

## Chapter 27: WATER, ELECTROLYTES, AND ACID-BASE BALANCE

### I. RELATED TOPICS

Integumentary system	Capillary dynamics
Synovial fluid	Lymph circulation
Cerebrospinal fluid	Edema
Aqueous humor	Metabolic water
Digestive juices	Diabetes
Feces	Thermoregulation
Blood flow	Renal dynamics
	Lactation

Systems which control fluid balance?

### II. THE FLUID COMPARTMENTS

#### A. Body Fluid Distribution (Table 27.1, p. 986 [902])

Total body water	_____				
Intracellular fluid	_____				
Extracellular fluid	_____	_____	_____	_____	_____
		Plasma		Interstitial fluid	

#### B. Intracellular Fluid

1. Location
2. Volume - \_\_\_\_\_ % body weight
3. Major electrolytes (charged ions or molecules)
  - a. Cations (+):             $K^+$              $Mg^{++}$
  - b. Anions (-):             $HPO_4^{-2}$             Protein<sup>-</sup>

C. Extracellular Fluid

1. Plasma

- a. Volume: \_\_\_\_\_ % body weight
- b. Major cation:
- c. Major anions:

2. Interstitial fluid

- a. Location:
- b. Volume: \_\_\_\_\_ % body weight
- c. Major cation
- d. Major anion
- e. Differs how from plasma?

3. Lymph

- a. Location
- b. Composition

**III. REGULATION OF WATER CONTENT**

A. Water intake and loss (Table 27.3, p. 987 [27.7, p. 912])

1. Sources of water

Fluid  
Food  
Metabolic H<sub>2</sub>O

2. Water loss routes

Urine  
Lungs  
Skin  
Feces  
Other  
Abnormal water loss

- B. Stimulation of thirst (Fig. 27.1, p. 987)
1. Increased osmolality of plasma affects hypothalamus --> thirst
  2. Low blood pressure affects baroreceptors --> thirst
  3. Renin --> --> --> angiotensin II --> thirst
- C. Regulation of extracellular fluid osmolality: Antidiuretic hormone (ADH) (Fig. 27.1, p. 987; 27.2, p. 989) See Notebook, p. 85.

D. Regulation of extracellular fluid volume:

1. Renin-angiotensin system (Fig. 21.40, p. 759; p. 27.4a, p. 992)

Decreased blood pressure

\_\_\_\_\_ blood pressure

Renin (juxtaglomerular cells)

\_\_\_\_\_ blood volume

Angiotensinogen ---> angiotensin I  
(inactive plasma protein)

\_\_\_\_\_ water reabsorption

Angiotensin II --> aldosterone release --->  
from adrenal cortex

\_\_\_\_\_ Na<sup>+</sup> reabsorption  
from nephrons

\_\_\_\_\_ vasoconstriction

\_\_\_\_\_ thirst

\_\_\_\_\_ salt appetite

\_\_\_\_\_ ADH secretion

2. Atrial natriuretic hormone (Fig. 27.4b, p. 992)

a. Source

b. Stimulus for release

c. Function

- E. Regulation of intracellular fluid (Fig. 27.5, p. 993 [27.1, p. 902])
  - 1. Major extracellular ion
  - 2. Role of osmosis

#### IV. ROLE OF ELECTROLYTES IN FLUID BALANCE

- A. Specific electrolytes
  - 1. Sodium ion ( $\text{Na}^+$ )
    - a. Location
    - b. Functions
    - c. Excretion
    - d. Control (Fig. 27.4, p. 992 [27.2, p. 904])

#### *Hypernatremia/ Hyponatremia*

- 2. Chloride ion ( $\text{Cl}^-$ )
  - a. Location
  - b. Functions
  - c. Control

3. Potassium ion ( $K^+$ )
  - a. Location
  - b. Function
  - c. Control (Fig. 27.6, p. 998)

*Hyperkalemia*

- d. If aldosterone is greatly reduced, increased  $[K^+]$  causes death. Why?

4. Calcium ion ( $Ca^{++}$ )
  - a. Location
  - b. Functions
  - c. Control (Fig. 27.7, p. 999 [ 27.3, p. 908])
    - i. PTH \_\_\_\_\_ blood  $Ca^{++}$
    - ii Calcitonin \_\_ blood  $Ca^{++}$

*Hypercalcemia/ Hypocalcemia*

- d. Role of vitamin D

5. Magnesium
  - a. Location
  - b. Function
  - c. Control (Fig. 27.8, p. 1001)

*Hypomagnesemia*

6. Phosphate ions ( $\text{HPO}_4^-$ )
  - a. Location
  - b. Function
  - c. Control (Fig. 27.9, p. 1002)

C. Causes of electrolyte imbalances:

1. Increased sweating
2. Vomiting
3. Diarrhea
4. Diuretic drugs
5. Inadequate nutrient intake
6. Renal insufficiency
7. Endocrine disorders

D. Note that these also can cause acid-base imbalances

## V. ACID-BASE BALANCE

A. Significance: Blood pH = 7.35 - 7.45

a. 7.0 - 7.35: acidosis                      Effect:

b. 7.45 - 7.8: alkalosis                      Effect:

c. NOTE: Intracellular pH = 6.0 - 7.4

B. Causes of imbalances (Table A, p. 1009)

1. Gastric vomiting

2. Diarrhea

3. Intestinal vomiting

4. Kidney dysfunction

5. Respiratory dysfunction

6. Ketosis

7. Ingestion of acidic or alkaline substances

C. Definitions

1. Acid: Electrolyte dissociating into  $H^+$  and anion<sup>-</sup>

a. Strong acid --> total dissociation

b. Weak acid --> partial dissociation

2. Base: Anion which can bind an  $H^+$

a. Weak base: weak  $H^+$  acceptor

b. Ex.:

3. Acids + bases --> salt +  $H_2O$



2. Respiratory system (Fig. 27.12, p. 1006)
- i. Extracellular (buffers carbonic acid in \_\_\_\_\_ blood)
  - a. Relatively rapid (minutes, hours); more effective than buffers  
Why?
  - b.  $\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-$
  - c. Decreased respiration --> \_\_\_\_\_ in  $\text{CO}_2$   
Increased respiration --> \_\_\_\_\_ in  $\text{CO}_2$
  - d. Increased  $\text{CO}_2$  --> \_\_\_\_\_  $[\text{H}^+]$  --> \_\_\_\_\_ pH  
Decreased  $\text{CO}_2$  --> \_\_\_\_\_  $[\text{H}^+]$  --> \_\_\_\_\_ pH
  - e. Control: decreased pH --> \_\_\_\_\_ ventilation rate  
Increased pH --> \_\_\_\_\_ ventilation rate
  - f. Respiratory acidosis: pH \_\_\_\_\_
    - i. Cause: \_\_\_\_\_ ventilation  
or \_\_\_\_\_ respiratory exchange
    - ii. Effect
  - g. Respiratory alkalosis: pH \_\_\_\_\_
    - i. Cause: \_\_\_\_\_ ventilation
    - ii. Effect
  - b. Respiratory stresses which affect acid-base balance
    - i. High altitude
    - ii. Pneumonia
    - iii. Emphysema

iv. Hyperventilation

## 3. Kidneys

a. Slow acting but exact:

b.  $H^+$  eliminated as

i.  $Na^+ H_2 PO_4$

ii.  $NH_4^+ Cl^-$

in exchange for  $Na^+ HCO_3^-$

c. Local control: pH of cells of distal tubule and collecting duct

i. If blood and tissue pH is too low, they secrete  $H^+$  and reabsorb

\_\_\_\_\_.

ii. If blood and tissue pH is too high,  $H^+$  is not secreted and  $Na^+ HCO_3^-$  is not reabsorbed -->

d. If urine contains more acid, blood becomes \_\_\_\_\_ acid

e. If urine contains less acid, blood becomes \_\_\_\_\_ acid

f. Metabolic acidosis

i. Causes

ii. Effect

g. Metabolic alkalosis

i. Causes

ii. Effect:

See also "Clinical Focus: Acidosis and Alkalosis," pp. 1008-1009 [918-919].