Major intra and extra cellular electrolytes

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Lecture 5

Electrolytes

Substances whose molecules dissociate into ions when they are placed in water.

CATIONS (+) ***ANIONS (-)***

Medically significant / routinely ordered electrolytes include***:***

Cation: Positively Charged particles.
Sodium (Na^+)
Potassium (K^+)
Calcium (Ca^{++})
Magnesium (Mg^{++})

Anion: Negatively charged particles.
Chloride (Cl^-)
Bicarbonate (HCO_3^-)
Phosphate (HPO_4^-)

Electrochemical Equivalence

- **Equivalent (*Eq/L*) = moles x valence**
- **Monovalent Ions** (Na^+, K^+, Cl^-):
  - – 1 milliequivalent (*mEq/L*) = 1 millimole
- **Divalent Ions** (Ca^{++}, Mg^{++}, and HPO_4^{2-})
  - – 1 milliequivalent = 0.5 millimole

Electrolyte Functions

-Volume and osmotic regulation
-Myocardial rhythm and contractility
-Cofactors in enzyme activation
-Regulation of ATPase ion pumps
-Acid-base balance
-Blood coagulation
-Neuromuscular excitability
-Production of ATP from glucose

Sodium

Functions

-Most abundant extracellular cation.

-Regulates body water distribution.

-Aids nerve impulse transmission.

-Aids transfer of calcium into cells.

Regulation of Sodium

-Concentration depends on:

- -intake of water in response to thirst
- -excretion of water due to blood volume or -osmolality changes

-Renal regulation of sodium

- -Kidneys can conserve or excrete Na+ depending on ECF and blood volume
  - -by aldosterone
  - -and the renin-angiotensin system
    - this system will stimulate the adrenal cortex to secrete aldosterone.

Aldosterone

From the (adrenal cortex)

Functions

promote excretion of K

in exchange for reabsorption of Na

Sodium normal values

Serum – 135-148 mEq/L

Clinical Features: Sodium

Hyponatremia: < 135 mmol/L

- Increased Na+ loss
  - Aldosterone deficiency
    - ***Addison's disease (hypo-adrenalism, result in ❧ aldosterone)***
  - Diabetes mellitus
    - ***In acidosis of diabetes, Na is excreted with ketones***
  - Potassium depletion
    - ***K normally excreted , if none, then Na***
  - Loss of gastric contents

- Increased water retention
  - Dilution of serum/plasma Na+
  - excretion of > 20 mmol /mEq urine sodium)
    - Renal failure
    - Nephrotic syndrome
- Water imbalance
  - Excess water intake
  - Chronic condition

Hypernatremia

Excess water loss resulting in dehydration
(relative increase)

- Sweating
- Diarrhea
- Burns
- Dehydration from inadequate water intake, including thirst mechanism problems
- Diabetes insipidus (ADH deficiency …↑ H_2O loss)

-Excessive IV therapy
-comatose diabetics following treatment with insulin. Some Na in the cells is kicked out as it is replaced with potassium***.***

-Cushing's syndrome ***- opposite of Addison’s***

Potassium

Functions

Most abundant intracellular cation.
Necessary for transmission and conduction of nerve impulses.
Maintenance of normal cardiac rhythm.
Necessary for smooth and skeletal muscle contraction.

the major cation of intracellular fluid

- Only 2 % of potassium is in the plasma
- Potassium concentration inside cells is 20 X greater than it is outside.
- This is maintained by the Na pump, (exchanges 3 Na for 1 K)

$$\frac{INSIDE}{OUTSIDE} = \frac{20}{1}$$

Regulation

Diet

easily consumed (bananas etc.)

Kidneys

Kidneys - responsible for regulation. Potassium is readily excreted, but gets reabsorbed in the proximal tubule - under the control of ALDOSTERONE

Potassium normal values

Serum (adults) – ***3.5 - 5.3 mEq/L***

Newborns slightly higher – ***3.7 - 5.9 mEq/L***

Hypokalemia

Decrease in K concentration

Effects

neuromuscular weakness & cardiac arrhythmia

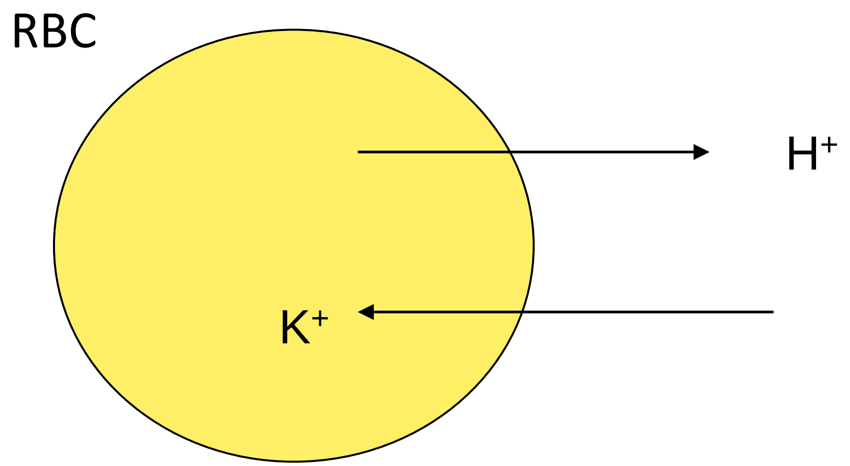
Causes of hypokalemia

-Excessive fluid loss (diarrhea, vomiting, diuretics)

-↑ Aldosterone promote Na reabsorption … K is excreted in its place (Cushing's syndrome = hyper aldosterone)

-Insulin IVs promote rapid cellular potassium uptake

Increased plasma pH (decreased Hydrogen ion)



K^+ moves into RBCs to preserve electrical balance, causing plasma potassium to decrease.
(Sodium also shows a slight decrease)

Hyperkalemia

Increased K concentration

Causes

- -IV'S or other increased intake
- -Renal disease – impaired excretion
- -Acidosis (Diabetes mellitus)
- -H+ competes with K+ to get into cells & to be excreted kidneys
  - -Decreased insulin promotes cellular K loss
  - -Hyperosomolar plasma (from ↑ glucose) pulls H_2O and potassium into the plasma

Calcium

Extracellular cation
Plays role in nerve impulse transmission.
Increases force of muscle contractions.
Functions as an enzyme co-factor in blood clotting.
Necessary for structure of bone and teeth.

Hypercalcemia [Ca > 5.8 mEq/L; Normal = 4.5-5.8 mEq/L]

- Causes
  - ✓ Hyperparathyroidism
  - ✓ Immobility
  - ✓ Increased vitamin D intake
  - ✓ Osteoporosis & osteomalacia [early stages]

Hypocalcemia [Ca < 4.5 mEq/L; Normal = 4.5-5.8 mEq/L]

- Causes
  - ✓Acute pancreatitis
  - ✓Diarrhea
    - ✓Hypoparathyroidism
  - ✓Lack of vitamin D In the diet
  - ✓Long-term steroid therapy

Magnesium

Intracellular cation.
Activates (ATP-ase) the primary energy source for the sodium potassium pump.
Plays important role in the relaxation of smooth muscle.
Stabilizes cardiac muscle cells - decreases fibrillation threshold.

Hyermagnesemia [Mg > 3.0 mEq/L; Normal = 1.5-3.0 mEq/L]

- ❑Causes
  - ✓Renal insufficiency, dehydration
  - ✓Excessive use of Mg-containing antacids or laxatives

Hypomagnesemia [Mg < 1.50 mEq/L; Normal = 1.5-3.0 mEq/L]

- ❑Causes
  - ✓Low intake of Mg in the diet
  - ✓Prolonged diarrhea
  - ✓Massive diuresis
  - ✓Hypoparathyroidism

Chloride

Chloride - the major anion of extracellular fluid

Chloride moves passively with Na^+ or against HCO_3^- to maintain neutral electrical charge

Chloride usually follows Na (if one is abnormal, so is the other)

Function **- *not completely known***

- ***body hydration***
- ***osmotic pressure***
- ***electrical neutrality & other functions***

Regulation via diet and kidneys

In the kidney, Cl is reabsorbed in the renal proximal tubules, along with sodium.

Deficiencies of either one limits the reabsorption of the other.

Normal values

Serum – ***100 -110 mEq/L***

Hypochloremia

Decreased serum Cl

- ***loss of gastric HCl***
- ***salt loosing renal diseases***
- ***metabolic alkalosis;***
  - ***increased HCO_3^- & decreased Cl^-***

Hyperchloremia

Increased serum Cl

dehydration (relative increase)

excessive intake (IV)

congestive heart failure

renal tubular disease

metabolic acidosis

decreased HCO_3^- & increased Cl^-

Bicarbonate

Principle buffer of body pH. (extracellular)
Neutralizes acids.
Plays important role in acid / base balance.
Acts as chemical sponge to soak up Hydrogen ions.
(Acidic metabolic waste) For every one Hydrogen ion twenty bicarbonate ions are released to maintain balance.

Carbon dioxide/bicarbonate –

* the major anion of intracellular fluid

2nd most important anion (2nd to Cl)

Note: most abundant <u>intra-</u>cellular anion

2nd most abundant <u>extra-</u>cellular

Total plasma CO_2 =

$$HCO_3^- + H_2CO_3^- + CO_2$$

HCO_3^- (carbonate ion) accounts for 90% of total plasma CO_2

$H_2CO_3^-$ carbonic acid (bicarbonate)

Regulation:

Bicarbonate is regulated by secretion / reabsorption of the renal tubules

Acidosis : ↓ renal excretion

Alkalosis : ↑ renal excretion

Kidney regulation requires the enzyme carbonic anhydrase - which is present in renal tubular cells & RBCs

carbonic anhydrase

Reaction: $CO_2 + H_2O \leftrightharpoons H_2CO_3 \rightarrow H^+ + HCO_3^-$

Normal values

Total Carbon dioxide (venous) – @ 22-30 mmol/L

includes bicarb, dissolved & undissociated H_2CO_3 - carbonic acid (bicarbonate)

Bicarbonate ion (HCO_3^-) – 22-26 mEq/L

Phosphate

Phosphate ($H_2PO_4^-$, HPO_4^{2-}, PO_4^{3-})

- Important ICF anions; plasma 1.7-2.6 mEq/liter
  - most (85%) is stored in bone as calcium salts
  - also combined with lipids, proteins, carbohydrates, nucleic acids (DNA and RNA), and high energy phosphate transport compound
  - important acid-base buffer in body fluids

Regulation - regulated in an inverse relationship with Ca^{2+} by PTH and Calcitonin

Homeostatic imbalances

Phosphate concentrations shift oppositely from calcium concentrations and symptoms are usually due to the related calcium excess or deficit