Intermediate & long-term regulation of ABP

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Objectives:

- By the end of this lecture the students are expected to:
- List the intermediate and long-term mechanisms regulating ABP.
- Explain the intermediate mechanisms in the regulation of ABP; Capillary fluid shift.
- Explains the role of the kidney in the long-term regulation of ABP.: the renin –angiotensin system and its components.

Intermediate mechanism

- Activated within 30 minutes up to hours.
- This effect lasts for long period as days.
- **Capillary fluid shift mechanism:**

Any time capillary pressure falls too low

- Fluid is absorbed by osmosis from the tissues into circulation

- Build up blood volume

- Increase PB toward normal

Capillary Hydrostatic Pressure

- High on arterial side – bulk flow out
- Low on venous side – bulk flow in
- Fenestrations &/or leaky joints speed exchange

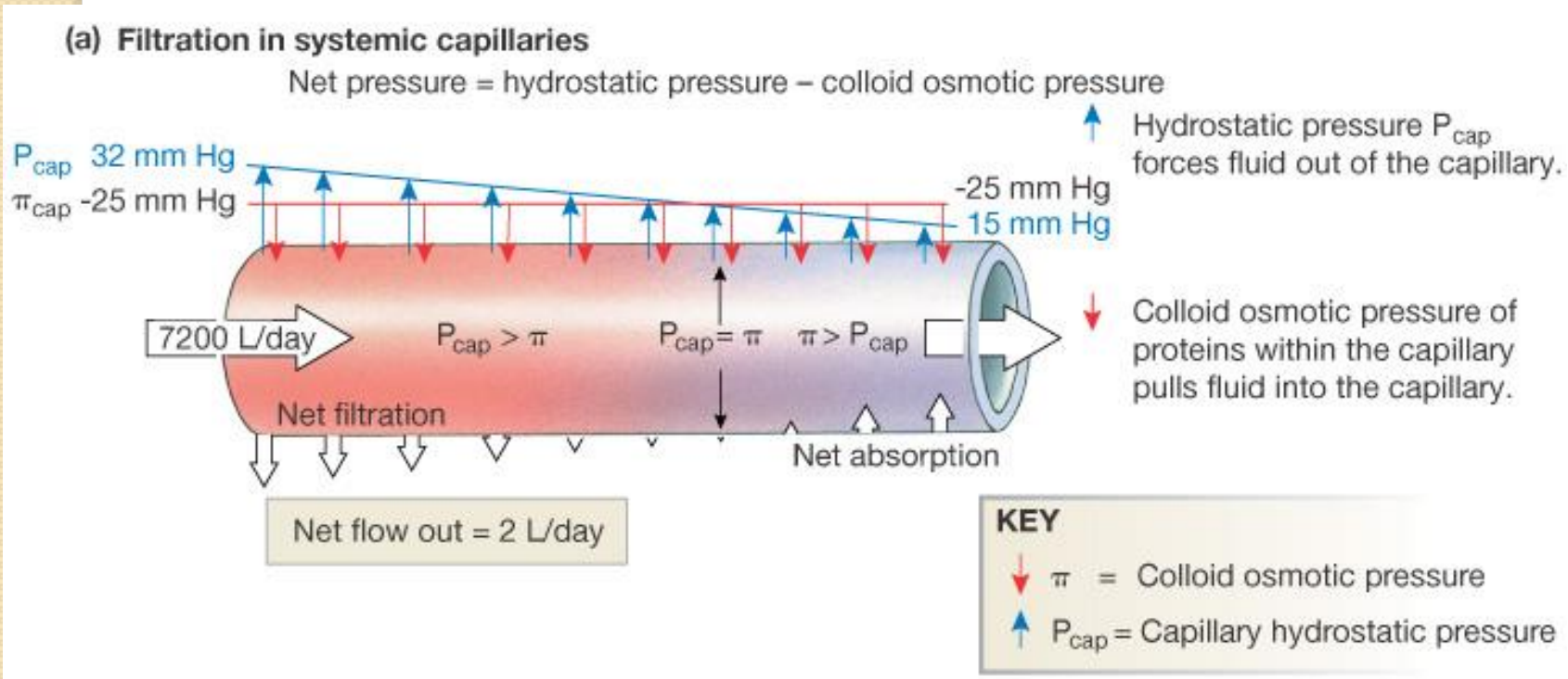


Figure 15-18a: Fluid exchange at the capillary

Capillary fluid shift, continued . . . .

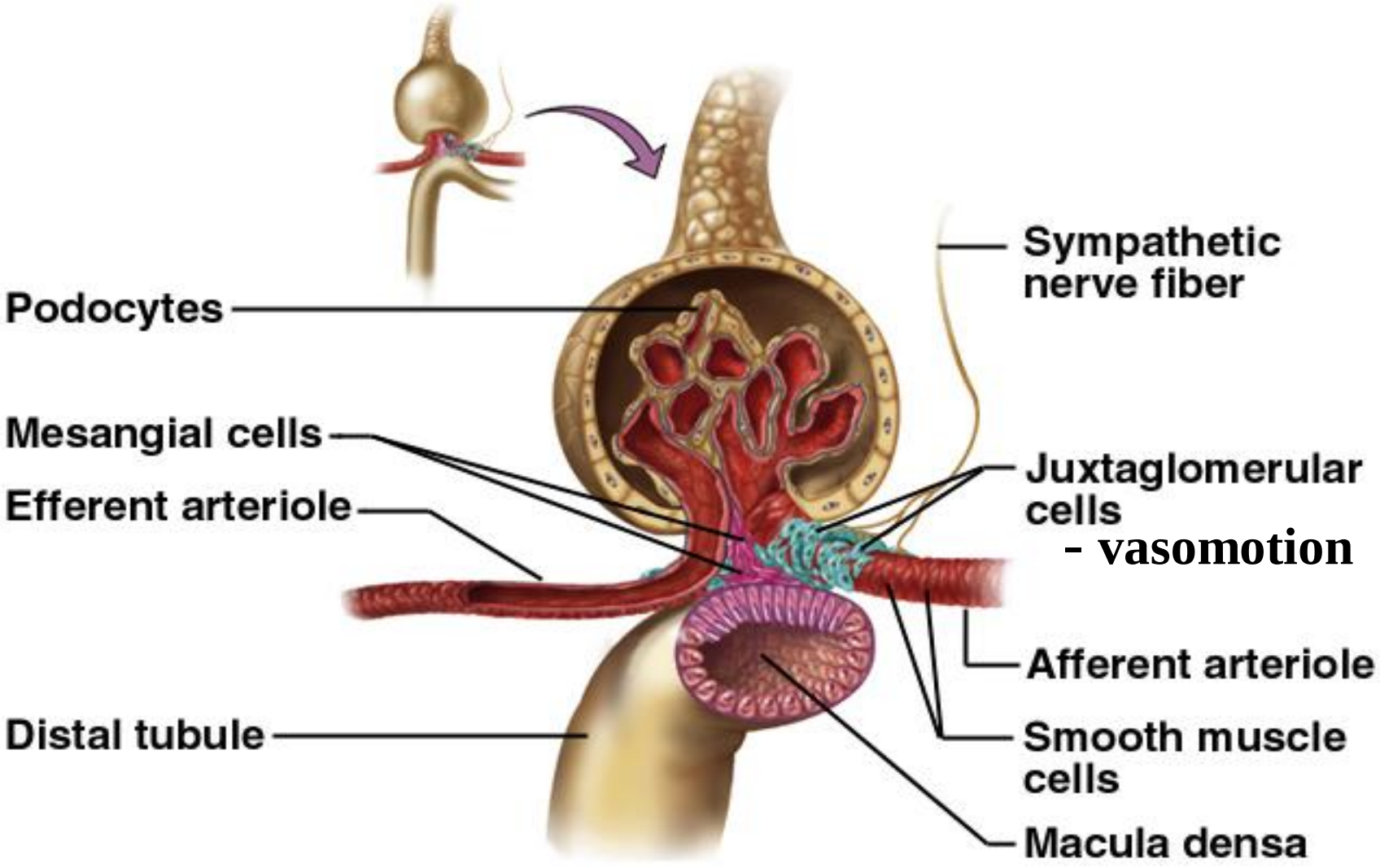
- If capillary pressure increases too high

- Fluid moves out of the circulation into the ECF-compartment

- Blood volume & pressure back to normal

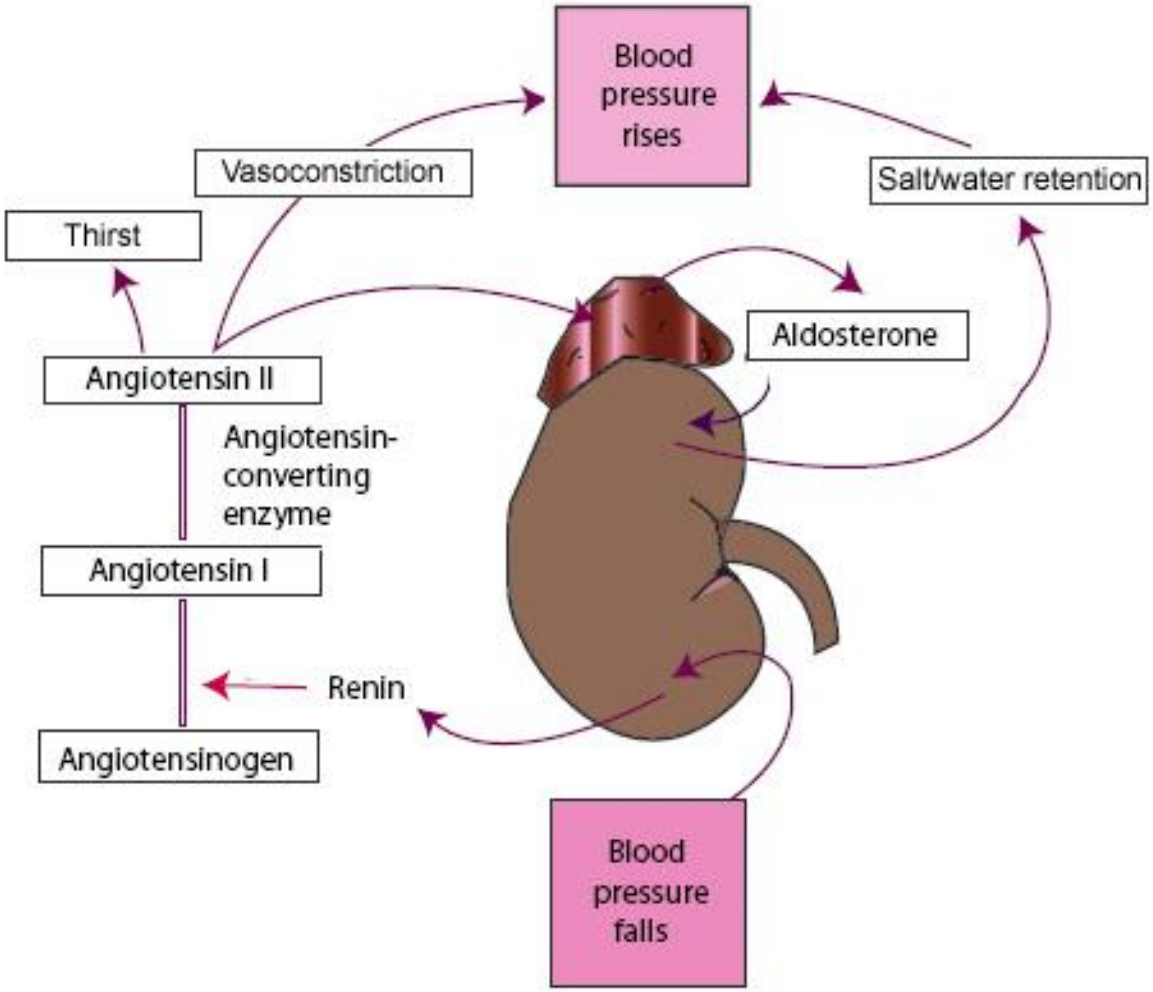
Long-term regulation of ABP: Role of the kidney & Renin-angiotensin system

Juxtaglomerular Apparatus



(monitor salinity)

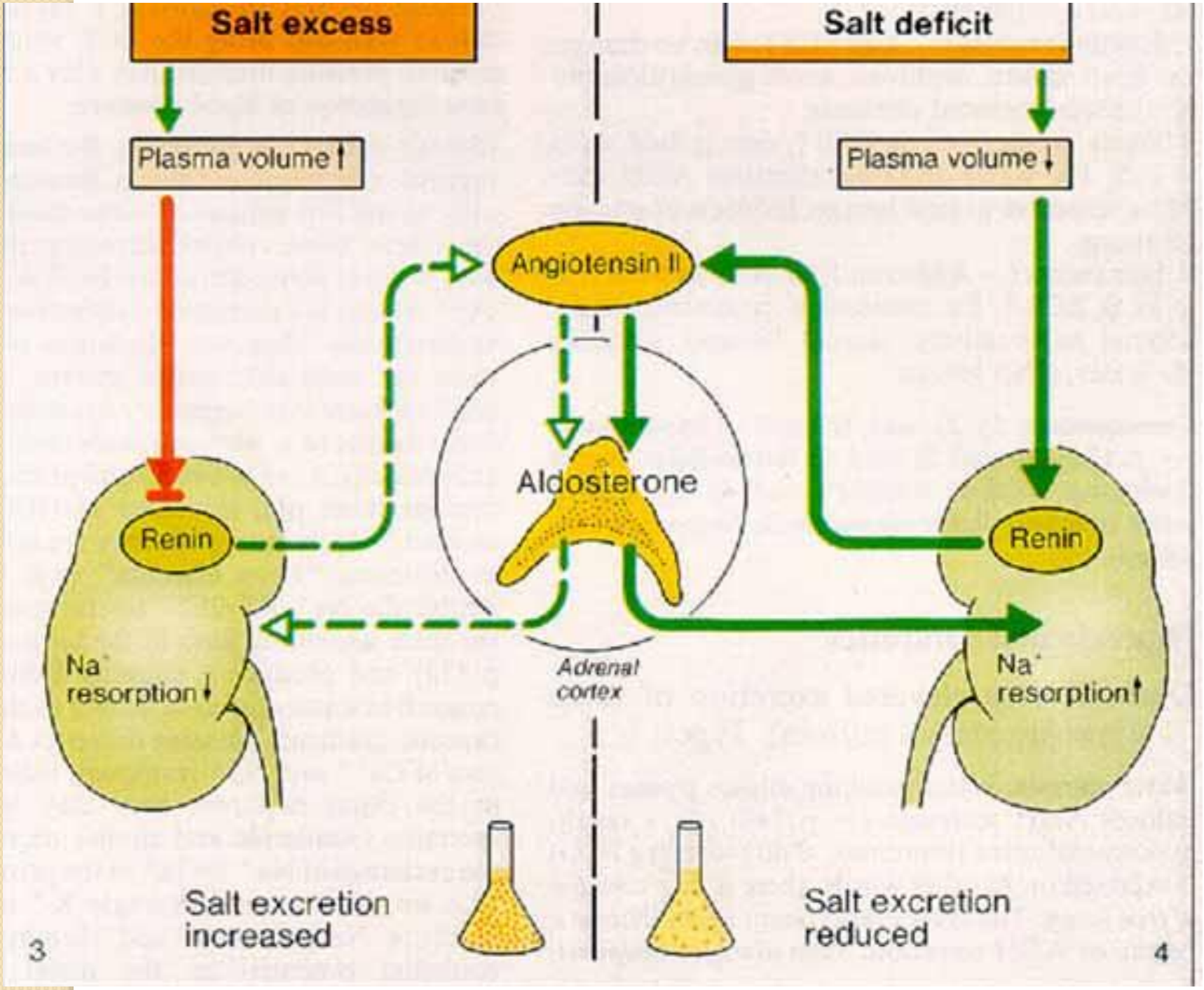
Renin-angiotensin system



Renin-angiotensin system

- Renin is synthesized and stored in an inactive form called prorenin in the juxtaglomerular cells (JG cells)of the kidneys. The JG cells are modified smooth muscle cells located in the walls of the afferent arterioles immediately proximal to the glomeruli.

When the arterial pressure falls, intrinsic reactions in the kidneys themselves cause many of the prorenin molecules in the JG cells to split and release renin. Most of the renin enters the renal blood and then passes out of the kidneys to circulate throughout the entire body.


Salt excess
Salt deficit
Plasma volume ↑
Plasma volume ↓
Angiotensin II
Aldosterone
Renin
Renin
Na⁺ resorption ↓
Na⁺ resorption ↑
Adrenal cortex
Salt excretion increased
Salt excretion reduced
3
4

Role of the Renin-Angiotensin System in Maintaining a Normal Arterial Pressure Despite Wide Variations in Salt Intake:

- One of the most important functions of the renin-angiotensin system is to allow a person to eat either very small or very large amounts of salt without causing great changes in either extracellular fluid volume or arterial pressure.

The initial effect of increased salt intake is to elevate the extracellular fluid volume and this in turn to elevate the arterial pressure

Role of renin-angiotensin system in BP control, continued,..

Then, the increased arterial pressure causes increased blood flow through the kidneys, which reduces the rate of secretion of renin to a much lower level and leads sequentially to decreased renal retention of salt and water, return of the extracellular fluid volume almost to normal, and, finally, return of the arterial pressure also almost to normal.

Role of renin-angiotensin system in BP control, continued,..

- Thus, the renin-angiotensin system is an automatic feedback mechanism that helps maintain the arterial pressure at or near the normal level even when salt intake is increased.
- When salt intake is decreased below normal, exactly opposite effects take place.

- Increased salt intake
- Increased extracellular volume
- Increased arterial pressure
- Decreased renin and angiotensin
- Decreased renal retention of salt and water
- Return of extracellular volume almost to normal
- Return of arterial pressure almost to normal

Functions of Ang II:

1. Vasoconstriction.

2. Decreases excretion of both salt and water by the kidneys (main mechanism for long-term regulation).

Renin-angiotensin system, continued,….

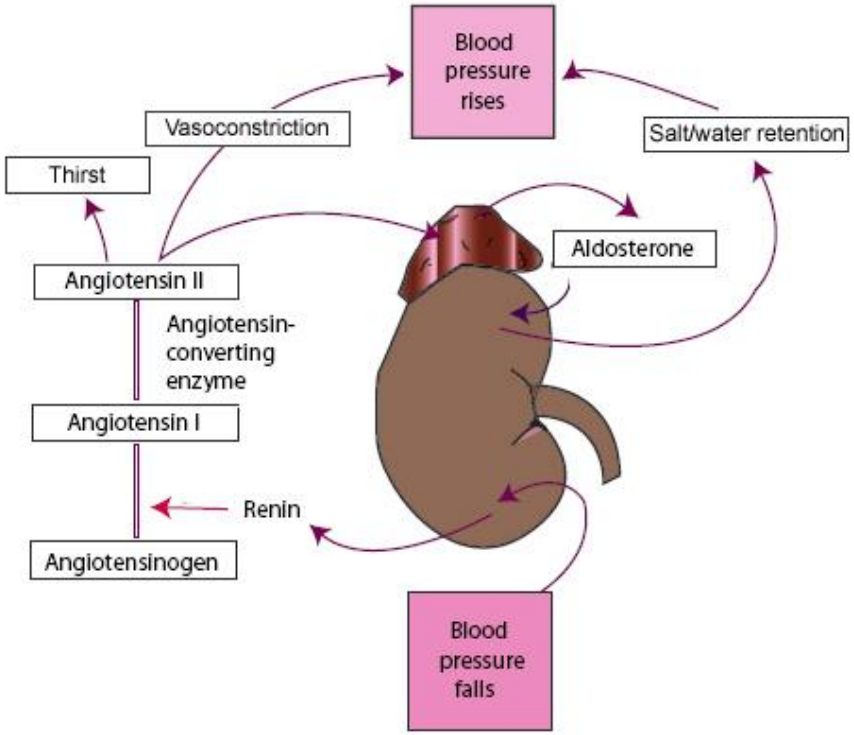
- Angiotensin causes the kidneys to retain both salt and water in two major ways:
- 1. Angiotensin acts directly on the kidneys to cause salt and water retention.

Renin-angiotensin system, continued,….

2. Angiotensin causes the adrenal glands to secrete **aldosterone**, and the aldosterone in turn increases salt and water reabsorption by the kidney tubules.

Thus, whenever excess amounts of angiotensin circulate in the blood, the entire long-term renal–body fluid mechanism for arterial pressure control automaticallybecomes set to a higher arterial pressure level than normal.

Control of blood volume



- **Aldosterone:**
- Secreted by the adrenal cortex triggered by angiotensin II
- Promotes sodium reabsorption by the kidney tubules (Na^+ moves back into the blood and
- H_2O follows by osmosis)
- Whereas ADH promotes H_2O reabsorption only (in response to dehydration), aldosterone promotes reabsorption of both H_2O and salt (in response to ↓ BP)