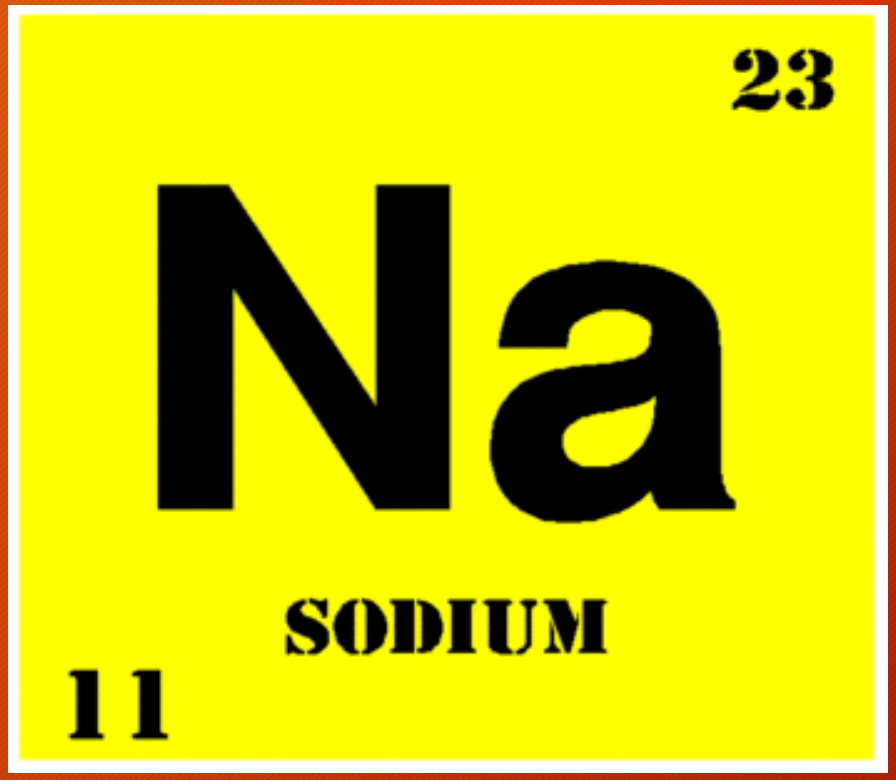
Diagnosis and Treatment of Electrolyte Disturbances.

Jessica A. Cannon MSN, ANP- BC

Objectives

- Identify the most commonly seen electrolyte disturbances in the adult population.
- Review function and physiology of common electrolytes in the body.
- Discuss the differential and work up of electrolyte disturbances commonly found.
- Discuss treatment of common abnormal findings.
- Identify need for further work up and referral to specialists for further treatment and work up of electrolyte disturbances.



Sodium

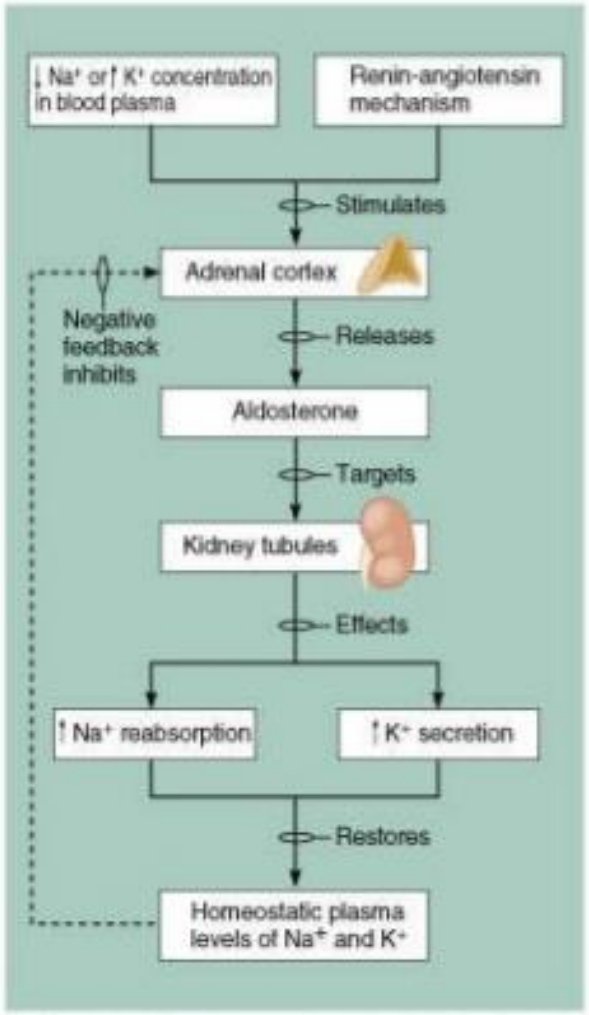
Overview of Sodium and its Role in the Body

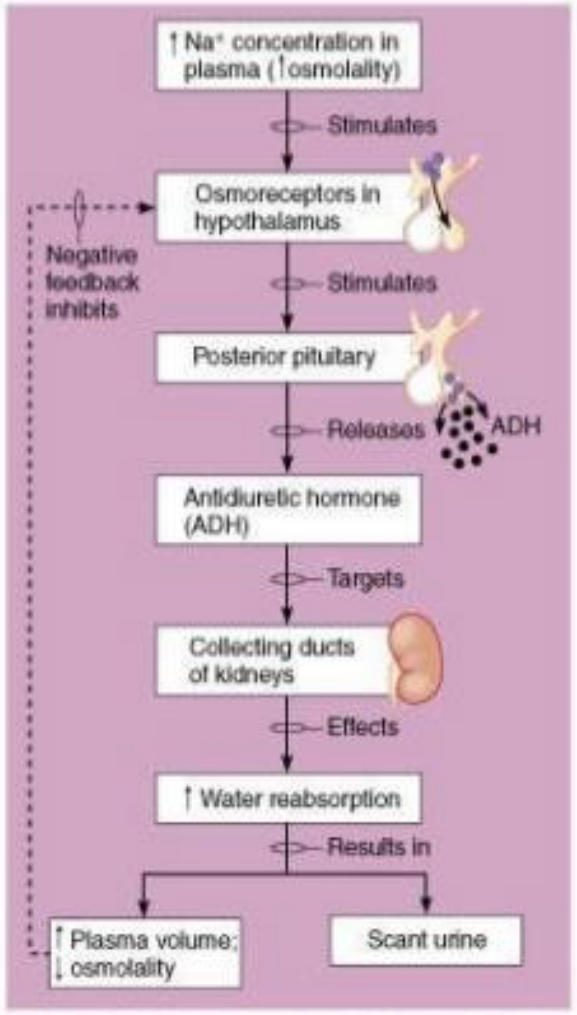
- Mostly located in fluid around the cells and in blood.
- Role: assists in maintaining normal fluid balance and assisting in muscle and nerve function.
- Sodium obtained through PO intake and lost via sweat and urine.
- Healthy kidneys assist in maintaining normal sodium levels by adjusting proportionally the amount of sodium secreted via urine.
- When intake and losses are not proportional , measured sodium levels are abnormal.

Overview (Continued)

- When sodium concentration and/or blood volume are too high, sensors in the heart, kidneys and vessels stimulate the kidneys to increase urine sodium excretion.
- If sodium concentration and/or blood volume are too low- sensors trigger two mechanisms to increase volume as follows:
    - Kidneys trigger the adrenals to release aldosterone which then causes renal sodium retention and increased excretion of potassium leading to decreased urine output and subsequent increased blood volume.
    - Pituitary gland secrete ADH (Vasopressin) which triggers the kidneys to conserve water.

Sodium regulation





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Hyponatremia

- Defined as serum sodium levels of less than 136mEq/L.
- Representative of increased body water in comparison to total body sodium concentration.

Causes of Hyponatremia

- **HYPOVOLEMIC (volume depletion):** (decreased total body water (TBW) AND Sodium levels). Proportionally, more sodium than water has been lost.
- Commonly from GI losses such as Vomiting/Diarrhea.
- Sources of renal loss are most commonly due to diuretic use (thiazides- increase Na+ excretion) and less likely mineralcorticoid deficiency, & nephropathies such as Interstitial nephritis, Polycystic Kidney Disease , etc. ***Urine Na+ will be very high > 20
- Less commonly- consider burns (obvious), rhabdomyolysis , SBO, etc. (3rd space losses)

Causes (continued)

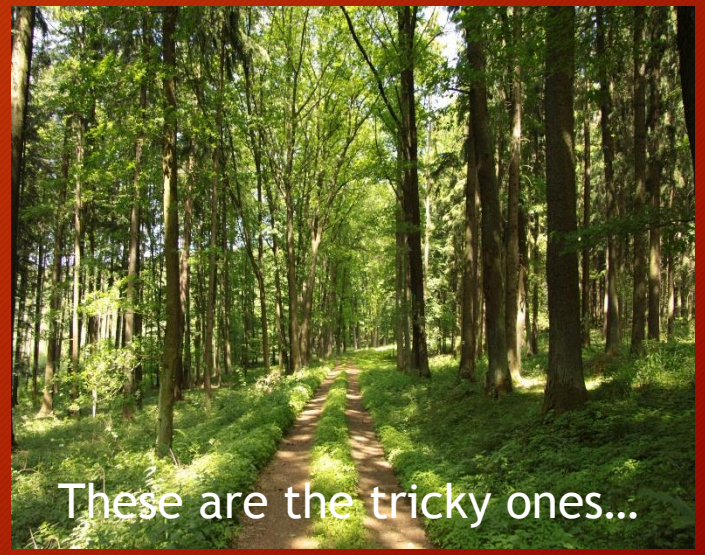
- **EUVOLEMIC(dilutional):** (Increased TBW & near normal sodium) Urine Sodium >20
- Commonly related to medications such as thiazides, carbamazepine, opioids (and others). ***Ecstasy is also a culprit***.
- In addition, also consider disorders such as Addisons, SIADH and Hypothyroidism.
- More common that realized—consider psychogenic polydipsia/crash diets, ect. (Water intake is more than kidneys can excrete)
- Also possibly RT increased stressors such as recent surgery (hypotonic fluids) , intractable pain, etc as these may increase release of ADH.

Causes (continued)

- **HYPERVOLEMIC:** (Increased sodium (total body) & TBW with greater increase in TBW)
- Common non renal causes include heart failure, liver disease (edematous diseases)
- Renal causes include acute renal failure (AKI), Chronic Kidney Disease (CKD) and nephrotic syndrome (rare).

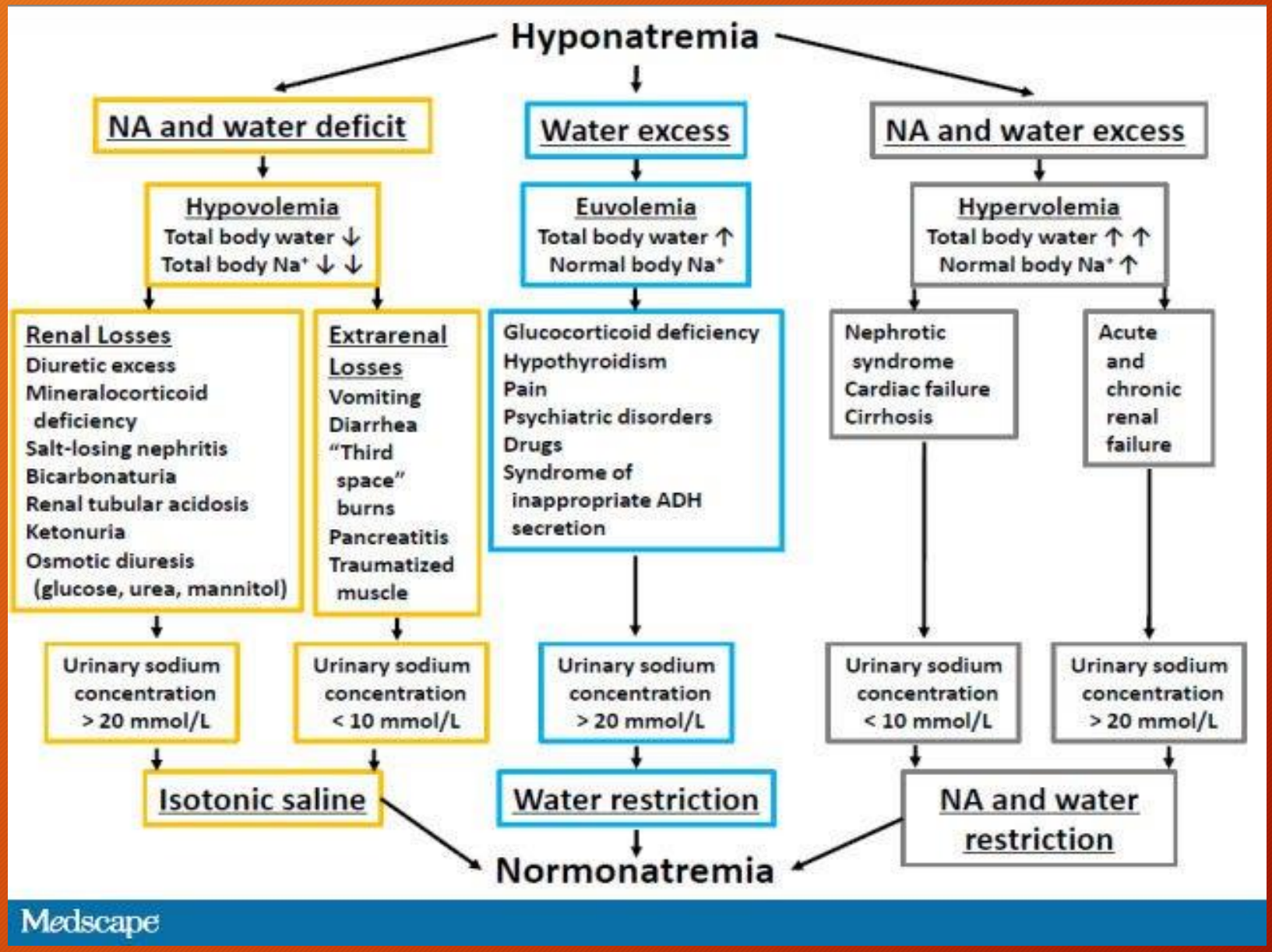
So….

Ask yourself, Does my patient look too dry (hypovolemic), too wet (hypervolemic) or just right (euvolemic)????



Recap…

SO…
If Hyponatremia noted/Suspected…assess volume status AND check Serum and Urine lytes and Osmolality…..If Euvolemic – add thyroid and adrenal function testing- consider specialist referral..


Hyponatremia
NA and water deficit
Water excess
NA and water excess
Hypovolemia
Total body water ↓
Total body Na+ ↓ ↓
Euvolemia
Total body water ↑
Normal body Na+
Hypervolemia
Total body water ↑ ↑
Normal body Na+ ↑
Renal Losses
Diuretic excess
Mineralocorticoid deficiency
Salt-losing nephritis
Bicarbonaturia
Renal tubular acidosis
Ketonuria
Osmotic diuresis (glucose, urea, mannitol)
Extrarenal Losses
Vomiting
Diarrhea
"Third space" burns
Pancreatitis
Traumatized muscle
Glucocorticoid deficiency
Hypothyroidism
Pain
Psychiatric disorders
Drugs
Syndrome of inappropriate ADH secretion
Nephrotic syndrome
Cardiac failure
Cirrhosis
Acute and chronic renal failure
Urinary sodium concentration > 20 mmol/L
Urinary sodium concentration < 10 mmol/L
Urinary sodium concentration > 20 mmol/L
Urinary sodium concentration < 10 mmol/L
Urinary sodium concentration > 20 mmol/L
Isotonic saline
Water restriction
NA and water restriction
Normonatremia
Medscape

Signs and Symptoms

- Primarily CNS in nature but may see s/s of volume depletion or overload as appropriate. Severity of s/s is proportionate to the rapidity of onset of hyponatremia. Rapid= more severe symptoms.
- Older, frail chronically ill generally exhibit more CNS symptoms as compared to younger counterparts
- Nausea and malaise may be subtle early findings (125-130mEq/L).
- May include subtle mental status changes such as lethargy, confusion, personality changes (115-120mEq/L. If hyponatremia is severe (serum sodium < 115- may see stupor, coma, seizures, death.
- Rarely- may see severe cerebral edema- with acute hyponatremia - leading to possible demyelination syndrome and brain stem herniation.

Remember….

- Sodium may be “falsely” elevated when patient is severely hypoglycemic. This is due to increased osmolality from water moving out of cells leading to decreased serum sodium. Thus, always calculate corrected sodium in uncontrolled diabetics if you note a low sodium on labs. “Translocational Hyponatremia”

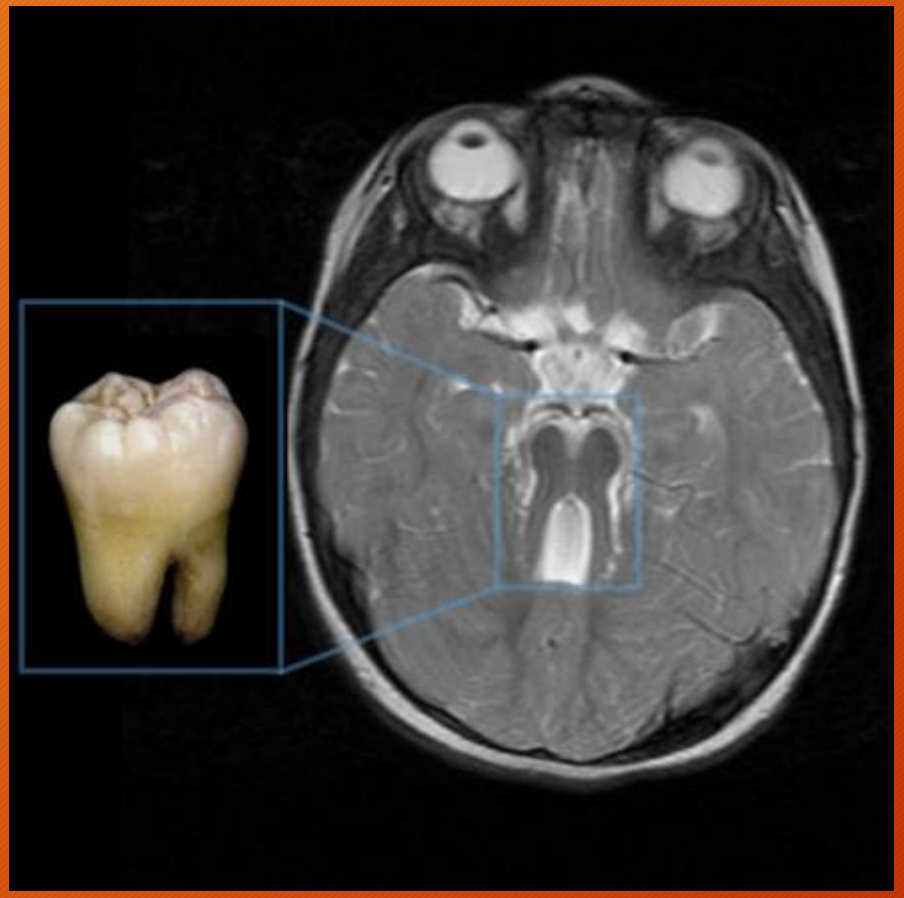
Treatment

- If hypovolemic- 0.9% Normal Saline
- Too rapid correction = risk for demylenation syndrome—never increase more than 8 mEq/L over first 24 hours- thus if significantly hyponatremic and symptomatic- hospitalization warranted for close monitoring and serial sodium levels.
- If hypervolemic- consider fluid restriction with treatment of underlying cause ie)heart failure, liver disease, etc. For example- CHF- consider ACE and loop diuretic. Monitor other lytes (K+ and Mg+_ and replace as indicated. Refractory hyponatremia often indicative of end stage disease.
- If euvolemic- treatment targeted at the cause- For example SIADH- severe fluid restriction,

If Hyponatremia is mild to moderate (between 121 and 135) GO SLOW- minor adjustments are generally all you need. Consider eliminating diuretic, adding salt tabs, etc

If severe and asymptomatic- consider if patient care reliably fluid restrict and monitor closely. If patient has neurologic symptoms- hospitalize for slow IV correction and close monitoring (often ICU- with critical care/nephrology support) . 0.3% normal saline can be used but requires close monitoring (frequent lab draws every 2-3 hours) . In severe , refractory cases- conivaptan (IV)and tolvaptan (PO) may be used- both are selective vasopressin receptor antagonists. Risk of rapid correction and used with caution thus reserved for severe, resistant hyponatremia. Not to be used in hypovolemic patients or those with severe liver disease or advanced CKD.

Osmotic Demyelination Syndrome



If you see it once , you'll never forget it

- Generally affects the pons- but can affects other areas of the brain
- Lesions generally seen in patient with malnutrition, chronic ETOH use, chronic illness
- Patients may develop flaccid paralysis, dysarthria, dysphagia
- Can occur over a few days or even weeks afterwards.
- Severe cases lead to "locked-in" state- general motor paralysis leading to inability to speak- will maintain vertical eye movements. Damage often PERMANENT.

https://radiopaedia.org/articles/osmotic-demyelination-syndrome " Trident Sign"

Hypernatremia

- Defined as serum sodium levels of greater than 145 mEq/L
- Representative of TBW deficit in relation to total body sodium.

Causes of Hypernatremia

- Again, must be considered in relation to fluid status: is the patient too wet ? Too dry" or looks just right??
- Generally related to impaired thirst mechanisim or lack of access to water.
- HYPOVOLEMIC: (Decreased TBW and Sodium but TBW loss is greater)- often due to GI losses, excessive sweating, burns, renal losses (same as for hypovolemic hyponatremia- just dependent on the amounts of Na+ and water lost in comparison to amount ingested prior to presentation.
- Renal losses also common- usually due to loop diuretics 2/2 mechanism of inhibited sodium reabsorption in the kidney leading to increased H2O clearance.
- Also – think about DKA/HHNK- hyperglycemia leading to increased osmotic diuresis-** high sugar ->higher urine output due to decreased ability to maximally concentrate urine=volume depletion.

Causes (Continued)

- EUVOLEMIC:decreased TBW with near to normal total body sodium leading to a pure water deficit.- Hypernatremia can occur BEFORE significant hypovolemia. (ie) extreme exercise, runners, etc. Diabetes Insipidus is also an example –
- HYPERVOLEMIC: much more rare than the other two. Results from grossly elevated sodium intake with limited or poor water intake.- more commonly likely due to excessive hypertonic fluids, etc.

- ESSENTIAL HYPERNATREMIA: may be seen in chronically ill elderly or children with CP, brain damage, etc. Due to impaired thirst mechanism.- Additionally, consider their dependence on others to obtain fluids, etc.

Signs and Symptoms

- THIRST- inability to sense , inability to communicate.
- CNS symptoms due to brain cell shrinkage- confusion, hyperreflexia, seizures or coma
- Symptoms less severe in chronic hypernatremia.

Treatment

- Replacement of free water (PO fluids) is generally all that is needed in awake , alert patients that do not have GI s/s (n/v/severe diarrhea).
- Replacement of intravascular fluids via IV rehydration may be warranted in those with intractable vomiting, altered mental status, etc. Rate of replacement dependent on how acute or chronic the hypernatremia is at time of presentation. Monitor other electrolytes and replace accordingly.
- Treatment of more serious conditions such as diabetes insipidus require specialty involvement.

Potassium

19

K

39.10

Overview of Potassium and its Role

- Most intracellular potassium is contained within muscle cells, thus total body potassium is proportional to lean muscle mass. Potassium plays a large role in determining intracellular osmolality.
- Role- influences cell membrane polarization which then influences cell processes such as muscle contraction and conduction of nerve impulses.
- Small fluctuations can lead to significant clinical symptoms.
- Potassium obtained through oral intake/ IV solution and excreted mostly via the urinary system.
- Serum potassium levels closely correlate with total body potassium content.
- ***A decrease in serum K+ of approximately 1 mEq/L = deficit of about 200-400 mEq.

• Overview (continued)

- Several variables effect intracellular potassium levels including b- adrenergic activity, acid-base levels and insulin concentrations.
    - Insulin pushes K+ into cells(mainly skeletal and hepatic cells)- thus higher levels of insulin= lower K+ (serum). Conversely, low insulin (think DKA)= high serum K+ due to pushing potassium out of the cells.
    - B-adrenergic AGONISTS push K+ into cells and b- blockers/blockade and alpha agonists help move K+ OUT of cells.
    - . Metabolic Acidosis pushes K+ out of cells, alkalosis moves it into cells. But- serum bicarb levels play a large role in this as well- more so than pH. For example- non anion gap acidosis may lead to hyperkalemia while gap acidosis may not. Therefore, the hyperkalemia of DKA (typically a GAP ACIDOSIS) has more to do with insulin deficiency than it does the acidosis
    - Respiratory acidosis/alkalosis will have less effect on serum K+ than a metabolic source.

Overview (continued)

- Dietary potassium intake is approximately 40-150 mEq/day. Generally GI losses are about 10% of dietary intake. Remainder is excreted via urinary system. Therefore, renal secretion greatly affects serum potassium balance.
- If potassium intake is persistently elevated, aldosterone kicks in and renal excretion rises and fecal excretion may decrease.
- If potassium intake falls, intracellular K+ acts as a buffer but renal conservation is a slow response thus hypokalemia is common in the clinical setting.

Hypokalemia

- Defined as serum potassium levels of < 3.5 mEq/L.
- Related to decrease in total body K+ or abnormal movement of potassium into the cells.
- Common problem in the clinical setting.

Causes of Hypokalemia

- Most commonly due to excessive renal or GI tract losses
- Examples of GI loss include diarrhea (usually profound or chronic). Sub considerations include laxative abuse, malabsorption syndromes such as Celiac, and due to bowel diversion surgeries (resection, bypass, etc
- Pica (clay ingestion) can bind potassium and decrease absorption.
- Villous adenoma of colon can lead to profound potassium secretion. RARE
- Protracted vomiting can cause renal losses as well. (Stimulates ADH due to volume depletion leading to excretion of K+).

Causes (continued)

- Disorders such as Cushings, Hyperaldosteronism, Renin secreting tumors (rare)and adrenal hyperplasia (adrenal steroid excess). Salt wasting nephropathies.
- Intracellular Shift:
    - Hyperalimentation/TPN- leading to stimulated insulin release (Glycogenesis)
    - After Administration of Insulin.
    - Administration of b2 agonists (albuterol, terbutaline, dobutamine)- this stimulates the sympathetic nervous system. Also think about ETOH withdrawals, Acute MI, etc- anything that prompts stress induced ephedrine release.
    - Thyrotoxicosis.

Causes (Continued)

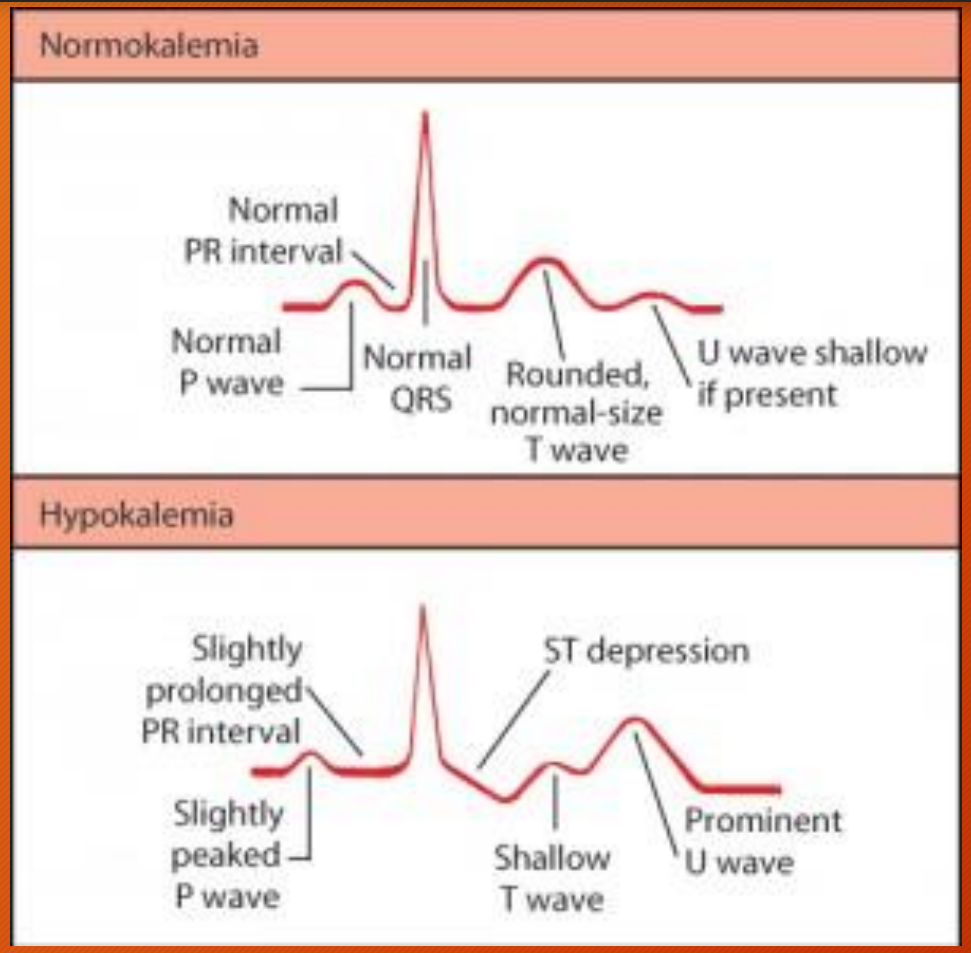
- Drugs- most commonly diuretics- especially the potassium wasting ones (loop, **thiazides**, osmotic)- mechanism is blockage of sodium reabsorption proximal to distal nephron leading to renal loss. Often dose dependent and self limiting.
- Drugs- laxatives- most often if abused-** Consider in patients preoccupied with weight loss.
- GI preps such as sodium phosphate, polyethylene glycol (PEG) preparations—not as commonly used now.
- Other culprits include high dose PCN, theophylline.
- Ingestion of pseudoephedrine, ephedrine such at found in OTC cough/cold meds and diet pills.
- Rare complication of some antipsychotics such as risperidone and quetiapine.

Causes (Continued)

- Increased sweat losses- usually loss is negligible but the body can produce 10L or more of sweat per day in very hot climates, exercise. Also a consideration in this with Cystic Fibrosis.
- Dialysis (peritoneal, hemodialysis)

Signs and Symptoms of Hypokalemia

- Mild- (K= 3.0 to 3.5mEq/L) – rarely cause symptoms.
- Moderate to Severe- (<3.0 mEq/L)- may experience muscle cramping and weakness, hypoventilation, hypotension, tetany and rhabdomyolysis. Persistent decreased potassium levels can cause polyuria and polydipsia due to impaired renal concentrating ability.
- Diagnostics include serum K+ and EKG. Consider 24 hour urine collection if cause is not clearly evident. K+ excretion of <15mEq/L is indicative of GI (extra renal)potassium loss while excretion of > 15mEq/L suggests renal cause.



Cardiac effects not usually noted until K+ < 3mEq/L. May see depression or shallowing of I wave, more prominent U wave and prolonged PR interval

May also result in more frequent PVCS, PACS, 2nd, 3rd degree heart block in more severe hypokalemia. Increasingly severe decline in levels ultimately may lead to ventricular fibrillation.

Patients with underlying CAD, on Digoxin may be more at risk of cardiac SE of even mild hypokalemia.

Treatment/Prevention

- Generally in the outpatient setting- hypokalemia can be treated with oral potassium supplementation.
- Consider admission for IV potassium replacement in severe hypokalemia and /or ongoing losses.
- Liquid KCL improves levels within 1-2 hours but is poorly tolerated due to taste.
- When considering replacement dosing- divided dosing is best due to less risk of GI irritation, bleeding. Replace at rate of 20-80 mEq/day
- Patient on diuretics do not routinely need daily replacement therapy- monitor serum potassium levels if risk of hypokalemia complications are high when initiating or adjusting therapy-Those at risk may include diabetics, those with decreased LV function, on digoxin, or asthmatics on b2 agonists.

HyperKalemia

- Defined as serum potassium > 5.5 mEq/L
- Generally related decreased renal potassium secretion and/or abnormal potassium movement out of the cells.

Causes

- Pseudohyperkalemia is common- due to hemolysis of RBCS from difficult sticks, excessive fist clinching by patient. Thrombocytosis and leukemias (CML- cause falsely elevated levels due to cell fragility) can also cause this.
- Decreased potassium excretion is the most common cause of elevated potassium levels
    - Drugs such as ACE inhibitors, potassium sparing diuretics, Heparin, Lithium, NSAIDS, Bactrim, Tacrolimus
    - Adrenal Insufficeny
    - Acute Renal Failure, Chronic Kidney disease, Obstructive Uropathies
    - Decreased circulating volume (volume depletion).

- Increased dietary intake (food sources, supplements- although rarely an isolated cause) or via IV route from blood transfusions, IVF with supplemental K+, TPN, or potassium containing drugs (PCN G)

Causes (Continued)

- Increased Movement of Potassium Out of Cells
    - GI bleeding, burns, rhabdomyolysis causing increased tissue catabolisim
    - Drugs such as b blockers, Digoxin (toxicity)
    - Disorders of insulin deficiency such as DM, extreme fasting

- ***Remember hyperkalemia is un common in CKD until GFR falls to less that 10mL/min…unless po/IV intake is extreme. In Oliguric states, elevated potassium levels are common especially in setting of AKI, rhabdomyolysis, burns.

Signs and Symptoms of Hyperkalemia

- . Again, there are generally no symptoms until arrhythmia occurs
- Consider/Suspect in high risk patients such as known renal disease, advanced HF, or concurrent ACE and potassium sparing diuretic therapies

Serum Potassium	Typical ECG Appearance	Possible ECG Abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (> 8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

http://hyperkalemiablog.blogspot.com/2013_12_01archiver.html

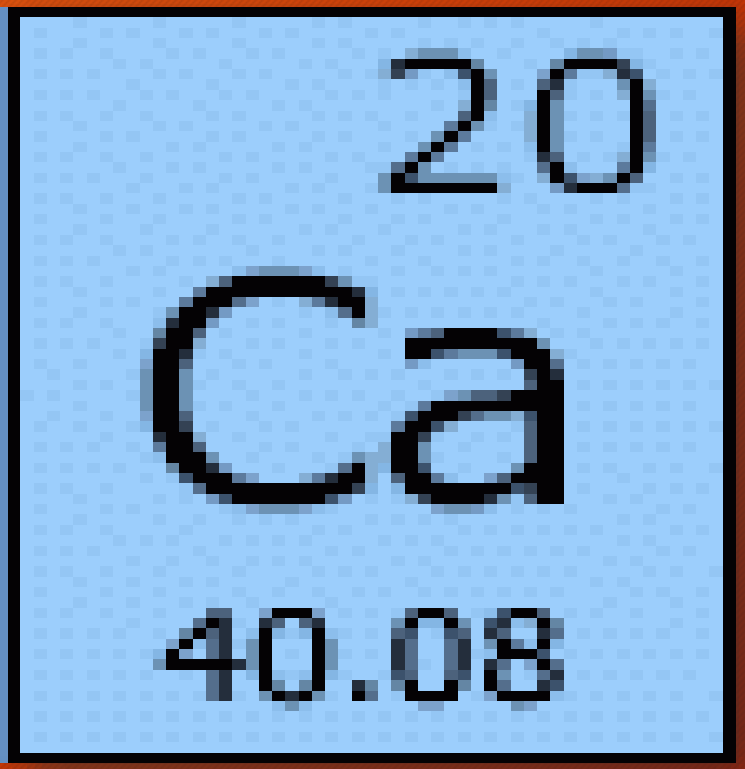
Early changes include widening of PR interval with shortening of the QT with tall, symmetric peaked T waves.

Higher levels lead to further slowing of conduction leading to further widening of QRS, loss of P wave

Ultimately, in severe Hyperkalemia, the QRS evolves into sine wave pattern and leads to fatal arrhythmia. (VF, asystole)

Treatment/Prevention

- In mild elevations- consider stopping the offending medications, limiting dietary intake.
- May consider addition of a loop diuretic if the offending medication cannot be stopped- increased renal K+ excretion but monitor for volume depletion.
- Sodium polystyrene sulfonate (Kayexelate) may also be given(dose 15-30 G) – mechanism is removal of potassium via the GI tract. May give PO or PR..Results are often slow. Can be ineffective in hypercatabolic states.
- Moderate to Severe elevations need more prompt attention and probable hospitalization
    - Consider addition of IV Insulin 5-10 units with immediate administration of D50
    - IV Calcium in addition to the above (gluconate) for more advance EKG changes such as loss of p wave or development of sine wave.
    - High dose albuterol in nebulized form can be added as adjuvant (0.5 to1.5mEQ/L decrease in Potassium level (DOES NOT PEAK FOR about 90 min)- do not use in acute MI/USA (b2 agonist)
    - Lastly, CCRT, acute hemodialysis can be considered – especially those with ESRD with significant EKG changes.



Calcium

Overview of Calcium and its Role

- Needed for muscle contraction, blood coagulation, hormone release and nerve conduction.
- Proper levels of calcium stores are dependent on dietary intake, GI absorption and renal calcium excretion
- Proper renal extraction is dependent on circulating PTH and calcitonin levels
- Remember IONIZED CALCIUM is the active form of Calcium in the body.
- Approximately 99% of the body's calcium is in bone. Only about 1% is freely available for buffering changes in serum calcium balance

Overview (continued)

- Normal serum calcium level 8.8- 10.4 mg/dL. This is generally a measurement of total calcium (about 40% of which is protein bound (albumin) and 60% of which is ionized calcium and calcium bound with phosphate and citrate. Again- ionized form is the ACTIVE form in the body thus the only physiologically important value.
- Ionized calcium is estimated at about 50% of total serum Ca.
- You can estimate ionized calcium if you have a serum calcium level and a serum albumin level.
- Normal Ionized Calcium levels are 4.7-5.2 mg/dL
- Calcium/phosphate balance are affected by the level of circulating PTH. Vitamin D, and calcitonin .

Overview (continued)

- PTH (Parathyroid Hormone) – secreted by parathyroid glands. Main role is to defend against hypocalcemia. Decreases in serum calcium cause the parathyroids to release PTH . This can increase serum calcium within minutes as it causes increased renal and GI absorption as well as mobilizing available calcium and phosphate from bone. Additionally, renal phosphate reabsorption is decreased- increasing renal loss.
- PTH stimulates conversion of Vitamin D to calcitrol (active form) which increases GI tract absorption of dietary calcium.
- Prolonged increased PTH secretion will lead to increased bone reabsorption due to inhibited osteoblast function and increased osteoclast activity.
- *** Vitamin D and PTH are important in regulation of bone remodeling and growth.
- If calcium levels are low- check intact PTH levels to asses parathyroid function.

Overview (continued)

- Calcitonin increases calcium concentration by increasing cellular uptake, renal excretion and bone formation. Secreted from the C cells (thyroid parafollicular cells). Calcitonin effects on bone are much less than that of Vitamin D or PTH.

Hypocalcemia

- Defined as serum calcium of < 8.8 mg/dL with normal plasma protein (albumin)concentrations OR ionized calcium of < 4.7mg/ dL

Causes

- Hypoparathyroidism-low calcium, low phosphate. Caused by low PTH (autoimmune disorders, accidental damage during thyroid surgery, removal of parathyroid due to adenoma (large). Characterized by high alk phos and phosphate, low PTH
- Vitamin D Deficiency/Dependency- Deficiency due to inadequate dietary intake, decreased absorption 2/2 GI malabsorption issues, or decreased exposure to UV light. Medications may also alter absorption (Dilantin, phenobarbital). Characterized by high PTH, low phos, high akl phos and low Vitamin D. Dependency is due to inabilitiy to convert Vitamin D to its active form or end organ decreased responsiveness to normal levels of circulating active Vitamin D. Two Subtypes.
- Psuedohypoparathyroidism-uncommon- not really an issue of PTH deficiency, rather characterized but target organ resistance. Genetic. Three subtypes.

Causes (continued)

- Renal Failure/Disease-
- Magnesium depletion- causes relative PTH deficiency
- Less commonly pancreatitis, hungry bone syndrome, large volume blood transfusions, septic shock

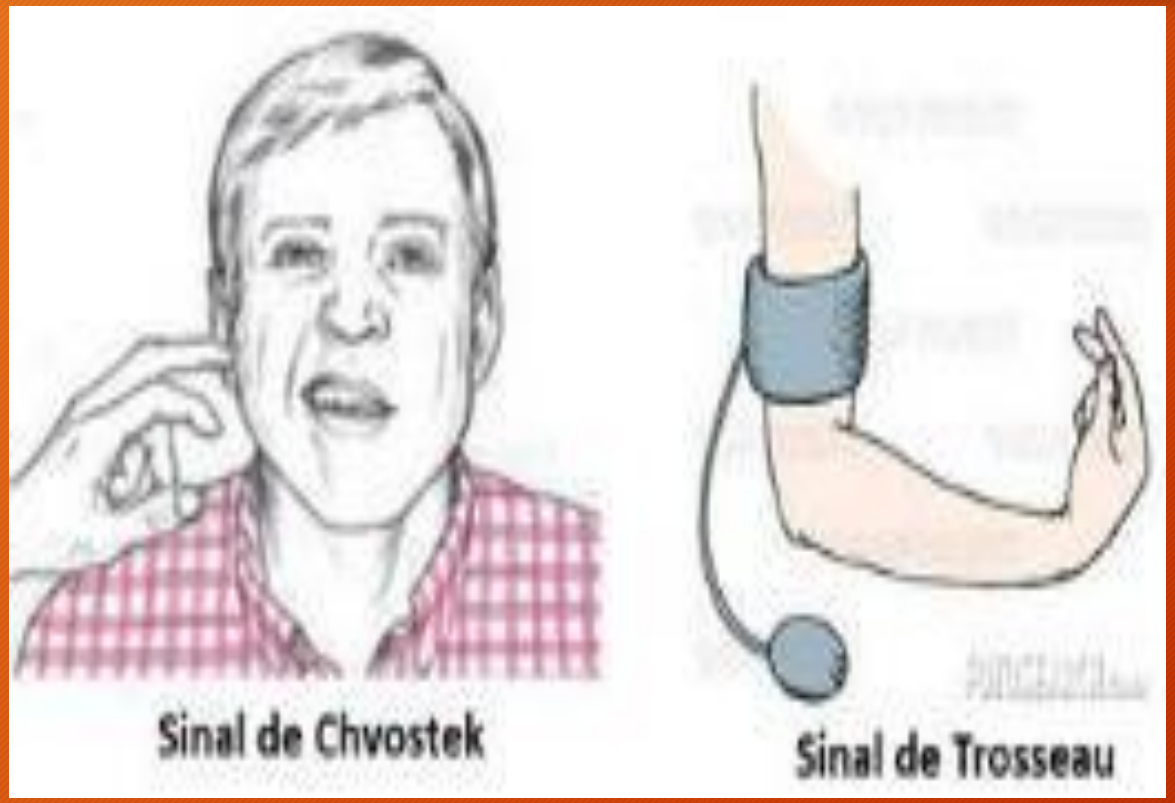
• Signs and Symptoms of Hypocalcemia

- Often asymptomatic

- Major symptoms are related to neuromuscular irritability- including muscle cramping, insidious mental status changes (dementia, depression).
- May see brittle nails, coarse hair, thin skin in chronic hypocalcemia
- Severe hypocalcemia can cause EKG changes – prolonged QTc , ulitimately heart block/arrhythmias.

Severe hypocalcemia (< 7 (serum), or < 1.75 (ionized))- may result in tetany , hyperreflexia, laryngospasm, or tonic clonic seizures. (s/s may sensory complaints of facial paresthesia, muscle aches, facial muscle spasms).

- Trousseau Sign
- Chvostek Sign



Trousseau-illicting carpal spasm with BP cuff or tourniquet- inflate cuff t oapproximately 29 mmHG above SBP and hold for 3 min- induces spasm of hand and forearm muscles- indicative of latent tetany-

Chvosteck- involuntary facial muscle twitching in response to tapping over the facial nerve- just anterior to the external ear meatus

www.ebah.com.br

Diagnosis

- If serum calcium is low- check ionized calcium. Additionally, consider checking Mg, alk phos, phosphate, and vitamin D levels as well as a renal panel
- If normal, further investigation with PTH intact
- EKG to assess for prolonged QTc which is typically proportional to severity of hypocalcemia.

Treatment

- If tetany present- IV calcium gluconate (need continuous EKG monitoring)
- Oral calcium for postoperative hypoparathyroidism- risk of severe hypocalcemia higher in those with CKD, large adenoma removal.
- Oral Calcium and Vitamin D for chronic hypocalcemia

Hypercalcemia

- Serum calcium > 10mg/dL or ionized calcium > 5.2mg/dL

Causes

- Generally, high calcium levels are due to problems that lead to excessive bone reabsorption

  - Primary Hyperparathyroidism (rarely > 12) - most common cause of hypercalcemia secondary to over secretion of PTH-generally due to a parathyroid adenoma. Increased incidence with aging, postmenopausal state and remotely after head/neck radiation. Generally, have low phos, high alk phos

  - Less commonly, can be caused by inherited conditions, parathyroid neoplasm.

  - End stage renal disease can cause tertiary hypoparathyroidism

  - Non parathyroid mediated etiologies include hypercalcemia of malignancy due to increased osteolytic activity of metastatic bone lesions(breast, NSCL CA, multiple myeloma) and/OR humoral hypercalcemia of malignancy(ovarian, renal cell, breast, prostate)- release of a PTH related peptide that is excreted and mimics PTH, increased calcitrol excretion.

Causes (continued)

- Certain medications including but not limited to- thiazide diuretics, theophylline. Additionally, excessive Vitamin A intake can contribute to hypercalcemia
- Prolonged immobilization (chronic bed bound state).
- Conditions such as acromegaly, pheochromcytoma and adrenal insufficiency.
- Hyperthyroidism (thyrotoxicosis), Addisons
- Milk- alkali syndrome
- Vitamin D Toxicity(due to large pharmacologic doses leading to excessive bone reabsorption, increased Ca absorption) or due to high endogenous levels RT lymphoma, T cell leukemia.
- Granulomatous Disorders such as sarcoidosis, TB, histoplasmosis.

Signs and Symptoms of Hypercalcemia

- Polyuria, polydipsia
- Nephrolithiasis
- Acute on Chronic renal failure
- Muscle weakness, bone pain
- N/V, appetite loss, constipation
- Pancreatitis
- Confusion, fatigue, decreased concentration
- Severe may lead to stupor, coma (> 12mg.mL)
- Shortened QT, elevated BP, bradycardia

Diagnosis

- Check serum calcium and ionized if available
- Chemistries, ALK PHOS, phosphate, BUN, creatinine
- CXR- screening for granulomatous dz, lung CA
- PTH

- *** Cause is often apparent based on preliminary testing and clinical exam, history alone.

Treatment

- If mild (< 11.5) consider oral phosphate supplementation if mild to no symptoms and no renal dieses
- Rapid correction (inpatient) for levels > 18 may include IV furosemide and IVF
- Biphosphanates (inhibit osteoclast activity) – DOC for cancer related hypercalcemia.
- Calcitonin.
- Hemodialyisis (>18), IV phosphate
- Surgical management of adenoma

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