**unfortunately the sheet does not involve the slides.**

** the doctor repeat a lot of things from the previous lecture so this sheet will begin from slide 139 to 174.

** really I do my best and so sorry for any mistake.**

**there is some information from the book that the doctor mentioned it in a way or in another.

** in the previous lecture we are talking about countercurrent mechanism

** as you know loop of henle is composed of ascending and descending tubules, also vasa recta has the same

** anything coming from vasa recta to interstitium and then to the renal tubules is called secreted substances.

** anything coming from tubules to interstitium then to vasa recta is called reabsorbed substances.

slides 139,140,141,142

** keep in your mind that there is two things imp to initiate or generate the countercurrent exchanger " in another way contribute in hyperosmotic renal medullary interstitium":

1. Mainly is the sodium ions and other ions and solute with it " contribute in 60% "

2. Recirculation of Urea " contribute in 40%"

** now how the urea generate and recalculate?

As water flows up the ascending loop of Henle and into the distal and cortical collecting tubules, little urea is reabsorbed because these segments are impermeable to urea, In the presence of high concentrations of ADH, water is reabsorbed rapidly from the cortical collecting tubule and the urea concentration increases rapidly because urea is not very permeant in this part of the tubule. Then, as the tubular fluid flows into the inner medullary collecting ducts, still more water reabsorption takes place, causing an even higher concentration of urea in the fluid. This high concentration of urea in the tubular fluid of the inner medullary collecting duct causes urea to diffuse out of the tubule into the renal interstitium.

The simultaneous movement of water and urea out of the inner medullary collecting ducts maintains a high concentration of urea in the tubular fluid and, eventually, in the urine, even though urea is being reabsorbed.
Recirculation of urea absorbed from the medullary collecting duct into the interstitial fluid. This urea diffuses into the thin loop of Henle, and then passes through the distal tubules, and finally passes back into the collecting duct. The recirculation of urea helps to trap urea in the renal medulla and contributes to the hyperosmolarity of the renal medulla. The heavy dark lines "in above picture", from the thick ascending loop of Henle to the medullary collecting ducts, indicate that these segments are not very permeable to urea.

** note that the rate of urea excretion is determined mainly by two factors: (1) the concentration of urea in the plasma and (2) the glomerular filtration rate (GFR) slide 143,144

** vasa recta not involve in generate countercurrent mechanism but only to maintain it.

** vasa recta is a pritubular capillary present in deep medullary nephron not in the cortical nephron.

** the countercurrent mechanism is mainly to concentrate urine when there is defect of water in the body so the excretion of urine significantly decreased and reabsorbtion of water increased.

** the minimum amount of urine to formed with highest osmolarity is about .5 litter

* the maximum osmolarity of urine is 1200 osm/l.

** in loop of henle the more deeper is going in descending the more osmolarity because the
reabsorption of water but the vice versa in ascending " as you going in ascending the osmolarity decreased " because the reabsorption of sodium and other solute.

** note that anything get out from tubules into interstitium either water from descending tubules or sodium from ascending it will removed by vasa recta to maintain the countercurrent mechanism going on as we said.

** this picture below will explain to you what I mean 😊

** one student asked " is the vasa recta only in the distal medullary nephron ?" the doctor answered him " in cortical there is pricapillary but not the vasa recta, so the concentration not much as medulla "

** IF WE NOT NEED WATER what happen is:

1. The medullary will not be functioning and this will be mainly regulated by the cortical only

2. Vasa recta have to keep functioning but in a minimum level, the blood flow in vasa recta is only 1-2% of renal blood flow " this enough to keep it function in minimal level " and sure its function will increase when there is a shortage of water to reduce the urine formation.

slide 145

** note that in loop of henle the minimum osmolarity is 600 while the maximum is 1200
**as we said collecting duct and distal tubule depend on level of ADH**

*With presence of ADH, these tubules are highly permeable to water, and significant amounts of water are reabsorbed. Urea, however, is not very permeant in this part of the nephron, resulting in increased urea concentration as water is reabsorbed so this will "preserve the high medullary interstitial fluid osmolarity"

*with absence of ADH this part still impermeable to water.

**The U-shaped structure of the vessels minimizes loss of solute from the interstitium but does not prevent the bulk flow of fluid and solutes into the blood through the usual colloid osmotic and hydrostatic pressures that favor reabsorption in these capillaries. Thus, under steady-state conditions, the vasa recta carry away only as much solute and water as is absorbed from the medullary tubules, and the high concentration of solutes established by the countercurrent mechanism is maintained

** one student ask " what we mean by starling capillary circulation ?"

the doctor answered him " it is the bulk flow in capillary ( the difference between two forces the capillary force and the interstitium force)"

**two primary systems are especially involved in regulating the concentration of sodium and osmolarity of extracellular fluid:
(1) the osmoreceptor-ADH system (2) the thirst mechanism.

** when there is shortage of water → ADH will increase → the absorption of water increase → the urine formation decrease .
* remember that ADH always try to save water and prevent the dehydration.

**on the other hand , if there is too much water , the body now need no more water so → no more ADH →more urine formation with low osmolarity " hypotonic urine"
**when you too much salt , the osmolarity will increase so there is two mechanism occur to bring the osmolarity to normal :**

1. Increased osmolarity → will stimulate the posterior pituitary to secrete ADH → increase level of ADH → increase water reabsorption → increasing ECF and bring osmolarity to normal.

2. Referring to slide 152, the same area along the anteroventral wall of the third ventricle that promotes ADH release also stimulates thirst center → increase water intake (drinking water) → bring osmolarity to normal and increase the ECF.

**as you see above the two mechanism increase the ECF and we dont need this increasing we only need to bring osmolarity normal so what happen to return the ECF normal is as follow :**

increase ECF → increase glomerular filtration → increase the load to descending tubule → increase load to the macula densa → decreased the angiotensin ii and aldosterone then decreased of ADH → this will reduce reabsorption → bringing the ECF to normal.

**so we eat too much salt drink so many water but the osmolarity and ECF must be always at normal level even when the opposite happened " when there is a dehydration , no water "**

**This what we called THIRST-ADH MECHANISM**

**as you know the urine osmolarity depends on ADH level lets repeat it again 😊 when the level of ADH is high → less urine with high osmolarity and vice versa .**

**note that the urea is just keep the hyperosmolarity in interstitium (renal medulla ) not in the renal collecting duct " do you remember urea recirculation ? plz go back to slide 141 because the doctor repeat it again and again and again ..😊 "**

**Urine Specific Gravity = Weight of solutes in a given volume, determined by size and numbers of solute molecules.**

**Urine osmolarity = Determined only by the number of solute molecules in given volume.**

slide 153

**anything decrease the ECF or increase the osmolarity will stimulate the thirst center .**

slide 154

**if one of them "ADH or thirst " are not working the other one can compensate for the osmolarity to bring it to normal but when the both are not working there nothing can bring the osmolarity to normal .**

**angiotensin ii and aldosterone increase both of sodium and water so they are not**
involved in regulation of concentration, they rather involved in regulation of Bulk "volume"

slide 155

** salt appetite mean that you take salt more than your need

** its mechanism to compensate decrease the ECF... HOW?!?

decreasing in ECF, [Na⁺], blood volume or BP all will stimulate salt appetite → so you will take more salt → this will increase the osmolarity → stimulate thirst center → so increase the ECF and bring osmolarity to normal as well.

slide 157, 158, 159, 160, 161

** the doctor read them as they are

slide 162

** in collecting duct we have two type of cells:
1. Principle cells → responsible for secretion of K⁺ in distal tubule and collecting duct.
2. Intercalated cells → which also have two type A and B, this cells can reabsorbed K⁺ by type A or secrete K⁺ by type B; depending on concentration of K⁺.

slide 163

** note that taking too much potassium it has no much effect as taking too much sodium.

** some people eat more fruit than protein so [K⁺] is much higher than [Na⁺] to keep hyperosmolarity in interstitium, so those people not vulnerable for hypertension or cardiovascular disease

slide 164

** chronic acidosis cause increase K Loss by (dec proximal tubule Na,Cl,H₂O absorption) → so the bulk flow coming to distal tubules increase → and this will reduce the aldosterone → and decreasing in aldosterone lead to → decrease the Na⁺ and then affect on secretion of K⁺
** increasing salt intake (mean increase the [Na+] ) \(\rightarrow\) increasing glomerular filtration rate \(\rightarrow\) decrease reabsorption of Na+ \(\rightarrow\) increase the flow in distal tubules \(\rightarrow\) which will increase the secretion of K+ ( the two K+ and Na+ are opposite each other when one increase the other decrease )

slide 165
The doctor read it

slide 166 -174

** The doctor read them as they are

read this note after finish the slide 171 ** as we said previously the Ca+2are not freely filtrated to the glomeruli rather it is bind with plasma protein or complexes with anions such as phosphate.