PHYSIOLOGY TEAM 432

LECTURE : 8

Renal Regulation of Body Fluids

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OBJECTIVES

- Identify and describe the role of the Sensors (receptors) and Effectors in the renal regulation of body fluid volume
- Identify and describe the role of the Sensors and Effectors in the renal regulation of body fluid osmolality
- Role of the kidney in volume regulation
- Role of the kidney in ECF osmolality

MIND MAP

- **Kidney is important organ in regulating ECV & osmolality.**

Renal regulation of ECV

Changes in:
ECV, Na & pressure

Sensor
Carotid sinus & volume receptors(large vein, atria, Internal artery) Right side of circulation

Effectors:
- Rennin/ angiotenssin, aldosterone
- renal sympathetic
- ANF
- ADH

Affecting: Na excretion

Renal regulation of ECV

Changes in:
ECV osmolarity

Sensor
Hypothalamic osmoreceptors

Effectors:
- ADH
- thirst

Affecting:
- Urine osmolality
- Water intake

Higher water intake

Lower water intake

General Notes

- **High water intake => ↓ plasma osmolarity**
- **Low water intake => ↑ plasma osmolarity**

- **This lecture is numbered 7 for boys & 8 for girls**

Additional notes

Boys Only

Girls Only

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).**

That means (sensor and effectors) are working with each other without interference to regulate any changes in total body Na and ECV by sensor (receptors).

- **Regulation of ECF volume = Regulation of body Na+= Regulation BP.**

When we regulate the Na level in the body, we also regulate the volume; because water follows Na.

- **Thus, regulation of Na+ also dependent upon baroreceptors.**

Baroreceptors are receptors which detect any changes in blood pressure .

ECF volume Receptors

Central vascular sensors	Sensors in the CNS	Sensors in the liver
• **Low pressure receptors (very important):** **(present in right side of circulation)** **- Cardiac atria** **- Pulmonary vasculature or Intrarenal vessel.** • **High pressure receptors (less important) :** **(present in left side of circulation)** **- Carotid sinus** **- Aortic arch** **- Juxtaglomerular apparatus (renal afferent arteriole)**	**(less important)**	**(less important)**

1. Renin-angiotensin Aldosterone

- Renin is released into plasma when plasma Na ↓ or volume is low
- 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.

Na reabsorption followed by water >> blood volume & blood pressure will be normal.

Notice: the concentration of Na in the urine will be low.

2. Renal Sympathetic

- (moderate) ↓ ECV → ↑ renal sympathetic activity stimulate Na absorption by direct tubular effect mediated through α-receptors on renal tubules (mainly PCT) to correct for low ECV
- Severe ↓ in ECV → no correction → vasoconstriction and diversion of the blood to vital organ.

3. ATRIAL NATRIURETIC PEPTIDE (ANP) or (ANF)

- ↑ ECV → ↑ Venous return → Stretch of right Atria → release ANP & acts on adrenal gland to inhibit aldosterone release → Na reabsorption by collecting duct
- ↑ Na excretion and water → correcting for the increase in ECV
- ANP can also inhibit Renin secretion.

So it has 2 main functions:

1- acts on the adrenal gland to inhibit aldosterone release

2- inhibit Renin secretion

In both cases, Na is not reabsorbed & it will be excreted.

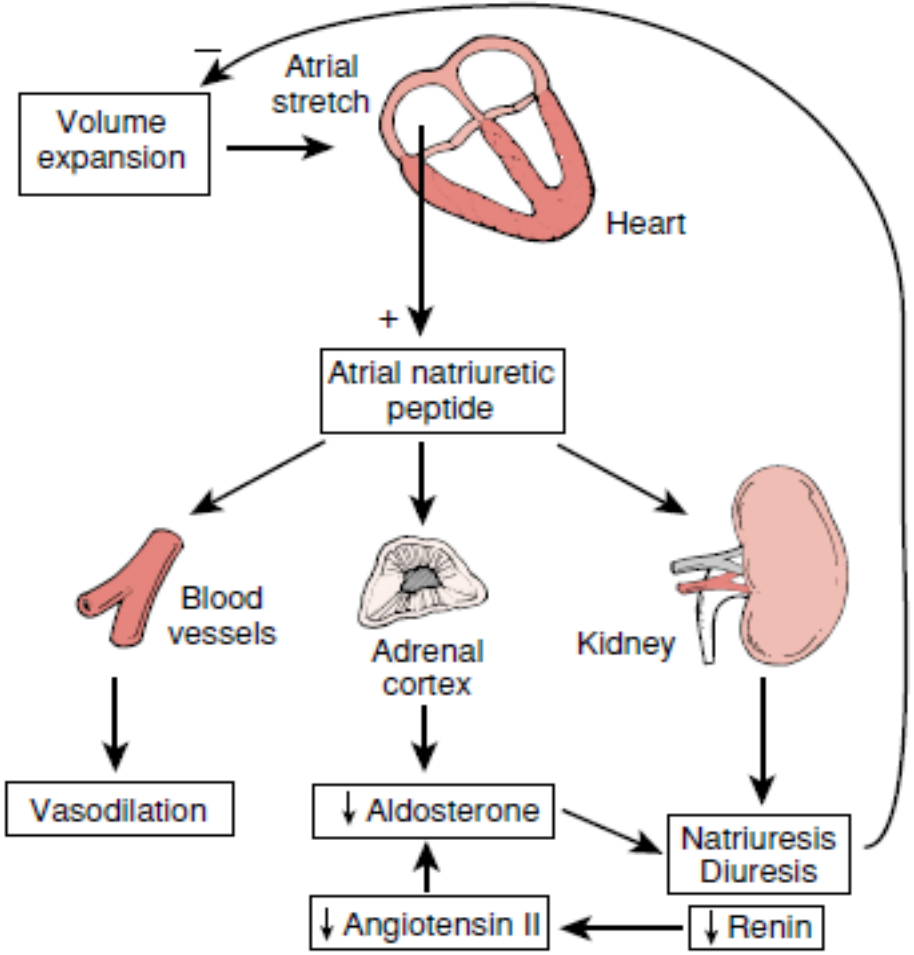


FIGURE 24.10 **Atrial natriuretic peptide and its actions.** ANP release from the cardiac atria is stimulated by blood volume expansion, which stretches the atria. ANP produces effects that bring blood volume back toward normal, such as increased Na^+ excretion.

4. Antidiuretic hormone

Increase in plasma osmolality

↓

osmoreeptor

↓

Trigger the release of ADH → Increase permeability of collecting duct (late distal tubules, cortical collecting tubules & medullary collecting ducts) to H_2O

↓

Increase H_2O reabsorption

↓

Correction of hyperosmolality of blood

ADH is synthesized at the supraoptic & Para ventricular nuclei of hypothalamus & is secreted from the pituitary gland

ADH release is stimulated by:

- Increase osmolality
- Decrease blood volume
- Decrease blood pressure
- Drugs: morphine, nicotine, cyclophosphamide

The relative of ADH is the osmolality, not only the volume.

ECV and Urinary Sodium Excretion

The relation between volume & Na in urine

- Regulation of urinary sodium excretion → regulation ECV
- ↑ ECV or Na is corrected by ↑ urinary sodium excretion and water by:
- Renin–aldosterone
- ANP
- Sympathetic
- ADH

- Increase in blood volume is corrected by increase Na in urine
- Decrease in blood volume is corrected by decrease Na in urine

Renal Regulation of ECF osmolality

- Is a reflex mechanism in which a changin plasma osmolalityis monitor by appropriate sensor which is (Hypothalmus osmoreceptor)

1. Osmoreceptor ADH Feedback System

Increase of osmolality

↓

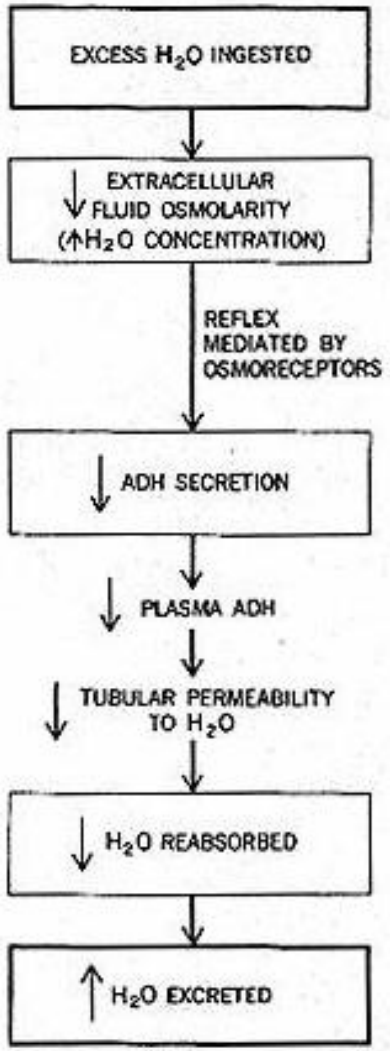
stimulates osmoreceptors

↓

release of ADH

↓

reabsorbed water(mainly) in collecting duct.



2. Role of Thirst in Controlling Extracellular Fluid Osmolarity

- **Thirst is stimulated by:**
  - **Mouth Dryness**
  - **increase Osmolarity**
  - **increase Angiotensin**
  - **decrease Blood Pressure**
  - **decrease Blood Volume**

2. Role of Thirst in Controlling Extracellular Fluid Osmolarity

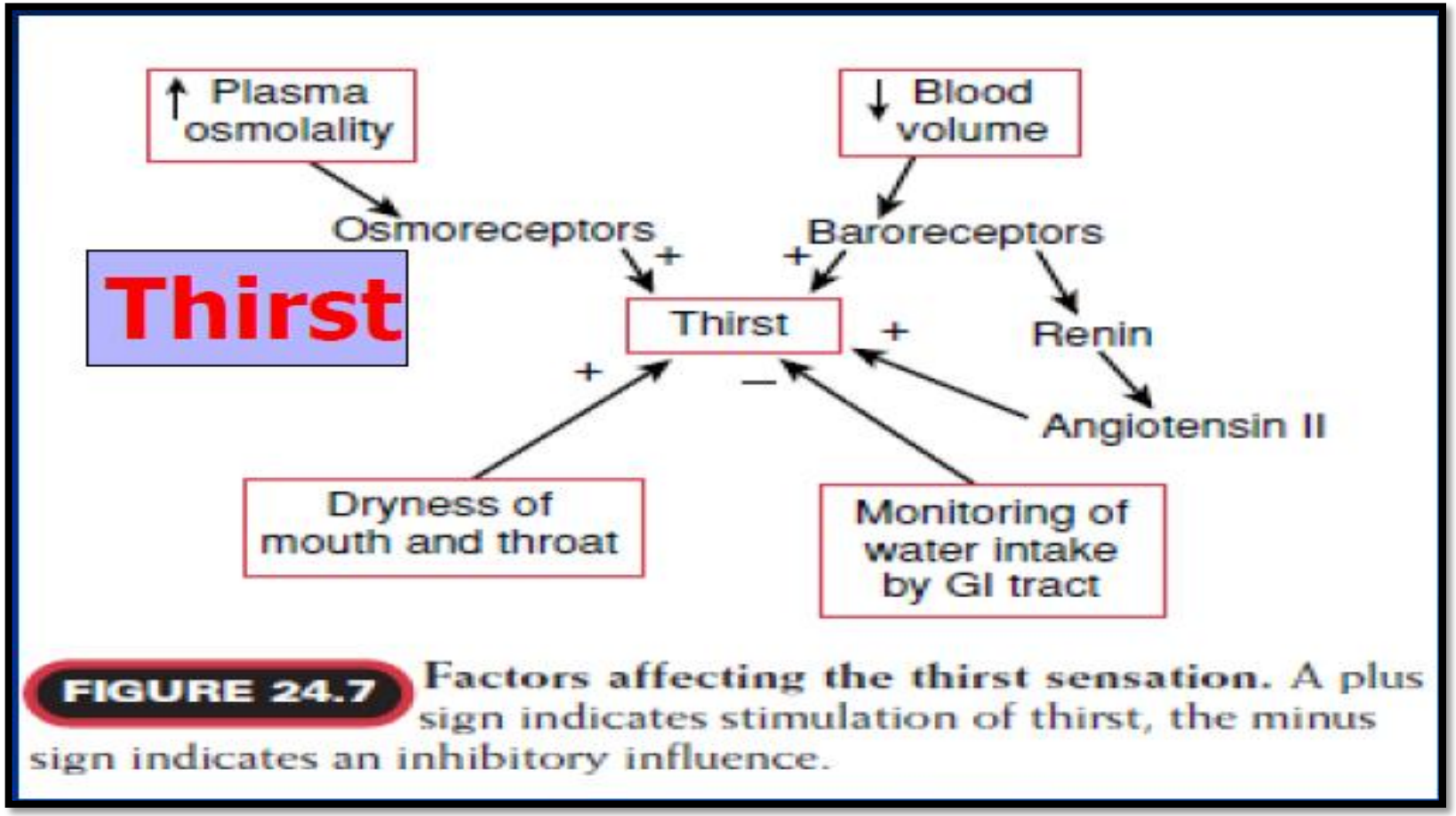


FIGURE 24.7 **Factors affecting the thirst sensation.** A plus sign indicates stimulation of thirst, the minus sign indicates an inhibitory influence.

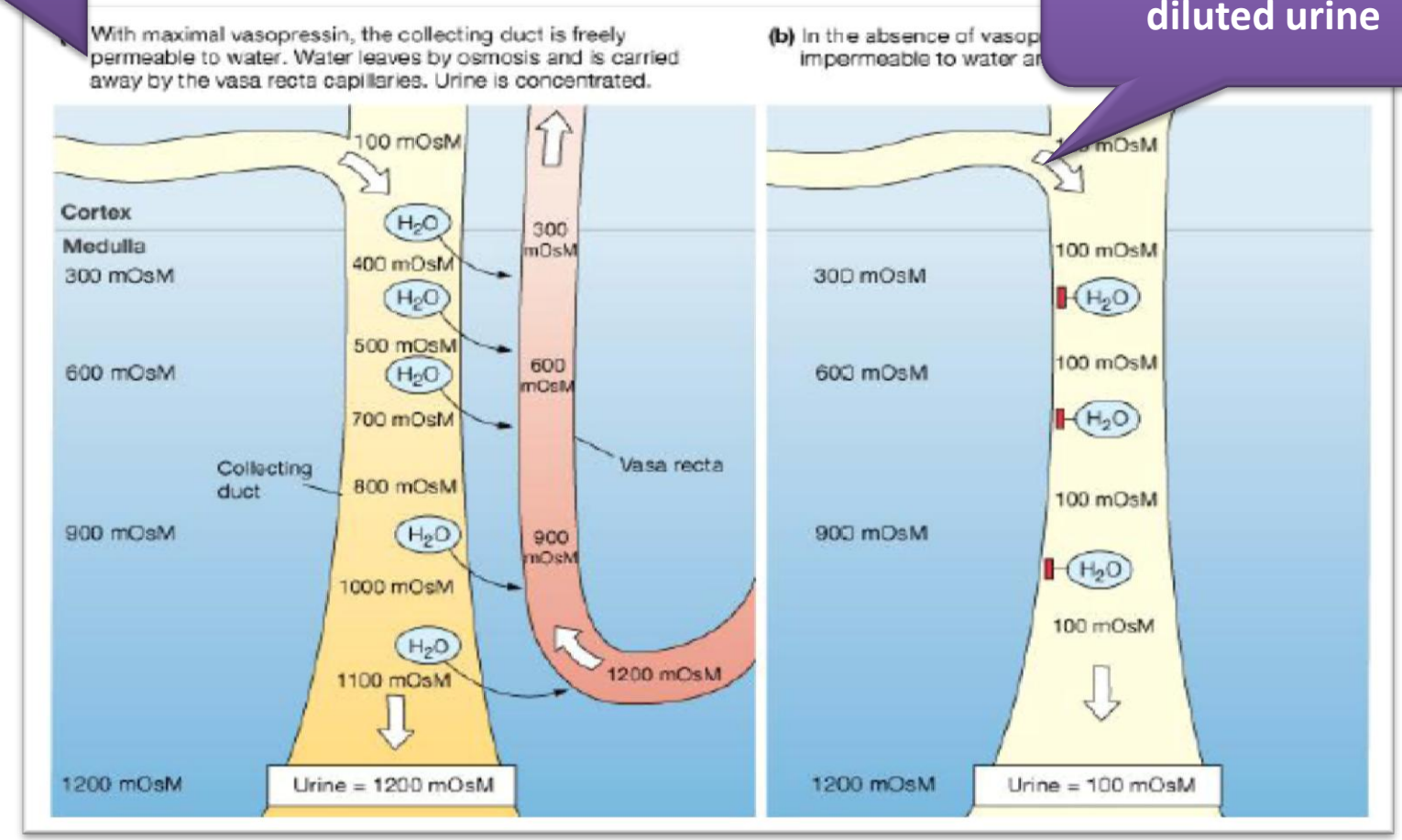
Higher & lower water intake

	High water intake	Low water intake
plasma osmolality	Drop	Increase
ADH	Inhibit ADH secretion	Stimulate ADH secretion
Collecting duct	impermeable to water	Permeable to water
Urine volume(excretion)	Large volume (diluted)	Small volume(concentrated)
	* increase plasma osmolality back to normal.	* Diluting plasma and a drop in osmolality back to normal. * Accompanied by thirst sensation.

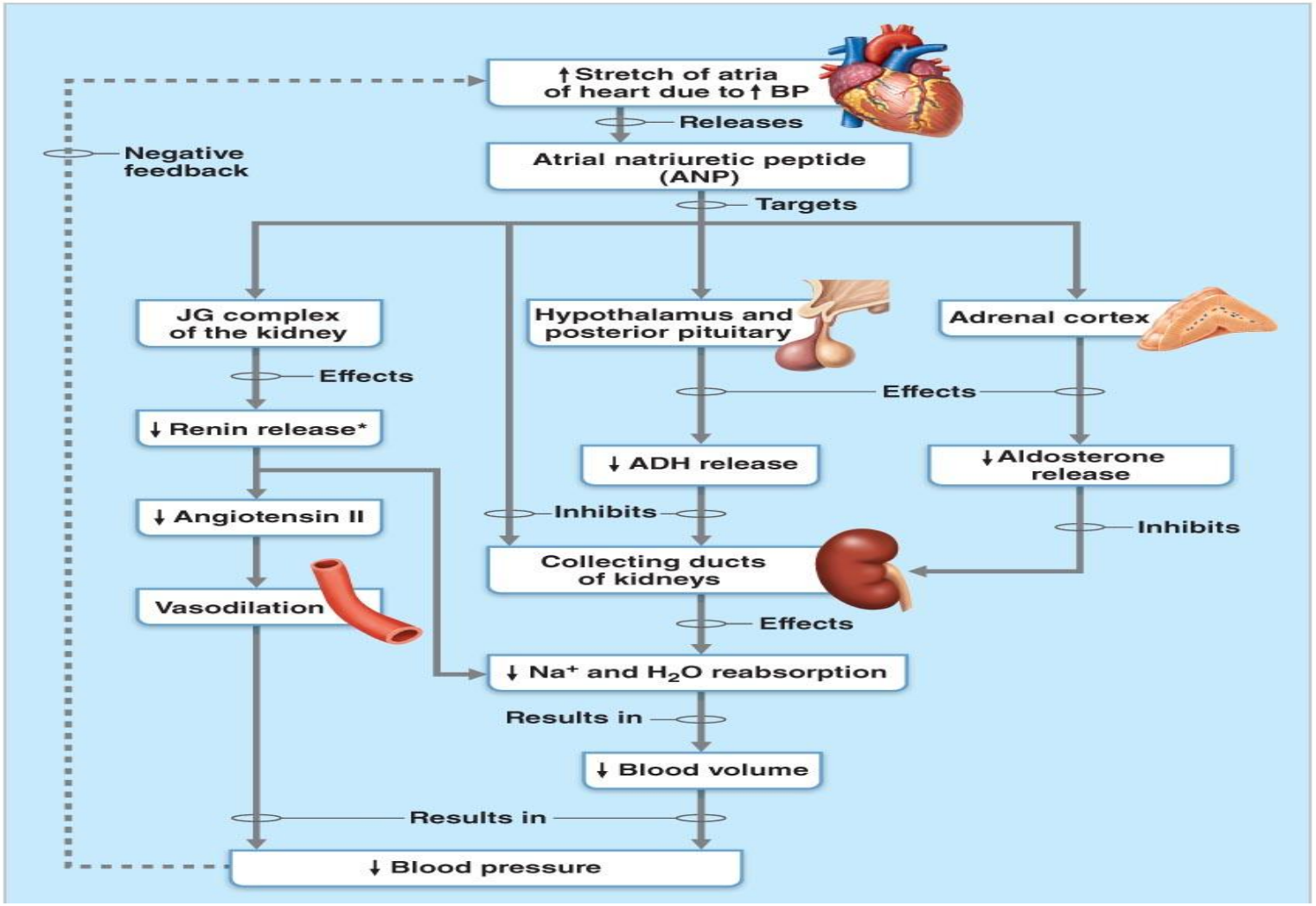
If the kidney can not concentrate the urine (the first sign of renal failure) & continuous passing large quantity of urine >> that will leads to dehydration

Low water intake >> concentrated urine

High water intake >> diluted urine



SUMMARY

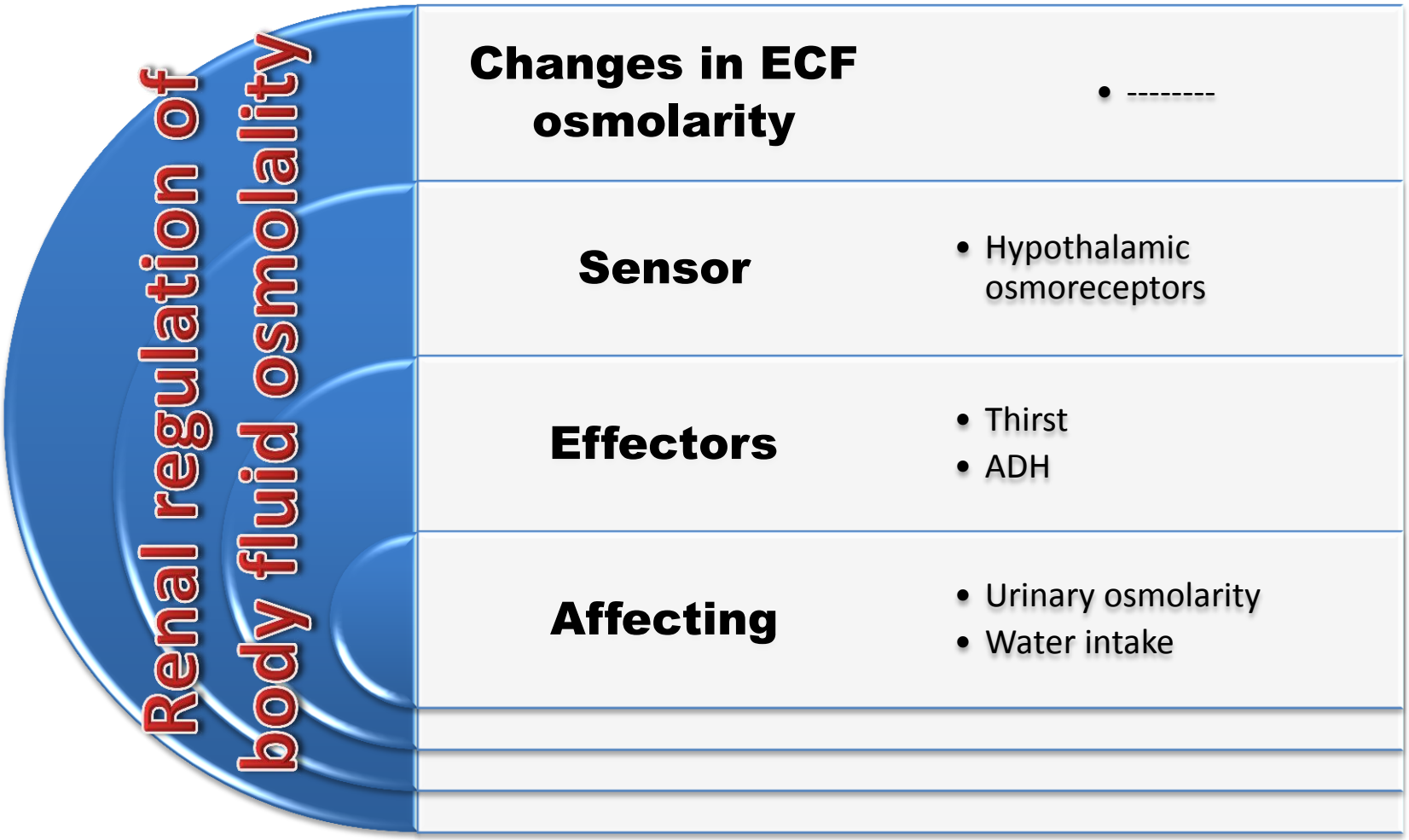

↑Stretch of atria of heart due to ↑ BP
Releases
Atrial natriuretic peptide (ANP)
Negative feedback
Targets
JG complex of the kidney
Effects
↓ Renin release*
↓ Angiotensin II
Vasodilation
Hypothalamus and posterior pituitary
Effects
↓ ADH release
Inhibits
Collecting ducts of kidneys
Adrenal cortex
Effects
↓Aldosterone release
Inhibits
Effects
↓ Na⁺ and H_2O reabsorption
Results in
↓ Blood volume
Results in
↓ Blood pressure

SUMMARY

Renal Regulation of ECV

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

SUMMARY


Renal regulation of body fluid osmolality
Changes in ECF osmolarity
• --------
Sensor
• Hypothalamic osmoreceptors
Effectors
• Thirst
• ADH
Affecting
• Urinary osmolarity
• Water intake

Questions

Q1: Renin:

a) Increase H2O reabsorption
b) Angiotensin II formation
b) decrease Na reabsorption
d) increase Na reabsorption

Q2: Atrial natriuretic peptide:

a) Increase H2O reabsorption
b) Angiotensin II formation
b) decrease Na reabsorption
d) increase Na reabsorption

Q3: ADH:

a) Increase H2O reabsorption
b) Angiotensin II formation
b) decrease Na reabsorption
d) increase Na reabsorption

Q4: What affect does ADH have on urine output?

a) Minimal
c) Decreases
b) increases
d) maintains

Q5: The action of the aldosterone is to increase:

a) Na elimination
c) K reabsorption
b) Na reabsorption
d) Cl excretion

C
B
A
C
B

THE END

If there are any problems or suggestions
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THANK YOU

Actions speak louder than Words