type & cross, emergent surgical consult, CT scan if stable

**Treatment**
- IV fluid resuscitation, PRBC if indicated
- Surgical repair

**Disposition**
- Surgical ICU admission

**Pearl**
- Mortality directly related to time to the OR

---

**LOWER GI BLEED**

(*Crit Care Clin.* 2016;32:241)
Diverticular Bleeding

- **History:** Painless bright red rectal bleeding often initiated by urge to defecate
- **Physical Findings:** NL abd exam, BRBPR, no etiology found on rectal exam
- **Evaluation:** Labs: CBC, LFTs, coagulation panel, type & cross
- **Treatment:** Usually self-limited. IV fluid resuscitation, PRBC if indicated
- **Disposition:** Admit for colonoscopy

Colorectal Cancer

**History**
- Chronic blood in stool, change in bowel habits, anorexia, weight loss, light-headedness

**Physical Findings**
- Pale, heme occult positive stools

**Evaluation**
- Labs: CBC, LFTs, coagubs; CT if concern for obstruction or significant bleeding

**Treatment**
- IV fluid resuscitation, PRBC if indicated
- Surgical consultation if significant bleeding (rare)

**Disposition**
- If stable, d/c for outpt colonoscopy/oncology w/u

Colonic Angiodysplasia

- **Definition:** Enlarged, fragile blood vessels, usually in cecum or proximal ascending colon (10–20% of LGIB)
- **History:** >60 y/o, small frequent bleeds. Usually coagulopathy or NSAID use precipitates bleed.
- **Physical Findings:** nl abd exam, BRBPR, or heme occult positive stools
- **Evaluation:** CBC, coagulation panel
- **Treatment:** IV fluid resuscitation, PRBC if indicated; endoscopic cautery or IR embolization
- **Disposition:** Admit for observation & colonoscopy
DIFFICULTY SWALLOWING

(\textit{Nat Rev Gastroenterol Hepatol.} 2015;12:259)

\textbf{Definition}

\begin{itemize}
  \item Dysphagia is difficulty swallowing, odynophagia is pain w/ swallowing
\end{itemize}

\textbf{Approach}

\begin{itemize}
  \item Nature: Time course, sudden or progressive, localization (oropharyngeal vs. esophageal)
  \item ROS, PMH, hx, or FH of GI disorders or neurologic disorders
  \item Labs: CBC, BMP
  \item Studies: Barium swallow or EGD for structural/mechanical lesions; motility studies
\end{itemize}

\begin{table}[h]
\centering
\begin{tabular}{|l|l|}
\hline
\textbf{Dysphagia} & \textbf{Differential} \\
\hline
Sols (mechanical obstruction) & Esophageal ring (intermittent), eosinophilic esophagitis (intermittent), esophageal cancer (progressive), oral/pharyngeal abscess (4d), neck cancer \\
\hline
Sols & liquids (motility disorder) & Spasm (intermittent), scleroderma (progressive), achalasia (progressive), neurologic (eg, myasthenia, ALS) \\
\hline
Odynophagia & Reflux esophagitis, infection (candida, herpes), radiation, chemotherapy \\
\hline
\end{tabular}
\end{table}

\textbf{Esophageal Food Impaction/Foreign Bodies}


\textbf{Definition}

\begin{itemize}
  \item Food or FB stuck in esophagus (70% lodge at the lower esophageal sphincter)
\end{itemize}

\textbf{History}

\begin{itemize}
  \item Sensation of food (often meat) or FB stuck in the esophagus, retching, unable to swallow secretions. A/w esophageal stricture, esophageal ring, or eosinophilic esophagitis
\end{itemize}

\textbf{Physical Findings}

\begin{itemize}
  \item Odynophagia, neck or chest pain, respiratory distress, drooling, retching
\end{itemize}
Evaluation
- CXR (may show dilated esophagus w/ air–fluid level or FB)

Treatment
- Airway management
- Historically glucagon given however no data to support its use.
  Effervescents, benzos are also often used.
- Endoscopy if a dangerous object is present (batteries, sharp object), or FB doesn’t pass w/i 12–24 h

Disposition
- If tolerating PO, d/c w/ outpt EGD

---

**DIARRHEA**

*(Emerg Med Clin North Am. 2011;29:211)*

**Definition**
- Frequent, watery stools. Specifically, >3 loose stools/d OR >200 g stool/d.
- Acute ≤14 d, persistent 14–30 d, chronic >30 d

**Approach**
- Nature: Bloody, mucus present, duration, frequency, volume; recent travel or abx
- Labs: Consider BMP for electrolyte derangement; consider CBC, LFTs, heme occult

<table>
<thead>
<tr>
<th>Diarrhea Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes</strong></td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td>Medications</td>
</tr>
<tr>
<td>-------------</td>
</tr>
</tbody>
</table>
| - ↑ secretion  
- ↑ motility  
- ↑ cell turnover |

<table>
<thead>
<tr>
<th>Inflammatory</th>
<th>IBD, radiation enteritis, ischemic colitis, diverticulitis</th>
</tr>
</thead>
</table>
| - Fever  
- Hematochezia  
- Abd pain |

<table>
<thead>
<tr>
<th>Malabsorption</th>
<th>Bile salt deficiency (cirrhosis, cholestasis, ileal dz, bacterial overgrowth), pancreatic insufficiency, mucosal abnormalities (celiac sprue, tropical sprue, Whipple dz), lactose intolerance</th>
</tr>
</thead>
</table>
| - Chronic  
- ↓ sx w/ fasting  
- ↑ osmotic gap  
- ↑ fecal fat  
- Vitamin deficient |

<table>
<thead>
<tr>
<th>Secretory</th>
<th>Hormonal (VIP, carcinoid tumor, medullary cancer of the thyroid, Zollinger–Ellison, glucagon, thyroxine), laxative abuse, neoplasm</th>
</tr>
</thead>
</table>
| - nl osmotic gap- ↓ sx w/ fasting  
- Nocturnal sx |

<table>
<thead>
<tr>
<th>Motility</th>
<th>IBS, scleroderma, hyperthyroidism, diabetic autonomic neuropathy</th>
</tr>
</thead>
</table>
- Abx: TMP-SMX, ciprofloxacin or azithromycin (recent travel, ill appearing, fever, immunocompromised), OR metronidazole (C. difficile, Giardia, E. histolytica)
- Antimotility agents may be used for traveler’s diarrhea
- Constipating diet (BRAT: Bananas, rice, applesauce, toast) for a short time

**Disposition**
- Admit if unable to keep up w/ volume loss or toxic

**Pearl**
- Significant abd pain in not common & should be evaluated further

<table>
<thead>
<tr>
<th><strong>Diarrhea Epidemiology</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathogen</strong></td>
</tr>
<tr>
<td>Norovirus</td>
</tr>
<tr>
<td><em>Campylobacter</em></td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td><em>Giardia</em></td>
</tr>
<tr>
<td>Enterotoxigenic <em>Escherichia coli</em></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Diarrhea Pathogen Characteristics</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathogen</strong></td>
</tr>
<tr>
<td><em>Campylobacter</em></td>
</tr>
<tr>
<td><em>Salmonella</em></td>
</tr>
<tr>
<td><em>Shigella</em></td>
</tr>
<tr>
<td><em>Yersinia</em></td>
</tr>
<tr>
<td>Pathogen</td>
</tr>
<tr>
<td>----------------------------------------------</td>
</tr>
<tr>
<td>Preformed toxin-mediated (Staphylococcus, Bacillus)</td>
</tr>
<tr>
<td>Enterotoxic Escherichia coli</td>
</tr>
<tr>
<td>Enterohemorrhagic Escherichia coli</td>
</tr>
<tr>
<td>Clostridium perfringens</td>
</tr>
<tr>
<td>Clostridium difficile</td>
</tr>
<tr>
<td>Vibrio parahaemolyticus</td>
</tr>
<tr>
<td>Giardia</td>
</tr>
<tr>
<td>Entamoeba</td>
</tr>
</tbody>
</table>

**Irritable Bowel Syndrome**  
*BMJ. 2015;350:h1622*

**Definition:** Disorder of the colon: Causes cramping, bloating, diarrhea, constipation (F > M)

**History:** Recurrent abd pain >3 d/mo over the last 3 mo. Plus 2 or more of the following: Improvement w/ defecation, onset w/ change in frequency of stools, onset w/ change in form of stools. No constitutional sxs.

**Physical Findings:** May have mild lower abd tenderness, heme-negative stools

**Treatment:** Fiber for constipation, antimitility for diarrhea, antispasmodics (Bentyl) for pain

**Disposition:** D/c, outpt management
**Pearl:** Dx of exclusion. Unlikely if age of onset >35 or associated constitutional sxs.

**CONSTIPATION**

*(JAMA. 2016;315:185)*

**Definition**
- Reduced frequency of stool (<3/wk), &/or difficult passage of hard stool

**Approach**
- Nature: Duration, severity, character of stool, pain, fever, medication use, prior episodes

<table>
<thead>
<tr>
<th>Constipation Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
</tr>
<tr>
<td>Functional</td>
</tr>
<tr>
<td>Obstruction</td>
</tr>
<tr>
<td>Medication</td>
</tr>
<tr>
<td>Neurologic</td>
</tr>
<tr>
<td>Metabolic</td>
</tr>
</tbody>
</table>

**Simple Constipation (Including Stool Impaction)**

**History**
- Poor diet, decreased fluid/fiber intake, decreased mobility, constipating medications

**Physical Findings**
- Firm stool in the rectal vault, palpable stool on abd exam, minimal abd ttp

**Evaluation**
- Abd x-ray or CT if need to r/o obstruction, or to confirm dx in high-risk pt

**Treatment**
- Manual disimpaction if needed
- Colace, magnesium citrate, enema (esp in elderly), bisacodyl (oral or
suppository)
- Natural bulking agents (Metamucil) when constipation resolves

**Disposition**
- Home

**Rectal Foreign Body**
*(Surgical Clin of North Am. 2010;90:173)*

**Physical Findings**
- FB in rectum on exam or anoscopy, peritonitis if perforation

**Evaluation**
- Abd x-ray to eval location/shape & presence of pneumoperitoneum

**Treatment**
- Removal w/ forceps traction while the pt bears down. Impacted object may cause proximal vacuum suction; can pass foley around object to break vacuum seal & use balloon to pull back on object.
- Removal in OR if unsuccessful or if sharp object w/ risk of perforation

**Disposition**
- Home if removed

**Pearl**
- Procedural sedation may be needed to sufficiently dilate anus to remove FB in ED

---

**JAUNDICE**

*(Prim Care. 2011;38:469)*

**Definition**
- Yellowning of the skin as a result of elevated bilirubin (&gt;3 mg/dL)

**Approach**
- Duration, associated pain, fever, recent travel, h/o liver dz or alcohol abuse
- Labs: CBC, BMP, UA, LFTs, lipase, ±ammonia if MS changes, paracentesis if ascites

<table>
<thead>
<tr>
<th>Jaundice Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperbilirubinemia</td>
</tr>
</tbody>
</table>
### Prehepatic:
Increased bilirubin production or impaired conjugation

- Unconjugated (indirect)
  - Hemolysis, hematoma resorption, prolonged fasting, Crigler–Najjar syndrome, Gilbert syndrome

### Hepatocellular

- Mixed, mostly conjugated
  - Infectious hepatitis, hepatotoxins, autoimmune, alcoholic (AST:ALT >2:1), drugs (eg, tylenol, amiodarone, statins), metabolic disorders (Wilson, Reye), hemochromatosis, α₁-antitrypsin deficiency, ischemic (“shock liver,” AST/ALT >1000 + ↑ LDH), nonalcoholic fatty liver dz

### Intrahepatic (nonobstructive):
Impaired excretion of conjugated bilirubin

- Conjugated (direct)
  - Cholestatic jaundice of pregnancy, Dubin–Johnson syndrome, rotor syndrome, primary biliary cirrhosis, sarcoidosis, graft-versus-host dz

### Extrahepatic (obstructive):
Impaired excretion of conjugated bilirubin

- Conjugated (direct)
  - Cholecystitis, choledocholithiasis, cholangitis, pancreatitis, carcinoma (ampulla, gallbladder, pancreas, CBD), biliary stricture (postsurgical), sclerosing cholangitis

### Viral Hepatitis

<table>
<thead>
<tr>
<th>Dz</th>
<th>Transmission</th>
<th>Serologic Pattern</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis A</td>
<td>Fecal–oral, contaminated food/water</td>
<td>Acute: IgM anti-HAV Prior: IgG anti-HAV</td>
<td>Incubation 2–6 wk, self-limiting, tx is supportive</td>
</tr>
<tr>
<td>Acute Hepatitis B&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Blood, sex, perianal</td>
<td>IgM anti-HBc: Acute HBeAg: Active infection HBsAg: May appear before sxs</td>
<td>Incubation 1–6 mo, 70% acute infections subclinical, 30% jaundice, 1% fulminant failure, acute tx is supportive, &lt;10% persist to chronic</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hepatitis B</td>
</tr>
<tr>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Chronic Hepatitis B&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Blood, sex, perianal</td>
<td>IgG anti-HBc</td>
<td>Major cause of hepatocellular cancer (10–390 × increased risk), tx: INF-α-2b, PEG INF-α-2b, lamivudine, adefovir, telbivudine, entecavir</td>
</tr>
<tr>
<td>Acute Hepatitis C</td>
<td>Blood, sex</td>
<td>HCV viral load</td>
<td>Incubation 2 wk–5 mo, 75% acute infections subclinical, 25% jaundice, 50–80% persist to chronic</td>
</tr>
<tr>
<td>Chronic Hepatitis C</td>
<td>Blood, sex</td>
<td>HCV &amp; anti-HCV</td>
<td>Major cause of cirrhosis (20–30%), 2–3% of cirrhotics develop HCC, tx: PEG INF-α-2b + ribavirin</td>
</tr>
<tr>
<td>Hepatitis D</td>
<td>Blood, sex</td>
<td>Anti-HDV</td>
<td>Exists only in association w/ hepatitis B, faster progression to cirrhosis</td>
</tr>
<tr>
<td>Hepatitis E</td>
<td>Fecal–oral (travel)</td>
<td>IgM anti-HEV</td>
<td>Self-limiting, mortality 10–20% in pregnancy</td>
</tr>
</tbody>
</table>


**Cirrhosis**
*(Lancet. 2014;383:1749)*

**Definition**
- Fibrosis & nodular regeneration resulting from hepatocellular injury
- Major etiologies include viral hepatitis (esp HCV), alcoholism, nonalcoholic steatohepatitis

**History**
- Abd pain, jaundice, pruritus, abd distension
Physical Findings

- Liver: Enlarged palpable liver or shrunken nodular
- Signs of liver failure: Jaundice, spider angioma, palmar erythema, gynecomastia, asterixis, encephalopathy
- Signs of portal HTN: Splenomegaly, ascites, caput medusae

Evaluation

- New onset: LFTs, BMP, CBC (for anemia, thrombocytopenia), INR (to evaluate synthetic function), abd US if pain, tenderness, or fever present to r/o acute biliary dz or if concern for Budd–Chiari, paracentesis if new-onset ascites
- Exacerbation/decompensation of known cirrhosis: CBC, BMP, INR, ammonia. Paracentesis to r/o SBP if fever, abd pain, new hepatic encephalopathy, GIB, significant leukocytosis.

Treatment

- Directed at treating cultures
- Hepatic encephalopathy (failure of liver to detoxify ammonia & other agents): Protein restriction, lactulose (goal 2–4 stools/d)

Disposition

- Admit if decompensated (increasing ascites/edema despite compliance w/ outpt regimen), pulmonary edema, renal failure, hypotensive, encephalopathic, febrile

Pearl

- Cxs: Portal HTN (ascites, varices), encephalopathy, hepatorenal syndrome, hepatopulmonary syndrome, infections (relative immunosuppression), HCC

Acute Liver Failure

(NEJM. 2013;369:2525)

Definition

- Acute hepatic dz often w/ coagulopathy & encephalopathy
- Fulminant liver failure is when encephalopathy occurs <8 wk since onset of 1st sx
- Etiologies: Viral hepatitis (A, B, E), drugs (acetaminophen), acute ischemic injury in critically ill pts, neoplastic infiltration, acute Budd–Chiari, mushroom ingestion, Wilson’s dz

History

- Abd pain, jaundice, toxic ingestion, nausea, vomiting, malaise,
confusion

Physical Findings

- Jaundice, abd tenderness, enlarged liver, encephalopathy, pulmonary edema, GIB (decreased clotting factors, DIC)

Evaluation

- Labs: CBC (anemia, thrombocytopenia), PT/INR, BMP (electrolytes, renal function), acetaminophen level, viral serologies

Treatment

- Treat underlying causes (eg, acetaminophen w/ NAC)
- If etiology unclear have low threshold for NAC regardless of acetaminophen level
- Abx: Broad-spectrum (Vancomycin + 3rd-generation cephalosporin)
- Coagulopathy/GIB: Vit K, FFP, platelets, cryoprecipitate if active hemorrhage
- Cerebral edema: Consider ICP monitoring, hypertonic saline/mannitol, avoid fever
- Transplantation improves survival but not universally available

Disposition

- Admit medicine. ICU if fulminant, hypotensive, or otherwise unstable.

RECTAL PAIN (PROCTALGIA)

(Medical Clin of North Am. 2014;98:609)

Approach

- Nature: Duration, consistency of stools, bleeding, fevers

<table>
<thead>
<tr>
<th>Proctalgia Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bleeding</strong></td>
</tr>
<tr>
<td><strong>No bleeding</strong></td>
</tr>
</tbody>
</table>

Anal Fissure

- **Definition:** Superficial tear of the anoderm that begins just below the dentate line
History: H/o passage of hard stools, sharp pain w/ defecation, blood on toilet paper
Physical Findings: Visible fissure, painful. If not midline, eval for cancer, HIV, IBD, STDs.
Management: Sitz baths (warm baths 15 min 3×/d), high-fiber diet, lidocaine jelly, topical nitroglycerin ointment, topical diltiazem gel

Hemorrhoids
Definition
- Dilated or bulging veins of the rectum & anus. Internal hemorrhoids may prolapse & become incarcerated (irreducible) or strangulated (ischemic).
History
- Bright red-coated stool/toilet paper/dripping into the bowl, pain w/ defecation, h/o hard stools, constipation, prolonged sitting

Physical Findings
- External hemorrhoids are visible on eversion of the anal orifice, internal hemorrhoids may be palpable & are only visible w/ anoscopy

Evaluation
- CBC only if significant blood loss suspected or concerning underlying condition

Management
- Outpt w/ stool softener (Colace, Senna), Sitz baths (15 min TID & after BMs), suppositories for symptomatic relief
- Acute thrombosis (<48 h since onset of pain) can be excised at bedside in ED
- If prolapsed hemorrhoid is incarcerated w/ signs of strangulation, consult surgery

Pearl
- Hemorrhoidal bleeding rarely a cause of significant anemia
FEVER

Background
- Temp >100.4°F/38°C
- Caused by response to bacteria, viruses, inflammation; ↑ metabolic rate, meds
- Distinct from hyperthermia (caused by exogenous factors)

Approach
- Careful hx: COLDER, associated sx (N/V, diarrhea, cough, abd pain, rash, AMS)
- Eval directed by pt hx & sx localization
- Assess VS for significant abnormalities that may indicate serious infection (↓ BP, ↑ HR)
- If immunosuppressed (HIV/AIDS, elderly, malnourished, chronic steroids, DM) or neutropenic, more intensive eval & testing: CBC, Chem, UA & cx, CXR; consider blood cx & admission
- Intermittent/relapsing fever, FUO, or occurring after foreign travel: Consider travel-related infectious etiologies, endocarditis

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Endocarditis, myocarditis (1j)</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Pneumonia (2b), bronchitis (2b), empyema, TB (2b), PE</td>
</tr>
<tr>
<td>GI</td>
<td>Intra-abd abscess, cholangitis (3a), diverticulitis (3a), appendicitis (3a), hepatitis (3g), cholecystitis</td>
</tr>
<tr>
<td>GU</td>
<td>UTI (6a), pyelonephritis (6b), PID (7e)</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Meningitis (5d), subarachnoid hemorrhage, TBI, dysautonomia</td>
</tr>
<tr>
<td>ENT</td>
<td>Pharyngitis (13b), sinusitis (13), otitis</td>
</tr>
<tr>
<td>Toxicology</td>
<td>Neuroleptic malignant syndrome (10l), malignant hyperthermia (10l)</td>
</tr>
<tr>
<td>Environmental</td>
<td>Hyperthermia (10k), drug-induced, vector-borne &amp; zoonotic</td>
</tr>
</tbody>
</table>
diseases (4h), parasitic infections (4l), Rocky Mountain spotted fever (8a)

<table>
<thead>
<tr>
<th>Infectious</th>
<th>Mononucleosis (4f), TB (2b), HIV (4g), rheumatic fever, viral infections (4f)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematologic</td>
<td>DVT (1b), PE (1b), sickle cell (11e)</td>
</tr>
<tr>
<td>Orthopedic</td>
<td>Osteomyelitis (19k), septic arthritis (12c)</td>
</tr>
<tr>
<td>Oncologic</td>
<td>Malignancy (11), neutropenic fever, tumor lysis syndrome</td>
</tr>
<tr>
<td>Immunologic</td>
<td>Autoimmune, Mediterranean fever, vasculitis, sarcoid</td>
</tr>
</tbody>
</table>

**ENDOCARDITIS**

*(NEJM. 2013;369:785)*

**History**
- RFs: IVDU, congenital or acquired valvular dz, prosthetic valves, structural heart dz, HD, indwelling venous catheters, cardiac surgery, bacteremia, HIV, previous endocarditis
- Dx difficult 2/2 nonspecific sx (lethargy, weak, anorexia, low-grade temp), or negative w/u

**Findings**
- Fever (80%), new murmur (48%), CHF, splenomegaly (11%), petechiae
- Classic physical exam findings
  - Roth spots (2%): Exudative, edematous retinal lesions w/ central clearing
  - Osler nodes (3%): Violaceous tender nodules on toes & fingers
  - Janeway lesions (5%): Nontender, blanching, macular plaques on soles & palms
  - Splinter hemorrhages (8%): Nonblanching, linear, reddish-brown under nails
  - Septic emboli (mitral valve vegetations)

**Diagnosis**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Requirements for Dx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite</td>
<td>Microorganism on culture or histology of vegetation/cardiac abscess OR</td>
</tr>
</tbody>
</table>
Clinical criteria: 2 major, 1 major & 3 minor, or 5 minor

<table>
<thead>
<tr>
<th>Possible</th>
<th>Criteria</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 major &amp; 1 minor, or 3 minor</td>
<td>Major</td>
<td>≥2 positive blood cultures, endocardial involvement, vegetation, new valvular regurgitation</td>
</tr>
<tr>
<td></td>
<td>Minor</td>
<td>Predisposing cardiac dz, IVDU, or other RFs, fever, vascular phenomena (septic infarcts, ICH, Janeway lesions), immune phenomena (glomerulonephritis, Osler’s nodes, Roth’s spots, RF), positive blood culture not meeting major criteria</td>
</tr>
</tbody>
</table>

**Evaluation**
- EKG, CBC, Chem, coags; CXR, ↑ ESR/CRP (nonspecific), at least 3 sets blood cx
- Typically *Staph aureus* or *Strep* species, also *Enterococcus, Candida* (prosthetic). Up to 10% never have organism identified
- Echo for vegetations or valve ring abscesses; TEE more sens than TTE

**Treatment**
- Hemodynamic stabilization if valve rupture, can present w/ acute pulm edema cultures
- Immediate abx in suspected cases, preferably after blood cultures (see table)

**Disposition**
- Admit w/ continuous telemetry & IV abx, ICU if hemodynamic compromise

**Pearls**
- Infection of endothelium of heart (including but not limited to valves)
- Consider cardiac surgery consultation for heart failure, uncontrolled infection or prevention of embolic events
- Mortality w/ native valve dz: ~25%; prosthetic valve higher
  - Worse prognosis if involves aortic valve, DM, *S. aureus* (30–40%)
  - Left-sided endocarditis (mitral 41%, aortic valve 31%) most common
  - IVDU: Tricuspid valve endocarditis; rheumatic valve dz: Mitral, then aortic valve

**Antimicrobial Treatment of Bacterial Endocarditis**

<table>
<thead>
<tr>
<th>Hx</th>
<th>Antibiotic</th>
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<tr>
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<tr>
<td>Native valve</td>
<td>Ampicillin-sulbactam 3 g IV q6h or Amoxicillin-clavulanate 3 g IV q6h + gentamicin 1 mg/kg IV q8h Vancomycin 15 mg/kg IV BID + gentamicin 1 mg/kg IV q8h + ciprofloxacin 400 mg IV BID (for patients allergic to beta-lactams)</td>
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<tr>
<td>Prosthetic valve (&lt;12 mo post-op)</td>
<td>Vancomycin 15 mg/kg IV BID + gentamicin 1 mg/kg IV q8h + rifampin 600 mg PO BID</td>
</tr>
<tr>
<td>Prosthetic valve (≥12 mo post-op)</td>
<td>Same as native valve</td>
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**ABSCESS**

**Approach**

- ↓ activity of infiltrated local anesthetic agents b/c of the low pH of abscess area; consider regional nerve or field blocks + IV procedural sedation/analgesia
- Gram stain & wound cx rarely necessary for skin or perirectal abscesses
- Cx from intra-abd, spinal, or epidural abscesses usually sent from OR to guide therapy
- Pharyngeal abscess cx can also help tailor antibiotic therapy
- In diabetic, immunocompromised, w/ systemic sx, septic, obtain labs & blood cultures, start IVF & abx & admit for IV abx

**Cutaneous Abscess**

*(NEJM. 2014;370:1039)*

**History**

- ↑ pain, tenderness & induration, usually w/o h/o fever or systemic tox
- Disruption of skin from trauma or penetrating injury, often pt cannot recall injury
- H/o IVDA/skin popping, prior MRSA abscesses

**Findings**

- Exquisitely tender, soft, fluctuant mass surrounded by erythema
- Most commonly *Staph* species, often polymicrobial
Evaluation
- Blood work rarely needed unless appear systemically ill; US may help w/ localization
- Culture from abscess only if tx w/ abx, severe infection, systemic illnesses, failed initial tx

Treatment
- Traditionally no abx indicated in healthy hosts unless cellulitis, systemic illness, immunosuppression, failed I&D. However, RCT of 1247 pts showed higher cure rate (80.5% v 73.6%) as well as lower rates of subsequent I&Ds, skin infections at new sites & infections in household members. (NEJM. 2016;374:823)
- I&D w/ regional nerve or field block ± procedural sedation
  - Create elliptical incision to prevent premature wound closure, deep enough to drain cavity. Follow tension lines to minimize scarring.
  - Break up loculations in abscess cavity w/ hemostat
  - Consider packing w/ 1/4-in gauze × 48 h (24 h if cosmetically important) for large abscesses
- Tx cellulitis if indicated (see Cellulitis section below)

Disposition
- D/c w/ wound care instructions, 2-d f/u
- Warm soaks TID × 2–3 d after removal of packing to allow continued wound drainage

Pearls
- Can develop essentially anywhere: Furuncle, acne, skin breakdown, insect bites
- Routine packing of abscesses after I&D is controversial

Paronychia
(J Hand Surgery. 2012;37:1068)

History
- Pain & swelling lateral to nail edge; abscess beneath eponychial fold
- Usually secondary to contaminated nail care instruments, hang nail, or trauma

Findings
- Purulent collection lateral to nail bed w/ minimal surrounding erythema
- Most commonly S. aureus, S. pyogenes, Pseudomonas or Proteus.

Evaluation
- No labs necessary

**Treatment**
- Oral antibiotics (cephalexin, clindamycin, amoxicillin + clavulanate) may be used
- Digital block w/ 1% lidocaine with or without epinephrine in each web space of affected digit
- #11 blade scalpel to lift cuticle from nail on affected side & express purulent material

**Disposition**
- D/c w/ wound care instructions, 2 d f/u
- Warm soaks to finger TID × 2–3 d to allow complete drainage

**Pearls**
- Often h/o manicure/pedicure, nail biting
- If recurrent or chronic paronychia, consider *Candida* infection
- May spread to pulp space of finger (felon) or deep spaces of hand, tendon if neglected

**Pilonidal Cyst**
*Emerg Med Clin North Am. 2016;34:251*

**History**
- Painful, tender abscess in midline pit between upper part of the gluteal clefts, often in obese or hirsute individuals
- More prevalent in males; fever & systemic tox very rare

**Findings**
- Painful, localized abscess in natal cleavage/midline sacrococcygeal region, 4–5 cm posterior to anal opening; surrounding erythema & fluctuance
- Mixed flora: *Staph* or *Strep* species, anaerobic cocci, mixed aerobic & anaerobic flora

**Evaluation**
- No labs necessary unless systemically ill

**Treatment**
- Same as for cutaneous abscess, I&D
- Antibiotics if overlying cellulitis, immunosuppressed or systemically ill
- Surgical referral for excision of follicle & sinus tract after acute episode subsides
Disposition
- D/c w/ wound care instructions, 2-d wound care f/u

Pearl
- Thought to be caused by hair penetrating into subcutaneous tissues creating abscess

**Bartholin Gland Cyst/Abscess**

**History**
- Severe localized pain in labia caused by obstructed Bartholin duct
- Difficulty walking & sitting secondary to pain
- Fever & signs of systemic tox are rare

**Findings**
- Painful, tender, cystic mass on inferior lateral margin of vaginal introitus, often w/ purulent drainage from sinus tract
- Typically mixed vaginal flora (*Bacteroides*, *E. coli*, *S. aureus*, gonorrhea, chlamydia)

**Evaluation**
- Culture for chlamydia, gonorrhea

**Treatment**
- I&D through mucosal surface, place Word catheter ×48 h
- Sitz baths TID for the 1st 2–3 d to assist drainage
- Gyn f/u for consideration of marsupialization to prevent recurrence

Disposition
- D/c w/ wound care instructions, 2-d wound care f/u

Pearl
- Recurrence rate still 5–15% after marsupialization; consider gyn malignancy

---

**PERIRECTAL ABScessses**

**History**
- Pain & swelling in rectal area w/ defecation & often w/ sitting down or walking
- High fever & signs of systemic tox are rare
Pts often have h/o IBD, obesity, DM, hemorrhoids, or rectal trauma.

**Findings**
- Rectal exam essential to ensure abscess localized outside of anal sphincter & to identify upper extent of abscess
- Typically mixed flora (*E. coli* species, *Enterococcus*, *Bacteroides* species, *S. aureus*)

**Evaluation**
- Lab studies unnecessary unless systemically ill
- DM or immunocompromised should have Chem, CBC
- CT/MRI if concern for intersphincteric or supralevator or postanal abscess or fistula

**Treatment**
- ED I&D of superficial abscesses outside the anal verge w/ visible indurated area
- Pain control; I&D extremely painful, procedural sedation often needed
- If abscess is only identified on rectal exam & no induration visible, refer to surgery for I&D under general anesthesia
  - DM or immunocompromised pts should undergo I&D in OR to ensure full drainage
- Pack w/ Vaseline gauze ×48 h, Sitz baths TID for 1st 2–3 d to assist drainage
- No abx for healthy host w/ superficial abscess
- Consider abx for immunocompromised, prosthetic device/valve, incomplete I&D
  - Levofloxacin 500 mg QD (ampicillin 1 g + gentamicin 80 mg q8h) + metronidazole 500 mg q8h, consider vancomycin

**Disposition**
- D/c w/ wound care instructions, 2-d wound care f/u
- Admit diabetic & immunocompromised for IV abx

**Pearls**
- 50–75% treated w/ I&D or spontaneous drainage will develop chronic anal fistula
- Consider adding stool softeners
Figure 4.1

INTRACRANIAL ABSCESS

(NEJM. 2014;371:447)

History

› Caused by contiguous spread (sinus, ear, dental), hematogenous seeding from distant infection, (endocarditis) or post-CNS surgery/penetrating trauma. Often predisposing factor such as underlying disease (eg, HIV, transplant patients)
› HA (most common), ± fever, meningismus, photophobia, sz (25%), vomiting, AMS frequently absent, may have CN palsy, gait disorder
› Subacute time course (vs. meningitis or encephalitis)
Findings
- Focal neuro deficits, low-grade fever, obtundation (mass effect), sz, AMS, nuchal rigidity, papilledema
- Wide variety of organisms depending on method of entry, 1/3 polymicrobial

Evaluation
- Blood cultures, CBC (WBC nonspecific), Chem, coags
- CT scan w/ & w/o IV contrast; MRI to help differentiate abscess from tumor
- Avoid LP if any concern for high ICP as may cause brain herniation

Treatment
- Emergency neurosurgical consult for drainage in OR; airway management, sz tx
- Early IV abx w/ good CSF penetration, tailored to likely pathogen
- Start broad-spectrum IV abx: Ceftriaxone 2 g + vancomycin 1 g + metronidazole 500 mg; consider adding coverage for toxoplasmosis, tuberculosis in immunocompromised
- Corticosteroids ONLY for tx of cerebral edema: Decadron 10 mg IV × 1 then 4 mg q6h

Disposition
- Neurosurgical intervention for operative washout, 6–8 wk IV abx then prolonged PO abx

Pearls
- Mortality 15%, unless abscess ruptures into ventricular system (mortality 27–85%)
- Morbidity from residual neuro deficits, new sz from scar tissue or neuropsych Δ (30%)

SOFT TISSUE INFECTIONS

Approach
- Careful hx, associated sxs (V/D, cough, abd pain, AMS), progression
- Check blood sugar if diabetic
- Assess VS for significant abnormalities that may indicate serious
infection (↓ BP, ↑ HR)
› If immunosuppressed (HIV/AIDS, elderly, malnourished, chronic steroids) or neutropenic, more intensive eval & testing: CBC, Chem, UA & cx, CXR; consider blood cx & admission
› If recent foreign travel: Consider travel-related infectious etiologies

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
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<tbody>
<tr>
<td><strong>Dermatology</strong></td>
<td>Subcutaneous cellulitis, erysipelas, impetigo (8a), SSSS, TSS, necrotizing fasciitis, abscess (4d), hidradenitis suppurativa, cat scratch (4h)</td>
</tr>
<tr>
<td><strong>GU</strong></td>
<td>Fournier gangrene</td>
</tr>
<tr>
<td><strong>Ophthalmology</strong></td>
<td>Periorbital cellulitis, orbital cellulitis</td>
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<tr>
<td><strong>ENT</strong></td>
<td>Ludwig angina</td>
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<tr>
<td><strong>Vector-borne</strong></td>
<td>Rocky Mountain spotted fever (8a), Lyme (8a)</td>
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<tr>
<td><strong>Bioterrorism</strong></td>
<td>Anthrax (4n)</td>
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</table>

**DERMATOLOGIC**

**Cellulitis**
(JAMA. 2016;316:325)

**History**
› Often no h/o broken skin; ± local trauma, recent surgery, FB
› May report fever, chills, malaise
› RF: Edema/lymphedema (facilitates bacterial growth)

**Findings**
› Warm, blanching erythema & tenderness to palpation, mild to moderate swelling
› May lead to dilated/edematous lymphatics (peau d’orange), bulla formation or linear streaking/lymphangitis
› ± distal skin disruption (eg, tinea pedis b/w toes w/ cellulitis of anterior shin)

**Evaluation**
› If elevated BS, check Chem, UA; rule out abscess clinically or with bedside ultrasound
› Consider blood cultures, CBC w/ differential, chemistries, CRP, CPK in
systemically ill pts
› Bacterial cultures of inflamed area not indicated; only 10–50% positive
› Most often caused by *Strep pyogenes* or *S. aureus* (including MRSA); can be from metastatic seeding

**Treatment**
› If LE cellulitis, recommend rest & elevation × 48 h, crutches if needed
› Typically aim to treat *Strep* & MSSA, but if purulent, MRSA coverage should be added. Duration of therapy 5–10 d outpt. IV therapy changed to oral after 48 h afebrile & regression from skin markings.
› Mild cellulitis:
  • Nonpurulent: Cephalexin, PCN VK, amoxicillin/clavulanate
  • Purulent: ADD trimethoprim-sulfamethoxazole, doxycycline, OR minocycline
  • PCN allergic: Clindamycin
› Moderate nonpurulent cellulitis (+ ≥2 SIRS criteria – T >38°C or <36°C, HR >90, RR >20, WBC >12 or <4):
  • Nonpurulent: IV cefazolin, IV ceftriaxone, IV PCN G, if PCN allergy clindamycin
  • Purulent: IV vancomycin, IV clindamycin or linezolid
› Severe nonpurulent (≥2 SIRS PLUS hypotension, immunocompromised or rapid disease progression):
  • Nonpurulent: IV vancomycin + IV piperacillin/tazobactam, IV imipenem, IV meropenem
  • Purulent: vancomycin, clindamycin, linezolid, daptomycin, tigecycline
› Pain control w/ NSAID/APAP; if severe pain, consider necrotizing infection
› Wound debridement if infected, contaminated, or devitalized wound
  • Surgery consult if aggressive/necrotizing infection/gas in soft tissue

**Disposition**
› D/c w/ PO abx & 24–48 h f/u, strict return instructions
› Admit if signs of systemic infection, DM, immunocompromise, failure of outpt tx

**Pearls**
› Due to inflammation of dermal & subcutaneous tissue due to nonsuppurative bacteria, infection does not involve fascia or muscles
› Consider Doppler vascular studies in single limb w/ diffuse swelling, posterior calf or medial thigh to rule out DVT
Mark border w/ permanent ink, write time & date
Mimics: Stasis dermatitis (more likely if bilateral), hematoma (consider if h/o trauma), gout (consider if over joint)

**Erysipelas**  

**History**
- Rapidly expanding, well-demarcated, painful plaque a/w swelling
- Extremes of age, obesity, DM, CHF, postop, nephrotic syndrome at higher risk
- Acute onset of fever, chills, malaise

**Findings**
- Skin painful superficial, indurated, raised; erythema w/ sharply demarcated border
- Irregular erythema w/ lymphangitis, may see desquamation, dimpling, vesicles, LAD
- Mostly found on lower extremities, sometimes on face, typically malar or “butterfly” pattern

**Evaluation**
- None indicated unless toxic appearing

**Treatment**
- Dicloxacillin, cephalexin, if c/f MRSA trimethoprim-sulfamethoxazole, doxycycline, clindamycin
- PCN allergic: Levofloxacin

**Disposition**
- D/c w/ PO abx & analgesics, elevate affected area, 24–48 h f/u, strict return instructions

**Pearls**
- Typically caused by group A β-hemolytic streptococcus; involves upper dermis & superficial lymphatics
- More superficial than cellulitis. Infection involving the ear “Milian’s ear sign” unique to erysipelas because ear does not contain deeper dermis tissues

**Staphylococcal Scalded Skin Syndrome (SSSS)**  
*(Am J Med. 2010;123:505)*

**History**
Young children <5 yr, fairly rapid progression of prodromal sore throat, conjunctivitis, fever, malaise to painful red skin w/ sloughing
Rare in adults, a/w chronic illness, immunosuppression, & renal failure

Findings
- No mucous membrane involvement (vs. TEN)
- Erythematous cellulitis followed by acute exfoliation: Bullae, vesicles → large sheets of skin loss resulting in scalded-appearing skin
- General malaise, fever, irritability, tenderness to palpation, does not appear severely ill

Evaluation
- None indicated unless systemically ill
- Positive Nikolsky sign (epidermis separates when pressure applied)

Treatment
- Similar to burns (IVF, topical wound care, burn consult)
- Vancomycin is antibiotic of choice

Disposition
- Admit for burn care, IVF; consider ICU

Pearls
- Caused by exfoliative exotoxins of S. aureus, reports of MRSA
- Separation of epidermal layers vs. more severe TEN (necrosis at level of basement membrane)
- Prognosis: Children (<5% mortality) often w/o significant scarring; adults (60% mortality)

Toxic Shock Syndrome (TSS)
(Lancet Infect Dis. 2009;9:281)

History
- Multiple sxes: Prodrome, pain at site of infection (out of proportion to findings), fever, GI upset, myalgia, confusion, lethargy, sore throat
- Recent surgery, infrequently changed packing (tampons, nasal packing), disruption of skin, commonly no source found

Findings
- Clinical Dx w/ findings from all organ systems:
  - Staph TSS: Temp >38.9°C, rash (diffuse macular erythoderma including palms/soles), desquamation (1–2 wk after onset), hypotension, multisystem involvement (≥3 GI, muscular, mucous
membranes, renal, hepatic, hematological, CNS), cultures negative except for blood culture for *S. aureus*

- **Strep TSS:** Culture positive for Strep (blood, CSF, tissue biopsy, throat, vagina, sputum); hypotension, multisystem involvement (≥2 renal impairment, coagulopathy, hepatic involvement, ARDS, generalized erythematous macular rash, soft tissue necrosis)

**Evaluation**
- CBC w/ differential, Chem, UA, LFTs, coags, cultures (blood, urine, throat, sputum, CSF)

**Treatment**
- Remove tampon or packing if still in place, drain abscesses if present; surgical debridement of necrotizing fasciitis or myositis; burn care
- Aggressive resuscitation, pressors if needed
- Abx may not have impact (toxin-mediated process); tx any identified source
- Staph: nafcillin, vancomycin, clarithromycin, linezolid + clindamycin (to suppress bacterial toxin synthesis)
- Strep: PCN G + clindamycin OR linezolid
- IVIG (blocks T-cell activation by superantigens) may be added if no clinical response to aggressive supportive therapy in first 6 h of treatment

**Disposition**
- ICU admission

**Pearls**
- Rate ↓ w/ ↓ in use of superabsorbent tampons
- Caused by inflammatory response to superantigen from toxin-producing Gram-positive organisms (*S. aureus, S. pyogenes*)
  - **Strep:** Usually after surgery or trauma; scarlet fever-like rash; 30–44% mortality, fulminant. Blood cultures positive ~60% of cases.
  - **Staph:** More indolent, 0–20% mortality. Blood cultures positive <5% of cases.

**Necrotizing Soft Tissue Infections**
*Crit Care Clin.* 2013;29:795

**History**
- Often diabetic, IVDU, obesity, EtOH abuse or nutritionally compromised
- Sudden onset of pain & swelling which progresses to anesthesia
Findings

- Cellulitis, skin discoloration/ecchymosis or gangrene, edema, spectrum of sensation from anesthesia to pain out of proportion
- Hemodynamic instability, crepitance (subcutaneous air due to gas-forming organisms), bullae & skin necrosis are rare but should trigger emergent surgical debridement
- Can progress to involvement of deeper layers, causing myositis or myonecrosis

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<thead>
<tr>
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<tr>
<td><strong>Points</strong></td>
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<td>Score of ≥6</td>
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Evaluation

- CBC w/ differential, Chem, UA, CRP, coags
- Plain radiographs less sens than CT/MRI in eval of gas w/i soft tissue

Treatment

- Early surgical consult for debridement (definitive tx); hemodynamic support
- Early & broad spectrum IV abx
  - Piperacillin/tazobactam 3.3 g IV q6–8h + clindamycin 600–900 mg IV q8h + ciprofloxacin 400 mg IV q12h + vancomycin 15–20mg/kg IV q12h (if c/f MRSA)
- Consider hyperbaric oxygen tx, IVIG after debridement (both controversial)

Disposition
ICU admission for surgical debridement

**Pearls**
- Mortality 16–46%, fatal if untreated
- Mostly *S. pyogenes* (group A), *Clostridium*, *S. aureus*, or mixed Gram + & – bacteria, anaerobes

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**GENITOURINARY**

**Fournier Gangrene**
*Surgeon. 2013;11:222*

**History**
- Men (10:1), >50 yo, diabetic, chronic EtOH abuse, immunocompromised
- Recent h/o instrumentation/surgery, urethral strictures or calculi, hemorrhoids, perirectal abscess, malignancies
- Fever, lethargy prodrome
- Rapidly progressing scrotal swelling, pain, erythema, warmth, possible purulent drainage

**Findings**
- Intensely tender, swollen, warm scrotum w/o clear fluctuance, pruritic genitalia
- Fever, chills, systemic sx (tachycardia, ↓ BP), ± crepitus, drainage
- Deep-space infection is often vastly greater than skin involvement would suggest

**Evaluation**
- CBC w/ differential, Chem, blood & urine cx, CRP, coags
- X-rays may show subcutaneous air; CT will show extent of infection & necrosis

**Treatment**
- Urology or general surgery consult for wide debridement & drainage
- Hemodynamic support & resuscitation w/ IVF, pressors
- Broad-spectrum abx: Vancomycin, Unasyn, Zosyn, clindamycin; Td prophylaxis
- Consider hyperbaric oxygen tx. IVIG after debridement.

**Disposition**
- ICU admission for surgical debridement, transfer for hyperbaric oxygen
therapy

Pearls

- Mortality 3–67%; early surgical debridement most strongly correlated w/ outcome
- Polymicrobial (*E. coli*, Proteus, Enterococcus, *Bacteroides*, & other anaerobes) necrotizing infection of perineum, scrotum, & penis characterized by obliterative endarteritis of the subcutaneous arteries resulting in gangrene
- Rapid destruction of fascial planes

**OPHTHALMOLOGIC**

(*Dis Mon.* 2017 Feb;63(2):30–32)

**Periorbital/Preseptal Cellulitis**

**History**

- Recent infection of sinuses, periorbital skin, trauma to periorbital area, insect bites

**Findings**

- Unilateral eyelid swelling, erythema, warmth, discoloration of skin
- Injected sclera, conjunctival ecchymosis
- No pain w/ extraocular movements, no proptosis, normal pupillary reaction & vision

**Evaluation**

- CBC w/ differential, blood cultures, CT scan of orbits to evaluate for orbital extension

**Treatment**

- Head elevation
- Abx: Ceftriaxone or Unasyn 3 g IV q6h (if need admission) or cephalexin, dicloxacillin, clindamycin or Augmentin 500 mg PO TID × 10 d if d/c

**Disposition**

- Admit if appears systemically ill or has other comorbidities
- O/w d/c w/ close ophthalmology f/u (2 d)

**Pearls**

- Infection of soft tissue of eyelids & periorbital region anterior to orbital septum
Most often caused by *Staph & Strep*, rarely *H. influenza* since vaccine
Distinguish from orbital cellulitis: No pain w/ EOM or proptosis in periorbital cellulitis

**Orbital Cellulitis**

**History**
- Orbital pain increased w/ extraocular movements, ↓ vision
- Recent infection of sinuses, periorbital skin, trauma to periorbital area, facial trauma

**Findings**
- Fever, HA, rhinorrhea, malaise
- Proptosis & ophthalmoplegia are cardinal signs
  - Unilateral eyelid swelling, erythema, warmth, discoloration of skin
  - Injected sclera, chemosis
  - Tenderness on gentle globe palpation, ↑ IOP
  - ↓ visual acuity, relative afferent pupillary defect, visual field abnormalities

**Evaluation**
- CBC w/ differential, CT scan of orbits, soft tissue aspirate if possible, blood cultures

**Treatment**
- Ophthalmology consult, head elevation
- Aggressive tx w/ immediate broad spectrum IV abx

**Disposition**
- Admission for abx

**Pearls**
- Infection of soft tissues of orbit posterior to orbital septum
- Most common: *Strep, Staph, H. influenzae*, polymicrobial
- Cx: Meningitis, brain abscess, death, cavernous sinus thrombosis
  (bilateral involvement, rapidly worsening, congestion of veins of face or conjunctiva)

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**Otolaryngologic**

**Ludwig Angina**

**History**
- A/w dental infection, mandible fractures, tongue piercings
- Typically males, a/w DM< HIV, malnutrition, alcoholism

**Findings**
- Fever, malaise, neck swelling, trismus, drooling, pain with tongue movement, stridor
- Swelling of submandibular/sublingual space feels hard & “board like” or woody

**Evaluation**
- CBC w/ differential, Chem, UA, blood cultures, coags
- CT scan head & neck

**Treatment**
- If severe swelling, aggressively ↑ infection, or airway threatening, endotracheal intubation may be difficult, fiberoptic nasotracheal intubation may be the best initial approach w/ cricothyrotomy as backup
- Consultation w/ otolaryngologist for admission
- Broad spectrum IV antibiotics (clindamycin, unasyn, zosyn)

**Disposition**
- Admit to ICU for IV abx, airway watch

**Pearls**
- Rapidly spreading bilateral cellulitis of submandibular space a/w displacement of tongue causing life-threatening airway obstruction
- Polymicrobial, includes group A strep, also Staph, *Fusobacterium*, *Bacteroides*
- Surgical debridement was tx in preantibiotic era; now only if unresponsive to IV abx or e/o purulent collections

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**VIRAL INFECTIONS**

<table>
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<tr>
<th>Pathophysiology</th>
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<tbody>
<tr>
<td>Cardiac</td>
<td>Myocarditis (1j), pericarditis</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Pneumonia (2b), URI/bronchitis (2b)</td>
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<tr>
<td>GI</td>
<td>Hepatitis (3g), gastroenteritis (3b), EVD</td>
</tr>
<tr>
<td>Dermatology</td>
<td>Herpes zoster (8a), rubella (8a), measles (8a), roseola (8a),</td>
</tr>
</tbody>
</table>
**EBOLA VIRUS DISEASE (EVD)**

*(Crit Care. 2016;20:217)*

**History**
- Fever, chills, myalgias, malaise, then 5 d later GI symptoms such as severe watery diarrhea, nausea/vomiting, abd pain. Other symptoms like chest pain, SOB, HA, confusion may also develop. Bleeding is not universally present, mild bleeding (30%), frank hemorrhage is uncommon.
- 2–21 d incubation period
- Recent travel to country with outbreak (primarily West Africa, check CDC website for current updates)

**Findings**
- Fever, abd pain
- Diffuse erythematous maculopapular rash may develop day 5–7
- Pts with fatal disease typically die day 6–16 from complications including MSOF, sepsis

**Evaluation**
- CBC (leukopenia, lymphopenia, late elevated neutrophils, thrombocytopenia), ↑amylase, ↑AST/ALT, ↑PT/PTT, ↑fibrinogen, UA (proteinuria)
- RT-PCR assay specific for ebola

**Treatment**
- Supportive care of complications such as hypovolemia, electrolyte abnormalities, hematologic abnormalities, hypoxia, MSOF, septic shock, DIC
- Volume repletion, pressors as needed, pain control, nutritional support

**Disposition**
- Strick contact isolation, prevent contact or splashes with blood & body fluids, equipment & surfaces
**Pearls**
- Can be confused with more common diseases (malaria, typhoid, PNA, meningitis)
- Enters through mucous membranes, breaks in skin or parenterally

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**INFECTIOUS MONONUCLEOSIS**

(*NEJM.* 2010;362:1993)

**History**
- Fever, pharyngitis, lymphadenopathy, HA, rash, nonspecific sx
- 4–6 wk incubation period, 1–2 wk prodrome: Fatigue, malaise, myalgias, low-grade temp

**Findings**
- Low-grade temp, pharyngitis, tonsillitis
- Tender & firm LAD for 1–2 wk, most often postcervical nodes, but can be generalized
- Rash: Papular erythematous on UE, erythema nodosum, erythema multiforme
- Splenomegaly; severe abd pain uncommon, may indicate splenic rupture
- May have petechiae, jaundice, hepatomegaly, periorbital edema

**Evaluation**
- CBC: ↑ WBC, ↑ atypical lymphocytes, ↓ platelets, ↑ LFTs (bilirubin, AST, ALT); monospot test
- Consider rapid strep if clinical ambiguity

**Treatment**
- Supportive, rest, analgesics, antipyretics
- Corticosteroids if airway edema

**Disposition**
- Admission rarely indicated; close PCP f/u
- Advise to avoid contact sports or vigorous exercise × 1 mo to prevent splenic rupture

**Pearls**
- Represents syndrome response to EBV (90% of people have EBV); most cases of mono caused by EBV but most EBV infections do not
result in mono

- Secondary etiology: CMV
- Transmission through saliva; infects epithelial cells of oropharynx & salivary glands
- B lymphocytes become infected → allows viral entry into bloodstream
- Self-limited; usually spontaneous resolution in 3–4 wk, complete in several months

---

**HIV/AIDS**


**History**

- Fever, fatigue, night sweats, pharyngitis, diarrhea, myalgia/arthralgias, HA, flu-like sx

**Findings**

- Generalized maculopapular rash, oral ulcers (thrush), fever, lymphadenopathy

**Evaluation**

- CBC: Leukopenia, thrombocytopenia, ↑ LFTs
- ELISA to test for HIV Ab; if + confirm w/ Western blot (VL >100 K in acute infection)
- PCR to detect viral load, CD4 count

**Treatment**

- Counseling pre- & post-HIV testing

**Disposition**

- D/c unless systemically ill, ID f/u for antiretroviral tx

**Pearls**

- Transmitted through sexual contact (70%), IVDU; mother-to-child transmission possible during pregnancy or birth
- Untreated HIV → AIDS (CD4 <200) w/ life expectancy of 2–3 yr

---

**Opportunistic Infection Prophylaxis**

<table>
<thead>
<tr>
<th>Infection</th>
<th>Indication</th>
<th>Prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB</td>
<td>+PPD (&gt;5 mm) or high-risk exposure</td>
<td>Isoniazid + Vit B6 × 9 mo</td>
</tr>
<tr>
<td>Condition</td>
<td>CD4 Count</td>
<td>Treatment</td>
</tr>
<tr>
<td>--------------</td>
<td>----------------------</td>
<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td>PCP PNA</td>
<td>CD4 &lt;200 or thrush</td>
<td>Bactrim QD OR dapsone 100 QD OR Atovaquone 1500 QD OR Pentamidine 300 q4wk</td>
</tr>
<tr>
<td>Toxoplasmosis</td>
<td>CD4 &lt;100 AND + toxoplasma serology</td>
<td>Bactrim QD OR dapsone 200 QD + pyrimethamine 75 QD + leucovorin 25 qwk</td>
</tr>
<tr>
<td>MAC</td>
<td>CD4 &lt;50</td>
<td>Azithromycin 1200 qwk OR clarithromycin 500 BID</td>
</tr>
</tbody>
</table>

### Complications of HIV/AIDS

<table>
<thead>
<tr>
<th>CD4 Count</th>
<th>Cx</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;500</td>
<td>Kaposi sarcoma, lymphoma, oral hairy leukopenia</td>
</tr>
<tr>
<td></td>
<td>Candidiasis: Oral, esophageal, vaginal</td>
</tr>
<tr>
<td></td>
<td>Recurrent bacterial infections</td>
</tr>
<tr>
<td></td>
<td>Pulmonary &amp; extrapulmonary TB</td>
</tr>
<tr>
<td></td>
<td>HSV, VZV</td>
</tr>
<tr>
<td>&lt;200</td>
<td>PCP PNA, *Toxoplasma, Bartonella, Cryptococcus, Histoplasma, Coccidioides, HIV encephalopathy</td>
</tr>
<tr>
<td>&lt;50–100</td>
<td>CMV, MAC</td>
</tr>
<tr>
<td></td>
<td>Disseminated <em>Bartonella</em>, invasive aspergillosis</td>
</tr>
<tr>
<td></td>
<td>CNS lymphoma, PML</td>
</tr>
</tbody>
</table>

### Organ Involvement of HIV/AIDS

<table>
<thead>
<tr>
<th>Organ</th>
<th>Manifestation/Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constitutional</td>
<td>Fevers: Bacterial, MAC, CMV, PCP, TB, lymphoma, drug rxn, endocarditis</td>
</tr>
<tr>
<td>Dermatologic</td>
<td>Kaposi sarcoma, lymphoma, VZV, HSV, HPV, <em>Molluscum contagiosum</em></td>
</tr>
<tr>
<td>Ophthalmologic</td>
<td>CMV retinitis</td>
</tr>
<tr>
<td>Oral</td>
<td>Oral hairy leukopenia, Kaposi sarcoma, thrush, aphthous ulcers</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Dilated cardiomyopathy, endocarditis, myocarditis, CAD, pericardial effusion, LVH</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>PCP PNA, TB, fungal PNA (aspergillosis, <em>Cryptococcus</em>, etc.), CMV</td>
</tr>
<tr>
<td>GI</td>
<td>Oral candida, hairy leukoplakia, esophagitis, enterocolitis, GIB (CMV, Kaposi, lymphoma), proctitis, hepatitis, diarrhea (<em>Cryptosporidium, Isospora</em>)</td>
</tr>
</tbody>
</table>
Renal
Nephropathy (drugs), HIV-associated nephropathy

Hematologic
Anemia (chronic dz), leukopenia, thrombocytopenia

Oncologic
NH & CNS lymphoma, Kaposi sarcoma, cervical cancer

Endocrine
Hypogonadism, metabolic syndrome, adrenal insufficiency, HIV wasting syndrome

Neurologic
Meningitis: Cryptococcus, bacterial, viral, TB, cocci, histoplasmosis
Neurosyphilis: Meningitis, CN palsy, dementia
Mass (toxoplasmosis), AIDS dementia, myelopathy, peripheral neuropathy, HIV encephalopathy, progressive multifocal leukoencephalopathy

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Drugs</th>
<th>Rxn</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nucleoside reverse transcriptase inhibitors</td>
<td>Zidovudine (AZT)</td>
<td>Bone marrow suppression (AZT)</td>
</tr>
<tr>
<td></td>
<td>Didanosine</td>
<td>pancreatitis (didanosine)</td>
</tr>
<tr>
<td></td>
<td>Stavudine</td>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td></td>
<td>Zalcitabine</td>
<td></td>
</tr>
<tr>
<td>Nonnucleoside reverse transcriptase inhibitors</td>
<td>Nevirapine</td>
<td>Steven–Johnson syndrome</td>
</tr>
<tr>
<td></td>
<td>Efavirenz</td>
<td></td>
</tr>
<tr>
<td>Protease inhibitors</td>
<td>Indinavir</td>
<td>N/V, diarrhea, hyperlipidemia,</td>
</tr>
<tr>
<td></td>
<td>Atazanavir</td>
<td>hyperglycemia, fat redistribution</td>
</tr>
</tbody>
</table>

**Rabies**


**History**
- Exposure to rabid (agitated, drooling, unprovoked attack) mammal (dog, cat, bat, raccoon)
- Prodrome lasts 2–10 d; nonspecific: fatigue, loss of appetite, HA, anxiety, irritability, fever

**Findings**
- Encephalitic (80%) & paralytic (20%) rabies, affects brain & spinal cord respectively
  - Encephalitic form: hypersalivation, sweating, piloerection, hydrophobia, impaired consciousness → quadripareisis → death
  - Paralytic form: weakness in bitten limb, progression to quadripareisis &
facial weakness, urinary incontinence → neurologic progression → death
• Dog-acquired cases: hydrophobia, aerophobia, encephalopathy
• Bat-acquired cases: symptoms at exposure site, abnl neuro findings (tremor, myoclonus, CN exam, motor/sensory exam)

Evaluation
› Neutralizing anti-rabies virus AB in serum (if not vaccinated), RABV antigen in tissues, RABV RNA in saliva or CSF
› CSF: pleocytosis, ↑ protein; Ab titer diagnostic, regardless of vaccine status
› Imaging (head CT, MRI) used to evaluate for other causes of encephalopathy

Treatment
› Supportive, palliative. Universally fatal within 14 d of initial symptoms
› No proven medical tx has been shown to be effective
› Therapeutic coma (ketamine, benzo’s) & antiviral therapy (amantadine, ribavirin) rarely a/w survival

Disposition
› ICU admission if neuro or resp sxes w/ inpt ID consult
› Notify public health department & animal control center
› Identify others at risk & initiate postexposure prophylaxis if indicated

 Pearls
› Caused by *Lyssavirus* in family *Rhabdoviridae* transmitted by animal bites
› IP is variable, typically 20–90 d but ranges from days to 1 year
› Dogs are the most commonly infected animals worldwide, but very rare in US & Canada
› Rabies PEP
  • Wound care (soap, water, irrigation w/ povidone–iodine solution), debridement of devitalized tissue, secondary closure, update Tetanus vaccination
  • If domestic dog or cat bite, determine vaccination status of animal from owner. If animal can be observed, start PEP only if animal develops symptoms
  • Assess rabies risk & need for human rabies immune globulin (HRIG) & human diploid cell vaccine (HDCV)
HRIG: 20 IU/kg; as much as possible at exposure site, remaining administered at distant site (eg, deltoid)
HDCV: 1 mL dose in deltoid in ED. F/u doses given days 3, 7, 14
HDCV 5th dose on day 28 if immunocompromised
Do not stop rabies immunization b/c of mild rxn to vaccine doses
Rabies cases from nonbite exposures > from known bite exposures; consider prophylaxis for any contact w/ high-risk animals (eg, bats, skunks, raccoons, coyotes, foxes)

**SYPHILIS**

_(Lancet. 2017;389:1550–1557)_

**History**
- Primary syphilis: hallmark is a chancre – painless, usually solitary, indurated, clean-based ulcerative lesion 2–3 wk after contact infected lesion.
- Secondary syphilis: painless, macular rash of 1–2 cm, lesions on palms & soles; but can vary in appearance (thus the “great imitator”). May be a/w malaise, myalgia, HA (syphilitic meningitis), sore throat.
- Latent disease
- Tertiary syphilis – late neurosyphilis (general paresis, tabes dorsalis), cardiovascular syphilis (aneurysm of ascending aorta, AV insufficiency, CAD), gummatous syphilis (reactive, granulomatous processes)

**Findings**
- Primary syphilis: chancre, ± nontender LAD
- Secondary syphilis: painless rash may be associated with fever, LAD, HSM, hepatitis
- Tertiary syphilis: General paresis causes progressive dementia, seizures, psychiatric syndromes. Tabes dorsalis “lightening” radicular pains, ataxia, Argyll Robertson pupil (small, do not react to light but accommodate), loss of reflexes, impaired vibratory sense.

**Evaluation**
- Treponemal test (eg, RPR, VDRL) → nontreponemal assay to confirm (eg, FT-ABS, TP-PA)
- Reactive CSF VDRL is diagnostic of neurosyphilis
**Treatment**
- Early (primary, secondary, or early latent): benzathine PCN G 2.4 million units IM ×1 OR doxycycline 100 mg PO BID × 14 d
- Late/unknown duration latent syphilis: benzathine PCN G 2.4 million units IM wk × 3 wk OR doxycycline 100 mg BID x 28 d
- Neurosyphilis: PCN G 3–4 million units IV q4H × 10–14d
- After treatment 30–50% patients have Jarisch–Herrxheimer reaction (fever, myalgia, worsening skin rash). Will self-resolve, can tx with IVF, antipyretics.

**Disposition**
- Pts with neurosyphilis or cardiovascular syphilis should be admitted for antibiotics.

**Pearls**
- Caused by *Treponema pallidum*
- Increases risk of HIV infection; HIV incidence up to 20% in the decade after syphilis Dx
- Spread through direct lesion contact, small proportion through blood transfer

---

**TETANUS**

*(Crit Care. 2014;18:217)*

**History**
- Acute onset hypertonia, painful muscular contractions esp. the masseter (“lockjaw”) → generalized muscle spasms/rigidity, dysphagia
- RFs: Inadequate vaccination status, chronic wound, IVDU

**Findings**
- Spasms of muscles in close proximity to site of injury, cephalic, lockjaw, risus sardonicus (characteristic grimace) tetanic sz, respiratory failure
- Autonomic Dysfxn: BP ↑ or ↓, dysrhythmias, cardiac arrest
- Cx include fractures & dislocations

**Evaluation**
- No spec tests available; clinical Dx

**Treatment**
- Heavy sedation (benzos, propofol) & paralysis supported by artificial ventilation
- Magnesium sulfate has been used to control muscle spasms
- Intrathecal, intramuscular antitetanus immunoglobulin hastens clinical improvement
- Abx: Metronidazole, PCN G, or doxycycline

**Disposition**
- ICU admission

**Pearls**
- *C. tetani* is obligate anaerobe, gram-positive spore forming bacillus, resistant to heat, desiccation, & disinfectants
- DTaP (diphtheria, tetanus, pertussis; inactivated) vaccine given at 2, 4, & 6 mo, booster given b/w 15–18 mo & at 4–6 yr; booster recommended q10y or if dirty wound
- Mortality 30–45%; if received tetanus toxoid at sometime in life mortality 6
- Slow recovery over 2–4 mo, usually complete resolution of sx

**Prevention**

<table>
<thead>
<tr>
<th>Wound</th>
<th>Vaccination Hx</th>
<th>Prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor, clean</td>
<td>&lt;3 doses of tetanus toxoid, &gt;10 yr since last dose or unknown immunization status</td>
<td>Td toxoid booster</td>
</tr>
<tr>
<td>All other wounds</td>
<td>&lt;3 doses of tetanus toxoid, &gt;5 yr since last dose or unknown immunization status</td>
<td>Td toxoid booster</td>
</tr>
<tr>
<td></td>
<td>&lt;3 doses of tetanus toxoid, or unknown immunization status</td>
<td>Tetanus immune globulin (250 mg or 500 IU IM)</td>
</tr>
</tbody>
</table>

- Clean & debride wound as needed
- Pts who have not completed primary immunization series should repeat Td booster in 4–8 wk & 6–12 mo

**SCABIES**
History
- Persistent pruritus, worse at night. Sometimes multiple family members involved
- Common in overcrowding, poor hygiene, elderly, homeless. More common in winter (survive longer on fomites, more crowded living)

Findings
- Small, pruritic, erythematous papules. Typically, web spaces between fingers & toes, flexor aspects of wrists, under armpits, around umbilicus, under knees, around nipples, genital region
- Burrow from mites: elevated thin red or gray line
- Secondary skin infections may also be present

Evaluation
- Clinical Dx. Other Dx tests (skin scrapings, shave biopsy, tape test, etc.) may increase certainty but negative results do not rule out

Treatment
- Permethrin 5% cream, 2 applications 1 wk apart OR oral ivermectin 200 μg/kg, 2 applications 2 wk apart
- Second line: crotamiton 10% cream, lindane 1% lotion
- Symptomatic relief, tx 2° infections & household members, clean clothes/linens

Disposition
- D/c w/ instructions for household to be treated, decontaminate clothing, bedding
- Exclude from school until treated, topical permethrin usually effective w/i 12 h

Pearls
- Caused by female mite, S. scabiei
- Delayed type IV hypersensitivity rxn to mite proteins (from saliva, feces, eggs, mite itself), symptoms initially develop 3–4 wk after exposure, then 1–2 d after re-exposure
- Skin-to-skin contact, indirect contact through bedding or clothing
<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tick-borne</td>
<td>Lyme, Rocky Mountain spotted fever, ehrlichiosis, babesiosis</td>
</tr>
<tr>
<td>Mosquito-borne</td>
<td>Malaria, yellow fever, dengue fever, West Nile, eastern equine encephalitis</td>
</tr>
</tbody>
</table>

**Tick-borne Diseases**

**Lyme Disease** *(Borrelia burgdorferi)*  
*(NEJM. 2014;371:684)*

**History**
- 1/3 recall h/o tick bite. Often by *I. scapularis* (deer tick) in endemic area b/w May & August, or of exposure to wooded areas, incubation period 3–31 d.
- Tick must be attached for >36 h to cause infection
- Erythema migrans (typical “bull’s eye” rash w/ central clearing, but can be uniform or enhanced central erythema w/o clearing; can last 3–4 wk if untreated); malaise, fatigue

**Findings**
- Progression can result in polyarthritis (late), cardiac conduction dz, neurologic sequelae
- Rash: Erythema migrans (absent in 20–40%)
- Lyme carditis – AV block &/or myopericarditis
- Lyme meningitis; does not present as classic bacterial meningitis
  - Early: HA, Bell’s palsy, radiculoneuritis, erythema migrans
  - Late: Neurocognitive Dysfxn (ie, encephalopathy)

**Evaluation**
- Testing not recommended for pts w/ only erythema migrans, poor sensitivity
- For pts w/ nonerythema migrans presentations: First antibody screen assay (EIA), if positive, obtain immunoblot. Both results positive required to confirm Dx.
- ECG to assess for HB, CSF may be considered in pts w/ neurologic involvement

**Treatment**
Tick removal: Using forceps or tweezers, grasp the tick as close to skin as possible, pull upward w/ steady pressure. Disinfect site, save tick for identification.

See table, consider Rheum consult
Avoid doxycycline in pregnant pts

**Dispositions**
- D/c w/ abx regimen, PCP f/u unless has symptomatic AV block/syncope

**Pearls**
- Deer tick tiny (head of pin) vs. dog tick (larger, more, common, don’t transmit Lyme)
- Most common tick-borne dz in US; 90% of cases in MA, CT, RI, NY, NJ, PA, MN, WI, CA

<table>
<thead>
<tr>
<th>Sxs/Findings</th>
<th>Onset After Bite</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic exposure</td>
<td>W/i 72 h</td>
<td>Doxycycline 200 mg PO ×1</td>
</tr>
<tr>
<td>EM rash, nonspecific viral syndrome (fever, fatigue, malaise), regional LAD</td>
<td>Few days–1 mo</td>
<td>Doxycycline 100 mg PO BID or amoxicillin 500 mg PO TID or cefuroxime 500 mg PO BID × 14 d</td>
</tr>
<tr>
<td>CN palsy w/o meningitis, asymptomatic carditis</td>
<td>Days–10 mo</td>
<td>Ceftriaxone 2 g IV QD × 14 d, up to 21 d for Lyme carditis</td>
</tr>
<tr>
<td>Musculoskeletal (arthritis), neurologic (encephalitis, meningitis, neuropathy), symptomatic carditis</td>
<td>Months–years</td>
<td>Arthritis alone: PO regimen as above × 28 d Neurologic sxs/findings: Ceftriaxone 2 g IV QD × 14–28 d</td>
</tr>
</tbody>
</table>

**Rocky Mountain Spotted Fever (Rickettsia Rickettsii)**
(Lancet Infect Dis. 2007;7:724)

**History**
- Tick exposure. IP 2–14 d.
- Sudden high fever, malaise, HA, myalgia, anorexia, N/V, abd pain, photophobia
- Rash 2–5 d after fever

**Findings**
- Multisystem dz; Temp >102°F, may be ↓ BP on presentation
- Rash (85–90%): Petechial rash typically starts at wrist & ankles; may
be diffuse at onset. Typically moves out (palms & soles) then in (arms, legs, & trunk). By end of first week, rash is maculopapular with central petechiae, spares face. ~10% have no rash

- Multiple systems can be involved: Cardiac (myocarditis), pulmonary (cough, PNA), GI (abd pain, N/V, hepatomegaly), renal (ARF), CNS (meningismus, photophobia, confusion), ocular (conjunctivitis, retinal hemorrhage, arterial occlusion), muscular (CK elevation)

Evaluation

- IFA assay most commonly used, cannot distinguish between rickettsial diseases
- Ab not detectable until 7–10 d after disease onset
- CBC (thrombocytopenia, anemia), Chem (hyponatremia, ↑ BUN), LFTs, coags, blood cx
- CXR if appear toxic or abnl lung findings
- CT or MRI for AMS may show infarction, edema, meningeal enhancement
- CSF may show pleocytosis, nl glucose, elevated protein

Treatment

- Intubation if indicated, resuscitation; dialysis, fluids, PRBC + platelets if indicated
- Abx: Tetracyclines (doxycycline), chloramphenicol

Disposition

- Most require hospitalization, consider ICU (rapid progression)

Pearls

- *R. rickettsii* obligate intracellular bacterium spread by ticks to human endothelial cells causing small, medium vessel vasculitis
- Found in US (primarily MD, VA, NC, SC, OK, TN, AR), also western Canada, western & central Mexico, & South America
- Mortality 5% treated, 20% untreated

**Ehrlichiosis and Anaplasmosis**

*(Prim Care. 2013;40:619)*

History

- Travel to endemic area in spring/early summer, tick bite; 5–14 d incubation
- Fever, myalgia, HA, malaise, cough, chills, rash (10–30%)

Findings
Fever, LAD (<25%), maculopapular, petechial, or macular rash on UE/trunk

**Evaluation**
- CBC (↓ WBC, ↓ plat), ↑ LFT, LDH, ↑ ESR; blood cultures not helpful
- PCR most sens during acute infection, serologies, peripheral smear
- CT/LP if severe HA to R/O meningitis, may show pleocytosis, mildly elevated protein

**Treatment**
- Analgesics, resuscitation, abx: Doxycycline 100 mg IV/PO BID × 10 d

**Disposition**
- Admit as needed for supportive tx

**Pearls**
- Obligate intracellular gram-negative bacteria; *Anaplasma* infects granulocytes (HGA), *Ehrlichia* targets monocytes (HME); distinct epidemiologically but same clinical picture
- HGA—American deer tick; found in NE & Midwest US in summer
- HME—American dog tick, lone star tick; found in SE & south-central US, April–September

**Babesiosis**
*(NEJM. 2012;366:2397)*

**History**
- Travel to endemic areas b/w May & September, tick bite; 1–4 wk incubation
- Usually asymptomatic in healthy host; affects elderly, immunocompromised, asplenic
- Fever, weakness, fatigue, HA, photophobia, AMS, cough, SOB, N/V, abd pain, arthralgias, chills, myalgias, anorexia, cough

**Findings**
- Fevers, rigors, hepatosplenomegaly, pharyngeal erythema, jaundice, retinopathy

**Evaluation**
- CBC (hemolytic anemia), ↓ haptoglobin, ↑ LFTs, ↑ LDH, ↑ reticulocytes, ↓ platelets, UA (proteinuria or hematuria)
- Wright or Giemsa peripheral blood smear; PCR, immunofluorescence Ab testing
- Serial blood smear may show parasites
Treatment
- Resuscitation, symptomatic tx, airway management
- Early abx: Atovaquone IV + azithromycin IV OR clindamycin PO or IV + quinine PO
- RBC exchange transfusion if parasite load >10%, severe anemia, end-organ Dysfxn

Disposition
- Admit for ongoing supportive therapy, abx
- Most pts recover spontaneously in 1–2 wk, fatigue may continue for months

Pearls
- Protozoan parasite Babesia transmitted by tick or blood transfusion from infected individual
- Peak in May–October; found in Europe & US (MA, NY, RI, CT, upper Midwest, Northwest)
- Mortality 10% (US), 50% (Europe); if symptomatic

MOSQUITO-BORNE DISEASES

Malaria
(WHO Guidelines for the Treatment of Malaria. 3rd ed. 2015.)

History
- Travel to Central & South America, Sub-Saharan Africa, India, SE Asia, Middle East, Caribbean, South Central Asia; incubation period 7–30 d, may present months after
- Paroxysmal chills, sweats, & high fevers q48–72h
- Fever, cough, fatigue, myalgias, malaise; less common anorexia, N/V, diarrhea, HA

Findings
- Fever, hypotension, tachycardia, may see jaundice, signs of anemia, splenomegaly, icterus
- Severe malaria: AMS, ≥2 szs, pulm edema, HD unstable, >40°C, DIC, severe anemia, renal failure, hypoglycemia, hyperparasitemia, acidosis, hyperbilirubinemia
- Cerebral malaria: AMS, meningitis, szs, encephalopathy; 15–20% mortality even w/ tx

Evaluation
CBC, Chem, haptoglobin, UA, blood cx, thick & thin blood smear, rapid antigen tests
- Triad of thrombocytopenia, ↑ LDH, atypical lymphocytes
- Head CT/LP if AMS or encephalopathy to look for cerebral malaria
- CXR if signs of pulm edema

**Treatment**
- Airway management, IV access & IV fluid resuscitation, infectious dz consultation
- Prophylaxis regimen often recommended; depends on region of travel
- Use DEET & insect repellent, bed nets w/ permethrin, long-sleeved clothing
- Tx regimen dependent on geography, which species, & severity of dz
- Watch QT interval when giving antimalarials

**Disposition**
- Admit if suspected or confirmed, if child, pregnant, or immunodeficient
- ICU if end-organ sxs noted, signs of cerebral malaria
- Thin & thick blood smears should be performed qwk × 4 after d/c to ensure resolution

**Pearls**
- *Plasmodium (ovale, vivax, malariae, falciparum)* cause malaria, transmitted through bite of infected female *Anopheles* mosquito, causing systemic infection of erythrocytes
- *P. falciparum* most severe: Can cause cerebral malaria, pulm edema, renal failure, anemia; highest occurrence in Sub-Saharan Africa
- *P. vivax* & *P. ovale* produce dormant form in liver, usually causes uncomplicated malaria
- 2 million deaths annually, majority in kids <5 y/o, ~90% in rural Sub-Saharan Africa
- Sickle cell trait, thalassemia, Hemoglobin C disease & G6PD deficiency are protective
- Pregnant women up to 10× more likely to contract & develop severe malaria, ↑ M&M

**Yellow Fever**
*(Clin Infect Dis. 2007;44:850)*

**History**
- Travel to endemic area (Sub-Saharan Africa [90%] & South America),
incubation 3–6 d
- Mild form sudden fever HA → more severe cases with high fever, chills, HA, myalgias, lumbosacral pain, anorexia, N/V, dizziness → 10–25% have severe recurrence 2 d later with multiple organ systems involved (GI, renal, cardiac, hematologic)

Findings
- High fever, relative bradycardia, N/V, epigastric tenderness
  - Severe cases: ↑ fever, HA, N/V, abd pain, somnolence, jaundice, hematologic complications (eg, hematemesis, melena, petechiae, epistaxis)
- Late: ↓ BP, shock, confusion, coma, DIC, hemorrhage
- Liver is the most affected organ: Hepatocellular damage (steatosis, necrosis); bleeding
- Kidney is also affected: Renal insufficiency, albuminuria, ATN
- Cardiac: Fatty infiltration of myocardium → myocarditis & arrhythmias

Evaluation
- CBC (leukopenia, thrombocytopenia), ↑ LFTs, abnl coags, ↑ BUN/Cr, fibrinogen (DIC), ↓ ESR, serology, viral IgG, IgM

Treatment
- Resuscitation, supportive, symptomatic tx; no antiviral meds approved
- Live attenuated vaccine available for prevention, extremely effective

Disposition
- Admit for supportive care

Pearls
- *Flavivirus* transmitted by *A. aegypti* mosquito during tropical wet & early dry season, causes viral hemorrhagic fever
- Up to 20–50% mortality in symptomatic patients
- Mandated reporting to WHO, local health dept

**Dengue Fever**  
(*NEJM.* 2012;366:1423)

History
- Travel to endemic areas: Mostly SE Asia, Central America, Western Pacific, sometimes from Eastern Mediterranean, Africa
- Sxs begin after 3–7 d incubation
- High fever: Abrupt onset × 1–7 d, biphasic, w/ HA, vomiting, myalgia, joint pain
Rash: Characteristically bright red blanching petechiae, usually 1st on lower limbs & chest → morbilliform, maculopapular & sparing palms & soles → desquamation

Findings
- Hemorrhagic fever (DHF) or shock syndrome (DSS) occur during 2nd infection by different dengue virus
- Fever, ↓ BP, rash, LAD, hemorrhage (petechiae, purpura, epistaxis, GIB, menorrhagia)
- DHF: High fever, hepatomegaly, hypotension, DIC; begins w/ sudden ↑ in temp & flu sx

Evaluation
- CBC (↑ Hct, ↓ plat, ↓ WBC), Chem (↑ BUN), ↑ LFTs, guaiac, DIC panel, ELISA, lactate
- CXR, head CT (if AMS), US, viral culture, dengue antigen tests, PCR, viral serologies

Treatment
- Aggressive supportive therapy, IVF, fluid status important 2/2 to plasma leakage, blood transfusions for severe bleeding

Disposition
- Admit for supportive tx

Pearls
- Caused by dengue virus (*Flavivirus*) infection, transmitted by *A. aegypti* mosquitoes
- Called “break-bone fever” due to acute onset severe HA, muscle & joint pains
- Benign acute febrile illness that can cause bleeding or DIC in small # of cases but can lead to lethal DHF

**West Nile Disease**
*(MMWR. 2014;63:521)*

History
- Outdoor exposures in area of outbreak during summer months, 2–14 d incubation
- Most infections asymptomatic. Symptomatic pts have fever, HA, malaise, myalgia, GI symptoms, rash.
- <1% have neuroinvasive Dx (meningitis, encephalitis, flaccid paralysis)

Findings
- Low-grade fever, hepatomegaly, splenomegaly, generalized LAD
- Rash: Erythematous maculopapular
- CNS: AMS, confusion, coma, meningismus, papilledema, CN abnormalities, flaccid paralysis, sz, ataxia, tremor, involuntary movements

**Evaluation**
- CBC (↓ WBC, ↓ lymphocytes, anemia), Chem (↓ Na), ↑ LFTs; ↑ lipase, viral IgM Ab
- CSF: Mild ↑ protein, mild ↑ leukocyte, nl glucose, serologies
- Brain MRI may be normal or show signal abnormalities in brainstem, basal ganglia, thalamus, anterior spinal cord

**Treatment**
- Supportive care, airway management, resuscitation
- Limited evidence for interferon & IVIG in case series & reports

**Disposition**
- Admit for supportive tx, may need rehabilitation from neuro cx

**Pearls**
- *Flavivirus* transmitted by several types of mosquito to horses, dogs, birds; crosses the blood–brain barrier to infect nervous system
- Has been reported throughout the world
- Excellent prognosis unless elderly or w/ other comorbid factors

**Eastern Equine Encephalitis**
*(MMWR. 2006;55:697)*

**History**
- Outdoor exposure to area of outbreak in summer or early fall
- Fevers, chills, malaise, weakness, HA, myalgias; progression to confusion, coma, N/V

**Findings**
- Similar to any other encephalitis; fever, tachycardia, tachypnea
- Neuro: Papilledema, sz, nuchal rigidity, focal neuro abn, CN abnormalities, spastic paralysis

**Evaluation**
- CBC (↑ WBC), Chem (↓ Na), serologies (IgM), viral isolation from CSF, blood, tissue
- Head CT: Punctuate/intraventricular hemorrhage, focal edema, meningeal enhancement
MRI, LP: CSF shows ↑ protein, ↑ RBC, ↑ WBC

**Treatment**
- Supportive care, airway management, resuscitation, corticosteroids, & anticonvulsants

**Disposition**
- Admit, likely to ICU; will need extensive rehab

**Pearls**
- *Arbovirus* transmitted subcutaneously by mosquito, birds serve as primary reservoir; virus causes acute inflammatory process mainly involving meninges
- Primarily found in North America (east of MS river; MI, MA, NY, NJ, NC, SC, FL, LA, GA); wooded areas near freshwater swamps, marshes; less commonly Central/South America
- Poor prognosis: 33–70% mortality in a few days, 90% morbidity, only 10% fully recover

**Chikungunya** *(Lancet. 2012;379:662)*

**History**
- IP 1–12 d. Sudden onset fever with joint pain, HA, photophobia, rash

**Findings**
- Polyarthralgia (can last months–years), joint swelling, fever, transient maculopapular rash

**Evaluation**
- CBC, viral PCR, serologies, r/o other possible culprits (eg, dengue)

**Treatment**
- NSAIDs for joint pain, supportive care

**Disposition**
- Admit as needed for supportive care

**Pearls**
- Caused by alphavirus transmitted by *Aedes* mosquitos
- Joint pain can last years
- Found in tropical/subtropical regions (African, Indian Ocean Islands, Asia)

**Zika Virus Disease** *(NEJM. 2016;374:1552)*
History
- Asymptomatic or mild Dx (fever, arthritis/arthralgia, rash, conjunctivitis, HA, myalgia)

Findings
- Fever, macular or popular rash, conjunctivitis

Evaluation
- Consider CBC, viral testing

Treatment
- Supportive care

Disposition
- Home

Pearls
- Caused by Flavivirus transmitted by Aedes mosquitoes
- Found in Southern US, central America, South America, Southeast Asia
- Temporal & geographic relationship with neurologic complications in adults such as Guillain–Barré syndrome, meningoencephalitis, as well as association with birth defects such as microcephaly. (Lancet. 2016;388:898)

BIOTERRORISM

(NEJM. 2015;372:954)

Background
- Characterized by low visibility, high potency, accessibility, easy delivery
- Only small amount of agent needed to kill large numbers of people
- Only plague, smallpox, & viral hemorrhagic fevers spread from person to person

Approach
- Take protective measures: Universal precautions w/ HEPA filter masks, decontaminate pt including remove clothing, shower w/ soap & water
- Isolation (negative pressure room) of affected, proper disposal of corpses
ANTHRAX (Bacillus Anthracis)

History
- Contact w/ infected goats, sheep, cattle, horses, swine, 1–6 d incubation period
- Most commonly cutaneous infection, also respiratory or GI; not human to human
- Fever, malaise, HA, cough, weakness, SOB, pruritus, N/V, diarrhea, abd pain
- Less likely than influenza to have sore throat or rhinorrhea

Findings
- Dependent on route of inoculation
- Cutaneous (most common): Incubation 1–12 d; starts as small papule → vesicle containing serosanguineous fluid (1–2 d) → vesicle rupture leaves painless necrotic lesion w/ surrounding edema → massive edema
  - Ulcer base develops 1–5 cm black eschar; after 2–3 wk separates & leaves scar
- Inhalational: Incubation 1–6 d; initial nonspecific sxs & cough × 2–3 d → sudden onset respiratory distress (dyspnea, stridor, cyanosis, ↑ CP, diaphoresis) → rapid onset shock & death in 24–36 h
- GI: From ingestion of infected meat; incubation 2–5 d; local oral/tonsillar ulcer, dysphagia & respiratory distress → abd pain, hematemesis, massive ascites, diarrhea
- Injectional: Characterized by skin lesions seen in “skin popping” drug users, may progress rapidly & require surgical debridement or may disseminate.

Evaluation
- Blood cultures; Gram stain or culture confirms cutaneous anthrax, serologies, rapid antianthrax antibody test can be performed w/i 1 h
- Difficult to diagnose inhalational or GI anthrax
- CXR (inhalational): Mediastinal widening, pleural effusion, e/o ARDS

Treatment
- Early abx for cutaneous Dx: PCN, doxycycline, ciprofloxacin IV. Multiple abx if systemic/extensive dz.
- Raxibacumab injection recently FDA approved for inhalational anthrax
Prophylaxis: Ciprofloxacin or doxycycline PO; anthrax vaccine
Corticosteroids may be useful in severe edema, meningitis

Disposition
Consider admission based on clinical findings

Pearls
Large, aerobic, gram-positive, spore-forming, nonmotile, pyogenic B. anthracis
Found in animals in South & Central America, Southern & Eastern Europe, Africa, Asia, Caribbean, Middle East
Death from respiratory failure, overwhelming bacteremia, septic shock, meningitis
Mortality variable: Cutaneous <1%, inhalational 45–92%, GI 25–60%, injectional 34%

---

**PLAQUE (YERSINIA PESTIS)**

History
- Contact w/ rat flea; 99% cases in SE Asia (Vietnam), rarely Southwest United States
- Acute onset high fevers, LAD, myalgias, cough, SOB, CP, hemoptysis, sore throat, GI sx

Findings
- Bacilli spread to lymph nodes → supportive lymphadenitis, producing bubo → spread to other organs (spleen, liver, lungs, skin) & septic shock if untreated
- Bubonic (85–90%): Incubation 1–8 d; buboes emerge in groin, axilla, or cervical regions w/ f/C/HA, N/V, AMS, cough → buboes visible in 24 h, severely painful
- Septicemia (10–15%): Result of hematogenous dissemination of bubonic plague
- Pneumonic (1%): From inhalation of aerosols or hematogenous dissemination; productive cough w/ blood-tinged sputum, rales, decreased breath sounds

Evaluation
- Presence of painful bubo; Gram stain of bubo aspirate; blood, sputum, & CSF cultures, lymph node aspiration
CXR (pneumonic): Bilateral alveolar infiltrates, consolidation

Treatment
- Isolate pts for 1st 48 h after tx; if pneumonic plague, isolate for 4 d
- Levofloxacin recently approved
- Streptomycin 15 mg/kg IM BID × 10 d ± doxycycline 200 mg IV × 1
- Alternative regimens: Chloramphenicol, gentamicin, Bactrim, ciprofloxacin
- Septicemia plague: Same as for other causes of sepsis
- Prophylaxis: Doxycyclin or ciprofloxacin PO × 7 d; use insecticides, reduce rodent populations

Disposition
- Admission, isolation

Pearls
- Y. pestis: Gram-negative nonmotile nonsporulating coccobacillus; can remain viable for days → weeks in water, moist soil, grain, buried bodies; reservoir: Rodents
- Mortality variable: Untreated bubonic 50%, Septic/pneumonic ~100%; tx reduced mortality to 10–15% overall

SMALLPOX (VARIOLA)

History
- High fever, HA rigors, malaise, myalgias, vomiting, abd pain, back pain, rash

Findings
- Virus multiplies in respiratory tract
- Incubation 10–14 d, spreads hematogenously → regional lymph nodes, blood vessels → skin changes
- 2 types: Major (30% mortality), minor (<1% mortality)
- 2–3 d after initial sxs, exanthema on face, hands, forearms → trunk & lower extremities
- Skin exanthem: Macules → papules (day 2) → vesicles (day 5) → umbilicated pustules (day 8); pustules form scabs after 8–14 d; death in 2nd wk from toxemia (mortality 25%)

Evaluation
Clinical Dx; centrifugal distribution, lesions all in same stage of development, PCR

**Treatment**
- Isolation, hemodynamic support, skin care, vaccination w/i 4 d of exposure (after fever, before rash)

**Disposition**
- Isolation × 17 d; pts most infectious on day 3–6 after onset of fever, remain infectious until all scabs separated

**Pearls**
- Variola virus: Highly infectious by aerosol, environmentally stable, prolonged infectivity
- Transmitted through respiratory droplets, bodily fluids
- Last occurrence in Somalia in 1977; routine vaccination stopped in 1972

---

**BOTULISM**

**History**
- 6 H after inhalation pt would have descending paralysis, CN dysfunction (diplopia, dysphagia, ptosis) progresses to ventilatory failure

**Evaluation**
- Clinical Dx, confirmed with mouse bioassays through culture

**Treatment**
- Antitoxin (equine derived), available exclusively from CDC
- Respiratory support, mechanical ventilation

**Disposition**
- ICU

**Pearls**
- Not contagious
- Inhalational or gastrointestinal form could be used as weapon. Other forms occur (infantile, wound, iatrogenic).
ALTERED MENTAL STATUS

Approach

- **Definition:** Any transient or fixed change in cognition &/or arousability including but not limited to disorientation, memory impairment, behavioral changes, hallucinations
- “AMS” can describe a wide spectrum of clinical severity & be 2/2 diverse causes (see table); encompasses mild confusional states → delirium → coma, or dementia
- **Approach:** Dictated by clinical severity of AMS; if unconscious or severely altered:
  - Immediate IV access, telemetry, ABCs, O₂ for hypoxia (caution if hx severe COPD)
  - Bedside glucose measurement: If low, give immediate 1–2 amps D₅₀W
  - If concern for narcotic o/d or h/o opiate meds: Naloxone 0.4–2.0 mg IV/IM/IN
  - If h/o ETOH abuse or malnutrition: empiric thiamine 100 mg IV (can give D₅₀W first if hypoglycemia; replete thiamine before prolonged dextrose 2/2 risk of Wernicke’s)
- **History:** Start by assessing baseline MS, degree of change, acuity/timing of change, any circumstances surrounding AMS (Δ meds, intoxication/substance use, trauma), PMH
  - Eyewitness accounts helpful: Contact eyewitness if not present with patient
- **Exam:** Assess for focal neurologic sxs (if present, consider CVA, ICH, space-occupying lesion), pupil exam (toxidrome, ↑ ICP/herniation), skin exam (diaphoresis may suggest tox; dehydration may suggest lyte d/o), asterixis (CO₂ or NH₃ excess), clonus
- **Evaluation:** All patients should get CBC, Chem 10, LFTs, UA, Tox screen, ECG, ± hcg; Consider CXR (esp if unable to give hx), TSH, VBG, NH₃, Drug levels, CO (esp if unwitnessed at home), Head CT, LP
### Organic Causes of Acute Altered Mental Status

<table>
<thead>
<tr>
<th>Category</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial</td>
<td><strong>Ischemia</strong>: CVA (can cause AMS if large ± swelling, brainstem)</td>
</tr>
<tr>
<td></td>
<td><strong>Hemorrhage</strong>: Epidural (if trauma), SDH (can be atraumatic), SAH</td>
</tr>
<tr>
<td></td>
<td>(traumatic or aneurysmal), IPH (2/2 neoplasm, HTN, AVM)</td>
</tr>
<tr>
<td></td>
<td><strong>Seizure (complex)</strong>: Sz, post-ictal, or consider nonconvulsive status</td>
</tr>
<tr>
<td></td>
<td><strong>Space-occupying lesion</strong>: Neoplasm, Abscess (esp IVDU, HIV)</td>
</tr>
<tr>
<td></td>
<td><strong>Other</strong>: HTN encephalopathy, PRES, concussion (traumatic), diffuse axonal</td>
</tr>
<tr>
<td></td>
<td>injury (traumatic), anoxic brain injury (esp if s/p ↓ O₂ or ↓ BP)</td>
</tr>
<tr>
<td>Metabolic</td>
<td><strong>Metabolic</strong>: Acidosis, ↑ CO₂, ↓ O₂, electrolyte Δ (Na, Ca), uremia, NH₃</td>
</tr>
<tr>
<td></td>
<td><strong>Endocrine</strong>: ↓ glucose, ↑ glucose (HHS, DKA), adrenal, thyroid (↑/↓)</td>
</tr>
<tr>
<td>Infectious</td>
<td><strong>Nutritional</strong>: Wernicke’s, B₁₂ deficiency</td>
</tr>
<tr>
<td></td>
<td><strong>Substances</strong>: Depressants: Opioids, antipsychotics, sedative-hypnotics (eg,</td>
</tr>
<tr>
<td></td>
<td>benzos), antihistamines, anticholinergics, alcohols (inc. toxic alcohols)</td>
</tr>
<tr>
<td></td>
<td><strong>Stimulants</strong>: Sympathomimetic agents, hallucinogens, w/d states</td>
</tr>
<tr>
<td>Medications</td>
<td>Psychotropic meds most often at fault, but always consider polypharmacy</td>
</tr>
<tr>
<td></td>
<td><strong>Serotonin syndrome</strong> (SSRI, NSRI, Linezolid, triptans, dextromethorphan,</td>
</tr>
<tr>
<td></td>
<td>meperidine, methadone, tramadol, ecstasy)</td>
</tr>
<tr>
<td></td>
<td><strong>Neuroleptic malignant syndrome</strong> (antipsychotics)</td>
</tr>
<tr>
<td>Trauma</td>
<td>Burns, electrocution, systemic inflammatory response, fat embolism, occult</td>
</tr>
<tr>
<td>Environment</td>
<td>trauma (eg, abuse/neglect)</td>
</tr>
<tr>
<td></td>
<td>CO, cyanide</td>
</tr>
</tbody>
</table>

### Physical Exam Clues in the Patient with Altered Mental Status

<table>
<thead>
<tr>
<th>Exam Finding</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>VS</td>
<td>↑ Temp Infxn, thyroid storm, adrenergic stim (drug o/d, w/d), SS/NMS</td>
</tr>
<tr>
<td></td>
<td>↓ Temp Environmental, hypothyroidism, sepsis</td>
</tr>
<tr>
<td></td>
<td>↑ RR Metabolic acidosis (DKA), stimulant, aspirin OD</td>
</tr>
<tr>
<td></td>
<td>↓ RR Narcotic o/d, CNS insult</td>
</tr>
<tr>
<td></td>
<td>↑ HR Fever, sepsis, dehydration, thyroid storm, OD (stimulant, TCA,</td>
</tr>
<tr>
<td></td>
<td>aspirin, theophylline, anticholinergic), acidosis</td>
</tr>
<tr>
<td></td>
<td>↓ HR Heart block, ingestion (BB, CCB, digoxin), ↑ ICP</td>
</tr>
<tr>
<td></td>
<td>↑ BP HTN emergency, preeclampsia, adrenergic stim (drug o/d, w/d),</td>
</tr>
<tr>
<td></td>
<td>PRES, ↑ ICP, pain</td>
</tr>
<tr>
<td></td>
<td>↓ BP Shock, sepsis, hemorrhage, toxins, GIB, adrenal crisis</td>
</tr>
</tbody>
</table>
Delirium

- **Definition**: Acute state of temporary or fluctuating disturbance of consciousness (eg, impaired cognition, perception disturbances, reduced attention, hypo- or hyperactivity) that is caused by an organic medical condition or medication/drug (ie, not psychiatric)
- Can have many causes (see table above); w/u & tx dependent on causal etiology
- Delirium (vs. dementia or psych) suggested by: Age <12 or >40, visual hallucinations (vs. auditory), acute onset, exam abnormalities
- Dispo: Admit all pts not at baseline MS or with recent unexplained AMS; delirium in ED may independently predict 6-mo mortality (*Ann Emerg Med* 2010;56(3):244–252)

Dementia

- **Definition**: Progressive, unremitting decline in cognitive function due to a variety of causes (Alzheimer’s, vascular, Lewy-body, etc.), classically marked by decline in short- & eventually long-term memory, but advanced cases may have behavioral chgs (hypo- or hyperactive, agitation) or even nonverbal.
- Much more subacute than delirium, though can predispose pts to delirium from otherwise occult pathology (eg, UTI, PNA) due to poor cognitive reserve; 50% of elderly pts w/ delirium have some degree of underlying dementia (*Ann Emerg Med* 2010;56(3):261–269)
- Important to screen for elder abuse (e/o physical trauma, neglect); EA is underdiagnosed but especially important in pts w/ dementia 2/2 inc risk of caregiver fatigue

<table>
<thead>
<tr>
<th>Eyes</th>
<th>Miosis</th>
<th>Opioid ingestion, clonidine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mydriasis</td>
<td>Sympathomimetic or anticholinergic toxidrome</td>
<td></td>
</tr>
<tr>
<td>Asymmetric</td>
<td>Intracranial process w/ mass effect or herniation</td>
<td></td>
</tr>
<tr>
<td>Papilledema</td>
<td>↑ ICP</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dementia Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>System</strong></td>
</tr>
<tr>
<td>Degenerative</td>
</tr>
<tr>
<td>Vascular</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
</tbody>
</table>
**Inflammatory**
- Lupus, demyelinating dz

**Neoplastic**
- Primary CNS tumor, metastatic dz, paraneoplastic syndromes

**Traumatic**
- TBI, SDH, anoxic brain injury

**Toxic**
- Alcohol, medications, heavy metals

**Metabolic**
- B$_{12}$/folate deficiency, thyroid, Wilson dz, lipid storage dz

**Psychiatric**
- Depression

**Hydrocephalus**
- NPH, noncommunicating hydrocephalus

## HEADACHE

### Approach
- Must differentiate life-threatening HA (minority) from benign HAs (majority)
- **History:** Essential to describe timing/acuity of onset, location, quality, radiation/movement, severity, & associated sx (fever, photophobia, emesis, vision chg, eye pain, neck pain, focal neuro sx, chg in speech or cognition, sinus congestion), circumstances surrounding onset (trauma, med chgs, environment)
- **PMH:** Always ask about HA hx (if present: obtain detailed info regarding how current HA is similar/different), IVDU, immunosupp, & current/recent meds (eg, A/C)
- **Red flags** requiring neuroimaging: Sudden/rapid onset (<1 h to peak), exertional onset, worst of life, AMS, 1st severe HA >age 35, fever, neck stiffness, immunosupp, daily HA, no similar prior HAs, abnl neuro exam, meningismus, papilledema

### Headache Differential

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary HA</td>
<td>Migraine, tension, cluster, trigeminal neuralgia, analgesia rebound</td>
</tr>
<tr>
<td>Trauma</td>
<td>ICH (SAH, SDH, EDH, IPH), postconcussive syndrome</td>
</tr>
<tr>
<td>CNS Infection</td>
<td>Meningitis, encephalitis, abscess</td>
</tr>
<tr>
<td>Vascular</td>
<td>HTN emergency, aneurysm/AVM growth, cerebral venous sinus thrombosis, carotid/vertebral art. dissection, temporal arteritis, preeclampsia; HA rarely presenting complaint w/ CVA</td>
</tr>
</tbody>
</table>
**Neoplastic Malignancy** (primary or metastatic), benign (eg, meningioma)

- CSF d/o (↑↓ ICP): Hydrocephalus, pseudotumor cerebri, dural leak/post-LP (↓ CSF)
- Otolaryngologic: Sinusitis, TMJ syndrome, mastoiditis
- Ophthalmologic: Glaucoma, Myopia/Presbyopia/Hyperopia/Astigmatism
- Environmental: CO poisoning (Ch. 10), noxious aerosols

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**PRIMARY HEADACHE SYNDROMES**

**Migraine Headache & Variants**

**History, Physical Exam, & Evaluation**

- HX: Slow in onset (over hours), unilateral throbbing or pulsatile, often w/ N/V (>50%), photophobia; visual or sensory aura/prodrome may precede HA (15%); duration 4–72 h
- Migraine variants discussed below
- Classically have migraine hx; beware of assuming migraine if first time & age >35
- EXAM: NL neuro exam (except in migraine variants)
- DX: No studies or consults routinely indicated unless need to exclude other cause (eg, CT, LP, MRI) as in the case of severe or prolonged sx (see *Migraine Variants*)

**Treatment**

- Abortive: Most effective if given w/i 15 m of onset; often involves combo tx w/ IVF, NSAIDs, APAP, antiemetic (check QTc; common options incl prochlorperazine, metoclopramide; give w/ diphenhydramine to ↓ extrapyramidal sx); additional options include triptans & DHE (both c/i in preg & CAD), & dexamethasone
- Prophylaxis: Indicated if >2/mo, duration > 24 h, major lifestyle disruption, failure of abortive tx; Options include AEDs, BB, TCAs, CCBs, SSRIs, behavioral/environmental chgs

**Disposition**

- Most pts able to return home within hours; may need Observation Unit if protracted

**Migraine Variants**

- Migraine variants are rare but can resemble other concerning conditions; often strong h/o similar events in the past, but if not,
requires involved w/u to r/o other more serious dx

- **Hemiplegic migraine**: HA a/w hemiplegia (± paresthesias); hemiplegia may resolve w/i hrs or persist days; HA may be subtle but classically pts have h/o similar prior sx
- **Basilar migraine**: HA w/ dizziness/vertigo, ± ataxia, N/V, tinnitus, AMS
- **Abdominal migraine**: Paroxysmal mid-abd pain a/w N/V, often w/o HA; strong h/o prior similar episodes & +FH; ddx inc. cyclic vomiting syndrome; more common in Peds
- **Ocular migraine**: Gradual loss of vision in one eye 2/2 transient vasospasm of retinal arteries; often h/o prior similar episodes & +FH; avoid triptans & DHE
- **Status migrainosus**: Migraine HA >72 h

**Pearl**

- Migraine HA are independent RF for ischemic CVA (RR 1.64) & silent CVA (avoid triptans/DHE) (*Neurol Sci 2017;38(1):33–40*)

### Tension Headache

**History, Physical Exam, & Evaluation**

- **HX**: Dull, aching or throbbing “vice-like” pressure HA w/ gradual onset, bilateral frontal &/or occipito-nuchal; rarely w/ N/V or prodrome; duration 30 min to 7 d; a/w insomnia, stress, anxiety, or depression
- **EXAM**: Normal neuro exam, no true photophobia
- **DX**: No studies or consults routinely indicated (unless need to exclude other cause)

**Treatment**

- NSAIDs or APAP, neck massage & heat, relaxation techniques, *not* narcotics

**Disposition**

- Most patients can go home within hours; if chronic HA, refer to HA specialist

### Cluster Headache

**History, Physical Exam, & Evaluation**

- **HX**: Sudden onset unilateral, paroxysmal, sharp, stabbing severe temporal/periorbital HA that may awaken from sleep; ± ipsilateral lacrimation, flushing, rhinorrhea or nasal congestion, conj injection or Horner syndrome (30% of pts); occur in clusters of short (15–180 min) episodes (1 QOD to >8/d) for up to 6–8 wk; more common in men
- **EXAM:** Normal neuro exam or +Horner’s; may have +lacrimation, flushing, conj injection
- **DX:** No studies or consults routinely indicated (unless need to exclude other cause)

**Treatment**
- **Abortive:** High-flow O₂ (12–15 L/min) by mask, sumatriptan (CI in pregnancy or CAD), intranasal lidocaine, NSAIDs.
- **Prophylaxis:** Prednisone 60 mg ×10 d then taper, ± verapamil or valproic acid

**Pearl**
- Make sure to distinguish from acute angle-closure glaucoma

---

**Trigeminal Neuralgia**

**History, Physical Exam, & Evaluation**
- **HX:** Unilateral paroxysmal pain in sensory distribution of CN V, commonly involves the maxillary (V2) or mandibular (V3) branches; ± brief facial spasm or tic (“tic douloureux”); may be triggered by light touch or vibration, shaving, face washing, chewing
- **EXAM:** No e/o CN dysfxn or other neurologic abnlty
- **DX:** Can be treated w/o w/u if characteristic hx; MRI if atypical features present (neuro deficit, age <40). Refer to neurology for outpt w/u.

**Treatment**
- Carbamazepine 100 mg BID, increase by 200 mg/d up to 1200 mg/d

**Pearl**
- Most common cause is compression of the nerve root by an aberrant vessel

---

**Acute Sinusitis**

**Overview**
- **Definition:** Inflammation of the paranasal sinuses, usually viral or allergic, though sometimes bacterial superinfxn (S. pneumo, nontypable H. influenzae, M. catarrhalis)
- Dangerous pathogens: Pseudomonas (esp HIV, CF, s/p instrumentation), invasive fungal sinusitis (Rhizopus) or mucormycosis (DM, immunosupp); require special tx

**History, Physical Exam, & Evaluation**
HX: Consider with positional HA, worse bending forward or head movement; pts often have mucopurulent d/c, postnasal drip, sinus pressure, ± tap tenderness; may be afebrile (if +fever, more likely bacterial); progresses over 7–10 d
- If no resolution w/i 7 d, suggests bacterial dz
EXAM: May have pharyngeal erythema from postnasal drip, ± tap tenderness
DX: Clinical. Imaging not routinely indicated; CT has high Se but low Sp.

Treatment
- Supportive (analgesics, antipyretics, decongestants, antihistamines if allergic)
- Decongestants: Neo-Synephrine nasal spray TID × 3 d, Afrin nasal spray BID × 3 d
- Abx not routinely indicated: Reserve for pts w/ sxs >7 d, worsening sxs, fever, purulent d/c, or high risk for severe infection or cx
  - First-line: Amoxicillin 500 mg PO TID × 10 d, TMP–SMX, or azithromycin
  - If no improvement: Amoxicillin–clavulanate, fluoroquinolone, clindamycin

Disposition
- Discharge w/ PCP follow-up; Consider admx if high fever, immunosupp, poor f/u

Pearl
- Sphenoid/ethmoid sinusitis is less common than maxillary sinusitis but has significant potential cx (eg, orbital cellulitis, cavernous sinus thrombosis)

Hypertensive Headache

History, Physical Exam, & Evaluation
- HX: Untreated HTN or other precipitants (pregnancy, drug use, serotonin syndrome)
- EXAM: BP often >240/140 (unlikely w/ DBP <120); May have papilledema, encephalopathy, ± focal neuro abnormalities or sz
- DX: Assess for e/o end-organ damage (HTN emergency): Head CT (r/o ICH, edema), ECG, e/o aortic injury, pulm edema, renal failure
Cerebral Venous Sinus Thrombosis

Overview (NEJM 2005;352(17):1791–1798)

- Pathophysiology: Thrombosis of the sinuses of the brain (e.g., sagittal, straight, occipital, transverse) with or without thrombosis of the cortical veins of the brain
- Sinus thrombosis impairs CSF absorption causing ↑ ICP (e.g., HA, AMS)
- Cortical vein thrombosis causes venous infarction & localized injury (e.g., focal deficits)
- Because of cerebral injury, secondary hemorrhage may develop (40% cases)
- Prothrombotic RF (trauma, hypercoagulable state [esp preg]) present in 85%; other causes include post-LP (2/2 downward traction on cortical veins from pressure chg), sinusitis (cavernous thrombosis)

History, Physical Exam, & Evaluation

- HX: HA present >90%, often gradual over days but can be sudden; ± N/V, vision chgs, focal neuro deficits (see above); Assess PMH for prothrombotic RFs
- EXAM: Assess for papilledema, focal neuro deficits; cavernous sinus thrombosis will have CN III/IV/VI compromise, periorbital chemosis/edema, & ↓ vision ipsilaterally
- DX: CBC, PT/INR, PTT, Upreg, ± D-dimer; MRI/MRV more Se than CTV but balance accuracy with urgency & desire to r/o other diagnoses (e.g., SAH, IPH)
  - D-dimer may help r/o CVST if low pre-TP (weighted Se 94%, Sp 90%)

Treatment

- Anticoagulation (heparin) preferred, often even in presence of hemorrhagic infarcts
- Endovascular thrombolysis can be used, but often reserved for those w/ worst prognosis
Disposition
- Admission to neurology; may warrant ICU care if e/o hemorrhagic conversion or AMS

**Temporal Arteritis (Giant Cell Arteritis)**

**Overview**
- **Definition:** Granulomatous inflammatory vasculitis of medium/large arteries occurring in pts > 50 y (peak incidence 70–80 y); largely affecting branches of ECA, vertebral, distal subclavian, axillary, arteries, & thoracic aorta; causes ischemic sx by vessel occlusion

**History, Physical Exam, & Evaluation**
- **HX:** Unilateral HA, jaw/tongue claudication, malaise, low-grade fevers, visual impairment
- **RFs:** Age >50 y/o (90% >60 y/o), F > M, h/o PMR (50% of pts)
- **EXAM:** May have tenderness over temporal art. (↓ Se) or ↓ visual acuity (↓ Se)

**Evaluation** *(NEJM 2014;371:50–57)*
- ↑ ESR (Se ~84%), ↑ CRP (Se ~86%), combined Se of ESR & CRP may be >95%, but poorly specific in pts w/ other causes for elevation
- Temporal art. bx: high Se for even low-levels of inflammation *if present at site of bx* (requires 1.5–2.0 cm segment); given focality of dz, may need repeat bx or imaging if high pre-TP & first-bx neg
- CTA &/or MRA: Not used to make dx, but may serve as adjunct if -bx &/or assess extent of dz if +bx

**Treatment**
- Prednisone 1 mg/kg/d (if vision sx, do not wait for bx results) ~2–4 wk, then prolonged taper
- Consult neurology, ophthalmology, rheumatology if concern for dx & to arrange f/u

**Disposition**
- Admit for visual deficits; Can be discharged on steroids w/ follow-up

**Pseudotumor Cerebri (Idiopathic Intracranial HTN)**

**Overview**
Pathophysiology: Set of sx of unclear etiology; due to obstructed venous/CSF outflow rather than ↑ CSF production

- **HX:** Gradual onset, global, daily/constant HA (>90%) or retrobulbar pain, ±, N/V, ↓ vision (~70%; may be transient), intracranial noises (~60%); sx can be worst in morning
- **RFs:** Young obese females, recent weight gain; some meds (tetracyclines, retinoids)
- **EXAM:** NL neuro exam (except possible CN VI palsy), check vision & for papilledema
- **DX:** Can be clinical dx (sx, papilledema); LP for opening pressure (>25 cm H₂O in lateral decubitus) confirms dx; Neuroimaging may be normal (or show swelling of optic discs)

**Treatment**
- Neuro consult (± ophthalmology) if new dx or refractory to tx
- Weight loss remains most effective treatment
- Diuretics to decrease ICP: Acetazolamide 1 mg PO QD
- May need repeated high-volume LPs; refractory cases may need VP shunt
- Use of steroids controversial & may worsen weight gain

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**Dural Leak/Post-LP Headache**

**Overview**
- **Definition:** HA caused by ICP caused by loss of CSF from recent dural injury (eg, due to LP, myelogram, spinal anesthesia, vigorous coughing)

**History, Physical Exam & Evaluation**
- **HX:** Occipital HA radiating to shoulders/neck, worse with sitting/standing (± alleviated w/ supine); Worsened by activities that ↑ ICP (eg, coughing, sneezing, Valsalva); Usually present 48–72 h postdural injury (but can be >1 wk); ± N/V, LH, photophobia, tinnitus
- **EXAM:** Nonfocal neuro exam; HA improved w/ lying flat, worse w/ sitting up
- **DX:** None spec; evaluate for other causes of HA, if indicated

**Treatment**
- As much as 8% will resolve w/o tx; tx indicated for severe or prolonged
Epidural blood patch (clot forms to seal dural defect): 70–98% success
- Methylxanthine derivatives (caffeine IV, aminophylline) may be helpful, limited data
- Surgical closure of dural gap is last-resort effort if blood patch &/or other options fail

**Pearls**
- Minimize risk of post-LP HA w/ small caliber spinal needle (24–27G), bevel alignment w/ dural fibers, atraumatic needles, minimized number of attempts
- Severe cases can precipitate Sz & SDH (2/2 ↓ ICP → brain pulled → bridging vein strain)

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**INTRACRANIAL NEOPLASM**

- **Definition:** Any of a spectrum of neoplasms, each with distinct biology, epidemiology, natural history, & management, & prognosis; often cause acute sx due to mass effect of tumor, vasogenic edema, or secondary hemorrhage on neural tissue, or sz
- Tumors either extraparenchymal (meningioma, pituitary neoplasm) or intraparenchymal
  - Intraparenchymal most commonly glioma (eg, oligodendroglioma, mixed glioma, astrocytoma), primary CNS lymphoma, or metastatic non-CNS primary cancer

**History, Physical Exam, & Evaluation**
- **HX:** Subacute onset, often daily morning HAs (2/2 ↑ ICP), ± N/V, focal deficits, chgs in personality or speech, sz; alleviation w/ NSAIDs/APAP does not help r/o dx
- **EXAM:** Look for signs of herniation:
  - Uncal (most common): ↓ mental status, blown unilateral pupil, decerebrate posturing
  - Central: AMS, yawning, Cheyne–Stokes breathing, miosis. Decorticate → decerebrate.
  - Tonsillar (posterior): Bradycardia, coma, respiratory arrest
  - Cushing reflex (due to ↑ ICP): HTN, bradycardia, irregular breathing
- **DX:** Neuroimaging w/ CT, usually followed by MRI (w/ contrast); In
those suspected w/ pituitary tumors, check lytes, cortisol, TSH
• Treatment & prognosis depend on tumor phenotype & grade

Treatment (glioma)
  ‣ Dexamethasone 4 mg TID for mass effect/minimize edema
  ‣ Antiepileptic treatment
  ‣ Consult NSGY (consideration of resection), Neuro-oncology

Disposition
  ‣ Admit all patients w/ new dx of intracranial neoplasm; or transfer to facility w/ NSGY
  ‣ Prognosis heavily dependent on phenotype & grade (mos to yrs)

CNS INFECTIONS

Meningitis
Overview
  ‣ Definition: Inflammation of the meninges overlying the brain due to either infectious (bacterial, fungal, viral) or noninfectious etiology; sx & tx differ widely based on etiology
  ‣ Bacterial meningitis rare in developed nations; common bacteria include S. pneumoniae, N. meningitidis, H. influenzae type b, L. monocytogenes (infants, elderly, pregnant), staph (VPS, trauma, NSGY); seeding of subarachnoid space 2/2 hematogenous spread (eg, from resp tract) or direct spread (eg, sinusitis, acute OM)
  ‣ Causes of noninfectious “aseptic” meningitis include drugs (eg, antimicrobials, vaccines, NSAIDs), inflammatory dz (SLE, Behcet); rarely malignancy can p/w leptomeningeal dz

History
  ‣ Bacterial: Typically acute (<1 d), high-grade fever, HA, nuchal rigidity, ill appearing, AMS
  • RFs: Extremes of age, immunosupp (esp HIV, steroids, MM/blood ca), crowded living environment (dorms, shelters), splenectomy, ETOH abuse / cirrhosis, IVDU, recent illness (esp sinusitis/OM), dural defect (recent trauma, surgery; congenital, VPS)
  • Classic triad for bacterial etiology: Neck stiffness, fever, AMS (3 of 3 present <50%, 2 of 3 present >95%) (Lancet 2016;388(10063):3036–3047)
Viral: Typically subacute (1–7 d), also w/ HA, fever, photophobia; unless HSV, usually normal mental status; HSV usually w/ AMS
Fungal/TB: Subacute (>1 wk), HA, low-grade fever, weight loss, night sweats, ± AMS

Physical Exam
- May have nuchal rigidity, disorientation/AMS, photophobia
  - Brudzinski sign (hip flexion elicited by passive neck flexion) & Kernig sign (inability or reluctance to extend knee when hip is flexed to 90):
    High Sp, but Se only 5%
- Petechial/purpuric rash suggests meningococcus (N. meningitidis)
- Expect subtle presentation in elderly or immunocompromised pts; may be AMS only

Evaluation
- LP is gold standard for dx but should not delay abx if high pre-TP for bacterial/HSV
  - See table below for indications for CT prior to LP
  - LP tubes: (1 & 4) cell count & diff, (2) gluc & protein, (3) Gram stain, cx ± HSV PCR
  - Gram stain Se depends on etiology: S. pneumoniae (Se >90%), H. influenzae (75%), N. meningitidis (50%), L. monocytogenes (33%); GS & Cx yield may ↓ by 40% if performed after abx, improved by PCR (Clin Infect Dis 2004;39(9):1267; Lancet 388:3036–3047)
- BCx, full infectious w/u (CBC, CXR, UA); thrombocytopenia suggests meningococcus

Treatment (Clin Infect Dis 2004;39(9):1267)
- Respiratory precautions if suspect bacterial
- Empiric abx based on suspected etiology (see table); acyclovir 10 mg/kg q8h if c/f HSV
- Early steroids may ↓ inflammatory cascade in bacterial meningitis; therefore, if proven or high pre-TP bacterial etiology, give dexamethasone 0.15 mg/kg w/i 20 min of 1st abx (↑ favorable neuro outcomes & ↓ mortality)
- Consult NSGY in all pts w/indwelling hardware (eg, VPS): removal improves tx success
- Postexposure ppx (if +N. meningitidis): Ciprofloxacin 500 mg PO × 1. Ceftriaxone 250 mg IM (peds), or Ceftriaxone 150 mg IM (pregnancy).
- If viral (non-HSV): Supportive care only, prophylaxis not needed
Disposition

- Admit bacterial, HSV, fungal; Non-HSV viral: D/C vs. Observation based on sx severity


<table>
<thead>
<tr>
<th>Indicator</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;60</td>
<td>Any neurologic abnormalities: ↓ GCS*, CN abnlty, abnl visual fields, pronator drift, abnl language (eg, aphasia)</td>
</tr>
<tr>
<td>Immunocompromised</td>
<td></td>
</tr>
<tr>
<td>History of CNS/neurologic dz</td>
<td></td>
</tr>
<tr>
<td>Recent seizure w/i 1 wk of presentation</td>
<td>Inability to follow two consecutive commands</td>
</tr>
<tr>
<td>Papilledema</td>
<td>Inability to answer two consecutive questions</td>
</tr>
</tbody>
</table>

*No clear data on precise GCS cut-off; some studies suggest CT for any GCS <13, while others suggest CT only if GCS <8. Others simply use “alert” or “not alert” (NEJM 2001;345:1727–1733).

### Interpretation of LP Results

<table>
<thead>
<tr>
<th>CSF Test</th>
<th>Normal</th>
<th>Implications of Abnormal Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>&lt;5 WBC</td>
<td>Bacterial: Marked ↑ WBC (usually &gt;1000), ↑ PMN</td>
</tr>
<tr>
<td></td>
<td>&lt;1 PMN</td>
<td>Viral: Usually ↑ WBC but &lt;500, mononuclear</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Traumatic tap: if serum WBC nl, expect 1 WBC for every 700 RBC (limited data)*</td>
</tr>
<tr>
<td>RBC</td>
<td>None</td>
<td>↑↑ RBC: traumatic LP (if T1 &gt; T4) or SAH (T1~T4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↑ RBC (&amp; ↑ WBC): Consider HSV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xanthochromia indicates RBCs present 4 h prior</td>
</tr>
<tr>
<td>CSF:Serum Gluc</td>
<td>0.6:1</td>
<td>↓ in bacterial/fungal meningitis or hyperglycemia</td>
</tr>
<tr>
<td>Protein</td>
<td>15–45 mg/dL</td>
<td>↑ in bacterial/fungal meningitis, syphilis, neoplasm, demyelination, bleed (SAH)</td>
</tr>
<tr>
<td>Opening pres.**</td>
<td>&lt;20 mmH$_2$O</td>
<td>↑ in bacterial, fungal or TB</td>
</tr>
<tr>
<td>Gram stain</td>
<td>Negative</td>
<td>Positive in 80% of bacterial meningitis (see above)</td>
</tr>
</tbody>
</table>

*Limited data from adult population. In infants 0–60 d, may be safe to use ratio of 1 WBC: 877 RBCs (Ann Emerg Med 2016;pii:S0196-064(16)31223–31229).

**Can be elevated 2/2 noninfectious etiologies as well.
<table>
<thead>
<tr>
<th>Age</th>
<th>Pathogens</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–24 mo</td>
<td>Group B strep, S. <em>pneumo</em>, H. <em>influenzae</em>, N. <em>meningitidis</em></td>
<td>Vancomycin AND Ceftriaxone</td>
</tr>
<tr>
<td>2–50 yr</td>
<td>S. <em>pneumo</em>, N. <em>meningitidis</em></td>
<td>Vancomycin 1 g AND Ceftriaxone 2 g</td>
</tr>
<tr>
<td>&gt;50 yr</td>
<td>S. <em>pneumo</em>, N. <em>meningitidis</em>, P. <em>aeruginosa</em>, L. <em>monocytogenes</em></td>
<td>Vancomycin 1 g AND Ceftriaxone 2 g AND Ampicillin 150 mg/kg/d div q4h</td>
</tr>
<tr>
<td>Surgery/Trauma</td>
<td>S. <em>aureus</em>, S. <em>epidermidis</em>, P. <em>aeruginosa</em></td>
<td>Vancomycin 1 g AND Cefepime 2 g</td>
</tr>
</tbody>
</table>

**Encephalitis**

**Overview**

- **Definition:** Inflammation of brain parenchyma usually due to infection (often viral); rarely autoimmune/paraneoplastic *(Lancet Neurol 2016;15(4):391–404)*
- HSV (5–10% encephalitis) life-threatening (>70% mortality w/o tx) *(BMJ 2012;344:e3166)*

**History & Physical Exam**

- **HX:** Acute onset fever (>90% w/ HSV), HA (>80% w/ HSV), behavior chgs (>70% w/ HSV), hallucinations/altered awareness (>60% w/ HSV), confusion/impaired memory (25% w/ HSV); ± diffuse or focal neuro sx (weakness, ataxia, speech disturbance), seizure, or e/o meningeal involvement (+photophobia, +neck stiffness) *(BMJ 2012;344:e3166)*
  - Absence of fever or HA strongly suggests against the dx of HSV encephalitis
  - HSV encephalitis often preceded by nonspecific viral prodrome (fever, malaise, N/V)
  - Assess for immunosupp, recent travel, tick/mosquito bites
- **EXAM:** AMS (may progress to coma), more likely to have focal neuro deficits than isolated meningitis (may progress to diffuse paralysis &/or ataxia); may have e/o concurrent meningeal involvement *(BMJ 2012;344:e3166)*
  - LP: CSF ↑ WBC, ± ↑ RBC/xanthochromia, ± ↑ protein levels, nl glucose
  - Important to send HSV PCR, but should not defer tx
  - MRI: Imaging modality of choice & helps r/o CI to LP; Se 90% in HSV
encephalitis
› EEG: Se 84%, Sp 32% for HSV encephalitis (may help guide need for AEDs)

Treatment
› If concern for HSV encephalitis: Acyclovir 10 mg/kg q8h IV, AEDs
› If low concern for HSV: Supportive ± anticonvulsants, steroids

Disposition
› Admit if confirmed/suspected HSV, not at baseline (eg, AMS), immunosupp

Abscess

Overview
› Definition: Purulent collections w/i the CNS (intraparenchymal, epidural, subdural, spinal); form by contiguous spread (sinus, dental) or hematogenous seeding (PNA, endocarditis)
› RFs: Immunosupp, IVDU, trauma/surgery, local infxn (mastoiditis, sinusitis, dental), & RFs for systemic infxn (eg, endocarditis, line infxn, bacteremia)
› Certain medical conditions predispose to certain pathogens: HIV (Toxoplasma gondii, M. tuberculosis), solid-organ tpx (aspergillus, candida), post-surgical (staph, gram-neg)

› HX: HA (most common), ± low-grade fever; neuro sx only late in dz; AMS often absent
› Up to 25% of pts may p/w seizures
› Assess for RFs & ROS suggesting possible source infxn
› EXAM: May have nl neuro exam depending on site of abscess & timing of presentation
› DX: ↑ WBC, ↑ ESR, blood cultures. CT w/ contrast (“ring-enhancing” lesion)
› Owing to risk of brain herniation & low Se (25%), LP not routinely performed
› NSGY consultation for diagnostic stereotactic aspiration for cx & decompression
› Antitoxoplasma IgG can confirm toxoplasma dx in HIV pts (no need for aspiration)

Treatment
Although diagnostic NSGY aspiration aims to decompress maximally, therapeutic NSGY aspiration (eg, if pathogen already known) indicated only for large abscesses or those w/ e/o IV abx failure (NEJM 2014;371(5):447–456)

- Early abx (before diagnostic aspiration, esp if acute or severe)
- Low threshold to intubate (may progress rapidly)

**Disposition**

- Admit all pts; may require ICU

### Empiric Antibiotics for Brain Abscess (NEJM 2014;371(5):447–456)

<table>
<thead>
<tr>
<th>Category</th>
<th>Antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard</td>
<td>Ceftriaxone* 2 g AND Metronidazole 500 mg ± Vancomycin 1 g</td>
</tr>
<tr>
<td>Transplant patients</td>
<td>Ceftriaxone* 2 g AND Metronidazole 500 mg ± Vancomycin 1 g AND Voriconazole AND Trimethoprim-Sulfamethoxazole</td>
</tr>
<tr>
<td>HIV-positive patients</td>
<td>Ceftriaxone* 2 g AND Metronidazole 500 mg ± Vancomycin 1 g AND Pyrimethamine AND Sulfadiazine Consider TB tx (INH, Rifampin, Pyrazinamide, Ethambutol)</td>
</tr>
</tbody>
</table>

*Meropenem can be substituted for cephalosporin if allergies present

### SEIZURE

**Overview**

- **Definition:** Spontaneous or provoked abnl synchronous cortical electrical activity; recurrent unprovoked seizures referred to as “epilepsy” (Ann Emerg Med 2014;63(4):437–447)

  - **Simple vs. Complex:** Refers to degree of change of mental status; simple sz cause no chg in mental status, while complex can cause AMS or complete LOC
  - **Partial vs. Generalized:** Refers to location(s) of brain involved & corresponding sx; partial sz are limited to one area of one hemisphere, while generalized are bilateral
  - **Status epilepticus:** >20 min continuous generalized sz activity or continued intermittent sz w/o return to baseline mental status (Ann Emerg Med 2014;63(4):437–447)

- Lifetime risk of nonfebrile seizure is 2–5% (Ann Emerg Med 2004;43(5):605–625)
- May be provoked by numerous etiologies (see table)
### Common Etiologies Provoking Seizure

<table>
<thead>
<tr>
<th>Location</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Ischemia (eg, CVA), ICH, Vascular malformations (aneurysm, AVM), Neoplasm (primary, met), Sinus thrombosis, Trauma, PRES, HTN enceph, inherited conditions (NF, Tub. sclerosis, Sturge–Weber, etc.)</td>
</tr>
<tr>
<td>Infection</td>
<td>Febrile sz (peds), meningitis, encephalitis, brain abscess, HIV OIs, HIV enceph, neurocysticercosis, neurosyphilis, malaria</td>
</tr>
<tr>
<td>Toxic</td>
<td>W/D (ETOH, benzo, barb), OD (sympathomimetic, TCA, anticholinergic, SSRI/NSRI, lidocaine, INH), Caffeine</td>
</tr>
<tr>
<td>Metabolic</td>
<td>↑ ↓ glucose, ↑ ↓ Na, ↓ Ca, ↓ Mg, ↓ O₂, uremia, liver failure, thyrotoxicosis</td>
</tr>
<tr>
<td>Obstetric</td>
<td>Eclampsia</td>
</tr>
<tr>
<td>Environmental</td>
<td>Heat stroke, Stress, Lack of sleep</td>
</tr>
<tr>
<td>Neonatal</td>
<td>CMV, congenital syphilis, rubella, inborn errors of metabolism (eg, PKU)</td>
</tr>
</tbody>
</table>

### Common Types of Seizures

<table>
<thead>
<tr>
<th>Location</th>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalized</td>
<td>Tonic, Clonic, Tonic–clonic</td>
<td>Abrupt LOC, often apneic; Rigid (tonic), rhythmic jerking (clonic), or tonic phase followed by clonic phase (tonic-clonic); Often a/w incontinence, tongue biting, trauma (eg, shoulder dislocation) Followed by post-ictal state of depressed mental status lasting minutes to hours; patients unable to recall event Affects all ages; In older adults, more likely 2/2 focal lesion that rapidly generalizes</td>
</tr>
<tr>
<td>Absence</td>
<td></td>
<td>Abrupt LOC; Staring spells or rhythmic blinking; no incontinence Minimal post-ictal state but patients unable to recall event School-aged children (usually resolves by adulthood)</td>
</tr>
<tr>
<td>Partial (focal)</td>
<td>Simple</td>
<td>Isolated unilateral motor (eg, convulsions, automatisms) or sensory (eg, loss or change), or autonomic activity No LOC, change in behavior or mental status, or postictal state; patients can recall event</td>
</tr>
<tr>
<td></td>
<td>Complex</td>
<td>Isolated unilateral motor (eg, convulsions, automatisms) or sensory (eg, hallucinations), or autonomic activity No LOC, but does have behavior change ± postictal confusion</td>
</tr>
</tbody>
</table>

### Approach
- If actively seizing: Immediate IV access, roll patient into decubitus
(avoid aspiration), suction airway, supplemental O₂, fingerstick glucose, magnesium (if pregnant), antiepileptic agents (IV, IO, IM, IN)

- If not actively seizing: Assess return to baseline mental status, focal deficits

**History**

- Description of events before, during, & after sz: prodrome, associated sx(s) (fever, vomiting, HA, trauma, photophobia, visual chg), any focal neuro sx(s), AEDs given, type of sz (partial vs. generalized; if partial: simple vs. complex) & duration, post-ictal state

- **First-time Sz:** Assess possible causal etiologies (see above)

- **Breakthrough Sz:** Assess similarities &/or differences from prior sz, typical sz frequency, last sz prior to presentation, any changes in AEDs, outpt provider, factors that may lower sz threshold (stress, sleep, noncompliance, new meds, toxins, alcohol, infxn)

- Differentiate from syncope (may have myoclonic jerks, no incontinence or tongue-biting, quick return to baseline mental status)

- Assess for sx of traumatic injury

**Physical Exam**

- Assess GCS, orientation, & memory (compare with baseline), neuro deficits

- Evaluate for e/o trauma (inc tongue biting) or ingestion; if persistent AMS & unwitnessed sz w/ e/o head trauma or fall, may need temporary C-spine immobilization until cleared


- **If active seizing:** Defer evaluation until cessation of sz (see Approach)

- **First-time Sz & back to baseline:**
  - Labs: CBC, BMP (glucose, Na), HCG; ± lactate, CPK (↑ lactate & ↑ CPK can help differentiate b/w unwitnessed convulsive sz & syncope w/ myoclonus), LP if immunosupp (even if afebrile); PRN based on hx: Tox screen, LFTs, LP
  - Neuroimaging: Obtain noncontrast CT in ED if feasible
    - May defer neuroimaging to o/p if: Age <40 y, normal neuro exam, no concern for intracranial path (no trauma, no hx malignancy or immunosupp, no fever, no HA, no A/C use), & good o/p f/u; preferred o/p study is MRI w/contrast
    - MRI w/contrast > CT for evaluation of tumors (esp in elderly, hx
cancer), but can be done as o/p in most pts if CT negative
• EEG: May be performed as outpt; indicated only for persistent AMS, SE, dx of viral encephalitis, intubated/paralyzed, r/o nonconvulsive SE
  • **Breakthrough sz:**
    • Labs: Electrolytes, UA, AED levels, ± CXR; ± lactate, CPK (↑ lactate & ↑ CPK can help differentiate b/w unwitnessed convulsive sz & syncope w/ myoclonus)
    • Neuroimaging: Consider if different from prior sz, prolonged duration since recent sz, trauma, or other c/f intracranial pathology
    • Keep differential broad even if known sz d/o, esp if therapeutic med levels

**Treatment**
  • **Airway:** Nasal trumpet, supplemental O₂, suction, positioning, may need to intubate if SE
  • **Abortive meds:** Benzodiazepines 1st line (available IV, IM, IN, buccal, PR); ongoing investigations to establish optimal second-line agent
    • IV lorazepam vs. IM midazolam: IM midazolam is noninferior & may be superior to IV lorazepam w/ regard to sz termination/need for rescue tx, & is quicker if no IV access *(Epilepsia 2015;56(2):254–262; NEJM 2012;366(7):591–600)*
    • IV lorazepam vs. IV diazepam: IV lorazepam superior to IV diazepam w/ regard to sz termination / need for rescue tx *(Cochrane 2014; (9):CD003723)*
    • Special cases w/ alternative 1st-line tx: Pregnant (Mg 4g IV), INH tox (pyridoxine 1g)
  • If not seizing, tx w/AED depends on risk of recurrence: *(Ann Emerg Med 2014;63(4):437–447)*
    • First-time sz (provoked or unprovoked): No AED indicated if back to baseline mental status, no current or known h/o structural brain disease/injury
    • If h/o sz d/o & ↓ AED levels, load w/ AED (PO or IV; home agent preferred)
    • If h/o sz d/o & nl AED levels (& no clear provoking trigger): Contact o/p prescriber to discuss ↑ o/p AED dose

<table>
<thead>
<tr>
<th>IV Treatment of Status Epilepticus</th>
</tr>
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<tbody>
<tr>
<td>Step</td>
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</tbody>
</table>
### Disposition

- **Provoked sz:** Disposition depends on underlying cause; if underlying cause cannot be rapidly reversed & pt remains at risk for recurrent provoked sz, admx vs. observation
- **Unprovoked sz:** Most can be safely discharged w/ close neuro f/u if nl mental status, exam, & w/u (above)
- If on long-term meds or 2nd sz, discuss w/ neurology regarding dose adjustments or starting a long-term med
- Explicit instructions to not drive, operate hazardous machinery or perform tasks where recurrent sz may cause harm; some states have mandatory reporting to DMV
- Admit all pts with 2+ sz in pre-hospital/ED or SE; may need ICU

### Pearl

- Treat alcohol w/d sz w/ BZD, almost never responsive to phenytoin

### VERTIGO

#### Definition

- The sensation of disorientation in space combined w/ sensation of motion/spinning
- May be due to benign (usually peripheral) or life-threatening (usually central) causes
  - Central comprise ~10% of cases; CVA comprises ~4% ([Mayo Clin Proc](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733053/))
  - RFs for central vertigo: Older age, males, HTN, CAD, DM, AF, h/o
Differential Diagnosis for Vertigo

<table>
<thead>
<tr>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>FB, cerumen impaction, acute otitis media, labyrinthitis, benign paroxysmal positional vertigo, Ménière’s dz, vestibular neuritis, perilymphatic fistula, trauma, motion sickness, acoustic neuroma, ototoxic medications (eg, gentamicin, furosemide)</td>
<td>Infection (encephalitis, meningitis, cerebritis), vertebrobasilar art. insufficiency, subclavian steal syndrome, cerebellar or brainstem hemorrhage or infarction, vertebrobasilar migraine, trauma (temporal bone fracture, postconcussive syndrome), tumor (brainstem or cerebellum), MS, temporal lobe epilepsy</td>
</tr>
</tbody>
</table>

History & Physical Exam

- **HX:** Onset & duration of sx; changes with position & direction; associated sx (HA, neuro sx, dysarthria, chg in hearing, CP/LH, palpitations); circumstances surrounding onset (trauma, torsional neck inj, neck manipulation, new meds); PMH including meds
- **EXAM:** Assess for neuro deficits, nystagmus, cerebellar exam, gait, ± Dix Hallpike; listen for carotid bruits, otoscopy, cardiac murmurs

Historical & Exam Features of Central vs. Peripheral Vertigo

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Timing</strong></td>
<td>Acute-onset (seconds)</td>
<td>Gradual-onset (min–hr)</td>
</tr>
<tr>
<td></td>
<td>Can be intermittent or constant</td>
<td>Progressive &amp; constant</td>
</tr>
<tr>
<td></td>
<td>Often self-resolves (sec–hrs)</td>
<td>Present later in course</td>
</tr>
<tr>
<td><strong>Intensity</strong></td>
<td>Severe</td>
<td>Mild–moderate</td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td>Always present: Unidirectional, fatigable horizontal or rotatory</td>
<td>May be absent, can be bidirectional. Vertical nystagmus almost always central in origin.</td>
</tr>
<tr>
<td></td>
<td><em>(never vertical)</em></td>
<td></td>
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<tr>
<td><strong>Associated sx</strong></td>
<td>Intense N/V</td>
<td>Mild nausea, often HA</td>
</tr>
<tr>
<td></td>
<td>Provoked by movement/position</td>
<td>Not affected by movement</td>
</tr>
<tr>
<td></td>
<td>± hearing loss or tinnitus</td>
<td>Usually no auditory sx</td>
</tr>
<tr>
<td></td>
<td>NI brainstem/cerebellar exam</td>
<td>May have abnl neuro exam</td>
</tr>
</tbody>
</table>

Evaluation

- ECG *(r/o arrhythmia)*, glucose & electrolytes, UA, HCG *(if child-bearing age)*
- Neuroimaging: Preferred modality is MRI; head CT PRN to r/o hemorrhage (eg, HA, trauma, A/C), limited utility for cerebellum/brainstem
- Consider CTA or MRA to evaluate for vascular dz (carotid, vertebrobasilar)

**Treatment**
- **Central:** Symptomatic relief (antiemetics, benzodiazepines); Neurology consult, ASA (if ischemic CVA); NSGY (if hemorrhagic CVA) & anticoagulation reversal
- **Peripheral:** usually supportive care w/ antivertigo medications (Diazepam 2–4 mg IV/5–10 mg PO, meclizine 25 mg PO, diphenhydramine, promethazine)
  - For BPPV, consider trying Epley maneuver (or modified self-Epley maneuver at home)
  - For acute bacterial labyrinthitis: ENT consult, IV abx, usually need admission
  - For Ménière’s: Supportive medications, encourage decreased salt intake, close ENT f/u

**Disposition**
- Home once sx improve w/ PCP/ENT f/u
- Admit if (a) central/CVA, (b) peripheral w/ refractory sx, (c) acute bacterial labyrinthitis

**Pearl**
- More than half of pts presenting to ED with a chief complaint of “dizziness” or “vertigo” may have nonneurologic processes (*Mayo Clin Proc* 2008;83:765–777)

<table>
<thead>
<tr>
<th>Common Causes of Peripheral Vertigo</th>
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<tr>
<td><strong>Etiology</strong></td>
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</table>
| Benign paroxysmal positional vertigo | - Due to otolith disruption w/ semicircular canals (often posterior)  
                                      - Most common cause of peripheral vertigo (lifetime prev 2.4%; annual rate of recurrence 15%) (*NEJM* 2014;370:1138–1147)  
                                      - **HX:** Brief (sec/min) spinning sensation, episodic (<1 min each), precipitated by chg in head position (eg, rolling over in bed), severe, a/w N/V  
                                      - **DX:** Dix-Hallpike causes sx & unidirectional nystagmus in >70% pts, depending on canal involved: start in seated position, rapidly lie flat on back, extend pt’s head back 45°, then immediately to left or right 45°, keep pt’s eyes open, monitor nystagmus & sx, repeat on other side. |
### Labyrinthitis
- **Viral**: Inflammatory d/o of inner ear 2/2 infection or external toxin
- **Acute bacterial**: Usually coexisting or recent URI/OM, may have hearing loss; usually nontoxic, may have mild fever; r/o VZV (Ramsay Hunt) requiring IV acyclovir & admx; usual tx is supportive care (antiemetics, hydration)
- **Toxic**: Due to medication ototoxicity; progressive sx, often w/ hearing loss, tinnitus, NO nystagmus.

### Vestibular neuronitis
- Noninflammatory d/o of vestibular system (unclear etiology)
- **HX**: Sudden onset, severe, isolated vertigo (no auditory sx); progressive over hours & then gradually subsides, but may have persistent mild sx for wks/mos; May have h/o prior infxn/toxin; ± nystagmus.

### Ménière’s Dz
- Increased pressure w/I inner ear endolymphatic system, either due to known cause (metabolic, endocrine, trauma, meds, etc.) or idiopathic
- **HX**: Classic tetrad: Episodic severe vertigo (a/w N/V, lasting min to hrs, often most severe sx), unilateral chg in hearing, tinnitus, & sensation of ear fullness or pressure; sx followed nonspecific fatigue & nausea × days, then prolonged sx-free remission
- **TX**: Supportive care, trigger avoidance, if severe may warrant trial of diuretics or steroids (limited supporting data)

### Acoustic neuroma
- Intracranial benign neoplasm arising from Schwann cells encasing vestibular or cochlear nerve; cause sx both by affecting signal transmission on affected nerve or by mass effect
- **HX**: Gradual onset, progressive unilateral sensorineural hearing loss (most common sx), ± tinnitus, HA, imbalance (rarely frank vertigo), facial weakness/numbness
- **DX**: Unlike other causes of peripheral vertigo, dx requires neuroimaging (MRI w/ contrast)
- **TX**: Observation (if few sx), surgical excision, stereotactic XRT

### FACIAL DROOP

**Approach**

- **Definition**: Unilateral weakness of the facial muscles, w/ or w/o other neuro deficits; either due to central (upper motor neuron) or peripheral (lower motor neuron) etiologies
Strength of eye closure & eyebrow elevation helps differentiate central vs. peripheral:
- Central etiology *spares* forehead due to bilateral innervation → *w/u for stroke*
- Bedside fingerstick blood glucose early b/c hypoglycemia can cause this

<table>
<thead>
<tr>
<th>Location</th>
<th>Differential</th>
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<tbody>
<tr>
<td>Peripheral</td>
<td>Bell's palsy (idiopathic), facial nerve injury, postsurgical (parotidectomy), infectious (Lyme dz, HSV, mastoiditis), acoustic neuroma, parotid malignancy, botulism</td>
</tr>
<tr>
<td>Central</td>
<td>CVA/TIA, intracranial bleed, Todd's paralysis, Guillain–Barré syndrome, cerebral vasculitis/arteritis, multiple sclerosis, myasthenia gravis, progressive supranuclear palsy, infection (meningitis, encephalitis, brain abscess), mass lesion, sarcoidosis, Lyme</td>
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### Differential Diagnosis for Facial Droop

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<th>Location</th>
<th>Differential</th>
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<td>CVA/TIA, intracranial bleed, Todd's paralysis, Guillain–Barré syndrome, cerebral vasculitis/arteritis, multiple sclerosis, myasthenia gravis, progressive supranuclear palsy, infection (meningitis, encephalitis, brain abscess), mass lesion, sarcoidosis, Lyme</td>
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<thead>
<tr>
<th>Location</th>
<th>Upper Face</th>
<th>Lacrim</th>
<th>Saliva**</th>
<th>Taste***</th>
<th>± Associated sx</th>
<th>Common Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortex****</td>
<td>Intact</td>
<td>Intact</td>
<td>Intact</td>
<td>UE weak</td>
<td>Infarct</td>
<td></td>
</tr>
<tr>
<td>Subcortical****</td>
<td>Intact</td>
<td>Intact</td>
<td>Intact</td>
<td>UE weak</td>
<td>Infarct</td>
<td></td>
</tr>
<tr>
<td>Pons</td>
<td>Weak</td>
<td>Intact</td>
<td>Intact</td>
<td>UE weak/numb, ataxia, nystagmus, CN VI palsy</td>
<td>Infarct, glioma, MS</td>
<td></td>
</tr>
<tr>
<td>CPA</td>
<td>Weak</td>
<td>Intact</td>
<td>Intact</td>
<td>Tinnitus, face numb ataxia, nystagmus</td>
<td>Neoplasm, AVM, sarcoid</td>
<td></td>
</tr>
<tr>
<td>IAC proximal to geniculate gang</td>
<td>Weak</td>
<td>Change</td>
<td>Change</td>
<td>Tinnitus, hearing loss, nystagmus</td>
<td>Bell's palsy, acoustic neur.</td>
<td></td>
</tr>
<tr>
<td>IAC/FC distal to geniculate gang</td>
<td>Weak</td>
<td>Change</td>
<td>Change</td>
<td>Tinnitus, hearing loss, nystagmus</td>
<td>Bell's palsy, acoustic neur., AOM</td>
<td></td>
</tr>
<tr>
<td>FN distal to SMF</td>
<td>Weak</td>
<td>Intact</td>
<td>Intact</td>
<td>None (except if trauma, parotid)</td>
<td>Head injury, parotid path</td>
<td></td>
</tr>
</tbody>
</table>

*Lacrimation innervated from nucleus superior salivatory nerve in pons, via nervus intermedius (traverses w/ CN VII in internal auditory canal).

**Salivation innervated from nucleus superior salivatory nerve in pons, via nervus intermedius (traverses w/ CN VII in IAC) & chorda tympani (traverses w/ CN VII in FC).*
Bell’s Palsy

History
- Acute onset (over hours) painless unilateral facial droop not sparing the forehead, ± aching of ear (60%), taste disturbances (60%), hyperacusis (30%), dry eye, ± cheek/mouth paresthesias (but true sensory loss suggests central lesion)
  - RFs: Adult, diabetics, pregnancy, tick exposure
  - Evaluate risk for more concerning pathology: RFs for TIA/CVA, sx of neoplasm, etc.
- Accounts for ~50% of all facial palsies. Can be bilateral, but this requires further w/u.
- Unclear etiology (proposed: ischemic mononeuropathy, HSV reactivation in geniculate g.)

Physical Exam
- Paralysis must include forehead; inability to smile or close eye, drooling, hyperacusis
  - Assess for changes in lacrimation, salivation, & taste (see above)
  - Look for findings of spec etiology; eg, erythema migrans (Lyme), vesicles (HSV)

Evaluation
- Labs & imaging not routinely indicated if typical presentation
- If atypical presentation, other signs, systemic sx: Neuroimaging & neuro consult

Treatment (Neurology 2012;79(22):2209)
- Artificial tears, tape eyelid before sleeping to prevent corneal injury (cannot close lids)
- Prednisone 60 mg QD × 5, then slow taper (NNT 11) (Cochrane 2010;3:CD001942)
- No empiric abx, but consider if concerned or severe: Acyclovir (HSV), doxycycline (Lyme)
  - No clear benefit of antiviral tx in Bell's (Cochrane 2009;4:CD001869)
Disposition
- Home w/ reassurance, neuro f/u if paralysis persists for months
- Prognosis: 80–90% complete recovery in 2–3 mo, 10% permanent, 14% recurrence.

INTRACRANIAL HEMORRHAGE

Overview

Approach
- Immediate IV access; low threshold for intubation if GCS <8 or declining; assess for e/o herniation (give empiric hyperosmolar tx); emergent neuroimaging, NSGY/neurology c/s
- Attend to concurrent life-threatening pathology (eg, ATLS, ACLS)
- Pts w/ ICH can decompensate rapidly 2/2 ↑ ICP

History & Physical Exam
- HX: Acuity/timing of onset, position, severity, duration, circumstances surrounding onset (trauma, exertion, Valsalva, cocaine), associated sx (HA, N/V, vision chgs, focal neuro sx, speech or behavior chg, fatigue, neck pain), PMH (HTN, cancer, connective tissue d/o), FHx (ICH, aneurysm/AVM, PCKD), Meds (A/C, anti-plt agents)
- EXAM: VS; Assess GCS, motor/sensory & coordination, meningeal signs, e/o trauma
  - Signs of impending herniation: Asymmetric nonreactive pupil, decorticate/decerebrate posturing, Cushing’s reflex (↑ BP, ↓ HR)

Evaluation
- CBC, BMP, PT/INR, PTT, Type & Screen
- STAT Noncontrast CT head to evaluate location & extent of bleeding; based on type of ICH, CTA may help clarify vascular etiology (eg, AVM, aneurysm)
- NSGY consultation: if ↑ ICP & ↓ GCS, may need bolt, drain, craniotomy vs. craniectomy

Treatment
- Reverse anticoagulation: See section on Anticoagulation Reversal (Ch. 11)
- Optimize BP (SBP 90–140 mmHg): Nicardipine, Labetalol, Esmolol gtt
Consider IV enalaprilat if unable to take PO but no need for continuous gtt

- Cerebral protection: HOB elevation to 30 degrees, minimize ↑↓ glucose, hyperthermia, ↓ BP, ↓ O₂, ↑ CO₂, seizures

### Subarachnoid Hemorrhage

#### Overview

- **Definition**: Acute bleed into subarachnoid space between pial & arachnoid mater; can be traumatic (often focal) or atraumatic (eg, ruptured aneurysm, AVM; often generalized)
- 80% of atraumatic SAH from ruptured aneurysm, but 30–50% of these may have had prior sentinel bleed (eg, leak); important to consider sentinel bleed in ddx of HA

<table>
<thead>
<tr>
<th>Grade</th>
<th>GCS</th>
<th>Clinical Appearance</th>
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<tbody>
<tr>
<td>1</td>
<td>15</td>
<td>No motor deficit</td>
</tr>
<tr>
<td>2</td>
<td>13–14</td>
<td>No motor deficit</td>
</tr>
<tr>
<td>3</td>
<td>13–14</td>
<td>Motor deficit</td>
</tr>
<tr>
<td>4</td>
<td>7–12</td>
<td>With or without motor deficit</td>
</tr>
<tr>
<td>5</td>
<td>3–6</td>
<td>With or without motor deficit</td>
</tr>
</tbody>
</table>

#### History & Physical Exam

- **HX**: Classically sudden “thunderclap” HA, max pain w/i 1 h, “worst HA of life”, ± neck pain, N/V, photophobia, syncope or AMS, focal neuro deficits, sz
- Red flags: Exertional/Valsalva, neck pain, arrival by ambulance, LOC, N/V
- RFs: Age >60, FH (4× risk), HTN, smoking, alcohol, cocaine,amphetamine use, PCKD, collagen/connective tissue d/o
- **EXAM**: Ranges based on severity; if sentinel bleed with low-grade, may have nl neuro exam, or if high-grade with low GCS, may be obtunded; Assess for photophobia & nuchal rigidity; May have ocular motor palsy 2/2 aneurysm compression.

#### Evaluation

- Noncontrast head CT: In pts with low pre-TP (*low risk* by hx & nl neuro exam): Se 100% (−LR 0.01) if performed w/i 6 h & read by
neuroradiologist, 89% after 6 h (BMJ 2011;343:d4277; Acad Emerg Med 2016;23(9):963–1003).

- LP (gold standard): ↑ opening pressure (>20 cm H$_2$O), Xanthochromia (100% Se if >12 h)
  - No established “lower limit” for RBCs: Multiple “test thresholds” have been studied, including visible xanthochromia (Se 31%, Sp 98%), RBC <1 K × 10$^6$/L in Tube 4 (pooled Se 76%, Sp 88%; +LR 5.7, −LR 0.21); Spectrophotometric bilirubin (Se 100%, Sp 95%; +LR 28.8, −LR 0.22) (Acad Emerg Med 2016;23(9):963–1003)

- CTA: Reaches sens of 98% for bleed, but is improving & will likely play greater role; obtain CTA if SAH is diagnosed in order to localize aneurysm/AVM
- Conventional angiography: gold standard for localizing aneurysm/AVM if CTA neg

**Treatment**
- See Approach above; early consultation to NSGY critical
- 70% of SAH will have vasospasm (usually 3–21d, peak 7–10d), causing delayed cerebral ischemia; nimodipine (60 mg q4h PO) should be started w/i 96h of SAH

**Disposition**
- Admit, may need ICU; prognosis dependent on WFNS Grade/GCS

**Subdural & Epidural Hematoma**

**Overview**
- **Definition:** Bleeding into the subdural or epidural spaces due either to trauma (EDH, SDH) or tearing of the bridging veins from rapid acceleration/deceleration injury (SDH), occasionally, no h/o trauma present (SDH)
- Both cause sx as a result of mass effect on brain parenchyma & are NSGY emergencies

**History, Physical Exam, Evaluation, Treatment**
- See section on EDH & SDH in Trauma chapter as well as General Approach above to ICH

**Nontraumatic Intraparenchymal Hemorrhage**

**Overview**
- **Definition:** Hemorrhage often within the subcortical white matter or
(less likely) brainstem, causing sx due to mass effect, vasogenic edema & localized inflammation

- Classified as either primary (HTN, amyloid angiopathy) or secondary (coagulopathy, AVM, neoplasm, cerebral venous sinus thrombosis, hemorrhagic conversion of ischemic infarct)

<table>
<thead>
<tr>
<th>Characteristic Appearance &amp; Location of Common Nontraumatic Etiologies</th>
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<tbody>
<tr>
<td><strong>Cause</strong></td>
</tr>
<tr>
<td>HTN</td>
</tr>
<tr>
<td>CAA</td>
</tr>
<tr>
<td>Coagulopathy</td>
</tr>
<tr>
<td><em>Neoplasm</em></td>
</tr>
</tbody>
</table>

*Common neoplasms: Primary CNS tumors, melanoma, lung, breast (Semin Roentgenol 2014:49(1):112–126)

**History, Physical Exam, Evaluation, Treatment**

- See Approach above to ICH

**Disposition**

- Admit to neurology if no e/o aneurysm/AVM; often require ICU

**ISCHEMIC STROKE**

**Overview**

**Approach**

- Requires immediate & rapid assessment; utility of thrombolysis is time-limited
- Immediate IV access, telemetry, supplemental O₂ if hypoxic, neuro c/s if tPA candidate
- Quick assessment of ABCs: if GCS < 8 but RR & O₂ nl, weigh risk-benefits of sending pt to CT scan w/o intubation, intubate if concern for imminent deterioration
- If possible, obtain patient advanced directive (goals of care) to guide resuscitation
- All patients need STAT fingerstick glucose to r/o hypoglycemia as cause of sx
Goal to R/O TIA, Sz, & ICH ASAP & provide lytics if w/i eligible time frame

History
- Establish time of onset (if unwitnessed, establish time “last seen nl”), sx progression (stable vs. improving), circumstances surrounding onset (recent health, trauma, sz, toxins)
- Complaints may be vague (AMS, numb, weak, vision Δ, dysarthria)
- Assess RFs for ischemic CVA (HTN/HLD, DM, AF, CAD/PVD, CHF, Valve dz, hypercoagulable states, PFO [5–10% of pop w/ clinically significant PFO])
- Assess for other causes (see table), inc. recrudescence (“unmasking”) of old CVA

<table>
<thead>
<tr>
<th>Differential Diagnosis for Ischemic Stroke</th>
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<tbody>
<tr>
<td><strong>CNS</strong></td>
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<tr>
<td><strong>Vascular</strong></td>
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<tr>
<td><strong>Tox/Metabolic</strong></td>
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<tr>
<td><strong>Hematologic</strong></td>
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<tr>
<td><strong>Other</strong></td>
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Physical
- Quick but detailed neuro exam using NIH Stroke Scale (see below)
- Check for arrhythmia, murmur, bruits, & rectal for occult blood if considering lytics

<table>
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<tr>
<th>NIH Stroke Scale</th>
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<tbody>
<tr>
<td><strong>Variable</strong></td>
</tr>
<tr>
<td>Consciousness</td>
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<td>Orientation (month, age)</td>
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<td>Commands (close eyes,</td>
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<td>grip)</td>
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<td>Best gaze</td>
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<td>Visual fields</td>
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<tr>
<td>Facial palsy</td>
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<tr>
<td>Motor arm (10 s drift)</td>
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<td>Motor leg (5 s drift)</td>
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<tr>
<td>Ataxia (finger/nose,</td>
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<tr>
<td>heel/shin)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Sensory</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
### Severe–total loss

<table>
<thead>
<tr>
<th>Language (writing if intubated) (Naming objects, pictures)</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>nl, no aphasia</td>
<td>0</td>
</tr>
<tr>
<td>Some loss of fluency or comprehension</td>
<td>1</td>
</tr>
<tr>
<td>Severe aphasia; fragmented</td>
<td>2</td>
</tr>
<tr>
<td>Mute, global aphasia</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dysarthria (Have pt read list of words)</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>nl</td>
<td>0</td>
</tr>
<tr>
<td>Slurs some words but understandable</td>
<td>1</td>
</tr>
<tr>
<td>Severe, unintelligible</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Extinction/Inattention (Bilateral stimulation)</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>No abnlty</td>
<td>0</td>
</tr>
<tr>
<td>Inattention or extinction to bilateral stimulation</td>
<td>1</td>
</tr>
<tr>
<td>Profound hemi-inattention</td>
<td>2</td>
</tr>
</tbody>
</table>

Score <5 = minor, Score>20 = severe neurologic deficit

### Evaluation:

- CBC, BMP, PT/INR, Troponin, UA, T & S
- ECG: May reveal AF; cerebral T waves (deep symmetric precordial) suggest ↑ ICP (rare)
- Noncontrast CT: r/o hemorrhage & exclude other etiologies; eval for early e/o infarction (hyperdense vessel segment; loss of grey-white differentiation, gyral effacement)
- CT Angiography: r/o arterial dissection; localize thrombus & map vasculature limitations (eg, stenosis, tortuosity) to help guide potential IA tx
- MRI: Highest Se & Sp for CVA in acute setting (initially ↑ DWI & ↓ ADC signal; after 6 h ↑ T2 FLAIR; after 16 h ↓ T1 signal), but often not immediately available
- Echocardiography: Eval for atrial/LV thrombus, mitral valve pathology, myxoma, PFO; diagnostic yield 4–10% (TTE) & 11–41% (TEE), but +findings often chg long-term mgmt; most useful in pts w/ abnl ECG or c/f embolic source (Postgrad Med J 2014;90(1066):434–438).
- Once CVA dx’ed: addx tests to evaluate RF (HgbA1c, Lipid panel) & guide 2° prevention

### Transient Ischemic Attack

#### Overview:

- **Definition:** Acute focal neurologic dysfxn 2/2 ischemia from arterial
occlusion (thrombotic or embolic) but completely resolving within 24 h (usually <1 h) & not a/w residual tissue infarction; signals ↑ CVA risk 2/2 shared underlying pathophysiology w/ CVA

- Etiologies: Often thromboembolic event (AF, PFO, Atherosclerosis), small vessel dz, or fixed large-vessel stenosis w/ transient ↓ BP
- Risk of CVA after TIA: 3% w/i 2 d, 5% w/i 7 d; ABCD² Score may predict individual risk, but should not supplant clinical judgment (see footnotes in table) or dictate f/u urgency

<table>
<thead>
<tr>
<th>ABCD² Score: Stroke Risk after TIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Factor</strong></td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>BP</td>
</tr>
<tr>
<td>Clinical signs</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Duration</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
</tbody>
</table>

**Pooled meta-analysis of 29 cohorts (Neurology 2015;85(4):304–305). Up to 1/3 of pts w/ TIA-mimics may have ABCD² ≥4, and 1/3 of pts w/ true TIA have ABCD² <4. Additionally, 1/5 pts w/ ABCD² <4 have >50% carotid stenosis, needing urgent f/u. Thus, score should accompany clinical judgement, eval of other CVA RFs (cervical arterial stenosis), and strong consideration should be given to ED neurology c/s to guide mgmt & urgency of f/u.

**History & Physical Exam**
- See Approach; If acute, emphasis should be placed on assessing degree of resolution

**Evaluation**
- If sx not fully resolved (NIHSS > 0): W/U as acute CVA (Noncontrast CT & CTA)
- If sx fully resolved (NIHSS = 0): May defer CT & obtain MRI/MRA w/i 24 h, unless c/f other etiologies (eg, partial sz 2/2 underlying neoplasm)
- Need to find etiology of TIA (echo, carotid imaging, Holter, MRI/MRA), usually as inpt

**Treatment** (Stroke 2013;44(3):870–947; Stroke 2014;45(7):2160–236)
- Tx focuses on short- & long-term risk reduction
- ASA (325 mg QD) ± clopidogrel (75 mg QD) based on severity of intracranial stenosis
- BP control: In acute setting, may be reasonable to avoid active BP control for >24 h unless markedly elevated (>220/120) or concurrent medical condition requiring ↓ BP; if BP remains elevated after first several days, tx w/ goal BP < 140/90
- Statin therapy if LDL-C >100 mg/dL
- Anti-coagulation if e/o Afib/Aflutter (VKA or NOAC); if Cl to A/C, ASA ± clopidogrel
- Carotid revascularization: Recommended for high-grade (>70%) & moderate (50–69%, NNT 15; if not otw high surgical risk) stenosis no risk reduction if stenosis <50%; if able to undergo CEA, CEA preferred over CAS (Cochrane 2012;9:10:662–668)
- Lifestyle modifications: Weight loss, diet/low-salt, exercise, ↓ ETOH, ↓ tobacco

**Disposition**
- Admit if 1st TIA, mx TIAs in short time, cardiogenic, or posterior circulation
  - In some stroke centers, select cases can be managed outpt (eg, recent full w/u)
- ABCD² is a useful data point but not well validated as a disposition tool

**Pearl**
- Recurrent TIAs w/ different sxes are likely cardiac emboli; if same sxes, likely cerebral

**Ischemic Stroke**

**Overview**
- **Definition:** Acute focal neurologic dysfxn 2/2 ischemia causing tissue infarction, often due to acute arterial occlusion (embolic >25% thrombotic > vascular dissection, etc) or fixed stenosis w/ hypotension

**History, Physical Exam, & Evaluation**
- See Approach; If acute, initial assessment should not delay imaging & decision to provide lytic tx (apart from reviewing Cls)
- Sxs & exam findings will depend on arterial distribution affected (see table)
  - Anterior circulation: Unilateral motor/sensory deficit (eg, numbness,
weakness, facial droop, monocular blindness [amaurosis fugax], aphasia)

- Posterior circulation: Nonlateralizing sx (eg, diplopia, dysarthria, dysphagia, ataxia)

### Common Ischemic Stroke Patterns

<table>
<thead>
<tr>
<th>Stroke Location</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ophthalmic Art.</td>
<td>Transient painless monocular vision loss (often embolic from ICA)</td>
</tr>
<tr>
<td>Internal Carotid Art.</td>
<td>See ACA &amp; MCA: profound motor &amp; sensory deficits</td>
</tr>
<tr>
<td>Ant. Cerebral Art.</td>
<td>C/L hemiparesis &amp; sensory loss (leg, perineum &gt; arm, face) ± impaired judgment/confusion, ± incontinence (pelvic floor weakness), ± disconnection syndrome (↓ awareness of I/L body, 2/2 corpus callosum infarction)</td>
</tr>
<tr>
<td>Mid. Cerebral Art.</td>
<td>C/L hemiparesis &amp; sensory loss (face, arm &gt; leg, perineum) ± aphasia (if dominant hemisphere; Broca/receptive [frontal] or Wernicke/expressive [temporal]) or neglect (if nondominant)</td>
</tr>
<tr>
<td>Post. Cerebral Art.</td>
<td>Homonymous hemianopsia ± cortical blindness ± Agnosia (object recognition), alexia (word recognition), prosopagnosia (face recognition), memory deficits ± Prominent contralateral sensory chgs w/o paralysis (thalamus)</td>
</tr>
<tr>
<td>Lacunar Art.</td>
<td>Pure hemiplegia (pons/internal capsule), pure sensory (thalamus), clumsy hand &amp; dysarthria syndrome (pons), unilateral leg paresis &amp; ataxia (pons/internal capsule)</td>
</tr>
</tbody>
</table>

### Common Posterior Fossa Syndromes (QJM 2013;106(7):607–615)

<table>
<thead>
<tr>
<th>Post. Inf. Cerebellar Art. (Lateral Medullary Synd, Wallenberg)</th>
<th>NO motor deficits Sensory: <em>Crossed</em> sensory loss on I/L face, C/L arm/leg Ataxia: I/L limb &amp; truncal (veer/leaning) ataxia Oculobulbar: Ocular (diplopia, nystagmus, ocular torsion), bulbar (dysarthria, dysphagia, hiccups, uvular deviation) Autonomic sx: Horner syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ant. Inf. Cerebellar Art. (Lat Pontine Synd.)</td>
<td>I/L facial weakness &amp; sensory loss I/L sensorineural hearing loss (labyrinthine art.) Ataxia, nystagmus</td>
</tr>
<tr>
<td>Basilar Art. (pontine)</td>
<td>Impaired or alternating responsiveness (may present w/ coma) Various B/L motor sx(s, including bulbar ± Visual impairment/cortical blindness • “Locked-in syndrome”: Only ocular muscles remain intact</td>
</tr>
</tbody>
</table>

**Treatment** *(Stroke 2013;44(3):870–947; Stroke 2014;45(7):2160–236)*
Early Neurology C/S & imaging: Recommended door-to-physician, ≤10 min; door-to-stroke team, ≤15 min; door-to-CT initiation, ≤25 min; door-to-CT interpretation, ≤45 min

ASA 325 mg PO/PR. May use clopidogrel, ticlopidine, or warfarin per neurology.

BP control: Labetalol (IV) & Nicardipine (gtt) first-line, use short-acting IV agents
- If TPA candidate: BP goal <185/110 (lysis contraindicated if >185/110 after 2 doses)
- If not TPA candidate: Treat only if persistently >220/120, sx other end-organ damage (eg, AMI), or alternative med condition needing BP control; lower ≤10–20%

Fibrinolytic therapy (rtPA 0.9 mg/kg): In selected pts w/i appropriate timeframe & w/o CIs
- Odds of favorable recovery decrease with time after sx onset (see table)
- Risks of tPA: ICH (6% risk, clinically significant 1–2%), angioedema (1-5), systemic bleeding; ↑ 7 d mortality, but no ↑ mortality at final f/u (Lancet 2012;379(9834):2364–2372)
- See table below for inclusion criteria, absolute & relative CIs
- Intra-arterial tPA (available at some stroke centers) may be preferred (w/ or w/o prior IV tPA) for proximal lesions (distal ICA, MCA, basilar), severe sx, CI to systemic tPA, or delayed presentation after sx onset (4.5–6 h, investigations ongoing 6–12 h)
- Thrombectomy (typically w/ tPA) (eg, Merci retrieval system): Recanalization w/ Merci retrieval 57%, in combo w/ IA tPA 70%; ICH risk 7–10% (Stroke 2008;39(4):1205–1212)
- If arterial dissection or suspected cardioembolic stroke, may consider heparin

<table>
<thead>
<tr>
<th>Time of TPA administration after sx</th>
<th>0–1.5 h</th>
<th>1.5–3.0 h</th>
<th>3.0–4.5 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Odds of favorable neuro recovery at 3 mo</td>
<td>2.81</td>
<td>1.55</td>
<td>1.40</td>
</tr>
</tbody>
</table>

Data pooled from 6 RCTs (Lancet 2004;363(9411):768–774); no change in mortality w/ different timing of tx.

Criteria for Thrombolysis in Acute Stroke
### Inclusion Criteria

- Age >18
- Clinical Dx of acute ischemic stroke w/ measurable neuro deficit
- Time of onset <3 h (well established), or <4.5 h in some centers

### Absolute CIs to Lysis

- CTH shows ICH or very large stroke (>33% of hemisphere)
- High clinical suspicion for SAH (even w/ nl CTH)
- Active internal bleeding (eg, GIB)
- Bleeding diathesis (PLT <100000, heparin in past 48 h, anticoagulation w/ INR >1.7)
- Stroke, intracranial surgery, or head trauma in past 3 mo
- LP in past 1 wk
- Recent arterial puncture at noncompressible site
- Prior ICH, AVM, or aneurysm
- Refractory HTN (SBP >185 mmHg & DBP ≥110 mmHg despite tx)

### Relative CIs (weigh risk–benefit)

- Minor or rapidly resolving sxks
- Witnessed sz at time of stroke onset
- Acute MI in past 3 mo
- Recent GI/GU hemorrhage in past 3 wk
- Major surgery or serious trauma in past 2 wk
- Pregnancy

### Additional Relative CIs (for use after 3 h & before 4.5 h)

- Age >80
- NIHSS >25 (suggests large stroke)
- Oral anticoagulant use (regardless of INR)
- Combination of prior ischemic strokes & diabetes mellitus

### Disposition

- Admit all patients; Large strokes may need ICU (risk of edema, hemorrhagic conversion)

### Pearls

- Inpt w/u includes carotid imaging, echo, Holter monitor, advanced serology (hypercoagulability, lipids, bleeding diathesis, ESR, ANA)
- NIHSS correlates w/ neurologic outcome at 3 mo but poor predictor for posterior CVAs

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**NEUROMUSCULAR SYNDROMES**

**MYASTHENIA GRAVIS**
Overview

- **Definition:** Autoimmune d/o (Abs against postsynaptic ACh nicotinic receptors) causing progressive weakness of incremental muscle groups, with intermittent crises marked my potential need for ventilatory support
- Epidemiology: Most commonly affects women in 20s–30s, men in 60s–70s (peak)
- DDx includes Lambert–Eaton syndrome (similar pathophysiology, paraneoplastic)

History

- Gradual onset, symmetric, fluctuating proximal & ocular muscle weakness
  - Common: Extraocular/ptosis (present in 50% initially), bulbar, limb (prox > distal); however, w/i 1 y most pts have generalized involvement
  - Sx least severe in morning; worsen w/ repetitive activity & throughout day
- Assess for triggers of crisis: Stress, infection, pregnancy, surgery, meds (abx, steroids)
- If advanced dz: Obtain clear goals of care in case need for intubation

Physical Exam

- Proximal weakness & fatigability worse w/ repetitive activity, relieved by rest
- CN affected early (ocular: Ptosis, diplopia; bulbar: Dysarthria, dysphagia)

Evaluation

- Neuro C/S: if new onset, poor o/p f/u, or probable need for admx
- If new dx: AChR Ab test has high Sp, but poor Se, esp in localized dz; Tensilon (edrophonium) test (2 mg IV over 15 s; binds to AChE, blocking ACh hydrolysis)
  - Tensilon test may precipitate bradycardia or heart block – have atropine at bedside
- If known dx: Differentiate MG crisis from cholinergic crisis (most pts on cholinergic meds)
  - Cholinergic tox: lacrimation, salivation, perspiration, bronchorrhea, N/V, diarrhea, brady
Measuring NIF (negative inspiratory force) can identify pts at risk of respiratory failure; NIF <20 cm H₂O suggests severe resp weakness

**Treatment**
- Ventilatory support as indicated (NIPPV, intubation)
- Long term txs include AChE-inh (pyridostigmine) & immunomod (steroids, etc.)
- Corticosteroids given in crisis but can worsen sx initially; minimal short-term effect
- Plasmapheresis & IVIG: mainstays of tx for acute crisis

**Disposition**
- Admit all pts w/myasthenia crisis
- If no e/o crisis & good o/p f/u, can d/c with close o/p f/u

---

**Guillain–Barré Syndrome**

**Overview**
- **Definition:** Acute autoimmune demyelinating peripheral neuropathy, often in response to external infectious exposure & characterized by loss of peripheral nerve reflexes
- Common associated pathogens: *Campylobacter* (~30%), EBV, HSV, HIV, *Mycoplasma*
- Slow recovery (can take mo in worst affected); 5% die from cx (sepsis, PE, dysautonomia)

**History, Physical Exam, & Evaluation**
- **HX:** Progressive ascending weakness; can start w/ numbness/paresthesias or pain in LE’s, f/b symmetric b/l weakness over hrs to wks
  - Usually (2/3) heralded by recent URI or diarrheal illness days – wks prior to sx
- **EXAM:** Acute ascending symmetric weakness, sensory chgs, ↓ DTRs (however, 10% of early cases will have DTRs), 20% will have autonomic dysfxn & potentially-fatal arrhythmias; ± CNS (hallucinations, psychosis, vivid dreams) *(NEJM 2012;366: 2294–2304)*
  - Miller-Fisher variant = ataxia, areflexia, ophthalmoplegia.
- **DX:** Clinical dx; w/u generally to r/o other dx
  - CPK nl (acute myopathy may present similarly but nl sensation & ↑
**Amyotrophic Lateral Sclerosis (ALS)**

**Overview**
- **Definition:** Degenerative dz of UMN & LMN
- **Epidemiology:** Age >40, M = F

**History, Physical Exam, & Evaluation**
- **HX:** Progressive motor weakness & atrophy, fasciculations, spasm; NO sensory loss.
- Assess pt goals of care regarding life-support interventions including airway
- **EXAM:** UMN & LMN findings, initially distal; fasciculations 2/2 denervation; may have bulbar findings (dysphagia, dysarthria) in advanced dz, spasticity, ↑ DTRs, +Babinski
- Bladder & bowel sphincters & ocular muscles often spared
- **DX:** If new onset, neuro consult & consider MRI brain & spinal cord

**Treatment**
- Supportive care (Respiratory support, antispasmodics)
- In pts w/ known dx, treat cx (DVT from immobility, asp PNA, UTI,
decubitus ulcers)

**Disposition**

- Depends on respiratory status, acuity

---

**Multiple Sclerosis (MS)**

**Overview**

- **Definition:** Progressive chronic immune-mediated demyelinating dz of the CNS
  - Generally follows relapsing-remitting (85–90%; may not have complete recovery b/w relapses) or primary progressive course (usually w/o relapses)
  - Epidemiology: Relapsing-remitting presents young (30 y) & F>M (3:1); primary-progressive presents older (40 y) & F=M; ↑↑ risk if 1st-degree relative also affected
  - Dx requires 2+ distinct episodes w/ differential neurologic sxs (ie, diff anatomic lesion)

**History**

- Acute episodes develop over hours–days (also remit over same time course)
  - Look for precipitating factors for exacerbation (eg, infection, hyperthermia)
  - Sx can be highly variable: vision, sensation, mobility/balance, cognition, sphincter control
  - See table for typical presentations, ocular sx common
  - Uhthoff phenomenon: Sxs worsen w/ ↑ body temp (exercise, hot bath, fever)

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**Typical Multiple Sclerosis Presentations** *(Lancet 2017;389(10076):1336–1346)*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute unilateral optic neuritis*</td>
<td>Sensory sx in a CNS pattern</td>
</tr>
<tr>
<td>Diplopia (2/2 INO or CN VI palsy)*</td>
<td>Lhermitte's sign</td>
</tr>
<tr>
<td>Facial sensory loss or trigeminal neuralgia</td>
<td>Asymmetric limb weakness</td>
</tr>
<tr>
<td>Cerebellar ataxia,</td>
<td>Urge incontinence, erectile dysfxn</td>
</tr>
</tbody>
</table>
nystagmus
Partial myelopathy

*Optic Neuritis: Painful EOM, afferent pupillary defect, decreased visual acuity, ± papilledema.
**INO: Inter-nuclear ophthalmoplegia (affected eye is able to abduct but not adduct; unaffected eye EOM nl) due to MLF lesion; Lhermitte's sign: Electric-shock sensation travelling down spine with neck flexion.

**Evaluation** *(Lancet 2017;389(10076):1336–1346)*
- Neuro consult indicated due to clinical benefit of early dx
- MRI Brain (Se 80% in pts w/ isolated syndrome): multifocal T2 hyperint white matter lesions
- MRI Spine (Se 50% in pts w/ isolated syndrome; mostly c-spine): indicated if sx localize to spinal cord or MRI Brain nondiagnostic
- LP: Indicated only if uncertainty based on MRI findings; may show pleocytosis (50%) & IgG oligoclonal bands (85–95%)

**Treatment**
- Treat any reversible underlying triggers (eg, infxn, dehydration, fevers)
- High-dose corticosteroids are 1st line for acute relapses; may consider addition of 2nd-line plasmapheresis in fulminant cases *(Neurology 2011;76(3):294–300)*
- Supportive: spasticity (baclofen, benzo), pain (carbamazepine/TCA), fatigue (amantadine)

**Disposition**
- Admit all new-dx for further w/u
- Most pts are admitted for relapses; can D/C if mild sx, nonprogressive, & close neuro f/u

**Pearl**
- Due to complexity of dx, rates of mis-dx pts (ie, w/o dz) may be as high as 10% *(Lancet 2017;389(10076):1336–1346)*

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**TRANSVERSE MYELITIS**

**Overview** *(NEJM 2010;363:564–572)*
- **Definition**: Acute or subacute inflammation & varying demyelination of a limited length of spinal cord causing motor, sensory, & autonomic dysfxn w/ sx correlating to level affected
- Often >2 vertebral seg involved; MS-associated TM can be <2 seg &
partial cord

- Epidemiology: All ages affected; bimodal peak (10–19y & 30–39y); M = F; unrelated to FHx
- Etiologies: Postvaccination (60% in children), postinfection, systemic autoimmune dz, or acquired demyelination dz (eg, MS), neuromyelitis optica, idiopathic (15–30%)

**History, Physical Exam, & Evaluation**

- **HX:** Acute or subacute paraplegia, sensory changes (with definitive spinal cord level), & sphincter loss (below level); onset over hours to days; bilateral but often asymmetric, often w/ neuropathic back/midline pain
- Ask about recent viral illness, immunization, FHx of MS, vision sx (NMO)
- **EXAM:** Symmetric or asymmetric weakness & sensory loss referable to a spinal cord level, hyperreflexia, +Babinski, +Lhermitte’s sign (+electric radiating back pain w/ neck flexion)
- **DX:** MRI entire spine w/ contrast; Neurology consult; If confirmed by imaging, may need LP (+pleocytosis) to help differentiate etiology & prognosticate

**Treatment**

- Ventilatory support as indicated by level of cord involvement & sx
- Supportive care: Analgesia (TCA, carbamazepine), Spasticity (baclofen, benzos), fatigue
- High-dose steroids (1-g methylprednisolone QD IV) are 1st-line, esp for postinfectious or demyelinating etiology (*NEJM* 2010;363:564–572)
- May consider plasmapheresis if fulminant or refractory to steroids; limited data

**Disposition**

- Admit to neurology
- Prognosis depends on etiology: Most recovery takes mo to yr
- MS-associated TM: Quicker & complete recovery; but ↑ risk of relapse c/w idiopathic, post-viral, or post-immunization
DYSURIA

(Am Fam Phys. 2015;92:778)

Definition
- Sensation of pain, burning, or discomfort on urination; generally indicates infection or inflammation of the bladder &/or urethra

Approach to the Patient
History
- ROS (fever, trauma, flank pain, abdominal or suprapubic pain, joint/back pain)
- PMH (STDs or PID, DM or immunocompromised)
- MEDS (topical irritants)
- SOCIAL (recent intercourse, multiple sexual partners)

Physical Exam
- Most pts should have at least an assessment of costovertebral angle tenderness & an abdominal exam
- Women: Consider pelvic exam if at risk for STDs, w/ vaginal sxs, postmenopausal
- Men: Should perform penile exam, testicular exam & prostate exam given risk of complicated dz

Evaluation
- Urine studies (clean catch UA, hCG ± culture, NAAT for GC/Chlamydia); CBC/Chemistries rarely indicated, unless suspected complicated dz (see below)
- Consider vaginal/urethral studies (smear w/ wet mount, cx), renal u/s, IV pyelography, CT abdomen/pelvis if warranted
- Further studies may include urine cytology, voiding cystourethrography, cystoscopy, urodynamic testing, but not routinely performed in ED
Dysuria Differential

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural</td>
<td>Urolithiasis, BPH, urethral stricture/diverticula, atrophic vaginitis</td>
</tr>
<tr>
<td>Infectious</td>
<td>Vulvovaginitis, urethritis, cervicitis, prostatitis, epididymo-orchitis,</td>
</tr>
<tr>
<td></td>
<td>cystitis, pyelonephritis, STDs</td>
</tr>
<tr>
<td>Meds</td>
<td>PCN, Cytoxan, topical hygiene products (vaginal spray/douche/lubricant)</td>
</tr>
<tr>
<td>Neoplastic/autoimmune</td>
<td>GU cancer (penile, vulvar/vaginal, prostate, bladder), Behçet, Reiter, SLE</td>
</tr>
<tr>
<td>Other</td>
<td>Instrumentation, urethral trauma, interstitial cystitis</td>
</tr>
</tbody>
</table>

**URINARY TRACT INFECTIONS**

*(Emerg Med Clin North Am. 2011;29:539)*

**Definitions**

UTIs are classified according to a spectrum of dz & the predominant clinical sxs: Asymptomatic bacteriuria, uncomplicated lower UTI (cystitis), uncomplicated pyelonephritis, complicated UTI w/ or w/o pyelonephritis, recurrent UTI

**Asymptomatic bacteriuria:** Absence of urinary sxs w/ Ucx ≥10⁵ cfu/mL uropathogen. Screening/ tx not recommended except in pregnant women

**Acute uncomplicated UTI:** Acute dysuria, urgency, frequency, suprapubic pain w/ UA ≥10 WBC/mm³ & Ucx ≥10³ cfu/mL

**Acute uncomplicated pyelonephritis:** Fever, chills, flank pain in the absence of alternative Dx & urologic abnlty w/ UA ≥10 WBC/mm³ & Ucx ≥10⁴ cfu/mL

**Complicated UTI:** Features of uncomplicated UTI/pyelonephritis AND 1 or more of the following—pregnancy, diabetes, male gender, immunosuppression (eg, chemo, AIDS), functional GU abnlty (indwelling catheter, neurogenic bladder), structural GU abnlty (renal stone, intestinal fistula, PCKD, kidney transplant pt)

**Recurrent UTI:** At least 3 episodes of uncomplicated UTI documented by culture in the last 12 mo in the absence of structural/functional abx

**Male urogenital tract infections:** Urethritis, prostatitis, epididymitis, orchitis
Asymptomatic Bacteriuria


Definition

› Absence of urinary sx with UA ≥10 WBC/mm³ & Ucx ≥10⁵ cfu/mL of the same uropathogen in 2 consecutive midstream urine samples ≥24 h apart; however, a single positive midstream urine is generally accepted as adequate & more practical

› USPSTF recommends screening for asymptomatic bacteriuria with Ucx for pregnant women at 12–16 wk gestation given increased risk of pyelonephritis, preterm labor & low birth weight

› USPSTF recommends against screening for asymptomatic bacteriuria in men or nonpregnant women

› The IDSA recommends against routine screening for or tx of asymptomatic bacteriuria in diabetic women, older persons >65 y/o residing in the community or institutionalized residents of long-term care facilities, spinal cord injury, & pts with indwelling urethral catheters

Treatment (Asymptomatic Bacteriuria in Pregnancy)

› 3–7-d course of nitrofurantoin or cephalosporin (cephalexin, cefpodoxime, cefdinir, cefaclor)

Pearl

› Given the high PPV of leukocyte esterase & nitrites on UA for bacteriuria, a positive test result in an asymptomatic pregnant pt in the ED should be considered for tx pending culture data

Acute Uncomplicated Urinary Tract Infection (Acute Cystitis)

Definition

Acute dysuria, urgency, frequency, suprapubic pain with UA ≥10 WBC/mm³ & Ucx ≥10³ cfu/mL, but ≥10⁵ cfu/mL also used to define UTI; absence of structural/functional UG tract abnormalities

Occurs when uropathogen from bowel or vagina colonize periurethral mucosa & ascend through urethra & bladder

Predominant uropathogens: E. coli (75–95%), K. pneumoniae, P. mirabilis, E. faecalis, S. saprophyticus, & S. agalactiae (group B Strep); rarely P. aeruginosa, Ureaplasma species

Probability of dz in pts presenting with 1 or more UTI sx is ~50%

History
Combination of dysuria, frequency, hematuria, fever, back pain, &/or self-diagnosis all increase the probability of UTI, whereas their absence decreases its probability.

- Vaginal d/c or irritation w/o the above sxs decreases probability of UTI
- RFs: Prior UTI, family h/o UTI, sexual intercourse, new sex partner (w/i 1 yr), use of spermicide

**Physical Exam**
- ±Fever; tenderness w/ suprapubic palpation; CVA tenderness
- GU exam if vaginal d/c or irritation present

**Evaluation**
- CBC/Chemistries rarely indicated
- Urine hCG, UA (+leukocyte esterase AND +nitrite has best diagnostic utility, where *either* +LE or +nitrite helpful w/ high pretest probability pts)
- Routine Ucx not needed in uncomplicated cases

**Treatment**


- Spontaneous resolution observed in 25–42% of untreated women
- Antibiotic regimens:
  - 1st-line:
    - Nitrofurantoin 100 mg BID × 5 d
    - Trimethoprim–sulfamethoxazole 160/800 mg (1 DS tablet) BID × 3 d (if <20% resistance in community)
    - Fosfomycin 3 g in a single dose
  - Alternative regimens:
    - Fluoroquinolones (ofloxacin, ciprofloxacin, levofloxacin) for 3 d
    - β-lactams (amoxicillin–clavulanate, cefdinir, cefaclor, cefpodoxime) for 3–7 d
- Symptomatic tx: NSAIDs, phenazopyridine (variable efficacy)

**Disposition**
- Home

**Pearls**
- Probability of cystitis >90% in women w/ sxs of UTI in the absence of vaginal d/c or irritation, thus consider empiric tx w/o UA or w/ nl UA (negative LE & nitrites do not reliably r/o UTI)
- UTI in males is rare thus consider STD, prostatitis
- Increasing *E. coli* resistance to amoxicillin & trimethoprim—
sulfamethoxazole

**Acute Uncomplicated Pyelonephritis**

**Definition**
- Upper UTI of renal pelvis & kidney secondary to ascending lower UTI (see *Acute Uncomplicated UTI for Pathogenesis & Uropathogens*)
- Fever, chills, flank pain in absence of alternative Dx & urologic abnlty w/ UA ≥10 WBC/mm³ & Ucx ≥10⁴ cfu/mL

**History**
- Highest incidence 15–29 y/o, followed by infants & elderly
- Combination of constitutional sx (fever, chills, malaise), lower urinary tract sx (dysuria, frequency, hematuria) & upper urinary tract sx (flank pain); N/V
- RFs: Prior UTI, sexual intercourse (esp ≥3/wk in last 30 d), new sex partner (w/ i 1 yr), use of spermicide, stress incontinence in previous 30 d, diabetes mellitus

**Physical Exam**
- ±Fever, tachycardia, hypotension; CVA tenderness (~25% bilateral)

**Evaluation**
- CBC may show leukocytosis, but can be nl (rarely guides decision making)
- Chemistries (esp BUN/Cr) if renal impairment suspected
- Urine hCG, UA (+leukocyte esterase AND + nitrite has best diagnostic utility, where *either* + LE or + nitrite helpful w/ high pretest probability pts; WBC casts)
- Ucx & susceptibility should always be performed (usually reveals ≥10⁵ cfu/mL of single uropathogen)
- Routine blood cultures not indicated
- Diagnostic imaging usually not indicated; can be considered to r/o alternative Dx, if complicated dz suspected, if sxs do not improve, or if recurrence → CT abdomen/pelvis study of choice over u/s

**Treatment**
- Outpt tx:
  - Ciprofloxacin 500 mg PO BID × 7 d
  - levofloxacin 750 mg PO QD × 5 d
  - Trimethoprim–sulfamethoxazole 160/800 mg (1 DS tablet) BID × 14 d
• Oral β-lactam for 10–14 d

*Above regimens can be given w/ (esp if resistance in community is known to exceed 10% or Bactrim/β-lactam are used) or w/o an initial 400 mg IV dose of ciprofloxacin, 1 g IV dose ceftriaxone, or consolidated 24-h dose of aminoglycoside

› Inpt tx:
• IV fluoroquinolone, an aminoglycoside (w/ or w/o ampicillin), an extended spectrum cephalosporin or PCN (w/ or w/o an aminoglycoside), or a carbapenem

Disposition
› Home: Most cases in o/w well appearing, healthy women
› ED Obs: Persistent emesis requiring IVFs or antiemetics
› Admit: Inability to take PO/intractable vomiting, age >65 y/o, toxic appearance, suspected sepsis, obstructive uropathy, inadequate f/u, poor social disposition (ie, homeless)

Pearl
› Cx: Emphysematous pyelonephritis, perinephric abscess, urosepsis, ARF, renal scarring

Complicated Urinary Tract Infection

History
(See Uncomplicated Cystitis & Pyelonephritis)

Physical Exam
(See Uncomplicated Cystitis & Pyelonephritis)

Evaluation
› CBC may show leukocytosis, but can be nl (rarely guides decision making)
› Chemistries (esp BUN/Cr)
› Urine hCG, UA (+leukocyte esterase AND + nitrite has best diagnostic utility, where either + LE or + nitrite helpful w/ high pretest probability pts; WBC casts)
› Ucx & susceptibility should always be performed (usually reveals ≥10⁵ cfu/mL of single uropathogen when positive)
› Routine blood cultures not indicated, but should be obtained in suspected sepsis
› Diagnostic imaging should be considered → CT abdomen/pelvis study of choice over u/s
› Urology consultation: Esp w/ known or suspected structural/functional abx, recent urologic procedure, UG tract FB, obstructive uropathy, UTI
in male

Treatment

- Empiric parenteral therapy w/ fluoroquinolone, carbapenem (ie, ertapenem, meropenem, or imipenem), or 3rd-generation cephalosporin (ie, ceftriaxone, cefotaxime), or piperacillin/tazobactam
- Duration: 7–10 d for complicated cystitis; 10–14 d for complicated pyelonephritis

Disposition

- Typically admit

Catheter-associated UTI (CA-UTI)


Definition

- **CA-UTI**: Sxs or signs compatible w/ UTI w/ no other identifiable source of infection w/ ≥10^3 cfu/mL uropathogen in pts w/ indwelling urethral, suprapubic, or intermittent straight catheter in urine sample obtained w/i 48 h of removal
- **Catheter-associated asymptomatic bacteriuria (CA-ASB)**: Presence of ≥10^5 cfu/mL uropathogen in a catheter urine specimen in a pt w/o sxs
- Pt scenarios may include pts transferred from long-term care facilities w/ chronic indwelling foley/suprapubic catheters, paraplegic pts w/ chronic indwelling catheters, pts w/ urinary obstruction w/ temporary foley catheter or intermittent straight catheterization, etc.

History

- New onset or worsening fever, rigors, AMS, malaise, or lethargy w/o identifiable cause in pt w/ catheter
- Dysuria, frequency, urgency, suprapubic pain, flank pain, hematuria in those whose catheters were recently removed

Physical Exam

- ±Fever, tachycardia, hypotension; CVA tenderness; suprapubic tenderness
- Cloudy/malodorous urine should not be used to differentiate CA-UTI & CA-ASB

Evaluation

(See *Complicated UTI*)
Treatment
(See *Complicated UTI for Antimicrobials*)

- Screening for & tx of CA-ASB are not recommended except pregnant women
- 3-d regimen may be considered in CA-UTI pts ≤65 y/o w/o upper tract sxs
- 5-d regimen of levofloxacin may be considered in CA-UTI pts not severely ill
- 7-d regimen recommended for CA-UTI pts w/ prompt resolution of sxs
- 10–14-d regimen recommended in those w/ delayed response

Prevention

- Strongly consider indication for catheter insertion, limit catheterization changes, aseptic technique w/ placement, among others

Disposition

- Home in majority of cases
- Admit: Age >65 y/o, toxic appearance, suspected sepsis, immunocompromised (DM, sickle cell, cancer on chemotherapy, organ transplant recipient, immunosuppressives), inadequate f/u, poor social disposition (ie, homeless)

Recurrent Urinary Tract Infection

Definition

- At least 3 episodes of uncomplicated UTI documented by culture in the last 12 mo in the absence of structural/functional abx
- *Relapse* (5–10% women) occurs w/i 2 wk of completing antimicrobial therapy & is caused by persistence of the same uropathogen, suggesting antibiotic resistance
- *Reinfection* occurs >2 wk after completing antimicrobial therapy & is generally secondary to infection w/ different organism or strain

History
(See *Uncomplicated Cystitis and Pyelonephritis*)

Physical Exam
(See *Uncomplicated Cystitis and Pyelonephritis*)

Evaluation

- CBC/Chemistries rarely indicated
Urine hCG, UA (+leukocyte esterase AND + nitrite has best diagnostic utility, where *either* + LE or + nitrite helpful w/ high pretest probability pts)

- Ucx should be obtained on representation to assess for antimicrobial resistance
- Postvoid residual if incomplete emptying suspected
- Imaging: Renal u/s, IV pyelography, CT abdomen/pelvis if warranted although not routine needed on emergent basis
- Further studies may include voiding cystourethrography, cystoscopy, urodynamic testing, but not routinely performed in ED

**Treatment**
(See *Uncomplicated UTI for Antimicrobials*)

- Consider starting prophylactic, continuous low-dose abx for 6-mo duration:
  - Nitrofurantoin 50–100 mg PO QD
  - Fosfomycin 3 g sachet PO q10d
  - Ciprofloxacin 125 mg PO QD
  - Cephalexin 125–250 mg PO QD, cefaclor 250 mg PO QD
  - Trimethoprim–sulfamethoxazole 40/200 mg QD or 3 times weekly
- May alternatively consider postcoital antimicrobial prophylaxis w/ a single dose w/i 2 h after intercourse (esp if UTI temporally a/w coitus):
  - Nitrofurantoin 50–100 mg
  - Trimethoprim–sulfamethoxazole 40/200 mg or 80/400 mg
  - Cephalexin 250 mg

- Self-start antibiotic therapy is an additional option (pt must be instructed to contact a medical provider w/i 48 h if sxs do not resolve)

**Disposition**

- Home w/ urology f/u to assess for anatomical/functional etiology

**Urethritis**

**Definition**

- Urogenital inflammatory condition characterized by urethral inflammation which can result from infectious & noninfectious etiologies
- Infectious causes include gonococcal (*N. gonorrhoeae*) & nongonococcal (*C. trachomatis, M. genitalium, T. vaginalis*, HSV, adenovirus)
- Rare causes include syphilis, CMV, & enteric bacteria
History
- Highest prevalence in adolescent, sexually active men
- Dysuria, urethral pruritus, mucopurulent or purulent urethral d/c; however, asymptomatic infections are common
- Urinary frequency & urgency typically absent
- Sexual hx: Current sexual activity, type (oral, vaginal, anal), MSM, number of sex partners, condom use, h/o STDs (esp GC/Chlamydia), sex w/ prostitutes
- Systemic sxos? (Fever, sore throat, arthritis, rash, back pain)

Physical Exam
- GU exam: Urethral meatus for skin lesions, erythema, d/c; milk urethra for d/c; testicular/epididymal exam in men, pelvic exam in women

Evaluation
- First-void (“dirty”) UA (may reveal + LE & ≥10 WBC/hpf), urine hCG
- Gram stain of urethral secretions w/ ≥5 WBC/hpf (presence of gram-negative intracellular diplococci c/w gonococcal dz) & culture
- Urine NAAT for N. gonorrhoeae & C. trachomatis most sens

Treatment
- GC & Chlamydia coinfection common so therapy should be geared toward both:
  - Azithromycin 1 g orally in a single dose OR doxycycline 100 mg PO BID × 7 d -AND-
  - Ceftriaxone 250 mg in a single IM dose
- Abstain from intercourse for 7 d & until all sex partners (w/i previous 60 d) are evaluated or empirically treated

Disposition
- Home w/ PCP referral for counseling & further STD testing

Pearl
- GC & Chlamydia are reportable to state health department


Male Urogenital Tract Infections

Acute Bacterial Prostatitis
(Curr Opin Infect Dis. 2016;29:86)
**Definition**
- The NIH consensus classification of prostatitis syndromes includes 4 categories:
  - I. Acute bacterial prostatitis
  - II. Chronic bacterial prostatitis (≥3 mo of sx)
  - III. Chronic bacterial prostatitis/chronic pelvic pain syndrome (CP/CPPS)
    - A. Inflammatory
    - B. Noninflammatory
  - IV. Asymptomatic inflammatory prostatitis
- Acute bacterial prostatitis is an acute bacterial infection of prostate w/ + Ucx, lower urinary tract sx, obstructive voiding sx, & systemic sx
- Bacterial prostatitis can be spontaneous or secondary to urologic intervention
- Bacterial spectrum similar to uropathogens seen in other UTIs (see Uncomplicated UTI); however, uropathogens of prostatitis carry greater number of virulence factors. Also, C. trachomatis, T. vaginalis, U. urealyticum, N. gonorrhoeae, & viruses rare causes

**History**
- Typical age 20–45 y/o; most common urologic Dx in men <50 y/o
- Acute onset fevers, chills, malaise, frequency, dysuria, poor urine stream, feeling of incomplete bladder emptying, & lower back/abdominal/pelvic pain
- Sexual Dysfxn (ejaculatory discomfort & hematospermia) may be present
- RFs: Recent urologic intervention/instrumentation, urethral stricture, urethritis

**Physical Exam**
- ±Fever; suprapubic abdominal discomfort
- Testicular exam should be performed to r/o epididymitis/orchitis
- DRE w/ warm, tender, swollen prostate

**Evaluation**
- Consider CBC & Bcx, esp if toxic appearing
- UA (+nitrites & LE, PPV 95%, NPV ~70%), Ucx
- Consider post-void residual urine measurement, urinary retention may not be evident
- Consider transrectal u/s if prostate abscess suspected (poor response
Prostate biopsy as an outpt

Treatment

- Systemically ill pts should receive parenteral abx: IV ciprofloxacin 400 mg BID, IV levofloxacin 500 mg IV QD OR ceftriaxone 2 g IV QD
- Clinically stable pts may be treated w/ oral therapy (usually fluoroquinolone)
  - Ciprofloxacin 500 mg PO BID or levofloxacin 500–750 mg PO QD × 2–4 weeks
  - Trimethoprim–sulfamethoxazole 160/800 mg (1 DS tablet) BID × 2–4 weeks
  - Sexually transmitted: Ceftriaxone 250 mg IM × 1 AND doxycycline 100 mg BID × 14 d

Disposition

- Home w/ urology f/u
- Admit if systemically ill, known antibiotic resistant pathogen, etc.

Pearls

- 10% men w/ acute bacterial prostatitis go on to suffer chronic prostatitis, & 10% progress to chronic prostatitis/chronic pelvic pain syndrome
- Cx: Chronic prostatitis (10%), acute urinary retention, prostatic abscess (~2%), sepsis

Epididymitis/Orchitis


Definition

- Epididymitis & orchitis are inflammation of the epididymis & testes, respectively, w/ or w/o infection
- Can be acute (<6 wk), subacute (6 wk–3 mo), or chronic (>3 mo) based on symptom duration
- Orchitis usually occurs when inflammation spreads from epididymis to adjacent testicle (epididymo-orchitis), but isolated orchitis w/o epididymitis can be seen w/ mumps
- Epididymitis can be sexually transmitted, caused by N. gonorrhoeae or C. trachomatis, or by ascending lower UTI by common uropathogens (see Uncomplicated UTI); M. tuberculosis should be considered in high-risk pts, & fungal or viral causes found in pts w/ immunodeficiency
- Noninfectious causes of epididymitis include postinfectious
inflammatory rxn to pathogens (ie, *M. pneumoniae*, adenoviruses), vasculitides, meds (ie, amiodarone)

**History**
- Primarily affects young men aged 18–35 y/o, bimodal distribution 16–30 y/o & 50–70 y/o
- Testicular pain, swelling usually beginning posteriorly overlying epididymis; lower urinary tract sx may be present
- RFs: Unprotected intercourse (esp anal), MSM, increased number of sex partners, h/o STDs (esp GC/Chlamydia), sex w/ prostitutes, structural/functional GU abnlty, urinary tract instrumentation

**Physical Exam**
- ±Fever; assess for CVA tenderness, suprapubic pain as e/o other urinary tract dz
- Testicular exam: Palpation of epididymis, testes, cremasteric reflex; tender, erythematous, swollen spermatic cord & testicular contents c/w epididymitis-orchitis
- Prehn sign: Relief of pain w/ elevation of testes can be seen w/ epididymitis. Inguinal exam for hernia or swollen, tender nodes.

**Evaluation**
- First-void (“dirty”) UA (+LE & ≥10 WBC/hpf suggests urethritis, favoring Dx of epididymitis); Ucx
- Gram stain of urethral secretions w/ ≥5 WBC/hpf (presence of gram-negative intracellular diplococci c/w gonococcal dz) & culture
- Urine NAAT for *N. gonorrhoeae* & *C. trachomatis* most sens
- Imaging: Testicular color Doppler ultrasonography (Findings: Thickened epididymis w/ increased blood flow suggesting hyperemia)

**Treatment**
- Sexually active men <35 y/o & older men w/ RFs for STDs:
  - Ceftriaxone 250 mg IM × 1
  -AND-
  - Doxycycline 100 mg PO BID × 10 d
- Abstain from intercourse for 7 d & until all sex partners (w/i previous 60 d) are evaluated or empirically treated
- Men >35 y/o or no RFs for STDs (thus likely caused by enteric organisms):
  - Levofloxacin 500 mg PO QD × 10 d
  - Ofloxacin 300 mg PO BID × 10 d
*Note: Above fluoroquinolones have activity against C. trachomatis & favorable UG tissue Penetration

- Supportive: NSAIDs for pain, ice/elevation of testes while at rest

**Disposition**

- Home

**Pearl**

- Pts <35 y/o likely to have an STD organism as etiology; >35 y/o more likely enteric pathogen

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**FLANK PAIN**

**Approach to the Patient**

**History**

- Onset (sudden vs. progressive)? Location? Dysuria/hematuria/urinary frequency? Prior h/o similar sx
- ROS (fever, rash, trauma, nausea, vomiting, weakness, abdominal pain), PMH (kidney stones, gout, cancer, AAA, congenital kidney dz, cardiac or vascular dz)

**Evaluation**

- CBC, Cr; consider renal u/s or noncontrast abdominal CT

<table>
<thead>
<tr>
<th>Flank Pain Differential</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal</td>
<td>Nephrolithiasis, urolithiasis, retroperitoneal hematoma, ruptured renal cyst, ureteral stricture</td>
</tr>
<tr>
<td>Infectious</td>
<td>Pyelonephritis, perinephric abscess, psoas abscess, pneumonia, discitis, vertebral osteomyelitis, epidural abscess</td>
</tr>
<tr>
<td>Vascular</td>
<td>Ruptured AAA, renal infarct, renal vein thrombosis, PE</td>
</tr>
<tr>
<td>GI</td>
<td>Biliary dz</td>
</tr>
<tr>
<td>Other</td>
<td>PCKD (ruptured cyst), renal malignancy, varicella-zoster</td>
</tr>
<tr>
<td>Trauma</td>
<td>Lumbar spasm, radiculopathy</td>
</tr>
</tbody>
</table>

**Urolithiasis (Nephrolithiasis and Ureterolithiasis)**


**Definition**
Urolithiasis denotes calculi (of mineral or organic solids) that form anywhere in the urinary tract; nephrolithiasis & ureterolithiasis more specifically denote calculi present in the kidney or ureter, respectively.

- Kidney stones form when urine becomes saturated with stone-forming salts.

Types of calculi:
- Calcium oxalate stones (~80%): Predisposing conditions include hypercalciuria (hyperparathyroidism, sarcoidosis, type I RTA, hypercalcemia of malignancy, thiazides) & hyperoxaluria (Crohn’s dz other ileal dz)
- Magnesium ammonium phosphate (struvite) stones (~15%): Requires combination of ammonia & alkaline urine. Source of ammonia from splitting of urea by urease-producing bacteria (Proteus, Klebsiella, Pseudomonas, & Staphylococcus)
- Uric acid stones (~5–10%): Secondary to hyperuricosuria (gout, DM2, HTN)
- Cystine stones: Secondary to inherited defects of tubular amino acid reabsorption
- Drug-induced calculi: 2/2 metabolic abnormalities that favor stone formation or crystallization of drug or metabolites

- Stones lodged in ureter are typically found in 3 locations: Ureteropelvic junction, at the level of the iliac vessels, & ureterovesicular junction.

**History**
- M:F, 2:1; Caucasian > Hispanic > Asian > African; peak incidence 20–50 y/o
- Renal colic (acute, spasmodic, unilateral flank pain radiating to groin/testes/labium) & visceral sxss (N/V/diaphoresis)
- Distal stones may cause lower abdominal pain & lower urinary tract sxss (dysuria, frequency, hematuria)
- PMH: FH nephrolithiasis, hyperparathyroidism, sarcoidosis, RTA, malignancy, Crohn’s, jejunoileal bypass, recurrent UTI, gout, DM2, HTN, structural urologic abnormalities
- Meds: Indinavir, loop/thiazide diuretics, laxatives, carbonic anhydrase inhibitors, ciprofloxacin, sulfonamides have been a/w drug-induced calculi

**Physical Exam**
- Fever? Tachycardic? Generally uncomfortable appearing, diaphoretic, cool/clammy skin
CVA tenderness; lower abdominal/pelvic tenderness (if stone has migrated)
Assess for midline spinal TTP, acute abdomen, etc. which suggest alternative dx

Evaluation
- UA (may show +RBCs, though sens 84% spec 48% for stone; proteinuria, crystalluria), Ucx
- Consider BUN/Cr; CBC usually nonspecific & not helpful
- Imaging:
  - Renal U/S (sens 45%, spec 94% for stones; sens 85–90% spec 90–100% for hydro):
    - May be initial radiographic exam w/ high pretest probability or if CT not possible (pregnancy); esp useful for detection of hydronephrosis or ureteral dilatation; not sens stones <5 mm; can be done point-of-care
    - Nonenhanced helical CT (sens 96–98%, spec 100%) [Indinavir stones not visible on CT]
  - Useul as initial radiographic exam, particularly w/ 1st presentation of suspected stone or low-moderate probability; able to make alternative diagnoses; modality of choice when available

*Indinavir stones not visible on CT

Treatment
- Data suggests IVFs likely not useful for acute renal colic from urolithiasis, but consider if pt appears dehydrated or has AKI
- Pain control: NSAIDs (ibuprofen 600 mg PO TID or ketorolac 15–30 mg IV if unable to take PO [caution in renal insufficiency]) & morphine 0.1 mg/kg × 1 then titrated for further relief
- Medical expulsive therapy: Tamsulosin 0.4 mg PO QD × 14 d or until stone passage; other alpha-antagonists (doxazosin, terazosin, alfuzosin) & nifedipine still used by many. SUSPEND trial (RCT 1167 pts tamsulosin, nifedipine or placebo) showed no increase in stone passage with MET. (Health Tech Assess. 2015;19:1)
- Urology consult: For concomitant infection, renal insufficiency, or low likelihood of stone passage (>10 mm)

Disposition
- Home: Adequate pain control in ED, nl Cr; f/u w/ urology in 24–48 h if stone >5 mm
- Admit: Intractable pain, unable to tolerate POs, renal failure, infection, renal transplant, single kidney, comorbid conditions (DM, baseline CRI),
infected stone w/ obstruction

**Pearls**

- Presence or absence of hematuria alone cannot be used to diagnose or exclude nephrolithiasis.
- Most stones ≤5 mm (70–98%) will pass spontaneously. Stones >5 mm have smaller chance (25–51%) of spontaneous passage & are more likely to need urologic intervention. *(J Urol. 2015;194:1009)*
- Send pts home w/ strainer, esp 1st-time stone formers for stone analysis.
- Cx: Obstructed infected kidney (urologic emergency requiring urgent decompression), renal insufficiency, failed expulsion.

**HEMATURIA**


**Definition**

- Hematuria is blood in the urine. Gloss hematuria is visible. Microscopic hematuria is ≥3 RBCs/hpf in urine sediment.
- Hematuria must be distinguished from pigmenturia (discoloration of urine). Pigmenturia can be caused endogenously by melanin, porphyrins, bilirubin, myoglobin, or hemoglobin or exogenously by meds (ie, warfarin, rifampin, phenazopyridine, phenytoin, etc.), beets.

**History**

- Onset (sudden vs. chronic)? Dysuria/urinary frequency/renal colic? During entire or part of urine stream? (hematuria at beginning of urination → urethral; throughout urination → upper urinary tract or proximal bladder; end of urination → bladder neck or prostatic urethra)
- Painless hematuria should raise suspicion for genitourinary malignancy.
- ROS (fever, weight loss, night sweats, rash, sore throat, abdominal pain, N/V, recent viral infection or UTI; trauma; excessive exercises; pelvic radiation)
- PMH (kidney stones, HTN, cancer, congenital kidney dz, vascular dz, bleeding diathesis, SCD, hereditary spherocytosis)
- MEDS:
  - Drugs that cause pigmenturia: Warfarin, rifampin, phenazopyridine,
phenytoin, azathioprine, deferoxamine, doxorubicin, riboflavin
- Drugs that cause myoglobinuria: Amphotericin B, barbiturates, cocaine, diazepam, ethanol, heroin, methadone, statins
- Drugs that cause hematuria: NSAIDs, anticoagulations, busulfan, cyclophosphamide, OCPs, quinine, vincristine
- Social (smoking, benzene or aromatic amine exposure)

**Physical**
- Evaluate for HTN, petechiae, arthritis, rash
- Assess for suprapubic & CVA tenderness; thorough GU exam including prostate exam
- Postvoid residual if concern for urinary retention

**Evaluation**

**Key question: Is this truly hematuria?**
- Urine dipstick + blood (can be seen w/ hematuria, hemoglobinuria, myoglobinuria, or other pigmenturias); urine sediment necessary to confirm >5 RBCs/hpf as well as identify protein, RBC casts (suggests glomerulonephritis), & crystalluria (suggests urolithiasis)
- Other urine studies: Urine cytology
- CBC, BUN/Cr, coags (if isolated hematuria—erythrocytes in sediment, but no protein—suggests bleeding diathesis)
- Outpt imaging: CT urography (1st line), renal u/s, MRI. Cystoscopy if ≥35 y/o.

**Disposition**
- Large, gross hematuria may warrant continuous monitoring of HCT & urology eval. If microscopic, can obtain further outpt eval by nephrology or urology.

<table>
<thead>
<tr>
<th>Hematuria Differential</th>
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<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
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<tr>
<td>Structural</td>
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<td>Infectious</td>
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<td>Vascular</td>
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<tr>
<td>Meds</td>
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<tr>
<td>Inflammatory</td>
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</tbody>
</table>
**Trauma/Other**
Renal trauma, urethral or ureteral trauma, recent instrumentation, paroxysmal nocturnal hemoglobinuria, vigorous exercise

**Neoplastic**
Renal Ca, urethral Ca, bladder Ca, prostate Ca

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**ACUTE KIDNEY INJURY**

*(Lancet. 2012;380:756)*


**Approach to the Patient**

**Definition & Staging**

- AKI is defined as any of the following:
  - Increase in serum Cr by $\geq 0.3$ mg/dL ($\geq 26.5$ μmol/l) w/i 48 h; or
  - Increase in serum Cr by $\geq 1.5$ times baseline, which is known or presumed to have occurred w/i prior 7 d; or
  - Urine volume $<0.5$ mL/kg/h for 6 h

- AKI is staged for severity according to the following criteria:

<table>
<thead>
<tr>
<th>Stage</th>
<th>RIFLE Criteria</th>
<th>Serum Cr</th>
<th>Urine Output</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Risk</td>
<td>1.5–1.9 times baseline -OR- GFR decrease $&gt;25%$</td>
<td>$&lt;0.5$ mL/kg/h for 6–12 h</td>
<td>- D/c nephrotoxins - Ensure volume status/perfusion pressure - Monitor Cr &amp; UOP - Avoid hyperglycemia - Consider alternative to using radiocontrast - Noninvasive w/u - Consider invasive w/u</td>
</tr>
<tr>
<td>2</td>
<td>Injury</td>
<td>2–2.9 times baseline -OR- GFR decrease $&gt;50%$</td>
<td>$&lt;0.5$ mL/kg/h for $\geq 12$ h</td>
<td>-AND- - Check for changes in drug dosing - Consider RRT - Consider ICU admit</td>
</tr>
<tr>
<td>3</td>
<td>Failure</td>
<td>3 times baseline -OR-</td>
<td>$&lt;0.3$ mL/kg/h for $\geq 24$ h</td>
<td>-AND- - Avoid subclavian</td>
</tr>
</tbody>
</table>
History
- ARF is usually asymptomatic & diagnosed when labs reveal renal abnormalities
- Sxs may include decreased urine output, weight gain, fluid retention (peripheral edema, anasarca, ascites), fatigue, anorexia, N/V, pruritus, altered sensorium, thirst/orthostasis (prerenal)
- ROS (fever, rash, flank pain, hematuria)
- PMH: Baseline renal impairment, CHF, liver dz, SLE, multiple myeloma
- MEDS (ACEI/ARB, NSAIDs, aminoglycosides, other abx, cisplatin, amphotericin B, diuretics)

Physical
- Assess volume status; myoclonus, pericardial or pl rub, rash, mental status, edema
- Stigmata of CHF, liver dz, collagen vascular dzs

Evaluation
- CBC, Chem 10 (BUN/Cr ratio), serum osmolality; consider VBG w/ STAT potassium
- Urinalysis/sediment, urine lytes (urine Na, urine K, urine Cr, urine osmolality)
  - $\text{FE}_{\text{Na}}\% = \frac{(\text{Urine Na} \times \text{Plasma Cr})}{(\text{Plasma Na} \times \text{Urine Cr})} \times 100$
- Consider LFTs, BNP if indicated
- EKG for cardiac electrical instability from potential electrolyte abx
- Consider point-of-care cardiac, IVC, renal u/s
- Imaging: Renal u/s (r/o obstruction, assess flow); consider CT abdomen if c/f pelvic mass, Doppler u/s of renal vasculature
- Other studies: Renal biopsy

| Loss | Persistent ARF = complete loss of kidney function for >4 wk |
| Loss | End-stage kidney disease (>3 mo) |

Differential Diagnosis of AKI/ARF

| Pathophysiology | Differential |
### Prerenal

**Hypovolemia:**
Dehydration, hypotension/shock, hemorrhage, vomiting/diarrhea, diuresis, burns, pancreatitis, severe hypoalbuminemia

**Altered Renal Hemodynamics:**
Low cardiac output states (CHF, severe valvular heart dz, tamponade, massive PE, abdominal compartment syndrome), sepsis, anaphylaxis, Meds (NSAIDs, ACEI/ARBs), hepatorenal syndrome

### Intrinsic renal

**Renovascular Obstruction:**
Renal artery
- atherosclerosis/thrombosis/embolism/dissection/vasculitis
Renal vein thrombosis/external compression

**Glomerular Dz:**
Glomerulonephritis, vasculitis, malignant HTN, preeclampsia, DIC, collagen vascular dzs (SLE, scleroderma)

**Intratubular Obstruction:**
Multiple myeloma, uric acid, acyclovir, MTX, indinavir

**Acute Tubular Necrosis:**
Profound ischemia, infection, radiocontrast, calcineurin inhibitors, abx (ie, aminoglycosides), antifungals (amphotericin B), chemo (ie, cisplatin), ethylene glycol, rhabdomyolysis, HUS/TTP

**Interstitial Nephritis:**
Allergic nephritis (β-lactams, fluoroquinolones, sulfa, NSAIDs), pyelonephritis, leukemia/lymphoma, sarcoid

### Postrenal

**Ureter:**
Calculi, clot, cancer (pelvic mass), external compression

**Bladder Neck:**
Calculi, clot, cancer (pancreatic), BPH, neurogenic bladder

**Urethra:**
Stricture, valves

---

### Interpreting Laboratory Data in AKI/ARF

<table>
<thead>
<tr>
<th></th>
<th>BUN/Cr</th>
<th>FE\textsubscript{Na}</th>
<th>Urine\textsubscript{Na}</th>
<th>SpGrav</th>
<th>Urine\textsubscript{osm}</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerenal</td>
<td>≥20</td>
<td>&lt;1%</td>
<td>&lt;10 mmol/L</td>
<td>&gt;1.018</td>
<td>&gt;500</td>
<td>Hyaline casts</td>
</tr>
<tr>
<td>Intrinsic renal</td>
<td>10–20</td>
<td>&gt;1%</td>
<td>&gt;20 mmol/L</td>
<td>&lt;1.015</td>
<td>300–500</td>
<td>- Muddy brown casts (ATN)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- RBC casts (glomerular injury, tubulointerstitial nephritis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- WBC casts (interstitial nephritis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- Broad granular casts (CKD)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- Eosinophiluria</td>
</tr>
</tbody>
</table>
Postrenal <10 >1% — — <350

Treatment

- **Prerenal:** Correct volume status/perfusion pressure (IVFs, pressors, PRBCs if indicated, diuresis/inotropes if cardiorenal)
- **Intrinsic:** Eliminate nephrotoxins, treat underlying cause, consider glucocorticoids
- **Postrenal:** Transurethral or suprapubic catheter placement; may require ureteric stents or percutaneous nephrostomy tube placement
- Consider sodium bicarbonate if pH <7.2 or HCO₃ <15 mmol/L as bridge to dialysis

**Indications for Emergent Dialysis and Renal Replacement Therapy**
“**A, E, I, O, U**”

- Acidosis (pH < 7.1)
- Electrolyte imbalance (hyperkalemia, hypocalcemia, hyperphosphatemia)
- Intoxication (lithium, salicylates, ethylene glycol, methanol, among others)
- Overload (volume overload)
- Uremia (pericarditis, encephalopathy, neuropathy, bleeding)

**Disposition**

- Home: Mild prerenal azotemia may be adequately treated w/ hydration; pts w/ postobstructive ARF can be sent home if obstruction is relieved (ie, w/ bladder catheter) & no significant comorbidities
- Admit: Pts w/ uremia, significant electrolyte abnormalities, volume overload, severe metabolic acidosis, unexplained ARF

**Pearl**

- Cx: Intravascular volume overload, hyponatremia, hyperkalemia, hyperphosphatemia, hypocalcemia, hypermagnesemia, metabolic acidosis, uremia, anemia, arrhythmias
TESTICULAR TORSION/TORSION OF TESTICULAR APPENDIX

(Emerg Med Clin North Am. 2011;29:469)

History
Testicular Torsion
- Sudden onset pain (± swelling) in scrotum w/ radiation into abdomen; pain may be intermittent; N/V; most commonly in puberty

Torsion of Appendix
- Similar presentation to testicular torsion but pain can be localized to superior pole of testicle; benign condition

Physical Exam
Testicular Torsion
- Ill appearing, very tender/swollen/elevated testicle that may lie horizontally or anteriorly rotated; presence of cremasteric reflex does not r/o dz

Torsion of Appendix
- Normal-appearing testes; tenderness localized to superior pole of testicle; may have nodular “blue dot” at superior pole of testicle

Evaluation
- Labs: Preop labs if surgery anticipated
- Imaging: Scrotal duplex u/s to assess flow to testicle, but imaging should not delay time to OR; HRUS if duplex equivocal

Treatment
- Consult urology immediately if concern for testicular torsion as time to OR is critical for survival of testicle; if delay to OR, may attempt manual detorsion in medial to lateral direction (“open book” technique)
- Analgesia
- Antiemetics

Pearls
- >90% salvage rate if detorsion occurs <6 h
- Continuous pain >24 h is a/w an infarcted testicle
**PHIMOSIS AND PARAPHIMOSIS**


**History**

**Phimosis**
- Inability to retract the distal foreskin over the glans penis; “ballooning” of the prepuce during urination; painful erection, preputial pain, weak urinary stream

**Paraphimosis**
- Inability to completely reduce foreskin distally back to natural position over glans penis. Entrapped foreskin forms constricting band, leads to pain & swelling.
- A/w vigorous sexual activity & chronic balanoposthitis
- Occurs exclusively in uncircumcised males & is a urologic emergency
- Pediatric: Often seen w/ forceful retraction or forgetting to reduce foreskin after bathing/voiding; irritability may be the only sign in nonverbal children

**Physical Exam**

**Phimosis**
- Inability to retract foreskin proximally over glans penis

**Paraphimosis**
- Foreskin retracted behind the glans & cannot be replaced to nl position; proximal shaft is soft (unless there is accompanying infection) w/ glans appearing erythematous/edematous & eventually blue/black & firm

**Treatment**
- If significant manipulation is expected, you may perform a penile block. On the dorsal aspect of the penis in the 2- & 10-o’clock positions, deposit 1% lidocaine; subsequently complete a ring block by depositing anesthetic circumferentially around the proximal shaft.

**Phimosis**
- No acute intervention needed unless infection suspected. Consider topical steroids (0.05–0.1% betamethasone) × 4–6 wk for mild–moderate cases.

**Paraphimosis**
Compress the foreskin & glans by snugly grasping it w/ the palm of the hand & apply pressure for several minutes. Other methods to reduce edema include:

- **Dundee micropuncture technique**: Make ~20 puncture holes in edematous foreskin tissue w/ a small needle (27 gauge) & express the fluid
- **Hyaluronidase technique**: Inject 1 cc of hyaluronidase (150 U/mL) using a tuberculin syringe into the site of edematous foreskin
- **Sugar technique**: Soak a swab of 50 mL of 50% dextrose solution & leave it wrapped around the foreskin for 1 h

Attempt manual reduction by placing index fingers on dorsal border of glans behind retracted prepuce & thumbs on glans; may facilitate w/ ice, elastic bandage over glans or spreading hyperosmolar agents (such as sugar/dextrose) over glans to reduce swelling

Consult urology if manual reduction unsuccessful

**Disposition**

- Home: Phimosis ± abx for accompanying infection; paraphimosis if skin is in the nl position. Urology f/u for all paraphimoses.
- Admit: Paraphimosis not reduced by conservative methods

**Pearls**

- Educate parents/caretakers of children on importance of avoiding forcible retractions & of gentle reduction of foreskin after bathing & voiding
- Paraphimoses that are not immediately treated are at risk for necrosis & autoamputation

---

**PRIAPISM**


**Definitions**

- Priapism is defined as a prolonged erection lasting generally >4 h in the absence of sexual stimulation
- Ischemic (low-flow) priapism is the most common subtype & is due to painful engorgement of the corpora cavernosa. This can lead to intracavernosal acidosis, sludging of blood, thrombosis of cavernal
arteries, & impotence

- Nonischemia (high-flow) priapism is rare, painless, & is caused by increased arterial inflow to the penis as a result of traumatic arterial–cavernosal fistulas

**History**

- Painful, persistent erection lasting >4 h, not relieved by ejaculation
- RFs: impotence agents (sildenafil), SCD, leukemia, urogenital malignancies (prostate, bladder), CVA, spinal cord injury antihypertensives (hydralazine, prazosin, doxazosin), antidepressants (trazodone, fluoxetine, sertraline), antipsychotics (phenothiazines & atypicals), phosphodiesterase inhibitors, cocaine, toxins (scorpion, black widow, CO)

**Physical Exam**

- Obvious erection, generally involving only the corporal cavernosa & flaccid corpora spongiosum

**Evaluation**

- Labs: Preoperative labs if contemplating OR
- May send a blood gas from penile aspirate

**Treatment**

- Pain control
- To reduce flow/vasoconstriction:
  - Oral/IM: Terbutaline 5 mg PO × 1; terbutaline 0.25–0.5 mg IM × 1 (unclear benefit)
  - Intracavernosal phenylephrine injection: Using a 25- or 27-gauge needle (or tuberculin syringe), inject 0.2–0.5 mg of phenylephrine into corpus q10–15min (maximum 4–5 doses) 2 cm distal to origin of shaft on dorsal penis at 2- or 10-o’clock position
  
  *Note: Must dilute phenylephrine solution. Take phenylephrine 1% solution (10 mg/mL) & extract 1 mL (10 mg) from solution. Add this 1 mL to 9 mL of saline, which will give you 1 mg/mL of phenylephrine solution. You can then extract 0.2–0.5 mL (0.2–0.5 mg) of this for intracavernosal injection.*

- If unsuccessful, aspiration/irrigation technique:
  - Perform penile nerve block: On the dorsal aspect of the penis in the 2- & 10-o’clock positions, deposit 1% lidocaine; subsequently complete a ring block by depositing anesthetic circumferentially around the
proximal shaft
- Prep & drape penis in sterile fashion
- At 2- or 10-o'clock position insert a 16–18 g needle (also consider 18-gauge dialysis butterfly access needle), & using a 10–30 mL syringe, slowly aspirate while milking corpus w/ other hand until return if bright red blood & detumescence occurs
- If this fails, you can attempt to irrigate by injecting 20–30 mL of phenylephrine & NS solution (10 mg phenylephrine in 500 mL NS) as exchange for 20–30 mL aspirate
  - W/ sickle cell crisis: IVFs, O₂, pain control, consider exchange transfusion
  - Consult urology for refractory priapism (may necessitate surgical decompression)

Disposition
- Recommended to observe for at least 2 h to assess for recurrence
- Home: Once detumescence achieved. Recommended to d/c w/ 3-d course of oral α-adrenergic agent (pseudoephedrine)
- Admit: If priapism not responsive to ED tx

Pearls
- >12 h of priapism a/w onset of tissue demise w/ >24 h a/w permanent impotence
- Cx: Hematoma, infection, systemic absorption of vasoactive agents (severe HTN), recurrence, impotence (this risk should be discussed w/ pt & is a possibility despite efforts & timeliness of therapy)

---

**EMERGENCIES IN DIALYSIS PATIENTS**


**Definition**
- Any complication involving dialysis catheters or fistulas as well as infection, electrolyte imbalances, cardiac complaints, or signs of fluid overload among others
- Common complaints & special considerations include:

<table>
<thead>
<tr>
<th>Common Chief Complaints and Special Considerations in Dialysis Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chief Complaint</td>
</tr>
<tr>
<td>-------------------------------------------------</td>
</tr>
<tr>
<td>Fever</td>
</tr>
<tr>
<td>------------------------------</td>
</tr>
<tr>
<td>Dyspnea</td>
</tr>
<tr>
<td>Chest pain</td>
</tr>
<tr>
<td>Syncope</td>
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<tr>
<td>Hypotension</td>
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<tr>
<td>Abdominal pain</td>
</tr>
<tr>
<td>Headache/AMS</td>
</tr>
<tr>
<td>Skin changes</td>
</tr>
<tr>
<td>HD access sx</td>
</tr>
<tr>
<td>Other</td>
</tr>
</tbody>
</table>

**Approach to the Patient**

**History**

› Should focus on assessing for common causes of respective chief complaints, w/ attention to special considerations unique to ESRD pt

**Physical**

› Attention to abnl vital signs
› Pulmonary & cardiac exam including assessment of friction rub, rhonchi, & rales
- Abdominal exam, esp in pts w/ PD catheters
- Extremity exam & JVP for signs of fluid overload
- Skin exam for e/o calciphylaxis
- Assess graft site for thrill & signs of bleeding, infection, edema, & bruising; assess tunneled catheter site for e/o cellulitis or underlying abscess formation

Diagnostics
- CBC, Chem 10; consider ABG w/ STAT potassium & to assess acid–base status
- Consider LFTs, BNP, cardiac markers if indicated
- Consider contacting PD access nurse for sample of PD dialysate fluid (cell count [WBC >50–100 cell/mm³ suggest peritonitis], Gram stain, culture)
- EKG for cardiac electrical instability from potential electrolyte abx, ischemia
- Consider point-of-care cardiac & lung u/s & FAST exam to assess for effusion & ascites, respectively
- Imaging: Appropriate imaging for respective complaints; Doppler imaging of AF fistula site if concern for thrombosis

Treatment
- Refer to appropriate sections for tx of conditions noted above
- Special considerations:
  - **Peritonitis**: Vancomycin 2 g AND cefepime/ceftazidime 1 g each added to 1 bag of dialysate infused into & allowed to dwell in the peritoneal cavity for 6 h
  - **Dialysis disequilibrium syndrome**: Reduce ICP (HOB elevation >30°, hyperosmolar therapy [mannitol, hypertonic saline], euglycemia, euthermia, eunatremia, MAP > 65, CO₂ 40 mmHg, CPP 50–70 mmHg); renal consult
  - **Clotted AV graft/fistula**: Immediate vascular surgery consultation for consideration of catheter-directed thrombolysis, pharmacomechanical thrombolysis, surgical thrombectomy
  - **Clotted Vascular Access Catheters**: Consult institutional policies; if feasible, attempt catheter-directed tPA via infusion of 2 mg tPA into occluded lumen & fill remainder w/ saline. After 15 min, inject 0.3 mL saline to move the active enzyme toward the tip of the catheter. After another 15 min, inject another 0.3 mL to move the active enzyme toward the tip of the catheter. After another 15 min, try to aspirate
catheter. If unsuccessful, send pt for catheter exchange.

- **Vascular Access Hemorrhage:** Apply direct pressure for 10–15 min; if occurs w/ hours of dialysis, consider protamine 1 mg per 100 U heparin received (or 10–20 mg if dose unknown) to reverse heparin anticoagulation; consider application of gelfoam, surgical, or other hemostatic agent; immediate vascular surgery consultation for uncontrolled hemorrhage

**Disposition**
- Depends upon presenting complaint, but most will invariably require admission

**Pearls**
- BP measurement over & use of AV fistula sites for blood draw/administering therapy is contraindicated
- BNP levels are not reliable in diagnosing fluid overload/HF in dialysis pts as basal BNP levels are typically elevated & increased BNP levels from baseline may not correlate w/ clinical HF
- Chronically elevated troponin common & a/w increased mortality; makes assessment of ACS challenging; however, the National Academy of Clinical Biochemistry (NACB) recommends a 20% change in troponin concentration from baseline for Dx of AMI
VAGINAL BLEEDING


History


Diagnostics

- CBC, type & screen (Rh), urine hCG; quantitative hCG if pt is pregnant; crossmatch (if heavily bleeding); consider pelvic u/s

Pearls

- Average pad holds 5–15 cc of blood
- Average tampon holds 5 cc of blood

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonpregnant</td>
<td>Abnl uterine bleeding, PCOS, IUD or oral contraceptives, endometritis, cervicitis, fibroids, uterus polyps, adenomyosis, endometrial hyperplasia or cancer, coagulopathies, postcoital bleeding</td>
</tr>
<tr>
<td>1st trimester</td>
<td>Implantation bleeding, miscarriage, ectopic pregnancy, hydatidiform mole</td>
</tr>
<tr>
<td>2nd/3rd trimester</td>
<td>Placenta previa, vasa previa, placental abruption, uterine rupture</td>
</tr>
<tr>
<td>Other</td>
<td>Postpartum hemorrhage, retained products of conception</td>
</tr>
</tbody>
</table>

Miscarriage


History

- Vaginal bleeding ± passage of clots or tissue at <20 wk; abd
pain/cramps

Physical Exam

- Speculum & bimanual exam to assess for passage of blood/POC & whether os is open or closed. (If copious bleeding, remove POC w/ gentle traction to allow uterus to clamp.)

Evaluation

- Labs: UA, quant hCG, HCT, type & screen (crossmatch if HD unstable). If products expelled, send to pathology.
- Imaging: Pelvic u/s to determine location of pregnancy

Classification of Miscarriage

- Threatened: Os closed, no passage of POC, viable fetus w/ heart tones, mild cramping/bleeding (~20% will eventually abort)
- Inevitable: Os dilated & effaced; POC not passed; cramps, moderate bleeding
- Complete: POC expelled, cervical os closed; little cramping or bleeding
- Incomplete: Some, but not all products have passed. Retained fetal or placental tissue
- Missed: Pregnancy loss after development of embryo/fetus, os closed

Treatment

- ED:
  - Supportive management: IVFs, O₂, monitoring, position on L side
  - Blood products: Transfuse if HD unstable
- Medication therapy:
  - Rh immunoglobulin: 50 mcg <12 wk, 300 mcg >12 wk if Rh-negative
  - Consult: Gyn service if HD unstable or if need for D&C anticipated (inevitable, incomplete or missed abortion)
- Home management:
  - Hormonal therapy: Methotrexate may be indicated under guidance of OB/Gyn
  - Consider prophylaxis w/ doxycycline or testing for STD if discharging home w/ open os

Disposition

- Home: Stable pts w/ complete or threatened abortion; f/u w/ OB/Gyn w/i 72 h to monitor hCG levels
- Admit: Uncontrolled bleeding or pts requiring immediate D&C

Pearl
Threatened & missed abortions can only be distinguished by pelvic u/s

**Ectopic Pregnancy**

**History**
- Unruptured: abd pain, cramping, amenorrhea or abd pain.
- Ruptured: hypotension, tachycardia, abd pain
- RFs: H/o PID, IUD, fertility tx, recent abortion or prior ectopic

**Physical Exam**
- Assess for HD stability. Signs of peritonitis if rupture has occurred.
  Speculum & bimanual exam may reveal pelvic tenderness &/or adnexal mass.

**Evaluation**
- Labs: Quant hCG, HCT, Rh screen, PT/PTT & type & crossmatch 4 U (if HD unstable)
- Imaging: Pelvic US (TVUS should identify IUP at 5.5 wk); if HD unstable, FAST exam to assess for free fluid

**Treatment**
- Supportive: 2 large-bore IVs, IVF resuscitation, monitor
- Transfusion: If HD unstable
- Rh immunoglobulin: 50 mcg <12 wk, 300 mcg >12 wk if Rh-negative
- Consult: Urgent Gyn eval for consideration of medical (MTX) vs. surgical (laparoscopy/laparotomy) tx options

**Pearl**
- Heterotopic pregnancies (co-occurrence of IUP & ectopic) have incidence of 1/30,000 in spontaneous pregnancies but 1/100 in assisted pregnancies.

**Placenta Previa and Abruptio Placentae**

**History**
- Placenta previa: Placental implantation adjacent to or over os. Presents as painless, bright red, vaginal bleeding usually after 28 wk. RFs: Multiple gestation, multiparity, advanced maternal age, previous placenta previa/C-section, maternal smoking, HTN
- Abruptio placentae: Separation of implanted placenta b/w 20 wk & delivery. Presents as painful, dark red bleeding (80%); may also present w/ signs/sxs of DIC. RFs: Eclampsia, DM, HTN, abdominal
trauma, cocaine, cigarette smoking

Physical Exam
- Check fundal height, contractions, & uterine tenderness:
  - Firm/tender uterus = placental abruption until proven o/w
- AVOID SPECULUM & VAGINAL EXAM

Evaluation
- Labs: CBC, Chem 7, LFTs, PT/PTT, fibrinogen (r/o DIC), UA, type/crossmatch 2 U
- Imaging: Doppler u/s (fetal heart tones); bedside abdominal u/s to assess placenta & signs of fetal movement, though may not always detect abruption

Treatment
- Supportive: Place on L side, 2 large-bore IVs, IVF resuscitation, monitor pt & fetus
- Transfusion: Blood products ± FFP (HD unstable or signs of DIC)
- Medications: Rh immunoglobulin 300 mcg if Rh-negative, magnesium for fetal neuroprotection if emergent delivery under 32 wk
- Consult: Urgent Gyn eval for possible STAT C-section

Disposition
- Admit: All pts to the OB service even if HD stable for close monitoring

Retained Products of Conception and Postabortion Sepsis
(Ob Gyn. 2015;125:1042)

History
- Infection of placenta &/or POC which can spread to the uterus → systemic
- Retained POC: Cramping, heavy bleeding
- Postabortion sepsis: Cramping, bloody or purulent d/c, fever

Physical Exam
- Fever, vaginal bleeding or purulent/bloody d/c, uterine tenderness

Evaluation
- Labs: Quant hCG, type & cross, preop labs
- Imaging: Pelvic u/s

Treatment
- Supportive: Stabilize (see Sepsis chapter), correct coagulopathy/anemia
Abx: If suspected infection, clindamycin 900 mg IV q8h PLUS gentamicin 5 mg/kg/d OR ampicillin 2 g q4h PLUS gentamycin PLUS metronidazole 500 mg q8h OR levofloxacin 500 mg QD PLU metronidazole OR piperacillin–tazobactam 4.5 g q8h.
Consult: Gyn service for D&C

Disposition
Admit: All pts to OB/Gyn for D&C

Postcoital Bleeding

History
- Trauma during intercourse? Vaginal d/c, assess domestic violence or abuse.
- RFs: Cervical abnormalities, STDs, postmenopausal

Physical Exam
- Ongoing bleeding; vaginal lacerations, abrasions

Evaluation
- Labs: Urine hCG, GC/Chlamydia testing; HCT

Treatment
ED:
- Abx: Treat STI appropriately (see Vaginal Discharge below)
- Consult: Gyn service for laceration requiring extensive repair; social services if concern for domestic violence

PREECLAMPSIA AND ECLAMPSIA


Definition
- Chronic HTN: Systolic BP >140/90 before 20 wk gestation or longer than 12 wk postpartum
- Gestational HTN: BP >140/90 on 2 occasions after 20 wk gestation.
- Preeclampsia: Gestational HTN & proteinuria, can be classified as mild to severe based on end-organ damage
- Eclampsia: Preeclampsia w/ szs or coma; generally 3rd trimester or postpartum

Approach to the Patient
History
- HA, visual disturbances, mental status changes, abd pain, edema.
  ROS plural gestation? PMH (prior preeclampsia, nulliparity, extremes of age, HTN, obesity, antiphospholipid antibody syndrome, DM, chronic renal dz, connective tissue disorder)

Physical Exam
- HTN, abdominal tenderness, hyperreflexia/clonus, peripheral edema, papilledema, AMS

Evaluation
- UA, CBC, Chem 7, LFTs, LDH, uric acid, coags, type & cross, fetal/maternal monitoring

Treatment
- BP: Hydralazine, labetalol, or nifedipine (goal BP <140/90)
- Sz prophylaxis: Magnesium 2–6 g IV load + 1–2 g/h
- Szs: Magnesium (2–4 g IV q5–10min); refractory szs: Diazepam (5 mg IV q5min up to 20 mg) OR phenobarbital (200 mg IV)
- Consult: Gyn for all pts; delivery = only definitive tx for eclampsia

Disposition
- Home: Mild preeclampsia; schedule OB f/u in 24 h
- Admit: Eclamptic & most severe preeclamptic pts need urgent delivery (pending BP & sz control) & ICU admission

HYPEREMESIS GRAVIDARUM

(ORIGINAL TEXT CONTINUED)
Disposition: Home if tolerating PO, admit if severe dehydration

---

**EMERGENCY DELIVERY**


**Definition**
- True labor: Regular uterine contractions of increasing intensity at decreasing intervals
- 1st stage: Cervical dilatation & effacement (up to 12 h)
- 2nd stage: Complete cervical dilatation, culminating in delivery (up to 2 h)

**Approach to the Patient**

**History**
- Frequency & intensity of contractions, rupture of membranes, fetal movement, has pt had prenatal care for eval of cx of pregnancy, screening tests, etc.

**Physical Exam**
- External exam: Assess for crowning or active bleeding (if so, defer speculum/bimanual exam)
- Sterile speculum exam: Confirm ROM by checking for ferning &/or Nitrazine test
- Bimanual exam: Assess cervical effacement & dilatation (10 cm = complete), position, presentation (fetal part in canal), lie (relation of long axis to mother → longitudinal or transverse), & station (−3 to +3; 0 is at level of ischial spines); cord prolapse?

**Diagnostics**
- Abdominal u/s if placenta previa of concern

**Treatment**
- Basics of Delivery
  - Cord prolapse: Manually place hand in vaginal vault, lift presenting part away from cord; place pt in knee–chest position or deep Trendelenburg. Administer tocolytics (magnesium 4–6 g IV, terbutaline 0.25 mg SQ).
  - Vaginal delivery: Place mother in lithotomy position; cleanse/drape perineum if possible; w/ contractions, ask mother to “bear down”
Head: One hand on occipital area & other on perineum, maintain fetal head in flexed position; if cord wrapped at neck reduce over head or bring cord caudally over shoulders & deliver baby through cord. In extreme circumstances can cut cord first.

Shoulders: Rotate head & exert gentle pressure until anterior shoulder delivered; lift head upward to deliver posterior shoulders, attempt to guide posterior shoulder over perineum.

Body: Support head & catch body w/ the other hand. Suction mouth & nose.

Cord: Clamp cord twice & cut, send cord blood for serology & Rh. Clamp cord 1–3 cm distal to navel.

Placenta: Apply pressure above symphysis w/ minimal traction on cord (too much traction will cause uterine inversion); sudden gush of blood & lengthening cord will signify imminent placental delivery.

Aftercare: Massage uterus ± oxytocin 20 U IV (can be given as 10 U IM if no IV access for ongoing hemorrhage); inspect & repair lacerations of cervix, vagina

Infant care: Suction mouth & nose, stimulate with warm blanket. BVM if no spontaneous respirations. If pulse <60 start CPR, neonatal resusc per PALS. Obtain Apgar scores at 1 & 5 min.

Shoulder Dystocia:

- McRoberts: Hyperflexion of hips to abdomen w/ external rotation & slight abduction
- Rubin I maneuver: Downward pressure just proximal to symphysis pubis
- Woods screw maneuver: Insert hand into vagina & apply pressure to anterior aspect of posterior shoulder to abduct/extend shoulder & free it
- Delivery of the posterior arm: Insert hand into vagina, flex posterior arm of the fetus, bringing it across the chest. Deliver posterior arm & then rotate fetus out
- Gaskin position: Place Mom in hands-and-knees position, allows gravity to help open space

Breech: Ideally OB present, or delivery in OR for c/s. If imminent, touch fetus as little as possible & let delivery happen spontaneously, do not pull on fetus which can entrap fetal head. If head becomes entrapped, uterine relaxant like terbutaline can be given.

Perimortem delivery: >23 wk gestational age (obvious gravid uterus),
should initiate w/i 5 min of maternal arrest. (Emerg Med Clin North Am. 2012;30:937)

- Vertical incision from epigastrium to pubic symphysis & extend through all layers to the peritoneal cavity.
- Uterus is exposed & incised at bladder reflection, retract bladder caudally
- Incision extended to uterine fundus, with operator’s hand used to palpate fetal parts & prevent damage. Infant extracted, clamp & cut umbilical cord.

**FEMALE PELVIC PAIN**

(Emerg Med Clin North Am. 2011;29:621)

**History**
- Dyspareunia, vaginal bleeding or d/c? Urinary sx, ROS PMH (STDs, recent procedure) MEDS (contraceptive devices, hormonal therapy), social (domestic violence)

**Physical Exam**
- Abdominal exam; Gyn exam (d/c or bleeding, masses or tenderness)

**Diagnostics**
- Labs: UA, GC/Chlamydia, Wet mount
- Imaging: Pelvic US (assess flow, torsion, mass, fluid)

**Ovarian Cyst**

**History**
- Dull, vague, unilateral sensation of pelvic pain or dyspareunia
- Rupture: Sudden, unilateral, sharp pelvic pain; can also present as diffuse peritonitis

**Physical Exam**
- Lower quadrant abdominal tenderness, adnexal tenderness/mass, vaginal bleeding

**Evaluation**
- Labs: CBC, type & screen (crossmatch if HD unstable)
- Imaging: Pelvic u/s to assess for size, complexity, torsion, presence of free fluid. Bedside FAST if HD unstable.

**Treatment**
Supportive: IVFs, transfuse if HD unstable
Analgesia: NSAIDs, Narcotics prn
Consult: Gyn Service for persistent pain, large-volume hemorrhage

**Disposition**
- Home: Stable, pain well controlled; f/u w/ Gyn or PCP in 1–2 mo for repeat u/s to reassess size
- Admit: HD unstable

**Ovarian Torsion**

**History**
- Acutely worsening unilateral lower abd/pelvic pain, N/V
- Can present as intermittent torsion w/ intermittent sx
- RFs: Ovarian cysts, dermoid & other tumors, pregnancy

**Physical Exam**
- Nonspecific & variable; Gyn exam reveals unilateral, adnexal mass in majority of cases ± tenderness (though tenderness absent ~30% of the time)

**Evaluation**
- Labs: Urine hCG, pre-op labs
- Imaging: Pelvic US to assess for ovarian edema, cyst/mass, blood flow

**Treatment**
- Analgesia/antiemetics
- Consult: Gyn service for urgent laparoscopy

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**VAGINAL DISCHARGE (SEXUALLY TRANSMITTED INFECTION)**


**History**
- RFs: Multiple sexual partners & unprotected intercourse

**Physical Exam**
External: Inspect for lesions, ulcerations; adenopathy
Speculum: Vaginal wall inflammation/d/c; cervical inflammation/d/c
Bimanual: If cervical motion tenderness or adnexal tenderness, think PID (see below)

**Evaluation**
- Labs: GC/Chlamydia testing; wet mount

**Treatment**
- *N. gonorrhoeae*: Ceftriaxone 125 mg IM × 1
- *C. trachomatis*: Azithromycin 1 g PO × 1 OR doxycycline 100 mg PO BID × 7 d OR levofloxacin 500 mg PO QD × 7 d
- *T. vaginalis*: Metronidazole 2 g PO × 1 OR 500 mg PO BID × 7 d
- Bacterial vaginosis: metronidazole 500 mg PO BID × 7d OR metronidazole 0.75% gel intravaginally 5 g/d × 5 d OR clindamycin 2% cream intravaginally 5 g × 7 d
- Candidiasis: Topical azoles (over the counter) × 7 d OR fluconazole 150 mg PO × 1

**Pearls**
- Educate pts on safe sex practices & advise pts to tell their partners to get tested/treated
- Encourage HIV testing outpt if not offered in ED
- Drinking alcohol on metronidazole can cause disulfiram-like reaction (flushing, ↑ HR, ↓ BP)

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**PELVIC INFLAMMATORY DISEASE AND TUBO-OVARIAN ABSCESS**


**Definition**
- Spectrum of inflammatory disorders, any combination of endometritis, salpingitis, tubo-ovarian abscess & pelvic peritonitis.
- Commonly a/w gonorrhea, chlamydia but <50% of pts with PID test positive for these organisms. Involves other bacteria (eg, GNR, anaerobes) & viruses (eg, *M. genitalium*)
- Cx include abscess, perihepatitis (Fitz-Hugh–Curtis), sepsis, chronic pain, increased risk of ectopic pregnancy, infertility
**History**
- Lower abd pain, vaginal d/c, dysuria, dyspareunia, nausea ± fevers
- RFs: Age <25, multiple sexual partners, unprotected sex, h/o PID, IUD placement in the last month, recent instrumentation of the cervix, douching, smoking

**Physical Exam**
- Lower abdominal tenderness, cervical discharge, cervical friability, cervical motion tenderness, adnexal tenderness/fullness
- Clinical exam has poor sensitivity; presentation is often atypical

**Evaluation**
- Labs: Always check pregnancy test; cervical cultures, UA, CBC (not sens)
- Abdominal CT or pelvic US only required if TOA is suspected (unilateral tenderness or palpable mass, systemically ill)

**Treatment**
- Low threshold for empiric tx: Minimum criteria in sexually active young women or others at risk are pelvic pain & cervical, uterine or adnexal tenderness
- Outpt: Ceftriaxone 250 mg IM × 1 + doxycycline for 14 d
- Consider adding metronidazole for anaerobes
- Azithromycin is considered insufficient for PID; may be used in isolated cervicitis or 2nd line
- If severe PCN allergy, options are hospitalization or azithromycin &/or levofloxacin depending on regional antibiogram
- Inpt: (Cefotetan or cefoxitin) + doxycycline OR clindamycin + gentamicin
- Consult: Gyn service if concern for TOA

**Disposition**
- Admit if toxic appearing, severe vomiting, TOA, failure of outpt therapy, pregnancy, immunocompromised, young age, poor f/u w/i 72 h
- Discharged pts need f/u in 3 d to ensure sx resolving. Partners should be tested.

**Pearls**
- Given ↑ resistance to antibiotic regimens, CDC updates recommendations frequently
PID in pregnancy is rare but does happen; alternative diagnoses should be considered
**RASH**

**Definition**
- One or more skin lesions originating from a common cause (often over a short prd) & having a spec distribution & morphology

**Approach**
- HPI: Onset (timing, location); evolution (distribution, morphology); periodicity (constant vs waxing & waning, temporal associations); sxs (pain, pruritus, burning, fever, bleeding); new exposures or inciting events (topical or systemic exposures, recent travel, occupational exposures, sick contacts, animals, sexual hx)
- PMH & Meds (including immunizations, new formulations or doses, supplements, illicits)
- Ensure a good ROS (rashes can be first sign of an occult internal process)
- PE: Determine distribution, shape (if applicable), morphology, & secondary changes
  - Distribution: Localized/grouped/regional/generalized, central/peripheral, flexor/extensor surface, dermatomal, acral, intertriginous, follicular, mucosal, sun-exposed areas
  - Shape (if applicable): Annular (ring), round/nummular/discoid (coin), targetoid, arcuate (arc), linear, serpiginous, reticular (net-like/lacey), whorled (marble-like), polycyclic (coalescing circular/ring-shaped lesions)
  - Morphology & secondary changes: See tables

<table>
<thead>
<tr>
<th>Common Dermatologic Morphologies</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Flat</strong></td>
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<tr>
<td>Macule</td>
</tr>
<tr>
<td><strong>Patch</strong></td>
</tr>
<tr>
<td><strong>Raised</strong></td>
</tr>
<tr>
<td>Papule</td>
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<tr>
<td><strong>Plaque</strong></td>
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<tr>
<td><strong>Nodule</strong></td>
</tr>
<tr>
<td><strong>Wheal</strong></td>
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<tr>
<td><strong>Fluid-filled</strong></td>
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<tr>
<td><strong>Pustule</strong></td>
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<tr>
<td><strong>Bulla</strong></td>
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<tr>
<td><strong>Cyst</strong></td>
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<tr>
<td><strong>Vascular</strong></td>
</tr>
<tr>
<td><strong>Purpura</strong></td>
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<tr>
<td><strong>Depressed</strong></td>
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<tr>
<td><strong>Erosion</strong></td>
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<tr>
<td><strong>Ulcer</strong></td>
</tr>
</tbody>
</table>

### Common Secondary Changes

| **Scaling** | Thickened outer epidermis (stratum corneum), usually white |
| **Crusting** | Dried liquid debris (eg, serum, blood, exudates), usually yellow–brown |
| **Lichenification** | Thickening of epidermis w/ accentuated skin lines/markings |
| **Excoriation** | Superficial abrasions, usually due to scratching |

**Typical Manifestations of Common or Critical Acute Disseminated Rashes**
## Viral Etiologies

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute HIV</td>
<td>Pink <strong>maculopapular</strong> 2–3 wk after initial infxn; a/w const sx</td>
</tr>
<tr>
<td>Dengue fever</td>
<td>Pink <strong>maculopapular/confluent macules</strong> w/islands of sparing a/w high fever, HA, retro-orbital &amp; severe body pain; lasts 2–3 d</td>
</tr>
<tr>
<td>Measles</td>
<td>Pink <strong>maculopapular</strong>, starts behind ears &amp; face/neck, spreads to trunk &amp; extremities (w/ palms/soles) w/ progression to trunk/buttocks (can last 3 wk)</td>
</tr>
<tr>
<td>Mononucleosis</td>
<td>Pink maculopapular; no palms/soles; a/w const sx</td>
</tr>
<tr>
<td>Parvovirus B19</td>
<td>Red (“slapped”) cheeks w/ circumoral pallor (lasts 1–4 d), then generalized <strong>reticular/face</strong> rash, esp extensor surfaces (spares palms/soles) w/ progression to trunk/buttocks (can last 3 wk)</td>
</tr>
<tr>
<td>Pityriasis rosea</td>
<td>Pink/salmon small oval <strong>plaques</strong> distributed on lines of cleavage on trunk/prox extremities (spares face, palms, soles); often 1–3 wk after herald patch (single 2–4 cm pink plaque w/ fine scaling borders &amp; depressed pale center); rash lasts 5 wk – 5 mo</td>
</tr>
<tr>
<td>Roseola</td>
<td><strong>Pink macules</strong>; starts neck/trunk after defervescence &amp; spreads to face/extremities; lasts 1–2 d</td>
</tr>
<tr>
<td>Rubella</td>
<td>Pink <strong>maculopapular</strong>, starts on face/forehead then to trunk/extremities, ± <strong>coalesce</strong>; a/w fever, HA, arthralgias; lasts ~3 d</td>
</tr>
<tr>
<td>Varicella (Chicken pox)</td>
<td>Pruritic <strong>macules</strong>, progress to <strong>papules &amp; vesicles</strong>, crusting w/i 48 h; trunk/face &gt; extremities; ± mucous membranes; crusts fall off after 1–2 wk; may leave hypopigmented scars long-term</td>
</tr>
</tbody>
</table>

## Bacterial Etiologies

<table>
<thead>
<tr>
<th>Condition</th>
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</thead>
<tbody>
<tr>
<td>Gonococcemia</td>
<td>Few scattered <strong>hemorrhagic pustules</strong> (often over joints) occurring after mucosal infxn; a/w arthralgias &amp; low-grade fever; tx w/ CTX (1 g IV QD × 7d) &amp; azithromycin (1 g PO ×1); admx</td>
</tr>
<tr>
<td>Leptospirosis</td>
<td>Initially warm &amp; flushed, can develop <strong>transient petechia</strong>, later <strong>purpura</strong>; a/w fever, HA, myalgias, GI sx, subconjunctival hem; sx can be bimodal; severe dz (Weil syndrome) a/w liver failure</td>
</tr>
<tr>
<td>Lyme</td>
<td>Mx <strong>erythema migrans</strong> in 20% of primary lyme; Secondary lyme (3–10 wk after infxn) small pink oval <strong>macules/patches</strong>, a/w neuro (CN), visual, cardiac (get ECG), msk complications</td>
</tr>
<tr>
<td>Meningococcemia</td>
<td>Rapidly progressive <strong>petechia &amp; purpura</strong>; pt toxic-appearing; ±2–3 d prodrome of HA, URI sx (but 20% pts present w/ sepsis); tx w/ CTX × 7d; mortality 10–15% w/ tx (Intern Med 2016;55(6):567–572)</td>
</tr>
<tr>
<td>RMSF</td>
<td>Numerous red <strong>macules w/ central petechia</strong> start on wrists/ankles, then palms/soles, then arms/legs/trunk; 10–15% may not have rash; a/w abrupt fever, severe HA, N/V, abd pain, myalgias</td>
</tr>
<tr>
<td>Condition</td>
<td>Description</td>
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<tr>
<td>Scarlet fever</td>
<td>Diffuse fine erythematous <em>coalescent</em> “sandpaper” eruption; starts on neck &amp; spreads to trunk/extremities, becomes <strong>macules coalescing into patches</strong>; flushed face w/ circumoral pallor; a/w recent strep infxn; lasts 7 d then fine desquamation</td>
</tr>
<tr>
<td>Staph scalded skin syndrome</td>
<td>Diffuse painful <strong>light erythema w/ widespread exfoliation</strong> of thin sheets of skin; no mucous membranes; a/w malaise, fever</td>
</tr>
<tr>
<td>Secondary syphilis</td>
<td>Diffuse <strong>macules, papules (± pustules)</strong>, including on palms/soles &amp; mucous membranes</td>
</tr>
<tr>
<td>Toxic shock syndrome</td>
<td>Diffuse erythematous <strong>coalescent “sandpaper” macules &amp; patches, ± hemorrhagic bullae</strong>; mucous membranes involved; a/w fever, GI sx, confusion, multiorgan failure</td>
</tr>
</tbody>
</table>

### Arthropod Bites

<table>
<thead>
<tr>
<th>Condition</th>
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</thead>
<tbody>
<tr>
<td>Bed bugs</td>
<td>Painless pruritic red <strong>papules</strong> on exposed areas; occasionally wheals, hemorrhagic nodules, vesicles 2/2 bug proteases</td>
</tr>
<tr>
<td>Lice</td>
<td>Painless pruritic red <strong>papules &amp; wheals</strong>, often concentrated in covered areas (axilla/groin/trunk) &amp; sparing extremities</td>
</tr>
<tr>
<td>Scabies</td>
<td>Painless pruritic red <strong>papules</strong>, often clustered, starts on hand/foot, spreads to trunk; look for intertriginous burrows</td>
</tr>
</tbody>
</table>

### Vascular/Hematologic

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>HSP</td>
<td>Erythematous <strong>macules &amp; papules</strong> becoming <strong>purpuric</strong> (palpable purpura) symmetrically on lower extremities; spares soles; a/w abd pain &amp; joint pain</td>
</tr>
<tr>
<td>ITP</td>
<td><strong>Petechia</strong>, esp in lower extremities &amp; palette; can be asx</td>
</tr>
<tr>
<td>TTP</td>
<td><strong>Petechia</strong>, a/w fever, AMS/neuro deficits, ± jaundice</td>
</tr>
<tr>
<td>DIC</td>
<td><strong>Petechia, purpura, hemorrhagic bullae</strong>, acral cyanosis, localized necrosis/gangrene (inc. extremities); a/w multiorgan failure</td>
</tr>
</tbody>
</table>

### Hypersensitivity Reactions

<table>
<thead>
<tr>
<th>Condition</th>
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</thead>
<tbody>
<tr>
<td>Acute generalized pustular psoriasis</td>
<td>Diffuse small sterile pruritic pustules on erythematous base, a/w fever; trunk &amp; intertriginous areas common (no mucous membranes); may have multiorgan dysfxn</td>
</tr>
<tr>
<td>Allergic contact dermatitis</td>
<td><strong>Poison Ivy/Oak</strong>: Wheals &amp; vesicles (± bullae) on exposed areas <strong>Topical (eg, hair dye)</strong>: <strong>Vesicles &amp; papules</strong> w/ crusting, edema</td>
</tr>
<tr>
<td>Bullous pemphigoid</td>
<td>Acute or subacute pruritic, diffuse, tense <strong>bullae</strong> on nl, erythematous, or urticarial base; many causal associations (diuretics, NSAIDs, captopril); bx dx</td>
</tr>
</tbody>
</table>
| DRESS Syndrome | **Maculopapular, papulosquamous, pustular, bullous, or urticarial** exanthem; a/w fever, LAN, systemic sx; 2/2 drug
<table>
<thead>
<tr>
<th>Condition</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Erythema multiforme</strong></td>
<td>Diffuse erythematous <strong>macules/papules</strong> w/ evolving morphology (become targetoid, then polycyclic &amp; annular configuration); trunk, extremities (inc palms/soles), face (mucous memb ~70%)</td>
</tr>
<tr>
<td><strong>Erythema nodosum</strong></td>
<td>Painful pink/purple, round, oval <strong>nodules</strong> (1–6 cm diam), can coalesce; symmetric &amp; usually anterior tibia (also knees, ankles, thighs, forearms); self-resolves after 1–6 wk</td>
</tr>
<tr>
<td><strong>Erythroderma</strong></td>
<td><strong>Generalized erythema</strong> (&gt;90% TBSA; spares palms/soles), progresses to scaling &amp; desquamation; pruritic; often w/ edema; LAN ± e/o high-output HF, hepatomegaly, splenomegaly</td>
</tr>
<tr>
<td><strong>Morbilliform drug eruption</strong></td>
<td>Diffuse erythematous morbilliform <strong>macules/papules</strong> (less commonly erythroderma, pustules, targetoid lesions), can coalesce &amp; become edematous; trunk, extremities, face</td>
</tr>
<tr>
<td><strong>Pemphigus vulgaris</strong></td>
<td>Diffuse small or confluent painful <strong>flaccid blisters &amp; erosions</strong> on erythematous base (+Nikolsky); inc on mucous membranes</td>
</tr>
<tr>
<td><strong>SJS/TEN</strong></td>
<td>Diffuse erythematous dusky confluent purpuric <strong>macules</strong> or <strong>patches</strong>, rapidly evolve to coalesce &amp; blister (+Nikolsky); often starts on trunk &amp; spreads to extremities (inc palms/soles) &amp; face (inc mucous membranes); eventually generalized sloughing</td>
</tr>
<tr>
<td><strong>Scromboid</strong></td>
<td>Diffuse <strong>macular</strong> or <strong>papular</strong> erythema w/ urticaria &lt;30 min after ingesting scombroid fish; a/w HA, N/V, palpitations</td>
</tr>
<tr>
<td><strong>Serum-sickness &amp; serum sickness-like rxn</strong></td>
<td>Diffuse <strong>urticarial</strong> or serpiginous <strong>macules &amp; patches</strong>, well-demarcated w/ intense red border &amp; central clearing; trunk, face, extremities (no palms/soles), a/w arthralgias</td>
</tr>
<tr>
<td><strong>Sweet syndrome</strong></td>
<td>Tender, violaceous, well-demarcated painful <strong>papules &amp; plaques</strong> (± central pustules, bullae, or ulcers), can evolve to coalesce; common esp on upper body (inc face, mucous memb), a/w fever</td>
</tr>
<tr>
<td><strong>Urticaria</strong></td>
<td>Pruritic, pink-erythematous <strong>wheals</strong>, ranging in size from a few mm to several cm in size; may be round or irregular in shape</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Guttate psoriasis</strong></td>
<td>Diffuse dewdrop-like 1–10 mm salmon-colored <strong>papules</strong> w/ fine scaling; can coalesce; may be preceded by URI or grp A strep infxn</td>
</tr>
<tr>
<td><strong>Photosensitive rash</strong></td>
<td><strong>Erythema</strong> or <strong>bulla</strong> (inc hemorrhagic) at area of UV exposure</td>
</tr>
<tr>
<td><strong>Pityriasis versicolor</strong></td>
<td>Subacute hypo- or hyperpigmented <strong>macules &amp; patches</strong>, often neck/trunk/UEs; 2/1 <em>malassezia</em> fungus, not contagious; RFs are humidity, immunosupp, OCPs, poor nutrition; tx topically for localized dz (azole, terbinafine), systemic for extensive dz (azoles)</td>
</tr>
</tbody>
</table>
Measles (Rubeola; “First Disease”)

Definition (Lancet 2012;379(9811):153–164)

- Highly contagious dz caused by the measles virus, spread by droplet contact
- Epidemiology: Usually nonimmunized or immunosuppr (1° vaccine failure <0.2%); can occur in immunized adults (2° vaccine failure 5% >15 yr after vaccination) esp if no herd immunity (though often milder); winter/spring common; incubation prd ~1 wk

History & Physical Exam

- Assess vaccination status
- Prodrome (lasts 3 d): Acute febrile illness, cough, coryza (nasal mucosal inflammation), conjunctivitis, Koplik spots (small irreg-shaped blue-white macules on buccal mucosa)
- Exanthem (lasts 3–7 d): Starts behind ears, on face/neck as discrete purple-red macules & papules; spreads to trunk & extremities (inc palms/soles), becoming confluent; disappears in same order as arrived

Evaluation

- Routine labs rarely indicated (CBC may show leukopenia)
- Lab confirmation: Measles serologies (enzyme immunoassay for measles IgG & IgM), throat or nasopharyngeal swab for viral isolation/RT-PCR. Contact lab specialist.

Treatment

- Supportive, consult ID
- Two-dose Vit A may reduce mortality in children <2 yr (Cochrane 2005; (4):CD001479)

Complications

- During/post-infxn: Otitis media (most common) & mastoiditis, keratitis, corneal ulcerations & blindness, croup, PNA (most common severe complication), myopericarditis, TTP, febrile sz, encephalomyelitis (1:1000 incidence ~2 wk after infxn; 2/2 autoimm demyelination; fever/sz / various neuro sx) (Lancet 2012;379(9811):153–164)
- Late complications (rare): Inclusion body encephalitis (mo after infxn; fatal), subacute sclerosing panencephalitis (yrs after infxn; fatal)
Disposition
- Home if absence if cx
- Notifiable infectious dz at national level by CDC; requires notification w/i 24 h

Rubella (German Measles; Three-day Measles; “Third Disease”)

Definition
- Contagious dz of childhood caused by rubella virus, spread by droplet contact
- Epidemiology: Usually children, but adults can get too (long-term vaccine failure <10%); winter/spring common; incubation prd 2–3 wk (Lancet 2004;363(9415):1127–1137)
- Rubella infxn in children/adults is distinct from congenital rubella syndrome (CRS), a severe teratogenic congenital infxn (not discussed here)

History & Physical Exam
- Assess vaccination status
- Prodrome: Malaise, low-grade fever, HA, sore throat, adenopathy, arthralgias
- Exanthem: Erythematous macules & papules begin on face/forehead, spread to trunk/extremities, may coalesce; typically lasts 3 d then resolves

Evaluation
- Routine labs rarely indicated (CBC may show leukopenia, thrombocytopenia)
- Lab confirmation: Rubella serologies (enzyme immunoassay, latex agglutination, IFA), throat or nasopharyngeal swab for viral isolation/RT-PCR. Contact lab specialist.
  - Confirmation important in pregnant patients

Treatment
- Supportive

Complications (Lancet 2004;363(9415):1127–1137)
- During/post-infxn: Inflammatory arthritis (most common), encephalopathy (1:5000–1:30,000), transient thrombocytopenia, hemolytic anemia (rare), GBS
- If pt is pregnant: Congenital rubella syndrome (highest risk in first
Disposition

- Home, avoid contact w/ pregnant women (severe congenital defects)
- Notifiable infectious dz at national level by CDC; requires notification w/i 24 h

**Erythema Infectiosum (“Fifth Disease”)**

**Definition**

- Highly contagious dz caused by Parvovirus B19, spread by respiratory droplets
- Epidemiology: Mainly school-age children (2–14 y/o); winter/spring common; incubation prd 1–2 wk; transmission via blood products rare (virus lives in RBC precursor)

**History & Physical Exam**

- Prodrome: Malaise, low-grade fever, HA, arthralgias
- Exanthem: Intensely red face (“slapped cheek”) w/ circumoral pallor (lasts 1–4 d), then generalized reticular/lacey rash, esp extensor surfaces (spares palms/soles) w/ progression to trunk/buttocks (can last 3 wk); rash uncommon in adults

**Evaluation**

- Routine labs rarely indicated; consider CBC, reticulocyte count, & haptoglobin in pts w/ hx hemolytic anemia (eg, hereditary spherocytosis) or hemoglobinopathies (ie, SCD)
- Lab confirmation: Serologic testing, DNA assays (direct hybridization)

**Treatment**

- Supportive
- If complicated (see below), can consider course of IVIG in consultation w/ hematology

**Complications**

- During infxn: Inflammatory arthritis (most common, esp adults), transient aplastic crisis (esp in pts w/ hemolytic anemias & hemoglobinopathies), pure red cell aplasia
- Parvovirus temporarily shuts down RBC production during infxn
- In pregnant pts, risk of fetal loss 5–10%; greatest in 2nd trimester (*CMAJ* 2005;172(6):743)

**Disposition**
Home; CDC does not recommend avoidance of school or workplace.

**Roseola Infantum (Exanthema Subitum; “Sixth Disease”)**

**Definition**
- Dz of children 6–36 mo (95%) caused by HHV-6 & HHV-7, spread by salivary secretions
- Three stages of infxn: acute, latent, reactivation
- Epidemiology: Acute dz is most common viral exanthem in children <3 yr (10–20% of all acute febrile illnesses in this age), no seasonality, incubation prd 1–2 wk; Reactivation can be severe in immunosupp (esp recent HSCT) *Clin Microbiol Rev* 2015;28(2):313–335.

**History & Physical Exam**
- Prodrome: Abrupt high fevers (± febrile sz), HA, coryza, periorbital edema
- Exanthem: Begins after defervescence; erythematous macules; starts neck/trunk & spreads to face/extremities; clears in 1–2 d

**Evaluation**
- Routine labs rarely indicated (CBC may show leukopenia)
- Primary infxn w/ HHV-6 is difficult to confirm diagnostically

**Treatment**
- Supportive for primary infxn
- Reactivation in immunosuppressed: May tx w/ ganciclovir or foscarnet

**Complications**
- Primary infxn: febrile sz (most common complication)
- Reactivation infxn (most severe in transplant pts, esp recent HSCT): encephalitis, bone marrow suppression, pneumonitis, hepatitis, transplant failure, GVHD

**Disposition**
- Home

**Herpes Simplex 1 and 2 (HHV-1 and HHV-2)**

**Definition**
- Historically, HSV-1 a/w orofacial dz & HSV-2 a/w genital dz; now both can cause both dz; transmission by contact w/ active lesions, but also by resp droplets & infected secretions
- Three stages: primary, latent (asx), reactivation; reactivation triggers include illness or fever, menstruation, sun exposure, psychological
stress (but usually spontaneous)

- **Herpes gingivostomatitis:** Affects oral/perioral mucous membranes, usually 1° infxn
- **Herpes labialis:** Affects perioral skin & mucous membranes; difficult to distinguish 1° & 2° dz; latent virus lives in trigeminal ganglion; reactivation in >1/3 pts
- **Herpes genitalis:** Affects genitals (inc suprapubic, perineum, thighs, perianal) & mucous membranes; difficult to distinguish 1° & 2° dz; 60–70% of primary infxn can be asx; reactivation is common & can be sx (1-yr reactivation 20–50% [HSV-1], 70–90% [HSV-2]) or asx (80–90% of HSV-2 pts have transient asx shedding); up to 25% of infected pts unaware they have dz *(NEJM 2016;375(7):666–674)*

**History & Physical Exam**

- Herpes gingivostomatitis & labialis:
  - Prodrome: Malaise, fever, localized pruritus/tingling/burning, dysphagia if intraoral
  - Rash (gingivostomatitis): Oral/perioral vesicles, oral ulcers, gingivitis (lasts 1–2 wk)
  - Rash (labialis): Clustered vesicles on erythematous base, often outside of mouth at vermillion border (but can be on nose); ± LAN & sore throat; distinguish from aphthous stomatitis (canker sore), which are discrete painful intraoral lesions
- Herpes genitalis:
  - Prodrome: Malaise, fever, HA, tender LAN; localized burning/pain in genital region
  - Rash: Clustered vesicles on erythematous base, crusted ulcers if on dry skin
  - Risk factors: Number lifetime sexual partners, mx partners, h/o STI/HIV

**Evaluation**

- Generally not needed in ED; if recurrent, determining HSV-1 vs HSV-2 guides prog & tx
- PCR (best), Tzanck smear (can’t differentiate HSV-1 vs HSV-2), viral cx (slow), biopsy

**Treatment**

- Pain control, hydration
- Herpes labialis: Topical therapy (docosanol 10% cream, penciclovir 1%
cream, acyclovir 5% ointment, cidofovir 0.3 or 1% gel) & oral therapy (Acyclovir, Famciclovir, Valacyclovir) may decrease sx & time to healing; sunscreen may decrease relapses.

- Herpes genitalis: Tx differs for first infxn, recurrent infxn, & suppressive tx
  - First infxn: Acyclovir 400 mg TID, Famciclovir 250 mg TID, Valacyclovir 1 g BID (7–10 d)
  - Recurrent: Acyclovir 400 mg TID, Famciclovir 125 mg BID, Valacyclovir 1g QD (5 d)
- Severe or disseminated dz: IV acyclovir 5–10 mg/kg q8h

**Complications:**

- Bacterial superinfxn (eg, impetigo), keratitis (2/2 autoinoculation), disseminated dz (eg, meningoencephalitis, hepatitis, pneumonitis) esp in neonates & immunosupp

**Disposition**

- Home unless severe/disseminated (requires IV antivirals) or unable to tolerate PO
- Herpes genitalis pts should be counseled on safe sex & prevention  
  *(MMWR 2010;59:1–110)*

**Varicella (“Chickenpox,” HHV-3)**

**Definition**

- Primary infxn w/ VZV; highly contagious (~90% transmission among household contacts; 10–35% w/ limited exposure); transmission by resp droplets or vesicle secretion
- Epidemiology: Mostly children (5–10 y/o), but can affect infants & adults (esp if from tropical regions, 2/2 childhood dz less common); mortality low, but ~4× higher in infants & ~25× higher in adults, including most among immunocompetent; winter/spring common; incubation prd ~14 d  
  *(Lancet 2006;368(9544):1365–1376)*

**History & Physical Exam**

- Exanthem: Pruritic macules, progress to papules & vesicles, crusting w/i 48 h; trunk/face > extremities; crusts fall off after 1–2 wk; may leave hypopigmented scars long-term
Mucous membranes can be involved: conjunctiva, genitals, oropharynx
“Breakthrough varicella” in immunized pts is similar but mild (ie, <50 lesions)

**Evaluation**
- Routine labs rarely indicated (CBC may show lymphopenia & transaminitis)

**Treatment**
- Healthy children: Supportive (calamine lotion, colloidal oatmeal baths; AVOID salicylates 2/2 Reye syndrome); oral acyclovir w/i 24 h of dz onset may reduce fever by 1 d & sx severity by 15–30%, but not recommended by CDC ([Lancet](https://doi.org/10.1016/S0140-6736(06)68722-8) 2006;368(9544):1365–1376)
- High-risk groups (infants, age >12, pregnant, steroids (inc inhaled) or any immunosupp, chronic skin or pulm dz, long-term ASA use, pregnant) or complicated dz: IV acyclovir
- Precautions: put pts on airborne (neg pressure) & contact precautions until crusted
  - Pts should not be managed by providers w/o immunity or those who are pregnant

**Complications**
- Bacterial superinfxn (impetigo, cellulitis; most common); invasive bacterial infxns (PNA, arthritis, osteomyelitis, necrotizing fasciitis, sepsis) & varicella PNA; neuro cx (cerebellar ataxia [1:4000], encephalitis, myelitis); heme cx (thrombocytopenia, purpura fulminans); vasculitis (inc intracranial); hepatitis ([Lancet](https://doi.org/10.1016/S0140-6736(06)68722-8) 2006;368(9544):1365–1376)
- In rare cases, maternal varicella in early gestation can result in a congenital varicella syndrome (microcephaly, mental retardation limb hypoplasias, cutaneous defects, etc.); later in pregnancy, varicella can cause preterm delivery & neonatal varicella

**Postexposure Prophylaxis**
- Antivirals not recommended for PEP
- If eligible for VZV vaccine: give vaccine w/i 3–5 d of exposure, if no e/o prior immunity
- If not eligible for VZV vaccine (allergy, immunosupp, pregnancy, infant): Varicella zoster immune globulin can prevent varicella or lessen severity, give w/i 96 h of exposure

**Disposition**
- Home for uncomplicated cases
Admission for high-risk groups or those w/ complications

Herpes Zoster ("Shingles," HHV-3)

**Definitions** *(NEJM 2013;369(3):255–263; Cochrane 2008;1:CD005582)*

- Reactivation of VZV from sensory ganglia; 20–50% lifetime risk (if nonvaccinated)
- **Herpes zoster ophthalmicus:** (V1 branch of CN V) Rash on forehead, periocular, nose
- **Herpes zoster oticus (Ramsay Hunt syndrome):** (CN VII/geniculate ganglion) Rash on ear, hard palate, anterior 2/3 of tongue; can get ipsilateral facial nerve palsy; a/w variable other CN findings (tinnitus, hearing loss, N/V, vertigo, nystagmus, etc.)
- Zoster sine herpete: Clinical features similar to VZV but w/o rash

**History & Physical Exam**

- Risk factors: Previous VZV infxn, age, immunosupp, neoplastic disease (esp hematologic)
- Prodrome (may be absent): 2–3 d of localized skin sensations (tingling, hot/cold sensation, pruritus, burning pain) prior to rash; can be a/w HA, photophobia, malaise,
- Exanthem: Grouped vesicles on erythematous base, eventually crusting; pain; distributed in dermatomal pattern, not crossing midline; overlap adjacent dermatomes in 20% cases
- Sensory changes vary: Paresthesias (tingling), dysesthesia (altered), allodynia (pain), hyperesthesia (exaggerated), pruritus

**Evaluation**

- Clinical dx; testing may be indicated if rash is atypical or pt has comorbidities
- DFA for VZV Ag (~80% Se), PCR (lesion base) (95–100% Se) *(NEJM 2013;369(3):255–263)*

**Treatment**

- Antivirals indicated if w/i 72 h of rash onset, but recommended even >72 h if new vesicles forming, complications present (inc eye), or pt risk factors (immunosupp, elderly)
- Antivirals (valacyclovir, acyclovir, famciclovir) decrease course & neuralgic pain
  - Acyclovir 800 mg PO q4h 5 times daily × 7–10 d
  - Valacyclovir 1000 mg PO q8h × 7 d (may have better bioavailability
than acyclovir)
• Famciclovir 500 mg PO q8h × 7 d (may have better bioavailability than acyclovir)
  ‣ Corticosteroids: Data equivocal; may accelerate healing & possibly reduce pain; may not help prevent postherpetic neuralgia (NEJM 2013;369(3):255–263; Cochrane 2008;1:CD005582)
  ‣ Herpes zoster ophthalmicus: Consult ophthalmology
  ‣ Supportive care (NSAIDs/acetaminophen; may need opiates during acute rash)
  ‣ Postherpetic neuralgia: tx disappointing (<50% pts have >50% reduction in pain); topical agents (lidocaine patch [NNT 2.0], capsaicin cream [NNT 3.3]), systemic tx (gabapentin [NNT 4.4], pregabalin [NNT 4.2], TCAs [NNT 2.6]); combo tx better than mono-tx (if tolerated); pain specialist c/s if considering opiates (NEJM 2014;371(16):1526–1533)

• Postherpetic neuralgia (pain >90 d after rash, can be long-term; ~20% incidence after VZV, risk inc w/ age); bell's palsy; transverse myelitis, cerebrovascular disease; disseminated dz (pneumonitis, hepatitis, pancreatitis, CNS) esp in immunosupp
• Herpes zoster ophthalmicus: ~50% can have ocular comp (eg, iritis, episcleritis, keratitis)

**Disposition**
• Home unless disseminated

**Pityriasis Rosea (associated HHV-6 and HHV-7)**

**Definition**
• Acute, self-healing exanthema of unclear etiology: may be viral (a/w HHV-6 & HHV-7), but can also be a/w drugs (esp if no herald patch & longer duration) (BMJ 2015;351:h5233)
• Epidemiology: Mainly adolescents/young adults (age 10–35); a/w asthma, eczema, URIs

**History & Physical Exam**
• Const sx in only ~50%: Fever, HA, arthralgia, cough, N/V, LAN (BMJ 2015;351:h5233)
• Herald patch present in 40–75%: Single pink/salmon-colored oval plaque 2–4 cm diam w/ fine scaling borders & pale depressed center; precedes rash by up to 3 wk
Exanthem: Numerous lesions similar in appearance to herald patch on trunk & prox. extremities but in characteristic lines of cleavage (“Christmas tree pattern”); spares face, scalp, palms, & soles typically; lasts 5 wk but can last up to 5 mo

Treatment
- Supportive (oatmeal baths & emollients may help): No recommended role for steroids, abx, or antivirals including acyclovir. In severe cases, topical agents may be tried locally, & if improvement can then use widely. (BMJ 2015;351:h5233)

Disposition
- Home; can f/u w/ dermatology esp if >3 mo duration

Molluscum Contagiosum (associated Poxvirus)

Definition
- Benign, self-limiting but long-lasting eruption 2/2 poxvirus; spread by fomite, skin-to-skin, & sexual contact & auto-inoculation
- Can serve as marker or opportunistic infxn in pts w/ HIV

History & Physical Exam
- Exanthem: Nonpainful smooth tan dome-shaped papules (2–5 mm diam) w/ umbilicated center on face, trunk, extremities (but can see in axilla, groin, a/c fossa, etc.); can last up to 12–18 mo

Treatment
- Self-limited & asx: no tx needed; can refer to dermatology for lesion eradication to dec risk of spread (cryotherapy, laser, curettage, imiquimod cream, trichloroacetic acid, or tretinoin), esp if numerous or HIV+ (Curr Opin Pediatr 2016;28(2):250–257)

Disposition
- Home ± dermatology f/u

BACTERIAL EXANTHEMS

Refer to Chapter 4 (“Soft Tissue Infections”) for the following: Cellulitis, Erysipelas, Staph Scalded Skin Syndrome, Toxic Shock Syndrome, & Necrotizing Fasciitis

Scarlet Fever (Scarlatina, “Second Disease”)
Definition
- Rash in children (3–12 yr) 2/2 erythrogenic toxin-producing strains of gpr A β-hemolytic streptococci; transmitted via airborne droplets & fomites from ppl w/ dz & asx carriers; incubation prd 1–4 d; winter/spring common

History, Physical Exam, & Evaluation
- PRODROME: Acute onset sore throat, fever, HA, vomiting, ± abd pain (can be severe)
- EXAM: Diffuse fine erythematous coalescent “sandpaper” eruption (“goosebump” appearance); starts on neck/axilla/groin & spreads to trunk/extremities (w/o palms/soles), becomes macules coalescing into patches; lasts 7d then fine desquamation
- Characteristic features: Flushed face w/ circumoral pallor; inc intensity at flexor folds (Pastia lines are transverse red streaks in skin folds); strawberry tongue; beefy red pharynx & tonsils w/ or w/o exudate
- DX: Rapid strep test (Se 60–90%, Sp 90%), throat cx; CBC rarely indicated but usually leukocytosis w/ PMN predominance present

Treatment
- PCN VK QID × 10 d, benzathine PCN 1.2 million U IM × 1, or macrolide in PCN-allergic

Disposition
- Home

Impetigo

Definition
- Highly contagious superficial infxn 2/2 S. aureus & group A β-hemolytic streptococci; transmitted via direct contact (inc autoinoculation) & fomites; summer common
- Two types: Nonbullous (majority of cases; represents host response to infxn), Bullous (caused by bacterial toxins, esp staph exfoliative toxins)
- Epidemiology: Affects mainly children (2–5 yr; most common pediatric bacterial skin infxn)

History & Physical Exam
- Nonbullous impetigo: Begins as red macule or papule that becomes a vesicle; vesicle ruptures to form an erosion, & its contents dry to form honey-colored crusts; usually on face (cheeks or under lips) or extremities; self-limited over 2 wk
Bullous impetigo: Begins as rapidly enlarging vesicles that form sharply demarcated bullae w/ little to no surrounding erythema; these rupture, forming yellow oozing crusts; usually moist intertriginous areas involved (neck fold, axilla, groin, perineum); self-limited

**Evaluation**
- Dx is clinical; gram stain & culture rarely indicated

**Complications**
- Cellulitis, lymphangitis, poststreptococcal GMN, TSS, SSSS; invasive bact infxns

**Treatment** *(Cochrane 2012;1:CD003261)*
- Most will resolve spontaneously, but abx recommended
- Topical abx equally if not more effective than oral abx (mupirocin 2% ointment TID 3–5 d)
- Oral abx may be indicated in those who cannot tolerate topical tx or w/ extensive dz: Amoxicillin/clavulanate, dicloxacillin, cephalexin, macrolide for PCN-allergic pts

**Disposition**
- Home w/ instruction to prevent spreading

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**FUNGAL EXANTHEMS**

**Dermatophytoses**

**Definitions**
- Superficial fungal infxns involving the stratum corneum, hair, or nails:
  - Tinea capitis: infxn of hair & scalp
  - Tinea corporis: infxn of smooth, hairless skin (except palms, soles, & groin)
  - Tinea cruris: infxn of groin, genitals, pubic area, or perineum
  - Tinea pedis: infxn of feet, commonly interdigital regions
  - Tinea manuum: infxn of hand, commonly interdigital regions
  - Tinea unguium/Onychomycosis: infxn of the nail

<table>
<thead>
<tr>
<th>Clinical Features and Treatment of Dermatophytoses</th>
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<tr>
<td><strong>Dermatophytosis</strong></td>
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<tr>
<td>Tinea capitis</td>
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</table>
- poor hygiene, overcrowding, ↓ SES
PE: Scalp w/ hair loss, scaling, pruritus
Dx: Clinical; wood's lamp may reveal green fluorescence
selenium shampoo can reduce transmission
- Systemic tx preferred:
  - Terbinafine
  - Itraconazole

| Tinea corporis | Hx: RFs - occlusive clothing, minor skin trauma, freq skin-to-skin contact | PE: Annular/polycyclic scaly plaque
Dx: Clinical; KOH prep w/ septate & branching hyphae |
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<td></td>
<td>• Localized dz: tx w/ topical (azoles are fungistatic; allylamines &amp; ciclopirox are fungicidal)</td>
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|               | • Extensive dz, immunosupp, hair follicles: systemic tx
  - Terbinafine
  - Fluconazole
  - Itraconazole |

| Tinea cruris  | Hx: RFs include occlusion & humidity | PE: Annular plaque & scaly raised borders; from inguinal folds; pruritic
Dx: Clinical; KOH prep w/ septate & branching hyphae |

| Tinea pedis   | Hx: RFs include communal bathing, locker rooms, pools | PE: Scaling, erythema, & maceration of interdigital spaces; bacterial superinfn causes erosions, pruritus, & malador (“athlete’s foot”) |

| Tinea manuum  | PE: Scaling of palms, interdigital region, & palmar creases |

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<tr>
<th>Tinea unguium/onychomycosis</th>
<th>Hx: RFs include nail trauma (tight shoes), immunosupp, DM, communal bathing</th>
<th>PE: Toenail varies from discoloration &amp; thickening of proximal, distal/subungal, or superficial portions of nail plate</th>
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|                            | • Topical tx only for limited dz (<50% distal nailbed), may have poor cure rates:
  - Ciclopirox (not as monox)
|                            | • Systemic tx preferred:
  - Terbinafine (preferred)
  - Itraconazole |

*Most tx regimens last from 2–6 wk. Onychomycosis can take months to adequately treat, thus pts require f/u w/ their primary provider or dermatologist.

**Disposition**

- Home w/ primary care or dermatology f/u

**Pearl**
Dermatophytid rxn: Delayed-type hypersensitivity rxn to fungal antigens in pts w/ dermatophytosis; pts p/w pruritic papules or vesicles on hands & feet; respond to tx of primary dermatophytosis infxn

Cutaneous Candidiasis/Intertrigo
Definition
- Fungal infxn by Candida species (C. albicans); predilection for colonizing skin folds (intertriginous areas) where the environment is warm & moist

History & Physical Exam
- Risk factors: Obesity, DM, occlusive clothes, immunosupp, poor hygiene
- Exanthem: Moist, red, shiny patch w/ scalloped borders & satellite macules & pustules, often in intertriginous areas (groin, axilla, pannus folds, gluteal fold, web spaces); can be pruritic, burning, or asx

Physical Findings
- Moist, red, shiny macules/patches w/ scalloped borders, adjacent satellite pustules

Treatment
- Keep dry, topical antifungals (Various preparations: Creams, lotions, powders, w/ or w/o mild steroid combinations; poor data to support 1 type over the other)

Disposition
- Home

HIGH-RISK EXANTHEMS

Pemphigus Vulgaris
Definition
- Rare but potentially life-threatening acute progressive autoimmune (2/2 autoantibodies) bullous dz involving skin/mucosa; mortality 5–10% w/ tx (Clin Dermatol 2013;31(4)374–381)
- Idiopathic, but a/w PMH/FH autoimmune dz

History, Physical Exam, & Evaluation
- HX: Subacute-onset (over dys to wks) additive blisters on mucosa &
skin (mucosa may precede skin by wks/mos); pain > pruritus; ask about PMH/FH autoimmune dz
- EXAM: Small or confluent flaccid blisters & erosions on erythematous base (+Nikolsky); diffuse, inc on mucous membranes (oropharynx, conjunctiva, anogenital)
- DX: Histologic dx (c/s derm for bx); labs to r/o other causes or complications

**Treatment**
- Supportive: analgesia, wound care
- Steroids (1 mg/kg/d pred or equiv); c/s derm, ENT, ophthalmology based on lesions

**Duration & Disposition**
- Duration can be lifelong, or can remit (w/ tx) w/ risk of recurrence
- If rapid progression or extensive dz, admit; c/s derm on all cases in ED

**Erythroderma (Generalized Exfoliative Dermatitis)**

**Definition** *(Clin Dermatol 1993;11(1):67–72)*
- Rare but potentially life-threatening acute generalized red rash, affecting >90% TBSA; more common in males; a/w high-output HF
- Idiopathic (25%) or 2/2 meds, malignancy, psoriasis, uncontrolled dermatitis, among others
- Common associated drugs: ACE inh, allopurinol, anticonvulsants, beta-blockers, beta-lactam abx, CCBs, furosemide, minocycline, NSAIDs, sulfonamides, others

**History, Physical Exam, & Evaluation**
- HX: Subacute-onset (over dys to wks) generalized rash w/ scaling, a/w malaise, chills ± fever; pruritic commonly; always ask about meds, recent inflxn sx, PMH/FH inflam dz & malignancy
- EXAM: Generalized erythema (>90% TBSA; spares palms/soles), progresses to scaling & desquamation; often w/ edema; LAN ± e/o high-output HF, hepatomegaly, splenomegaly
- DX: Elevated ESR, hypoalbuminemia, hyperglobinemia (2/2 antibodies), mild anemia; may see e/o heme malignancy necessitating additional w/u; consult dermatology
Treatment

- Tx underling cause or d/c causal drug if known
- Supportive: skin moisture, antihistamines, topical steroids; systemic steroids usually warranted (unless c/f SSSS or underlying h/o psoriasis); watch fluid balance (given risk of both dehydration & HF)

Duration & Disposition

- Resolution depends on cause & ability to control/remove it
- Admit, esp if rapid or unstable; consult dermatology

Acute Generalized Exanthematous Pustulosis (AGEP)

Definition

- Rare but life-threatening immunologically-mediated diffuse acute pustular exanthema, often w/ multiorgan dysfxn in ~17% (esp elderly) (J Am Acad Dermatol 2015;73(5):843–848)
- Common associated drugs: Beta-lactam abx, quinolones, sulfonamides, carbamazepine, terbinafine, diltiazem, hydroxychloroquine, others


- HX: Acute-onset (w/ hrs) diffuse pustular rash; often occurs w/i 48 h of starting drug (or longer if not 2/2 abx); pruritic; always ask about meds, recent infxn sx
  - May also have sx of systemic organ involvement: SOB, abd pain, N/V, skin infxn
- EXAM: Numerous small sterile pustules on erythematous base, a/w fever; trunk & intertriginous areas common (rarely mucous membranes); pruritic
  - Assess for systemic dz: pleural effusions/hypoxia, hepatic dysfxn, rarely systemic superinfxn & DIC
- DX: CBC w/ leukocytosis (± eosinophilia), LFTs, ± CXR; c/s dermatology for bx

Treatment

- D/C causal drug or tx underling infxn
- Supportive: Moist dressings & antiseptic solutions; high-potency topical steroids may help pruritus, but no role for systemic steroids; ± empiric abx if unstable
Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)

**Definition**
- Rare but potentially life-threatening immune-mediated diffuse rash with multiorgan dysfxn
- Common associated drugs: Allopurinol, anticonvulsants, sulfonamides, others

**History, Physical Exam, & Evaluation**
- HX: Prodrome of pruritus & fever, f/b acute-onset (hrs to dys) diffuse rash; occurs within 2–6 wk of starting drug
- EXAM: Diffuse erythematous morbilliform macules/papules (less commonly erythroderma, pustules, targetoid lesions), can coalesce & become edematous; trunk, extremities, face (can involve mucous membranes)
  - Eval for multiorgan dysfxn: liver (>70% of pts; major source of morbidity/mortality), hematologic, lymphatic, renal, pulm (pneumonitis, ARDS), cardiac (myocarditis), gastroenteritis, meningoencephalitis
- DX: CBC w/ diff (WBC can be >50 k/L; Eos >1.5 k/L; +atypical lymphs), Chem 20 (Cr, lytes, LFTs), Troponin, CXR, dermatology c/s for bx

**Treatment**
- D/C causal drug
- Supportive: antihistamines, tx underlying organ dysfxn
- Systemic steroids w/ gradual taper over 3–6 mo

**Duration & Disposition**
- 10% mortality, esp immunosupp
- Resolves months after causal drug d/c-ed
- Admit (may need ICU)

Stevens-Johnson Syndrome (SJS)/Toxic Epidermal Necrolysis (TEN)

**Definition**
- Acute generalized mucocutaneous desquamative eruption with various
associated causes; immune-mediated but precise mechanism unknown

- Mucous membranes a dz hallmark: 80% cases (first sx in 30%); oropharyngeal, ocular (80% cases), anogenital, GI, endotracheal/bronchial; all w/ severe complications

- Differentiation b/w SJS & TEN depends on TBSA desquamated:
  - SJS: <10% TBSA w/ detachable epidermis w/ horizontal shear (“+Nikolsky sign”)
  - SJS/TEN overlap: 10–30% TBSA w/ +Nikolsky
  - TEN: >30% TBSA w/ +Nikolsky
  - Idiopathic (20%), but often a/w meds (most common cause), infxn (*M. pneumoniae*, HSV), less likely food additives, fumigants, malignancy
  - Common associated drugs: Allopurinol, anticonvulsants, beta-lactam abx, nevirapine, piroxicam, sulfonamides, others

**History, Physical Exam, & Evaluation**

- HX: Flu-like prodrome (fever, malaise, HA, sore throat, rhinitis, myalgias) f/b acute-onset (over dys) diffuse rash ± mucosal sx (dysphagia, etc.); pain; always ask about meds (usually w/i 4 wk), recent infxn sx, PMH/FH inflam dz & malignancy

- EXAM: Diffuse erythematous dusky confluent purpuric macules or patches, rapidly evolve to coalesce & blister (+Nikolsky); often starts on trunk & spreads to extremities (inc palms/soles) & face (inc mucous membranes); eventually generalized sloughing

- Extent of TBSA w/ +Nikolsky dictates SJS (<10%) vs SJS/TEN (10–30%) vs TEN (>30%)

- DX: CBC, BMP, Lactate & blood cx (esp if hypotensive), derm c/s for bx

**Treatment**

- Treat underlying cause or D/C causal drug

- Supportive: analgesia, thermoregulation (28–32°; esp important if high TBSA), IVF, airway protection prn, nutritional support (helps healing) wound care (debridement, bacitracin)

- Limited high-quality data supports specific systemic tx modalities:
  - Cyclosporine may improve survival over IVIG (*J Am Acad Dermatol* )
Complications

- Mostly from mucosal ulcerations in trachea & bronchi (resp distress), esophagus (GIB, malnutrition), eyes (uveitis, ulceration, blindness), genitourinary (dysuria, retention)
- Sepsis can occur 2/2 superinfxn from skin breakdown

Duration & Disposition

- Mortality 5–30%; TEN prognosis predictable using SCORTEN (see table)
- All patients get admitted; TEN requires burn unit admission
- ED or early inpt c/s to ophthalmology, urology (early foley), ± GI, pulm

<table>
<thead>
<tr>
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<tbody>
<tr>
<td><strong>Point</strong></td>
</tr>
<tr>
<td>Age &gt;40</td>
</tr>
<tr>
<td>HR &gt; 120 bpm</td>
</tr>
<tr>
<td>Comorbid malignancy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Score</th>
<th>Mortality</th>
<th>Score</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1</td>
<td>3.2%</td>
<td>4</td>
<td>58.3%</td>
</tr>
<tr>
<td>2</td>
<td>12.2%</td>
<td>≥5</td>
<td>90.0%</td>
</tr>
<tr>
<td>3</td>
<td>35.5%</td>
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</tbody>
</table>

OTHER EXANTHEMS

Allergic/Urticarial Reactions

Definition

- Acute (can be chronic or recurrent) histamine-mediated exanthem, often due to IgE, direct mast cell activation, complement, or dysmetabolism of arachidonic acid (eg, NSAIDs)
- Triggers: systemic exposures (foods, meds, insect stings, contact w/ external allergens, parasites), physical triggers (eg, cholinergic, exercise, pressure, aquagenic, cold, etc.)
Common associated drugs: ASA, ACE inh, beta-lactam abx, NSAIDs, sulfonamides, others

**History, Physical Exam, & Evaluation**
- HX: Acute-onset (over mins) diffuse urticaria, ± SOB, N/V, LH; pruritus (no pain); usually occurs within hrs of trigger
- EXAM: Diffuse or localized erythematous wheals, variably sized (mm to cm), round or irregular in shape, can be excoriated 2/2 pruritic nature; occur anywhere on skin
  - Assess for signs of anaphylaxis (wheezing, hypotension)
- DX: Clinical dx; if +myalgias, LFTs to r/o acute hepatitis

**Treatment**
- D/C causal drug or other trigger
- Antihistamines (H1 & H2), steroids for severe cases, epinephrine if anaphylaxis

**Duration & Disposition**
- Resolves hours after trigger removed; no mortality risk (unless anaphylaxis)
- Home; RX EpiPen in case recurrence; F/U with allergy for allergen testing &/or desensitization

**Serum Sickness & Serum Sickness-like Reactions**

**Definition**
- Acute diffuse immune-mediated (type III hypersensitivity) rash, often 2/2 drug exposure
- Common associated drugs: Barbiturates, beta-lactam abx, fluoxetine, sulfonamides, thiazides, vaccines/anti-serum, others

**History, Physical Exam, & Evaluation**
- HX: Fever, severe arthralgias, malaise, & acute-onset (over hrs) diffuse rash; pain > pruritus; usually occurs within 2 wk of starting drug
- EXAM: Diffuse urticarial or serpiginous macules & patches (though other morphologies possible), well-demarcated w/ intense red border & central clearing; trunk, face, extremities (no palms/soles); joint ROM limited 2/2 pain
- DX: Clinical dx, no testing indicated

**Treatment**
- D/C causal drug or other trigger
- Supportive: antihistamines, NSAIDs for pain
Steroids for severe dz

**Duration & Disposition**
- Resolves 2–3 wk after trigger removed; no mortality risk
- Home if pain controlled; F/U with allergy

**Exanthematous (Morbilliform) Eruption**

**Definition**
- Acute diffuse immune-mediated rash (type IV hypersensitivity) 2/2 drug exposure
- Common associated drugs: Allopurinol, anticonvulsants, beta-lactam abx, NSAIDs, sulfonamides, others

**History, Physical Exam, & Evaluation**
- HX: May have low-grade fever, f/b acute-onset (over hrs–dys) diffuse rash; pruritus > pain; usually occurs within 2–6 wk of starting drug
- EXAM: Diffuse erythematous macules or papules (but can be pustular or bullous), becoming confluent; viral (morbilliform) appearance; trunk & extremities (no palms/soles & face)
- DX: Clinical; elevated CRP, CBC may have mild eosinophilia (if markedly elevated eosinophils, consider DRESS), LFTs nl (if elevated, consider DRESS)

**Treatment**
- D/C causal drug (if unknown, d/c all non-necessary drugs); rarely, the causal medication can be continued through the rash if it is essential; discuss with dermatology
- Supportive care: antihistamines
- Steroids for severe dz

**Duration & Disposition**
- Resolves ~2 wk after med d/c-ed; no significant mortality risk
- Home; F/U with dermatology

**Fixed Drug Eruptions**

**Definition**
- Acute but recurrent localized immune-mediated skin eruption 2/2 repeat drug exposure
- Common associated drugs: ASA, NSAIDs, quinine, sedatives, sulfonamides, tetracyclines, others

**History, Physical Exam, & Evaluation**
HX: Rash w/o systemic sx; pruritic; occurs within hrs–dys of starting drug; on repeat exposure, lesions occur in same location as prior (new lesions may be present as well)
EXAM: Solitary or small group of erythematous or hyperpigmented oval macules evolving to plaques (may become brown); pruritus; common sites include lips, extremities, genitals
DX: Clinical

Treatment
D/C causal drug (hyperpigmented area may remain)
Supportive: antihistamines

Duration & Disposition
Resolves days after med d/c-ed; no significant mortality risk
Home; F/U with dermatology

Erythema Multiforme

Definition
Acute (but sometimes recurrent or persistent) diffuse immune-mediated rash which can have mucosal involvement
- *Erythema multiforme major* – mucosal involvement
- *Erythema multiforme minor* – no mucosal involvement
Idiopathic, but can be a/w infxn (90% cases; esp HSV, M. pneumonia, HCV, EBV), meds, malignancy, XRT, inflamm dz (*Int J Dermatol* 2012;51(8):889–902)
Common associated drugs: Anticonvulsants, beta-lactam abx, NSAIDs, phenothiazines, sulfonamides, others

History, Physical Exam, & Evaluation
HX: Prodrome (fever, malaise) present in EM major, f/b acute-onset (over dys) diffuse rash (see exam); always ask about meds, recent infxn sx, PMH/FH inflamm dz & malignancy
EXAM: Diffuse erythematous macules/papules w/ evolving morphology (become targetoid, then polycyclic & annular configuration); trunk, extremities (inc palms/soles), face (mucous memb ∼70%) (*Int J Dermatol* 2012;51(8):889–902)
- Compared to SJS: Less purpuric, less truncal, less painful, less mucosa
DX: Clinical & histopathologic dx (c/s derm for bx); labs inc HSV PCR/IgM (esp if recurrent episode), ± CXR to r/o causes & complications
**Treatment**
- Tx underlying cause or d/c causal med if known
- Supportive: antihistamines, analgesia, ± oral anesthetic solutions/antiseptic rinses
- Topical steroids if mild, systemic steroids if severe dz (esp mucosal);

**Duration & Disposition**
- Resolves w/i weeks; no significant mortality risk, but can progress to SJS/TEN if offending agent not removed
- Home; f/u w/ dermatology & ophthalmology (if ocular involvement)

**Erythema Nodosum**

**Definition**
- Panniculitis (inflam of subcutaneous fat) w/ unknown mechanism; thought 2/2 immune complex deposition in connective tissue *(Clin Dermatol 2007;25(3):288–294)*
- Idiopathic ~30%, infxn ~30% (TB, recent Grp A strep infxn), sarcoidosis ~20%, inflamm dz, malignancy, pregnancy, drugs
- Common associated drugs: Sulfonamides, OCPs/estrogens, others

**History, Physical Exam, & Evaluation**
- HX: Prodrome (fever, fatigue, malaise, polyarthralgia (symmetric, additive, lg joint), HA, GI sx), f/b acute-onset (over dys to wks) generally-localized rash, ± fever, fatigue, malaise, polyarthralgia (symmetric, additive, lg joint), HA, GI sx; painful; always ask about meds, recent infxn sx, PMH/FH inflamm dz & malignancy
- EXAM: Scattered tender erythematous or purple oval nodules (1–6 cm diam), can coalesce; symmetric & usually anterior tibia (also knees, ankles, thighs, forearms)
- DX: Clinical; labs prn for underlying dz (CBC, ESR/CRP, CXR [r/o e/o Tb, sarcoidosis])

**Treatment**
- Supportive: NSAIDs for pain
- Short course oral corticosteroids in severe dz

**Disposition**
- Most cases self-resolve w/i 6 wk, but EN may recur; no significant mortality risk *(Clin Dermatol 2007;25(3):288–294)*
Leukocytoclastic Vasculitis

Definition
- Acute, chronic, or intermittently recurrent immune-mediated (immune complex, ANCA) rash, sometimes with systemic organ involvement
- Idiopathic (~50%), or with recent infection (viral [esp HBV/HCV], bacterial, parasites, fungi), inflammation, meds (see table), illicit drugs, malignancy (*J Am Acad Dermatol* 2003;48(3):311–340)
- Common associated drugs: Allopurinol, abx, anticoagulants (oral), anticonvulsants, NSAIDs, thiazide diuretics, thiouracil, others

History, Physical Exam, & Evaluation
- HX: Acute-onset (over days) rash; ± systemic sx (fever, arthralgias, GI sx [diarrhea, abd pain], hematuria, hemoptysis); pruritic/burning; always ask about meds, recent infection sx, PMH/FH inflammation dz & malignancy
- EXAM: Diffuse or localized tender palpable purpura or purpuric urticaria, can evolve to coalesce to form plaques or bullae; often lower extremities
  - Multiorgan involvement possible: MSK, GI, cardiac, lungs, ocular, kidneys
- DX: Clinical; labs to rule out systemic dz (CBC, ESR/CRP, Chem 20, UA, ± CXR)

Treatment
- Tx definitive cause (if known) or d/c causal drug
- Supportive (elevation of legs, compression stockings), antihistamines, analgesia with NSAIDs
- Colchicine (± dapsone) if no response to NSAIDs (0.6 mg BID); short-course steroids if still refractory (*J Am Acad Dermatol* 2003;48(3):311–340)

Duration & Disposition
- Resolves within 2 wk if 2/2 drug; if 2/2 underlying dz may persist or recur
- Home if no systemic indications for admx; refer to dermatology & rheumatology

Sweet’s Syndrome (Acute Febrile Neutrophilic Dermatosis)

Definition
Idiopathic (~2/3 cases; F>M); malignancy (second most common cause; often undx’ed), inflam dz, infxn (esp URI, GI sx, others), meds, pregnancy *(Dermatol Online J 1999;5(1):8)*

Common associated drugs: Vaccines, G-CSF, TMP-SMX, minocycline, others

**History, Physical Exam, Evaluation**

- **HX:** Acute-onset (over hrs) rash w/ fever (may precede rash), a/w arthralgias, HA; painful
- **EXAM:** Tender, violaceous, well-demarcated papules & plaques (can get central pustules, bullae, or ulcers – esp if paraneoplastic), can evolve to coalesce; common esp on upper body (inc face, mucous memb)
- **DX:** Bx required for dx; elev ESR (>90%), CBC (WBC >8 k in 80%, +bands), anemia, low plts; LFTs, ± CXR; ± imaging to eval for malignancy dx *(Dermatol Online J 1999;5(1):8)*

**Treatment**

- Supportive: Analgesia
- Systemic steroids for acute dz; mx agents used for suppressive tx

**Duration & Disposition**

- Rapid resolution w/ steroids (w/o tx may persist wks to mo); can recur
- Home if pain controlled & stable; fl/u w/ dermatology

**Photosensitive Reaction**

**Definition**

- Any exanthem appearing in photodistribution after exposure to UV light
  - Drug-related causes: exaggerated sunburn, photosensitive drug rxn (phototoxic, photoallergic), pseudoporphyria
  - Common associated drugs: Diuretics, NSAIDs, phenothiazines, quinolones, sulfonamides, sulfonylureas, tetracyclines, others
  - Not drug-related: porphyria cutanea tarda (PCT), inflamm dz (lupus, dermatomyositis)

**History, Physical Exam, & Evaluation**

- **HX:** Acute-onset (over hrs) rash localized to sun-exposed areas; h/o UV exposure (tanning booth, phototherapy, sunlight); always ask about meds (usually w/i 4 wk), recent infxn sx, PMH/FH inflamm dz
- **Phototoxic rxn:** Onset min–hrs after UV
- **Photoallergic rxn:** Onset 24–72 h after UV
- Pseudoporphyria: Onset hrs after UV
  - EXAM: Exanthem only present on sun-exposed area, morphologically diverse:
    - Phototoxic drug rxn: Exaggerated sunburn response (erythema, edema ± blistering)
    - Photoallergic drug rxn: Erythema ± eczematous changes
    - Pseudoporphyria: Erythema, tense bulla (inc hemorrhagic) & erosions (as opposed to PCT, lacks chronic pigment, hair, or sclerodermal chgs)
  - DX: Clinical; further testing prn to r/o PCT or inflmm dz (eg, porphyrins, ANA, etc.)

**Treatment**
- Supportive: Sun protection (clothing, high-SPF sunscreen), cool compresses, wound care
- Topical steroids, systemic steroids for severe dz
- N-acetylcysteine may speed pseudoporphyria resolution *(Br J Dermatol 2000;142(3):580–581)*

**Duration & Disposition:**
- Resolution variable, but drug-induced pseudoporphyria may take months
- Home; F/U w/ dermatology & rheumatology prn to exclude PCT or inflammatory causes

**Dermatitis**
- Class of skin inflammatory-mediated skin dz marked by similar s/sx: erythema, pruritus, scaling, fissures, varying degrees of lichenification & blistering
- Usually chronic/subacute, but pts may come to ED if bad flare (esp if recent long remission)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Presentation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopic dermatitis</td>
<td>Definition: Chronic, relapsing/remitting; mostly children (10–20% of kids; 1–3% of adults), most w/i first 1 yr. RFs: Hx/FH atopy (asthma, allergic rhinitis, food allergies)</td>
<td>Skin hydration: Soaking baths f/b moisturizer w/i 10 min Topical Steroids: Low-potency for maintenance, mid- &amp; high-potency for flares Antihistamines: For pruritus; do not use topical agents 2/2 risk</td>
</tr>
</tbody>
</table>
| **Contact dermatitis (allergic)**  
*(Ann Allergy Asthma Immunol 2006;97:S1–38)* | **Contact dermatitis (irritant)**  
*(Ann Allergy Asthma Immunol 2006;97:S1–38)* | **Nummular dermatitis** | **Seborrheic dermatitis** |
|---|---|---|---|
| **Definition:** Inflammatory rxn 2/2 direct contact w/ exogenous agent & subsequent type IV hypersensitivity rxn; in Allergic CD, antigen reacts w/ proteins in skin to cause inflammation; in irritant CD, antigen chemically abrades or damages skin to cause inflammation  
Triggers: Latex, plant substances, metals (esp nickel), plant resins, soaps, detergents, fragrance, hair products, sunscreen, top meds  
Exam: Erythematous, papulovesicular w/ varying lichenification, fissuring, scaling, excoriation; often localized to exposed area  
Avoid any suspected trigger  
Sx relief: cold compresses, colloidal baths, emollients  
Topical steroids: Start w/ high-potency if localized (not on face, genitals), then transition to mid- or low-potency as sx improve  
Systemic steroids for severe or extensive dz  
Evaluate & tx superinfxn  
Antihistamines can be ineffective  
Refer to dermatology for patch testing | 
| **Triggers:** Temp, humidity, irritants, infxn, foods, allergens, stress  
Exam: Dry pruritic erythematous papulovesicular w/ excoriations & serous exudate ± lichenification; in kids often on face, neck, extensor surf; in adults often flexor folds  
Prevention: Avoid irritants  
F/U w/ derm: May need tx w/ topical calcineurin inhibitors | **Definition:** Morphologically unique type of atopic dermatitis; can occur older in adulthood; M>F  
Risk factors: Dry skin, atopy, skin injury/abrasion, poor vascular flow, Vit A containing meds  
Exam: Round/oval pink/brown pruritic papulovesicular rash w/ serous exudate, evolving to plaque (2–10 cm diam) w/ crust then scale; often on extremities (but can be torso) | **Definition:** Dz of the sebum-rich areas (scalp/face/trunk), possibly 2/2 abnl immune  
Frequent bathing w/ keratolytic shampoos (eg, selenium, zinc-based), reduce oil |
| **Avoid any suspected trigger**  
**Avoid any suspected trigger**  
**Avoid any suspected trigger**  
**See atopic dermatitis** |
Table:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xerotic dermatitis</td>
<td>Definition: Skin dz characterized by changes 2/2 dry skin; common among elderly</td>
<td>Skin hydration: Soaking baths f/b moisturizer w/ 10 min Topical Steroids: Low-potency &amp; short duration</td>
</tr>
<tr>
<td>Pompholyx</td>
<td>Definition: Subtype of eczema with edematous fluid accumulation in areas w/ thick epidermis; affecting palmoplantar skin; acute, recurrent, or chronic Triggers: a/w atopic &amp; contact dermatitis, drug rxns, stress, id rxn in pts w/ tinea pedis Exam: Nonerythematous pruritic vesicles or bulla on palms or soles</td>
<td>Topical steroids: High-potency Systemic steroids if severe F/U w/ derm: May need tx w/ topical calcineurin inhibitors</td>
</tr>
</tbody>
</table>

**Topical Steroid Preparations by Potency (Generic)**

<table>
<thead>
<tr>
<th>High Potency</th>
<th>Upper-Mid Potency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clobetasol propionate C,G,O,So</td>
<td>Fluocinonide C,G,O</td>
</tr>
<tr>
<td>Betamethasone dipropionate C,G,O,So</td>
<td>Betamethasone valerate O</td>
</tr>
<tr>
<td>Betamethasone valerate O</td>
<td>Mometasone furoate C,O</td>
</tr>
</tbody>
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<tr>
<th>Lower-Mid Potency</th>
<th>Low Potency</th>
</tr>
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<tbody>
<tr>
<td>Triamcinolone acetonide C,O,Sp</td>
<td>Fluticasone propionate C,L,O</td>
</tr>
<tr>
<td>Hydrocortisone valerate C,O</td>
<td>Hydrocortisone 1%, 2.5% C,L,O,Sp</td>
</tr>
<tr>
<td>Desonide C,L,O</td>
<td></td>
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</tbody>
</table>

**Special Notes on Administration**

(1) Vehicles: Ointment (O) most soothing for dry skin. Cream (C) most cosmetically acceptable. Lotion (L), gel (G) & solution (So) most ideal for scalp. Spray (Sp) in unique circumstances.
(2) Avoid high potency or prolonged upper-mid potency for pediatric pts (high absorption), skin folds inc genitals (causes striae), and face (causes atrophy/rosacea/ocular complications).
ACID–BASE DISORDERS

Approach to the Patient

Diagnostics

- BMP; consider LFTs, CBC, urine electrolytes, ABG/VBG, & serum osmoles

Note: HCO₃ from ABG is calculated & should be w/i 2 mmol/L of BMP total CO₂

Step-wise Approach

- **Step 1**: Is there an acidemia or alkaelemia?
  
  Acidemia: pH <7.36; Alkalemia: pH >7.44

- **Steps 2 & 3**: Is the primary disturbance metabolic or respiratory? Is there compensation?

| Assessing Primary Metabolic Disturbances and Physiologic Compensation |
|-----------------------------|--------|--------|--------|-----------------------------|
| Primary Disorder           | pH     | pCO₂   | HCO₃   | Compensation Formula        |
| Metabolic acidosis         | Low    | Low    | Low    | Decr pCO₂ = 1.25 × ΔHCO₃    |
| Metabolic alkalosis        | High   | High   | High   | Incr pCO₂ = 0.75 × ΔHCO₃    |
| Acute respiratory acidosis | Low    | High   | High   | Incr HCO₃ = 0.1 × ΔPCO₂      |
| Acute respiratory alkalosis| High   | Low    | Low    | Decr HCO₃ = 0.2 × ΔPCO₂      |
| Chronic respiratory acidosis| Nl or low | High   | High or nl | Incr HCO₃ = 0.4 × ΔPCO₂ |
| Chronic respiratory alkalosis| Nl or high | Low    | Low or nl | Decr HCO₃ = 0.4 × ΔPCO₂ |

- **Step 4a**: Is there an anion gap?

  **Anion gap acidosis**: (Na – (Cl + bicarb)) > 14 (see chart)

Note: Needs to be corrected for albumin; a fall in serum albumin 1 g/dL from the nl value (4.2 g/dL) decreases the anion gap by 2.5 meq/L.
Corrected AG = AG + (2.5 × [4.2 – albumin]).

- **Step 4b:** If an anion gap is present, is there an osmolar gap?

**Osmolar gap:** Measured serum Osm – Calculated Osm >10 mOsm/L, where Calculated Osm = (2 × [Na⁺]) + glucose/18 + BUN/2.8 + Ethanol/4.6

- **Step 4c:** If no anion gap is present, what is UAG?

**Urinary anion gap:** Na + K – Cl

Note: The UAG can help differentiate GI & renal causes of non-AG (hyperchloremic) metabolic acidosis, as base can be lost from the gut or kidney (negative UAG: GI loss [i.e., diarrhea, small bowel fistula, ileostomy]; positive UAG: Renal loss, particularly RTA types I & IV)

- **Step 5:** What is the delta ratio, also known as the “delta/delta”?

\[
\frac{\text{AG} - \text{nl AG}}{\text{nl HCO}_3^- - \text{HCO}_3^-}, \text{ or simply } \frac{(\text{AG} - 12)}{(24 - \text{HCO}_3^-)}
\]

If delta/delta >+6, suggests concomitant metabolic alkalosis, or prior compensated respiratory acidosis
If delta/delta = 0, suggests uncomplicated AG metabolic acidosis
If delta/delta <-6, suggests concomitant hyperchloremic non-AG metabolic acidosis

<table>
<thead>
<tr>
<th>Metabolic Acidosis</th>
<th>Nonanion Gap Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anion Gap Acidosis</td>
<td>Nonanion Gap Acidosis</td>
</tr>
<tr>
<td>“A CAT’S MUDPILE”</td>
<td>“FUSED CARD TIP”</td>
</tr>
<tr>
<td>Alcoholic ketoacidosis</td>
<td>Fanconi syndrome</td>
</tr>
<tr>
<td>Carbon monoxide, cyanide</td>
<td>Ureteroenterostomy</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Small bowel fistula</td>
</tr>
<tr>
<td>Toluene</td>
<td>Excessive Cl⁻ (NaCl, Ammonium Cl⁻)</td>
</tr>
<tr>
<td>Starvation ketoacidosis</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>Methanol, metformin, methemoglobinemia</td>
<td>Carbonic anhydrase inhibitors</td>
</tr>
<tr>
<td>Uremia</td>
<td>Addison’s dz</td>
</tr>
<tr>
<td>DKA</td>
<td>Renal tubular acidosis</td>
</tr>
<tr>
<td>Paraldehyde, phenformin, propylene glycol</td>
<td>Drugs (spironolactone, amiloride, cholestyramine, triamterene)</td>
</tr>
<tr>
<td>Isoniazid, iron</td>
<td>Toluene (chronic, secondary to RTA)</td>
</tr>
<tr>
<td>Lactic Acidosis types A &amp; B</td>
<td>Ileostomy</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>Pancreatic fistula, parenteral nutrition, posthypocapnia</td>
</tr>
</tbody>
</table>

### Osmolar Gap Causes

#### Toxic Alcohols:
- *Methanol*
- *Ethylene glycol*
- Isopropyl alcohol
- *A/w anion gap*

#### Others:
- Acetone
- Mannitol
- Sorbitol
- Glycerol
- Ether trichloroethane

### Low Anion Gap (<6)

- Lab error
- Lithium tox
- Bromide tox
- Hypoalbuminemia
- Paraproteinemias
- Severe hypercalcemia/hypermagnesemia

### Metabolic Alkalosis

#### Pathophysiology
- **Exogenous HCO<sub>3</sub>**
- **NaCl responsive conditions** *(urine Cl <1<0–1<5 mEq/L)*
- **NaCl unresponsive conditions** *(urine Cl >1<5 mEq/L)*

#### Differential
- Acute alkali administration: Citrate loads from blood transfusions, acetate loads from TPN, administration of NaHCO<sub>3</sub> solution, excessive antacids
- Milk–alkali syndrome
- GI loss of H⁺: V/D, NGT drainage, adenomas
- Renal loss of H⁺: Diuretic use
- Posthypercapnia
- Volume expansion/hypertensive/mineralocorticoid excess: Hyperaldosteronism, Cushing syndrome, exogenous mineralocorticoid, licorice, renal artery stenosis
- Volume contraction/normotensive/2° hyperaldosteronism: Hypokalemia, hypomagnesemia, hypercalcemia/hypoPTH, Bartter's syndrome, Gitelman's syndrome
- Volume expansion/hypertensive/hypoaldosteronism: Liddle's syndrome

### Respiratory Acidosis

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central respiratory depression</td>
<td>Drugs (opioids, sedatives), brainstem infarct, high C-spine injury, obesity hypoventilation syndrome (ie, Pickwickian syndrome)</td>
</tr>
<tr>
<td>Nerve or muscular disorders</td>
<td>Paralysis, muscular dystrophy or other myopathies, myasthenia gravis, toxins (ie, organophosphate, snake envenomations), Guillain–Barré, ALS</td>
</tr>
<tr>
<td>Airway issues</td>
<td>Upper airway obstruction, laryngospasm, bronchospasm</td>
</tr>
<tr>
<td>Respiratory issues</td>
<td>Asthma, COPD, CHF, pneumonia, ILD, aspiration, ARDS, inadequate mechanical ventilation</td>
</tr>
<tr>
<td>Chest wall trauma</td>
<td>Flail chest, PTX, hemithorax, diaphragmatic paralysis, kyphoscoliosis</td>
</tr>
</tbody>
</table>

### Respiratory Alkalosis

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac, respiratory</td>
<td>Pulmonary edema, pulmonary embolus, restrictive lung dz, mechanical hyperventilation</td>
</tr>
<tr>
<td>Psychiatric, neurologic</td>
<td>Hyperventilation syndromes (eg, anxiety, pain, stress), meningoencephalitis, tumor, trauma, CVA</td>
</tr>
<tr>
<td>Infection</td>
<td>Fever, pneumonia, sepsis</td>
</tr>
<tr>
<td>GI</td>
<td>Liver failure</td>
</tr>
<tr>
<td>Meds, other</td>
<td>Salicylates, hyperthyroidism, high altitude, anemia, pregnancy</td>
</tr>
</tbody>
</table>

### Treatment and Disposition
- Both will largely depend on severity & underlying etiology of the disorder
- Limited role for bicarbonate in the absence of hemodynamic collapse
Hyponatremia

Definition
- Na <135, excess of water relative to sodium, usually from elevated ADH; generally not symptomatic at Na >125

History
- Most sx are nonspecific: Fatigue, weakness, muscle cramps, thirst, or postural dizziness. Severe sx include confusion, agitation, delirium, lethargy, somnolence, coma, or szs.
- Other helpful historical features include h/o CHF, cirrhosis, renal dz, cancer, adrenal or pituitary dysfxn, recent GI surgery, thiazide or loop diuretics use, alcoholism

Physical Exam
- Look for signs to assess pt fluid status:
  - Hypervolemia: Elevated JVP, peripheral edema, crackles, ascites, anasarca
  - Hypovolemia: Tachycardia, hypotension, dry mucous membranes, oliguria, poor skin turgor, IVC collapsibility
- Look for signs of profound hyponatremia: Lethargic, disoriented/abnl sensorium, depressed reflexes, hypothermic, pseudobulbar palsy, Cheyne–Stokes respiration

Diagnostics
- Labs: BMP, FSG, urine electrolytes (Na, Cr, Osm), serum Osm, albumin
- VBG w/ stat sodium & Osm may provide more rapid turnaround
- Corrected Na$_{glucose}$ = Serum Na + [0.016 × (serum glucose – 100)]
  - up to 400 mg/dL
  - for glucose >400 mg/dL, 4 mEq/L should be added to every additional 100 mg/dL

Step-wise Approach to Hyponatremia
- **Step 1:** What is the serum osmolality?

<table>
<thead>
<tr>
<th>Causes of Hyponatremia by Serum Osmolality</th>
<th>Hypertonic HypoNa</th>
<th>Isotonic HypoNa</th>
<th>Hypotonic HypoNa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hyperglycemia</strong></td>
<td></td>
<td>Lab/blood draw error</td>
<td>Etiology based on volume status. *See Step 2</td>
</tr>
<tr>
<td>Manntitol</td>
<td></td>
<td>Hyperparaproteinemia</td>
<td></td>
</tr>
<tr>
<td>Glycerol</td>
<td></td>
<td>HL</td>
<td></td>
</tr>
<tr>
<td>Sorbitol</td>
<td></td>
<td>Post TURP (bladder irrigation</td>
<td></td>
</tr>
</tbody>
</table>
NI serum osmolality = 275–290 mosmol/kg

- **Step 2:** What is the pt’s volume status? Hypervolemic, euvoletic, or hypovolemic?
- **Step 3:** What are the urine Na, urine Osm, & FeNA values?
  - **Fractional Excretion of Sodium = FeNa = (Na_{urine} \times \text{Cr}_{serum})/(Na_{serum} \times \text{Cr}_{urine})**

### Assessing Causes of Hypotonic Hyponatremia by Volume Status and Urine Analysis

<table>
<thead>
<tr>
<th>Volume Status</th>
<th>Urine Na</th>
<th>Urine Osm</th>
<th>FeNa</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypervolemic</td>
<td>&gt;20</td>
<td>&gt;1%</td>
<td></td>
<td>Renal failure</td>
</tr>
<tr>
<td></td>
<td>&lt;10</td>
<td>&lt;1%</td>
<td></td>
<td>CHF, cirrhosis, nephrosis</td>
</tr>
<tr>
<td>Euvolemic</td>
<td>&gt;100</td>
<td></td>
<td></td>
<td>SIADH, *hypothyroidism, glucocorticoid deficiency</td>
</tr>
<tr>
<td></td>
<td>&lt;100</td>
<td></td>
<td></td>
<td>Psychogenic polydipsia (&gt;12 L fluid/d), low solute (beer potomania, tea/toast diet, dilution of infant formula)</td>
</tr>
<tr>
<td>Variable</td>
<td></td>
<td></td>
<td></td>
<td>Chronic malnutrition (anorexia), pregnancy</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>&gt;20</td>
<td>&gt;1%</td>
<td></td>
<td>Renal losses: Diuretic use, osmotic diuresis, salt-wasting nephropathy, mineralocorticoid deficiency, nonoliguric ATN</td>
</tr>
<tr>
<td></td>
<td>&lt;10</td>
<td>&lt;1%</td>
<td></td>
<td>Extrarenal losses: Vomiting, diarrhea, NGT drainage, 3rd spacing (pancreatitis, SBO), sweating</td>
</tr>
</tbody>
</table>

*Pneumonia, asthma, COPD, SCLC, pneumothorax, trauma, CVA, hemorrhage, tumors, infection, hydrocephalus, antipsychotics/antidepressants, chemo, vasopressin, postoperative.

### Treatment
- Asymptomatic or mild sxs of hyponatremia: Correct serum Na at ≤0.5 mEq/L/h
- Severe manifestations of hyponatremia: RAPID correction serum Na at 2 mEq/L/h × 2–3 h OR until sxs resolve

### IV Fluid Management

Total Body Water (TBW) = Weight (kg) × 0.6 (use 0.5 if female or elderly, 0.6 for infants)
\[
\text{Rate of Infusion (cc/h)} = \frac{1000 \times [\text{TBW} \times (\text{desired Na} - \text{serum Na})]}{[\text{Na}(\text{mmol/L})_{\text{infuse}} \times \text{time(h)}]}
\]

Infusate concentrations:
LR: 130 mmol/L  NS: 154 mmol/L  3% NS: 513

Requires checking serum Na *(& Glu) q1h

- Euvolemic hyponatremia
  - Asymptomatic: Free water restrict (500–1000 mL/day)
  - Symptomatic: See above
- SIADH
  - Free water restrict + treat underlying cause
  - Caution if using hypertonic or nl saline esp if IVF Osm < urine Osm, serum sodium may worsen (higher Osm will draw out fluid)
  - Consider lithium or demeclocycline – nephrogenic DI (*NEJM 2007;356:2064*)
- Hypovolemic hyponatremia
  - Volume replete w/ nl saline, as above (once dehydration resolved, stimulation of ADH will decline & Na will correct)
- Hypervolemic hyponatremia
  - Free water restrict (0.5–1.5 L/d)
  - Increase arterial volume: W/ vasodilators (Nitro), loop diuretics; consider albumin in cirrhosis
  - Severe hyponatremia: Consider diuresis + Na replacement

Disposition
- Home: Mild asymptomatic hyponatremia
- Admit: Symptomatic, comorbidities, elderly. May require ICU admission if severe.

Pearl
- Rapid correction >10–12 mEq/L/d may result in central pontine myelinolysis (dysarthria, szs, quadriplegia due to focal myelin destruction in pons & extrapontine areas)

Hypernatremia

Definition
- Na >145, usually from free water loss or sodium gain (eg, infusion of hypertonic fluid)
- Appropriate response to hypernatremia is increased free water intake
stimulated by thirst & renal excretion of a minimal volume of maximally concentrated urine as regulated by ADH

History
- Mild sx include increased thirst or polyuria
- Severe sx: AMS (irritability, lethargy, confusion, delirium, coma)
- RFs: Elderly, infants, debilitated. Endocrine pathology; cardiac, renal, liver dz; psychiatric disorder (see etiology of Central and Nephrogenic Diabetes Insipidus); MEDS (see below chart), living situation (access to free water).

Physical Exam
- Look for signs to assess pt fluid status:
  - Hypervolemia: Elevated JVP, peripheral edema, crackles, ascites, anasarca
  - Hypovolemia: Tachycardia, hypotension, dry mucous membranes, oliguria, poor skin turgor, IVC collapsibility
- Severe hypernatremia: Lethargy, muscle spasticity, tremor, hyperreflexia, respiratory paralysis, ataxia

Diagnostics
- Labs: BMP, FSG, urine electrolytes (Na, Cr, Osm), serum Osm, albumin
- VBG w/ stat sodium & Osm may provide more rapid turnaround
- Corrected Na\textsubscript{glucose} = Serum Na + [0.016 \times (serum glucose – 100)]
  up to 400 mg/dL
- For glucose >400 mg/dL, 4 mEq/L should be added to every additional 100 mg/dL

Step-wise Approach to Hypernatremia
- Step 1: What is the serum osmolality?
  - NI serum osmolality = 275–290 mosmol/kg
- Step 2: What is the pt's volume status? Hypervolemic, euvoletic, or hypovolemic?
- Step 3: What are the urine Na & urine Osm values?

<table>
<thead>
<tr>
<th>Assessing Causes of Hypernatremia by Volume Status and Urine Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>---------------------------------------------------------------</td>
</tr>
<tr>
<td>Hypervolemic</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
## mineralocorticoid excess

<table>
<thead>
<tr>
<th>Euvolemic</th>
<th>&lt;300</th>
<th>Complete DI (central &amp; nephrogenic)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>300–600</td>
<td>Partial DI (central &amp; nephrogenic)*</td>
</tr>
<tr>
<td></td>
<td>&gt;600</td>
<td><strong>Exogenous Sodium Intake:</strong> Hypertonic saline, sodium bicarb tablets, sea water ingestion, concentrated infant formula</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypovolemic</th>
<th>&gt;20</th>
<th>300–600</th>
<th><strong>Renal Water Losses:</strong> Loop diuretic, osmotic diuresis (mannitol, urea)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;20</td>
<td>&gt;600</td>
<td><strong>Extrarenal Water Losses:</strong> Vomiting, diarrhea, NGT drainage, szs, exercise, severe burns, fever, 3rd spacing <strong>Decreased Water Intake:</strong> Defective thirst mechanism, dementia, AMS, infancy, intubation</td>
</tr>
</tbody>
</table>


## Treatment

### IV Fluid Management*

| Free water deficit (Liters) = Total Body Water × [1 – (140/serum Na)] |
|--------------------------|-------------------------------------------------------------------|
| Total body water (TBW) = Weight (kg) × 0.6 (use 0.5 if female or elderly; 0.6 for children) |
| Hourly maintenance (mL/h) = Free Water Deficit (mL)/24 h |
| Rate of Infusion (cc/h) = \[ \frac{1000 \times [TBW \times (serum \ Na - desired \ Na)]}{[Na]_{infusate} \times time \ (h)} \] |

<table>
<thead>
<tr>
<th>Infusate concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td>D5W: 0 mEq</td>
</tr>
</tbody>
</table>

*Requires checking serum Glucose & Na q1h. Rate of Na correction should NOT exceed 0.5 mEq/L/h to avoid cerebral edema. Urine output: >0.5 cc/kg/h.

- Hypovolemic hypernatremia
  - Treat underlying disorder
  - Replace free water deficit (as above)
Euvolemic hypernatremia
- Replace free water deficit (as above)
- Treat underlying etiology
- Central DI: Vasopressin 10 U SQ

Hypovolemic hypernatremia
- Restore volume 1st then replace free water deficit (as above); add 40 mEq KCl IV to fluid replacement once pt is urinating

Disposition
- Home: Mild hypernatremia which can be corrected in <24 h
- Admit: Likely admit

Hypokalemia

Definition
- K⁺ <3.5 mEq/L (ie, decreased intake, shift into cells, loss); 98% of potassium is intracellular

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Poor oral intake; diarrhea, vomiting, &amp; NG tube drainage</td>
</tr>
<tr>
<td>Endocrine</td>
<td>High insulin levels, hyperaldosteronism, alkalosis, DKA, Cushing dz, hypomagnesemia</td>
</tr>
<tr>
<td>Renal</td>
<td>Renal tubular acidosis (type 2), renovascular dz, Bartter syndrome, Liddle syndrome</td>
</tr>
<tr>
<td>Meds/toxins</td>
<td>Thiazide &amp; loop diuretics, insulin, β-2 agonists, α-antagonists, amphotericin B, laxative abuse, exogenous mineralocorticoid use, massive blood transfusions, barium tox, toluene tox</td>
</tr>
</tbody>
</table>

History
- Usually not symptomatic until K⁺ <3 mEq/L
- Nausea, vomiting, weakness, fatigue, myalgia, muscle cramps.
- Pts at highest risk for electrocardiac cx of hypokalemia include those w/ acute ischemia, prolonged QT syndrome, & those taking digoxin

Physical Exam
- Paresthesias, depressed reflexes, proximal muscle weakness, ileus
- Severe hypokalemia: Hypoventilation, spasm, paralysis, rhabdomyolysis, myoglobinuria
- ARF, polymorphic VT, asystole
Diagnostics

- **Labs:** BMP, UA, urine electrolytes, urine Osm; consider blood gas, CPK, serum Osm
- Urine K+ <15 mmol/d suggests extrarenal, while urine K+ >15 mmol/d suggests renal etiology
- Transtubular K+ concentration gradient (TTKG) is helpful, but rarely used in the ED:
  \[ \text{TTKG} = \frac{(\text{Plasma Osm} \times \text{Urine K}^-)}{(\text{Plasma K}^- \times \text{Urine Osm})} \]

Note: Hypokalemia w/ TTKG >4 suggests renal K+ loss due to distal K+ secretion

- **ECG:** T-wave flattening/inversion, ST depression, U-waves, prolonged QT/QU interval; may also see PR prolongation, decreased voltage, QRS widening, atrial/ventricular dysrhythmias

Treatment

- **ED**
  - Potassium replacement: Potassium chloride, Potassium bicarbonate, Potassium phosphate
    
    (Drop 1 mEq/L = 200–400 mEq total body loss)
    
    Mild (K+ >2.8 mEq/L): 40 mEq K+ PO q4–6h
    
    Moderate/severe: 40 mEq K+ PO q4h (if tolerating oral) + KCl 10 mEq/h IV, recheck K+ q4h

  - Treat underlying cause
  - Replace Mg as needed (*Note: Concurrent Mg & K+ deficiency could lead to refractory K+ repletion)
  - Goal K+ = 4 mEq/L in pts at highest risk

- **Home**
  - Counsel pts to increase dietary intake of K+ (dried fruits, nuts, avocados, wheat germ lima beans, vegetables [spinach, broccoli, cauliflower, beets, carrots], fruits [banana, kiwi, etc])
  - Discuss w/ PCP: Decrease diuretic dose; start/substitute for K+-sparing med (βB, ACE, ARB, K+-sparing diuretic)
  - Potassium replacement: KCl 20 mEq PO QD for prevention; KCl 40–100 mEq PO QD for tx

Disposition

- **Home:** Mild hypokalemia w/ close f/u to recheck labs
- Admit: Moderate/severe hypokalemia, acid–base abnormalities, arrhythmia, severe sx
Pearl

- Avoid dextrose solutions (stimulate insulin & inward shift of $K^+$)


Hyperkalemia

Definition

- $K^+>5$ mEq/L (ie, $K^+$ release from cells, decreased renal losses, iatrogenic)

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine/metabolic</td>
<td>Hypoaldosteronism, DKA, other acidoses</td>
</tr>
<tr>
<td>Renal</td>
<td>Renal insufficiency, end-stage renal failure, renal tubular acidosis (type 4), diabetic nephropathy, Gordon’s syndrome</td>
</tr>
<tr>
<td>Other</td>
<td>Tumor lysis syndrome, hemolysis, rhabdomyolysis, *pseudohyperkalemia (hemolyzed blood sample, prolonged tourniquet), exercise</td>
</tr>
<tr>
<td>Meds</td>
<td>NSAIDs, ACE−, ARBs, heparin, TMP–SMX, pentamidine βBs, digoxin poisoning, $K^+$ sparing diuretics, exogenous KCl supplements, cyclosporine, *succinylcholine</td>
</tr>
</tbody>
</table>

*Pseudohyperkalemia should be suspected in o/w asymptomatic pts w/o underlying causes. Repeated $K^+$ should be obtained prior to initiating tx in such cases.

History

- Weakness, muscle cramps, paresthesias, nausea, palpitations. Meds (see Differential table).

Physical Exam

- Paresthesias, tetany; assess fluid status
- Severe hyperkalemia: Flaccid paralysis, hypoventilation, PEA arrest, or asystole

Diagnostics

- Labs: BMP; consider blood gas w/ stat $K^+$, UA, urine electrolytes, urine Osm, CPK
- ECG: Early: Peaked & symmetric T waves, flattened P waves, PR prolongation, 1° AVB. Late: Widening/slurring of QRS → sinusoidal waveform → VFib or asystole
<table>
<thead>
<tr>
<th>Intervention</th>
<th>Dose</th>
<th>Onset</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium gluconate OR Calcium chloride***</td>
<td>1–2 amps IV</td>
<td>Few minutes</td>
<td>Stabilizes cell membrane; used in pts w/ cardiac conduction abnormalities (no direct effect on K⁺)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>1–2 amps</td>
<td>15–30 min (up to 2 h)</td>
<td>Transient K⁺ into cells in exchange for H⁺ (may ↓ K⁺ 0.47 mmol/L)</td>
</tr>
<tr>
<td>Albuterol (β-agonist)</td>
<td>10–20 mg inh or 0.5–2.5 mg IV</td>
<td>30–90 min</td>
<td>Transient K⁺ into cells (↓ K⁺ 0.3–0.99 mmol/L)</td>
</tr>
<tr>
<td>Insulin + D₅₀W</td>
<td>10 U IV + 1 amp D₅₀W</td>
<td>15–30 min, lasts 2–4 h</td>
<td>Transient K⁺ into cells (↓ K⁺ 0.45–1 mmol/L)</td>
</tr>
<tr>
<td>Kayexalate***</td>
<td>30–90 g PO/PR</td>
<td>90 min for PO, 30 min for PR</td>
<td>Decreases total body K⁺ by exchanging Na for K⁺ in gut</td>
</tr>
<tr>
<td>Diuretics (Furosemide)</td>
<td>≥40 mg IV</td>
<td>30 min</td>
<td>Decreases total body K⁺</td>
</tr>
<tr>
<td>HD (emergent)</td>
<td></td>
<td></td>
<td>Decreases total body K⁺ (pts w/ cardiac cx or new/worsened renal failure)</td>
</tr>
</tbody>
</table>

*Standard teaching is not to use calcium in digitalis tox → hypercalcemia may potentiate the tox; however, recent data shows that this may be inaccurate.

**Calcium chloride contains 3 times more calcium ion, onset in seconds to minutes & lasts 30 min, but much more caustic to veins than Calcium gluconate.

***May cause intestinal necrosis in pts w/ postoperative ileus; may also worsen pulmonary edema in pts w/ fluid overload; data on its efficacy at reducing total body potassium is poor.

**Treatment**

- Continuous cardiac monitoring
- Treating underlying cause
- Check electrolytes every 2–4 h until normalized

**Disposition**

- Home: Only if mild, stable hyperkalemia with good outpatient f/u
- Admit: Most pts will require admission; may require ICU admission

**Pearls**

- Think “ABCD” (albuterol, bicarbonate, calcium, dextrose/insulin, dialysis, diuretics)
Combination therapy is proven more efficacious than any therapy alone
HD is the most rapid & effective way of lowering plasma K⁺

**Hypocalcemia**

**Definition**
- Ca <8.5 mg/dL (2 mmol/L) OR ionized Ca <4.5 mg/dL (1.1 mmol/L);
- 50% bound to albumin, 40% is free, 10% complexed to anions

<table>
<thead>
<tr>
<th>Hypocalcemia Etiology</th>
<th>Pathophysiology</th>
<th>PTH</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine</td>
<td></td>
<td>↓</td>
<td>Hypoparathyroidism [familial, autoimmune, infiltrative, iatrogenic: Surgery, neck irradiation], DiGeorge syndrome, hypomagnesemia</td>
</tr>
<tr>
<td>Vit D deficiency</td>
<td></td>
<td>↑</td>
<td>Nutritional/sunlight deprivation; malabsorption; drugs (anticonvulsants, rifampin, ketoconazole, 5-FU/leucovorin); genetic; renal insufficiency (impaired production)</td>
</tr>
<tr>
<td>Renal</td>
<td></td>
<td>↑</td>
<td>Chronic renal failure, ARF (elevated phosphorous)</td>
</tr>
<tr>
<td>Neoplasm</td>
<td></td>
<td>↑</td>
<td>Osteoblastic metastases, tumor lysis (elevated phosphorous)</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>↑</td>
<td>Pancreatitis, multiple blood transfusions, rhabdomyolysis, burns, prematurity, pseudohypoparathyroidism</td>
</tr>
</tbody>
</table>

**History**
- Weakness, muscle cramps, paresthesias, irritability, depression, tetany, AMS. Meds (see **Differential table**).

**Physical Exam**
- Paresthesias; Chvostek sign (tap over facial nerve causing facial twitching); +Trousseau sign (inflate a BP cuff to 20 mmHg above systolic BP over bicep × 3 min to cause carpal spasm); may also see psychosis, szs, ↑ ICP, bronchospasm, laryngospasm

**Diagnostics**
- Labs: BMP w/ Ca/Mg/Phosphorus testing. Check ionized calcium level, albumin, consider PTH for continued inpt w/u:

\[
\text{Corrected Ca} = \text{measured serum calcium (mg/dL)} + [0.8 \times (4-\text{serum albumin (g/dL)})]
\]
- ECG: *Prolonged QTc*, heart blocks, ventricular dysrhythmias, torsade

**Treatment**
- Asymptomatic: Oral elemental Ca (1–3 g/d in divided doses)
- Symptomatic: [10% Calcium gluconate (1–2 g IV over 20 min) OR 10% Calcium chloride (1–2 g IV diluted in 100 cc D$_5$W to decrease tissue irritation)], ± Vit D, ± Mg (50–100 mEq/d)

**Disposition**
- Home: Asymptomatic, w/ oral regimen described above & PCP f/u in 5–7 d to recheck electrolytes
- Admit: Severe hypocalcemia, comorbid conditions, HD unstable

**Hypercalcemia**

**Definition**
- Ca >10.5 mg/dL; usually asymptomatic at levels up to 11.5 mg/dL

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>PTH</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excess PTH prod</td>
<td>↑</td>
<td>1° hyperparathyroidism* (adenoma, hyperplasia, rarely adenoCa), 3° hyperparathyroidism (renal insufficiency), FHH</td>
</tr>
<tr>
<td>Vit D excess</td>
<td>↓</td>
<td>Sarcoidosis, TB, histoplasmosis, Wegener granulomatosis, Vit D intoxication, lymphoma</td>
</tr>
<tr>
<td>↑ bone resorption</td>
<td>↓</td>
<td>Hyperthyroidism, immobilization</td>
</tr>
<tr>
<td>Neoplasm*</td>
<td>↓</td>
<td>PTHrP-producing solid tumors (squamous cell, renal bladder), lytic lesions (breast, myeloma), Paget dz</td>
</tr>
<tr>
<td>Other</td>
<td>↓</td>
<td>Meds (lithium, Vit A, thiazides, Ca-based antacids), massive dairy, consumption (milk–alkali syndrome), TPN, endocrine d/o (adrenal insufficiency, VIPoma)</td>
</tr>
</tbody>
</table>

* Most common causes of hypercalcemia.

**History**
- Polyuria, polydipsia, dehydration, nausea, vomiting, depression, confusion, coma, AMS; abdominal pain, anorexia, constipation, bone pain, Meds (see Differential table)
- May cause pancreatitis, nephrolithiasis, pathologic fractures thus suspect hypercalcemia in pts presenting w/ sx consistent w/ these diagnoses
Physical Exam
- General weakness, epigastric tenderness, depressed deep tendon reflexes, coma

Diagnostics
- Labs: BMP w/ Ca/Mg/Phosphorus testing, ionized Ca, lipase (if considering pancreatitis), urine electrolytes, albumin (see corrected Ca equation above), consider PTH
- ECG: Shortened QTc, PR prolongation, QRS widening; rarely BBB, sinus bradycardia or high-degree AV block

Treatment
- Address/treat underlying causes

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Dose</th>
<th>Onset/Duration</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>NS</td>
<td>4–6 L/d</td>
<td>Hours</td>
<td>Promote calcium excretion (Ca can drop 2 mEq)</td>
</tr>
<tr>
<td>Furosemide</td>
<td>20–60 mg IV q6h</td>
<td>Hours</td>
<td>Promotes calcium excretion; hold if intravascularly dry</td>
</tr>
<tr>
<td>Bisphosphonates (pamidronate, zoledronic acid, alendronate)</td>
<td>Variable</td>
<td>Days</td>
<td>Inhibit osteoclasts (esp useful in malignancy), caution in renal failure pt</td>
</tr>
<tr>
<td>Hypercalcemic antidote (calcitonin, plicamycin)</td>
<td>Calcitonin: 4 IU/kg q12h Plicamycin: 25 mcg/kg IV over 4 h</td>
<td>Hours, lasts days</td>
<td>Direct RNA inhibitor, may develop tachyphylaxis</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>200–300 mg IV QD</td>
<td>Days</td>
<td>Useful only for Vit D toxic pts, multiple myeloma, sarcoid &amp; lymphoma pts</td>
</tr>
<tr>
<td>HD</td>
<td></td>
<td></td>
<td>Useful in renal failure pts</td>
</tr>
</tbody>
</table>

Disposition
- Home: Mild stable hypercalcemia
- Admit: Most will need admission until resolution

Pearl
- Hypercalcemia = stones, bones, moans, abdominal groans, & psychiatric overtones
Hypomagnesemia

**Definition**
- Mg <0.7 mmol/L

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>CHF</td>
</tr>
<tr>
<td>GI</td>
<td>V/D, NGT suctioning, malabsorption</td>
</tr>
<tr>
<td>Renal</td>
<td>Chronic renal failure (causing tertiary hypoparathyroidism)</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Hyperaldosteronism, Vit D deficiency</td>
</tr>
<tr>
<td>Other/Meds</td>
<td>Alcoholism, pregnancy, thiazide &amp; loop diuretics, aminoglycosides, amphotericin, gentamicin, pentamidine, tobramycin</td>
</tr>
</tbody>
</table>

**History**
- Weakness, AMS, muscle cramps. Meds (see *Differential table*).

**Physical Exam**
- Tetany, Chvostek/Trousseau signs, papilledema, hyperreflexia

**Diagnostics**
- Labs: BMP w/ Ca/Mg/Phosphorus testing, ionized Ca, albumin, consider PTH for continued inpt w/u.
- ECG: Similar to hypokalemia & hypocalcemia (prolonged intervals, T-wave flattening, widening of QRS, U waves)

**Treatment**
- Address underlying cause
- Magnesium replacement: 50% magnesium sulfate 2–4 g (16.6–33 mEq) IV over 30 min. Oral form may cause diarrhea (eg, magnesium citrate, milk of magnesia).
- Alcoholics: Consider thiamine; phosphorous & potassium replacement as needed

**Disposition**
- Home: Mild hypomagnesemia
- Admit: Severe hypomagnesemia w/ other associated electrolyte abnormalities (potassium, calcium), comorbid conditions

**Pearl**
- Most exogenously administered Mg will be excreted in urine; full Mg
replacement takes days

**Hypermagnesemia**

**Definition**
- Mg >3 mEq/L

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Chronic constipation, bowel obstruction</td>
</tr>
<tr>
<td>Renal</td>
<td>Acute or chronic renal failure</td>
</tr>
<tr>
<td>Autoimmune/endocrine</td>
<td>DKA, adrenal insufficiency, hyperparathyroidism, hypothyroidism</td>
</tr>
<tr>
<td>Other/Meds</td>
<td>Hemolysis, lithium, exogenous Mg infusions, opioids, anticholinergics, tumor lysis syndrome, milk-alkali syndrome, rhabdomyolysis</td>
</tr>
</tbody>
</table>

**History**
- Nausea, vomiting, lethargy, weakness, AMS; depends on level (renal insufficiency, GI motility disorder, adrenal insufficiency, hyperparathyroidism), Meds (anticholinergic, narcotic, lithium)

**Physical Exam**
- Depends on level
  - Mg >3 mEq/L: N/V cutaneous flushing
  - Mg >4 mEq/L: Hyporeflexia
  - Mg >5 mEq/L: Hypotension
  - Mg >9 mEq/L: Respiratory depression, shock, coma
  - Mg >10 mEq/L: Asystole

**Diagnostics**
- Labs: BMP w/ Ca/Mg/Phosphorus testing, ionized Ca, albumin
- ECG: QRS widening, QT prolongation, prolonged AV conduction → complete block

**Treatment**
- Calcium:
  - Immediate: Calcium gluconate IV or Calcium chloride (see hypocalcemia)
  - Continuous: 10% Calcium gluconate 2–4 mg/kg/h if indicated
- Diuretics: Loop diuretics + aggressive hydration (improve excretion)
- Dialysis: Particularly for pts in renal failure
Disposition
- Home: Asymptomatic, stable
- Admit: All need admission until sx & lab values have normalized

Pearls
- Magnesium abnormalities are often seen w/ K⁺ or calcium abnormalities
- Check serial DTRs to assess toxicity in preeclamptic pts receiving Mg

Hypoglycemia

Definition
- Glucose <60 mg/dL; however, clinical hypoglycemia is any plasma glucose level low enough to cause sx or signs c/w hypoglycemia (see below). Usually <55 mg/dL causes sx.
- Whipple’s triad: Sign/sx of hypoglycemia, low plasma glucose, resolution of sx when plasma glucose is raised

<table>
<thead>
<tr>
<th>Hypoglycemia Differential</th>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Medications</strong></td>
<td></td>
<td>Insulin, sulfonylureas (glyburide, glipizide, glimepiride), Meglitinides (repaglinide, nateglinide), alcohol</td>
</tr>
<tr>
<td>GI</td>
<td></td>
<td>Liver failure, post-gastrectomy/gastric bypass</td>
</tr>
<tr>
<td>Renal</td>
<td></td>
<td>ARF</td>
</tr>
<tr>
<td>Endocrine</td>
<td></td>
<td>Hypothyroidism, insulinoma (including MEN-1), hypopituitarism, adrenal insufficiency, insulin autoimmune hypoglycemia (Ab to insulin or its receptor)</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>Sepsis, starvation, accidental/surreptitious/malicious hypoglycemia</td>
</tr>
</tbody>
</table>

*Most common cause of hypoglycemia.

History
- Neurogenic/autonomic sx: Agitation, tremor, diaphoresis, palpitations, pallor, hunger
- Neuroglycopenic sx: Fatigue, HA, AMS, lethargy, somnolence, coma, sz
- Take detailed med hx (see Differential table): consider new meds, med dose changes, incorrect use, intentional/accidental overdose, OTC/naturopathic meds, AKI
Diabetics:
- Inquire recent FSG values (if taken), last meal, dietary changes, excess exercise
- ROS of contributing causes: Fever, chills, cough, abdominal pain, diarrhea, urinary sx, etc.
- RFs: Diabetics (esp on insulin), alcoholics, infants, elderly, s/p gastric bypass, critically ill

**Diagnostics**
- Labs: FSG, BMP; consider infectious w/u (CBC, UA, CXR)
  *In o/w healthy, nondiabetics, consider LFTs, TSH, insulin, β-hydroxybutyrate, proinsulin, & C-peptide (low in exogenous insulin, high in insulinoma or sulfonylureas) in consultation w/ an endocrine specialist

- Serial glucose assessments may be necessary when prolonged hypoglycemia is expected in pts unable to communicate (eg, dementia, delirium, comatose, infants)

**Treatment**
- Glucose replacement:
  - PO: Glucose paste/tablets (20 g), fruit juice, soft drinks, candy, a meal, etc.
  - IV: 1 amp D₅₀; infusion may be needed
  - IM: 0.5–1 mg IM or SC glucagon (may cause N/V)

**Disposition**
- Home: Identifiable cause, does not need further monitoring
  - Prompt f/u w/ primary care or endocrinologist should be arranged
  - Pts should keep a glucose diary & should become concerned about the possibility of developing hypoglycemia when self-monitored glucose levels fall rapidly or is no greater than 70 mg/dL
- Admit: Long-acting hypoglycemic agents, unable to tolerate POs, HD unstable

**Pearls**
- βBs can mask adrenergic signs of hypoglycemia
- Efforts should be made to contact pt’s primary physician or endocrinologist

### HYPERGLYCEMIC EMERGENCIES (DKA/HHS)

<table>
<thead>
<tr>
<th>Criteria for Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycemic State</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DKA (glucose &gt;250 mg/dL)</strong></td>
</tr>
<tr>
<td><strong>Mild</strong></td>
</tr>
<tr>
<td>Arterial pH</td>
</tr>
<tr>
<td>Serum bicarbonate (mEq/L)</td>
</tr>
<tr>
<td>Urine ketone</td>
</tr>
<tr>
<td>Serum ketone</td>
</tr>
<tr>
<td>Serum osmolality</td>
</tr>
</tbody>
</table>

#### Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State

**Definition**

- See above for consensus diagnostic criteria. DKA characterized by uncontrolled hyperglycemia, metabolic acidosis, & increased ketone body concentration. HHS characterized by profound hyperglycemia & serum hyperosmolality, nl arterial pH & bicarbonate, & AMS.
- Marked by insulin deficiency & increased counter-regulatory hormones
- HHS generally occurs in Type II diabetes; DKA generally occurs in Type I diabetes, but may occur in Type II diabetes w/ stressors:

### 5 I's of DKA

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin deficiency</td>
<td>New-onset T1DM, failure to take enough insulin</td>
</tr>
</tbody>
</table>
Infection* Pneumonia, UTI, cellulitis, etc.
Inflammation Pancreatitis
Intoxication Alcohol, drugs
Iatrogenesis Glucocorticoids, thiazides, sympathomimetics, antipsychotics
Other AMI, CVA, eating d/o in pts w/ T1DM

*Most common precipitating factor.

History
- DKA often more acute in onset, c/w HHS which evolves over days to weeks
- Polyuria, polydipsia, N/V, dehydration, weight loss, abdominal pain, visual changes, AMS
- Take detailed med hx (see Differential table); consider new meds, med dose changes, incorrect use, intentional/accidental overdose, OTC/naturopathic meds, insulin pump use
- ROS of contrib causes: Fever, chills, cough, abdominal pain, diarrhea, urinary sx, depression
- RFs: Insulin pump users

Physical Exam
- Appears dry, Kussmaul respiration, lethargy, coma; abdominal tenderness (ileus)

Evaluation
- Labs: FSG, BMP (elevated anion gap acidosis, pseudohyponatremia, total body K+ generally depleted despite lab value), Ca/Mg/Phosphorus, urine/serum ketones, β-hydroxybutyrate, nitroprusside test, UA, CBC, lactate, lipase, LFTs, serum osmolality, VBG, urine hCG; ABG if HD unstable or comatose; blood cultures, urines cultures if clinically indicated

<table>
<thead>
<tr>
<th>Equations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anion gap (AG) = (Na – (Cl + bicarb))</td>
</tr>
<tr>
<td>Corrected AG = AG + (2.5 × [4.2 – albumin])</td>
</tr>
<tr>
<td>Calculated Osm = (2 × [Na+]) + glucose/18 + BUN/2.8 + Ethanol/4.6</td>
</tr>
<tr>
<td>Corrected Na = Serum Na + [0.016 × (serum glucose – 100)]</td>
</tr>
</tbody>
</table>

(<up to 400 mg/dL; for glucose >400 mg/dL, 4 mEq/L should be added to every additional 100)
- ECG: If older than 30 yr
- Imaging: CXR (r/o infection); may need abdominal CT or U/S if clinically indicated

**Treatment**
- Supportive: Continuous cardiac monitoring, 2 large-bore IVs
- Electrolyte monitoring: Glucose fingerstick q1h; BMP, Ca/Mg/Phosphorus, VBG q2–4h

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose/Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IV hydration</strong>*</td>
<td>NS bolus + NS 15–20 cc/kg/h (adjust for dehydration &amp; cardiovascular status); usually 1–1.5 L during 1st hour</td>
</tr>
<tr>
<td></td>
<td>→ Continue NS 250–500 cc/h if corrected Na low</td>
</tr>
<tr>
<td></td>
<td>→ Δ IVF to ½ NS 250–500 cc/h if corrected Na nl or high</td>
</tr>
<tr>
<td></td>
<td>→ Δ IVF to D5 ½ NS 150–250 cc/h when glucose ≤200 mg/dL</td>
</tr>
<tr>
<td>Insulin</td>
<td>0.1 U/kg (regular insulin) IV push × 1, followed by 0.1 U/kg/h</td>
</tr>
<tr>
<td></td>
<td>Persistent anion gap: Continue drip</td>
</tr>
<tr>
<td></td>
<td>Resolution of anion gap: Change to SC insulin (overlap IV w/ SC by 1–2 h)</td>
</tr>
<tr>
<td></td>
<td>→ When glucose ≤200 mg/dL in DKA &amp; ≤300 mg/dL in HHS, reduce insulin infusion to 0.02–0.05 U/kg/h IV, or Δ to rapid acting insulin at 0.1 U/kg q2h</td>
</tr>
<tr>
<td>Electrolyte repletion</td>
<td>Potassium: Goal to maintain K⁺ 4–5 mEq/L</td>
</tr>
<tr>
<td></td>
<td>→ Add 20–40 mEq/L IVFs if serum K⁺ &lt;4.5 (insulin promotes K⁺ entry into cells, but careful w/ renal pts)</td>
</tr>
<tr>
<td></td>
<td>→ Hold insulin &amp; give K⁺ 20–40 mEq/h if K⁺ &lt;3.3</td>
</tr>
<tr>
<td></td>
<td>HCO₃⁻: If cardiac unstable or pH &lt;7</td>
</tr>
<tr>
<td></td>
<td>Phosphate: Replete if &lt;1 (20–30 mEq/L KPhos added to IVF)</td>
</tr>
</tbody>
</table>

*After volume resuscitation, choice of fluid replacement will depend on hemodynamics, hydration status electrolytes, etc.

**IVF volume should be used w/ caution in pts w/ cardiac or renal impairment.


**Disposition**
- Home: None
Admit: All pts will require admission, may need ICU monitoring

Pearls
- ~10% of the DKA population may present w/ glucose ≤250 mg/dL
- An initial insulin bolus may not be necessary as some pts respond to fluid resuscitation
- Consider increasing continuous insulin dose if glucose does not decrease 50–75 mg/dL/h
- Tx w/ SC rapid-acting insulin q1–2h is an effective alternative to IV regular insulin
- Cx: Hypoglycemia, hypokalemia, fluid overload, cerebral edema

THYROID EMERGENCIES

Hypothyroidism/Myxedema Coma

Definition
- Hypothyroidism is characterized by insufficient production of thyroid hormone by the thyroid gland. Cretinism is a form of hypothyroidism found in infants.
- Hypothyroidism can be classified on the basis of its time of onset (congenital or acquired), the level of endocrine Dysfxn (1° [thyroid] or 2° [pituitary or hypothalamic]), & its severity (subclinical, clinical, severe [myxedema coma])
- Myxedema coma is a rare, extreme expression of severe hypothyroidism. Myxedema coma typically occurs in pts who develop systemic illness superimposed on previously undiagnosed hypothyroidism.

<table>
<thead>
<tr>
<th>Hypothyroidism Differential</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine</td>
<td>Hashimoto (autoimmune thyroiditis), subacute thyroiditis (de Quervain’s thyroiditis), lymphocytic thyroiditis (postpartum thyroiditis), hypothalamic or pituitary failure, iodine deficiency</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Surgical removal &amp; XRT</td>
</tr>
<tr>
<td>Meds/toxins</td>
<td>Radioactive iodine (therapeutic or environmental), amiodarone, lithium, stavudine, interferon α, polybrominated/polychlorinated biphenyls, resorcinol (textile workers)</td>
</tr>
</tbody>
</table>
### Other

| Congenital hypothyroidism (endemic iodine deficiency, thyroid gland dysgenesis, defective thyroid hormone biosynthesis); hemochromatosis |

Adapted from: Roberts, CG, Ladenson PW. Hypothyroidism. 

<table>
<thead>
<tr>
<th>Factors Precipitating Myxedema Coma</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infection</strong> <em>(sepsis, PNA, UTI)</em></td>
</tr>
<tr>
<td><strong>CVA</strong></td>
</tr>
<tr>
<td><strong>CHF</strong></td>
</tr>
<tr>
<td><strong>Hypothermia</strong></td>
</tr>
<tr>
<td><strong>GIB</strong></td>
</tr>
<tr>
<td><strong>Trauma, burns</strong></td>
</tr>
<tr>
<td><strong>Metabolic disturbances</strong> <em>(hypoglycemia, hyponatremia, acidosis, hypercapnia, hypercalcemia)</em></td>
</tr>
<tr>
<td><strong>Meds</strong> <em>(anesthetics, sedatives, opioids, amiodarone, lithium, withdrawal of L-thyroxine)</em></td>
</tr>
<tr>
<td><strong>Ingestion of raw bok choy</strong></td>
</tr>
</tbody>
</table>

Adapted from: Klubo-Gwiezdzinska J, Wartofsky L. Thyroid emergencies. 

### History

- **Hypothyroidism:** Weakness, fatigue, myalgias, HA, depression, cold intolerance, weight gain, constipation, menorrhagia, dry skin, brittle hair, hoarseness
- **Myxedema coma:** Severely altered mental status/coma
- **Meds** *(see Differential table)*
- **RFs:** Postpartum women, family h/o autoimmune thyroid disorders, prior H&N surgery or irradiation, other autoimmune disorders *(ie, Type 1 DM, adrenal insufficiency, autoimmune polyendocrine syndrome types 1 & 2 etc.), Down’s syndrome, Turner’s syndrome

### Physical Exam

- **Hypothyroidism:** Obese, delayed DTRs, diastolic HTN, dry, thick skin SQ tissue *(myxedema)*, bradycardia, pl/pericardial/peritoneal effusion, hypothermia, hypotension, hypoventilation, altered sensorium
- **Myxedema coma:** Hypothermia & severely altered mental status/coma are hallmark
- **Vitals/Pulm/CV:** Hypothermia, hypoventilation, hypoxia, hypotension, or bradycardia
- **HEENT:** Facial swelling, periorbital edema, macroglossia
- **Neuro:** Lethargy → comatose, cerebellar signs, poor memory & cognition, delayed reflexes
- **Psych:** “Myxedema madness” disorientation, paranoia, depression, hallucinations, etc.

### Evaluation
Labs: TFTs (TSH elevated); BMP (hyponatremia, hypoglycemia), CBC (anemia); consider T4, free T4, T3, antimicrosomal Ab, antithyroid peroxidase Ab, antithyroglobulin Ab
- ↑ TSH, ↓ free T4 confirms primary hypothyroidism of any cause
- ↑ TSH, ↓ free T4, +antithyroid abs confirms Hashimoto thyroiditis
- variable TSH, ↓ free T4 consistent w/ secondary hypothyroidsm disorders
- mild ↑ TSH, nl free T4, & subtle sxs consistent w/ subclinical hypothyroid

ECG: Myxedema-bradycardia, AV block, low voltage, flattened/inverted T-waves, prolonged QTc, atrial/ventricular dysrhythmias.

Bedside cardiac u/s: Pericardial effusion/tamponade may be seen in myxedema

Treatment (Only Start Empiric Treatment if Severely Symptomatic/Coma)
- Thyroid replacement: (Start in ED if severely symptomatic/coma)
  - Levothyroxine: 5–8 mcg/kg IV × 1, then 50–100 mcg QD; consider synthetic T3 5–10 mcg IV q8h (b/c peripheral conversion impaired, but is more arrhythmogenic)
  - Adrenal replacement: Hydrocortisone 100 mg IV × q8h (decreased reserve in coma)

Disposition
- Home: Discuss w/ PCP prior to starting any thyroid medications; usual starting dose of Levothyroxine 1.8 μg/kg PO QD (required repeat TFTs at 4–6 wk)
- Admit: All pts w/ severe hypothyroidism/myxedema; may require ICU admission

Thyrotoxicosis/Hyperthyroidism/Thyroid Storm

Definition
- Thyrotoxicosis is a disorder of excess thyroid hormone
- Hyperthyroidism specifically describes overproduction & secretion of excess of free thyroid hormones: Thyroxine (T₄), triiodothyronine (T₃), or both
- Thyroid storm/crisis is a rare, extreme expression of severe thyrotoxicosis
- Precise criteria for thyroid storm have been defined (Endocrinol Metab Clin North Am 1993;22:
**Differential**

**Thyrotoxicosis w/ Hyperthyroidism**

<table>
<thead>
<tr>
<th>Endocrine</th>
<th>*Graves’ dz, **toxic multinodular goiter, **solitary toxic adenoma, TSH-secreting pituitary adenoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasm</td>
<td>Metastatic follicular thyroid carcinoma, struma ovarii, choriocarcinoma (hCG secretion)</td>
</tr>
<tr>
<td>Other/Meds</td>
<td>Amiodarone, iodine, &amp; radiographic contrast agents</td>
</tr>
</tbody>
</table>

**Thyrotoxicosis w/o Hyperthyroidism**

<table>
<thead>
<tr>
<th>Thyroiditis</th>
<th>Early Hashimoto (autoimmune thyroiditis), subacute thyroiditis (de Quervain’s thyroiditis), lymphocytic thyroiditis (postpartum thyroiditis), acute infectious thyroiditis, drug-induced thyroiditis (amiodarone, lithium, interferon α), radiation thyroiditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other</td>
<td>Exogenous thyroid hormone, “Hamburger” thyrotoxicosis, infarction of thyroid adenoma</td>
</tr>
</tbody>
</table>

*Most common cause of hyperthyroidism caused by autoantibodies to & stimulation of TSH receptors.

**Next most common causes of hyperthyroidism caused by autonomous overproduction of thyroid hormone secondary to activating mutations in TSHR or a focus of functional autonomy, respectively.


---

**Factors Precipitating Thyroid Storm**

- Infection (sepsis)
- Sz
- PE
- Parturition
- Emotional stress
- Trauma (including vigorous thyroid palpation), burns
- Postthyroidectomy
- Metabolic disturbances (hypoglycemia, DKA)
- Meds (amiodarone, radioactive iodine tx, iodinated contrast, thyroxine/triiodothyronine OD, ASA OD, withdrawal of PTU/methimazole)


---

**History**

- Neck fullness, double vision, restlessness, anxiety, palpitations, sweating, heat intolerance, tremor, weight loss, diarrhea, irregular menses, periodic paralysis, lethargy, hair thinning/loss
- Thyroid storm: AMS (delirium, agitation, coma), sz, fever, tachycardia, N/V, diarrhea
Meds (see Differential table; assess h/o hyperthyroidism)

**Physical Exam**
- Thyrotoxicosis: Cachexia, diaphoretic, agitation, tremor, tachycardia, AFib, systolic HTN, widened pulse pressure
- Thyroid storm: Hyperthermia & severely AMS are hallmark
- Vitals/Pulm/CV: Hyperthermia, hyperventilation, tachycardia
- GI: Nausea, vomiting, diarrhea, diffuse abdominal pain (may mimic acute abdomen)
- Neuro: AMS (delirium, agitation, coma), sz
- Psych: Disorientation, paranoia, psychosis, etc.

**Evaluation**
- Labs: TSH (low) w/ elevated free T4 (if TSH low & free T4 nl, free or total T3 concentration should also be measured to identify potential T3 toxicosis; consider thyroxine-binding globulin in pregnancy); BMP/Ca/Mg/Phosphorus, LFTs, UA, urine hCG; consider TRH or thyroid peroxidase
- ECG: Tachycardia, supraventricular ectopy, AFib

**Treatment (Only Start ED Treatment if Severely Symptomatic/Thyroid Storm)**
- Thyrotoxicosis: Therapies include antithyroid meds (methimazole/PTU), radioiodine, surgery
- Thyroid storm: βB → PTU or methimazole → iodine or lithium → steroids w/ supportive care
  - βB: Propranolol or esmolol (improve α-adrenergic activity & tachycardia)
    - Propranolol 1 mg IV over 10 min, then 1–3 mg boluses q3h
    - Propanolol 60–80 mg q4h if taking PO
    - Esmolol 250–500 mcg/kg loading dose, then 50–100 mcg/kg/min
  - PTU: Blocks hormone synthesis, inhibits peripheral conversion of T4 to T3
    - Loading dose of 500–1000 mg, then 250 mg q4h
    - Preferred to methimazole, particularly if pregnant & 1st trimester
  - Methimazole: Blocks hormone synthesis
    - Dose 20 mg q4h (60–80 mg/d)
    - Pts should receive baseline CBC & LFTs prior to tx
  - Iodine: Blocks thyroid hormone release but give >1 g after PTU (can
potentiate thyroid storm if given before)
- Potassium iodide 5 drops (0.25 mL or 250 mg) PO q6h
- For iodine allergic pts, can use lithium carbonate 300 mg 6 h
- Steroids: Hydrocortisone 100–300 mg IV bolus, then 100 mg IV × q8h (can decrease conversion of T4–T3)
- Consider plasmapheresis & therapeutic plasma exchange (Graves’ dz)
  ‣ Supportive care: Hyperpyrexia – APAP as needed; avoid aspirin (can increase T3 conversion)
  ‣ Treat underlying precipitant (often infection)

Disposition
  ‣ Home: TSH low but no severe sx: F/u w/ PCP or endo RE: outpt meds ± surgery.
  ‣ Admit: All pts w/ severe hyperthyroidism. Pts w/ thyroid storm require ICU admission.


## ADRENAL INSUFFICIENCY

**Definition**
- Condition in which the adrenal glands, do not produce adequate amounts of steroid hormones, primarily cortisol, but may also include impaired aldosterone production
- Primary adrenal insufficiency (Addison’s dz) refers to pathology of the adrenal cortex, where secondary adrenal insufficiency may occur as a result of pituitary or hypothalamic dzs.

<table>
<thead>
<tr>
<th>Adrenal Insufficiency Differential</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infiltrative dz</td>
<td>Tuberculosis, CMV, histoplasmosis/cryptococcosis/blastomycosis, amyloidosis, sarcoidosis, histiocytosis AIDS (opportunistic dz)</td>
</tr>
<tr>
<td>Vascular*</td>
<td>Hemorrhage, thrombosis, necrosis (meningococcemia, sepsis, **APLAS)</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Autoimmune adrenalitis (alone or as component of autoimmune polyglandular syndromes types 1 &amp; 2), pituitary failure</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>Metastatic dz (lung, breast, kidney), lymphoma, pituitary tumor (primary or mets), craniopharyngioma, hypothalamic tumors</td>
</tr>
<tr>
<td>----------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Meds</td>
<td>Ketoconazole, etomidate, rifampin, anticonvulsants, megestrol, glucocorticoid withdrawal</td>
</tr>
<tr>
<td>Other*</td>
<td>Trauma (esp head trauma, burns) postpartum pituitary necrosis (Sheehan's syndrome), empty sella syndrome, pituitary radiation/surgery</td>
</tr>
</tbody>
</table>

*Causes of acute onset adrenal insufficiency.
**APLAS: Antiphospholipid antibody syndrome.

- Dysfxn of the hypothalamic–pituitary–adrenal axis in critical illness is termed critical illness-related corticosteroid insufficiency (CIRCI)

**History**
- Weakness, fatigue, anorexia, nausea, vomiting, presyncope, craving for salt
- Meds (see Differential table); also elicit if pt is on chronic steroids at baseline

**Physical Exam**
- Orthostatic hypotension, hyperpigmentation, vitiligo

**Evaluation**
- Labs: BMP (may see hypoglycemia, hyponatremia, hyperkalemia, acidosis), CBC (may see mild normocytic anemia, lymphocytosis, & eosinophilia); send serum cortisol/ACTH level for inpt w/u
  - Serum cortisol >25 μg/dL in a pt requiring intensive care likely rules out adrenal insufficiency
  - CIRCI is best diagnosed by a delta cortisol (after 250 μg cosyntropin) of <9 μg/dL or a random total cortisol <10 μg/dL
  - ACTH stimulation test is rarely used in the ED
- Imaging: Consider head MR (assess pituitary), adrenal CT

**Treatment (Only Start ED Treatment if Symptomatic/Hypotensive)**
- Steroids: Hydrocortisone 100 mg IV bolus, followed by continuous infusion at 10 mg/h; may also give 200 mg/d in 4 divided doses
- IV hydration: Volume resuscitation w/ nl saline
- Steroids (particularly, hydrocortisone) should be considered in the
management strategy of pts w/ septic shock, particularly those pts who have responded poorly to fluid resuscitation & vasopressors (SBP <90, despite IVF & vasopressors)

Disposition
- Home: Stable, already on meds
- Admit: All pts w/ new onset adrenal insufficiency; may require ICU admission if concomitant infection or HD unstable

Pearls
- Acute adrenal insufficiency should be suspected in the presence of fluid & pressor-refractory hypotension, esp in a pt w/ signs & sx as noted above
- Pts w/ known adrenal insufficiency & concomitant febrile illness should be instructed to increase their home dose of steroid by 2–3 times until recovery to prevent possible adrenal crisis. Stress dose steroids can be given in the ED prior to disposition.

DEHYDRATION

Approach
- Careful hx: Understand whether pt has had excessive fluid loss or inadequate intake
- Attempt to quantify fluid deficit
- Check FSG to r/o hypoglycemia, electrolytes

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Arrhythmia (1f)</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Adrenal insufficiency, DI, DKA, SIADH, thyroid Dysfxn</td>
</tr>
<tr>
<td>Infectious</td>
<td>Encephalitis (4b), meningitis (4b), Lyme dz (4h), sepsis (16b), syphilis (4)</td>
</tr>
<tr>
<td>GI</td>
<td>Bowel obstruction, diarrhea, gastroenteritis, intestinal volvulus, vomiting, GIB</td>
</tr>
<tr>
<td>FEN/GU</td>
<td>Electrolyte disturbances, renal insufficiency</td>
</tr>
<tr>
<td>Neurologic</td>
<td>GBS, myasthenia gravis, ALS, stroke, migraine</td>
</tr>
<tr>
<td>Hematologic/oncologic</td>
<td>Metastatic dz</td>
</tr>
<tr>
<td>Toxic</td>
<td>Drug induced</td>
</tr>
<tr>
<td>Environmental</td>
<td>Hyperthermia</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>Anorexia, bulimia, laxative abuse, psychosis</td>
</tr>
</tbody>
</table>

History
- Excessive fluid loss (V/D, sweating, polyuria, diuretic/laxatives, bowel regimen), inadequate intake (debilitated, institutionalized, NM d/o, H&N pathology), altered thirst mechanism (intoxication, systemic illness, malignancy, antipsychotic use)

Findings
- ↑ HR w/ standing (Δ >20 bt/min lying → standing) 75% sens & spec; skin tenting, dry MM
Evaluation
- CBC (hemoconcentration), BMP (↓ bicarb, ↑ BUN/Cr, abnl Na, K), ECG abnl
- UA: Ketones, hyaline casts, spec grav >1.02: Uroconcentration, >1.03 = Severe dehydration

Treatment
- Initial fluid resuscitation w/ NS or LR (avoid NS if concern for hyponatremia), then tailor to electrolyte abnlty/pathology (labor: Nonglucose IVF, malnourishment: D5 NS)
- nl LV fxn: 2–3 L NS, follow clinical sxes, VS, UOP
- Compromised LV fxn: 500 cc/h, watch pulmonary status (O₂ sat, SOB)
- Consider antiemetic if N/V contributes to dehydration

Disposition
- Home once dehydration adequately treated unless concerning electrolyte abnormalities, pt able to maintain hydration status
- Consider care coordination/placement if pt lives alone & unable to hydrate self

Pearls
- Up to 30% of healthy pts are orthostatic w/o dehydration (βBs, autonomic Dysfxn (DM))
- Oral rehydration w/ glucose to facilitate intestinal absorption of Na & water if pt tolerates, “recipe” is 2 tbl sugar: 0.5 tsp salt: 1 quart water; ½ dilute apple juice also effective
- Healthy adults tolerating PO rarely require IVF & PO rehydration is usually adequate

<table>
<thead>
<tr>
<th>Types of Dehydration</th>
<th>Losses</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotonic</td>
<td>Na loss &gt; water loss</td>
<td>Diuretics</td>
</tr>
<tr>
<td>Isotonic</td>
<td>Na loss = water loss</td>
<td>Vomiting, diarrhea</td>
</tr>
<tr>
<td>Hypertonic</td>
<td>Na loss &lt; water loss</td>
<td>Fever, sweating, faulty thirst mechanism</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Degrees of Dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degree</td>
</tr>
<tr>
<td>--------</td>
</tr>
</tbody>
</table>
### Bites and Stings

**Approach**
- Treat anaphylaxis; give tetanus prophylaxis
- Consider x-ray for underlying fx or FB
- Assess for joint space violation, copious wound irrigation/wash out w/ NS; if heavily contaminated, do not close
- 24–48 h wound check for high-risk bites, esp in kids or unreliable pts
- National Poison Control Center (PCC): (800) 222-1222

### Human & Animal Bites

**Human**

**History**
- Laceration near MCP joint during altercation should be considered a human bite (“fight bite”); bacteria spread along tendon sheath deep into hand

**Evaluation**
Consider x-ray to assess for fracture, air in joint, tooth fragments; no serology needed
Extend & explore periarticular MCP joint injuries, including in that position that injury occurred

Treatment
- Preferred regimen: Amoxicillin/Clav acid) 875/125 mg BID × 5–10 d
- Alternatives: Doxycycline or TMP-SMX or pen VK or fluoroquinolone or cefuroxime PLUS clindamycin or metronidazole
- If late/complicated/needs admit, IV ampicillin/sulbactam 1.5 g q6h
- Delayed 1° closure if closure needed

Disposition
- Scheduled strict f/u in 24–48 h

Pearl
- *Eikenella* (most common), *Staph/Strep* species found in mouth, anaerobes

Cat
Evaluation
- Consider x-ray to assess for fracture, air in joint, tooth fragments
- Extend & explore joint injuries including in the position that injury occurred

Treatment
- Amoxicillin/Clav Acid 875/125 mg BID, cefuroxime 500 mg BID or doxycycline 100 mg BID
- Delayed 1° closure only if cosmetically needed; 80% of cat bites become infected!

Disposition
- Scheduled strict f/u in 24 h

Pearls
- *Pasteurella multocida* most common organism
- Consider cat-scratch dz if pt has tender LAD 1 wk after bite/scratch
- Very high infection rate despite abx use
- Consider rabies prophylaxis (rabies immunoglobulin + vaccine) if unknown cat (4i)

Dog
Evaluation
Consider x-ray to assess for fracture, air in joint, tooth fragments

**Treatment**
- Amoxicillin/Clav acid 875/125 mg BID or clindamycin 300 QID + ciprofloxacin 500 mg BID
- 1° closure after copious irrigation possible except on hand/foot; only 5% become infected

**Disposition**
- Scheduled strict f/u in 24 h

**Pearls**
- Polymicrobial infections
- Consider rabies prophylaxis if unknown dog as above w/ cats (4i)

---

**Snake Bites**

**Crotalinae/Pit Vipers (Rattlesnakes, Copperheads, Water Moccasins)**

**History**
- Pain & swelling around fang marks, attempt identification of snake if possible

**Findings**
- Local (pain, swelling, ecchymosis), systemic (↓ BP, ↑ HR, paresthesias), coagulopathy (↓ PLTs, ↑ INR, ↓ fibrinogen), pulmonary edema, acidosis, rhabdomyolysis, neuromuscular weakness if Mojave rattlesnake

**Evaluation**
- Consult PCC/toxicologist; CBC, BMP, coags w/ fibrinogen & split products, CK, T&C, x-rays to r/o retained fang; watch compartment pressures

**Treatment**
- Remove rings, constrictive clothing, general wound care, tetanus
- Antivenom (Crotalidae) if systemic effects or coagulopathy; surgical assessment if compartment syndrome; supportive care; no proven benefit w/ abx or steroids

**Disposition**
- D/C if absence of any findings 8–12 h post bite envenomation in healthy adults, 12–24 h in children/elderly, 12–24 h if concerns for
Mojave rattlesnake
› ICU admission if antivenom given

**Pearls**
› Avoid oral or mechanical suction of wound, tourniquets, incision, & suction
› 25% of bites are “dry strikes” (no effect); pit vipers identified by 2 fangs

<table>
<thead>
<tr>
<th>Grades of Pit Viper Envenomation (Dynamic)</th>
<th>Signs/Sxs</th>
<th>Vials of Antivenom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Local pain, edema. No signs of systemic tox. nl labs.</td>
<td>None</td>
</tr>
<tr>
<td>Moderate</td>
<td>Severe local pain, edema &lt;50 cm around wound. Systemic tox: N/V. Labs abnl (↓ Hct, ↓ PLTs).</td>
<td>4–6</td>
</tr>
<tr>
<td>Severe</td>
<td>Generalized petechiae/ecchymosis, compartment sx, bleeding, ↓ BP, AMS, renal dysfxn, markedly abnl coags</td>
<td>Initial dose 8–12</td>
</tr>
</tbody>
</table>

**Elapidae/Coral Snake** (*Micrurus fulvius*)

**History**
› Bitten by brightly colored snake (black, red, & yellow bands), primarily in tx, FLA

**Findings**
› Neurotoxic effects from venom: Tremor/sz, ↑ salivation, respiratory paralysis, bulbar palsy (dysarthria, diplopia, dysphagia), usually less local tissue damage than Crotalinae

**Evaluation**
› Consult PCC/toxicologist; CBC, BMP, coags/DIC eval not usually indicated, consider pulmonary fxn testing

**Treatment**
› Consult PCC before giving antivenom as higher risk for allergic rxn; surgical assessment if concern for compartment syndrome; supportive care (esp respiratory support)

**Disposition**
› 12–24 h observation; ICU admission if antivenom given or respiratory compromise

**Pearl**
› True coral snakes have red on yellow banding, nonvenomous snakes
have red band on black background: “Red on yellow: Kill a fellow. Red on black: Poison lack.”

**SCORPION BITES**

**Scorpion (Centruroides exilicauda)**

**History**
- Burning & stinging w/o visible injury at bite site

**Findings**
- Usually no visible local injury; possible systemic effects include roving eye movements (pathognomonic), opisthotonos, ↑ HR, diaphoresis, fasciculations
- Mydriasis, nystagmus, hypersalivation, dysphagia, restlessness
- Severe envenomation may cause pancreatitis, respiratory failure, coagulopathy, anaphylaxis

**Evaluation**
- “Tap test”: Exquisite tenderness w/ light tapping in exilicauda stings; consult PCC/toxicologist
- CBC, BMP, coags, LFTs, CK, ECG

**Treatment**
- Most bites are self-limited, provide supportive care
- BZD for muscle spasm/fasciculations, pain control, tetanus, reassurance
- If severe systemic sxrs, 1–2 vials scorpion antivenom; avail from AZ PCC

**Disposition**
- Admission for observation; ICU admission if antivenom given

**Pearl**
- Only *C. exilicauda* (bark scorpion) found in Western US produces systemic toxicity

**SPIDER BITES**

**Brown Recluse (Loxosceles reclusa)**

**History**
- Pt may not remember bite & initially have no pain; pain & pruritus
develops over 2–8 h
- Severe rxn: Immediate pain & blister formation, necrosis & eschar over next 3–4 d
- Loxoscelism: Systemic rxn 1–3 d after envenomation; N/V, f/c, muscle/joint aches, sz, rarely renal failure, DIC, hemolytic anemia, rhabdomyolysis

Findings
- Necrotic blister w/ surrounding erythema, petechiae

Evaluation
- Consult PCC/toxicologist, surgery/plastics consult for lesion >2 cm
- CBC, BMP, coagulation profile, UA

Treatment
- No antivenom; wound care, tetanus, supportive care (eg, hydration, abx, transfusion, HD), local debridement
- May consider dapsone 50–100 mg BID to prevent necrosis, hyperbaric O₂, steroids (all are controversial)
- Dapsone causes hemolysis, hepatitis; monitor LFTs, check G6PD level

Disposition
- Admission for observation

Pearl
- Located in S. Central & SW (desert) of US; violin-shaped marking on back

Black Widow (*Latrodectus mactans*)

History
- Immediate pain, then swelling, possible target-shaped lesion, can have unexplained severe abd/back pain, muscle cramps w/i 1 h
- Pain may continue intermittently for 3 days, is often a/w muscle weakness & spasm for wk to mo

Findings
- Severe rxns: HTN, respiratory failure, abd rigidity, fasciculations, shock, coma

Evaluation
- CBC, BMP, CK, coagulation profile, UA, abd CT (r/o acute abdomen), ECG

Treatment
- Antivenom if severe rxn: 1–2 vials over 30 min (after cutaneous test dose)
- Wound care, tetanus, supportive care: BZD, analgesia

**Disposition**
- Consider admission for observation & pain control

**Pearls**
- Painful abd muscle cramps can mimic peritonitis
- Red hourglass-shaped marking on spider’s abdomen

---

**Hymenoptera (Bee, Wasp, Stinging Ant)**

**History**
- Immediate pain & swelling at site of bite

**Findings**
- Local & systemic signs of allergic rxn can occur

**Treatment**
- Treat anaphylaxis/allergic rxn; local rxn treated w/ cleansing, ice packs, & elevation
- If present, stinger should be removed immediately by scraping it from the wound (bees)

**Disposition**
- Close wound care f/u; prescribe epinephrine auto-injectors in cases of anaphylaxis

**Pearls**
- The more rapid onset of sx(s), the more severe the rxn; IgE-mediated allergic rxn
- Rapid onset: 50% D in 30 min, 75% in 4 h; usually see fatal rxn following prior mild rxn
- Delayed rxn similar to serum sickness can present 10–14 d after a sting/bite

---

**Jellyfish Stings**

**History**
- Swimming in seawater w/ jellyfish
Findings
- Painful papular lesions & urticarial eruptions last min to h
- Systemic rxns rare; vomiting, muscle spasm, paresthesias, weakness, fever, respiratory distress, Irukandji syndrome: Rare, severe chest/abd/back pain, HTN, GI sx

Evaluation
- CBC, BMP, CK, coagulation profile, ECG

Treatment
- Analgesia, supportive care
- Tentacles should be removed w/ forceps; nematocysts should be scraped off w/ a knife/blade after dusting w/ talcum powder & covering w/ shaving cream
- Analgesia & after nematocyst removal wash w/ hot (40°C) salt water (helps w/ pain)
- Antivenom available for serious systemic effects (cardiopulmonary arrest, severe pain) from the Commonwealth Serum Laboratory in Melbourne, Australia

Disposition
- D/C if mild & pain controlled, admission for observation o/w

Pearls
- Box jellyfish are severely toxic, can induce respiratory & myocardial arrest in min
- Use seawater/acid/vinegar (not urine!) to wash; freshwater causes nematocysts to fire

OCCUPATIONAL EXPOSURE

Approach
- Institutional guidelines vary regarding occupational exposures of HC workers to bodily fluids
- Refer to CDC/local experts for recs on postexposure prophylaxis (PEP)
- National Clinicians’ Postexposure Prophylaxis Hotline (PEPline): (888) 448-4911

History
Any percutaneous injury, mucous membrane exposure, or exposure of nonintact skin to any blood & other bodily fluids considered potentially infectious

RFs: High-risk procedures, use of equipment w/o newer safety designs, failure to follow universal precautions

Findings

Physical examination nl; should be documented for future reference

Evaluation

Consent & test source pt for HIV, HBsAg, Hep C Ab (direct viral assays not rec)
Test HC worker for HIV & Hep C Ab, draw HBsAb titers if unknown immune status
Check serum hCG, CBC, BMP, LFTs, & UA before starting prophylaxis
If source pt is HIV+, ID consult for appropriate regimen based on source pt’s regimen

Treatment

HIV: 2 drug regimen (Combivir) × 4 wk; 3 drug regimen (Nelfinavir) for high-risk exposures
Hep B: Start vaccination series if unvaccinated, Hep B immune globulin (HBIG) if HBsAg+
Multiple doses HBIG w/i 1 wk of exposure provides 75% protection from infection
Hep C: CDC does not recommend use of interferon or ribavirin for HCV exposure
Consider interferon & ribavirin tx as soon as HCV seroconversion is documented

Disposition

F/u w/ ID specialist; fully inform risks & benefits of tx & nontx

Pearls

~80% ↓ rate of transmission w/ immediate initiation (w/i 2 h) of HIV PEP
Rates of occupational transmission after percutaneous exposure
HIV + source pt: 0.3%; Hep B + source pt: 5–20%; Hep C + source pt: 1–10%
<table>
<thead>
<tr>
<th>Name</th>
<th>Regimen</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combivir [Zidovudine (AZT)/Lamivudine (3TC)]</td>
<td>1 tablet (300 mg AZT, 150 mg 3TC) every 12 h</td>
<td>HA, malaise, fatigue, nausea, diarrhea, myalgias</td>
</tr>
<tr>
<td>Nelfinavir (Viracept)</td>
<td>1 tablet (250 mg) TID</td>
<td>Diarrhea, nausea, rash, fatigue, stomach cramps</td>
</tr>
</tbody>
</table>

**Occupational Exposure Risk Assessment**

<table>
<thead>
<tr>
<th>Low-risk Exposure</th>
<th>High-risk Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Instrument used for giving injection</td>
<td>Instrument visibly contaminated w/ source pt blood, directly placed in pt vein/artery</td>
</tr>
<tr>
<td>Superficial puncture in employee</td>
<td>Deep puncture in employee</td>
</tr>
<tr>
<td>Splash or mucosal exposure in employee</td>
<td>Terminal source pt (high viral load)</td>
</tr>
</tbody>
</table>


**BURNS**

**Approach**
- Early airway assessment, determine need for intubation (soot in airway, edema, voice Δ, deep facial burns, ↓ O₂ sat; transfer to burn center if intubated)
- 100% O₂ or O₂ by NRB mask until CO (10e) & other inhalation tox assessed
- Evaluate for concomitant trauma (fall, blast injury); maintain c-spine precautions
- Start IVF resuscitation early (almost universally required)
- Keep room warm to ↓ insensate losses

**History**
- How burn occurred (explosion? closed space?), duration of exposure, type of burn

**Findings**
- Assess burn
Evaluation
- Mental status on extrication, assess degree of burn, % of total body surface area
- Check CO level (10e), CBC, BMP, lactate, ABG, LFTs, coags, tox, T&S, UA, CXR

Treatment
- Early & generous analgesia: Morphine IV q5–10min titrated to pain
- Airway management: Intubate early
  - Toxic inhalation (cough, dyspnea, carbonaceous sputum, soot in oropharynx): Intubate or perform fiberoptic airway exam early; delay could cause ↑ airway edema → airway compromise, difficult/impossible intubation
- If >15% TBSA, aggressive IVF resuscitation, 2 LBIV through unburned skin
  - Parkland formula calculates IVF requirement in 1st 24 h after burn:
    - 4 mL × weight (kg) × BSA (2nd- & 3rd-degree burns)
    - Give ½ over 1st 8 h, other ½ over next 16 h; use LR to avoid NAGMA w/ NS
- Urinary catheter placement: Target urine output: 30–50 mL/h
- Burn mgmt: Irrigate w/ NS, remove debris, clothing, jewelry, & ruptured blisters (prevent future infection)
  - Apply silver sulfadiazine (antipseudomonal) ointment to denuded areas
  - Bacitracin only on face (silver sulfadiazine may cause discoloration)
- Immediate escharotomy for full-thickness circumferential burns that compromise distal neurovascular status or significantly ↓ chest compliance
- Tetanus prophylaxis, no role for steroids or immediate IV abx

Disposition
- Admit 2nd-degree burns 10–20% BSA (or 5–10% if <10 y/o), circumferential or if meet criteria below

Pearls
- Burns often progress in severity, watch for worsening burns
- Remove tar (asphalt burns) w/ mineral oil
- Consider cyanide w/ industrial/closed space fires, check lactate, treat w/ hydroxocobalamin
<table>
<thead>
<tr>
<th>Degree</th>
<th>Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st: Epidermis</td>
<td>Painful, erythematous, indurated area w/o blisters</td>
</tr>
<tr>
<td>2nd: Dermis</td>
<td>Blisters, painful, erythematous or mottled, indurated</td>
</tr>
<tr>
<td>3rd: Full-skin thickness</td>
<td>Charred, leathery, mottled/white, painless</td>
</tr>
<tr>
<td>4th: Full-tissue thickness</td>
<td>Includes SQ tissue, muscle, fat, blood vessels &amp; nerves, to bone. Catastrophic.</td>
</tr>
</tbody>
</table>

**Criteria for Transfer to Burn Unit**

- Burns >20% BSA (or >10% if age <10 or >50)
- 3rd-degree burns >5% BSA or 2nd-degree burns >20% BSA
- Burns involving face, eyes, ears, hands, feet, or perineum
- Burns a/w significant electrical, chemical, inhalational, or traumatic injury
- Burns suspected to be related to abuse
- Burns to pts w/ special psychosocial or rehabilitative care needs
**Approach**
- Early airway assessment, determine need for intubation (AMS)
  - 100% O₂ or O₂ by NRB mask until CO assessed
  - Pulse oximetry not useful b/c it will detect carboxyhemoglobin (COHgb) as oxyhemoglobin

**History**
- Exposure to CO from combustion, faulty heating, closed-space fire, defective automobile exhaust; often multiple people exposed/symptomatic
  - Mild poisoning: Frontal HA, N/V, DOE, dizziness/confusion
  - Severe exposure: Syncope, coma, or sz

**Findings**
- Mild confusion progressing to agitation, sz, coma
- May have subtle psychomotor abnormalities: Ataxia, muscle rigidity, tachycardia, hypotension, retinal hemorrhage, ↓ visual acuity, cyanosis, or pallor
- Neurologic findings primarily cerebellar: Dysmetria, ataxia, etc.

**Evaluation**
- ABG alone not useful b/c pO₂, a measure of dissolved O₂, will be nl; check ABG for COHgb via co-oximetry
  - Level is weakly correlated w/ tox but it confirms significant exposure
  - Level of <10–15% may be nl in smokers
  - Higher risk for myocardial injury: Check ECG esp if baseline CAD, risk fx, or high CO
- Assess for suicidal gesture; may need psychiatry consult

**Treatment**
- O₂ via NRB (60% O₂) at least, ideally deliver 100% O₂
- Airway management: If AMS, hypoxemia or shock → intubate
- Cardiac monitoring; admission if dysrhythmia or e/o ischemia on ECG
- Hyperbaric O₂ tx controversial but recommended by Undersea & Hyperbaric Med Society, potential long-term neuro sequelae benefit
- Fetal Hgb has higher affinity for CO than adult Hgb; lower threshold for hyperbaric O₂ in pregnant women

**Disposition**
Admission based on level & clinical findings; D/C asymptomatic pt w/ HbCO <10%

**Pearls**
- CO is the most common cause of D from acute poisoning & fires; reversibly binds Hb more avidly than O$_2$ → functional anemia
- May see delayed neurologic sequelae (personality Δ, HA, sz, parkinsonian Δ) 2–40 d after exposure; virtually universally resolve w/i 6 mo
- Half-life of COHgb: 300 min on RA, 90 min on 100% NRB, 30 min hyperbaric

<table>
<thead>
<tr>
<th>Indications for Hyperbaric O$_2$ in CO Poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transient or prolonged unconsciousness (syncope, coma)</td>
</tr>
<tr>
<td>CO level &gt;25–40%</td>
</tr>
<tr>
<td>Persistent neurologic disturbances</td>
</tr>
<tr>
<td>Cardiovascular Dysfxn</td>
</tr>
<tr>
<td>Severe acidosis</td>
</tr>
<tr>
<td>Pregnancy w/ CO level &gt;20% or signs of fetal distress</td>
</tr>
</tbody>
</table>

**DYSBARISM**

**Background**
- Atmospheric pressure at sea level = 760 mmHg = 14.7 psi: 1 atm
  - Each descent of 33 ft under water ↑ pressure by 1 atm
  - Dive tables & computers set standards for rate & depth of ascent to avoid dysbarism

**Approach**
- Careful hx: Length, depth, # of dives, interval btw dives, comorbid dz, sinus pain during dive, intoxication, onset of sxs, dive relative to decompression limits
- Divers Alert Network, Duke University: (919) 684-8111, 24-h med advice
**Decompression Sickness (DCS)**

**History**
- Improper dive time, depth, & ascent; sx can develop during or after (1–24 h) ascent, longer if air travel

**Findings**
- Fatigue, AMS, visual defects, lingual pallor, tachypnea, tachycardia, N/V, ↓ UOP, sz, neuro Δ, joint pain, lymphedema, pruritus

**Evaluation**
- Cardiac monitor, CBC, CMP, O$_2$ sat, tox screen, CO level, coags, CXR, head CT

**Treatment**
- 100% O$_2$ (NRB mask), place pt in L lateral decub & mild Trendelenburg, hyperbaric O$_2$, IVF (UOP 1.5 mL/kg/h) for recompression
- Goal of recompression to ↓ mechanical obstruction of air bubbles, ↑ tissue O$_2$ delivery
- Sx tx: Intubation (inflate cuff w/ saline), needle decompression, sz control

**Disposition**
- Ground transport, or low-flying air transport (cabin pressure <1000 ft)
- Admit to institution w/ hyperbaric O$_2$ capability

**Pearls**
- Spectrum of illness: Formation of small nitrogen gas bubbles in blood & tissues
- Depends on location & degree of bubble formation
- ↑ freq w/ longer & deeper dives, comorbid illness (COPD, CAD, PFO, asthma)
- Residual paralysis, myocardial necrosis, other ischemic injuries possible; early recognition & tx imperative
- Wait >12–48 h btw diving & flying, no diving for 7 d after DCS I, 28 d after DCS II

| Types of Decompression Sickness |
## Type I: Pain “the bends”
- Extremity/joint pain w/o localized tenderness or erythema
- Skin: Pruritus, rash, mottling or marbling of skin, violaceous rash
- Lymphatics: Venous stasis
- Inflate BP cuff to 150 mmHg over affected joint; if relieves pain, confirms dx

## Type II: CNS or pulm gas embolism
- Pulmonary sx: Pleuritic pain, respiratory distress, nonproductive cough
- Hypovolemic shock: Tachycardia, postural hypotension, cyanosis
- Nervous system: Mimics spinal cord trauma; ext weakness & paresthesias, moves proximally, focal neuro deficit, plegia, AMS, sz

---

### MIDDLE EAR BAROTRAUMA

#### History
- Usually occurs on descent; ↑ pain w/ ↑ water pressure on TM, equilibration via Eustachian tubes, rupture occurs b/w 5 & 17 ft → pain relief; vertigo, N/V, hearing loss

#### Findings
- Reversible Bell palsy from increased pressure to facial nerve in severe cases

#### Evaluation
- Concomitant eval for inner ear barotrauma

#### Treatment
- Nasal vasoconstrictor drops/spray to open fluid from middle ear; antihistamines, analgesia, pinch nose & swallow to displace fluid through Eustachian tube

#### Disposition
- ENT f/u in 2 wk

#### Pearl
- No benefit w/ abx; use occlusive earplugs when diving/showering until TM healed

---

### Other Dysbarisms
| Inner ear barotrauma | Occurs during descent; nausea, vertigo, tinnitus, hearing loss |
- Insufflation in ear canal using otoscope produces nystagmus
- Conservative mgmt, 1 wk bed rest, elevated HOB, no Valsalva

| Nitrogen narcosis | - “Rapture of the deep” from ↑ tissue nitrogen concentration
|                  | - Euphoria, false sense of well being, confusion, loss of judgment, disorientation, inappropriate laughter, ↓ motor control, paresthesias
|                  | - Start around 100 ft, resolves w/ ascent

| Facial barotrauma | - Neg. pressure generated in airspace created by mask over face
|                  | - If pt doesn’t force exhale through nose, get conjunctival edema, petechial hemorrhages over face, subconjunctival hemorrhages

| Arterial gas embolism (AGE) | - “The chokes” occur when diver doesn’t exhale properly during ascent
|                            | - Sudden onset stroke sx in 10 min of surfacing, dyspnea, hemoptysis
|                            | - Look for PFO, shouldn’t dive again, emergent recompression

| PTX/Pneumomediastinum | - Results from barotrauma, seen on CXR
|                       | - Pleuritic pain, dyspnea, subcutaneous emphysema/crepitus
|                       | - Unless hemodynamic compromise or tension, not life-threatening

---

**ELECTRICAL INJURY**

**Background**
- Current: Measure of amount of energy flowing through an object; in amperes (A)

**Approach**
- Early & continuous cardiac monitoring for dysrhythmias
- Evaluate for concomitant trauma (fall, injury); maintain c-spine precautions
- Divided in low voltage <500 & high voltage

**History**
- Usually obvious & reported (eg, occupational injury of electrician, home handyman); pt reports minor shock (tingling) related to home appliance use
- “3rd rail” contact from light-rail mass transportation system
- Toddler w/ burns to corners of mouth (chewing) or hands (playing w/...
- Bimodal distribution w/ most pts <6 or adult workers

**Findings**
- VF more common w/ low voltage AC, asystole w/ high voltage AC or DC
- Respiratory arrest via chest wall paralysis or respiratory center of brain possible
- Skin wounds may appear minor & entry/exit wounds may be present (examine bottoms of feet for exit); may be more severe than they appear due to deep-tissue injury
- Long-bone fx, scapular fx, shoulder dislocation, spinal fx from mechanical trauma caused by whole body tetanic contractions or trauma of being blown back
- Perforated TMs, delayed cataracts in 6% of pts

**Evaluation**
- ECG, CBC, BMP, cardiac enzymes (rhabdomyolysis), UA (myoglobin)

**Treatment**
- Resuscitate, eval for trauma, immobilize c-spine, continuous cardiac monitoring
- High-volume IV crystalloid (NS, avoid K-containing fluids)
- Urinary catheter placement: Target urine output: 0.5–1 mL/kg/h
- If rhabdomyolysis (↑ CK, +UA dip), maintain high UOP until urine dip neg.
- Goal serum pH 7.45–7.55
  - Alkaline urine (pH > 6.5) to ↑ excretion of acidic myoglobin by ↑ solubility; D$_5$W + 150 mEq NaHCO$_3$ OR D$_5$ ¼ NS or D$_5$ ½ NS + 100 mEq NaHCO$_3$
  - Diuresis w/ Lasix 20–40 mg IV or mannitol 25 g IV (then 12.5 g/kg/h) prn
- Treat wounds the same as thermal burns (10 d)
- Compartment pressures ± fasciotomy if sx of compartment syndrome
- Splint injured extremities in best “position of fxn” to minimize contractures

**Disposition**
- If asx & nl exam, can be D/C
- If mild cutaneous burns & nl ECG, nl urine dip, observe for 2 h, then
D/C
- ECG Δ, myoglobinuria, entry/exit burns, partial/full thickness burns: Admit burn center

**Pearls**
- Electrical injuries are often minor, but may be more serious than they 1st appear. If any concern, observe for 6–12 h.
- Pediatric oral “bite” burns may develop delayed labial artery bleed at 2–3 wk

<table>
<thead>
<tr>
<th>Types of Current</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct current (DC)</td>
</tr>
<tr>
<td>Occupational, high voltage: Current flows in 1 direction only; most pts are “blown” from this exposure &amp; suffer blunt trauma</td>
</tr>
<tr>
<td>Alternating current (AC)</td>
</tr>
<tr>
<td>Home, low voltage: 3× more dangerous than DC of same voltage due to continuous muscle contraction/tetany from current alternating direction of flow; pt “can’t let go”</td>
</tr>
<tr>
<td>Arc injury</td>
</tr>
<tr>
<td>Pt trapped in electrical arc b/w 2 objects; mostly serious b/c of ↑ risk of blunt trauma &amp; temp as high as 2500–5000°C causing burn</td>
</tr>
</tbody>
</table>

**HIGH-ALTITUDE ILLNESS**

**Background**
- Caused by acute exposure to hypobaric hypoxia (low PO2) usually above 8K ft
- Altitude illness is generally considered as a progressive spectrum from AMS to HACE
- Acclimatization allows body to minimize effects of hypoxia; ↑ RR (↓ PaCO2), ↑ CO, ↑ hematopoiesis & 2,3-DPG production (favors O2 release to tissues)
- Takes 5–7 d for full effect; inherent acclimatization ability varies by individual

**Approach**
- O2, descent, symptomatic relief; HAPE can be fatal w/i h unless treated

**History**
- Rapid ascent to altitude >8K ft, risk increased by exertion, past h/o
altitude illness
- Flu-like sxs, “hangover,” HA, fatigue, DOE, sleep disturbance, N/V, dizziness, paresthesias
- Sxs manifest 6–12 h after ascent, subside in 1–2 d or may progress to HAPE, HACE
- Watch for sxs of HAPE (dry cough, fever, SOB at rest) or HACE (ataxia, emesis, LOC)

Findings
- Depends on severity of altitude illness
- HAPE: Tachycardia, tachypnea, rales/wheeze, fever, orthopnea, pink/frothy sputum
- HACE: AMS, ataxia, sz, slurred speech, stupor, coma, D from brain herniation

Evaluation
- Clinical Dx
- HAPE: CXR (patchy infiltrates), US (comet tails), pulse oximetry (relative hypoxia)
- HACE: Head CT neg., MRI (white matter Δ showing ↑ edema)

Treatment
- Descent! If unable: O₂, symptomatic relief, bed rest
- Hyperbaric O₂ chamber: Used as temporizing measure until descent
- Meds: Unclear benefit but low risk:
  - Acetazolamide: 125–250 mg PO q12h; for ppx start 1d prior to ascent
  - Dexamethasone: 8 mg PO × 1, then 4 mg PO q6h
- In HAPE:
  - Nifedipine (pulm vasodilation): 10 mg PO q6h, SR 30 mg PO q8–12h (<90–120 mg/d)
  - Inhaled β-agonist (Salmeterol; clears alveolar fluid): Inhalation q12h
  - PDE-5 inhibitors (tadalafil, sildenafil) have shown efficacy in HAPE ppx & can be considered in tx: Tadalafil 10 mg q12h, sildenafil 50 mg q8h

Disposition
- Admit if hypoxic, dyspnea at rest; prognosis excellent for survivors

Pearls
- Avoid abrupt ascent, spend 1–2 nights at intermediate elevation, descend to sleep
Underlying medical conditions (COPD, CAD, HTN, SSD, pregnancy) affected more
Consider other causes of sx: PNA (HAPE does not usually cause fever), PE, SDH, CVA
Descent is the mainstay of any tx

<table>
<thead>
<tr>
<th>Dz</th>
<th>Signs &amp; Sxs</th>
<th>Altitude &amp; Course</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMS</td>
<td>Viral illness, HA + GI upset, insomnia, fatigue, lightheaded, Lake Louise Scoring system</td>
<td>8–10K ft; onset 6–12 h, peak 1–2 d, duration 3–5 d</td>
<td>Acetazolamide, O₂, Ibuprofen, avoid further ascent</td>
</tr>
<tr>
<td>HAPE</td>
<td>SOB at rest, fatigue, HA, anorexia, cyanosis, rales, tachypnea, tachycardia</td>
<td>&gt;14500 ft; onset 2–4 d, resolution 1–2 d after descent</td>
<td>O₂, descent, rest, nifedipine; hyperbaric if severe</td>
</tr>
<tr>
<td>HACE</td>
<td>HA, ataxia, slurred speech, AMS (hallucinations), insomnia, stupor, coma</td>
<td>&gt;12K ft; onset 1–3 d, peak 5–9 d, resolves 3–7 d after descent</td>
<td>O₂, descent, rest; dexamethasone; hyperbaric if severe</td>
</tr>
<tr>
<td>High altitude retinal hemorrhage</td>
<td>Usually asymptomatic, sometimes central scotoma</td>
<td>&gt;17500 ft; ? onset/peak; resolves 1–3 wk</td>
<td>No emergent tx</td>
</tr>
</tbody>
</table>

HYPOTHERMIA

Background
- Multiple classifications of hypothermia based on severity & etiology

Approach
- Careful hx: Determine etiology of hypothermia: Environmental exposure vs. medical
- Environmental hypothermia can occur even in the absence of freezing weather (malnourished pt, elderly)
- Many medical etiologies: Hypothyroidism (myxedema coma), hypoglycemia, hypoadrenalism, sepsis, hypothalamic lesion (eg, 2/2 trauma, tumor, stroke), dermatologic conditions that prevent heat conservation (burns, erythrodermas)
If unresponsive, check BS/give D50, give naloxone 2 mg

History
- Environmental exposure, drug use, trauma, comorbid illnesses

Findings
- Based on degree of hypothermia (table below)

Evaluation
- Obtain core temp (bladder, rectum, esophagus: All may be inaccurate)
- Cardiac monitor, CBC (Hct ↑ 2% for every 1° ↓ temp), CMP (↑ K bad sign), tox screen, coags, CXR, lipase (cold-induced pancreatitis), CK, UA (rhabdo), ABG, head CT
- ECG shows Osborn waves (J pt deflection in same direction as QRS), <32°C/90°F
- Interval prolongation (PR, QRS, QT), AF w/ slow ventricular response (common)

Treatment
- Rewarm as per table below; intubate as needed, remove wet clothing
- Maintain horizontal position, avoid movement, limit manipulation to essential tasks. However, this should not prevent CPR or other critical interventions.
- Monitor ECG, check for pulse q1min; chest compressions may cause ventricular dysrhythmias, perform only if no pulse
- If no cardiac activity, start CPR
  - VF or VT: Defibrillate up to 3 times
  - Core temp <30°C, cont compressions/rewarming, no ACLS meds/shock until >30°C
  - Core temp >30°C, ACLS protocol w/ meds/shock, allow longer time b/w doses
  - Cont resuscitation until core temp >32°C/90°F
    - Consider hydrocortisone 250 mg IV or levothyroxine 250–500 μg if doesn’t rewarm w/ above

Disposition
- Based on severity of hypothermia (table below)

Pearls
- Hypothermic bradycardia is refractory to atropine since not vagally
mediated; no indication for temporary pacing
- Core temp afterdrop: Peripheral vasodilation from rewarming extremities may cause return of cooler peripheral blood to core
- Consider femoral line placement if needed to avoid cardiac stimulation (vs. IJ, SC)
- “You’re not dead until you’re warm & dead”; aggressively rewarm before stopping efforts

### Classifications of Hypothermia

<table>
<thead>
<tr>
<th></th>
<th>Physiologic Response</th>
<th>Clinical Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (90–95°F, 32–35°C)</td>
<td>Increased: HR, BP, CO, RR, metabolic activity, shivering, cold diuresis</td>
<td>Dizziness, lethargy, confusion, amnesia, apathy, dysarthria, nausea, ataxia, loss of fine-motor skills</td>
</tr>
<tr>
<td>Moderate (86–90°F, 30–32°C)</td>
<td>Decreased: HR, BP, CO, RR, metabolic activity, cold diuresis, shivering stops</td>
<td>Delirium (paradoxic undressing), stupor, pupillary dilatation, ↓ reflexes</td>
</tr>
<tr>
<td>Severe (&lt;86°F, &lt;30°C)</td>
<td>Decreased: HR, BP, CO, RR, metabolic activity, no shivering</td>
<td>Unresponsive, fixed &amp; dilated pupils, rigid, very cold skin, coma, pulm edema; ↑ risk of ventricular fibrillation &amp; asystole</td>
</tr>
</tbody>
</table>

### Rewarming Strategies by Severity of Hypothermia

<table>
<thead>
<tr>
<th></th>
<th>Rewarming Strategy</th>
<th>Tx</th>
<th>Disposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (90–95°F, 32–35°C)</td>
<td>Passive rewarming (PR)</td>
<td>Warm blankets, heat lamps, ACLS if cardiac arrest</td>
<td>Likely D/C</td>
</tr>
<tr>
<td>Moderate (86–90°F, 30–32°C)</td>
<td>PR, active external rewarming (AER) to trunk only</td>
<td>Hot water bottles (45–65°C) to axilla &amp; groin, ACLS for cardiac arrest</td>
<td>Admit, cardiac monitoring</td>
</tr>
<tr>
<td>Severe (&lt;86°F, &lt;30°C)</td>
<td>PR, AER, active internal rewarming</td>
<td>Warm IVF (NS 45°C), warm humidified O₂ (45°C), If cardiac arrest, shock (no ACLS meds); CP bypass/pl lavage, central venous “radiator” cath</td>
<td>Admit, likely ICU</td>
</tr>
</tbody>
</table>
**Frostbite**

**History**
- Cold exposure, numbness of body part → loss of sensation

**Findings**
- Distal body part most commonly affected (fingers, nose, toes, ears)
- Caused by both immediate cell D from cold & delayed injury from inflammatory response
- Skin initially white, waxy, insensate → erythematous, edematous, painful 48–72 h after rewarming → bleb formation, devitalized tissue demarcation over weeks

**Evaluation**
- Check core temp to look for systemic hypothermia
- Superficial: Areas of pallor & edema, local anesthesia, potentially clear blisters, erythema, no tissue loss
- Deep: Hemorrhagic blisters, eschar, if severe extends to muscle/bone, mummification

**Treatment**
- Handle tissue gently, keep extremity elevated, sterile/nonadherent dressing
- Rapid rewarming of frozen extr in gentle warm water bath (40–42°C), ROM exercise in bath, avoid water temp falling outside of range; 30 min if superficial/
  60 min if deep
- Consider intra-arterial tPA in severe cases
- Topical aloe vera q6h
- Aspirate & débride clear blisters, only aspirate (do not débride) hemorrhagic blisters to avoid desiccation, infection of deeper tissues
- Tetanus prophylaxis, consider ppx abx
- Early surgical intervention not indicated other than escharotomy for circumferential limb lesions (very uncommon)

**Disposition**
- Refer to burn service; consider admission for 24–48 h to observe for progression

**Pearls**
Long-term cx: Cold insensitivity, paresthesias, nail loss, joint stiffness
Avoid refreezing, if unable to maintain warmth to affected part (e.g. prehospital) do not rewarm

HYPERTERMIA

Background
- Spectrum of heat-related illnesses including heat rash, cramps, syncope, stroke

Approach
- Careful hx: Determine etiology of hyperthermia: External (environmental) or internal (toxic/metabolic) factors, environmental hypothermia can occur even in absence of exertion (malnourished pt, chronically ill, elderly)
- Look for medication related hyperthermia: MH, NMS, SS
- Use rectal thermometer to determine core temperature

EXTERNAL HEAT EMERGENCIES

Heat Cramps

History
- Brief, intermittent, severe muscle cramping usually following cessation of strenuous activity. Often in abd or calf muscles.

Findings
- Euthermic, clinical signs of dehydration

Evaluation
- BMP (↓ Na, ↓ Cl), urine lytes optional (↓ urinary Na & Cl from sweating)

Treatment
- Oral salt or electrolyte repletion tablets or sports drinks; IV hydration rarely required

Disposition
- Home after observation for sx relief

Pearl
- Related to electrolyte deficiency; electrolyte enhanced sports drinks
may be helpful although may cause diarrhea due to the high sugar content

**Heat Edema**

**History**
- Swollen feet/ankles after long periods of sitting/standing due to hydrostatic pressure, vasodilation & orthostatic pooling → vascular leak, interstitial fluid accumulation
- No underlying hepatic, lymphatic, cardiac, or venous dz

**Findings**
- Euthermic, B LE pitting edema w/o signs of CHF or renal failure

**Evaluation**
- BMP, UA for proteinuria, CXR for pulm edema, ECG for e/o LVH, RH strain

**Treatment**
- Elevate lower extremities, provide support hose
- No evidence that diuretics help

**Disposition**
- Home after reassurance, PCP f/u

**Pearl**
- Dx of exclusion

**Heat Rash (Prickly Heat, Miliaria, Lichen Tropicus)**

**History**
- Sweat gland blockage w/ localized inflammatory response
- Often seen in pts newly arrived to subtropical/tropical areas or during heat waves

**Findings**
- Euthermic, erythema w/ pruritic vesicles, primarily in intertriginous areas, then becomes anhidrotic
- Occasionally will become superinfected, usually *Staph*

**Evaluation**
- None

**Treatment**
- Treat pruritus: Diphenhydramine 25–50 mg PO or hydroxyzine 25 mg PO
Desquamate skin w/ chlorhexidine antibacterial soap or salicylate-containing topical scrub

Disposition
- Home, PCP f/u

Pearl
- Avoid routine talcum powder application, which may block sweat glands

Heat Syncope

History
- Syncopal event in warm/humid weather or following strenuous activity
- Heat → vasodilation → peripheral intravascular blood pooling, ↓ central venous return

Findings
- Euthermic, nl exam

Evaluation
- EKG, eval for other causes of syncope (see 1c)
- Syncope/presyncope sx should resolve w/i 30 min, if not consider further w/u

Treatment
- PO or IV hydration

Disposition
- Home, PCP f/u

Pearl
- Dx of exclusion, diagnose only in young healthy pts w/ no cardiac dz

Heat Exhaustion

History
- Gradual onset, extreme fatigue in warm/humid weather following strenuous activity, profuse sweating, dizziness, N/V; often pale w/ cool, moist skin
- Inadequate PO intake

Findings
- Mild hyperthermia, may reach 40°C (104°F), nl mental status

Evaluation
- BMP for electrolyte imbalance, UA (rhabdomyolysis uncommon)
Treatment
- IV hydration (PO if pt tolerates), replace w/ NS (or alternate w/ ½ NS if ↑ Na)

Disposition
- Observation w/ continued hydration until normothermic w/ good UOP

Pearl
- No value w/ fever-reducing medications

Heat Stroke

History
- Acute onset when compared to heat exhaustion
- Classic: Occurs during heat waves, affects susceptible pts: Elderly, chronically ill, scleroderma, CF, burns, alcoholics, homeless, mentally ill, on diuretics or anti-chol
- Exertional: Occurs in pts who are overwhelmed by heat overproduction: Athletes, military recruits, thyroid storm, pheochromocytoma, sympathomimetic overdose

Findings
- Hyperthermia >41°C/106°F, CNS Dysfxn: Confusion, disorientation, delirium
- Classic: Anhidrotic, tachypnea
- Exertional: Diaphoretic until “sweat gland fatigue”
- Muscles usually flaccid in HS, if rigid consider NMS, etc.

Evaluation
- BMP (electrolyte imbalance, ↓ blood sugar), LFTs (hepatic damage common), coags (DIC possible but uncommon), CK & UA (rhabdo common in exertional heat stroke)

Treatment
- Aggressive fluid resuscitation: Cooling procedures → vasoconstriction, can ↑ BP so may need to guide fluid status by UOP, IVC US, CVP, etc.
- Rapid cooling indicated, ↓ by 0.2°C/min → 39°C/102.2°F to avoid overshooting
- Ice water immersion: Can ↓ core temp in 10–40 min
- Evaporation: Spray water mist & use fan, maintains cutaneous vasodilation, avoids heat generation by shivering, 7× more efficient than ice packing but 2× as fast
- Adjunctive cooling strategies: Strategic ice packs near large blood
vessels (ant neck, axilla, groin), ice water gastric lavage at NS 200 mL/h
• Mannitol 50–100 g IV ↑ renal blood flow, ↓ cerebral edema
• Treat rhabdo w/ IVF, HD if anuric, tx coagulopathy w/ FFP

Disposition
• Admit for ongoing tx & cooling

Pearls
• Avoid alcohol sponge baths, dantrolene
• Avoid antipyretics (APAP damages liver, salicylates aggravate bleeding)
• Avoid α-adrenergic drugs (promote vasoconstriction, ↑ hepatic/renal damage, CO same)
• Avoid atropine/anticholinergics that ↓ sweating; use BZD to stop shivering
• Avoid neuroleptics (chlorpromazine): ↓ sz threshold, interfere w/ thermoregulation, etc.

INTERNAL HEAT EMERGENCIES

Malignant Hyperthermia (MH)

History
• Acute ↑ body temp after administration of inhaled anesthetic or succinylcholine
• Genetic abnlty of skeletal muscle sarcoplasmic reticulum → inappropriate Ca release → severe tetany & spasm (heat); often FH of adverse rxn to anesthesia

Findings
• Acute hyperthermia after anesthetic, hypercapnia (early sign), muscular rigidity, masseter muscle spasm, acidosis, tachycardia, rhabdomyolysis

Evaluation
• Check core temp, electrolytes, CK

Treatment
• Stop offending agent, increase ventilation rate, dantrolene 2.5 mg/kg bolus IV, repeat doses of 1 mg/kg until sx subsides; MH protocols

Disposition
• Usually occurs in OR, admission for supportive care
Pearl
• MH hotline: 1-800-MH-HYPER (1-800-644-9737), ask for “Index Zero”

Neuroleptic Malignant Syndrome (NMS)

History
• Antipsychotic use (phenothiazines, butyrophenones, thioxanthenes, lithium, TCAs); recent initiation or dose ↑ (2/3 of cases in 1st wk)
• Anti-Parkinson medication withdrawal
• Dopamine receptor blockade → severe muscle spasticity & dystonia, heat overproduction

Findings
• Triad: Hyperthermia, muscular rigidity (lead pipe), autonomic Dysfxn
• AMS, dyskinesia, tachycardia, dyspnea, diaphoresis, dysphagia, tremor, incontinence

Evaluation
• UA for myoglobin, CK for rhabdomyolysis, ↑ WBC, Chem, tox

Treatment
• Stop offending agent, mainstay is supportive tx: IVF, BZDs
• May consider danthrolene (as for MH), whole body cooling w/ evaporating fans
• Dopamine antagonists (bromocriptine 2.5 mg PO q8h, amantadine 200 mg PO q12h)
• Treat rhabdomyolysis w/ IVF, alkaline urine (pH > 6.5) to ↑ myoglobin excretion
• Keep Na in IVF close to 154 mEq/L; add NaHCO₃

Disposition
• Admission; mortality 10–20%

Pearl
• NMS hotline: 1-888-667-8367

Serotonin Syndrome (SS)

History
• Drug & food interactions: MAOI + tyramine (found in aged cheese, wine, etc.); caused by excessive serotonin activity in spinal cord & brain

Findings
• Hunter criteria: Combination of clonus, hyperthermia, agitation,
- diaphoresis, ocular clonus, hyperreflexia, tremor, hypertonia
- Diarrhea, cramps, hypersalivation (similar to NMS), autonomic Dysfxn

### Evaluation
- UA for myoglobin, CK for rhabdomyolysis, CBC, Chem, tox
- Clinical dx, must confirm h/o 2 serotonergic agents, r/o toxic, metabolic, infectious cause

### Treatment
- Stop offending agent, supportive tx, whole body cooling, treat rhabdo w/ IVF**
- BZD: May require high doses
- Dantrolene not recommended: May ↑ central serotonin metabolism & production
- Nonsp serotonin inhib: Cyproheptadine 12 mg PO then 2 mg PO q2h

### Disposition
- Admission; most resolve w/ no sequelae in 24–36 h after starting tx

### Pearl
- Pts must stop MAOI for 6 wk prior to starting SSRI

#### Differentiating NMS and Serotonin Syndrome

<table>
<thead>
<tr>
<th></th>
<th>NMS</th>
<th>SS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
<td>● A/w neuroleptic use</td>
<td>● A/w serotonergic agents</td>
</tr>
<tr>
<td></td>
<td>● Idiosyncratic rxn to therapeutic doses</td>
<td>● Manifestation of tox; often from combination of 2 serotonergic drugs</td>
</tr>
<tr>
<td><strong>Timing</strong></td>
<td>● Slow onset (days → weeks)</td>
<td>● Rapid onset &amp; progression</td>
</tr>
<tr>
<td></td>
<td>● Slow progression (24–72 h)</td>
<td></td>
</tr>
<tr>
<td><strong>Sxs</strong></td>
<td>● Bradykinesia, lead-pipe rigidity</td>
<td>● Hyperkinesia, less rigidity</td>
</tr>
<tr>
<td><strong>Tx</strong></td>
<td>● BZD</td>
<td>● BZD</td>
</tr>
<tr>
<td></td>
<td>● Dantrolene</td>
<td>● Serotonin inhibitors</td>
</tr>
<tr>
<td></td>
<td>● Dopamine antagonists</td>
<td>● NOT dantrolene</td>
</tr>
</tbody>
</table>
restarts in SR but CNS injury & concussion may cause respiratory arrest w/ secondary cardiac arrest

**Approach**
- Early & continuous cardiac monitoring for dysrhythmias
- Evaluate for concomitant trauma (fall, injury); maintain c-spine precautions
- Reverse triage in field: Lightning victims that appear dead should get CPR as pts can be pulseless w/ fixed, dilated pupils & still have good survivability

**History**
- Usually obvious, reported lightning strike near pt; often witnessed collapse

**Findings**
- TM rupture, transient vasospasm (cool ext), symp nervous system instability
- Various burn patterns
  - Linear: Caused by steam production during flashover (charge passes over surface of body only) where sweat accumulates
  - Punctate: Multiple cigarette-like burns
  - Feathering: Not actual burns; electron showers make a ferning pattern on skin (Lichtenberg figures)
  - Thermal: Usually from burnt clothing
- Ocular pathology: Corneal lesions, hyphema, vitreous hemorrhage, retinal detachment, cataracts develop long term
- Keraunoparalysis: Transient paralysis that can occur, likely 2/2 vasospasm, LE > UE, usually resolves in hours, still will need eval for true spinal cord pathology 2/2 trauma

**Evaluation**
- ECG, CBC, BMP, CK (rhabdomyolysis), UA (myoglobin), head CT if unresponsive

**Treatment**
- Resuscitate, eval for trauma, immobilize c-spine, continuous cardiac monitoring
- High-volume IV crystalloid (NS); same tx as electrical injury (10f)
- Urinary catheter placement: Target urine output 1–1.5 mL/kg/h (200–
300 mL/h)
- If rhabdomyolysis (↑ CK, +UA dip), maintain high UOP until urine dip neg.
- Treat wounds the same as thermal burns (10 d), tetanus, wound care, etc.
- Splint injured extremities in best “position of fxn” to minimize contractures & edema

Disposition
- If asx & nl exam, can be discharged; good prognosis if survive in field
- ECG Δ, myoglobinuria, entry/exit burns, partial/full thickness burns: Admit to burn center

Pearl
- Lightning causes ~50–300 Ds in US each year, 25–30% of lightning strike victims die, of those that survive ~75% have permanent disability

DROWNING

Background
- AHA guidelines suggest a broad definition of drowning to include D from drowning, near drowning (no longer used!), wet drowning, etc.
- Definition: Respiratory impairment from being submerged under a liquid
- >4000 drowning Ds annually in US; toddlers & teenage boys at greatest risk
- Freshwater vs. saltwater vs. chlorinated pool water: No difference, theoretical diff only
- 1° insult to lung; water moves across alveolar–capillary membrane, destroys (freshwater) or washes out (salt water) surfactant → hypoxia
- Diving reflex = immersion of face in water <68°F, blood shunts from periphery → brain & heart → apnea, bradycardia, hypothermia → ↓ metabolic demand prevents/delays severe cerebral hypoxia

Approach
- Careful hx: Possible diving (cervical spine or head) injury vs. 1° drowning, intoxicants, comorbidity, submersion time, water temp, initial rescuer response (ACLS)
- Extricate pt, remove wet clothing, ABCs, ACLS, intubation as
appropriate
- Bedside glucose or D50 if AMS
- Cervical spine immobilization if suspicion for head or neck injury (diving, pool accident)

History
- Submersion event

Findings
- Variable presentation (awake, coma, cardiac arrest)
- Wheezes/rales/rhonchi, ecchymosis/crepitus/other signs of trauma on exam

Evaluation
- CBC, BMP, LFTs, tox, CXR may show pulmonary edema or aspiration 2–6 h after event, CT head & c-spine if concern for trauma, AMS

Treatment
- ABCs, intubation or supplemental O₂, CPR, ACLS, Foley placement
- Measure core temp, treat for hypothermia if indicated to temp 30°C/86°F
- Ventilator PEEP 5–10 mm H₂O to ↓ intrapulmonary shunting

Disposition
- Admission for continued tx, watch for signs of ARDS/VALI
- May develop pulm Δ even after mild submersion, observe asx pts for at least 8 h

Pearls
- Prophylactic abx & steroids not indicated
- Artificially induced hypothermia does not improve outcome

---

**BOTULISM**

Background
- Caused by neurotoxin produced by anaerobic gram-positive rod *C. botulinum*
- Spore-forming bacterium found in soil & water, particularly in CA, UT, PA
• Blocks ACh release at neuromuscular jxn & autonomic ganglions (nicotinic receptors)

**Approach**
• Early airway management & ventilatory support
• Contact CDC Botulism center (404-639-2206/3311) for antitoxin, BabyBIG from CA

**History**
• 3 main etiologies: Infant, foodborne, or wound; also potential for bioterrorism
• Infant: Consumption of unpasteurized honey or likely exposure to endemic spores (feeding through a nipple dropped on the ground, sucking on fingers after playing in dirt)
• Foodborne (adult): Ingestion of food contaminated w/ spores, usually home-canned goods
• Wound: Spores infiltrate skin wounds, germinate, & release tox into the bloodstream

**Findings**
• Weakness, flaccid paralysis, respiratory arrest, autonomic dysfxn; CN affected 1st
• Infant: Weak cry, poor sucking, flaccid/hypotonic muscles
• Foodborne (12–36 h) & wound (several days): Autonomic dysfxn, descending symmetric motor paralysis, nl sensorium

**Evaluation**
• None needed prior to intervention
• Collect serum, stool, wound, & food samples for CDC testing

**Treatment**
• ABCs, intubation or supplemental O₂
• Administer antitoxin 1 vial IV to adults & children
• Infants need only supportive care, no antitoxin

**Disposition**
• Admission to ICU for ventilatory support

**Pearls**
• Consider botulism in all infant sepsis workups
• Artificially induced hypothermia does not improve outcome
- AGs, magnesium contraindicated as they potentiate neuromuscular blockade
- Recovery of strength may take ~4 mo; may require respiratory support for months
ANAPHYLAXIS AND ANGIOEDEMA

Approach
- Eval & treat anyone w/ potential anaphylaxis immediately; can deteriorate rapidly
- Anaphylaxis median time to resp/card arrest: 5 min (2/2 drug), 15 min (2/2 venom), 30 min (2/2 food) (Curr Opin Allergy Clin Immunol 2013;13(3):263)

Definition
- Anaphylaxis – acute-onset occasionally life-threatening IgE-mediated rxn causing multisystem Dysfxn: skin (eg, urticaria), mucosa (eg, angioedema), GI (eg, n/v), respiratory (eg, bronchospasm), circulatory (eg, hypotension, syncope), neuro (eg, AMS)
- Absence of cutaneous sx rare (J Allergy Clin Immunol 2005;115:S485)
- DDx: anaphylactoid rxn, angioedema, neurocardiogenic syndromes, malignancies a/w flushing (eg, carcinoid), scombroid toxicity, systemic mastocytosis, other causes of shock or respiratory collapse (J Allergy Clin Immunol 2005;115:S485)

<table>
<thead>
<tr>
<th>Anaphylaxis &amp; Anaphylactic-like Syndromes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Anaphylaxis</td>
</tr>
<tr>
<td>Anaphylactoid rxn</td>
</tr>
<tr>
<td>Angioedema</td>
</tr>
</tbody>
</table>

History
- Sx: sudden-onset is key; sx can involve many organ sx – hives, swelling of tongue/throat, hoarseness, dyspnea, N/V, abd cramps,
presyncope

- Assess recent exposures: Foods (nuts, egg, shellfish), meds (abx, NSAIDs, vancomycin, iodine contrast; ACEi for angioedema), enzymes (insulin, trypsin, etc), airborne allergens (pollen, mold), venoms (bees, fire ants, snakes), exercise-induced, latex, idiopathic
- Check PMH for atopy, hypersensitivity syndromes, or hereditary angioedema

**Physical Exam**

- Urticaria, conjunctival injection, diffuse erythema, facial or oropharyngeal swelling, drooling, hoarseness, stridor, wheezing, ↓BP

**Evaluation & Treatment**

- Labs not routinely indicated; consider serum tryptase during acute event esp if idiopathic or dx uncertain (tryptase elevation sp but insensitive for IgE-mediated rxns)

<table>
<thead>
<tr>
<th>Tx</th>
<th>Type of rxn</th>
<th>Tx Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Most allergic rxns</td>
<td>Remove allergen</td>
</tr>
<tr>
<td></td>
<td></td>
<td>H1-blocker: Diphenhydramine 25–50 mg PO or IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>H2-blocker: Ranitidine 150 mg PO or 50 mg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prednisone 60 mg or Methylprednisolone 125 mg IV</td>
</tr>
<tr>
<td><strong>Intubation:</strong></td>
<td>Anaphylaxis (<a href="#">J Allergy Clin Immunol 2005;115:S485; Ann Emerg Med 2006;47:373</a>)</td>
<td>Consider early, consider fiberoptic/awake</td>
</tr>
<tr>
<td>All txs for allergic rxn (IV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVF bolus</td>
<td>Epinephrine: (Repeat prn, ± infusion)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IM: 0.3–0.5 mg (1:1000)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IV: 0.1–0.25 mg (1:10000) if severe (IM is safer)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neb: 0.5 mL 2.25% epi in 2.5 mL NS (No IV)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epi gtt: 1–4 μg/min (titrate to stability)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C1 esterase deficiency: FFP</td>
<td></td>
</tr>
<tr>
<td>Stable: Fiberoptic eval ± intubation</td>
<td>Unstable: Cricothyrotomy</td>
<td></td>
</tr>
</tbody>
</table>
H1-blocker + H2-blocker > H1-blocker alone for urticaria (NEJM 2004;351:2203)

Epi IM vs. SC: IM preferred → more rapid absorption (J Allergy Clin Immunol 2001;108:871)

Epi IM vs. IV: IM preferred → safer (World Allergy Organ J 2015;8(1):32)

Epi & cardiac dz: Place on monitor → epi is relatively contraindicated w/ CAD, but mortality of anaphylaxis w/o epi >> mortality from arrhythmia 2/2 epi

Epi vs. glucocorticoids: Epi first-line in anaphylaxis; glucocorticoids best for delayed sx, but evidence on acute benefit inconclusive (Cochrane 2012;4:CD007596)

Disposition

Home: pts w/ either local rxns (w/o airway involvement) or delayed-presentation generalized rxn (w/o airway involvement)

- Provide EpiPen Rx (esp if unknown cause) & allergist f/u

Biphasic rxns: may occur in up to 20% of cases; median onset 11–15 h after initial sx resolve; risk reduced if 2/2 food, & increased if 2/2 drug or idiopathic; higher risk if initially hypotensive (Immunol Allergy Clin North Am 2007;27(2):309; J Allergy Clin Immunol Pract 2015;3(3):408; Ann Allergy Asthma Immunol 2015;115(4):312)

- Risk of clinically important biphasic rxn small (<0.5%) (Annals Emerg Med 2014;63(6):736)

Consider observation if required epi or if high risk for biphasic rxn; however, there is no clear data on recommended duration of observation, & early d/c may be appropriate (Allergy 2014;69(6):791)

Admit to ICU: Severe anaphylactic rxn (mx epi, epi gtt, airway compromise)

Pearls

- ACEI can cause angioedema at any time, independent of length of use
- PCN allergy: IgE-mediated allergy confers low (~1%) risk of cross-reactivity w/ cephalosporins; however, avoid if rxn is severe (NEJM 2006;354:601)

See also Chapter 1 (Cardiac tamponade), Chapter 2 (Respiratory
distress, Hemoptysis), Chapter 5 (Altered mental status, Seizure, Brain tumor, CNS infections), Chapter 9 (SIADH, Hypercalcemia), Chapter 12 (Cauda equine syndrome)

NEUTROPENIC FEVER

Overview (Clin Infect Dis 2011;52(4):e56)

- **Definition:** Fever (single temp >38.3°C or temp >38°C for 1 h) + Neutropenia (ANC <500 or predicted <500 w/l 24 h or “functional neutropenia” [eg, AML])
- **Approach:** Early IV access, IVFs, & abx; most pts will not end up having identifiable infxn (only 20–30%), but those who do can deteriorate quickly
- **Etiology:** Infxn found in 20–30% (bacteremia in 10–25%); fungal infxn rare unless neutropenia >1 wk; consider also viral, drugs (chemo)

### Differential for Fever in Neutropenic pt

<table>
<thead>
<tr>
<th>Category</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial</td>
<td>HEENT (sinusitis, mucositis, oitis, pharyngitis), Pulm (PNA, TB, pneumonia), GI (colitis, mucositis, hepatobiliary), GU (UTI, pyelo), cardiac (endocarditis), neuro (meningoencephalitis, epidural abscess), skin (cellulitis, line-related infxn, abscess [inc perianal])</td>
</tr>
<tr>
<td>Viral</td>
<td>Influenza (in epidemics), RSV, other viral pathogens, HSV, CMV, EBV</td>
</tr>
<tr>
<td>Fungal</td>
<td>Candidiasis, aspergillosis (most common life-threatening)</td>
</tr>
<tr>
<td>Drug-related</td>
<td>Many chemo agents can cause immune-mediated febrile rxns</td>
</tr>
</tbody>
</table>

**History**

- Date of fever onset & last chemo (ANC nadirs 10–14 d after chemo); thorough ROS
- Assess RFs warranting inpt care: MASCC score (see Disposition)

**Physical Exam**

- Examine skin, mouth, lung, abdomen, catheter/surgical sites, perirectal area (No DRE)

**Evaluation**

- CBC w/ diff, Chem 20, coags, UA/urine cx, blood cx (at least 2 + any catheter port if present), ± CXR if resp sx
• ±Additional labs: Coags, culture (stool/sputum/peritoneal/CSF)
• Imaging: Consider imaging of chest, abdomen/pelvis, sinuses, brain

<table>
<thead>
<tr>
<th>Low-risk Criteria for Neutropenic Fever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (J Clin Oncol 2000;18:3038)</td>
</tr>
<tr>
<td>Pediatrics (≤16 y/o) (J Clin Oncol 2000;18:1012)</td>
</tr>
</tbody>
</table>

Treatment (Clin Infect Dis 2011;52(4):e56)
• Empiric tx depends on risk level (see above): low-risk (PO, outpt) vs. high-risk (IV, inpt)
  • Low risk: Ciprofloxacin + (amoxicillin/clavulanate or clindamycin [if PCN allergic])
    • If no RFs, mortality & tx failure w/ oral tx = similar to iv tx (Cochrane 2013;10:CD003992)
  • High risk: Antipseudomonas (ceftazidime, cefepime, carbapenem [not ertapenem])
    • If PCN-allergic: levofloxacin + aztreonam or AG
    • If cx: Can add AG or quinolone for additional GNR synergy
    • If line-infxn, PNA, hypotension: Add vancomycin
    • If MDRO, consider carbapenem (if ESBL), vancomycin (if MRSA), linezolid (if VRE)
    • Antifungals generally not indicated except if strong hx, shock, or recent HSCT

Disposition
• Admit all high-risk pts; low-risk pts can be d/c-ed only if close outpt f/u guaranteed
• CISNE score may outperform MASCC Risk Index in identifying low-risk pts who present to ED for eval (Ann Emerg Med 2015;PMID 28041827)

<table>
<thead>
<tr>
<th>Multinational Association for Supportive Care in Cancer (MASCC) Risk Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criteria</td>
</tr>
<tr>
<td>No or mild sxs</td>
</tr>
<tr>
<td>No hypotension (SBP &gt; 90 mmHg)</td>
</tr>
<tr>
<td>No active COPD (O₂, steroids, bronchodilators)</td>
</tr>
</tbody>
</table>

*PPV (%) = Predictive Positive Value
<table>
<thead>
<tr>
<th>Criteria</th>
<th>Pts</th>
<th>Rate of Adverse Outcome by Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECOG performance status ≥2*</td>
<td>2</td>
<td>Outcome</td>
</tr>
<tr>
<td>Stress-induced hyperglycemia</td>
<td>2</td>
<td>0 (%)</td>
</tr>
<tr>
<td>Active COPD</td>
<td>1</td>
<td>1-2</td>
</tr>
<tr>
<td>Chronic cardiovascular dz</td>
<td>1</td>
<td>3 (%)</td>
</tr>
<tr>
<td>Mucositis NCI grade ≥2**</td>
<td>1</td>
<td>Mortality</td>
</tr>
<tr>
<td>Monocytes &lt;200/μL</td>
<td>1</td>
<td>0 (%)</td>
</tr>
</tbody>
</table>


**TUMOR LYSIS SYNDROME**


- **Definition:** ≥2 metabolic abnlty (<25% ↑ K, ↑ PO₄, ↓ Ca, ↑ uric acid) w/i 3 d before or 7 d after the start of chemo, AND e/o AKI (GFR ≤ 60), arrhythmia, or sz
- **Etiology:** Rapid destruction neoplastic cells → release of nucleic acids, K, PO₄ → uric acid (from nucleic acids), AKI (2/2 uric acid), ↓ Ca (2/2 PO₄)

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*Am J Clin Oncol* 1982;5:649. **NCI Mucositis Grades:** 0 (none), 1 (painless ulcers, erythema, or mild soreness in absence of lesions), 2 (painful erythema, edema, or ulcers but eating/swallowing possible), 3 (painful erythema, edema, or ulcers requiring IV hydration), 4 (severe ulceration or requiring parenteral or enteral nutritional support or prophylactic intubation). Source: *Cancer* 2004;100(9 Suppl):1995.
Typically 48–72 h after starting cytotoxic cancer tx, a/w large, rapidly proliferating, tx-responsive tumors (esp acute leukemia, NHL, Burkitt)

**Approach:** Obtain ECG immediately (look for signs ↑ K), & put on cardiac monitor

**History**
- Diverse sx (need high index of suspicion prior to labs): Lethargy, edema, CHF, hematuria, cardiac dysrhythmia, sz, muscle cramps, tetany, syncope, sudden D
- Assess last chemo, but may be presenting signs of malignancy

**Evaluation**
- Serial Chem 10 (↓ Ca, ↑ PO₄, ↑ BUN/Cr, ↑ K), ↑ uric acid (draw on ice), ↑ LDH (marker of tumor proliferation), UA (urine pH), CBC w/ diff, VBG, Foley or close UOP measurement

**Treatment** *(NEJM 2011;364(19):1844)*
- Correct electrolyte d/o (↑ K, ↑ PO₄, ↓ Ca), except: use Ca tx in hyperK cautiously (may worsen CaPO₄ crystals in kidneys & worsen AKI) *(see also Chapter 9)*
- Maintain UOP > mL/kg/h: IVF ± loop diuretic PRN
- Reduce uric acid: allopurinol (prevents uric acid formation) or rasburicase (more effective; breaks down existing uric acid); give rasburicase in consultation w/ oncology
- Rasburicase (compared to allopurinol): lowers mean peak PO₄ & uric acid, less AKI, reduces need for HD; superior in mx trials
- Avoid NaHCO₃ for urine alkalinization: ↓ uric acid crystals, but also ↑ CaPO₄ crystals
- Consider only if no rasburicase available AND nl serum PO₄
- HD: If persistent ↑ K, severe acidosis, volume overload, uremia, severe ↑ PO₄ or ↓ Ca

**Disposition**
- Admit (floor vs. ICU, depending on severity)

---

**SUPERIOR VENA CAVA SYNDROME**

**Overview**
- **Definition:** Intrinsic/extrinsic SVC obstruction causing upstream high venous pressures
- **Etiology:** Malignancy (eg, lung, mediastinum) in 90%, thrombosis (eg, a/w implantable device), rarely TB & syphilitic aortitis (*NEJM* 2007;356(18):1862)

**History**
- Subacute onset (usually wks) of facial swelling ± laryngeal edema (cough, hoarseness, stridor), cerebral edema (HA, confusion), or ↓ venous return (hypotension)
- Severity of sx, acuity of worsening, & type of malignancy dictate intervention & urgency

**Physical Exam**
- Check for facial/oropharyngeal edema, JVD, abnml lung sounds, neuro deficits

**Evaluation**
- Chem 10, CBC, CXR, CT Chest (w/ IV contrast) ± CT Head (r/o ICH), bedside echo

**Treatment** (*NEJM* 2007;356(18):1862; *Crit Care Med* 2012;40:2212)
- Elevate head of bed (decrease ICP), intubate if impending airway obstruction
- If 2/2 malignancy: Chemo, XRT, Endovascular stenting (if no tissue dx)
  - Glucocorticoids commonly used to reduce swelling, but uncertain benefit
- If 2/2 thrombosis: anticoagulation (if concurrent HA: r/o ICH first), remove line if able
- If cerebral edema or airway obstruction: c/s Rad-Onc, Interventional Radiology from ED

**Disposition**
- Admit for expedited w/u (eg, bx) & tx

---

**Neutropenic Enterocolitis (Typhlitis)**

**Overview** (*World J Gastroentrol* 2017;23(1):42)
- **Definition:** Rare life-threatening dz of neutropenic pts, characterized by mucosal injury, bowel edema & distention, bacterial translocation;
cecum ± ileum & other colon

- **Approach:** Early IV access, IVF, abx, surgical consultation

**History**
- Abd pain (RLQ or diffuse), diarrhea, fever; ± N/V, distention, GIB
- Onset corresponds to WBC nadir (10–14 d after chemo)

**Physical Exam**
- Always assess for rebound tenderness; No DRE given neutropenia

**Evaluation**
- CBC, Chem 20, lactate, blood cx, CT A/P (bowl thick/dilated, pneumatosis), ±Upright abd XR if CT delay (can show intramural gas) or c/f perforation (subdiaphragmatic air)

**Treatment** *(World J Gastroentrol 2017;23(1):42)*
- Supportive: IVF, analgesia, antiemetics, NGT for bowel decompression
- Early abx as for neutropenic fever (see above); add flagyl if hx or c/f Cdif
- May need surgical intervention if e/o perforation or bowel necrosis

**Disposition**
- Admit, may need ICU

---

**Hyperviscosity Syndrome** *(Emerg Med Clin N Am 2014:32:495)*

**Overview**
- **Definition:** Rise in serum viscosity 2/2 proteins, causing low-flow & prolonged bleeding
- Most common in Waldenström macroglobulinemia (leading cause; can be presenting sx), multiple myeloma, acute leukemia (2/2 cellular proteins)
- **Approach:** Early IV access, IVF, abx, surgical consultation

**History**
- Classic triad: mucosal bleeding, visual deficits, focal neuro signs; can also see CHF, pulm edema, AKI, confusion

**Physical Exam**
- Assess mucosal bleeding, fundoscopic exam, neuro findings
Evaluation
- CBC w/ diff, Chem 20, lactate ± NCHCT (if neuro sx), BNP, Troponin, CXR

Treatment
- Supportive: IVF
- Consultation w/ oncology for emergent plasmapheresis or expedited chemotx

Disposition
- Admit, may need ICU

---

**SICKLE CELL DISEASE**

**Overview**

- **Pathophysiology:** Recessive β-globin mutation → structurally abnl HbS → deoxygenated form polymerizes → RBC sickles → hemolysis/microvascular occlusion → tissue ischemia

<table>
<thead>
<tr>
<th>Acute Presentations of SCD</th>
</tr>
</thead>
</table>
| Acute anemia | **Aplastic crisis:** reduced marrow production (eg, parvo B19) combined w/ short existing RBC half-life  
**Splenic sequestration:** sequestration of RBCs in spleen  
**Hyperhemolytic crisis:** 2/2 hemolysis |
| Vaso-occlusive crisis (VOC) | Can manifest as pain, tissue infarction (stroke, renal necrosis, aseptic necrosis, hepatic), priapism |
| Acute chest syndrome (ACS) | Resembles PNA; fever, dyspnea, hypoxia, CXR infiltrates; 13% will require mechanical vent; mortality up to 9%; 80% occur w/ VOC (Chest 2016;149(4):1082) |

- **Approach:** Initiate empiric IVF & analgesia early, can decompensate quickly; maintain high index of suspicion for infection from encapsulated organisms 2/2 functional asplenia

**History & Physical Exam**

- **HX:** Assess location, duration, & severity of pain; similarity to prior crises; infectious sx
- **PE:** Assess joint ROM in areas affected; respiratory status; neuro deficits; priapism
Evaluation

- CBC w/ diff (compare to baseline), Chem 20 (↑ bili in hemolysis), reticulocyte count (↑ in hemolysis or sequestration; ↓ in aplastic crisis), LDH (elevated), ABG (if hypoxic)
- Imaging as directed by sx: CXR (if CP or c/f ACS), x-ray/MRI (osteomyelitis or avascular necrosis), CTA chest (PE); CTA/MRI (stroke)

Treatment

<table>
<thead>
<tr>
<th>Acute tx in SCD (Chest 2016;149(4):1082)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aplastic crisis</td>
</tr>
<tr>
<td>Hyperhemolytic crisis</td>
</tr>
<tr>
<td>Splenic sequestration</td>
</tr>
<tr>
<td>VOC</td>
</tr>
<tr>
<td>Acute chest syndrome</td>
</tr>
<tr>
<td>Acute stroke</td>
</tr>
<tr>
<td>Other severe organ injury (eg, hepatic)</td>
</tr>
<tr>
<td>Sepsis (any cause)</td>
</tr>
</tbody>
</table>

Chronic


Disposition

- Home: If pain controlled, no e/o hemolysis; close hematology f/u
- Admit: Any acute cx as detailed above

ABNORMAL BLEEDING

Overview
Etiology & tx depend on nature of problem (plt count, plt fxn, clotting time, combo)

### Differential Dx for Abnl Bleeding

<table>
<thead>
<tr>
<th>1st Problem</th>
<th>Potential Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ plt count</td>
<td>ITP, HUS/TTP, DIC, HELLP syndrome (if pregnant), SLE, HIT, splenic sequestration (eg, NHL, myelofibrosis, cirrhosis), bone marrow failure (eg, aplastic anemia, heme malignancy), massive transfusion/dilution, chronic systemic dz</td>
</tr>
<tr>
<td>↓ plt fxn</td>
<td>vWD, meds (eg, ASA/NSAIDs, clopidogrel, GP IIb/IIA inhibitor), chronic systemic dz (eg, uremia, cirrhosis, leukemia/MDS)</td>
</tr>
<tr>
<td>↓ clotting cascade</td>
<td>DIC, meds (eg, coumadin, DOACs), factor deficiency (eg, hemophilia), chronic systemic dz (eg, malnutrition, cirrhosis), massive transfusion/dilution</td>
</tr>
</tbody>
</table>

### History & Physical Exam

- Bleeding syndromes can present in many organ systems, including several at once:

<table>
<thead>
<tr>
<th>Hx System</th>
<th>Manifestation</th>
<th>Hx System</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Gingival bleeding, epistaxis</td>
<td>GU</td>
<td>Hematuria, menorrhagia</td>
</tr>
<tr>
<td>CNS</td>
<td>ICH, epidural hematoma</td>
<td>MSK</td>
<td>Hematoma, hemarthrosis</td>
</tr>
<tr>
<td>Pulm</td>
<td>Hemoptysis</td>
<td>Skin</td>
<td>Petechiae, purpura, ecchymosis</td>
</tr>
<tr>
<td>GI</td>
<td>Hematemesis, melena, hematochezia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Physical exam may suggest sp bleeding problem:
  - Petechiae: ↓ plt count
  - Purpura: ↓ plt count, ↓ plt fxn, problem w/ blood vessels or connective tissue
  - Ecchymosis: ↓ clotting cascade, problem w/ connective tissue, trauma

### Evaluation

- CBC w/ diff, Chem 7, Coags; consider LFTs, peripheral blood smear, DIC panel (fibrinogen, D-dimer, LDH, haptoglobin), direct Coombs

---

**Immune Thrombocytopenia**
Overview

- **Definition:** Immune Ab-mediated destruction of PLTs (PLT count <100 k/μL); either 1° (idiopathic) or secondary (2/2 virus, meds, autoimmune dz, pregnancy, vaccine)
  - Common associated meds: quinine, antimicrobials (linezolid, rifampin, vancomycin, sulfa), anticonvulsants (phenytoin, VPA, carbamazepine), thiazides, H2-blockers, NSAIDs/tylenol, chemotx (*NEJM* 2007;357(6):580)
- Dx of exclusion: r/o TTP/HUS, HIT, hematologic d/o (eg, malignancy, MDS, HIV)

**History & Physical Exam**

- Acute/subacute petechiae ± e/o bleeding (see above; assess for ICH, GIB, etc.); always ask about new meds, recent infectious sx, pregnancy

**Evaluation** (*Blood* 2010;115(2):168)

- CBC w/ diff (↓ plt count, otw nml), peripheral smear (no schistocytes), Coags (nml), T+S
- HIV/HCV recommended on all pts to r/o alt cause, *H. pylori* can be associated as well
- If dx confirmed or highly suspected: obtain baseline IgG/A/M levels, direct antiglobulin

**Treatment** (*Blood* 2010;115(2):168; *NEJM* 2007;357(6):580)

- Adults: If asx, tx rarely indicated if PLTs >50k & no bleeding, trauma, or surgery
- Children: Manage mild cases expectantly, treat if PLTs <20k or active bleeding

<table>
<thead>
<tr>
<th>Tx for ITP</th>
<th>Adults</th>
<th>No bleeding, Plt &gt;50k</th>
<th>May manage expectantly; close f/u</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No bleeding, Plt &lt;50k</td>
<td>Prednisone (0.5–2 mg/kg/d until plts), OR dexamethasone (40 mg/d), OR IV methylprednisolone (1 g/kg/d) if severe</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IVIG (1 g/kg/d) × 1–2 d</td>
<td>Anti-D Ig (Rh+ pts only) (75 μg/kg/d)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bleeding, Plt &lt;50k</td>
<td>Combine tx above, transfuse</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Adults</th>
<th>No bleeding, Plt &lt;20k</th>
<th>Steroids: Prednisone 4 mg/kg/d PO × 4 d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Anti-Rh(D) Ig: 75 μg/kg IV × 2 d</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Children</th>
<th>No bleeding, Plt &lt;20k</th>
<th>Steroids: Prednisone 4 mg/kg/d PO × 4 d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Anti-Rh(D) Ig: 75 μg/kg IV × 2 d</td>
</tr>
</tbody>
</table>
IVIG: 0.8–1 g/kg IV × 1 dose

| Bleeding, Plt <20k | Combine tx above, transfuse |

- Transfuse platelets as per guidelines below (see Transfusion section)
- Steroids: 50–75% response, often by 3 wk
- IVIG: Equivalent but quicker remission compared to steroids; consult w/ hematology
- Anti-D Ig: As effective as IVIG, but shorter infusion, longer response; consult w/ hematology
- Second-line tx should be discussed w/ consultant: azathioprine, cyclosporine, cyclophosphamide, dapsone, mycophenolate, rituximab, splenectomy, others
- No role for plasmapheresis in tx of ITP

**Disposition**
- Home: If no active bleeding, PLTs >20k
- Admit: Any pt w/ PLTs <20k &/or active bleeding
- Long-term relapse is common (>70%) despite tx modality *(NEJM 2007;357(6):580)*

---

**Heparin-Induced Thrombocytopenia (HIT)**

**Overview**
- **Definition:** TCP (<150k) or 30–50% drop in plt count after starting heparin
  - More common w/ unfractionated heparin, but occurs as well w/ LMWH
- **Pathogenesis:** Heparin-dependent IgG binds PF4 → plt activation → ↑ thrombosis
  - Thrombosis causes sx in 50% pts: PE, DVT > limb ischemia, stroke > MI

**History & Evaluation**
- Can have sx of TCP (if plts very low), thrombosis (even w/ nl plt count), or be incidental discovery; very rarely causes bleeding
- Usual occurrence 5–10 d after starting heparin, more rapid onset if recent heparin exposure
- Anti-PF4 enzyme assays highly se, poorly sp (good NPV); combine lab testing w/ clinical probability (hi, med, low) based on: Δ plt count (>50%, 30–50%, <30%), timing (5–10 d, >10 d, <4 d), thrombotic cx, &
alternative causes (yes, possible, no)

**Treatment** *(NEJM 2006;355:809; NEJM 2001;344:1286)*

- STOP heparin + any device/flush that contains heparin (even if thrombotic cx)
- Initiate nonheparin a/c (even if no thrombosis): argatroban (first-line), bivalirudin, lepirudin
  - Avoid vitamin-K antagonists, as will decrease protein C → ↑ thrombosis
- Avoid PLT transfusions unless bleeding or high risk of bleeding
- Recurrence w/ future heparin may be low if neg. for PF4 Ab >100 d after Dx

**Disposition**

- Admit for monitoring & intravenous anticoagulation

---

**Hemolytic-Uremic Syndrome (HUS) & Thrombotic Thrombocytopenic Purpura (TTP)** *(NEJM 2006;354(18):1927; NEJM 2014;371(7):654)*

**Overview**

- **Definition:** Systemic (TTP) or intrarenal (HUS) microvascular occlusive d/o 2/2 PLT aggregation → MAHA + TCP
- **Pathogenesis:** TTP a/w ADAMTS13 dysfxn (ADAMTS13 normally cleaves vWF, resultant inability to cleave vWF → plt activation → microthrombi); HUS a/w *E. coli* O157:H7

**History & Physical Exam**

- **TTP:** Acute/subacute, can be subtle & non-sp (classic pentad rare); adults > children
  - MAHA: Vague sx (abd pain, nausea, weakness) 2/2 diffuse microvascular thrombi
  - TCP: e/o bleeding, skin findings (petechial, purpura)
  - Neuro sx: AMS, stroke, szs; neuro sx present only 50%
  - AKI: Can be mild
  - Fever: Uncommon, low-grade when present); no other cause
- **HUS:** Acute-onset, bloody diarrhea & abd pain, f/b TCP & renal failure
  - Classic triad: MAHA + TCP + Renal failure
  - Always ask about triggers: HUS (contaminated food), TTP (meds,
systemic dz, HSCT)

**Evaluation**
- CBC w/diff (↓ Hct, ↓ Plt), Chem 20 (↑ Cr, ↑ LFTs), peripheral smear (schistocytes), coags (nml), ↑ LDH, ↓ haptoglobin, fibrinogen, D-dimer, UA

**Treatment** *(NEJM 2006;354(18):1927; NEJM 2014;371(7):654)*
- Consult hematology, consider renal consult early
- TTP: Plasma exchange (↑ survival @ 6 mo, 78%), FFP (if delay to plasma exchange), Steroids (prednisone 1–2 mg/kg/d, methylprednisolone 1 g/d, data on efficacy limited)
- HUS: Supportive; pts commonly need dialysis
- Do not give PLTs → ↑ microvascular thrombosis

**Disposition**
- Admit, may require ICU
- Risk of relapse is low in acquired TTP, but can occur even years after initial episode
- HUS in kids often resolves w/o long-term renal dz (but >45% mortality in adults)

---

**DISSEMINATED INTRAVASCULAR COAGULATION (DIC)**

**Overview**
- **Definition:** Acquired life-threatening consumptive coagulopathy a/w diverse dz
- **Pathogenesis:** Widespread activation of coagulation → thrombosis of small/midsized vessels → organ dysfxn, ↓ PLTs/coagulation factors → bleeding & thromboembolism

<table>
<thead>
<tr>
<th>Causes of DIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer (top cause)</td>
</tr>
<tr>
<td>Infectious dz</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Obstetrics</td>
</tr>
<tr>
<td>Immunologic</td>
</tr>
</tbody>
</table>
**History, Physical Exam, Evaluation**

- Underlying systemic dz is necessary for dx (see above)
- Assess for s/sx of bleeding, end-organ damage
- CBC w/ diff (↓ PLTs [usually <100]), ↑ PT, ↑ PTT, ↑ D-dimer, ↓ fibrinogen, ↑ LDH, ↑ Fibrin degradation products, e/o end-organ damage (↑ lactate, ↑ Cr); imaging PRN by sx
- Different scoring systems used to help in dx of DIC (Thromb Haemost 2011;105(1):40)

**Treatment**

- Treatment of underlying disorder is mainstay of tx
- Transfuse if bleeding, risk of bleeding, or need for procedure: PLTs (if bleeding + plt <50k, or plt <20k), FFP (if INR >1.5; beware large doses may be required [15–30 mL/kg]), cryoprecipitate (if fibrinogen <1.5 g/dL despite FFP), prothrombin complex concentrate (if bleeding & FFP delayed/not possible [eg, volume])
- Heparin/LMWH if thromboembolism predominates (LMWH may be superior to UH)

**Disposition**

- Admit to ICU

---

**Von Willebrand Disease (vWD)**

**Overview**

- **Definition:** Most common inherited bleeding d/o caused by ↓ vWF (↓ quantity [type 1, 3] or fxn [type 2]); vWF carries factor VIII & enables plt adhesion/aggregation; rarely acquired
- **Etiology:** Precursor vWF cleaved by ADAMTS13 (see TTP above) → circulating vWF activated by vascular damage → vWF binds collagen → plt aggregation
  - ↓ vWF leads to ↓ plt aggregation & ↑ factor VIII degradation

**History & Physical Exam**

- Mucocutaneous bleeding, menorrhagia, bruising, epistaxis, hemarthrosis, hematomas
- 5–20% of pts w/ menorrhagia will have vWD (sometimes requiring TAH)
Most pts aware of their hx (60–80% will have bleeding after surgery)

**Evaluation**
- CBC w/ diff, T&S, coags (↑ PTT); ↓ vWF Ag, ↓ vWF activity, ↓ factor VIII activity

**Treatment**
- Desmopressin (DDAVP): Most useful in Type 1 vWD, where sufficient & functional vWF exists in noncirculating form; DDAVP causes endothelial release of vWF & ↑ circulating vWF levels (no chg in overall vWF); efficacy variable; dose 0.3 μg/kg IV
- vWF replacement: most useful in Type 2 or 3, where endogenous vWF either dysfxnal or absent; options in include cryoprecipitate (requires up to 8–12 bags), plasma-derived vWF/factor VIII concentrate (Humate-P), or recombinant vWF + factor VIII
- Life-threatening bleeds: Important to increase levels of both vWF & factor VIII
- Adjunct tx: Antifibrinolytic amino acids (eg, TXA); high-quality data lacking

**Disposition**
- If severe or significant bleeding, admx; if d/c, f/u w/ hematology

---

**Hemophilia A/B**


**Overview**
- Definition: X-linked (males) d/o 2/2 ↓ factor VIII (hemophilia A) or factor IX (hemophilia B)

<table>
<thead>
<tr>
<th>Hemophilia Dz Severity</th>
<th>Factor Concentration (% of nml)</th>
<th>Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>5–40 IU/dL</td>
<td>Spontaneous IM bleeding</td>
</tr>
<tr>
<td>Moderate</td>
<td>1–5 IU/dL</td>
<td>Severe bleeding w/ minor trauma</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;1 IU/dL</td>
<td>Severe bleeding w/ major trauma</td>
</tr>
</tbody>
</table>

**History & Physical Exam**
- Bleeding (GI, GU, mucosa), hematoma, hemarthroses, bruising
Evaluation
• CBC, nml INR, ↑ PTT, T&S, Imaging PRN

Treatment
• Minor bleeds: desmopressin/DDAVP (0.3 μg/kg IV, 150 μg IN kids/300 μg IN adults) increases factor VIII concentration 3–5 × 2/2 release of vWF; tranexamic acid (25–50 mg/kg/d); DDAVP for hemophilia A, TXA for hemophilia A/B
• Moderate–Severe bleeds: factor VIII/IX concentrate based on extent of deficiency; refer to product-sp dosing instructions (Haemophilia 2013;19(1):e1)
  • If severe (eg, ICH), give factor even prior to diagnostic testing
  • If pts have developed inhibitors to factor VIII, consider use of factor VIIa or prothrombin complex (bypass factor VIII in clotting cascade)

TRANSFUSIONS

See Chapter 18 for transfusions in trauma.

Overview
• Approach: Obtain type/screen for any pt suspected of needing transfusion

<table>
<thead>
<tr>
<th>Transfusion Product Sp</th>
<th>Use for</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irradiated</td>
<td>pts w/ cellular immunosuppression (eg, neonates, HSCT, congenital immune def); destroys donor T-cells (↓ GVHD)</td>
</tr>
<tr>
<td>Leuko-reduced</td>
<td>pts requiring mx transfusions; removes WBCs (↓ infxn [eg, CMV], ↓ rejection against donor product)</td>
</tr>
<tr>
<td>Washed</td>
<td>pts w/ hx of transfusion allergic rxns; removes plasma components (↓ allergic rxns); very time intensive</td>
</tr>
<tr>
<td>Pheresis</td>
<td>Single-donor (vs. pooled-donor); refers to platelet transfusion</td>
</tr>
</tbody>
</table>

• When to give: restrictive strategies ↓ adverse events, mortality (Cochrane 2012;(4):CD002042)
  • All pts: Hgb ≤7, Hgb >7 + acute, ongoing, or significant sx
  • Cardiovascular dz: Hgb ≤8, Hgb >8 + acute, ongoing, or significant sx
• What to give: always obtain T&S in anyone who may need transfusion
  • Emergent transfusion: O neg. blood to females, O positive to males
  • In massive transfusion (expected >10 U PRBC/d), give FFP & PLTs as
How much to give:
- Neonates: ↑ Hgb by 3 g/dL for 10–15 mL/kg of PRBCs
- Adult: ↑ Hgb by 1 g/dL or Hct by 3%, for each 1 U PRBCs

What to monitor:
- Electrolytes: esp if large volume PRBC (↑ K, ↓ Ca)
- O₂: Pts w/ CHF may require concurrent diuresis

Platelet Transfusion

When to give:
- PLT < 10K: regardless of s/sx of bleeding or comorbidities
- PLT < 20K: non-bleeding but high risk if deteriorates (eg, chronically ill, high-risk onc)
- PLT < 50K: active bleeding or need for invasive procedure
- PLT < 100K: need for ophthalmologic or neurosurgical procedure

How much to give: ↑ PLTs by 5000–10000 for each 1 U PLTs

Fresh-Frozen Plasma Transfusions (Transfusion 2010;50(6):1227)

When to give: Mass transfusion (see Trauma section); reversal of warfarin; coagulopathy (eg, DIC); TTP/HUS (if delay to plasmapheresis); replacement of factor deficiencies

How much to give: 10–20 mL/kg will ↑ coagulation factors by 20–30% (1U FFP = 200 mL); consider giving concurrent diuretic if e/o or known hx of CHF

Cryoprecipitate Transfusions

How much to give: fibrinogen level <1 g/dL; factor XIII deficiency; hemophilia or vWD

How much to give: 1 U/5–10 kg body weight to maintain fibrinogen >1 g/L

ANTICOAGULATION REVERSAL

Approach
- Hold further doses of anticoagulant
- Supportive care: Gastric lavage or activated charcoal (if overdose w/i 1 h), apply pressure to site of bleeding if possible, transfuse (as indicated)
- Agent-sp reversal: If significant/life-threatening bleeding (eg, ICH,
hemoptysis, GIB) or need for emergent procedure/surgery

<table>
<thead>
<tr>
<th>Reversal of Common Anticoagulants</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antiplatelet agent</strong></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
| | | Consider plt transfusion (though new data suggests may cause harm in case of ICH)\(^1,\(^2\)

| **Heparin** | Protamine (1 mg/90–100 U heparin in last 2–3 h; max 50 mg)\(^2\) |
| **LMWH** | Protamine may be partially (60–80%) effective (dosing above)\(^2\) |
| | Andexanet may be effective (under study)\(^3\) |

| **Warfarin\(^4\)** | INR <5 | Decrease or omit single warfarin dose F/U for INR check w/i 48–72 h |
| | No bleeding | |
| | INR 5–9 | Omit 1–2 warfarin doses |
| | No bleeding | Vitamin K 2.5 mg PO × 1 if risk for bleeding F/U for INR check w/i 24–48 h |

| | INR >9 | Hold warfarin (until F/U INR recheck) |
| | No bleeding | Vitamin K 5 mg PO × 1 F/U for INR check w/i 12–24 h |

| | INR >1.5 | Hold warfarin |
| | Serious bleed | Vitamin K 5–10 mg IV (risk of anaphylactoid rxn) |
| | | Prothrombin clotting complex (25–50 IU/kg) |
| | | FFP (10–20 mL/kg) only if PCC unavailable |

| **DOAC** | **Dabigatran** | Idarucizumab reverses bleeding w/i minutes\(^5\) 4-factor PCC may have effectiveness |
| | | |
| | **Rivaroxaban** | 4-factor PCC may have effectiveness Andexanet may be effective (under study)\(^6\) |
| | **Apixaban** | 4-factor PCC may have effectiveness Andexanet may be effective (under study)\(^6\) |

| **NOTE:** At time of publishing, several additional DOACs & reversal agents are under investigation. Data is limited. |

---


**Pearls**

- Irreversible antiplatelet agents inhibit life of platelet (7–10 d)
- Vitamin K takes 6 (IV) to 24 (PO) hours to reverse warfarin; IV may cause anaphylactoid rxn in rare instances (push slowly over 30 min)
4F-PCC superior to FFP, may have fewer adverse events ([Lancet](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(15)00482-6/fulltext) 2015;385(9982):2077)

- Adverse events: w/ 4F-PCC mostly 2/2 thrombosis; w/ FFP mostly 2/2 volume

### Transfusion Complications

#### Approach

- Always obtain consent if possible before giving a transfusion
- If e/o rxn: Stop transfusion, check bag, label, & send remaining products to blood bank
- May be possible to resume transfusion in mild allergic rxn only (see below)
- If febrile, obtain CBC, smear, direct Coombs, UA, gram stain, BCx (pt & product)

<table>
<thead>
<tr>
<th>Common or Critical Transfusion rxns</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rxn</td>
</tr>
<tr>
<td>Febrile (nonhemolytic)</td>
</tr>
<tr>
<td>1:100</td>
</tr>
<tr>
<td>Incidence*</td>
</tr>
<tr>
<td>Recipient Ab against donor cytokines; dx of exclusion</td>
</tr>
<tr>
<td><strong>HX:</strong> Fever, discomfort ± transient HTN</td>
</tr>
<tr>
<td><strong>TX:</strong> Antipyretics, monitor to r/o infxn (septic trans fus) or hemolysis (acute hemolytic rxn)</td>
</tr>
<tr>
<td>Allergic Anaphylactic</td>
</tr>
<tr>
<td>1:1000</td>
</tr>
<tr>
<td>1:10,000</td>
</tr>
<tr>
<td>Histamine-mediated; more common w/ platelet transfusion</td>
</tr>
<tr>
<td><strong>DX:</strong> Pruritus, urticaria ± angioedema, e/o anaphylaxis</td>
</tr>
<tr>
<td><strong>TX:</strong> H1B; if anaphylaxis then add H2B, glucocorticoids, IM epi; can restart transfusion if sx were local only &amp; full resolution (stop if recurs)</td>
</tr>
<tr>
<td><strong>Pearls:</strong> If hx of transfusion allergic rxn, consider washed products &amp; H1B prem ed; no role for steroid prem ed</td>
</tr>
<tr>
<td>Febrile (acute hemolytic)</td>
</tr>
<tr>
<td>1:10,000</td>
</tr>
<tr>
<td>Blood product error (eg, ABO incompatibility, incorrect preparation): can be immune or nonimmune</td>
</tr>
<tr>
<td><strong>DX:</strong> Fever (first sign) → flank pain, AKI, hemoglobinuria, anemia, DIC, shock w/i 24 h</td>
</tr>
<tr>
<td><strong>TX:</strong> If fever, monitor for other sx; IVF/diuretics for UOP</td>
</tr>
</tbody>
</table>
| **Pearls:** Differs from delayed hemolytic rxn (1:2.5K [1:10 in SCD]; 2/2 Abs against non-ABO groups;
<table>
<thead>
<tr>
<th>Condition</th>
<th>Incidence</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRACO (transfusion-associated circulatory overload)</td>
<td>1:10,000</td>
<td>Excess volume/rate of transfusion, ↑ risk if hx of CHF, CKD, large volume or rapid rate (eg, hemorrhage)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>DX:</strong> SOB, ↑ BNP, ↑ CVP, pulm edema w/i 6 h of transfusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>TX:</strong> O₂, diuresis, NIPPV prn, restart transfusion slowly</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Pearl:</strong> Give diuresis w/t transfusion high-risk for TRACO</td>
</tr>
<tr>
<td>TRALI (transfusion-related lung injury)</td>
<td>1:100,000</td>
<td>Proposed etiology: donor Abs bind recipient WBCs → pool in pulm capillaries → ↑ permeability → edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>DX:</strong> SOB, ↑ O₂, CXR w/ b/l infiltrates; fever/hypothermia, hypotension/HTN; w/i 72 h of transfusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>TX:</strong> Supportive (O₂, low tidal-volume vent if intubated)</td>
</tr>
<tr>
<td>Septic</td>
<td>1:100,000</td>
<td>Infected blood product, most common w/ platelets</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>DX:</strong> Fever, rigors, hypotension; +BCx (pt &amp; product)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>TX:</strong> Broad-spectrum abx (inc antipseudomonal)</td>
</tr>
<tr>
<td>Viral infection</td>
<td>Variable</td>
<td>HBV (1:250K), HCV (1:1.6M), HIV (1:1.8M)</td>
</tr>
</tbody>
</table>


**EMERGENCIES IN THE TRANSPLANT PATIENT**

**Infectious Complications**
- Incidence is 25–80% in 1st year after transplant
- Can be subtle: Immunosuppression can diminish classic sx (eg, fever, localizing sx), radiographic signs, or serology results; maintain high index of suspicion
- Timing of infection after tpx a/w type of infection
  - Nosocomial: Asp PNA, wound infxn, UTI (Foley), donor infxn, line infxn, *C, difficile*
  - Opportunistic: *PJP, Histoplasma, Coccidioides, Cryptococcus*, HBV, HCV, BK polyomavirus, CMV, TB, EBV; assess if pt is on ppx (eg, PJP, CMV, fungal ppx)
  - Community: PNA, Influenza, EBV, RSV, Legionella, UTI
- If septic, strongly consider adjunct stress-dose steroids

<table>
<thead>
<tr>
<th>Infections in Posttransplant pts</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>0–1 mo posttransplant</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>1–6 mo posttransplant</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>&gt;6 mo posttransplant</strong></td>
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<td></td>
</tr>
</tbody>
</table>

**Graft Rejection**
- Recipient immune-mediated rxn against transplanted organ (esp microcirculation)
- Frequency & sx of rejection vary by organ txp type
  - 20% kidney; 64% (acute)/23% (late) liver txp, 30% cardiac (acute), 30% lung (first yr)
- Essential to involve transplant team if considering rejection
- TX: High-dose intravenous steroids; may require additional tx

<table>
<thead>
<tr>
<th>Signs &amp; sx of Transplant Rejection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Renal (20%)</strong></td>
</tr>
<tr>
<td>SX: Often asx; fever, malaise, oliguria, graft pain; HTN</td>
</tr>
<tr>
<td>DX: Labs (↑ BUN/Cr, abnml lytes); urine lytes (r/o other dx); Renal US w/ ↑ resistive indices</td>
</tr>
<tr>
<td><strong>Liver (60% &lt;6 mo; 25% late)</strong></td>
</tr>
<tr>
<td>SX: Fever, malaise, abd pain, organomegaly, ascites</td>
</tr>
<tr>
<td>DX: Labs (↑ LFTs); RUQUS (r/o other dx [eg, thrombosis])</td>
</tr>
<tr>
<td><strong>Cardiac (30% &lt;6 mo)</strong></td>
</tr>
<tr>
<td>SX: SOB, orthopnea, palpitations, near-syncope, GI sx if R heart predominance (RUQ pain, nausea)</td>
</tr>
<tr>
<td>DX: Labs (↑ Troponin, BNP), ECG w/ ST/Tw chgs, BSUS w/ systolic/diastolic dysfuxn; CXR w/ edema (if L heart)</td>
</tr>
<tr>
<td><strong>Lung (30% &lt;1 yr)</strong></td>
</tr>
<tr>
<td>SX: SOB, cough, lung exam variable</td>
</tr>
<tr>
<td>DX: Labs (↑ eos), CXR can be nml, Chest CT</td>
</tr>
</tbody>
</table>

**Organ-specific Complications**

**Renal Transplant**
- UTI (most common): always cx; consider strongly dual-abx & admx
- Arterial stenosis (10%): HTN, ↓ UOP, edema; U/S w/ flow limitation; tx w/ stent
- Venous thrombosis (4%): Graft pain/erythema, ↓ UOP, N/V; U/S (may need contrast CT); often results in graft failure & need for graft
replacement

- Ureteral obstruction (3–6%): ↓ UOP, edema; U/S w/ hydronephrosis; if no correction w/ Foley, may need percutaneous intervention for urinary drainage
- Urinary leakage (2–5%): ↓ UOP, perineal leakage; U/S w/ peritpx fluid collxn; urology c/s
- Lymphocele (5–15%): abd swelling; US w/ hydrocele (CT for definitive dx)

Liver Transplant

- Hepatic artery thrombosis (4–12%): Doppler U/S 90% Se; mortality 80% w/o tx
- Hepatic artery stenosis (14%): Doppler U/S 70% Se; may require angiography for dx
- Pseudoaneurysm: Can cause hemobilia, hemoperitoneum, GIB; dx w/ U/S or CT
- Biliary strictures: May be asx 2/2 denervation; U/S (Se 66%), MRCP (Se 95%); tx stent
- Biliary leaks (2–25%) & bilomas: tx w/ stenting over leak, drain & abx (if biloma)

Cardiac Transplant

- Allograft vasculopathy (30–70%): Chronic & progressive; rapid atherosclerosis in tx organ; may have s/sx of ischemia, or be asx 2/2 denervation (inc sudden cardiac arrest)
- Bradycardia: 2/2 sinus or AVN trauma; refractory to atropine 2/2 loss of vagal innervation
- Tachyarrhythmias: BB > CCB for Afib/flutter (2/2 med interaction); half-dose adenosine 2/2 sensitivity of transplanted heart

Lung Transplant

- Airway anastomotic stenosis, tracheobronchomalacia (increase in granulation tissue), necrosis; SOB/stridor; CT may help, but bronch is definitive dx; may need stent
- Fistula: Depending on tract, may present w/ PTX, crepitus, hemoptysis
- Pulm artery stenosis: Early & late after tx; SOB, ↓ O₂, LE edema, ↓ BP; dx w/ CTA chest
- Pulm vein thrombosis: Early after tx; SOB, ↓ O₂, LE edema, ↓ BP; dx w/ CTA chest
- Phrenic nerve injury (3–9% lung tx; 40% heart–lung tx): SOB; CXR ↑
hemidiaphragm
LEG PAIN AND SWELLING

Approach
- Careful hx: Anatomic distribution, unilateral vs. bilateral, acute vs. chronic, a/w erythema or dermatologic findings; hx of trauma
- Assess for paresthesia, hyperesthesia, or neuropathy
- Complete neurologic & vascular exam, assess for motor weakness

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>CHF (edema, venous stasis)</td>
</tr>
<tr>
<td>Vascular</td>
<td>DVT (see Ch. 1), PVD, arterial occlusion, vascular ulcers, thrombophlebitis</td>
</tr>
<tr>
<td>Infection</td>
<td>Osteomyelitis, necrotizing fasciitis, septic joint, cellulitis/abscess (see Ch. 4)</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Fracture, sprain, dislocation, hematoma, compartment syndrome; spinal stenosis (pseudoclaudication)</td>
</tr>
<tr>
<td>FEN/GU</td>
<td>Electrolyte abnltys, glomerulonephritis, nephritic syndrome</td>
</tr>
<tr>
<td>OB/GYN</td>
<td>Pregnancy, HELLP syndrome</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Guillain–Barré, peripheral neuropathy</td>
</tr>
<tr>
<td>Environmental</td>
<td>Heat edema</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>Sarcoma, SVC syndrome</td>
</tr>
</tbody>
</table>

PERIPHERAL VASCULAR DISEASE

Claudication

History
- Ischemic muscle pain reproducible w/ exertion, improves w/ rest
- Pts often place legs in dependent position to improve flow
- 1–2% have chronic critical limb ischemia: Pain at rest, nonhealing ulcers, dry gangrene
Findings: May have nl exam at rest w/ or w/o ↓ peripheral pulses; shiny, smooth skin

Evaluation
- ABI <0.9 is diagnostic of PVD (sens & spec)
- Careful pulse exam, w/ Doppler if difficult to palpate
- Look for signs of critical ischemia (rest pain, nonhealing ulcers)

Management: If concern for critical ischemia or acute dz, consult vascular surgery

Disposition
- Admit acute dz
- D/C home if chronic w/ vascular surgery f/u, strict return instructions

Acute Extremity Arterial Occlusion

History
- Known PVD +/- RFs (HTN, tobacco, known CAD, AF)
- Abrupt onset of pain w/ distal paresthesias
- Late (concerning findings): poikilothermia, pallor, paresthesia, pulselessness

Findings
- Cold, mottled extremity, ↓ pulse, motor weakness, ± bruit
- Tenderness to palpation out of proportion of exam or ↓ sensation

Evaluation
- Bedside Doppler of all pulses, including unaffected extremities; ABI
- Labs: CBC, BMP, baseline coags, ± lactate
- U/S can demonstrate level of occlusion
- CTA or angiography
- ECG for arrhythmia, may need echo to look for embolic source

Treatment
- Immediate vascular surgery consultation for possible embolectomy
- Anticoagulation (discuss w/ vascular): Heparin 18 U/kg/h IV w/o bolus

Disposition
- Transfer to facility w/ vascular surgery capability if none available

Pearl
- Ischemic tissue D starts by 4 h; sooner in pts w/ chronic arterial insufficiency
Measurement of Ankle–Brachial Indices (ABIs)

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<tr>
<td>1</td>
<td>W/ pt lying supine, measure SBP at ankle &amp; ipsilateral wrist. Place cuff over biceps to measure wrist SBP &amp; over calf to measure ankle SBP. Place Doppler U/S over radial pulse for wrist measurement &amp; over posttibialis or DP for ankle measurement. Inflate cuff until Doppler pulse no longer heard, record this pressure.</td>
</tr>
</tbody>
</table>

2 | Divide ankle SBP by wrist SBP. nl ABI = 1, <0.9 defines PVD. ABI < 1 indicates lower-extremity diminished flow. ABI > 1 indicates upper-extremity diminished flow. |

**TRAUMA**

**Compartment Syndrome**

**History**
- Can occur in any closed fascial space, most commonly in distal lower extremity (calf)
- Hx of trauma (esp crush), burns, rhabdomyolysis, tight cast/dressing, hemorrhage (anticoagulants, coagulopathy), postischemic swelling, snakebites, IVDU

**Findings**
- Pain out of proportion to exam, pain w/ passive stretch of muscles that run through compartment (see the table below), paresthesias, pallor of the extremity, taut or rigid compartment. LATE: Decreased pulse, sensory/motor deficits.

**Evaluation**
- Measure compartment pressures: nl <8 mmHg; emergent fasciotomy if >30 mmHg
- Stryker instrument: Enter each compartment perpendicular to the skin
- A-line manometer: Attach 18G needle to A-line manometer; check that the compartment pressure being measured is at the same height as the manometer transducer

**Treatment**
- Immediate orthopedic/surgical consult for fasciotomy

**Disposition**
- Admit to ortho for serial manometry & neurovascular checks if compartment pressures <30 mmHg but evolving compartment
syndrome suspected

**Pearls**

- Nl compartment pressure does *not r/o* compartment syndrome; clinical Dx
- 6% incidence w/ open tibia fx; 1% in closed tibia fx; 30% w/ arterial injury; 14% w/ venous

<table>
<thead>
<tr>
<th>Lower-Extremity Compartments &amp; Associated Muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep posterior</td>
</tr>
<tr>
<td>Superficial posterior</td>
</tr>
<tr>
<td>Lateral</td>
</tr>
<tr>
<td>Anterior</td>
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</tbody>
</table>

**LOWER BACK PAIN**

**Approach**

- Careful hx: Anatomic distribution, unilateral vs. bilateral, acute vs. chronic, fever, abd pain, groin pain, syncope hx of trauma; worse at rest or at night; incontinence?

<table>
<thead>
<tr>
<th>Historical Red Flags for Back Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>For fracture</td>
</tr>
<tr>
<td>For malignancy</td>
</tr>
<tr>
<td>For infection</td>
</tr>
<tr>
<td>For aortic/vascular</td>
</tr>
</tbody>
</table>

- Physical exam w/ thorough neurologic exam, straight leg raise, pulses, rectal tone, gait
- Always check urine pregnancy test in females of childbearing age
- X-rays not routinely indicated: Use for red flags above, abnl exam, point tenderness
- Most require only analgesia & f/u but always consider life- & limb-threatening conditions
<table>
<thead>
<tr>
<th>Differential Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI (see Ch. 3)</td>
<td>Abd aneurysm/dissection, pancreatitis, cholecystitis, ulcer (±perforation)</td>
</tr>
<tr>
<td>Trauma</td>
<td>Acute lumbosacral strain, vertebral compression fracture, retroperitoneal bleed (minor/no trauma but on anticoagulant)</td>
</tr>
<tr>
<td>Infectious</td>
<td>Spinal epidural abscess, discitis, osteomyelitis, pyelonephritis/perinephric abscess</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Cauda equina syndrome, herniated disc, spinal stenosis</td>
</tr>
<tr>
<td>Rheumatologic</td>
<td>Rheumatoid arthritis, ankylosing spondylitis, OA</td>
</tr>
<tr>
<td>FEN/GU</td>
<td>Nephrolithiasis</td>
</tr>
<tr>
<td>Vascular</td>
<td>Spinal hematoma/dissection</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>Malignancy (multiple myeloma in elderly), bony metastasis</td>
</tr>
</tbody>
</table>

**TRAUMA**

**Acute Lumbosacral Strain**

**History**
- Usually hx of precipitating event: twisting, lifting, new workout.
- Acute/subacute onset
- Should have no fever or radicular sx

**Findings:** Paravertebral muscle spasm & tenderness, nl neuro exam

**Evaluation:** No indication for imaging acutely

**Treatment**
- NSAIDs; if severe, short course opioids or BZD; early activity (no bed rest!)
- Muscle relaxants of no proven value, many side effects (anticholinergic, dependence)

**Disposition:** D/C home w/ PCP f/u, strict return instructions

**Pearl:** Lumbar strain is the #1 cause of LBP in ED but a dx of exclusion

**Vertebral Compression Fracture**

**History:** Acute-onset LBP usually in elderly pts w/ osteopenia, smoking, on steroids

**Findings:** Focal tender area on spine, usually no neuro findings
**Evaluation:** Plain film of affected thoracic, lumbar, or sacral spine

**Treatment**
- Usually stable fractures; analgesia ± brace for comfort
- Consult ortho or spine for >50% compression or multiple fractures

**Disposition:** Admit for intractable pain, any neuro findings, >50% compression, multiple fractures

**Pearl:** Look for neoplastic cause if no other RFs or hx, esp in elderly

---

**NEUROLOGIC**

**Cauda Equina Syndrome**

**Definition:** Large central disk herniation of distal spinal cord – *neurosurgical emergency*

**History**
- Severe LBP shooting down 1 or both legs & neuro sxs: Saddle paresthesias, urinary retention w/ overflow incontinence, loss of bowel control or sexual Dysfxn; pts w/ recent trauma or cancer w/ possible mets

**Findings:** ↓ rectal tone, urinary retention, saddle anesthesia, areflexia, weakness

**Evaluation**
- MRI is imaging test of choice
- Postvoid residual is the most sens initial finding

**Management:** Emergent neurosurgery consult, admit

**Lumbar Spinal Stenosis**

**Definition:** Narrowing of lumbar spinal canal from degeneration, facet arthritis, or subluxation

**History:** 40+ y/o, bilateral low back pain, pseudoclaudication (pain w/ walking), age >40, improves w/ rest & flexion of back (walk hunched over to keep back flexed)

**Findings:** nl exam, nl SLR, pain w/ back extension

**Evaluation:** Emergent imaging not needed if nl neuro exam; CT, MRI are diagnostic

**Treatment:** Pain mgmt w/ NSAIDs; hip flexor & abd exercises; surgery if severe

**Disposition:** Close f/u w/ PCP, spine
Herniated Disc

History
- 30–40 y/o, hx of waxing/waning back pain shooting down leg (past the knee) ± paresthesias
- Exacerbated by leaning forward, coughing, sneezing, & straining (stretches nerve root)

Findings
- See table below (L4–5 is most common)
- SLR test correlates w/ nerve root irritation only if reproduced sx extend below knee; Ipsilateral is sens, contralateral is spec.

Management
- Neuro intact: Analgesia, DC home. MRI or CT myelogram if no improvement in 4–6 wk.
- Neuro deficits (or acute traumatic herniation): MRI to eval for cord involvement

Disposition: D/C if no cord findings; o/w needs spine consultation

Pearl: Sciatica is lumbar disc herniation impinging on sciatic nerve

<table>
<thead>
<tr>
<th>Lumbar Nerve Root Compression</th>
</tr>
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<tbody>
<tr>
<td>Root</td>
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<tr>
<td>------</td>
</tr>
<tr>
<td>L4</td>
</tr>
<tr>
<td>L5</td>
</tr>
<tr>
<td>S1</td>
</tr>
<tr>
<td>S2–S4</td>
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</tbody>
</table>

Infectious

Spinal Epidural Abscess

History
- Classic triad of fever, local spine tenderness, extremity neurologic deficit
- High-risk population: IV drug abusers, immunocompromised, recent instrumentation, DM
Findings
- Classic sequence: Back pain → root pain/radiculopathy → motor weakness, sensory Δ, bowel/bladder dysfxn → paralysis

Evaluation: MRI w/ IV contrast is test of choice

Treatment
- Cover *Staph, Strep*, gram-neg. organisms: (nafcillin 2 g OR oxacillin) & (ceftiraxone 2 g OR ciprofloxacin) ± vancomycin, + antipseudomonal abx if instrumentation hx
- Spine surgery consultation; ±steroids; may want biopsy prior to abx

Disposition: Admit, usually to spine surgery; operative washout

Pearl: Avoid LP to prevent introduction of organisms into CSF unless meningitis highly suspected

---

**NEOPLASTIC**

Bony Metastasis

History: >50 y/o, 1 mo of sx, weight loss. Commonly breast, lung, kidney, prostate, thyroid

Findings: Tenderness of lumbar spine to palpation

Evaluation
- Plain film. CT/MRI/bone scan if plain film not definitive
- MRI & spine/oncology consultation if cord syndrome or findings

Treatment
- Pain control, Oncology referral
- If cord compressed, administer dexamethasone 10 mg IV or methylprednisolone 30 mg/kg IV, immediate consult

Disposition: Tx per spine surgery; possible operative decompression

Pearls
- 1° malignancy (esp multiple myeloma) should also be considered, esp in elderly
- Many bony mets missed on x-rays/CT; review films w/ radiologist specifically

---

**JOINT PAIN**
**Approach**

- Careful hx; anatomic distribution, single vs. multiple joints, acute vs. chronic, a/w fevers, skin Δ; hx of trauma
- Eval for systemic sxs in conjunction w/ chief complaint of joint pain
- If considering septic arthritis, evaluate need for arthrocentesis

<table>
<thead>
<tr>
<th>Differential</th>
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<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td>Rheumatologic</td>
</tr>
<tr>
<td>Musculoskeletal</td>
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<table>
<thead>
<tr>
<th>Etiology of Common Regional Joint Pains</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Region</strong></td>
</tr>
</tbody>
</table>
| Shoulder | Rotator cuff injury | Inflammation or tear of rotator cuff tendons from direct trauma or overuse
Pain in deltoid area of shoulder, worse w/ moving arm overhead or w/ direct pressure (sleeping)
Tenderness to palpation
“Empty Can Test”: Pain & weakness w/ resisted abduction of arm elevated at 90°, adducted forward 30°
Tx w/ NSAIDs, avoidance of aggravating movements, PT, subacromial steroid injections if no improvement |
| Frozen shoulder (adhesive capsulitis) | | Gradual ↓ ROM (active & passive) of glenohumeral joint due to joint capsule pathology; no known injury
Pain at extremes of ROM
Tx w/ NSAIDs, PT, 2–4 wk oral corticosteroids |
| Acromioclavicular syndrome | Arthritis or injury to AC ligaments
Acute or chronic, possible hx of trauma
TTP & swelling of AC joint, pain worse w/ ↓ traction or forced passive adduction
Acute injury tx w/ sling |
| Elbow | Lateral epicondylitis (tennis elbow) | Pain along lateral epicondyle at attachment of extensor tendons of forearm
↑ pain w/ resisting wrist dorsiflexion
Tx w/ rest, NSAIDs, ±steroid injections |
| Medial epicondylitis (golfer elbow) | Less common than tennis elbow
Pain along medial epicondyle at insertion of common flexor tendon |
|  |  |  |
### Tendon

**Resistance to wrist flexion w/ elbow in extension ↑ pain**
Tx w/ rest, NSAIDs, ±steroid injections

| Hip | Trochanteric bursitis | Most common cause of pain in hip (lateral aspect)  
|     |                        | Pain ↑ w/ walking, squatting, climbing stairs, ↓ at rest  
|     |                        | Resisted abduction of hip reproduces pain  
|     |                        | Tx w/ NSAIDs, corticosteroid injections |

| Knee | Patellar tendonitis | Pain at inferior aspect of patella during repetitive running, jumping, kicking  
|      | (jumper’s knee)     | Tx w/ rest, NSAIDs, knee brace, PT, strengthening exercises for quads & hamstrings |

| Ankle | Achilles tendonitis | Pain, swelling, tenderness, over Achilles tendon from repetitive trauma & microscopic tears from overuse (ballet, distance running, basketball)  
|       |                    | ↑ pain w/ passive dorsiflexion  
|       |                    | Tx w/ rest, heat, NSAIDs, shoe modification, heel lift to ↓ tendon stretching, PT, stretching exercises |

---

**TENOSYNOVITIS**

**Definition**

- Inflammation of the tendon & tendon sheath. Can result in chronic disability, ↓ ROM, chronic pain, amputation if not treated appropriately.

<table>
<thead>
<tr>
<th>Types of Tenosynovitis</th>
<th>Hx &amp; Findings</th>
<th>Management &amp; Disposition</th>
</tr>
</thead>
</table>
| De Quervain tenosynovitis | Repetitive pinching of thumb & fingers  
|                         | Pain improved w/ rest; no hx of acute trauma  
|                         | Most common in middle-aged women  
|                         | Pain at radial aspect of wrist, worse w/ passive ROM of thumb, ulnar deviation of wrist w/ thumb cupped in closed fist (Finkelstein test) | Rest, NSAIDs  
|                         | Thumb spica  
|                         | Steroid injection  
|                         | Surgery if needed  
|                         | Good prognosis  
|                         | DC home w/ f/u |
| Stenosing flexor tenosynovitis (trigger finger) | Locking of thumb or ring finger in flexion followed by sudden release, pain radiates to fingers  
|                                              | Most common in middle-aged women, diabetics  
|                                              | Pain in proximal tendon sheath in distal palm  
|                                              | ±Palpable tendon thickening or nodularity  
|                                              | May require manipulation to release | NSAIDs  
|                                              | Splint 4–6 wk  
|                                              | ±Steroid injection  
|                                              | Surgical release if injection fails  
|                                              | Good prognosis  
|                                              | D/C home w/ f/u |
| Infectious tenosynovitis | Puncture wound, laceration, bite, cracked skin, high-pressure injury; usually *S. aureus, Strep* | Admit ortho/hand  
|                         | Abx |
GOUT

History
· Middle-aged pt w/ abrupt (often recurrent) onset single joint pain, swelling, erythema, warmth; may be precipitated by minor trauma or illness
· RFs: HTN, HLD, DM, obesity. Systemic etiologies: Cancer, hemolysis.
· 75% monoarticular, classically affects 1st MTP joint (aka “podagra”)

Findings
· Red, swollen, tender, warm joint (MTP > ankle > dorsal area > knee); mimics cellulitis
· Tophi overlying effected joints indicate chronic gouty dz

Evaluation
· Arthrocentesis if: 1st episode (no prior tap), unclear Dx, concern for septic joint
· Joint fluid: Needle-shaped, neg. birefringent crystals; always send for culture
· Serum uric acid level is of no value; 30% will have nl levels
· X-ray findings in chronic gout include bony erosions, punched out lesions, calcified tophi

Treatment
· NSAIDs (no aspirin). Eg, indomethacin 50 mg PO TID for duration of attack (~3–10 d).
· Alternatively: Colchicine (0.5 mg PO q1h up to 8 mg; if nl renal fxn) OR steroids
· Allopurinol for chronic prevention but has no role in acute mgmt of gout attack

Disposition
· D/C home w/ pain control unless intractable pain

Kanavel signs:
1. Fusiform (“sausage”) swelling of finger
2. Flexed position of finger
3. Severe pain w/ passive extension
4. Tenderness along flexor tendon sheath

Splint & elevation
Fair prognosis even w/ abx, surgery
Pearl
› Gout is a result of monosodium urate crystal deposition

PSEUDOGOUT

History
› Elderly pt w/ abrupt-onset, single-joint pain, swelling, erythema, warmth; precipitated by minor trauma or illness; usually in large joints (unlike gout)

Findings
› Red, swollen, tender, warm joint (knee > wrist > ankle = elbow)

Evaluation
› If unclear Dx, concern for septic joint, perform arthrocentesis
› Joint fluid: Rhomboid-shaped, positively birefringent crystals
› X-ray findings: Chondrocalcinosis, subchondral sclerosis, radiopaque calcifications

Treatment
› Same as gout

Disposition
› D/C home w/ pain control

Pearls
› Pseudogout is the result of calcium pyrophosphate crystal deposition
› Most common cause of new monoarticular arthritis in pts >60 y/o
› RFs: ↑ Ca, ↓ Mg, ↓ PO₄, hemochromatosis, hemosiderosis, parathyroid dz

BURSITIS

Definition
› Inflammation of bursa, which are flattened sacs lined w/ synovial fluid that helps facilitate movement; bursitis is usually due to overuse, trauma, or OA, but can be septic

History
› Discrete area of pain, swelling, erythema, warmth over a joint
Less than half of bursitis is septic, but 70% of septic bursitis has preceding trauma
Most common in joints that are subject to repetitive stresses (elbow, knee), but can be deep (hip) esp in setting of instrumentation (eg, acupuncture, surgery)

**Findings**
- Warm, swollen, fluid-filled pocket outside the joint ± erythema
- Tenderness, fever, associated cellulitis suggest septic bursitis
- Should have minimal pain w/ passive ROM; o/w consider septic arthritis

**Evaluation**
- If any concern for septic bursitis, perform bursa aspiration (WBC >5K is suggestive)
- Deep bursae may require aspiration by ortho or IR
- Often clinically difficult to differentiate from septic arthritis; may need arthrocentesis

**Treatment**
- Rest, ice, elevation, analgesia, ± steroid injection
- If septic bursitis: Abx for *Staph* coverage (ie, dicloxacillin, TMP–SMX, or clindamycin)
- Consult ortho for f/u as these have high outpt failure rate & may need surgical bursal excision or serial aspirations

**Disposition**
- D/C w/ pain control if no ortho intervention, ± abx
- Admit for fulminant infection, immunocompromised pt, significant surrounding cellulitis

**Pearl**
- Prepatellar (carpet layer’s knee) & olecranon bursitis (student’s elbow) are usually due to *Staph* infection from local trauma

---

**Infectious**

**Septic Arthritis (Nongonococcal)**

**History**
- Acute onset of painful, swollen, warm, tender joint, often w/ fever
- Hallmark is severe pain w/ any passive ROM
All joints are at risk but most commonly knee > hip. In pediatrics, hip is most common.

High-risk groups include IV drug users, immunocompromised

**Findings**
- Usually single joint involvement; can see multijoint in disseminated GC dz
- Pain w/ minimal passive ROM or axial load; warmth, redness, swelling

**Evaluation**
- X-ray to identify effusion, FB, fracture, or osteomyelitis
- Arthrocentesis: Gram stain & culture, cell count, protein & glucose, crystal analysis, synovial lactate; positive: WBC >50000 w/ PMN predominance
- Labs: Consider ESR, CRP, blood cx to isolate; UA, CXR for infectious w/u

**Management**
- Arthrocentesis (hip may need orthopedics or IR), ortho consult, splint in physiologic position
- Supportive care: hydration, antipyretics, pain control
- Abx after arthrocentesis & blood cultures taken; S. aureus is most common
- Adults: Vancomycin & 3rd-generation cephalosporin OR quinolone
- Children <14 yr: Vancomycin & 3rd-generation cephalosporin
- Prosthesis, immunocompromised: Vancomycin & antipseudomonal (piperacillin/tazobactam OR fluoroquinolone)

**Disposition:** Admit for abx, ortho observation, likely need for operative washout

**Pearls**
- Septic hips do not present w/ classic signs; can be very subtle
- Presence of crystals in the joint fluid does NOT r/o a septic joint
- Overlying cellulitis is relative CI for arthrocentesis; avoid cellulitic area during tap
- If hardware is present, discuss risk/benefit w/ orthopedics prior to arthrocentesis
- Intra-articular steroid injection for pain relief in septic arthritis is contraindicated
Gonococcal Septic Arthritis

**History**
- Young, sexually active pt usually c/o single painful, swollen, warm, & tender joint
- May be polyarticular or migratory; smaller joints (elbow, wrist, ankle) commonly involved
- Urethral or vaginal D/C of GC infection may be present

**Findings**
- Any clinical manifestations of *Neisseria* GC infection (cervicitis, malodorous, purulent vaginal D/C in female or dysuria & penile D/C in male)
- Swollen, tender, warm, & extremely painful small joint(s), usually slightly flexed at rest, more painful w/ ROM; may have tenosynovitis
- A painless diffuse maculopapular rash w/ necrotic/pustular centers may be present
- RUQ abd pain may indicate Fitz-Hugh–Curtis syndrome

**Evaluation**
- Same as non-GC septic arthritis + cervical (female) & urethral (male) cultures; blood, pharynx & rectal cultures to ↑ likelihood of definitive dx

**Treatment**
- Arthrocentesis, ortho consult, splint joint in physiologic position for comfort
- 3rd-generation cephalosporin (ceftriaxone 1 g IV QD) OR quinolone, add doxycycline for chlamydia
- Supportive care: Hydration, antipyretics, pain control

**Disposition:** Admit for abx, ortho observation, possible need for operative washout

**Pearls**
- GC septic arthritis is the only septic arthritis that does not necessarily need operative washout; however, serial arthrocentesis to remove fluid may be indicated
- Gram stain & culture from GC septic arthritis more often neg. than non-GC septic joints
- Intra-articular steroid injection for pain relief in septic arthritis is contraindicated
EAR PAIN

Approach
- Nature of pain, associated sx, duration, fevers, hearing loss; diabetes

<table>
<thead>
<tr>
<th>Ear Pain Differential</th>
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</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
</tr>
<tr>
<td>Outer ear</td>
</tr>
<tr>
<td>Middle ear</td>
</tr>
<tr>
<td>Mastoid air cells</td>
</tr>
</tbody>
</table>

Otitis Externa (Swimmer’s Ear)

Definition
- Infection (Acute: *Pseudomonas*, *S. aureus*, *S. epidermidis*; Chronic: *Aspergillus*, *Candida*) of the outer ear due to breakdown of natural barriers

History
- Summer, water exposure, cotton swab trauma, hearing aids, pain/itching/drainage

Physical Findings
- Pain w/ movement of tragus/helix, localized LAD, redness/exudate in canal, white/gray debris, ± green d/c/yellow crusting, ± abscess, conductive hearing loss if severe

Treatment
- Remove debris, dry canal w/ suction, drain abscess if present
- Mild infections: Cleanse w/ 2% acetic acid, hydrogen peroxide, OR sterile saline; no good evidence for these. Avoid w/ ruptured TM
- Severe infections: Topical abx (eg, ofloxacin) + steroid × 7 d
  - Use wick (cotton, gauze, or cellulose) 10–12 mm into canal × 2–3 d to allow med delivery
  - If TM rupture, consider oral abx
No swimming × 48 h, keep ear dry in shower × 1 wk (ear plugs or Vaseline gauze seal)

Disposition
- Home: Diabetics w/ simple OE should get close f/u

Malignant (Necrotizing) Otitis Externa
Definition: Aggressive infection (95% *Pseudomonas*) of the outer ear canal to skull base/bony structures, usually in diabetics/immunocompromised

History: Ear pain extending to TMJ (pain w/ chewing), nocturnal pain, swelling, otorrhea

Physical Findings: Granulation tissue, severe inflammation, may have CN palsy

Evaluation: Consider CT scan to eval extent, underlying osteomyelitis, & intracranial extension

Treatment
- 1st line: IV ciprofloxacin; Increasing rates of resistance; 2nd line: Ceftazidime, imipenem, OR piperacillin/tazobactam
- Consider amphotericin B or voriconazole for aspergillus in HIV/immunocompromised

Disposition: Admission for IV abx ± operative débridement

Pearls
- 10% mortality
- Cx: Cerebral/epidural abscess, osteomyelitis, dural sinus thrombophlebitis, meningitis

Otitis Media
Definition
- Inflammation of the middle ear
- Acute OM: Infection (50% *S. pneumoniae*, 20% *H. influenzae*, 10% *M. catarrhalis*, viral 50–70%) + effusion <3 wk
- Chronic OM: Effusion w/o infection

History: Unilateral ear pain, fever (25%), winter/spring, 2–10 y/o, URI

Physical Findings: Bulging TM, loss of light reflex/TM mobility (most sens), effusion, erythema (not sufficient alone to diagnose OM), purulent drainage
Treatment
- Many improve w/ no abx w/o cx
- Pain control: APAP/ibuprofen, auralgan (topical)
- Nonsevere acute OM: Amoxicillin to start in 2–3 d if sxs do not improve
- Severe (<6 mo, bilateral, bulging TM, otorrhea, fever >39°C, systemically ill) = immediate abx
- Pediatric: Amoxicillin 80–90 mg/kg/d (1st line) 7–10 d, amoxicillin/clavulanate if recent abx or concurrent conjunctivitis (Pediatrics 2010;125(2):384)
- Adult: Amoxicillin 500 mg BID (mild to mod), 875 mg BID (severe), Cefpodoxime OR cefuroxime if PCN allergic

Disposition: Home, PCP f/u 2–3 d

Pearls
- Cx (rare): Meningitis, mastoiditis, persistent effusion → hearing loss
- TM perforation does not require any Δ in management

Mastoiditis
Definition: Extension of infection from the middle ear into the mastoid air cells

History: Unilateral ear pain, fever, HA

Physical Findings: Tenderness, erythema, fluctuance over mastoid, outward bulging pinna

Evaluation: CT scan to eval extent/destruction of the septa of the air cells, MRI for intracranial cx, ENT consult

Treatment
- Abx: Nafcillin/cefuroxime/ceftriaxone
- ±Myringotomy/tympanostomy; mastoidectomy (if 50% of air cells involved)

Disposition: Admission, possible operative débridement
Pearl: Cx include meningitis, dural sinus thrombosis, brain abscess, subperiosteal abscess, hearing loss

HEARING LOSS

Approach
Nature, acuity of onset, unilateral/bilateral, associated pain/systemic sx$s$

<table>
<thead>
<tr>
<th>Hearing Loss Differential</th>
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<tbody>
<tr>
<td><strong>Cause</strong></td>
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<tr>
<td>Infections</td>
</tr>
<tr>
<td>Vascular</td>
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<tr>
<td>Metabolic</td>
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<tr>
<td>Conductive</td>
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<tr>
<td>Medications</td>
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<tr>
<td>Neoplasm</td>
</tr>
</tbody>
</table>

**Cerumen Impaction/Foreign Body**

**Definition:** Buildup of earwax or FB in the external canal

**History:** Unilateral hearing loss, placement of FB in ear, drainage, pain

**Physical Findings:** Visualization or cerumen/FB in ear

**Treatment**

- Irrigate the external canal w/ room temperature NS (cold/hot NS can cause nystagmus/vertigo/nausea), past FB if possible. Do not irrigate batteries
- For live insects, consider liquid lidocaine, isopropyl alcohol, or mineral oil to asphyxiate prior to removal
- For cerumen: Instill colace, cerumenex, or $H_2O_2$ for 15 min to dissolve, then irrigate
- For FB: Alligator forceps OR cyanoacrylate (glue) to cotton-tipped applicator, hold against object for 60 sec; OR try suction for smooth objects
- Re-examine ear postextraction for TM rupture, canal damage, or residual FB. Consider topical abx if canal damaged

**Disposition:** D/c

**Ruptured Tympanic Membrane**

**Definition:** Rupture of the TM. Etiologies include trauma (open hand slap
History: Pain, hearing loss
Physical Findings: Perforation of TM, ± blood in the canal

Treatment
- Keep ear dry (earplugs during shower, no swimming)
- Abx needed if pre-existing infection; treat as usual OM; consider abx if contaminated water exposure
- Operative repair if >¼ of TM damaged

Disposition
- D/c, ENT f/u 2–4 d for audiogram; perforations usually heal in 2–3 mo
- Admit in acute trauma w/ associated facial nerve injury, incapacitating vertigo

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**SORE THROAT**

**Approach**
- Nature, acuity of onset, duration, associated sxs (cough, fever, drooling, voice Δ, dysphagia, difficulty breathing)

<table>
<thead>
<tr>
<th>Sore Throat Differential</th>
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<tbody>
<tr>
<td><strong>Cause</strong></td>
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<tr>
<td>Systemic</td>
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<tr>
<td>Trauma</td>
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<tr>
<td>Tumor</td>
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</tbody>
</table>

**Group A Streptococcus Pharyngitis (“Strep Throat”)**
Definition: Infection of the oropharynx caused by GABHS
History: Sore throat, odynophagia, myalgias, fever; no cough
Physical Findings: Erythematous oropharynx, tonsillar exudate, cervical LAD
Evaluation
- Centor criteria: Fever >38°C, tonsillar exudate, tender LAD, absence of cough
- Rapid strep: Sens 60–90%, spec 90% (send culture if neg. given low sens)
- GABHS culture: 90% sens
- Consider culture for gonorrhea (if oral sex exposure), or Monospot for EBV

Treatment
- There are multiple conflicting guidelines (NEJM 2011;364:648). One reasonable approach:
  - If 0–1 Centor criteria met: No testing, no tx
  - If 2–3 Centor criteria met: Rapid strep, treat if positive, confirm w/ culture
  - If all Centor criteria met: No testing, yes tx
- Abx
  - Benzathine penicillin 25000 U/kg max 1.2 million U IM ×1 OR penicillin VK, OR amoxicillin OR azithromycin. If refractory: Clindamycin, augmentin
  - Dexamethasone 8 mg ×1 may ↓ time to pain relief (J Emerg Med 2008;35(4):363)

Disposition: D/c
Pearl: Treat w/ abx to prevent scarlet fever, rheumatic fever, abscess, mastoiditis. Poststrep glomerulonephritis is not prevented w/ abx

Croup (Laryngotracheobronchitis)
Definition
- URI in children (6 mo–6 yr) usually by parainfluenza virus causing inflammation/exudate/edema of subglottic mucosa

History: Barky cough, worse at night, low-grade fever, following 2–3 d of URI sxss
Physical Findings: High-pitched inspiratory stridor, barking cough, hoarse voice, tachycardia, tachypnea

<table>
<thead>
<tr>
<th>Croup Severity Score (Westley Score)</th>
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<tbody>
<tr>
<td>Inspiratory stridor</td>
</tr>
<tr>
<td>None = 0, w/ agitation = 1, at rest = 2</td>
</tr>
<tr>
<td>Retractions</td>
</tr>
<tr>
<td>------------------</td>
</tr>
<tr>
<td>Air entry</td>
</tr>
<tr>
<td>Cyanosis</td>
</tr>
<tr>
<td>Level of alertness</td>
</tr>
<tr>
<td>Score ≤2 = mild, 3–5 = moderate, &gt;6 = severe</td>
</tr>
</tbody>
</table>

**Evaluation:** Neck film is typically of no clinical value → narrowing of subglottic trachea (“steeple sign”)

**Treatment**
- Calm child, monitor pulse oximetry
- Cool mist (no clear benefit)
- Dexamethasone 0.3–0.6 mg/kg (↓ time to improvement) ([Cochrane Syst Rev](Cochrane Syst Rev 2004;(1):CD001955))
- Moderate–severe or stridor at rest: Nebulized racemic epinephrine 0.5 mL of 2.25%

**Disposition**
- Admit if no improvement in ED, hypoxic, persistent stridor at rest, <6 mo old, unable to tolerate PO, requiring multiple doses of epinephrine
- Croup severity score ≤4 can usually be D/C, score >6 may require ICU

**Pearl:** If epinephrine given, should observe for >3–4 h for rebound stridor

**Epiglottitis**

**Definition**
- Inflammation of the epiglottis caused by *H. influenzae* >> *Staph/Strep, B. catarrhalis*
- Can lead to rapidly progressing, life-threatening airway obstruction

**History**
- Sore throat, muffled “hot potato” voice, odynophagia, respiratory distress, fever
- ↓ Pediatric incidence since vaccination, now more common in adult diabetics

**Physical Findings:** Dysphonia, stridor, drooling, sitting in tripod position

**Evaluation**
- Lateral neck XR (90% sens): Epiglottis >7 mm (“thumbprint”), loss of
vallecular air space

- Adult: If nl x-ray → indirect or fiberoptic laryngoscopy (have surgical airway ready)
- Pediatric: Avoid agitation (↑ risk of acute airway obstruction), do NOT attempt to visualize in the ED. To OR for DL w/ anesthesia & ENT/surgery

Treatment: Abx (ceftriaxone OR ampicillin–sulbactam, add clindamycin or vancomycin if concern for MRSA); no proven benefit w/ steroids

Disposition: ICU admission

Pertussis (Whooping Cough)
Definition: Lower respiratory tract infection by B. pertussis (gram-neg. rod)

Presentation
- Commonly a prolonged course (aka “hundred-day cough”)
- Stages: (1) Catarrhal (most infectious): 2 wk mild URI sx; (2) Paroxysmal: 1–2 wk intense paroxysmal cough ± posttussive emesis, inspiratory “whoop”; (3) Convalescent: Several weeks of chronic cough
- ↑ Risk if unvaccinated, but immunity wanes after ~12 yr; ↑ morbidity if <6 mo old

Evaluation
- Rapid PCR may be useful esp during epidemics
- May develop PNA; consider CXR if refractory to abx

Treatment
- Droplet precautions × 7 d, abx (only effective in catarrhal stage)
- Azithromycin or clarithromycin, albuterol prn, treat household contacts
- Low threshold for empiric tx in infants, pregnant, healthcare workers

Disposition: Admit <6 mo–1 y/o or ill appearing

Lemierre Syndrome
Definition
- Suppurative thrombosis of internal jugular vein w/ F. necrophorum
- Septic emboli to lung are common (can be confused w/ R-sided endocarditis)

History
- Usually previously healthy young adults w/ high fever, sore throat ±
cough

- Typical course is pharyngitis that improves & then followed by severe sepsis

**Physical Findings:** Unilateral neck swelling, tenderness, induration

**Evaluation:** Contrast CT of neck

**Treatment:** Abx: Ampicillin–sulbactam, piperacillin–tazobactam or a carbapenem. Consider adding vancomycin if catheter-associated. Anticoagulation is controversial

**Disposition:** Admit

---

**SINUSITIS**

**Acute Sinusitis**

**Definition**

- Inflammation of the paranasal sinuses
- Usually viral or allergic
- Common bacterial etiologies: *S. pneumoniae*, nontypable *H. influenzae*, *M. catarrhalis*
- *Pseudomonas* is seen in HIV, cystic fibrosis, or after instrumentation
- Mucormycosis is invasive fungal sinusitis (*Rhizopus*) in diabetics or immunocompromised

**Presentation**

- Mucopurulent d/c, postnasal drip, cough, sinus pressure, HA, ±fever
- Typically progresses over 7–10 d & resolves spontaneously
- Sxs >7 d, worsening course, or worsening after improving, all suggest bacterial dz
- Consider sinusitis w/ positional HA that is worse when bending forward
- Sphenoid sinusitis is a difficult Dx, often presents late; classically worse w/ head tilt

**Evaluation**

- Clinical, no routine imaging. CT sens but not spec, can r/o cx
- Cx include orbital cellulitis, osteomyelitis, cavernous sinus thrombosis, cerebral abscess, meningitis, frontal bone abscess (Pott puffy tumor)

**Treatment**

- Supportive (analgesics, antipyretics, decongestants, antihistamines if
allergic)

- Decongestants: Neo-Synephrine nasal spray TID × 3 d, Afrin nasal spray BID × 3 d
- Abx not routinely indicated. Reserve for pts w/ sxs >7 d, worsening sxs, fever, purulent d/c, or high risk for severe infection or cx
  - Amoxicillin–clavulanate 500 mg PO TID × 5–7 d
  - RFs for resistance: high-dose amoxicillin–clavulanate (2000 mg BID)

**Disposition**

- Vast majority are managed outpt
- Admit if toxic, severe HA, high fever, immunocompromised, poor f/u

**Pearl**

- Sphenoid/ethmoid sinusitis is less common than maxillary sinusitis but has significant potential cx (eg, orbital cellulites, cavernous sinus thrombosis)

---

**EPISTAXIS**

**Definition:** Bleeding from the nose. 90% of cases are anterior & involve Kiesselbach plexus on the septum. 10% of cases are posterior & arise from a branch of sphenopalatine artery

**History**

- Etiologies include URI (most common), trauma, nose picking, environmental irritants (dry air), intranasal drug use, neoplasm, FB, polyps, anticoagulation/TCP
- RFs: Alcoholism, diabetes, anticoagulation, HTN, hematologic disorder

**Physical Findings**

- Evaluate w/ nasal speculum after having pt blow nose to express clots

**Evaluation**

- Can usually identify anterior source on exam; posterior bleeds are heavy, brisk, can cause airway compromise. If still bleeding after anterior packing, consider posterior source
- Check hematocrit if extensive/prolonged bleeding, INR if on warfarin

**Treatment**

- If significantly hypertensive, consider antihypertensive to help w/ hemostasis
Anterior: Start w/ oxymetazoline (Afrin) 3 sprays & hold pressure for 15 min
- May also insert cotton pledgets soaked in cocaine/lidocaine/epinephrine/phenylephrine
- Once vasoconstricted, try to identify a focal bleeding site, then use silver nitrate cautery in ring around bleeding (will not work on active bleeding; caution on septum)
- If bleeding has stopped, observe for 60 min; if recurs, insert a lubricated nasal tampon or vaseline gauze packing
- If nasal tampon is not successful, pack the contralateral side

Posterior: Bleeding can cause airway compromise & be life-threatening
- Commercial double balloon device OR pass Foley catheter through nose into posterior pharynx, fill balloon, hold gentle traction

Disposition
- Anterior: D/c w/ 48 h f/u, typically w/ prophylactic abx for TSS (unproven) (eg, clindamycin, augmentin, or dicloxacillin)
- Posterior: Admit w/ ENT consult

EYE PAIN/REDNESS

Approach
- Ask about FB exposure, chemicals, trauma, contact lens use, freshwater exposure
- Always check visual acuity. Use topical anesthetics (tetracaine, proparacaine) for exam
- Complete eye exam: Visual acuity (corrected), visual fields, external inspection, periorbital soft tissue & bones, extraocular movement, pupils (including swinging light test for afferent pupillary defect), pressure (tonometry), slit lamp (lids, conjunctiva, sclera, cornea w/ fluorescein, anterior chamber, iris, lens), fundoscopy

Acute Angle-closure Glaucoma
Definition: Increased IOP due to ↓ aqueous outflow. Generally due to reduction in the angle of the anterior chamber in setting of the dilated pupil pushing against trabecular meshwork

History
- Sudden onset of severe unilateral pain, HA, N/V, blurry vision, halos
- May be triggered by dim light, mydriatic drops, stress, sympathomimetics

**Physical Findings**
- Unilateral perilimbal eye injection, ↓ VA, “steamy” (cloudy) cornea, nonreactive midsize pupil (5–7 mm), shallow anterior chamber, ↑ IOP >22 mmHg, firm globe

**Treatment**
- *Immediate optho consult; need for urgent laser peripheral iridotomy*
- *Reduce aqueous production:* Timolol 0.5% 1–2 drops q30min (avoid if CI to systemic βB), acetazolamide 500 mg IV, then 250 q6h (avoid in sulfur-allergic pts) or brimonidine 1 drop TID
- *Facilitate aqueous outflow* (miotics): Pilocarpine 2% 1 drop q15min until pupil constricts
- *Decrease vitreous volume* (osmotics): Mannitol 1–2 mg/kg IV over 30–60 min

**Disposition**
- Per optho recommendations. Admit for intractable vomiting or need for systemic agents

<table>
<thead>
<tr>
<th>Critical Dx</th>
<th>Etiology</th>
<th>Features</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Caustic injury (chemical)</td>
<td>Hx: Chemical exposure PE: Corneal burns (esp w/ alkali), pain, blepharospasm</td>
<td>Immediate optho consult Immediate copious (2–4 L) irrigation until pH = 7</td>
</tr>
<tr>
<td></td>
<td>Acute angle-closure glaucoma</td>
<td>See discussion above</td>
<td>See discussion above</td>
</tr>
<tr>
<td></td>
<td>Retrobulbar hematoma</td>
<td>Hx: Often due to trauma, but also spontaneous in coagulopathy or due to tumor PE: Decreased acuity, diplopia, proptosis, afferent pupillary defect ± pale optic disc</td>
<td>IOP &gt;20 = orbital compartment syndrome Immediate optho consult Lateral canthotomy if: - Conscious, ↓ IOP, ↓ VA - Unconscious, IOP &gt;40 &amp; proptosis - CI: ruptured globe</td>
</tr>
<tr>
<td></td>
<td>Penetrating trauma/scleral penetration</td>
<td>Hx: Blunt (blow to orbit or globe) or penetrating PE: ↓ acuity, afferent papillary defect, classically teardrop-shaped pupil, Seidel sign (aqueous leak on fluorescein)</td>
<td>Apply eye shield Immediate optho consult IV abx Tetanus prophylaxis CT scan to assess for FB</td>
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**Critical Dx**

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<td>Apply eye shield Immediate optho consult IV abx Tetanus prophylaxis CT scan to assess for FB</td>
</tr>
<tr>
<td>Corneal ulcer/keratitis</td>
<td>Hx: Pain, FB sensation, photophobia, tearing, blurry vision. Recent contact lens use, UV light exposure, Bell palsy or abrasion PE: Fluorescein: Corneal infiltrate (white spots/haze) around sharply demarcated “scooped out” epithelial defect - Herpes: Dendritic - UV keratitis: Many punctate ulcers (snowfall pattern)</td>
<td>Immediate optho consult May need to débride or culture prior to abx - Ciprofloxacin - Cycloplegics - Acyclovir if possibly HSV</td>
</tr>
<tr>
<td>Orbital cellulitis (vs. preseptal cellulitis)</td>
<td>Orbital cellulitis: Posterior to orbital septum, drains into cavernous sinus Both orbital &amp; preseptal: - May have fever, leukocytosis - Lid swelling, erythema, warmth - Eye tenderness - Conjunctivitis, chemosis Suspect orbital cellulitis if: - Ill appearance, high fever - Pain w/ EOM movement - Ophthalmoplegia/diplopia - Visual impairment - Proptosis - Increased IOP</td>
<td>Immediate optho consult for orbital cellulitis IOP &gt; 20: Optho emergency CT orbit to r/o FB, abscess Obtain blood cx Start IV abx (vancomycin + ceftriaxone or ampicillin/sulbactam) In diabetics, consider mucormycosis Admit all orbital cellulitis If preseptal cellulitis, outpt abx w/ amoxicillin/clavulanate &amp; optho recheck in 1 d Cx: vision loss, cavernous sinus thrombosis, CNS involvement, abscess, osteomyelitis</td>
</tr>
</tbody>
</table>

**Emergent Diagnoses**

| Hyphema | Hx: Pain, ↓ visual acuity, usually after blunt trauma PE: Gross or microscopic blood layering in anterior chamber, ±fixed & dilated pupil | First r/o open globe Discuss w/ optho-“eight ball” hyphema requires urgent f/u IOP > 30: Treat as glaucoma IOP > 20: Use cycloplegic to prevent iris motion Elevate HOB 45 degrees Screen for FH of sickle cell Most can be D/C home w/ 1–2 d recheck Return for ↑ pain or ↓ vision |
| Corneal abrasion/FB | Hx: Pain worse w/ blinking, photophobia, FB sensation PE: Conjunctival injection. Evert | If high velocity: XR or CT to r/o ocular penetration If embedded FB, remove w/ 25 |
lids to look for FB. Use fluorescein to eval -Rust ring = metallic FB -Seidel test to r/o corneal penetration

g needle tip under magnification, or burr
Give tetanus prophylaxis
Abx (erythromycin), use quinolone if contact use or freshwater exposure
DC home w/ optho f/u in 1–2 d for recheck, rust ring removal if needed. No contacts until resolved

| Anterior uveitis/iritis | Def: Inflammatory process involving anterior chamber, iris, ciliary body, or choroid | • Traumatic iritis: Cycloplegic for comfort, optho f/u in 1–2 d
• Inflammatory: Cycloplegics, consult optho for possible steroids |
<table>
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<tbody>
<tr>
<td>Hx: Usually due to trauma, autoimmune dz, or infection (HSV, Lyme). Unilateral painful red eye, &quot;deep&quot; pain, blurred vision, photophobia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Findings: Perilimbal injection, photophobia (consensual suggests iritis), ± ↓ visual acuity, slit lamp shows anterior chamber cell &amp; flare</td>
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</tr>
</tbody>
</table>

Other Causes of Red Eye

<table>
<thead>
<tr>
<th>Conjunctivitis (allergic, viral &gt; bacterial)</th>
<th>Def: Inflammation of mucus membranes that line sclera/lids. Usually viral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hx: Drainage, irritation, pruritus, crusting, concurrent URI</td>
<td></td>
</tr>
<tr>
<td>PE: Injection/edema, usually sparing limbus</td>
<td></td>
</tr>
<tr>
<td>nl exam o/w</td>
<td></td>
</tr>
<tr>
<td>Gonorrhea = copious, green exudate</td>
<td></td>
</tr>
<tr>
<td>Culture if neonate or concern for Chlamydia, gonorrhea</td>
<td></td>
</tr>
<tr>
<td>Warm soaks, artificial tears</td>
<td></td>
</tr>
<tr>
<td>Antihistamine if allergic</td>
<td></td>
</tr>
<tr>
<td>Abx if concern for bacterial:</td>
<td></td>
</tr>
<tr>
<td>- Erythromycin, Polytrim</td>
<td></td>
</tr>
<tr>
<td>- Quinolone if contact lens or freshwater exposure</td>
<td></td>
</tr>
<tr>
<td>D/c home, optho f/u in 2 d if not improving</td>
<td></td>
</tr>
<tr>
<td>Consult optho if gonorrhea suspected</td>
<td></td>
</tr>
</tbody>
</table>

Etiology  | Features  | Management |
|----------|-----------|------------|
| Lid disorders (blepharitis, chalazion, dacrocystitis, hordeolum/stye) | **Blepharitis**: Inflamed eyelid margins **Chalazion**: Inflamed meibomian gland (subcutaneous lid nodule) **Dacrocystitis**: Inflamed lower eye lid w/ redness, tenderness **Hordeolum (stye)**: Abscess in | **Blepharitis**: Warm compresses
**Chalazion**: Warm compresses, gentle massage
**Dacrocystitis**: R/o periorbital or orbital cellulitis. If mild, d/c w/ clindamycin & warm compresses. Admit if systemically ill
**Hordeolum:** |
eyelash follicle or lid margin (can be external or internal)

- External: Warm compresses ± abx ointment for Staph
- Internal = PO abx for Staph

VISION CHANGE & VISION LOSS

Approach

- Complete eye exam: Visual acuity (corrected), visual fields, external inspection, periorbital soft tissue & bones, extraocular movement, pupils (including swinging light test for afferent pupillary defect), pressure (tonometry), slit lamp (lids, conjunctiva, sclera, cornea w/ fluorescein, anterior chamber, iris, lens), fundoscopy, & full neurologic exam

<table>
<thead>
<tr>
<th>Differential of Vision Δ &amp; Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Differential</strong></td>
</tr>
<tr>
<td>Painful</td>
</tr>
<tr>
<td>Trauma, glaucoma, uveitis, corneal ulcer, temporal arteritis, optic neuritis</td>
</tr>
<tr>
<td>Painless</td>
</tr>
<tr>
<td>Amaurosis fugax/TIA, central retinal artery/vein occlusion (CRAO/CRVO), vitreous hemorrhage, retinal detachment, lens dislocation, hypertensive encephalopathy, pituitary tumors, macular disorders, toxic ingestions (toxic alcohols, heavy metals)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differential of Diplopia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Differential</strong></td>
</tr>
<tr>
<td>Monocular</td>
</tr>
<tr>
<td>Astigmatism, cataracts, lens dislocation</td>
</tr>
<tr>
<td>Binocular</td>
</tr>
<tr>
<td>Entrapment, CN palsy, intracranial mass effect, thyroid dz, microvascular dz</td>
</tr>
</tbody>
</table>

Central Retinal Artery Occlusion

**Definition:** Retinal artery occlusion, most commonly embolic

**History**

- Sudden painless, monocular vision loss (or visual field cut if branch of retinal artery), may have transient loss prior to complete loss (amaurosis fugax)
- RFs: HTN, DM, CVA, AF, carotid dz, hypercoagulable, vasculitis, endocarditis, sickle cell anemia
Physical Findings
- Afferent pupillary defect, funduscopic exam shows cherry-red spot at fovea (spared), pale disc (late finding)
- May have carotid bruit, irregular HR, murmur; r/o temporal arteritis

Evaluation
- CBC, ESR
- For embolic w/u: Neuroimaging (CT/CTA or MRI/MRA), carotid imaging, echo, EKG

Treatment
- Initiate immediately (>2 h = irreversible vision loss)
- Immediate ophthalmologic consult
- Intermittent globe massage (to try to dislodge embolus & move it further downstream)
- Reduce IOP as in glaucoma (eg, acetazolamide, mannitol, timolol)
- Anterior chamber paracentesis
- Surgical decompression, anticoagulation, intra-arterial thrombolysis, hyperbaric $O_2$

Disposition: Admit
Pearl: Cardiac embolus most common in >40 y/o, coagulopathies most common in <30 y/o

Central Retinal Vein Occlusion
Definition: Retinal vein occlusion, usually thrombotic

History
- Sudden painless monocular vision loss (may be gradual onset)
- RFs: CAD, HTN, glaucoma, venous stasis, hypercoagulable, DM, vascular dz

Physical Findings: Afferent pupillary defect, funduscopic exam w/ retinal hemorrhages/disk edema (“blood & thunder”), cotton wool spots
Management: Immediate optho consult. Start ASA, outpt hypercoagulability w/u
Disposition: Home

Temporal Arteritis (Giant Cell Arteritis)
Definition: Granulomatous inflammatory vasculitis of medium/large arteries
History
- Unilateral HA, jaw/tongue claudication, malaise, low-grade fevers, visual impairment
- Usually >50 y/o (90% >60 y/o), F > M, hx of PMR (50% of pts)

Physical Findings: Tenderness over temporal artery, decreased visual acuity, afferent pupillary defect
Evaluation: ↑ ESR, ↑ CRP, temporal artery biopsy

Management
- If visual deficits: IV methylprednisolone 1g daily × 3 days
- No visual deficits: Prednisone 60 mg/d (do not withhold pending biopsy results) & biopsy w/i 2 wk. Consult rheumatology, ophthalmology

Disposition: Admit only for visual deficits

Pearls
- Failure to diagnose & treat may result in permanent blindness
- 75% of pt w/ visual deficits in one eye will develop contralateral deficits w/i 3 wk
- 20× higher risk of thoracic aortic aneurysm

Optic Neuritis

Definition
- Inflammation of the optic nerve usually due to focal demyelination
- A/w MS (¼ pts will be diagnosed w/ MS), but also sarcoidosis, SLE, leukemia, alcoholism, syphilis, idiopathic, postviral

History: Vision loss (minimal → complete), ↓ color perception, pain w/ eye movement

Physical Findings
- ↓ Visual acuity, afferent pupillary defect, central scotoma, funduscopic exam
- Disk swelling/pallor

Evaluation: MRI shows inflammation of optic nerve, 20% have other demyelinating lesions
Treatment: Immediate ophthalmology/neurology consult, steroids
Disposition: Admit
Retinal Detachment

History
- Painless, classically “curtain-like” visual field deficit, “coal dust” or “spider webs,” floaters, photopsia (scintilla)
- RFs include myopia, trauma, surgical hx (cataract removal), DM, HTN, malignancy (breast CA, melanoma, leukemia), SCD, eclampsia, prematurity

Physical Findings: Visual field cut, “billowing” retina, may see pigmented vitreous or visible line demarcating detachment (usually by indirect ophthalmoscopy)

Evaluation: Bedside ED ocular U/S highly sens for detachment

Management
- Immediate optho consult if suspected
- If macula still attached, surgical repair indicated w/i 24–48 h
- Most inflammatory retinal detachments are treated medically (NSAIDs, steroids), but sometimes require emergent surgery depending on etiology, size, location

Disposition: Admit if acute

TOOTHACHE

<table>
<thead>
<tr>
<th>Toothache Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Atraumatic</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tooth Numbering</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper right 1, 2, 3, 4, 5, 6, 7, 8 (midline)</td>
</tr>
<tr>
<td>Lower right 32, 31, 30, 29, 28, 27, 26, 25 (midline)</td>
</tr>
<tr>
<td>Upper left (midline) 9, 10, 11, 12, 13, 14, 15, 16</td>
</tr>
<tr>
<td>Lower left (midline) 24, 23, 22, 21, 20, 19, 18, 17</td>
</tr>
</tbody>
</table>

Dental Fractures

Definition
- Ellis I: Enamel; Ellis II: Enamel + dentin; Ellis III: Involves pulp (+ bleeding)

**Evaluation:** Consider CXR in trauma pt for aspirated fragments

**Management**
- Dental blocks & oral analgesia
- Ellis I: Smooth sharp edges if needed, dental f/u in 2–3 d
- Ellis II: Cover w/ calcium hydroxide paste, zinc oxide paste, glass ionomer composites (pulp necrosis 1–7%), *dental f/u in 24 h*
- Ellis III: Cover w/ calcium hydroxide paste, zinc oxide paste, glass ionomer composites (pulp necrosis 10–30%), dental consult or urgent referral for pulpotomy/pulpectomy
- High risk infection. Rx abx
- Need urgent (<24 h) dental f/u
- If bleeding → gauze soaked in epinephrine, inject lidocaine w/ epinephrine into pulp

**Tooth Subluxation & Avulsion**
**Definition:** Loose teeth or loss of teeth due to trauma

**Evaluation:** X-ray if mobility suggests alveolar fracture

**Management**
- Dental blocks & oral analgesia
- Minimal mobility: Soft diet 1–2 wk, dental f/u in 2–3 d
- Grossly mobile: Stabilize w/ periodontal paste or splint, *dental f/u in 24 h*
- Avulsion: Only permanent teeth. Transport tooth in Hank’s solution or milk (preserves up to 8 h), do not clean tooth, replace tooth to socket w/ stabilization if w/i <60 min. 1% loss of tooth survival for every minute out. *Immediate dental consult w/ f/u in 24 h*

**Dental Caries**
**Definition:** Bacterial infection of hard tooth structure (enamel, dentin, & cementum)

**Presentation:** Tooth pain, poor dentition

**Management:** Dental block & oral analgesia, dental f/u in 1–2 d

**Periapical Abscess**
**Definition:** Bacterial infection of alveolar space

**Presentation:** Severe tooth pain, often fluctuant abscess

**Management:** Dental block. I&D if fluctuant. Abx (penicillin V or
clindamycin), warm saline rinses, dental f/u in 1–2 d.

**Acute Necrotizing Ulcerative Gingivitis (Trench Mouth)**

**Definition**
- Polymicrobial infection of gums causing bleeding, deep ulcers, & necrotic gums
- RFs: Poor oral hygiene, local trauma, smoking, immunodeficiencies

**Presentation**
- Rapid onset diffuse mouth pain, halitosis, fever, gum bleeding
- Gingival erythema/edema, interdental papillae ulceration, gray pseudomembrane

**Management:** Oral anesthetic solution (viscous lidocaine), dilute hydrogen peroxide rinses QID or chlorhexidine, abx if extensive or systemic (penicillin, clindamycin), *dental f/u in 1–2 d*

**Pearl:** Cx: Vincent angina–spread to pharynx & tonsils

**Alveolar Osteitis (Dry Socket)**

**Definition:** Irritation of bone exposed to the oral cavity after premature disintegration of blood clot 3–5 d after tooth extraction

**History:** Sudden onset, severe pain after dental extraction, foul odor/taste

**Management:** Dental block, oral analgesia, irrigate socket, pack with iodoform gauze soaked in medicated dental paste or eugenol. Abx (penicillin, clindamycin). *Dental f/u in 1–2 d.*
RESUSCITATION

Broselow Tape (equipment size & drug doses based on child length), Handtevy method

Airway: RSI (See 17-1)
- Pretreatment: Atropine (0.02 mg/kg, max 1 mg) prn bradycardia; lidocaine (1.5 mg/kg) prn if ↑ ICP
- Sedation: Etomidate (0.3 mg/kg); thiopental (3–5 mg/kg); ketamine (1–2 mg/kg)
- Paralysis: Succinylcholine (1–2 mg/kg); rocuronium (0.6–1.2 mg/kg)
- ETT size: 3 mm cuffed (newborns); (age/4 + 4) – 0.5 mm cuffed (>1 mo); depth (cm) = ETT size × 3
- Laryngoscope size: 0 (<2.5 kg); 1 (<3 yr); 2 (3–12 yr); 3 (12 yr to adult)

Shock
- nl SBP (mmHg) = 70 + (age in years × 2) b/w 1 & 10 yr
- Start w/ 20 cc/kg NS, up to 3 boluses
- Dopamine (2–20 μg/kg/min); epinephrine (0.05–1 μg/kg/min) for cold shock; norepinephrine (0.05–1 μg/kg/min) for warm shock; dobutamine (2–20 μg/kg/min) for cardiogenic shock
- Consider hydrocortisone if at risk for adrenal insufficiency
- In trauma, start w/ 20–40 mL/kg NS; then add 10–20 mL/kg PRBCs

ABDOMINAL PAIN

Approach
- Nature of pain: Location, constant or intermittent, relation to eating, associated sxs
- PMH: Previous abd surgeries, prematurity
- Exam: Always perform genital exam in males to r/o testicular torsion
- Labs: CBC, CRP, BMP, UA, LFTs, lipase if in the upper abdomen
<table>
<thead>
<tr>
<th>Location</th>
<th>Infancy</th>
<th>Childhood/Adolescence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanical</td>
<td>Malrotation w/ midgut volvulus, intussusception, hernia, Meckel diverticulum, Hirschsprung</td>
<td>Constipation, hernia, Meckel diverticulum, bowel obstruction (3a)</td>
</tr>
<tr>
<td>Inflammatory/infectious</td>
<td>NEC</td>
<td>Gastroenteritis, appendicitis, HSP, pancreatitis, gastritis, biliary tract dz (3a), colitis (3a), pancreatitis</td>
</tr>
<tr>
<td>GU</td>
<td>UTI (14bb)</td>
<td>UTI (14bb), renal colic (6b), pregnancy/ectopic (7), PID (7), testicular/ovarian torsion (7)</td>
</tr>
<tr>
<td>Other</td>
<td>Colic, trauma (abuse)</td>
<td>DKA (14r), trauma, sickle cell (14aa), toxic ingestions, PNA, strep pharyngitis</td>
</tr>
</tbody>
</table>

**APPENDICITIS**

**Definition**
- Inflammation of the appendix

**History**
- Diffuse/periumbilical pain → localizing to RLQ, anorexia, N/V, irritability (may be the only sx in age <2), fever

**Physical Findings**
- RLQ tenderness, rebound/guarding, Rovsing sign (RLQ pain w/ palpation in LLQ), psoas sign (RLQ pain w/ hip extension), obturator sign (RLQ pain w/ leg flexion + internal hip rotation)

**Evaluation**
- Labs: CBC, UA (sterile pyuria/mild hematuria), hCG
- Imaging: U/S (90% sens: Much lower if perforated/large habitus/operator dependent), abd plain films (fecalith 10%), CT scan (95% sens/spec)

**Treatment**
- Surgical consult for operative management, abx (ampicillin 50 mg/kg, gentamicin 1 mg/kg + metronidazole 15 mg/kg or cefoxitin 20–40 mg/kg)
**Disposition**
- Admit

**Pearls**
- 90% of children <2 y/o have perforation at presentation (thinner walled/looser omentum → ↑ perforation)
- Young children may not have anorexia

---

**INTUSSUSCEPTION**

**Definition**
- Invagination of bowel into another, most commonly ileocolic (most frequent cause of SBO in <6 y/o)

**History**
- Age 3 mo–3 yr (peak 5–9 mo), M > F, lethargy, vomiting, intermittent fussiness/crying/inconsolability w/ drawing legs to chest, cramping abd pain

**Physical Findings**
- Not tender b/w episodes, abd tenderness, RUQ sausage-like mass, heme + stool, “currant jelly” stool (late finding in <1/3 of pts)

**Evaluation**
- Upright plain abd film to r/o free air, crescent sign, U/S (95% sens/spec): Target, bull’s eye, doughnut, pseudokidney sign; barium/air/water enema: Diagnostic/therapeutic (90% successful)

**Treatment**
- Barium/air/water enema, NGT, surgical consult for operative management in case barium enema fails, hydration (severe dehydration is common), NPO

**Disposition**
- Admission for 24 h observation

**Pearls**
- <3 y/o likely idiopathic
- Barium enema is contraindicated if peritoneal signs
  - If >2 y/o, consider abnl lead point (tumor, Meckel’s, polyp)
MALROTATION WITH MIDGUT VOLVULUS

Definition
- Malrotation & weak fixation of the duodenum & colon during embryologic development → twisting of the mesentery causing duodenal obstruction/SMA compression → necrosis

History
- Neonate (3 y/o) acute abd pain, bilious vomiting, ±distension, irritability/lethargy, FTT, mostly occur w/i 1st year of life

Physical Findings
- Ill appearing/dehydration, heme + stool/grossly bloody, abd tenderness, often peritoneal

Evaluation

Treatment
- Immediate surgical consult for operative management, NGT, NPO, abx, fluids

Disposition
- Admission

INCARCERATED/STRANGULATED HERNIA

Definition
- Defects in the abd wall that allow protrusion of abd contents through the inguinal canal

History
- More commonly male, abd/groin/testicular pain, inguinal fullness w/ prolonged standing/coughing, vomiting, irritability in infants

Physical Findings
- Intestine/BS in scrotal sac

Evaluation
Scrotal/abd U/S if physical exam is unclear, x-ray can be used to r/o free air

**Treatment**
- Reduction: Place in Trendelenburg → gentle pressure ± ice analgesic/BZD; >12 h concern for perforation/gangrene → surgical management

**Disposition**
- Admission if operative management required

---

**MECKEL DIVERICULUM**

**Definition**
- Omphalomesenteric duct remnant w/ 60% containing heterotopic gastric (80%) or pancreatic tissue

**History**
- Any age (sxs usually begin <2 y/o), ±LLQ pain, melanotic stool (acid secretion → ulceration/erosion of mucosa), vomiting, sx of SBO, intussusception

**Physical Findings**
- LLQ mass, heme + stool/brisk bleeding, abd distension

**Evaluation**
- Technetium scan (Meckel scan): Identifies heterotopic gastric tissue (90% sens)

**Treatment**
- Type & cross/transfuse for brisk bleeding, surgical consult for Meckel diverticulectomy

**Disposition**
- Admit

<table>
<thead>
<tr>
<th>Meckel's Rule of 2s</th>
</tr>
</thead>
<tbody>
<tr>
<td>2% of the population</td>
</tr>
<tr>
<td>Only 2% of those w/ Meckel are symptomatic</td>
</tr>
<tr>
<td>2 in long</td>
</tr>
</tbody>
</table>
Necrotizing Enterocolitis (NEC)

**Definition**
- Inflammatory condition of intestinal wall due to bacterial overgrowth w/ translocation

**History**
- Preterm neonate (90%), age < 1 mo (usually first days of life), bilious vomiting, abd distension, bloody stool, feeding intolerance

**Physical Findings**
- Ill appearing, hypotension, lethargic, abd tenderness, heme + stools, diarrhea

**Evaluation**
- Abd x-ray: Pneumatosis intestinalis (75%), portal venous air; barium enema if x-ray is ambiguous

**Treatment**
- NPO, hydration/transfusion, NGT, abx (ampicillin/gentamicin/metronidazole), surgical consult

**Disposition**
- Admit

**Pearls**
- Bell stages: I. Vomiting/ileus, II. Intestinal dilation/pneumatosis on x-ray, III. Shock/perforation
- Cx: DIC, strictures, obstruction, fistulas, short gut syndrome

**Hirschsprung Disease**

**Definition**
- Absence of ganglion cells in the myenteric plexus of the colon → constant contraction & proximal dilation → constipation, obstruction
(4:1 male predominance)

**History**
- Chronic constipation, delayed 1st meconium, FTT, abd distension, vomiting

**Physical Findings**
- Sx in 1st days to weeks of life, palpable stool in abdomen, tight sphincter, fecal mass in LLQ, no stool in rectal vault, “squirt” – explosive release of stool when finger is withdrawn

**Evaluation**
- Abd plain film: Dilated colon/fecal impaction/air fluid levels; barium enema; Dx → biopsy (aganglionosis) or anal manometry

**Treatment**
- Outpt surgical eval

**Disposition**
- D/c unless cx: Toxic mega colon, perforation, enterocolitis

---

### Cyanosis

**Approach**
- Differentiate cyanosis that is central (mucous membranes, tongue, trunk, 2/2 right-to-left shunt) vs. peripheral (feet, hands, lips, 2/2 peripheral vasoconstriction)

**Definition**
- Acrocyanosis: Blueness in hands/feet only seen in newborns, 2° perfusion of the extremities → nl & resolves w/i 1st few days of life
- Breath-holding spell: Prolonged period w/o attempt to breathe a/w intense crying from pain, anger, fright → benign, but Dx of exclusion

<table>
<thead>
<tr>
<th>Cyanosis Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Hypoventilation</td>
</tr>
<tr>
<td>Respiratory</td>
</tr>
</tbody>
</table>
Cardiovascular | Cyanotic congenital cardiac dz
---|---
Other | Sepsis, hypothermia, methemoglobinemia, CN−, acrocyanosis of the newborn

**History**
- Age of onset, central or peripheral, med ingestion, recent illness, environmental exposures
- Δ w/ crying: Improvement → respiratory etiology (↑ alveolar recruitment); exacerbation → cardiac etiology (↑ CO)

**Findings**
- Appearance (ill or well), VS, respiratory distress, heart murmur

**Evaluation**
- Provide O₂, obtain CXR, ECG
- Hyperoxygenation test: Compare ABG on RA on 100% O₂ for 10 min, P O₂ of >250 excludes hypoxia 2/2 congenital heart dz
- Improvement in O₂ sat w/ O₂, lack of murmur, nl ECG → pulmonary process
- No Δ in O₂ sat w/ O₂, murmur, abnl ECG → cardiac cause → obtain echo (see 14-19)

**Treatment**
- O₂, identify then tx underlying condition
- Consider PGE₁ for pts <2 wk of age in circulatory failure

**Disposition**
- Admit any pt who is ill appearing, low O₂ sat or PaO₂
- Consult cardiology for any pt w/ suspected congenital cardiac dz

---

**PEDIATRIC FEVER**

**Approach**
- Fever (38°C or 100.4°F) management is different in pediatric population compared to adults
- ABCs, check O₂ saturation, rectal temperature
- Need for abx & hospitalization depends on age, tox, exposures, immune status, identified source, seriousness of source
Introduction of *H. influenzae* & pneumococcal vaccines have changed the incidence & etiology of febrile illness in pediatric populations

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary</td>
<td>Bronchiolitis, croup, pertussis, pharyngitis, PNA</td>
</tr>
<tr>
<td>GI</td>
<td>Appendicitis, gastroenteritis, rotavirus</td>
</tr>
<tr>
<td>GU</td>
<td>UTI, pyelonephritis</td>
</tr>
<tr>
<td>Noninfectious</td>
<td>SCD, Kawasaki’s Dz, rheumatologic &amp; oncologic etiologies</td>
</tr>
<tr>
<td>Misc infections</td>
<td>Cellulitis, HIV, sepsis, varicella, epiglottitis, measles, meningitis, mumps, OM, omphalitis, roseola, rubella, scarlet fever, osteomyelitis, HSV, enterovirus, bacterial conjunctivitis, nonsp viral syndromes</td>
</tr>
</tbody>
</table>

### FEBRILE INFANT 0–90 D OLD

#### History
- Difficult to obtain localizing hx; standardized w/u to Dx serious bacterial illnesses, high-risk 2/2 immature immune sz
- Exposures (travel, ill family members) & immunizations are helpful

#### Findings
- Fever >38°C or 100.4°F rectal considered standard; fussy, irritable, poor feeding
- Assess frequency & # of wet diapers, cap refill, fontanelles, tears, to estimate dehydration
- Ask about any rashes (viral exanthems, meningococcus)

#### Evaluation
- Sepsis w/u: See table

#### Treatment
- Less than 1 mo: Cefotaxime 50 mg/kg IV q12h + ampicillin 25–50 mg/kg IV q8h
- 1–3 mo: Ceftriaxone 50 mg/kg IV q24h, consider IM ceftriaxone 50 mg/kg if being D/C
- Higher doses for suspected meningitis, consider adding acyclovir 20 mg/kg IV (see 14-16)
Treat other identified bacterial source appropriately
If LP was not performed, consider withholding abx in well-appearing infant w/ nl WBC

Disposition
If <30 d or <90 d & toxic appearing, admit & follow cx even if all labs nl
Can d/c 30–90 d w/ neg. sepsis w/u & well appearing/feeding w/ f/u in 24 h. Consider 1 dose ceftriaxone prior to d/c

Pearl
Due to inability to localize source of infection, relative immaturity of immune systems, & prevalence of occult bacteremia, all pts receive extensive sepsis w/u

---

**Febrile Child 3–36 mo**

**History**
- Vulnerable immune system, esp to encapsulated organisms’ exposures
- Exposures (travel, ill family members) & immunizations helpful

**Findings**
- Irritable, poor feeding; elicit hydration status via # of wet diapers, tears, fontanelle, cap refill
- Ask about any rashes (viral exanthems, meningococcus)

**Evaluation**
- See table

**Treatment**
- If ill appearing w/ fever, 1 dose ceftriaxone (50 mg/kg IV & 24-h admission for cx)
- Treat identified bacterial source appropriately

**Disposition**
- If well appearing w/ neg. w/u & fully immunized, d/c home w/ close f/u
- If well appearing w/neg. w/u & incomplete immunization:
  - WBC >15K (ANC >9000), give empiric abx (ceftriaxone IV or IM) & 24 h f/u or admit if f/u uncertain
  - WBC <15K (ANC <9000), d/c w/o abx, but close f/u in 24–48 h

**Pearl**
Prevalence of occult bacteremia in well-appearing children <36 mo is now 0.25–0.4% \((\text{Acad Emerg Med 2009;16(3):220; Arch Dis Child 2009;94(2):144})\)

### EVAL of Pediatric Fever by Age

<table>
<thead>
<tr>
<th>Age</th>
<th>Temp</th>
<th>Appearance</th>
<th>Eval</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–90 d</td>
<td>&gt;38°C</td>
<td>Any</td>
<td>Straight cath urine &amp; culture</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CBC w/ differential, blood culture, CRP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CXR if ↑ RR, respiratory sxs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CSF culture, cell count, glucose/protein, ±HSV/enterovirus PCR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stool culture if diarrhea is present</td>
</tr>
<tr>
<td>3–36 mo</td>
<td>&lt;39°C</td>
<td>Any</td>
<td>UA &amp; Ucx</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CXR if ↑ RR, resp sxs</td>
</tr>
<tr>
<td>&gt;39°C</td>
<td>Well</td>
<td></td>
<td>UA &amp; Ucx</td>
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<td></td>
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<td></td>
<td>CBC w/ differential, blood culture, CRP</td>
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<td></td>
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<td></td>
<td>CXR if ↑ RR, resp sxs</td>
</tr>
<tr>
<td>Ill</td>
<td></td>
<td></td>
<td>UA &amp; Ucx</td>
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<td></td>
<td>CBC w/ differential, blood culture, CRP</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>If neg., LP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CXR if ↑ RR, resp sxs</td>
</tr>
</tbody>
</table>

**JAUNDICE**

**Definition**

- Yellowish discoloration of the skin/tissue/body fluids caused by ↑ bilirubin production or ↓ excretion

**Approach**

- Bilirubin: Formed from degradation of hemoglobin → bound to albumin in blood (unconjugated/indirect) → conjugated in liver by glucuronyl transferase (conjugated/direct) → excreted in bile

**History**

- Differential depends on age (neonates ≤4 wk), gestational age, breast-feeding status
- Time of onset of sx: Yellowing of skin, dark urine

**Physical Findings**
Scleral icterus, jaundice

Labs
- Total/fractionated bilirubin (visible >5 mg/dL in neonates), LFTs, CBC (hemolysis/anemia → Coombs test, smear, ABO/Rh type), reticulocyte count, serum haptoglobin
- Neonates → unconjugated (can be physiologic, treat to prevent kernicterus)/ conjugated (always pathologic)

<table>
<thead>
<tr>
<th>Differential Dx of Unconjugated Hyperbilirubinemia in Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemolytic disorders</td>
</tr>
<tr>
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<td></td>
</tr>
<tr>
<td>Enterohepatic recirculation</td>
</tr>
<tr>
<td>Other</td>
</tr>
</tbody>
</table>

**Physiologic Jaundice**

Definition
- Elevated unconjugated bilirubin in the 1st wk of life, 60% newborns will be jaundiced (peaks 2–5 d), due to low activity of glucuronyl transferase, increased production, & increased enterohepatic circulation

Evaluation
- Total/fractionated bilirubin, CBC (hemolysis/anemia → Coombs test, smear, ABO/Rh type), total bilirubin usually <6 mg/dL, up to 12 mg/dL in premature infants

Treatment
- No tx necessary
**Disposition**
- Home

**Pearls**
- Pathologic: In the 1st 24 h of life, peak >17 mg/dL in breast-fed/ >15 mg/dL in formula-fed infants, persists beyond 1st wk of life, ↑ bilirubin >5 mg/dL/d
- Cx of severe hyperbilirubinemia: kernicterus (bilirubin deposition in basal ganglia → neurodevelopmental deficits)
- Sepsis can rarely present as jaundice

---

**BREAST-FEEDING JAUNDICE**

**Definition**
- ↑ unconjugated bilirubinemia in breast-fed infants possibly due to hormonal mediators or altered intestinal secretion/absorption of bile, early onset after birth
  - May be related to caloric deprivation or insufficient frequency of feeding

**Evaluation**
- Total/fractionated bilirubin, CBC

**Treatment**
- No tx necessary if bilirubin <17 mg/dL, encourage breast feeding, phototherapy

**Disposition**
- Home

---

**BREAST MILK JAUNDICE**

**Definition**
- Due to substances in breast milk that prevent conjugation & excretion of bilirubin. Occurs after 3–5 d of life, persists for weeks.

**Evaluation**
- Total/fractionated bilirubin, CBC

**Treatment**
If bilirubin <17 mg/dL, continue breast feeding, phototherapy
If >17 mg/dL, stop breast feeding, will not recur when resumed

Disposition
› Home

ABO and Rh Incompatibility/Hemolytic Disease

Definition
› Hemolytic dz caused by maternal antibodies against fetal A or B type proteins or maternal Rh antibodies (sensitized from previous pregnancy) against Rh-positive fetus (Rh incompatibility)

History
› Yellowing of skin w/i 1st 24 h of life, dark urine, lethargy

Physical Findings
› Severe jaundice, scleral icterus, ill appearing

Evaluation
› Total/fractionated bilirubin, CBC (hemolysis/anemia → Coombs test, smear, ABO/Rh type)

Treatment
› Phototherapy, exchange transfusion (see table)

Disposition
› Admit

<table>
<thead>
<tr>
<th>Age</th>
<th>Consider Phototherapy (mg/dL)</th>
<th>Phototherapy (mg/dL)</th>
<th>Consider Exchange Transfusion if Phototherapy Fails (mg/dL)</th>
<th>Exchange Transfusion (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤24 h</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>25–48 h</td>
<td>≥12</td>
<td>≥15</td>
<td>≥20</td>
<td>≥25</td>
</tr>
<tr>
<td>49–72 h</td>
<td>≥15</td>
<td>≥18</td>
<td>≥25</td>
<td>≥30</td>
</tr>
</tbody>
</table>
**Conjugated Hyperbilirubinemia**

**Definition**
- Pathologic increase in direct bilirubin leading to jaundice (conjugated bilirubin >20% of total, or >2 mg/dL)

**History**
- Yellowing of skin, dark urine, lethargy, ± genetic syndrome/metabolic syndromes/sepsis

**Physical Findings**
- Severe jaundice, scleral icterus, ill appearing

**Evaluation**
- Total/fractionated bilirubin, CBC, blood cultures, blood smear, LFTs, blood type, KUB if signs of obstruction, U/S: Biliary obstruction, UA, Ucx

**Treatment**
- Hydration, tx based on cause (see below)

**Disposition**
- Admit

<table>
<thead>
<tr>
<th>Differential Dx of Conjugated Hyperbilirubinemia in Children</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biliary obstruction</strong></td>
</tr>
<tr>
<td>Choledochal cyst</td>
</tr>
<tr>
<td>1° sclerosing cholangitis</td>
</tr>
<tr>
<td>Gallstone (usually pigmented stone from hemolysis in sickle cell/thalassemia)</td>
</tr>
<tr>
<td><strong>Infections</strong></td>
</tr>
<tr>
<td>Bacterial sepsis</td>
</tr>
<tr>
<td>UTI</td>
</tr>
<tr>
<td>Viral hepatitis</td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
</tr>
<tr>
<td>Galactosemia</td>
</tr>
</tbody>
</table>
**LIMP**

**Approach**
- Examine abdomen, genitalia, spine, hips, long bones, knees, ankle, feet; observe gait
- Careful hx from pt & care giver: Acute vs. chronic, fevers, skin Δ; trauma
- Obtain x-rays although pain is often referred (classically, knee pain referred from hip)
- Consider systemic sxs in conjunction w/ chief complaint of joint pain

<table>
<thead>
<tr>
<th>Limp Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathophysiology</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Hematologic</td>
</tr>
<tr>
<td>Neuromuscular</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td>Rheumatologic</td>
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<tr>
<td>GI/GU</td>
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<tr>
<td>Musculoskeletal</td>
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<tr>
<td>Neoplastic</td>
</tr>
</tbody>
</table>
INFECTIOUS

Septic Arthritis of the Hip

History
- Most commonly in children <3 y/o, but can occur at any age
- Limp or refuse to walk, hx of fever & irritability (sxs may be far more subtle in infants)

Findings
- Febrile & toxic appearing
- Flexed, externally rotated, abducted hip; antalgic gait (if walking)
- Significant pain w/ ROM but not necessarily warm, swollen or erythematous

Evaluation
- ↑ WBC, ↑ CRP, ↑ ESR; arthrocentesis shows ↑ WBC, +Gram stain & culture
- X-rays & U/S may show effusion

Treatment
- Orthopedic consultation for drainage & washout in the OR
- Abx: β-lactamase–resistant PCN (IV nafcillin or oxacillin 50–100 mg/kg/d QID) & 3rd-generation cephalosporin (cefotaxime or ceftriaxone 50 mg/kg); consider vancomycin
- Pain control

Disposition
- Admit for surgical wash-out

Pearl
- Hip > knee > elbow likely to be septic in children

Toxic (Transient) Synovitis

History
- 3–6 y/o, M:F 2:1, acute or chronic unilateral hip, thigh, or knee pain
- May be mildly febrile, possibly recent URI

Findings
- Nontoxic appearing
- Limited hip ROM 2/2 pain; mild restriction of passive ROM to internal rotation & extension; most sens to log roll
Antalgic gait, painful to palpation

Evaluation
- X-ray of hip nl; may show effusion
- WBC & ESR nl or slightly ↑; afebrile children w/ nl labs can avoid arthrocentesis
- U/S can diagnose effusion, but cannot differentiate type

Treatment
- Pain control w/ NSAIDs, heat, & massage

Disposition
- Orthopedic f/u, crutches to keep weight off hip until pain resolves

Pearls
- Most common cause of acute hip pain in children from 3–10 yr; arthralgia & arthritis secondary to transient inflammation of the synovium of the hip
- Recurrence rate <20%, most develop w/i 6 mo, no ↑ risk for juvenile chronic arthritis, may go on to develop Legg–Calvé–Perthes dx

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>16.9</td>
<td>0.2</td>
</tr>
<tr>
<td>1</td>
<td>36.7</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>62.4</td>
<td>40</td>
</tr>
<tr>
<td>3</td>
<td>82.6</td>
<td>93.1</td>
</tr>
<tr>
<td>4</td>
<td>93.1</td>
<td>99.6</td>
</tr>
<tr>
<td>5</td>
<td>97.5</td>
<td></td>
</tr>
</tbody>
</table>

Factors: Temp >38.5°C, WBC >12, ESR >40, refusal to bear weight ± CRP >20 (if using modified Kocher criteria)

**MUSCULOSKELETAL**

Legg–Calvé–Perthes Disease (<i>Avascular Necrosis of Femoral Head</i>)

History
- Most commonly in 5–7 y/o w/ limp & pain in groin, thigh, or knee; worse
w/ ↑ activity
- No fever or irritability, no hx of trauma

Findings
- Nontoxic appearing, antalgic gait
- ↓ Hip ROM secondary to pain w/ possible thigh atrophy, ↑ w/ internal rotation & abduction

Evaluation
- WBC & ESR nl
- X-rays often nl initially; frog-leg views helpful
  - Widening of cartilage space, diminished ossific nucleus
  - Subchondral stress fx of femoral head; linear lucency in femoral head epiphysis
  - Femoral head opacification & flattening known as coxa plana
  - Subluxation & protrusion of femoral head from acetabulum

Treatment
- Goal is to avoid severe degenerative arthritis, maintain ROM, relieve weight bearing
- Orthopedic eval; bone scan & MRI more rapidly diagnostic than x-rays

Disposition
- Orthopedic f/u, crutches to keep weight off hip until pain resolves

Pearls
- Idiopathic osteonecrosis of capital epiphysis of femoral head; 15–20% bilateral
- Caused by interruption of blood supply to capital femoral head → bone infarction
- Better prognosis at younger onset; proportional to degree of radiologic involvement

Slipped Capital Femoral Epiphysis (SCFE)

History
- 12–15-y/o boy or 10–13-y/o girl, c/o limp & groin, thigh, or knee pain
- If sxs >3 wk, considered chronic
- If unable to bear weight, considered unstable (higher cx rate)

Findings
- Affected leg externally rotated, shortened w/ pain when flexing hip; antalgic gain
Evaluation
- nl temp, WBC, ESR
- X-ray: Femoral head is displaced posteriorly & inferiorly in relation to femoral neck w/i confines of acetabulum; AP & frog-leg views best

Treatment
- Orthopedic consult for operative internal fixation; goal to prevent AVN of femoral head

Disposition
- Admission for orthopedic surgery

Pearls
- Obesity is the RF; genetics play role; bilaterality more common in younger pts who also tend to have metabolic/endocrine disorders
- If traumatic hip injury w/ obvious external rotation & shortening of the leg, do not force ROM as this can worsen epiphyseal displacement

Osgood–Schlatter Disease

Definition
- Microtrauma to the tibial tubercle tuberosity apophysis occurring during use

History
- Preteen boy w/ knee pain, worse w/ activity & better w/ rest

Findings
- Edema & pain of tibial tubercle; enlarged & indurated tibial tuberosity
- Tender over anterior knee, esp over thickened patellar tendon
- Pain reproduced by extending knee against resistance, stressing quads or squatting w/ knee in full flexion, running, jumping, kneeling, squatting, stairs

Evaluation
- Clinical dx. X-ray may show swelling over tuberosity & patellar tendon; no effusion

Treatment
- Guided by severity: Range from decreasing activity in mild cases to rest in severe cases
- NSAIDs for pain control, ice, ±crutches

Disposition
D/c home w/ pain control

Pearls
- One of the most common causes of knee pain in adolescent; benign & self-limited
- Bilateral in 25% of cases; 50% give hx of precipitating trauma

Pediatric Seizure

Definition
- Abn, paroxysmal d/c of CNS neurons leading to abn neurologic fxn

Approach
- ABCs, check O₂ saturation, temperature, determine if still seizing
- Immediate bedside glucose fingerstick & tx, consider administering empiric glucose
- If actively seizing, quickly administer suppression medications
- Careful hx: Description of events before & after sz, associated sxs (HA, photophobia, vomiting, visual Δ, ocular pain), focal neurologic sxs
- Assess for head or neck trauma, meningismus, skin finding (petechiae, café-au-lait spots, port-wine stain, ash leaf spots), ↑ICP (bulging fontanelle)
- Thorough neurologic exam; Todd paralysis: Transient paralysis after a sz
- CBC, CMP, tox screen, UA, CXR: Tox screen, anticonvulsant levels, infectious w/u
- Consider CT if persistent AMS, neurologic deficit, or trauma
- Consider LP after head CT if persistently AMS, fever, & therapeutic med levels
- 1st-time sz w/u: Consider head CT, ECG, CBC, CMP, tox screen, LP
- EEG days to weeks after sz unless concern for nonconvulsant status epilepticus
- Status epilepticus is recurrent or continuous sz activity lasting >30 min w/o return to baseline MS
- Can result in cerebral hypoxia, lactic & respiratory acidosis, hypercarbia, hypoglycemia
- Disposition: Admission for abnl neuro exam, others w/ neurology f/u
### Sz Differential

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neurologic</strong></td>
<td>1° sz, status epilepticus, febrile, sz degenerative CNS dz (neurofibromatosis, tuberous sclerosis, Sturge–Weber syndrome), epilepsy, cerebral palsy</td>
</tr>
<tr>
<td><strong>Head injury</strong></td>
<td>IPH, SAH, SDH, epidural (19b)</td>
</tr>
<tr>
<td><strong>Infection</strong></td>
<td>Meningitis (5d, 14i), encephalitis (5d), brain abscess, toxoplasmosis, tetanus, neurocysticercosis</td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
<td>Hypoglycemia, hyperglycemia, hyponatremia, hypernatremia, hypocalcemia, hypomagnesemia, alkalosis (5e), pyridoxine deficiency</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Toxic</strong></td>
<td>Lead, PCP, amphetamine, cocaine, aspirin, CO, organophosphates, theophylline, lidocaine, lindane, drug withdrawal (anticonvulsants), s/p DPT immunization</td>
</tr>
<tr>
<td><strong>Neoplasm</strong></td>
<td>Brain tumor</td>
</tr>
<tr>
<td><strong>Pediatric</strong></td>
<td>Reye syndrome, CMV, congenital syphilis, maternal rubella, PKU</td>
</tr>
<tr>
<td><strong>Vascular</strong></td>
<td>Embolism, infarction, HTN encephalopathy, malformations</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Psychological, hyperventilation, breath-hold spells, inadequate drug level, neurocutaneous syndromes, inborn errors of metabolism</td>
</tr>
</tbody>
</table>

### Primary Seizures

**History**
- Presence/absence of aura, abrupt onset & termination of sz activity, stereotyped purposeless behavior, fecal or urinary incontinence, postictal confusion or lethargy

**Findings**
- Depends on type of sz, LOC secondary to simultaneous activation of entire cerebral cortex

**Evaluation**
- As above

**Treatment**
- Acute vs. chronic meds, airway mgmt often w/ only nasal trumpet, supplemental O₂
- Abortive tx
- BZD are 1st line (lorazepam 0.1 mg/kg up to 4 mg IV)
- BZD: Diazepam \(t_{1/2} 15-20\) min, lorazepam \(t_{1/2} 12-24\) h, midazolam \(t_{1/2} <12\) h
- 2nd-line: fosphenytoin (20 PE/kg IV)
- Phenobarbital (20 mg/kg IV load), 1st line in neonates, watch for hypotension & bradypnea
- If refractory szs, give pyridoxine 100 mg IV; consider thiamine 100 mg IV in adolescents

### Long-term Anticonvulsant Medications
- If known sz disorder & subtherapeutic levels, load w/ chronic med
- Long-term anticonvulsants not routinely indicated in 1st unprovoked sz

### Disposition
- Explicit instructions to not drive, operate hazardous machinery, or perform tasks where recurrent sz may cause harm; some states have mandatory reporting to department of motor vehicles

### Pearls
- Keep differential broad even if known sz d/o, esp if tx med levels
- If meningitis suspected, give abx pre-emptively while awaiting confirmation
- Pseudosz is Dx of exclusion
- Tx EtOH withdrawal sz w/ BZD, almost never responsive to antiepileptic meds
- Consider Neurology consult if starting new long-term med in 1st-time sz (will need close f/u)

### Pediatric Sz Suppression Steps

<table>
<thead>
<tr>
<th>Tx of Siz</th>
<th>Antiepileptic</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lorazepam (slower)</td>
<td>0.1 mg/kg IV or prn, repeat 0.05 mg/kg q5min</td>
</tr>
<tr>
<td>2 (&gt;30 min)</td>
<td>Phenobarbital (consider intubating)</td>
<td>20 mg/kg (&lt;20 kg) or 10 mg/kg (&gt;20 kg) IV</td>
</tr>
<tr>
<td></td>
<td>Phenytoin</td>
<td>20 mg/kg IV at 1 mg/kg/min</td>
</tr>
<tr>
<td></td>
<td>Fosphenytoin</td>
<td>20 mg PE/kg at 3 mg PE/kg/min</td>
</tr>
<tr>
<td></td>
<td>Levetiracetam</td>
<td>20 mg/kg IV</td>
</tr>
<tr>
<td>3 (&gt;1 h)</td>
<td>Pentobarbital, midazolam, valproic acid, propofol infusions, general anesthesia</td>
<td></td>
</tr>
</tbody>
</table>
Epilepsy

History
- Typical sz recurrence, may be a/w lip biting, incontinence of bowel or bladder followed by lethargy/combativeness & confusion (postictal period)

Findings
- Depends on type of sz, LOC secondary to simultaneous activation of entire cerebral cortex

Evaluation
- As above

Treatment
- Acute vs. chronic meds, airway mgmt often w/ only nasal trumpet, supplemental O₂

Disposition
- Neurology f/u for medication adjustment if indicated

Pearls
- Keep differential broad even if known sz d/o, esp if tx med levels
- Systemic illness such as URI or fever can lower sz threshold

Cerebral Palsy

History
- Nonprogressive lesion sustained during brain development → motor, speech, & learning disabilities, high risk (50%) for szs. Prematurity is the biggest RF

Findings
- Depends on type of CP:
  I. Quadriplegia: Hypotonic trunk & spastic extremities
  II. Diplegia: Spastic lower extremities, ↑ DTRs, clonus, & “scissoring”
  III. Hemiplegia: Unilateral spasticity, usually UE > LE
  IV. Athetoid: Writhing, involuntary movements of extremities
  V. Ataxic: Unsteady, uncoordinated movements
  VI. Hypotonic: Lacking muscle tone

Evaluation
- Head CT if new onset sz or recent trauma
- Outpt EEG if new onset sz or Δ in sz pattern or frequency
Treatment
› Standard sz tx

Disposition
› Neurology f/u for medication adjustment if indicated

Pearls
› Pts w/ CP often have breakthrough sz & low sz thresholds, look for underlying illness (URI, PNA, UTI, etc.), adjust outpt meds w/ 1° neurologist
› CP pts also commonly present to the ED w/ chronic aspiration, PNA, feeding difficulties, G-tube malfunction, UTIs

<table>
<thead>
<tr>
<th>Pediatric Sz Types</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sț Type</td>
</tr>
<tr>
<td>Generalized absence</td>
</tr>
<tr>
<td>Generalized tonic–clonic</td>
</tr>
<tr>
<td>Myoclonic</td>
</tr>
<tr>
<td>Simple partial</td>
</tr>
<tr>
<td>Complex partial</td>
</tr>
<tr>
<td>Somatosensory</td>
</tr>
<tr>
<td>Autonomic</td>
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<tr>
<td>Psychomotor</td>
</tr>
</tbody>
</table>

Febrile Seizures

History
› T ≥38.3°C (101°F) in child b/w 6 mo & 5 yr of age
› No hx of sz; 1 generalized sz lasting <15 min a/w rapidly ↑ temp

Findings
› Generalized sz activity, usually lasts <15 min; high fever, postictal period
› Complex febrile sz: Last >15 min, >1× in 24-h period, or focal component

Evaluation
- Evaluate for underlying (infectious) cause: CXR, UA, labs, bedside glucose, ±LP

**Treatment**
- Antipyretic, observation until pt back to baseline, parental reassurance
- Anticonvulsants like BZD & phenobarbital are not indicated

**Disposition**
- 1st febrile sz, nonfocal exam, neg. ED w/u can be D/C w/ neuro f/u

**Pearls**
- Focal sz do not present as simple febrile sz
  - Consider meningitis/encephalitis in unvaccinated children
- Febrile sz not a/w an epilepsy or brain damage
- Incidence of another febrile sz is 35
- >2 febrile sz/yr or >3 total febrile sz must be evaluated for other etiologies

**NAUSEA AND VOMITING**

**Approach**
- Common sxs of many dz processes (eg, intra-abd causes, metabolic derangements, toxic ingestions, neurologic causes)

**History**
- Relation to eating, bilious (require eval for obstruction), ability to tolerate POs, urine output (making wet diapers), presence of bloody stools, HA, AMS

**Labs**
- BMP, serum glucose (↑ risk of hypoglycemia)

**Treatment**
- Treat under lying cause, antiemetics, hydration (PO or IV)

<table>
<thead>
<tr>
<th>Location</th>
<th>Infancy</th>
<th>Childhood/Adolescence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanical</td>
<td>GERD, malrotation w/ midgut volvulus (14a), intussusception (14a)</td>
<td>Constipation, hernia (14a), Meckel diverticulum (14a), bowel obstruction (3a)</td>
</tr>
<tr>
<td>Category</td>
<td>Conditions</td>
<td>Conditions</td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Inflammatory/ infectious</td>
<td>NEC (14a), gastroenteritis, sepsis (14j), meningitis (14i), PNA, OM</td>
<td>Gastroenteritis, OM, appendicitis (14a), pancreatitis (14a), HSP (14a), biliary dz (3a)</td>
</tr>
<tr>
<td>GU</td>
<td>UTI (14bb)</td>
<td>UTI (14bb), renal colic (6b), pregnancy/ectopic (7), PID (7), testicular/ovarian torsion (7)</td>
</tr>
<tr>
<td>CNS (persistent vomiting w/o systemic/GI sx)</td>
<td>Hydrocephalus, intracranial injury/tumor (18b)</td>
<td>Hydrocephalus, intracranial injury/tumor (18b), migraine (5d)</td>
</tr>
<tr>
<td>Metabolic</td>
<td>DKA (14r), urea cycle defects, fatty acid oxidation disorders, amino acidopathies, organic acidurias</td>
<td>DKA (14r), urea cycle defects, fatty acid oxidation disorders, RTA, adrenal insuf</td>
</tr>
<tr>
<td>Other</td>
<td>Toxic ingestions, trauma, Reye syndrome</td>
<td>Trauma, sickle cell (14aa), toxic ingestions</td>
</tr>
</tbody>
</table>

**Pyloric Stenosis**

**Definition**
- Hypertrophy of the antrum of the stomach, 5:1 male-to-female ratio

**History**
- 2–5 wk of age (rare after 3 mo), nl feeding after birth → nonbilious/+ blood streaked projectile vomiting after feeding, weight loss, lethargy

**Physical Findings**
- RUQ olive-size mass, dehydration (loose skin, sunken eyes, dry mucous membranes)

**Evaluation**
- BMP (hyperchloremic metabolic alkalosis), U/S (+ pylorus >4 mm thick, >16 mm long, 95% sens, study of choice), upper GI series: “String sign,” abd x-ray: Dilated stomach

**Treatment**
- Hydration, surgical consult for pyloromyotomy

**Disposition**
- Admit
**GASTROESOPHAGEAL REFLUX DISEASE**

**Definition**
- Loose esophageal sphincter → retrograde passage of food into esophagus

**History**
- <2 y/o, nonbilious vomiting/spitting during/after eating, type of formula (cow vs. soy)

**Physical Findings**
- Sandifer syndrome: Startled/jerky movements after eating, often confused for sz

**Evaluation**
- Outpt w/u: 24 pH probe (most sens), nuclear milk scan, barium swallow, heme + stools (if esophagitis present), bloody diarrhea can indicate formula allergy

**Treatment**
- Small feeding volumes w/ burp breaks, keep semiupright for 30–40 min after eating, thicken feeds by adding cereal
- Acid-reducing agents: Ranitidine 2–4 mg/kg/d divided q8h, PPI, metoclopramide 0.1–0.2 mg/kg q12h

**Disposition**
- Home

**Pearls**
- Cx: FTT, apnea, laryngospasm, esophagitis, PNA
- Usually resolves by 1 yr

---

**GASTROENTERITIS**

**Definition**
- Vomiting & diarrhea caused by infectious source

**History**
- Vomiting, diarrhea, sick contacts, recent abx, travel

**Physical Findings**
Lethargy, dehydration (skin turgor, cap refill, mucus membranes, tears, VS)

Evaluation
- BMP (if severely dehydrated), stool culture/ova/parasites (protracted/bloody diarrhea)

Treatment
- Electrolyte correction, hydration (PO preferred, IV prn), most self-resolve, avoid antimotility agents (↑ pain/cramping/prolonged sx)

Disposition
- Home or admit (severe dehydration, bicarb <16 mEq/L, inability to tolerate POs)

<table>
<thead>
<tr>
<th>Etiology-sp Sxs &amp; tx of Gastroenteritis</th>
<th>Agents</th>
<th>Classic Hx &amp; Findings</th>
<th>ED Intervention</th>
<th>Clinical Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viral</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Rotavirus</td>
<td></td>
<td>Watery diarrhea, in fall (Southwest)/winter (Northeast) months; common among children attending daycare or preschool</td>
<td>Hydration</td>
<td>~70% of children under age 2 yr admitted for diarrhea dehydration are infected w/ rotavirus; very infectious</td>
</tr>
<tr>
<td>Adenovirus</td>
<td></td>
<td>Watery diarrhea w/ concurrent respiratory illness usually in spring or early summer</td>
<td>Hydration</td>
<td></td>
</tr>
<tr>
<td>Norwalk virus</td>
<td></td>
<td>Watery diarrhea w/ fever, HA, &amp; myalgia</td>
<td>Hydration</td>
<td>Major cause of diarrhea epidemics</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Agents</th>
<th>Classic Hx &amp; Findings</th>
<th>ED Intervention</th>
<th>Clinical Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. jejuni</td>
<td>Watery or bloody diarrhea w/ fever &amp; crampy abd pain</td>
<td>Hydration &amp; azithromycin, erythromycin, or ciprofloxacin</td>
<td>Contracted through contaminated food or water</td>
</tr>
<tr>
<td>Bacterial Pathogen</td>
<td>Clinical Presentation</td>
<td>Management</td>
<td>Remarks</td>
</tr>
<tr>
<td>--------------------------</td>
<td>---------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Shigella</strong></td>
<td>Diarrhea possibly w/ blood/mucus/pus a/w fever, HA, &amp; abd pain</td>
<td>Hydration; fluoroquinolones, Bactrim, ampicillin, or azithromycin</td>
<td>Contracted through contaminated food or water; increasing abx resistance</td>
</tr>
<tr>
<td><strong>Salmonella</strong></td>
<td>Bloody diarrhea w/ fever</td>
<td>Hydration; ciprofloxacin, azithromycin, ampicillin, or Bactrim</td>
<td>Abx can induce a carrier state; Treat only if risk of invasive dz (&lt;3 mo of age, sickle cell, immunosuppression)</td>
</tr>
<tr>
<td><strong>E. coli</strong></td>
<td>Watery diarrhea</td>
<td>Hydration; fluoroquinolones, azithromycin, or Bactrim</td>
<td>Tx w/ abx may trigger HUS in pts w/ E. coli 0157 (controversial)</td>
</tr>
<tr>
<td><strong>V. cholerae</strong></td>
<td>Watery diarrhea</td>
<td>Hydration; tetracycline or erythromycin</td>
<td></td>
</tr>
<tr>
<td><strong>V. parahaemolyticus</strong></td>
<td>Rice-water diarrhea in pt who ingested inadequately cooked seafood</td>
<td>Bactrim 10 mg (TMP)/kg/24 h BID for 7–10 d in severe cases</td>
<td></td>
</tr>
<tr>
<td><strong>Y. enterocolitica</strong></td>
<td>Diarrhea possibly w/ blood/mucus/pus a/w fever, vomiting, &amp; RLQ pain</td>
<td>Hydration</td>
<td>Mimics appendicitis</td>
</tr>
<tr>
<td><strong>C. difficile</strong></td>
<td>Diarrhea a/w recent abx use</td>
<td>Metronidazole 15–30 mg/kg/24 h PO TID or vancomycin 40 mg/kg/24 h PO q6h</td>
<td>Toxic megacolon very rare in children, but possible</td>
</tr>
<tr>
<td><strong>S. aureus</strong></td>
<td>Foodborne toxin mediated w/ abrupt, dramatic onset of sx w/ 2–6 h of ingestion</td>
<td>Hydration, supportive care</td>
<td></td>
</tr>
<tr>
<td><strong>Parasitic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>G. lamblia</strong></td>
<td>Watery diarrhea &amp; excessive, particularly malodorous, flatulence in pt exposed to children in daycare or mountain streams</td>
<td>Hydration &amp; supportive care; metronidazole 15–30 mg/kg/24 h PO TID for 5 d</td>
<td></td>
</tr>
</tbody>
</table>
**E. histolytica**  
Diarrhea w/ blood & mucus  
Hydration; metronidazole 15–30 mg/kg/24 h TID  
A/w hepatic abscesses  
Consider paromomycin to treat intraluminal infection

QID, 4 times daily; BID, twice daily; q6h, every 6 h; CBC, complete blood count; BUN, serum urea nitrogen; Cr, creatinine; RLQ, right lower quadrant; TMP, trimvibrio colethoprim; PO, by mouth; TID, 3 times daily.

### PEDIATRIC MENINGITIS (SEE 5D)

**History**
- HA, fever, neck stiffness, lethargy (AMS), N/V, rash, irritability, sz, somnolence

**Findings**
- Meningismus (stiff neck) occurs <15% of the time in children <18 mo old, petechial rash, irritability/lethargy, hemodynamic instability, fever, sz

**Evaluation**
- If bacterial etiology suspected, abx should be given immediately, then LP (see 4d)

**Treatment**
- Dexamethasone 0.15 mg/kg IV before 1st dose of abx (↓ ICP, risk of hearing loss 2/2 H. influenzae)
- Abx
  - <1 mo: Ampicillin (100 mg/kg) + gentamicin (2.5 mg/kg) OR cefotaxime (50 mg/kg)
  - 1–2 mo: Ampicillin + ceftriaxone (100 mg/kg) OR cefotaxime
  - >2 mo: Vancomycin (15 mg/kg) + ceftriaxone OR cefotaxime
- Consider adding acyclovir 20 mg/kg IV

**Clinical Pearl**
- Ampicillin necessary to cover Listeria in infants

### NEONATAL COMPLAINTS
**Approach**
- Differentiate nervous parents from a child w/ true dz

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Complaint</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Poor feeding, reflux/regurgitation, vomiting, diarrhea, constipation, jaundice</td>
</tr>
<tr>
<td>Infectious dz</td>
<td>Fever</td>
</tr>
<tr>
<td>Other</td>
<td>Crying/colic, ALTE, sudden infant death syndrome (SIDS)</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Stridor, apnea, cyanosis</td>
</tr>
</tbody>
</table>

**History**
- Events during pregnancy, delivery, gestational age & weight @ birth, alertness, diet, frequency of diaper Δ, crying patterns, color Δ; FH

**Findings**
- Weight, VS, color; undress baby → full exam

**Pearl**
- Many signs/sxs are nonsp: Abnl tone, weak suck, decrease PO intake, jaundice, abnl breathing, peripheral cyanosis, vomiting

---

**POOR FEEDING**

**Approach**
- Check for appropriate weight gain (5–7% wt loss during 1st week nl, then gain 1 oz/d for 1st 3 mo), take a careful hx/physical exam to identify any other abnlty

**Treatment**
- If weight gain is appropriate & pt has no other issues, attempt feeding trial

**Disposition**
- Pts w/ appropriate wt gain who tolerate POs in ED may be D/C home w/ parental reassurance & outpt f/u; all other pts require further eval (see w/u for inconsolability below)
CONSTITUTION

Approach

- Differentiate functional (no underlying condition) from pathologic constipation

<table>
<thead>
<tr>
<th>Constipation Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Obstruction</td>
</tr>
<tr>
<td>Metabolic</td>
</tr>
<tr>
<td>Neuro</td>
</tr>
<tr>
<td>Other</td>
</tr>
</tbody>
</table>

History

- Sx time course, Δ in stool consistency, baseline stooling patterns, 1st meconium passage after birth (>24–48 h = abnl), recent illness, V/D, fever, ingestion of honey

Findings

- Abd (distension), rectal exam (patency, stool @ vault), neuro exam (CNs, muscular tone)

Evaluation

- KUB (if obstruction is suspected); consider Chem 7, TSH, Ca, heavy metal screen

Treatment

- For functional constipation: Glycerin suppository, disimpaction, increased water b/w feedings, consider bisacodyl, lactulose, enemas, high fiber diet in older children
- Fleets enemas may cause hypocalcemia, avoid in young infants

Disposition

- Functional constipation → d/c w/ PCP f/u: Pathologic causes warrant further w/u & may require admission
**Crying and Colic**

**Definition**
- Colic: Recurrent pattern of inconsolable crying & irritability lasting >3 h/d on >3 d/wk, 3 wk–3 mo of life. Benign GI colic is Dx of exclusion.

**Approach**
- Excessive crying/colic are nonsp complaints that can be the presenting signs of benign GI distress or life-threatening dz

<table>
<thead>
<tr>
<th>Colic Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>System</strong></td>
</tr>
<tr>
<td>CNS</td>
</tr>
<tr>
<td>HEENT</td>
</tr>
<tr>
<td>Cardiac</td>
</tr>
<tr>
<td>GI</td>
</tr>
<tr>
<td>GU</td>
</tr>
<tr>
<td>Other</td>
</tr>
</tbody>
</table>

**History**
- Timing of crying, trauma, fever, medication ingestion, feeding hx, complete ROS & PMH

**Findings**
- Observe behavior, thorough physical exam

**Evaluation**
- UA; consider further testing (eg, abd U/S, x-ray, LP, tox screen) to r/o spec etiologies

**Treatment**
- Treat the underlying d/o

**Disposition**
- Home: If etiology is thought to be benign & pt has cry-free period in ED
- Admit: Any pt w/o clear etiology identified & no cry-free period in ED

Definition

• Observed episode frightening to the observer & characterized by ≥1 of the following: Apnea, color Δ, Δ in muscle tone, choking, &/or gagging
• Separate clinical entity from SIDS & represents a wide spectrum of etiologies

<table>
<thead>
<tr>
<th>ALTE Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiology</strong></td>
</tr>
<tr>
<td>Cardiac</td>
</tr>
<tr>
<td>GI</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td>Metabolic</td>
</tr>
<tr>
<td>Neuro</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>Respiratory</td>
</tr>
</tbody>
</table>

History

• Obtain 1st-hand account of event when possible, appearance of child (central vs. peripheral cyanosis, pallor, etc.), preceding sxs, prior episodes, presence of apnea or gagging, muscle tone, sz-like activity, spontaneous or facilitated recovery

Findings

• Through physical exam

Evaluation

• No standard diagnostic strategy exists. Testing should be guided by hx & physical exam. Consider: CBC, Chem 7, Ca, UA, urine/blood cx, ECG, RSV swab; CT head/LP (based on clinical suspicion); also consider ABG, serum/urine tox, pertussis screen, EEG

Disposition

• Observe in the ED; pts w/o true ALTE (eg, breath-holding spell) can be D/C w/ f/u in 24 h
Infant w/ hx of apnea, pallor, cyanosis, limp, unresponsive req stimulation or CPR or have inadequate f/u require admission for observation & further revaluation

**Pearl**
- Definitive etiology of the ALTE is found in only ~50% of pts

---

### SIDS

#### Definition
- D of child <12 mo of age that is unexplained after careful investigation, autopsy, exam of the D scene, & hx; most common @ 2–5 mo

#### Approach
- Approach parents of SIDS pts w/ sympathy, as child abuse rare in SIDS (<1–5%)

#### Risk Factors for SIDS

<table>
<thead>
<tr>
<th>Category</th>
<th>fro</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant</td>
<td>Male, preterm or multiple birth, low birth weight, low Apgar scores, ICU tx, congenital dz, neonatal respiratory abnlty, recent viral illness, previous ALTE, sibling w/ SIDS, prone sleeping position, heavy layers</td>
</tr>
<tr>
<td>Maternal</td>
<td>Age &lt;20 y/o, unmarried, low socioeconomic status, low educational level, inadequate prenatal care, illness during pregnancy, smoking during pregnancy, use of illicit drugs, bed sharing</td>
</tr>
</tbody>
</table>

#### Prevention
- Remind parents to lay their children in the supine position, avoid smoking, head covering, soft sleeping surfaces, & multiple layers to reduce risk

---

### CONGENITAL HEART DISEASE

#### Approach
- Consider Dx in pts w/ sudden onset cyanosis, hypoxemia, &/or shock, typically in the 1st 1–2 wk of life, though some pts present weeks to
years later

- Differentiate cyanotic vs. noncyanotic, & ductal vs. nonductal dependent congenital heart dz
- Hyperoxia test: Compare ABG on room air & on 100% O₂ for 10 min, P₀₂ of >250 makes hypoxia 2/2 congenital heart dz unlikely
- Give PGE₁ to any pt w/ suspected ductal-dependent lesion & circulatory compromise

**Definition**

- Cyanotic lesions: Congenital cardiac dz w/ right-to-left shunt
- Ductal dependent lesions: Congenital cardiac dz in which fetal life depends on a PDA, either from impaired systemic or pulmonary blood flow

<table>
<thead>
<tr>
<th>Congenital Heart Dz Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type of Lesion</strong></td>
</tr>
<tr>
<td>Noncyanotic</td>
</tr>
</tbody>
</table>

*Ductal dependent.

**History**

- Cyanosis, fussy baby, poor feeding

**Findings**

- ↓ O₂ sat, cyanosis, ↓ BP, cardiac murmur, hepatomegaly, check 4-extremity BPs

**Evaluation**

- ABG, response to O₂, CXR, ECG, echo

**Treatment**

- O₂, consider PGE₁ (alprostadil): 0.05–0.1 μg/kg/min (max 0.4 μg/kg/min) if ductal-dependent lesion suspected, side effects: bradycardia, hyperthermia, hypotension, & apnea
- Inotropic support w/ milrinone, dopamine, or dobutamine & intubation
Disposition
- Cardiology consult, ±cardiac surgery consult, admit

Pearls
- Pts w/ ductal-dependent lesions p/w circulatory failure, usually during 1–2 wk of life
- Acyanotic lesions may p/w CHF

Tetralogy of Fallot

Approach
- Recognize/tx Tet spells

Definition
- PA stenosis, VSD, RV hypertrophy, & deviation of aortic origin to the right (overriding); degree of severity dictated by degree of RV outflow tract obstruction

History
- Presentation usually w/i 1st few years of life, though occasionally into adulthood
- Cyanosis (often during feeds), ↓ PO intake, agitation, ↑ RR; ↑ sxs w/ exercise, szs, CVA
- “Tet” spell: Infundibulum spasm → ↑ RV outflow obstruction → cyanosis, respiratory distress

Findings
- ↓ O₂ sat, systolic ejection murmur, cyanosis, squatting pt

Evaluation
- See above, ECG (RAD, RVH, RAE, RBBB), CXR (boot-shaped heart), CBC, VBG

Treatment
- See above, 100% O₂, calm child, bring knees to chest; consider morphine & IV fluid bolus, correct hypoglycemia, consider propranolol, phenylephrine, intubation

Disposition
› Cardiology, cardiac surgery consult, admit

Pearl
› Onset determined by slowly ↑ infundibulum hypertrophy → ↑ RV outflow tract obstruction → ↑ RV hypertrophy → ↑ right-to-left shunt; thus presentations at later age have poorer long-term outcomes

RESPIRATORY COMPLAINTS

PNEUMONIA

History
› Fever, cough; quality of sputum usually unascertainable (children often swallow secretions); recent URI, malaise, lethargy, N/V, SOB, nasal flaring & grunting
› Older children: Abd pain, neck stiffness
› Infants/neonates: Difficulty feeding, tachypnea, restlessness, or lethargy
› RFs: Lack of immunizations/incomplete immunizations, travel, daycare

Bacterial (10–40%)
› Abrupt, follows URI, appearing ill, usually <5 yr

Atypical
› Fever, malaise, & myalgia, HA, photophobia, sore throat, & gradually worsening nonproductive cough

Viral
› Nontoxic, associated upper airway sxs (runny nose, nasal congestion)

Physical Exam
› Fever, tachypnea (most sens), O₂ saturation; full pulmonary exam (rales, rhonchi, decreased breath sounds)

Evaluation
› Labs: Chem 7 (severe dehydration), CBC (elevated WBC), blood cultures (if seriously ill); consider viral panel (including RSV)
› Imaging: CXR

Treatment
• Supportive: IVFs (if dehydrated), O₂ monitoring & therapy
• Viral: Supportive
• Abx (duration is 14 d for neonates, o/w 7–10 d), add vancomycin if critically ill
  • Neonate – ampicillin + gentamicin inpt
  • 1–3 mo – 3rd-generation cephalosporin + macrolide inpt
  • 3 mo–5 yr – 3rd-generation cephalosporin + macrolide (inpt) or high-dose amoxicillin (outpt)
  • 5–18 yr – 3rd-generation cephalosporin + macrolide (inpt) or macrolide alone (outpt)

Disposition
• Home: Immunizations up to date, HD stable, on room air, >3 mo
• Admit: <3 mo, temp >38.5°C, tachypnea (>70 breaths in <12 mo & >50 breaths in older children), retractions in infants, respiratory distress, nasal flaring, cyanosis or hypoxemia (O₂ <92%), intermittent apnea, grunting, poor POs, signs of dehydration, social concerns, inadequate f/u, sepsis, immunosuppressed, comorbidities, cx, virulent pathogens

(Thorax 2002;57(suppl 1))

Asthma and Bronchiolitis

History

Asthma
• Cough (usually early), dyspnea & wheezing (generally worse at night). Consider frequency, severity, duration, home txs, required past txs, baseline peak flow, number of ED visits, hospitalizations, ICU admissions, intubations
• Triggers: Exercise, infection, cold air, allergens, any respiratory irritant

Bronchiolitis (usually <2 yr of age)
• Fever (usually ≤38.3°C), cough, wheezing, mild respiratory distress; etiology from viral exposure (usually RSV, but also parainfluenza, adenovirus, influenza, rhinovirus). Often preceded by a 1–3-d hx of nasal congestion & mild cough
• RFs for severity: Prematurity, low birth weight, <12 wk old, congenital dz, immunodeficiency, neurologic dz

Physical
Asthma

- Tachypnea, tachycardia, inspiratory/expiratory wheezes, decreased or no air movement, use of accessory muscles, anxious/agitated, signs of dehydration

Bronchiolitis

- Same as asthma; may hear crackles & have signs of other infections such as OM

Evaluation

- **Pulse ox**: Continuous, unless very mild sxs
- **Labs**: Usually not necessary, consider RSV testing (bronchiolitis) if admission
- **Imaging**: CXR only if concomitant PNA suspected or 1st-time wheezer
- **Peak flow** (asthma): In children >6 yr (compare to predicted based on height)

Treatment

- Supportive: ABCs, O₂ therapy (O₂ sat >90%)

Asthma

- Mild/moderate:
  - **Albuterol**: 0.15 mg/kg (max 5 mg) q20–30min × 3 doses (short acting β-agonist)
  - **Ipratropium bromide**: 250 μg/dose (<20 kg) OR 500 μg/dose (>20 kg) q20–30min × 3 doses may decrease need for hospitalization
  - **Steroids**: Prednisolone/prednisone 2 mg/kg PO (max 60 mg) OR methylprednisolone 1–2 mg/kg IV (max 125 mg) OR dexamethasone 0.6 mg/kg PO (max 16 mg)
- Severe (add):
  - **Albuterol**: As above but may be used continuously
  - **Magnesium**: 75 mg/kg IV (max 2.5 g) over 20 min (optimal dose unknown)
  - **Heliox**: 80% helium/20% O₂. Use only if O₂ saturation can be maintained above 90%
  - **Terbutaline or epinephrine**: Terbutaline 0.01 mg/kg SC (max 0.4 mg) q20min × 2 doses &/or epinephrine 0.01 mg/kg SC (max 0.4 mg) q20min × 3 doses then repeated q4–6h
- Ventilation:
  - **Noninvasive** (BiPAP): May reduce respiratory fatigue & improve
oxygenation/ventilation

- **Intubation:** For impending respiratory failure; use large ETT; consider permissive hypercapnia (increased expiratory time & low VTs to prevent barotraumas). Consider ketamine for induction (bronchodilating properties)

**Bronchiolitis**

- Supportive tx is the mainstay including humidified O₂, suctioning, oral hydration
- Trial of albuterol, can continue only after documented response
- Nebulized hypertonic saline not a/w decreased hospital LOS after controlling for heterogeneity (*JAMA Pediatr* 2016;170(6):577)
- Racemic epinephrine may be helpful
  - <2 yr: 0.25 mL of 2.25% solution via nebulizer diluted in 3 mL NS
  - ≥2 yr: 0.5 mL of 2.25% solution via nebulizer diluted in 3 mL NS
- Consider ribavirin if documented RSV bronchiolitis w/ severe dz or immunosuppression &/or HD unstable

**Disposition**

**Asthma**

- Reassess pt in 3 h (more frequent if sx more severe) after nebulizers, steroids, O₂ therapy
- Home: Improved peak flow (to >70% predicted), significant improvement in RR/O₂ saturation; d/c w/ inhaled β-agonist, steroid burst × 5 d (see *Adult Asthma Table for further home management*) w/ close f/u
- Admission:
  - Floor: Persistent wheezing w/ nasal flaring, tachypnea, hypoxia, & unable to tolerate POs
  - ICU: If pt maintains severe wheezing/poor air movement w/ peak flow <50% & worsening tachypnea or possible impending respiratory fatigue, PCO₂ >42 mmHg, intubated, requiring continuous nebs, heliox, or terbutaline

**Bronchiolitis**

- Home: Age >2 mo, no hx of intubation, eczema, RR <45, no/mild retractions, O₂ sat >93%, tolerating PO, fewer albuterol/epinephrine txs in 1st hour (*Pediatrics* 2008;121(4):680)
- Admission: Age <6 wk, hypoxia, persistent respiratory distress,
significant comorbidities, or immunosuppression

**BRONCHOPULMONARY DYSPLASIA**

**Definition**
- Chronic lung dz in preterm neonates w/ hx of ICU, malnutrition, exposure to high O\textsubscript{2} concentrations, inflammation, infection (sepsis, chorioamnionitis, funisitis, postnatal infections), & PPV → impaired alveolar/pulmonary vascular development

<table>
<thead>
<tr>
<th>Dz Severity</th>
<th>O\textsubscript{2} Supplement &gt;36 wk Postmenstrual Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity of BPD</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>None</td>
</tr>
<tr>
<td>Moderate</td>
<td>&lt;30% O\textsubscript{2}</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;30% O\textsubscript{2} &amp;/or positive pressure</td>
</tr>
</tbody>
</table>

**History**
- Preterm birth, hx of ICU stay w/ mechanical ventilation, recent respiratory infection, poor feeding, increased O\textsubscript{2} requirement

**Physical Exam**
- Abnl VS, nasal flaring, retractions, grunting, wheezes, rales, decreased breath sounds

**Evaluation**
- CXR – hyperinflation, cystic areas, scarring; RSV testing will identify those who require hospitalization

**Treatment**
- Supportive; O\textsubscript{2}, consider inhaled & systemic corticosteroids, abx (see *Pediatric Pneumonia*), bronchodilators (see *Pediatric Asthma*), furosemide (1 mg/kg q6–12h, titrate to effect)

**Disposition**
- Admission: If increased respiratory distress, hypoxia, hypercarbia, new pulmonary infiltrates, inability to maintain oral hydration, RSV infection

---

**UPPER AIRWAY EMERGENCIES**
Definition
- Actual or impending obstruction of the upper airway

Approach to the Patient

History
- Agitation or fidgeting, cyanosis, AMS, choking, SOB, increased work of breathing, panic, unconscious, unusual breathing noises
- ROS (fever, drooling), PMH/MEDS (see differential chart)

Diagnostics
- CXR or neck films, esp if abnl O₂ sat & temp

Treatment
- O₂, calm the child, head tilt, chin lift, “position of comfort” (upright while leaning forward)

Disposition
- Largely will depend on hemodynamic stability & airway issues

<table>
<thead>
<tr>
<th>UAE Differential</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathophysiology</td>
<td>Differential</td>
</tr>
<tr>
<td>Structural</td>
<td>Tracheomalacia, laryngomalacia, tumors, macroglossia</td>
</tr>
<tr>
<td>Infectious</td>
<td>Peritonsillar abscess, epiglottitis, retropharyngeal abscess, bacterial tracheitis, croup</td>
</tr>
<tr>
<td>Other</td>
<td>Allergic rxn, chemical burns, FB aspiration, trauma</td>
</tr>
</tbody>
</table>

FOREIGN BODY/UPPER AIRWAY OBSTRUCTION (SEE ADULT RESPIRATORY SECTION)

Croup (Laryngotracheobronchitis)

Definition
- Viral infection primarily of the larynx & trachea (often parainfluenza), age 6 mo–6 yr

History
- Hoarseness, barking cough, & inspiratory stridor w/ variable degree of respiratory distress; preceded by nonsp respiratory sxs (rhinorrhea, sore throat, cough); fever is usually low grade
Physical Exam

- Inspiratory stridor, retractions, decreased air entry

Evaluation

- Labs: None
- Imaging: not routinely indicated
- CXR: PA view may show steeple sign, (subglottic narrowing), lateral view may reveal a distended hypopharynx (ballooning) during inspiration

Treatment

- Supportive: Humidified air, O₂, keep child as comfortable as possible
- Steroids: Dexamethasone (0.6 mg/kg ×1, max of 10 mg)
- Racemic epinephrine: Below dosing mixed w/ 3 cc NS (may repeat q20–30min), for children w/ stridor at rest, requires 2–3 h observation for “rebound stridor”
  - <20 kg: 0.25 mL
  - 20–40 kg: 0.5 mL
  - >40 kg: 0.75 mL

Disposition

- Home: If maintaining O₂ saturation; advise symptomatic tx w/ Tylenol & humidified air
- Admit: If hypoxia, depressed sensorium, moderate to severe respiratory distress, stridor at rest, poor oral intake, dehydration

Epiglottitis

Definition

- Pharyngeal infection classically due to H. influenzae; incidence in children has declined since introduction of H. influenzae vaccine, most common organisms now include S. pyogenes, S. aureus, S. pneumoniae, Moraxella

History

- Fever is usually 1st sx w/ abrupt onset sore throat, stridor, labored breathing, drooling muffled/hoarse voice, age 2–7 yr, lack of cough

Physical Exam

- Toxic, irritable, anxious, sitting in tripod or sniffing position (chin hyperextended & leaning forward), drooling, retractions, adenopathy; may visualize edematous epiglottis on oral exam
**Evaluation**

- **Labs:** Postpone IV & labs until airway secured; CBC, blood cultures, Chem 7
- **Imaging:** Lateral neck x-ray: Swollen epiglottis (ie, thumbprint sign), thickened aryepiglottic folds, obliteration of the vallecula, & dilation of the hypopharynx

**Treatment**

- Supportive: O₂ therapy, keep child as comfortable as possible; place child & mom in a quiet & controlled for complete eval/tx
- Airway: Preferable secured in OR under controlled environments but if not available, consider partial sedation & fiberoptic intubation. Cricothyrotomy kit at bedside for emergent surgical airway; tracheostomy.
- Abx: Ceftriaxone 100 mg/kg IV q12h (max 2 g/d) + Vancomycin OR clindamycin if concern for MRSA
- Consult: ENT or anesthesia for STAT OR airway

**Disposition**

- Admit: All to the ICU

**Pearls**

- Avoid procedures which may cause distress to the pt & further thereby compromise airway
- Give child or parent Yankauer suction to maintain secretions & alleviate associated anxiety

**Bacterial Tracheitis**

**Definition**

- Infection of subglottic region causing edema, pseudomembrane formation; polymicrobial (S. aureus, S. pneumoniae, H. influenzae, Pseudomonas, Moraxella), average age 3 yr

**History**

- Preceding URI infection w/ rapid deterioration, high fevers, age 3 mo–5 yr

**Physical Exam**

- Stridor, retractions, tachypnea, barking cough, wheezing, high fevers, toxic appearing

**Evaluation**
Labs: None
Imaging: X-ray shows subglottic & tracheal narrowing, irregular tracheal margins, PNA

Treatment
- Supportive: O₂, frequent suctioning, use one size smaller ETT
- Broad spectrum abx (3rd-generation cephalosporin, vancomycin)

Disposition
- ICU

---

**DIABETIC KETOACIDOSIS**

History
- Fatigue & malaise, N/V, abd pain, polydipsia, polyuria, polyphagia, weight loss, AMS/HA (may be signs of cerebral edema), fever/sxs of infection (cough, URI sxs, dysuria, rash); toddlers may not present w/ classic sxs
- RFs: Infection, poor compliance w/ insulin, puberty, inadequate caregiver

Physical Exam
- AMS, tachycardia, tachypnea, Kussmaul respirations, normo- or hypotensive, delayed capillary refill, mottled, lethargy/weakness, fever, N/V, acetone on breath (metabolic acidosis)

Evaluation
- Labs: FSG, Chem 10 (elevated anion gap acidosis, pseudohyponatremia, total body K generally depleted despite lab value, ↓ phosphorus, ↓ Mg), urine/serum ketones, β-hydroxybutyrate, UA, CBC, lactate, lipase, LFTs, urine hCG, VBG; ABG if HD unstable or comatose; blood & Ucxs if febrile
  - Corrected Na = measured Na + [1.6 × (measured glucose – 100)/100]
  - Definition: Glucose >200, venous pH <7.3 or bicarb <15, ketonemia & ketonuria
- ECG: T wave Δ (hyper/hypokalemia)
- Imaging: If concern for focal infection

Treatment
Supportive: Continuous cardiac monitoring, O₂ sat monitoring, 2 large-bore IVs, intubate if necessary, evaluate & treat sources of infection

Electrolyte monitoring: Glucose fingerstick q1h (goal = 150); Chem 7, Ca, Mg, phosphorus q2h

### Acute Tx

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose/Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IV hydration</strong></td>
<td>Slow NS bolus 10–20 cc/kg over 1–2 h + maintenance (weight based) (adjust for dehydration) Add dextrose once serum glucose &lt;250 mg/dL</td>
</tr>
</tbody>
</table>
| **Insulin**                             | 0.1 U/kg/h<br>*Persistent anion gap:* Continue drip  
Resolution of anion gap: Δ to SC insulin (overlap IV w/ SC by 2–3 h) |
| **Electrolyte repletion**               | *Potassium:* Add 20–30 mEq/L IVFs (K⁺: 3.5–5) OR 40 mEq/L IVFs (K⁺ <3.5) as insulin promotes K⁺ entry into cells  
*HCO₃⁻:* ↑ risk of cerebral edema. Avoid use  
*Phosphate:* Replete if <2, monitor for hypocalcemia |
| **Mannitol or hypertonic saline**       | *Mannitol:* 0.25–1 g/kg IV over 20 min (may repeat in 2 h if no improvement)  
*Hypertonic saline:* 5–10 cc/kg over 30 min × 1 |

### Disposition

- Admit: All pts; HD unstable, pts w/ cerebral edema/AMS or newly diagnosed diabetes pts should go to the ICU

### Pearl

- Children more likely than adults to develop cerebral edema; carry a 25% mortality rate; avoid insulin bolus & large-volume isotonic fluid boluses

### HYPOGLYCEMIA

#### Definition

- Glucose <50 in children; glucose <40 w/ age 3–24 h; glucose <45 in infants >24 h of age

#### Hypoglycemia Differential
### Pathophysiology vs. Differential

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congenital</strong></td>
<td>Glycogen storage disorders, disorders of gluconeogenesis, disorders of fatty acid or amino acid metabolism</td>
</tr>
<tr>
<td><strong>Autoimmune/ endocrine/neoplasm</strong></td>
<td>Hypothyroidism, insulinoma, hypopituitarism, adrenal insufficiency, glucagon deficiency, GH deficiency</td>
</tr>
<tr>
<td><strong>GI</strong></td>
<td>Liver pathology, Reye syndrome</td>
</tr>
<tr>
<td><strong>Other/meds</strong></td>
<td>Oral hypoglycemics, pentamidine, alcohol ingestion, βB, salicylates, INH, sepsis, burns, cardiogenic shock</td>
</tr>
</tbody>
</table>

### Approach to the Patient

**History**
- Irritability, sweating, jitteriness, feeding problems, lethargy, cyanosis, tachypnea, &/or hypothermia. May be a/w sepsis, congenital heart dz, ventricular hemorrhage, tox, & respiratory distress syndrome, PMH/meds (see chart)

**Physical Exam**
- Hypotonia, lethargy, cyanotic, hypothermic, apneic, tachycardic, pallor, vomiting, tremulousness, ataxia, sz, diplopia, signs of CVA

**Evaluation**
- **Labs:** FSG, Chem 7, LFTs, serum insulin, UA (ketones), C-peptide (low in exogenous insulin, high in insulinoma or sulfonylureas); growth hormone, cortisol, glucagon levels; tox screen if indicated

**Treatment**
- Glucose replacement
  - PO: Glucose paste, fruit juice (preferred)
  - Infants: IV bolus: 10% dextrose: 2 mL/kg followed by infusion at 6–9 mg/kg/min
  - Children: IV bolus: 10% dextrose at 5 mL/kg followed by infusion at 6–9 mg/kg/min
  - IM: Glucagon 0.03–0.1 mg/kg/dose SC q20min prn; not to exceed 1 mg/dose

**Disposition**
- Home: Obvious cause treated, sx reversed, after high-carbohydrate meal
- Admit: No obvious cause, toxic ingestion w/ oral hypoglycemic, long acting insulin, persistent sxs
# FLUID AND ELECTROLYTE ABNORMALITIES

## Definition
- See *Adult Metabolic Abnormalities* for etiologies

## History
- **Hyponatremia:** Fatigue, weakness, lethargy, agitation, szs; ask re: renal dz or GI distress
- **Hypernatremia:** Irritability, lethargy, szs, fever, over or lack of urination
- **Hypokalemia:** Weakness, smooth muscle dysfxn, lethargy, confusion, decreased GI motility, respiratory insufficiency, rhabdomyolysis, polyuria
- **Hyperkalemia:** Asymptomatic to generalized weakness, paralysis, paresthesias
- **Hypocalcemia:** Tetany, weakness, fatigue, paresthesias, laryngospasm, sz, irritability
- **Hypercalcemia:** Weakness, respiratory distress, apnea, HA, sz, abd pain, lethargy, anorexia, constipation, bone pain, signs of kidney stones, pancreatitis, N/V, psychosis
- **Hypomagnesemia:** Anorexia, nausea, weakness, nonsp psychiatric sx
- **Hypermagnesemia:** Lethargy, confusion, respiratory distress

## Physical Exam
- **Hyponatremia:** May appear euvoletic, dehydrated or hypovolemic; severe hyponatremia = lethargy, hyporeflexia, Cheyne–Stokes respiration
- **Hypernatremia:** Poor skin turgor, increased muscle tone, altered sensorium; severe hypernatremia = spasticity, lethargy, hyperreflexia, respiratory paralysis
- **Hypokalemia:** Skeletal muscle weakness, hyporeflexia, lethargy, confusion
- **Hyperkalemia:** Paralysis, hyporeflexia, confusion
- **Hypocalcemia:** Tetany, wheezing/inspiratory stridor, Chvostek/Trousseau
- **Hypercalcemia:** Respiratory distress, apnea, hyporeflexia, epigastric tenderness, ↑ BP
Hypomagnesemia: Anorexia, nausea, weakness, clonus, tetany, Chvostek/Trousseau
Hypermagnesemia: Lethargy, hyporeflexia, hypotension, respiratory failure

Evaluation
- Labs: R/o spurious lab draws, hemolysis (hyperkalemia); CBC, Chem 7, Ca/Mg/phosphorus, urine electrolytes; ABG if acidotic & respiratory decline, UA, lipase
- ECG: U wave (hypokalemia), peaked T/widened QRS/ventricular tachycardia (hyperkalemia), prolonged QT (hypocalcemia), shortened QT (hypercalcemia), ventricular arrhythmia/torsades de pointe (hypomagnesemia)

Treatment
- Supportive: Continuous cardiac monitoring, O₂ sat monitoring, 2 large-bore IVs
- Electrolyte monitoring: Chem 7, Ca, Mg, phosphorus q4h
- Electrolyte correction:
  - Hyponatremia: See Adult Metabolic section; determine volume status; children should not be corrected >10 mEq/L/d in hypovolemia; acute onset <48 h hyponatremia can be corrected more quickly over 24 h; 3% NS 3–5 mL/kg for severe neuro sx (ie, szs); consider loop diuretics; tx underlying cause
  - Hypernatremia: See Adult Metabolic section for Na correction (goal Na reduction rate of 0.5–1 mEq/L/h); consider vasopressin/DDAVP for DI
  - Hypokalemia: Correct alkalosis, hypomagnesemia
    - IV: 0.5–1 mEq/kg/h IV (max 40 mEq/dose) over 1–2 h. Goal ↑ potassium by 0.3–0.5 mEq/L (require ECG monitoring)
    - PO: 1–4 mEq/kg/d PO in divided doses (max 20 mEq/dose)
  - Hyperkalemia:
    - Calcium gluconate: 50–100 mg/kg/dose IV, up to adult dose
    - Calcium chloride (code situation): 10–20 mg/kg/dose IV, up to adult dose over 2–5 min
    - Glucose + insulin: 1 g/kg IV of D₂₅W + 0.25 U/kg IV insulin
    - Sodium bicarbonate: 1–2 mEq/kg/dose IV over 5–10 min
    - Albuterol: 2.5–5 mg nebulized
    - Furosemide: 1–2 mg/kg IV/PO; hydrochlorothiazide 1 mg/kg PO up to 200 mg
- Kayexalate: 1 g/kg PO
- Dialysis
- Hypocalcemia: Send ionized calcium
  - Symptomatic:
    - Calcium gluconate 10%: 50–100 mg/kg IV slowly over 5–10 min to control szs; IV infusion at 50–75 mg/kg/d over 24 h; use calcium chloride (dose as in hyperkalemia) in code situation
  - Asymptomatic:
    - Calcium carbonate: Neonates: 30–150 mg/kg/d PO divided QID; children: 20–65 mg/kg/d PO divided BID/QID
- Hypercalcemia: Send ionized calcium
  - NS: (Weight-based bolus + 1.5 times maintenance); furosemide
  - Consider bisphosphonates, calcitonin
  - Dialysis: In extreme hypercalcemia & renal failure
- Hypomagnesemia:
  - PO: Magnesium gluconate 10–20 mg/kg TID/QID
  - IV: Magnesium sulfate 25–50 mg/kg IV over 2–4 h
- Hypermagnesemia:
  - NS infusion, furosemide 1 mg/kg/dose q6–12h; titrate to effect
  - Calcium gluconate/calcium chloride (same dose as hyperkalemia)
  - Dialysis: Severe renal failure, cardiac or neuromuscular Dysfxn

**Disposition**
- Home: Mild, asymptomatic electrolyte abnlty may be D/C home w/ PCP f/u in 1–2 d for repeat labs
- Admit: All pts w/ symptomatic electrolyte abnlty should be admitted & monitored; consider ICU level care for HD unstable or those w/ severe cardiac or neurologic disturbances

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**PEDIATRIC EXANTHEMS**

**ERYSIPelas**

**Definition**
- Infection caused most commonly by group A *Strep*
**History**
- Any age but > in children <3 yr

**Physical Findings**
- Red/hot tender area of skin, purulent d/c at entry site, ±fevers

**Treatment**
- Penicillin G, dicloxacillin

**Disposition**
- Home

---

**Viral Exanthem**

**Definition**
- Diffuse rash caused by nonpolio enteroviruses (coxsackievirus, echovirus, enterovirus) & respiratory viruses (adenovirus, parainfluenza virus, influenza, RSV)

**History**
- Any age, recent viral illness

**Physical Findings**
- Diffuse blanchable erythematous macules on trunk & extremities

**Treatment**
- Supportive

**Disposition**
- Home

---

**Hand-Foot-and-Mouth Disease**

**Definition**
- Caused by coxsackievirus B

**History**
- Summer/fall, 1–4 y/o

**Physical Findings**
- Ulcerative oral lesions on soft palate, macular → pustular → crusted
lesions on palms/soles, resolves in 5–6 d

**Treatment**
- Supportive

**Disposition**
- Home

---

**IMPETIGO**

**Definition**
- Secondary infection in pts w/ underlying dermatoses caused by *S. aureus* & group A *Strep*

**History**
- Warm humid summer months, any age

**Physical Findings**
- Papule/vesicle → golden crusted lesions commonly around mouth & on cheeks

**Treatment**
- Topical abx (2% mupirocin, dicloxacillin, 1st-generation cephalosporins)

**Disposition**
- Home

---

**KAWASAKI DISEASE**

**Definition**
- Systemic vasculitis of microvessels of unknown etiology, often self-limited

**History**
- Febrile illness, peak onset 18–24 mo, usually in children <5 yr of age

**Physical Findings**
- To make the Dx, requires unexplained fever × 5 d + 4 of the following:
  - Edema/desquamation of extremities
  - Bulbar conjunctivitis
  - Polymorphous rash
• Cervical LAD
• Mucous membrane Δ (ie, strawberry tongue)

Evaluation
› CBC (↑ WBC, ↑ PLT), ↑ LFTs, ↑ ESR, ↑ CRP, sterile pyuria, ECG, echocardiography, RUQ U/S

Treatment
› High-dose ASA 100 mg/kg/d divided QID
› IVIG 2 g/kg infused over 8–12 h single dose (reduces risk of coronary artery aneurysms)

Disposition
› Admit

Complications
› #1 cause of acquired heart dz in children
› Cx: coronary artery aneurysm, CHF, MI, dysrhythmias, valvular insufficiency, gallbladder hydrops, uveitis

SERUM SICKNESS

Definition
› Immune-complex–mediated type III hypersensitivity rxn

History
› Any age but > in children <3 yr, fever, arthralgias, rash, possible etiologies include blood products, antitoxins (ie, spider or snake envenomations), clostridial infections, meds

Physical Findings
› Fever, rash (urticarial, serpiginous)

Treatment
› Supportive as dz is self-limited, resolves in 2–3 wk, discontinue offending agent
› Short course of corticosteroids can be used for severe arthralgias

Disposition
› Home
HENOCHE–SCHÖNLEIN PURPURA

Definition
¬ Small-vessel vasculitis

History
¬ Age 2–11 yr; preceding respiratory infection (Group A β-hemolytic Strep); fever, arthralgia, abd pain, bloody stools, hematuria

Physical Findings
¬ Palpable purpura in dependent regions, fever, joint swelling, guaiac positive, scrotal edema

Diagnosis
¬ Clinical; CBC (↑ WBC, ↑ PLT, anemia), ↑ ESR, antistreptolysin antibodies (+ in 50%), UA (hematuria, proteinuria, pyuria), abd U/S (intussusception), scrotal U/S

Treatment
¬ Majority is self-limiting w/ resolution in a few weeks; supportive, NSAIDs, treat underlying infection
¬ Corticosteroids does not prevent recurrences, which occur in 50%; but can be used for severe arthritis, renal involvement, GI, scrotal or CNS cx

Disposition
¬ Home unless cx: HTN, oliguria, obstruction, intussusception, GIB

Complications
¬ Bowel obstruction, perforation, intussusception, renal failure, hypertensive encephalopathy, acute scrotum (mimics torsion), pancreatitis, CNS cx (sz, coma, neuro deficits)

URINARY TRACT INFECTION

(Pediatrics 2011;128;595)

History
¬ Adolescents: Dysuria, urgency, frequency, hematuria; fever; flank pain,
abd pain
- Younger children: Enuresis, foul-smelling urine, abd pain, N/V
- Infants: Fever, irritability, poor feeding, vomiting, jaundice, FTT

Physical Exam
- Fever, suprapubic tenderness, bladder fullness; CVA tenderness; GU exam to assess for vaginitis

Evaluation
- **Labs:** UA/Ucx (may require straight cath for clean specimen); Chem 7 (dehydration), CBC/blood cultures (if considering sepsis)
- Renal U/S in febrile infant or young child b/w 2 mo & 2 yr w/ 1st UTI
- VCUG for recurrent infections, poor urinary stream, palpable kidneys, unusual organism, HTN, bacteremia or sepsis that fails to respond to abx, unusual presentation, or hydronephrosis/scarring seen on renal U/S

Treatment
- Supportive: Oral rehydration if child able to tolerate o/w establish IV for hydration
- Abx (usually *E. coli*):
  - IV: Cefotaxime, ceftriaxone, gentamicin
  - PO: Augmentin, Bactrim, cefixime, cefpodoxime

Disposition
- Home: Stable, tolerating POs, nontoxic appearing; PCP f/u in 2–3 d
- Admit: <2 mo old, toxic appearing, unable to tolerate POs, signs of urinary obstruction, suspected sepsis, underlying comorbidities, ↑ Cr
PSYCHIATRIC PATIENT

Approach
- Always consider medical disorders → esp if no previous psych hx
- Anticipate need for psychiatry consult & restraints (meds, physical) early to assure safety

Definition
- Medical clearance: An ambiguous term suggesting no “organic” cause for pt's psych complaint; however, pts can have medical condition that exacerbates their psychiatric presentation (ie, drug abuse, infection)
- Focused medical assessment: The process of excluding medical illnesses that require acute care to determine who is medically stable (Ann Emerg Med 2006;47:79)

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurologic</td>
<td>Brain tumor, head trauma, encephalopathy, epilepsy, dementia, hydrocephalus, CVA, ICH, migraine, vasculitides</td>
</tr>
<tr>
<td>Other</td>
<td>Porphyria</td>
</tr>
<tr>
<td>Infections</td>
<td>Meningitis, encephalitis, UTI, PNA</td>
</tr>
<tr>
<td>Medications</td>
<td>Polypharmacy, benzos, anticholinergics, SSRIs, opioids, Dig, furosemide, warfarin, hydrochlorothiazide</td>
</tr>
<tr>
<td>Toxicologic</td>
<td>EtOH, substance abuse, overdoses, withdrawal</td>
</tr>
<tr>
<td>Metabolic/endocrine</td>
<td>Hypo/hyperglycemia, hypoxia, thyroid, parathyroid dz, electrolyte abnlty, hyper/hypocortisolism</td>
</tr>
</tbody>
</table>

Depression/Suicidality (Emerg Med Clin North Am 2015;33:765)

History
- Ask open-ended questions about thoughts, feelings, personal relationships; drug use; prior hospitalizations/psych hx; psych medications; physical/sexual abuse
Sxs (SIG E CAPS): Sleep, Interest, Guilt, Energy level decreased, Concentration, Appetite, Psychomotor activity, Suicidal ideation
SI/HI: Access to weapons, plan, prior SI/HI or attempt; command hallucinations
Risk of suicide (SAD PERSONS): Sex (male), Age (<19, >45), Depression, Previous attempt, EtOH abuse, Rational thinking loss, Social support lacking, Organized plan, No spouse, Sickness

Findings
- Abnl VS; appearance, mental status exam
- Head-to-toe exam: E/O trauma, pupils, nystagmus, thyroid, pulm/cardiac/abdomen, skin
- Neuro: CNs, DTRs, motor, sensory, cerebellar, asterixis, gait, catatonia

Evaluation
- There is no data to support routine use of lab testing in psych pts whose H&P exclude significant medical illness
- βhCG (all women reproductive age), consider ECG & psych med levels (ie, Li)
- Tox: If concern for unreported drug abuse or ingestion (ie, APAP)
- Psychiatry consult: If needed for hospitalization, suicide/homicide attempt, uncertain at risk of danger to self/others
- Other labs: If concern for “organic” d/o or required for psych hospital: CBC, Chem 7, LFTs, UA, TSH, ammonia, CXR
- More thorough w/u is necessary for new onset psych Dx: Consider RPR, CT head, LP, EEG

Treatment
- Treat any underlying medical illness
- Typically antidepressants are not initiated by the ED physician

Anxiety/Panic Disorder

History
- Associated physical sxs (CP, SOB), substance use, prior similar episodes, current life stressors
- SI/HI (see above)

Findings
- Look for clues for underlying medical conditions: abnl VS (tachycardia, hypoxia), trauma, thyroid, nystagmus, cardiopulmonary exam
Evaluation
- Consider EKG, CXR or other cardiopulmonary testing

Treatment
- Treat any underlying medical illness
- BZD may help in acute setting but are a/w possible abuse & rebound

Psychosis *(Emerg Med Clin North AM 2015;33:739.)*

History
- Often challenging if pt is unable to interact appropriately
- Hallucinations? Delusions? Disorganized thoughts or speech?

Findings
- Look for clues for underlying medical conditions: abnl VS, trauma, nystagmus, thyroid, asterixis, focal neurologic deficits, fluctuating sx, nonauditory hallucinations

Evaluation
- There is no data to support routine use of lab testing in psych pts whose H&P exclude significant medical illness
- βhCG (all women reproductive age), consider ECG & psych med levels (ie, Li)
- Tox: If concern for unreported drug abuse or ingestion (ie, APAP)
- Psychiatry consult: If needed for hospitalization, suicide/homicide attempt, uncertain at risk of danger to self/others
- Other labs: If concern for “organic” d/o or required for psych hospital: CBC, Chem 7, LFTs, UA, TSH, ammonia, CXR
- More thorough w/u is necessary for new onset psych Dx: Consider RPR, CT head, LP, EEG

Treatment
- Nonpharmacologic strategies: creating a safe environment, seclusion, verbal de-escalation
- Medications:
  - Haldol (IM/IV), ziprasidone (IM), olanzapine (PO/SL/IM); side effects ↑QT, akathisia, dystonia
  - Lorazepam/diazepam (PO/IV/IM): preferred for drug-related agitation; avoid in the elderly
- Physical restratints: Soft/leather (1–4 point), posy, mitts. Use as
temporizing measure in conjunction w/ pharmacologic tx & sitter.

- Should attempt to use least restrictive strategies for the shortest time possible

**Pearls**

- Signs suggestive of “organic disorder”: Age >40 w/ no prior psychiatric hx, abnl VS, recent memory loss, clouded consciousness
- Engage family members/friends/partners for collateral whenever possible
GENERAL APPROACH TO THE INTOXICATED PATIENT

Approach
- (1) ABCs, resuscitate/stabilize → (2) decontaminate (GI tract, skin, eyes)/enhance elimination (charcoal, dialysis) → (3) treat w/ antidote, if available & indicated
- Consider empiric naloxone, dextrose, thiamine in pts w/ depressed MS. Use flumazenil w/ caution as it may precipitate sz
- Call Poison Control Center: 1 (800) 222-1222

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Toxidrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticholinergics</td>
<td>↑ Temp, ↑ HR, dry skin, mydriasis, dry MM, AMS, urinary retention, sz, coma</td>
</tr>
<tr>
<td>Sympathomimetics</td>
<td>↑ Temp, ↑ HR, diaphoresis, mydriasis, agitation, dysrhythmia, sz, coma</td>
</tr>
<tr>
<td>Sympatholytic</td>
<td>↓ HR, ↓ BP, miosis, ↓ peristalsis</td>
</tr>
<tr>
<td>Opioids</td>
<td>AMS, ↓ RR, miosis</td>
</tr>
<tr>
<td>Anticholinesterases</td>
<td>DUMBELS* + muscle weakness, AMS, sz, coma</td>
</tr>
<tr>
<td>Sedative-hypnotics</td>
<td>↓ BP, ↓ RR, AMS, ↓ temp, slurred speech, ataxia</td>
</tr>
<tr>
<td>Alpha-adrenergic</td>
<td>↑ BP, ↓ RR, mydriasis, moist skin</td>
</tr>
</tbody>
</table>

*Defecation, urination, miosis, bronchorrhea, bronchospasm, bradycardia, emesis, lacrimation, salivation

History
- Always consider Drug, Dose, & Pt: timing, quantity of ingestion/exposure, access to household chemicals/other meds, coingestions, enteric-coated/extended-release substances

Physical Exam
VS, pupils, skin, neuro findings (AMS, nystagmus, myoclonus, tremor), peristalsis, smell

**Evaluation**
- ECG, FSG, CBC, BMP, LFTs, UA, ABG, hCG, osmolar/anion gap
- Drug levels
  - Exposures for which drug level is useful: APAP, salicylates, theophylline, lithium, Dig, EtOH, carboxyhemoglobin, methemoglobin, iron, methanol, ethylene glycol, lead, mercury, arsenic, organophosphate, anticonvulsants

**Treatment**

<table>
<thead>
<tr>
<th>GI Decontamination</th>
<th>Tx</th>
<th>Indications</th>
<th>Dose</th>
<th>Relative CIs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activated charcoal</td>
<td>Given ideally w/i 1 h from ingestion</td>
<td>50 g (adults) 25 g (children) Give w/ antiemetics</td>
<td>Concern for bowel perforation, obstruction, aspiration, acid/alkali ions, EtOH, lithium, iron poorly adsorbed, AMS</td>
<td></td>
</tr>
<tr>
<td>Whole bowel irrigation</td>
<td>Significant ingestion not absorbed by charcoal or bags of illicit drugs</td>
<td>PEG via NGT 2 L/h (children 500 mL/h) until clear rectal effluent</td>
<td>Low-risk ingestion, risk of aspiration, toxin absorbed by charcoal, ileus or obstruction, obtundation</td>
<td></td>
</tr>
</tbody>
</table>

**Dermal Decontamination**
- Irrigation w/ copious volumes of H₂O (unless metallic Na, K, or phosphorus)

**Ocular Decontamination**
- Irrigation w/ copious volumes of H₂O, check pH after irrigation

**Enhanced Elimination**
- Urinary alkalinization w/ NaHCO₃ (eg, salicylates, phenobarbital, formic acid)
- HD (eg, ethylene glycol, methanol, lithium, salicylates, severe acidosis)

<table>
<thead>
<tr>
<th>Common Toxicology Tx</th>
<th>Toxicologic Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antivenom</td>
<td>Snake, black widow spider, brown recluse spider, scorpion</td>
</tr>
<tr>
<td>Drug</td>
<td>Mechanism</td>
</tr>
<tr>
<td>------</td>
<td>-----------</td>
</tr>
<tr>
<td>Botulinum antitoxin</td>
<td><em>Clostridium botulinum</em></td>
</tr>
<tr>
<td>Calcium</td>
<td>CCB, ↑ K, ↑ Mg, ↓ Ca, hydrofluoric acid</td>
</tr>
<tr>
<td>Edetate calcium disodium</td>
<td>Lead</td>
</tr>
<tr>
<td>Cyanide kit (amyl nitrite, sodium nitrite, thiosulfate); Cyanocobalamin</td>
<td>Cyanide, smoke inhalation</td>
</tr>
<tr>
<td>Deferoxamine</td>
<td>Iron</td>
</tr>
<tr>
<td>Dig antibody fragments</td>
<td>Dig</td>
</tr>
<tr>
<td>Dimercaprol</td>
<td>Arsenic, lead, mercury</td>
</tr>
<tr>
<td>ETOH</td>
<td>Ethylene glycol, methanol</td>
</tr>
<tr>
<td>Flumazenil</td>
<td>Benzos</td>
</tr>
<tr>
<td>Fomepizole</td>
<td>Ethylene glycol, methanol</td>
</tr>
<tr>
<td>Glucagon</td>
<td>βB, CCB</td>
</tr>
<tr>
<td>Hyperinsulinemia–euglycemia therapy</td>
<td>βB, CCB</td>
</tr>
<tr>
<td>N-acetylcysteine</td>
<td>APAP</td>
</tr>
<tr>
<td>Naloxone</td>
<td>Opioids</td>
</tr>
<tr>
<td>Octreotide</td>
<td>Sulfonylureas</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>Anticholinergics</td>
</tr>
<tr>
<td>Pralidoxime</td>
<td>Organophosphates</td>
</tr>
<tr>
<td>Protamine</td>
<td>Heparin</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>TCAs</td>
</tr>
<tr>
<td>Succimer</td>
<td>Arsenic, lead, mercury</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>Coumadin</td>
</tr>
</tbody>
</table>

**Disposition**

- Admit for any significant ingestion/exposure; consider transfer for complex presentations & inadequate hospital resources

**Pearl**

- Hospital tox screens vary → know your hospital’s screen to guide your practice
ANTICHOLINERGIC INGESTION

Definition
- Antagonists @ **muscarinic** cholinergic receptor → inhibit parasymp system

<table>
<thead>
<tr>
<th>Class</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Belladonna alkaloids</td>
<td>Atropine, scopolamine, ipratropium</td>
</tr>
<tr>
<td>Antiparkinsonian agents</td>
<td>Benztropine</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>Diphenhydramine (Benadryl), meclizine (Antivert), promethazine (Phenergan), hydroxyzine (Atarax), dimenhydrinate (Dramamine)</td>
</tr>
<tr>
<td>Cyclics</td>
<td>Cyclobenzaprine (Flexeril)</td>
</tr>
<tr>
<td>Psychopharmacologics</td>
<td>TCA, phenothiazines</td>
</tr>
</tbody>
</table>

History
- AMS w/ medication exposure, ingestion hx, teas, supplements, or polypharmacy

Differential
- Sympathomimetic OD, EtOH/benzo withdrawal, thyroid storm, sepsis, meningitis, hypoglycemia

Findings
- ↑ HR, ↑ temp, dilated pupils, dry MM/skin, ↓ bowel sounds, urinary retention, myotonic activity, choreoathetosis, confusion/delirium, sz; “blind as a bat, dry as a bone, hot as a hare, mad as a hatter, red as a beet, bloated as a toad”

Evaluation
- ECG (↑ QRS, QT_c → TCAs, neuroleptics); electrolytes; total CK (rhabdomyolysis); tox screen → r/o other ingestions; pulse ox; tele

Treatment
- **Supportive:** IV hydration, external cooling
- **Decontamination/elimination:** Activated charcoal (1 dose, w/i 1 h),
HD

- **BZD (IV):** For agitation, szs
- **Physostigmine (IV):** Reverses anticholinergic effects via acetylcholinesterase inhibition
  - NOT for routine use due to risk of intractable szs, AV block, asystole
  - Half-life of physostigmine often shorter than toxidrome!

**Disposition**

- Admit; ICU for pts w/ cardiac instability or szs

**Pearl**

- Rarely fatal unless significant hyperthermia is present

---

**PSYCHOPHARMACOLOGIC INGESTION**

**Selective Serotonin Reuptake Inhibitors and Serotonin Syndrome Approach**

- Spectrum for serotonin intoxication ranges from mild lethargy to serotonin syndrome
- Consider serotonin syndrome for anyone on meds w/ serotonin activity, esp ≥2 agents
- Greatest risk w/i minutes to hours after starting new med or increasing dose of old med

**Definition**

- SSRI: Selective serotonin reuptake inhibitors; SRIs: Serotonin reuptake inhibitors (also exhibit activity on epinephrine, norepinephrine, dopamine)

<table>
<thead>
<tr>
<th>Common Drugs That Inhibit Serotonin Reuptake</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SSRIs</strong></td>
</tr>
<tr>
<td>Fluoxetine, paroxetine, sertraline, citalopram</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Drugs w/ Serotonin Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antidepressants</strong></td>
</tr>
<tr>
<td>SSRIs, SRIs, MAOIs,</td>
</tr>
<tr>
<td>lithium</td>
</tr>
</tbody>
</table>

### Differential for Serotonin Intoxication

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toxic ingestion</td>
<td>Sympathomimetics (16f), MAOIs, lithium, salicylates (16g), anticholinergics (16b), NMS</td>
</tr>
<tr>
<td>Chemical withdrawal</td>
<td>EtOH (16e), sedative-hypnotics (16d)</td>
</tr>
<tr>
<td>Infection</td>
<td>CNS (4c), SIRS (1f)</td>
</tr>
<tr>
<td>Other</td>
<td>Thyrotoxicosis (9d), tetanus (4i), malignant hyperthermia (10k)</td>
</tr>
</tbody>
</table>

### History
- Akathisia, AMS, szs

### Findings
- ↑ HR, ↑ temp, ↑ reflexes, diaphoresis, mydriasis, ↑ ↓ BP, tremor, clonus, neuromuscular rigidity, ataxia

### Evaluation
- VS, CBC, BMP, CK (rhabdo), ECG (↑ QRS, ↑ QT_c, torsades), pulse ox, Tele

### Treatment

#### Acute Overdose
- Activated charcoal, admit for monitoring

#### Serotonin Syndrome
- Supportive: IV fluids, electrolyte correction, external cooling (may require sedation/paralysis for severe hyperthermia)
- Benzos (IV): For agitation, rigidity, szs
- (Controversial) Consider cyproheptadine (12 mg initially, 4 mg PO q1h), chlorpromazine 25–50 mg IV for severe sx

### Pearls

#### Characteristics of Serotonin Syndrome vs. Neuroleptic Malignant Syndrome

<table>
<thead>
<tr>
<th>Signs &amp; Sxs</th>
<th>Serotonin Syndrome</th>
<th>NMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Sudden</td>
<td>Often over days to weeks</td>
</tr>
<tr>
<td>Resolution</td>
<td>w/i 24 h</td>
<td>Over ~1 wk</td>
</tr>
<tr>
<td>Condition</td>
<td>Commonity</td>
<td>Uncommonity</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>Common</td>
<td>VERY common</td>
</tr>
<tr>
<td>AMS</td>
<td>Common</td>
<td>VERY common</td>
</tr>
<tr>
<td>Autonomic dysfxn</td>
<td>Common</td>
<td>VERY common</td>
</tr>
<tr>
<td>Muscle rigidity</td>
<td>Common</td>
<td>VERY common</td>
</tr>
<tr>
<td>↑ Total CK</td>
<td>Uncommon</td>
<td>VERY common</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Uncommon</td>
<td>VERY common</td>
</tr>
<tr>
<td>↑ Reflexes</td>
<td>VERY common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Myoclonus</td>
<td>VERY common</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>

### Neuroleptics, Neuroleptic Malignant Syndrome

#### Definition
- Characterized by D₂ antagonism ± serotonin receptor antagonism

#### Common Neuroleptics

<table>
<thead>
<tr>
<th>Typical Neuroleptics</th>
<th>Atypical Neuroleptics</th>
<th>Antiemetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine, haloperidol</td>
<td>Aripiprazole, clozapine, olanzapine, quetiapine, risperidone, ziprasidone</td>
<td>Promethazine, prochlorperazine, droperidol</td>
</tr>
</tbody>
</table>

#### History
- Slurred speech, sedation, anticholinergic toxicidrome, extrapyramidal sxes (dystonia, akathisia, parkinsonism, tardive dyskinesia)
- **NMS:** ↑ HR, rigidity, AMS, szs, autonomic instability, metabolic acidosis, rhabdomyolysis

#### Evaluation
- CBC, BMP, CK (rhabdo), ECG (↑QT<sub>c</sub>, torsades, dysrhythmia), UA (myoglobin)

#### Treatment
- Dystonia/akathisia: Diphenhydramine, benztropine, BZD
- **NMS:** External/Internal cooling, IV fluids, benzos, nondepolarizing neuromuscular blockade, dantrolene, bromocriptine, amantadine

---

**Lithium**
### Clinical Effects

<table>
<thead>
<tr>
<th>System</th>
<th>Side Effects</th>
<th>Acute Overdose</th>
<th>Chronic Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>N/V, diarrhea, abd pain</td>
<td>N/V, diarrhea</td>
<td>N/V</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Tremor, weakness</td>
<td>Tremor, rigidity, clonus, ↑ reflexes, lethargy, sz, coma</td>
<td>Tremor, rigidity, pseudotumor cerebri, tinnitus, ataxia, blurred vision, sz, coma</td>
</tr>
<tr>
<td>CV</td>
<td>Sinus node dysfxn</td>
<td>↓ BP</td>
<td>↓ BP, ↓ T-wave, ↓ ST seg, sinus node dysfxn, ↑ QTc</td>
</tr>
<tr>
<td>Renal</td>
<td>Polyuria</td>
<td>—</td>
<td>Nephrogenic DI (↑Na), interstitial nephritis, renal acidosis</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Goiter, ↓ thyroid</td>
<td>—</td>
<td>Goiter, ↑ or ↓ thyroid, ↑ Ca</td>
</tr>
</tbody>
</table>

### History
- **Acute tox:** GI sx initially; neurologic findings may develop later
- **Chronic tox:** Neurologic sxs

### Severity of Lithium Toxicity

<table>
<thead>
<tr>
<th>Grading of Toxicity</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>N/V, tremor, ataxia, muscle weakness, ataxia</td>
</tr>
<tr>
<td>2</td>
<td>Rigidity, hypertonia, ↓ BP, stupor</td>
</tr>
<tr>
<td>3</td>
<td>Coma, sz → D</td>
</tr>
</tbody>
</table>

### Evaluation
- VS, ECG, CBC, BMP, Ca, Mg, PO4, TSH, free T4, UA
- Lithium level: Not useful in acute ingestion (development of neurologic sx is better reflection of tox); in chronic tox, level >1.5 mEq is significant
- Assess for causes of decreased lithium clearance (eg, dehydration, renal failure)

### Treatment
- IV fluids: Decreases tox & promotes Li excretion, NS bolus then ½ NS
- GI decontamination: Activated charcoal ineffective, whole bowel irrigation may be useful
- Sodium polystyrene sulfonate (Kayexalate), consider thiazides, indomethacin, or amiloride for nephrogenic DI
- BZD for szs (avoid phenytoin, which ↓ Li renal excretion)
- HD: For pts w/ severe neurologic szs &/or clinical deterioration, Li level >3.5

**Disposition**
- Admit all pts w/ sustained release ingestions, Li level >1.5 mEq, or new neurologic signs; lesser ingestions can be treated & observed 4–6 h → rev level ± psychiatry eval

**Pearl**
- Li has very narrow therapeutic window; consider Li tox in pts w/ ARF/↓ UOP

---

**TRICYCLIC ANTIDEPRESSANTS**

**Approach**
- Sxs of overdose almost always occur w/i 6 h of ingestion

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Physiologic Mechanism of Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histamine antagonist</td>
<td>Clinical Manifestations</td>
</tr>
<tr>
<td></td>
<td>Sedation, coma</td>
</tr>
<tr>
<td>ACh (muscarinic) antagonist</td>
<td>↑ HR, ↑ BP, mydriasis, dry skin, ileus, urinary retention</td>
</tr>
<tr>
<td>α1-adrenergic antagonist</td>
<td>Sedation, orthostatic ↓ BP, miosis (can counteract muscarinic mydriasis)</td>
</tr>
<tr>
<td>Amine reuptake inhibition</td>
<td>↑ HR, myoclonus, ↑ reflexes</td>
</tr>
<tr>
<td>Na channel inhibition</td>
<td>↑ PR/QRS intervals, RAD, ↓ cardiac contractility, heart block</td>
</tr>
<tr>
<td>K channel antagonist</td>
<td>↑ QT interval → torsades de pointes</td>
</tr>
<tr>
<td>GABA-A antagonist</td>
<td>Szs</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Severity of TCA Toxicity</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degree of Toxicity</td>
<td>Drowsiness, confusion, slurred speech, ataxia, dry MM, ST,</td>
</tr>
</tbody>
</table>
urinary retention, myoclonus, ↑ reflexes

| Severe | SVT, ↑ QRS, ↑ PR, ↑ QT, VT, ↓ BP, sz, coma |

### Evaluation
- **ECG**, CBC, BMP, Ca/Mg/PO4, CK, UA tox screen, pulse ox, Tele

### Treatment
- Supportive: IV fluids
- GI decontamination/elimination: Activated charcoal ± **gastric lavage**, intralipid for clomipramine
- **Sodium bicarbonate**: 1–2 mEq/kg boluses titrated to pH 7.45–7.55
- **Indications**: QRS >100, new RAD, ↓ BP, &/or ventricular dysrhythmia
- BZD: For szs
- Lidocaine: For ventricular dysrhythmias refractory to NaHCO₃, avoid procaainamide or other type Ia or Ic antiarrhythmics
- Lipid emulsion: Case reports only, 1.5 mg/kg bolus followed by 400 mL infusion over 30 min

### Disposition
- Admit all pts w/ e/o cardiotoxicity or sz; d/c pts w/o sxs at 6 h after ingestion

### Pearl
- Antimuscarinic effects are absent in many cases of TCA overdose

---

## ALCOHOLS

### Definition
- Ingestions of toxic alcohols

### Approach
- Hx
- Type of alcohol ingested, time of ingestion, coingestants
- PE: Monitor for airway protection, occult trauma (head injury)
- Labs: FSG (may be all that’s needed), consider BAL (declines ~20 mg/dL/h), anion gap, serum/urine tox (if coingestants suspected), osmolar gap for alcohols other than EtOH
- Osmol calc = 2 × Na + BUN/2.8 + glucose/18 + EtOH/4.6
- Osmol gap = Osmol measured – osmol calc
- Tx: Charcoal doesn’t bind alcohol, ±thiamine/folate

<table>
<thead>
<tr>
<th>Alcohol Ingestion Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td>EtOH</td>
</tr>
<tr>
<td>Methanol</td>
</tr>
<tr>
<td>Ethylene glycol</td>
</tr>
<tr>
<td>Isopropyl alcohol</td>
</tr>
</tbody>
</table>

**Ethanol**

**History**
- EtOH ingestion, found down, lethargy, N/V, ± associated trauma, ± aspiration, gastritis

**Physical Findings**
- CNS, respiratory depression, slurred speech, ataxia, nystagmus

**Evaluation**
- FSG (hypoglycemia common in alcoholics), ± BAL (if ingestion uncertain), ± CBC/BMP/LFTs/lipase, ± ECG (if pulse if irregular), ± magnesium level

**Treatment**
- Maintain airway, serial exams, ± IVF/thiamine/folate (given but may not be necessary)

**Disposition**
- Ambulating w/o ataxia + speaking clearly → d/c

**Pearls**
- R/o head trauma, CNS infection, Wernicke encephalopathy, alcoholic ketoacidosis, hypoglycemia, alcohol withdrawal/DT, coingestions, SI/HI
- Known EtOH ingestion/intoxication in pt w/ hx of same does not require lab & can be observed until clinically sober

**Methanol**
**Definition**
- Ingestion of methanol (peak levels 30–60 min, 24–30 h ½ life, hepatic metabolism)

**History**
- Drinking: Paint solvents/antifreeze/windshield-washing fluid/canned fuels/gasoline additives, shellac/copy machine fluid/home heating fuels

**Physical Findings**
- CNS depression, vomiting, papilledema/hyperemia, visual Δ/loss, gastritis

**Evaluation**
- ↑ Methanol level, ↑ Osmol gap, ↑ anion gap (profound), BMP, ABG

**Treatment**
- Based presumptive Dx if levels delayed, maintain airway
- Fomepizole: Loading dose (15 mg/kg in 100 mL D5W over 30 min) → maintenance (10 mg/kg q12h × 4 doses → 15 mg/kg q12 to methanol concentration <20/dL)
- Folate 50 mg IV q4h until resolution of acidemia (cofactor to convert formic acid → CO₂ + H₂O)
- Dialysis: Absolute indications → visual impairment + detectible methanol level or >50 mL/dL, osmol gap >10, ingestion >1 mg/kg, severe acidosis, renal failure

**Disposition**
- Admit

---

**Ethylene Glycol**

**Definition**
- Ingestion of ethylene glycol (peak levels 30–180 min, 3–7 h ½ life, 70% hepatic metabolism)

**History**
- Drinking: Antifreeze, coolants, paint, polishes, detergents, fire extinguishers

**Physical Findings**
3 phases: <12 h → ↓ CNS (like EtOH), gastritis; 12–24 h → ↑ HR/RR/BP/SOB; >12 h → ATN (oxalate crystal deposition)

**Evaluation**
- Ethylene glycol level, ↑ osmol gap, ↑ AG, calcium oxalate crystals in urine, beta-hydroxybutyrate (used to distinguish from alcoholic ketoacidosis)

**Treatment**
- Based presumptive Dx if levels delayed, maintain airway
- Fomepizole: Loading dose (15 mg/kg in 100 mL D₅W over 30 min) → maintenance (10 mg/kg q12h × 4 doses → 15 mg/kg q12h to ethylene glycol concentration <20/dL)
- Folate/thiamine 100 mg IV q6h/pyridoxine 50 mg IV q6h until resolution of acidemia (cofactors in oxalic acid metabolism)
- HD: Severe acidosis (pH <7.25) + osmol gap >10, renal failure (Cr >1.2 mg/dL), ethylene glycol level >50 mg/dL, deterioration despite supportive care

**Disposition**
- Admit

**Clinical Pearl**
- Urine/gastric contents fluoresce w/ Wood lamp due to antifreeze additives (early)

---

**ISOPROPYL ALCOHOL**

**Definition**
- Ingestion of isopropyl alcohol (peak levels 30–180 min, 3–7 h ½ life, 80% hepatic metabolism, lethal dose 2–4 mL/kg)

**History**
- Drinking: Rubbing alcohol, paint thinner, solvents, skin/hair products, nail polish remover

**Physical Findings**
- Profound ↓ CNS (2–4 × EtOH), fruity odor on breath, respiratory depression, ↓ BP, gastritis

**Evaluation**
- BMP, UA, FSG, isopropyl level, nl AG, ↑ osmol gap, falsely ↑ Cr (from acetone)

**Treatment**
- Based presumptive Dx if levels delayed
- Supportive (rarely lethal)
- Dialysis: Refractory hypotension, levels >500 mg/dL

**Disposition**
- Admit if severe toxicity, may D/C 2 h after resolution of sx if no coingestions or SI

---

**ALCOHOL WITHDRAWAL**

**Definition**
- Abrupt cessation or significant reduction in alcohol intake (begins 6–24 h/peaks 48–72 h after last drink)

**History**
- Heavy alcohol use w/ cessation, insomnia, anorexia, N/V, restlessness, diaphoresis, sz

**Physical Findings**
- Tremulousness, szs (25% of pts at 6–48 h), delirium, hallucinations (visual > auditory), autonomic hyperactivity (tachycardia, HTN, irritability, hyperreflexia), delirium tremens (rare/serious, 24 h–5 d after last drink): Tremor/autonomic hyperactivity/confusion/hallucinations/low-grade fever

**Evaluation**
- FSG, CBC, BMP, LFTs/coags (if liver dysfxn suspected), BAL

**Treatment**
- Glucose (if hypoglycemic), thiamine, lorazepam 2 mg IV for sz, IV/IM/PO long-acting BZD (ie, lorazepam 1–4 mg IV q10–30min to sedation, diazepam 5–10 mg IV q5–10min to sedation, chlordiazepoxide 25–100 mg PO q1h), phenobarbital as 2nd-line

**Disposition**
- Admit if requiring IV medication/DTs ± ICU
Clinical Pearls
- Rarely fatal (increased w/ aspiration due to sz) when treated appropriately
- May require very large doses of IV BZD to control/treat

---

**DRUGS OF ABUSE**

<table>
<thead>
<tr>
<th>Focused Differential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class</strong></td>
</tr>
<tr>
<td>Sedative-hypnotics</td>
</tr>
<tr>
<td>Stimulants/sympathomimetics</td>
</tr>
</tbody>
</table>

---

**BENZODIAZEPINES**

**Definition**
- GABA agonists

**History**
- Usually suicidal gesture or abuse, hypnotic/sleep agents (zaleplon, zolpidem, eszopiclone) have similar effects as BZD in overdose

**Physical Findings**
- CNS, respiratory depression, slurred speech, ataxia, hyporeflexia, midpoint/small pupils, hypothermia, hypotension

**Evaluation**
- FSG, consider ABG, Serum/urine tox, ETCO₂ monitor, Tele, pulse ox if severe toxicity

**Treatment**
- Supportive (airway protection if needed), Flumazenil 0.1–0.2 mg,
repeat up to 3 mg → may precipitate szs, indications are rare, use only to reverse when known benzo is overadministered as part of procedural sedation & must be reversed for life-threatening sx/s, monitor for resedation after 1–2 h, may require repeat dose

- Decontamination: Activated charcoal if ingestion occurred w/i 30 min

### Disposition
- Home (rarely require admission) if resolution of sx/s after monitoring & if no SI

### Pearls
- Monitor for withdrawal, which is similar in presentation (agitation, szs) & tx to EtOH withdrawal
- Isolated benzo OD rarely life-threatening although usually presents as polysubstance OD

---

**Gamma-Hydroxybutyrate (GHB)**

### Definition
- GABA & GHB receptor agonist

### Physical Findings
- Initial euphoria, AMS/obtundation, hypothermia, bradycardia, hypotension, sz, respiratory depression, myoclonus, aspiration, rarely pulmonary edema & sz

### Evaluation
- FSG, ± Serum/urine tox screen (rapidly metabolized → GHB levels not readily available)

### Treatment
- Supportive, maintain airway, recovery w/i 2–4 h, resolution w/i 8 h

### Disposition
- Home

---

**Opioids**

### Definition
- Opioid receptor agonist
History

- Witnessed or reported use of opioids (heroin, methadone, morphine, hydromorphone, fentanyl, oxycodone)

Physical Findings

- ↓ CNS, ↓ RR/BP, apnea, ± miosis, track marks, aspiration, noncardiogenic pulmonary edema

Evaluation

- Glucose, serum/urine tox screen (for coingestants), end-tidal CO₂ monitor, Tele, pulse ox

Treatment

- Maintain airway
- Naloxone (titrate to effect) 0.2–0.4 mg IV → 1 mg IV → 2 mg IV → IV drip (duration 1–2 h)
- Activated charcoal (recent ingestion), whole bowel irrigation (long-acting opioid)

Disposition

- May require ICU admission for long-acting opioid toxicity on naloxone drip

Pearls

- Pts die from untreated apnea, often in prehospital setting
- Pts w/ hypoxia/cyanosis have risk of aspiration/ARDS
- Pts w/ recurrent apnea after naloxone likely have longer-acting opioid

Opioid Withdrawal

Definition

- Cessation or rapid reduction of opioid use in a dependent individual

History

- Chronic opioid use, anxiety, N/V, abd pain, diarrhea, myalgias

Physical Findings

- Yawning, rhinorrhea, mydriasis, piloerection, tachycardia

Treatment

- Clonidine 0.1 mg PO q30–60min (central α-agonist) → ↓ duration, methadone (not indicated in the ED), IVF

Disposition
COCAINENot life-threatening, do not require admission, may be precipitated by administration of naloxone & caution should be used before treating w/additional opioids

Definition
Snorting, injecting, smoking, ingesting (body packing vs. stuffing) cocaine (peak 5–15 min, duration 1–4 h, releases norepinephrine/blocks reuptake)

History
Cocaine use, anxiety, CP, focal weakness (CVA/ICH), sz, psychosis

Physical Findings
↑ HR, ↑ BP, hyperthermia, diaphoresis, agitation, nasal septal perforation, mydriasis

Evaluation
Serum/urine tox screen, cardiac markers (if CP present), ECG (↑ QRS, ischemia), Cr (renal failure), CK (rhabdomyolysis), head CT (if ICH suspected), consider aortic dissection, intestinal infarction, stroke

Treatment
Supportive care, BZD for anxiety/agitation/CP, treat hyperthermia (ice packs, cooling blankets, cooling mist), avoid βBs (unopposed α-adrenergic stimulation)
Activated charcoal (recent ingestion), whole bowel irrigation (packers/stuffers)

Disposition
Varies depending on severity & mechanism of toxicity

Pearl
Cocaine wash-out syndrome: After cocaine binging, MS (lethargy, obtundation), lasts up to 24 h

METHAMPHETAMINE (“METH”)
**Definition**
- Norepinephrine release, dopaminergic (causes addiction)

**History**
- Ingestion, snorting, smoking, injection, rectal insertion of methamphetamines & derivatives (LSD, bath salts), ADHD, & narcolepsy medications

**Physical Findings**
- ↑ HR, ↑ BP, hyperthermia, diaphoresis, agitation, poor dentition (“meth mouth”), poor hygiene, compulsive scratching lesions (“meth mites”), tremors, sz

**Evaluation**
- Serum/urine tox screen, ECG, consider CT head (ICH), UA, CK (rhabdomyolysis), BMP, cardiac enzymes (CP), Tele

**Treatment**
- Supportive care, BZD for anxiety/agitation/CP, cool hyperthermic pts (ice packs, cooling blankets, cooling mist)
- Activated charcoal (recent ingestion), whole bowel irrigation (packers/stuffers)

---

**METHYLENEDIOXYMETHAMPHETAMINE (MDMA, “Ecstasy”), LYSERGIC ACID DIETHYLAMIDE (LSD)**

**Definition**
- Serotonergic

**History**
- Ingestion of MDMA, LSD, other hallucinogens

**Physical Findings**
- ↑ HR, ↑ BP, hyperthermia, anxiety, mydriasis, hallucinations, sz, diaphoresis, bruxism

**Evaluation**
- Serum/urine tox screen, BMP (↓ Na due to excessive water ingestion), ECG, consider CT head (ICH), INR, UA, CK (rhabdomyolysis), cardiac enzymes (CP), Tele
Treatment

- Supportive care, BZD & haloperidol for agitation, cool hyperthermic pts (ice packs, cooling blankets, cooling mist)
- ± Activated charcoal (recent ingestion)

**ANALGESIC OVERDOSE**

**ACETAMINOPHEN (APAP) POISONING**

**History**

- Witnessed or reported ingestion of any APAP-containing meds (many Rx & OTC drugs)
- Often coingestions w/ other substances

**Findings**

- 4 stages of APAP poisoning
  i. Asymptomatic (0–24 h)
  ii. GI upset, N/V, abd pain (24–72 h)
  iii. Jaundice, fulminant liver failure, encephalopathy (3–5 d)
  iv. Recovery (1 wk after) if survive phase III or multisystem organ failure

**Evaluation**

- APAP level 4 h after ingestion, serum/urine tox for coingestants, baseline LFTs & coags, BMP for calculation of anion gap, preop labs if potential for need for transplant, ECG

**Treatment**

- N-Acetylcysteine (NAC) is glutathione substitute used as antidote
- Cardiac monitor, 2 large-bore IVs, ± NGT for anticipated NAC tx (PO NAC noxious), antiemetics
- Begin NAC if (acute ingestion, APAP level >140 μg/mL), (chronic ingestion of >200 mg/kg, 150 mg/kg, 100 mg/kg over 1, 2, 3 d, or ↑ LFTs, detectable serum APAP, or high risk)
- NAC 140 mg/kg per NGT × 1, then 70 mg/kg PNGT or 150 mg/kg IV × 1, then 50 mg/kg IV q4h × 5 doses, prolong therapy past initial 20 h if persistent serum APAP detected or ↑ LFTs until improvement in LFTs
- Activated charcoal (recent ingestion), HD (APAP >1,000 mg/L +
coma/hypotension)

**Disposition**
- Admission to hospital vs. ICU based on clinical picture; transfer to transplant facility
- Consider Psych eval

**Pearls**
- Maximal safe APAP dose 15 mg/kg (up to 1000 mg) QID, max daily dose 3–4 g/24 h
- APAP metabolism produces NAPQI (toxic metabolite) → direct hepatocyte damage
- When coupled to glutathione, NAPQI made inert & is excreted in urine; APAP tox results from overwhelmed/depleted glutathione stores
- Many unintentional APAP OD from confusion b/w pediatric vs. infant APAP preparations
- Infant: 80 mg/0.8 mL = 100 mg/mL; children: 160 mg/5 mL (5 mL = 1 tsp) = 32 mg/mL
Figure 16. Relation between plasma APAP level and hepatotoxicity correlated with time after ingestion. Reprinted with permission from Helms RA, Quan DJ. *Textbook of Therapeutics: Drug and Disease Management*. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.

**SALICYLATE POISONING (ASA)**

**History**
- Witnessed or reported ingestion of ASA or ASA-containing meds
- Often coingestions w/ other substances, occasionally inadvertent (elderly)
- Suspect ASA poisoning in any pt who reports tinnitus
Findings
- 1st 8–12 h: Fever, hyperventilation (respiratory alkalosis + metabolic acidosis), hyperpnea, tachycardia, hypotension, diaphoresis, dysrhythmias, N/V, epigastric pain, confusion
- By 24 h: Coma, cerebral edema, szs, noncardiogenic pulmonary edema, DIC

Evaluation
- Serum tox screen for ASA level & coingestants, BMP for anion gap, CBC, baseline coags, ABG, CXR, ECG
- Check ASA level q4h to ensure that levels are not rising due to bezoar formation or enteric-coated formulation delayed metabolism
- Bedside ferric chloride test (sens but not spec), serum quantitative assay preferred
- Add 2–4 drops 10% ferric chloride to 2 mL urine: Bluish purple color indicates + ASA; acetone & phenylpyruvic acid cause false + (pt w/ DM, ketoacidosis alcoholics)
- Ferric chloride testing will be + if as little as 2 ASA tablets ingested 24 h prior to test; takes 2 h from time of ingestion for ASA to be renally cleared

Treatment
- Airway protection if pt tires, hyperventilate & maintain respiratory alkalosis w/ vent
- Cardiac monitor, 2 large-bore IVs, Foley to monitor UOP & pH, dextrose for hypoglycemia
- Alkalinize urine to enhance ASA excretion
- 3 amps NaHCO₃ to 1 L D₅W or 2 amps NaHCO₃ to 1 L ½ NS, bolus over 30 min
- Continue NaHCO₃ IV fluid to maintain serum pH >7.45, <7.55, UOP 1.5 mL/kg/h
- Add 20–40 mEq K⁺ to replete K⁺ exchanged into cells for H⁺ ions; hypokalemia prevents effective alkaline diuresis
- Activated charcoal (recent acute ingestion), consider whole bowel irrigation
- Arrange for HD for symptomatic pt, if chronic ASA poisoning w/ ASA >60 mg/dL or acute ASA poisoning w/ ASA >90 mg/dL w/ severe acidosis
Disposition

- Admission to floor vs. ICU (if symptomatic), observe for at least 6 h (asymptomatic, nonenteric-coated, smaller ingestions), screen for SI/psych eval if indicated

Pearls

- ASA uncouples oxidative phosphorylation, causes a 1° metabolic acidosis & 1° (centrally mediated) respiratory alkalosis
- Methyl salicylate (found in BenGay, Icy Hot muscle balm, oil of wintergreen food flavoring) produces ASA tox in very small amounts (1 tsp of oil of wintergreen contains 7 g of ASA)
- Done nomogram created for ASA in the same way as the APAP-tox nomogram; considered to be inaccurate & of no clinical value due to the wide metabolic swings that occur w/ salicylate tox; is no longer used

<table>
<thead>
<tr>
<th>Amount Ingested</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150 mg/kg</td>
<td>None → mild tox</td>
</tr>
<tr>
<td>150–300 mg/kg</td>
<td>Mild → mod tox</td>
</tr>
<tr>
<td>301–500 mg/kg</td>
<td>Serious tox</td>
</tr>
<tr>
<td>&gt;500 mg/kg</td>
<td>Potentially lethal tox</td>
</tr>
</tbody>
</table>

CARDIAC MEDICATION OVERDOSE

α-Blocker (βB) Overdose

History

- Witnessed or reported overingestion of βB
- Children who have been at homes of older relatives taking prescribed medications

Findings

- Symptomatic bradycardia, hypotension, AMS, weakness, bronchospasm
- Lipid-soluble βB (propranolol) – sz; sotalol – ↑ QTc, torsades de pointes
May have hypoglycemia, N/V, hyperkalemia

Evaluation
- ECG shows bradycardia, AV or intraventricular block, asystole
- Check cardiac enzymes, BMP; drug levels not available

Treatment
- Continuous Tele, 2 large-bore IVs, place transcutaneous pacer pads on pt
- Place a cordis in the R IJ or L subclavian vein if transvenous pacing indicated
- For symptomatic or refractory βB OD, administer:
  - Atropine 0.5–1 mg IV (ACLS protocol) for severe bradycardia &/or hypotension
  - Glucagon 5–10 mg IV bolus followed by infusion of 1–5 mg/h if hypotensive
  - Pressors if indicated (epinephrine), cardiac pacing prn
  - Sodium bicarbonate 1–2 mEq/kg for wide-complex conduction defects
  - Consider hyperinsulinemia–euglycemia therapy &/or IV lipid emulsion (benefit in animals & case reports)
- No role for activated charcoal or whole bowel irrigation unless massive recent OD
- HD only useful for βB w/ low volume of distribution (acebutolol, atenolol, nadolol, timolol, sotalol) if unresponsive to medical intervention, or if pressors/glucagon necessary to maintain BP

Disposition
- Admission to floor vs. ICU (if symptomatic)
- Clinically significant βB OD develop sx&s w/i 6 h; if remain asymptomatic, can be D/C unless ingested sustained release formulation (24 h observation)

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**Calcium Channel Blocker (CCB) Overdose**

History
- Witnessed or reported overingestion of CCB
- Children who have been at homes of older relatives taking prescribed medications

Findings
Symptomatic bradycardia, hypotension, AMS, N/V, weakness
- Transient hyperglycemia; sz rare

**Evaluation**
- ECG shows bradycardia, ventricular escape rhythm, 2nd- or 3rd-degree AV block; usually nl QRS complex (vs. βB OD)
- Check cardiac enzymes, BMP; drug levels not available

**Treatment**
- Continuous Tele, 2 large-bore IVs, place transcutaneous pacer pads on pt
- Place a cordis in the R IJ or L subclavian vein if transvenous pacing indicated
- Continue supportive therapy including volume resuscitation & pressors for hypotension & depressed inotropy
- For either symptomatic βB or CCB OD, administer:
  - Atropine 0.5–1 mg IV (ACLS protocol)
  - Glucagon 5–10 mg IV bolus followed by infusion of 1–5 mg/h if hypotensive
  - Calcium gluconate 3 g slow IV push or calcium chloride 1 g IV q5–10min prn
  - Can reverse depression of cardiac contractility; no effect on sinus node depression or peripheral vasodilation; variable effect on AV node conduction
  - Pressors if indicated (dopamine, norepinephrine, amrinone)
  - For CCB OD, hyperinsulinemia–euglycemia therapy can provide fuel for enhanced myocardial contractility
    - If glucose <200 mg/dL, give dextrose 0.25 g/kg D_25 up to 1 amp D_50
    - If K+ <2.5 mEq/dL, administer 40 mEq IV; monitor & replete K+ prn
    - Administer regular insulin 0.5–1 U/kg IV bolus, followed by infusion of 0.5–1 U/kg/h
  - Start D_10 ½ NS at 80% maintenance rate
  - Recheck glucose q20min × 1 h, then qh; titrate insulin infusion to maintain glucose b/w 100 & 200
  - Consider IV lipid emulsion (promising in animal studies & case reports), glucagon
  - No role for activated charcoal or whole bowel irrigation unless massive recent OD of extended-release formulation; then use multidose charcoal
- HD not useful for CCB OD due to extensive protein binding

**Disposition**
- Admission to floor vs. ICU (if symptomatic)
- CCB should be monitored for 6 h or 24 h for sustained release formulations

---

**Di**goxin **Overdose**

**History**
- Usually in pts on chronic Dig, occasional acute intentional OD occurs
- Weakness, fatigue, palpitations, syncope, AMS, N/V, diarrhea, HA, paresthesias
- Yellow-green vision or other vision disturbances pathognomonic in chronic OD (not always present)
- Recent worsening renal fxn, dehydration, electrolyte abn, recent addition of new med

**Findings**
- GI sxs (common), generalized neuro sxs, visual Δ w/ few objective findings
- Hemodynamic instability related to dysrhythmias or acute CHF

**Evaluation**
- ECG may show a number of cardiac dysrhythmias (see table)
- Dig level, cardiac enzymes, BMP (↑ K in acute OD, nl or ↓ K, ↓ Mg in chronic OD)

**Treatment**
- Continuous Tele, trend Dig & serum K levels w/ ECG & clinical picture
- Correct electrolyte abnl
- Acute overdose
  - ↑ K is bad prognostic sign; treat immediately w/ calcium, glucose/insulin & bicarb (the notion that calcium is contraindicated in Dig overdose is based on very weak evidence from animal models)
  - Magnesium, lidocaine, antiarrhythmic until Digibind available
  - Dig spec Ab (antidote) if level >6, K >5, high-deg AV block, ventricular arrhythmias, AMS, hemodynamic compromise
  - Each vial of Dig spec Ab binds 0.5 mg of Dig
  - # of Dig spec Ab = (serum Dig [ng/mL] × TBW [kg])/100
For unknown amount/level, empirically treat w/ 10 vials, repeat once prn for acute ingestion, 6 vials for chronic ingestion
Phenytoin & lidocaine safe to control tachydysrhythmias
Activated charcoal (if recent ingestion), dialysis ineffective due to large Vd
› Chronic tox
› Stop Dig
› Verify need for Dig spec Ab, check Cr, electrolytes

Disposition
› Admission to floor vs. ICU (if hemodynamic instability, refractory dysrhythmia)
› If asymptomatic, no cardiac dysrhythmias, nl K & dig level, can d/c after 6 h

Pearl
› Many drug interactions (BZD, βB, CCB, diuretics, succinylcholine, some abx)

<table>
<thead>
<tr>
<th>Dysrhythmias Suggestive of Dig Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVCs (most common); bigeminy or trigeminy</td>
</tr>
<tr>
<td>Slow AF w/ regularized ventricular rate (AV dissociation)</td>
</tr>
<tr>
<td>NPJT (rate 70–130)</td>
</tr>
<tr>
<td>AT w/ block</td>
</tr>
<tr>
<td>Bidirectional ventricular tachycardia</td>
</tr>
<tr>
<td>Asystole or ventricular fibrillation</td>
</tr>
</tbody>
</table>

CAUSTIC INGESTIONS

Background
› Cause tissue injury by acidic or alkaline chemical rxn
› pH <2 is considered strong acid, pH >12 considered strong base
› Severity of tissue injury determined by duration of contact, pH, concentration, type of substance (liquid vs. solid)

Approach
ACID/ALKALINE INGESTIONS

History
- Alkaline: Ingestion of ammonia, cleaning agents: drain, oven, swimming pool, dishwasher detergents, bleach, cement, hair relaxers
- Acid: Ingestion of battery liquid, toilet bowl cleaners, rust or metal cleaning products, drain cleaners, cement cleaning products

Findings
- Alkaline: Liquefactive necrosis – severe injury starts rapidly after ingestion, w/i min of contact, tissues that 1st contact alkali are most severely injured (oropharynx, hypopharynx, esophagus). Tissue edema occurs immediately, may persist for 48 h, progress → airway obstruction. Over 2–4 wk get scar tissue thickening → strictures (depends on depth of burn).
- Acid: Coagulation necrosis → desiccation → eschar formation; stomach most commonly affected, small bowel exposure possible. Eschar sloughs in 3–4 d, then granulation tissue development. Perforation after 3–4 d as eschar sloughs; gastric outlet obstruction if scar tissue contracts over 2–4 wk. Pyloric sphincter spasm may delay gastric emptying & ↑ contact time to 90 min.
- In hydrofluoric acid (HF) ingestion, ↓ Ca may lead to arrhythmias, sudden cardiac arrest
- Both may cause esophageal perforation

Evaluation
- pH of product & of saliva, CBC, BMP, ABG, baseline LFTs, UA, preop labs, tox screen; cardiac monitoring, ECG; x-rays, consider CT for extraluminal air
- Endoscopy if symptomatic, small child, AMS but not if e/o perforation or...
airway edema

**Treatment**
- Airway protection, large-bore IV access; surgical/GI consultation, antiemetics
- Gastric lavage controversial
- Activated charcoal not helpful due to poor adsorption
- Dilution w/ small amts of water/milk may be beneficial if done w/i 30 min after ingestion
- Abx if e/o perforation, pain control

**Disposition**
- Admit to ICU if symptomatic

---

**CELLULAR ASPHYXIATES**

**Etiology**
- By-product of nitroprusside, acrylonitrile (nail polish, plastics, some tattoo ink), cyanogenic glycosides (apricot pits, cassava), cyanide gas (house fires)
- Mechanism: Binds to cytochrome oxidase, blocks aerobic utilization of $O_2$, leading to cellular asphyxia

**History**
- Difficulty breathing, confusion, HA, n/v, AMS, syncope, sz, cardiovascular collapse
- Sxs develop immediately after inhalational exposure, delayed sxs after exposure to nitroprusside, cyanide salts, acrylonitrile, cyanogenic glycosides

**Physical Exam**
- $O_2$ saturation often nl; dyspnea/tachypnea, confusion, tachycardia; agonal respirations & cardiovascular collapse a/w severe poisoning
- “Bitter almond” smell (unreliable), bright red venous blood due to high venous $O_2$ content

**Evaluation**
- **Labs:** BMP, ↑↑ lactate, ABG (metabolic acidosis), VBG (assess
venous/arterial $O_2$ gradient), cyanide level, carboxyhemoglobin (if smoke inhalation)

**Treatment**
- Supportive: Maintain airway, $O_2$ therapy, IV fluids
- Activated charcoal (presenting <2 h)
- Cyanide antidote:
  - Cyanocobalamin adult: 5 g (child: 70 mg/kg) IV over 15 min
  - Cyanide kit (amyl nitrite, sodium nitrite, sodium thiosulfate) – causes methemoglobinemia

**Disposition**
- Admit: All pts; consider ICU for pts w/ szs, coma, acidosis, hypotension

---

**Carbon Monoxide Poisoning and Methemoglobinemia**

**Etiology**

**Carbon Monoxide**
- Smoke inhalation, methylene chloride exposure
- Mechanism: Reduces $O_2$ carrying capacity, shifts $O_2$ dissociation curve to L

**Methemoglobinemia**
- Nitrites, dapsone, sulfa drugs, lidocaine/benzocaine, antimalarials, water contamination
- Mechanism: Disequilibrium of methemoglobin to hemoglobin; overwhelmed methemoglobin reductase

**History**

**Carbon Monoxide**
- Mild: Mild HA, DOE; mod: HA, N/V, dizziness, poor concentration; severe: CP, syncope, coma, LOC & persistent AMS

**Methemoglobinemia**
- SOB, HA, light-headedness, fatigue, nausea, tachycardia, CP, syncope

**Physical Exam**

**Carbon Monoxide**
- Lethargy, szs, tachycardia, tachypnea, rales, confusion, red skin, or cyanosis
Methemoglobinemia

- “Chocolate” cyanosis, tachycardia; coma, sz, D a/w severe exposure

Evaluation

- ABG & Pulse ox: May be falsely reassuring

Labs

- CO
  - CO oximeter, CO level (mild: 10–20%, mod: 20–40%, severe: >40%), urine hCG; mod/severe: ABG (metabolic acidosis), BMP, CBC, cardiac enzymes, UA, CPK, lactate, consider cyanide level
  - ECG: Arrhythmias, signs of MI

- Methemoglobinemia
  - CO oximeter, methemoglobin level; severe exposure: ABG, hemolysis labs (LDH, peripheral smear, haptoglobin, reticulocyte count), type & crossmatch
  - Bedside test: Drop of blood on white filter paper will turn chocolate brown (compared to regular venous blood)

Treatment

Carbon Monoxide

- O₂: 100% NRB until sxs improved
- Hyperbaric O₂: Sz, respiratory failure, LOC, CO level >25% (if pregnant, >15%), infants, severe acidosis, neuro deficits, CV dysfxn, exposure >24 h, age >36 yr

Methemoglobinemia (symptomatic exposures, level >20%)

- Methylene blue (reducing agent): 1–2 mg/kg of 1% solution IV qh × 2 doses
- Exchange transfusion/hyperbaric O₂: Severe sxs not responsive to methylene blue or if methylene blue is contraindicated (eg, G6PD deficiency)

Disposition

- Admit if CO level >25%, methemoglobin level >20%, dapsone tox, LOC, pts w/ underlying cardiac/neurologic/respiratory dz

Pearls

- Do NOT use methylene blue in pts w/ G6PD deficiency (hemolytic anemia)
Large amounts of methylene blue may paradoxically elevate methemoglobin levels

**Hypoglycemics**

**History**
- Oral ingestion of sulfonylureas, meglitinides (eg, repaglinide), or SC/IV insulin (oral insulin is not toxic)
- Agitation, coma, convulsions, confusion, blurry vision, n/v, rapid heartbeat, sweating, tingling of tongue & lips, tremor, dizziness, poor feeding; children may show sx with 5 min of ingestion
- RF: Extremes of age, polypharmacy, renal or hepatic dz, suicide attempt

**Physical Exam**
- AMS, generalized weakness, diaphoresis, tachycardia, tachypnea, transient neurologic deficit, pallor, sz, cyanosis, coma, hypothermia

**Evaluation**
- **Labs:** FSG q1h, BMP, urine hCG; tox screen (if intentional overdose or ingestion unknown), C-peptide (present w/ endogenous insulin secretion)

**Treatment**
- Supportive: ABCs, activated charcoal if recent ingestion
- Dextrose:
  - Oral: Glucose paste, juice
  - IV: 0.5–1 g/kg IV D$_{50}$W (adults), D$_{25}$W (children), D$_{10}$W (neonates) × 1 dose; persistent hypoglycemia: 0.5 g/kg/h D$_{10}$W (titrate to glucose >100)
  - Glucagon: 1 mg/dose IV/IM/SC (if <20 kg, 0.5 mg/dose)
  - Octreotide for sulfonylurea or meglitinide overdose

**Disposition**
- Home: Pts w/ unintentional isolated insulin overdose may be treated & released after effect of insulin wears off depending on rapid vs. long-acting
- Admit: Pts w/ sulfonylurea overdose must be monitored for at least 8 h
## OTHER INGESTIONS

### Etiology, Hx, & Sxs of Other Ingestions

<table>
<thead>
<tr>
<th>Overdose</th>
<th>Etiology</th>
<th>Sxs of Acute Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Insecticides</strong></td>
<td><strong>Organophosphate</strong> nerve gas (Sarin, Tabun)</td>
<td>SLUDGE: Salivation, lacrimation, urinary incontinence, defecation, GI distress, emesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other: Muscle weakness/paralysis, diaphoresis, bronchospasm, miosis, bronchorrhea, tachycardia, HTN, sz, respiratory depression, garlic-like odor; AVOID succinylcholine</td>
</tr>
<tr>
<td><strong>Carbamates</strong> (used to treat myasthenia gravis)</td>
<td></td>
<td>Similar to organophosphates but shorter acting &amp; may not have neuro sxs</td>
</tr>
<tr>
<td><strong>Chlorinated Hydrocarbons</strong> (DDT, chlordane, lindane)</td>
<td></td>
<td>Tremors, paresthesias, szs, AMS, muscle twitching, hyperthermia, arrhythmias, rhabdomyolysis, chemical pneumonitis</td>
</tr>
<tr>
<td><strong>Iron</strong></td>
<td>Any iron supplement, tox &gt;20 mg/kg</td>
<td>&lt;12 h: GI (emesis/diarrhea/abd pain; Severe: Bloody emesis/diarrhea, large fluid losses)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6–24 h: Latent phase w/o sxs</td>
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<tr>
<td></td>
<td></td>
<td>24–72 h: Hepatorenal failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2–6 wk: Chronic GI strictures</td>
</tr>
<tr>
<td><strong>Phenytoin/fosphenytoin</strong></td>
<td></td>
<td>Lethargy, dysarthria, ataxia, dizziness, confusion, horizontal nystagmus, N/V</td>
</tr>
<tr>
<td><strong>Hydrocarbons/volatiles</strong></td>
<td>Baby oil, mineral oil, furniture polish, paint thinner, petroleum jelly, solvents, gasoline, lamp oil kerosene, lighter fluid</td>
<td>Hydrocarbon odor, glue sniffer’s rash, chemical pneumonitis, aspiration, confusion, depression, szs, dysrhythmias, N/V, liver failure, burns, cerebellar dysfxn</td>
</tr>
<tr>
<td><strong>Herbicides</strong></td>
<td>Paraquat, diquat, Roundup (glyphosate), Glufosinate, Atrazine, Mecoprop, Acetochlor, Dichamba, Pentachlorophenols, Chlorophenoxy, Nitrophenolic, Metolachlor</td>
<td>Dermatologic irritant, mediastinitis, peritonitis, N/V/D, liver failure, CV shock, coma, sz, muscle weakness, renal failure/tubular necrosis/myoglobinuria, rhabdomyolysis, pulmonary edema, pulmonary fibrosis (paraquat), ICH (diquat)</td>
</tr>
<tr>
<td>Overdose</td>
<td>Etiology</td>
<td>Sxs of Acute Toxicity</td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Heavy metals</strong></td>
<td>Arsenic, bismuth, cadmium, chromium, cobalt, copper, lead, manganese, mercury, nickel, selenium, silver, thallium, zinc</td>
<td>Varies based on poisoning; in general N/V, GI distress, renal failure/ATN, pneumonitis, encephalopathy, abd pain; zinc smells like fish</td>
</tr>
</tbody>
</table>
| **Rodenticides** | Red squill, strychnine, yellow phosphorous, warfarin type/brodifacoum     | Red squill: Cardiac glycoside-like sxss  
Strychnine: Sz-like appearance w/ extensor posturing, rhabdomyolysis  
Yellow phosphorous: Garlic odor, oral burns, vomiting, phosphorescent smelling feces, GIB, electrolyte abnl, sz, arrhythmias, renal/hepatic failure  
Warfarin type/brodifacoum: Long-acting anticoagulation, bleeding risk |
| **Household products** | Acids (toilet bowel cleaners), bases (bleach, ammonia), detergents, all-purpose cleaners (glass cleaner, pine oil, turpentine), chlorine, cosmetics | Bases/acids: GI irritation  
Bases: Pneumonitis, pneumomediastinum  
Perfume/mouthwash: Depends on alcohol level  
Pine oil/turpentine: Hemorrhagic pulmonary edema  
Detergents: GI irritants/corrosives, pulmonary edema  
Glass cleaner: Ocular, o/w well tolerated |

**Eval & Tx of Other Ingestions**

<table>
<thead>
<tr>
<th>Overdose</th>
<th>Labs/Imaging</th>
<th>Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Organophosphates</strong></td>
<td>BMP, ECG, plasma cholinesterase level, lactate, CK, LFTs, CXR</td>
<td>Decontamination; atropine (2–5 mg) IV q5min (endpoint = dried secretions); 2-PAM 1–2 g IV over 30–60 min, 500–1000 mg/h (will not work on skeletal muscle); BZD (prn szs/agitation)</td>
</tr>
<tr>
<td><strong>Carbamates</strong></td>
<td>Same as organophosphates</td>
<td>Supportive, decontamination, atropine (dose same as organophosphates)</td>
</tr>
<tr>
<td><strong>Chlorinated hydrocarbons</strong></td>
<td>Electrolytes (metabolic acidosis, ATN), ECG (arrhythmias), CK</td>
<td>Supportive, decontamination, activated charcoal, cholestyramine (do not use in bowel obstruction),</td>
</tr>
<tr>
<td>Table Title</td>
<td>Individual Subheading</td>
<td>Special Instructions</td>
</tr>
<tr>
<td>-------------</td>
<td>------------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td><strong>Iron</strong></td>
<td>Fe level q4h; check BMP, LFTs, lactate, CBC, coags if symptomatic; KUB may show radiopaque tablets</td>
<td>Decontamination &amp; whole bowel irrigation; supportive/IVFs; deferoxamine 15 mg/kg/h (max 1 g/h) over 6 h (for severe sx, may induce hypotension)</td>
</tr>
<tr>
<td><strong>Phenytoin/fosphenytoin</strong></td>
<td>Check phenytoin or fosphenytoin level; calculate free phenytoin level, albumin level, ECG, BMP</td>
<td>Supportive, activated charcoal, treat hypotension w/ fluids/pressors</td>
</tr>
<tr>
<td><strong>Hydrocarbons &amp; other Volatiles</strong></td>
<td>BMP (renal tubular acidosis, hypokalemia), ECG, LFTs (elevated); CXR (infiltrate, bronchovascular markings)</td>
<td>Remove all exposed clothing; supportive care – if intubated, PEEP beneficial</td>
</tr>
<tr>
<td><strong>Overdose</strong></td>
<td>Labs/Imaging</td>
<td>Tx</td>
</tr>
<tr>
<td><strong>Herbicides</strong></td>
<td>BMP (tubular necrosis, hypernatremia), lipase, CK, urine myoglobin, ECG (dysrhythmias), LFTs, CXR</td>
<td>Irrigate all areas of exposure (skin, eyes, gastric lavage), IVFs, electrolyte replacement, BZD (sz, agitation); activated charcoal Paraquat/Diquat/Glufosinate: Hemoperfusion Chlorophenoxy: Alkaline diuresis via 1–2 amps bicarb + KCl (urine output: 4–6 cc/kg/h) Pentachlorophenols/nitrophenolic: Aggressive cooling, treat hyperkalemia/rhabdomyolysis</td>
</tr>
<tr>
<td><strong>Heavy metals</strong></td>
<td>Send off individual levels, BMP (electrolyte abnl, renal failure), CBC (HCT), CXR (pneumonitis)</td>
<td>Supportive; may require intubation BAL: Copper, arsenic, lead, mercury NAC: Chromium, cobalt D-Penicillamine: Copper EDTA: Cobalt, lead MDAC: Thallium Prussian Blue: Thallium Selenium: Silver Succimer: Lead, copper, arsenic</td>
</tr>
<tr>
<td><strong>Rodenticides</strong></td>
<td>CXR, BMP, LFTs, EKG, CXR, CK, Urine hCG, PT/PTT, may check individual levels</td>
<td>Decontaminate, activated charcoal, whole bowel irrigation, supportive, renal failure may require dialysis, exchange transfusion for severe hemolysis</td>
</tr>
<tr>
<td><strong>Household material</strong></td>
<td>BMP (hypernatremia w/ bleach), CXR (aspiration PNA)</td>
<td>Supportive: IVFs, intubation if necessary; copious irrigation of skin, eye</td>
</tr>
<tr>
<td>Ingestion: Water, milk to reduce irritation; Pine oil/turpentine: GI decontamination, endoscopy</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
AIRWAY MANAGEMENT

Approach
- Assess need for intubation

<table>
<thead>
<tr>
<th>Indications for Intubation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inability to oxygenate</td>
</tr>
<tr>
<td>Inability to ventilate</td>
</tr>
<tr>
<td>Impending clinical course</td>
</tr>
</tbody>
</table>

- Anticipated need for airway management in pts at risk for deterioration
- Assess difficulty of intubation early
- In pts w/ acute respiratory failure, BVM ventilation or noninvasive positive pressure ventilation (NiPPV) can be a bridge, but not a substitute, to intubation
- **Choose appropriate intubation algorithm**

<table>
<thead>
<tr>
<th>Airway Algorithms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Presentation</strong></td>
</tr>
<tr>
<td>Standard</td>
</tr>
<tr>
<td>Anticipated difficult airway</td>
</tr>
<tr>
<td>Failed airway: Can't intubate</td>
</tr>
<tr>
<td>Failed airway: Can't intubate, can't ventilate</td>
</tr>
<tr>
<td>Crash airway (near D)</td>
</tr>
</tbody>
</table>

- **Choose appropriate intubation tool**
  - VL: 1st choice, if available; higher 1st pass success rate vs. DL
  - DL: Most commonly used (Mac or Miller blade)
  - Awake sedated airway (when difficult laryngoscopy is anticipated):
    Inhalation ± topical application of local anesthetic, parenteral sedation,
evaluate airway, intubate via VL or fiber optic, paralyze/sedate when airway established → requires cooperative pt, noncrash airway

**Pearls**

- Have rescue devices at the ready: EGD, cricothyrotomy kit
- Consider glycopyrrolate 0.2 mg IV to w/ ketamine to minimize secretions
- Good BVM technique saves lives

---

**RAPID SEQUENCE INTUBATION**

**The “7 Ps”**

<table>
<thead>
<tr>
<th>Preparation</th>
<th>Preoxygenation</th>
<th>Pretreatment</th>
<th>Positioning</th>
<th>Paralysis w/ induction</th>
<th>Placement w/ proof</th>
<th>Postintubation management</th>
</tr>
</thead>
</table>

- Preparation
  - Monitor $O_2$ sat, BP, rhythm, ≥1 IV
  - BVM, suction, ET $CO_2$ detector, oral airway, Bougie
  - Intubation equipment (eg, laryngoscope): Blade, backup blade, check video monitor/light
  - ETT: 7.5–8 (male), 7–7.5 (female); check cuff, load stylet/10-cc syringe; pediatrics tube size: $= 4 + (age in y/4)$ → or use Broselow tape
  - RSI medications/doses
  - Assess for difficult BVM, difficult intubation, & difficult cricothyrotomy → prepare appropriately

---

**Assessing Difficult BVM → MOANS**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>M – mask seal</td>
<td>Beard, lower facial trauma</td>
</tr>
<tr>
<td>O – obesity, obstruction</td>
<td>Includes angioedema, Ludwig angina, trauma, etc.</td>
</tr>
<tr>
<td>A – age</td>
<td>Age &gt;55 yr</td>
</tr>
<tr>
<td>N – no teeth</td>
<td>Difficulty obtaining seal</td>
</tr>
<tr>
<td>S – stiff</td>
<td>Stiff lungs (asthma/COPD, PNA, ARDS, etc.)</td>
</tr>
</tbody>
</table>
**Assessing Difficult Airway → LEMON**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>L – look externally</td>
<td>Overall gestalt of difficulty</td>
</tr>
<tr>
<td>E – evaluate 3-3-2</td>
<td>Can fit 3 fingers into open mouth, 3 fingers b/w tip of chin &amp; chin/neck jxn, 2 fingers b/w chin/neck jxn &amp; thyroid notch</td>
</tr>
<tr>
<td>M – Mallampati class</td>
<td>From I (soft palate, uvula, pillars seen) to IV (only hard palate seen)</td>
</tr>
<tr>
<td>O – obesity/obstruction</td>
<td>Look for muffled voice, difficulty handling secretions, stridor, sense of dyspnea</td>
</tr>
<tr>
<td>N – neck mobility</td>
<td>Eg, C-spine immobilization, ankylosing spondylitis, RA</td>
</tr>
</tbody>
</table>

**Assessing Difficult Cricothyrotomy → SHORT**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>S – surgery</td>
<td>Eg, Halo device, recent thyroid surgery</td>
</tr>
<tr>
<td>H – hematomata</td>
<td>Anything distorting neck anatomy (includes infection, abscess)</td>
</tr>
<tr>
<td>O – obesity</td>
<td>Also consider short neck, SC emphysema</td>
</tr>
<tr>
<td>R – radiation</td>
<td>Distorts anatomy</td>
</tr>
<tr>
<td>T – tumor</td>
<td>Distorts anatomy, ↑ bleeding</td>
</tr>
</tbody>
</table>

- Preoxygenation: BVM (provides \(\sim 100\% \text{FiO}_2\)) × 3 min or 8 vital capacity breaths
- Consider passive/apneic oxygenation: Place NC on high flow throughout intubation, prolongs time to desaturation
- Pretreatment: Give 3 min prior to intubation—lidocaine 1.5 mg/kg IV (↓ ICP, in pts w/ ↑ ICP, ↓ bronchospasm in pts w/ reactive airway dz); fentanyl 3 μg/kg IV (↓ ICP in pts w/ ↑ ICP, ↓ HTN response in pts w/ cardiac ischemia, aortic dissection, head bleed)
- Paralysis w/ induction: Always induce prior to paralysis
- Induction: Etomidate (0.3 mg/kg IV), midazolam (0.3 mg/kg IV), ketamine (1–3 mg/kg IV), thiopental (3 mg/kg IV)
- Paralysis: Succinylcholine (1.5 mg/kg IV, if no CI), rocuronium (1–1.2 mg/kg IV)
- Succinylcholine CIs: Large burns, paralysis, crush injury (w/ in 3 d–6 mo), abd sepsis (>3 d), elevated ICP or intraocular pressure, hx of MH, neurologic d/o (muscular dystrophy, MS, Amyotrophic Lateral Sclerosis)
- Rocuronium has no CIs but longer half-life often leads to delayed sedation
- Positioning: ± Cricoid pressure (prevents gastric regurgitation but may worsen DL view) before/during intubation until tube placement confirmed
- Placement w/ proof: Insert ETT via direct visualization of vocal cords, inflate cuff
- Confirm placement: ET CO$_2$ detector, auscultate lungs (assess for R-side intubation)
- Secure ETT, release cricoid pressure
- Postintubation management: Oral gastric tube, CXR, sedation (benzos, propofol) ± paralytics (vecuronium 0.1 mg/kg IV), analgesia (fentanyl), initiate mechanical ventilation

## CRICOTHYROTOMY

### Purpose
- Failed airway (can’t intubate/can’t oxygenate or ventilate); severe facial trauma, trismus, upper airway obstruction

### Equipment
- Scalpel (11 blade), Trousseau dilator, tracheal hook, Bougie, tracheostomy tube (6.0–6.5 ET tube if none immediately available)

### Positioning
- Pt supine, hyperextend neck if no CI

### Procedure
- Sterile technique if time allows; see RSI for preparation & postintubation management
- **Open Technique:**
  - Hold larynx w/ nondominant hand
  - Make **vertical incision** w/ dominant hand from thyroid cartilage to cricoid membrane (2–3 cm), through skin & soft tissue
  - Palpate cricothyroid membrane through incision using nondominant index finger, **not** visualization
  - Make **horizontal incision** <1 cm through cricothyroid membrane
  - Place finger into stoma, then replace w/ tracheal hook-pointed
caudad, then rotate cephalad. Alternatively, place Bougie (instead of tracheal hook) deep into stoma then slide ETT over Bougie & into place.

- Place Trousseau dilator in stoma w/ handle **perpendicular** to neck & dilate **vertically**
- Rotate dilator **parallel** to neck, then place tracheostomy tube w/ obturator in place, thumb over the obturator or ET tube
- Remove obturator (if tracheostomy tube), inflate cuff, suture in place

**Complications**
- Bleeding, misplaced tube, vocal cord damage
- Contraindicated in children <10 y/o, consider needle cricothyrotomy in peds

**Pearl**
- The hardest part of performing cricothyrotomy is deciding to do it → therefore, always consider this procedure in your airway algorithm
**PRIMARY SURVEY**

**Definition**
- Initial survey of the trauma pt for rapid identification of life-threatening injuries

**Approach**
- Eval in ABCDE order: Airway, breathing/ventilation, circulation, disability, exposure/environmental control

| 1° Survey |
|-------------------|--------------------------------------------------|
| **Airway maintenance w/c-spine immobilization** | Talking → airway patent → frequent reassessment<br>Unable to talk → eval for FB/facial fractures/tracheal/laryngeal injury/other obstruction → if obstruction not reversible w/ chin lift jaw/thrust or GCS <8 → intubation w/c-spine immobilization<br>Severe facial/neck trauma be prepared for surgical airway (see Chapter 17 for further details of airway management) |
| **Breathing/ventilation** | Eval chest wall excursion/bilateral breath sounds/chest wall (flail chest, crepitance, open chest wound, tracheal injury) → identify/repair injuries that impair ventilation; tension ptx (needle decompression/finger or tube thoracostomy), flail chest w/ pulm contusion, massive hemothorax (tube thoracostomy → >1500 cc blood out or >200 cc/h or unstable HD → OR), open ptx |
| **Circulation** | Hypotension/altered MS/confusion/mottled skin/thready pulse/diminished pulse = hemorrhage/hypovolemia until proven o/w → place multiple large-bore IVs/control external hemorrhage → resuscitate w/ 2 L NS → persistent hypotension; transfuse PRBC (males O⁺, females O⁻), consider massive transfusion protocol (1 PRBC:1 FFP:1 PLTs) if persistent transfusion requirements, consider permissive hypotension (SBP 70–100 mmHg) & restrictive use of fluids<br>FAST exam to evaluate for intra-abd hemorrhage → + FAST + persistent hypotension = OR |
| **Disability** | Rapid neurologic assessment; AVPU (Alert, responds to Verbal stimuli, responds to Painful stimuli, Unresponsive), GCS |
| **Exposure/environmental** | Remove clothes, avoid hypothermia (massive transfusions/environmental exposures) can lead to coagulopathies (warmed blankets/IVF) |
Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eye opening</th>
<th>BEST motor response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4—Open eyes spontaneously</td>
<td>6—Obeys commands</td>
</tr>
<tr>
<td>3—Open eyes to command</td>
<td>5—Localizes pain</td>
</tr>
<tr>
<td>2—Open eyes to pain</td>
<td>4—Withdraws to pain</td>
</tr>
<tr>
<td>1—No eye opening</td>
<td>3—Decorticate posturing (abnl flexion)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Verbal response</th>
</tr>
</thead>
<tbody>
<tr>
<td>5—Oriented, fluent speech</td>
</tr>
<tr>
<td>4—Confused conversation</td>
</tr>
<tr>
<td>3—Inappropriate words</td>
</tr>
<tr>
<td>2—Incomprehensible words</td>
</tr>
<tr>
<td>1—No speech</td>
</tr>
</tbody>
</table>

HEAD TRAUMA

Background
- Leading cause of traumatic D in pts <25
- 80% mild (GCS 14–15), 10% mod (GCS 9–13), 10% severe (GCS <9) injuries
- CPP = MAP – ICP, poor outcome if CPP <70 mmHg, CPP constant when MAP b/w 50 & 160
- 1° brain injury: Mechanical, irreversible damage caused by mechanical cell damage
- 2° brain injury: Alteration in cerebral blood flow → cerebral ischemia,
membrane disruption, cerebral edema, free radical generation

**Approach**
- Careful hx: Associated sxs (photophobia, vomiting, visual Δ, ocular pain), focal neurologic sxs
- Assess for head or neck trauma, medications, substance abuse
- Check fingerstick blood sugar to r/o hypoglycemia as cause for AMS
- Warning signs for neuroimaging: severe HA, vomiting, worsening over days, aggravated by exertion or Valsalva, neck stiffness, AMS, abnl neuro exam, peri- or retro-orbital pain

**Skull Fractures**

**History**
- Direct blow to the head, pt c/o pain

**Findings**
- Skull depression
- Basilar skull fx: Periorbital ecchymosis (raccoon eyes), retroauricular hematoma (Battle sign), otorrhea & rhinorrhea (CSF leak), 7th nerve palsy, hemotympanum

**Evaluation**
- Noncontrast head CT. CBC, Chem, coags, type & screen, tox screen; plain films not indicated
- CTA to eval for vascular injury if basilar skull fx present

**Treatment & Disposition**
- Airway management; management guided by underlying brain injury
- Linear skull fx: If no other IC injury may be observed 4–6 h & D/C
- Depressed skull fx: Admit to NSGY, surgical elevation if depressed skull fx > thickness of skull, update tetanus, give ppx abx & consider anticonvulsants
- Basilar skull fx: Admit to NSGY

**Pearl**
- GCS more indicative of underlying brain injury or hemorrhage

**Scalp Laceration**

**History**
- Direct blow to the head, direct bleeding from scalp

**Findings**
- Often blood has clotted upon ED arrival; has potential for large blood loss
- Blood loss may not be evident in ED, eval for blood loss in field

**Evaluation**
- Noncontrast head CT if indicated. CBC, Chem, coags, type & screen, tox screen if significant blood loss
- Thoroughly evaluate & explore skull for depressions & large lacerations

**Treatment**
- Hemostasis & irrigation: Wounds often contaminated despite rich blood supply, direct venous drainage into the venous sinuses can cause significant CNS infections
- Staples can be used if galea not involved
- Interrupted or vertical mattress sutures w/ 3-0 nylon or Prolene
- Galea must be repaired w/ absorbable sutures if lacerated; continued bleeding → subgaleal hematoma that often becomes infected

**Disposition**
- If no other injuries, can d/c. Otherwise admission & observation.

**Pearl**
- Abx not indicated for properly managed head wound unless gross contamination

### Head Injury Classifications

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GCS</strong></td>
<td>14–15</td>
<td>9–13</td>
<td>&lt;9</td>
</tr>
<tr>
<td><strong>Hx</strong></td>
<td>Transient LOC, amnestic to event</td>
<td>LOC, amnestic to event</td>
<td>Pt unable to provide hx</td>
</tr>
<tr>
<td><strong>Sxs</strong></td>
<td>Mild HA, nausea</td>
<td>Confused or somnolent, often unable to follow commands</td>
<td>Obtunded, cannot follow simple commands</td>
</tr>
<tr>
<td><strong>Head CT</strong></td>
<td>Only if indicated (head CT rules); usually neg</td>
<td>All pts</td>
<td>All pts</td>
</tr>
<tr>
<td><strong>Eval</strong></td>
<td>Evaluate C-spine, no other testing needed</td>
<td>CBC, glucose, Chem, tox, coags, UA, hCG</td>
<td>CBC, glucose, Chem, tox, coags, UA, hCG</td>
</tr>
<tr>
<td><strong>Tx</strong></td>
<td>Observation w/ neuro checks, d/c w/ careful return instructions</td>
<td>24-h admission even if head CT neg, repeat CT if ↓ GCS, AMS</td>
<td>Intubation, NSGY eval, IVF, tight BP control (SBP &gt;90), treat ↑ ICP (mannitol, hypertonic)</td>
</tr>
</tbody>
</table>
Canadian Head CT Imaging Rule; Must Have Initial GCS 13–15

Indications for CT Scan
- GCS <15 at 2 h postinjury
- Suspected open or depressed skull fx
- Age >64
- Retrograde amnesia to event at >30 min
- Any sign of basilar skull fx
- 2 or more episodes of vomiting
- Dangerous mechanism

Postconcussive Syndrome

History
- Closed head injury, ± LOC (brief); HA, memory problems, dizziness, etc. may last 6 wk

Findings
- nl neurologic exam, wide spectrum of mild neuro complaints

Evaluation
- Noncontrast CT shows no bleed but clinically insignificant SAH may have occurred

Treatment
- Symptomatic HA control

Disposition
- D/c w/ careful head injury instructions
- Progressive return to full activity only after complete resolution of concussive sxs

Pearls
- Thought to be secondary to stretching of white matter fibers at time of injury
- 2nd head injury more dangerous than 1st

Intracerebral/Intraparenchymal Hemorrhage

History
Depends on size & location of bleed

**Findings**
- Pts commonly c/o HA, N/V

**Evaluation**
- Noncontrast head CT; CBC, Chem, coags, type & screen

**Treatment**
- Airway management
- Emergent neurosurgical eval although most pts are managed nonoperatively; ICP monitor if significant bleed present
- Mannitol for ↑ ICP, anticonvulsant medication to all pts
- Reverse coagulopathy emergently w/ appropriate agent (Vit K vs. FFP vs. PCC vs. factor conc) depending on underlying cause of coagulopathy

**Disposition**
- Follow

**Pearl**
- Frontal lobe hematoma may cause disinhibition & personality Δ

**Subarachnoid Hemorrhage (SAH)**

**History**
- Pt c/o “worst HA of life”; acute onset & rapid progression, meningismus, vomiting, photophobia; can often pinpoint exact moment of onset
- Spontaneous (ruptured cerebral aneurysm [~75%], AVM [~10%]) or traumatic

**Findings**
- HA, N/V, sz, syncope, acute distress
- Acute AMS is indicative of large bleed, usually requires emergent intervention

**Evaluation**
- Noncontrast CT scan of head, ancillary studies (CBC, BMP, coags, T&S)
- Head CT 95–99% sens for acute SAH (w/i 6–24 h); perform LP if CT neg
- If concern for ruptured cerebral aneurysm, should also obtain CT angiogram
- Large # RBC in CSF highly suggestive of SAH
- RBCs are hemolyzed in CSF, may not be present in large numbers after 12 h or may not be present at all after 2 wk
- Xanthochromia highly suggestive of bleed b/w 12 h & 2 wk (yellow discoloration due to RBC breakdown)
- Check fingerstick blood sugar to R/O hypoglycemia as cause for AMS

**Treatment**
- Airway management if comatose or not protecting airway, neurosurgical consultation
- ICP & BP monitoring if bleed is significant; a-line, elevate head of bed to 30°
- SPB b/w 90 & 140 mmHg, HR b/w 50 & 90 bpm, nicardipine or labetalol prn
- Mannitol for significant bleed w/ increased ICP
- Nimodipine to decrease vasospasm 60 mg PO q4h × 21 d
- Sz prophylaxis (phenytoin, Keppra)

**Disposition**
- To neurologic ICU

**Pearls**
- Outcome directly related to amount of intracranial blood
- 30–50% have “sentinel HA” days to weeks prior to SAH

### Clinical Findings of SAH

<table>
<thead>
<tr>
<th>Findings</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>HA</td>
<td>95–100%</td>
</tr>
<tr>
<td>Meningismus</td>
<td>Frequent</td>
</tr>
<tr>
<td>Transient LOC/syncope</td>
<td>50%</td>
</tr>
<tr>
<td>Retinal subhyaloid hemorrhage</td>
<td>6–30%</td>
</tr>
</tbody>
</table>

### Hunt–Hess Scale for SAH

<table>
<thead>
<tr>
<th>Grade</th>
<th>Findings</th>
<th>Percent Survival (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>Asymptomatic or mild HA</td>
<td>70</td>
</tr>
<tr>
<td>II.</td>
<td>Mod-to-severe HA, nuchal rigidity, no neuro deficits or other CN palsy</td>
<td>60</td>
</tr>
<tr>
<td>III.</td>
<td>Confusion, drowsiness, mild focal signs</td>
<td>50</td>
</tr>
</tbody>
</table>
Subdural Hematoma (SDH)

**History**
- Often caused by acceleration/deceleration tearing injury of bridging veins
- Can be acute (<48 h), subacute (2 d–3 wk) or chronic (>3 wk)

**Findings**
- Varied. Range from HA w/ nausea to comatose & flaccid

**Evaluation**
- Noncontrast head CT shows crescent-shaped mass. Check CBC, Chem, Coags, type & screen

**Treatment**
- Airway management, emergent neurosurgical eval
- If e/o ↑ ICP or midline shift, mannitol & anticonvulsant
- Reverse coagulopathy emergently w/ appropriate agent (Vit K, PCC, FFP, factor conc.)

**Disposition**
- Follow

**Pearls**
- More common than epidural hematoma
- Comatose & flaccid pts w/ SDH have an extremely poor prognosis, should discuss w/ family

Epidural Hematoma

**History**
- Brief LOC followed by “lucid interval,” then rapidly progressive deterioration
- Head injury usually in area of temporal bone, causes damage to middle meningeal artery

**Findings**
- Ipsilateral pupil deviation, occasionally contralateral hemiparesis, N/V, sz, hyperreflexia, + Babinski

**Evaluation**
Noncontrast CT often shows lenticular biconcave mass, possible fx of temporal bone
CBC, Chem, coag panel, type & screen

**Treatment**
- Airway management, emergent neurosurgical consultation
- Mannitol & anticonvulsant
- Reverse coagulopathy emergently w/ appropriate agent (Vit K, PCC, FFP, factor conc.)

**Disposition**
- Follow

**Pearl**
- Bleeding b/w the dura mater & skull

<table>
<thead>
<tr>
<th>Indications for Sz Prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed skull fractures</td>
</tr>
<tr>
<td>Paralyzed &amp; intubated, severe head injury</td>
</tr>
<tr>
<td>Sz at the time of injury or during ED presentation</td>
</tr>
<tr>
<td>Penetrating brain injury</td>
</tr>
<tr>
<td>GCS ≤8</td>
</tr>
<tr>
<td>Acute SDH, EDH, or ICH</td>
</tr>
<tr>
<td>Hx of szs prior to injury</td>
</tr>
</tbody>
</table>

**Diffuse Axonal Injury (DAI)**

**History**
- Result of tremendous shearing forces seen in high-speed MVCs

**Findings**
- Pts often present in coma; document best neuro response: May have prognostic value

**Evaluation**
- Noncontrast CT often nl, must r/o bleed
- CBC, Chem, coag panel, type & screen, tox; look for other etiology for coma
- MRI (nonemergent) will show Δ & can guide prognosis

**Treatment**
Airway management
Emergent neurosurgical consultation for ICP monitor to avoid 2° injury from edema
Mannitol & anticonvulsants

Disposition
Follow

Pearl
Prognosis determined by clinical course & difficult to predict

MAXILLOFACIAL INJURY

Definition
Injuries to the soft tissue or bones of the face (50% caused by MVCs)

Approach

Inspection
Deformities, enophthalmos (orbital blowout fracture), jaw malocclusion, dentition step-offs, nasal septal/auricular hematomas, rhinorrhea (CSF leak), trigeminal/facial nerve deficits, abnl EOM, diplopia, gross visual acuity

Palpation
Facial prominences for tenderness/bony defects/crepitance/false motion, FB

Radiology
Panoramic x-ray for mandibular/dental fractures, maxillofacial CT scan for most injuries, CTA in injuries at high risk for vascular trauma

Soft Tissue Injury
Definition
Injury to the soft tissue of the face

History
MVC/bites/assault

Evaluation
CT only if bony injury/FB suspected

Treatment
Irrigate/eval for FB/1° closure w/ in 24 h, abx (cefazolin, Ampicillin/Sulbactam, amoxicillin/clavulanate) for contaminated wounds (eg, bites), plastic surgery repair for nerve damage/extensive repair

Disposition

• Home

Septal/Auricular Hematomas

Definition

• Hematoma of nasal septum/ear

History

• Direct trauma to the nose (a/w nasal bone fractures)/ear (classically in wrestlers)

Physical Findings

• Swelling/purple discoloration

Treatment

• Septal: Apply topical anesthetic, incise/evacuate w/ elliptical incision, pack bilateral nares, abx (amoxicillin/clavulanate) (failure to drain → cartilage necrosis → saddle nose deformity)
• Auricular: Anesthetize area (lidocaine 1%) or auricular block, needle aspiration (chronic hematomas) or incise along skin folds, evacuate, apply compression dressing (failure to drain/compress → cauliflower ear/infection)

Disposition

• Home, f/u in 24 h

Nasal Fractures

Definition

• Fractures of the nasal bone

History

• Direct trauma to the nose

Physical Findings

• Swelling/deformity note: Patency of nares & appearance of septum

Evaluation

• CT only if significant deformity/persistent epistaxis/rhinorrhea

Disposition
Isolated nasal fractures → Most home w/ plastic/ENT f/u in 5–7 d for reduction, consider reduction in ED if displaced, (pediatric pts → 3 d, ↑ risk for growth dysplasia)

**Pearl**

- Septal hematoma requires immediate I&D to prevent necrosis

**Zygomatic Fracture**

**Definition**

- Fractures of the zygomatic arch or fracture at the zygomaticotemporal suture/zygomaticofrontal suture/infraorbital foramen (tripod fracture)

**History**

- Direct trauma to face

**Physical Findings**

- Shallow depression over temporal region, trismus, edema, diplopia/vertical dystopia/infraorbital nerve anesthesia (tripod fracture)

**Evaluation**

- Maxillofacial CT

**Treatment**

- ENT/OMFS/Plastics consult

**Disposition**

- Home, ENT/OMFS/plastics f/u for delayed ORIF, sinus precautions

**Mandibular Fractures**

**Definition**

- Fracture of the mandible (>50% multiple fracture sites)

**History**

- Direct trauma to mandible (assaults usually = body/angle fractures, MVC usually = symphysis/condylar fractures)

**Physical Findings**

- Malocclusion, trismus, associated dental & lingual injury

**Evaluation**

- Panorex (isolated mandibular fractures): Can miss condylar fracture, maxillofacial CT (preferred): Condylar fractures/additional facial trauma

**Treatment**
OMFS or plastic surgery consult: Temporary immobilization (wiring of jaw) or delayed ORIF, abx (PCN, clindamycin) if gingival bleeding

Disposition
› Home

Pearls
› Pts D/C w/ temporary wiring must be D/C w/ wire cutters
› Tongue blade test has high sens for mandibular fx

Maxillary Fractures

Definition
› Fracture of the maxilla, rare in isolation, a/w significant mechanism, greatest risk of airway compromise, traditionally classified by Le Fort system

History
› Significant mechanism trauma to the face (high-speed MVC)

Physical Findings
› Midface swelling/mobility, malocclusion of mandible, CSF rhinorrhea

Evaluation
› Maxillofacial CT
› CTA in Le Fort II & III should be strongly considered

Treatment
› Airway management (eval for difficult airway, Le Fort II/III highest risk), hemorrhage control (nasal packing/nasal Foley/elevation of head), abx (ceftriaxone) for CSF communication, ENT/OMFS consult

Disposition
› Admit

---

<table>
<thead>
<tr>
<th>Le Fort Classification</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Le Fort I</td>
<td>Involves only maxilla at level of nasal fossa; Free-floating jaw</td>
</tr>
<tr>
<td>Le Fort II</td>
<td>Involves maxilla, nasal bones, &amp; medial aspects of the orbits &amp; is described as pyramidal Dysfxn</td>
</tr>
<tr>
<td>Le Fort III</td>
<td>Involves the maxilla, zygoma, nasal bones, &amp; ethmoids. Extends through the maxillary sinuses &amp; infraorbital rims bilaterally across the bridge of the nose. Is described as craniofacial Dysfxn</td>
</tr>
</tbody>
</table>
EYE INJURY

Definition
- Injury to eye caused by trauma

Approach
- Assess visual acuity (use lid retractors if needed) & extraocular muscles (EOM), remove contact lenses

Orbital Fracture

Definition
- Fracture to the wall of the orbit (floor/medial wall most common)

History
- Blunt trauma to eye by object larger than the orbital rim

Physical Findings
- Periorbital swelling/crepitance, tenderness/irregularities to bony orbit, vertical diplopia/limited range of motion (ROM) w/ upward gaze (inferior rectus/inferior oblique entrapment), diplopia/limited ROM w/ lateral gaze (medius rectus entrapment), hypoesthesia of lower lid/cheek (infraorbital nerve entrapment), enophthalmos, ptosis

Evaluation
- Orbital CT (opacification of maxillary sinus = orbital floor fracture)

Treatment
- Abx (cover sinus flora), ophthalmology consult (rarely require surgery unless diplopia/entrapment) if any EOM entrapment or visual acuity Δ, “sinus precautions” (no nose blowing/sneezing, no sucking on straws/smoking)

Disposition
- Home

Pearls
- Orbital floor fractures are rare but a/w CNS trauma/infection
- Pts are at ↑ risk zygomatic tripod fractures/Le Fort II & III fractures

Globe Rupture

Definition
› Full-thickness defect in the cornea/sclera

**History**
› Blunt (most common at muscle insertion sites/corneoscleral junction) or penetrating (more common) trauma, decreased vision, pain

**Physical Findings**
› ↓ visual acuity, teardrop-shaped pupil, hyphema, + Seidel test (bright stream of aqueous humor after fluorescein) for corneal perforations, intraocular content extrusion, flattening of anterior chamber, oculocardiac reflex can cause bradycardia

**Evaluation**
› Orbital/head CT (for FB/intracranial injury), US—but must be careful to not apply pressure

**Treatment**
› Ophthalmology consult (for surgical repair), tetanus, abx (fluoroquinolones, vanc/gent), avoid pressure on eye/topical agents/Valsalva (antiemetics), protective shield

**Disposition**
› Admit

---

**Chemical Burns**

**Definition**
› Burns to sclera/conjunctiva/cornea/lid caused by alkali (oven cleaner, dish soap, detergents, cement, bleach) or acid (less severe)

**History**
› Chemical exposure, severe pain, FB sensation, photophobia

**Physical Findings**
› ↓ visual acuity, conjunctival injection, corneal edema, lens opacification, limbal blanching

**Evaluation**
› pH testing of effluent in fornixes

**Treatment**
› Topical anesthetics, irrigation (>2 L NS), use Morgan lens/manual retraction to keep eye open, check pH every 30 min until pH 7.3–7.7 & 10 min later, ↑ IOP treat like glaucoma, cycloplegics (cyclopentolate, tropicamide) if ciliary spasm, antibiotic ointment, ophthalmology consult
for corneal haziness/perforation/conjunctival blanching

Disposition
› Admit for increased IOP/intractable pain, minor burns: f/u in 24 h

Pearls
› Hydrofluoric acid exposure: Administer 1% calcium gluconate drops during irrigation
› If no pH paper available can use urine dipstick, for nl pH compare to unaffected eye

Retrobulbar Hematoma
Definition
› Bleeding in the space surrounding the globe

History
› Blunt trauma, recent eye surgery, pain, vomiting, ↓ visual acuity

Physical Findings
› Afferent papillary defect, restricted EOM, ↑ IOP, proptosis, periorbital ecchymosis, subconjunctival hemorrhage

Evaluation
› Orbital CT

Treatment
› Immediate ophthalmology consult, treat ↑ IOP (timolol, acetazolamide), decompress w/ lateral canthotomy

Disposition
› Admit

Retinal Detachment
Definition
› Detachment of the retina

History
› Floaters/flashing lights, “mosca volante”—solitary large floater, ↑ IOP, visual loss (macula involvement)

Physical Findings
› Visual field deficit (curtain being pulled down), dilated retinal exam: Retinal tears/detachment

Evaluation
β-scan u/s w/ undulating, hyperechoic membrane

**Treatment**
- NPO, bed rest, restrict EOM, immediate ophthalmology consult for surgical repair

**Disposition**
- Admit

**Hyphema**

**Definition**
- Accumulation of blood in the anterior chamber caused by rupture iris root vessel (trauma) or sickle cell/DM/anticoagulation

**History**
- Blunt or penetrating trauma to the globe, dull eye pain, photophobia

**Physical Findings**
- Microhyphemas: Visualized w/ slit lamp, larger hyphemas: Visualized w/ tangential pen light, total hyphema (high association w/ globe rupture): ↑ IOP

**Evaluation**
- INR if on Coumadin
- If any FH of hemoglobinopathy pt should be screened

**Treatment**
- Immediate ophthalmology consult for >10%/↑ IOP, treat ↑ IOP (timolol, acetazolamide), metal eye shield, cycloplegics (cyclopentolate, tropicamide) if ciliary spasm
- HOB >45% (upright allows blood to settle in anterior chamber/avoid retinal staining)
- Topical anesthesia if no globe rupture, PO/IV analgesia
- Topical steroids may help prevent rebleeding & synechiae

**Disposition**
- Admit for >50%, ↑ IOP, coagulopathy or sick cell
- Urgent ophthalmology f/u

**Pearls**
- Sickle cell: Avoid acetazolamide/pilocarpine/hyperosmotic, ↑ risk of rapid ↑ IOP → optic nerve injury
- Avoid ASA/NSAIDs 2/2 ↑ rebleed risk
- 10% rebleed (usually more severe) in 2–5 d
**Vitreous Hemorrhage**

**Definition**
- Blood in the vitreous humor

**History**
- Blunt trauma, floaters, blurry vision, vision loss, sickle cell/DM

**Physical Findings**
- Loss of light reflex, poorly visualized fundus

**Evaluation**
- β-scan u/s: For associated retinal detachment
- Consider noncontrast CTH if a/w trauma

**Treatment**
- Immediate ophthalmology consult, HOB >45%, bed rest

**Disposition**
- Admit if retinal tear/unknown cause

**Pearl**
- Avoid ASA/NSAIDs b/c ↑ risk rebleed

**Subconjunctival Hemorrhage**

**Definition**
- Hemorrhage b/w the conjunctiva & sclera caused by trauma, Valsalva (coughing/straining/vomiting), HTN, coagulopathy

**History**
- Painless red eye

**Physical Findings**
- Blood b/w the conjunctiva & sclera

**Treatment**
- BP control, avoid Valsalva, avoid ASA/NSAIDs, artificial tears for comfort

**Disposition**
- Home, ophthalmology f/u in 1 wk

**Pearls**
- Resolution in 2 wk
- Blood chemosis (large/circumferential) ↑ risk globe rupture
NECK TRAUMA

Definition
- Injuries soft tissue & structures of the neck

Approach
- Evaluate 3 main categories: vascular, pharyngoesophageal, laryngotracheal (do not place NGT if esophageal/laryngeal injury suspected)

Inspection
- Violation of platysma (↑ incidence of underlying structure injury, may indicate need for surgical exploration) (Trauma 1979;19:391), pulsatile/expanding hematomas

Penetrating Trauma Zones
- Anterior triangle: Bordered by anterior SCM, midline, mandible. Posterior: Posterior to SCM, anterior to trapezius, superior to clavicle, most significant structures are anterior
- Zone I: Below cricoid cartilage (highest mortality), Zone II: B/w cricoid & angle of mandible, Zone III: Above angle of mandible

<table>
<thead>
<tr>
<th>Recommended Imaging for Penetrating Neck Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injury</td>
</tr>
<tr>
<td>Vascular</td>
</tr>
<tr>
<td>Pharyngoesophageal</td>
</tr>
<tr>
<td>Laryngotracheal</td>
</tr>
</tbody>
</table>

Penetrating Neck Trauma

Definition
- Injury to the neck from GSW, stabbings, projectile objects (shrapnel/glass)

Physical Findings
- Laryngotracheal injuries may have stridor, respiratory distress, hemoptysis, SQ air, dysphonia
Esophageal injuries may have dysphagia, hematemesis, SQ air
Vascular injuries may have neuro deficits, expanding/pulsatile hematoma/bleeding, bruit/thrill, hypotension

Evaluation
- CXR/(ptx/htx), lateral neck x-ray in trauma bay, CT, CTA
- Trauma labs: CBC, BMP, type & cross, PTT/PT, ABG

Treatment
- Airway management (may be difficult airway), surgical consultation if platysma violation, abx (if ↑ risk contamination from aerodigestive perforation)
- Treat as trauma resuscitation (ABCs, transfusion, etc.)

Disposition
- Admit if surgical intervention/observation needed

Pearl
- Arrest due to penetrating neck trauma is indication for ED thoracotomy

Strangulation

Definition
- Neck trauma due to strangulation (3500 D/y)

History
- Strangulation, voice Δ, attempt to obtain “height of drop” from EMS

Physical Findings
- Dysphonia/dyspnea (indicators serious injury), petechial hemorrhages (Tardieu spots), ligature/finger marks, neuro deficits/coma

Treatment
- Airway management (may be difficult airway), surgical consultation (if needed), consider CTA, abx (if ↑ risk contamination from aerodigestive perforation)

Disposition
- Admit if needed

Pearls
- ↑ incidence of ARDS & long-term neuropsychiatric sequelae (selective vulnerability of hippocampus to anoxic injury)
- Self-inflicted hanging rarely a/w C-spine injury, see Hangman fracture (Chapter 18)
CERVICAL SPINE TRAUMA

Definition
- Injury to the bony/ligamentous structure of the cervical spine (C2 24%, C6 20%, C7 19%)

Approach
- Maintain C-spine immobilization until cleared clinically w/o imaging (see table) or radiographically

Palpation
- Midline cervical tenderness, step-offs, neurologic deficits

Radiology
- Plain c-spine x-rays: 52% sens (limited use), C-spine CT: 98% sens → persistent midline tenderness/obtunded → Flex/ex films: 94% sens for ligamentous injury if adequate ROM (30° flexion/extension), MRI: 98% sens for ligamentous injury (J Trauma 58(5):902; 53(3):426)

<table>
<thead>
<tr>
<th>Cervical Spine Clearance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NEXUS Low-risk Criteria</strong></td>
<td><strong>Canadian Cervical Spine Rule</strong></td>
</tr>
<tr>
<td>No posterior midline tenderness</td>
<td>Age ≥ 16</td>
</tr>
<tr>
<td>No focal neurologic deficits</td>
<td>GCS 15</td>
</tr>
<tr>
<td>nl alertness</td>
<td>nl VS (RR 10–14, SBP &gt; 90 mmHg)</td>
</tr>
<tr>
<td>No intoxication</td>
<td>Injury w/i 48 h</td>
</tr>
<tr>
<td>No painful distracting injury (long bone fracture, visceral injury, large laceration, degloving, burns, injury causing functional impairment)</td>
<td>Blunt trauma</td>
</tr>
<tr>
<td></td>
<td>No paralysis/paresthesia</td>
</tr>
<tr>
<td></td>
<td>No known vertebral dz</td>
</tr>
<tr>
<td></td>
<td>Not evaluated previously for same injury</td>
</tr>
<tr>
<td></td>
<td>Not pregnant</td>
</tr>
<tr>
<td></td>
<td>Not high risk (&lt;65 y/o, dangerous mechanism: MVC rollover/ejection/&gt;62 mph, fall from ≥3 ft, bicycle accident)</td>
</tr>
<tr>
<td>Presence of ≥1 low risk finding (simple rear-end MVC, sitting position in ED, ambulatory after trauma, delayed onset neck pain, no midline tenderness)</td>
<td>Presence of ≥1 low risk finding (simple rear-end MVC, sitting position in ED, ambulatory after trauma, delayed onset neck pain, no midline tenderness)</td>
</tr>
<tr>
<td>99.6% sens, 12.9% spec for significant C-spine injury</td>
<td>99.4% sens, 45.1 spec for significant C-spine injury</td>
</tr>
</tbody>
</table>

(NEJM 2000;342:94; 2003;349:2510)
C1 Burst Fracture (Jefferson Fracture)
Definition
- Unstable burst fracture of atlas (C1) causing widening of lateral masses (33% a/w C2 fracture)

History
- Axial load

Physical Findings
- C1 tenderness, neurologic deficit rare (wide canal at C1)

Evaluation
- CT/CTA, MRI for ligamentous injury

Treatment
- C-spine immobilization, spine consult for operative management

Disposition
- Admit

C2 Hangman Fracture
Definition
- Unstable fracture of bilateral C2 pedicles (↑ risk of C2 anterior subluxation/C2–C3 disk rupture → high mortality)

History
- Hyperextension
- Named due to judicial hangings in which knot is in front of pt & “height of drop” is at least as long as victim

Physical Findings
- C2 tenderness, high-impact trauma, neurologic deficits

Evaluation
- CT/CTA, MRI for ligamentous injury

Treatment
- C-spine immobilization, spine consult for operative management

Disposition
- Admit

Odontoid Fracture (C2 Dens)
Definition
- Fracture through the dens w/ variable stability (see table)
History
  ‣ Flexion injury

Physical Findings
  ‣ C2 tenderness

Evaluation
  ‣ CT scan, MRI for ligamentous injury

Treatment
  ‣ C-spine immobilization, spine consult

Disposition
  ‣ Likely admit

<table>
<thead>
<tr>
<th>Dens Fracture Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classification</td>
</tr>
<tr>
<td>----------------</td>
</tr>
</tbody>
</table>
| Type I | Avulsion fracture through upper part of odontoid process  
Stable & does not require surgical intervention |
| Type II | Fracture at the junction of the odontoid process w/ the vertebral body  
Potentially unstable fracture  
Nondisplaced: Halo often used to treat  
Displaced/angulated: Surgery often performed |
| Type III | Fracture at base of odontoid that extends down into body of atlas  
Immobilize w/ halo, does not usually require surgical intervention |

Tear Drop Fracture

Definition
  ‣ Unstable avulsion of cervical vertebral body at insertion of anterior ligament in extension injury (C2 common) or posterior in flexion injury (C5–C6)

History
  ‣ Flexion (MVC, diving in pool) or extension (elderly fall on chin)

Physical Findings
  ‣ C-spine tenderness, anterior cord syndrome (flexion), central cord syndrome (extension)

Evaluation
  ‣ CT/CTA, MRI for ligamentous injury

Treatment
C-spine immobilization, spine consult

**Disposition**
- Admit

**Clay Shoveler Fracture**

**Definition**
- Stable avulsion fracture of spinous process (most common in low C-spine, >C6)

**History**
- Forceful flexion (as when clay sticks to a shovel when trying to throw it)

**Physical Findings**
- C-spine tenderness, no neurologic deficits

**Evaluation**
- CT scan

**Treatment**
- C-spine immobilization, spine consult

**Disposition**
- D/c

**Subluxation/Ligamentous Injury**

**Definition**
- Unstable rupture of ligaments w/o bony injury, anterior slipping of vertebrae one over the other

**History**
- Flexion

**Physical Findings**
- C-spine tenderness, no neurologic deficits

**Evaluation**
- CT/CTA scan, MRI

**Treatment**
- C-spine immobilization, spine consult

**Disposition**
- May require admission
Definition
- Injury to the bony/ligamentous structure TLS spine

Approach
- Maintain logroll precautions
- Palpation: Spinal tenderness, step-offs, neurologic deficits

Anterior Wedge/Compression Fracture
Definition
- Stable compression fracture of the vertebral body (wedge → only anterosuperior vertebral body endplate). May be unstable if >50% height loss of vertebral body

History
- Flexion

Physical Findings
- Focal tenderness, no neurologic deficits

Evaluation
- CT scan

Treatment
- Spine consult

Disposition
- D/c if pain controlled

Burst Fracture
Definition
- Stable compression fracture of anterior & posterior vertebral body (may be complicated by retropulsed bony fragments → cord injury)

History
- Axial load/vertical compression

Physical Findings
- Focal tenderness, ± neurologic deficit
Evaluation
- CT scan

Treatment
- Spine consult, bracing/orthosis

Disposition
- Likely admit

**Chance Fracture**

**Definition**
- Often stable fracture through the vertebra, can also include body/pedicles/laminae

**History**
- Back pain after head-on MVC when wearing only a lap belt from flexion injury

**Physical Findings**
- Focal tenderness, rare neurologic deficit

**Evaluation**
- CT scan

**Treatment**
- Spine consult, orthosis

**Disposition**
- Admit

**Sacral Fracture**

**Definition**
- Fractures of the sacrum (may be a/w pelvic fractures in above S4)

**History**
- Buttock/perirectal/posterior thigh pain after direct trauma to sacrum (fall or force from behind)

**Physical Findings**
- Focal tenderness, neurologic deficits (above S4), careful eval for cauda equina

**Evaluation**
- CT scan

**Treatment**
Anterior Cord Syndrome
Definition
- Injury to the anterior cord from blunt or ischemic injury
History
- Flexion/axial load (major trauma), minor trauma (arthritis/spinal stenosis/OA/spinal cord pathology)
Physical Findings
- Bilateral loss of motor/pain/temperature sensation, dorsal column intact (proprioception/vibratory sense) *(See Sensory & Motor deficit tables)*
Evaluation
- MRI
Treatment
- Spine consult
Disposition
- Admit

Central Cord Syndrome
Definition
- Trauma to central cord → injury of corticospinal motor tracts of UE > tracts of LE (buckling of ligamentum flavum)
History
- Hyperextension of neck, hx of elderly, arthritis, OA, spinal stenosis
Physical Findings
- Loss of motor fxn in UE >LE, variable sensory loss *(See Sensory & Motor deficit tables)*, loss of pain & temperature if nontraumatic
Evaluation
- MRI
Treatment
- Spine consult
Disposition
Admit

Brown–Sequard Syndrome (Lateral Cord Syndrome)

Definition
› Hemicord transection from penetrating trauma

History
› Penetrating trauma

Physical Findings
› Ipsilateral motor/proprionception/vibration loss, contralateral pain/temperature sensation loss, deficits occur 2 levels below lesion

Evaluation
› MRI

Treatment
› Spine consult

Disposition
› Admit

<table>
<thead>
<tr>
<th>Deficit by Level of Spinal Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sensory-deficit Landmarks</strong></td>
</tr>
<tr>
<td>C2 Occiput</td>
</tr>
<tr>
<td>C4 Clavicular region</td>
</tr>
<tr>
<td>C6 Thumb</td>
</tr>
<tr>
<td>C8 Little finger</td>
</tr>
<tr>
<td>T4 Nipple line</td>
</tr>
<tr>
<td>T10 Umbilicus</td>
</tr>
<tr>
<td>L1 Inguinal region</td>
</tr>
<tr>
<td>L3 Knee</td>
</tr>
<tr>
<td>S1 Heel</td>
</tr>
<tr>
<td>S5 Perineal area</td>
</tr>
</tbody>
</table>

Spinal Shock

Definition
› Loss of vascular tone caused by cord trauma lasting 24–48 h, rarely can last several weeks
History
- Spinal cord trauma

Physical Findings
- Hypotension, bradycardia, flaccid paralysis, hyporeflexia

Treatment
- Phenylephrine (Neosynephrine peripheral alpha agonist) for BP support

Disposition
- Admit

Pearls
- There is NO evidence to support the administration of steroids in spinal trauma
- SCIWORA (spinal cord injury w/o radiologic abnl): In pediatric pts, if focal tenderness/ neurologic deficits → treat as cord injury regardless of imaging

THORACIC TRAUMA

Definition
- Injuries to the thorax & its structures caused by penetrating or blunt trauma (25% all trauma Ds; immediate: heart/great vessel injury, early: Airway obstruction/tamponade/tension PTX, Late: PNA/PE)

Approach
- Evaluate anatomical categories although many injuries do not occur in isolation: Cardiac/vascular, pulmonary, skeletal, esophageal, diaphragmatic

Inspection
- External trauma: Open wounds (do not probe wounds: Clot dislodgement → hemorrhage), exit/entrance wounds, flail segments (may require external fixation or PPV), seat belt marks, impaled objects (stabilization → removal in OR)

Palpation
- Crepitance (PTX), unequal pulses (vascular trauma, mediastinal hematoma), wounds below nipple line/tip of scapula ↑ risk abd trauma (25% have both intra-abd + thoracic trauma) (J Trauma 1998;45:87)
Radiology
- See table

Thoracotomy
- Blunt Traumatic Arrest
  - CPR >10 min, do not perform
  - CPR <10 min or profound refractory shock
- Penetrating Trauma Arrest
  - CPR >15 min, do not perform
  - CPR <15 min or profound refractory shock or CPR <5 min penetrating neck or extremity trauma
- Do not transport pt only if pulseless & no electrical cardiac activity in field
- Survival rate in pt w/ arrest from blunt trauma 1.6%, survival rate for arrest from penetrating trauma w/ some signs of life is 31.1% (J Trauma Acute Care Surg 2012;73(6):1359)

<table>
<thead>
<tr>
<th>Thoracic Trauma</th>
<th>General Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blunt trauma</td>
<td>If e/o thoracic trauma exists: CXR, chest CT</td>
</tr>
<tr>
<td>Penetrating trauma—traverses mediastinum</td>
<td>Agonal: Thoracotomy</td>
</tr>
<tr>
<td></td>
<td>Unstable: Place bilateral chest tubes</td>
</tr>
<tr>
<td></td>
<td>Stable: CXR, chest CTA, esophagoscopy, bronchoscopy</td>
</tr>
<tr>
<td>Penetrating trauma—does not traverse mediastinum</td>
<td>CXR &amp;/or chest CT for intrathoracic or extrathoracic injury</td>
</tr>
</tbody>
</table>

Traumatic Aortic Rupture

Definition
- Traumatic rupture of the aorta (descending aorta → fixed to thorax) caused by deceleration injury (fall from height, high-speed MVC, T-boned MVC)

History
- Retrosternal/intrascapular pain (80% die immediately)

Physical Findings
- Exam has poor sens for detecting injury, must have high index of suspicion w/ high mechanism
- Hypotension, asymmetric pulses/BP
Evaluation
- CXR (>8 cm widening of mediastinum, esophageal/trachea deviation, loss of aortic knob/aortopulmonary window, L apical cap, fractures of 1st rib/2nd rib/sternum, widening of paravertebral strip), CTA, TEE

Treatment
- BP control (labetalol/esmolol/nitroprusside): Allow permissive hypotension (SBP 70–90), surgical consult

Disposition
- Admit

Pearl
- 90% who survive have contained hematoma near ligamentum arteriosum
- nl CXR does not rule or aortic injury

Pneumothorax
Definition
- Air in the plural space (simple: w/o shift/communicating w/ outside air, tension: Injury acts as one-way valve/increased intrapleural pressures, open: wall deficit/collapse on inspiration/expansion on expiration/ineffective ventilation)

History
- Blunt (simple) or penetrating (tension/open) trauma

Physical Findings
- Decreased BS, hyperresonance, tension: Tracheal deviation/neck vein distension/ hypotension, open: Chest wound w/ “sucking”

Evaluation
- US, CXR (treat tension PTX prior to imaging), chest CT

Treatment
- 100% O₂
- Tension: Needle decompression (large-bore needle/IV catheter → 2nd intercostal space, midclavicular line), chest tube to 4th–5th intercostal space mid/anterior axillary line
- Open: Sterile occlusive dressing to taped down on 3 sides → allows efflux/not influx of air, chest tube
- Simple: <10% → serial CXR, mod/large → chest tube directed
anteriorly/serial CXR
› Occult: No tx other than O₂
› PPx abx indicated in tube thoracostomy in setting of trauma (World J Surg 2006;30:1843)

Disposition
› Admit

Pearl
› Chest tube must be placed if mechanical ventilation required

Hemothorax
Definition
› Blood in the plural space, most common from lung lacerations

History
› Blunt/penetrating trauma

Physical Findings
› Pain, decreased BS, dullness to percussion

Evaluation
› CXR: Costophrenic angle blunting (upright)/diffuse haziness (supine), US, chest CT

Treatment
› Large-bore chest tube directed inferiorly, surgical consult → OR if >1.5 L bloody output initially (>20 mL/kg)/>200 cc/h (>3 mL/kg/h) or if unstable (↑ likelihood of injury to intercostal/internal mammary/hilar vessels)
› PPx abx indicated in tube thoracostomy in setting of trauma

Pearl
› ~300 cc needed to see hemothorax on CXR

Disposition
› Admit

Flail Chest
Definition
› Fracture >3 or more ribs in 2 or more places → discontinuous segment of chest wall → paradoxical movement w/ respiration (5% of thoracic trauma)
History
- Blunt trauma, SOB

Physical Findings
- Respiratory distress, tenderness, crepitus, paradoxical movement of chest wall

Evaluation
- CXR

Treatment
- External stabilization (pillow), CPAP (1st line if poor oxygenation/ventilation in awake/cooperative pt → lower mortality/PNA rates vs. intubation) (EMJ 22(5):325), ± chest tube placement, pain control (rib block catheter/epidural is best), intubate only if necessary (obtunded, airway obstruction, respiratory distress)

Disposition
- Admit

Pearl
- 35–50% mortality → related to underlying injuries & cx (pulmonary contusions, PNA)

Pulmonary Contusion
Definition
- Injury to lung parenchyma → hemorrhage/edema → V/Q mismatch

History
- Blunt trauma, SOB

Physical Findings
- Respiratory distress, tenderness, tachypnea, tachycardia, hemoptysis, hypoxia ↑ 1–2 d/resolve 7 d

Evaluation
- CXR: May be nl initially, bilateral alveolar infiltrates

Treatment
- Restrict IVF goal euvolemia, intubate if needed

Disposition
- Admit

Cardiac Tamponade
**Definition**
- Hemopericardium → constriction of the heart → decreased CO, most commonly due to penetrating injury (rarely blunt trauma)

**History**
- Penetrating trauma

**Physical Findings**
- Beck triad (hypotension/JVD/muffled heart sounds), tachycardia, pulsus paradoxus

**Evaluation**
- Bedside/formal US: Pericardial effusion/diastolic collapse of RA/RV, ECG: Low voltage/electrical alternans, CXR: Usually unremarkable

**Treatment**
- Aggressive IVF (preload dependent)
- Hypotension + pericardial effusion → OR/pericardiocentesis (blood usually clotted, if fresh may be in RV)
- Arrest → thoracotomy

**Disposition**
- Admit

**Pearl**
- JVD is rare in trauma pts given hypovolemia

**Cardiac Contusion**

**Definition**
- Contusion of the myocardium/coronary vessels/valves/septum

**History**
- Blunt trauma

**Physical Findings**
- Tachycardia, hypotension

**Evaluation**
- ECG: New BBB, dysrhythmias (rare after 1st 24 h), ST Δ/conduction abnl/RV Dysfxn, ± cardiac enzymes (poor sens, levels not predictive of outcome)

**Treatment**
- IV fluid resuscitation (RV damage → preload dependence), see table

**Disposition**
Admit to Tele

**Pearl**

- New ECG Δ consider 1° cardiac event → trauma

<table>
<thead>
<tr>
<th>Cardiac Contusion</th>
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<tbody>
<tr>
<td>Asymptomatic, no ECG Δ, no dysrhythmias</td>
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<tr>
<td>ECG Δ or dysrhythmia in HD stable pt</td>
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<tr>
<td>ECG Δ or dysrhythmia in HD unstable pt</td>
</tr>
<tr>
<td>Life-threatening dysrhythmias</td>
</tr>
</tbody>
</table>

**Esophageal Injury**

**Definition**

- Injury to the esophagus most commonly from penetrating trauma (possible w/ significant epigastric blunt trauma)

**History**

- Penetrating trauma

**Physical Findings**

- Respiratory distress, neck/chest crepitus, hematemesis
- Often will have severe other injuries in blunt trauma

**Evaluation**

- CXR: Mediastinal/deep cervical air, neck films: Esophageal + laryngeal injury → air column in the esophagus, flexible esophagoscopy + esophagram (90% sens), CT

**Treatment**

- Surgical consult for operative management, broad-spectrum abx

**Disposition**

- Admit

**Tracheobronchial Tear**

**Definition**

- Tear to trachea/bronchus, most commonly due to penetrating trauma

**History**

- Penetrating trauma or severe deceleration injury, often die at scene

**Physical Findings**

- Crepitance, large persistent air leak or recurrent ptx after chest tube
placement (if cervical injury may not have air leak)

**Evaluation**

- CXR: PTX/pneumomediastinum/“fallen lung sign,” chest CT, bronchoscopy (gold standard, may miss injuries >2 cm above carina)

**Treatment**

- Fiberoptic intubation (in major bronchial lesions → consider double lumen ETT), chest tube placement (may require >1 chest tube)

**Disposition**

- Admit

**Pearl**

- May p/w difficulty passing ETT/difficulty w/ ventilation after ETT intubation

---

**ABDOMINAL TRAUMA**

**Definition**

- Trauma to the abdomen & its structures

**Approach**

**Evaluate 4 Main Areas**

- Anterior abdomen: nipple line → inguinal ligaments/pubic symphysis → anterior axillary line, Flank: B/w anterior & posterior axillary lines from 6th rib → iliac crest, Back: Inf scapular tips → iliac crest, gluteal region: Iliac crest → gluteal fold

**Inspection**

- Entrance/exit wounds (check b/w buttock/thigh/axilla/neck), seat belt sign (↑ risk mesenteric tear/avulsion, bowel perforation, aorta/iliac thrombosis, chance fracture of L1/L2), do not remove objects, cover eviscerated organs in saline soaked gauze

**Palpation**

- Peritoneal signs (operative management), rectal exam (high-riding prostate/blood/tone)

**Labs**

- CBC (Hct may be nl initially in setting of hemorrhage), ABG, lactate, LFTs, lipase, UA
Radiology
- FAST (90–100% sens for hemoperitoneum, not spec), CXR (abd free air), pelvic x-ray (loss of psoas shadow → retroperitoneal injury, location of bullets), CT (definitive test, low sens for early pancreatic/diaphragmatic/bowel injury)

Diagnostic Peritoneal Lavage (DPL)
- Rarely used given FAST/CT scans, positive study → >10 cc gross blood or enteric contents, blunt trauma >100000 RBCs, penetrating trauma >5000–10000 RBC

Liver Laceration

Definition
- Laceration to liver (most commonly injured organ)

History
- Blunt or penetrating trauma

Physical Findings
- ± RUQ tenderness

Evaluation
- LFTs, HCT, FAST, CT scan: Grading of laceration (I–VI)

Treatment
- Surgical consultation for operative vs. conservative management (HD stable, serial exams/HCT)

Disposition
- Admit ICU vs. floor

<table>
<thead>
<tr>
<th>Approach to Abd Trauma</th>
<th>Abd Trauma</th>
<th>Examples</th>
<th>Most Common Injuries</th>
<th>General Guidelines</th>
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<tbody>
<tr>
<td></td>
<td>Blunt trauma</td>
<td>Motor vehicle crash, falls, assaults</td>
<td>Spleen, liver, intestine, kidney</td>
<td>Unstable + distention → OR Unstable → FAST Stable → CT (IV contrast only), FAST, or serial abd exams</td>
</tr>
<tr>
<td></td>
<td>Penetrating trauma— anterior abdomen</td>
<td>GSW, SW</td>
<td>Small bowel, colon, liver, &amp; vascular</td>
<td>GSW to anterior abdomen → OR</td>
</tr>
</tbody>
</table>
structures (GSW); liver, small bowel, diaphragm

| Penetrating trauma—flank & back | GSW, SW | Unstable w/ non-GSW trauma → OR
Stable w/ non-GSW trauma → local wound exploration, CT

Notes:
(1) 1° objective is to identify need for surgical exploration.
(2) Peritoneal irritation often seen w/ hollow viscus injury, but not w/ hemoperitoneum.
(3) If fascia penetration is found, f/u w/ DPL, CT, or ex lap. If not pt can be D/C after wound care.
(4) Intra-abd organ injury occurs in 20% of non-GSW flank injuries & 5–10% of non-GSW back injuries.

**Splenic Laceration**

**Definition**
- Laceration to spleen (most commonly injured organ in blunt trauma)

**History**
- Blunt or penetrating trauma, L shoulder pain (Kehr sign)/chest/flank/upper quadrant pain

**Physical Findings**
- LUQ pain

**Evaluation**
- FAST, CT scan: Grading of laceration (I–V)

**Treatment**
- Surgical consultation for operative vs. conservative management (HD stable, serial exams/HCT), IR for embolization

**Disposition**
- Admit ICU vs. floor

<table>
<thead>
<tr>
<th>Splenic Laceration Grading</th>
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<tbody>
<tr>
<td>Grade</td>
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<td>III</td>
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<td>IV</td>
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<td>V</td>
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</table>

**Small Bowel Injury**

**Definition**
- Injury to small bowel (GSW > SW > blunt trauma)

**History**
- Blunt or penetrating trauma, classically handlebar injury

**Physical Findings**
- Seat belt sign (MVC), peritoneal signs (may be delayed)

**Evaluation**
- Unstable → FAST/DPL, Stable → CT scan (low sens, fluid collection/bowel-wall thickening/stranding/free air) CXR (rarely shows free air), Lumbar XR (Chance fracture)

**Treatment**
- Surgical consultation for operative management (perforation or devascularization), abx (ampicillin/ciprofloxacin/metronidazole)

**Disposition**
- Admit

**ColoRectal Injury**

**Definition**
- Injury to colon or rectum (transverse colon most common)

**History**
- Penetrating trauma (GSW)

**Physical Findings**
- Hypoactive bowels, peritoneal signs, gross rectal blood

**Evaluation**
• Triple contrast CT scan (Gastrografin, barium is irritating), KUB (air lining psoas), f/u sigmoidoscopy

**Treatment**

• Surgical consultation for operative management (perforation or devascularization), abx (ampicillin/ciprofloxacin/metronidazole)

**Disposition**

• Admit

**Duodenal Injury**

**Definition**

• Injury to duodenum (80% a/w other injury)

**History**

• Penetrating trauma, N/V (obstructing hematoma)

**Physical Findings**

• Epigastric tenderness, heme positive stool, bloody NGT aspirate

**Evaluation**

• Upright CXR (free air), CT scan (duodenal wall hematoma), Upper GI (“coiled spring” area)

**Treatment**

• Surgical consultation for operative management (perforation or devascularization), abx (ampicillin/ciprofloxacin/metronidazole), NGT placement

**Disposition**

• Admit

**Pearls**

• 2nd portion most commonly injured (contains bile/pancreatic duct openings)
• Mortality 40% if dx delayed 24 h

**Gastric Injury**

**Definition**

• Injury to stomach, uncommon

**History**

• Penetrating trauma

**Physical Findings**
Epigastric tenderness, heme positive stool, bloody NGT aspirate

**Evaluation**
- Upright CXR (free air)

**Treatment**
- Surgical consultation for operative management, abx (ampicillin/ciprofloxacin/metronidazole)

**Disposition**
- Admit

---

**Pancreatic Injury**

**Definition**
- Injury to pancreas (75% penetrating trauma)

**History**
- Penetrating trauma, direct epigastric trauma (steering wheel, bicycle handles)

**Physical Findings**
- Minimal epigastric tenderness (retroperitoneal structure)

**Evaluation**
- CT scan (low sens early), lipase (may be nl), ERCP for ductal injury

**Treatment**
- Surgical consultation

**Disposition**
- Admit

**Pearl**
- A/w other injuries 90% of the time

---

**Vascular Trauma**

**Definition**
- Injury to abd vasculature (10% of SW, 25% of GSW)

**History**
- Penetrating trauma

**Physical Findings**
- Distension, expanding hematoma, Grey–Turner sign (flank ecchymosis)/Cullen sign (periumbilical ecchymosis) → retroperitoneal hemorrhage
**Evaluation**
- FAST, CT scan (if stable), wound exploration

**Treatment**
- Surgical consultation, unstable → OR

**Disposition**
- Admit

**Pearl**
- Avoid LE venous access

**Diaphragmatic Tear**

**Definition**
- Tear to diaphragm from blunt trauma, ↑ lateral impact (large, L-sided 2–3× more likely than R, posterolaterally located) or penetrating trauma (small but enlarge w/ time)

**History**
- Penetrating/blunt trauma, delayed presentation; pain, ± obstruction

**Physical Findings**
- BS over chest

**Evaluation**
- CXR (50% sens): Hemothorax/PTX (penetrating), abnl diaphragmatic shadow (blunt), US, CT scan, careful NGT placement (may be seen in hemithorax)

**Treatment**
- Respiratory distress → NGT placement for decompression, surgery consult for operative repair. CXR may be misinterpreted as hemothorax, avoid chest tube placement

**Disposition**
- Admit

**Pearl**
- Intrapericardial diaphragmatic rupture/bowel herniation → tamponade

---

**GENITOURINARY TRAUMA**

**Definition**
Trauma to the structures of the genitourinary tract, uncommon to be life-threat unless significant renal/vascular injury

**Approach**

**Inspection**
- Blood at meatus (urethra trauma), blood in vagina, perineal lacerations (do not probe → hemorrhage), scrotal ecchymosis/lacerations, flank bruising

**Palpation**
- Rectal exam (high-riding prostate/boggy → membranous urethral injury, blood → rectal laceration), testicular disruption

**Labs**
- UA (microscopic hematuria → no eval, gross blood → serious GU trauma)

**Radiology**
- RUG: Males w/ blood at meatus before Foley placement (to prevent full urethral tear/false passage), inject 50 cc contrast into urethra → pelvic x-ray for extravasation
- Cystogram: Instill 400–500 cc contrast into bladder via Foley → AP film or CT scan → repeat image after contrast is washed out (posterior bladder tears)
- IV pyelogram: Rarely indicated
- CT scan (IV contrast): Complete eval of kidneys

**Renal Laceration**

**Definition**
- Laceration to kidney (major: Extend to medulla/collection system, minor: No involvement of collecting system/medulla, no extraversion of urine, pedicle: injury to renal vasculature)

**History**
- Penetrating trauma

**Physical Findings**
- Flank wound, gross hematuria, ± hypotension

**Evaluation**
- CBC, UA, other trauma labs as needed, CT scan: Eval extent of injury

**Treatment**
• Surgery consult, minor lacerations may be nonoperative

**Disposition**

• Admit

**Renal Contusion**

**Definition**

• Contusion to kidney

**History**

• Blunt trauma

**Physical Findings**

• Flank ecchymosis

**Evaluation**

• UA (if neg → no further testing), CT scan

**Treatment**

• Surgery consult, subcapsular hematoma → 24 h observation/serial HCT/serial UA/bed rest, microscopic hematuria → avoid strenuous exercise/repeat UA in 2 d + until clear

**Disposition**

• Admit: Major/subcapsular hematoma
• Home: Microscopic hematuria

**Renal Pedicle/Vascular Injury**

**Definition**

• Injury to renal pedicle or vasculature

**History**

• High-velocity deceleration, penetrating trauma

**Physical Findings**

• Flank ecchymosis, hypotension

**Evaluation**

• UA, CBC, Coags, BMP, CT scan: Nonenhancing kidney/± perirenal hematoma

**Treatment**

• Surgery consult for operative management → repair (20% salvage rate in pedicle lacerations) vs. nephrectomy

**Disposition**
Renal Pelvis Rupture

Definition
- Rupture of the renal pelvis

History
- High-velocity deceleration, penetrating trauma

Physical Findings
- Flank ecchymosis, hypotension

Evaluation
- UA, CBC, Coags, BMP, CT scan: Extravasation of urine in perirenal space

Treatment
- Urology consult for operative repair

Disposition
- Admit

Pearl
- ↑ risk of infection in delayed repair

Ureteral Injuries

Definition
- Injury to ureter (very rare), majority are iatrogenic from gyn/uro procedures

History
- Hyperextension, penetrating trauma, forced flexion of L-spine → rupture below UPJ, delayed necrosis from microvascular injury after GSW (rare)

Evaluation
- UA, HCT, CT scan: Extravasation of urine, IVP (limited sens)

Treatment
- Urology consult for operative ureteroureterostomy

Disposition
- Admit

Intraperitoneal Bladder Rupture
**Definition**
- Laceration at dome of bladder w/ intraperitoneal communication

**History**
- MVC, blunt trauma (burst injury)

**Physical Findings**
- Lower abd tenderness, ↓ UOP, hematuria

**Evaluation**
- UA, HCT, CT cystogram/cystogram: Extravasation of urine

**Treatment**
- Urology consult for urgent operative repair

**Disposition**
- Admit

---

**Extraperitoneal Bladder Rupture**

**Definition**
- Rupture of the bladder w/ extraperitoneal spillage

**History**
- MVC, blunt trauma

**Physical Findings**
- Lower abd tenderness, ↓ UOP, hematuria

**Evaluation**
- UA, HCT, CT cystogram/cystogram w/ washout: Extravasation of urine

**Treatment**
- Urology consult (usually nonoperative unless extends to bladder neck), Foley 10–14 d

**Disposition**
- Admit

---

**Male Urethral Injuries**

**Definition**
- Injury to posterior (prostatomembranous) urethra (a/w pelvic fractures, esp bilateral or both ipsilateral pubic rami fx & posterior disruption injuries) & anterior (bulbous/penile) urethra (a/w direct trauma to penis/penile fracture/saddle injuries/falls/GSW)
History
- Blunt or penetrating trauma

Physical Findings
- Blood at meatus, gross hematuria, inability to void

Evaluation
- UA, HCT, RUG (prior to Foley)

Treatment
- Suprapubic bladder decompression if needed, urology consult for 1° repair/
  fluoroscopic catheter placement/suprapubic cystotomy

Disposition
- Admit

Female Urethral Injuries
Definition
- Injury to female urethra associated most commonly w/ pelvic fractures
  (rarely saddle injuries, falls, GSW, instrumentation)

History
- Blunt or penetrating trauma, much less common than in males

Physical Findings
- Vaginal bleeding, inability to place Foley, labial edema

Evaluation
- RUG not useful, passage of Foley precludes complete tear

Treatment
- Suprapubic bladder decompression if needed, urology consult for
  surgical repair

Disposition
- Admit

Testicular Contusion/Rupture
Definition
- Blunt trauma to the testicle leading to contusion or rupture (disruption of
tunica albuginea)

History
- Blunt trauma, pain, swelling
Physical Findings
- Ecchymosis, edema, tenderness, inability to palpate testicle due to dislocation

Evaluation
- Testicular US (mod sens/spec for rupture)

Treatment
- Urology consult for surgical repair/clot evacuation (early intervention \( \rightarrow \) ↓ morbidity)

Disposition
- Admit

Penile Fracture

Definition
- Blunt injury to the erect penis when penis is forcefully bent leading to rupture of the tunica albuginea/rupture of corpora cavernosa

History
- “Cracking sound” usually during sexual activity \( \rightarrow \) severe pain

Physical Findings
- Swelling, discoloration (vascular engorgement), ecchymosis, blood at meatus (10–20% a/w urethral injury)

Evaluation
- RUG for urethral injury (concomitant injury in 15–20%)

Treatment
- Urology consult for surgical urethral repair/clot evacuation

Disposition
- Admit

Penile Amputation/Laceration

Definition
- Complete or partial amputation/laceration of the penis

History
- Penetrating trauma, zipper injury

Evaluation
- RUG or testicular US if associated injuries suspected

Treatment
Amputation: Urology consult (best results in reimplanted in 18 h)
- Simple laceration: Repair w/ absorbable suture
- Zipper injury: Remove zipper w/ mineral oil/wire cutters at zipper median bar to break apart

Disposition
- D/C unless reimplantation required

Female Genital Injuries
Definition
- Injury to ovary, uterus, fallopian tube, vagina (difficult to Dx usually found when evaluating for other injury), a/w pelvic fractures

History
- Blunt or penetrating trauma, vaginal bleeding

Physical Findings
- Blood in vaginal vault, lower abd tenderness

Evaluation
- CT scan, pelvic US (in gravid pt, ↑ risk)

Treatment
- Open vaginal lacerations open → abx (ampicillin, gentamicin, Flagyl)
- GYN consult
- Simple vaginal lacerations: Repair w/ absorbable suture

Disposition
- Admit if needed

HIP/PELVIC TRAUMA

Definition
- Trauma to hip or pelvis

Approach

Pelvis Anatomy
- Sacrum, coccyx, & R/L innominate bones (ileum, ischium, pubis) fuse at acetabulum

Inspection
- Perineal edema/lacerations/ecchymosis, deformities (length
discrepancy, internal/external rotation)

**Palpation**
- Rectal exam (blood, high-riding prostate, tone), pulses, pelvic stability (limit manipulation if unstable → clot dislodgement), neurologic exam (strength, sensation, DTRs), in females pelvic exam

**Radiology**
- AP pelvis (can miss sacral fractures/SI joint disruptions → inlet/outlet views), CT scan (superior for acetabular fractures/associated injuries), hip x-ray

**Pelvic Fractures**

**Definition**
- Fractures of the pelvis usually caused by significant mechanism (↑ association w/ other injuries)

**History**
- Blunt trauma, lateral/AP compression, vertical shear (fall)

**Physical Findings**
- External contusion/abrasion/ecchymosis, caution w/ manual compression/distraction of pelvis (may dislodge clot → hemorrhage), evaluate for open pelvic fx as these have 40–50% mortality, hypotension (42–50% mortality), blood at meatus, perineal trauma, neurologic abnl (cauda equina syndrome, plexopathies, radiculopathies)

**Evaluation**
- FAST, AP pelvis, CT scan, evaluate carefully for intra-abd trauma as >15% w/ pelvic fx will have intra-abd injury

**Treatment**
- Unstable: Temporizing measures (wrapped sheet/external binders/external clamps), immediate orthopedic & trauma surgery consult (reduction/external fixation & pelvic packing), IR for hemorrhage control
- Stable: Orthopedic consult

**Disposition**
- Admit

**Pearls**
- Type A (inferior pubic rami/avulsion) & type B2 (bucket handle) → most
common

- Type B3 (open book) & C (70% have major associated injuries) → most life-threatening

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<th>Pelvic Fracture Classification (Tile Classification System)</th>
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<tr>
<td><strong>Type</strong></td>
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<tr>
<td>Type A: Stable pelvic ring fracture</td>
</tr>
<tr>
<td>A1: Avulsion of innominate bone → sudden muscle contraction, iliac wing (Duverney) fracture → direct lateral to medical trauma</td>
</tr>
<tr>
<td>A2: Stable or minimally displaced fracture of pelvic ring (ramus/ischium) → elderly fall</td>
</tr>
<tr>
<td>A3: Transverse fracture of sacrum/coccyx → fall in sitting position</td>
</tr>
<tr>
<td>Type B: Partially stable pelvic ring injuries (rotationally unstable/vertically stable)</td>
</tr>
<tr>
<td>B1: Unilateral open book (disruption symphysis pubis + SI joint hinge rotation) → AP compression</td>
</tr>
<tr>
<td>B2: Bucket handle fractures → lateral compression</td>
</tr>
<tr>
<td>B3: Bilateral open book fracture → severe AP compression</td>
</tr>
<tr>
<td>Type C: Unstable pelvic ring fractures</td>
</tr>
<tr>
<td>Distracting vertical sacral fractures/other vertical shear fractures → vertical shear injuries</td>
</tr>
</tbody>
</table>

Vascular Pelvic Injuries

**Definition**

- Injury to vascular structures of pelvis a/w pelvic fractures (most commonly AP trauma or vertical shear)

**History**

- Blunt trauma, lateral/AP compression, vertical shear (fall)

**Physical Findings**

- Unstable pelvis, hypotension resistant to resuscitation

**Evaluation**

- FAST, AP pelvis, CT scan (if stable), pelvic angiography, consider DPA if FAST neg but HD unstable

**Treatment**

- Stabilization of pelvis, orthopedic & trauma surgery consult (external fixation & pelvic packing to control hemorrhage), IR embolization for continued hypotension (less effective for venous bleed → high collateralization)

**Disposition**

- Admit

Acetabular Fractures
Definition
- Fractures to the acetabulum (MVC → knee striking dashboard or lateral intrusion), fall in elderly

History
- Blunt trauma, pain w/ movement of hip

Physical Findings
- Pain w/ movement of hip/compression of sole of foot or greater trochanter

Evaluation
- AP pelvis, lateral hip films (± Judet views), CT scan (if plain films unrevealing)
- 3 types (although some fit in multiple categories
  - Wall: Anterior, posterior, posterior wall/column, transverse/posterior wall
  - Column: Anterior, posterior, both, posterior wall/column, anterior/transverse
  - Transverse: Transverse, T, transverse/posterior wall, anterior column/transverse

Treatment
- Orthopedic consult for operative management

Disposition
- Admit

Hip Fractures

Definition
- Fractures of the hip (femoral head/neck/trochanter)

History
- Elderly → fall from standing, young → significant mechanism trauma (MVC)

Physical Findings
- External rotation, shortening of leg

Evaluation
- AP pelvis, lateral hip films, CT (if unable to bear weight + neg plain films)

Treatment
- Orthopedic consult for operative management (femoral neck fractures
→ ↑ risk avascular necrosis of femoral head, surgical repair in <6 h)

**Disposition**
- Admit

**Pearl**
- Hip fracture in elderly → 25% 1-y mortality

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracapsular</td>
<td>Femoral head: Rare in isolation, a/w posterior dislocations&lt;br&gt;Femoral neck: ↑ risk avascular necrosis of femoral head, most common in elderly women</td>
</tr>
<tr>
<td>Extracapsular</td>
<td>Intertrochanteric: Markedly externally rotated/shortened, elderly fall&lt;br&gt;Subtrochanteric: ↑ risk bleeding into thigh, elderly fall/MVC</td>
</tr>
</tbody>
</table>

**Hip Dislocations**

**Definition**
- Dislocation of femoral head from acetabulum (90% posterior)

**History**
- Elderly fall w/ hx of hip total hip replacement, MVC (knee hitting dashboard, a/w other injuries), athlete running & lands w/ hip flexed/internally rotated & adducted

**Physical Findings**
- Flexed/adducted/internally rotated hip (posterior)

**Evaluation**
- AP pelvis, lateral hip films

**Treatment**
- Orthopedic consult if fracture or prosthetic hip, reduction under conscious sedation (in <6 h, ↑ risk avascular necrosis of femoral head)

**Disposition**
- Admit if needed

**EXTREMITITY INJURY**

**Definition**
Injuries to the extremities (vascular/bony/soft tissue/nerve)

**Approach**

**History**
- Last tetanus (booster if >5 y), hand dominance, time of injury, mechanism (crush/penetrating), neurologic deficit (loss of sensation/motor), environmental exposures (burn/cold), preinjury functional status

**Inspection**
- Color (discoloration/ecchymosis/perfusion), soft tissue defects (control hemorrhage during 1° survey), deformities (angulations/shortening), swelling

**Palpation**
- Pulses, all joints/bones (tenderness), FB, crepitance, strength, sensation, DTRs, range all joints, joint effusions

**Radiology**
- Plain films guided by PE

**Consults**
- Orthopedic &/or vascular for open fractures/amputations/vascular injuries/compartment syndrome, hand surgery for significant hand injuries

**Extremity Vascular Injury**

**Definition**
- Injury to the vasculature of the extremities

**History**
- Blunt trauma (fracture/dislocation → tearing of vessels) or penetrating trauma

**Physical Findings**
- Obvious vascular compromise → pulseless/pallor/pain/paresthesia/cold, indicators of vascular injury → swelling/pain/↓ cap refill/mottled skin/↓ pulses

**Evaluation**
- Plain films (blunt trauma), CTA, or angiography (if stable), Ankle/Brachial index or Ankle/Ankle index: Abnl if <0.9

**Treatment**
Vascular surgery consult for immediate surgical repair (↓ salvage rate if >6 h)

Disposition
- Admit if needed

**Extremity Orthopedic Injuries**

**Definition**
- Bony fractures or joint dislocations of the extremities

**History**
- Blunt trauma or penetrating

**Physical Findings**
- Deformities, pain, swelling, crepitance, neurologic deficits, diminished pulses

**Evaluation**
- Plain films, image joint above & below for significant fracture, CT in certain injuries (tibial plateau)

**Treatment**
- Open fractures: Immediate orthopedic consult for operative washout/fixation (<6 h), abx (cefazolin 1–2 g)
- Closed UE fractures + intact neuro exam: Splint, outpt f/u (see table)
- Closed LE fractures + intact neuro exam: Splint, outpt f/u if able to use crutches (see table)
- Dislocations: ED reduction, pt f/u

**Disposition**
- Admit if needed

<table>
<thead>
<tr>
<th>Fracture Sites</th>
<th>Splint/Immobilization Technique</th>
<th>Referral Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral fractures</td>
<td>Temporary traction splints</td>
<td>Emergent ED consult</td>
</tr>
<tr>
<td>Knee injuries (w/ no e/o dislocation &amp; no neurovascular compromise)</td>
<td>Knee immobilizer/long leg cast w/ leg flexed 10°</td>
<td>Orthopedic f/u w/i 1 wk</td>
</tr>
<tr>
<td>Tibia fractures (not tibial plateau)</td>
<td>Lower leg posterior splint</td>
<td>Orthopedic f/u in 1–2 d</td>
</tr>
<tr>
<td>Ankle fractures</td>
<td>Lower leg posterior U-splint</td>
<td>If fracture fragments well aligned, orthopedic f/u in 1 wk. If angulation or distraction, needs next-day f/u</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Hand fractures</td>
<td>Thumb &amp; index finger—radial gutter splint Middle, ring, &amp; little fingers—ulnar gutter splint</td>
<td>If fracture fragments well aligned &amp; fracture is closed, f/u w/ hand surgeon w/i 1 wk</td>
</tr>
<tr>
<td>Wrist fractures</td>
<td>Wrist splint/immobilizer unless scaphoid fracture then thumb spica cast</td>
<td>Orthopedic f/u in 7–10 d unless scaphoid displacement—then f/u in 1–2 d</td>
</tr>
<tr>
<td>Distal radius/ulna fractures</td>
<td>Short arm cast</td>
<td>Orthopedic f/u in 1–2 d unless closed reduction results in good anatomic alignment—then f/u in 7–10 d</td>
</tr>
<tr>
<td>Humerus fractures</td>
<td>Sling, coaptation splint rarely used</td>
<td>Orthopedic f/u in 7–10 d, sooner if articular surface or tuberosity</td>
</tr>
<tr>
<td>Shoulder dislocations</td>
<td>Sling, early ROM to prevent frozen shoulder</td>
<td>Orthopedic f/u in 7–10 d</td>
</tr>
</tbody>
</table>

**Pearls**
- 5th MCP fractures or “Boxer fractures” have a high rate of infection secondary to breaks in skin from opponent’s tooth. Always r/o FB w/plain radiographs & f/u in 1–2 d in ED or in hand clinic
- Scaphoid tenderness w/o radiologic e/o fracture requires splinting & orthopedic f/u & repeated x-rays w/i 7 d
- ED, emergency department; MCP, metacarpal

**Extremity Soft Tissue Injury**

**Definition**
- Injury to the soft tissue of the extremities

**History**
- Blunt trauma or penetrating (polytrauma, industrial accidents)

**Physical Findings**
- Soft tissue defects, FBs

**Evaluation**
- Plain films for FB/underlying fractures, US, CPK (if extensive injury)

**Treatment**
- Irrigate, explore for FB (↑ risk wound infection → poor cosmetic outcome), plastic surgery consult (extensive injuries), hand consult for palmar injuries as exploration w/ potential for iatrogenic injury, abx
(grossly contaminated wounds)

**Disposition**
- Admit if extensive, e/o rhabdomyolysis/compartment syndrome

**Extremity Nerve Injury**

**Definition**
- Injury to the nerves of the extremities (a/w fractures/dislocations/lacerations/vascular ischemia/compartment syndrome)

**History**
- Blunt trauma or penetrating

**Physical Findings**
- See table

**Evaluation**
- Plain films for fracture/dislocation

**Treatment**
- Reduce fracture/dislocation (↓ pressure on nerve), fasciotomy (compartment syndrome), orthopedic/plastic surgery consult

**Disposition**
- Admit if needed

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Motor</th>
<th>Sensation</th>
<th>Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulnar</td>
<td>Index finger abduction</td>
<td>Tip of little finger</td>
<td>Elbow injury</td>
</tr>
<tr>
<td>Median (distal)</td>
<td>Thenar opposition</td>
<td>Tip of index finger</td>
<td>Wrist dislocation</td>
</tr>
<tr>
<td>Median (anterior interosseous)</td>
<td>Index tip flexion</td>
<td></td>
<td>Supracondylar fx of humerus in children</td>
</tr>
<tr>
<td>Musculocutaneous</td>
<td>Elbow flexion</td>
<td>Lateral forearm</td>
<td>Anterior shoulder dislocation</td>
</tr>
<tr>
<td>Radial</td>
<td>Thumb, finger MCP extension</td>
<td>1st dorsal web space</td>
<td>Distal humeral shaft, anterior shoulder dislocation</td>
</tr>
<tr>
<td>Axillary</td>
<td>Deltoid</td>
<td>Lateral shoulder</td>
<td>Proximal humerus fx, anterior shoulder dislocation</td>
</tr>
<tr>
<td>Femoral</td>
<td>Knee extension</td>
<td>Anterior knee</td>
<td>Pubic rami fx</td>
</tr>
<tr>
<td>Compartment Syndrome</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Definition</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‣ A condition in which perfusion pressures &lt; tissue pressures in closed space (fascial compartments) → ↓ circulation/tissue fxn (↑ risk injuries: Tibial/forearm fractures, crush injuries, burns, immobilized injuries in tight dressing/cast)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>History</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>‣ Blunt trauma or penetrating, pain &gt; than expected/worse w/ passive muscle stretching</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Physical Findings</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>‣ Tenderness, tense swelling, classically: Pain, pallor, paresthesias, paralysis, pulselessness (late finding). Pain w/ passive stretching is early sign but not always reliable</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Evaluation</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>‣ Compartment pressures (measure w/ Stryker or 18 G IV + arterial line transducer) &gt;30 mmHg or &lt;20–30 mmHg difference b/w DBP &amp; compartment pressure (if hypotensive necrosis occurs at ↓ pressures), CK</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Treatment</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>‣ Remove restrictive dressings/casts, elevate extremity, correct BP, surgery consult for fasciotomy (do not delay fasciotomy for surgical availability)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Disposition</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>----------------</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Obturator</th>
<th>Hip adduction</th>
<th>Medial thigh</th>
<th>Obturator ring fx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior tibial</td>
<td>Toe dorsiflexion</td>
<td>Sole of foot</td>
<td>Knee dislocation</td>
</tr>
<tr>
<td>Superficial peroneal</td>
<td>Ankle eversion</td>
<td>Lateral dorsum of foot</td>
<td>Fibular neck fx, knee dislocation</td>
</tr>
<tr>
<td>Deep peroneal</td>
<td>Ankle/toe dorsiflexion</td>
<td>Dorsal 1st to 2nd web space of foot</td>
<td>Fibular neck fx, compartment syndrome</td>
</tr>
<tr>
<td>Sciatic</td>
<td>Plantar &amp; dorsiflexion</td>
<td>Foot</td>
<td>Posterior hip dislocation</td>
</tr>
<tr>
<td>Superior gluteal</td>
<td>Hip abduction</td>
<td>Acetabular fx</td>
<td></td>
</tr>
<tr>
<td>Inferior gluteal</td>
<td>Gluteus maximus hip extension</td>
<td>Acetabular fx</td>
<td></td>
</tr>
</tbody>
</table>

---
Admit

**Crush Syndrome/Rhabdomyolysis**

**Definition**
- Crush injury → release in cellular contents of muscle cells → CK levels >5000 U/L

**History**
- Crush injury

**Physical Findings**
- May have minimal external injury, dark brown/orange urine

**Evaluation**
- CK levels >5000 U/L, ↑ Cr (15–47% a/w ARF), ↑ potassium, UA (+ myoglobin), observe closely for reperfusion syndrome, esp if in field

**Treatment**
- IV fluids for UOP >1 mL/g/h, traditionally alkalization of urine (sodium bicarbonate, 1 amp/1 L NS → urine pH >7 → prevents tubular precipitation of myoglobin) → no difference than NS in prevention of renal failure, treat hyperkalemia *(J Trauma 2004;56:1191)*

**Disposition**
- Admit

**Partial/Complete Amputation**

**Definition**
- Amputation of extremity

**History**
- Blunt or penetrating trauma (polytrauma, industrial accident)

**Physical Findings**
- Document motor/neurologic/vascular fxn in remaining limb

**Evaluation**
- Plain films of stump + amputated fragment, ± angiography (if not going directly to OR)

**Treatment**
- Limit mobility, hemostasis w/ direct pressure, immediate surgery consult for replantation, abx (cefazolin 1–2 g IV), pack stump w/ sterile NS soaked gauze, wrap amputated part in cold NS soaked gauze/place on
ice (do not place in direct contact w/ ice or NS)

**Disposition**

- Admit

**Pearl**

- Replantation depends on age, vocation, injury severity

---

**WOUND MANAGEMENT**

**Approach**

**History**

- Time of event (>12 h → irrigate/heal by secondary intention or delayed 1° closure, face/significant soft tissue defect → 1° closure in <24 h), location (suture selection/time until removal), mechanism (↑ risk FB/contamination), tetanus (booster if >5 y)

**Inspection**

- FB, wound approximation

**Palpation**

- Pulses, strength, sensation distal to injury

**Laceration**

**Definition**

- Cut or tear to skin & soft tissues

**History**

- Penetrating or blunt trauma

**Physical Findings**

- Skin defect, ↓ pulses/sensation/motor (neurovascular injury)

**Evaluation**

- Plain films only if FB/fracture suspected

**Treatment**

- Hemostasis: Direct pressure, lidocaine w/ epinephrine if needed (avoid in digits, nose, ears, penis), hemostatic agents (eg, thrombin, Surgicel), proximal tourniquet
- Analgesia: Use regional blocks when possible (↓ wound distortion/amount of analgesic needed)
### Commonly Used Local Anesthetics

<table>
<thead>
<tr>
<th>Agent</th>
<th>Trade Name</th>
<th>Class</th>
<th>Concentrations (%)</th>
<th>Maximum Safe Dose</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine w/epinephrine</td>
<td>Xylocaine</td>
<td>Amide</td>
<td>0.5–2</td>
<td>4.5 mg/kg 7 mg/kg</td>
<td>~5 min</td>
<td>1–2 h 2–4 h</td>
</tr>
<tr>
<td>Bupivacaine w/epinephrine</td>
<td>Marcaine</td>
<td>Amide</td>
<td>0.125–0.25</td>
<td>2 mg/kg 3 mg/kg</td>
<td>~5 min</td>
<td>4–8 h 8–16 h</td>
</tr>
<tr>
<td>Procaine w/epinephrine</td>
<td>Novocaine</td>
<td>Ester</td>
<td>0.5–1</td>
<td>7 mg/kg 9 mg/kg</td>
<td>~5 min</td>
<td>15–45 min 30–90 min</td>
</tr>
</tbody>
</table>

- Irrigation: >500 cc NS (no benefit over tap water) ([Ann Emerg Med](1999;34:356)), 8 psi of pressure (18 g IV catheter or Zerowet splash shield in 30–60 cc syringe), caution on delicate tissues (eye lids)
- Exploration (through a full ROM): FB, tendons (including in position of injury), fascial planes
- Repair:

<table>
<thead>
<tr>
<th>Suture Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body Part</strong></td>
</tr>
<tr>
<td>Scalp</td>
</tr>
<tr>
<td>Face</td>
</tr>
<tr>
<td>Chest</td>
</tr>
<tr>
<td>Back</td>
</tr>
<tr>
<td>Forearm</td>
</tr>
<tr>
<td>Finger/hand</td>
</tr>
<tr>
<td>LE</td>
</tr>
</tbody>
</table>

- Abx: Not routinely required (must be given for certain bites)

**Disposition**
- Home

**Pearls**
- Scarring: Take up to 1 y to fully develop, apply sunscreen/keep covered even on cloudy days, apply Vit E
- Hand flexor tendon lacerations: Emergent 1° repair by hand surgeon, splint (wrist 30° flexion, MP joint 70° flexion, DIP/PIP 10° flexion)
Hand extensor tendon lacerations: Zone IV & VI repair 1° in ED, splint, hand surgery f/u

**Foreign Body**

**Definition**
- Retained FB in wound (most common hand/foot) → ↑ risk delayed infection/
  granuloma/formation/local compression of structures/embolization/allergic rxns (reactive FBs: Wood, organic matter, clothing, skin fragments)

**History**
- Know FB, ↑ risk wounds: Stepping on glass/punching windows/MVC w/ glass exposure/
  fall on gravel/pain at IVD site/persistent wound infections/failure to heal (41% wounds caused by glass)

**Physical Findings**
- Visible/palpable FB

**Evaluation**
- Explore wound (adequate anesthesia/hemostasis/probe w/ instrument),
  plain films for radiopaque FBs (glass, metal, bone, teeth, graphite, gravel), US (use 100 cc bag of NS or other transducing material for superficial FB location)

**Treatment**
- Not all FB require removal (deep, small, inert, asymptomatic, away from vital structures), removal (significant pain, functional impairment, reactive, contamination, near vital structures, cosmetic concerns): May require wound extension, irrigation, fine tip forceps

**Disposition**
- D/C

**Fingertip Wounds**

**Definition**
- Amputations/laceration/crush to fingertip (skin/volar pulp/distal phalanx/nail/nail bed)

**History**
- Cutting/crushing injury
Physical Findings
- Amputation, nail bed lacerations, subungual hematoma

Evaluation
- Finger plain film (FB, fracture)

Treatment
- Amputation: Distal to DIP joint → wound care/secondary intention (may need to trim back bone/should always be covered by soft tissue)/abx, significant bone/soft tissue loss → emergent hand surgery consult
- Subungual hematoma: Large → nail trephination, small → no intervention
- Nail bed laceration: 1° repair → remove nail, repair w/ 6-0 absorbable suture, replace nail into nail fold (suture or secure w/ tape) to splint nail bed/maintain nail fold (nail growth → 70–160 d)

Disposition
- D/C

ABUSE

Approach

History
- Delays in seeking care, hx inconsistent w/ injury, multiple past injuries, injuries in various stages of healing

Team Approach
- Social work, child protective services, trained sexual assault nurses, pt advocate

Documentation
- Record factual events/injuries, avoid judgments, informed consent for forensic collection/release of information, mandatory reporting for child/elder abuse

Child Abuse

History
- Story inconsistent w/ injuries/child’s developmental age, inconsistent stories by caretakers

Physical Findings
- Child neglect: Flattening/alopca of occiput (supine for long periods of time), decreased SC tissue/prominent ribs/loose skin over buttocks (FTT)
- Child abuse: Bruises/fracture varying stages, bruises in areas not prone to trauma (lower back, buttock, thighs, cheeks, ear pinna), geometric-shaped bruising (belts, cords), scald burns w/o splash marks or in “dip” pattern, multiple deep contact burns, unexplained extremity swelling (long bone spiral fracture, metaphyseal chip fractures, femur fractures in <3 y), posterior rib fractures, MS Δ (shaken baby), suspicious oral/facial trauma (torn frenulum, dental trauma present in 50% of abuse)
- Child sexual assault: Penile/vaginal d/c (STDs), UTI, genital/rectal trauma (inner thigh bruising, rectal tears, loss rectal tone), often no physical findings if delay in presentation

Evaluation
- Skeletal series (children <5), head CT (suspected intracranial injury), dilated eye exam (retinal detachment/hemorrhage → shaken baby), CBC, Coags, LFTs, tox screen, growth measurements, vaginal/rectal/oral swabs

Treatment
- Social work/child protective services, treat injuries as appropriate

Disposition
- D/C per child protective services

Pearls
- 2–3% children (physical abuse associated low SES)
- ↑ risk in children w/ mental/physical disabilities/chronic medical problems
- Consider Munchausen syndrome by proxy in cases w/ extensive/neg prior w/u
- Most important to suspect abuse & allow trained specialists to opine if abuse occurred

Sexual Assault

History
- Time, date, number/description of assailants, threats made, weapons used, type of assault, drugs used, LOC, post assault activity (Δ of clothing, urination, showering, tampon use), last time of voluntary
intercourse

**Physical Findings**
- Document: Appearance of clothes, scratches, bruising, lacerations (can use toluidine dye to identify vaginal lacerations), d/c

**Evaluation**
- Imaging as needed, have pt advocate present, pregnancy test, ± STD testing, full rape kit if <72 h (modify as appropriate), vaginal/rectal secretions for acid phosphatase/glycoprotein p30, tox screen
- Many areas will have SANE services available & pt may need transfer for SANE exam, must medically clear pt 1st

**Treatment**
- Pregnancy prophylaxis (levonorgestrel 0.75 q12h × 2 doses), STD prophylaxis (gonorrhea: Ceftriaxone 125 mg IM × 1, Chlamydia: Azithromycin 1 g PO × 1, Hep B: 1st of 2 vaccines, HIV), antiemetics

**Disposition**
- D/C w/ f/u counseling

**Pearl**
- 1/5 women is sexually assaulted in lifetime, only 7% reported

**Intimate-partner Violence**

**History**
- Story inconsistent w/ injuries, frequent ED visits, vague medical complaints, chronic pain (>abd pain), overbearing/controlling partner, injury during pregnancy

**Physical Findings**
- Injuries face/head/neck/areas covered by clothes (most common)

**Evaluation**
- Imaging as needed

**Treatment**
- Photographs as appropriate, determine safety of home/immediate risk (escalating violence, treats, firearms), devise safety plan (avoid sedative/arguments in small rooms/access to firearms, teach children to call 911), social work consult

**Disposition**
- D/C to shelter if unsafe home
Pearls

- ↑ risk during pregnancy/attempts to leave partner
- Universal screening for all pts should be done in the ED

Elder Abuse

History

- Delayed presentation, hx of med noncompliance/missed appointments, often lives w/ abuser, have dementia, are dependent on abuser for ADLs, RFs for abusive caretaker: Mental illness, substance abuse, hx of family violence/financial stress/stress of being caretaker

Physical Findings

- Poor hygiene, malnutrition, decubitus ulcers, “urine rash,” unexplained injuries to face/head/torso/back/buttocks/limb contractures (restraints)/bilateral upper extremities (grabbing)

Evaluation

- Imaging as needed, CBC, BMP, CK (rhabdomyolysis)

Treatment

- Photographs as appropriate, arrange for support services to relieve stress on caretaker (VNA, meals-on-wheels), arrange for home safety eval

Disposition

- Admit if unsafe to go home

Pearls

- May be as high as 5–10% in elderly
- Decreased reporting for fear of institutionalization
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A/P</td>
<td>assessment and plan</td>
</tr>
<tr>
<td>a/w</td>
<td>Associated with</td>
</tr>
<tr>
<td>AAA</td>
<td>Abdominal aortic aneurysm</td>
</tr>
<tr>
<td>βB</td>
<td>Beta-blocker</td>
</tr>
<tr>
<td>ABC</td>
<td>Airway, breathing, circulation</td>
</tr>
<tr>
<td>ABG</td>
<td>Arterial blood gas</td>
</tr>
<tr>
<td>ABI</td>
<td>Ankle-brachial index</td>
</tr>
<tr>
<td>abnl</td>
<td>Abnormal</td>
</tr>
<tr>
<td>abnlty</td>
<td>Abnormality</td>
</tr>
<tr>
<td>abx</td>
<td>Antibiotics</td>
</tr>
<tr>
<td>AC</td>
<td>Acromioclavicular</td>
</tr>
<tr>
<td>ACE-I/ACEI</td>
<td>Angiotensin-converting enzyme inhibitor</td>
</tr>
<tr>
<td>ACS</td>
<td>Acute coronary syndrome</td>
</tr>
<tr>
<td>ADH</td>
<td>Antidiuretic hormone</td>
</tr>
<tr>
<td>ADHD</td>
<td>Attention-deficit hyperactivity disorder</td>
</tr>
<tr>
<td>AED</td>
<td>Antiepileptic drug</td>
</tr>
<tr>
<td>AF</td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>AFL</td>
<td>Atrial flutter</td>
</tr>
<tr>
<td>AG</td>
<td>Aminoglycoside</td>
</tr>
<tr>
<td>AGE</td>
<td>Arterial gas embolism</td>
</tr>
<tr>
<td>AI</td>
<td>Aortic insufficiency</td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
</tr>
<tr>
<td>AIVR</td>
<td>Accelerated idioventricular rhythm</td>
</tr>
<tr>
<td>AKD</td>
<td>Acute kidney disease</td>
</tr>
<tr>
<td>AKI</td>
<td>acute kidney injury</td>
</tr>
<tr>
<td>ALI</td>
<td>Acute lung injury</td>
</tr>
<tr>
<td>ALT</td>
<td>Alanine transaminase</td>
</tr>
<tr>
<td>ALTE</td>
<td>Apparent life-threatening event</td>
</tr>
</tbody>
</table>
AMS  Acute mountain sickness
ANC  Absolute neutrophil count
APAP Acetaminophen
ARB  Angiotensin receptor blocker
ARDS Acute respiratory distress syndrome
ARF  Acute renal failure
AS   Aortic stenosis
ASA  Acetylsalicylic acid (aspirin)
ASD  Atrial septal defect
AST  Aspartate aminotransferase
AT   Atrial tachycardia
ATN  Acute tubular necrosis
AVA  Aortic valve area
AVM  Arteriovenous malformation
AVN  Atrioventricular node
AVNRT AV node re-entrant tachycardia
AVR  Aortic valve replacement
AVRT AV reciprocating tachycardia
b/c  Because
b/w  Between
BAL  Blood alcohol level
BBB  Bundle branch block
BiPAP Bilevel positive airway pressure
BMP  Basic metabolic panel
BMS  Bare metal stent
BNP  Brain natriuretic peptide
BP   Blood pressure
BPH  Benign prostatic hyperplasia
BPPV Benign paroxysmal positional vertigo
BSUS Bedside ultrasound
BVM  Bag-valve mask
BZD  Benzodiazepines
C/o  Complaint of
C/w  Compared with
CABG Coronary artery bypass graft
CAD  Coronary artery disease
CAP  Community-acquired pneumonia
CBC  Complete blood count
CBD  Common bile duct
CCB  Calcium channel blocker
CCU  Coronary care unit
CHF  Congestive heart failure
CKD  Chronic kidney disease
Cl   Contraindication
CMP  Cardiomyopathy
CMV  Cytomegalovirus
CN   Cranial nerve
CNS  Central nervous system
CO   Carbon monoxide, cardiac output
CO₂  Carbon dioxide
COPD Chronic obstructive pulmonary disease
CP   Chest pain
CPAP Continuous positive airway pressure
CPK  Creatine phosphokinase
CPP  Cerebral perfusion pressure
CPR  Cardiopulmonary resuscitation
Cr   Creatinine
CRAO Central retinal artery occlusion
CRP  C-reactive protein
CRVO Central retinal vein occlusion
CSF  Cerebrospinal fluid
CT   Computed tomography
CTA  Computed tomography angiography
CTH  Chronic tension headache
cTn  Cardiac troponin
CTV  Computed tomography venography
CTX  ceftriaxone
CVA  Cerebral vascular accident
cx   Complications
CXR  Chest x-ray
d    Day(s)
D/C     Discharge
DAI    Diffuse axonal injury
DBP    Diastolic blood pressure
DCS    Decompression sickness
DDx    Differential diagnosis
DI     Diabetes insipidus
DIC    Disseminated intravascular coagulation
DIP    Distal interphalangeal joint
DKA    Diabetic ketoacidosis
DL     Direct laryngoscopy
DMS    Altered mental status
DOAC   Direct oral anticoagulant
DOE    Dyspnea on exertion
DP     Dorsalis pedis
DPA    Diagnostic peritoneal aspiration
DPL    Diagnostic peritoneal lavage
DTR    Deep tendon reflex
DUB    Dysfunctional uterine bleeding
DVT    Deep vein thrombosis
Dx     Diagnosis
Dysfxn Dysfunction
dz     Disease
e/o    Evidence of
EBV    Epstein–Barr virus
ED     Emergency Department
EDH    Epidural hemorrhage
EEG    Electroencephalogram
EF     Ejection fraction
EGD    Extraglottic device
EKG    Electrocardiogram
EMS    Emergency medical services
EOM    Extraocular muscles
EP     Electrophysiologist
ERCP   Endoscopic retrograde cholangiopancreatography
ESBL   Extended spectrum beta-lactamase
esp    Especially
ESR  Erythrocyte sedimentation rate
EtOH  Ethanol
ETT  Endotracheal tube
eval  Evaluation
f/b  followed by
f/u  Follow-up
FAST  Focused assessment with sonography for trauma
FB  Foreign body
FFP  Fresh frozen plasma
FH  Family history
FHH  Familial hypocalciuric hypercalcemia
FSG  Fingerstick glucose
FTT  Failure to thrive
FUO  Fever of unknown origin
G6PD  Glucose-6-phosphate dehydrogenase
GC  Gonococcal
GCS  Glasgow coma scale
GERD  Gastroesophageal reflux
GI  Gastrointestinal
GIB  Gastrointestinal bleeding
GNR  gram-negative rods
GP  glycoprotein
GSW  Gunshot wound
GVHD  Graft versus host disease
h  Hour(s)
H&P  history and physical
HA  Headache
HACE  High altitude cerebral edema
HAPE  High altitude pulmonary edema
HCAP  Health-care associated pneumonia
Hct  Hematocrit
HD  Hemodialysis, hemodynamically
HEENT  head, eyes, ears, nose, and throat
HELLP  Hemolysis, Elevated Liver enzymes, and Low Platelets
HHS  Hyperosmolar hyperglycemic state
HI  Homicidal ideation
HIDA  Hydroxy iminodiacetic acid
HIT  Heparin-induced thrombocytopenia
HIV  human immunodeficiency virus
HK  Hypokinesia
HL  Hyperlipidemia
HLD  Hyperlipidemia
HOB  Head of bed
HR  Heart rate
HRUS  High-resolution ultrasound
HSCT  hematopoietic stem cell transplantation
HSP  Henoch–Schönlein purpura
HSV  Herpes simplex virus
HTN  Hypertension
HUS  Hemolytic uremic syndrome
hx  History
I&D  Incision and drainage
IABP  Intra-aortic balloon pump
IBD  Inflammatory bowel disease
ICH  Intracranial hemorrhage
ICP  Intracranial pressure
ICU  intensive care unit
IFA  Immunofluorescence antibody
ILD  Interstitial lung disease
inpt  inpatient
INR  International normalized ratio
IO  Intraosseous
IOP  Intraocular pressure
IPH  Intraparenchymal hemorrhage
IR  Interventional radiology
ITP  Immune thrombocytopenic purpura
IVDA  Intravenous drug abuse
IVDU  Intravenous drug use
IVIG  Intravenous immunoglobulin
IVP  Intravenous pyelogram
JVD  Jugular venous distension
JVP  Jugular venous pressure
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>KUB</td>
<td>Kidney ureter bladder</td>
</tr>
<tr>
<td>L</td>
<td>Left</td>
</tr>
<tr>
<td>LAD</td>
<td>Lymphadenopathy, Left-axis deviation</td>
</tr>
<tr>
<td>LAE</td>
<td>Left atrial enlargement</td>
</tr>
<tr>
<td>LAFB</td>
<td>Left anterior fascicular block</td>
</tr>
<tr>
<td>LBBB</td>
<td>Left bundle branch block</td>
</tr>
<tr>
<td>LBP</td>
<td>Lower back pain</td>
</tr>
<tr>
<td>LDH</td>
<td>Lactate dehydrogenase</td>
</tr>
<tr>
<td>LE</td>
<td>Lower extremity</td>
</tr>
<tr>
<td>LFT</td>
<td>Liver function test</td>
</tr>
<tr>
<td>LGIB</td>
<td>Lower gastrointestinal bleeding</td>
</tr>
<tr>
<td>LLL</td>
<td>Left lower lobe</td>
</tr>
<tr>
<td>LLQ</td>
<td>Left lower quadrant</td>
</tr>
<tr>
<td>LMN</td>
<td>Lower motor neuron</td>
</tr>
<tr>
<td>LMP</td>
<td>Last menstrual period</td>
</tr>
<tr>
<td>LOC</td>
<td>Loss of consciousness</td>
</tr>
<tr>
<td>LP</td>
<td>Lumbar puncture</td>
</tr>
<tr>
<td>LPFB</td>
<td>Left posterior fascicular block</td>
</tr>
<tr>
<td>LV</td>
<td>Left ventricle</td>
</tr>
<tr>
<td>LVAD</td>
<td>Left ventricular assist device</td>
</tr>
<tr>
<td>LVH</td>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td>MAHA</td>
<td>Microangiopathic hemolytic anemia</td>
</tr>
<tr>
<td>MAP</td>
<td>Mean arterial pressure</td>
</tr>
<tr>
<td>MAT</td>
<td>Multifocal atrial tachycardia</td>
</tr>
<tr>
<td>MCP</td>
<td>Metacarpophalangeal</td>
</tr>
<tr>
<td>MDR</td>
<td>Multiple drug resistance</td>
</tr>
<tr>
<td>MDRO</td>
<td>multi-drug resistant organism</td>
</tr>
<tr>
<td>MDS</td>
<td>myelodysplastic syndrome</td>
</tr>
<tr>
<td>MH</td>
<td>Malignant hyperthermia</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>min</td>
<td>Minute(s)</td>
</tr>
<tr>
<td>MM</td>
<td>Mucous membrane</td>
</tr>
<tr>
<td>mo</td>
<td>Month(s)</td>
</tr>
<tr>
<td>mod</td>
<td>Moderate</td>
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<tr>
<td>MR</td>
<td>Mitral regurgitation</td>
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<td>MRA</td>
<td>Magnetic resonance angiography</td>
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<tr>
<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>MRCP</td>
<td>mental retardation/cerebral palsy</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>MRSA</td>
<td>Methicillin-resistant <em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>MRV</td>
<td>Magnetic resonance venography</td>
</tr>
<tr>
<td>MS</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>MSK</td>
<td>musculoskeletal</td>
</tr>
<tr>
<td>MV</td>
<td>Mitral valve</td>
</tr>
<tr>
<td>MVC</td>
<td>Motor vehicle collision</td>
</tr>
<tr>
<td>MVP</td>
<td>Mitral valve prolapsed</td>
</tr>
<tr>
<td>N/V</td>
<td>Nausea/vomiting</td>
</tr>
<tr>
<td>NAPQI</td>
<td>N-acetyl-p-benzoquinone imine</td>
</tr>
<tr>
<td>NCHCT</td>
<td>non-contrast head computed tomography</td>
</tr>
<tr>
<td>NEC</td>
<td>Necrotizing enterocolitis</td>
</tr>
<tr>
<td>neg</td>
<td>Negative</td>
</tr>
<tr>
<td>NGT</td>
<td>nasogastric tube</td>
</tr>
<tr>
<td>NHL</td>
<td>non-Hodgkin’s lymphoma</td>
</tr>
<tr>
<td>NIF</td>
<td>Negative inspiratory force</td>
</tr>
<tr>
<td>NIPPV</td>
<td>non-invasive positive-pressure ventilation</td>
</tr>
<tr>
<td>NIV</td>
<td>Noninvasive ventilation</td>
</tr>
<tr>
<td>nl</td>
<td>Normal</td>
</tr>
<tr>
<td>NMS</td>
<td>Neuroleptic malignant syndrome</td>
</tr>
<tr>
<td>NPJT</td>
<td>Nonparoxysmal junctional tachycardia</td>
</tr>
<tr>
<td>NPO</td>
<td>Nil per OS</td>
</tr>
<tr>
<td>NPV</td>
<td>Negative predictive value</td>
</tr>
<tr>
<td>NRB</td>
<td>Nonrebreather</td>
</tr>
<tr>
<td>NSAID</td>
<td>Nonsteroidal anti-inflammatory drug</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>Non-ST-elevation MI</td>
</tr>
<tr>
<td>NSTV</td>
<td>Nonsustained ventricular tachycardia</td>
</tr>
<tr>
<td>NTG</td>
<td>Nitroglycerin</td>
</tr>
<tr>
<td>o/w</td>
<td>Otherwise</td>
</tr>
<tr>
<td>O₂</td>
<td>Oxygen</td>
</tr>
<tr>
<td>OA</td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td>OCP</td>
<td>Oral contraceptive pill</td>
</tr>
<tr>
<td>OE</td>
<td>Otitis externa</td>
</tr>
<tr>
<td>OM</td>
<td>Otitis media</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>OMFS</td>
<td>Oral and maxillofacial surgery</td>
</tr>
<tr>
<td>OR</td>
<td>Operating room</td>
</tr>
<tr>
<td>ORIF</td>
<td>Open reduction and internal fixation</td>
</tr>
<tr>
<td>OSA</td>
<td>Obstructive sleep apnea</td>
</tr>
<tr>
<td>OTC</td>
<td>Over-the-counter</td>
</tr>
<tr>
<td>otw</td>
<td>otherwise</td>
</tr>
<tr>
<td>outpt(s)</td>
<td>Outpatient(s)</td>
</tr>
<tr>
<td>p/w</td>
<td>Presents with</td>
</tr>
<tr>
<td>PAN</td>
<td>Polyarteritis nodosa</td>
</tr>
<tr>
<td>PCI</td>
<td>Percutaneous coronary intervention</td>
</tr>
<tr>
<td>PCKD</td>
<td>Polycystic kidney disease</td>
</tr>
<tr>
<td>PCN</td>
<td>Penicillin</td>
</tr>
<tr>
<td>PCOS</td>
<td>Polycystic ovary syndrome</td>
</tr>
<tr>
<td>PCP</td>
<td>Pneumocystis jirovecii pneumonia, primary care physician</td>
</tr>
<tr>
<td>PCR</td>
<td>Polymerase chain reaction</td>
</tr>
<tr>
<td>PDA</td>
<td>Patent ductus arteriosus</td>
</tr>
<tr>
<td>PE</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>PEEP</td>
<td>Positive end-expiratory pressure</td>
</tr>
<tr>
<td>PEFR</td>
<td>Peak expiratory flow rate</td>
</tr>
<tr>
<td>PGE$_1$</td>
<td>Prostaglandin E$_1$</td>
</tr>
<tr>
<td>PHT</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>PID</td>
<td>Pelvic inflammatory disease</td>
</tr>
<tr>
<td>PIP</td>
<td>Proximal interphalangeal joint</td>
</tr>
<tr>
<td>pl</td>
<td>Pleural</td>
</tr>
<tr>
<td>PLT</td>
<td>Platelet</td>
</tr>
<tr>
<td>PM</td>
<td>Pacemaker</td>
</tr>
<tr>
<td>PMH</td>
<td>Past medical history</td>
</tr>
<tr>
<td>PMR</td>
<td>Polymyalgia rheumatica</td>
</tr>
<tr>
<td>PMV</td>
<td>Percutaneous mitral valvuloplasty</td>
</tr>
<tr>
<td>PNA</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>PND</td>
<td>Paroxysmal nocturnal dyspnea</td>
</tr>
<tr>
<td>PostTP</td>
<td>Posttest probability</td>
</tr>
<tr>
<td>PPM</td>
<td>Permanent pacemaker</td>
</tr>
<tr>
<td>PPV</td>
<td>Positive predictive value, positive pressure ventilation</td>
</tr>
<tr>
<td>PQRST</td>
<td>Palliation, quality, radiation, severity, timing</td>
</tr>
</tbody>
</table>
PRBC  Packed red blood cell
PreTP  Pretest probability
PRWP  Poor R-wave progression
PS  Pulmonary stenosis
PT  Prothrombin time, Posterior tibialis
pt(s)  Patient(s)
PTX  Pneumothorax
PUD  Peptic ulcer disease
PVD  Peripheral vascular disease
Qw  Q wave
R  Right
r/o  Rule out
RAD  Right axis deviation
RAE  Right atrial enlargement
RBBB  Right bundle branch block
RF  Risk factor
RLQ  Right lower quadrant
RMSF  Rocky Mountain spotted fever
ROM  Range of motion
ROS  Review of systems
ROSC  Return of spontaneous circulation
RPR  rapid plasma reagin
RR  Respiratory rate
RSI  Rapid sequence intubation
RSV  Respiratory syncytial virus
RTA  Renal tubular acidosis
RUG  Retrograde urethrogram
RUQ  Right upper quadrant
RV  Right ventricle
RVH  Right ventricular hypertrophy
RVOT  Right ventricular outflow tract
rxn  Reaction
SAH  Subarachnoid hemorrhage
SBE  Subacute bacterial endocarditis
SBO  Small bowel obstruction
SBP  Systolic blood pressure
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD</td>
<td>Sickle cell disease</td>
</tr>
<tr>
<td>SCFE</td>
<td>Slipped capital femoral epiphysis</td>
</tr>
<tr>
<td>SCM</td>
<td>Sternocleidomastoid</td>
</tr>
<tr>
<td>SDH</td>
<td>Subdural hemorrhage</td>
</tr>
<tr>
<td>sec</td>
<td>Second(s)</td>
</tr>
<tr>
<td>sens</td>
<td>Sensitive, Sensitivity</td>
</tr>
<tr>
<td>SFV</td>
<td>Superficial femoral vein</td>
</tr>
<tr>
<td>SI</td>
<td>Suicidal ideation</td>
</tr>
<tr>
<td>SIRS</td>
<td>Systemic inflammatory response syndrome</td>
</tr>
<tr>
<td>SJS</td>
<td>Stevens–Johnson syndrome</td>
</tr>
<tr>
<td>SLE</td>
<td>systemic lupus erythematosus</td>
</tr>
<tr>
<td>SMA</td>
<td>Superior mesenteric artery</td>
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<tr>
<td>SOB</td>
<td>Shortness of breath</td>
</tr>
<tr>
<td>spec</td>
<td>Specific, Specificity</td>
</tr>
<tr>
<td>SS</td>
<td>Serotonin syndrome</td>
</tr>
<tr>
<td>SSRI</td>
<td>Selective serotonin reuptake inhibitor</td>
</tr>
<tr>
<td>SSSS</td>
<td>Staphylococcal scalded skin</td>
</tr>
<tr>
<td>ST</td>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td>ST-T</td>
<td>ST-segment-T wave</td>
</tr>
<tr>
<td>STD</td>
<td>Sexually transmitted disease</td>
</tr>
<tr>
<td>STEMI</td>
<td>ST-segment elevation MI</td>
</tr>
<tr>
<td>SVC</td>
<td>superior vena cava</td>
</tr>
<tr>
<td>SVT</td>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td>sxs</td>
<td>Symptoms</td>
</tr>
<tr>
<td>sz</td>
<td>Seizure</td>
</tr>
<tr>
<td>T+S</td>
<td>type and screen</td>
</tr>
<tr>
<td>TAH</td>
<td>total abdominal hysterectomy</td>
</tr>
<tr>
<td>TB</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>TBI</td>
<td>Traumatic brain injury</td>
</tr>
<tr>
<td>TBW</td>
<td>Total body weight</td>
</tr>
<tr>
<td>TCA</td>
<td>Tricyclic antidepressants</td>
</tr>
<tr>
<td>TCP</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>TEE</td>
<td>Transesophageal echocardiogram</td>
</tr>
<tr>
<td>Tele</td>
<td>Telemetry</td>
</tr>
<tr>
<td>TEN</td>
<td>Toxic epidermal necrolysis</td>
</tr>
<tr>
<td>TIA</td>
<td>Transient ischemic attack</td>
</tr>
</tbody>
</table>
TM  Tympanic membrane
TMJ  Temporomandibular joint
TOA  Tubo-ovarian abscess
tox  toxicity
TRACO  transfusion-associated circulatory overload
TRALI  transfusion-associated lung injury
TSH  thyroid stimulating hormone
TSS  Toxic shock syndrome
TTE  Transthoracic echocardiogram
TTP  Thrombotic thrombocytopenic purpura
TWI  T-wave inversion
tx  Treatment
TXA  tranexamic acid
u/s  Ultrasound
UA  Urine analysis, Unstable angina
UAG  Urinary anion gap
Ucx  Urine culture
UGIB  Upper gastrointestinal bleeding
UH  unfractionated heparin
UMN  Upper motor neuron
UOP  Urinary output
UPJ  Urinary pelvic junction
URI  Upper respiratory infection
UTI  Urinary tract infection
V/D  Vomiting/diarrhea
VBG  Venous blood gas
VCUG  voiding cystourethrogram
VL  Video laryngoscopy
VN  Visiting Nurse (Association)
VRE  vancomycin-resistant enterococcus
VS  Vital signs
VSD  Ventricular septal defect
VT  Tidal volume
vWD  von Willebrand disease
vWF  von Willebrand factor
VZV  Varicella zoster virus
<table>
<thead>
<tr>
<th>Medication</th>
<th>Route</th>
<th>nl Dose</th>
<th>Incremental Dose</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Rapid IV push</td>
<td>0.1 mg/kg (up to 6 mg)</td>
<td>0.2 mg/kg</td>
<td>12 mg</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Rapid IV/IO</td>
<td>5 mg/kg</td>
<td></td>
<td>15 mg/kg/d</td>
</tr>
<tr>
<td>Atropine</td>
<td>IV/IO/ET</td>
<td>0.02 mg/kg</td>
<td>0.04 mg/kg</td>
<td>0.5 mg single dose</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>IV/IO</td>
<td>20 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>IV/IO</td>
<td>2–20 μg/kg/min</td>
<td></td>
<td>Titrate to effect</td>
</tr>
<tr>
<td>Epinephrine PEA, bradycardia</td>
<td>IV/IO: 0.01 mg/kg (1:10000) ET: 0.1 mg/kg (1:1000)</td>
<td>q3–5min during CPR</td>
<td>0.1 mL/kg</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>IV/IO</td>
<td>0.5–1 g/kg</td>
<td></td>
<td>2–4 mL/kg 25%</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>IV/IO/ET</td>
<td>1 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mg sulfate</td>
<td>IV/IO</td>
<td>25–50 μg/kg</td>
<td></td>
<td>2 g</td>
</tr>
<tr>
<td>Naloxone</td>
<td>If &lt;5 yr or &lt;20 mg: 0.1 mg/kg If &gt;5 yr or &gt;20 kg: 2 mg</td>
<td></td>
<td></td>
<td>Titrate to effect</td>
</tr>
</tbody>
</table>
Cardioversion in PALS

<table>
<thead>
<tr>
<th>Reason</th>
<th>nl Dose</th>
<th>Incremental Dose</th>
</tr>
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<tbody>
<tr>
<td>Tachycardia</td>
<td>0.5–1 J/kg</td>
<td>2 J/kg if ineffective</td>
</tr>
<tr>
<td>VF/pulseless VT</td>
<td>2–4 J/kg</td>
<td>4 J/kg if ineffective w/i 30–60 sec after med</td>
</tr>
</tbody>
</table>

MECHANICAL VENTILATION

*(NEJM 2001;344:1986)*

**Approach**
- Choose invasive ventilation vs. NIV → choose type of NIV or invasive mode → adjust settings
- In the ED, NIV is used to avoid intubation, esp in acute COPD/RAD or CHF/pulm edema

**Indications for Ventilation**

<table>
<thead>
<tr>
<th>Type of Ventilation</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noninvasive</td>
<td>CPAP: Hypoxemia (ie, CHF exacerbation)</td>
</tr>
<tr>
<td></td>
<td>BiPAP: Hypoventilation (ie, COPD)</td>
</tr>
<tr>
<td>Invasive</td>
<td>Apnea, impending respiratory failure, airway protection, failed NIV</td>
</tr>
</tbody>
</table>

**Noninvasive Ventilation**

<table>
<thead>
<tr>
<th>CPAP</th>
<th>BiPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opens atelectatic alveoli, can improve respiratory mechanics &amp; hemodynamics</td>
<td>CPAP + pressure support → directly reduces work of breathing</td>
</tr>
<tr>
<td>In ACPE → ↓ intubation, mortality</td>
<td>In COPD, PNA → ↓ intubation, mortality</td>
</tr>
</tbody>
</table>

Relative CIs: Aspiration risk, vomiting, UGIB/epistaxis, agitation or lethargy precluding compliance, hemodynamic instability

**Invasive Ventilation**

<table>
<thead>
<tr>
<th>Invasive Ventilation Modes</th>
<th>Description</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assist control</td>
<td>All breaths fully assisted by vent</td>
<td>Most useful in apneic pts (eg, chemically paralyzed)</td>
</tr>
<tr>
<td><strong>Invasive Ventilation Modes</strong></td>
<td><strong>Description</strong></td>
<td><strong>Comments</strong></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation</td>
<td>Set # of supported breaths, synchronized to pt’s effort; all other pt-initiated breaths determined by pt</td>
<td>Useful for weaning pts from ventilator</td>
</tr>
<tr>
<td>Volume targeted</td>
<td>Set TV for assisted breaths</td>
<td>Standard setting</td>
</tr>
<tr>
<td>Pressure targeted</td>
<td>Set inspiratory pressure for assisted breaths</td>
<td>Useful for pts at risk for barotrauma</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Other Ventilator Settings</strong></th>
<th><strong>Standard initial settings</strong></th>
<th><strong>Other modes</strong></th>
<th><strong>PEEP</strong></th>
<th><strong>Auto-PEEP</strong></th>
<th><strong>Inspiratory flow rate</strong></th>
<th><strong>Pplat</strong></th>
<th><strong>PIP</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Assist control ventilation, TV 4–8 mL/kg, RR 12–14, FiO2 100%, PEEP 5 cm H2O, Wean FiO2 as quickly as tolerated</td>
<td>See above</td>
<td>Positive pressure present during exhalation → maintains patent alveoli → ↓ shunting &amp; ↑ oxygenation (cardiac effects dictate CO &amp; oxygenation); 5 cm H2O = “physiologic” PEEP</td>
<td>Presence of flow at end expiration due to “breath stacking”: ↓ time for exhalation → incomplete expiration → lungs “trap” air → leading to potential compromise of respiratory mechanics &amp; hemodynamics (↓ preload)</td>
<td>↓ Flow rate → ↓ inspiratory time → ↑ expiratory time (ie, ↓ I:E ratio) → improves ventilation &amp; minimizes auto-PEEP in obstructive dz (asthma, COPD)</td>
<td>Plateau pressure, measured at end expiration; determined by respiratory system compliance</td>
<td>Peak pressure measured during inspiration affected by airway resistance plus lung/chest wall compliance</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Preload: Decreased via ↑ intrathoracic pressure → ↓ venous return</td>
<td>Afterload: Decreased via ↓ transmural cardiac pressure</td>
<td></td>
<td>↑ Pplat w/ obesity, pulmonary edema, ARDS → auto-PEEP, asynchronous breathing</td>
<td>If ↑ PIP &amp; nl Pplat → cause = airway resistance (bronchospasm, secretions, etc.)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Making Changes to the Ventilator</strong></th>
<th><strong>Improve oxygenation</strong></th>
<th><strong>↑ PEEP, ↑ FiO2</strong></th>
</tr>
</thead>
</table>
Improve ventilation | ↑ TV, ↑ RR
Reduce auto-PEEP | ↓ RR, ↑ expiratory time, ↑ insp flow rate
Permissive hypercapnia | Low TV (4–6 mL/kg) to reduce baro/volutrauma in ALI/ARDS

**ANALGESIA & CONSCIOUS SEDATION**

### Opioids

<table>
<thead>
<tr>
<th>Medication</th>
<th>nl Dose</th>
<th>Incremental Dose</th>
<th>Onset of Action</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>0.1 mg/kg</td>
<td>½ nl dose</td>
<td>5–10 min</td>
<td>3–4 h</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.5–2 mg</td>
<td>½ nl dose</td>
<td>3–5 min</td>
<td>2–4 h</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5–1 μg/kg (25–100 μg)</td>
<td>25 μg</td>
<td>1–2 min</td>
<td>10 min–1 h</td>
</tr>
</tbody>
</table>

### Benzodiazepines

<table>
<thead>
<tr>
<th>Medication</th>
<th>nl Dose</th>
<th>Incremental Dose</th>
<th>Onset of Action</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>5–10 mg</td>
<td>2.5 mg</td>
<td>1–5 min</td>
<td>30 min–2 h</td>
</tr>
<tr>
<td>Midazolam</td>
<td>1–5 mg</td>
<td>0.5–1 mg</td>
<td>1–2 min</td>
<td>15–60 min</td>
</tr>
</tbody>
</table>

### Conscious Sedation Medications

<table>
<thead>
<tr>
<th>Medication</th>
<th>nl Dose</th>
<th>Incremental Dose</th>
<th>Onset of Action</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketamine*</td>
<td>1–2 mg/kg IV or 2–4 mg/kg IM</td>
<td>1 mg/kg</td>
<td>1–2 min</td>
<td>10–30 min</td>
</tr>
<tr>
<td>Chloral hydrate**</td>
<td>50–75 mg/kg prn</td>
<td>25–75 mg/kg</td>
<td>20–30 min</td>
<td>2–6 h</td>
</tr>
<tr>
<td>Propofol</td>
<td>1–3 mg/kg</td>
<td>0.5–5 mg/kg</td>
<td>&lt;1 min</td>
<td>8–10 min</td>
</tr>
<tr>
<td>Etomidate</td>
<td>0.2–0.5 mg/kg</td>
<td>0.05 mg/kg</td>
<td>&lt;1 min</td>
<td>5–8 min</td>
</tr>
<tr>
<td>Nitrous oxide</td>
<td>30–50%</td>
<td>Constant</td>
<td>1–2 min</td>
<td>5 min</td>
</tr>
</tbody>
</table>

*Consider administration w/ glycopyrrolate (0.01 mg/kg) or atropine (0.01 mg/kg) as an antisialogogue
**Pediatric only, rarely used

### Reversal Agents
### ICU MEDICATIONS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
<th>Incremental Dose</th>
<th>Onset of Action</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naloxone (opioids)</td>
<td>0.4–2 mg</td>
<td>0.04 mg</td>
<td>1–2 min</td>
<td>30 min–1 h</td>
</tr>
<tr>
<td>Flumazenil (benzo)</td>
<td>1 mg</td>
<td>0.2 mg</td>
<td>1–2 min</td>
<td>30 min–1.5 h</td>
</tr>
</tbody>
</table>

### ICU Medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pressors, Inotropes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>$\alpha_1$</td>
<td>50–200 μg/min</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>$\alpha_1 &gt; \beta_1$</td>
<td>0.05–0.5 μg/kg/min, max ~5 μg/kg/min</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>$V_1$</td>
<td>0.04 U/min</td>
</tr>
<tr>
<td>Dopamine</td>
<td>$D$, $\beta$, $\alpha$, $\beta$, $D$</td>
<td>0.5–2 μg/kg/min, 2–10 μg/kg/min, &gt;10 μg/kg/min, max 50 μg/kg/min</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>$\beta_1 &gt; \beta_2$</td>
<td>2–20 μg/kg/min</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>$\alpha_1$, $\alpha_2$, $\beta_1$, $\beta_2$</td>
<td>0.05–0.5 μg/kg/min, max ~5 μg/kg/min</td>
</tr>
<tr>
<td><strong>Vasodilators</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>NO</td>
<td>5–1000 μg/min</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>NO</td>
<td>0.1–10 μg/kg/min</td>
</tr>
<tr>
<td>Enalaprilat</td>
<td>ACEI</td>
<td>0.625–2.5 mg over 5 min then 0.625–5 mg q6h</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Vasodilator</td>
<td>5–20 mg q20–30 min</td>
</tr>
<tr>
<td>Labetalol</td>
<td>$\alpha_1$, $\beta_1$, $\beta_2$ blockers</td>
<td>20 mg over 2 min then 20–80 mg q10min or 10–120 mg/h</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>NO</td>
<td>2.5–15 mg/h</td>
</tr>
<tr>
<td><strong>Antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Class III</td>
<td>150 mg over 10 min, then 1 mg/min × 6 h, then 0.5 mg/min × 18 h</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Class IB (Na channel)</td>
<td>1–1.5 mg/kg (100 mg) then 1–4 mg/min</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Class IA (Na channel)</td>
<td>17 mg/kg (1 g) over 60 min, then 1–4 mg/min</td>
</tr>
<tr>
<td>Drug</td>
<td>Class</td>
<td>Dose</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
<td>-------------------------------------------</td>
</tr>
<tr>
<td>Ibutilide</td>
<td>Class III (K channel)</td>
<td>1 mg over 10 min, may repeat × 1</td>
</tr>
<tr>
<td>Propranolol</td>
<td>βB</td>
<td>0.5–1 mg q5min then 1–10 mg/h</td>
</tr>
<tr>
<td>Esmolol</td>
<td>β₁ blocker &gt; β₂ blocker</td>
<td>500 μg/kg (20–40 mg) over 1 min, then 25–300 μg/kg/min (2–20 mg/min)</td>
</tr>
<tr>
<td>Verapamil</td>
<td>CCB</td>
<td>2.5–5 mg over 1–2 min repeat 5–10 mg in 15–30 min prn, 5–20 mg/h</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>CCB</td>
<td>0.25 mg/kg (20 mg) over 2 min, reload 0.35 mg/kg (25 mg) × 1 prn, then 5–15 mg/h</td>
</tr>
</tbody>
</table>

**Drug**       | **Class** | **Dose**                                      |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Purinergic</td>
<td>6 mg rapid push, if no response 12 mg rapid push, repeat × 1 prn</td>
</tr>
</tbody>
</table>

**Sedation**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>Opioid</td>
<td>1–unlimited mg/h</td>
</tr>
<tr>
<td>Etomidate</td>
<td>Anesthetic</td>
<td>0.2–0.5 mg (100–300 mg)</td>
</tr>
<tr>
<td>Propofol</td>
<td>Anesthetic</td>
<td>1–3 mg/kg (50–200 mg) then 0.3–5 mg/kg/h (20–400 mg/h)</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Benzo</td>
<td>1–5 mg q1–2h then q6 prn</td>
</tr>
<tr>
<td>Midazolam</td>
<td>Benzo</td>
<td>0.5–2 mg q5min prn or 0.5–4 mg then 1–10 mg/h</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Anesthetic</td>
<td>1–2 mg/kg (60–150 mg)</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>Antipsychotic</td>
<td>2–5 mg q20–30min</td>
</tr>
</tbody>
</table>

**Paralysis**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>Depolarizing</td>
<td>0.6–1.1 mg/kg (70–100 mg)</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>nACh</td>
<td>0.08 mg/kg (2–4 mg) q30–90min</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>nACh</td>
<td>0.6 mg/kg (60–100 mg)</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>nACh</td>
<td>0.08 mg/kg (5–10 mg) over 1–3 min, then 0.5–0.1 mg/kg/h (2–8 mg/h)</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>nACh</td>
<td>5–10 μg/kg/min</td>
</tr>
</tbody>
</table>

**Miscellaneous**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin</td>
<td></td>
<td>0.1 U/kg/h</td>
</tr>
<tr>
<td>Glucagon</td>
<td></td>
<td>5–10 mg then 1–5 mg/h</td>
</tr>
<tr>
<td>Octreotide</td>
<td>Somatostatin analog</td>
<td>50 μg then 50 μg/h</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Antiepileptic</td>
<td>20 mg/kg (1–1.5 g) over 20–30 min</td>
</tr>
<tr>
<td>Drug</td>
<td>Category</td>
<td>Dosage Description</td>
</tr>
<tr>
<td>------------------</td>
<td>-------------------</td>
<td>---------------------------------------------------------</td>
</tr>
<tr>
<td>Fosphenytoin</td>
<td>Antiepileptic</td>
<td>20 mg/kg (1–1.5 g) over 10 min</td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>Barbiturate</td>
<td>20 mg/kg (1–1.5 g) over 20 min</td>
</tr>
<tr>
<td>Thiopental</td>
<td>Barbiturate</td>
<td>3–5 mg/kg (200–400 mg) over 2 min</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Osmotic</td>
<td>1.5–2 g/kg over 30–60 min, repeat q6–12h to keep oSm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>310–320</td>
</tr>
<tr>
<td>Naloxone</td>
<td>Opioid antagonist</td>
<td>0.4–2 mg q2–3min to total 10 mg</td>
</tr>
<tr>
<td>Flumazenil</td>
<td>Benzo antagonist</td>
<td>0.2 mg over 30 sec then 0.3 mg over 30 sec, may repeat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.5 mg over 30 sec to max 3 mg</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Opioid</td>
<td>50–100 μg then 50–unlimited μg/h</td>
</tr>
</tbody>
</table>

**EQUATIONS**

**Metabolic**

**Anion Gap:** Na – (Cl + Bicarb)

**Delta/Delta** = (actual anion gap – normal gap)/(normal HCO₃ – actual HCO₃)

**Total body water (TBW)** = weight (kg) × 0.6 (use 0.5 if female/elderly, 0.6 for infants)

**Corrected Na** = measured Na + [2.4 × (measured glucose – 100)]

**Calculated osmoles** = (2 × Na) + (glucose/18) + (BUN/2.8) + (EtOH/4.6)

**Osmolal gap** = measured osmoles – calculated osmoles (mL)

**Estimated Cr clearance** = \[
\left(\frac{140 - \text{age (y)}}{\text{wt (kg)}}\times\frac{\text{Serum Cr (mg/dL)}}{72}\times(0.85 \text{ in women})\right)
\]

**Pediatric fluid maintenance (4-2-1 Rule):**

(4 cc/kg for 1st 10 kg) + (2 cc/kg for 2nd 10 kg) + (1 cc/kg for remainder kg)

**Hyponatremia:**

\[
\Delta[\text{Na}] \text{liter infused} = \frac{[\text{Na}]_{\text{infuse}} \times [\text{Na}]_{\text{serum}}}{\text{TBW} + 1}
\]

\[
\text{Rate of infusion (mL/h)} = \frac{1000 \times [\text{TBW} \times (\text{desired Na} - \text{serum Na})]}{[\text{Na (mmol/L)}]\text{infuse} \times \text{time (h)}}
\]

**Hyponatremia:**
Free water deficit = total body water \times \left[\frac{(140/\text{serum Na}) - 1}{\text{TBW} + 1}\right]

\[
\Delta [\text{Na}] \text{liter infusate} = \frac{([\text{Na}]_{\text{infusate}} + [K]_{\text{infusate}}) - [\text{Na}]_{\text{serum}}}{\text{TBW}}
\]

Total infusion (L) = \frac{\text{Desired [Na (mEq/L)]} - \text{Serum [Na (mEq/L)]}}{\Delta [\text{Na}] \text{liter infusate}}

Rate of infusion (mL/h) = \frac{\text{total infusion (mL)}}{24 \text{ h}}

**CARDIOPULMONARY**

A-a gradient = PAO\(_2\) – PaO\(_2\) (nl ≈ 4 + (age/4))

Stroke volume = cardiac output/heart rate

Mean arterial pressure = \frac{[\text{SBP} + (\text{DBP} \times 2)]}{3} (nl 70–100 mmHg)

**PROCEDURES**

<table>
<thead>
<tr>
<th>Type</th>
<th>Procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>Airway management (Ch. 17), mechanical ventilation (see above), thoracentesis, tube thoracostomy</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Cardiac pacing, pericardiocentesis, ED thoracotomy</td>
</tr>
<tr>
<td>Vascular</td>
<td>Arterial puncture/catheterization, peripheral IV, central venous catheterization &amp; CVP monitoring, venous cutdown, IO placement</td>
</tr>
<tr>
<td>Anesthesia</td>
<td>Conscious sedation, nerve blocks</td>
</tr>
<tr>
<td>Skin &amp; soft tissue</td>
<td>Wound closure, FB removal, I&amp;D</td>
</tr>
<tr>
<td>GI</td>
<td>Nasogastric intubation, balloon tamponade of esophageal varices, paracentesis, anorectal procedures</td>
</tr>
<tr>
<td>Orthopedic</td>
<td>Fracture/dislocation reductions, splinting, arthrocentesis, compartment pressure measurement</td>
</tr>
<tr>
<td>GU</td>
<td>Bladder catheterization (urethral, suprapubic)</td>
</tr>
<tr>
<td>OB</td>
<td>Emergency delivery</td>
</tr>
<tr>
<td>Neuro</td>
<td>LP, Dix–Hallpike/Epley maneuver</td>
</tr>
<tr>
<td>Ophtho</td>
<td>Eye irrigation, FB removal, lateral canthotomy</td>
</tr>
</tbody>
</table>
**Arterial Puncture and Catheterization**

**Purpose**
- Puncture to obtain ABG; catheterization for continuous real-time BP monitoring or need for repeat arterial blood sampling

**Equipment**
- Puncture: Local anesthetic, 3-mL syringe, 22-gauge needle (or insulin syringe)
- Catheterization: Arm board, tape, angiocath (size depends on artery cannulated), guidewire, pressure tubing, pressure transducer, suture, needle driver, sterile dressing

**Positioning**
- Ideally placed in the radial, femoral, or DP artery; brachial & axillary are also useable but they are terminal (no collateral supply) so worse prognosis if thrombosis occurs. Document Allen's test before & after catheterization of radial artery.

**Procedure**
- Sterilize area, use sterile gloves, but generally drape & gown are not required
- Puncture: Palpate artery w/ nondominant hand, insert needle distal to palpated artery at a 30° angle to skin, advance until flash in syringe or angiocath, remove 1–2 mL blood, remove air bubbles, & send immediately to lab on ice. US w/ sterile cover may help.
- Catheterization: Immobilize wrist in slight dorsiflexion using tape/arm board, insert needle as above until flash is observed, then advance another 2–3 mm, remove needle & leave catheter, pull back slowly until arterial blood flow is observed, pass guidewire into artery, advance catheter to hub along the guidewire, remove wire, check flow, attach to pressure tubing, suture in place, apply sterile dressing

**Complications**
Hematoma, AV fistula, pseudoaneurysm, bleeding, PAIN. Rarely: Catheter infection, thrombosis or stenosis of artery, hand/limb ischemia.

**Central Venous Catheterization**

**Purpose**
- Rapid volume resuscitation, emergency venous access, administration of spec medicines (ie, pressors, high concentration electrolytes), central venous pressure monitoring
- Sometimes used when peripheral access is not obtainable, but 1st consider external jugular, basilic, or cephalic vein catheterization, or IO access

**Choice of Site**
- Each site has advantages & disadvantages. Overall, no compelling evidence that one site is uniformly superior to others, or definite difference in infection risk. CDC recommends weighing risk/benefits for each pt, but avoiding femoral when possible (“Guidelines for the Prevention of Intravascular Catheter-related Infections”, 2011, CDC: www.cdc.gov).

<table>
<thead>
<tr>
<th>Site</th>
<th>Pros</th>
<th>Cons</th>
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<td>Internal jugular</td>
<td>Bleeding easily controlled</td>
<td>Can be more time consuming w/ U/S</td>
<td>U/S guidance is now the standard of care for IJ</td>
</tr>
<tr>
<td></td>
<td>Low mechanical cx rate</td>
<td>Intermediate risk of infection</td>
<td></td>
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<tr>
<td></td>
<td>when used w/ U/S</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infraclavicular subclavian</td>
<td>Fast, considered ↓ infection rate</td>
<td>↑ Risk of PTX</td>
<td>± U/S guided</td>
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<tr>
<td></td>
<td></td>
<td>↓ Bleeding control</td>
<td></td>
</tr>
<tr>
<td>Supraclavicular subclavian</td>
<td>Practical for cardiac arrest</td>
<td>↑ Risk of PTX</td>
<td>± U/S guided</td>
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<tr>
<td></td>
<td></td>
<td>↓ Bleeding control</td>
<td></td>
</tr>
<tr>
<td>Femoral</td>
<td>Fast, practical during arrest</td>
<td>Thought to have ↑ risk of infection</td>
<td>± U/S guided</td>
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**Equipment**
- Chlorhexidine, cap, mask, sterile drape/gloves/gown, catheter device kit (includes 1% lidocaine w/ 10-mL syringe & 25-gauge needle, catheterization needle/syringe, guidewire, scalpel, dilator, catheter, needle driver, scissors, suture, sterile dressing)
Positioning
- Supine pt, Trendelenburg position for IJ; can do subclavians upright (eg, in CHF)
- Internal jugular: Bedside U/S guidance recommended if available
  - Locate the IJ vein (compressible) & carotid artery (pulsatile, noncompressible) using a sterile U/S probe w/i the triangle created by the clavicle & the sternal & clavicular heads of the sternocleidomastoid muscle
  - Advance the needle toward the IJ vein & away from the carotid artery w/ needle at 30° angle to skin while observing the needle penetrate the vein on U/S towards ipsilateral nipple
  - Confirm venous cannulation via U/S once the wire is in place
- Infraclavicular: Insert needle 2 cm inferior & 2 cm lateral to the angle of the clavicle (located along the middle third), point toward spot just superior to the suprasternal notch & advance just posterior to the clavicle
- Supraclavicular: Insert needle at the junction of the middle & medial thirds of the clavicle, just posterior to the clavicle, point toward the contralateral nipple
- Femoral: Palpate femoral artery, then advance needle at 45° angle to skin toward the head just medial to the palpable artery

Procedure
- Rate of CVL-associated infection ↓ w/ use of observer & checklist (NEJM 2006;355:2725)
- Sterile technique. Attach catheterization needle to syringe, advance while aspirating.
- Remove syringe once vein is entered & check for free return of nonpulsatile blood
- Place the curved end of the guidewire into the needle & advance, check that the wire passes easily, & advance to estimated location of SVC
- Remove the needle while keeping the wire in position
- Make a 1-cm incision through the dermis where the wire meets the skin
- Advance the dilator over the wire several centimeters, then remove the dilator
- Advance catheter over wire, advance to the estimated location of the SVC, remove wire
- Suture in place, cover w/ sterile dressing, obtain CXR to r/o PTX (for all
but femoral lines)

Complications

- Arterial puncture (if needle/wire puncture & compressible, apply prolonged pressure). If a major artery was *dilated*, leave in place & consult IR & vascular surgery.
- PTX: Always r/o w/ XR. Always stat XR if SOB during line placement.
- Bloodstream infection, air embolus, nerve injury (phrenic, brachial plexus, femoral)

Pearls

- A triple-lumen CVC has a slower infusion rate than most PIVs, consider percutaneous introducer if large-volume resuscitation needed

---

**INCISION AND DRAINAGE**

Purpose

- Definitive tx of a soft-tissue abscess

Equipment

- Consider bedside U/S prior to procedure to confirm fluid collection. Hemostat, scissors, forceps, scalpel, packing gauze, 1–2% lidocaine w/ 10-mL syringe & 25-gauge needle.

Procedure

- Anesthetize skin over the most fluctuant area. Make a single, linear incision w/ scalpel over the entire length of the abscess cavity
- Dissect wound using a hemostat & probe into all corners of the cavity to break up loculations & evaluate for an FB, then irrigate wound
- Place enough packing gauze to prevent wound closure but do not pack tightly

---

**INTRAOSSEOUS CATHETERIZATION**

Purpose

- Rapid temporary vascular access. Increasing use in adults & nonemergent cases.

Equipment

- IO needle w/ stylet & syringe, EZ-IO drill if available, gauze
**Positioning**
- Anteromedial aspect of the proximal tibia, 1–3 cm distal to the tibial tuberosity
- Secondary options include distal femur or proximal humerus

**Procedure**
- Sterile technique. Advance IO needle/stylet perpendicular to the bone w/ firm pressure & a twisting motion until the cortex is penetrated, remove stylet, attach syringe & aspirate to correct positioning of the needle. Secure in place w/ gauze pads.

**Complications**
- Infection, bleeding, fracture, retained FB, pain

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**Lumbar Puncture**

**Purpose**
- Dx of meningitis (in the absence of elevated ICP), SAH, idiopathic intracranial HTN, other infectious, inflammatory, neoplastic processes

**Equipment**
- Careful neurologic exam beforehand (avoid in any pt w/ focal neurologic findings), sterile technique, 20–22-gauge spinal or Whitacre needle, LP tray (w/ collecting tubes, lidocaine 1%, manometer/stopcock, 25-gauge needle, 10-cc syringe, sterile drapes)
- Consider U/S in obese pts to identify nonpalpable landmarks
- Consider CTH prior to procedure if concern for mass effect/elevated ICP

**Positioning**
- Lateral decubitus w/ shoulders/hips perpendicular to the bed (preferable & necessary to measure opening pressure) or sitting up on the edge of the bed
- Have the pt maximally flex neck, hips, & knees, & arch back, into a fetal position
- L4 spinous process is found at the intersection of a line b/w the spine & the iliac crests; enter through the interspace above or below this location

**Procedure**
- Anesthetize locally with lidocaine 1% using 25-gauge needle, then advance needle while aspirating → inject lidocaine into the interspinous ligament
- Advance spinal needle toward the umbilicus with bevel pointed toward the pt's side (left or right) until a "pop" or sudden decrease in resistance is felt → remove the stylet
- Once clear fluid is obtained, attach the manometer & record opening pressure
- If fluid is not found, replace the stylet, pull back the needle to the level of SQ tissue, confirm that you are in midline, & reangle your needle slightly
- Obtain at least 1 cc in each collecting tube (more if extensive studies are necessary)
- Replace stylet, remove needle, place sterile dressing over wound
- Tests: Send for cell count (tubes #1, 4), protein/glucose (#2 or #3), gram stain & culture (#2 or #3)

Complications
- HA (5–40%), localized infection, epidural hematoma (rare), herniation (in cases of elevated ICP)

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### Nasogastric Intubation

**Purpose**
- Aspiration of stomach contents in pts at risk for recurrent vomiting (eg, GI obstruction), stomach decompression during trauma or after intubation

**Equipment**
- 16- or 18-gauge NG tube, lubricant, 60-cc syringe, cup of water w/ straw, towel, tape, stethoscope, topical anesthetic jelly, nasal vasoconstrictor

**Positioning**
- Sitting up, chin down

**Procedure**
- Place towel over chest, estimate distance to stomach (from xiphoid to earlobe to stomach)
- Lubricate tube, spray patent nare w/ vasoconstrictor, apply anesthetic
jelly, wait a few minutes

- Advance tube posteriorly along the floor of the nose until it enters the oropharynx, then have pt continuously sip water through straw while the tube is advanced into esophagus; once in the esophagus, quickly advance the tube to the desired distance
- Confirm placement by insufflating the NG tube w/ air using 60-cc syringe & listening over stomach for gush of air, & aspiration of GI contents. Obtain upright CXR if any concern.
- Secure tube using tape attached to the nose & wrapped around the tube from each side; tape should also be used to attach a 2nd segment of the NG tube to the gown

**Complications**

- Vomiting during placement, tracheal intubation, small risk of intracranial penetration (contraindicated in facial fractures), bleeding, esophageal rupture (hx of esophageal stricture/alkali injury), pain

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**Paracentesis**

**Purpose**

- Diagnostic: Removal of peritoneal fluid in a pt w/ ascites to (a) diagnose the cause of new ascites; (b) assess for spontaneous bacterial peritonitis
- Therapeutic: Relieve sxs in pts w/ tense ascites (eg, hypoxia from mass effect)

**Equipment**

- Use bedside U/S prior to procedure to confirm ascites & identify large pocket
- Sterile technique
- 25-gauge needle, 1% lidocaine. For diagnostic tap, only need 20–22-gauge needle & large syringe to aspirate fluid. For therapeutic tap, use paracentesis kit w/ 18-gauge needle, catheter sheath, & vacuum-sealed collection bottles.

**Positioning**

- Supine; identify entry site: Usually 4–5 cm cephalad & medial to anterior superior iliac spine, lateral to the rectus muscle sheath, being careful to avoid any visible veins
**Procedure**
- Check for severe coagulopathy prior to procedure
- Perform w/ real-time bedside U/S if possible
- Anesthetize locally w/ lidocaine 1% using 25-gauge needle
- Z-tract: Pull the skin 2 cm caudad before advancing the larger-bore needle, then place the needle perpendicular to the skin, advance needle slowly while occasionally aspirating, until ascitic fluid is aspirated, then release skin
- It may be necessary to make a 0.5 cm stab incision at the dermis to allow passage of the needle/catheter
- After aspirating fluid, advance catheter 1–2 cm & remove needle → connect catheter to stopcock, & collect fluid into sterile containers
- Fluid: Send for cell count, albumin, culture. Consider total protein, glucose, LDH, amylase, gram stain

**Complications**
- Hypotension (can have severe fluid shifts in large-volume tap), ascitic fluid leakage, abdominal wall hematoma, infection, hemoperitoneum, visceraperforation

---

**PERICARDIOCENTESIS**

**Purpose**
- Emergent tx of pericardial effusion/tamponade in a pt w/ cardiac arrest (often PEA) or periarrest; hemorrhagic tamponade is best treated w/ thoracotomy

**Equipment**
- 16- or 18-gauge spinal needle attached to a 30- or 60-cc syringe

**Positioning**
- Supine pt, angle needle 30°–45° to the skin, insert b/w xiphoid process & left costal margin, aim needle toward left shoulder (parasternal technique: 90° angle above 5th/6th rib L sternal border)

**Procedure**
- Sterile technique. Bedside U/S guidance recommended if available. Advance needle slowly while aspirating until fluid is removed (presence of blood suggests ventricular puncture)
Complications
- “Dry tap,” PTX, myocardial laceration, coronary vessel laceration, hemopericardium, ventricular penetration, visceral injury

Thoracentesis

Purpose
- Diagnostic eval (new/unclear etiology) or therapeutic tx of pl effusion

Equipment
- 20- or 22-gauge needle w/ catheter or thoracentesis kit

Positioning
- Pt sitting upright, needle angled 90° to skin, insert in intercostal space above rib (no lower than 8th intercostal space) in midscapular line

Procedure
- Sterile technique. Bedside U/S guidance is recommended for locating height of effusion & distance of lung from the parietal pleura.
- Anesthetize locally w/ lidocaine 1% using 25-gauge needle, then advance needle while aspirating → inject lidocaine → advance while aspirating further, until pl fluid is aspirated
- Remove needle, make a small 0.5 cm incision at the insertion site, then insert 20- or 22-gauge needle w/ catheter → advance while aspirating
- After aspirating fluid, advance catheter & remove needle
- Connect catheter to stopcock, & collect fluid into sterile containers
- Goal: Diagnostic (50–100 mL), therapeutic (relief of dyspnea, up to 1500 mL)
- Fluid: Send for LDH, protein, glucose, cell count, amylase, cytology, gram stain, culture
- Obtain postprocedure CXR

Complications
- PTX, bleeding (caution if PLT <50000 or >1.5 × nl PT/PTT), cough, infection, hemothorax, diaphragmatic penetration

Tube Thoracostomy

Purpose
Drainage of air (PTX), blood (hemothorax), or fluid (pleural effusion, empyema) in the pleural space that threatens cardiac or pulmonary function

**Equipment**
- #10 scalpel, Kelly clamp, #0 or 1 suture, scissors, chest tube (28F minimum, larger for hemothorax; may consider pigtail catheter for small PTX)

**Positioning**
- Supine pt, shoulder abducted (raised overhead), enter at midaxillary line @ 4th–5th intercostal space (nipple line), lateral to pectoralis major

**Procedure**
- Sterile technique
- Create wheal using lidocaine 1% w/ epinephrine (1:100000) & a 25- or 27-gauge needle, then advance needle while aspirating, & infiltrate broadly through muscle, periosteum & parietal pleura, staying above the rib; ± intercostal nerve block (at level, above, & below)
- Make 3–4 cm incision parallel & just over rib, through skin & fat overlying the rib
- Perform blunt dissection w/a Kelly or scissors down to the rib & just above it
- Apply firm pressure w/ the Kelly closed to pop through the parietal pleura
- Look/listen for rush of fluid or air. Leave Kelly in place & spread to open the pleura further.
- Insert finger into the chest wall (Kelly still in place) to verify that it is the pleural space (feel lung, ensure no abdominal organs)
- Keep finger in place, remove Kelly, pass the tube over finger while gently spinning the tube
- Typically, direct tube superiorly & posteriorly (can go anteriorly if certain there is only air)
- Rotate the tube 360° to ↓ kinking & ensure all the tube holes are in the pleural space
- Attach to water seal or suction. Never clamp a chest tube
- Confirm placement: Condensation w/ respiration, bubbles in water seal w/ coughing, CXR
- Suture in place, place petroleum gauze over wound, cover w/ dry gauze & tape in place
Complications

- Infection, intercostal vessel/nerve laceration, lung laceration, intra-abdominal entry, solid organ tube placement, subcutaneous placement
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