MNEMONICS AND PEARLS HANDBOOK

For Residents, Medical Students, Nurses and Pre-Hospital Personnel

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Hello and thank you for having an interest in *Mnemonics and Pearls For Residents, Medical Students and Pre-hospital Personnel*.

This book contains mnemonics that will assist you in rapidly learning the essentials in medicine. The “pearls” will help you answer questions frequently asked in rounds or on board exams.

**HOW TO USE THIS HANDBOOK**

Each mnemonic is presented in the following format:

<table>
<thead>
<tr>
<th>Causes of Chest Pain</th>
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<tbody>
<tr>
<td><strong>Mnemonic</strong> - <em>(MAPLE)</em> 3 [1, with modifications]</td>
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- **Causes of Chest Pain** - This mnemonic covers the causes of chest pain
- **(MAPLE)** - the mnemonic
- **3** - indicates that each letter is used three times, for example the three “M’s” for this mnemonic are:

| Myocardial Infarction - Musculoskeletal - Myocarditis |

- The superscript enclosed by brackets are references. In this example, reference 1 is Rogers PT: *The Medical Student’s Guide to Board Scores*. Reference materials can be found on pages xx-xx. In this example, modifications were made to Dr. Roger’s mnemonic. Therefore, the reference is listed as, [1, with modifications]
- If the reference/author is now known, ANK is placed next to the mnemonic.

Many of the “pearls” have been obtained from the references below:


Every attempt has been made to ensure accuracy.

If you have any suggestions, corrections, mnemonics or pearls you would like to share, please email: stevesfmc@gmail.com, billgossmanmd@gmail.com
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Good luck and we hope this handbook is helpful.

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HISTORY OF PRESENT ILLNESS

Mnemonic - (O P₃ Q R S₃ T)

<table>
<thead>
<tr>
<th>O</th>
<th>Onset - What time did the symptoms start - What activity caused the symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>P₃</td>
<td>Pain location</td>
</tr>
<tr>
<td></td>
<td>Palilative (what makes the pain better)</td>
</tr>
<tr>
<td></td>
<td>Provocative factors (what makes the pain worse)</td>
</tr>
<tr>
<td>Q</td>
<td>Quality (sharp, dull, heavy, burning, squeezing, etc.)</td>
</tr>
<tr>
<td>R</td>
<td>Radiation (arms, jay, back, groin, etc.)</td>
</tr>
<tr>
<td>S₃</td>
<td>Severity (use pain scale 1 to 10, ten being the most severe pain)</td>
</tr>
<tr>
<td></td>
<td>Symptoms associated (nausea, vomiting, diaphoresis, SOB, F/C)</td>
</tr>
<tr>
<td></td>
<td>Similar episodes in past</td>
</tr>
<tr>
<td>T</td>
<td>Timing (how long, constant vs. intermittent)</td>
</tr>
</tbody>
</table>

DETERMINING THE ETIOLOGY OF DISEASE PROCESSES

Mnemonic - (AN INDICATIVE DIFFERENTIAL DIAGNOSIS DD) ANK with Modifications

| A  | Allergy                           |
| N  | Neoplasm                          |
| I  | Infection                         |
| N  | Nosocomial                        |
| D  | Drugs                             |
| I  | Intoxication                      |
| C  | Congenital                        |
| A  | Autoimmune                        |
| T  | Trauma                            |
| I  | Inflammation                      |
| V  | Vascular                          |
| E  | Endocrine                         |
| D  | Deficiency                        |
| D  | Degenerative                      |

If you come up empty with all the above consider PSYCH
MORE HISTORY

Mnemonic - (AMPLE)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
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<tbody>
<tr>
<td>A</td>
<td>Allergies</td>
</tr>
<tr>
<td>M</td>
<td>Meds</td>
</tr>
<tr>
<td>P</td>
<td>Previous medical history</td>
</tr>
<tr>
<td>L</td>
<td>Last meal/ LMP</td>
</tr>
<tr>
<td>E</td>
<td>Events</td>
</tr>
</tbody>
</table>
CAUSES OF CHEST PAIN

Mnemonic - (MAPLE)3 (PCP2) [1, with modifications]

| M | Myocardial Infarction |
| M | Musculoskelatal |
| M | Myocarditis |
| A | Aortic dissection |
| A | Angina |
| A | Anxiety |
| P | PE |
| P | Pneumothorax |
| P | Pneumonia |
| L | Low H/H |
| L | Lung CA |
| L | Lesions, Skin (Herpes Zoster) |
| E | Esophageal rupture |
| E | Esophagitis |
| E | GERD |
| P | Pyelonephritis |
| C | Cholecystitis |
| P | Pancreatitis |
| P | Pericarditis |

SERUM MARKERS IN THE DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION

<table>
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<th>Marker</th>
<th>Earliest Rise</th>
<th>Peak</th>
<th>Normalize</th>
</tr>
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<tr>
<td>Myoglobin</td>
<td>1-2 hr</td>
<td>4-8 hr</td>
<td>1st Day</td>
</tr>
<tr>
<td>CK-MB</td>
<td>3-4 hr</td>
<td>10-24 hr</td>
<td>2nd Day</td>
</tr>
<tr>
<td>MB-Isoforms</td>
<td>2-4 hr</td>
<td>6-12 hr</td>
<td>1st Day</td>
</tr>
<tr>
<td>Troponin</td>
<td>2-4 hr</td>
<td>10-24 hr</td>
<td>5-12 Days</td>
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TIMI RISK SCORE FOR UNSTABLE ANGINA / NON-ST ELEVATION MI

Mnemonic - (AMERICA) [Emergency Medicine Journal 2008;25:122, provided by Dr. Jeff Kovar]

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<tr>
<td>A</td>
<td>Age (&gt; 65 years)</td>
</tr>
<tr>
<td>M</td>
<td>Markers (raised serum cardiac markers)</td>
</tr>
<tr>
<td>E</td>
<td>ECG (ST-segment depression at presentation; ST change &gt; 0.5mm)</td>
</tr>
<tr>
<td>R</td>
<td>Risk factors (at least 3/5 for CAD – DM, HTN, HL, Family Hx of CAD, smoking)</td>
</tr>
<tr>
<td>I</td>
<td>Ischemia (at least two anginal events in previous 24 hours)</td>
</tr>
<tr>
<td>C</td>
<td>Coronary stenosis (prior stenosis of 50% or more)</td>
</tr>
<tr>
<td>A</td>
<td>Aspirin (use in previous seven days)</td>
</tr>
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TIMI RISK SCORE OF 30 DAY MORBIDITY / MORTALITY [JAMA. 2000;284:835-842]

4.7% for a score of 0/1
8.3% for 2
13.2% for 3
19.9% for 4
26.2% for 5
40.9% for 6/7

MAJOR RISK FACTORS ASSOCIATED WITH ISCHEMIC HEART DISEASE

Age > 40
Male sex
Family history
Cigarette smoking
HTN
DM
Hypercholesterolemia
Obesity
Known CAD

HEART SCORE FOR MAJOR CARDIAC EVENTS

[Neth Heart J. 2008 Jun; 16(6): 191–196]

Predicts 6-week risk of major adverse cardiac event (MACE)

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<th>EKG</th>
<th>AGE</th>
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<td>Highly suspicious +2</td>
<td>Significant ST-depression +2</td>
<td>≥ 65 +2</td>
</tr>
<tr>
<td>Moderately suspicious +1</td>
<td>Non specific repolarisation disturbance +1</td>
<td>45-65 +1</td>
</tr>
<tr>
<td>Slightly suspicious 0</td>
<td>Normal 0</td>
<td>≤ 45 0</td>
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<th>RISK FACTORS</th>
<th>TROPONIN</th>
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<td>Hypercholesterolemia, Hypertension, Diabetes,</td>
<td>≥ 3× normal limit +2</td>
</tr>
<tr>
<td>Smoking, + Fam Hx, Obesity</td>
<td>1-3× normal limit +1</td>
</tr>
<tr>
<td>No risk factors known 0</td>
<td>≤ normal limit 0</td>
</tr>
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</table>
The HEART Score is a prospectively studied scoring system to help emergency departments risk-stratify chest pain patients: who will have a MACE within the next 6 weeks and who will not?

- It involves only a 1-time troponin, at admission.
- The rest of the score is based on age, history, risk factors, and EKG.
- Low risk patients have a score 0-3 and have a less than 2% risk of MACE at 6 weeks.
- MACE is defined as: all-cause mortality, myocardial infarction, or coronary revascularization.
- All other scores are high risk (risk increasing exponentially) and require further management and admission.

CAUSES OF ST SEGMENT ELEVATION

**Mnemonic (ELEVATION) 2** [Acad Emerg Med, 1999;6:930, with modifications]

| E | Electrolyte abnormalities (↑ K+)
Excitation (WPW→ Delta wave) |
| L | Left bundle branch block
Left ventricular hypertrophy |
| E | Early repolarization
Embolism |
| V | Ventricular paced rhythms
Variant angina (Prinzmetal’s angina) |
| A | Aneurysm (left ventricular)
AMI |
| T | Trauma (contusion)
Treatment (pericardiocentesis) |
| I | Intracranial hemorrhage
Inflammation (pericarditis/myocarditis) |
| O | Osborn J waves (hypothermia) The amplitude of the J-wave is proportional to the degree of hypothermia; does not relate to pH and is not prognostic\[10, 5th ed., pg. 1981\]
Overdose-cocaine |
| N | NSSTT – wave change
Nonocclusive vasospasm - (Prinzmetal’s angina, cocaine) |

EKG FINDINGS IN AMI

EKG diagnosis of AMI = > 1 mm (0.1 mV) of STE in limb leads, and at least 2 mm elevation in the precordial leads

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<th>Coronary Artery Involvement</th>
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<td>V1-V2</td>
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<td>Anterior</td>
<td>V3-V4</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL plus V5, V6</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Posterior</td>
<td>V8-V9</td>
</tr>
<tr>
<td></td>
<td>STD w/upright T V1</td>
</tr>
<tr>
<td></td>
<td>R/S ratio &gt; 1 V1-V2</td>
</tr>
<tr>
<td>Right Ventricle</td>
<td>V4R (II, III, aVF)</td>
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QUICK EKG INTERPRETATION OF STEMI V1-V6
Mnemonic - (SSAALL)

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<td>S</td>
<td>A</td>
<td>A</td>
<td>L</td>
<td>L</td>
</tr>
<tr>
<td>V1 SEPTAL</td>
<td>V2 SEPTAL</td>
<td>V3 ANTERIOR</td>
<td>V4 ANTERIOR</td>
<td>V5 LATERAL</td>
<td>V6 LATERAL</td>
</tr>
</tbody>
</table>

AMI IN LBBB (SGARBOSSA CRITERIA)

STE > 1mm concordant with QRS  5 points
ST depression > 1mm in V1, V2, V3  3 points (STD in V1-V3 concurrently = 3 points)
STE > 5mm discordant with QRS  2 points

Score > 3 suggest MI (90% specific, however 36% sensitive)

Clinical utility of criteria are insensitive and probably have relatively low utility

SGARBOSSA CRITERIA / LBBB PACED RHYTHM

BRUGADA SYNDROME

Common cause of sudden death; genetic disease

ST segment elevation V1-V3 and/or “saddle deformity” of ST-T segment, with RBBB with or without the terminal S waves in the lateral leads that are associated with a typical RBBB

ENRs of Brugada Types 1-3 go to
http://lifeinthefastlane.com/ecg-library/brugada-syndrome
TREATMENT OPTIONS IN ACUTE CORONARY SYNDROME

Mnemonic - (HE BE MOAN) [21, with modifications]


<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>HE</td>
<td>UNFRACTIONATED HEPARIN (UFH) IV [Ref 29, 2010 AHA pg. 49]</td>
</tr>
<tr>
<td></td>
<td>STEMI and NSTEMI</td>
</tr>
<tr>
<td></td>
<td>• 60 U/kg IV bolus (max 4,000 U),</td>
</tr>
<tr>
<td></td>
<td>• Followed with 12 U/kg/hr infusion (max 1,000 U/hr)</td>
</tr>
<tr>
<td></td>
<td>LOVENOX (LMWH) DOSAGES [Ref 29, 2010 AHA pg. 50]</td>
</tr>
<tr>
<td></td>
<td>STEMI – age &lt; 75 years with normal CrCl</td>
</tr>
<tr>
<td></td>
<td>• Fibrinolysis = 30 mg IV bolus</td>
</tr>
<tr>
<td></td>
<td>• Follow with 1mg/kg SC q 12 hr (first dose 15 minutes after IV dose, with maximum of 100 mg for the first two doses only)</td>
</tr>
<tr>
<td></td>
<td>• Dose for patients &gt; 75 y/o = 0.75mg/kg SC q 12 hr without IV bolus (max 75 mg/dose for first 2 doses)</td>
</tr>
<tr>
<td></td>
<td>• Dose for patients with CrCl &lt; 30 mL/min = 1mg/kg SC q 24 hours</td>
</tr>
<tr>
<td></td>
<td>• PCI = If last SC dose of lovenox was &lt; 8 hours before</td>
</tr>
<tr>
<td></td>
<td>• PCI → no additional dosing needed; if &gt; 8 hrs bolus with</td>
</tr>
<tr>
<td></td>
<td>• 0.3 mg/kg IV [<a href="http://www.druglib.com/druginfo/lovenox/indications_dosage">www.druglib.com/druginfo/lovenox/indications_dosage</a>]</td>
</tr>
<tr>
<td></td>
<td>LOVENOX (LMWH) DOSAGES [Ref 29, 2010 AHA pg. 50]</td>
</tr>
<tr>
<td></td>
<td>NSTEMI</td>
</tr>
<tr>
<td></td>
<td>• Loading dose 30 mg IV bolus</td>
</tr>
<tr>
<td></td>
<td>• Maintenance dose = 1mg/kg SC q 12 hours</td>
</tr>
<tr>
<td></td>
<td>• Reduce dose if creatinine clearance &lt; 30 mL/min = 1mg/kg SQ daily</td>
</tr>
<tr>
<td></td>
<td>EMS: In systems in which UFH is currently administered in the prehospital setting for patients with suspected STEMI who are being transferred for PPCI, it is reasonable to consider prehospital administration of enoxaparin as an alternative to UFH. (Class IIa, LOE B-R) [*AHA, 2015, page 491]</td>
</tr>
<tr>
<td>BE</td>
<td>BEta Blocker</td>
</tr>
<tr>
<td></td>
<td>NSTEMI</td>
</tr>
<tr>
<td></td>
<td>Oral beta-blocker within 24 h (Class I, LOE A); Metoprolol 50 mg po</td>
</tr>
<tr>
<td></td>
<td>Lopressor 5 mg IV at 5 min intervals x 3 = total 15 mg</td>
</tr>
<tr>
<td></td>
<td>(Class IIa, LOE B) In the absence of contraindications</td>
</tr>
<tr>
<td>M</td>
<td>Morphine</td>
</tr>
<tr>
<td></td>
<td>NSTEMI patients: downgraded from Class 1, LOE C → IIa, LOE B potentially adverse effects of morphine in patients with UA/NSTEMI [*AHA, 2015]</td>
</tr>
<tr>
<td></td>
<td>STEMI patients (Class I, LOE C) [Ref 29, 2010 AHA pg. 26]</td>
</tr>
<tr>
<td>O</td>
<td>Oxygen</td>
</tr>
<tr>
<td></td>
<td>The usefulness of supplementary oxygen therapy has not been established in normoxic patients. In the prehospital, ED, and hospital settings, the withholding of supplementary oxygen therapy in normoxic patients with suspected or confirmed acute coronary syndrome may be considered. (Class IIb, LOE C-LD) [*AHA, 2015, page 491]</td>
</tr>
</tbody>
</table>
**Cardiology**

<table>
<thead>
<tr>
<th>Mnemonics &amp; Pearls</th>
<th>Cardiology</th>
</tr>
</thead>
</table>
| **Antiplatelet**   | ASA162-325 mg po/crushed/chewed; vomiting = 300mg rectal; ASAP  
If ASA contraindicated: clopidogrel (Plavix) 300 mg po [Ref 29, AHA pg. 39] |
| LOE = Level of Evidence | **NSTEMI Conservative Therapy** (Class I, LOE A)  
ASA + add clopidogrel (Plavix) 300 mg po asap after admission |
| Clopidogrel Limited evidence patients > 75 y/o | **NSTEMI Invasive Strategy** (PCI within 4 to 24 hours)  
< 75 years old  
Give ASA + clopidogrel (Plavix) 300 to 600 mg or IV glycoprotein IIb/IIIa (GP IIb/IIIa) inhibitor |
| A | **STEMI** < 75 years old  
ASA + add Prasugrel (Effient) 60 mg po prior to primary PCI (Class I, LOE B)  
or  
ASA + add clopidogrel (Plavix) 300 to 600 mg po before PCI or non-primary PCI [Ref 29, 2010 AHA pg. 39]; (Class I, LOE C)  
Fibrinolytic therapy: ASA + add clopidogrel 300 mg po (Class I, LOE B) |
| **Nitroglycerin** (Class I, LOE B) | NTG 0.4 mg sublingual or spray every 5 minutes; if no improvement after 3 tablets/sprays, start IV NTG at 10 mcg/min continuous infusion, ↑ 10 mcg/min every 3 to 5 min until relief or hypotension  
Hold if patients have recently taken ED meds sildenafil (Viagra) or vardenafil (Levitra), tadalafil (Cialis)  
Hold if extreme bradycardia (<50 bpm) or tachycardia (>100 bpm) in absence of heart failure  
Hold in patients with RV infarct [Ref 29, 2010 AHA pg. 54] |

**TREATMENT OPTIONS IN ACS**

ASA alone relative reduction in cardiovascular mortality = 20 to 25% [10, 5th ed., pg 1042]  
ASA with lytics 42% reduction in mortality

ASA → Inhibits cyclooxygenase → ↓ thromboxane A2 production → ↓ platelet aggregation and less arterial constriction

Clopidogrel (Plavix) and Prasugrel (Effient) → irreversibly inhibit ADP receptor on platelet cell membranes → block activation / transformation glycoprotein IIb/IIIa receptor

If CABG planned → hold Clopidogrel for 5 days, Prasugrel for 7 days [Ref 29, 2010 AHA pg. 39]

Prasugrel (Effient) is contraindicated in patients with history of TIA, CVA; use with caution in patients > 75 years old or < 60 kg due to risk of fatal bleeding, ICH and uncertain benefit; Not recommended in STEMI patients managed with fibrinolysis [Ref 29, 2010 AHA pg. 39, 40]
GLYCOPEPTIDE IIb/IIIa INHIBITORS

→ prevent fibrinogen/vWF crosslinking → ↓ platelet aggregation

[Ref 29, 2010 AHA pg. 48]

Abciximab (ReoPro)
- STEMI with emergent PCI
  - 0.25 mg/kg IV bolus 10 to 60 minutes before PCI followed by
  - 0.125 mcg/kg/minute (max of 10 mcg/minute) IV infusion for 12 hours
- NSTEMI Invasive Strategy (PCI planned within 24 hours)
  - 0.25 mg/kg IV bolus, then
  - 10 mcg per minute IV infusion for 18 to 24 hours, end 1 hour after PCI
  - Must use with heparin

Eptifibatide (Integrilin) – NSTEMI Invasive Strategy
- 180 mcg/kg IV bolus over 2 minutes then begin 2 mcg/kg/min IV infusion repeat 180 mcg/kg IV bolus over 2 minutes in 10 minute
- Maximum dose (121-kg patient) for PCI: 22.6 mg bolus; 15 mg per hour infusion
- Infusion duration 18 to 24 hours after PCI
- Reduce infusion rate by 50% if CrCl < 50 mL/min

Tirofiban (Aggrastat) – NSTEMI Invasive Strategy
- 0.4 mcg/kg/min IV for 30 minutes and then continued at 0.1 mcg/kg/min IV infusion for 18 to 24 hours after PCI
- Reduce infusion rate by 50% if CrCl < 30 mL/min

MANAGEMENT OF COCAINE-INDUCED ACS

• SL NTG and a CCB (e.g., diltiazem 20 mg IV); avoid B-blockers
• If ST-segment elevation is present and the patient is unresponsive to initial treatment, immediate coronary angiography is preferred over fibrinolytic therapy.

• UA/NSTEMI → observed and managed medically for 9 to 24 h. If EKG and biomarkers are normal and the patient is stable, the patient can be discharged.

PERCUTANEOUS CORONARY INTERVENTION (PCI) - STEMI

• When performed within 90 minutes of patient arrival has been shown to be superior to fibrinolysis in combined end points of death, stroke, and reinfarction in many studies
• Primary PCI performed at a high-volume center within 90 minutes of first medical contact by an experienced operator that maintains an appropriate expert status (>200 PCI/year) is reasonable, as it improves morbidity and mortality as compared with immediate fibrinolysis

[Class I, LOE A] [https://eccguidelines.heart.org/index.php/circulation/cpr-ecc-guidelines-2/part-9-acute-coronary-syndromes/]

DIAGNOSTIC ANGIOGRAPHY IN NSTEMI PATIENTS

• Persistent chest pain/symptoms/ischemia, heart failure, or arrhythmias, then diagnostic angiography should be performed (level of evidence: A)
THROMBOLYTIC AGENTS RECOMMENDED (AMI)
[Ref 29, 2010 AHA pg. 25, 46]

Presentation < 12 hours in context of signs and symptoms of AMI
• ST-segment elevation (> 1 mm in > 2 contiguous leads)
• Posterior-wall MI
• New or presumably new left bundle-branch block
• No exclusion criteria

Fibrinolytic therapy is generally not recommended for patients presenting between 12 and 24 hours after onset of symptoms based on the results of the LATE and EMERAS trials, unless continuing ischemic pain is present with continuing ST-segment elevation. (Class IIb, LOE B)

Fibrinolytic therapy should not be administered to patients who present greater than 24 hours after the onset of symptoms. (*Class III, LOE B)

Alteplase (tPA) – Accelerated infusion regimen is given over 1.5 hours
Give 15mg IV Bolus
Then 0.75 mg/kg (max 50 mg) over next 30 minutes
Then 0.50 mg/kg (max 35 mg) over next 60 minutes

Reteneplase (Retavase)
10 U IV over 2 min
30 minutes later give second 10 U IV bolus over 2 minutes

Tenecteplase (TNKase)
Bolus: 30 to 50 mg, weight adjusted (not to exceed 50 mg)

ALTEPASE (TPA) AND HEPARIN DOSING IN AMI, CVA AND PULMONARY EMBOLISM

AMI
Alteplase (tPA) Accelerated infusion regimen is given over 1.5 hours [AHA, 2005 page 52]
Give 15mg IV Bolus
Then 0.75 mg/kg (max 50 mg) over next 30 minutes
Then 0.50 mg/kg (max 35 mg) over next 60 minutes

Heparin [AHA, 2005 page 55]
Begin heparin with fibrin-specific lytics (tPA, Retavase and TNKase)
Unfractionated Heparin (UFH) or Lovenox = ancillary therapy w/ fibrinolytic

CVA
tPA dose = 0.9 mg/kg, max 90mg
First 10% bolus over 1 min, remaining infused over next 60 minutes
Do not administer heparin or ASA during the first 24 hrs of fibrinolytic therapy [AHA, 2005 page 52]

Pulmonary Embolism = You will find three different protocols for tPA
• 100 mg over 2 hours (FDA approved regimen, most textbooks) or
• 15 mg bolus, then 85 mg continuous infusion over 2 hours or
• Accelerated infusion regimen used in AMI

Hold heparin during fibrinolytic infusion. At the conclusion of alteplase infusion begin heparin infusion without a bolus when aPTT has decreased to less than < 80 seconds [Circulation. 2005;112:e28-e32 Management of Massive Pulmonary Embolism]
CONTRAINDICATIONS AND CAUTIONS FOR FIBRINOLYSIS USE IN STEMI
[Circulation. 2004;110:588-636]

Absolute contraindications
- Any prior ICH
- Known structural cerebral vascular lesion (eg, AVM)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 months

Relative contraindications
- History of chronic severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP > 180 mm Hg or DBP > 110 mm Hg)
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (> 10 minutes) CPR or Major surgery (< 3 weeks)
- Recent internal bleeding (within 2 to 4 weeks)
- Noncompressible vascular punctures
- For streptokinase/anistreplase: prior exposure (> 5 days) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA
[2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care]

- Start CPR, provide maximum oxygen, attach monitor/defibrillator →
- Unsynchronized Cardioversion = Biphasic preferred, manufacturer recommendation (120 to 200J); if unknown use max available; If only Monophasic available 360J →
- Resume CPR immediately – 2 minutes → check rhythm → shockable rhythm →
- 200J Biphasic or 360J Monophasic → Resume CPR immediately
- When IV available →
  Epinephrine 1 mg IVP repeat every 3 to 5 minutes
- CPR 2 minutes → check rhythm → shockable rhythm →
- 200J Biphasic or 360J Monophasic →
  Amiodarone 300 mg once IVP; second dose (if needed) 150mg IVP
- Extracorporeal membrane oxygenation: ECPR (or ECMO) techniques
  ECPR refers to venoarterial extracorporeal membrane oxygenation during cardiac arrest, including extracorporeal membrane oxygenation and cardiopulmonary bypass. These techniques require adequate
vascular access and specialized equipment. The use of ECPR may allow providers additional time to treat reversible underlying causes of cardiac arrest (eg, acute coronary artery occlusion, pulmonary embolism, refractory VF, profound hypothermia, cardiac injury, myocarditis, cardiomyopathy, congestive heart failure, drug intoxication etc) or serve as a bridge for left ventricular assist device implantation or cardiac transplantation.

There is insufficient evidence to recommend the routine use of ECPR for patients with cardiac arrest. In settings where it can be rapidly implemented, ECPR may be considered for select cardiac arrest patients for whom the suspected etiology of the cardiac arrest is potentially reversible during a limited period of mechanical cardiorespiratory support. ([Class IIb, LOE C-LD](https://eccguidelines.heart.org/index.php/circulation/cpr-ecc-guidelines-2/part-7-adult-advanced-cardiovascular-life-support/))

- Give 5 cycles of CPR → check rhythm → shockable rhythm → Start at top again
- Treat reversible causes
- CPR = Compressions, push hard > 2 inches (5cm), and fast > 100-120/min and allow complete chest recoil. Minimize interruptions in compressions.
- Rotate compressor every 2 minutes or sooner if fatigued. If no advanced airway 30:2 compression-ventilation ratio. If advanced airway – continue compressions and give 8 to 10 breaths/minute and check rhythm every 2 minutes. Avoid excessive ventilation. If PETCO2 < 10mmHg, attempt to improve CPR quality
- Narcan recommendation for agonal respirations [AHA, 2015]

**REVERSIBLE CAUSES OF PULSELESS V-FIB/V-TACH**

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo / hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

**MONOPHASIC VS BIPHASIC DEFIBRILLATORS**

(2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care; S708)

- Monophasic waveforms deliver current of one polarity (i.e., direction of current flow)
- Biphasic is like an AC jolt, part of the shock goes from one paddle to the next and then reverses from the opposite paddle back. Less energy is required for the same effect.
MEDICATIONS THAT CAN BE GIVEN DOWN ET TUBE

Mnemonic **(NAVEL)**

| N | Narcan |
| A | Atropine |
| V | Valium |
| E | Epinephrine |
| L | Lidocaine |

Also, isoproterenol [10, 5th ed., pg. 78]

ACC/AHA CLASSIFICATION OF RECOMMENDATIONS


<table>
<thead>
<tr>
<th>Class</th>
<th>Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective</th>
</tr>
</thead>
</table>
| Class I | Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.  
Ia. Weight of evidence/opinion is in favor of usefulness/efficacy  
Iib. Usefulness/efficacy is less well established by evidence/opinion. |
| Class III | Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective, and in some cases may be harmful. |

Level of Evidence

- **Level of Evidence A**  
  Data derived from multiple randomized clinical trials
- **Level of Evidence B**  
  Data derived from a single randomized trial, or non-randomized studies
- **Level of Evidence C**  
  Consensus opinion of experts

CAUSES OF ATRIAL FIBRILLATION

Mnemonic **(ME WITH MITCH PhD)**  
Drs. Archer and Christos

Atrial rate = 350-500, ventricular rate = 100-160; Irregular

| M | Mitral valve disease (MS, MR) |
| E | Electrolytes |
| W | WPW |
| I | Intoxication/ETOH (holiday heart) |
| T | Thyrotoxicosis |
| H | HTN |
| M | Myocarditis |
| I | Idiopathic |
| T | Tox (CO, cocaine, amphetamines, heroin) |
| C | CAD |
| H | Hypoxia (COPD) although more commonly Multifocal Atrial Tachycardia (MAT) |

<table>
<thead>
<tr>
<th>P</th>
<th>PE</th>
</tr>
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<tbody>
<tr>
<td>h</td>
<td>Hypothermia</td>
</tr>
<tr>
<td>D</td>
<td>Drugs (see tox above)</td>
</tr>
</tbody>
</table>
**ATRIAL FIBRILLATION**

If unstable (ie hypotensive) → “Synchronized” Cardioversion

*Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex. This synchronization avoids shock delivery during the relative refractory portion of the cardiac cycle, when a shock could produce VF.*

- Biphasic 120 to 200 J preferred, if not available then monophasic cardioversion at 200J
- If the initial shock fails, providers should increase the dose in a stepwise fashion
- ↑ success of cardioversion if pretreatment with Amiodarone
- Electrical cardioversion in Digoxin Toxicity → malignant ventricular arrhythmias
- Avoid electrical cardioversion if patient is on Digoxin unless condition is life-threatening, then use lower dose → 10 to 20 J

**APPROACH TO SELECTING DRUG THERAPY FOR VENTRICULAR RATE CONTROL**

[http://circ.ahajournals.org/content/early/2014/04/10/CIR.0000000000000041.full.pdf]

2014 AHA/ACC/HRS Atrial Fibrillation Guidelines

1 Drugs are listed alphabetically.

2 Beta blockers should be instituted following stabilization of patients with decompensated HF. The choice of beta blocker (cardio selective, etc.) depends on the patient’s clinical condition.

3 Digoxin is usually not first line-therapy. It may be combined with a beta blocker and/or a nondihydropyridine calcium channel blocker when ventricular rate control is insufficient and may be useful with patients in HF.

4 In part, because of concerns over its side-effect profile, use of amiodarone for chronic control of ventricular rate should be reserved for patients who do not respond or are intolerant of beta blockers or nondihydropyridine calcium antagonists.
**THE ACC/AHA GUIDELINES FOR PHARMACOLOGIC CONVERSION OF ATRIAL FIBRILLATION (AF)**

(JACC Vol. 48, No.4, 2006; August 15, 2006:e149-e246. Section 8.1.5.)

<table>
<thead>
<tr>
<th>Conversion of AF &lt; 7 days</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class I</strong></td>
</tr>
<tr>
<td>Dofetilide (Tikosyn) oral</td>
</tr>
<tr>
<td>Ibutilide (Corvert) IV</td>
</tr>
<tr>
<td>Flecaïnide (Tambocor) oral or IV</td>
</tr>
<tr>
<td>Propafenone (Rythmol) oral or IV</td>
</tr>
<tr>
<td>Amiodarone (Cordarone) IIa oral or IV</td>
</tr>
<tr>
<td><strong>Class IIb</strong></td>
</tr>
<tr>
<td>(Less effective or incompletely studied agents)</td>
</tr>
<tr>
<td>Disopyramide (Norpace) IV</td>
</tr>
<tr>
<td>Procainamide (Pronestyl) IV</td>
</tr>
<tr>
<td>Quinidine (Biquin) oral</td>
</tr>
<tr>
<td><strong>Class III</strong></td>
</tr>
<tr>
<td>Digoxin oral or IV</td>
</tr>
<tr>
<td>Sotalol (Betapace) oral or IV</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Conversion of AF &gt; 7 days</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class I</strong></td>
</tr>
<tr>
<td>Dofetilide (Tikosyn) oral</td>
</tr>
<tr>
<td>Ibutilide (Corvert) IV</td>
</tr>
<tr>
<td>Amiodarone (Cordarone) IIa oral or IV</td>
</tr>
<tr>
<td><strong>Class IIb</strong></td>
</tr>
<tr>
<td>(Less effective or incompletely studied agents)</td>
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<tr>
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</tr>
<tr>
<td>Flecaïnide (Tambocor) oral or IV</td>
</tr>
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<td>Quinidine (Biquin) oral</td>
</tr>
<tr>
<td><strong>Class III</strong></td>
</tr>
<tr>
<td>Digoxin oral or IV</td>
</tr>
<tr>
<td>Sotalol (Betapace) oral or IV</td>
</tr>
</tbody>
</table>

**WPW AND RAPID AF**

- **AVOID** digoxin, CCBs, or beta-blockers!
- They may paradoxically accelerate the ventricular response → ventricular fibrillation.  

(JACC Vol. 48, No.4, 2006; pg e175) (10, 5th ed, pg. 1083)

- Consider Amiodarone or Procainamide (Class IIb)

**WPW = TACHYCARDIA WITH**
a. Short PR interval (less than 0.12 second)
b. QRS duration > 0.10s
c. The clinical hallmark = narrow complex supraventricular tachycardia at a rate of 150 to 300 beats/min
   [12, 5th ed, pgs 188-190]
d. Slurred upstroke to QRS complex ("delta wave")

**WPW:** PSVT in 40 to 80%, Atrial Fibrillation in 10 to 20% and Atrial Flutter in 5%

### CHA₂DS₂-VASc SCORE

In patients with nonvalvular AF, the CHA₂DS₂-VASc score is recommended for assessment of stroke risk

[http://circ.ahajournals.org/content/early/2014/04/10/CIR.0000000000000041.full.pdf]

#### Definition and Scores for CHADS₂

<table>
<thead>
<tr>
<th>CHADS₂</th>
<th>SCORE</th>
</tr>
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<tbody>
<tr>
<td>Congestive HF</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Age greater than or = to 75</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke / TIA / TE</td>
<td>2</td>
</tr>
<tr>
<td>Maximum Score</td>
<td>6</td>
</tr>
</tbody>
</table>

#### CHADS₂ acronym¹ Adjusted stroke rate( % per year)

<table>
<thead>
<tr>
<th>CHADS₂ acronym¹</th>
<th>Adjusted stroke rate( % per year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.9%</td>
</tr>
<tr>
<td>1</td>
<td>2.8%</td>
</tr>
<tr>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td>3</td>
<td>5.9%</td>
</tr>
<tr>
<td>4</td>
<td>8.5%</td>
</tr>
<tr>
<td>5</td>
<td>12.5%</td>
</tr>
<tr>
<td>6</td>
<td>18.2%</td>
</tr>
</tbody>
</table>

#### CHA₂DS₂-VASc acronym² Adjusted stroke rate( % per year)

<table>
<thead>
<tr>
<th>CHA₂DS₂-VASc acronym²</th>
<th>Adjusted stroke rate( % per year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>1</td>
<td>1.3%</td>
</tr>
<tr>
<td>2</td>
<td>2.2%</td>
</tr>
<tr>
<td>3</td>
<td>3.2%</td>
</tr>
<tr>
<td>4</td>
<td>4.0%</td>
</tr>
<tr>
<td>5</td>
<td>6.7%</td>
</tr>
<tr>
<td>6</td>
<td>9.8%</td>
</tr>
<tr>
<td>7</td>
<td>9.6%</td>
</tr>
<tr>
<td>8</td>
<td>6.7%</td>
</tr>
<tr>
<td>9</td>
<td>15.20%</td>
</tr>
</tbody>
</table>

¹These adjusted stroke rates are based on data for hospitalized patients with AF and were published in 2001 (201). Because stroke rates are decreasing, actual stroke rates in contemporary nonhospitalized cohorts might vary from these estimates.

²Adjusted stroke rate scores are based on data from Lip and colleagues (202). Actual rates of stroke in contemporary cohorts may vary from these estimates.
ANTITHROMBOTIC THERAPY FOR ATRIAL FIBRILLATION

- **AF and mechanical heart valves**: warfarin is recommended; target INR = 2.0 to 3.0 or 2.5 to 3.5 based on the type and location of the prosthesis

- **Nonvalvular AF, prior stroke, TIA or a CHA₂DS₂-VASc score > 2**: oral anticoagulants are recommended. Options include: warfarin (INR 2.0 to 3.0), dabigatran, rivaroxaban or apixaban

- **For patients with nonvalvular AF unable to maintain a therapeutic INR level with warfarin**: use of a direct thrombin or factor Xa inhibitor (dabigatran, rivaroxaban, or apixaban) is recommended.

- **Renal function** should be evaluated prior to initiation of direct thrombin or factor Xa inhibitors and should be re-evaluated when clinically indicated and at least annually

- **For patients with atrial flutter**, antithrombotic therapy is recommended according to the same risk profile used for AF.

- **CHA₂DS₂-VASc score of 1**: no antithrombotic therapy or treatment with an oral anticoagulant or aspirin may be considered.

MULTIFOCAL ATRIAL TACHYCARDIA (MAT)

1. > 3 differently shaped P waves
2. Varying PP, PR and RR intervals
3. Atrial rhythm between 100 and 180 [12]

- “Irregularly irregular” rhythm, “wandering pacemaker”, “chaotic atrial rhythm”

- Most common cause of MAT = COPD. Other causes = CHF, sepsis and theophylline toxicity.

- Treatment of MAT is directed toward underlying disorder

ATRIAL FLUTTER

Atrial Rate = 250 to 350 beats/min

The 2:1 conduction ratio accounts for the classic (although not exclusive) EKG appearance of atrial flutter as a **narrow complex tachycardia** with a regular ventricular rate usually in the high 130’s to 140’s beats/min [10, 5th ed. pgs. 1082-1083]

- Cardioversion often responds to lower energy levels then Atrial Fibrillation

- Use 50 to 100 J with either a monophasic or biphasic device

[2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, S712]
ANTIDYSRHYTHMIC DRUGS

Mnemonic: (SOme Block Potassium Channels) [ANK, provided by Dr. Alan Lazzara]

<table>
<thead>
<tr>
<th>Class</th>
<th>Blockers</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Sodium Channel Blockers (SOme)</td>
<td>Fast Na+ channel blockers → slow phase 0 depolarization in His-Purkinje and ventricular myocytes = slow conduction and membrane-stabilization</td>
</tr>
<tr>
<td></td>
<td>Class IA (Quinidine, Procainamide, Disopyramide)</td>
<td>Toxicity of Class IA, IC meds → ↑ QT → torsade de pointes</td>
</tr>
<tr>
<td></td>
<td>Class IB (Lidocaine, Tocainide, Mexiletine, Phenytoin, Aprindine)</td>
<td>Toxicity of Class IB Lidocaine = CNS disturbances (lightheadedness, confusion) CV effects (↓ BP, AV block)</td>
</tr>
<tr>
<td>2</td>
<td>Beta Blockers (BLOCK)</td>
<td>Metoprolol, Atenolol, Esmolol, Propanolol, Timolol</td>
</tr>
<tr>
<td>3</td>
<td>Potassium Channel Blockers (POTASSIUM)</td>
<td>Prolong action potential duration and refractory period duration, such that another AP cannot take place immediately following its predecessor; antifibrillatory properties</td>
</tr>
<tr>
<td></td>
<td>K+ Channel Blockers (CLASS 3)</td>
<td>Amiodarone, Dofetilide, Dronedarone, Azimilide Sotalol (non-selective beta-blocker) and Ibutilide also share activity with class II agents</td>
</tr>
<tr>
<td>4</td>
<td>Ca2+ (slow) Channel Blockers (CHANNELS)</td>
<td>Verapamil, Diltiazem</td>
</tr>
</tbody>
</table>

CAUSES OF PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Mnemonic: (MI PhD) 2 (CREW)

Rate = 130-220, usually 160; Regular

<table>
<thead>
<tr>
<th>MI</th>
<th>MI</th>
<th>MI Disease (MV Prolapse, MV stenosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MV</td>
<td>MV Disease (MV Prolapse, MV stenosis)</td>
</tr>
<tr>
<td></td>
<td>Pneumonia</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>P</td>
<td>Pericarditis</td>
<td>Pericarditis</td>
</tr>
<tr>
<td>H</td>
<td>Hypertension</td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Hyperthyroidism</td>
<td>Hyperthyroidism</td>
</tr>
<tr>
<td>D</td>
<td>Digitalis toxicity</td>
<td>Digitalis toxicity</td>
</tr>
<tr>
<td></td>
<td>Drop volume (hypovolemia)</td>
<td>Drop volume (hypovolemia)</td>
</tr>
<tr>
<td>C</td>
<td>COPD</td>
<td>COPD (much more common rhythm for COPD = MAT)</td>
</tr>
<tr>
<td>R</td>
<td>Rheumatic Heart Disease</td>
<td>Rheumatic Heart Disease</td>
</tr>
<tr>
<td>E</td>
<td>ETOH</td>
<td>ETOH</td>
</tr>
<tr>
<td>W</td>
<td>WPW</td>
<td>WPW (40-80% PSVT, 10-20% A fib, 5% Flutter) [12, pg. 159]</td>
</tr>
</tbody>
</table>
**PSVT TREATMENT OPTIONS**

[2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care]

If unstable → “synchronized” Cardioversion → 50 to 100J monophasic or biphasic [AHA: 2011]

Vagal maneuvers → Adenosine (Adenocard) 6 mg IVP → Adenosine 12 mg IVP in proximal vein (short half life); may repeat 12 mg IVP dose once → rhythm does not convert → Narrow or Wide QRS?

→ Narrow QRS consider → Diltiazem or β-blockers
→ Wide QRS consider → Procainamide or Amiodarone

Methylxanthines (Theophylline) and caffeine → antagonize Adenosine
Dipyridamole (Persantine) and Carbamazepine (Tegretol) → potentiate Adenosine

- Adenosine ultra short acting, 20 secs → AV block; converts >90% of reentrant SVT [12, pg. 148]
- Most common pediatric dysrhythmia = PSVT heart rate usually > 220 (in adults it’s less)
  Treatment = Adenosine 0.1 mg/kg (max 6 mg) → 0.2 mg/kg (max 12 mg), (may repeat x1)
- Verapamil is contraindicated in infants

**CAUSES OF PULSELESS ELECTRICAL ACTIVITY**

**Mnemonic:** (MI OD PATCH<sub>6</sub>)

<table>
<thead>
<tr>
<th>MI</th>
<th>MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>OD</td>
<td>Overdose</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>P</th>
<th>PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Acidosis</td>
</tr>
<tr>
<td>T</td>
<td>Tension Ptx</td>
</tr>
<tr>
<td>C</td>
<td>Cardiac tamponade</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>H6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypo-xia</td>
</tr>
<tr>
<td>Hypo-thermia</td>
</tr>
<tr>
<td>Hypo-glycemia</td>
</tr>
<tr>
<td>Hypo-olemia</td>
</tr>
<tr>
<td>Hypo-kalemia</td>
</tr>
<tr>
<td>Hyper-kalemia</td>
</tr>
</tbody>
</table>
TREATMENT OPTIONS: ASYSTOLE / PEA
[2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, 8.2]

- Epinephrine 1 mg IVP repeat every 3-5 minutes
- Vasopressin was removed from cardiac arrest algorithm in 2015
- Atropine was removed from the cardiac arrest algorithm in 2010
- Available evidence suggests that the routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit.

CAUSES OF PROLONGED QT ON EKG
[23, pg. 130]

- Hypo-calcemia
- Hypo-magnesemia
- Hypo-kalemia
- Hypo-thyroidism
- Hypo-thermia (also, see mnemonics: causes of ST segment elevation and Afib)
- Hereditary
- Drugs
  - TCA’s
  - Lithium
  - Phenothiazine
  - Diphenhydramine
  - Cocaine
  - Class IA, IC, III antiarrhythmics
  - Erythromycin IV
  - Zofran (ondansetron)
- Miscellaneous
  - AMI
  - ↑ICP
  - SAH (also, diffuse deep T-wave inversion with SAH)

CAUSES OF LOW VOLTAGE EKG
Mnemonic: (ME BIRP)

<table>
<thead>
<tr>
<th>M</th>
<th>Myxedema</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Effusion, pericardial</td>
</tr>
<tr>
<td>B</td>
<td>Barrel chest/obese</td>
</tr>
<tr>
<td>I</td>
<td>Improper lead placement</td>
</tr>
<tr>
<td>R</td>
<td>Restrictive cardiomyopathy</td>
</tr>
<tr>
<td>P</td>
<td>Pericarditis / Myocarditis</td>
</tr>
</tbody>
</table>
FOUR PHASES OF PERICARDITIS

[10, 5th ed, pg. 1132]

<table>
<thead>
<tr>
<th>Phase</th>
<th>Description</th>
</tr>
</thead>
</table>
| Phase 1 | ST-Segment ↑ I, V5, V6 → subepicardial ventricular injury  
ST segment in concave upward, in AMI = convex upward  
PR Depression II, aVF and V4 to V6 → subepicardial atrial injury  
Low voltage  
ST-segment ↓ aVR or V1 |
| Phase 2 | ST-Segment returns to isoelectric line, and T-wave amplitude decreases (flattens) |
| Phase 3 | T-wave ↓ inversion in leads which were previously ST ↑  
Begins at the end of the second or third week and lasts several weeks |
| Phase 4 | Resolution of repolarization abnormalities |

CAUSES OF PERICARDITIS

MNEMONIC: (AMP CARDIAC RIND) [Provided by Dr Mark Postel, with modifications]

<table>
<thead>
<tr>
<th>Letter</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Autoimmune</td>
</tr>
<tr>
<td>M</td>
<td>Myxedema</td>
</tr>
<tr>
<td>P</td>
<td>Post-traumatic (4 to 12 days post-injury)</td>
</tr>
<tr>
<td>C</td>
<td>Collagen Vascular Disease (SLE, RA, scleroderma, dermatomyositis, sarcoid, amyloid)</td>
</tr>
<tr>
<td>R</td>
<td>Aortic dissection</td>
</tr>
<tr>
<td>A</td>
<td>Radiation = most spontaneously resolve</td>
</tr>
<tr>
<td>D</td>
<td>Drug-Induced (procainamide, hydralazine, cromolyn sodium, dantrolene, methysergide)</td>
</tr>
<tr>
<td>I</td>
<td>Idiopathic = Most Common; viral = #2 most common cause of pericarditis</td>
</tr>
<tr>
<td>A</td>
<td>Acute Renal Failure</td>
</tr>
<tr>
<td>C</td>
<td>Cardiac Infarction</td>
</tr>
<tr>
<td>R</td>
<td>Rheumatic Fever</td>
</tr>
<tr>
<td>I</td>
<td>Infectious = viral (coxsackie B, echovirus, influenza, adenovirus, HIV, EBV, CMV), Staph, Strep pneumo, Strep pyogenes (acute rheumatic fever), Mycoplasma, C. trachomatis, Rickettsia, parasites, TB, Salmonella, Haemophilus influenzae</td>
</tr>
<tr>
<td>N</td>
<td>Neoplasm, mainly metastatic</td>
</tr>
<tr>
<td>D</td>
<td>Dressler syndrome = several weeks after MI</td>
</tr>
</tbody>
</table>
PERICARDITIS PEARLS

- Uremia = few EKG changes; → serous or hemorrhagic effusions; hemorrhagic effusions more common secondary to uremia-induced platelet dysfunction
  - Treatment = hemodialysis, steroids 1-2 weeks; avoid NSAIDs → bleeding

- Pericarditis is the most common cardiac manifestation of SLE in 30%
  - Most common symptom = CP which ↑ when the patient is supine, ↓leaning forward
  - Most common physical finding = pericardial friction rub

CHF CAUSES / PRECIPITATIONS FACTORS / PEARLS

Mnemonic: (HEART MISHAPS) 2 5, with modifications

Most common cause of Acute CHF = CAD 10, 5th ed., pg 1053,1115-1118
Most common causes of Chronic CHF = see below

<table>
<thead>
<tr>
<th>H</th>
<th>HTN</th>
<th>High output failure vs low output failure*</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Endocarditis</td>
<td>ETOH</td>
</tr>
<tr>
<td>A</td>
<td>Acute Anemia</td>
<td>Arrhythmias</td>
</tr>
<tr>
<td>R</td>
<td>Rheumatic fever</td>
<td>Renal failure</td>
</tr>
<tr>
<td>T</td>
<td>Thyroid (hyper)</td>
<td>Tox (Causes of NON-cardiogenic pulmonary edema → see Toxicology)</td>
</tr>
<tr>
<td>M</td>
<td>MR/MS/AR/AS</td>
<td>Myopathies (Dilated / Hypertrophic / Restrictive)***</td>
</tr>
<tr>
<td>I</td>
<td>Ischemia</td>
<td>Infection (Coxsackie B, Dipheria &amp; many more...) Chaga’s disease (Trypanosoma cruzi) leading form of CHF in Central America</td>
</tr>
<tr>
<td>S</td>
<td>Sodium load</td>
<td>Stiff ventricles (noncompliant)***</td>
</tr>
<tr>
<td>H</td>
<td>Heat</td>
<td>Humidity</td>
</tr>
<tr>
<td>A</td>
<td>pAgets</td>
<td>Anti-inflammatory (NSAIDS)</td>
</tr>
<tr>
<td>P</td>
<td>PE</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>S</td>
<td>Steroids</td>
<td>Saline overload</td>
</tr>
</tbody>
</table>

*High output failure examples include: hyperthyroid, Paget’s, pregnancy, anemia, AV fistula and beriberi (thiamine (B1) deficiency → dilated cardiomyopathy) → heart cannot meet elevated circulatory demands of conditions listed

Low output failure myocardial dysfunction prevents normal metabolic requirements being met

**Cardiomyopathies

Dilated = infective myocarditis, collagen vascular disease, EOTH, idiopathic, DM, ↑ or ↓thyroidism, uremia and beriberi

Hypertrophic obstructive cardiomyopathy (HOCM) has also historically been known as idiopathic hypertrophic subaortic stenosis (IHSS)
Restrictive = idiopathic (most common), amyloid, sarcoid and hemochromatosis

***Stiff ventricles /noncompliant - due to hypertrophy or ischemia, requires ↑LVEDP to achieve diastolic filling

CHF ON CXR

Kerley A lines: straight, non-branching lines in the mid and upper lung fields radiate towards the hila, 3 to 4cm long [8]

Kerley B lines: horizontal, non-branching lines seen laterally in lower zones never > 2 cm long

Pulmonary edema, pleural effusions (usually bilateral with right > left, if unilateral almost always right sided), cardiac enlargement without specific chamber enlargement and evidence of ↑pulmonary venous pressure (enlargement of vessel in upper lung zones “cephalization”) [8]

TREATMENT OPTIONS IN CHF
Mnemonic: (L M N O P)

| L | Lasix = 2 mechanism of action: 1) Vasodilation within few minutes 2) Diuretic response in 10-20”  
   | Dose = equal twice the patient’s daily usage, up to a maximum of 180 mg IV  
   | If the patient previously has not been on a loop diuretic, initial dose = 40mg IV  
   | If bumetanide (Bumex) is used, 1 mg of Bumetanide equals 40 mg furosemide  
   | Ethacrynic acid is useful if the patient has a serious sulfa allergy  
| M | Morphine 2-6 mg IV  
| N | Nitroglycerin 0.4 mg SL q 1-5 min, IV starting dose = 0.2-0.4mcg/kg/min  
| O | Oxygen, CPAP, intubate  
| P | Position – elevate HOB  

• CHF occurs if there is acute impairment of at least 25% of the left ventricle

• Most common symptom of left-sided failure = SOB

• The most common and earliest finding on CXR in CHF = cephalization  
CXR findings may precede symptoms

• LEFT sided failure (reduced flow into aorta and systemic circulation) → fatigue, dyspnea, orthopnea, ↑HR, ↑RR, ↑BP, ↑CO, S3 gallop, diaphoresis, rales/wheezing

• RIGHT sided failure (secondary to elevated systemic venous pressure) → JVD, ↓BP, ↓CO, RUQ pain, peripheral edema, hepatomegaly and hepatojugular reflux
ABDOMINAL AORTIC ANEURYSM (AAA)

- AAA = localized dilation of aorta, involves all three layers of aorta (intima, media and adventitia ... don’t confuse with Aortic Dissection
- Aortic Dissection = blood enters the media of the aorta and dissects the aortic wall
- Men > Female
- Average age at time of diagnosis = 65 to 70 y/o (75% of AAAs occur in patients > 60 y/o)
- AAAs have traditionally been attributed to Atherosclerosis but other factors probably contribute to their formation → biochemical abnormalities and uncertain genetic basis → Society for Vascular Surgery recommend labeling AAA as “nonspecific” rather than “atherosclerotic” [10, 6th ed., pg. 1331]
- Strong risk factor for AAA = Family history → patients with 1st degree relative & AAA have 10-20-fold ↑risk of developing AAA [10, 6th ed., pg. 1331]; other risk factors = smoking, HTN, history of CAD or PVD
- Most important risk factor for AAA rupture = size; most ruptures > 5cm [10, 6th ed., pg. 1331]
- KUB /cross-table lateral = suggest diagnosis of AAA in > 65% of symptomatic patients (Calcified aorta/loss of renal/psoas shadow) [12, 5th ed., pg. 414]
- Classic triad for ruptured AAA = ↓BP, pulsatile abdominal mass, and flank/back pain\nTriad may be incomplete in as many as 50%
- Mechanism of pain = 1. Rapid expansion of AAA or 2. Pressure of AAA on surrounding structures like nerves or 3. Presence of free blood in the abdomen or retroperitoneum from leaking or ruptured aneurysm
- Most common site AAA ruptures = Retroperitoneal 75-90% of cases (most common on left)\nMost common segment = below renal arteries\n10 to 30% rupture intraperitoneal = if intraperitoneal → rapidly fatal [10, 6th ed., pg. 1331]

AORTIC DISSECTION

- Thoracic aortic dissection is the most common lethal disease affecting the aorta
- 2-3x more common as ruptured AAA [10, 4th ed., pg 1819] [EM Reports, Vol. 21, #1]
- Major risk factor for aortic dissection = HTN [10, 5th ed., pg 1324]
  Other = pregnancy, aortic valve disease, connective tissue disease, stimulant use
- Age > 60 y/o; 3:1 male:female
- Type A (proximal) = ascending aorta +/- descending aorta = 75% cases = surgery
  Type B (distal) = confined to descending aorta = medical management
- BP difference between upper extremities of > 20 mmHg or a loss/reduction of lower extremity pulses suggests aortic dissection

CXR Findings

- Widened mediastinum (> 8cm) - most common/reliable abnormality occurs 75% of cases
- Tracheal deviation to the RIGHT
- Depressed LEFT mainstem bronchus
- LEFT apical cap
- Loss of aortic arch
• Separation (> 5mm) of intimal calcium from the outer border of the aorta
• LEFT pleural effusion (see mnemonic “causes of pleural effusions”)

**Cardiovascular** [10, 5th ed., pg 1325]
• 90% CP; Migration of pain down back or from chest to back is highly specific for dissection
• Ascending dissections → pain anterior chest
• Arch dissections → neck and jaw pain
• Descending dissections → pain in the interscapular area
• Aortic Insufficiency (diastolic murmur) = common > 50% of patients → Acute CHF
• EKG is abnormal in most patients – varying degrees of heart block, AMI
• Tamponade (blood spreads proximally to open the pericardial space to aortic blood flow)

**Neurologic**
• 9% syncope (most common cause = tamponade; other = cerebral ischemia) [10, 5th ed., pg 1325]
• Type A→ carotid artery dissection/obstruction → CVA
• Spinal artery of Adamkiewicz blockage → cord ischemia → acute paraplegia
• Horners syndrome = compression of sympathetic chain from enlarged aorta

**GI**
• Hematemesis
• Accompanied mesenteric ischemia BAD → death rate 88%

**Renal**
• Hematuria, oliguria
• Decrease blood flow → renin release → refractory ↑BP

**HYPERTENSIVE EMERGENCY**
Severe elevation in BP accompanied by acute target organ damage; treatment = parenteral therapy

\[ \text{MAP} = \text{CO} \times \text{SVR} \quad \text{MAP} = \text{DBP} + \frac{1}{3} (\text{SBP} - \text{DBP}) \quad \text{CPP} = \text{MAP} - \text{ICP} \]

**HYPERTENSIVE EMERGENCIES – TREATMENT OPTIONS**

1. HTN encephalopathy = Nitroprusside or Labetalol or Clevidipine (Cleviprex, new CCB drug, recently approved, may see but will be institution dependent)

2. Stroke syndromes = Labetalol or Nicardapine (Cardene)
   Nimodipine (Nimotop) useful in SAH, 60mg po (Note: Patients with acute ischemic stroke-in-evolution are most often not given antihypertensive drugs unless they are candidates for tissue plasminogen activator and their initial blood pressure is ≥185/110 mmHg [http://www.uptodate.com/contents/evaluation-and-treatment-of-hypertensive-emergencies-in-adults])

3. Acute coronary insufficiency = NTG
   Avoid Nitroprusside because of “coronary steal” syndrome [12, 5th ed, pg. 407]

4. Acute pulmonary edema treatment = NTG and Lasix; Drugs that increase cardiac work (eg, hydralazine) or acutely decrease cardiac contractility (eg, labetalol or other beta blockers) should be avoided [http://www.uptodate.com/contents/evaluation-and-treatment-of-hypertensive-emergencies-in-adults]
5. Aortic Dissection = Nitroprusside + esmolol or labetalol, give β-blocker first to prevent reflex tachycardia. Goal - decrease aortic pressure wave contour (dp/dt); ↓ HR 60

6. Eclampsia = Hydralazine; MgSO4 for seizures. Avoid Nipride → fetal cyanide toxicity

7. Hyperadrenergic states (MAOI, Pheochromocytoma, Anit-HTN withdrawal)
   Phentolamine + Propranolol; Nitroprusside + Propranolol; Labetalol
   Trimetaphan (ganglion blocker) if Beta Blocker contraindication

Do not drop BP too quickly. Ischemic damage can occur in vascular beds that have grown accustomed to the higher level of blood pressure (ie, autoregulation). For most hypertensive emergencies, mean arterial pressure should be reduced by about 10 to 20 percent in the first hour and then gradually during the next 23 hours so that the final pressure is reduced by approximately 25 percent compared with baseline.[http://www.uptodate.com/contents/evaluation-and-treatment-of-hypertensive-emergencies-in-adults]

**Dosing**
- Labetalol = 20mg IV over two minutes – repeat or double every 10min to max of 300mg; Infusion rate 1 to 2 mg/min up to 8 mg/min
- Nicardipine (Cardene) = 5mg/hr, increase infusion 2.5mg/hr every 10 min to a max of 15mg/hr
- Nitroprusside = start at 0.5 mcg/kg/min IV infusion

**Hypertensive Urgency**
Severe elevations in BP (DBP > 115), with mild or no acute target organ damage; may reduce BP within hours to days usually with oral medications

**HTN without Emergency / Urgency = does not mandate urgent therapy**
- Essential HTN = chronic problem and referral to PMD will greatly enhance management of HTN
- Treatment of HTN as an outpatient basis is usually not the responsibility of EM doc [10, 5th ed. 1166-1177]

**CARDIOLOGY PEARLS**
- Most common complications of Anterior Wall MI = Mobitz type II, ventricular aneurysm, CHB (unlike CHB with IWMI, CHB with AWMI = grave prognosis) [12, 5th ed., pg. 363]
- Most common complications of Inferior Wall MI = Acute MR, ↓HR and ↓BP
  First degree AVB, Mobitz type I (Wenckebach, accounts for 90% of 2nd-degree AVB and AMI) and CHB (stable, usually resolves) [12, 5th ed., pg. 363]
- VF occurs in 5% of patients with AMI, 80% present within 12 hours [10, 6th ed., pg. 1160]
- Most common cause of VT/VF = AMI / ischemia
- Low serum potassium, but not magnesium, has been associated with ventricular arrhythmias in AMI
  Maintain serum potassium >4 mEq/L and magnesium >2 mEq/L [2010 AHA Guidelines for Cardio Resusc and Em Cardiovascular Care]
- Most common conduction disturbance in AMI = First degree AV block, 15%; more common with IWMI
• Mortality rate with IWMI and CHB = 15%; if RV involved MR rises to > 30% [12, 5th ed., pg. 363]

• Most common rhythm disturbances in AMI = PVC’s (>90%), PACs (50%) Sinus tach (33%) [12, 5th ed., pg. 362]

• % of patients with AMI who have diagnostic changes on their first EKG = 50% [12, 5th ed., pg. 359]

• Most common complications of thrombolytic therapy in patients with AMI = Reperfusion arrhythmias; accelerated idioventricular rhythm most common arrhythmia

• Most common rhythm disturbance in digitals toxicity = PVC’s 60% > SVT 25% > AV block 20%

• Earliest EKG findings associated with AMI = hyperacute T → giant’ R wave (“tombstone”) → typical ST segment elevation – which is either flat (horizontally or oblique) or convex

• ST-segment elevation in V1, in the absence of ST-segment elevation in the other anteroseptal leads (V2-V3), is suggestive of right-ventricular ischemia/infarction

• Dressler’s syndrome = fever, pleuritis, leukocytosis, pericardial friction rub, and evidence of pericarditis or pleural effusion occurring several weeks after MI. Autoimmune. Treatment = NSAIDs or ASA [10, 6th ed., pg. 1283]

• LV dysfunction > 40%= Cardiogenic shock [10, 6th ed., pg. 47]

• Cardiogenic shock may not occur immediately post-MI. The median delay from AMI to clinical development of cardiogenic shock may be up to 7 hours

• Most common type of CVA after MI = ischemic thromboembolic [10, 6th ed., pg. 1161]

• Most common permanent pacer dysfunction = oversensing

**CARDIOLOGY PEARLS - INFECTIVE ENDOCARDITIS**

• In patients with rheumatic heart disease, the mitral valve is the most common site of IE involvement

• Most common cause of Native Valve Infective Endocarditis (IE) with valvular or Congenital HD =
  - Strep viridans (30-40%)
  - Staph aureus + S. epidermidis = 20-35%,
  - “Other” Strep (15-25%)
  - Enterococci (5-18%)  
  - Treat = Pen G or AMP + Nafcillin + Gentamycin [Sanford, 2009, pg. 25]; Most textbooks = AMP + Gent
  - Gentamycin for synergy; Nafcillin does not cover Enterococci
  - or Vancomycin + Gentamycin [Sanford, 2009, pg. 25]

• Most common cause of Native Valve Right-Sided IE = Staph aureus (IVD abusers)
  Most common valve affected = tricuspid; Treatment = Vancomycin [Sanford, 2009, pg. 25]

• Culture negative IE = Haemophilus parainfluenzae, H. aphrophilus, Actinobacillus, Cardiobacterium, Eikenella, and Kingella species (HACEK organisms); Treatment = Ceftriaxone 2gm IV q 24hrs or Amp + Gent
Add *Bartonella henselae*, *B. quintana* = (HABCEK); Treatment = Ceftriaxone + Gentamycin + Doxy
Also, *Coxiella burnetii* Treatment = Doxy + Hydroxychloroquine  
[Sanford, 2009, pg. 27] and  
[10, 6th ed., pg. 1301]

- Most common cause of Prosthetic Valve IE < 60 days post-op = coagulase negative Staph. (*S. epidermidis*) and *Staph aureus*. Treat = Vancomycin + Gentamycin + Rifampin  
[Sanford, 2009 pg. 28]

- Most common cause of Prosthetic Valve IE > 60 days post-op = similar to Native Valve with Staph aureus now most common etiology  
[Sanford, 2009 pg. 28, JAMA 297:1354,2007]

- Association between *Streptococcus bovis* IE and coexisting GI malignancy  
[10, 6th ed., pg. 1301]

- IE = petechiae, splinter hemorrhages (dark red vertical lesions in nailbeds), Osler nodes (painful, red, raised lesions on distal finger pads), Janeway lesions 35% (flat, red-bluish, painless lesions on palms/soles), fever, murmur, anemia, malaise, Roth spots (retinal hemorrhages with pale center)
  Fever is the most common presenting symptom

- Most common cause of Acute Aortic Regurgitation (AR) = Infective endocarditis

- In developed countries, the frequency of RHD has declined, and MVP is now the most common underlying condition in patients with endocarditis

**INFECTIVE ENDOCARDITIS**

Mnemonic **(FROM JANE)**

| **F** | Fever (most common presenting symptom) |
| **R** | Roth spots (retinal hemorrhages with pale center) |
| **O** | Osler nodes (painful, red, raised lesions on distal finger pads) |
| **M** | Murmur |
| **J** | Janeway lesions 35% (flat, red-bluish, painless lesions on palms/soles) and petechiae |
| **A** | Anemia |
| **N** | Nailbeds - splinter hemorrhages (dark red vertical lesions in nailbeds) |
| **E** | Emboli |
CARDIOLOGY PEARLS - VALVULAR HEART DISEASE

- Most common cause of Chronic Aortic Regurgitation (AR) = Rheumatic heart disease (RHD)
- Most common cause of Aortic Stenosis < 70 y/o = Congenital Heart Disease (Bicuspid Valve 50%), 2nd RHD
- Most common cause of Aortic Stenosis > 70 y/o = Idiopathic calcification/degen. heart disease [10, 5th ed. pg. 1155]
- Most common cause of Acute MR = Rupture of chordae tendineae or papillary muscle (IWMI)
- Most common cause of Chronic MR = Rheumatic heart disease (RHD)
- Most common cause of Mitral Stenosis (MS) = Rheumatic heart disease (RHD)
- Most common presenting symptom of all cardiac valvular diseases = exertional dyspnea
- Most common symptom of Mitral Stenosis (MS) = Exertional Dyspnea (symptom specific to MS = hemoptysis)
- Aortic Stenosis (AS) = exertional Syncope, Angina and Dyspnea (mnemonic=SAD)
- Most common rapidly lethal complication of Aortic Stenosis (AS) = sudden death
- Most common valvulopathy due to chest trauma = Aortic Regurgitation (AR)
- Aortic Regurgitation (AR) = High Pulse Pressure, head bobbing = prominent ventricular impulse (Musset sign), soft diastolic murmur (Austin-Flint murmur), bounding peripheral pulses (water hammer), pulsations of uvula and nailbed, and SBP of LE > UE (Hill sign); causes: trauma, IE, aortic dissection, Marfan’s, syphilis
- Systolic murmurs = AS (mid-systolic ejection crescendo-decrescendo) and MR (holosystolic)
- Diastolic murmurs = AR (blowing decrescendo) and MS (holodiastolic with opening snap) [http://www.wilkes.med.ucla.edu/Systolic.htm]
- MS, MR, Pulmonary Insufficiency = decrease pulse pressure (AR high pulse pressure) [10, 5th ed., pg 1155]
- Most common symptoms of MVP = CP (sharp, localized) & palpitations; affects 10% of population; majority asymptomatic; EKG abnormal; patients may have pectus excavatum or scoliosis; ↑ migraines, anxiety and CVA
  ↑ incidence of sudden death and dysrhythmias. ↑ incidence of TIA’s under the age of 45; MVP with regurgitation (both leaflets involved; usually affects one – the posterior leaflet) → ↑ risk of IE [10, 5th ed., pg. 1153]
- MVP murmur = mid-systolic click (snapping of chordae tendineae during prolapse of valve) followed by late systolic crescendo murmur heard best at apex or LSB with patient in left lateral decub; If ↓ preload (↓EDV) (valsalva, standing position) → click moves closer to S1 → ↑ previously unheard click, murmur is longer not louder; handgrip → ↑ louder murmur [10, 5th ed., pg. 1153]
- Valsalva → ↑intrathoracic pressure → ↓venous return → ↓preload → ↓most murmurs except for Hypertrophic Cardiomyopathy murmur which ↑ because dynamic LV outflow obstruction is accentuated by ↓preload

- Hypertrophic Cardiomyopathy murmur = harsh mid-SEM crescendo-decrescendo; loud S4; heard best at apex & left sternal border; does not radiate to neck (AS radiates to carotids) \[10, 5th ed., pg. 1144\]
  ↓ preload (standing, valsalva diuretics or nitrates) or ↓ afterload (vasodilators) → ↑gradient → ↑murmur

**CARDIOLOGY PEARLS**

- Amount of fluid in normal pericardial space = 25-50 ml
- Need 250 ml of fluid in pericardial space before cardiac silhouette ↑ on CXR
- Beck’s Triad: 1) Muffled heart tones 2) ↓BP 3) JVD (↑CVP) = cardiac tamponade
- Most common echocardiographic findings with cardiac tamponade = right ventricular diastolic collapse
- Electrical alternans on EKG is pathognomonic for tamponade
- Most common cause of pericardial tamponade = malignancy 30-60%; uremia 10-15%, idiopathic pericarditis 5-15%, ID 5-10%, anticoagulation 5-10%, connective tissue diseases 2-6%, and Dressler syndrome 1-2%
- Causes of pulsus paradoxus = cardiac tamponade, and obstructive lung disease (asthma, COPD)
- Pulsus Paradoxus = measuring the variation of SBP during expiration and inspiration \[10, 5th ed., pg 398\]

  Slowly decrease cuff pressure until systolic sounds are first heard during expiration but not during inspiration, (note this reading)
  Slowly continue decreasing the cuff pressure until sounds are heard throughout the respiratory cycle, (inspiration and expiration) = note this second reading
  If the pressure difference between the two readings is >10mmHg = pulsus paradoxus
- Most common cause of restrictive cardiomyopathy = idiopathic \[10, 5th ed. pg. 1145\]
  Other causes = amyloid, sarcoid and hemochromatosis
- Most common infectious cause of myocarditis = Coxsackie B
  Consider CMV & Toxoplasma gondii in Transplant or HIV patient
  Kawasaki = 50% myocarditis
  Chaga’s disease (Trypanosoma cruzi) = leading cause of death in Central America; 3/4 no symptoms
  Spread by the reduvid, “kissing” or “assassin” bug
  Pathognomonic finding in Chaga’s = Romaña sign = painless unilateral periorbital edema; uncommon
  Chagoma = painful cutaneous edema at the site of skin penetration
  Treatment = Nifurtimox (Lampit) or Primaquine
- Shock = imbalance of tissue O2 supply and demand;
  All patients with shock should receive as the first priority → Supplemental oxygen
• Four mechanistic classifications of shock \[^{12, \text{5th ed., pg 215}}\]
  1) Hypovolemic (inadequate circulatory volume)
  2) Cardiogenic (inadequate cardiac pump function)
  3) Distributive (Peripheral vasodilation and maldistribution of blood flow); examples →
      neurogenic shock (↓BP and ↓HR), anaphylactic shock, pancreatitis, burns, trauma, adrenal
      insufficiency, drug or toxin reactions, heavy metal poisoning, hepatic insufficiency
  4) Obstructive (extra-cardiac obstruction to blood flow) cardiac tamponade, PE, tension pneumothorax

• Pure alpha adrenergic agent = phenylephrine

• Mixed alpha and beta adrenergic agents = epinephrine, norepinephrine and dopamine

• Pure beta or primary beta-agonists = dobutamine and isoproterenol

• S1Q3 RAD = Left Posterior Fascicular (LPFB)
  S3Q1 LAD = Left Anterior Fascicular (LAFB)

• LPFB has more serious implications since it implies compromise to both the right and left coronary
  arteries as well as damage to large areas of myocardial muscle and to the electrical conduction system
  in the left ventricle \[^{\text{Sensible analysis of the 12-lead ECG By Kathryn Monica Lewis, Kathleen A. Handal, pg. 170}}\]
### RISK FACTORS FOR DVT / PULMONARY EMBOLISM

**Mnemonic: (MOIST CAMEL)** [3, w/modification]

<table>
<thead>
<tr>
<th><strong>M</strong></th>
<th><strong>O</strong></th>
<th><strong>I</strong></th>
<th><strong>S</strong></th>
<th><strong>T</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mobility = Stasis (prolonged travel, bed rest, paralysis (CVA, spinal cord injury), leg cast</td>
<td>Obesity</td>
<td>Inflammatory Conditions (IBD, SLE, PVD)</td>
<td>Surgery (especially orthopedic, pelvic &amp; major abdominal surgery)</td>
<td>Trauma = Intimal damage (trauma, IVDA, surgery, central lines)</td>
</tr>
</tbody>
</table>

### VIRCHOW’S TRIAD

1) Hypercoagulable state  
2) Venous stasis  
3) Venous injury

### HYPERCOAGULABLE (THROMBOPHILIA) STATES

- Previous DVT/PE  
- Nephrotic syndrome (loss of antithrombin)  
- Malignancy  
- Inflammatory Conditions (IBD, SLE, PVD)  
- Sepsis

### COAGULATION DISORDERS – INHERITABLE VS ACQUIRED

- Protein C or S deficiency  
- Resistance to activated Protein C  
- Antithrombin deficiency  
- Disorders of Fibrinogen or Plasminogen  
- Antiphospholipid antibodies (lupus anticoagulant & anti-cardiolipin)

### INCREASED ESTROGEN (CAUSES URINARY LOSS OF PROTEIN S AND ANTITHROMBIN)

- Pregnancy  
- Postpartum status < 3 months  
- OCPs  
- Elective abortion or miscarriage
**D-DIMER**

Fibrin fragments found in fresh fibrin clot & in fibrin degradation products. Elevated in many conditions (poor Sensitivity 77%) including = DVT/PE, CVA, trauma, cancer, surgery, infection/sepsis, sickle cell anemia, postpartum within 1 week and pregnancy (elevated in 75% of patients with a normal pregnancy)

**WELLS CLINICAL SCORE FOR DVT**

<table>
<thead>
<tr>
<th>Clinical Parameter Score</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active cancer (treatment ongoing, or withing 6 mo. or palliative)</td>
<td>+1</td>
</tr>
<tr>
<td>Paralysis or recent plaster immobilization of the lower extremities</td>
<td>+1</td>
</tr>
<tr>
<td>Recently bedridden for &gt;3 days or major surgery &lt; 4wks.</td>
<td>+1</td>
</tr>
<tr>
<td>Localized tenderness along the distribution of the deep venous system</td>
<td>+1</td>
</tr>
<tr>
<td>Entire leg swelling</td>
<td>+1</td>
</tr>
<tr>
<td>Calf swelling &gt;3 cm compared with the asymptomatic leg</td>
<td>+1</td>
</tr>
<tr>
<td>Previous DVT documented</td>
<td>+1</td>
</tr>
<tr>
<td>Collateral superficial veins (non varicose)</td>
<td>+1</td>
</tr>
<tr>
<td>Alternative diagnosis (as likely or greater than that of DVT)</td>
<td>-2</td>
</tr>
</tbody>
</table>

**Total of Above Score**

<table>
<thead>
<tr>
<th>Score</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>High probability</td>
<td>&gt;3</td>
</tr>
<tr>
<td>Moderate probability</td>
<td>1 or 2</td>
</tr>
<tr>
<td>Low probability</td>
<td>0</td>
</tr>
</tbody>
</table>

**D-DIMER + WELLS CLINICAL SCORE FOR DVT- DIAGNOSTIC STRATEGY**

A negative D-dimer result in the unlikely group (Wells DVT score < 2) ----> rules out DVT
All patients with a positive D-dimer result and all patients in the likely group (Wells DVT score > 2) ----> require a diagnostic study (duplex Ultrasonography)

**ANTICOAGULATION TREATMENT OPTIONS FOR VENOTHROMBOEMBOLISM (VTE) - DVT/PE**

1. Injectable indirect factor Xa and IIa (thrombin) inhibitors
   The effects of indirect inhibitors are mediated through antithrombin (AT)
   a. Unfractionated heparin (UFH); Xa and to a lesser extent IIa (thrombin) Also, inhibits factors XIIa, Xia and IXa
   b. Enoxaparin (Lovenox), low-molecular-weight-heparin (LMWH); Xa and to a lesser extent IIa (thrombin) inhibitor
   c. Fondaparinux (Arixtra); exclusive indirect factor Xa inhibitor

2. Target-Specific Oral Anticoagulants (TSOACs) or Direct Oral Anticoagulants (DOACs)
   a. Direct Thrombin Inhibitors (DTIs)
      i. Dabigatran (Pradaxa)
   b. Direct Factor Xa Inhibitors (“Xabans”)
      i. Rivaroxaban (Xarelto)
      ii. Apixaban (Eliquis)
      iii. Edoxaban (Lixiana, Savaysa)
VTE PROPHYLAXIS

UFH 5,000 units SQ every 8 hours

Enoxaparin (Lovenox)
- 40 mg SQ once daily or 30 mg SQ once daily if CrCl < 30 mL/min

Fondaparinux (Arixtra)
- 2.5 mg SQ daily if CrCl > 30 mL/min and actual body weight > 50kg

Rivaroxaban (Xarelto) - Surgical VTE Prophylaxis
- Knee replacement - 10 mg once daily for 12 days
- Hip replacement - 10 mg once daily for 35 days
- Initial dose should be taken at least 6-10 hours after surgery once hemostasis has been established
- Avoid use if CrCl < 30 mL/min

Apixaban (Eliquis) - Surgical VTE Prophylaxis
- Knee replacement – 2.5mg BID for 12 days
- Hip replacement - 2.5mg BID for 35 days
- Initial dose should be given 12 to 24 hours after surgery once hemostasis has been established
- No dose adjustment with moderate renal impairment for above Apixaban VTE indications
- (not studied in patients with CrCl < 25 mL/min)

VTE TREATMENT

UFH Bolus 80 units/kg followed by infusion of 18 units/kg/hr

Enoxaparin (Lovenox)
- 1 mg/kg SC every 12 hours or
- 1.5 mg/kg SC daily - “suggested over twice-daily regimen”

Fondaparinux (Arixtra)
- Weight <50 kg: 5 mg SQ daily
- Weight 50-100 kg: 7.5 mg SQ daily
- Weight >100 kg: 10 mg SQ daily

ARIXTRA is contraindicated:
- Creatinine clearance <30 mL/min
- DVT prophylaxis if weight < 50 kg

Rivaroxaban (Xarelto)
- 15 mg BID x 21 days
- On day #22 transition to
- 20 mg once daily

Reduce Risk of Recurrent DVT or PE
- Following 6 months of treatment
- 20 mg once daily
Apixaban (Eliquis)
- 10 mg BID x 7 days, then 5 mg BID

Reduce Risk of Recurrent DVT or PE
- Following 6 months of treatment
- 2.5 mg BID

Edoxaban (Lixiana, Savaysa)
- >60 kg: 60 mg daily
- <60 kg: 30 mg daily
- Treat with parenteral anticoagulation for 5-10 days (dual therapy)
- CrCl 15 to 50 mL/min: decrease dose to 30 mg daily

Dabigatran (Pradaxa)
- 150 mg BID
- Treat with parenteral anticoagulation for 5-10 days (dual therapy)
- Avoid use CrCl < 30 mL/min

TREATMENT STRATEGIES FOR MAJOR BLEEDING FROM TSOACS

<table>
<thead>
<tr>
<th>Direct Factor Xa Inhibitors (Rivaroxaban, Apixaban, Edoxaban)</th>
<th>Direct Thrombin Inhibitors (Dabigatran (Paradaxa))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider charcoal - YES</td>
<td>Consider Charcoal - YES</td>
</tr>
<tr>
<td>Consider Anti-Fibrinolitics - YES</td>
<td>Consider Anti-Fibrinolitics - YES</td>
</tr>
<tr>
<td>Consider Hemodialysis - NO</td>
<td>Consider Hemodylasis - YES</td>
</tr>
<tr>
<td>Consider non-activated PCC (Kcentra)</td>
<td>Consider activated PCC (FEIBA)</td>
</tr>
<tr>
<td></td>
<td>If antidote available - consider Praxbind (Idarucizumab)</td>
</tr>
</tbody>
</table>

Praxbind (Idarucizumab)
Idarucizumab is a humanized, monoclonal, antibody fragment that specifically binds with high affinity to dabigatran. Dabigatran has an affinity for idarucizumab that is 350 times greater than its affinity for thrombin.

Andexanet Alfa
Antidote in pipeline for oral direct and injectable indirect Factor Xa Inhibitors. Andexanet alfa is a modified recombinant factor Xa molecule administered intravenously. Antidote to reverse the anticoagulant activity of oral direct (apixaban, edoxaban, and rivaroxaban) and injectable indirect (enoxaparin and fondaparinux) factor Xa inhibitors. Andexanet alfa acts as a decoy to target and sequester with high specificity both oral and injectable factor Xa inhibitors.
ANTICOAGULATION CASCADE
Contact Activation (intrinsic) Pathway

MECHANISM OF ANTICOAGULATION

Unfractionated heparin
Low-molecular-weight heparins
Vitamin K antagonists
Direct thrombin inhibitors
Factor Xa inhibitors
WELL'S CRITERIA FOR ASSESSMENT OF PRETEST PROBABILITY FOR PE

<table>
<thead>
<tr>
<th>Clinical Signs and Symptoms of DVT</th>
<th>3 points</th>
</tr>
</thead>
<tbody>
<tr>
<td>An alternative diagnosis in less likely than PE</td>
<td>3 points</td>
</tr>
<tr>
<td>Heart Rate &gt; 100</td>
<td>1.5 points</td>
</tr>
<tr>
<td>Immobilization at least 3 days, or Surgery in the Previous 4 weeks</td>
<td>1.5 points</td>
</tr>
<tr>
<td>Previous, objectively diagnosed PE or DVT</td>
<td>1.5 points</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>1 point</td>
</tr>
<tr>
<td>Malignancy w/ Treatment within 6 months, or palliative</td>
<td>1 point</td>
</tr>
</tbody>
</table>

Low-risk patients, score < 2 had a mean probability of 3.6% for PE
Intermediate-risk patients, score 2-6 had a mean probability of 20.5% for PE
High-risk patients, score > 6 had a mean probability of 66.7% for PE

Modified Wells Criteria PE
If < 4 Proceed to PERC
If > 4 CT angio or V/Q

PULMONARY EMBOLISM RULE-OUT CRITERIA (PERC)

- Age >= 50y
- Tachycardia, HR > 100
- O2 sat < 95%
- Prior DVT or PE
- Recent trauma or surgery
- Hemoptysis
- Exogenous estrogen (hormone use)
- Unilateral leg swelling

If NO criteria are met, PE can be ruled out
If any criteria are met, unable to rule out PE

PERC RULE
MNEMONIC - (HAD CLOTS)

The patient must meet the following:

- H: no Hormones
- A: no Age > 50
- D: no DVT/PE history
- C: no Coughing blood
- L: no Lower extremity swelling unilaterally
- O: no O2 saturation <95
- T: no Tachycardia >100
- S: no Surgery/trauma within past 28 days
PULMONARY EMBOLISM - PEARLS

- Most Common **Symptom** = dyspnea, 90% > CP\(^{[10, 6th ed., pg 1371-1372]}\)
- Most Common **Sign** = tachypnea, 70%
- Triad of Dyspnea, CP and Hemoptysis < 20%
- Most common cause of death from pulmonary thromboembolism = hemodynamic collapse\(^{[10]}\)
- Most common **EKG** finding = Non specific ST / T Wave Changes
  (50% of patients diagnosed with PE have a HR < **100** beats/min \(^{[12, 6th ed., pg 388]}\)

EKG FINDINGS IN PULMONARY EMBOLISM

Mnemonic - (A STRIPS)

<table>
<thead>
<tr>
<th>A</th>
<th>Atrial fibrillation, new onset (see mnemonic AFIB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>S1Q3T3</td>
</tr>
<tr>
<td>T</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>R</td>
<td>RAD, RBBB</td>
</tr>
<tr>
<td>I</td>
<td>Inverted T-wave in V1-V4</td>
</tr>
<tr>
<td>P</td>
<td>Pulmonale (peaked P waves in lead II)</td>
</tr>
<tr>
<td>S</td>
<td>ST segment elevation</td>
</tr>
</tbody>
</table>

The CXR is most often abnormal in PE

- Conflicting literature:
  Most common CXR finding from PIOPED trail = atelectasis
  Most common CXR finding from UPET (Urokinase PE Trail) = elevated hemidiaphragn

- Most common CXR in admitted patients = cardiomegaly\(^{[12, 6th ed., 390]}\)
- Most common CXR in outpatient patients = basilar atelectasis\(^{[12, 6th ed., 390]}\)
- Other CXR findings: Hampton’s hump, (wedge-shaped consolidation in lung periphery), Westermark’s sign, (dilation of proximal arteries with collapse of distal vasculature)

PULMONARY EMBOLISM - PEARLS

- **ABG** = acute respiratory alkalosis, hypoxemia, an abnormal AaO2 gradient or normal
- **A-a gradient** = should be widened; Sn 90%, Sp 15%
- A-a gradient = 150 – **PaO2** – (**PaCO2** x 1.25)
  Normal = 5-20 (calculate on room air)
• Lack of hypoxemia does NOT rule out diagnosis of PE

• Hypoxia helps with risk stratification → correlates with degree of pulmonary vasculature occlusion from PE → shown to predict poor outcome

[12, 6th ed., 389]

• Consider spiral CT of chest, V/Q, or pulmonary angiography

• ECHO = right heart strain in 40%
  McConnell’s sign = RV hypokinesis in the presence of normal RV apical contractility

PULMONARY EMBOLISM – PEARLS PREGNANCY

• Threshold for human teratogenesis = 10 rad; fetus most vulnerable 8 to 15 weeks gestation

[12, 6th ed., pg. 675]

• V/Q — Total fetal exposure to xenon-133 and technetium-99m = 0.5 rad; void bladder x 3

• CXR = 0.00005 rad; CT head < 0.1 rad, CT chest = < 1 rad; CT abdomen = 3.5 rad

[12, 6th ed., pg. 675]

• British Journal of Radiology (2006) [79, 441-444] recommends:
  D-dimer → positive → bilateral lower extremity venous doppler → non-diagnostic
  → CT chest over V/Q to diagnose PE

TREATMENT OPTIONS IN PULMONARY EMBOLISM


• **Heparin**: bolus (80U/kg or 5,000 Units) then infusion (18U/kg/hr or 1,300 u/hr)

• In massive PE consider 10,000 U bolus, followed by a continuous intravenous infusion of at least 1,250 U/h (however 80U/kg bolus followed by 18U/kg/hr, infusion acceptable)

• **LMWH**: Enoxaparin (Lovenox) = 1mg/kg SQ or IV every 12 hours or 1.5mg/kg q 24 hours

  Circulation. 2005;112:e28-e32 Management of Massive Pulmonary Embolism did not mention Lovenox as a treatment option for “Massive” PE

• **tPA** = You will find three different protocols for tPA
  ▫ 100 mg over 2 hours (FDA approved regimen, most textbooks) or
  ▫ 15 mg bolus, then 85 mg continuous infusion over 2 hours or
  ▫ Accelerated infusion regimen used in AMI
Hold **heparin** during fibrinolytic infusion. At the conclusion of alteplase infusion begin heparin infusion without a bolus when aPTT has decreased to < 80 seconds.

- **Hypotension**
  - 0.9% NS cautiously because ↑ RV wall stress can → ↓ the ratio of RV oxygen supply to demand → may result in ischemia, deterioration of RV function, and worsening RV failure and → further ↑ interventricular septal shift toward the left ventricle, thereby worsening left ventricular compliance and filling. Consider only 500 to 1,000 cc
  - Norepinephrine and dobutamine → permits increased myocardial contractility, while minimizing both vasodilation and the risk of hypotension

- Morphine 4 to 6 mg IV PRN

**Massive PE** = arterial hypotension + cardiogenic shock
- Hypotension: SBP < 90 mm Hg or drop in SBP of at least 40 mm Hg for at least 15 minutes
- Shock = tissue hypoperfusion & hypoxia, altered LOC, oliguria, or cool, clammy extremities

### TREATMENT OPTIONS IF MAJOR BLEEDING STARTS AFTER THROMBOLYTIC THERAPY

- Stop the lytic agent
- FFP and cryoprecipitate ASAP
- Aminocaproic acid (Amicar) Inhibits plasminogen activators - 5 gm bolus infused over 1 hr then infusion of 1 gm/hr until bleeding has stopped; Rapid administration may result in hypotension, bradycardia and arrythmias. [10, 5th ed., pg. 1229]

### TREATMENT OPTIONS IF MAJOR BLEEDING STARTS AFTER HEPARIN THERAPY

- Most common cause of drug related death in hospitalized patients = heparin
- Antidote = Protamine = 1 mg neutralizes 100 units of heparin; administer over 15 minutes. Empiric 25 to 50 mg IV [12, 6th ed., pg. 1017]

### CAUSES OF DYSPNEA

**Mnemonic: (SPACE) 2** [19, with modifications]

Dyspnea = a subjective shortness of breath with abnormal and uncomfortable awareness of breathing [10, pg. 1030]

<table>
<thead>
<tr>
<th>S</th>
<th>Spontaneous pneumothorax</th>
<th>Shock ↑ hemodynamic stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>Pneumonia</td>
<td>Pulmonary edema (CHF/ARDS)</td>
</tr>
<tr>
<td>A</td>
<td>Asthma</td>
<td>Atelectasis</td>
</tr>
<tr>
<td>C</td>
<td>COPD (bronchitis/emphysema)</td>
<td>Cardiac (CHF, AMI, valvular, AS, MS, AMR)</td>
</tr>
<tr>
<td>E</td>
<td>Embolism-pulmonary (PE)</td>
<td>Effusions (pulmonary)</td>
</tr>
</tbody>
</table>
CAUSES OF PLEURAL EFFUSIONS  
**Mnemonic: B (CHAMPS) 3** [1, with modifications] [10, 5th ed., pg. 1005]

<table>
<thead>
<tr>
<th>B</th>
<th>Boerhaave syndrome (left sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>CHF Most common cause of effusions in Western countries</td>
</tr>
<tr>
<td>H</td>
<td>Hemorthorax</td>
</tr>
<tr>
<td>A</td>
<td>AFB positive = TB Most common cause of effusions developing countries</td>
</tr>
<tr>
<td>M</td>
<td>Malignancy</td>
</tr>
<tr>
<td>P</td>
<td>Pneumonia “parapneumonic effusion”</td>
</tr>
<tr>
<td>S</td>
<td>SLE (also RA) 12% pts SLE = Exud Effusion</td>
</tr>
</tbody>
</table>

| C | Cirrhosis (with ascites) |
| H | Hypothyroidism |
| A | Asbestos |
| M | METS |
| P | PE (with infarct) |
| S | Saline overload |

| H | Chylothorax |
| H | Hepatic infection (with upward spread) |
| A | ↓ Albumin |
| S | Side effect of drugs (NSAIDS, etc.) (see below) |

CAUSES OF PLEURAL EFFUSIONS - SIDE EFFECTS OF DRUGS  
**Mnemonic: (MAP)**

| M | Macrodantin, sustained release = Macrobid (Nitrofurantoin) |
| A | Apresoline (Hydralazine) |
| P | Procainamide (Pronestyl, Procan) |

All three drugs above (MAP), Dilantin or INH, can all cause **Lupus-Like Syndrome**  
Amiodarone (Cordarone) causes pulmonary toxicity by pulmonary fibrosis
PLEURAL EFFUSION PEARLS

Transudative pleural effusions

- Protein content < 3 gm/dl
- Pleural fluid vs. Serum protein RATIO < 3
- Pleural fluid vs. Serum LDH RATIO < 0.6
- LDH content less than 200

Transudative pleural effusions = CHF, cirrhosis, starvation (↓albumin), constrictive pericarditis, nephrotic syndrome and SVC obstruction

Exudative effusions have high amounts of protein and LDH; pH < 7.3
Examples = pneumonia, SLE, TB and malignancy

Exudates – Exceed (high amounts of protein and LDH effusion to serum ratios)
Effusion protein/serum protein > 0.5
Effusion LDH/serum LDH > 0.6

Pleural effusions: Need 200 cc of fluid to be seen on PA or lateral CXR
Need <50 cc of fluid if lateral decubitus film

CAUSES OF HEMOPTYSIS

Mnemonic: (BIC) 3 [1, with modifications]

<table>
<thead>
<tr>
<th>Common Causes</th>
<th>Less Common Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>Bbronchitis</td>
</tr>
<tr>
<td>I</td>
<td>Infection (TB, lung abscess, bronchitis, pneumonia)</td>
</tr>
<tr>
<td>C</td>
<td>Cancer</td>
</tr>
</tbody>
</table>

B - Bronchiectasis

I - Idiopathic Cryptogenic hemoptysis
Common = 28%

C - Cardiac (CHF, MS)
CHF = 75% of cardiac cases

Cocaine “Crack lung”

Other causes
Alveolar hemorrhage syndromes: Bechet, Goodpasture syndrome and Wegener granulomatosis
Hematologic: platelet dysfunction, anticoagulant therapy and uremia (platelet dysfunction)
Cystic Fibrosis
Trauma

Epidemiology (in industrialized countries) [12, 6th ed., pg., 465]
Idiopathic = “cryptogenic hemoptysis” = 28%
Neoplasm = 28%
Infectious, non-TB = 25%
Miscellaneous = 13%
TB = 5%

Mild Hemoptysis < 20 ml in 24hrs; Moderate = 20 to 600ml; Severe = > 600 ml [12, 6th ed., pg., 465]

TB = cough is the most common presenting symptom, not hemoptysis; hemoptysis is usually minor in acute infection; later → major cause of massive hemoptysis [10, 5th ed., pg., 1906]
SOB is unusual; 15% get extrapulmonary manifestations
Aspergilloma as a superinfection with TB can also cause massive hemoptysis \(^{[10, 5th ed., pg., 1907]}\)

HIV patient with hemoptysis = consider Strep. pneumo. or TB \(^{[10, 5th ed., pg., 1847]}\)

Children with TB, most common CXR findings = hilar adenopathy, mediastinal lymphadenopathy or consolidated pneumonia

**Evaluation**\(^{[12, 6th ed., pg., 466]}\)
CXR = abnormal in 70-85%; if neoplasm → abnormal in 80-90%
Massive bleeding = rigid bronchoscopy
Less severe bleeding = fiberoptic bronchoscopy
CT chest

**COMMUNITY ACQUIRED PNEUMONIA (CAP)**
[Clinical Infectious Diseases 2007;44:S27-S72]
The initial treatment of CAP is **empiric** and must cover these 6 bugs

**“Typical” pathogens**
- *Streptococcus pneumoniae*
- *Moraxella catarrhalis*
- *Haemophilus influenzae, nontypeable*

**“Atypical” pathogens = not detectable on GS or cultivatable on standard bacteriologic media**
- *Mycoplasma pneumoniae*
- *Chlamyphilia pneumoniae*
- *Legionella*

**MILD (AMBULATORY) CAP**
[Clinical Infectious Diseases 2007;44:S27-S72]
The most common pathogens →
*Strep. pneumoniae, M. pneumoniae, C. pneumoniae, and H. influenzae*

- *Mycoplasma* = most common among patients <50 years of age without significant comorbid conditions or abnormal vital signs, whereas *Strep. pneumoniae* was the most common pathogen among older patients and among those with significant underlying disease.
- *Hemophilus* infection was found in 5%—mostly in patients with comorbidities
- The use of fluoroquinolones to treat ambulatory patients with CAP without comorbid conditions, risk factors for DRSP, or recent antimicrobial use is discouraged because of concern that widespread use may lead to the development of fluoroquinolone resistance.

**COMMUNITY ACQUIRED PNEUMONIA (CAP)**
[Clinical Infectious Diseases 2007;44:S27-S72]
- Blood cultures are low yield = 5 to 14%
- Blood cultures are **optional** for all hospitalized patients with CAP but should be performed selectively
Get blood cultures if severe CAP, immunocompromised or if your yearly merit increase is affected by a hospital core measure list which includes blood cultures for CAP.
- There is strong evidence for the recommendation of **combination** empirical therapy for non-ICU and ICU
TREATMENT FOR NON-ICU – CAP

- Ceftriaxone or Cefotaxime or Unasyn or Ertapenem (Invanz) + Macrolide
- A respiratory fluoroquinolone can be used for penicillin-allergic patients in non-ICU CAP

TREATMENT FOR ICU – CAP

- Therapy with a respiratory fluoroquinolone alone is not established for severe ICU CAP
- Ceftriaxone or Cefotaxime or Unasyn plus either azithromycin (level II evidence) or a respiratory fluoroquinolone (level I evidence) (strong recommendation)
- For penicillin-allergic patients → respiratory fluoroquinolone + aztreonam

Pneumonia Severity Score for Elderly Patients

<table>
<thead>
<tr>
<th>CHARACTERISTIC</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Historical Findings</strong></td>
<td></td>
</tr>
<tr>
<td>Age - Men</td>
<td></td>
</tr>
<tr>
<td>Men (years)</td>
<td></td>
</tr>
<tr>
<td>Women (years - 10)</td>
<td></td>
</tr>
<tr>
<td>Nursing home resident</td>
<td>10</td>
</tr>
<tr>
<td>Coexisting disease</td>
<td></td>
</tr>
<tr>
<td>Neoplastic disease</td>
<td>30</td>
</tr>
<tr>
<td>Liver disease</td>
<td>20</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>10</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>10</td>
</tr>
<tr>
<td>Renal disease</td>
<td>10</td>
</tr>
<tr>
<td><strong>Physical Examination Findings</strong></td>
<td></td>
</tr>
<tr>
<td>Altered mental status (acute)</td>
<td>20</td>
</tr>
<tr>
<td>Respiratory rate &gt; 30</td>
<td>20</td>
</tr>
<tr>
<td>Systolic BP &lt; 90 mmHg</td>
<td>20</td>
</tr>
<tr>
<td>Temperature &lt; 35°C or &gt; 40°C</td>
<td>15</td>
</tr>
<tr>
<td>Pulse &gt; 125/min</td>
<td>10</td>
</tr>
<tr>
<td><strong>Diagnostic Testing Findings</strong></td>
<td></td>
</tr>
<tr>
<td>Arterial pH &lt; 7.35</td>
<td>30</td>
</tr>
<tr>
<td>BUN &gt; 30 mg/dL</td>
<td>20</td>
</tr>
<tr>
<td>Sodium &lt; 130 mmol/L</td>
<td>20</td>
</tr>
<tr>
<td>Glucose &gt; 250 mg/dL</td>
<td>10</td>
</tr>
<tr>
<td>Hematocrit &lt; 30%</td>
<td>10</td>
</tr>
<tr>
<td>PaO2 &lt; 60 mmHg (or SaO2 &lt; 90%)</td>
<td>10</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>10</td>
</tr>
</tbody>
</table>
Fine and colleagues developed a **prognostic model**, the **Pneumonia Severity Index (PSI)**, for thirty day **mortality** in patients with CAP. Patients are assigned to one of five risk classes (1=lowest risk and 5=highest risk) based upon a point system. Outpatient management is suggested for Class 1 and 2, brief inpatient for class 3 and traditional hospitalization for Classes 4 & 5. Severe pneumonia may require intensive care unit (ICU) admission. Guidelines should not supersede clinical judgment.

**SEVERITY OF ILLNESS SCORE**

**Mnemonic: (CURB 65)** *(Sanford, 2009, pg. 36; AmJ 118:384,2005)*

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>C</strong></td>
<td>Confusion</td>
<td>1 Point</td>
<td></td>
</tr>
<tr>
<td><strong>U</strong></td>
<td>Uremia - BUN &gt; 19 mg/dl</td>
<td>1 Point</td>
<td></td>
</tr>
<tr>
<td><strong>R</strong></td>
<td>Respiratory rate - RR &gt; 30/min</td>
<td>1 Point</td>
<td></td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Blood pressure, low - BP &lt; 90/60</td>
<td>1 Point</td>
<td></td>
</tr>
<tr>
<td><strong>65</strong></td>
<td>Age &gt; 65</td>
<td>1 Point</td>
<td></td>
</tr>
</tbody>
</table>

If score = 1, outpatient ok; If > 1 hospitalize. The higher the score the higher the mortality.

**IDSA / ATS GUIDELINES ICU ADMISSION DECISION** *(Clinical Infectious Diseases 2007;44:S27-S72)*

**Major Criteria**
- Mechanical ventilation or
- Shock (SBP < 90 mmHg) requiring pressors

**Minor Criteria – presence of 3 criteria → admit ICU**
- Respiratory rate > 30 breaths/min at admission
- Arterial oxygen pressure / fraction inspired oxygen (PaO2/FI02) < 250 mmHg
- Multilobar infiltrates
- Confusion / disorientation
- BUN > 20 mg/dL
- Leukopenia from infection (< 4,000 cells/mm3)
- Thrombocytopenia (<100,000 cells/mm3)
- Hypothermia < 36°C
- Hypotension requiring aggressive fluid resuscitation
EPIDEMIOLOGIC CONDITIONS AND/OR RISK FACTORS RELATED TO SPECIFIC PATHOGENS IN CAP

(Clinical Infectious Diseases 2007;44:S27-S72)

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>COMMONLY ENCOUNTERED PATHOGEN(S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholism</td>
<td><em>Streptococcus pneumoniae</em>, oral anaerobes, <em>Acinetobacter</em> species, <em>Mycobacterium tuberculosis</em></td>
</tr>
<tr>
<td>COPD and/or smoking</td>
<td><em>Haemophilus influenzae</em>, <em>Pseudomonas aeruginosa</em>, <em>Legionella</em> species, <em>S. pneumoniae</em>, <em>Moraxella catarrhalis</em>, <em>Chlamydia pneumoniae</em></td>
</tr>
<tr>
<td>Aspiration</td>
<td>Gram-negative enteric pathogens, oral anaerobes</td>
</tr>
<tr>
<td>Lung abscess</td>
<td><em>CA-MRSA</em>, oral anaerobes, endemic fungal pneumonia, <em>M. tuberculosis</em>, atypical mycobacteria</td>
</tr>
<tr>
<td>Exposure to bat or bird droppings</td>
<td><em>Histoplasma capsulatum</em></td>
</tr>
<tr>
<td>Exposure to birds</td>
<td><em>Chlamydia psittaci</em> (if poultry: avian influenza)</td>
</tr>
<tr>
<td>Exposure to rabbits</td>
<td><em>Francisella tularensis</em></td>
</tr>
<tr>
<td>Exposure to farm animals or parturient cats</td>
<td><em>Coxiella burnettii</em> (Q fever)</td>
</tr>
<tr>
<td>HIV infection (early)</td>
<td><em>S. pneumoniae</em>, <em>H. influenzae</em>, <em>M. tuberculosis</em></td>
</tr>
<tr>
<td>HIV infection (late)</td>
<td>The pathogens listed for early infection plus <em>Pneumocystis jirovecii</em>, <em>Cryptococcus</em>, <em>Histoplasma</em>, <em>Aspergillus</em>, atypical mycobacteria (especially <em>Mycobacterium kansasi</em>), <em>P. aeruginosa</em>, <em>H. influenzae</em></td>
</tr>
<tr>
<td>Hotel or cruise ship stay in previous 2 weeks</td>
<td><em>Legionella</em> species</td>
</tr>
<tr>
<td>Travel to or residence in SW United States</td>
<td><em>Coccidioides</em> species, Hantavirus</td>
</tr>
<tr>
<td>Travel to or residence in SE and East Asia</td>
<td><em>Burkholderia pseudomallei</em>, avian influenza, SARS</td>
</tr>
<tr>
<td>Influenza active in community</td>
<td><em>Influenza</em>, <em>S. pneumoniae</em>, <em>Staph aureus</em>, <em>H. influenzae</em></td>
</tr>
<tr>
<td>Cough &gt;2 weeks with whoop or posttussive V</td>
<td><em>Bordetella pertussis</em></td>
</tr>
<tr>
<td>Structural lung disease (e.g., bronchiectasis)</td>
<td><em>Pseudomonas aeruginosa</em>, <em>Burkholderia cepacia</em>, <em>S. aureus</em></td>
</tr>
<tr>
<td>Injection drug use</td>
<td><em>S. aureus</em>, anaerobes, <em>M. tuberculosis</em>, <em>S. pneumoniae</em></td>
</tr>
<tr>
<td>Endobronchial obstruction</td>
<td>Anaerobes, <em>S. pneumoniae</em>, <em>H. influenzae</em>, <em>S. aureus</em></td>
</tr>
<tr>
<td>In context of bioterrorism</td>
<td><em>Bacillus anthracis</em> (anthrax), <em>Yersinia pestis</em> (plague), <em>Francisella tularensis</em> (tularemia)</td>
</tr>
</tbody>
</table>
**PULMONARY PEARLS**

- Adequate sputum specimen must have > 25 PMN’s and < 10 squamous epithelial cells
- Most common cause of bacterial pneumonia = *Strep pneumoniae* \(\checkmark\) rusty/blood sputum
- Most common non-bacterial pneumonia in adults < 50 y/o = *Mycoplasma pneumonia*
- Neonatal pneumonia = GBS, *E. coli* and *Klebsiella*
- Pneumonia 1-3 months = Chlamydia, CMV, Pneumocystis
- Most common cause of pneumonia in children < 6 months and 3-5 years old = RSV and parainfluenza. Children > 5 y/o and young adults *Mycoplasma* is common
- Most common cause of viral pneumonia in adults = *Influenza*
  - *Strep pneumoniae* = most common cause of bacterial pneumonia …. However \(\rightarrow\) also consider *Staph* pneumonia during outbreaks of influenza.
  - *Staph* pneumonia = necrotizing with cavitations and pneumatically formation. \([10, 5th ed., pg., 987]\)
- The majority of deaths from viral pneumonia occur during winter months
- Most common source of infection in septic patient = respiratory system \([10, 5th ed., pg., 1961]\)
- Organisms that cause pneumonias which present with fever and relative bradycardia = Legionella, Q fever, Psittacosis and Tularemia (note: all 4 also ↑LFTs) \([12, 6th ed., pg., 971]\)
- Most common pathogens to generate cavitary lesions = TB, anaerobic bacteria, aerobic gram-negative bacilli, *S. aureus*, and fungal disease \([10, 5th ed., pgs 990-993]\)
- Most common complication of cavitary TB = endobronchial spread (seen on x-ray as 5 to 10 mm poorly defined nodules clustered in dependent portions of the lungs) \([10, 5th ed., pg. 1907]\)
- TB = world’s leading cause of infectious death; 1/3 of world population is infected \([10, 5th ed., pg 1903]\)
- PPD often negative in late HIV; minimum triple drug therapy for TB, if HIV + TB four drugs \([10, 5th ed., pg 1848]\)
- Carcinoma suspected on CXR \(\rightarrow\) remember L.A. (Los Angeles) is located on the periphery of the US \(\rightarrow\) Large cell and Adenocarcinoma present as peripheral masses on CXR; Squamous cell and Small cell (most malignant) present as central or hilar tumors.
- Most common cause of cancer deaths in males and females = bronchogenic CA
- Paraneoplastic syndromes = ↑Na+/ SIADH, ↑Ca2+/ parathormone, ↓Ca2+/ Calcitonin, ↑ACTH/ Cushing syndrome, gynecomastia/ gonadotropins, ↑Serotonin/ Carcinoid syndrome)
• CXR findings of COPD = flat diaphragm, ↑AP diameter, enlarged retrosternal space, blebs

• If you suspect chest pain is due to pneumothorax, ask for inspiratory and **expiratory** views on chest film

• Albuterol β 2 agonist ↑ **cAMP** → bronchodilation

• Atrovent is an anticholinergic agent that ↓ **cGMP** → bronchodilation

• Epi 1:1000 IM in the thigh (vastus lateralis) results in higher and more rapid maximum plasma concentrations

• Most common occupational lung disease worldwide = Silicosis (found in sand, granite, sandstone, flint, slate, and in coal and metallic ores. Cutting, breaking, crushing, drilling, grinding, or abrasive blasting of these materials may produce fine silica dust
  ▫ Fever, SOB, CP, Cor pulmonale and **cyanosis**
  ▫ Susceptible to TB (silicotuberculosis) suspect silica damages pulmonary macrophages, inhibiting their ability to kill mycobacteria.
  ▫ Silicosis is an irreversible condition with no cure. Treatment = Lung transplantation
CAUSES OF HEMATURIA

Mnemonics: (IN STITCHES) [ANK]

| I | Infection (pyelonephritis, hemorrhagic cystitis, prostatitis, TB, Schistosomiasis) |
| N | Necrosis = Papillary Necrosis (NSAIDs, DM, sickle cell disease) |

| S | Stones, Kidney  
Stenosis of the urethra |
| T | Toxins (direct kidney injury or rhabdo)  
Toxemia of pregnancy |
| I | Intrinsic Kidney Disease (glomerulonephritis, PKD, Medullary Sponge, IgA nephropathy (Berger disease), hereditary nephritis (Alport syndrome)  
Iatrogenic (post procedure) |
| T | Trauma  
Thrombosis of renal vein |
| C | Coagulopathy (Hemophilia, ↑coumadin or heparin toxicity)  
Cancer (renal, bladder, prostate, Wilms’ tumor/nephroblastoma) |
| H | Hemoglobinopathy (sickle cell disease)  
Huge prostate (BPH) |
| E | Endocarditis  
Expanding AAA may erode into urogenital tract; also Aortic Dissection |
| S | SLE (50% nephritis) other immunologic disease = (ITP, HSP (IgA complexes), Goodpasture syndrome, Wegener granulomatosis, polyarteritis nodosa (PAN); nonimmune causes = TTP, HUS  
Serum sickness |

Don’t confuse Berger with Buergers’s disease (thromboangiitis obliterans) = recurring inflammation and thrombosis of small and medium arteries and veins of the hands and feet. Strongly associated with smoking

Berger, Buergers, Bugger, Burger say that fast 20x

Triad of sinusitis, pulmonary infiltrates, and nephritis = Wegener granulomatosis
**KIDNEY STONES**

3 x more common males > females

**Calcium oxalate or Ca phosphate stones**
- Most common 75%; radio-opaque
- Causes
  - Hypercalciuria → GI = reabsorption; Bone = resorption (primary hyperPTH); Kidney = Ca2+ leak
  - Idiopathic, sarcoid, hyper-thyroidism,
  - Paget's disease, increased incidence from genetic predisposition, PUD taking antacids, IBD

**Magnesium-ammonium-phosphate (Staghorn or struvite stones)**
- 15%, associated with alkaline urine (pH > 7.6),
- secondary to recurrent infections: Proteus, Pseudomonas, Klebsiella
- Urea → split to NH3 by Urease → binds H+ → NH4+

**Uric acid**
- 10%, radiolucent, associated with acidic urine; 25% patients with gout get stones

**Cystine stones**
- 1%, 2 0 inborn error in metabolism → ↑secretion of cystine → form staghorns

**Medications That Can Cause Stones**
- Indinavir (Crixivan) - radiolucent stones; HIV med, protease inhibitor

**Kidney Stone Pearls**
- 90% stones < 4 mm pass spontaneously
- 50% stones 4 to 6mm pass spontaneously
- 10% stones > 6mm pass spontaneously

- Diagnostic test = non-contrast spiral CT – may find alternate diagnosis …. AAA or appy consider Ultrasound to evaluate for hydronephrosis/ hydroureret in patients with known history of kidney stones
- Most common location of stone at first diagnosis = distal ureter 70%
- Most common site = ureterovesical junction (UVJ)
- Treatment: Fluid, NSAIDs decrease ureteral spasm, narcotics and Tamsulosin (Flomax)

**NEPHRITIC SYNDROME**

Mnemonics: (PHARAOH) [ANK, Mnemonic provided by Dr. Justino Dalio]

<table>
<thead>
<tr>
<th>P</th>
<th>Proteinuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hematuria</td>
</tr>
<tr>
<td>A</td>
<td>Azotemia</td>
</tr>
<tr>
<td>R</td>
<td>RBC casts</td>
</tr>
<tr>
<td>A</td>
<td>Immunoglobulin A (IgA) nephropathy glomerulonephritis (Berger disease) = Most common cause of glomerulonephritis worldwide</td>
</tr>
<tr>
<td>O</td>
<td>Oliguria</td>
</tr>
<tr>
<td>H</td>
<td>HTN</td>
</tr>
</tbody>
</table>
TESTICULAR TORSION

- Absent cremasteric reflex
- Pain may be constant, intermittent but is not positional
- Manually detorsion of testicle → stand at patient’s feet → detorsion in similar fashion to opening a book → patient’s right testicle → rotated counterclockwise; left testis → clockwise
- Manually detorsion is not curative, attempt while waiting for surgery [10, 5th ed., pg. 1423]
- If you cannot exclude torsion by H&P and imaging studies → surgery for scrotal exploration, regardless of time since onset of symptoms [12, 6th ed., pg. 617]
- “Blue-dot sign” on testicular exam = Torsion of the testicular appendix

PENILE FRACTURE

- Traumatic rupture of the tunica albuginea and corpus cavernosum urologic emergency.
- Sudden blunt trauma of the penis in an erect state causes rupture in one or both corpora.
- Concomitant urethral injury may also occur.
- Clinical diagnosis: Patients usually hear a “popping” or “cracking” sound, followed by significant pain, immediate flaccidity and skin hematoma/ecchymosis.
- Conservative management has fallen out of favor because of high complication rates.
- Surgical repair is necessary to expedite relief of pain and to prevent potential complications → surgical evacuation of hematoma and suture apposition of the disrupted tunica albuginea [12, 6th ed., pg. 617]
- Complications of conservative management include: missed urethral injury, penile abscess, nodule formation at the site of rupture, permanent penile curvature, painful erection, painful coitus, erectile dysfunction, corporal urethral fistula, arterial venous fistula and fibrotic plaque formation.

PRIAPISM

Engorgement of the corpora cavernosa. Ventral corpora spongiosum and glans penis usually remain flaccid

Two types
- **Low-flow (ischemic) priapism** (most common)
  - Veno-occlusion; painful
  - Most common cause = Sickle cell anemia
- **Arterial (non-ischemic) high-flow** priapism
  - Secondary to a rupture of a cavernous artery.
  - Rare; usually not painful; Causes = penetrating penile trauma or a blunt perineal injury

Other Causes of Low-flow priapism

- Medications
Injectable medications to induce an erection = papaverine, phentolamine (non-selective alpha-1, alpha-2 adrenergic-receptor antagonist), and prostaglandin E1
Psychotropic medications = chlorpromazine, trazodone, quetiapine, thioridazine and citalopram (SSRI)
Rebound hypercoagulable state with anticoagulants = heparin and warfarin
Hydralazine, metoclopramide, omeprazole, hydroxyzine (atarax; vistaril), prazosin, tamoxifen, and androstenedione
Cocaine, marijuana, ecstasy, and ethanol

• Neurologic
  Spinal cord injury and anesthesia
  Cauda equina syndrome

• Neoplastic
  Primary or metastatic; leukemia and multiple myeloma

• Infection
  Mycoplasma pneumoniae (Mechanism is thought to be a hypercoagulable state induced)
  Malaria

Treatment Low-flow (vaso-occlusive) priapism [12, 6th ed., pg. 616]
Terbutaline 0.25 to 0.5 mg SQ in deltoid, may be repeated in 20 to 30 min
Oral pseudoephedrine, 60-120 mg orally due to its alpha-agonist effect (sympathomimetic amine → ↑endogenous norepinephrine from storage vesicles in presynaptic neurons)
Next line of therapy = aspiration of corpus cavernosum first and injection of alpha-1-adrenergic agent phenylephrine (Neo-Synephrine)
In adults, phenylephrine should be diluted with normal saline to provide a final concentration of approximately 100 mcg to 500 mcg per mL [http://www.uptodate.com/contents/priapism]
Phenylephrine (Neo-Synephrine) 10mg/mL which = 10,000 mcg/mL
Dilute 10mg/mL Phenylephrine in 100mL of normal saline =
100 mcg/mL of Phenylephrine (Neo-Synephrine) solution → draw up 1 to 5 mL to get recommended treatment dose of 100 mcg to 500 mcg per mL

Treatment High flow
Observation alone may be sufficient as erectile function is usually unimpaired.
Compression therapy may be successful in certain cases, especially children

UROLOGY PEARLS - TRAUMA
• Most common urologic injury = renal; 80% have concurrent injuries
• Most common renal injury = contusion 90% (Grade I)
• Renal injuries are Graded I to V; order CT with IV contrast
  Surgery: Uncontrolled hemorrhage, penetrating injuries, avulsed major renal vessel, extensive urine extravasation, Grade V injury (shattered kidney with avulsed hilum)
• Most common urethral injury = posterior (90%), above the urogenital diaphragm, associated with pelvic fractures (high riding or absent prostate on rectal exam)
• Anterior urethral injury = 10%, direct trauma (kicks or straddle injuries)

• Blood at meatus = urethral trauma, no foley without first performing retrograde urethrogram

• Retrograde urethrogram = Toomey syringe into urethral meatus → inject 60 ml contrast over 30 to 60 seconds → xray during the last 10ml contrast injection

• Retrograde cystogram / retrograde CT cystography = after normal retrograde urethrogram → allow 300 to 400 ml contrast to flow by gravity from a Toomey syringe through a Foley catheter into bladder → clamp foley → AP and lateral films or CT of bladder taken → unclamp Foley → postevacuation film / CT

• Bladder injuries
  ▫ Contusion = most common bladder injury → incomplete tear of bladder mucosa; large hematomas can alter bladder shape on cystogram = “pear-shaped” bladder
  ▫ Extraperitoneal bladder rupture → heal spontaneously in 14 days with Foley
  ▫ Most common type of bladder rupture 80% (rupture usually at bladder neck)
  ▫ Intraperitoneal bladder rupture (injury is in the dome posteriorly, the only portion of dome covered by the peritoneum) → surgical repair [12, 6th ed., pg. 1626]

UROLOGY PEARLS

• Most common cause of hematuria in females = infection

• Most common cause of hematuria in men = BPH → urinary retention

• Most common cause of hematuria worldwide = Schistosomiasis → Schistosoma haematobium (blood fluke) S. mansoni & S. japonicum = esophageal varices; Dx = eosinophilia and identification of eggs in first-morning urine or stool or biopsy; Treatment = Praziquantel

• Degree of hematuria after blunt trauma does not correspond to degree of injury [12, 6th ed., pg. 1622]

• Pseudo-hematuria = blood tinged but no RBC’s on UA consider myoglobinuria (from trauma, seizures, burns, sepsis – any cause of rhabdo), pyridium, rifampin, porphyria

• Spontaneous complete drainage of distended bladder ok, no need to clamp. Transient gross hematuria or hypotension is usually insignificant [12, 6th ed., pg. 620]

• If urinary retention is chronic or insidious → postobstructive diuresis; observe 4 to 6 hours

• Fournier’s gangrene = surgical debridement, fluid and antibiotic cover aerobic/anaerobic per [Sanford, 2009, pg. 52]

• Acute hydrocele is most likely associated with = testicular cancer (7-25% of patients)

• Acute Right-sided varicocele is most likely associated with = IVC thrombosis or compression of IVC by tumors

• Acute Left-sided varicocele is most likely associated with = Renal cell CA or obstruction of left renal vein
Paraphimosis inability to reduce the retracted foreskin over the glans; ice, manual decompression; dorsal slit
Phimosis inability to retract the foreskin to visualize the glans; topical steroids 4-6 weeks; circumcision
Balanitis inflammation of the glans penis; Candida 40%, Group B Strep., Gardnerella and anaerobes; occurs in 1/4 male sex partners of women infected with candida
Treatment = Fluconazole 150 mg po x 1 [Sanford, 2009, pg. 24] or antifungal creams [12, 6th ed., pg. 615]
Posthitis inflammation of the foreskin; consider adding 1st generation cephalosporin for bacterial infection
Balanoposthitis is inflammation of both the glans penis and surrounding foreskin
Recurrent Balanoposthitis can be the sole presenting symptom of = diabetes mellitus

UROLOGY PEARLS – STDS
[CDC STD UPDATE 2010]
Epididymitis [Sanford, 2009, pg. 24]
- < 35 y/o GC (ceftriaxone 250 mg IM) and Chlamydia (doxy 100 mg po bid x 10 day not zithro x1)
- > 35 y/o E. coli (quinolone x 10 to 14 days)
- Prehn’s sign = scrotal elevation → ↓ pain in epididymitis and not in torsion; know sign for test then forget it, since insensitive to distinguish from epididymitis from testicular torsion

Chancroid
- Haemophilus ducreyi, gram-negative bacillus = painful chancres (Do Cry with H. ducreyi)
- High rates of HIV infection among patient who have chancroid
- 10% of persons who have chancroid acquired in the US are co-infected with T. pallidum or HSV
- Treatment
  - Azithromycin 1 gm orally
  - Ceftriaxone 250 mg IM in a single dose
  - Ciprofloxacin 500 mg twice daily x 3 days

Granuloma Inguinale (Donovanosis)
- Calymmatobacterium granulomatis, gram-negative pleomorphic bacillus
- Beefy-red, velvety, painless ulcer with rolled border
- Diagnosis = organism within macrophages (Donovan bodies) in biopsy specimens taken from the advancing edge of the ulceration. Macrophages engulf clusters of organisms that look like microscopic safety pins = Donovan bodies
- Treatment = Doxy 100 mg po bid x 3 weeks minimum

Lymphogranuloma Venereum
- Chlamydia trachomatis serotypes L1-3
- Painless papules, vesicles, or ulcers
- Typically unilateral, painful inguinal lymphadenopathy (“groove” sign)
- Treatment = Doxy 100 mg po bid or Erythromycin base 500 mg four times daily x 3 weeks

Syphilis is Divided into Clinical Stages
<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>Painless chancre</td>
</tr>
<tr>
<td>Secondary</td>
<td>9-90 d, after the onset of the chancre; maculopapular rash, + palms/soles</td>
</tr>
<tr>
<td>Latent</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Late</td>
<td>Manifestations 10-20 yrs following primary infection CNS, CV, skin, and/or bone may be involved</td>
</tr>
</tbody>
</table>
• **Diagnosis** - corkscrew-like spirochetes under dark field microscopy; serologic tests (VDRL, RPR) = non-specific

• **Treatment** - Primary, Secondary, Early Latent = Benzathine Penicillin G, 2.4 million units IM

• Jarisch-Herxheimer reaction = acute onset of fever, rigors, and possibly hypotension may occur within 24 hours of initiating treatment

• Condyloma lata = raised, flat, grayish papular lesions which are found in moist areas of the body including the anus, vulva, and scrotum; seen in secondary syphilis

**Trichomoniasis**

• Parasite *Trichomonas vaginalis*

• Treatment = Metronidazole 2 gm orally in a single dose

• Diagnosis = saline microscopy of vaginal secretions on wet slide prep (motile, flagellated trichomonads)

• Sensitivity = 60-70%

• Immunochromatographic capillary flow dipstick technology, and a nucleic acid probe test performed on vaginal secretions; more sensitive than vaginal wet preparation, however ↑false positives

• Culture if need definitive diagnosis

**Bacterial Vaginosis (BV)**

• Polymicrobial infection

• Clue cells on saline microscopy (bacteria adhered to vaginal epithelial cells)

• Metronidazole 500 mg twice daily for 7 days

• All symptomatic pregnant women with BV should be treated, regardless of trimester

• Woman’s response to therapy and the likelihood of relapse or recurrence **not** affected by treatment of sex partner
DIALYSIS CRITERIA

Mnemonic: (AEIOU)\[^{[AWK]}\]

Note: Mnemonic AEIOU also used in “Causes of Coma”

<table>
<thead>
<tr>
<th>A</th>
<th>Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Electrolyte abnormalities (↑K+)</td>
</tr>
<tr>
<td>I</td>
<td>Ingestion of toxins (Isopropanol, Barbiturates, Amphetamines, INH, Lithium, Theophylline, Ethylene glycol, ASA, methanol)</td>
</tr>
<tr>
<td>O</td>
<td>Overload-fluid</td>
</tr>
<tr>
<td>U</td>
<td>Uremic Pericarditis</td>
</tr>
</tbody>
</table>

INDICATIONS FOR DIALYSIS OF TOXINS

Mnemonic: (I  BAIL TEAM)

Dialysis can BAIL your medical TEAM out of a life threatening situation

<table>
<thead>
<tr>
<th>I</th>
<th>Isopropanol (isopropyl alcohol, rubbing alcohol; note: normal AG ↑ osmolar gap) [^{[12, 4th ed. pg 769]}]</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>Barbiturates → long acting (charcoal hemoperfusion); Beta blockers (water soluble = atenolol)</td>
</tr>
<tr>
<td>A</td>
<td>Amphetamines</td>
</tr>
<tr>
<td>I</td>
<td>INH</td>
</tr>
<tr>
<td>L</td>
<td>Lithium (level &gt; 4 mEq/L or 2-4 if poor clinical condition)</td>
</tr>
<tr>
<td>T</td>
<td>Theophylline (charcoal hemoperfusion)</td>
</tr>
<tr>
<td>E</td>
<td>Ethylene glycol (levels &gt; 20 mg/dL; nephrotoxicity; metabolic acidosis is present; Triad = history, clinical presentation &amp; lab results consistent with Ethylene glycol poisoning)</td>
</tr>
<tr>
<td>A</td>
<td>ASA (if seizures, CNS alteration, acidosis, serum levels &gt; 100mg/dl)</td>
</tr>
<tr>
<td>M</td>
<td>Methanol (visual or CNS dysfunction; methanol levels &gt; 20 mg/dL; ingestion &gt; 30 ml; pH &lt; 7.15) Mushrooms = Amanita (charcoal hemoperfusion)</td>
</tr>
</tbody>
</table>

Note: dialysis has NOT been shown to be effective in Acetaminophen, BZP, Clonidine, Digoxin, Dilantin \[^{[12, pg 810]}\], MAOI OD, Heroin (↑ Vd), Organophosphate poisonings \[^{[12, pg 826]}\] TCA OD-highly bound and high volume of distribution; Iron \[^{[12, 6th ed., 1123]}\]

Drugs which can be dialyzed have:
- Small molecular weight
- Small volume of distribution
- Water Solubility
- Lack protein binding
# Toxicology

## Mnemonics & Pearls

### OVERDOSES WHERE BICARB MAY BE A TREATMENT OPTION

**Mnemonic:** (LCD BITS)

| L | Lamictal (lamotrigine) |
| C | Cocaine overdose: treat if wide-complex tachycardia (Ref 29, 2010 AHA pg. 57) |
| D | Diphenhydramine Darvon = propoxyphene (similar to type IA antidysrhythmic agents) |
| B | Barbiturates - Long acting only, eg phenobarbital and barbital; note: butalbital found in Fioricet is short-acting, 25% of phenobarbital is excreted in urine; alkaline urine pH → ↑ excretion |
| I | INH (Isoniazid); treatment = pyridoxine (B6) |
| T | TCAs: treat if wide-complex tachycardia, hypotension and ventricular arrhythmias |
| S | Salicylates: favors the formation of ionized salicylate → ↓ ASA reabsorption → ↑ exertion |

### CHARCOAL INEFFECTIVE

**Mnemonic:** (MP LICE)

| M | Metals-heavy (Treatment = whole bowel irrigation = PEG (polyethylene glycol)) |
| P | Petroleum distillates (Hydrocarbons) |
| L | Lithium |
| I | Iron |
| C | Caustics |
| E | Ethylene glycol (Also → ethanol, methanol and isopropyl alcohol) |

### WHOLE BOWEL IRRIGATION

**Mnemonic:** (SLIMS)

| S | Sustained release |
| L | Lithium |
| I | Iron |
| M | Metals (heavy) |
| S | Stuffers |
RADIOPAQUE SUBSTANCES
Mnemonic: *(BET A CHIP)*  \[ANK/13\]

<table>
<thead>
<tr>
<th>B</th>
<th>Barium</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Enteric coated ASA</td>
</tr>
<tr>
<td>T</td>
<td>TCA’s</td>
</tr>
<tr>
<td>A</td>
<td>Antihistamines</td>
</tr>
<tr>
<td>C</td>
<td>Chloral hydrate</td>
</tr>
<tr>
<td>H</td>
<td>Heavy metals (Iron, lead, etc. Note: MVI with Iron-not seen on x-ray)</td>
</tr>
<tr>
<td>I</td>
<td>Iodine</td>
</tr>
<tr>
<td>P</td>
<td>Phenothiazine → Examples: chlorpromazine (Thorazine), prochlorperazine (Compazine), mesoridazine (Serentil), thioridazine (Mellaril) and promethazine (Phenergan)</td>
</tr>
</tbody>
</table>

CAUSES OF NON-ANION GAP ACIDOSIS
Mnemonic: *(HARD CUP)*  \[ANK\]

<table>
<thead>
<tr>
<th>H</th>
<th>Hyperalimentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Acetazolamide (Diamox)</td>
</tr>
<tr>
<td>R</td>
<td>RTA (proximal)</td>
</tr>
<tr>
<td>D</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>C</td>
<td>Cholestyramine</td>
</tr>
<tr>
<td>U</td>
<td>Uterosigmoidostomy</td>
</tr>
<tr>
<td>P</td>
<td>Pancreatic fistulas</td>
</tr>
</tbody>
</table>

(Causes of ↓ AG = Acetazolamide, Ammonium CI, Bromide, Iodide, Lithium, Polymyxin B, Spironolactone, Sulindac)

CAUSES OF ANION GAP ACIDOSIS
Mnemonic: *(CAT MUDPILES)*  \[ANK\]

<table>
<thead>
<tr>
<th>C</th>
<th>CO, CN (inhibit cytochrome oxidase a-a3 → ↑ lactate)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Alcoholic ketoacidosis</td>
</tr>
<tr>
<td>T</td>
<td>Toluene (secondary) to acidic metabolites</td>
</tr>
<tr>
<td>M</td>
<td>Methanol, Metformin</td>
</tr>
<tr>
<td>U</td>
<td>Uremia</td>
</tr>
<tr>
<td>D</td>
<td>DKA</td>
</tr>
<tr>
<td>P</td>
<td>Paraldehyde</td>
</tr>
<tr>
<td>I</td>
<td>INH (isoniazid, inhibits lactate ↔ pyruvate, therefore → ↑ lactate) Iron (hypovolemia and anemia → tissue hypoperfusion → ↑ lactate)</td>
</tr>
<tr>
<td>L</td>
<td>Lactic acidosis</td>
</tr>
<tr>
<td>E</td>
<td>Ethylene glycol</td>
</tr>
<tr>
<td>S</td>
<td>Salicylates</td>
</tr>
</tbody>
</table>
Salicylates
- Inhibit Kreb’s cycle dehydrogenases → ↑ lactate. Also, uncouple oxidative phosphorylation → ↑ heat (fever) and ↑ glycolysis → hypoglycemia → ↑ lipid metabolism → ↑ ketones → ↑ acidosis

ETOH
- Normal AG and ↑ osmolal gap; however if ETOH ketoacidosis, AG is ↑

Causes of Lactic Acidosis
- CO, CN, ASA, INH, Fe toxicity, ETOH abuse, hypoxemia, metformin, ritodrine, seizure and shock [9, pg. 199]

METHANOL AND ETHYLENE GLYCOL
- Both inhibit mitochondrial respiration → ↓ intracellular NAD/NADH ratio →
- ↑ anaerobic glycolysis → ↑ lactate

Methanol (windshield washer fluid)
- → ADH formaldehyde → formic acid → accounts for most of the AG metabolic acidosis and ocular toxicity; symptoms may be delayed for up to 12 to 18 hrs

Methanol Treatment
- ETOH or Fomepizole (4-methylpyrazole)
- Folate 50 mg IV q 4 hours for several days
- Bicarb for severe acidosis
- Fomepizole →
  - 15 mg/kg over 30 minutes
  - 10 mg/kg q 12 hours x 4 doses
  - More frequent dosing required during dialysis (Fomepizole is removed with dialysis)

Methanol Indications for dialysis [12, 6th ed., pg. 1069]
- Visual or CNS dysfunction
- Peak methanol levels > 20 mg/dL
- Ingestion > 30 ml
- pH < 7.15

Ethylene Glycol
- → ADH Glycoaldehyde → Glycolic acid (main player for ↑ AG metabolic acidosis)
- → → Formic acid and Oxalic acid; Oxalic acid → binds Ca 2+ → urine crystals
- Used as de-icer, antifreeze and coolant

Ethylene Glycol 3 Phases of Toxicity
1) CNS = 1 to 12 hrs; ataxia, slurred speech, hallucinations, seizures, nystagmus, coma, death
2) Cardiopulmonary = 12 to 24 hrs; ↑ HR, ↑ BP, ↑ RR = most common; CHF, ARDS, CV collapse
3) Nephrotoxic = 24 to 72 hrs; flank pain, ATN, Ca Oxalate crystals → ↓ Ca (hypocalcemia in EG toxicity is a common test question)

Ethylene Glycol Treatment
- ETOH or Fomepizole
- Thiamine (B1) 100 mg and Pyridoxine (B6) 100 mg IM or IV (cofactors needed in the metabolic pathway of ethylene glycol for the conversion to nontoxic compounds)
- Replace Ca 2+ if necessary; 1 amp Ca Gluconate
- Bicarb for severe acidosis [12, 6th ed., pg. 1068]
Ethylene Glycol Indications for dialysis [12, 6th ed., pg. 1070]
- Triad = history, clinical presentation & lab results consistent with Ethylene glycol poisoning
- Ethylene glycol levels > 20 mg/dL
- Signs of nephrotoxicity
- Metabolic acidosis is present

SUBSTANCES CAUSING AN OSMOLAR GAP
Mnemonic: (I MADE GAS)
Note: You would too, if you ate GYROS everyday!

| I | Isopropyl alcohol |
| M | Methanol / Mannitol |
| A | Alcohol (ETOH) |
| D | DKA (due to acetone) |
| E | Ethylene Glycol |
| G | Glycerol |
| A | Acetone |
| S | Sorbitol |

RESPIRATORY COMPENSATION FOR METABOLIC ACIDOSIS
PCO2 = 1.5 [HCO3] + 8 ± 2

| Anion Gap = Na – (CL + HCO3) | Normal = 12 ± 2 |
| Osmolarity = 2 [Na] + glucose/18 + BUN/2.8 | Normal = 280-290 |
| Osmolal Gap = Lab determined osmolarity – calculated osmolarity | Normal = 5-10 |

Note: every 4.2mg/dl of alcohol = 1 milliosmol; therefore if Osmolal Gap is elevated, order serum ethanol level and make correction; if Osmolal Gap still elevated think of the mnemonic, “I MADE GAS”

FACTORS INCREASING THEOPHYLLINE HALF-LIFE
Mnemonic: (COP CELICA) Imagine a COP chasing a Toyota CELICA [4, with modifications and 12, pg. 796]

| C | Contraceptive-oral |
| O | Obesity |
| P | Pregnancy, 3rd trimester reduces the clearance of theophylline |
| C | Cipro |
| E | Erythromycin / Elderly and Neonates |
| L | Liver Disease |
| I | Inderal (Propranolol) |
| C | Cimetidine (Tagamet) |
| A | Allopurinol |
### FACTORS DECREASING THEOPHYLLINE HALF-LIFE

**Mnemonic:** *(SMOKING CPR)*  
*If you smoke, you may need CPR*

<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>S (SMOKING)</td>
<td>Carbamazepine (Tegretol)</td>
</tr>
<tr>
<td>M</td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>Phenytoin and Phenobarbital</td>
</tr>
<tr>
<td>N</td>
<td>Rifampin</td>
</tr>
</tbody>
</table>

### DRUGS THAT INCREASE DIGOXIN LEVELS

**Mnemonic:** *(VAN – PQ)*

<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>V (Verapamil)</td>
<td>(Isoptin, Calan, Sustained release = Isoptin SR, Calan SR, Verelan, Covera)</td>
</tr>
<tr>
<td>A (Amiodarone)</td>
<td>Cordarone</td>
</tr>
<tr>
<td>N</td>
<td>Nifedipine (Procardia, Adalat)</td>
</tr>
<tr>
<td>P</td>
<td>Prozac</td>
</tr>
<tr>
<td>Q</td>
<td>Quinidine</td>
</tr>
</tbody>
</table>

### POISONING ASSOCIATED WITH FEVER

**Mnemonic:** *(SAL2T3 ASAP)*

<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>Salicylates</td>
</tr>
<tr>
<td>A</td>
<td>Amphetamines</td>
</tr>
<tr>
<td>L</td>
<td>LSD (rare) [12, pg. 781]</td>
</tr>
<tr>
<td>T</td>
<td>TCA’s</td>
</tr>
<tr>
<td>A</td>
<td>Anticholinergics (antihistamines, phenothiazine, TCA’s)</td>
</tr>
<tr>
<td>S</td>
<td>Sympathomimetics (cocaine, amphetamines)</td>
</tr>
<tr>
<td>A</td>
<td>Antihistamines</td>
</tr>
<tr>
<td>P</td>
<td>PCP (may be hypo-or hyperthermic)</td>
</tr>
</tbody>
</table>

MAO Inhibitor overdose

### POISONING ASSOCIATED WITH HYPO-THERMIA

**Mnemonic:** *(COOLS)* [ANK]

<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>Carbon monoxide</td>
</tr>
<tr>
<td>O</td>
<td>Opiates</td>
</tr>
<tr>
<td>O</td>
<td>Oral hypoglycemics, insulin</td>
</tr>
<tr>
<td>L</td>
<td>Liquor</td>
</tr>
<tr>
<td>S</td>
<td>Sedative hypnotics (barbiturates, benzodiazepines, chloral hydrate, etc.)</td>
</tr>
</tbody>
</table>
DIAPHORETIC SKIN
Mnemonic: (SOAP)

| S | Sympathomimetics |
| O | Organophosphates |
| A | ASA (salicylates) |
| P | PCP |

SIGNS AND SYMPTOMS OF CHOLINERGIC EXCESS
Mnemonic: (SLUDGE BAM) [MNC]

| S | Salivation |
| L | Lacrimation |
| U | Urination |
| D | Diaphoresis |
| G | GI – motility (diarrhea) |
| E | Emesis |

| B | 1) Bradycardia 2) Bronchoconstriction → Bronchospasm 3) Bronchorrhea |
| A | Abdominal cramps |
| M | 1) Myosis 2) Muscle cramps, weakness and fasciculations |

- Acetylcholinesterase (ACHE) inhibitors → cholinergic excess.

- Examples of acetylcholinesterase inhibitors include organophosphate and carbamate insecticides, nerve gases (sarin, soman, tabun and VX) and therapeutic agents (edrophonium/tensilon, pyridostigmine, physostigmine, and neostigmine)

- Organophosphate poisoning: the insecticides’ phosphate radicals covalently bind to active serine sites on ACHE → enzymatically inert proteins; irreversible inhibition. Examples include: isoflurophate, echothiophate, pesticides such as malathion and parathion and toxins such as sarin and other nerve gases [12, pg. 823;10, 4th ed. pg. 1403]

- Carbamates → carbamylation of ACHE → reversible inhibition, because of the bond spontaneously breaks within 4 to 8 hours with regeneration on ACHE to the active form. Examples include: pyridostigmine, physostigmine, neostigmine and pesticides like carbaryl

TREATMENT CHOLINERGIC EXCESS

1) ABC’s
2) Decontamination, lavage and charcoal 1 gm/kg
3) Atropine 3 mg IV, (peds 0.05 mg/kg IV), large amounts (10-20 mg) may be needed over 24 hours. Atropine blocks the action of acetylcholine at muscarinic (SLUDGE BA presentation), not nicotine receptors responsible for Muscle cramps, weakness and fasciculation and tachycardia
4) **Pralidoxime (2-PAM)** adults 1-2 gm IV, (peds 23-30 mg/kg over 3-5 minutes)\(^9\), \(^{355}\) 2-PAM reverses the cholinergic **nicotinic** effects that are unaffected by atropine alone. Use in carbamate poisoning is controversial \(^{12\text{, }5\text{th ed.}\text{, pg. 2189}}\)

**Mechanism:**
- Reactivation of cholinesterase by cleaving the phosphorylated active sites
- Direct detoxification of unbound organophosphate and
- Endogenous ANTI-cholinergic effect \(^{12\text{, pg. 826}}\)

**SIGNS AND SYMPTOMS OF ANTI-CHOLINERGIC TOXICITY**

- Dry as a bone (dry skin first symptom)
- Hot as Hades (hyperthermia)
- Blind as a bat (mydriasis)
- Mad as a hatter (delirium, hallucinations)
- Red as a beet (flushing with hyperthermia)

Tachycardia, Urinary Retention, Hypoactive or absent bowel sounds

**ANTI-CHOLINERGIC TOXICITY EXAMPLES**

- Anti-parkinsonian drugs, anti-histamines (H1-receptor blockers), phenothiazine, some mushrooms, TCAs, belladonna alkaloids (**jimson weed**, atropine, scopolamine), Atrovent and chemically related drugs to TCA's such as carbamazepine (Tegretol), cyclcobenzaprione (Flexeril)
- Note: sympathomimetics present with similar symptoms as anti-cholinergic excess .... however, **sympathomimetics → sweating and + bowel sounds**
- Treatment: Physostigmine 1-2 mg IVP slowly over 5"... if give too fast → SLUDGE BAM or seizures
- Also, most common side effect of physostigmine = seizures; other side effects = ↓HR, ↓BP and SLUDGE BAM
- \(t\frac{1}{2}=30-60\) minutes, may repeat in 20 minutes if no effect

**SUBSTANCE CAUSING NYSTAGMUS**

**Mnemonic:** (**PCP To PETS MEALS**)

*If you want to see your pets go crazy and demonstrate nystagmus, add PCP to your pets’ meals*

<table>
<thead>
<tr>
<th>P</th>
<th>PCP (Phencyclidine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ph</td>
<td>Phenytoin</td>
</tr>
<tr>
<td>E</td>
<td>ETOH</td>
</tr>
<tr>
<td>T</td>
<td>1) Tegretol  2) Thiamine depletion</td>
</tr>
<tr>
<td>S</td>
<td>Solvents</td>
</tr>
<tr>
<td>M</td>
<td>Methanol</td>
</tr>
<tr>
<td>E</td>
<td>Ethylene glycol</td>
</tr>
<tr>
<td>A</td>
<td>Alcohols (isopropyl alcohol)</td>
</tr>
<tr>
<td>L</td>
<td>Lithium (^{10\text{, 4th ed.\text{, pg. 1392}})}</td>
</tr>
<tr>
<td>S</td>
<td>Sedative hypnotics</td>
</tr>
</tbody>
</table>
### TOXICOLOGY

**MIOSIS**

**Mnemonic:** (COPS) 2

| C | Clonidine | Cholinergics |
| O | Opiates   | Organophosphates |
| P | Phenothiazine | Pilocarpine |
| S | Sedative hypnotics | Stroke (pontine bleed) |

PCP Intoxication (cholinergic) = **miosis** (2.1%) or (anticholinergic findings) = **mydriasis** (6.2%)

Horner syndrome = miosis

### MYDRIASIS

**Mnemonic:** (4 - AAAA)

| A | Antihistamines |
| A | Antidepressants (TCAs) |
| A | ANTI-cholinergics |
| A | Amphetamines & other Sympathomimetics (cocaine) |

### CAUSES OF SEIZURES

**Mnemonic:** (U HIT)2 (OTIS CAMPBELL) 3

**MEDICAL**

| U | Uremia | Used up oxygen (hypoxia) |
| H | Hypo’s (Na, Ca, Mg) | Hypo-glycemia |
| I | Infection | Idiopathic |
| T | Trauma (SAH, etc.) | Tumor (brain) |

**TOXICOLOGY - Otis Campbell = The “town drunk” on “The Andy Griffith Show”**

| O | Organophosphates | Opiates | Olanzapine (Zyprexa) |
| T | TCA’s | Theophylline | Tramadol (Ultram) |
| I | INH | Iron | Inhalants |
| S | Sympathomimetics | Salicylates | SSRIs |

| C | Cocaine | CO | CN and Camphor |
| A | Amphetamines | Anticholinergics | Anesthetics - local |
| M | MDMA | Meperidine (Demerol) | Mushrooms (Gyromitra esculenta) |
| P | PCP | Phystostigmine | Propoxyphene (Darvon) |
| B | B-blockers | Benadryl | Bupropion (Wellbutrin, Zyban) |
| E | ETOH withdrawal | Etomidate | Ephedra |
| L | Lithium | LSD | Lexapro (escitalopram) |
| L | Lead | Lidocaine | Lindane |
SEIZURE HISTORY
**Mnemonic (B COLD)**

<table>
<thead>
<tr>
<th>B</th>
<th>Bladder or Bowel incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>Character (type of seizure)</td>
</tr>
<tr>
<td>O</td>
<td>Onset (when did it start, what was the patient doing)</td>
</tr>
<tr>
<td>L</td>
<td>Location (where did the activity start)</td>
</tr>
<tr>
<td>D</td>
<td>Duration (how long did it last)</td>
</tr>
</tbody>
</table>

Also, previous episodes of seizure activity, previous medical history, meds and trauma history.

**MORE CAUSES OF SEIZURES**

Treat INH (isoniazid) seizures with IV pyridoxine (Vitamin B6), 1 mg for each mg INH, if unknown amount ingested, give 5 mg IV; HCO3, for alkaline diuresis; consider dialysis.

Water Hemlock (*Cicuta douglasii*) → intractable seizures occur 1hr after ingestion; ↑ mortality; Treatment = hemodialysis

Jimson Weed (*datura stramonium*)

**Anti-epileptics that may Cause Seizures:**
Lamictal (lamotrigine), Phenytoin, Fosphenytoin, carbamazepine (Tegretol)

**CAUSES OF SEIZURES – WITHDRAWAL**
**Mnemonic - (BEGS)**

<table>
<thead>
<tr>
<th>B</th>
<th>Baclofen</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>ETOH</td>
</tr>
<tr>
<td>G</td>
<td>Gamma-hydroxybutyrate (GHB)</td>
</tr>
<tr>
<td>S</td>
<td>Sedative Hypnotics</td>
</tr>
</tbody>
</table>
NON-CARDIOGENIC PULMONARY EDEMA
Mnemonic - (MOP CD)2  ASAP

| M | Methamphetamine smoking meth | Mountain sickness (HAPE= high altitude pulmonary edema) |
| O | Opiates (heroin, methadone) | Overwhelming Sepsis |
| P | Phenobarbital | Pancreatitis |
| C | Carbon Monoxide / Chlorine | CVA (neurogenic pulmonary edema) |
| D | Drugs | Drowning |
| A | ASA (Salicylates - more common in chronic poisoning and adults more > common than peds) | |
| S | Sympathomimetics (smoking cocaine or methamphetamines) | |
| A | APAP | |
| P | Phosgene | |

ANTI-cholinergic toxicity = cardiogenic pulmonary edema secondary to depressed myocardial contraction

Choking Agents
Phosgene and Chlorine = non-cardiogenic-pulmonary edema
Delayed symptoms with phosgene; immediate symptoms are noted with chlorine

LITHIUM TOXICITY
[10, 6th ed., pgs 1048-1051; 5th ed., pg 2172]

- Hand tremor 65%
- N / V / D, anorexia, abdominal cramping → common early
- Polyuria → nephrogenic diabetes insipidus (DI) → inhibits arginine vasopressin
- (Antidiuretic hormone, ADH); more common in chronic toxicity
- Antithyroid effect → blocks thyroid hormone release → myxedema coma
- Parathyroid hyperplasia or adenoma → mild ↑Ca 2+
- EKG = flat T waves, ↓ST, ↓HR, U waves, ↑QTc (inhibits Na/K pump → ↓K+ in cell), ventricular arrhythmias, sinus arrest, asystole
- CNS = lethargy, confusion, spasticity, cogwheel rigidity, ↑DTRs, coma and seizures
- Acute overdose more GI and less CNS toxicity
- Normal range 0.6 to 1.2 mEq/L (lithium is slowly eliminated from cells, half-life = 24±12 hours, therefore patients may be toxic with levels in therapeutic range)
- Serum levels especially do not predict CNS levels (brain may → 2 to 3x higher concentrations)

LITHIUM TOXICITY TREATMENT

Mild/moderate toxicity
- Aggressive hydration with 0.9% NS (if dehydrated kidney reabsorbs lithium preferentially)
- Kayexalate 15gm po (risk for hypokalemia)
- Charcoal is ineffective → consider charcoal in polydrug toxicity
- Whole-bowel irrigation → helpful, especially if sustained-release lithium products
Severe toxicity → Hemodialysis
- Coma, seizures
- Level > 3.5 or 4.0 mEq/L in acute overdose
- Patients with minimal change in their levels after 6 hours of hydration
- Patients with sustained levels > 1.0 mEq/L after 36 hours
- Seizures = BZP, Phenobarbital; Dilantin ↓ renal excretion of lithium and is ineffective
- If patient is asymptomatic after 6 hours of treatment of acute, non-sustained release, ingestions → psych consult

Interesting trivia provided by Dr. Bryan Bluhm → when the soft drink 7-UP was introduced in 1929 it originally contained lithium; fyi the molecular weight of lithium = 7

5 STAGES OF IRON TOXICITY
[12, 6th ed., pg 1122-1123]

Stage 1 (Gastrointestinal) Within 6 hours of ingestion
- N/V/ D abdominal pain; GI bleeding common

Stage 2 (Latent) usually occurs 6-12 hours after ingestion and may lasts 24 hours
- Resolution of GI symptoms
- This deceptive phase as the patient appears to improve and recover
- Metabolic abnormalities during this phase may include ↓BP, metabolic acidosis, and coagulopathy

Stage 3 (metabolic/cardiovascular – systemic toxicity) starts as early as 6-8 hours, lasts up to 2 days
- Most patients die during this phase
- Acute Renal Failure
- Recurrent GI bleeding
- Cardiomyopathy with CV collapse
- Coagulopathy – worsens bleeding; leukocytosis
- CNS symptoms = lethargy, encephalopathy and coma
- Anion-Gap metabolic acidosis (intracellular iron disrupts cellular metabolism)
- Combination of fluid and blood loss, with additional third-spacing → hypovolemia or shock

Stage 4 (hepatic) 2 to 5 days after exposure
- ↑ LFTs, BILI and ↑ coagulopathy (hepatic injury → ↓ factor production / hepatic failure)
- Hepatic injury → ↓ Glucose (hypoglycemia)
- ↑ Ammonia → encephalopathy, coma

Stage 5 (delayed) Usually → 4 to 6 weeks after a severe poisoning
- Gastric outlet obstruction or proximal bowel scarring / obstruction; rare

Moderate Toxicity = 20 to 60 mg/kg of elemental iron
Severe Toxicity = > 60 mg/kg of elemental iron

IRON TOXICITY TREATMENT
- Fluid, vitamin K, FFP, blood, antiemetics
- Consider gastric lavage if presentation within 1st hour
- Whole-bowel irrigation
- EGD to remove pills
- Deferoxamine 90 mg/kg IM (up to 1 gm in PEDS), q 4 to 6 hours as clinically indicated; binds directly to free iron; Change in urine color (vin rosé urine) → should not be the sole factor in deciding toxicity.
TRICYCLIC ANTIDEPRESSANT (TCA) PATHOPHYSIOLOGY

1) **Inhibition of amine uptake** → ↑ serotonin → serotonin syndrome and ↑ norepinephrine, dopamine → early sympathomimetic effects (↑ HR, early mild HTN, followed by hypotension, arrhythmias)

2) **Anticholinergic effects** → only muscarinic, NOT nicotinic → central anticholinergic symptoms delirium, hallucinations, seizures, sedation and coma peripheral symptoms please see anticholinergic toxicity

3) **Inhibition of adrenergic post-synaptic receptors (α1 and α2)** → ↓ BP and reflex ↑ HR. Inhibition of ocular α adrenergic receptors → miosis which frequently offsets anticholinergic-induced mydriasis

4) **Na+ channel blockade** → Bradycardia, QRS prolongation, RAD, hypotension & seizures
   
   Na+ channel blockade → negative chronotropic → ↓ HR (the ↑ HR from anticholinergic activity partially offsets the ↓ HR; if patients has ↓ HR and **wide QRS = ↑ toxicity/Na channel blockade**)
   
   Na+ channel blockade → ↓ Na+ influx and delayed depolarization → ↑ QRS & PR on EKG
   
   ↓ rapid Na+ influx → ↓ release of intracellular Ca++ → ↓ myocardial contractility → ↓ BP
   
   Na+ channel blockade → (other: VT/VF, heart blocks, seizures)

5) **K+ channel antagonist** → ↓ K+ efflux during repolarization → ↑ QT → torsades

6) **GABA antagonism** → seizures

CARBON MONOXIDE (CO) POISONING – PATHOPHYSIOLOGY

- The affinity of CO for hemoglobin is 250x > than the affinity of O2 for hemoglobin → ↓ of the O2 carrying capacity in the blood → tissue hypoxia

- Shifting of the oxygen-hemoglobin dissociation curve → **LEFT** → ↓ O2 available at cellular level → ↓ tissue hypoxia

- Binds to cytochromes A, and P450 → inhibit cellular (mitochondrial) respiration → lactic acid

- Binds to myoglobin → ↓ O2 available to myocardium → ischemia / arrhythmias

- Brain lipid peroxidation → neuronal damage

- See mnemonics, atrial fibrillation, seizures and non-cardiogenic pulmonary edema

- Classic cherry red skin is rarely seen

- Visual disturbances are frequent and correlate with the duration of exposure. Flame-shaped retinal hemorrhages, bright red retinal veins, papilledema, blindness is uncommon.

- Rhabdomyolysis → renal failure
• Check EKG, cardiac enzymes, ABG, lactate and COHB level

• N/V/D, hepatic necrosis, hematochezia, melena, non-pancreatic ↑amylase, DI, ↑glucose, ↓Ca, ↑BUN/Cr, DIC, ↑WBC

• CO is removed almost exclusively via the pulmonary circulation through competitive binding of hemoglobin by O2

• Serum elimination half-life of carboxyhemoglobin (COHb)
  Remember: ↓ 1/3 → 1/3 → 1/3
  Breathing room air = 180 minutes → 100% O2 = 60 min → 20 min with HBO [12, 6th ed., pg. 1240]
  Breathing room air = 300 minutes → 100% O2 = 90 min → 30 min with HBO [Up to Date]

HYPERBARIC OXYGEN (HBO) DEFINITE INDICATIONS
[12, 6th ed., pg. 1240]

• ↓BP
• Coma
• Seizure
• Myocardial ischemia
• ↑ Prolonged exposure
• LOC or near syncpe
• Pregnancy with COHB > 15%; fetal distress
• MS changes and /or abnormal neuro exam

ACETAMINOPHEN (N-ACETYL-PARA-AMINOPHENOL, OR APAP) OVERDOSE PEARLS
[12, 6th ed., pg. 1088-1093]

• 90% of APAP is metabolized in the liver to sulfate and glucuronide conjugates.
  Conjugated metabolites lack biologic activity and are not hepatotoxic → excreted in urine

• 5% remaining APAP is → excreted unchanged in the urine and

• 5% remaining is metabolized via the hepatic cytochrome P450 pathway to a hepatotoxic metabolite → N-acetyl-p-benzoquinone-imine (NAPQI)

• The amount of NAPQI formed during the metabolism of APAP at therapeutic doses can be detoxified by conjugation with hepatic glutathione → nontoxic NAPQI conjugates → excreted in urine

• At toxic doses, acute ingestion = 150 mg/kg peds, 7.5 grams adults → sulfate and glucuronide conjugation become saturated → ↑APAP is shunted into the cytochrome P450 pathway for further metabolism → ↑ NAPQI → glutathione stores are depleted → hepatic necrosis

• Drugs that stimulate/induce the P450 pathway may enhance APAP toxicity: (chronic use of: antihistamines, phenytoin, barbiturates, carbamazepine and chronic ETOH abuse)

• Drugs that inhibit the P450 pathway protect against APAP toxicity: cimetidine (Tagamet)

• Acute ingestion of ETOH protects against APAP toxicity by competitive inhibition via the cytochrome P450 pathway → ↓ amount of NAPQI produced
**PEARL:** children are less susceptible to hepatotoxicity than adults (increased rate of sulfation and increased relative size of liver affords hepato-protective effects to pediatric patients)

At **toxic doses** sulfate and glucuronide pathways become saturated → ↑APAP metabolized by cytochrome P450 enzymes. Once glutathione stores are depleted → ↑ NAPQI → hepatic injury

**ACETAMINOPHEN (N-ACETYL-PARA-AMINOPHENOL, OR APAP) OVERDOSE PEARLS**

**FOUR STAGES OF ACETAMINOPHEN POISONING**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Time Following Ingestion</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>First day</td>
<td>Anorexia, N/V/malaise, lethargy, pallor, diaphoresis</td>
</tr>
<tr>
<td>II</td>
<td>1 to 3 days</td>
<td>Abdominal pain, liver tenderness, elevated LFT's, lipase, oliguria (ATN)</td>
</tr>
<tr>
<td>III</td>
<td>3 to 4 days</td>
<td>Peak LFT's and ↑ PT/INR, jaundice, confusion (hepatic encephalopathy), ↑ammonia, lactic acidosis Hypoglycemia</td>
</tr>
<tr>
<td>IV</td>
<td>4 days to 2 weeks</td>
<td>Resolution of hepatotoxicity or progressive failure</td>
</tr>
</tbody>
</table>

- The serum APAP concentration should be measured at 4 and 24 hours after a single large overdose of an immediate-release preparation. The level should be evaluated according to the **Rumack-Matthew nomogram** to determine the risk of hepatotoxicity and need for therapy
- Other labs: serum tox, urine tox, pregnancy, UA, LFTs, lipase, coags, CBC, electrolytes, glucose, BUN/Cr; severe overdoses – ammonia level
- ETOH and chronic APAP poisoning hepatitis AST (GOT)/ALT (GPT) ratio > 2
- **Acute** APAP poisoning hepatitis AST/ALT ratio < 2
- An early marker for subclinical hepatic injury following APAP overdose is serum alpha glutathione S-transferase (a-GST), which is both released into and cleared from the circulation more rapidly than AST
- See mnemonic, “Drugs that cause pancreatitis”
- Elevated PT / INR indicate impaired synthetic liver function
- Non-cardiogenic pulmonary edema may develop in severe APAP poisoning
• The proximate cause of death in APAP-induced hepatic failure is usually cerebral edema
• APAP crosses the placenta, which places the fetus at potential risk of hepatotoxicity
• NAPQI does not cross the placenta
• The fetus metabolizes APAP → hepatotoxicity

ACETAMINOPHEN OVERDOSE PROGNOSTIC INDICATORS

[Mahadevan et al, 2006]

1. Delayed presentation to the emergency department
2. Delay in treatment
3. Prothrombin time > 100 seconds
4. Serum creatinine > 200 mcL/L (2.26 mg/dL)
5. Hypoglycemia
6. Metabolic acidosis
7. Hepatic encephalopathy grade III or higher

Acetaminophen Antidote = Acetylcysteine; other names = N-acetyl-L-cysteine or N-acetylcysteine (NAC)
Trade names = Mucomyst (oral) and Acetadote (IV)

• If given within 8 hours NAC almost 100% effective in preventing hepatotoxicity
• Most toxicologists believe mucomyst prevents APAP hepatotoxicity by restoring hepatic glutathione stores; N-acetylcysteine also increases oxygen extraction by the liver, prevents WBC migration to the liver and acts as a free radical scavenger → may be mechanism for improved hemodynamic and decrease in cerebral edema

Mucomyst (acetylcysteine)
1. **140 mg/kg po** loading dose
2. Next, give 70 mg/kg every 4 hours x 17 doses

Acetadote (acetylcysteine)
1. 150 mg/kg IV in 200cc D5W over 60 minutes
2. 50 mg/kg IV in 500cc D5W over 4 hours
3. 100 mg/kg IV in 1,000cc D5W over 16 hours

Activated charcoal
• Binds NAC, however the reduction in NAC absorption is insignificant
Dose of charcoal = 1 gm/kg (max dose 50 mg)

Hemodialysis
• Because of its low volume of distribution and minimal protein binding, acetaminophen can be removed via hemodialysis
• However, considering N-acetylcysteine is so effective in the management of acetaminophen toxicity, the role for dialysis is **minimal**

Gastric lavage
• No proven efficacy in isolated acetaminophen overdose
Rumack-Matthew nomogram for the single acute acetaminophen poisoning. Semilogarithmic plot of plasma acetaminophen levels versus time. Caution s for use of this chart: (1) The time coordinates refer to time of ingestion. (2) Serum levels drawn before 4 hours may not represent peak levels. (3) The graph should be used only in relation to a single acute ingestion. (4) The lower solid line 25% below the standard nomogram is included to allow for possible errors in acetaminophen plasma assays and estimated time from ingestion of an overdose. (Adapted from Rumack BH, Matthew H: Pediatrics 55:871-876, 1975.)
ACETAMINOPHEN OVERDOSE PEARLS
(N-acetyl-para-aminophenol, or APAP)

The 150 Rule
- Toxic dose is 150 mg/kg
- Give NAC if level is >150 mcg/mL four hours post-ingestion
- Initial loading dose of NAC is 150 mg/kg IV (140mg/kg PO)

SALICYLATE OVERDOSE
(12, 6th ed. pgs., 1085-1088)

- Acute ingestion of 150 to 300 mg/kg produce mild/moderate toxicity = N/V/diaphoresis/↑RR/tinnitus (often first symptom reported)
- 300 – 500 mg/kg = severe toxicity
- > 500 mg/kg = potentially lethal

SALICYLATE OVERDOSE PATHOPHYSIOLOGY

1) Krebs cycle inhibition → ↑ lactate → ↑ metabolic acidosis
2) Uncouples oxidative phosphorylation
   → ↓ ATP production → release of energy in form of ↑ heat (fever)
   → ↓ ATP production → cerebral edema
   → ↓ ATP production → acidosis → cardiac depression / hypotension / VT/VF
   → ↑ anaerobic glycolysis → hypoglycemia → ↑ lipid metabolism → ↑ ketones → ↑ metabolic acidosis
3) Stimulates respiratory centers in brain stem → Kussmaul breathing → respiratory alkalosis
4) ↑ pulmonary capillary permeability → noncardiogenic pulmonary edema /ARDS
   a. More common in adults > peds
   b. More common in chronic > acute ASA poisoning
5) ↓ clotting factor VII synthesis, → prolonged ↑ PT /INR

SALICYLATE OVERDOSE DONE NOMOGRAM
(12, 6th ed. pgs., 1085-1088)

- Predict the degree of toxicity after an acute single ingestion (cannot use if ASA ingestion within the last 24 hours or ingestion occurred over several hours or chronic salicylate poisoning or ingestion of enteric coated ASA tablets)
- Serum level must be drawn at least 6 hours after ingestion
- [12, 6th ed. pg 1087] = Done nomogram has limitations, deceptive utility and not recommended

SALICYLATE OVERDOSE TREATMENT

- Charcoal
- Oxygen; avoid intubation. Fluid resuscitation unless pulmonary edema
- Dextrose drip if AMS, regardless of serum glucose
- Bicarb and K+ are needed to produce alkaline urine
- Why K+? → When Na+ is reabsorbed, the kidney preferentially secretes H+ ions into the tubular lumen rather than K+, you need K+ ions to compete with H+ ions → produce more alkaline urine
- Monitor ABG (keep arterial pH > 7.4), electrolytes and urine pH every 2 hours
- Bicarb 1 mEq/kg IV boluses until arterial pH > 7.4
- Continuous IV infusion of 1 L 5% dextrose in water, which is added 50 to 100 mmolNaHCO3 and 40 mmol KCL, started at 2x maintenance rate
- ASA is eliminated by renal excretion
- Ionized ASA cannot be reabsorbed and → excreted
- Alkaline urine favors the formation of ionized salicylate → ↑ excretion
- Hemodialysis

**METHEMOGLOBINEMIA**

- A form of hemoglobin wherein the ferrous (Fe2+) has been oxidized → ferric (Fe3+)
- The oxidized hemoglobin is incapable of carrying oxygen
- Causes: benzocaine, lidocaine, prilocaine, dapsone, sulfonamides, nitrofurantoin phenazopyridine (Pyridium), antimalarials (primaquine, chloroquine), nitrates and nitrites, acetaminophen, acetanilid, phenacetin, celecoxib
- Cyanosis is usually the first presenting symptom
- Blood sample = chocolate in color → turns red on exposure to air
- Treatment: Methylene Blue 1-2mg/kg, one 10 ml 10% solution (100mg) is initial adult dose \(^{12, 6th\ ed., \ pg\ 1017}\); treat if symptomatic or levels above 30%

Methemoglobin (MetHb) and carboxyhemoglobin (COHb) → ABG = normal PaO2 and calculated oxygen saturation, because the dissolved oxygen is unaffected, hence normal PaO2

The calculated oxygen saturation is based on the PaO2, therefore, will also be normal

**You must order** MEASURED oxygen saturation (measures the % hemoglobin bound to oxygen) – abnormally low ↓
MUSHROOM POISONING

Toxicology

Mushrooms symptom onset
- If onset < 6 hours after ingestion clinical course benign
- > 6 hours possible hepatotoxic, nephrotoxic, and erythromelalgic (hemolytic anemia) syndromes

- Amanita species (Cyclopeptides) = nearly all mushroom fatalities; 3 phases of illness:
  1) GI → 2) quiescent → 3) hepatic failure → hepatorenal syndrome
  Onset of symptoms delayed 6 to 24 hours; does not cross placental barrier
  No specific antidote, however treatment options include:
  - Activated charcoal
  - N-acetylcysteine (NAC)
  - High dose PCN G = blocks uptake of amatoxin in liver
  - Silymarin (silibinin) / Milk Thistle oral = hepatoprotective (available in Europe) – occupies receptor sites
  - Cimetidine, HBO
  - Liver transplant

- Gyromita esculenta → CNS and hepatotoxic; Methemoglobinemia; onset < 6 to 24 hours; heat labile; more toxic/fatal then Amanita however fewer fatalities because poisoning much less common
  Neuro symptoms treated with High Dose Pyridoxine (B6) – no effect on liver

- Psilocybe → structurally similar to LSD → psychedelic effects; onset < 30 min

- Cortinarius orellanus → norleucine toxin = nephrotoxic → delayed onset renal failure

- Inocybe and Clitocybe → muscarine → onset < 30 min SLUDGE BAM; tx = atropine

- Amanita pantherina (panther mushroom) → anticholinergic symptoms treatment of severe cases = physostigmine

- Coprinus → inky cap” or “shaggy mane” = disulfiram (Antabuse) reaction with ETOH onset 2 to 72 hours and < 30 min after ETOH (HA, flushing, SOB, ↑RR, ↑HR) [10, 5th ed., pg. 2203]
  Treatment = beta-blockers for SVT, Norepinephrine for refractory ↓ BP

- GI toxins with onset of symptoms < 2 hours = most commonly ingested mushroom
  N/V/D (occasionally bloody) / abdominal pain
  Chlorophyllum molybdites (green gill) – most common
  Omphalotus illudens (jack-o’-lantern)
  Boletus piperatus (pepper bolete)
  Agaricus arvensis (horse mushroom)

  Hypoglycemia is one of the most common causes of death in mushroom toxicity
PEARLS - COCAINE, OPIATE, BARBITURATE, PCP, GHB, NMS, SYMPATHOMIMETICS

- **Cocaine** acts as a type IA sodium channel blocker → prolongs the QT

- **Cocaine** overdose: beta-blockers are contraindicated → unopposed a-adrenergic receptor stimulation → will worsen cocaine-induced coronary and peripheral vasoconstriction

- **Cocaine** use cause 90% of strokes in young adults (3rd and 4th decades of life), and is the most common cause of drug-associated stroke

- **Opiate**-induced pulmonary edema: treatment with diuretics is not effective. Use of naloxone and supportive treatment is all that usually is needed, and typically clears rapidly in 24-36 hours

- Miosis is a well-known side effect of opiate use however meperidine causes mydriasis instead

- Naloxone (Narcan) = 0.4 mg IV initial test dose should be given (avoid violent withdrawal symptoms). If no response is observed, give 2 mg doses q three minutes up to 10 mg total IV

- Clinical effects of **opiate** reversal with naloxone = 30-60 minutes → monitor for return of s/s of OD

- Cutaneous bullae occur in 4-6% of patients with **barbiturate** coma and in 50% of patients who die from barbiturate overdose

- Haloperidol (Haldol) use in **PCP** overdose may trigger a syndrome similar to neuroleptic malignant syndrome; consider benzodiazepines for sedation to avoid this potential complication

- Bruxism, or jaw clenching, is seen in nearly 100% of **MDMA** users. They often resort to use of pacifiers or lollipops to relieve jaw tension

- **GHB** overdose = rapid and profound CNS depression; deep levels of anesthesia last only 1-4 hours and spontaneously resolve with only supportive treatment

- **Sympathomimetics** present with similar symptoms as **anti-cholinergic** excess.... however, sympathomimetics → sweating and + bowel sounds

- Both **NMS** and **Serotonin Syndrome** = autonomic instability, altered MS and muscle rigidity

- NMS medical treatment options – controversial, include:
  - Dantrolene, a direct-acting skeletal muscle relaxant; 1 to 2.5mg/kg IV in adults, can be repeated to a maximum dose of 10 mg/kg/day
  - Bromocriptine, a dopamine agonist, restores lost dopaminergic tone. 2.5 mg (through nasogastric tube) every six to eight hours are titrated up to a maximum dose of 40 mg/day

- R rigidity in NMS more severe (“lead pipe” rigidity) >>> vs Serotonin Syndrome

- Hyperreflexia and clonus can coexist in Serotonin Syndrome, which is NOT typical in NMS
PEARLS - TODDLER TOX KILLERS

- Even a small amount of following can result in death: B-blockers, especially Inderal (propranolol), CCBs, camphor, ethylene glycol (tx dialysis), lomotil (tx naloxone), Amanita phalloides, Methyl salicylate, sulfonylureas (D5W & octreotide), TCAs and theophylline [Dr. Rangan, ACEP News, Vol. 30, No. 2, Feb 2011]

- Liquid nicotine commonly used in e-cigarettes now recognized as “one pill killer” [Journal of Paediatrics and Child Health Volume 50, Issue 2, pages 164-165, February 2014]

SULFONYLUREA OD, WITHDRAWAL SYNDROMES, CLONIDINE, LEAD, HEMLOCKS, DIG

- Treatment option of refractory hypoglycemia after sulfonylurea OD = Octreotide → somatostatin analog → inhibits insulin secretion from the pancreas that are a result of both the sulfonylurea and dextrose

- Acute ETOH withdrawal = anxiety, tremulousness, agitation, autonomic hyperactivity (↑HR, HTN, arrhythmias), hallucinations, and/or seizure

- Heroin withdrawal = yawning, N/V/D, abdominal pain, piloerection, restlessness, mydriasis and rhinorrhea

- Cocaine withdrawal = simulates depression

- Clonidine toxicity = ↓BP, ↓HR, ↓ RR, MS change, and miosis → closely mimics opioid toxicity

- Bluish lines on the gingival = lead lines from lead toxicity

- Lead concentrates in metaphyses of growing bones: distal femur, both ends of tibia or distal radius

- Aldrich - Mees Lines are horizontal lines of discoloration on the nails of the fingers and toes Poisonings = arsenic, thallium; or renal failure

- Water Hemlock (Cicuta douglasii) → intractable seizures occur 1 hour after ingestion; ↑ mortality

- Poison Hemlock (Conium maculatum) was reportedly used to execute Socrates (I had to add a Greek pearl)

- Root contains the greatest concentration of toxin in both species above

- Digoxin toxicity presentation = weakness, fatigue, nausea/vomiting/diarrhea, confusion, and a visual disturbance hallmarked by yellow/green halos around objects [12, 5th ed., pg. 1140]
PEARLS - BETA-BLOCKER, CCBS, SEIZURES, ANESTHETICS, STRYCHNINE, ODORS

- Treatment options for Beta-blocker and CCBs Overdose
  - Glucagon 3 mg IV (max 10mg), then begin infusion 3 to 5 mg/hour [Ref 29, 2010 AHA pg. 47, 63]
  - Glucagon → enhances Ca entry and usage in cell → ↑ cardiac inotropy
  - High dose insulin 1.0 unit/kg bolus with Dextrose bolus (0.5g/kg IV); Insulin drip 0.5 unit/kg/hr with D25W
  - With CCB OD give Ca Gluconate or Ca Chloride (central line)
  - Vasopressors
  - Milrinone (Primacor) or Inamrinone (Inocor)
  - Intravenous lipid emulsion
  - Charcoal

- Beta-blocker cause seizures (see seizure mnemonic) while CCBs rarely cause seizures; tx BZPs

- Children can have significant life-threatening toxicity from minor accidental ingestions of B-blockers/CCBs

- Treatment options for local anesthetic toxicity = Intravenous lipid emulsion [Ref 29, 2010 AHA pg. 47, 63]

- Most common cause of drug-induce seizures = Bupropion (Wellbutrin, Zyban)

- Isopropyl alcohol → “pseudo renal failure” = early clue for diagnosis. ↑ Cr, normal BUN and ↑ acetone level

- Intoxications that improve with Narcan: heroin, clonidine, tramadol, captopril, ethanol and valproic acid [10, 5th ed, pgs. 1791-1793]

- Strychnine poisoning resembles = tetanus infection; Strychnine blocks glycine receptor (glycine is an inhibitory neurotransmitter)→absorbed rapidly→ acute generalized seizure like skeletal muscles contractions [10, 5th pg. 1792]

- The most commonly abused substance among adolescents presenting to the ED = alcohol

- In non-diabetic children ages 2 to 10, the most common drug induced cause of hypoglycemia = alcohol (Other causes, Reyes, sepsis, aspirin, adrenal insufficiency, hypothyroidism)

- Toxic odors:
  - Fruity – isopropanol, DKA
  - Pear like – chloral hydrate
  - Garlic – arsenic, organophosphates, DMSO, selenium, Mustard agent
  - Mustard – Mustard (blister) agent
  - Rotten Eggs – hydrogen sulfide, sulfur dioxide
  - Fresh hay – phosgene
  - Wintergreen mint – methylsalicylate
  - Moth balls – camphor, naphthalene
  - Onion - Mustard (blister) agent
TRUE EMERGENT CAUSES OF SYNCOPE
Mnemonic - (CRAPS) [16]

<table>
<thead>
<tr>
<th>C</th>
<th>Cardiac arrhythmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>Ruptured AAA or Ruptured Ectopic</td>
</tr>
<tr>
<td>A</td>
<td>Aortic stenosis and IHSS</td>
</tr>
<tr>
<td>P</td>
<td>PE</td>
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<td>S</td>
<td>SAH</td>
</tr>
</tbody>
</table>

CAUSES OF SYNCOPE
(OH MY HEAD AND VESSELS ARE IN PAIN)

<table>
<thead>
<tr>
<th>O</th>
<th>Orthostatic hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hypovolemia</td>
</tr>
<tr>
<td>MY</td>
<td>MI</td>
</tr>
<tr>
<td>H</td>
<td>Hypoxia (CO/Anemia)</td>
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<tr>
<td>E</td>
<td>Seizure</td>
</tr>
<tr>
<td>A</td>
<td>Aortic stenosis</td>
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<tr>
<td>D</td>
<td>Drugs</td>
</tr>
<tr>
<td>AND</td>
<td>Anemia</td>
</tr>
<tr>
<td>V</td>
<td>Vasovagal</td>
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<tr>
<td>E</td>
<td>Ectopic</td>
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<tr>
<td>S</td>
<td>SAH</td>
</tr>
<tr>
<td>S</td>
<td>Sensitivity (Hyper)-Carotid Sinus</td>
</tr>
<tr>
<td>E</td>
<td>Electrolyte abnormally → dysrhythmia</td>
</tr>
<tr>
<td>L</td>
<td>Low SVR (sepsis)</td>
</tr>
<tr>
<td>S</td>
<td>Subclavian steal</td>
</tr>
<tr>
<td>ARE</td>
<td>Anxiety → vasovagal</td>
</tr>
<tr>
<td>IN</td>
<td>IHSS</td>
</tr>
<tr>
<td>PAIN</td>
<td>Psychiatric; Pericardial tamponade</td>
</tr>
</tbody>
</table>

EVALUATION OF SYNCOPE

- The most Sn and Sp test in evaluation of patient with syncope = H&P
- Cause of syncope determined by thorough H&P in 50 to 85% of patients [10, 5th ed., 173]
SAN FRANCISCO SYNCOPE RULE (SFSR)
Mnemonic - (CHESS)

| C | History of CHF |
| H | Hematocrit < 30% |
| E | Abnormal EKG |
| S | Shortness of Breath |
| S | Triage SBP < 90 |

A patient with any of the above measures is considered at high risk for a serious outcome (death, MI, arrhythmia, PE, CVA, SAH, significant hemorrhage).

CAUSES OF HEADACHE
Mnemonic - (MGM Studios Say They Can Create Happy Positive Movies to C) [ANK]

| M | Migraine / Tension / Cluster = Causes of “Primary” HAs [12, 6th ed., pg 1377] |
| G | Causes of Secondary HAs |
|   | Glaucoma / Iritis / Optic neuritis |
| M | Meningitis / Encephalitis/ Cerebral Abscess |

Studios Subdural / Epidural SAH / ICH
Say Sinusitis
They Temporal arteritis
Can CO poisoning
Create Carotid or Vertebral a. dissection
Happy HTN
Positive Pseudotumor cerebri
Post LP
Movies Mastoiditis
To Tumor (CNS masses)
C Cavernous sinus thrombosis

HEADACHE PEARLS

Most common recurrent head pain syndrome = Tension HA [10, 5th ed., pg 1460]
Women > men; 75% population affected

Post Dural Puncture HA (PDPH)
- Most common complication of LP 40% [10, 5th ed., pg 1464]
- Aggravated in the upright position and diminished in the supine position
- The amount of time supine does not affect incidence of HA
Factors to ↓PDPH

a. Smaller diameter needles → will cause less leakage  
b. Insert needle bevel parallel to long axis of spine → will minimize dura fiber damage  
c. Using atraumatic needles or pencil-point needles (Whitaker or Sprotte)

Treatment if bed rest and analgesics fail = epidural blood patch (autologous blood clot)

**TEMPORAL ARTERITIS (TA) / GIANT CELL ARTERITIS (GCA)**

3 of the following 5 items must be present

1. Age > 50 years  
2. New-onset headache or localized head pain  
3. Temporal artery tenderness to palpation or reduced pulsation  
4. Erythrocyte sedimentation rate (ESR) greater than 50 mm/h  
5. Abnormal arterial biopsy

Complications of GCA/TA = visual disturbances, diplopia and blindness.

- Diplopia is due to ophthalmoplegia usually of CN III or VI  
- Blindness is sometimes preceded by a visual field cut, amaurosis fugax, blurred vision or diplopia  
- Blindness in GCA/TA is due to posterior ciliary artery occlusion → ↓ blood flow to optic nerve → ischemia → blindness (less common blindness is due to central retinal artery occlusion or ischemic retrobulbar optic neuritis)  
- Definitive Diagnosis is made by temporal artery biopsy. Biopsy should be done within 1 week of the initiation of steroid therapy  
- Few studies exist regarding dosing protocols for corticosteroids  
- Start prednisone 60 mg/day po in the ED  
- Improvement of systemic symptoms typically occurs within 72 hours of initiation of therapy

**MENINGITIS PEARLS**

LP may be performed without Head CT in some patient groups during evaluation for meningitis

- Age less than 60  
- Immunocompetent  
- No history of CNS disease  
- No recent seizure  
- Alert and oriented  
- No papilledema  
- No focal neurologic deficits

Signs suspicious for space-occupying lesions = papilledema and focal neurologic deficits

The Infectious Disease Society of America (IDSA) states in its 2004 guidelines that the administration of antibiotics for suspected bacterial meningitis should be “emergent” but does not specify a time frame.

The decision to perform a head CT before lumbar puncture should not prevent the immediate administration of antibiotics

A paper published in 1989 found that the average time to antibiotic administration for bacterial meningitis is **three hours**
Although the yield of CSF cultures and CSF Gram stain may be diminished by antimicrobial therapy given prior to LP, pretreatment blood cultures and CSF findings (i.e., elevated WBC count, diminished glucose concentration, and elevated protein concentration) will likely provide evidence for or against the diagnosis of bacterial meningitis.

*(More meningitis pearls – see Pediatrics Section and Neurology Pearls)*

**STROKE SYNDROMES**

**Anterior Cerebral Artery** [ICEP 2009 Board Review and 12, 6th ed., pg 1384-1385]
- Paralysis of opposite leg > worse than arm
- Sensory deficit paralleling paralysis
- AMS; confusion
- Bowel or bladder incontinence

**Middle Cerebral Artery (Most common)**
- Paralysis of opposite body, arm, face > worse than leg
- Sensory deficit paralleling paralysis
- Hemianopsia = Blindness in lateral half of visual field
- Agnosia = inability to recognize objects
- Aphasia (receptive, expressive or both) is present if dominant hemisphere involved

Right-handed and 80% left-handed patients are left hemisphere dominant
Non-dominant hemisphere involved = inattention, neglect, dysarthic (difficulty articulating words) but not aphasic

**Posterior Cerebral Artery**
- Hemianopsia
- AMS
- Cortical blindness
- CN III paralysis
- Most ischemic strokes will not be seen on CT for at least 6 hours
- 80% of strokes are ischemic. Patients with AfiB are 10 to 20x more likely to develop stroke, and the majority of these are embolic events
- 30 day mortality after stoke = 20 – 25%; in-hospital mortality = 15% [10, 5th ed., pg 1433]
- Most common cause of new onset seizures in elderly = strokes
- Acute painless vision loss → anterior circulation stroke
  Common Carotid a. → Internal Carotid a. → first branch = ophthalmic artery → CN II (optic) and retina
- Internal Carotid artery terminates by branching → Anterior and Middle Cerebral arteries at the Circle of Willis
STROKE SYNDROMES

Mnemonic - Brainstem Stroke (5 D’s) [10]

<table>
<thead>
<tr>
<th>D</th>
<th>Diplopia</th>
</tr>
</thead>
<tbody>
<tr>
<td>D</td>
<td>Dysphagia</td>
</tr>
<tr>
<td>D</td>
<td>Dysarthria</td>
</tr>
<tr>
<td>D</td>
<td>Dizziness – vertigo, nystagmus</td>
</tr>
<tr>
<td>D</td>
<td>Drop attacks (syncope)</td>
</tr>
</tbody>
</table>

Contralateral loss of pain and temperature
Bilateral spasticity
Facial numbness / paresthesias

- **Wallenberg Syndrome (Lateral Medullary Syndrome)** = Ipsilateral absence of facial pain and temperature, with contralateral loss of these senses over the body; ataxia; Horner syndrome; dysphagia and dysarthria (ipsilateral CN V, IX, X, XI involvement)

BASILAR ARTERY OCCLUSION

- Coma
- Severe quadriplegia
- *Locked in syndrome* (pontine lesion → complete muscle paralysis except for upward gaze)

CEREBELLAR INFARCTION

- N/V/HA/Neck pain
- Central Vertigo
- CN abnormalities often present
- Drop attacks
- 6 to 8 hours delay in cerebral edema → ↑ brainstem pressure → decrease LOC

CVA PEARLS

- Intracerebral lesions - gaze = TOWARDS the affected side
- Brainstem abnormalities - gaze = AWAY from the affected side
- Dysconjugate gaze (failure of the eyes to turn together in the same direction) [10, 5th ed., pg 141]
  - Vertical plane = Pontine or Cerebellar lesions
  - Horizontal plane = drowsiness, sedated states (ETOH intoxication)

STROKE MIMICS


<table>
<thead>
<tr>
<th>MI</th>
<th>Migraine (hemiplegic migraine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hypo or Hyperglycemia</td>
</tr>
<tr>
<td>E</td>
<td>Epilepsy (focal seizures)</td>
</tr>
<tr>
<td>M</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>I</td>
<td>Infections – CNS (encephalitis, meningitis or abscess)</td>
</tr>
</tbody>
</table>
STROKE CAUSES IN YOUNG PATIENTS

Mnemonic - (MI HEMI 7 C’s) [Step Up to USMLE Step 2, Van Kleunen JP. Lippincott, 2nd Edition 2008 from Dr Postel, with modifications]

1. Cocaine
2. Cancer (brain tumor)
3. Cardiogenic Emboli
4. Coagulation (sickle cell)
5. CNS infection (septic emboli)
6. Congenital vascular lesion
7. Consanguinity (genetic disease like Von Hippel-Lindau (VHL) syndrome, neurofibromatosis)

LACUNAR INFARCTS

[10, 5th ed., pg. 1434]

- Small vessel strokes (DM, HTN); 80-90% patients have HTN
- More common in African-American patients
- Most common sites: subcortical structures of cerebrum (BG, thalamus, internal capsule), & brainstem (pons)
- Most common lacunae syndromes = pure motor or pure sensory strokes or ataxic hemiparesis
- Subcortical, so rarely → cognitive deficits, aphasia, LOC, simultaneous motor-sensory findings or memory impairment

ALTEPASE (tPA) IN CVA

>18 y/o
Ischemic stroke
Time of symptom onset to drug administration < 4.5 hours [Stroke, 2009;40:2945-2948]
No contraindications to tPA

tPA / HEPARIN

tPA dose = 0.9 mg/kg, max 90mg
First 10% bolus over 1 min, remaining infused over next 60 min
No not administer heparin or ASA during the first 24 hrs of fibrinolytic therapy [AHA, 2005 page 52]

CEREBRAL PERFUSION PRESSURE

CPP = MAP – ICP
Ideal CPP = > 70 mmHg
Normal intracranial pressure (ICP) < 20 mmHg

TIA

Focal symptoms, usually weakness or numbness, resolve within < 24 hours
Majority of TIAs last less than 30 minutes
(New proposed definition = A brief episode of neuro dysfunction caused by focal brain or retinal ischemia with clinical symptoms lasting < 1 hour without evidence of infarction)
ABCD2

A simple score to identify individuals at high early risk of stroke after transient ischemic attack

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<table>
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<tbody>
<tr>
<td>A</td>
<td>Age</td>
</tr>
<tr>
<td>B</td>
<td>Blood pressure &gt; 140/90 mmHg</td>
</tr>
<tr>
<td>C</td>
<td>Clinical features</td>
</tr>
<tr>
<td>D</td>
<td>Symptom Duration</td>
</tr>
<tr>
<td>D</td>
<td>Diabetes</td>
</tr>
</tbody>
</table>

Total scores ranged from 0 (lowest risk) to 7 (highest risk)

Stroke risk at 2 days, 7 days, and 90 days:
- Scores 0-3: low risk
- Scores 4-5: moderate risk
- Scores 6-7: high risk

Go to www.mdcalc.com/abcd2-score-for-tia → will calculate ABCD2 score for you and give you stroke risk at 2 days, 7 days, and 90 days

For example according to the validation study, an ABCD2 score of 4-5 points = Moderate Risk
- 2-Day Stroke Risk: 4.1%.
- 7-Day Stroke Risk: 5.9%.
- 90-Day Stroke Risk: 9.8%.

OCULOVESTIBULAR TESTING (COLD CALORICS) - DIRECTION OF FAST COMPONENT
Mnemonic - (COWS)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>Cold</td>
</tr>
<tr>
<td>O</td>
<td>Opposite</td>
</tr>
<tr>
<td>W</td>
<td>Warm</td>
</tr>
<tr>
<td>S</td>
<td>Same</td>
</tr>
</tbody>
</table>

Exam of comatose patient (Test the integrity of the brainstem) →

Inject 50 cc ice water → TM, normal =
1) **Rapid** nystagmus away (**Opposite**) from the irrigated ear
2) **Slow** compensatory nystagmus toward the irrigated side

- If the eye does not move in any direction, despite bilateral testing the brainstem is structurally or physiologically functionless
- Absence of oculovestibular reflexes = severe hypo-thermia, drug overdose and brainstem herniation. [12, 4th ed., pg. 228]
- If warm water is used = opposite will occur. The fast component of nystagmus = **toward** (**Same**) irrigated side, and the slow component will be away from the irrigated side.
OCULOCEPHALIC RESPONSE (DOLL’S EYES MANEUVER)

Tests integrity of pontine gaze centers; clear c-spine first; + if involuntary movement of the eyes upward/downward on passive flexion/extension of patient’s head.

GLASGOW COMA SCORE

<table>
<thead>
<tr>
<th>Eye Opening (4)</th>
<th>Verbal Response (5)</th>
<th>Motor Response (6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 = Spontaneous</td>
<td>5 = Alert and oriented</td>
<td>6 = Follows commands</td>
</tr>
<tr>
<td>3 = To voice</td>
<td>4 = Disoriented conversation</td>
<td>5 = Localizes pain</td>
</tr>
<tr>
<td>2 = To pain</td>
<td>3 = Speaking, but not coherent</td>
<td>4 = Movement or withdrawal to pain</td>
</tr>
<tr>
<td>1 = None</td>
<td>2 = Moans or unintelligible words</td>
<td>3 = Decorticate flexion</td>
</tr>
<tr>
<td></td>
<td>1 = None</td>
<td>2 = Decerebrate extension</td>
</tr>
</tbody>
</table>

HORNER SYNDROME

Mnemonic - (SPAM)

Sympathetic nerve impulses are disrupted and the pupil constricts due to more parasympathetic than sympathetic stimulation.[12, 6th ed., pg. 1463]

<table>
<thead>
<tr>
<th>S</th>
<th>Sunken eyeball (enophthalmos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>Ptosis</td>
</tr>
<tr>
<td>A</td>
<td>Anhidrosis</td>
</tr>
<tr>
<td>M</td>
<td>Miosis</td>
</tr>
</tbody>
</table>

Causes = internal carotid artery dissection, tumors, CVA, Pancoast tumor, herpes Zoster, trauma, Wallenberg Syndrome.

ACUTE TRANSVERSE MYELITIS (ATM)

1. Back pain
2. Weakness of legs and arms
3. Transverse sensory impairment
4. Bowel and bladder dysfunction (incontinence or constipation/ retention)

- 1-4 new cases per million people per year
- All ages with bimodal peaks between the ages of 10-19 and 30-39 years of age
- 1/3 = no sequelae; 1/3 = moderate degree and 1/3 = severe degree of permanent disabilities
- Rapid progression = poor recovery
- Inflammation or demyelination of the spinal cord
- Several segments involved
- Bilateral findings
- Thoracic cord region 60-70%
- Causes: unknown, 30% follow viral infection[10, 5th ed. pg1501]
- Order spine MRI and CSF (elevated protein)
- Fever/back pain, ddx = spinal epidural abscess (may skip a level)
- Back pain with neuro symptoms ddx = malignancy or hemotoma
- ↑ reflexes, sensory > weakness, clonus and Babinski ddx = MS (consider adding brain MRI)
INCOMPLETE SPINAL CORD LESIONS

Central cord syndrome: usually in the elderly when the neck is subjected to hyperextension The ligamentum flavum buckles into the cord → contusion to the central portion of the cord → neurological deficits in upper extremity >>> lower extremities

Mnemonic - (MUD-E) [http://boringem.org/2015/11/16/a-boring-guide-to-spinal-cord-syndromes/]

| M | Motor > Sensory |
| U | Upper extremity > Lower extremity |
| D | Distal > Proximal |
| E | Extension injury |

Anterior cord syndrome: flexion injuries → anterior cord compression → paralysis and pain-temperature loss distal to the lesion. Preservation of the Posterior Columns (fine touch, conscious proprioception and vibratory sense)

Brown-Sequard syndrome: hemisection of the spinal cord usually from penetrating GSW or knife wound, may be seen in lateral mass fractures of cervical spine →
- ipsilateral motor paralysis and loss of position-vibratory sensation
- contralateral sensory (pain and temperature) loss

SPINAL CORD TRACTS
Mnemonic - (SCALP)

<table>
<thead>
<tr>
<th>TRACT</th>
<th>FUNCTION</th>
<th>SITE OF Crossover</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>Spinocebeellar tract</td>
<td>Muscle tone Unconscious proprioception</td>
</tr>
<tr>
<td>C</td>
<td>Corticospinal tract</td>
<td>Voluntary motor Medulla</td>
</tr>
<tr>
<td>A</td>
<td>Anterior Spinothalamic</td>
<td>Crude touch</td>
</tr>
<tr>
<td>L</td>
<td>Lateral Spinothalamic</td>
<td>Pain Temperature</td>
</tr>
<tr>
<td>P</td>
<td>Posterior columns</td>
<td>Fine touch Conscious proprioception Vibratory sense</td>
</tr>
</tbody>
</table>

- The maximum neurologic deficit after blunt spinal cord trauma is seen over many hours and is not seen immediately
- Factors that worsen spinal cord injury [10, 5th ed., pg 345]
  Hypoxia, hypoglycemia, ↓ BP, hyperthermia, mishandling by medical personnel

CAUDA EQUINA SYNDROME

- The spinal cord ends at L1-L2 vertebrae → The most distal of the spinal cord = conus medullaris, distal to this → collection of horsetail-like nerve roots = cauda equina (Latin for horse’s tail) → Nerve root injury rather than a true spinal cord injury
- Most consistent finding = urinary retention [10, 5th ed., pg 1499]
• Present with fecal or urinary incontinence (overflow), impotence, distal motor weakness and
• Sensory loss = saddle distribution over the perineum
• Common cause is ruptured disc, most common L4-L5; other causes = tumors, trauma, vascular
• Treatment = surgery; steroids controversial

**COMPLETE SPINAL CORD SYNDROME ACUTE OR SUBACUTE**

Total loss of sensory, autonomic and voluntary motor distal to spinal cord level of injury
DTRs present, may be ↑ or ↓

**Spinal Shock triad** → ↓ BP, ↓ HR, and peripheral vasodilation resulting from autonomic dysfunction and the interruption of Sympathetic Nervous System control

**Spinal Shock** → loss of bulbocavernous reflex (reflex contraction of anal sphincter in response to squeezing the glans penis or tugging on an indwelling Foley catheter)
  • Associated head injury occurs in about 25% of patients with spinal cord injury
  • Judicious fluids – avoid pulmonary edema; atropine for bradycardia; rarely dopamine

**INSUFFICIENT EVIDENCE TO SUPPORT STEROIDS IN CORD INJURY**

ATLS 8th Ed. 2008 [J Trauma 2008;64:1638-1650]

In case old school doc /institution requests / or test questions lag ... the steroid protocol →

**Steroids**: Treatment must be started within 8 hours of injury [12, 6th ed., pg. 1581]

1) Methylprednisolone 30 mg/kg bolus over 15 minutes
2) Followed by 45 minutes pause
3) Infusion of Methylprednisolone at 5.4 mg/kg/h is continued for 23 hours

Steroid complications: thromboembolism, sepsis, pneumonia, wound infection, delayed healing, GI bleed

**CRANIAL NERVES**

**Mnemonic** - *(Oh, Oh, Oh, To Touch A Funky Vest Gives Very Amazing Happiness)*

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>O</td>
<td>I. Olfactory nerve</td>
</tr>
<tr>
<td>O</td>
<td>II. Optic nerve</td>
</tr>
<tr>
<td>O</td>
<td>III. Oculomotor nerve – EOMs, pupillary constriction via PNS, upper lid elevation (levator palpebrae)</td>
</tr>
<tr>
<td>T</td>
<td>IV. Trochlear nerve (LR6SO4) → superior oblique mm → eye downward and laterally Patients compensate for CN IV compression by head tilt</td>
</tr>
<tr>
<td>T</td>
<td>V. Trigeminal nerve – chew, face/mouth touch and pain</td>
</tr>
<tr>
<td>A</td>
<td>VI. Abducens nerve – lateral rectus muscle → eye laterally</td>
</tr>
<tr>
<td>F</td>
<td>VII. Facial nerve – face muscles, tears, saliva, taste</td>
</tr>
<tr>
<td>V</td>
<td>VIII. Vestibulocochlear nerve/Auditory nerve – hearing, equilibrium</td>
</tr>
<tr>
<td>G</td>
<td>IX. Glossopharyngeal nerve – taste, senses carotid BP</td>
</tr>
<tr>
<td>V</td>
<td>X. Vagus nerve – senses aortic BP, slows HR, stimulates digestive organs, taste, Unilateral palatal elevation</td>
</tr>
<tr>
<td>A</td>
<td>XI. Accessory nerve/Spinal accessory nerve – trapezius, SCM, swallowing</td>
</tr>
<tr>
<td>H</td>
<td>XII. Hypoglossal nerve – tongue motor</td>
</tr>
</tbody>
</table>
CORD INJURY SENSORY LEVELS

<table>
<thead>
<tr>
<th>Sensory Level</th>
<th>Corresponding Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clavicle</td>
<td>C5</td>
</tr>
<tr>
<td>Thumb</td>
<td>C6</td>
</tr>
<tr>
<td>Index &amp; middle finger</td>
<td>C7</td>
</tr>
<tr>
<td>Ring &amp; small finger</td>
<td>C8</td>
</tr>
<tr>
<td>Nipples</td>
<td>T4</td>
</tr>
<tr>
<td>Umbilicus</td>
<td>T10</td>
</tr>
<tr>
<td>Medial thigh</td>
<td>L2-3</td>
</tr>
<tr>
<td>Knee</td>
<td>L4</td>
</tr>
<tr>
<td>Lateral calf</td>
<td>L5</td>
</tr>
<tr>
<td>Perineum</td>
<td>S2,3,4</td>
</tr>
</tbody>
</table>

NERVE INJURIES
Mnemonics - (DR CUMA) [ANK]

<table>
<thead>
<tr>
<th>Letter</th>
<th>Mnemonic</th>
</tr>
</thead>
<tbody>
<tr>
<td>D</td>
<td>Drop wrist</td>
</tr>
<tr>
<td>R</td>
<td>Radial nerve</td>
</tr>
<tr>
<td>C</td>
<td>Claw hand</td>
</tr>
<tr>
<td>U</td>
<td>Ulnar nerve</td>
</tr>
<tr>
<td>M</td>
<td>Median nerve</td>
</tr>
<tr>
<td>A</td>
<td>Ape hand</td>
</tr>
</tbody>
</table>

MEDIAN NERVE

1) Wrist flexion and the Flexor Digitorum Superficialis (FDS), finger flexion
2) Median nerve branches → to anterior interosseus nerve (controls deep finger flexors in the forearm - FDProfundus, Flexor Pollicis Longus, and Pronator Quadratus
3) → sensory branch provides sensation to most of palm plus a motor branch →
4) → recurrent branch of the median nerve → innervates thenar motor muscles of the thumb

Evaluation of Median Nerve
Test anterior interosseus nerve → make a circle or, “OK” sign, with thumb/index finger
Test recurrent branch of median nerve → ABDuction of the thumb
Sensation on radial side of palm – from the thumb to radial half of ring finger
Eponym “LOAF weakness” = Lumbricals (flex MCP), thumb: Opposition, ABDuction, Flexion

ULNAR NERVE

Controls intrinsic muscles and provides sensation to the little finger and ulnar half of ring finger
Eponym for ulnar n palsy “Tardy Ulnar Palsy” – palsy can manifest years after injury.
RADIAL NERVE

Wrist extension then branches → posterior interosseus n. → finger extension
Dorsal hand sensation. Test sensation over the dorsum of the thumb-index finger web space
Eponym for radial n palsy = “Saturday Night Palsy “ and “Bridegroom Palsy”

LIFE THREATENING CAUSES OF ALTERED MENTAL STATUS
Mnemonics - (WHHHIMP) [ANK, 14]

| W | Wernicke’s encephalopathy (give thiamine) |
| H | Hypoglycemia |
| H | HTN encephalopathy |
| H | Hypoxia |
| I | Intracerebral hemorrhage |
| M | Meningitis |
| P | Poisonings |

CAUSES OF COMA
Mnemonic - (AEIOU SET TIPS) [ANK, 12, pg. 230 with modifications]
Note: AEIOU is the mnemonic used for “Dialysis Criteria”

| A | Alcohol and other drugs – (opiates) |
| E | Endocrine (↓↑ glucose, myxedema coma -↓ T3) |
| I | Increased BP or NH3 (Hypertensive or Hepatic Encephalopathy) |
| O | Oxygen |
| U | Uremia |

| S | SAH/Stroke |
| E | Electrolytes (Na+, Ca++) |
| T | Trauma |

| T | Temperature (heat stroke) |
| I | Infection |
| P | Psychiatric |
| S | Space occupying lesions Shock Seizure → Post Ictal |
ALTERED LEVEL OF CONSCIOUSNESS TREATMENT OPTIONS
Mnemonics - (DON’T) [28 Volume 31, Number 12]

| D | Dextrose - one AMP of D 50% |
| O | Oxygen |
| N | Narcan (Naloxone) 2 mg IV |
| T | Thiamine 100 mg IV, give BEFORE glucose |

CAUSES OF PERIPHERAL NEUROPATHY
Mnemonics - (HIT DANG SPARTEN) [ANK]

| H | Hereditary |
| I | Infectious (Diphtheria, Mono, syphilis, hepatitis, HIV) |
| T | Toxic (heavy metals: lead, arsenic, mercury, thallium) |

| D | Diabetes |
| A | Alcohol |
| N | Nutritional Deficiencies: Thiamine (B1), Niacin (B3), Cobalamin (B12), Vit E |
| G | Guillain-Barre syndrome |

| S | Systemic (SLE, PA, sarcoid, hypothyroid) |
| P | Porphyria |
| A | Amyloid |
| R | Renal failure |
| T | Trauma |
| E | Emaciation (see nutritional above) |
| N | No known cause |

NORMAL PRESSURE HYDROCEPHALUS - TRIAD
Mnemonic - (Wet - Wacky Wobbly) [ANK]

1) Wet (Urinary incontinence)
2) Wacky (Mental confusion)
3) Wobbly (Ataxia)

HA and papilledema are ABSENT. Drop attacks may occur. Normal pressure hydrocephalus may follow SAH, meningitis or head trauma, although the cause is usually unknown [10, 4th ed., pg. 2223]

PSEUDOTUMOR CEREBRI (IDIOPATHIC INTRACRANIAL HYPERTENSION)
Most frequently in young, overweight women between the ages of 20 and 45
Headache is the most common presenting complaint; papilledema on exam
Causes = pregnancy, medications (OCPs, steroids, vitamin A) [10, 5th ed., pg. 1518]
TABES DORSALIS
Progressive demyelination of Posterior Column and Dorsal Nerve Roots. (Know how to distinguish Tabes Dorsalis from Normal Pressure Hydrocephalus)

1) Ataxia
2) Urinary incontinence and loss of sexual function
3) Leg pain (Lancinating - appearing suddenly, spreading rapidly, and disappearing) often is an early symptom

Other neurologic presentations: progressive loss of pain sensation, loss of peripheral reflexes, impairment of vibration and position senses

NEUROLOGY PEARLS

- Status epilepticus has an associated mortality of up to 20%
- Bell’s Palsy = CN VII Palsy; lose ipsilateral forehead strength; steroids, antivirals [12, 6th ed. pg 1421]
- Most common CN involved in cephalic tetanus = CN VII (facial nerve)
- If you suspect SAH and head CT is normal → perform LP; note: 12 hours before you can see xanthochromia
- Possible complications weeks after SAH: seizure, cerebral artery vasospasm, hydrocephalus, rebleeding
- Basilar skull fracture = 1) Raccoon eye (peri orbital ecchymosis) 2) Battle sign (mastoid ecchymosis) 3) hemotympanum 4) CSF rhinorrhea/otorrhea.
- Spinal shock = hypotension and bradycardia; Tx = Trendelenburg position and fluid
- ↑ICP → Cushing Reflex = ↑ BP and ↓ HR, with respiratory irregularities
- Most common presenting symptom of Multiple Sclerosis (MS) = optic neuritis
- Most common cause of neonatal (0-28 days) meningitis = Group B Strep, E. Coli and Listeria [12]
- Most common cause of meningitis 1st – 3rd month of life = GBS, Listeria and now include Haemophilus, Strep pneumo and Neisseria meningitidis [12]
- Most common cause of meningitis 3 months-18 years = Strep pneumo, Haemophilus, and Neisseria [12]
- Most common cause of focal encephalitis in patients with AIDS = Toxoplasma gondii (obligate intracellular parasite). Contrast CT → ring-enhancing lesions with surrounding areas of edema; seizures common and 80% focal neurologic deficits [10, 5th ed. pg 1849]
  Treatment = Pyrimethamine + Folinic acid (prevents heme toxicity from pyrimethamine) + Sulfadiazine or Bactrim IV [Sanford, 2009, pg. 129]
- Most common opportunistic CNS fungal infection = Cryptococcus neoformans; ↑↑ ICP; seizures uncommon CSF cryptococcal antigen = 100 % Sn & Sp; treatment = Amphotericin IV + Flucytosine po [Sanford, 2009, pg. 103]
- HIV encephalopathy or AIDS Dementia Complex = 1/3 patients with HIV; early = impairment in recent memory, difficulty concentrating; later MS changes and seizures [10, 5th ed. pg 1849]
NEUROLOGY PEARLS (CONT)

- *Listeria monocytogenes* risk > 60x ↑ in AIDS patients and 3/4 present = meningitis [Sanford, 2009, pg. 10]

- > 1st year of life, you can assess nuchal rigidity. Two signs of meningeal irritation = Kernig’s and Brudzinski’s

- Kernig’s sign with patient lying supine, hip and knee flexed to about 90°, knee extension → in meningeal irritation → neck pain [12, 6th ed., pg. 741]

- Brudzinski’s = with the patient supine, passive flexion of the neck → involuntary flexion in the hips, if there is meningeal irritation [12, 6th ed., pg. 741]

- DeCORticate posturing: hyperextension of legs with flexion of the arms; remember arms are in flexion with hands over the heart (“cor”) in de “cor”ticate posturing – results from damage to the descending motor pathways above the central midbrain [9]

- Decerebrate posturing: hyperextension of both upper and lower extremities – refers to damage to the midbrain and upper pons; is a grave sign [9]

- Meniere’s Disease = 1) Vertigo 2) Unilateral diminished hearing and 3) Tinnitus intermitent

- Ddx acoustic neuroma = tinnitus is constant with acoustic neuroma; Meniere’s = true vertigo vs acoustic neuroma patient describes imbalance and dysequilibrium

- Peripheral Vertigo: Nystagmus = unidirectional and fatiguing and suppressed with fixation. Usually have hearing loss. Caloric testing shows abnormal function on involved side. N/V/diaphoresis common

- Examples: Benign paroxysmal positional vertigo (BPPV), Meniere’s disease, vestibular neuronitis, perilymph fistula, labyrinthitis [12, 6th ed, pgs. 1404-1408]

- BPPV, vestibular neuronitis → NO hearing loss

- Treatment options: 1) Sedative: diazepam 2) Antiemetic: hydroxyzine (Vistaril), promethazine (Phenergan), metoclopamide (Reglan) 3) Anticholinergic: Scopolamine patch 4) Anti-histamines: Antivert, Benadryl 5) Calcium antagonists: Nimodipine, Cinnarizine, Flunarazine [Table 231-4 in ref. 12, 6th ed, pg. 1404]

- Central vertigo (disorders affecting the cerebellum or brainstem): Nystagmus = multidirectional and Non-fatiguing and not suppressed with fixation. Caloric testing often normal

- Associated N/V/diaphoresis = rare and hearing loss is unlikely

- Examples = brainstem stroke, multiple sclerosis, acoustic neuroma, cerebellar stroke, Wallenberg Syndrome, vertebrobasilar insufficiency, vertebral artery dissection.

- The most common cause of delirium in the elderly = medications. 22-39% of cases. Delirium symptom onset acute vs Dementia progressive over months. Alterations in sleep-wake cycles are common

- Delirium = various hallucinations (visual, auditory, olfactory, tactile, gustatory) vs Functional Psychosis patient who only experience auditory hallucinations

- EEG = abnormal; bilateral diffuse symmetric abnormalities; relative generalized slowing with or without superimposed fast activity [10, 5th ed., pg. 1471, 6th ed., 1645-64]

- One of the hallmarks of Acute Delirium = short-term memory impairment; remote memory preserved

- Post-traumatic seizures: more common PEDS > adults; if dura disrupted the incidence of seizures with neuro deficits ↑; incidence of seizures ↑ than general population
- Dilantin loading dose = 18 to 20 mg/kg IV with infusion < 50 mg/min
- Treatment for Trigeminal Neuralgia = oral Carbamazepine (Tegretol)
- Most common ocular motor palsy = **CN VI** (abducens nerve) innervates the ipsilateral lateral rectus
  Causes = aneurysm, vascular disease (DM, HTN, atherosclerosis), trauma, neoplasm, MS, MG, meningoitis, cavernous sinus mass, thyroid eye disease, ↑ ICP → downward displacement of the brainstem (30% of patients with pseudotumor cerebri have an isolated abducens nerve palsy)
- **CN III (Oculomotor) Palsy causes** = Infarction, hemorrhage, neoplasm, aneurysm, abscess, meningitis, vascular disease (DM, microvascular ischemia/atherosclerosis), cavernous sinus mass, thyroid eye disease
- Pinpoint pupils = Pontine hemorrhage; also, see TOX - (COPS) 2
- SLE CNS manifestations = seizures, CVA, psychosis, migraines and peripheral neuropathy
- Initial work-up of CSF shunt malfunction = CT head and shunt series (consider in any patient with shunt and decreased LOC)
- **Myasthenia gravis (MG)** = autoimmune disease - antibodies directed against the acetylcholine receptor at NMJ → diplopia and ptosis most common presenting symptom, also = dysphagia, dysarthria then muscle weakness
  Females > males; teens to early 30’s; thymomas thought to ↑ acetylcholine receptor antibodies
  Tension test helps with diagnosis. Edrophonium (Tensilon) temporarily blocks acetylcholinesterase → prolongs muscle stimulation and temporarily improves strength (measure distance from the upper to the lower eyelid in the most severely affected eye before and after edrophonium). 1 – 2 mg test dose
- Treatment of MG = **Anticholinesterase medications**
  ▫ Neostigmine (Prostigmin) and Pyridostigmine (Mestinon)
- Lambert Eaton with repeated stimulation → increase strength, opposite of MG
- **Nerve Agents** (sarin, soman, tabun, GF, VX) are organophosphates which are potent inhibitors of acetylcholinesterase → SLUDGE BAM syndrome. Treatment = atropine and 2-PAM (pralidoxime)
- **Botulism** = neurotoxins → block the release of acetylcholine → descending symmetrical paralysis, ptosis, generalized weakness UE > LE, proximal muscles > distal, dizziness, dry mouth, diplopia, dilated/ fixed pupils & blurred vision, dysphonia, dysarthria, dysphagia, and respiratory failure. ↓ DTR’s
  Treatment = antitoxin → binds circulating toxin only; intubate if vital capacity <30%; saline enema to cleanse GI tract of residual toxin
- **Guillain-Barre Syndrome (GBS)** = ascending paralysis; in most cases GBS is caused by an autoimmune attack on myelinated motor nerves. Variable sensory findings; subjective sensory disturbances (numbness and tingling of lower extremities); urinary retention may occur - ddx = spinal cord lesion and cauda equina syndrome
- **GBS** – order vital capacity
- Hallmark of **GBS** = loss of deep tendon reflexes (↓ DTR’s). CSF = high protein, normal glucose and cell count.
  Treatment = plasma exchange or intravenous immunoglobulin [12, 6th ed, pg. 1420]
- Most common CNS complication of pertussis = seizures 3%; other = encephalopathy and ICH
- **Tick Paralysis** = mimics GBS
# Causes of Microcytic Hypochromic Anemias

Mnemonic - **(TAILS)** [ANK, provided by Dr. Matt Jordan]

<table>
<thead>
<tr>
<th>T</th>
<th>Thalassemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Anemia of chronic disease</td>
</tr>
<tr>
<td>I</td>
<td>Iron deficiency</td>
</tr>
<tr>
<td>L</td>
<td>Lead Poisoning</td>
</tr>
<tr>
<td>S</td>
<td>Sideroblastic</td>
</tr>
</tbody>
</table>

# Causes of Thrombocytosis

Mnemonic - **(Crazy Navy Men SHIP)** [ANK]

<table>
<thead>
<tr>
<th>Crazy</th>
<th>Cirrhosis</th>
<th>CML</th>
<th>Collagen vascular Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Navy</td>
<td>Neoplasm (GI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>Myelofibrosis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>S</th>
<th>Splenectomy (post)</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hemophilia</td>
</tr>
<tr>
<td>I</td>
<td>Infection</td>
</tr>
<tr>
<td>P</td>
<td>Pancreatitis</td>
</tr>
</tbody>
</table>

| P | Postpartum | Post trauma | Polycythemia vera |

# Causes of Eosinophilia

Mnemonic - **(NAAACP)**

<table>
<thead>
<tr>
<th>N</th>
<th>Neoplasms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Allergy</td>
</tr>
<tr>
<td>A</td>
<td>Asthma</td>
</tr>
<tr>
<td>A</td>
<td>Addison’s</td>
</tr>
<tr>
<td>C</td>
<td>Collagen vascular disease (CVD)*</td>
</tr>
<tr>
<td>P</td>
<td>Parasites</td>
</tr>
</tbody>
</table>

*CVDs = systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), progressive systemic sclerosis (PSS) or scleroderma (SD), dermatomyositis (DM) and polymyositis (PM), ankylosing spondylitis (AS), Sjögren syndrome (SS), and mixed connective-tissue disease (MCTD)*

Churg-Strauss syndrome (CSS), or allergic granulomatous angiitis = Eosinophilia; Wegener granulomatosis, another ANCA vasculitic syndrome similar to CSS = NO eosinophilia
THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP)

Mnemonic - (FAT RN)

<table>
<thead>
<tr>
<th>F</th>
<th>Fever in 90%</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Anemia - Microangiopathic hemolytic anemia (MAHA) - schistocytes on smear</td>
</tr>
<tr>
<td>T</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>R</td>
<td>Renal failure</td>
</tr>
<tr>
<td>N</td>
<td>Neurologic sequelae (HA, confusion, CN palsies, seizures and coma)</td>
</tr>
</tbody>
</table>

- Pentad only present in 40%
- ↑LDH, ↑IBIL, ↑Reticulocyte count, ↓haptoglobin, ↑Cr, normal DIC panel
- Abdominal pain
- Affect any age but majority 10 to 40 years
- Women 60% of cases
- 1/3 of patients who survive initial episode experience relapse within 10 years
- Survival rate = 80-90% with early diagnosis and treatment. 95% MR with no treatment
- Treatment = Intravenous (IV) plasma exchange, also called plasmapheresis

IDIOPATHIC THROMBOCYTOPENIC PURPURA (ITP)

Syn = primary immune thrombocytopenic purpura and autoimmune thrombocytopenic purpura

- Patients having antibodies to platelet membrane glycoproteins
- Most often in children (usually 2 to 6 yrs); male = female; follows viral infection
- Usually isolated thrombocytopenia; M&M low; recovery may take weeks
- Treatment is supportive as the course is self limited with 90% spontaneous remission
- Platelet count < 20,000 to 30,000 μL require treatment
- Platelet count < 50,000 μL with bleeding or risk factors for bleeding require treatment
- Treatment = steroids; life threatening bleeding high dose steroids + IV immunoglobulin
- Add conjugated estrogen 25 mg IV x once if uterine bleeding
- Admit if ITP-related bleeding and consider admit if platelet count < 20,000 μL

HEMOLYTIC UREMIC SYNDROME (HUS)

Use TTP Mnemonic with Pearls below:

- Can occur at any age however majority of cases < 5 years old
- Low grade fever 5-20%; abdominal pain
- Renal manifestations more prominent than neurologic ones in HUS vs TTP
- 75% of cases post-infectious: E. coli 0157:H7, Shigella, Strep. pneumoniae
- Majority = gastroenteritis up to 2 weeks before illness. E. coli 0157:H7 [12, 6th ed., pg. 820]
- 15% children vs 5% adults infected with E. coli 0157:H7 go on to develop HUS
- Mild HUS → steroid therapy may be beneficial
- Severe disease plasmapheresis = equivocal results [12, 6th ed., pg. 1347]
- Hemodialysis if renal failure (shorter duration = better chances of recovery from HUS)
- Infected with E. coli 0157:H7 should not be treated w/ antimotility drugs → ↑risk HUS
**METS TO BONE**

**Mnemonic** - *(Many Kinds of Tumors Leaping Promptly To Bone)*

<table>
<thead>
<tr>
<th>Many</th>
<th>Myelomas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kinds</td>
<td>Kidney</td>
</tr>
<tr>
<td>Of</td>
<td>Ovary</td>
</tr>
<tr>
<td>Tumors</td>
<td>Testicular</td>
</tr>
<tr>
<td>Leaping</td>
<td>Lung Lymphoma</td>
</tr>
<tr>
<td>Promptly</td>
<td>Prostate</td>
</tr>
<tr>
<td>To</td>
<td>Thyroid</td>
</tr>
<tr>
<td>Bone</td>
<td>Breast</td>
</tr>
</tbody>
</table>

- Mets most commonly to pedicles on thoracic vertebrae
- Most common site of malignant spinal cord compression = thoracic vertebrae.
- Most common symptom of spinal cord compression = pain (WORSE recumbent position)
- Most common early finding of spinal cord compression = motor weakness
- Check CT, MRI or myelogram; Treatment options = steroids, radiation and surgery

**HEME /ONC PEARLS**

- Cancer with JVD, dyspnea →
  - **Superior vena cava syndrome** = facial plethora, dilated veins chest/arms, HA
  - Most common cause = Small (oat) cell lung CA > bronchogenic (squamous) cell lung CA > lymphoma; CXR = right sided mass; discuss with ONC – consider: radiation, steroids, lasix, endovascular shunts, and thrombolytics if thrombotic etiology
  - **Cardiac tamponade;** also ↓BP
  - Massive PE → acute cor pulmonale; also ↓BP

- Cancer with back pain → **Spinal cord compression** treatment = Decadron 10mg IV, radiation

- Cancer with Paraneoplastic syndromes:
  - ↓Na+ → SIADH (euvolemic hyponatremia); small cell Lung CA
  - ↑Ca2+ → Calcitonin
  - ↑Ca2+ → Parathormone → “Stones (kidney), Abdominal groans (constipation, N/V, anorexia, pancreatitis), Psychic moans (mental status changes, seizures, coma, Bones (pain))”
  - ↑ACTH → Cushing syndrome (↑ cortisol), ↑Na+, ↓K+, (small cell Lung CA)
  - ↑ACTH → Gonadotropins / gynecostasia
  - ↑Serotonin → Carcinoid syndrome
  - Thymoma → ↑ acetylcholine receptor antibodies → Myasthenia gravis
  - Lambert-Eaton myasthenic syndrome (LEMS) → autoimmune attack on pre-synaptic motor nerve voltage-gated calcium channels (VGCC) → decrease in amount of acetylcholine in synapse → proximal muscle weakness, ↓DTR’s & autonomic changes → (small cell Lung CA)

- FUO consider malignancy → lymphomas, acute leukemias, sarcomas, renal cell carcinomas, GI malignancies

- Neutropenic = neutrophil count < 500/mm3
• Emergent treatment of hyperviscosity syndrome secondary to symptomatic polycythemia = phlebotomy (not more than 500 ml of blood is removed and the volume is replaced with an equal volume of normal saline)
• Other Hyperviscosity Syndromes = Multiple Myeloma and Waldenstrom’s Macroglobulinemia (sludge immunoglobulins) and Leukemia (sludge wbc’s) tx = plasmapheresis or leukapheresis
• Tumor Lysis Syndrome = cell lysis → ↑ K+ (dysrhythmia), ↑ Phos, ↑ Uric Acid (renal failure) and subsequent ↓ Ca 2+ (muscle cramps/tetany); Treatment options: Chemo pretreatment with fluid and allopurinol
  Urine alkalinization controversial – improves uric acid diuresis, but may worsen ↓ Ca 2+ tetany
  Dialysis = Treatment of Choice

HEME PEARLS

• Vitamin K dependent factors = II, VII, IX, X
• Extrinsic pathway and Common Pathways = Measured by PT/INR
• Extrinsic = VII
• Common Pathway = I, II, V ---- remember
  • 1 x 2 = 2
  • 2 x 5 will get you to = 10
• Intrinsic Pathway = Measured by PTT
• Cryoprecipitate = vWF, Fibrinogen, Factor VIII and Factor XIII
• Prothrombin Complex Concentrate (PCC) (trade name Beriplex or Octaplex) = Vit K dependant coagulation factors II, VII, IX and X as well as protein C and S
• Desmopressin (DDAVP) = synthetic analog of vasopressin – causes release of vWF
  ▫ vWF → carries additional factor VII in the plasma
  ▫ vWF can increase factor VIII by 3-5x (onset 30 minutes)
  ▫ Dose = 0.3 mcg/kg/dose IV
  ▫ vWF → is also required for normal platelet adhesion
• Fresh Frozen Plasma (FFP) = contains all the plasma clotting factors
• Hemophilia A = Factor VII deficiency = most common cause of hemophilia in the US; 1 in 5,000 male births; (↑ PTT)
• Hemophilia B = Christmas disease = Factor IX deficiency; 1 in 30,000 male births; (↑ PTT)
• Hemophilia A and B = X-linked recessive disorder – therefore a disease of men
• Intracranial hemorrhage is the most common noninfectious cause of death in hemophiliacs
• von Willebrand’s disease = most common inherited bleeding disorder (autosomal dominant); ↑ bleeding time
• Most common cause of vaginal bleeding related to primary coagulation disorder = Von Willebrand’s disease
  [12, 5th edn, pgs. 673-674 and 1377-1382]
• Treatment of non-traumatic hemarthrosis in Hemophilia A patient =
  ▫ Desmopressin (DDAVP) or if N/A give Factor VIII 12.5 U/kg
  ▫ Most patients will require 25 U/kg every 24 hours x 2-3 days for most bleeds
  ▫ If severe bleeding, Factor VIII 50 U/kg
  ▫ Factor VIII = Each U/kg = an Increase of Factor by 2%

• Treatment of mild bleeding in von Willebrand’s disease = Desmopressin (DDAVP)

• Treatment of severe bleeding in von Willebrand’s disease = Cryoprecipitate

• 1 unit of platelets = will increase platelet count 5-10,000 in adults, 20,000/20kg in Peds
  Ask for single-donor platelets when possible (contain the equivalent of 6 units of random donor platelets)

• When platelet levels decrease < 20,000/uL = concerned about risk of spontaneous bleeding

• 1 unit PRBC will increase Hgb 1 gm

• Peds FFP = 30 cc/kg

• Most common cause of acute hemolytic transfusion reactions = transfuse wrong blood due to clerical error

• Most common adverse effect from blood transfusion = febrile, non-hemolytic reaction. Reaction to anti-leukocyte and antiplatelet antibodies (prevent by using washed RBCs)

• DIC most helpful labs = ↑ PT, ↓ platelet count and ↓ fibrinogen

• Heparin has selective use in DIC when fibrin deposits and thrombosis predominate. Heparin should be considered in purpura fulminans, retained dead fetus before delivery, giant hemangioma and acute promyelocytic leukemia [10, 5th ed., pg. 1698]

• Sickle cell patients = functional asplenia; risk of encapsulated organisms – S. pneumo. H. influenza, Salmonella

• Major cause of anemia worldwide = hookworm infection (Necator americanus); infective filariform larva penetrate skin → adult worms penetrate into intestinal mucosa and feed → luminal blood loss [10, 5th ed., pg 1871]

• Most common human enzyme defect = deficiency of RBC enzyme Glucose-6-phosphate dehydrogenase (G-6-PD); affects 1/10th of world population
  Oxidant stress → hemolytic anemia, renal failure, low platelets
  Causes: infections, metabolic acidosis (DKA), exposure to oxidant drugs and ingestion of fava beans
  Drugs associated = antimalarials (primaquine), nitrofurantoin, phenazinepyridine (pyridium), sulfas
  Self limited because only the older RBCs hemolyze [10, 5th ed., pg1676-1677]
CONGENITAL HEART DISEASE - CYANOTIC
Mnemonic - (5 Terrible T’s) [ANK]
Right to left shunts causing early cyanosis

<table>
<thead>
<tr>
<th>T</th>
<th>Truncus Arteriosus (single arterial trunk exits ventricular portion of heart)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Transposition of the Great Vessels</td>
</tr>
<tr>
<td></td>
<td>Most common cause of cyanosis or CHF within the first 3 days of life</td>
</tr>
<tr>
<td></td>
<td>Aorta originates from RV and pulmonary artery from the LV</td>
</tr>
<tr>
<td></td>
<td>Systemic veins drain → RA and pulmonary vein → LV</td>
</tr>
<tr>
<td></td>
<td>Tricuspid Valve Atresia</td>
</tr>
<tr>
<td></td>
<td>Tetralogy of Fallot (Most common cyanotic congenital heart disease in kids &gt; 4y/o)</td>
</tr>
<tr>
<td></td>
<td>Total Anomalous Pulmonary Venous Return</td>
</tr>
</tbody>
</table>

When trying to remember the 5 T's think of counting the fingers on your hand
[ANK from Drs. Lisa McQueen, Nathan Allen]

<table>
<thead>
<tr>
<th>1 finger</th>
<th>One arterial trunk exits the ventricle (Truncus Arteriosus)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 fingers</td>
<td>Cross middle finger over index finger) = 2 vessels transposed Transposition of the Great Vessels</td>
</tr>
<tr>
<td>3 fingers</td>
<td>Tricuspid Valve Atresia (3 = Tri)</td>
</tr>
<tr>
<td>4 fingers</td>
<td>Tetralogy of Fallot (4 = Tetra)</td>
</tr>
<tr>
<td>5 fingers</td>
<td>Palm open all (total) fingers up = Total Anomalous Pulmonary Venous Return Also, 5 = 5 words</td>
</tr>
</tbody>
</table>

TETRALOGY OF FALLOT
Mnemonic - (POSH) [ANK]

<table>
<thead>
<tr>
<th>P</th>
<th>Pulmonary stenosis (RV outflow tract obstruction - RVOTO)</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>Overriding aorta (Dextroposition of the aorta); the aortic valve is situated above the VSD with biventricular connection (connected to both the RV and LV)</td>
</tr>
<tr>
<td>S</td>
<td>Septal defect - VSD; holosystolic murmur</td>
</tr>
<tr>
<td>H</td>
<td>Hypertrophy – RV</td>
</tr>
</tbody>
</table>

CXR = boot-shaped heart
MANAGEMENT OF HYPERCYANOTIC OR TET SPELL
Mnemonic - (5P’s) [ANK from Dr. Angela McCormick]

- **Position** - Knees to chest or squatting → ↑ venous return to heart and ↑ SVR
- **Pain control** - O2 and Morphine 0.2 mg/kg SQ or IM per dose
- **Propranolol** 0.05 to 0.1 mg/kg IV or Esmolol (with consultation) → Relaxes infundibular muscle spasm that causes RVOTO
- **Phenylephrine** 10 μg/kg bolus followed by infusion 2 to 5 μg/kg/min → ↑ SVR → ↑ BP [12, 6th ed. pg. 761]
- **Prostaglandins in neonates may be lifesaving** = 0.1μg/kg/min [10, 5th ed. pg. 2283]

If hypercyanotic episode is not recognized and treated early it may be fatal. Other complications = seizures, cerebral thrombosis, profound lactic acidosis and cardiac dysrhythmias [12, 6th ed. pg. 761].

<table>
<thead>
<tr>
<th>CONGENITAL DISORDER</th>
<th>CV MANIFESTATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Downs</td>
<td>Atrial Septal Defect (ASD) SEM at left sternal border&lt;br&gt;Left to right intra-cardiac shunt; present &gt; 6 month = CHF</td>
</tr>
<tr>
<td>Turners</td>
<td>Coarctation of the aorta (↓pulse LEs, HTN UEs, SEM at cardiac base radiates → interscapula) = CHF</td>
</tr>
<tr>
<td>Rubella</td>
<td>Patient ductus arteriosus (PDA)&lt;br&gt;Left to right intra-cardiac shunt; present &lt; 6 months = CHF</td>
</tr>
</tbody>
</table>

CONGESTIVE HEART FAILURE
[12, 6th ed., pgs. 761-763]

Infants present with poor feeding, labored breathing and sweating,

Most common cause of CHF < 1 day = non-cardiac (↓H/H, ↓glucose, ↓O2, ↓Ca2+, sepsis, acidosis) or premature neonate with PDA

Most common cause of cyanosis or CHF within the first 3 days of life of life = Transposition of the Great Vessels (TGV)

Most common cause of CHF 1st week of life in full-term newborns = Hypoplastic LV (HPLV)

Most common cause of CHF 2nd week of life in full-term newborns = Coarctation of the aorta

VSD = CHF 4 to 12 weeks of life, unless complicated by other cardiac disease then earlier

TREATMENT OF CHF

Lasix 1 to 2 mg/kg IV<br>Digoxin 0.05 mg/kg per day, in infants up to 2 y/o. Give first digitalizing dose in ED (50% daily dose), followed by one-fourth of the daily dose IV at 6 to 8 hour intervals

Cardiogenic shock → inotropic agents<br>Dopamine or Dobutamine
If Inotropic Support Fails
Combination of Nitroprusside or NTG + Dopamine

- Blue baby = Terrible T’s
- Mottled or gray baby = Coarctation of the aorta or aortic stenosis
- Pink baby = VSD, PDA

**COMMON CAUSES OF NEONATAL SEPSIS / MENINGITIS (< 1 MONTH)**

**Mnemonic: GEL** [Sanford Guide 2015]

<table>
<thead>
<tr>
<th>G</th>
<th>Group B Strep. (Strep. agalactiae) Most common cause 49%</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>E. coli 18%</td>
</tr>
<tr>
<td>L</td>
<td>Listeria 7%</td>
</tr>
</tbody>
</table>

**TREATMENT OF NEONATAL MENINGITIS**

[Ampicillin (will cover Listeria) 50 mg/kg q 6 hrs (max dose 2 grams) +
Cefotaxime (Claforan) 200 mg/kg/day divided q 6-8 hrs (max dose 2 grams)]

or Amp + Gentamycin

If CSF pleocytosis and negative gram stain consider adding Acyclovir empirically for HSV

**MENINGITIS > 1 MONTH**

[Sanford Guide 2015]

1) Strep. pneumoniae
2) Neisseria meningitidis
3) H. influenzae

**TREATMENT OF MENINGITIS > 1 MONTH TO 50 YEARS**

[Dexamethasone + Ceftriaxone or Cefotaxime + Vancomycin
Ceftriaxone = 100 mg/kg (2 gm IV max) q 12 hrs
Dexamethasone = 0.15 mg/kg IV q 6 hrs x 2 to 4 days; give 15 min prior or con-comitant with first dose of antibiotic to prevent neurologic complications.
Vancomycin = 15 mg/kg IV q 6 hours; Adults max dose of 2-3 gm/day is suggested:
500 to 750mg IV q 6 hours]

Most common cause of aseptic meningitis in US = Enterovirus (coxsackie)

*Neisseria meningitidis* prophylaxis rule of 2s = 2 hours within 2 feet require prophylaxis; also if direct mucosal contact with patient’s secretions (mouth-mouth, intubation or nasotracheal suctioning) [10, 5th ed., pg. 1538]

If > 50 years or alcoholism or other debilitating associated diseases or impaired cellular immunity add, Ampicillin 2gm IV q 4h to regimen above cover possible *Listeria*
TYPICAL CSF CHARACTERISTIC OF NORMAL & INFECTED HOSTS

<table>
<thead>
<tr>
<th>Case</th>
<th>Color</th>
<th>Opening Pressure</th>
<th>WBC</th>
<th>Glucose</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Infant</td>
<td>Clear</td>
<td>&lt; 180 mm</td>
<td>&lt; 10 mm³</td>
<td>&gt; 40 mg/dL</td>
<td>90 mg/dL</td>
</tr>
<tr>
<td>Normal child or Adult</td>
<td>Clear</td>
<td>&lt; 180 mm</td>
<td>0</td>
<td>&gt; 40</td>
<td>&lt; 40</td>
</tr>
<tr>
<td>Bacterial Meningitis</td>
<td>Cloudy</td>
<td>&gt; 200 mm</td>
<td>200-10,000 (&gt; 80% PMN)</td>
<td>&lt; 40</td>
<td>100 – 150</td>
</tr>
<tr>
<td>Viral Meningitis</td>
<td>Clear</td>
<td>&lt; 180 mm</td>
<td>25-1,000 (&lt; 50% PMN)</td>
<td>&gt; 40</td>
<td>50-100</td>
</tr>
<tr>
<td>Cryptococcal Meningitis</td>
<td>Clear</td>
<td>&gt; 200 mm</td>
<td>50-1,000 (&lt; 50% PMN)</td>
<td>&lt; 40</td>
<td>50-300</td>
</tr>
</tbody>
</table>

- Also, ask for Gram’s Stain, Culture and Sensitivity and consider: + India Ink, cryptococcal antigen, HSV-PCR, AFB and antigen detection studies (counterimmunoelectrophoresis), latex agglutination and coagglutination are methods for detecting specific antigens


- [Rev Neurol Dis. 2006;3(2):57-60] → does not recommend lactate → nonspecific

- CSF glucose is dependent on serum glucose. Rough guideline = 
  - CSF glucose is normally > serum glucose/2
  - CSF/blood glucose ratio less or equal to 0.4 → suggests bacterial meningitis

- When peripheral cell counts are normal, CSF from traumatic LP should contain 1 WBC per 700 RBCs [10, 5th ed., pg. 1533]

FEBRILE SEIZURES
[Dr. Collucci, 12, 6th ed, pg. 803 and PEDIATRICS Volume 121, Number 6, June 2008]

- History of febrile seizure in first degree relative = most consistently identified risk factor for febrile seizure
- 3% of all children
- 6 months to 5 years
- Simple Febrile Seizures = < 15 minutes, generalized tonic-clonic, no focal neuro deficits, mild post-ictal period, occurs once in 24 hours
- Complex Febrile Seizures = prolonged (>15 minutes), are focal, or occur > once in 24 hours
- 30% recurrence (especially if first seizure occurs in child < 1 year old)
- Risk factors for recurrence:
  - Young age of onset < 18 months old (strongest and most consistent risk factor for recurrence)
  - History of febrile seizure in first degree relative
  - Low grade temperature in ED
  - Brief duration between fever and seizure
• Simple vs. complex is NOT predictive of the risk of recurrence

• Viral infections are common causes: influenza, adenovirus and parainfluenza. Human herpes 6 (HHV-6) infection is a particular risk for febrile seizures

• Risk of epilepsy = 1%, same as general population

• If onset < 12 months, multiple seizures or family hx of epilepsy – risk of epilepsy = 2.4%

• Neither a decline in IQ, academic performance or neurocognitive inattention nor behavioral abnormalities have been shown to be a consequence of recurrent simple febrile seizures

• Long-term therapy antiepileptic therapy is not recommended for simple febrile seizures

• In situations in which parental anxiety associated with febrile seizures is severe, intermittent oral diazepam at the onset of febrile illness may be effective in preventing recurrence

• Antipyretics may improve comfort of the child, they will not prevent febrile seizures

• The AAP stance on Febrile seizures from 1996
  ◦ “Strongly recommend” lumbar puncture (LP) in patients < 12 months
  ◦ “Consider” LP 12 to 18 months
  ◦ LP is not routinely necessary in patients > 18 months.

STREPTOCOCCUS IDENTIFICATION

Alpha-hemolytic
  • Streptococci from a GREEN zone around their colonies as a result of incomplete lysis of red blood cells (RBC’s) in the agar
  • Streptococcus pneumoniae
  • Strep viridans (eg, Strep mitis and Strep mutants)

Beta-hemolytic
  • Streptococci form a CLEAR zone around their colonies as a result of complete lysis of RBC’s. Beta-hemolysis is due to the production of enzymes called hemolysins
  • Streptococcus pyogenes (Group A)
  • Streptococcus agalactiae (Group B)

Group (Lancefield Groups A-U)
  • Streptococci are determined by antigenetic differences in C carbohydrate in the cell wall
  • Group A Streptococcus Pyogenes
  • Group B Streptococcus agalactiae
  • Group D Enterococci (eg, Streptococcus faecalis)
    → causes urinary, biliary/abdominal and cardiovascular infections
  • Group D Non-enterococci (eg, Streptococcus bovis, alpha hemolytic) → Gastric CA association
  • Group D hemolytic reaction is variable. Some are beta, alpha or non-hemolytic
  • Non-group = Streptococcus pneumoniae and Strep viridans
  • Groups C, E, F, G, H and K-U streptococci infrequently cause human disease
**M protein**
- Associated with virulence and determines the type of Group A β-hemolytic Strep. It interferes with ingestion by phagocytes

**Anaerobic /microaerophilic Strep**
- Peptostreptococci, variable hemolysis, cause mixed GI infections

### STREPTOCOCCUS PNEUMONIAE “PNEUMOCOCCUS”

**Mnemonics - (COMMONPLACES)**

<table>
<thead>
<tr>
<th>C</th>
<th>Conjunctivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>Otitis</td>
</tr>
<tr>
<td>M</td>
<td>Media</td>
</tr>
<tr>
<td>M</td>
<td>Meningitis</td>
</tr>
<tr>
<td>O</td>
<td>Optochin sensitive</td>
</tr>
<tr>
<td>N</td>
<td>Nasal Sinusitis</td>
</tr>
</tbody>
</table>

- **Strep viridans eg, Strep mitis and Strep mutants** - both alpha hemolytic, are NOT inhibited by Optochin

### STREPTOCOCCUS PYOGENES

**Mnemonic - (PIECES)**

*Group A, beta-hemolytic Strep, Gram + spherical cocci in pairs or chains*

<table>
<thead>
<tr>
<th>P</th>
<th>Pharyngitis (get circumoral pallor)</th>
</tr>
</thead>
<tbody>
<tr>
<td>L</td>
<td>Penicillin (Drug of choice)</td>
</tr>
<tr>
<td>A</td>
<td>Lobar pneumonia</td>
</tr>
<tr>
<td>C</td>
<td>Alpha hemolytic (Non-Grouped)</td>
</tr>
<tr>
<td>E</td>
<td>Capsule</td>
</tr>
<tr>
<td>E</td>
<td>Elderly are candidates for vaccination</td>
</tr>
<tr>
<td>S</td>
<td>Sputum-rusty</td>
</tr>
</tbody>
</table>

- If PCN allergy you can use Zithromax
- Zithromax Dose = 12mg/kg/day x 5 days – not the traditional 10mg/kg day #1 than 5mg/kg days #2 to 5

<table>
<thead>
<tr>
<th>I</th>
<th>Impetigo</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Erysipelas</td>
</tr>
<tr>
<td>C</td>
<td>Cellulitis</td>
</tr>
<tr>
<td>E</td>
<td>Erythrogenic Toxin → Scarlet Fever</td>
</tr>
<tr>
<td>S</td>
<td>Sequelae: 1) Rheumatic Fever (most common after pharyngitis) 2) Poststreptococcal GN (most common after skin infection-impetigo)</td>
</tr>
</tbody>
</table>
• Rheumatic Fever (RF) is a NON-infectious autoimmune disease typically occurring 2-4 weeks after Group A Strep pharyngitis. The immunologic reaction results from cross-reactions between streptococcal antigens and antigens of joint and heart tissue.

• RF is prevented if strep infection treated within 8 days after onset.

• Early antibiotic treatment = earlier resolution of symptoms & shortens course of illness by 1 day \[10, 5th ed., pg. 969-972]\)

• Not common < 2 y/o

• Occurs winter, spring

• Responsible for < 15% of pharyngitis in patients > 15 years old \[10, 5th ed., pg. 969-972]\)

• Rapid Strep sensitivity = 60 – 95% \[10, 5th ed., pg. 969-972]\)

• Acute glomerulonephritis (AGN) = HTN, edema face/ankles and “smoky” urine. May be prevented if strep treated early, however cannot be prevented with PCN after onset of symptoms.

• Reinfection with strep rarely leads to recurrence of AGN.

• “Doughnut lesions” = erythematous papules with a pale center located on both the soft and hard palate, pathognomonic for Group A Strep pharyngitis.

5 MAJOR MODIFIED JONES CRITERIA FOR RHEUMATIC FEVER
Mnemonic - (EM Physicians Can Snuggle Continuously) \[7, with modifications]\)

<table>
<thead>
<tr>
<th>EM</th>
<th>Erythema Marginatum (pink rings on the trunk and inner surfaces of the arms and legs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physicians</td>
<td>Polyarthritis (symmetric, migratory-see mnemonic on migratory arthritis)</td>
</tr>
<tr>
<td>CA</td>
<td>Carditis (steroids may be helpful)</td>
</tr>
<tr>
<td>Snuggle</td>
<td>Subcutaneous Nodules</td>
</tr>
<tr>
<td>Continuously</td>
<td>Chorea (Sydenham’s chorea = “Saint Vitus’ Dance”)</td>
</tr>
</tbody>
</table>

Clinical diagnosis of Rheumatic Fever is made if 2 Major or 1 Major and 2 Minor criteria are present in patient with preceding Strep infection evidenced by:

1) ↑ ASO titer
2) Positive throat culture
3) Recent Scarlet Fever

Minor Criteria
• Fever
• Arthralgia
• ↑ PR interval
• ↑ ESR or CRP
• Previous rheumatic fever
CAUSES OF MIGRATORY ARTHRITIS
Mnemonic - (RF HSP LSD)

A tough one to remember, Try “if you had rheumatic fever, or HSP, you would want LSD.”

<table>
<thead>
<tr>
<th>RF</th>
<th>Rheumatic Fever</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>HSP</td>
</tr>
<tr>
<td>S</td>
<td>Sepsis (Strep, Staph, GC, Meningococcal)</td>
</tr>
<tr>
<td>P</td>
<td>Pulmonary Infection (Mycoplasma, Histo, Coccidia)</td>
</tr>
<tr>
<td>L</td>
<td>Lyme disease</td>
</tr>
<tr>
<td>S</td>
<td>SBEndocarditis</td>
</tr>
<tr>
<td>D</td>
<td>Drugs (Ceclor)</td>
</tr>
</tbody>
</table>

SINUSES PRESENT AT BIRTH
Mnemonic - (ME)

<table>
<thead>
<tr>
<th>M</th>
<th>Maxillary</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Ethmoid</td>
</tr>
</tbody>
</table>

Fyi: adult patients →
Leaning forward exacerbates maxillary sinusitis
Supine position exacerbates ethmoid sinusitis.
CT scan is the gold standard for diagnosis of sinusitis.

CONGENITAL TOXOPLASMOSIS (TOXOPLASMA GONDII )
Mnemonic - (THC3) [1]

<table>
<thead>
<tr>
<th>T</th>
<th>Toxoplasmosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hydrocephalus</td>
</tr>
<tr>
<td>C</td>
<td>Chorioretinitis</td>
</tr>
<tr>
<td></td>
<td>Cerebral Calcification</td>
</tr>
<tr>
<td></td>
<td>Cat feces and raw meat sources</td>
</tr>
</tbody>
</table>

Note: The acquired form in adults may present as a mononucleosis-like syndrome.

DIAGNOSTIC CRITERIA FOR KAWASAKI SYNDROME
- Fever for at least 5 days duration
- Illness that is not explained by other known disease process
AND 4 of the following 5 For Diagnosing Kawasaki Syndrome
1. Bilateral conjunctivitis
2. Changes of lips and oral mucosa (strawberry tongue, red/fissured lips, oropharyngeal edema)
3. Changes of the extremities (erythema of the palms and soles, edema of the hands and feet, periungual desquamation)
4. Polymorphous rash
5. Cervical lymphadenopathy

Complication = coronary artery aneurysms; high suspicion tx = Gamma-globulin, high dose ASA

REYE SYNDROME
- ↓ Glucose
- ↑ LFT’s, except normal BILI, ↑ ammonia
- Mental Status changes with ↑ ICP and seizures

DIPHTHERIA
Corynebacterium diphtheriae, club-shaped G+ aerobic rod, no capsule
Spread person-person nasopharyngeal secretions (place patient in respiratory droplet isolation)
Gray-green pseudomembrane, do not remove → ↑ bleeding; “bull neck” appearance
Exotoxin → disrupts protein synthesis → leads to cardiac and CNS disease
Myocarditis → cardiomyopathy, CHF, Dysrhythmias
CNS & PNS myelin sheath deterioration → peripheral neuropathy, muscle weakness (palate 1st)
Diagnosis: by culture of pharynx and nose (alert lab as special media is needed)

Diphtheria Treatment
Active immunization +
Antitoxin (dose depends on site of infection and duration of symptoms) +
Erythromycin or Penicillin x 7-14 days  [Sanford, 2015]

CHILD ABUSE
1. Neglect  52%
2. Physical  24%
3. Sexual  12%
4. Emotional  6%

- Greatest risk factor = lower socioeconomic status
- Head trauma is the most common cause of mortality
- Shaken Baby / Shaken-Impact Syndrome = SDH, retinal hemorrhages, rib fractures, metaphyseal fractures of long bones
HENOCH-SCHÖNLEIN PURPURA PEARLS
Mnemonic - (PANDAS) [ANK, mnemonic provided by Dr. Collucci] [12, 6th ed, pg. 886]

| P | Purpura, palpable – blanching, buttocks and lower legs |
| A | Abdominal pain 85%, nausea & vomiting (may have → melena/hematochezia; 8% massive GI bleed or intussusception) |
| N | Nephritis 25 – 50% |
| D | Diarrhea |
| A | Arthritis, polymigratory 60 – 80% |
| S | Scrotal edema (2 – 35%) can mimic torsion |

- IgA dominant immune complexes → systemic vasculitis - mainly arterioles and capillaries
- 75% cases 2 to 4 years
- Typically follows URI, in spring
- Intussusception = ileal-ileal secondary to small bowel vasculitis
- Corticosteroids → improvement in GI, Renal and CNS complications

MECKEL'S DIVERTICULUM – RULE OF 2'S

- TWO % of population is affected (of those patients 96% never have problems)
- TWO years of age = most will have presented
- TWO feet from terminal ileum = diverticulum location
- TWO x more common in male > female

- Most common cause of massive rectal bleeding = Meckel's (painless, brick or bright red bleeding)
- Most common cause of minor rectal bleeding (peds) = Anal fissures (painful)
- Child may be well appearing, or may have bilious vomiting & abdominal distention with rectal bleeding
- The bleeding occurs from ulcers in the diverticulum secondary to ectopic gastric mucosa → which is what takes up the Technetium (85% sensitivity and 95% specificity)
- May cause intussusception
- Definitive treatment = surgery

RECTAL PROLAPSE (PROCIDENTIA)

Disease of the extremes of age; PEDS think Cystic Fibrosis; boys > girls;
Elderly = history of excessive staining; women > men

GASTROCHISIS AND OMPHAEOCLE

[10, 5th ed, pgs. 103]

- Gastrochisis = defect in abdominal wall → evisceration of abdominal structures without a sac
- Omphalocele = defect in umbilical ring → intestines protrude in a sac
- Gastrochisis 2x more common than Omphalocele

ED Management = NGT and place plastic covering to prevent heat and water loss.
ANAL PRURITUS

- Most common cause of anal pruritus in kids = Pinworms (Enterobius vermicularis) [12, pg. 487]
- Most prevalent parasite in the US = Enterobius vermicularis (pinworm); 20 to 30% kids infected [10, 9th, pg. 1318]
- Most common cause of perianal cellulitis in young children = group A Streptococcus

HIRSCHSPRUNG’S DISEASE
[10, 5th ed, pgs. 2305-2306]

- Most common cause of obstruction in newborn (functional obstruction)
- 75% recto-sigmoid involvement
- Diagnosis made in nursery as there is no passage of meconium for 24-48 hours
- Bilious vomiting, tarry diarrhea, distended abdomen, poor feeding
- 5x more common in boys
- Associated with Down Syndrome
- Diagnosis
  - Suspected on barium enema - normal segment of colon with proximal dilation
  - Confirmed by biopsy – congenital absence of parasympathetic ganglion cells
- Complication: Toxic Megacolon = progressive enlargement of proximal segment
- Complication: Enterocolitis (abdominal distension, fever, ↑WBC and bloody stool)
- Treatment is surgical; NGT, rectal tube

INTUSSUSCEPTION

- Most common cause of obstruction in children < 3 y/o; male > female
- Siblings of affected patients have a relative risk 20x > general population
- Most common site ileo-cecum – 80%
- Causes: Meckel’s Diverticulum, inverted appendix, tumors, polyps, duplication, lymphoma, HSP (if HSP site = ileal-ileal)
- 50% preceded by viral illness
- Colicky abdominal pain (acute pain, patient draws up legs)
- **Bilious** vomiting
- Check for “currant jelly” stools (late finding) only present in about 20% of cases
- Dance’s Sign = RUQ sausage shaped mass (intussusception) & empty space in RLQ (cecum → RUQ)
- Diagnosis & Treatment = Air contrast or Barium enema; notify Peds surgery before enema (Air Contrast Enema advantages: better control of colonic pressure used for the reduction, safer, faster, less expensive and more effective than barium enema
- Complications = 5 to 10% re-intussusception within 48 hours; severe sepsis or septic shock; bowel perforation

MALROTATION

- Acute bilious emesis, poor feeding, irritability and bloody stool in neonate
- Volvulus is a complication of malrotation
- Duodenal obstruction without volvulus
- Diagnosis: Upper GI with small bowel follow-through (“cork-screw sign”)
- Surgery is the only definitive treatment
HYPERTROPHIC PYLORIC STENOSIS

- 95% of cases between 3-12 weeks; rare before 1 week and after 3 months
- Male > female
- More frequently in first born males
- Familial incidence in 50%
- NON-bilious projectile vomiting just after feeding
- ↓Cl-, ↓K+; hypo-chloremic, hypo-kalemic metabolic alkalosis
- “Olive” in RUQ/pyloric tumor is pathognomonic – 70 to 80% of patients
- Diagnosis: Ultrasound is the gold standard; if US N/A then UGI
- Treatment: fluid, correct electrolytes and surgery

PEDIATRIC PEARLS

- Most common cause of croup = Parainfluenza virus; usually < 3 y/o
- “Previous” most common cause of epiglottitis = H. influenzae now Strep Pneumo & GABHS; 3 to 6 y/o
- Most common cause of bronchiolitis = RSV
- Most common serious bacterial illness (SBI), causes 5% of fever without a source = UTI
- Most common cause of infantile diarrhea = Rotavirus (ROTA = Right Out The Anus)[17]
- Neonatal conjunctivitis (ophthalmia neonatorum): First month of life
  Day 1-2 = chemical, Day 3-5 Gonorrhea, Day 6 to first month of life = Chlamydia trachomatis
- Most common viral cause of conjunctivitis = Adenovirus
- Most common viral cause of otitis media = RSV
- Most common bacterial cause of otitis media = Strep pneumoniae; other two = Haemophilus and Moraxella. Treat all with High Dose Amoxicillin 90 mg/kg/day div q 12 or q 8 hours
- OM best confirmed = decreased mobility of TM and loss of normal landmarks
- Most common complication of otitis media = hearing (conductive) loss [10, 6th ed., pg. 930]
  Another common complication of otitis media = perforation, which usually heals within 7 days
- Most common intracranial complication of otitis media = meningitis
- Most common bacterial cause of pharyngitis = Strep pyogenes (Group A, beta hemolytic strep)
- Monospot is positive in 30% of children 0 to 20 months with mononucleosis; Monospot may be negative the first week of illness [10, 5th ed., pg. 971]
- Most common cause of dysuria in school age girls = non-specific vulvovaginitis
- Most common manifestation of GC infection in children = vaginitis. Obtain specimen from vaginal introitus [10, 5th ed., pg. 1399]
• Roseola infantum (exanthem subitum) → human herpes 6-infection → febrile 3-5 days → ↓ fever → ↑ macular rash. Most common exanthem in children < 2 year of age

• Rubella (German measles) → maculopapular rash spreads in a centrifugal pattern (head to feet); lymphadenopathy typical → postauricular and occipital; “blueberry muffin” skin rash
Rubella and EBV are the most common viral cause of arthritis

• Most common cause of cataracts in newborn = Rubella (German measles)

• Rubeola (Measles) → maculopapular rash spreads in a centrifugal pattern (head to feet); 3-4 day prodrome = C x 3 = Cough, Coryza, Conjunctivitis and Koplik’s spots (white lesions on buccal mucosa = pathognomonic)

• Erythema Infectiosum → fifth disease → parovirus B 19 → “slapped cheek” disease

• Most common cause of acute ataxia is = post-infection (especially varicella); r/o tox, cerebellar tumors

• Mumps = 5-15 y/o; infective 3 days before → 7 days after salivary gland swelling; 70-80% bilateral; Spread = respiratory droplets.
Complications: meningitis, encephalitis, orchitis 15-25% postpubertal men (sterility uncommon; 70% unilateral), uncommon = GBS, transverse myelitis, oophoritis, mastitis, myocarditis, pancreatitis

• Most common growth plate fracture = Salter-Harris type II (physial and metaphysis) 75% [12, pg. 674]

• Most common Salter-Harris fracture likely to result in bone-growth arrest = type V [27, Vol. 14, No. 9, pg. 111]

• Most common cause of hip pain in children < 10 y/o = transient tenosynovitis; ↑ in boys; follows viral illness

• Most common elbow fracture in childhood = supracondylar fracture of the distal humeral metaphysis – the distal fragment is most commonly displaced posteriorly
Complication = Volkman’s ischemic contracture (compartment syndrome) [12, pg. 677, 1230]

• Legg-Calve-Perthes disease = avascular necrosis of pediatric femoral head; age 2-10y/o; 5x ↑ in boys; b/l 15%

• Hip pain → knee in adolescent obese patient → SCFE (slipped capital femoral epiphysis); 2.5x ↑ in boys; ORIF

• Nursemaid’s elbow common 1 to 5 years old – stretch annular ligament and subluxation of radial head;
Reduce: downward pressure on child’s radial head by the docs thumb, supinate and flex elbow

• Greenstick fracture = incomplete, angulated fractures of long bones; may need to complete fracture to achieve anatomic reduction [10, 5th ed., pg. 169]

• Most common fractured bone in children = clavicle

• Fractures suspicious of abuse: spiral fractures of long bones, metaphyseal chip fractures

• Maximum dose of lidocaine of infiltration without epi = 5 mg/kg and with epi = 7 mg/kg
• Codeine dose = 0.5 to 1.0 mg/kg (Acetaminophen with codeine = 120 mg/12mg per 5cc)

• Most common Chronic Disease of childhood = Asthma \(^{[10, 5th \text{ ed.}, \text{pg. } 939]}\)

• Most common cause of home accidental death < 6 y/o = FB aspiration (usually in right mainstem bronchus)

• Age Group most likely to aspirate a FB = 1 to 3 years with peak incidence at age 2 \(^{[10, 5th \text{ ed.}, \text{pgs. } 757-758]}\)

• Most common foods aspirated = nuts \(^{[10, 5th \text{ ed.}, \text{pgs. } 757-758]}\)

• Common cause of pediatric fatal aspiration = food (especially hot dogs) and toy balloons

• Most common Esophageal impaction → kids = coins; adults = food (meat/bones)

• If button battery has passed esophagus and patient is asymptomatic → no retrieval. If cell has not passed pylorus in 48 hours = endoscopic retrieval

• Most common cause of metabolic acidosis in kids = prolonged diarrhea

• Congenital adrenal hyperplasia = adrenal insufficiency → ↓Aldosterone →↓Na+ more common > K+ and ↓ cortisol; Refractory ↓BP: Girls = ambiguous genitalia (look both male and female) → treatment = Hydrocortisone 2 mg/kg IV/IO bolus (max 100mg) \(^{[PALS \text{ provider manual}]}\)

• Most common cause of death 1 month to 1 year = SIDS (RARE before 1 month, risk factors for SIDS = winter, male > female if infectious etiology otherwise, male=female, if mom < 20 y/o, smokes, drugs, no pre-natal care and lower socioeconomic group) \(^{[12, \text{pg. } 599]}\)

• Pott’s puffy tumor = complications of frontal sinusitis → subperiosteal abscess with soft tissue swelling causes pitting edema over the frontal bone; Although it can affect all ages, it is mostly found among teenagers and adolescents

• Most common pediatric dysrhythmia = PSVT heart rate usually > 220, in adults less

  Treatment = Adenosine (Adenocard) 0.1 mg/kg (max 6mg) → 0.2 mg/kg (max 12 mg), may repeat x 1

  SVT Cardioversion = 0.5 to 1.0 J/kg → 2 J /kg \(^{[2010 \text{ AHA Guidelines for Cardiopulmonary Resuscitation and Em Cardiovascular Care, S712}]}\)

• Verapamil is contraindicated in infants

• Most common cause of cardiac arrest = respiratory arrest

• Most common rhythm disturbance in pediatric arrest = bradycardia

• Bradycardia is most commonly an indicator of hypoxemia in the newborn

• First line drug for bradycardic arrest = Epinephrine 0.01 mg/kg of 1:100,000 IV/IO; ETT = 0.1 mg/kg 1:1,000

  Repeat Epi every 3 to 5 minutes

  If ↑ vagal tone or 10 AV Block → Atropine 0.02 mg/kg (minimum dose 0.1mg; max total dose for child = 1 mg) → consider pacing \(^{[PALS \text{ 2006 provider manual, pg 123}]}\)
• Vfib/ Pulseless VT = 2 J/kg Biphasic → CPR 5 cycles → 4 J/kg biphasic [2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care; S708]

  Epi 0.01 mg/kg; Amio 5mg/kg

• Most common category of shock in the pediatric population = hypovolemic; fluid = 20cc/kg 0.9 NS or LR

• Peds Post-arrest - the drug of choice in treating hypotension → epinephrine infusion [10, 5th ed, pg. 98]

• ETT size in PEDS can be estimated = 16 + (age in years) / 4 or age/4 + 4
  ETT x 2 = NG/Foley catheter size
  ETT x 3 = Depth of ETT insertion
  ETT x 4 = Chest Tube size

• < 2y/o ETT size = size of patients small finger (fifth digit!)

• Position of the ET tube at the lips (in cm’s) should = 3 x size of ETT

• Uncuffed ETT in children under 6-8 years of age

• Pretreatment with atropine in RSI for children < 10 years old has fallen out of favor (lack of evidence to support)

• Treatment of hypoglycemia
  Neonate = D10 5 to 10 mL/kg
  Child = D25 2 to 4 mL/kg

• Upper limit of SBP = (Age x 2) + 80

• Weight in kilograms = (Age x 2) + 8

  Newborn = 3 kg
  1 y/o = 10 kg
  5 y/o = 20 kg
  10 y/o = 30 kg

APGAR SCORE
1 and 5 Minutes after birth. Score 0-2

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Appearance (color)</td>
<td>blue/pale</td>
<td>acrocyanosis</td>
</tr>
<tr>
<td>P</td>
<td>Pulse</td>
<td>Absent</td>
<td>&lt;100 beats</td>
</tr>
<tr>
<td>G</td>
<td>Grimace (reflex)</td>
<td>no response</td>
<td>grimace</td>
</tr>
<tr>
<td>A</td>
<td>Activity (muscle tone)</td>
<td>limp</td>
<td>some flexion</td>
</tr>
<tr>
<td>R</td>
<td>Respiration</td>
<td>absent</td>
<td>weak cry/hypovent.</td>
</tr>
</tbody>
</table>

Want score at least 7
SMALL BOWEL OBSTRUCTION - HISTORY
Mnemonic - (VODKA) \(^{(6)}\)

<table>
<thead>
<tr>
<th>V</th>
<th>Vomiting</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>*Obstipation - Old Scar</td>
</tr>
<tr>
<td>D</td>
<td>Distension</td>
</tr>
<tr>
<td>K</td>
<td>(K) Crampy</td>
</tr>
<tr>
<td>A</td>
<td>Abdominal Pain</td>
</tr>
</tbody>
</table>

*Obstipation = 1) the act or condition of obstructing 2) extreme constipation due to obstruction

CAUSES OF SMALL BOWEL OBSTRUCTION
Mnemonic - (BEN VIP) \(^{[3, \text{with modification}]}\)

<table>
<thead>
<tr>
<th>B</th>
<th>Bands (adhesions) = most common cause of SBO; from previous Sx or Crohn’s</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>External Hernias = 2(^{nd}) most common cause of SBO</td>
</tr>
<tr>
<td>N</td>
<td>Neoplasm (note: neoplasm is the most common cause of LBO)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>V</th>
<th>Volvulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Intussusception</td>
</tr>
<tr>
<td>P</td>
<td>Packets (swallowed FBs, bezoars, etc)</td>
</tr>
</tbody>
</table>

SBO PEARLS
\(^{(10, 5th ed. pg. 1283, 1284 & 1287)}\)

SBO → ↑ intraluminal pressure → capillary and lymphatic obstruction → ↑bowel wall edema
Aggressive treatment has ↓Mortality Rate from 60% in 1900 to less than 5% today
35 to 50% of patients with complete SBO → resolution without surgical intervention

CAUSES OF LARGE BOWEL OBSTRUCTION
Mnemonic - (CA\(t\)ch VD)

| CA\(t\)ch | CAncer = most common cause (> 65%)
Most common cancer = adenocarcinoma |
|---|---|
| V | Volvulus = 3\(^{rd}\) most common cause (5%)
Most common site = sigmoid (70%) and cecum (30%) |
| D | Diverticulitis = 2\(^{nd}\) most common cause (20%), secondary to scarring |
• Other causes of LBO:
  Inflammatory disorders, benign tumors, foreign bodies, radiation and fecal impaction
• Ogilvie syndrome = enormous dilation of the RIGHT colon without mechanical obstruction (pseudo obstruction)
• History of LBO = diffuse colicky pain, obstipation...vomiting LATE or ABSENT; distension most common and prominent physical finding

CECAL VOLVULUS
[10, 5th ed., pg. 1335]
• Occurs in all ages, but most common in 25 to 35 years of age
• Not associated with chronic constipation (unlike Sigmoid)
• Onset of pain = acute; in sigmoid volvulus = more gradual
• Treatment = surgery, non-op decompression often unsuccessful

Treatment of non-strangulated Sigmoid Volvulus = decompression and detorsion using a rectal tube via the sigmoidoscope = 85-95% success[10, 5th ed., pg. 1334]

CAUSES OF ILEUS

Mnemonic - (Nurses (RN) and Physician Assistants (PA) Fix MI Burn GAP)

| R | Retroperitoneal hematoma |
| N | Nephrolithiasis |
| P | Pylonephritis or Pneumonia |
| A | Abdominal Surgery |
| Fix | Fx (fractures – lumbar, rib) |
| MI | Myocardial Infarction |
| Burn | Burns, especially if > 20% |
| G | Gallstone ileus or Gastroenteritis |
| A | Appendicitis |
| P | Pancreatitis |

ILEUS

| Minimal abdominal pain | Crampy abdominal pain |
| ↓ or absent bowel sounds | ↑ or normal bowel sounds |
| Gas in SI & colon on x-ray | Gas in SI only on x-ray |
| Nausea & vomiting | Nausea & vomiting |
| Obstipation & failure to pass flatus | Obstipation & failure to pass flatus |
| Abdominal distention | Abdominal distention |
APPENDICITIS

Mnemonic - (PAVEL’S P’S) [1, with modifications]

<table>
<thead>
<tr>
<th>P</th>
<th>Pain-periumbilical → RLQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Anorexia</td>
</tr>
<tr>
<td>V</td>
<td>Vomiting</td>
</tr>
<tr>
<td>E</td>
<td>Elevated temperature, mild/normal. If elevated, think perforation</td>
</tr>
<tr>
<td>L</td>
<td>Leukocytosis – may be normal</td>
</tr>
<tr>
<td>S</td>
<td>Signs [10, 5th ed, pg 1294]</td>
</tr>
<tr>
<td></td>
<td>Rovsing’s = palpate LLQ → tender RLQ</td>
</tr>
<tr>
<td></td>
<td>Obturator = patient supine, flex and internally rotate right hip → tender RLQ</td>
</tr>
<tr>
<td></td>
<td>Iliopsoas = patient asked to extend right hip → tender RLQ</td>
</tr>
</tbody>
</table>

Pathophysiology = lumen obstruction (fecaliths – most common, lymphoid hyperplasia, dietary matter, worms, tumors, granulomatous disease, adhesions) → increased edema as mucus continues to be secreted → edema and vascular compromise → followed by bacterial invasion

Most common cause of surgical abdomen in children = appendicitis
> 66% cases are in patients < 30y/o (peak incidence = 11 to 20 y/o)

Condition most commonly mistaken for acute appendicitis in children = mesenteric adenitis and gastroenteritis

Most common surgical emergency in pregnancy = appendicitis
Higher perforation rate in pregnancy [10, 5th ed, pgs 2425-2427]
Fetal loss 20% with perforated appy
Pain may be in RUQ [10, 5th ed, pgs 2425-2427]
Most patients with appendicitis will have pain in RLQ, even in 3rd Trimester [Am J of Obst and Gyne 2000]

Threshold for human teratogenesis = 10 rad; fetus most vulnerable 8 to 15 weeks gestation
CT abdomen = 3.5 rad [12, 6th ed., pg. 675]

1 rad (Radiation Absorbed Dose) = 10 mgray (Gy); Gray = the SI unit
Gray (Gy) = amount of radiation required to deposit 1 joule of energy in 1 kg of any kind of matter

1 rad = 0.01 sievert (Sv) = 10 mSv

Sievert (Sv) = a unit used to derive a quantity called dose equivalent. This relates the absorbed dose in human tissue to the effective biological damage of the radiation. Not all radiation has the same biological effect, even for the same amount of absorbed dose. [www.bt.cdc.gov/radiation/glossary.asp] 1 sievert (Sv) = 100 rem (Roentgen Equivalent in Man)
CAUSES OF PANCREATITIS

Mnemonic - (ABCDEF SHIP LIST) [with modifications]

A  Alcohol
    Alcohol and biliary make up 85% of cases
B  Biliary (Gall Stones)
C  Cancer – pancreatic
D  Drugs (see below)
E  ERCP (3% post procedure)
F  Familial

S  Scorpion stings
H  ↑Hyper-calcemia (rare)
I  Idiopathic = 3rd most common cause (thought to be a form of microlithiasis)
P  Posterior duodenal ulcer erosion
P  Pancreas divisum

L  Lipids ↑TG > 1,000 mg/dL
I  Infection (mumps, coxsackie, HBV, EBV, influenza, legionella, mycoplasma, West Nile Virus)
S  Surgery – post op
T  Trauma

DRUGS THAT CAUSE PANCREATITIS

Mnemonic - (PAST DATE) 2

P  Pentamidine
A  Azathioprine (Imuran) common cause
S  Sulfa
T  Thiazides

D  Depakote (valproic acid)
A  ASA and NSAIDs
T  Tetracyclines
E  Ethacrynic acid

Propofol
Amiodarone
Steroids
Tylenol
[12, pg. 788]
Diphenoxylate (lomotil)
Amlodipine
Tamoxifen
Estrogen

HIV medications → all 4 are nucleoside reverse transcriptase inhibitors (NRTIs), inhibit viral reverse transcriptase, thus preventing / interfering with the production of a DNA copy of viral RNA

Didanosine (ddI) (also 15% neuropathy)
Zalcitabine (ddC) (also 30% neuropathy)
Stavudine (d4T) (also 30% neuropathy)
Lamivudine (Epivir)
PANCREATITIS - RANSON’S CRITERIA

Mnemonic - (All Wild Girls Like Soccer)  

<table>
<thead>
<tr>
<th>On admission</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>Age &gt; 55</td>
</tr>
<tr>
<td>Wild</td>
<td>WBC &gt; 16,000</td>
</tr>
<tr>
<td>Girls</td>
<td>Glucose &gt; 200</td>
</tr>
<tr>
<td>Like</td>
<td>LDH &gt; 350</td>
</tr>
<tr>
<td>Soccer</td>
<td>SGOT (AST) &gt; 250</td>
</tr>
</tbody>
</table>

Within 48 hours
- Hct ↓ > 10%
- BUN ↑ > 5
- Ca2+ < 8
- PO2 < 60
- Base deficit > 4
- Fluid sequestration > 61

“Although they serve as a reminder of features that portend a worse prognosis, the Ranson Criteria have poor predictive value in the acute setting that does not improve on clinical judgment” [12, 6th ed. pg. 575]

- Pathophysiology: Inappropriate activation of trypsin → pancreatic autodigestion and ↑ local inflammatory mediators are released → which cause distant extra-pancreatic dysfunction
- Amylase rises within 6 to 24 hours however returns to normal in 3 to 7 days
- Lipase rises within 4 to 8 hours and stays elevated for 7 to 14 days
- ↑ lipase > 3x upper limit of normal with history c/w pancreatitis to make the diagnosis
- The absolute level of serum amylase and lipase do not correlate with for disease severity and have no prognostic value [12, 6th ed. pg. 575]
- Lipase and amylase both exist in other tissues
- Experts recommend lipase over amylase when seeking diagnosis of pancreatitis [10, 5th ed. pg. 1276] and [12, 6th ed. pg. 575]
- Overall mortality rate (MR) = 5%
- Mortality rate of severe pancreatitis (see complications below) = 14 - 25%

Complications of acute pancreatitis: pleural effusions (left), ARDS, myocardial depression, DIC, renal failure, shock, phlegmons, abscesses, pseudocysts and necrosis

Signs of Hemorrhagic Pancreatitis [12, pg. 513] Not common (3%), but if present MR = 37%

- **Cullen sign** = periumbilical ecchymosis
- **Turner sign** = flank ecchymosis

Treatment: aggressive fluid management, analgesia, oxygen administration and early nutrition
GALLSTONES

Mnemonic - (4 F’s) [ANK]

<table>
<thead>
<tr>
<th>F</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>Fat</td>
</tr>
<tr>
<td>F</td>
<td>&gt; Forty</td>
</tr>
<tr>
<td>F</td>
<td>Fertile</td>
</tr>
</tbody>
</table>

- Gall stones < 20% picked up on KUB, cholesterol GS = 80% and these are radiolucent
- Kidney stones > 90% picked up on KUB

Charcot’s Triad: present in 50 to 70% patients with cholangitis
1) Fever
2) RUQ pain
3) Jaundice

Reynolds’s Pentad: Charcot’s triad plus
4) Mental status changes
5) Hypotension

- Most common organisms E. coli (27%), Klebsiella species (16%), Enterococcus species (15%), Streptococcus species (8%), Enterobacter species (7%) and P. aeruginosa (7%) [http://emedicine.medscape.com/article/74245-overview]
- Murphy’s Sign = inspiratory arrest, due to pain, with palpation of RUQ / over GB
- Tender RUQ → radiates to the right scapula

CHOLECYSTITIS

Labs = leukocytosis with or without a left shift, ↑ or normal aminotransferases; bilirubin usually within normal limits [10, 6th ed., pgs. 1491-1492]

- 5 to 10% acalculous
- Most common surgical emergency in elderly with abd. pain = Acute Cholecystitis [12 6th ed., pg. 503]
- Bilirubin will be elevated in cholangitis and in general have a higher fever and appear more ill then patient with cholecystitis
- Ultrasound is the procedure of choice for investigating the gallbladder [10, 5th ed., pg. 1266]
- Most sensitive and specific test for cholecystitis = nuclear scintigraphy (HIDA scan) [10, 5th ed., pg. 1267]

Cholelithiasis = gallstones in the gallbladder
Biliary colic = pain caused by a stone temporarily obstructing the cystic duct
Cholecystitis = inflammation of the gallbladder from obstruction of the cystic duct
Cholelithiasis = stone in the common bile duct – often significant ↑Alk phos and transaminases
Cholangitis = bacterial infection superimposed on an obstruction of the biliary tree gallstone, neoplasm or stricture

PORCELAIN GALLBLADDER

- Uncommon manifestation of chronic cholecystitis, characterized by intramural calcification of the gallbladder wall
- The diagnosis is suggested by an abdominal radiograph revealing an incidental calcified gallbladder.
- Patients with a porcelain gallbladder are often asymptomatic, but are at increased risk for the development of gallbladder carcinoma (poor prognosis)
HEPATITIS PEARLS

[10, 6th edn, pgs. 1402-3 and 5th ed, pg. 1251] and [12, 6th edn, pgs. 568, 990]

Incubation period
HAV is 15-45 days
HBV is 60-90 days
HCV is 30-90 days

- ETOH Hepatitis → AST (GOT) > 2 x ALT (GPT); ratio AST/ALT > 2
- Viral Hepatitis → ALT generally >>AST; both 10 to 100x normal
- Scleral icterus when serum BILI > 2.5 mg/dl
- ↑ PT = not good, clue to complicated course; PT reflects hepatic synthetic function

HAV = RNA virus; spread = fecal → oral route
Fecal excretion of HAV usually occurs prior to symptoms of acute HAV infection
IgM antibody to HAV → indicates acute infection; no chronic carrier state

HBV = DNA virus, transmitted hematogenously and sexually
HB cAb-IgM = antibody to core antigen (cAg) → indicates acute infection
HB cAb-IgG = antibody to cAg → prior infection
HB sAb = antibody to surface antigen (sAg) → indicates acute or prior infection or immunization
HB eAb = antibody to eAg → resolving infection and decreased infectivity

HB sAg surface antigen indicates acute or chronic infection; measurable before clinical illness
HB eAg antigen associated with active acute or chronic infection, and indicates high infectivity
HB eAg comes from the core gene and is a marker of active viral replication

HCV = RNA virus
Anti-HCV = antibody that defines infection with HCV, acute or past
Risk of HCV = 0.03% per unit of blood transfused

50% HCV develop chronic hepatitis, 20% of this group develop cirrhosis within 10 years
10% of adults and 90% of neonates infected with HBV develop chronic hepatitis
In addition to chronic hepatitis and cirrhosis, HBV associated with hepatocellular CA

HEPATITIS PROPHYLAXIS

Hepatitis A = Immune Serum Globulin (ISG) = 0.02 ml/kg IM within 14 days of exposure Vaccine is available
[10, 5th ed, pg. 1255]

Hepatitis B if previously unvaccinated = HBIG 0.06 ml/kg IM, simultaneously with HBV vaccine series with first shot in the deltoid
[10, 5th ed, pg. 1256]
HEPATOTOXINS
→ hepatocellular necrosis \([12, \text{6th edn, pgs. 568}]\)

- Acetominophen (NSAIDS)
- Amphotericin
- INH (Ketoconazole \([10, \text{5th ed. pg. 1261}]\))
- Phenytoin (Valproic acid)
- Iron (Amiodarone \([10, \text{5th ed. pg. 1261}]\))
- Cocaine (Halothane)
- Mushrooms (Ecstasy (MDMA))
- White Phosphorus (Carbon tetrachloride)
- Inorganic arsenicals, thallium & borates

Reye Syndrome: resembles fulminant liver failure, but microvesicular fatty infiltration occurs without hepatocellular necrosis

Cholestatic picture = chlorpromazine, haldol, anabolic or oral contraceptive steroids and erythromycin estolate \([10, \text{5th ed. pg. 1261}]\)

LIVER ABSCESS

Usually polymicrobial, \textit{Escherichia coli} and \textit{Klebsiella pneumonia} = 2 most frequently isolated pathogens; other pathogens = anaerobes, \textit{Bacteroides}, \textit{Fusobacterium}, microaerophilic/anaerobic \textit{Strep} (\textit{Peptostreptococcus}); aerobic \textit{Strep} = \textit{Strep. faecalis}, and \textit{Pseudomonas}

A colonic source usually initial source of infection (diverticulitis > biliary disease > appendicitis)

If hematogenous spread = \textit{Staphylococcus aureus}

Patients with Crohn’s disease = \textit{Staphylococcus milleri}

Most common test question = \textit{Entamoeba histolytica} → causes 10\% of liver abscess cases

CHILD-PUGH CLASSIFICATION OF SEVERITY OF LIVER DISEASE

Mnemonic - (I BEAN) \(^{[10]}\)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>1 Point</th>
<th>2 Points</th>
<th>3 Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>I INR</td>
<td>&lt;1.7</td>
<td>1.7-2.3</td>
<td>&gt;2.3</td>
</tr>
<tr>
<td>B Bilirubin (g/dl)</td>
<td>&lt; 2.0</td>
<td>2.0 - 3.0</td>
<td>&gt; 3.0</td>
</tr>
<tr>
<td>E Encephalopathy</td>
<td>None</td>
<td>Grade 1-2</td>
<td>Grade 3-4</td>
</tr>
<tr>
<td>A Ascites</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
</tr>
<tr>
<td>N Nutrition = Albumin, (g/dl)</td>
<td>&gt; 3.5</td>
<td>2.8 - 3.5</td>
<td>&lt; 2.8</td>
</tr>
</tbody>
</table>

Score of
5-6 = grade A (well-compensated disease)
7-9 = grade B (significant functional compromise)
10-15 = grade C (decompensated disease)

These grades correlate with one- and two-year patient survival:
Grade A - 100 and 85\%
CAUSES OF FECAL LEUKOCYTES
Mnemonic - (I Can SEE leukocytes In Your Shample) \[18, with modifications\]

<table>
<thead>
<tr>
<th>I</th>
<th>Ischemic colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Can</td>
<td>Campylobacter (Guillain Barre Syndrome; poultry, eggs)</td>
</tr>
<tr>
<td>S</td>
<td>Salmonella (rose spots, fever with relative bradycardia; sickle cell or asplenic patients; pet turtle or inguana; after eating poultry, eggs)</td>
</tr>
<tr>
<td>E</td>
<td>E. coli 0157:H7 (EHEC = enterohemorrhagic E. coli) HUS → microthrombi → RF</td>
</tr>
<tr>
<td>E</td>
<td>E. coli (EIEC = enteroinvasive E. coli)</td>
</tr>
</tbody>
</table>

**LEUKOCYTES**

<table>
<thead>
<tr>
<th>In</th>
<th>Inflammatory bowel disease (IBD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Your</td>
<td>Yersinia enterocolitica (mimics appendicitis; erythema nodosum)</td>
</tr>
<tr>
<td>Shample</td>
<td>Shigella (seizures, Reiter’s syndrome and HUS; mucoid bloody diarrhea + ↑ Fever)</td>
</tr>
</tbody>
</table>

Reiter syndrome = can’t see, can’t pee, can’t climb a tree = urethritis, iritis, arthritis

- Organisms that produce variable findings on microscopic stool examination, depending on the invasive properties of the strain and the degree of colonic involvement
  - \textit{Clostridium difficile}
  - \textit{Aeromonas}
  - \textit{Vibrio parahaemolyticus} (most common cause of gastroenteritis in Japan)

- Viruses cause the majority of infectious diarrheas followed by bacteria \[12, 6th ed., 556\]

- Suggests treating diarrheal illnesses regardless of whether or not diarrhea is invasive, therefore “ascertaining the presence or absence of fecal leukocytes is superfluous” \[12, 6th ed., pg. 555\]

**NON-VIRAL DIARRHEA PEARLS**

- Most common cause of acute food poisoning in US = \textit{Clostridium perfingens} \[10, 5th ed., pgs. 1308\]

- From CDC → \textit{Campylobacter} is one of the most common causes of diarrheal illness in the US

- \textit{Clostridium perfingens} = Patients ingest heat-resistant spores which produce an enterotoxin in the GI tract (non-invasive/non-bloody diarrhea; poultry, meat, eggs)
DIARRHEA PEARLS

- Most common cause of travelers diarrhea = *Enterotoxigenic E. coli* (ETEC) produces a toxin that acts on the intestinal lining (non-invasive / non-bloody diarrhea)

- *Enteropathogenic E. coli* (EPEC) attach to the intestinal mucosa, causing diarrhea in children and adults → exact mechanism unclear. Subtle changes in the microvillus surface have been noted in association with attached EPEC, and this damage may cause diarrhea.

- *Entamoeba histolytica* (causes 10% of liver abscess cases) = noninvasive colitis / fecal leukocyte negative, with bloody diarrhea; 10% of world population infected, however only 10% = clinical disease Infects colon → mimics UC; Treatment = Flagyl [10, 5th ed., pg. 1317]

- Most common causes of diarrhea in AIDS patients = *Cryptosporidium* and *Cytomegalovirus* (CMV) [10, 5th ed., pg. 1319]

- Most common cause of chronic diarrhea in AIDS patients = *Cryptosporidium* or *Isospora belli*

- Most common symptom in AIDS patient = diarrhea

- Most common infection of GI tract in AIDS patient = oral Candida

- *Bacillus cereus* = enteritis after eating fried rice

- Ciguatera fish poisoning = caused by consumption of reef fish that feed on dinoflagellates (algae); most common ciguatoxin carriers: red snapper, grouper, amberjack, sea bass, sturgeon, barracuda; symptoms = N/V/D, paresthesias, *paradoxxal temperature reversal*, teeth feel loose, vertigo; treatment = supportive (antihistamines, amitriptyline, fluids); if brady → atropine and dopamine; consider Mannitol in severe cases.

- Scombroid fish poisoning = caused by consumption of dark meat fish (tuna, mackerel, skipjack, bonito, marlin); nonscombroid species (mahi-mahi sardine, yellowtail, herring, and bluefish); histamine-like reaction: flushing, palpitations, HA, N/V/D, diffuse, macular, blanching erythema, peppery bitter taste; treatment = antihistamines

- Diarrhea in patients with pet turtle or inguana, asplenic or with sickle cell = *Salmonella*

- Diarrhea after potato salad or mayonnaise = *Staph aureus*

- Diarrhea after eating raw oysters = *Vibrio cholera*

- Most common water-borne diarrhea US = *Giardia lamblia*; Parasite however no Eosinophilia

- Most common symptom with *Giardiasis* = acute watery or pale explosive, offensive smelling diarrhea 90%; abdominal colicky pain / distention /flatulence in 75%. Asymptomatic 15%; [10, 5th ed., pg. 1316]

- Most common intestinal parasite in the US = *Giardia lamblia*; treatment = Metronidazole (Flagyl)
### CAUSES OF POST-OP FEVERS

**Mnemonic - (7 W’s)**

<table>
<thead>
<tr>
<th>W</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>W</td>
<td>Wind (atelectasis or pneumonia) – first 24 hours</td>
</tr>
<tr>
<td>W</td>
<td>Water (UTI) – days 3 to 5</td>
</tr>
<tr>
<td>W</td>
<td>Wanes → veins → check IV sites – days 3 to 5</td>
</tr>
<tr>
<td>W</td>
<td>Walk (DVT/PE) &gt; 5 days post-op</td>
</tr>
<tr>
<td>W</td>
<td>Wound &gt; 5 days post-op</td>
</tr>
<tr>
<td>W</td>
<td>Wonder drugs → drug fever</td>
</tr>
<tr>
<td>W</td>
<td>Women → endometritis</td>
</tr>
</tbody>
</table>

### ARTERIAL OCCLUSION

**Mnemonic - (6 P’s)**

<table>
<thead>
<tr>
<th>P</th>
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</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>Pain</td>
</tr>
<tr>
<td>P</td>
<td>Pulselessness</td>
</tr>
<tr>
<td>P</td>
<td>Palor (limb initially white, with time cyanosis may appear indicating desaturation of blood with ongoing ischemia)</td>
</tr>
<tr>
<td>P</td>
<td>Polar (for cold, and rhymes with pallor)</td>
</tr>
<tr>
<td>P</td>
<td>Paresthesias (complete anesthesia = immediate surgical intervention)</td>
</tr>
<tr>
<td>P</td>
<td>Paralysis (last finding)</td>
</tr>
</tbody>
</table>

- Most common cause of arterial occlusion = embolism
- Embolism originates from the heart (thromboembolism) in 80 to 90% of cases
  Afib and recent MI are the two most common causes of mural thrombus within the heart
  Note: atheroembolism = microemboli from blood vessels
- Most common site of arterial embolism occlusion = bifurcation of common femoral artery
- Ankle Brachial Index: normal > 90%, mild 70-90%, moderate 50-70% and severe arterial insufficiency < 50% (inflate cuff above ankle, doppler → DP or PT, compare to brachial BP)

### POST-OP COMPLICATIONS OF THYROIDECTOMY

**Mnemonic - (THYROIDS)**

<table>
<thead>
<tr>
<th>T</th>
<th>Tetany-hypoparathyroidism</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Y</td>
<td>Yell – recurrent laryngeal → stridor &amp; superior laryngeal nerve → voice fatigue</td>
</tr>
<tr>
<td>R</td>
<td>Recurrent hyperthyroidism</td>
</tr>
<tr>
<td>O</td>
<td>Oesophageal damage</td>
</tr>
<tr>
<td>I</td>
<td>Infection</td>
</tr>
<tr>
<td>D</td>
<td>Deaths</td>
</tr>
<tr>
<td>S</td>
<td>Storm-thyroid storm - tachypnea, tachycardia and fever</td>
</tr>
</tbody>
</table>
**SURGERY / GI PEARLS**

- Most common GI disease in the US = gastroenteritis
- Most common cause of vomiting in adults = medications
- Most common cause of upper neuromuscular swallowing dysfunction = CVA
  
  2nd = polymyositis or dermatomyositis
- Most common cause of lower swallowing dysfunction = intrinsic motility disorder (achalasia, spasm)
- Most common cause of significant LGI bleed = diverticulosis; note 10-15% of BRPR is from UGI source
  
  [10, 5th ed., pg. 1332]
- Diagnose Bleeding Site: Angiography requires a brisk bleeding rate (0.5-2 mL/min)
  
  Technetium-Labeled Tagged RBC Nuclear Scan or GI bleeding scan- is more sensitive and can detect bleeding sites of 0.1mL/min
- Most common cause of UGI bleed = PUD; melena present in 33% of LGI bleeds (need 150-200cc of blood in GI tract for minimum of 8 hours for stool to turn black)
  
  Consider Protonix 80mg IV bolus, then drip of 8mg/hr
- Most common location of gastric ulcer = lesser curvature of body and antrum
- Most common location of stress ulcers = body and fundus; examples = Curling’s ulcers (burns);
  
  Cushing’s ulcers (associated with ICP → head trauma and CNS tumors); sepsis and shock
- Duodenal ulcers > more common than gastric ulcers
  
  Duodenal ulcers improve with food, gastric ulcers worsen with food
- Gastric ulcers bleed more often than duodenal
- Most common cause of UGI bleeding in pregnancy = esophagitis
- Most common cause of esophageal varices in USA = ETOH abuse
- Most common cause of esophageal varices worldwide = Schistosomiasis
- Start Octreotide on patients with UGI bleed & known esophageal varices for the first 24 hours of hospitalization
  
  **Octreotide (Sandostatin)** mimics natural somatostatin (50mcg IV bolus followed by 50mcg/hr drip)
  
  → splanchnic vasoconstriction → variceal bleeding (similar to vasopressin without coronary vasoconstriction)

  In addition to Octreotide treat esophageal varices with: PPI [Protonix (Pantoprazole) 80 mg IV bolus, 8
  
  mg/hr drip], IV fluids, blood products (PRBCs, FFP, platelets) and antibiotics
  
  Erythromycin 250 mg IV x1 before EGD → promotility → improves EGD quality
  
  Ceftriaxone 1 gm /day x 5 days → ↓ release of endotoxins → ↓systemic vasodilation → prevents coagulopathy and early rebleeding
  
  TOX Pearl → Octreotide is used in the treatment of refractory hypoglycemia from oral hypoglycemic agents
No evidence that NGT placement aggravates hemorrhage from varices or Mallory-Weiss tears \([10, 5th \text{ ed, pg. } 1319]\).  
Mallory-Weiss tears = partial thickness esophageal tear with bleeding after vomiting  
Boerhaave syndrome = full thickness esophageal rupture after vomiting  
Classic triad presentation of Boerhaave syndrome = vomiting, CP, and subcutaneous emphysema  
SOB may be secondary to pleuritic CP or left-sided pleural effusion (common), or pneumothorax  
Hamman’s crunch = Pneumomediastinum; 20% of patients  
Esophagram helps confirm diagnosis; Gastrografin (water-soluble contrast) = 90% sensitivity  
Most common cause of esophageal perforations = iatrogenic perforations (others: FBs, caustic burns, Boerhaave’s)  
Most common site of iatrogenic perforations = pharyngoesophageal junction or the esophagogastric junction \([10, 6th \text{ edn, pgs } 482-485]\)  
> 90% of spontaneous esophageal perforations occur in the distal esophagus  
Foreign Bodies → Most common in kids = 80%  
Most common site for Esophageal foreign bodies to lodge  
#1 = Level of cricopharyngeus muscle (C6)  
#2 = Aortic arch (T4)  
#3 = GE junction  
Most common esophageal impaction in kids = coins  
Most common esophageal impaction in adults = food (meat/bones); tx = glucagon IV, NTG, procardia ....  
EGD  
Glucagon 1 to 2mgIV  
Relaxes esophageal smooth muscle and the LES  
Little effect on the motility of the proximal esophagus  
Less effective in patients with structural abnormalities, such as strictures or rings  
Success rate 15 to 50%  
NTG, Procardia (may cause hypotension)  
Get EGD!  
Papain (meat tenderizer) – NO! → meat tenderizer may cause necrosis of the esophagus  
If perforation suspected → water-soluble contrast (Gastrografin)  
CXR esophageal foreign body = frontal plane; tracheal = sagittal plane  
Most common age group to aspirate FB = 1 to 3 years  
Button battery ingestion: if in duodenum → observe; if in esophagus → immediate removal; if not removed in 8 hours there is a risk of esophageal perforation secondary to rapid erosion  
Most common malignancy of esophagus = adenocarcinoma, (no longer squamous cell); adenocarcinoma has continued to rise since 1970 and is now > 50% of all esophageal carcinomas
• Barrett’s esophagus is a risk factor for adenocarcinoma; up to 10% of patients with Barrett’s esophagus will develop adenocarcinoma

• Most common benign stomach neoplasms = Polyps (90% hyperplastic/inflammatory polyps, 10% adenomatous – single cauliflower-like malignant transformation risk)

• Most common malignancy of stomach = Adenocarcinoma; Check → CEA, Virchow supraclavicular-node; blacks > whites; lymphoma second most common stomach malignancy

• Krukenberg tumor → Primary stomach or breast CA metastasis to ovary (usually both ovaries; accounts for 5% of ovarian cancers)

• Klatskin tumor = cholangiocarcinoma – cancer of biliary tree

• Most common chronic infection in liver or kidney transplant = CMV

• Most common malignancy of small intestine in the US = Adenocarcinoma

• Most common malignancy of large intestine = Adenocarcinoma

• Most common site of large bowel perforation = cecum

• Most common cause of mesenteric ischemia = arterial embolus; get angiogram not CT
  Other causes = mesenteric arterial thrombosis (15%) or venous thrombosis (15%), non-occlusive mesenteric ischemia (20%) and hypercoagulable states \[10, 5th ed., pg. 1288\]. Pain out of proportion to exam; heme+ stool; ↑lactate

• Toxic megacolon occurs in 5% of case of Ulcerative Colitis; cause unclear; transverse colon more common

• Ulcerative Colitis lesions = erythema nodosum, pyoderma gangrenosum and aphtous stomatitis

• Crohn’s = peri-anal disease (fistulas and fissures), bowel malignancies 3x more common

• Most common anorectal abscess = perianal (may be first presentation of Crohn’s)

• Most common cause of SBP = E. coli 50%, Enterococcus, Strep. Pneumo (Tx = Amp + Gent)

• APT-Downey Test = differentiates if a bloody stool contains maternal or fetal blood → add 1% NaOH to bloody stool → fetal Hgb resists oxidation → remains pinkish-red, whereas maternal Hgb changes to dark brown color \[10, 5th ed, pgs. 2307\]

TRAUMA PEARLS

• Monocular diplopia = lens dislocation

• Leading cause of traumatic death in adults and PEDS = severe Traumatic Brain Injury; #2 = thoracic trauma

• Severe Traumatic Brain Injury (TBI) = GCS < 8
- Most common cause of severe TBI = Falls 28% > MVC 20% [CDC, National Center for Injury Prevention and Control; 2006]

- SBP < 90 mmHg, O2 saturation < 90%, PaCO2 < 35 mmHg and T > 100.4 °F correlate with poor outcomes and are secondary insults to avoid in TBI [26, Vol. 30, No. 21, pg. 258]

- Routine hyperventilation in TBI should be avoided, unless evidence of herniation [J. Neurotrauma 2007;24 Suppl 1:S1-106]

- A single episode of hypotension in patients with traumatic brain injury = double mortality

- Seizure > 20 min after trauma – worse; ↑ possibility of internal injury and development of seizures later

- Seizure Prophylaxis [10, 5th ed., pg 296]
  - Depressed skull fracture
  - Penetrating brain injury
  - Intracranial Hemorrhage
  - Acute SDH or Epidural
  - Prior history of seizures
  - Seizure at time of injury or ED presentation
  - Intubated and paralyzed head injury patient

- Most common traumatic herniation syndrome = Uncus of temporal lobe → transtentorial herniation; CN III compressed → ipsilateral, dilated, non-reactive pupil; contralateral hemiparesis; herniation progresses → decerebrate posturing

- Most common bleed = traumatic SAH

- Most common cause of post-traumatic coma = diffuse axonal injury

- Subdural=crescent shape on CT; bridging veins much more common than epidural and ↑mortality vs epidural

- Epidural = lens / biconvex/ football shaped on CT; middle meningeal artery; “lucid interval”

- Most common CT abnormality after severe closed head injury = traumatic SAH [10, 5th ed., pg. 310]

- Most common bone fractured in children with skull fractures = parietal bone (60-70%)

- 15 to 30% of linear skull fractures in children have been associated with an intracranial injury

- Growing Skull Fractures = linear skull fractures in children that enlarge over time and produce a cranial defect. Result from tear in dura, present months to years following the initial injury, usually require surgical correction

- Indications for Obtaining Head CT in Children with Head Trauma
  - AMS Focal neurologic deficits
  - HA Evidence of basilar or depressed skull fractures
  - LOC Irritability or behavior changes
  - Amnesia Scalp hematoma in children < 2 years old
  - Seizure Persistent vomiting

- Significant head or facial trauma have 5 to10% associated C-spine injuries
• Nexus Criteria for C-spine imaging (if any criteria present, cannot clear C-spine clinically)
  ▫ Midline spinal tenderness
  ▫ Focal neurological deficit
  ▫ Altered level of consciousness
  ▫ Intoxication
  ▫ Distracting injury present

• Patients with spinal cord injury have 25% associated head injury
• Most common level of C-spine injury in Elderly = C1 to C3 (higher than younger, non-peds patients)
• Most common C-spine fx in Elderly = Type 2 odontoid fx
• Most common cervical fracture in kids = higher cervical (especially odontoid); more common in older kids
• Most common facial fracture = nasal (rule-out septal hematoma); #2 mandible
• Most common Mandible fx = condyle 36% (jaw deviates towards fx on maximal opening) > body 21% angle 20% > symphysis 14% >> ramus 3% \[10, 5\textsuperscript{th} ed., pg 326\]; most common side = left; similar to pelvic ring usually 2 fractures; subungal or buccal ecchymosis is pathognomonic for mandibular fracture

• LeFort Fractures\[10, 5\textsuperscript{th} ed., pg. 324\]
  ▫ I = Horizontal fracture involving only the maxilla at level of nasal fossa Motion of hard palate but not nose; if SQ air \(\rightarrow\) sinus fracture
  ▫ II = Pyramidal fracture = vertical fx through maxilla, nasal bones, medial aspects of the orbits Motion hard palate and nose, not eyes; blood in nares, rhino or otorrhea; swollen mid-face
  ▫ III = Craniofacial disjunction; “dishface”; entire face moves but not head; CSF rhinorrhea

• Orbital Blow-Out Fracture = Vertical diplopia - entrapment of inferior rectus muscle \(\rightarrow\) results in limited upgaze and may cause pain on attempted upgaze; endophthalmos Fractures along the floor usually affect the -- infraorbital nerve \(\rightarrow\) hypoesthesia of the cheek and upper
• Weakest area of Orbit = floor (contents prolapse into maxillary sinus)
• Retrobulbar hematoma = Eye pain, diplopia, visual loss, reduction of ocular motility, proptosis, ↑IOP, ecchymosis of eyelids, chemosis, ophtalmoplegia, APD; Treatment = lateral canthotomy
• Most common Zygomatic fracture = arch > tripod \[12, 6\textsuperscript{th} ed., pg. 1588\]; both uncommon
• Tripod fracture
  Zygomatico - Maxillary articulation - infraorbital rim fx
  Zygomatico - Temporal articulation - at the arch
  Zygomatico - Frontal articulation
• Flat cheek, diplopia, anesthesia to cheek/upper lip, cheek or periorbital edema = Tripod fracture

• Zones of the Neck\[10, 5\textsuperscript{th} ed., pg. 371\]
  Zone I - (base of neck) extends superiorly from the sternal notch and clavicles \(\rightarrow\) cricoid cartilage
  Zone II - (midneck) cricoid cartilage \(\rightarrow\) angle of the mandible
  Zone III - (upper neck) angle of the mandible \(\rightarrow\) base of skull
• Most commonly injured intra-abdominal structure in PEDS = spleen; Kehr’s sign (referred pain shoulder)
• Most commonly solid organ damaged after blunt trauma = spleen
• Most commonly solid organ damaged after penetrating trauma = liver
• Tension pneumothorax = ↓ BP, distended neck veins, ↓ breath sounds and tracheal deviation
• Flail Chest = > 3 consecutive rib fractures in 2 or more places; produces a free-floating, unstable segment of chest wall → paradoxical chest wall movement
• Major cause of respiratory insufficiency in Flail Chest = pulmonary contusion; Treatment = aggressive pulmonary toilet, intercostal nerve blocks, indwelling epidural catheters, CPAP; judicious fluid administration \[10, 5th ed., pgs. 384-385\]; ↑ morbidity (pneumonia, sepsis, pneumothorax, ↑ admission)
• Most common significant chest injury in PEDS = pulmonary contusion; > 25% often require mech. ventilation
• Indications for thoracotomy = initial chest tube drainage > 20ml/kg or 1,500 ml of blood; unstable VS; > 300-400 cc/hr (or 7 cc/kg/hr) for 4 hours; ↑ing hemothorax on CXR; patient decompensates after initial response to resuscitation; use large chest tube (34 – 40F)
• Hemothorax can be seen on upright CXR with 200 to 300cc of blood
• Communicating/open pneumothorax (“sucking chest wound”) treatment = EMS = occlusive dressing taped on three sides; ED treatment = chest tube and occlusive dressing; GSW to chest leaves defect (hole) in thoracic wall
• No association of sternal fractures and aortic rupture
• 90% of Blunt Aortic Injuries occur in descending aorta at the isthmus between left subclavian artery and the ligamentum arteriosum; 80% die at scene; 50% who survive die in 24 hours
• Most common area of heart injured in blunt trauma = Right ventricle; most common rhythm = sinus tachycardia; other = A. Fib
• Most common valvulopathy due to chest trauma = Aortic Regurgitation (AR)
• Beck’s Triad: 1) Muffled heart tones 2) ↓BP 3) JVD (↑CVP) = cardiac tamponade
• Most common echocardiographic findings with cardiac tamponade = right ventricular diastolic collapse
• Electrical alternans on EKG is pathognomonic for tamponade
• Air Embolism – place patient head and left side down → decrease air leaving through RV outflow tract
• Esophagus is the most rapidly fatal perforation of the GI tract; Hamman’s crunch
• Most common site of diaphragmatic injury = Posterolateral; left >> right (liver protects on right); 5% bilateral. FYI: most common site herniated disks rupture, and almost all spinal hematomas = Posterolateral location
• Diaphragmatic injuries are more common in penetrating trauma; CXR diagnostic only 10 to 40%;
• Needle cric if < 8 years old

**Indications for Percutaneous Transtracheal Ventilation (“Needle Cric”)**
1. Cannot control airway with standard interventions or LMA
2. Severe maxillofacial trauma
3. Obstructive processes

**Contradintiations for Percutaneous Transtracheal Ventilation (“Needle Cric”)**
1. Damaged cricoid cartilage
2. Tracheal rupture
3. Caution with complete upper airway obstruction

**Indications for Tube Thoracostomy**
- Pneumothorax - Open or closed (moderate to large) or Tension
- Respiratory symptoms regardless of size of pneumothorax
- Hemothorax, Hemopneumothorax, Hydrothorax, Chylothorax, Empyema, large Effusion
- Patients with pneumothorax who are intubated or about to be intubated
- Patients with pneumothorax about to undergo air transport
- Bilateral Ptx regardless of size

**Complications of Tube Thoracostomy**
pulmonary edema, contralateral ptx, infection (cellulitis, empyma), bronchopleural fistula, pleural leak, formation of hemothorax (lung parenchyma, vs intercostal artery injury), placement (in abdomen, SQ tissue etc), intercostal vessel or nerve injury (avoid inferior margin of rib)

**Indications for Emergency Department Thoracotomy (EDT) are determined by**
- Presence of signs of life
- Mechanism of injury
- Location of injury

**Increased thoracotomy survival rates are associated with**
- Signs of life in the emergency department (ED)
- Thoracic injuries (as opposed to abdominal injuries)
- Penetrating injuries (as opposed to blunt injuries): Survival in blunt cardiac injury is less than with penetrating cardiac injury secondary to poor cardiac function (due to cardiac contusion) and a higher incidence of associated injuries (cardiac rupture and aortic rupture)
- Stab wounds (as opposed to GSW): GSW injuries are usually unable to spontaneously seal because of large injury pattern. If patients present with any signs of life, they are usually in profound hemodynamic compromise

**“Relative” indications for EDT**
- Penetrating thoracic injury with traumatic arrest without previously witnessed cardiac activity
- Penetrating nonthoracic injury (eg, abdominal, peripheral) with traumatic arrest with previously witnessed cardiac activity (pre-hospital or in-hospital)
- Blunt thoracic injuries with traumatic arrest with witnessed cardiac activity (pre-hospital or in-hospital)

**OB TRAUMA PEARLS**
- Primary cause of fetal death in trauma = maternal shock and death
- Second most common cause of fetal death in trauma = placental abruption; 30% of placental abruptions after trauma will not have vaginal bleeding[ATLS, 8th ed., J Trauma 2008;64:1638-1650]
- Placental abruption is a clinical diagnosis = vaginal bleeding, abdominal pain, uterine tenderness, uterine contractions, and fetal distress
- Most common cause of blunt abdominal trauma in pregnancy = MVC 70%; others = falls, direct assault
DESCRIBING ORTHOPEDIC RADIOGRAPHS

Open vs. Closed

Fractures line

Relates to long axis of involved bone (spiral, oblique or transverse)
Simple vs. Comminuted (more than 2 fractures segment)

Location

Which bone is fractured, left vs. right, dominant vs. non-dominant hand, approximate location-proximal, middle or distal 1/3 for long bones, use standard reference points – humeral neck, tibial plateau or intertrochanteric region of femur, extra/intraarticular extension etc.

Position of bone fragments

Displacement: describe the DISTAL fragment in relation to the proximal one (describe in %)
Alignment: describes relationships of longitude axis of one fragment to another
Angulation: any deviation from normal alignment, describe by direction of the apex of the angle formed by the two fragments – give degree and direction (dorsal vs. volar or radial vs ulnar) of deformity. The angle is OPPOSITE the direction of displacement of the distal fragment.

Lateral

Proximal ← Medial → Distal

Distraction without displacement or angulation
Lateral displacement (25% - 50%) without angulation
Complete 100% lateral displacement with shortening and without angulation
Lateral angulation (30°) without displacement
Lateral displacement about 50% and lateral angulation (45°)
Complete medial displacement with shortening and lateral angulation (about 45°)
INTERPRETING C-SPINES
Mnemonic - (ABC’s) [ANK/9, with modifications]

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Alignment</td>
</tr>
<tr>
<td>B</td>
<td>Bones</td>
</tr>
<tr>
<td>C</td>
<td>Cartilage</td>
</tr>
<tr>
<td>s</td>
<td>Soft-tissue</td>
</tr>
</tbody>
</table>

See all 7 cervical vertebrae down to the top of T1

**Alignment** check 3 lines → smooth lordotic curve at 1) anterior and 2) posterior aspect of vertebral bodies and the 3rd line, which is spinolaminar line

- PEDS: 4% of kids < 8 years have pseudosubluxation of C2-C3

**Bones** check vertebral bodies; ensure anterior and posterior heights are similar (> 3mm difference suggests fracture)

**Cartilage** intervertebral joint spaces and facet joints

**Soft-tissue**

**Prevertebral swelling, 6mm at C2 and 22mm at C6**
Measure from anterior border of C2 to posterior wall of pharynx (6-at-2 and 22-at-6)
PEDS, < 15 years old, same holds for C2, however at C6 <14mm

**Predental space** = space from the anterior aspect of the odontoid process and the posterior aspect of the anterior ring of C1; normal predental space =

- < 3mm in adults
- < 5mm in children

Widen predental space = C1-C2 injury

**HISTORY FOR C-SPINE**
Mnemonic - (A MUST) [9]

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Altered mental status</td>
</tr>
<tr>
<td>M</td>
<td>Mechanism</td>
</tr>
<tr>
<td>U</td>
<td>Underlying condition</td>
</tr>
<tr>
<td>S</td>
<td>Symptoms</td>
</tr>
<tr>
<td>T</td>
<td>Timing (when symptoms began in relation to event)</td>
</tr>
</tbody>
</table>

**Nexus Criteria for C-spine imaging (if any criteria present, cannot clear C-spine clinically)**
- Midline spinal tenderness
- Focal neurological deficit
- Altered level of consciousness
- Intoxication
- Distracting injury present
**UNSTABLE C-SPINE FRACTURES**
**Mnemonic - (Jefferson Bit Off a Hangman’s Thumb)**

<table>
<thead>
<tr>
<th>Jefferson</th>
<th>Jefferson fx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bit</td>
<td>Bilateral facet dislocation</td>
</tr>
<tr>
<td>Off a</td>
<td>Odontoid fx</td>
</tr>
<tr>
<td>Hangman’s Thumb</td>
<td>Hangman’s fx</td>
</tr>
<tr>
<td>Thumb</td>
<td>Teardrop fx</td>
</tr>
</tbody>
</table>

**POSTERIOR HIP DISLOCATIONS**
**Mnemonic - (DIP)**
Patients who take a “DIP” may have a posterior hip dislocation, most common hip dislocation 80%.

| D | ADDucted          |
| I | Internally rotated |
| P | Posterior dislocation (most common hip dislocation) |

**GALEAZZI’S FRACTURE**
**Mnemonic - (SURF-surf the sea of Galilee)**

| S | Subluxated          |
| U | Ulna (distal radial-ulnar joint (DRUJ)) |
| R | Radial shaft (middle/ distal junction) |
| F | Fracture            |

**MONTEGGIA FRACTURE**
**Mnemonic - (BURD)**

| B | Broken             |
| U | Ulna (proximal)    |
| R | Radial head        |
| D | Dislocation (anterior in 60%) |

√ Radial nerve → wrist extension; also → posterior interosseus branch → finger extension; and another branch of radial nerve, which is purely sensory → sensation dorsum of hand [12, 6th ed., pg. 1691]

- Usually requires surgical fixation
**COLLES FRACTURE**
(Remember Collie = Dog)

<table>
<thead>
<tr>
<th>D</th>
<th>Distal Radial fracture</th>
</tr>
</thead>
<tbody>
<tr>
<td>D</td>
<td>Dorsal displacement of radial fragment</td>
</tr>
<tr>
<td>D</td>
<td>Dinner-fork deformity</td>
</tr>
</tbody>
</table>

60% with Colles fractures have ulnar styloid fracture

Check median nerve
Reverse Colles = Smith’s Fracture
Barton Fracture = dorsal or volar rim fx of distal radius – often fx/dislocations or subluxations

**EVALUATION OF ELBOW RADIOGRAPHS IN KIDS**
Mnemonic - (Careful Resurrection Medical Training Offers Learning)

<table>
<thead>
<tr>
<th>Ossification Center</th>
<th>First Appears (year/old)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Careful</td>
<td>Capitellum</td>
</tr>
<tr>
<td>Resurrection</td>
<td>Radial head</td>
</tr>
<tr>
<td>Medical</td>
<td>Medical epicondyle</td>
</tr>
<tr>
<td>Training</td>
<td>Trochlea</td>
</tr>
<tr>
<td>Offers</td>
<td>Olecranon</td>
</tr>
<tr>
<td>Learning</td>
<td>Lateral epicondyle</td>
</tr>
</tbody>
</table>

Comparison views are helpful for evaluating elbow radiographs in kids
Other mnemonic = CRITOE → I = Internal (Medial) and E = External (Lateral epicondyle)

**THE OTTAWA ANKLE RULES**
Ankle x-ray series are only required if there is pain in the malleolar zone and any one of the following findings:

1) Bone tenderness along the distal 6 cm of the posterior edge of the fibula or tip of lateral malleolus
2) Bone tenderness along the distal 6 cm of the posterior edge of the tibia or tip of medial malleolus
3) Inability to bear weight both immediately and in the ED for 4 steps

Foot x-ray series are only required if there is tenderness in the midfoot zone and any one of the following findings:

1) Bone tenderness at the base of the fifth metatarsal
2) Bone tenderness at the navicular bone
3) Inability to bear weight both immediately and in the ED for 4 steps

Ottawa Ankle Rules not developed for patients < 18 years old
Clinical judgment should prevail = if exam is unreliable (ETOH, lack of cooperation, distracting injuries or diminished sensation in the leg) get x-rays
BONES OF THE WRIST

Mnemonic - (Some Lovers Try Positions That They Can’t Handle) [ANK]

<table>
<thead>
<tr>
<th>Some</th>
<th>Scaphoid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lovers</td>
<td>Lunate</td>
</tr>
<tr>
<td>Try</td>
<td>Triquetrum</td>
</tr>
<tr>
<td>Positions</td>
<td>Pisiform</td>
</tr>
<tr>
<td>That</td>
<td>Trapezium</td>
</tr>
<tr>
<td>They</td>
<td>Trapezoid</td>
</tr>
<tr>
<td>Can’t</td>
<td>Capitate</td>
</tr>
<tr>
<td>Handle</td>
<td>Hamate</td>
</tr>
</tbody>
</table>

Begin mnemonic:
1st row → RADIAL, proximal row → ULNARLY; scaphoid → pisiform
2nd Row → RADIAL, distal row → ULNARLY; trapezium → hamate

ROTATOR CUFF MUSCLES

Mnemonic - (SITS)

<table>
<thead>
<tr>
<th>S</th>
<th>Supraspinatus → ABDuction; most common rotator cuff muscle injured (85-90%) (“Empty Can Test”)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Infraspinatus → External rotation</td>
</tr>
<tr>
<td>T</td>
<td>Teres Minor → External rotation and ADDuction</td>
</tr>
<tr>
<td>S</td>
<td>Subscapularis → Internal rotation</td>
</tr>
</tbody>
</table>

All 4 muscles originate on the scapula, transverse the glenohumeral joint and insert on the proximal humerus

FELTY’S SYNDROME

Mnemonic - (FAULTS) [1]

<table>
<thead>
<tr>
<th>F</th>
<th>Felty syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Arthritis – chronic Rheumatoid arthritis (affects &lt; 1% patients with RA)</td>
</tr>
<tr>
<td>U</td>
<td>Ulcers of leg (pyoderma gangrenosum)</td>
</tr>
<tr>
<td>L</td>
<td>Leukopenia and neutropenia (low PMN’s)</td>
</tr>
<tr>
<td>T</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>S</td>
<td>Splenomegaly (spleen is often FELT)</td>
</tr>
</tbody>
</table>
SYNOVIAL FLUID ANALYSIS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Appearance</th>
<th>WBC's/mm</th>
<th>% PMN's</th>
<th>Glucose; % Serum Level</th>
<th>Crystals Under Polarized Light</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear</td>
<td>&lt;200</td>
<td>&lt;25</td>
<td>95-100</td>
<td>None</td>
</tr>
<tr>
<td>Non-Inflammatory (i.e. DJD)</td>
<td>Clear</td>
<td>&lt;400</td>
<td>&lt;25</td>
<td>95-100</td>
<td>None</td>
</tr>
<tr>
<td>Acute Gout</td>
<td>Turbid</td>
<td>2000-5000</td>
<td>&gt;75</td>
<td>80-100</td>
<td>Negative birefringence; needle-like crystals</td>
</tr>
<tr>
<td>Pseudogout</td>
<td>Turbid</td>
<td>5000-50,000</td>
<td>&gt;75</td>
<td>80-1000</td>
<td>Positive birefringence; rhomboid crystals</td>
</tr>
<tr>
<td>Septic Arthritis</td>
<td>Purulent/ turbid</td>
<td>&gt;50,000</td>
<td>&gt;75</td>
<td>&lt;50</td>
<td>None</td>
</tr>
<tr>
<td>Inflammatory (i.e. Rheumatoid arthritis)</td>
<td>Turbid</td>
<td>5000-50,000</td>
<td>50-75</td>
<td>Approx. 75</td>
<td>None</td>
</tr>
</tbody>
</table>


ORTHOPEDIC PEARLS

- Most commonly fractured bone in the body = clavicle (middle third most common site, 85%); S-shaped bone

- Most common dislocation of shoulder = ANTERIOR; check axillary nerve (pinprick sensation over the skin of deltoid muscle)\[12, pg. 1240\]; if shoulder dislocation secondary to seizure or electric shock than → posterior dislocation (subacromial being common type of posterior dislocations)

- Most common nerve injury with humeral shaft fx = radial nerve (wrist and finger extension, sensation dorsum of hand). Other injuries: brachial artery or vein, ulnar and median nerves\[10, 5th ed, pg. 563\]

- Most common dislocation of elbow = POSTERIOR; check ulnar nerve (intrinsic muscles of hand and sensation over ulnar side of hand); vascular injuries 5 to 13%, most common = brachial artery

- Most common bursitis = olecranon bursitis

- Tennis elbow (lateral epicondylitis) confirmed = reproduce pain with Elbow in extension, wrist flexion and forearm pronation against resistance\[12, pg. 1298\] or With forearm pronated actively extend fingers or wrist against resistance or Pinching with the wrist in extension

- Posterior fat pad on x-ray = supracondylar or radial head fracture (most common elbow fx in adults)

- Most common carpal fx = scaphoid > triquetrum 2nd > lunate 3rd
• Most common dislocated carpal bone = lunate

• Scapholunate gap > 3mm = “Terry Thomas” sign, an indicator of scapholunate dissociation

• Boutonniere Deformity = disruption of extensor hood near the PIP → PIP flexion and DIP extension

• Mallet finger = disruption of extensor tendon at the DIP; immobilize in extension for 8 weeks

• Jersey finger = Avulsion of flexor digitorum profundus tendon (flexed finger is hyperextended); Ring finger most commonly affected

• Boxer’s fracture = fx of the 5th metacarpal neck, most common angle in volar direction

Acceptable Metacarpal Angulation to avoid functional impairment
  ▫ 2nd, 3rd = < 15°
  ▫ 4th = 20°
  ▫ 5th = 40°

• Bennett’s fracture = intraarticular base fractures of thumb; unstable; often require surgery

• Rolando’s fracture = comminuted Bennett’s fracture at the metacarpal base; require surgery; worse prognosis

• Most common cause of Gamekeeper’s thumb skiing (not twisting the necks of hares); Ulnar collateral ligament (UCL) ruptures 10x more often then radial CL. Treat complete rupture of UCL with surgery, incomplete tear – thumb spicca cast 4 weeks

• UCL rupture if > 35% joint laxity or > 15% more laxity than is present in uninjured thumb; know Stenner’s lesion (UCL is prevented from healing by the interposed adductor aponeurosis → chronic instability)

• Finkelstein’s test = diagnose De Quervain’s tendonitis (tendonitis of the abductor pollicis longus and the extensor pollicis brevis)

• Phalen’s test more sensitive test for carpal tunnel syndrome vs Tinel’s sign; nerve conduction studies used to confirm the diagnosis with Sn 90%

• Most sensitive bedside test for nerve injury in a finger = two-point discrimination

• Most common dislocation of hip = POSTERIOR; check sciatic nerve and acetabular fx

• Most common complication with Femoral Neck Fracture = avascular necrosis

• Femoral shaft fracture may result in loss of > 1 liter of blood

• Tibial plateau fx – lateral more common, check peroneal nerve. Cause: direct force driving femoral condyles into articulating surface of the tibia or direct trauma. Check for lipohemarthrosis on x-ray; more than 5mm of depression = surgical repair

• Knee dislocation = ortho emergency = 50% injury to popliteal artery

• Most common ligamentous knee injury = medical collateral

• Most common dislocation of patella = lateral
• Most common cause of acute traumatic knee hemarthrosis = injury to anterior cruciate ligament (ACL)

• Accuracy increases from 70 % using Anterior Drawer test to 99% using Lachman’s Test in dx ACL injury

• Most common meniscal injury = medical meniscus – less mobile. Knee locks up; Get delayed swelling

• Most common long bone fx = tibia [12, pg. 1263]

• Most common open bone fx = tibia

• Most common site for compartment syndrome in lower extremity = anterior compartment (note: anterior tibial artery deep & peroneal nerve) [12, pg. 1264]; normal pressure = 0 to 8 mmHg; pressures > 30 mmHg cause ischemia

• Most common presenting symptom of compartment syndromes = pain

• Most commonly injured ankle ligament = anterior TALOfibular ligament [12, pg. 1267]

• Maisonneve fracture = eversion mechanism → proximal fibular fx + disruption of the deltoid ligament or a medial malleolar fx + partial or complete disruption of the syndesmosis

• Pilon fracture = comminuted fracture of the distal tibial metaphysis combined with disruption of the talar dome

• Most common tarsal bone fracture = calcaneus; 75% intraarticular [10, 5th ed., pg. 724]

• Calcaneal fxs rule of 10’s = 10% bilateral, 10% vertebral compression fractures, 10% compartment syndrome; 25% lower extremity injuries (tibial plateau fxs, etc)

• Bohler’s angle = normal 20 to 40 degrees, if less suspect calcaneal fracture

• Most common midfoot fracture = Navicular (uncommon)

• Most common forefoot fracture = Phalangeal fractures

• Most common metatarsal base fracture = 5th metatarsal

• Most common metatarsals fractured = 3rd metatarsal [10, 5th ed., pg. 725, 728,729]

• Jones Fracture = transverse fracture through the base of 5th metatarsal, 1.5cm distal to the proximal part of the metatarsal; treatment = NWB cast for 6 weeks [12, 6th ed., pg. 1745]

35-50% patients develop persistent nonunions requiring bone grafting and internal fixation


• Pseudo-Jones = avulsion fx, more common, peroneus brevis tendon pulls off a portion of the bone where it inserts; treatment = cast shoe [12, 6th ed., pg. 1745]

• Most common undisplaced metatarsal shaft fracture 2nd-5th tx = below-knee walking cast 2 to 4 weeks

Non-displaced 1st metatarsal fractures treated with cast 4 to 6 weeks and NWB

• Most common metatarsals involved in stress – “March” – fractures = 2nd and 3rd (fixed)
• 2 bones in the hindfoot (calcaneus, talus), 5 bones in the midfoot (navicular, cuboid, 3 cuneiforms), and 19 bones in the forefoot (5 metatarsals, 14 phalanges)

• Hindfoot connects to the midfoot at the Chopart joint; forefoot connects to the midfoot at the Lisfranc joint

• Lisfranc Fracture = disruption of tarsal-metatarsal joint, fracture at base of 2\textsuperscript{nd} metatarsal

• Thompson test check Achilles tendon rupture (plantar flexion weakness/absent)\textsuperscript{[12, pg. 1303]}
  Forced dorsi flexion, palpable gap
  Treatment = open repair or cast immobilization for 8 weeks
  ED management = Gravity Equinus Splint = below-the-knee with ankle plantar flexion

• Kanavel’s 4 cardinal signs for flexor tendon sheath infection
  1) slight flexion of digit
  2) Swelling (“Sausage Finger”)
  3) Tenderness over flexor tendon sheath
  4) Pain on passive extension

• Most common infection with puncture wound to foot = \textit{Pseudomonas}

• Most common cause of osteomyelitis = \textit{Staph aureus}; in sickle cell patients, think Salmonella after staph

• Most common complication associated with leg fractures = infection

• Most common bacteria causing septic joint = \textit{Staph aureus}; most common joint = knee

• Fight bite injury = \textit{Eikenella corrodens}

• Infection of the deep space of the fingertip = felon; treatment = I&D

• Paronychia involves the dorsal aspect of the nail area; treatment = I&D

• Most common viruses causing arthritis = Rubella (German measles) and HBV; others = Parvovirus B19, EBV, mumps, adenovirus and enteroviruses

• Most common joint involved in Gout = Toe; crystals = uric acid; needle shaped, blue; neg. birefringence

• Most common joint involved in Pseudogout = knee; crystals = calcium pyrophosphate; rhomboid, yellow

• Acute Gout/ Pseudogout therapy options = NSAIDs, oral colchicines (more effective with Gout; bad vomiting and diarrhea); cold compresses

\textbf{If contraindication to NSAIDs}
  a) Adrenocorticotropic (ACTH) = Day 1 80 IU IM, q 8h; Day 2 40 IU q 12; Day 3-14 40 IU daily
    → stimulates cortisol → inhibits inflammation\textsuperscript{[10, 5th ed. Pg. 1592][Drugs 2008:68:407-415]}
  b) Oral prednisone 30 -60 mg daily with taper over 10 to 14 days
    If > 5 joints involved → 3 week taper
  c) Intra-articular steroid injection – triamcinolone 10mg in knees, 8 mg in smaller joints\textsuperscript{[Drugs 2008:68:407-415]}

• Long term Gout/ Pseudogout therapy = allopurinol (↓ uric acid production) or probenecid (↑ uric acid excretion)
RISK FACTORS FOR SUICIDE

Mnemonic - (SAD PERSONS) [ANK]

<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Characteristic</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>Sex</td>
<td>Male</td>
</tr>
<tr>
<td>A</td>
<td>Age</td>
<td>&lt; 19 or &gt; 45</td>
</tr>
<tr>
<td>D</td>
<td>Depression</td>
<td>↓concentration, appetite</td>
</tr>
<tr>
<td>P</td>
<td>Previous psych hx</td>
<td>Panic, depression, schizo</td>
</tr>
<tr>
<td>E</td>
<td>ETOH</td>
<td>Stigmata of ETOH abuse</td>
</tr>
<tr>
<td>R</td>
<td>Rational thinking loss</td>
<td>Organic brain syndrome</td>
</tr>
<tr>
<td>S</td>
<td>Separated</td>
<td>Separated &gt; divorced &gt; widow &gt; single &gt; married (least likely)</td>
</tr>
<tr>
<td>O</td>
<td>Organized attempt</td>
<td>Plan</td>
</tr>
<tr>
<td>N</td>
<td>No social support</td>
<td>No close family or friends</td>
</tr>
<tr>
<td>S</td>
<td>Stated future attempts</td>
<td>Determined to repeat</td>
</tr>
</tbody>
</table>

Total scores > 6 should undergo psychiatric care directly from the ED

- Females attempt more frequently; males succeed more often
- 20% retry within one year
- 5% of repeaters succeed
- Personality disorders (antisocial, histrionic, narcissistic and paranoid) least likely to commit suicide
- Major Mood Disorder = greatest risk for suicide

DEPRESSION

Mnemonic - (IN SAD CAGES) [ANK]

<table>
<thead>
<tr>
<th>IN</th>
<th>Interest decrease in everything</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>Sleep disorder</td>
</tr>
<tr>
<td>A</td>
<td>Appetite alteration</td>
</tr>
<tr>
<td>D</td>
<td>Dysphoric mood</td>
</tr>
<tr>
<td>C</td>
<td>Concentration decreases</td>
</tr>
<tr>
<td>A</td>
<td>Affect blunted</td>
</tr>
<tr>
<td>G</td>
<td>Guilt</td>
</tr>
<tr>
<td>E</td>
<td>Energy diminishes</td>
</tr>
<tr>
<td>S</td>
<td>Suicide risk</td>
</tr>
</tbody>
</table>
• 20% of all psych referrals have organic etiologies
  Medical features = abrupt onset, age > 40, visual or tactile hallucinations, abnormal vital signs

• Haldol till they crawl-keep ED safe

• BS2 = drop “bomb” on agitated patients = Haldol 5 mg + Ativan 2 mg (can mix the two drugs together in
  same syringe, “compatible”, then give one shot IM

• Most common psychiatric disease = schizophrenia (delusions-most often persecutory, auditory
  hallucinations, loose associations, catatonia, flat affect)

• Most common cause of dementia in elderly patients = Alzheimer’s disease

• Major Mood Disorder = greatest risk for suicide

• The most common DSM-IV diagnostic group for pediatric patients in the ED = substance disorders

  If you have a “difficult patient” – insolvable problems, multiple visits, hostility, name dropping excessive
  need for attention and threats consider BPD [10, 5th ed., 2005].

**BORDERLINE PERSONALITY DISORDER (BPD)**
Diagnostic & Statistical Manual (DSM) IV Criteria

A pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity
beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Frantic efforts to avoid real or imagined abandonment. Note: Do not include suicidal or self-mutilating
   behavior covered in Criterion 5.

2. A pattern of unstable and intense interpersonal relationships characterized by alternating between
   extremes of idealization and devaluation

3. Identity disturbance: markedly & persistently unstable self-image or sense of self

4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse,
   reckless driving, binge eating). Note: Do not include suicidal or self-mutilating behavior covered in
   Criterion 5.

5. Recurrent suicidal behavior, gestures, threats, or self-mutilating behavior

6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability or
   anxiety usually lasting a few hours and only rarely more than a few days).

7. Chronic feelings of emptiness

8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant
   anger or recurrent physical fights)

9. Transient, stress-related paranoid ideation or severe dissociative symptoms
ECTOPIC PREGNANCY (EP) PEARLS

- 15% of clinically recognized pregnancies terminate in miscarriage
- Mean gestational age for ectopic rupture = 7.0 + 2 weeks
- Recent estimate of heterotopic pregnancy = 1 in 4,000 pregnancies: if women have undergone embryo transfer or use of ovulation-inducing drugs → incidence = 1 in 100
- Most common site of EP implantation = ampullary (80%) portion of the fallopian tube
- ↑maternal mortality rate if EP implantation = cornual location
- Monoclonal antibody assays detect presence of β-hCG as soon as 2-3 d postimplantation
  The earliest a serum beta-hCG test can detect pregnancy = shortly before missed period
  Usually reaches 200 IU/ml at time of menses
- In a normal pregnancy, β-hCG doubles every 2 days or increase by 66% every 3 days for the first 6-7 weeks beginning 8-9 days after ovulation
- After 9-10 weeks gestation, beta-hCG levels decline
- 10% of normal pregnancies can manifest abnormal doubling times
- 15% of EP’s can have normal doubling times
- Evidence of IUP should be seen by transabdominal ultrasound with beta-hCG levels of 6,500 mIU/mL, or at least 1,500 mIU/mL using TVUS (discriminatory threshold)
- Rupture can occur in patients with beta-hCG levels as low as 100 mIU/mL
- The most common clinical presentation of patients with EP = abdominal pain (80%); vaginal bleeding 50-80%)

RISK FACTORS FOR ECTOPIC PREGNANCY

<table>
<thead>
<tr>
<th>LESSER RISK</th>
<th>GREATER RISK</th>
<th>GREATEST RISK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous pelvic or abdominal sx</td>
<td>Previous PID</td>
<td>Pervious ectopic pregnancy</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>Infertility (IVF)</td>
<td>Pervious tubal surgery or sterilization</td>
</tr>
<tr>
<td>Vaginal douching</td>
<td>Multiple sexual partners</td>
<td>Diethylstilbestrol exposure to utero</td>
</tr>
<tr>
<td>Age of 1st intercourse &lt; 18 yrs</td>
<td></td>
<td>Documented tubal pathology (scarring)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use of IUD</td>
</tr>
</tbody>
</table>
ECTOPIC PEARLS

<table>
<thead>
<tr>
<th>TV Ultrasound findings</th>
<th>beta-hCG (mIU/mL)</th>
<th>Gestational Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational sac (GS)</td>
<td>1,000</td>
<td>4-5 weeks</td>
</tr>
<tr>
<td>First sonographic finding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yolk sac</td>
<td>1,000 to 7,000</td>
<td>5 weeks</td>
</tr>
<tr>
<td>Small hyperechoic ring in GS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fetal pole (embryo)</td>
<td>1,000 to 7,000</td>
<td>5-6 weeks</td>
</tr>
<tr>
<td>Seen adjacent to yolk sac</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fetal cardiac activity</td>
<td>10,000 to 23,000</td>
<td>6 weeks</td>
</tr>
<tr>
<td>Normal rate early preg. = 112 to 136</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slower rates in 2nd and 3rd trimester</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Menstrual age = 2 weeks older than embryonic age (ovulation typically takes place at midpoint of a typical 28-day menstrual cycle) \([12, \text{6th ed., pgs. 717-721}]\)

**Double-decidual sac sign ("double ring sign")** = Two distinct hyperechoic decidua layers (decidua capsularis and decidua vera) surround the gestational sac (GS). The earliest reliable US finding seen in normal intrauterine pregnancies \([12, \text{6th ed., pg. 717}]\). Should be visualized by 5 weeks after last menstrual period

Earliest definitive sign in the initial diagnosis of an IUP = yolk sac in the uterus; Should be visualized by 5.5 weeks after last menstrual period

Most common cause of misdiagnosis of ectopic pregnancy by TVUS = misinterpretation of the pseudogestational sac as an IUP

**Pseudogestational sac** = anechoic fluid collection without a clear double decidual reaction

Seen in 10 to 20% of ectopics

**Double-decidual sac sign** can be used to differentiate true GS from pseudo-GS

Other US findings in Ectopic = free fluid, adnexal mass, or tubal ring

Ectopic pregnancies are due to anatomic abnormalities of the salpinx (tube), prior tubal infection or an abnormal endometrium

**OB / GYN PEARLS**

- Blighted ovum = inability to visualize yolk sac or fetal pole (embryo) in a large gestational sac on TV ultrasound → major criteria for fetal demise
- Subchorionic hematoma = more than ↑double the chance pregnancy loss in threatened abortion\([12, \text{6th edn., pg. 722}]\)
- **HELLP Syndrome** = Hemolysis, Elevated Liver Enzymes, Low Platelets
  More common in multigravid patient
  Can also occur postpartum \([12, \text{5th edn, pg. 697}]\)
- Preeclampsia = HTN, edema, and proteinuria → more common in primigravida patient \([12, \text{5th edn, pg. 697}]\)
- Treatment of Ecclamptic Seizures or prophylactic treatment of patients with severe preeclampsia = Magnesium sulfate first-line agent
  Loading dose 4 to 6 grams over 15 min, followed by IV infusion of 1 to 2 gm/hour maintained for 24 hours after the last seizure
Observe for nausea, somnolence;
Serum magnesium level 4 to 8 mEq/L = ↓DTRs
Serum magnesium level > 8 mEq/L = apnea
Treatment of hypermagnesemia = Ca Gluconate [10, 5th edn, pg. 2430; eMedicine]

- Most common cause of death in Toxemia = cerebral hemorrhage [10, 5th edn, pg. 2407]
- Most common medical cause of death in pregnant patient = PE
- Most common cause of death in pregnant patient overall = injury (homicide most common injury)
- The leading cause of death in first trimester is ectopic pregnancy [12, 5th edn, pg. 697]
- Placental abruption usually occurs spontaneously (though trauma can cause abruption) and manifests clinically as painful third trimester vaginal bleeding; clinical diagnosis = dark VB, uterine pain or tenderness is seen in 2/3 and uterine irritability or contractions are seen in 1/3 [12, 5th edn, pg. 697][10, 5th edn, pg. 2420] US only 50% accurate in diagnosis. Cocaine is a risk-factor.
- Complications of placental abruption = fetal/maternal death, DIC, amniotic fluid embolism, and fetal/maternal hemorrhage; treatment = delivery
- Placenta previa = common cause of third trimester vaginal bleeding, painless vaginal bleeding occurs. Do not perform vaginal exam if suspected previa/abruption ... it may lead to worsened bleeding
- Most common cause of post-partum hemorrhage = uterine atony
- Most common cause of vaginal bleeding related to primary coagulation disorder = Von Willebrand’s disease [12, 5th ed., pgs 673-674 and 1377-1382]
- Most common causes of vaginal bleeding in prepubertal girls (without precocious puberty) = vulvovaginal abnormalities (vaginitis, vaginal FBs, trauma, and tumors)
- Most common organism in lactational mastitis = is S. aureus Treatment = penicillinase-resistant antibiotic and continued emptying of the breast milk (continued nursing or manual extraction). The breast milk will not harm the nursing infant. Women are encouraged to continue to nurse if able. [12, 5th ed., pg 726]
- Uterine size greater than dates = gestational trophoblastic disease and multiple gestation pregnancies
- Molar pregnancy = 1 in 1,700 pregnancies [12, 6th ed., pgs. 677, 723]
  - 80 % present as hydatidiform mole and follow benign course
  - Malignant forms = Invasive Mole (12-15%) and choriocarcinoma (5-8%)
  - Larger then expected uterine size for gestational age and markedly ↑ β-hCG (>100,000 mIU/mL) are risk factors for malignant disease
  - Choriocarcinoma → may metastasize to vagina/lung/liver/brain and is sensitive to chemotherapy
- Hydatidiform mole US findings = intrauterine echogenic mass with multiple small hypoechoic vesicles interspersed = “grape-like” appearance or “snowstorm appearance”
- Hyperemesis Gravidarum = seen in first 12 weeks; occurs in 2% pregnancies; Zofran is ok in pregnancy; abdominal pain is highly unusual and should suggest another diagnosis [12, 5th ed., pg 677]
- Simple cervicitis = ceftriaxone 250 mg IM and a single dose of azithromycin 1gm po
- Chlamydia urethritis = 20% of women with dysuria (sterile pyuria)
- Two most common causes of PID = GC and Chlamydia. Ceftriaxone IM covers GC, the CDC recommends doxycycline for 10 days in PID
• Acute complications of PID include
  - Tubo-ovarian abscess (TOA)
  - Peritonitis
  - Peri-hepatitis (Fitz-Hugh-Curtis syndrome)

• Treat asymptomatic bacteriuria in pregnancy = 3 -7 days, amox, nitrofurantoin, cephalosporin [Sanford, 2009, pg. 31]

• RhoGAM is indicated for Rh-negative mothers who are exposed to a clinical event (miscarriage, ectopic, placenta previa, abruptio placenta, term pregnancy, trauma) that puts them at risk for Rh isoimmunization as this can negatively impact both their current and subsequent pregnancies → hydrops fetalis
  - RhoGAM must be administered within 72 hours of the event
  - 50 mcg IM < 12 weeks gestational age
  - 300 mcg IM > 12 weeks

• Threshold for human teratogenesis = 5-10 rad; fetus most vulnerable 8 to 15 weeks gestation [12, 6th ed., pg. 675]
  - 1 mGy = 0.1 rad

• V/Q — Total fetal exposure to xenon-133 and technetium-99m = 0.5 rad
  - CXR = 0.00005 rad; CT head < 0.1 rad, CT chest = < 1 rad; CT abdomen = 3.5 rad [12, 6th ed., pg. 675]


• Absolute contraindication to ED speculum and manual pelvic exam = 3rd trimester bleeding

• Tocolytic agents used for the treatment of preterm labor: MgSO4, indomethacin and nifedipine. In the past, terbutaline or ritodrine, were the agents of choice, but in recent years their use has been significantly curtailed due to maternal and fetal side effects = maternal tachycardia, hyperglycemia and palpitations.

• Nitrazine paper pH 7.1 to 7.3 = amniotic fluid; normal vaginal pH in pregnancy = 3.5 to 6.0 [10, 5th ed., pg. 2407]

• Perimortem c-section > 26 weeks gestation, FHTs present; 4 minutes for procedure and 1 min for actual delivery time (70% children who survived were delivered < 5 min) [10, 5th ed., pg. 264]

• Expected physiological changes in pregnancy
  - ↑ HR
  - ↓ SVR
  - ↑ Cardiac Output (HR x SVR)
  - ↑ Blood volume
  - ↓ CVP
  - ↓ SBP which normalizes near term
  - ↑ Minute Ventilation
  - ↑ WBC – mild
  - ↓ BUN / Cr

• 10% of insulin-dependent patients will develop DKA during pregnancy; occurs more rapidly and lower levels of glucose in pregnant patients; hyperemesis and non-compliance most common causes

• pH may be normal in DKA, because the initial pH is ↑ in pregnancy due to physiologic hyperventilation

• Mondor’s Syndrome = superficial phlebitis of the veins in the SQ tissue of the breast; may occur post-op or minor trauma. (Mondor’s disease of penis also described) [10, 5th ed., pg. 1257]

  Menorrhagia = menstruation at regular cycle intervals but with excessive flow and duration
  Metrorrhagia = irregular vaginal bleeding outside normal cycle
  Menometrorrhagia = irregular vaginal bleeding, excessive bleeding, outside normal cycle
  Polymenorrhea = frequent, light, bleeding at intervals < 21 days
  Dysfunctional uterine bleeding = abnormal vaginal bleeding due to anovulation [12, 6th ed., pg. 648]
ACUTE ANGLE-CLOSURE GLAUCOMA (AACG)

Classic history = patient walks into dark room from daylight or by administering a mydriatic → pupil dilation → occlusion of the chamber angle (the canal of schlemm) → aqueous humor build up → ↑IOP → AACG → periorbital pain, ipsilateral HA, blurry vision / vision loss / halos, abdominal pain

TREATMENT ACUTE ANGLE-CLOSURE GLAUCOMA (AACG)

1) Block aqueous humor production
   a. Topical beta-blocker (Timoptic 0.5% one drop) ↓ IOP in 30 to 60 minutes
   b. Oral /IV Acetazolamide (Diamox) 500mg
   c. Topial alpha-2 agonist – Apraclonidine (Iopidine) one drop

2) Reduce vitreous humor volume
   a.Systemic hyperosmotic agent = Mannitol 1 – 2 gm/kg IV

3) Facilitate aqueous outflow
   a. Pilocarpine 1 or 2% one drop 4x daily → after pressure reduced < 40 mm Hg makes pupil miotic → pulling the peripheral iris away from the angle
      (will not work if given early because pressure-induced ischemic paralysis of iris)

4) Decrease the inflammatory reaction and reduce optic nerve damage
   a. Topical steroid = Pred Forte 1% one drop every 15 min for four doses then hourly

Other: analgesics, antiemetics and supine position (the lens falls away from the iris decreasing pupillary block)

Differentiate AACG from Iritis = Iritis has normal cornea, constricted to mid-range pupil, normal IOP, ciliary flush (perilimbal injection = dilation of the blood vessels adjacent to cornea), debris in anterior chamber (cell and flare)

HYPHEMA

If hyphema < 1/3 the anterior chamber = manage outpatient – bedrest, elevate HOB 30 to 45 degrees, limit eye movement (reading)

Treatment of ↑ IOP = use approach above except the pupil needs to be diluted = Atropine 1% one drop three times daily. Atropine will help avoid “papillary play” - constrict/dilate → which stretches iris vessels and promotes bleeding

Avoid Acetazolamide (Diamox) if etiology of hyphema is due to sickle cell disease or if patient is allergic to sulfa

Antifibrinolytic = aminocaproic acid (AMICAR) per ophthalmologist

Major complication = rebleeding after 3 to 5 days; other complications = corneal blood staining, acute/chronic glaucoma, and anterior or posterior synechia formation
CENTRAL RETINAL ARTERY OCCLUSION (CRAO)

- Acute, painless vision loss
- Episodes of Amaurosis fugax
- Pale/gray retina with macular “cherry red spot” (macula is thinnest portion of retina; intact choroidal circulation remains visible through this section of retina)
- Optic disc = boxcar segmentation
- Afferent pupillary defect (APD) (usually not seen with CRVO)
- Causes: embolus (carotid, heart), thrombosis, giant cell arteritis, vasculitis (Lupus), sickle cell disease, hyperviscosity syndromes and trauma \[12, 6th ed., 1460-1461\]

CENTRAL RETINAL ARTERY OCCLUSION TREATMENT

1. Consult ophtho – determine if anterior chamber paracentesis to lower IOP is indicated
2. Massage 15 seconds with sudden release
3. Topical beta-blocker (Timoptic 0.5% one drop)
4. Oral /IV Acetazolamide (Diamox) 500mg
5. Consider having the patient breath into paper bag for 5-10 minutes if no contraindications → ↑ PaCO2 → vasodilation

Treatment futile if > 90 minutes

CENTRAL RETINAL VEIN OCCLUSION (CRVO)

Dilated tortuous retinal veins, cotton-wool spots, macular edema, and optic disc edema; Retinal hemorrhages, may be mild, moderate or large giving a “blood and thunder appearance”

Ischemic = severe visual loss, extensive retinal hemorrhages and cotton-wool spots, presence of relative APD; complication → neovascular glaucoma

Nonischemic = milder form of the disease, less vision loss, no APD

Causes: HTN, DM, CVdisease, Polycythemia vera, Lymphoma, Leukemia, Cloting disorders, Multiple myeloma, Syphilis, Sarcoidosis, Autoimmune disease – SLE and Oral contraceptive use

No known effective medical treatment is available for either the prevention of or the treatment of CRVO. Identifying and treating any systemic medical problems to reduce further complications is important

ACUTE VISUAL LOSS

Mnemonic - (CAN U GO STARE AT HIM) [http://canadiem.org/2016/01/12/tiny-tip-can-u-go-stare-at-him/ and Dr. Postel]
**OPHTHALMOLOGY PEARLS**

- Right eye = OD, left eye = OS. OD acuity appears above the OS acuity when written on the chart.

- 20/200 vision OD = right eye sees at 20 feet what a normal eye sees at 200 feet [12, 5th ed., pg 1505]

- Normal intraocular pressure (IOP) = 10 to 20 mm Hg (the # 20 seems important: compartment syndrome suspected if pressures > 20, normal intracranial pressure (ICP) < 20 mmHg)

- Afferent pupillary defect (APD) (Marcus-Gunn pupil) = affected pupil dilates in response to light. Conditions with APD = CRAO and optic neuritis (lesion on retina or optic nerve)

- Most common cause of acute reduction of vision due to optic nerve dysfunction in pt 20 - 40 years of age = optic neuritis; APD; VA reduced rapid and painful vision loss; papilledema; IV steroids = slightly lower 2-year risk of developing MS; oral steroids contraindicated (ONTT = optic neuritis treatment trial) [12, 6th ed., pg 1460]

- Amaurosis fugax = Greek amaurosis = darkening, Latin fugax =fleeting, is a transient monocular visual loss

- Orbital Cellulitis = Proptosis, ophthalmoplegia, edema & erythema of the eyelids, pain on eye movement, fever, headache, and malaise; CT orbits, IV Abx, consult; Up to 11% of cases result in visual loss

- Most common cause of Orbital Cellulitis = ethmoid sinusitis (90%); Risk for cavernous sinus thrombosis

- Hutchinson’s sign = HZ lesions on tip nose – prognostic of corneal involvement; herpes zoster ophthalmicus → HZ travels down V1 → nasociliary nerve (branches innervate cornea and skin)

- Retinal detachment = flashing lights, floaters and vision loss. Vision loss: cloudy, irregular, or curtain-like

- Synechia = iris adheres to either the cornea (anterior synechia) or lens (posterior synechia)
  - Anterior synechia → prevent drainage of aqueous humor → closed angle glaucoma
  - Posterior synechia → block aqueous from posterior chamber to anterior chamber → increased IOP

- Causes = trauma, iritis or iridocyclitis

- Hallmark physical finding in Bechet’s Syndrome = hypopyon uveitis is seen rarely; recurrent painful aphthous ulcers of oral mucosa and genitals are more common findings [10, 5th ed., pg 1615]

- Uveitis = uveal tract inflammation; tract consists of three segments, the iris, the ciliary body and the choroid

- Most common cause of blindness in AIDS patient = CMV

- CMV retinitis occurs in 10 to 30% HIV-infected patients [10, 5th ed., pg 1853]

- Treat super glue/crazy glue (cyanoacrylate) = erythromycin ointment [12, 6th ed., pg 1459]

- Alkali burns (liquefactive necrosis) more destructive than acid burns → irrigate copiously, check pH (Normal eye pH = 7.0 to 7.4)
• Cotton wool spots = ↓ retinal blood flow → damage to nerve fibers → puffy white patches on the retina; most common causes = HTN and DM

• Corneal abrasion resulting from tree branch → secondary infection = Pseudomonas aeruginosa

• Corneal abrasion resulting from prolonged contact lens wear → secondary infection = Pseudomonas aeruginosa
CAUSES OF HYPERKALEMIA

Mnemonic - (RAD MY LAD) \[^3\]

<table>
<thead>
<tr>
<th>R</th>
<th>Renal Insufficiency; RTA type 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Adrenal Insufficiency</td>
</tr>
<tr>
<td>D</td>
<td>Drugs</td>
</tr>
<tr>
<td></td>
<td>K+ sparing = Triamterene and Spironolactone</td>
</tr>
<tr>
<td></td>
<td>Transcellular shifts = Succinylcholine, β-blockers and digoxin</td>
</tr>
<tr>
<td></td>
<td>Drug induced hypoaldosteronism = ACE inhibitors, NSAIDs, Heparin, Cyclosporin and Bactrim</td>
</tr>
<tr>
<td>MY</td>
<td>MYonecrosis / Cell injury (rhabdomyolysis, burns, crush / acute tumor lysis syndrome</td>
</tr>
<tr>
<td>L</td>
<td>Lack of Insulin → DKA (transcellular shift)</td>
</tr>
<tr>
<td>A</td>
<td>Acidosis (transcellular shift)</td>
</tr>
<tr>
<td>D</td>
<td>Digitalis Toxicity (↓K+, ↓Mg2+ and ↑Ca2+→ ↑dig toxicity)</td>
</tr>
</tbody>
</table>

Most common cause of markedly ↑ K+ = lab error \[^10, 5th ed., pg 1730\]

**RHABDOMYOLYSIS**

- CPK = 5x normal
- Urine = + blood with 0 RBCs
- Most common metabolic abnormality in rhabdo = ↓Ca2+
  Early in rhabdo you get calcium deposition in injured muscle. Later, ↑ Ca2+ secondary to mobilization of deposited calcium and secondary hyperparathyroidism
- Other lab findings: ↑ K+, metabolic acidosis, acute renal failure, DIC
- ↓K+ or ↓Phos = contribute to development of rhabdomyolysis (K+ and phos released from injured muscle so later serum levels may be falsely normal or elevated) \[^10, 5th ed., pg 1729, 1741\]
- Causes: traumatic, exercise induced, toxicologic (CO, toluene, statins, ASA, caffeine, ETOH, neuroleptics/antipsycotics, cocaine/sympathomimetics), environmental (hypo or hyper-thermia, metabolic ↓K+, ↓Phos, ↑ or ↓Na+, hypo or hyper-thyroidism, DKA or HHS), infectious (influenza most common), snake bites, black widow spider bites, immunologic and inherited
- Treatment: fluid (isotonic crystalloid 500 mL/h and titrate to maintain a urine output of 200 mL/h), urinary alkalinization, mannitol and loop diuretics
- Hemodialysis: persistent hyperkalemia despite therapy, severe acid-base disturbances, refractory pulmonary edema and progressive renal failure.
**TREATMENT OF HYPERKALEMIA**  
Mnemonic - (C BIG K DROP)[16]

<p>| | |</p>
<table>
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</table>
| C | **Calcium** chloride or gluconate  
Calcium gluconate 10cc of a 10% solution slowly over 2 min (stop if bradycardic)  
Peds = 1.0 mL/kg, not to exceed 10ml, of 10% calcium gluconate solution over 3-5 min  
If CaCl use central line (CaCl has 3x more Ca2+ than Ca gluconate)  
Most rapid and effective treatment, stabilizes cardiac membrane without changing serum K+ level  
Avoid in Digitals toxicity; can use 2gm of Mag sulfate over 5 min if dig-toxic arrhythmia  
Onset of action = < 5 minutes  
Duration of action = 30 to 60 minutes  
Consider repeat dose if EKG changes do not normalize in 3-5 min |
|   |   |
| B | **Bicarb** (rarely indicated in DKA, unless pH < 7.0 or cardiac arrhythmias) 1 amp  
Peds  
Infants = 0.5 mEq/kg IV over 5-10 min  
Children = 1 mEq/kg IV over 5-10 min  
In infants use the 4.2% solution and the 8.4% solution in children and adults  
Onset of action = within minutes  
Duration of action = 15 to 30 minutes  
Because of short duration consider Bicarb drip |
| I | **Insulin**  
5-10 units regular insulin IVP (with the dextrose solution)  
Peds = 0.1 U/kg regular insulin (1 unit regular insulin/ per 5 gram of glucose infused)  
Onset of action = 20-30 minutes |
| G | **Glucose**  
1-2 amps of D50  
Peds = 0.5 gm/kg (2mL/kg) 25% dextrose solution (with insulin over 30 min)  
Bicarb/insulin/glucose combo “shifts” K+ into cells |
| K | **Kayexalate**  
30-50 gm PO/PR mixed with 100cc of 20% sorbitol  
Peds = 1 gm/kg/dose PO/PR  
Resin that exchanges Na+ for K+ in colonic mucosa; Ca/Bicarb/Insulin/Glucose are temporizing measures; kayexalate/diuretics/dialysis = definitive loss of excess K+  
Onset of action = 2 to 12 hours  
Duration of action = 4 to 6 hours |
| D | **Diuretic / Dialysis**  
Albuterol 10 mg Neb → ↑ plasma insulin → shifts K+ into intracellular space  
Little controversial → tachycardia and chest discomfort  
Can be beneficial in patient with renal failure when fluid overload is a concern |
**CAUSES OF HYPERCALCEMIA**

Mnemonic - (VITAMINS TRAP)\(^{[14]}\)

| V | Vitamin D intoxication  
Vitamin A intoxication (transcription factor in osteoclast stimulation) |
<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Immobilization</td>
</tr>
<tr>
<td>T</td>
<td>Thyrotoxicosis → direct stimulation of osteoclastic bone resorption</td>
</tr>
</tbody>
</table>
| A | Adrenal insufficiency (Addison’s)  
Lack of hypocalcemic effect of corticosteroid → ↑ active Vitamin D → ↑ bone resorption & ↑ GI Ca absorption; also decrease renal clearance of calcium |
| M | Myeloma / Milk-alkali syndrome → excessive consumption of Ca and absorbable antacids |
| I | Insufficiency → acute renal failure → ↓ Ca\(^{2+}\)↑ Phos → ↑ PTH → ↑ Ca\(^{2+}\) from bone and GI and ↓ phosphate reabsorption from kidneys → ↑ phos urine excretion  
In chronic renal failure the calcium will remain ↓ low and ↑ phos because kidney cannot excrete |
| N | Neoplasm (squamous cell lung, head and neck, breast, renal, multiple myeloma, leukemia) |
| S | Sarcoid (other granulomatous disorders = TB & Wegener’s; fungal = Histoplasmosis, Coccidiomycosis)  
Overproduction of vitamin D by macrophages and ↑ extrarenal alpha1-hydroxylase activity (enzyme, which converts Vit D to active form) |
| T | Thiazides calcium carbonate and lithium → alters PTH set-point for inhibition of hormone secretion by circulating Ca |
| R | Rhabdomyolysis |
| A | Aids |
| P | ↑ PTH (parathyroid adenoma 80%)  
Paget disease |

Note: HYPER Ca\(^{2+}\) is associated with HYPOkalemia (33% of patients)

90% of cases of Hypercalcemia = malignancy and primary hyper-parathyroidism

**PAGET DISEASE**

- Disorder of bone remodeling → excessive bone resorption (osteoclastic activity) followed by a compensatory ↑ bone formation (osteoblastic activity) → structurally disorganized mosaic of bone (woven bone), which is weaker, larger, less compact, more vascular and more susceptible to fracture than normal adult lamellar bone
- Etiology is unknown
- ↑ Ca\(^{2+}\)
- ↑ bone vascularity may → high-output CHF; increased likelihood bleeding complications following surgery
- Most common neurological problem is hearing loss → compression of CN VIII
- Vertebral involvement may lead to nerve-root compressions and cauda equina syndrome
SIGNS AND SYMPTOMS OF HYPERCALCEMIA

Mnemonic - Stones, Bones, Psychic Moans, Abdominal groans

<table>
<thead>
<tr>
<th>Stones</th>
<th>renal calculi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bones</td>
<td>osteolysis = bone pain</td>
</tr>
<tr>
<td>Psychic moans</td>
<td>mental status change, seizures, apathy, stupor, coma</td>
</tr>
<tr>
<td>Abdominal groans</td>
<td>N/V/anorexia, constipation, PUD, pancreatitis</td>
</tr>
</tbody>
</table>

TREATMENT OF HYPERCALCEMIA

- Initial and most important = rehydration followed by forced diuresis (0.9% NS + Lasix) →↑ GFR and therefore, excretion in urine. Lasix inhibits resorption of Ca in ascending tubule

- Glucocorticoids – hydrocortisone 200mg/day → inhibits activation of Vitamin D → inhibits bone resorption and GI absorption of calcium

- Plicamycin (mithramycin) → inhibits RNA synthesis in osteoclasts

- Bisphosphates act by inhibiting osteoclastic bone resorption and ↓ viability of osteoclasts (slows the release of calcium from bones to lower serum calcium)
  - Pamidronate (Aredia) 60 to 90 mg over 2 hours
  - Zometa (Zoledronic Acid - ZA) 4 mg IV over 15 minutes; considered agent of choice for malignancy-associated hypercalcemia because it is more potent and effective then Pamidronate

- Calcitonin 4 international units (IU)/kg SC or IM every 12 hours; reduces serum Ca by increasing renal Ca excretion and by decreasing bone resorption via interference with osteoclastic function.

- Dialysis

- Correction of underlying condition

HYPOCALCEMIA CAUSES

Renal failure, ↓PTH, ↓Mg, massive transfusions, shock or sepsis, pancreatitis, rhabdomyolysis, Vit D deficiency, alcoholism and drugs

Drugs = cimetidine, phenytoin, phenobarbital, gent, tobra, heparin, protamine, theophylline, nipride, phosphate enemas/laxatives, cisplatin, norepi, loop diuretics, steroids and mag sulfate

HYPOCALCEMIA SIGNS / SYMPTOMS

Depends on serum level and rapidity of decline

Chvostek and Trousseau signs, tetany, seizures, psychosis, ↓ BP, CHF and prolonged QT

Most symptoms = neuromuscular → progressive neuromuscular hyperexcitability [10, 5th ed., pg 1733]

Chvostek sign: a twitch at the corner of the mouth when the examiner taps over the facial nerve (CN VII), just in front of the ear [12, 6th ed., pg. 175]
**Trousseau sign:** more reliable indicator of ↓Ca = BP cuff maintains a pressure above systolic for 3 minutes → positive if carpal spasm produced \(^{[12, \text{6th ed., pg. 175}]}\)

**HYPOCALCEMIA TREATMENT**

- Ca Chloride = 360 mg elemental / 14 meq calcium
- Ca Gluconate 90 mg elemental / 4 meq calcium
- 1 amp = 10 cc of 10% solution (either CaCl or Ca Gluconate)
- The ionized calcium will increase for only 1 to 2 hours → follow by repeated doses or an infusion at a rate of 0.5 to 2 mg/kg/hr \(^{[10, \text{5th ed., pg 1733}]}\)

**ELECTROLYTE PEALS**

- **Categories of Hyponatremia**
  - Hypervolemic (CHF, cirrhosis, nephrotic syndrome)
  - Euvolemic (SIADH, psychogenic polydipsia)
  - Hypovolemic (Addison’s, renal-GI-third space losses)
  - Pseudohyponatremia (hyperlipidemia, hyperproteinemia)
  - Redistributive (hyperglycemia) – water drawn from cell dilutes Na+

- Correct acute hyponatremia = 1 to 2 mEq/L/hr
  Correct chronic hyponatremia = 0.5 mEq/L/hr

- Do not increase serum sodium more than 10 meq/L during the first 24 hours

- Required dose of hypertonic saline to correct Hyponatremia =
  (desired Na+ – measured Na+) x 0.6 (weight in kg) = mEq Na+ administered

- In patients who require emergency therapy = Hypertonic Saline 100 mL bolus given over 10 minutes; should raise the serum sodium by 2 meq/L. If seizure persist or worsen repeat bolus one or two more times at 10 minute intervals

- Overaggressive correction of hyponatremia = Central Pontine Myelinolysis (CPM) → destruction of myelin in Pons → CN palsies, quadriplegia or coma. More likely to occur in patients with chronic hyponatremia \(^{[10, \text{5th ed., pg 1726}]}\)

- Most likely EKG findings in severe hypo-Mg = PVCs and ventricular dysrhythmias (VT, torsades)
  Other EKG findings = AFIB, MAT, PSVT, ↑QT \(^{[10, \text{5th ed., pg 1738}]}\)

- Mg is essential cofactor for the Na⁺-K⁺-ATPase pump
  - Refractory hypo ↓K⁺ if ↓Magnesium is not corrected along with hypokalemia correction
  - ↓Magnesium → worsen digoxin toxicity induced dysrhythmias

- Most common cause of hyperphosphatemia and hypermagnesemia = renal failure

- 50% of alcoholics = ↓Phos

- Prominent muscle weakness = ↓K⁺ (paralysis may occur with serum levels < 2.0 mEq/L); also ↓Phos

- Decrease in the serum K⁺ of 1.0 mEqL = 370 mEq deficit of total potassium (in the absence of acute shifts caused by acid-base disturbances) \(^{[10, \text{5th ed., pg 1727}]}\)

- Pathophysiology of Hypokalemia from vomiting =
  Alkalosis = K⁺→ into cells in exchange for H⁺
  Volume loss → hypovolemia → aldosterone secretion → preserve Na⁺ & Bicarb in exchange for K⁺
ELECTROLYTE PEARLS

- Most important blood protein buffer = hemoglobin
- ↑ or ↓ in pH of 0.10 causes a ↓ or ↑ (opposite change) in PaO2 of about 10% [10, 5th ed., pg 37-38]
  - PaO2 = partial pressure of oxygen dissolved in blood
  - ↑ acidosis → Hgb gives up O2 more readily → ↑ PaO2 for a particular oxy-Hgb saturation → rightward shift of oxygen-hemoglobin dissociation curve
- pH < 7.35 = Acidosis  pH > 7.45 = Alkalosis
- Normal values:  pH 7.35 to 7.45  PaCO2 35 to 45
- PaCO2 and pH move in the same direction = Respiratory process
  - PaCO2 and pH move in the opposite direction = Metabolic process
- Acute: for every change of 10 in PaCO2 → the pH moves in the opposite direction (↓ or ↑) by 0.08 + 0.02
- Chronic: for every change of 10 in PaCO2 → the pH moves in the opposite direction (↓ or ↑) by 0.03
- Anion Gap = Na – [HCO3 + Cl]
  - Normal range = 5 to 12
  - Most common acid-base disorder in seizing patient = respiratory acidosis

CAUSES OF ANION GAP ACIDOSIS

Mnemonic: (CAT MUDPILES) [ANK]

| C | CO, CN (inhibit cytochrome oxidase a-a3 → ↑ lactate) |
| A | Alcoholic ketoacidosis |
| T | Toluene (secondary) to acidic metabolites |
| M | Methanol, Metformin |
| U | Uremia |
| D | DKA |
| P | Paraldehyde |
| I | INH (Isoniazid, inhibits lactate ↔ pyruvate, therefore → ↑ lactate) Iron (hypovolemia and anemia → tissue hypoperfusion → ↑ lactate) |
| L | Lactic acidosis |
| E | Ethylene glycol |
| S | Salicylates |

CAUSES OF NON-ANION GAP ACIDOSIS

Mnemonic: (HARD CUP) [ANK]

| H | Hyperalimentation |
| A | Acetazolamide (Diamox) |
| R | RTA (proximal) |
| D | Diarrhea |
| C | Cholestyramine |
| U | Uterosigmoidostomy |
| P | Pancreatic fistulas |
ADRENAL INSUFFICIENCY (AI)

- Most common cause of Adrenal insufficiency = autoimmune
- Most common infectious cause of Adrenal insufficiency worldwide = TB
- Most common infectious cause of Adrenal insufficiency in US = HIV
- ↓Na+ more common about 90%
- ↑K+ (65%) aldosterone production failure (see hyperkalemia mnemonic)
- ↑Ca2+ is seen in 6 to 33%
- ↓Glucose is present in 66% - significant cause of morbidity and mortality
- ↓BP
- Azotemia from hypovolemia
- ↑hematocrit from hypovolemia; lymphocytosis and eosinophilia (rare)
- Weakness, weight loss, abdominal pain \([12, 6th ed. pgs. 1315-1318]\)

Oral mucous membrane hyperpigmentation is pathognomonic primary AI (Addison’s Disease) \(\rightarrow\) result of compensatory adrenocorticotropic hormone (ACTH) and melanocyte-stimulating hormone (MSH) secretion

Hyperpigmentation is not present in secondary AI (adrenal insufficiency from pituitary infarction or hypothalamic insufficiency)

Treatment in confirmed cases of Adrenal insufficiency = Hydrocortisone 100 mg IV bolus

Treatment in non-confirmed /suspected cases of AI = Dexamthasone \([10, 5th ed. pgs. 1782]\)

Dexamthasone will not affect the serum cortisol level \(\rightarrow\) not interfere with the diagnosis of AI using the cosyntropin stimulation test

Measure cortisol levels \(\rightarrow\) administer cosyntropin (Cortrosyn), a synthetic form of ACTH, \(\rightarrow\) measure serum cortisol levels at 60 min \(\rightarrow\) AI excluded if basal or post-stimulation level > 550 nmol/L
NORMAL ADRENAL PHYSIOLOGY

1. Stress → corticotropin-releasing hormone (CRH) (hypothalamus) → Adrenocorticotropic hormone (ACTH) (anterior pituitary) → stimulates the adrenal cortex (zona fasciculata, middle layer of adrenal cortex) → ↑ cortisol

   ACTH is produced from a large precursor protein → in the process, other hormones are generated → ↑ MSH

2. ↓ renal blood flow → renin secretion → angiotensin II production → aldosterone secretion from adrenal cortex (zona glomerulosa /outer layer of adrenal cortex) → ↑ reabsorption of Na+ / water and ↑ secretion of K+ → ↑ BP

   Aldosterone is also produced to a lesser extent by ACTH

   ACTH deficiency doesn’t cause mineralocorticoid deficiency, but ACTH excess does cause mineralocorticoid excess

3. Then inner most layer of adrenal cortex = zona reticularis → androgens (DHEA)

4. Adrenal Medulla = core of adrenal gland – catecholamines (epi and norepinephrine)

TREATMENT OF THYROID STORM

1) Block peripheral effects of thyroid hormone
   a. Propranolol 1 to 2 mg IV q 5 min prn
   b. Guanethidine (inhibits NE release from post-ganglionic adrenergic nerve endings)
   c. Reserpine (depletes stored catecholamines both centrally and peripherally → inhibits release)

2) Inhibit hormone synthesis
   a. Propylthiouracil (PTU) 600 to 1,000 mg po, followed by 250 mg po q 4-6h
   b. Methimazole

3) Block hormone release
   a. Lugol solution or potassium iodide (SSKI) (give one hour after PTU)
   b. Lithium carbonate – difficult to titrate and toxic effects common

4) Prevent peripheral conversion of T4 → T3
   a. Propranolol
   b. PTU
   c. Glucocorticoids (may also be useful in preventing relative adrenal insufficiency due to hyperthyroidism)
     - Hydrocortisone 100mg IV q 8 hours

5) Provide general support: airway, monitor, fluids, cooling blanket, Tylenol (avoid ASA → ↑ Free T4) [170, 5th ed. pgs. 1772-1774]

Give beta-blocker first = blocking peripheral adrenergic hyperactivity of thyroid crisis may be the most important factor in reducing mortality and morbidity. If asthma, COPD or CHF substitute for B1 selective medication = esmolol; guanethidine or reserpine = alternatives.
Avoid ASA (see above) and Amiodarone - iodine-rich antidysrhythmic with poorly-defined effects on thyroid function that has been associated with both hyperthyroidism and hypothyroidism

**NORMAL PHYSIOLOGY**

Thyrotropin-Releasing Hormone (TRH) (hypothalamus) → Thyroid Stimulating Hormone (TSH) or Thyrotropin (anterior pituitary gland) → thyroid gland → capture iodine from blood to synthesize, store & release thyroxine (T4)

Most common cause of hyperthyroidism in US = Graves' disease 80%

**MYXEDEMA COMA**

Remember - HYPO's

- Hyponatremia - common
- Hypoglycemia
- Hypoventilation → respiratory acidosis
- Hypothermia - virtually all cases; Correlates with survival (worst if < 90 degrees F)
- ↓ HR (Bradycardia)
- ↓ Hct (Normochromic, normocytic anemia)
- ↑ Cholesterol > 250 mg/dL
- ↑ Transaminases, CPK, LDH
- Pericarditis → cardiac tamponade
- Mental status changes, lethargy, seizures
- Erythema nodosum, dry coarse hair, alopecia (lateral 1/3 of eyebrow), hoarse voice, bilateral carpal tunnel syndrome

**Myxedema Diagnosis**↓ T4 and ↑TSH

- Most common precipitating factor = infection; others = cold exposure, CVA, CHF, drugs, anesthetics, GI bleed, metabolic disturbances, trauma, surgery
- Amiodarone has been associated with both hyperthyroidism and hypothyroidism

**Treatment**
Levothyroxine IV + hydrocortisone 300 mg IV followed by 100 mg IV q 6 to 8 hrs

**DIABETES INSIPIDUS (DI)**

Lose large amounts of dilute urine because of the loss of concentrating ability of distal nephron

Central = lack of ADH secretion from Posterior Pituitary
Nephrogenic = lack of responsiveness to circulating ADH

**Causes** [50, 5th ed, pgs. 1727]

- Central = idiopathic, infection, tumor, bleed, granulomatous disorders, head trauma
- Nephrogenic = Obstructive uropathy, PKD, renal dysplasia, congenital disorders
- Systemic with renal involvement = Sickle cell, sarcoid, amyloid
- Drugs = Lithium, amphotericin, phenytoin, aminoglycosides
Lab
- ↓ Urine specific gravity
- ↓ Urine osmolality
- ↑ Na+

Treatment of Central DI = parenteral or IN vasopressin

NORMAL PHYSIOLOGY

↑ Plasma osmolarity
↓ BP
↑ Angiotensin II
↓
→ Arginine Vasopressin (AVP) or antidiuretic hormone (ADH) hormone formed in the hypothalamus → transported via axons to, and released from posterior pituitary → collecting ducts of kidney → reabsorption of water back into the circulation (↑ blood volume)
Vasopressin also → vasoconstrictor (↑ MAP)

ANTERIOR PITUITARY HORMONE RELEASE
Mnemonic: (FLAT PEG) [Ann]

| F | Follicle Stimulating Hormone (FSH) |
| L | Luteinising Hormone (LH) |
| A | Adrenocorticotropic Hormone (ACTH) |
| T | Thyroid Stimulating Hormone (TSH) |
| P | Prolactin (PRL) |
| E | Endorphin |
| G | Growth hormone (GH) |

VITAMIN DEFICIENCIES

Thiamine (B1) deficiency
- Alcoholics
- Anorexia, malaise, skin anesthesia, palpitations, calf tenderness, leg heaviness
- Beriberi = edema, JVD, CHF, ↑HR, ↑BP, ↓UO
- Polyneuritis
- Wernicke-Korsakoff Syndrome

Riboflavin (B2) deficiency
- Sore mouth/tongue, stomatitis, glossitis, purple swollen tongue (seen in other B deficiencies)
- Photophobia, loss of VA, corneal ulcers
- Seborrheic dermatitis
Niacin (B3) deficiency - constituent of NAD+ and NADP+
- Pellagra = 3 D’s
  - Diarrhea
  - Dermatitis
  - Dementia
- Other: sore tongue, tremors, muscle weakness, anorexia indigestion

Pyridoxine (B6) deficiency
- Peripheral neuritis

Cobalamin (B12) deficiency
- Megaloblastic and pernicious anemias
- Glossitis, hypospermia, GI disorders

Ascorbic Acid (C) deficiency
- Cofactor in collagen synthesis
- Scurvy; swollen/inflamed gums, loosening of teeth, follicular hyperkeratosis
- Impaired wound healing

Vitamin A deficiency
- Night blindness
- Loss of mucous membrane integrity →↑host susceptibility → infection

Vitamin E deficiency
- Peripheral neuropathy, anemia

ENDOCRINE PEARLS
- Most common cause of coma in patient with diabetes = hypoglycemia
- Primary reason for mental status changes in DKA = elevated osmolarity
- Urine dipstick for ketones uses a nitroprusside reaction which measures = acetoacetate ... not beta-hydroxybutyrate; usual ratio in DKA 1:3 acetoacetate / beta-hydroxybutyrate (may be as high as 1:30), therefore urine dip stick does not reflect true level of ketosis
- Average adult fluid deficit in DKA = 5 to 10 liters
- Correct Na+ in DKA = add 1.6 for every 100 mg/dl over the norm
- Diagnostic Features of Hyperosmolar Hyperglycemic State (HHS) per American Diabetic Association
  - Plasma glucose > 600 mg/dL
  - Serum osmolarity > 320 mOsm/kg
  - Serum pH > 7.30
  - Bicarbonate > 15 mEq/L
  - Small ketonuria and absent-to-low ketonemia
  - Profound dehydration up to an average of 9L
  - Some alteration in consciousness
• Most common precipitating factor for Hyperosmolar Hyperglycemic State (HHS) = infection (UTI and pneumonia most common; others = uremia, viral illness, ACS, drugs, metabolic and iatrogenic

• HHS → Start fluid resuscitation first with isotonic saline (0.9% NaCl)
  ○ Insulin can precipitate vascular collapse if given prior to volume expansion [10, 5th ed., pg 1756]

• Initial treatment of alcoholic ketoacidosis = intravenous D5NS; also, correct K+, add thiamine [10, 5th ed., pg 2524]

• Most common cause of Hyperparathyroidism = Adenoma 80%; hyperplasia 15-20%, carcinoma < 1%
  ↑PTH → ↑Ca

• The most common presentation of primary hyperparathyroidism = Asymptomatic hypercalcemia

• Pheochromocytoma = catecholamine secreting tumors, usually adrenal; 5 P’s = Pressure (BP), palpitations, perspiration, pallor and pain (chest pain, abdominal pain or headache)
  Definitive treatment = surgery
HYPOTHERMIA, HYPERTERMIA

- Chilblains (Pernio) = inflammatory erythematous to violaceous acral lesions after exposure to cold; pruritic and/or painful; tx = rewarming, gently bandage, elevate; consider Nifedipine (Procardia)[10, 5th ed., pg. 1974]
- Hypothermia = temperature of less than 95°F (35°C)
- Hypothermic patient – move the patient as little as possible – irritable myocardium → VF
- The amplitude of the J-wave (Osborne wave) is proportional to the degree of hypothermia; does not relate to pH and is not prognostic; may appear temp < 32°C (89.6°F)[10, 5th ed., pg. 1981]
- Hypothermia – sequence cardiac deterioration: sinus brady→AF (< 32°C) →VF→Asytole (< 25°C / 77°F)
- Cerebral metabolism ↓ decreases 6% for each 1°C ↓ decline in temperature
- Severe Hypothermia (< 32.2°C, CV instability) → active rewarming → immersion in bath maintained at 40°C (104°F); more practical in ED = Bair Hugger → heat transfer via convection [10, 5th ed., pg. 1991]

Active core rewarming = peritoneal, bladder and pleural lavage with fluids heated to 45°C (113°F). From my experience I recommend pleural irrigation by placing two thoracostomy tubes (36 to 40 French) in both hemithoraces

Lactated Ringer solution is not recommended because hypothermic liver cannot metabolize lactate [http://emedicine.medscape.com/article/770542-treatment]

- DKA/hypothermia → insulin is not effective at core temp < 30°C (86°F)
- Defibrillation/hypothermia → rarely effective < 30°C (86°F)
- “Core-temperature afterdrop” = further ↓ in core temperature & clinical deterioration after rewarming. Peripheral tissues are warmed → vasodilation → sudden return of cooler, acidic, hyperkalemic blood from the extremities → central circulation → core-temperature afterdrop → dysrhythmogenic [10, 5th ed., pg. 1975]

- Most common presenting symptom of frostbite = numbness 75%[10, 5th ed., pg. 1973]
- Heat cramps = related to Na+ (salt) deficiency; lab = ↓Na+, ↓Cl, ↓UNa+ and ↓UCl levels; salt your beer!
- Tylenol/ASA not recommended in heat stroke, may be deleterious; tx = evaporative cooling [12, 6th ed., pg. 1188]
**SPIDERS, SNAKES, BEES, SCORPION, SEA CREATURES**

- **Black Widow bites** = 60% erythematous macule; other findings: target lesions, tiny dual fang marks
  
  Pathognomonic bite = patch of sweat and a little red dot
  
  [Dr. Rangan, ACEP News, Vol. 30, No. 2, Feb 2011]

- **Black Widow Pathophysiology**
  
  Neurotoxin → ↑ Ach and NE release → SLUDGE BAM + HTN (severe abdominal pain). Tx = supportive, observe 4 hours → if no symptoms discharge home; if HTN and tachycardia do not respond to supportive measures → Equine derived Antivenin available

- **Brown recluse spider** = venom causes local necrotic skin lesion surrounded by an erythematous ring

- **Tarantula** = irritating venomous barbed abdominal hairs, which can be ejected several feet like a javelin → allergic reactions; more painful than damaging (only serious if hairs get into eyes)

- **Most common poisonous snake in the US** = Rattlesnake

  **Rattlesnake** snakebite = admit patients to the hospital for 24 to 48 hours → serial determinations of platelets, prothrombin time, and urinalysis to check for myoglobin and hemoglobin

  Treatment = Crotalidae Polyvalent Immune Fab (Ovine) (CroFab; FabAV) antivenin - indicated even if envenomation is minimal or mild


- **Coral Snakes** admit patients for 24 to 48 hours for observation (delayed signs and symptoms may occur)

  Red on yellow, kill a fellow

  Red on black, venom lack

- **Anyone** bitten by Eastern Coral Snake should be given antivenin IV → 3 to 5 vials in 500 cc NS

- **Most consistent symptom after pit viper bites** = immediate burning pain; petechiae may occur, as well as anaphylaxis if there is an immune response; severe edema, however no compartment syndrome

- **Most dangerous venom from hymenoptera family** = honey bee → venom causes greater histamine release per gram than any other hymenopteran venom [10, 5th ed., pg 793]

- **After stinging a victim honey bees release a pheromone that attracts other bees**

- **Box Scorpion** = do not treat with narcotic and barbiturates → ↑ toxic effects of venom; Antivenin available

- **Jellyfish /man-o-war treatment** = wash off with salt water; 5% acetic acid (vinegar) neutralizes nematocytes

- **Sting ray, starfish, sea urchin, sea cucumbers lion fish treatment** = immersion in hot water [12 6th ed., pg. 1210]

- **Hemorrhagic bullous lesions with history of sea water-contaminated abrasions or eating raw seafood** = consider *Vibrio vulnificus*

  Treatment = Cipro 750 mg po bid [Sanford, 2009, pg. 51]
**ELECTRICAL, RADIATION, HAPE /HACE**

- Low voltage alternating current (AC) → ventricular fibrillation; High voltage AC → Asystole
- Direct current (DC) → Asystole
- Most common arrest arrhythmia after electrical injury = ventricular fibrillation
- Lightning strike = high voltage DC depolarization; current pathway = “flashover”, not horizontal (hand to hand) or vertical (hand to foot) seen with low or high-voltage AC\(^{[12, \text{6th ed., pg 1236}]}\)
- “Lichtenberg figures” = superficial ferning pattern = pathognomonic for lightning strike\(^{[12, \text{6th ed., pg 1238}]}\)
- Lightning strike = bilateral cataracts and TM perforation common; arrest rhythm = asystole
- Immediate Cause of Death\(^{[12, \text{6th ed., pg 1236}]}\)
  - Low voltage alternating current (AC) → ventricular fibrillation
  - High voltage AC → apnea
  - Lightning → apnea

- Absolute lymphocyte count 24 hours after radiation exposure is a good indicator of patients clinical course → > 1,200 → no lethal dose. If lymphocyte count 300-1,200 at 48hrs → lethal dose of radiation expected
- LD50 from exposure to ionizing radiation = 4.5 Gy (450 rad); at 10 Gy (1,000 rad) = 100% MR
- Most sensitive physical finding in HACE = cerebellar ataxia; treatment = descent, O2, dexamethasone
- Most common fatal manifestation of severe high-altitude illness = HAPE; treatment = rest, oxygen, descent, Nifedipine (Procardia, Adalat) 10 to 20 mg po or SL q 6 hours
  New study = Dexamethasone and tadalafil (cialis) both decrease systolic pulmonary artery pressure and may reduce the incidence of HAPE in adults with a history of HAPE.\(^{\text{Respir Physiol Neurobiol. Dec 15 2011;179(2-3):294-9}}\)
- Acetazolamide (Diamox) and dexamethasone have been shown to be effective agents for prophylaxis (not in the treatment) of HAPE
DERMATOLOGY

DERMATOLOGY PEARLS

- **Nikolsky’s sign** = Pemphigus vulgaris (autoimmune), TEN and Staph. scalded skin syndrome (the outer epidermis separates easily from the basal layer on exertion of firm sliding manual pressure)

- **Hutchinson’s sign** - with HZ infection V1 distribution = vesicle at the tip of the nose

- **Erythema multiforme** = target lesions; 70% mucosal involvement; palms/sole involvement
  Steroids provide symptomatic relief, but unproven benefit in duration and outcome
  Causes: drugs, bugs, immunizations, malignancy (leukemia), idiopathic
  Most common causes: *Mycoplasma*, HSV 1, *Strep. pyogenes* as well as and sulfa, phenytoin and Penicillin drugs

- Most common cause of **impetigo** = *Staph. aureus*; Group A Strep a distant second. Without treatment impetigo heals in 3 to 6 weeks [10, 5th ed., pg. 1639]

- **Tinea versicolor** = yeast infection, *Pityrosporum ovale* or *P. orbiculare* are synonyms for Malassezia furfur; variety of colors (tan, pink white), hypopigmented scaly macules or patches on chest or back. Usually seek medical attention because spots do not tan; lesions resolve in 1-2 months without permanent scar
  Recurrence is common [10, 5th ed. pg. 1637]
  Treatment = oral Ketoconazole (Nizoral) 400mg x1 or 200mg q24 hr x 7days or 2% cream 1x q24 hr x 2 weeks; rule-out erythrasma [Sanford, 2009, pg. 104]

- **Erythrasma** = chronic superficial infection intertriginous areas of skin; *Corynebacterium minutissimum*
  well-demarcated brown-red macular patches; treatment = Erythromycin 250mg q 6hr x 14 d [Sanford, 2009, pg. 51]

- **Porphyria cutanea tarda** = erosions and bullae to sun-exposed areas exposed to trauma

- **Wood’s light** = organism → fluorescent pattern [12, 5th ed., pg. 1572]
  *Porphyria cutanea tarda* → urine color change to orange or yellow
  Erythrasma → coral red or pink
  *Tinea versicolor* → green or yellow
  *Pseudomonas* → yellow or green

- **Pityriasis rosea** = pink or pigmented papules or plaques 1 to 2cm, forming a “Christmas tree-like” distribution on trunk. Usually children and young adults; asymptomatic or pruritis; Eruption is preceded by a week by the appearance of a “herald patch” (2 to 6 cm plaque); self-limiting, resolves in 8 to 12 weeks without treatment; etiology unclear, possibly viral [10, 5th ed., pg. 1638]

- **Poison Ivy** = allergen not in bullae of vesicles; so after washing of the involved site, contact with rash does not cause it to spread; Treatment = antihistamines, oatmeal baths and topical steroids; if widespread oral steroids 30 to 80 mg/day tapered over 21 days [10, 5th ed., pg. 1648] Bentoquatam (IvyBlock) [12, 6th ed. pg. 1249]
• Steroids in Zoster = treat if within 3 days of onset of rash; ↓ discomfort during acute phase of zoster; does not ↓ incidence of post-herpetic neuralgia [Sanford, 2009, pg 144] or lessen rate of the healing of lesions [10, 5th ed. pg. 1656]

21 day steroid taper = 30mg bid (days 1-7), 15mg bid (days 8-14) 7.5mg bid (days 15-21) [Sanford, 2009, pg 144]

• Erythema nodosum causes = Ulcerative colitis, Yersinia enterocolitica, Srep., Chlamydia, TB, sarcoid, histoplasmosis, coccidiomycosis, hypothyroidism, pregnancy, idiopathic and drugs

• Most common drug-induced cause of erythema nodosum = oral contraceptives
**SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)**

More Than 2 Criteria Must be Met

1) Temperature  
   a. > 38°C (100.4°F) or  
   b. < 36°C (96.8°F)

2) Heart Rate > 90 beats/min

3) Respiratory Rate (respiratory alkalosis is often the first sign of SIRS)  
   a. > 20 breaths/min or  
   b. PaCO2 < 32 mmHg

4) WBC =  
   a. > 12,000 or  
   b. < 4,000 cells or  
   c. > 10% bands

Sepsis = SIRS + suspected or documented source of infection

- Severe Sepsis = Sepsis + infection induced organ dysfunction or hypoperfusion
- Organ dysfunction /hypoperfusion = oliguria; lactic acidosis

Septic Shock = Severe Sepsis + refractory ↓ BP despite adequate fluid resuscitation

Sepsis articles  
[Crit Care Med. 2008 Jan;36(1):296-327]  
[Crit Care Med. 2004 Mar;32(3):858-73]

- Most common source of infection in septic patient = respiratory system  
- Overwhelming Post-Splenectomy Infection (OPSI) = septic shock, DIC and adrenal hemorrhage; may be post surgery or inadequate splenic function (sickle cell)

**HIV/AIDS**

- Risk of seroconversion after needlestick from HIV + patient = 0.3%
- Risk of HIV from blood transfusion = 1/600,000 to 2,000,000
- Primary HIV occurs 2 to 4 weeks after exposure (Acute Retroviral Syndrome); presentation = mono-like syndrome; Fever, fatigue, sore throat, lymphadenopathy (cervical most common), weight loss, myalgias, HA, N/V/D/, and maculopapular erythematous rash (most commonly on trunk) and leukopenia
• Most common opportunistic infection in AIDS patients = Pneumocystis jiroveci (carinii) (PCP)
  ▫ Classic presentation = fever, non-productive cough and DOE which → dyspnea at rest \cite{12, 6th ed., pg. 930}
  ▫ Treatment = Bactrim; if sulfa allergic = clindamycin + primaquine or pentamidine \cite{Sanford, 2009, pg. 41}
  ▫ Steroids if PaO2 < 70 mmHg; steroids 15-30min before meds; reduce resp. failure and death \cite{Sanford, 2009, pg. 41,128}

• Most common cause of serious opportunistic viral infection in HIV patient = CMV

• Most common systemic opportunistic infection in AIDS patient = Mycobacterium avium complex (MAC)
  ▫ Diagnosis AFB in stool or other body fluids
  ▫ Treatment = Rifabutin + Clarithromycin + Ethambutol \cite{10, 5th ed., pg. 1845}

• Most common cause of pneumonia in HIV / AIDS patients = still Strep. pneumo

LYME DISEASE

• Borrelia burgdorferi = spirochete \cite{12, 6th ed., pgs. 970-972}
• Vector = Ixodes dammini / Ixodes scapularis – deer tick
• Zoonotic reservoirs = white-tail deer and white footed mouse
• Most prevalent in Northeast

3 Stages of Lyme Disease Infection

<table>
<thead>
<tr>
<th>Stage 1</th>
<th>Erythema Chronicum Migrans (ECM) large annular rash with central clearing 2 to 20 days after tick bite</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>Disseminated phase = 3 days to 6 months after tick bite; fever, adenopathy arthralgias, 50% get multiple annular lesions = most characteristic component of the 2nd stage of illness Carditis 8% = 1st degree AVB, Wenkebach, CHB CNS = unilateral or bilateral CN palsies (most common neuro symptom); peripheral neuropathy</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Late phase = years after infection = chronic arthritis (knee most common), myocarditis, polyneuropathy or leukencephalopathy</td>
</tr>
</tbody>
</table>

BABESIOSIS

• Babesia microti (NE US) and Babesia gibsonii (NW US) and B. divergens (Europe) = protozoan
• Vector = Ixodes dammini – deer tick (same vector as Lyme disease)
• Zoonotic reservoirs = deer, rodents and domesticated mammals (cattle, horses, dogs and cats)
• Malaria-like disease; protozoan similar in structure and life-cycle to plasmodia
• Blood transfusions have been implicated in transmission of babesiosis
• 20% have concurrent Lyme disease
• Viral syndrome presentation with ↑ spiking fevers; hepatosplenomegaly; emotional lability
• ↑thrombocytopenia, ↑leukopenia, ↑LFTs, renal failure (dark urine)
• Hemolytic anemia = ↑IBIL, ↑reticulocyte count, ↑LDH, ↓haptoglobin
• Diagnosis = intra-erythrocytic parasite on Giemsa stained peripheral blood smear
  ▫ (“Maltese Cross” formation)
• Treatment = Clindamycin + Quinine or Atovaquone + Zithro \cite{12, 6th ed., pg 973} and \cite{10, 5th ed., pg 1869}
RELAPSING FEVER

- *Borrelia recurrentis* = spirochete \[12, 6th ed., pg 1256\]
- Vector = lice (singular: louse) or ticks
- Zoonotic reservoirs = humans and wild rodents \[12, 6th ed, pg 973\]
- Viral syndrome presentation
- ↑LFTs, ↓thrombocytopenia, ↓BP; severe cases → meningoencephalitis, DIC, liver failure, myocarditis
- Relapsing fevers
- Diagnosis = thick smear (similar to Malaria)
- Treatment = Tetracycline 200 mg po or Erythromycin 1gm po x one dose

EHRlichOSIS

- *Ehrlichia chaffeensis*; *G – Neg coccobacilli*
- Vector = Ixodes
- Zoonotic reservoirs = deer, dogs and other mammals
- Infects circulating leukocytes → fever, viral like syndrome, maculopapular rash 20%
- ↓Leukopenia ↓thrombocytopenia, ↑LFTs; rarely encephalitis and renal failure
- Treatment = Doxy 100mg po bid x 7 to 14 days \[12, 6th ed., pg 973\]

Q-FEVER

- *Coxiella burnetii*; intracellular, small G Neg
- Vector = tick; more common route of infection = inhalation of organisms from air that contains airborne barnyard dust contaminated by dried placental material, birth fluids, and excreta of infected herd animals
- Zoonotic reservoirs = livestock (cattle, sheep, goats) or cats
- 50% infected with C. burnetii show signs of clinical illness. ↑ Temp (104-105° F), severe HA, myalgias, non-productive cough, N/V/D, abdominal pain, and chest pain. 30-50% of symptomatic infection will develop pneumonia.
- Atypical pneumonia, meningitis, endocarditis, granulomatous hepatitis
- Treatment = Doxy, Azithro or Quinolone

ROCKY MOUNTAIN SPOTTED FEVER

- *Rickettsia rickettsii*; intracellular, Gram Neg
- Vector = Dermacentor tick
- Zoonotic reservoirs = deer, horses, cattle, cats, dogs or rodents
- Triad = fever, HA and rash (begins wrists/ankles spread → trunk/face)
- Treatment = Doxy or Chloramphenicol if allergic

TULaremIA

- *Francisella tularensis*, small, Gram Neg, aerobic, rod
- Forms of *F. tularensis* infection recognized in humans include: Ulceroglandular (80%, chancer- like ulcer - with raised margins), Glandular, Oculoglandular, Oropharyngeal, Typhoidal/Septicemic and Pneumonic
- Transmission:
  - Direct penetration of the skin (hair follicles, or cuts/abrasions or contaminated by exposure of an infected animal)
**Indirectly from bites of deerflies, ticks or mosquitoes (bacterium not isolated in saliva; scratch after bite → introduce infected feces)**

- Exposure of mucous membranes with blood or tissue of infected animals
- (rabbits, squirrels, foxes, skunks, mice, rats)
- Ingestion of contaminated food or water

**Inhalation**

- Regardless of presenting form of tularemia systemic symptoms of **fever with relative bradycardia in 42%**, chills and rigors, myalgias (often prominent in low back), weakness, malaise and headache
- Treatment: Streptomycin; alternative = Gentamicin

**TRAVEL CHEMOPROPHYLAXIS**

- [http://www.cdc.gov/travel](http://www.cdc.gov/travel)
- Travel Clinic = Robert Citronberg, MD. Traveler’s Health and Immunization Center
- Telephone: (847) 663-9500

**DENGUE FEVER**

- Arbovirus; Most common serious febrile tropical disease after malaria
- Mosquito transmission = *Aedes aegypti* (day biting mosquito)
- Incubation period = 4 to 7 days

- Asymptomatic or viral syndrome presentation with rash:
- Acute onset of severe HA, myalgias and arthralgias ("Break-Bone Fever")
- Facial flushing, conjunctival injection, retro-orbital pain and facial edema ("Dengue Facies")
- Rash = macular or maculopapular on trunk spreads → extremities and face

- Note: West Nile virus (transmitted by *Culex* mosquito) = lymphadenopathy → **absent** in Dengue

**DENGUE HEMORRHAGIC FEVER (DHF)**

- A small percentage of previously infected patients develop → DHF
- Begins as classic Dengue Fever followed by:
  - Hemorrhagic pleural effusions, purpura, petechiae, bleeding diathesis
- Diagnosis = ELISA IgM; lab = ↓leukopenia, ↓thrombocytopenia, false ↑Hct, ↑LFTs; ↓Na (most common electrolyte abnormality); ↑PT/PTT, ↓fibrinogen and ↑fibrin degradation products
- Supportive care; Treat fever with Tylenol not NSAIDs due anticoagulant properties, [12, 6th ed., pg 1253]

**DENGUE SHOCK SYNDROME**

- Circulatory failure (↓BP, Altered mental status, ↑HR, Altered MS, cool/clammy, narrow pulse pressure with ↑ peripheral vascular resistance

**HANTAVIRUSES**

- Inhale material contaminated with mouse urine/feces → hemorrhagic fever + renal failure or
- Syndrome of severe respiratory failure and shock
MALARIA

Most deadly vector-borne disease in the world

- *Plasmodium vivax, ovale, malariae* and *falciparum* = protozoa
- Mosquito transmission = Female Anopheles (bite at dusk and dawn)
- Also direct transmission → blood transfusion & mother → fetus
- Viral syndrome presentation: Paroxysm shivering and chills → followed by ↑fever → when ↓ fever → patient diaphoretic /exhausted → paroxysms of malaria (correspond to length of asexual erythrocytic cycles; merozoites invade RBC's → cells lyse → new merozoites further invade uninfected RBCs); less common symptoms = N/V/D/HA and jaundice
- *P. falciparum* = most deadly form of malaria; complications = cerebral malaria, ↓hypoglycemia (parasites metabolize glucose from RBCs; especially children), metabolic acidosis, severe ↓anemia, renal failure, pulmonary edema, DIC and death
- Blackwater fever = dark urine secondary to RBC hemolysis from high parasitemia

Malaria Diagnosis

- Giemsa or Wright’s - stained thin and thick blood smears

Malaria Treatment

- *Plasmodium vivax, ovale, malariae* = Chloroquine + Primaquine (covers exoerythrocytic parasites)
- Chloroquine-sensitive *P. falciparum* → treatment as above
- Chloroquine-resistant *P. falciparum* →
  - Quinine + Doxy (clinda if contraindication, or < 8y/o) +/− Primethamine-sulfadoxine (Fansidar) or
  - Mefloquine +/- Doxy or
  - Atovaquone-proguanil (Malarone)

Do not use Primaquine if glucose-6-phosphate dehydrogenase deficiency → hemolysis of RBCs

- Quinine given po or IV; if rapid infusion → ↓ profound hypoglycemia
- Other side effects = ↓BP and cardiac dysrhythmias

LEPTOSPIROSIS

World’s most widespread zoonotic infection; common in tropical climates

- *Leptospira interrogans* = spirochete
- Fresh water contaminated by bovine, pig, canine or rat urine; 2 to 20 days incubation
- Leptospires multiply in the small blood vessel endothelium
- Two syndromes: anicteric (which is self-limiting) 90% of cases and icteric leptospirosis (Weil’s disease) which is more severe form characterized by multi-organ failure

- ↑leukocytosis with atypical lymphs, ↓thrombocytopenia
- CXR = bilateral interstitial infiltrates in dependent areas
- Death from CV collapse; Mortality rate 6%, if respiratory syndrome mortality much higher
Two distinct phases of illness observed in the mild anicteric form → septicemic (acute) phase and the immune (delayed) phase. In icteric leptospirosis, the 2 phases of illness are often continuous and indistinguishable.

Viral syndrome presentation with severe †HA, petechial rash which may involve the palate, conjunctival injection, myalgias (calf, low back) and **fever with relative bradycardia**

→ Symptoms resolve after 4 to 7 days, followed by → asymptomatic period or progress directly→more severe disease

Aseptic meningitis, hepatitis/liver failure, nephritis/RF, uveitis, rash (jaundice and purpura), TTP, HUS, DIC, pneumonia/consolidation due to alveolar hemorrhage, acalculous cholecystitis, pancreatitis, myocarditis → CHF, Afib and rarely CV collapse

→ May last up to 4 weeks

Mortality rate in icteric leptospirosis (Weil’s disease) = 5 to 40%

Diagnosis = isolate leptospires from blood or CSF

Oral Doxycycline or Amoxicillin if treated within first 3 days

Penicillin or Ampicillin IV for severe cases

**LEISHMANIASIS**

Leishmania = intracellular protozoan

Transmission = Lutzomyia or Phlebotomus → sandflies

(Rural Africa, Asia, Mediterranean basin, Central/South America, Brazil, India and Sudan

† Leishmaniasis in returning U.S. military personnel and their dependents from the Middle East, especially from Iraq (mainly cutaneous)

Clinical Syndromes

1) Cutaneous = most common
2) Mucocutaneous = chronic and relentless disease complicated by secondary infections and pneumonia
3) Diffuse Cutaneous = chronic, difficult to treat, few resulting deaths
4) Visceral (Kala-azar or Black fever) = most fatal form caused by Leishmania donovani

Darkening of skin is characteristic = Kala-azar or black fever; lymphadenopathy

“Kala-azar” comes from India → Hindi for black fever

Infiltration of the hematopoietic system → pancytopenia

† mortality due to secondary infections (pneumonia, TB, dysentery); hemorrhage or severe anemia

Pentad = fever, weight loss, † hepatosplenomegaly, ↓ pancytopenia and hypergammaglobulinemia

Diagnosis = aspirate bone marrow, spleen, lymph nodes or punch biopsy from ulcer edge

Stained smears = Leishman-Donovan bodies = stained amastigotes in macrophages

Treatment of cutaneous leishmaniasis where the potential for mucosal spread is low, topical Paromycin

Treatment = **Pentavalent Antimonial compounds**; injection only; call CDC for this one

If failure/resistance, use Amphotericin
**HOT TUB FOLLICULITIS, FRESH / SEA WATER CELLULITIS, LUDWIG’S ANGINA, NEMATODES**

- Hot tub folliculits = **Pseudomonas folliculitis** = generally self-limited; no antibiotics \[\text{Sanford, 2009, pg. 52}\]

- Cellulitis after wound in fresh water lake = consider Aeromonas hydrophilia
- Treatment = Quinolone, alternate = Bactrim \[\text{Sanford, 2009, pg. 63}\]

- Ludwig’s angina = cellulitis of the soft tissues of the neck and floor of the mouth; life-threatening complication = airway obstruction. Broad-spectrum antibiotics cover gram-positive, gram-negative, and anaerobic organisms, ENT consult, CT

- **Nematodes** Treat all 3 with Albendazole or Mebendazole \[\text{Sanford, 2009, pg. 52}\]
  1) **Necator americanus** (hookworm) major cause of anemia worldwide; eosinophilia may be absent
  2) **Enterobius vermicularis** (pinworm) most common parasite infection in the US → anal pruritis
  3) **Ascaris lumbricoides** (round worm) most common nematode infection in the world →
     - Asymptomatic, bowel perforation, cholecystitis, intestinal obstruction, malabsorption, pneumonitis and pancreatitis
     - Mortality rate = 5% if complications occur

- **Cystercercosis** = **Taenia solium** (pork tapeworm)
  - New onset seizures
  - CT = calcified lesions
  - Treatment = Mebendazole for acute infection
WMD PEARLS - DERM

CUTANEOUS ANTHRAX

Painless, depressed, black necrotic eschar (anthrax = Greek for “coal”)

BUBONIC PLAGUE

- Regional lymph node infection (femoral most common > inguinal > axillary > cervical)
- Erythematous, warm, very painful, tender, swollen lymph nodes (buboes) with considerable surrounding edema
- Buboes usually nonfluctuant and rarely, suppurate
- Acral gangrene (“black death”) = Septicemic plague
- Common Characteristic Features of VHFs & Plague = Petechiae, purpura, ecchymosis, DIC
- Hemoptysis is present in both VHFs and Plague, however other pulmonary findings are uncommon with VHFs

ULCEROGLANDULAR TULAREMIA

- Erythematous, tender papule at the inoculation site → becomes pustular → ulcerates within days
- → The local ulcer is raised with sharply demarcated margins with depressed center (chancriform)

BLISTER AGENTS

- Lewisite
- Nitrogen and sulfur mustards
- Phosgene oxime

- Most sensitive are warm, moist thin areas → perineum, genitalia, axilla, neck & antecubital fossa (thicker skin of hands may be spared)
- On the skin mustard causes no immediate pain sensation (delayed symptoms, hours after exposure)
- Lewisite and phosgene oxime cause immediate pain
- Lewisite antidote = Dimercaprol (British Anti-Lewisite - BAL)

TRICHOSTROTHECENE MYCOTOXINS (“YELLOW RAIN”)

- Protein synthesis inhibitors and inhibit mitochondrial respiration and cause bone marrow suppression; 400x more potent than mustard in producing skin injury/↑blisters
- Charcoal binds mycotoxins
SMALLPOX

- Rash begins on face, and upper extremities and all lesions are synchronous, umbilicated, deeply embedded in dermis, painful, involve palm and soles
- Patients are infectious from the time the rash first appears until all scabs fall off
- Smallpox vaccine can lessen the severity/prevent illness if given within 3 days of exposure

WMD PEARLS - PULMONARY

INHALATIONAL ANTHRAX

- Non-specific flu-like illness with non-productive cough; rhinorrhea uncommon
- Patient may improve before acute deterioration within 24 to 48 hours → diaphoresis, dyspnea, stridor, cyanosis, hemorrhagic mediastinitis, septic shock, death; 50% hemorrhagic meningitis
- CXR
  - Wide mediastinum, hemorrhagic pleural effusions. Results from U.S. anthrax attacks 2001 from first 10 patients = 7 had infiltrates, multilobar in 3 patients
- Treatment
  - Cipro + 1 or 2 additional antimicrobials: Vancomycin, rifampin, PCN, ampicillin, imipenem, clarithromycin, clindamycin or chloramphenicol
  - IV treatment initially before switching to oral antimicrobial therapy
  - Continue oral and IV treatment for 60 days
  - Consider steroids: meningitis, cutaneous or mediastinal edema

PNEUMONIC PLAGUE

- Productive cough → Sputum: bloody, watery or less commonly purulent; F /CP / Dyspnea
- CXR = bilateral infiltrates or lobar consolidation; any pattern possible, including ARDS
- Within 24 hours without treatment → fulminant pneumonia associated with hemoptysis, septic shock, DIC, respiratory failure, circulatory collapse and death; 6-10% get meningitis
- Treatment
  - Preferred choices = Streptomycin or Gentamicin
  - Alternative choices = Doxycycline or ciprofloxacin or chloramphenicol
  - Treatment for 10 days

PULMONARY TULAREMIA

- Initial picture of systemic illness without prominent signs of respiratory disease: abrupt onset of high fever with relative bradycardia in 42%, chills, rigors, malaise, sore throat, headache and pleuritic CP, myalgias (often prominent in low back) and non-productive cough
- CXR
  - Earliest radiographic finding = peribronchial infiltrates
  - Advancing to bronchopneumonia in one or more lobes
  - Hilar lymphadenopathy and effusions are common
- Treatment - Preferred Choices [JAMA June 6, 2001-Vol 285, No. 21:2763-2773]
  - Streptomycin – adult: 1 gm IM twice daily; Peds: 15mg/kg IM or IV daily
  - Gentamicin – adult: 5 mg/kg IM or IV daily; Peds. 2.5 mg/kg IM or IV three times daily
  - Treatment for 10 days
WMD PEARLS

- Inhalational anthrax, pneumonic plague, Q-fever, Ebola = abdominal symptoms
- Inhalational anthrax, bubonic and septicemic plague, pulmonary tularemia, Q-fever pneumonia: Person to person transmission = has not been reported
- Person to person transmission = Pneumonic plague, viral hemorrhagic fevers (not yellow-fever), and smallpox
- Decontamination → bleach effective: anthrax, plague, tularemia, Q-fever, cholera, ricin, VHFs, nerve agents, blister agents
- Hypochlorite solution does not inactivate Trichothecene Mycotoxins

BOTULISM

- 7 types of botulism neurotoxins known as types A-G
- Exotoxin (botulinus toxin) → descending symmetrical paralysis (GBS: ascending paralysis)
- Pathogenesis
  - Inhale / digest → circulation → peripheral nerve synapses → blocks the release of acetylcholine → descending symmetrical paralysis → ptosis, generalized weakness, dizziness, dry mouth, diplopia, blurred vision, dysphonia, dysarthria, dysphagia and respiratory failure
- Treatment = equine antitoxin

CHOLERA

- Enterotoxin →↑ cAMP → secretion of water and chloride ions → massive “rice-water” stool; fluid losses may exceed 5 to 10 liters per day
- Treatment
  - Fluid and electrolyte replacement
  - Ciprofloxacin 1.0 gm po x once OR
  - Doxycycline 300 mg po x once
  - For children and in pregnancy: erythromycin or trimethoprim-sulfamethoxazole

RICIN

- Inhibits protein synthesis
- Charcoal binds GI exposure of Ricin

BLOOD AGENT

Cyanide

- Is a tissue toxin - the military incorrectly categorizes with blood agent
- Binds cytochrome oxidase and disrupts oxidative phosphorylation →↑ anaerobic metabolism
- HA/N/V/confusion/combativeness/seizures and coma
- Reddish lips (blue in dark skinned patient)
- Odor = almonds
- Signs: Initially ↑ HR and BP followed by → ↓ HR and BP and profound metabolic acidosis
- → Respiratory, CNS and myocardial depression (bradycardia → asystole) within minutes of significant exposure
• **Treatment**
  - Na-Nitrite → converts RBC hemoglobin (Hgb) → to Methemoglobin
  - Methemoglobin → combines with cyanide to form → cyanometh-Hgb
  - Cyanometh-Hgb and free cyanide are detoxified by sulfur transferase (rhodanese) → thiocyanate
  - Thiocyanate is → eliminated in urine

  • Rhodanese function ↑s with the availability of sulfur donor
  • Na-Thiosulfate is a sulfur-containing compound
  • Na-Thiosulfate + cyanometh-Hgb, via rhodanese enzyme → Na-thiocyanate
  • Na-thiocyanate → eliminated in urine
  • If simultaneous carbon monoxide and cyanide poisoning Na-Thiosulfate should be used ALONE
  • Adjunctive therapy: sodium bicarbonate to correct metabolic acidosis & benzodiazepines for szs

**NERVE AGENTS**

- Sarin, Soman, Tabun, GF & VX
- Inhibitors of acetylcholinesterase → cholinergic excess → SLUDGE BAM Syndrome
- Treatment
  - Atropine: large amounts, (10-20 mg), may be needed over 24 hours
  - Pridaloxime chloride (2-PAM, Protopam) reverses the cholinergic nicotinic effects

**CHOKING AGENTS**

- Phosgene and Chlorine = non-cardiogenic-pulmonary edema
- Delayed symptoms with phosgene; immediate symptoms are noted with chlorine
- Nebulized 3.75% sodium bicarbonate symptomatic improvement to treat chlorine exposures

**RSI - PREMEDICATION**

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSE</th>
<th>PURPOSE</th>
<th>SIDE EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Premedication</strong></td>
<td>LOAD = Lidocaine, Opioid analgesic, Atropine, Defasciculating agents</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lidocaine</strong></td>
<td>1.5 mg/kg</td>
<td>Suppresses cough reflex. May blunt ICP response to intubation</td>
<td>HYPOTENSION</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Seizure</td>
</tr>
<tr>
<td><strong>Fentanyl (Sublimaze)</strong></td>
<td>3-5 μg/kg</td>
<td>Seldom causes hypotension. Analgesia 70x morphine. Give slowly → over 1&quot; because if fast → muscle rigidity</td>
<td>Apnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Seizure</td>
</tr>
<tr>
<td><strong>Atropine</strong></td>
<td>0.02 mg/kg</td>
<td>↓ salivation / bradycardia; min dose 0.1mg</td>
<td>Tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Brady from (a) laryngoscope stimulating laryngopharynx parasympathetic receptors (b) Sux → direct muscarinic receptor stim</td>
<td></td>
</tr>
<tr>
<td><strong>Vecuronium (Norcuron)</strong></td>
<td>0.01 mg/kg</td>
<td>Pre-paralytic; dose to block fasciculations due to succinylcholine; give 3&quot; before sux</td>
<td>Tachycardia</td>
</tr>
</tbody>
</table>
## RSI - SEDATION

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSE</th>
<th>PURPOSE</th>
<th>SIDE EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etomidate</td>
<td>0.3 mg/kg</td>
<td>&lt; 1 min, durn 5 min, ↓ ICP &amp; IOP</td>
<td>N/V, myoclonic excitation</td>
</tr>
<tr>
<td>Thiopental (Pentothal)</td>
<td>3-5 mg/kg</td>
<td>Rapid sedn. 30 sec, durn 10 min. ↓ ICP; No analgesia</td>
<td>HYPOTENSION Apnea, asthma</td>
</tr>
<tr>
<td>Methohexital (Brevital)</td>
<td>1 mg/kg</td>
<td>Onset &lt; 1 min, durn 5-10min, No analgesia</td>
<td>HYPOTENSION apnea, hiccup, laryngospasm</td>
</tr>
<tr>
<td>Midazolam (Versed)</td>
<td>0.1 mg/kg IV or IM</td>
<td>Rapid sedation &lt; 2 min, duration &lt; 30 min No analgesia; + amnesia and muscle relaxation</td>
<td>HYPOTENSION, Apnea ↓ dose if COPD or age &gt; 60</td>
</tr>
<tr>
<td>Ketamine (Ketalar)</td>
<td>1-2 mg/kg over 1 min</td>
<td>Status asthmaticus (Bronchodilation) Beneficial in hypotension; Amnesia Analgesia. Onset 1 min, durn 10 min.</td>
<td>HTN, ↑ HR, ↑ ICP, ↑ IOP, emergence reactions ↑ secretions</td>
</tr>
<tr>
<td>Fentanyl (Sublimaze)</td>
<td>5 μg/kg</td>
<td>Useful in hypotensive patient. Analgesia. Onset 1 min, durn 30 -60 min.</td>
<td>Apnea; Seizure muscle rigidity if given fast</td>
</tr>
</tbody>
</table>

## RSI - PARALYSIS

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSE</th>
<th>PURPOSE</th>
<th>SIDE EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine (Anectine)</td>
<td>1.5 mg/kg 2.0 mg/kg kids</td>
<td>Depolarizing Rapid onset &lt; 1” Short duration &lt; 10” Give IM if no IV access</td>
<td>↓ HR, ↓ BP, Fasciculations ↑ IOP, V+, ↑ intragastric P, ↑ ICP, ↑K+, myoglobinuria,</td>
</tr>
<tr>
<td>Vecuronium (Norcuron)</td>
<td>0.1 mg/kg</td>
<td>Non-depolarizing agent Onset 1 to 3 minutes Duration 20 to 60 minutes</td>
<td>Long duration</td>
</tr>
<tr>
<td>Pancuronium (Pavulon)</td>
<td>0.1 mg/kg</td>
<td>Non-depolarizing agent Onset 2 minutes Duration 45 minutes</td>
<td>↑ HR, HTN, No CAD</td>
</tr>
<tr>
<td>Cisatracurium (Nimbex)</td>
<td>0.4 mg/kg</td>
<td>Non-depolarizing Onset 2 minutes Duration 90 to 120 minutes</td>
<td>Minimal CV effects</td>
</tr>
<tr>
<td>Rocuronium (Zemuron)</td>
<td>1.0 mg/kg</td>
<td>Non-depolarizing Onset 1 minute Duration 20 to 60 minutes</td>
<td>Bronchospasm, ↑ HR, Dysrhythmias</td>
</tr>
</tbody>
</table>
10 P’S OF RAPID SEQUENCE INTUBATION (RSI)

P 1. Perform H & P (MAPLE), consider indications-risks-alternatives
P 2. Preparation: personal-drugs-equipment
P 3. Pulse oximetry-monitor-automated BP device
P 4. Preoxygenate (5 minutes)
P 5. Premedicate: Atropine, Norcuron, Lidocaine or Fentanyl (wait 3 minutes)
P 6. Pressure (cricoid) and continue to assist ventilation with BVM
P 7. Prime (sedation): Etomidate, Thiopental, Versed, Ketamine or Fentanyl
P 8. Paralyze: Succinylcholine, Norcuron or Zemuron
P 9. Placement of ETT
P 10. Post-intubation; verify tube placement and assure adequate sedation for prolonged paralysis

ENDOTRACHEAL TUBE (ETT) SIZE

- # = the inner diameter in mm [12, pg. 40]
- ETT size adult males = 8.0-8.5 advance tube to 23cm (from carina to corner of mouth) [12]
- ETT size adult females = 7.5-8.0 advance tube to 21 cm (from carina to corner of mouth) [12]
- After intubation and NGT get PCXR, ETT should be 2 cm above the carina; check end-tidal CO2

PRE-INTUBATION ASSESSMENT FOR DIFFICULT AIRWAY
Mnemonic: (LEMON)

<table>
<thead>
<tr>
<th>L</th>
<th>Look externally</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>Evaluate (3:3:2 rule) = 3 fingers between incisors; mandible length 3 fingers from tip of chin to Hyoid bone and distance of the hyoid to the thyroid – 2 fingers distance</td>
</tr>
<tr>
<td>M</td>
<td>Mallampati classification (I-IV); I fully visible tonsils; IV only hard palate visible</td>
</tr>
<tr>
<td>O</td>
<td>Obstruction</td>
</tr>
<tr>
<td>N</td>
<td>Neck</td>
</tr>
</tbody>
</table>
POST-INTUBATION PROBLEMS

Mnemonic: (DOPE)

| D | Dislodged ETT |
| O | Obstructed ETT |
| P | Pneumothorax |
| E | Equipment failure |

KETAMINE PEARLS

- Do NOT give Ketamine: < 6 months, weight < 5 kg, active pulmonary infection, PMH of cardiac-thyroid-psych or porphyria.


- In conscious sedation may give 4-5 mg/kg IM onset 5 minutes, duration 30 minutes
- Consider atropine (decreases secretions), however not routinely recommended anymore [Acad Emerg Med. 2008 Apr;15(4):314-318]
- Consider versed, IV or IM = 0.1 mg/kg, PR 0.75 mg/kg onset 15” durn 45” - drug of choice for emergence reaction

PEDIATRIC AIRWAY PEARLS

ETT size can be estimated:

a) 16 + (age in years) / 4
b) Age / 4 + 4
c) < 2 y/o = size of patients little finger (fifth digit)
d) Broslow Tape

- Position of the ET tube at the lips (in cm’s) should = 3 x size of ETT
- ETT x 2 = NG and Urinary Catheter size

PEDIATRIC TUBE SIZES

<table>
<thead>
<tr>
<th>Tube Type</th>
<th>Neonate</th>
<th>6 months</th>
<th>1 to 2 years</th>
<th>5 years</th>
<th>8 to 10 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest tube</td>
<td>12-18</td>
<td>14-20</td>
<td>14-24</td>
<td>20-32</td>
<td>28-38</td>
</tr>
<tr>
<td>NGT</td>
<td>5-8</td>
<td>8</td>
<td>10</td>
<td>10-12</td>
<td>14-18</td>
</tr>
<tr>
<td>Urinary Catheter</td>
<td>5-8 (feeding)</td>
<td>8</td>
<td>10</td>
<td>10-12</td>
<td>12</td>
</tr>
</tbody>
</table>
### PEDIATRIC AVERAGE WEIGHTS AND ENDOTRACHEAL TUBE SIZES

<table>
<thead>
<tr>
<th>Age</th>
<th>Weight range (kg)</th>
<th>Endotracheal Tube Sizes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature</td>
<td>1.0-2.5</td>
<td>2.5-3.0</td>
</tr>
<tr>
<td>Newborn– 3 months</td>
<td>2.5-6.0</td>
<td>3.0-3.5</td>
</tr>
<tr>
<td>4-18 months</td>
<td>6-12</td>
<td>4.0-4.5</td>
</tr>
<tr>
<td>1.5-3 years</td>
<td>12-15</td>
<td>4.0-4.5</td>
</tr>
<tr>
<td>3-5 years</td>
<td>15-20</td>
<td>4.5-5.0</td>
</tr>
<tr>
<td>5-7 years</td>
<td>20-25</td>
<td>5.5-6.0</td>
</tr>
<tr>
<td>8-10 years</td>
<td>25-35</td>
<td>6.0 cuffed</td>
</tr>
<tr>
<td>11-12 years</td>
<td>35-40</td>
<td>7.0 cuffed</td>
</tr>
</tbody>
</table>

[Reference for above chart: “Tube Sizes” and “Average Weights – Endotracheal Tube Sizes” = Larry B. Mellick, MD]

Weight in kilograms = (Age x 2) + 8  
Upper limit of SBP = (Age x 2) + 80

Newborn = 3 kg  
1 y/o = 10 kg  
5 y/o = 20 kg  
10 y/o = 30 kg

### DRUG INFUSIONS

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSAGE RANGE</th>
<th>INFUSION RATE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DOPAMINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conc: 100mg/5cc</td>
<td>2-30 mcg/kg/minute</td>
<td>1 cc/hr = 10 mcg/minute</td>
</tr>
<tr>
<td>60 mg/100 cc D5W</td>
<td>Starting dose: 3 mcg/kg/minute</td>
<td></td>
</tr>
<tr>
<td><strong>DOBUTAMINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conc: 250 mg/20 cc</td>
<td>2-30 mcg/kg/minute</td>
<td>1 cc/hr = 10 mcg/minute</td>
</tr>
<tr>
<td>60 mg/100 cc D5W</td>
<td>Starting dose: 3 mcg/kg/minute</td>
<td></td>
</tr>
<tr>
<td><strong>PROSTAGLANDIN</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conc: 0.5 mg/cc</td>
<td>0.05-0.2 mcg/kg/minute</td>
<td>1 cc/hr = 0.1 mcg/minute</td>
</tr>
<tr>
<td>Premix: 0.5 mg/83 cc D5W</td>
<td>Starting dose: 0.05 mcg/kg/minute</td>
<td></td>
</tr>
<tr>
<td><strong>ISUPREL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conc: 1 mg/5cc</td>
<td>0.05-1.0 mcg/kg/minute</td>
<td>1 cc/hr = 0.2 mcg/minute</td>
</tr>
<tr>
<td>3 mg/250 cc D5W</td>
<td>(add 1 mg to premix)</td>
<td></td>
</tr>
<tr>
<td><strong>EPINEPHRINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dilution: 1:10,000</td>
<td>0.05-1.0 mcg/kg/minute</td>
<td>1 cc/hr = 0.2 mcg/minute</td>
</tr>
<tr>
<td>Conc: 1 mg/10 cc</td>
<td>Starting dose: 0.05 mcg/kg/minute</td>
<td></td>
</tr>
<tr>
<td>Premix: 3mg/250 cc D5W</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>NOREPINEPHRINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conc: 1 mg/cc</td>
<td>0.05-1.0 mcg/kg/minute</td>
<td>1 cc/hr = 0.2 mcg/minute</td>
</tr>
<tr>
<td>Premix: 3 mg/250 cc D5W</td>
<td>Starting dose: 0.05 mcg/kg/minute</td>
<td></td>
</tr>
</tbody>
</table>
**LIDOCAINE**  
Conc: 100 mg/5 cc  
Premix: 2 gm/250 cc D5W  
Starting dose: 20-50 mcg/kg/minute  
IV Rate: Run at 0.25-0.6 cc/kg/hr = 20-50 mcg/kg/minute  
1.5 cc/hr = 200 mcg/minute

**NIPRIDE**  
Conc: 50 mg/5 cc  
Premix: 50 mg/250 cc D5W  
Starting Dose: 0.5 mcg/kg/minute  
1.5 cc/hr = 5 mcg/minute

**NITROGLYCERINE**  
Premix: 50 mg/250 cc  
Starting dose: 0.5 mcg/kg/minute  
1.5 cc/hr = 5 mcg/minute

**CA CHLORIDE**  
Premix: 10 gm/100 cc  
Starting dose: 10-20 mg/kg/hr  
1 cc/hr = 100 mg/hour

---

**Anaphylactic shock: Give Epinephrine IV over 10 minutes**

Epi 1:1,000 ---> take 0.1 cc = 100 μg (0.1mg) ---> dilute in 10 cc NS = 100,000 concentration  
Or  
Epi off crash cart = 1:10,000 (1mg) ---> take 1cc = 100 μg (0.1mg) ---> dilute in 10cc of NS to get 1:100,000 concentration ---> give slowly over 10 minutes

**LOCAL ANESTHESIA PEARLS**

- 2 “i’s” in generic denotes an amide  
  Examples: Lidocaine, Bupivacaine, Prilocaine, Mepivacaine and Etidocaine

- If allergic to amides (see above) and esthers, for example procaine (novocaine) → consider benadryl → dose = 1cc (50mg/ml) diluted in 9cc of NS

- Maximum dose of lidocaine of infiltration →  
  *Without* epinephrine = 5 mg/kg  
  *With* epinephrine = 7 mg/kg

- Avoid TAC (tetracaine, adrenaline, cocaine) or LET (lidocaine, epinephrine and tetracaine) on mucosal membranes, pinna of the ear, nose, penis, fingers and toes \[^{10, 5th ed. pg., 2574}\]

- XAP = lidocaine, adrenaline (epinephrine) and pontocaine (tetracaine)  
- LET = lidocaine, epinephrine and tetracaine

- Bupivacaine is not recommended for use in children < 12 y/o \[^{10, 5th ed. pg., 2573}\]

- Consider 6-0 fast-absorbing catgut sutures with facial lacerations [Dr. Robert Rifenburg]
THE #20 - AN IMPORTANT NUMBER

- Normal IOP < 20 mm Hg
- Normal intracranial pressure (ICP) < 20
- Compartment syndrome suspected if pressures > 20 mm Hg
- If pressure > 30 mmHg → ischemia and pain
- Peak methanol levels > 20 mg/dL = Indication for dialysis\(^{[12, 6th ed., pg. 1069,1070]}\)
- Ethylene glycol levels > 20 mg/dL = Indication for dialysis\(^{[12, 6th ed., pg. 1069,1070]}\)

POSTEROLATERAL - AN IMPORTANT WORD

- Most common site of diaphragmatic injury = Posterolateral
- Most common site herniated disks rupture = Posterolateral
- Almost all spinal hematomas = Posterolateral
- Most common site of esophageal tear in Boerhaave’s syndrome = left Posterolateral aspect (distal esophagus)
[ANK] Author is Not Known


[2] Dr. Antonio Carlino

[3] Dr. Karen Spurgash

[4] Dr. Tajudeen Ogbara

[5] Author = Dr. Eric Farinas, mnemonic provided by Dr. Nancy Bauer.


[13] ANK/Author is Not Known-mnemonic provided by Dr. John Carroll.

[14] Dr. Dane Nichols


[16] Dr. Thomas R. Scaggs


[18] Author = Dr. Gueyikian, mnemonic provided by Dr. Suzanne Ahn

[19] Dr. Nancy Bauer
[20] Dr. George Hevesy


[24] Dr. Robert Rifenburg


[26] Adult EM Reports

[27] Pediatric EM Reports


[29] 2010 AHA Handbook of Emergency Cardiovascular Care for Healthcare Providers