

# Renal Regulation of ECV and osmolality

*Dr Sitelbanat Awadalla*

# Objectives

**At the end of this lecture student should be able to describe:**

- 1. Identify and describe the role of the Sensors and Effectors in the renal regulation of body fluid volume**
- 2. Identify and describe the role of the Sensors and Effectors in the renal regulation of body fluid osmolality**
- 3. Role of the kidney in volume regulation**
- 4. Role of the kidney in ECF osmolality**

# Renal regulation of Extra Cellular Volume

Is a reflex mechanism in which variables reflecting **total body sodium** and **ECV** are monitor by appropriate sensor (receptors)

Regulation of ECF volume = **Regulation of body Na<sup>+</sup>** = Regulation BP

Thus, regulation of **Na<sup>+</sup>** also dependent upon baroreceptors.

# Summary of Renal Regulation of ECV

- **Changes in ECV, Na and Pressure**
- **Sensor**
  - **Carotid sinus**
  - **Volume receptors (large vein, atria, intrarenal artery)**
- **Effectors**
  - **Renin/angiotensin, aldosterone**
  - **Renal sympathetic nerve**
  - **ANF**
  - **ADH**
- **Affecting**
  - **Urinary Na excretion**

# ECF volume Receptors

1. Central vascular sensors
  - **Low pressure receptors** (very important)
    - Cardiac atria
    - Pulmonary vasculature
  - **High pressure receptors** (less important)
    - Carotid sinus
    - Aortic arch
    - Juxtaglomerular apparatus (renal afferent arteriole)
2. Sensors in the CNS (less important)
3. Sensors in the liver (less important)

# 1. Renin-angiotensin Aldosterone

- Renin is released into plasma when plasma Na ↓
- Renin → angiotensinogen → Angiotensin I
- Angiotensin I → ACE → angiotensin II
- angiotensin II act on adrenal cortex → aldosterone secretion → ↑ Na reabsorption in distal & collecting duct of nephron

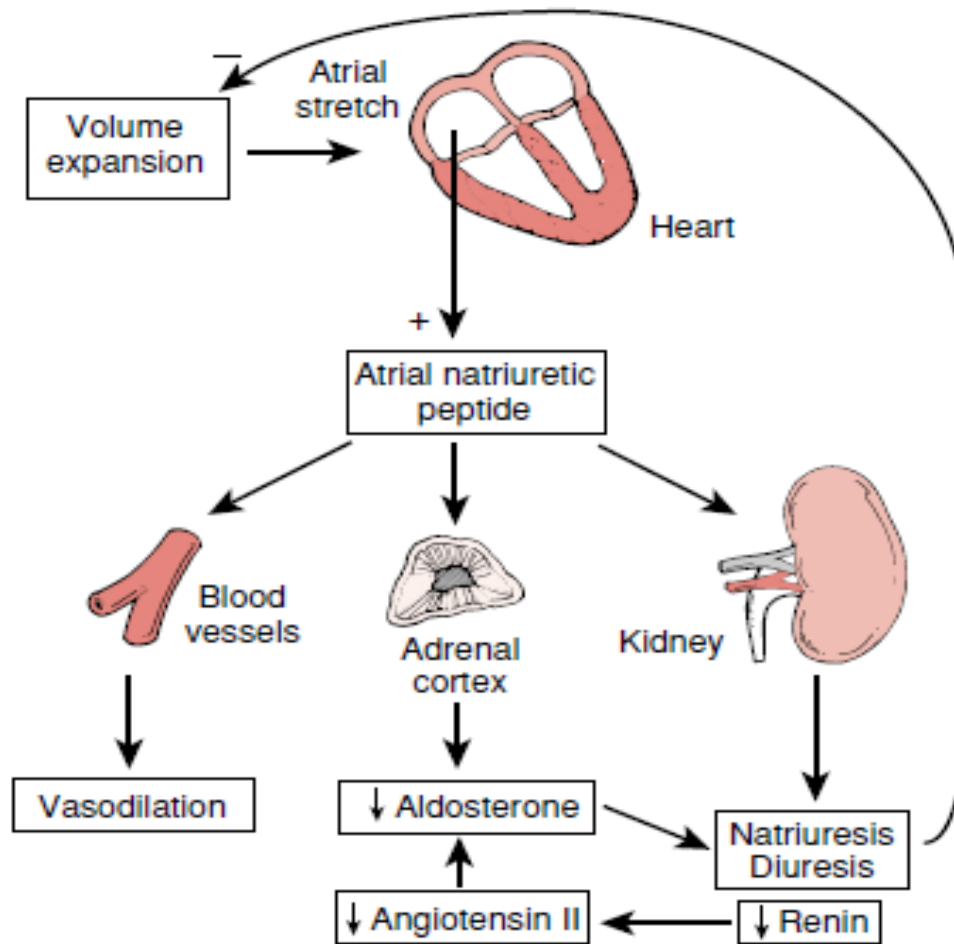
## 2. Renal Sympathetic

- **↓ ECV → ↑ renal sympathetic activity → stimulate Na absorption by direct tubular effect mediated through  $\alpha$ -receptors on renal tubules (mainly PCT) to correct for low ECV**

# 3. ATRIAL NATRIURETIC PEPTIDE (ANP)

- **↑ ECV → Stretch of Atria → release ANP → inhibit aldosterone release → ↓ sodium reabsorption by collecting duct**
- **↑ sodium excretion and water → correcting for the increase in ECV**
- **ANP can also inhibit Renin secretion**





**FIGURE 24.10** Atrial natriuretic peptide and its actions. ANP release from the cardiac atria is stimulated by blood volume expansion, which stretches the atria. ANP produces effects that bring blood volume back toward normal, such as increased  $\text{Na}^+$  excretion.

# 4. Antidiuretic hormone

- Increase of plasma osmolality → osmoreceptor → trigger the release of ADH.
- ADH → ↑ permeability of collecting duct to  $H_2O$  → ↑  $H_2O$  reabsorption → correction of hyperosmolality of blood.

## ADH release stimulated by

- ↑ Osmolarity
- ↓ Blood volume
- ↓ Blood pressure
- Drugs: Morphine; Nicotine; cyclophosphamide

# ECV and Urinary Sodium Excretion

- Regulation of urinary sodium excretion → regulation EC volume
- ↑ ECV or Sodium is corrected by ↑ urinary sodium excretion and water by:
  - **Renin – aldsterone**
  - **ANP**
  - **Sympathetic**
  - **ADH**

# Renal Regulation of ECF osmolality

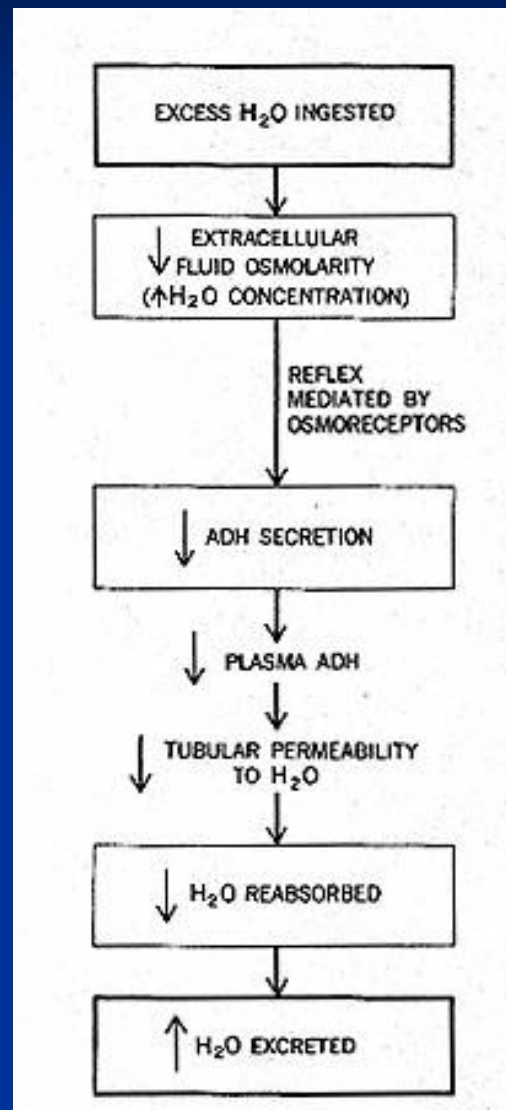
# Summary of Renal regulation of body fluid osmolality

- **Changes in ECF osmolarity**
- **Sensors**
  - **Hypothalamic osmoreceptors**
- **Effectors**
  - **ADH**
  - **thirst**
- **Affecting**
  - **Urine osmolaity**
  - **Water intake**

# Renal regulation of Extra Cellular Osmolality

Is a reflex mechanism in which a change in **plasma osmolality** is monitored by appropriate sensor (**osmoreceptors**)  
hypothalamus osmoreceptor

# 1. Osmoreceptor ADH Feedback System



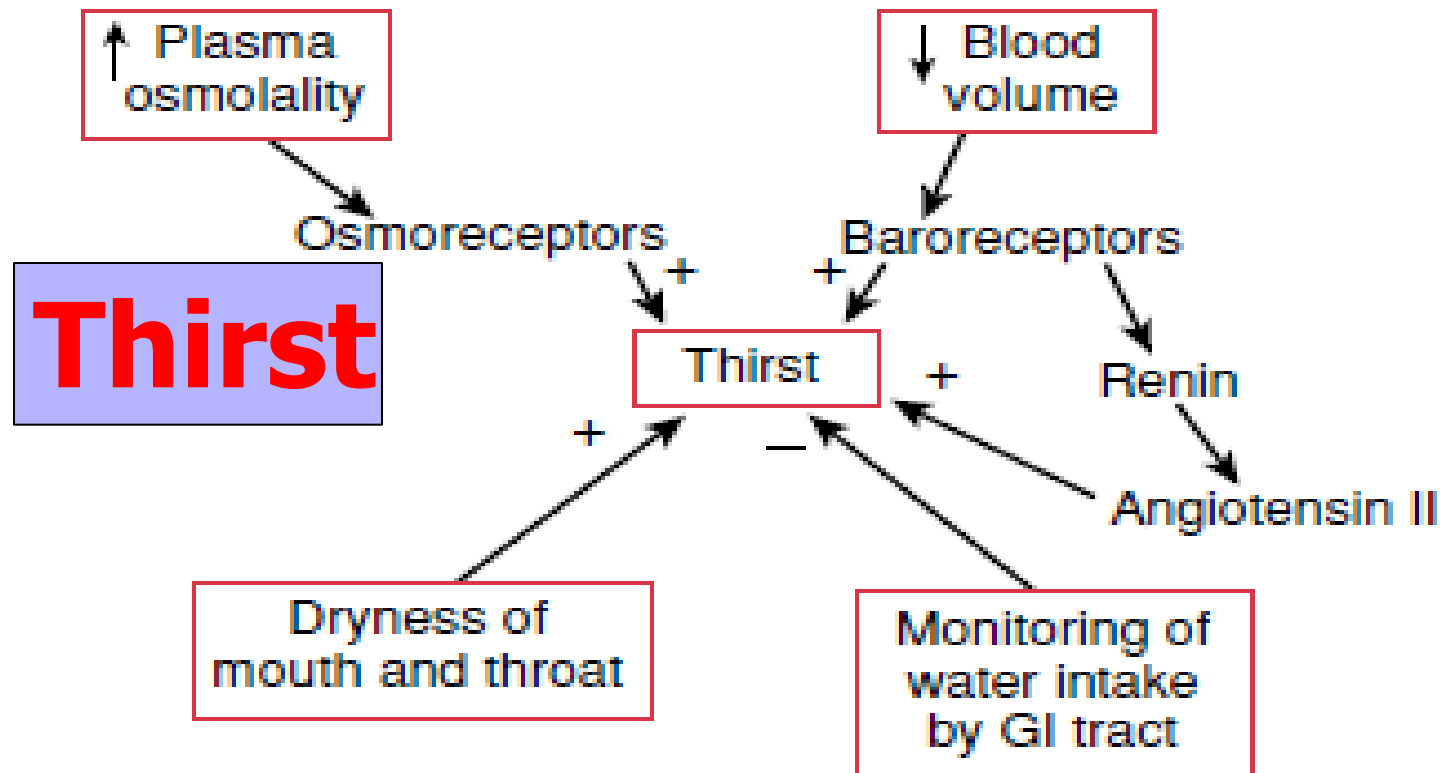
## 2. Role of Thirst in Controlling Extracellular Fluid Osmolarity

**Thirst sensation stimulated by:**

1. ↑ Osmolarity
2. ↓ Blood volume
3. ↓ Blood pressure
4. ↑ Angiotensin
5. Dryness of mouth



## 2. Thirst



**FIGURE 24.7** Factors affecting the thirst sensation. A plus sign indicates stimulation of thirst, the minus sign indicates an inhibitory influence.

# High water intake

- **Drop** in plasma osmolality
- inhibit ADH secretion
- Collecting impermeable to water
- excretion of large volume of urine
- **increases** plasma osmolality back to normal.

# Low water intake

1. **Increases** plasma osmolality
2. **Stimulate** ADH secretion
3. **Making** Collecting duct permeable to water
4. **Excretion** of small volume of urine
5. **Diluting** plasma and a **drop** in osmolality back to normal.
6. **Accompanied** by thirst sensation.

# Urine Osmolarity Regulation: Collecting Duct

**(a)** With maximal vasopressin, the collecting duct is freely permeable to water. Water leaves by osmosis and is carried away by the vasa recta capillaries. Urine is concentrated.

**(b)** In the absence of vasopressin, the collecting duct is impermeable to water and the urine is dilute.

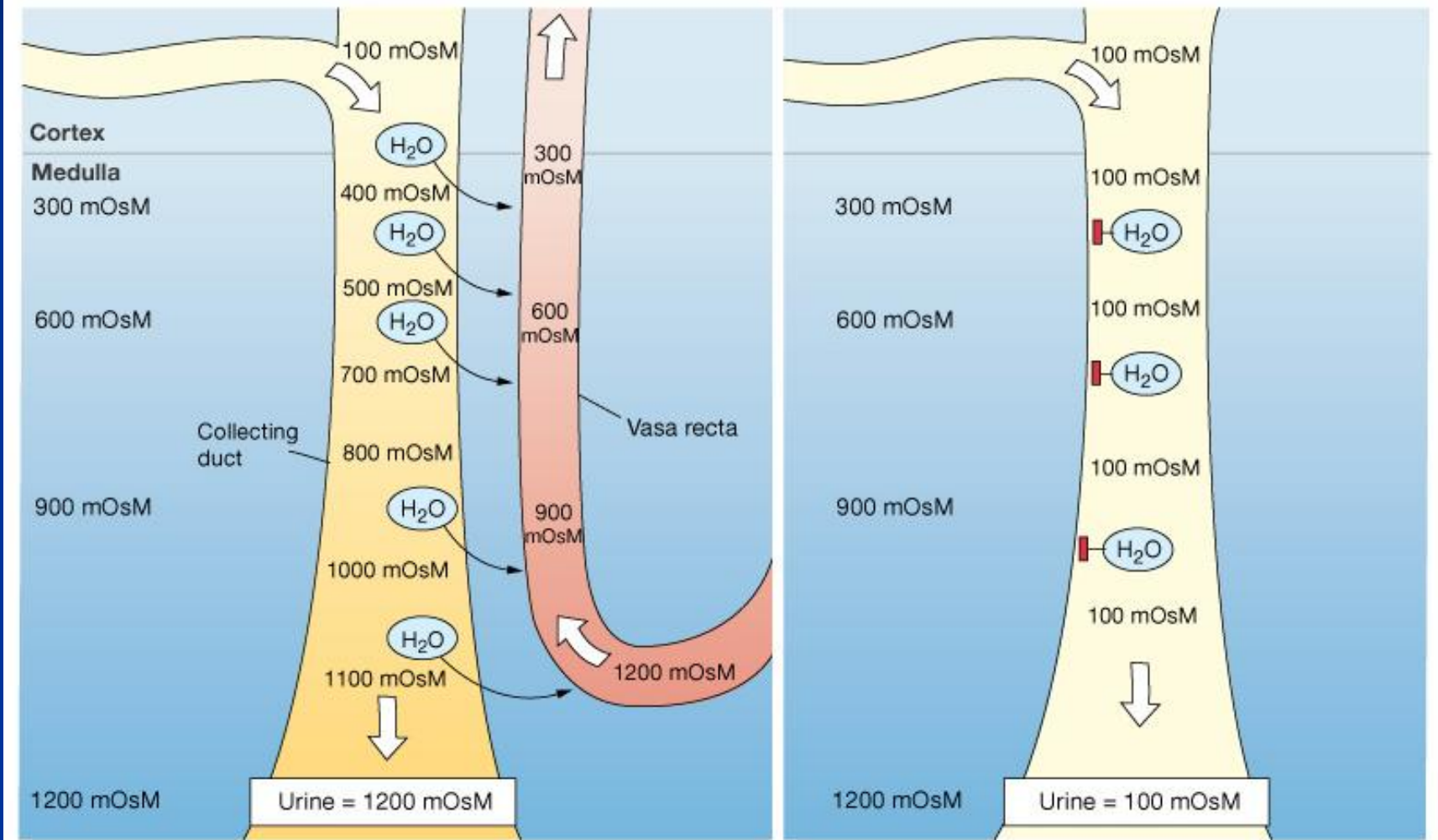


Figure 26-57. Water movement in the collecting duct in the presence and absence of