

# PHM Board Review: Last Minute Review

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## Not for clinical decision making.

**Note:** This document is intended for educational purposes only, as a rapid review of key clinical topics that may appear on certification examinations. This information is not intended for use in clinical decision making, nor does it represent an exhaustive review of these topics. While every reasonable effort has been put forth to ensure the accuracy of this information, no guarantee is made that this information is free from errors or omissions.

## Part 1: Neurology

### 1. Weakness & Paralysis

- Acute Cerebellar Ataxia
- **Dx:** Wide staggering gait, dysmetria, slurred speech typically in a 1-3 year old.
  - **Dx:** normal reflexes, normal mentation and absence of infectious signs
  - **Rx:** Supportive care
  - Typically following viral illness
- Acute Disseminated Encephalomyelitis
  - **Dx:** Encephalopathy with progressive focal neurological signs including cranial nerve dysfunction, flaccid paralysis, and even seizures over 4-7 days. Typically affects children 3-7 years old.
  - **Dx:** Evidence of demyelination on MRI of brain and/or spine
  - **Dx:** Oligoclonal banding of CSF, serum anti-Mog IgG positive
  - **Rx:** Broad spectrum antibiotics until infection ruled out
  - **Rx:** Steroids are first line treatment.
  - **Rx:** IVIG if poor response to steroids (then plasma exchange if still poor response)
  - Note: in contrast to acute cerebellar ataxia, imaging and CSF analysis is indicated for suspected ADEM due to the presence of encephalopathy and focal signs raising the concern for alternative infectious or vascular diagnoses.
  - Typically follows a viral illness, although some bacteria have been implicated as well
  - Like Guillaine-Barré Syndrome, thought to be due to cross reactivity ("molecular mimicry") of myelin components and infectious organisms
- Multiple Sclerosis
  - **Dx:** Diagnosis requires multiple inflammatory demyelinating attacks separated in time and space.
  - **Dx:** Diagnostic criteria include multiple possible combinations of attacks and imaging lesions (and CSF oligoclonal banding)
  - **Dx:** Note that ADEM can count as one of the clinical attacks. While most patients with ADEM do not have MS, a small subset of these patients go on to meet criteria for MS.
  - **Rx:** Steroids for acute attacks.
  - **Rx:** The vast majority of pediatric cases are the relapsing remitting type. Remissions may be prevented to some extent by disease modifying agents, most commonly the CD20 antibody Rituximab.
- Guillaine-Barré Syndrome
  - **Dx:** Ascending paralysis, decreased reflexes
  - **Dx:** Increased CSF protein, oligoclonal bands
  - **Rx:** IVIG or, in severe cases, plasmapheresis

- **Rx:** Monitoring of respiratory status, supportive care.
  - Typically following URI or GI infection
  - Botulism
    - Infantile botulism results from colonization of the intestines.
    - The source is usually the environment (soil), hardly ever honey.
    - **Dx:** **Descending** paralysis starting with cranial nerves (e.g. ptosis).
    - **Dx:** Isolation of spores or toxin from stool (serum is often negative in infantile botulism).
    - **Dx:** But do NOT wait for confirmatory test! If botulism is clinically suspected, treat.
    - **Dx:** Anti-toxin. Call (510) 231-7600 for help from **Infant Botulism Treatment and Prevention Program**.
    - **Rx:** If possible, **avoid** antibiotic treatment at least until antitoxin has been started.
  - Tick Paralysis
    - **Dx:** Similar to GBS, but a tick is present. Sometimes may have unilateral symptoms.
    - **Dx:** Unlike GBS, CSF analysis is normal.
    - **Rx:** Remove the tick!
  - Acute Flaccid Myelitis
    - **Dx:** Acute, rapidly progressive paralysis of part or all of body after a viral illness (especially enterovirus)
    - **Dx:** Abnormal spinal cord MRI
    - **Rx:** Supportive care. Return to full function is unfortunately rare.
  - Peripartum nerve injuries of the newborn
    - Facial nerve injury
      - **Dx:** Asymmetric facies
      - **Rx:** Nothing. Typically resolves in a couple weeks
    - Brachial plexus injury
      - **Dx:** C5-C6 nerve roots (Erb's palsy) -- Arm adducted, internally rotated, forearm extended, hand movement in tact
      - **Dx:** C5-C7 (Erb plus) -- Similar to above, and wrist and fingers are flexed
      - **Dx:** C5-T1 -- Paralysis of arm and hand, Horner syndrome (small pupil, drooping eyelid)
      - **Dx:** C8-T1 (Klumpke) -- Hand paralysis, Horner syndrome
      - **Rx:** Immediate referral for PT. If poor response, surgery in several months may or may not be indicated.
  - Sturge Weber
2. Elevated ICP / Stroke
- Idiopathic intracranial hypertension (IIH)
    - **Dx:** Headache, visual changes, opening pressure greater than about 25 cmH<sub>2</sub>O
    - **Rx:** Therapeutic lumbar puncture, acetazolamide, optic nerve sheath fenestration, shunt
  - IIH secondary to Lyme disease
    - See "Lyme disease" under "CNS infections" below
  - Stroke
    - **Dx:** Clinical suspicion based on focal neurological signs
    - **Dx:** MRI with diffusion weighted imaging is the gold standard
    - **Rx:**
      - ABC's
      - Neuroprotection: maintain normal blood glucose levels, normal temperature, normal electrolyte levels, head elevation (maybe), permissive hypertension.
      - Select patients may be candidates for TPA and/or thrombectomy.
      - Needless to say, patients with suspected or known acute stroke belong in a pediatric ICU under the care of a multidisciplinary team.
3. CNS infections
- Lyme disease

- **Dx:** May present as IHH, but may be accompanied by other symptoms of Lyme disease (erythema migrans, systemic signs)
- **Dx:** CSF typically exhibits pleocytosis. Serum and CSF serologies are positive.
- **Rx:** Doxycycline, 14 days)
- Extension from ear / orbits / sinuses / throat: see ENT section below.
- Primary amebic meningoencephalitis (N. Fowleri)
  - **Dx:** History of water exposure, visualization of amoeba in CSF and/or CSF PCR
  - **Rx:** Combination of amphotericin B, Rifampin, Fluconazole, Miltefosine, Azithromycin
  - Unfortunately, almost universally fatal.
- Cysticercosis: *Taenia solium*.
  - **Dx:** Characteristic cysts on CNS imaging, Serum ELISA, CSF PCR (test available from NIH, useful if ELISA negative but clinical suspicion high).
  - **Rx:** Albendazole + praziquantal (if > 2 cysts) with coadministration of steroids
    - Do NOT initiate treatment if evidence of elevated ICP or hydrocephalus or only calcified lesions

#### 4. Seizures

- **Dx:** EEG =). Characteristic tracings:
  - Absence seizures: 3 hz spike and wave
  - Benign Rolandic Seizures: Centrotemporal spikes
  - West syndrome / infantile spasms: Hypsarrhythmia (high amplitude waves with background of multifocal spikes).
- **Dx:** Simple Febrile seizures are: less than 15 minutes long, do not recur in 24 hours, do not have a focal onset, feature return to a completely normal neurological baseline (possibly after a brief postictal phase), and occur in a child 6 months to 5 years of age who are experiencing a febrile illness.
- **Dx:** Complex Febrile seizures are similar to simple febrile seizures but have focal onset, reoccur within 24 hours, and/or last longer than 15 minutes.
- **Rx:**
  - Simple febrile seizures: assuming the child has a reassuring exam (e.g. normal neuro exam, no evidence of meningitis or other serious bacterial infection), no further evaluation or treatment is required.
  - Complex febrile seizure:
    - There is some evidence that checking electrolytes is useful for risk stratification since there is an association between complex febrile seizures and hyponatremia (which is in turn predictive of reoccurrence).
    - Otherwise, assuming the child has a reassuring exam (e.g. normal neuro exam, no evidence of meningitis or other serious bacterial infection), no further urgent evaluation or treatment is required.
    - However, children with complex febrile seizures may warrant prompt outpatient EEG and/or MRI, particularly if the onset was focal or the seizure was prolonged.
  - assuming the child has a reassuring exam (e.g. normal neuro exam, no evidence of meningitis or other serious bacterial infection),
  - Status epilepticus:
    - Emergently identify and correct electrolyte abnormalities
    - Give ativan → more ativan → levetiracetam → fosphenytoin → Phenobarb or versed drip
    - (There are other options for steps 3 and 4. Recommendation is not to use the same medication twice.)
  - Absence seizures: ethosuxamide
    - Side effects are GI upset and sedation
  - Infantile spasms: ACTH, high dose steroids, or vigabatrin
  - Lennox-Gastaut syndrome: Valproate, then add lamotrigine, then add others
  - Partial seizures: oxcarbazepime / carbamazepine
    - Monitor for hyponatremia

## Part 2: ENT

### 1. Stridor

- Inspiratory
  - Croup (laryngotracheitis)
    - Typically responds to supportive care,  $\pm$  racemic epinephrine and dexamethasone
    - Persistent or recurrent croup may be a sign of airway abnormality such as a vascular ring/sling
      - **Dx:** Chest CTA or MRA (ECHO or Upper GI / Barium swallow are alternatives, but the latter is quite old school.)
      - **Dx:** Also consider laryngoscopy to evaluate for hemangioma, tracheomalacia, etc.
  - Tracheitis (without an artificial airway)
    - **Dx:** similar to croup, but patient appears more toxic and does not respond to racemic epi
    - **Rx:** Typically due to staph, strep, H. flu, M. catarrhalis. Treat with Vancomycin and either ceftriaxone or ampicillin/sulbactam
- Expiratory
  - Foreign body aspiration (most commonly peanuts)
    - **Dx:** Acute onset of expiratory stridor, respiratory distress, cough, asymmetric air trapping on bilateral decubitus films.
    - **Rx:** Get it out (typically via rigid bronchoscopy).
  - Anaphylaxis
    - **Dx:** Diagnostic criteria: Acute onset of illness featuring:
      - Involvement of skin and/or mucous membranes AND either respiratory symptoms, hypotension, GI symptoms OR:
      - Exposure to LIKELY allergen AND TWO of: skin/mucosal tissue involvement, respiratory symptoms, hypotension, GI symptoms OR
      - Exposure to KNOWN allergen AND hypotension.
      - Clinically diagnosis: accompanied by other symptoms including hives, swelling of lips and/or face, nausea, diarrhea, hypotension, with or without exposure to known allergen.
    - **Rx:** **IM epinephrine** (0.01 mg/kg up to 0.5mg). Also diphenhydramine, pepcid, other histamine blockers.
      - Note: delayed epinephrine administration may be associated with biphasic response
      - Note: Epi-pen Jr is 0.15mg and suitable for children up to 25kg. Above that Api-pen (0.3mg) should be used.
      - **Rx:** Patients with severe presentation or requiring more than one dose of epinephrine likely should be admitted for observation.
  - Other intrathoracic compression of the airway

### 2. Head & Neck infections

- Paratonsillar abscess, retropharyngeal abscess
  - **Dx:** Exam findings of muffled voice, enlarged tonsil with uvular deviation, trismus
    - (Obviously, don't even examine the oropharynx if there is concern for the airway. Just head to the OR.)
  - **Dx:** If signs consistent with deep space involvement (trismus, limitation of neck movement, neck swelling), then CT should be obtained to evaluate extent of infection.
  - **Micro:** Infection is often polymicrobial including Staph, Strep, anaerobes (fusobacterium, prevotella, veillonella). Thus, e
  - **Rx:** If any concern for airway compromise, proceed directly to the OR.
  - **Rx:** If significant abscess (>2.5 cm?) or airway compromise  $\rightarrow$  surgical drainage (or, for paratonsillar abscess and perhaps even some retropharyngeal abscesses, needle aspiration).
  - **Rx:** If no improvement on antibiotics after 24-48 hours, re-image.
  - **Rx:** Empiric therapy would be ampicillin/sulbactam or clindamycin. Vancomycin can be added for severe disease
- Lemierre Syndrome
  - **Dx:** Patient present with a history of recent pharyngitis or other parapharyngeal infections and associated neck pain and are typically quite ill appearing.
  - **Dx:** Contrast CT neck, demonstrating internal jugular vein thrombus

- **Micro:** Classically, *Fusobacterium necrophorum*, though other mouth flora may be implicated.
- **Rx: Piperacillin-tazobactam.** Interestingly, there are higher rates of fusobacterium resistance to ampicillin-sulbactam than piperacillin-tazobactam. Vancomycin may be added if the patient is very ill or there is suspicion for MRSA.
- **Rx:** In the very rare case that the CNS is involved, ceftriaxone + metronidazole + vancomycin should be used for better CNS penetration.
- Periorbital cellulitis
  - **Dx:** Clinical presentation of facial and eye lid swelling, but without proptosis, pain on eye movement, or vision changes.
  - **Dx:** Contrast CT of the sinuses and orbits are frequently obtained to rule out orbital involvement and determine likelihood of sinus disease as source (as the source may be either skin or sinuses.)
  - **Micro:** skin (staph/strep) or sinus bugs (including untypable H. flu, M. catarrhalis, S. pneumo, and anaerobes)
  - **Rx:** For straightforward cases in a well appearing child > 1 year of age without concern for orbital or CNS involvement, outpatient management with amoxicillin-clavulanate acid is appropriate. MRSA coverage should be added if no response in 24-48 hours.
  - **Rx:** For hospitalized patients (those who are toxic, have severe disease, or have failed outpatient management), the antibiotic selection would be the same as for orbital cellulitis below.
- Orbital cellulitis
  - **Dx:** Clinical presentation of eye lid swelling, proptosis, pain with eye movement, diplopia, possibly even decreased pupillary light reflex.
  - **Dx:** Imaging is not necessarily always needed, but always seems to be done. Contrast orbit and sinus CT will identify the pathology, but MRI / MR venogram is gold standard to evaluate for CNS extension and/or sinus venous thrombosis.
  - **Micro:** Causative organisms are often polymicrobial and typically include staph or strep, untypable H. Flu and, more rarely, anaerobes (*fusobacterium*, *eikenella*, *peptostreptococcus*, and occasionally even *pseudomonas* or *klebsiella*).
  - **Rx:** There seems to be some variation on empiric therapy:
    - Textbook answer: Vancomycin + ceftriaxone. And, if sinuses are thought to be the source OR if CNS involvement is not ruled out, add metronidazole.
    - Textbook answer alternative: Vancomycin + ampicillin / sulbactam IF there is no concern for CNS extension (since ampicillin / sulbactam does not have great CNS penetration).
    - What I am actually seeing done: ampicillin / sulbactam alone unless concern for CNS involvement.
  - **Rx:** Surgery rarely required, but is indicated in cases of failure of medical therapy or intracranial abscess.
- Neonatal conjunctivitis
  - **Dx:** Inflamed bulbar conjunctiva with copious discharge in the first 4 weeks of life
    - Ocular discharge that does not immediately reaccumulate and is not associated with conjunctival injection likely represent dacryostenosis or even a reaction to the erythromycin ointment applied after birth.
  - **Micro:** Responsible organisms may be either gonorrhea or chlamydia.
    - Recall that chlamydia is not susceptible to erythromycin ointment.
    - Mothers should be screened and treated (and rescreened) for chlamydial infections during pregnancy
      - Maternal treatment would be azithromycin since doxycycline is contraindicated during pregnancy.
  - **Rx:** Therefore, treatment is 1 dose of ceftriaxone (IV or IM), and three doses of Azithromycin.
    - Counsel parents on risk of pyloric stenosis related to azithromycin
- Pott (or Pott's) Puffy Tumor (frontal bone subperiosteal abscess)
  - **Dx:** Clinical evidence of frontal bone abscess plus or minus signs and symptoms consistent with elevated ICP or meningitis, typically in setting of sinusitis or trauma.
  - **Dx:** Contrast enhanced MRI with MR Venography is the gold standard imaging to evaluate for intracranial extension, epidural abscess, and/or sinus venous thrombosis.
  - **Micro:** Essentially the same as periorbital cellulitis above.
  - **Rx:** Broad spectrum antibiotics including coverage for anaerobes (e.g. Vancomycin + Ceftriaxone + Metronidazole)
  - **Rx:** If sinus venous thrombosis is present, anticoagulation (Heparin drip)

- Likely needs long term (3 months?) anticoagulation with LMWH
- **Rx:** Emergent neurosurgery consult for possible drainage (possibly with collaboration with ENT for evacuation of sinuses).
- Otitis media / Mastoiditis with intracranial extension and/or venous sinus thrombosis
  - See Pott Puffy Tumor above
  - Note that mastoiditis is a clinical diagnosis and no imaging is required unless complications (such as CNS involvement) is suspected.
- Dental abscess
  - **Dx:** Clinical presentation of pain and swelling in region of affected tooth, possibly with lymphadenopathy and systemic signs.
  - **Micro:** Polymicrobial, mouth flora including Strep, fusobacterium nucleatum, Bacteroides, Peptostreptococcus, Actinomyces. Staphylococcus may also participate.
  - **Rx:** Extraction, debridement, and ampicillin-sulbactam. Transition to amoxicillin-clavulanate when clinically improving.
- Ludwig Angina
  - **Dx:** Clinical presentation of a rapidly progressing, bilateral cellulitis of the submandibular region, typically without lymphadenopathy.
    - Note: the infection can spread rapidly down the neck and into the mediastinum with accompanying airway compromise.
  - **Dx:** Contrast CT. (In this scenario, an MRI takes too long.)
  - **Micro:** Same as dental abscess. (The source of the infection is usually a mandibular molar tooth)
  - **Rx:** airway management and ampicillin-sulbactam  $\pm$  vancomycin. There is usually no abscess to drain.
    - Some recommend a 2-3 week course of IV ampicillin-sulbactam. Others transition to amoxicillin-clavulanate after clear clinical improvement.

## Part 3: Pulmonology

### 1. Bronchiolitis

- This is a very rare condition that is outside the scope of this review.

### 2. Asthma

- Definition of "well controlled asthma"
  - $\leq 2$  episodes of night time coughing per month
  - $\leq 2$  episode of rescue inhaler use per week (not counting exercise prophylaxis)
  - Unrestricted daily activity
  - (There are also PFT criteria, which I would say are outside the scope of PHM)
- Risk factors for exacerbations
  - Obesity, sinusitis, GERD, anxiety, depression
  - Exposure to cigarette smoke, allergens (if allergic to them), air pollution
  - Socioeconomic barriers to care
- **Dx:** There are a variety of inhaled corticosteroids each with different dosing at different ages. Hopefully we need not commit these to memory.
- **Rx:** Unfortunately, there are competing guidelines (NAEPP, GINA) and practices.
- **Rx: The fundamental principles are:**
  - Start with PRN SABA, escalate to daily ICS, then escalate to daily ICS-LABA (e.b. budesonide/formoterol).
  - If low dose ICS-LABA does not achieve control, escalate the ICS dose.
  - ICS-LABA can be used as both controller and rescue med (**SMART therapy**) for children 5 and over. (The GINA guideline only allows for this in adolescents and adults).
  - High dose ICS-LABA should not be used as a rescue med. Use PRN SABA if you get to that point
  - Children 4 and under with intermittent asthma should have a **short course ICS with viral illnesses**.
- **Rx:** The NAEPP guidelines are summarized below. The GINA guidelines are similar but not exactly the same.

**Classification**

	<b>Symptoms</b>	<b>Nighttime waking</b>	<b>Activity limitation</b>
NAEPP: Intermittent (Step 1)	≤2 / week	≤2/month	None
NAEPP: Mild persistent (Step 2)	3 - 6 / week	3 -4 /month	Minor
NAEPP: Moderate persistent (Step 3)	Daily	3 -4 /month	Moderate
NAEPP: Severe Persistent (Step 4)	Throughout	> weekly	Extreme
NAEPP: Severe Persistent (Step 5)	(Same as above, failing step 4 therapy)		

**Treatment (NAEPP)**

	Age	ICS at onset		Daily low-dose ICS	Daily low dose ICS-LABA	PRN low dose ICS-LABA	Daily med. dose ICS-LABA	PRN med dose ICS-LABA	Daily high dose ICS-LABA	Med. Dose ICS-LABA + LAMA
		SABA	PRN							
Step 1	≤ 4 years	X*	X							
	5-11 years	X								
	>11 years	X*								
Step 2	≤ 4 years	X		X						
	5-11 years	X		X						
	>11 years	X		X						
Step 3	≤ 4 years	X			X					
	5-11 years	*			X	X*				
	>11 years				X	X				
Step 4	≤ 4 years	X					X			
	5-11 years	*					X	X*		
	>11 years						X	X		
Step 5	≤ 4 years	X							X	
	5-11 years	X							X	
	>11 years	X								X

\* The GINA guidelines do not endorse ICS-LABA as a rescue med in this age group. Escalating ICS and using PRN SABA would be the alternative.

3. Community acquired pneumonia

- **Dx:** Focal lung exams, hypoxia, fever.
- **Dx:** Patients requiring hospitalization (<6 or so months of age or hypoxic, distressed, and/or toxic) should in most cases get a chest x-ray to evaluate for potential complications (e.g. empyema, abscess, etc.)
- **Dx:** If moderate to large (>1/4 chest diameter) effusion is present, obtain US to characterize the effusion.
- **Micro:** In preschool children 80% of cases of pneumonia are viral!
- **Micro:** S. pneumo (both non-vaccine types, and vaccine types--the vaccine is far from perfect!), Staph, Strep, non-typhable H. Flu, M. catarrhalis.
- **Micro:** In school aged children, M. pneumonia is an important cause of community acquired pneumonia.
- **Rx:** Ampicillin for uncomplicated pneumonia in a fully vaccinated patient requiring hospitalization.
- **Rx:** Ceftriaxone for uncomplicated pneumonia in an under vaccinated patient requiring hospitalization.
- **Rx:** Ceftriaxone for complicated pneumonia (empyema, necrosis, or abscess).
  - Add clindamycin or vancomycin if concern for MRSA or very severe disease. (Personally, I am concerned for MRSA if there are abscesses, if there in concurrent or recent influenza infection, or if there is a family history.)
- **Rx:** Small effusions require no special treatment. Moderate effusions may require drainage, especially if there is significant respiratory distress. Large effusions (> 1/2 the chest diameter) should be drained. TPA and/or VATS is indicated if the effusion is complicated as demonstrated by US.

4. Tracheitis: See ENT section above.

5. ARDS

- At my institution, ARDS seems mainly constrained to the PICU. However, I have encountered some practice questions on the topics.
- Pearl #1: ARDS features worsening respiratory distress combined with worsening lung opacities on CXR or CT not explained by

collapse or pulmonary edema (from fluid overload), combined with impaired oxygenation.

- Pearl #2: The pathology involves alveolar injury, filling of alveolar with proteinaceous fluid, and surfactant loss.
- Pearl #3: Treatment is focused on the underlying disease process combined with supportive care that is beyond the scope of the pediatric hospitalist.

6. Foreign body aspiration: See section on stridor in ENT section above.

## Part 4: Cardiovascular

### 1. Endocarditis

- Major Duke Criteria
  - Two blood cultures positive for organism consistent with infective endocarditis
    - Strep
    - Staph
    - Haemophilus species
    - Aggregatibacter actinomycetemcomitans
    - Cardiobacterium hominis
    - Eikenella corrodens
    - Kingella kingae
  - Echocardiographic evidence of endocarditis
- Minor Duke Criteria
  - Predisposing heart condition or injection drug use
  - Fever: temperature  $>38^{\circ}\text{C}$
  - Vascular phenomena: (Septic or vascular emboli, Janeway lesions, etc)
  - Immunologic phenomena: (GN, Osler's nodes, etc.)
  - Microbiological evidence: single positive blood culture with organism consistent with infective endocarditis
- **Dx:** Definitive endocarditis is diagnosed based on both major, 1 major + 3 minor, or all 5 minor criteria
- **Dx:** Possible endocarditis is diagnosed based on 1 major + 1 minor or 3 minor criteria
- **Rx:** Treatment for native valve endocarditis is based on the organism isolated. Typical regimens are penicillin or ceftriaxone for 4-6 weeks.
- **Rx:** Prosthetic valve endocarditis is most commonly due to S. Aureus. Treatment is ampicillin or oxacillin or nafcillin AND gentamicin, and rifampin. Vancomycin instead of penicillin derivative if MRSA.
- **Rx: Prophylaxis** with amoxicillin prior to dental, pulmonary, or cardiac procedures is recommended for individuals with any of the following:
  - History of infective endocarditis
  - Unrepaired cyanotic congenital heart defects
  - Partially repaired congenital heart defects
  - Any cardiac prosthetic material (if material relates to complete repair of a CHD, prophylaxis is only needed for first six months)

### 2. Pericarditis

- **Dx:** Clinical constellation of chest pain relieved by sitting up and forward, friction rub on exam, and widespread ST elevation on ECG.
- **Rx:** NSAIDs. If that fails then colchicine, possibly steroids, possibly surgery.
- **Rx:** Note--bacterial pericarditis is substantially more rare than idiopathic/inflammatory pericarditis. But in toxic appearing patients, consideration should be given to broad spectrum antibiotics and surgical drainage of pericardial sac.

### 3. Cardiomyopathies

- Dilated cardiomyopathy with pump failure (diminished ventricular function)



- Typically secondary to myocarditis, which is typically secondary to viral infection (most common is Coxsackie B27)
- **Dx:** Early findings may be subtle (fatigue, poor feeding, irritability).
- **Dx:** Later findings are more obvious and include hepatomegaly, liver down, pulmonary edema, hypotension.
- **Rx:** Treatment for myocarditis itself is supportive (fluid management) and possibly IVIG.
- **Rx:** Treatment of dilated cardiomyopathy with pump failure begins with ACE inhibitors (e.g. enalapril), followed by numerous other therapies that will be managed by your friendly neighborhood pediatric cardiologist.
- Hypertrophic obstructive cardiomyopathy(HOCM)
  - **Dx:** Systolic ejection murmur worse with valsalva.
  - **Dx:** Exertional angina, presyncope, and/or syncope.
  - **Dx:** EKG, Cardiac echo
  - **Major risk factors for sudden cardiac death related to HOCM:**
    - Close relative with sudden cardiac death under 50 years of age likely due to SCD
    - **Syncope**
    - Massive LVH
    - Prior sustained ventricular arrhythmia
    - EF < 50%
  - **Rx:** In the absence of risk factors for SCD, first line treatment for symptomatic HOCM is beta blocker (unless asthma, or possibly diabetes).
    - Patients with risk factors for SCD may require implantable defibrillator.

#### 4. Congenital heart defects

- Heart defects with ductal dependant systemic circulation (and/or oxygenation)
  - These are defects affecting the left sided outflow tract
  - Hypoplastic left heart
  - Aortic stenosis
  - Aortic coarctation
  - Transposition of the great vessels (depending on systemic and pulmonary vascular resistance)
- Heart defects with ductal dependant pulmonary circulation
  - These are defects affecting the right sided outflow tract
  - Tetralogy of fallot
  - Pulmonary atresia / stenosis
  - Ebstein's anomaly (ASD, downward displacement of the tricuspid valve)
- **Rx:** Infants with ductal dependant circulation may present with circulatory collapse and/or marked respiratory distress. In addition to standard resuscitative maneuvers (PALS), emergent therapy includes Prostaglandin E2 administration.
- **Dx:** Characteristics chest x-ray findings
  - Egg on a string: Transposition of the great vessels
  - Boot shaped: Tetralogy of fallot
  - Cardiomegaly or "box shaped" heart: Ebstein's anomaly
- Heart Defect ↔ Genetic Disorder associations
  - Di-George :: Truncus Arteriosus ("22 Truncus Two" -- credit to Dr. Andrea Hadley)
  - Trisomy 13 :: PDA, VSD, ASD
  - Trisomy 18 :: VSD
  - Trisomy 21 :: AV canal defects
  - Turner syndrome :: Aortic coarctation (the aorta is turning and getting kinked)
  - Williams syndrome :: aortic stenosis, pulmonary atresia

#### 5. Syncope

- **Syncope red flags**
  - No prodrome

- Palpitations as a prodrome
  - Syncope in the context of hyperplastic obstructive cardiomyopathy
  - If these red flags are present, prompt cardiac workup is warranted.
  - Sudden Vasovagal syncope
    - **Dx:** Drop in blood pressure and heart rate with prolonged standing or vagal maneuvers (e.g. defecation)
  - Orthostatic hypotension
    - **Dx:** Drop in blood pressure, increase in heart rate upon position change.
    - Usually only occasional
  - Postural orthostatic tachycardia syndrome (POTS)
    - **Dx:** Dramatic rise in heart rate with minimal change in blood pressure upon position change
    - Typically presyncope
    - Interferes with quality of life due to frequency of tachycardia symptoms
  - **Rx:** Increase fluids and salt intake, adequate exercise,  $\pm$  compressive stockings
  - **Rx:** For POTS, beta blockers, and counseling to help with coping strategies
  - **Rx:** For vasovagal syncope, avoidance of provoking activities
6. Arrhythmias / Cardiac Arrest
- Sudden cardiac death in the context of a structurally normal heart (e.g. no congenital heart disease, no ischemia) is usually the result of ventricular arrhythmia.
  - Examples of causes of ventricular arrhythmia
    - Brugada syndrome (right bundle branch block and elevated ST segment on V1 and V2 on ECG)
      - **Rx:** Implantable defibrillator may be indicated
    - Prolonged QT
      - 99th percentile for QTc is 470 in adult males and 480 in adult females
      - **Rx:** First line treatment is beta blocker
    - Pre-excitation (e.g. Wolf-Parkinson-White, delta waves on ECG)
      - **Rx:** Ablation if high risk (e.g. persistent delta waves on ECG including during exercise and high risk electrophysiology demonstrated with invasive testing).
    - Complete heart block
    - Trauma (commotio cordis)
  - Supraventricular Tachycardia (SVT)
    - **Dx:** narrow complex tachycardia with minimal variability and absent (or dysmorphic) p-waves, with a heart rate typically > 180 (>220 in infants)
    - **Rx:** If unstable, synchronized cardioversion (0.5 - 1J/kg, then 2J/kg if initial shock does not work).
      - Ok to give adenosine while someone is running for the crash cart, but do not delay cardioversion for these maneuvers (especially if you do not already have IV or IO access).
    - **Rx:** If clinically stable, vagal maneuvers and adenosine (0.1mg/kg to max of 6mg for first dose, 0.2mg/kg to max of 12 mg for second dose if needed).
      - Use an IV as close to central location as possible. Administer quickly and quickly flush with saline.
  - Ventricular Tachycardia
    - **Dx:** Wide complex tachycardia, absent or dissociated p-waves.
    - **Rx:** Synchronized cardioversion, same as SVT above.
  - Ventricular Fibrillation
    - **Dx:** Disorganized, irregular jagged QRS complexes
    - **Rx:** Defibrillation, 2J/kg, then 4, then continue to progress up to 10J/kg or adult dose (whichever is lower)
    - **NOTE:** Full review of PALS is (way) outside the scope of this outline. See UpToDate or your PALS cards for review.
7. Hypertension

- In the majority of cases, hypertension in the acute care environment is best addressed by follow up with PCP after resolution of acute illness.
  - **Dx:** In some cases, such as during prolonged hospitalization, hypertension in a very young patient, or concerns regarding access or follow up, inpatient evaluation including ECG, renal ultrasound with doppler, possible ECHO, and renal function panel may be desired.
  - **Rx:** Whatever you do, NEVER give ACE inhibitors or ARBs to a hypertensive pediatric patient in whom bilateral renal artery stenosis has not been rule out (as this can precipitate renal failure).
  - **Rx:** PRN hydralazine or isradipine are fine choices for short term treatment of asymptomatic hypertensive inpatients below the hypertensive urgency range.
- However, hypertensive urgencies and emergencies obviously warrant acute treatment.
- **Dx:** If less than 6 years old, should get an ECHO (to, among other things, rule out coarctation)
- **Dx:** Hypertension classification
  - Elevated: 90th-95th percentile or 120-129 / <80
  - Stage 1: 95th percentile - 95th percentile + 12 or 130 - 139 / 80 - 89
  - Stage 2: > 95th percentile + 12 or > 140/90
  - Hypertensive urgency: BP 30mmHg above 95th percentile for age/sex/height (or >180/120 for an adolescent)
  - Hypertensive emergency: Stage 2 hypertension or higher and symptoms of end organ dysfunction (hematuria, proteinuria, seizures, vision changes, papilledema, etc.)
- **Dx:** Look for evidence of causes of secondary causes
  - History of prematurity (Reno-vascular hypertension)
  - Cushingoid appearance (Cushing disease or syndrome)
  - Hearing loss (Alport disease→Kidney disease)
  - Rash, arthritis (rheumatologic disorder with possible kidney involvement)
  - etc.
- **Rx:** First step is ALWAYS manual confirmation with appropriate cuff.
- **Rx:** Identify and treat causes (elevated ICP, pain, fever, toxic exposure, etc.)
- **Rx:** Do NOT treat hypertension in setting of elevated ICP without consultation with neurosurgery and, where possible, pediatric neurocritical care.
- **Rx:** Labetalol to achieve 25% of goal reduction over 8 hours (ideally this should be done in the PICU).
  - Goal reduction: current BP - 95th percentile, or 130/80 for adolescents

## Part 5: Gastrointestinal

### 1. Appendicitis

- Non-operative management of early appendicitis
  - **Dx:** Criteria
    - <48 hour of symptoms
    - WBC < 18,000
    - Normal CRP
    - No appendicolith and appendix < 1.1 cm in diameter
    - No evidence of peritonitis or rupture
  - **Rx:** Piperacillin-tazobactam or ceftriaxone + metronidazole, de-escalating amoxicillin-clavulanate for 7-10 total days
  - Benefits
    - Avoidance of surgery and its complications
  - Risks
    - May need appendectomy in the future

- Not a risk per se, but length of stay seems to be the same with medical management vs. surgery
- If above criteria or not met, appendectomy is recommended. (And, in fact, adoption of non-operative management is evolving. Your mileage with your consultants may vary.)
- Advanced appendicitis (presence of rupture)
  - **Rx:** Initiate piperacillin-tazobactam
  - **Rx:** Urgent surgery consult for possible appendectomy/washout
- Intrabodinal abscess
  - **Rx:** piperacillin-tazobactam
  - **Rx:** If ill appearing, urgent surgery consult for possible appendectomy/washout
  - **Rx:** Consider drainage if not responding to treatment

## 2. Cholecystitis / Cholangitis

- Acalculous cholecystitis
  - **Dx:** Clinical presentation of abdominal pain, possibly fever, very rarely increased bilirubin.
  - **Dx:** US showing gall bladder thickening without signs of stones or obstruction
  - **Rx:** Although the primary etiology is usually inflammatory, infectious can be either a cause or complication. Treatment with piperacillin-tazobactam is appropriate.
  - **Rx:** If therapy fails, drainage of the gall bladder may be indicated.
- Calculous cholecystitis
  - **Dx:** Clinical presentation of abdominal pain, possibly fever, possible elevated bilirubin if obstructing stone.
  - **Dx:** US showing gall bladder stones, and there may be signs of inflammation and/or obstruction.
  - **Rx:** Uncomplicated calculous cholecystitis is managed conservatively with pain management. Cholecystectomy may be performed once inflammation has abated.
  - **Rx:** Piperacillin-tazobactam to prevent infectious complications are typically given.
  - **Rx:** If there is evident of necrosis, rupture, or if the patient is septic, urgent cholecystectomy may be indicated.
- Ascending cholangitis
  - **Dx:** Clinical presentation of abdominal pain, fever, an jaundice.
  - **Dx:** Typically occurs in the context of gall stones or congenital biliary malformations
  - **Rx:** Piperacillin-tazobactam
  - **Rx:** ERCP should be considered for patients that are septic (in which case, urgent drainage may be required), that fail medical management, or have ongoing biliary obstruction.

## 3. Pancreatitis

- **Dx:** Two of: epigastric pain, amylase or lipase > 3 time upper limit of normal, imaging consistent with pancreatitis.
- **Dx:** US to evaluate for obstructive process or trauma.
- **Rx:** Stop potential instigating medications (valproic acid, prednisone, 6MP, etc.)
- **Rx:** Aggressive fluid resuscitation followed by 1.5 MIVF
- **Rx:** Pain control
- **Rx:** ERCP if bile duct obstruction (ASAP if evidence of cholangitis)
- **Rx:** MRCP if < 3 years of age or recurrent pancreatitis.
- **Rx:** No antibiotics indicated if no or limited pancreatic necrosis and no evidence of cholangitis
- **Rx:** If patient has gallstones, consult pediatric surgery for cholecystectomy prior to discharge.
- **Rx:** Low fat diet as soon as tolerated (consider slow advance of NG feeds if cannot tolerate PO after 24 hours, or TPN if enteral feeding not possible.)
- Discharge when pain controlled and tolerating PO. No need to trend labs.

## 4. GI bleeding

- Common causes of hematochezia in infants
  - Ingested maternal blood
  - Milk protein intolerance

- **Dx:** Infant (typically < 6 months of age) with loose, mucousy, blood streaked stools with no other evidence of illness.
- **Rx:** Milk (and, perhaps, soy) elimination by mother is an option.
- **Rx:** Otherwise, extensively hydrolyzed formula. (e.g. Nutramigen or Alimentum). (Many of these infants are also allergic to proteins in soy based formula).
- Necrotizing enterocolitis (typically observed in preterm infants, but can occur in term infants)
- Anal fissures
- Dehydration with urate crystals mistaken for blood.
- Meckel's diverticulum
  - **Dx:** Clinical presentation of painless GI bleeding.
  - MOST commonly diagnosed in first year of life, but may be diagnosed at any age including adulthood.
  - **Dx:** Typically diagnosed with Meckel's (99m technetium pertechnetate) scan, but arteriography or CTA may be used in the rare case of very brisk bleeding.
  - **Rx:** Treatment includes stabilization and consideration of PPI (until upper GI source is ruled out), followed by surgical resection.

## 5. Obstruction

- Pyloric stenosis
  - **Dx:** Progressive non-bloody, non-bilious vomiting, classically projectile. Peak incidence is around 4 weeks of age.
  - **Dx:** Classically accompanied by hypokalemic hypochloremic metabolic alkalosis, although this is often not apparent with early diagnosis.
  - **Rx:** Fluid resuscitation, MIVF, consult to pediatric surgery for pyloric myotomy.
- Intussusception
  - Typically ileo-colic unless there is a pathologic lead point leading to ileo-ileal intussusception (e.g. tumor, IgA vasculitis, Meckel's diverticulum.)
    - Older children are more likely to have pathologic lead point.
  - Note: Note bleeding (e.g. "red currant jelly stools") is a late finding with intussusception. (I personally have never encountered intussusception late enough the course to be accompanied by bleeding.)
  - **Dx:** Severe, often intermittent abdominal pain. Following resolution of bouts, patient may seem sleepy or even listless.
  - **Dx:** Diagnosis can sometimes be made by means of abdominal plain films, but abdominal US is the diagnostic test of choice.
  - **Rx:** Air enema is both diagnostic and therapeutic.
  - **Rx:** Recurrent intussusception may require exploratory laparotomy, particularly if there are multiple recurrences and/or suspected pathologic lead point.
- Small bowel obstruction
  - **Note:** Bilious vomiting is a surgical emergency until proven otherwise.
  - **Dx:** Abdominal distension and tenderness, vomiting, hemodynamic instability
  - **Dx:** Abdominal plain films can demonstrate obstructive gas pattern and/or free air.
  - **Dx:** An upper GI with small bowel follow through is the preferred modality for confirming bowel configuration (e.g. malrotation with or without volvulus) and patency if patient condition will permit.
  - **Rx:** NPO, gastric decompression via NG tube to low intermittent suction, MIVF, emergent consult to pediatric surgery for surgical correction.

## 6. Foreign bodies

- **Rx:** Magnets, batteries, and sharp objects in the esophagus, stomach, or duodenal bulb should be emergently removed endoscopically.
  - Although there is some difference of opinion regarding button batteries in the stomach.
  - It is also an option to simply observe a single ingested magnet in the esophagus or stomach.
- **Rx:** Any foreign body causing airway compromise or remaining in the esophagus for more than 12 hours should be endoscopically removed.
- **Rx:** Any foreign body past the duodenum in an asymptomatic patient can be observed at home for passage.

- "Magnet precautions" -- avoid prolonged contact with metal things that might cause magnet to adhere and erode through bowel wall.
- Carefully counsel on symptoms that should prompt return for care.
- **Rx:** Any foreign body past the duodenum in a symptomatic patient should be surgically removed.

## Part 6: Nephrology and Urology

### 1. UTI / Pyelonephritis

- This topic is pretty straightforward and well protocolled by the AAP guidelines so is omitted (for now) from this review.

### 2. Nephrolithiasis

- Remember that pyelonephritis with an obstructing stone is a urological emergency, particularly if any signs of sepsis.
- While acute management of nephrolithiasis is relatively straightforward a couple special cases bear mentioning:
  - Uric acid stones
    - Alkalinization of urine may help avoid surgical removal of a stubborn kidney stone.
  - Struvite (magnesium ammonium phosphate and calcium carbonate-apatite) stones
    - Tend to form stag horn calculi and to be a nidus of infection
    - Thus typically need to be removed by your friendly neighborhood pediatric urologist

### 3. Nephrotic syndrome

- **Dx:** Most common diagnosis is minimal change disease, most frequently diagnosed in children less than 6 years of age
- **Rx:** Nephrotic range proteinuria (**>50mg/kg/day, or  $U_p/U_{Cr} > 3$** ) can be empirically treated with steroids IF the following criteria are met:
  - Age 1 - 10 years
  - Normal blood pressure
  - No gross hematuria
  - Creatinine normal for age
- **Rx:** Steroid regimens varies. Here is one option: prednisone 2mg/kg day is given for 4 weeks, followed by 1.5mg/kg/every other day for 4 more weeks. (Note that all published guidelines involve steroids for a minimum of 8 weeks, and most for at least 12 weeks)
- If nephrotic syndrome recurs, or for patients who do not meet above criteria, referral to pediatric nephrology for biopsy and further management is indicated.

### 4. Asymptomatic microscopic hematuria

- **Dx:** Red flags
  - Hypertension
  - Hematuria accompanied by proteinuria with  $U_p/U_{Cr} > 0.2$  or proteinuria that persists for more than a week or two
- **Rx:** Absent these red flags, isolated hematuria in an asymptomatic patient may be followed expectantly
  - **Repeat weekly UA x 2.** (Most hematuria is transient and related to minor illness or exercise)
  - If UA continues to be positive but patient remains free of red flag signs and symptoms and UTI has been ruled out, follow **every three months** with repeat UA and blood pressure.
  - **If hematuria persists > 12 months** or patient develops red flag signs or symptoms, **refer** to pediatric nephrology.
- Glomerulonephritis is a wide ranging and complex subject. Hopefully the boards will let us simply refer patients with suspected glomerulonephritis to our friendly neighborhood nephrologist (which I would do in a hot minute in real life).
  - Although it is worth noting that post-streptococcal glomerulonephritis is the most common cause of GN and is usually self limited, with resolution of hematuria within six months and full recovery of kidney function.

# Part 7: Urogenital / Gynecology

## 1. Ectopic pregnancy

- **Dx:** Like pelvic inflammatory disease and ovarian torsion, ectopic pregnancy must be considered in any female patient of reproductive age presenting with abdominal pain.
- **Dx:** Typically presents 6-8 weeks after last menstrual cycle.
- **Dx:** Assuming history and physical exam are suggestive, serum HcG is initial screen. If positive, transvaginal ultrasound can confirm the location of gestational sac.
- **Rx:** A hemodynamically unstable patient with known or suspected HcG needs emergent consult to Ob/Gyn for surgical management.
- **Rx:** There are a couple of options (surgery, methotrexate, or very rarely expectant management) for management of ectopic pregnancy in a hemodynamically stable patient, so a consult to Ob/Gyn is required anyway. But if hemodynamically stable you can walk swiftly to the phone instead of running.

## 2. Ovarian torsion

- **Dx:** Ovarian torsion should be suspected in any patient with lower quadrant abdominal pain with normal appendix and (where applicable) negative evaluation for ectopic pregnancy.
- **Dx:** Diagnosis is supported by abdominal ultrasound showing an adnexal mass and, possibly, appearance consistent with torsion.
- **Dx:** Blood flow demonstrated by doppler ultrasound **does not** rule out torsion.
- **Dx:** The gold standard for ruling out torted ovary is visualizing a non-torted ovary in the operating room. 😊
- **Rx:** So when in doubt, ask your friendly neighborhood gynecologist to urgently take your patient to the operating room.

## 3. Pelvic Inflammatory Disease

- **Dx:** Should be suspected in any sexually active female patient with lower abdominal pain without a clear alternative diagnosis.
  - Diagnosis is supported by systemic signs, vaginal discharge, cervical motion tenderness, or adnexal tenderness.
- **Rx:** PID can be treated as an outpatient unless the patient is pregnant, cannot tolerate PO, there is concern for tubulo-ovarian abscess, or fails outpatient management.
- **Rx:** Parenteral therapy for PID is doxycycline plus cefotetan, cofoxitin, or ceftriaxone + metronidazole.
  - When clinically improving for 24-48 hours, step down to doxycycline + metronidazole for 14 total days of therapy
- Note: Fitz-Hugh-Curtis syndrome (inflammation of liver capsule with associated adhesion formation) complicate about 10% of cases of PID

## 4. Sexually transmitted illnesses

- **Rx:** Ceftriaxone 500mg + doxycycline x 5-7 (?) days (Azithromycin is no longer first line for treatment of chlamydia urethritis / vulvovaginitis due to resistance. Azithromycin should be used in pregnant patients or those who are allergic to doxycycline.)
- **Rx:** For Trichomonas, metronidazole x 7 days is the treatment
- Test of cure is only indicated for pregnant patients. High risk patients do not require test of cure, but should be prescreened in 6 months.

## 5. Dysfunctional uterine bleeding

- "Normal" menstrual bleeding occurs every 21-45 (some sources say 24-38) days, vaginal bleeding lasts 2-7 days, and inter-cycle variability is less than 10 days
- If bleeding is prolonged with every cycle and/or patients hemoglobin is <8g/dl, bleeding disorder (most commonly Von Willebrand disease) should be suspected.
- Mild to moderate abnormal (anovulatory) bleeding (hemodynamically stable, Hgb > 10g/dl) can usually be managed outpatient.
- Severe abnormal (anovulatory) bleeding (Hgb < 10g/dl and/or hemodynamically unstable) requires inpatient management:
  - Thorough gynecologic, obstetric, and reproductive history
  - Physical exam including complete pelvic exam to assess for non-uterine sources of bleeding, infectious processes, uterine or adnexal abnormalities
  - CBC
  - Urine (and possibly serum) hCG

- Transvaginal US
- Possibly endometrial sampling
- Possible coags and VWD testing
- Treatment (assuming not pregnant):
  - Hemodynamically unstable
    - Uterine curettage (tamponade may be performed as temporizing measure)
  - Hemodynamically stable
    - High dose combination oral contraceptive (if no thrombosis risk)
    - e.g. A OCP containing 35mcg ethinyl estradiol/pill: 5 pills on day 1, tapering to 1 pill on day 5

#### 6. Testicular torsion

- **Dx:** Clinical presentation of unilateral testis with absent cremasteric reflex, high riding, swollen, exceedingly tender.
- **Dx:** Doppler ultrasound reveals absent blood flow to testis.
- **Rx:** Emergency detorsion by pediatric urologist (typically along with bilateral orchopexy)
- **Note:** torsion of the testicular appendix presents similarly, but blood flow to the testis is preserved and a "blue dot" (allegedly) can be visualized through the scrotal skin. This condition is managed supportively with pain control.

#### 7. Epididymitis

- **Dx:** Presentation is similar to testicular torsion, but both blood flow and cremasteric reflex are preserved.
- **Dx:** Elevation of the testis is alleged to alleviate symptoms.
- **Dx:** Two incidence peaks: < 2 and post-pubertal.
- **Dx:** Pyuria is usually but not always present.
- **Micro:** GC/Chlamydia (if sexually active), or typical UTI bugs (if not)
- **Rx:** Targeted toward suspected or known organisms (doxycycline + ceftriaxone for patient and partner(s) if sexually active, keflex or ceftriaxone if not)
- **Rx:** In prepubescent boys with negative UTI testing, supportive care alone should be sufficient.

#### 8. Orchitis

- **Dx:** In the absence of torsion or epididymitis, viral orchitis is a likely explanation of testicular pain. (Testicular lie should be symmetric, cremasteric reflex should be present, and blood flow should be intact.)
- **Rx:** Treatment is supportive.
- Rarely, brucellosis can be a cause of orchitis. (Diagnosis and management of brucellosis is beyond the scope of this review, but spoiler alert: the treatment is doxycycline for 6 weeks plus either rifampin for 6 weeks or gentamycin for the first 7-10 days)

## Part 8: Orthopedics

### 1. Compound fractures

- Compound (open) fractures require antibiotic prophylaxis. Selection of antibiotic depends on the Gustilo Andrsen classification:
  - Class 1 or 2: < 10 cm laceration, mild or moderate contamination, minimal periosteal stripping, and no skin grafting or vascular repair required.
    - Cefazolin. Add metronidazole if soil contamination
  - Class 3: > 10 cm, severe contamination, and/or incomplete soft tissue coverage (grafting require) and/or vascular repair required.
    - Cefazolin + gentamycin, or ceftriaxone. Add metronidazole if soil contamination.
    - If water contamination, use piperacillin-tazobactam for pseudomonas coverage.
      - And add doxycycline if salt water contamination. 🤖

### 2. Septic joint / osteomyelitis

- **Dx:** Kocher criteria can help distinguish septic arthritis from non-infectious causes of joint pain (such as post infectious tenosynovitis)



- Non-weight bearing (1 point)
- Fever > 38.5 (1 point)
- ESR > 40 (1 point)
- WBC > 12,000 (1 point)
- Score 0 = < 0.2 % risk, 1 = 3% risk, 2 = 40% risk, 3 = 93% risk, 4 = 99.6% risk
- **Rx:** Treatment options
  - Cefazolin is a good choice as it targets MSSA (by far the most common etiologic agent), as well as some of the more common other bugs (notably, K. King)
  - ADD clindamycin or vancomycin is concern for MRSA
  - ADD ceftriaxone AND vancomycin if ill appearing / unstable.
  - Also ADD ceftriaxone if under vaccinated against HiB or S. Pneumonia, or patient has sickle cell (covers salmonella), or patient is sexually active (covers N. Gonorrhoea).
  - There are numerous other options. Can tailor treatment according to gram stain and culture.
- Treatment for osteomyelitis is similar
  - In a hemodynamically stable patient, hold antibiotics until culture specimens obtained intraoperatively.

## Part 9: Rheumatology

### 1. Systemic Lupus Erythematosus

- **Dx:** Typically vague systemic symptoms at outset, including fatigue, fever, weight loss, malar rash, other rashes, even chronic thrombocytopenia.
  - There are diagnostic criteria (e.g. EULAR, SLICC) which are fairly extensive but assign points for Skin, Kidney, Mucous membrane, Joint, Neuro, Lab, and Neuropsychological symptoms.
  - For example, +ANA + Thrombocytopenia + Lupus Nephritis on biopsy would meet EULAR criteria.
  - So would +ANA + Malar Rash + Arthritis
- **Dx:** Initial presentation can occasionally be dramatic, such as:
  - MAS
  - Thromboembolic event
  - Neuropsychiatric illness
  - Renal failure
- **Dx:** Screening lab is ANA, followed by anti-dsDNA/Rho/Smith/La/RNP/cardioliipin, Lupus anticoagulant, direct Coomb's.
- **Rx:** Treatment consists of Hydroxychloroquine, steroids (try to minimize). Mycophenolate, and other immunomodulators.

### 2. Juvenile Idiopathy Arthritis

- **Dx:** Clinical presentation of 6 or more weeks of synovitis in one or more joints < 16 years of age and exclusion of other diagnoses.
  - There are 7 subtypes: Systemic, oligoarticular, polyarticular RF-, polyarticular RF+, psoriatic, enthesitis-related, undifferentiated.
  - Oligoarticular and systemic JIA peak at around 2 years of age
- **Dx:**ANA is NOT useful for diagnosing JIA, but can identify patients at risk of complications, particularly uveitis.
- **Dx:** Patient with oligoarticular JIA are particularly at risk for uveitis, even in the absence of joint symptoms.
  - Patients with JIA require regular slit lamp exams, as often as every 3 months depending on risk factors.
- **Rx:** Treatment starts with NSAIDs (and possibly intrarticular steroids), followed by disease modifying drugs (most commonly methotrexate).
- **Rx:** Steroids can be used for flares, but goal is to minimize steroid use.

### 3. Reactive arthritis

- **Dx:** Clinical constellation of urethritis, conjunctivitis, arthritis
- **Rx:** Commonly a result of S. Pyogenes infection, but other organisms can also be causative, notably GI bugs (Yersinia, salmonella,

shigella, Giardia, C. Diff, campylobacter), chlamydia, and Lyme disease.

#### 4. Kawasaki disease

- I think we are pretty familiar with this thoroughly protocolized condition so I will skip this topic for now.

#### 5. IgA vasculitis (Henoch-Schönlein Purpura)

- "Immune mediated leukocytoclastic vasculitis with neutrophil invasion and IgA deposition"
- **Dx:** Clinical features in decreasing order of prevalence: skin lesions, joint symptoms (arthritis/periartthritis), GI symptoms (pain, occult bleeding, rarely overt bleeding), IgA mediated nephropathy (almost always self limited), orchitis, pulmonary hemorrhage.
- **Rx:** Treatment is typically supportive with the following caveats:
  - Associated hematuria/nephritis may require consultation of pediatric nephrologist.
  - Severe abdominal pain is an indication for steroids, in which case a long taper (4+ weeks) should be prescribed.
  - Arthritis can usually be managed with NSAIDs unless there is active GI bleeding or evidence of glomerulonephritis.
  - Bleeding is rare, but can occur anywhere (including CNS)
- **Rx:** Assuming the patient does not have hematuria at discharge (in which case nephrology follow up would be indicated), regular screening for hematuria and hypertension is indicated. One possible regimen is: weekly for two months, monthly for 6 months, every other month for 6 more months, then annually.

#### 6. Behçet disease

- **Dx:** Clinical presentation of recurrent oral ulcers, with or without genital and corneal ulcers.
  - May progress with worsening vasculitis affecting other mucous membranes, joints, even CNS.
- **Rx:** Steroids. Colchicine for arthritis. May require other immunomodulators (azathioprine, etc.)

#### 7. Macrophage activation syndrome

- MAS is essentially hemophagocytic lymphohistiocytosis in the context of an underlying rheumatologist disease.
- Clinical presentation is one of marked systemic inflammation including fever, cytopenias, elevated ferritin and triglycerides, splenomegaly, elevated CD25.
- Unlike other forms of HLH, dexamethasone alone (to treat underlying rheumatologist disease) may be sufficient. If disease is severe or rapidly progressing, the usual HLH94 protocol (dexamethasone, etoposide, cyclosporine,  $\pm$  intrathecal methotrexate) can be used, although newer clinical trial protocols are becoming quite prevalent.

## Part 10: Endocrine

### 1. Type I Diabetes

- **Dx:** Typically present with hyperglycemia, abdominal pain, polyuria, polydipsia, weight loss
  - Most commonly present with out acidosis, but DKA upon presentation is also quite common
- **Dx:** Formal criteria are fasting glucose > 126 on **more than one occasion** (or random > 200). HgbA1C > 6.5% is also suggestive (confirm with fasting glucose).
- Physical exam findings do not definitively differentiate between Type I and Type II DM.
- Pancreatic autoantibodies or insulin antibodies are indicative of Type I DM
  - More antibodies = increased risk of development of Type I DM, but not severity of Type I DM
- **Treatment of DKA**
  - Goal is to rectify acidosis, dehydration, hypophosphatemia, and (effective) hypokalemia.
  - First step is to begin to restore intravascular volume. How to do this while minimizing risk of cerebral edema (due to rapid osmolar shifts) is a topic that remains controversial.
    - An **initial 10ml/kg fluid bolus** of normal saline is consistent with most guidelines.
    - Replacement of remainder fluid deficit (which can be assumed to be about 6-8% of body weight, typically at the higher end of that spectrum with new onset diabetes or severe acidosis) can be achieved over subsequent 48 hours with either NS or 1/2NS
      - The latest PECARN data suggests that half of this deficit can actually be safely replace over 12 hours, and the other

half over the following 24 hours.

- These fluids are in **addition** to maintenance fluid requirements
- One hour after initial fluid bolus, **insulin drip** can be started at 0.1U/kg to start to correct acidosis.
- Add dextrose to fluid once glucose < 300g/dl to keep glucose between 200 and 300 until gap closes. (This can be achieved by titrating D10NS and NS along with hourly glucose checks. Both bags should contain KPhos and KCl once potassium is nearing the normal range--see below).
- Phosphorous should be replaced, and **potassium** should be replaced once <4.5 (NOTE: other experts recommend replacement once potassium < 6, and some stay to start when the patient starts urinating) (since profound intracellular shifts will occur as pH corrects). Addition of 40meq/L of a combination of KPhos and KCl (or K acetate) will achieve these replacements.
- Monitor Glucose (hourly), Sodium, Potassium, pH, Bicarb (q 2 hours), Phos, Calcium, Magnesium (q 4-6 hours) throughout treatment.
- Wean to subcutaneous insulin when bicarb > 17.
- Readiness for discharge
  - Once patient is established on subcutaneous insulin regimen and tolerating PO, they can be discharged once initial education is complete
  - Initial education focuses on survival skills (glucose monitoring and insulin administration, carb counting, symptoms of and response to hypo and hyperglycemia, ketonuria testing)
- As of 2020, HgbA1C goal is 7.0%, perhaps higher (7.5%) if patient has barriers to recognizing and treating hypoglycemia or history of severe hypoglycemia.

## 2. HHS

- Same management?
- Cerebral edema?
- (Pseudohyponatremia)

## 3. Hyperglycemia in acute illness

- Elevated blood sugar during hospitalization is very common, presumably due to the anti-insulin effects of cortisol.
- Sub-cutaneous insulin should be given if blood sugar > 200 in the context of critical illness (but make sure the patient is not in fact diabetic, in which case you would manage accordingly)
- Insulin drip should be initiated if the patient becomes ketotic.

## 4. Thyroid disorders

DiGeorge

Poor tone: Trisomy 21, Prader Willi, Beckwith Wiedeman (?) -- BW also omphalocele

Screen : TSH surge in first 12 hours, so wait 24 hours

- Hypothyroidism
  - Causes include congenital hypothyroidism, hypothyroid phase of chronic autoimmune thyroiditis, congenital or acquired pituitary dysfunction (resulting in low TSH), or thyroid ablation (e.g. as treatment for Grave's disease).
    - "Sick euthyroid" -- low TSH, low T3, and normal to low free T4 in an acutely ill patient.
    - Iodine deficiency is another cause, though very rare in the US.
  - **Dx:** Clinical presentation of congenital hypothyroidism: many are asymptomatic, otherwise listless, poor feeding, coarse facies, umbilical hernia, large fontanelles.
  - **Dx:** Elevated TSH, low free T4 (unless central cause, in which case TSH may be low)
  - **Dx:** Thyroglobulin (TGO) and thyroid peroxidase (TPO) antibodies will be positive in cases of acquired (autoimmune) thyroid disease.
  - The most common cause of congenital hypothyroidism is thyroid dysgenesis
    - Congenital central hypothyroidism, while it does occur, is exceedingly rare and would typically present in the context of other pituitary disorders, although it may be isolated if related, for example, to a TSH gene mutation.
  - **Dx:** Clinical presentation of acquired hypothyroidism: fatigue, impaired growth

- **Rx:** Treatment: levothyroxine
  - Prompt initiation of treatment in hypothyroid infants is essential (do not delay for completion of diagnostic workup)
  - Sick euthyroid syndrome, which may feature mild depression of free T4, typically requires no treatment
- Hyperthyroidism
  - **Dx:** Causes include Graves disease (neonatal or acquired), hyperthyroid phase of chronic autoimmune thyroiditis
  - **Dx:** Clinical presentation of neonatal Grave's: irritable, not sleeping well, SGA, flushed.
    - **Note:** Mother may be hypothyroid (and taking levothyroxine) due to thyroid ablation, but will still have stimulatory antibodies in her circulation that cross the placenta.
    - Only a small minority of infants born to mothers with Grave's disease will develop neonatal graves.
  - **Dx:** Clinical presentation of acquired Grave's disease: tachycardia, weight loss, emesis, tremor.
  - **Dx:** For acquired Grave's disease, positive anti-TSHR or thyroid stimulating (TSI) antibodies, elevated free T4, undetectable TSH.
    - TPO and TGO antibodies may also be positive in Grave's disease. Positive TPO and TGO but negative TSHR and TSI may indicate hyperthyroid phase of chronic autoimmune thyroiditis.
  - **Rx:** Beta blockers, Methimazole (PTU is not used in children due to risk of hepatotoxicity, agranulocytosis, etc.), ablation (followed by levothyroxine replacement for resulting hypothyroidism).
- Solitary nodule
  - Chance of malignancy 25%
  - **Dx:** Diagnosis via fine needle aspiration

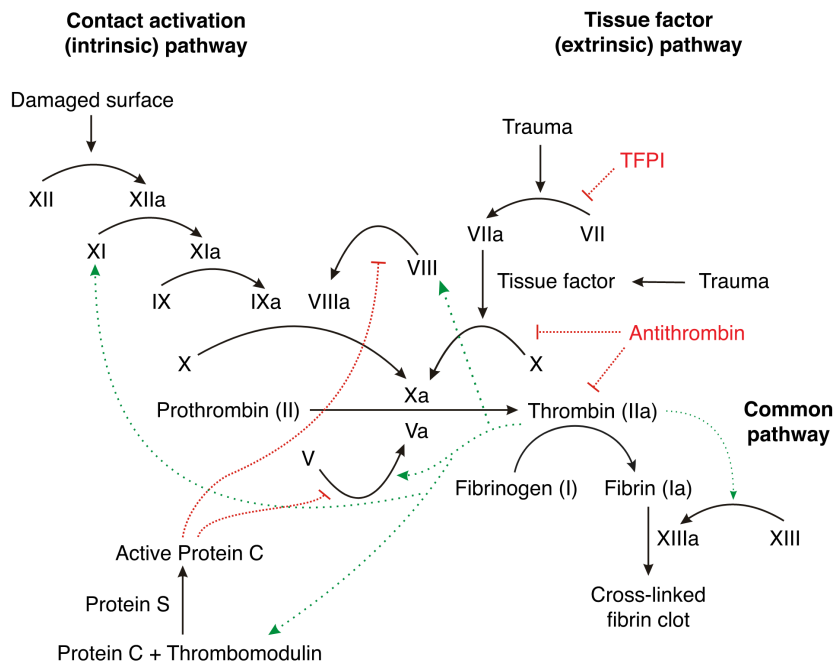
## 5. Electrolyte derangements

- Hyper/hyponatremia: these are common conditions that are not discussed here (yet)
  - Except to say, "Don't correct too fast!"
  - And, "Those fancy calculations never seem to achieve the clinical result I was looking for!"
- Cushing disease / syndrome
  - Increased glucocorticoid production due to genetic defect, ACTH secreting tumor (Cushing Disease), or exogenous steroid administration.
  - **Dx:** Hypoglycemia, obesity, hypertension, bone loss.
  - **Dx:** Diagnosis requires two of the following: abnormal late night serum cortisol, lack of ACTH suppression in overnight dexamethasone suppression test, and/or elevated 24 hour urine cortisol. (Confirm abnormal results by repeating test).
    - Note these tests are essentially meaningless in the face of exogenous steroid use. If your patient has Cushingoid symptoms and is taking steroids, the diagnosis of iatrogenic Cushing syndrome can be assumed.
  - **Rx:** Preferred treatment of Cushing disease is removal or ablation of the tumor.
  - **Rx:** Preferred treatment of Cushing syndrome is tapering the steroid where achievable
- Adrenal Insufficiency
  - **Dx:** Symptoms include fatigue, hypoglycemia, hypotension (the latter two are typically present only during stress, i.e. during adrenal crisis). In primary adrenal insufficiency, chronic ACTH elevation may result in increased skin pigmentation.
  - **Dx:** AM cortisol, ACTH. If cortisol low, perform ACTH (cosyntropin) stim test.
    - Inadequate response = adrenal insufficiency
    - Inadequate response + low or normal AM ACTH = secondary or tertiary adrenal insufficiency
      - Paradoxically, ACTH response is inadequate even in cases of secondary adrenal insufficiency (due to inadequate ACTH production by the pituitary). This is because the adrenal gland need regular ACTH stimulation to maintain normal synthetic function.
    - Inadequate response + elevated AM ACTH = primary adrenal insufficiency.
  - **Dx:** If above testing suggests secondary or tertiary adrenal insufficiency, CNS imaging is warranted.
  - **Dx:** Causes of primary adrenal insufficiency include congenital adrenal hyperplasia (see below), hemorrhage, trauma, and autoimmune destruction (Addison's disease).
  - **Rx:** Replace mineralocorticoid and glucocorticoid.

- **Rx:** In the event of adrenal crisis, draw stat glucose and electrolytes and administer a dextrose bolus (e.g. 5ml/kg of D10) and NS bolus(es) to correct blood sugar and maintain adequate blood pressure. Administer stress dosing hydrocortisone immediately (IM if necessary) and every 4 hours over subsequent 24 hours.
- Congenital adrenal hyperplasia
  - Vast majority of cases are due to 21-hydroxylase deficiency, resulting in decreased production of cortisol and aldosterone, with resulting increased in ACTH and androgens.
  - **Dx:** Symptoms include hypoglycemia, virilization, hyponatremia, hyperkalemia, hypotension.
    - In contrast, central causes of cortisol deficiency (ACTH deficiency related to pituitary injury, dysgenesis, or dysfunction) is characterized by hypoglycemia and hypotension, but not hyponatremia or hyperkalemia since the renin-angiotensin-aldosterone axis should still be in tact).
  - **Rx:** See adrenal insufficiency above.
- Hypercalcemia
  - 90% of cases are due to hyperparathyroidism (secondary to an adenoma) or malignancy increasing bone turn over.
  - **Dx:** Hypercalcemia may be associated with vague symptoms of mild neuropsychiatric symptoms, nausea, constipation. Diabetes insidious, kidney stones, and kidney dysfunction.
  - **Dx:** Severe hypercalcemia (>14mg/dL) can cause altered mental status and coma.
  - **Rx:** Aside from diagnostic workup to establish the cause, mild to moderate hypercalcemia (<14 mg/dl) can often be managed by hydration and avoidance of exacerbation medications (thiazides, lithium, calcium supplements, excessive vitamin D).
  - **Rx:** Patients with severe hypercalcemia (>14mg/dl) or significant symptoms require treatment.
    - IV fluid bolus first.
    - Calcitonin
    - Bisphosphanates

## Part 10: Hematology

### 1. Obligatory clotting cascade figure (Licensed under CC-SA license.)



### 2. Bleeding disorders

- Vitamin K deficiency

- Dx: Decreased activity of factors II, VII, IX, X ("1972")
- Dx: **Prolonged PT and INR**, if severe prolonged aPTT
- Rx: Vitamin K!
- Von Willebrand Disease (VWD)
  - Most common bleeding disorder
  - Autosomal recessive
    - Most commonly diagnosed in women as may lead to heavy menstrual bleeding.
  - **Dx:** Easy bruising, mucosal bleeding, major bleeding with injury, childbirth, or menstruation.
  - **Dx:** Prolonged aPTT, thrombocytopenia, anemia
  - **Dx:** VWF antigen, VWF activity, Factor VIII activity (VWF is a carrier for factor VIII)
  - **Rx:** DDAVP (if responsive) or VWF concentrate (if not) prophylaxis for procedures
  - **Rx:** For heavy menstrual bleeding, both hormonal contraceptives and DDAVP (or VWF concentrate) are options.
  - **Rx:** Heavy bleeding or major surgery generally require VWF concentrate as DDAVP response will not be strong enough.
  - **Rx:** Note that VWF factors rise during pregnancy. So pregnant women may or may not need VWF concentrate for delivery depending on levels, and vaginal delivery is not contraindicated in women with VWD.
- Factor V Leiden
  - Mutation in factor V results in resistance to the inhibitory effects of Protein C
  - **Dx:** Suspect if unprovoked clot or clot in unusual location or context
  - **Dx:** Genetic testing and/or activated protein C resistance assay.
  - **Dx:** In these patients, clotting cascades are normal (but cannot be shut off via Protein C). As a result, **coags are normal**.
  - **Rx:** Frequently none, but should be treated as high risk in circumstances where VTE prophylaxis would ordinarily be considered (surgery, immobilization, etc.)
- Hemophilia
  - Types A (factor VIII) and B (factor IX)
  - Hemophilia A is about 5 times more common.
  - Both demonstrate a wide range of severity related to individual variation in factor activity.
  - X-linked recessive, affect predominantly males
  - **Dx:** Typically presents between 1 year and 3 years of life (depending on severity). Suspect in cases of easy bruising, hemoarthrosis or excessive bleeding following procedure, particularly in young children.
  - **Dx: Prolonged aPTT** that corrects with mixing with donor blood (unlike patients with anticoagulants such as lupus anticoagulants)
    - Side note: lupus anticoagulants prolong aPTT in a test tube, but actually increases clotting in people.
  - **Rx:** Treatment options include prophylaxis vs. on-demand therapy, and decision of which to pursue is highly individualized depending on factor levels and patient preferences.
  - **Rx:** Treatment can be in the form of factor infusion or emicizumab (for Factor VIII deficiency only)
  - **Rx:** Factor levels rise with infusion and then fall following infusion. Thus, they should be emergently replaced whenever there is severe bleeding in a patient with hemophilia.
- 3. Sickle Cell Disease (SCD)
  - Hemoglobin genetics
    - HgbF :  $\alpha_2\gamma_2$
    - HgbA:  $\alpha_2\beta_2$
    - Sickle trait:  $\alpha_2\beta_1\beta^{glu7val}2_1$
    - HgbS:  $\alpha_2\beta^{glu7val}2_2$ 
      - Glutamic acid to valine mutation at amino acid 7
  - Acute Chest Syndrome (ACS)
    - **Dx:** Always maintain a high index of suspicion in patients with SCD presenting with pain, fever, or respiratory distress.
    - **Dx:** Obtain CXR. Any new radio density in combination with pain, fever, or respiratory distress should be assumed to be

ACS absent a clear alternative diagnosis.

- **Dx:** Pulmonary Embolism can present in a similar fashion and should be kept on the differential.
- **Rx:** Fluids, oxygen (maintain sats  $\geq 95\%$ ), pain control (typically with opioids, typically with PCA), ceftriaxone and azithromycin.
- **Rx: Transfusion.** Most patients admitted with ACS will require prompt transfusion. Goal is raising hemoglobin to 10-11 (and not higher).
- Sequestration Crisis
  - **Dx:** Splenomegaly and abrupt drop in hemoglobin
  - **Rx:** Fluid resuscitating and transfusion **but** bear in mind that fluid administration can liberate blood from the spleen. So great care must be taken to avoid raising the Hgb too high and causing hyperviscosity syndrome. So discuss with your friendly neighborhood hematologist.
- Pain crisis
  - **Dx:** Acute severe pain
  - **Rx:** Fluid rehydration and aggressive pain control, typically with opioids, typically with PCA.
  - All the while, be vigilant for ACS.

#### 4. Idiopathic Thrombocytopenia

- Grading of bleeding
  - Grade 0-2: No bleeding up to and including numerous petechiae and large bruises
  - Grade 3 low risk: Oral purport and epistaxis that stops quickly (<5 minutes)
  - Grade 3 high risk: Epistaxis that does not stop quickly, blood in stools or urine, menorrhagia.
  - Grade 4: Mucosal or internal bleeding needing "immediate medical attention" (?).
  - Grade 5: Life threatening bleeding including intracranial bleeding.
- Grade 0-3 & platelet count > 30,000: Watchful waiting (except in certain circumstances like planned surgery).
- Grade 0-3 & platelet count < 30,000: Still watchful waiting unless other bleeding disorder or antithrombotic medication use (or planned surgery as above).
- Grade 4: Treat with IVIG or steroids, or both. (And possibly other therapies as well.)
- Grade 5: Treat with IVIG and steroids and platelet transfusion. (And possibly other therapies as well.)
- **Note:** IVIG will raise platelet count faster, but steroids are less expensive and avoids the risk of anaphylaxis.
  - Neither guarantees permanent rise in platelets.

## Part 11: Newborns

Depending on your practice mix, this content is either second nature or a fuzzy distant memory with little in between.

### 1. NRP

- The Three Questions: Term? Good Tone? Breathing or Crying?
  - If any are no, we are off to the races!
- First minute: dry, stimulate, suction.
  - If apnea or gasping or HR < 100, PPV
- Next minute: Ensure adequate ventilation
  - If HR < 100 even with PPV then MR. SOPA
    - Mask adjustment, reposition (try again),
    - suction, open mouth (try again),
    - pressure increase (try again),
    - alternative airway
  - Recheck pulse in 15 seconds 🤔

- If HR > 100 then yay! Post resuscitation care.
  - If HR < 100 but > 60, keep working on ventilation.
  - If HR < 60, intubate, get access, then give epinephrine
    - New epinephrine dose: 0.02 **mg/kg** (0.2**ml/kg**) IV, 0.1**mg/kg** (1**ml/kg**) ETT.
  - Rinse and repeat.
2. Group B Strep sepsis risk mitigation
- All pregnant women should be screened for GBS by rectovaginal cultures between 36/0 and 38/0 weeks.
  - **Dx:** Adequate intrapartum prophylaxis is recommended for all women meeting any of the following criteria:
    - Prior infant affected by GBS
    - Positive urine culture at any time during pregnancy
    - Positive rectovaginal screen
    - Labor prior to 37/0 weeks.
    - Intrapartum fever, or rupture of membranes  $\geq$  18 hours.
    - NAAT screen positive (when available and performed).
    - GBS unknown and demonstrated colonization during prior pregnancy.
  - **Rx:** Acceptable antibiotics for intrapartum antibiotic prophylaxis (IAP)
    - Penicillin or ampicillin preferred,  $\geq$ 4 hours prior to deliver (= "**Adequate IAP**")
    - Cefazolin if low risk penicillin allergy,  $\geq$ 4 hours prior to deliver (= "**Adequate IAP**")
    - Clindamycin if high risk clindamycin (vancomycin if clindamycin resistance demonstrated).
    - **Note:** while clindamycin and vancomycin provide risk reduction for the infant, they are not considered adequate prophylaxis for the purposes of subsequently risk stratifying the infant.
  - **Dx/Rx:** Infant risk stratification and workup (for infants born  $\geq$ 35/0 weeks)
    - Obviously, a symptomatic infant needs full sepsis evaluation and empiric antibiotics (e.g. ampicillin and gentamicin).
    - Blood cultures and empiric antibiotics are indicated in any case of **intrapartum fever** (>38.0)
    - The only other situation in which something other than routine newborn care is indicated is if the adequate IAP was indicated for the mother, but not achieved (e.g., antibiotics given less than 4 hours prior to delivery, or antibiotics other than penicillin, ampicillin, or cefazolin given).
3. Screening and treatment for neonatal hypoglycemia
- Who needs screening?
    - Late preterm infants (34 - 36/6)
    - Large for gestational age infants (LGA) and small for gestational age infants (SGA)
    - Infants born to mothers with gestational diabetes.
    - Infants experiencing perinatal stress such as neonatal depression, polycythemia, etc.
  - **Dx/Rx:** Screening thresholds
    - Give IV glucose to any symptomatic infant with glucose < 40mg/dl
    - Feed within 1 hour of birth, then screen in 30 minutes. And then...
    - The First Four Hours
      - If initial screen < 25mg/dl → Feed and recheck in 1 hour 😞
      - If recheck is still <25, give IV dextrose. Otherwise feed and/or give IV dextrose based on your best judgement.
    - After the first four hours
      - Continue to feed every 2-3 hours, screening before each feeding
      - If any screen < 35mg/dl → IV dextrose
      - Otherwise feed and/or give IV dextrose based on your best judgement.
    - After 24 hours, treat any glucose < 50mg/dl
    - After 48 hours, treat any glucose < 60mg/dl
  - **Rx:** Dextrose dose
    - Initial bolus: 2ml/kg of D10



- Infusion: 5-8 mg/kg/minute
  - = 300 to 2400mg/kg/hour
  - = 3 to 4.8ml/kg/hr D10

4. **Rx: Neonatal fever in a well appearing infant.**

- 0-21 days: full work up, antibiotics
  - ampicillin/gentamycin or, if pleocytosis in CSF, ampicillin and ceftazidime, for CNS penetration)
  - HSV testing and acyclovir if risk factors (which include hypothermia, leukopenia, maternal fever 48 hours before to 48 hours after delivery, in addition to the obvious ones like fever, vesicles, etc.)
- 22-28 days: usually a full workup unless normal inflammatory markers (IMs).
- 29-60 days: at least UA, urine culture, blood culture, and inflammatory markers. And then...
  - If IMs normal you can observe--including at home if follow up is assured.
    - Treat with oral antibiotics if UA positive while awaiting culture results.
  - If inflammatory markers abnormal, you have the option to either:
    - Obtain CSF and if no pleocytosis observe (even at home?!?!)
    - Treat any UTI identified with UA either orally or IV.
    - If CSF has pleocytosis then treat as for meningitis
    - Or you can forego LP, give antibiotics, and observe in the hospital (or even at home?!?!?) while you await culture results.
- **Note:** infants with bronchiolitis are excluded from this pathway and bronchiolitis can safely be assumed to be the cause of fever in these infants if reasonably in the physician's judgement.
  - Concurrent meningitis or bacteremia seems to be exceedingly rare.
  - This does **NOT** apply to other viral illnesses, or just a positive viral test. It only applies to the clinical syndrome of bronchiolitis.
  - Practically speaking there is some lower age limit for this approach which I presume varies by physician.

## Part 12: Trauma

### 1. Burns

- Rule of 9's -- **only** count area affected by second degree and above (blistering, through the epidermis)
  - Head = 9%
  - Arms = 9% each
  - Legs = 9% each for the fronts, 9% each for the backs
  - Torso = 18% for front, 18% for back.
  - Only count significant, blistering burns
- Parkland formula
  - $4ml * weight * \% burn area$ 
    - again, only account second degree and above
    - Give half over first 8 hours
    - Give the rest over next 16 hours
  - To be given as Lactated Ringer's solution.

### 2. Drowning

- Mortality risk factors
  - Submersion > 5 minutes
  - Age > 14
  - Delay of > 10 minutes in receiving BLS
  - pH < 7.1
  - Comatose on presentation

- Need for CPR > 25 minutes
- **Rx:** A totally symptomatic individual with full recall of event can be observed at home.
- **Rx:** Individuals with any respiratory symptoms whatsoever or abnormal CXR should be observed at least 8 hours for evolution of symptoms.
- Hypothermia
  - **Rx:** Immediately institute external warming measures.
  - **Rx:** If temp < 32, give IV normal saline heated to 40-44C.
  - **Rx:** If that fails, or hypothermia is extreme (<28C), can lavage body cavities with warmed saline.

### 3. Ingestions

- Anticholinergics (diphenhydramine, glycopyrrolate, tricyclic antidepressants, etc.)
  - Mad as hatter, dry as a bone, blind as a bat (mydriasis), hot as a hare, red as a beet.
- Organophosphates (landscaping and farm chemicals)
  - DUMBBELLS (i.e., everything-rhea, mitosis)
- Acetaminophen pitfalls
  - Don't base any decision on a low or undetectable level less than 4 hours after ingestion.
  - A patient with elevated AST or ALT and **any** detectable acetaminophen level probably should be started on NAC (due to risk of being a late presenter with ongoing injury).
- Serotonin syndrome
  - May result from many drugs of abuse or overdose of medications
  - **Dx:** Clinical presentation of agitation, clonus, hyperthermia, tremor, hyperreflexia, tachycardia
  - **Rx:** First line treatment is ativan to normalize vitals and suppress tremor.
  - **Rx:** If that fails, cyproheptadine is second line.
  - **Rx:** Room temperature IV fluids can be used for temperatures > 41C.
  - (Don't confuse with malignant hyperthermia which is exceedingly rare and triggered by anesthesia.)
    - (Treatment for malignant hyperthermia is dantrolene)

## Part 13: Genetics

There are hundreds of genetic diseases. Nevertheless, I have encountered many practice questions that wanted me to know the inheritance of the more common ones. So here they are.

### 1. Major Autosomal Recessive Diseases

- Sickle cell disease
- Cystic Fibrosis
- Tay-Sachs disease
- Gaucher disease
- Von Willebrand Disease
- Congenital Adrenal Hyperplasia
- Most metabolic diseases, such as CPS deficiency (urea cycle defect)

### 2. Major autosomal dominant diseases

- Huntington's disease
- Achondroplasia
- Marfan syndrome
- Spinal Muscular Atrophy
- DiGeorge Syndrome

### 3. Major X-linked recessive diseases

- Duchenne muscular dystrophy
- X-linked agammaglobulinemia
- Glucose-6-phosphate dehydrogenase deficiency
- Wiskott-Aldrich syndrome

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