Physiology | Lecture 11B

Body Fluids pt.2

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Regulation of Fluid Volumes and Osmolality

In this sheet, we will talk about many things related to the regulation of fluid volumes and osmolality, divided into:

A. Regulation of Na^+ and Water -> B. Disorders of Volumes

-> C. Disorders of Osmolality -> D. Disorders of Volumes and Osmolality

-> E. Oedema.

Important note: our doctor didn't explain the mechanisms in this sheet deeply, but he did last years, so anything in this color will be extra information from the last years to make everything obvious and for more understanding, should you study it? Yes, without it you won't fully understand the sheet.

A. Regulation of Na^+ and Water:

That involves regulation of Osmolality and Volume of ECF.

1. Osmolality (Osmoregulation):

Osmoreceptors: They are receptors that sense changes in osmolarity.

We can regulate both the intake and the output of water, by:

-Regulation of Intake

a. Osmoreceptors in hypothalamus sense the increase in plasma osmolality and sends signals toward hypothalamic thirst center, leading you to feel thirst and drink water, increasing the water intake.

-Osmolality ↑ -> thirst ↑ -> water intake ↑

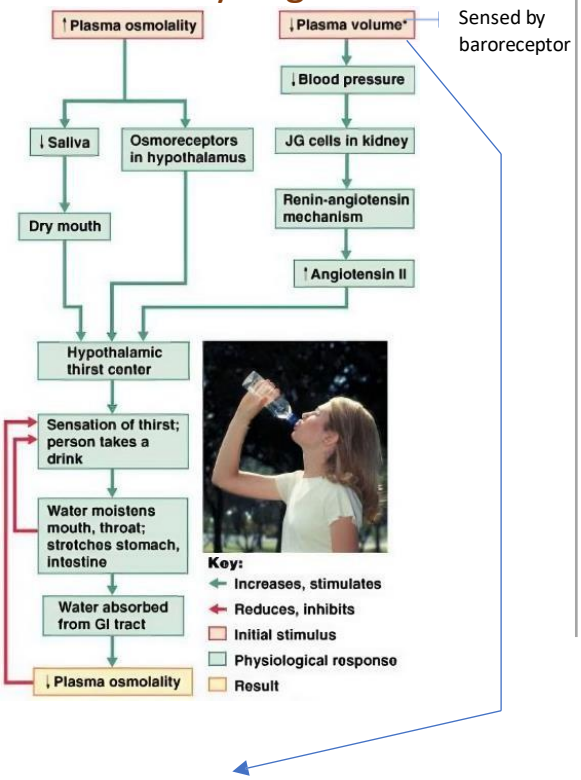
b. The increase in plasma osmolarity reduces salivation which results in a dry mouth , which stimulates the thirst center to activate thirst sensations.

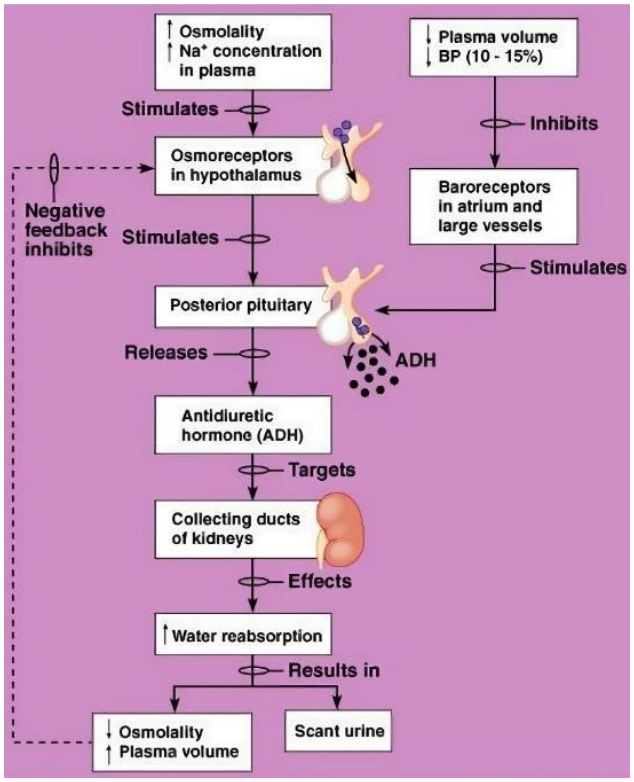
Regulation of Output

Again, Osmoreceptors in hypothalamus sense the increase in plasma osmolality and stimulate the posterior pituitary to release ADH, which decreases water output by acting on renal collecting ducts to increase water reabsorption.

b. - Osmolality ↑ -> releasing ADH ↑ -> water reabsorption ↑ -> decrased water output ↓

c. Decreased plasma volume means decreased blood pressure and that will activate juxtaglomerular cells in the kidney leading to activate Renin-angiotensin-aldosterone system, this system will activate the thirst centers in hypothalamus by angiotensin II.





2. Volume of ECF:

That regulation depends on Na^+ excretion in urine, you will understand the reason later.

Controlled by **a. Renin-Angiotensin Aldosterone system**:

a. the plasma volume <u>decreases.</u>

b. the blood pressure <u>decreases.</u>

c. Baroreceptors sense this change in blood pressure and stimulate the Juxtaglomerular cells (Kidney).

d. Juxtaglomerular cells release Renin.

e. Renin converts Angiotensinogen into Angiotensin I.

f. Lungs convert Angiotensin I into Angiotensin II.

g. Angiotensin II stimulate the suprarenal gland to release the Aldosterone.

h. Aldosterone <u>increases</u> the reabsorption of Na^+.

- When the plasma volume decreases (e.g. from dehydration and blood loss) , the body activates mechanisms to restore blood pressure and volume . Two major hormones involved are :

1.ADH
(antidiuretic hormone)

2. Angiotensin II

- ADH hormone .

Released by : the posterior pituitary gland in response to low blood pressure and high blood osmolarity

Effects : acts on kidneys to reabsorb water and reducing urine output , causes vasoconstriction (narrowing of blood vessels) .

- Angiotensin II

Part of the renin angiotensin system (RAAS)

Triggered by : low blood pressure , or low sodium levels

Effects : strong vasoconstrictor ---> increases blood pressure , Stimulates Aldosterone release from the adrenal glands --> increases water and sodium reabsorption in the kidneys , Stimulates ADH release from the posterior pituitary gland.

-Together , they restore blood volume and pressure by conserving water , increasing vascular tone and promoting sodium reabsorption .

So while ADH is strongly stimulated by

High osmolarity and low plasma volume ;

Angiotensin II is stimulated by a low plasma volume as part of the renin Angiotensin Aldosterone system .

Quick Summery of hormones involved in regulation of fluid output :

a) **Antidiuretic Hormone (ADH / Vasopressin)**

- Secreted by the posterior pituitary.
- Increases water reabsorption in the kidneys (collecting ducts).
- Reduces urine volume → conserves water.

b) **Aldosterone**

- Produced by the adrenal cortex.
- Promotes sodium reabsorption and potassium excretion in the kidneys.
- Water follows sodium → increases blood volume and pressure.

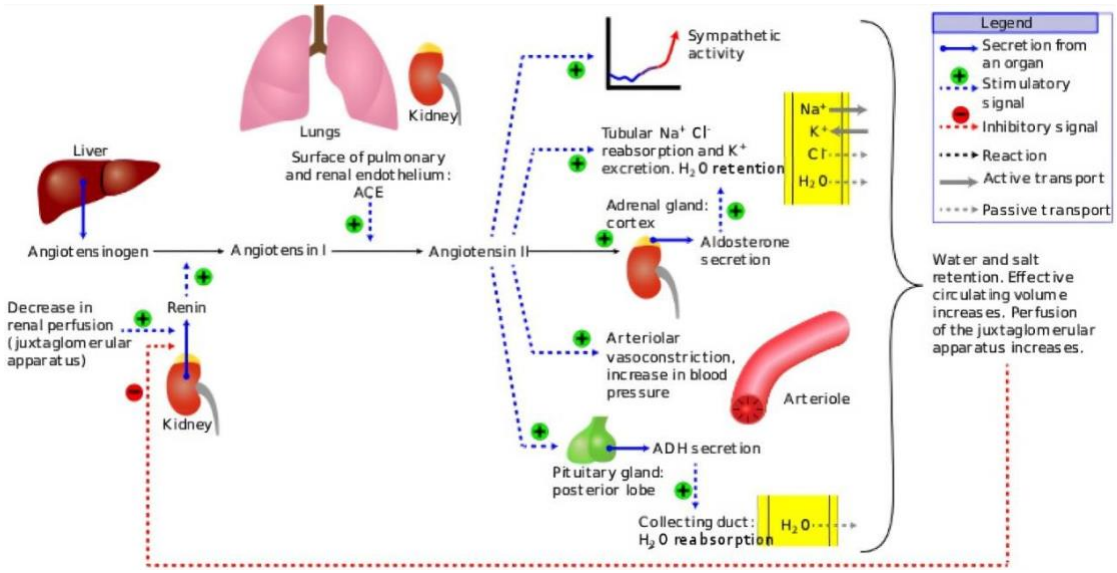
c) **Atrial Natriuretic Peptide (ANP)**

- Released by the atria of the heart when blood volume is high.
- Increases sodium and water excretion.
- Lowers blood pressure and volume.

d) **Renin-Angiotensin-Aldosterone System (RAAS)**

- Activated when blood pressure or volume drops.
- Leads to aldosterone release, vasoconstriction, and water retention.

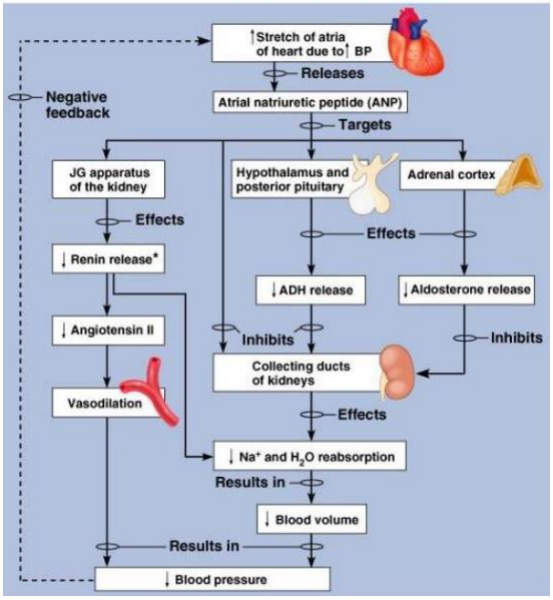
. Angiotensin II constricts blood vessels too.
Which increases blood pressure



-Controlled by **b. Atrial natriuretic peptide**:

When the blood pressure increases (that means increased plasma volume too) the Atria will be stretched which releases ANP, ANP will inhibit the secretion of Angiotensin II, Aldosterone and ADH that will lead to vasodilation and decreasing of water and Na^+ reabsorption that will decrease blood volume and reduce blood pressure.

-> Vasodilation because of the inhibition of Angiotensin II.



B. Disorders of Volumes:

Hypovolemia:	hypervolemia:
decrease in volume.	Increase in volume.

1. Hypovolemia (Dehydration): Results by excessive loss of fluids, caused by diarrhea, vomiting and blood loss excessive sweating , excessive urination ...., it leads to dehydration.

-> Decreased release of ADH can cause Hypovolemia too.

2. Hypervolemia (Overhydration): Results by excessive intake or administration of Fluids if that high intake of water is not accompanied by water output from the body

- **Disorders of osmolality :**

1. Hyponatremia: Results by excessive loss of Na+ or administration of hypotonic fluids.

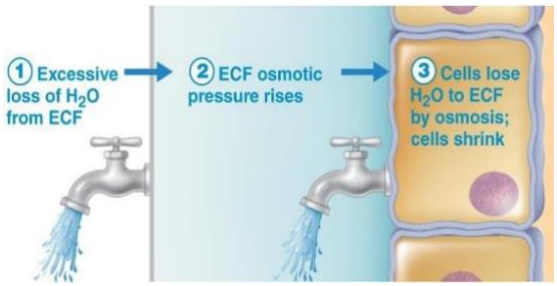
2. Hypernatremia: Results by excessive intake of Na+ or administration of hypertonic fluids

D. Disorders of Volumes and Osmolality:

Disorders of Volumes and Osmolality
–Isonatremia with hypovolemia
–Isonatremia with hypervolemia
–Hyponatremia with hypovolemia
–Hyponatremia with hypervolemia
–Hyponatremia with isovolemia
–Hypernatremia with hypovolemia
–Hypernatremia with hypervolemia
–Hypernatremia with isovolemia

Disorders of Volumes and Osmolality
–Combinations are according to the fluid loss or gain: (hypo-, hyper- or isotonic)

Here we just need to understand the overlapping between the two types of disorders.

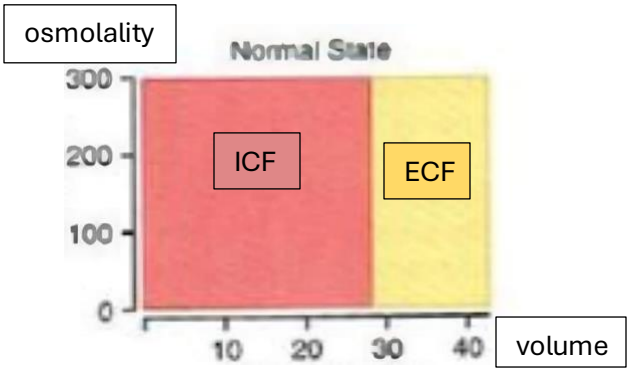


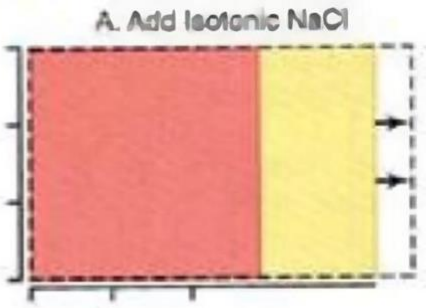
1. ECF becomes Hypovolemia (Dehydration).
2. ECF becomes Hypernatremia.
3. Water move from ICF toward ECF by osmosis .
4. The cell shrinks.

1. ECF becomes Hypervolemia (Overhydration).
1. ECF becomes Hyponatremia.
2. Water moves from ECF toward ICF by osmosis.
3. The cell Swells.

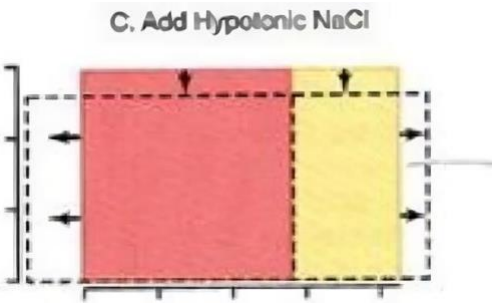
1. High release of ADH: Hypervolemia, Hyponatremia.
2. Low release of ADH: Hypovolemia, Hypernatremia.
3. High release of Aldosterone: Hypervolemia, Hypernatremia.
4. Large amount of Water: Hypervolemia, Hyponatremia.

Can we achieve Hypervolemia and Isonatremia? Yes, by adding isotonic solution, such as Normal Saline (9 grams of NaCl per Liter) the volume of ECF will increase but the osmolality won't change! Here is an example:

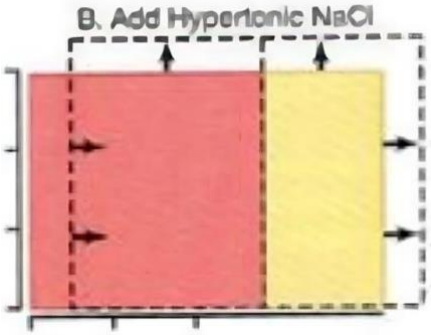




If we added a hypotonic NaCl solution to the ECF, the osmolality would decrease and fluids would move from the ECF toward ICF, so the osmolality of both ECF and ICF would decrease and the volume of ECF and ICF would increase.

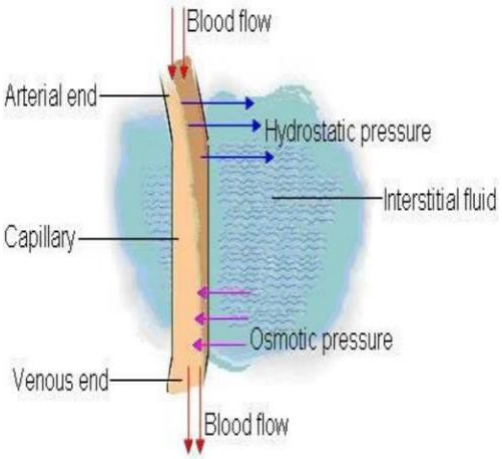


If we added a hypertonic NaCl solution to the ECF, the osmolality would increase and fluids would move from the ICF toward ECF, so the osmolality of both ECF and ICF would increase and the volume of ECF would increase while the volume of ICF would decrease.



At the Arterial End of the capillaries, we have high hydrostatic pressure, which is forcing fluids to move from capillaries toward interstitial fluid compartment.

At the Venous End of the capillaries there is oncotic pressure (colloid osmotic pressure of proteins) which moves the water back to the capillaries.



E. Oedema:

Oedema is a localized increase in the volume of interstitial fluid.

Causes of Oedema:

1. Increased capillary hydrostatic pressure:

-> Kidney causes: more retention of water and salts (as in Renal failure), that will increase the hydrostatic pressure.
-> Excess of Mineralocorticoids (aldosterone) means increased osmolality and increased hydrostatic pressure.

2. Decreased oncotic (colloid) pressure:

Can be caused by:

a. Increased loss of proteins:

-> From Kidney in nephrotic syndrome.
-> from skin in burns and severe wounds.

b. Decreased production of proteins:

-> Liver diseases.
-> Decreased intake of proteins in malnutrition.

3. Increased capillary permeability:

Can be caused by:

a. During immune reactions by release of histamine | **b.** Toxins | **c.** Infections
d. Vitamin C deficiency | **e.** Ischemia | **f.** Burns.
These can get more proteins passing from capillaries toward interstitial fluid and affecting the localization of fluids.

4. Decreased lymph drainage:

As we know the capillaries aren't permeable for proteins, but some of them can escape the filtration from the capillaries toward interstitial fluid, these proteins are removed by the lymphatic circulation (think about it like washing out proteins from the interstitial compartment and getting these proteins back to the general circulation), that keeps low colloid pressure in the interstitial fluid.

Can be caused by:

a. Cancer | **b.** Infections | **c.** Surgery | **d.** Absence or abnormality of lymphatic vessels.

5. High venous pressure **:**

Increased hydrostatic pressure in the capallires end leads to no reabsorption of water in venous end (obstruction, decreased venous pump activity) and retention of water in the interstitial fluid compartment.

-> It is very low in normal conditions, may be caused by heart failure.

6. Decreased arteriolar resistance**:**

When vasodilation occurs (decreased resistance), more blood will flow leading to more filtration.

-> Vasodilation could be caused by excessive body heat, insufficiency of sympathetic nervous system and vasodilators.

Safety factors for preventing oedema:

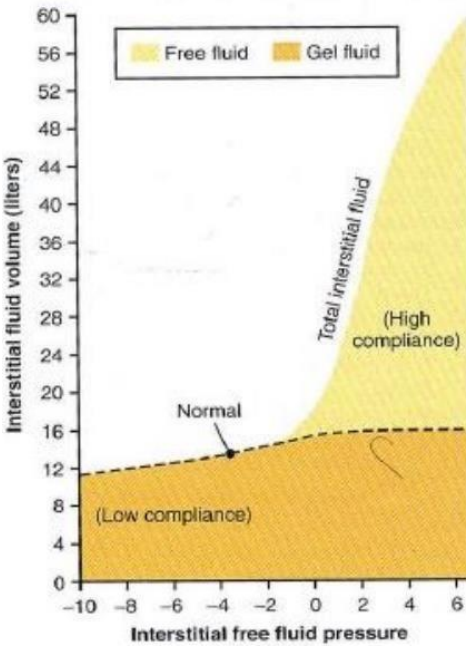
a. Low tissue compliance:

The fluid is a gel like fluid that has the property of low compliance.

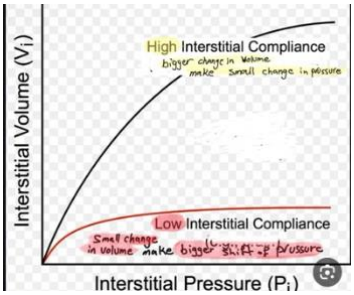
-> Low compliance means that any small increase in its volume can cause a big shift in its pressure.

-> At normal condition, the interstitial fluid will be at negative pressure range.

-> At that level, any increase in volume of the fluid (even if this increase is small) will result in bigger shift in its pressure which prevents more filtration of fluids from capillaries and will prevent Oedema.



-> We should mention that once we get accumulation of free fluids we shift from low compliance to high compliance, at positive pressure range any big increase in the volume can cause only a small changes in the pressure, if you pressed over the region you have oedema at, you will get pitting oedema as a sign.



B. increased lymph flow (from the interstitial fluid toward general circulation)

-Lymph flow can increase up to 10-50 folds more than usual. By this increasing, the lymph flow can carry away large amounts of fluids from the interstitial fluid compartment. Leading to reducing the amount of fluids and preventing the interstitial fluid pressure from rising towards the positive ranges.

C. increased protein wash-down from interstitial fluid

Increased lymph flow can carry with it large amount of protein from the interstitial fluid, this results in decreasing colloid osmotic pressure in interstitial fluid leading to decreasing net filtration forces and decreasing of fluids from the capillaries toward interstitial fluid, which results in preventing accumulation of fluids.

-In addition, the decreasing protein content in the interstitial fluid by the protein washout, you are increasing and reabsorption of these fluid at the venous end of capillaries.

-the difference between the oncotic pressure inside capillaries and the interstitial fluid will be higher, which is favoring more movements of fluids from the interstitial compartment toward capillaries. This process prevents accumulation of fluids in interstitial compartment.

Additional Resources

رسالة من الفريق العلمي:

لا إله إلا الله، وحده لا شريك له، له الملك، وله الحمد، وهو على كل شيء قدير

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