physiology

premed 2018 - JU

◉ Sheet ○ Slides

Number

22

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Last time we talked about regulation of body fluids mechanisms by regulating:

1-volume

2- osmolality

For example: levels of angiotensin II, ADH…. etc.

Now let's talk about ANP which is involved in regulatory mechanisms (output regulation)

ANP: Atrial natriuretic peptide

Atrial: related to atrium (so ANP released from the heart)

Natriuretic: sodium (involved in sodium homeostasis)

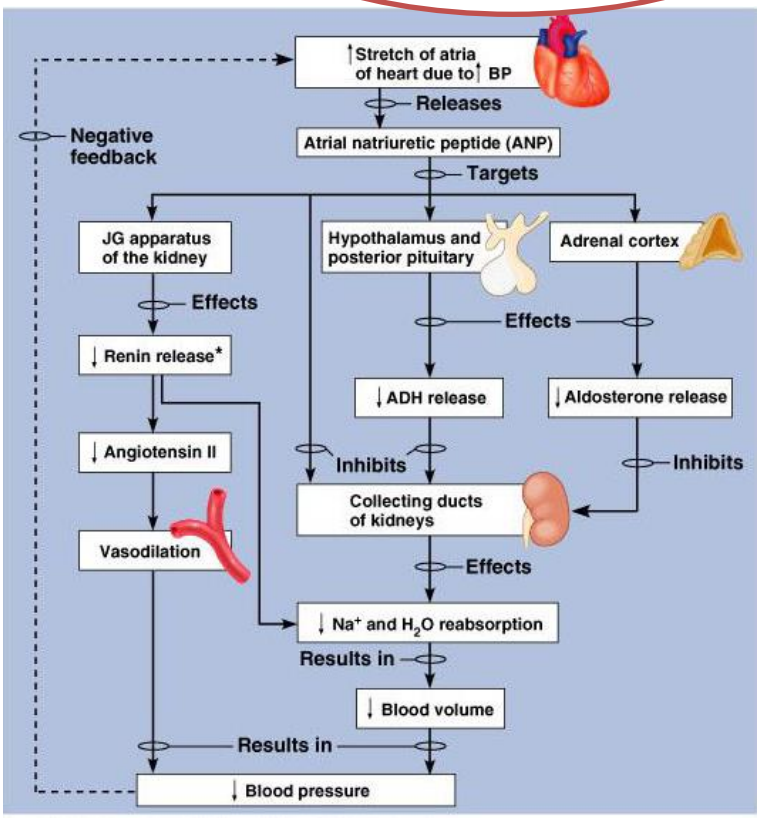
Note: the doctor said the details in the figure below is not required just focus on what's written beside it

we are interested in effects of ANP over blood vessels:

there are many effects of ANP that causes reducing blood pressure; and that's by interfering with many mechanisms for example

1-it **reduces the release of the ADH** from the posterior pituitary, **and aldosterone** from the adrenal cortex which reduces the efficiency of the salts and water reabsorption by the kidneys resulting in a decrease in the blood volume and consequently reducing its pressure

2-it **reduces the angiotensin II levels** which result in vasodilation (remember angiotensin II can function as a vasoconstrictor)



Vasodilation →less resistance →lowering blood pressure

Now we are going to discuss some disorders of **volume** and **osmolality**;

1- Changes the could happen in our body about osmolality (mainly related to sodium)

a- Hyponatremia

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

b- Hypernatremia

Results by excessive intake of Na+ or administration of hypertonic fluids

2- Changes the could happen in our body regarding volume

a-Hypovolemia

Results by excessive loss of fluids

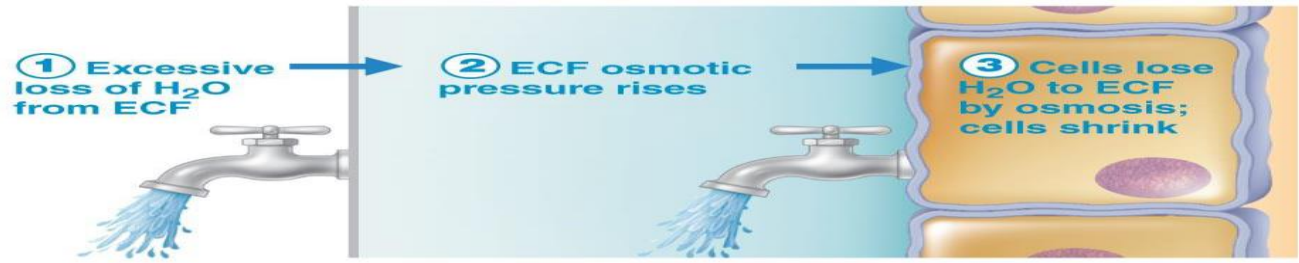
b- Hypervolemia

Results by excessive intake or administration of fluids

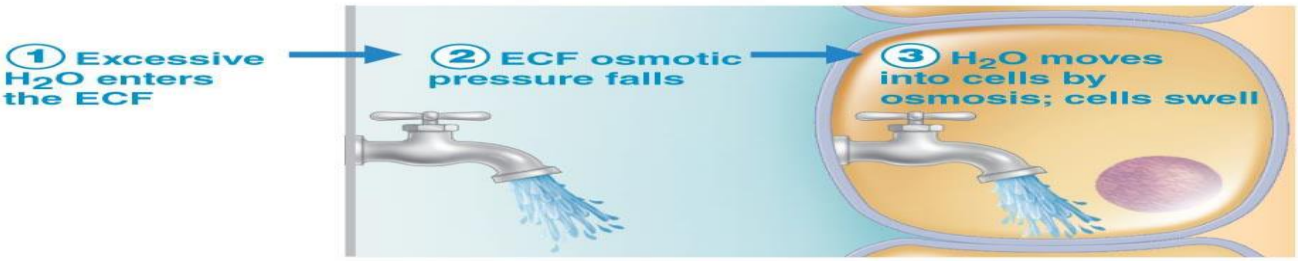
Note: **Overhydration and dehydration vs hypervolemia and hypovolemia**:

Dehydration: loss of H2O only ……. Hypovolemia: loss of H2O and solutes.

Overhydration: gain of H2O only ……. Hypervolemia: gain of H2O and solutes.



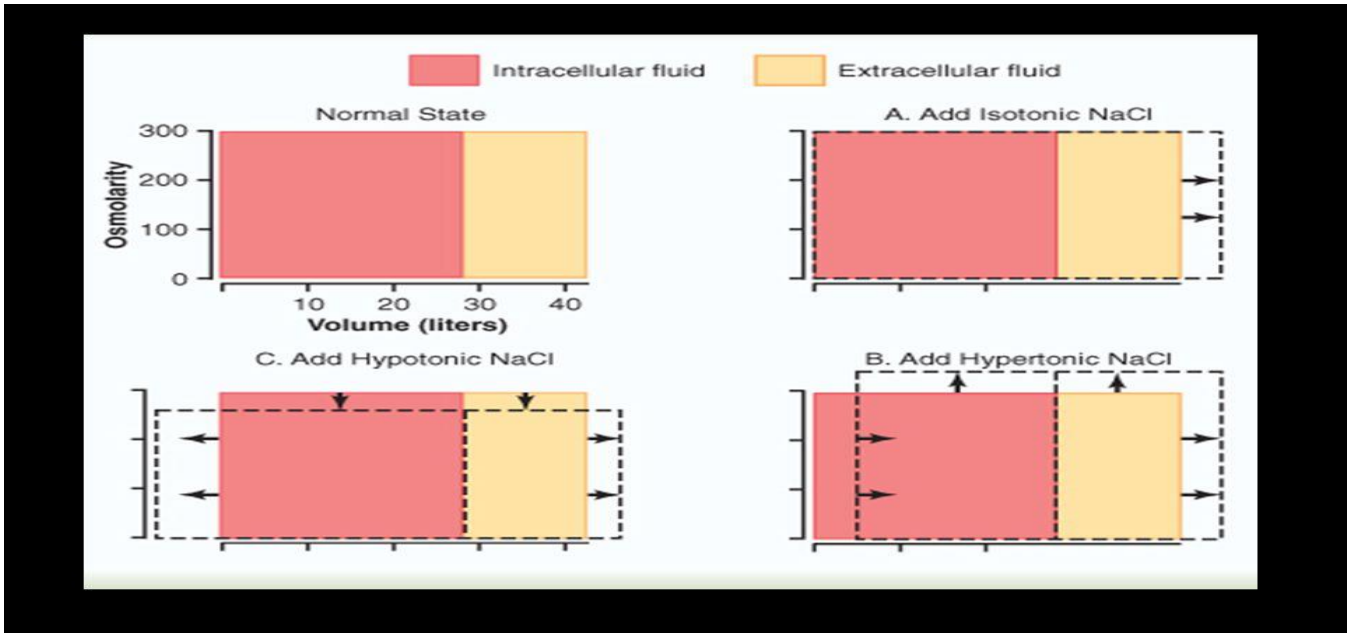
(a) Consequences of dehydration. If more water than solutes is lost, cells shrink.



(b) Consequences of hypotonic hydration (water gain). If more water than solutes is gained, cells swell.

This slide has been mentioned earlier so the doctor skipped it

Administration of fluids: infusion of fluids to ECF.



Now let's discuss every situation individually;

A. Adding isotonic solution to the ECF →increased ECF volume

Why?? When we add an isotonic solution the osmolarity won't be affected so we are just adding volume to the ECF. (no fluid exchange between the ICF and ECF).

B. Adding hypertonic solution to the ECF → increased ECF volume and osmolarity

decreased ICF volume, increased ICF osmolarity

why?? When we have an administration of a hypertonic solution: firstly we are having an increase in the volume of the ECF, and because its hypertonic (high conc. Of salts) we can also see an increase in its osmolarity which leads to the movement of H20 from the ICF to the ECF by osmosis meaning that the volume of the ICF will be decreased and its osmolarity will be heightened (the same amount of salts will remain in a lower quantity of H2O)

C. Adding hypotonic solution to ECF→increased ECF volume, decreased ECF osmolarity

increased ICF volume, decreased ICF osmolarity

why?? when we add an hypotonic solution: firstly we are having an increase in the volume of the ECF, and because its hypotonic we can also see a decrease in its osmolarity which leads to the movement of H20 from the ECF to the ICF by osmosis meaning that the volume of the ICF will be increased and its osmolarity will be lowered(the same amount of salts will be in a higher quantity of H2O).

** notice that the ECF volume gets larger in all cases (because we are adding the solution to it)

After discussing the cases of disorders, can these disorders take place simultaneously?

Yes, and those are some possible combinations:

–Hyponatremia with dehydration

–Hyponatremia with overhydration

–Hypernatremia with dehydration

–Hypernatremia with overhydration

–hypernatremia with isovolemia

–isonatremia with hypervolemia

Remember:
Iso = normal
Hyper = high
Hypo = low

How they can appear?

For clarification let's have a look on some of them:

Hyponatremia with overhydration: may be caused by excessive intake of water, also by high release of ADH (note that ADH causes the reabsorption of H2O only).

Hypernatremia with dehydration: decreased level of ADH.

Hypernatremia with overhydration: If we have increased aldosterone levels. (if there is intake of water).

Hypernatremia with isovolemia: increased levels of aldosterone without dinking any more water (we are just absorbing salts due to lack of water so there is no considerable change in volume).

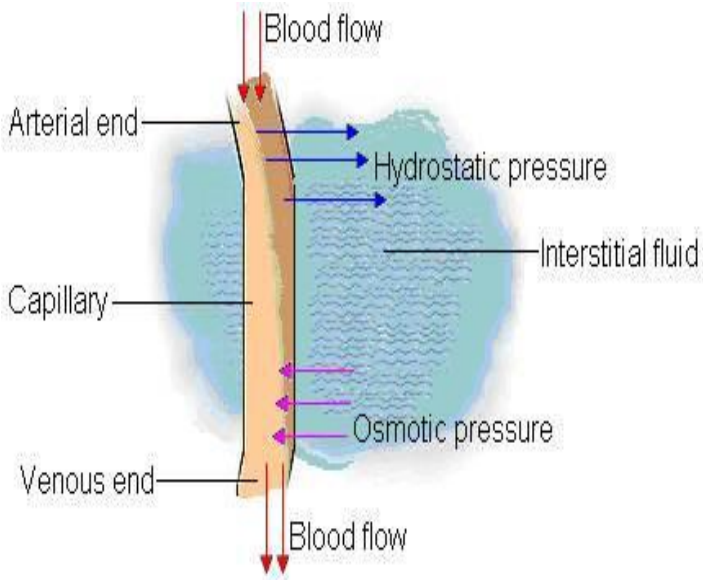
Hypervolemia with isonatermia: administration of isotonic solution.

Oedema

Oedema: accumulation of fluids in the interstitial compartment.

There is a dynamic movement of interstitial fluids toward cells, vessels, etc. this dynamic movement is caused by both colloid osmotic and hydrostatic pressures.

To make better understanding for Oedema let's look at this capillary:



each capillary has two ends arterial and venous, blood flow in the capillary is influenced by colloid osmotic and hydrostatic pressures.

Blood pumped by the heart passes along the arteries then into narrower arterioles and then finally into capillaries so this creates a pressure called the hydrostatic pressure at the arterial end of the capillary and this tend to force the liquid out of the blood towards the interstitial fluid, there are many other forces that opposes this for example the hydrostatic pressure of the interstitial fluid and the lower water potential of the blood due to the plasma proteins (high oncotic pressure of the blood)but the combined effects of all these forces tend to favor the net movement of fluids out of the capillaries at the arterial end. This process is called filtration, so you can conclude that as the hydrostatic pressure inside the capillaries increases, the filtration increases. Now as fluids are lost the pressure in the capillaries reduces so as we are reaching the venous end of the network the hydrostatic pressure is lowered, now the colloid osmotic pressure of the capillary will force the fluid to move back from the interstitial fluid to the blood capillary. This process is called reabsorption.

to sum up

At the arterial end —> higher hydrostatic than osmotic pressure—> filtration

At the venous end —> higher osmotic than hydrostatic pressure—>reabsorption

Note:

in normal cases the colloid osmotic pressure of the blood doesn't change considerably, so the variable here is the hydrostatic pressure and its capability of overcoming the osmotic pressure.

The colloid osmotic pressure here appears due to the oncotic pressure which is a form of osmotic pressure induced by proteins, so don't be confused although they're not the same but we can use them interchangeably here.

Now let's talk about oedema, so as we said the oedema is the abnormal accumulation of fluids in the interstitial compartment so what causes this accumulation?

Causes of oedema (there are many causes we will go through them in details)

A- increasing capillary filtration:

this is caused by

1-increased capillary hydrostatic pressure:

- kidney causes: more retention of water and salts (renal failure)
  so, when the kidney reabsorbs more water and salts than normal, the blood volume and hydrostatic pressure will increase which ends in an increase in filtration and the appearance of oedema. (this may also cause hypertension)

- excess of mineralocorticoids(aldosterone)
  aldosterone increases the retention of water and salts by the kidney, so the same procedure as said above will happen.

2- High venous pressure

- Heart failure, decrease of venous return (obstruction, decreased venous pump activity)
  Here The heart fail to pump blood normally from the veins into the arteries, which raises venous pressure(hydrostatic), causing increased capillary filtration (decreasing the reabsorption). This also leads to a reduce of blood flow to the kidneys and as we have seen before this will stimulate the renin, angiotensin, aldosterone pathways which causes an additional retention of water and salts and serious developing of oedema.

3- Decreased arteriolar resistance

Now what do we mean by decreasing resistance?
Suppose we have a tube with flowing fluids inside, any increase in the diameter of this tube will actually decrease the resistance and vice versa any decrease in the diameter of this tube will increase the resistance. So, when the arterioles are dilated the resistance is decreased and we are having more flow of blood at the arterial end which gives us more surface area for filtration and thus the appearance of oedema but what causes this vasodilation of arterioles?

- Excessive body heat (to increase sweating and vaporization)
- Insufficiency of sympathetic nervous system (any error or decrease in the SANS tone can leave behind more dilated arterioles.)
- vasodilators

B- decreasing oncotic pressure

Remember that: oncotic pressure is caused by the presence of proteins in plasma inside blood vessels and capillaries.

failure to produce normal amounts of proteins or leakage of proteins from the plasma causes the oncotic pressure to fall. This leads to increased capillary filtration throughout the body and oedema.whyy?

because when we reduce the amount of proteins in the plasma and blood the osmolarity (oncotic, colloid osmotic pressure) will decrease meaning that fluids and water will leave the capillaries (filtration) trying to restore this change in the osmotic pressure.

Now what can cause such a decrease in the protein numbers

1- increased loss of proteins

- From Kidney in **nephrotic syndrome**

Nephrotic syndrome: high protein content in the urine; some people may have some structures that prevents protein reabsorption, the membranes become leaky to plasma proteins and often allowing large quantities of these proteins to pass into the urine.

- from skin in burns and severe wounds:
Severe wounds like accident; body loses so much blood with serum, body tries to recruit this, but it only makes blood vessels not serum.

Serum = protein rich liquid, separates out when blood coagulates

2- decreased production of proteins:

- Liver diseases (disfunction of protein synthesis): Which results in a decrease in oncotic pressure and oedema at certain locations.

- Decreased intake of proteins in malnutrition → less protein in plasma → less oncotic pressure.

C- Increasing capillary permeability

By increasing the permeability of capillaries, we are having more filtration because more fluids can move through, and at the same time were permitting the movement of proteins from the plasma to the interstitial fluid which reduces the oncotic pressure and leads to oedema, so what causes this increase in permeability?

1-During immune reactions by release of histamine

Here we have two factors combined, the first one is that immune reactions lead to vasodilation which as we discussed earlier is responsible for oedema. And the second one is that the immune products such as histamine can lead to an increase in the capillaries permeability which also develops oedema

2-Toxins and Infections

Here our body needs the white blood cells and the antibodies inside the blood to fight infections, so we must increase the permeability of the capillaries to allow these cells to move to the infected area. This is an effect of the cytokines which are released as inflammatory particles and causes this change in permeability.

3- Vitamin C deficiency

4-Ischemia

5-burns

D- Decreased lymph drainage

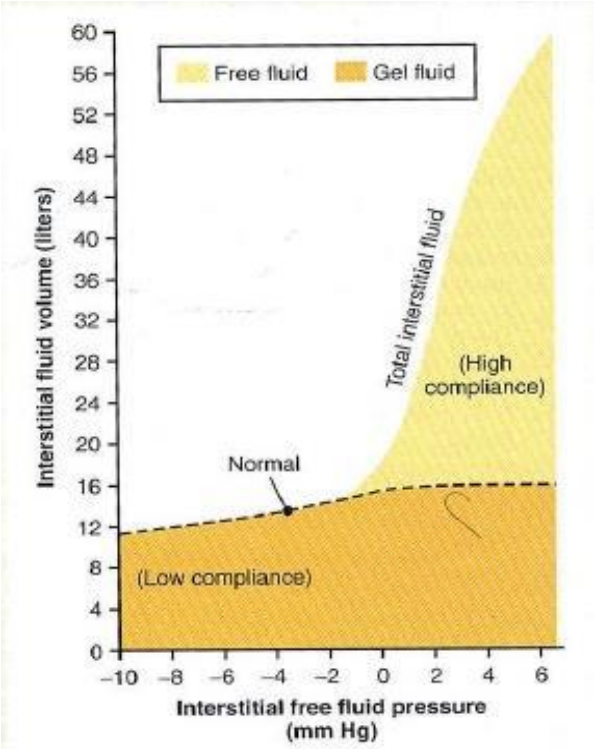
A major function of the lymphatic system is to return to the circulation the fluid and proteins filtered from the capillaries, and without this continuous return to the blood the plasma volume will be depleted and oedema will occur. Now what can cause this decrease in the lymphatic drainage?

1- Cancer: may block the lymph vessel →preventing proteins wash.
2- Infection: for example, elephantiasis (the enlargement and hardening of limbs or body parts and its characterized by oedema because of a decrease in the lymphatic drainage)
3- Surgery: extensive removal of lymphatic vessels
4- Absence or abnormality of lymphatic vessels

**** In our body there are safety factors for preventing Oedema:**

•Low tissue compliance
•Increased lymph flow
•Increased protein wash-down from interstitial fluids

•Low tissue compliance



Interstitial free fluid normally exists in negative pressure ranges and shows low compliance.

Low compliance: a small change in the interstitial fluid volume results in a big change in interstitial fluid hydrostatic pressure.

Higher interstitial hydrostatic pressure makes the movement of fluids from the capillary towards the interstitial compartment hard →decreasing filtration→ preventing fluids accumulation in interstitial compartment → prevents Oedema.

Note that the hydrostatic pressure inside the capillary and the interstitial hydrostatic pressure are opposed to each other for example an increase the hydrostatic pressure inside the capillary will increase the filtration while an increase in the interstitial hydrostatic pressure will decrease the filtration

** In positive pressure ranges (Not normal)

High compliance

Once interstitial fluid pressure rises above the 0 the compliance of the tissues increases markedly, allowing large movement of fluids to accumulate in the tissues with relatively small additional increases in interstitial fluid hydrostatic pressure; thus, in the positive range this safety factor is lost → pitting oedema

Pitting Oedema: observable swelling of body tissues due to fluid accumulation that may be demonstrated by applying pressure to the swollen area (such as by depressing the skin with a finger)

- Increased lymph flow

Lymph flow can increase up to 10-50 folds Carrying away large amounts of fluids → prevents interstitial pressure from rising into POSITIVE ranges

- Increased protein wash-down from interstitial fluids

Carry away large amounts of proteins (Protein washed out from interstitial fluids) → decrease Colloid osmotic pressure in interstitial fluid → Lowering net filtration forces → Prevents accumulation of fluids.

The end.

Good luck, happy birthday to you all.

Special thanks to Hasan Barakat and mutaz abuhnaneh for the motivation.