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:

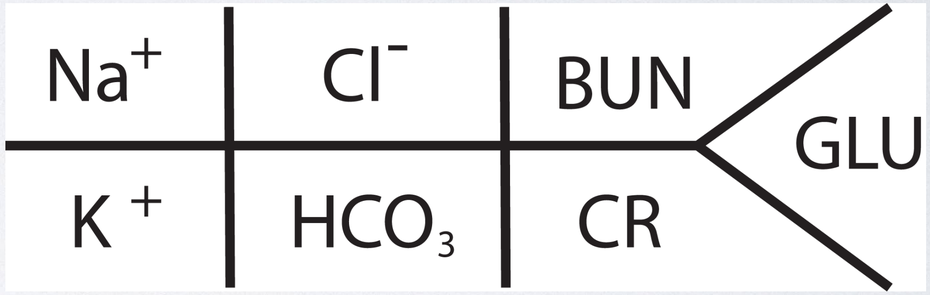
- Recognize the presentation of common electrolyte abnormalities
- Discuss the evaluation of patients with suspected electrolyte disturbances
- Evaluate laboratory and physical exam findings in patients with electrolyte disturbances
- Discuss possible etiologies of electrolyte disturbances
- Develop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

FLUID BALANCE

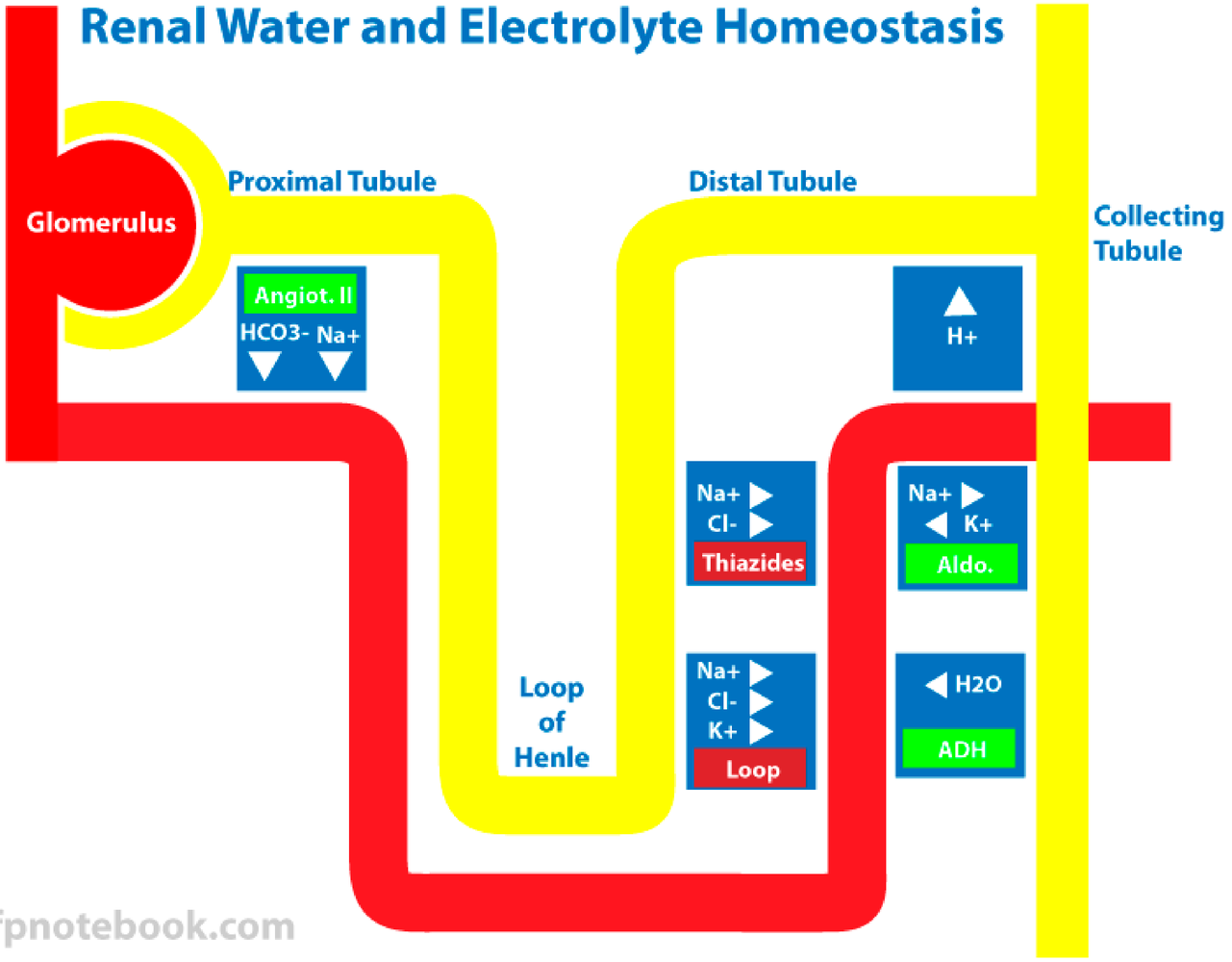
Electrolytes are dissolved in body fluid — water balance important to consider

- 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

FLUID BALANCE



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 hypo-osmolar hyponatremia (serum Osms <280)
    - Exception: Severe hyperglycemia (hyperosmolar hyponatremia)

3. Obtain Bedside Glucose
    - Pseudohyponatremia (normo-osmolar hyponatremia) is rare
    - Remember to calculate a corrected Na^+ for patients with hyperglycemia

HYPONATREMIA WORK-UP

Consider the Etiology

Hypovolemic Hypo-osmolar Hyponatremia

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

Euvolemic Hypo-osmolar Hyponatremia

- SIADH
- Water Intoxication
- Hypothyroidism
- Medications

Hypervolemic Hypo-osmolar Hyponatremia

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

Hyperosmolar Hyponatremia (Serum Osms >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)

Normo-osmolar Hyponatremia (Serum Osms 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 m*M*/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 m*M*

Relatively uncommon: 1% of hospitalized elderly patients

Prognosis: Mortality

- Children
  - Acute Hypernatremia: ***43%***
  - Chronic hypernatremia: ***7-29%***
- Adults
  - Acute hypernatremia: ***60%***

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

Severe symptoms: typically begin at >160mM

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

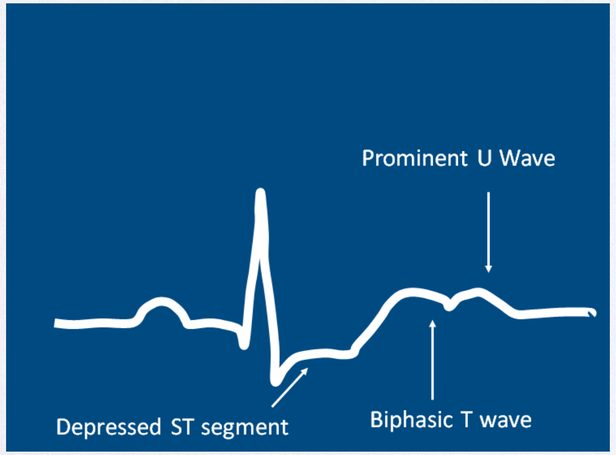
HYPOKALEMIA WORK-UP

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 hyperK^+
    - 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^+ >5.2 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

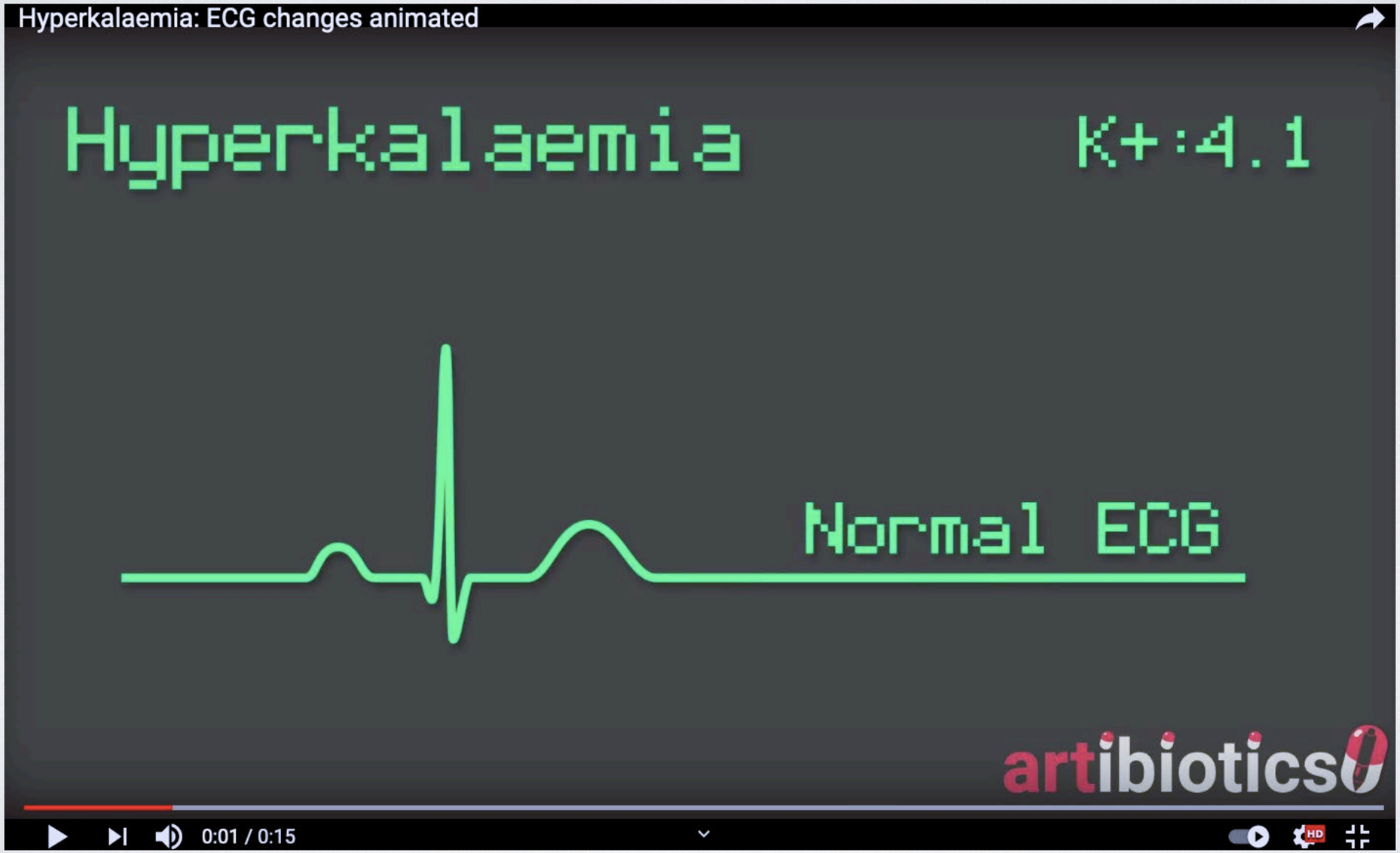
- Possible 24-hour urine potassium, sodium, and creatinine
  - Fractional excretion of K^+
  - Urine sodium <25 mEq/L suggests decreased distal renal flow

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



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 treatment
    - Rapid and recent rise in K^+
    - Renal insufficiency
    - Metabolic acidosis
    - EKG changes consistent with hyperK
    - Non-emergent if serum K^+ <6.0 mEq/L

HYPERKALEMIA MANAGEMENT

The Over-Simplified Version

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 0.1unit/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 Mg 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

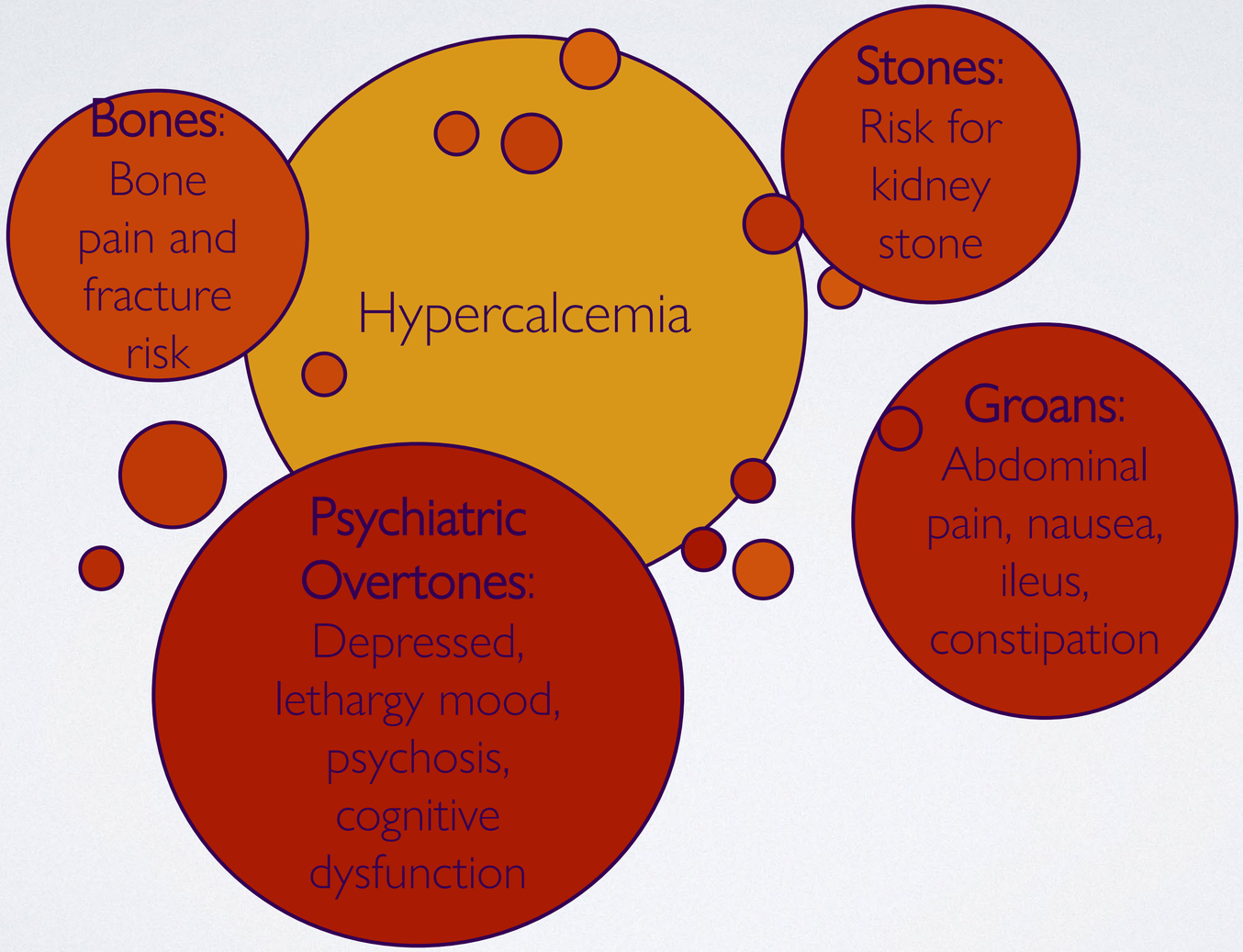
Skin
- Pruritis

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 CO2, 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
- Hypernatremia

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

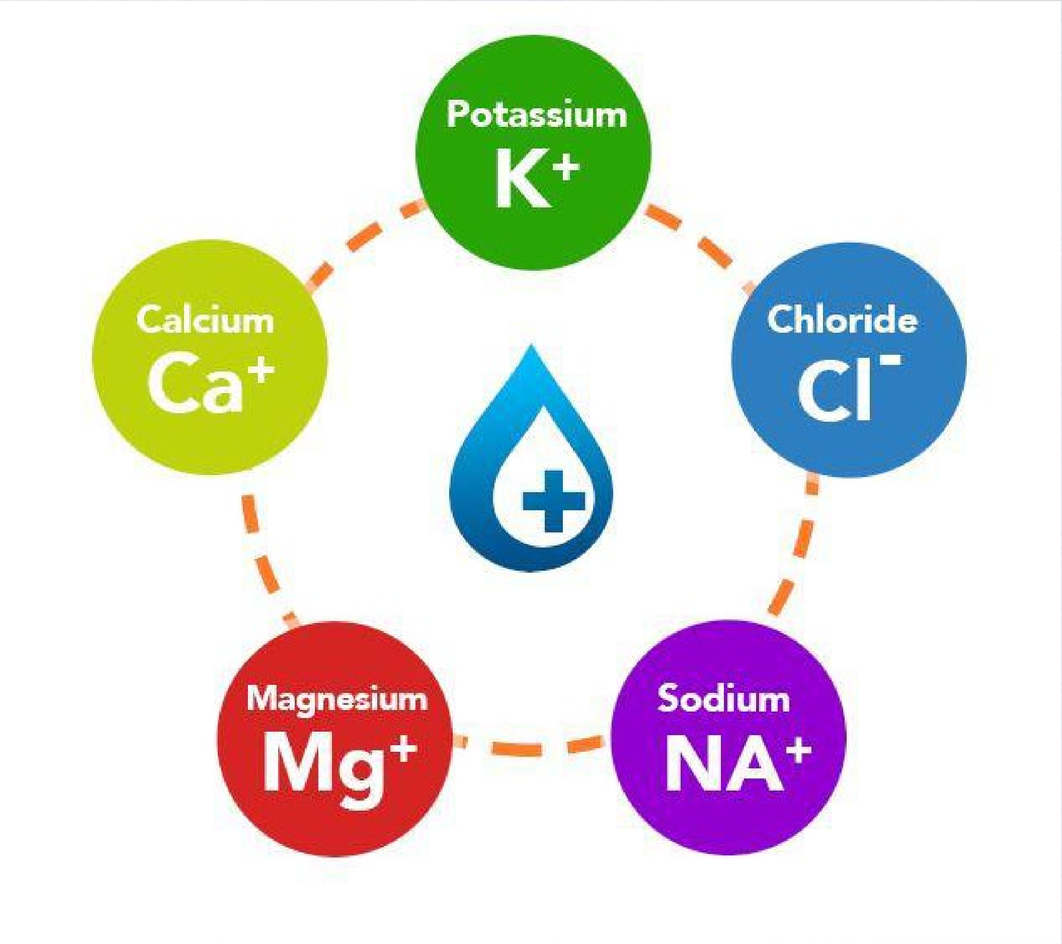
- Severe metabolic acidosis
- Prolonged cardiac arrest
- DKA with pH <6.9
- Hyperkalemia

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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