Fluid, Electrolytes, Hypovolemia & Resuscitation

- **Physiology**
  - Total body water constant
  - Total body water fat and age dependent
    - Male 60%, Female 50%
    - Infants 80%, 1 year old 65%
  - TBW = ICF (2/3) + ECF (1/3)
  - ECF = Intravascular (25%) + Interstitial (75%)
  - Water in intracellular, intravascular, interstitial compartments in “Dynamic Equilibrium” because of semipermeable membranes and osmotic gradients
  - Sodium and Potassium – dominant cations
  - Sodium - extracellular
  - Potassium – intracellular
  - Colloid oncotic pressure (plasma protein intravascular)
  - Osmoregulation – hypothalamus
    - ADH/thirst/renal/angiotensin
  - Volume control
    - Osmoreceptors – “day to day”
    - Baroreceptors – 10-20% change in volume
    - Atria – natriuretic peptide
  - Baroreceptor modulation of volume control
    - Receptors in aorta, carotid, renal
    - Sympathetic/parasympathetic trigger hormonal changes
    - Renin angiotensin, aldosterone, atrial natriuretic peptide, renal prostaglandins

- **Surgical Patients**
  - Prone to fluid and electrolyte abnormalities because of disease and surgical care
  - “Effective” circulating volume
    - Perfuse organs
    - Third space loss ineffective
    - Not helpful with CHF or AVF
  - Normal water exchange
    - Sensible – urine, stool, vomit, sweat
    - Insensible – evaporation of skin and respiratory tract
    - GI tract – net secretory to Jejunum, reabsorption in small bowel and colon
• Fluid and Electrolyte Therapy
  o Parenteral solutions
    ▪ Lactated ringers – similar to plasma, edema, SB losses. Sodium 130
  o Isotonic saline (.9%, sodium 154)
    ▪ Possible sodium overload and hyperchloremic acidosis
  o Hypotonic (D51/4 NS, D5 ½ NS)
  o Hypertonic (3% Na, “hot salt”)
  o Plasma expanders – problems with microvascular permeability
  o **Goal is normalization of hemodynamic parameters and electrolyte concentrations while avoiding complications from too rapid correction or overcorrection**

• Volume deficit (hypovolemia)
  o Chronic – decrease skin turgor, dry mucous membranes, sunken eyes, orthostasis, tachycardia, hypothermia, BUN/Cr >15:1, HCT increased, Na decreased excretion
  o Acute – change in vital signs without tissue changes; decreased urine output
  o **Response to Hypovolemia:**

<table>
<thead>
<tr>
<th>Blood Volume</th>
<th>Supine</th>
<th>Sitting</th>
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<tbody>
<tr>
<td></td>
<td>BP</td>
<td>HR</td>
</tr>
<tr>
<td>500 (5%)</td>
<td>NML</td>
<td>NML</td>
</tr>
<tr>
<td>1000 (10-15%)</td>
<td>NML</td>
<td>NML or ↑</td>
</tr>
<tr>
<td>1500 (20%)</td>
<td>NML or ↓</td>
<td>↑</td>
</tr>
<tr>
<td>2000 (30%)</td>
<td>↓</td>
<td>↑ or ↓</td>
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</tbody>
</table>

• Resuscitation
  o Isotonic solution – lactated ringers
  o Blood and blood products
  o Colloid
  o Goal – normalization of BP, HR, Urine output
  o Monitor – Foley, CVP, Base deficit, lactate, Swan Ganz catheter
  o **Volume excess** – over resuscitation, mobilization 3rd space fluids, post op or trauma patients with increased ADH

• Maintenance fluid
  o 70 kg  2500cc water, 140 meq Na, 70 meq K
- Includes sensible and insensible losses
- Does not factor deficits or on-going losses
- Younger or smaller patients require more cc/kg because high percentage TBW in relation to body weight
- Ca, PO4, Mg, trace elements later

- Electrolyte concentration in GI secretions

<table>
<thead>
<tr>
<th>Meq/L</th>
<th>NA</th>
<th>K</th>
<th>Cl</th>
<th>HCO3</th>
<th>H</th>
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<tbody>
<tr>
<td>Salivary</td>
<td>50</td>
<td>20</td>
<td>40</td>
<td>30</td>
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</tr>
<tr>
<td>Gastric Basal</td>
<td>100</td>
<td>10</td>
<td>140</td>
<td>–</td>
<td>30</td>
</tr>
<tr>
<td>Gastric Stim</td>
<td>30</td>
<td>10</td>
<td>140</td>
<td>–</td>
<td>100</td>
</tr>
<tr>
<td>Bile</td>
<td>140</td>
<td>5</td>
<td>100</td>
<td>60</td>
<td>–</td>
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<tr>
<td>Pancreas</td>
<td>140</td>
<td>5</td>
<td>75</td>
<td>100</td>
<td>–</td>
</tr>
<tr>
<td>Duo</td>
<td>140</td>
<td>5</td>
<td>80</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Ileum</td>
<td>140</td>
<td>5</td>
<td>70</td>
<td>50</td>
<td>–</td>
</tr>
<tr>
<td>Colon</td>
<td>60</td>
<td>70</td>
<td>15</td>
<td>30</td>
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</tr>
</tbody>
</table>

- Replacement ongoing fluid losses
  - Intraoperative losses
    - Anesthesia disrupts baroreceptor reflexes
    - No increase HR or increase PVR
    - Most tolerate <500 cc blood loss
    - LR 500-1000 cc/hour common
  - Post op fluid losses
    - NG, Ileostomy, fistula, edema
    - Ileus
    - 3rd space
  - Post op monitoring
    - VS, CVP, I/O, daily wts, Swan Ganz Catheter
    - Electrolytes, lactate, base Deficit
    - Urine specific gravity (1.012)
    - Urine osmolality
• **Hyponatremia**
  - Excess free water most common cause
  - Self-limiting; rarely <130 meq/L if no SIADH
  - Hyperosmolar hyponatremia 2\(^\circ\) hyperglycemia
  - Symptoms if <120 meq/L
    - Weakness
    - Fatigue
    - Muscle cramps
  - Treatment
    - Free water restriction
    - “Hot salt” if symptomatic

• **Hypernatremia**
  - Uncommon in surgical patients
  - 2\(^\circ\) free water loss
  - Associated with head trauma or post-surgical with decreased ADH
  - CNS symptoms if >160 meq/L
  - Treat slowly to avoid cerebral edema and herniation

• **Potassium**
  - Major intracellular cation (2% in ECF)
  - Concentration differences between ICF and ECF creates transmembrane potential
  - Profound effects on cardiac, skeletal, and smooth muscle
  - Total body stores dependent on muscle mass

• **Hypokalemia**
  - Raises membrane excitation potentials
  - Nerve and muscle less excitable
  - **NOTE** – Life threatening SVT in patients on Cardiac Glycosides (Digoxin)
  - Symptoms - < 2.5 meq/L
    - Muscle weakness
    - Ileus
    - **Arrhythmias**
  - Treatment
    - Oral or IV
    - No more than 40 meq/hr.
    - Monitor serum K
    - Arrhythmia meds
• Hyperkalemia
  o Decrease membrane excitation potentials
  o Nerve and muscle cells more excitable
  o Acidosis leads to K shift out of cells raising serum K
  o Alkalosis leads to K shift into cells lowering serum K
  o Insulin promotes K entry into muscle/hepatic cells lowering serum K
  o Rare in absence of renal disease
  o Excessive cellular breakdown secondary to trauma (crush injuries), vascular insufficiency, sepsis
  o Symptoms
    ▪ Arrhythmias, V-Fib
    ▪ Weakness
    ▪ Paralysis
  o Treatment
    ▪ Calcium Gluconate
    ▪ Sodium Bicarbonate
    ▪ Glucose/Insulin
    ▪ Kayexalate
    ▪ Dialysis

• Calcium
  o Homeostasis between bone, ECF, renal excretion, intestinal absorption
  o Role of PTH (Parathyroid)
  o Ionized (45%), Non-ionized, Protein bound
  o pH
    ▪ Acidosis
      • ↓ Protein bound
      • ↑ Ionized
    ▪ Alkalosis
      • ↑ Protein bound
      • ↓ Ionized
  o Albumin
    ▪ Decrease
      • ↓ Protein bound
      • ↑ Ionized
    ▪ Increase
      • ↑ Protein bound
      • ↓ Ionized
  o Key role in neuromuscular transmission, muscle contraction, enzyme regulation
• Hypercalcemia
  o Hyperparathyroidism
  o Vit D intoxication
  o Skeletal mets
  o Tumors secreting PTH “like” peptides
  o Symptoms
    ▪ Muscle fatigue
    ▪ Weakness
    ▪ Mental changes
    ▪ Pancreatitis
    ▪ Kidney stones
  o Treatment
    ▪ Surgery
    ▪ Treat etiology
    ▪ Meds if acute (>14 mg/dl)

• Hypocalcemia
  o Rare
  o S/P Thyroidectomy or Parathyroidectomy
  o Hyperventilation Syndrome causing acute alkalosis
  o Pancreatic, small bowel fistula
  o Symptoms - <8 mg/dl
    ▪ Perioral paresthesia
    ▪ Stridor
    ▪ Tetany
    ▪ Seizures
  o Treatment
    ▪ Asymptomatic
      • None
    ▪ Symptomatic
      • IV and/or PO Calcium

• Magnesium
  o Role in Neuromuscular function
  o Bone and Intracellular
  o < 1% in ECF

• Hypermagnesemia
  o Rare
- Renal failure #1 Etiology
  - Burns, crush injuries
  - Symptoms
    - Decreased DTR, Paralysis
  - Treatment
    - Calcium Gluconate
    - Ventilator
    - Pacing
    - Dialysis

- Hypomagnesaemia
  - Causes
    - Low intake
    - GI losses
    - Burns
    - Pancreatitis
    - Alcoholics
  - Symptoms
    - Neurologic
    - Neuromuscular
    - Arrhythmias
    - Contributes to hypokalemia and hypocalcemia
  - Treatment
    - If mild – Mag-oxide PO
    - If severe – Magnesium Sulfate IV
• **Pearls and Tricks for Hypotensive Patients**

- If hypotension secondary to bleeding in trauma – always stop visible or accessible bleeding **BEFORE** IV access
  - Direct pressure
  - Tourniquet
  - BP Cuff
  - Combat quaze
  - Balloon catheters
  - Mast trousers
- Understand permissive hypotension and why
- Understand transfusion protocols and coagulopathy
  - 1:1:1 transfusion
  - Hypothermia
  - Acidosis
  - Damage control surgery
- Both trauma and sepsis resuscitation can require **MASSIVE** fluid requirements secondary to ongoing losses and 3rd spacing. **EXPECT** Severe Edema.
- Days later after patient is stabilized watch for signs of fluid overload and CHF as a result of the 3rd spaced fluid becoming mobilized back into intravascular space
- **Emergency Vascular Access in Children**
  - 2 short large diameter lines
  - Hand, Arm, Saphenous
  - Intraosseous if < 6 yr.
    - Gravity flow and remove in 6 hours
  - Cutdown on saphenous or basilic vein
  - Central line
    - High complication rate
    - Femoral route preferred
- Most rapid resuscitation accomplished with saphenous cutdown and sterile IV tubing.