Urinary System: Renal Physiology for Medical Students, L4-8

Urine Formation by the Kidneys: II. Tubular Reabsorption and Secretion

Reference: Guyton & Hall, Jordanian first edition Chapter27

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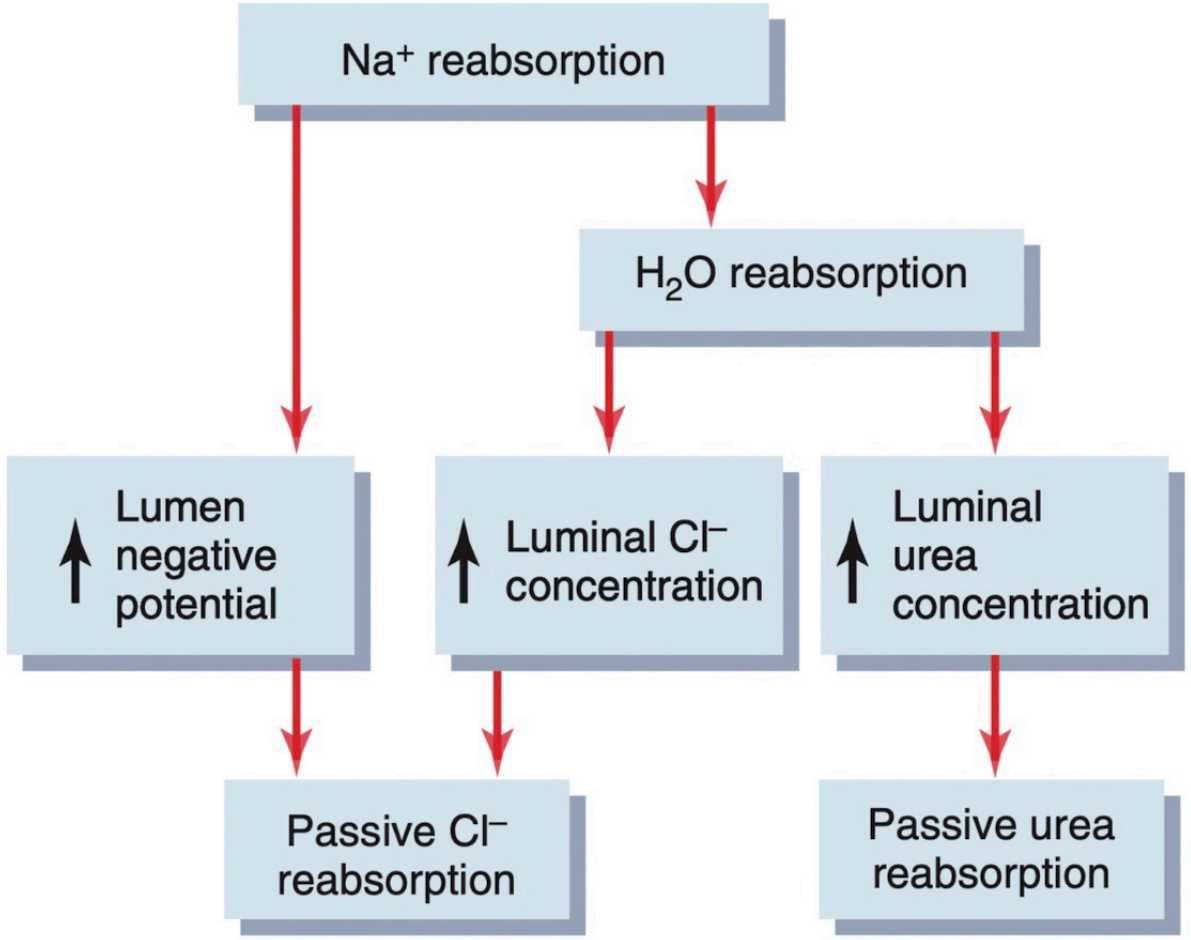
Modified by Toqa Abushanab

Reference : GUYTON & HALL 13th edition, Doctor's notes and 020 sheet.

Mechanisms by which water, chloride, and urea reabsorption are coupled with sodium reabsorption.



Proximal Tubules Have a High Capacity for Active and Passive Reabsorption.

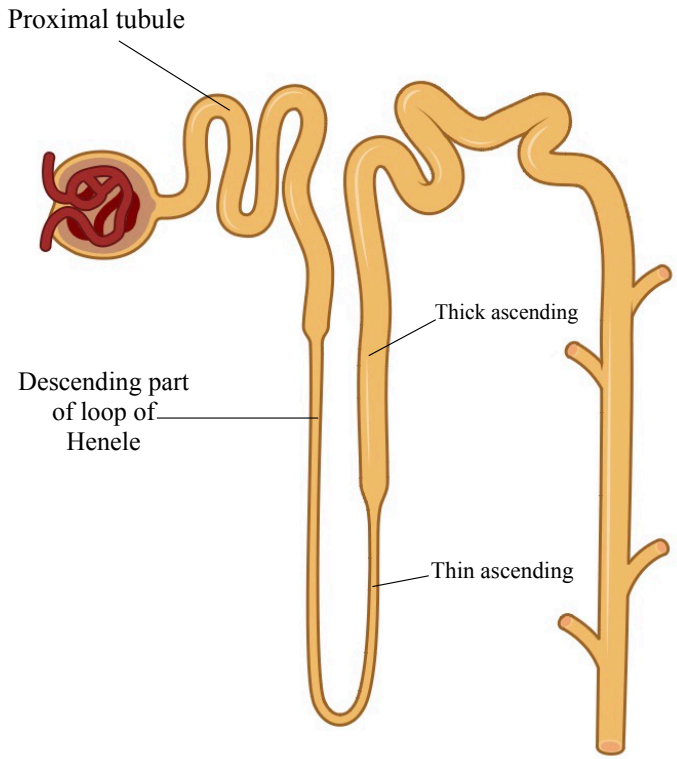
When Na^+ is reabsorbed along with water in the proximal tubule, the luminal concentration of Cl^- increases. This rise in Cl^- concentration drives its passive reabsorption through two main mechanisms: Electrochemical Potential or Concentration Gradient as mentioned in the previous lecture.

Later in the proximal tubule, the Na^+- Cl^- co-transporter protein facilitates the active reabsorption of Na^+ and Cl^- ions together. This co-transporter utilizes the energy gradient established by the Na^+/K^+ ATPase pump to drive the reabsorption of Na^+, with Cl^- on this transport (by mean of secondary active transport).
The reason the Na^+-Cl^- co-transporter operates after Cl^- accumulates in the lumen is primarily due to its location in the later segment of the proximal tubule. There are special characteristics in the reabsorption processes occurring in the early and later segments of the proximal tubule:

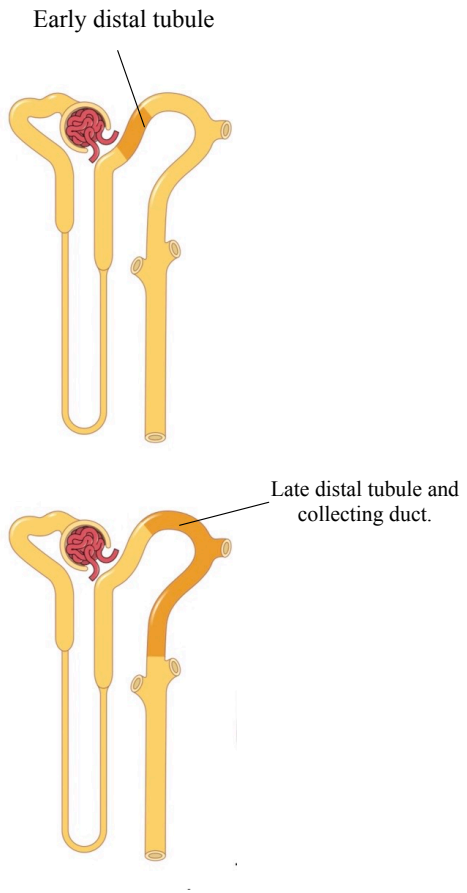
- **Early Segments**: In the early segments, Na^+ reabsorption is often coupled with the reabsorption of glucose and amino acids. This co-transport mechanism utilizes the concentration gradient of these solutes to drive Na^+ reabsorption.
- **Later Segments**: In the later segments, Na^+ reabsorption is primarily coupled with Cl^- reabsorption through the Na^+-Cl^- co-transporter. This shift occurs because the concentration of glucose and amino acids in the tubular fluid has significantly decreased by the time it reaches the later segments.

As Na^+ reabsorption progresses in the early segments, the luminal concentration of Cl^- increases due to its lower rate of reabsorption compared to Na^+. This buildup of Cl^- then drives its passive reabsorption through the mechanisms mentioned earlier (paracellular diffusion), as well as Na^+-Cl^- Co-transporter.

The doctor wanted to clarify the points mentioned above. However, let's remember that we have already discussed the different segments of the nephron, including the proximal tubule, as well as the various parts of the loop of Henle (descending, thin ascending, and thick ascending), in the previous lecture. Now, let's continue from where we left off.



Distal tubule



The thick segment of the ascending limb of the loop of Henle empties into the distal tubule. The distal tubule can be divided into two categories: **the early distal tubule** and the **late distal tubule with the collecting duct.** The late distal tubule shares similar characteristics and functions with the collecting duct, which is why they are grouped into the same category.

The first portion of the early distal tubule forms the macula densa, which is part of the juxtaglomerular complex and provides feedback control of GFR and blood flow in this same nephron.
The next part of the distal tubule is highly convoluted and has many of the same reabsorptive characteristics of the thick segment of the ascending limb of the loop of Henle. That is, it effectively reabsorbs most of the ions, including Na^+, K^+, and Cl^-, but is virtually impermeable to water and urea. For this reason, it is referred to as the **diluting segment** because it further dilutes the tubular fluid (Na^+ is reabsorbed without water being reabsorbed).

Indeed, we have also referred to the thick ascending limb of the loop of Henle as the diluting segment.

Early Distal Tubule

The Na^+-Cl^- co-transporter, located in the early distal tubule, facilitates the movement of NaCl from the tubular lumen into the cell, while the Na^+-K^+ ATPase pump transports sodium out of the cell across the basolateral membrane, providing the gradient for the co-transporter, Chloride diffuses out of the cell into the renal interstitial fluid through chloride channels present in the basolateral membrane.

Thiazide diuretics, which are commonly used to treat conditions like hypertension and heart failure, inhibit the sodium-chloride co-transporter. This inhibition results in an increase in tubular fluid volume, leading to greater urinary excretion. As a consequence, blood pressure decreases.

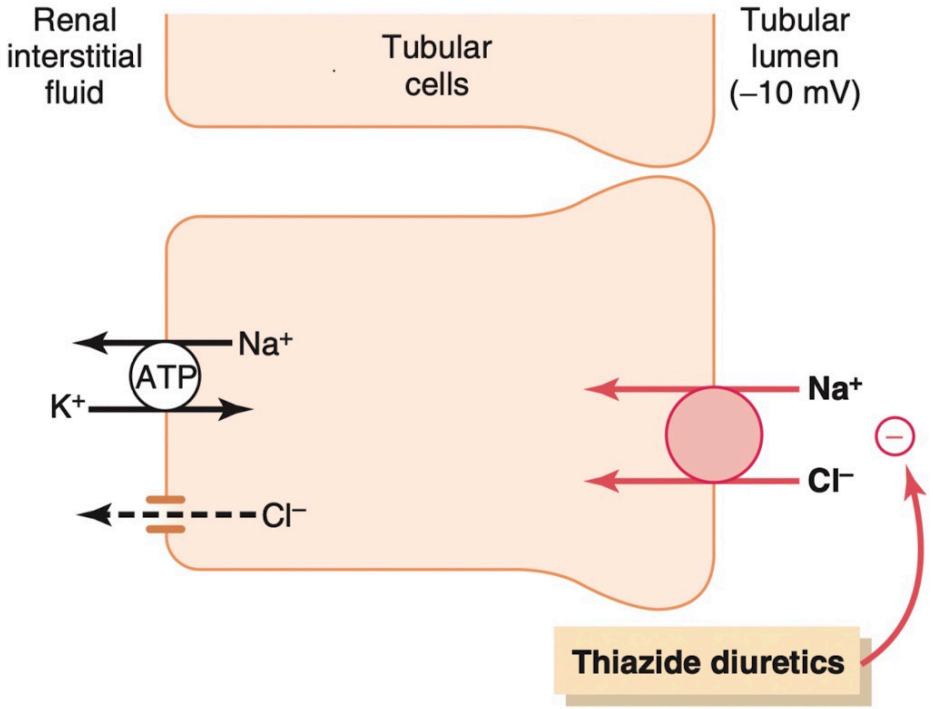


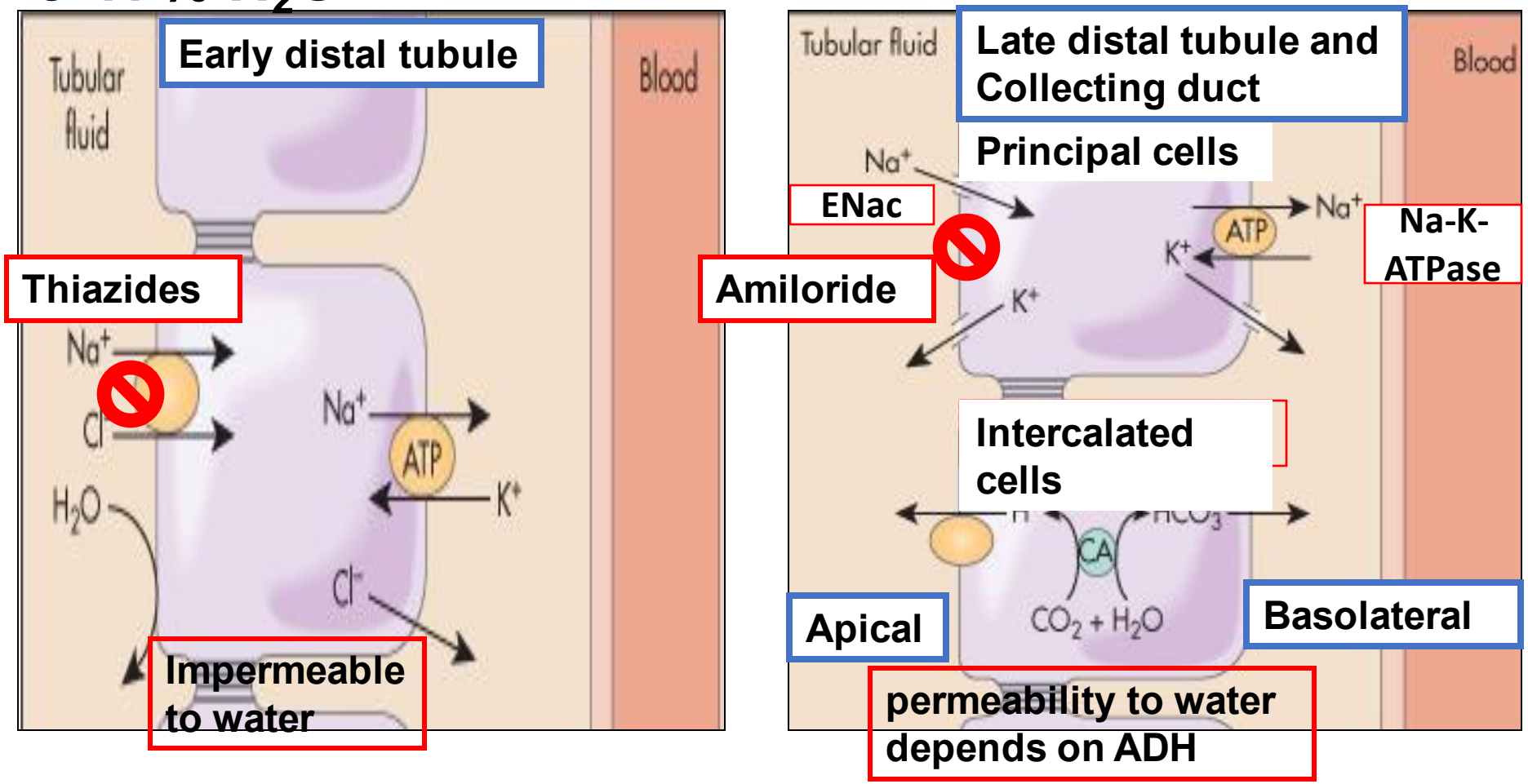
Figure 28-10. Mechanism of sodium chloride transport in the early distal tubule. Sodium and chloride are transported from the tubular lumen into the cell by a co-transporter that is inhibited by thiazide diuretics. Sodium is pumped out of the cell by sodium-potassium ATPase adenosine triphosphatase, and chloride diffuses into the interstitial fluid via chloride channels.

Early Distal Tubule

- Functionally similar to thick ascending loop
- Not permeable to water (called diluting segment)
- Active reabsorption of Na^{+}, Cl^{-}, K^{+}, Mg^{++}
- Contains macula densa

Distal tubule and collecting duct

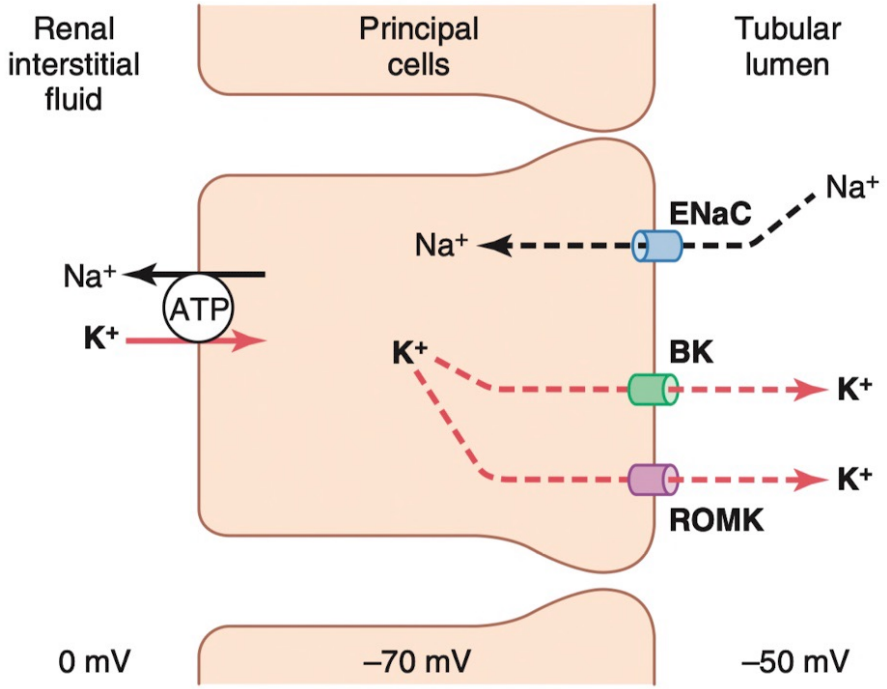
- **Reabsorbs 7% NaCl, secrets K+ and H+ and reabsorbs 8-17% H_2O**



Early and Late Distal Tubules and Collecting Tubules.

The second half of the distal tubule and the cortical collecting tubule have similar functions.
They consist of two types of cells: **principal cells** and **intercalated cells**.

- **Principal cells** → characterized by the presence of ENaC (Epithelial sodium channels). These channels play a key role in the reabsorption of Na^+ from the tubular lumen. The reabsorbed Na^+ ions are dependent on the action of the Na^+/K^+ ATPase pump, which creates a driving force for Na^+ from the luminal side. As a result, the intracellular concentration of K^+ increases. This buildup of intracellular K^+ ions is subsequently secreted into the tubular fluid or lumen. It's important to note that the direction of Na^+ and K^+ movement is opposite to each other: the more Na^+ is reabsorbed, the more K^+ is secreted.



Aldosterone hormone is responsible for increasing the activity of ENaC as well as the Na^+/K^+ ATPase channels. The adrenal cortex can by stimulated to secrete aldosterone by :

1. Angiotensin II → secreted in conditions such as hypotension, hypovolemia or hemorrhage.
2. Hyperkalemia.

Aldosterone plays a crucial role in K^+ regulation and maintaining hemostasis. Its role in K^+ regulation is considered more important than its role in Na^+ regulation. Therefore, aldosterone is considered the major regulator of K^+ in the body.

Amiloride is a synthetic inhibitor of ENaC. Na^+ accumulate in the lumen, K^+ secretion is impaired, the K stay in the blood. Therefore, it causes diuresis but conserve K those called Potassium sparing drugs. potassium ATPase pump. This, in turn, decreases transport of potassium into the cells and ultimately reduces potassium secretion into the tubular fluid. For this reason the sodium channel blockers, as well as the aldosterone antagonists, decrease urinary excretion of potassium and act as potassium-sparing diuretics.

Early and Late Distal Tubules and Collecting Tubules.

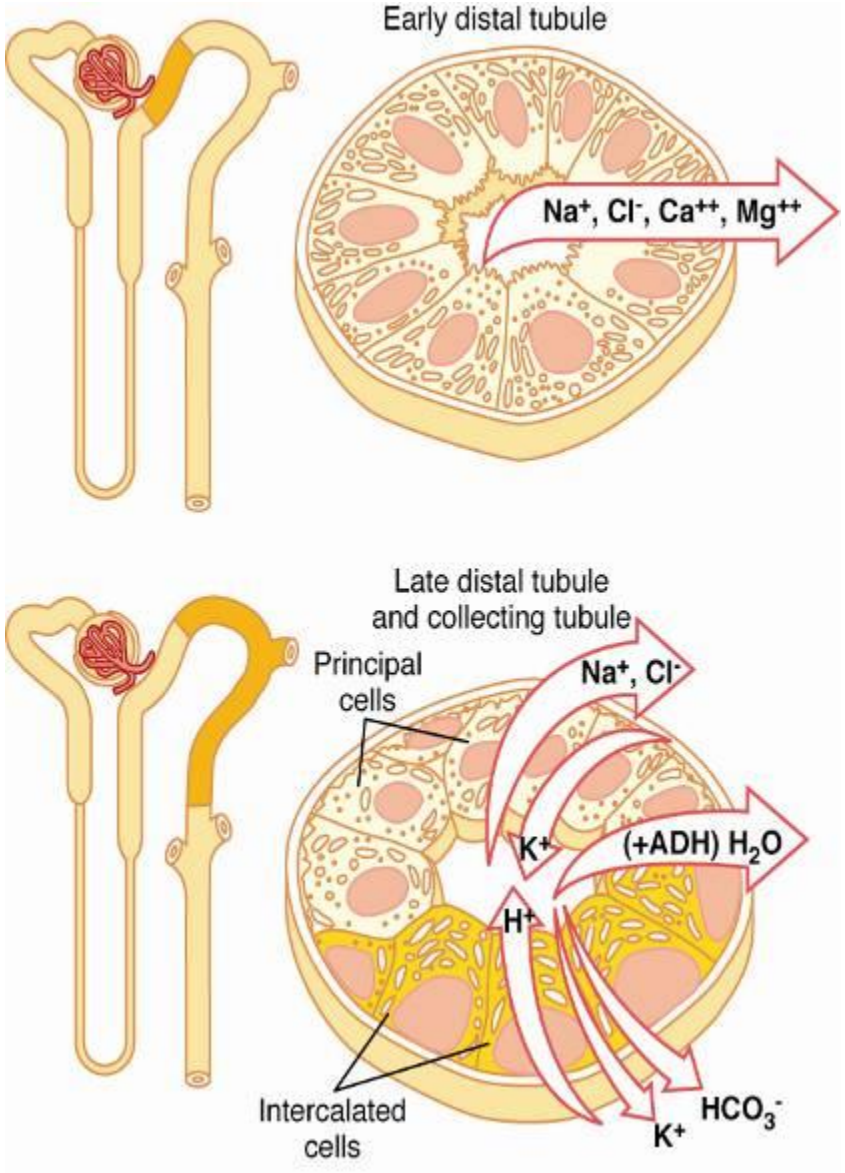


Figure 27-11

The permeability of the late distal tubule and cortical collecting duct to water is controlled by the concentration of ADH, which is also called vasopressin. With high levels of ADH, these tubular segments are permeable to water, but in the absence of ADH, they are virtually impermeable to water.

~ 5% of filtered load NaCl reabsorbed

- not permeable to H_2O
- not very permeable to urea

- permeablility to H_2O depends on ADH
- not very permeable to urea

Late Distal and Cortical Collecting Tubules Principal Cells – Secrete K^+

Aldosterone hormone is responsible for increasing the activity of ENaC as well as the Na^+/K^+ ATPase channels. The adrenal cortex can by stimulated to secrete aldosterone by :
1. Angiotensin II → secreted in conditions such as hypotension, hypovolemia or hemorrhage.
2. Hyperkalemia.

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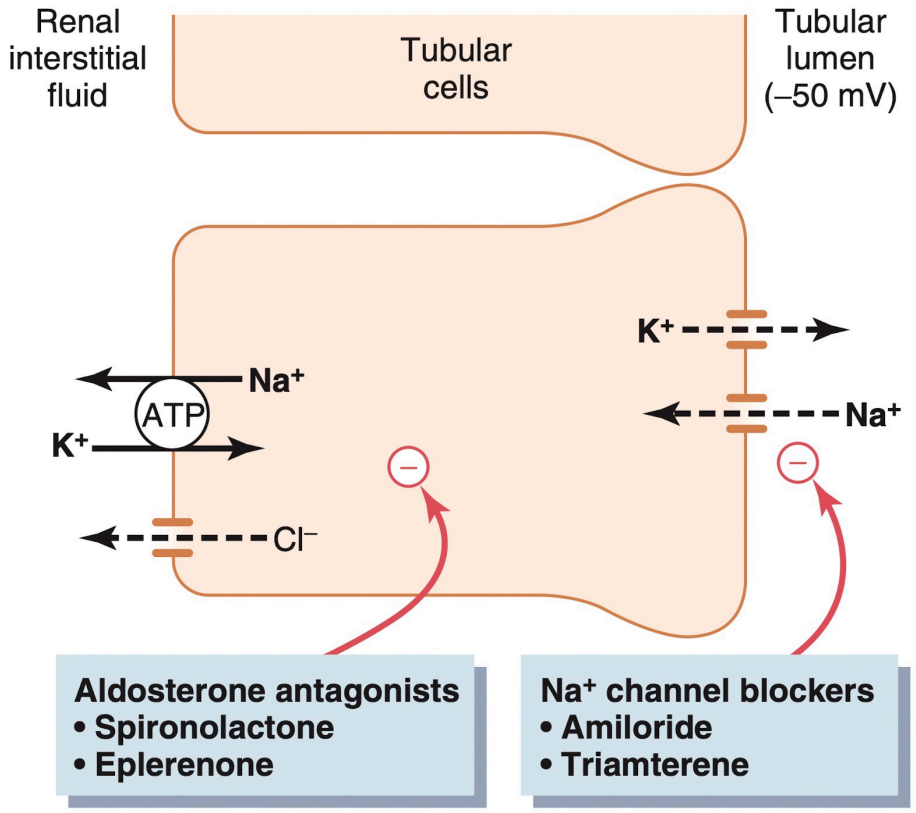
The principal cells are the primary sites of action of the potassium-sparing diuretics, including spironolactone, eplerenone, amiloride, and triamterene. Spironolactone and eplerenone are mineralocorticoid receptor antagonists **that compete** with aldosterone for receptor sites in the principal cells and therefore inhibit the stimulatory effects.

Amiloride and **triamterene** are sodium channel blockers (ENaC inhibitors). that directly inhibit the entry of sodium into the sodium channels of the luminal membranes and therefore reduce the amount of sodium that can be transported across the basolateral membranes by the sodium-potassium ATPase pump. This, in turn, decreases transport of potassium into the cells and ultimately reduces potassium secretion into the tubular fluid.

For this reason the sodium channel blockers, as well as the aldosterone antagonists, decrease urinary excretion of potassium and act as **potassium-sparing diuretics.**



Intercalated Cells Secrete or Reabsorb Hydrogen, Bicarbonate, and Potassium ions.

2. **Intercalated cells** → play a major role in acid-base balance, by secreting H^+ ions and reabsorption of HCO_3^- and vise versa. Depending on the type of the intercalated cell.
- Type A→ especially important in eliminating hydrogen ions while reabsorbing bicarbonate in acidosis.
- Type B → have functions opposite to those of type A cells and secrete bicarbonate into the tubular lumen while reabsorbing hydrogen ions in alkalosis.

Both intercalated cells and principal cells are affected by aldosterone. In the absence of ADH, they are impermeable to water. However, in the presence of ADH, they become permeable to water.

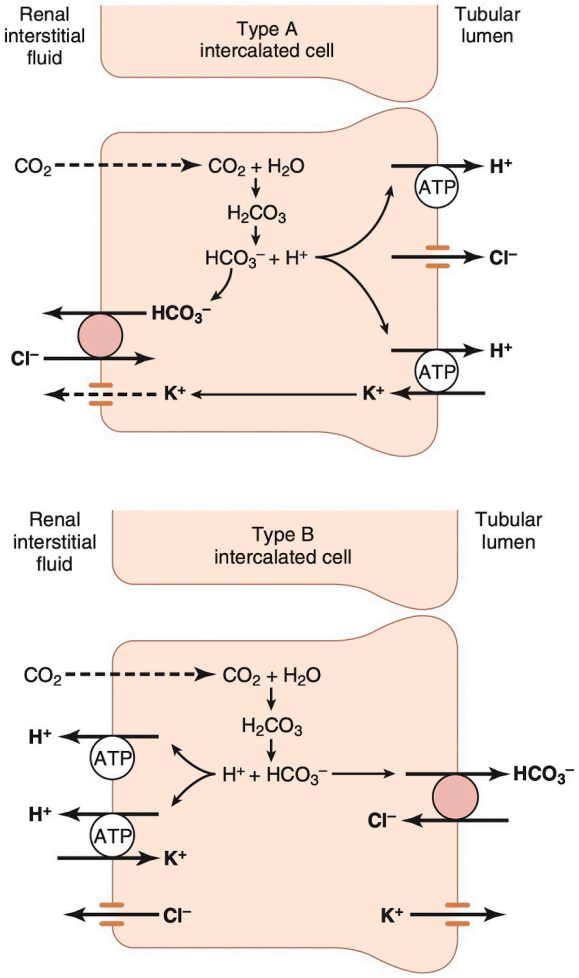
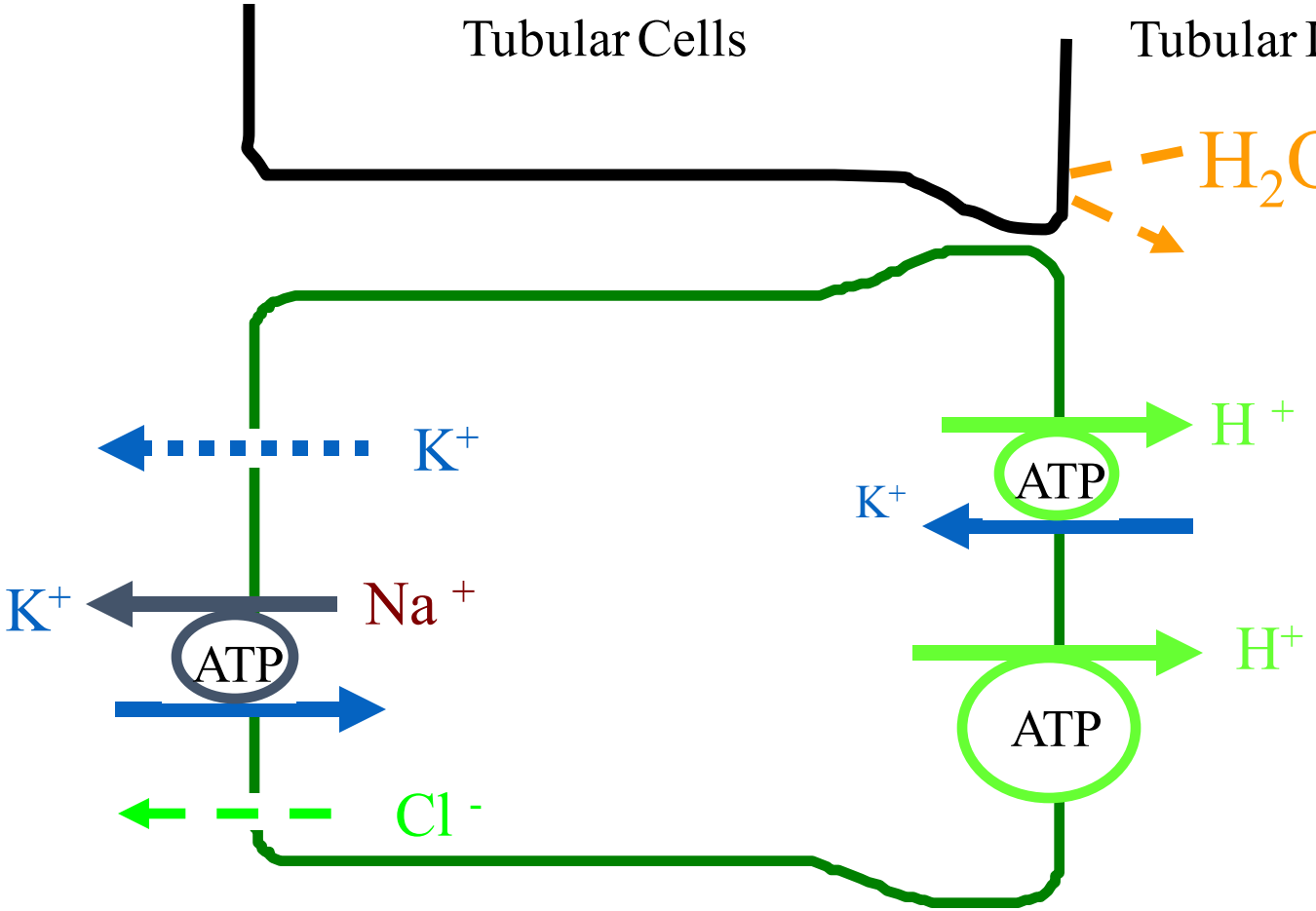


Figure 28-13. Type A and type B intercalated cells of the collecting tubule. Type A cells contain hydrogen-ATPase and hydrogen-potassium-ATPase in the luminal membrane and secrete hydrogen ions while reabsorbing bicarbonate and potassium ions in acidosis. In type B cells the hydrogen-ATPase and hydrogen-potassium-ATPase transporters are located in the basolateral membrane and reabsorb hydrogen ions while secreting bicarbonate and potassium ions in alkalosis.

Late Distal and Cortical Collecting Tubules <u>Intercalated Cells</u> –Secrete H^+

Note that the Na^+/K^+ ATPase pump is located on the basolateral side of the cell on the interstitial side. On the luminal side, there is the H^+/K^+ ATPase pump as well as the H^+ ATPase.

The presence of Na^+/K^+ ATPase on the interstitial side facilitates the reabsorption of K^+. As H^+ are secreted, K^+ accumulate within the cell. This increase in intracellular potassium leads to an increase in the reabsorption of K^+.



Because the H^+ pumps are located on the luminal side of the cell, and they secrete H^+. Then they are type A intercalated cells.

Transport characteristics of medullary collecting ducts

Unlike the cortical collecting tubule, the medullary collecting duct is permeable to urea, and there are special urea transporters that facilitate urea diffusion

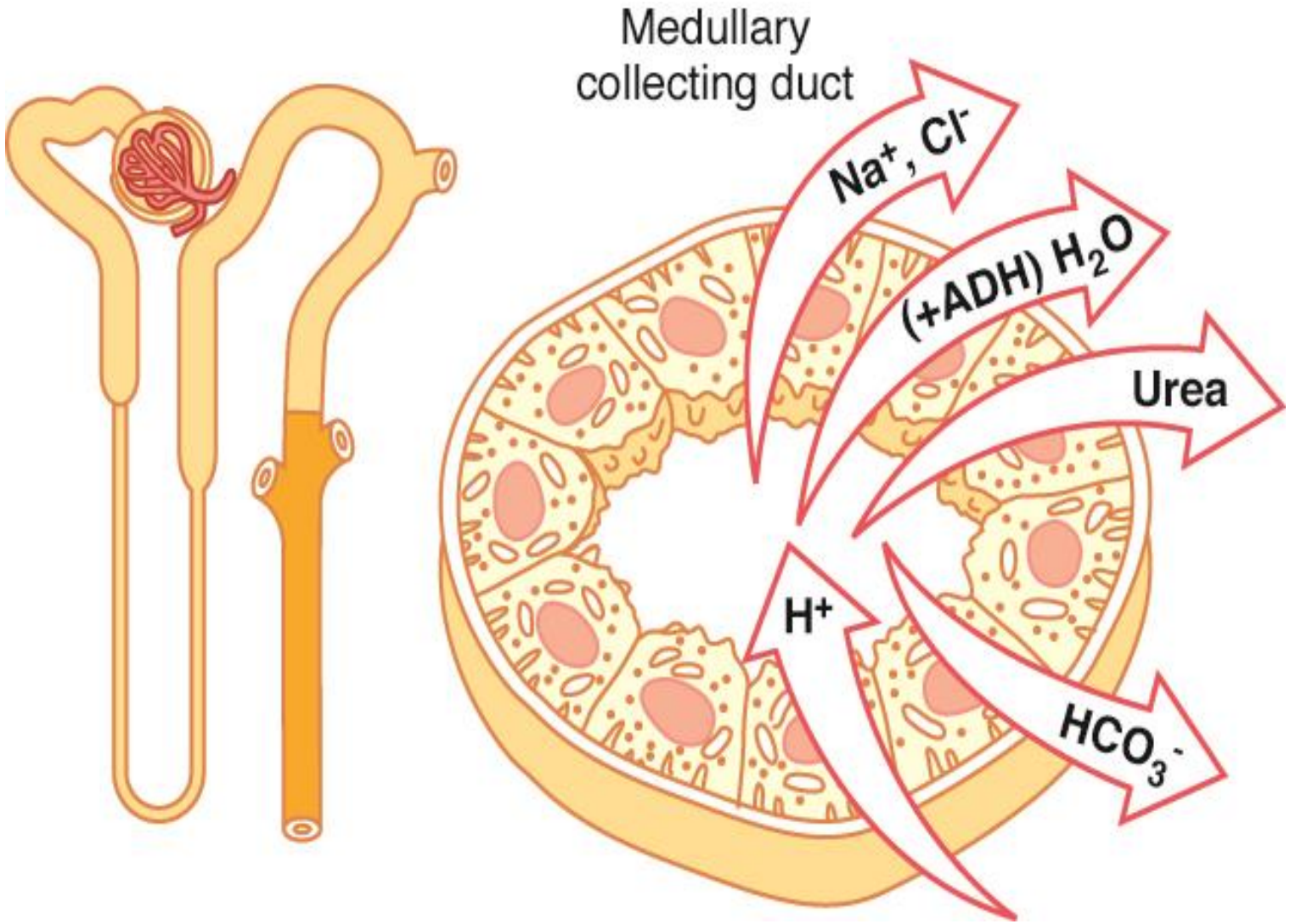
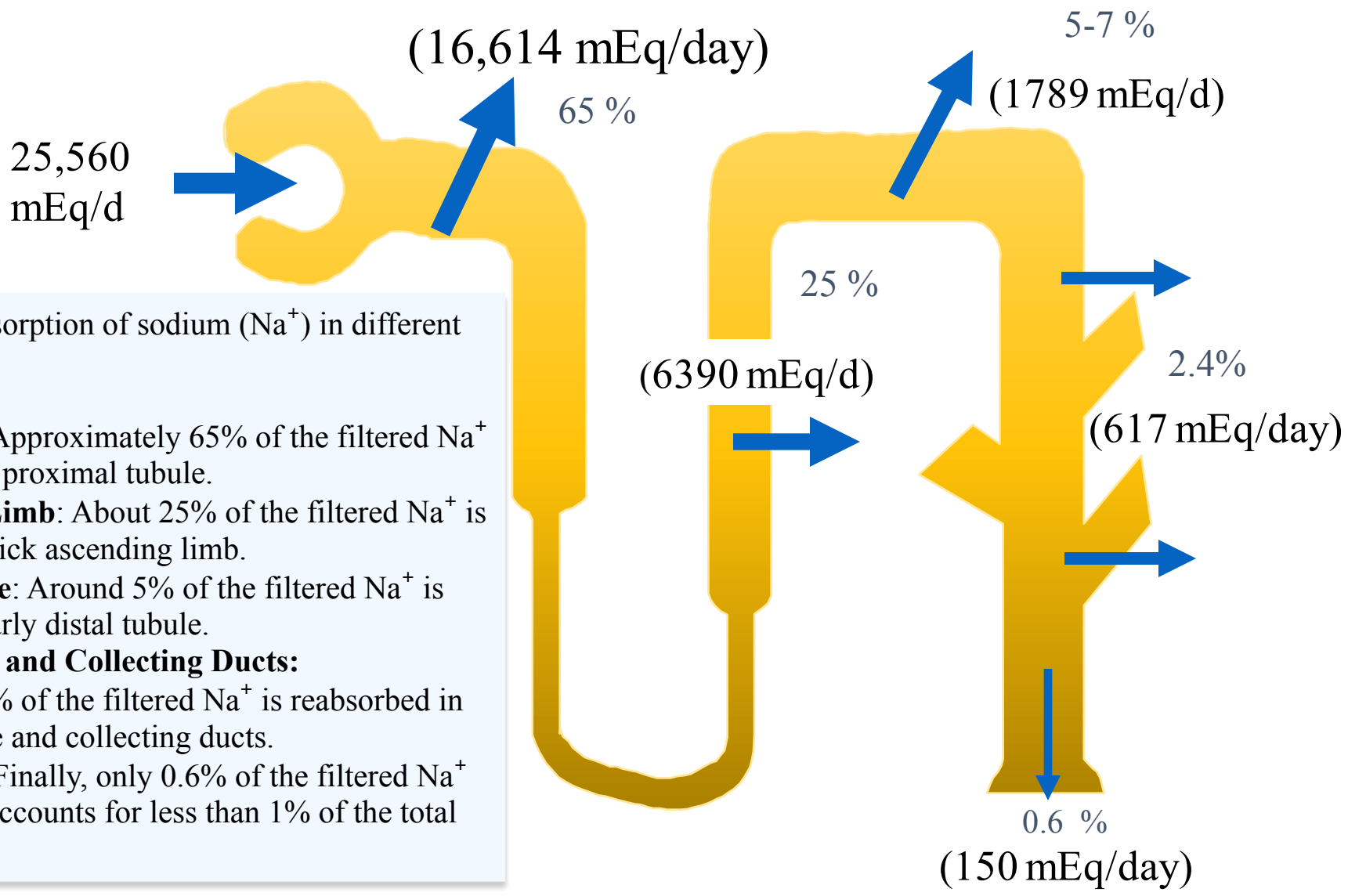


Figure 27-13

Normal Renal Tubular Na^+ Reabsorption



If we examine the reabsorption of sodium (Na^+) in different parts of the nephron:

- **Proximal Tubule**: Approximately 65% of the filtered Na^+ is reabsorbed in the proximal tubule.
- **Thick Ascending Limb**: About 25% of the filtered Na^+ is reabsorbed in the thick ascending limb.
- **Early Distal Tubule**: Around 5% of the filtered Na^+ is reabsorbed in the early distal tubule.
- **Late Distal Tubule and Collecting Ducts:** Approximately 2.4% of the filtered Na^+ is reabsorbed in the late distal tubule and collecting ducts.
- **Excretion of Na^+**: Finally, only 0.6% of the filtered Na^+ is excreted, which accounts for less than 1% of the total filtered Na^+.

Concentrations of solutes in different parts of the tubule depend on relative reabsorption of the solutes compared to water

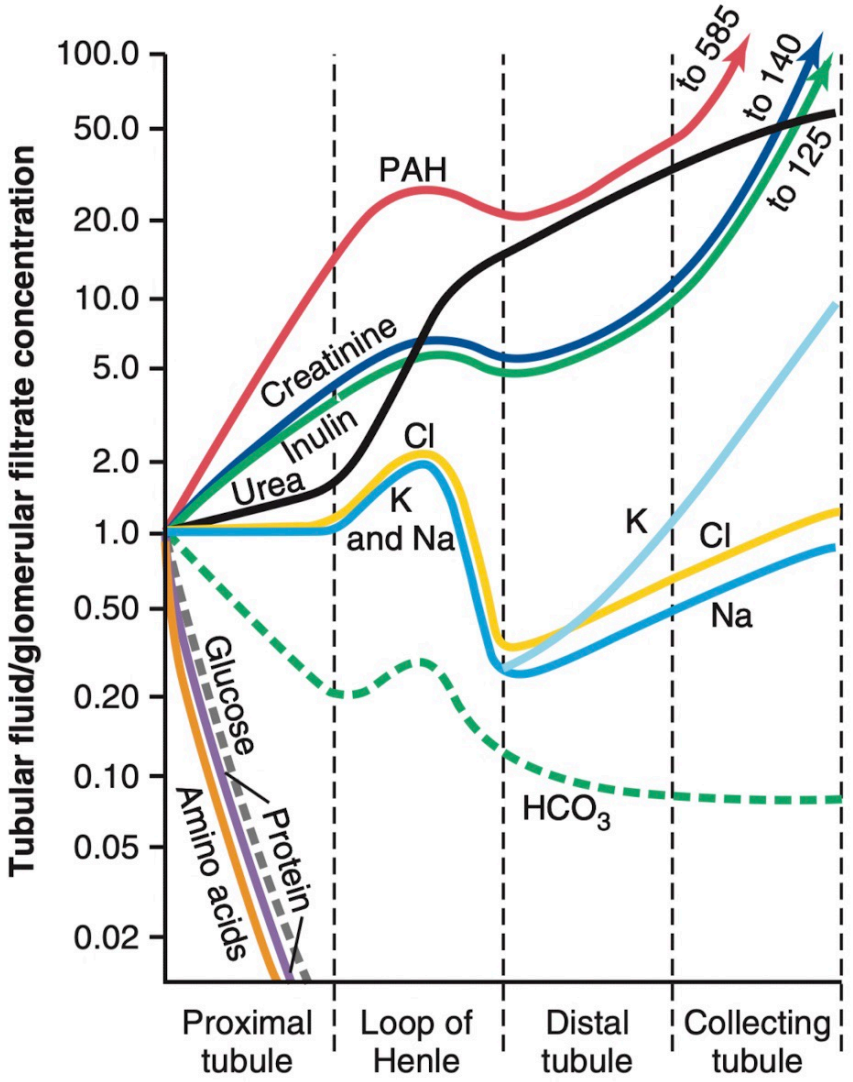
- If water is reabsorbed to a greater extent than the solute, the solute will become more concentrated in the tubule (e.g. creatinine, inulin)

- If water is reabsorbed to a lesser extent than the solute, the solute will become less concentrated in the tubule (e.g. glucose, amino acids)

Changes in average concentrations of different substances at different points in the tubular system relative to the concentration of that substance in the plasma and in the glomerular filtrate. A value of 1.0 indicates that the concentration of the substance in the tubular fluid is the same as the concentration of that substance in the plasma. Values below 1.0 indicate that the substance is reabsorbed more avidly than water, whereas values above 1.0 indicate that the substance is **reabsorbed to a lesser extent** than water or is **secreted into the tubules.**

- The secretion and reabsorption rates determine the concentration of a substance in the tubule.

Inulin, a polysaccharide used to measure GFR, is **not reabsorbed or secreted** by the renal tubules. Changes in inulin concentration at different points along the renal tubule, therefore, **reflect changes in the amount of water present in the tubular fluid.**
inulin clearance = GFR.



Changes in concentrations of substances in the renal tubules

Concentrations of solutes in different parts of the tubule depend on relative reabsorption of the solutes compared to water

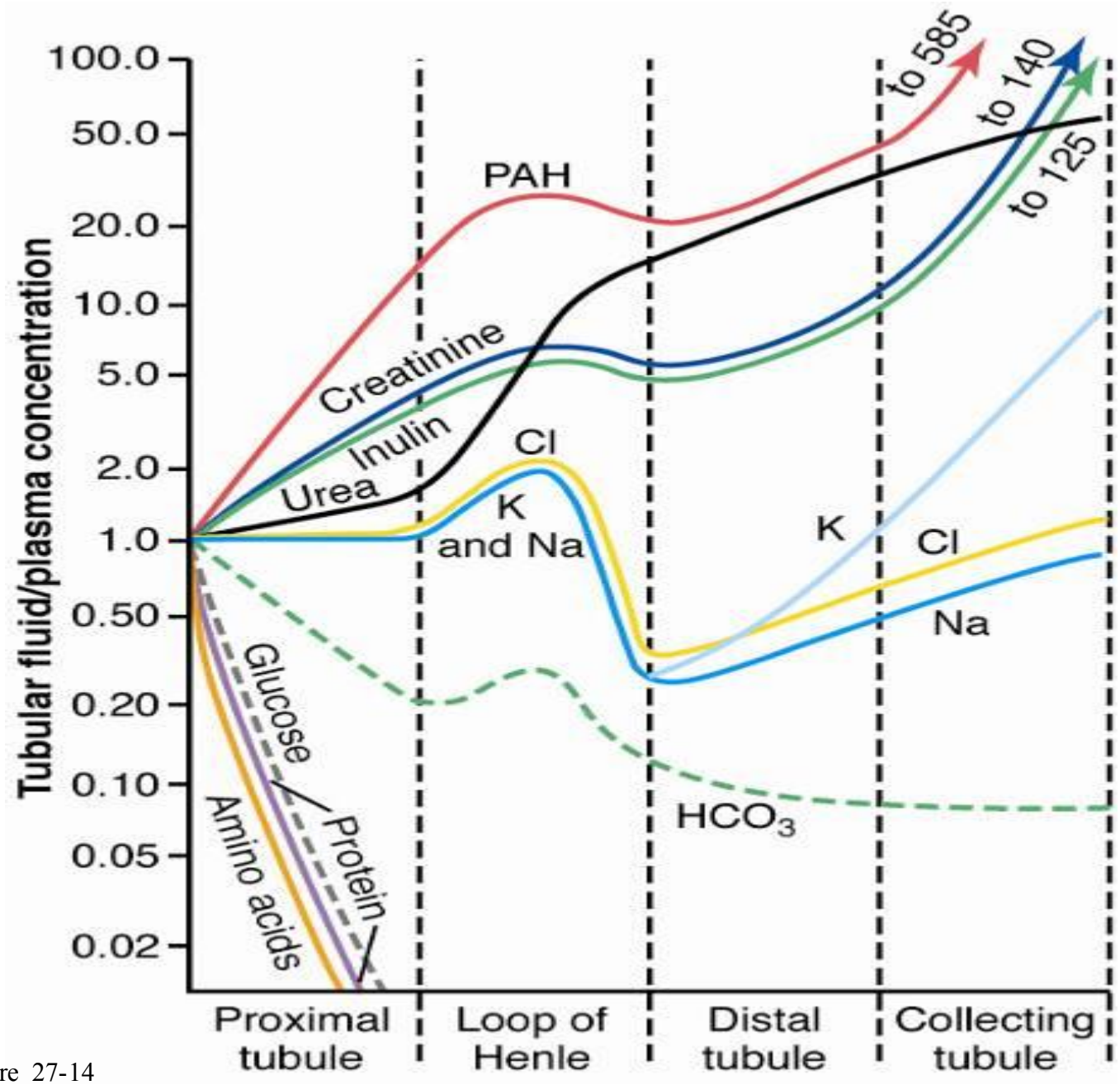


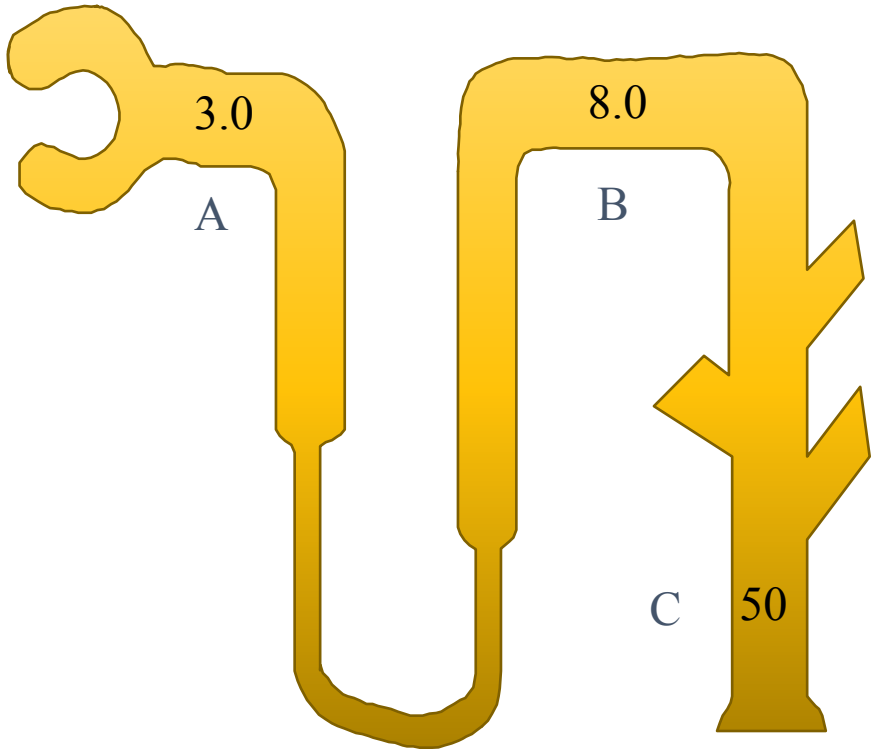
Figure 27-14

The figure below shows the concentrations of inulin at different points along the tubule, expressed as the tubular fluid/plasma (TF/P_{inulin}) concentration of inulin. If inulin is not reabsorbed by the tubule, what is the percentage of the filtered water that has been reabsorbed or remains at each point ? What percentage of the filtered water has been reabsorbed up to that point?

A = 1/3 (33.33 %) remains
66.67 % reabsorbed

B = 1/8 (12.5 %) remains
87.5 % reabsorbed

C = 1/50 (2.0 %) remains
98.0 % reabsorbed



The ratio of tubular fluid to plasma inulin concentration can serve as a measure of water reabsorption by the renal tubules. When inulin is filtered, its concentration in the tubular fluid is the same as in the plasma, resulting in a ratio of 1. It is important to note that while plasma concentration of inulin remains relatively constant, the concentration in the tubular fluid can vary. Therefore, by comparing the tubular fluid/plasma inulin concentration ratio, we can assess the extent of water reabsorption by the renal tubules.

- **Point A** → the tubular fluid/plasma concentration ratio for inulin increases to about 3. This indicates that the concentration of inulin in the tubular fluid is three times higher than in the plasma and glomerular filtrate. Since inulin is neither secreted nor reabsorbed in the tubules, a tubular fluid/plasma concentration ratio of 3 means that only one third (33.33%) of the filtered water remains in the renal tubule, while two thirds (66.67%) of the filtered water has been reabsorbed as the fluid passes through the proximal tubule. This change is primarily due to the reabsorption of water, which leads to a concentration change, rather than any changes in the behavior of inulin itself.

- **Point B** → the tubular fluid/plasma inulin concentration ratio becomes 8. This means that only 1/8 (12.5%) of the water that was initially filtered remains in the tubule, while the remaining 7/8 (87.5%) of the filtered water has been reabsorbed.

- **Point C** → the tubular fluid/plasma inulin concentration ratio increases to 50. This indicates that only 1/50 (2%) of the water that was originally filtered remains in the tubule, while the remaining 49/50 (98%) of the filtered water has been reabsorbed.

Regulation of Tubular Reabsorption

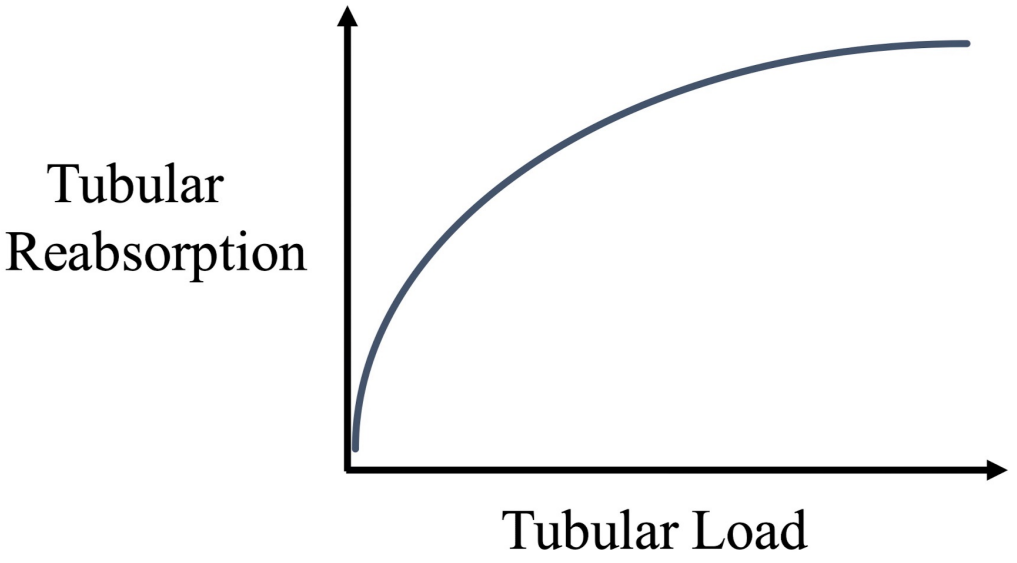
- Glomerulotubular Balance
- Peritubular Physical Forces
- Hormones
    - aldosterone
    - angiotensin II
    - antidiuretic hormone (ADH)
    - natriuretic hormones (ANF)
    - parathyroid hormone
- Sympathetic Nervous System
- Arterial Pressure (pressure natriuresis)
- Osmotic factors

The tubular reabsorption mechanism involves the transport of essential nutrients from the tubular lumen into the tubular cells. From there, these nutrients traverse the interstitium and continue their journey until they reach the peritubular capillaries. The transfer of nutrients from the tubular cells to the peritubular capillaries is facilitated by a process known as **bulk flow.** *2020 sheet*

Glomerulotubular Balance

Glomerulotubular balance is defined as the **intrinsic** ability of the tubules to increase their reabsorption rate in response to increased tubular load (increased tubular inflow).

- Tubular load = GFR*Plasma concentration of the substance.
- As the tubular load increases, the tubular reabsorption increases as well (Until it reaches the plateau where there is no further increase in reabsorption).
- This mechanism **operates intrinsically** within the kidney, meaning that it functions even when the kidney is isolated from external influences.



Tubuloglomerular balance regulates the GFR based on tubular flow rate, while **glomerulotubular balance** adjusts tubular reabsorption based on the filtered load of substances. Both mechanisms contribute to maintaining the overall balance of fluid and solute handling in the kidney.

Importance of Glomerulotubular Balance in Minimizing Changes in Urine Volume

GFR	Reabsorption	% Reabsorption	Urine Volume
	no glomerulotubular balance		
125	124	99.2	1.0
150	124	82.7	26.0
	"perfect" glomerulotubular balance		
150	148.8	99.2	1.2

Importance of Glomerulotubular Balance in Minimizing Changes in Urine Volume

1. No Glomerulotubular Balance

When glomerulotubular balance is absent, an increase in the glomerular filtration rate (GFR) from the normal value of 125 ml/min to 150 ml/min leads to a rise in urine volume from 1 ml/min to 26 ml/min. In normal conditions, around 99.2% of filtered substances are reabsorbed, resulting in minimal urine production. However, without glomerulotubular balance, there is a loss of filtered substances and reabsorption does not adjust to the elevated GFR. Consequently, the reabsorption percentage decreases, calculated as 124/150 * 100, which is 82.7%. This decrease in reabsorption percentage contributes to the increased urine volume observed in the absence of glomerulotubular balance.

2. Perfect glomerulotubular balance

In the presence of glomerulotubular balance, reabsorption is increased to match the increase in tubular load, thereby maintaining a constant glomerular filtration rate (GFR).
In a normal glomerulotubular balance scenario, the percentage of reabsorption is very close to 100%. For example, if the reabsorption amount is 148.8 ml out of a tubular load of 150 ml, the reabsorption percentage would be calculated as (148.8/150) * 100% = 99.2%.
In the case of perfect glomerulotubular balance, the overall urine excretion does not increase significantly. This indicates that the balance between filtration and reabsorption is maintained, resulting in minimal changes in urine output.

Therefore, the presence or absence of glomerulotubular balance significantly impacts urine volume. Conversely, a well-functioning glomerulotubularween filtration and reabsorption occurs, leading to increased urine output. Conversely, a well-functioning glomerulotubular balance maintains a stable GFR and minimal urine production

The table compares the reabsorption in two situations: one where glomerulotubular balance is absent and the other where glomerulotubular balance is functioning perfectly.

GFR	Reabsorption	% Reabsorption	Urine Volume
	no glomerulotubular balance		
125	124	99.2	1.0
150	124	82.7	26.0
	“perfect” glomerulotubular balance		
150	148.8	99.2	1.2

Perfect glomerulotubular balance encompasses both tubuloglomerular balance + glomerulotubular balance.

Peritubular capillary reabsorption

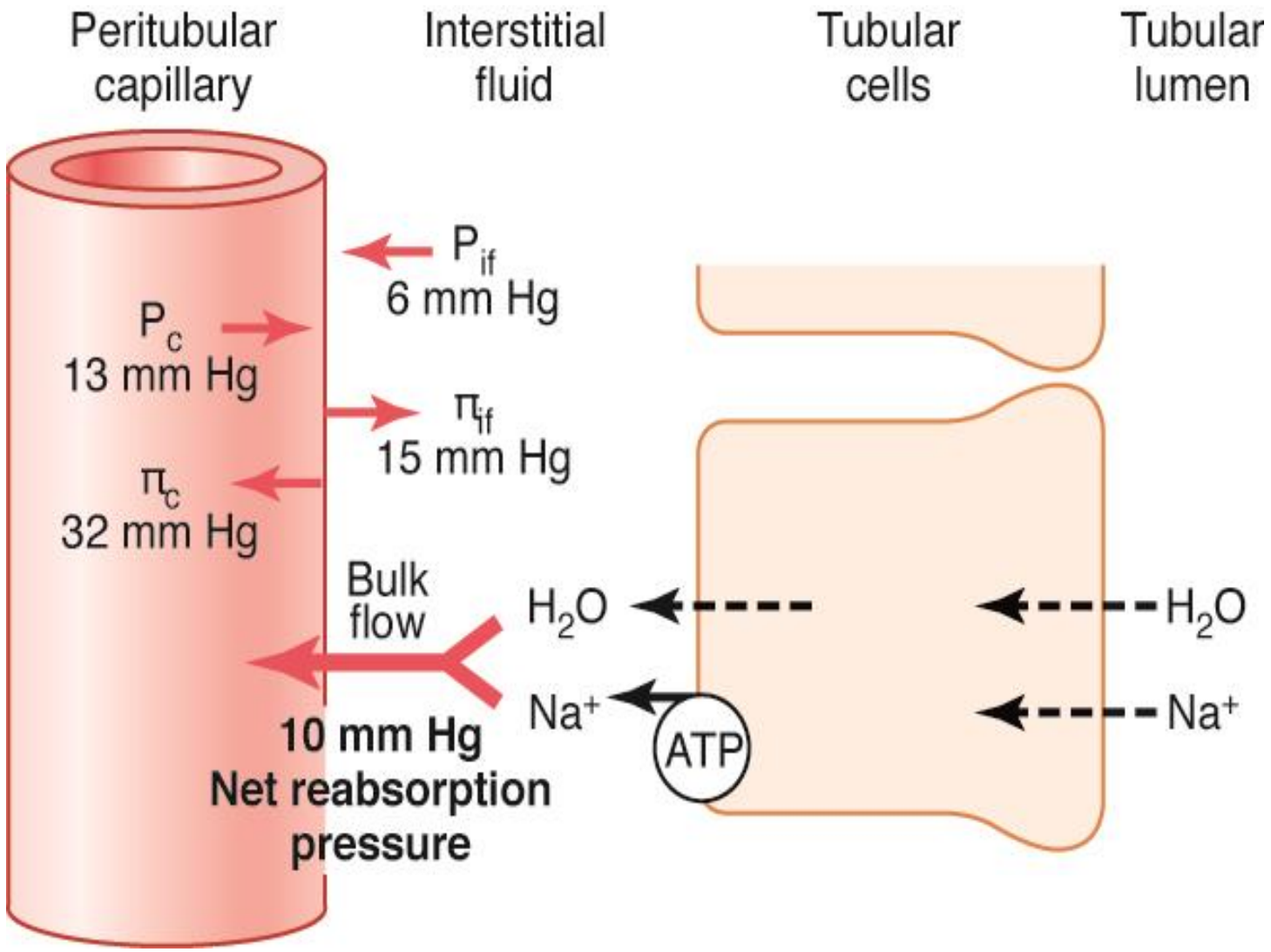


Figure 27-15

Peritubular capillary reabsorption

The net reabsorptive force represents the sum of the hydrostatic and colloid osmotic forces that either favor or oppose reabsorption across the peritubular capillaries.
These forces include:
(1) Hydrostatic pressure inside the peritubular capillaries (peritubular hydrostatic pressure [P_c]), which opposes reabsorption.
(2) Hydrostatic pressure in the renal interstitium (P_{if}) outside the capillaries, which favors reabsorption.
(3) Colloid osmotic pressure of the peritubular capillary plasma proteins (π_c), which favors reabsorption.
(4) Colloid osmotic pressure of the proteins in the renal interstitium (π_{if}), which opposes reabsorption.

Reabsorption = K_f × Net reabsorptive force

[P_c] = -13 mm Hg and (P_{if})= +6 mm Hg, there is a positive hydrostatic pressure gradient from the peritubular capillary to the interstitial fluid of about -7 mm Hg, which **opposes fluid reabsorption**. This opposition to fluid reabsorption is more than counterbalanced by the colloid osmotic pressures that favor reabsorption.
(π_c)= +32 mm Hg, and (π_{if}) = -15 mm Hg, causing a net colloid osmotic force of about +17 mm Hg, **favoring reabsorption.**

The net hydrostatic forces that oppose reabsorption + the net colloid osmotic forces that favor reabsorption = net reabsorptive force.

17 −7= +10 mm Hg → Represent the driving force of the bulk flow toward the capillaries.

- The value that favors reabsorption is positive (+), while the value that opposes reabsorption is negative (-).

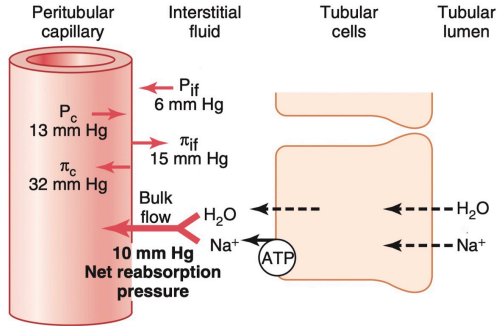


Figure 28-16. Summary of the hydrostatic and colloid osmotic forces that determine fluid reabsorption by the peritubular capillaries. The numerical values shown are estimates of the normal values for humans. The net reabsorptive pressure is normally about 10 mm Hg, causing fluid and solutes to be reabsorbed into the peritubular capillaries as they are transported across the renal tubular cells. ATP, adenosine triphosphate; P_c, peritubular capillary hydrostatic pressure; P_{if} , interstitial fluid hydrostatic pressure; π_c, peritubular capillary colloid osmotic pressure; π_{if}, interstitial fluid colloid osmotic pressure.

Not mentioned by the doctor. however, it's quite important to note :
The other factor that contributes to the high rate of fluid reabsorption in the peritubular capillaries is a large filtration coefficient (Kf) because of the high hydraulic conductivity and large surface area of the capillaries. Because the reabsorption rate is normally about 124 ml/min and net reabsorption pressure is 10 mm Hg, K normally is about 12.4 ml/min/mm Hg.

Peritubular capillary reabsorption

If any changes occur and the net reabsorption pressure decreases, this results in a decrease in bulk flow, and fluid will accumulate intracellularly and backflow due to the presence of loose or less tight "**tight junctions"**. Reabsorption is not solely dependent on transporters within the tubular cells, but also on hemodynamic forces present in the peritubular capillaries and interstitiumorce decreases by less than 10 mmHg, the back leak increases accordingly.

Reabsorption is not solely dependent on transporters within the tubular cells, but also on hemodynamic forces present in the peritubular capillaries and interstitium.

Calculation of tubular reabsorption or secretion from renal clearances

The renal tubules play a crucial role in determining whether a substance is **reabsorbed** or **secreted** by the kidneys.

- By comparing the rates of glomerular filtration and renal excretion of a substance, we can determine if there is a net reabsorption or net secretion occurring in the tubules.

Calculation of Tubular Reabsorption

(when Excret s < Filt s)

Filtered load

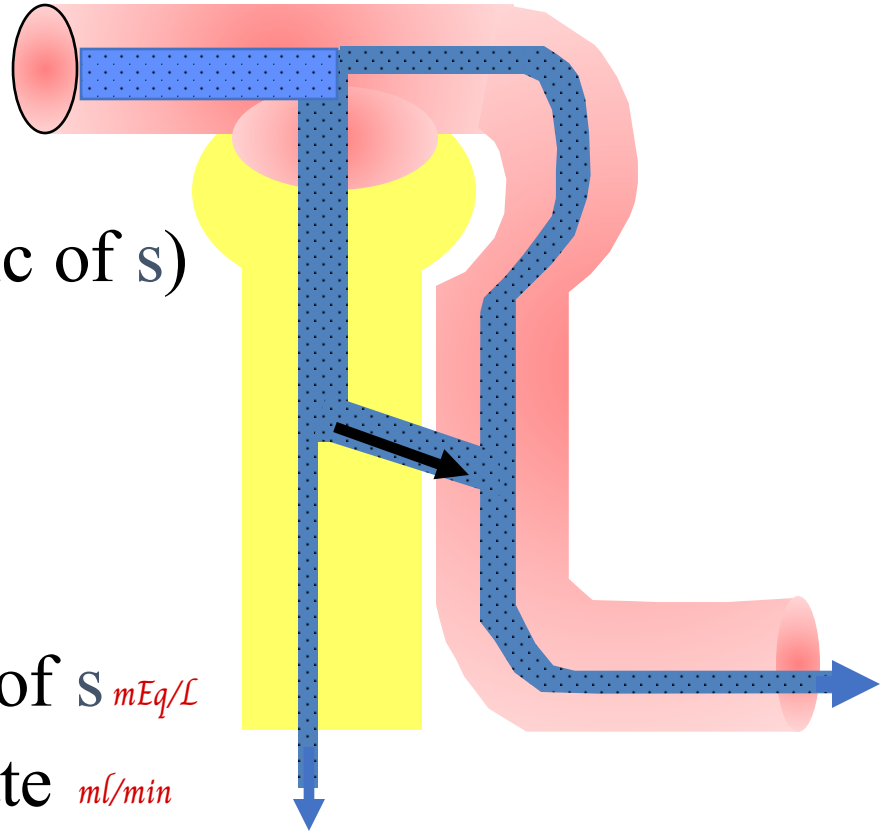
Reabsorption = Filtration -Excretion

ml/min

Filt s = GFR x Ps

(Ps = Plasma conc of s)

mEq/L



Excret s = Us x V

Us = Urine conc of s mEq/L

V = urine flow rate ml/min

V (usually) = 1 ml/min

concentration of a substance in a 24-hour urine collection

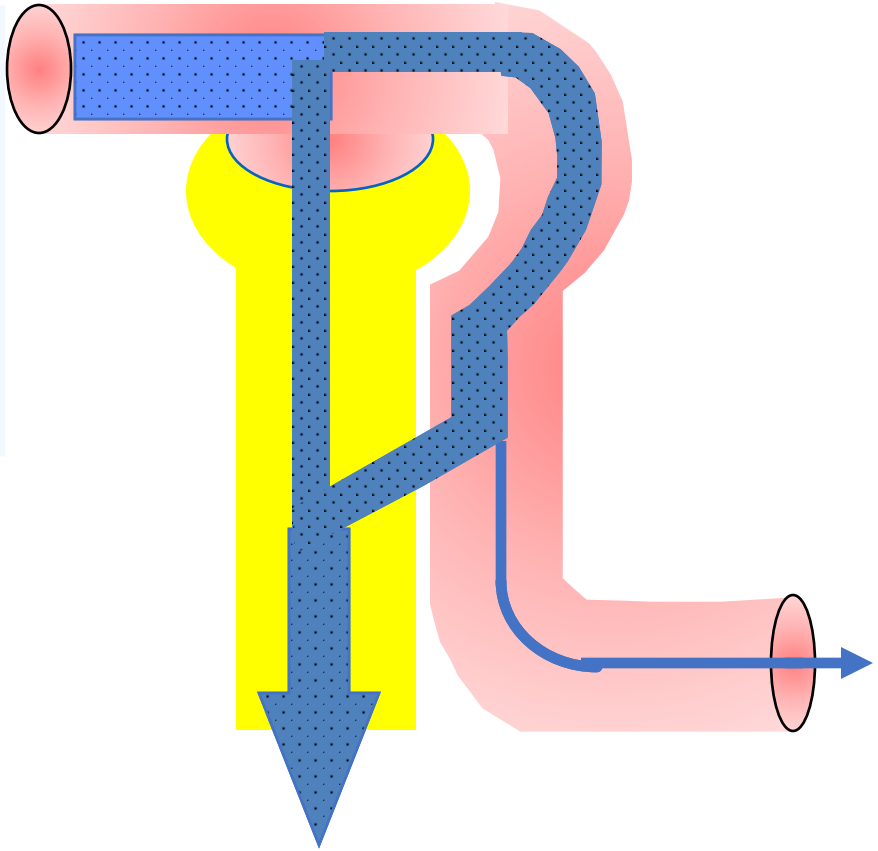
Calculation of Tubular Secretion

(when Excret s > Filt s)

Secretion = Excretion - Filtration

If, after applying the reabsorption formula, the result is a negative number, it suggests that the substance has been secreted rather than reabsorbed by the renal tubules. The negative value indicates that the rate of excretion (Us X V) is greater than the filtered load (GFR X Ps) of the substance.

Therefore, a negative value obtained from the reabsorption formula indicates that the substance is undergoing tubular **secretion** rather than reabsorption in the kidneys.



Filt s = GFR x Ps

Excret s = Us x V

Example: Given the following data, calculate the rate of Na^+ filtration, excretion, reabsorption, and secretion

GFR =100 ml/min (0.1 L/min)

P_{Na} = 140 mEq/L

urine flow = 1 ml/min (.001 L/min)

urine Na conc = 100 mEq/L

Make sure to carefully consider the units used when doing calculations!

Filtration Na = GFR x P_{Na}

= 0.1 L/min x 140 mEq/L = 14 mEq/min

Excretion Na = Urine flow rate x Urine Na conc

=.001 L/min x 100 mEq/L

= 0.1 mEq/min

Example: Given the following data, calculate the rate of Na^+ filtration, excretion, reabsorption, and secretion

GFR =100 ml/min;
P_{Na} = 140 mEq/L
urine flow = 1 ml/min;
urine Na conc = 100 mEq/L

Filtration Na = 0.1 L/min x 140 mEq/L = 14 mEq/min
Excretion Na = .001 L/min x 100 mEq/L = 0.1 mEq/min
Reabsorption Na = Filtration Na - Excretion Na

Reabs Na = 14.0 - 0.1 = 13.9 mEq/min

Secretion Na = There is no net secretion of Na since Excret Na < Filt Na

However, if the answer is negative, it indicates that secretion is occurring rather than reabsorption

Transport Maximum

Some substances have a maximum rate of tubular transport due to saturation of carriers, limited ATP, etc

- Transport Maximum: Once the transport maximum is reached for all nephrons, further increases in tubular load are not reabsorbed and are excreted.

- Threshold is the tubular load at which transport maximum is exceeded in some nephrons. This is not exactly the same as the transport maximum of the whole kidney because some nephrons have lower transport max's than others.

- Examples: glucose, amino acids, phosphate, sulphate

Does Na+ have Transport Maximum?

Back to the previous lecture……

Substances that are actively transported but don't exhibit a transport maximum

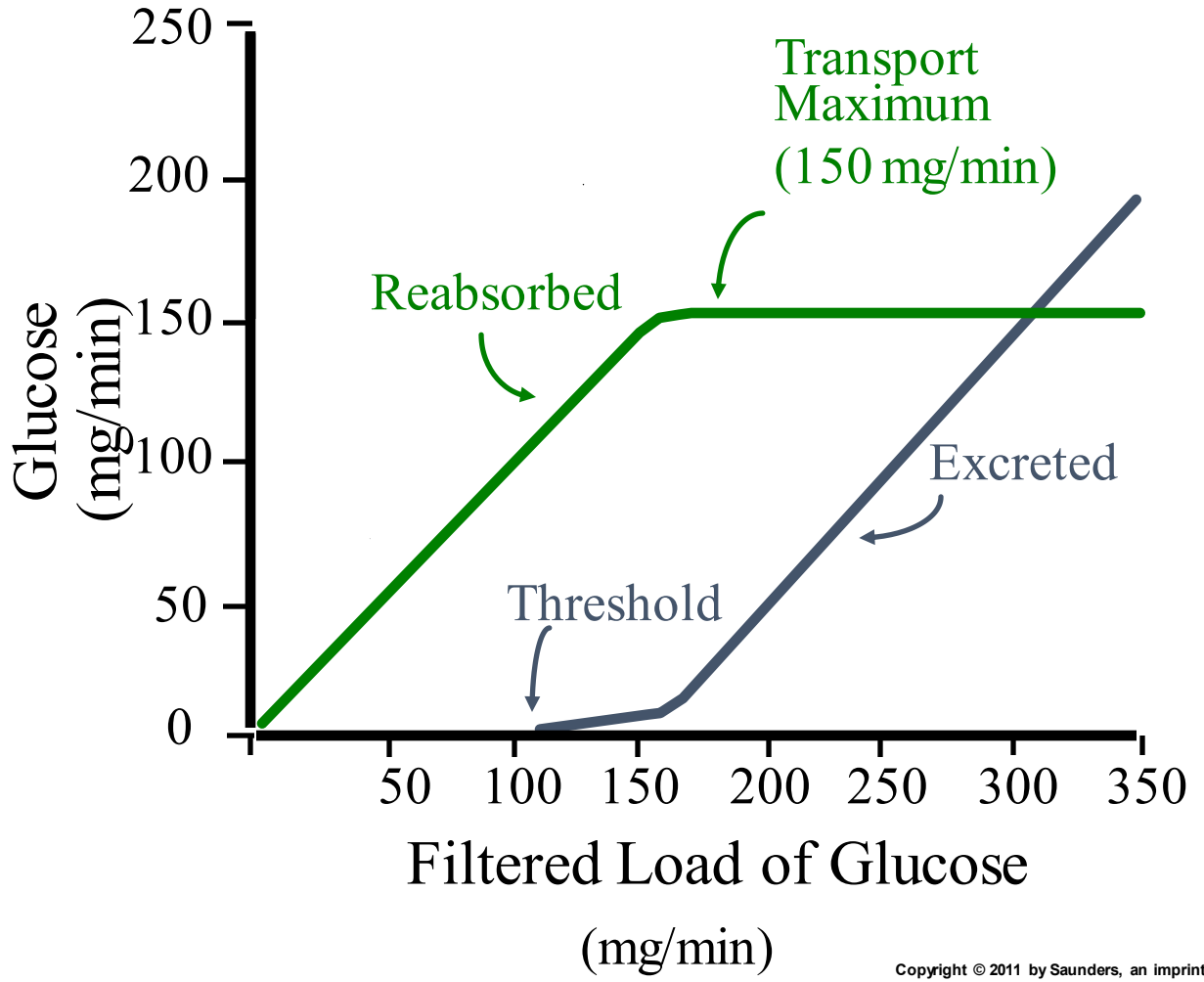
Unlike glucose and other substances that have a transport maximum, sodium follows the **gradient-time transport mechanism**. This means that the filtration rate of sodium (Na^+) depends on two factors:

1. Na^+ electrochemical gradient: The concentration gradient and the electrical charge difference across the membrane influence the movement of sodium. The greater the gradient, the higher the rate of sodium reabsorption or secretion.
2. Time of contact: The duration of time that the fluid containing the sodium remains in contact with the luminal membrane of the tubule also affects the rate of sodium transport. The longer the fluid remains in contact with the membrane, the more time there is for sodium to be reabsorbed or secreted.

This observation means that the greater the concentration of sodium in the proximal tubules, the greater its reabsorption rate. Also, the slower the flow rate of tubular fluid, the greater the percentage of sodium that can be reabsorbed from the proximal tubules.
In the more distal parts of the nephron, the epithelial cells have much tighter junctions and transport much smaller amounts of sodium. In these segments, sodium reabsorption exhibits a transport maximum similar to that for other actively transported substances. Further-more, this transport maximum can be increased by certain hormones, such as aldosterone.

A uninephrectomized patient with uncontrolled diabetes has a GFR of 90 ml/min, a plasma glucose of 200 mg% (2mg/ml), and a transport max (Tm) shown in the figure. What is the glucose excretion for this patient?

1. 0 mg/min
2. 30 mg/min
3. 60 mg/min
4. 90 mg/min
5. 120 mg/min



Answer: $Filt_{Glu}$ = $(GFR \times P_{Glu}) = (90 \times 2) = 180$ mg/min

$Reabs_{Glu}$ = T_{max} = 150 mg/min

$Excret_{Glu}$ = 30 mg/min In normal individuals, the urinary excretion of glucose = zero.

Answer : B (30 mg/min)

GFR = 90 ml/min
P_{Glu} = 2 mg/ml
T_{max} = 150 mg/min

- Tm = 150 mg/min, which means that the transporters in the kidney can only handle a maximum transport rate of 150 mg/min. However, the tubular load is 180 mg/min, resulting in an excess of 30 mg/min that cannot be fully reabsorbed. Based on the figure, it can be observed that the transport maximum was already reached at a filtered load of 180, causing the remaining 30 mg/min to be excreted. Therefore, the answer is B.