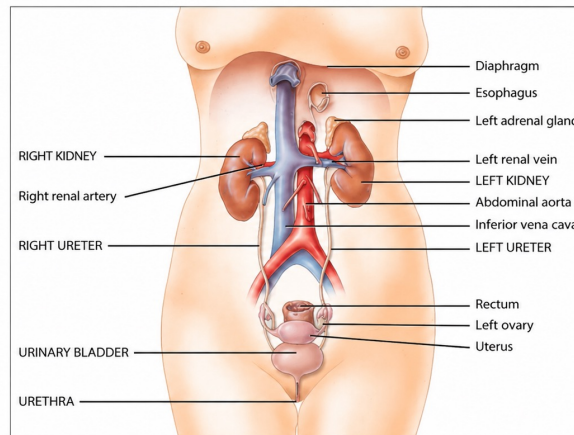


The Urinary System
Focus: The Kidney

Overview of the Urinary System

The urinary system is made up of several organs:

- **two kidneys;**
- **two ureters;**
- **urinary bladder;**
- **urethra.**



The urinary system consists of:
two kidneys,
two ureters,
one urinary bladder,
one urethra.

Its main function is to maintain a constant composition of the body's internal environment.

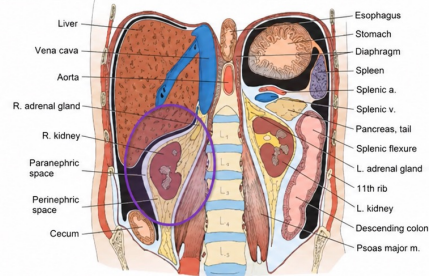
Among these organs, we will study the kidney in detail, since it represents the main functional organ of the urinary system. The kidney is the great guardian of internal balance: an organ specialized in blood filtration, capable of precisely controlling water, salts, pH, and the volume of body fluids, thereby maintaining a stable environment in which our cells can survive.

Anatomical review

The kidneys are a pair of bean-shaped **organs**.

They occupy the **posterior topographic region of the abdomen** (specifically they are **retroperitoneal** organs) and are **located on the sides of the vertebral column** in a space called the “renal lodge”.

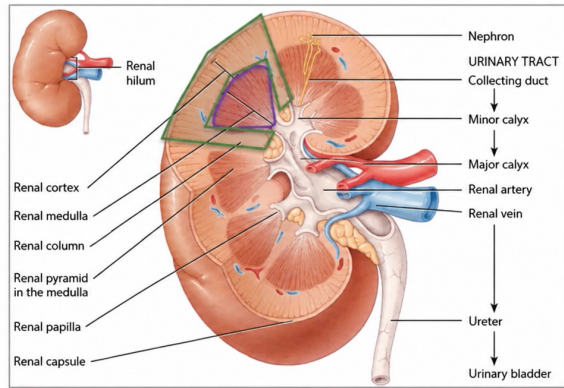
It is interesting to note how the **right kidney**, due to the bulk of the liver in the right hypochondrium, is positioned a little **lower** (2cm) than the **left**.



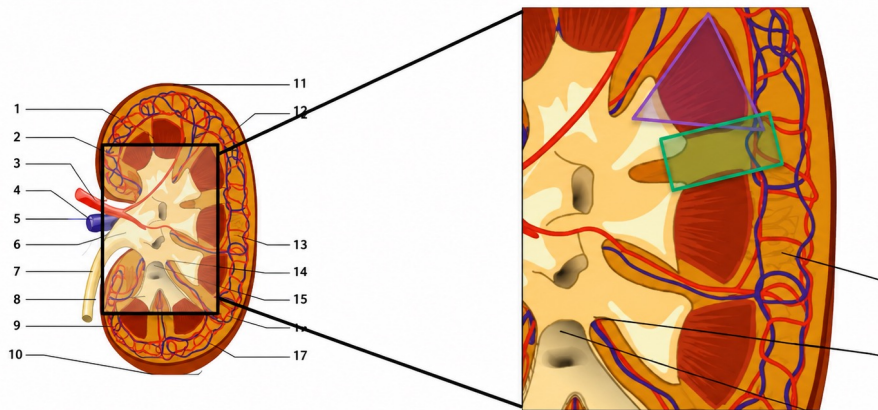
Each kidney is surrounded by the **renal capsule**, a transparent connective tissue that provides containment and protection, and by **adipose tissue**.

Internally, they are distinguishable:

- **cortical zone**, more external;
- **medullary zone**, more internal.



The renal medulla is organized into different **renal pyramids** (Malpighi's pyramids). The spaces between one pyramid and another are occupied by extensions of the cortex called **renal columns**.

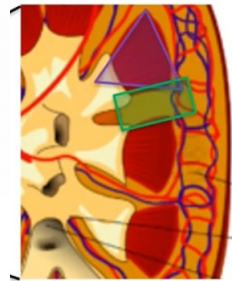


Each renal pyramid ends, at the apex, in the **renal papilla**. The surface of the renal papilla is characterized by a series of holes (cribriform area), through which the urine produced by each nephron contained in the pyramid is poured into the **minor calyces**.

The minor calyces are funnel-shaped structures that surround the base of the renal pyramids. Several minor calyces converge to form the **major calyces**, which in turn converge in the **renal pelvis**.

Therefore:

MINOR CALYCES → MAJOR CALYCES → RENAL PELVIS

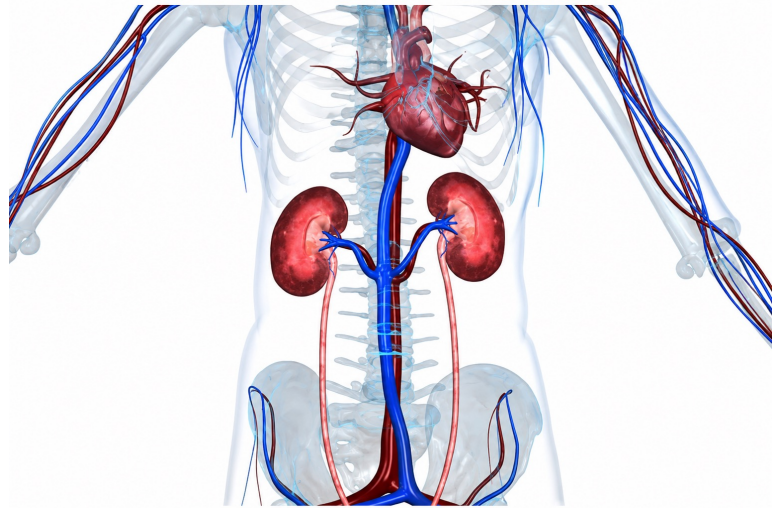


Through these openings, the urine produced by the nephrons is discharged into the:
minor calyces.
The minor calyces merge into the:
major calyces,
which in turn convey the urine into the:
renal pelvis.

Blood flow pathway

From the heart to the kidneys

From the kidneys to the systemic circulation



The kidneys are part of the systemic circulation and continuously receive blood from the heart.

The circulatory system does not function in series, as if blood first passed through all tissues and only afterward through the kidneys. Instead, it functions in parallel: the heart simultaneously distributes blood to all organs of the body — the brain, muscles, liver, intestines, and also the kidneys.

Oxygenated blood is pumped from the left ventricle into the aorta. The aorta descends into the abdomen as the abdominal aorta, from which two large vessels arise: the right renal artery and the left renal artery, which carry blood to the kidneys.

It is interesting to note that, although the kidneys are relatively small organs, they receive about 20–25% of the cardiac output. This enormous blood flow is necessary because the kidneys must continuously monitor the composition of the blood throughout the entire body.

The blood reaching the kidneys is blood that continuously circulates through the body: during its passage through tissues, it has delivered oxygen and nutrients and collected carbon dioxide, metabolites, and substances that must be eliminated or regulated. The kidneys are specifically responsible for filtering this blood, correcting its composition, and maintaining its balance constant.

The kidneys return blood to the systemic circulation through the renal veins.

Each kidney has a right renal vein and a left renal vein.

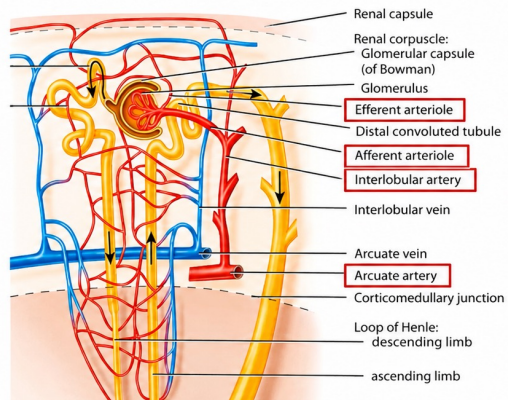
The renal veins collect the filtered blood. They empty directly into the inferior vena cava, which is the large venous vessel that carries blood back to the heart.

Kidney vascularization

The vascularization of the kidney is provided by the **renal arteries**, arterial branches paired with the **abdominal aorta**.

The renal arteries along their course divide into vessels of progressively smaller caliber until they reach the **afferent arterioles**.

The afferent arteriole branches into a **network of capillaries** called **renal glomerulus**. The mass of capillaries of the glomerulus resolves into an **efferent arteriole**, which by progressively subdividing into the **peritubular capillaries** drains the blood into the **renal vein**.



The kidney function

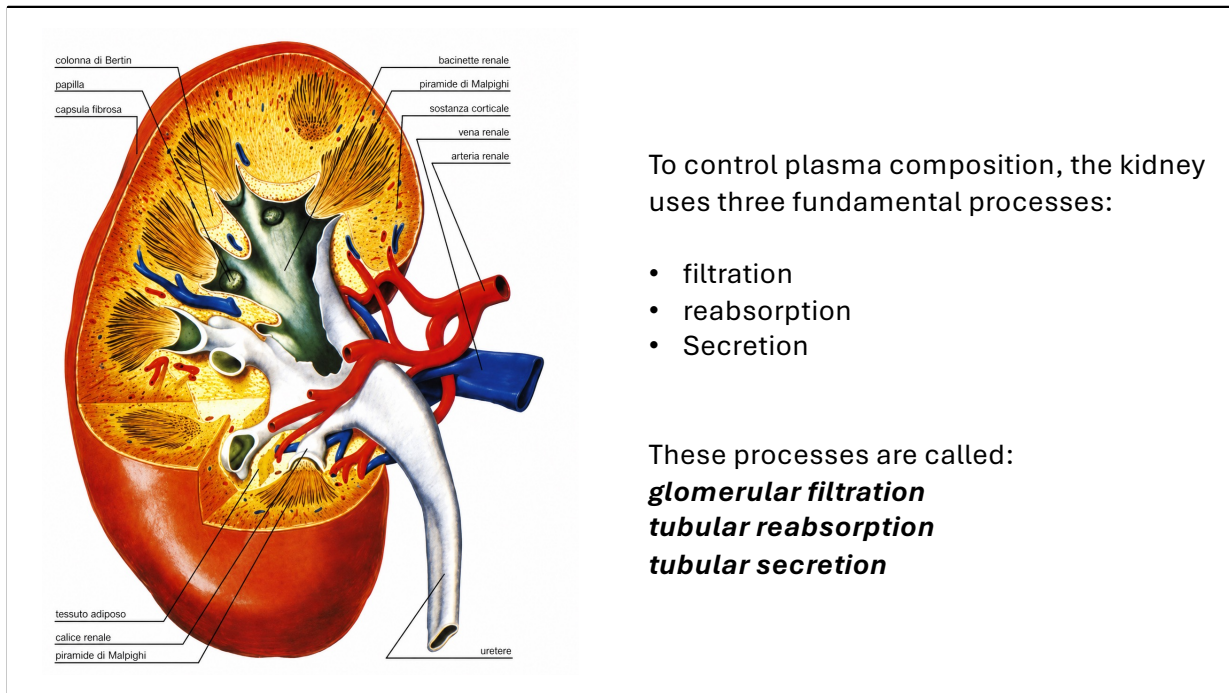
The kidney is responsible for the regulation of dynamic homeostasis, including:

Regulation of blood volume,
regulation of blood pressure,
regulation of pH,
elimination of waste products,
regulation of ionic concentration.

The kidneys also have an endocrine function and produce:

Calcitriol (the active form of vitamin D)

Erythropoietin (stimulate bone marrow to produce red blood cells)



To control plasma composition, the kidney uses three fundamental processes:

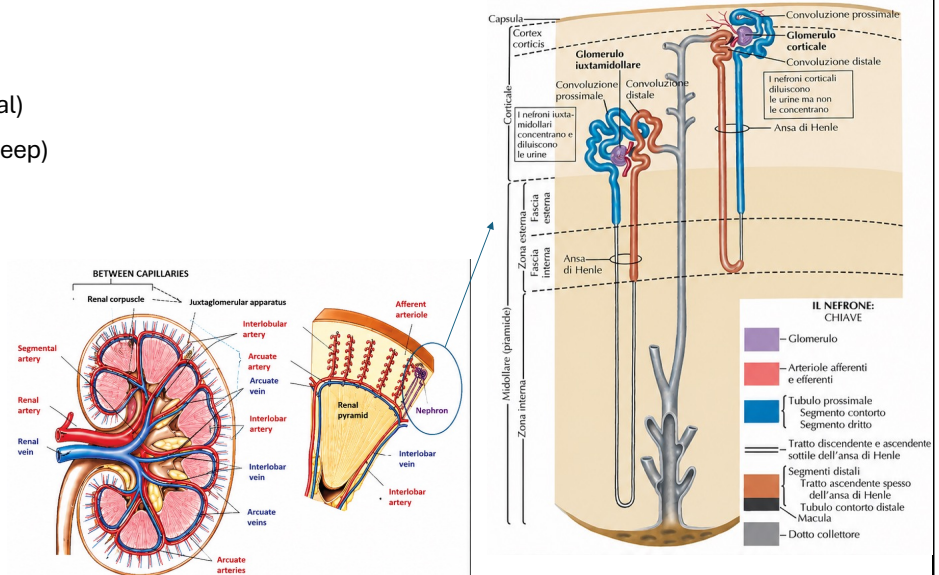
- filtration
- reabsorption
- Secretion

These processes are called:

glomerular filtration
tubular reabsorption
tubular secretion

The nephron

- **cortical** (superficial)
- **juxtamedullary** (deep)



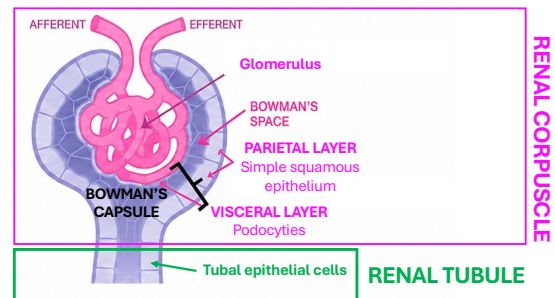
The nephron is the functional unit of the kidney.
 Each kidney contains more than 1 million nephrons.
 The kidneys contain two types of nephrons:
 cortical (superficial) nephrons,
 juxtamedullary (deep) nephrons.
 Most nephrons are cortical (about 80%).
 They are similar and differ mainly in their location and in the length of their segments.

The nephron is made up of two main elements:

- the **renal corpuscle**, where the **blood plasma** is **filtered**;
- the **renal tubule**, in which the **filtered fluid flows** (glomerular filtrate).

Each **renal corpuscle** is made up of:

- renal **glomerulus**;
- **glomerular capsule** (or **Bowman's capsule**).

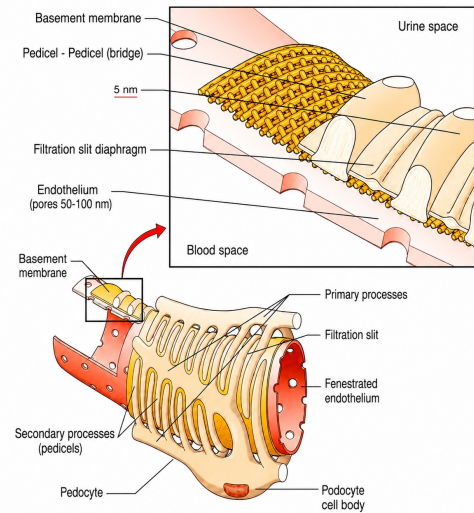


The renal glomerulus and Bowman's capsule

- **Tangle of capillaries**

The capillaries have a **fenestrated endothelium** and a **basement membrane** that allows filtration while keeping red blood cells, proteins, and many macromolecules out of the ultrafiltrate.

It is surrounded by a single layer of epithelial cells (**podocytes**) responsible for the filtration barrier

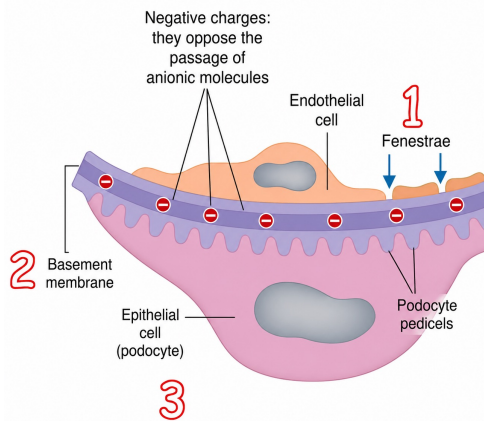
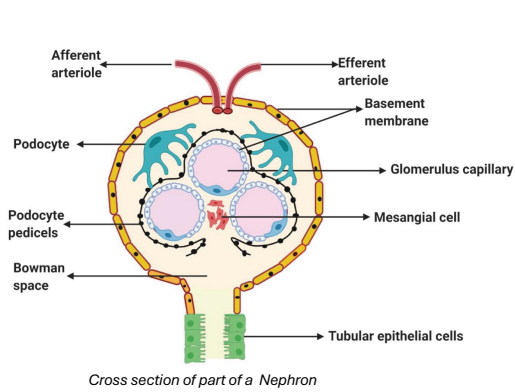


GLOMERULAR FILTRATION BARRIER

Fenestrated endothelium:
allows water and salts to pass through

Basal membrane
Rich in collagen, proteoglycans negatively charged

Podocytes
Cells with extensions called foot processes (pedicels)



Cr

Between one foot process and another, there are small gaps called:

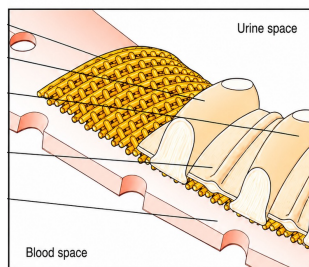
slit pores / filtration slits

These slits are closed by a thin protein membrane that acts as a final sieve:

slit diaphragm

Therefore, the filtrate:

- does NOT enter the podocytes,
- passes through the space between the foot processes.



Filtration occurs according to:

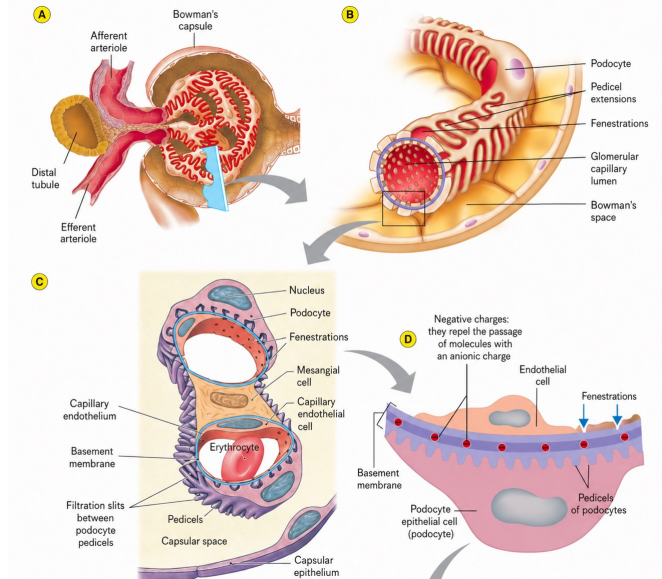
- **Size**
- **Shape**
- **charge**

The basement membrane and the podocytes are negatively charged; therefore, proteins are not filtered.

The filtrate is collected in Bowman's space and then conveyed into the renal tubule.

- The filtrate contains water, ions, glucose, urea, and many other small molecules with a radius smaller than about 2 nm or a molecular weight lower than 6–7 kDa.
- Almost all proteins are excluded.

Microscopic anatomical details of the glomerular filtration barrier



Dettagli anatomici microscopici della barriera di filtrazione glomerulare

The renal tubule

The renal tubule is the structure through which the glomerular filtrate flows.

Structurally, the renal tubule can be divided into:

proximal convoluted tubule, loop of Hente:

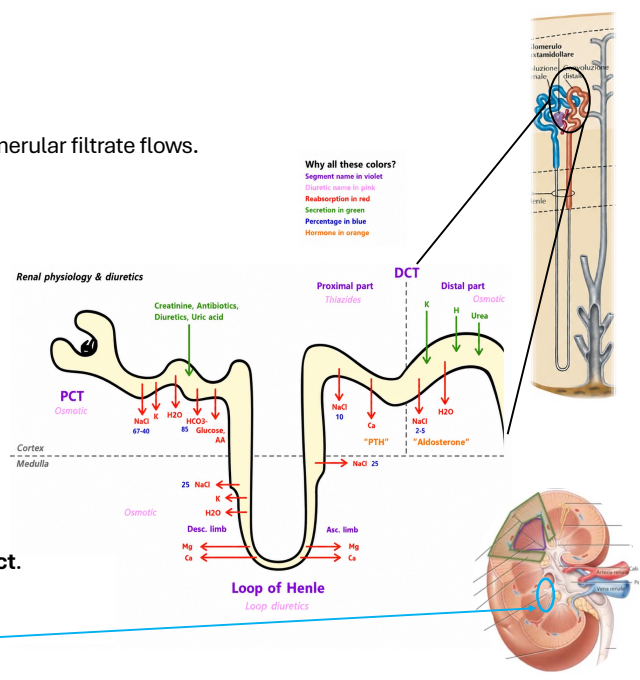
- thin descending limb,
- loop proper,
- thin ascending limb,
- thick ascending limb,

distal convoluted tubule

The renal tubule of a nephron ends in the **collecting duct**.

Several tubules empty into the same duct.

The duct opens at the **renal papilla**.



The glomerulus

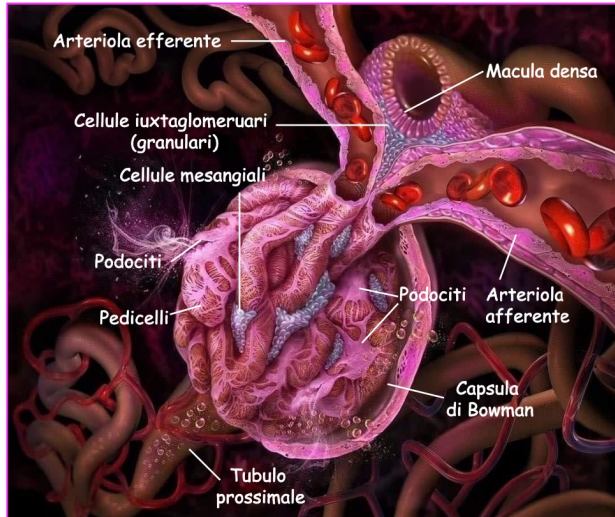


Immagine ricostruita che dettaglia la struttura del glomerulo

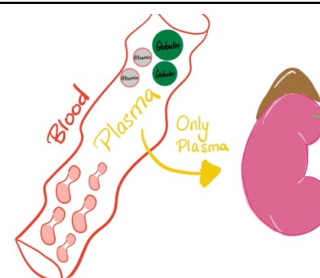
1. Glomerular filtration

The kidneys receive about 20% of the cardiac output.
 Average cardiac output:
 ~5 L/min
 Blood reaching the kidneys:
 ~1 L/min

Only the plasma is filtered.
 Plasma reaching the kidneys
 55% of 1 L/min:
 ~550 mL/min of plasma

Not all the plasma is filtered.
 Opposing forces:
 1. **Blood pressure** pushing out the liquid
 2. **Plasma proteins** (retaining water in the capillars) and the **filtration barrier** (endothelium, basement membrane, podocytes) opposing resistance.

⇒ 1/5 out of the plasma = 20%



Remember that blood contains:

Cellular components (45%)
 erythrocytes (red blood cells)
 leukocytes (white blood cells)
 platelets
Liquid component: Plasma (55%)
 H₂O (90%)
 plasma proteins
 hormones
 nutrients
 waste products (urea and creatinine)
 dissolved gases
 electrolytes

Let us now see how the kidney uses this functional unit to carry out its functions.

Each time the heart contracts and pumps blood into the systemic circulation, about 20% of the blood — one fifth — reaches the kidneys.

If you remember, the body's cardiac output is typically around 5 liters per minute. Twenty percent of this corresponds to approximately 1 liter of blood per minute reaching the kidneys.

However, the kidneys filter only one portion of the blood: the plasma.

Remember that blood is composed of:

a cellular portion (45%):

erythrocytes,
 leukocytes,
 platelets;

a liquid portion, called plasma (55%):

water (H₂O, about 90%),
 plasma proteins,
 hormones,
 nutrients,
 waste products (such as urea and creatinine),
 dissolved gases,
 electrolytes.

Therefore, 55% of 1 liter per minute corresponds to approximately 550 mL of plasma per minute.

However, at the level of the glomerulus there are physical forces that oppose filtration.

On one side:

blood pressure pushes fluid outward.

On the other side:

proteins in the blood pull water inward (they retain water within the capillaries by osmosis),
 and the filtration barrier (endothelium + basement membrane + podocytes) provides resistance.

An equilibrium is therefore established, preventing the glomerulus from completely emptying the plasma.

Only a fraction of the plasma is filtered (about 1/5 = 20%).

As a result, approximately 100–120 milliliters of plasma per minute are filtered through this filtration membrane. This quantity defines the glomerular filtration rate (GFR).

Glomerular Filtration Rate GFR

- Homeostatic variable that must be kept constant.
- It represents a measure of how well we are filtering the blood.

100–120 milliliters per minute

Why does the filtrate leave the glomerulus?

Forces involved

1. Capillary hydrostatic pressure

(+55 mmHg)

It is the main force.

Blood pushes outward.

It FAVORS filtration.

2. Colloid osmotic pressure

(-30 mmHg)

Plasma proteins retain water inside the capillary.

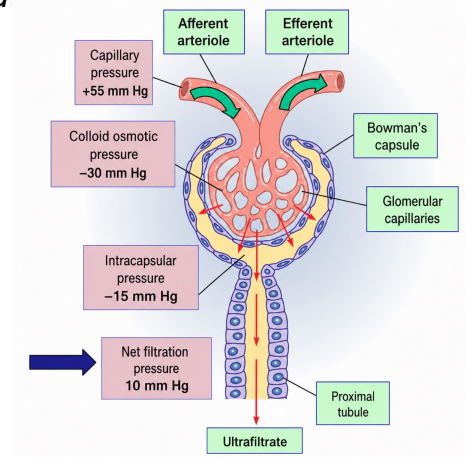
It OPPOSES filtration.

3. Intracapsular pressure

(-15 mmHg)

The fluid already present in Bowman's capsule creates resistance.

It OPPOSES filtration.



FINAL RESULT

$$55 - 30 - 15 = 10 \text{ mmHg}$$

Net filtration pressure

The filtration depends on three factors

Pressure difference

Between:
the glomerular capillary,
Bowman's capsule.

Colloid osmotic pressure

Proteins draw water back into the blood.

Hydraulic permeability

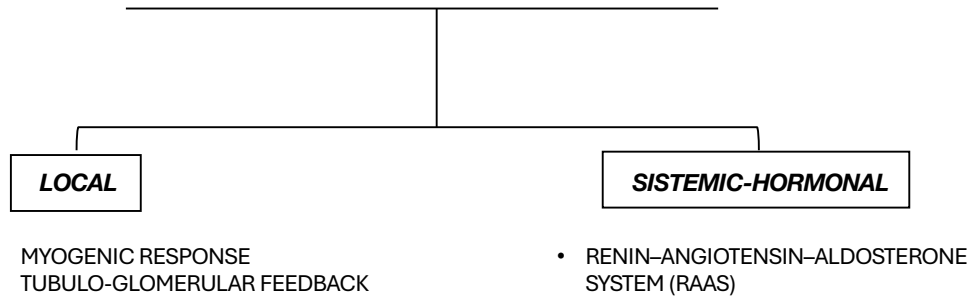
How easily the filter allows water to pass through.
It depends on:

the endothelium,
the basement membrane,
the podocytes.



Glomerular Filtration Barrier

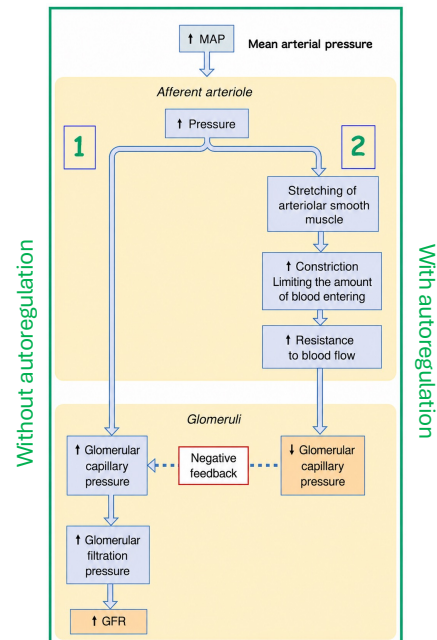
GFR regulatory mechanisms



Glomerular filtration is regulated both by local autoregulatory mechanisms, such as the myogenic response and tubuloglomerular feedback, and by systemic hormonal systems such as the RAAS (Renin–Angiotensin–Aldosterone System).

Myogenic response

A negative feedback mechanism that protects the glomerulus from excessive changes in blood pressure.



Let us now see what would happen if the kidney did not possess autoregulatory mechanisms.

When arterial pressure (more precisely, mean arterial pressure) increases, the pressure of the blood reaching the afferent arteriole also increases. If no control system existed, this would directly increase the pressure inside the glomerular capillaries.

The increase in glomerular capillary pressure would also increase the filtration pressure, leading to an increase in the glomerular filtration rate (GFR).

In other words, every increase in systemic arterial pressure would automatically cause an increase in renal filtration, which would be harmful to the glomerulus.

To prevent this, the kidney possesses an autoregulatory mechanism called the myogenic response.

When pressure increases, the wall of the afferent arteriole is stretched. The smooth muscle cells in the vessel wall detect this stretch through mechanosensitive channels located in the cell membrane.

Activation of these channels promotes calcium entry into the smooth muscle cells. The increase in intracellular calcium induces contraction of the smooth muscle of the afferent arteriole.

The result is vasoconstriction of the afferent arteriole, which increases resistance to blood flow and limits the amount of blood entering the glomerulus.

In this way, glomerular capillary pressure returns toward normal values, and the glomerular filtration rate remains relatively stable.

This is therefore a negative feedback mechanism that protects the glomerulus from excessive changes in arterial pressure.

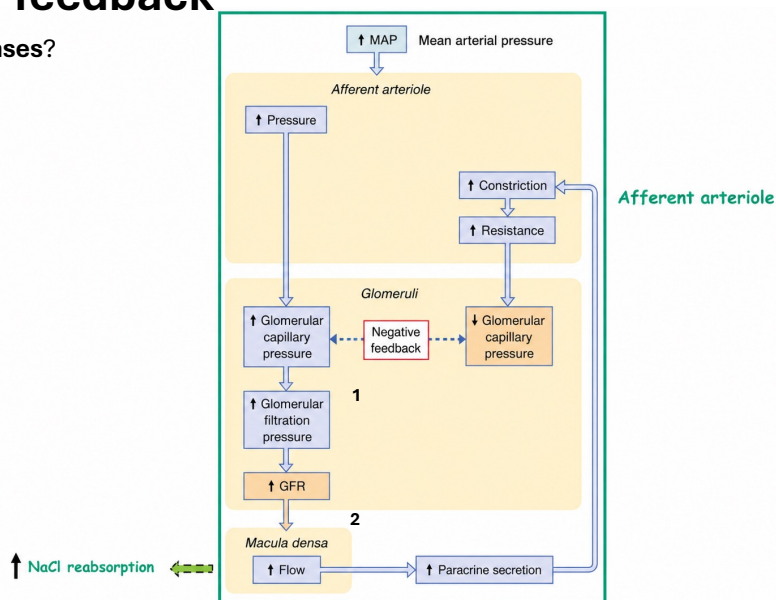
Curiosity

Why do we speak about mean arterial pressure?

Because blood flow through tissues — including the kidney — depends on the average pressure exerted throughout the entire cardiac cycle.

Tubulo-glomerular feedback

What happens when the GFR increases?



A control system that works in coordination with the myogenic response is the tubuloglomerular feedback mechanism. Its purpose is also to maintain a stable GFR.

Let us see what happens when the glomerular filtration rate increases.

An increase in mean arterial pressure causes an increase in pressure within the glomerular capillaries and therefore an increase in GFR.

If the GFR increases, the filtrate flows more rapidly through the tubule, and a greater amount of sodium reaches the macula densa.

The macula densa uses the NKCC2 transporter as a sensor of the amount of NaCl present in the tubular filtrate.

When GFR increases, more NaCl reaches the macula densa, and therefore a greater amount of sodium enters the cell through NKCC2. This ion influx modifies the electrical activity of the cell and increases its metabolic activity.

The more sodium enters the cell, the harder the cell must work to pump it back out through the Na⁺/K⁺ ATPase pump, a process that requires ATP consumption and turnover.

The increased metabolic activity therefore leads to greater extracellular release of ATP, which can then be converted into adenosine.

The macula densa interprets the elevated NaCl concentration as a signal of excessive glomerular filtration and releases these local paracrine signals (ATP and adenosine), which induce vasoconstriction of the afferent arteriole and reduction of the GFR.

ATP and adenosine bind to specific receptors on the membrane of smooth muscle cells (A1 receptors), causing Ca²⁺ influx and subsequent contraction.

This constriction increases resistance to blood flow, reduces the amount of blood entering the glomerulus, and restores glomerular capillary pressure and GFR toward normal values.

This too is therefore a negative feedback mechanism.

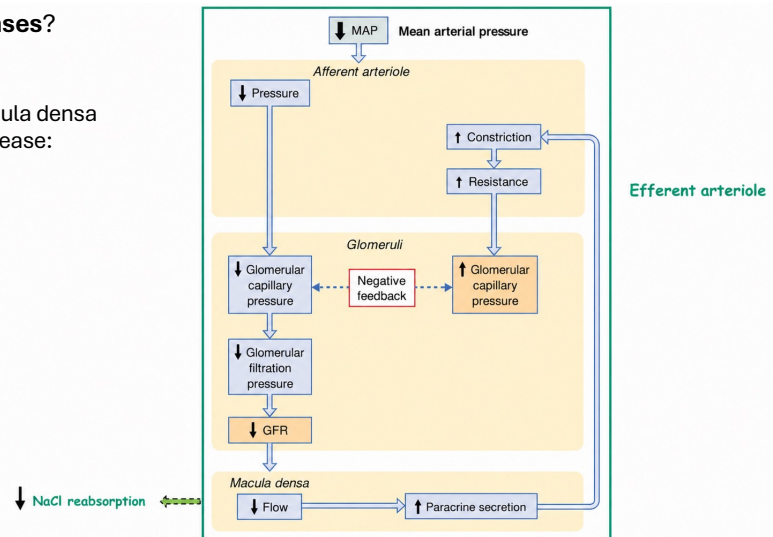
Tubulo-glomerular feedback

What happens when the GFR **decreases**?

Activation of two key enzymes in the macula densa cells such as **COX-2** and **nNOS** which release:

Paracrine mediators:

- **Nitric oxide**
- **Prostaglandins**



What happens when the GFR decreases?

The macula densa senses a low sodium concentration. A reduced NaCl entry corresponds to a reduced Cl⁻ entry, which serves as a signal for the activation of two key enzymes in the macula densa cells:

COX-2 (cyclooxygenase-2), the enzyme responsible for prostaglandin production;
 nNOS (neuronal nitric oxide synthase), the enzyme that synthesizes nitric oxide (NO).
 These are precisely the paracrine mediators that are released.

Nitric oxide induces vasodilation by diffusing into vascular smooth muscle cells, where it activates guanylate cyclase, promoting the formation of intracellular cGMP. cGMP reduces the calcium available for contraction and causes relaxation of the vessel wall.

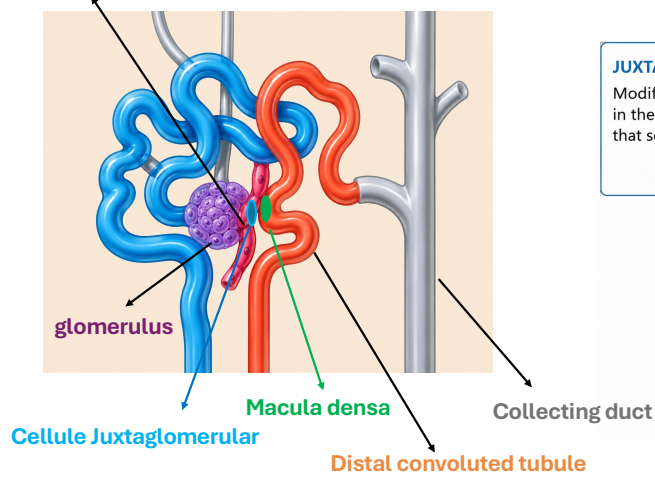
In fact, cGMP inhibits actin-myosin interaction by activating a protein called PKG (protein kinase G). PKG:
 promotes the removal of Ca²⁺ from the cytoplasm;
 reduces Ca²⁺ entry into the cell;
 activates myosin phosphatase.

Prostaglandins stimulate the juxtaglomerular cells, promoting renin release and consequently activating the renin-angiotensin-aldosterone system (RAAS).

Renin–Angiotensin–Aldosterone System (RAAS)

Juxtaglomerular apparatus

Arterioles (afferent and efferent)



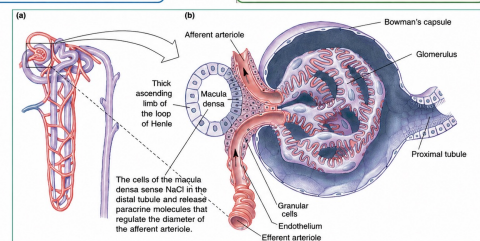
Specialized structure formed by:

JUXTAGLOMERULAR CELLS

Modified smooth muscle cells in the afferent arteriole that secrete **renin**

MACULA DENSA

A group of specialized epithelial cells in the distal tubule that detects NaCl concentration in the filtrate



The juxtaglomerular apparatus is responsible for the RAAS.

It is a specialized structure composed of:

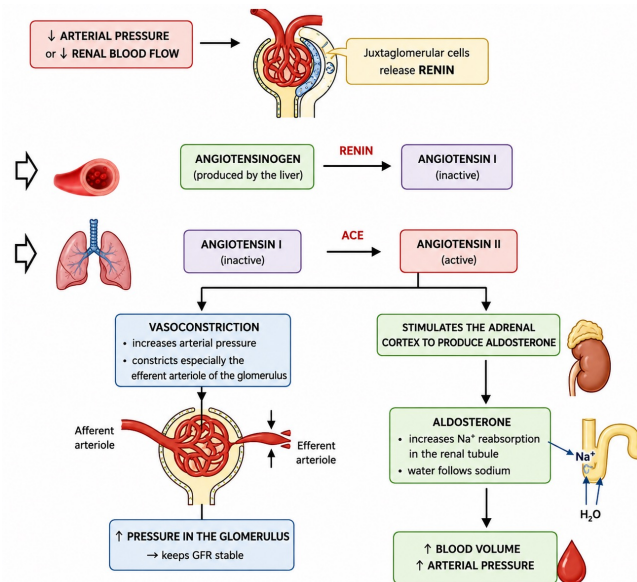
Juxtaglomerular cells (or granular cells)

A group of specialized smooth muscle cells located in the afferent arteriole.

Macula densa

A group of specialized epithelial cells of the distal convoluted tubule.

Renin–Angiotensin–Aldosterone System (RAAS)



When arterial pressure or blood flow to the kidney decreases, the juxtaglomerular cells release an enzyme called renin. Renin enters the bloodstream and acts on a protein produced by the liver, called angiotensinogen, converting it into angiotensin I.

Subsequently, mainly in the lungs, the enzyme ACE converts angiotensin I into angiotensin II, which is the active molecule of the system.

Angiotensin II causes vasoconstriction, increasing arterial pressure, and in particular constricts the efferent arteriole of the glomerulus. In this way, it increases pressure within the glomerulus and helps maintain a stable glomerular filtration rate.

In addition, angiotensin II stimulates the adrenal gland to produce aldosterone. Aldosterone increases sodium reabsorption in the renal tubule; water follows sodium, leading to an increase in blood volume and arterial pressure. Overall, the renin–angiotensin–aldosterone system is a compensatory mechanism that allows the kidney to maintain a stable GFR even when blood pressure or blood volume decreases.

Explanation of why angiotensin II acts mainly on the efferent arteriole

Imagine a water-filled tube.

If you tighten the outlet:

water accumulates,

pressure inside the tube increases.

This is exactly what happens in the glomerulus.

Physiological consequence

Constriction of the efferent arteriole:

slows blood outflow from the glomerulus,

increases glomerular hydrostatic pressure,

maintains filtration,

even when less blood reaches the kidney.

Why does it not mainly constrict the afferent arteriole?

If it strongly constricted the afferent arteriole:

less blood would enter the glomerulus,

glomerular pressure would decrease,

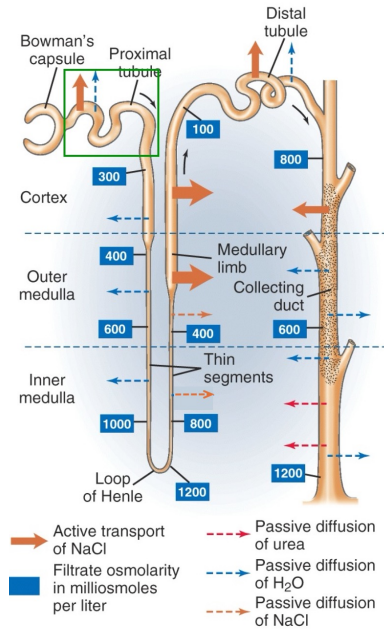
GFR would worsen.

Therefore, it would be counterproductive.

How does angiotensin II preferentially act on the efferent arteriole?

Because the efferent arteriole is more sensitive to angiotensin II than the afferent arteriole, since it expresses a greater number of functionally active AT1 receptors — the receptors that “sense” angiotensin II.

Tubular reabsorption Transport systems



Water, sodium, glucose, amino acids, and many other useful substances enter the filtrate.

The role of the renal tubule is therefore to recover what the body needs and leave in the urine what must be eliminated.

To accomplish this, different transport systems are involved.

Reabsorption involves transepithelial transport.

It can be either active or passive.

The concentration gradient of a molecule determines the mode of reabsorption:

passive diffusion through ion channels and/or transporters (movement along the concentration gradient);

primary and secondary active transport (movement against the concentration gradient).

1. Proximal tubule

In the proximal tubule, most of the glomerular filtrate is reabsorbed.
It is the hardest-working segment of the nephron.

Huge amounts of the following are reabsorbed here:

- sodium: 65–70%
- water: 65–70%
- glucose: almost 100%
- amino acids: almost 100%
- bicarbonate: most of it.

Type of transport

Active and passive.

The initial driving force is the active transport of sodium.

Primary active transport

The Na^+/K^+ ATPase on the basolateral membrane (interstitial side) transports:

Na^+ out of the cell,
 K^+ into the cell,
 consuming ATP.

Inside the cell:
 Na^+ remains at a low concentration.
 This creates a gradient:
 sodium tends to enter the cell from the tubular lumen.

Secondary active transport

Sodium carries other substances along with it.

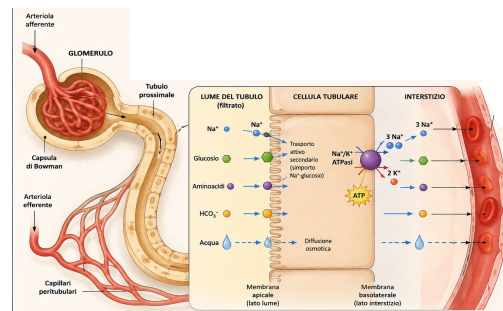
Na^+ enters together with:

glucose,
 amino acids,
 other solutes.

These substances enter through the symport mechanism with Na^+ (e.g., Sodium-Glucose Linked Transporter, SGLT).

Water follows osmotically

When sodium is reabsorbed:
 interstitial osmolarity increases,
 water follows passively.
 This is passive transport.



But is transport passive or active?

The correct answer is:

both.

However:

the initial driving force is the active transport of sodium.

Simple mechanism explanation**1. The Na^+/K^+ ATPase**

(located on the basolateral membrane)

transports:

Na^+ out of the cell,

K^+ into the cell,

while consuming ATP.

This is primary active transport.

2. Consequence

Inside the cell:

Na^+ concentration remains low.

This creates a gradient:

sodium tends to enter the cell from the tubular lumen.

3. Sodium carries other substances with it

Na^+ enters together with:

glucose,

amino acids,

other solutes.

This is secondary active transport.

Because:

it indirectly uses the energy generated by the Na^+/K^+ ATPase.

4. Water follows osmotically

When sodium is reabsorbed:

interstitial osmolarity increases,
water follows passively.
This is passive transport.
Therefore, bulk reabsorption is:
active for sodium,
secondarily active for glucose and amino acids,
passive for water and some ions.

2. Loop of Henle

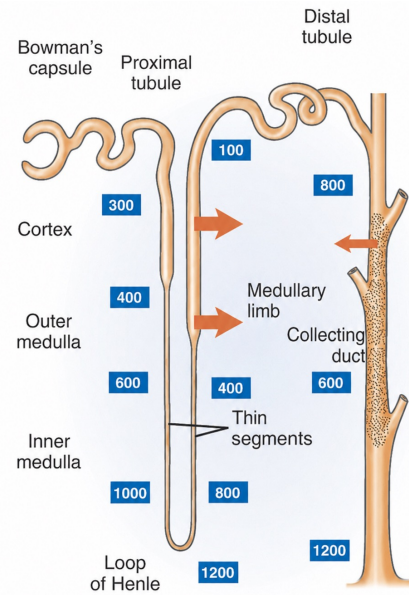
The loop of Henle has different properties in its two limbs.

The thin **descending limb** is permeable to water but only slightly permeable to solutes. H_2O progressively leaves the tubule and moves into the medullary interstitium. About 15% of water is reabsorbed (entering the peritubular capillaries).

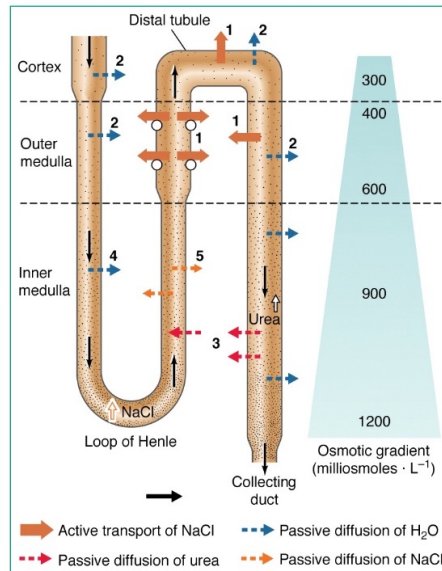
The **ascending limb** is impermeable to water but reabsorbs sodium, potassium, and chloride through the NKCC2 cotransporter. In this way, the filtrate, initially concentrated in the deep part of the loop, becomes progressively more diluted as it ascends toward the cortex. About 25% of sodium, potassium, and chloride is reabsorbed.



Therefore, the loop of Henle creates the medullary osmotic gradient necessary to concentrate the urine.



Corticomedullary osmolarity gradient



We have said that water (H₂O) is reabsorbed at the level of the descending limb of the loop of Henle. Therefore, here water leaves the tubule.

For water to leave the tubule, the external environment must be highly concentrated.

The renal medulla is the ideal environment for this process. In fact, it is characterized by being hyperosmotic.

The concentration of the renal interstitium progressively increases from the cortex toward the inner medulla.

What creates this high osmolarity?

Two substances:

NaCl (in the loop of Henle),

urea (in the medullary portion of the collecting duct).

As regards the loop of Henle, we have two compartments (ascending and descending limbs) that work in opposite but coordinated ways: one supports the function of the other.

Urea derives from protein catabolism, is produced by the liver, and reaches the glomerulus where it is filtered and enters the filtrate.

It then passes through the proximal tubule, travels along the entire loop of Henle, crosses the distal tubule, and finally reaches the collecting duct.

This portion is characterized by the presence of urea receptors/transporters that allow urea to leave the tubule and enter the deepest part of the renal medullary interstitium.

3. Distal tubule

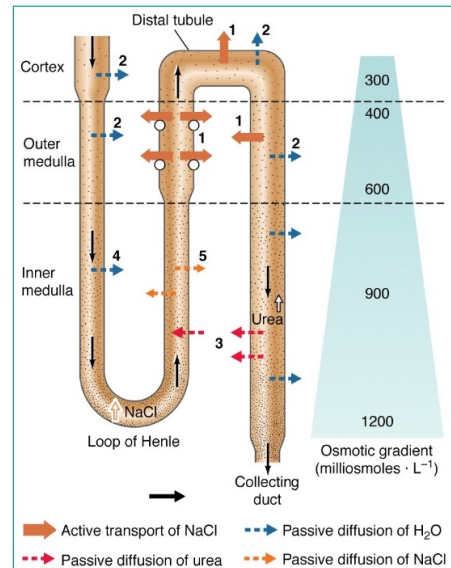
Reabsorbed:
10% of NaCl
a variable amount of water (8–17%)

Control of natremia

Control of kalemia

Importance of K⁺
It controls:

- Cardiac excitability
- Nervous excitability
- Muscular excitability



After passing through the loop of Henle, the filtrate enters the distal tubule, where about 10% of NaCl and a variable amount of water (8–17%) are reabsorbed.

Here, the following processes occur:

1. Regulation of natremia

(the concentration of Na⁺ in body fluids)

The distal tubule:

can increase or decrease Na⁺ reabsorption, depending on the body's needs.

2. Regulation of kalemia

(the concentration of K⁺ in body fluids)

The distal tubule:

regulates K⁺ secretion;

potassium is moved from the blood

→ into the tubule

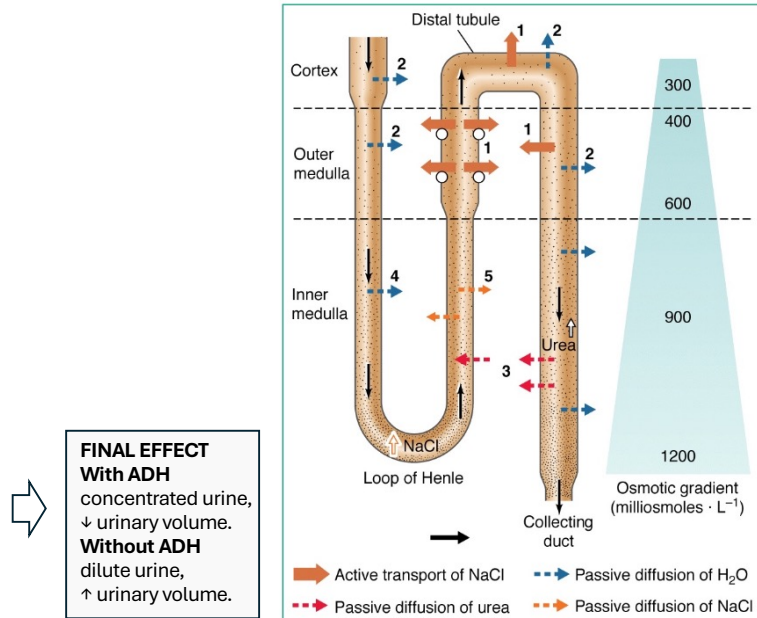
→ and then eliminated in the urine.

K⁺ is important because it controls cardiac, nervous, and muscular excitability.

Natremia and kalemia are hormonally regulated by aldosterone.

Aldosterone (produced by the adrenal gland), being a steroid hormone, crosses the cell membrane and binds to an intracellular receptor. The hormone–receptor complex enters the nucleus and stimulates the transcription of genes that increase the synthesis of ENaC channels and Na⁺/K⁺ ATPase pumps, thereby promoting sodium reabsorption and potassium secretion.

4. Dotto collettore



This is followed by passage into the collecting duct, where the amount of H₂O to be reabsorbed or eliminated is regulated by antidiuretic hormone (ADH).

When the body is dehydrated or plasma osmolarity increases, ADH is released by the posterior pituitary (neurohypophysis) and reaches the kidney through the bloodstream.

ADH acts on the cells of the collecting duct by inducing the insertion of water channels called aquaporins into the cell membrane.

In the presence of aquaporins, the collecting duct becomes permeable to water.

At this point, water can leave the tubule by osmosis because the renal medulla is highly hyperosmotic, thanks to the gradient created by the loop of Henle and the contribution of urea, which in this segment can leave the tubule through the presence of urea transporters (UT).

In the preceding segments, the expression of these transporters is not abundant. This is one of the ways by which the kidney concentrates urea specifically in the deep medulla, where it is necessary to increase osmolarity.

Water therefore passes:

from the lumen of the collecting duct,
 into the medullary interstitium,
 and subsequently into the vasa recta, returning to the bloodstream.

As a consequence:

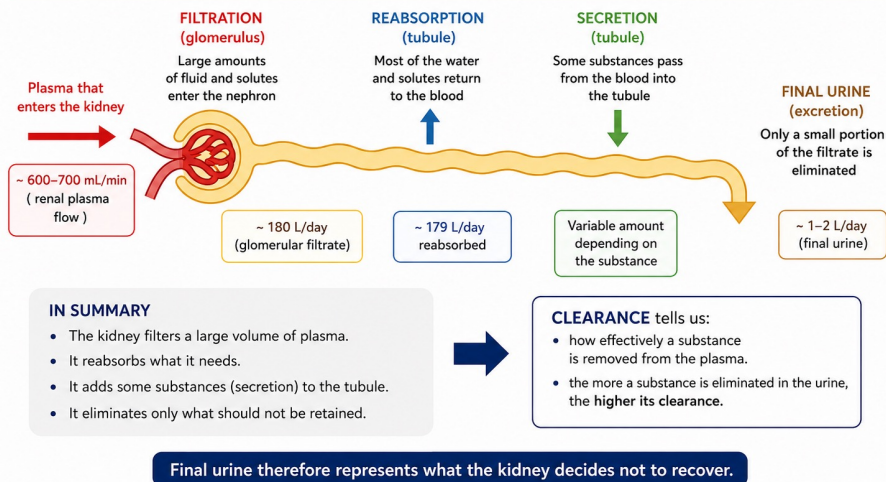
urine volume decreases,
 and urine becomes more concentrated.

Conversely, in the absence of ADH, the collecting duct remains poorly permeable to water. In this case, water is not significantly reabsorbed and is eliminated in the urine, which becomes more abundant and dilute.

We can therefore say that the collecting duct represents the main site for regulation of the body's water balance.

RENAL CLEARANCE – THE CONCEPT

The renal clearance of a substance is the **volume of plasma** that is **completely cleared** of that substance per unit of time.



We have seen that the kidney initially produces a large amount of glomerular filtrate.

This filtrate contains water, sodium, glucose, amino acids, urea, and many other substances present in the plasma. However, the kidney does not eliminate everything it filters.

Along the renal tubule, intense reabsorption takes place:

in the proximal tubule, large amounts of water and useful solutes are recovered;

in the loop of Henle, the medullary osmotic gradient is generated;

in the distal tubule and collecting duct, fine regulation of water and electrolytes occurs under hormonal control.

We have therefore seen that during passage through the nephron, some substances:

are reabsorbed,

others are secreted,

while only a fraction is actually eliminated in the urine.

At this point, an important question arises:

How efficiently is the kidney able to eliminate a given substance from the plasma?

To answer this question, we introduce the concept of renal clearance.

Clearance represents the volume of plasma completely cleared of a substance per unit of time.

In other words, clearance allows us to understand how rapidly the kidney is able to remove a substance from the body.

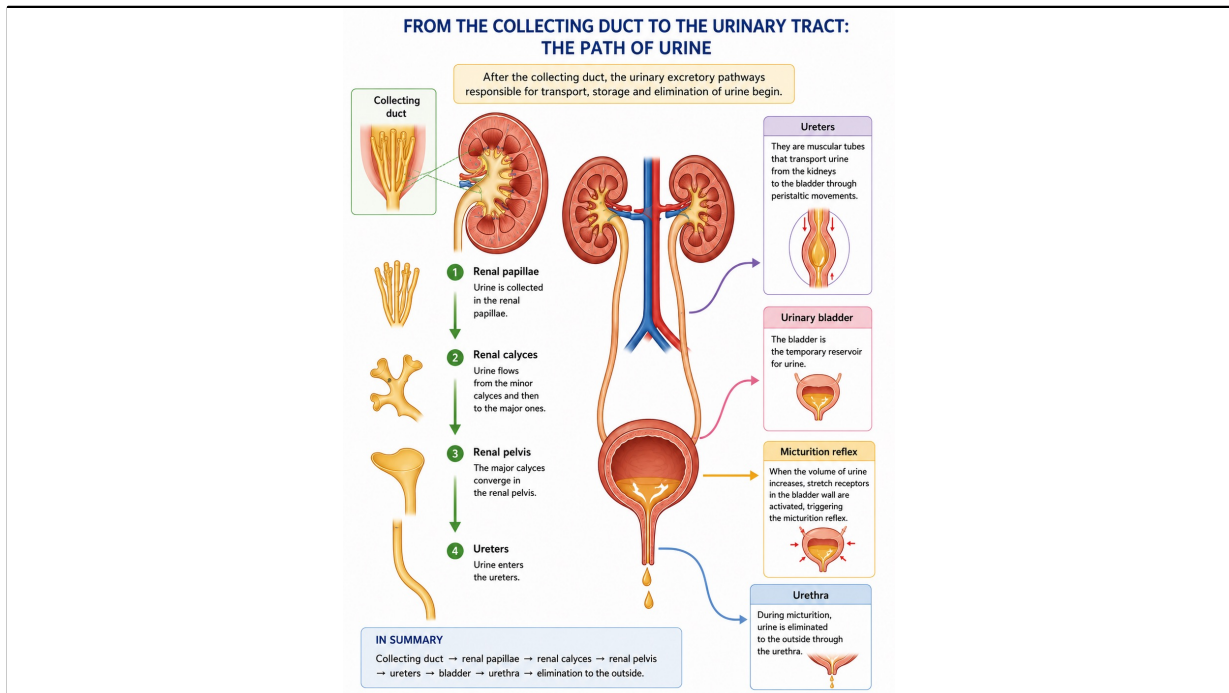
A substance that is highly excreted will have a high clearance, whereas a substance that is extensively reabsorbed will have a lower clearance.

Finally, we can remember that the amount excreted in the urine always depends on the balance between:

the amount filtered,

the amount reabsorbed,

and the amount secreted.



After the collecting duct, the urinary excretory pathways begin; these structures are responsible for the transport, storage, and elimination of urine.

Urine therefore passes through:

the papillary ducts,

the renal calyces,

the renal pelvis,

and subsequently enters the ureters.

The ureters are muscular tubes that transport urine to the bladder through peristaltic movements.

The bladder acts as a temporary reservoir for urine.

When urine volume increases, stretch receptors in the bladder wall activate the micturition reflex.

During micturition, urine is expelled from the body through the urethra.