Geisinger
Janet Weis Children's Hospital

Janet Weis Children's Hospital Geisinger Medical Center
PICU Reference Guide

2nd Edition- April 2025
Editor- Robin Craig, DO

General PICU Objectives: *Appreciate and begin to recognize basic pathophysiology of critically ill children. Understand the basic technical skills involved in resuscitation and care of a critically ill child.*

CORE COMPETANCIES:

- Patient Care: compassion, professionalism, empathy
- Medical knowledge: (display through presentations) clinical knowledge and physical exam skills.
- Practice-based learning and improvement: use of literature and learning from each patient experience to display a graded improvement in clinical acumen and understanding of the critically ill patient.
- Interpersonal and communication skills: effective hand-offs, working with a team and family to provide comprehensive patient care.

Timing of overnight sign out and AM rounds:

6:00-7:00 am: Resident sign out.
8:00-8:10 am: IDT rounds- charge RN, service attending, second service attending, care manager/SW, PT/OT/Speech
8:10-8:30 am: Attending sign out occurs
8:30 am: PICU rounds will commence at 8:30 am.
5:30pm: Evening resident sign out

Rounds generally begin with CVTS patients however severity of illness may require that a non-CVTS patient be rounded on first.
The presenting resident, PA or NP, should examine the patient, review radiographs and the electronic flow sheet prior to rounds. All data should be readily available when rounds begin.

THIS COPY BELONGS TO: _____

Inspiration from University at Buffalo- Oishei Children's Hospital. Sections used and adapted from Dr. Amanda Hassinger, MD; Oishei Children's Hospital, Buffalo NY with her expressed permission.

PICU DAILY SCHEDULE

Where PICU rounds occur:
Rounds occur in the PICU at the bedside. Careful consideration of the child is essential. It is inappropriate to have rounds create undo anxiety for the child. The team should commence rounds close enough to the bedside to allow the RN and family to have an active role while being at a distance that is comfortable for the child.

Who participates in PICU rounds:
A consistent approach to rounds ensures that *every* critically ill child receives the detailed attention they deserve. Rounds allow *all* members of the team to participate in the assessment and formulation of care plans for each child. Lastly, rounds provide an invaluable forum for ongoing education of the health care team.

Participants should include:

- Bedside nurse
- Parent(s), guardians
- PICU NP or PA
- Residents
- PICU attending, and subspecialty attending when necessary (CVTS, Trauma)
- Charge RN
- Pharmacist
- Respiratory therapist
- Nutrition

Who presents on PICU rounds:
PICU residents are primarily responsible for presentations on their individual patients.
Exceptions:

- PICU PA/NP may present patients in the absence of covering residents or when PICU is at high volume/acuity. This should be decided on by the residents and APP prior to beginning rounds.

How PICU rounds are performed:
The importance of morning rounds dictates that a ***formal*** and ***consistent*** format be utilized.

Ground rules:

- Respect – All opinions should be heard and respected.
- Listen – Often, it is more important to listen and think than to speak. The resident should present the data with his or her assessment and plans prior to attending input. Interruptions should be limited to only those times when inaccurate data is being presented.
- Nurses are vital to the formulation of care plans. As such, their assessment is crucial. After the resident presentation, the bedside RN should augment any resident data and be allowed to express his or her concerns regarding the child.
- Education– Attending input and finalization of decisions are imperative. This is done after the formal resident presentation.

Data Presentation
<u>New admissions: Order set: Pediatric ICU Admission (4108)</u>
Children who are newly admitted should be presented as a complete history and physical exam. This includes past medical history, past surgical history, previous PICU admissions, medications, allergies, and social history.
Once the history and physical has been presented, an **overall assessment** should be given including the working diagnosis and differential diagnosis.

- *This is a 15-day-old male who presents with sudden onset apnea, fever and nasal congestion progressing to respiratory failure. Other diagnoses to consider include meningitis, sepsis, intracranial event…*

After the overall assessment has been given, each organ system should be addressed in a manner as outlined below. All new admissions require careful attention to the initial diagnostic workup (pending labs, future diagnostic studies).

Established patients:

- **Brief identifying statement:**
  - *This is PICU day 6 for 4-month-old Jack who was admitted for with seizures and found to have pneumococcal meningitis.*
  - *This is POD # 1 for Michael who is an 8-month-old s/p patch closure of a moderate sized membranous VSD.*
- **Overnight significant events*:***
  - Only major events should be listed (i.e., intubation/extubation, new procedures/complications). Minor changes should be discussed within the organ system (i.e., electrolyte replacement, advancement of feeds, wean of ventilator, wean of cardiovascular agent).
  - *Jack had episodes of agitation requiring increased sedation and also had an episode of desaturation secondary to mucous plugging of the ETT..*
  - *Michael did well overnight with stable hemodynamics, good gas exchange and well controlled pain**.***
- **Vital signs and Examination**
  - Most attendings prefer the entire examination and vitals to be given after the major O/N events are listed. ***Alternatively**, examination findings can be placed in the corresponding system assessment*.

  Include positives and only pertinent negatives

  Pulmonary: breathing rate, work of breathing, air entry, lung sounds, trach site
  Cardiac: heart rate, heart sounds, murmur, blood pressure, distal perfusion (temperature, color, capillary refill, edema)
  Abdomen: distention, bowel sounds, girth, tenderness, GT site
  Neuro: Comfort level, level of consciousness, motor exam (GCS if trauma), cranial nerve exam, focality, withdrawal s/sx
  MSK: skin, ortho, C-spine
  Include invasive devices in place
- **Organ system approach**
  - **System based objective data**
  - **Current support (therapeutics)**
  - **Plan by systems**

  Using a systematic approach, the child's organ specific data should be incorporated into the appropriate system. Important data include patient monitoring data (i.e., ventilator feedback, waveform analysis, CVP, ICP, $EtCO_2$), laboratory results, operative findings and imaging results.

System Based Approach

Specific data to be presented and analyzed followed by a specific plan for each system

Respiratory

Objective data:

- Ventilator settings, mode, PIP, PEEP, spontaneous Vt (cc/kg)
- Blood gas data, PaO_2/FiO_2 ratio, OI
- CXR
- Quality of secretions, cough
- Chest tube output, air leak
- Spontaneous breathing trial results

Current support:

- Oxygen, HFNC, CPAP, BiPAP, CPT, Nebulized/MDI treatments

Cardiovascular

Objective data:

- Rhythm/Ectopy
- Invasive hemodynamic data (CVP, arterial pressure variation, LAP)
- Recent echocardiography and or catheterization data
- Lactate, cvO_2, Pro-BNP, troponin

Current support:

- Cardiovascular medication support (inotropes, pressors)
- Antiarrhythmics
- Diuretic therapy
- Pacer settings

Fluids/Electrolytes/Nutrition (often **GI** is also incorporated here unless GI issues are complex)

Objective data:

- I/Os. Specify type and amount of fluid.
- cc/kg/day (and % of maintenance)
- Weight!
- Kcal/kg/day (especially in infants), TPN breakdown if necessary
- Electrolytes, acid base
- PO or enteral access site (gastric vs. postpyloric)
- GI labs – LFTS, amylase, lipase, gastric pH, heme
- Stool quality, heme

Current support:

- NPO, IVF, TPN or enteral
- Gut prophylaxis, bowel regimen, promotility agents

Neurologic/Pain Control/Mobility

Objective data:

- Trend in level of consciousness, GCS
- If ICP or EVD – neurophysiologic data ICP, CPP
- Pain and anxiety status
- Imaging CT, MRI
- EEG
- AED levels

Current support:

- Sedative and analgesia regimen- SBS goal
- EVD drainage

- AED
- Mobility goal (PICUup level)
- PT/OT, Speech, neuropsychology

Hematological

Objective data:

- Hgb/Hct
- Coagulation labs
- TEG

Current support:

- Blood product requirements
- Pro/anticoagulant therapy

Renal (if no active issues may have been covered in FEN)

Objective data:

- BUN/create
- Acid/Base
- Urine glucose, ketones, specific gravity

Current support:

- Diuretics
- Renal replacement therapy (i.e., HD, PD)

Infectious Disease / Inflammation

Objective data:

- Fever curve
- WBC and other inflammatory indicators (i.e., CRP, ESR, PCT)
- Culture results
- Abx levels

Current support:

- Antibiotics /antivirals/antifungals - Day x/y
- Antipyretics, anti-inflammatory

Social

- Family updated, and family input considered
- Social service input/ Child life input
- Discharge or ward transfer planning

Other systems as needed include: Musculoskeletal, Transplant, Endocrine, Rheum, Derm

Check **ALL M**eds **– mnemonic to avoid iatrogenic complications**

Check **– Close loops**

Airway - Is the airway secure? Is there a risk of self extubation?

Lines and devices (central lines, ICP, PICC lines, Foley, Drains, Pacer etc)

- Have they been accounted for during rounds?
- Where are they and for how long?
- Can any line or device be removed?
- Are the pacer thresholds current? Is the battery fresh?

Lab schedule

- Have the appropriate labs been ordered?
- Can the lab draws be reduced?

Medications

- Have all medications been accounted for and verified by pharmacy?

TEAM DEBRIEF



Announce: “Team, let’s have a 5-min debrief”



Emotional Decompression: “How is everyone feeling?”



Summarize: “So we can all be on the same page, let’s summarize the events”



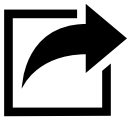
Praise: “What did we as a team do well?”



Opportunities: “What were the challenges?”



Team Inclusion & Take-Away: “Did every profession have a chance to speak?” & “What is something that you will take away from this experience”?



Forward: “I will pass this summary to the Quality/Safety & Improvement champions for the unit”

GROUND RULES:

Discuss: Reactions, processes, team communications, and identified system issues (i.e., equipment, resources, etc.)	**Avoid Blame Game**: Don’t blame individuals or teams
Validate: restate the points brought forth & team emotions	**Avoid Critiques:** Don’t critique individuals or teams
Safe Forum: Allow each profession to speak up	**Don’t focus on “Solutions”:** No need to solve challenges today

Appreciate: “Everyone: Thank you for your time and input!”

PULMONARY

Respiration: the process of gas exchange at the alveolar-capillary interface to diffuse oxygen into (oxygenate) and CO_2 out of (ventilate) the blood.

Oxygenation at the alveolar level is directly dependent on:

- The concentration of oxygen in the alveoli (alveolar oxygen or P_AO_2), determined by F_iO_2 and P_aCO_2:

  $P_AO_2=(P_{Bar}-P_{H2O})xF_iO_2 – (P_aCO_2/RQ)$

  Barometric P at sea level = 760 torr, P_{H2O} = 47 torr

  Normal P_AO_2 in RA = (760-47) x 0.21 - (40/0.8) = 100 torr

- The number of alveoli with oxygen receiving adequate blood flow (V/Q).
  - Manipulated by increasing the number of alveoli open using PEEP or mean airway pressure or recruitment with open-lung strategies.
  - Perfusion to the lungs can be impaired by a pulmonary embolus, an AVM, pneumonia, congenital heart disease, pulmonary hypertension.
    - Using inhaled pulmonary vasodilators like nitric oxide can increase perfusion to the alveoli being ventilated.

VQ mismatch and its effect on PCO2. PaO2

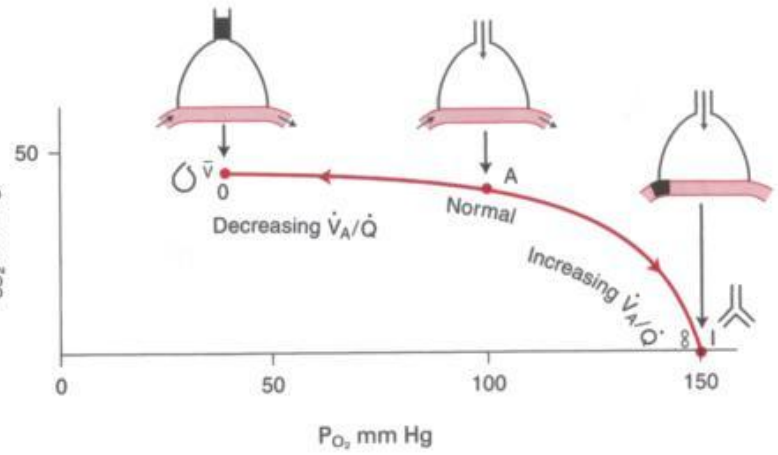


Figure 5-7. West JB. " Respiratory Physiology: the Essentials." Eigth Edition, 2008.

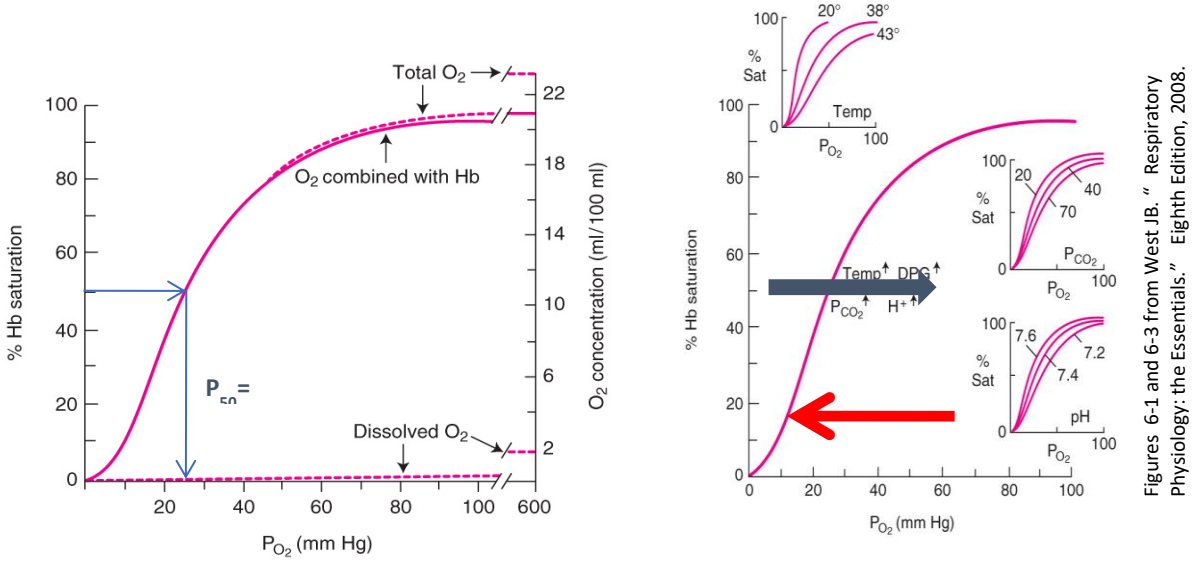
- The surface area of gas-exchange membrane allowing oxygen to diffuse into the blood.
  - Impaired in ARDS or ALI where there is inflammation, scarring or fluid impairing the ability of the oxygen to diffuse across the alveolar membrane.
  - Measure with the *A-a gradient* = the difference between the oxygen in the alveoli P_AO_2 and that in the artery P_aO_2. (Normal A-a gradient is 10-20 on room air, higher indicates disease.)
- The mixed venous oxygen content.
  - Assuming a fixed F_iO_2, pulmonary blood flow and alveolar gas exchange, if the blood that enters the pulmonary vasculature is desaturated from poor CO, shock, anemia, etc., the arterial blood will be relatively desaturated as it leaves the lungs.
- Oxygen delivery to the tissues is dependent on the oxygen content of the blood (C_aO_2) which relies on the amount bound to hemoglobin more than the O_2 diffused in the blood:

  C_aO_2 = (Hgb x 1.34 x S_aO_2) + **(P_aO_2 x 0.003)***.

*This is why in severe anemia a patient should be on a 100% non-rebreather mask to exploit the **second-half** of this equation until you can increase the Hgb.

These factors all add up to the **5** main reasons for **arterial desaturation**:

1. **Alveolar hypoventilation** (high PCO_2 lowers P_AO_2)
2. **Pulmonary venous desaturation** (impaired gas exchange as in ARDS)
3. **Right to left shunt**, intra-cardiac or intra-pulmonary (eg. VSD, pulmonary AVM)
4. **V/Q mismatch** (eg. atelectasis, foreign body)
5. **Decreased affinity of Hgb for oxygen** (CO poisoning)



Figures 6-1 and 6-3 from West JB. " Respiratory Physiology: the Essentials." Eighth Edition, 2008.

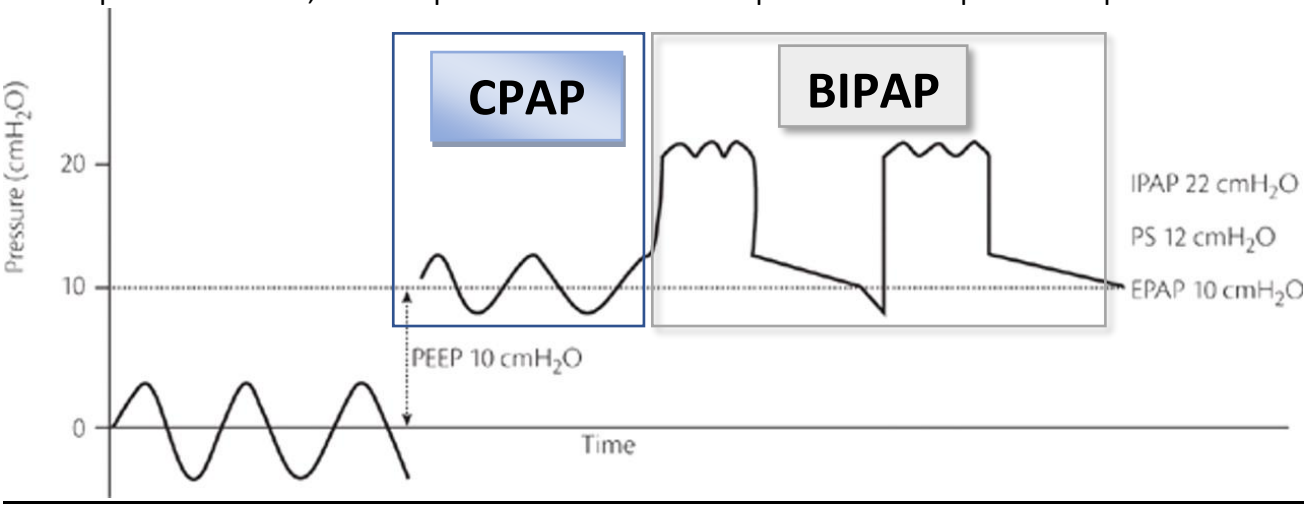
- **Oxygen-hemoglobin dissociation curve**: demonstrates the P_aO_2 at a given Hgb saturation.
  - Fetal hemoglobin has a P_{50} of 20mmHg demonstrating higher affinity of Hgb for oxygen = easier to pick up and harder to release.
  - Shift of the curve to the **right** in hyperthermia, acidosis and hypercarbia makes the affinity for oxygen ***lower*** meaning it's **easier to release** at the tissue level.
  - Hypothermia, alkalosis and low 2,3-DPG shift the curve to the **left** making Hgb have a higher affinity for oxygen = **harder to release to the tissues.**
- **Ventilation:** removal of CO_2 out of the blood into the exhaled gas relies on:
  - **Minute ventilation** as determined by the respiratory rate and tidal volume per breath minus the anatomic and physiologic dead space (dead space=part of the respiratory tree without gas exchange, includes conducting airways and alveoli not receiving perfusion).
  - **The gas-exchange membrane**: only profound V/Q mismatch will impair ventilation.

NON-INVASIVE RESPIRATORY SUPPORT:

- **Low Flow Nasal Cannula:** 1-6L/m. Non humidified oxygen.
- **Venturi Mask:** similar to a non-rebreather mask but with adjustable amounts of FiO2.
- **High- Flow Nasal Cannula:** humidified air to make higher flow volumes more comfortable to the upper airway.
  - Initiated at 1.5- 2 liter per min/kg and titrated to patient effort and oxygenation

Figures 6-1 and 6-3 from West JB. " Respiratory Physiology: the Essentials." Eighth Edition, 2008.

- **CPAP (continuous positive airway pressure):** positive pressure delivered through a tight fitting mask either over the nose or nose and mouth. The patient can breathe above the CPAP at the rate they are comfortable.
  - CPAP should be above physiologic (>/=5cm H_2O)
- **BiPAP (biphasic positive airway pressure):** same mask as CPAP but will deliver a CPAP and a Pressure support.
  - Will set an IPAP (inspiratory positive airway pressure= PS and CPAP) and an EPAP (expiratory positive airway pressure/ CPAP).
  - BiPAP will deliver the IPAP each time the patient triggers a breath, at the rate the provider has set, or if the patient has a set "back-up rate" and the patient is apneic.



RESPIRATORY FAILURE- INTUBATION

Order set- Pediatric ICU Intubation / Mechanical Ventilation (4171)

Indications**:**

- *Inability to protect the airway*: CNS injury (GCS <8), seizures, neuromuscular dysfunction affecting swallowing or vocal cord movement (Guillain-Barre), overdose or encephalopathy
- *Inability to meet the metabolic demands of respiration:* shock, arrest, extremis
- *Inability to overcome severe pulmonary disease*: hypoxia or hypoventilation, secretions, poor compliance from parenchymal dysfunction or impaired gas-exchange
- *Airway compromise*: infection, allergic reaction, trauma, foreign body, or ingestion causing narrowing or collapse of the airway
- *Sedation or procedural airway protection*

Intubation Methods:

- ***"Awake"*** *– no paralysis utilized*
  - Indications: arrest, cervical spine injury, airway abnormalities, hemodynamic instability
  - Use local anesthetic sprays: nebulized lidocaine 2mL 1%
- ***RSI (rapid sequence intubation)***
  - Indications: aspiration risk, e.g. trauma, recent meal, high abdominal pressure

- Technique:
    - Pre-oxygenate with 100% oxygen using either a LFNC or HFNC, **NO BAGGING**.
    - Have suction ready.
    - Sedative + analgesia + paralytic (+/- lidocaine or atropine) given all at once.
    - Hold cricoid pressure ONCE MEDS GIVEN UNTIL ETT IN PLACE.
    - Intubate once medications take effect.

- **Technique:**
    - Pre-oxygenate and bag-mask the patient until ready to intubate- (can keep a LFNC or HFNC in place if able to maintain a seal for apneic oxygenation).
    - Have all equipment ready and IVF available to bolus if concerned about BP.
    - ***Pre-medicate 2-3 minutes*** *before* giving sedation.
    - ***Sedate and allow 1-2 minutes*** to take effect.
    - **Paralyze and *wait 30-60 seconds*** to take effect. Start bagging immediately after administration even if not totally paralyzed yet.
    - Insert laryngoscope and visualize white vocal cords anterior to esophagus. IF YOU CANNOT SEE CLEARLY, DO NOT ATTEMPT TO INTUBATE, REPOSITION OR PULL OUT AND BAG UNTIL S_pO_2 is 100% and it is safe to try again.
    - Hold corner of lip to open mouth, insert ETT through the vocal cords past the cuff.
    - Remove the stylet and immediately begin bagging through ETT. Watch chest rise and S_pO_2. Have someone auscultate over all lung fields and stomach.
    - Place CO_2 detector, aka capnograph, or in-line end tidal and look for color change. **Yellow=yes!**
    - If placement confirmed, take off capnograph and bag until CXR confirms position of the tip of the ETT. Desired position around T2-T3 but at least 1 cm above the carina.
- ***The difficult airway! Fiber-optic scopes, LMAs, emergency tracheostomy.***
    - **ANTICIPATION**: Check the medical record for anesthesia reports or previous intubation attempts and ask the family if it has been a problem for this patient before.
        - Red flags: obese, redundant neck folds, difficult to bag mask, ANY FACIAL TRAUMA, Down syndrome (C1/C2 instability), h/o SGS or OSA, micrognathia, anterior mediastinal mask
    - Call anesthesia to assist you if you think it'll be a problem. They have a fiberoptic scope.
    - LMA's are easy to insert and can buy time if anesthesia is not around.
        - LMA sizing is based on weight: **< 5kg=1, 5-10kg=1.5, 10-20kg=2, 20-30kg=2.5, 30-50kg=3, >50kg=4.**

INTUBATION EQUIPMENT:

- Bag and appropriately sized mask with inflated cushion
- Suction: "Yankauer" is the large rigid catheter, ensure is hooked up to working suction
- 100% oxygen

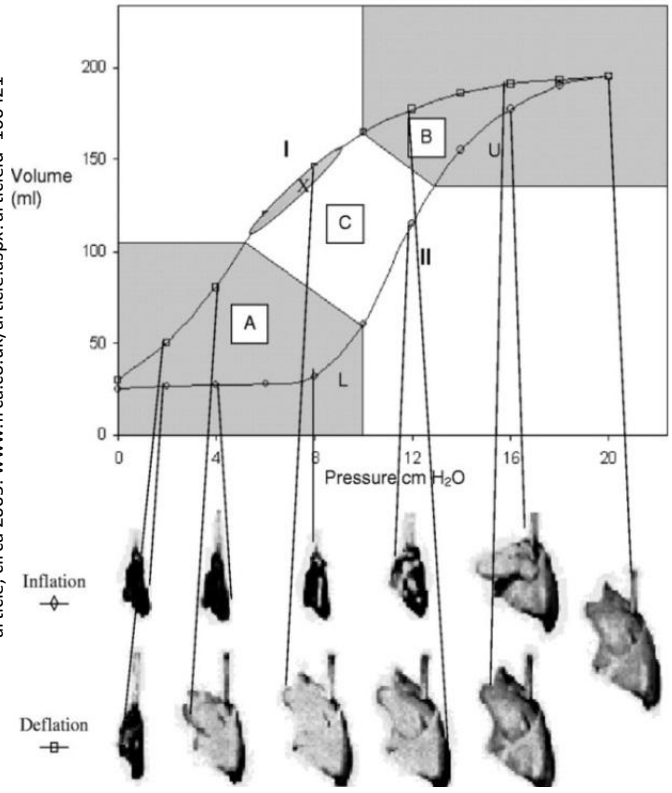
- Oral airway (measure from tragus to lips to ensure correct size)
- Laryngoscope: sizes and types as shown below, have a miller and a mac, CHECK BULB
- Stylet in the ETT (lubricated) and bent into the hockey stick shape you prefer
- ETT: cuffed, ensure one size below anticipated size, make sure cuff works
- Capnograph or end tidal CO_2 detector
- Emergency equipment if concerned about airway: LMA, Anesthesia, etc.

Age (regardless of wt)	Laryngoscope	ETT Size = (Age/4) + 4
Term newborn	Miller 0-1	3.0 cuffed
6 months – 1 year	Miller 1	3.5 cuffed
1 – 2 years	Miller 1, Mac 1	4.0 cuffed
2 – 4 years	Miller 2, Mac 2	4.0 cuffed
4 – 6 years	Miller 2, Mac 2	4.5 cuffed
6 – 8 years	Miller 2, Mac 2	5.0 cuffed
8 – 12 years	Miller 2-3, Mac 2-3	6.0 cuffed
12 years and up	Miller 3, Mac 3	7.0 cuffed

Macintosh blade=curved edge slides just behind epiglottis and holds it up, *Miller*=straight blade

Conventional Mechanical Ventilation

AKA invasive Positive Pressure Ventilation (IPPV)= forced inhalation with passive exhalation



Graph adapted from AnesthesiaUK, Pressure Control Ventilation article, circa 2005. www.frca.co.uk/article.aspx?articleid=100421

Figure taken from website article:
http://mechandz.blogspot.com/2009/12/ventilation-mechanical.html

Aim to ventilate along Zone II as shown here, at the highest compliance along the natural lung hysteresis curve. Adequate PEEP will avoid Zone A and avoiding high PIP will avoid Zone B.

Poor compliance = curve shifted down = smaller Zone C & larger Zone B.

Follow these steps to determine settings:

1.When the vent breathes for the patient: "SIMV"

<u>Synchronized intermittent mechanical ventilation:</u> set a mandatory rate and the patient can breathe above it with PS, if taking a breath near time for a mandatory breath, the machine gives the mandatory breath with full PC or VC in synch with the patient's effort.

2. How the ventilator delivers the breath: With a set **pressure** or set **volume**?

a. <u>Pressure control (PC)</u>: delivers a set pressure above the PEEP over the entire I_t. Creates a deceleration flow

pattern (see below) just like blowing up a balloon. As you reach the full expansion of the balloon and the lungs, less volume goes in at the same pressure since neither can continue to expand as it approaches maximal volume.

b. Volume control (VC): delivers a set volume over the I_t by using a set flow. Pressure increases over the breath since the same flow will need more pressure to expand the lung as it reaches maximum capacity. This is less physiologic and not often used as a result. Usually hits higher PIP than PC or PRVC.
c. Pressure-regulated volume control (PRVC)/ APV on Hamilton: actually, a pressure control mode with a smarter ventilator. Set a goal V_t and the ventilator tests the compliance of the lung to determine what pressure is needed to deliver that tidal volume over the set I_t. This mode can adjust to dynamic compliance changes.
d. Spontaneous Mode: Set the PEEP ("CPAP") above which the patient breathes with PS to overcome ETT resistance. This is a weaning to extubation mode. No mandatory RR is set.

Compliance: will determine what tidal volumes you achieve at what pressures.

- Definition: distensibility of the lung, ease of expansion of the lungs and thorax.
  - Determined by pulmonary volume and elasticity, can be impacted by abdominal pressures (hepatomegaly, ascites) and chest wall rigidity.
  - Compliance will be *high* in healthy, easily distensible lungs and *low* in ARDS, PNA, or asthma.
  - Normal= 1ml/kg/cmH_2O.

▪ $C = \Delta V/\Delta P$

▪ At the bedside: $C = (V_{te}/kg)/(PIP-PEEP)$

Ventilator Parameters:

Vent Parameter	Definition	Starting Setting
Tidal Volume (V_t)	Volume given with each mandatory breath	6-8ml/kg; if very stiff lungs (poor compliance) aim lower = 4-6ml/kg
Pressure control (PC)	Inspiratory pressure over PEEP, this is not PIP (PIP=PC+PEEP)	Set for a goal tidal volume of 6-8ml/kg
Pressure support (PS)	Support given by the vent for each spontaneous breath	Usually 10cmH_2O for OETT, lower for tracheostomy
Positive end-expiratory pressure (PEEP)	Pressure left in the circuit at the end of each breath, used to maintain FRC	5cmH_2O for normal lungs, higher in atelectasis.
Respiratory Rate	# of mandatory breaths/min	Age appropriate

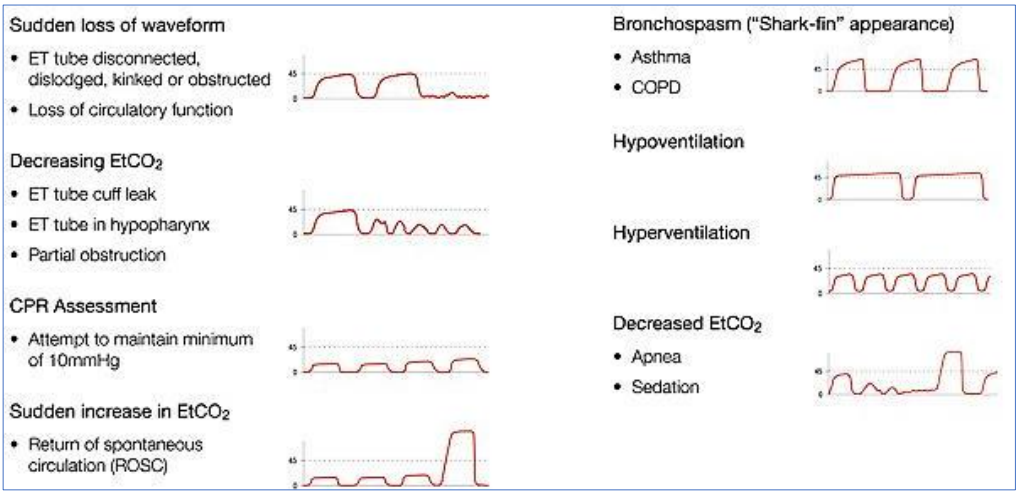
Inspiratory time (I_t)	Amount of time over which the vent will deliver the set V_t or PC ****Remember I_t determines E_t.**	Newborn to 1yo: 0.50 – 0.70 s >1 yo: 0.60 – 1 second. **E_t= (60/RR) - I_t****
F_iO_2	Fraction of inspired air that is O_2	Titrate as soon as possible to <60%
Mean Airway Pressure	*Not Set → measured by ventilator*	Physiologic MAP 8-16cmH_2O
Peak inspiratory pressure (PIP)	PEEP + PC, *not set → observed*	Goal < 30 cmH_2O to avoid barotrauma

Other important considerations:

- Compliance factor? The amount of pressure or volume lost to the tubing. This is important in PRVC/APV as the V_t you set is not what the alveoli see.
- Leak? The ETT is too small for the airway and a leak occurs around the tube into the mouth and atmosphere. This can be heard audibly or auscultated. Pressure mode preferred with a leak- as need decelerating flow pattern to deliver flow as quickly as you can. *****CLUE: large difference between V_{ti} and V_{te}.**

VENTILATOR MODE CHEAT SHEET	Mode	What you set	Clinical scenarios for use	Advantages	Disadvantages
	PRVC/APV	V_t, PEEP, RR, PS, I_t, F_iO_2	Bronchiolitis, asthmatics, pneumonia, sepsis-related respiratory failure	Adjusts for dynamic compliance, precise control over ventilation	Small patients (<5kg) the V_t may be imprecise, not good with large leak around ETT
	PC/PS	PC, PEEP, RR, PS, I_t,F_iO_2	Neonates with resp failure, airway protection, procedural	Minimize damaging PIP, better in normal or fixed compliance	Problematic in dynamic compliance, less precise control over ventilation
	VC/PS	V_t, PEEP, RR, PS, I_t, F_iO_2	Older patients intubated for airway protection with normal compliance	Precise control over ventilation	No pressure limit, higher PIPs than PRVC in same V_t, cannot use with leaks,

End Tidal CO_2 Monitoring



High Frequency Oscillatory Ventilation

- **Definition:** A gentler way to oxygenate and ventilate a patient with hypoxia and poor compliance requiring PIPs >30 on CMV. Deliver rapid "breaths" with tidal volumes of 0.5-3mL, smaller than the anatomic dead space.

- HFOV holds a mean airway pressure (used to recruit and maintain lung volumes) and has small oscillations around the MAP
- Hertz determines the frequency of the oscillations. The higher the Hertz = more oscillations, ↓area under the curve = ↓ventilation.
- Amplitude (ΔP) determines height of oscillation. Adjust to achieve "wiggle" to upper thighs.
- F_iO_2 is targeted to maintain S_pO_2. ***If able to wean F_iO_2<60%, wean MAP.***

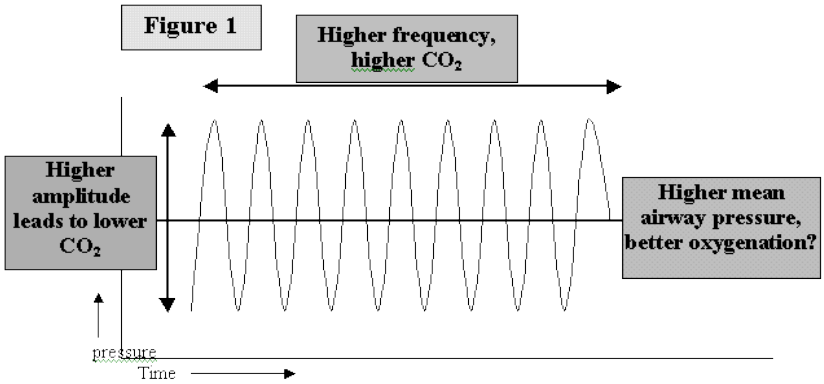


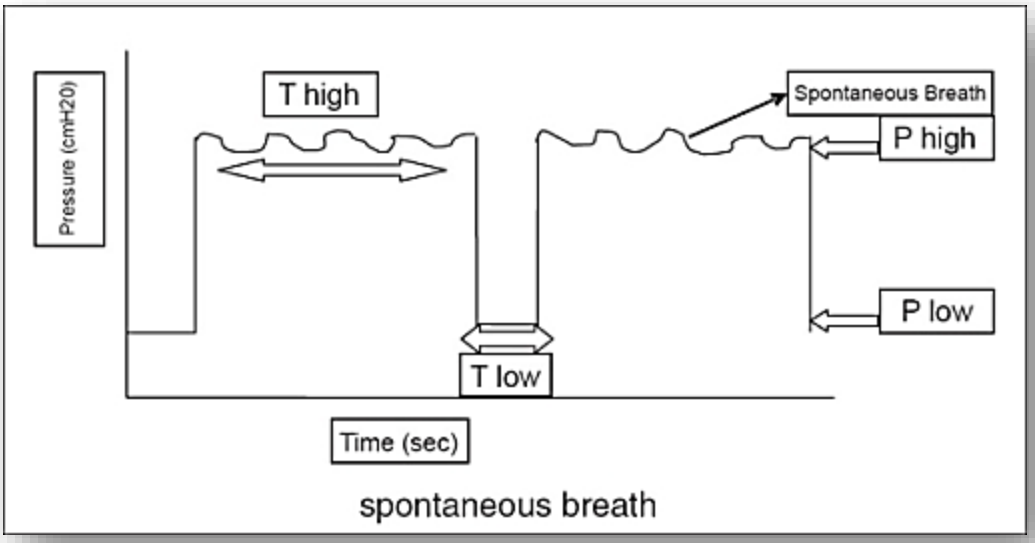
Figure from: http://www.sswahs.nsw.gov.au/rpa/neonatal/html/newprot/hfov.html

Parameter	Initial Settings	Effects of manipulation
Mean airway pressure (MAP)	**10%** or **5 cmH_2O** over MAP on conventional vent ; Optimal MAP ≤**8** ribs expansion on CXR	Increase MAP = increase P_aO_2 Wean by 1cmH_2O, takes >4hr to see effect of change.
Frequency, Hertz (Hz)	10-15 in newborns, 6-8 children	**RR=60 x Hz**; Lower Hz = lower PCO_2
Amplitude (ΔP)	Adjust to wiggle in thighs, usu 40-80	Increasing ΔP = lowering PCO_2
F_iO_2	Start at 100% and titrate to S_pO_2	Lower F_iO_2 = lower P_aO_2; Once F_iO_2 < 60%, wean MAP.

- **Weaning on HFOV:** Titrate FiO2 and MAP. Once able to wean MAP to <18-22 consider switching to conventional ventilator as should be able to obtain that MAP without high Peak Pressures.

Airway Pressure Release Ventilation (APRV)

- Baseline airway pressure is the upper CPAP level, and the pressure is intermittently "released" to a lower level, thus eliminating waste gas
  - Not designed for use in paralyzed patients.
  - Ventilation is achieved with spontaneous breathing and occasional releases.



Stawicki SP, et al. HFOV and APRV: A Practical Guide. *J Intensive Care Med* 2009;24(4):215-229.

Parameter	Initial Settings	Effects of manipulation
P_{high}	Add 2-4cmH_2O to MAP conventional vent	↑P_{high} = ↑P_aO_2
P_{low}	0 cmH_2O	↑P_{low} = ↓ΔP = ↑PCO_2
T_{high}	Adjust to "RR" as needed for vent; usually 2-5 seconds	↑ T_{high}= fewer cycles = ↑PCO_2
T_{low}	Titrate to avoid total volume loss; usually < 0.5 second	↑T_{low} = ↑E_t =↓PCO_2 as long as pt doesn't lose PEEP
Parameters you DON'T SET on APRV:		
RR	# of respiratory cycles/min = 60/(T_{high}+T_{low})	Pt should breathe around this
PEEP	This is the pressure left at the end of the time spent at Plow	The ventilator will read the intrinsic PEEP in the upper right corner.

  - T_{low}= time the pressure is released to P_{low}.
    - T_{low} is determined by the patient's lung compliance.
    - Want to set the T_{low} so that the patient *does not completely exhale* and lose FRC or RV.
  - **The "RR" then is **60/(T_{high} + T_{low}).** Manipulate T_{high} to change the number of respiratory cycles.**

- **Weaning on APRV:**
  - ⬆ T_{high}: Lengthen the amount of time on P_{high} making the patient breathe spontaneously more often over P_{high} to maintain ventilation.
  - ⬇ P_{high} : by 1-2cmH_2O as oxygenation improves.

Extubation

- **Criteria** for extubation vary on the clinical scenario, but generally should meet the following:

- **CALMSS pneumonic**
  - C- CNS: are they awake enough to protect their airway
  - A- Airway: do they have a leak, are they a difficult airway
  - L- lung disease: improved- atelectasis present?
  - M- Medication: any meds on board that may affect respiratory drive?
  - S- Secretions: are they manageable?
  - S- Studies: do they have any upcoming studies or surgeries that require an airway?

- **Equipment for extubation**:
  - Appropriately sized mask for BMV, re-intubation equipment at bedside if high-risk, or nearby if not high-risk
  - Suction (Yankaeur and in-line)
  - Racemic epinephrine neb ready if suspicious of post-extubation swelling and stridor
  - Support intended to use, eg HFNC, CPAP/BiPAP, NC, etc.
  - Adhesive remover, towel for ETT and gastric tube if also coming out
  - Nasal or oral airway if suspect UAW obstruction

Pediatric ARDS

Age	Exclude patients with peri-natal lung disease			
Timing	Within 7 days of known clinical insult			
Origin of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload			
Chest imaging	Chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease			
Oxygenation	**Non-invasive mechanical ventilation**		**Invasive mechanical ventilation**	
	Mild-moderate	Severe	Mild- Moderate	Severe
	PaO2/FiO2 >100 Or SpO2/FiO2 >150	PaO2/FiO2 </= 100 Or SpO2/FiO2 </= 150	OI <16 Or OSI <12	OI ≥ 16 OSI ≥ 12
OI = (F_iO_2 x Mean airway pressure x 100)/P_aO_2			OSI = (F_iO_2 x MAP x 100)/SpO_2	
PALICC-2; *PCCM* 2023				

- **Causes:** Primary pulmonary (PNA, pneumonitis), vs. non-pulmonary (multi-organ failure, sepsis, pancreatitis, TRALI, hepato-pulmonary syndrome).
- **Pathophysiology:** Infiltration of the lung parenchyma and migration of neutrophils and inflammatory mediators ito the alveolar space causing a relative surfactant deficiency and impaired gas exchange.
- **Treatment:** ARDS is characterized by poor lung compliance and issues ventilating and oxygenating.
  - "Open-lung strategy" = low tidal volumes (5-7ml/kg), high PEEP (10-15 cmH2O) to maintain FRC in a setting of poor compliance, permissive hypercapnia (P_aCO_2 50-60, arterial pH > 7.2); APRV, HFOV or ECMO V-V may be necessary.

- Conservative fluid management (**AVOID fluid overload**)
- Early enteral nutrition (within 72 hours)

Status Asthmaticus

Order set: Pediatric ICU Asthma and Status Asthmaticus (4136)

- **Diagnosis**: Evidence of bronchospasm with poor oxygenation refractory to conventional therapy. Manifests as prolonged exhalation, poor aeration, accessory muscle use, dyspnea.
- **Concerning Historical and PE findings**:
  - Previous ICU stays or intubations
  - Tachypnea or bradypnea with dyspnea (no more than a few words per breath)
  - Pulsus paradoxus: inability to fully exhale leads to air trapping which increases the intra-thoracic pressure to the point that during inspiration, the venous return to the heart is obstructed and cardiac output decreases. Defined as a SBP >20mmHg lower during inspiration.
  - LACK OF WHEEZING with poor aeration can be a very bad sign!
  - Crepitus in neck or chest can be seen in severe obstruction leading to air leak
- **Initial imaging:**
  - CXR: look for expansion, flattening of the diaphragms, asymmetry (foreign body), pneumothorax, and cardiomegaly.
- Look for the other causes of wheezing or obstructed breathing in a pediatric patient:
  - Viral infections like bronchiolitis or pneumonia
    - Bacterial or fungal pneumonia
    - Laryngo-, tracho- or bronchomalacia **(BRONCHODILATORS MAKE THESE WORSE)**
    - Foreign bodies anywhere in the respiratory tree
    - Anaphylaxis
    - Congenital heart defects, vascular slings.
- **Treatment:**
  - *Standard therapy*: steroids and beta-agonists (inhaled or systemic)
  - *Adjunctive therapy*: Atrovent (inhaled anticholinergic), trial x 24 hours
  - *Alternative therapy*: Magnesium, Heliox, ketamine, IM/IV epinephrine, inhaled anesthetics.
  - *CPT*: secretion clearance is important (NOTE: 3% NS nebs can worsen bronchospasm)- can cause wheeze to worsen in some asthmatics- monitor closely
  - NIPPV may be necessary and can stave off intubation (Trial CPAP 1st, then BiPAP)
  - GI prophylaxis is warranted if NPO for a prolonged period while on steroids.

MEDICATION	MECHANISM/CLASS	DOSE	SE, notes
Albuterol	Aerosolized β_2-agonist = relaxation of bronchial smooth muscles	2.5mg neb, can give up to 40mg/hr	Tachycardia, jitteriness, hypokalemia, diastolic hypotension, ST elevations
Atrovent	Anticholinergic, inhibits cGMP = bronchodilation	0.5mg neb Q6hr if responsive	Best in 1st 24-48hrs, stop after if no response
Corticosteroids (Solumedrol IV or Prednisolone PO)	Systemic anti-inflammation	2mg/kg load, then 0.5-1mg/kg Q6hr (Max dose 80mg/d)	Hyperglycemia, hypertension, myopathy, gastritis, agitation
Magnesium sulfate	Causes smooth muscle relaxation through unknown mechanism	25-50mg/kg/dose IV, can schedule Q6hr	Hypotension, not all patients are "Mg responders"
Heliox	Mixture of helium and oxygen=laminar flow around airway resistance	80:20, 70:30, 60:40 depending on FiO_2 requirement	Immediate response if will have effect, cannot use if on >50% FiO2
Terbutaline	Systemic beta$_2$-agonist	Load 10mcg/kg, infusion 0.1-1mcg/kg/min, max 10	Same as albuterol, watch for ST depression and chest pain, hyperglycemia
Ketamine	Dissociative anesthetic that causes inadvertent bronchodilation	0.5-1mg/kg, infusion 0.5-1 mg/kg/hr titrate Q30m	Secretions, hallucinations, hypertension, myocardial depression
Theophylline	Methylated xanthine derivative, ↑cAMP and ↓inflammation	6-7mg/kg, infusion of 0.4-1.3mg/kg/h, titrate to levels	Nausea, tachycardia, anxiety, seizures, HA *Use ONLY if home med*
Isoflurane	Inhaled anesthetic agent which causes airway relaxation	0.4-3%	Hypotension requiring IVF and vasopressors is expected

- **Mechanical ventilation in asthma:**
  - Intubation of an asthmatic is VERY dangerous because it can precipitate an arrest, cause a pneumothorax and *will* worsen bronchospasm by irritating the airway. Prior to intubation, have IVF and albuterol ready to be given down the ETT.
  - Ventilation is challenging. Permissive hypercapnia may be necessary.
  - Try to match a patient's I/E ratio to allow them to fully exhale by using low RR and short I_t ****Remember: E_t (sec)= (60 / RR)- I_t**

- Titrate V_t to keep ***plateau*** pressure <30cmH_2O and still maintain ventilation with a low RR.
- Low or normal PEEP as patient is already hyper-expanded.
- Ensure adequate sedation

Pulmonary Hypertension (PHTN)

- **Definition:** After birth, PVR should drop precipitously to less than 1/3rd SVR to allow passive blood flow from the relatively weaker RV into the pulmonary bed and onto the LA.
- **Causes**: BPD/CLD, chronic hypoxia (congenital heart disease, CLD), unrepaired pulmonary venous obstruction—PAPVR or TAPVR, idiopathic pulmonary fibrosis, recurrent PE, mitral regurgitation or LVOT obstruction, untreated OSA.
- **Presentation:** episodes of hypoxia and sequalae of right heart strain, see dilated RV and RA on EKG and CXR, ECHO shows tricuspid regurgitation (the "jet" approximates the RV pressures), the ventricular septum may bow into the left ventricular upon systole causing a relative LVOT obstruction:
- **Treatment**: Acute pulmonary hypertensive crisis can be *life-threatening* if the RV cannot overcome PVR and deliver preload to the left side of the heart AND there is no abnormal intra-atrial (ASD or PFO) or intra-ventricular (VSD) connection to allow blood to shunt from the right side of the heart to the left.
  - **Oxygen**: Hypoxia causes pulmonary vasoconstriction, **hyperoxia** = pulmonary vasodilation.
  - **Relative respiratory alkalosis:** Hypercapnia causes pulmonary vasoconstriction = worse PHTN
  - **iNO**: ubiquitous local vasodilator produced by the endothelium in all tissue beds with an extremely short half-life, can only cause vasodilation of capillaries in alveoli being ventilated
  - **Sedation and paralysis**: avoid any sympathetic stimuli leading to systemic HTN or PHTN
  - **Pulmonary vasodilators**

CARDIOVASCULAR

Cardiac output (CO) = Stroke volume x heart rate
Blood pressure = CO x Systemic Vascular Resistance
$\dot{D}O_2$ (oxygen delivery) = C_aO_2 (O2 content of blood) x CO

Shock

Definition: Failed oxygen delivery, transport or uptake at the cellular level necessitating anaerobic metabolism, if prolonged leads to multi-organ failure and death.

- **Compensated shock:** Intrinsic systemic and cellular mechanisms are supporting metabolic needs, subtle overt clinical signs: irritability, *tachycardia*, hypertension, poor cap refill.
- **Uncompensated shock:** Compensations are overcome and perfusion to microvasculature fails causing cellular function to deteriorate = overt organ failure. Present with hypotension, lactic acidosis, and evidence of organ failure such as anuria or lethargy.

Delivery Problem (Low S_vO_2)	Distribution Problem (Not reaching vital organs, ↑S_vO_2)	Extraction Problem (High S_vO_2)
Cardiogenic shock	Anaphylactic shock	CO poisoning
Hypovolemic shock	Septic Shock	Methemoglobinemia
Obstruction (PE or PTX)	Neurogenic shock	CN poisoning
Severe anemia		

Commonly used terms in shock:

- **Warm shock:** warm extremities, flash cap refill, bounding peripheral pulses, evidence of poor vital organ perfusion (AMS, oliguria). Usually signifies distributive shock or low SVR.
- **Cold shock:** cool extremities with delayed cap refill, poor peripheral pulses, mottling, evidence of poor vital organ perfusion (AMS, oliguria). Consistent with septic shock or high SVR states.
- **Fluid refractory shock:** hypotension, tachycardia despite 60ml/kg of IVF boluses
- **Catecholamine resistant shock:** hypotension despite maximal doses of epinephrine and norepinephrine
- **Refractory shock:** resistant to IVF, inotropes, vasopressors, steroids and metabolic correction

Treatment: Therapeutic efforts are geared towards stabilizing cellular function by improving oxygen supply, delivery, correcting metabolic derangements and decreasing oxygen demands.

- Increasing oxygen supply and delivery
  - Oxygen delivery = **$\dot{D}O_2 = CO \times C_aO_2$**
    - **$CO = HR \times SV$** → improve HR with chronotropic agents, use inotropes to improve cardiac contractility and manipulate preload with IVF and vasopressors to improve SV.

        - **C_aO_2=(Hgb x 1.34 x S_aO_2) + (P_aO_2 x 0.003)**→ Hgb >7gm/dL, improve S_aO_2 and P_aO_2 with CMV if needed, manipulate the Hgb-O_2 dissociation curve to unload O_2 at the tissue level, i.e. shift to the right = **AVOID ALKALOSIS** and **HYPOTHERMIA**.
- <u>Decreasing oxygen demands</u>
    - Sedation to prevent anxiety, mechanical ventilation to help with work-of-breathing, avoid hyperthermia or seizures, avoid cardiac arrhythmias, neuromuscular blockade
- <u>Correct any metabolic abnormalities</u>
    - Metabolic acidosis is the result of anaerobic metabolism from inadequate tissue perfusion, it will correct as oxygenation and perfusion to tissues improves.
        - pH correction is occasionally warranted before tissue perfusion can be restored, pH <7.2 and profound lactic acidosis in the setting of renal and hepatic failure.
    - Ensure normal iCa for vasomotor tone and cardiac membrane stability
    - Ensure normal K for cardiac conduction and contraction
    - Ensure normal glucose for metabolic demands.

Inotropes and Vasopressors

	Receptors	Doses	Indications	SE/Notes
Dopamine	*D_1*:dilated renal & mesenteric vessels *β_1*:↑HR and contractility α_1: art & veno-constriction	*Low (D_1)*=1-5 *Middle (β_1)*=5-10 *High (α_1)* = 10-20 mcg/kg/min	Fluid refractory septic shock, hypovolemic and cardiogenic shock	Tachycardia, immune-suppression; may increase PVR at doses >7mcg/kg/min
Epinephrine	β_1: ↑HR and ↑contractility β_2: vasodilation α_1: arterial & venous constriction	0.02-1 mcg/kg/min As escalate, goes from β_1 to β_2 to α_1	Fluid refract. septic shock, Anaphylaxis, Cardiogenic shock, Post-arrest	Tachyarrhythmia, myocardial ischemia by ↑metabolic demand and ↑afterload on LV
Norepinephrine	α_1: vasocon-striction Minimal β_1	0.01-2 mcg/kg/min	Warm septic shock	Tachyarrhythmia (< risk than Epi)
Vasopressin	V_2: constriction of capillaries and arterioles. V_1: ↑H_2O uptake by the renal collecting ducts	0.3-2 milliunits/kg/min; *GI Bleed (2-10), DI (0.5-10 mu/kg/hr)*	Warm septic shock, catecholamine-resistant shock	Venous thrombosis, hyponatremia
Milrinone	Phosphodiester-ase Inhibitor= ↑cAMP, ↑ contractility & lusitrophy, ↓SVR =↓LV afterload	0.2-1 mcg/kg/min	Cardiogenic shock, Cold septic shock, Pulmonary hypertension	Hypotension, ventricular ectopy, long-half life (3hrs), renal excretion (must renally dose)
Phenyl-ephrine	α_1: vasoconstriction	0.05-0.5 mcg/kg/min	Neurogenic shock	Reflexive bradycardia

	Excessive tachycardia when at each body temperature								SBP 5%ILE FOR AGE
Age	**37.8**	**38.3**	**38.9**	**39.4**	**40**	**40.6**	**41.1**	**41.7**	
< 2 years *	>180	>185	>190	>195	>200	>205	>210	>215	*<30 days: 60* *1mo- 2y: 70*
2 - 5 years	>140	145	150	155	160	165	170	175	75
6 - 12 years	>130	135	140	145	150	155	160	165	85
13 - 18 years	>110	115	120	125	130	135	140	145	90
*Also qualify if bradycardic:			< 1 month: HR <100				1 - 23 months: HR < 90		

Pheonix Sepsis Score

	0 points	1 Point	2 Points	3 Points
Respiratory (0-3 points)	PaO2: FiO2 >/= 400 or SpO2:FIO2 >/= 292	PaO2: FiO2 >/= 400 and any resp support or SpO2:FIO2 >/= 292 and any resp support	PaO2: FiO2 100-200 or SpO2:FIO2 148-220 and IMV	PaO2: FiO2 <100 and IMV or SpO2:FIO2 < 148 and IMV
Cardiovascular (0-6 points)		1 point each (up to 3) for	2 points each (up to 6) for	
	No vasoactive meds	1 vasoactive med	>/= 2 vasoactive meds	
	Lactate < 5 mmol/L	Lactate 5-10.9 mmol/L	Lactate >/= 11 mmol/L	
Mean arterial pressure by age				
<1 month	>30	17-30	<17	
1-11 month	>38	25-38	<25	
1-< 2 years	>43	31-43	<31	
2- <5 years	>44	32-44	<32	
5- <12 years	>48	36-48	<36	
12- 17 years	>51	38-51	<38	
Coagulation (0-2 points)		1 point each for		
	Platelets >100	Platelets < 100		
	INR </= 1.3	INR >/= 1.3		
	D Dimer </= 2	D Dimer > 2		
	Fibrinogen >/= 100	Fibrinogen <100		
Neurologic (0-2 points)	GCS > 10	GCS </= 10	Pupils fixed bilaterally	

Heart Failure

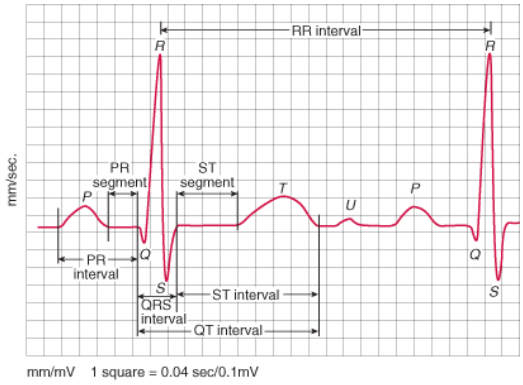
- **Right-sided heart failure (*cor pulmonale*)**: inability of the right side of the heart to overcome its afterload, usually pulmonary vascular resistance, and deliver an adequate amount of preload to the left side of the heart to create systemic CO.
    - *Causes:* HLHS, unrepaired VSD, Pulmonary valve obstruction or insufficiency, RVOT obstruction, tricuspid regurgitation, long-standing or acute pulmonary hypertension, unrepaired TAPVR
    - *Presents with:* hypoxia without pulmonary edema, HSM (portal HTN), peripheral edema, high CVP, high BNP.
    - *Treatment:* optimize preload to the right side of the heart (goal CVP > RAP), decrease PVR (O_2, iNO, milrinone), inotropes to support right-heart.
- **Left-sided heart failure**: the systemic ventricle cannot produce adequate CO to the body.
    - *Causes*: Coarctation of the aorta, Aortic atresia or insufficiency, mitral regurgitation, HLHS, hypertrophic cardiomyopathy, long-standing systemic hypertension
    - *Presents with*: pulmonary edema, multi-organ failure, peripheral edema, dyspnea on exertion, clubbing, high CVP, low S_vO_2
    - *Treatment*: limit afterload on the LV using diuretics, inotropes and vasodilators (ACE-I, milrinone), limit oxygen/metabolic demands on the heart →potentially sedate, paralyze and intubate.**

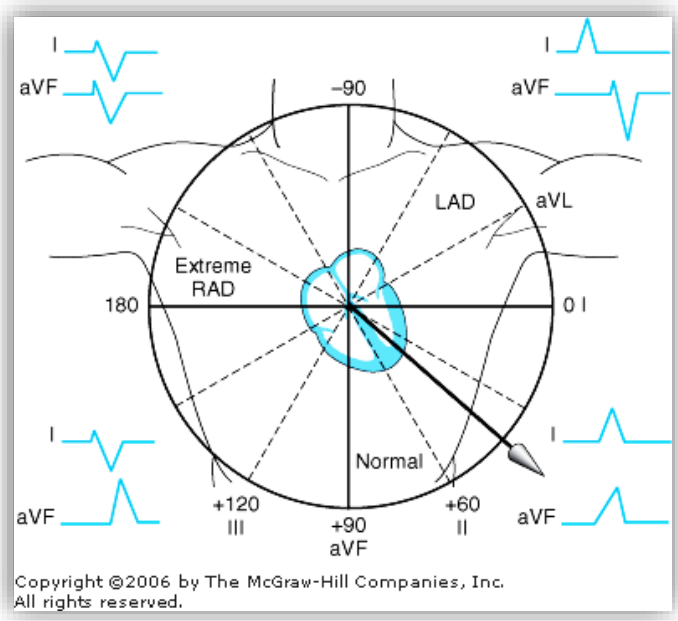
****REMEMBER:** intubation in cardiogenic shock or any form of cardiac dysfunction is life-threatening because of the change of the intra-thoracic pressure from negative to positive limiting the venous return to the right side of the heart. IVF are often needed and a code chart should be open with code medications ready.

- **BI-Ventricular Heart Failure**
    - *Presentation*: feeding intolerance, constipation or diarrhea, dyspnea, FTT, global or pulmonary edema, newborn from 7 to 60 days of life with irritability (remember ductal dependent lesions) presenting in shock with cyanosis:
    - For newborn in shock, suspect congenital heart disease and ***ASAP*** start prostaglandin (PG_E) 0.01-0.4mcg/kg/min and watch for **APNEA**.
    - ***Red flags!*** The patient is on ANY of these outpatient CHF medications:
    - Labs: VBG with **S_vO_2** and **lactate**, troponin, BNP, EKG, chemistry (want K>4.0), iCa (ideal>4.5), Mg (ideal >2.0), Phos, LFTs, coagulation panel, CBC, CXR to evaluate heart size and check for pulmonary edema.
    - Initial resuscitation: if any sequelae of heart failure are present on exam (hepatomegaly, rales, poor peripheral perfusion, gallop or murmur on exam, refractory hypoxia), fluid resuscitation may be detrimental, give only in 5-10ml/kg aliquots and assess carefully after for worsening clinical status, lasix +/- inotropes may be in order. Check for an **arrhythmia** which could worsen heart function.

EKG Basics

- Confirm EKG is your patient's.
- Confirm EKG is at 10mm/mV with a speed of 25mm/sec.
- Determine ventricular rate.
- Check rhythm: is there a p before every QRS?
    a. Yes: sinus.
    b. No: are there p-waves at all?
- Determine Axis looking at I and aVF.
- Check intervals:





- 1 small block = 0.04sec, large =0.2s.

PR = 0.08-0.15 sec
QRS = 0.03-0.08 sec

QTc = QT /(√RR);
Normal QT_c for <6months = 490sec, >6mo = 440sec

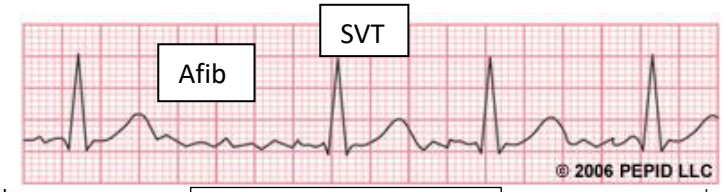
- Look at other waves/heights.
    a. The T-wave changes with age:

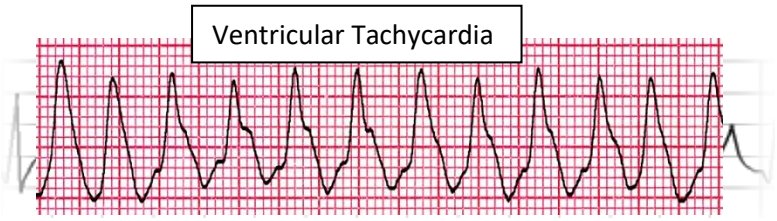
Age	V1-V3	aVF	I, V5,V6
0-7d	↑	↑	↑/↓
1w-16y	↓*	↑	↑
16y-Adlt	↑	↑	↑
*Inverted T-waves can correct by 8yo			

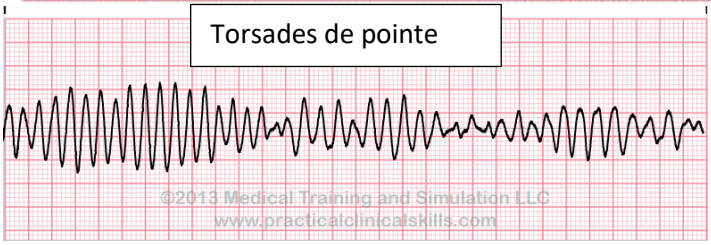
LVH: R-wave >95%ile in V5/V6 or <5%ile in V1/V2, Q wave > 4mm in V5/V6, inverted T in V6
RVH: R-wave >98%ile in V1, Q-wave in V1, S-wave >98%ile in I/V6), ↑T in V1/V4
Myocarditis: Low QRS amplitudes, long PR
Pericarditis: Low QRS amp, ST depression
BBB: QRS>120msec, RAD in RBBB

**Q-waves are nml if <0.03s, present in V1-3 and aVF and less than 5mm deep.

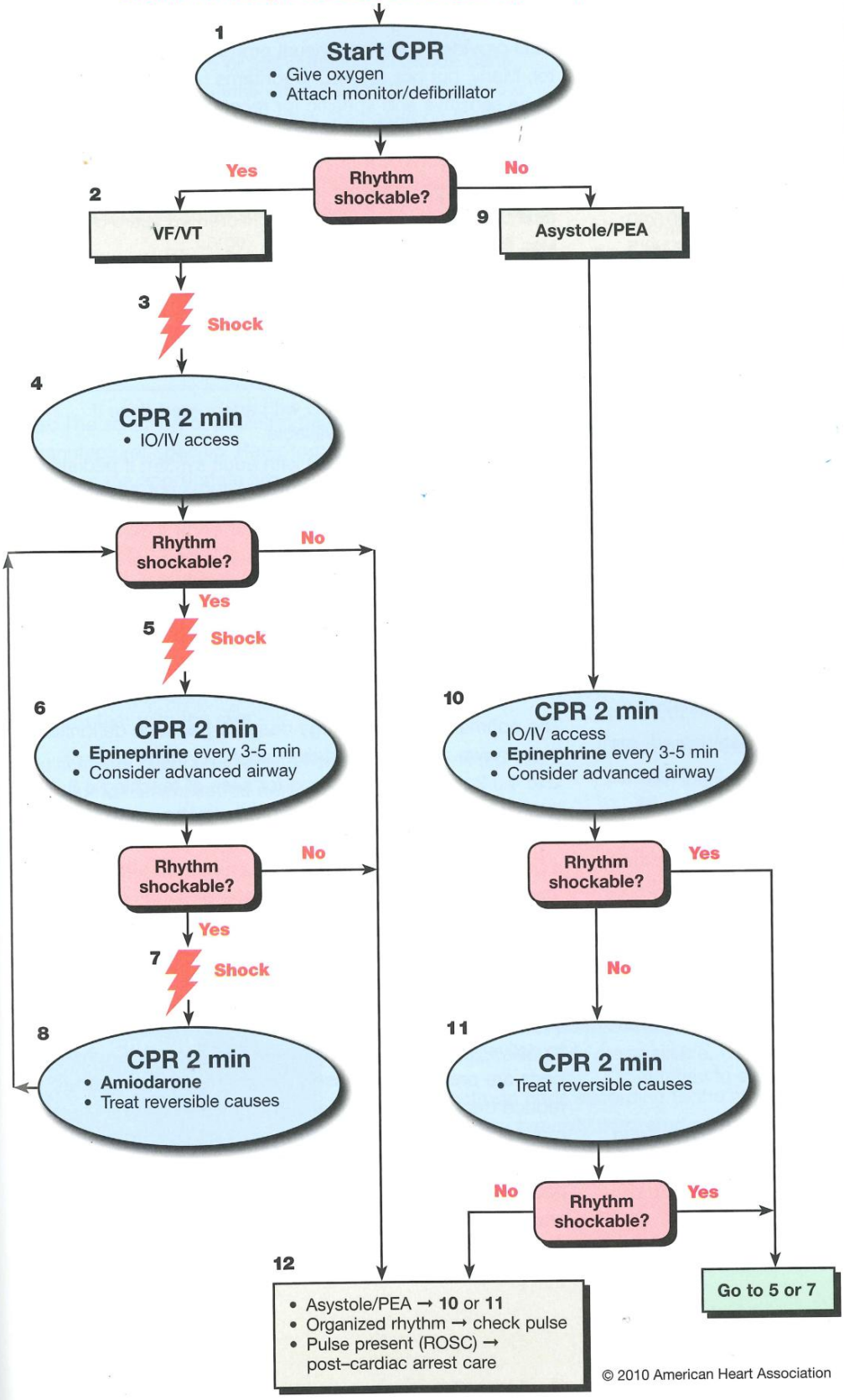
Common Pediatric Arrhythmias:







Pediatric Cardiac Arrest



Doses/Details

CPR Quality

- Push hard (≥1/3 of anterior-posterior diameter of chest) and fast (at least 100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 15:2 compression-ventilation ratio. If advanced airway, 8-10 breaths per minute with continuous chest compressions

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 IO/IV Dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV Dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place give 1 breath every 6-8 seconds (8-10 breaths per minute)

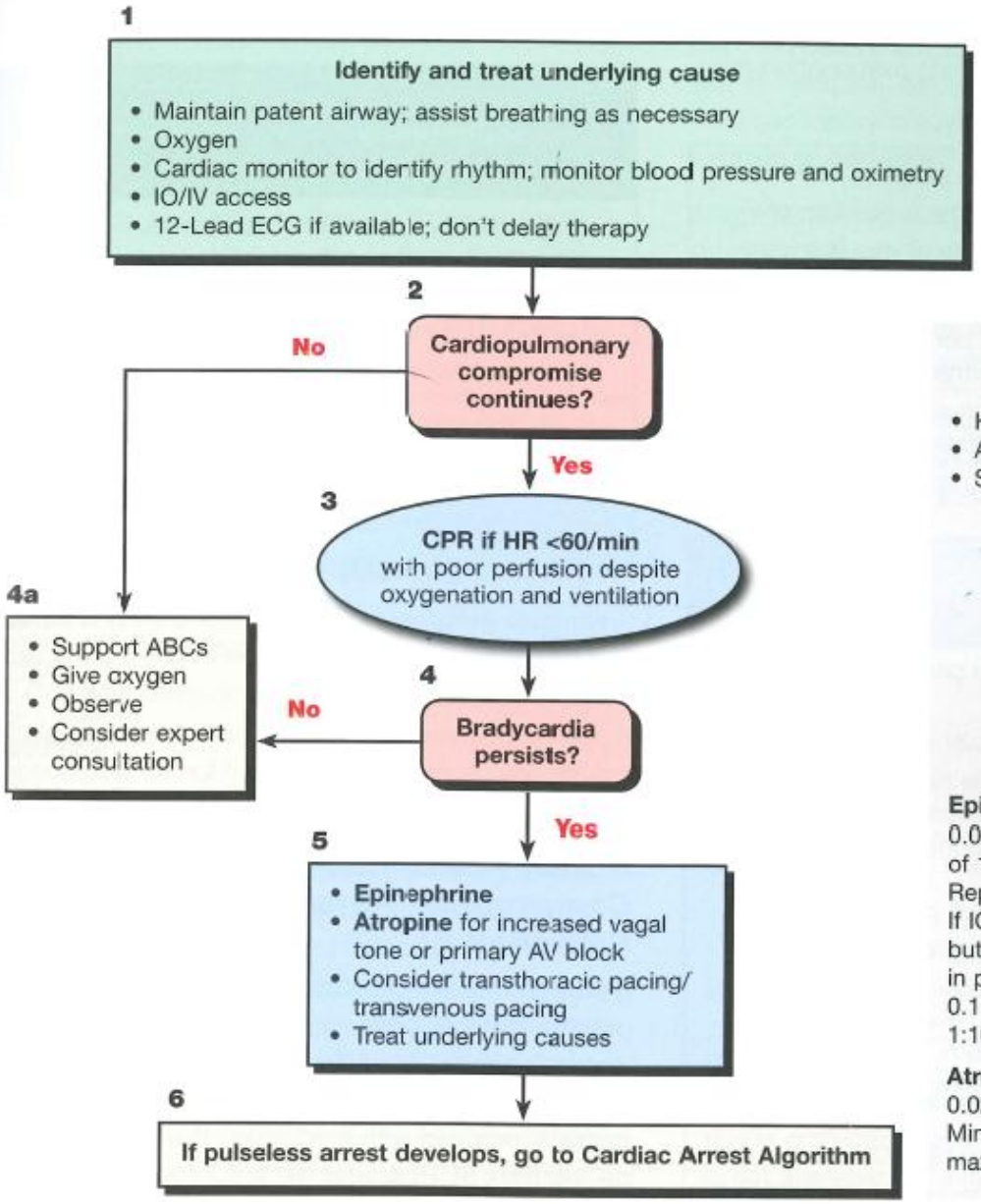
Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

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

Management: Pediatric Bradycardia With a Pulse and Poor Perfusion Algorithm



Cardiopulmonary Compromise

- Hypotension
- Acutely altered mental status
- Signs of shock

Doses/Details

Epinephrine IO/IV Dose: 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If IO/IV access not available but endotracheal (ET) tube in place, may give ET dose: 0.1 mg/kg (0.1 mL/kg of 1:1000).

Atropine IO/IV Dose: 0.02 mg/kg. May repeat once. Minimum dose 0.1 mg and maximum single dose 0.5 mg.

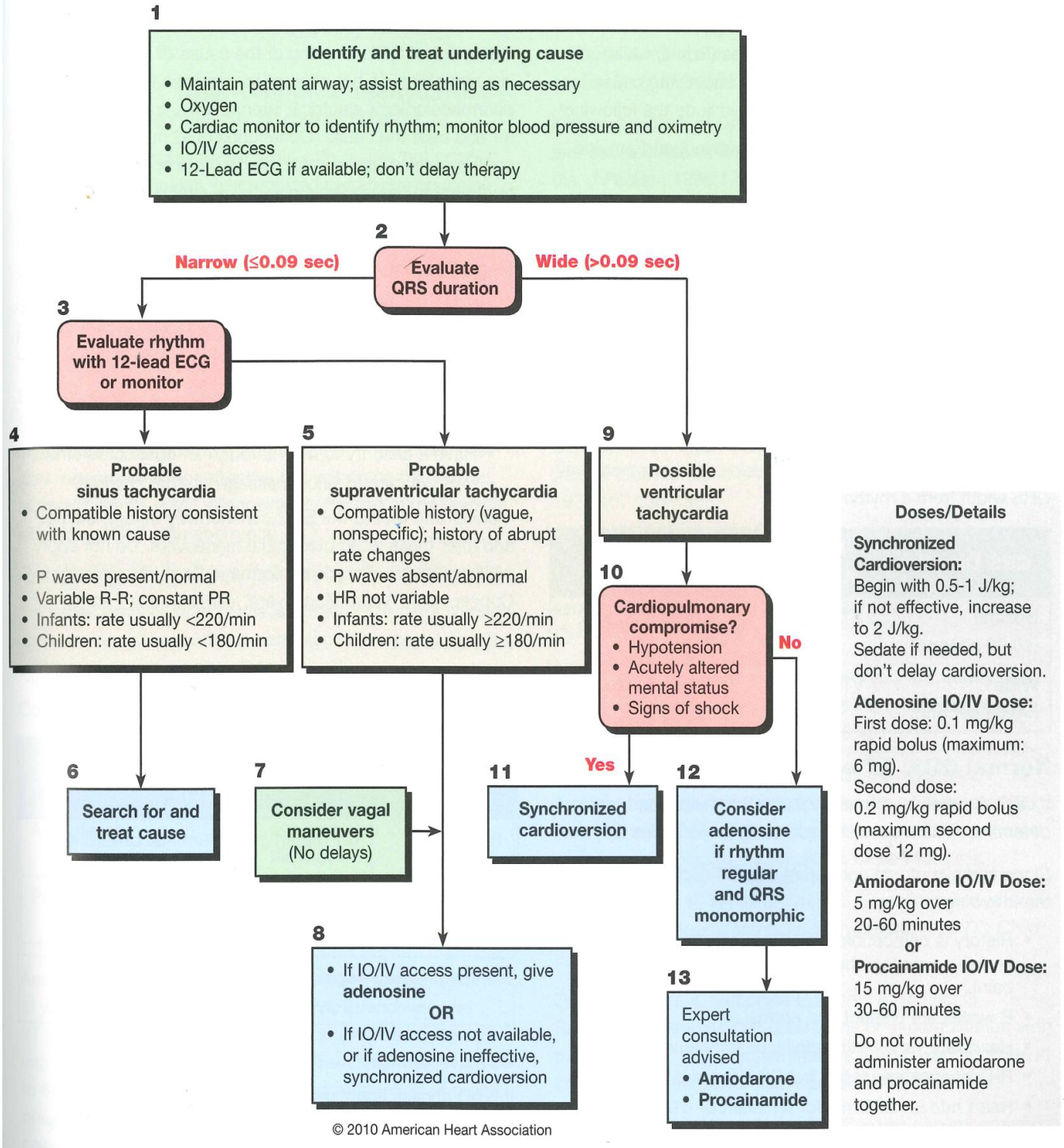
BRADYCARDIA

Check for pulses and perfusion (mental status, BP)

Start CPR and give **epinephrine 0.01mg/kg** or **atropine 0.02mg/kg.**

Tachyarrhythmia	Treatment
SVT	Adenosine 0.1-0.2mg/kg → synch cardioversion 0.5-1J/kg
JET	Cooling, sedation, stop catecholamines → digoxin → amiodarone 5mg/kg IV over 30-60mins
Vtach	+pulse → synch cardioversion; -pulse → defibrillation 2-4J/kg; lidocaine, amiodarone, procainamide
Torsades de pointe	Magnesium 25mg/kg IV/IO, then Vtach management

Pediatric Tachycardia With a Pulse and Poor Perfusion Algorithm



NEUROCRITICAL CARE

ELEVATED ICP

Order set: Pediatric ICU Severe Traumatic Brain Injury (16192)

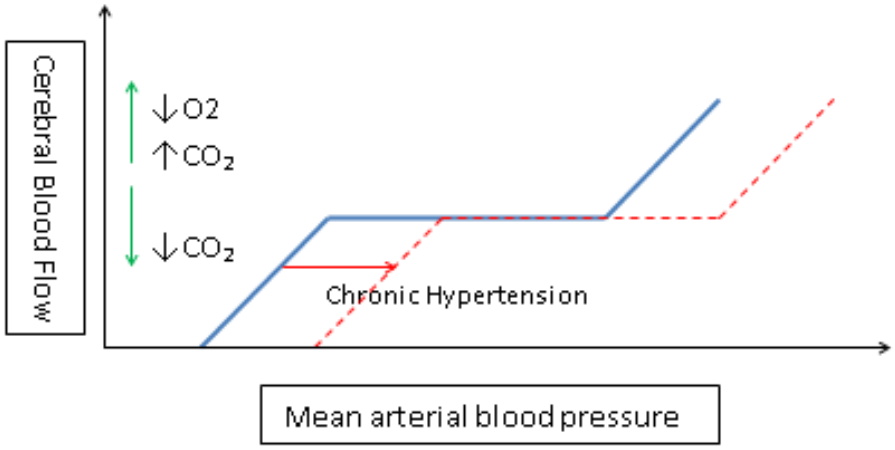
The intracranial vault is a closed compartment composed of blood, brain, and CSF. If one compartment enlarges, the others must be compressed and eventually intracranial pressure will rise. Once ICP rises above mean arterial blood pressure, blood flow is compromised to the brain according to this relationship:

Cerebral perfusion pressure (CPP)=mean arterial pressure - intracranial pressure

Goal CPP should be 40-60mmHg, normal ICP is < 20mmHg.

Cerebral blood flow is preserved over varying BPs by **cerebral auto-regulation.**

This curve can be shifted for patients with chronic HTN and chronic CO_2 retention. That is you **never quickly** correct a chronically HTN patient to BPs.



with why normal

Causes of Elevated ICP:

1. *Cerebral edema:*
   i) *Vasogenic*: capillary leak from the brain endothelium as seen in SIRS, meningitis, ICH, CVA, lead intoxication
   ii) *Cytotoxic*: the cells in the brain themselves swell from cellular energy failure (ATP-production) as seen in HIE, DAI, osmolar injury as in hyperosmolar correction (CE-DKA).
   iii) *Interstitial*: obstructed CSF = ↑water content of the periventricular white matter.
2. *Intracranial lesions*: ICH, tumor, abscess, AVM, focal edema from a contusion or CHI
3. *CSF obstruction*: anatomic abnormality, mass, or meningeal dysfunction from infection or resolving ICH.

Presentation: Altered LOC, vomiting, posturing, abnormal CN exam, impaired pupillary reflex, lateral rectus palsy, sun-downing, papilledema, bulging fontanelle (especially at rest), Cushing's triad.

Cushing's triad: brainstem's attempt to maintain perfusion = increased MAP to maintain CPP causes reflexive bradycardia, hyper or hypoventilation possible.

↑BP + ↓HR + abnormal RR = VERY LATE AND BAD SIGN!

Reference Guide for the appearance of intracranial hemorrhages on MRIs

MRI APPEARANCE OF HEMORRHAGE

Stage	Biochemistry	Pathophysiology	Location	Magnetism	Appearance T1W/T2W
Hyperacute (hrs)	Oxy-Hb	Serum + RBCs	Intracellular	Diamagnetic	◐ ○
Acute (1-2 days)	Deoxy-Hb	Deoxygenation	Intracellular	Paramagnetic	◐ ●
Early subacute (2-7 days)	Met-Hb	Oxidation/ denaturation	Intracellular	Paramagnetic	○ ●
Late subacute (1-4 wks)	Met-Hb	RBC lysis	Extracellular	Paramagnetic	○ ○
Chronic	Hemosiderin Ferritin	Iron storage	Extracellular	Ferromagnetic	● ◎

SEVERE TBI TREATMENT Algorithm

Initial Resuscitation and Trauma Assessment

A: Assess airway and secure if **GCS<8** or gag/cough lost
B: Avoid hypoxia, hyper or hypoventilation, P_aCO_2 40mmHg
C: Avoid hypotension with fluid resuscitation
E: Do not rewarm unless hemodynamically compromised

Head CT → **Surgical evacuation**

Tier 1 Therapies:
1. ICP Monitor: Maintain CPP >40-65 and ICP < 20mmHg
2. Neuroprotective measures†
3. Sedation and analgesia
4. Anti-epileptics, if indicated

ICP>20 → **Drain CSF from EVD**

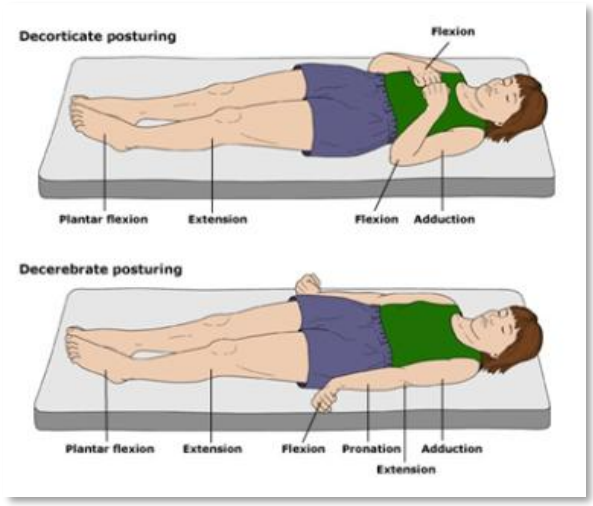
ICP>20 → **Neuromuscular blockade**

ICP>20 → **Hyperosmolar Therapy**

ICP>20 → Repeat Head CT → **Decompressive Craniectomy**

Tier 2 Therapies:
1. Hypothermia 32-33° C x 48hrs
2. Coma
3. Lumbar Drain
4. Continuous EEG

†Neuroprotective measures =
1. Avoid swings in temperature (no fever), Na, CO_2 and glucose.
2. Head midline and HOB at 30°
3. Optimize sedation
4. Avoid/treat seizures
5. Lidocaine with suctioning.



http://uptodateinmedicine.blogspot.com/2012_02_01_archive.html

PICU TBI management

Goal to prevent ***secondary*** injury to the brain.

Start with the A/B/C's on initial presentation:

A. Check GCS to determine if can protect airway. **GCS < 8 = severe TBI = intubate.**

	1	2	3	4	5	6
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

B. Intubation should be in a controlled setting using *maximal* sedation to avoid ICP spikes from the noxious stimuli of intubation.

C. Avoid sedation meds which ↓BP significantly to ↓CPP (morphine, versed, etc.)

D. Ideal medications:

i. Pre-medicate with **atropine** and **lidocaine**

ii. Sedate with propofol, etomidate, or fentanyl. Paralyze with Rocuronium.

iii. ***NO SUCCINYLCHOLINE as could ↑ ICP***

E. Ensure normal hemodynamics and fluid status. Use only isotonic fluids: D5NS if hypoglycemic or simple NS if not. Resuscitate with normal saline always.

1. **Neuroprotective measures**: HOB at 30 degrees, head midline, avoid glucose or Na variability, avoid fever, sedate and treat pain to avoid sympathetic nervous system stimulation of ICP.
2. **Hyperventilation and O_2**: manipulation of the brain's own homeostatic mechanism. ↓PCO_2 and↑O_2 decreases CBF to temporarily decrease ICP by dropping blood flow to the brain.
   a. Hyperventilation is <u>only warranted</u> in impending herniation.
   b. **NEVER drop $PaCO_2$ <35mmHg (PvO2<40)** or CBF will be severely ↓leading to more ***<u>ischemia</u>***.
   c. Only a temporizing measure (works for <24 hours), worsens outcomes if persistent.
3. **Hyperosmolar therapy**: Neither 3% NS nor mannitol cross the BBB, therefore, they have rheologic and osmotic properties which lower cerebral blood volume and free water content of healthy brain cells.
   a. <u>Mannitol</u>: immediately decreases blood viscosity causing a decrease in cerebral blood vessel diameter = decreased cerebral blood volume and ICP, gradually draws free water out of the cells by creating an osmotic gradient (lasts 6 hours). Requires an intact BBB.
      i. 0.5-1gm/kg can be given every 4-6 hours. Causes osmotic diuresis so ensure adequately volume-resuscitated and not oliguric or anuric. Foley helpful. <u>*Maximum serum osmolarity < 320mOsm/L*</u> or can precipitate renal failure.
   b. <u>Hypertonic saline (3%):</u> unlike mannitol, 3% NS also restores normal cellular resting membrane potential and cell volume, inhibits inflammation, and ↑cardiac output.
      i. 5mL/kg bolus or continuous drip to goal Na 145-155, <u>*goal serum osm< 360*</u>.
4. **Neurosurgical intervention**:
   a. <u>ICP monitor</u>: subarachnoid bolt, intraventricular drain, intraparenchymal catheter (Camino), directly measure the patient's ICP to allow titration to goal CPP.

b. EVD: extraventricular drain allows CSF removal if ICP >20mmHg for > 5minutes, can also measure ICP if needed. Is set at the bedside at a certain height to deliver a certain pressure (negative or positive) to the ventricle in cmH_2O. Set at the level of the tragus.
c. Decompressive craniectomy: effective in recoverable brain injury before significant secondary injury has occurred.

ICU Management in TBI:

1. **FEN**: use isotonic IVF and ensure adequate hydration. Remember, energy expenditure can be increased. Inititate full enteral nutrition ≤ 72 hours after injury.
    a. In hyperglycemia→ give insulin to control serum glucose. Goal glucose >100 and < 200.
2. **Resp**: maintain normal ventilation and adequate oxygenation. MUST follow end-tidal CO_2 or blood gases closely.
3. **Neuro**: Neuro-protective measures are VERY important as mentioned above
    a. Steroids: for limiting *focal* vasogenic edema ONLY, neurosurg will indicate when to use.
    b. Anti-epileptics: prolonged seizures increase metabolic demand of injured brain, raise ICP and cause secondary brain injury. TBI= ↑hepatic metabolism → follow serum AED levels if using phenytoin.- Empiric therapy with Keppra or phenobarbital more common.
    c. Normothermia: do not actively warm if cold- can passively warm, avoid shivering with paralytic
    d. Coma: suppress the metabolic rate of the brain, lower ICP, and inhibit free-radical mediated lipid peroxidation and stabilize cell membranes. Peak metabolic effect seen when burst suppression achieved on EEG.
        i. Pentobarbital: Load with 6-8mg/kg over 30 minutes, then 1-4mg/kg/hr
            1. SE: myocardial depression, **hypotension**
            2. Used only for *refractory* Intracranial HTN.
4. **GI**: PPI or H_2 blocker important to avoid CNS-related gastric ulcer.

STATUS EPILEPTICUS

Definition: Seizure activity lasting > 30 minutes or clusters of seizures for > 30 minutes without return to baseline mental status in between seizures.
Causes: seizure disorder with sub-therapeutic AEDs, hypoglycemia, electrolyte abnormalities, encephalitis, meningitis, stroke, sepsis, ingestion, trauma, metabolic or mitochondrial disorders, vasculitis, increased ICP, hypertensive crisis, PRES, ADEM, complex febrile seizure.

Status Epilepticus pathway:

Seizure > 5 minutes → **Ativan 0.1mg/kg** IV or **Diastat 0.2-0.5mg/kg** PR → *sz continue* → can give another **Ativan 0.1mg/kg** or if <1mo give **Phenobarbital 20mg/kg**, if > 1mo give **Keppra 60mg/kg** → *sz continue* → give additional **phenobarbital 20mg/kg** OR **Keppra**→ if *still seizing*, consider **versed/Propofol** drip

- Provide supplemental O_2 and assess ABCs. (May need respiratory or hemodynamic support.)
- Neurology should be called early to start EEG if subclinical status suspected.
- **RULE OF THUMB**: during a seizure eyes are usually open and dolls eyes (oculocephalic) reflex is **not** intact.

Potential Status Epilepticus Labs: ***immediate d-stick***, electrolytes, Head CT, consider LP, tox screen, LFTs, coagulation panel, metabolic screen (lactate, pyruvate, urine for ketones, ammonia, urine and serum amino and organic acids), call about the Newborn Screen, CK.

BRAIN DEATH

Order set: Pediatric ICU Gift of Life Organ Donation (16440)

Definition: *Irreversible* loss of all function (including brainstem) due to total necrosis of the cerebral neurons. Three essential findings: Coma, Absent brainstem reflexes, Apnea. **Once brain dead, a patient is clinically dead.**

To determine brain death (BD):

1. Ensure cause of neurologic state is *irreversible*.
    a. Exclude any complicating medical conditions (eg electrolyte, acid-base or endocrine disturbances) or confounding factors like hypotension, hypothermia (T<32°C) or drug intoxication (barbiturate levels need not be 0 but in a range not causing CNS depression).
2. Initiate notification of next-of-kin.
3. Conduct and document the first clinical assessment of brain stem reflexes:
    a. *Pupillary light reflex*: BD c/w no response to light
    b. *Oculocephalic reflex*: normally eyes stay midline when head rotated to left or right
    c. *Cold caloric*: normally the eyes will have lateral nystagmus toward the ear into which 50mL of ice water is quickly irrigated, in BD no nystagmus is produced
    d. *Corneal reflex*: blink and grimace or withdrawal from pressure +/- rubbing over the cornea
    e. *Withdrawal* from noxious stimuli: nail bed pressure, pressure over supraorbital ridge
    f. *Gag or cough reflex*: BD consistent with no response to stimulation of the posterior pharynx or suctioning into the tracheobronchial tree
4. Observe the patient during the period before the next exam for any signs inconsistent with brain death.
    a. Birth to 30 days: 24 hours after birth/injury and 24-hour waiting period (or interval shortened with ancillary study)
    b. 31 days to 18 years: 24 hours after injury/arrest and 12-hour waiting period (or interval shortened with ancillary study)
    c. Conduct the second assessment of brainstem reflexes as outlined in Step 3.
5. Perform and document the apnea test with both exams
    a. Ensure a normal PCO_2, pre-oxygenate with 100% oxygen. Obtain an ABG.
    b. Disconnect the ventilator and watch for any signs of breathing.
    c. Wait 5 to 8 minutes and obtain a repeat ABG. If PCO_2 > 20mmHg above initial PCO_2, apnea test is positive for brain death.
    d. If hypotension or desaturation develops, obtain ABG earlier and reconnect to the ventilator. **If PCO_2 is >60mmHg or >20mmHg above baseline, then apnea test is positive.** If not, the apnea test is indeterminate and should consider confirmatory testing.
6. Perform confirmatory testing, if indicated
    a. CTA, MRA or conventional angiography: an absence of intra-cerebral filling at the level of carotid bifurcation or Circle of Willis
    b. EEG: at least 30 minutes of recording showing absence of electrical activity
    c. Nuclear brain imaging showing absence of uptake of isotope into brain parenchyma
    d. Somatosensory evoked potentials: bilateral absence of N20-P22 response to median nerve stimulation
    e. Transcranial Doppler Ultrasound: small systolic peaks in early systole without diastolic flow or reverberating flow indicates high vascular resistance (high ICP)

7. Certify brain death and withdraw cardiorespiratory support in accordance with policies.
    a. Need 2 separate physicians from 2 different departments to certify brain death.

PROCEDURAL SEDATION

Sedation represents a continuum. Varying levels are needed to minimize patient discomfort, pain or anxiety, and control behavior during procedures to maintain patient safety.

1. **Minimal sedation** = drug-induced state during which patients respond normally to commands although coordination or cognitive function may be impaired. Respiratory and cardiac function unaffected
2. **Moderate sedation** = depressed consciousness during which a patient can respond to commands either alone or after light tactile stimulation. Airway and spontaneous respirations are intact
3. **Deep sedation** = drug-induced depressed consciousness requiring repeated or painful stimulation for arousal; may have impaired respiratory effort and/or airway.
4. **Anesthesia =** loss of consciousness, no arousal even by painful stimulation.

Monitoring and Equipment: HR, SpO_2, blood pressure and RR must all be continuously monitored. Emergency intubation equipment and appropriately sized bag mask need to be available.
Personnel: the proceduralist *cannot* be the person providing sedation.
NPO for: solids>8 hours, milk and formula> 6 hours, breast milk >4 hours, clears >2 hours

Drug	Dose	Duration	Side Effects	Reversal
Fentanyl	1-2mcg/kg IV (max 4-5mcg/kg)	60 minutes	Apnea, rigid chest (if pushed)	Narcan 5-10mcg/kg IV
Midazolam	0.05-0.2mg/kg IV (can give intra-nasally or IM)	60 minutes	Apnea, ↓BP, paradoxical agitation	**Flumazenil 0.01mg/kg IV **Can cause seizures**
Morphine	0.05-0.2mg/kg IV	60 minutes	Apnea, ↓BP, hives	Narcan 5-10mcg/kg IV
Ketamine	0.5-2mg/kg IV/IM	15-30 minutes (Onset confirmed by nystagmus)	Hallucinations, secretions (have robinul or atropine ready)	None
Dexmedetomidine	0.2-1mcg/kg/hr IV infusion	<15 minutes	Bradycardia, hyperglycemia	None
Propofol	1-2 mg/kg IV	<15 minutes	Apnea, ↓BP, metabolic acidosis	None

HEMATOLOGY

ANEMIA

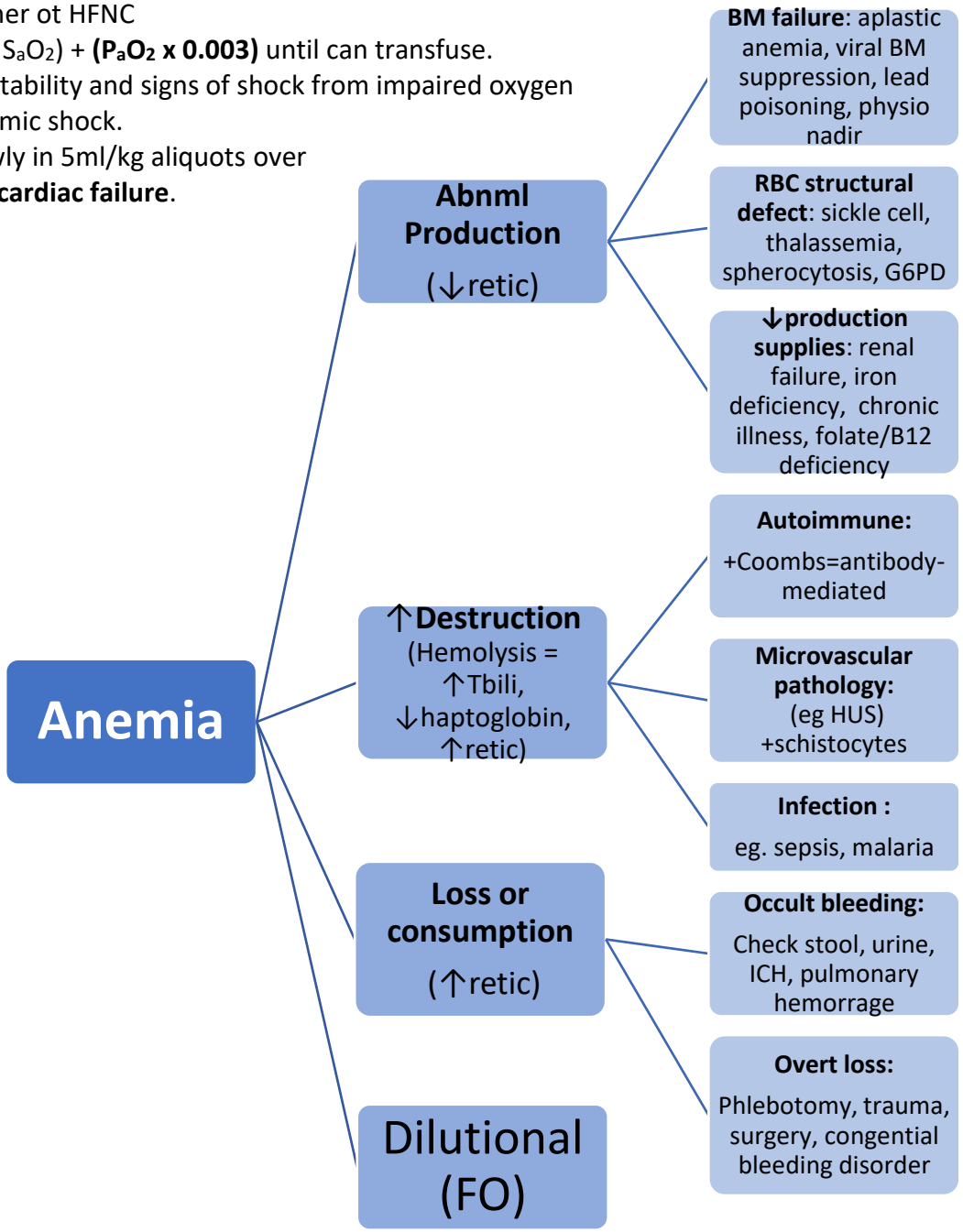
Definition: Hgb < 10gm/dL

Causes: can differentiate types by looking at MCV, reticulocyte count, peripheral smear

Labs: CBC with MCV, RDW and peripheral smear, reticulocyte count, Coombs, Type and Cross, Hgb electrophoresis, viral studies, osmotic fragility test (for spherocytosis), Tbili and dbili, UA, heme check stools, imaging for occult blood, coagulation panel.

Treatment:

1. 100% oxygen with non-rebreather ot HFNC to maximize C_aO_2=(Hgb x 1.34 x S_aO_2) + **(P_aO_2 x 0.003)** until can transfuse.
2. Must assess for hemodynamic stability and signs of shock from impaired oxygen delivery and potential hypovolemic shock.
3. If chronic, should transfuse slowly in 5ml/kg aliquots over 3-4 hours to avoid **high-output cardiac failure**.



THROMBOCYTOPENIA

Definition: **mild** = platelet count<100,000/mm^3, **severe** < 50,000/mm^3.
Causes:

1. ↓production of platelets: BM suppression, medication effect, Fanconi anemia, Wiskott Aldrich
2. ↑destruction or consumption: infection/sepsis, DVT, TTP, ITP*, HUS, ECMO, heparin induced thrombocytopenia* (HIT), transfusion-induced platelet antibody* (*Antibody mediated)
3. Sequestration: hypersplenia, Kasabach-Merritt syndrome
4. Dilutional thrombocytopenia: whole blood loss without repletion of platelets.

DISSEMINATED INTRAVASCULAR COAGULATION (aka Consumptive Coagulopathy)

Definition: uncontrolled thrombin generation leading to microvascular thrombotic disease with resultant end-organ dysfunction and bleeding diathesis related to the consumption of coagulation proteins and fibrinogen.

Diagnosis: largely clinical, adult ICU's use the following scoring system:

Laboratory finding	Range	Points
Platelet Count (x10^3/mm^3)	<80 or >50% ↓ in 24hr	3
	>80 and <120 or >30% ↓ in 24hr	1
	>120	0
INR	>1.2	1
Fibrinogen (mg/dL)	<350	1
Fibrin degradation products	>25	3
	>10 and <25	1
	Total Score >5 consistent with DIC	

Treatment:

1. Treat underlying sepsis or inflammatory condition.
2. ATIII and Protein C have shown promise in adult sepsis-induced DIC; however, there may be an increased risk of IVH in pediatrics with APC use.
3. Replace platelets if < 20,000/mm^3, FFP if PT >30 seconds (INR>2), cryoprecipitate if fibrinogen <50-100mg/dL.

BLOOD COMPONENT REPLACEMENT

**If a patient has significant bleeding (estimated blood volume is 60-80ml/kg), must replace all components of whole blood. If just replace pRBCs, will see "Dilutional DIC and thrombocytopenia."

pRBCs

Source of exogenous Hgb to improve O_2 delivery according to C_aO_2=(**Hgb** x 1.34 x S_aO_2) + (P_aO_2 x 0.003).

1 unit of pRBCs= approx. 250-300mL with a Hct of 50-60%.

To determine volume to transfuse: aprox 10ml/kg of pRBCs = 2.5gm/dL increase in Hgb

Indications:

1. Severe symptomatic anemia (Hgb < 7gm/dL)
2. Hypoxemia from pulmonary pathology and Hgb < 7gm/dL
3. Shock, MODS and Hgb < 7gm/dL
4. Unrepaired cyanotic heart disease and Hgb < 10gm/dL
5. Ongoing bleeding or anticipated blood loss from surgery.

Preparations:

Leukocyte reduction: donor RBCs filtered to remove any remaining WBC, used to prevent febrile, non-hemolytic reactions

Irradiated: destroys any nucleated cells, i.e. WBC, to prevent GVHD.

Washed: To reduce the remaining plasma which includes potential allergens and cellular debris from old blood, i.e. in renal failure to avoid a K^+ load or in previous allergic reaction to pRBCs.

Complications: Hyperkalemia, hypocalcemia, low DPG = shifts of oxygen-Hgb dissociation curve to the left making it harder to unload O_2 at the tissues. Risk of blood-borne illness (rare). Transfused RBCs are less malleable so cannot fit into capillaries as well and have free heme and NO which cause inflammation and immunosuppression = ↑the risk of secondary HAI.

PLATELETS

Available as pools of platelet concentrates (spun down portion of 1 unit of donated whole blood=$5.5x10^{10}$ plt each) OR apheresis platelets in single or double units (one donor, single=$3x10^{11}$). No difference clinically, although apheresis units are easier to handle in the blood bank.

1 unit PC = 40-50mL and 1 pheresis unit = 200-300mL.

1 PC = 5-10,000 plt response on CBC.

All platelets are leukocyte reduced to reduce risk of GVHD.

Transfused platelets last 4-5 days in healthy patients, up to 7-10,000 plts are consumed daily in critical illness.

Indications: severe thrombocytopenia with active bleeding or at risk for spontaneous bleeding.

1. Spontaneous bleeding not likely until <5,000. Risk increases if coagulopathic or infected, consider transfusion when <10-20,000 without bleeding.
2. Transfuse if undergoing a procedure when < 50,000, unless a CNS procedure or ICH then transfuse if < 100,000.
3. Transfusion NOT indicated in ITP, TTP and HUS unless having life-threatening bleeding.
4. Contraindicated in HIT because may precipitate DIC.
5. Disorders of platelet function.
   a. NOTE: Transfused platelets will still be dysfunctional in uremia.

Complications:

1. Rare risk of Hep C, B, HIV
2. Febrile non-hemolytic reactions FAR more common than after pRBC transfusion.
3. Higher risk of bacterial infection and anaphylaxis because stored at room temperature (cytokines can accumulate).

****DO NOT PUSH platelets****

FFP (Fresh frozen plasma)

After 1 unit of whole blood is spun down to extract RBCs and remove WBCs, the whole plasma is stored and frozen, contains coagulation factors as needed for correcting prolonged PT and PTT.

1 unit = 250mL, needs to be ABO compatible.

10-15mL/kg, to provide ~15-20% rise in factor levels.

Indications:

1. Coagulopathy in the setting of potential bleeding (procedures, surgery, trauma) or active bleeding.
2. DIC
3. Acute liver failure with bleeding
4. Massive blood loss (>60ml/kg) and multiple pRBC transfusions

CRYOPRECIPITATE

The insoluble portion of FFP that precipitates when plasma is thawed = primarily concentrated fibrinogen.

1 unit from a single donor = 10-15mL;
1 unit=80IU of Factor VII, 150mg of fibrinogen, von Willebrand factor and factor XIII.
Approximately 2 units/10kg= 100mg/dL increase in serum fibrinogen.

Indications:

1. Hypofibrinogenemia and active/suspected bleeding or prior to an invasive procedure
2. VWDz when conventional treatments not available (eg. DDAVP)
3. Dysfibrinogenemias, inherited or acquired, as seen in acute liver failure
4. DIC with active bleeding

BLOOD TRANSFUSION REACTIONS

1. Acute hemolytic transfusion reactions: Antigen/antibody response to incompatible blood leading to complement activation and intravascular hemolysis.
    a. *Presentation*: fever, rigors, chest pain, tachycardia, hypoxemia and tachypnea, urticaria, angioedema, hypotension and shock, hemoglobinuria
    b. *Diagnosis*: positive DAT (direct antiglobulin test)
    c. *Treatment*: stop transfusion, send donor blood to blood bank for testing, support respiratory and cardiovascular systems as needed, may need red cell exchange
2. Febrile non-hemolytic reactions: preformed anti-HLA antibodies in the recipient which react to donor WBC or platelets triggering cytokine release.
    a. *Presentation*: fever within 4 hours of transfusion, chills/rigors, headache, nausea and vomiting, tachycardia
    b. *Treatment*: antipyretic (acetaminophen), leukocyte reduction if pRBC, specialized cross-match
3. Allergic, anaphylactic reactions: recipient has an IgE antibody directed against an antigen in the donor plasma; seen in IgA deficient patients who may have an antibody to IgA.
    a. *Presentation*: wheezing, urticaria, vomiting/diarrhea, angioedema, airway swelling, cyanosis, hypotension, shock, AFEBRILE
    b. *Diagnosis*: can send IgA levels to diagnose deficiency
    c. *Treatment*: anaphylaxis = IM epinephrine, may need an infusion, Benadryl, H2 blocker, steroids;
        i. Benadryl and steroids prior to all future transfusions
        ii. IgA deficient patients can be given washed pRBCs in the future to minimize rxn.
4. TRALI: HLA-mediated compliment activation and neutrophil migration to the lungs. Neutrophils cause capillary leakage and pulmonary edema/damage.
    a. *Presentation*: respiratory distress, hypoxia within 6 hours after transfusion, bilateral pulmonary infiltrates on CXR, fever, mortality rate of 10%
    b. *Diagnosis*: check for antibodies against HLA or neutrophil specific antigens, the blood bank will test the donor blood and cross-match.
    c. *Treatment*: Provide oxygen, +/-mechanical ventilation, steroids not indicated

5. GVHD: engraftment of donor T-cells that are HLA-incompatible with the recipient causing immune mediated attack on BM, liver, skin, GI tract.
    a. *Presentation*: fever, diarrhea (bloody or watery), maculopapular rash, hepatitis, pancytopenia occurring 8-10 days after a transfusion
    b. RARE, usually seen in immune-compromised patients, can be avoided by **irradiating** blood components
6. ***PICU Transfusion considerations***
    a. **Hyperkalemia**, **hypocalcemia** (low **iCa**) are common side effects from pRBCs
    b. **Fluid overload** is a potentially dangerous side effect of multiple transfusions
    c. **DO NOT push PLATELETS**. Can cause refractory hypotension.

PRO-COAGULANTS

1. *Amicar (Aminocaproic acid):* binds competitively to plasminogen to inhibit fibrinolysis
    a. Indications: controls hemorrhage in hemophilia, platelet dysfunction, after ECMO or CPB
    b. Dosage: 50-100mg/kg PO/IV Q6hr, 25-30mg/kg/hr continuous infusion
    c. SE: risk of anaphylaxis, hypotension, thrombus, PE
2. *Vitamin K*
    a. Indications: liver disease, nutritional deficiency, prolonged INR
    b. Dosage: 1-5mg IV qday x 3 days to replace if in hepatic dysfunction, 5mg IV x 1 for severe bleeding due to anticoagulation
    c. SE: IV formulations carry a risk of anaphylaxis, especially if administered quickly.
3. *Recombinant Factor VIIa (NovoSeven):* activates the extrinsic pathway of the coagulation cascade
    a. Indications: Initially developed for acute bleeding in hemophilia, potential use in DIC and life-threatening hemorrhagic shock.
    b. Dosage: 35-120mcg/kg/dose, round to full vial size (1.2 or 2.4mg).
    c. SE: Can worsen DIC and potentiate active thrombus formation. ONLY for use with known factor VII deficiency.

ANTI-COAGULANTS

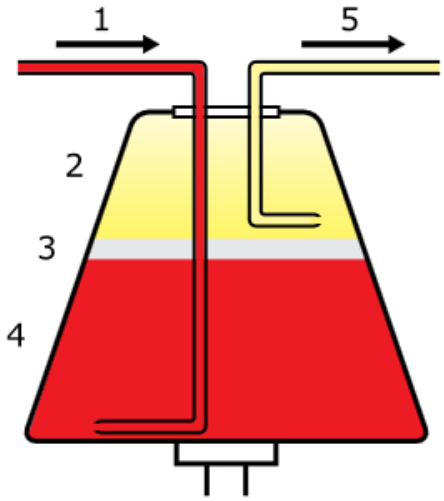
Order set: Pediatric ICU Heparin Anticoagulation

1. *Heparin*: Naturally occurring anticoagulant produced by basophils and mast cells which prevents the formation of clots and extension of existing clots by inhibiting factor Xa. No thrombolysis although it allows the body's natural mechanisms to break down existing clots
    a. Indications: thromboemboli, DVT prophylaxis, ECMO, pro-coagulant state
    b. Dose: load 100units/kg, infuse at 20units/kg/hr, titrate to goal Hep assay of 0.3-0.5
    c. Complications: bleeding, anemia, thrombocytopenia, HIT (send antibody to diagnose)
    d. Reversal:

Time since last Heparin dose	Mg of Protamine to reverse 100 units heparin
<30 mins	1 (max=50mg)
30-60 mins	0.5-0.75
60-120 mins	0.375-0.5
>120 mins	0.25-0.375

2. *Tissue plasminogen activator (TPA, alteplase*): local fibrinolysis by binding to fibrin in the thrombus and converts entrapped plasminogen to plasmin to break down the clot.
3. *Lovenox* is a low molecular weight formulation of heparin that can be injected BID. Dosage: 0.75-1mg/kg SC, titrate to anti-Xa level of 0.1-0.3 for prophylaxis and 0.5-1 for treatment.

APHERESIS



Apheresis is the process of drawing all blood out of a patient (1) and then spinning it in a centrifuge to create separation of the components and removing one part of the blood before returning the remainder to the patient.

The components settle out in layers based on density:

1. Whole blood
2. Plasma: immunoglobulins, cytokines
3. Buffy coat: Leukocytes, platelets
4. Erythrocytes
5. Plasma being removed → TPE.

Indications for each modality:

- **Therapeutic plasma exchange (TPE): removal of the plasma with replacement with albumin or FFP. Accomplishes the removal of all cytokines and antibodies which is helpful in auto-immune pathology.**
  May be used for the following conditions
  - Acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome)
  - ANCA-associated rapidly progressive glomerulonephritis
  - Anti-glomerular basement membrane disease (Goodpasture's syndrome)
  - Chronic inflammatory demyelinating polyradiculoneuropathy
  - Cryoglobulinemia
  - Focal segmental glomerulosclerosis
  - Hemolytic-uremic syndrome; Atypical HUS due to autoantibody to factor H
  - Hyperviscosity in monoclonal gammopathies
  - Liver transplantation, ABO incompatible
  - Myasthenia gravis
  - PANDAS (exacerbation)
  - Paraproteinemic polyneuropathies
  - Renal transplantation, ABO incompatible
  - Sydenham's chorea
  - Thrombotic microangiopathy, drug-associated
  - Thrombotic thrombocytopenic purpura
  - Wilson disease, fulminant
  - Acute disseminated encephalomyelitis
  - Antiphospholipid syndrome, catastrophic
  - Autoimmune hemolytic anemia
  - Familial hypercholesterolemia
  - Hematopoietic cell transplantation, major ABO incompatibility (recipient has anti-A or anti-B)
  - Hemolytic-uremic syndrome
  - Atypical HUS due to complement factor gene mutations
  - Lambert-Eaton myasthenic syndrome
  - Multiple sclerosis
  - Acute CNS inflammatory demyelinating disease
  - Neuromyelitis optica (Devic's syndrome), acute
  - Overdose, venoms, and poisoning
  - Mushroom poisoning
  - Phytanic acid storage disease (Refsum's disease)

- Renal transplantation, ABO incompatible
- Humoral rejection
- Systemic lupus erythematosus, severe (eg, cerebritis, diffuse alveolar hemorrhage)
- Voltage gated potassium channel antibodies

- **RBC Exchange: Remove erythrocytes to replace with donor RBCs. Effective in Hemoglobinopathies.**
  CATEGORY: I (RBC EXCHANGE IS FIRST LINE THERAPY FOR THE FOLLOWING CONDITIONS)
  - Sickle cell anemia and stroke
  - Babesiosis

  CATEGORY: II (RBC EXCHANGE IS SECOND LINE THERAPY FOR THE FOLLOWING CONDITIONS)
  - Malaria, severe
  - Sickle cell disease: Acute chest syndrome, Prophylaxis for primary or secondary stroke
  - Prevention of iron overload from recurrent transfusions
- **Leukopheresis: removal of WBC without replacement.**
  - Hyperleukocytosis

Complications of APHERESIS:

ANY REPLACEMENT FLUID used in the circuit (e.g. albumin, FFP)

1. **Citrate-induced hypocalcemia:** Symptoms of hypocalcemia include perioral and distal extremity paresthesia or numbness. Severe reactions can include tetany, prolongation of the QT interval, arrhythmias, or hypotension
2. **Citrate-induced metabolic alkalosis:** especially seen in patients with renal insufficiency
3. **Inadvertent removal of a medication**
4. **Vascular catheter complications:** infection, thrombosis, perforation, dissecting hematomas, air embolism, or arteriovenous fistulas

NON-PLASMA REPLACEMENT FLUIDS (e.g. albumin)

1. **Hypokalemia:** up to a 25% reduction in serum potassium may be seen
2. **Hypocalcemia**
3. **Coagulation factor depletion**: Immediately after a single plasma volume exchange, the prothrombin time (PT) may increase by 30 percent and the activated partial thromboplastin time (aPTT) may double; these changes tend to return towards normal within four hours. May be more severe and long lasting if multiple treatments in a week
4. **Immunoglobulin depletion:** Baseline IgG levels should be measured. If IgG levels less than 500 mg/dl or if concern for infection IVIG is indicated
5. **Angiotensin converting enzyme (ACE) inhibitor-related complications:** symptoms resembling anaphylaxis may be seen. Recommended that ACE inhibitors be withheld for 24 hours prior to apheresis with albumin

DONOR PLASMA OR RED BLOOD CELL EXPOSURE

1. **Anaphylactic reactions**: Check for IgA deficiency if anaphylaxis occurs
2. **Transfusion-related acute lung injury (TRALI)**
3. **Infectious risks of plasma or RBC**
4. **Mortality:** reported case fatality rate for therapeutic plasma exchange (TPE) is 3 to 5 per 10,000

ENDOCRINE

DIABETIC KETOACIDOSIS (DKA)

Order set: Pediatric ICU Diabetic Ketoacidosis (4143)

Diagnosis: Metabolic acidosis (pH <7.30 arterial blood gas), Hyperglycemia (BG>200), Ketonuria (MOD-LRG ketones on U/A)

Initial Evaluation:

1. *History*: CC and HPI including any ingestions or exposures, family history of any endocrinopathy is very important, identifying a *trigger* is essential if a known diabetic
2. *PE:* VS for Cushing's triad from cerebral edema, **mental status,** degree of dehydration, respiratory pattern, cellulitis around home injection sites
3. *Labs:* D-stick, urinalysis, chemistry with Mg and Phos, iCa, CBC, Hgb_A1_C, pH, [New diagnosis: include insulin level, c-peptide, thyroid studies, celiac screen]

ICU Treatment:

1. **Fluids**: initial fluid bolus should be around 10mL/kg, only give 20mL/kg if in shock
    a. Two bag system: D10 Isolyte with potassium and Isolyte with potassium (as KPhos or KAcetate);

Serum Glucose	Isolyte	D10 Isolyte	Dextrose conc
>300	100%	0	0%
200-249	50%	50%	5%
100-149	0	100%	10%
<100	0	125%	12.5%

    b. If not voiding, do not put any electrolytes (potassium) in the IVF.
2. **Insulin**: continuous infusion to start at 0.1units/kg/hr. Ideally serum glucose ***should not fall by more than 100mg/dL in one hour***. Do not stop the infusion if serum glucose is less than 100 if the patient is still acidotic and making ketones- increase the dextrose in the fluids.
3. **Mannitol**: at any signs of altered mental status, obtain head CT and consider mannitol, 0.5-1gm/kg

1. Once corrected- ensure patient received daily Lantus. Then correct for sugar and turn insulin drip off 1 hour later.
2. Transition d-sticks to before meals, bedtime and at 3am. Call endocrine and transfer to floor.

SODIUM DYSREGULATION

**Never attempt to correct serum sodium by more than 0.5mEq/L/hr- 1mEq/L/hr, especially if asymptomatic or if the sodium abnormality has existed for longer than 48 hours. **

Use the fractional excretion of sodium to help determine if the kidneys are handling sodium appropriately in each clinical setting. **Remember, the FE_{Na} is not clinically relevant if diuretics given in the last 12 hours. >3% in ATN or post-renal obstruction; <1% in pre-renal azotemia or acute GN.

$$FE_{Na} = (U_{Na} \times S_{Cr}) / (U_{Cr} \times S_{Na}) \times 100\%$$

Vasopressin (aka Anti-diuretic hormone or ADH)

- Synthesized in the hypothalamus and stored and released from the posterior pituitary.
- Released in response to increased plasma osmolarity (>285mOsm/L), decreased stretch of the baroreceptors in the Aorta (decreased BP).
- Acts on collecting ducts of the kidney to increase intravascular free water and stimulates the V_2 receptors in the peripheral vasculature to cause vasoconstriction.

↑ADH = ↑BP+↑free water reabsorption in kidney = concentrated urine + ↓ Na.

HYPERNATREMIA

Differential diagnosis: dehydration, DI, iatrogenic, excess Na load

Diabetes insipidus (DI) = deficiency of (*Central*) or lack of response to ADH (*Nephrogenic*)

Diagnosis: Large amounts of dilute urine (6-8ml/kg/hr, urine osmolarity <300 or spec gravity <1.005) and hypernatremia with high serum osmolarity (>305mOsm/L), volume depletion. Loss of free water causes further aldosterone secretion to stimulate more sodium absorption and worsens the hypernatremia.

Causes:

1. *Central DI* (lack of ADH production): Injury to pituitary and/or hypothalamus as seen in CHI, tumors, seizures, meningitis, brain death, CVA or ICH.
2. *Nephrogenic DI* (lack of response to ADH): AKI, genetic defects

Treatment:

1. Calculate free water deficit, give IVF at insensible rate + urine output 1:1. Start with D5 1/2NS- may need D5 ¼ NS- closely monitor Na,
   Free water deficit (mL) = [(Na_{actual}/Na_{goal}) -1] x 1000mL/L x 0.6 mL/kg x wt(kg). Divide by 24-48 hours to calculate replacement by no more than 0.5mEq/hr using enteral water or D5W running with another IVF fluid.
2. Vasopressin drip at 0.5-10milliunits/kg/hr can replace an ADH deficiency in *central DI*, titrate to goal normal urine output.

HYPONATREMIA

Differential diagnosis: SIADH, CSW, iatrogenic, diuretics, heart failure or liver failure, excess free water

Syndrome of Inappropriate ADH (SIADH) = pathologic excess of ADH

Diagnosis: Hyponatremia from free water retention, low urine output, concentrated urine (urine osmolarity > 500mOsm/L, spec gravity > 1.015) and decreased serum osmolarity (<275mOsm/L). Presents with hypervolemia

Causes:

1. CNS infection, TBI or CHI, neurosurgery, brain tumors.
2. Lung pathology: PNA, high PEEP, mechanical ventilation
3. Para-neoplastic syndromes
4. Medication side-effect, adrenal-cortical dysfunction from other causes.

Treatment:

1. Fluid restriction to insensible fluid rate.
2. Sodium replacement with 3% NS or NaCl and a loop diuretic.
   a. **Sodium deficit (mEq) = 0.6mL/kg x wt(kg) x (Na_{goal}-Na_{actual})**
3. 3% replacement in *symptomatic* hyponatremia

Cerebral Salt Wasting (CSW) = inappropriate sodium handling, lose more sodium than free water

Diagnosis: hyponatremia from loss, high urine output and high FE_{Na} in a setting of hypovolemia.

Causes: Neurosurgical procedure, intracranial mass, TBI or CHI with ICH, HIE

Treatment:

1. Aggressive rehydration with 0.9NS and 3% sodium replacement using the above calculation
2. Fludrocortisone can stop polyuria, urine replacement with 3% or NS may be necessary.
3. CSW usually resolves on its own in 4-6weeks after the initial CNS insult.

ADRENAL INSUFFICIENCY (AI)

AI can be either primary – Addison's, panhypopituitarism, or secondary- sepsis, chronic steroid therapy. It is suspected in the presence of unexplained catecholamine-resistant shock or recurrent episodes of hypotension.

Diagnosis: *ACTH stimulation test*: draw baseline cortisol, administer an ACTH analog (Corsitropin 1mcg dose IV), and obtain repeat Cortisol levels at 30 to 60 minutes after to determine adrenal response. If cortisol level is low at baseline and after the stim test (△<9), the patient has adrenal insufficiency.

Treatment: Hydrocortisone supplementation at stress doses: **2mg/kg** as a loading dose & then **2mg/kg/day divided q 6h** , wean to physiologic (one third the stress dose) once condition improving. Repeat stim test when acute illness is resolved.

	Equivalent dose (mg)	Stress dose	Glucocorticoid FX	Mineralocorticoid FX	Half-life
Cortisone	25		0.8	0.8	8-12 hours
Hydrocortisone	20	200-300mg	1	1	8-12 hours
Prednisone	5	50-100mg	4	0.8	18-36 hours
Prednisolone	5		4	0.8	18-36 hours
Methylprednisolone	5	40-80mg	5	0.5	18-36 hours
Dexamethasone	0.75	7.5-30 mg	25	0	36-54 hours

GLUCOSE VARIABILITY

Order Set Pediatric ICU Non-DKA Insulin Infusion (15105)

Hyperglycemia, **hypoglycemia** and **glucose variability** are independently associated with increased mortality in PICU patients. Proposed theories include increased inflammatory cytokine production, acute dyslipidemia, endothelial dysfunction, hypercoagulation, and accelerated cellular apoptosis. Glucose variability increases production of reactive oxygen species leading to cell damage and death. High risk pts for glucose variability: Post-arrest, Exogenous steroids, Post-trauma or surgery, Septic patients, actively cooling or rewarming.

Definitions/Treatments:

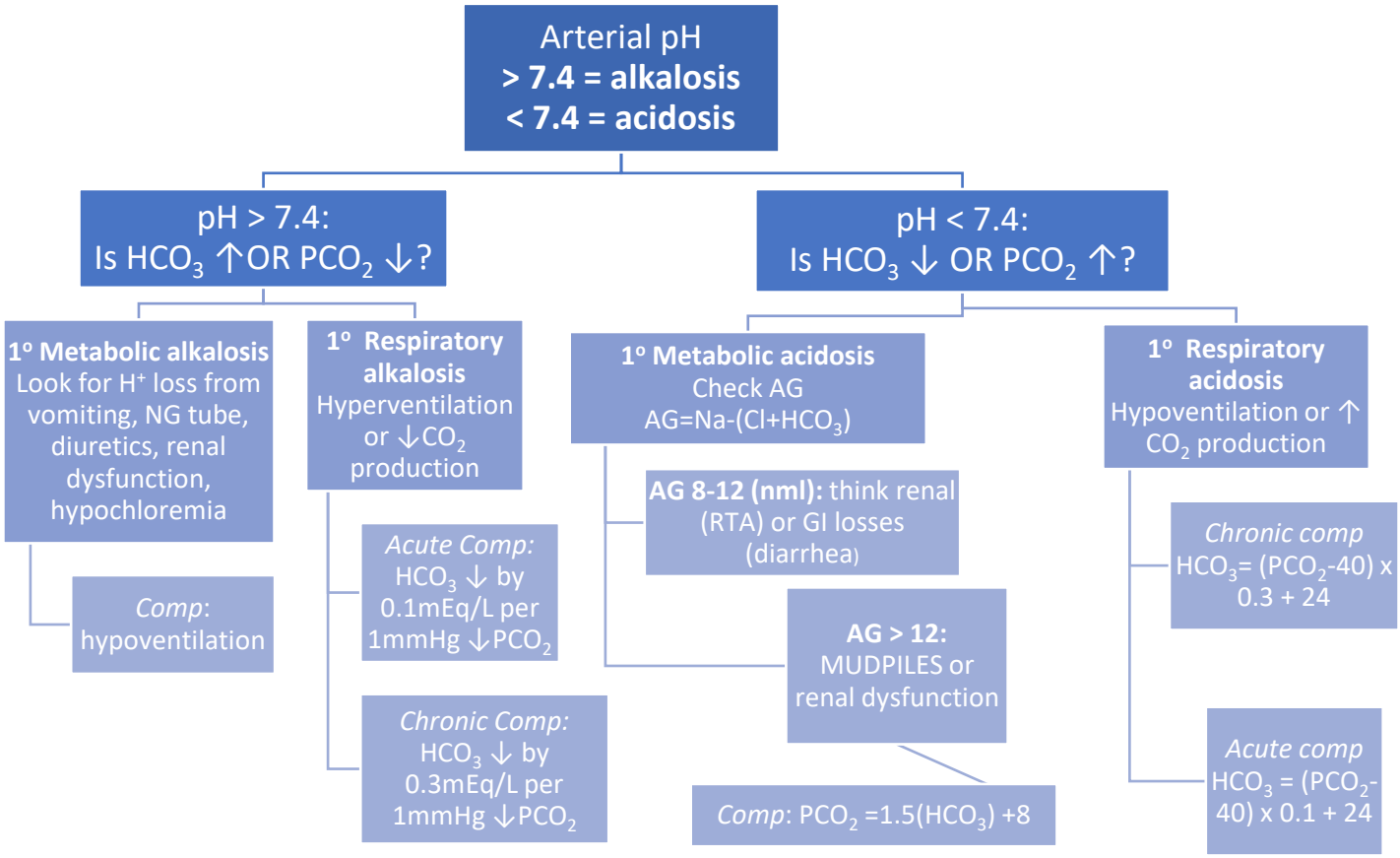
- Hyperglycemia = mild glucose >120, moderate >150mg/dL, severe > 200mg/dL.
  - Treat to keep serum glucose- 120-180 using an insulin infusion starting at 0.05-0.1units/kg/hr and titrate by 0.1units/kg/hr to avoid any hypoglycemia.
- Hypoglycemia = glucose < 60, in neonates <35 in the first 24 hours and less than 50 up to 28 days.
  - Treat with "Rule of 50" = %Dextrose x volume = 50.
  - D5 give 10ml/kg, D10 give 5ml/kg, D25 give 2ml/kg*, D50 give 1ml/kg*.

  *Through central venous line.

ACID-BASE BALANCE

$$pH = pK + \log[HCO_3]/0.03 \times PCO_2$$

Approach to reading a blood gas:



For every **10mmHg ΔPCO2 = pH Δ0.08** For every **10mEq ΔHCO₃ = pH Δ0.15**

IMPORTANT ELECTROLYTE CHANGES IN ACID/BASE DYSRUPTION:

2) *Potassium*: serum level affected by H^+-K^+ transporter
   a) Alkalosis = **l**ow serum **K^+** because shifted into cells, total body K^+ is the same
   b) Acidosis = high serum potassium, if prolonged the kidneys will compensate by excreting K^+
3) *Calcium*: ionized component (unbound) affected by H^+ concentration as it reacts to albumin
   a) Acidosis = ↑ iCa because H^+ competes for calcium binding sites
   b) Alkalosis = ↓iCa

BUFFERS

IN ACIDOSIS:

1. Sodium Bicarbonate: Dose 1mEq/kg IV; *must* have ability to ventilate as HCO_3 converts to PCO_2 which is then exhaled decreasing total body proton load, can cause hypernatremia+↓CSF pH.

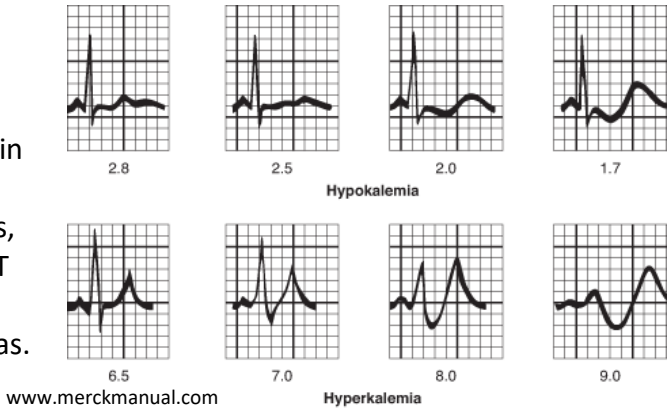
IN ALKALOSIS:

1. Chloride replacement if Cl < 90: use NaCl or KCl, **Cl deficit = 0.3 x wt x (100-present Cl)**
2. Ammonium Chloride: 1mEq/kg/dose IV Q6hr x 4 doses at max rate of 1mEq/kg/hr
3. Hydrochloric acid: via CVL, **H^+ deficit = 0.5 x wt x ($HCO_{3current}$-$HCO_{3\ goal}$)**, 0.05-0.1mEq/kg/hr.
4. Diamox 5mg/kg/dose q6hr, promotes renal excretion of HCO_3. NOTE: weak diuretic effect = ↓ K.

RENAL

HYPERKALEMIA

Definition: Serum K > 6mEq/L is life threatening because the intracellular potassium gradient is pivotal to the regulation of cardiac action potentials and conduction.
Causes: acute acidosis, renal failure, tumor lysis, massive blood transfusions, iatrogenic



www.merckmanual.com

Treatment:

1. Send confirmatory testing from a free-flowing serum sample (from central line or arterial puncture), place on cardiac monitoring and obtain EKG.
    a. EKG changes: initially peaked T-waves, then widening of the QRS complex, ST depression and sinusoidal pattern preceding fatal ventricular arrhythmias.
2. Stop all sources of potassium: IVF, blood transfusions, enteral feeds.
3. Call Nephrology to alert about emergency hemodialysis and immediately order the following:
    a. Calcium: stabilizes the cardiac myocyte membrane, avoids arrest or v-fib.
        i. Through CVL preferably, calcium gluconate can be given peripherally but slowly and must have an intact hepatic metabolism to generate active form.
        ii. Calcium gluconate: 100mg/kg IV over 15-20minutes.
        iii. Calcium chloride: 10-20mg/kg IV over 5 minutes.
    b. Glucose + Insulin: 0.1units/kg IV regular insulin + 0.5-1gm dextrose over 15-30 minutes
        i. Even if hyperglycemic, give glucose with insulin to avoid any hypoglycemia.
    c. Albuterol: continuous nebulized treatment will shift some potassium into the cells.
    d. Kayexalate: 1gm/kg PO (give through OG or NG) or PR
        i. Avoid in any patients with risk of bowel ischemia
    e. Sodium bicarbonate: 1mEq/kg IV over 5 minutes
        i. Must have control of ventilation because will convert into PCO_2 and **worsen acidosis and hyperkalemia** if already not ventilating well.
4. Establish venous access for emergency hemodialysis.

Acute Kidney Injury (AKI) and Acute Renal Failure (ARF)

Definitions:
AKI: increase in serum creatinine by 50% or CrCl decrease by 50%.
ARF: inability to perform renal functions including acid/base balance, fluid balance, electrolyte regulation, excretion of waste (can be manifested as polyuria, oliguria, or anuria) regardless of SCr.
Diagnosis: azotemia (increased BUN and SCr) and oliguria 0.5ml/kg/hr or anuria or fluid overload.
Remember there is a 24-48 hour delay between occurrence of AKI and increase in SCr.

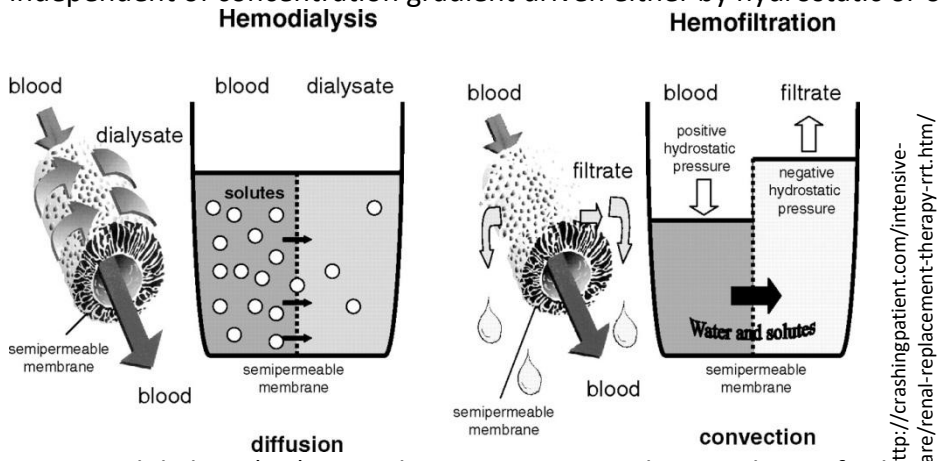
pRIFLE	R– "Risk"	eCrCl* decreases by ≥ 25%	<0.5ml/kg/hr x 8 hours
	I—"Injury"	eCrCl* decreases by ≥ 50%	<0.5ml/kg/hr x 16 hours
	F—"Failure"	eCrCl* decreases by ≥ 75% or eCrCl*<35ml/min/1.73m^2	<0.3ml/kg/hr x 24 hours OR No urine output for 12 hours
	L– "Loss"	Failure > 4weeks	
	E—"End stage"	Failure >3months	*Can replace eCrCl with SCr if unavailable*

Management:

1. **FEN**: Limit fluid intake (insensible Losses= 300-400/m^2/d or about ⅓MIVF if don't know BSA)
   a. If taking PO: avoid free water, potassium containing fluids or high protein-load
   b. If giving TPN: avoid any potassium and minimize protein to < 2gm
2. **Labs**: Chemistry, iCa, Mg, Phos, CBC with diff, T&C, UA, urine protein: creatinine ratio (proteinuria)
   a. If transfusing, *wash* the pRBCs to avoid high potassium load
3. **Renal replacement therapy (RRT)**: indicated for toxic ingestion, severe electrolyte imbalance not amenable to medical therapy, symptomatic uremia (BUN 80-100), fluid overload >20%

   %FO = [(Fluid in (L) – Fluid out (L))/admission weight] x 100%.

   RRT performs the exchange of solute and water between two solutions (blood to dialysate) across a membrane based on:
   a. Hemodialysis uses **Diffusion** = solute exchange across a membrane between two solutions based on concentration gradient, permeability of the membrane and surface area of the membrane
   b. Hemofiltration uses **Convection** = solute movement or "drag" with filtration across a membrane independent of concentration gradient driven either by hydrostatic or osmotic pressures



   c. Peritoneal dialysis (PD): uses the peritoneum as the membrane for both convection and diffusion solute clearance. Need healthy, intact peritoneum (Contraindications: diaphragmatic hernias, adhesions, previous surgeries, or active peritonitis)
      i. *Complications*: hernias, peritonitis, increased risk of any infection because of clearance of immunoglobulins
   d. Intermittent hemodialysis (HD): convection and diffusion to clear fluids and electrolytes from the serum, can set certain amount of fluid to remove, run for 3-4-hour sessions; good for hyperkalemia, ingestions, drug toxicity, tumor lysis, hyperammonemia.
   e. Continuous renal replacement therapy (CRRT): through a temporary vascular catheter, intended to run 24 hours a day to provide slow, gentle removal of fluid and waste over time; more precise in reaching solute clearance and UF goals than PD. To be used in hemodynamically unstable patients.
   f. ***Complications from RRT*:** hemodynamic instability (especially upon initiation), disequilibrium syndrome (if urea cleared from blood too quickly = acute cerebral edema and death), hypothermia from the CRRT circuit, air embolism, anaphylaxis, bradykinin release syndrome (reaction of acidic blood with the biocompatible membrane causes a massive bradykinin release within 10-15 minutes = profound hypotension)

To assess if medication doses must be adjusted when on CRRT, go to the **Dialysis of Drugs Handbook**: http://renalpharmacyconsultants.com/sitebuildercontent/sitebuilderfiles/2012dialysisofdrugsbooklet.pdf

HYPERTENSIVE EMERGENCY

Definition: severely elevated BP in the setting of end-organ damage, i.e. seizure, visual changes, oliguria, MI.

Causes: high renin states or increased sympathetic tone, pain, seizures, elevated ICP, renal vascular disease, ingestion, coarctation of the aorta, PRES (chemotherapy), exogenous steroids
Treatment: ↓BPs **by 20-25%** acutely to control symptoms and then ↓to ***baseline*** within 24-48hours.

Med	Mechanism	Dose	Primary Indication	Function on SVR + CO	Function on CBF
Sodium Nitro-prusside	Metabolized into nitric oxide = direct vaso- and veno-dilation	0.3 to 8 mcg/kg/min (with sodium thiosulfate)	Hypertensive emergency without elevated ICP	Decreases preload and afterload by dropping SVR	Dilates cerebral arteries ↑CBF + ICP
Esmolol	Cardio-selective beta-1 receptor antagonist	100 to 500 mcg/kg/min, load 0.3 to 0.5mg/kg	Aortic Dissection (with Nicardipine)	Reduces CO by ↓heart rate and stroke volume	Little to no effect on CBF
Labetalol	Selective alpha-1 and non-selective beta-antagonist	0.25 to 3 mg/kg/hr; load 0.2 to 1mg/kg	Aortic Dissection, ICH or CVA	↓SVR without ↓CO, no tachycardia	Little to no effect on CBF
Nicard-ipine	2nd generation Ca-channel blocker acting on smooth muscle in all blood vessels and the myocardium	0.5 to 5 mcg/kg/min	Hypertensive encephalopathy, Aortic dissection (with esmolol), Hypertensive emergency ARF, CVA, Sympathetic crisis (with benzo)	Arterial and venous dilation, ↓cardiac contractility and heart rate by slowing AV node conduction	Dilates cerebral arteries ↑CBF+ ICP if mean arterial pressure is still ↑

HYPERTENSIVE *URGENCY:*

Definition: severely elevated BP without organ dysfunction- asymptomatic

- **Hydralazine:** arterial vasodilator, 0.1-1mg/kg IV/PO Q6hr, lasts 4-6h
- **Enalapril:** ACE-Inhibitor, 0.04-0.1mg/kg/d Q12-24hours, lasts 12-24h
- **Amlodipine:** Ca-channel blocker, 2.5-5mg PO Qday, 1/2life 30-50h, peak at 6-12h

	Class	Drugs	Mechanism	SE/Notes
DIURETICS	**Osmotic diuretics**	Mannitol, glycerol, urea	Freely filtered to prevent H_2O reabsorption by the proximal tubule	Works for acute management of elevated ICP or IOP
	Loop diuretics	Furosemide (0.5-1mg/kg q6), Bumetadine (0.02mg/kg q6), Torsemide (0.5mg/kg qd), Ethacrynic Acid (0.5-1mg/kg BID)	Inhibit Na/K/2Cl transport in the thick ascending loop	Hypercalciuria (potential stones), ototoxicity and ↓K
	Thiazides	Hydrochlorothiazide (1-1.5mg/kg BID), chlorothiazide (10-20mg/kg PO BID)	Inhibit NaCl cotransport in early distal convoluted tubule	Used for nephrogenic DI and hypercalciuria, Weaker than loops.
	K^+ sparing diuretics	Sprinoloactone (1.5-3.5mg/kg qd); Amiloride and Triamterene	Blocks aldosterone action on the cortical collecting ducts; A/T inhibit Na/K pump	Weak diuretics alone
	Carbonic anhydrase (CA) Inhib.	Acetazolamide (5mg/kg Q8), metolazone (0.1-0.2mg/kg q12-24), dichlophenamide	Inhibit CA in the luminal membrane of the proximal tubular = reduced HCO_3 reabsorption.	Also used in glaucoma, altitude sickness and as adjunctive anti-epileptic.

IVF COMPOSITION

Serum osmolality = (2xNa) + (BUN/2.8) + (glucose/18) **NORMAL SERUM OSM 285-295mOsm/L.**

	Sodium	Chloride	Bicarbonate	mEq/L
0.9%* NS	154mEq/L	154 mEq/L	--	308
3% NS	513mEq/L	513mEq/L	--	1026
D5% W	--	--	--	252
D5% 0.2%NS	34	34	--	320
D5% 0.45% NS	77	77	--	406
D5% 0.9%NS	154mEq/L	154mEq/L	--	514
LR	130	109	28 (Potassium 4)	273
Plasmalyte	140	98	27 (Potassium 5)	294

*Percentage amount = grams per 100mL. For example, 0.9%=0.9gm of NaCl per 100mL of water.

ELECTROLYTE DERANGEMENTS

Abnormality	Differential Dx	Symptoms	Treatment
Hyponatremia (Na<130)	SIADH, CSW, Free water intox, iatrogenic, CAH, diuretics, hepatic or cardiac failure	Lethargy, seizures, coma from cerebral edema	3%NS in mL (if symptomatic) = 0.6*wt*(Na_{goal}-Na_{actual})
Hypernatremia (Na>145)	DI, dehydration, iatrogenic, free water loss from skin, drugs	Seizures, renal failure, lethargy and coma	Free water deficit (mL)= [(Na_{actual}/Na_{goal}) - 1]*1000*0.6*wt
Hypocalcemia (iCa<1.20)	Hypoparathyroidism, multiple pRBC tx, diet, alkalosis, CRRT, Lasix, malabsorption, hyperphosphatemia, hyperlipidemia	Paresthesias, bronchospasm, apnea, seizures, prolonged QT, Rickets, Chvostek sign (facial mm spasm), Trousseau sign (carpopedal spasm)	CaCl (via CVL) 10-20mg/kg IV Calcium gluconate (PIV) 100-200mg/kg IV
Hypercalcemia (iCa>1.4)	Iatrogenic, dietary intake, increased renal absorption or bone destruction, malignancy, Williams syndrome, salicylate ingestion, familial, acidosis	Poor feeding, emesis, FTT, confusion, psychosis, weakness, short QT, renal failure, Nephrogenic DI, Calcinosis	Hydration (IVF at 2-3xmaint), low dose loop diuretics; Calcitonin and bisphosphate; if severe CRRT
Hypokalemia (K<3.5)	Diet, medications, alkalosis, diarrhea, Adrenal-cortical excess, Bartter syndrome	Arrhythmias (PACs, PVCs), mild muscle weakness	KCl 0.5-1mEq/kg IV or PO *KPhos is poor K supplier*
Hyperkalemia (K>5.0)	Renal failure, acute acidosis, tumor lysis, rhabdomyolysis, pRBCs tx, medications, adrenal insufficiency	Fatal arrhythmias, fatigue, mm weakness, disorientation, palpitations, paresthesias	CaCl, Lasix, kayexalate, $NaHCO_3$, insulin/glucose, albuterol, +/-CRRT
Hypomagnesemia (Mg<2)	Diarrhea, iatrogenic, diet, insulin for DKA, massive RBC tx, CPB, burns	Symptoms usu from resultant ↓Ca: mm weakness, tetany, seizures, hypokalemia	$MgSO_4$ 25-50mg/kg IV or MgOxide PO
Hypermagnesemia (Mg>4)	Diet, iatrogenic, massive cellular release	Mm weakness, resp depression, prolonged QT and PR	Hydration and loop diuretic if causing arrhythmias, CRRT if in renal failure
Hypophosphatemia (Phos<1.5)	↓intake or absorption, ↑bone formation, re-feeding syndrome	ATP depletion = hemolysis, WBC failure, plat dysfunction, mm atrophy and weakness, respiratory failure, AKI, AMS	KPhos 0.16-0.24mmol/kg IV q6hr or NaPhos 0.16-0.24mmol/kg IV q6hr
Hyperphosphatemia (Phos>9.5)	↑intake or ↓renal excretion	Refractory hypocalcemia	Hydration, stop Phos sources; Phos binders

NUTRITION

PHASES of CRITICAL ILLNESS	Duration	Systemic Characteristics	Metabolism
Acute	6-8hr	Fever, hypoglycemia, ↑HR	Growth is inhibited, energy diverted to stress response; pre-albumin levels begin to decline.
Ebb	Varies	↑catecholamines, cortisol and glucagon = ↑glucose, ↑GH, cytokine production = gluconeogenesis, ↑TGs, ↑ADH	Limit calories at this phase as excess nutrients can be deleterious; weight gain is not from growth but fluid retention.
Flow – Catabolic	Varies	Endogenous catabolism of fat, carbs, and protein stores = inflammation, ↑glucose, glucose intolerance, negative nitrogen balance, ↑CRP	Nutrient provision should remain toward basal metabolic needs, provide extra protein to protect stores of albumin and pre-albumin.
Flow-Anabolic	Varies	Restoration of tissue composition, positive nitrogen balance	Increase nutrient provision to promote repletion, growth will resume.

Total energy expenditure (TEE) = basal metabolic rate + (energy for thermogenesis + activity + growth + healing)

Resting energy expenditure: **REE** = **basal metabolic rate + energy for thermogenesis**

REE needs during the **Acute** and **Ebb** phases:

Age	REE (kcal/kg/day)
0-36 months	55
4-6 years	45
7-10 years	40
11-18 years	25

In healing/active phase: estimated caloric needs are around 80-100kcal/kg/day.

Initiating nutrition within **72** hours of PICU admission has shown to lower mortality.
Start nutrition as soon as it is safe to do it from a respiratory/GI/cardiac standpoint.

Enteral feeds: the gut is ***always*** the preferred mode of nutrition.

- Can feed into the stomach with an NG/OG or across the pylorus into the duodenum with an ND/OD to avoid aspiration from reflux if patient is at high risk.
- If feeding through an ND, bolus feeds are **not tolerated**.
- Bolus feeds into a NG are preferred since they are more physiologic.

Parenteral feeds aka Total parenteral nutrition (TPN): expensive, risk of infection from the TPN/IL itself and from the central line, need a CVL to optimize calories (through dextrose) and protein delivery.

- Dextrose = 3.4kcal/gm (gm=%dextrose x volume); AA = 2-3kcal/gm.
- Fat as intralipids = 10kcal/gm or 2kcal/ml
- Check serial LFTs and TG to monitor for TPN cholestasis or hypertriglyceridemia.
- *Peripheral parenteral nutrition- PPN-* limits the amount of dextrose that can be given but can be given via a peripheral IV.

INFECTIOUS DISEASES

BACTERIA by GRAM STAIN

Order Set: Pediatric Sepsis (13712)

GRAM POSITIVE	GRAM NEGATIVE	FUNGAL PATHOGENS
Streptococci group A, B, C, G (C)	*Neisseria* (C)	***Candida***
Staphylococcus (C)	*Haemophilus influenza* (CB)	***Aspergillus***
*Bacillus** (B)	*Escherica coli* (B, L+)	***Cryptococcus***
Enterococcus (C)	*Klebsiella* (B, L+)	***Histoplasma***
Corynebacterium (B)	*Salmonella* (B, L-)	***Pneumocystis carinii***
*Actinomyces** (B)	*Shigella* (B, L-)	
*Clostridium**(B)	*Proteus mirabilus* (B, L-)	
Listeria (B)	*Enterobacter* (B, L+)	
Nocardia (C)	*Serratia* (B, L+)	
*Peptostreptococcus** (C)	*Pseudomonas* (B, L-)	***Addt'l GRAM –'s:***
	Bordetella (CB)	*Helicobacter pylori* (B, L-)
***Mycoplasma* and *Ureaplasma* = NO CELL WALL, not G+ or G-**	*Legionella pneumophila* (CB)	*Yersinia pestis* (B, L-)
	Chlamydia trachomatis	*Campylobacter jejuni* (B, L-)
B = bacilli	*Citrobacter* (B, L+)	*Moraxella catarrhalis*
C = cocci	*Vibrio cholera* (B, L-)	*Acinetobacter*
CB = coccobacilli	*Bacteroides fragilis** (B, L-)	*Franciscella tularensis* (CB)
L+ = lactose fermenter	*Stenotrophomonas*	*Pasturella multocida* (CB)
***Anaerobic**	*Fusobacterium**	*Brucella* (CB)

COMMON PICU INFECTIONS

Infection	Likely Pathogens	Antibiotic Choice	NOTES
Meningitis	<u>≤1mo</u>: GBS, enteric bacilli (*E. coli, Klebsiella, Proteus*), *Listeria monocytogenes* <u>1-3mo</u>: same + *Strep pneumo, Neisseria meningitides, Hib*	<u>≤1m:</u> ampicillin & gentamicin <u>1-3m:</u> Ceftazidime/cefotaxime & vancomycin X 14days, re-LP to see if clears and determine stoppage vs. 21 d	
	<u>3mo-5yo</u>: *S. pneumo, N. meningitides, Hib* <u>>5yo</u>: *S. pneumo, N. mening*	<u>>3mo:</u> ceftriaxone/vancomycin <u>Complicated:</u> meropenem	
	<u>CSF shunt:</u> *CONS, Staph aureus,* enterics	<u>Shunt</u>: ceftazidime & gent & vancomycin	
Pneumonia	<u>CAP</u>: (Lobar) *S. pneumonia (#1 cause), H. influenza* (Lobar with effusion) *S. pneumo*, MSSA, MRSA or GAS (Atypical) *C. trachomatis, B. pertussis*; >5y *M. pneumonia, C. pneumoniae*	<u>CAP:</u> Ampicillin, ceftriaxone <u>With effusion:</u> Clindamycin + ceftriaxone <u>Atypical:</u> azithromycin Uncomplicated= 7-10 days Complicated = 2-4 weeks	Atypical pneumonia is unusual between infancy and 4 years of age unless there are school age children at home <u>VAP:</u> *GPB, Staph epi* and *enterococci* are contaminates; <u>not</u> pulmonary pathogens
	<u>Nosocomial (VAP):</u> *GNB, Staph aureus, H. influenzae*	<u>Nosocomial:</u> ceftriaxone or ampicillin/sulbactam <u>If high MDR</u>: piperacillin/tazobactam	
	<u>Aspiration:</u> *Bacteroides, fusobacterium, alpha-heme strep, E. Coli, Klebsiella*	<u>Aspiration:</u> ampicillin/sulbactam or Clindamycin; piperacillin/tazobactam or Vancomycin if nosocomial	
Spont. bacterial peritonitis	*E. coli, Klebsiella, Staph, Strep*	<u>1st line</u>: 3rd gen cephalosporin +/- Metronidazole <u>Complicated:</u> meropenem	Diagnose with ascites fluid, treat x 5 days and reassess, may need repeat ascitic tap
UTI/pyelo-nephritis	<u>GNeg:</u> *E. coli (80%), Proteus, Klebsiella, Enterobacter, Citrobacter;* <u>GPos:</u> *Staph saphro, Enterococcus, Staph aureus*	3rd gen cephalosporin + amoxicillin or ampicillin if have instrumentation in place No fever: 3-5d, Fever: 10d	UTI by Clean catch if >100,000 CFU; and by cath if>10,000 CFU
Life-threatening gastro-enteritis	HUS *(E.coli);* Toxic megacolon *(C. diff, Shigella);* Amebiasis; **Majority will be viral**	***NO antibiotics*** *unless specific bacterial pathogen found* <u>*C.diff*</u>: PO vancomycin or metronidazole in MOD or SEVERE disease (metronidazole can be IV)	
Osteo-myelitis	*Staph aureus* (>50%), Grp A and B *Strep*, *E. coli* <u>Rare:</u> *Kingella kingae, Bartonella, Brucella, TB, Salmonella* (SSDz)	<u>Neonates:</u> cefotaxime & vancomycin <u>Infants and older:</u> oxacillin, clindamycin or vancomycin (if>10% CA-MRSA), linezolid if fails <u>3-36m:</u> cefazolin (*K. kingae)*	Duration of IV therapy depends on response in first 5-10 days: follow ESR, WBC, fever and CRP. May need surgical intervention.
Cellulitis, Skin Abscess	*Staph aureus*, Beta-hemolytic Strep*, gram negatives in rare cases	TMP-SMX OR clindamycin, Vancomycin, OR cefotaxime + metronidazole for 5-10days <u>Complicated:</u> linezolid, meropenem, vancomycin	Drainage alone may be therapeutic

ANTIBIOTICS

Blue=gram negative coverage; Pink=gram positive; Purple= gram positive & negative; Gray=anaerobes

Name	Class	Cidal?	Dosing	Gram +	Gram -	Anaer-obes	Side Effects
Amikacin	Amino-glycoside	Y	5-7.5mg/kg IV Q8hr (Peak goal 30-40)	0	++++	0	Oto- and nephro-toxic, poor CNS penetration, ↓Ca
Ampicillin	Amino-PCN, Beta-lactam	Y, if conc 3-4xMIC	150-250 mg/kg/d IV/PO Q6hr, 400mg/kg/day for GBS meningitis	VRE, +++	++ *H. flu, E.coli Proteus*	*+Actino*	Anaphylaxis, rash, C. diff, reversible neutropenia
Cefazolin	1st gen cephalos	Y	25-100mg/kg/d IV/IM Q6-8hr	+, No MRSA	++, No *Enterobac*	0	SJS, anemia, anaphylaxis
Azithro-mycin	Macrolide	**No**	10mg/kg Qd→ 5mg/kg Qd x 4d(IV/PO); 10mg/kg/d for pertussis <6mo	++, No MRSA	++, GNB resistant	0	GI, anaphylaxis, C. diff
Aztreonam	Monocyclic Beta-lactam	Y	30mg/kg IV/IM Q8hr	0	++++ *Pseudo+*	0	TEN, cross-rxn with PCN allergy, C. diff
TMP-SMX (Bactrim)	Sulfon-amide	?	5mg/kg PO/IV BID	+MRSA	++++	0	SJS, BM Suppress, liver injury
Cefepime	4th gen cephalo	Y	50mg/kg IV Q12, Q8hr for pseudomonas or CF	++++	++++, *Pseudo+*	0	Anaphylaxis, C. diff, renal insuff, +Coombs
Cefotaxime	3rd gen cephalo	Y	50mg/kg IV Q6hr Crosses BBB	+++	++, no *Entero* or *Serratia*	0	Anaphylaxis, C. diff, renal insuff
Ceftazidime	3rd gen cephalo	Y	30-50mg/kg IV/IM Q8hr	++	++, no *Entero* or *Serratia*, *+Pseudo*	0	Anaphylaxis, C. diff, renal insuff, seizures
Ceftriaxone	3rd gen cephalo	Y	50mg/kg Qd, mening 100mg/kg IV/IM Qd (ADLT 2gm IV q12)	++	++, no *Enterobac* or *Serratia*	0	Anaphylaxis, Biliary sludging (neonates)
Cipro-floxacin	Flouro-quinolone	Y	6-10mg/kg Q8hr IV, 10mg/kg PO Q12hr; chelates with cont. feeds	0	++++, infectious diarrhea	0	↑QTc, ↑levels of other meds, Allergy, ↓glu
Clindamycin	Linco-samide	**No**	40mg/kg/d÷Q8hr IV/PO *Adjunct for TSS*	++, MRSA	Aerobic Resist.	++++, *PCP*	SJS, hypotension, C. diff
Metronida-zole (Flagyl)	Nitro-imidazole	**No**	30mg/kg/d IV Q6h, available PO (good CNS penetration)	0	0	++++ (*C. diff* give ent	SJS, peripheral neuropathy, disulfuram rxn

						eral)	
Gentamicin	Amino-glycoside	Y	<5yo 2.5mg/kg IV Q8h >5yo 2-2.5mg/kg Q8h	+MSSA	++	0	Ototoxic, ↓Ca, nephrotoxic
Linezolid	Oxazo-lidinone	**No**	<12y 10mg/kg IV/PO Q8hr >12y 600mg IV/PO Q12	+++VRE +MRSA	0	Some *C. diff* & *act-inomyces*	Fungal infxn, pancreatitis, optic neuritis, periph neuropathy
Meropenem	Carba-penem	Y	20mg/kg IV Q8hr, 40mg/kg/dose Q8hr (max 2gm) IV for menin	++++	++++ *Pseudo+*	++ ++	Anaphylaxis, ↓K, sz, renal insuff; **AVOID with valproic acid**
Cefdinir	3rd gen. cephalosporin	Y	14mg/kg/day either Q12 or Q24hr PO (max 600mg Qday)	++, ↑ resistant *Strep*	++, no *Pseudo*	0	Red stool, C. diff, agranulocytosis, allergy, SJS
Oxacillin	PCNase-R-beta-lactam	Y, if 4xMIC	100-150mg/kg/d IV Q6hr	+++	0	0	Anaphylaxis, hepatotox, ↑LFTs, neutropen
Penicillin	Beta-lactam	Y	PCN G 25-50,000 units/kg IM; neonatal GBS mening 100,000units/kg Q6h	++++	+*Neisseria*	+++, Syphilis+	Anaphylaxis, sz neurotoxicity
Rifampin	Rifamycin	Y	20mg/kg/day IV Q12hr (penetrates CSF)	MRSA, TB	0	0	Flu-like, suppress BM, ↑bili
Tobramycin	Amino-glycoside	Y	2.5mg/kg IV q8hr; 300mg neb Q12h	Some *Staph*	+++	0	Ototoxicity, nephrotox, vertigo
Ampicillin-Sulbactam (Unasyn)	PCN+beta-lactamase Inhibitor	Y	50-100mg/kg IV q6hr	+++	+++, NO *Pseudo, Serratia, Entero*	+++	Anaphylaxis, C. diff, rash (with EBV infxn)
Vanco-mycin	Glyco-peptide	Y **Static a/g enterococci**	40mg/kg/day div q6, CNS 60mg/kg/day [Goal peak 25-40, trough <10: ↑by 5mg/kg/dose	++++, MRSA	0	*C. diff* (enteral form only)	Nephrotoxic, poor CSF penetration, SJS, anaphylaxis, pancytopenia
Pipericillin Tazobactam (Zosyn)	PCN+beta-lactamase Inhibitor	Y	<6mo 150-300mg/kg/d IV q6-8h, >6mo 300-400mg/kg/d IV	++++, No MRSA	++++, NO *Pseudo, Serratia, Entero*	*Bacteroides*	Anaphylaxis, C. diff, bleeding, AKI

SEDATION, ANALGESIA, & NEUROMUSCULAR BLOCKADE

Order Set: Pediatric ICU Sedation, Analgesia, Delirium, Withdrawal (15093)

Doses are provided in the next table

Sedative or Hypnotic	SE	Duration	Reversal	Notes
Ativan	↓RR, ↓BP, paradoxical rxn	4h	Flumazenil 0.01 mg/kg	FLUMAZENIL = ↓Sz threshold
Etomidate*	↓ICP,↓sz, Adrenal suppression	3-5m	—	Intubation med TBI Can cause adrenal insufficiency
Fentanyl*	↓HR,↓BP, rapid tolerance, chest rigidity if pushed	15m	Naloxone 0.1mg/kg Drip 0.1-2mg/kg/hr	Intubation if ⬇BP, hepatic metabolism
Ketamine*	↑ICP, ↑HR, ↑BP,↓CO, hallucinations, bronchodilation, salivation	15-30m	—	Asthma = atropine for salivation & benzo for hallucinations Physician must give
Morphine*	Urticaria,↓BP, HR & RR, constipation, urinary retent	60m	Naloxone 0.1mg/kg	Histamine release = worse bronchospasm and ↓BP
Pentobarbital	Apnea, ↓BP	50-75m	—	Barbiturate coma for sz
Dexmedetomidine (Precedex)	↓↓HR, ↓BP, hyperglycemia	Mins	—	
Propofol	↓BP, Metabolic syndrome, ↓ICP,↓sz	Secs	—	Physician to give
Remifentanil	↓HR,↓BP, constipation, IWS	Ultra-short	Naloxone 0.1mg/kg	1mcg/kg/hr F = 0.1mcg/kg/min R
Sufentanil	↓HR,↓BP, constipation, withdrawal	30m	Naloxone 0.1mg/kg	Replaces high doses of fentanyl 5mcg/kg/hr F = 1mcg/kg/hr SuF
Versed*	↓RR, ↓BP	60m	Flumazenil 0.01 mg/kg	Can give IM or intranasal

Paralytic**	SE**	Class	Duration	Reversal	Notes
Cisatracurium	Histamine release, ↑HR	Non-depolarizing	25m	Neostigmine 70mcg/kg	↑effect in ↓temps,
Rocuronium*	↑HR	Non-depolarizing	Onset 30s, Dur 1h	Neostigmine 70mcg/kg	Vagolytic
Vecuronium	Histamine release, ↑HR	Non-depolarizing	60m	Neostigmine 70mcg/kg	Accumulates over days
Pancuronium	Histamine release, ↑HR	Non-depolarizing	60-120m	Neostigmine 70mcg/kg	Renal and hepatic clearance, vagolytic
Succinylcholine*	Dysrhythmias, ↓HR, ↑K, MHS, ↑ICP, fasiculat.	Depolarizing	Ultra-short (5-8m)	Neostigmine 70mcg/kg	**Atropine** to avoid ⬇HR, causes Vfib in DMD (rhabdo & hyperkalemia). Avoid in neonates

***Medications used for intubation.**

****Paralytic use for prolonged periods of time can lead to fluid retention (lymph stasis), myopathies and neuropathies→ it is recommended to assess the optimal dose of paralytic needed to achieve effect using train-of-four testing and give "holidays" as often as daily to limit the damage to peripheral nerves.**

COMMON PICU MEDICATIONS (in alphabetical order)

Medication	Dose	Special Instructions or Notes
Acetazolamide (Diamox)	20 mg/kg/day IV/PO divided q6-12h	Max 1g/day, use x 24 hours only.
Adenosine	100 mcg/kg (0.1mg/kg)	May double and repeat in 1-2 mins. Max 6mg
Albumin (25%)	1 gm/kg/dose IV; equals 4cc/kg	
Albumin (5%)	1 gm/kg/dose IV; equals 20cc/kg	Contraindicated as IVFB in TBI
Albuterol	Continuous aerosolized 10-20 mg/hr	↑ by 5-10 mg/hr to max of 40mg/hr
Alprostadil	0.01-0.05 mcg/kg/min	
Alteplase (TPA)	0.5mg/kg/hr	6 hours maximum
Aminocaproic acid	100 mg/kg load, 30mg/kg/hr	
Aminophylline	6mg/kg IV load; maintenance 1mg/kg/hr	Theophylline level Q4hr, target 15-20mcg/ml. Bolus 1mg/kg to raise level by 2mcg/ml
Amiodarone	5mg/kg **OVER 20-60 minutes;** Infusion: 0.4-0.6 mg/kg/hr	May repeat x 2; max dose 300mg. Monitor for cardiac suppression
Arginine	2 mEq/kg/dose over 4 hours	
Atenolol	0.25-1 mg/kg/dose q12 PO	Max 100 mg/day
Atropine	0.02mg/kg IV/IM/ET	Min 0.1mg and max 1mg
Na Bicarbonate	1-2meq/kg IV	
Bumetanide	0.03-0.1 mg/kg/dose IV/PO	Bumetanide 1 mg = furosemide 40 mg = torsemide 20 mg = ethacrynic acid 50 mg; max 10mg daily
Calcium	Calcium Chloride 20-30mg/kg IV; Calcium gluconate 100mg/kg slow IV	Gluconate requires intact liver function, chloride must be given through IO or CVL
Captopril	Infants 0.1-0.5 mg/kg/dose PO q8; Children 0.3-0.5mg/kg/dose PO q8h	
Chlorothiazide	20-30 mg/kg/day IV/PO divided q12h	max 1g/day
Cisatracurium (Nimbex)	Infusion: 0.5-2 mcg/kg/min.	
Clonidine	5-10 mcg/kg/day divided q8	Must taper if discontinuing
Dexmedetomidine (Precedex)	0.5-1 mcg/kg Infusion: 0.2-2mcg/kg/hr	Give over 5 minutes; maximum dose may be limited by ↓HR
Dextrose	0.5-1.0 gram/kg	2-4 ml/kg of D25; 1-2 ml/kg of D50
Diamox	20 mg/kg/day IV/PO divided q6-12h	Max 1g/day, use x 24 hours only.
Diazepam	0.1-0.3 mg/kg/dose	
Digoxin	30 mcg/kg initial dose- 1/2 STAT, 1/4th at 8 hours, 1/4th at 16 hours	Maintenance dose 8-10 mcg/kg/day divided q12; monitor dig levels for toxicity.
Dobutamine	2-20 mcg/kg/min	
Dopamine	2-20 mcg/kg/min	
Enalapril	0.05-0.25 mg/kg/dose q12 PO	Max 5mg/day
Enoxaparin	1-1.5 mg/kg/dose SC q12h	Check Xa levels 4 hour post second dose
Epinephrine (1:10,000)	0.01mg/kg IV/IO; 0.1mg/kg ETT; Infusion 0.02-2mcg/kg/min	
Esmolol	500 mcg IV bolus Infusion 50-250mcg/kg/min	
Ethacrynic Acid	1mg/kg/dose IV	

Medication	Dose	Special Instructions or Notes
Etomidate	0.2-0.3 mg/kg IV	*In sepsis* one dose can causes adrenal suppression
Factor VII	90 mcg/kg IV ONCE	Can precipitate DIC
Famotidine	0.5 mg/kg/dose IV q12h	Max 20mg IV q12
Fenoldopam	0.2-1.5mcg/kg/min	
Fentanyl	2-4 mcg/kg; Infusion 1-10mcg/kg/hr	Rapid push can cause rigid chest
Flumazenil	0.01 mg/kg IV	Lasts less than 1 hour**, can cause seizures if chronic benzo user**
Fosphenytoin	10-20 mg/kg IV; give over 15-20 mins Maintenance dose 6mg/kg/day divided q8-12	Target levels 15-25 mcg/ml; Check levels 4 hrs and 24 hours after load. Check albumin levels with drug levels
Furosemide	1mg/kg/dose IV/IM/PO Q6hr; Infusion 0.05-0.2 mg/kg/hr	
Glucagon	0.025-0.1mg/kg IV/IM/SC	Max 1mg; may repeat every 20 mins
Glycopyrrolate	5-10 mcg/kg IV or 20-40mcg/kg PO	Can be given up to 4 times/day
Haloperidol	5-10 mcg/kg/dose IV	Can be given up to 4 times/day
Heparin	100 units/kg bolus→20u/kg/hr unless a neonate then 28u/kg/hr	Check Xa levels q4h until therapeutic (0.3-0.5)
Hydralazine	0.1-0.5 mg/kg/dose	Max 20mg/dose
Insulin	0.05-0.1 units/kg/hr for DKA 0.1-0.2u/kg IV for hyperkalemia	Bolus with 0.5gm of glucose if using for hyperkalemia
Ipratropium	250 mcg (<5yr); 500 mcg (>5yr) nebulized q6h	
Isoproterenol	0.05-1 mcg/kg/min	
Kayexalate	1 gm/kg PO; 1.5-2mg/kg PR	
Ketamine	0.5-2 mg/kg IV; 3 to 7 mg/kg IM Infusion 0.5-2mg/kg/hour	Give with a benzodiazepine to avoid hallucinations +/- atropine for secretions
Labetalol	0.3 mg/kg IV bolus	May repeat 0.15 mg/kg every 5 mins
Levetiracetam	30mg/kg load; Maintenance 30 mg/kg/day divided q8-12	Watch for agitation with load or increasing dosage
Lidocaine	1mg/kg IV/ET; Gtt 20-40 mcg/kg/min	
Lorazepam (Ativan)	0.05-0.2mg/kg/dose Q4-6hr	⬇respiratory drive, 10% paradoxical reaction
Magnesium sulfate	25-50 mg/kg/dose	Max dose 2gms; watch for hypotension
Mannitol	0.5-1 gm/kg/dose IV can give Q6hr	Contraindicated in anuric renal failure
Methylene Blue	1-2 mg/kg IV over 5 mins	Pulse oximeter not reliable immediately after
Methylprednisolone	0.5-1 mg/kg IV q6h; 2mg/kg load	Max dose 60mg/day
Metoclopramide	0.1mg/kg/dose PO	Can be given 2-4 times per day
Midazolam (Versed)	0.05-0.3 mg/kg Q1-2 hr, Gtt 0.05-0.5mg/kg/hr	Hypotension can occur with sedation doses
Milrinone	0.25-1 mcg/kg/min	Can cause hypotension, long half-life
Morphine	0.05 - 0.3 mg/kg/hr	Can precipitate bronchospasm
Naloxone (Narcan)	0.1mg/kg IV, Drip: 0.1-2mg/kg/hour	Very short acting
Neostigmine	0.07 mg/kg IV	Reversal for paralytics
Nicardipine	0.5-5 mcg/kg/min	Titrate every 15 minutes to effect
Nifedipine	0.5 mg/kg/dose PO/SL	
Nitroglycerin	0.5-10 mcg/kg/min	

Medication	Dose	Special Instructions or Notes
Nitroprusside	1-4 mcg/kg/min	Watch for CN poisoning at higher doses
Norepinephrine	0.05-1 mcg/kg/min	
Octreotide	1 mcg/kg/hr - GI bleed; 1-4 mcg/kg/hr - Chylothorax	
Omeprazole	2mg/kg/day divided q12	
Ondansetron	0.1-0.15 mg/kg/dose PO/IV q8h prn	
Pantoprazole	0.8 to 2.4 mg/kg/day; maximum single dose: 80 mg	40 mg/1.73 m2/day; may titrate up to a maximum dose of 80 mg/1.73 m2/day
Pentobarbital	1-3mg/kg, drip 1-5mg/kg/hr	Max dose 100mg; see apnea and ↓BP
Phenobarbital	20 mg/kg IV ; Give over 5-10 min Maintenance dose 6mg/kg/day ÷q12	Target levels of 20-40 mcg/ml
Phenylephrine	0.1-4 mcg/kg/min	May load with 10mcg/kg
Phosphate	Potassium phosphate 0.3 mmol/kg	
Potassium	0.5-1 mEq/kg IV/PO; Max 30mEq.	
Procainamide	10mg/kg load over 15 minutes, 2 mg/kg/dose every 3-5 minutes; Infusion: 10-30 mcg/kg/min	
Propofol	0.5-2 mg/kg/dose; Infusion 25-200mcg/kg/min	May repeat with 0.5-1 mg/kg every 3-5 minutes until level of sedation achieved
Propranolol	0.01 - 0.1 mg/kg/dose IV; 0.5-2mg/kg/day divided q6-12 h	
Protamine	2mg/kg	
Ranitidine	4-6 mg/kg/day PO divided q12h	
Remifentanil	0.1-1mcg/kg/min	10 times more potent than fentanyl
Rocuronium	1 mg/kg/dose	
Sildenafil	0.5 to 3 mg/kg/dose Q 6 to 12h	
Spironolactone	2-4mg/kg/day PO divided q12h	
Succinylcholine	1-2 mg/kg/dose	Give with atropine. C
Sufentanil	1-5mcg/kg/hr	5 times more potent than fentanyl
Terbutaline	10 mcg/kg IV slow bolus → 0.4-10mcg/kg/min	Monitor for ST changes, chest pain. Do not escalate if HR >200.
Ursodiol	20mg/kg/day q12h PO	
Valproic acid	15-30 mg/kg/dose load	15-60 mg/kg/day divided q6
Vasopressin (DI)	0.5 milliunits/kg/***hour*** (0.0005 units/kg/**hour**)	Double dosage as needed every 30 minutes to a maximum of 40 milliunits/kg/hour
Vasopressin (Shock)	0.17 to 8 milliunits/kg/***minute***	
Vecuronium	0.1-0.2mg/kg; 0.05-0.2mg/kg/hr	

NURSING QUICK REFERENCE CARDS

Sedative/Analgesic/Paralytics/Vasoactive infusions	
Ativan 0.01 - 0.02 mg/kg/hr	Dopamine 2 – 20 mcg/kg/min
Dexmedetomidine 0.2 – 0.7 mcg/kg/hr	Dobutamine 2 - 20 mcg/kg/min
Propofol 50 -150 mcg/kg/hr	Epinephrine 0.02 – 1 mcg/kg/min
Versed 0.05 - 0.35 mg/kg/hr	Esmolol 25-500 mcg/kg/min
Ketamine 0.3 - 1 mg/kg/hr	Fenoldopam 0.2 – 0.8 mcg/kg/min
Pentobarbital 0.5 – 4 mg/kg/hr	Norepinephrine 0.02-1 mcg/kg/min
Cisatracurium 0.5 – 8 mcg/kg/min (monitor train of four)	Milrinone 0.1-1 mcg/kg/min
Vecuronium .05 - 0.2 mg/kg/hr (monitor train of four)	Nitroprusside (Nipride) 0.25 – 5 mcg/kg/min
Fentanyl 0.1 - 5 mcg/kg/hr	Phenylephrine (Neosynephrine) 0.1 – 0.5 mcg/kg/min
Morphine 0.001 - 0.2 mg/kg/hr	Vasopressin (0.4 units/ml – shock) 0.6 -2.4 units/hr
Remifentanil 0.05 - 1.3 mcg/kg/min	Vasopressin (0.05 units/ml- DI) 2.5 units/hr – titrate based on urine output

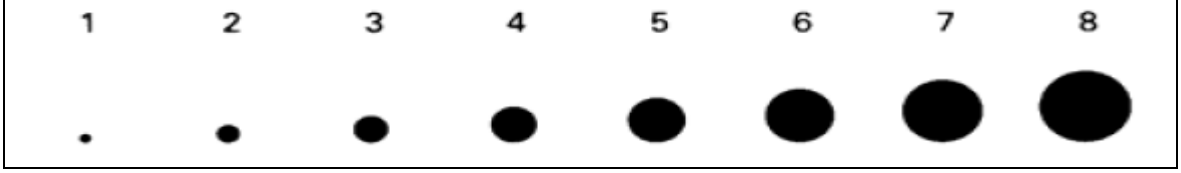
Calculations	
Concert MCG→MG = Divide by 1000 Convert MG → MCG = Multiply by 1000	**D**esired (Dose Ordered) / **H**ave (Concentration of drug available) X **Q**uantity (Volume per have)
(KG)(**MCG**/KG/HR) / DRUG CONC. (**MCG**/ML) = ML/HR	(KG)(**MG**/KG/HR) / DRUG CONC. (**MG**/ML) = ML/HR
(KG)(**MCG**/KG/MIN)(60min/hr) / DRUG CONC. (**MCG**/ML) = ML/HR	(KG)(**MG**/KG/MIN)(60min/hr) / DRUG CONC. (**MG**/ML) = ML/HR
Fluid Maintenance Calculation	
Patient's weight	Amount per kg/hr
0-10kg	4mL/kg/hr
>10-20kg	40mL + 2mL/kg (for kgs 11-20)
>20kg	60mL + 1mL/kg (for every kg over 20kg)
Add each section for hourly rate.	
SHORTCUT FOR PTS >20KG = WT. IN KG + 40 = ML/HR (IV RATE)	

Rapid Sequence Intubation (RSI)

Medications	Intubation Checklist
Atropine 0.01-0.02 mg/kg	- Mapleson with manometer & correct size mask connect to O_2 source - Yankauer & working suction - ETCO2 detector (Pedi Cap for <1month) - Intubation box (pre-check handle for working light source) - Glide scope, if requested - Roll (for under shoulders) - ETT tape and skin prep
Fentanyl 1-5mcg/kg	
Ketamine 1-4mg/kg (RN cannot give)	
Propofol 0.5 - 1mg/kg (RN cannot give)	
Rocuronium 0.6 - 1.2 mg/kg	
Succinylcholine 1-2 mg/kg	
Thiopental 1-5 mg/kg	ETT Formula = $\frac{\text{age in years}}{4} + 4$ Subtract 0.5cm for cuffed tubes
Vecuronium 0.1 - 0.2 mg/kg	
Versed 0.05- 0.1 mg/kg	Approx. Depth of ETT = Size of ETT x 3

Emergency Meds/Electricity

Under 50kgs	50kgs and over
Epinephrine 0.1mL/kg (max 1mg) **Adenosine** 0.1mg-0.2 mg/kg (max 6mg 1st dose, max 12 mg 2nd dose) **Amiodarone** 5mg/kg (max 300mg) **Atropine** 0.02 mg/kg (min 0.1 mg /max 0.5 mg) **Bicarb** 1mEq/kg (4.2% <1 month of age) **Calcium CHLORIDE** 20mg/kg (max 1gm) **Calcium GLUCONATE** 60-100mg/kg **Mannitol** 0.25gm – 1 gm/kg (22-micron filter) **Narcan** 0.1mg/kg (max dose 2mg) ***Defibrillation*** 2-4 Joules/kg ***Cardioversion*** 0.5 – 1 Joules/kg	**Epinephrine** 1mg (q 3-5 min) **Adenosine** 6 mg 1st dose, 12mg 2nd dose **Amiodarone** 300mg (dilute in 20mL D5W) **Atropine** 0.5mg (q 3-5 min/ max 3mg) **Bicarb** 1mEq/kg **Calcium CHLORIDE** 0.5-1gm **Narcan** 0.04 - 0.4mg (max 10mg) **Defibrillation** Biphasic 100-200Joules / Monophasic 360Joules **Cardioversion** 50-200 Joules



Mnemonics	
X-ray interpretation (I AM ABLE)	**Extubation readiness (CALMS)**
I- image (quality, rotation) **A**- artificial things (tubes, wires) **M**-median (heart, lymph vessels) **A**- Airway **B**- borders (heart & lung borders) **L**-lines **E**- extraneous things (surgical clips, foreign body, etc.)	**C**- CNS (alert, awake) Chest X-ray **A**- Airway (? ETT leak, cough quality) **L**- Lung function (PF ratio >200) PF ratio = PaO_2 / FiO_2 Ventilation (PS/CPAP trials, Insp. Volumes) **M**- medications (off or weaned) **S**- secretions (how many passes to clear?)

Mnemonics	
DOPE (troubleshooting deterioration in the intubated patient)	**ALL MedS**
D- Dislodgement/Displacement of ETT ✓ Is the ETT out of the trachea or in a mainstem bronchus? **O**- Obstruction of the ETT ✓ Is the tube kinked? Are there secretions or foreign body obstructing the ETT? **P**- Pneumothorax ✓ Is there symmetrical chest rise and breath sounds bilaterally? **E**- Equipment ✓ Is there an equipment problem? Take patient off vent and manually ventilate.	**A**irway- Is ETT secure? Issues w/ Trach? **L**ines- Any central lines/ urinary catheters? Can they be removed? St cath schedule? **L**abs- Appropriate labs/frequency? **M**eds- Appropriate meds? **S**leep- Is sleep protocol appropriate? **Other questions for rounds:** weight frequencies, any ??s about dressing/wound care, concerns with skin/ wounds/incisions, inconsistencies between services, etc.

Room Set Up	Admission Checklist
○ Code sheet completed with maintenance IV rate and ETT size for reference ○ Flow inflating bag with manometer (1/2-liter bag for weight ≤ 10kgs, 1-liter bag for weight >10kgs) ○ Yankauer & 2 suction set ups (cannister, tubing & suction regulator) ○ 1 large volume IV pump and 1 syringe pump ○ Appropriately sized blood pressure cuff, pulse oximetry sensor & ECG leads ○ Tape measure & Stethoscope ○ Alarms are set to correct defaults according to age	-Permission for treatment signed -Photography consent signed -Code sheet filled out w/ 2 signatures -Admission Database -Admission note documented -Height, Weight, Head Circ. ≤ 36 months -Physical Assessment -Learning Assessment & Education -Care Plans w/ end dates (5 days) -MRSA swab -Signed Crib/Isolation Waiver (if applicable) -Bands applied and charted -Review iPAD, policies and bedside shift report process

Transfer Checklist	Discharge Checklist
○ Verify transfer order ○ Report called ○ Meds/feeds collected (check fridge) ○ Belongings packed up ○ Caregivers notified if not with patient ○ Transfer note documented (include who report was given to by name)	-Verify discharge order -Verify all documentation (flowsheet/daily notes) is completed. -Review AVS with patient/caregiver -Document any LDA removals -Resolve education and care plans -Complete discharge checklist in EPIC (correct time corresponding with AVS print out) -Document discharge note -Accompany patient/caregiver to vehicle -Empty contents from blue bedside binder for processing -

Vital Signs & Normal Values

Age (KG)	Respirations Min-Max	Heart Rate Min-Max	Systolic BP Min-Max	Circulating Blood Volumes
Newborn (3-5)	30-60	90-180	50-70	Neonates 85-90 mL/kg Infants 75-85 mL/kg Children 70-75 mL/kg Adults 65-70mL/kg
6 months (7)	24-40	85-170	65-106	
1 yr (10)	20-40	80-140	72-110	
3 yr (15)	20-30	80-130	78-114	Minimum Urinary Output 0.5 - 1mL/kg/hr
6 yr (20)	18-25	70-120	80-116	
8 yr (25)	18-25	70-110	84-122	
10 yr (30)	16-20	65-110	90-130	Normal CSF production ~20mL/hr
12 yr (40)	14-20	60-110	94-136	
15 yr (50)	12-20	55-100	100-142	Normal ICP 5-15 (report ICPs >20 unless otherwise ordered)
18 yr (65)	12-18	50-90	104-148	

Equipment

Age (KG)	ETT (cuffed)	Laryngoscope Blade	Suction Cath (FR)	NG Tube (FR)	Urinary Cath (FR)
Newborn (3-5)	3.0-3.5	1 Miller	6-8	8	6
6 months (7)	3.5-4.0	1 Miller	6-8	8	8
1 yr (10)	3.5-4.0	2 Miller	10	8	8
3 yr (15)	4.0-4.5	2 Miller	10-12	10	10
6 yr (20)	5.0-5.5	2 Miller	12	10	10
8 yr (25)	5.5-6.0	2 Miller/Mac	12	10	10
10 yr (30)	6.0-6.5	3 Miller/Mac	14	16	12
12 yr (40)	6.5-7.0	3 Miller/Mac	14	16	12
15 yr (50)	7.0-7.5	3 Miller/Mac	16	16	14
18 yr (65)	7.5-8.01	3-4 Miller/Mac	16	16	16
	Miller = Straight				
	Mac = Curved				

Central Line and Ventriculostomy Care Checklist	Procedural Sedation Checklist
- Sterile Procedure sign on door - All fans off and visitors out of room - Utilize assistant for infants/small children and for all Ventric procedures - Disinfect work space - Hat and Mask for operator and assistant - Masks for child (if able) and caregiver - Use pre-assembled dressing change or blood draw kits - Hand hygiene before entering room & with each glove change - Scrub the access point with a NEW alcohol swab with every access - Document all procedures including output from CSF specimen collection.	- Ready procedural medications - Monitoring equipment (Capnometry, ECG monitor, pulse oximetry) - NSS and tubing (for bolus if needed) - Airway equipment (Mapleson, Yankauer, suction set-up- CHECK THAT ALL ARE WORKING PROPERLY) - Verify that IV is patent. - Verify consent for sedation & procedure - Complete pre-sedation checklist in narrator - Verify NPO status

Road Trip Checklist	Documentation reminders

- Refer to Sedation checklist if procedural sedation is part of the road trip.
- Obtain equipment
  o Monitor/Cables, O_2 tank, Capnoline (if needed), Mapleson (need gas source), Ambu bag, Suction machine (if needed), stethoscope
  o Emergency drug sheet
  o Trach basket (if applicable)
  o Red transport bag
  o Transport med box (in Omnicell), if needed. 911 will unlock box (Restock and return to Omnicell after trip)
	- Note placed for admissions, transfers, discharges and all unplanned events.
	- Did you do your:
	  o Restraint documentation
	  o Care plan review w/ end dates
	  o Education documentation (including isolation & MDRO)
	  o Daily goal & eval
	- Document wasted meds (email if out of 1 hr window)
	- Refer to PICU routines in PICU Standards of care policy for complete list

ARTERIAL BLOOD GAS ANALYSIS				
Abnormality		**pH (7.35-7.45)**	**CO_2 (35-45 mmHg)**	**HCO_3 (22-26mEq/L)**
Respiratory Acidosis Severe asthma, Hypoventilation	Uncomp	↓	↑	Normal
	Partially comp	↓	↑	↑
	Fully Comp	Normal	↑	↑
Respiratory Alkalosis Hyperventilation, Aspirin OD	Uncomp	↑	↓	Normal
	Partially comp	↑	↓	↓
	Fully Comp	Normal	↓	↓
Metabolic Acidosis DKA, shock, renal failure	Uncomp	↓	Normal	↓
	Partially comp	↓	↓	↓
	Fully Comp	Normal	↓	↓
Metabolic Alkalosis Loss of acid (GI), vomiting, loss of K^+	Uncomp	↑	Normal	↑
	Partially comp	↑	↑	↑
	Fully Comp	Normal	↑	↑

NOTES