Conflict of Interest Disclosures

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I have no conflicts of interest to disclose

Agenda

Distribution of Total Body Fluid
Fluid resuscitation vs. Maintenance IV Fluid
Crystallloid vs. Colloid
Osmolarity of IV Fluids
Hypertonic Saline and Hyponatremia
Hypotonic Fluid
Hypernatremia
Hypo- and Hyperkalemia
Other Electrolytes and Shortages
Enteral and Parenteral Nutrition

SAQ #1: Fluids

- 74yo female with cough, temp 102, and lethargic
- BP 72/40, HR 115, UO 10 ml/hr, WBC 18K, Cr 1.7 (baseline 1.2), wt 72kg
- After 500ml NS IV bolus, BP 80/46, HR 113
- CXR – pneumonia
- PMH – CAD, arthritis

Which is best treatment?

A. Furosemide 40mg IV
B. 5% albumin 500ml + NE for SBP ≥ 90
C. 1000ml bolus with D5W/.9% NaCl
D. 1000ml bolus with 0.9% NaCl

To answer SAQ #1, think about…

- How do we recognize intravascular volume depletion?
- How do IV fluids distribute in total body fluid?
- What IV fluids can be used to optimize intravascular volume?
**Total Body Fluid**

- 60% Intracellular (IC)
- 40% Extracellular (EC)

**Total Body Water**

- 60% Intracellular (IC)
- 40% Extracellular (EC)
- 75% Interstitial (IS)
- 25% Intravascular

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**Intravascular Space**

- Not exactly "extracellular" because there are cells in this space (RBC's)
- The extracellular fluid in the intravascular space is known as plasma, and is about ~3 L
- There's an additional ~2L of fluid in RBC's, making the total blood volume about 5L
- Intravascular fluid is analogous to the fluid in your car's gas tank

**Intravascular volume makes us go...**

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**The Starling Curve**

![Graph](image)

**Measures of Intravascular Volume**

(e.g., CVP, LVEDP, MAP)

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**Distribution of IV Crystalloid**

- 0.9% NaCl or LR
  - Na+ and Cl+ do not freely enter cells
  - Therefore, evenly distribute in EC
  - Each L provides ~250ml into intravascular space

- D5W
  - Dextrose is metabolized to H2O and CO2
  - Water crosses any membrane
  - Each L provides 100 ml into intravascular space
Distribution of IV Colloid

- Colloids are too large to cross capillary membrane (theoretically) – therefore 500ml infused provides 500ml intravascular volume replacement
- PRBC
- Pooled human plasma (albumin 5%, plasma protein fraction or plasmanate)
- Semi-synthetic glucose polymers (dextran)
- Semi-synthetic hydroxyethyl starch (hetastarch)

Page 150 B3 and Table 1

Distribution of IV Colloid

- Unlike 5% albumin, 25% albumin will result in volume expansion 5 x’s the volume administered
  - 100ml 25% results in 500ml intravascular volume replacement.
  - Use caution because fluid redistribution can cause cellular dehydration
  - May be useful in patients with ascites or pleural effusions where fluid redistribution is the goal

Page 150 B3 and Table 1

Fluid Resuscitation

- Intravascular fluid depletion commonly due to:
  - Hemorrhagic shock
  - Septic shock
- S/S usually occur when 15% (~750ml) lost
  - Hypotension, tachycardia, ↓ UO, Improvement after fluid bolus
  - Reduced CO and organ hypoperfusion
- Need prompt intravascular fluid replacement
  - Through a central venous catheter
  - Administer as a 500-1000ml bolus, reassess and continue as long as S/S are improving

Page 150-151 and Table 2

Crystalloids vs. Colloids

- Crystalloids (NS, LR) are recommended
  - LR is historically used in trauma patients because the lactate is metabolized to bicarbonate and theoretically useful for metabolic acidosis…but no evidence of superiority over NS
- Colloids “seem” better than crystalloids based on distribution properties
  - No evidence to demonstrate improved outcomes
  - Higher cost

Page 151 C6-C7

Colloids: Popular but Controversial

- Consider after fluid resuscitation with crystalloid (usually 4-6 L) has failed to achieve hemodynamic goals or when clinically significant edema limits further administration of crystalloid
  - e.g., Pulmonary edema causing hypoxia
- Considered if albumin < 2.5 and required large volume of crystalloid and have a relative contraindication for semi-synthetic colloid
  - e.g., Avoid hetastarch if ↑ risk bleeding

Page 151 C7

Colloids: Popular but Controversial

- Consider albumin (preferably 25%) in conjunction with loop diuretics for patients with clinically significant edema and an albumin concentration < 2.5 when appropriately dosed diuretics are ineffective
  - e.g., pulmonary edema or effusion causing respiratory failure
  - Although, theoretically loop diuretics (highly protein bound) will bind to albumin resulting in reduced efficacy.

Page 151 C7
**Maintenance IV Fluid**
- Not for patients with S/S of intravascular volume depletion
  - When asked for a stat D5W/0.45%NaCl + KCl 20 for a hypotensive patient, consider alternative
- Typical maintenance IV fluid is D5 0.45% NaCl + KCl 20 - 40meq/L
- Omit KCl if elevated K or kidney failure
- 0.9% NaCl, LR, or colloids are generally not appropriate maintenance IV fluids
- Evaluate IV fluids daily

**How much fluid?**
- Remember for resuscitation, administer 500-1000ml bolus, then re-evaluate.
  - Continue until S/S no longer improve
- For daily fluid maintenance, many use 1500 ml for first 20kg, then 20ml/kg thereafter (~2500ml/day)
  - Adjust rate based on I/O, estimated insensible loss (e.g., fever)

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**Plasma Osmolality**
- Posm ~ 275-290 mOsm/kg
- Primary determinant of Posm is sodium salts (hence 2 x 140 = 280 ~ Posm)
- Changes in Posm cause fluid shifts across cell membranes
  - Increased Posm causes cellular dehydration
  - Decreased Posm causes cellular swelling
  - Can cause permanent neurologic damage
- Major changes in serum sodium can result in changes in Posm

**Osmolarity of IV Fluids**
- Isotonic
  - No osmotic gradient, no fluid shift
- Hypertonic IV Fluid
  - Cell dehydration/shrinkage
- Hypotonic IV Fluid
  - Cell over hydration can occur if < 150 mOsm/L
  - RBC swelling = hemolysis
  - Brain cell swelling = cerebral edema/herniation
Calculate Osmolarity of IV Fluid

0.9% NaCl = 0.9 gm/100ml = 9gm/L
MW of NaCl = 58.5gm/mol
Osmotic Coefficient NaCl = 0.93

\[
\begin{array}{c}
\text{9 gm} \\
\text{L}
\end{array} \times \begin{array}{c}
\text{1 mol} \\
\text{58.5gm mol}
\end{array} \times \begin{array}{c}
\text{2 osm} \\
\text{mol}
\end{array} \times \begin{array}{c}
0.93 \\
\text{1 osm}
\end{array} \times 1000\text{mosm} = 287 \text{ mOsm/L (isotonic)}
\]

Page 154, Table 6

Patient Case #3

- 72yo female with HTN started HCTZ 3 weeks ago; with dizziness, fatigue, nausea
- Na 116, Wt 60kg, BP 86/50, HR 122
- Which regimen is best?
  A. NS 100ml/hr
  B. NS 500ml bolus
  C. 3% NaCl 60ml/hr
  D. 23.4% NaCl 30ml bolus as needed

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Patient Case #4

- What is the treatment goal for the 1st 24 hours in correcting her serum Na? (initial value is 116 mEq/L)
  A. Goal Na 140
  B. Goal Na 132
  C. Goal Na 126
  D. Maintain 116 - 120

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Hypertonic Saline and Hyponatremia

Hypertonic Saline

- Typically 3%, 7.5%, or 23.4%
- Osmolarity ~ 950, 2400, 7400 respectively
- Administer via central line
- Use
  - Traumatic Brain Injury to reduce elevated ICP and/or increase BP (3%, 7.5%, 23.4%)
  - Acute Symptomatic Hyponatremia (usually 3%)
    - Acute = symptom onset within 48h or less
    - Symptomatic = lethargy, psychosis, seizure, coma

Page 155 III A-B

Symptoms of Hyponatremia

<table>
<thead>
<tr>
<th>Serum Sodium (mEq/L)</th>
<th>Symptoms</th>
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<tbody>
<tr>
<td>120-125</td>
<td>Nausea, malaise</td>
</tr>
<tr>
<td>115-120</td>
<td>Headache, lethargy, obtundation, Unsteadiness, confusion</td>
</tr>
<tr>
<td>110-115</td>
<td>Seizure, coma</td>
</tr>
</tbody>
</table>

Page 159, Table 11
Hypertonic Saline is NOT for:
- Chronic asymptomatic hyponatremia
  - e.g., SIADH usually chronic and treated with fluid restriction
- Hyponatremia secondary to DKA
  - As glucose is corrected, Na will too
- Hyponatremia associated with CHF
  - Usually chronic and asymptomatic
  - If symptomatic, caution regarding volume overload with hypertonic saline

Avoid this Error
- 150 mEq Sodium Bicarbonate mixed in 850 ml IV Fluid (typically to prevent RCN).
- If mixed in 0.9% NaCl, the result is equivalent to 3% sodium (hypertonic).
- I suggest using D5W instead.
- Sterile water could be used as well, but I avoid this due to risk of error.

Safe Use of Hypertonic Saline for Symptomatic Hyponatremia
- Goal is a SMALL but QUICK rise in Na by 0.75-1 mEq/L/hr to a concentration of 120 mEq/L, then slow to 0.5 mEq/L/hr
- Can be achieved using 3% NaCl 1-2 ml/kg/hr or 250ml bolus over 30 min
- Treat until:
  - Symptoms stop
  - Safe, serum Na range (120-125 mmol/L)
  - Obtained max safe change in serum Na...

Complications of HS:
Avoid Rapid Change in Na+
- Max change is 10-12 mmol/L in 24 hours
- Rapid correction of serum sodium can cause central pontine myelinolysis or osmotic demyelination syndrome
  - Characterized by paraparesis, quadriplegia, coma
  - Permanent neurologic damage
  - Highest risk is patients with chronic hyponatremia (some things don’t need fixin’)

Other Considerations in Hyponatremia
- Correct hypokalemia first
  - Hypokalemia can cause hyponatremia due to an intracellular shift of Na to maintain electroneutrality
  - As K+ is replaced, serum Na will also increase due to an extracellular shift
- Also consider volume status...

Hyponatremia and Volume

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<td>Example</td>
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<td>SIADH</td>
<td>Fluid loss</td>
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<tr>
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<td>Restrict H2O</td>
<td>Restrict H2O</td>
<td>Fluid resuscitation</td>
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<tr>
<td>Underlying Cause</td>
<td>Vaptan</td>
<td>Vaptan</td>
<td></td>
</tr>
</tbody>
</table>
Hypovolemic Hyponatremia

- Hypovolemia is a potent stimulus for ADH secretion which perpetuates hyponatremia…once restore volume, ADH secretion slows
- Treat with fluid resuscitation using 0.9% NaCl
- Once ADH secretion is reduced, serum Na can rapidly correct, so…
- Careful monitoring is required to prevent overly rapid correction (a medication error that can cause permanent neurologic damage!)

Patient Case #3

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Hyponatremia and Volume

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<td></td>
<td></td>
<td>Fluid resuscitation</td>
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Vaptans

- Vasopressin receptor antagonists
- AVP secreted by hypothalamus to regulate osmolality
- IV conivaptan, oral tolvaptan
- Safe and efficacious for normovolemic (SIADH) or hypovolemic hyponatremia (CHF, Cirrhosis) persisting despite fluid restriction
  - Facilitate aquaresis (electrolyte-free water excretion)
  - Increase Na
  - Alleviate symptoms and reduce weight in CHF

Vaptans

- No evidence of improved clinical outcomes in prospective randomized controlled studies
  - i.e., fall prevention, hospitalization, hospital length of stay, mortality
- Substrates and inhibitors of CYP 450 3A4 isoenzymes, so monitor for drug interactions
  - 3A4 inhibitors could increase vaptan effect leading to a rapid increase in serum Na
- Fluid restriction in combination with a vaptan during the first 24 hours can also increase risk of overly rapid correction of serum Na
  - Avoid fluid restriction in first 24 hours of vaptan use

Here’s an opposite problem: Hypernatremia in Patient Case # 6

- 74yo female on Jevity TF’s at 60 ml/hr for 8 days through PEG, 50kg
- Recent CVA, non-communicative
- Na 142 → 149 → 156 → 163 on days 3 → 8
- Treatment?
Hypotonic Fluid and Hypernatremia

Hypotonic Fluid

- Albumin 25% diluted with SW to make albumin 5% has osmolarity of ~ 60 mOsm/L
  - Associated with hemolysis and death
- 0.2% NaCl ("quarter saline") is hypotonic with an osmolarity of 68 mOsm/L
- Avoid IV fluid with osmolarity < 150 mOsm/L

Page 157-158 IV A-C

Appropriate Use of Hypotonic IV Fluid

Is Hypernatremia a good reason to use hypotonic saline?

- Generally patients with hypernatremia need water, not NaCl (as in 0.2% NaCl)
- But we NEVER give water IV...
- So if possible, give water by mouth...
- Or if NPO, give free water IV (D5W)
  - Dextrose is metabolized to CO2 and water, so that provides free water and can be given IV

Page 157-158 Section IV B2

What to do with those orders for "quarter saline"?

- This is a potentially fatal error
- Generally ordered in error
- Eliminate risk by changing to
  - D5W (best case scenario if need IV)
  - D5 / 0.2% NaCl (a recommendation that’s more likely to succeed)
  - D2.5 / 0.2% NaCl (for those who complain about risk of hyperglycemia with 5% dextrose)
  - Addition of KCl will also increase osmolarity
  - Enteral water (best case scenario)

Page 158 Section IV C

Treatment of Hypernatremia

- Rapid correction of chronic asymptomatic hypernatremia is a potentially fatal medical error.
- In symptomatic hypernatremia, serum Na+ should be corrected by NMT 10-12 mEq/L/day
- Replace water deficit

Page 162 F1-3

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Treatment of Hypernatremia

- Estimate Water Deficit in patients with hypernatremia:
  - 0.5 to 0.6 x LBW x [(Na/140) -1] for men
  - 0.4 to 0.5 x LBW x [(Na/140) -1] for women

- Replace water deficit slowly following guidelines for safe changes in serum Na
- If serum Na is decreasing too rapidly, can administer D5W / 0.2% or D5W / 0.45% NaCl to slow rate of decrease

Hypernatremia in Patient Case # 6

- 74yo female on Jevity TF’s at 60 ml/hr for 8 days through PEG, 50kg
- Recent CVA, non-communicative
- Na 142 → 149 → 156 → 163 on days 3 → 8

- Sterile water IV 80 ml/hr
- D5W IV 80 ml/hr
- D5W 0.2% NaCl 80 ml/hr
- Water per PEG 200ml q6 hours

By the way…

- Hypernatremia in the setting of Case 6 is completely preventable and should be considered a medication error
- Can be prevented by being proactive with enteral administration of water in patients receiving TF’s (especially if 1.5-2 kcal/ml)
  - Check the TF label for water content
  - Make sure patients receive at least 1 ml water per calorie administered (will need extra per FT)
  - Especially in patients who can’t communicate thirst

Potassium (K+)

- The primary intracellular cation
- Balance K+ between IC and EC compartments is maintained by
  - β2-stimulation promotes cellular uptake
  - Insulin promotes cellular uptake
  - Plasma K+ concentration can cause passive shifts in or out of cells

Hypokalemia (K+ < 3.5 meq/L)

- Seldom caused by reduced K+ intake because of ↓ kidney excretion
- Causes of hypokalemia
  - A shift of K+ into cells can occur with
    - ↑ pH
    - Insulin or carbohydrate load
    - β2 stimulation (stress, drugs)
    - Hypothermia
  - GI loss (V, D, fistula, laxative abuse)
  - Urinary loss (diuretics)
  - ↓ Mg++ (increase renal loss of K)

Treatment Pearls of Hypokalemia

- How much?
  - There is no precise calculation of K+ loss based on a plasma K+ concentration
  - K+ replacement is guided by plasma K+

- How fast?
  - 10-20 mEq/hr, max 20-40 mEq/hr requires continuous EKG monitoring (regardless of route)

- Route?
  - Oral KCL should be considered if no symptoms
  - If peripheral IV, max concentration is 60 meq/L
Treatment Pearls of Hyperkalemia

- Is it real? Does it fit the clinical scenario?
  - K⁺ can be artificially elevated if traumatic venipuncture (hemolysis)
  - Artificially elevated if serum rather than plasma K⁺ (due to K⁺ release during coagulation)
- Is the patient experiencing severe muscle weakness or EKG changes (peaked t-waves, wide QRS) or is K⁺ > 6.5 meq/L (VF can be first sign)?
  - No – Kayexalate
  - Yes…

Urgent Treatment of Hyperkalemia

1. Calcium gluconate 1-2gm IV over 2-10 minutes can prevent hyperkalemia-induced arrhythmias (will come back to CaGluc)
2. Drugs that cause an intracellular shift of K⁺:
   - Insulin 10 units IV (with glucose to prevent hypoglycemia) – within ~60min
   - Sodium bicarbonate 50mEq – within 30-60 min although efficacy is disputed
   - Albuterol (β₂ agonist) – within 90 min inhaled; 40% won’t respond; consider use in combination with insulin

After Urgent Treatment, ↑ K⁺ excretion with…

- Diuretics to ↑ renal excretion (if kidneys functioning)
- Cation-exchange resin (Kayexalate) PO or as retention enema
  - Do not use sorbitol as vehicle for oral or rectal use due to risk of colonic necrosis
  - Caution in kidney or heart failure due to Na retention
- Dialysis

More on Calcium use in Hyperkalemia

- Calcium gluconate can be administered peripherally and is preferred over calcium chloride because of reduced risk of tissue necrosis
  - 10ml of 10% = 1gm (90mg elemental Ca)
- Calcium chloride can be used if central IV access is available, but adjust dose
  - 10ml = 1gm (270mg elemental Ca)
- Calcium will not reduce K⁺
- Avoid calcium if digoxin used (↑ risk dig toxicity)

Replacement of Other Electrolytes

- Mg
  - Oral Mg limited by diarrhea
  - If symptomatic (tetany, seizure, HTN), administer 1-4gm IV slowly (1gm/hr) to avoid hypotension and/or increased renal excretion; if torsades can give IV push
- PO₄
  - Major cause of low PO₄ is refeeding syndrome; also during treatment of DKA
  - Prevent refeeding syndrome by supplementing with 15 mmol/L as egg phospholipids likely sufficient for most
- Ca
  - Don’t treat (or use PO) if asymptomatic hypocalcemia associated with low albumin
  - Asymptomatic hypocalcemia can be treated with oral calcium
  - Don’t give CaCl via peripheral IV (limb ischemia)

Electrolyte Shortages (not in text)

- Phosphate shortage (pg 168)
  - Reserve for pediatric and neonatal patients
  - Reserve for DKA, refeeding syndrome
  - IV fat emulsions contain 15 mmol/L as egg phospholipids…likely sufficient for most
- Calcium shortage (pg 169)
  - If no gluconate, don’t add Ca chloride to PN
  - Use multi-electrolyte products
  - Unknown if can dilute Ca chloride and administer peripherally (but people are doing this)
Enteral and Parenteral Nutrition

Enteral Nutrition: Indication

- Used in hemodynamically stable patients at risk of malnutrition in whom it is anticipated that oral feedings will be inadequate for at least 1-2 weeks
- Most well-nourished adults without excessive metabolic stress can tolerate little to no nutrition for up to 7 days

Enteral Nutrition - Delivery

- Gastric vs. duodenal feeding tubes
  - Improved tolerance with duodenal
  - Reduced risk of aspiration with duodenal
  - Duodenal tubes clog easier
  - Must use continuous infusion (not bolus) for duodenal feeding
- Continuous infusion is most common in hospitalized patients
  - Reduced risk of aspiration compared to bolus feedings

Enteral Formulations

- Intact or polymeric formulas used for normal digestive processes
- Elemental formulas used for malabsorptive processes (e.g., short bowel)
  - Typically 1-2 kcal/ml
- Specialty formulas
  - Renal: concentrated, reduced protein/lytes
  - Pulmonary: more fat and less CHO
  - Diabetic: more fat, less CHO, fiber

Enteral Nutrition Example

A 60kg patient requires ~25 kcal/kg
60kg x 25 kcal/kg = 1500 kcal
Ultracal provides 1 kcal/ml
1500/24 = 62.5 ml/hr

Start Ultracal at 20 ml/hr and advance every 4-6 hours as tolerated to a goal rate of 62 ml/hr

Enteral Nutrition: Don’t forget to add water

- Ultracal provides 830 ml water/L, therefore if infusing 1500 kcal/day, need to add ~250 ml water extra daily
- Order 60-70ml water per feeding tube every 6 hours
Complications of EN

- Aspiration pneumonia
  - HOB 30-45 degrees
  - Hold if gastric residuals are > 250-500ml
  - Consider pro-motility agents
  - Duodenal feedings

- Diarrhea
  - More common with higher osmolarity enteral feedings
  - Consider other causes (antibiotics, infection, lactose intol, Mg++, sorbitol, etc)

Pro-Motility Agents

- Metoclopramide 5-20mg IV q6h
- Erythromycin 250mg IV q6-8h x 72 hours or until tolerating EN x 24 h
- Can combine, but monitor for diarrhea
- D/C if diarrhea
- Avoid prolonged use or prophylactic use due to increase risk of adverse effects

Enteral vs. Parenteral

- If the gut works, use it.
- Enteral feedings maintains integrity of GI mucosa and can prevent bacterial translocation
- Early enteral feeding improves outcomes in surgery patients.
- No evidence that early parenteral feeding improves outcomes.
- PN if unable to feed at least 7d with a nonfunctioning or inaccessible GI tract

Developing a PN Regimen

1. Calories: 25-35 kcal/kg/day
   - Applies to EN too
   - Some say less (“permissive underfeeding”)
   - If BMI > 30, give 22-25 kcal/kg based on IBW
2. Fluid
   - Typically 2500-3500 ml/day for maintenance
   - Do not use PN for fluid resuscitation

Developing a PN Regimen

3. Protein 0.8-2 g/kg/day
   - Less protein if not dialyzed; more if dialysis
   - Consider kcal from protein (4 kcal/gm)
   - Protein buffers 3-in-1’s so keep at 2.5-4%
4. Provide about 20-30% of calories as lipid (~10 kcal/g) ...or less is fine too
5. Provide rest of calories as dextrose (3.4 kcal/g)
   - Infuse no faster than 4-6 mg/kg/min (adults only)
     - Closer to 4 in patients with hyperglycemia

Central PN: 70kg Patient

- Total kcal 30 kcal/kg 2100 kcal
- Protein 1.5 g/kg 105 g (420 kcal)
- Lipid 25-30% of total 500 kcal

<table>
<thead>
<tr>
<th>Total</th>
<th>Protein</th>
<th>Lipid</th>
</tr>
</thead>
<tbody>
<tr>
<td>2100</td>
<td>105</td>
<td>420</td>
</tr>
<tr>
<td>-500</td>
<td></td>
<td>1180</td>
</tr>
</tbody>
</table>

- Dextrose: 1180 kcal / 3.4 kcal /g ~ 350 g
**Peripheral PN**
- Unlike central PN which is guided by patient’s nutritional needs...
- Peripheral PN is guided by final concentration of dextrose and amino acids in formula to maintain osmolarity < 900 mOsm/L
  - Dextrose 10% or less
  - AA 2.5-4%
- Electrolyte restrictions
  - Calcium ≤ 5 mEq/L
  - K 40 ≤ mEq/L

**Calcium – Phosphate Precipitation**
- Risk is low if Ca < 6 mEq/L & PO4 ≤ 30 mmol/L
- Calcium chloride ppt > calcium gluconate
- Final AA should be at least 2.5%
  - Forms soluble complexes with Ca and PO4
  - Buffer to maintain a lower pH
- As temp ↑, so does risk (so, refrigerate)
- Filters decrease risk of embolism (0.22 for 2-in-1, or 1.2 for 3-in-1)
- Phosphate is first electrolyte added, then mix well, then add Ca last

**Complications of PN**
- Catheter-related infection (S. aur, C. alb)
- Gut atrophy is linked to bacteremia
- Overfeeding
  - Hepatic steatosis
  - Hypercapnia
  - Hyperglycemia
  - Azotemia
- EFAD (impaired wound healing) if no lipid for ~ 1-3 weeks

**Complications of PN**
- Refeeding syndrome in acutely or chronically malnourished patients
  - Reductions in PO4, K, Mg
  - Cardiac and respiratory dysfunction
- Patients at risk (anorexia, alcoholism, cancer, chronically ill, poor intake x 1-2 wks, wt loss)
- Prevent by infusing ≤ 50% of calories and advancing over several days to goal
- Provide more PO4, K, Mg in 1st week, monitor daily and replace as needed

**Monitoring PN**
- Infection – temp, WBC, site
- Fluid – wt, edema, vitals, in/out, temp
- Prealbumin is useful for chronic nutrition support in patients who are not critically ill (normal 16-40 mg/dL)
- Blood glucose
- Electrolyte and acid-base status
- TG (withhold lipid if > 400 mg/dL)
- Readiness for enteral or oral nutrition

**Premixed PN (Not in Chapter)**
- 2-compartment bags containing AA in one compartment and dextrose in other are available in U.S. (Clinimix; has electrolytes)
  - Lipid can be added to container following mixing compartments or can be administered by Y-site
- Procalamine contains AA (3%), glycerin (4.3 kcal/g), and electrolytes in a single container does not meet needs of most patients due to insufficient protein and calories
- Insufficient evidence to show that customized PN is superior to standardized pre-mixed

**Calcium – Phosphate Precipitation**

**Complications of PN**

**Monitoring PN**

**Premixed PN (Not in Chapter)**

Premixed PN

**Pros**
- Fewer manipulations
- ↓ risk contamination
- ↓ compounding errors
- ↓ labor costs
- Good fit for stable pts
- Non-electrolyte formula available
- Good when AA shortage

**Cons**
- Still need additives (MVI, TE)
- Poor if fluid restriction
- Poor if high protein needs
- Only 1 or 2 L bags, so standardized admin times problematic
- Clinimix uses CaCl
- Additive stability??


Thank You

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Conflic of Interest Disclosures

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I have no conflicts of interest to disclose

Agenda

- Sepsis-Related Hemodynamics
- Initial Resuscitation
- Sepsis Definitions
- Vasopressors
- Antimicrobials
- Corticosteroids

- Other Critical Care Issues
- Hypothermia/Cardiac Arrest
- Analgesia & Sedation
- Delirium
- Hyperglycemia
- Stress Ulcer
- DVT Prophylaxis
- VAP Prevention
- Nutrition

Hemodynamics: Indicators of Volume

- Mean arterial pressure (MAP)
  - MAP = DBP + \(\frac{1}{3}(SBP-DBP)\)
  - Indicates global perfusion pressure
  - An MAP > 60 is needed for organ perfusion
  - Mostly DBP...coronary perfusion during diastole
- Central Venous Pressure (CVP) is the pressure in the thoracic vena cava near the right atrium
  - Reflects preload / intravascular volume
  - Heart function (CO) optimal at a CVP of 8-12
Indicators of Blood flow and Heart Function

- Cardiac output – amount of blood pumped per minute
  - Normal 4-8 L/min, but larger CO in larger people
- Cardiac index is CO standardized to BSA
  - Normal is 2.5-4 L/min regardless of body size
- CVP (and PCWP) can indicate heart function because as forward flow (CO) is impaired, then volume (and thus pressure) will increase

Indicators of O2 Transport / Use

- Lactic Acid – anaerobic metab / hypoxia
- Venous saturation (SvO2) indicates oxygen transport and use...How?
  - Normal arterial saturation is 98-100%
  - Normal venous saturation is 70-75%
  - Normal oxygen extraction ratio is 25-30%
- During hypoperfusion, extraction ratio is larger (lower SvO2) due to ↑ tissue demand for oxygen in a setting of ↓ supply
  - Low SvO2 despite fluid resuscitation can be a sign of insufficient CO

Indicators of vascular tone

- Systemic vascular resistance (SVR) is a calculated number, and not a measured number (like BP, CVP, CO)
- SVR can be used as a diagnostic tool, but does not necessarily need to be treated if high or low
- Treatment (with vasodilators or vasopressors) should be based on the clinical assessment (not SVR)

Shock

- Septic Shock
- Hypovolemic Shock (includes hemorrhagic)
- Cardiogenic Shock

Regardless of etiology, shock is associated with a state of hypoperfusion, organ dysfunction, and a high risk of death

SAQ # 5

- 92yo female with urosepsis and septic shock
- PMH: MI, HTN, CHF
- BP 72/44, HR 120, Cr 2.7, UO 20 ml/hr
- Empiric antibiotics initiated
- What's next?
  - A. Dopamine 1 mcg/kg/min
  - B. Furosemide 60mg IV now
  - C. NS 500ml bolus
  - D. Albumin 5% 500ml bolus

What is Sepsis?

- Systemic inflammatory reaction triggered by an infection
- Release of cytokines and inflammatory mediators
- Systemic vasodilation causes hypotension
- Increased vascular permeability causes fluids to shift out of intravascular space
- Intravascular volume depletion leads to reduced cardiac function which perpetuates hypotension and organ hypoperfusion.
Sepsis Definitions

- Sepsis – Systemic inflammatory response triggered by a documented or suspected infection
- Severe Sepsis – Sepsis complicated by organ dysfunction or hypoperfusion
- Septic Shock – Sepsis-induced hypoperfusion that responds poorly to fluid resuscitation and vasopressors

Hypoperfusion & Organ Dysfunction

<table>
<thead>
<tr>
<th>Organ System</th>
<th>Some (not all) Signs of Dysfunction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>SBP &lt; 90 or MAP &lt; 70 Unresponsive to fluid resuscitation</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Need for mechanical ventilation</td>
</tr>
<tr>
<td>Kidney</td>
<td>↓ UO or ↑ Creatinine</td>
</tr>
<tr>
<td>Hematologic Coagulation</td>
<td>↓ Platelet, ↓ WBC, or ↑ INR</td>
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</tbody>
</table>

Initial Resuscitation for Poor Perfusion

- Complete initial resuscitation in 1st 6 hours
- Goals are to optimize intravascular volume (and thus CO) and organ perfusion
  - CVP 8-12 mmHg (12-15 if intubated)
  - MAP ≥ 65 mmHg
  - Urine output ≥ 0.5 ml/kg/hr
  - SvO2 ≥ 70%
- This "early goal-directed resuscitation" improves survival

Initial Resuscitation Protocol

- Fluid resuscitation using crystalloid or colloid
  - 1000ml NS or LR administered as fast as possible and repeat as needed to achieve goals
  - 300-500 ml colloid (e.g., albumin, hetastarch) administered over 30 minutes
  - No evidence that one strategy superior to another
- Continue fluid resuscitation as long as hemodynamic parameters are improving (MAP, CVP, SvO2)
- Patients may require aggressive fluid resuscitation during 1st 24 hours

Fluids → Vasopressors

- If unable to achieve goals with fluids, then...
  - PRBC if HCT < 30%
  - Dobutamine to improve cardiac output
  - Vasopressors
- Fluid resuscitation should be achieved 1st
  - Vasopressors can be ineffective or detrimental if administered prior to fluid resuscitation
  - However, vasopressors may be needed when hypovolemia has not been resolved in cases of life-threatening hypoperfusion

Vasopressors

- Norepinephrine and dopamine are initial vasopressors of choice to maintain MAP ≥ 65
- Grade 1C
  - Strong recommendation, but low quality evidence
- No high-quality evidence (Grade A) to recommend one vasopressor over another
  - Clinical practice has been driven by expert opinion
  - However, a recent study found no mortality difference comparing NE to DA, but ↑ arrhythmias with DA
Norepinephrine (Levophed)

**Efficacy**
- $\alpha_1$-induced vasoconstriction maintains perfusion

**Safety**
- $\downarrow$ CO
- $\downarrow$ Renal perfusion
- Peripheral ischemia
- $\beta_1$-induced arrhythmias or myocardial ischemia
- $\alpha_1$ vasoconstriction at higher doses

Page 201, Table 6

Dopamine

**Efficacy**
- Low-dose $\beta_1$-induced ionotropy could complement vasoconstrictive effect of norepinephrine
- $\alpha_1$ vasoconstriction at higher doses

**Safety**
- $\beta_1$-induced arrhythmias or myocardial ischemia
- Endocrine changes ($\downarrow$ Prolactin, GH, TH)
- Inappropriate use - unfounded belief of renal protection

Page 201, Table 6

Epinephrine

**Efficacy**
- $\alpha_1$-induced vasoconstriction maintains perfusion

**Safety**
- $\beta_1$-induced arrhythmias or myocardial ischemia
- Reduced splanchnic circulation $\rightarrow$ gut ischemia?
- $\uparrow$ lactate-hypoperfusion

Page 201, Table 6

Phenylephrine

**Efficacy**
- $\alpha_1$-induced vasoconstriction maintains perfusion
- No $\beta_1$ stimulation
- Reflex slowing of HR

**Safety**
- $\downarrow$ CO
- $\downarrow$ Renal perfusion

Page 202, Table 6

Vasopressin

**Efficacy**
- V1-receptor stimulation causes profound vasoconstriction
- Relative VP deficiency in SS
- Effective during acidosis and hypoxia

**Safety**
- Vasoconstriction leads to coronary and peripheral ischemia at doses $>0.04-0.06$ unit/min
- Not titrated like traditional vasopressors
- Dosing errors with unit/min

Page 202, Table 6

Dobutamine is an Ionodilator

**Ionotrope ($\beta_1$)** $\rightarrow$ Tachyarrhythmia

**Vasodilator ($\beta_2$)** $\rightarrow$ Vasodilation

Patients with septic shock can have a relative myocardial dysfunction (SvO2 $<70\%$)

Dobutamine can improve CO and thus improve BP and organ perfusion, but monitor for arrhythmia’s and hypotension

Page 202, Table 6
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  d. Albumin 5% 500ml bolus

Agenda

- **Sepsis-Related**
  - Hemodynamics
  - Initial Resuscitation
  - Sepsis Definitions
  - Vasopressors
  - Antimicrobials
  - Corticosteroids

- **Other Critical Care Issues**
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  - Delirium
  - Hyperglycemia
  - Stress Ulcer
  - DVT Prophylaxis
  - VAP Prevention
  - Nutrition

Antimicrobials in Sepsis
- Most common sources of infection in patients with severe sepsis or septic shock are lung, abdomen, and urinary tract
  - Assess sputum / tracheal secretions, urine, and stool cultures as appropriate depending on the suspected source
- Early antimicrobial, but earlier cultures!
- Obtain at least 2 blood cultures before antimicrobial therapy
  - 1 drawn percutaneously

Initial Empiric Antibiotic Therapy
- Early and appropriate empiric antibiotic therapy can be life-saving
- Start Antibiotics within 1st hour of recognition of sepsis
- If multiple antibiotics, give broadest coverage first and administer as quickly as possible
- De-escalation therapy based on cultures and clinical situation
- Typical duration is 7-10 days

Consider Empiric Fungal Therapy if Risk Factors Present
- Recent abdominal surgery
- Chronic PN
- Indwelling central venous catheter
- Immunocompromised
  - Chronic corticosteroids or other immunosuppressants
  - Neutropenia
  - Malignancy
  - Organ transplant

Corticosteroids:
**Efficacy in Septic Shock**
- Hydrocortisone 50mg IV q6h can improve short-term mortality in patients with hypotension that is poorly responsive to fluid resuscitation and vasopressors... "septic shock" (Annane JAMA 2002:862)
- Use of hydrocortisone in this scenario is a Grade 2C recommendation
  - Weak recommendation based on low quality evidence (the study was significantly underpowered)

Page 203 b i (a-b)

Page 203 b ii-vi

Page 193
Corticosteroids: Not Always Beneficial

- CORTICUS trial evaluated same regimen of hydrocortisone in patients with sepsis requiring vasopressors, but not necessarily with persistent hypotension.
- No mortality benefit
- Increased risk of hyperglycemia
- Increased risk of new sepsis or septic shock

**Corticosteroids**

- Fludrocortisone 50ug daily is optional
  - Unnecessary in theory because hydrocortisone has mineralocorticoid activity
  - Recently substantiated lack of benefit with fludrocortisone in COIITSS study
- No need to assess adrenal function with ACTH, or other measures (e.g., cortisol)
- Once vasopressors no longer required, taper, then d/c corticosteroids

Patient Case #4

- 65yo female in CCU for MI
- New on day 4: BP 80/50, HR 125, RR 30, hypoxic, temp 102, confused, CXR infiltrates
- Given NS 1L x 2, intubated, started on pip/tazo + ciproflox for nosocomial pneumonia
- Further fluid bolus ineffective

**Patient Case #4: Which is best?**

A. Add clinda for aspiration pneumonia
B. Add dobutamine titrated for MAP > 65
C. Add norepinephrine titrated for MAP > 65
D. Add hydrocortisone 50mg q6h IV

Patient Case #6

- 42yo Man unresponsive with BP 72/30
- Covered in vomit; intubated; r/o aspiration
- WBC 20, LA 15, AST 78, Cr 2 (1), Ptt 118, INR 1.4, UO 15 ml/hr
- Receiving NS (~ 5 L) + Albumin 5%
- BP 87/56 (MAP 66) on NE 40 mcg/min
- Started on Zosyn
- What next?

**Patient Case #6**

A. Hydrocortisone 50mg q6h IV
B. Check random cortisol
C. Add low-dose dopamine
D. Add enoxaparin 40mg subQ daily
Sepsis Resuscitation and Management
Bundle Includes:
- Measure serum lactate (a sign of hypoperfusion)
- Obtain blood cultures before antibiotics
- Timely broad-spectrum antibiotics
- Early goal-directed therapy
- Consider corticosteroids
- Consider rhAPC (withdrawn from market)
- Glucose control

Agenda

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Patient Case # 7
- 51yo woman collapses; family call 911 / CPR
- Unresponsive, HR 20
- In ER: Map 68 after fluids + NE
- Hypothermia protocol initiated x 24 hours – starting re-warming process when you arrive to ICU
- Insulin continuous infusion 15 units/hr, √ q6h
- Propofol + Nimbex (cisatracurium)

Patient Case # 7: What’s next?
A. Increase BG testing to q1-2 hours
B. Adjust cisatracurium for TOF 0/4
C. D/C propofol to facilitate extubation
D. Increase insulin to prevent hyperkalemia

Inducing Hypothermia post Cardiac Arrest
- Inducing hypothermia (32-34°C) for 12-24 hrs post cardiac arrest can improve neurologic recovery and mortality
- Consider if successfully resuscitated but remain comatose
- Achieved using ice packs, cooling blankets, and infusion of cold IV fluid
- Patients will need sedation and analgesia during period of hypothermia

Complications of Hypothermia
- Shivering warms body temperature
  - Prevent with sedatives, anesthetics, opiates, Magnesium, buspirone, or paralytics
  - Shivering more common during cooling
- Reduced drug clearance
  - Can include sedatives, opiates, paralytics
  - Use normal bolus doses, but reduce maintenance doses
- Intravascular volume depletion
  - Replace with crystalloids
More Complications of Hypothermia
- Electrolyte imbalances – be proactive
  - ↓ K, Mg, PO4 during cooling
  - ↑ K during rewarming
- Increased risk of infection
- Blood Glucose
  - Hyperglycemia during hypothermia
  - Hypoglycemia during rewarming – be proactive
- Bedsores
- Impaired bowel function
  - Hold nutrition during hypothermia

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Consider more about Patient Case #7
- Do comatose patients who are paralyzed need to be sedated?
- What if they become hypotensive with propofol? Can the sedation be stopped?

Patient Case #8: Sedation
- Elderly woman admitted for ADHF and AKI
- Intubated for pulmonary edema
- RASS +2 (agitated); CAM-ICU +, denies pain
- BP 120/70, HR 88
- Which is best for sedation?
  A. Propofol at 5 mcg/kg/min and titrate
  B. Haldol 5mg IV, double every 20 min prn
  C. Lorazepam 1mg q 20 min prn
  D. Morphine 4mg IV q4h prn pain

Analgesia & Sedation Assessment
- Consider non-pharmacologic strategies (window, lighting, music, patient positioning)
  - Not all patients will require drug therapy
- Determine goals using a validated scale
- Not all patients will require drug therapy
- Assess pain using a tool that is valid and reliable for adult ICU patients
  - Behavioral Pain Scale
  - Critical-Care Pain Observation Tool
- Vital signs are cues (to use a tool above)
Assess sedation using a tool that is valid and reliable for adult ICU patients
- Richmond Agitation-Sedation Scale (RASS)
- Sedation-Agitation Scale (SAS)

Treat pain first and add a sedative if needed
- Opioids have analgesic and sedative properties
- Use analgesics before potentially painful procedures
- Opioids are first DOC for pain
- Non-opioid + opioid analgesics to improve efficacy and safety
  - APAP, ibuprofen, ketorolac, ketamine
- Non-benzo sedatives preferred

Goal: Use validated scales to achieve patient goal without over-sedation
- Intermittent Dosing - routinely or PRN
- Continuous Infusion
  - If using continuous infusion, use a bolus dose before or instead of rate ↑ for opioids and benzodiazepines (if BP tolerates)
  - Use bolus dosing proactively (dressing changes)
- Hybrid Approach
  - Intermittent drug 1 + continuous infusion drug 2

Protocols should incorporate
- Regular reduction in infusion rates
- Daily interruption and subsequent re-assessment of dose
  - This can decrease duration of mechanical ventilation and ICU LOS
- Laxatives + stool softener should be used proactively in patients receiving opioids

Infusion rates get increased, but not decreased (unless during ventilator weaning)
- Protocols need to specify times for rate reductions
- Bolus dosing is under-used during continuous infusions of opioids and benzodiazepines
  - Bolus dosing has faster onset than increasing infusion rate, and can eliminate need for a rate ↑
  - Bolus dosing is not necessary with very short acting sedatives (propofol, dexmedetomidine) and can cause hypotension or bradycardia

Opioid Analgesics

- Morphine
  - Histamine release
  - Hypotension
  - Itching
  - Rash
  - Active metabolites (renally eliminated)
- Hydromorphone
  - Dosing errors (≥ 2mg)
  - No active metabolites
- Fentanyl
  - No histamine release
  - Useful if ↓BP
  - Short acting with single doses, not CI
  - ↑ duration if obese
- Remifentanil
  - Short acting
  - Expensive
**Benzodiazepines**

- **Lorazepam**
  - Predictable if kidney/liver disease
  - Propylene glycol toxicity at high infusion rates

- **Midazolam**
  - Drug metabolism changes during critical illness
  - Drug interactions
  - Active metabolites

---

**Propofol**

- Most commonly used sedative
- Very rapid onset, short duration (minutes)
- Hypotension, especially with bolus doses
- Respiratory depression
- No significant analgesic effect
- 10% lipid emulsion
  - Consider calories (1.1 kcal/ml)
  - Monitor TG
- Propofol-infusion syndrome more likely if prolonged infusion > 50 mcg/kg/min

---

**Propofol Infusion Syndrome S/S**

- Metabolic acidosis
- Cardiac failure
- Arrhythmias
- Cardiac arrest
- Rhabdomyolysis
- Hyperkalemia
- Kidney failure
- With prolonged high doses of propofol, I recommend a 2nd agent to titrate up while titrating propofol down

---

**Benzodiazepines vs. Propofol**

- Longer duration compared to propofol
- Not as titratable
- Not as predictable
- Some patients will require very large doses of benzodiazepines to achieve sedation goals
- Benzodiazepines are associated with increased risk of delirium

---

**Dexmedetomidine (Precedex®)**

- Sedative and analgesic activity mediated through central and peripheral α₂ receptor stimulation
- Extent of analgesic activity not well described…surgical patients typically need additional pain medications
- No respiratory depression
- Titratable – rapid onset, short duration

---

**Dexmedetomidine**

- Bradycardia and hypotension – especially with bolus dosing (avoid if possible)
- FDA approved for max 24 hour infusion, but evidence of safety/efficacy is accumulating for longer infusions
- Associated with reduced incidence of delirium when compared to benzodiazepines
- Need evidence comparing to propofol
Patient Case #8: Sedation

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- RASS +2 (agitated); CAM-ICU +, denies pain
- BP 120/70, HR 88

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C. Lorazepam 1mg q 20 min prn
D. Morphine 4mg IV q4h prn pain

Delirium

- Acute change in cognitive function characterized by disorganized thought, altered level of consciousness, and inattentiveness.
- Associated with increased mortality and LOS
- Use validated tool to identify and assess
  - CAM-ICU
  - Intensive Care Delirium Screening Checklist

Delirium Treatment

- Nonpharmacologic – verbal reassurance, uninterrupted sleep, natural lighting, early mobility
- Avoid benzodiazepines, anticholinergics
- Haldol 1-10mg, double every 20 min
  - Once patient calm, add total mg administered and divide into 4 daily doses given q6h
  - Monitor BP, QT baseline and daily, drug interactions, EPS (laryngeal dystonia and dysphagia), seizures
- Atypical antipsychotics are alternatives

Preventing Hyperglycemia: Timeline

- 2001: 80-110 reduced mortality; single ctr; surgery pts; Hypoglycemia 5.1 vs 0.8%
- 2002-2008: ICU’s adopt intensive insulin
- 2002-2008: Unable to reproduce ’01 results
- 2006: 80-110 MICU; Hypoglycemia 18 vs 3%
- 2007: CCM recommends < 150 mg/dL
- 2009: NICE SUGAR

2009: NICE-SUGAR

- > 6,000 medical and surgical ICU patients
- Compared 80-108mg/dL vs. ≤ 180 mg/dL
- ↑ Mortality
  - 27.5% vs. 24.9%, (OR 1.14; 95% CI 1.02-1.26; p=0.02)
- ↑ Hypoglycemia (BG ≤ 40mg/dL)
  - 6.8% vs. 0.5% (p< 0.001)

Preventing Hyperglycemia: Moving Forward

- Evidence suggests a target range of < 180
  - Many suggest 140-180, but exact range unknown
- Regardless of the range, glucose control is important in all critically ill patients…with and without diabetes
- Caution that point-of-care testing of capillary blood (i.e., “fingerstick”) can overestimate plasma glucose values

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- Many suggest 140-180, but exact range unknown
- Regardless of the range, glucose control is important in all critically ill patients…with and without diabetes
- Caution that point-of-care testing of capillary blood (i.e., “fingerstick”) can overestimate plasma glucose values
Preventing Hyperglycemia:
Moving Forward

- Avoid sole therapy with an insulin sliding scale
  - Consider a baseline of insulin (e.g., Lantus, NPH) and adjust baseline daily based on sliding scale requirements
- Patients may require IV insulin due to variable SC absorption
  - Insulin protocol adjustments should take into consideration: BG value, rate of change, and insulin infusion rate

Patient Case #12: SUP

- 73yo female after cardiac arrest; intubated
- Vitals stable, OGT with TF’s
- Meds – amiodarone, simvastatin, clopidogrel, asa, metoprolol, UFH, NS
- Which is best?
  a. Famotidine 20mg per OGT q12h
  b. Esomeprazole 40mg per OGT daily
  c. Sucralfate 1g per OGT qid
  d. Ranitidine 50mg IV q8h

Stress Ulcer

Two major risk factors:

1. Respiratory failure requiring mechanical ventilation
2. Coagulopathy
   - Platelet < 50K
   - INR > 1.5
   - aPTT > 2 x’s control
   - Note prophylactic or treatment doses of anticoagulants does NOT = coagulopathy

Stress Ulcer Prophylaxis

- H₂-blockers (more evidence) or proton pump inhibitors (less evidence)
- Evidence demonstrating efficacy used IV administration, however enteral route is commonly used for drugs
- We typically remember to start SUP, but frequently forget to stop SUP
- Increased risk of C. diff, HAP, and CAP (if discharged on PPI) with increasing gastric pH

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- Vitals stable, OGT with TF’s
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  c. Sucralfate 1g per OGT qid
  d. Ranitidine 50mg IV q8h
Patient Case #13: SUP

- Patient from case #12 is extubated
- Vitals stable
- Poor appetite
- Which is correct?
  1. Continue SUP till ICU discharge
  2. D/C SUP now
  3. Continue SUP until eating
  4. D/C SUP when discharged from hospital

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Preventing VTE

- LMWH is superior to UFH in trauma / SCI
- LMWH and UFH equivalent in medical pts
  - Heparin SQ q8 vs. q12 should be based on patient’s risk of thrombosis vs. bleeding
  - LMWH is suggested in very high risk medical patients (Grade 2C) not because evidence supports this, but because LMWH is superior to UFH in other high risk patients (e.g., major trauma, spinal cord injury)
- What about patients with kidney dysfunction?
- What about obese patients?

Page 220, Table 18

VTE Prevention in CKD

- For CrCl 20-30 ml/min:
  - Enoxaparin dose should be reduced to 30mg SC daily (rather than the normal dose of 40mg daily)
  - No apparent adjustment for dalteparin or tinzaparin for short-term use (< 10d)
- For CrCl < 20 ml/min or patients on HD:
  - Dosing information is limited for LMWH for prophylaxis (and treatment)...consider UFH
- Anti Xa monitoring may not be reliable in dialysis patients

Page 219 VIII C1

VTE Prevention (and treatment) in Obese Patients

- Consider increasing LMWH prophylaxis doses by 30% in patients with BMI > 40 kg/m2
  - No consensus, but suggest peak (4 hrs post dose) anti-Xa 0.2-0.4 IU/ml for prophylaxis
- For treatment of VTE in obese patients, use total body weight for LMWH (not in text)
  - Avoid once-daily dosing of enoxaparin in obesity
  - Anti-Xa monitoring is not needed if < 190kg
  - Peak anti-Xa (0.5-1 IU/ml) is suggested if > 190kg

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Preventing VAP

- Analgesia/Sedation
  - Daily interruption
  - Use validated score to assess sedation goals
    - Regularly try to reduce doses to prevent over-sedation
  - Daily assess readiness to wean from vent
- HOB 30-45° if administering tube feedings
- Daily oral care (chlorhexidine, povidone iodine)
- Avoid gastric over distention
  - Assess tolerance to tube feedings
  - Prevent opioid-induced constipation

Page 220 A-D

Nutrition Support

- Enteral nutrition improves outcomes
  - Reduced post-operative infections
  - Improved wound healing
- Parenteral nutrition…?
  - When supplement with EN, late initiation (after 1 week) of PN in critically ill patients had reduction in LOS, infection, cholestasis, and costs compared to early initiation (within 48 hours)...benefits even evident in those with contraindication to EN (so no nutrition for a week)

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Enteral Nutrition: Preventing Aspiration

- Monitor gastric residuals and hold TF if > 250-500ml
- HOB 30-45 degrees
- Feeding post-pyloric valve (controversial)
- Metoclopramide & erythromycin can ↑ GI motility and ↓ gastric residuals, and can improve tolerance of EN, but ↔ in preventing aspiration pneumonia

**Enteral Nutrition: Caloric Goals**

- A recent study showed that “trickle” or “trophic” feeds (10-30 ml/hr), which provided about 16% of caloric goals, had similar outcomes (time on vent, ICU stay) but reduced gastric residual volume.
- Alternatively, caloric deficits have been associated with longer time on vent, pressure ulcers, increased LOS and mortality

**Predicting kcal in Critically Ill**

- Indirect Calorimetry is the best for critically ill patients, but usually unavailable ($$$)
- See “Predictive Equations for Energy Needs for the Critically Ill” ref below
  - Harris-Benedict equation is not recommended
  - ACCP recommendation of 25 kcal/kg also not recommended
- Underfeeding is associated with prolonged ventilator dependence and LOS

**Predictive Equations in Critically Ill**

- Penn State 2003 equation is one of most accurate predictive equations for intubated critically ill patients (*equation not in chapter*)

\[(0.85 \times \text{value from Harris-Benedict Eqn}) + (175 \times T_{\text{max}}) + (33 \times V_E) - 6433\]

\[V_E = \text{minute volume (in L/min)}\]

**Agenda**

- Sepsis-Related Hemodynamics √
- Initial Resuscitation √
- Sepsis Definitions √
- Vasopressors √
- Antimicrobials √
- Corticosteroids √
- rhAPC √

- Other Critical Care Issues Hypothermia/Cardiac Arrest √
- Analgesia & Sedation √
- Delirium √
- Hyperglycemia √
- Stress Ulcer √
- DVT Prophylaxis √
- VAP Prevention √
- Nutrition √

**Thank You**

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