جامعة الملك سعود
King Saud University

Index:
- Important
- Extra information
- Doctor's notes
- Only in female slides
- Only in male slides

Tubular Reabsorption & Secretion

Lecture 5, 6

RENAL BLOCK

PHYSIOLOGY TEAM 437

Editing file

Objectives:

by the end of this lecture you will be able to:

- Define tubular reabsorption, tubular secretion, transcellular and paracellular transport.
- Identify and describe mechanisms of tubular transport.
- Revise tubuloglomerular feedback and describe its physiological importance.
- Describe tubular reabsorption of sodium and water.
- Identify and describe mechanism involved in Glucose reabsorption.
- Study glucose titration curve in terms of renal threshold, tubular transport maximum, splay, excretion and filtration.
- Identify the tubular site and describe how Amino Acids, HCO_3^-, PO_4^- and Urea are reabsorbed.

Tubular Reabsorption

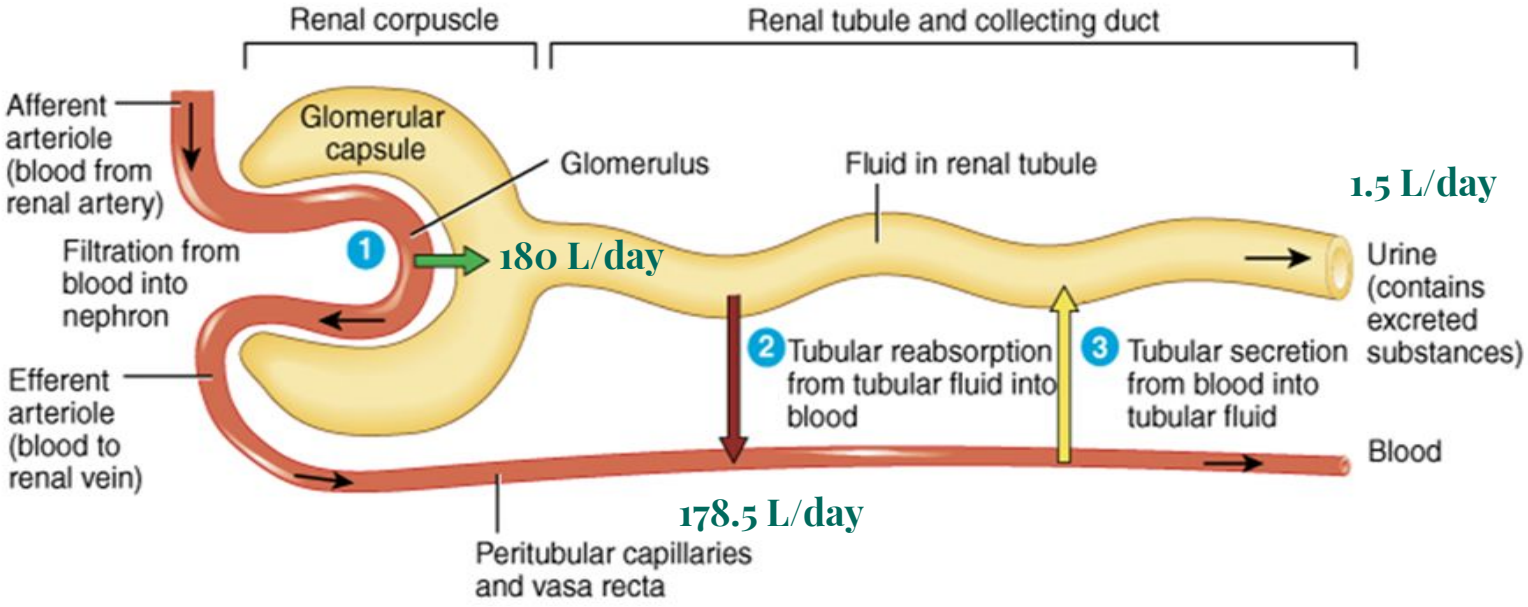
ONLY in female slides

Tubular Processing of Ultrafiltrate

- After glomerular filtration the ultrafiltrate gets modified as it passes through the tubules before it is finally excreted.
- Tubular processing includes:
  - **Tubular reabsorption** = reabsorption of substances from the glomerular filtrate into peritubular capillary blood.
  - **Tubular secretion** = secretion of substances from peritubular capillary blood into tubular fluid.
- What is the importance of tubular processing?

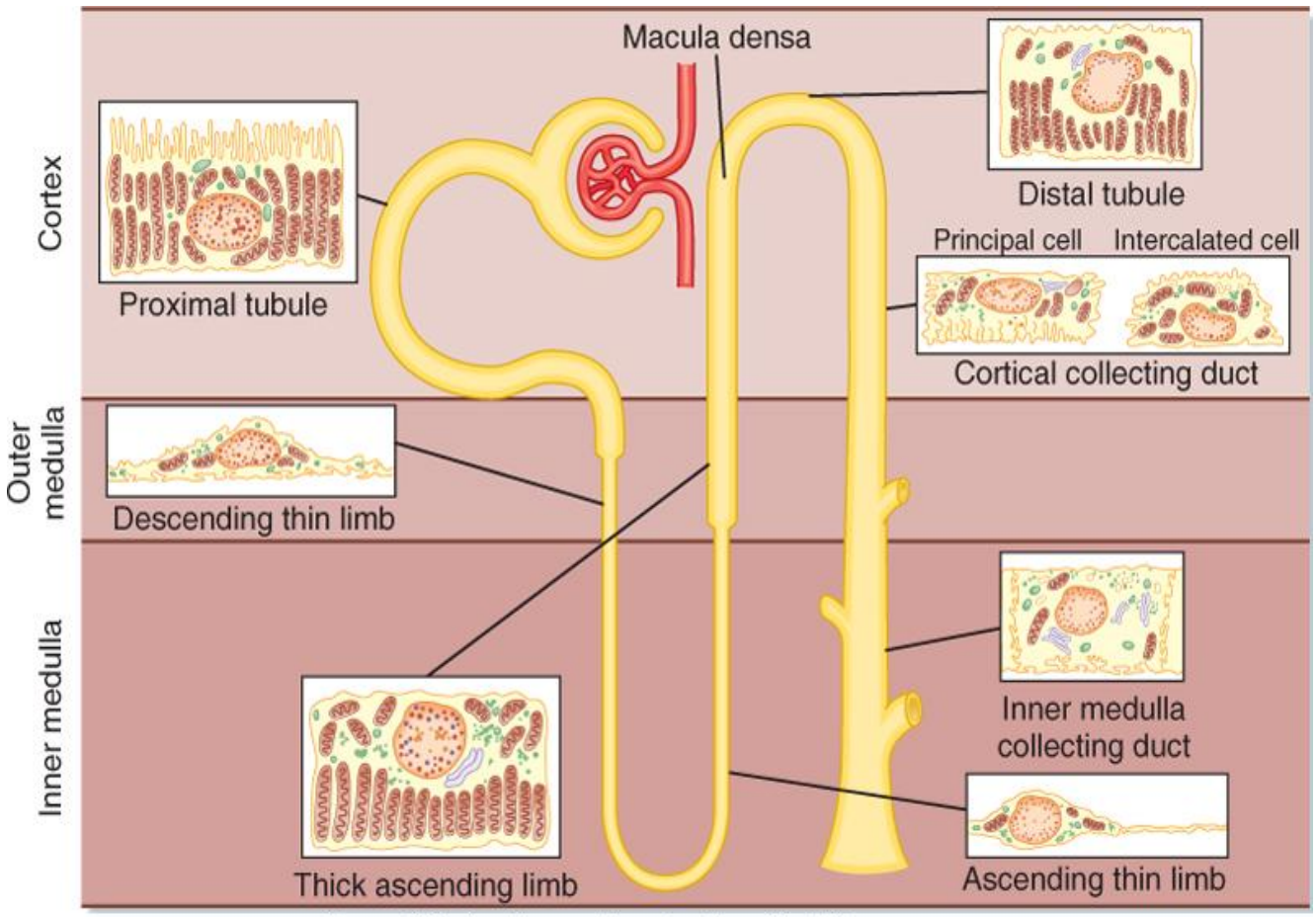
Tubular Reabsorption

- Glomerular filtration and tubular reabsorption are quantitatively very large relative to the amount excreted!
- Glomerular filtration is non-selective whereas tubular reabsorption is highly selective.



Differences in Renal Tubular Cells Reflect Their Function in Tubular Processing

ONLY in female slides



Transport Mechanisms Across the Tubule

Active Transport

- Requires energy
- Moves substances against their electrochemical gradient.

Primary active
Directly coupled to energy source.
e.g. Na^+-K^+ ATPase.
Na^+ - H^+ ATPase.

Secondary active
Indirectly coupled to energy source.
Carrier protein.
e.g. Glucose & amino acid.

Passive Transport

- Does not need energy.
- Moves substances down their electrochemical gradient.

Passive diffusion
Osmosis
Water Solutes like Cl-Urea

ONLY in female slides

How Does the Nephron Reabsorb Substances?

Reabsorption

2 Step Process

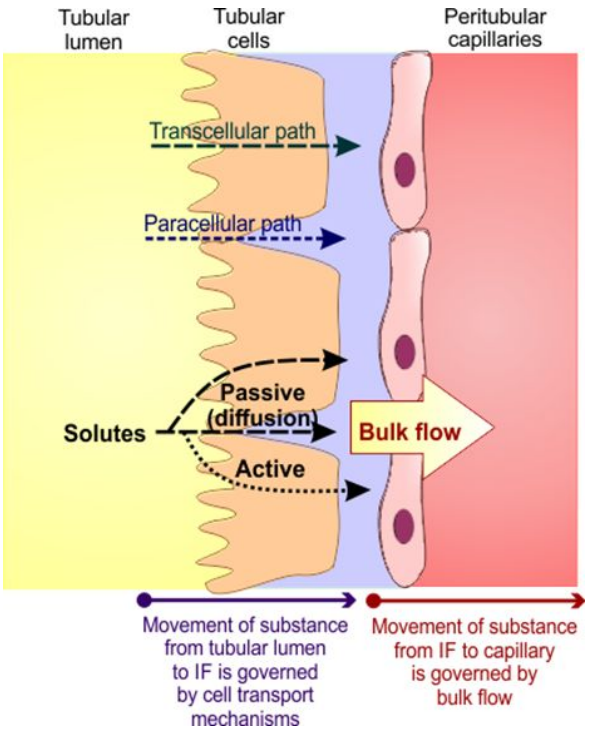
1-Transport of substances from tubular lumen to IF.

Transport involves ***Active & Passive*** mechanisms.

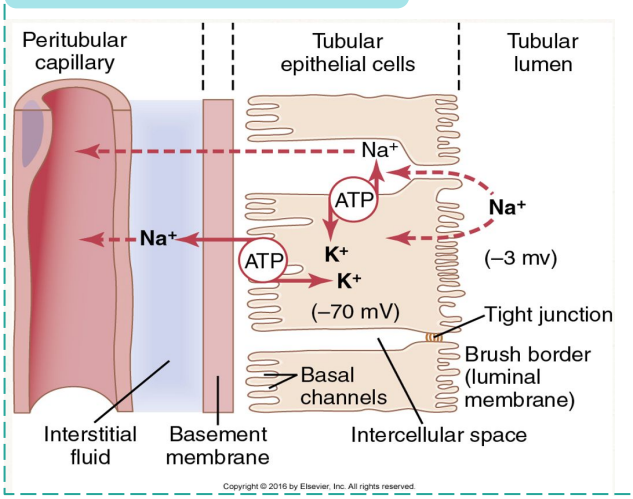
Occur through ***Paracellular*** and/or ***Transcellular*** routes.

2-Transport from IF to blood.

By ultrafiltration (bulk flow).



ONLY in male slides



Bulk flow results from the imbalance of osmotic or hydrostatic forces at the peritubular capillary.

Overview

TUBULAR REABSORPTION

Loops of henle

Descending limb

Ascending limb

Proximal Convoluted Tubule

Early part

Late part

Distal convoluted Tubule

Collecting Ducts

Early part

Late part

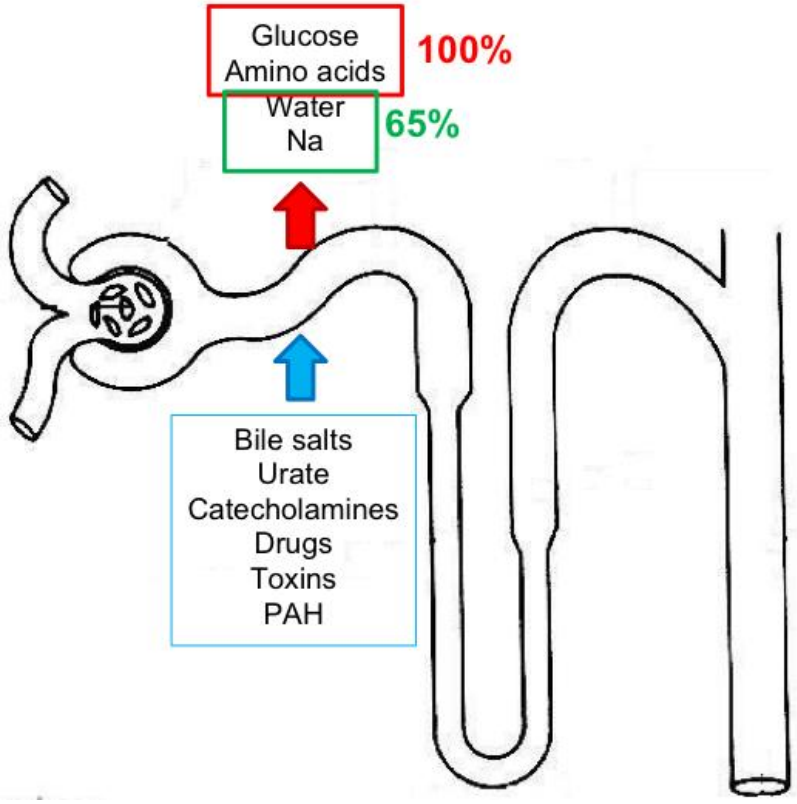
ONLY in female slides

Tubular Reabsorption in Each Part of the Nephron

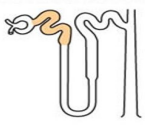
Proximal Convoluted Tubule

Most of the reabsorption occurs in the PCT.. ***Why***?

- Highly metabolic cells.
- Extensive brush border.
- Lots of mitochondria.



Early PCT



PCT Duration 4:34 mins

- **70%** of Na^+, Cl^-, K^+ and water.

1. **NHE**:

(Na^+ for H^+),, HCO_3^- reabsorbed. how?*

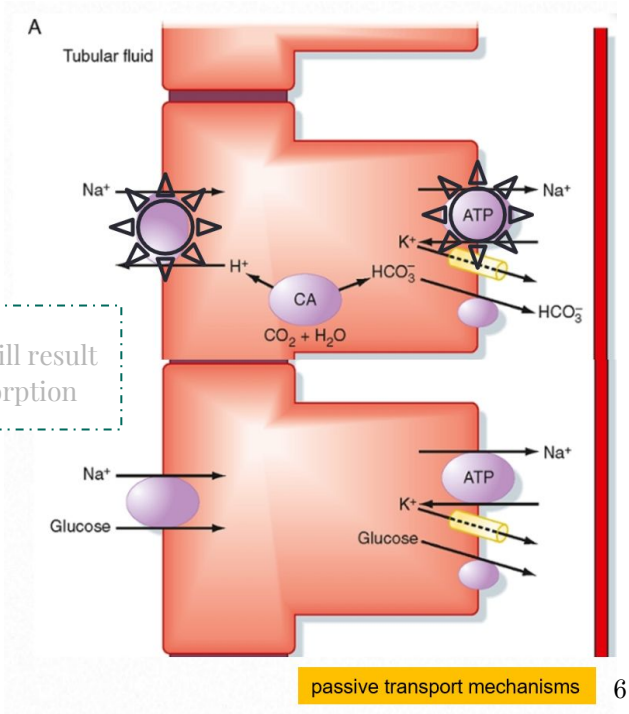
2. **Symporters (Cotransporters)**:

Na^+-glucose, Na^+-amino acid,

Na^+-Pi(Phosphate), Na^+-lactate.

- Organic molecules will be completely removed from the filtrate in the first half of the PT

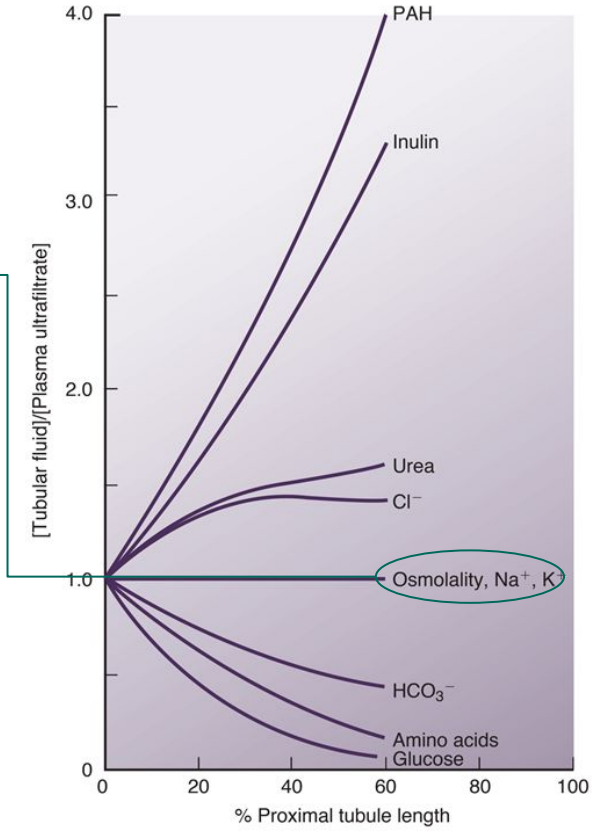
*H+ formation will result in HCO_3 reabsorption



passive transport mechanisms

Absorption in PCT

- The *amount* of Na^+ in the tubular fluid ↓↓ along PCT.
- The [Na^+] (and total osmolarity) remains relatively **constant.**
- Because water permeability of PCT is so great → **water reabsorption keeps pace with Na^+ reabsorption.**

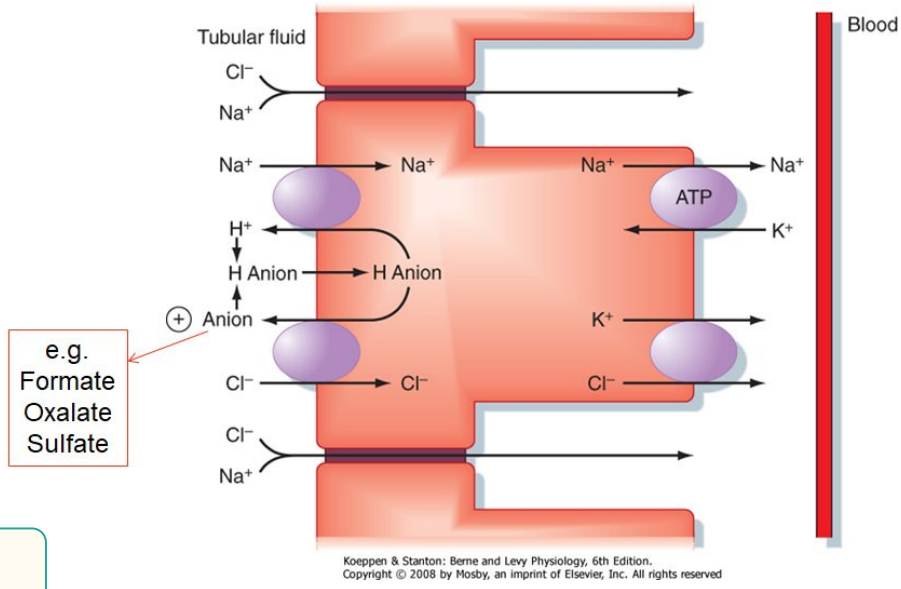


Inulin and PAH are less reabsorbed than Na or K. Bicarbonate, Amino Acids, and glucose are more reabsorbed than Na or K

Late PCT

Na^+ Reabsorbed with Cl^- . Why?

- Due to different transport mechanisms in late PCT.
- Lack of organic molecules.



By two methods

Paracellular "Directly"

Na^+ and **Cl^-** will be absorbed passively.

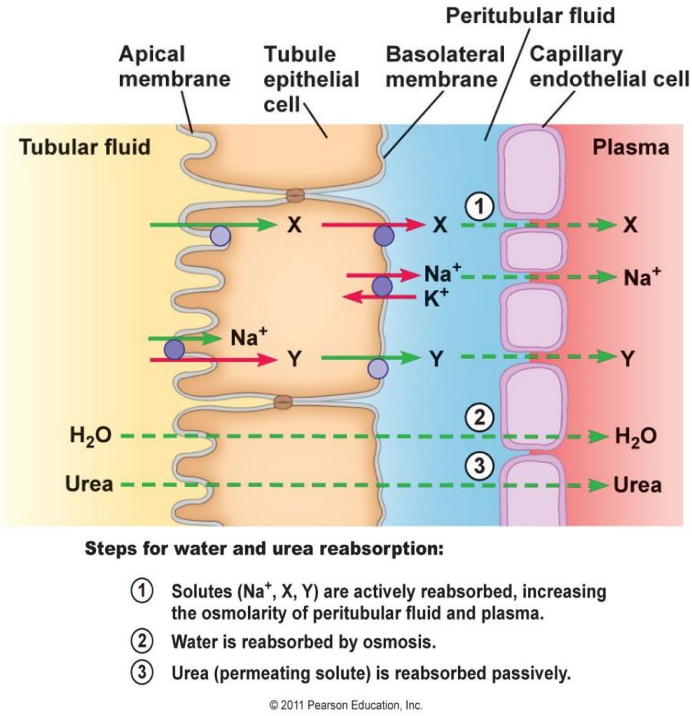
Transcellular "Indirectly"

H anion complex: this is reabsorbed by the tubules into the cell, when it become inside, it will disseminate to **H** and **anion**, Here we need to get rid of them to the tubular lumen again and that by:

- **Na^+ -H^+** Exchanger: **Na^+** inside the cell and **H^+** outside cell.
- **Anion -Cl^-** Exchanger: **Anion** outside the cell and **Cl^-** inside it.

Why the complex have to be formed outside? Because **H** and **Anion** are not permeable to the membrane, so the complex help them to be reabsorbed again.

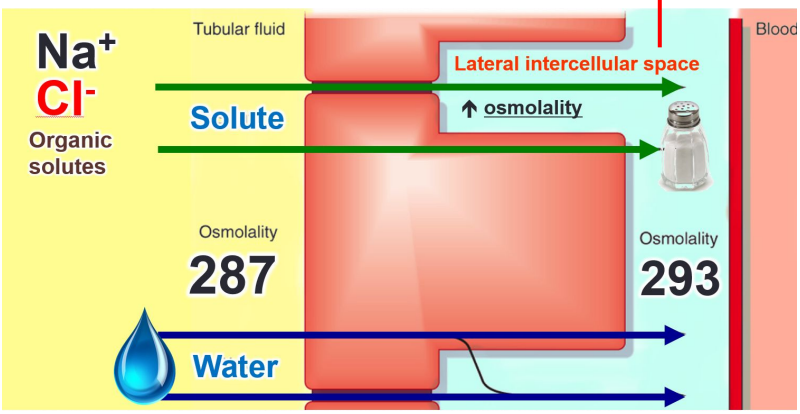
Water Reabsorption in the PCT



Steps for water and urea reabsorption:

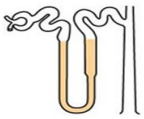
1. Solutes (Na^+, X, Y) are actively reabsorbed, increasing the osmolarity of peritubular fluid and plasma.
2. Water is reabsorbed by osmosis.
3. Urea (permeating solute) is reabsorbed passively.

The driving force for osmotic water reabsorption.



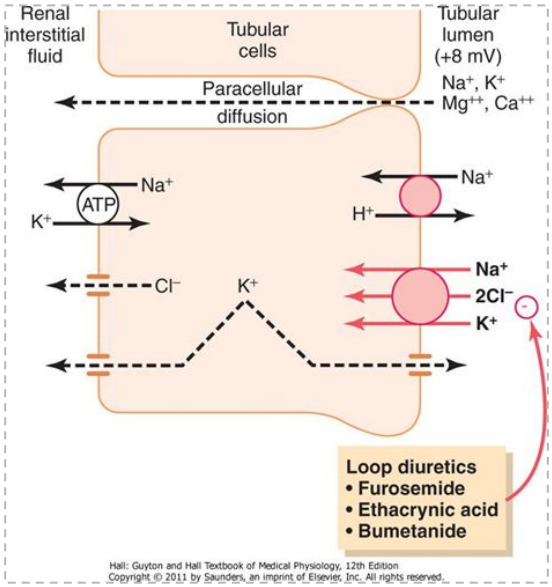
Passive, due to osmotically active substances that are absorbed.

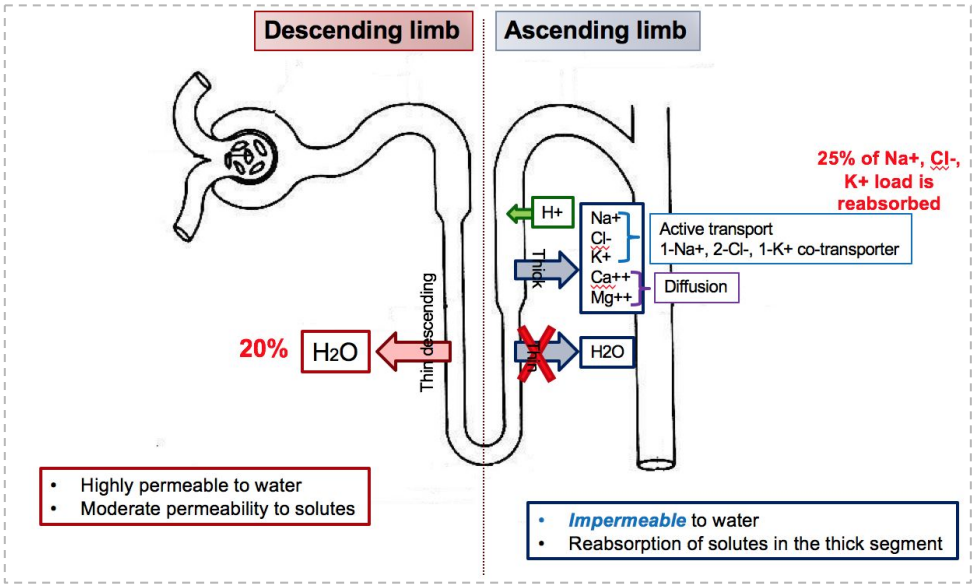
Loop of Henle



Sodium regulation in the thick ascending limb
Duration 6:06 mins

- **25%**of filtered **NaCl** and **K^+** is reabsorbed as well as Ca^{2+}, HCO_3^- in thick ascending limb (TAL)
- The ascending limb is **impermeable to water (diluting segment).**
- 15% water absorbed in thin descending limb (**permeable to water**)
- The descending thin limb **does not reabsorb NaCl.**





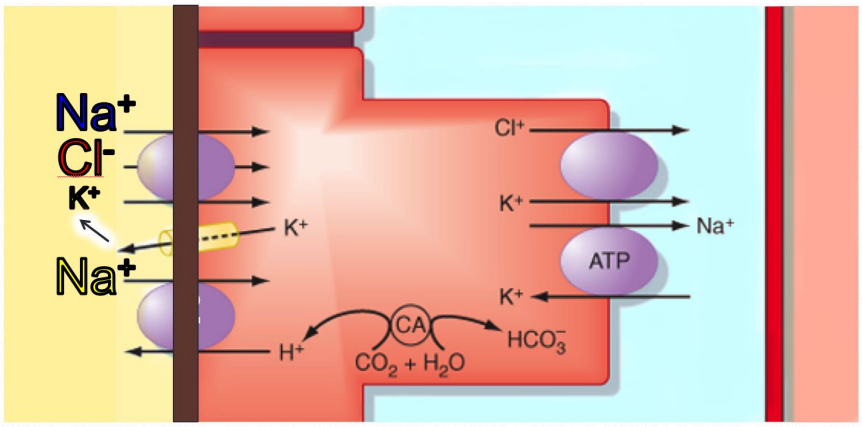
Solute Absorption

Ascending limb

1) **Transcellular (50%)**

- $1Na^+$-$2Cl^-$-$1K^+$ co-transporter
- NHE Na-H Exchange transporter

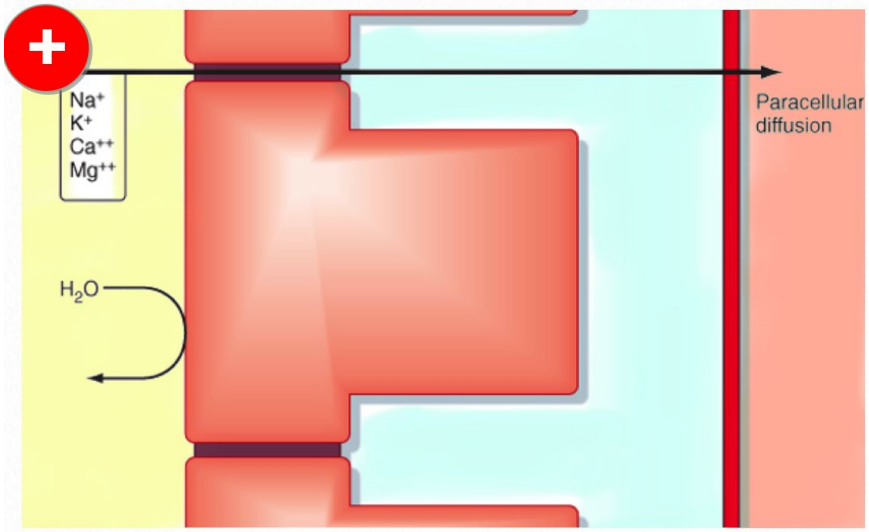
Bridge to pharma:
Loop diuretics work on this cotransporter.



Transport mechanisms for NaCl reabsorption in the thick ascending limb of Henle's loop. 19

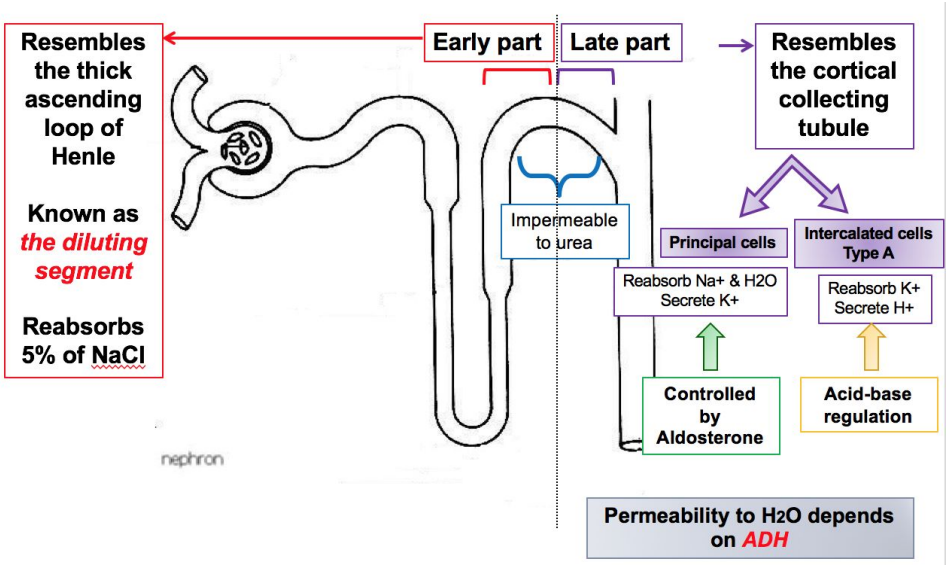
2) **Paracellular (50%)**

Loss of NaCl in tubule →↑ +ve compared to blood drives absorption



-↑ Salt transport by the thick ascending limb ↑- the magnitude of the positive charge in the lumen.

Distal convoluted tubule & collecting ducts

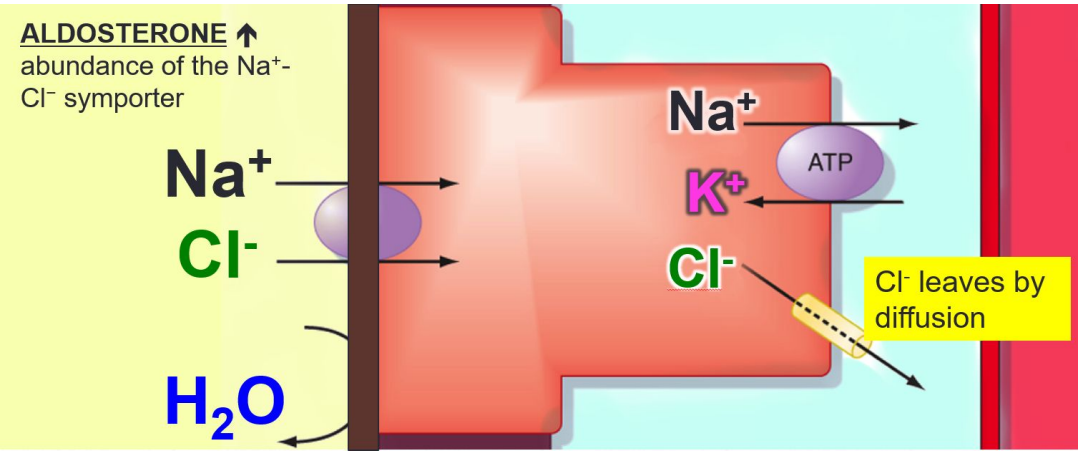


- Reabsorb **8%** of filtered NaCl, ~10% water (**needs ADH**).
- Some K^+, H^+ secreted ***into*** tubule.

Early DCT

- **This segment is impermeable to water.**
- Reabsorbs Na^+, Cl^- and Ca^{2+}

Bridge to pharma:
Thiazide diuretics work on Na/Cl transporter



Late DCT

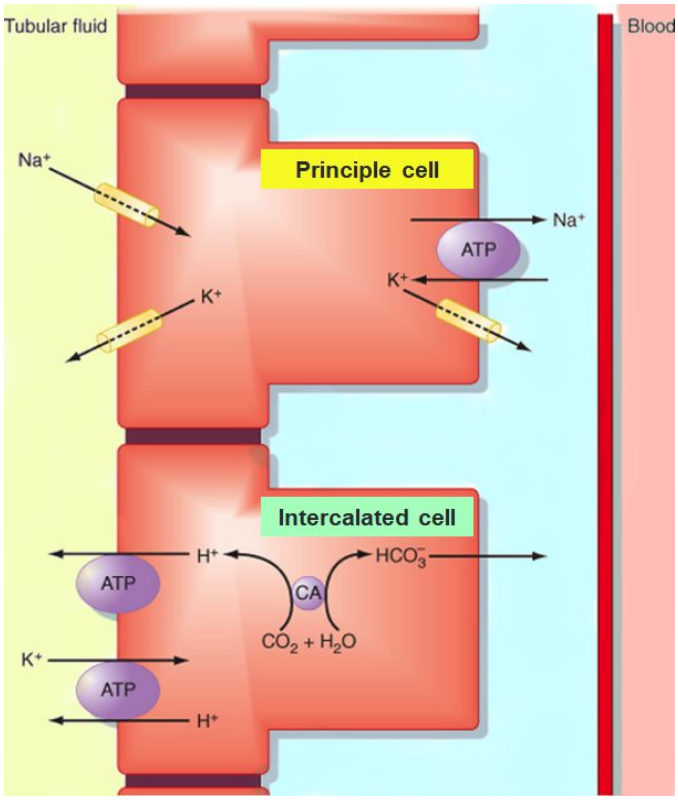
1) Principle cells: reabsorb Na^+, water, secrete K^+

2) Intercalated cells: secrete or reabsorb H^+ (opposite for HCO_3^-) [**important for acid base**], reabsorb K^+

- Na^+ diffuses via selective channels
- K^+ secreted down concentration, reabsorbed by an H^+/K^+-ATPase located in the apical (luminal membrane) cell membrane.

ALDOSTERONE increase NaCl reabsorption, **How?** **increase the amount of Na^+/K^+-ATPase** in the basolateral membrane.
↑ expression of the ENaC* in the apical cell membrane.

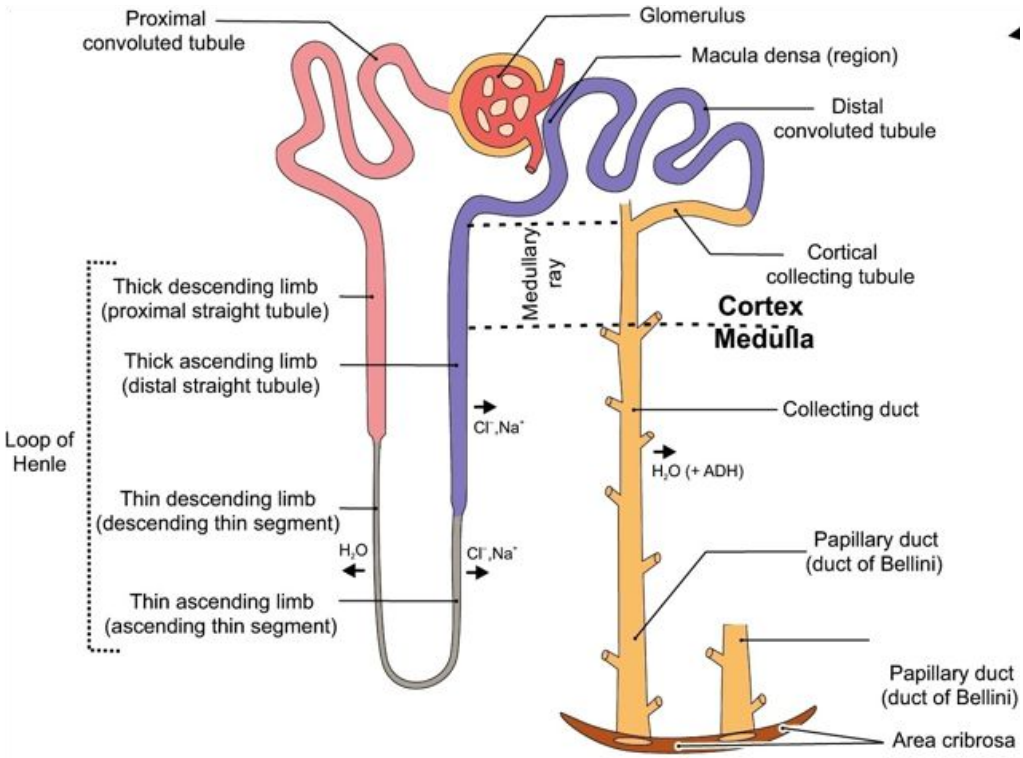
*: Epithelial Sodium Channels

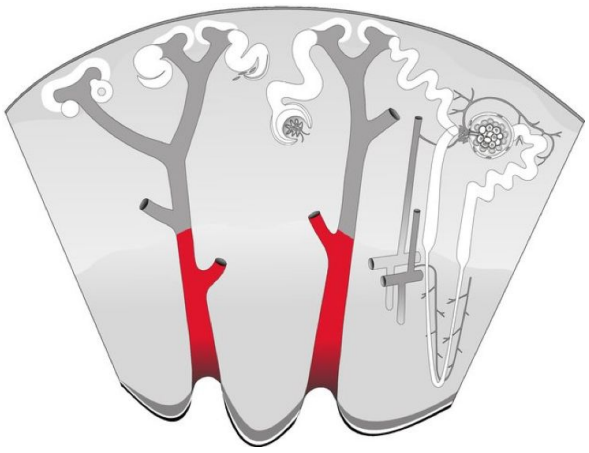


Bridge to pharma:
K sparing diuretics work in this segment of the nephron.

Medullary Collecting Duct

DCT and collecting duct
Duration 4:44 mins





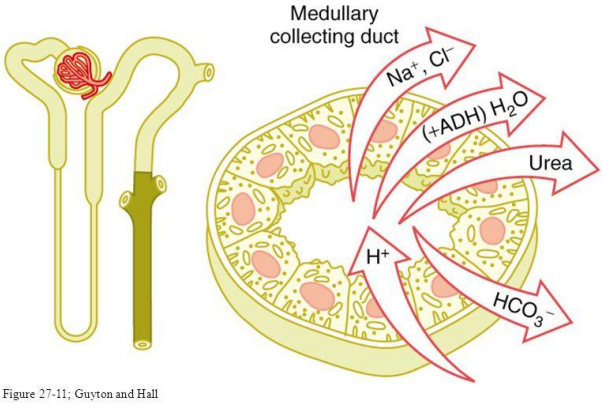


Figure 27-11; Guyton and Hall

- **Permeability to water is under ADH control**
- **Permeable to urea**
- **Secrete H+**

Regulation of Tubular Reabsorption

Regulation of tubular reabsorption depends on:

1. Physical forces that govern reabsorption.
2. Hormonal and neural mechanisms.

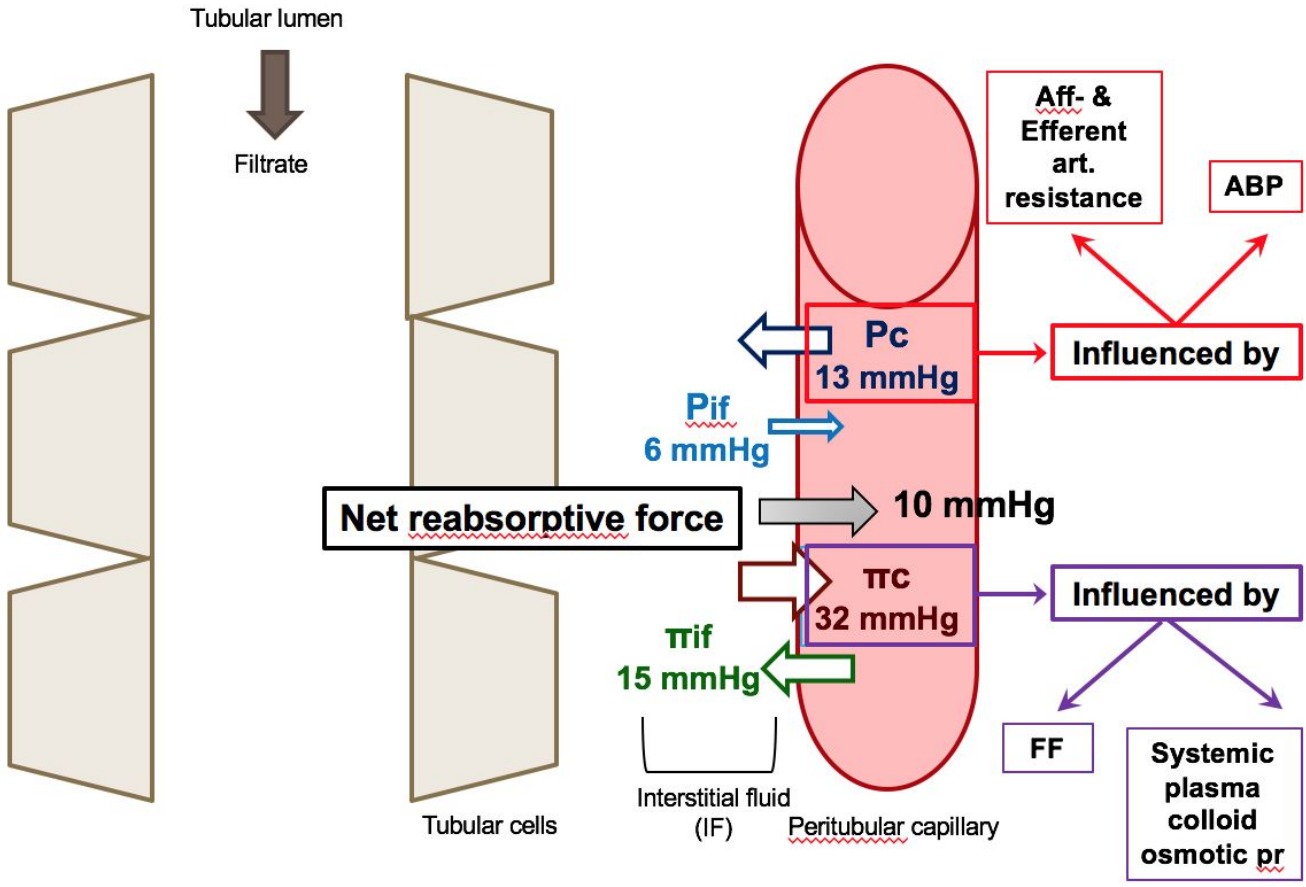
Tubules can increase their reabsorption in response to increased tubular load ↠ ***glomerulotubular balance.***

What are the physical forces that govern tubular reabsorption?

Starling Forces

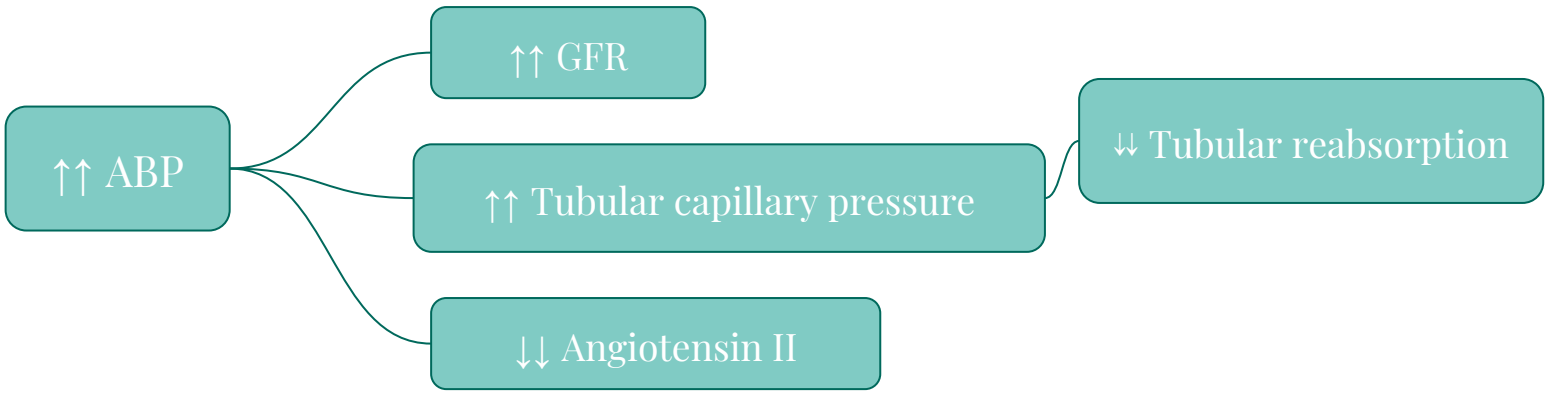
1- Physical Forces that Govern Tubular Reabsorption

P=hydrostatic pressure π=osmotic C=capillary if=interstitium



Pressure Natriuresis & Pressure Diuresis

- Increasing urinary excretion of Na+ and H2O in response to increases in ABP.
- Autoregulation should limit this! ***What happens if autoregulation is impaired?***



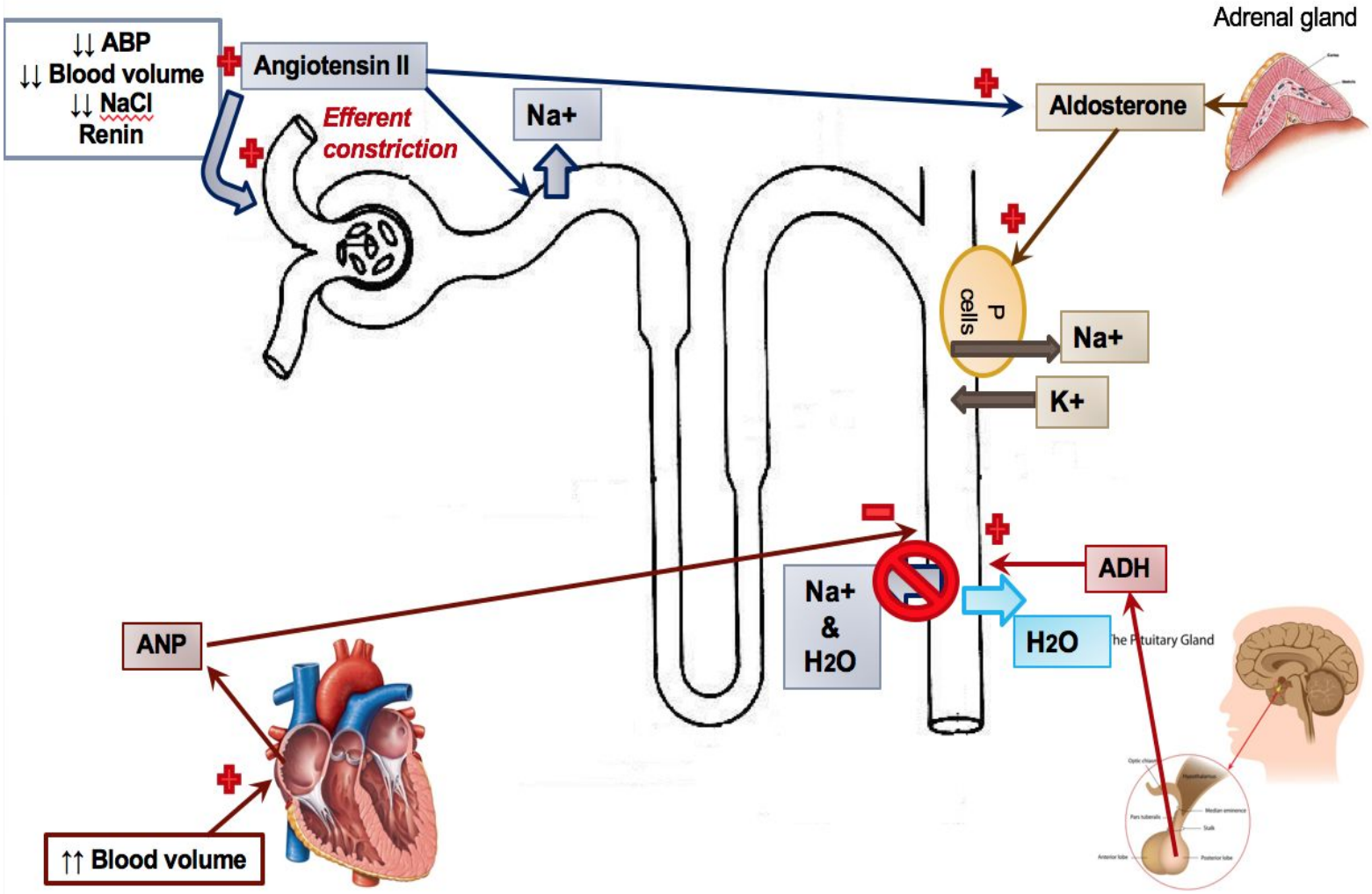
Natriuresis = excretion of sodium in urine
Diuresis = excretion of water in urine

ONLY in female slides

2- Hormonal Regulation of Tubular Reabsorption

Hormones are:

- Renin
- Angiotensin ii
- Aldosterone
- ANP
- ADH



Organic Anion/Cation Secretion

Here just memorize them and know that they secreted to PCT

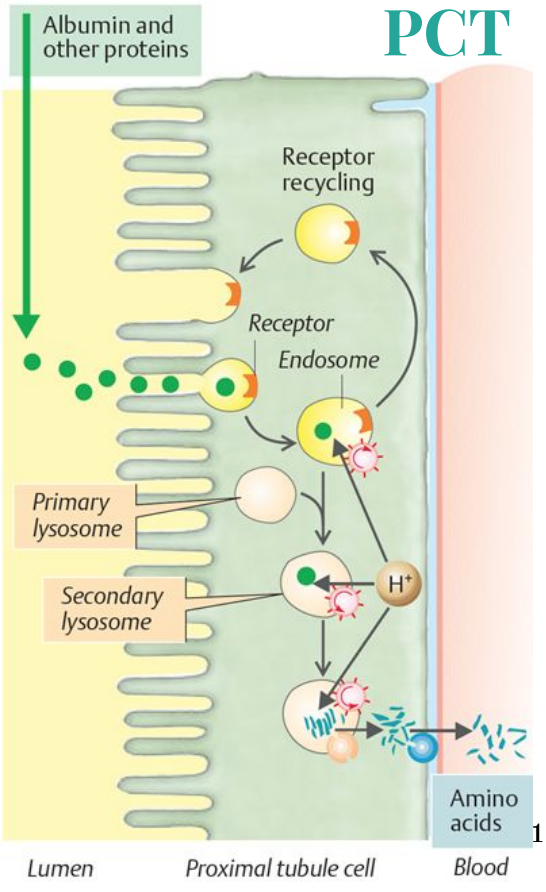
Organic Anions	Organic cations
1-Endogenous: • Bile salts. • Oxalate. • Urate. • Vitamins (ascorbate, folate). **2-Exogenous:** • Acetazolamide. • Furosemide. • Salicylates. • Penicillin.	**1-Endogenous:** • Creatinine. • Dopamine. • Epinephrine. • Norepinephrine. **2-Exogenous:** • Atropine. • Morphine. • Amiloride. • Procainamide

Many of these organic compounds can be bound to plasma proteins and are not readily filtered. Therefore, excretion by filtration alone eliminates only a small portion of these potentially toxic substances from the body. Such substances are also secreted from the peritubular capillaries into the tubular fluid. These secretory mechanisms are very powerful and remove virtually all organic anions and cations from the plasma entering the kidneys. Hence, these substances are removed from the plasma by both filtration and secretion.

ONLY in male slides

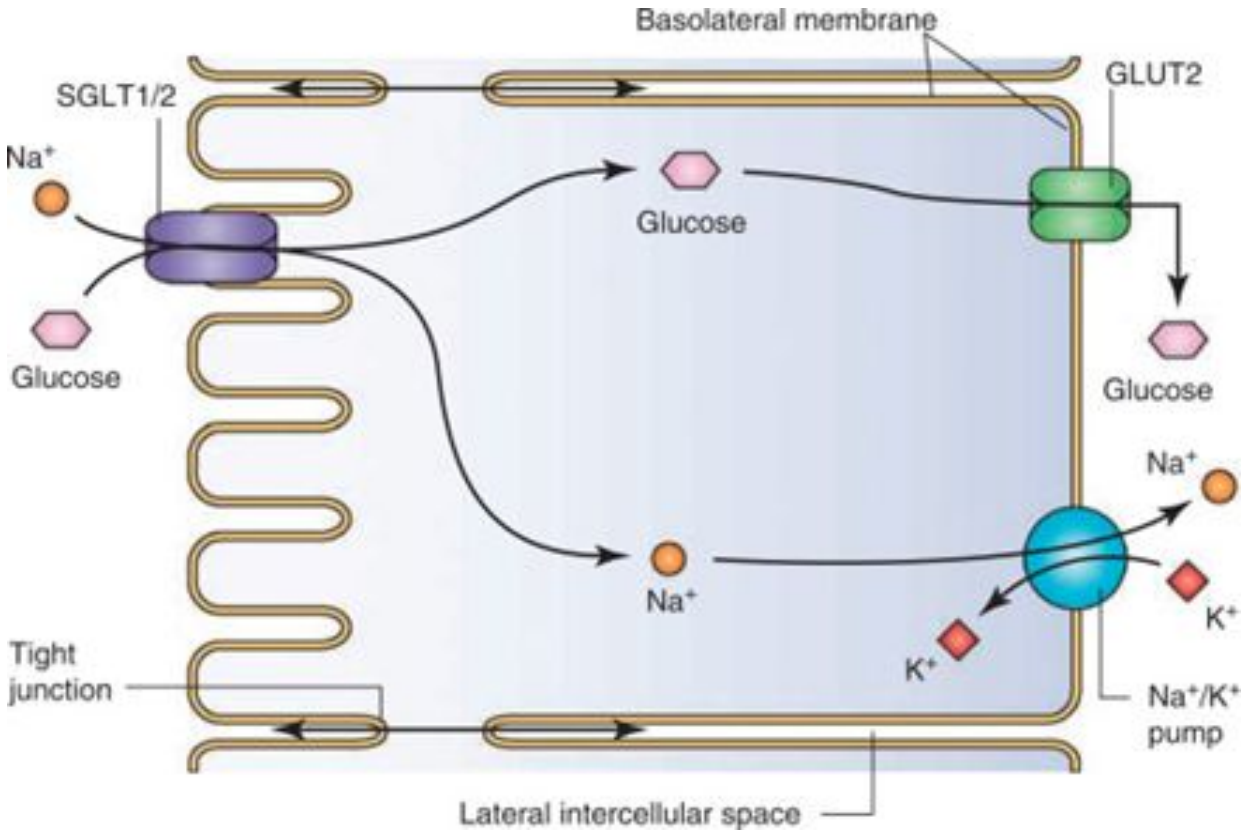
Protein Reabsorption

- Peptide hormones, small proteins & amino acids
- **Endocytosis** either intact or after being partially degraded by enzymes.
- Has a maximum capacity - too much protein filtered = **proteinuria**

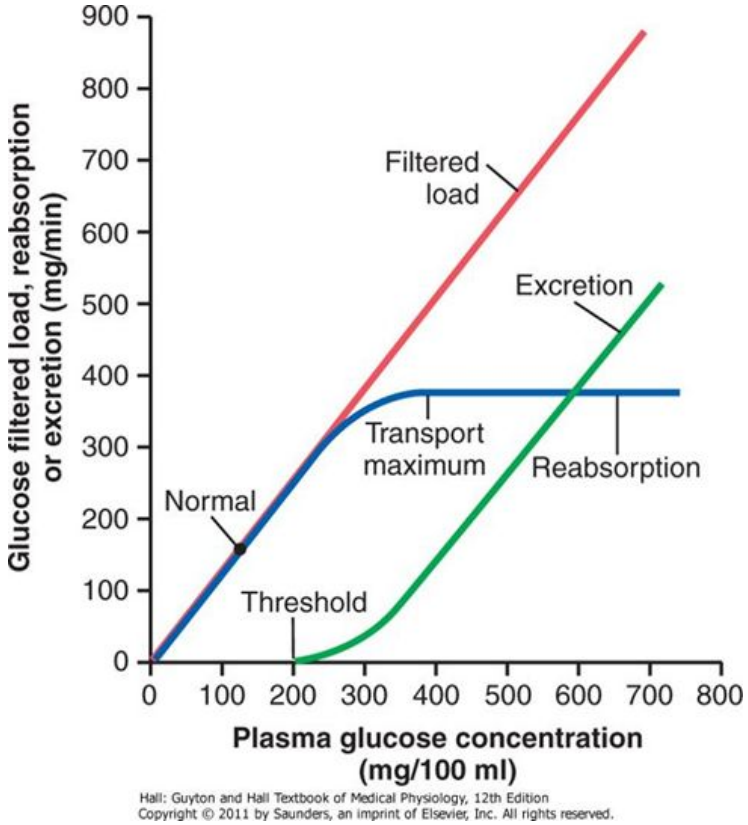


Glucose Reabsorption

Mentioned Previously in Lecture 3



Transport Maximum for Glucose



What is meant by transport maximum?

It is the maximal amount of a substance (in mg) which can be transported (reabsorbed or secreted) by tubular cells/min.

Why does it occur?

Due to saturation of the carriers. If Tm is exceeded, then the excess substrate enters the urine.

What happens if blood glucose level increased to 400 mg/dl?

If plasma [glucose] = 400 mg/dl, 400 mg/dl will be filtered, 180 mg/dl reabsorbed and 220 mg/dl excreted.

Sodium Reabsorption

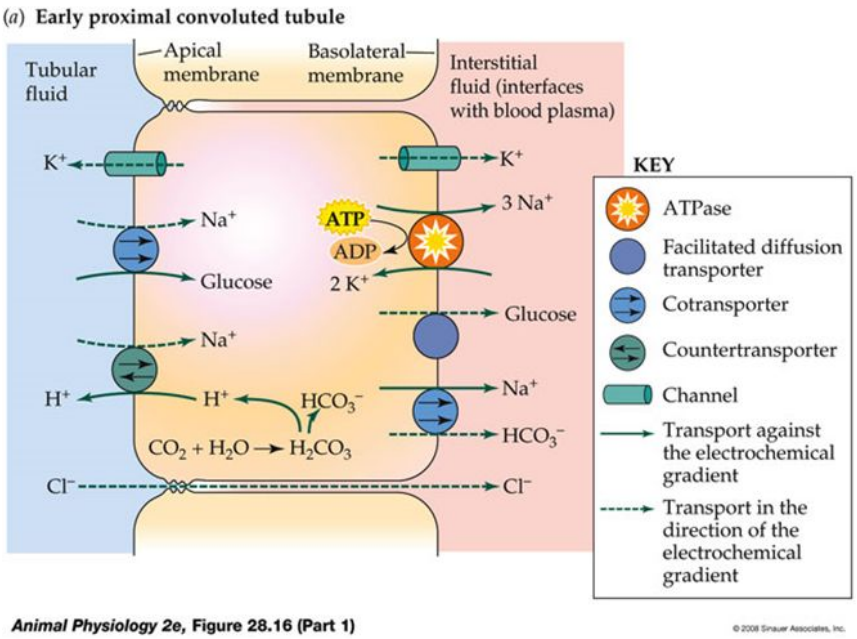
Basolateral Na^+-K^+ATPase pumps $3Na^+$ out and $2K^+$ into the cell

↓

Results in low [Na^+]inside the cell

↓

This gradient favours Na^+ entry across the apical membrane via transporter proteins.



Factors Affecting Na Reabsorption

ONLY in male slides

GFR	Aldosterone	Estrogens	Osmotic diuresis
Natriuretic hormone	Diuretic Drugs (Laxis)	Poorly reabsorbed anions	

- When GFR increased causes an increase in filtration of Na which sensitise the macula densa.
- Poorly reabsorbed anions causes retention of equal amount of Na.
- Estrogens cause Increase reabsorption of Na and decrease Na excretion.
- Osmotic diuresis such as Glucose, Mannitol and Urea when their conc. Increase in the filtered load then causes a decrease in water reabsorption and Na.

ONLY in male slides

HCO_3^- Reabsorption

- The renal tubules are poorly-permeable to HCO_3^-. However, it is still reabsorbed but in the form of CO_2 (to which the tubules are very highly permeable).

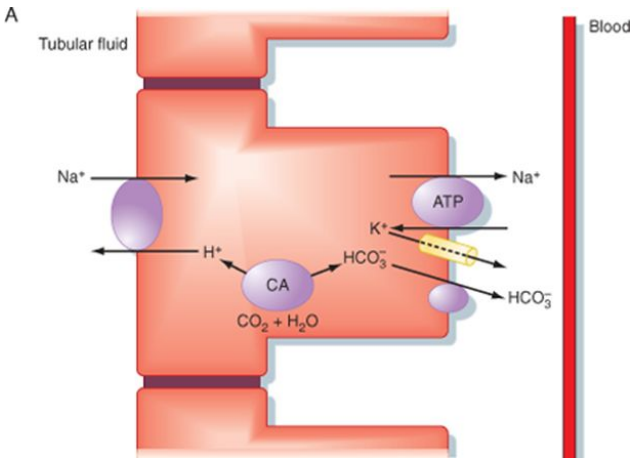
This occurs through the following steps:

1. H^+ is formed inside the cells then secreted in the tubular fluid.
2. H^+ combines with HCO_3^- in the tubular fluid forming H_2CO_3.
3. By activity of the **carbonic anhydrase enzyme (C.A.)** in the tubular cells, H_2CO_3 dissociates into CO_2 & H_2O.
4. CO_2 diffuses into the cells where it combines with H_2O (by activity of an intracellular C.A.), forming H_2CO_3 which dissociates into HCO_3^- & H^+.

HCO_3^- passively diffuses into the interstitial fluid (then to the blood) while H^+ is secreted into the tubular fluid to help more reabsorption of HCO_3^-.

- Factors affecting **HCO_3^-** reabsorption:
  - Arterial Pco_2
  - Plasma$[K^+]$*
  - Plasma Aldosterone.*
  - Plasma $[Cl^-]$**

*They will affect H level.
**Chloride depletion enhances Bicarbonate reabsorption by inhibition of Chloride/Bicarbonate exchanger.

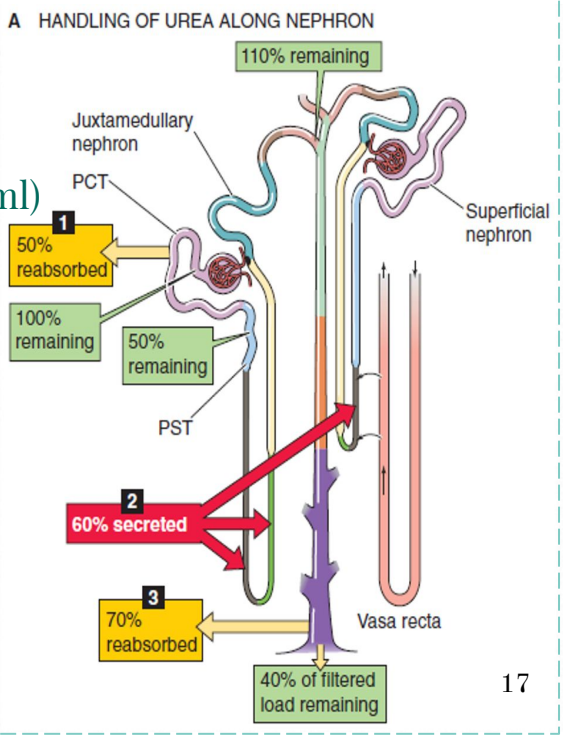


Urea Reabsorption

- Normal plasma level of urea 2.5-6.5 mM/L (15-39 mg/100ml)

Mechanism of urea reabsorption:

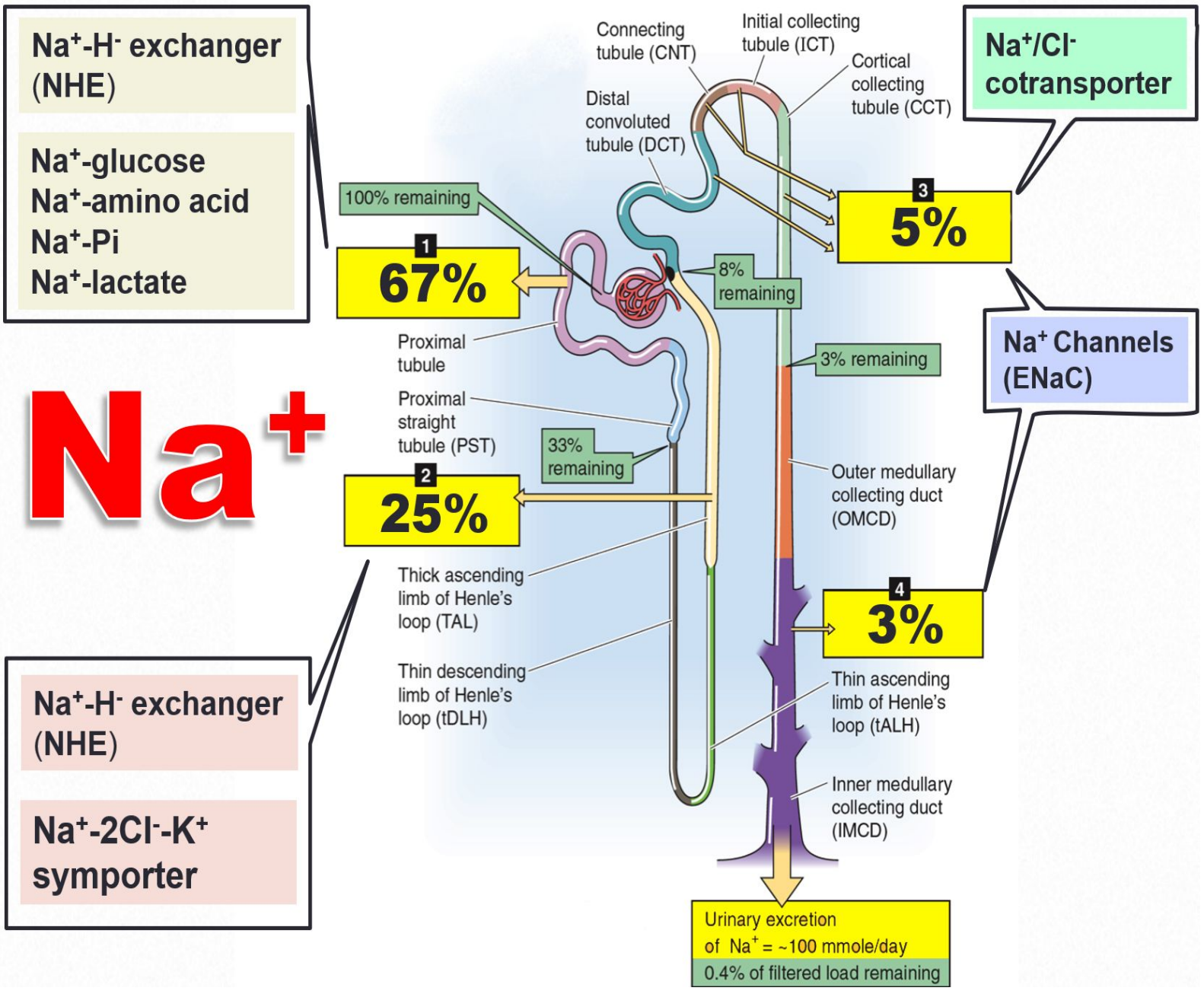
- About **40-70%** of filtered load of urea is reabsorbed in:
  - Second half of PCT.
  - Medullary CT and CD (ADH dependent)
- Due to water reabsorption in the first half of PCT, the conc. of urea is increased in the second half and urea is reabsorbed by simple diffusion (downhill)



Transport Process in the Nephron

ONLY in male slides

Know the transport mechanisms at each point and the percentage of Na Reabsorption at each point.

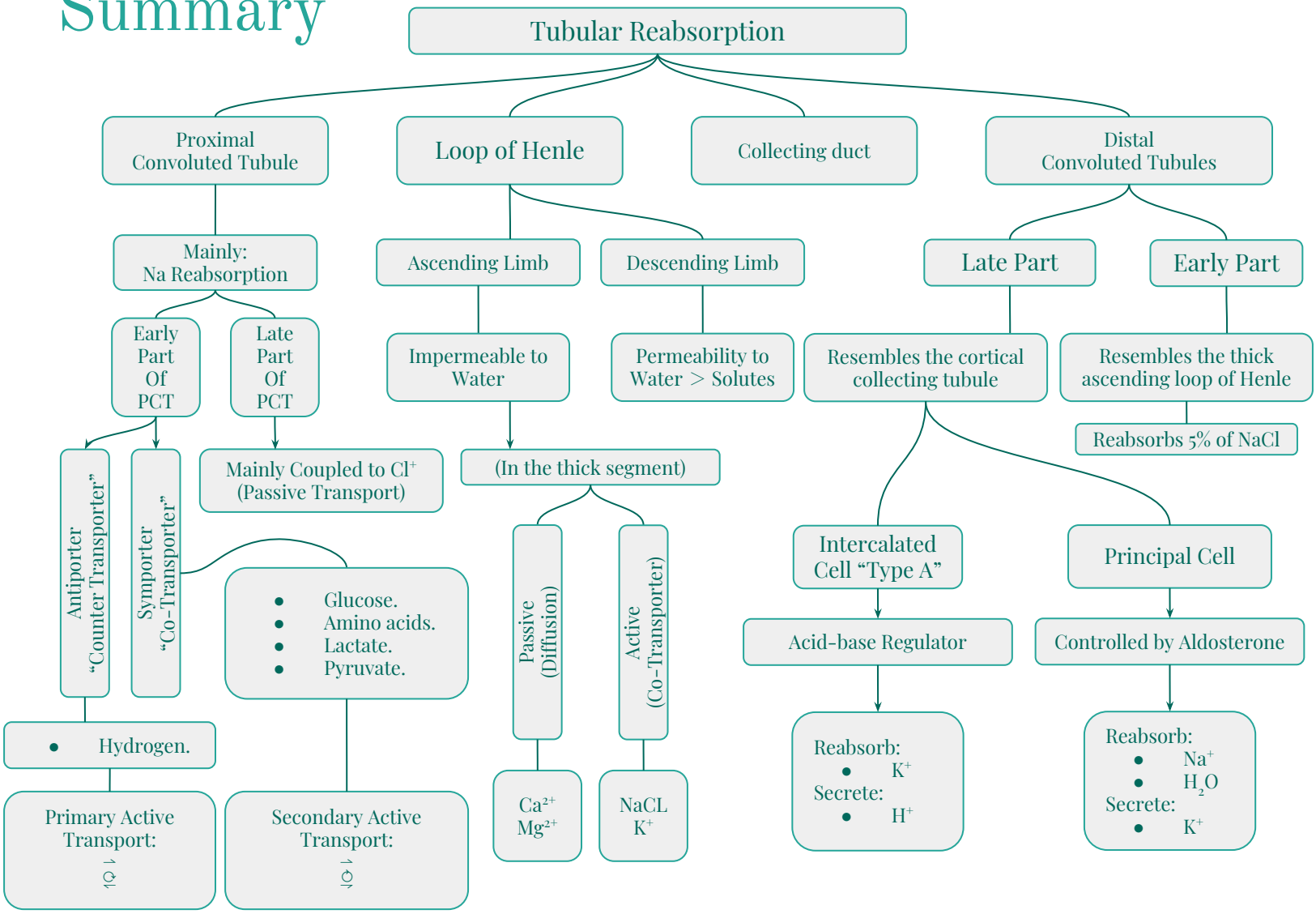


Tubular Secretion

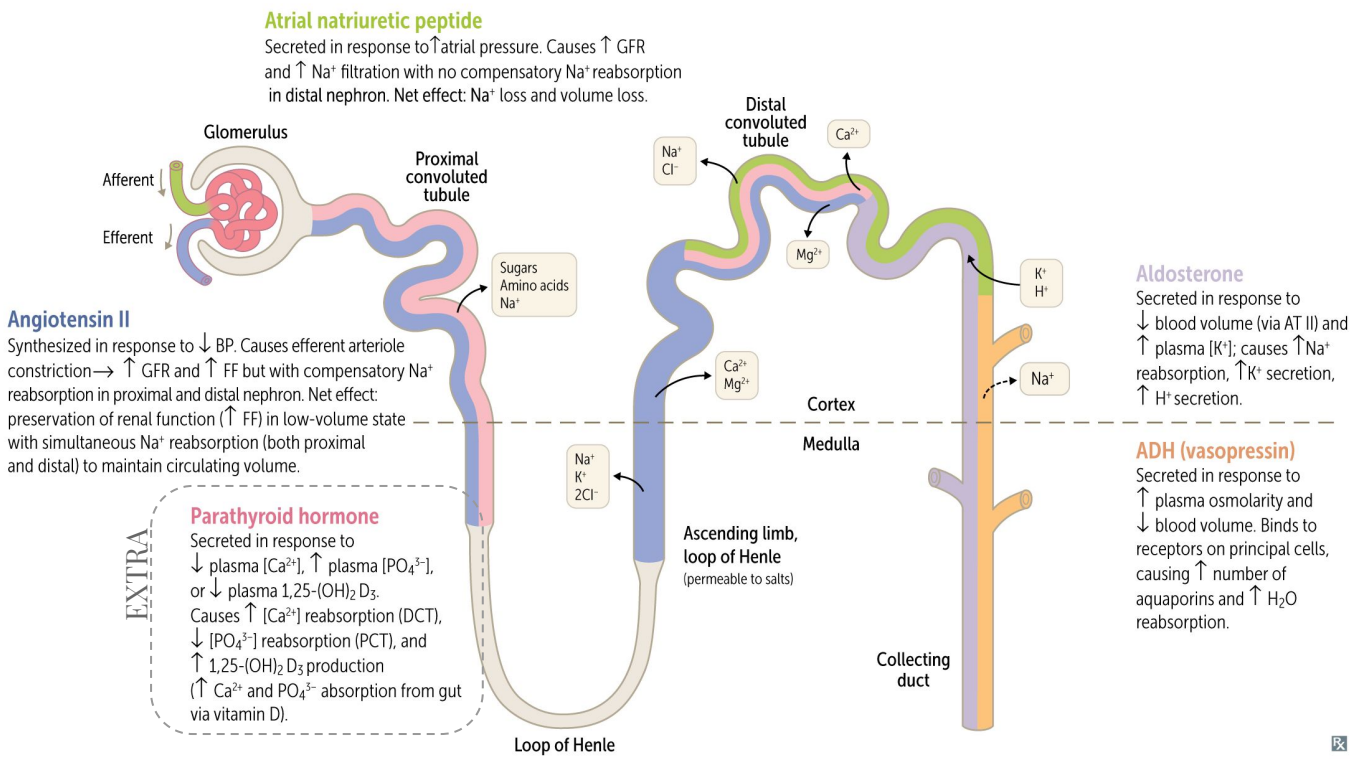
Summary of reabsorption and secretion in PCT & DCT, Duration 4:21 mins (1)

Review of reabsorption and secretion (armando), Duration 13 mins

Summary



Hormones acting on kidney

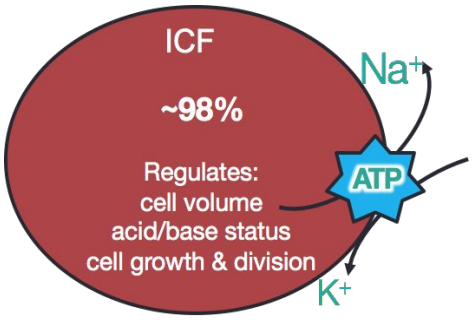


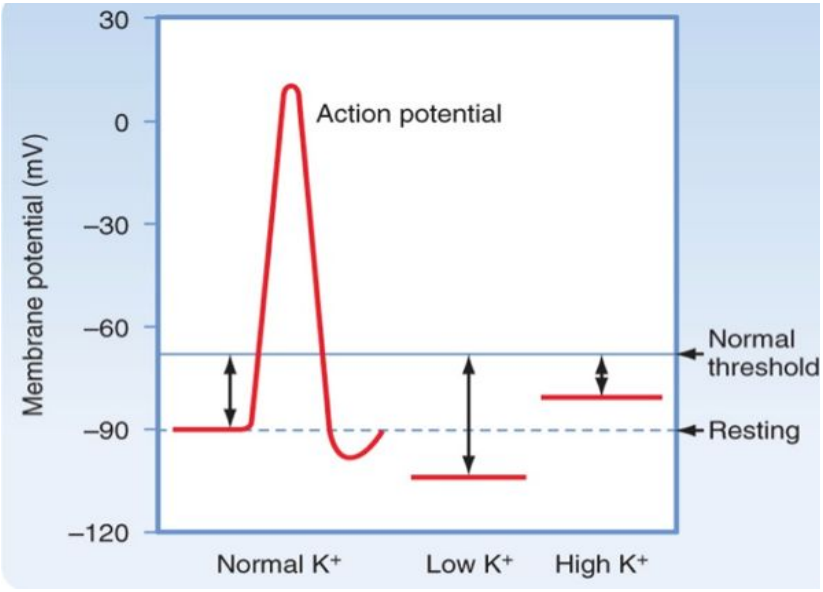
ONLY in male slides

K^+ is the most abundant cation in the body

- K^+ concentrations in equilibrium → Equal diffusion into and out of cell
- **Decrease** EC K^+ → **increase** diffusion of K^+ out of cell → cells hyperpolarized
- **Increase** EC K^+ → **decrease** diffusion of K^+ out of cell → cells partially depolarized

- K is the major determinant of the resting membrane potential and plays a crucial role in the normal functioning of all cells, especially those with inherent excitability.
- K 98% intracellularly , 2% extracellularly.
- This very high concentration difference is maintained by Na-K-ATPase enzyme that actively pumps potassium into the cell while moving sodium out of the cell.





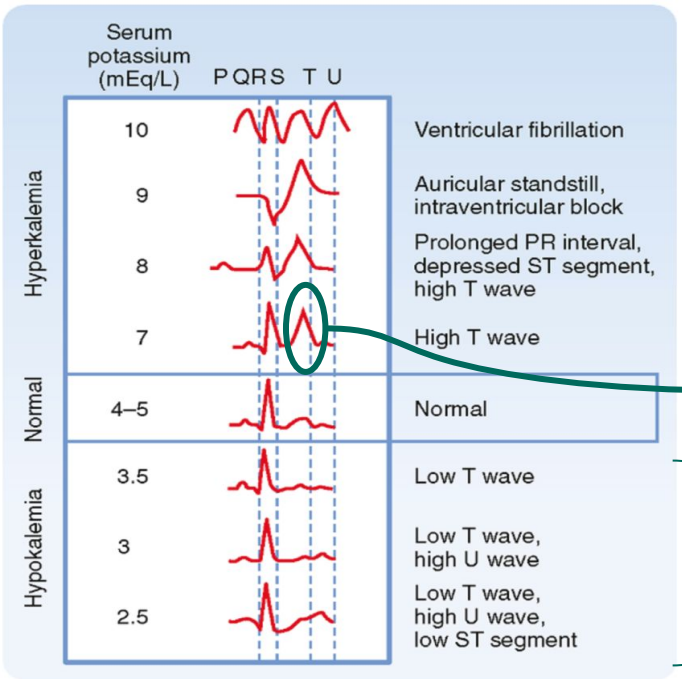
precise control mechanisms

Plasma [K^+] **3.5-4.8 mmol/L**

Regulates:
membrane potentials in excitable cells

ECG Changes due to Serum K Changes

Changes in plasma K^+ affect the ECG wave form



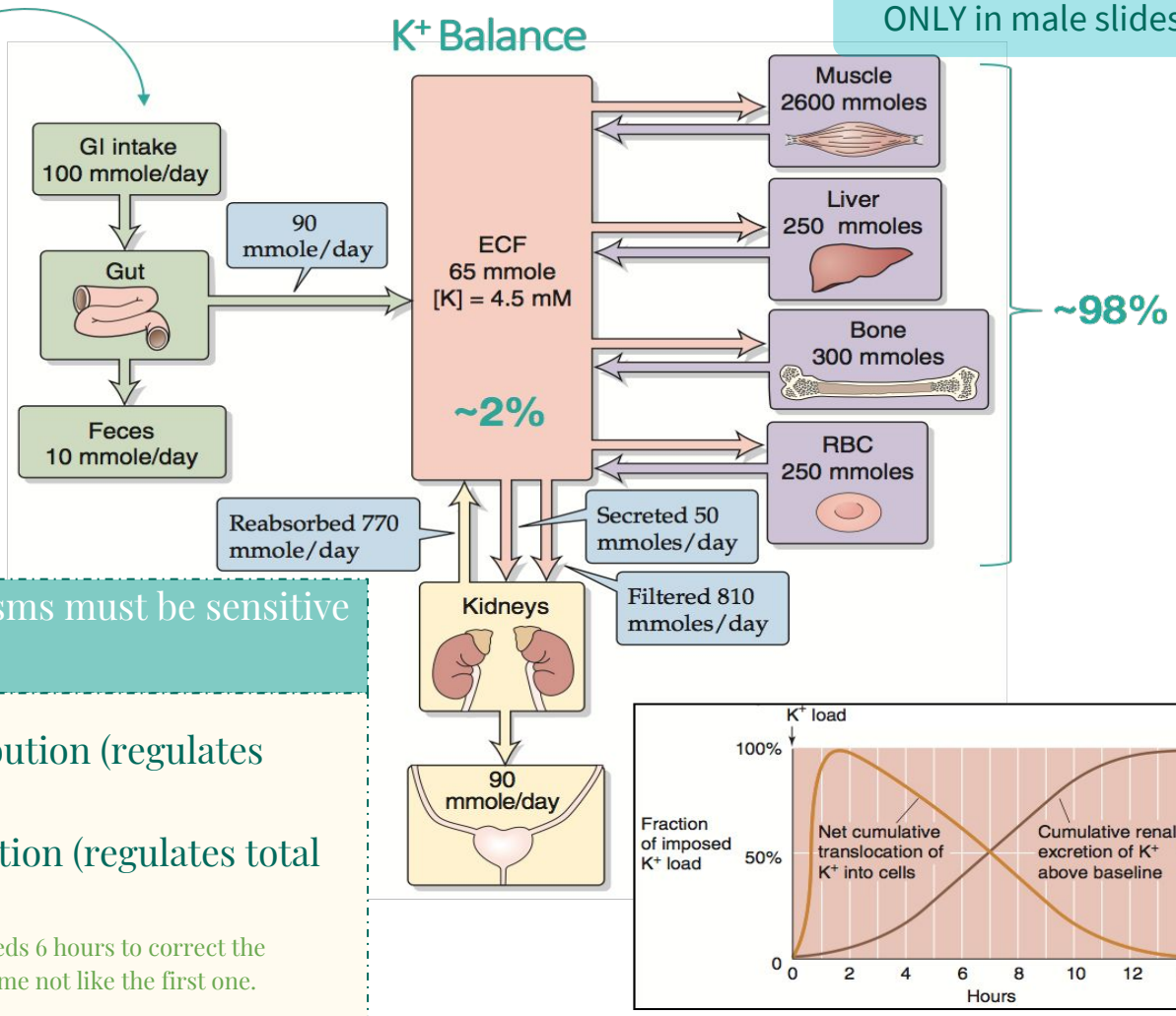
P wave disappears, QRS broadens, **(sine wave)**

Prolong PR, depress ST, and lengthen QRS

The first sign of hyperkalemia is a tall, thin T waves

Hypokalemia prolongs the QT interval, inverts the T wave, and lowers the ST segment

ONLY in male slides



Control mechanisms must be sensitive and rapid:

1- Internal distribution (regulates extracellular [K^+])

2- Renal K^+ excretion (regulates total body K^+)

Renal K^+ excretion may Needs 6 hours to correct the situation, so it takes long time not like the first one.

Control Mechanisms of Potassium K

Late

Renal Excretion

Rapid

Internal Regulation

The internal regulation mechanism function by distributing excess amount of K to the cells until the renal excretion mechanism begins to function.

Factors that shift K out of the cell

1- Acidosis: ICF K^+ is "exchanged" for extracellular H^+.

2-Increased **Osmolality** → K^+ moves out secondary to H_2O movement out of cells

3- Exercise → loss of K^+ from muscles

4- Cell lysis → release of cellular contents

Factors that shift K into the cell

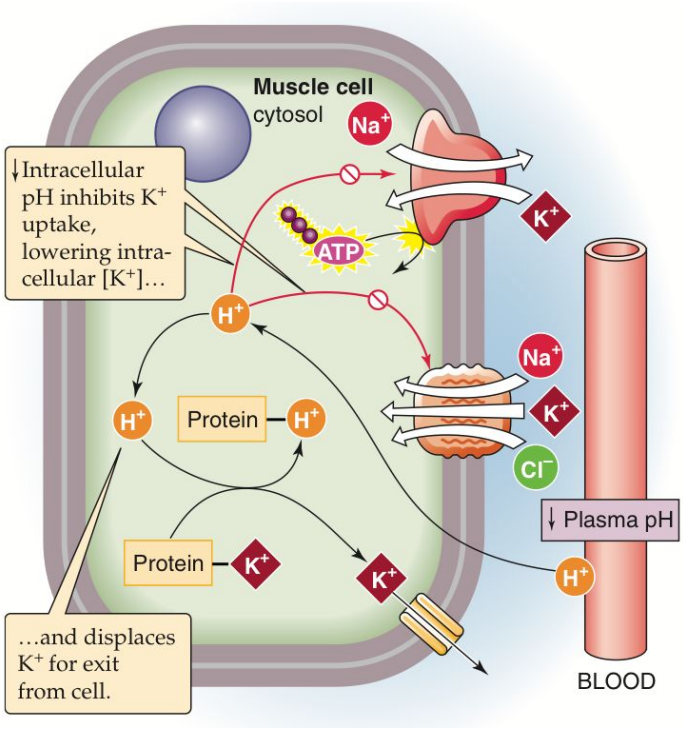
1- Insulin: after high K^+ meal Insulin + glucose conc. Increase to treat hyperkalemia.

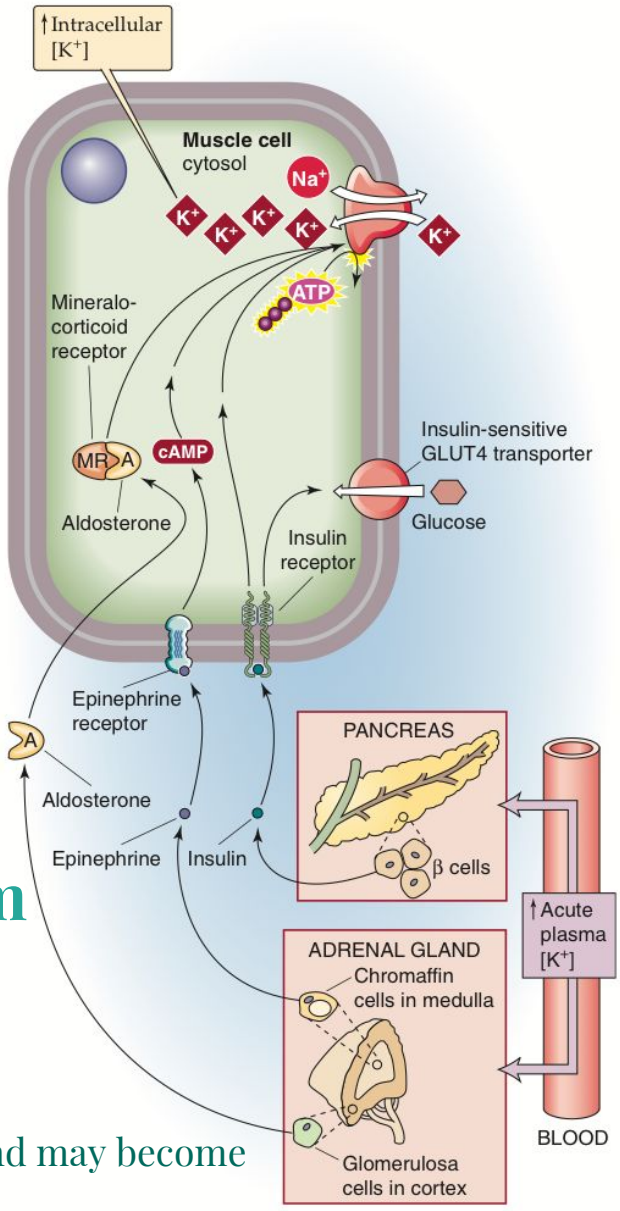
2- Catecholamines (Adrenaline): Via stimulation of b_2 b blockers increase plasma K^+ after a meal or an exercise

3- Aldosterone

4- Alkalosis: H^+ is *exchanged* for extracellular K^+.

ONLY in male slides





Renal Excretion of Potassium

- 90-95% of Dietary K excreted via the kidneys
- 5-10% in Sweat & Feces (This is unregulated and may become significant in diarrheas)
- In normal individual intake is matched by excretion and potassium balance is maintained.
- Filtered load of potassium ~ 720 mmol/day
- Bulk absorbed by proximal tubule and loop of Henle.

Renal K^+ Transport Mechanisms

ONLY in male slides

- Cell membrane transporters:

Na^-K ATPase, H^-K ATPase.

K^+ channels, K:Cl cotransport.

Na:K:2Cl cotransport.

- K^+ is Reabsorbed in PT, TAL and intercalated cell in CCD
- K^+ Secreted in late distal tubule and in principal cells of late DT and CCD

Plasma $[K^+]$

Hyperkalemia stimulates secretion of K^+ within minutes

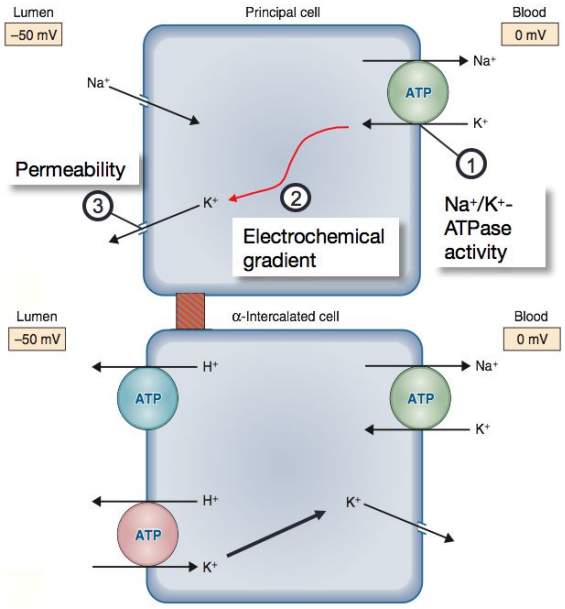
How?

1. Stimulates Na/K-ATPase → ↑ K^+ uptake (*basolateral*) → ↑ electrochemical gradient.

2. ↑ permeability to K^+ (*apical*).

3. ↑ aldosterone → ↑ secretion of K^+.

<u>Hypokalaemia produces an opposite effect</u>.



Aldosterone

↑ K^+ secretion by:

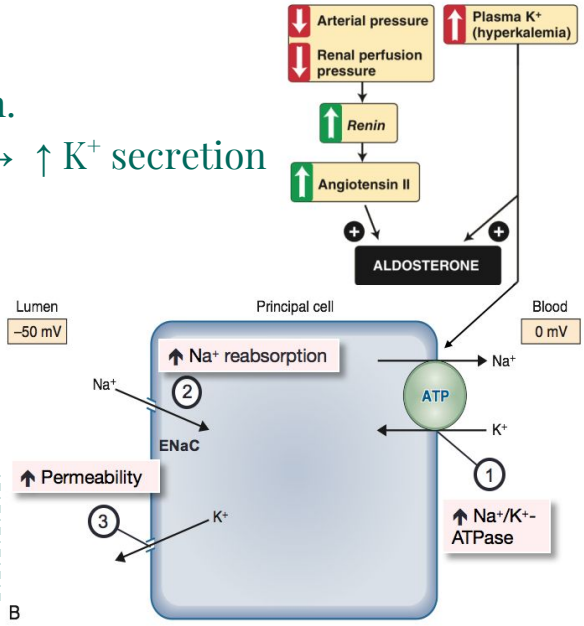
1. ↑ Na/K ATPase → ↑ Na^+ reabsorption → ↑ K^+ secretion.

2. ↑ Na+ reabsorption* (↑ ENaC) → -ve lumen potential → ↑ K^+ secretion

3. ↑ permeability of apical membrane → ↑ K^+ secretion

Conn's syndrome (↑ aldo) → hypokalaemia

Addison's disease (↓ aldo) → hyperkalaemia



*المقصود هنا أن قناة الصوديوم هذي راح تدخل الصوديوم للخلية وبالتالي راح تقل الشحنة الموجبة في الخارج وهذا يساعد في إخراج البوتاسيوم

B

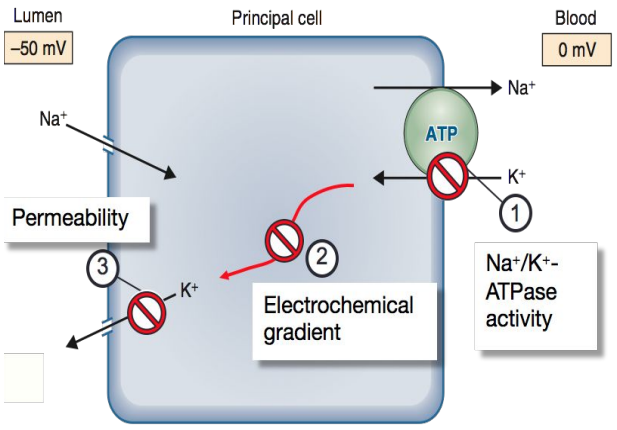
Acid-base Balance

ONLY in male slides

Acidosis inhibits K^+ secretion in principal cells by INHIBITING:

- Na/K ATPase → ↓ K^+ uptake from blood → ↓ conc. gradient for K^+ efflux into the lumen.
- K^+ channels (*apical*) → ↓ K^+ secretion directly → hyperkalemia.

Alkalosis has the opposite effect, promoting K^+ secretion and hypokalemia.



Effects of acidosis on K^+ secretion

Factors Affecting Potassium Secretion

Briefly: Aldosterone, Insulin, Epinephrine, ECF ph and Luminal flow (Diuresis)

In details:

ONLY in male slides

Luminal factors:

1- Diuresis: increase volume of urine and decrease conc of K in lumen which causes secretion via chemical gradient. (increase secretion and excretion)

2- Increased urinary excretion of Na: increase in Na-K exchange at luminal membrane causes an increase in secretion and excretion of K.

3- Increased urinary excretion of bicarbonate, phosphate, sulphate and ketone acids: increase negativeness of lumen then increase electrochemical gradient between cell and lumen causes secretion and excretion of K.

2- when the sodium excretion in PCT increase, more Na arrive to DCT and that will stimulate Na/K ATPase, so more K secretion.

Peritubular factors:

1- Hyperkalemia: increase K in tubular cells, increase chemical gradient of K between tubular cell and tubular lumen which lead to increase in the secretion and excretion of K.

2- Hyper-aldosteronism: increase aldosterone will increase secretion and excretion of K.

3- Alkalosis: increase H-K exchange at baso-lateral membrane then increase secretion and excretion of K.

3-in case of alkalosis, more H is needed so the exchanger in the basolateral membrane will allow more H to blood and more K to tubular cell.

Regulation of Potassium

Body Potassium Balance

Input:

- Dietary intake = 80-120 mmol/day.
- The ECF K^+ content ≈ 70 mmol.

Output:

- GI loss ≈ 5-10%
- Renal excretion ≈ 90-95%.

Control External balance Between body and environment

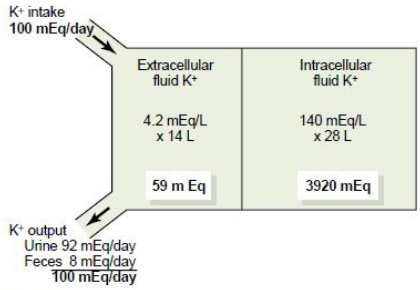


Figure 29–1

Normal potassium intake, distribution of potassium in the body fluids, and potassium output from the body.

Internal balance (within the body) is regulated by modifying the distribution of K^+ between the ICF & ECF.

The Importance of Regulating K+

Table 37-1 Physiological Role of K^+ Ions

A. Roles of Intracellular K^+	
Cell-volume maintenance	Net loss of K^+ → cell shrinkage Net gain of K^+ → cell swelling
Intracellular pH regulation	Net loss of K^+ → cell acidosis Net gain of K^+ → cell alkalosis
Cell enzyme functions	K^+ dependence of enzymes (e.g., some ATPases, succinic dehydrogenase)
DNA/protein synthesis, growth	Lack of K^+ → reduction of protein synthesis, stunted growth
B. Roles of Transmembrane $[K^+]$ Ratio	
Resting cell membrane potential	Reduced $[K^+]_i/[K^+]_o$ → membrane depolarization Increased $[K^+]_i/[K^+]_o$ → membrane hyperpolarization
Neuromuscular activity	Low plasma K^+: muscle weakness, muscle paralysis, intestinal distention, respiratory failure High plasma K^+: increased muscle excitability; later, muscle weakness (paralysis)
Cardiac activity	Low plasma K^+: slowed conduction of pacemaker activity, arrhythmias High plasma K^+: conduction disturbances, ventricular arrhythmias, and ventricular fibrillation

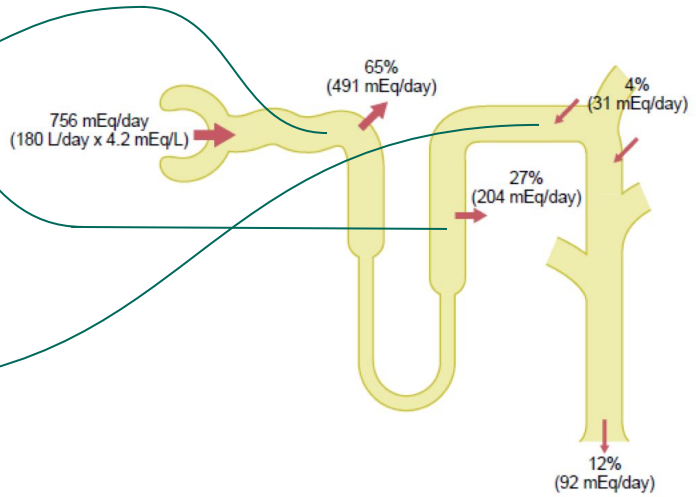
Secretion of K^+, Duration 6 mins

Reabsorption of K^+, Duration 4 mins

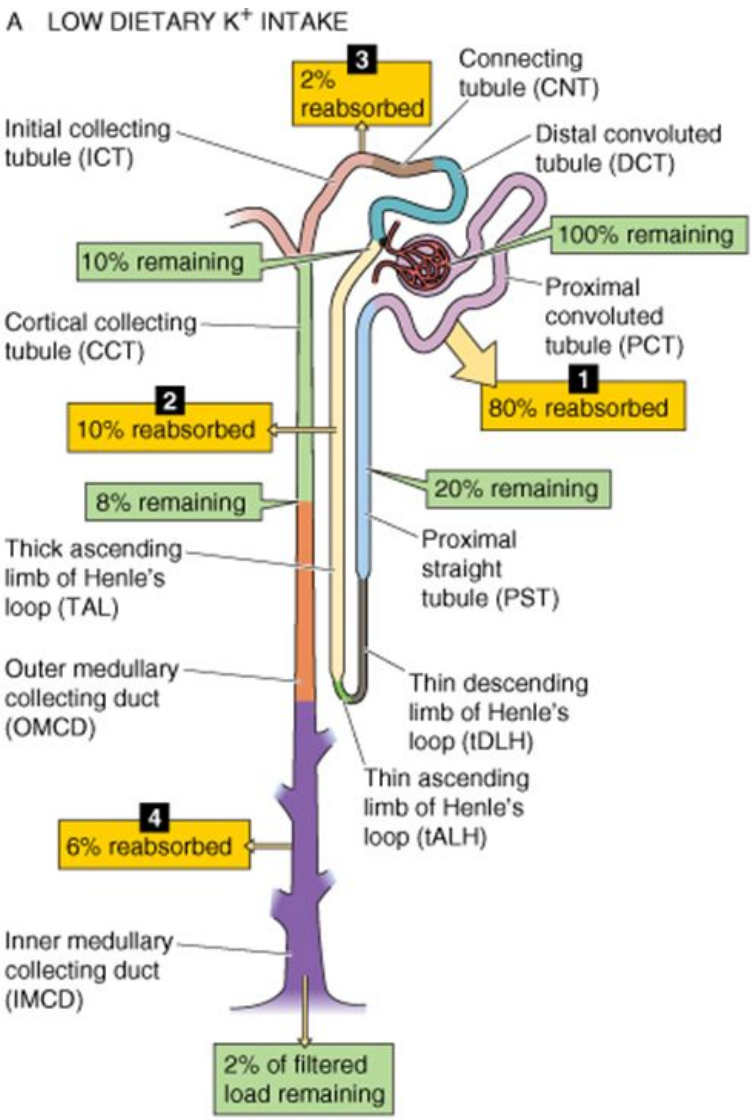
Renal Potassium Handling

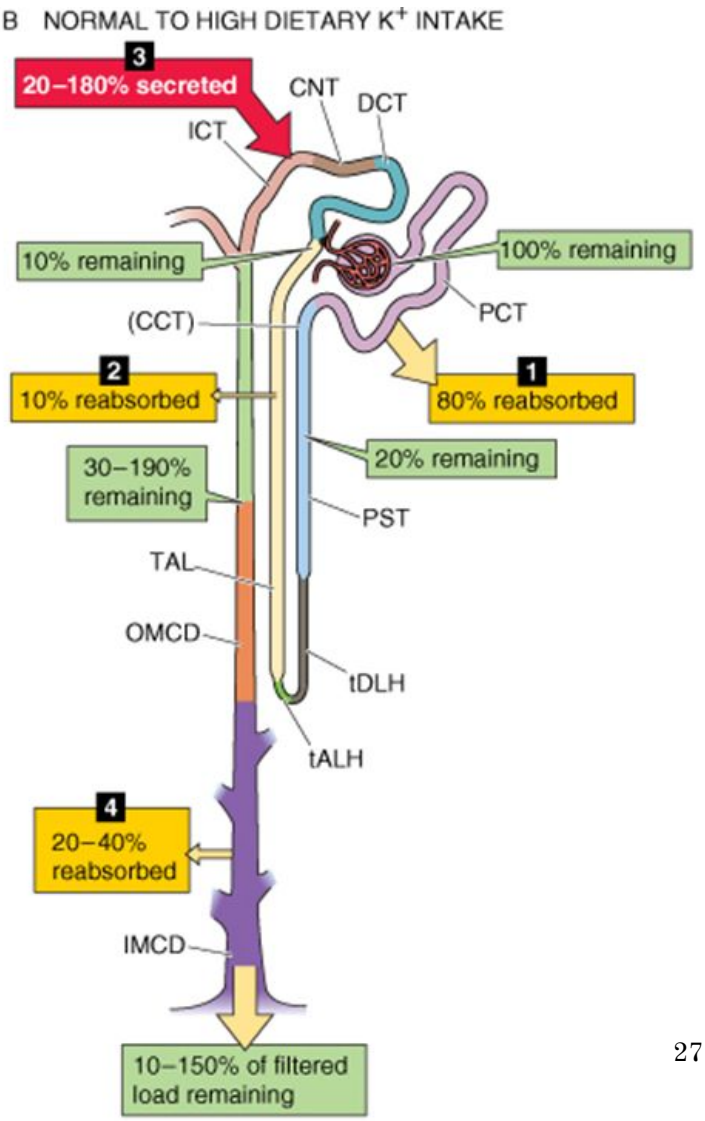
- ≈ 90% of K+ is reabsorbed in the PCT & TAL.

- ≈ 10% enters the distal portions of the nephron.
  - **If K+ intake is low** only 1-3% of filtered K+ will be excreted.
  - **If K+ intake is normal/high**, 10-15% of filtered K+ will be excreted.



ONLY in male slides

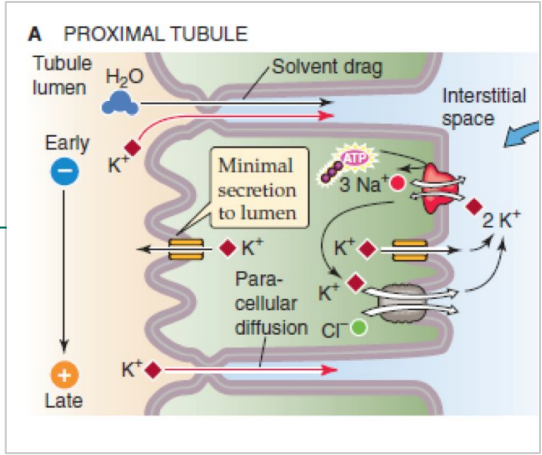




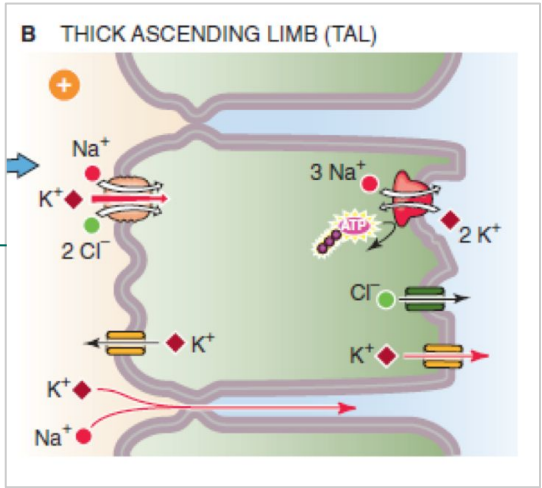
Potassium Handling by the kidney

It is the sum of = filtration – reabsorption + secretion

- **In the PCT →** K+ reabsorption is a passive process. **How?**
- Water reabsorption (driven by Na+ absorption) through the paracellular route drags K+ with it **(solvent drag).**



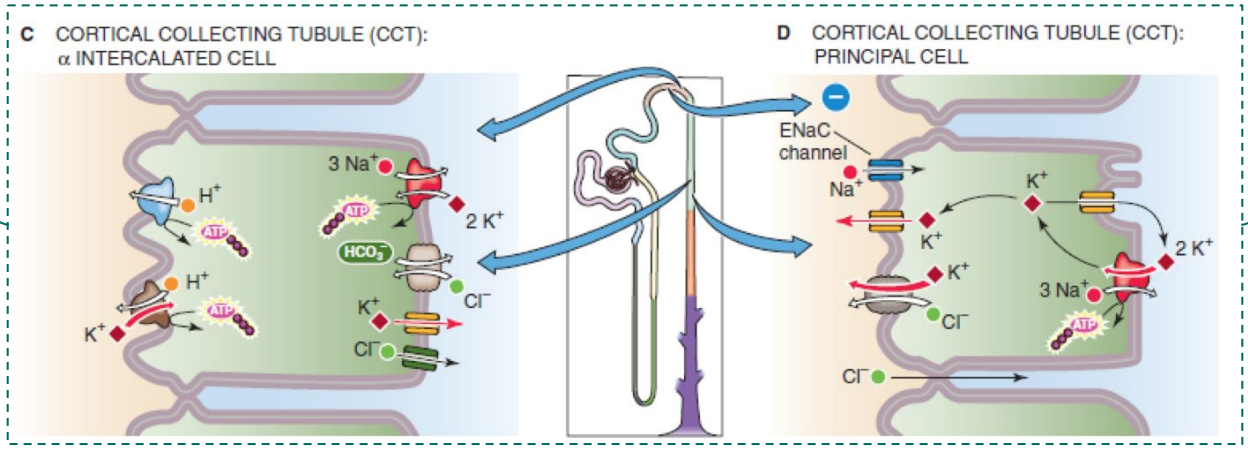
- In the TAL → By secondary active transport using the apical triple transporter (Na:K:2Cl).
- K:Cl co-transport in baso-lateral membrane.



Potassium Handling by the CT

Alpha-Intercalated cells

Principal cells



Secrete H+ and reabsorb K+ via a luminal H-K ATPase

Reabsorb Na+ and water & secrete K+ via luminal K channels and basolateral Na-K ATPase

Quiz

1- Which of the following is true about glucose reabsorption from the tubular lumen to the tubular cell?
A. It is done by carrier-mediated passive transport.
B. Direction of transport is downhill.
C. No energy is consumed in the process.
D. It is done by secondary active transport of sodium

2- Which of the following is false about sodium reabsorption?
A. Sodium is reabsorbed in the early proximal convoluted tubule.
B. Sodium enters the tubule at the luminal membrane
C. Sodium is passively transported out using the Na-K ATPase pump.
D. All of the above are correct

3- Which change tends to increase peritubular capillary fluid reabsorption?
A. Increased blood pressure
B. Increased efferent arteriolar resistance
C. Decreased angiotensin II
D. Increased renal blood flow

4- The primary renal site for the secretion of organic ions e.g urate, creatinine is :
A. proximal tubule
B. loop of Henle
C. distal tubule
D. collecting duct

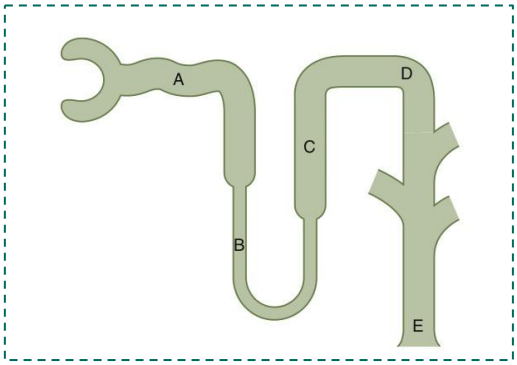
5- Principal cells are responsible for reabsorption of?
A. Ca
B. Phosphorus
C. Hydrogen
D. Sodium

1- D 2- C 3- B 4- A 5- D

6- Which of the following tubules never reabsorbs water?
A. Proximal convoluted tubule
B. Descending loop of Henle
C. Ascending limb
D. Collecting tubules

7- Where is Sodium-potassium specific pumps?
A. Basement membrane
B. Basolateral membrane
C. Interstitial wall
D. Cytoplasmic membrane

For Questions 8-10 choose the appropriate nephron site in the above figure.67.



8- In a person on a very low potassium diet, which part of the nephron would be expected to reabsorb the most potassium?
A. A
B. B
C. C
D. D

9- Which part of the nephron normally reabsorbs the most water?
A. A
B. B
C. C
D. D

10- In a normally functioning kidney, which part of the tubule has the lowest permeability to water?
A. A
B. B
C. C
D. D

6- C 7- B 8- A 9- A 10- C

Thank you for checking our work

Male Team:

أنس السويداء
أنس السيف
خالد شويل
ريان الموسى
سعد الهداب
سلطان الناصر
سعود العطوي
سيف المشاري
عبدالجبار اليماني
عبدالرحمن آل دحيم
هشام الموسى

فهد الفايز
خالد المطلق
نواف الهلال
هشام الشايع
خالد العقيلي
عبدالله الزيد
حسين علامي
سلطان الفهيد
خالد المطيري
فهد النهابي
عمر اليابس

Female Team:

آلاء الصويّغ
رناد المقرن
روان مشعل
ريم القرني
نوره بن حسن
مجد البرّاك

مها النهدي
ريناد الغريبي
عائشة الصباغ
ميعاد النفيعي
مها القحطاني

Team Leaders:

عبدالمجيد الوردي ~ ساره البليهد

@physio437

physiologyteam437@gmail.com