Renal Block

Physiology Team

6th Lecture

Renal regulation of body fluids

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***JUST TO DIFFERENTIATE BETWEEN OSMOLARITY AND OSMOLALITY* (NOT IMP)**

Osmolarity and Osmolality:

The osmolal concentration of a solution is called:

- Osmolality when the concentration is expressed as "osmoles per Kg of Water"

- Osmolarity when the concentration is expressed as "osmoles per Liter of Solution"

Plasma osmolality is affected by changes in water content. In comparison, the plasma osmolarity is slightly less than osmolality, because:

The total plasma weight (the divisor used for osmolality) excludes the weight of any solutes, while the total plasma volume (used for osmolarity) includes solute content.

Otherwise, one liter of plasma would be equivalent to one kilogram of plasma, and plasma osmolarity and plasma osmolality would be equal.

The Extracellular osmolality (Keep in mind) (below: just read)

* The blood osmolality depends mainly on Na^+ concentration because:

Regulation of ECF volume = Regulation of body Na+= Regulation BP (regulated together)

Thus, regulation of Na^+ also dependent upon baroreceptors.

* Macula Densa and Carotid and Aortic baroreceptors work to regulate BP

* Osmoreceptors (Hypothalamus) → stimulate:

- Thirst Center

- Hunger Center

- Satiety (الشبع التام) Center

Atrial Natriuretic Peptide (**ANP**) =
Atrial Natriuretic Factor (**ANF**) =
Atrial Natriuretic Hormone (**ANH**)

Or **Atriopeptin.**

* Osmoreceptors → ↑ Plasma Osmolality

* Baroreceptors → ↓ Blood Volume by RAAS (Renin-Angiotensin-Aldosterone System)

↑ Water intake → ↑ BV → ↓ osmolality → ↓ ADH → ↑ Urine

↑ Water salt intake → ↑ BV → Osmolality not changed → needs 3 days of correction

* Angiotensin II has: **(VERY IMP!!)**

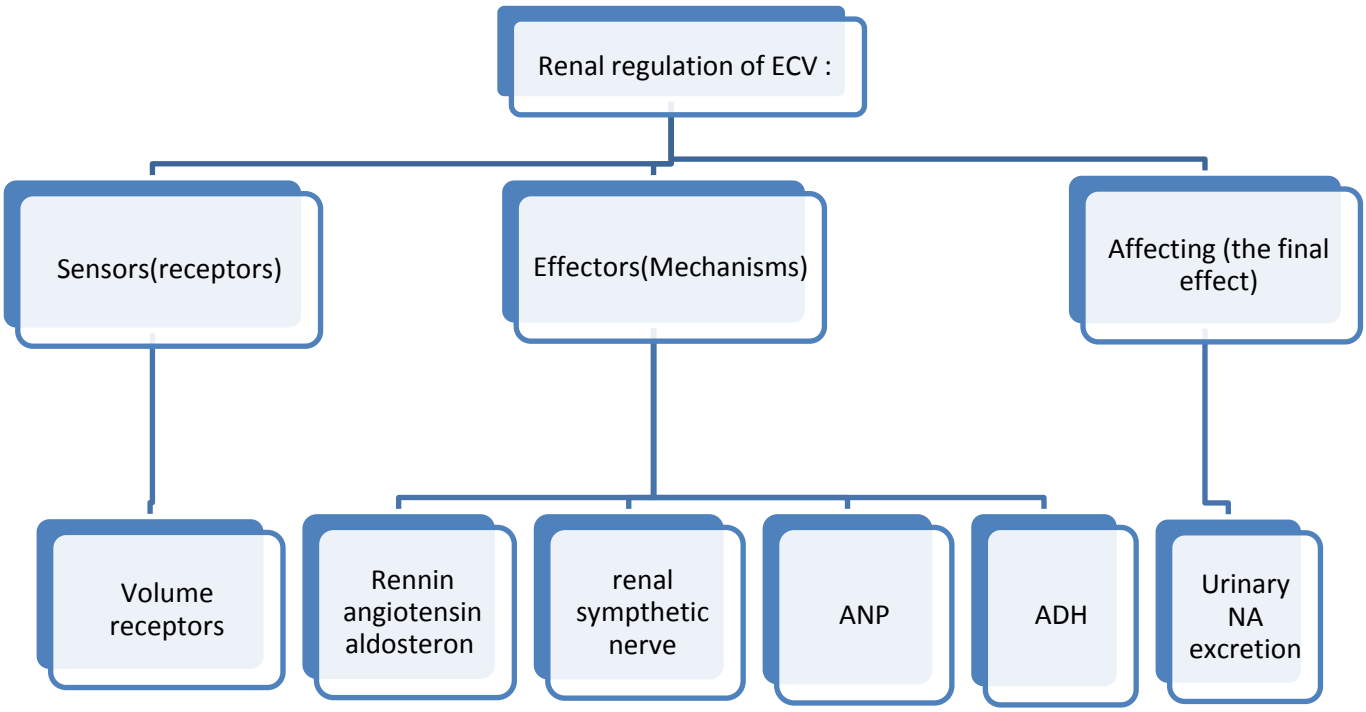
- Minimal effect on Afferent Arteriole

- Maximal effect on Efferent Arteriole

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). I.e.: renal function is regulated by neural and hormonal influences

Regulation of ECF volume = Regulation of body Na+= Regulation BP
Thus, regulation of Na+ also dependent upon baroreceptors .



Renal regulation of body fluids :

First :

Receptors : they are known as volume receptors, They have three types :

1.Central vascular sensors

- Low pressure receptors (very important) **which are found in :**
  - Cardiac atria
  - Pulmonary vasculature
- High pressure receptors (less important) **which are found in :**
  - Carotid sinus
  - Aortic arch
  - Juxtaglomerularapparatus (renal afferent arteriole)

2.Sensors in the CNS (less important)

3.Sensors in the liver (less important

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Second :

Renal mechanisms that regulate ECV:

1- Renin-angiotensinAldsterone:

Rennin is an enzyme that is synthesized ,stored and secreted by granular cells of the affrent and the efferent arterioles of the glomerulus , in a specialized region known as the juxtaglomerular apparatus which have special type of cells known as" macula densa "

That respond to changes in the composition of tubular fluid

Rennin secretion is stimulated by three factors :

1- when there is a decrease in the NA delivery to macula densa , I.e. Plasma NA is decreased :

Reninis released into plasma when plasma Na ↓

•Renin → angiotensinogen → AngiotensinI

•AngiotensinI → ACE→ angiotensinII

•angiotensin II act on adrenal cortex → aldosteronesecretion → ↑Na reabsorptionin distal & collecting duct of nephron

2- Increased renal sympathetic activity

3- Reduced renal perfusion pressure (For more information):
The actual stimulus seems to be a reduction in the pressure within the afferent arterioles and the resulting reduction in the wall tension in this arteriole . This can occur as a direct result of a reduction in systemic arterial blood pressure . It may be reinforced by constriction caused by an increase in sympathetic activity to the kidney

Cont.. of the mechanisms :

2- Renal sympathetic :

↓ 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

3. ATRIAL NATRIURETIC PEPTIDE (ANP):

↑ ECV → Stretch of Atria → release ANP → inhibit aldosteronerelease →↓sodium reabsorptionby collecting duct

- ↑sodium excretion and water → correcting for the increase in ECV
- ANP can also inhibit Renin secretion

For more understanding :

ANP is synthesized and relased by myocardial cells of the atrium , it is released by stretch of the atrium . In heart failure , it is released by the ventricles as well.

ANP has several actions which are not fully understood yet , but generally : they tend to oppose the actions of rennin angiotensin system include the followings :

Vasodilatation of the kidney, inhibit rennin and aldosteron secretion

4. Antidiuretichormone

- Increase of plasma osmolality → osmoreceptor → trigger the release of ADH.
- ADH → ↑permeability of collecting duct to H2O → ↑ H2O reabsorption → correction of hyperosmolalityof blood.

ADH release stimulated by

↑Osmolarity

↓ Blood volume

↓ Blood pressure

Drugs: Morphine; Nicotine; cyclophosphamide

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Renal regulation of ECF osmolality :

Is a reflex mechanism in which a changin plasma osmolalityis monitor by appropriate sensor (osmoreceptors) Hypothalmusosmoreceptor

■ It is regulated by : ADH and thirst

1- Role of ADH:

■ in the case of High water intake

1) there will be Drop in plasma osmolality
2) this will inhibit ADH secretion
3) Collecting ducts are impermeable to water(I.e. decrease in the reabsorbtion)
4) excretion of large volume of urine
5) increases plasma osmolalityback to normal.

■ **in the case of 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 osmolalityback to normal.
6) Accompanied by thirst sensation

2- Role of thirst :

Thirst sensation stimulated by:

1.↑Osmolarity

2.↓ Blood volume

3.↓ Blood pressure

4.↑ Angiotensin

5.Dryness of mouth

Feeling thirsty lead to consume more water – correcting the hyperosmolality