HANDBOOK

2022 Rapid Reference

Boston Children's Hospital
BOSTON MEDICAL CENTER
Boston Combined Residency Program

HARVARD MEDICAL SCHOOL

BOSTON UNIVERSITY SCHOOL of Medicine

Department of Pediatrics
Boston Children's Hospital
Boston Medical Center

Rapid Reference
First Edition
Last Revised 8/1/2022

Dear BCRP,

This is the first edition of the BCRP Rapid Reference Handbook. It is based upon contributions from generations of past residents- thank you to all.

This Rapid Reference is intended to be an on the fly reference for all residents to provide an approach to management of common conditions, increase clinical efficiency, and improve patient care. All clinical information contained herein is subject to change, particularly medication dosing depending on the clinical situation and medical advancements in the field. Please double check continuously up-dated, evidence based resources (clinical pathways, UpToDate, and Lexicomp) before making clinical decisions.

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Rapid Reference

Important numbers
Chief on call pager: 4891

BCH Ph #'s (617-355-)
- CODE BLUE: 5-5555
- ICU eval/STAT: p3825
- Operator: 0
- Pharmacy 5-6807
- Lab 5-6351
- Rads 5-6286
- Interpreters 5-7198
- CVS 617-975-3500

BMC Ph #'s (617-414-)
- CODE BLUE: 4-7777
- ICU support p6789
- Operator 8-7243
- Pharmacy 4-7687
- Lab 4-4060
- Rads 4-3892
- Interpreters 4-5549

BCH Rapid Responses			
Level of Support	**Number**	**Indication**	**Who Comes**
Medical Assist Team (MAT)	5-5555 and state "Medical Assist Team to floor # and room #"	Non-patient requires medical attention Only allowed to give juice, transport to ER Other needs requires code blue	Gen Peds Seniors, ED RN, COPP, Critical Care Transport Team, Security
ICU Eval	Page 3825 with your extension	Evaluate whether patient needs escalation of care (not time sensitive)	ICU Fellow
ICU STAT	5-5555 "ICU stat to floor # and room #"	When a patient needs to go to the ICU urgently (time sensitive)	ICU fellow, ICU charge RN, RT, Gen Peds seniors, DOM
Code Blue	5-5555 and state "Code Blue to floor # and room #"	CPR required for patients or non-patients	ICU fellow, attending, charge, RN Anesthesia, ED RN, RT, Pharmacist, Crit Care Transport team, COPP, security, Gen Peds Seniors and Social Worker

BMC Rapid Responses			
Level of Support	**Number**	**Indication**	**Who Comes**
Anesthesia Stat	4-7777	Emergent airway support	Anesthesia fellow
ICU Eval	4789	Evaluate whether patient needs escalation of care	PICU senior, PICU attending
Code Blue	4-7777	CPR required for patients or non-patients	PICU senior, PICU attending, PICU charge RN, RT

Normal Vital Signs by Age:

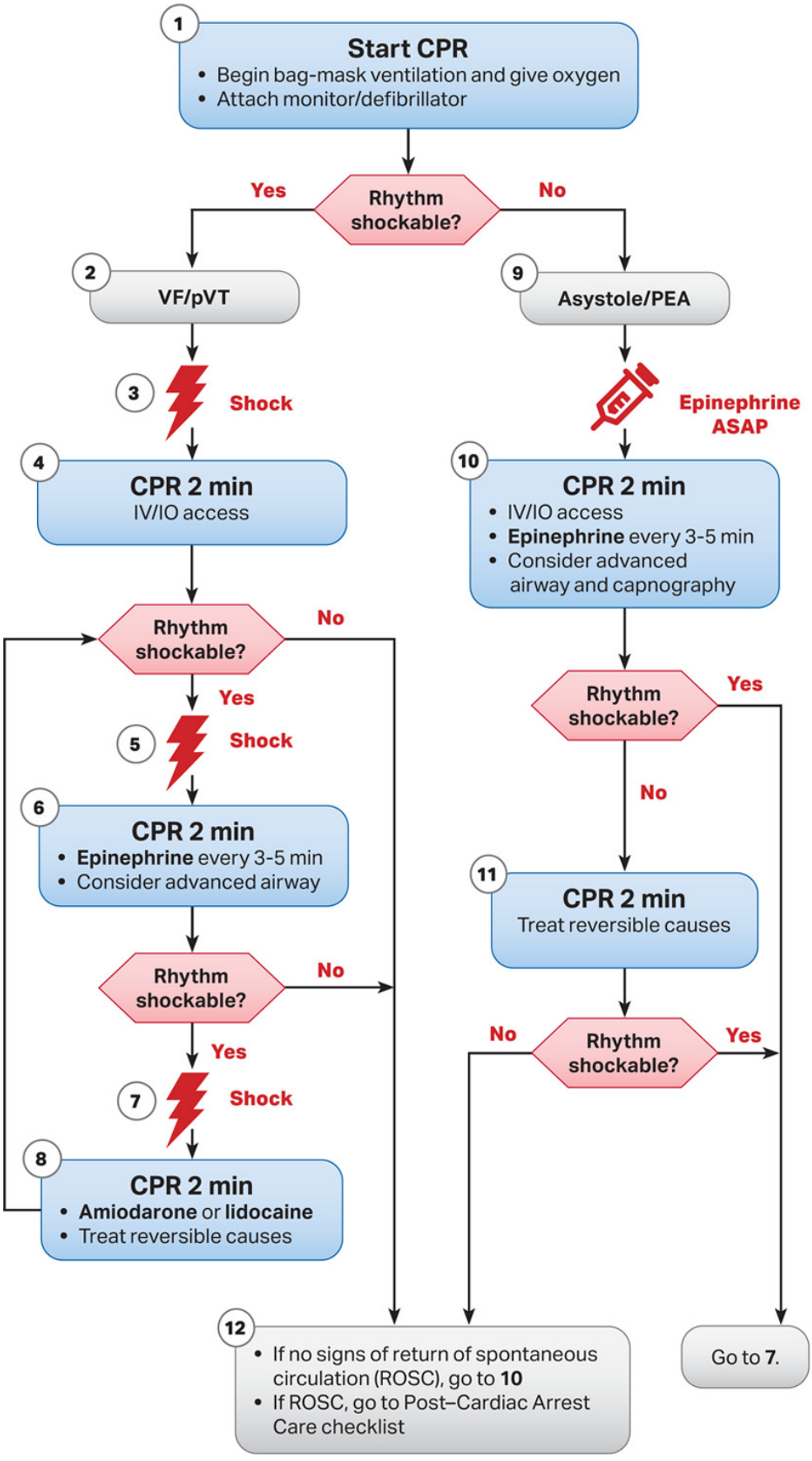
Age	HR (awake)	HR (asleep)	RR	SBP/DBP	MAP
Neonate (0-1m)	100-205	90-160	35-55	60-84 / 35-53	45-60
Infant (1-12m)	100-180	90-160	30-53	72-104 / 37-56	50-62
Toddler (1-2y)	98-140	80-120	22-37	86-106 / 42-63	49-62
Pre-schooler (3-5y)	80-120	65-100	20-28	89-112 / 46-72	58-69
School-aged (6-11y)	75-118	58-90	18-25	97-115 / 57-76	66-72
Adolescent (12-18y)	60-100	50-90	12-20	110-120 / 64-70	73-84

Emergent Medication Doses:

PALS			
Medication	**Indication**	**Dosage**	**Max Dose**
Adenosine	SVT	1st dose: 0.1 mg/kg IV 2nd dose: 0.2 mg/kg IV	1st dose: 6mg 2nd dose: 12 mg
Atropine	Symptomatic bradycardia	0.02 mg/kg IV may repeat dose once in 3-5 min	Single dose: 0.5 mg Total dose child: 1mg Total dose adol: 3 mg
Calcium Gluconate	Hypocalcemia, hyperkalemia, hypermagnesemia, calcium channel blocker overdose	20 mg/kg IV, repeat PRN	2000 mg
Dexamethasone	Croup	0.6 mg/kg PO/IM/IV	16 mg
Dextrose	Hypoglycemia	Rule of 50: 50/dextrose content = mL/kg dose	
Epinephrine (CPR)	Pulseless arrest, symptomatic bradycardia	IV: 0.01 mg/kg q3-5 min ET: 0.1 mg/kg q3-5min Strongly prefer IV	Single dose max IV: 1 mg
Epinephrine (shock)	Hypotensive shock	0.05-1 mcg/kg per min IV, start at 0.1	
Epinephrine (anaphylaxis)	Anaphylaxis	IM autoinjector 0.3 mg (pt > 25 kg) IM junior autoinjector 0.15 mg (pt 10-25kg) 0.01 mg/kg IM q15min	Single dose max IM: 0.5 mg Consider using longer needle if significant adipose tissue
Hydrocortisone	Adrenal Insufficiency	2 mg/kg IV bolus	100 mg
Prostaglandin E1 (alprostadil)	Ductal dependent congenital heart disease	0.05-0.1 mcg/kg per min IV	0.4 mcg/kg/min
Sodium Bicarbonate	Severe metabolic acidosis, hyperkalemia, sodium channel blocker overdose (TCA)	1-2 mEq/kg IV	

NRP		
Medication	**Indication**	**Dose**
IV Epinephrine	Persistent bradycardia (HR < 60) following PPV and chest compressions	0.2 mL/kg
Trach Epinephrine	Persistent bradycardia (HR <60) following PPV and chest compressions	1 mL/kg

Pediatric Cardiac Arrest Algorithm



CPR Quality

- Push hard (≥⅓ of anteroposterior diameter of chest) and fast (100-120/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Change compressor every 2 minutes, or sooner if fatigued
- If no advanced airway, 15:2 compression-ventilation ratio
- If advanced airway, provide continuous compressions and give a breath every 2-3 seconds

Shock Energy for Defibrillation

- First shock 2 J/kg
- Second shock 4 J/kg
- Subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose

Drug Therapy

- **Epinephrine IV/IO dose:** 0.01 mg/kg (0.1 mL/kg of the 0.1 mg/mL concentration). Max dose 1 mg. Repeat every 3-5 minutes. If no IV/IO access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of the 1 mg/mL concentration).
- **Amiodarone IV/IO dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 3 total doses for refractory VF/pulseless VT
  or
  Lidocaine IV/IO dose: Initial: 1 mg/kg loading dose

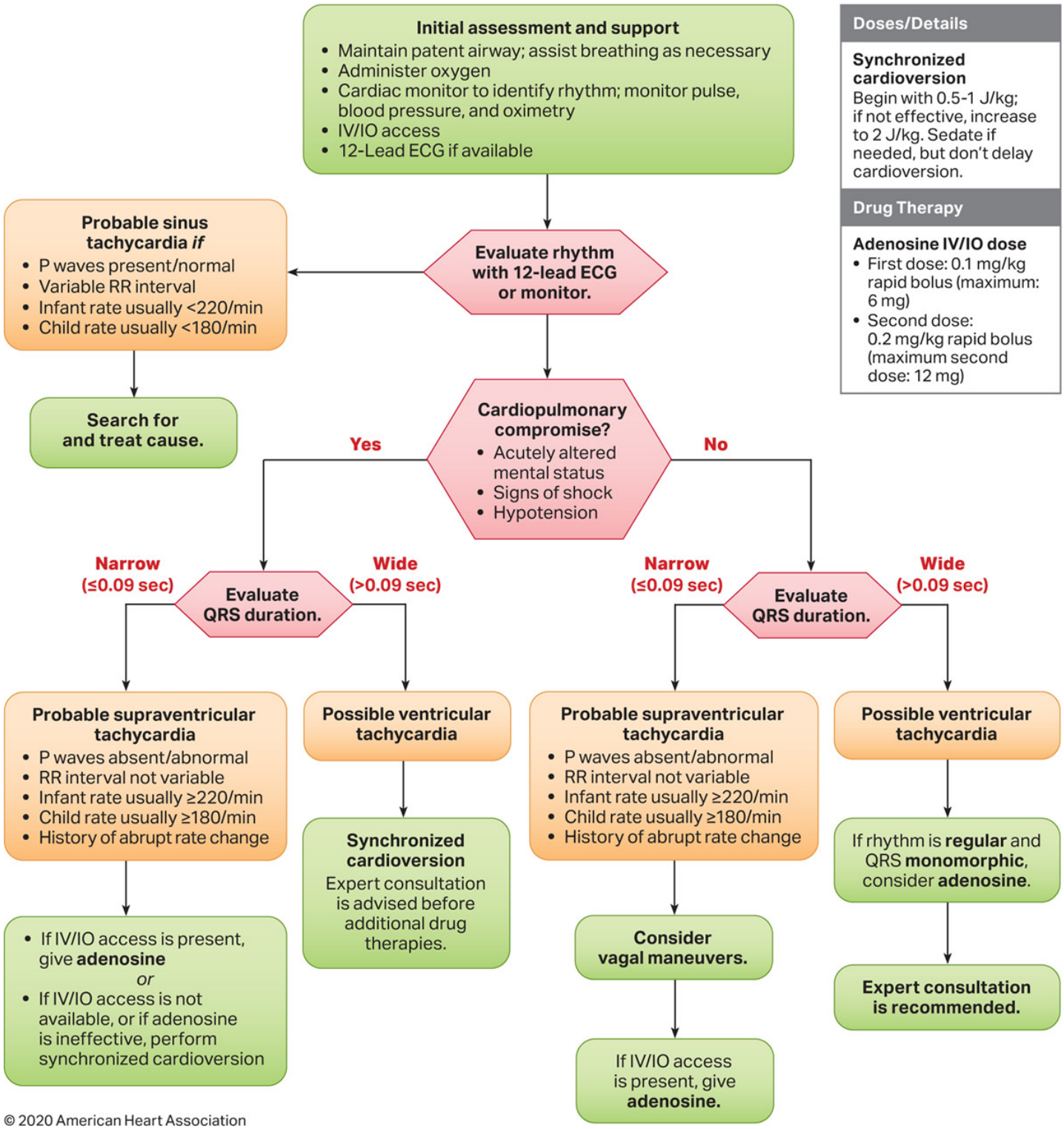
Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement

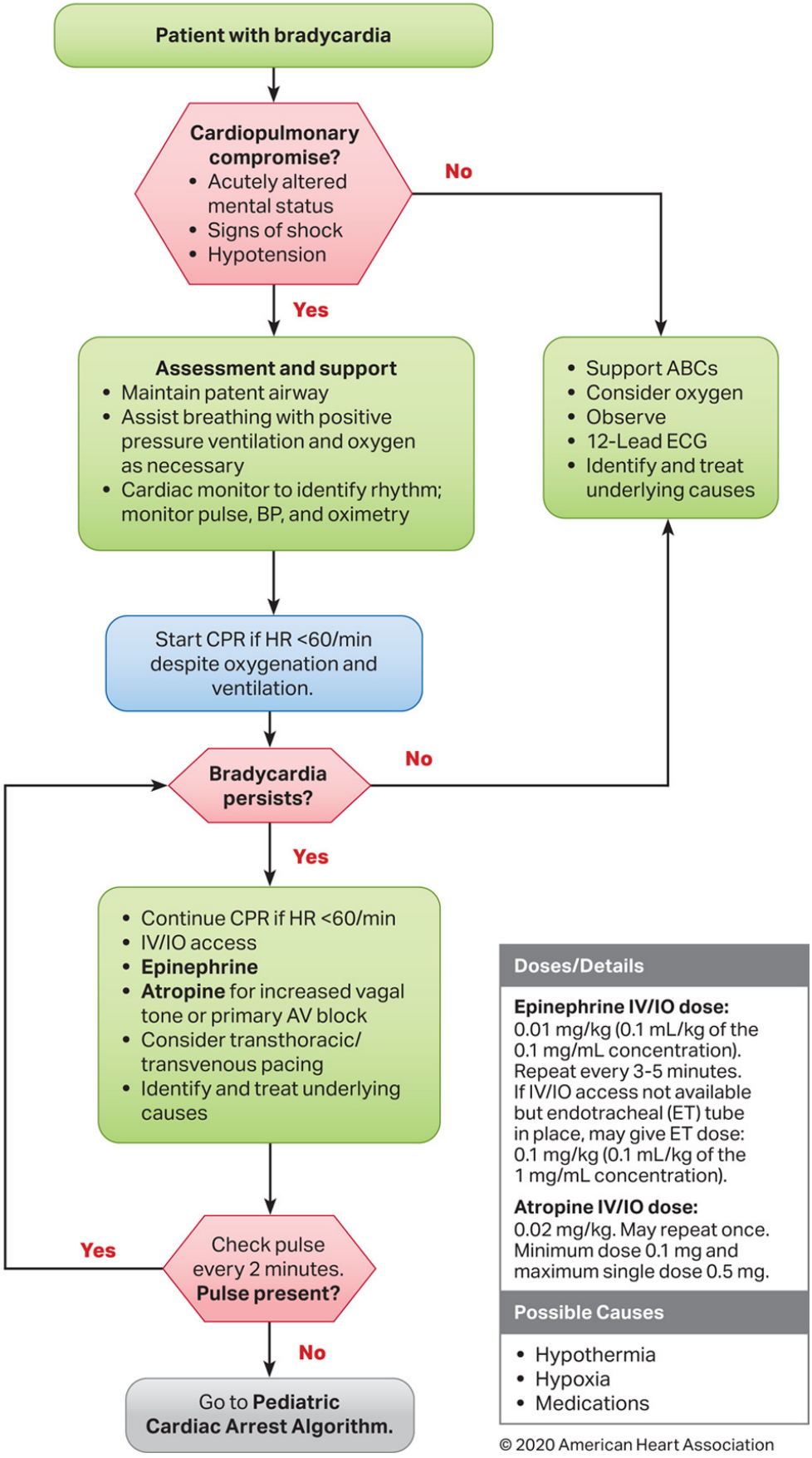
Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypoglycemia
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Pediatric Tachycardia With a Pulse Algorithm

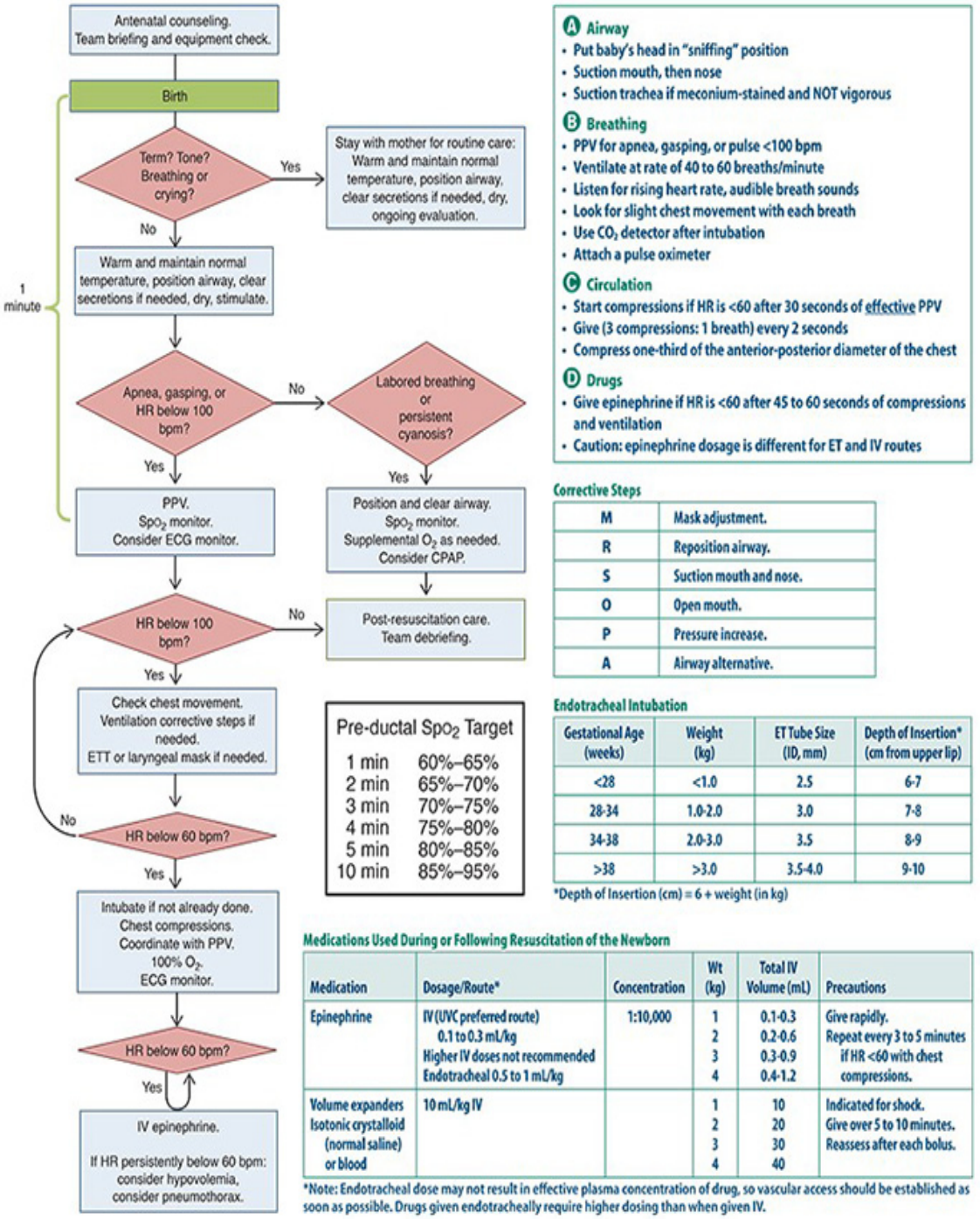


Pediatric Bradycardia With a Pulse Algorithm



Neonatal Resuscitation Program® - Reference Chart

The most important and effective action in neonatal resuscitation is ventilation of the baby's lungs.



A Airway
- Put baby's head in "sniffing" position
- Suction mouth, then nose
- Suction trachea if meconium-stained and NOT vigorous

B Breathing
- PPV for apnea, gasping, or pulse <100 bpm
- Ventilate at rate of 40 to 60 breaths/minute
- Listen for rising heart rate, audible breath sounds
- Look for slight chest movement with each breath
- Use CO_2 detector after intubation
- Attach a pulse oximeter

C Circulation
- Start compressions if HR is <60 after 30 seconds of effective PPV
- Give (3 compressions: 1 breath) every 2 seconds
- Compress one-third of the anterior-posterior diameter of the chest

D Drugs
- Give epinephrine if HR is <60 after 45 to 60 seconds of compressions and ventilation
- Caution: epinephrine dosage is different for ET and IV routes

Corrective Steps

M	Mask adjustment.
R	Reposition airway.
S	Suction mouth and nose.
O	Open mouth.
P	Pressure increase.
A	Airway alternative.

Pre-ductal Spo_2 Target

1 min	60%–65%
2 min	65%–70%
3 min	70%–75%
4 min	75%–80%
5 min	80%–85%
10 min	85%–95%

Endotracheal Intubation

Gestational Age (weeks)	Weight (kg)	ET Tube Size (ID, mm)	Depth of Insertion* (cm from upper lip)
<28	<1.0	2.5	6-7
28-34	1.0-2.0	3.0	7-8
34-38	2.0-3.0	3.5	8-9
>38	>3.0	3.5-4.0	9-10

*Depth of Insertion (cm) = 6 + weight (in kg)

Medications Used During or Following Resuscitation of the Newborn

Medication	Dosage/Route*	Concentration	Wt (kg)	Total IV Volume (mL)	Precautions
Epinephrine	IV (UVC preferred route) 0.1 to 0.3 mL/kg Higher IV doses not recommended Endotracheal 0.5 to 1 mL/kg	1:10,000	1 2 3 4	0.1-0.3 0.2-0.6 0.3-0.9 0.4-1.2	Give rapidly. Repeat every 3 to 5 minutes if HR <60 with chest compressions.
Volume expanders Isotonic crystalloid (normal saline) or blood	10 mL/kg IV		1 2 3 4	10 20 30 40	Indicated for shock. Give over 5 to 10 minutes. Reassess after each bolus.

*Note: Endotracheal dose may not result in effective plasma concentration of drug, so vascular access should be established as soon as possible. Drugs given endotracheally require higher dosing than when given IV.

Allergy/Immunology

Adverse Drug Reactions: Type A vs B ADRs	
Type A	Predictable, dose/duration dependent (ex overdose, SE, drug reactions).
Type B	Unpredictable hypersensitivity reactions (intolerance, immunologic)
I - Immediate (min-hours); IgE mediated	Anaphylaxis, angioedema, hives, hypotension, N/V/D
II - Delayed (variable); cytotoxic	Hemolysis, thrombocytopenia, neutropenia
III - Delayed weeks); Immune-complex	Serum sickness, arthus reaction, vasculitis
IV - Delayed (days to weeks); cell mediated	Contact dermatitis, SJS/TEN, DRESS

Desensitization: give increasing doses over hours → mast cells/basophils unreactive to Ag activation (Only for Type I HSRs) Result: Temporary tolerance → patient can receive the drug at usual intervals. When the drug is stopped, desensitization ends (d-wk). Avoid desensitization if an alternative abx exists for treatment.

Primary Immunodeficiencies

- Pathophys: genetic defects in adaptive (B or T cell) or innate (phagocytes, complement) immune systems lead to recurrent infections.
- Over 200 disorders: B defects (65%), combined T and B (15%), phagocytic (10%), T cell (5%), and complement (5%).
- Incidence 1:10K. Prevalence 1:2K
- Presentation can be nonspecific: poor growth, TFT, chronic diarrhea, rashes, recurrent infections, autoimmune diseases, FHx of consanguinity, fhx immunodeficiency
- Initial labs: CBCd, CMP, CRP, ESR< UA, albumin, Ig levels, IgG subclacsses, vaccine antibodies
- FOllow up studies: HIV testing, B and T cell subsets (aka flow cytometry), complement C3/C4/AH40, vaccine challenge (give vaccines and 6 week later measure titers), DHR flow assay (CGD), LAD testing.
- Treatment varies; ppx abx, IVIG, bone marrow transplant

Primary immunodeficiencies by Cell Type

B/T Cell Defects

- **DiGeorge** (no thymus, T cell issues), **SCID** (on NBS, dx absolute T cell count <300, abnormal T cell proliferation, OR presence of maternal T cells in circulation.), **CVID** (impaired B cell differentiation, poor vaccine response, low Ig levels), **Wiskott Aldrich** (WAS gene, thrombocytopenia, eczema, chronic otitis media/sinusitis), **Ataxia-Telangiectasia, Hyper IgM/ Hyper IgE, XLP, NEMO, DOCK8 def**
- **B:** Generally presents <12 mo old (3-6 mo, due to loss of maternal antibody), BACTERIAL infxn (sinusitis, otitis, PNA), recurrent abscesses, diarrhea/gastro, FTT, enteroviral meningoencephalitis if chronic. Bugs: Encapsulated: S. pneumo, HiB, Neisseria, Burkholderia, Klebsiella, Nocardia & Salmonella.+ S. aureus, pseudomonas. Can also have IgG subclass or selective IgA deficiency with nl B cell levels (i.e. class switch issue)
- **T:** Typically presents at birth/early infancy. Severe VIRAL, FUNGAL, bacterial, opportunistic, mucocutaneous candidiasis, warts/eczema, diarrhea, FTT. Bugs: HSV/VZV/CMV, Candida, PJP, Mycobact, S. typhii

Phagocytic Defects

- **Chediak Higashi** (CHS1, giant cell granules on smear), **Chronic Granulomatous Disease** (CGD, X-linked, abnormal DHR, subunit defect of protein complex), and **Lymphocyte Adhesion Def** (LAD, delayed cord separation, integrin defect). Presents in infancy, poor would healing, candidiasis, HSM, oral dz, lymphadenitis

Complement Defects

- Affecting the Classical, Lectin or Alternative pathways; C2 most common in Caucasians, C1-est involved in hereditary angioedema. P/W recurrent sinopulmonary infections, Neisseria (with C5-C9 def), autoimmune phenotype, meningitis; associated with atypical HUS

Less common are NK cell deficiencies (Herpesviral infections), Basophils and Eosinophils defects, and Neutrophil defects (ELANE mutation). Not mentioned here but can have Treg (IPEX, CD25 def, ALPS) and Th17 defects (Hyper IgE/STAT3 mutation)

Rheumatology

Common meds (route; mechanism; indications; common SE/PPX/Screens)

Small Molecule:
- Hydroxychloroquine: oral; alters lysosomal pH. Used in SLE, Sjogren's etc. Risk of retinopathy so baseline eye exam & monitoring; G6PD trigger.
- Azathioprine (AZA): oral; anti-metabolite. Used in SLE, JDM, vasculitis, autoimmune hepatitis. Risk of bruising, myelosuppression
- Methotrexate (MTX): oral vs. subQ weekly. DHFR inhibitor. Used in JIA/arthritis, JDM. Risk of transaminitis, nausea, stomatitis.
- Mycophenolate mofetil (MMF): oral; lymphocyte proliferation. RIsk of diarrhea.
- Cyclophosphamide: IV alkylating agent. Used for CNS vasculitis, JDM, Lupus nephritis. Causes immunosuppressive, alopecia, hemorrhagic cystitis (give with MESNA), later cancer.
- JAK inhibitors: tofacitinib, ruxolitinib, & baricitinib. PO. Blocks JAK-STAT receptor. Used in systemic JIA, macrophage activation syndrome, COVID PNA, JIA; risk of infection, transaminitis.

Biologics (all IV or subQ):
- Abatacept: CTLA-IgG. Promotes tolerance. Used in JIA, immune dysregulation
- Rituximab: anti-CD20 to clear B cells. Used in SLE. Risk of permanent hypogammaglobulinemia; check vaccine titers before
- Tocilizumab: anti-IL-6. Used in JIA, vasculitis. Increased infection risk; infusion reaction.
- TNF inhibitors: adalimumab, etanercept, & infliximab. Used in JIA, vasculitis, IBD; need TB test beforehand.
- Anakinra & canakinumab: anti-IL1. Used in systemic JIA, MAS, autoinflammatory disease. Risk of transaminitis.

Common Rheumatologic Diseases

Kawasaki's Disease (see BCH pathway) – medium vessel vasculitis
- Most common and has more classic presentation in ages 1-5 but higher risk of aneurysm in <6 months
- Presents with "CRASH and burn" (bilateral, non-exudative, limbal sparing), Conjunctivitis, Rash, Adenitis (unilateral >1.5cm), Strawberry tongue (or red, cracked lips), Hand/foot swelling + Fever (burn) at least 4 days
- Often irritable, tachycardic even when afebrile 2/2 myocardial inflammation
- Infants <6 months may have no physical criteria at all; low threshold for echo if persistently febrile
- A normal initial echocardiogram does not rule out KD; may not develop changes for weeks
- Eval: Complete (clinical) and Incomplete (clinical + labs); get a cardiac echo (coronary aneurysm risk, abnl= z-score >2.5) and baseline labs (CBC, CRP/ESR, Chem, LFTs, UA+ micro)
- TX:Treatment is to prevent coronary changes. IVIG (2g/kg; use Powerplan) + 10mg/kg ASA q6h; +/- repeat IVIG/steroid/additional therapy if abnormal echo or persistent fever 36 hours out from IVIG completion (discuss with rheum/KD team). D/C on low dose ASA when without fever and KD team deems ready

Juvenile Dermatomyositis (JDM)
- Rare, (1/1000,000) but seen and diagnosed at BCH; not associated with cancer unlike adult DM
- Proximal weakness plus pathognomonic rashes including Gottron's papules, heliotrope rash (also with peri-orbital swelling); note that rashes may be more subtle based on different skin tones
- Most dangerous clinical sequelae are ILD and GI bleeding from vascular inflammation in these organs
- Pulse steroids and methotrexate inpatient followed by oral steroids and MTX outpatient (may escalate therapy based on clinical severity); note that oral medications may be absorbed less well i/s/o GI inflammation at disease onset

Periodic Fever, Aphthous Stomatitis, Pharyngitis, Adenitis (PFAPA)
- Most common Periodic Fever Syndrome (PFS) in childhood; not due to a monogenic defect; clinical diagnosis
- "Clockwork" episodes ~every 4 weeks with the symptoms as indicated in the name (patients don't always have all)
- Steroids can be used as an abortive treatment, although this often increases the frequency of episodes
- Definitive treatment with tonsillectomy (only rheum diagnosis fixed with surgery!)

IgA Vasculitis ("Henoch Schonlein Purpura" (HSP)) – small vessel vasculitis
- Most common vasculitis in children; classically 2-3 weeks after URI or immunization
- Symptoms: palpable purpura, often lower extremities and buttocks; arthralgias & swelling in ankles/knees, abdominal pain (GI vasculitis), renal involvement (hematuria +/- HTN, often 1-2 weeks into clinical course)
- DX/Eval: Clinical: palpable purpura (w/o low plts or coag issues) + ≥1 of the other sx above. Get UA, CBC +/- abdominal US for intussusception. NO IgA level.
- TX: supportive; steroids generally reserved for patients with so much abdominal pain they require admission; the taper is very slow & steroids do not prevent renal disease; f/u with PCP with weekly UA + BP check

Multisystem inflammatory syndrome in children (MIS-C) – see ED pathway
- 2-8 weeks after primary COVID infection
- Fever for at least 24h + >1 system involvement (Neuro (AMS), GI (N/V/D), mucocutaneous (rash/conjunctivitis/oral mucosa changes), respiratory, cardiac (abnl echo, cardiac enzymes, EKG), shock
- Labs often show anemia, thrombocytopenia, high ANC, low ALC, elevated d-dimer, and elevated CRP (may not have all features but this would be the classic scenario)
- Echocardiogram to evaluate for myocarditis and coronary dilation
- TX: IVIG (2g/kg) + steroids (1mg/kg BID max 30mg BID); may need pressors for shock

Systemic Lupus Erythematosus

- Multiorgan systemic autoimmune disorder with markedly variable presentations/course
- Dying cells→ DNA/RNA→ Dendritic cell activation→ B & T cell activation→ autoantibodies and immune complexes; all steps feed into each other (not entirely linear)
- Under age 5 more concerning for possible monogenic etiology (e.g. complement deficiency)
- In adolescents F>M; more common in non-white patients but can still be seen in all race/ethnicities
- A diagnosis of lupus requires a positive ANA (usually high titer); h/w 20% of post-pubertal females have a positive ANA (usually low titer) and are totally healthy
- TX: all patients should be on hydroxychloroquine; many patients on chronic steroids; if mild symptoms (rash/arthritis), may add MTX or AZA; if mild-moderate kidney disease use mycophenolate mofetil; if moderate-severe kidney disease or CNS involvement (more likely to be inpatient), use cyclophosphamide +/- rituximab
- Neonatal lupus also exists (born to mom with anti-Ro/La), can have AV block (auto-ab to cardiac conduction system), photosensitive annular rash, thrombocytopenia, transaminitis, and thyroid issues; aside from heart block, other features self-resolve with antibody clearance in first 6-9 months

SLE Entry criterion: ANA titer ≥1:80 on HEp-2 cells or an equivalent positive test

- If absent, do not classify as SLE
- If present, apply additive criteria

Additive Criteria

- Do not count a criterion if there is a more likely explanation other than SLE
- Occurence of a criterion on at least one occasion is sufficient
- SLE classification requires at least one clinical criteria and ≥10 points
- Criteria need not occur simultaneously
- W/n each domain, only the highest weighted criterion is counted toward the total scores
  - Constitutional: fever 2
  - Hematologic
    - Leukopenia 3
    - THrombocytopenia 4
    - Autoimmune Hemolysis 4
  - Neuropsychiatric
    - Delirium 2
    - Psychosis 3
    - Seizures 5
  - Mucocutaneous
    - Non-scarring alopecia 2
    - Oral ulcers 2
    - Subacute cutaneous or discoid 4
    - Acute cutaneous lupus 6
  - Seorsal
    - Pleural/pericardial effusion 5
    - Acute pericarditis 6
  - Musculoskeletal
    - Joint 6
  - Renal
    - Proteinuria >0.5g/24h 4
    - Renal bx class II or V nephritis 8
    - Renal bx class III or IV nephritis 10
  - Antiphospholipid Antibodies 2
    - Anti-cardiolipin antibodies or anti-Beta2GP1 antibodies or lupus anticoagulant
  - Complement proteins
    - Low C3 or low C4 3
    - Low C3 AND low C4 4
  - SLE specific Antibodies 6
    - anti-dsDNA antibody or anti-smith antibody

Total score: classic as SLE with a score of 10 or more if entry criterion fulfilled.

Approach to Joint Disease

Inflammatory vs non-inflammatory	**Inflammatory:** swollen, warm, tender joint, worse w/ prolonged inactivity, morning stiffness, decreased ROM, improves w/ NSAIDS/steroids/movements **Noninflammatory/mechanical:** due to injury/degeneration. Pain with motion or activity, improvement with rest, can have mild swelling, effusion or bony abnormalities.
	Central Sensitization/Pain amplification: pain that is constant but often worse at night when trying to sleep. Not stiff. Sometimes has neuropathic description (burning, stabbing) or allodynia. Hypermobility is a risk factor as chronic microtrauma can cause neurons to require so they fire even in the absence of damage. Anxiety/depression worsening experience of pain. Treated with PT and CBT.
Distribution	Monoarticular, oligoarticular (<2), Polyarticular (>4)
Joint involvement	Peripheral vs axial; large vs small; symmetric vs asymmetric
Timing	Acute vs chronic (>2 months); episodic vs constant; migratory vs localized
Precipitation	Infection (GI/Gu), use, medications/diet, unprotected sex, IV drugs, family history
Useful associations	Monoarthritis of the knee- think Lyme disease Migratory polyarthralgias- consider onc, but also seen in acute rheumatic fever Hip effusion after viral infection- think transient synovitis Painful polyarthritis after recent infection- think reactive arthritis Morning stiffness for several weeks- think JIA

Juvenile Idiopathic Arthritis (JIA)

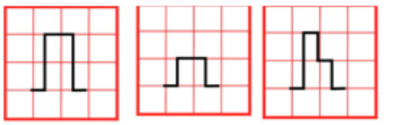
- autoimmune arthritis of childhood; requires symptoms for six weeks in the absence of alternative diagnosis; most often treated outpatient, but systemic JIA is a unique illness that is likely to be encountered inpatient.
- An ANA is not part of the diagnosis of JIA; it is used to assess risk for uveitis; ANA+ leads to more frequent screening for uveitis; JIA is a clinical diagnosis and except for the RF designation for subtype, no lab test is used in diagnosis
- Systemic JIA classically presents with quotidian or double quotidian fevers with rash that worsens during fever; the rash may be pruritic; patients may have anemia, thrombocytosis, and other laboratory features of chronic inflammation; it is not uncommon for arthritis to be absent in the first few months (name is thus confusing), although some patients do have this feature at onset; important because has a high rate of macrophage activation syndrome; treated with IL-1 or IL-6 inhibition
- Macrophage Activation Syndrome (MAS): often thought of as “secondary HLH”. multisystem inflammatory process (cytokine storm); can be complication of systemic JIA, SLE, KD, viral illnesses (e.g. EBV), or cancer. Clinical features may include fever, erythrodermic rash, hepatosplenomegaly, and organ dysfunction; laboratory features may show pancytopenia, transaminitis, and hyperferritinemia; look for atypically low ESR due to consumption of fibrinogen. See MAS/HLH pathway.

JIA subtypes:

- **Systemic:** age 1-5 years. Polyarticular pattern. Associated with fever, rash, pericarditis. Treated with IL1- and IL6 inhibitors. Can be associated with MAS.
- **Oligo** (<5 joints in the first 6 months): ages 1.5-4 years. Knee, ankle, finger pattern. Associated with uveitis. Treat with NSAIDs, intra-articular steroids, MTX. TNFi if incomplete response.
- Poly RF (-): ages 2-4 years and 10-14 years. Symmetrical and Asymmetric; small and large joints. Associated with uveitis. Treat with MTX, TNFi.
- Poly RF (+): ages 9-12 years. Symmetric polyarthritis pattern. Associated with rheumatoid nodules, fever. Treat with MTX, TNFi, rituximab.
- Psoriatic: ages 2-4 years and 9-11 years. Asymmetric, small to medium joints pattern. Associated with dactylitis, uveitis, psoriasis. Treat with NSAID, steroids, MTX, TNFi, ustekinumab
- Enthesitits: ages 9-12 years. Lower limb and axial pattern. Associated with acute anterior uveitis, IBD. treat with NSAIDs, steroids, MTX, TNFi.

Cardiology

EKG: Standardizations and Dimensions:


Full, half and mixed standards

Paper speed: Standard 25mm/s → Small box = 0.04s, Big box = 0.2s
Standardization marker:
- 2 big boxes tall = "full standard" and 10 mm = 1 mV
- 1 big box tall = "half standard" and 5 mm = 1 mV
- "Chair" = "mixed standard", limb leads in full standard and precordial are in half standard

EKG Reading:
Evaluate: Ventricular Rate (300/# large boxes), Rhythm (1:1 P:QRS), axis (I, aVF)

PR Interval	**Short:** pre-excitation, WPW; **Long:** AV block
QRS Interval	**Long:** Ventricular arrhythmia, pre-excitation, conduction delay, BBB
QT interval	**QTc** = QT/√RR; **Long**: congenital, hypo-K/Mg/Ca, hypothermia, iatrogenic
Q wave	**Normal:** Small, inferior/L leads; **Abnormal**: >0.04s, amplitude > 25% QRS, V1/V2
ST segment	**Abnormal:** >1mm in limb, >2mm in precordial; territorial distribution
T wave	**Inverted**: Normal age 10d-adol in V1-V3; always abnormal in V5, V6 (strain)
U wave	**Normal:** <25% amp of T, middle isoelectric segment, upright; **Abnormal**: Hypo-K
Atrial size	**RAE**: Height > 2.5mm in II; **LAE:** Duration > 100ms in II
LV/RV size	**LVH**: R wave large in I, II, aVL, V5, V6; **RVH**: R wave large in aVR, V1, V2
Strain	QRS-T axis difference > 90%

Normal EKG Values by Age:

PR Interval (ms)	**<1 mo**, 80-120; **2mo-1y**, 80-140; **1-5y**, 100-160; **6y-12y**, 110-180ms
QRS Duration (ms)	**<1 mo,** <65; **1mo-8y**, <80; **8-16y**, <100
QRS Axis (degrees)	**<1 mo,** over +120°; **6mo-1y**, under +120°; **>1y**, under +100°

Source: Galli and Danzi, A Guide to Neonatal and Pediatric ECGs, 2013

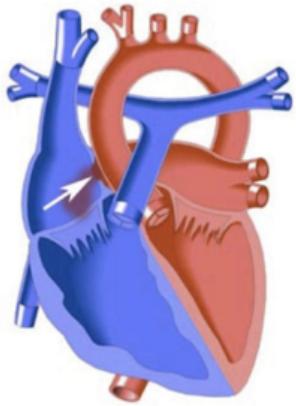
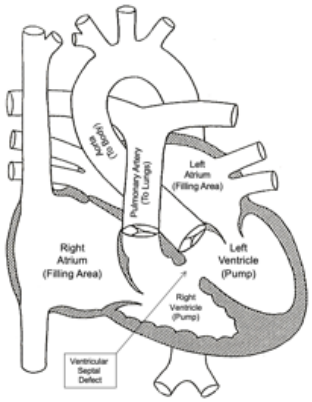
Cardiac/Heart Failure Pharmacology:

HF Stage	Treatment
A: Nl fxn, size	None
B: Abnl struct, no sx	ACE inhibitor
C: Sx of HF	ACE inhibitor and aldo antagonist; diuretic per volume status
D: End-stage HF	IV diuretics/inotropes; may need PPV, CRT, MCS

Drug	Receptor Activity				Clinical Effects
	Alpha 1	Beta 1	Beta 2	Dopa	
Phenylephrine	+++	0	0	0	SVR↑↑, CO↔/↑
Norepinephrine	+++	++	0	0	SVR↑↑, CO↔/↑
Epinephrine	+++	+++	++	0	CO↑↑, SVR↓ at low dose but ↑ at high dose
Dopamine (mcg/kg/min)					
0.5 to 2	0	+	0	++	CO↑
5 to 10	+	++	0	++	CO↑, SVR↑
10 to 20	++	++	0	++	SVR ↑↑
Dobutamine	0/+	+++	++	0	CO↑, SVR↓
Isoprotenerol	0	+++	+++	0	CO↑, SVR↓

Congenital Heart Disease

Acyanotic Heart Defects

<table>
<tr>
<td>  </td>
<td>Atrial Septal Defect: ASD
Problem: volume overload
SxS: asymptomatic vs. poor growth vs. CHF
PE: fixed and widely split S2. SEM from increased flow across PV
Tx: Closure if symptomatic or Qp:Qs >1.5 : 1</td>
<td> 
</td>
<td>Ventricular Septal Defect:
Problem: Volume overload +/- R pressure overload
Sxs: asymptomatic to sweating and tiring with feeds, tachypnea, poor growth
PE: Early or holosystolic regurgitant type murmur +/- left-sided heave
Tx: Monitor for spontaneous closure +/- closure</td>
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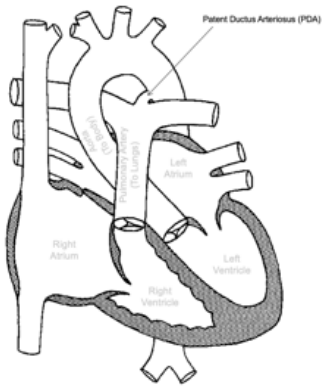
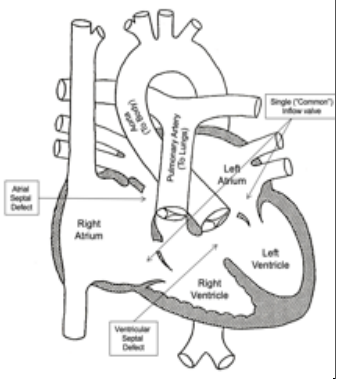
<td> 
</td>
<td>Patent Ductus Arteriosus:
Problem: volume overload
SxS: Asymptomatic vs. respiratory distress, feeding fatigue, poor growth
PE: continuous, machine-like murmur at LUSB, wide pulse pressure, bounding pulses
Tx: Indomethacin/Tylenol vs. ligation</td>
<td> 
</td>
<td>Complete Common AV canal Defect:
Problem: volume overload
Components: primum ASD, AV canal type VSD, AV valve defects
SxS: poor growth, sweating and fatigue with feeds
PE: murmurs of ASD, VSD, and MR +/- gallop
Tx: surgery</td>
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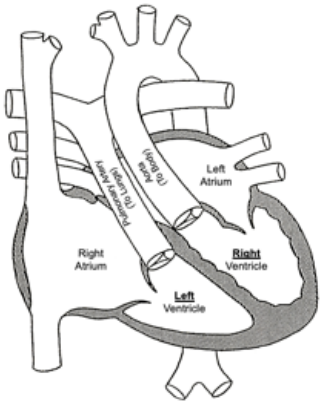
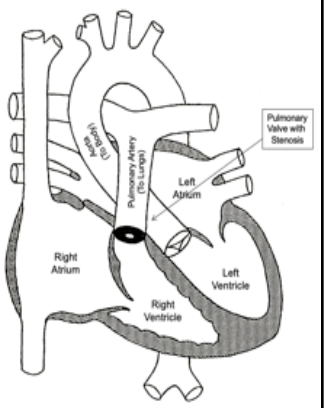
<td> 
</td>
<td>L Transposition of the Great Arteries: (Congenitally Corrected)
Problem: switched RV and LV (often associated with other cardiac defects)
SxS: R heart failure in early adulthood
PE: loud S2 +/- symptoms associated with other defects
Tx: Double switch (arterial and atrial level switch)</td>
<td> 
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<td>Valvar Pulmonary Stenosis:
Problem: pressure overload
SxS: asymptomatic vs. RV failure +/- cyanosis
PE: SEM at LUSB, ejection click +/- TR murmur
Tx: if critical, start PGE. Ultimately, balloon valvuloplasty vs. surgery</td>
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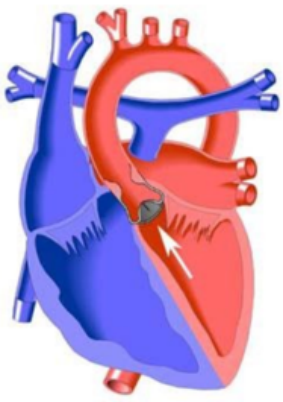
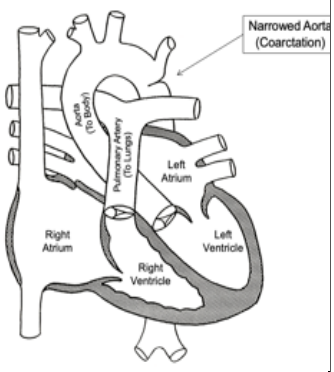
<td>  </td>
<td>Valvar Aortic Stenosis
Problem: pressure overload
SxS: Asymptomatic vs. CHF vs. cardiogenic shock
PE: Harsh SEM at base radiating to neck, ejection click, LV heave
Tx: if critical, start PGE. Balloon valvuloplasty vs. surgical repair/replacement</td>
<td> 
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<td>Coarctation of the Aorta
Problem: Pressure overload
SxS: poor growth, sweating and fatigue with feeds vs. cardiogenic shock
PE: UE HTN with drop in LE BPs. SEM at LUSB radiating to back. Brachiofemoral delay +/- decreased/absent femoral pulses
Tx: PGE if evidence of cardiogenic shock; surgical excision vs. balloon dilation and stenting</td>
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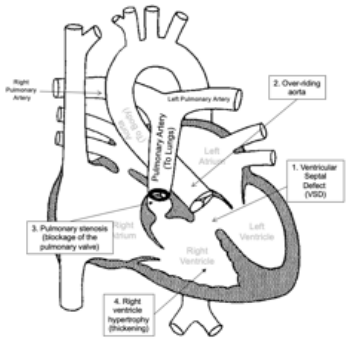
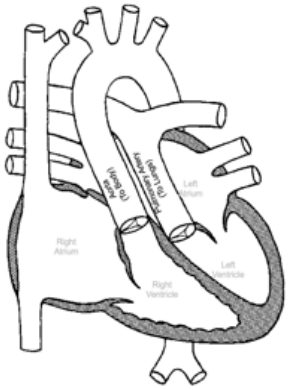
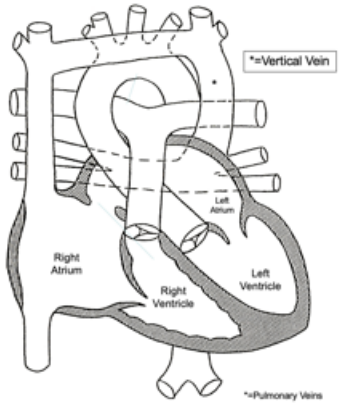
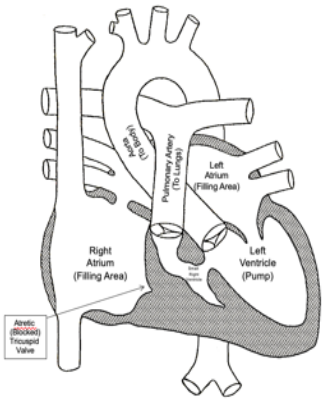
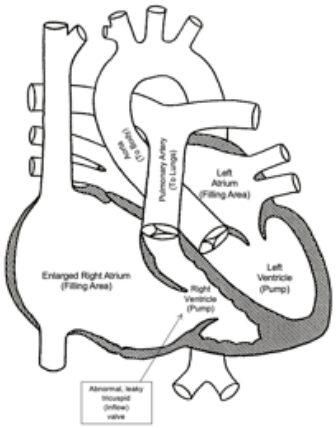
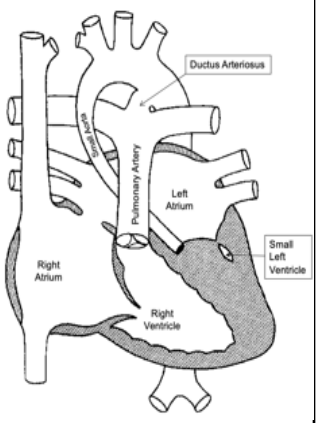
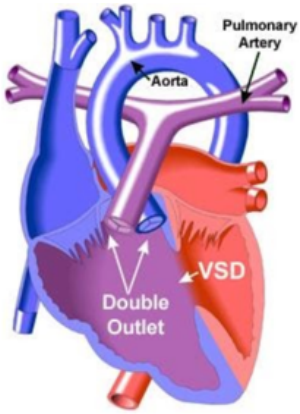
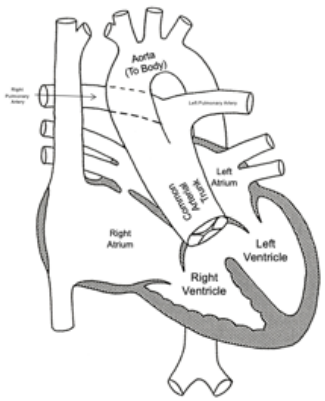
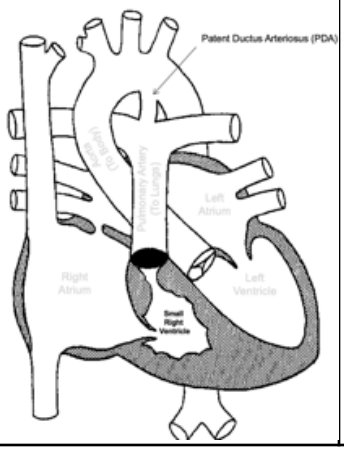
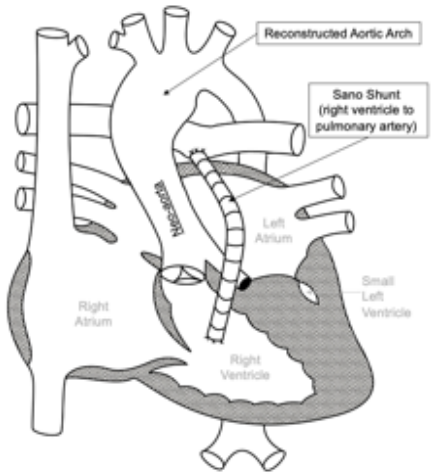
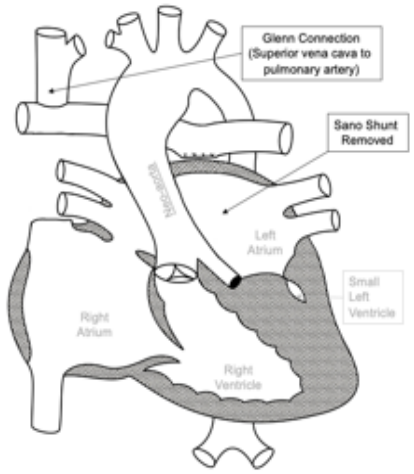
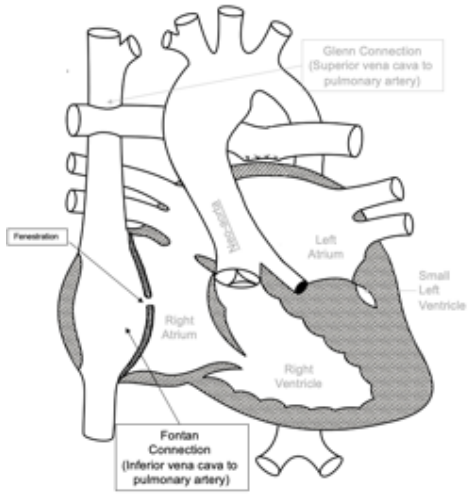
Cyanotic Heart Defects

	**Tetralogy of Fallot:** Problem: 4 components leading to decreased pulmonary flow SxS: tachypnea +/- cyanosis PE: SEM at LUSB, absent/soft P2 Tx: PGE if critical, surgery to close VSD and relieve RVOT obstruction		**D- Transposition of the Great Arteries** Problem: two parallel circuits requiring mixing (ASD vs VSD) SxS: profound cyanosis and tachypnea at birth PE: often no murmur if no VSD +/- single S2 Tx: PGE emergent balloon atrial septostomy arterial switch
	**Total Anomalous Pulmonary Venous Return:** Problem: PV don't drain into LA. 4 types: supracardiac, intracardiac, infradiaphgramatic, mixed. Requires ASD or VSD. SxS: cyanosis +/- tachypnea, crackles PE: Increased RV impulse, SEM at LUSB, diastolic TV rumble, +/- fixed split S2 (if ASD) +/- single loud S2 (if PV obstruction) Tx: surgery (emergent if severe PV obstruction)		**Tricuspid Atresia** Problem: No outlet from RA to RV. Supply to LA via PFO/ASD SxS: cyanosis and tachypnea (onset varies based on VSD size and PS degree) PE: Single S2 +/- VSD murmur Tx: PGE (if cyanotic) +/- atrial septostomy. Staged palliation (BT shunt bidirectional Glenn Fontan)
	**Ebstein Anomaly of the TV:** Problem: apically displaced TV, atrialization of RV, RA enlargement. Requires ASD. SxS: Varies. Neonates with cyanosis and RH failure vs. adults with murmurs and arrythmias PE: systolic murmur (TR) +/- gallop Tx: Surgical repair +/- PGE		**Hypoplastic Left Heart Syndrome:** Problem: Left-sided obstruction 2/2 underdeveloped L heart; three types (MS/AS, MS/AA, MA/AA) with unrestrictive ASD vs. restrictive ASD vs. intact atrial septum (IAS) SxS: cyanosis & pulmonary edema. Cardiogenic shock if unrestrictive ASD PE: Increased RV impulse, single S2, poor pulses, cold Tx: PGE, balloon atrial septostomy if IAS, three stage univentricular palliation
	**Double outlet RV:** Problem: RV drains into PA and Aorta (multiple types) + VSD SxS: Depends on type PE: depends on type Tx: medical management (balance Qp:Qs) and surgery		**Truncus Arteriosus:** Problem: failure of embryonic truncus arteriosus to septate; several subtypes SxS: cyanosis and pulmonary overcirculation as PVR falls PE: loud single S2, ejection click. SEM at LUSB. Diastolic decrescendo murmur from truncal regurg. Bounding pulses from diastolic run off. Tx: Surgery

Cyanotic lesions Continued			
	**Pulmonary Atresia with Intact Ventricular Septum** Problem: Atretic PV with ASD and varying degrees of RV hypoplasia PE: cyanosis at birth (worsens with PDA closure), PDA murmur Tx: PGE; depending on coronary circulation, single ventricle palliation vs. RV-PA conduit		

Single Ventricle Palliation

After **Stage 1 Norwood w/ Sant Shunt**	After **Stage 2 "Glenn"**	After **Stage 3 "Fontan"**
		
1) Establish Qs: neo-aortic valve, AscAo anastomosis, aortic arch repair 2) Provide Qp: RV-PA conduit (Sano shunt) or aorto-PA conduit (BT shunt) Post-op sats: 75-85%	1) Maintain Qp: anastomose SVC to RPA 2) Reduce volume to systemic ventricle (remove Sano or BT shunt) Post-op sats: 80s	1) Increase Qp: anastomose IVC to RPA (extra-cardiac tunnel vs lateral aka within atrium) 2) Provide fenestration (hole connecting Fontan pathway to systemic atrium) Post-op sats: >90%

Critical Care: ICU/ICP

SEDATIVES/HYPNOTICS/ANESTHETICS

SEDATIVES				
Name (Trade Name)	**Onset & Duration of Action**	**Dosage**	**Pros & Uses**	**Cons & Contraindications**
diazepam (Valium)	4-5 minutes / LONG	IV 0.1 mg/kg/dose (typical max 4mg)	Anxiolysis, amnesia, muscle spasm; reversal w/ Flumazenil	Respiratory dep, synergistic w opioids
lorazepam (Ativan)	15-30 minutes / MODERATE	IV 0.05-0.1 mg/kg/dose q4-12h	Anxiolysis, amnesia, muscle relaxation; reversal w/ Flumazenil	Respiratory dep, synergistic with opioids
midazolam (Versed)	1-5 minutes / SHORT	IV 0.05-0.1 mg/kg/dose q1-2h	Short-acting, preoperative anxiolysis; amnesia, muscle relaxation; reversal w/ Flumazenil	Respiratory dep, synergistic with opioids. Benzos associated with higher risk of delirium, especially shorter-acting
ketamine	30 seconds / SHORT to MODERATE	IV induction: 2-3 mg/kg (+ 0.5 mg/kg/dose q5min PRN) Conscious sedation: 0.2-1 mg/kg (load) + 0.5 mg/kg q10min PRN	Mild analgesia, bronchodilation, amnesia	Causes dissociative/trance-like state - can cause hallucinations/delirium; myocardial depressant but also releases catecholamines- causes hypotension in catecholamine-depleted pts
dexmedetomidine (Precedex)	5 minutes / LONG as an infusion	Infusion: 0.2-2 mcg/kg/hour	Anxiolysis, deep sedation mimics natural sleep EEG, no respiratory depression	Dose-dependent bradycardia (common); hypotension
propofol	30 seconds / SHORT (LONG as an infusion)	Bolus: 2-3 mg/kg Infusion: 20-200 mcg/kg/min (*only for <12 hours in children)	Short duration of action with bolus doses; can facilitate rapid intubating conditions or level of anesthesia	Respiratory dep, dose-dependent hypotension (vasodilation *and* myocardial depression); prolonged/high-dose infusions increase risk of propofol infusion syndrome (cardiac failure, arrhythmias, rhabdo, lactic acidosis, hyper-TG, etc.)
ANALGESICS				
morphine	20 minutes / LONG (3-5 hours)	IV 0.05-0.1 mg/kg/dose q1-2h	Quick onset, sedation	Can cause histamine release → hypotension, pruritus, flushing; nausea, constipation, respiratory depression
hydromorphone (Dilaudid)	15 minutes / LONG (5 hours)	IV 0.015 mg/kg/dose q3h	Often used as a PCA	Nausea, constipation, respiratory depression
fentanyl	Immediate / MODERATE (30-60 minutes)	IV 1-2 mcg/kg/dose q1h	Minimal hemodynamic instability; less sedating, no histamine release	Large/rapid bolus doses → muscle (chest) rigidity
Naloxone for opioid reversal: (full reversal with 5-10 mcg/kg/dose [max 200 mcg/dose] 1-2mc/kg/dose for respiratory depression.				

ICU Respiratory Support

	Flow rate	**FiO2**	**Titration**	**Dispo**
BBO2	>10 L/min	<30%	n/a	floor
LFNC	0.25-6 L/min	24-40%	Flow rate only	Up to 4L/min on the floor
Simple mask	6-12 L/min	35-60%	Flow rate only	floor
Non-rebreather	10-15 L/min	100%	non-titratable	Bridge therapy
HFNC	1-2 L/kg	21-100%	Flow rate & FiO2	ICP

Effect of vent changes on O2 and ventilation		
Vent change	PaO2	PCO2
↑ PIP or TV	↑	↓
↑ PEEP	↑	↑
↑Rate	Small ↑	↓
↑I:E ratio	↑	+/- small ↑
↑ Fio2	↑	No change

Troubleshooting:

- Bag masking w/ MR SOPA
  - **M**ask - reposition mask, consider 2 hands
  - **R**eposition head (neutral, slightly extended, “sniffing”)
  - **S**uction mouth and nose
  - **O**pen mouth, move jaw forward
  - **P**ressure - increase PIP
  - **A**rtificial airway (intubate or LMA)
- Mechanically ventilated patients w/ DOPE:
  - **D**islodgement of ETT → look for ETCO2 reading, mask ventilate, call staff assist
  - **O**bstruction (mucus plug) → suction, call RT
  - **P**neumothorax → obtain CXR, consider needle decompression if concern for tension physiology
  - **E**quipment Failure → bag-ETT ventilate, call RT

Modes of Ventilation

Non-Invasive Positive Pressure Ventilation

- **Continuous Positive Airway Pressure (CPAP)**
  - Provides continuous PEEP. No breaths delivered, so patient MUST be spontaneously breathing.
  - Alveolar recruitment improved → better V/Q matching → improves oxygenation. Can also adjust FiO2.
- **Bilevel Positive Airway Pressure (BiPAP)**
  - Provides inspiratory pressure (IPAP) and expiratory pressure (EPAP). **Present settings as "IPAP/EPAP"**
  - In addition to alveolar recruitment, delta pressure (IPAP - EPAP) influences Vt to improve ventilation. IPAP can also reduce the work of breathing. Can also adjust FiO2.
  - Machine breaths that are not aligned w/ patient efforts do not result in good Vt due to the noninvasive interface - not a good choice for patients w/ inconsistent respiratory drive. Not a good choice for patients w/ altered mental status or who cannot protect their airway (ie. no cough or gag) from aspiration.

Invasive Mechanical Ventilation

Basics:

- 2 goals: oxygenation and ventilation
  - To improve **oxygenation** (increase pO2) by recruiting alveoli and optimizing V/Q matching → titrate **PEEP, MAP, FiO2, I:E ratio**
  - To improve **ventilation** (decrease pCO2) by increasing alveolar ventilation → titrate **RR, Vt.** *Caution in patients with obstructive respiratory physiology: increasing RR decreases expiratory time, providing less time for alveoli to empty despite needing more time – this can place patients at risk of dynamic hyperinflation/air trapping.*
  - *To protect the airway in patients who cannot (invasive mechanical ventilation)*
- Compliance = ∆Volume / ∆Pressure
- MBR = Mandatory breath rate: # of breaths ventilator delivers per minute
- RR = MBR + spontaneous breaths
- PIP = Peak inspiratory pressure: highest pressure pt sees during respiratory cycle.
- PEEP = Positive end expiratory pressure: pressure lungs see in between delivered breaths
- Vt = Tidal volume: volume delivered to pt during inspiration
- IT = Inspiratory time: length of inspiration
- ET = Expiratory time: time over which exhalation occurs (aka time between inspirations) (I:E ratio usually 1:2 - 1:3)
- MAP = Mean airway pressure

Targets in Conventional Mechanical Ventilation

- **Pressure Control (PC)**: set pressure (PIP), Vt varies from breath to breath
  - May reduce barotrauma by limiting peak pressures
- **Volume Control (VC)**: set Vt, peak inspiratory pressure varies from breath to breath
  - Avoids changes in Vt with changing compliance, but sudden changes in compliance can result in BIG changes in pressure.

Modes of Mechanical Ventilation

- **AC (Assist Control):** *Can target pressure (PC-AC) or volume (VC-AC)*
  - Breaths can be triggered by patient (assisted breaths) or elapsed time if patient not able to trigger (controlled breaths). Every breath is machine supported and has the same parameters, whether patient- or machine-triggered.
  - Risk of overventilation if patient's spontaneous RR is high for other reasons (fever, agitation) or if ventilator is inappropriately triggering
  - **AC/VC Settings: Volume/PEEP/RR/IT/FiO2**
  - **AC/PC Settings: PIP/PEEP/RR/IT/FiO2**
- **SIMV (Synchronized Intermittent Mandatory Ventilation):**
  - Machine will synchronize breath delivery to align w/ patient's efforts
  - If patient does not trigger a breath during assist window (when machine is looking for patient effort), machine will provide a mandatory breath to patient. This way it ensures delivery of the set mandatory breath rate
  - Often paired w/ pressure support ventilation (SIMV + PSV) to support breaths above mandatory breath rate
  - **The main difference between AC and SIMV is what happens when pt triggers a breath: in AC pt gets full machine support, the same as a machine triggered breath. In SIMV, pt gets full machine support for any breath triggered during assist window up to the MBR; for breaths above MBR, pt gets whatever pressure support level has been set; if no PSV specified, then the patient receives no active support beyond bias flow (this would be unfusual).*
- **PRVC (Pressure Regulated Volume Control)**
  - Ventilator adjusts pressure depending on tidal volume generated by a "test breath" with fixed characteristics (PIP, IT)
  - Optimizes lowest pressure possible to achieve set tidal volume by constant adjustments
- **Pressure Support (PS)**
  - **Settings: IPAP/PEEP/RR/FiO2**
  - Requires patient to initiate all breaths. When patient triggers a breath, machine delivers a set level of pressure above PEEP
  - Flow cycled: inspiration terminated when flow drops below a set percentage of peak inspiratory flow (aka patient determines IT)

SHOCK

Shock Definition: Metabolic demands of body > delivered oxygen to tissues

- Oxygen delivery (DO2) = content of arterial oxygen (CaO2) x cardiac output (CO)
- CaO2 = (1.34 x Hgb x % O2 Sat) + (0.003 x PaO2)
- CO = SV x HR, SV determined by preload, afterload, and contractility

Type of Shock	Causes	Physiology	Findings	Treatment
Hypovolemic	Dehydration, hemorrhage, osmotic diuresis, third-spacing fluid, burns	Not enough fluid in vasculature → decreased preload & CVP → low CO → decr. O2 delivery	Dry mucous membranes, oliguria, weak pulses w/ delayed capillary refill	Fluid resuscitation, stop fluid losses (e.g. treat bleeding) Rapid transfusion protocol if hemorrhage Rapid infuser in ICUs, ED, OR
Distributive	**Septic & anaphylactic:** Vasodilation & increased capillary permeability) **Neurogenic:** Loss of sympathetic innervation to vascular tone)	Poor tone and/or leaking of vasculature → low SVR → relative hypovolemia/ preload, low DBP. Contractility may be depressed later in sepsis presentation, CVP will vary. Septic shock has cardiogenic + hypovolemic features	Bounding pulses & brisk capillary refill if capillaries are leaky → warm extremities (NOT always true in pediatric septic shock!) Low DBP (especially in neurogenic), widened pulse pressure	Hemodynamically unstable after 40-60cc/kg fluids given → vasopressors Vasopressors: Anaphylactic → EPI Neurogenic → NE Septic (no c/f cardiac dysfunction) → NE Septic (c/f cardiac dysfunction) → EPI
Cardiogenic	Arrhythmias, myocarditis, CHF, cardiomyopathy, trauma, cardiac tamponade, pulmonary embolism	Poor contractility or ability to relax → ineffective systolic output → decreased CO w/ high SVR	Weak pulses w/ narrow pulse pressure (due to low SBP), pallor, cold extremities, delayed capillary refill, heart failure (respiratory distress, hepatomegaly, JVD)	LIMIT fluid resuscitation (5-10cc/kg) Inotropic agents (low dose dopamine, or epinephrine, less commonly dobutamine) Can consider milrinone if BP normal to decrease afterload
Obstructive	Pulmonary embolism, cardiac tamponade, CoA, pHTN	Ability to produce adequate CO is impaired because of obstruction	Tamponade - Pulsus paradoxus or electrical alternans, narrow pulse pressure w/ increased diastolic	Specific to the underlying cause

VASOPRESSORS & INOTROPES

	Receptor	Dose Range (mcg/kg/min)	Notes
Dobutamine	B1 > B2	0.5-20	Heart failure, cardiogenic shock (inotropic > chronotropic)
Dopamine	D1 = D2 > B1 > α1	1-20 - 1-5 mostly affects D-R - 6-10 B1 - 11-20 α1	Low dose (more B1) → increase CO, HR – can be arrythmogenic High dose (more α) → vasoconstricts, increases SVR. Can decrease CO
Epinephrine	B1 = B2 > α1	0.01-1	Anaphylaxis, asthma, septic shock. Increases CO, SVR. α effects predominate at high doses. Stronger B2 (inotropic) effect relative to increase in SVR particularly at lower doses.
Norepinephrine	α1 > α2 > B1 > B2	0.01-1	Increases SVR. Minimal HR effect.
Milrinone	Phosphodiesterase inhibitor	0.25-1	Decreases SVR → reduces afterload. Useful in cardiogenic shock in normo or hypertensive pts.

Dermatology

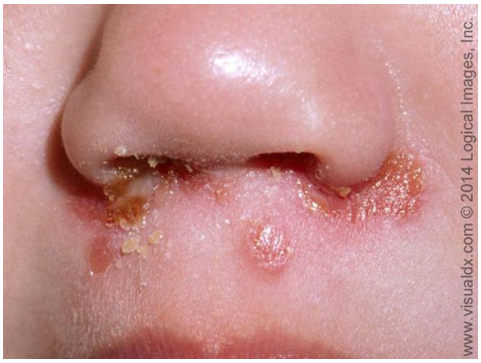
Erythroderma
- Nikolsky Positive:SJS/TEN, acute CVHD, SSSS
- Nikolsky negative
  - Fever positive: TSS, DRESS, Kawasaki, Scarle fever
  - Fever negative: anaphylaxis, angioedema, Sezary syndrome,scombroid

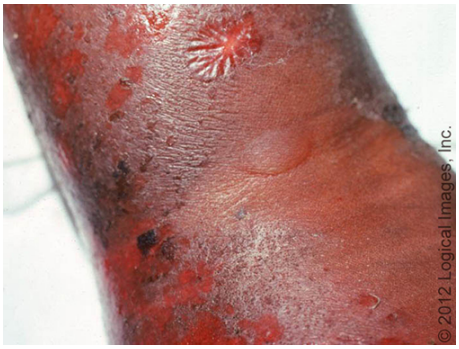
Fluid Filled
- Pustular: GC, candidiasis, folliculitis, ance, rosacea
- Vesiculobullous:
  - Fever positive
    - Diffuse: SJS, TEN, Disseminated HSV/ZVZ
    - Localized: NSTI, eczema herpeticum, hand-foot-mouth
  - Fever negative:
    - Diffuse: BP, PV, bullous impetigo
    - Localized: Burn, HSV/VZV, contact dermatitis, poison ivy

Petechiae
- Fever positive:
  - Palpable: Meningococcemia, RMSF
  - Nonpalpable: DIC, endocarditis, TTP
- Fever negative
  - Palpable: Autoimmune vasculitis
  - Nonpalpable: APLS, HIT, ITP, Cholesterol embolism, amyloidosis, nutritional deficiency

Maculopapular
- Fever positive:
  - Targetoid: EM, Lyme disease
  - Non targetoid: Meningococcemia, RMSF, Syphilis
- Fever Negative
  - Red:
    - Scaling: psoriasis, tinea, SLE, atopic dermatitis, eczema, candidiasis, scabies
    - Nonscaling: exanthem, cellulitis, drug reaction, insect bite
  - White: pityriasis, tinea versicolor, vitligo
  - Brown: melanoma, nevus, seborrheic keratosis
  - Yellow: Actinic keratosis, crusted lesion, xanthelasma
  - Skin: Verruca, BCC, SCC, Molluscum contagiosum, lipoma

Impetigo: *Non-bullous* *Bullous*





- Nonbullous: on traumatized skin, S aureus and S pyogenes.
- Bullous: infants and young children, caused by S aureus toxin causing cleavage in the superficial skin layer
- Tx: topical mupirocin if limited, oral cephalexin if extensive; clindamycin if MRSA

Baby Rashes
- Sebaceous hyperplasia: yellow filled papules forehead/nose/lips
- Milia: pearly white filled cysts on nose
- Neonatal ache: papules and pustules w/o comedonal
- Erythema toxicum: red base papules/pustules
- Transient Neonatal Pustular Melanosis: TNPM: pustules in stages of rupture w/ hyperpigmented macules
- Seborrheic dermatitis: cradle cap
- Congenital dermal melanocytosis: slate gray patch
- Congenital Melanocytic nevus: moles
- Nevus simplex: stork bite/angel kiss/salmon patch
- Nevus flammeus: port wine stain
- Cutis marmorata: mottling 2/2 environment

Emergency Medicine

Trauma Primary Survey	
Airway	• Obstruction - Open airway; suction secretions; Administer 100% O2 • Midface fracture/difficult airway or Direct airway injury - Surgical Airway
Breathing	• Tension pneumothorax - Needle decompression; place chest tube or pigtail catheter • Massive hemothorax - chest tube • Open pneumothorax - 3-sided occlusive dressing • Flail chest - Perform bag-valve-mask ventilation • Impaired oxygenation/ventilation - Rapid sequence intubation
Circulation	• Absent circulation - Cardiac compressions • External hemorrhage - Apply direct pressure to control external hemorrhage • Signs of shock - IV access; Obtain laboratory studies; Fluid resuscitation • Cardiac tamponade - Pericardiocentesis; may require thoracotomy • Pelvic fracture - Wrap or bind pelvis
Disability	• Level of consciousness (GCS) - Endotracheal intubation for rapidly declining GCS, GCS ≤8 or herniation • Signs of spinal cord injury - Immobilize spine (especially cervical) • Signs of impending herniation - Elevate head of bed to 30° if no signs of shock; Hyperventilate (pCO2 30 to 35); Osmotic agents if normotensive • Neurosurgical consultation
Exposure	• Toxins/Hazards - Remove clothing • Hypothermia - Initiate rewarming

Trauma Secondary Survey	
Head	Any scalp/skull injury, periorbital or postauricular bruising
Eye	Corneal reflex Fundoscopic exam
Neck	· C-spine tenderness or deformity · Hematoma · Trachea midline · Bruit
Chest	Clavicle deformity or tenderness Breath sounds, heart sounds Chest wall symmetry, paradoxical movement, rib deformity, fracture
Abdomen	• Serial exams to evaluate tenderness, distension, ecchymosis • Shoulder pain suggests subdiaphragmatic process • Orogastric aspirates with blood or bile • Splenic laceration suggested by left upper quadrant rib tenderness, flank pain, flank ecchymoses, “seatbelt sign”
Pelvis	Tenderness, symmetry, deformity, stability
GU	Laceration, ecchymoses, hematoma, bleeding Rectal tone, blood, displaced prostate Blood at urinary meatus → don’t catheterize, suggests urethral injury
Back	Evaluate for step offs along spinal column, tenderness
Extremities	Neurovascular: pulse, perfusion, pallor, paresthesias, paralysis, pain.Motor/sensory
Skin	Lacerations, abrasions, contusions

Salter-Harris Classification (for physeal fractures)					
	Type I  **S** **S**traight across	Type II  **A** **A**bove	Type III  **L** **L**ower or Be**L**ow	Type IV  **T** **T**wo or **T**hrough	Type V  **E R** **ER**asure of growth plate or C**R**ush
Details	Only involves the growth plate	Growth plate + metaphysis (Most common)	Growth plate + epiphysis + joint space	Metaphysis + growth plate + epiphysis + joint space	Compression of growth plate
Implications	Good prognosis	Good prognosis	Threatens growth and articular integrity	Threatens growth and articular integrity	Very high risk for growth arrest

Common MSK Injuries

Upper Extremities:

Supracondylar fracture ● Usually FOOSH with elbow hyperextension ● Exam: Gross deformity, limited active elbow motion ● Imaging: Get AP and lateral XR. Findings may be subtle (posterior fat pad sign on lateral film) ● Ortho consult: Usually surgical fixation for displaced fractures	**Nursemaid's Elbow (AKA subluxation of radial head)** ● Traction on arm with extended elbow (e.g. swinging child through the air) ● Exam: no deformity, elbow held in passive pronation with slight flexion, refusing to use arm · Imaging: Unnecessary unless suspect fracture based on H&P, or if reduction unsuccessful ● Stabilize elbow w/ one hand → supinate forearm and flex elbow (will usually feel/hear click)
Distal Radius Fracture ● Most common pediatric fracture; typically FOOSH ● Exam: Pain, ecchymosis, swelling ● Imaging: AP + lateral of wrist and forearm; consider AP+lateral of elbow if tender or if diaphyseal fractures present ● Ortho consult: Depending on severity may require anything from immobilization to ORIF	**Proximal Humeral Fracture** ● FOOSH or Direct blow to lateral shoulder ● History of trauma, severe shoulder pain, pain w/ arm movement ● Exam: tenderness, swelling, shoulder asymmetry, arm shortened and held in extension ● Imaging: AP and axillary XR views of humerus Get scapular "Y" view in addition if concerned for shoulder injury ○ Suspect Salter-Harris I if negative XR + tenderness at physis ● Immobilization ● Likely ortho consult (esp if more severe - assoc. w/ shoulder dislocation, neurovascular compromise, etc.)

Hip and Knee:

SCFE (Slipped capital femoral epiphysis) ● Displacement of the capital femoral epiphysis from the femoral neck through the physeal plate; commonly ages 10-16, M > F ● Groin pain, knee pain, limp ● Exam: decreased hip ROM, hip externally rotated at rest, leg length discrepancy ● Imaging: AP and frog leg lateral hip XR ·Look for "ice cream scoop falling off the cone ● Ortho consult	**Legg Calve Perthes** ● Avascular necrosis of the hip, most common age 5-7, M > F ● Activity-related hip pain and/or limp (acute or chronic) ● Exam: Trendelenburg gait, decreased hip abduction and internal rotation ● Imaging: XR often normal early in course, bone scan or MRI more suggestive of dx ● Non-weight bearing and restoration of motion - crutches, NSAIDS, PT, aquatherapy ● Severe cases may require spica casting or surgery
ACL Injuries ● Cutting/pivoting motion causing valgus stress on knee, can be 2/2 direct blow causing hyperextension/valgus deform. ● Medial meniscus and MCL often injured at same time (Unhappy Triad) ● "Pop" at time of injury, swelling, feeling of knee "giving out" ● Exam: Joint effusion, positive anterior drawer test ● Imaging: MRI > XR, but can get XR to evaluate for associated injury/fracture ● Ortho/Sports Medicine referral ● Operative management in majority of cases, ideally w/ period of pre-operative rehabilitation to optimize outcomes	**Meniscus Injuries** ● Direction change w/ knee rotation, planted foot, and flexed knee. Common in sports w/ deceleration and direction change ● Often insidious onset of pain/swelling in 24h after injury. Pain worse w/ twisting/pivoting ● Can have a locking/popping/catching sensation. ● Exam: joint line tenderness, inability to fully extend/squat/kneel, positive McMurray test ● Imaging: MRI > XR (plain films often negative) ● Ortho/Sports Medicine referral ● Management varies from conservative to operative (usually arthroscopic)

Foot and Ankle

Ankle Sprain
- Ligamentous stretching/tearing
- Lateral: inversion of plantarflexed foot - injures ATFL most commonly
- Medial: eversion or abduction/ external
- Pain, swelling (diffuse or localized), +/- inability to bear weight
- Exam: swelling, TTP, positive anterior drawer/talar tilt (lateral sprain), positive mid-calf squeeze (high sprain)
- Imaging: not routinely indicated unless concern for or clinical uncertainty
- Short period of complete immob. (longer depending on severity), supportive device (lace-up brace or elastic bandage)
- ROM/strength exercises (can be w/ formal PT, esp in case of recurrent ankle sprains) critical to restoring function and proprioception
- For HIGH ankle sprains, consult ortho/sports medicine (may need acute surgical stabilization if severe)

Ottawa ankle rules: when to get XR of the ankle/foot (validated age >18yo)
- Ankle: pain localized to malleolar zone and EITHER of:
  - Bony tenderness at post edge of lateral/medial malleolus
  - Inability to bear weight both immediately after injury and at time of exam
- Foot: pain in midfoot zone and EITHER of:
  - Bony tenderness at base of 5th met or navicular
  - Inability to bear weight both immediately after injury and at time of exam

Sever's Disease
- Traction apophysitis of calcaneal growth plate at site of Achilles insertion; often children who play sports w/ jumping/heel striking and/or are undergoing rapid growth spurt
- Essentially Osgood Schlatter at the calcaneus
- Chronic heel pain w/ insidious onset, worse w/ activity or wearing non-supportive footwear
- Exam: TTP at calcaneal apophysis or w/ "calcaneal compression test" ·Imaging: not routinely indicated unless diagnosis unclear or to rule out fracture
- Painful activity → gradual return to play, use of heel cup for support, ice and stretching

Spiral/Oblique Fracture
- "Toddler's fracture" in 9mo-3yr
- Rotation around fixed foot → distal tibial fracture; often minimal trauma in toddlers, higher impact injury in older children
- Approx 30% of tibial fractures have associated fibular fracture
- Spiral fractures in NON ambulatory child → concern for NAT
- Limp, refusal to bear weight
- Exam: point tenderness over distal 1⁄3 of tibia
- Imaging: AP and lateral XR of the tibia and fibula; fractures may be occult (not seen on imaging)
- Immobilization in long leg posterior splint/cast
- Ortho referral

Laceration Repair

Equipment

- Basics: light, mask, sterile gloves & gown, betadine
- Irrigation: sterile bowl, sterile water, 20-50 cc syringes with splash guard (all except water come in irrigation kit)
- Local anesthesia or digital block:
  - LET gel (lidocaine, epinephrine, tetracaine) – apply for 15-20 minutes (surrounding skin should be blanched)
  - 1% lidocaine (10mg/mL): onset 2-5 minutes, lasts 15-20 minutes. Toxic dose 5mg/kg (0.5cc/kg)
  - 1% lidocaine with epinephrine (1:200,000): onset 2-5 minutes, duration ~60 minutes. Do not use in digits, penis, pinna, tip of nose
  - Use buffered lidocaine if available (buffered with sodium bicarbonate)
- Suture tray (sterilized and packaged together): forceps, scissors, needle driver, hemostats, sterile gauze
- Suture material: Nonabsorbable sutures vs. Absorbable sutures. Use absorbable for deep wounds and in small children when suture removal would be just as traumatic as placement

<u>Technique</u>

- Simple interrupted - most common, closes superficial layer
- Deep subcutaneous - reduces tension of deep wounds
- Buried horizontal dermal - closes deep layer in shallow lacs
- Horizontal/vertical mattress- reinforce SC tissue, relieves wound-edge tension
- Corner stitch - repair flap-type, corner lacerations

Absorbable sutures:
- Braided: vicryl or vicryl rapide
- Monofilament: monocryl, fast absorbing gut, chromic gut

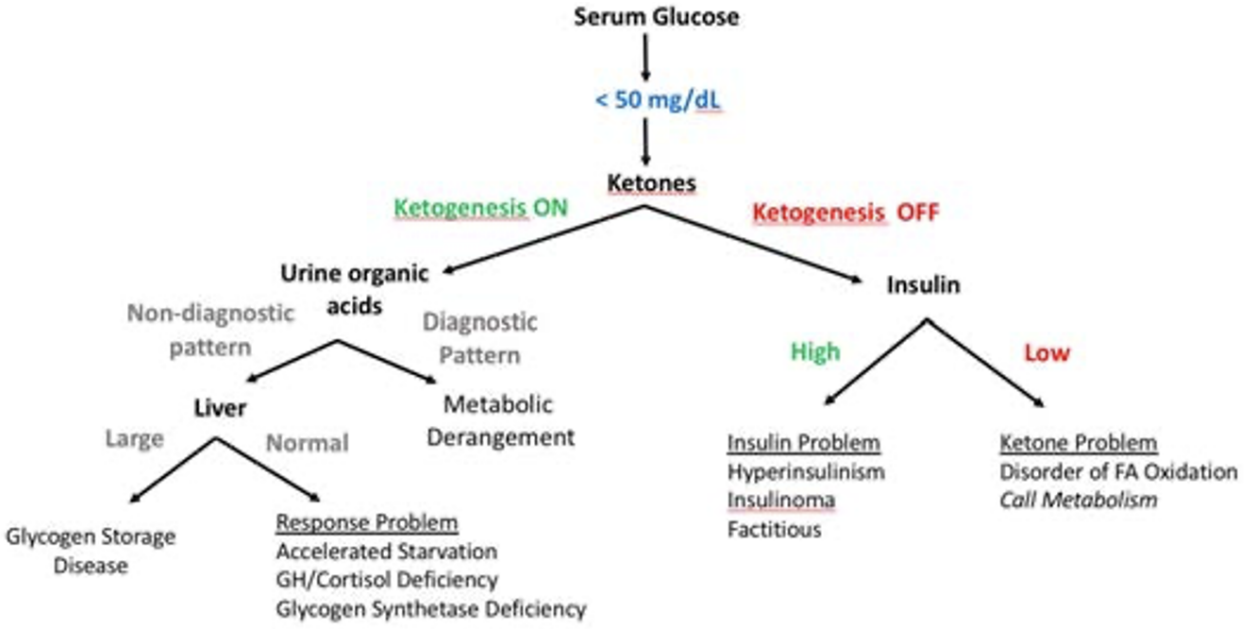
Non-absorbable sutures:
- Braided: ethibond, silk
- Monofilament: ethilon

Suture selection
- Face: 5-0 to 6-0
- Scalp: 3-0 to 5-0
- Chest: 3-0 to 4-0
- Back: 3-0 to 4-0
- Abdomen: 3-0 to 4-0
- Extremities: 4-0 to 5-0
- Joints: 3-0 to 4-0
- Soles of foot: 3-0 to 4-0
- Oral: 3-0 to 5-0 absorbable

Alternatives to sutures:
- Dermabond (tissue adhesive) +/- Steri-Strips: use for linear wounds with minimal tension. No removal needed.
- Staples: Best for scalp wounds. Requires remover.

<table>
<tr><th colspan="4">Febrile Infant</th></tr>
<tr><td>Definition</td><td colspan="3">Temperature ≥38.0 (100.4 C) in infant ≤60 days; Temperature ≥38.5 (101.3 C) in child >2-24 months</td></tr>
<tr><td>Etiology</td><td colspan="3">● Rates of serious bacterial infection (SBI) in febrile infants/young children range from 7-38% of infants aged 0-28 days seen in emergency department for fever.
● UTI is the most common (5.9%), followed by bacteremia (1%), meningitis (0.3%).</td></tr>
<tr><td>Pathogenesis</td><td colspan="3">● Bacterial: UTI, pneumonia, bacteremia, meningitis, enteritis, osteomyelitis
● Viral: Enterovirus, HSV, influenza, RSV, rotavirus, aseptic meningitis
● Neonate: (within first 7 days of life) often vertical transmission
● Less common: recent immunizations, malignancy, medications (antibiotics, antineoplastic drugs, biologics), immunological (Kawasaki), immunodeficiency (HIV, SCID, humoral deficiency), hereditary autoinflammatory syndromes of periodic fever, other periodic fever syndromes</td></tr>
<tr><td rowspan="4">Most Common Pathogens by Age</td><td>Age</td><td>Bacteremia/Meningitis</td><td>Other pathogens</td></tr>
<tr><td>0-28 days</td><td>Group B Strep
Gram negative enterics (E. coli, Klebsiella) Listeria</td><td>HSV
Conjunctivitis: Gonorrhea, Chlamydia, S. aureus
Pneumonia: Chlamydia, S. aureus Diarrhea: Salmonella</td></tr>
<tr><td>28-90 days</td><td>GBS (Late onset) Gram negative enterics Strep Pneumo
H. flu
N. meningitides</td><td>Pneumonia: Chlamydia, Staph aureus, Pertussis, RSV and other viruses Diarrhea: Salmonella</td></tr>
<tr><td>3-36 mos</td><td>Strep Pneumo H. flu
N. meningitides</td><td>UTI: E. coli, other GNR, enterococcus</td></tr>
<tr><td>Clinical Presentation</td><td colspan="3">● Non-specific symptoms: poor feeding, lethargy or irritability, fever or hypothermia
● History: Full pre- and perinatal history including GBS status, need for intrapartum antibiotics, evidence of maternal HSV or other infections
● Physical exam: bulging fontanelle (Meningeal signs unlikely in infants), respiratory distress or focal lung findings, conjunctivitis, oral lesions, vesicles, cellulitis, rash, vomiting, diarrhea, swelling of a joint or extremity
● Otitis media/URI symptoms, if present, do not preclude need for further eval.</td></tr>
<tr><td>Workup</td><td colspan="3">See the AAP 2022 Guideline for Febrile Infant for most up to date recommendations. Take away points:
● 0-21 days: CBC w/ diff, Blood Culture, Procalcitonin, UA and Urine Culture, Viral swab, LP (Cell count, protein, glucose, culture, gram stain, hold +/- HSV) +/- CXR.
● 8-21 day old: UA and urine culture, Blood culture, and LP. Optional inflammatory markers. Add HSV if at risk.
● 22-28 day old: optional LP based on inflammatory markers/UA. Dispo hinges on LP.
● 29 - 60 days: CBC w/ diff, Blood Culture, Procalcitonin, UA, Viral swab, CXR. Send LP and urine culture if inflammatory markers elevated.
● 2-24 months: Workup varies significantly based on presentation; recommend viewing EBG for more details</td></tr>
<tr><td>Treatment</td><td colspan="3">See the AAP 2022 Guideline for Febrile Infant for most up to date recommendations. Take away points:
● For ≤21 day olds, empiric therapy while awaiting culture results (see below table)
● For ≥22 day olds, empiric therapy depends on CSF pleocytosis, inflammatory markers, and dispo
● In patients with positive UA or cultures, therapy should be tailored appropriately</td></tr>
<tr><td rowspan="5">Empiric Antibiotic Treatment Based on Age</td><td>Age</td><td>Empiric Antibiotics</td><td>Other antigens to consider</td></tr>
<tr><td><or=14 days</td><td>Ampicillin + Ceftazidime</td><td>Gentamicin can replace Ceftazidime
If pleocytosis or ill-appearing, substitute Cefepime for Ceftazidime and add acyclovir</td></tr>
<tr><td>15-28 days</td><td>Ceftriaxone (50 mg/kg)</td><td>Add ampicillin and acyclovir if CSF pleocytosis or ill- appearing
Meningitic dose (100 mg/kg/day) if CSF pleocytosis</td></tr>
<tr><td>29-60 days</td><td>Ceftriaxone</td><td>Meningitic dosing and Vancomycin if CSF pleocytosis</td></tr>
<tr><td>2-24 months</td><td colspan="2">Antibiosis varies significantly based on presentation and workup; recommend viewing EBG for more details</td></tr>
</table>

Endocrinology

Type 1 Diabetes – SubQ Insulin Regimen *(Non-DKA)*	
Common Definitions & Equations	TDD (total daily dose) = total units insulin (50% basal + 50% bolus) per day (ie. 30 units) · TDD in Puberty = 0.5 - 0.75 units/kg/day · TDD not in Puberty = 0.25 - 0.5 units/kg/day **Bolus** = correction + carb coverage | **Basal** = given once daily, is half the TDD (ie. 15 u) *Correction Factor* = 1500/TDD (ie. 1500/30 = 50. CF = 1:50 or 1u insulin drops BG by 50) *Carb Ratio* = 500/TDD (ie. 500/30 = 16. CR = 1:16 or give 1u insulin for every 16g of CHO)
Hypoglycemia	
Acute Management	If glucose <70: Juice/feed/glucose tab/gel then recheck in 15 minutes. Repeat PRN If unable to take PO (NICU, obtunded): "Hawaii 5-0 rule," bolus x dextrose content = 50 · IV bolus: 10 cc/kg bolus of D5W, 5 cc/kg bolus of D10W, 2 cc/kg bolus of D25W · IV gtt: GIR (mg/kg/min) = [(IV rate x Dextrose conc (g/dL) x1000)/(Wt (kg) x 60 x 100)] · No IV: Glucagon 0.03 mg/kg (max 1 mg/dose) IM, subQ, IV, or IN · *Note: Effective for hyperinsulinemia, not effective for glycogen storage disease*
Workup	
Critical Labs	***Blood***: Plasma glucose (<50 mg/dL to be "critical sample"), Insulin, BHB, Chem 10, VBG, Lactate, Pyruvate, NH_3, GH, Cortisol, FFA, Total/Free Carnitine, Acylcarnitines, Serum Amino Acids. ***Urine***: Urinalysis, Urine Organic Acids, Acylglycines
Central Diabetes Insipidus	
Diagnosis	Serum Osm > 300 AND urine Osm < 600 AND Serum Na > 145
Treatment	Acute: Give 1x breakthrough DDAVP when: UOP > 4 mL/kg/hr AND Urine Osm < 300 Chronic: Desmopressin (DDAVP) daily (up to q12 or q8) – start at 0.05 mg PO or 5 mcg IN
Adrenal Insufficiency	
Acute Management	**STRESS DOSE STEROIDS** (Fever >101F, surgery/anesthesia, fracture, dehydration) 1) Hydrocortisone 50-100 mg/m^2/dose IV x1 + 20 cc/kg Normal Saline Bolus 2) Hydrocortisone 25 mg/m^2/dose IV Q6hr + 1.5-2x maintenance IVF of D5NS or D5LR · Continue until 24hrs after stressor then transition to maint. dosing over 3-5 days *FYI SDS hydrocort covers both glucocorticoid + mineralocorticoid needs*
Chronic Management	**Glucocorticoid (Cortisol) Replacement** · Hydrocortisone 8-10 mg/m^2/day divided 2-3 times daily (largest dose in AM) *Note: Double dose if GI absorption problems. AM dose ~9 am, PM dose ~2-3 pm.* **Mineralocorticoid (Aldosterone) Replacement** - Infants: Fludrocortisone 0.15-0.3 mg/day PO QDay. May need Na supps. - Children & Adults: Fludrocortisone 0.1 mg/day PO QDay (0.05 – 0.2 mg/day)

Steroid Conversion Table				
	Equivalent doses (mg)	**Relative Anti-nflammatory Activity**	**Relative Mineralocorticoid Activity**	**Duration of action (Hours)**
Hydrocortisone, Cortisol (IV or PO)	20	1	1	8 to 12
Cortisone acetate (PO)	25	0.8	0.8	8 to 12
Prednisone (PO)	5	4	0.8	12 to 36
Prednisolone (PO)	5	4	0.8	12 to 36
Methylprednisolone (IV or PO)	4	5	0.5	12 to 36
Triamcinolone (IV)	4	5	0	12 to 36
Fludrocortisone (PO)	0	0	125	12 to 36
Dexamethasone (IV or PO)	0.75	30	0	36 to 72
Betamethasone (IV or PO)	0.6	30	0	36 to 72

Calcium

Diagnostics

- Confirm with iCal and/or albumin (1g/dL ↓ albumin = 0.8 mg/dL ↓ calcium)
- Hypocalcemia: PTH, magnesium, phosphate, BUN, creatinine, 25 OH-Vitamin D
- Hypercalcemia: PTH, PTHrP, 25 OH-Vitamin D, 1,25 OH-Vitamin D, FeCa (Urinary excretion)

	Calcium	PTH	25-OHD	Alk Phos	FeCa	1,25-OHD	PTHrP
Hypoparathyroidism	Low	Low	Normal	Normal			
PTH Resistance	Low	High	Normal	Normal			
Vit D Deficiency	Low	High	Low	Normal/high			
Vit D Resistance	Low	High	Normal	Normal			
Renal Disease	Low	High	Normal/low	Normal/high			
Hypomagnesemia	Low	Normal	Normal/low	Normal			
Metastatic Disease	High	High	Normal	High			
Primary Hyperparathyroidism	High	Normal/ High			>0.01		
Familial Hypocalciuric Hypercalcemia (FHH)	High	Normal/ High			<0.01		
Vit D Intoxication	High	Low	High				
Granulomatous Dz, CMV, Malignancy	High	Low	Normal/Low			High	
Malignancy, HIV	High	Low	Normal/Low			Normal/Low	High
See below*	High	Low	Normal/Low			Normal/Low	Normal

** Maternal hypocalcemia, Williams Syndrome, SubQ Fat Necrosis, Blue Diaper Syndrome, Disaccharide Deficiency*

Management

Hypocalcemia:

- **Chronic:** PO Calcium 10-15 mg/kg, up to QID (elemental calcium, max 1500 mg/dose)
- **Acute:** IV Ca Gluconate 100 mg/kg or CaCl 20 mg/kg (Emergencies only, dangerous irritant)
  - ***Hypocalcemia****: Replete Vit D and/or Mg as needed*
  - ***Vit D Deficiency****: Replete Vit D, always give Ca to prevent hungry bone syndrome*
  - ***Hypoparathyroidism:*** *Give calcitriol (1,25-OHD), not ergocalciferol or cholecalciferol*
  - ***Hyperphosphatemia:*** *Avoid Ca^{+} x PO^{4-} >55 because of metastatic calcification risk*

Hypercalcemia:

- **Severe (>14 mg/dL) or symptomatic**: Dilute/Excrete Calcium then decrease bone resorption
  - Dilute/Excrete: IV NS then Furosemide
  - Decrease resorption: Calcitonin 2-4 u/kg Q12hr, max 8 u/kg Q6hr or Pamidronate 0.5-1 mg/kg (monitor for hypocalcemia, hypophosphatemia, hypomag). Most pts on calcitonin will develop tachyphylaxis w/n 48 hrs
- **Primary Hyperparathyroidism**: Parathyroidectomy

GI/Nutrition/Hepatology

GI Bleeds		
Upper (UGIB): Hematemesis, coffee grounds, hematochezia, melena. DDx: swallowed maternal blood (apt test), foreign body, esophagitis, esophageal varices, mallory weiss, gastric ulcer, gastritis, duodenitis, duplication cysts, vascular malformations, hemobilia, pulmonary hemorrhage.	Lig of Treiz	**Lower (LGIB):** hematochezia, melena. Ddx: fissure, hemorrhoids, milk protein allergy, Meckles, intussusception, duplication, epistaxis, vascular, infectious, typhlitis, IBD, juvenile inflammatory polyp, HSP, varices, NEC, ischemic, swallowed mat blood.
Work up: CBC, Chem 10, Coags, LFTs, T&S, +/- Amylase/lipase, +/-KUB Tx: fluid, transfusions, NPO, PPI, coagulopathy correction, Octreotide for UGIB (ICU only), endoscopy.		

IBD		
Crohn's ● Nose to anus; skip lesions ● Transmural inflammation, +granulomas ● Fistulas, abscesses, obstructions ● Crampy abdominal pain ● Th2 cytokine response Tx: hydrocortisone, budesonide, prednisolone, sulfasalazine, olsalazine, mesalazine, balsalazide, infliximab, adalimumab, natalizumab, vedolizumab, ustekinumab	**UC** ● Continuous from rectum up ● Submucosal/mucosal inflammation ● Hemorrhage, toxic megacolon ● Bloody diarrhea; PUCAI score ● Th17 cytokine response Tx: EEN, 5ASA, sulfasalazine, balsalazide, infliximab, azathioprine, mercaptopurine	**Work up:** CBC, Chem, ESR, CRP Fecal Cal pro, SSYCE, Cdiff Celiac testing, iron studies HepA, B, C, testing TB testing TPMT testing Colonoscopy, endoscopy MRI/CT

Constipation Management	
Gush: osmotic. **Miralax, GoLytely, Lactulose, MagOx, Sorbitol (prune juice).** s/e: bloating, nausea, diarrhea, hypoK. Need to be well hydrated to work.	**Mush**: Softener. Must be given with a gush. **Docusate/colace**
Push: stimulant. **Senna, bisacodyl, glycerin.** s/e: cramping, decrease transient time can decrease nutrient absorption at high dose	**Woosh**: lubricant **Mineral oil**

- **Chocolate Bomb**= 4oz icecream, 15mL senna, 30 mL mineral oil, 30 mL MOM
- **SMOG enema**= 20 mL NS, 20 mL mineral oil, 20 mL GLycerin
- **GI cleanout:** Miralax 34g q 30 min x8 doses, or GoLytely via NG tube +IVF

Antiemetics Class	**Drug**	**AE**	**Good for**
Prokinetics (D2-R)	Metoclopramide Domperidone Prochlorperazine Chlorpromazine	Extrapyramidal side effects, QTc prolongation, hypotension.	Gastroenteritis, Migraine
ST Antagonists (5HT3-R)	Ondansetron Granisetron Olanzapine	QTc prolongation. Constipation, headache.	Gastroenteritis
Antimuscarinic (M1-R)	Scopolamine Atropine Hysocine	Anticholinergic side effects	Motion sick
Antihistamine (H1-R)	Diphenhydramine Hydroxyzine	Anticholinergic side effects	Motion sick
Neurokinin-1 Antagonists (NK1-R)	Aprepitant	Cyp inhibitor	Chemo
Gaba Agonist	Lorazepam Alprazolam	Sedation, respiratory depression	Functional
Others: Dexamethasone, Cannabinoids, Ginger, Peppermint.			

Formulas			
Cow: Enfamil (cheap), Sim advance (cheap), enfacare (premie), neosure (premie)	**Partially hydrolyzed**: Goodstart	**Hydrolyzed, semi elemental:** nutramigen (cheap), Alimentum (sweet), Pregestimil	**AA/elemental:** $$ neocate, Elecare
Soy: Prosobee (enfamil), Isomil (Similac), Goodstart Soy			

Gtube considerations:
- Clogged tube slurry= 1tab NaBiC (324 mg) +1tab Vlokase 8 (Pancrelipase) in 5mL water. Instill into tube, wait 30-60 minutes, withdraw, and flush. (enteral/gastrostomy tube obstruction orderset).
- Granulation: stabilize tube, silver nitrate, triamcinolone cream.
- Leakage: check balloon, fit to abdominal wall.

Pancreatitis:
- Chem 10, Amylase, lipase, lipids, albumin, LFTs, ultrasound, ketorolac, acetaminophen (=/- opiates risk of ileus), NPO x24 hours with NS vs LR boluses and then 2xmivf, enteral feeds at 24 horus if stable.
- Ddx: gallstones, ETOH, hypertriglyceridemia, ERCP, Genetics, Medications, ductal injuries, annular pancreas, biliary cysts.

Nutrition:
- "Multiple vitamins and minerals"= MVI order
- Cholecalciferol 10mcg=200 IU
- 20-30g growth per day
- See Vitamin D-Calcium EBG for dosing of supps

Oral Electrolyte Repletion:
Ca: 10-15 mg elemental calcium/kg/dose, up to QID (Max 1500 mg/dose)
Mg: 10-20 mg elemental magnesium/kg/dose, BID-QID
K: <4yr: 1 tab/packet, up to QID; ≥**4yr:** 1-2 tab/packets PO up to QID

Single electrolyte	Dosage Form	Elemental Content
Calcium Carbonate	250 mg/mL Liquid	100 mg/mL liquid
	750 mg Tablet	300 mg/tab
	1250 mg Capsule	500 mg/cap
	1500 mg Tablet	600 mg/tab
Calcium Glubionate	360 mg/mL Liquid	23 mg/mL liquid
Magnesium Oxide	140 mg Capsule	84.5 mg/cap
	400 mg Tablet	241 mg/tab
Mag Sulfate	500 mg/mL oral soln.	49 mg/mL
Mag Carbonate	200 mg/mL oral soln.	10.8 mg/mL

Combination Electrolyte	Phos Content	K Content	Na Content
Phos-NaK (Powder Packet)	250 mg (8 mmol)	7.1 mEq (278 mg)	7.1 mEq (164 mg)
Phospha 250 Neutral (Tablet)	250 mg (8 mmol)	1.1 mEq (45 mg)	13 mEq (298 mg)
Potassium Phosphate (Oral Solution)	93 mg/mL (3 mmol/mL)	4.4 mEq/mL (170 mg/mL)	None

	Intermittent IV repletion			Continuous IV repletion		
Electrolyte	**Dosage**	**Infusion Time**	**Max Concentration**	**Max rate**	**Max *PIV* Conc.**	**Max *CVL* Conc.**
Potassium Chloride	0.5-1 mEq/kg/dose (Max 30 mEq/dose)	2 hr (Max 0.5 mEq/kg/hr)	PIV: 0.1-0.2 mEq/mL CVL: 0.5 mEq/mL	0.25 mEq/kg/hr (Max 7.5 mEq/hr, w/o ECG monitoring)	80 mEq/L	200 mEq/L
Potassium Phosphate (For low K, consider KCl over KPhos)	0.1-0.4 mmol/kg/dose (Max 15 mmol/dose)	6 hr (Max 0.06 mmol/kg/hr)	PIV: 0.05-0.07 mmol/mL CVL: 0.12-0.17 mmol/mL	0.06 mmol/kg/hr	50 mmol/L	120 mmol/L
Calcium Gluconate	100 mg/kg/dose (Max 2 g/dose)	1 hr (Max 100 mg/min)	PIV: 20 mg/mL CVL: 50 mg/mL	100 mg/min	4 g/L	50 g/L
Magnesium Sulfate	25-50 mg/kg/dose (Max 2 g/dose)	2 hr (Max 125 mg/kg/hr)	PIV: 125 mg/mL CVL: 125 mg/mL	125 mg/kg/hr	2 g/L	Start at 2 g/L. May titrate up based on Mg level

Hepatology Pearls		
Physiologic differences:	**Specific Pathology red flags**	**Meds to avoid**
- Fever: lower threshold even than Onc. >38Cx1. - Sources for infection: ascites, biliary tree - Common to have low diastolic BPs and wide PP. Don't need to bolus. - Normal Na can be high 120s-low 130s (esp if on diuretics). Lower Na= more advanced cirrhosis - Hepatic metabolism of drugs is altered	- Esophageal varices→ UGIB. Tx: IV pantoprazole, drip octreotide (ICU), CTX, access, T&S, CBCd, - Ascites w/ fever → SBP. Tx: CBCd, BCIx, Abx (often zosyn), tap (use cell counts to diagnose- don't hold abx for culture) - Vitamin Deficiencies → A deficiency results in hemolytic anemia (high bili load, anemia).	- NSAIDS (risk of plt inhibition, hepatorenal syndrome, AKI). Alternative= acetaminophen intermittent (don't order PRN). - Normal Saline (too high of sodium load, ascites). Alternative= ½ NS (older kids) vs ¼ NS (infants, young kids). Oral and NG are preferred to IV (ascites, fluid overload risk). Consider ¾ mIVF w/ dextrose. - Benzos (not metabolized, overly sedating)- rarely ever ordered - Narcotics (metabolized slower)- much smaller doses required

LFT Patterns			
Hepatocellular: ALT (specific), AST, LDH >>> ↑ GGTP, alk phos, bilirubin	**Cholestatic**: ↑ Alk phos, GGTP & Direct Bili >> AST, ALT	**Infiltrate**: ↑ Alk phos w/ nl bili (send GGT to determine if from liver or bone)	**End stage:** ↓ Alb, PT, PTT, factor VII, V & serum lipids

Hematology

<table>
<tr><th colspan="2">Transfusions</th></tr>
<tr><td>Normal Hematologic Lab Values: see Nathan and Oski's textbook available online. Varies by age.
Blood Products/Transfusion
● pRBCs: 10-15 mL/kg (max rate: 5 mL/kg/hr). Order by mL or unit (1 unit = 300-350 mL)
● Severe anemia: 5cc/kg over 4 hours (repeat as needed)
○ Expected increase: 3 mL/kg = Hb 1 g/dL & Hct 3%. 10 mL/kg = Hb 3 g/dL & Hct 10%
○ Quick formula for pRBC desired mL = estimated blood volume (80 mL) x kg x (goal Hct - current Hct) / Hct of pRBCs (60%)
● Platelets: goal 5-10 mL/kg over 1 hr. 1U (0 - <12 kg), 3U (12-36 kg), 8U (36-96 kg)
● FFP: 10-15 mL/kg over 1 hr. Expected increase of factor levels by 15-20%
Cryoprecipitate 1-2 U/10 kg over 1 hr. Expected increase of fibrinogen of 60-100 mg/dL</td><td><u>Blood products preparation</u>
Irradiated = BMT, Hem/Onc, T-cell immunodeficiency, sometimes neonates. Washing = prior anaphylaxis/severe allergic reaction or hyperkalemia. Leukoreduction = chronically transfused. BCH does this for all cell-containing products except granulocytes and stem cells.

<u>Transfusion in SCD:</u>
Leukodepleted irradiated pRBCs, goal: Hct 28-33%. Consider if Hb drop >2 g/dL from baseline, low reticulocytes, ACS, pre-op, splenic sequestration. Indications for exchange transfusion: severe ACS, acute stroke or TIA, pre-op w/ hx of severe ACS, chronic transfusion protocol w/ elevated ferritin.</td></tr>
<tr><td><u>Transfusion reactions:</u>
Allergic reaction: from plasma proteins. Pre-med w/ antihistamine, if severe, hydrocortisone 2 mg/kg (max 100 mg) q6h x 2 doses. Consider washed product
Fever: from cytokines from WBCs or platelets. Use antipyretic
Acute hemolytic reaction: Intravascular. Within 6 hours (usually minutes). ABO incompatibility. Stop transfusion
Delayed hemolytic reaction: Extravascular. 3-10 days after. Sx: fever, jaundice, decreased Hct. Not life-threatening
TRALI: non-cardiac pulm edema (resp Sx, fever, CXR infiltrates). 2-4 days of supportive care, can be life-threatening
TACO: intravascular volume expansion CARDIAC pulm edema and decompensation. Diuretics + oxygen.</td><td><u>Antibody testing w/ antihuman IgG (Coombs reagent):</u>
Direct Coomb's Test: positive when patient's RBCs coated w/ Ab agglutinate in the presence of Coombs reagent that binds to the Ab on RBCs.
Indirect Coomb's Test: positive when patient's Ab-containing serum binds donor RBCs so when Coomb's reagent is added, agglutination occurs.</td></tr>
</table>

Platelet Disorders	
<u>Causes:</u> Immune-mediated (ITP, neonatal alloimmune thrombocytopenia, HIT/drug-induced). Non-immune: (MAHA, DIC, vascular malformation, HLH, type 2b vWD, cardiopulm bypass, thrombus, many genetic conditions w/ decreased production.	<u>ITP:</u> ASH 2019 guidelines recommend outpatient management, observation only if no or minor bleeding, steroids (rather than IVIG or anti-D) if non-life-threatening bleeding. For life-threatening bleeding, consider IVIG, steroid, continuous platelet transfusion, Romiplostim 5-10 ug/kg, splenectomy.

Neutropenia		
<u>General work up:</u> Hx includes duration/periodicity, serious infections, drugs/toxins, family history. Labs: CBCd (often 3x in 1 mo), LDH, uric acid, alk phos. Consider IgG/A/M, HIV, T+B subsets, gene sequencing.	<u>Acquired causes:</u> secondary to infections or drugs, chronic benign neutropenia (onset 2-24 mo, resolution by 4-5 yo; ER visits not needed), autoimmune (treat underlying disorder, ER visits not needed), alloimmune (profound in neonates up to 3 mo, GCSF), chronic idiopathic neutropenia (mod to severe w/ infections, AYA population, GCSF)	<u>Congenital causes:</u> severe congenital neutropenia (early infancy, severe bacterial infections, GCSF to keep ANC >1000), benign ethnic neutropenia (ANC 800-1200, no increased risk of infection, often African, Middle Eastern or West Indian descent), many syndromic disorders (e.g, cartilage hair hypoplasia, SDS), cyclic neutropenia (fever, pharyngitis, ulcers, periodontitis q21 days), qualitative disorders with normal or high ANC (e.g., CGD, leukocyte adhesion deficiencies, hyper IgE syndrome)

Anemia by MCV

Iron Studies	IDA	ACD	Sideroblastic	Thalassemia
Iron	Low	Low	High	Normal
TIBC	High	Low	Low or normal	High or normal
% saturation	Low	Low or normal	High or normal	Normal
Ferritin	Low (<50)	High or normal	High	Normal

* transferrin saturation = Fe/TIBC, <10% in IDA. Mentzer index: MCV/RBC, < 13 in thalassemia, > 13 in IDA.

Anemia Approach: Hx includes family hx (transfusions, gallstones, splenectomy), diet (pica, milk intake, vegan, malnourishment), drugs, travel, blood loss, lead exposure. MCV and reticulocyte count, +/- hemolysis labs (LDH, bilirubin, haptoglobin, UA), iron studies, Hb electrophoresis, lead, NBS.	Hemolytic Anemias **Extravascular:** HUS, TTP, DIC, artificial valves, warm antibody (IgG) immune-mediated (idiopathic, rheum, drug-associated), tick exposure, hemoglobinopathies, membrane defects, enzyme deficiencies, hypersplenism **Intravascular:** cold agglutinin (IgM; associated w/ mycoplasma, EBV), PNH **Mixed:** transfusion reactions (acute vs delayed), drugs/toxins, malaria	Elemental iron dose (salt from dose): ● Severe anemia 4-6 mg/kg/day (20-30 mg/kg/day) divided TID. ● Mild/moderate anemia 3 mg/kg/day (15 mg/kg/day) divided daily or BID. ● Prophylaxis 1-2 mg/kg/day with max 15 mg (5-10 mg/kg/day with max 75 mg). ● Adult dosing prophylaxis 325 mg ferrous sulfate daily, treatment dosing 2-4 x/day.

Sickle Cell Anemia

Supportive care: ● IV + PO = 1-1.25xM (or 0.75xM if ACS) ● bowel regimen ● incentive spirometry, +/- BiPAP if ACS hx ● albuterol if asthma hx Fever: (> 3mo old + 101.5F) ● Eval: CBCd, retic, blood Cx, +/- UA/UCx (if M <6m; F <2y), +/- CXR (decreased O2, resp Sx) ● Abx: Ceftriaxone 50 mg/kg IV or IM q24 (max 1g) ● Other Abx: azithromycin 12 mg/kg/dose IV or PO q24h (+CXR or resp Sx), vancomycin 15-20 mg/kg/dose q8h (septic shock, CNS) ● * See BCH ED pathway for management and admission criteria	Pain management **NSAIDs:** ketorolac x 72 hours, then ibuprofen **PCA: Morphine** 0.015 mg/kg/hr basal (usual max 1 mg), 0.025 mg/kg bolus (usual max 1.8 mg) or **hydromorphone** 0.003 mg/kg/hr basal (usual max 0.2 mg), 0.005 mg/kg bolus (usual max 0.3 mg); lockout times 6-12 minutes +/- naloxone drip if n/v or pruritis: 0.001 mg/kg/hr Increase basal rate by 10-20% for uncontrolled pain, goal basal rate ⅔ of total use **IV analgesics: morphine** 0.05-0.1 mg/kg/dose q4h or **hydromorphone** 0.015 mg/kg/dose q4h (starting max dose 1 mg/dose) **Oral analgesics:** **Morphine** (immediate): 0.2 - 0.5 mg/kg q2-4h (max 60 mg/dose) or morphine (sustained; MS contin) 0.3-0.5 mg/kg q8-12h (max 60 mg/dose)**. Oxycodone** 0.1-0.2 mg/kg q4-6h (max 10-15 mg/dose)**.** **Hydromorphone** 0.03-0.08 mg/kg q3-4h (max 2-4 mg/dose) * See BCH and BMC ED pathways for ED pain management and admission criteria.

Bone Marrow Failure

DDx of pancytopenia: inherited BMF syndromes (e.g., SDS, FA, DBA, dyskeratosis congenita), infections (EBV, parvo, TB, sepsis, HIV, CMV, HHV6), immunologic (SLE, autoimmune, CVID, CTLA4), infiltrative (heme malignancy, storage diseases, histiocytic disorders, metastases), marked, nutritional deficiencies, drugs/toxins, idiopathic or acquired aplastic anemia Important history: family history of early deaths, fetal loss, cancer, transfusions, FTT. Ask about recurrent infections, congenital anatomic anomalies, developmental delay	Important PE: short stature, dysmorphic facies, radial anomalies, hyper/hypopigmented skin, dystrophic nails Work up: Depends on suspicion but may include CBCd, chemistry and LFTs, Hb electrophoresis, immunologic studies, nutritional labs, Fanconi screen, BMA/Bx, viral studies, pancreatic isoamylase, genetic testing

Bleeding Disorders

Platelets	PT	PTT	Differential
WNL	Prolonged	WNL	Moderate liver dx, warfarin, vit K deficiency, factor VII deficiency
WNL	WNL	Prolonged	Factor VIII, IX, XI, XII deficiencies, vWD, heparin, lupus anticoagulant
Decreased	Prolonged	Prolonged	DIC, coagulopathy from large venous malformations, HLH
WNL	Prolonged	Prolonged	Severe liver dx, warfarin, vit K deficiency, heparin, Factor II, V, or X deficiency
WNL	WNL	WNL	Not a bleeding disorder, vWD w/ adequate VIII levels, qualitative platelet dysfunction, factor XIII deficiency

* 1:1 mixing study ddx between factor deficiency (corrects) and presence of inhibitor (doesn't correct). Lupus anticoagulant not associated w/ bleeding but can be prothrombotic.

Hemophilia A (VIII) and B (IX): A more common than B. Hallmark of hemarthrosis and soft tissue bleeding. Complicated by arthropathy, CNS bleeds, inhibitor development from treatments. Standard of Care for children is initiation of factor prophylaxis in toddlers prior to onset of recurrent hemarthrosis. Bleed/injury evaluation and management for minimal bleeding (e.g., epistaxis, subcutaneous, mouth) = none or TXA. Everything else requires factor replacement for varying goal levels of factor correction (25-50% for minor bleeds up to 100% for major or life-threatening bleeds).	**vWD**: Evaluation starts w/ vWF panel measuring vWF Ag, vWF activity (RCoF), VIII level to determine subtype. Treatment varies on indication (minor/major procedure, severity of bleeding episode) but may include DDAVP, vWF concentrates, or anti-fibrinolytic therapy (e.g, Amicar, TXA), combination OCPs (for menorrhagia).

Thrombophilia and Thrombosis	
Evaluation after VTE: None if provoked (e.g., central line). Send if unprovoked (APLA panel prior to anticoagulation INITIATION of therapy; protein C, S, antithrombin, factor V Leiden, prothrombin prior to DISCONTINUATION.	Anticoagulation: Generally enoxaparin in children (though can be unfractionated heparin, warfarin, and less commonly DOACs). 3 mo for provoked, may transition and keep on ppx dosing if persistent VTE after treatment period. Generally 6-12 mo for unprovoked with consideration of indefinite treatment for APLA syndrome, recurrence, and/or life-threatening VTE. Enoxaparin stand risk goal level 0.5 to 1, high risk bleed level 0.4 to 0.6.

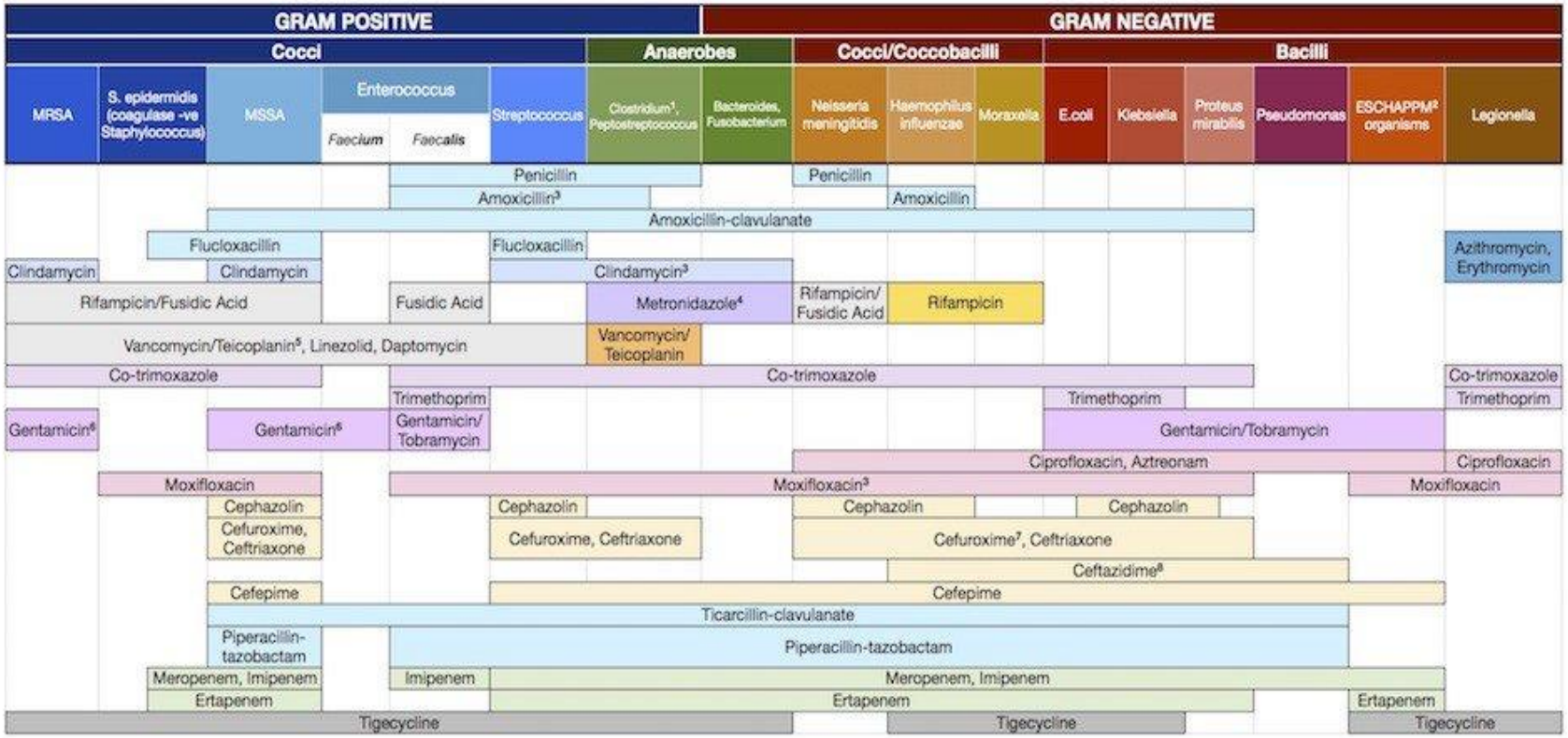
Lead poisoning

PCP screen: CBC (causes sideroblastic anemia) & Pb level 9-12mo, 1y, 2y, 3y, and 4y, or new to U.S.

Capillary Pb level testing: failed screen, sibling w/ failed screen, pica, development/behavior disorders, severe anemia, ingestion, parent occupation, home renovation

	BMC	BCH
2-4	Retest in 6-12mo. Environmental Hx, lead safe education.	Retest in ~ 12 mo or at regular testing times (9-12m, 2y, 3y, 4y). Environmental hx (CPG risk assessment), nutrition recs for Ca/vit D/iron/vit C/Mg (CPG parent nutrition handout)
5-9	Steps above + retest in 1-3 mo & nutrition recs for Ca/vit D/iron. Add "elevated lead level" to problem list. CBC, ferritin, CRP. Iron supps if needed. Test sibs < 6 yo.	Steps above + CBC, iron studies, retic. Iron supps if needed (2 mg/kg ferrous sulfate BID). Retest lead AND anemia studies in 1-2 mo.
10-19	Steps above + add "lead poisoning" to problem list. Refer to lead clinic. Steps above + repeat level in 1-4 weeks.	Steps above + MDPH mandated home inspection/remediation. Retest in 2-4 weeks.
20-44*	Steps above + PE, consider KUB. Consider chelation Tx w/ PO succumer in consultation w/ lead specialist.	*NOTE: 25-44 for BCH. Steps above + page env health physician. Additional labs: zinc protoporphyrin. Obtain KUB, if + refer to ED for bowel decontamination, if - refer to BCH environmental health center. Repeat labs.
45-69	Steps above + consider hospitalization for IV chelation Tx w/ EDTA. Retest within 48 hrs.	Steps above + complete neuro exam (assess for lead encephalopathy). Refer to ED for eval and admit to hospital for chelation Tx. Additional labs: chem7, LFTs, UA.
70+		Steps above + IMMEDIATE page to env health physician, URGENT ED referral for ICU admit.

ANTIBIOTIC SUSCEPTIBILITIES IN INTENSIVE CARE*



For simplicity, atypical organisms are not included above. Partial columns indicate incomplete coverage. ESBL-producing organisms are not susceptible to most antibiotics containing a beta-lactam ring; carbapenems are the usual agent of choice.
1: *C. difficile* should only be treated with metronidazole or vancomycin. **2**: ESCHAPPM are β-lactamase producing organisms. These are *Enterobacter, Serratia, Citrobacter freundii, Hafnia, Acinetobacter/Aeromonas, Proteus* (**not** *mirabilis*), *Providencia & Morganella morganii*.
3: Not effective against *Clostridium*. **4**: Metronidazole is not effective against *Peptostreptococcus*. **5**: Teicoplanin is not effective against *Enterococcus faecium*. **6**: Gentamicin is not appropriate mono therapy for *Staphylococcus aureus* & should only be used in conjunction with a β-lactam.
7: Due to increasing MIC, Cefuxorime is not recommended therapy for *Moraxella*. **8**: Although it has other actions, Ceftazidime should only be used for *Pseudomonas*.

ANTIBIOTIC CLASS KEY

PENICILLINS	LINCOSAMIDE	MACROLIDES	NITROIMIDAZOLE	RIFAMYCIN	GLYCOPEPTIDES
SULFONAMIDES	AMINOGLYCOSIDES	FLUOROQUINOLONES	CEPHALOSPORINS	CARBAPENEMS	GLYCYLCYCLINE

*This chart is intended as a guide, pending specific identification & sensitivities - **it does not replace expert ID advice. Local antibiotic sensitivities & preferences will vary.**

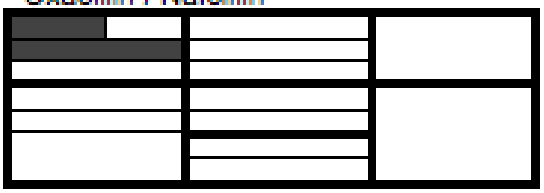
v2h 2017-09 @pcrides Taken from Appendix 5, Wellington ICU Drug Manual, Wellington, New Zealand

Common Antimicrobial Quick Reference (first line antibiotic selections)	
Acute Otitis Media (AOM)	● Amoxicillin 45mg/kg BID (max 3g/d) x 10d for <2y, 7d for 2-5y, 5-7d for 6y and older ● If >6mos w/ no severe symptoms (fever, ear pain >2d) can choose close surveillance (exception: <24mos w/ bilateral AOM). ● If penicillin allergy (non-anaphylactic): cefdinir 14mg/kg daily (max 600mg/d) OR cefuroxime 15mg/kg BID (max 1g/d) ● CTX 50mg/kg daily IM/IV x 3d if not able to take oral antibiotics
Acute sinusitis	Amoxicillin-clavulanate 45 mg amox/kg/dose PO BID (1g) x 10 days (ES-600 formulation, 600mg/5mL amoxicillin component).
C. diff	Metronidazole 10 mg/kg/dose PO QID x 10 days
Cellulitis,	Nonpurlent: Cefazolin 25 mg/kg/dose IV q8h (1g) OR cephalexin 25 mg/kg/dose PO TID (1g) x 5-7 days Purulent or Abscess: TMP-SMX 6 mg TMP/kg/dose IV/PO q12h (160 mg) x 5-7 days
Community-acquired pneumonia (CAP)	● Outpatient: Amoxicillin 30 mg/kg/dose PO TID (500 mg-1g) x 7 days (cephalosporin if mild PCN allergy, clindamycin OR levofloxacin if severe PCN/ cephalosporin allergy). ● If ill/ requires ICP/ICU: CTX 50 mg/kg/dose IV q24h (2g). Add vancomycin 15-20 mg/kg/dose IV q6-8h (1g) for severe or worsening symptoms. ● If suspect atypical organism, add azithromycin 10mg/kg day 1, 5mg/kg daily days 2-5. ● If associated influenza, add oseltamivir for 5d. ● Hospitalized (on the floor): ampicillin 50 mg/kg/dose IV q6h (2g) x 7 days w/ or w/o atypical organism coverage. Treat influenza if associated. ● Aspiration PNA: Ampicillin-sulbactam 50 mg amp/kg/dose IV q6h (2g) x 7 days
Deep neck infections	Ampicillin-sulbactam 50mg ampicillin/kg/dose IV q6h (2g) x 10-14 days
Fever of Unclear Source (non-sepsis)	Cetriaxone 50 mg/kg/dose IV Q24h (2g)
Mastoiditis	Empiric vancomycin 15mg/kg IV q6h or linezolid 10mg/kg IV q8-12h until able to narrow based on culture data x 7-10d IV
Meningitis	● 0-14day: Ampicillin 75-100 mg/kg/day (divided q6-q8h) AND ceftazidime 50mg/kg q8-12h. Add acyclovir if pleocytosis on LP, and transition ceftazidime to cefepime. ● 14d-1month Ceftriaxone for empiric treatment*. Add ampicillin and acyclovir if pleocytosis on LP, and transition CTX to cefepime. ● 1-3months: CTX 50 mg/kg/dose q12h AND vanc 15mg/kg/day q6h (goal trough 15-20) ● >3months: CTX 50 mg/kg/dose q12h AND vanc 15mg/kg/day q6h (goal trough 15-20 ● HSV risk factors: Include HSV coverage (Acyclovir 20 mg/kg/dose q8h) if HSV risk factors above are present. * Contraindications for ceftriaxone in neonates: - Patients < 44 wks PMA w/ hyperbilirubinemia - Patients < 44 wks PMA receiving calcium-containing intravenous fluids or parenteral nutrition), then amp + ceftazidime or cefepime
Orbital Cellulitis	Ampicillin-sulbactam 50mg ampicillin/kg/dose IV q6h (2g) x 14-21 days (can transition to Augmentin)
Periorbital cellulitis:	Ampicillin-sulbactam 50mg ampicillin/kg/dose IV q6h (2g) x 5-7 days (can transition to Augmentin)
Osteomyelitis	Cefazolin 50 mg/kg/dose IV q8h (2g) x 4wks
Strep pharyngitis	Amoxicillin 50 mg/kg daily (1g) x 10 days OR PCN G benzathine IM x 1 (weight based dosing). If PCN allergy, cephalexin 20mg/kg BID x 10d
PID	Outpatient: Ceftriaxone 50 mg/kg/dose IM x1 (250mg) + Doxycycline 2.5 mg/kg/dose PO BID (100 mg) x 14 days + Metronidazole 10 mg/kg/dose PO BID (500 mg) x 14 days Inpatient: Cefoxitin 40 mg/kg/dose IV q6h (2g) + Doxycycline IV/PO 2.5 mg/kg/dose PO BID (100 mg)
Pyelonephritis	Ceftriaxone 50 mg/kg/dose IV q24h (2g) x 10 days
Sepsis	Previously healthy: CTX 50 mg/kg/dose IV Q24h (2g) PLUS vancomycin 15-20 mg/kg/dose IV q6-8h (1g) Hospital onset, comorbidity: cefepime 50 mg/kg/dose IV Q8h PLUS vanc 15-20 mg/kg/dose IV q6-8h (1g)
Septic arthritis	Cefazolin 50 mg/kg/dose IV q8h (2g) x 3wks
Strep Pharyngitis/Tonsillitis	Amoxicillin 50 mg/kg/dose daily x 10 days (1 g) x 10 days
UTI	3-23 mo, febrile, healthy, outpatient: Cephalexin 25 mg/kg/dose TID (500 mg) x 10 days >24 months, healthy, outpatient: Cephalexin 25 mg/kg/dose PO TID (500 mg) x 3-5 days

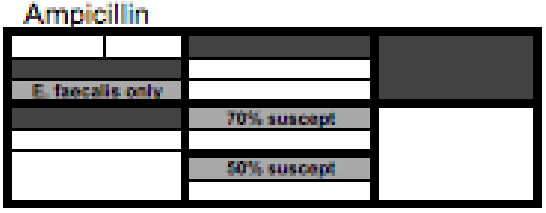
	Gram Positive		Gram Negative	Anaerobes
Cocci	S.aureus	SNA	Meningococcus	Above the diaphragm
	Strep		Gonococcus	
	Enterococcus		Moraxella	
Rod	Listeria		H. influenza	Below the diaphragm
	Corynebacterium		Pseudomonas	
	Bacillus		E. coli	
			Enterics	

Penicillin

Oxacillin / Nafcillin

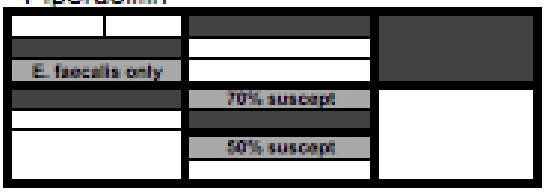


Ampicillin

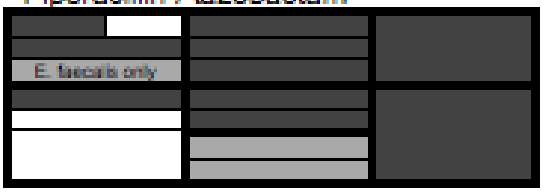


Ampicillin / sulbactam

Piperacillin

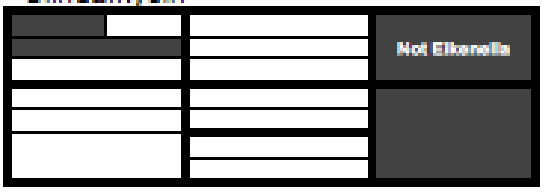


Piperacillin / tazobactam

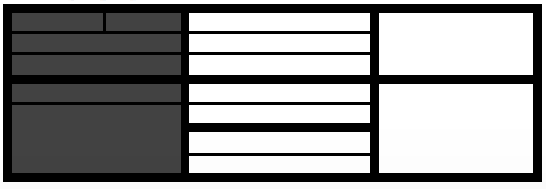


Vancomycin

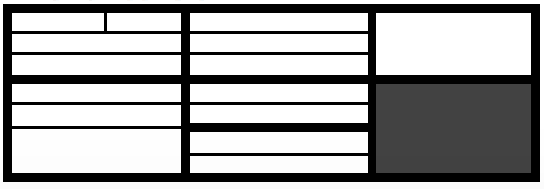
Clindamycin



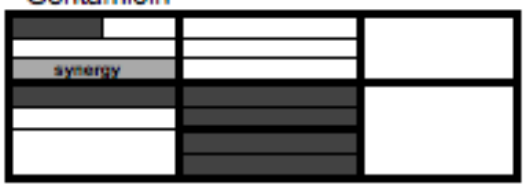
Linezolid



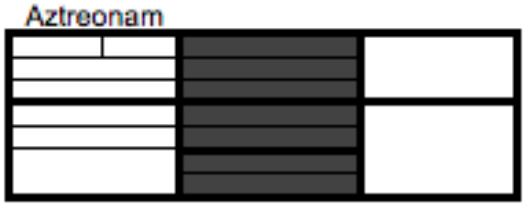
Metronidazole



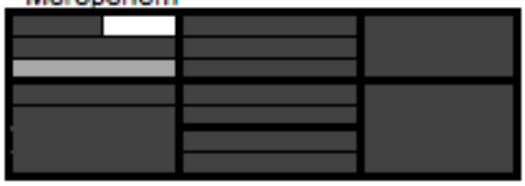
Gentamicin



Aztreonam



Meropenem



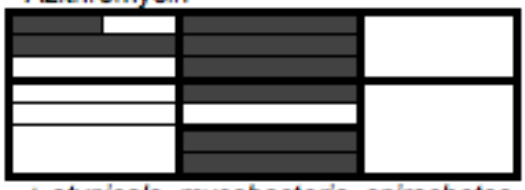
Ciprofloxacin

+ atypicals (2nd line)

Moxifloxacin

+ atypicals (2nd line), mycobacteria

Azithromycin

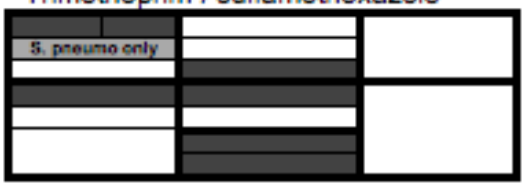


+ atypicals, mycobacteria, spirochetes

Doxycycline

+ atypicals, mycobacteria, spirochetes, and plasmodium

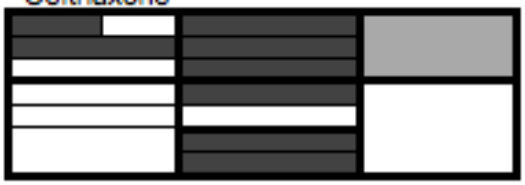
Trimethoprim / sulfamethoxazole



+ Pneumocystis, Burkholderia

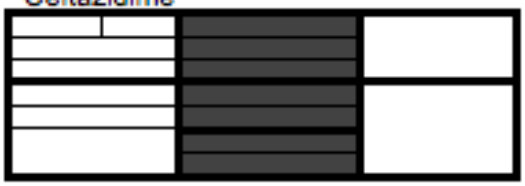
Cefazolin

Ceftriaxone



+ spirochetes

Ceftazidime



Metabolism

Approach
- ABCs, sick vs. not sick
- Page metabolism STAT - we will give you immediate next steps in terms of stabilizing measures (e.g. IVF, medication recs), labs/other work up

Patient has a known diagnosis? Look for a critical contingency plan in Powerchart
- Look for "Critical Contingency" on top blue bar --> Hover over this to see what date contingency note was created --> find this note in documents
- In the ED, find the relevant order set in **ED Metabolism Plan**
- On admission, select the relevant **Admit Plan**
- Read clinic notes, ask the family if they have a “sick day plan,” which often include a modified formula plan which has less of the potentially toxic substrate

Abnormal Newborn Screen? Page metabolism
- Immediate next steps can be found in ACT sheets (https://www.acmg.net/ACMG/Medical-Genetics-Practice-Resources/ACT_Sheets_and_Algorithms.aspx)

History/Physical
- Identify the trigger: infection (commonly viral), fever, other illness, dietary or medication non-compliance, fasting, other recent catabolic stressor such as surgery, trauma, vaccination, extensive exercise, new medications
- Ask about symptoms pertinent to the specific disorder, if known (for example: hyperammonemia presents with AMS, seizures, lethargy, other symptoms of increased ICP while fatty acid oxidation defects present with symptoms of hypoglycemia)
- Ask about whether they brought their home medications and formula (both their regular "well" formula and their "sick day" formula)

Seizures
- Many can’t miss metabolic disorders (e.g. urea cycle defects, fatty acid oxidation defects) can present with seizures. Work up involves special testing; if standard workup for seizures is unrevealing, consider metabolism consults

Peroxisomal Disorders X-Linked Adrenoleukodystrophy	**Mitochondrial Disorders**

• Pathophys: X-linked mutations in ABCD1 gene whose protein likely helps transport VLCFAs into the peroxisome where they are normally degraded. Dysfunctional protein leads to extra-peroxisomal accumulation of VLFCAs which can be toxic to tissues, particularly the nervous system and adrenal glands
• Presentation: various subtypes with different ages of onset; presents with chronic neuropsych symptoms and symptoms of adrenal insufficiency (e.g. tan skin, hypoglycemia, GI upset, weakness, dizziness)
• Diagnosis: elevated plasma VLCFAs, particularly C26:0. ABCD1 gene sequencing
• Management
  ○ Treat adrenal insufficiency
  ○ HSCT
  ○ Trials are investigating gene therapy, anti-oxidant therapy
	• Dextrose containing fluids: goal is to provide enough glucose to provide anabolic support without providing too much which can precipitate dangerous lactic acidosis (mitochondrial dysfunction means dysfunctional TCA cycle and electron transport chain; therefore excess glucose --> pyruvate will be shunted to lactate)
	• Don’t give valproic acid (impairs mitochondrial function) or Lactated Ringers (avoid lactic acidosis).

Amino Acid Disorder & enzyme physiology	**Presentation**	**Treatment**
Phenylketonuria Blocked Phenylalanine hydroxylase (Phe → Tyr). Accumulates Phenylalanine	Neurotoxicity, intellectual deficits, microcephaly, GDD, eczema Note that PKU does not cause acute metabolic decompensation	Avoid Phe, give special Phe-free diet, consider cofactor tx (sapropterin), enzyme substitution (adults)
Maple Syrup Urine Disease Blocked Branched-chain alpha-keto acid dehydrogenase. Accumulates BCAAs: Leu, Ile, Val. Leu is neurotoxic, causes hypoNa	Catabolic stress, high Leu intake → HA, confusion, hallucination, lethargy, N/V → coma/death	Stop all Leu, give Leu-free feeds, consider dex-containing IVF, AVOID hypotonic fluids (cerebral edema)
Homocystinuria Blocked cystathionine β-synthase Accumulates Homocysteine (pro-thrombotic), Methionine (potentially acute cerebral edema)	FTT, GDD, ID, seizures, psychiatric illness, marfanoid body habitus, premature atherosclerosis, "down and in" lens subluxation, myopia and glaucoma later in life, kyphoscoliosis, osteoporosis, thromboembolism	• Met-free formula, B6 (cofactor for cystathionine β-synthase) in responsive patients, B9, B12, betaine (Hcy → Met) • risk of cerebral edema with rapid methionine correction
Tyrosinemia Blocked Fumaryl- acetoacetase (fumaroaceto-acetate → fumarate + acetoacetate) Accumulates Tyrosine (blood), Succinylacetone (urine)	Liver failure, RTA - due to accumulation of succinylacetone	Nitisinone (blocks early step in Tyr metab - can’t make succinylacetone), Tyr restriction

Hyperammonemia

Neonates:
- Healthy: NH3 <110 µmol/l
- Sick: up to 180 µmol/l
- suspect metabolic disease with NH3 >200 µmol/l

After neonatal period:
- Normal: <50 µmol/l
- suspect metabolic disease with NH3 >100 µmol/l

Pathophys: high levels of ammonia cause neurotoxicity and cerebral edema.

Presentation: Headaches, irritability, nausea/vomiting, central hyperventilation/resp alkalosis, lethargy, delirium, seizures, AMS, coma, increased ICP, brain herniation, death.

Diagnosis: free-flowing sample in Na heparin tube w/o tourniquet, send to lab on ice STAT

Management of hyperammonemia:
- Reverse the underlying cause
- Ammonul (IV Na-phenylacetate & Na-benzoate) and Ravicti (PO glycerol phenylbutyrate) provide substrates to react w/ NH3 to create alternative metabolites that are eliminated in the urine instead of urea cycle
  - Ammonul: only ordered by metabolism for severe hyperammonemia- can cause a salicylate-like poisoning, death.
- Dialysis (severe cases)
- Long term: Low-protein diet, avoid catabolism, include missing UC intermediates, liver transplant

Differential:
- Metabolic: urea cycle disorders, organic acidurias, long chain fatty acid oxidation defects, hyperinsulinism-hyperammonemia syndrome, hyperammonemia-hyperornithine-hyper citrulline (HHH) syndrome, systemic carnitine deficiency
- Liver failure
- Porto-systemic shunt
- Increased muscle activity (ex postictal)
- Transient hyperammonemia of the newborn:
  - From the umbilical vein, the ductus venosus bypasses the liver, so circulating NH3 is not be metabolized by the urea cycle.
  - In babies w/ prematurity or respiratory distress, the ductus venosus stays open longer which can cause hyperammonemia
- Valproic acid
- Infection with urease-positive organism (e.g., Proteus, H pylori)

Urea cycle disorders: (key examples) Ornithine Transcarbamylase Deficiency, Citrullinemia Type 1 (Arginosuccinate Synthase Deficiency), Arginosuccinic aciduria (Arginosuccinate Lyase Deficiency), Carbamoyl phosphate synthetase (CPS) I deficiency & NAGS deficiency

Most are autosomal recessive; however OTC deficiency is X-linked. Boys are at highest risk of severe disease, but carrier females can get very ill.

Hypoglycemia: Overview (see further sections for more specifics)

For a more detailed approach see Endocrine ch.
Definition: Neonates < 2 days old, see NICU chapter. After 2 days: < 70 mg/dL
Differential diagnosis:
- Ketotic Hypoglycemia
  - Disorders of carbohydrate metabolism including GSDs and disorders of gluconeogenesis
  - Disorders of ketolysis (e.g. SCOT deficiency, Beta ketothiolase deficiency)
  - Idiopathic Ketotic Hypoglycemia (aka accelerated starvation)- see sidebar.
- Non-ketotic Hypoglycemia
  - Disorders of fatty acid oxidation
  - Hyperinsulinism
  - Disorders of ketogenesis (e.g. HMG-CoA Synthase deficiency, HMG Co-A Lyase deficiency)

Idiopathic Ketotic Hypoglycemia (aka accelerated starvation)
- Pathophys: not fully understood but thought to be related to below average physiologic fasting tolerance. Risk factors: small body size and subQ fat (reduced energy stores).
- Presentation: Often presents in the 2-6 year age range with ketotic hypoglycemia, often in setting of illness which causes the child to skip a meal.
- Diagnosis: high clinical suspicion, diagnosis of exclusion
- Management: avoid fasting and known triggers. Children expected to "grow out of it" by around age 6, although it may persist into adulthood

Hypoglycemia, Disorders of Carbohydrate metabolism: Glycogen Storage Diseases		
Disorder & Pathophys	**Presentation**	**Diagnosis & Management**
GSD1 Type 1a (von Gierke). AR, enzymes include glucose 6 phosphatase and glucose 6 phosphate translocase	classically an infant a few months into life (when feeds start to be spaced out) ketotic hypoglycemia w/ other metabolic abnormalities detailed here. Type 1b may have neutropenia and an IBD-like disorder	● STAT POCT glucose, chem, BHOB, lactate, uric acid, triglycerides, free fatty acids, plasma amino acids; ● Later: consider enzyme studies, genetic testing ● Findings: ketotic hypoglycemia, AGMA (ketones, lactic acidosis), ↑ uric acid gout, kidney stones, ↑ TG, FFAs, ↑ Alanine Tx: Frequent meals, uncooked corn starch. If neutropenia in 1b, Filgrastim (G-CSF). Avoid steroids in IBD-like disease (avoid catabolic stress)
GSD Type IIa (Pompe). Defect in Lysosomal acid α-glucosidase --> glycogen accum. in skeletal & cardiac muscle	hypotonia w/ progressive loss of motor, cardiac, respiratory muscle control; macroglossia; cardiomyopathy	enzyme studies, genetic testing, other biochemical tests. Tx: ERT (alglucosidase alfa), cardiomyopathy mgt, protein rich diet with Ala + Leu supplementation
GSD Type IIIa & IIIb (Cori). Defect in debranching enzyme --> glycogen accum. in liver & muscle	similar to 1a but milder: ketotic hypoglycemia, less likely to see AGMA/hyperuricemia, ↑ TG, FFAs	similar to GSD1a. Tx: Uncooked cornstarch + continuous feeds to maintain normoglycemia, high-protein diet
GSD Type V (McArdle). Defect in Muscle phosphorylase --> glycogen accumulates in muscle	Exercise intolerance, “second wind” phenomenon (once FA mobilized by muscle), +/- rhabdomyolysis	enzyme studies, genetic testing Tx: Carbohydrate administration before exercise, high-protein diet

Hypoglycemia, Fatty Acid Oxidation Defects	
Disorders: Medium chain acyl-CoA dehydrogenase deficiency (MCAD deficiency), Very long-chain acyl-CoA Dehydrogenase deficiency (VLCHAD deficiency), primary carnitine deficiency Pathophys: ● deficient enzymes in beta oxidation of fatty acids --> Unable to extract ATP from fatty acids efficiently --> end organ damage, particularly liver and skeletal muscle, although cardiac muscle can be involved esp in longer chair defects (a/w arrhythmias) ● Causes hypoketotic hypoglycemia because fatty acids produce acetyl CoA which is used for ketone synthesis ● Hyperammonemia: accumulation of fatty acids in the liver may alter NH3 metabolism	Presentation ● Classically present with hypoketotic hypoglycemia +/- liver failure and hepatomegaly, acidosis & hyperammonemia, muscle breakdown with myalgias +/- rhabdomyolysis, +/- cardiomyopathy/arrhythmia ● there have been numerous reports of ketosis in FAODs so elevated ketones does not rule out a FAOD. Diagnosis: STAT POCT glucose, consider acylcarnitine profile, urine organic acids, chem, LFTs, NH3, BHOB, CK, troponin

Hypoglycemia, Disorders of Carbohydrate metabolism: Other	
Classic Galactosemia ● Defect in Gal-1-P Uridyltransferase (GALT) ● Accumulation of Gal-1-P→ sequester P→lack of ATP→ hypoglycemia due to lack ATP needed for glycogen breakdown/ gluconeogenesis. Liver failure in general ● Presentation classically neonate/young infant (e.g. exposed to lactose in breast milk and standard formulas) presenting with hypoglycemia, liver failure, cataracts, risk of E Coli Sepsis ● Diagnosis: GALT activity, Galactose and GAL-1-P levels, genetic testing ● Management: avoid galactose (including lactose, i.e. milk/dairy products)	**Hereditary Fructose intolerance** ● Defect in Aldolase B ● Accumulation of F-1-P→ sequester P→ lack of ATP→hypoglycemia due to lack ATP needed for glycogen breakdown and gluconeogenesis. Liver failure in general ● Presentation classically in older infant (4-6+ months) after initiation of solid foods such as fruits, juices, which contain fructose. ● Presentation similar to classic galactosemia, but no cataracts. ● Diagnosis: enzyme studies, genetic testing ● Management: avoid fructose (including sucrose and sorbitol)

Hypoglycemia, Organic Acidemias	
Key examples: Propionic Aciduria (PA), Methyl Malonic Aciduria (MMA), Isovaleric Aciduria (IVA) **Presentation**: ● AG metabolic acidosis (N/V, tachypnea) w/ increased lactate and ketones, liver dysfunction, hyperammonemia, hypoglycemia ● Possibly basal ganglia stroke, cardiomyopathy/arrhythmias, pancreatitis, pancytopenia **Diagnosis**: ● Standard labs/work up: chem, LFTs, lactate, BHOB, NH3, CBC with diff, lipase, EKG ● Metabolic labs: urine organic acids, acylcarnitine profile, F/T carnitine, plasma AAs **Management**: ● Acute: transiently stop protein intake, glucose infusion, correct acidosis, supplement with carnitine ● Long term: low protein diet, carnitine, vitamins, NH3 scavengers	Glutaric Aciduria Type 1: **Pathophys**: defect in Glutaryl-CoA Dehydrogenase--> accumulated metabolites are neurotoxic and can cause acute metabolic stroke. **Presentation**: ● Neonatal onset: Non-specific neuropsychiatric symptoms, devol delay ● Adolescent/Adult Onset p/w chronic neurodegeneration and psychiatric illness ● Subdural hemorrhages and retinal hemorrhages that can look like NAT ● Macrocephaly ● Catabolic triggers: acute stroke like episodes, neurological/encephalopathic crises, seizures, coma and death **Imaging**: frontal temporal atrophy ("bat wing appearance" of the sylvian fissures)

Lysosomal Storage Disorders

In general LSDs cause progressive end organ damage and do not p/w acute decompensation.

Disorder & Enzyme Physio.	Presentation	Treatment
Gaucher Disease Blocked β-glucosidase (glucocerebrosidase) Accumulates Glucocerebroside	Type 1:HSM, bone disease, anemia & thrombocytopenia, absence of CNS disease Type 2&3: Primarily neurologic with DD, regression, early death	ERT, substrate reduction therapy
Tay-Sachs Disease Blocked Hexosaminidase A Accumulates GM2 gangliosides	By age 1 - DD, exaggerated startle, seizures, macular cherry-red spot	Supportive
Niemann-Pick Disease Blocked Sphingomyelinase Accumulates Sphingomyelin	Massive HSM, cherry red spot; neuronopathic or non-neuronopathic	HSCT for non- neuronopathic
Krabbe Disease Blocked Galactocerebrosidase Accumulates Galactocerebroside	Infantile onset: by age 1 - irritability, rapid neurologic deterioration, early childhood death. Later-onset: variable	Early HSCT
Metachromatic Leukodystrophy Blocked Arylsulfatase A Accumulates Cerebroside Sulfate	Onset age 1-2 with spasticity, neuropathy, loss of motor skills --> optic atrophy and worsening neurodegeneration, fatal by age 3-6. Juvenile and adult forms present later	HSCT in pre-symptomatic or early symptomatic juvenile onset patients
Fabry Disease Blocked α-galactosidase Accumulates Ceramide trihexoside	X-linked recessive. 1st decade: Paresthesias, hypohidrosis and recurrent fever, angiokeratomas. Adult complications: stroke, hearing loss, cardiomyopathy, renal failure	ERT
Hurler Syndrome (MPS I). Blocked α-L-iduronidase Accumulates Glycosamino- glycans (GAGs): dermatan + heparan sulfate	Coarse facies, DD, ID, hydrocephalus, corneal clouding, hearing loss, recurrent upper respiratory infections, cardiac disease, HSM, hernias, dysostosis multiplex	ERT, HSCT
Hunter Syndrome (MPS II) Blocked Iduronate-2-sulfatase Accumulates GAGs as above	X-linked recessive. Similar to MPSI but corneal clouding is rare.	ERT, HSCT

ERT = enzyme-replacement therapy. HSCT = hematopoietic stem-cell transplant. Autosomal recessive unless otherwise specified

Nephrology

Dialysis

Indications for Acute Dialysis: "AEIOU"
- **A**cidosis: metabolic acidosis refractory to medical therapy
- **E**lectrolyte/metabolic derangements: chiefly hyperkalemia and hyperammonemia
- **I**ngestions: typically low molecular weight toxins with a small volume of distribution and low protein binding (e.g., lithium, metformin)
- **O**verload: fluid overload not responding to diuretics, or inability to provide nutrition without resulting in fluid overload
- **U**remia: symptomatic uremia manifesting with encephalopathy, pericarditis, or bleeding

Hemodialysis (HD) or **continuous renal replacement therapy** (CRRT) is typically preferred in the acute setting given the ability for rapid solute and fluid removal.
- Typically place a temporary double lumen CVC in an IJ or femoral vein
- HD is more efficient with rapid solute removal but can predispose to rebound (e.g., hyperammonemia) and causes greater hemodynamic shifts
- CRRT is less effective for rapid solute removal but has a lower risk of rebound and is preferred when hemodynamics cannot tolerate rapid fluid shifts

Peritoneal dialysis (PD) can also be performed in the acute setting (e.g., after cardiopulmonary bypass for repair of congenital heart disease).
- Works by instilling a solution with a high concentration of dextrose into the peritoneal cavity, creating an osmotic gradient for fluid and toxin removal

Electrolyte Emergencies

Hyponatremia: Main ddx: hypovolemic hypoNa vs SIADH; don't forget pseudohypoNa w/ ↑TG and hyperglycemia-related hypoNa
- If symptomatic (seizing/coma): give 3% HTS 3-5 mL/kg, repeat until symptoms resolve
- If asymptomatic: acute (<48h) vs chronic? If unknown, assume chronic.
  - Generally can fix at rate that Na became deranged
  - If corr hypoNa too quickly (>10 mEq/L/24h), risk central pontine myelinolysis
- Hypovolemic hypoNa vs SIADH:
  - In both, urine osms >300; urine Na <40 in hypovolemic vs >40 in SIADH
  - Tx of hypovolemic hypoNa – give isotonic to correct deficit + addnl losses
  - Tx of SIADH – free water restriction; Na supps drive additional UOP

Hypernatremia: Main ddx: insufficient free water vs excessive Na supplementation vs excessive free water loss (diabetes insipidus)
- If d/t dehydration, calculate free water deficit ([Current Na^+]/[Desired Na^+] – 1)*TBW (approx. 0.6*weight in kg) – restore ½ of FWD in first 24h + replace ongoing losses
- Risk of cerebral edema with overly rapid correction – no more than 10 mEq/L/24h

Hypokalemia: Main ddx: GI losses (stool, emesis), renal losses (post-obstructive diuresis, loop/thiazide diuretic use, inherited tubulopathy; check urine/serum Cr and K to calculate fractional excretion of K), states of aldosterone excess, metabolic alkalosis (trans-cellular shifts)
- Check [Mg^{2+}] – cannot replete K^+ if hypomagnesemia (can give 25-50 mg/kg IV, max 2g)
- If K >2.5/asymptomatic, give 1 mEq/kg PO, max 40 mEq/dose
- If K <2.5 or symptomatic (EKG changes), give KCL 0.5-1 mEq/kg (max 30 mEq) IV w/ EKG monitoring during infusion; check w/ charge RN re: max infusion rate allowed on unit

Hyperkalemia: Main ddx: pseudohypoK (hemolyzed sample), renal failure/decreased GFR, ↑ cell turnover (tumor lysis, true hemolysis), acidosis (trans-cellular shifts), aldosterone deficiency/resistance (Type IV RTA)

if real (non-hemolyzed) and with EKG changes:
- **STOP** K^+ supplementation, K^+-containing IVF, and K^+-sparing medications.
- **Stabilize cardiac membrane**
  - Calcium gluconate 10% @ 0.5 mL/kg (=100 mg/kg) IV over 5 min
  - Calcium chloride 20 mg/kg IV over 5-10 min if impending cardiac arrest
- **Drive K^+ into cells**
  - Insulin 0.1 U/kg, max 10U IV w/ glucose, infuse over 30 min:
    - <5 yo: D10 (100 mg/mL) @ 5 mL/kg
    - ≥5 yo: D25 (250 mg/mL) @ 2-4 mL/kg IV (max 25g)
  - Albuterol nebs
  - Bicarb: 1 mEq/kg IV (max 50 mEq) over 10-15 min
- **Excrete total body K^+**
  - Kayexalate 1 g/kg, max 50g PO/PR q4h PRN
  - Furosemide 1-2 mg/kg IV (max 40 mg or 80 mg if renal insufficiency) q6h
  - Dialysis if emergent or if ongoing source of K^+ release (TLS, rhabdo)

Metabolic Acidosis
- Calculate the serum anion gap: $Na^+ – (Cl^- + HCO_3^-)$; normal is 2.5-3.0*albumin
- If elevated AG: **MUDPILES**: **M**ethanol | **U**remia | **D**iabetic ketoacidosis/starvation ketoacidosis | **P**araldehyde | **I**nfection/**I**soniazid/**I**ron/**I**EM | **L**actic Acidosis | **E**thylene Glycol | **S**alicylates (cause primary met acidosis and resp alkalosis)
- If normal AG: main ddx diarrhea vs RTA (history!)
  - Calculate **urine** anion gap to help differentiate: Urine $Na^+ + K^+ - Cl^-$.
    - If negative: suggests NH_4^- is present in urine (unmeasured cation), RTA unlikely
    - If positive: suggests little NH_4^- is present in urine, RTA possible (see below)

Renal Tubular Acidosis: Hyperchloremic Metabolic Acidosis w/ positive Urine AG

RTAs	Proximal (Type 2)	Distal (Type 1)	Hyperkalemic (type 4)
Defect	Bicarb reabsorption	H^+	↓ aldo effect
K^+	Normal/decreased	Normal/decreased	Increased
urine pH	<5.5	>5.5	<5.5
Renal stones	No	Yes (high urine pH → CaPhos stones, low urine citrate)	No
Clinical context	Fanconi Synd. (generalized proximal tubular dysfunction → lose glucose, phos, AAs)	Lithium, autoimmune (SLE, Sjogren), ampho B, ifosfamide, hypercalciuric conditions, medullary sponge kidney	DM, primary adrenal insufficiency, use of ACEIs/aldo antagonists, CNIs (tacro/CsA), TMP/SMX

Diuretics	CA inhibitors	Loop diuretics	Thiazides	K-sparing
Examples	Acetazolamide	Furosemide, Torsemide, Ethacrynic acid (no sulfa)	Hydrochlorothiazide, chlorothiazide, chlorthalidone	Spironolactone (S), eplerenone (E), amiloride (A), triamterene (T)
Mechanism (potency)	Block carbonic anhydrase in prox tubule: HCO_3^- loss. (low)	Block NKCC in thick ascending limb. (high)	Block NCC in distal convoluted tubule. (mid)	Block aldo effect (S/E) or block ENaC (A/T) in distal nephron. (low)
Common uses	Metabolic acidosis, CSF overproduction/IIH	Fluid overload, hypercalcemia, hyperkalemia	Fluid overload, HTN, hypercalciuria, hyperkalemia	Cirrhotic ascites, aldo excess, heart failure, acne (S), DI (A)
Side effects	Metabolic alkalosis, ↓ CSF production, metallic taste	↓ serum K^+/Ca^{2+}/Mg^{2+}/ uric acid ↑ urine Ca^{2+} in (stone risk) Disrupts medullary conc gradient poss. ↑Na^+	↓ serum K^+/Na^+ ↑ serum glucose, uric acid, and Ca^{2+} ↓ Ca^{2+} in urine, helpful for stones	↑ serum K^+ Anti-androgen (S) Nephrotox (T)
Clinical Correlate	Fanconi syndrome (proximal tubular dysfunction)	Bartter syndrome	Gitelman Syndrome	type IV RTA

Neurology

Neurologic Exam

Mental status: Awake, comfortable, somnolent, obtunded. Oriented. Follow directions. Maintain attention. Fund of knowledge appropriate for age. Memory (3 word recall at 1, 5 minutes). Speech.

Motor:
- Tone (axial, appendicular), strength, abnl movements
- Reflexes
  - Biceps/brachioradialis C5/C6
  - Triceps C7
  - Patellar L2-L4
  - Ankle S1
  - Plantar reflexes L5-S1

Cranial Nerves
- CN I: smell
- CN II: visual acuity, visual fields, PERRLA, fundoscopic exam
  - normal pupil size ~3 mm in a room with adequate light
  - physiologic anisocoria: difference by ~1 mm in size
- CN III- IV-VI: H test
- CN V: facial sensation
- CN VII: facial movements (smile, grimace, cheek puff)
- CN VIII: test hearing finger rub
- CN IX-X: palate elevation, gag
- CN XI: neck rotation, shoulder shrug
- CN XII: tongue movements

Sensation
- T4 Nipple Line
- T10 Umbilicus
- L1 Femoral Pulse
- L4 Knee
- S2-S4 Perineal Region

Cerebellar/coordination
- Finger-nose-finger
- Rapid alternating hand movements
- Heel to shin test

Gait
Normal gait
Heel, tip toe, tandem gait

Romberg test

Neurologic Emergencies

Stroke
Any acute-onset focal neurologic finding including unilateral weakness/numbness, visual loss, aphasia, altered mental status, new-onset focal seizures

Suspect in trauma, vasculitis/vasculopathy, infection, tumor/malignancy, prothrombotic state, and anemia.

Arterial strokes- Sickle Cell Disease, Cardiac Disease, Vasculitis
Venous strokes- IBD, autoimmune, infections, dehydration

Work up
- Brain MRI/MRA stroke protocol, +/- MRV.
- TTE to look for cardiac etiologies
- Coags.
- Metabolic w/u for newborns

Manage
- ABC's!
- Stroke STAT – x52170
- Head of bed flat
- IVF at maintenance
- SBP 50-90th% for age.
- Euglycemia & normothermia
- Treat seizures if present
- Consider PICU admission and neurosurgical consult.
- Exchange transfusion in Sickle Cell disease

Complications: edema, herniation, hemorrhagic conversion

Status Epilepticus
Seizure lasting > 5 min or two sequential seizures w/o return to baseline in between. Refractory when resistant to multiple drugs.

Manage
- ABC's!
- Electrolytes
- Neuro consult
- Code Blue, x55555

First line Rescue Medication
- IV lorazepam: 0.05 - 0.1 mg/kg/dose (max 4 mg), OR
- Diazepam PR: (0.5 mg/kg if < 5yo; 0.3 mg/kg if 6-11yo; 0.2 mg/kg if > 11yo)

Second line: After 5-15 min, consider:
- repeat IV lorazepam AND
- Levetiracetam IV (non-neonates)
- Phenobarbital 20/mg/kg IV (neonates)

Work-up (after treatment)
POC glucose, chem, UA/blood/urine cultures if febrile, urine tox screen, AED levels, consider LP, imaging if examination is focal.
- Seizure history: baseline frequency, duration, semiology, history of status.
- Medication history: changes to medications, current AEDs, missed doses, when is next AED due.

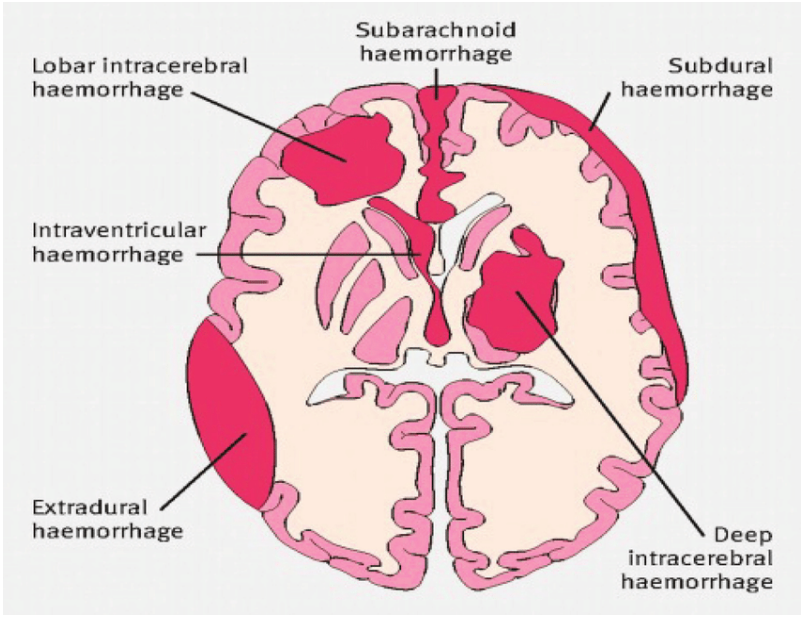
Elevated ICP
Findings: Headache, emesis, increased BP, altered mental status, delirium, confusion, unequal/dilated pupils, cranial nerve palsy, papilledema, incontinence, bradycardia, respiratory irregularity or arrest, sudden polyuria.

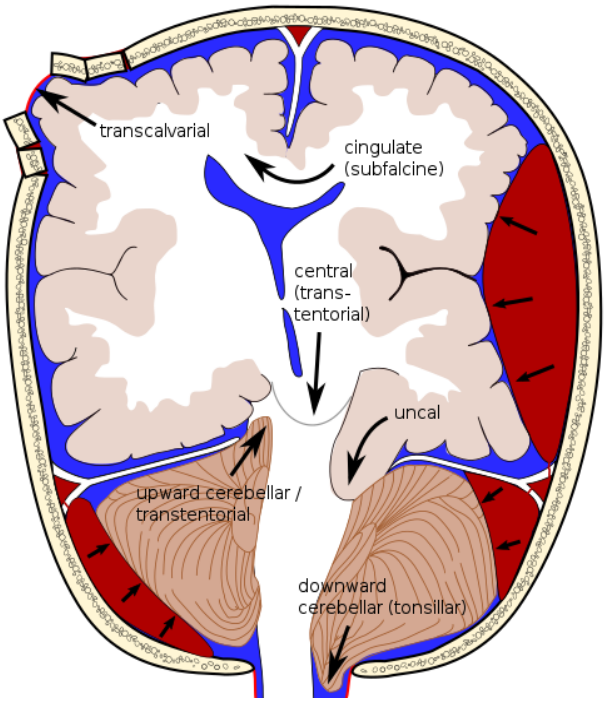
Management
- HOB to 30°
- Mannitol 1 g/kg IV over 15 min, follow UOP and VS
- Consider ETT placement for airway control, hyperventilate
- After intubation, $PaCO_2$ should be maintained between 35 and 40 mmHg
- Dexamethasone for vasogenic edema (tumors, abscesses, etc)
- Avoid hypotonic fluids, hypovolemia.
- Antipyretics to ppx fever
- Avoid hypothermia

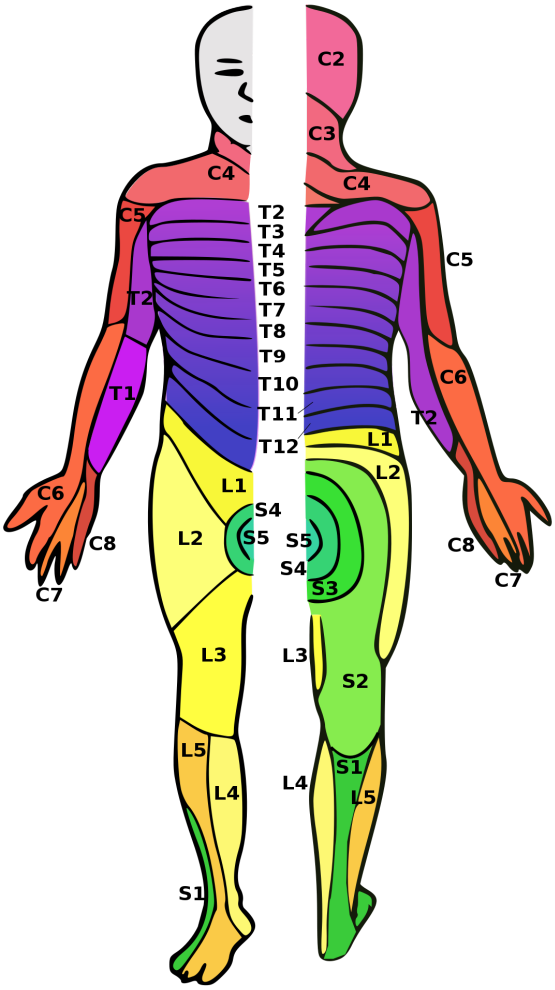
Work up (after treatment)
- Stat head CT when airway stabilized
- Neurosurgery consult

Headaches		
Type	**Characteristic**	**Red Flags**
Migraine	throbbing, pulsating pain Unilateral in 60-70% Worse with exertion, better with hydration, rest, darkness	● Acute onset ● atypical headache for patient ● neck stiffness ● worse when supine or with Valsalva ● waking from sleep ● vomiting w/o nausea or diarrhea ● focal neurologic symptoms ● altered mental status ● blurry/double vision
Tension	Bilateral pressure that waxes and wanes, usually bifrontal	
IIH: idiopathic intracranial hypertension (Pseudotumor cerebri)	Variable HA. Gradual, Some constant, some throbbing, variable location, though often retrobulbar. Worse when supine, Valsalva. Blurry vision.	
Trigeminal Autonomic headache (cluster)	unilateral, around the eye or temple. Rapid onset (minutes), pain is continuous, excruciating.	
Medication Overuse Headaches	characteristics vary, but usually preceded by another HA disorder.	

Acute Weakness Differential		
Guillain-Barré Syndrome (GBS) Antibody and complement mediated injury to myelinated peripheral nerves.	● Preceded by viral infection ● Ascending weakness ● Areflexia ● +/- autonomic dysfn	Dx: CSF w/ albuminocytologic dissociation Tx: IVIG or plasmapheresis. Consult PT.
Miller-Fisher variant of GBS Antibody-mediated (anti-Gq1b) demyelination of the cranial nerves +/- peripheral nerves.	● Preceding viral infection ● Areflexia, ataxia ● Ophthalmoplegia ● Sensorium intact.	Dx: MRI of the brain and spine LP (if no space-occupying lesion) CSF w/ albuminocytologic dissociation Tx: IVIG 2g/kg over 2-5 days
Bell's Palsy: aka idiopathic facial paralysis. Ddx: HSV, AOM, Lymes, varicella, coxsackie.	● Sudden (over hours) unilateral facial paralysis. ● Prodrome of ear pain/hearing loss	Dx: Lyme serology, consider brain imaging Tx: eye care, steroids. Consider valacyclovir.
Transverse Myelitis: neuronal inflammation w/n spinal cord resulting in rapid onset weakness, sensory alterations, and bowel/bladder dysfunction.	● Postinfectious, autoimmune ● Sudden sensory, motor, and autonomic dysfunction that are localizable to spinal cord segments. ● Initial flaccidity followed by spasticity with sensory level	Dx: MRI spine. Tx: IV steroids, +/- plasma exchange.
Myasthenia Gravis Antibody blockade of the NMJ postsynaptic ACh receptor. **Myasthenic Crisis:** inability to clear secretions or maintain oxygenation	● Fatigable weakness ● Diplopia and ptosis (provoked by sustained upgaze) ● Bulbar weakness	Dx: Ice pack for ptosis (should improve); anti-AchR, anti-MuSK antibodies; EMG; CXR to screen for thymoma. Tx: Pyridostigmine IVIG (0.4 g/kg/d x 5d) Plasmapheresis if severe Monitor FVC/NIF; Intubate if FVC< 15mL/kg, NIF< -20.
Infantile Botulism C. botulinum toxin disrupts vesicle binding to the presynaptic membrane blocking Ach release at the NMJ	● Descending paralysis ● Honey or corn syrup ● Living near construction, agriculture, soil disruptions	Isolation of organisms in stool. EMG w/ short-duration, low-amplitude motor unit potentials. Immune globulin Avoid aminoglycosides (block presynaptic NMJ)
Acute Flaccid Myelitis Inflammation of gray matter of spinal cord (anterior horn cells). Assoc w/ enterovirus D68/A71, adenovirus, flavivirus and West Nile virus. Enterovirus A71 can cause brainstem encephalitis and non-cardiogenic pulmonary edema.	● Limb weakness following non-specific viral symptoms ● Quadriparesis in 30% of cases. ● +/- UMN pattern (lateral corticospinal tracts) ● +/- bowel/bladder symptoms ● +/- cranial neuropathies.	● MRI brain and spine w/wo contrast: Spinal gray matter T2 hyperintensities ● CSF analysis (lymphocytic pleocytosis) ● Nasopharyngeal and rectal swabs for viral PCR ● anti-MOG Supportive care. Glucocorticoids, IVIG.


Subarachnoid haemorrhage
Lobar intracerebral haemorrhage
Subdural haemorrhage
Intraventricular haemorrhage
Extradural haemorrhage
Deep intracerebral haemorrhage


transcalvarial
cingulate (subfalcine)
central (trans-tentorial)
uncal
upward cerebellar / transtentorial
downward cerebellar (tonsillar)


C2
C3
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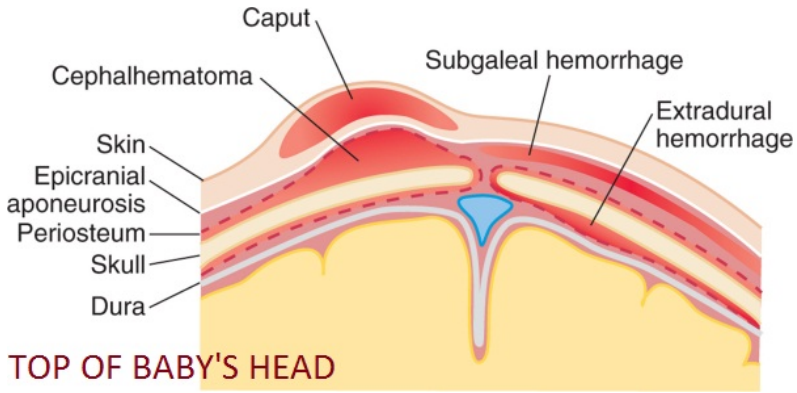

Caput
Cephalhematoma
Subgaleal hemorrhage
Extradural hemorrhage
Skin
Epicranial aponeurosis
Periosteum
Skull
Dura
TOP OF BABY'S HEAD

Neonatology/Newborn

APGARs			
	0	**1**	**2**
HR	<60	60-100	>100
Color	Blue throughout	Pink body, Blue extremities	Pink throughout
Resp Effort	No effort	Weak cry, hypoventilation	Strong cry
Tone	Limp	Some flexion w/ active movement	Active movement
Irritability	No response	Grimace	Cough, sneeze, cry

Blood Pressure Goals

- DOL 1-2: MAP= gestational age
- DOL 3- few months: MAP=GA+5
- 1+ year old: MAP = 90+2*age

Common Neonatal Respiratory Disorders		
TTN (Transient Tachypnea of the Newborn)	**RDS (Respiratory Distress Syndrome)**	**Meconium Aspiration**
Definition: retention of amniotic fluid causing neonatal respiratory distress Risk Factors: C section Diagnosis: CXR with prominent vasculature and fluid in the fissures Management: Supportive	Definition: surfactant deficiency causing neonatal respiratory distress Risk Factors: Prematurity Diagnosis: CXR w/ ground glass opacities, low lung volumes, air bronchograms Management: ● CPAP vs. intubation ● Surfactant: up to 2 doses if still intubated 12 hours after 1st dose ● Minimize FiO2	Definition: aspiration of meconium/meconium stained fluid causing neonatal respiratory distress Diagnosis: CXR with prominent vasculature and fluid in the fissures Management: supportive care +/- deep suctioning Complications: ● lung inflammation ● direct surfactant inactivation

NICU respiratory support	
Non-Invasive	**Invasive**
LFNC + HFNC Mechanism of Action: provide oxygen without pressure (some heavily debated discussion about if HFNC provides any important positive pressure, but it is a small amount of pressure at best) **CPAP** Mechanism of Action: keeps alveoli open, improves oxygenation by reducing the amount of blood shunted through atelectasis areas while the infant breathes spontaneously Note: *"bubble CPAP" is a low-tech way of providing CPAP with the outflow tubing immersed in water to provide expiratory resistance. Some studies suggest a benefit of bubble CPAP in premature neonates given the vibratory effect.* **NIPPV** Mechanism of Action: same as CPAP while also delivering PIP, so helps babies breathe off CO2 (ventilation) in addition to oxygenation	**SIMV** (Synchronized Intermittent Mandatory Ventilation) Mechanism of Action: delivers a set rate of mandatory breaths (either volume or pressure-controlled) while allowing the baby to also breathe over the set rate on its own volition. The ventilator tries to time the mandatory breaths when baby is about to inspire. Often the non-mandatory breaths are supported with pressure support Utility: wide-array of situations **A/C** (Assist / Control) Mechanism of Action: delivers a set rate of mandatory breaths (either volume or pressure-controlled). Unlike SIMV, there is no differentiation between mandatory and non-mandatory breaths Utility: best for babies that are sick / requiring maximally guaranteed and controlled breaths **Oscillator / Jet** Mechanism of Action: delivery fast "vibratory" breaths around a set mean airway pressure. Of note - the parameters on these ventilators can be quite different from conventional ventilators - eg amplitude, frequency, MAP, etc. Often the baby's chest jiggles because of how fast the breaths are. Utility: best for babies that are really sick / failing conventional ventilators (escalating oxygenation / worsening blood gases despite maximum settings). Also useful to temporize babies or allow more time with family in palliative care settings

Indirect Hyperbilirubinemia

Ddx
- ABO set-up (mother blood type O, baby blood type A or B)
- G6PD
- Sepsis
- Infants of Diabetic Mothers
- Breastmilk Jaundice (not fully understood why, something about proteins in human milk!)
- Breastfeeding Jaundice (limited caloric intake)
- Rarer causes: Gilbert, Crigler-Najjar

Management
- >35 weeks gestation: BiliTool
- <35 weeks gestation, varies by NICU

(Ptx level//exchange level)
- <28w0d 5//11
- 28w0d to 29w6d 6//12
- 30w0d to 31w6d 8//13
- 32w0d to 33w6d 10//15
- 34w0d to 34w6d 12//17

If Approaching Exchange Transfusion Management:
- Aggressive phototherapy
- Aggressive hydration (IV + PO)
- IVIG if Coombs positive
- Consider steroids

Direct Hyperbilirubinemia ddx:
- PN-associated liver injury (PNALD) - trial switching from intralipid to SMOF or omegaven
- Sepsis or TORCH infections
- Biliary atresia or other congenital biliary pathology
- Rotor, Dubin-Johnson, or other metabolic defects
- Hypothyroidism

Neonatal Cardiology

Patent Ductus Arteriosus

Definition: failure of ductal tissue to close

Frequency: 60% of infants <28 weeks

Clinical Manifestations:
- Continuous, machine like murmur
- In first few weeks of life, higher pulmonary vascular resistance favors shunting R→L across PDA - predisposing babies to desats, cyanosis, and ischemia to peripheral organs
- Once pulmonary vascular resistance begins decreasing, shunting is more likely to go L→R across PDA - which may engender pulmonary over circulation (eg decreased lung compliance, greater ventilator needs, tachypnea, ABDs) and ultimately pulmonary hypertension

Diagnosis: Echo, with attention to features like PDA diameter, velocity across PDA (higher velocity / gradient suggests narrower PDA), and aortic reverse flow

Management:
- Medical: NSAID therapy (indomethacin or ibuprofen, sometimes contra-indicated if large IVH, NEC or severe oliguria) or acetaminophen (monitor LFTs)
- Procedural: catheter-based closure (eg Piccolo) vs surgical ligation. Current evidence favors initial catheter-based closure first given the hemodynamic changes are more gradual
- Watch and wait: until the neonate is older. Around ⅔ of PDAs will close on their own by the time a neonatal reaches full-term

Neonatal EKG Red Flags

Adapted from Dr. Sharon O'Brien's Cheat Sheet for Interpreting Neonatal EKG:
- HR > 200, which could suggest SVT
- R wave > 12mm in V6 (LVH)
- Q waves in V1, V2 or V3
- Irregular or non-sinus rhythm (eg heart block associated with maternal SLE)
- Negative QRS in lead aVF at any age (suggest superior axis)
- Positive T wave in lead VI after DOL10
- Prolonged QTc
- Delta waves to suggest WPW

***Normal to see diffuse T wave flattening in first 48hr of life

Hypoglycemia protocol

Definition: Depends on age
- 0-4 HOL: >40
- 4-24 HOL: >45
- 24-48HOL: >50
- >2 DOL: >60

Risk Factors:
- Infant of diabetic mother
- Birth weight <2500g
- SGA (<10th percentile)
- LGA (>90th percentile)
- Preterm (<37 weeks)
- Post-dates (>42 weeks)
- APGARS <7 at 5mins
- Maternal Medications: Terbutaline, beta blocker
- Respiratory Distress >1 hour
- Midline abnormalities
- Family History of Hypoglycemia

Management:
- Mild: Feed, glucose gels x3 if near-term, can also try brief formula instead of MBM because of higher sugar content
- Severe: D10W 2mL/kg bolus and/or maintenance D10W at 60mL/kg/day (titrating to goal glucose). Ways to troubleshoot refractory hypoglycemia include: 1) increasing dextrose-containing IVF flow rate, 2) increasing dextrose concentration (remember any dextrose concentration above 12.5% requires central access), 3) increasing enteral feed fortification or feeding duration (eg extending over 2hr instead of 1hr), and 4)

Intraventricular Hemorrhage

Definition: premature blood vessels are fragile
Screening in :

- GA <32 weeks or BW <1500g
- Low Hct, low platelets
- Unstable BPs, prolonged hypotension
- Cardiopulmonary arrest
- Pneumothorax
- Asphyxia
- Pre/During ECMO

Timing of Screening: DOL 3, 7, 30, 60
IVH Grading

1. Grade 1: Germinal matrix hemorrhage
2. Grade 2: IVH without ventricular dilation
3. Grade 3: IVH with ventricular dilation
4. Grade 4: IVH with ventricular dilation + parenchymal hemorrhage

Therapeutic Hypothermia Protocol

Eligibility Criteria

1. >34 weeks gestation
2. Any 1 of the following:
   a. Apgar score <5 at 10 mins
   b. Needs PPV, intubation or CPR at 10 mins
   c. pH <7.1 from cord or blood gas within 60 mins of birth
   d. Abnormal base excess <-10 mEq/L from cord or blood gas
3. Any 1 of the following:
   a. Neonatal encephalopathy score >4
   b. Seizure or clinical concern for seizure

Management

- Cardiovascular monitoring
- Sedation: morphine loading dose + infusion
- Neuromonitoring: HUS on admission, EEG for 24 hours→ aEEG if without seizures, MRI on DOL 4 after rewarming
- Antibiotics through completion of rewarming
- Fluid restriction to 60 cc/kg/day with Na goal 140-145 and Mag coa
- Labs:
  - On Admission: Lactate, blood gas, CBC, PT/PTT/INR, fibrinogen, blood culture
  - At 12 hours: BMP, Mg, ALT, AST

Exclusion Criteria

- Absolute: <34 weeks gestation
- Relative: severe IUGR, <1750g, metabolic disorders, major IVH, overwhelming sepsis, coagulopathy

Neonatal Encephalopathy Exam Scoring

Stage	Normal	Mild	Moderate	Severe
Level of Consciousness	Normal 0	Hyper-alert/irritable 1	Lethargic/obtunded 2	Super/Coma 3
Spontaneous Activity	Normal 0	Normal 0	Decreased 2	Absent3
Muscle tone	Normal 0	Normal 0	Mild hypotonia 2	Flaccid 3
Primitive Reflexes				
Suck	Normal 0	Weak 1	Uncoordinated 2	Absent 3
Moro	Normal 0	Strong1	Weak 2	Absent 3
Autonomic Function				
Pupils	Normal 0	Mydriasis 1	Miosis 2	Unequal/fixed 3
Heart rate	Normal 0	Tachycardia 1	Bradycardia 2	Variable 3
Respiration	Normal 0	Normal 0	Periodic Breathing 2	Apnea 3

Formula guide:

- Term formula: Carnation Good Start, Enfamil w/ Iron, Similac w/ Iron
  - With DHA and AA: Enfamil lipil, Good Start DHA and Amp, ARA, Similac Advance
- Preterm:
  - <34 weeks or<1800g: Enfamil 24 Premature, Preemie SMA 24, Similac 24 Special Care
  - 34-36 week or >1800g: Enfacare, Similac Neosure
- Food restrictions
  - Soy based (corn based carbs, soy protein): Enfamil Prosobee, Good Start Soy, Similac Isomil
  - Lactose free (corn based carbs, cow-milk protein): Enfamil Lactofree, Similar Sensitive
  - Hypoallergenic (extensively hydrolyzed proteins): Similac Alimentum, Enfamil Nutramigen, Enfamil Pregestimil
  - Nonallergenic (amino acid based): Elecrae, Neocate, Alfamino, Nutramigen AA.
- Antireflux (thickened with rice starch): Enfamil AR, Similac Sensitive RS.
- Toddler: Enfamil Next step, Good Start 2, Similac Go and Grow

Oncology

Management of Emesis

CAN use dexamethasone in solid tumors excluding neuro-onc: ● 6mons or older: ondansetron, dexamethasone, aprepitant ● <6mos: ondansetron and dexamethasone	CANNOT use dexamethasone in heme malignancy, neuro-onc, or on trial that prohibits dexamethasone ● 6mons: ondansetron, aprepitant, and olanzapine (if QTc <500, no h/o extrapyramidal effects w/ dopaminergic agents or other intolerance) ● <6mos: ondansetron

- If breakthrough: add olanzapinef already on olanzapine, consider discontinuing and trying metoclopramide and, if >40kg, scopolamine.
- Anticipatory nausea and vomiting:
  - Ensure receiving appropriate
  - Lorazepam night before and morning of first day of chemo

Tumor Lysis Syndrome (TLS)

Pathogenesis

- Purines are metabolized to hypoxanthine and xanthine, and then to uric acid via xanthine oxidase. Uric acid is poorly soluble in water crystal precipitation and deposition in the renal tubules and AKI.
- Cancer cells have ~4X higher hos than normal cells. Hyperphosphatemia can lead to secondary hypocalcemia and renal calcium phosphate precipitation. Hypocalcemia may also cause cardiac arrhythmias.
- Elevated and phosphate worsen the severity of AKI (increases precipitation of each other)

Clinical Manifestation

- **Hyperuricemia:** ethargy, nausea, vomiting
- **Hyperphosphatemia/hypocalcemia:** norexia, cramping, vomiting, spasm, tetany, seizures, altered consciousness, cardiac arrest
- **Hyperkalemia:** idened QRS; peaked T waves
- **Acute Renal Failure:** 2/2 ric acid and calcium phosphate deposition

Diagnostic Studies

- TLS labs: focus on K, Ca, Phos, BUN/Cr and uric acid. LDH also helpful, but does not necessarily need to be trended, q4-8h initially and space pending stability
- Urinalysis may show many crystals but can be normal due to lack of output from the obstructed nephrons
- Monitor urine output closely

Treatment

- **Hydration:** Goal of 3L/m^2/day or 1.5x mIVF Consider D5W NS or D5W1/2NS, w/o K.
  - Benefits unclear for alkalinization of urine (pH 7-8); can consider if appropriate
- **Hyperuricemia:**
  - **1st line = Allopurinol:** *Prevents further UA formation but does not reduce preexisting UA*. Competitively inhibits xanthine oxidase. Do not use in AKI (xanthine less soluble than UA).
  - **Rasburicase:** *Rapidly reduces existing UA*. Recombinant urate oxidase degrades to allantoin (excreted renally). Consider if rapidly rising, >5, or unable to hyperhydrate.
    - *Test for G6PD first*. Contraindicated in patients with G6PD deficiency produces hydrogen peroxide methemoglobinemia hemolytic anemia.
    - Order "Uric acid, post-rasburicase" (in Order Set) to check levels at 72h post.
- **Hyperkalemia:** Calcium gluconate to reduce risk of dysrhythmia. Insulin plus glucose or beta-agonists for quick control. Kayexalate for excretion.
- **Hyperphosphatemia:** Hyperhydration
- **Hypocalcemia:** IV calcium, high threshold to replete as there is a risk of worsening calcium phosphate deposition if phos still high.

Infectious Prophylaxis Agents

	ALL	**AML**
Antibiotic Agent	**Levofloxacin** - 6mos to 5yrs: 10mg/kg/dose IV/PO q12h - >5yrs: 10mg/kg/dose (max 500mg) IV/PO q24h - For Induction 1A, Consolidation 1B, 1C* - d/c when ANC > 200 x 2d and rising *Refractory/relapsed ALL- use cefepime.	**Cefepime** - 50mg/kg/dose IVq12h (all ages) - For ANC <1000 and falling during each phase - d/c when ANC >100 and rising
Antifungal Agent	**Micafungin** 1.5mg/kg/dose (max 50mg) IV qD For patients on doxorubicin during induction and relapsed patients on anthracycline or high dose cytarabine.	**Posaconazole** (preferred): all phases of tx o Voriconazole is alternative to Posa o Micafungin: <2y, prolonged QTc, hepatic dysfunction, or CYP3A4 interaction
PJP	**Bactrim** (preferred, dosed 3 days/wk 5mg/kg up to 160mg PO), atovaquone, pentamidine ● New ALL: start at induction (except infants who start at diagnosis). ● Relapsed/refractory ALL: during tx and until 6mos after completion	**Bactrim** (preferred, dosed 3 days/wk 5mg/kg up to 160mg PO), atovaquone, pentamidine ● AML: start at diagnosis
Antiviral	**Valacyclovir:** reserved for patients with h/o HSV infection during prior cycles. If prolonged viral symptoms, obtain IgG level, if quite low, consider IVIG therapy	

Fever and Neutropenia- Overview

Definition

1. ANC <500 cells/uL **OR** ANC will be <500 cells/uL during the next 48 hours)
2. Fever > 38.5C once or > 38.0C twice (separated by ≥1 hour) in a 24h period

AML and ALL patients considered "functionally neutropenic"

Clinical Manifestations

- **Fever:** Focal infection (skin/soft tissue/lungs/etc)
- **Physical exam:** vitals, skin folds, line sites, oropharynx, perineum. Inflammation can be subtle.
- *** **NO rectal exam or rectal temperatures** ***

Microbiology

- **Gram-positive predominate:** Coag-negative staph, strep pneumo, staph aureus, strep viridans, B. Cereus
- **Gram-negative also common:** Pseudomonas, stenotrophomonas maltophilia, E. coli, Serratia, Klebsiella

Treatment: Use Onc Sepsis/F&N Order Set

Key Principle:

- Pseudomonas coverage until ANC recovers (even if GP isolated); Vanc for 48hr for empiric Cereus coverage.
- ABx depend on high vs low risk and pt presentation. See below.

"High Risk" Population:

- Hemodynamically unstablesx of septic shock, clinical concern for typhlitis, or neutropenic enterocolitis (e.g. abdominal pain, distention, rectal pain, rectal sores, diarrhea).
- Patient with prolonged and profound neutropenia (ANC <100/mm3 for >7-10 days)
  - AML in all phases (except APML maintenance)
  - ALL in all phases EXCEPT continuation
  - Relapsed ALL and AML
  - Lymphoma: advanced NHL, recurrent NHL, HL
- Patients with Down Syndrome with ANY oncologic diagnosis

"Low Risk" Population

- Age >=12 months and no external central line (Port-A-Cath is OK, broviac is not).
- Not on PN, no bacteremia in 6mo, no recent surgery.
- Solid tumor malignancy (excluding neuro-oncology, solid organ transplant, and HR neuroblastoma)
- Able to be seen at Jimmy Fund Clinic or BCH ED **AND** has good follow up.
- Not allergic to levofloxacin.

Intermediate Risk: not high and not low risk.

Labs: CBCd. LFTs, +/- amylase lipase, +/- RVP.

Cultures:

- Anaerobic and aerobic cultures from each lumen of any indwelling catheters.
- Re-culture after 24h for temperature > 38.5C from one lumen thereafter.
- rinalysis and urine culturekin, sputum, throat swabs and cultures.
- CSF usually not obtained unless clinically warranted (seizure, change in mental status)

Imaging: CXR in patients with respiratory symptoms. KUB with abdominal symptoms.

Antibiotics in High-Risk Patient

Clinical Condition	Empiric Treatment
Hemodynamically stable	Cefepime 50mg/kg/dose q8h **AND** Vanc x48hrs If **cephalosporin allergy:** Aztreonam 30mg/kg/dose q6h (not cefe)
Abd/perirectal pain	ADD metronidazole 7.5mg/kg/dose q6h
Hemodynamically UNSTABLE	Meropenem 20mg/kg/dose q8h **AND** Vancomycin x48hrs
Pts receiving cefepime prophylaxis at time of fever	Meropenem 20mg/kg/dose q8h **AND** Vancomycin x48hrs
Carbapenem allergy or anaphylactic PCN allergy	Aztreonam 30mg/kg/dose q6h **AND** Vancomycin q8h x48hrs **AND** Tobramycin
Fever lasting > 5-7 days	Consider Micafungin 3mg/kg/dose q24h - Obtain serum galactomannan & BD-glucan PRIOR to initiation

Antibiotics in Standard-Risk Patient

Clinical Condition	Empiric Treatment
Hemodynamically stable	Cefepime 50mg/kg/dose q8h If **cephalo allergy:** Aztreonam 30mg/kg/dose q6h **AND** Clindamycin 10mg/kg/dose
Abd/perirectal pain	**ADD** Metronidazole 7.5mg/kg/dose q6h
Hemodynamically UNSTABLE	Use High Risk Algorithm
+ Skin/soft tissue infection/mucositis	**ADD** Vanc
Fever lasting > 72 hrs	Discontinue clindamycin (if receiving) and **ADD** Vancomycin
Fever lasting > 5-7 days	Consider Micafungin 3mg/kg/dose q24h - Obtain serum galactomannan & BD-glucan PRIOR to initiation

Typhlitis (AKA neutropenic enterocolitis): Microbial infection leads to necrosis of layers of bowel wall. Cecum typically but can also involve ascending colon and terminal ileum.

- **Signs/symptoms:** Abdominal pain (often RLQ), distention, cramping, nausea/vomiting, watery/bloody diarrhea, hematochezia. If peritoneal signs and shock, consider bowel wall perforation.
- **Work-up:** Plain film to r/o free air, Contrast CT, blood and stool cultures, and C. diff assay
- **Diagnosis:** CT with contrast

Anterior Mediastinal Mass & Superior Vena Cava Syndrome

Pathogenesis

Compression of mediastinal structures by an anterior mediastinal mass leading to upper body venous congestion and airway obstruction

Differential: "4 T's"

- Thyroid mass
- Thymoma
- Teratoma (malignant)
- (Terrible) lymphoma/ T-ALL

Clinical Manifestations

- Cough/dyspnea/wheezing (40-70% of pts)Arm, neck and/or facial swelling (>60%) from decreased blood returnvenous clot Plethoric/ruddy facies (13-23%)ysphagia, orthopnea, hoarseness.Symptoms exacerbated when lying supine or valsalva.
- Headache, anxiety, altered mental status (to CO2 retentionncreased ICP
- Pleural effusion ~40-60%
- Shock if cardiopulmonary compromise; pericardial effusion possible

Diagnostic Studies

- **Imaging:** CXR, thoracic and abd CT, echo (if suspicion for cardiac compromise) and chest with Doppler (for SVC thrombosis)
- **Labs:** CBC, tumor lysis labs, consider tumor marker evaluation, if peripheral blasts present
- Diagnosis by least invasive method possible to avoid sedation (peripheral lymph node biopsy, bone marrow, pleurocentesis, pericardiocentesis)

Management

- Anesthesia and ORL consult. Consider ICU.
- Immediate supportive care: O2, elevate HOB to 30 degrees
- Empiric chemotherapy may be necessary based on specific circumstances
- Therapy depends on most likely diagnosis, but radiation therapy, steroids, chemotherapy and diuretics are options to consider
- Surgical resection of chemo/radio-resistant tumors (in rare cases)
- Anticoagulation as appropriate if SVC syndrome is due to thrombus

Spinal Cord Compression

Pathogenesis

- Epidural compression from perivertebral tumors extending through intervertebral foramen bulky metastatic disease in vertebral bodies
- Most common etiologies: Sarcoma, neuroblastoma, germ cell tumors, lymphoma and CNS metastases
- Compression of venous plexus leads to cord edema, hemorrhage, and ischemia
- Prognosis is based on duration of symptoms and time to diagnosis and treatment; in general survival for patients with spinal cord compression is <1 year
- May occur at any spinal level (15% cervical spine, 60% thoracic spine, 25% lumbosacral spine)

Clinical Manifestations

- **Focal back pain in a known oncology patient is considered spinal cord compression until proven otherwise!**
- Back pain (80-90% of patients), weakness (35-75%), paresis, sensory abnormalities, paraplegia or quadriplegia, urinary and/or fecal incontinence, or constipation
- Prolonged cord compression causes irreversible paralysis, sensory loss and sphincter incompetence

Physical Exam

- Complete neurologic evaluation including rectal tone, with attention to level of deficit and sensory abnormalities
- Pain is often aggravated by movement, straight-leg raise, neck flexion, recumbency or Valsalva maneuver

Diagnostic Studies

- MRI w/ + w/o gadolinium. Obtain emergently if back pain is associated w/ focal neurologic deficits or refusal/inability to walk
- Following MRI, consider LP with cytology studies
- Spine radiographs are generally not helpful (positive in 1/3rd of cases)

Treatment: Goal is rapid decompression

- Dexamethasone load 1-2 mg/kg, then 0.25– 0.5mg/kg IV q6hr (children) or 10mg IV bolus (adolescents/adults) followed by 6mg q6hr
- Consult Neurosurgery to evaluate for surgical decompression and laminectomy
- Consult Radiation Oncology to evaluate for emergent XRT
- Chemotherapy may be helpful in select tumors if specific tumor type is known or highly suspected and is likely therapy-responsive (e.g. lymphoma, neuroblastoma)
- Surgical resection may be best option if tumor type unknown or if mass persists despite radiotherapy, steroids, and/or chemotherapy

Increased ICP

Definition: Varies w/ age. 5-10 mmHg in infants 10-15 mmHg i. Symptoms when ICP >20 mmHg.

Pathogenesis: Blockage of CSF flow, usually by compression of the third fourth ventricle by an infratentorial tumor

Clinical Manifestations

- **Infants:** Personality changes, head holding or banging, vomiting, lethargy, loss of milestones, seizures, increased head circumference, bulging fontanelle, distension of scalp veins, strabismus
- **Older children:** Headache (classically i morning and occipital), vomiting (often without nausea), diplopia, ataxia, hemiparesis, dizziness, lethargy, speech disturbances, neck stiffness

Physical Exam

- **Vital signs:** Classic Cushing's triad hypertension (systolic widened pulse pressure), irregular respirations bradycardia (late sign of increased ICP)
- **Physical exam:** Complete neurologic exam with attention to mental status and cranial nerves
- **Classic herniation syndromes:**
  - **Transtentorial:** Ipsilateral papillary dilation +/- contralateral hemiparesis
  - **Foramen magnum:** Depressed LOC, Cushing's triad

Diagnostic Studies

- **Labs:** NO LP given risk of herniation!
- **Imaging:** Emergent CT or MRI

Treatment

- Goals are to maintain cerebral perfusion, control ICP and prevent herniation or seizures
- Transfer to ICU; involve Neurosurgery
- Neuroprotective measures. See critical care chapter.

Hyperleukocytosis & Leukostasis

Definition: varies by disease.

- AML: WBC >100,000 (more common)
- ALL: WBC >300,000 (very rare)
- Chronic phase CML: WBC >600,000

Pathogenesis

- Increased blood viscosity (leukemic blasts are less deformable than mature leukocytes) plugs in the microvasculature, decreased tissue perfusion, local hypoxia, cytokines release, and endothelial damage

Clinical Manifestations

- **Neurological:**
  - Visual changes, headache, dizziness, tinnitus, gait instability, confusion, somnolence, occasionally coma
  - Increased risk of intracranial hemorrhage (persists for at least a week after the reduction of white cell count)
- **Pulmonary:**
  - Dyspnea, hypoxia, possible diffuse interstitial or alveolar infiltrates on imaging studies
  - Occasionally, patients develop dyspnea and worsening hypoxemia following the initiation of chemotherapy due to the lysis of leukemic cells trapped in the lungs (eg, acute lysis pneumopathy)
  - Note: Measured arterial pO2 can be falsely decreased in patients with hyperleukocytosis, since the WBCs in the test tube utilize oxygen. Pulse oximetry provides a more accurate assessment of O2 saturation in this setting
- **ID:** ~80% of patients with leukostasis are febrile, which may be due to inflammation or infection
- **Other:** Less common symptoms signs of myocardial ischemia or right ventricular overload, renal insufficiency, priapism, acute limb ischemia, or bowel infarction

Physical Exam: Careful neurologic exam including fundoscopic exam

Diagnostic Studies

- **Labs:** CBCd, tumor lysis labs (see above), coagulation panel
- **Imaging:** CXR and/or non-contrast head CT/MRI for neurologic abnormalities

Treatment

- **Supportive care:**
  - **Hyperhydration**
  - Close monitoring for DIC (especially AML & APML patients)
  - Maintain platelets >20K
  - Judicious use of pRBC
- **Leukapheresis:** mayWBC >100,000. Contraindications include hemodynamic unable to have central access, cardiovascular comorbidities
  - **Low dose-chemo:**enerally "pre-induction" therapy with cytarabine or hydroxyurea
  - May rapidly lower WBC count and cause

Pain Control and Sedation

Initial Opioid Dosage Suggestions (> 6 mo):

****Start low and titrate as needed; see lexicomp for more details*

	IV		PO		PCA#	GTT	Comments
	Initial Dose	Max (>50 kg)	Initial Dose	Max (>50 kg)			
Morphine	0.05 - 0.1 mg/kg q2-4h	5 mg	0.2 - 0.5 mg/kg q3-4h	10 - 20 mg	**Basal**: 0.0 - 0.03 mg/kg/hr **Bolus**: 0.025 mg/kg — (max ~1.8 mg) **Max hr rate:** 1 mg / hr	0.010 - 0.040 mg/kg/hr	
Hydro-morphone	5 - 15 mCg/kg q2-4h	200 - 600 mCg	30 - 80 mCg/kg q4h	2 mg	**Basal**: 1 - 5 mCg / kg / hr max 300 mCg **Bolus**: 2 - 6 mCg / kg max 200 mCg / hr **Max hr rate:** 200 mCg / kg / hr	3 - 5 mCg/kg/hr	
Fentanyl	0.5 -1 mCg/kg q1-2h	50 mCg			**Basal**: 0.1 mCg / kg / hr **Bolus**: 0.25 mCg / kg (max ~18 mCg) **Max hr rate:** 1 mCg / kg/ hr	1 - 3 mCg/kg/hr	**Intra-nasal;** 1-2 mCg / kg / dose (max 100 mCg / dose)
Oxycodone			0.1 - 0.2 mg/kg q4-6h	5 - 10 mg			
Hydro-codone			0.1 - 0.2 mg/kg q4-6h	5 - 10 mg			

#PCA Notation: Basal Rate (Continuous) with a Bolus (Demand), Lock out (minutes) and maximum hourly rate
#To convert PCA to IV/PO, calculate total daily dose and divide into hourly (or PO regimen)

Approximate Opioid Conversion (Equianalgesic dose)

****Approximate equivalent of opioid to 10 mg IV morphine*

Drug	IV	PO	IV to PO Conversion
Morphine	**10 mg**	**30 mg**	**1:3**
Hydromorphone	**2 mg**	**10 mg**	**1:5**
Fentanyl	**100 mCg**		
Oxycodone		**20 mg**	
Hydrocodone		**20 mg**	

Non-opioid Analgesics

	Dose	Max Dosages	Comments
Acetaminophen	**PO/IV:** 15 mg/kg q4-6h	650-1000 mg/dose 60 mg/kg/day or 4000 mg/day	
Ibuprofen	10 mg/kg q6-8h	40 mg/kg/day	
Naproxen	5 mg/kg q12h	15 mg/kg/day	
Ketorolac	0.5 mg/kg q6h	15 mg/dose Max 72 hours	15 mg equivalent to higher doses
Diazepam	0.1 mg/kg q8h	10 mg/dose	Use PRN Spasms
Gabapentin	5-40 mg/kg/day Divided TID		Neuropathic pain Start low and titrate

Procedural Sedation

******For Sedation Requests: Page sedation triage NP On Call, Call NP Booking 5-2652, or Submit Request Online***

****See the Procedural Sedation Medication Card for additional guidance*

****Choose sedation based on need for procedure (+/- pain, length of procedure, etc)*

	Dosing	Kinetics	Adverse Reactions	Additional Considerations
Midazolam *–Anxiolytic* *–Amnestic*	**IN**: 0.5 mg/kg (max 10 mg) **IV:** 0.05 - 0.1 mg/kg (max 2 mg) – push over 10-20 sec – titrate PRN to max 4 mg ***Consider 2-3 doses fentanyl per midazolam	Onset: 1-3 min Duration: 45-60 min (peak 20 min)	– Respiratory depression – Paradoxical reaction – IN discomfort – Infusion pain/burning	– Preoxygenate for 3 min with 12-15 L via non-rebreather or facemask to increase time prior to desaturation in case of apnea – Use CO2 monitor to detect apneas earlier than O2 Sat **– ETT Sizing (Uncuffed)** ET size = (age + 16) / 4 *Down size by ½ for cuffed ET *Have ½ size smaller available **– Laryngoscope::** — <2 yrs: Miller 1 — 2-12 yrs: Miller 2 / Mac 2 — >12 yrs: Mac 3 **– Laryngospasm mgmt:** — Expectant (wait 30 sec) — laryngospasm notch (jaw thrust with pressure toward midline) — PPV — Deepen sedation (propofol or midazolam) — Succinylcholine (0.1 mg/kg IV, then 1 mg/kg IV)
Fentanyl *–Analgesic*	**IN:** 2 mCg (max 100 mCg/dose) **IV:** 1-2 mCg/kg (max 50 mCg/dose) – Titrate 1 mCg/kg q3min PRN – push over 10-20 sec – Max 5-7 mCg/kg	**Onset:** – IN: 2-3 min – IV: 1-3 min **Duration:** – 30 min (peak 15 min)	– Respiratory depression	
Ketamine *–Dissociative analgesic* *–Anxiolytic* *–Sedative* *–Amnestic*	**IV:** 1-2 mg/kg (max 100 mg/dose) – 50 mg often need – may repeat 0.5 mg/kg q5-10 min PRN (max 50 mg/dose) **IM:** 4-5 mg/kg – repeat 2-4 mg/kg after 10-20 min PRN **IN:** 0.5 mg/kg (repeat PRN pain)	**IV:** Onset 0.5-1 min Duration 5-15 min **IM:** Onset 3-5 min Duration 15-30 min **IN:** Onset 1-2 min Duration 30-60 min	– Laryngospasm – Hallucinations – Agitation upon waking up; Emesis – Myoclonus/flushing ***Avoid with URIs, poorly controlled asthma, increased ICP w/ obstruction	
Propofol *–Sedative* *–Hypnotic*	**Induction:** 0-4 yrs; 2-3 mg/kg 5-10 yrs; 1.5-2 mg/kg >10 yrs; 1 mg/kg — may need additional 0.5-1 mg/kg bolus q30-90 sec for induction **Maintenance**: 100 - 250 mCg/kg/min	Onset: <1min Duration: 5-15 min	– Hypotension (expected) – Apnea – Bradycardia – Infusion pain (try midaz/lidocaine prior to infusion)	
Ketofol *–Sedative* *–Hypnotic* *–Analgesic*	Ketamine Induction: 0.5 mg/kg Then Bolus Propofol for Induction: 0-4 yrs: 2 mg/kg 5-10 yrs: 1.5 mg/kg >10 yrs: 1 mg/kg **Propofol IV Maintenance:** 100-250 mcg/kg/min **Propofol**: 0.5 - 1 mg/kg bolus PRN pain **Ketamine**: 0.5 mg/kg bolus PRN pain or if longer procedure	Onset: <1 min Duration: 5-15 min	– Hypotension – Laryngospasm – Apnea	

Primary Care

BMC Contacts/Resources

	Resources	Contact	
Patient Navigator	Urgent housing needs Urgent food insecurities; Newborn visits follow ups	**Non-urgent:** Message 'Amb Primary Care Pedi Navigation' **SOFAR PN:** Kristin Reed, p6571 **Heme PN:** Miriam Guerrier p1228 **Housing PN:** Yesenia Valentin p0961 (page first or message if non-urgent) **LTSS PN:** Nicole De Olmos, p9394 (Message Kristin Reed to refer patients) Use ***.LTSSREFERRAL*** in Epic Message to Nicole & your team navigator	
Integrated Behavioral Health	One-time diagnostic or medication recommendations	"**Integrated Behavioral Health**" referral in Epic IBH Psychiatrists: Andrea Spencer p5333 and Amy Yule p4976	
Social Work	Short term therapy/referrals IPV, trauma, CPT, etc	Liz Renzella p3433	
Asthma Educator	Asthma education	Leah Shafer, p7024 (or Epic Message)	
Breastfeeding Clinic	Lactation Support	Epic referral "Referral to Breastfeeding Clinic" Refer to phone#: 617-414-MILK (6455)	
Food Insecurity	Food	BMC Food Pantry Referral, Project Bread Referral	
Tax Assistance	Tax Filing and Advice	Ambulatory referral to StreetCred)	**BMC Clinic Resources**
Prior Authorizations	Insurance Issues	Staff Message to "BMC Amb Pharmacy Liaison"	
Early Intervention	Developmental evaluation or intervention	Order referral in Epic Message patient navigator or self refer	

BCH Contacts/Resources

	Resources	Contact
ED Expect Line	To refer to ED	52170
Lactation	Lactation Nurse (M-F)	56455 (5-MILK)
Patient Navigator	Transportation, Housing, Utilities, Food insecurity	Non-urgent; message CHPCC Social Needs Pool; **.pcpsocialneedsref*** Urgent: page p5931
CHPCC SW	Immediate needs: – imminent homelessness – Crisis Intervention, IPV, CPT – Behavioral Health	Page p0171
Referrals	Specialty Clinic Referrals	**Neurology, Dermatology, Allergy, Endocrinology, ASK** – Send/Save message to Specific Consult Pool; – Add order to message with specific details of consult **Other specialities:** Place order for Primary Care Patient Referral
Early Intervention	Development Evaluation or Support	Message CHPCC Early Intervention Pool; use "**.pcpeiref"**
Behavioral Health	+PSC or other +Mental Health screener	Non-urgent: Message CHPCC Behavioral Health Pool; **".pcpbhhref"** Emergent needs; page Social Work

Well Child Visit Checklist:

	Screening	Vaccines	Developmental Milestones*	Guidance
Newborn	Edinburgh Bilirubin	HepB (hospital)	G: Lifts head briefly F: Hands in fists V: Cries	Safe Sleep, Car Seat Fevers, Formula Mixing Postpartum Depression
1 mo	Edinburgh	None	G: Lifts head briefly F: Hands in fists, opens occasionally V: Beginning to coo, starting to smile	Safe Sleep, Fevers Formula Mixing, Tummy time Reading
2 mo	Edinburgh	Vaxelis (DTaP, HiB, IPV, HepB) PCV13, Rota	G: Lifts head while prone F: Opens hands, moves both arms/legs V: Social smile, attends to face	Safe Sleep, Car Seat, Formula Mixing, Tummy time, Reading
4 mo	Edinburgh	Pentacel (DTaP, HiB, IPV), PCV13, Rota	G: +head control, Pushes up when prone F: Grasps objects, brings hands to mouth V: Chuckles, babbling, Looks to caregiver	Safe Sleep, Car Seat Solid Food, food allergy Tummy time, Reading
6 mo	PEDS	Vaxelis (DTaP, HiB, IPV, HepB) PCV13, Rota, Flu	G: Rolls tummy to back, sits w/ tripod support F: Bangs toys V: Laughs, smiles at reflection, reciprocal sounds	Solid Food, food allergy, bottles in bed, baby proofing Poison Control #
9 mo	PEDS	Flu	G: Sits w/o support, pulls up, crawls F: feeds self, moves toys from hand to hand V: Dada/Mama non-specific, peek-a-boo, stranger danger, responds to name	Baby Proofing, sippy cups, poison control
12 mo	PEDS Hb, Lead 1st dental visit	MMR, VZV HepA, Flu	G: pulls up to stand, cruising, starting to walk F: Pincer grip, points V: waves bye-bye, Dada/Mama specific, object permanence	Dental, baby proofing Cups, limit juice Lower mattress, screen time
15 mo	PEDS	DtaP, Hib, PCV13, Flu	G: Squats to pick up, starting to walk/walking F: Starting to draw V: 1-2 words, follows direction with gestures, points for things, imitates other children	Limit setting, no bottles Dental, screen time
18 mo	MCHAT PEDS	HepA #2	G: Walks up steps, carries toy while walk F: Scribbles, drinking from cup/spoon V: 1-step directions, 3+ words, Helps dress/undress	Limit setting Toilet training Screen time
24 mo	Hb, Lead MCHAT PEDS	Flu	G: Jumps, kicks ball, walks up stairs F: Stacks, draws line V: 2 word phrases, 50% intelligible, 2+ body parts	Limit setting, tantrums, toilet training, dental, Screen time
3 yo	Hb, Lead BP, Hearing, Vision PEDS	MMR, VZV, Flu	G: Tricycle, good runner F: Draws circle, can cut, starting to dress self V: 3 word phrases, 75% intelligible, Imaginative play, asks who/what/where, states first-name	Limit setting, tantrums, screen time, car seat, dental
4 yo	Hb, Lead BP, Hearing, Vision PEDS	MMR, VZV (if not already given) Kinrix (DTap + IPV), Flu	G: Skips, climbs stairs alternating feet F: Draws cross, mature pencil grip, brushes teeth V: cooperative play, 100% intelligible, tells stories	Limit setting, screen time Safety, school readiness
5-12 yo	BP, BMI PSC-17	9 yo: HPV #1 and 2, Flu		Sleep hygiene, outdoor safety, screen time, Sexuality/Gender Identity
13-18 yo	BP, BMI PHQ, GAD2 Lipids if @ risk GC/CT if @risk	11 yo: Tdap, MCV 16 yo: MCV, flu		Sleep hygiene, Mood/SI Firearm, seatbelts Drugs and alcohol safety Sexuality/Gender Identity

**G: Gross Motor Milestones; F: Fine Motor Milestones; V: Verbal or Social-emotional Milestones*

Common Primary Care Screening Tests and Follow up

	Positive Test	Follow Up	BCH Pathways*
MCHAT-R	Answered No (except 2, 5, 12)	Score 3-7: MCHAT follow up Q's: If ≥2, EI and DBP referral Score ≥ 8: Early Intervention, Developmental Evaluation	
PSC-17	Total Score ≥ 15 I ≥ 5, A ≥ 7, E ≥ 7	ADHD (A), Mood disorder evaluation (I), Conduct disorder evaluation (E)	
Elevated BP	**Elevated**: >90th%ile **Stage 1**: >95th%ile **Stage 2:** >95%ile + 12 mm/hg	Follow up visit, sooner if higher BP Diet/lifestyle counseling Eval for secondary HTN	
Elevated BMI	>95%ile Or >85%ile + risk factors (FHx, dyslipidemia, kidney disease, etc)	For >9yo: – Total Cholesterol/HDL or fasting lipids – TSH, ALT, HbA1c or fasting/random glucose For >12yo: above tests and; – Consider PCOS, SCFE, OSA, Binge Eating screening as indicated	
Lead	Any Detectable Result	– counsel on lead inspection – assess for anemia, initiate Fe as indicated – repeat Pb as indicated (see pathway) – for levels >25 page environmental health doc – consider BCH pediatric environmental health center outpatient referral	
Anemia	Hb < 11 g/dL Fe deficient on iron studies	Ferrous Sulfate 15 mg/ml elemental iron (2 mg elemental Fe/kg/dose BID x 1-3 mo) Repeat in CBC in 1-3 mo	
Concussion	Recent Head Injury with Consistent Symptoms: – Headaches, dizziness, irritability, fatigue, sleep issues Eval for Red Flag Symptoms (weakness, LOC, focal findings)	Track PCSS (Post-concussion symptoms scale) Targeted symptom management (cognitive rest, meds etc) Create Physical and Cognitive Rest Plan – Initial 24-48 hrs rest before gradual return to school/sports Sports Clearance Letter (must do one time MA training) – Use the "**.pcpconcussionletter**" dot-phrase	

**For BMC clinics, refer to the BMC resources QR Code above, otherwise, refer to specific clinic or AAP guidelines*

**Access the BCH Pathways must be on VPN or on the Children's Network,*

CDC Pediatric Vaccine Schedule

CDC Catch-Up Vaccine Schedule

CDC Travel Vaccine Guidance

Pulmonology

Pneumothorax (PTX)

Causes	● Primary : idiopathic, possibly due to apical blebs ● Secondary: traumatic, iatrogenic, due to infection, CF, asthma, underlying lung disease, connective tissue disease
Work-up	● CXR; Bedside US ● Consider testing if suspicion for underlying dx or recurrence, including: Sweat test, NBS, Chest CT, genetics consult
Tx	● **Small PTX (<30% lung volume + HDS)** ○ Conservative Management including: serial CXR, optimize tx of underlying disease if any ○ If new O2 requirement or failure to re-expand within 24-48hrs, consider chest tube/aspiration ● **Large PTX (>30% lung volume, or significant resp distress)** ○ Chest Tube +/- needle aspiration ○ If failure to re-expand within 3-5 days, consider surgical intervention ● **Recurrent PTX or failure to spontaneously regress:** Pleurodesis via VATS (video-assisted thoracoscopic surgery)
Tension PTX	Sx: respiratory distress, **decreased/absent breath sounds, hypotension,** tracheal deviation, distended neck veins Dx: Physical exam + vitals usually diagnostic, consider beside US Tx: Needle decompression at 2nd intercostal space at midclavicular line -> chest tube/pigtail catheter placement

Cystic Fibrosis Exacerbation Management *(Refer to **BCH Cystic Fibrosis Guidelines** for additional guidance)*

System	Problem	Intervention
Infection (Antibiotics)	Common Bacteria: H. Flu S. aureus P. aeruginosa Personal Microbiology	Choose antibiotics based on prior exacerbations and susceptibilities or current cultures ● ≥1 abx targeting isolated bacteria from respiratory culture ○ If MRSA, consider vancomycin vs. linezolid vs ceftaroline ● 2 abx with pseudomonas coverage (zosyn + fluoroquinolone/tobramycin/amikacin) ○ Only if culture + pseudomonas or history of pseudomonas on prior cultures **Duration:** variable based on clinical response, PFTs, often 10-14-day course
Pulm	Secretion burden and respiratory infections (above)	Supplemental O2 as needed for sat goal >92% Optimizing airway clearance via chemical and physical ways to dislodge and clear secretions Airway clearance regimen: Albuterol MDI → Hypertonic saline (HTS) → +/- DNase → Chest PT → +/- inhaled antibiotics→ +/- corticosteroids ● **Increase frequency** beyond home regimen (typically TID) ● If patients do not tolerate HTS due to cough/throat irritation, lower % or consider NS nebs Continue CFTR modulators including: Kalydeco, Orkambi and Trikafta
GI	Constipation Pancreatic insufficiency Nutritional deficiencies Hepatobiliary disease	Continue or intensify home bowel regimen Continue pancreatic enzyme replacement therapy Continue fat-soluble vitamins (ADEKs), Optimize nutritional status **If abdominal pain: consider KUB to r/o DIOS
Endocrine	Diabetes	Ensure glucose control

Hemoptysis:

Ask:	Frank blood or streaking? How much? Can patient localize bleed to one side? Does patient have a history of hemoptysis? If so, chronic, small volume, or larger volume? Has the patient required embolization? When?
Assess	Vital signs (tachycardia, hypotension) and exam (oropharyngeal source, pulmonary source, crackles)
Send	CBC, coags, T&S
Stop	All chest PT, HTS, or pulmozyme Avoid BiPAP unless patient uses routinely
Give:	Consider Vit K: 10mg PO or 5mg SQ x3d Discuss w/ fellow inhaled TXA or DDAVP
Call:	Fellow, Senior, consider IR (if large volume >200 or repeated events)
ICU:	Discuss transfer if patient unstable, hemoptysis volume is massive, or unable to stop bleeding

Pediatric Hypoxia

Cause	Findings	Diagnoses
Hypoventilation	Normal A-a gradient High PaCO2 Responsive to O2	CNS depression, CNS lesions affecting respiratory center, muscular weakness, poor chest wall elasticity, genetic disorders
V/Q Mismatch	Increased A-a gradient Responsive to O2	Obstructive lung disease, pHTN, ILD, pulmonary vascular disease, PNA, PE
Shunting	Increased A-a gradient Not responsive to O2	Anatomic shunts: intracardiac shunting, pulmonary AVMs, hepatopulmonary syndrome Physiologic shunts: ARDS, pulmonary edema
Diffusion Limitations	Hypoxemia often during exercise	ILD, pulmonary fibrosis
Reduced O2	Responsive to O2	High altitude

Asthma - REFER TO **BCH ASTHMA CLINICAL PATHWAYS** FOR FURTHER GUIDANCE

Outpatient Asthma Management: Initial assessment requires severity classification

Classification	Intermittent	Mild	Moderate	Severe	Outpatient Asthma Treatment Guidelines
Symptoms	<2 days per wk	>2 days/wk	Daily	Daily	
Nighttime awakenings	0-4 yr: 0 >5 yr: <2 per mo	0-4 yr: 1-2/ mo >5 yr: 3-4/ mo	0-4 yr: 3-4/mo >5 yr: >1/week	0-4 yr: >1/week >5 yr: >7/week	
Activity Limitation	None	Minor	Some	Extreme	
SABA use	<2 days/week	<2 per mo	Daily	Daily	
FEV1% predicted)	>80%	6%	40-60%	<40%	

Common Inhaled Corticosteroids:

Inhaler (Brand)	Type	Low dose	Medium Dose	High Dose
Fluticasone (Flovent)	ICS	<12: 44 mcg/puff 2 puffs BID >12: 220 mcg	<12: 110 mcg/puff 1-2 puffs BID >12: 220-440 mcg	<12: 220 mcg/puff 1 puff BID >12: >440 mcg
Beclomethasone (Qvar)	ICS	40-80 mcg BID 80-160 mcg	80-160 mcg BID 160-320 mcg	240-320 mcg BID >320-640 mcg
Budesonide (Pulmicort)	ICS	160/4.5 mcg or 2 puffs BID	180/4.5 mcg 2 puffs BID	160/4.5 mcg 2 puffs BID
Budesonide-formoterol (Symbicort)	ICS-LABA	80 mcg/4.5 mcg 2 puffs BID	160 mcg/4.5 mcg 2 puffs BID	
Fluticasone-salmeterol (Advair HFA)	ICS-LABA	45 mcg/21 mcg 2 puffs BID	115 mcg/21 mcg 2 puffs BID	230 mcg-21 mcg 2 puffs BID

Asthma Inpatient Management

HASS Scoring

Dyspnea	Full Sentences Phrases/Short cries Single Words/Grunts	1 2 3
Resp Rate (per min)	*2-5y: <30 6-12y: <25 >12: <20* *2-5y: <30-40 6-12y: 25-30 >12: 20-25* *2-5y: >40 6-12y: >30 >12: >25*	*1* *2* *3*
WOB	*None - 1 muscle groups* *2 muscle groups* *3 muscle groups*	*1* *2* *3*
Breath Sounds	*Clear or end expiratory wheeze* *Expiratory wheeze* *Inspiratory/expiratory wheeze or diminished*	*1* *2* *3*
O2 Sat	*>94%* *90-94%* *<90%*	*1* *2* *3*

HASS Management

<6 (Mild)	Space Albuterol to goal of q4h
7-9 (Moderate)	Continue Albuterol
>10 (Severe)	Consider repeat UNINEB Consider CXR, VBG, IV Mag (if not given) Consider ICU eval if needs > q1h or unstable

Acute Exacerbation Medications

Albuterol	**MDI:**	**Nebulizer**
	<10 kg: 4 puffs q2-3h	0.25 ml q2-3h
	10-30 kg: 6 puffs q2-3h	0.5 ml q2-3h
	>30 kg: 8 puffs q2-3h	1 ml q2-3h
Steroids	**Dexamethasone**: 0.6 mg/kg QD x2 doses (max 16mg) **Prednisone (PO)**: 1 mg/kg q12h x 5d (max 60mg/d) **Methylprednisone (IV):** 2mg/kg (max 80 mg) then 0.5-1 mg/kg q6h (max 60 mg)	
MgSO4 +bolus	**40 mg/kg/dose** (max 2000mg), may repeat in 4 hrs Give with 20cc/kg bolus	

ED/Urgent Care

Initial HASS	Initial Treatment	Treatment after reassessment
HASS <7	Albuterol MDI or Neb	d/c if HASS<7, otherwise, escalate care
HASS 7-9	Systemic steroids within 1 hr UNINEB OR Albuterol MDI x3 +/- ipratropium MDI	Consider IV MgSO4 if HASS the same or worse Admit if Q2H albuterol required
HASS >10	Systemic steroids within 1 hr. UNINEB OR Albuterol MDI x3 +/- ipratropium MDI. Consider IM epi if poor air entry	Consider continuous albuterol, BiPAP, Heliox Admit to ICU/ICP if continuous/BiPAP required

Floor Care:

- Continue Systemic Steroids (usually dexamethasone)
- Continue albuterol treatment, space as tolerated for HASS <7
- Consider NPO+IV fluids if unstable resp status
- Titrate O2 to >92% awake, >90% asleep
- ICU eval if more than q2h albuterol required

ICP/ICU:

- Continue Systemic Steroids (IV methylprednisone)
- Consider NPO+IV fluids if unstable resp status
- If on continuous albuterol, max time = 16 hrs before trial off
- Wean as tolerated for HASS <7
- If HASS >12 while on continuous, consider Heliox/BiPAP

Toxicology

Resources

- Tox pager number: 6612
- Poison control number: 1-800-222-1222
- Ex-trip: https://www.extrip-workgroup.org/ (guidance on use of extracorporeal tx in poisonings)
- Material Safety Data Sheets (MSDS): ingredients in household object ingestions

One Pill Can Kill (most common, not extensive list):

Agent	Minimal Potential Fatal Dose	Potential Fatal Dose	Toxicities
β-Blockers	Unclear	1-2 tablets	Bradycardia, hypotension, seizures, hypoglycemia
Ca Channel Blockers	<40 mg/kg	1-2 tablets	Bradycardia, hypotension
Clonidine	Unclear	1 tablet	Bradycardia, CNS depression
Hypoglycemics (i.e. glyburide)	~1 mg/kg	2 tablets	Hypoglycemia
Methyl Salicylate	~200mg/kg	½ tsp of oil of wintergreen 2 tsp Icy Hot Balm	Seizures, cardiovascular collapse
Opioids	Variable by potency	1-2 tablets	CNS and respiratory depression
TCAs	~20mg/kg	1-2 tablets	Seizures, arrhythmia, hypotension

Stabilization: ABCDDDEF: **A**irway, **B**reathing, **C**irculation, **D**isability, **D**rugs/**D**-sticks, **D**econtamination, **E**KG, **F**ever/temperature
Labs: EKG, Serum tox, UTox (expanded in some cases), VBG, Chem, LFTs, D-stick, Upreg
Toxicology Screens: Differ from hospital to hospital (substances included, limits of detection, etc); false + and - common on utox
Always ask: Can I decontaminate? Can I enhance elimination? Is there an antidote? How do I provide targeted supportive care?

Common Treatments

Treatment	Indications	Dosage
Activated Charcoal*	Within 1-2 hours of ingestion of adsorbable* chemicals, alert patient with intact airway. If large ingestion, delayed release, anticholinergic, opioid, or decreased intestinal motility properties, consider charcoal outside of initial window	0.5-1 g/kg (max 50g)
Naloxone	Suspected opioid ingestion: AMS, respiratory depression, miosis Can consider for clonidine poisoning	Full dose: 0.1 mg/kg q2min (max 2 mg) • Titrate to respiratory effect • Can use smaller doses for milder symptoms or chronic opiate use
Sodium Bicarbonate	TCA ingestion Other QRS prolonging medications with QRS > 100ms Salicylate toxicity	Bolus: 1-2 mEq/kg Infusion: 150 mEq in 1L D5W @1.5-2x M Can repeat as needed for QRS

* Contraindications to Activated Charcoal: Unprotected airway, altered mental status, emesis, intestinal obstruction, risk of GI perforation/bleed, need for endoscopy or poorly adsorbed materials: heavy metals (arsenic, mercury, lead, iron), corrosives, inorganic ions (Li, Na, Ca, K), corrosives (acid, alkali), hydrocarbons, alcohols, essential oils

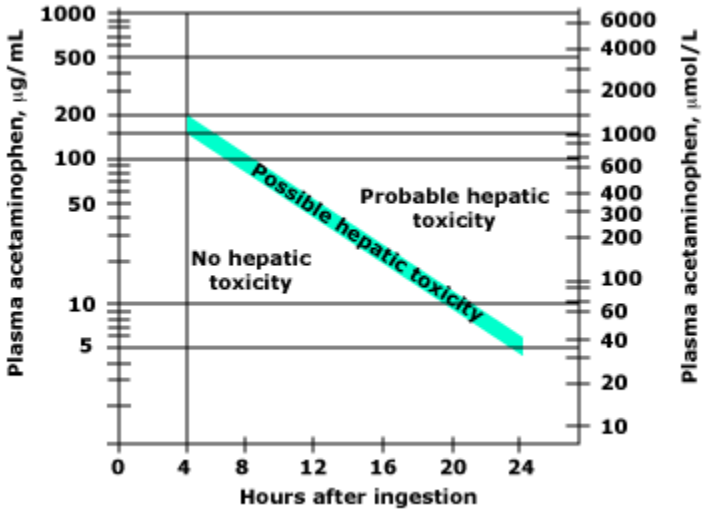
QRS prolonging agents (>100ms)		QTc prolonging agents (>450ms)
• TCAs • Bupivacaine • Diphenhydramine • Cocaine • Carbamazepine/oxcarbamazepine • Class 1a antiarrhythmics (procainamide/quinidine/disopyramide)	• Class 1c antiarrhythmics (flecainide/propafenone) • Propranolol • Loperamide • Lamotrigine • Bupropion	**Antiarrhythmics:** amiodarone, sotalol, procainamide **Antibiotics:** levofloxacin, ciprofloxacin, erythromycin, clarithromycin, ketoconazole, itraconazole **Antidepressants:** amitriptyline, SSRI (Citalopram most common), SNRIs (eg: venlafaxine) **Antipsychotics:** haloperidol, quetiapine, ziprasidone **Others**: sumatriptan, methadone

Acetaminophen toxicity:

Acetaminophen			
Toxic Dose: 200 mg/kg (7.5-10g in older/adult patients)			
Stage	**Signs/Symptoms**	**Labs**	**Time Frame**
1	Nausea, Vomiting, Malaise, Pallor	Normal AST/ALT, INR	0-24 hrs
2	Hepatotoxicity RUQ tenderness	Rising AST/ALT INR nl/elevated	12-72 hrs
3	Fulminant hepatic failure Encephalopathy Coma	AST/ALT > 10000 INR elevated Elevated Cr, acidosis, lactemia, hypoglycemia	72-96 hrs
4	Recovery	Normalization of AST/ALT, INR	> 96 hrs

Antidote: N-Acetyl Cysteine**:**

- Use plasma Acetaminophen level (at least 4hrs post ingestion) and nomogram (adjacent) to determine whether to initiate NAC
- Nomogram only useful IF single acute acetaminophen ingestion with reliable time course and rapid presentation <24 hrs (Poly-ingestion can alter elimination, absorption etc, and nomogram not as reliable and serial measurements indicated)
- Timing of NAC administration:
  - Goal: within 8 hrs of ingestion
  - Start ASAP if >8 hrs from ingestion
- Dosage:
  - loading dose of 150mg/kg over 1 hour
  - then 50 mg/kg over 4 hours
  - then 100 mg/kg over 16 hours



Toxidrome Diagnosis PE Algorithm:
(After rapid stabilization and supportive care)

- **Miosis (**pinpoint**)**
  - **Dry**
    - **Opioid**: Fentanyl, morphine, codeine, methadone
    - **Sedative**: benzodiazepines, barbiturates, ethanol
  - **Diaphoretic=** cholinergic= pesticides, nerve gas, alzheimer's medications, Nicotine
- **Mydriasis (dilated)**
  - **Dry=** anticholinergic: antihistamines, antimuscarinic, antispasmodic, TCA
  - **Diaphoretic**
    - **Normal muscle tone**
      - **Sympathomimetic**: amphetamine, cocaine, cathinone
      - **Sedative withdrawal**: benzodiazepines, barbiturates, ethanol
    - **Increased muscle tone**
      - **Decreased reflexes**: neuroleptic Malignant syndrome. Antipsychotics, antiemetics.
      - **Increased reflexes:** serotonin syndrome. MAOI, SSRI, TCA