Common Electrolyte and Fluid Problems in the Hospital

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Educational Objectives

• Understand the concept of maintenance vs replacement fluid
• Understand the mechanism behind gastric fluid loss and its consequence and treatment
• Understand the mechanism behind lower GI (small and large bowel) fluid loss and its consequence and treatment
• Understand the indications for normal saline and its risks
• Understand the utility of lactated Ringer’s solution and its risks
• Be able to recall the sodium content in 3% saline, 0.9% (normal saline), Ringer’s lactate, 0.45% (1/2 normal) saline and an ampule of sodium bicarbonate
References


5. Khan L *Gastroenterology Research and Practice*; 2011:1-7


Disclosures

• None, just workin’ for The Man, like all of us
A good heart and set of kidneys can withstand all but the most woefully incompetent fluid regime

Fluid compartments

- Human body is approximately 60% water

Total body water 42 l

- Intracellular fluid (2/3) 28 l
- Extracellular fluid (1/3) 14 l
- Transcellular fluid (7%) 1 l
  - Plasma (23%) 3 l
  - Interstitial fluid (70%) 10 l
Resting Fluid Balance

Minimal obligatory water intake
- Ingested: 500 ml
- Water in food: 600 ml
- Water from oxidation: 500 ml
- Total: 1600 ml/day

Minimal obligatory water output
- Urine: 500 ml
- Skin: 500 ml
- Respiratory: 400 ml
- Stool: 200 ml
- Total: 1600 ml/day

Quick and easy estimate of Total Body Water (TBW)

- TBW Men = 0.6 x weight (kg)
  - 83 kg x 0.6 = 49.8 L

- TBW Women = 0.55 x weight (kg)
  - 73 kg x 0.55 = 40.15 L
How the Body Responds to Volume Depletion

Hypoperfusion & CO/BP (Volume depletion, shock, sepsis)

↑ SNS (Epi N.E.) → V.C. / Ventricular Contraction

RAAS (AT II/Aldo) V.C., Na/H₂O ®

↑ ADH V.C. H₂O / Na ®

↑ C.O. / BP

↑ Atrial Naturetic Peptide (ANP)

Suppresses: SNS, RAAS, ADH

Glossary

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Definition</th>
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<tbody>
<tr>
<td>SNS</td>
<td>Sympathetic Nervous System</td>
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Fluid Requirements

- **Maintenance**: the amount of fluid required to meet the normal metabolic requirements and compensate for normal losses e.g. sweat, stool, skin and urine

- **Replacement**: the amount of additional fluid needed to correct for abnormal fluid and electrolyte losses e.g. urinary, vomiting, NG suction, diarrhea, skin, lung or “3rd space losses”
Baseline Fluid Requirements

• For normal adults:
  ~20-30 ml/kg/day (Remember YMMV depending on the clinical scenario)

Cellular Response to Water

(a) Consequences of dehydration. If more water than solutes is lost, cells shrink.

(b) Consequences of hypotonic hydration (water gain). If more water than solutes is gained, cells swell.
# Initial Distribution of IV Fluids

<table>
<thead>
<tr>
<th>70 kg man, euvolemic</th>
<th>Intracellular (2/3) 42L</th>
<th>Extracellular (1/3) 28L Divided into:</th>
<th>Extracellular Plasma/Transcellular (1/4) 4L AND</th>
<th>Extracellular Interstitial(3/4) 14 L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal saline/LR 1,000 ml</td>
<td>0 ml</td>
<td>1,000 ml</td>
<td>1,000 ml</td>
<td>0 ml</td>
</tr>
<tr>
<td>1/2 Saline (.45%) (500 ml NS+500ml free water)</td>
<td>333 ml</td>
<td>666 ml</td>
<td>583 ml AND</td>
<td>83 ml</td>
</tr>
<tr>
<td>5% dextrose in water</td>
<td>666 ml</td>
<td>333 ml</td>
<td>83 ml AND</td>
<td>250 ml</td>
</tr>
</tbody>
</table>
Glucose Content

- 5% dextrose may be added to normal saline, 1/2 normal saline, or Ringer’s

  50gm of dextrose=200 kcal/l

This can raise the glucose levels in diabetics, pre-diabetic, patients on corticosteroids or sepsis/multisystem organ failure

This can lead to a **HYPEROSMOLAR** state and osmotic diuresis and further fluid losses

**BUT** under normal conditions, the glucose is rapidly taken up and you are giving **FREE WATER** which diffuses into **ALL** compartments
Normal Saline

**Advantage/Use**
- Still fluid of choice for initial volume resuscitation in hypovolemic shock and head injuries (stays within the vascular space)
- Fluid of choice in hypovolemic hyponatremia
- Na 154 mmol/l  286 mOsm/l
- Cl 154 mmol/l

**Disadvantage**
- Trend toward increased mortality
- Metabolic acidosis (adding chloride to the body)
- pH 5.4
- May lead to hypernatremia and metabolic acidosis
Ringer’s Lactate

**Advantage/Use**
- Emerging as the “fluid of choice” in many clinical scenarios
- Has added Ca and K which may or may not be needed
- Na 130 mmol/l, K 4 mmol/l
- Cl 109 mmol/l, Ca 1.5 mmol/l
- Lactate 28 mmol/l
- 273 mOsm/l, pH 6.5

**Disadvantage**
- Hyponatremic to serum and use with caution in a hyponatremic patients
- Metabolic alkalosis (lactate is converted to bicarbonate in the liver)
- Use with caution in advanced liver disease
- Added Ca$^{++}$ and K$^+$ may not be appropriate in certain patients (e.g. ESRD)
Normal Saline vs Ringer’s Lactate

End Points in Fluid Resuscitation

“The evil that men do lives after them; the good is oft interred with their bones.”
Marc Antony, *Julius Caesar*, Act 3

- **Follow your patient!!!!**
  - Blood pressure (SBP>100 mmHg)
  - Pulse<90 BPM
  - CVP/ PCWP
  - Intraabdominal pressure
  - Urine output
  - Oxygenation
  - Serial lab, correct as needed
Fluid Management

FACTT Trial 2011 Follow-up

• Multicenter trial, 306 AKI patient

• Liberal vs conservative fluid management + furosemide dosage in AKI

• **Liberal fluid group** (10.2 L fluid accumulation over 6 days) had a *higher mortality* vs the conservative group (3.2 L over 6 days)

• **Higher furosemide** dosage (562 mg vs 159 mg) had a *lower mortality* rate, but little effect on fluid balance

Common Fluid Management Scenarios
# Composition of body fluid

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Gastric Fluid Losses

- Recurrent vomiting
- Nasogastric suction
- Gastric fistulas

- Loss of $H^+$ but *greater* loss of $Cl^-$
Volume Depleted Metabolic Alkalosis

Increased $\text{HCO}_3^-$ due to loss of $\text{H}^+$ or $\text{Cl}^-$

No Change
$\text{UC}^+ = \text{unmeasured cations}$

$\text{Na}^+$

$\text{UC}^+ = \text{unmeasured cations}$

$\text{UA}^-$ = Unmeasured anions

$\text{UA}^-$

$\uparrow \text{HCO}_3^-$

$\downarrow \text{Cl}^-$
Volume Depleted/ Chloride Sensitive

• In volume depleted metabolic alkalosis, the body holds onto Cl\(^-\) to compensate for loss of HCO\(_3\)\(^-\) (Maintain charge balance/electrical neutrality)

• In this type of presentation the spot urine Cl\(^-\) will be <20 mEq/l
How the Body Responds to Volume Depletion

Hypoperfusion & CO/BP (Volume depletion, shock, sepsis)

$\uparrow$ SNS (Epi N.E.) $\rightarrow$ V.C. / Ventricular Contraction

RAAS (AT II/Aldo) V.C., Na/H$_2$O $\bigcirc$

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Volume Depleted Metabolic Alkalosis

• **2 Phases**: Initiation and Propagation Phases

• *Initiation Phase*: Loss of chloride *and* volume i.e. vomiting, NG suction, over diuresis, cystic fibrosis

• The volume loss leads to *increase* in SNS, RAAS, and ADH in an attempt to preserve cardiac output and blood pressure
Normal Gastric-Pancreatic Function

- HCl *stimulates* the production of **Secretin** which then *stimulates* the secretion of bicarbonate into the duodenum
Vomiting or NG Suction

• With the *loss of H*⁺ *stimulation*, Secretin is NOT produced, therefore *no* stimulus for bicarbonate secretion. Bicarbonate is then partially reabsorbed into the bloodstream.

• *Loss of Chloride* then leads to *increased* bicarbonate production in the kidney to generate *more* anions.
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Metabolic Alkalosis-Volume Depleted

Volume Depleted (UCl<20 mEq/l) contraction of the bicarbonate space, aka *Hypochloremic* or *contraction alkalosis* - the “space” is smaller hence the bicarbonate concentration will be higher.

**Causes:** vomiting, NG suction, proximal fistulas, colonic villous adenomas, congenital chloridorrhea (Chloride wasting diarrhea), cystic fibrosis
Volume Depleted Metabolic Alkalosis

• 2 Phases: Initiation and propagation phases

• Initiation Phase: Loss of chloride, H⁺, Cl⁻, and volume i.e. vomiting, NG suction, gastric fistulas

• Remember: proportionally more Cl⁻ lost > H⁺

• The volume loss leads to increase in: SNS, RAAS, and ADH activity, which then helps to propagate the alkalosis while trying to maintain perfusion
Propagation of a Volume Depleted Metabolic Alkalosis

• 1. Loss of Cl\(^-\): leads to *more* HCO\(_3^-\) regeneration in the proximal tubule to maintain electrical neutrality (equal number of anions)

• 2. *Volume depleted* stimulation of the RAAS (especially AT II) leads to *increased* sodium reabsorption in the proximal tubule in exchange for H\(^+\) secretion into the lumen

• Thus a “double whammy effect” of a gastric alkalosis: *loss of* H\(^+\) *and gain of* HCO\(_3^-\)
Blood CO\textsubscript{2} + H\textsubscript{2}O

Proximal Tubule Bicarbonate Regeneration

Lumen

CO\textsubscript{2} + H\textsubscript{2}O → H\textsubscript{2}CO\textsubscript{3}

ATII\textsuperscript{*} ATPase

H\textsuperscript{+} + HCO\textsubscript{3}\textsuperscript{-}

Na\textsuperscript{+}

Blood

Na\textsuperscript{+} → K\textsuperscript{+}

ATPase
Propagation of a Volume Depleted Metabolic Alkalosis (2)

Propagation (cont.)

3. Distal Tubule: elevated aldosterone levels from volume depletion lead to enhanced Na\(^+\) uptake and secretion of K\(^+\) and H\(^+\) into the lumen.

Thus in an attempt to hold onto water and sodium, the alkalosis (loss of K\(^+\) and H\(^+\)) worsens.
Role of **Potassium** in Propagation of a Volume Depleted Metabolic Alkalosis

Propagation (cont.)

3. Distal Tubule: elevated aldosterone levels from volume depletion lead to *enhanced* Na\(^+\) uptake and secretion of K\(^+\) and H\(^+\) into the lumen

If there is a K\(^+\) deficiency present, the tubules will secrete *more* H\(^+\) to maintain electrical neutrality
To Regurgitate What Happens

1. Low Chloride stimulates *increased* $\text{HCO}_3^-$ regeneration/reabsorption in the PCT

2. AT-II stimulates *increased* $\text{Na}^+$ reabsorption in *exchange* for $\text{H}^+$ secretion in the PCT (double whammy)

3. Aldosterone stimulates *increased* $\text{Na}^+$ reabsorption AND secretion of $\text{H}^+$ and $\text{K}^+$ in the distal nephron

Principle of Treatment of Gastric Fluid Loss

• Restore baseline fluid and electrolytes deficiency, if present
• Anticipate problems
• Replace~ 1:1 NG output with 0.45% saline with 20-40 mEq/l KCl (every 4-6 hours depending on losses/serial lab) on top to maintenance IV fluids (if a patient loses 900 ml, replace 900 ml)
• Unless Cl⁻ is adequately replaced, the alkalosis will continue
• Unless K⁺ is adequately replaced, the Distal nephron will over-secrete H⁺ in an attempt to maintain electrical neutrality and worsen the alkalosis
Principle of Treatment of Gastric Fluid Loss

• Monitor not only the routine electrolytes but also the Magnesium, Phosphate and Calcium (Remember low Mg^{++} can worsen hypokalemia!)

• Certain condition such as a bowel obstruction will lead to *INCREASED* fluid losses via an NG tube

• *Anticipate* problems rather than having to play catch up
Example

• 82 kg male has an NG tube inserted and attached to suction. He is not eating and has a maintenance fluid running, the am lab was normal

• In the last 6 hours he has lost 1,200 ml in NG fluids

• His replacement fluid would be 0.45% saline with 20-40 mEq/l of KCl (based on lab) over the next 6 hours at approximately 200 ml/hr. (1200ml/6hrs)

• In the following 6 hours he lost 900 ml of NG fluid

• His subsequent replacement fluid would be the same at 150 ml/hr. (900ml/6hrs)
Lower GI Losses/ Metabolic Acidosis
Metabolic Acidosis and Fluids

• **Dilutional**: adding fluids without bicarbonate/lactate *dilutes* the remaining bicarbonate (*dilutional acidosis*)

• Normal saline has a pH 5.4!

• **Hyperchloremic**: saline or other IV fluids, adding Cl⁻

• **Lower GI losses**: Loss of fluids but also *loss* of bicarbonate and *GAIN* of Cl⁻ e.g. ileostomy, colostomy if little large intestine left, ileal conduit/neobladder, remember *much* of the intestinal water is reabsorbed in the colon

• Diarrheal diseases, Crohn’s disease, Ulcerative colitis,
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Diarrhea Associated Metabolic Acidosis

- Na\(^+\) is **reabsorbed** as is Cl\(^-\) which is **"recycled"** and water is osmotically transported into the lumen
- **Loss** of bicarbonate in the stool
- Hypokalemic metabolic acidosis
- Urinary anion gap will be **NEGATIVE** (due to excess urinary NH\(_4^+\) )
Ileal Conduit/ Neobladder
Ileal Conduit, Neobladder, Ileostomy

- 1 Na\(^+\) & 2 Cl\(^-\) are *reabsorbed* and K\(^+\) is blocked by *competing* NH\(_4\)^-.

- Cl\(^-\) is “recycled” via the CTFR Cl\(^-\) channel in exchanged for HCO\(_3\)^- which is secreted.

- Hypokalemic metabolic acidosis.

- Increased risk for stones.

- Urinary anion gap will be **NEGATIVE** (due to excess urinary NH\(_4\)^+).

Hypercalcemia of Malignancy

• **Malignancy**: Thyroid, lung, breast, renal cell, prostate, myeloma, leukemias/lymphomas, Tumor lysis syndrome
• **Very** poor prognostic sign
• **Always** (or almost always) presents with *dehydration*

**Mechanisms**:
1. Direct bone invasion/osteolysis
2. Elevated PTHrP
3. **Humoral hypercalcemia of malignancy**: production of cytokines/osteoclast activating factors-releasing Ca\(^{++}\) from bone
4. **Excess Calcitriol-like factors** (increased GI absorption) granulomatous diseases (Sarcoid, TB)
Hypercalcemia of Malignancy

**Mechanism of Hypercalcemia**
- **PTHrP**: Bone calcium release
  - 80% Breast, lung, NHL
- **PTH**: Bone calcium release
  - Rare case reports
- **1,25(OH)₂D**: Intestinal calcium absorption
  - <1% Lymphomas
- **Osteolysis**: Bone calcium release
  - 20% Breast, lung, myeloma

**Frequency & Tumor Types**
- **PTHrP**: Stimulates resorption
- **PTH**: Stimulates PTHrP expression
- **1,25(OH)₂D**: Stimulates Ca²⁺ reabsorption

**References**
- Reagan P, AJKD 2014;63:141-147
- Mundy G, JASN 2008;19:672-675
Treatment of Hypercalcemia of Malignancy

1. **Aggressive fluid replacement** (normal saline)
   - Restores BP and tissue perfusion
   - Increased Na\(^+\)/water delivery to the kidney decreases reabsorption of Na\(^+\) and Ca\(^{++}\)

2. Once hydrated: **Loop diuretic** (reduces Ca\(^{++}\) uptake in the TAL)
   - do NOT use Thiazides!!!!! WHY?

3. Calcitonin

4. Bisphosphonates (if renal function is normal and phosphorus is not elevated)

5. Steroids

6. Denosumab
## Treatment of Hypercalcemia of Malignancy

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal saline 2-4l/da</td>
<td>Immediate</td>
<td>2-3 days</td>
</tr>
<tr>
<td>Calcitonin 4-8 U/kg SQ 6-12 hours</td>
<td>4-6 hours</td>
<td>2-3 days</td>
</tr>
<tr>
<td>Loop diuretic (once hydrated!)</td>
<td>~5-10 minutes</td>
<td>1-4 hours</td>
</tr>
<tr>
<td>Bisphosphonates</td>
<td>48 hours</td>
<td>~3 weeks</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>7 days</td>
<td>~7 days</td>
</tr>
<tr>
<td>Denosumab</td>
<td>7-10 day</td>
<td>3 months</td>
</tr>
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Sternlicht *Ther Clin Risk Manag* 2015; 11: 1779-1788
Hungry Bone Syndrome
In advanced CKD, PTH levels increase significantly. This leads to a decrease in calcium levels and an increase in phosphate levels.

FGF-23 levels also increase, which disrupts the relationship with vitamin D. The persistent increase in PTH yields PO₄⁻³ excretion.
Hungry Bone Syndrome

• Seen in severe 2° or 3° HyperPTH

• Chronic elevations of PTH lead to increased osteoblastic bone formation and osteoclastic bone resorption

• PTH surgery leads to an abrupt DROP in PTH levels and alteration in the osteoblastic/clastic ratio < 24 hours of surgery

• This leads to massive UPTAKE of Ca++, Mg++, Phosphorus and K by the bone and can lead to severe electrolyte disorders

• Lasts 2-4 days until the cells can downregulate

• Monitor: Ca++, Mg++, phosphorus and K+ levels every 6 hours and replace as needed

• Vitamin D as needed
Treatment of Hypocalcemia

• Use IV only in severe cases
• Ca Gluconate: 10ml=94 mg Ca
• CaCl: 10 ml=273 mg Ca
  Know differences!
• Continuous infusion to optimal Ca$^{++}$ level

• Less severe cases: oral calcium, Tums
• Thiazides increase Ca$^{++}$, used in prevention of Ca$^{++}$ renal stones
• Replace Vitamin D$_3$, Phosphorus, Magnesium if needed
Magnesium Reabsorption in the Thick Ascending Limb (TAL)

• Na\(^+\) and K\(^+\) are reabsorbed (with 2 Cl\(^-\)); this allows an *electrostatic* environment so that Ca\(^{++}\) and Mg\(^{++}\) can be taken up at the tight junction (RED ARROW)

• Mg\(^{++}\) via the *CaSR* regulates the Renal Outer Medullary K Channel (ROMK) which *secretes* K\(^+\) into the lumen
Role of Magnesium in Hypokalemia (TAL)

- In **refractory hypokalemia**, hypomagnesemia is usually the cause
- Mg\(^{++}\) via the **CaSR** regulates the Renal Outer Medullary K Channel (ROMK) which secretes K\(^{+}\) into the lumen
- In **Hypokalemia**, Mg\(^{++}\) acts on the ROMK to decrease K\(^{+}\) secretion
- **If Mg\(^{++}\) is low**: K\(^{+}\) continues to be secreted, despite replacement

http://www.clinsci.org/content/112/4/203.figures-only
Hypomagnesemia

• More common and under-appreciated
• Frequently associated with hypocalcemia and hypokalemia
• Not on routine chemistry panels
Causes of Hypomagnesemia

**GI causes:** low intake, PPI’s, malabsorption, Crohn’s, ileal resection, chronic diarrhea

**Renal Causes:** chronic ETOH, recovery from AKI, thiazide and loop diuretics, hypercalcemia, “hungry bone syndrome”, Bartter’s (TAL), Gitleman's Syndrome (DCT), cisplatin, cyclosporine, Ampho B, aminoglycosides

- Pregnancy and lactation
- Refeeding syndrome: treatment of DKA or hyperglycemia
- Genetic mutations
Presentation of Hypomagnesemia

- Weakness
- Paresthesia
- Tremors
- Seizures
- Tetany
- +Chvostek's and Trousseau's signs
- Hypokalemia or Hypocalcemia
- Cardiac arrhythmias, Torsades de Pointes
Severe Hypomagnesemia: EKG Findings

- Prolonged QT
- U waves
- PVCs
- V fibrillation
- Torsades de Pointes

http://www.imrespdx.com/imrespdx-blog/2017/8/16/syncope-secondary-to-torsades-de-pointes
Hypomagnesemia: Evaluation and Treatment

- Remember serum levels *may not* reflect *total* Mg stores
- i-PTH
- Ca and ionized Ca\(^{++}\), Phosphorus, and Potassium levels
- 24-hour urine for Magnesium

- **IV**: Magnesium Sulfate 2-4 gm
- **Oral**: Magnesium oxide or other forms BUT remember Mg is a laxative and may lead to further losses
- **Amiloride** reduces Mg\(^{++}\) excretion
Phosphorus

• Exists as *organic* and *inorganic* forms

• **Organic**: phospholipids, phosphoesters, nucleic acids, etc.

• **Inorganic**: Ca Phosphates salts, hydroxyapatite in bone, titratable acidity (Na$_2$HPO$_4$/NaH$_2$PO$_4$), excrete excess H$^+$

• **Sources**: meat, fish, milk, lentils, nuts, grains, and beans

• Absorbed in the small intestine

• Essential for ATP and other high energy bond formation and cellular function
Hypophosphatemia

- Phosphate is needed for high energy bonds (ATP, etc.) which fuel much of the active transporters, muscle contraction, cardiac function
- A Medical Emergency

https://americansongwriter.com/2012/12/behind-the-song-jackson-browne-running-on-empty/
Hypophosphatemia: Causes

- **GI**: Malnutrition, *starvation*, low Vit. D/resistance, hypercatabolic, Phosphate binders/TUMS, Crohn’s, resection, diarrhea

- **Renal**: Diuretic phase of AKI, chronic ETOH use, hypoparathyroidism, acidosis, steroids, Fanconi’s syndrome

- **Transcellular Shifts**: Treatment of DKA/hyperglycemia w/ insulin, refeeding syndrome, excess catecholamines, respiratory alkalosis, rapid cell proliferation

- **Genetic**: Hypophosphatemic Rickets (multiple forms), osteomalacia
Presentation and Evaluation of Hypophosphatemia

- **Symptoms**: weakness, confusion, difficulty breathing, difficulty swallowing, dysrhythmias, rhabdomyolysis, cardiac or respiratory arrest, other associated electrolyte abnormalities: hypokalemia, hypomagnesemia

- **Anticipate** in critically ill patients

- **Lab**: i-PTH, K, Ca, Mg, glucose alkaline phosphatase, 24-hour urine for phosphorus, 25 and 1,25 D₃
Hypophosphatemia: Treatment

**IV-Emergent:** (K⁺ or Na⁺ Phosphate):
- **NaPhos**: 4 mmol/ml Na, 3 mmol/ml phosphorus
- **KPhos**: 4.4 mmol/ml K, 3 mmol/ml phosphorus

Be sure to monitor K⁺ and Mg²⁺ levels!

**Oral**: preferred
- 1 qt. skim milk = 1000mg phosphorus
- **NeutraPhos**: 250mg/capsule
Refeeding Syndrome

• Recognized after WW2
• Rapid shift of metabolism from starvation (gluconeogenesis) to influx of glucose and amino acids/protein and increased metabolism
• Seen today in critical care units, immune disorders, and cancer
• Common in chronic ETOH
• IV, oral, or tube feedings

• Mechanism of refeeding:
• Large amounts of glucose are rapidly metabolized
  • In the process, phosphorus, Mg++, K+, and thiamine levels are consumed/depleted
• Hypophosphatemia, hypomagnesemia, hypokalemia
• Frequent monitoring and replace as needed
Chronic Alcohol and the Kidney

- Underappreciated target organ
- ETOH is directly toxic to various pumps and co-transporters in the tubules
- Reversible with abstinence
- Hypertension: increased sympathetic tone
- Dehydration: diuretic effect of ETOH and inhibition of ADH secretion (Central Diabetes Insipidus)
- Hyponatremia: excess water and too few solutes (Beer Potomania)


- Hypokalemia: excess losses and treatment w/ glucose
- Hypomagnesemia: excess losses and treatment w/ glucose
- Hypocalcemia: excess losses, ETOH suppresses PTH, Vitamin D deficiency, low albumin
- Hypophosphatemia: excess losses, poor dietary intake, insulin administration, treatment w/ glucose
- Acute Thiamine deficiency leading to Wernicke’s encephalopathy or Beriberi and CHF
Closing points

• Try to anticipate fluid requirements and losses if possible
• In a volume depleted metabolic alkalosis do not forget that Cl− replacement is essential to restore much needed anions and correct the alkalosis
• Hypercalcemia of malignancy is treatable, but does carry a poor prognosis
• Refeeding syndrome is seen in a variety of hospital settings and can be deadly (hypomagnesemia, hypophosphatemia) if not anticipated
• Alcohol has a number of direct and indirect effects on renal function
Thank you,
Questions?
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