Fluid and Electrolyte Management of the Surgical Patient

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The key to understanding fluids and electrolytes stems from a complete understanding of basal metabolism.

- **SITE OF FLUID LOSS**
  - **Skin, Resp. Tract**: 50% Fluid, None Electrolytes
  - **Urine**: 50% Fluid, All Electrolytes

**Normal Water Exchange: Sensible and Insensible Losses**

<table>
<thead>
<tr>
<th>Sensible Losses</th>
<th>Avg. Daily Volume (ml.)</th>
<th>minimal amount of water needed/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary</td>
<td>800-1500 ml</td>
<td>300 ml</td>
</tr>
<tr>
<td>Intestinal</td>
<td>0-250 ml</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Insensible Losses</th>
<th>Avg. Daily Volume (ml.)</th>
<th>minimal amount of water needed/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lungs and skin</td>
<td>600-900 ml</td>
<td>600-900 ml</td>
</tr>
</tbody>
</table>
PHYSIOLOGY of a 70kg Man: 42L = 60%TBW

Extracellular Fluid
- Plasma: 28 Liters
- Interstitial Fluid: 10 Liters
- Total extracellular fluid = 38 Liters

Intracellular Fluid
- 28 Liters
- 40% TBW

Total osmolarity across membranes = 280mOsm/L

**ISOTONIC SOLUTION = 0.9 NS**

- Osmolarity = 290 mOsm/L

**HYPOTONIC SOLUTION = D5W**

- Osmolarity < 280 mOsm/L

**HYPERTONIC SOLUTION = 3% NS**

- Osmolarity > 290 mOsm/L

<table>
<thead>
<tr>
<th>Substance</th>
<th>Plasma</th>
<th>Interstitial</th>
<th>Intracellular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>142</td>
<td>139</td>
<td>14</td>
</tr>
<tr>
<td>K</td>
<td>4.1</td>
<td>4</td>
<td>100</td>
</tr>
<tr>
<td>Ca</td>
<td>1.3</td>
<td>1.2</td>
<td>0</td>
</tr>
<tr>
<td>Mg</td>
<td>0.8</td>
<td>0.7</td>
<td>20</td>
</tr>
<tr>
<td>Cl</td>
<td>108</td>
<td>108</td>
<td>4</td>
</tr>
<tr>
<td>HCO3</td>
<td>24.1</td>
<td>28.3</td>
<td>10</td>
</tr>
<tr>
<td>HPO4, H2PO4</td>
<td>2</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>Phosphocreatine</td>
<td>0</td>
<td>0</td>
<td>45</td>
</tr>
<tr>
<td>Creatine</td>
<td>0.2</td>
<td>0.2</td>
<td>9</td>
</tr>
<tr>
<td>Lactate</td>
<td>1.2</td>
<td>1.2</td>
<td>1.5</td>
</tr>
<tr>
<td>ATP</td>
<td>0</td>
<td>0</td>
<td>0.5</td>
</tr>
<tr>
<td>Hexose Monophosphate</td>
<td>0</td>
<td>0</td>
<td>2.7</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.6</td>
<td>5.6</td>
<td>0</td>
</tr>
<tr>
<td>Protein</td>
<td>1.2</td>
<td>0.2</td>
<td>0</td>
</tr>
<tr>
<td>Urea</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Others</td>
<td>4.9</td>
<td>3.9</td>
<td>10</td>
</tr>
<tr>
<td><strong>TOTAL mOsm/L</strong></td>
<td>282</td>
<td>281</td>
<td>281</td>
</tr>
</tbody>
</table>
Starling’s Equilibrium for Capillary Exchange

\[ Q_f = K_f \left( (P_{cap} - P_{int}) - \sigma (\pi_{cap} - \pi_{int}) \right) \]

Fluid Flux
Perm. of water
Hydrostatic Press
Perm. To protein
Colloid osmotic Press

Net Outward Force
Net Inward Force

(17.3 - (-3.0))
(28 - 8.0)

Composition of Parenteral Fluids:
Electrolyte Content (mEq/L)

<table>
<thead>
<tr>
<th>SOLUTION</th>
<th>CATIONS</th>
<th>ANIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na</td>
<td>K</td>
</tr>
<tr>
<td>Extracellular Fluid</td>
<td>142</td>
<td>4</td>
</tr>
<tr>
<td>Lactated Ringers</td>
<td>130</td>
<td>4</td>
</tr>
<tr>
<td>0.9% Normal Saline</td>
<td>154</td>
<td></td>
</tr>
<tr>
<td>D₅₄% Normal Saline</td>
<td>77</td>
<td></td>
</tr>
</tbody>
</table>

Plasma Expanders

- Human Albumin 5% and 25%
- Plasma protein fractions: Albumin 4.4%
- Dextrans and Starches
Correction of Volume Abnormalities

- **Volume deficits: Acute vs. Chronic**
  - skin turgor, weight loss, sunken eyes, hypothermia, oliguria, orthostatic hypotension, tachycardia, increased BUN/Cr ratio >15:1, increased HCT (4-8 points/liter deficit), high urine SG and low sodium excretion

**Maintenance Fluid Therapy**

- Replacement of fluids (sensible and insensible losses) and electrolytes lost throughout the day
- 4-2-1 rule
- 100-50-20 rule

**Calculation of Maintenance Fluids**

- **100/50/20 Rule**
  - For 0-10 kg 100ml/kg/d
  - Next 10-20 kg 50ml/kg/d
  - Each 10kg after 20ml/kg/d

- **4/2/1 Rule**
  - 4cc for the 1st 10 kg
  - 2cc for the 2nd 10kg
  - 1cc for each 10 kg after

**Replacement of Ongoing Fluid Losses**

<table>
<thead>
<tr>
<th>Secretion</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>HCO3</th>
<th>H2CO3</th>
<th>Rate (ml/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salivary</td>
<td>50</td>
<td>20</td>
<td>40</td>
<td>30</td>
<td>0</td>
<td>100-1000</td>
</tr>
<tr>
<td>Gastric (basal)</td>
<td>100</td>
<td>10</td>
<td>140</td>
<td>0</td>
<td>30</td>
<td>1000</td>
</tr>
<tr>
<td>Gastric (stim.)</td>
<td>30</td>
<td>10</td>
<td>140</td>
<td>0</td>
<td>100</td>
<td>4200</td>
</tr>
<tr>
<td>Bile</td>
<td>140</td>
<td>5</td>
<td>100</td>
<td>60</td>
<td>0</td>
<td>500-1000</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>140</td>
<td>5</td>
<td>75</td>
<td>100</td>
<td>0</td>
<td>1000</td>
</tr>
<tr>
<td>Duodenum</td>
<td>140</td>
<td>5</td>
<td>80</td>
<td>0</td>
<td>0</td>
<td>1000-2000</td>
</tr>
<tr>
<td>Bilem</td>
<td>140</td>
<td>5</td>
<td>70</td>
<td>50</td>
<td>0</td>
<td>100-2000</td>
</tr>
<tr>
<td>Colon</td>
<td>60</td>
<td>70</td>
<td>15</td>
<td>30</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>
Intraoperative and Postoperative Fluid Therapy

- Anesthesia interrupts normal baroreceptor reflexes
- Fluid loss from blood loss, sequestration, trauma and manipulation of tissues, evaporative losses
- 500ml of blood loss tolerated fairly well
- Replacement of losses with isotonic fluid at rate of 500-1000ml/hr
- Central venous pressure and/or Swan-Ganz catheter
- Special situations in the immediate postoperative period

Special situations in the immediate postoperative period

- Volume Excess: look for weight gain (daily weights), peripheral edema, pulmonary edema, AFIB
- Hyponatremia:
- Hypernatremia:
- Replacement of Na loss with water:
- Decreased Urine volume
- Endogenous water release
- Intracellular shifts
- High-output renal failure

Multiple Trauma Patient / Patient in Shock

What is Shock??

The inadequate delivery of oxygen to the cells of the body

How do we define the presence of shock?

Clinical and laboratory data +/- invasive monitoring
Pulmonary Artery Catheter (S/G)

- Cardiac Output and Index
- Mixed venous blood: P\textsubscript{vo2} and S\textsubscript{vO2} ↑ or ↓
- AVDO\textsubscript{2}
- Oxygen extraction ratio
- DO\textsubscript{2} and VO\textsubscript{2}: Flow dependent VO\textsubscript{2} (Shoemaker)

Flow dependent Oxygen Consumption (VO\textsubscript{2}, Shoemaker)

Adequate resuscitation from shock requires that the patient's hemodynamic status must be pushed to the state of flow-independent oxygen consumption.

Flow Dependent

Flow Independent

O\textsubscript{2} consumption (VO\textsubscript{2})

O\textsubscript{2} Delivery (DO\textsubscript{2})

Resuscitation of the Burn Patient

<table>
<thead>
<tr>
<th>Formula</th>
<th>First 24 Hours</th>
<th>Second 24 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Solution</td>
<td>Calcular</td>
</tr>
<tr>
<td>P.H. Mayo</td>
<td>LR 1-2 L</td>
<td>5-7% of body wt</td>
</tr>
<tr>
<td>Evans</td>
<td>0.9% Lactate</td>
<td>1.0-1.5 ml/kg/hr</td>
</tr>
<tr>
<td></td>
<td>Inj/kg/burn</td>
<td></td>
</tr>
<tr>
<td>Brookes</td>
<td>1.8 ml/kg/hr</td>
<td>1.5-2 ml/kg/hr</td>
</tr>
<tr>
<td></td>
<td>sum</td>
<td></td>
</tr>
<tr>
<td>Parkland</td>
<td>1.8 ml/kg/hr</td>
<td></td>
</tr>
<tr>
<td>Hypertonic</td>
<td>Na</td>
<td></td>
</tr>
<tr>
<td></td>
<td>250mg/kg/hr</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lactated Ringer</td>
<td></td>
</tr>
<tr>
<td>Modified</td>
<td>Brookes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.5 ml/kg/hr</td>
<td></td>
</tr>
</tbody>
</table>
Disorders of Sodium Homeostasis

Maintenance of a normal serum sodium is intimately associated with control of plasma osmolarity (P_{osm})

\[ P_{osm} = 2 \times \text{plasma [Na]} + \frac{\text{[glucose]}}{20} + \frac{\text{[BUN]}}{3} \]

- Plasma [Na] is the major determinant of P_{osm}
- P_{osm} depends on the ability of the kidneys to excrete water
- ADH is the principal regulator of serum osmolarity

Hyponatremia

- Dilutional (increased P_{osm}), pseudo (unnl P_{osm}), true (reduced P_{osm})
- TRUE: reduction in ECF volume; plasma, skin, GI, renal losses; Expanded ECF volume: CHE, cirrhosis, nephrotic syndrome, malnutrition; Normal ECF volume; SIADH, hypothyroid, hypoadrenal, drugs ie: MSO4, TCA’s, chemo agents, pain and nausea

Rule #1: For every 100mg/dl rise in glucose above 100, the [Na] falls by 2mEq/L. Hyperglycemia shifts water from the cells, causing a dilutional hyponatremia

Rule #2: Na deficit = 0.60 X lean body weight (kg) X (140-measured plasma Na)
Hypernatremia

- Usually a result of excessive free water loss, frequently associated with hypovolemia and oliguria
- Extrarenal losses most common, fever, hypervent., burns, hypotonic fluid loss secondary to diarrhea or perspiration
- Polyuria secondary to osmotic diuresis (check urine for glucose); Central DI (give DDAVP)
- Do not correct any faster than 0.5 to 1.0 mEq/L per hour

\[
\text{Water deficit} = \text{lean body weight} \times 0.6 \times \left\{ \frac{\text{plasma} \ [\text{Na}]}{140} - 1 \right\}
\]

Potassium Homeostasis

- Major intracellular cation; Only 2% in ECF
- Slight alterations in homeostasis can cause major effects upon muscle contraction and nerve conduction
- Hypokalemia: losses from GI tract, kidneys and skin; vomiting, NGT suction, diarrhea, burns, diuretics, drugs, metabolic alkalosis, insulin, Conn’s hyperaldosteronism
- Hyperkalemia: Almost always caused by rapid administration of transcellular flux of K+; sustained hyperkalemia implies impairment of renal excretion.

Hypokalemia

- Main danger is cardiac arrhythmias
- Exacerbated by metabolic alkalosis, digoxin or hypercalcaemia
- ECG changes seen usually with levels <3mmol/L
- T-wave flattening or inversion, depressed ST segments, prolonged Q-T interval
Hyperkalemia

- Main danger is weakness and cardiac arrhythmias
- ECG changes begin with an increase in T-wave amplitude, leading to a narrow, peaked and symmetrical T wave, followed by reduced P-wave amplitude and widened QRS.
- If left untreated: sinusoidal ECG complex leading to asystole

Hyperkalemia: Treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mechanism of Action</th>
<th>Time Frame</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV calcium gluconate</td>
<td>Antagonizes the effects of hyperkalemia on the cell</td>
<td>Seconds to minutes</td>
</tr>
<tr>
<td>Glucose, Insulin, NaHCO3</td>
<td>Translocation of potassium into the cells</td>
<td>30-60 minutes</td>
</tr>
<tr>
<td>Kayexalate, Potassium Binding Resin (Oral and Rectal)</td>
<td>Binds and speeds excretion of potassium secreted into colon</td>
<td>1-4 hours rectal &gt;6 hours oral</td>
</tr>
<tr>
<td>Dialysis</td>
<td>Movement across a conc. Gradient and excreted</td>
<td>Immediate</td>
</tr>
</tbody>
</table>

Mineral Homeostasis Calcium

- Total calcium stores 1000g with 99% in the bone
- 1% in ECF of which 40% is free, 60% bound
- Only the free calcium is biologically active
- Bound to albumin as a carrier protein
- Corrected calcium = calcium level + 0.8 X (4-serum albumin)
- Daily intake about 500 to 1500 mg
- GI tract excretes most
Mineral Homeostasis

Hypocalcemia

- Most frequent cause is low serum albumin
- Other causes: acute pancreatitis, infection, fistula, hypothyroidism, massive blood transfusion (chelation of calcium with citrate)
- Perioral numbness and tingling; tetany, seizures; Trousseau’s sign, Chvostek’s sign; prolonged QT;
- Treat with IV calcium, or PO with 1-3 grams of elemental calcium per day

Mineral Homeostasis

Hypercalcemia

- Most frequent cause outpatient: primary hyperparathyroidism; inpatient: malignancy
- Symptoms: confusion, lethargy, weakness, anorexia, nausea, vomiting, pancreatitis and constipation, renal stones, shortened QT interval, nephrogenic DI, polyuria, muscle weakness, fatal arrhythmias

Mineral Homeostasis

Hypercalcemia: Treatment

- > 15mg/dl requires urgent treatment in assoc. with ECG changes
- Vigorous hydration and diuretics
- Diphosphonates and calcitonin (inhibit osteoclast resorption) or reduce serum calcium levels by forming calcium-phosphate complexes (diphosphonates: pamidronate and etidronate)
Mineral Homeostasis
Magnesium and Phosphate

- Magnesium is the primary intracellular divalent cation with 50% found in bone and not exchangeable
- Low levels seen in alcoholics
- High levels are very rare
- Phosphate is the most abundant intracellular anion with only 0.1% in ECF
- Low phosphate means less high energy bonds available, impaired tissue oxygen delivery due to decreased levels of 2,3-DPG, muscle weakness and rhabdomyolysis
- Hyperphosphatemia seen secondarily to impaired renal phosphate excretion and frequently assoc. with hypocalcemia