Fluids and Electrolytes

Learning Objectives

- Total Body Fluid
- Intravascular Volume Depletion
- Fluid resuscitation vs. Maintenance IV Fluid
- Osmolarity of IV Fluids
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Hypomagnesemia
- Hypermagmesemia
- Hypophosphatemia
- Hyperphosphatemia
- Hypocalcemia
- Hypercalcemia

### Body Fluid Compartments:

- Male 60% of LBW is fluid
- Female 50% of LBW is fluid
- 70 kg male
- BW x 0.6 = TBW
- 70kg x 0.6 = 42 L
- ICF = 2/3 x 42 = 28L
- ECF = 1/3 x 42 = 14L

- ECF
  - 1/4 is intravascular plasma
  - 1/4 x 14 = 5.6L

- 3/4 is interstitial
  - 3/4 x 14 = 8.4L

TBW

ICF

Extravascular

\rightarrow Interstitial

Fluid

8.4L

Intravascular

\rightarrow plasma

5.6L
Water Steady State

- Amount Ingested = Amount Eliminated

- Pathological losses
  - vascular bleeding (H2O, Na+)
  - vomiting (H2O, H+)
  - diarrhea (H2O, HCO3-).

Fluid Requirement

- The average adult requires approximately 35-45 ml/kg/d
- NRC* recommends 1 to 2 ml of water for each kcal of energy expenditure

*NRC= National research council
### Fluid Requirement

- 1st 10 kilogram 100 cc/kg
- 2nd 10 kilogram 50 cc/kg
- Rest of the weight 20 to 30 cc/kg

Example: 50 kg patient

1st 10 kg x 100cc = 1000 cc  
2nd 10 kg x 50cc = 500cc  
Rest 30 kg x 30cc = 900cc  
**total** = 2400 cc

### Fluid

- Fluid needs are altered by the patient's functional cardiac, hepatic, pulmonary, and renal status
- Fluid needs increase with fever, diarrhea, hemorrhage, surgical drains, and loss of skin integrity like burns, open wounds
Regulation of Fluids:

Response to Decreased volume and Blood pressure

1. Blood pressure falls
2. Renin
3. Angiotensin
4. Aldosterone
Salt retention
Blood pressure rises

Regulation of Fluids:

Response to Increased volume and Blood pressure

ADH (antidiuretic hormone)

Decrease in NaCl concentration

Decrease ADH output

Plummary decreases output of ADH

Decrease Thirst

Diuresis (water loss)

Reduces water content of ECFV

Increases osmotic concentration (NaCl)
Hypovolemia

Causes of Hypovolemia

- Hypovolemia
- Abnormally low volume of body fluid in intravascular and/or interstitial compartments
- Causes
  - Vomiting
  - Diarrhea
  - Fever
  - Excess sweating
  - Burns
  - Diabetes insipidus
  - Uncontrolled diabetes mellitus
Other Causes of Water Loss

- Fever
- Burns
- Diarrhea
- Vomiting
- N-G Suction
- Fistulas
- Wound drainage

Signs and Symptoms

- Acute weight loss
- Decreased skin turgor
- Concentrated urine
- Weak, rapid pulse
- Increased capillary filling time
- Sensations of thirst, weakness, dizziness, muscle cramps
Signs of Hypovolemia:

- Diminished skin turgor
- Dry oral mucus membrane
- Oliguria
  - <500ml/day
  - normal: 0.5~1ml/kg/h
- Tachycardia (100 beats/min)
- Hypotension (SBP < 90 mm Hg)
- Hypoperfusion → cyanosis
- Altered mental status
Clinical Diagnosis of Hypovolemia:

- Thorough history taking: poor intake, GI bleeding…etc
- BUN : Creatinine > 20 : 1
- Increased specific gravity
- Increased hematocrit
- Electrolytes imbalance
- Acid-base disorder

Complications

- Reduced cardiac function, organ hypo perfusion and multi-organ failure, renal failure, shock and death.
Fluid Replacement

- Crystalloids
  - Normal saline (0.9% NaCl)
  - Dextrose 5%
- Colloids
  - Albumin 5%, 25%
  - Hetastarch

Parenteral Fluid Therapy:

- Crystalloids: (0.9% NaCl)
  - Contain Na, and Cl as the main osmotically active particle do not freely cross into cells but they will distribute evenly in the EC (IV + IT)
- Crystalloids: (D5W)
  - D5W -> H2O + CO2
  - Water will distribute in TBW
**Body Fluid Compartments:**

If 1 liter of NS is given, only 250 ml will stay in intravascular.

1000 ml x \(\frac{1}{4}\) = 250 ml

If 1 liter of D5W is given, only about 100 ml will stay in intravascular.

1000 ml x \(\frac{1}{3}\) = 333 ml

333 ml x \(\frac{1}{4}\) = 83 ml

**Crystalloids:**

- Isotonic crystalloids
  - Lactated Ringer’s, 0.9% NaCl
  - only 25% remain intravascularly

- Hypotonic solutions
  - D5W
  - less than 10% remain intra-vascularly, inadequate for fluid resuscitation
**Colloid Solutions:**

- Contain high molecular weight substances → too large to cross capillary walls
- Preparations
  - Albumin: 5%, 25%
  - Dextran
  - Hetastarch

**Body Fluid Compartments:**

If 1 liter of 5% albumin is given, all will stay in intravascular because of its large molecule that will not cross cell membrane.

1000 ml x 1 = 1000 ml

If 100 ml of 25% albumin is given, it will draw 5 times of its volume into intravascular compartment.

100 ml x 5 = 500 ml
The Influence of Colloid & Crystalloid on Blood Volume:

Fluid Resuscitation

- Calculate the fluid deficit base on serum sodium level (assume patient Na is 120 mmole/l and patient weight is 70 kg)

\[
\text{Fluid deficit} = BW \times 0.5 \left( \frac{\text{Avg Na} - \text{pt Na}}{\text{Na avg}} \right)
\]

\[
= \frac{70 \times 0.5 \left( 140 - 120 \right)}{140}
\]

\[
= 5 \text{ L}
\]
**Fluid Resuscitation**

- Calculate the fluid deficit based on patient actual weight.
  - If you know the patient weight before the dehydration, simply subtract patient current weight from patient previous weight.
  - Pt wt before dehydration – pt current wt
  - Exp if pt weight was 70 kg before and now pt weight 65 kg then
  - 70 kg – 65 kg = 5 kg equal to 5 L of water loss (s.g for water is 1)

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**Fluid Resuscitation**

- Use crystalloids (NS or Lactate Ranger).
- Colloids is not superior to crystalloids.
- Administer 500-1000 ml/hr bolus (30-60 mins) and then 250-500 ml/hr for 6 to 8 hours and rest of the fluid within 24 hours.
- Maintain IV fluid (D5 ½ NS) until vital signs are normalized and patient is able to take adequate oral fluid.
Osmolarity

- Isotonic solution
- Hypotonic solution
- Hypertonic solution

Osmolarity

- Plasma osmolarity
  \[ p\text{Osm} = \text{Na} + \text{Cl} + \text{BUN} + \text{Glucose} \]
  exp: if pt Serum Na = 145 mmol/l and Glucose is 6 mmole/l and BUN is 6 mole/l, then osmolarity of serum is
  \[ 145 + 145 + 6 + 6 = 302 \]
Osmolarity

- Calculate the osmolarity of 1L NS?
  MW of Na = 23, Cl = 35.5
  0.9% NaCL of 1 L
  9 gm NaCl
  $9/23 + 35.5 = 0.154$ mole (154 mmole)
  1 mole of NaCl = 1 mole Na + 1 mole Cl
    $= 2$
  154 mmole/l x 2 = 308

Osmolarity

- Calculate the osmolarity of 1L 3%NaCl?
  MW of Na = 23, Cl = 35.5
  3% NaCL of 1 L
  30 gm NaCl
  $30/23 + 35.5 = 0.154$ mole (513 mmole)
  1 mole of NaCl = 1 mole Na + 1 mole Cl
    $= 2$
  513 mmole/l x 2 = 1026
Osmolarity

- Calculate the osmolarity of 1L D5W?
  - MW of dextrose 180
  - D5W of 1 L
  - 50 gm dextrose
  - $\frac{50}{180} = 0.278$ mole (278 mmole)
  - $278 \text{ mmole} / \text{l} \times 1 = 278 \text{ mosm/l}$

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Osmolarity

- Calculate the osmolarity of D5WNS?
Osmolarity

- What happen if you infuse hypotonic solution?

RBC will swell and rapture
Also will cause brain edema

Osmotic Conditions: H = High
Solution is Hypotonic

Cell: L water, H solute
Environment: H water, L solute, hypotonic to cell

Osmolarity

- What happen if you infuse hypertonic solution to you RBC?

RBC will shrink and will not carry oxygen properly

Osmotic Conditions: H = High
Solution is Hypertonic

Cell: H water; L solute
Environment: L water, H solute
### Common parenteral fluid therapy

<table>
<thead>
<tr>
<th>Solutions</th>
<th>Volumes</th>
<th>Na⁺</th>
<th>K⁺</th>
<th>Ca²⁺</th>
<th>Mg²⁺</th>
<th>Cl⁻</th>
<th>HCO₃⁻</th>
<th>Dextrose</th>
<th>mOsm/L</th>
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<td>28</td>
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<td>0.9% NaCl</td>
<td>154</td>
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<td>308</td>
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<tr>
<td>0.45% NaCl</td>
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<td>154</td>
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<td>Lactated</td>
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<td>103</td>
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<td>1026</td>
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<td>6% Hetastarch</td>
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<td>310</td>
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<tr>
<td>5% Albumin</td>
<td>250-500</td>
<td>130-160</td>
<td>&lt;2.5</td>
<td>130-160</td>
<td>330</td>
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<tr>
<td>25% Albumin</td>
<td>20-30,100</td>
<td>130-160</td>
<td>&lt;2.5</td>
<td>130-160</td>
<td>330</td>
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</tbody>
</table>

### Hypervolemia

- Excess fluid in the extracellular compartment as a result of fluid or Na retention when compensatory mechanisms fail to restore fluid balance or from renal failure
Causes

- Cardiovascular – Heart failure
- Urinary – Renal failure
- Hepatic – Liver failure, cirrhosis
- Other – Drug therapy (i.e., corticosteroids), high sodium intake, protein malnutrition

Sign and Symptoms

- Tachycardia
- Tachypnea
- Dyspnea
- Lung sound (Crackles)
- Pitting edema or generalized edema
- S3 gallop (added heart sound)
- Increase CVP and PCWP
- Raise JVP (distended neck vein)
- Weight gain and change in mental status
Lab Abnormalities

Lab data
- ↓ Hct (dilutional)
- Low serum osmolality
- Low specific gravity
- ↓ BUN (dilutional)

Management

- Sodium restriction with No more than 2 grams of salt per day
- Fluid restriction if necessary
- Diuretic
  1. Furosemide dose and route depends on patient condition and underlining diseases
**IV Loop diuretic (Furosemide)**

- Patient with a cute CHF with pulmonary edema and difficult in breathing
- Patient with a cute or chronic renal failure with massive fluid overload
- Patient with liver cirrhosis and refractory to oral diuretic (furosemide)
- Dose can be range from 80-240 mg/day
- Can be bolus in divided doses or continuous infusion range from 5-10mg/hour

**Monitoring Parameters**

- Fluid intake and output (trying to create at least 1-2 liters of negative fluid balance)
- Patient weight
- Monitor the vital sign BP, RR, PR
- ABG or oxygen saturation
- Chest auscultation If dyspnea or orthopnea
- Urea and electrolytes (make sure that patient does not develop renal impairment or hyponatremia or hypokalemia)
Composition of Body Fluids and electrolytes:

### Cations
- Ca\(^{2+}\)
- Mg\(^{2+}\)
- Na\(^+\)
- K\(^+\)

### Anions
- Cl\(^-\)
- PO\(_4^{3-}\)
- HCO\(_3^-\)
- Organic anion

Summary of Ionic composition

- Plasma
- Interstitial
- Cell

- Na
- K
- Cl
- P

- Protein
- Organic Phos.
- Inorganic Phos.
- Bicarbonate
- Chloride
**Sodium**

- Normal 135-145 mEq/L
- Major cation in ECF
- Regulates voltage of action potential; transmission of impulses in nerve and muscle fibers
- Main factors in determining ECF volume
- Helps maintain acid-base balance

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**Hyponatremia**

- Results from excess Na loss or water gain
  - GI losses (vomiting and diarrhea)
  - Diuretic therapy
  - Severe renal dysfunction (ATN)
  - Administration of hypotonic fluid (1/2NS)
  - DKA, HHS
  - Unregulated production of ADH (pneumonia, brain trauma, lung cancer etc)
  - Some drugs (Li, thiazide)
Sign and Symptoms

- Clinical manifestations
  - ↓ BP
  - Confusion, nausea, malaise
  - Lethargy and headache (115-120 mmol/l)
  - Seizure and coma (110-115 mmol/l)
  - Decreased muscle tone, twitching and tremors
  - Vomiting, diarrhea, and cramps

Assessment

- Labs
  - Decreased Na, Cl, Bicarbonate
  - Urine specific gravity ↓ 1.010
  - Estimated Na deficit (calculation)
    Na deficit = 0.6 x LBW (140 – patient serum Na)
    Exp: if patient is 70 kg and his serum Na=120
    = 0.6 x 70 (140 – 120)
    = 42 x 20
    = 840 mmole
Treatment

- Interventions
  - If patient is normovolemic or edematous
    - Fluid restriction
  - If patient is intravascular volume depletion
    - IV 0.9% NS or LR
    - Avoid rapid Na correction
    - A change of no more than 10-12 mmole/day
    - Rapid correction of Na can cause central pontine myelinolysis and death
  - 120-125 mmole/l is a reasonable goal and safe

Hypertonic Saline 3% NaCl

- Use in patient with symptomatic hyponatremia such as in seizure, comatose patient, or patient with brain edema
- 3% NaCl 250ml with an infusion rate of 1-2ml/kg/hr
  - exp; 70 kg patient
  - 70kg x 1ml/kg = 70 ml
  - 250ml/70ml = 3.5 hours
**Hypernatremia (> 145mmol/l)**

- Gain of Na in excess of water or loss of water in excess of Na
- **Causes**
  - Deprivation of water
  - Hypertonic tube feedings without water supplements
  - Watery diarrhea
  - Increased insensible water loss (burn, fever)
  - Renal failure (unable to excrete Na)
  - Use of large doses of adrenal corticoids
  - Excess sodium intake (NS or HS)

**Signs/Symptoms**

- Early: Generalized muscle weakness, faintness, muscle fatigue, headache, tachycardia, nausea and vomiting
- Moderate: Confusion, thirst
- Late: Edema, restlessness, thirst, hyperreflexia, muscle twitching, irritability, seizures, possible coma (Na > 158 mmol/l)
- Severe: Permanent brain damage form cerebral dehydration and intracerebral hemorrhage, hypertension (Na > 158 mmol/l)
**Labs**

- Increased serum Na
- Increased serum osmolality
- Increased urine specific gravity

**Treatment** *(Euvolemic with hypernatremia)*

- IV D5W to replace ECF volume if patient is symptomatic with hypernatremia

\[
\text{D5W need} = 0.4 \times \text{LBW} \times \frac{\text{pt serum Na} - \text{Na normal}}{\text{Na}}
\]

exp: patient 70 kg serum Na = 158, normal Na = 135

\[
= 0.4 \times 70 \frac{(158 - 135)}{135} \\
= 4.77 \text{ L}
\]

- Gradual lowering with Na level with D5W
  - Decrease by no more than 0.5 mmol/l/hr or 12 mmol/l/day
**Treatment (hypovolemic with hypernatremia)**

**Non-symptomatic patient**
- Orally (plain water) to replace ECF volume if patient is not symptomatic with excessive free water losses

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**Treatment (Hypervolemic with hypernatremia)**

- If patient is hypervolemic with hypernatremia; then loop diuretic is the drug of choice
Treatment *(Hypovolemic with hypernatremia)*

- Hypotonic fluid loss is the most commonest cause of hypernatremia
  - Exp: osmotic diuretic and gastroenteritis
- Signs of intravascular depletion
- Treat with NS or 1/2NS followed by correction of free water deficit

Evaluation

- Normalization of serum Na level over days
- Resolution of symptoms
Potassium

- Normal 3.5-5.5 mEq/L
- Major ICF cation
- Vital in maintaining normal cardiac and neuromuscular function, influences nerve impulse conduction, important in glucose metabolism, helps maintain acid-base balance, control fluid movement in and out of cells by osmosis

Hypokalemia

- Serum potassium level below 3.5 mEq/L
- Causes
  - Loss of GI secretions (diarrhea)
  - Excessive renal excretion of K
  - Movement of K into the cells with insulin (Rx DKA)
  - Prolonged fluid administration without K supplementation
  - Diuretics (some) and beta agonist (albuterol)
  - Alkalosis
**Signs/Symptoms**

- Skeletal muscle weakness, ↓ smooth muscle function, ↓ respiratory muscle function
- EKG changes, possible cardiac arrest
- Paralytic ileus
- Nausea, vomiting
- Metabolic alkalosis
- Mental depression and confusion

**Treatment**

- Deficit can be estimated as 200 -400 mmol K for every 1 mmol/l reduction in plasma K
<table>
<thead>
<tr>
<th>Plasma K levels Mmol/l</th>
<th>Treatment</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 – 3.5</td>
<td>Oral KCl 60-80 mmol/d if no sign or symptoms</td>
<td>Plasma K level rise by about 1.5 mmol/l</td>
</tr>
<tr>
<td>2.5 -3</td>
<td>Oral KCl 120 mmol/d or IV 10 -20 mmol/hr if sign or symptoms</td>
<td>Plasma K level rise by about 2.0 mmol/l</td>
</tr>
<tr>
<td>2 -2.5</td>
<td>IV KCl 10 -20 mmol/hr</td>
<td>Consider continuous EKG monitoring</td>
</tr>
<tr>
<td>Less than 2</td>
<td>IV KCl 20 -40 mmol/hr</td>
<td>Requires continuous EKG monitoring</td>
</tr>
</tbody>
</table>

**Caution**

- Don’t mix K in dextrose
- No more than K 10 mmol/hr to be infused in general ward
- If rate exceed more than 10 mmol/hr, then consider EKG monitor
Monitoring

Monitor

- Potassium level
- EKG
- Bowel sounds
- Muscle strength

Hyperkalemia

- Serum potassium level above 5.3 mEq/L
- Causes
  - Excessive K intake (IV or PO) especially in renal failure
  - CRF
  - Tissue trauma
  - Acidosis
  - Catabolic state
  - ACE inhibitors, K-sparing diuretics, B blockers
**Signs/Symptoms**

- ECG changes – tachycardia to bradycardia to possible cardiac arrest
  - Peaked, narrowed T waves
- Cardiac arrhythmias (VF
- Muscle weakness and paralysis
- Paresthesia of tongue, face, hands, and feet
- N/V, cramping, diarrhea
- Metabolic acidosis

**Treatment**

Asymptomatic elevation of plasma K

- Use cation exchange resin (calcium or sodium polystyrene sulfonate *Kayexalate*)
- 15- 30 grams 3 to 4 times/day as orally or rectal enema
- Specially used in chronic renal failure patient with hyperkalemia.
- Avoid K containing food
**Treatment (symptomatic)**

Urgent immediate treatment is needed if patient
1. Plasma K+ of 8mmol/l
2. Severe muscle weakness
3. ECK changes
   10% Ca gluconate 20ml should be given immediately if a patient has hyperkalemia-induced-arrhythmias (2 grams IV bolus)

**Treatment (symptomatic)**

- Sodium bicarbonate 1 mmol/kg can be given if patient has acidosis (pH of < 7)
- 50% glucose solution 50 ml (25 gm) with 10 units of insulin → push K+ intracellular and lower serum K+ level by 1 to 1.5 mmol/l in one hour
- B2 adrenergic agonist → salbutamol 10 -20 mg in NS as nebulizer over 10 mins → lower K+ level by 1 to 1.5 mmol/l in one hour to two hours
- Kayexalate PO or PR
- Hemodialysis
- Avoid K in foods, fluids, salt substitutes
**Evaluation**

- Normal serum K values
- Resolution of symptoms
- Treat underlying cause if possible

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**Calcium**

- Normal 2.25-2.75 mmol/L
- 99% of Ca in bones, other 1% in ECF and soft tissues
- ECF Calcium – $\frac{1}{2}$ is bound to protein – levels influenced by serum albumin state
- Ionized Calcium – used in physiologic activities – crucial for neuromuscular activity
**Calcium**

- Required for blood coagulation, neuromuscular contraction, enzymatic activity, and strength and durability of bones and teeth
- Nerve cell membranes less excitable with enough calcium
- Ca absorption and concentration influenced by Vit D, calcitriol (active form of Vitamin D), PTH, calcitonin, serum concentration of Ca and Phos

**Causes of Hypocalcemia**

- Hypoparathyroidism (depressed function or surgical removal of the parathyroid gland)
- Hypomagnesemia
- Hyperphosphatemia
- Administration of large quantities of stored blood (preserved with citrate)
- Renal insufficiency
- ↓ Absorption of Vitamin D from intestines
Signs/Symptoms

- Abdominal and/or extremity cramping
- Tingling and numbness
- Positive Chvostek or Trousseau signs
- Tetany; hyperactive reflexes
- Irritability, reduced cognitive ability, seizures
- Prolonged QT on ECG, hypotension, decreased myocardial contractility
- Abnormal clotting

Treatment

- Asymptomatic hypocalcaemia associated with hypoalbuminemia → check for corrected Ca++
  Corrected Ca = Serum Ca + (normal S albumin – pt serum albumin) x 0.02
  Exp: if patient serum Ca is 1.8 mmol/l and albumin is 20 gm/l then corrected Ca is (assume Normal Ca is 45 gm/l)
  \[
  = 1.8 + (45 - 20) \times 0.02 \\
  = 1.8 + 25 \times 0.02 \\
  = 1.8 + 0.5 \\
  = 2.3
  \]
Treatment

Asymptomatic hypocalcemia
- Oral calcium salts (mild) – 2 – 4 gm of elemental Ca++/day with Vit D supplementation

Symptomatic hypocalcemia
- IV calcium as 10% calcium chloride 10 ml or 10% calcium gluconate 20ml (270 mg elemental Ca)– give with caution over 5-10 mins followed by continuous infusion of Ca at a rate of 0.5 – 2 mg/kg/hr
- Don’t exceed infusion rate 60 mg/min
- Close monitor for hypotension and bradycardia
- Vitamin D supplementation

Monitoring

- Close monitoring of serum Ca++
- Phosphorus level
- Magnesium level
- Vitamin D level
- Albumin level
**Hypercalcemia**

- Causes
  - Mobilization of Ca from bone
  - Malignancy (non-small cell and small cell lung cancer, breast cancer, lymphomas, renal cell)
  - Hyperparathyroidism
  - Immobilization – causes bone loss
  - Thiazide diuretics and hormonal therapy
  - Thyrotoxicosis
  - Excessive ingestion of Ca or Vit D

**Signs/Symptoms**

- Anorexia, constipation
- Generalized muscle weakness, lethargy, loss of muscle tone, ataxia
- Depression, fatigue, confusion, coma
- Dysrhythmias and heart block
- Deep bone pain and demineralization
- Renal calculi
- Pathologic bone fractures
Hypercalcemic Crisis

- Emergency - level of 4-4.5 mmol/L
- Intractable nausea, dehydration, stupor, coma, azotemia, hypokalemia, hypomagnesemia, hypernatremia
- High mortality rate from cardiac arrest

Treatment

- NS IV infusion 3 – 6 L over 24 hours followed by loop diuretic to prevent over load
- I&O hourly to avoid over hydration
- Biphosphonate- pamidronate 60mg IV once (inhibit bone resorption)
- Corticosteroids (HC 100 q6 hr) and Mithramycin in lymphomas and myeloma patient
- Calcitonin 2-8 IU/kg IV or SQ q6 to q12 to inhibit PTH effect
- Phosphorus in patient with hypophosphatemia
- Encourage fluids
- Dialysis in renal patient with hypercalcemia
**Evaluation**

- Normal serum calcium levels
- Improvement of signs and symptoms specially heart block, PVC, tachycardia, mental status

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**Magnesium**

- Normal 0.7 to 1.25 mmol/l
- Important in CHO and protein metabolism
- Plays significant role in nerve cell conduction
- Important in transmitting CNS messages and maintaining neuromuscular activity
- Causes vasodilatation
- Decreases peripheral vascular resistance
Hypomagnesemia

- Causes
  - Decreased intake or decreased absorption or excessive loss through urinary or bowel elimination
  - Acute pancreatitis, starvation, malabsorption syndrome, chronic alcoholism, burns, prolonged hyperalimentation without adequate Mg supplement
  - Hypoparathyroidism with hypocalcemia
  - Diuretic therapy

Signs/Symptoms

- Tremors, tetany, ↑ reflexes, paresthesias of feet and legs, convulsions
- Positive Babinski, Chvostek and Trousseau signs
- Personality changes with agitation, depression or confusion, hallucinations
- ECG changes (PVC’S, V-tach and V-fib)
Treatment

- **Mild**
  - Diet – Best sources are unprocessed cereal grains, nuts, green leafy vegetables, dairy products, dried fruits, meat, fish
  - Magnesium salts (MgO 400mg/d)
- **More severe**
  - MgSO4 IM
  - MgSO4 IV slowly

Treatment of Severe Symptomatic Hypomagnesemia

- Treated with 1-2g Mg sulfate (4mEq/ml) IV over 15 min, followed by infusion of 6g Mg sulfate in 1L or more IV fluid over 24hrs
- Need to replenish intracellular stores, the infusion should be continued for 3-7 days
- Serum Mg should be measured q24h and the infusion rate adjusted to maintain a serum Mg level of <1.25 mmol/L

Treatment of Severe Symptomatic Hypomagnesemia

- In patient with normal renal function, excess Mg is readily excreted, and there is little risk of causing hypermagnesemia with recommended doses.
- Mg must be given with extreme caution in renal failure due to the risk of accumulation of Mg and can cause hypermagnesemia.

Monitoring

- Monitor Mg level q 12 – 24 hrs
- Monitor VS
- Knee reflexes
- Check swallow reflex
**Hypermagnesemia**

- Most common cause is renal failure, especially if taking large amounts of Mg-containing antacids or cathartics
- DKA with severe water loss
- Signs and symptoms
  - Hypotension, drowsiness, absent DTRs, respiratory depression, coma, cardiac arrest
  - ECG – Bradycardia, cardiac arrest

**Treatment**

- Withhold Mg-containing products
- Calcium chloride or gluconate IV for acute symptoms (10% Ca gluconate 10-20ml over 15-30 mins)
- NS IV hydration and diuretics
- Glucose(25gms) and Insulin(10 units)
- Hemodialysis
Evaluation

- Serum magnesium levels WNL
- Improvement of symptoms

**Phosphorus** Normal 2.5-4.5 mg/dL

- The primary anion in the intracellular fluid
- Crucial to cell membrane integrity, muscle function, neurologic function and metabolism of carbs, fats and protein
- Functions in ATP formation, phagocytosis, platelet function and formation of bones and teeth
- Influenced by parathyroid hormone and has inverse relationship to Calcium
Hypophosphotemia

- Causes
  - Malnutrition
  - Hyperparathyroidism
  - Certain renal tubular defects
  - Metabolic acidosis (esp. DKA)
  - Disorders causing hypercalcemia

Sign and Symptoms

- Musculoskeletal
  - Muscle weakness
  - Respiratory muscle failure
  - Osteomalacia
  - Pathological fractures

- CNS
  - Confusion
  - Anxiety
  - Seizures
  - Coma
**Sign and Symptoms**

- Cardiac
  - hypotension
  - decreased cardiac output
- Hematologic
  - hemolytic anemia
  - easy bruising
  - infection risk

**Treatment**

- Treatment of moderate to severe deficiency
  - Oral or IV phosphate (do not exceed rate of 10 mEq/h)
  - Monitor levels during treatment
**Hyperphosphatemia**

- **Causes**
  - Chronic renal failure (most common)
  - Hyperthyroidism, hypoparathyroidism
  - Severe catabolic states
  - Conditions causing hypocalcemia

Net effect of PTH → ↑ serum calcium  ↓ serum phosphate

Net effect of calcitriol → ↑ serum calcium  ↑ serum phosphate

**Role of PTH**

- Stimulates renal reabsorption of calcium
- Inhibits renal reabsorption of phosphate
- Stimulates bone resorption
- Inhibits bone formation and mineralization
- Stimulates synthesis of calcitriol

Net effect of PTH → ↑ serum calcium  ↓ serum phosphate
Sign and Symptoms

- Cardiac irregularities
  - Hyperreflexia
  - Eating poorly
  - Muscle weakness
  - Nausea

Treatment

- Prevention is the goal
- Restrict phosphate-containing foods
- Administer phosphate-binding agents (Ca carbonate, sevelamar, lanthanum)
- Diuretics
- Cinacalcet – increase the sensitivity of Ca receptor on PTH gland to Ca conc $\rightarrow \downarrow$ PTH
- Treatment may need to focus on correcting calcium levels
Evaluation

- Lab values within normal limits
- Improvement of symptoms
Regulation of blood pH

- The lungs and kidneys play important role in regulating blood pH.
- The lungs regulate pH through retention (hypoventilation) or elimination (hyperventilation) of CO₂ by changing the rate and volume of ventilation.
- The kidneys regulate pH by excreting acid, primarily in the ammonium ion (NH₄⁺), and by reclaiming HCO₃⁻ from the glomerular filtrate (and adding it back to the blood).

Normal Values for Blood Buffer in Arterial Blood.

- The following values are determined by blood gas analyzer:
  - pH 7.35 – 7.45
  - PCO₂ 35 – 45 mm Hg
  - H₂CO₃ 2.4 mmoles/L of plasma
  - HCO₃⁻ 24 mmoles/L of plasma
  - PO₂ 80 – 110 mm Hg
Blood Gases

- In the lungs, $O_2$ enters the blood, while $CO_2$ from the blood is released.
- In the tissues, $O_2$ enters the cells, which release $CO_2$ into the blood.

Four Basic Types of Imbalance

- Respiratory Acidosis
- Respiratory Alkalosis
- Metabolic Acidosis
- Metabolic Alkalosis
Respiratory Acidosis

- Carbonic acid excess
  - Exhaling of CO2 inhibited
  - Carbonic acid builds up
  - pH falls below 7.35
  - Cause = Hypoventilation (see chart)
  - When CO2 level rises hypoventilation, producing more H2CO3, the equilibrium produces more H3O+, which lowers the pH – acidosis.

\[
\text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}_3\text{O}^+ + \text{HCO}_3^-
\]

Respiratory Acidosis: CO2 ↑ pH ↓

- Symptoms: Failure to ventilate, suppression of breathing, disorientation, weakness, coma
- Causes: Lung disease blocking gas diffusion (e.g., emphysema, pneumonia, bronchitis, and asthma); depression of respiratory center by drugs, cardiopulmonary arrest, stroke, poliomyelitis, or nervous system disorders
**Acid-Base Imbalances**

- **Normal**

  1.2 mEq/L \( \text{H}_2\text{CO}_3 \) \[ \text{HCO}_3 \] 24 mEq/L

- **Respiratory Acidosis**

  1.84 mEq/L \( \text{H}_2\text{CO}_3 \) \[ \text{HCO}_3 \] 24 mEq/L

  pH 7.21
Respiratory Acidosis

- Respiratory acidosis compensates by metabolic alkalosis
  - Compensated by the kidney increasing production of bicarbonate

**Acute Hypercapnia:**
HCO₃ increases 1 mmol/L for each 10 mmHg increase in PaCO₂ >40

**Chronic Hypercapnia:**
For each 10 mmHg increase in PaCO₂ >40 HCO₃ incr. 3.5 mmol/L

### Simple Acid-Base Disturbances

<table>
<thead>
<tr>
<th>Acid-Base Disorder</th>
<th>Primary Abnormality</th>
<th>Expected Compensation</th>
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<td>↓pCO₂</td>
<td>pCO₂ = 1.5 × [HCO₃⁻] + 8</td>
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<td>↑↑↑[HCO₃⁻]</td>
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<td>↓↓[HCO₃⁻]</td>
<td>↓ in [HCO₃⁻] = 2 × ΔpCO₂/10</td>
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<td>↓↓↓pCO₂</td>
<td>↓↓[HCO₃⁻]</td>
<td>↓ in [HCO₃⁻] = 5 × ΔpCO₂/10</td>
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</table>
**Acute Respiratory Acidosis:**

25 y.o. IV drug user s/p heroin overdose:

pH 7.10  pCO\(_2\) 80  Bicarbonate 24

80 – 40 = 40. For every 10 CO\(_2\) inc 3.5 mmol HCO\(_3\) increases

10----------- 3.5
40----------- 40/10 = 4 x 3.5 = 14
24 + 14 = 38 HCO\(_3\)

**Chronic Respiratory Acidosis:**

65 y.o. patient with stable COPD:

pH 7.32  pCO\(_2\) 70  Bicarbonate 35

Significant Renal Compensation

But when he arrives in the ED, this is the only ABG you have:

- 7.23/85/pO\(_2\)/35
- 35-24=11. 11/3.5 = 3. 3 x 10 =30. 40 + 30 = 70
  - Baseline  pCO\(_2\) = 70. Pt. has acute resp acidosis.
Respiratory Alkalosis

- Decreasing of CO₂ level due to a hyperventilation, which expels large amounts of CO₂, leads to a lowering in the partial pressure of CO₂ below normal and the shift of the equilibrium from H₂CO₃ to CO₂ and H₂O. This shift decreases H₃O⁺ and raises blood pH – alkalosis.

\[ \text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \downarrow \text{H}_3\text{O}^+ + \text{HCO}_3^- \]

Respiratory Alkalosis: CO₂ ↓ pH ↑

- **Symptoms:** Increased rate and depth of breathing, numbness, light-headedness, tetany
- **Causes:** Hyperventilation due to anxiety, hysteria, fever, exercise; reaction to drugs such as salicylate, quinine, and antihistamines; conditions causing hypoxia (e.g., pneumonia, pulmonary edema, and heart disease)
- **Treatment:** Elimination of anxiety producing state, rebreathing into a paper bag
Acid-Base Imbalances

**Normal**

\[
\begin{align*}
\text{H}_2\text{CO}_3 & \quad \ldots \quad \text{HCO}_3 \\
1 & \quad \triangle \quad 20 \\
24 \text{ mEq/L} & \quad 1.2 \text{ mEq/L}
\end{align*}
\]

**Respiratory Alkalosis**

\[
\begin{align*}
\text{H}_2\text{CO}_3 & \quad \ldots \quad \text{HCO}_3 \\
1 & \quad \triangle \quad 40 \\
0.6 \text{ mEq/L} & \quad 24 \text{ mEq/L}
\end{align*}
\]
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### Acute Hypocapnia:
- HCO₃ decreases 2 mmol/L for every 10 mmHg decrease in PaCO₂ <40

### Chronic Hypocapnia:
- For every 10 mmHg decrease in PaCO₂ <40
- HCO₃ decreases 5 mmol/L
Respiratory Alkalosis:

15 y.o. girl who just had panic attack
pH 7.70 pCO₂ 20 Bicarbonate 24

Reality: 7.65/20/pO₂/20, because hypocapnia leads to lower bicarb as well.

40 – 20 = 20. For every 10 CO₂ HCO₃ dec by 5 mmol
20/10 = 2 x 5 = 10
24 – 10 = 14

3 most important equations so far

- Chronic resp. acidosis: steady-state pCO₂ is increased by 10 for every 3.5 increase in HCO₃
- Acute metabolic acidosis:
  - pCO₂ = 1.5 x HCO₃ + 8 (+/- 2)
- Acute metabolic alkalosis:
  - pCO₂ = 0.9 x HCO₃ + 15
METABOLIC ACIDOSIS

- Metabolic acidosis represents an increase in acid in body fluids.
- Reflected by a decrease in [HCO3 -] and a compensatory decrease in pCO2.
### Metabolic Acidosis

- Impaired cardiac contractility
- Decreased threshold for v fib
- Decreased Hepatic and Renal perfusion
- Increased Pulm Vasc resistance
- Inability to respond to catecholamines
- Vascular collapse
Test Case

23 year old AIDS patient c/o weakness and prolonged severe diarrhea. He appears markedly dehydrated.

pH 7.25  pCO₂ 25  pO₂ 110  HCO₃ 11

151  129  60

2.0  12  2.0

Acute metabolic acidosis:

\[ pCO₂ = 1.5 \times HCO₃ + 8 \pm 2 \]

\[ = 1.5 \times 11 + 8 \]

\[ = 24.5 \]

Metabolic Acidosis

18 y.o. WF presents in DKA

ABG: pH 7.00  pCO₂ 25  Bicarbonate 6

If Pure metabolic acidosis, then \( pCO₂ = (1.5)(6) + 8 = 17 \)

\[ pCO₂ = 1.5 \times HCO₃ + 8 +/− 2 \]

\[ = 1.5 \times 6 + 8 \]

\[ = 9 + 8 \]

\[ = 17 \]
Respiratory Compensation

**Metabolic Acidosis:**
- Occurs rapidly
- Hyperventilation
  - "Kussmaul Respirations"
  - Deep > rapid (high tidal volume)

**Metabolic Alkalosis:**
- Calculation not as accurate
- Hypoventilation
- Restricted by hypoxemia
- PCO₂ seldom > 50-55

\[ pCO_2 = 1.5 \times HCO_3 + 8 \pm 2 \]
Winter’s formula

\[ pCO_2 = 0.9 \times HCO_3 + 15 \]

**METABOLIC ALKALOSIS:**
- Metabolic alkalosis represents an increase in [HCO₃⁻] with a compensatory rise in pCO₂.
Test Case

An 80 year old man has been confused and c/o SOB for one week. He also has a hearing problem and has seen 3 ENT docs in the past month. Family denies medications.

pH 7.53  pCO2 15  pO2 80  HCO3 12

\[
\begin{align*}
\text{AG} & = 140 - 121 = 19
\end{align*}
\]
### First Step - Simple disorders

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<td>&lt; 22</td>
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