

Renal Regulation of Body Fluid

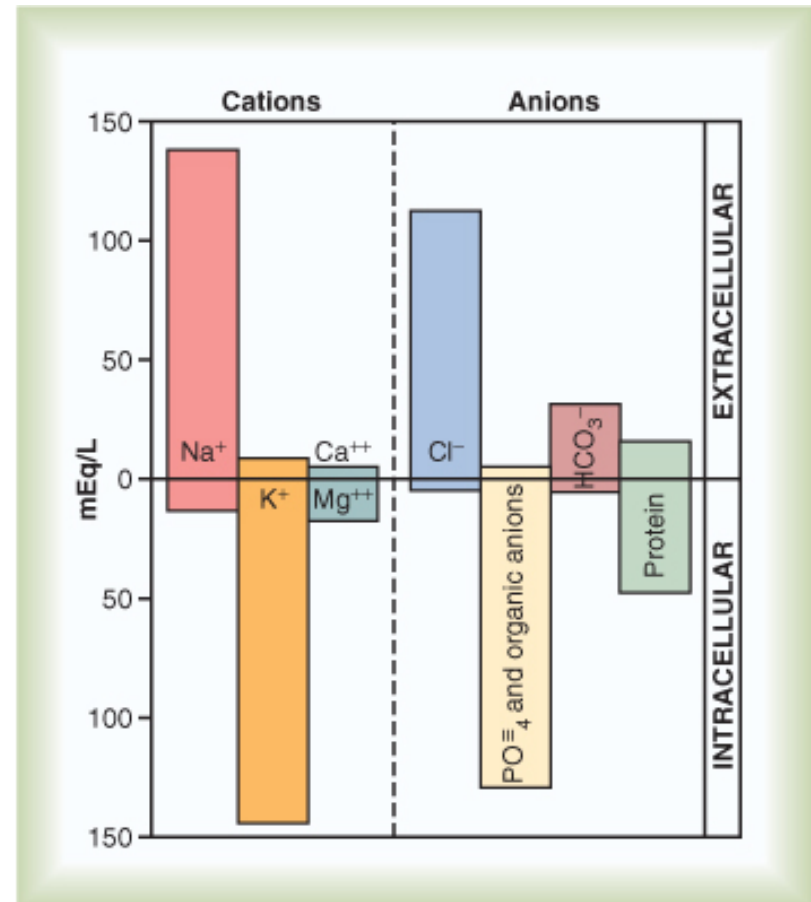
**Prof. Mona Soliman, MBBS, MSc, PhD
Head, Medical Education Department
Professor of Physiology
& Medical Education
College of Medicine
King Saud University**

Learning Objectives:

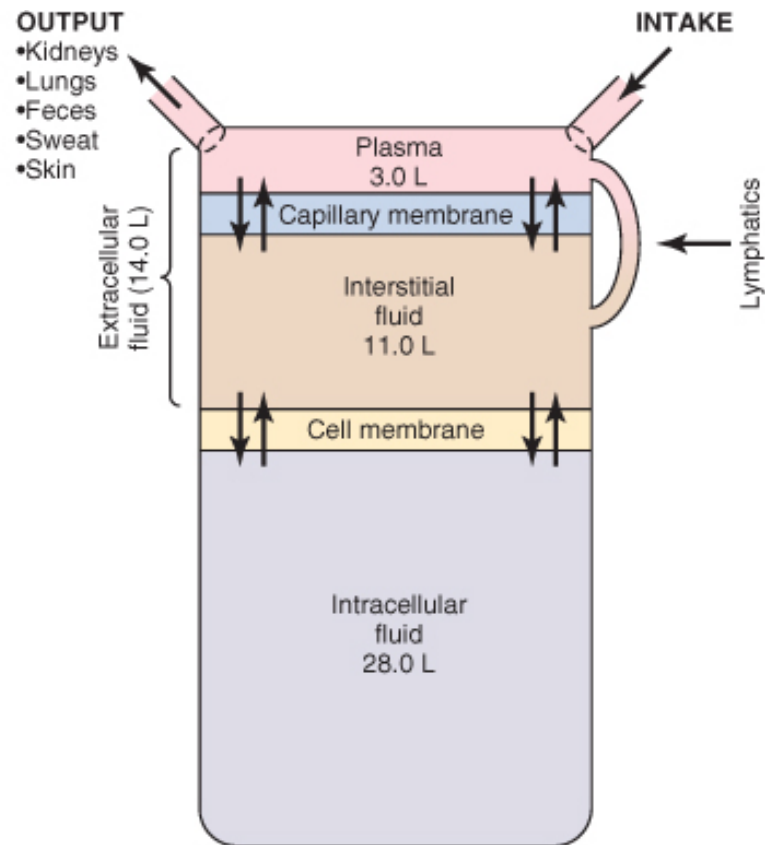
- Identify and describe the role of the Sensors and Effectors in the renal regulation of body fluid volume & osmolality
- Describe the role of the kidney in regulation of body fluid volume & osmolality
- Understand the role of ADH in the reabsorption of water and urea
- Identify the site and describe the influence of aldosterone on reabsorption of Na^+ in the late distal tubules.

Solute Overview: Intracellular vs. Extracellular

- Ionic composition very different
- Total ionic concentration very similar
- Total osmotic concentrations virtually identical



The major body fluid compartment

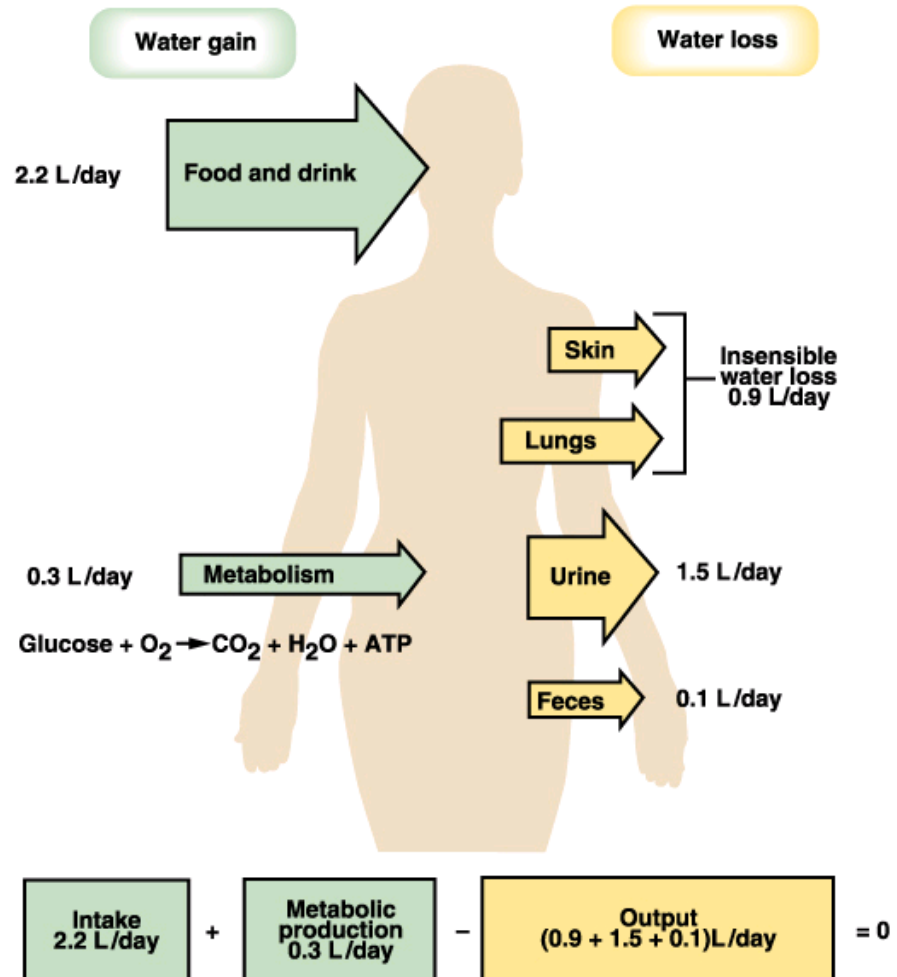


Regulation of volume & osmolality

- Body water balance must be maintained.
- Kidneys concentrate or dilute urine.
- To remain properly hydrated, water intake must equal water output.
- Increases in plasma osmolality trigger thirst and release of antidiuretic hormone (ADH)

Water Steady State:

- Amount ingested = amount eliminated.
- Pathological losses:
 - Vascular bleeding.
 - Vomiting.
 - Diarrhea.



Control of circulating volume

- **Volume sensors:**

- a) Vascular:

1. Low pressure sensors: Cardiac atria (ANP), pulmonary vasculature.
2. High pressure: carotid sinus, aortic arch and juxtaglomerular apparatus of the kidney.

- b) Central nervous system.

- c) Hepatic.

Control of circulating volume

- **Volume sensor signals/Mediators:**

A) Neural:

If pressure ↓

Renal sympathetics:

a) afferent & Efferent arterioles constrict

i) GRF ↓

ii) less Na⁺ filtered

iii) more Na⁺ absorbed by PCT

b) renin released

i) ↑ aldosterone

ii) ↑ angiotensin II

Control of circulating volume

B) Hormonal:

1) Renin-angiotensin-aldosterone system (\downarrow pressure):

- Renin secreted, by:
 - a) Sympathetic stimulation
 - b) \downarrow perfusion pressure
 - c) \downarrow Na^+ reaching **macula densa**
- Angiotensin II:
 - i) aldosterone release by adrenal cortex
 - \uparrow Na^+ reabsorption in TAL, DT, CD
 - ii) Vasoconstriction
 - iii) ADH release
 - iv) \uparrow Na^+ reabsorption in PCT

2) ANP:

From **atrial** myocytes

Released by stretch of atrium

⇒ ↑ NaCl & water excretion

Regulation of volume & osmolality

- If \uparrow water intake \Rightarrow hypoosmotic urine
dilute (~ 50 mOsm/kg)
large volume (up to 18 L/d!!)
- If \downarrow water intake \Rightarrow hyperosmotic urine
concentrated (up to 1200 mOsm/kg)
small volume (0.5 L/d)
- Renal water excretion mechanism(s) independent of solute excretion mechanism(s)
 \therefore allows water balance maintenance without damaging solute homeostasis (e.g. Na^+ , K^+)

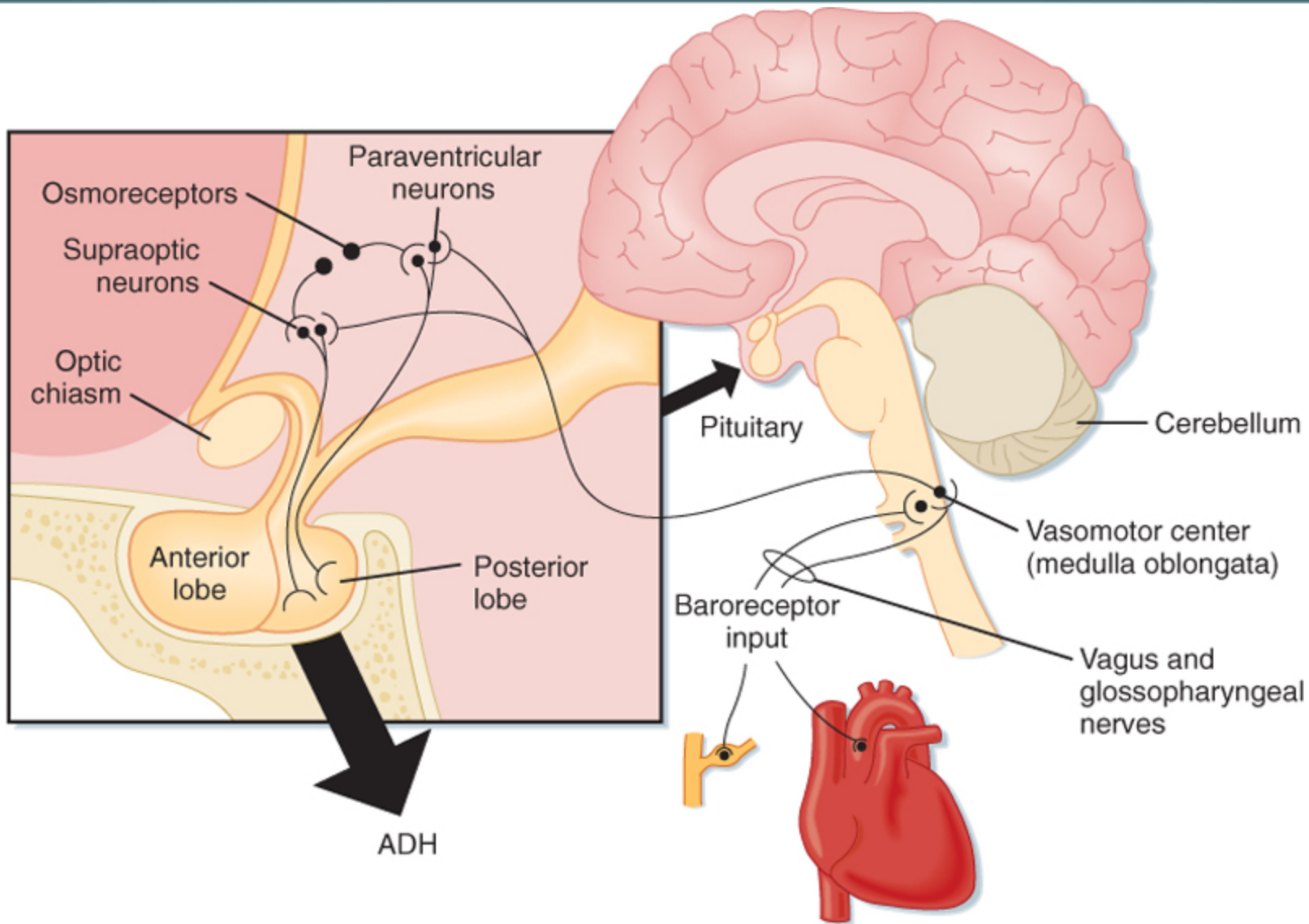
Antidiuretic hormone (ADH)/ Vasopressin

- It is synthesized in neuroendocrine cells located within the supraoptic and paraventricular nuclei of the hypothalamus
- The synthesized hormone is packaged in granules that are transported down the axon of the cell and stored in nerve terminals located in the neurohypophysis (posterior pituitary).
- prevents water loss
- small protein hormone (only 9 amino acids)
- fast acting, short half life in circulation
- ↑ thirst

Antidiuretic hormone (ADH)/ Vasopressin

- Factors influencing release:

- 1) Osmolality
 - 2) Haemodynamic factors
 - 3) Nausea → stimulates
 - 4) Atrial natriuretic peptide (ANP) → inhibits
 - 5) Angiotensin II → stimulates
- } Main physiological factors



Koeppen & Stanton: Berne and Levy Physiology, 6th Edition.
 Copyright © 2008 by Mosby, an imprint of Elsevier, Inc. All rights reserved

- A rough estimate of ECF osmolality can be obtained by doubling Plasma sodium concentration
- $145\text{mEq/l} \times 2 = 290$ (Normal 285-295 mOsm/kg H₂O)
- ∴ Sodium concentration gives best estimate of effective osmolality of ECF.
- In clinical situations glucose & urea concentrations (mmols) are also taken into account, useful in cases of patients with diabetes mellitus or chronic renal failure.

Osmolality

- Osmoreceptors in hypothalamus, outside blood-brain barrier.
- \uparrow osmolality \Rightarrow ADH release
- \downarrow blood volume \Rightarrow ADH release

ADH renal target

- Collecting duct cells only permeable to water in presence of ADH
- ADH causes \uparrow in **urea** permeability in inner medullary CD
- ADH stimulates reabsorption of NaCl by the thick ascending limb of Henle's loop and by the DCT and cortical segment of CD

Regulation of Water Intake

- The hypothalamic thirst center is stimulated:
 - By a decline in plasma volume of 10%–15%
 - By increases in plasma osmolality of 1–2%
 - Via baroreceptor input, angiotensin II, and other stimuli.

Regulation of Water Intake

- Thirst is quenched as soon as we begin to drink water
- Feedback signals that inhibit the thirst centers include:
 - Moistening of the mucosa of the mouth and throat
 - Activation of stomach and intestinal stretch receptors

Actions of Angiotensin II

1. Angiotensin II receptors are found on the zona glomerulosa cells of the adrenal cortex.
 - Activation of these receptors leads to an immediate and rapid increase in aldosterone secretion.
 - Aldosterone acts on the distal tubule and collecting duct to cause *sodium* retention.
 - This is likely to be an important mechanism for determining long-term sodium balance.

Actions of Angiotensin II

2. Vascular actions

- Angiotensin II is one of the most potent vasoconstrictors known.
- Constriction of vascular smooth muscle leads to a prompt rise in blood pressure.
- It plays an important role in maintaining vascular tone and blood pressure in volume depleted states, for example haemorrhage and fluid depletion.

Reference

- Guyton and Hall Textbook of Physiology
- Chapter 25 & 28

Thank You