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Welcome to the PICU!

Welcome to the Children's National PICU - we know the PICU rotation can invoke anxiety and anticipation but we hope you will learn more in one week in the PICU than a month on another rotation and gain confidence to take on whatever comes your way as a pediatrician.

Tips, Pearls, and Rules

- **Be present**- we know there are high demands on you but grab your laptop, avoid the backroom, and stay on the unit. You hard work will be obvious and you will learn so much more.
- **Be prepared**
  - Review your schedule of clinics, calls, and REACH time. Be aware of each other's schedules including visiting residents in the PICU – find discrepancies early
  - At the start of rounds notify the team that you are post call and by what time you need to leave. If you have any concerns about work hours violations, contact the chiefs
  - Be ready for rounds at 7:30am… computers on, at the bedside, all patients seen!
- **On doctoring** – do not be scooped
  - Examine your patients each day (no brainer right?)
  - Know diagnoses, culture results, lab trends, consultant recommendations
  - Attend family meetings and support each other in this endeavor
  - Communication - use appropriate PICU documentation Notes and minimize cutting and pasting from other notes
  - Use your fellows and attendings as resources if you need help
- **Education** - this is a great place to learn
  - Review your PICU Goals & Objectives located on Resident Book
  - Please attend teaching sessions and forward your phone to the fellows
  - Be at the bedside – again, this is the best place to learn
  - Seek out feedback on a <u>weekly basis</u> from your attendings and fellows
- **Other details**
  - If asked to put in someone else's orders, please redirect to the ordering provider
  - If you are treated unprofessionally, please let your attending or rotation director know!

<u>General Schedule for a PICU Day</u>

5:30-6am – Arrive and pre-round on your patients
7:30 or 8 am – Rounds start
10am – Radiology Rounds
9:30am – Post-call resident must be gone
Resident teaching session – either 7:30 or 11:15 or 11:30 am
Noon-1pm – Residents going to clinic or REACH sign-out
5pm – Afternoon residents not on call sign-out and leave

IMPORTANT PICU FORMULAS

RESPIRATORY

Size of Endotracheal Tube (ETT) = [Age (yr)/4] + 4

ETT distance from lip = 3 x ETT size

Airway Compliance: ΔVolume(mL/kg)/ ΔPressure(cmH_2O)

Static Compliance: [V_t(mL/kg)]/[($P_{plateau}$-PEEP)], *nl 0.6 -1 mL/cmH_2O/kg*

Dynamic Compliance: [V_t (mL/kg)]/[(PIP-PEEP)], *nl 10-20% < than static*

Minute ventilation: (V_t-V_d) x RR

Alveolar dead space ratio: V_d/V_t = (P_aCO_2 – $P_{ET}CO_2$)/ (P_aCO_2), *normal 0.2-0.3*

Alveolar Gas equation: $P_AO_2 = P_iO_2 – (P_ACO_2 /RQ)$

$P_iO_2 = F_iO_2$ x (P_B-47 torr), at sea level P_B=760 torr;

A-a gradient: P_AO_2-P_aO_2, *normal 20-65 torr on 100% O_2, 5-20 on RA*

P:F ratio: P_aO_2/F_iO_2; *If < 300 ALI, if < 200 ARDS*

Oxygenation Index (OI)= (MAP x F_iO_2)/P_aO_2;*If > 25 consider HFOV, if >30 consider ECMO*

Oxygen content of arterial blood: C_aO_2 = (Hgb x 1.34 x S_aO_2) + (P_aO_2 x 0.003)

Expiratory time E_t = (60/RR) -I_t

V = Volume
P = Pressure
V_t = Tidal V
V_d = Dead space V
PEEP = Positive end expiratory pressure
PIP = Peak Inspiratory Pressure
RR = Respiratory Rate
RQ = Respiratory Quotient (nl 0.8)
P_aO_2= *arterial* O_2
P_AO_2 = *alveolar*O_2
P_iO_2 = inspired O2
$P_{ET}CO_2$= end tidal CO_2
MAP = Mean Airway Pressure

CARDIOLOGY

RIGHT SIDE of Heart			LEFT SIDE of Heart		
Parameter	*Calculation*	*Normal Value*	*Parameter*	*Calculation*	*Normal Value*
CVP		1-6 mmHg	PCWP		6-12 mmHg
RV P		15-30 mmHg	LV Pressure		110/8 mmHg
MPA P		25/6-12 mmHg	Systemic BP		110/60mmHg
PVR*	[(MPAP-CWP)/CO]x79.9	150-250dyne*s/cm^5	Mean BP	(SBP/3)+[(2xDBP)/3)]	Age dependent
SVR*	[(MAP-CVP)/CO]x79.9	800-1500 dyne*s/cm^5	Oxygen extraction ratio	(S_aO_2-S_vO_2)/S_aO_2	0.24-0.28
CO	SV x HR	3.5-5.5 L/min/m^2	Systemic BP	SVR x CO	
SYSTEMIC HEMODYNAMICS	Pulmonary (Qp) to systemic blood flow (Qs) with a mixing cardiac lesion (Qp:Qs)			(S_aO_2-S_vO_2)/ (100-S_aO_2)	Ideal= 1.0
O_2Delivery	DO_2=CO x10xCaO_2	540-670 mL/min			
O_2 Consumption	VO2=CO x 10 x (CaO_2-CvO_2)	Infant 160-180 mL/min/m^2; Child 100-130 mL/min/m^2			
Bedside Fick	**CO = VO_2 / [1.34 x Hgb x (SaO_2 -SvO_2) x 10]**				

VITALS

Age	Pulse (bpm)	Respirations (/min)	BP	MBP
Newborn- 5 mo	100-160	30-60	60-80/30-50	40-50
6 – 11 months	110-160	24-38	60-90/40-55	45-55
1-2 years	90-150	22-30	70-90/45-60	50-60
3-4 years	80-125	22-30	70-90/45-60	50-60
5-9 years	70-115	20-24	80-100/50-65	60-70
10-13 years	60-100	16-22	85-110/55-70	65-75
14 years and older	60-100	14-20	90-120/60-80	70-80

Harriet Lane. NIH

RENAL / FEN

Glucose infusion rate: GIR = [(%glucose in solution x 10) x rate of IVF]/ [60 x Wt(kg)]
Anion Gap: AG= Na – (Cl +HCO_3), *normal 8-12*
Serum Osmolality: 2(Na) + (glucose/18) + (BUN/2.8), *normal 275-295*
Osmolar Gap = Measured Osmolality (from lab) – Calculated Serum Osmolality (above), Nl < 10
FENa = (U_{Na} x S_{Cr})/(U_{Cr}xS_{Na}) x 100%; >3% intrinsic renal, <1% pre-renal
Free water deficit (mL) = [(Na_{actual}/Na_{goal}) -1] x 1000mL/L x 0.6 mL/kg x wt(kg)
Sodium deficit (mEq) = 0.6mL/kg x wt(kg) x (Na_{goal}-Na_{actual})
3% Na replacement *symptomatic* hyponatremia: 3% NS mL = (Na_{goal}-Na_{actual}) x wt(kg) x 0.6ml/kg
IVF rate in DKA = [(3500mL/m^2 x BSA) – initial fluid bolus]/24
Corrected Calcium in hypoalbuminemia (mg/dL) = Measured total Ca + 0.8 (4.0-$S_{albumin}$)
Sodium correction in hyperglycemia = 1.6 x[(Glucose-150)/100] + measured S_{Na}
Maintenance fluid calculations (hourly):
0-10 kg: 4mL/kg/hr
11-20 kg: 40mL/hr for 1st 10kg + 2mL/kg/hr for each g>10kg
21+ kg: 60mL/kg for 1st 20kg + 1mL/kg/hr for each kg>20kg **Insensible Fluid rate** = 300-400 ml/m^2/day
Body Surface Area = $\sqrt{Ht(cm) \times Wt\ (kg/3600)}$ m^2
Parkland Formula
Total fluid = 4mL x wt (kg) x TBSA (%)
Give half in the first 8 hours *from the time of the burn* and half over the next 16 hours.
Subtract the volume of IV fluid given in the prehospital setting from the half to be given in the first 8 hours *from the time of the burn*.
Use Lactated Ringer's (LR). For kids <30kg, add D5 1/2 NS maintenance fluid to the above.

NEURO

Cerebral perfusion pressure: CPP=MAP-ICP or MAP-CVP if CVP > ICP, *goal >50-60, normal ICP <20 mm Hg*
Fosphenytoin correction = [level (measured)]/[(0.2xalbumin)+0.1]; *goal level 10-20mcg/ml*
Fosphenytoin reload (mg/kg) = Goal – Actual [Reload 1mg/kg for every "point" you want to ↑]

CONVERSIONS

2.2 lbs = 1 kg
1 in = 2.54 cm
1 torr = 1 mm Hg
Fahrenheit = (Celsius) x 1.8 + 32
Celsius = (Fahrenheit – 32) / 1.8

Celcius	Fahrenheit
36	96.8
37	98.6
37.8	100.1
38.6	101.5
40	104

RESPIRATION

Definition: Exchange of gases at the alveolar-capillary interface to diffuse oxygen into (**oxygenate**) and CO_2out of (**ventilate**) the blood.

Oxygenation at the alveolar level is dependent on:

1. Concentration of oxygen in the alveoli (alveolar oxygen or P_AO_2), determined by F_iO_2 and P_aCO_2:

   $P_AO_2 = (P_{Bar} - P_{H2O}) \times F_iO_2 - (P_aCO_2/RQ)$

   Barometric P at sea level = 760torr, P_{H2O} = 47 torr
   Normal P_AO_2 in RA = (760-47)x0.21-(40/0.8)=100torr
   RQ = 0.8

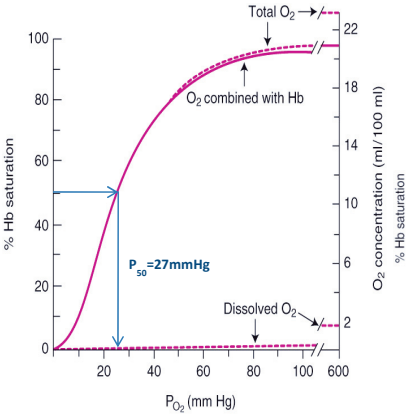
2. Amount of alveoli with oxygen receiving adequate blood flow (V/Q).
   a. ⬆number of open alveoli -⬆PEEP, mean airway pressure, or recruitment (open-lung strategies)
   b. Fix perfusion impairments - Caused by a pulmonary embolus, an AVM, pneumonia, congenital heart disease, pulmonary hypertension. Can be improved with inhaled pulmonary vasodilators like nitric oxide in some patients
3. Surface area of gas-exchange membrane allowing oxygen to diffuse into the blood.
   a. Impaired in ARDS or ALI - inflammation, scarring or fluid impairing the ability of the oxygen to diffuse across the alveolar membrane.
   b. 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.
4. The mixed venous oxygen content.
   a. 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.
5. Oxygen delivery to the tissues - 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**:

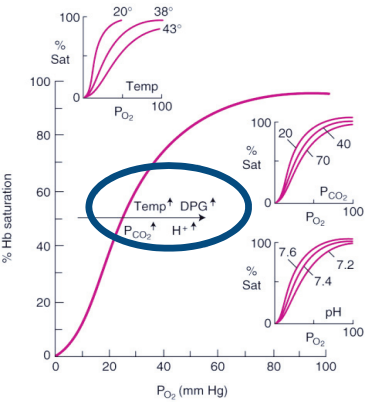
   C_aO_2 = (Hgb x 1.34 x S_aO_2) + **(P_aO_2 x 0.003)***.

   *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 (e.g. VSD, pulmonary AVM)
4. **V/Q mismatch** (e.g. 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.

6. Oxygen-hemoglobin dissociation curve-demonstrates the P_aO_2 at a given Hgb saturation.
   a. Shift to the Left = increased affinity for O2 = decreased released to tissue (Fetal Hgb, carbon monoxide, alkalosis)
   b. Shift to the Right = decreased affinity for O2 = more oxygen is released to tissue (Acidosis, hypercarbia, hyperthermia, DPG)

Ventilation of CO_2 out of the blood into the exhaled gas relies on:

1. Minute ventilation -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)
2. The gas-exchange membrane: only profound V/Q mismatch will impair ventilation

PULMONARY TOILET

1. Manual Chest Physical Therapy – 5-10 minutes of "tapping" on chest with cupped hand or airway mask to mobilize secretions, followed by laying on side to enhance drainage
2. Vest Chest Physical Therapy – Vibrating vest to mobilize secretions
3. Cough-assist Device– provides alternating positive and negative pressure to help with airway secretion clearance (range -10/+10 to -40/+40), can be done on extubated and intubated patients
4. Acapella – exhalation against vibratory valve that assist with airway secretion clearance
5. Intermittent Positive Pressure Ventilation (IPPV) – Bipap for 20 minutes every 4-6 hours for alveolar recruitment
6. Intra-pulmonary Percussive Ventilation (IPV) – high-frequency ventilation that delivers small bursts of high-flow respiratory gas with frequency higher than 1 Hz (4-10 Hz) can be used in patients on and off ventilator; consider in patients who need help with secretions

RESPIRATORY FAILURE

1. Hypoxemic or Hypercapneic or both
2. Chronic hypoxemia may be manifested by polycythemia, pulm hypertension, cor pulmonale
3. Acute Hypercapnia may be manifested by flushing, agitation, confusion, tachycardia, headache

NON-INVASIVE RESPIRATORY SUPPORT

These are the other modalities available if nasal cannula or blow-by oxygen is not enough:

1. High-flow nasal cannula: humidified air to make higher flow volumes more comfortable to the moist upper airway, each size cannula has a maximal flow because of the limitations of smaller diameter tubing.
2. Venturi mask: similar to a non-rebreather with specific concentrations of oxygen (F_iO_2) based on color of the tubing and volume of flow. You should state the $F_iO_2$and ensure flow is dialed in correctly (as stated on tubing).
3. CPAP (continuous positive airway pressure): positive pressure delivered through a tight fitting mask either over the mouth and nose or just nose. The patient can breathe above the CPAP at any rate. CPAP should be above physiologic (>+5cmH_2O) or else one could potentially suffocate.

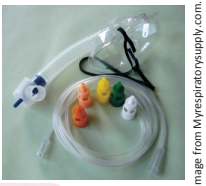


Image from Myrespiratorysupply.com.

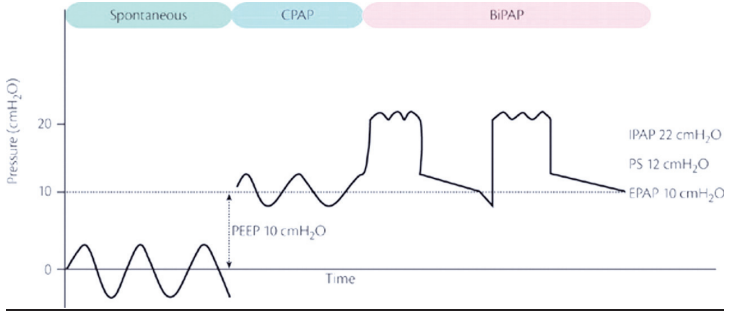


Image from "The ESC Textbook of Intensive and Acute Cardiac Care Online."

4. BiPAP (biphasic positive airway pressure): same mask as CPAP.
   a. Set an IPAP (inspiratory positive airway pressure) and an (EPAP) expiratory positive airway pressure.
   b. So the ΔP, which determines tidal volume= IPAP-EPAP
   c. BIPAP will deliver full IPAP every time the patient triggers or can set mandatory rate, "back-up rate," if you think your patient is hypoventilating

RESPIRATORY FAILURE: INTUBATION

Indications:

1. Inability to protect the airway: CNS injury (GCS <8), seizures, neuromuscular dysfunction (e.g. Guillain Barre)
2. Inability to meet the metabolic demands of respiration: shock, arrest, extremis
3. Inability to overcome severe pulmonary disease: hypoxia or hypoventilation, secretions
4. Airway compromise: croup, allergic reaction, trauma, foreign body, or ingestion
5. Sedation or procedural airway protection

Intubation Methods:

1. "Awake" aka no meds – rare!
   a. Indications: arrest, cervical spine injury, airway abnormalities, hemodynamic instability
   b. Use localized anesthetic sprays: nebulized lidocaine 2mL 1%
2. RSI (rapid sequence intubation)
   a. Indications: aspiration risk, e.g. trauma, recent meal, high abdominal pressure
   b. Technique:
      i. Pre-oxygenate with 100% oxygen, **NO BAGGING**.
      ii. Have suction ready.
      iii. Sedative + analgesia + paralytic (+/- lidocaine or atropine) given all at once.
      iv. Hold cricoid pressure ONCE MEDS GIVEN UNTIL ETT IN PLACE.
      v. Intubate once medications take effect.
3. Controlled setting in the ICU
   a. Pre-oxygenate and bag-mask the patient if need be.
   b. Have all equipment ready and IVF available to bolus if concerned about BP.
   c. Sedate and allow 1-2 minutes to take effect, bag-mask ventilate and ensure you can before administering paralytic
   d. Paralyze and wait 30-60 seconds to take effect. Start bagging immediately after administration even if not totally paralyzed yet.

Technique

1. 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.**
2. Hold corner of lip to open mouth, insert ETT through the vocal cords past the cuff.
3. 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.
4. Place CO_2 detector, aka capnograph, or in-line end tidal and look for color change (**Yellow = yes!)** or end tidal tracing
5. 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

1. May require Fiber-optic scopes, LMAs, emergency tracheostomy.
2. **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.

3. Red flags: obese, redundant neck folds, difficult to bag mask, ANY FACIAL TRAUMA, Down syndrome (C1/C2 instability), h/o SGS or OSA, micrognathia
4. Call anesthesia to assist you if you think it'll be a problem. They have a fiberoptic scope.
5. 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.**

Equipment

1. Bag and appropriately sized mask with inflated cushion
2. Suction: "Yankauer" is the large rigid catheter, ensure is hooked up to working suction
3. 100% oxygen
4. Oral airway (measure from tragus to lips to ensure correct size)
5. Endotracheal tube: preferably cuffed, ensure one size below anticipated size, make sure cuff works (Size = 4 + Age/4)
6. Laryngoscope: check bulb to make sure it lights up and that handle and blade attach, Macintosh blade – curved, placed behind epiglottis, Miller blade – straight, placed in front of epiglottis
7. Stylet to place through ETT if needed
8. Capnograph or end tidal CO_2 detector
9. Complications of Intubation: Airway injury, bleeding, pressure-induced ischemia, unrecognized eosophageal intubation, long-term subglottic stenosis

Age (regardless of wt)	Laryngoscope	ETT Size = (Age/4) + 4
Term newborn	Miller 0-1	3.0 cuffed, 3.5 uncuffed
6 months – 1 year	Miller 1	3.5 cuffed, 4.0 uncuffed
1 – 2 years	Miller 1, Mac 1	4.0 cuffed, 4.5 uncuffed
2 – 4 years	Miller 2, Mac 2	4.0 cuffed, 4.5 uncuffed
4 – 6 years	Miller 2, Mac 2	4.5 cuffed, 5.0 uncuffed
6 – 8 years	Miller 2, Mac 2	5.0 cuffed, 5.5 uncuffed
8 – 12 years	Miller 2-3, Mac 2-3	6.0 cuffed, 7.0 uncuffed
12 years and up	Miller 3, Mac 3	7.0 cuffed, 8.0 uncuffed

Intubation Medications

1. Premedication
   a. Infants and young children susceptible to vagal stimulation – Atropine
   b. Head Injury/Elevated ICP – consider Lidocaine
2. Sedation
   a. Normotensive – Midazolam + Fentanyl/Morphine OR Etomidate
   b. Hypotensive – Etomidate or Ketamine, may consider High-dose Fentanyl
   c. TBI – Etomidate (Thiopental in past but not currently on market)
   d. Asthma – Ketamine and Midazolam
3. Muscle Relaxant – ONLY IF YOU KNOW YOU CAN EFFECTIVELY BAG-MASK VENTILATE
   a. Rocuronium or Vecuronium
   b. Succinylcholine – beware of hyperkalemia, rabdomyolysis, prolonged paralysis in neuromuscular kids

EXTUBATION

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

1. Reversal or improvement of original reason for intubation.
2. Maintaining adequate ventilation, oxygenation, and work-of-breathing independent of the ventilator
3. Is strong enough to meet work of breathing needs: lifting the head or legs off the bed signifies enough strength to control the muscles of respiration.
4. Negative inspiratory force (NIF) > -20; Vital Capacity (VC) appropriate for age (think of V_t in mL/kg)
5. Mental status is adequate to protect airway upon extubation (e.g. following commands)
6. Airway will be patent <-- no significant airway swelling = leak around ETT at 20cmH_2O.
7. Minimal secretions, i.e. suctioning less frequently than every hour.
8. If no leak, consider decadron 0.25mg/kg IV q6hr x 24hrs to start at least 12 hours prior to extubation.

Equipment for extubation

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

CONVENTIONAL MECHANICAL VENTILATION

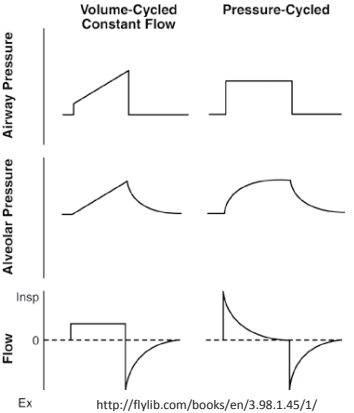
AKA Invasive Positive Pressure Ventilation= forced inhalation with *passive* exhalation.

1. ***When*** the vent breathes for the patient: "SIMV" or "AC"
   a. Synchronized intermittent mechanical ventilation (SIMV)
      i. set a mandatory rate and the patient can breathe above it with support (PS)
      ii. 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.
   b. Assist control (AC): get full pressure control with every spontaneous AND mandatory breath, to be used in only certain circumstances (e.g. SMA Type I, or paralysis)

2. ***How*** the ventilator delivers the breath: With a set **pressure** or set **volume**?
   a. Pressure control (PC): Delivers a set pressure above the PEEP, "decelerating flow pattern" just like blowing up a balloon.
   b. Volume control (VC): Delivers a set volume using a set flow.

c. Pressure-regulated volume control (PRVC):
   i. actually a pressure control mode with a smarter ventilator.
   ii. 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
   iii. This mode can adjust to dynamic compliance changes in the lung.

d. CPAP + PS: Set the PEEP ("CPAP")above which the patient breathes with PS to overcome ET tube resistance. No mandatory rate is set.



http://flylib.com/books/en/3.98.1.45/1/

Vent Parameter	Definition	Starting Setting
Tidal Volume (V_t)	Volume given with each mandatory breath	6-10ml/kg, if very stiff lungs (poor compliance) aim lower = 4-6ml/kg
Pressure control (PC)	Inspiratory pressure over PEEP, not the same as Peak Inspiratory Pressure (PIP=PC+PEEP)	Usually around 14-20cmH_2O, look for good chest rise and V_t
Pressure support (PS)	Support given by the vent for each spontaneous breath	Usually 10cmH_2O for ETT, 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. If >10, paralysis is recommended to avoid a PTX.
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-just observed	Goal < 30 cmH_2O to avoid barotrauma

VENTILATOR MODE CHEAT SHEET	Mode	What you set	Clinical scenarios for use	Advantages	Disadvantages	How to Adjust
	PRVC	V_t, PEEP RR PS 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	Oxygenation – improve with PEEP or FiO2 Ventilation – increase RR or V_t
	PC/PS	PC PEEP RR PS 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	Oxygenation – same Ventilation – increase RR or PC
	VC/PS	V_t, PEEP, RR PS 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, less physiologic, cannot use with leaks	Oxygenation – increase PEEP or FiO2 Ventilation – increase RR or V_t

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COMPLIANCE will determine what tidal volumes you can 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.

$C = \Delta V/\Delta P$
At the bedside: $C = (V_{te}/kg)/(PIP-PEEP)$

Compliance will be *high* in healthy, easily distensible lungs and *low* in ARDS, PNA or asthma. Following the compliance <u>daily</u> will show you the clinical progress your intubated patient is making.

Normal compliance is≈ 1mL/kg/cmH_20.

HIGH FREQUENCY OSCILLATORY VENTILATION (HFOV)

Definition: A gentler way to oxygenate and ventilate a patient with low compliance requiring PIPs >30 on conventional ventilation. Deliver rapid "breaths" with tidal volumes of 0.5-3mL

1. HFOV holds a mean airway pressure (MAP) - used to recruit and maintain lung volumes
2. Small oscillations around that MAP.
3. Hertz determines the frequency of the oscillations. Higher the Hertz = more oscillations.
4. Amplitude (ΔP) determines height of oscillation. Adjust to achieve "wiggle" to upper thighs.

5. F_iO_2 is targeted to maintain S_pO_2. If able to wean F_iO_2<60%, wean MAP.

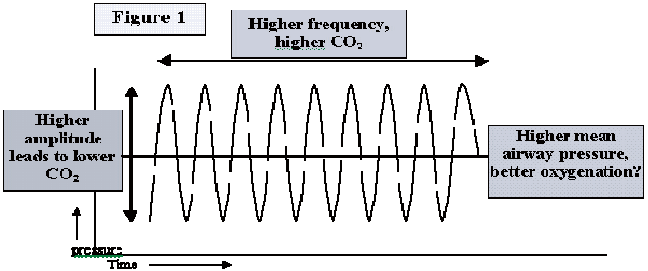


Figure from: http://www.sswahs.nsw.gov.au/rpa/neonatal/html/newprot/hfov.html

Weaning on an HFOV: Titrate FiO_2 and MAP aggressively, once able to wean MAP to < 20-24, consider switch to conventional ventilator. Titrate amplitude before Hz

Parameter	Initial Settings	Effects of manipulation
Mean airway pressure (MAP)	**10%** or **5cmH_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.

ACUTE LUNG INJURY AND ARDS

Clinical diagnosis:

1. Acute onset
2. Bilateral changes on x-ray concerning for pulmonary edema
3. Normal left-sided heart function without obstruction to pulmonary venous flow
4. **P/F ratio of ≤ 300 is ALI, ≤ 200 is ARDS. (REMEMBER P/F ratio= P_aO_2/F_iO_2)**

Causes: Primary pulmonary (PNA, aspiration, RSV, inhalation injury) vs. non-pulmonary (multi-organ failure, sepsis, pancreatitis, SIRS, TRALI, hepato-pulmonary syndrome).

Pathophysiology: infiltration of the lung parenchyma and migration of neutrophils and inflammatory mediators into the alveolar space causing a relative surfactant deficiency and impaired gas exchange. Marked by hypoxemic respiratory failure. Complications include Multi-organ system failure, pulmonary fibrosis and abnormal lung function and bronchoreactivity.

Treatment: ARDS and ALI cause poor lung compliance and issues ventilating and oxygenating.

1. "Open-lung strategy" has been proven most effective = low tidal volumes 4-6ml/kg, higher PEEP to maintain FRC if needed, permissive hypercapnia (PCO_2 50-60, pH > 7.2); APRV has worked, HFOV or ECMO V-V may be necessary.
2. Steroid-course if within two weeks is not improving (Meduri protocol 2mg/kg IV to start)
3. Conservative fluid management (AVOID fluid overload)

STATUS ASTHMATICUS

Diagnosis: Evidence of bronchospasm with poor oxygenation refractory to conventional therapy. Manifests as prolonged exhalation, poor aeration, accessory muscle use, dyspnea.

Concerning Historical or exam findings:

1. Previous ICU stays or intubations
2. Daily symptoms and frequent ER visits
3. Tachypnea or bradypnea with dyspnea (no more than a few words per breath)
4. Pulsus paradoxus: air trapping is raising the intra-thoracic pressure during inspiration to the point that it impairs venous return to the heart and therefore cardiac output
5. LACK OF WHEEZING with poor aeration can be a very bad sign!
6. Crepitus in neck or chest can be seen in severe obstruction leading to air leak
7. Impending respiratory failure if PCO_2 high or NORMAL on an ABG (MUST BE AN ABG!)

Initial Labs:

1. A free flowing arterial blood gas, remember a **normal P_aCO_2** in severe asthma is **concerning**.
2. CXR: look for expansion, flattening of the diaphragms, asymmetry (foreign body), pneumothorax, and cardiomegaly.
3. Look for the other causes of wheezing or obstructed breathing in a pediatric patient:
   a. Viral infections like bronchiolitis or pneumonia
   b. Bacterial or fungal pneumonia
   c. Laryngo-, tracheo- or bronchomalacia (BRONCHODILATORS MAKE THESE WORSE)
   d. Foreign bodies anywhere in the respiratory tree
   e. Anaphylaxis
   f. Congenital heart defects, vascular slings.

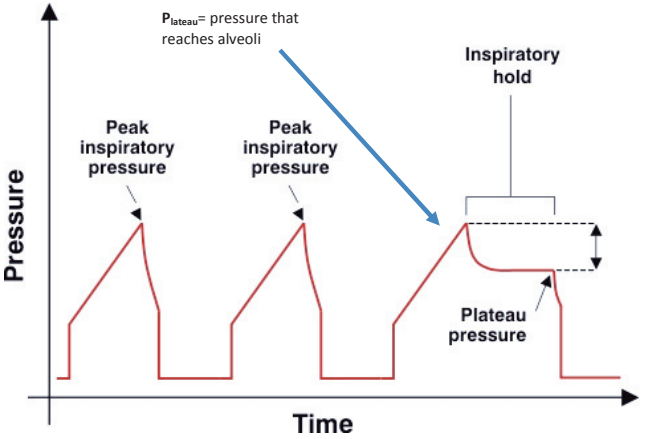
Treatment:

1. *Standard therapy*: steroids and beta-agonists (inhaled or systemic)
2. *Adjunctive therapy*: Atrovent (inhaled anticholinergic)
3. *Additional therapy*: Magnesium, Heliox, ketamine, inhaled anesthetics, IM epinephrine
4. *CPT*: secretion clearance is important (NOTE:3% NS nebs can worsen bronchospasm)
5. NIPPV may be necessary and can stave off intubation (Trial CPAP 1st, then BiPAP)
6. Ensure adequate hydration
7. GI prophylaxis is warranted if NPO and on steroids.

Mechanical ventilation in asthma:

1. Intubation of an asthmatic is VERY dangerous - 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.
   a. Lidocaine is a necessary pre-medication to blunt the bronchospasm caused by intubation, atropine may be relatively contra-indicated if already severely tachycardic.
   b. **Versed** + **ketamine** + **rocuronium** (or vecuronium) are the ideal medications.
2. Ventilation is challenging. Permissive hypercapnia may be necessary.

a. 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**
b. Titrate V_t to keep ***plateau*** pressure < 30cmH_2O and still maintain ventilation with a low RR.
c. Low or normal PEEP as patient is already hyper-expanded.
d. Watch the peak to plateau difference and aim for PLATEAU pressures < 30cmH_2O. As the obstruction from bronchospasm improves, the PIP – P_{plat} difference will lessen because it will be easier to overcome the initial resistance in the airway, i.e. the PIP will come down.
e. Ensure adequate sedation and paralysis to avoid pneumothorax at higher PIP.



Rotta A, Steinhorn D.Mechanical ventilation, acute lung injury, acute respiratory distress syndrome, status asthmaticus, permissive hypercapnia.*J Pediatr (Rio J).* 2007;83(2 Suppl):S100-8.

PULMONARY HYPERTENSION (PHTN)

Definition: The pulmonary vascular resistance (PVR) is elevated above systemic vascular resistance in utero to shunt blood away from non-ventilated lungs and through the PDA toward to body for systemic oxygen delivery. 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. There are certain pathologic conditions **where PVR fails to fall after birth or becomes elevated later in life**.

Causes: BPD and CLD, chronic hypoxia (congenital heart disease, CLD), unrepaired pulmonary venous obstruction—PAPVR or TAPVR, idiopathic pulmonary fibrosis, recurrent PE, mitral regurgitation or LVOT obstruction.

Presentation: episodes of hypoxia and sequelae 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 ventricle 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.

1. Oxygen: Hypoxia causes pulmonary vasoconstriction, hyperoxia= pulmonary vasodilation.
2. Relative respiratory alkalosis**:** Hypercapnia causes pulmonary vasoconstriction = worse PHTN
3. 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
4. Sedation and paralysis: avoid any sympathetic stimuli leading to systemic HTN or PHTN
5. Epoprostenol (Flolan): prostaglandin that directly vasodilates pulmonary and systemic arterial vascular beds and inhibits platelet aggregation. Continuous SQ/IV infusion at 2ng/kg/min, then titrate to SE tolerance. SE: chest pain, syncope, hypotension, flushing.

PNEUMOTHORAX/PNEUMOMEDIASTINUM

Diagnosis: Respiratory distress, absent breath sounds, tracheal deviation, hemodynamic compromise due to poor right-sided filling

Causes: Asthma, ARDS, malignancy, infection, emesis, perforation of the esophagus, pulmonary fibrosis, pulmonary histiocytosis, complication of CPR or mechanical ventilation, may be spontaneous, especially in young, asthenic boys.

Treatment: 100% oxygen, needle thoracentesis or subxiphoid for tension pneumomediastinum, chest tube for longer term evacuation, pleurodesis, treatment of underlying problem.

CARDIOVASCULAR

Cardiac output (CO) = Stroke volume (SV) x heart rate (HR)
Blood pressure = CO x Systemic Vascular Resistance (SVR)

SHOCK

Definition: Inadequate oxygen at cellular level necessitating anaerobic metabolism, if prolonged leads to multi-organ failure and death.

1. Compensated shock: Intrinsic systemic and cellular mechanisms are supporting metabolic needs. Subtle overt clinical signs: irritability, tachycardia, hypertension, poor cap refill.
2. Hypotensive / Uncompensated shock: Compensations overcome and perfusion to microvasculature fails causing cellular function to deteriorate = overt organ failure. Present with hypotension, lactic acidosis, lethargy. Hypotension is a late sign of shock!

Early detection is crucial to outcomes. Look for potential historical facts which could lead to shock, determine the type of shock, and identify subtle signs of compensated shock (delayed capillary refill). Normal blood pressure does not exclude shock - hypotension is a late sign!

Delivery Problem (Low S_vO_2)	Distribution Problem (not reaching vital organs, high S_vO_2, can be low)	Extraction Problem (Low S_vO_2)
Cardiogenic Shock	Septic Shock	Septic Shock
Hypovelemic Shock	Anaphylactic Shock	CO Poisoning
Obstruction (PE or Pneumothorax)	Neurogenic Shock	Methemoglobinemia
Severe Anemia		

Most common types of pediatric shock are septic, hypovolemic and cardiogenic. Septic shock often has attributes of hypovolemic, cardiogenic, and sometimes even distributive shock, so the clinical picture is highly variable.

	CO	SVR	MAP	PCWP	CVP
Cardiogenic	↓↓	↑↑↑	↔ or ↓	↑↑	↑↑
Hypovolemic	↑	↑	↔ or ↓	↓↓↓	↓↓↓
Obstructive	↓	↑	↔ or ↓	↑↑	↑↑
Distributive	↑↑	↓↓↓	↔ or ↓	↔ or ↓	↔ or ↓

CO = Cardiac output SVR = Systemic Vascular Resistance MAP = Mean arterial Pressure PCWP = Pulmonary Capillary Wedge Pressure CVP = Central Venous Pressure

Commonly used terms in shock:

1. Warm shock: warm extremities, flash cap refill, bounding peripheral pulses, evidence of poor vital

organ perfusion (AMS, oliguria)

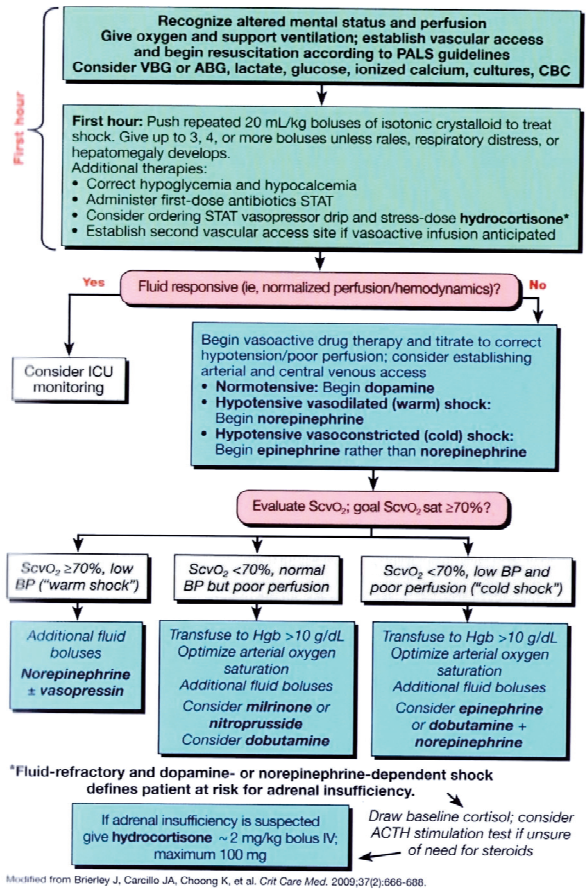
2. Cold shock: cool extremities with delayed cap refill, poor peripheral pulses, mottling, evidence of poor vital organ perfusion (AMS, oliguria)
3. Fluid refractory shock: hypotension, tachycardia despite 60ml/kg of IVF boluses
4. Catecholamine refractory shock: hypotension despite appropriate fluid resuscitation and vasoactive support.
5. Refractory shock: resistant to IVF resuscitation, inotropes, vasopressors, steroids and metabolic correction

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

1. Increasing oxygen supply and delivery:
   Oxygen delivery (DO_2) = CO (Cardiac Output) x C_aO_2 (arterial oxygen content)
   a. **CO = HR x SV** - stroke volume is dependent on preload, contractility and afterload; assure adequate filling volumes with appropriate fluid resuscitation and address contractility with inotropes if there is an inadequate response to fluid or contractility is documented to be decreased.
   b. C_aO_2=(Hgb x 1.34 x S_aO_2) + (P_aO_2 x 0.003) – optimize hemoglobin (data unclear on most appropriate hemoglobin but consider transfusion if < 10 g/dL, improve P_aO_2 with supplemental oxygen or respiratory support.
   c. Ensure distribution is to vital organs. Patients with excessive vasodilation may benefit from vasopressor medications such as norepinephrine but this is less commonly seen in the pediatric population when compared to adults.
2. Decreasing oxygen demands: Sedation to prevent anxiety, mechanical ventilation** to decrease with work-of-breathing, neuromuscular blockade, avoid hyperthermia, seizures, and cardiac arrhythmias
3. Correct any metabolic abnormalities: Lactic acidosis is the result of anaerobic metabolism from inadequate tissue perfusion, it will correct as oxygenation and perfusion to tissues improves

****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.

Pediatric Septic Shock Algorithm



Modified from Brierley J, Carcillo JA, Choong K, et al. *Crit Care Med.* 2009;37(2):666-688.

HEART FAILURE/CARDIOGENIC SHOCK

Definition: Inability of the heart to provide the necessary cardiac output to meet oxygen delivery demands and end-organ perfusion. Right heart failure occurs when the right ventricle is unable to overcome its afterload (pulmonary vascular resistance or PVR) to deliver adequate preload to the left heart. Left heart failure occurs when the systemic ventricle cannot produce adequate CO to the body.

Type	Causes	Presentation	Treatment
Right	Unrepaired VSD, pulmonary valve obstruction or insufficiency, RVOT obstruction, Tricuspid regurgitation, long-standing or acute pulmonary HTN, Unrepaired TAPVR	Hypoxia without pulmonary edema, HSM (portal HTN), peripheral edema, high CVP, high BNP	Optimize preload to right side of heart, decrease PVR (O2, Nitric Oxide, Milrinone), inotropes to support right heart
Left	Aortic coarctation, aortic atresia or insufficiency, mitral regurgitation, hypertrophic cardiomyopathy	Pulmonary edema, multi-organ failure, peripheral edema, dyspnea on exertion, clubbing, high CVP, low S_vO_2	Inotropes to increase contractility; Limit afterload on LV using diuretics and vasodilators (ACE-I, milrinone), limit oxygen consumption of and metabolic demands on heart → consider +/- intubation **see warning
Bi-ventricular	Post-arrest, myocarditis, endocarditis, pericarditis, cardiomyopathy, uncontrolled arrhythmias.	Feeding intolerance, constipation or diarrhea, dyspnea, FTT, global or pulmonary edema, newborn (7-60 dys) with irritability (remember ductal depend lesions) presenting in shock with cyanosis	Newborn in shock---suspect congenital heart disease. Prostaglandin (PG_E) 0.01-0.5 mcg/kg/min ***ASAP*** and watch for ***APNEA*** - Be careful with fluids (10 ml/kg aliquots), consider inotropes

Medications frequently used for outpatient management of heart failure

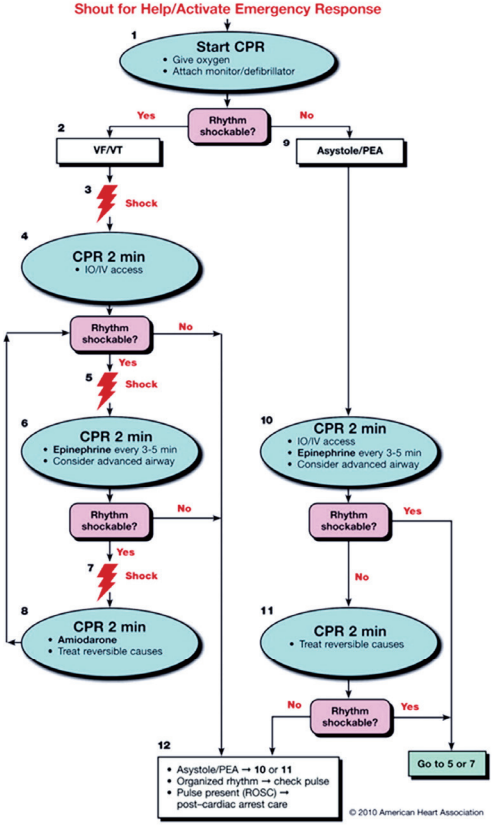
1. Carvedilol (Coreg): non-selective beta-blocker with alpha-1 blocking activity, used in conjunction with dig, diuretics, and afterload reduction. Side effects: bronchospasm, hypotension and bradycardia.
2. Digoxin: increases vagal activity through the AV node by inhibition of the Na-K ATPase pump, and is used in heart failure and to treat dysrhythmias. Side effects: heart block, bradycardia, in setting of altered electrolytes (low potassium or magnesium) will excacerbate dig toxicity
3. Enalapril (Vasotec): ACE-I which helps with afterload reduction to support left-sided heart dysfunction and LV remodeling, Side Effects: hyperkalemia, AKI, angioedema, hypotension
4. Diuretics (Aldactone and Lasix)

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.

THINK ECHO early!

PEDIATRIC ADVANCED LIFE SUPPORT (PALS)

Pediatric Cardiac Arrest



Doses/Details

CPR Quality
- Push hard (≥⅓ 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.

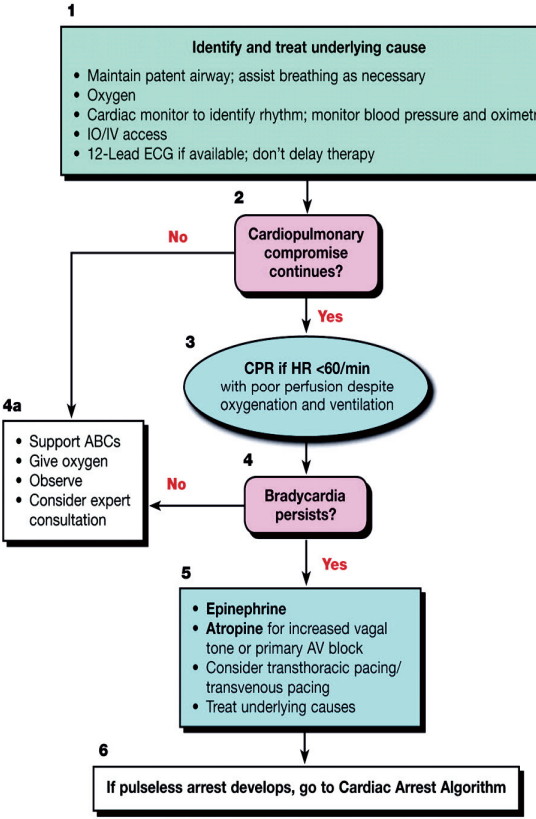
Drug Therapy
- **Epinephrine IO/IV Dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes.
  If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV Dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2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

Pediatric Bradycardia
With a Pulse and Poor Perfusion



Cardiopulmonary Compromise

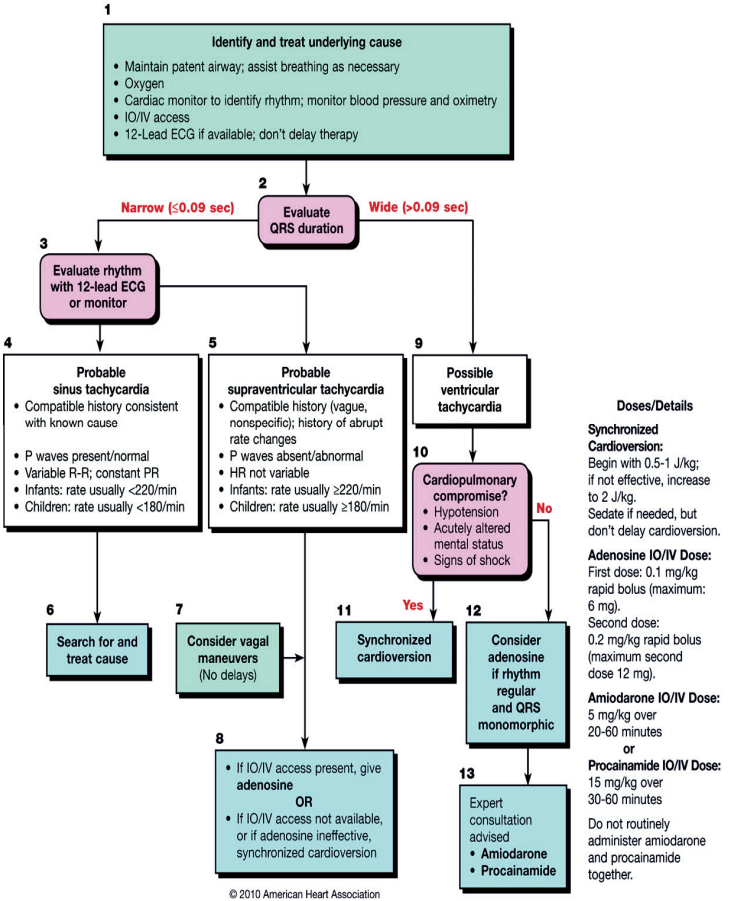
- Hypotension
- Acutely altered mental status
- Signs of shock

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.

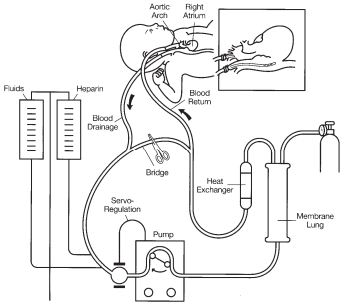
Pediatric Tachycardia
With a Pulse and Poor Perfusion



ECMO (Extracorporeal Membrane Oxygenation)

Indications: acute and reversible refractory respiratory and/or heart failure, Wt > 2 kg, >34 weeks GA.

Contraindications: IVH > grade II or active bleeding, irreversible disease process, fatal congenital disease, profound neurologic dysfunction.



http://www.nichd.nih.gov/publications/pubs/ecmo_report.cfm

V-V (venovenous): provides respiratory support

1. Cannula in vein removes deoxygenated blood from IVC, SVC and returns oxygenated blood to the RA, keeps pulmonary blood flow intact and does not ligate the carotid artery.
2. Only used in primary respiratory failure, heart function must be normal.
3. While on ECMO, use low to normal ventilator support to avoid lung collapse.

V-A (venoarterial): supports the heart and lungs

1. Deoxygenated blood removed from the SVC junction at the RA goes to a pump ("heart") which generates flows to propel the blood through the oxygenator and ventilator ("lung") and back to the aortic arch. (Can also be cannulated in the groin, or a combination of neck and groin)
2. Cardiac ouput generated by the ECMO pump (CO=flow); Oxygenator and sweep gas perform gas exchange.
3. Patient can be extubated or allow lungs to be on full rest vent settings.

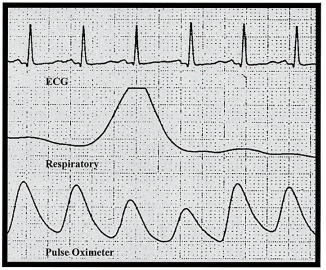
PERICARDIAL TAMPONADE

Definition: rising pericardial pressure obstructs venous return and cardiac output

Causes: Pericardial contusions or lacerations leading to hemopericardium, infection, post-operative reactive effusions, malignancy, rheumatic conditions

Presentation: Beck's triad - pulsus paradoxus, quiet precordium/distant heart sounds, and distended neck veins; may be less obvious presenting as shock, unexplained tachycardia. Echocardiogram (right atrial diastolic collapse) or CT may be helpful but diagnosis is clinical.

Treatment: Expand intravascular volume – patients are preload dependent!, be very ca Pediatrics, April 2002, 109(4) decrease preload and cause cardiovascular collapse, pericardiocentesis



SODIUM DYSREGULATION

Correction: never attempt to correct serum sodium by more than 1mEq/L/hr, especially if asymptomatic or if the sodium abnormality has existed for longer than 48 hours. Correct it gently by 0.5mEq/L/hr.

FE_{NA} (fractional excretion of sodium) - to help determine if the kidneys are handling sodium appropriately in each clinical setting.

1. Remember, the FE_{Na} is not clinically relevant if diuretics given in the last 12 hours.
2. FE_{Na} <1% in pre-renal azotemia or acute GN, 1-2% Intrinsic renal disease, > 2-4% (depending on source) post- renal obstruction, ATN.

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

3. Prerenal azotemia – hypovolemia, hypotension etc.
4. Intrinsic renal disease – ATN, glomerulopathies, etc.
5. Post-Renal – posterior urethral valves, nephrolithiasis, etc.

Hypernatremia

1. Differential diagnosis: dehydration, DI (see endocrine section), iatrogenic, excess Na load, increased free water loss from renal concentrating defect, burns, etc.
2. Presentation: Primarily neurologic – lethargy, weakness, AMS, irritability, seizures
3. Dehydration Hypernatremia: Calculate
   Free Water deficit= $[(Na_{actual}/Na_{goal}) -1] \times 1000mL/L \times 0.6\ mL/kg \times wt(kg)$to give over time to expect correction at 0.5 mEq/L/hr.

Hyponatremia

1. Differential diagnosis: SIADH (see endocrine section), cerebral salt wasting, iatrogenic, diuretics, heart failure or liver failure, excess free water, Falsely low Na from hyperlipidemia, hyperproteinemia, and hyperglycemia
2. Calculate: Sodium deficit (mEq) = 0.6mL/kg x wt(kg) x (Na_{goal}-Na_{actual})
3. Cerebral Salt Wasting (CSW) = inappropriate sodium handling, lose more sodium than free water
   a. Diagnosis: hyponatremia from loss, high urine output and high FE_{Na} in a setting of hypovolemia.
   b. Causes: Neurosurgical procedure, intracranial mass, TBI or CHI with ICH, HIE
   c. Treatment:
      i. Aggressive rehydration with 0.9NS and 3% sodium replacement via central line or large bore IV using the above calculation
      ii. Fludrocortisone can stop polyuria, urine replacement with 3% or NS may be necessary.
   d. CSW usually resolves on its own in 4-6weeks after the initial CNS insult.

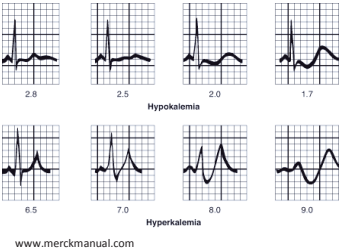
OTHER ELECTROLYTE DERANGEMENTS

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 (Ca<4.5)	Hypoparathyroidism, multiple pRBC tx, diet, alkalosis, CRRT, lasix, malabsorption, hyperphosphatemia, hyperlipidemia	Paresthesias, bronchospasm, apnea, seizures, prolonged QT, Rickets, Chvostek sign (facial spasm), Trousseau sign (carpopedal spasm)	Calcium supplementation, need to replete magnesium as well
Hypercalcemia (Ca>10)	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 bisphosphates if severe; CRRT
Hypokalemia (K<3)	Diet, medications, alkalosis, diarrhea, Adrenal-cortical excess, Bartter syndrome	Arrhythmias (PACs, PVCs), mild muscle weakness	KCl supplementation *KPhos is poor K supplier*
Hyperkalemia (K>6)	Renal failure, acute acidosis, tumor lysis, rhabdomyolysis, pRBCs tx, medications, adrenal insufficiency	Fatal arrhythmias, fatigue, mm weakness, disorientation, palpitations, paresthesias	CaCl, Bicarb ($NaHCO_3$), insulin/glucose, kayexalate (C BIG K pneumonic) also albuterol, Lasix, +/-CRRT
Hypomagnesemia (Mg<2)	Diarrhea, iatrogenic, diet, insulin for DKA, massive RBC tx, CPB, burns	Symptoms usually from resultant ↓Ca: mm weakness, tetany, seizures, hypokalemia	Magnesium supplementation
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	Phosphate supplementation with KPhos or NaPhos
Hyperphosphatemia (Phos>9.5)	↑intake or ↓renal excretion	Refractory hypocalcemia	Hydration, stop Phos sources; Phosphate binders

Hyperkalemia

Definition: Serum K > 6mEq/L is life threatening because the intracellular potassium gradient is pivotal in 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 + 0.5-1 gm dextrose over 15-30 minutes (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 (use through OG or NG) or PR, avoid in any patients with risk of bowel ischemia
   e. Sodium bicarbonate: 1mEq/kg IV over 5 minutes, must have control of ventilation because will convert into PCO_2 and **worsen acidosis and hyperkalemia** if already not ventilating well.
   f. Lasix: 1 mg/kg to augment diuresis
4. Establish venous access for emergency hemodialysis.

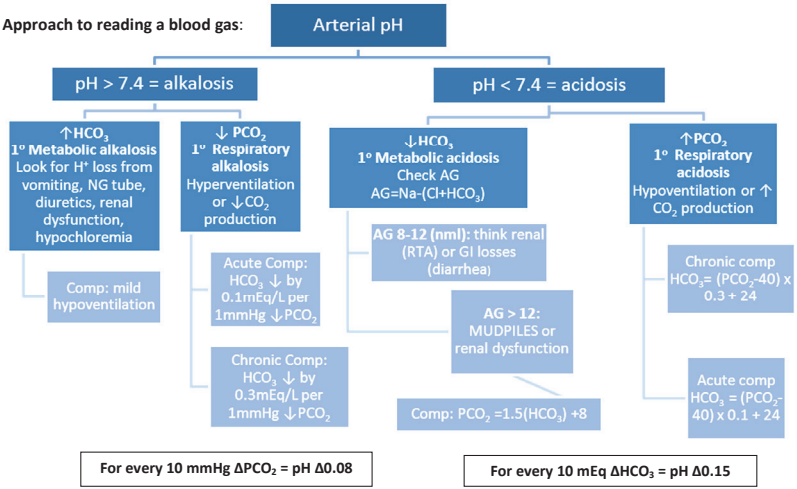
ACID-BASE BALANCE

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

Electrolyte changes in Acid/Base disruption:

1. Potassium: serum level affected by H^+-K^+ transporter
   a. A**lk**alosis = **l**ow serum **K**$^+$ because shifted into cells, total body K^+ is the same
   b. Acidosis = high serum K^+, if prolonged the kidneys will compensate by excreting K^+
2. 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

Approach to reading a blood gas:



Pneumonic

1. Anion Gap Acidoses: MUDPILES – Methanol, Uremia, DKA, Paraldehyde/ Propylene Glycole, INH, Lactate, Ethanol, Salicylates
2. Normal Gap Acidoses: HARDUP – Hyperalimentation / Hyperchloremia, Acetazolamide, RTA, Diarrhea, Uretero-enteric fistula, Pancreatic-duodenal fistula

Buffers

1. In Acidosis:
   a. 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.
   b. Tromethamine (THAM): Dose based on **THAM (mL of 0.3molar solution) = wt (kg) x BD (mmol/L)**. Give over 2-4 hours. Inert amino alcohol buffer which supplements the buffering capacity of the blood by accepting a proton and decreasing P_aCO_2. Monitor for hypoglycemia.
2. In Alkalosis:
   a. Chloride replacement if Cl < 90: use NaCl or KCl, **Cl deficit = 0.3 x wt x (100-present Cl)**
   b. Ammonium Chloride: 1mEq/kg/dose IV Q6hr x 4 doses at max rate of 1mEq/kg/hr
   c. Hydrochloric acid: via CVL, **H^+ deficit = 0.5 x wt x ($HCO_{3current}$-$HCO_{3\ goal}$)**, 0.05-0.1mEq/kg/hr.
   d. Diamox: 5mg/kg/dose q6hr, promotes renal excretion of HCO_3. NOTE: weak diuretic effect = ↓ K.

HYPERTENSIVE EMERGENCY

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

Causes: Multifactorial→ high renin states or increased sympathetic tone, pain, seizures, elevated ICP, renal vascular disease, ingestion, coarctation of the aorta, Posterior Reversible Encephalopathy Syndrome (PRES) , exogenous steroids

Treatment: ↓BPs **by 20-25%** acutely to control symptoms and then ↓to ***baseline*** within 24-48hours.
*Medications can be found in cardiac medications

RENAL FAILURE AND RENAL REPLACMENT

Definitions:

1. AKI: Cl_{Cr} decrease by 60% and oliguria for > 16 hours.
2. ARF: Cl_{Cr} decrease by 75%, oliguria for > 24 hours or anuria for 12 hours. Inability to perform renal functions including acid/base balance, fluid balance, electrolyte regulation, excretion of waste.

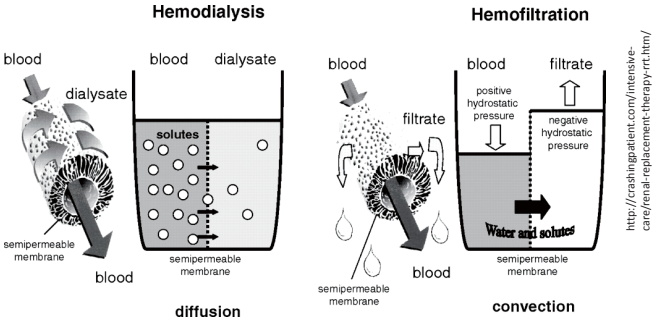
Diagnosis: azotemia (increased BUN and SCr) and oliguria 0.5ml/kg/hr or anuria or fluid overload.

Etiology: Prerenal (most common in children), renal or Postrenal obstruction. FE_{Na} >2% in ATN or post-renal obstruction; <1% in pre-renal azotemia or acute GN.

Management:

1. FEN: Limit fluid intake (insensible = 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 prot:cr ratio (proteinuria)
3. If transfusing, may *wash* the pRBCs to avoid high potassium load
4. Renal replacement therapy (RRT): performs the exchange of solute and water between two solutions (blood to dialysate) across a membrane
   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

INDICATIONS FOR RRT
A – Metabolic Acidosis
E – Electrolyte imbalance not amenable to medical therapy
I – Toxic ingestions
O – Fluid overload
U – Uremia (symptomatic)



c. Peritoneal dialysis (PD): uses the peritoneum as the membrane for both convection and diffusion solute clearance. Need healthy, intact peritoneum (no diaphragmatic hernias, adhesions or previous surgeries or active peritonitis). *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. ***Types of CRRT:***
    i. SCUF (slow continuous ultrafiltration): free water and small molecule clearance
    ii. CVVH (continuous venovenous hemofiltration): convective based solute clearance
    iii. CVVHD (continuous venovenous hemodialysis): diffusion based solute clearance, removes small molecules down the concentration gradient
    iv. CVVHDF (continuous venovenous hemodiafiltration): both convection and diffusion based clearance.
    v. Complications: 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

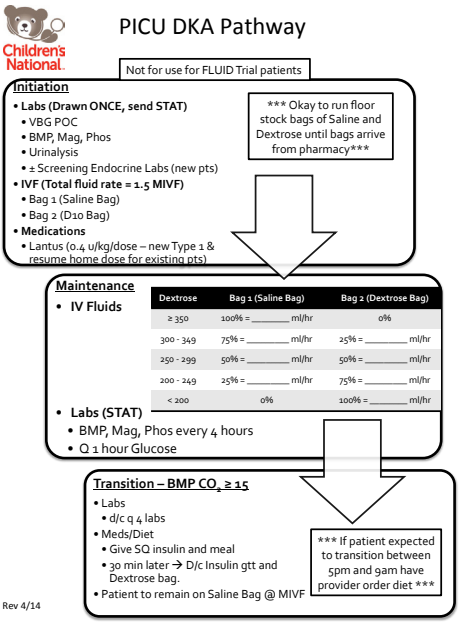
DIABETIC KETOACIDOSIS (DKA)

Diagnosis: Metabolic acidosis, Hyperglycemia >200, Presence of Ketones

Initial Evaluation:

1. History: CC and HPI including any ingestions or exposures, family history of endocrinopathy, identifying a *trigger* is essential if a known diabetic.
2. PE*:* VS for Cushing's triad from cerebral edema, **mental status, mental status, mental status,** degree of dehydration, respiratory pattern, goiter, cellulitis around home injection sites
3. Labs*:* D-stick, urinalysis, chemistry with Mg and Phos, iCa, CBC, Hgb_{A1c}, pH (arterial is preferable) [New diagnosis: include insulin level, c-peptide]
4. Complications: arrhythmias due to hypo/hyperkalemia, cerebral edema, shock, hypoglycemia, cerebral edema

ICU Treatment - please refer to Children's DKA Pathway, in brief:



1. Fluids:
   a. Initial fluid bolus should be around 10mL/kg, only give 20mL/kg if in shock, often this is given in ED.
   b. Infusion rate after bolus = [(3500mL x BSA (m^2)) – initial bolus]/48 ≈ 1.5x Maintenance
   c. Two bag system: D10NS with 40mEq potassium and NS with 40mEq potassium (as KPhos, KAcetate or KCl), may be ½ NS for some patients, refer to the protocol
   d. If not voiding, do not put any electrolytes (potassium) in the IVF. If K<3, increase total potassium in bag to 60mEq/L. If K <2, increase to 80mEq/L.
   e. If Phosphorous is < 2 at any point, increase KPhos in IVF to 40mEq/L
2. **Avoid** giving bicarbonate unless pH <7.0 and patient is hemodynamically unstable – discuss with fellow. Bicarb may help acidemia but cannot cross the blood brain barrier. The patient will get paradoxical CNS acidosis because the Bicarb converts to CO2, crosses the blood-brain-barrier. This CO2 will then increase cerebral venodilation potentiating increased ICP and will combine with water to generate more H+ thus leading to worsened cerebral acidosis.
3. Insulin: continuous infusion to start after initial IVF bolus at 0.1units/kg/hr. Ideally serum glucose should not fall by more than 150mg/dL in one hour. Do not stop the infusion if serum glucose is less than 150 if the patient is still acidotic and making ketones. Just increase the dextrose.
4. Mannitol: at any signs of altered mental status, obtain head CT and consider mannitol, 0.5-1gm/kg.

Once arterial pH >7.3 (venous pH>7.25) and HCO_3 is > 15 on BMP, convert to intermittent insulin per Endo's recommendations. Call endocrine and transfer to floor.

ADRENAL INSUFFICIENCY (AI)

AI is suspected in the presence of unexplained catecholamine-resistant shock or recurrent episodes of hypotension ("septic" episodes), chronic steroid therapy or endocrinopathies (e.g. pan-hypopit).

Diagnosis: *ACTH stimulation test*: draw baseline cortisol, administer an ACTH analog (Cosyntropin 1mcg dose IV), then obtain repeat Cortisol levels at 30 and 60 minutes 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: **50mg/m^2** or **2mg/kg** if surface area is not available as a loading dose & then **50mg/m^2/day** or **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
Prednisilone	5		4	0.8	18-36 hours
Methylprednisilone	5	40-80mg	5	0.5	18-36 hours
Dexamethasone	0.75	7.5-30 mg	25	0	36-54 hours

DISORDERS OF ANTI-DIURETIC HORMONE

Vasopressin (aka Anti-diuretic hormone or ADH)

1. Synthesized in the hypothalamus and stored and released from the posterior pituitary.
2. Released in response to increased plasma osmolarity (>285mOsm/L), decreased stretch of the baroreceptors in the Aorta (decreased BP).
3. 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.

Syndrome of Inappropriate ADH (SIADH) - Pathologic excess of ADH

1. Diagnosis: Hyponatremia from free water retention, low urine output, concentrated urine (urine osm > 500mOsm/L, spec grav > 1.015) and decreased serum osmolarity (<275mOsm/L). Initially mild hypervolemia then euvolemia.
2. Causes:
   a. CNS infection, traumatic brain injury neurosurgery, brain tumors.
   b. Lung pathology: PNA, high PEEP, mechanical ventilation
   c. Para-neoplastic syndromes
   d. Medication side-effect, adrenal-cortical dysfunction from other causes.
3. Treatment:
   a. Fluid restriction to insensible fluid rate (approximately 1/3 maintenance or 400/m^2).
   b. Sodium replacement with 3% NS or NaCl and a loop diuretic.
   a. Calculate **Sodium deficit (mEq) = 0.6mL/kg x wt(kg) x (Na_{goal}-Na_{actual})**
   c. 3% replacement in *symptomatic* hyponatremia: **3% NS mL = (Na_{goal}-Na_{actual}) x wt x 0.6mL/kg**

Diabetes insipidus (DI)

Deficiency of ("Central") or lack of response to ADH ("Nephrogenic")

1. Diagnosis: Large amounts of dilute urine (6-8ml/kg/hr, urine osm <300 or spec grav <1.005) and hypernatremia with high serum osm (>305mOsm/L), volume depletion. Loss of free water causes further aldosterone secretion to stimulate more sodium absorption and worsens the hypernatremia.
2. Causes:
   a. Central DI (lack of ADH production): Injury to pituitary and/or hypothalamus as seen in CHI, tumors, seizures, meningitis, brain death, CVA or ICH.
   b. Nephrogenic DI (lack of response to ADH): AKI, genetic defects of the kidney itself.
3. Treatment:
   a. Calculate free water deficit, give IVF at insensible rate + urine output 1:1 with D5W.
   b. **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.
   c. Vasopressin drip at 0.5-10milliunits/kg/hr can replace an ADH deficiency in *central DI*, titrate every 30 minutes until urine output stops and then decrease dose slowly.

ANEMIA

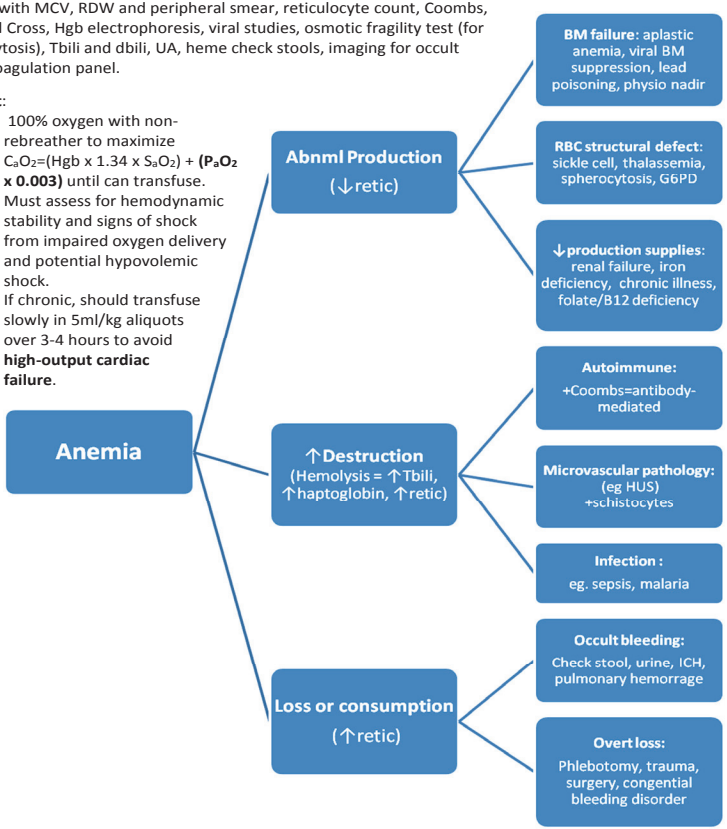
Definition: Hgb< 10-12gm/dL

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 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 = platelet count< 50,000/mm^3.

Causes:

1. ↓production of platelets: BM suppression, medication effect, Fanconi anemia, Wiskott Aldrich
2. ↑destruction or consumption: infection/sepsis, 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, thrombocytopenia, elevated INR, low fibrinogen, high fibrin degradation products

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

TRANSFUSION MEDICINE

PICU Pearls

Hyperkalemia, **hypocalcemia** (low **iCa**) are common side effects from pRBCs
Fluid overload is a potentially dangerous side effect of multiple transfusions
DO NOT push PLATELETS. Can cause refractory hypotension.

Blood Component to Replace

1. If a patient has significant blood volume loss consider transfusion with all blood components
2. Estimated blood volume is dependent on age of patient:
   Preterm neonate = 100-110 mL/kg; term
   Neonate = 85-90 mL/kg;
   >1 month – Adult = 75 mL/kg;
   Adults (male) = 65 mL/kg, adults (female) = 60 mL/kg

Packed RBCs - this is a source of exogenous Hgb to improve O_2 delivery according to:

$$C_aO_2 = (\mathbf{Hgb} \times 1.34 \times S_aO_2) + (P_aO_2 \times 0.003).$$

1. Content: 1 unit of pRBCs= approx. 250-300 mL with a Hct of 50-60%.
2. Dosing
   a. for patients <50 kg, use volume care-set: approximately 10 mL/kg of pRBCs = 2-3 gm/dL increase in Hgb
   b. for patients > 50 kg, use unit care-set: 1 – 2 units given will increase Hct by 3 to 6%

3. Indications:
   a. Severe symptomatic anemia (Hgb< 7 gm/dL)
   b. Hypoxemia from pulmonary pathology and Hgb< 10 gm/dL
   c. Shock, MODS and Hgb< 10 gm/dL
   d. Unrepaired cyanotic heart disease and Hgb< 12 gm/dL
   e. Ongoing bleeding or anticipated blood loss from surgery.
4. Preparations:
   a. Leukocyte reduction:
      i. Removes WBC by 10^3 fold so that blood product has < 5 x 10^6 WBCs
      ii. Decreases febrile, non-hemolytic reactions
      iii. Reduces platelet alloimmunization
      iv. Reduces transfusion associated CMV infection
      v. Reduces but does not eliminate risk of transfusion associated graft vs. host disease.
   b. Irradiation:
      i. Purpose: 1) prevents proliferation of donor lymphocytes, 2) eliminates risk of transfusion associated graft vs. host disease.
      ii. LIPs are required to notify blood bank of patients with potential oncologic or immunodeficient diagnoses so proper irradiation restrictions can be placed.
   c. Volume reduction:
      i. Reduce plasma protein content in platelets/RBCs
      ii. Reduces risk of hemolytic transfusion reaction to ABO incompatible platelets when there is need to give ABO incompatible platelets
      iii. Decreases volume of cellular products (RBCs and platelets) when volume restriction is needed.
   d. Washing:
      i. *R*educes the plasma of RBCs/Platelets and supernatant K+ content of RBCs
      ii. Used in patients with risk of anaphylaxis due to anti-IgA or severe allergic reactions not prevented by premedication or patients with high risk of hyperkalemia related effects.
      iii. Note: fresher RBCs or additive RBC units can be used instead of washing RBCs.

3. Complications: Hyperkalemia, hypocalcemia, hypothermia, low 2,3 DPG of transfused blood shifts the oxygen-Hgb dissociation curve to the left making it harder to unload O_2 at the tissue level. Risk of blood-borne illness (rare) and other reactions as below.

Platelets

Available as apheresis platelets in single or double units (one donor, single = $3x10^{11}$). . .

1. Content: 1 apheresis unit = 200-300mL or 4-6 equivalent whole blood derived units (EU); One EU has 5.5 x 10^{10} platelets and 25-50 mL.
2. Dosing: For all patients
   a. 1 EU/5 kg - Bleeding patients)
   b. 1 EU/10 kg - Non-bleeding patients
   c. Max 6EU for both dosing schemes.
3. Expected increment: 1 EU/5 kg expected to increase platelet count by 50,000 to 100,000 in bleeding pediatric patients. Similar increment in non-bleeding patients getting 1 EU/10 kg.. Note: At CHILDREN'S NATIONAL all platelets are leukocyte reduced

4. Indications: severe thrombocytopenia with active bleeding or at risk for spontaneous bleeding.
   a. Spontaneous bleeding not likely until <5,000. Risk increases if coagulopathic or infected, consider transfusion when <10-20,000 without bleeding.
   b. Transfuse if undergoing a procedure when < 50,000, unless a CNS procedure or ICH then transfuse if < 100,000.
   c. Transfusion NOT indicated in ITP, TTP and HUS unless having life-threatening bleeding.
   d. Contraindicated in HIT because may precipitate thrombosis
   e. Acquired disorders of platelet function
      a. NOTE: Transfused platelets will still be dysfunctional in uremia.
5. Complications:
   a. Rare risk of Hep C, B, HIV I/II, HTLV I/II, West Nile Virus, Chagas disease, syphilis, bacterial contamination (Note: RBCs not bacterially tested. Infections disease risks apply to all blood components besides platelets)
   b. Febrile non-hemolytic reactions FAR more common than after pRBC transfusion.
   c. Higher risk of bacterial infection because stored at room temperature.

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. Content: 1 unit = 250mL, needs to be ABO compatible.
2. Dosing:
   a. For <50 kg, use volume care-set 10-15mL/kg, to provide ~15-20% rise in factor levels.
   b. For > 50, unit care-set 2-3 units of FFP will increase ~15-20% rise in factor levels.
3. Indications: Coagulopathy in the setting of potential bleeding (procedures, surgery, trauma) or active bleeding.
   a. DIC
   b. Acute liver failure with bleeding
   c. Massive blood loss (>60ml/kg)
   d. Multiple pRBC transfusions resulting in dilutional coagulopathy

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

1. Content:
   a. 1 unit from a single donor = 10-15mL;
   b. 1 unit=80IU of Factor VII, 150 mg of fibrinogen, von Willebrand factor and factor XIII.
2. Dosing: (For all ages) 1-2 bags/10 Kg (average volume 10-15 mL); Max: 6 bags
3. Expected Increment: 60-100 mg/dL rise in fibrinogen
4. Indications:
   a. Hypofibrinogenemia and active/suspected bleeding or prior to an invasive procedure
   b. Von Willebrand disease when conventional treatments not available (e.g. DDAVP or factor concentrates)
   c. Dysfibrinogenemias, inherited or acquired, as seen in acute liver failure
   d. DIC with active bleeding

BLOOD TRANSFUSION REACTIONS

In all cases please contact Transfusion Medicine Attending on-call

Acute hemolytic transfusion reactions: Antigen/antibody response to incompatible blood leading to complement activation and intravascular hemolysis.

1. Presentation: fever, rigors, chest pain, tachycardia, hypoxemia and tachypnea, urticaria, angioedema, hypotension and shock, hemoglobinuria
2. Diagnosis: positive DAT (direct antiglobulin test)
3. Treatment: stop transfusion, send donor blood to blood bank for testing, support respiratory and cardiovascular systems as needed, may need red cell exchange

Febrile non-hemolytic reactions: preformed anti-HLA antibodies in the recipient which react to donor WBC or platelets triggering cytokine release or endogenous cytokines present in the blood product

1. Presentation: fever during or immediately after transfusion, chills/rigors, headache, nausea and vomiting, tachycardia
2. Treatment: antipyretic (acetaminophen),

Allergic, anaphylactic reactions: recipient has an IgE antibody directed against an antigen in the donor plasma (allergic); seen in IgA deficient patients who may have an IgG antibody to IgA (anaphylactic)

1. Presentation: wheezing, urticaria, vomiting/diarrhea, angioedema, airway swelling, cyanosis, hypotension, shock, AFEBRILE
5. Diagnosis: can send IgA and anti-IgA levels to diagnose deficiency
6. 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 or IgA negative products if available.

TRALI: Usually HLA-mediated compliment activation and neutrophil migration to the lungs. Neutrophils cause capillary leakage and pulmonary edema/damage.

1. Presentation: respiratory distress, hypoxia within 6 hours after transfusion, bilateral pulmonary infiltrates on CXR, fever, mortality rate of 10%
2. Diagnosis: check for antibodies against HLA or neutrophil specific antigens, the blood bank will test the donor blood and cross-match.
3. Treatment: Supportive, provide oxygen, +/-mechanical ventilation, steroids not indicated

GVHD: engraftment of donor T-cells that are HLA-incompatible with the recipient causing immune mediated attack on BM, liver, skin, GI tract.

1. Presentation: fever, diarrhea (bloody or watery), maculopapular rash, hepatitis, pancytopenia occurring 8-10 days after a transfusion
2. RARE, usually seen in immune-compromised patients
3. Avoid by **irradiating** blood components

INDICATIONS FOR THERAPEUTIC APHERESIS

Definition:

1. Removal and/or replacement of WBC, RBCs and/or plasma using an automated cell separation as a means of treatment.
2. Process depends on the density of various constituents of blood as means of separation.
3. In the case of extracorporeal photopheresis, WBCs are removed, exposed to 8-methoxypsoralen (methoxalen) and UV-A irradiation and infused back to the patient.

RBC exchange

1. Primary indications are for sickle cell disease patients with acute stroke and acute chest syndrome.
2. Procedure: Sickle RBCs are exchanged for antigen matched RBCs. Procedure takes about 1.5 to 2 hours to complete. There is a requirement to know what kind of antibodies the patient has, if any, in order to get the units needed for exchange in a timely fashion.

Peripheral Blood Stem Cell Collection (PBSC):

1. Primary indication: autologous/allogeneic collection of peripheral blood stem cell for stem cell rescue or hematopoietic stem cell transplantation.
2. Procedure: Generally 3-6 blood volumes are processed with removal of the buffy coat. Process takes between 2.5- 5 hours for completion. Logistically, WBC count and CD34 counts are followed in order to optimally time the start of harvest.

Plasma Exchange

1. Primary indications:
   a. Removal of pathologic substances from the plasma (e.g. antibodies [TTP, myasthenia gravis, Guillain Barre, antibody mediated rejection, etc.] overdose medications, abnormal proteins)
   b. Replenishment of normal constituents of plasma (e.g. Factor I (aHUS), ADAMTS13 deficiency, [TTP], coagulation factors for liver failure)
2. Procedure: Depending on the disease or condition, generally 1.0-1.5 plasma volumes are removed and replaced with either FFP or 5% albumin.

Leukoreduction

1. Primary indication is for removal of WBCs (blasts) in patients with acute lymphoblastic leukemia or acute myelogenous leukemia with leukostasis or high risk of leukostasis.
2. Procedure: Generally 2-3 blood volumes are processed as in PBSC, except that collect rate is much higher 3-5 x higher with a greater potential for volume depletion.

Extracorporeal Photopheresis

1. Primary indication: Cutaneous T-cell lymphoma (FDA only), acute or chronic refractory GVHD
2. Procedure: Procedure involves collection of buffy coats (mononuclear cells) in either a semi-continuous or discontinuous fashion using a cell separator specifically approved for this purpose, exposure of the cells to 8-methoxypsoralen, and subsequent UV-A irradiation, with return of the treated cells back to the patient. Anticoagulation uses either heparin or citrate depending on patient condition. Procedure time depends on the instrument used but can range from 1.5 hours to 5 hours. Because there is a sensor that detects the presence of the WBC layer, interferences such as lipids may interfere in detection of this layer, so that intralipids in the TPN must be discontinued 24 hours prior to procedure.

3. Cautions: Because of the potential for heparin use and large fluid shifts, procedure is not recommended for use on day of dialysis, and if the patient is hemodynamically unstable. Generally, platelet counts should be > 50 K/microL (may be lowered to > 30 K/microL if using citrate anticoagulation) and Hct>30% (may be lower depending on predicted extracorporeal volume) on day of procedure.
4. Complications:
   a. Most common: Citrate toxicity, hypotension/hypertension, medication and/or endogenous antibody removal, transfusion reactions (if replacement with blood products is required or if RBC prime is need), and line occlusion/problems,
   b. Photosensitivity is of concern with ECP patients who need to protect eyes and skin 24 hours post procedure. Complications depend on the type of procedure being performed.
5. Monitoring:
   a. Vital signs are measured every 5 minutes for the first 30 minutes, then every 15 minutes thereafter. Ionized calcium measurements are needed before, during (may be optional if previous procedures have had stable iCa), and after to monitor for hypocalcemia.
   b. Often a calcium drip will be used for hypocalcemia prophylaxis. Electrolyte abnormalities should be corrected prior to start of procedure.
   c. Other laboratory monitoring includes post procedure CBC to monitor for cellular depletion and/or target completion (e.g. appropriate target hematocrit in the case of RBC exchange, hemoglobin S % (to determine if appropriate percentage of sickle cells has been removed). There may be other testing required depending on patient disease or condition.
6. Other considerations:
   a. Minimal required information for starting the procedure includes patient weight and height, and pre procedure CBC. This is to determine blood volume of the patient and target parameters depending on procedure type.
   b. Patients may need RBC prime if extracorporeal volume is greater than 10% (generally ≤ 25 kg)
   c. Patients may need premedications, especially if blood products are part of the replacement fluids or prime.
   d. Access: Generally, for patients <10 kg, 10-20 kg, and >20 kg, 7 Fr, 8 Fr, and 9 French (or greater with the larger kids) double lumen medcomp catheter placed in the IJ or femoral vein will be suitable. Access being used for hemodialysis may also be used with permission from nephrology. Other access used includes: Cook Turbo Flow 5 Fr single lumen for extracorporeal photopheresis and vortex port (double lumen titanium port accessed with special 16 ga**noncoring** needles required).
   e. Transfusion Medicine consultation is required for consideration of any therapeutic apheresis procedure prior to line placement.

PRO-COAGULANTS

Recombinant Factor VIIa (NovoSeven)*:* activates the extrinsic pathway of the coagulation cascade

1. Indications: Initially developed for acute bleeding in hemophilia, potential use in DIC and life-threatening hemorrhagic shock.
2. Side Effects: Can worsen DIC and potentiate active thrombus formation. ONLY for use with Hematology approval.

Vitamin K

1. Indications: liver disease, nutritional deficiency, prolonged INR
2. Side Effects: IV formulations carry a risk of anaphylaxis, especially if administered quickly.

MASSIVE TRANSFUSION PROTOCOL

The Massive Blood Transfusion Process is a systematic process to obtain large amounts of blood products – activate anytime you may need more than 2 units of uncross-matched blood quickly.

1. Packet with all the necessary paperwork in the bottom drawer of each code cart, at all main desks in the units, on each code cart, outside the trauma bay in the ER, and in each OR room
2. Four key roles in the Massive Blood Transfusion Process: **Treating Physician,** Charge Nurse, Bedside Nurse, Transporter. Each role has a Checklist and necessary paperwork in the packet **– PHYSICIANS IN YELLOW**!
3. Activate the Massive Blood Transfusion Protocol by calling Blood Bank at x5347
   a. Know Patient name, MRN, Wt, Age, Gender, Hospital location, your ASCOM #
   b. Complete Emergency Release of Blood Products form in the packet → give to Charge RN
   c. Order: Type and Screen, CBC, PT/PTT, Fibrinogen, BMP, Blood Gas
   d. Order Blood Products in Cerner – Follow instructions in MBTP packet!

ONCOLOGIC EMERGENCIES

Tumor Lysis Syndrome

1. Etiology: Rapid lysis of tumor cells, releasing intracellular contents into the bloodstream, spontaneous or after starting treatment → hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia
2. Presentation: lethargy, nausea, vomiting and renal colic, CNS dysfunction, renal failure due to uric acid and calcium phosphate crystals
3. Treatment
   a. Allopurinol blocks the production of uric acid by inhibiting xanthine oxidase
   b. Rasburicase -recombinant urateoxidase, catalyzes conversion of UA to allantoin.
   c. Promote solubility by alkalinizing the urine using NaBicarb in iv fluid to pH > 7.0
   d. Decrease concentration by volume expansion (2x maintenance fluids)
   e. Frequent monitoring and replacement of lytes (Mg, Ca)

Hyperleukocytosis

1. Etiology: Leukocyte count > 100,000, seen in acute leukemia (AML > ALL), polycythemia vera
2. Presentation: Hyperviscosity→Sludging of viscous blood →causes visual disturbance, mental status changes, respiratory distress, hypoxia, acute renal failure
3. Treatment
   a. Avoid diuretics and PRBC transfusion
   b. Correct coagulopathy and thrombocytopenia if seen
   c. Hydration, alkalinization, Allopurinol as above
   d. Exchange transfusion vs. leukapheresis
   e. Treat the disease
   f. Treat the Tumor Lysis if also present

Spinal Cord Compression

1. Rare in children
2. Etiology: most often epidural compression from extension of paravertebral tumor through the intervertebral foramina or extension of tumor in the vertebral column.
3. Presentation: back pain (localized/radicular) in 80%, weakness, sensory loss, change in bowel/bladder function.
4. Prognosis - based on duration and level of disability at presentation.
5. Diagnosis – neuro exam, emergent MRI of spine
6. Treatment – Decadron, may be amendable to emergent radiotherapy or chemotherapy (discuss with Onc/Neurosurgery), surgery to decompress the spine.

Superior Vena Cava Syndrome / Mediastinal Mass

1. Due to SVC clot or compression of SVC usually from mediastinal mass or mediastinal mets
2. Presentation: respiratory distress, often positional, anxiety/air hunger, periorbital or facial edema, dizziness, syncope, visual discoloration of head and neck

BONE MARROW TRANSPLANT

STANDARD LABS

Allogeneic BMT patients: matched sibling bone marrow, peripheral blood, or cord blood

1. Weekly infectious surveillance labs drawn from the blood – drawn **Mondays** as the PCR lab runs the PCRs on Tues/Fri
   a. Adenovirus PCR
   b. CMV PCR
   c. Aspergillus Galactomannan
   d. Beta D Glucan/Fungitell
   e. EBV PCR
2. Other labs that should be obtained at least weekly: IgG, LFTs

BLOOD PRODUCTS TRANSFUSIONS

1. Most patients are transfused for Hemoglobin < 7 and Platelets < 10,000
2. Some patients also require pre-medications (Tylenol, Benadryl, Hydrocortisone) – which will be specified in BMT note
3. Can vary depending on the current clinical scenario (supplemental O2, bleeding, etc.)
4. BMT for Sickle Cell Disease require higher hemoglobin (>9) and platelet thresholds (>50,000) due to intracranial vasculopathy putting them at an increased risk for bleeding.
5. Clinical symptoms are always more important than the number and hemorrhage can be a serious concern in our patients. General rule of thumb is if there is bleeding, transfuse platelets even before seeing the result of the count.

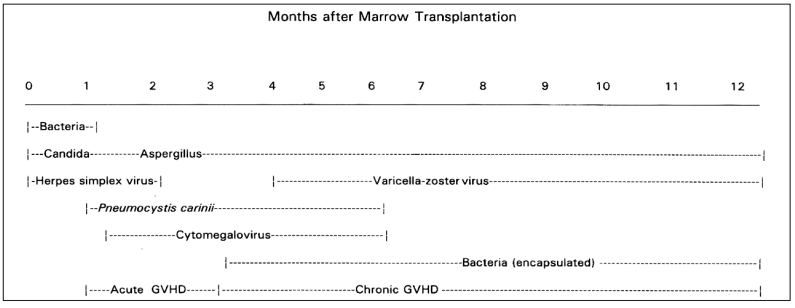
TRANSPLANT SPECIFIC MEDS AND MONITORING:

1. Calcineurin inhibitors
2. Cyclosporine, Tacrolimus
3. Standard medications for almost all allogeneic BMT
4. Graft versus host disease prophylaxis
5. Side effects – hypertension and hypomagnesemia
6. Levels usually followed MWF, more as
7. Have narrow therapeutic ranges
8. Levels are drawn as trough and before the morning dose is given
9. Medication should be scheduled/given at 10a/10p in order to keep steady state levels and to allow ease of monitoring

FEVER/INFECTION

1. Fever is defined as the following:
   a. 38.3 C by mouth once **OR** 38C that persists for one hour
   b. 37.8 C axillary one **OR** 37.5 C that persists for one hour (axillary temps are 0.5 less than oral)
2. Any BMT patient with fever or change in clinical status should be
   a. Vancomycin typically although may vary based on previous resistance patterns
   b. Blood cultures drawn (anaerobic, aerobic, and fungal from ALL lumens/lines).

3. Remember some BMP patients are on steroids and therefore may be unable to mount a fever.



Infection is ALWAYS on the differential!

TRANSPLANT – RELATED COMPLICATIONS

Infection (see above)

Graft versus Host Disease (GVHD)

1. Etiology: The new cells recognize the recipient's (patient's) body as foreign.
2. Presentation: diffuse rash, clinically significant diarrhea (>10mL/kg/day or 500 mL) with/without abdominal pain, and liver dysfunction (elevations in bilirubin).
3. Treatment is often with steroids.
4. **It is important to closely monitor stool output in all of our patients for this reason.**

Engraftment Syndrome

1. Occurs near the time of count recovery.
2. Presentation: fever (without an infection), an erythematous rash, and signs of capillary leak syndrome.
3. Treatment: strict fluid management and steroids.
4. Imperative to rule out infection prior to starting steroids.

Diffuse Alveolar Hemorrhage (DAH)

1. Etiology: can occur as a result of infection or damage from chemotherapy.
2. Presentation: bloody discharge from ET tube, hemoptysis, opacification on CXR
3. Treatment: blood and platelet transfusions, consider treating with high dose steroids, IVIG, and Novo7.

Idiopathic Pneumonia Syndrome (IPS)

1. Etiology is unknown but thought to be secondary to damage caused by inflammatory cytokines and is often fatal to those affected (50-75%). Infection needs to be ruled out by BAL if possible.

2. Presentation: diffuse, non-infectious lung injury that occurs acutely (typically before day +120) after transplant
3. Treatment is supportive. May consider using steroids to decrease inflammation, and Etanercept.

Veno-Occlusive Disease (VOD) or Sinusoidal Obstructive Syndrome (SOS)

1. Etiology: secondary to chemotherapy and damage to small venules of liver.
2. Presentation: Rising levels of bilirubin and other liver dysfunction, and fluid retention.
3. Treatment: respiratory support if becomes severe enough, strict fluid management and sometimes Defibrotide (on study medication).

Posterior Reversible Encephalopathy Syndrome (PRES)

1. May occur at any point after transplant
2. Etiology: secondary to tacrolimus or cyclosporine with the combination of steroids (both causing hypertension).
3. Presentation: visual disturbances, seizures, headaches, or altered mental status.
4. Treatment: tight blood pressure control, anti-epileptics and transitioning to a different calcineurin inhibitor.

Mucositis

1. The time period is from day 0 until the patient recovers their counts.
2. Etiology: disruption of the mucosal lining from mouth to anus as a result of the chemotherapy and/or total body irradiation that was given to prep the patient for the bone marrow transplant.
3. Presentation: Pain and denudement of mucosal membranes
4. Treatment: pain control, often with a PCA.

TRAUMA

Epidemiology: 10,000 children die from injuries each year, injury is the leading cause of death and disability in children

Evaluation - ATLS® sequence

1. Primary survey and resuscitation
2. Secondary survey – goal is to identify other injuries
3. Treat Pain

Key things to consider in children

1. Less fat, are more elastic so more likely to have multisystem injury
2. Larger BSA to BMI so higher risk for hypothermia and insensible fluid loss
3. Can maintain near normal BPs in face of 30% blood volume loss
4. Children have better outcomes when cared for in pediatric trauma centers or in adult centers that have qualifications to care for children.

Primary Survey - identify life-threatening conditions**,** should take only a few minutes**,** starts at the scene

1. C-A-B sequence
   a. Check Pulse → Start compressions
   b. Airway: determine presences of blood, stomach contents, edema, foreign bodies, facial trauma; altered mental status may lead to stridor or inability to maintain patency of airway;
   c. Breathing: may be impaired by neuro status or airway obstruction, assess respiratory distress, evaluation for asymmetric breath sounds due to pneumothorax or malpositioned ET tube from the field, gross chest wall deformity (flail chest, sucking chest wound). Evaluated oxygen saturation, consider carbon monoxide poisoning that may have falsely normal or high SpO2. Always use inline stabilization when intubating, capnography to ensure no dislodgement of tube and adequacy of circulation, always evaluation appropriate size and position of invasive airway placed in the field or elsewhere.
   d. Circulation: Control major hemorrhage (i.e. pelvic fracture)
2. Disability
   a. Quick evaluation of neurologic status
   b. Cervical Spine Injuries – assume the patient has an injury; while rare (incident is < 2%), can have devastating consequences, 30-40% of children have a spinal cord injury without radiological abnormality (SCIWORA), may choose not to do cervical spine imaging if: no midline cervical tenderness, no focal neurologic deficit, normal alertness, no intoxication, and no painful, distracting injury. Initial imaging is AP view, cross-table lateral and open-mouth view, followed by either clinically clearing the patient or MRI at 72 hrs. post injury
   c. Other imaging – CT imaging for focused assessment

Secondary Survey

1. Focused Hx "SAMPLE"
   a. Signs/Symptoms
   b. Allergies
   c. Medications

d. PMH
e. Last oral intake
f. Events leading to injury

2. Physical assessment – may be more rapid or focused on more unstable patients
   a. Vital Signs
   b. Neck – deformity/pain suggestive of C-Spine injury, tracheal deviation
   c. Head – lacs, edema, deformity of skull, injury to eye or foreign bodies, pupillary exam, evaluate ears and noses for clear fluid that may be CSF or blood
   d. Chest – lacs, bruising, foreign bodies, symmetry of chest movement
   e. Abdomen – lacs, bruising, foreign bodies, tenderness, rigidity
   f. Spine – point tenderness, deformity, "step offs"
   g. Pelvis – press on lateral wings of pelvis to eval for deformity or pain
   h. Genitals – lacerations, impaled objects, blood at the urethral meatus and rectum, priapism in males, rectal tone
   i. Extremities – deformities, lacs, bruising, edema, pulses, cap refill, motor function

More on Specific Traumatic Injuries

1. Intrathoracic Injuries
   a. Most injuries in children suffer from are blunt trauma (car and bicycle injuries)
   b. Types: Pulmonary contusions, pneumothoraces, tracheal disruption, cardiac injuries, flail chest or rib fractures
      i. Flail Chest – segment of one or more ribs fractures in two points → paradoxical movement with respirations, caused by high-energy mechanisms, rare in children due to chest wall elasticity, likely associated with a pulmonary contusion that causes hypoxemia and VQ mismatch
      ii. Hemothorax – blood in the pleural space due to rupture of intercostal vessels or bone bleeding from fractured rib, can lead to hemorrhagic shock, dullness on percussion or asymmetric auscultation, treat with chest tube and transfusion as needed
   c. Injuries may not be obvious due to compliance of the rib cage but can cause profound hypoxemia
   d. Commotio cordis – sudden impact to the ant chest wall may cause the heart to stop or induce an arrhythmia
2. Intra-abdominal injuries
   a. **Beware!** Most common type of injury to go unrecognized and be fatal! Distracting injuries may contribute to this
   b. 1/3 of children with major trauma will have significant intra-peritoneal injuries.
   c. Consider NG tube and Foley decompressions
   d. Evaluate for abrasions that may be clues (i.e. seat belt sign. The stomach and bladder need to be decompressed
   e. Evaluate typically by CT
3. Near Drowning
   a. 15,000-70,000 per year in US
   b. < 1 yr olds 55% occur in bathtubs, 1-4 year olds , 56% in pools, older children, 63% in freshwater
   c. Hypoxemia due to apnea and breatholding, laryngospasm, and pulmonary aspiration with lung injury → may lead to ARDS

d. ARDS may occur after an initial period of apparent recovery
e. Causes of death associated with ARDS– sepsis, multiorgan failure, air leak
f. Treatment: bystander CPR, treat hypothermia!, respiratory support, no indication for steroids or antibiotics
g. Prognosis poor for submersion > 25 mins, delay in CPR, Resuscitation > 25 mins, severe acidosis, arrival to an ED still pulseless, elevated blood sugar on arrival, dilated and fixed pupils on arrival, abnormal initial CT, initial GCS < 5

Exposure – children are at high risk of hypothermia which can lead to arrhythmias, acidosis, and coagulopathy, ensure adequate monitoring and warming strategies for temp < 36 such as increasing room temperature, removal of wet cloths, use hat and warm blankets, warmed fluids and convection air blanket

TOXIDROMES

Resources: Online Formulary , American Association of Poison Control Centers: 1 (800) 222-1222 (www.aapcc.org); National Capital Poison Control (www.poison.org) Cornell University Poisonous Plants (www.ansci.cornell.edu/plants.html)

By Agent			
Agent	**Examples**	**Symptoms**	**Treatment**
Acetaminophen	Tylenol Paracetamol	Nausea, vomiting, pallor, delayed hepatic failure	Use Rumack-Matthews Nomogram for use of N-Acetylcystine
Alcohols	Methanol Ethylene Glycol Ethanol	Intoxication/Stupor, Coma, LARGE OSMOLAR GAP (>10), METABOLIC ACIDOSIS, HYPOTENSION, HYPOTHERMIA, ABRUPT HYPOGLYCEMIA, Blindness- Methanol, Urinary Crystals, Renal Failure- Ethylene Glycol, PROLONGED QT Beware: May mask other ingestions, may be due to over-the-counter medications like mouthwashes	NO GI Contamination Supportive Care- Oxygen/IVF IV Dextrose for Hypoglycemia Fomepizol for toxic ingestions Hemodialysis
Alpha 2 Adrenergic Agonist	Clonidine	Mild HTN and tachycardia followed by HYPOTENSION and Bradycardia, Mydriasis, CNS Depression	IVF Supportive Care
Amphetamines	Amphetamine Salts Dextroamphetamines ADHD Medications Pseudoephedrine Cough/Cold formulations, Ma Huang, Meth, "Ice"	Tachycardia, HTN, Hyperthermia, N/V, Abdominal Pain, Diaphoresis, Anorexia, Tremors, MYDRIASIS, Tachypnea, Delirium, Psychosis	GI Decontamination Benzodiazepines for Seizures Cooling blanket
Anticholinergic	Benadryl Promethazine Hydroxyzine Scopolamine	MYDRIASIS, ABSENT Sweating, Abdominal Ileus, Tachycardia, Delirium, Disorientation, Ataxia, Hallucinations, Seizures, Extrapyramidal Sx, Hyperexia, Urinary Retention, Dry Flushed Skin, Prolonged QRS **Red As A Beet, Dry As A Bone, Blind As A Bat, Mad As A Hatter, Full As A Flask**	Benzodiazepines for Seizures - Then Barbiturates IV Bicarbonate for Prolonged QTc CK Levels to R/O Rhabdomyolysis Physostigmine for SVT or narrow QRS Lidocaine for Ventricular Arrhythmias

Cholinergic	Organophosphate Pesticides	**SLUDGE: Salivation, Lacrimation, Urination, Defecation/Diarrhea, Gastric Cramping**	Hemodialysis Ineffective
Beta Blockers	Atenolol, Propranolol, Labetalol	HYPOGLYCEMIA, HYPOTENSION, BRADYCARDIA	Glucagon NE Lipids
Calcium Channel Blockers	Verapamil, Amlodipine, Nifedepine, Nicardipine	CLASSIC HYPOTENSION AND BRADYCARDIA, Hyperglycemia, Lactic Acidosis with increased K+, AV Block	Atropine for Bradycardia Ca+ CHL/Gluconate reverses block NE for Hypotension Glucagon Promotes CA Influx Lipids Trap drug in Bloodstream Insulin For Hyperglycemia Whole Bowel Irrigation
Camphor	Vicks Vapo-Rub Campo-Phenique	Abrupt seizures, emesis, confusion, agitation	Immediate Dilution/Irrigation NO CHARCOL OR EMESIS
Caustics	Alkaline Corrosives Acids Drain/Oven/ Toilet Bowel Cleaners Rust Removers	Pain, dysphagia, drooling, vomiting, hematemesis, oropharyngeal burns, ocular burns, wheezing, stridor	Immediate Dilution/Irrigation NO CHARCOL OR EMESIS H2 Blockers, steroids, antibiotics (reduce stricture formation)
Cocaine	Cocaine, Crack	May be taken IV, inhaled, intranasally, smoked Irritability, tremor, hyperreflexia, diaphoresis, dilated pupils, flushing, hypertensive crisis, dysrhythmias, seizures, coma, coronary vasospasm and cardiovascular collapse	Supportive care
Cyclic Antidepressants	Amitriptyline	Lethargy, Coma, Seizures, Hypotension, Ventricular Arrhythmias, Prolonged QRS	IV Bicarbonate Vasopressors: NE/EPI Benzodiazepines then Barbiturates
Hallucinogens	LSD, Mescaline, Psilocybin (magic mushrooms), Ecstasy	Ingested, visual hallucinations, distortions of time, psychosis	Supportive care
Hydrocarbons	Gasoline, Kerosene, Lamp Oil, Furniture Polish	Coughing, choking, tachypnea, wheezing, N/V	Oxygen, respiratory support, may require intubation

Inhalants	Airplane glue, gasoline, spray paint, aerosols, cleaning fluids	Inhaled often from plastic bags or rags → rapidly absorbed in lungs, altered mental status, pneumonitis, hepatic, renal, hematologic damage; gasoline may cause lead toxicity	Respiratory support, supportive care
Iron	MV, Prenatal Vitamin	N/V, Hematemesis, hemorrhagic diarrhea, shock, Hypotension, coma, metabolic acidosis, hepatitis, arrhythmias	Chelating Agent- Desferoxime Hemodialysis Whole Bowel irrigation
Marijuana	THC (delta-9-tetrahydrocannabinol)	Euphoriant Withdrawal syndrome – flu-like illness	Trazadone for insomnia
Narcotics	Morphine Tramadol Meperidine Codeine Oxycodone Heroin Fentanyl Propoxyphene	Confusion, Lethargy, Ataxia, Coma, Respiratory Depression, HYPOTENSION, HYPOTHERMIA, **MIOSIS,** Constipation, Bradycardia, Pulmonary Edema, Seizures- with Meperidine and Tramadol	Narcan- May need frequent dosing since duration is 20-60 minutes Toxicology Screen Benzodiazepines for Seizures IV Bicarbonate for Widened QRS
Salicylates	Aspirin	N/V, Tinnitus, Tachypnea, Confusion, Fever, early alkalosis followed by metabolic acidosis Beware: Salicylates are found in anti-diarrheal products	Charcoal?, N -Acetylcystine
Sulfonylureas	Sulfonylurea	HYPOGLYCEMIA, HYPOKALEMIA, lethargy, dizziness, coma	Glucose- D25 or D50 Octreotide

By Signs and Symptoms	
Hypothermia	Opiates, Sedatives, Alcohols, Phenothiazines, Carbamazepine, Barbiturates
Hyperthermia	Salicylates, Anticholinergics, Amphetamines, Antihistamines, Aspirin, Atropine, Cocaine, Iron, Thyroid Hormone, PCP, Phenothiazines
Tachycardia	Anticholinergics, Antihistamines, Sympathomimetics, TCA's and SSRI's
Bradycardia	Beta-Blockers, CCB's, Clonidine- Alpha 2 Agonists
Tachypnea	Direct pulmonary insult or non-cardiogenic pulmonary edema or as a compensatory mechanism with metabolic acidosis
Respiratory Depression	Clonidine, Opioids, Sedative-Hypnotics
Hypertension	Cocaine, Sympathomimetics/Amphetamines, Anticholinergics, TCA
Hypotension	TCA's, BB's, CCB's, Arsenic, Carbon Monoxide, Clonidine, Nitrates, Cyanide,

	Colchicine, Opiates, Barbiturates
Hypoglycemia	BB's, Sulfonyureas, Alcohols
Hyperglycemia	CCB's
Coma / Altered Mental Status	Alcohols, Anticonvulsants, Barbiturates, Benzos, Opiates, Antihistamines, Carbon Monoxide, Chloral Hydrate, Clonidine, Cyanide, TCS, GHB (Gamma-Hydroxybutyrate), hydrocarbons, hypoglycemic, inhalants, anticonvulsants, lithium, inhalants
Seizures	Amphetamines, Atropine, Camphor, Carbon Monoxide, Cocaine, Gyromitra mushrooms, INH, Lead, lithium Nicotine, Pesticides, PCP, Propoxyphene, Salicylates, TCAs
Mydriasis (Dilated Pupils)	Amphetamines, Anti-Cholinergic, Antihistamines, Atropine, Cocaine, Tricyclic AD, MDMA, LSD
Miosis (Constricted Pupils)	Narcotics, Alcohols, Organophosphates, Phenothiazines

Initial Evaluation and Treatment		
Initial Labs CBC/CMP/MAG/PO4 LFT's/PT/PTT/INR Ammonia U/A Ethanol level Salicylate Level Aspirin Level POC Glucose Check Blood Gas Serum Drug Screen Urine Drug Screen Serum Osm Level	**Blood Gas**--→ Check For Anion Gap • Increased > 15: Formaldehyde, Iron, INH, Methanol, Paraldehyde, or Salicylate, Ethylene Glycol, Ethanol • Decreased: Chloride, Bromide or Lithium **Calculate Osmolar Gap** **Serum Osmolality =** 2x NA + BUN+ Glucose/ 18 Osmolar Gap = Calculated Osm – measured Osm Normal -3 to 10: **Elevated: Methanol, Ethanol, Ethylene Glycol, Acetone and Isopropranolol** **EKG:** Check For Prolonged QRS > 100 **X-rays:** Radiopaque: Chloral hydrate, cocaine packets, heavy metals, iron, Calcium Carbonate, phenothiazines, slow release enteric tablets, Iodine, K+ Compounds	**Initial Treatments** **Charcoal:** **Effective For:** Carbamazepine, barbiturates, Quinine, Theophylline, Salicylates, Digoxin, Phenytoin **NOT Effective For:** Camphor, Caustics, Hydrocarbons, Alcohols, Metals, Pesticides **Whole Bowel Irrigation:** Using polyethylene glycol May be effective for systained release or enteric coated drugs, packets of illicit drugs **Consider Antidotes**

ANTIDOTES/REVERSALS		
Toxin/Med	**Antidote**	**Dose**
Narcotics (opioids) Clonidine	Naloxone	Partial Reversal: 0.01 mg/kg IV/IO/IM; Total Reversal: 0.1 mg/kg IV/IO/IM (max: 2 mg) Duration of effect 20-60 minutes: May need frequent dosing
Benzodiazepines	Flumazenil	0.01 mg/kg – 0.02 mg/kg IV (max: 0.2 mg)
Alcohols	Fomepizol (Dialysis!)	Patients **NOT requiring** dialysis: Loading Dose 15 mg/kg, then 10 mg/kg Q12 x 4 doses then 15mg/kg until ethylene glycol or methanol levels < 20 mg/dl Patients **requiring** hemodialysis: dosing depends on many dialysis and drug administration timing. See LexiComp formulary for further information. Provides 12 hours of alcohol inhibition

Sulfonylureas	Dextrose Octreotide	Dextrose: 0.5 - 1 gram/kg Octreotide: 1-1.5 mcg/kg/dose SQ; repeat in 6-12 hours as needed
Acetaminophen	N - Acetylcysteine	Loading Dose 150 mg/kg IV over 1 hour, followed by 50 mg/kg IV over 4 hours, then 100 mg/kg IV over 16 hours (may continue beyond 21 hours if clinically indicated)
Tricyclic Antidepressants	Sodium Bicarbonate (for prolonged QRS complex)	1 mEq/kg IV (max: 50 mEq) IV
Calcium channel blockers (CCB)	Calcium Chloride Glucagon	Calcium chloride: 20 mg/kg/dose IV (max: 1000 mg) every 10 minutes until a response is seen Glucagon: Loading Dose 0.03-0.15 mg/kg IV (max :10 mg), then 0.07 mg/kg/hour (max: 5 mg/hr)
Beta Blockers	Glucagon Atropine, Isoproterenol	Glucagon: Loading Dose 0.03-0.15mg/kg IV (max: 10 mg), then 0.07 mg/kg/hour (max: 5 mg/hr)
Pure Anticholinergic	Physostigmine	0.01-0.03 mg/kg IV (Max 2 mg) administer slowly over 5 minutes; may repeat after 15-20 minutes to a maximum total dose of 2 mg
Organophosphates	Atropine Pralidoxime	Atropine: 0.1 mg/kg every 5-10 minutes until secretions subside Initial Dose 20 – 50 mg/kg (max: 2000 mg/dose) IV; then maintenance infusion of 10-20 mg/kg/hour
Heparin	Protamine	Dosing is determined by the most recent dosage of heparin/LMWH and time since last heparin dose. See Lexicomp formulary for further information.
Coumadin	Vitamin K (phytonadione)	2 – 5 mg IV/Subcutaneous
Cyanide	Amyl nitrate, Sodium Nitrate, Sodium thiosulfate (Cyanokit)	Call Poison Control
Digoxin	"Digibind" – Digoxin- Specific Antibioties	Call Poison Control
Lead	Edetate Calcium (EDTA) British Anti-Lewisite (BAL) Pencillamine Succimer 2,3 dimercaptosuccinic acid (DMSA)	Call Poison Control
Mercury	BAL	5 mg/kg IM ASAP
Nitrites	Methylene Blue (for	1-2 mg/kg IV, repeat every 30-60 min, treat for levels > 30%

	methemoglobinemia)	
Iron	Deferoxamine	Initial dose 20 mg/kg (max: 1000 mg) IV, then 10 mg/kg (max: 500 mg) over 4 hour intervals for 2 doses
Insulin, Oral Hypoglycemics	Glucose (PO) or dextrose (IV)	Dextrose: 0.5 - 1 gram/kg

MALIGNANT HYPERTHERMIA

Clinical Diagnosis: associated with anesthesia, cocaine, neuroleptic meds, Duchenne muscular dystrophy or other myopathies; rapid rise in body temperature, muscle rigidity, spasms, tachycardia, acidosis, hypoxemia, myoglobinuria, elevated CK level, hyperkalemia, hypermetabolism, increased CO2 production

Causes: 1/15,000 children, some have an autosomal dominate inheritance of MH vulnerability (mutation of the RYR1 gene that codes for voltage-sensitive Ca^{2+} channel in the sarcoplasmic reticulum)

Treatment: remove precipitating agents, Dantrolene, oxygen, treat hyperkalemia, rapid cooling, alkalinize the urine, hydrate

NEUROCRITICAL CARE

(*Please also see Children's Intranet for Order sets/Flow sheets on: Acute Ischemic Stroke, Sinovenous Thrombosis (CSVT), Diabetes Insipidus, Severe TBI, Encephalitis, Refractory Status Epilepticus, and Pentobarbital Infusion and Weaning)

COMA

Etiology: metabolic derangements, abnormal electrolytes or glucose, azotemia, toxins, hepatic dysfunction, hydrocephalus, infections, psychiatric disorders, seizures, tumors, vasculopathies or cerebral vascular accidents, traumatic brain injury, increased ICP

ELEVATED INTRACRANIAL PRESSURE (ICP)

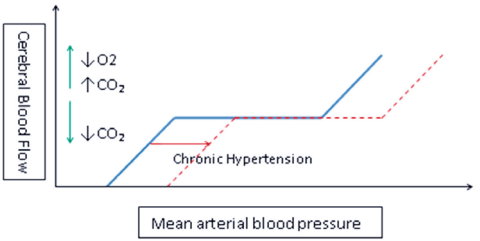
The intracranial vault is a closed compartment composed of blood, brain, and CSF. If one compartment enlarges, the others must be compressed and eventually intracranial pressure will rise. Once ICP rises above mean arterial blood pressure, blood flow is compromised to the brain according to this relationship:

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

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

Normal cerebral blood flow is maintained over a range of MAPs by **cerebral auto-regulation**:

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



Causes of Elevated ICP:

1. Cerebral edema:
   a. Vasogenic: capillary leak from the brain endothelium as seen in SIRS, meningitis, ICH, CVA, lead intoxication
   b. Cytotoxic: the cells in the brain themselves swell from cellular energy failure (ATP-production) as seen in HIE, DAI, osmolar injury.
2. Interstitial: obstructed CSF = ↑water content of the periventricular white matter.
   a. Intracranial lesions: ICH, tumor, abscess, AVM, focal edema from a contusion or CHI
   b. 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.

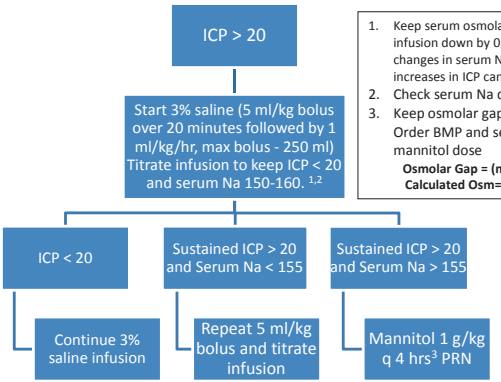
Cushing's triad: brainstem's attempt to maintain perfusion = increased MAP to maintain CPP causes reflexive bradycardia, hyper or hypoventilation possible.

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

Treatment:

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.
   a. Hyperventilation is only warranted in impending herniation.
   b. NEVER drop PCO_2 <30-35mmHg or CBF will be severely compromised leading to more ischemia.
   c. Only a temporizing measure (works for <24 hours) and can worsen outcomes if persistent.

3. Hyperosmolar therapy: Neither 3% NS nor mannitol cross the BBB, therefore, they have osmotic properties which lower cerebral blood volume and free water content of healthy brain cells.
   a. Mannitol: 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.
   b. Hypertonic saline (3%): unlike mannitol, 3% NS also restores normal cellular resting membrane potential and cell volume, inhibits inflammation, and ↑cardiac output.



1. Keep serum osmolality less than 360. If Na >165, titrate infusion down by 0.25 ml/kg/hr every 12 hours. Avoid changes in serum Na >10 mEq/24 hrs. Note that increases in ICP can occur with decreasing serum Na.
2. Check serum Na q8hrs
3. Keep osmolar gap <20 prior to mannitol infusion. Order BMP and serum osmolality 2hrs after mannitol dose

Osmolar Gap = (measured osm)-(calculated osm)
Calculated Osm= 2 (Na) + BUN/2.8 + Glucose/18

Please Note:

1. **Patient serum Na should be <160 mEq/L and serum osmolality <360 mOsm/kg**
2. **Notify neurosurgical service and CCM attending when initiating hypertonic saline and when initiating mannitol**
3. **Use of hypertonic saline should be limited to ICP >20 mmHg or symptomatic hyponatremia**

4. Neurosurgical intervention:
   a. ICP monitor: Intraparenchymal monitor or externalize ventricular drain (EVD), directly measure the patient's ICP to allow titration to goal CPP.
   b. EVD: allows CSF removal if as well as measure ICP if needed. Is set at the bedside at a specified height to deliver a certain pressure (negative or positive) to the ventricle in cmH_2O. Zeroed at the level of the tragus.
   c. Decompressive craniectomy: effective in recoverable brain injury before significant secondary injury has occurred (only shown in case studies)

TRAUMATIC BRAIN INJURY (TBI)

Glascow Coma Scale

Score	Eyes	Verbal	Motor
6			Obeys Commands
5		Oriented, converses normally	Localizes to painful stimuli
4	Opens eyes spontaneously	Confused, disoriented	Withdrawals to painful stimuli
3	Opens eyes in response to voice	Utters inappropriate words	Flexion (decorticate) posturing to painful stimuli
2	Opens eyes in response to painful stimuli	Incomprehensible sounds	Extension (decerebrate) posturing to painful stimuli
1	Does not open eyes	Makes no sounds	Makes no movements

Pediatric Glascow Coma Scale (<2 yo)

Score	Eyes	Verbal	Motor
6			Infant moves spontaneously and purposefully
5			Infant withdrawals from touch
4	Opens eyes spontaneously	Cries, irritable	Infant withdrawals from pain
3	Opens eyes in response to voice	Cries to pain	Flexion (decorticate) posturing to painful stimuli
2	Opens eyes in response to painful stimuli	Moans to pain	Extension (decerebrate) posturing to painful stimuli
1	Does not open eyes	Makes no sounds	Makes no movements

1. The GCS exam should be done **AFTER** airway, breathing and circulation have been assessed and corrected (and ideally before sedation and paralytics are given)
   GCS 13-15- **mild traumatic brain injury**
   GCS 9-12- **moderate traumatic brain injury**, close monitoring in PICU, do not intubate unless clinically indicated by respiratory exam
   GCS 3-8- **severe traumatic brain injury**, in presence of abnormal CT scan intubation and placement of ICP monitor as soon as possible is indicated
2. If CT scan is normal, consider medication effect as etiology of depressed mentation or earlier repeat of CT to follow for evolving lesions.
3. Skull fractures – in child < 1, consider non-accidental trauma

4. Epidural hematoma – caused by injury of penetrating arteries, blood collects in epidural space, can lead to rapid onset coma and herniation but this may be delayed.
5. Subdural hematoma – due to lacerations of brain parenchyma and bridging veins, occupies space between dura and brain, poorer prognosis due to brain injury
6. Spinal Cord Trauma
    a. Causes: May occur due to bony fragments causing cord injury or transection, contusions due to direct impact or contra coup, atlanto-axial dislocation, disrupted blood supply which may cause infarction
    b. Diagnosis: Neurologic deficit, neurogenic shock, must be vigilant and low index of suspicion because many patient s have distracting injuries.

    c. Treatment: surgical stabilization, steroids are controversial (some benefit in adults), treat spinal shock, ventilator management to ensure lowest possible intrathoracic pressure to allow for venous drainage and close attention to arterial pH and CO2 due to effects on cerebral and spinal blood flow

Children's National Medical Center

Pediatric Severe TBI Acute Phase Management Flowsheet

Sept 2011

Check

- Patient position (head neutral, HOB at 30°)
- Equipment functioning properly
- No recent interventions (respiratory, nursing)
- Exclude seizure activity

Seizures

- Prophylaxis:
  - Load– Levetiracetam (Keppra) 20 mg/kg IV
  - Maintenance– Levetiracetam 10 mg/kg/dose IV Q12 hrs
- Treatment
  - notify Neurology service immediately
  - Versed 0.1 mg/kg bolus for acute control
  - Load fosphenytoin 20 mg PE/kg

Fluid Therapy, Vasopressors

- Maintain CVP 5 to 10 mmHg (NS for fluid resuscitation)
- If <6 mos, use D5W/NS for maintenance
- If >6 mos use NS for maintenance
- Maintain serum glucose between 80-150
- Maintain Hgb >8 g/dl
- Once volume loaded, use inotropic/vasopressor
  - 1st line– Dopamine
  - Once Dopamine >10 mcg/kg/min, start Norepinephrine (warm ext) or Epinephrine (cool extremeties)

Sedation and Analgesia

- Versed
- Morphine or Fentanyl
- Avoid hypotension secondary to sedative/analgesic agents
- Consider NMB agents for ICP control– see NMB algorithm

CSF Drainage Options

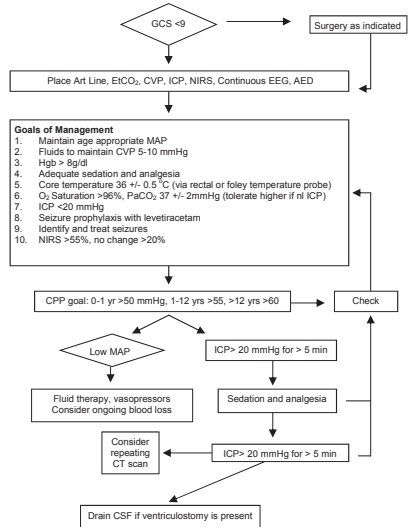
- Initial settings and changes to drainage level per Neurosurgical service
- Drain CSF for 15 minutes, then re-evaluate ICP. If persistent ICP >20, consider continuous CSF drainage with intermittent setting of ICP (close drain for 5 min to obtain reading)

Hyperosmolar Therapies

- Hypertonic Saline (3%) bolus 5 ml/kg (max 250 ml), and continuous infusion at 1 ml/kg/hr, titrate for serum Na 150-160 and/or serum osmolality <360
- Mannitol 1gm/kg
- See hyperosmolar algorithm for more details

Consider 2nd Tier Therapies

- Consider **transient** controlled hyperventilation ($PaCO_2$ 25-35 mmHg) and monitor effect on markers of cerebral blood flow (NIRS, licox)
- Is the patient salvageable?
  - Assess: mech of injury, best GCS, age, pupil reactivity, CT scan
  - Frontal focal contusions with initial good GCS, consider decompressive craniotomy
  - Barbiturate therapy: bolus pentobarbital 5 mg/kg q30 minutes until 2-3 burst per screen. Then start infusion of 1 mg/kg/hr. If # of bursts increase, repeat bolus until appropriate # of bursts are seen and then increase infusion
  - Stop infusion if brain death is suspected (do not wean)



NIRS

- If rSO2 <55% or, change > 20%, evaluate for changes in-
  - Ventilation (EtCO2 appropriate?)
  - Perfusion (MAP and CPP appropriate?)
  - Increased metabolic demand (fever, seizure, shivering?)
  - Consider transcranial doppler to evaluate for vasospasm

ICP monitoring

- Indications- GCS </= 8
- Contraindations-
  - Coagulopathy (consider Factor VII if >20 ml/kg of FFP is required to correct)
  - Keep INR <1.35, plts >100K
- Ventriculostomy if open ventricles

BP parameters

Age	MAP*
1-2 yrs	53-59
3-4 yrs	61-65
5-6 yrs	67-69
7-8 yrs	70-72
9-10 yrs	73-75
11-12 yrs	75-77
13-14 yrs	77-79
15-17 yrs	80-84

*MAP value is 50%ile for 50% of height

STATUS EPILEPTICUS

Definition – Status Epilepticus: Seizure activity lasting more than 5 minutes or clusters of seizures for > 60 minutes without return to baseline mental status in between seizures.

Definition - Refractory Status Epilepticus: Seizures persistent after the use of benzodiazepine and one Anti-epileptic drug (AED). Associated with worse neurologic outcome and increased mortality

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, Posterior Reversible Encephalopathy Syndrome (PRES), Acute Disseminated Encephalomyelitis (ADEM), complex febrile seizure.

Status Epilepticus pathway:

Seizure > 5 minutes → **Ativan 0.1mg/kg** IV or **Diastat 0.2-0.5mg/kg** PR →

Seizure continues? → can give another **Ativan 0.1mg/kg** →

Seizure continues? → give if <1mo give **Phenobarb 20mg/kg**, if > 1mo give **Fosphenytoin 20mg/kg** →

Seizure continues? → give additional **phenobarb 10mg/kg** OR **fosphenytoin 10mg/kg**→

If still seizing, consider **versed** load potential continuous infusions.

1. Provide supplemental O_2 and assess ABCs. (May need respiratory or hemodynamic support.)
2. Neurology should be called early to start EEG if subclinical status suspected.
3. **RULE OF THUMB**: during a seizure eyes are usually open and dolls eyes (oculocephalic) reflex is **not** intact.
4. Labs/ Studies: immediate d-stick, electrolytes, 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. Head CT, consider LP

Indications for seizure prophylaxis

1. TBI (as per Brain Trauma Foundation)
   - GCS < 10
   - Cortical contusion
   - Depressed skull fracture
   - Subdural hematoma
   - Epidural hematoma
   - Intracerebral hematoma
   - Penetrating head wound
   - Seizure within 24 hours of injury
2. Supratentorial neurosurgery
3. Non-traumatic Intracerebral hemorrhage (e.g. AVM rupture)

BRAIN DEATH

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.**

Prerequisites to determine brain death (BD):

1. Ensure cause of neurologic state is *irreversible*.
2. Exclude any complicating medical conditions (e.g. electrolyte, acid-base or endocrine disturbances) or

3. Exclude 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).
4. Neuromuscular block effects ABSENT
5. Spontaneous respirations ABSENT

Clinical Exam: (All must be ABSENT)

1. Motor movements to noxious stimulation of all four extremities (spinally mediated reflexes permitted)
2. Movement to noxious stimulation of supraorbital nerve, temperomandibular joint, or nasal passage
3. Pupillary reaction to bright light
4. Corneal reflex*:* blink and grimace or withdrawal from pressure over cornea
5. Oculocephalic reflex (Doll's eyes) or not performed due to concern for cervical spine injury
6. Oculovestibular reflex (cold calorics): No nystagmus is produced
7. Cough reflex with tracheal suction
8. Gag reflex with stimulation of posterior pharynx
9. Apnea Test: Spontaneous respirations absent throughout trial
   a. Prerequisites: Hemodynamically stable, pre-oxygenated with 100% FiO2 for > 10 minutes, Ideal $PaCO_2$ on initial ABG 35-45 mmHg
   b. Disconnect from ventilator with oxygen via
      i. Self-inflating bag with patient valve OPEN, PEEP 5-10 cm H_2O
      ii. T-piece with flow of 100% Oxygen at 15L/min
      iii. Flow-inflating bag with 100% Oxygen with PEEP 5-10 cmH_20
   c. Check blood gas every 3-5 minutes and end when PaCO2 ≥ 60 mmHg and rise of 20 mmHg over baseline $PaCO_2$

Ancillary Studies:

1. *If* unable to complete clinical exam, apnea test, medication effect present of to reduce observation period between clinical exams
2. *Types:* Conventional 4 vessel angiogram, radionuclide angiography study or EEG

Number and Timing of Examinations:

1. According to age
   a. 37 wga – 30 dys of age: 2 exams separated by 24 hrs.
   b. > 30 dys to 18 yrs.: 2 exams separated by 12 hours
   c. > 18 yrs.: 2 exams separated by 6 hours
2. Assessment of neurologic function may be unreliable immediately after CPR or other severe acute brain injuries, therefore, brain death should be deferred for > 24 hours from time of insult to 1st exam
3. Exams must be performed by 2 separate physicians. 1 exam by a critical care fellow or attending. 2nd exam by a second CCM attending, neurology or neurosurgical attending

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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

2. **Moderate sedation** = "Conscious 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; 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.
NPO for >6 hours if **<2years old**; **if >2yrs**: >8hrs solids or >3hrs clears.

Children's National Medical Center

Ventricular Shunt Malfunction Diagnostic Algorithm

June 2013

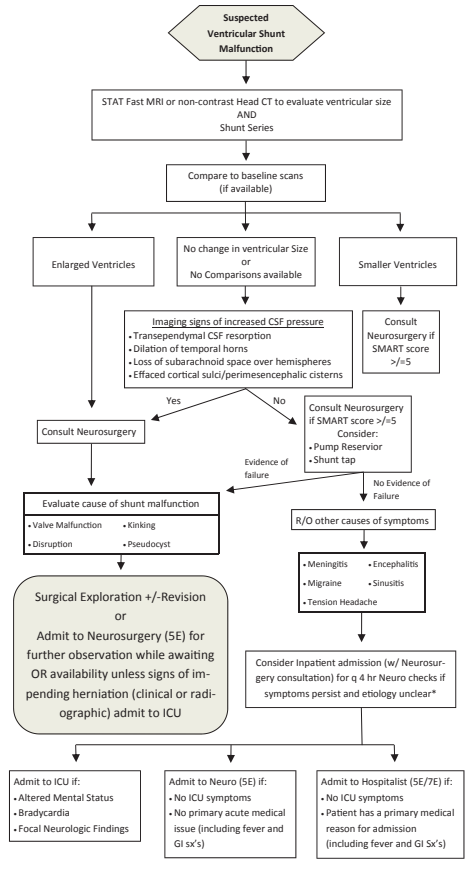
Acute Symptoms		
• Nausea	• Headache	• Irritability
• Vomiting	• Positional Headache	• Lethargy
• Hypertension	• Double Vision	• Stupor
• Bradycardia	• Sundown Sign	• Coma
• Seizures	• Transient visual obscurations (e.g. visual blackouts)	

Subacute/Chronic Symptoms	
• Change in behavior (e.g. agitation)	• Developmental regression
• Altered gate	• Change in cognitive function (e.g. attention span)
• Change in feeding patterns	• Daily headaches
• Change in school performance	• Increased head size

History to be Obtained Prior to Neurosurgical Consult
• Prior history of shunt failure
• Size of ventricles at last shunt failure
• Prior history of shunt failure without change in ventricular size
• Presence or absence of fevers
• Presence or absence of above acute and chronic symptoms

Fast MRI vs Head CT
• Available weekdays 8 am– 10 pm and weekends 8 am – 4 pm
• Patient must be able to lie still/cooperate for 10 minutes without sedation (roughly age >/=5 yrs)

Contraindications to Fast MRI	
• History of trauma	• Altered mental status
• r/o hemorrhage or pneumocephalus	• If shunt catheter needs to be visualized
• Programmable VP shunts	• Patients with other MRI contraindication (ex– pacemaker)
• Unable to obtain within 1 hour of presentation	



*Neurosurgical Attending notification at time of admission is expected

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SMART SCORE
(SHUNT MALFUNCTION ASSESSMENT AND REASSESSMENT TOOL)

Inclusion Criteria: Patient with VA or VP shunt and age > 1 month
Exclusion Criteria: Patients in the NICU

Major Criteria: (10 points each)
* Clinical signs of herniation (including Cushing's Triad)
* Imaging with signs of herniation/impending herniation
* Papilledema

Minor Criteria:

Tier 1 (5 points each)
* Positional headache (include awakening from sleep due to headache pain)
* New diplopia/CN VI palsy
* Altered MS (e.g. irritability, lethargy) in a patient w/ no (or mild) prior neurological deficit
* Neuroimaging with increased size of ventricles and/or decrease in sulci and/or cisterns (more sensitive if baseline large ventricles)
* History of VP shunt failure without prior change in ventricular size
* Head circumference increasing across percentiles (e.g. > 10%)

Tier 2 (2 points each)
* Nausea/vomiting
* Headache
* Bradycardia (not baseline)
* Increased seizure frequency from baseline
* Recent manipulation of VP shunt (e.g. change of setting of valve, revision in the last 30 days)
* Change in baseline in a patient with moderate to severe baseline deficits
* Neuroimaging with inc size of ventricles and no change in sulci and/or cisterns
* Radiographic evidence of shunt tubing disconnection

Recommended response based on assigned score
10 = immediate response from neurosurgery attending

5 -9 = high suspicion for shunt malfunction, neurosurgical consult. If no intervention prescribed, consider escalation to neurosurgical attending.

4 = moderate suspicion for shunt malfunction. Observation recommended. If symptoms not easily explained by other medical condition, consult neurosurgery.

** If score remains ≥ 4 consider repeat imaging Q2-3 days and/or ophthalmology evaluation for papilledema, even if other explanations plausible for observed symptoms.

BACTERIA BY GRAM STAIN

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-)	***Francisella tularensis* (CB)**
L+ = lactose fermenter	*Stenotrophomonas*	***Pasturella multocida* (CB)**
***Anaerobic**	***Fusobacterium****	***Brucella* (CB)**

COMMON PICU INFECTIONS

Infection	Likely Pathogens	Antibiotic Choice	NOTES
Meningitis	<1mo: GBS, enteric bacilli (*E. coli, Klebsiella, Proteus*), *Listeria monocytogenes* 1-3mo: same + *Strep pneumo, Neisseria meningitides, Hib*	<1m: Ampicillin + cefotaxime 1-3m: Vancomycin + CTX or cefotaxime	Corticosteroids ↓Vancomycin CSF penetration; If on steroids, rifampin may be better choice.
	> 3mo-5yo: *S. pneumo, N. meningitides, Hib*	>3mo: Vancomycin + CTX or cefotaxime	
	CSF shunt: *Staph aureus,* enteric organisms	Shunt: ceftazidime and gent and vancomycin	
Pneumonia	CAP: (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*	CAP: CTX ± azithromycin (atypical) With effusion: CTX + Clindamycin/vancomycin	Atypical pneumonia is unusual between infancy and 4 years of age
	Nosocomial (VAP): *Staph aureus, GNB, H. influenzae*	Nosocomial: Vancomycin + ceftazidime + Tobramycin Abnl Renal: Vancomycin + zosyn	
Spontaneous bacterial peritonitis	*E. coli, Klebsiella, Staph, Strep*	1st line: Vancomycin + ceftazidime + Flagyl Abnl Renal: Vancomycin + zosyn	Diagnose with ascites fluid

UTI/pyelo-nephritis	GNeg: *E. coli (80%), Proteus, Klebsiella, Enterobacter, Citrobacter;* GPos: *Staph saphro, Enterococcus, Staph aureus*	3rd gen cephalosporin or amp	UTI by Clean catch if >100,000 CFU; and by cath if >50,000 CFU
Life-threatening gastroenteritis	HUS *(E.coli);* Toxic megacolon *(C. diff, Shigella);* Amebiasis; **Majority will be viral**	***NO antibiotics*** *unless specific bacterial pathogen found* *C.diff*: enteral flagyl or vanc	
Osteomyelitis	*Staph aureus* (>50%), Grp A and B *Strep*, *E. coli* Rare: *Kingella kingae, Bartonella, Brucella, TB, Salmonella* (SSDz)	Neonates: cefotax & vanc Infants and older: oxacillin, clindamycin or vanc (if>10% CA-MRSA), linezolid if fails 3-36m: cefazolin (*K. kingae)*	May need surgical intervention.
Cellulitis, Skin Abscess	*Staph aureus, Beta-hemolytic Strep*, gram negatives in rare cases	Clindamycin or Vancomycin	Drainage alone may be therapeutic

NUTRITION SUPPORT IN THE PICU

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. Marked by ↑prealbumin and ↓ CRP	Increase nutrient provision to promote repletion, growth will resume

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

For critically ill or injured infants and children, provide basal needs. During the Acute, Ebb and Flow-Catabolic stages, children do not need calories for activity, growth or healing; therefore, energy needs are significantly decreased.

Basal Metabolic Rate (kcal/kg/day)		
Age	Male	Female
0-36 months	55	55
4-8 years	50	45
9-13 years	35	30
14-18 years	30	25

*** Initiating nutrition within 48-72 hours of PICU admission has been shown to lower mortality.**
If expected to be NPO >3-5 days, start enteral nutrition ASAP
Start TPN/IL after 3-5 days of NPO or failed enteral nutrition in well-nourished infants and children
Start TPN/IL after 7 days of NPO or failed enteral nutrition in well-nourished adolescents and adults

***Special Consideration for patients with poor cardiac output, renal or hepatic failure, or burns patients with high needs and high fluid losses**

ENTERAL NUTRITION (EN)

1. The gut is ***always*** the preferred mode of nutrition!

2. Maintains gut motility, improves mesenteric blood flow, supports gut-associated lymphoid tissue, releases trophic factors from the gut and pancreas to maintain cell lining and enterocyte mass, lower cost.
2. 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
3. When feeding through an ND, bolus feeds are **not tolerated**
4. Bolus feeds into a NG are preferred since they are more physiologic
5. Complications of tube feeding may include refeeding syndrome, overfeeding and excessive CO2 production, increased metabolic activity can exceed cardiac capacity, complications in patient with renal failure or hepatic failure, mechanical issues with the enteric tube such as perforation or displacement.

Common formulas to use when initiating EN:

1. Infants (<1 year): Use breast milk or home formula. Consider concentrating formula to 24 kcal/oz if patient is fluid restricted.
2. Children (1-10 years): NutrenJr or Pediasure
3. Adolescents/Adults (>10 years): Nutren 1.0 or Boost
4. High-Protein (burn patients, obese patients): Isosource HN or Replete w/ Fiber

Indications for a Nutrition Consult prior to initiation enteral feeds: if patient has a true milk-protein allergy (NOT lactose intolerance – all pediatric and adult formulas are lactose-free), requires a special formula (renal, semi-elemental, elemental, soy, etc.), or concentrated breast milk,

TOTAL OR PERIPHERAL PARENTERAL NUTRITION (TPN/PPN)

1. Unless malnourished, do not use unless patient is expected to be NPO >3 days
2. Expensive; risk of infection from the TPN/IL itself and from the central line, risks of electrolyte and acid-base disturbance, cholestasis, excessive CO2 production, and increased risk of bacterial and fungal infection.
3. PPN osmolarity limits (<900 mOsm/L)often prevent adequate delivery of calories and protein

For TPN, refer to "Parenteral Nutrition Guidelines" on the Intranet

1. Fill in **weight** and determine total **volume** and means of **access** (central or peripheral)
   a. Total volume = TPN + enteral feeds + continuous infusions (including intralipids)
   b. Non-TPN volume = enteral feeds + continuous infusions (including intralipids)
2. Dextrose: Initiate at D10%, refer to PN Guidelines for appropriate GIR initiation rates (GIR found in lower right corner of TPN order form in BAXA). To calculate GIR (mg/min/kg):
   a. first determine milligrams of dextrose by multiplying percent dextrose x total volume (mLs) received x 1000 (i.e. 1000 mLs of D10% = 100 grams dextrose x 1000 = 100,000 milligrams).
   b. then divide by minutes that patient received infusion, then divide by patient's weight in kg
3. Protein: Initiate at 2 gm/kg/day, advance by 1 gm/kg daily per PN Guidelines, if appropriate
4. Lipids: Initiate at 1 gm/kg/day for non-overweight/obese patients
   a. for overweight and obese patients, adjust lipid dose to provide no more than 30% kcal from lipids (% kcal from lipids found in lower right corner of TPN order form, written as L=_%")

b. *Lipid infusion rate*: For infants <1 year, lipids run over 24 hours (max infusion rate is 0.15 gm/kg/hr). Children >1 year should receive lipids over 12 hours. Click on "Lipids" tab and check "Special Lipid Duration"; change "Lipid Duration" to 12 hours

5. Ensure total kcals is no more than 90% BMR – decrease dextrose, protein, and/or lipids as needed
6. Refer to Parenteral Nutrition Guidelines for recommended electrolyte dosages
7. Add Ranitidine to TPN, if applicable – Click "Add Ingredient", choose Ranitidine
8. Monitor daily BMP, Mg, Phos until stable. Check CMP, TG, PAB, CRP upon initiation of TPN and weekly thereafter

ALL TPN ORDERS MUST BE ENTERED AND SIGNED PRIOR TO 1pm

SUPPORTIVE AND PALLIATIVE CARE

COMMUNICATION PEARLS

Families want an honest, timely, consistent message delivered with empathy, respect and protection of dignity. What you say to families is as important as how you say it. Try to remember that families are vulnerable and look to you as the one person who can save their child. When a child is sick the parent also exhibits "sick" behaviors, which means they often suppress the cognitive areas of their brains and cannot process lots of new information, have short-term memory loss, and limited ability to decode complex language or abstract thoughts. Below are some pearls that may improve your communication with families of critically ill children.

- Be honest – say you don't know if you don't know. It can be confusing for families to receive different messages from providers. Defer to your attending for more complex discussions.
- Show empathy – a smile goes a long way. When you look sad, families internalize those emotions and perceive you must have bad news about their child that you are withholding. This leads to anxiety and fears. Make eye contact, lean in, take a seat.
- Respect the patient and family's space – Families are confined to a limited space when in the PICU. Knock before entering, introduce yourself, and ask permission to touch the patient. Common courtesies go a long way toward establishing trust.
- Choose simple language – We all know not to use medical jargon when communicating with families, but we don't always realize that general speech patterns of physicians are far more complex and sophisticated than speech patterns of the general public. Choose your words wisely – say walk instead of ambulate, try breathe in lieu of ventilate, etc.
- "Ask-Tell-Ask" is a back-and-forth cycle between the family and physician that addresses four essential components: the family's perspective, information that needs to be delivered, response to the family's emotions, and recommendations by the physician. This method allows you to use the language the family uses to enhance their understanding and tailor the information you provide.

DELIVERING BAD NEWS

Bad news is defined in the medical literature as news that results in a cognitive, behavioral or emotional deficit in the person receiving the news and that these deficits persist. You can't change the news itself, but the way you give it can shape the experience for the family. When delivering bad news to a family there are a few tips that can improve their understanding, build trust and not have the family pass out – the last thing you want is an adult "code blue."

- Prepare – know the facts, find a quiet space, find the time without interruptions to talk
- Assess the family's understanding (Ask-Tell-Ask). "I want to make sure were on the same page, please tell me your understanding of Joey's illness."

- Fire a warning shot – Tell the family you are going to deliver bad news to prepare them to listen intently. “I wanted to meet with you to discuss Joey’s recent events, they have not been good.” “I have some bad news about the results of Joey’s brain scan”
- Deliver the news using the tips above
- Allow silence for the information to sink in
- Solicit questions to assess the family’s understanding
- Don’t offer false hope – It is natural to want to make people feel better when they are sad, but you will only confuse the family or delay healing by offering false hope. You can however still offer hope even if the situation is grim. “We are doing everything we can to make sure Joey is comfortable and feels no pain.”
- Plan a follow up – Families often need some time to process the information they received. Give them some space then make sure a social worker, nurse or physician checks in with the family within an hour of the bad news.

PALLIATIVE CARE IN THE PICU

The goal of the palliative care team in the PICU is to support informed decision-making ensuring that the patient and family’s wishes are respected, promote quality of life for the patient and family, and meet the needs of body, mind and spirit of the patient and family. Palliative care can occur simultaneously with cure-directed therapies. Consider a palliative care consult for patients with chronic, life-threatening illnesses, patients with acute illnesses and life expectancies <6 months, or patients and families who appear to need an extra layer of support.

CARING FOR THE CAREGIVER (that means you)

The PICU can be a tough place, emotionally, physically and intellectually. You are better able to care for your patients and families if you prioritize caring for yourself. Coping with death can lead to moral distress, PTSD and grief among caregivers. Rely on the coping strategies that have gotten you this far in life. Talk with your friends and colleagues, ask your attending to lead a debriefing session, exercise, do yoga, meditate and get some rest. The PANDA care team and chaplains are hospital resources available to you as well.

COMMON PICU MEDICATIONS

(Please also see Children's National Formulary)

Asthma Medications

	MECHANISM/CLASS	***DOSE***	***SE, notes***
Albuterol	Aerosolized beta$_2$-agonist = relaxation of bronchial smooth muscles	Intermittent Neb: 0.15-0.3 mg/kg (minimum 2.5 mg; max 10 mg) Continuous Neb: 0.5 mg/kg/hr (max: 25 mg/hr) MDI: 4 to 8 puffs every 20 minutes	Tachycardia, jitteriness, hypokalemia, diastolic hypotension, ST elevations
Ipratropium	Anticholinergic, inhibits cGMP = bronchodilation	0.25 to 0.5mg neb Q6hr if responsive	Best in 1st 24-48hrs, stop after if no response
Corticosteroids (Methylprednisolone IV or Prednisolone PO)	Systemic anti-inflammation	methylprednisolone: 0.5 to 1 mg/kg q6hr (max: 80mg/day) Prednisolone: 2mg/kg PO daily (max: 80mg/day)	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
Terbutaline	Systemic beta$_2$-agonist	Load 10 mcg/kg, infusion 0.5-1mcg/kg/min titrate as needed to a max of 10 mcg/kg/min	Same as albuterol, watch for ST depression and chest pain, hyperglycemia
Ketamine	Dissociative anesthetic that causes inadvertent bronchodilation	0.5-1mg/kg, infusion 0.5-1mg/kg/hr titrate Q30min	Secretions, hallucinations, hypertension, myocardial depression
Aminophylline	Methylated xanthine derivative, ↑cAMP and ↓inflammation	Loading dose: 6mg/kg, infusion of 1mg/kg/h, titrate to theophylline levels	Nausea, tachycardia, anxiety, seizures, HA, diuresis (transient)
Isoflurane	Inhaled anesthetic agent which causes airway relaxation	See Children's National Protocol	Hypotension requiring IVF and vasopressors is common

Inotropes and Vasopressors

	Receptor	Indication	SE/Notes	Dose
Dopamine	DA, β, α_1	• Fluid refractory shock • Hypovolemic shock • Septic shock	Tachycardia, Immune-suppression	Low (DA) = 1-5 **mcg**/kg/**min** Med (β) = 5-10 **mcg**/kg/**min** High (α) = 10-20 **mcg**/kg/**min**
Dobutamine	β1>β2	• Cardiogenic shock	Tachyarrhythmias, Hypotension	2-20 **mcg**/kg/**min**
Epinephrine	β1=β2>α	• Fluid refractory shock • Distributive shock • Cardiogenic shock • Arrest • Post-arrest	Tachyarrhythmia, Myocardial ischemia 2/2 increase metabolic demand	0.02-1 **mcg**/kg/**min**, as ↑ go from β1 to β2 to α *Code* – (1:10,000) IV 0.01 mg/kg = 0.1 mL/kg (1:1,000) ETT 0.1 mg/kg = 0.1 mL/kg *Ultra low dose* (1:100,000)– 0.001mg/kg = 0.1 mL/kg
Milrinone	PDE type III inhibitor: ↑cAMP, ↓ SVR, ↑HR and contractility	• Cardiogenic shock • Cold septic shock	Hypotension, ventricular ectopy. Long half-life, renally dose if have AKI	0.2-1 **mcg**/kg/**min**, load 25-75 mcg/kg
Norepinephrine	α_1>β	• Warm septic shock	Tachyarrhythmia	0.01-2 **mcg**/kg/**min**
Phenylephrine	α_1	• Neurogenic shock	Reflexive bradycardia	0.05-0.5 **mcg**/kg/**min**
Vasopressin	V1, V2 : vasoconstriction	• Catecholamine resistant shock • Warm septic shock	Venous thrombosis, hyponatremia	0.3-2 mU/kg/min

β1: ↑ionotropy (contractility) and chronotropy (HR), β2: bronchodilation and vasodilation, α: vasoconstriction, DA: dilate renal and mesenteric vessels

Anti-arrhythmics and Anti-hypertensives

	Receptor	Mechanism of Action	Indication	SE/Notes	Dose
Adenosine	Adenosine receptor	• Transiently blocks AV node conduction	• SVT	Make sure on ECG while give	0.1 mg/kg /dose IV (max: 6 mg) repeat 0.2 mg/kg/dose (max: 12 mg) (rapid push with flush)
Amiodarone	K channel block: Class III anti-arrhythmic	• Slows AV and SA nodal conduction by ↑ refractory period (↓ repolarization	• VF/VT • Chronic atrial fibrillation rate control	Bolus can cause cardiac arrest	5 mg/kg/dose IV repeat up to 15 mg/kg (rapid push in arrest, otherwise slow push over 20 to 60 minutes)
Atropine	Anticholinergic - Muscarinic acetylcholine	• Blocks vagal nerve action	• Bradycardia • Heart block		0.02 mg/kg/dose IV Max–1mg

B-blockers **Labetalol** **Esmolol**	β1, β2, mild α β1 only	• ↓ SVR without ↑ HR or SV • ↓chronotropy and inotropy	• Hypertension • Aortic dissection (with vasodilator) • Rate control atrial fibrillation/flutter	Hypotension, bradycardia, bronchospasm if B2, fatigue	0.25 – 3 **mg**/kg/**hr** load 0.2 to 1 mg/kg 100-500 **mcg**/kg/**min** Load 0.3-0.5 mg/kg
Ca Channel Blocker ***Dihydropyridine*** **Amlodipine (Aml)** **Nicardipine (Nic)** ***Non-dihydropyridine*** **Diltiazem** **Verapamil**	Calcium Channel In vascular smooth muscle In myocardium > vasc smooth muscle	• Arterial and venous dilation • ↑ blood flow in coronaries • ↓ ionotropy/ chronotropy (↓ AV Node conduction) • Mild vasodilation (Verapamil)	• Hypertension • Angina • Hypertension • Angina • Rate Control Afib/Flutter	Hypotension Heart block, depressed ventricular function	Aml-PO Children 1 to 5 years: 0.05 to 0.1 mg/kg/day Children 6 to 17: 2.5-5 mg qday Nic-0.5-5 **mcg**/kg/**min**
Hydralazine	Direct smooth muscle relaxant	• ↑cGMP, vasodilation	• Hypertension	Hypotension	0.1-0.2 mg/kg
Isopreterenol	β1 and β2	• ↑ chronotropy and ionotropy	• Bradycardia • Heart Block	Tachyarrhythmias, hypotension	0.05-2 **mcg**/kg/**min**
Lidocaine	Na Channel block : Class 1B anti-arrhythmic	• Shortens action potential • ↑ refractory period	• Ventricular arrhythmia	Hypotension, bradycardia,	1% - 1 mg/kg/dose IV/ETT Continuous infusion: 20-50 **mcg**/kg/**min**
Sodium Nitroprusside	Direct smooth muscle relaxant	• Metabolized into NO	• Hypertension	Hypotension, dilates cerebral arteries (↑ ICP) cyanide toxicity at doses >4	0.3-8 **mcg**/kg/**min**
Procainimide	Na channel block: Class 1a anti-arrhythmic	• ↓conduction • ↑ refractory period	• Afib • WPW • Wide complex stable tachycardia	Hypotension, arrhythmia	3-6 mg/kg load over 5 min (max: 100mg) Continuous infusion: 20 to 80 **mcg**/kg/**min**

Other Code Meds and Electrolytes

	Indication	*SE/Notes*	*Dose*
Calcium Chloride **Calcium Gluconate**	Hypocalcemia, cardiac membrane stabilization, improve vasomotor tone	Use Central line or IO, can give calcium gluconate peripherally if 20 mg/mL	10-20 mg/kg/dose IV Max 1000mg 100 -200 mg/kg IV Max 2000 mg
Dextrose	Hypoglycemia and increase metabolic demands	Use central line if 25% or higher concentration	0.5 to 1 gram/kg use rule of 50s: 10% - 5 ml/kg; 25% - 2 ml/kg; 50% - 1 ml/kg
Hydrocortisone	Shock	Hyperglycemia, impaired wound healing	1-2 mg/kg or 50 mg/m^2 IV
Magnesium Sulfate	Hypomagnesemia, torsades de pointes	Hypotension	25-50 mg/kg/dose IV Max 2000mg
Phosphate **Potassium Phosphate** **Sodium Phosphate**	Hypophophatemia	Incompatible with Ca++ containing solutions	0.15-0.5 mmol Phos/kg Max 30 mmol Phos
Potassium Chloride	Hypokalemia	May burn given peripheral IV – must give slow and lower concentration	0.25-1 mEq/kg, Max 40 mEq
Sodium Bicarbonate	Metabolic acidosis		1-2 mEq/kg/dose IV Use 4.2% if patient is < 6 mo

Diuretics

Class	Drugs	Mechanism	SE/Notes
Osmotic diuretics	Mannitol (0.5-1 gm/kg IV once)	Freely filtered and non-absorbed by the proximal tubule to prevent H_2O reabsorption	Works for acute management of elevated ICP or IOP
Loop diuretics	Furosemide (0.5-1mg/kg IV/PO q6h) Bumetadine (0.02mg/kg q6h)	Inhibit Na/K/2Cl transport in the thick ascending loop	Hypercalciuria (potential stones), ototoxicity and ↓K
Thiazides	Chlorothiazide (10-20mg/kg PO BID, 5-10 mg IV BID)	Inhibit NaCl cotransport in early distal convoluted tubule	Used for with loop diuretics, in nephrogenic DI and hypercalciuria, Weaker than loop diuretics.
K$^+$ sparing diuretics	Sprinoloactone (1-3.3mg/kg qday)	Blocks aldosterone action on the cortical collecting ducts; A/T inhibit Na/K pump	Used to counteract the hypokalemia from other diuretics, weak diuretics alone
Carbonic anhydrase (CA) Inhib.	Acetazolamide (5mg/kg once daily) Metolazone (0.1-0.2mg/kg/dose q12-24)	Inhibit CA in the luminal membrane of the proximal tubular = reduced HCO_3 reabsorption.	Used to counteract metabolic alkalosis, Also used in glaucoma, altitude sickness.

Sedation

Drug	Dose	Mech of Action	Side Effects/Notes	Reversal
Fentanyl	1-2mcg/kg IV (max 5mcg/kg) 1-5 mcg/kg/hr infusion	Opioid mu receptor	Apnea, rigid chest (if pushed)	Naloxone 0.01-0.1 mg/kg IV
Midazolam	0.05-0.2mg/kg IV 0.05-0.5 mg/kg/hr IV infusion (can give intra-nasally or IM)	Gaba agonist	Apnea, ↓BP, paradoxical agitation	**Flumazenil 0.01mg/kg IV ****lowers seizure threshold****
Morphine	0.05-0.2mg/kg IV 0.01-0.2 mg/kg/hr IV infusion	Opioid	Apnea, ↓BP, hives	Naloxone 0.01-0.1mg/kg IV
Ketamine	0.5-1mg/kg IV/IM 0.1-0.2 mg/kg/hr IV infusion- adjunct to narcotics	NMDA agonist	Hallucinations, secretions (have robinul or atropine ready) – onset confirmed by nystagmus	None
Dexmedetomidine	0.5-1 mcg/kg 0.5-2 mcg/kg/hr IV infusion	Central α agonist	Bradycardia, hypotension, hyperglycemia	None
Propofol	1-2 mg/kg IV 50-200 mcg/kg/min	Gaba agonist	Apnea, ↓BP, metabolic acidosis – rapid onset, short duration	None (NOTE: contraindicated in egg allergy)

Paralytics

Drug	Dose	Onset/Duration	Notes
Rocuronium	1 mg/kg	30-60 sec/45-70 min	Urine and bile excretion
Vecuronium	0.1 mg/kg	1-3 min/20-30 min	Renal and hepatic excretion
Cisatricurim	0.15 mg/kg	1-4 min/30-45 min	Good in hepatorenal dysfxn
Succinylcholine	1 mg/kg	30-60 sec/4-6 min	Good for RSI, Raises ICP, sinus bradycardia

Antibiotics

** Please refer to online formulary for dosing**

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

Name	Cidal?	Gram +	Gram -	Anaerobes	Side Effects
			ß -Lactams		
Oxacillin	Y	+++	0	0	Anaphylaxis, hepatotoxicity, ↑LFTs, neutropenia
Ampicillin	Y	VRE, +++	++ *H. flu, E.coli Proteus*	+*Actino*	Anaphylaxis, rash, reversible neutropenia
Penicillin	Y	++++	+*Neisseria*	+++, Syphillis+	Anaphylaxis, sz neurotoxicity
Ampicillin/Sulbactam (Unasyn)	Y	+++	+++, NO *Pseudo, Serratia, Entero*	+++	Anaphylaxis rash (with EBV infxn)
Piperacillin/Tazobactam (Zosyn)	Y	++++	++++, NO *Pseudo, Serratia, Entero*	*++, Bacteroides*	GI, Anaphylaxis,
			Cephalosporins		
1st Generation **Cefazolin (Ancef)**	Y	+, No MRSA	++, No *Enterobac*	0	SJS, anemia, anaphylaxis, GI
3rd Generation **Cefotaxime Ceftazidime Ceftriaxone Cefdinir (Omnicef)**	Y	+++, *no MRSA*	++ +pseudo (ceftaz)	0	Anaphylaxis, , renal insuff, seizures (ceftaz), biliary sludging (Ceftriaxone) Cefdinir-red stool, agranulocytosis, allergy, SJS
4th Generation **Cefepime**	Y	++++, +MRSA	++++, *Pseudo+*	0	Anaphylaxis, , renal insuff, +Coombs
			Aminoglycosides		
Amikacin	Y	0	++++	0	Oto- and nephro- toxic, poor CNS penetration, ↓Ca
Genta-micin	Y	+MSSA	++	0	Ototoxic, ↓Ca, nephrotoxic
Tobra-mycin	Y	Some *Staph*	+++	0	Ototoxicity, nephrotox, vertigo
			Other		
Carbapenem **Meropenem**	Y	++++	++++ *Pseudo+*	++++	Anaphylaxis, ↓K, sz, renal insuff; **AVOID with valproic acid**
Fluoroquinolone **Ciprofloxacin**	Y	0, MSSA	++++, infectious diarrhea	0	↑QTc, ↑levels of other meds, Allergy,
Macrolide	**No**	++, No MRSA	++, GNB resistant	0	GI, anaphylaxis,

Azithromycin					
Sulfonamide **Bactrim (TMP/SMX)**	Both	+MRSA	++++	0	SJS, BM Suppress, liver injury
Aztreonam	Y	0	++++ *Pseudo+*	0	Rash, cross-rxn with PCN allergy,
Clindamycin	**No**	++, MRSA	Aerobic Resist.	++++, *PCP*	SJS, hypotension, C. diff
Metronidazole (Flagyl)	**No**	0	0	++++ (*C. diff* give enteral)	SJS, peripheral neuropathy, disulfuram rxn
Linezolid	**No**	+++VRE+MRSA	0	Some *C. diff & act-inomyces*	Fungal infxn, pancreatitis, optic neuritis, periph neuropathy
Rifampin	Y	MRSA, TB	0	0	Flu-like, suppress BM, ↑bili
Vancomycin	**No**	++++	0	*C. diff* (enteral form only)	Nephrotoxic, ototoxic, poor CSF penetration, SJS, pancytopenia, anaphylaxis, Redman's

Organism	Penicillins						Cephalosporins					Carba	Mona	FQ		Macs	AG	Vanco	Syner Linez Dapto	Tets	Sulfonamide	Clinda	Metro
	PenG PenV	Meth Naf Oxa	Amp Amox	Una Aug	Pip	Tim Zos	1st	2nd	3rd	4th	5th	Imi Mero Erta Dori	Aztreo	Cipro	Levo Moxi Gati	Ery Clar Azi	G, T, A			Doxy Mino Tige	TMP/SMX		
MSSA		X		X		X	X	X	X[δ]	X	X	X		X	X	?X	X	X	X	X	X	X	
MRSA											X							X	X	X[□]	X	X	
PSSP	X	X	X	X	X	X	X	X	X[δ]	X	X	X		?X	X	X		X	X	X		X	
PRSP									X[δ]	X	X	?X			X			X	X				
Viridans	X	X	X	X	X	X	X	X	X[δ]	X	X	X			X	X		X	X			X	
Group Strep	X	X	X	X	X	X	X	X	X	X	X	X		?X	X	X		X	X	X	X	X	
Enterococcus	X		X	X	X	X					?X	X[!]			?X		X	X	X[€]				
Listeria	X[∩]		X	X[◊]	X							X		X	X	X	X	X	X		X		
H. influenz			X[$]	X	X	X	X	X	X	X	X	X	X	X	X	X[¶]	X			X	?X		
M. catarr				X	?X	X		X	X	X	X	X	X	X	X	X	X			X	X		
Niesseria	X[∩]		X	X	X	X		X	X	X	X	X	X	X	X	X				X			
E. Coli			X	X	X	X	X	X	X	X	X	X	X	X	X		X				?X		
Proteus			X	X	X	X	X	X	X	X	X	X	X	X	X		X						
Klebsiella				X	?X	X	X	X	X	X	X	X	X	X	X		X				?X		
Enterobacter					X	X			X	X	?X	X	X	X	X		X						
Serratia						X			X	X	X	X	X	X	X		X				?X		
Salmonella			X	X	X	X			X	X	X	X		X	X						?X		
Pseudomonas					X	X			X[¶]	X		X[!]	X	X	X[%]		X						
Citrobacter					X	X		?X	X	X		X	X	X	X								
Legionella														X	X	X				X	X		
ADA	X		X	X	X	X		X[#]				X						X				X	?X
BDA				X	X	X						X										?X	X
C. diff																		X					X

MSSA = methacillin-susceptable Staph. aureus

MRSA = methacillin-resistant Staph. aureus

PSSP = Penicillin-suspectable Strep pneumoniae

PRSP = Penicillin-resistant Strep pneumoniae

Una = Unasyn (ampicillin/sulbactam) Aug = Augmentin (amoxicillin/clav)

Tim/Zos = Timentin (ticarcillin/clav) and Zzosyn (piperacillin/tazo)

ADA = Above the Diaphragm anaerobes

BDA =Below the diaphragm anaerobes (especially Bacteroides sp.)

□ = TIGECYCLINE EXPANDED COVERAGE AGAINST GRAM +/- AEROBES (EXCEPT PROTEUS AND PSEUDOMONAS) AND ANAEROBES

∩ = Pen G only

◊ = Una only

* = BETA-LACTAMASE NEGATIVE STRAINS ONLY

= CEFOTETAN AND CEFOXITIN

$ = CEFTRIAXONE AND CEFOTAXIME ONLY

& = CEFTAZIDIME ONLY

¶ = AZITH AND CLARITH ONLY

! = NOT ERTAPENEM

€ = ACTIVITY VERSUS VRE

% = NOT MOXIFLOXACIN

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Notes: