

THE HIGHS AND LOWS OF
Electrolyte Evaluation and Management

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DISCLOSURES

I have no conflicts of interest to disclose.

OBJECTIVES

AT THE CONCLUSION OF THIS SESSION, PARTICIPANTS SHOULD BE ABLE TO:

- Recognize the presentation of common electrolyte abnormalities
- Discuss the evaluation of patients with suspected electrolyte disturbances
- Evaluate laboratory and physical exam findings in patients with electrolyte disturbances
- Discuss possible etiologies of electrolyte disturbances
- Develop a management plan for patients with electrolyte abnormalities

ELECTROLYTES

Substances in the body's fluid that dissociate into anions and cations

Broad functionality in human physiology:

- Acid-base homeostasis
- Fluid balance
- Cellular function
- Cardiac physiology

Sodium, potassium, calcium, phosphorous, magnesium, chloride, and bicarbonate

Na ⁺	Cl ⁻	BUN	GLU
K ⁺	HCO ₃	CR	

FLUID BALANCE

Electrolytes are dissolved in body fluid — water balance important to consider

- Vasopressin, water ingestion, renal water transport contribute to maintenance of normal osmolality of 280-295 mosmol/kg

Hypovolemia - volume depletion, salt and water loss

- Renal causes: osmotic diuresis, drug-related
- Extrarenal causes: loss from GI tract, skin, respiratory system, accumulation of fluid in tissue compartments

SODIUM DISORDERS

HYPONATREMIA

Plasma Na^+ < 135 mM

Common - 15-30% of hospitalized patients, potentially even higher!

- Premenopausal women at highest risk for severe neurologic sequelae

Pathophysiology: Principal osmole of extracellular fluid - generally a **water excess state**

- Hypoosmolar hyponatremia most common
- MDMA ingestion

Prognosis: Mortality

- Acute Hyponatremia (Serum Sodium < 120 meq/L): **50%**
 - Mortality associated with cerebral edema, osmotic demyelination syndrome
- Chronic Hyponatremia: 10%
 - Mortality associated with underlying condition



HYPONATREMIA SYMPTOMS

Acute (<24-48 hours)

Symptom onset generally at serum sodium <125 meq/L

- Nausea
- Vomiting
- Headache
- Lethargy
- Dizziness

Later or Severe Hyponatremia (serum sodium <120 meq/L)

- Seizure
- Coma
- Confusion
- Ataxia
- Respiratory depression

HYPONATREMIA SYMPTOMS

Chronic (developing over >48 hours)

Development and symptom onset variable - days, weeks, months

- Lethargy
- Confusion
- Muscle cramps
- Neurologic impairment - encephalopathy, cerebral edema

HYPONATREMIA CAUSES

Excessive free water replacement (with or without sodium replacement)

- Nausea, Vomiting, or Diarrhea
- Excessive, prolonged sweating with exercise
- Psychogenic polydipsia
- Hypotonic saline infusion (e.g. D5 1/2NS)

Excessive sodium renal excretion (salt-wasting)

- Diuretics (esp. Thiazide Diuretics, trimethoprim)
- Cerebral salt wasting (underlying neurologic disorder)
- Low Aldosterone (Mineralcorticoid deficiency)

HYPONATREMIA CAUSES

Syndrome of Inappropriate ADH Secretion (SIADH)

- Malignancy (e.g. Small Cell Lung Cancer, Pancreatic Cancer)
- Lung Infections (e.g. Pneumonia, Empyema, Tuberculosis, Legionella, ARDS)
- Neurologic disorders (e.g. brain mass, Meningitis, Intracranial Hemorrhage, CVA)
- Medications
 - Amiodarone
 - Neuropsychiatric agents (e.g. Amitriptyline, Carbamazepine, SSRI, Haloperidol)
 - Opioids and NSAIDs

Edematous States

- Renal Failure or Nephrotic Syndrome
- Congestive Heart Failure
- Cirrhosis or other severe liver disease

HYPONATREMIA WORK-UP

1. Is the patient hypervolemic?

- Hyponatremia due to edematous state (Cirrhosis, CHF, Nephrotic Syndrome) or renal failure?

2. Measure Serum Osmolarity

- Smaller labs may be unable to provide a measured serum osmolality
- Most cases are hypoosmolar hyponatremia (serum Osms <280)
- Exception: Severe hyperglycemia (hyperosmolar hyponatremia)

3. Obtain Bedside Glucose

- Pseudohyponatremia (normoosmolar hyponatremia) is rare
- Remember to calculate a corrected Na^+ for patients with hyperglycemia



HYPONATREMIA WORK-UP

Consider the Etiology

Hypovolemic Hypoosmolar Hyponatremia

- Fluid losses (e.g. Gastroenteritis)
- Third spacing (e.g. Pancreatitis)
- Renal sodium losses

Euvolemic Hypoosmolar Hyponatremia

- SIADH
- Water Intoxication
- Hypothyroidism
- Medications

Hypervolemic Hypoosmolar Hyponatremia

- Edematous State (Cirrhosis, CHF, Nephrotic Syndrome)
- Renal Failure

Hyperosmolar Hyponatremia (Serum Osm >300)

- Hyperglycemia (typical cause) with water shifting from cells to the extracellular compartment
- Serum sodium falls 1.6 mEq/L per serum glucose increase of every 100 mg/dl (over 100 mg/dl)
- May also occur with hypertonic infusions (Glucose, mannitol, glycine)

Normoosmolar Hyponatremia (Serum Osm 280-300)

- Known as Pseudohyponatremia - Rare
- Consider if known comorbidity:
 - Severe hypertriglyceridemia (>1500 mg/dl)
 - Serum Protein >10 g/dl (e.g. Multiple Myeloma)

HYPONATREMIA MANAGEMENT

The Over-Simplified Version

Hypovolemic Hypoosmolar Hyponatremia

- Slow normal saline replacement (no more than 0.5 mmol/L/hr)
- Be aware of patients needing more rapid correction – risk of cerebral edema
 - Hypertonic only for those with severe symptoms
- Correct water deficit (plus ongoing and insensible losses) over 48-72 hours



- Avoid increasing plasma Na^+ by >10 mM/24 hour

Isovolemic Hypoosmolar Hyponatremia

- Water restriction
- Stop offending agents – thiazides, medications causing SIADH

Hypervolemic Hypoosmolar Hyponatremia

- Water and sodium restriction

Hyperosmolar Hyponatremia

- Treat hyperglycemia – corrects with normalization of serum glucose

Normoosmolar Hyponatremia

- No sodium management required (lab abnormality only)
- Treat the underlying condition – triglycerides, multiple myeloma, etc.

HYPERNATREMIA

Plasma Na^+ > 145 mM: Severe symptoms typically begin at > 160 mM

Relatively uncommon: 1% of hospitalized elderly patients

Pathophysiology: Generally a **water deficit state**

- Issue is total body water, not a total body sodium excess
- Lacks normal physiologic response to free water loss
 - Excess water loss
 - Rarely excess sodium intake or sodium retention

Prognosis: Mortality

- Children
 - Acute Hyponatremia: **43%**
 - Chronic hyponatremia: **7-29%**
- Adults
 - Acute hyponatremia: **60%**

HYPERNATREMIA SYMPTOMS

Children

- Tachypnea
- Muscle weakness
- Motor restlessness
- High-pitched crying
- Lethargy
- Coma

Adults

- Anorexia
- Nausea/Vomiting
- Muscle weakness
- Lethargy
- Restlessness
- Hyperreflexia
- Spasticity
- Seizures

HYPERNATREMIA WORK-UP

Serum Labs

- CBC, CMP
 - Specifically serum Na, Ca, glucose, creatinine, BUN

Urine Labs

- Urine volume
- Urine sodium
- Urine potassium
- Urine chloride
- Urine calcium

Laboratory Calculations

- Serum osmolality
- Free water deficit
- Serum to urine electrolyte ratio (Na + K for each)
 - Helps determine role of kidney in excreting or retaining electrolyte free water

HYPERNATREMIA MANAGEMENT

The Over-Simplified Version

Hypervolemic Hypernatremia

- Discontinue hypertonic saline administration
- Administer diuretics
 - Furosemide AND high dose thiazide diuretic
 - Indapamide 2.5-5mg PO daily OR
 - Chlorothiazide 500mg IV Q12hr
- Consider primary hyperaldosteronism
 - If hypokalemic and with hypertension

Hypovolemic Hypernatremia

- Reverse underlying causes (renal)
- First, restore volume to correct hypotension
 - Normal saline
- Second, sodium correction
 - Administer free water (feeding tube or D5W) to slowly correct Na⁺

Isovolemic Hypernatremia

- Mild to moderate – Increase free water intake
- Moderate to severe – Replace free water deficit with D5W over 48 hours
 - Replace chronic hypernatremia slowly – 0.5 mEq/hr
- Then, external water sources (feeding tube) preferred
- Monitor electrolytes closely
- Do not replace water faster than a decrease of 1-2 mOsm/kg water/hr

POTASSIUM DISORDERS

HYPOKALEMIA

PLASMA K^+ <3.5 MEQ/L

Pathophysiology

- Transcellular K^+ shift
 - Medications (excess insulin, beta-agonists)
 - Metabolic acidosis
 - Thyrotoxicosis
- Renal K^+ loss
 - With hypertension – possible renovascular disease, renin-secreting tumor, or MH, Liddle's Syndrome, aldosterone abnormalities (e.g., Cushing's, 1° hyperaldosteronism)
 - Renal tubular acidosis
 - Diuretic use – thiazides and loop diuretics
- Extrarenal K^+ loss
 - Metabolic alkalosis – vomiting
 - GI or skin losses
 - Metabolic acidosis – DKA, diarrhea, laxatives
- Other causes
 - Anorexia
 - Inadequate intake (TPN)
 - Pseudohypokalemia – delayed lab analysis, severe leukocytosis (>75k)

HYPOKALEMIA SYMPTOMS

TYPICALLY BEGIN AT SERUM K^+ < 2.5 mEq/L

General

- Malaise
- Fatigue

Gastrointestinal

- Constipation
- Ileus
- Exacerbated Hepatic Encephalopathy

Cardiovascular

- Orthostatic Hypotension
- Hypertension
- Arrhythmias – V. Fib (risk 5-fold with acute or recent MI), Torsade de Pointes
- Fatigue

Renal

- Metabolic Alkalosis
- Polyuria, Polydipsia
- Decreased GFR
- Glucose Intolerance

Neurologic

- Weakness
- Decreased DTRs
- Paresthesias
- Cramps
- Restless Leg Syndrome
- Rhabdomyolysis
- Paralysis

HYPOKALEMIA WORK-UP

Serum Labs

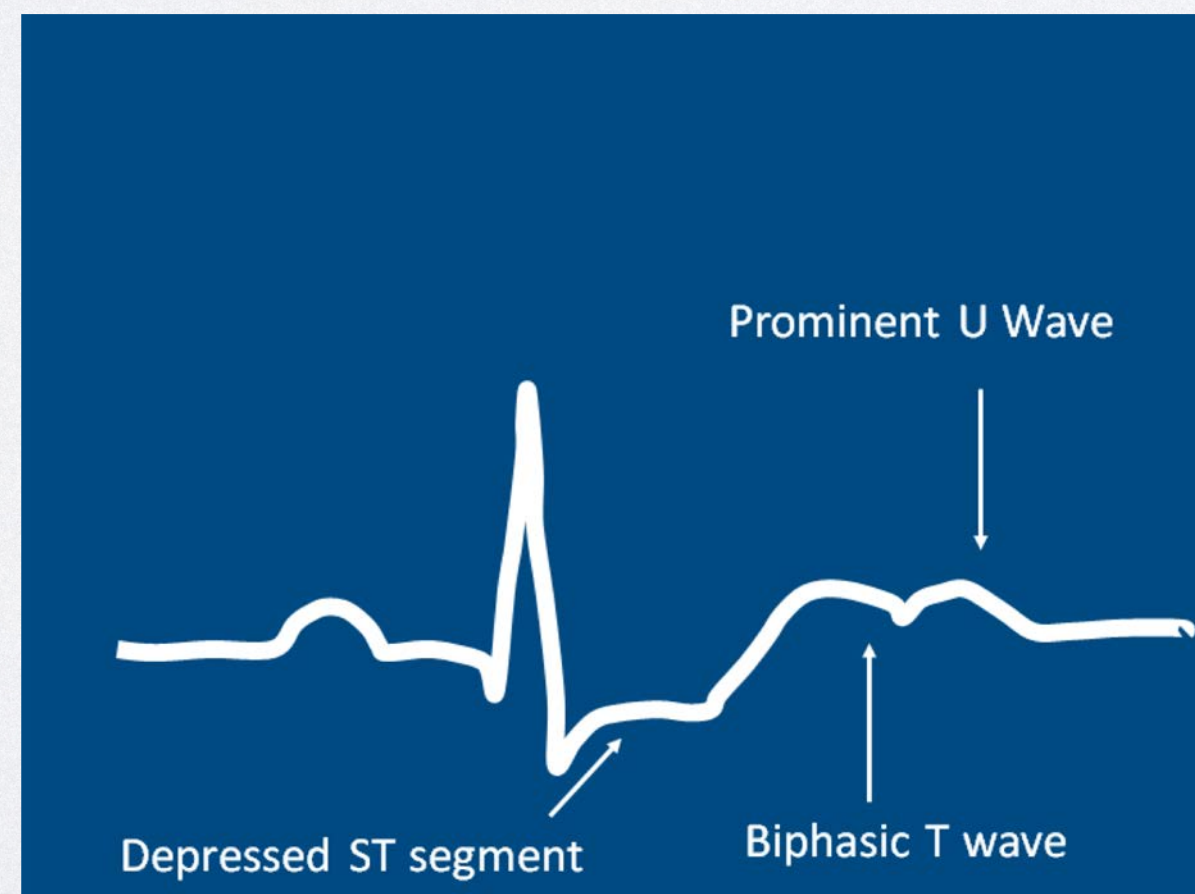
- CBC, CMP
 - Serum magnesium

Urine Labs

- Possible 24-hour urine potassium and sodium
 - Most accurate measure of K^+ excretion
- Urine potassium to creatinine ratio
 - Assess for renal vs. extrarenal loss

Electrocardiogram

- Early changes
 - T-waves - decreased amplitude to flattening
- Later changes
 - Prominent U-waves
 - ST depression
 - T-wave inversion
 - PR prolongation
 - QTc prolongation
- Associated arrhythmias
 - Sinus bradycardia
 - V. tach or V. fib
 - Torsade de Pointes



HYPOKALEMIA MANAGEMENT

The Over-Simplified Version

Replace Potassium

- Goal: $K^+ > 3.5$ mEq/L (4.0 in CAD, CHF)
- Administer potassium
 - < 2.5 mEq/L or serious EKG changes – consider larger dose/IV route
 - < 3.0 mEq/L: KCl 20 mEq orally Q 2 hrs for 4 doses, recheck
 - 3.0-3.5 mEq/L: KCl 20 mEq Q 2 hrs for 2 doses or 40 mEq once
- Likely continue at 20 mEq BID x 4-5 days
- Approximately half of replacement typically excreted by kidneys
 - Risk of hyperK low with normal kidney function and hydration
- Replace magnesium
 - Particularly if hypokalemia is refractory



Serum Potassium	Total Potassium Deficit
< 3.5 mEq/L	100 mEq
3.2 mEq/L	200 mEq
2.9 mEq/L	300 mEq
2.6 mEq/L	400 mEq

HYPERKALEMIA

PLASMA K^+ >5.2 MEQ/L

Pathophysiology (1 of 2)

- Decreased Renal Excretion
 - Hypoaldosteronism
 - Intrinsic renal disease
 - DM – esp. diabetic nephropathy
 - Interstitial nephritis
 - Adrenal insufficiency
 - Decreased distal renal flow
 - AKI, CKD, CHF
 - Primary tubular defects
 - Renal tubular acidosis
 - Obstructive uropathy
 - Medication-induced
 - ACE or ARB – responsible for 50% of med-induced hyper K^+
 - Also, heparin, Spironolactone, NSAIDs, Lithium
- Transcellular Shift or Redistribution (ICF to ECF)
 - Metabolic acidosis
 - Insulin deficiency or resistance (DM)
 - Rapid ECF rise - hypertonic glucose or mannitol
 - Dialysis
 - Coronary bypass
 - Cell lysis - any significant cause of cell turnover releases significant K^+ (98% of K^+ is intracellular)
 - Hypertonicity – hyperglycemia, mannitol
 - Medications – Succinylcholine, beta-blockers, Digitalis, arginine, somatostatin

HYPERKALEMIA

PLASMA K^+ >5.5 MEQ/L

Pathophysiology (2 of 2)

- Potassium Load
 - Potassium supplementation
 - Blood transfusion (PRBCs)
 - High dose penicillin G (1.7 mEq K^+ /1 million units)
- Pseudohyperkalemia
 - Lab issues
 - Sample analysis delayed
 - Sample clotted, hemolyzed, or cooled
 - Severe blood cell hyperplasia
 - Thrombocytosis
 - Leukocytosis ($>75k$)
 - Family History

HYPERKALEMIA SYMPTOMS

(TYPICALLY BEGIN WITH SERUM K^+ IS > 6.0 mEq/L)

Cardiovascular

- Bradycardia to asystole or V. fib
- AV prolonged transmission to complete heart block

Neurologic

- Weakness
- Paresthesias
- Areflexia
- Ascending paralysis
- Respiratory Failure

HYPERKALEMIA WORK-UP

Serum Labs

- CBC, CMP
- Other labs to consider: aldosterone and renin

Urine Labs

- Possible 24-hour urine potassium, sodium, and creatinine
 - Fractional excretion of K⁺
 - Urine sodium <25 mEq/L suggests decreased distal renal flow

EKG changes exacerbated by:

Hyponatremia
Hypocalcemia
Metabolic Acidosis
Hypermagnesemia
Chronic Renal Failure with frequent,
recurrent hyperkalemia

Electrocardiogram

- Initial changes
 - T-waves peaked or tented in V2, V3, II, III
- Next
 - ST depression
 - First degree AV block
 - QT interval shortening
- Next (ominous signs)
 - QRS widening (>110 msec)
 - Loss of p-wave (junctional rhythm)
 - Sine wave appearance
 - New bundle branch block
- Final
 - Biphasic wave with QRS and t-wave fusion
 - Severe bradycardia
 - Asystole, V. tach, V. fib

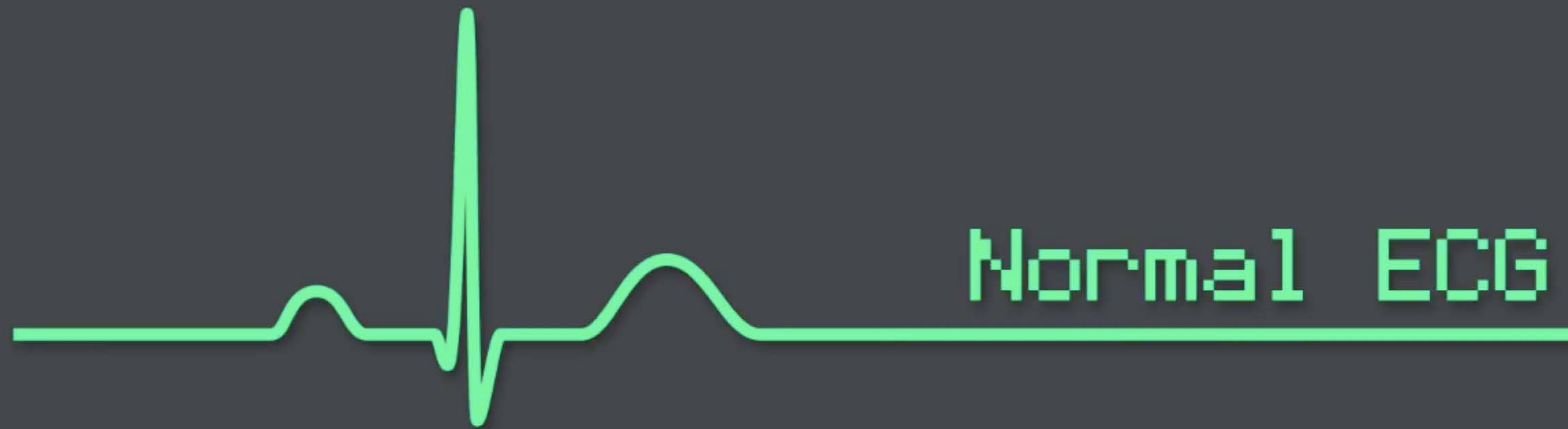


HYPERKALEMIA WORK-UP

Hyperkalaemia: ECG changes animated

Hyperkalaemia

K⁺: 4.1



artibiotics

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HD

HYPERKALEMIA MANAGEMENT

The Over-Simplified Version

Hyperkalemia Protocol

1. **Begin hyperkalemia workup**
 - Stop exogenous K⁺ sources
 - Fluids: NS vs. LR
 - NS is acidotic, LR contains K⁺
2. **Determine need for emergent Tx:**
 - Rapid and recent rise in K⁺
 - Renal insufficiency
 - Metabolic acidosis
 - EKG changes consistent with hyperK
 - Non-emergent if serum K⁺ <6.0 mEq/L

Emergent Treatment

1. Stabilize myocardium: Calcium chloride or calcium gluconate
 - Effect in 1-3 min, lasts 30-60 min, caution in dig tox
 - CaCl (5mL over 10min) or Ca Gluconate (10mL in 100mL of D5 infused over 20-30min, or faster if emergent; second dose in 5min if EKG not improved)
2. Temporarily shift K⁺ into intracellular space: Insulin+glucose, nebulized albuterol
 - Insulin regular 0.1 unit/kg up to 5-10 units IV AND D50W 50mL
 - Beware of hypoglycemia – repeat fingerstick glucose
 - Onset 15-30min, duration 2-6 hours
 - Lowers serum K⁺ 0.6 to 1 mEq/L
 - Albuterol: 10mg lowers K⁺ 0.5 mEq
3. Enhance K⁺ secretion: ~~Kayexalate~~, furosemide, hemodialysis
 - Binding agents not recommended in emergent hyperK⁺; Kayexalate has marginal efficacy, is poorly tolerated, has delayed onset of action, and risk of bowel necrosis
 - Furosemide 40-80mg IV push

MAGNESIUM DISORDERS

HYPOMAGNESEMIA

PLASMA < 1.8 mg/dL

Pathophysiology

- Decreased Intake
 - Alcoholics
 - Prolonged IV nutrition
- Decreased Absorption
 - Sprue, Crohn's
- Excess GI Loss
 - NG suction
 - Laxatives
 - Severe diarrhea
- Excess Renal Loss
 - Diuretics
 - Acute renal failure
 - Primary aldosteronism
 - Hypercalcemia
- Acute pancreatitis
- Endocrine
 - Hyperthyroidism
 - Hyperparathyroidism
 - Poorly controlled DM
- Medications
 - Cyclosporin
 - Gentamycin

HYPOMAGNESEMIA

PLASMA < 1.8 mg/dL

Signs and Symptoms

- Loss of appetite
- Nausea or vomiting
- Fatigue
- Weakness
- Vertigo
- Dysphagia
- Paresthesias
- Seizures
- Muscle Cramps to tetany
- Chvostek's sign
- Vertical nystagmus
- Arrhythmias

HYPOMAGNESEMIA MANAGEMENT

Assess Potassium Status

- Often related to magnesium status

Replace Magnesium

- Contraindicated in myasthenia gravis, caution in renal failure
- Do NOT exceed 100 meq/day
- Adjust replacement for decreased renal function
- Can be given PO or IV when more severe
 - Side effects- flushing, sweating, mild bradycardia, hypotension

HYPERMAGNESEMIA

PLASMA >2.5 mg/dL

Pathophysiology

- Renal Failure
- Medication overuse
 - Antacids
 - Milk of Mg
- Adrenal insufficiency (Addison's)
- Hypothyroidism (myxedema)
- Massive Mg dosing
 - Preeclampsia or eclampsia management

HYPERMAGNESEMIA MANAGEMENT

- Stop All Magnesium Sources
- Supportive Care with ABC Management
- Dialysis
- Cardiotoxicity Management
 - Calcium chloride or gluconate
 - Ca gluconate preferred

CALCIUM DISORDERS

HYPOCALCEMIA

PLASMA Ca^{2+} <8.5 mEq/L

Pathophysiology

Multifactorial causes

- Hypoalbuminemia
- Gram-negative sepsis

Low PTH

- Hypoparathyroidism

High PTH

- As a response to hypocalcemia

Other Causes

- Medication induced
- Pancreatitis
- Vitamin D deficiency
- Rhabdomyolysis
- Tumor lysis
- Pseudohypoparathyroidism
- After blood transfusion

HYPOCALCEMIA

PLASMA Ca^{2+} <8.5 mEq/L

Symptoms

Cardiovascular

- Dyspnea
- Edema
- Palpitations
- Syncope

Neurologic

- Headache
- Muscular fasciculations, cramping, stiffness
- Seizures
- Paresthesias
- Circumoral numbness
- Nervousness
- Weakness

Gastrointestinal

- Vomiting
- Diarrhea
- Dysphagia
- Abdominal pain

Signs

Neurologic

- Tetany
- Carpopedal spasm
- Chvostek's sign

Dermatologic

- Hair loss: dry, brittle hair
- Dry, puffy skin

Ophthalmologic

- Premature cataracts
- Papilledema

HYPOCALCEMIA

Serum Labs

- CBC, CMP
 - Low total corrected serum Ca^{2+} – albumin OR
 - Low ionized Ca^{2+}
- Magnesium
- Parathyroid hormone
- Vitamin D
- Confirm hypocalcemia with albumin correction

Electrocardiogram

- Paying close attention to QT interval

Management

- PO for low level, asymptomatic deficits
- Immediately replace calcium if symptomatic or <10 mEq/dL
 - Calcium gluconate (preferred) or calcium chloride
- Monitor calcium levels Q 4-6 hours
- Beware of
 - Bradycardia/asystole with rapid administration
 - Local injury – vein sclerosis, local chemical burns
 - Coronary vasospasm
 - Dig toxicity

HYPERCALCEMIA

PLASMA Ca^{2+} > 10.9 mEq/L

Pathophysiology

Malignancy

- Breast with bony mets, lung, RCC, head and neck squamous cell CA, MM, Hodgkin's lymphoma

Hyperparathyroidism

- Primary hyperparathyroidism (most common cause)
- Lithium treatment

Medications

- Thiazide diuretics
- Vit. A and Vit. D deficiencies
- Theophylline

Endocrine

- Adrenal insufficiency
- Thyrotoxicosis
- Pheochromocytoma
- Acromegaly

Other Causes

- Familial Hypocalciuric Hypercalcemia
- Prolonged immobilization
- Granulomatous disease (Sarcoidosis, Tuberculosis)

Paget's Disease of Bone

HYPERCALCEMIA

Symptoms - Often Asymptomatic

Cardiovascular

- Chest pain
- Dyspnea
- Palpitations
- Syncope

Gastrointestinal

- Anorexia
- Constipation
- Epigastric pain
- N/V

Renal

- Polydipsia
- Polyuria
- Renal colic

Neurologic

- Anxiety
- Confusion
- Fatigue
- Lethargy
- Weakness

Musculoskeletal

- Bone Pain
- Arthralgias

Skin

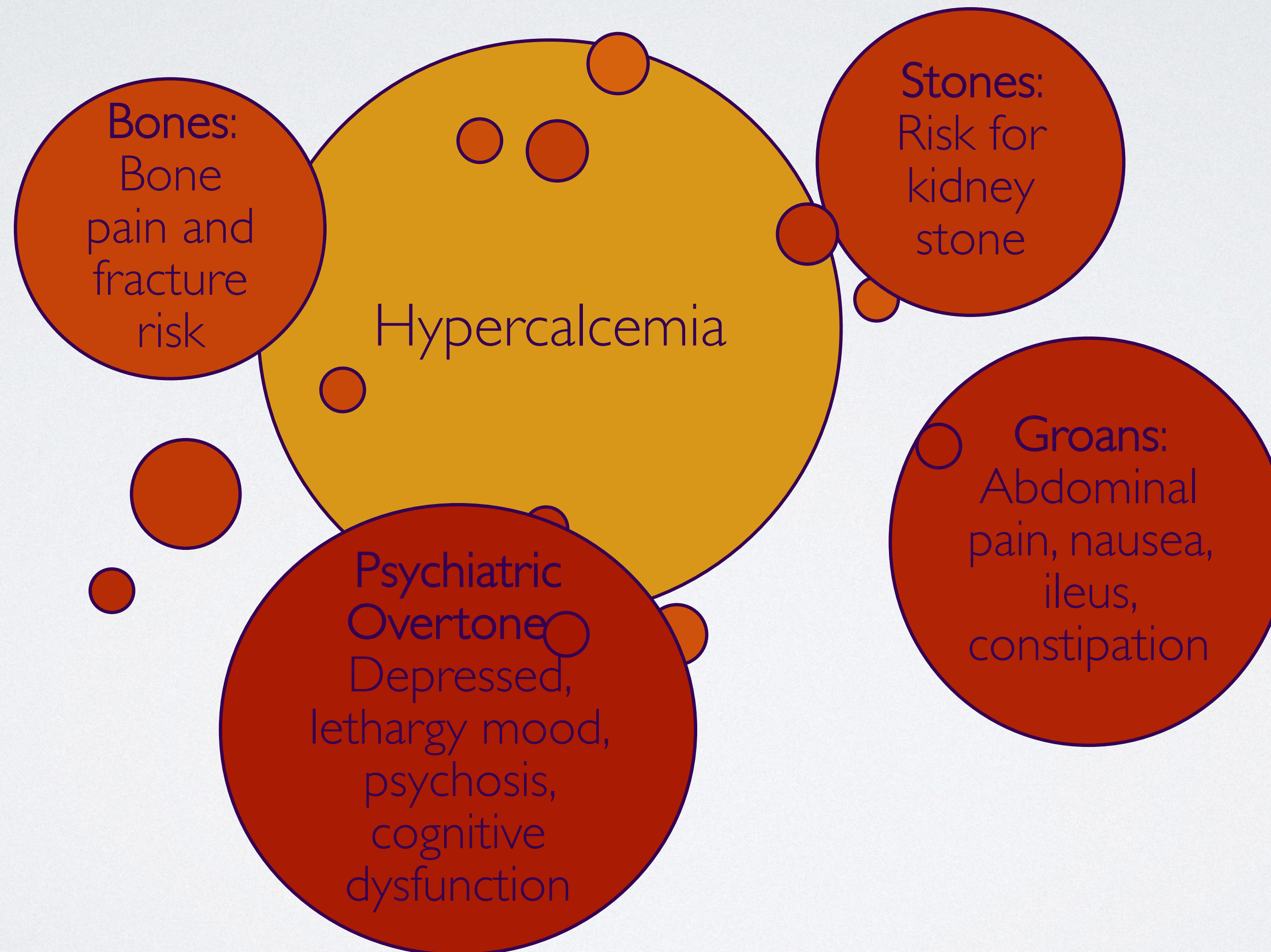
- Pruritis

Symptoms and signs are related to serum calcium levels:

Serum Calcium Level	Symptoms
> 11.5 mg/dl (2.9 mmol/L)	Symptom onset
> 13 mg/dl (3.2 mmol/L)	Calcium Nephrocalcinosis Acute Renal Failure
> 14 mg/dl (3.5 mmol/L)	Severe Hypercalcemia (or Parathyroid crisis)

HYPERCALCEMIA

Complications



HYPERCALCEMIA

Serum Labs

- CBC, CMP
 - High total corrected serum Ca^{2+} – albumin OR
 - High ionized Ca^{2+}
- Magnesium
- Parathyroid hormone
- 25-hydroxyvitamin D
- Serum creatinine

Electrocardiogram

- Short QT interval
- Prolonged PR interval
- Wide QRS complex
- Bradycardia

Management

- Immediate treatment for symptomatic or severe levels (serum calcium > 14 mg/dL)
- Eliminate potential causative medications

Mild Hypercalcemia (< 12 mg/dL)

- Adequate hydration: > 2 L/day
- Maximize mobility
- Diuretics if symptomatic: Lasix 40-160 mg/day

Moderate to Severe Hypercalcemia (> 14 mg/dL)

- Normal saline 2-4 L/day for 1-3 days
- May use Lasix
- Calcitonin (rapid onset, but weak effect)
- Bisphosphonates
- Oral phosphate
- Steroids – chronic granulomatous disease
- Dialysis

PHOSPHORUS DISORDERS

HYPOPHOSPHATEMIA

PLASMA <2.5 mg/dL

Pathophysiology

- Starvation
- DKA
- TPN
- Insulin
- NG suction
- Diuretics
- Vomiting
- Vitamin D Deficiency
- Hypoparathyroidism
- Pseudohypoparathyroidism
- Medications
 - Corticosteroids
 - Aluminum hydroxide antacids
 - Gram negative sepsis

HYPOPHOSPHATEMIA WORK-UP

Check Urinary Phosphate Excretion

- Low urine phosphorus (under 100mg/d)
 - Internal redistribution
 - GI losses
- High urine phosphorus (over 100mg/d)
 - Check for wasting in urine of other electrolytes

Treat Underlying Problem

- Replacement generally not needed

Replace Phosphate

- With symptoms (muscle weakness, rhabdomyolysis)
 - Typically when < 1.0 mg/dL
- Can be given PO or IV when more severe

HYPERPHOSPHATEMIA

PLASMA >5.0 mg/dL

Pathophysiology

- Renal Failure
- Dehydration
- Addison's Disease
- Hypervitaminosis D
- Hypoparathyroidism
- Magnesium deficiency
- Milk-Alkali Syndrome
- Transfusions
- Hemolysis
- Sarcoidosis
- Bone metastases
- Myelogenous Leukemia

HYPERPHOSPHATEMIA WORK-UP

Check Urinary Phosphate Excretion

- Normal urine phosphorus (under 1500mg/d)
 - Increased reabsorption
- High urine phosphorus (over 1500mg/d)
 - Endogenous source
 - Exogenous source

Management

- Manage kidney injury or CKD
- Antacid binding gel (AlternaGel)
- Low phosphate diet

CHLORIDE DISORDERS

HYPOCHLOREMIA

SERUM < 95 mEq/L

Pathophysiology

- Metabolic acidosis
 - Vomiting
 - Diarrhea
 - Diuresis
 - NG suction
 - Respiratory losses
 - Corticosteroids
- Cystic fibrosis

Together with K^+ , Na^+ , and CO_2 , Cl^- maintains acid-base balance

- Hyponatremia
 - Addison's disease
 - Edematous states – CHF
 - Salt-losing nephritis
 - SIADH
 - Renal failure
 - Pseudohyponatremia
 - Excessive sweating
 - Excessive IV D5W
 - Thiazide diuretics
 - Burns

HYPOCHLOREMIA WORK-UP

Consider Na⁺ status

Treat Underlying Problem

- Explicit replacement generally not needed

Replace Sodium as Indicated

HYPERCHLOREMIA

PLASMA > 105 mEq/L

Causes

Metabolic and endocrine

- Hyperparathyroidism
- Renal tubular acidosis
- Metabolic acidosis
- Hyponatremia

Gastrointestinal

- Dehydration
- Prolonged diarrhea

Medications

- Bromide intoxication
- Acetazolamide

- Boric acid
- Triamterene
- Excessive IV normal saline

Pathophysiology

- Metabolic acidosis with normal anion gap

Management

- Treat underlying problem
- Stop/alter offending medications
- Hydration

BICARBONATE DISORDERS

THE ROLE OF BICARBONATE

Primary acid-base buffer

Not a first line drug for resuscitation

- Treat acidosis with ventilation and perfusion
- Bicarbonate has not been shown to improve survival

Possible Indications

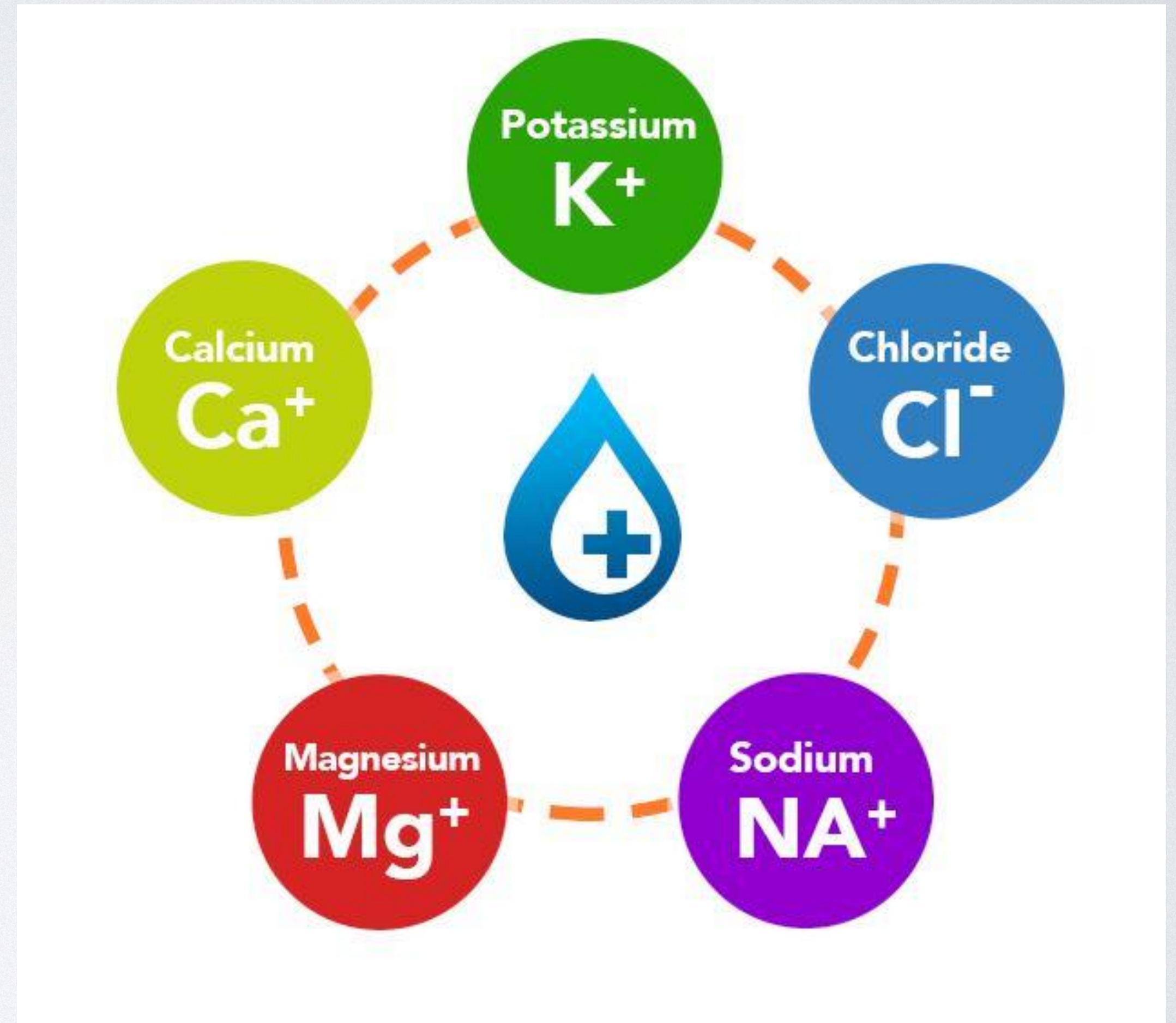
- Severe metabolic acidosis
- Prolonged cardiac arrest
- DKA with pH <6.9
- Hyperkalemia

Bicarbonate may transiently depress CV parameters

- Cardiac function
- Coronary artery perfusion

TAKE HOME POINTS

- Always consider the etiology of the electrolyte disturbance.
 - Treat the patient, not the number!
- Unless patient severely symptomatic, utilize a slow, gradual treatment perspective.
- For low electrolyte levels, consider possible etiologies of loss or dilutional effects.
- For elevated electrolyte levels, consider dehydration or fluid balance implications.



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