Disclaimer: This handbook contains general guidelines for the management of critically ill pediatric patients. Always discuss specific patient care issues with the ICU attending or fellow. Always communicate changes in patient status or plan with the ICU attending and all services involved.

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Goals and Objectives

OVERALL EDUCATIONAL GOAL:
To teach residents to care for children with serious medical and surgical problems and to learn the principles to identify and manage children with a critical illness. Residents will acquire an understanding of the pathophysiologic basis of common disease processes in the PICU, learn the technical skills to resuscitate and care for critically ill children, and understand end of life care for children and their families.

OBJECTIVES:
This rotation should allow the resident to accomplish the following objectives (in no particular order):

1. Recognize and appropriately respond to acute life threatening events. Acquire the necessary skills to resuscitate and provide initial stabilization of the critically ill child. (PC)

2. Demonstrate mastery of basic airway skills (use of oxygen delivery devices and demonstration of bag-mask ventilation) and placement of peripheral intraosseous and intravenous catheters. Advanced airway skills (intubation) and advanced intravenous access skills (central venous catheter and arterial line placement) may be performed if mastery of basic skills. (PC)

3. Understand the pathophysiology and treatment of common medical disorders in the PICU: respiratory failure (bronchiolitis, asthma, ARDS), shock (septic, cardiogenic, hypovolemic), neurologic critical care (status epilepticus, altered mental status, traumatic brain injury, brain death), renal and liver failure. (PC, MK)

Core Competency Codes
- PC=Patient Care
- MK=Medical Knowledge
- I&CS=Interpersonal & Communication Skills
- P=Professionalism
- PBL=Practice-Based Learning
4. Understand the indications, perioperative management and complications of common surgical admissions to the PICU: congenital heart disease, trauma, ENT, orthopedic, neurosurgical and organ transplant. (PC, PBL)

5. Understand the different monitoring techniques in critical care: vascular hemodynamics, intracranial devices, blood pressure, arterial saturations, end-tidal CO₂, and a variety of common laboratory tests. (PC, PBL)

6. Understand pediatric critical care pharmacology: inotropes and vasoactive agents, basic antibiotic therapy, common sedatives and analgesics, drug pharmacokinetics and monitoring of side effects. (MK, PBL)

7. Understand techniques for enteral and parenteral nutritional supplementation in the PICU patient. (MK)

8. Understand the ethical and legal issues which emerge during the care of critically ill and/or dying children (do not resuscitate orders, withholding and withdrawing life support, right of patients). (MK, SBP)

9. Understand the importance of psychosocial issues related to the care of critically ill or dying children. Learn to provide support and deliver difficult information to the family of a critically ill child. (MK, PBL)

10. Succinctly present an ICU patient on rounds, formulate a coherent assessment of a patient’s problems and present an appropriate therapeutic/diagnostic plan. Further, effectively communicate this plan to nurses, respiratory therapists, and sub-specialists/consultants. (PC)
Clinical Responsibilities
Rounds are standardized in the following manner:

- Rounds start at 0730 with the PICU fellow/attending running through the list of patients at the board to assess which patients can be transferred/discharged and to go over potential admissions.
- X-rays are often reviewed next on PACS. Each resident is responsible for interpreting their patient’s x-rays
- Rounds then begin at Room 1
- The format for rounds is:
  - Brief synopsis of major events in the past 24 hrs by the post-call resident
  - RT report of ventilator settings and airway maintenance issues
  - RN report of drips, access, and any concerns
  - Pharmacy report of current meds for reconciliation with CPOE
  - Resident report of assessment and plan
  - Family questions and concerns
  - PICU fellow summary of plan

Responsibilities include

- Reading about your patient’s condition, disease process, medications, etc.
- Pre-rounding on your patients (includes knowing overnight events, reviewing vital signs, conducting full physical exam, reviewing and reconciling patient medications from CPOE and MAR)
- Writing daily progress notes, transfer notes, accept notes, significant event notes and discharge notes
- Entering orders in CPOE (please use the same dosing weight (usually the admission weight) on every patient every day despite any new documented weights. You
should only change the dosing weight after discussion with PICU fellow/attending

- Reviewing and correcting/updating orders on all patients every day (for example, post surgical patients may return to the PICU with orders written by surgery that you need to evaluate)

- Don’t forget your prophylaxis if applicable
  - Famotidine for gastritis prophylaxis in patients who are NPO, on steroids, or on post-pyloric feeds
  - SCDs for DVT prophylaxis in post-pubertal or high risk patients
  - Peri-op antibiotics (check with the surgeons)

- When performing a “rule out” for fever, panculture your patient (blood, urine, sputum, CSF) wherever applicable. Don’t forget to get a peripheral blood culture as well in a patient with a central line

- When leaving the unit (for a break, to nap, to eat, etc), provide a quick sign out to resident/s who remain in the unit. Please also notify the fellow/attending that you are leaving and summarize any patient care tasks that still need to be done prior to your return

Specific responsibilities

On call resident
- Listen to everyone’s presentations as you are responsible for knowing every patient in the unit
- Take notes on the post-call resident’s “to-do” list as their presentation is considered sign out
- Divide the patients for pre-rounding among the 3 residents who will be present on your post-call day (including yourself)
Post call resident
- Complete daily progress notes prior to rounds
- Present the overnight events for each person
- Depart immediately after rounds

Short call resident
- Enter orders in CPOE
- Sign out to on call resident prior to noon lecture
- Attend noon lecture prior to departure

Afternoon resident
- Arrive at 1200 for lecture
- Obtain sign out from the on call resident regarding things that need to be done at 1300

Helpful tips
- PICU nurses are very experienced and invested in the care of these patients. Learn from them. Take their advice and concerns seriously.
- If a nurse asks you to call the fellow/attending, do it.
- If in doubt, call the fellow/attending.
- The only stupid question is the one you didn’t ask.
- Follow up on anything that was supposed to happen (including labs and x-rays and CT scans. Even if you aren’t a neurologist, you will likely notice something really bad that we should know about).
- Keep the surgical residents apprised of any changes in their patients.
- If in doubt about orders on surgical patients, ask the fellow/attending the best course of action.
- The PICU nurses perform arterial punctures and place peripheral IV access. You should make an effort to ask them to take over these procedures.
• When a PICU patient requires a central line, chest tube, or endotracheal tube, they are often too ill for a resident (and sometimes a fellow) to complete. These procedures are fellow level procedures but may be offered to you if you are readily available at the bedside and the patient is deemed appropriate by the fellow/attending.

### Educational Responsibilities

**Educational conferences**

<table>
<thead>
<tr>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
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<tbody>
<tr>
<td>* Mock code/Chalk talk 12:00</td>
<td># Fellow lecture (11-12)</td>
<td># Fellow lecture (11-12)</td>
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<tr>
<td># Cath conf (3:30-5:30)</td>
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* Required  
# Optional

Take charge of your education by reading about your patients, asking questions, using the resources discussed during orientation to find and read pertinent articles, and making sure the attendings and fellows give your twice weekly formal lectures. Remember that there can be a lot of teaching on rounds so listen up and ask questions.
PICU PHONE NUMBERS

Phone #s
Fellow: 45488
Unit:
Charge Nurse:
RT: 5-7250 and 5-7251
Radiology reading room: 67554
ICU Progress Notes

Past 24 Hour Summary
- Include only pertinent events such as intubation/extubation, CPR, new neurologic deficit, etc
- Do not include non-acute events such as titration of infusions, dietary changes, etc.

Care Checklist
- DVT prophylaxis required in every post-pubertal and/or high risk patient
- GI prophylaxis indicated in any patient who is NPO for longer than 24 hours, on steroids, or receiving post-pyloric feeds
- Spont breathing trials and daily awakenings are indicated in most intubated patients on sedation/paralytic
- HOB should be at 30° for every patient except those in the first 48 hours of a suspected ischemic stroke
- Beta blockade is only recommended in patients > 18 years of age
- Central line assessment is required in every patient with a central line including PICC

Medications
- Unclick non-pertinent medications such as IVF, most PRN meds such as electrolyte replacement
- Only include pertinent meds such as infusions, antibiotics, cardiac meds, etc.

Physical Exam
- Document at least General, HEENT, CV, Pulm, Abd, Ext, and Neuro exam on every patient

Physician Assessment
- First line should include patient age, pertinent PMH, reason for admission, and current status.

Examples:
3 yo with Trisomy 21 and AVSD admitted s/p AVSD repair on 11/22. Remains intubated and on inotropic support.

12 yo with panhypopituitarism involving DI admitted for hypernatremia in the setting of dehydration. Following rehydration with IVF, sodium has stabilized between 145-155 on vasopressin infusion.

6 yo with no sig PMH admitted s/p MVC with the following injuries: 1. Grade II splenic lac, 2. Right femur fracture, 3. Multiple abrasions. s/p ORIF of right femur. Remains in c-collar.

A plan by systems should follow. The systems should include those that are pertinent to your patient. There is no need for repetition.

For example, if you decide to include the diuresis plan in the CV system, you do not need to mention it in the FEN/GI system.

Remember, your note is geared toward the patient’s primary physician and subspecialists. It is not as necessary to mention the details of the plan. It is more important to mention the overall goals and how you anticipate achieving them.

Examples:

- the patient will need to remain NPO until return of bowel function. The patient may need supplemental nutrition with TPN if he remains NPO beyond 4-5 days.
- patient has been on narcotics and benzos for a prolonged period of time and is at risk for withdrawal as we titrate down his infusions. We will plan to titrate the midazolam infusion first, supplement with enteral lorazepam and monitor for signs/symptoms of withdrawal.
• Avoid use of the word “stable” when possible. A patient’s vital signs can be stable without being appropriate and therefore does not convey any information to your reader. Instead, use “hemodynamically appropriate” or “Hgb unchanged at range of 10-11”.

• Avoid use of the phrase “as tolerated”. Instead define specific parameters.
  o For example, “titrate vent rate for pH > 7.25 and < 7.4” or “titrate supplemental oxygen to maintain SpO2 > 93%”.

• Avoid the use of the word “wean” when possible. Instead use “titrate” or “titrate down”.

• Use generic drug names when possible.

• Use medical terminology such as “hypertension” instead of “elevated BP” or “thrombocytopenia” instead of “low platelets”.

• DO NOT “cut and paste” the note each day. This sets you up for contradictions and unnecessary repetition.
PALS Algorithms

1. PULSELESS ARREST
   - BLS Algorithm: Continue CPR
   - Give oxygen when available
   - Attach monitor/defibrillator when available

2. Check rhythm

3. Shockable
   - VF/VT
     - Give 1 shock
       - Manual: 4 J/kg
       - AED: >1 year of age
       - Use pediatric system if available for 1 to 8 years of age
       - Resume CPR immediately
     - Give 5 cycles of CPR
       - Check rhythm
       - Shockable
     - Continue CPR while defibrillator is charging
       - Give 1 shock
         - Manual: 4 J/kg
         - AED: >1 year of age
         - Resume CPR immediately
         - Give epinephrine
           - IV/IO: 0.01 mg/kg
             (1:10,000: 0.1 mL/kg)
           - Endotracheal tube: 0.1 mg/kg
             (1:10,000: 0.1 mL/kg)
           - Repeat every 3 to 5 minutes

4. Not Shockable

5. Check rhythm
   - Shokable

6. If asystole, go to Box 10
   - If electrical activity, check pulse. If no pulse, go to Box 10
   - If pulse present, begin postresuscitation care

7. Give 5 cycles of CPR

8. Continue CPR while defibrillator is charging
   - Give 1 shock
     - Manual: 4 J/kg
     - AED: >1 year of age
     - Resume CPR immediately
     - Consider antiarrhythmics (eg, amiodarone 5 mg/kg IV/IO or lidocaine 1 mg/kg IV/IO)
     - Consider magnesium 25 to 50 mg/kg IV/IO, max 2 g for torsades de pointes
     - After 5 cycles of CPR, go to Box 5 above

9. Asystole/PEA

10. Resume CPR immediately
    - Give epinephrine
       - IV/IO: 0.01 mg/kg
         (1:10,000: 0.1 mL/kg)
       - Endotracheal tube: 0.1 mg/kg
         (1:10,000: 0.1 mL/kg)
       - Repeat every 3 to 5 minutes

11. Check rhythm
    - Shokable

12. If asystole, go to Box 10
    - If electrical activity, check pulse. If no pulse, go to Box 10
    - If pulse present, begin postresuscitation care

13. Go to Box 4

During CPR

- Push hard and fast (100/min)
- Ensure full chest recoil
- Minimize interruptions in chest compressions
- One cycle of CPR: 15 compressions then 2 breaths; 5 cycles = 1 to 2 min
- Avoid hyperventilation
- Secure airway and confirm placement.
- After an advanced airway is placed, rescuers no longer deliver “cycles” of CPR. Give continuous chest compressions without pauses for breaths. Give 8 to 10 breaths/minute. Check rhythm every 2 minutes.

- Rotate compressors every 2 minutes with rhythm checks
- Search for and treat possible contributing factors:
  - Hypovolemia
  - Hypoxia
  - Hypertension
  - Hypo-arrhythmia
  - Acidosis
  - Alkalosis
  - Hypoglycemia
  - Hypothermia
  - Toxins
  - Tamponade, cardiac
  - Tension pneumothorax
  - Thrombosis (coronary or pulmonary)
  - Trauma
Common PICU medication dosages

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
<th>Instructions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>0.1 mg/kg</td>
<td>max 6 mg for 1\textsuperscript{st} dose, 12 g for 2\textsuperscript{nd} dose</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>5 mg/kg IV load over 25 min</td>
<td>150 mg max</td>
</tr>
<tr>
<td>Atropine</td>
<td>0.02 mg/kg IM/IV/ET</td>
<td>0.1 mg min and 1 mg max</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>30 mg/kg IV</td>
<td>central only</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>50-100 mg/kg IV</td>
<td></td>
</tr>
<tr>
<td>Dextrose</td>
<td>1 g/kg</td>
<td>1 ml/kg D50, 2 ml/kg D25, 5 ml/kg D10</td>
</tr>
</tbody>
</table>
| Dopamine        | D 1-3 mcg/kg/min  
β 3-10 mcg/kg/m  
α 10-20 mcg/kg/m |                                       |
| Enalapril       | 0.05 – 0.25 mg/kg PO   |                                       |
| Epinephrine     | 0.01 mg/kg IV  
0.1 mg/kg ET  
β 0.01-0.1 mcg/kg/m  
α 0.1 - 1 mcg/kg/min | 1:10,000, 0.1ml/kg  
1:1000, 0.1 ml/kg |
| Fentanyl        | 25 mcg/kg IV  
1 mcg/kg/hr |                                       |
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose/Rate</th>
<th>Maximum Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydralazine</td>
<td>0.1 – 0.2 mg/kg IV</td>
<td>max 20 mg/day</td>
</tr>
<tr>
<td>Magnesium</td>
<td>25-50 mg/kg IV</td>
<td>2 g max</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.05 mg/kg IM/IN/IV 0.05 mg/kg/hr</td>
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</tr>
<tr>
<td>Milrinone</td>
<td>0.25 – 1 mcg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>0.05 mg/kg IV</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>β 0.01-0.1 mcg/kg/m α 0.1 - 1 mcg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>1 – 2 meq/kg IV</td>
<td></td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.5 – 40 mU/kg/hr</td>
<td></td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.1 mg/kg IV</td>
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</table>
Respiratory
1. Airway / intubation
   a. Decision to intubate is clinical and based on poor respiratory effort, difficulty oxygenating or ventilating, need for decreased oxygen consumption, unable to protect airway, altered mental status, need for anesthesia, etc.
   b. Equipment to prepare: SOAP ME
      i. Suction: Yankauer and flexible catheter hooked to suction canister and ready to use
      ii. Oxygen source: bag and appropriate size mask, pre-oxygenate patient
      iii. Airway equipment: ETT size = (16 + Age) / 4 for uncuffed tube, subtract 0.5 size for cuffed tube, have other sizes available; check bulb on laryngoscope
      iv. Pharmacy: choose a sedative and a paralytic, make sure you have secure IV access, premedicate with atropine in patients < 5 yo
      v. Monitoring Equipment: have SpO2, HR, cycle BP q3 min, CO2 detector
   c. Confirmation: auscultate bilateral breath sounds, check end tidal CO2, obtain CXR
   d. Troubleshooting: DOPE
      i. Displaced ETT (Are you still intubated? Do you have end tidal?)
      ii. Obstructed ETT (Have you tried suctioning?)
      iii. Pneumothorax (Have you listened for bilateral breath sounds?)
      iv. Equipment failure (Do you have disconnected vent tubing?)
e. Rapid sequence intubation: do not apply PPV, do not place NG prior to intubation, hold cricoid pressure until ETT placement is confirmed
f. Assess for air leak around the ETT q12 hrs. A leak should be audible between 20 and 30 cmH$_2$O
g. Obtain CXR qAM to check ETT placement

2. Infant Airway
   a. larynx more cephalad (C2-3 vs. C4-5) and anterior
   b. larger tongue (and head), larger tonsils and adenoids.
   c. hyoid bone attached to thyroid cartilage $\rightarrow$ epiglottis protrudes into airway (omega shaped)
   d. cricoid cartilage—narrowest point $\rightarrow$ cylindrical shaped larynx (glottis is narrowest in adults)

3. Predicting Difficult Airway
   a. mallampati class > 2
   b. thyromental distance < 3 fingerbreaths (< 6 cm)
   c. submandibular space, neck movement, mouth opening
   d. cricoid pressure and BURP: move thyroid cartilage back, up, right, and with pressure

4. Mechanical ventilation
   a. Conventional ventilation
      i. Pressure control (set PIP) vs Volume control (set tidal volume): choose PEEP, Rate, FiO$_2$ in both modes. Monitor variable parameter (PIP in volume control and tidal volume in pressure control). Aim for tidal volume of 5-7 ml/kg.
      ii. Pressure Control: improved patient comfort, improved delivery of breath to lung units with different compliance and resistance
      iii. Volume Control: guaranteed minute ventilation
b. PRVC is a pressure mode. The desired tidal volume is set but the pressure used per breath varies and is based on patient involvement and preset limit. So, the tidal volume per breath may actually vary significantly.

c. APRV and HFOV are modes that are used to improve oxygenation. HFOV is also used for air leaks and for pulmonary hemorrhage

i. HFOV settings include FiO$_2$ and MAP that affect oxygenation and Amplitude and Frequency that affect ventilation
   1. Set the MAP about 2 cmH$_2$O above previous MAP and adjust based on oxygenation parameters, lung expansion on CXR
   2. Set the Amp based on patient “jiggle” and ventilation parameters
   3. Frequency: 1 Hz = 60 cycles/sec. lower limit is 3 Hz. the lower the frequency, the lower the CO$_2$
   4. exhalation is active
   5. cytokine production is ↓
   6. good for air leak: prevents overdistension of more compliant alveoli

ii. APRV settings include FiO$_2$, Phigh set near MAP on conventional vent, Plow set at zero, Thigh and Tlow set to maximize recruitment.

d. Monitor serum pH. Aim for pH 7.3-7.4. Correlate at least one serum pH with continuous End tidal monitoring

5. Minute ventilation = tidal volume x RR. In volume control, tidal volume is guaranteed. So if you want to decrease CO$_2$ by 25%, increase RR by 25%

6. Titrating vent settings toward extubation: patient must demonstrate ability to initiate spontaneous breaths,
achieve adequate tidal volumes per each spontaneous breath, have intact bulbar reflexes, have adequate muscle strength to initiate breaths, and be sufficiently alert.

7. Hypoxia differential diagnosis
   a. Low partial pressure of oxygen (high altitude)
   b. Hypoventilation
   c. Shunt (intracardiac or intrapulmonary)
      i. Calculating $P_{A\text{O}_2}$: $[\text{FiO}_2 \times (\text{Patm} - \text{P}_\text{H}_2\text{O} \text{vapor})] - [\text{PaCO}_2/RQ]$ where RQ is 0.8
      ii. Normal A-a gradient is 10 accounting for physiologic shunting from bronchial and thebesian circulations
   d. Alveolar dead space
      i. Physiologic dead space = anatomic dead space + alveolar dead space
      ii. Anatomic dead space = ventilated but not perfused areas (vent tubing, large airways)
      iii. Alveolar dead space = lung disease
      iv. Physiologic dead space = $(\text{PaCO}_2 - \text{P}_{\text{ETCO}_2}) / \text{PaCO}_2$
   e. Diffusion abnormality
   f. Shunt, alveolar dead space, and diffusion abnormality give you an A-a gradient $> 10$

8. Monitoring
   a. SpO$_2$: Can be falsely low with nail polish, methylene blue, indocyanine green, Methemoglobin levels $< 15\%$, increased venous pulsations, and other light sources. Can be falsely high with CO poisoning, vasoocclusive disease in HgbSS, and methemoglobinemia $> 15\%$. Hyperbilirubinemia does not affect the SpO$_2$.
   b. SaO$_2$: estimated on blood gas, measured on co-oximetry
9. ARDS: endothelial cell injury and increased permeability. Acute onset pulm edema, decreased lung compliance, hypoxia, and bilateral infiltrates on CXR
   a. 3 stages
      i. Exudative phase (week 1)
      ii. Proliferative phase (week 2-3)
      iii. Fibrotic phase (week 4 and beyond)
   b. Lung protective strategies
      i. minimize volutrauma (maintain tidal volumes 6-8 ml/kg)
      ii. minimize barotrauma (maintain PIP < 30 cmH₂O)
      iii. minimize oxygen trauma (maintain FiO₂ < 0.6)
      iv. Achieve this by permissive hypercapnea (maintain pH > 7.25) and maintain SpO₂ > 88%
   c. Follow oxygenation parameters
      i. P/F ratio = PaO₂/FiO₂. P/F ratio < 200 is ARDS
      ii. Oxygenation index = FiO₂ x MAP / PaO₂

10. Pulmonary Hypertension
    a. Desaturation followed by bradycardia and hypotension
    b. If patient is intubated, sedate and paralyze
    c. 100% oxygen as a pulmonary vasodilator
    d. Correct acidosis (acidosis causes vasoconstriction)
    e. Inhaled Nitric Oxide: increases cGMP to cause pulmonary vasodilation

11. Asthma
    a. Inhaled beta agonists: bronchial smooth muscle relaxation, increase ciliary clearance
       i. Albuterol, levalbuterol
    b. Systemic beta agonists: bronchodilate and vasodilate
       i. Terbutaline 10 mcg/kg bolus followed by 0.2 mcg/kg/min. salbutamol. Isoproterenol.
c. Subcutaneous beta agonists: epinephrine 0.01 ml/kg of 1:1000

d. Corticosteroids: anti-inflammatory, act on leukocytes, eicosanoids, and vascular endothelium
   i. Methylprednisone 2 mg/kg IV followed by 1 mg/kg q12
   ii. Inhaled steroids: beclomethasone, fluticasone, budesonide

e. Anticholinergics: inhibit parasympathetic bronchoconstriction
   i. Ipratropium bromide

f. Methylxanthines: unclear mechanisms. Possibly via adenosine receptor antagonism or decreasing cGMP

g. Magnesium sulfate: inhibits Ca channels to cause smooth muscle relaxation. can cause hypotension, respiratory depression, heart block

h. Heliox: Helium/Oxygen mixture. Helium is colorless, odorless. lowest density of any gas (except hydrogen) and higher viscosity than nitrogen so produces more laminar flow. requires tight fitting mask for delivery and an oxygen requirement <30% for effectiveness
**Cardiovascular**

1. Cardiopulmonary interactions
   a. During spontaneous inspiration, negative intrathoracic pressure decreases venous return to the heart. This is the basis of pulsus paradoxus.
   b. During PPV, positive intrathoracic pressure decreases venous return to the heart. Thus, it is important to assure adequate preload prior to initiating PPV. Patients with obstructive lung disease such as asthma also have decreased venous return which is dramatically worsened upon initiation of PPV and can lead to cardiac arrest.
   c. PPV decreases afterload on left ventricle. This decrease is most clinically relevant in heart failure. However, PPV also increases afterload to the right ventricle which can be clinically relevant even in a heart with normal function.

2. Oxygen Delivery: The main goal in critical care medicine is to provide adequate oxygen delivery to meet tissue requirements and balance that with oxygen consumption in order to maintain aerobic metabolism and organ function.
   a. Oxygen delivery = cardiac output x arterial oxygen content
   b. Cardiac output depends on HR and SV. SV depends on preload, contractility, and afterload
   c. Arterial oxygen content = bound content + dissolved content = (1.36 x Hgb x SaO2) + (0.003 x PaO2)
   d. Note that the dissolved content does not contribute significantly to overall arterial oxygen content especially when the hemoglobin is normal.
   e. The bound content is affected by the oxyhemoglobin dissociation curve. Factors that shift the curve to the
right make it easier for oxygen to unload to the tissues.

f. Note that if you want to increase BP, you can either increase the cardiac output, increase the SVR, or both. If just SVR is increased, then the heart is working against a resistance and the cardiac output can actually decrease.

g. Normally, 1 L O2/min is delivered to tissues and 250 ml O2/min is consumed by the tissues giving an oxygen extraction ratio of 25%. You can see this number as the SvO2 on a VBG obtained from a central venous line positioned near the RA. Assuming an SaO2 of 100% in a patient with no lung disease, the SvO2 should be approximately 75%. In sepsis, this number will often be lower implying an increase in oxygen consumption.

h. Assessment of oxygen delivery and consumption: end organ perfusion (skin perfusion, mental status, UOP), vital signs including NIRS, ABG with pH, base deficit and lactate, Hgb, SvO2

3. Shock: inadequate oxygen delivery +/- increased oxygen consumption or maldistribution of blood flow. Evidenced
by tachycardia and normotension if compensated, hypotension if uncompensated.


b. Types of shock

<table>
<thead>
<tr>
<th></th>
<th>pulse</th>
<th>SV R</th>
<th>CO</th>
<th>CVP</th>
<th>PCWP</th>
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</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>thready</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Septic</td>
<td>thready</td>
<td>↓</td>
<td>↓ unless compensated</td>
<td>↓ (can ↑ w/ cardiac dysfxn)</td>
<td>↓ (can ↑ w/ cardiac dysfxn)</td>
</tr>
<tr>
<td>Distributive (anaphylaxis or spinal shock)</td>
<td>bounding w/ widened pulse pressure</td>
<td>↓</td>
<td>↑</td>
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</tr>
<tr>
<td>Cardiogenic</td>
<td>thready</td>
<td>↑</td>
<td>↓ (can be nml with diastolic dysfxn)</td>
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</tr>
</tbody>
</table>

i. Cardiogenic shock can be an issue with

1. Preload (dilated cardiomyopathy, regurgitant valves)

2. Contractility (systolic or diastolic dysfunction)
   a. Diastolic dysfunction can maintain BP and CO for a longer period of time

3. Afterload (aortic coarctation, pulmonary or aortic stenosis, pHTN, etc)

4. Obstruction (tamponade, tension pneumothorax)

ii. Treatment
1. Volume resuscitate hypovolemic, septic, and distributive shock  
   a. Use isotonic fluids (NS or LR)  
   b. Reassess after each bolus  
   c. After 60 ml/kg, consider supplementing with inotrope/pressor support but do not stop providing isotonic fluids if the patient is still in shock  
   d. Epinephrine is often used for cold shock and NE for warm shock  
   e. For shock unresponsive to fluids and pressors, consider vasopressin and steroid replacement  

2. Broad spectrum antibiotics for suspected septic shock  


4. Pressors and Inotropes  
   a. work via the Autonomic Nervous System.  

<table>
<thead>
<tr>
<th></th>
<th>Preganglionic receptor (and neurotransmitter)</th>
<th>Postganglionic receptor (and neurotransmitter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parasympathetic Nervous System</td>
<td>Nicotinic (Ach)</td>
<td>Muscarinic (Ach)</td>
</tr>
<tr>
<td>Sympathetic Nervous System</td>
<td>Nicotinic (Ach)</td>
<td>Noradrenergic (Noradrenaline)</td>
</tr>
</tbody>
</table>

b. noradrenergic receptors are further subdivided into alpha, beta, and dopa receptors.  

c. $\alpha$ is pure vasoconstriction. A pure $\alpha$-agonist will increase SVR and can cause some reflex decrease in
cardiac output, although it has no direct effect on the heart itself.
d. $\beta$ is much more complicated in that it has several effects:
   i. Inotropy (desired)
   ii. Chronotropy (generally not desired as it increases $\text{MvO}_2$)
   iii. Mild vasodilation through $\beta_2$ effects (explains how this can work against SVR)
e. An $\alpha$ and $\beta$ agonist can often work together in blood pressure control. The $\alpha$-agonist will increase BP by increasing SVR. The $\beta$-agonist will increase cardiac output.

<table>
<thead>
<tr>
<th>Pure $\alpha$</th>
<th>$\alpha &gt; \beta$</th>
<th>$\alpha = \beta$</th>
<th>$\beta &gt; \alpha$</th>
<th>Pure $\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenylephrine</td>
<td>NE</td>
<td>Epi</td>
<td>dobutamine</td>
<td>Isoproterenol</td>
</tr>
</tbody>
</table>

- In an effort at simplification, anything to the left of middle in the chart above is a pressor and anything to the right of the middle is an inotrope.
- Epinephrine at doses > 0.1 mcg/kg/min becomes almost all $\alpha$
- Norepinephrine may have enough $\beta$ to counteract the reflex decrease in CO but remember that any $\alpha$ vasoconstricts more than any $\beta$ vasodilates
- Dopamine is difficult to put on the chart as it is the most dose dependent. At < 10 mcg/kg/min, it is mainly an inotrope. At > 10 mcg/kg/min, it is mainly a pressor. It also has a tendency to increase PVR.
- Remember that calcium is a short acting inotrope and vasoconstrictor. There are many other inotropes including milrinone and methylene blue

5. CHD
a. Acyanotic lesions
   i. Increased pulmonary blood flow (L → R shunts, present with CHF, FTT)
   ii. Decreased pulmonary blood flow
b. Cyanotic lesions
   i. Increased pulmonary blood flow
   ii. Decreased pulmonary blood flow
c. Pre-op management
   i. Secure diagnosis (Echo, cardiac cath) and management plan
   ii. Evaluate for syndrome associated with cardiac defects such as Trisomy 21 (AV canal defects, pulmonary HTN), Turners (coarctation), DiGeorge (conotruncal and aortic arch defects), etc.
   iii. Ensure adequate oxygen delivery
   iv. Prevent acidosis
   v. PGE for ductal dependent lesions
      1. Ductal dependent pulmonary blood flow: monitor PaO2
      2. Ductal dependent systemic blood flow: monitor end organ perfusion, limit supplemental oxygen
d. Post-op management
   i. Know the surgical procedure and the maneuvers required to achieve them (for example, was a ventriculotomy required? Was the ASD closed primarily or with a patch?)
   ii. Know the results of the post-op TEE for assessment of heart function and residual defects
   iii. Obtain CXR immediately post-op to assess placement of new lines and tubes
iv. Dysrhythmias: obtain EKG immediately post-op as new baseline, assess tachycardias with atrial EKG

v. Bleeding: monitor platelet count, Hgb, coags (PT, PTT, Fibrinogen). Goal Hgb > 10 (> 13 for single ventricle physiology)

vi. Adequate oxygen delivery: monitor pH, base deficit, lactate, NIRS. Assess volume status and maintain adequate preload

vii. Know of any alterations in loading conditions:
   1. Residual shunts or stenotic valves may have an adverse effect on preload
   2. Shunt closures or PA banding may have an adverse effect on afterload

viii. Monitor electrolytes closely post-op. Patients should be on ¾ maintenance IVF without potassium as the urine output will diminish over time and there is potential for renal injury. Avoid TPN immediately post-op.

ix. For single ventricle physiology (post-op Norwood and Glenn): maintain SpO₂ 75-85% as a non-invasive way to maintain PaO₂ 40-45 mmHg as a way to optimize pulmonary and systemic blood flows (Qp:Qs). Qp:Qs is determined using saturations: \[\frac{(SaO_2 – SvO_2)}{(SpvO_2 – SpaO_2)}\]
   where SpvO₂ is assumed to be 100% if there is no lung disease and SpaO₂ is assumed to be SaO₂ in complete mixing lesions.

x. For the first 12-18 hours post-op, any decrease in urine output may indicate low cardiac output, poor cardiac function, or hypovolemia. After the first 18-24 hours, a low urine output may indicate the need for diuresis.
xi. Patients with a BT shunt or CVL may be at risk for clots and may need a non-titrated, prophylactic heparin infusion at 10 U/kg/hr (discuss with fellow/attending)

xii. Tamponade: triad of muffled heart sounds, JVD, and hypotension. On EKG, look for low voltages. Clinically look for electrical alternans, narrow pulse pressure and tachycardia. Management involves supporting preload and relieving the tamponade with pericardiocentesis

xiii. Chylothorax: normally, thoracic duct takes lymph, fatty acids, and lymphocytes from the small intestine to the subclavian vein.
   1. Causes: surgical trauma, high CVP, congenital malformations, thrombosis
   2. Contents of chylothorax: triglycerides, fat soluble vitamins, T-lymphocytes, proteins (albumin, immunoglobulins), all blood components except RBCs and platelets
   3. Management: MCTs still meet the fatty acid needs of the body but are absorbed directly into the portal blood. Octreotide is a long acting somatostatin analog that decreases splanchnic blood flow. Surgical duct ligation for drainage > 100 ml / year of age for 5 days or persistent drainage for 2 weeks.

e. Cardiopulmonary bypass
   i. On bypass, patient is receiving fully oxygenated blood to all organs. However, the blood flow is non-pulsatile and the blood is being continuously exposed to tubing. Therefore, there is potential for end organ injury with prolonged bypass. There is also an inflammatory response that peaks 10-12
hours post bypass that manifests as low cardiac output, capillary leak, decreased lung complaints, etc. Patients also have thrombocytopenia and/or platelet dysfunction and hyperglycemia post-op.

ii. Cross clamp prevents blood flow inside the heart and maintains non-pulsatile flow to the rest of the organs. Watch for signs of cardiac ischemia post-op.

iii. Deep hypothermic circulatory arrest (DHCA): Patient is cooled to 18°C and there is no flow through the bypass circuit. This allows a bloodless field where the surgeon can also remove all clamps and tubing that sits in the way for intricate repairs external to the heart. Data suggests that up to 30 min of DHCA can be safe. Monitor for end organ injury including renal and neurologic injury.

f. Repairs for management of the single ventricle
   i. Stage I Norwood with BT shunt or Sano: The pulmonary artery trunk is used to create a neo-aorta. A shunt is placed to direct part of the systemic blood flow to the PAs. Remember that shunt flow is determined by the balance of PVR and SVR. Thus, blood from the IVC/SVC enters the RA, then the RV, then the neo-aorta where part enters the pulmonary system and part continues down the descending aorta. From the pulmonary veins, blood enters the LA and then the RA through an ASD.

   ii. Stage II bidirectional Glenn: The BT shunt or Sano is taken down. The SVC is disconnected from the RA and attached directly to the PA. Blood from the SVC must passively enter the pulmonary circulation.
iii. Stage III Fontan: The IVC is now also disconnected from the RA and attached to the PA often via an extracardiac conduit. This conduit may or may not be fenestrated to provide a right to left shunt to maintain cardiac output in the setting of pulmonary disease or pHTN. All blood flow to the lungs is now passive and the RV functions as the systemic ventricle. SpO2 should now be in the 90s.

6. Pacemaker Notation

<table>
<thead>
<tr>
<th>Chamber Paced</th>
<th>Chamber Sensed</th>
<th>Response to sensing</th>
<th>Rate Modulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>R for rate modulation</td>
</tr>
<tr>
<td>A = atrium</td>
<td>A = atrium</td>
<td>T = triggered</td>
<td></td>
</tr>
<tr>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td>I = inhibited</td>
<td></td>
</tr>
<tr>
<td>D = dual</td>
<td>D = dual</td>
<td>D = dual</td>
<td></td>
</tr>
</tbody>
</table>

Ex: VVIR pacer implies ventricle is paced, ventricular impulses are sensed, inhibited from pacing if pulse is greater than the programmed threshold, is rate responsive (adjusts rate according to patient’s activity)

7. Extracorporeal Membrane Oxygenation (ECMO) or Extracorporeal Life Support (ECLS)
   a. Requires the presence of a reversible disease process
   b. Requires the use of systemic heparin (screen with head ultrasound for any intraventricular hemorrhage)
   c. Types
      i. VV ECMO (veno-venous): provides pulmonary support only. Often employs one cannula with two lumens placed via the right IJ into the RA.
ii. VA ECMO (veno-arterial): provides pulmonary and cardiac support. Employs two catheters. The venous catheter is directed into the RA via the right IJ and the arterial cannula is directed into the ascending aorta via the right common carotid. The right carotid artery is then ligated. The cannulas can also be positioned directly in the RA and aorta via an open chest.

d. Cardiac output is determined by pump flow and LV output. Pump flow is determined by the amount of venous blood withdrawn from the patient which is dependent on cannula size and systemic venous return. Avoid hypovolemia, pneumothorax, and tamponade. LV output is determined by patient’s heart function.

e. Monitoring
   i. Ask perfusionist about the pump (presence of clots, membrane oxygenator failure)
   ii. Patient ABG evaluates the blood passing through the circuit and through the pulmonary circulation. The circuit ABG and VBG assess the adequacy of the membrane function and of the oxygen delivery to the tissues.
   iii. Examine patient’s perfusion, lines, tubes, sites of entry
   iv. Monitor for excessive bleeding, heparin dose required
   v. Daily CXR to assess lung fields, position of lines and tubes

f. Emergency management: If there is a problem with the circuit, disconnect the patient from the circuit (clamp the venous line first, then unclamp the bridge, then clamp the arterial line), increase the ventilator
settings to the predetermined emergency settings, proceed to routing PALS protocols.
FEN/GI

1. IVFs
   a. Isotonic fluids include NS and LR
   b. normal saline can cause a non-anion gap metabolic acidosis when given in significant amounts
   c. lactated ringers has lower chloride and sodium concentrations compared to NS

2. Potassium
   a. Hyperkalemia: manifests as EKG changes with peaked T waves initially (K 6-8 meq/L) that progresses to widened QRS, then disappearance of the P wave, and finally a sine wave. The etiology can range from acidosis to tumor lysis to renal failure.
      Treatment:
      i. Make sure to d/c exogenous sources (IVF, TPN, K supps)
      ii. Calcium will not decrease the potassium level but will act quickly to increase threshold potential
      iii. Medications that shift K intracellularly (temporary solution, takes approx 30 min): sodium bicarbonate, beta agonists, insulin (0.2 U/kg), glucose (1 g/kg)
      iv. Increase elimination (permanent solution but can take longer): Polystyrene sulfonate with sorbitol (Kayexalate – 1-2 g/kg PO or PR), dialysis
   b. Hypokalemia: manifests as weakness and ileus with EKG changes with flat T waves, U waves. Remember that potassium supplements can inadvertently cause hyperkalemia. PO supplementation is safer but should be given with feeds. KCl dose is 0.5 – 1 meq/kg.

3. Sodium
   a. Hypernatremia
i. Effects: cellular dehydration from osmotic fluid shifts, cerebral dehydration (brain cell volume ↓ 10-15%) → venous sinus thrombosis, bridging vein rupture (SDH, ICH), high pitched irritable cry, rhabdomyolysis, hyperreflexia, hyperglycemia from peripheral insulin resistance

ii. Causes
1. Too much intake: Na polystyrene
2. Not enough fluid (↑ serum osm): hypothalamic d/o or abnormal thirst function
3. Water loss
   a. In kidney (urine osm < 800, plasma osm > 275)
      i. Renal water loss: DI, diuretics
      ii. Osmotic water loss: ↑ glucose, mannitol, ↑ solute feeds
   b. Outside of kidney (urine osm > 800)
      i. Insensible losses
      ii. GI losses

iii. Management: rapid drop in Na leads to cerebral edema. Aim to decrease the Na by 12 meq/day. If the hypernatremia is chronic, body compensated with idiogenic osmoles.

b. Hyponatremia
   i. Causes
      1. Serum osm > 280
         a. No osmolar gap → pseudohyponatremia (lipids and proteins)
         b. Osmolar gap → translational hyponatremia (glucose, mannitol, maltose, etc)
            i. Corrected Na = measured Na + [((gluc – 100)/100) x 2]
      2. Serum osm < 280
a. Urine osm < 100: polydipsia, iatrogenic water intake
b. Urine osm > 100: impaired water excretion
   i. Hypovolemia: V/D, burns, CSW
   ii. Euvolemia: SIADH, hypothyroidism
   iii. Hypervolemia: CHF, nephritic syndrome, cirrhosis
ii. Management: rapid increase in Na leads to central and extrapontine demyelination (quadriplegia, seizures, pseudobulbar palsy). Aim to increase Na by 12 meq/day unless symptomatic. If symptomatic, correct rapidly to Na 125 meq/L. Assuming ongoing isotonic fluid losses, 1 ml/kg/hr of 3% will increase Na by 1 meq/L/hr.

4. Calcium
   a. Hypercalcemia
      i. Causes: hyperparathyroid, adrenal insuff, MEN, thyrotoxicosis, Vit D and A excess, immobilization, thiazides, lithium, theophylline, glucocorticoid withdrawal

<table>
<thead>
<tr>
<th></th>
<th>Phos</th>
<th>1,25(OH) -Vit D</th>
<th>PTH</th>
<th>Urine Ca</th>
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<tbody>
<tr>
<td>Immobilization</td>
<td>Nml/↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Malignancy</td>
<td>↓</td>
<td>Nml/↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Abrupt glucocorticoid w/d</td>
<td>Nml/↑</td>
<td>Nml</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>Nml/↑</td>
<td>↑</td>
<td>↓</td>
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</table>
### Familial Hypercalciuric Hypercalcemia

<table>
<thead>
<tr>
<th></th>
<th>Nml/↓</th>
<th>Nml/↑</th>
<th>Nml/↑</th>
<th>↓</th>
</tr>
</thead>
</table>

- **ii.** Signs/symptoms: Bone pain, Kidney stones, Polyuria/polydipisia, Constipation, ↓ reflexes, EKG: heart block, wide QRS, short QT
- **iii.** Management: IVFs, furosemide, calcitonin, bisphosphonates, edentate disodium chelation

#### b. Hypocalcemia

- **i.** Causes: hypoparathyroid, Vit D deficiency, alkalosis (iCa ↓ 0.42 mmol/L for every 0.1 mmol/L change in pH), hyperalbuminemia (corrected Ca = measured Ca + (0.8 x (4-albumin))), drugs (AEDs, barbs), DKA, TLS, rhabdomyolysis
- **ii.** Signs/symptoms: perioral paresthesias, Trousseau, Chvostek, bronchospasm, laryngospasm, stridor, seizures, cramps, EKG: prolonged QT, inverted T waves
- **iii.** Management: CaCl comes already ionized but must be given centrally. CaGluconate can be given peripherally but must be conjugated in the liver

#### 5. Magnesium

- **a.** Hypermagnesemia
- **i.** Causes: renal failure (initially low Mg from tubular injury then high Mg from decreased elimination)
- **ii.** Signs/symptoms: anticholinergic signs (inhibits prejxnal release of ACh due to displacement of memb bound Ca), ↓ reflexes, hypotension, resp arrest, EKG: short QT (breaks torsades)
- **iii.** Management: CaGluc, lasix, dialysis
b. Hypomagnesemia
   i. Causes: ↓ intake, refeeding, malnutrition, hyperthyroidism, transfusion with citrated blood, acidosis shift, ↑ loss (lasix, ampho B, cisplatin, cyclosporin, V/D, laxatives, malabsorption, gent (toxic to proximal tubule where 30% Mg reabsorbed), volume expansion (SIADH, burns), sweating, hyperaldosteronism, ↓ albumin
   ii. Signs/symptoms: muscle weakness, seizures, VT, nystagmus, Trousseau/Chvostek, low K/Ca, suppressed parathyroid hormone, EKG: ↑ PR, ↑ QRS, ↑ QT, flat T

6. Phosphorus
   a. Hypophosphatemia
      i. Causes: refeeding syndrome, malabsorption, respiratory alkalosis, β agonists, hypomagnesemia, antacids, sepsis, burns, renal losses
      ii. Effects
         1. ↓ 2,3 DPG → decreased oxygen delivery
         2. ↓ ATP → hemolytic anemia
         3. ↓ cardiac contractility
         4. Muscle weakness
         5. Paresthesias and neuropathy
         6. Seizures
         7. Platelet dysfunction
         8. Liver failure

7. NPO guidelines
   a. Nothing by mouth at least 2 hours before
   b. Clears/breast milk at least 4 hours before (8 oz limit)
   c. Simple solid foods, formula at least 6 hours before
   d. Heavy and fatty foods at least 8 hours before

8. Gastritis prophylaxis with famotidine for patients who are NPO, on post-pyloric feeds, or on corticosteroids
Endocrine

1. DKA
   a. State of relative or absolute insulin deficiency with increases in glucagon, cortisol, and growth hormone leading to hepatic gluconeogenesis, hepatic and skeletal muscle glycogenolysis, and hepatic lipolysis
   b. Patients will be hypovolemic but remember that rapid shifts in osmolality with aggressive fluid administration may result in cerebral edema
   c. The increased glucose and osmolality causes the brain to grab idiogenic osmoles to avoid shrinkage. Thus, lack of adequate compensation and/or aggressive treatment of DKA results in cerebral edema
   d. Risk factors for development of cerebral edema include age < 5, male patients, new onset diabetes, and protracted illness.
   e. Note their initial mental status and order q1 neuro checks for any altered mental status
   f. Monitor acidosis, hypokalemia, and hypophosphatemia with Chemistries q2
   g. Monitor ketonuria with U/As each void and serum ketones (Acetest for acetoacetate and β hydroxybutyrate)
   h. Monitor glucose by finger stick q1
   i. Remember that you will have a pseudohyponatremia while hyperglycemic
      i. Corrected Na = measured Na + [(serum glucose – 100) / 100] x 1.6
   j. Start insulin gtt at 0.1 units/kg/hr and do not titrate while patient is acidotic and ketotic
   k. 2 bag correction method
      i. NS + 20 meq/L KPhos + 20 meq/L KCl (initial fluids for any BG > 250)
ii. D10NS + 20 meq/L KPhos + 20 meq/L KCl (start for any BG < 250)
l. Run total fluids above maintenance fluid requirements (discuss with PICU fellow)
m. Do not treat acidosis with sodium bicarbonate as this may cause a paradoxical CNS acidosis, impair oxygen extraction, further hypokalemia, and hypocalcemia.

2. DI  
a. Causes: CNS infection, TBI, intraventricular hemorrhage, neurosurgery, brain death  
b. Signs/Symptoms: polyurea, ↓urine osm, ↑ serum osm and Na, hypovolemia  
c. Management: fluid resuscitation, replace UOP > 3 ml/kg/hr 1:1 with NS, electrolyte correction, vasopressin infusion 1-3 mU/kg/hr and titrate to UOP

3. SIADH  
a. Causes: CNS infection, TBI, drugs, PPV  
b. Signs/Symptoms: ↑ urine osm, ↓ serum osm and Na, low UOP  
c. Management: fluid restriction, furosemide, 3% saline for symptomatic patients

4. Adrenal Insufficiency  
a. Causes  
   i. Primary: CAH, Addisons  
   ii. Secondary: insufficient ACTH production or destruction of pituitary (tumors, autoimmune)  
   iii. Tertiary: suppression from exogenous steroids  
b. Signs/Symptoms: weight loss, dehydration, N/V, hypotension, shock unresponsive to fluids/inotropes, ↓Na, ↑K, ↑Ca, acidosis  
c. Cortisol stim test:  
   i. Obtain blood sample for cortisol and ACTH  
   ii. Inject synthetic ACTH
1. low-dose short test: inject 1 µg
2. conventional-dose short test: inject 250 µg
   iii. obtain blood sample 60 minutes after injection
d. Management: fluid resuscitation, hydrocortisone 50 mg/m2/d divided q8, electrolyte correction
Heme/Onc
1. DVT
   a. Risk factors: central venous lines, cancer, L-asparaginase, trauma, immobilization, pregnancy, obesity, smoking, HgbSS, vasculitis, sepsis, dehydration, cardiomyopathy
2. Blood replacement
   a. Older pRBCs from the blood bank have undergone metabolic, chemical, and molecular changes including decreased NO containing groups, loss of deformability, decreased 2,3 DPG, decreased ATP
3. Transfusion associated disease
   a. Acute lung injury
      i. Immune response directed against HLA antibodies that occurs within 30 minutes to 6 hours after transfusion manifested as respiratory distress and hypoxia with bilateral fluffy infiltrates on CXR. May also see fever and hypotension.
      ii. Management: for future transfusions, consider obtaining washed pRBCs, newer pRBCs and minimizing the number of donors
   b. Transfusion associated GVHD
      i. Associated with pRBCs, platelets, granulocytes but not FFP
      ii. Occurs in immunocompromised patients or those with HLA haplotype issues (1st degree relatives)
      iii. Fever, rash, cough, cholestatic jaundice, lymphopenia
      iv. Diagnosis: HLA analysis of lymphocytes
      v. Prevention: gamma irradiation, HLA matched blood products
4. Leukocytosis
   a. WBC count > 100,000/mL
b. Hyperviscous state leading to pulmonary and CNS findings

c. Management: avoid diuresis, avoid pRBCs. Leukapherese with tumor lysis precautions

5. Tumor Lysis Syndrome

a. Physiology: patients with a high tumor burden or those undergoing chemotherapy are at risk for massive lysis of cells.

\[
\text{Nucleic Acid} \rightarrow \text{Hypoxanthine} \rightarrow \text{Xanthine} \rightarrow \text{Uric Acid (excreted in urine)} \rightarrow \text{allantoin} \quad \text{(more soluble in H2O)}
\]

\[
\text{Xanthine} \quad \text{Oxidase} \quad \text{Urate} \quad \text{Oxidase}
\]

b. At risk patients include those with T cell lymphoma or leukemia that present with leukocytosis.

c. Monitor q6 labs for hyperkalemia, hyperuricemia, and hyperphosphatemia

d. Management: aggressive IVF hydration, alkalinize urine, mannitol. Allopurinol competitively inhibits xanthine oxidase and will prevent further production of uric acid. Rasburicase is a recombinant urate oxidase. Avoid giving calcium to prevent calcium-phosphorus binding.

6. Anticoagulation

a. Heparin induced thrombocytopenia: type I is not immune mediated and is transient, resolving without intervention. Type II is immune mediated antibodies formed to platelet factor 4. All sources of heparin must be removed from the patient. Platelet counts will normalize within one week of discontinuation but the risk for thrombotic events can persist for weeks. Management involves use of heparinoids or direct thrombin inhibitors.
Infectious Disease

1. Hospital acquired infections
   a. Ventilator associated pneumonia: more likely to occur in the first week with early onset VAP usually from gram positive organisms and late onset VAP from gram negative organisms
   b. Central line associated blood stream infection

2. Necrotizing Fasciitis
   a. Necrosis of subcutaneous tissue with mix of aerobic and anaerobic organisms. Single organisms are usually GAS. Oral anaerobes such as fusobacteria can especially cause necrotizing fasciitis of the head and neck. Management is with beta lactam or beta lactamase inhibitors (Pip/Tazo). If GAS is suspected, treat with penicillin and clindamycin.

3. Staph Toxic Shock
   a. Can occur with any Staph infection with 2-3 days of prodrome preceding fever and erythroderma, followed by desquamation in 1-2 weeks resulting from TSST-1 and enterotoxins
   b. 25% of S. aureus strains are toxigenic but Blood culture is only positive in 5-15% for S. aureus

4. Strep Toxic Shock
   a. Excruciating pain, confusion, hypoalbuminemia resulting from strep pyogenic exotoxins A and B
   b. Blood culture positive in 50%
   c. Mortality rate 5 x that of staph toxic shock
   d. Treatment: vancomycin + cefuroxime. Clindamycin can be used as an adjunt. Consider IVIg.

5. Meningitis
a. Neonates are at risk for GBS, E.coli. Children are at risk for S. pneumo, N. meningitides, Hib

<table>
<thead>
<tr>
<th></th>
<th>WBC count</th>
<th>%PMNs</th>
<th>Glucose</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0-5</td>
<td>0-15</td>
<td>45-65</td>
<td>20-45</td>
</tr>
<tr>
<td>TB/fungal</td>
<td>25-100</td>
<td>Lymph/monos</td>
<td>30-45</td>
<td>100-500</td>
</tr>
<tr>
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<td>25-500</td>
<td>&lt; 50</td>
<td>45-65</td>
<td>50-100</td>
</tr>
<tr>
<td>Bacterial</td>
<td>&gt; 100</td>
<td>90</td>
<td>&lt; 40</td>
<td>&gt; 150</td>
</tr>
<tr>
<td>Aseptic/Partially treated</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


c. N. meningitidis: encapsulated gram negative diplococcus with humans as the only natural host
   i. Serogroup B causes the most cases but is not included in the quadrivalent conjugate vaccine
   ii. Complications result from malfunction of the coagulation system from endotoxin release

d. HSV meningitis: EEG may show periodic lateralized epileptiform discharges (PLEDs) early in the course. Head CT may show temporal lobe edema. Diagnosis: HSV PCR.

e. High CSF protein can also result from prolonged seizures and disrupted blood brain barrier with albumin-cytologic dissociation (malignancy, Guillain Barre, multiple sclerosis)
Neurology
1. Sedation and paralytic medications
   a. Opiates
      i. Morphine, Fentanyl, Hydromorphone, Nalbuphine
      ii. Side effects include respiratory depression, itching, hypotension especially if combined with a benzodiazepine, histamine release with morphine
      iii. Reversal agent = naloxone. Remember that naloxone has a shorter ½ life than your opiates so the dose may need to be repeated
      iv. Watch for withdrawal signs and symptoms (diarrhea, sweating, agitation) if your patient is coming off an opiate infusion after a greater than 5-7 day exposure
      v. Uses
         1. Post-op pain control – for extubated patients, use morphine 0.05-0.1 mg/kg/dose q3-4 hours PRN
         2. For older post-op patients – consider morphine PCA using demand dosing only (no continuous without discussing with PICU fellow)
         3. For intubated patients, typically start fentanyl infusion at 1 mcg/kg/hr with an equivalent PRN (i.e. 1mcg/kg/dose q1 PRN) combined with a benzodiazepine
      vi. For itching, consider a mu antagonist/kappa agonist such as nalbuphine
   b. Benzos
      i. Midazolam, Diazepam, Lorazepam
      ii. Watch for propylene glycol toxicity with lorazepam (solvent). Watch for benzyl alcohol toxicity with continuous infusion of midazolam (antibacterial).
iii. Side effects include respiratory depression, hypotension
iv. Watch for withdrawal signs and symptoms (diarrhea, sweating, agitation) if your patient is coming off a benzodiazepine infusion after a greater than 5-7 day exposure
v. Uses
   1. For intubated patients, typically start midazolam 0.05 mg/kg/hr or lorazepam 0.1 mg/kg IV q4 with an equivalent PRN (i.e. midazolam 0.05 mg/kg/dose q1 PRN or lorazepam 0.1 mg/kg/dose q4 PRN)
   2. For posterior spinal fusion and similar surgeries, use diazepam 0.05-0.1 mg/kg/dose q6 PRN muscle spasm
vi. Reversal agent = flumazenil
c. Barbiturates
   i. Thiopental: rapid onset. Decreases cerebral oxygen consumption and ICP. Side effects: cardiac depression, bronchospasm.
d. Propofol
   i. GABA agonist and NMDA-R inhibitor
   ii. Very lipid soluble so quickly redistributes. No active metabolite. Hepatic metabolism
   iii. Contraindications: egg yolk allergy, metabolic disorder
   iv. Side effects: decreases inotropy. Hypotension. Impairs free fatty acid use and mitochondrial activity leading to propofol infusion syndrome (refractory metabolic acidosis, rhabdomyolysis, hepatomegaly)
e. Chloral hydrate
i. Metabolized by liver to its active metabolite trichloroethanol.
ii. ½ life 4-6 hours, excreted in urine
iii. Side effects: arrhythmias, withdrawal, agitation

f. Ketamine
i. NMDA-R antagonist, increases sympathetic stimulation, bronchodilates, increases cerebral blood flow
ii. Side effects include tachycardia, hypertension. Large doses can cause laryngospasm and apnea. Muscarinic effect of drooling.
iii. Contraindications: catecholamine depleted state such as septic shock, increased ICP

g. Dexmedetomidine
i. α 2 agonist
ii. biphasic response with transient hypertension initially with bolus followed by hypotension and bradycardia
iii. potential for hypothermia in neonates with decrease in brown fat use

h. Etomidate
i. Carboxylated imidazole derivative, GABA agonist
ii. Effect lasts 5-10 minutes with ½ life of 75 minutes
iii. No cardiac effects. Decreased cerebral oxygen consumption
iv. Side effects: adrenal insufficiency (reversibly inhibits 11 β hydroxylase and 17 α hydroxylase), decreased seizure threshold

i. Depolarizing neuromuscular blocker (succinylcholine)
   i. Side effects
      1. Fasciculations, constipation, myoglobinemia
      2. Increased intraocular, gastric, and intracranial pressures
3. Hyperkalemia from increased acetylcholine receptors from 48 hours up to 16 months s/p any disease process resulting in large mass of denervated muscle (burns, crush injuries, neuromuscular dx)

4. Bradycardia, malignant hyperthermia, Guillain Barre

ii. Succs effects can be prolonged with plasma pseudocholinesterase deficiency, liver disease, pregnancy, or with repeated dosing

j. Non-depolarizing neuromuscular blockers: competitive inhibitors of acetylcholine. Muscles of the eyelids and fingers are the first to be affected and the last to recover. Muscles of the intercostals, diaphragm, and larynx are the last to be affected and the first to recover.

i. Aminosteroids: vecuronium, pancuronium, rocuronium. Roc is safe to give in renal failure and can be given IM. All aminosteroids have some vagolytic activity with panc having the most.

ii. Benzylisoquinolines: Cisatracurium, mivacurium. not vagolytic but can cause histamine release and bronchospasm. Cisatracurium uses Hofmann degradation. Mivacurium is degraded via plasma cholinesterase.

2. Status epilepticus

a. Goal is to control seizures ASAP as the longer the seizure continues, the harder it is to break

b. Remember that it’s still ABCs first

i. Might need to establish an airway if the patient’s mental status is altered, the patient has poor cough/gag, the patient is apneic
ii. Might need to support blood pressure – consider phenylephrine infusion
c. Video EEG is necessary to assess for subclinical seizures
d. Look for treatable causes (electrolyte imbalance, hypoglycemia, etc)
e. Start with lorazepam 0.1 mg/kg
f. If the patient is still seizing, continue to provide lorazepam but quickly add a second and then a third agent:
   i. Fosphenytoin 20 mg/kg
   ii. Phenobarbital 20 mg/kg
   iii. Levetiracetam 20 mg/kg
g. Consider pentobarbital infusion with goal to achieve burst suppression on EEG
3. Glasgow Coma Scale
   a. Used in ER setting to assess neurologic status
   b. Scored from 3-15 with a score of 9-12 suggesting moderate head injury and a score below 9 suggesting severe head injury

<table>
<thead>
<tr>
<th>Motor</th>
<th>Verbal</th>
<th>Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 – none</td>
<td>1 – none</td>
<td>1 - none</td>
</tr>
<tr>
<td>2 – extension, decerebrate</td>
<td>2 – incomprehensible sounds, ininconsolable</td>
<td>2 – opens eyes to pain</td>
</tr>
<tr>
<td>3 – abnormal flexion, decorticate</td>
<td>3 – inappropriate words, irritable, inconsistently consolable</td>
<td>3 – opens eyes to command/speech</td>
</tr>
<tr>
<td>4 – flexion withdrawal to pain</td>
<td>4 – confused, cries but consoles</td>
<td>4 – opens eyes spontaneously</td>
</tr>
<tr>
<td>5 – localize, withdraw to touch</td>
<td>5 – oriented, coos, smiles</td>
<td></td>
</tr>
<tr>
<td>6 – obeys commands</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. TBI and stroke / neuroprotective guidelines
   a. Goal is to prevent secondary brain injury
i. Avoid hyperthermia (place continuous rectal probe, may need around the clock acetaminophen)

ii. Avoid hyponatremia (must place all patients on isotonic fluid (normal saline))

iii. Avoid hyper and hypoglycemia (older children should have NS only, younger children can have D5NS, everyone should have frequent glucose checks)

iv. Treat seizures aggressively

b. Order q1 hour neuro checks
c. If patient has an EVD, monitor ICPs
   i. Call Fellow and Neurosurgery for any sustained ICP > 20 mmHg
d. Signs of increased ICP: Cushing’s triad (bradycardia, hypertension, agonal respirations), dilated or unequal pupils.
e. Cerebral perfusion pressure (CPP) = Mean arterial pressure – ICP (or CVP if it’s higher like in SVC syndrome or Glenn physiology). Avoid hypotension.
f. Exam:
   i. evenly distributed high pressure: CN VI palsy, HA, vomiting;
   ii. in early herniation: CN III palsy, poor pupillary response, posturing
g. Types of herniation
   i. Transtentorial results from any supratentorial mass or cerebral edema
   ii. Uncal results when the mass is lateralized. anteromedial portion of the hippocampus herniates over the edge of the tentorium

h. Cerebral edema
   i. Types
1. Vasogenic: increased capillary membrane permeability secondary to endothelial injury. Examples include meningoencephalitis, vasculitis, tumors, DKA, and hypertensive encephalopathy
2. Interstitial: transudation of CSF into the brain from increased ICP. Examples include communicating and non-communicating hydrocephalus
3. Cytotoxic: intracellular edema secondary to brain cell (oligodendrocytes, astrocytes, neurons) injury seen in the gray matter. Examples include diffuse axonal injury, HIE, and chronic hepatic encephalopathy

ii. Imaging: head CT reveals hypodense cortical gray matter and subcortical white matter with obliterated cisterns around the midbrain
iii. Steroids: helpful in vasogenic edema only to inhibit lipid peroxidation
i. Mannitol vs. 3% saline
   i. Mannitol: Start with 0.25 g/kg. ↓ edema, alters blood rheology \(\rightarrow\) improved blood flow to ischemic areas. Its diuretic effect can lead to hypotension which will adversely decrease CPP
   ii. 3% saline: Start with 3-5 ml/kg bolus. Can continue as an infusion. Monitor serum osm and maintain < 360 mOsm/L
j. Corticosteroids do not benefit head trauma, HIE, or stroke. only ↓ vasogenic edema around tumors and abscesses
k. Cerebral autoregulation
1. C-spine precautions: maintain c-spine precautions until the spine is cleared by trauma surgery. Remember that c-spine cannot be clinically cleared in a patient with altered mental status, distracting injuries, or a patient that is non-verbal. These patients will likely need a c-spine MRI prior to clearance. Remember also that patients under age 8 are at risk for SCIWORA.

m. Stroke
   i. Types
      1. Ischemic/thrombotic: worse long-term outcome
      2. Hemorrhagic: higher immediate mortality
   ii. Specific treatment
      1. Maintain NPO status
      2. Elevate HOB to 30 degrees for hemorrhagic stroke and lay patient flat for ischemic stroke
      3. Anticoagulation should only be considered in conjunction with a stroke team

5. Pediatric brain death criteria
   a. Varies from hospital to hospital.
   a. The 1987 Ad Hoc Task Force guidelines for determination of brain death in children involves:
a. Rule out any reversible causes of coma such as high levels of sedating/paralyzing medications, electrolyte disturbances, etc
b. Patient must not be hypothermic (defined by individual hospital)
c. Physical exam includes absence of brain and brainstem function
   i. Midposition or fully dilated/non-reactive pupils
   ii. Absence of oculocephalic and oculovestibular responses
   iii. Absence of movement of bulbar musculature, corneal, gag, cough, sucking, and rooting reflexes
   iv. Flaccid tone and absence of posturing or other brainstem mediated movement
   v. Absence of respiratory effort on apnea test
d. Spinal reflexes such as myoclonus, triple flexor withdrawal of lower extremities to pain, and hiccups do not preclude brain death
b. Observation period according to age
   a. Less than 7 days: cannot be done
   b. 7 days to 2 months: 2 examinations and EEGs 48 hours apart
   c. 2 months to 12 months: 2 examinations and EEGs 24 hours apart
   d. > 12 months: 2 examinations 12 hours apart
c. Confirmatory tests include
   a. Radionuclide imaging
   b. Cerebral angiography