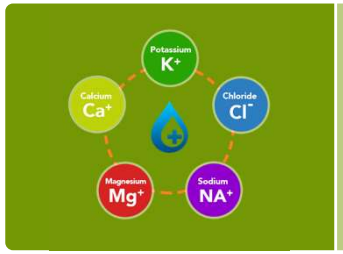


Electrolyte Disturbances: General Practice and Emergency Considerations

Sara Lefman, DVM DACVECC
Consultant to Heska

September 15, 2022

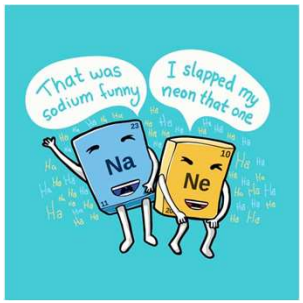
HESKA

1

Overview

- Disorders of:
  - Sodium and water
  - Chloride
  - Potassium
  - Calcium
  - Phosphorus

2



Sodium

3

Sodium in the body

- Most prevalent cation in extracellular fluid (ECF)
- Serves as the most important osmole, holding water in circulation
- Helps to determine plasma volume and thus patient's blood volume status
  - Euvolemic, hypovolemic, hypervolemic

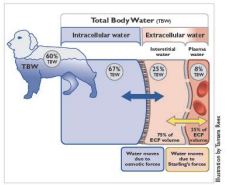


Figure 1. The distribution of body fluids by compartment. Starling's forces are hydrostatic and oncotic forces that determine fluid movement across capillary membranes as a result of filtration.

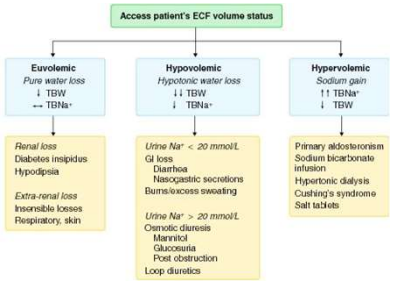
4

Hypernatremia

5

Approach to patient with hypernatremia

- What is the patient's **BLOOD VOLUME STATUS??**
  - Perfusion parameters



6

Causes of hypernatremia

- **Euvolemic hypernatremia=** loss of pure water
- **Water quickly moves from the IC to EC space via osmosis to replace what was lost**
  - Primary hypodipsia (mini Schnauzers, neuro animals)
  - Diabetes insipidus (central or nephrogenic)
  - High environmental temperatures/fever
  - Inadequate access to water

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Causes of hypernatremia

- **Hypovolemic hypernatremia=** loss of fluid that is similar to plasma
- **Movement from the ICF→ECF is slow**
  - Extra-renal
    - Gastrointestinal losses (activated charcoal)
    - Third spacing
    - Burns
  - Renal
    - Osmotic diuresis (mannitol)
    - Diuretics
    - Acute or chronic renal failure
    - Post-obstructive diuresis

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Causes of hypernatremia

- **Hypervolemic hypernatremia=** gain of sodium
- **Extra sodium expands ECF**
  - Salt poisoning
    - Paintballs!
  - Hypertonic fluid administration
    - Hypertonic saline
    - NaHCO3
    - Parenteral nutrition
  - Hyperaldosteronism

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Clinical signs of hypernatremia

- Neurologic signs
  - Ataxia, seizures, altered mentation
    - Often seen at Na >170mEq/L
  - Associated with movement of water out of brain cells and rapid brain shrinkage
  - Signs correlated to speed of development
- Anorexia, lethargy, weakness, vomiting

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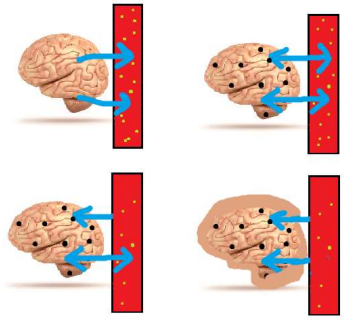
Chronic hypernatremia: Idiogenic osmoles



- The brain produces intracellular solutes to prevent dehydration
- If chronic hypernatremia is corrected too quickly, this can lead to **CEREBRAL EDEMA**

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Treatment of hypernatremia

- Replace the water as fast as it was lost!
- Ideally, calculate free water deficit

$$\underset{(L)}{\text{Water deficit}} = \underset{(kg)}{\text{Weight}} \times \left(\frac{\text{Na [measured]}}{\text{Na [desired]}} - 1 \right)$$

Na = sodium

Do not correct faster than 0.5meq/hr or a total of 10-12mEq/24 hours in chronic hypernatremia (more than 1-2 days)

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Treatment of hypernatremia

Start with a fluid which is closest to patient Na!

TABLE 1 Composition of Common Veterinary Fluids

FLUID TYPE	COMPONENT (unit)							BUFFER(S)	PRIMARY USE
	pH	Sodium (mEq/L)	Chloride (mEq/L)	Potassium (mEq/L)	Magnesium (mEq/L)	Calcium (mEq/L)	Osmolarity (mOsm/L)		
0.9% Saline	5.5	154	154	0	0	0	308	None	Replacement
0.45% Saline	5.6	77	77	0	0	0	154	None	Maintenance
Plasmalyte A	7.4	140	98	5	3	0	294	Acetate (27 mEq/L) Gluconate (23 mEq/L)	Replacement
Plasmalyte S6	5.0	40	40	13	3	0	363	None	Maintenance
Normosol-R	7.4	140	98	5	3	0	294	Acetate (27 mEq/L) Gluconate (23 mEq/L)	Replacement
Normosol-M	5.0	40	40	13	3	363	363	Acetate (16 mEq/L)	Maintenance
Lactated Ringer's solution (LRS)	6.5	130	109	4	0	2.7	273	Lactate (28 mEq/L)	Replacement
Hetastarch	5.5	154	154	0	0	0	309	None	Colloid

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Hyponatremia

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Approach to hyponatremia

- What is the patient's **OSMOLARITY and BLOOD VOLUME STATUS?**
  - Osmolarity= $2Na\ (mEq/L) + 2K\ (mEq/L) + \frac{glucose\ (mg/dL)}{18} + \frac{BUN\ (mg/dL)}{2.8}$

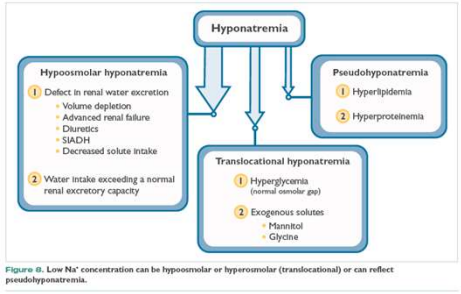

Figure 8. Low Na^+ concentration can be hypoosmolar or hyperosmolar (translocational) or can reflect pseudohyponatremia.

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Causes of hyponatremia

- **Normosmolar euvolemic hyponatremia**
  - Pseudohyponatremia from lab interference
    - Hyperlipidemia/cholesterolemia
    - Hyperproteinemia

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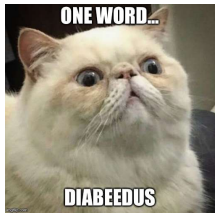
Causes of hyponatremia



- **Hyperosmolar hyponatremia**
  - Osmoles other than sodium pull water intravascularly and dilute sodium
    - Glucose
    - Mannitol infusion
- With hyperglycemia, each 100mg/dL increase in glucose decreases the Na by 1.6mEq/L

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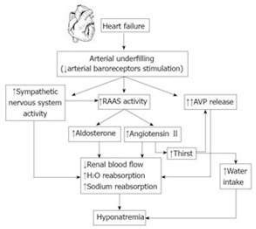
Causes of hyponatremia



- **Hypoosmolar hyponatremia with hypervolemia (edematous states)**
  - Hormone release and underlying disease increases water in vasculature and dilutes sodium
    - Severe liver disease
    - Congestive heart failure
    - Nephrotic syndrome/advanced renal failure

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Causes of hyponatremia

- **Hypoosmolar hyponatremia with normovolemia (rare)**
  - Inappropriate release of hormones or inappropriate drinking/fluids increases water in vasculature and dilutes sodium
    - Psychogenic polydipsia
    - SIADH
    - Antidiuretic drugs
      - Barbiturates, isoproterenol, narcotics
    - Myxedema coma of hypothyroidism
    - Hypotonic fluid administration

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Causes of hyponatremia

- **Hypoosmolar hyponatremia with hypovolemia (most common)**
  - During hypovolemia, drinking and appropriate release of hormones increases water in vasculature and dilutes sodium
    - Gastrointestinal losses
    - Third spacing (ascites, pleural, pericardial effusion, SQ edema)
    - Pancreatitis
    - Burns
    - Hypoadrenocorticism
    - Diuretics
    - Renal losses

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Clinical signs of hyponatremia

- Related to rapidity of onset and severity of hypoosmolality
- Neurologic signs
- Lethargy, nausea

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Treatment of hyponatremia

- Correct it as fast as it happened
- With chronic, do not raise Na more than **10-12mEq/24 hours**
- Not all patients require correction
  - With chronic, asymptomatic hyponatremia, water restriction may be enough (psychogenic polydipsia)

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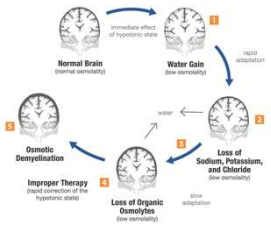
Treatment of hyponatremia



- Central pontine demyelination/myelinolysis
  - Develops several days after overly aggressive correction
  - Ataxia, lethargy, weakness, quadriparesis
  - Recovery is possible but prolonged

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Chloride

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Chloride in the body

- Dominant anion in ECF
- Most prevalent anion in gastric juice and intestinal fluids; secreted in jejunum, ileum and colon
- Important in acid-base balance

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Clinical approach to chloride disorders

- Changes in chloride can result from changes in water balance or by gain/loss of Cl^-
- Na and Cl usually go in the same direction EXCEPT:
  - Renal failure/tubular acidosis
  - Potassium bromide
  - Upper GI obstruction

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Treatment of chloride disorders

- Treatment of underlying cause
- Fluids (NaCl) or chloride containing salts if fluids are unnecessary

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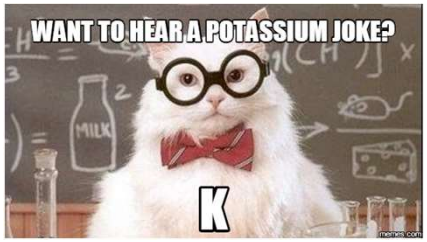
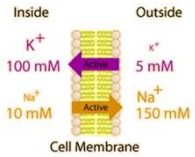
Potassium



28

Potassium in the body



- Major intracellular cation (95% or > in cells)
  - Muscle cells contain majority of total body potassium
- Required for maintenance of normal cell volume and growth
- Excreted by the kidneys* and colon
- Generates the normal negative resting membrane potential

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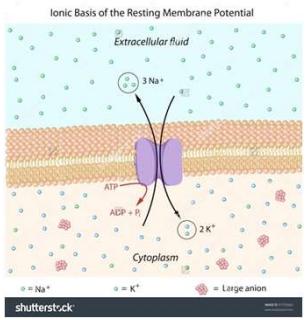
Membrane potentials



- The major determinant of the **resting cell membrane potential** is the ratio of the intracellular to extracellular [ ] of K
  - Na-K-ATPase pumps 3 Na++ out of cells and 2 K+ into cells
  - K+ ions diffuse out of cell down [ ] gradient

30

Membrane potentials

- The threshold potential must be reached to achieve an action potential
- Tissue excitability is determined by the difference between resting and threshold potentials

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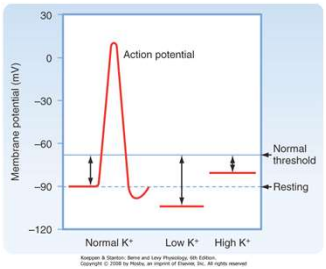
Membrane potentials



- Hypokalemia hyperpolarizes the cell
- Hyperkalemia makes the cell hyperexcitable
- Hypercalcemia affects the threshold potential and normalizes the difference between the two potentials

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Internal potassium balance

- Changes in K result from **changes in intake, distribution or excretion/loss**
- Intake= how much you eat
- Distribution
  - Translocation of K between ECF and ICF helps maintain balance
    - Acute acidosis causes shift of K from ICF→ECF in exchange for H+
- Excretion is mainly renal
  - Aldosterone is the most important hormone affecting excretion

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Hypokalemia

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Causes of hypokalemia

- **Inadequate intake**
  - Starvation
  - Cat litter ingestion (bentonite clay)
- **Distributional/translocation**
  - Alkalemia
  - Insulin/glucose containing fluids
  - Epinephrine, norepinephrine
  - Beta-agonist (albuterol) overdose
  - Hypokalemic periodic paralysis in Burmese cats

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Causes of hypokalemia

- **Loss**
  - Vomiting
  - Diarrhea
  - Chronic renal failure
    - 20-30% of cats
  - Distal renal tubular acidosis (type I)
  - Post-obstructive diuresis
  - Dialysis
  - Furosemide/hydrochlorothiazide
  - Hyperaldosteronism** (severe low K)

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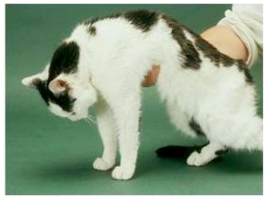
Clinical signs of hypokalemia



- Musculoskeletal
  - Weakness if K <3
  - CK increases with K <2.5
  - Rhabdomyolysis <2
    - Cervical ventroflexion
    - Broad based HL stance
    - Respiratory muscle paralysis

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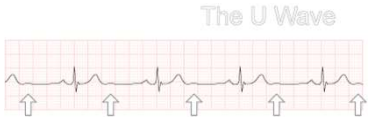
Clinical signs of hypokalemia

- Cardiac
  - Arrhythmias
    - Delayed ventricular repolarization
  - ECG changes: decreased amplitude T waves, ST segment depression, U waves (<2)
  - Refractoriness to lidocaine/procainamide



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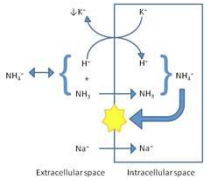
Clinical signs of hypokalemia



- Renal
  - Increases ammonia production in the kidney
    - **Bad with portosystemic shunt!!**

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Treatment of hypokalemia

- Parenteral supplementation (KCl or KPhos)
  - Mix the bag!
  - Use central catheters with >60mEq/L of K
  - "K-max" : 0.5meq/kg/hr
- Oral supplementation
  - K gluconate (Tumil-K, Renakare Kaon)
  - Recommend 2-4mEq/d divided into BID or TID

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Hyperkalemia

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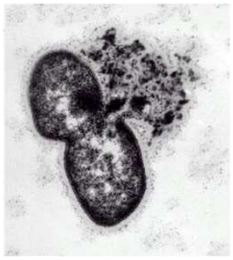
Causes of hyperkalemia



- **Translocation from ICF→ECF**
  - Acute acidosis (renal, respiratory)
  - Insulin deficiency
  - Massive tissue breakdown/tumor lysis
  - Ischemia:reperfusion injury
  - Nonspecific beta-blockers (propranolol)
  - Cardiac glycosides- digoxin

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Causes of hyperkalemia

- Hypothyroidism
  - Decreased activity of Na-K-ATPase
- Hemolysis or thrombocytosis
  - In breeds with high RBC [K] such as Akitas, Shibas, Jindos or in neonates
- Whipworm infestation, *Salmonella*

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Causes of hyperkalemia

- **Decreased urinary excretion**
  - Urethral obstruction (within 48 hrs)
  - Ruptured bladder
  - Oliguric/anuric renal failure
  - Hypoadrenocorticism
  - Repeated drainage of large volume effusion

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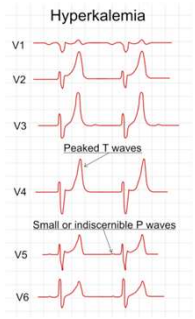
Clinical signs of hyperkalemia



- Musculoskeletal
  - Weakness if >8
- Cardiac
  - ECG changes
    - Tall/tented T, shortened Q-T interval, prolonged PR interval and widening of QRS; finally, decreased/absent P
  - Bradycardia
  - Ventricular arrhythmias, fibrillation or asystole

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Treatment of hyperkalemia

- Enhancing excretion/removal
  - Fluid therapy (any)
  - Diuretics (loop or thiazide)
  - Dialysis
- Promoting intracellular shifts
  - Regular insulin and/or dextrose
    - Insulin 0.25-1 U/kg IV- works within 1 hr
    - Dextrose 0.5g/kg IV bolus followed by CRI
  - Albuterol (1-2 puffs once)
  - NaHCO3
- Protecting the heart -*Does NOT lower K+*
  - Calcium gluconate
    - 0.5-1.5ml/kg bolus of 10% calcium gluconate; dilute 1:1 and give over 10 min with ECG

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Calcium

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Calcium in the body

- Used for skeletal support, muscle contraction
- Required for enzymatic reactions, coagulation, nerve conduction
- Homeostatic control via PTH hormone and calcitonin, vitamin D
  - Reabsorption of Ca from kidney, intestines and redistribution from bone
- PTHrP is a hormone generated from certain neoplastic cells which mimics PTH

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Calcium in the body

- In various forms including bound, complexed and ionized
- Total calcium can work as a surrogate but ionized most important!
  - Depends on acid/base status

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Hypercalcemia

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Causes of hypercalcemia

- Excess absorption/decreased excretion/increased release from bone
  - Malignancy (#1 in dog)
  - Primary and secondary hyperparathyroidism
  - Hypoadrenocorticism
  - Renal failure (chronic > acute)
  - Vitamin D toxicity
  - Inflammatory disorders/granulomatous diseases
- Idiopathic (#1 in cat)
- Spurious (lipemia)

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Clinical signs of hypercalcemia

- Alters cell membrane permeability→cell death
- Gastrointestinal tract
  - Vomiting, anorexia, constipation
- Renal
  - PU/PD*, dehydration, azotemia, acute/chronic renal failure
- CNS
  - Weakness, lethargy, seizures/twitching
- Cardiac
  - Arrhythmias
    - PR prolongation and QT shortening

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Treatment of hypercalcemia

- Usually indicated if tCa >16mg/dL
- Enhance excretion
  - NaCl
  - Furosemide
    - 2-4mg/kg any route BID to TID
  - NaHCO3
    - Can give up to 4meq/kg total dose
  - Glucocorticoids
    - 1-2mg/kg any route BID
- Decrease release from bone and absorption from intestines
  - Calcitonin
  - Bisphosphonates

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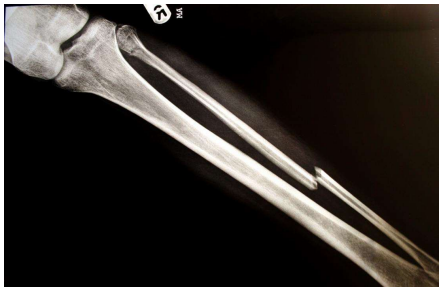
Hypocalcemia



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Causes of hypocalcemia

- Hypoalbuminemia= does not reflect ionized levels
- Excessive utilization
  - Eclampsia**
  - Sepsis/SIRS/critical illness
- Loss
  - Lymphangiectasia

J Am Anim Hosp Assoc. 2015 Nov-Dec;51(6):300-4. doi: 10.5326/JAAHA-MS-6205.

Seizures Associated With Hypocalcemia in a Yorkshire Terrier With Protein-Losing Enteropathy.

Whitehead J[1], Quimby J[1], Bayless D[1].

- Insufficient release/production
  - Hypoparathyroidism
  - Renal failure from decreased calcitriol
- Chelation/saponification/protein binding
  - Multiple blood transfusions (citrate)
  - Pancreatitis
  - Bicarb administration
  - Ethylene glycol

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Clinical signs of hypocalcemia

- Gastrointestinal tract
  - Anorexia
- CNS/muscular
  - Tremors/fasciculations, muscle cramping/stiffness
  - Hyperthermia
- Facial rubbing, panting, anxiety
- Cardiac
  - Arrhythmias
    - Tachycardia, prolonged QT interval

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Treatment of hypocalcemia

- Ca+ gluconate
  - 0.5-1.5ml/kg bolus of 10% calcium gluconate; dilute 1:1 and give over 10 min with ECG
  - Can put on CRI
- SQ Ca gluconate in dogs when diluted 1:1?
- Supplemental elemental calcium (TUMS)
  - 25-50mg/kg/d
- Wean puppies!
- Calcitriol

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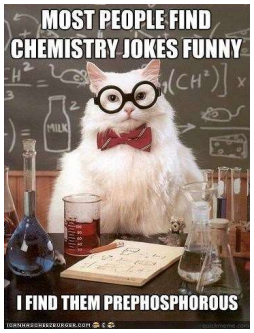
Phosphorus



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Phosphorus in the body

- Body's major intracellular anion
- Primarily stored in bone, muscle
- Function
  - Part of nucleic acids
  - Energy source (ATP, 2-3 DPG), component of glycogen
  - Urinary buffer which helps with acidosis

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Hyperphosphatemia

60

Causes of hyperphosphatemia

- Decreased renal excretion (azotemia)*
- Maldistribution
  - Tumor lysis syndrome
    - P high in myloblasts/lymphoblasts
  - Rhabdomyolysis
  - Hemolysis
  - Metabolic acidosis
- Increased intake
  - Phosphate enemas
  - Vitamin D intoxication (rodenticides)
- Hypoparathyroidism
- Normal in puppies <8wks

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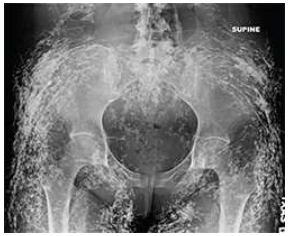
Clinical effects of hyperphosphatemia

- Decreases serum calcium concentration
- Soft tissue mineralization from abnormal Ca and P ratio



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Treatment of hyperphosphatemia

- Fluids enhance renal excretion
- Restrict protein in diet / phosphate binders

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Hypophosphatemia

64

Causes of hypophosphatemia

- Maldistribution
  - Insulin and/or dextrose administration*
  - Respiratory alkalosis/hyperventilation*
  - Refeeding
- Increased loss
  - Primary hyperparathyroidism*
  - Proximal tubule diuretics- acetazolamide
  - Renal tubular disorders
- Decreased intake
  - Phosphate binders
  - Vitamin D deficiency, malnutrition

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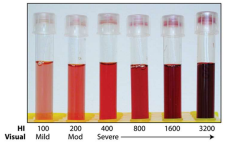
Clinical effects of hypophosphatemia



- Hemolysis
  - Observed when P <1mg/dL
- Impaired O2 delivery to tissues from RBCs (2-3 DPG)
- Impaired leukocyte and platelet function
- Muscle weakness/pain
- Metabolic encephalopathy
- Bone demineralization over time

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Treatment of hypophosphatemia

- Only indicated if clinical signs present, patient is at risk or if levels <2mg/dL
- NaPhos or Kphos injectable
  - 0.01-0.06mmol/kg/hr
- Do not give with LRS! (precipitation)

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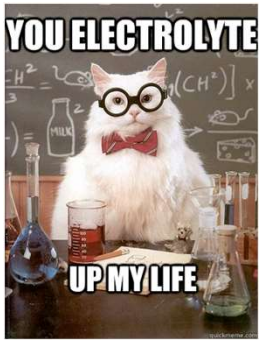
Questions?



Remember to
download the CE certificate
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NOTE: CE certificate not available
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Questions about CE?
events@heska.com

Questions about topic?
saracytosis@gmail.com

Thank you for joining us!

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