ABGs & Electrolyte Disorders

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Objectives

- Review acid / base imbalance
- Review anion gap and non-anion gap metabolic acidosis
- Review electrolyte disorders
  - Na
  - K
  - Ca
  - HCO3
  - BUN
  - Cr
pH

- pH is the inverse log of the Hydrogen ion concentration
- It is a logarithmic function so small changes in the number represent large changes in H concentration
- Normal physiologic pH is 7.35-7.45
- Normal enzymatic function occurs only within a very narrow pH
- Rapid derangement or more gradual but more severe derangement causes a loss of enzymatic function and inability to maintain cellular processes resulting in death
Figure 5: Diagnosis using Serum Acid-Base Values:

Davenport Diagram

Henderson-Hasselbalch:

\[
pH = pK + \log [\text{HCO}_3^-] \times PCO_2
\]

or,

\[
pH = 6.1 + \text{Kidney} \quad \text{Lung}
\]
ABGs

- pH/pO2/pCO2/HCO3/BE/Sat
- HCO3 in ABG is calculated NOT measured
- HCO3 in a Chem 7 is measured
- Base deficit/excess is the amount of bicarbonate that would have to be added to achieve a normal pH
Acid / Base Disorders

- Respiratory
  - Acidosis
  - Alkalosis
- Metabolic
  - Acidosis
    - Anion gap
    - Non-anion gap
  - Alkalosis
- Compensation mechanisms
Compensatory Mechanism

- Intravascular buffers
  - HGB / plasma proteins
  - Phosphate
  - Bicarbonate buffer system (1/2 the buffer effect)
    - \( \text{H}_2\text{O} + \text{CO}_2 = \text{H}_2\text{CO}_3 = \text{H}^+ + \text{HCO}_3^- \)

- Respiratory
  - Respiratory control of CO2 concentration

- Renal compensation
  - retention/excretion of \( \text{HCO}_3^- \)
Fundamentals

- Look at the pH
- Primary respiratory disorder
  - Look at CO2
- Primary metabolic disorder
  - Look at HCO3
- Any compensation that occurs will not exceed the magnitude of the primary disorder
ABG Interpretation

- Key to success is systematic approach
- Remember the principal rule….
  - The body does not compensate past the primary disturbance
- Identify your abnormalities in pH (7.35-7.45), pCO2 (35-45) and HCO3 (22-26)
- Let's look at isolated disorders to start
Example # 1

- 7.5 / pO2 / 29 / 22 / BE / SaO2
- pH high / low / normal?
- CO2 high / low / normal?
- Bicarb high / low / normal?
- What is the diagnosis?

- Acute respiratory alkalosis
Respiratory Alkalosis

- Primary derangement is caused by excessive off-gassing of $\text{CO}_2$
- Long term compensation is $\text{HCO}_3^-$ excretion by the kidney
Respiratory Alkalosis

- Anxiety
- Hypoxia
- Lung ds with or without hypoxia
- Central nervous system ds
- Drugs – salicylates, catecholamines
- Pregnancy
- Sepsis
- Hepatic encephalopathy
- Mechanical ventilation
Example # 2

- 7.25 / pO2 / 60 / 26 / BE / SaO2
- pH high / low / normal?
- CO2 high / low / normal?
- Bicarb high / low / normal?
- What is the diagnosis?

- Acute respiratory acidosis
Respiratory Acidosis

- Primary derangement is caused by accumulation of CO2
- Long term compensation by renal retention $\text{HCO}_3^-$
  - Why COPD pt have CO2 in 50-70s without significant acidosis
Causes of Respiratory Acidosis

- CNS depression
  - Drugs, cns event, cns masses
- Neuromuscular disorders
  - Myopathies, neuropathies
- Acute airway obstruction
  - Upper airway, laryngospasm, bronchospasm
- Impaired lung motion
  - Hemothorax, pneumothorax, flail chest
- Ventilator dysfunction
Example # 3

- 7.34 / pO2 / 60 / 31 / BE / SaO2
- pH high / low / normal?
- CO2 high / low / normal?
- Bicarb high / low / normal?
- What is the diagnosis?

- Chronic respiratory acidosis with metabolic compensation (alkalosis)
Example # 4

- 7.50 / pO2 / 48 / 36 / BE / SaO2
- pH high / low /normal?
- CO2 high / low /normal?
- Bicarb high / low /normal?
- What is the diagnosis?

- Metabolic alkalosis with respiratory compensation (acidosis)
Metabolic Alkalosis

- Caused by loss of endogenous acids or introduction of exogenous base
- Primary compensatory mechanism is CO$_2$ retention (hypoventilation)
- Causes are subdivided by chloride response
Metabolic Alkalosis

- Urinary chloride low
  - Vomiting, nasogastric suctioning
  - Diuretic use
  - Posthypercapnia

- Urinary chloride high/normal
  - Excess mineralocorticoids – Conn/Cushing syndrome, exogenous steroid, licorice
  - Diuretics
  - Excess alkali administration
Example # 5

- 7.20 / pO2 / 21 / 8 / BE / SaO2
- pH high / low /normal?
- CO2 high / low /normal?
- Bicarb high / low /normal?
- What is the diagnosis?

- metabolic acidosis with respiratory compensation (alkalosis)
Metabolic Acidosis

- Causes are divided by anion gap vs non-anion gap metabolic acidosis
- Anion Gap is the normal gap between positive ions and negative ions
Metabolic Acidosis

- May be anion gap or non-anion gap
- Calculate the anion gap
  - Most labs provide this for you
  - Positive ions – negative ions
  - Na – (HCO₃ + Cl)
  - Potassium is excluded because of its very small contribution to extracellular cations
Anion Gap Metabolic Acidosis

**M** - methanol
**U** - uremia
**D** - DKA
**P** – Phenol, paraldehyde
**I** – INH, Iron
**L** - lactate
**E** – ethanol, ethylene glycol
**S** - salicylates
Non-Anion Gap Metabolic Acidosis

- Usually the result of derangement of the bicarbonate system
  - GI Bicarbonate loss
    - Diarrhea
  - Renal Bicarbonate loss
    - RTA
    - Early renal failure
    - Carbonic anhydrase inhibitors
    - Aldosterone inhibitors
- Not usually a concern in the acute setting
- Requires more extensive work-up including urine studies
Non-Anion Gap Metabolic Acidosis

- Hyperchloremic Metabolic Acidosis
  - May be induced by excessive volume resuscitation with saline
  - \( \text{NaCl} + \text{H}_2\text{O} = \text{HCl} + \text{NaOH} \)
    - Strong acid and base should balance?
  - Normal ratio of Na : Cl is 140 : 100
    - Now provide Na and Cl in 1:1 ratio
    - Excessive Cl leads to acidosis
Ok now it gets tricky

- Mixed acid base disorders
- Respiratory acidosis w/(AG or NAG) metabolic acidosis and metabolic alkalosis
- OR  Respiratory alkalosis w/(AG or NAG) metabolic acidosis and metabolic alkalosis
- OR  (AG or NAG) metabolic acidosis and metabolic alkalosis
- Luckily you can’t have 4 disorders at the same time because you can’t hyper and hypo ventilate at the same time!
ABG Interpretation

- **Step 1**
  - Look at pH if <7.4 is acidosis if >7.4 is alkalosis as primary disorder

- **Step 2** : Calculate the anion gap
  - Na – (HCO3 + Cl)
  - If gap is > or = 20 there is a primary metabolic acidosis regardless of pH or bicarbonate

- **Step 3** : Calculate the excess anion gap
  - Calc AG – Norm AG (12)
  - Add this to the measured bicarb
    - If > normal bicarb (>30) have metabolic alkalosis
    - If < normal bicarb (<23) there is a non-AG metabolic acidosis
Example # 6

- 7.50 / pO2 / 20 / 15 / BE / SaO2
- pH high / low / normal?
- CO2 high / low / normal?
- Bicarb high / low / normal?
- What is the diagnosis?

- This looks like respiratory alkalosis...and it is. But there is more to it.
- Look at the bicarb...
Example # 6

- 7.50 / pO2 / 20 / 15 / BE / SaO2
- Na 140; Cl 103; HCO3 15
  - AG = 140-(103+15) = 22
  - Rule # 2 if AG >/= to 20 there is a primary metabolic acidosis regardless of pH or HCO3
- Now rule #3
  - Excess gap = 22-12 =10 Add to bicarb
    - 10+15=25
- >30 or <23? No
Example #6

- Where does this stuff come from?
- This patient had a centrally mediated respiratory alkalosis (salicylates directly stimulate the medulla) but....
- They also ingested a large quantity of ASA and had a primary metabolic acidosis as well.
- No additional disorder is present.
Example # 7

- 7.40 / pO2 / 40 / 24 / BE / SaO2
- pH high / low / normal?
- CO2 high / low / normal?
- Bicarb high / low / normal?
- What is the diagnosis?

- Nothing wrong here....right?
- Calculate the anion gap
Example # 7

- 7.40 / pO2 / 40 / 24 / BE / SaO2
- Na 145; Cl 100; HCO3 24
  - AG = 145-(100+24) = 21
  - Rule # 2 if AG >/= to 20 there is a primary metabolic acidosis regardless of pH or HCO3
- Now rule #3
  - Excess gap = 21-12 =9 Add to bicarb
    - 9+24=33
  - >30 or <23? Yes!
  - There is a metabolic alkalosis as well!
Example # 7

- This patient had chronic renal failure and developed uremia... And a metabolic acidosis.

- As his uremia worsened he developed vomiting...and a metabolic alkalosis.
Example # 8

- 7.50 / pO2 / 20 / 15 / BE / SaO2
- pH high / low /normal?
- CO2 high / low /normal?
- Bicarb high / low /normal?
- What is the diagnosis?

- There is an alkalosis but the bicarb is low and the CO2 is low....this ain’t right?
- Calculate the anion gap
Example # 8

- 7.50 / pO2 / 20 / 15 / BE / SaO2
- Na 145; Cl 100; HCO3 15
  - AG = 145-(100+15) = 30
  - Rule # 2 if AG >/= to 20 there is a primary metabolic acidosis regardless of pH or HCO3
- Now rule #3
  - Excess gap = 30-12 =18 Add to bicarb
    - 18+15=33
- >30 or <23? Yes!
- There is a primary metabolic alkalosis but there is also a metabolic acidosis (rule #2) and a respiratory alkalosis as well!
OK how does that happen?

- This patient had pneumonia which resulted in the respiratory alkalosis.
- They also vomiting which caused the metabolic alkalosis and...
- They had alcoholic ketoacidosis which caused the metabolic acidosis.
Example # 9

- 7.10 / pO2 / 50 / 15 / BE / SaO2
- pH high / low /normal?
- CO2 high / low /normal?
- Bicarb high / low /normal?
- What is the diagnosis?

- There is an acidosis with both a high CO2 and a low bicarb.
- Calculate the anion gap
Example # 9

- 7.10 / pO2 / 50 / 15 / BE / SaO2
- Na 145; Cl 100; HCO3 15
  - AG = 145-(100+15) = 30
  - Rule # 2 if AG >/= to 20 there is a primary metabolic acidosis regardless of pH or HCO3

Now rule #3

- Excess gap = 30-12 =18 Add to bicarb
  - 18+15=33

- >30 or <23? Yes!
- There is a primary respiratory acidosis but there is also a metabolic acidosis (rule #2) and a metabolic alkalosis as well!
OK how does that happen?

- This patient was obtunded and hypoventilating hence the respiratory acidosis; had vomiting leading to the metabolic alkalosis and had diabetic ketoacidosis which caused their anion gap metabolic acidosis.
Example # 10

- 7.15 / pO2 / 15 / 5 / BE / SaO2
- pH high / low /normal?
- CO2 high / low /normal?
- Bicarb high / low /normal?
- What is the diagnosis?

Clearly there is a metabolic acidosis and a respiratory alkalosis. By now you know there must be something else...
Example # 10

- 7.15 / pO2 / 15 / 5 / BE / SaO2
- Na 145; Cl 100; HCO3 15
  - AG = 140-(110+5) = 25
  - Rule # 2 if AG >/= to 20 there is a primary metabolic acidosis regardless of pH or HCO3
- Now rule #3
  - Excess gap = 25-12 =13 Add to bicarb
    - 13+5=18
- >30 or <23? Yes!
- This looks like a AG metabolic acidosis with respiratory compensation. However the change in bicarb does not correspond to the AG acidosis alone. There is a NAG acidosis as well!
OK how does that happen?

- This patient had DKA with a respiratory alkalosis. However the patient also had a NAG acidosis caused by hypercholoremic metabolic acidosis.
Had enough?
Osmosis

- Concentrations across a semipermeable membrane want to become equal.
- If solute cannot pass through the membrane, water will move into or out of the cell to make the concentrations equal.
- Osmotic pressure describes the driving force of this mechanism.
RBCs in solution

Hypotonic

Hypertonic
Sodium Regulation (135-145)

Sodium is regulated by:
- Renin-Angiotensin system
- Renin triggers secretion of Aldosterone
- Aldosterone
  - Increases sodium reabsorption
  - Increases potassium excretion
Water regulation

- Water homeostasis is maintained by the hypothalamus
- Senses rise in osmolarity and triggers
  - Thirst
  - Release of antidiuretic hormone
    - ADH increases water reabsorption in the distal tubule
- Usually Na and H2O changes are proportionate and do not cause changes in sodium concentration
Disorders of Na and H2O

- Sodium disorders are the result of an abnormal ratio of sodium to water.
- Whether hypo or hyper natremic they may exist in states of:
  - Hypervolemia
  - Euvolemia
  - Hypovolemia
Renin-angiotensin-aldosterone system

Decrease in renal perfusion (juxtaglomerular apparatus) → Renin → Angiotensinogen → Angiotensin I → Angiotensin II → Aldosterone secretion

- Surface of pulmonary and renal endothelium: ACE
- Tubular Na⁺ Cl⁻ reabsorption and K⁺ excretion, H₂O retention
- Water and salt retention. Effective circulating volume increases. Perfusion of the juxtaglomerular apparatus increases.

Legend:
- Secretion from an organ
- Stimulatory signal
- Inhibitory signal
- Reaction
- Active transport
- Passive transport
Hyponatremia

- Hyponatremia is Na <130
- Is the most common electrolyte abnormality
- Most patients are stable
- Profound hyponatremia or rapid change can cause cerebral edema and increased ICP due to influx of water into the cell.
Factitious Hyponatremia

- Caused by how sodium is measured
- Pseudohyponatremia (normal osmolarity)
  - Hyperlipidemia or hyperproteinemia
- Redistributive hyponatremia (osm >295)
  - Osmotic particles in ECF cause fluid shift from cells in to ECF giving appearance of low Na
    - Hyperglycemia (na falls 1.5 for every 100 glu)
    - Mannitol administration
True hyponatremia (Osm < 280)

- May occur with hypovolemic, hypervolemic or euvoolemic states depending on extracellular volume.
- These are differentiated by urine sodium excretion studies.
- We don’t collect 24 hr urine studies so.....don’t sweat the details.
Hypovolemic

Addisons is mineralcorticoid deficiency

Hypervolemic

Euvolemic

Hyponatraemia

Is the patient dehydrated?

Yes

Is urinary Na > 20 mmol/L?

Yes

Renal Na⁺ loss
- Addison’s
- Renal failure
- Diuretic excess
- Osmolar diuresis

No

Loss elsewhere
- Diarrhoea
- Vomiting
- Fistulae
- Burns
- Small-bowel obstruction
- Trauma
- CF
- Heat exposure

No

Is the patient oedematous?

Yes

Urine osmolality > 500 mmol/kg?

Yes

Inappropriate ADH

No

Nephrotic synd.
Cardiac failure
Cirrhosis
Renal failure

No

Water overload
Severe hypothyroidism
Glucocorticoid insufficiency
Signs and Symptoms

- Anorexia
- Nausea and vomiting
- Difficulty concentrating
- Confusion
- Lethargy
- Agitation
- Headache
- Seizures

- Variable degrees of cognitive impairment (eg, difficulty with short-term recall; loss of orientation to person, place, or time; frank confusion or depression)
- Acute severe hyponatremia: coma; fixed, unilateral, dilated pupil; decorticate or decerebrate posturing; and respiratory arrest
- Volume depletion: Dry mucous membranes, tachycardia, diminished skin turgor, orthostatics
- Volume overload: Pulmonary rales, S3 gallop, jugular venous distention, peripheral edema, or ascites
Treatment of Hyponatremia

- Treatment of hyponatremia is dependent on the symptoms
- Overriding theme is SLOW correction
- More aggressive therapy if na < 120 and significant change in mental status or coma, or seizures. ("hot salts")
- Rapid correction can cause central pontine myelinolysis (destruction of cells of pons)
Signs and Symptoms of CPM

- Dysarthria
- Dysphagia
- Seizures
- Altered mental status
- Quadriparesis
- Hypotension
- Typically begin 1-3 days after correction of serum sodium level.
Treatment of Hyponatremia

- Should not be corrected faster than 0.5meq/Hr to target > 120
- Na to be infused =TBW x (desired – actual)/ na concentration of solution
- Example: 50kg pt with Na 105
  \[(.6\times50\text{kg})(120-105)/513\text{mEq/L}(\text{if 3\% saline})\]
  \[=0.877\text{L} \text{ or } 877\text{mL}\]
  \[15\text{meq}/0.5\text{meq/hr} = 30 \text{ hrs or 29 mL/hr}\]
Treatment of Hyponatremia

- If NS is used contains 154meq Na
- Na to be infused = TBW x (desired – actual)/ na concentration of solution
- Example: 50kg pt with Na 105
  - (.6x50kg)(120-105)/154mEq/L
  - =2.9L or 2900 mL
  - 15meq/0.5meq/hr = 30 hrs
  - or 97 mL/hr
Hypernatremia

- Na > 145
- Is rare in those able to control own fluid intake (seen in infants and elderly)
- Because pt is usually debilitated these have worst prognosis of electrolyte d/o.
- Causes cerebral cellular degeneration causes brain shrinkage and puts mechanical traction on vessels.
Signs and Symptoms

- Anorexia
- Restlessness
- Nausea and vomiting
- Altered mental status
- Lethargy or irritability
- Stupor or coma.
- Twitching, hyperreflexia, ataxia, tremor, seizure
- Focal deficits such as hemiparesis have been reported.
Causes of Hypernatremia

- Poor PO intake
- Excess excretion
  - Diuretics
  - Lithium
  - Diabetes insipidus
- Intake of hypertonic solution
- Mineral/glucocorticoid excess
  - Conn or Cushings Syndrome
  - (hyperaldosteronism hypercortisolism)
Treatment of Hypernatremia

- Treatment caveats are the same for hyponatremia
- Correct imbalance slowly (<1meq/hr) this is often complicated as the patient is frequently volume depleted and may require rapid volume resuscitation for hypovolemic shock.
Free Water Deficit =
(kg) X 0.6 (TBW) X ([Serum Na/140] - 1)
Example 60 kg male with na of 155
= 60 x 0.6 x [(155/140)-1] = 3.9L
Must add to maintenance fluids and correct over 48hrs
Priority is volume replacement. Once volume replacement is achieved patients may be switched to free water.
Potassium (3.5-5)

- Is the major intracellular ion
- Only 2% of potassium is extracellular
- The large ratio of intracellular to extracellular K is responsible for the membrane charge in excitable tissue
- 90% of daily potassium load is secreted by kidneys
- Aldosterone increases K secretion by the kidney (retains sodium and indirectly water)
Hypokalemia

- 2 most common causes are malnutrition due to alcohol abuse and diuretics
- Also affected by acid base status with alkalosis causing movement into cells and acidosis out of cells
Hypokalemia Treatment

- K 3.0 - 3.5meq/L is rarely symptomatic
- Underlying cardiovascular disease and digitalis toxicity predispose patients to more severe dysrhythmias and should be more aggressively corrected.
- Each 0.3meq deficit represents 100meq of total body deficit.
- Max IV replacement should be 0.3-1meq/kg/hr
Hyperkalemia

- Is most deadly of electrolyte disorders
- Lab error is the #1 cause of hyperkalemia
- May be divided into external and internal balance disorders
Potassium

- Excess exogenous K
  - Dietary, supplement, iatrogenic
- Acute/chronic renal failure
- Aldosterone deficiency
- Drugs represent 1/3 cases
  - 75% due to KCl supplement or potassium sparing diuretic
- Acute acidosis
- Acute cell lysis
  - Rhabdo, hemolysis, burns
Hyperkalemia

- Neuromuscular
  - Weakness, paresthesias, tetany
- EKG changes
  - Peak T (5.5-6)
  - PR prolongation/loss of p (6.0-6.5)
  - Gradual widening of QRS
  - AV nodal and BB blocks
Hyperkalemia

Management

- Temporizing measures
  - CaCl
  - HCO3
  - Albuterol
  - Insulin

- Increased excretion
  - Furosemide
  - Kayexylate
BUN

- Blood urea nitrogen
- Urea is formed from protein catabolism and is produced by the liver
  - Ingested proteins
  - Body proteins (including blood in GI tract)
- Elevated nitrogen levels is called azotemia
Creatinine is a breakdown product of creatinine phosphate from muscle.

- Breakdown and renal filtration are usually at a fairly fixed rate.
- Creatinine is not reabsorbed by the kidney.
- Creatinine is used as a proxy for evaluating renal function.
Low Cr

- Low levels of Cr can be seen in
  - Malnutrition due to decreased mm mass
  - Late stage muscular dystrophy
  - Myasthenia gravis

- Not common
- Not useful
Elevated Cr

- Elevated Cr levels may be seen in renal failure
  - pre renal
    - Failure to perfuse the kidney
  - Renal
    - Injury to kidney, renal tubules
  - Post renal
    - Failure to excrete urine
    - Causes back pressure in system
    - Stops glomerular filtration
- **Pre-renal**
  - Failure to perfuse the kidney adequately
    - Shock states
    - Dehydration
    - ACEI / diuretic use
Renal

- Infection (sepsis, pyelonephritis)
- Toxins/drugs (NSAIDS, aminoglycosides, iodinated contrast, lithium)
- Rhabdomyolysis
- Hemolysis (hemoglobin damages the tubules)
- Nephropathies (nephrotic & nephritic syndromes)
- Post renal
  - Atonic bladder (dm, drugs, spinal cord injury)
  - Bladder outlet obstruction
  - BPH
  - Catheter obstruction
  - Kidney stones (not really)
How do I know?

- **BUN : Cr ratio**
  - Normal ratio is roughly 10:1
  - Elevated ratio indicates pre-renal failure
  - Ratios greater than 20-30 :1 suggest pre-renal failure
  - Caveat: ratios greater than 40:1 may be suggestive of Upper GI BLEED...clinical correlation recommended

- The best measure of renal function is urinary output...should be 0.5-1ml/kg/hr
Calcium homeostasis is regulated by parathyroid hormone and calcitonin.

- PTH causes release from body stores, increases reabsorption from the kidneys and raises blood calcium.
- Vitamin D increases absorption from the GI tract.
- Calcitonin raises calcium excretion from the kidney, increases trapping in the bones and lowers serum levels.
Role of Calcium

- Bone formation and maintenance
- Muscle contraction
- All processes that involve exocytosis including synaptic transmission and hormone release.
- 40% of calcium is bound to albumin
Hypercalcemia

- Stones
  - Polyuria
  - Nocturia
  - Dehydration
  - Renal stones
  - Renal failure

- Bones
  - Osteopenia/Ca deposits

- Abdominal moans
  - Constipation
  - Nausea
  - Anorexia
  - Pancreatitis
  - Gastric ulcer

- Psychiatric groans
  - Lethargy
  - Weakness
  - Confusion
  - Coma
  - Hypotonia
  - Hyporeflexia
  - Paresis

- Cardiac effects
  - Syncope
  - Arrhythmia
  - Hypotension
  - Shortened QT
90% of hypercalcemia is caused by malignancy or hyperparathyroidism.

Cancer
- 80% causes by bony metastasis
- 20% caused by PTH produced by cancer cells
Emergent Treatment

- Acute management is directed at elimination of excess calcium
  - Volume replacement
  - Furosemide
Hypocalcemia

- Numbness and tingling in the perioral area, fingers, toes
- Muscle cramps may progress to carpopedal spasm and tetany
  - Chvostek sign – tapping the facial nerve causes facial mm spasm
  - Trousseau sign - carpal spasm after 3 minutes of inflation of a pressure cuff 20 mm Hg above the patient's systolic pressure
- Irritability, impaired intellectual capacity, depression, and personality changes
- Parkinsonism, Choreoathetosis
- Grand mal, petit mal, focal seizures
- Laryngospasm and bronchospasm
- Signs of mental retardation may be present in children, adults may have dementia.
- Prolongation of the QTc interval, arrhythmias, hypotension, and heart failure.
Treatment of Hypocalcemia

- Calcium
Adrenal Insufficiency

- Is the loss of gluco and mineralocorticoid production from the adrenals
- Primary adrenal insufficiency is related to failure of the organ (50 per 1,000,000)
- Secondary adrenal insufficiency is related to disorders of the hypothalamic pituitary system. Commonly exogenous steroid.
Adrenal Crisis

- Results from acute exacerbation of chronic adrenal insufficiency
- Usually caused by some stressor
  - Surgery
  - Anesthesia (e.g., etomidate)
  - Volume loss
  - Trauma
  - Asthma
  - Hypothermia
  - Alcohol
  - Myocardial infarction
  - Fever / infection / sepsis
  - Hypoglycemia
  - Pain
  - Psychoses or depression
  - Exogenous steroid withdrawal
Signs and symptoms

- Weakness (99%)
- Pigmentation of skin (98%)
- Weight loss (97%)
- Abdominal pain (34%)
- Salt craving (22%)
- Diarrhea (20%)
- Constipation (19%)
- Syncope (16%)

Lab Abnormalities
- Hypoglycemia (67%)
- Hyponatremia (88%)
- Hyperkalemia (64%)
- Hypercalcemia (6-33%)

Findings are non-specific and usually symptoms relate to illness that precipitated event

Look for shock unresponsive to fluids or pressors
Treatment

- Volume replacement
- Correct lab abnormalities as needed
- Administer
  - Hydrocortisone 100mg IV or
  - Dexamethasone 4 mg IV
- Treat the underlying disorder