Emergency Medicine – 200 Level Review Packet, Justin Bosley MS3
(Evaluation of poison patients and expanded ultrasound review, Caroline Nelson, MS2)

Lectures covered:

- **Altered mental status**
- Wound management
- **ABx use in ED**
- **Dizziness**
- Pediatric respiratory illness
- **Headache**
- **Chest pain**
- Environmental emergencies
- **Pediatric common injuries**
- Evaluation of poison patients
- GYN emergencies
- **Shock**
- Emergency uses of ultrasound (covered separately at end with orientation lecture on ultrasound)
- **Fever in pediatrics**
- Ultrasound Stuff

** Don't forget – Abdominal Pain

Note: EKGs are important (according to the grapevine). Consider USMLE step 2 questions at http://www.learntheheart.com/

CHEST PAIN:

- Immediate Goals of Care in Patient w/ suspected MI = ABC/Stabilization/Resuscitation
    - IV, oxygen, monitors, ECG+/- CXR
- Acute Coronary Syndromes
    - Epidemiology
        - >6 million Americans w/ CAD
        - 500K deaths p.a. in U.S. from CAD
        - 250K pts. w/ pre-hospital CA à 6% survive to hospital discharge
        - >4 million ED visits p.a. for acute chest pain
        - $100B in care per year
    - Etiology = ischemia vs. fixed atherosclerotic lesion vs. evolving plaque/thrombus vs. spasm
    - Spectrum
        - Stable = transient, episodic chest discomfort, predictable, reproducible
        - Unstable = new in onset, occurs at rest
            - Acute MI (12.5% of acute MIs are clinically silent)
            - Substernal chest discomfort > 15', assoc. w/ dyspnea, diaphoresis, LH, palps, N/V
                - NSTEMI
                - STEMI
    - Risk Factors
        - Smoking, HTN, DM, Hypercholesterolemia, age
        - Family hx of CAD <55 y.o. in 1st degree relative, previous hx of CAD or PVD
        - Cardiac risk factors are poor predictors of risk for ACS in the ED
    - Physical Exam
        - Normal cardiopulmonary exam is most common
        - S3 in 15-20% of pts. w/ MI, chest wall TTP in ~15% w/ ACS
    - ECG: best test, but sensitivity is poor
        - ST elevation in 50% of AMIs

        - 1-5% of AMI's have normal initial ECG, 4-23% of patients w/ UA have normal ECG
    - Cardiac Markers:
        - CK-MB = >90% sensitive for MI 5-6h after symptom onset, but only 50% sensitive shortly after presentation, elevate @ 3-12 hours, peak @ 18-24 hours, duration 2 days
        - Troponin = Tn-I similar to CK-MB but duration is 5-10 days; TN-T is less sensitive, but is independent marker of CV risk
    - Treatment – "OH BATMAN!"
        - Oxygen, heparin, beta-blocker, aspirin, thrombolytic, morphine, anti-platelet agent, nitrates
        - Aspirin to inhibit thromboxane A2, decreasing PLT aggregation
        - Nitrates to decrease preload and afterload, increase coronary perfusion
        - Beta blocker to decrease infarct size, CV complications, and mortality
        - Consider heparin, direct thrombin inhibitors, fibrinolytics, PCI
            - Fibrinolytics if ST elevation > 0.1mV in 2+ continguous leads or new LBBB and time to therapy < 12 hours (class I), 12-24 hours (class IIb)
- Cocaine-Related Chest Pain
    - Epidemiology
        - 6% of patients w/ cocaine-associated chest pain have an AMI (often atypical chest pain)
        - 20-60% have transient myocardial ischemia
        - Can be delayed hours to days after most recent use
    - Etiology = spasm, inc. myocardial O2 demand, clot formation, accelerated atherosclerosis w/ LVH
    - Diagnosis = ECG is less sens/spec than for MI, CK-MB less sens, Tn-I may be more useful
    - Prognosis = favorable short-term, 1 year mortality due to comorbidities or cont. cocaine use
    - Treatment = benzodiazepines, avoid beta blockers
- Aortic dissection = intimal tear w/ entry of blood into media, dissecting btw. intima and adventitia
    - Epidemiology
        - Stanford A involves ascending aorta (80% of dissections), B involves descending only
        - Increased risk in pts. >50 y.o. w/ HTN, younger pts. w/ Marfan's, Ehler-Danlos, pregnancy
        - Mortality of A = 75% if untreated, 15-20% if sx, B = 32-36% w/ or w/out surgery
    - History
        - 90% w/ abrupt+severe pain in the chest (type A) or mid-back (type B), "tearing" or "ripping", can be dull or pressure-like, N/V and diaphoresis common
        - Involved areas and associated symptoms:
            - Carotid arteries: stroke
            - Spinal arteries: paraplegia
            - Abdominal aorta/renal arteries/iliacs: Abdominal/flank pain
            - Coronary arteries: aortic insufficiency; pericardial effusion/tamponade
            - Laryngeal nerve compression: hoarseness
            - Tracheal compression: dyspnea/stridor/wheezing
            - Esophageal compression: dysphagia
    - Diagnosis
        - Physical Exam w/ signs/symptoms as before + most commonly normal CV/pulm exam
            - AI murmur in only 16-20% of patients, w/ abnl. periph. pulses in only ~50%
        - CXR abn. in ~85%
            - Most often see widened mediastinum, L pleural effusion, indistinct aortic knob, displaced calcified intima > 6mm from outer aortic wall
        - CT vs. TEE vs. aortogram
    - Treatment
        - 2 large bore IVs, monitors, T&C, ECG
        - Drop BP to decrease the shear force on the intima, lower both ABP and LV contractility
            - Goal SBP 90-100 mmHg, HR 60-80 à use IV nitroprusside+esmolol or labetolol
- Pulmonary Embolus
    - Epidemiology
        - 650K cases/yr in the U.S., source is lower ext. DVT in 80-90% of cases
        - Mortality is 2-10% if dx/tx, but 30% if undx
    - Presentation = classic triad of dyspnea, hemoptysis, pleuritic CP seen in only 20% of pts
        - Pleuritic CP in 74%, dyspnea in 84%, RR > 16 in 92%, HR > 100 in only 44%
    - Dx

        - Wells Criteria à 3 pts. for suspected DVT or alt. dx. being less likely than PE, 1.5 for HR>100, recent immobilization, previous DVT/PE, 1 for hemoptysis, malignancy
            - 3-6 pts is 20.5% mean probability of PE; >6 points à 67% chance of PE
        - ECG often normal, w/ >40% showing non-specific ST+T wave abnl., see sinus tach most often, S1Q3T3 seen only in 6% of patients w/ PE
        - CXR nl in 30%, concerning in setting of dyspnea and hypoxemia w/o RAD
            - ATX in 50%, elevated hemidiaphragm in 40%, Hampton's Hump = pleural-based wedge-shaped infiltrate, Westermark sign = prox. Dilated pulmonary artery w/ abrupt cut-off
    - Treatment
        - IV, oxygen, monitors
        - If high pre-test probability, anticoagulate 1st then order study
            - Heparin 80 U/kg IV bolus, 18 U/kg/hr IV drip
- Spontaneous PTX
    - Epidemiology: often in tall, thin males, 10-20% occur w/ exertion, most though to result from rupture of subpleural bleb, symptoms vary w/ size+rate of ptx progression
    - Presentation
        - Acute pleuritic CP in 95%, dyspnea in 80%, decreased breath sounds in 85%, tachypnea >24 in only 5%, hyperressonance in <30%
    - Dx
        - Tension ptx à needs immediate decompression
        - Non-tension ptx à upright PA CXR is 83% sensitive
    - Tx
        - Tube thoracostomy using minicatheter or standard chest tube
        - Catheter aspiriation (single or sequential)
        - Observation x 6 hours w/ repeat CXR
- Esopahageal rupture is last of "Big Five" life-threatening cause of chest pain

DIZZINESS:

Assessment: key to the evaluation of dizziness is in the hx and PE; lab tests/imaging are of little benefit

Key: recognize that dizziness is a subjective complaint and the most important thing is to understand the patient's reference point. Meaning of this word will vary based on age, sex, and ethnic background. If a patient gives only vague responses then focus on a specific event. At least attempt to assess whether the symptoms are staying the same, worsening, or getting better.

Categorize the symptoms using the patient's own description of the dizziness, aiming to categorize the symptoms into 5 broad categories which can be supported with physical examination findings and more specific history taking: vertigo, pre-syncope/syncope, dysequilibrium, ill-defined lightheadedness, and true muscular weakness

1) Vertigo: perception of rotation or a spinning sensation which can be of themselves or of the room. Patients will often say, "it feels like I'm drunk" or "I'm sea sick." The key to vertigo is to figure out if it is peripheral or central and if it is associated with any hearing loss or tinnitus

a) Is it central or peripheral?

	CENTRAL	PERIPHERAL
Onset	slow	rapid

Severity	mild	worse
CN findings	+	-
Latency	-	+
Nylen-Barany*	nystagmus persists	nystagmus extinguishes
(* positive if nystagmus present, fast phase toward affected ear = ear closest to ground)		
Etiologies	brain stem ischemia	acoustic schwannoma
	posterior fossa tumors	Meniere' disease
	multiple sclerosis	labyrinthitis (infection)
	drugs: anticonvulsants,	benign positional vertigo
	PCP, ethanol	trauma (endolymphatic fistula)
		labyrinthine concussion

Peripheral = vertigo that recurs and abates every few hours suggests a peripheral cause, the more violent and severe the vertigo, the more likely it's peripheral

Central = vertigo that is gradual onset, constant and not affected by movement is characteristic of central vertigo. Brain stem or cerebellar symptoms including dysphasia, dysphonia, ataxia, diplopia, miosis or bilateral blurred vision are common. May be acute onset if acute ischemia is cause but will see cranial nerve findings

b) if (+) hearing loss = acute labyrinthitis: typically after URI's, otitis media

if (+) hearing loss and tinnitus – classic triad of Meniere's disease: often occurs in middle age, can recur and symptoms increase with each recurrence until peaks and slowly decrease in intensity, hearing loss typically persists between episodes

c) trauma-related vertigo: perilymphatic fistula results in leakage of endolymph from the round or oval window into the middle ear. These patients complain of acute worsening of dizziness when middle ear pressure increases during coughing, sneezing or straining. Nonspecific dizziness may be seen as part of post-concussive syndrome but the increase in symptoms with coughing is not seen. Labyrinthine concussion or post-traumatic positional vertigo

Physical Examination: key is a good neurologic examination particularly the cranial nerves, cerebellar function, nystagmus and positional testing. Nystagmus is seen in both peripheral and central causes of vertigo. Best observed in dark room because if the patient has something to fixate their vision on, any peripherally-induced nystagmus can be extinguished. That's why its helpful to "look at the horizon" (i.e., a stationary object) when you have "sea sickness." Peripheral nystagmus is rotatory or horizontal. Centrally-induced nystagmus can be vertical or dysconjugate.

Nylan-Barany maneuver (aka Dix-Hallpick)

Pt is sitting near top of gurney. Have them rapidly lay down and extend their neck 45 degrees below horizontal and 45 degrees to left. If this induces nystagmus, then the test is positive. Fast phase is towards affected ear (the ear closest to the ground is being tested)

2) PRESYNCOPE/SYNCOPE: These terms refer to the degree of symptoms with syncope actually defined by LOC. Symptoms may be better referred to as a near faint due to decreased cerebral blood flow. We have all experienced near syncope when we stand after crouching for a prolonged period, particularly if in the sun. Anything that altered the body's normal vascular reflexes to maintain central perfusion can cause this including drugs esp. antihypertensives and ethanol, hypovolemia, and rarely poor cardiac output secondary to a dysrhythmia or aortic stenosis.

This is the only time I'll ask a leading question to a dizzy patient: "Do you ever feel dizzy while sitting? How about while

standing? How about while lying down." If they only get dizzy with standing, I ask about postural changes or exertion.

Assess for any potential for volume loss: anorexia, nausea, vomiting, blood in stool, vaginal bleeding

If associated with a profound and new symptom may lead you to the diagnosis: acute headache – subarachnoid hemorrhage, acute abdominal pain – increased vagal tone from perforated bowel, ruptured AAA, or a ruptured ectopic pregnancy

Examination: orthostatic vital signs à there is no correlation between having symptoms of dizziness or lightheadedness upon standing and any measurable change in orthostatics. But perform thorough cardiovascular examination particularly for murmurs.

3) DYSEQUILIBRIUM: best classified as a gait disturbance; often described as an unsteadiness or stumbling or having difficulty getting around. Caused by loss of a significant sensory function such as sight, light touch, or proprioception. Often lose their dizziness when holding onto a stationary object like a wall or furniture.

Physical Examination: visual acuity, fundoscopic exam for cataracts, retinal disease; complete neurologic examination but focus on light touch, pin prick, proprioception in lower ext. Patients may have a wide based or stumbling gait but not ataxic – can test cerebellar function with finger-nose-finger and rapid alternating movements in upper extremities. Must be careful to do a good neurologic examination to evaluate for true weakness or ataxia.

Etiologies include poor vision, diabetic or ethanol induced neuropathy, B12 deficiency, and tabes dorsalis. Dysequilibrium is also symptom of motor gait disorders due to cerebellar degeneration or Parkinson's dz but the neuro exam will help differentiate.

4) ILL-DEFINED LIGHTHEADEDNESS: category of dizziness for patients without a specific etiology elicited from the H&P. Other terms these patients may use include "it feels like my head is full of air", "I just feel strange," or they may describe feeling of being distant from the environment or others, derealization or depersonalization respectively. If anyone describes their symptoms to you like this strongly suggests depression as an etiology (PSYCH). The etiology of this nonspecific feeling may be difficult to identify but hyperventilation, anxiety, depression, and medications should be very high on the list in otherwise healthy patients. However in patients who are slightly ill appearing or who have multiple medical problems, hyperCa, hyperMg, uremia, anemia, chronic subdural hematoma, and myocardial ischemia can all present with this nonspecific complaint.

Physical Examination: typically have a normal physical examination but pay attention to more subjective findings of flat affect and other clues to depression or stressors. Look for evidence of anemia with pale conjunctiva or under tongue (frenulum)

DIAGNOSTIC STUDIES: FSBG, ECG (unless patient has acute vertigo), lab levels of any quantifiable therapeutics the patient may be on (e.g., anticonvulsants, ethanol), CT/MRI (if symptoms are vertiginous but possibly central in etiology or if any localizing weakness is identified). The older the patient and the more nonspecific the symptoms, esp. in patients with dysequilibrium and lightheadedness, the greater number of tests should be done: CBC, lytes w/ Ca, Mg, Phos, renal function

MANAGEMENT:

Vertigo à treat any dehydration with isotonic fluids, antiemetics for any vomiting; if central, then probably admit (unless drug related); if peripheral, then treat etiology (to ENT if traumatic perilymphatic fistula, ABX if otitis media); or reassurance and symptomatic treatment

I tell patients (depending on their level of sophistication) that there is a problem or inflammation in the balance center in the inner ear and that is will take a few days to readjust. Tell patients to sit rather than lay down (this is because have more

stationary objects to fixate one), to move slowly, and no driving. Elevate head on a few pillows and take medications just prior to lying down to improve sleep.

Can use a wide range of medications and none have been demonstrated to be significantly better than another: meclizine (Antivert) 25-50 mg TID (antihistamine), scopolamine transdermal Q 3 days (anticholinergic), diazepam 5-10 mg BID (benzodiazepine), proclorpherazine 5-10 mg PO TID or 25 mg PR (antiemetic). These should only be used for 3 days because, while this blunts the symptoms they also slow the resetting of the vestibular system. Inform patients that recurrence over the next 3-4 days is expected but if it persists, recommend reevaluation

Dysequilibrium à improve home environment (not an easy task), glasses or ophthalmologic care, rehabilitation to train patients how to walk, NO MEDICATIONS! Remember that many of these can cause sedation and actually worsen these symptoms

FEVER IN PEDIATRICS: typically defined as temp > 38 C (100.5 F), core temp most accurate but rectal is most frequently used

- Background:
  - Fever is not disease, but a manifestation of disease process, host defenses release endogenous pyrogens (IL-1, IL-6) or can be 2/2 endogenous chemicals (e.g., cocaine, anti-cholinergics)
  - Hypothalamus regulates core temperature, infections alter this set point, and hypothalamic injury can cause erratic control
  - Hyperpyrexia = purposeful temp. elevation >= 41.5 C
  - Hyperthermia = uncontrolled temperature regulation
  - Treatment? Not necessary! (though potential role for preventing febrile seizures?) Elucidating/treating underlying cause is more important
    - Anti-pyretics make children more comfortable but do not cure fever!
  - Fever is very non-specific sign of infection and most cases are not life-threatening
  - Fever represents 20% of all pediatric ED visits w/ youngest children at highest risk of severe infection 2/2 impaired host defenses (neutrophil fxn, lymphocyte production, complement fxn), decreased pathogen clearance, and impaired ability to localize infection
- Common Febrile Infections:
  - Viral = URI (e.g., RSV, influenza), GI (e.g., rotavirus), enteroviruses
  - Bacterial = otitis media, strep pharyngitis, sinusitis, pneumonia
- Groups: infections and management differ significantly by age
  - 0-56 days = febrile infant
    - Risk of serious bacterial infections: bacteremia, meningitis, UTI, bacterial enteritis, skin/soft tissue infection, bone/joint infections
    - See fever, irritability, lethargy, poor feeding, resp. distress, N/V, or NO SYMPTOMS!
    - Organisms = E. coli, GBS, Listeria (< 30 days); HSV ½ may also produce severe infection if < 21 days old
    - Age 0-28 days: ABX + anti-virals started and all get septic work-up à CBC, UA, UCx, BCx, LP, CXR if respiratory symptoms
    - Age 29-56 days: Philadelphia Criteria
      - Needs reliable caregiver and ability to f/u at 24 hours in ED
      - Low-risk criteria: if met, can be f/u as outpatient and NO ABX!
        - PE: well-appearing and w/out focus of infection
        - Hx: normal past history and normal perinatal hx
        - Lab criteria:
          - WBC 5-15K and I/T ratio <0.2
          - UA < 10 WBC/hpf + GS(-)
          - CSF < 10 WBC/hpf + GS(-)
          - CXR w/out infiltrate
  - 2-36 months = febrile young child
    - Occult bacteremia (often Hib, S. pneumo)
      - Increased incidence w/ age <36 months and fever >= 39.0 C
      - Manage w/ WBC, ANC, blood culture and t/c empiric ABX tx
      - "In most of our minds, OB is a dead issue. For FWS-WELL APPEARING, NO SOURCE, do not need anything"

- Occult UTI (E. coli or GN enterics, enterococcus)
  - Fever is often only presenting sign, 3.3-5.3% prevalence, can occur despite source of otitis, URI, or gastroenteritis
  - Risk factors: Caucasian > Latino > AA, age <= 12 months, fever >= 39C, duration of fever >= 2 days, no source of fever à >= 3 risk factors = 88% sensitivity, 10x risk increase in uncircumcised males
  - Test catheterized or suprapubic urine only
    - Positive UA if urine dipstick is (+) nitrites+/- moderate leukesterase, or microscopic UA has >10-15 WBC/hpf
    - Always send urine cx b/c false-negative rate is 12%
  - Tx for Gram(-) enterics à cefixime, TMP-SMX,

- > 3 years = similar to adolescents/adults
  - Overwhelming number of cases are viral à no testing/treatment
  - Use sx to guide tx: pharyngitis merits rapid strep test, dysuria warrants UA/cx, tachypnea and crackles suggest need for CXR

ALTERED MENTAL STATUS: frequently seen in the ED

- Background: 2-4% of ED patients have AMS, as do 50% of elderly hospitalized patients. 12% of patients with AMS are intubated, and 72% of patients have had symptoms <24 hours
  - Delirium = state of disturbed consciousness associated w/ motor restlessness, transient hallucinations, disorientation, or delusions
    - An acute confusion state, ranging from hypoactivity to hyperactivity. People who may be actively hallucinating, or intermittently exhibiting strange behaviors are also delirious
    - Delirium always has an organic and reversible cause
  - Confusion is a symptom, not a diagnosis.
    - Major categories include primary intracranial disease, systemic diseases affecting the CNS, exogenous toxins, and drug withdrawal
  - Consciousness is an interaction between the reticular activating system (RAS, in brainstem or medulla) and the cerebral cortex. Both must be functioning for a patient to be fully conscious.
    - Disorders of consciousness grouped as diseases that affect arousal fxn, consciousness fxn, or both
  - Coma = any depressed LOC, a complete failure of the arousal system w/ no spontaneous eye opening = brainstem dysfxn and/or bilateral cortical disease
    - Minimally conscious state = inconsistent but discernable evidence of consciousness, an altered state, able to follow commands/purposeful behaviors, simple commands
    - Stupor = patients awaken w/ stimuli but little motor/verbal activity when aroused
    - Obtundation = awake but not alert, and patient exhibits psychomotor retardation
  - Delirium vs. Dementia
    - Delirium à always has an organic and reversible cause
      - Fluctuating course of confusion, may worsen w/ agitation, reversal of sleep-wake cycle often present
      - Acute onset
      - Reversible cause
      - Difficult to distinguish from acute psychosis
      - Depressed consciousness
    - Dementia = stable course of confusion w/ isidious onset
      - Irreversible and slowly progressive
      - No impairment of consciousness

- ED Evaluation:
  - Altered MS? à diminished level of consciousness?
    - Yes = coma, stupor, obtunded
    - No à acute focal neurological deficit? Hemparesis, aphasia, visual field cut?
      - Yes = stroke, mass
      - No à Abnormal attention span, mental status testing?
        - Yes = confusion, delirium (+/- agitation)

    - No = thought disorder, possible psychiatric disorder
  - HPI from paramedics/caretakers
    - Duration of patient's symptoms
    - Patient's baseline function
    - When was patient last at baseline
    - Any medications added or changed recently?
    - Any empty pill/alcohol bottles noticed?
    - Home environment?
  - ABC's come first as always…
  - Evaluating delirium:
    - MMSE
    - GCS à prognosis relies most heavily on motor response
- Differential Dx of Delirium
  - 2 categories: toxic/metabolic vs. structural dz, or medical vs. surgical
  - Mnemonic = AEIOU TIP
    - A: alcohol
      - i.e., respiratory depression, wernicke's encephalopathy
    - E: endocrinopathy, encephalopathy, electrolytes
      - Most common cause of altered MS is hypoglycemia, extreme hypothyroidism, hyponatremia, hypercalcemia, adrenal insufficiency or adrenal crisis, severe thyroid storm, encephalopathy: hepatic, uremic, hypertensive
    - I: insulin, infection, increased ICP
      - Diabetes, systemic infections: meningitis, sepsis, urosepsis/pneumonia in elderly, trauma: SAH, subdural, epidural hemorrhage, hydrocephalus, tumor
    - O: opiates, oxygen
      - Heroin, anoxia: anemia, low cardiac output, pulmonary disease, cardiac disease
    - U: uremia
    - T: trauma, toxic (alcohol, barbiturates, benzos, antidepressants, GHB), tumor, temperature (hypothermia/hyperthermia)
    - I: inborn errors of metabolism
    - P: psychiatric, post-ictal: Todd's paralysis
    - S: seizure, stroke, shock, space-occupying lesions
- Labs + Dx Testing = CBC, BMP, LFT, NH3 level, PT/PTT, tox screen, CXR, ECG, CT head, U/S (GB), LP, exam (skin/soft tissues)
- Tx: disease specific, but consider empiric ABX coverage, most patients are admitted for additional tx
- Do not miss conditions:
  - Hypoxia/diffuse cerebral ischemia
    - Respiratory failure
    - Congestive heart failure
    - Myocardial infarction
  - Systemic processes
    - Hypoglycemia
  - CNS infections
  - Hypertensive encephalopathy
  - Elevated intracranial pressure of medical and surgical origin
  - Hypoxia/diffuse cerebral ischemia
    - Severe anemia
  - Systemic diseases
    - Electrolyte and fluid disturbance
    - Endocrine disease (e.g., thyroid, adrenal)
    - Hepatic failure
    - Nutrition/Wernicke's encephalopathy
    - Sepsis, infection *(occult: skin, GB, impacted stone)*
  - Intoxications and withdrawal
    - CNS sedatives
    - Ethanol
    - Other medication side effects, particularly anticholinergics
  - CNS disease

        - Trauma
        - Infections
        - Stroke
        - Subarachnoid hemorrhage
        - Epilepsy/seizures
            - Postictal state
            - Nonconvulsive status epilepticus
            - Complex partial status epilepticus
    - Neoplasms

SHOCK: Physiologic state characterized by decreased tissue perfusion and inadequate oxygen delivery; SHOCK LECTURE SHOULD BE CALLED LACTATE LECTURE; GET ONE ON EVERYONE!

- Compensated shock (aka warm, early)
    - Physiology:
        - Ogran fxn maintained
        - BP remains normal, only sign may be slightly increased CO
    - Compensatory mechanisms work increase CO via preload, afterload, contractility, HR
        - BP = CO x SVR = (SV x HR) x SVR = ((EDV – ESV) x HR) x SVR
        - Baroreceptors in aortic arch and carotid sinus sense hypotension and stimulate vasoconstriction and increase in HR
        - Chemoreceptors in carotid body respond to cellular acidosis w/ vasoconstriction and respiratory stimulation
        - RAAS system activated by decreased kidney perfusion: increased renin secretion, angiotensin II causes vasoconstriction and aldosterone release
        - Humoral response = cathecholamine release increasing contractility and vasoconstriction
- Uncompensated shock (aka cold, late)
    - Hypotension à microvascular perfusion decreases à organ and cellular fxn deteriorate
        - Decreased urine output causing ARF
        - Restlessness progressing to agitation to obtundation to coma
        - Tachypnea leading to respiratory muscle hypoxia causing worsening acidosis and then respiratory failure
        - Tachycardia causing increased myocardial oxygen demand resulting in increased catecholamines causing more tachycardia and myocardial ischemia
    - Initially reversible, becomes irreversible
- Hypotension: SBP < 90 or MAP < 65
    - Cryptic shock = normal SBP despite profound tissue hypoxia, normotensive but lactate >=4
    - Baseline hypertension = drop of > 40mmHg in SBP is suggestive of shock
    - Baseline hypotension = some patients live in 80s/50s
- Classifications:
    - Hypovolemic: decreased preload
        - CV changes: decreased preload, increased afterload, nl/high contractility
        - Drop in SV, leads to drop in CO, and drop in delivery of oxygen
    - Cardiogenic: problem in contractility
        - CV changes: high preload, normal/high afterload, decreased contractility
        - Think of cardiomyopathies, arrhythmias, valvular/mechanical issues
    - Distributive: essential derangement of homeostasis (drop in SVR, functional hypovolemia)
        - CV changes: decreased preload, decreased afterload, high contractility
    - Neurogenic: loss of sympathetic function leads to loss of vascular tone
        - CV changes: decreased, preload, decreased afterload, decreased contractility
- Spectrum of shock:
    - SIRS criteria
        - Temperature: <96.8 F or >100.4 F
        - HR: >90
        - RR: >20 or PCO2 < 32
        - WBC: <4 or >12 or bands > 10%
    - Sepsis = SIRS + known infection
    - Severe sepsis = SIRS + known infection + organ dysfunction
        - Elevated creatinine, elevated INR, altered MS, elevated lactate, hypotension responding to IVF

- Septic shock = SIRS + known infection + organ dysfunction but NO RESPONSE TO FLUID
- Early Goal-Directed Therapy (EGDT) (Rivers et al., NEJM)
  - Key early measure is CVP (nl in healthy person is 0-4, but for EGDT want 8-12 mmHg before giving vasopressors)
    - Estimates preload
  - MAP goal is 65-90
    - Vasopressors used most often are norepinephrine and dobutamine
    - Use vasodilators if too high (nitro)
  - ScVO2 (central venous oxygen saturation)
    - Goal is >70% (manipulate w/ CO and carrying capacity of oxygen)
    - Normoxia had 20% mortality, but hypoxia had 40% and hyperoxia had 34% à worse outcome if pushing for hyperoxic ScVO2
  - Lab = lactate, Rivers trial used > 4, but nl is <= 2, so 2-4 range is "cryptic"
- "Time to Antibiotic"
  - Early ABX initiation is key: 7.6% absolute increase in mortality per hour

ANTIBIOTIC USE IN THE ED: Key role in bacterial meningitis, PID, SBP, and sepsis
- Goal = learn 1-2 antibiotics for each core infectious indication
- Mechanisms:
  - Inhibit nucleic acid synthesis
    - Sulphonamides – inhibits dihydropteroate synthetase (pre-DHFR step)
    - Trimethoprim – inhibits dihydrofolate reductase (DHFR)
    - Quinolones – inhibit DNA gyrase
    - Nitroimidazoles – Nucleic acid analogues
  - Inhibit cell wall synthesis
    - Penicillins
    - Cephalosporins
    - Vancomycin
  - Inhibit protein synthesis
    - Aminoglycosides
    - Tetracyclines
    - Macrolides
    - Chloramphenicol
- Prescribing 101
  - Every prescription needs: generic drug name, dose (including units), frequency, duration
  - Always assess for medication allergies, warn about SE, and be aware of drug interactions
- Core infectious indications
  - Meningitis
    - Community acquired: ceftriaxone 2g IV q 12h +/- vancomycin 1g IV q 12 h +/- ampicillin 2g IV q4h
    - Brain abscess: ceftriaxone 2g IV q12h +/- metronidazole 500mg IV q12h
  - Sinusitis
    - Acute (<3 weeks): no ABX recommended
    - Chronic (>3 weeks): TMP-SMZ DS BID x 10 days, amoxicillin/clavulanic acid 875 mg BID x 10 days, azithromycin, quinolone
  - Otitis (OM/OE)
    - OM: 81% of cases improve w/out abx, but if tx, consider 1x IM ceftriaxone 50mg/kg
    - OE: mild = acetic acid drops, moderate = corticosporin otic drops, severe/nonresponsive = ofloxacin or ciprofloxacin HC otic drops, malignant OE = levofloxacin 500mg q24h
  - Pharyngitis: most GAS, others are C. diptheriae, N. gonorrheae, EBV, HIV, staphylococcus
    - Acute bacterial pharyngitis: penicillin V 250-500mg PO BID, amoxicillin 250-500mg PO BID, benzathine PCN 1.2 MU IM, azithromycin 500mg 1st dose, f/u 250mg PO x 4 days
  - Bronchitis
    - Acute bronchitis: ABX NOT RECOMMENDED
    - COPD/chronic bronchitis: doxycycline 100mg BID or TMP-SMX DS BID or azithromycin 500mg x1 then 250mg QD
  - Pneumonia
    - Community acquired:

        - S. pneumo à azithromycin 500mg QD + PCN 3 MU IV q4H
        - Mycoplasma/C. pneumo à azithromycin 500mg QD + doxycycline 100 mg BID
        - Multiple lobes (as above, Legionella) à Levofloxacin 500mg IV q24H
    - Aspiration PNA:
        - Out-patient (oral flora) à PCN 3MU IV q4h + metronidazole 500mg q12h
        - Nosocomial à Cefipime 1g IV q12h
- UTI
    - Simple cystitis: TMP-SMX DS BID x 3 days
    - Complicated cystitis: Treat 7 days, quinolone (Levo 250mg QD or cipro 500mg BID)
    - Pyelonephritis: Treat 14 days
- Urethritis
    - Tx for both C. trahomatis & N. gonorrhea à CFTX 125mg IM x1 + Azithromycin 1g PO or 100mg BID x 7days
- Vaginal infections
    - Trichomoniasis à metronidazole 2g PO 1x
    - B. vaginosis à metronidazole 2g PO 1x
    - Cervicitis à CFTX 125mg IM + Azithromycin 1g PO or 100mg BID
    - PID à CFTX 125mg IM + Azithromycin 1g PO or 100mg BID
- Skin infection (including bites)
    - Folliculitis/Furuncle need anti-staphylococcal agents +/- I&D
    - Erysipelas (GAS) needs empiric ABX
- Intestinal infection
    - Often GNR and anerobic organisms, needs early broad-spectrum coverage à ampicililn/sulbactam 1.5g IV q8h + metronidazole 500mg IV q12h –OR--> levofloxacin 500mg IV QD + metronidazole 500mg IV q12h

EMS HISTORY: Brief 2/2 Dickinson stating likely test questions (as below)

- EMS personnel have curriculum set by the Federal Department of Transportation
    - Scope of practice set by each state (therefore, no uniform regulation)
    - Length of training minimums set by DOT, but overseen by states
- In Pennsylvania, physician medical director annually verifies paramedic's authority to provide care
- Paramedics need permission for special procedures and for controlled substance dispensation

HEADACHE EVALUATION IN THE ED: algorithmic approach

- Basic pathway: Starts w/ CC of headache
    - Alarms: evidence of serious HA disorder by H&P?
        - Yes: work-up to identify/exclude secondary HA etiology
        - No: dx of primary HA disorder?
            - Yes: tx primary HA
            - No: consider work-up for secondary HA
- Anatomy:
    - Pain-sensitive cranial structures
        - Venous sinuses w/ afferent veins
        - Arteries at base of brain and their major branches
        - Arteries of the cura
        - Dura near base of brain and large arteries
        - All extracranial structures
    - Pain-insensitive cranial structures
        - Brain parenchyma
        - Ependyma

    - Choroid
    - Pia
    - Arachnoid
    - Dura over convexity
    - Skull
- Mechanisms of HA:
    - Traction on major intracranial vessels
    - Distension/dilation of intracranial arteries
    - Inflammation near pain sensitive structures
    - Direct pressure on cranial or cervical nerves
    - Sustained contraction of scalp or neck muscles
    - Stimulation from disease of eye, ear, nose, or sinuses
- Epidemiology:
    - 60-75% of adults have at least 1 HA per year, w/ 5-10% seeking medical evaluation
    - Less than 10% of ED patients w/ CC of HA will have emergent secondary cause
- Etiology:
    - Primary: tension (69%), migraine (15%), cold stimulus HA (15%), cluster (0.1%)
    - Secondary: hangover (72%), fever (63%), metabolic disorder (22%), nose/sinus (15%), head trauma (4%), eye (3%), vascular (1%)
    - In the ED…
        - Primary: tension (32%), migraine (22%), cluster (<1%)
        - Secondary: Miscellaneous illness (33%), no specific dx (7%), CNS tumor (3%), SAH (<1%), meningitis (<1%), temporal arteritis (<1%), subdural hematoma (<1%)
- Headaches Types
    - Tension: duration 30' to 7 days, at least 2 of (pressing/tightening quality, mild-moderate severity, bilateral, no aggravation by routine physical exam), associated w/ no vomiting and no more than one of (nausea, photophobia, phonophobia); H&P has no suggestion of underlying organic disease
    - Migraine w/out aura: 4-72 hours if untreated or unsuccessfully treated, at least 2 of (unilateral location, pulsating quality, moderate-severe intensity, aggravation by physical activity), associate w/ at least 1 of (N/V, photophobia, or phonophobia); H&P has no suggestion of underlying organic disease
    - Migraine w/ aura: aura defined as at least 3 of (1+ full reversible aura symptoms indicated focal cerebral cortical or brain-stem dysfxn, at least 1 aura symptom developing gradually over >4 minutes or 2+ symptoms occurring in succession, no single aura sx lasting >60', HA beginning within 60' of aura onset); H&P has no suggestion of underlying organic disease
    - Cluster: 15-180' untreated, severe unilateral orbital/supraorbital/temporal pain, associated w/ at least 1 (ipsilateral to pain à conjunctival injection/lacrimation, nasal congestion, rhinorrhea, forehead/facial swelling, miosis/ptosis, eyelid edema), between QOD to 8 per day
    - Secondary: intracranial hemorrhage (SAH, intracerebral, subdural/epidural), meningitis/encephalitis, HTN encephalopathy, ischemic stroke, venous sinus thrombosis, hypoxia, hypercarbia, carbon monoxide, temporal arteritis, mass lesions (tumor, abscess, AVM), altitude sickness, metabolic (hypoglycemia, fever, hypothyroid, anemia), glaucoma, pseudotumor cerebrii (benign intracranial HTN), trigeminal neuralgia, post-concussion syndrome, sinusitis w/out complication, post-LP, diet, medications, fatigue, post-exertion, post-coital
- ED evaluation:
    - History: first/worst, new/frightening feature, last straw? How did HA start? Previous?
    - Pain: Where (unilateral/bilateral, frontal/occipital/facial), character (pulsatile, steady, shocklike, tightness), other symptoms (N/V, LOC, flushing, lacrimation, drop attack, neck stiffness, photophobia, dizziness)
    - Precipitating/aggravating factors: trauma, exertion, noise, position, foods, drugs, weather, anxiety, menstruation
    - Relieving factors: darkness, position, pressing on scalp, medication
    - Medical history: HIV, cancer, HTN, recent procedure (LP), change in meds
    - Family hx: migraines, SAH
    - Environment: carbon monoxide
- Diagnostic alarms: onset >50 y.o., sudden onset, increased freq./severity, new onset w/ risk factors (HIV, cancer), associated w/ systemic illness, altered MS/FND, papilledema, significant trauma
- Treatment:
    - Tension: oral analgesia
    - Migraine: reglan or compazine (10mg IV), serotonin agonists (triptans), narcotics
    - Cluster: 100% oxygen, intranasal lidocaine, NSAIDS
- Diagnostics: CT, LP, limited role for MRI/MRA/angiography, labs based on suspected etiologies

TEMPERATURE-RELATED EMERGIENCIES:

- Heat-related maladies
  - Heat edema: seen early in heat exposure, mostly in elderly, increased aldosterone, self-limited
  - Heat tetany: results from hyperventilation (paresthesias, carpopedal spasm, tetany 2/2 resp. alkalosis), self-limited, remove person from heat
  - Heat rash: pruritic, erythematous rash on clothing-covered parts of body, inflammation of obstructed sweat glands, tx w/ antihistamines, avoid heat, light/loose clothing
  - Heat syncope: early in heat exposure, combo of vasodilation, decreased vasomotor tone, mild dehydration, tx in cool enviro w/ rehydration, check for injuries and other syncope causes, esp. in elderly patients
  - Heat cramps: seen when cooling muscles off, likely 2/2 hypoNa/hypoCl from water w/out electrolytes, needs salt + fluids; may be predisposed to malignant hyperthermia
  - Heat exhaustion: excessive water/salt loss, generally develops over days, non-specific symptoms (HA, N/V, diarrhea, LH, diaphoresis, malaise, myalgias), temp < 40C, normal neuro exam, may be hard to distinguish from resolving heat stroke, tx as above
  - Heat stroke: altered MS w/ core temp > 40.5C (105F), peripheral cooling precedes central cooling so temp may be a bit lower, seen often in patients w/ compromised homeostatic mechanism (i.e., elderly, small children, chronically ill/addicted, obese, those w/out AC)
    - Physical exam: temperature should be measured rectally, anyhydrosis not a criterion for diagnosing heat stroke, may have wet lungs 2/2 vascular endothelial dysfxn
    - Labs: ABG/CXR to r/o ARDS, CBC/COAGS to assess DIC, may see leukocytosis 20-30K w/ thrombocytopenia, UA to screen for ARF/rhabdo, elevated AG from lactic acidosis, elevated LFTs in almost all cases (carry prognostic significance, AST > 1000 = poor), ECG shows QT and ST prolongation, RBBB, sinus tach, atrial fibrillation, SVT, evidence of MI
    - Treatment: aggressive cooling is crucial, options = submerse in ice water, hose w/ cold water, ice-soaked towels, wet+windy, iced lavage, endovascular cooling, cold hemodialysis, stop cooling when ~39C to avoid overshoot hypothermia
- Pathophysiology of heat-related illnesses:
  - Heat dissipation achieved by evaporation, conduction, convection, and radiation, thermoregulation controlled at hypothalamus, Ox-Phos is uncoupled once > 42C
  - Major complications include DIC, ARDS, rhabdomyolysis, ARF, liver failure, seizures
- Cold-related maladies
  - ED Evaluation
    - ABG, electrolytes (BUN may be elevated, possible hyperK), CBC (may see ß WBC, PLT, Ý Hb/Hct due to hemoconcentration), PT/PTT (can see cold-induced coagulopathy), amylase may be elevated 2/2 cold-induced pancreatitis
    - CXR may show evidence of aspiration PNA or pulmonary edema
  - Hypothermia = hypothermia at core temperature < 35C, may be mild (33-35C), moderate (28-32C), or severe (<28C)
    - Types:
      - Mild = shivering and increased pulse/RR/BP/CO, ataxia, hyperreflexia, dysarthria, impaired judgment, cold diuresis, bronchorrhea, bronchospasm, decreased GI motility
      - Moderate = shivering stops, decrased pulse/RR/CO, CNS depression, hyporeflexia, paradoxical undressing, potential cardiac dysrhythmmias (sinus, brady, atrial fib w/ slow ventricular response, VFib, asystole), can see Osborn J wave, patient is sensitive to movement and jostling can precipitate VFib
      - Severe = pulmonary edema, oliguria, loss of reflexes (patellar last to go), hypotension, acidosis, coma, VFib, asystole
    - Management: minimize further heat loss, ABCs
      - Lay person flat to avoid worsening hypotension, likely volume depleted (give IVF)
      - Give glucose, but avoid caffeine
      - Watch for hyper K
      - Hemodynamically unstable patients should be rewarmed faster (>2C/hour), if stable just @ 1-2C/hour
  - Frostnip: mild case of cold injury, reversible
  - Pernio/chilblains: chronic vasculitis resulting from repeated exposures, see red/purple macules/papules/plaques/nodules often on feet
  - Trenchfoot: aka immersion foot, characterized by redness/swelling/throbbing pain/ulcers, can occur at temps up to 60F if feet are constantly wet

- Surfer's ear: exostosis of bone in ear canal, seen in cold water surfers
- Frostbite: frozen tissue, smokers, people w/ prolonged arm/hand vibration, and people w/ vascular disease are at increased risk
  - Management: rewarm w/ water 40-42C, analgesia, leave blood-filled blisters alone, drain clear blisters, aloe vera to affected area, tetanus prophylaxis if not current

INJURIES IN PEDIATRIC PATIENTS:

- Epidemiology:
  - Average pediatric trauma victim = 8 y.o. male weighing 50 lbs. (23kg), blunt trauma more common than penetrating (age + locality specific)
  - Causes:
    - Falls are most common cause of injury in patients 0-14 years
    - MVAs are top cause of *fatal* injury in children 1-18 y.o., followed by drowning (1-14 y.o.) and firearms (15-18 y.o.), other causes = poisoning, suffocation, and fires
  - Affected areas: head (59%), extremities (26%), torso (12%), spine (2%)
- Unique aspects of pediatric trauma:
  - Anatomy/physiology: overall small size, organs more compact, proportionately larger head, smaller/narrower/funnel-shaped upper airway, flatter facet joints, more elastic cervical ligaments, thinner cheset wall, more flexible ribs, horizontal ribs, weaker intercostal muscles, more mobile mediastinum, abdominal organs are more anterior and less subcutaneous fat, softer bones/thicker periosteum, active unfused bony growth plates, compensatory vasoconstriction, larger body surface area/mass ratio
  - Injury response: multiple injuries more common, higher frequency of head trauma, higher frequency of soft-tissue obstruction, greater propensity for spinal cord injury w/out radiologic abnormality (SCIWORA), higher frequency of pulmonary injury, young children are diaphragm breathers, tension PTX poorly tolerated, higher risk of intra-abdominal injury and bleeding, higher frequency of incomplete fractures, disturbed growth after growth plate fractures, normal blood pressure w/ early shock, greater heat loss from exposed body surfaces
  - Psychological differences: fear, pain, anxiety, parental separation, stranger anxiety, lack of ability to reason
  - Therefore..requires an age-appropriate assessment and interactions!
- Primary Survey: Life Support
  - Two major causes of preventable death in pediatric trauma are airway compromise and unrecognized hemorrhage; goal of primary survey is to immediately recognize these and intervene simultaneously
- Pediatric Injury Patterns
  - Wadell Triad = closed head injury, intra-abdominal injury, mid-shaft femur fracture (from getting hit by auto)
- Pediatric vital signs:
  - Infants = HR 140-160, BP min. systolic 70-80, RR 40
  - Preschool = HR 120, BP min. systolic 70+2/yr, RR 30
  - Adolescent = HR 100, BP min. systolic 90-100, RR 20
- Pediatric injury treatment:
  - Avoid hyperventilation unless herniation
  - IVF bolus = 20 cc/kg crystalloid
  - Most solid organ injuries due to blunt trauma in pediatric patients can be managed conservatively with ICU monitoring
- Secondary Survey:
  - AMPLE Hx = allergies, medications, PMHx, last meal, event
- Head trauma case:
  - Trauma is leading cause of death in children > 1 y.o.
  - TBI is leading cause of trauma death/disability: blunt head trauma (BHT) causes 3K deaths, 50K hospitalizations, 650K ED visits w/ 325K CT scans à 90% are negative
- Index of suspicion for intracranial injury (ICI)
  - GCS 15 à ICI prevalence 2-3%, GCS 14 à ICI prevalence 7-8%, GCS 13 à ICI prevalence 25%
  - However, 40-50% of patients w/ ICI have a GCS of 14-15
  - Predictors:
    - If adjust for mental status, LOC is **not** an independent predictor of ICI
    - Seizures **are** an independent predictor of ICI
  - Children < 2 y.o. are a bit different
    - Typical mechanism is fall, cannot verbalize, greater risk of TBI in young infants
    - Abuse must be a consideration

        - 25-30% of infants <= 2 years who are hospitalized w/ BHT are abused
        - Up to 10% of all infants in ED for head trauma are abused
        - High risk of abuse if "no history" of trauma
    - Infants <= 2 y.o. w/ TBI may have subtle signs
        - ~50% are asymptomatic, but scalp hematomas will be present in >90% of otherwise asx infants / TBI, and >95% of infants w/ skull fx

- CT Scans
    - Risks: malignancy
        - 1 lethal malignancy per 2K scans in 1 y.o.
        - 1 lethal malignancy induced per 5K scans in 10 y.o.
        - Up to 3x as many nonlethal malignancies
    - Warning signs:
        - In children >= 2 y.o. include altered MS, LOC, hx of vomiting, mechanism of injury, signs of basilar skull fracture, severe HA
        - In children <= 2 y.o. include altered MS, sclap hematoma in occipital/parietal/temporal region, any LOC, mechanism of injury, palpable skull fracture, acting normally per parents
    - Kuppermann rules (2009) for children >= 2 years old
        - GCS 14 or other signs of AMS or signs of basilar skull fx?
            - Yes (14% of population, 4.3% risk of TBI) à CT recommended
            - No à any hx of LOC, vomiting, severe mechanism of injury, or severe HA?
                - Yes (27.7% of population, 0.9% risk of TBI) à observation v. CT on basis of other clinical factors including MD experience, multiple vs. isolated findings, worsening symptoms or signs, parental preference
                - No (58.3% of population, <0.05% risk of TBI) à CT not recommended
    - Kuppermann rules (2009) for children < 2 years old
        - GCS 14 or other signs of AMS or palpable skull fracture?
            - Yes (13.9% of population, 4.4% risk of ciTBI) à CT recommended
            - No à occipital/parietal/scalp hematoma or history of LOC >= 5 seconds or severe mechanism of injury or not acting normally per parent?
                - Yes (32.6% of population, 0.9% risk of TBI) à observation v. CT on bassis of other clinical factors including MD experience, multiple v. isolated findings, worsening s/sx after ED observation, age < 3 months, parental preference
                - No (53.5% of population, <0.02% risk of ciTBI) à CT not recommended
- Additional Abuse Evaluation Criteria
    - Opthomological exam: **retinal hemorrhages** are seen in <1% of serious unintentional head injury but commonly seen in intentional injury
    - Risk factors include: younger children (<5 y.o.), drug/alcohol use, hx of domestic violence, family hx of maltreatment

COMMON PEDIATRIC RESPIRATORY EMERGENCIES:
- Epidemiology of respiratory complaints
    - 10% of peds ED visits, 20% of peds hospital admissions, major cause of arrest/mortality
- H&P
    - Upper airway symptoms = stridor, supraclavicular and suprasternal retractions
    - Lower airway symptoms = wheezing, subcostal and intercostal retractions
    - Activity level, oral intake/hydration, acuity of symptoms
    - Physical Exam & Vitals
        - Pulse ox = 5th vital sign, normal SpO2 > 95% in children, not sensitive in predicting outcomes, supplemental oxygen is useful even with normal SpO2
- DDX:
    - Croup (lareyngotrachelobronchitis)
        - 6 months – 3 years peak ages, always viral etiology (RIPAM = RSV, influenza, parainfluenza > 60%,

adenovirus, mycoplasma)
    - Invasion of pharyngeal epithelium à spread to larynx, à mucous production and edema à subglottic larynx and vocal cords effected
    - Hx: preceeding URI symptoms, fever, abrupt onset of barking cough, distress w/ crying/agitation, improvement on way to ED
    - Physical Exam: mild-moderately ill, non-toxic, rarely cyanotic or hypoxemic, WOB increased, inspiratory stridor, barky, seal-like cough, lungs clear
    - Radiology to help rule out other dx (e.g., FB aspiration/obstruction, viral URI, tracheitis), see subglottic narrowing, **steeple sign**
    - It is a clinical diagnosis!
    - Labs generally not useful, but may see leukocytosis on CBC
    - Therapy: cool mist/hot shower, dexamethasone 0.6 mg/kg (8mg) PO or IM, racemic epinephrine nebulized if audible stridor at rest
    - Dispo: most are outpatient management, admit if dx is questionable, there is continued audible stridor, toxic appearance, dehydration and vomiting, very young (<3 months?)
- Asthma = airway hyperresponsiveness + chronic inflammation
    - "Late phase" reaction at 4-12 hours – inflammatory cells and mediators, muscle constriction, edema, mucous production, air trapping à dead space ventilation à V/Q mismatch resulting in hypoxemia, hypercapnea, and resp. acidosis, followed by metabolic acidosis 2/2 increased oxygen demand, increased energy consumption, and respiratory failure
    - History: brief and focused on duration/severity of symptoms, current medication use, hx of severe exacerbations, s/sx of infection; later be comprehensive w/ triggers, possible FB aspiration, activity level, oral intake, and ROS
        - Risk factors for severe exacerbations and mortality: hx of sudden/severe attacks, prior intubation and ICU admission, 2+ hospitalizations in last year, 3+ ED visits in last month, hospitalization in the last month, current or recent use of systemic steroids, medical comorbidity, psychosocial problems, age < 5 years
    - Assessment of Physical Findings:
        - RR increased in mild/moderate categories, +/- increased or decreased in severe
        - Retractions mild in mild, moderate in moderate, severe in severe
        - Wheezing is moderate, often end-expiratory in mild cases, loud and throughout exhalation in moderate cases, and both inspiratory and expiratory in severe cases
    - Radiology: needed if there is focal exam, minimal improvement, chest pain, severe exacerbations or this is the first time wheezing
        - See hyperinflation, peribronchial thickening, atelectasis
    - DDX includes anaphylaxis, FB aspiration, bronchiolitis, rings/slings, GER, cardiac dz
    - Therapy: based on pathophysiology
        - Bronchodilators = albuterol (salbutamol), likely to cause tachycardia, hypoK
            - Severe à nebulized 2.5-5.0 mg q20' x 3
            - Mild à MDI 4-8 pufss w/ spacer +/- mask
        - Anticholinergic agent = ipratropium, less potent bronchodilator cf albuterol, don't use along
            - Nebulized 0.25-0.5 mg w/ albuterol
        - Corticosteroids = prednisone/prednisolone/solumedrol
            - Multiple benefits, use early in **all** acute attacks, PFTs show improvement in 2-4 hours and peak in 6-12 hours, oral is as good as parenteral, questionable value of inhaled steroids
        - Other therapies = continuous albuterol, MgSO4, parenteral beta-agonists
    - Dispo: observe 60-90' after last tx, have action plan in place, steroid therapy (oral 2mg/kg per day for 4 days), follow up with PMD
- Bronchiolitis
    - Background: 85% due to RSV, remainder are 2/2 RIPAM, seen most in winter/early spring, peak incicence in 2-8 months old, may be seen up to 2 y.o.
    - Pathophysiology: RSV invades nasopharyngeal epithelium, cell-to-cell transfer to lower airways, cell death and sloughing, mucous production, airway edema and mucous plugging, minor role for airway hyperresponsiveness, medium and small airways are narrowed, mucous plugging/hyperinflation/atelectasis, hypoxia, hypercapnea, respiratory failure, **apnea (in very young infants < 1 month, may be first sign of distress)**

- History: begins as URI +/- fever, progression over 2-5 days, activity level and ability to feed deteriorate, lower UOP, apnea or cyanosis may be seen
- Risk factors: prematurity (< 35 weeks GA), bronchopulmonary dysplasia, heart disease, immunodeficiency, young age (< 3 months)
- Physical Findings: generally lower level of activity, speaking/crying less, WOB increased, exam shows grunting, nasal flaring, retractions, lung exam has wheezing/crackles/decreased aeration
- Labs: rapid antigen testing, respiratory culture, labs if patient is febrile and < 3 months of age (UCx, BCx, and sepsis work-up)
- DDX includes asthma, FB aspiration, bacterial pneumonia, viral myocarditis, vascular abnormality, GER
- Treatment = supportive care, oxygen as needed, IVF, close monitoring, nasal suction, pulmonary toilet, ventilatory support, use albuterol if benefit seen in that patient, racemi epinephrine for more severe distress (0.25-0.5 ml of 2.25% racemic epi), corticosteroids of no benefit but consider if this may be first appearance of asthma
- Dispo: consider current age, gestational age, general appearance, RR, atelectasis, POx

o Foreign Body Aspiration
- Diagnostic challenge seen in younger children, only slightly more common on right side, FB exerts ball-valve effect, and missed diagnosis may lead to PNA
- Physical Examination: classic triad = wheezing, coughing, diminished unilateral breath sounds (seen in only 1/3 of patients), 20% of patients are asymptomatic, hx present in 75% of cases
- Radiology: may see visible foreign body, hyperinflation, infiltrates
- Treatment: ENT consult for bronchoscopy after diagnosis

o Anaphylaxis
o Rings and Slings
o GE reflux
o Pulmonary Disease (CF, PNA)
- Pneumonia
  - Etiologies
    - Neonates – GBS, GN enterics
    - 2 weeks to 2 months – Chlamydia, viruses, S. pneumo, S. aureus, H. flu
    - 2 months to 3 years – viruses, S. pneumo, S. aureus, H. flu
    - 3 years – 19 years – viruses, S. pneumo, mycoplasma pneumoniae
  - Pathophysiology: arrives by aspiration or hematogenous spread, get inflammatory rxn w/ exudate and PMNs, fibrin deposition, macrophage invasion, leads to accumulation of fluid in lobe and effusions and/or empyema may occur
  - Abdominal pain? Shared central pathways for afferent neurons lead to the T9 (LLQ + RLQ) dermatome manifesting pain when lung parenchyma is affected
  - History: bacterial is abrupt onset, fever +/- chills, cough is common but often absent in early disease, chest pain is pleuritic, and vomiting/abd pain are common
  - Physical Exam: tachypnea out of proportion to fever is common, T 38.5-41C, patient may grunt, decreased breath sounds/rales/egophany, dullness to percussion, and referred pain (neck pain/meningismus from upper lobes)
  - Radiology: lobar infiltrate suggests bacterial process, infiltrates are less obvious in dehydrated patients, diffuse/intersitial infiltrates suggests viral vs. mycoplasma
  - Labs: increased WBC w/ left shift strongly suggests bacterial process, pneumococcus associated w/ marked leukocytosis
  - DDX = bronchiolitis, asthma, FB aspiration, sepsis, abdominal process
  - Treatment: ABX = amoxicillin, 3rd generation cephalosporin, macrolides; supportive care = oxygen, fluids, antipyretics, analgesics
  - Dispo = admit if toxic-appearing, in resp. distress, pleural effusion, failed outpatient therapy, young age, underlying disease

o Cardiac Disease

COMMON GYNECOLOGICAL EMERGENCIES:

- DDX for pelvic pain:
  - Gynecologic: ovarian cyst rupture/hemorrhage, torsion ovary/tube, PID, leiomyomata degeneration or torsion, endometriosis, dysmenorrheal, uterine perforation
    Non-gynecologic: appendicitis, gastroenteritis, diverticulitis, cystitis, nephrolithiasis, MSK

- Acute pelvic pain: 5% of ED visits, ~10% of these patients in ED have severe or life-threatening cause or require surgery
- Common OB/GYN issues: ovarian torsion, PID, eclampsia, ectopic pregnancy, hyperemesis gravidarum, Rh incompatibility, uterine prolapse, vaginitis, vulvovaginitis
    - Ovarian torsion: 5th most common, most common in reproductive years (avg. mid-20s), pregnancy predisposes to adnexal torsion (1 in 5), ovarian tumors discovered in 50-60% of cases, abnormal color Doppler flow highly predictive of torsion (but seen in only 50% of cases), but edema is most sensitive marker, **most cases are due to an underlying ovarian pathology that predisposes the ovary to twisting**
        - Common masses include adenocarcinoma in post-menopasual women, polycystic ovaries, large ovarian cysts (young adult women), endometriomas, dermoid tumors
        - 2/3 of torsions occur on R side, increasing likelihood of being confused w/ appy; sigmoid colon in LLQ helps keep L ovary from twisting
        - Aim to dx within 4 hours to save ovary from infarction
        - Lab studies: urine and/or serum beta-hCG, CBC is usually normal, chem panel, LFTs, lipase
        - Imaging: U/S, MRI w/o contrast
    - PID: 11% incidence of tubal occlusion after one episode, any combination of endometritis, salpingitis, peritonitis, and TOA
        - Organisms: N. gonorrhoeae, C. trachomatis, G. vaginalis, anaerobes, ascending infections bypass natural barriers
        - Risk factors = multiple sexual partners, history of STIs, recent intrauterine instrumentation (e.g., endometrial biopsy, D&C)
        - Protective factors: pregnancy after 12th week, barrier contraception, spermicide, OCP
        - Dx: PPV of clinical findings only 65-90%, upper ABD pain/TTP = Fitz-Hugh-Curtis Syndrome seen in 5-10% of cases
            - CDC criteria = low abdominal pain + no other identifiable cause + 1 or more minimum criteria (uterine TTP, adnexal TTP, or CMT)
            - Additional criteria = oral temp > 101F, abnl cervical or vaginal discharge, elevated ESR/CRP, large # WBC on microscopy, lab documention of C/G testing
        - Treatment: ceftriaxone 250mg IM single dose, doxycycline 100mg PO BIDx14d +/- Flagyl 500mg PO BIDx14d (b/c BV/Trich present in many PID patients)
        - Admission: if cannot tolerate PO, severe illness, pregnant, failed outpatient tx, TOA
        - Sequellae of PID:
            - Short-term can get peritonitis
            - Long-term can develop infertility (risk increases w/ each episode and severity of disease)
    - Ectopic Pregnancy:
        - Background: 50% of EPs occur in women who have been pregnant >= 3 times, ~1% heterotopic EP rate for all IVF pregnancies, most common cause of maternal death in 1st trimester
        - Risk factors: contraception reduces risk, but failure of IUDs may increase risk (progesterone IUDs inhibit tubal contractility)
        - Signs of rupture: adnexal tenderness (75-90% of patients, abd tenderness in 80-95% of patients, uterine enlargement, adnexal mass (may be contralateral))
        - Diagnosis: all woman must have urine hCG (11-58 y.o., even s/p tubal ligation), serum hCG, pelvic U/S, pelvic exam, T&S and CBC if vaginal bleeding
            - hGG < 3K in half of EPs, 85% of EPs have serum hCG levels lower than those seen in normal pregnancy at similar gestational age
            - Transvaginal U/S works at hCG levels of 1,500-2,000
        - DDX: salpingitis, threatened/incomplete abortion, ruptured corpus luteum, appendicitis, DUB, adnexal torsion, degenerative uterine leiomyoma, endometriosis
        - Non-operative management: methotrexate (folinic acid antagonist, usually singl IM dose), hCG level should fall at least 15% 4-7 days after dose, and at least 15% weekly thereafter

WOUND EVALUATION:
- Wounds matter least! Always start w/ ATLS principles
- Wound evaluation focuses on:
    - Mechanism: laceration, tear, crush
    - Age of wound: no closure of wounds >12 hours old unless on face or scalp
    - Contaminated wounds: saliva, stool, dirt, wood, organic matter
    - Adequately probe the wounds maximum depth

- Nerves and arteries
- Tendons: explore through full range of motion
- Foreign Bodies: x-ray if necessary

- Wound biology: epithelialization at 48 huors, collagen formation starts days out (about time sutures are removed), wound contractures follow Kraissel's lines of minimal tension, wound strength is 20% at 3 weeks, 60% at 4 months, wounds continue to improve for 6-12 months
- Anesthesia: general ED types include brutane, topical, local, digital, field, conscious sedation
  - Lidocaine (Marcaine) = amide, allergy is rare but can tx w/ 1% Benadryl, give local injection at dermal-subcutaneous junction, with digital locations do neurovascular exam first
    - No epinephrine in fingers/toes, penis, nose, scrotum, ears, or reverse flaps
    - Use 27 gauge needle into the cut skin
- Wound preparation:
  - Cleansing with normal saline is probably best, under pressure, debridement and exploration are critical
- Suturing
  - Techniques:
    - Plan out repair w/ wound approximation w/ central alignment suture
    - Assess the type of repair suitable (layered, interrupted, steri-strip)
    - Plan so margins are slightly everted
      - Eversion is achieved by perpendicular entry through skin and exit in wound
    - Consider skin tension, cosmetics in suture width, spacing, placement
    - Avoid necrosis by appropriate wound tension with adequate loops
    - Minimize damage to the wound—forceps only when needed, debridement is critical
    - Blood pressure cuff for bloodless site
  - Suture materials:
    - Non-absorbable (Ethilon, Prolene) – monofilament, springy, colored
    - Absorbable (Vicryl, Dexon) – braided, 2-3 week life, good for internal/subcutaneous
    - Perform instrument tie with double throw in first knot to avoid tissue strangulation
    - By site:
      - Scalp à 4-0 Prolene, in place for 7 days
      - Face à 5-0 or 6-0 Ethilon, in place for 4 days
      - Inside mouth à 5-0 Vicryl, Silk
      - Fingers à 5-0 Ethilon, in place for 8-10 days, splint as needed
      - Arm à 4-0 Ethilon, in place for 8-12 days
      - Leg à 4-0 Ethilon, in place for 10-14 days
    - Other options include staples and cyanoacrylate glue (e.g., Dermabond)
  - Caveats by site:
    - Always think about injuries to deep or other tissues
    - Scalp: avoid galea hematoma, glass foreign bodies, depressed skull fracture
    - Face: don't shave eyebrows, avoid eye structures, facial nerve, parotid duct
    - Face: think about various eye injuries - test visual acuity
    - Face: ear and nose cartilage is special - Get help
    - Mouth: **ALIGN VERMILION BORDER**
    - Mouth: check for loose teeth, check for teeth in wound
    - Neck: Get help, concern for underlying vascular injury
    - Fingers: check neurovascular exam before lidocaine
    - Fingers: examine both deep and superficial flexor tendons, "through full ROM"
    - Fingers: if any deficits or joint space involvement => call Hand Service
    - Fingers: longitudinal scars can contract
    - Palm: Get help
    - Legs: shins heal very poorly - often require plastics
    - Skin: abrasions require removal of dirt to prevent "tattooing"
    - Skin: patient can debride by themselves to avoid pain
- Tetanus prophylaxis: give for all wounds, 40% of wounds are either minor or not apparent (e.g., burns) so index of suspicion must remain high

    - dT is ok in pregnancy
    - TIG if not fully immunized and current (within last 10 years)
- Dressings and Immobilization:
    - Wounds around joints or in tissues under tension benefit from immobilization and evaluation
    - Xeroform, Adaptic, gauze, paper tape, finger stockinette are options
    - Gas permeable membranes needed for abrasions, burns, road rash, and ulcers
- Wound Care Instructions:
    - Signs and symptoms of infection: rubor, tumor, dolor, calor, functio laesa
    - Elevation
    - Invite back
    - When to do wound checks at one or two days
    - Suture removal instructions
    - Washing - showering - avoid long baths, pools, ocean
    - TIME-SPECIFIC instructions
    - ACTION-SPECIFIC instructions

------- SURGERY SHOCK LECTURE by David Gaieski, MD --------

Critical care is a concept not a location; way of treating patients that begins in pre-hospital setting w/ EMS care, continues in the ED, and is completed in the ICU

Shock = physiologic state characterized by inadequate oxygen delivery
Sepsis = infection plus inflammation
CVP is a surrogate for preload
Severe sepsis = SIRS + infection + end-organ dysfunction (but still responsive to fluid therapy) (can impact brain, liver, kidneys, pulmonary system, cardiac system)

3 causes of shock: hypovolemic, cardiogenic (pump), distributive (resistance)

SIRS criteria:
Temp < 96.8 or >100.4
HR > 90
RR > 20 or PCO2 < 32
WBC < 4 or >12 or bandemia of >= 10%

EVALUATION OF POISON PATIENTS

- Most common: ethanol, acetaminophen, aspirin, psych meds, toxic alcohols
- Whole bowel irrigation for CCBs, lithium, body packers
- Classic toxidromes
- Opioid (note propoxyphene and “opioid-like” clonidine (alpha2 adrenergic agonist), imidazolidines, tramadol (centrally-acting opioid analgesic (u receptors), releases serotonin, and inhibits norepi reuptake))
- Pinpoint pupils
- Respiratory depression
- Lethary to coma
- Bradycardia, hypothermia, borderline hypotension
- Anti-cholinergic (diphenhydramine (OTC cold/sleep meds), antiparkinson/anticholinergic meds (benztropine), misidentified or contaminated plant/herbal products e.g. toxic jimsonweed)
- “Blind as a bat, mad as a hatter, red as a beet, hot as a hare, full as a tick, dry as a bone, the bowel loses tone, and the heart runs alone”
- AKA mydriasis, AMS/delirium, flushing, hyperthermia, urinary retention, dry eyes/skin, decreased bs, tachycardia
- Sympathomimetic (cocaine, amphetamines, anorectics, otc stimulants, “herbal” stimulants)
- Same as anti-cholinergic: tachycardia, hyperthermia, mydriasis

- Unique: hypertension, hyperactive bowels, diaphoresis
- Cholinergic (organophosphates, carbamates e.g. pesticides, donepezil (AChEI for Alzheimer's), physostigmine (myasthenia gravis, glaucoma, Alzheimer's, delayed gastric emptying), pyridostigmine (myasthenia gravis), nerve gas agents)
- D diarrhea/diaphoresis
- U urination
- M miosis
- B bradycardia
- B bronchorrhea
- E emesis
- L lacrimattion
- S salivation/seizures
- Also: AMS, fasciculations, weakness
- Sedative-hypnotic (anticonvulsants, barbiturates, benzos, GHB, methaqualone (insomnia, sedative, muscle relaxant), ethanol)
- Deterioration of CNS function (ataxia, blurred vision, coma, confusion, delirium, diplopia, dysesthesias, hallucinations, nystagmus, paresthesias, sedation, slurred speech, stupor, apnea)
- Most are anticonvulsant but GHB and methaqualone lower the seizure threshold leading to paradoxical seizures

US REVIEW

- Physics
- Velocity = wavelength x frequency
- Distance = velocity x time therefore distance to an object is ½ x time to detect echo "pulse-echo principle"
- Infrasound < 20 Hz, 20 Hz < Acoustic < 20 kHz, US > 20 kHz with medical US between 2 MHz and 20 MHz (water is the medium)
- Increased "resolution" aka frequency gives increased resolution 2/2 shorter wavelength (medical US has mm accuracy). Use max resolution for superficial structures. Decreased resolution gives increased penetration 2/2 longer wavelength.
- Applying AC to piezoelectric crystals à wave transmitted à echo wave à crystals à AC read by voltmeter. Crystals are both transmitters (1% of the time) and receivers (99% of the time)
- Detect depth of the object with time and direction of the object by direction of crystals in the probe
- US is "B-mode" (B for brightness) representing the amplitude of the echos.
- Reflection is a special type of scatter that is concerted and organized
- 3 properties of surfaces that determine propensity to reflect U/S waves
- Impedance mismatch (high impedance = good transmission)
- Specular reflection (smooth regular surfaces reflect uniformly, e.g., diaphragm-pleura interface and most facial planes. Lung and bowel are poor specular reflectors, as is subQ tissue with cellulitis).
- Angle of insonation (i.e., right angles work best. Especially important for deep veins. Tendons can be "anisotropic" meaning that they are echogenic at right angle but anechoic at other angles)
- Sonographic windows are liver, spleen, and bladder (heterogenous tissue).
- EMBU use
- Pointer on probe will project image to left side of screen. Transverse left of screen shows right of patient's body. Longitudinal left of screen shows cephalad. When organ is not oriented in the transverse or longitudinal planes of the body, the probe is oriented according to the organ e.g. neck of gallbladder is cephalad (left of screen).
- Fan to stay in window but look perpendicular to plane, rock to stay in window but look in the plane, and slide if changing windows is ok.
- Gain increases the strength of the returning echoes "black is black"
- Time to gain compensation adjusts gain for depth of object—"near gain" and "far gain"
- Start with max depth then decrease so object fills most of the screen "use all your real estate"
- Resolution pen (low), gen (middle), res (high)
- Also contrast, multibeam processing, color Doppler (toward red, away blue),
- Abnormal findings related to echogenicity:
- Anechoic: free fluid collection
- Hyperechoic: collection of gas or calcium in the liver or kidneys in renal disease (relative to liver)
- Hypoechoic: kidneys in advanced cirrhosis (relative to liver)
- Artifacts

- Acoustic shadowing (dark behind highly reflective objects e.g. bones, calcifications, and gas. This happens when impedence mismatch is so high that no waves are transmitted through)
- Normal = ribs (all bones), bowel gas (dirty shadows)
- Abnormal = renal stones, gall stones, bladder stones
- Posterior acoustic enhancement (opposite)
- Normal = behind bladder and gallbladder
- Gain artifact ("black is black")
- Reverberation artifact (echo bouncing back and forth between two or more highly reflective parallel surfaces that are perpendicular to the beam aka "comit T-tails"
- Normal = bowel gas
- Abnormal = subQ emphysema, foreign bodies (e.g. needles during US-guided venipuncture), alveolar-interstitial disease = pulmonary edema
- Contact artifact (poor probe-patient interface)
- Mirror artifact: (appearance of structure on both sides of specular reflector, seen w/ liver or spleen on both sides of diaphragm)
- Abnormal = mirror artifact is lost when pleural effusion or hemothorax is present
- Also wide beam artifact, side lobe artifact, lateral cystic shadowing (edge artifact)
- Emergency US uses
- Moore's law: use predicted by miniaturization, processing power, cost
- Criteria:
- Urgency/emergency: progressive and potentially lethal (e.g. limited compression US to for DVT in ED vs. duplex US)
- Utility for common illnesses
- Efficiency and convenience: alternatives?
- Safety
- Executability (e.g. limited compression US is more feasible than duplex)
- Simplicity (e.g. global wall motion/EF vs. focal wall motion abnormalities that require specialists)
- Diagnostic:
- Gallbladder disease
- FAST: 10 spaces:
- 4 spaces in RUQ = pleural, subphrenic, hepatorenal, infrarenal
- 1 subxiphoid space
- 4 spaces in LUQ = pleural, subphrenic, splenorenal, infrarenal
- 1 suprapubic space = retrovesicular or pouch of douglas
- Soft tissue infections
- Dehydration/volume status
- DVTs
- AAA
- Pregnancy
- Procedural adjunct:
- Central line placement
- Peripheral venous access
- Thoracentesis
- Paracentesis
- T&A drainage

Worksheet analysis of likely utility of clinician-performed emergency ultrasound for selective common emergency problems

Cardiothoracic symptoms

Symptom / disease process	Anatomic finding of interest	Amenable to emergency bedside ultrasonography? Why? / Why not?
Chest pain 2^0 to Cardiac ischemia	Identification of occluded coronaries	*No*: arteries too small to be clearly visualized
	Focal wall motion abnormalities	*Not usually*: special skills … difficult to master
SOB 2^0 CHF	Global wall motion abnormality	*Yes:* EF estimation can be mastered
	Pulmonary edema	*Yes:* sonography increasingly used to identify 'wet lung' in critical care setting
	2^o to Acute MI	*No:* as for ischemia, above
SOB 2^0lge pleural effn	Pleural effusions	*Yes:* Easily identified. Sono guides thoracentesis
SOB or CP 2^0 to Pericardial effusion vs. Tamponade	Cardiac effusion Tamponade	*Yes:* Findings usually not subtle. No other convenient test *Yes:* Time sensitive, life threatening illness: diastolic collapse of any chamber is abnormal
Massive pulmonary embolus	Clot in lung …… Cardiac abnormalities	…. *No:* lung prevents view of hilum …. *Yes:* RV:LV size reversal, paradoxical septal motion, occasionally clot in heart
Cardiac arrest - PEA	Consider especially for reversible causes hypovolemia …… tamponade …… EMD …… Tension pneumothorax …… Assessment of cardiac standstill …	*Yes*: ….empty hyperdynamic heart, empty IVC and IVC …. As above …. Electrical activity w/o cardiac motion …. Absence of lung sliding + physical exam … absence of motion / gel-like intracardiac densities (clotted blood): consider cessation of resuscitative efforts.
Shock and unexplained hypotension	Hypovolemic	*Yes*: IVC: size, collapse, Doppler flow patterns SVC: ditto, but technically more challenging Internal jugular not useful in most shock patients (cannot sit up) Heart small hyperdynamic heart. LVEF > 75%
Cardiogenic		*Yes*: Heart: LVEF < 30% IVC: plethoric Lung: +/- lung rockets
Mechanical Massive P.E. Tension PTX Pericardial tamponade		*Yes*: R:L ventricle ratio reversal, paradoxical septal motion, plethoric IVC Clinical Dx. Absence of lung sliding Circumferential effusion with diastolic collapse (RV most sensitive and specific). Plethoric IVC. Early maybe only >25% respiratory variation of MV inflow velocity ("tamponade physiology")
Distributive		*Yes*: Heart: hyperdynamic, well-filled with normal/high LVEF and warm, well perfused extremities. High central venous O_2 satuaration.
Septic and neurogenic		Shock caused by one or more of the above mechanisms: hypovolemic, cardiogenic, distributive. Use EMBU to target therapy. Address intravascular volume first.

Abdominal pain: cause	Finding	Amenable to emergency bedside ultrasonography?

		Why? / Why not?
AAA	AAA: Aortic diameter enlarged	*Yes:* ++ Urgency Criterion. Also Efficiency, Executability, Simplicity. Occasionally bowel gas makes technically limited.
Aortic dissection	Intimal flap	+ for Emergency Criterion, but (-) for Executability and Simplicity. TTE Up to 90% accuracy for echocardiologists. Lower for Non-specialists. If +, high specificity.
Gallstones +/- cholecystitis	Stones +/- shadowing, wall thickness, evidence of CBD obstruction, +/- intrahepatic cholestasis	*Yes.* Not so emergent, but convenience and efficiency criteria.
Kidneys	Ureteral stones	*+/-:* may be revealed indirectly by hydronephrosis. Decreasingly by convenience criterion, although in pts w/ recurrent stones EMBU may r/o obstruction and avoid excessive lifetime radiation exposures (Safety Criterion: 1 abd CT = 90 CXRs).
	Pyelonephritis, acute renal failure	*No:* not reliably identified
Trauma	Specific organ injury	*No:* not reliably identified
	Indirect evidence: abnormal collections of free fluid. Search peritoneal, pericardial and pleural spaces	*Yes:* threshold of sensitivity about 500-650 cc free fluid. ++ emergency criteria.
Problems with bowel: appendicitis, diverticulitis, colon cancer, Meckel's, pneumatosis coli	Visualize enlarged, noncompressible appendix at location of maximal tenderness	*No:* air-filled bowel is hard to image by sonography due to diffuse gas. Sono eval requires special skills (the test is performed in pediatrics by specialist sonographers). Possible exceptions: intussusception, appendicitis: may or may not be seen. If +, high specificity.