# Fluids, Electrolytes and Acid-Base Balance

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### Objectives

- Define normal ranges of electrolytes
- Compare/contrast intracellular, extracellular, and intravascular volumes
- Outline methods of determining fluid and acid/base balance
- Describe the clinical manifestations of various electrolyte imbalances.

## Normal Plasma Ranges of Electrolytes

#### Cations

- Sodium
- Potassium
- Calcium
- Magnesium
- Anions
  - Chloride
  - Bicarbonate
  - Phosphate
  - Sulfate
  - Organic Acids (Lactate)
  - Total Protein

Concentration

- 135-145 mEq/L
- 3.5-5.0 mEq/L
- 8.0-10.5 mEq/L
- 1.5-2.5 mEq/L
- 95-105 mEq/L
- 24-30 mEq/L
- 2.5-4.5 mEq/L
- 1.0 mEq/L
- 2.0 mEq/L
- 6.0-8.4 mEq/L

### Normal Ranges of Electrolytes

#### Sodium (Na<sup>+</sup>)

- Range 135 145 mEq/L in serum
- Total body volume estimated at 40 mEq/kg
- 1/3 fixed to bone, 2/3 extracellular and available for trans-membrane exchange
- Normal daily requirement 1-2 mEq/kg/day
- Chief extracellular cation

### Normal Ranges of Electrolytes

#### Potassium (K<sup>+</sup>)

- Range 3.5 5.0 mEq/L in serum
- Total body volume estimated at 50 mEq/kg
- 98% intracellular
  - concentration of 150 mEq/L
  - extracellular concentration of 70 mEq/L
- Normal daily requirement 0.5 0.8 mEq/kg/day
- Chief intracellular cation

### Normal Ranges of Electrolytes

#### Intracellular v Extracellular

- Electrolyte composition is different
  - Intracellular  $K^+$ ,  $Mg^+$ ,  $PO_4^-$ ,  $SO_4^-$ , and proteins
  - Extracellular Na<sup>+</sup>, Ca<sup>+</sup>, Mg<sup>+</sup>, Cl<sup>-</sup>, HCO<sub>3</sub><sup>-</sup> and lactate
- Compositions of ions are maintained
  - selective permeability of cell membranes
  - active ion pumps
- Movement of water is passive
  - colloid osmotic gradients intravascular v interstitial spaces (extracellularly)
  - osmolar gradients intracellularly v extracellularly

### Fluid Balance

Calculation of Osmolarity  $\Box Osm = 2 \times [Na_s] + [glu / 18] + [BUN / 2.8]$ ■ Normal osmolarity is 280-300 mOsm/L ■ Na<sup>+</sup> resorption and excretion are the driving forces for osmolarity Calculating TBW deficit • TBWD males =  $[(140 - SNa^+) \ge 0.6 \ge IBW (kg)]/140$ • TBWD females =  $[(140 - SNa^+) \ge 0.5 \ge IBW (kg)]/140$ 

### Fluid Balance

Here's a trick

For every 3.5 mEq the Na<sup>+</sup> is over 140, there is an estimated free water deficit of 1 L.

## Normal Physiology

#### Total body water

- 60% IBW of males
- 50-55% IBW of females
  - directly related to muscle mass (70% water)
  - inversely related to fat content (10% water)
  - This is why witches float

#### **Compartments**

- Intracellular
- Extracellular
- Interstitial





**Body Fluid Compartments** 2/3 (65%) of TBW is intracellular (ICF) ■ 1/3 extracellular water ■ 25 % interstitial fluid (ISF) ■ 5-8 % in plasma (IVF intravascular fluid) ■ 1-2% in transcellular fluids – CSF, intraocular fluids, serous membranes, and in GI, respiratory and urinary tracts (third space)

## Normal Physiology



 Intravascular water
 Interstitial water
 Intracellular water



## Normal Physiology

#### Two main compartments

- Intracellular
  - 2/3 of TBW
  - 40% body weight
- Extracellular
  - Intravascular and Interstitial compartments
  - 1/3 of TBW
  - 20% body weight



 Intravascular water
 Interstitial water
 Intracellular water



- Fluid compartments are separated by membranes that are freely permeable to water.
- Movement of fluids due to:
  - hydrostatic pressure
  - osmotic pressure
- Capillary filtration (hydrostatic) pressure
- Capillary colloid osmotic pressure
- Interstitial hydrostatic pressure
- Tissue colloid osmotic pressure

### Cell in Isotonic Solution



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### Cell in a hypertonic solution



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### Cell in a hypotonic solution



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### Balance

- Fluid and electrolyte homeostasis is maintained in the body
  - Neutral balance: input = output
  - Positive balance: input > output
  - Negative balance: input < output</p>

Solutes – dissolved particles Electrolytes – charged particles Cations – positively charged ions  $\blacksquare Na^+, K^+, Ca^{++}, H^+$ Anions – negatively charged ions  $\square Cl^-, HCO_3^-, PO_4^{3-}$ Non-electrolytes - Uncharged ■Proteins, urea, glucose, O<sub>2</sub>, CO<sub>2</sub>

Body fluids are:
Electrically neutral
Osmotically maintained
Specific number of particles per volume of fluid

### Homeostasis maintained by:

Ion transport

■ Water movement

Kidney function

### **Basic Definitions**

- MW (Molecular Weight) = sum of the weights of atoms in a molecule
- mEq (milliequivalents) = MW (in mg)/ valence mOsm (milliosmoles) = number of particles in a solution

Movement of body fluids "Where sodium goes, water follows."

**Diffusion** – movement of particles down a concentration gradient.

**Osmosis** – diffusion of water across a selectively permeable membrane

Active transport – movement of particles up a concentration gradient ; requires energy



## Normal Physiology

Na<sup>+</sup> resorption secondary to aldosterone
Occurs in distal convoluted tubules
Active exchange for K<sup>+</sup> and H<sup>+</sup> ions
Maintains extracellular volume and osmolarity
Water resorption secondary to antidiuretic hormone

Occurs in collecting ducts

Modulated by intracranial osmoreceptors and atrial stretch receptors

### Normal Physiology Na<sup>+</sup> modulation

Renal perfusion decreases

JG apparatus secretes renin

Renin secretion ceases

Renal perfusion increases



Renin cleaves angiotensinogen to angiotensin I

> Angiotensin I converted to II by ACE

Extracellular volume expansion as water follows Na<sup>+</sup> Angiotensin II stimulates aldosterone secretion from adrenal cortex

Aldosterone increases Na<sup>+</sup> resorption in exchange for K<sup>+</sup> in DCT



Normal Physiology Na+ modulation

Result:

increased water consumption increased water conservation Increased water in body increased volume and decreased Na+ concentration Dysfunction and/or Trauma

Leads to:

Decreased amount of water in body Increased amount of Na<sup>+</sup> in the body Increased blood osmolality Decreased circulating blood volume

**Regulation of body water** ■ ADH – antidiuretic hormone + thirst Decreased amount of water in body ■ Increased amount of Na+ in the body Increased blood osmolality Decreased circulating blood volume Stimulate osmoreceptors in hypothalamus ADH released from posterior pituitary Increased thirst

### Normal Physiology Free H<sub>2</sub>O modulation

Intracranial osmoreceptors detect increased plasma osmolarity

Plasma osmolarity increases

Renal collecting ducts become less permeable to water



Adenohypophysis (posterior pituitary) secretes ADH

Renal collecting ducts become more permeable to water

Adenohypophysis ceases ADH secretion

Plasma osmolarity decreases

Intracranial osmoreceptors detect decreased osmolarity

### Acid/Base Balance

- The management of Hydrogen ions
   measured as pH
   maintained at 7.4 +/- 0.05
- Three mechanisms (differing effective intervals)
   buffering systems in plasma
   ventilatory changes for CO<sub>2</sub> excretion
   Renal tubular excretion of Hydrogen ions

### Acid/Base Balance

Henderson-Hasselbalch
 Remember

 H<sub>2</sub>O + CO<sub>2</sub> = H<sub>2</sub>CO<sub>3</sub> = HCO<sub>3</sub><sup>-</sup> + H<sup>+</sup>

 $pH = 6.1 + \log[HCO_3^{-}]/0.03 \times PaCO_2$ 

or

 $[H^+] = 24 \text{ x PaCO}_2 / [HCO_3^-]$ 

### Acid/Base Balance Extracellular Regulation

Pulmonary regulation of PaCO<sub>2</sub> and renal tubular regulation of HCO<sub>3</sub><sup>-</sup> are important determinants of extracellular pH.

Basically, the pH is determined by the ratio of  $[HCO_3^-/H_2CO_3]$ 

Normally 20:1 (7.40)
As one increases, the other increases to re-establish the 20:1 ratio

### Acid/Base Balance Intracellular Regulation

#### Intracellular buffering

Excessive CO<sub>2</sub> retention or excretion
50% of fixed acid loads (lactate)
95% of hydrogen ion changes
Reciprocal K<sup>+</sup> ion exchange
Alkalosis

H<sup>+</sup> moves extracellularly
K<sup>+</sup> moves intracellularly
Acidosis
H<sup>+</sup> moves extracellularly
K<sup>+</sup> moves extracellularly
K<sup>+</sup> moves extracellularly

This can have significant clinical effect

This can have significant clinical effect, especially regarding myocardial function

### Fluids and Electolytes in the Postoperative Period

- Maintenance
- Resuscitation
- Replacement of Losses

### Fluids and Electolytes in the Postoperative Period

#### Maintenance

Normal daily outputs • Urine = 12-15 cc/kg• Stool = 3 cc/kg• Sweat = 1.5 cc/kg• Respiratory and Skin insensible losses = 10 cc/kg■ Increased by 8%/degree F for fever Normal daily endogenous input • Oxidation of carbohydrates and fat = 3 cc/kgStandard nomograms estimate daily requirements





## Fluids and Electolytes in the Postoperative Period

- Third Space Fluid Losses
  - Fluids sequestered into extracellular and intersitial spaces
    - Peritonitis
    - Intestinal obstruction
    - Soft tissue inflammation/edema
    - Traumatic losses
  - Evidence of diminished volume
    - Hemodynamic changes
      - Tachycardia
      - Narrowed pulse pressures
      - Hypotension



The accumulation of fluid within the interstitial spaces.

Leads to: increased hydrostatic pressure lowered plasma osmotic pressure increased capillary membrane permeability lymphatic channel obstruction Hydrostatic pressure increases **Venous obstruction:** thrombophlebitis (inflammation of veins) hepatic obstruction tight clothing on extremities prolonged standing Salt or water retention congestive heart failure renal failure

### Decreased plasma osmotic pressure

↓ plasma albumin (liver disease or protein malnutrition)

plasma proteins lost in :

glomerular diseases of kidney

hemorrhage, burns, open wounds and cirrhosis of liver Increased capillary permeability: Inflammation

immune responses

Lymphatic channels blocked: surgical removal infection involving lymphatics lymphedema

### Electrolyte imbalances: Sodium

Hypernatremia (high levels of sodium)
Plasma Na+ > 145 mEq / L
Due to ↑ Na + or ↓ water
Water moves from ICF → ECF
Cells dehydrate



### Hypernatremia

### Causes

Hypertonic IV soln. Oversecretion of aldosterone ■ Loss of pure water ■Long term sweating with chronic fever  $\blacksquare$  Respiratory infection  $\rightarrow$  water vapor loss ■Diabetes – polyuria Insufficient intake of water (hypodipsia)

Clinical manifestations of Hypernatremia

#### Thirst

- Lethargy
- Neurological dysfunction due to dehydration of brain cells
- Decreased vascular volume

## Treatment of Hypernatremia

Lower serum Na+
 Isotonic salt-free IV fluid
 Oral solutions preferable

## Hyponatremia

Overall decrease in Na+ in ECF Two types: depletional and dilutional Depletional Hyponatremia Na+loss: diuretics, chronic vomiting Chronic diarrhea Decreased aldosterone Decreased Na+ intake

#### Dilutional Hyponatremia:

- Renal dysfunction with <sup>↑</sup> intake of hypotonic fluids
- Excessive sweating→ increased thirst → intake of excessive amounts of pure water
- Syndrome of Inappropriate ADH (SIADH) or oliguric renal failure, severe congestive heart failure, cirrhosis all lead to:
  - Impaired renal excretion of water
- Hyperglycemia attracts water

### Clinical manifestations of Hyponatremia

Neurological symptoms

 Lethargy, headache, confusion, apprehension, depressed reflexes, seizures and coma

Muscle symptoms

Cramps, weakness, fatigue

Gastrointestinal symptoms

■ Nausea, vomiting, abdominal cramps, and diarrhea

Tx – limit water intake or discontinue meds

## Hypokalemia

- Serum K<sup>+</sup> < 3.5 mEq /L</li>
  Beware if diabetic

  Insulin gets K<sup>+</sup> into cell
  Ketoacidosis H<sup>+</sup> replaces K<sup>+</sup>, which is lost in urine
- $\beta$  adrenergic drugs or epinephrine

## **Causes of Hypokalemia**

Decreased intake of K<sup>+</sup> ■ Increased K<sup>+</sup> loss Chronic diuretics ■ Acid/base imbalance ■ Trauma and stress ■ Increased aldosterone Redistribution between ICF and ECF

## Clinical manifestations of Hypokalemia

Neuromuscular disorders

- Weakness, flaccid paralysis, respiratory arrest, constipation
- Dysrhythmias, appearance of U wave
- Postural hypotension
- Cardiac arrest
- Others table 6-5
- Treatment-
  - Increase  $K^+$  intake, but slowly, preferably by foods

## Hyperkalemia

- Serum K+ > 5.5 mEq / L
- Check for renal disease
- Massive cellular trauma
- Insulin deficiency
- Addison's disease
- Potassium sparing diuretics
- Decreased blood pH
- Exercise causes K+ to move out of cells

## Clinical manifestations of Hyperkalemia

- Early hyperactive muscles, paresthesia
- Late Muscle weakness, flaccid paralysis
- Change in ECG pattern
- Dysrhythmias
- Bradycardia , heart block, cardiac arrest

## Treatment of Hyperkalemia

- If time, decrease intake and increase renal excretion
- Insulin + glucose
- Bicarbonate
- Ca<sup>++</sup> counters effect on heart

### **Calcium Imbalances**

Most in ECF Regulated by: Parathyroid hormone ■↑Blood Ca<sup>++</sup> by stimulating osteoclasts ■↑GI absorption and renal retention Calcitonin from the thyroid gland Promotes bone formation ■↑ renal excretion

## Hypercalcemia

#### Results from:

- Hyperparathyroidism
- Hypothyroid states
- Renal disease
- Excessive intake of vitamin D
- Milk-alkali syndrome
- Certain drugs
- Malignant tumors hypercalcemia of malignancy
   Tumor products promote bone breakdown
   Tumor growth in bone causing Ca<sup>++</sup> release

## Hypercalcemia

Usually also see hypophosphatemia

Effects:

- Many nonspecific fatigue, weakness, lethargy
- Increases formation of kidney stones and pancreatic stones
- Muscle cramps
- Bradycardia, cardiac arrest

Pain

GI activity also common
 Nausea, abdominal cramps
 Diarrhea / constipation
 Metastatic calcification

## Hypocalcemia

- Hyperactive neuromuscular reflexes and tetany differentiate it from hypercalcemia
- Convulsions in severe cases
- Caused by:
  - Renal failure
  - Lack of vitamin D
  - Suppression of parathyroid function
  - Hypersecretion of calcitonin
  - Malabsorption states
  - Abnormal intestinal acidity and acid/ base bal.
  - Widespread infection or peritoneal inflammation

## Hypocalcemia

Diagnosis:
Chvostek's sign
Trousseau's sign
Treatment
IV calcium for acute
Oral calcium and vitamin D for chronic

