Cardiovascular System

Diagram of the cardiovascular system

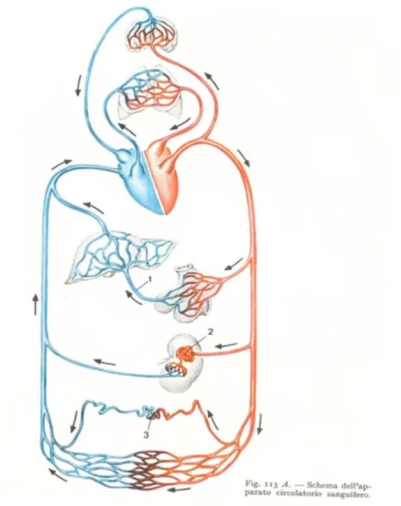


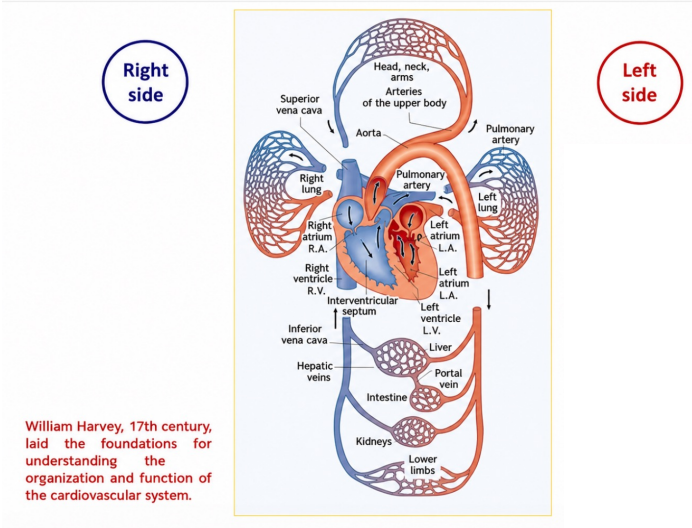
Fig. 113 A. — Schema dell'apparato circolatorio sanguifero.

FUNCTION
Deliver nutrients
Remove waste products from all organs

COMPONENTS
Arteries
Veins
Heart

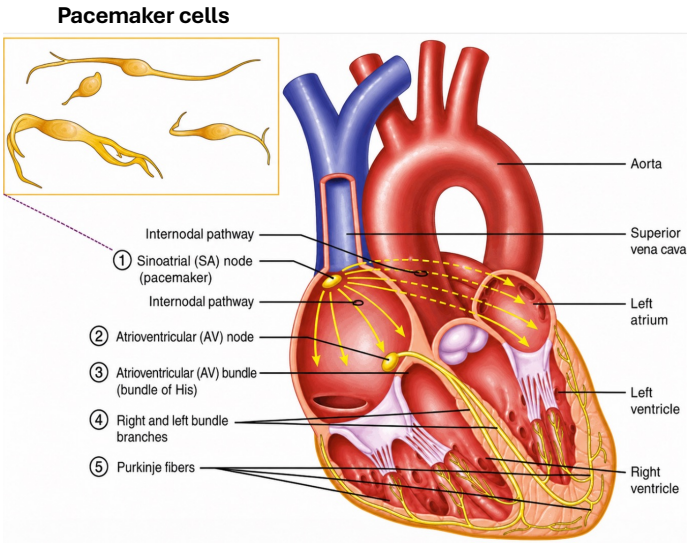
The cardiovascular system is responsible for distributing nutrients and removing waste substances from all organs. It is made up of arteries and veins, which carry blood to a pumping organ: the heart, divided into four chambers.

Cardiovascular system organization



This image represents the general organization of the cardiovascular system, a closed and well-organized system composed of two main circulation circuits, corresponding to the right and left sides.
The right side of the heart receives oxygen-poor blood from the tissues through the venae cavae and pumps it to the lungs via the pulmonary artery. Here, gas exchange occurs: the blood becomes oxygenated and releases carbon dioxide.
The left side of the heart, on the other hand, receives oxygenated blood from the lungs through the pulmonary veins and distributes it throughout the body via the aorta and systemic arteries. This circuit is called systemic circulation.
The image therefore highlights the two circuits:
Pulmonary circulation (heart → lungs → heart)
Systemic circulation (heart → tissues → heart)
From a functional point of view, the cardiovascular system performs several fundamental roles:
Transport of oxygen and nutrients to tissues
Removal of metabolic waste products
Communication between cells through hormones and signaling molecules
Defense of the organism through the transport of immune cells and molecules
It was William Harvey, in the 17th century, who first described blood circulation as a closed and organized system.

Cardiac conduction system



The heart, in order to function properly, requires a conduction system that generates and distributes electrical impulses in an organized way.
This system can be imagined as an internal electrical network. It is made up of specialized structures: the sinoatrial node, the atrioventricular node, the bundle of His, and the Purkinje fibers, which guide the wave of activation from the atria to the ventricles.
The sinoatrial node is the natural pacemaker of the heart. It is a small group of specialized cells located in the right atrium, near the opening of the superior vena cava.
These cells have the ability to spontaneously generate electrical impulses at a rate of about 60–70 per minute at rest, thus establishing the so-called sinus rhythm.
From there, the impulse spreads through the atria and, via the conduction system, reaches the ventricles, producing first atrial contraction and then ventricular contraction in sequence.
The sinoatrial node is autonomous, but not isolated: it is regulated by the sympathetic and parasympathetic nervous systems, which can increase or decrease its firing rate depending on the body's needs (exercise, rest, stress).

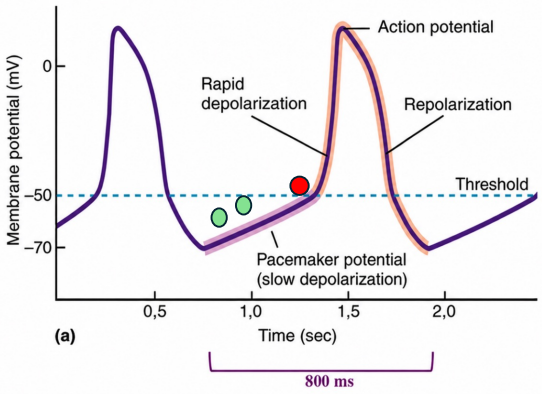
In the cells of the sinoatrial node, the resting potential is not stable

Because always moves toward the threshold

Opening of special ion channels
called If channels or "funny" channels allows positive ions (Na^+) to enter

Progressive closure of potassium channels
K^+ does not leave the cell

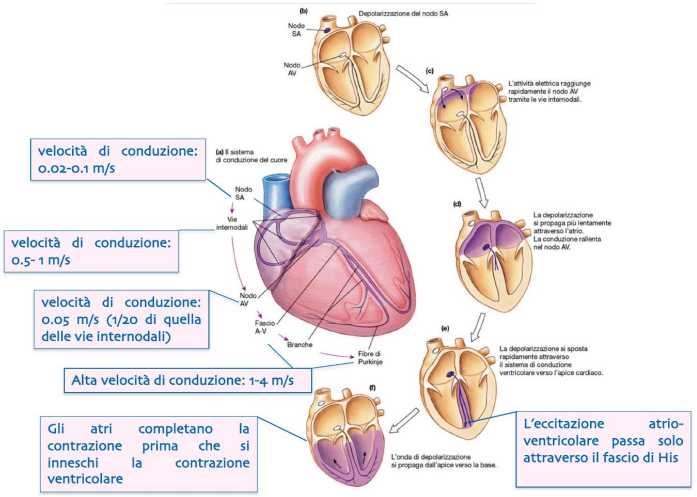
Gradual activation of calcium channels
when the potential has risen sufficiently, **voltage-dependent calcium channels** open, completing the depolarization and triggering a new action potential



The sinoatrial node does not wait for a stimulus: it generates it on its own.

At this point, a natural question is: how do sinoatrial node cells generate electrical impulses on their own? Why doesn't their charge stay stable, but always moves toward the threshold?
The answer is that pacemaker cells are different from normal muscle cells.
In typical muscle fibers, the resting membrane potential is stable: the cell stays "quiet" until it receives a signal from outside.
In sinoatrial node cells, instead, the resting potential is not truly stable. After repolarization, the cells slowly depolarize by themselves.
This slow rise toward the threshold is possible thanks to three main mechanisms working together:
Opening of special ion channels, called If ("funny") channels, which activate when the membrane is very negative and allow positive ions (mainly sodium) to slowly enter
Progressive closing of potassium channels: as these channels close, less K^+ leaves the cell, so the inside becomes less negative
Gradual activation of calcium channels: when the potential rises enough, voltage-dependent calcium channels open, completing depolarization and triggering a new action potential
Together, these processes cause the membrane of pacemaker cells to slowly "drift" toward the threshold, instead of staying still, until a new impulse is generated.
After the impulse, the cell repolarizes and the cycle starts again, creating a continuous and automatic rhythm.
For this reason, the sinoatrial node does not wait for a stimulus: it generates it on its own.

Electrical conduction in the heart



1. **Sinoatrial (SA) node** → activates the atria
The atria contract

2. The impulse reaches the **atrioventricular (AV) node** → there is a short delay (needed to fill the ventricles)

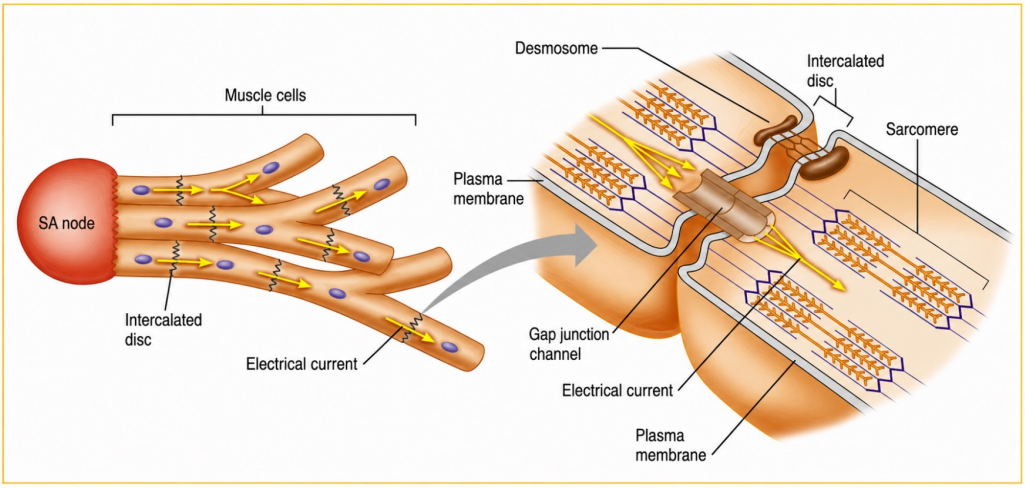
3. Then it passes through:
Bundle of His
Purkinje fibers

4. It reaches the **ventricles** → they contract

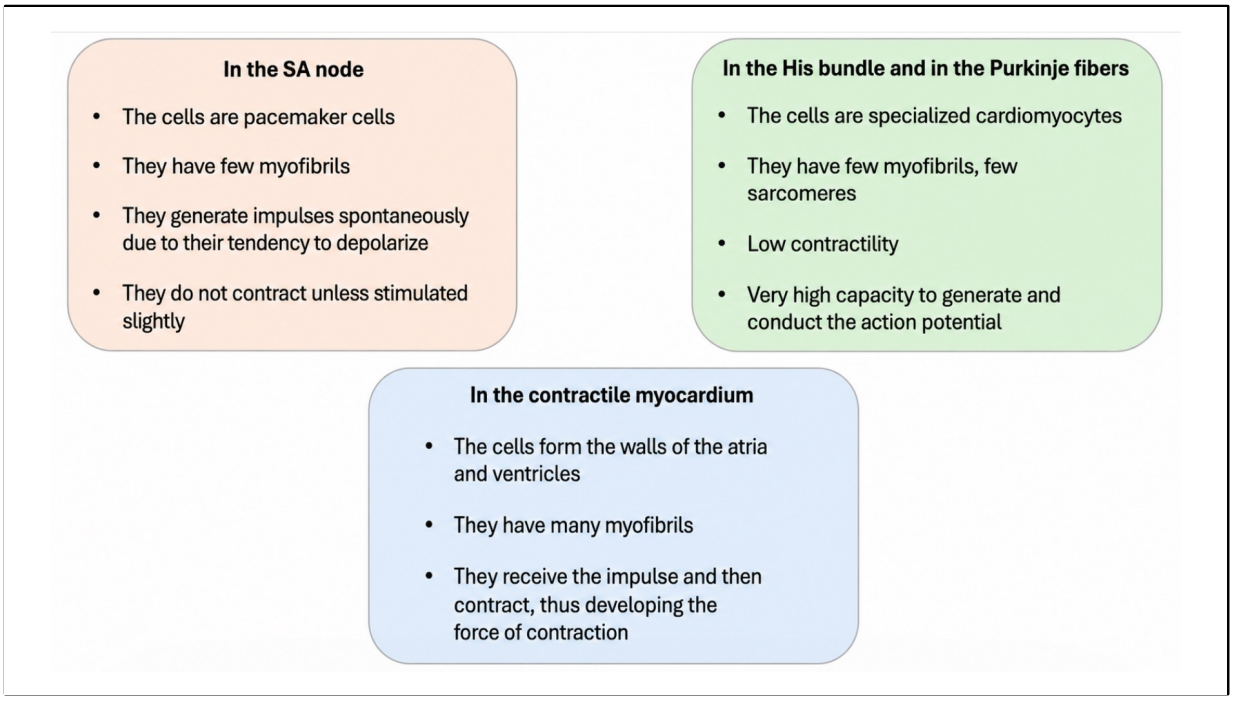
Low contractility
High conductivity

The bundle of His and the Purkinje fibers are regions where cardiomyocytes are specialized for conduction.
These cells have low contractility (they are poor in sarcomeres) but a very high ability to generate and conduct the action potential.
They are different from atrial and ventricular cardiomyocytes, which are specialized for contraction (rich in sarcomeres) and have lower conductivity.

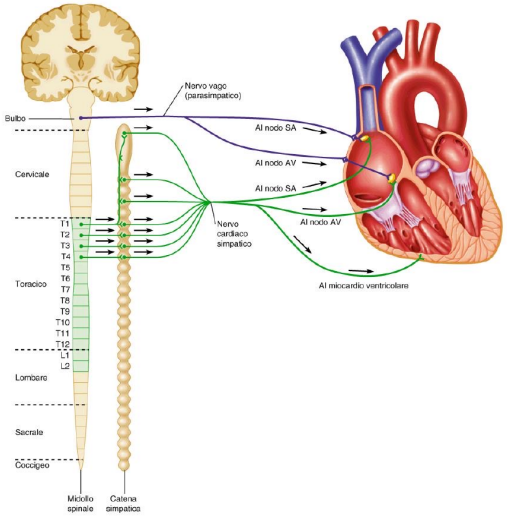
Intercalated discs are regions that allow the passage of the electrical impulse



The ends of the SA node fibers are directly connected to the atrial muscle fibers, called cardiomyocytes.
The impulse therefore spreads from cell to cell in the atrial myocardium thanks to gap junctions, which are true "electrical synapses" between cardiomyocytes.
Myocardial cells are not isolated: at their ends they have specialized contact regions called intercalated discs, which contain gap junctions, that is, pores connecting directly the inside of one cell with the inside of the neighboring cell.
These gap junctions act as electrical synapses: when one cell depolarizes, the ionic current flows through these very low-resistance pores and depolarizes the next cell as well.


In the SA node
The cells are pacemaker cells
They have few myofibrils
They generate impulses spontaneously due to their tendency to depolarize
They do not contract unless stimulated slightly
In the His bundle and in the Purkinje fibers
The cells are specialized cardiomyocytes
They have few myofibrils, few sarcomeres
Low contractility
Very high capacity to generate and conduct the action potential
In the contractile myocardium
The cells form the walls of the atria and ventricles
They have many myofibrils
They receive the impulse and then contract, thus developing the force of contraction

Autonomic control of heart rate



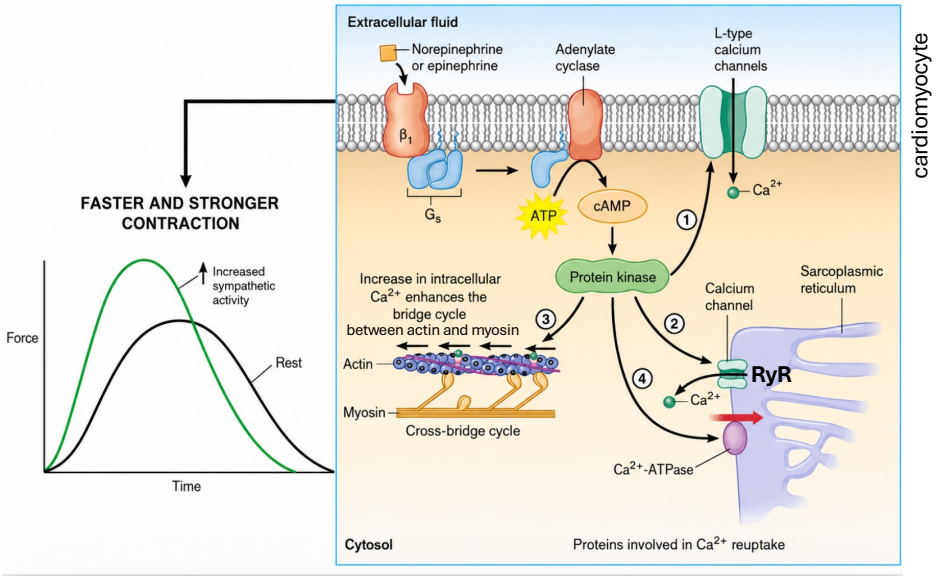
The heart is regulated by the autonomic nervous system.

The parasympathetic system (vagus nerve) slows the heart rate and reduces AV conduction.

The sympathetic system increases the heart rate and speeds up the propagation of the impulse.

The final effect on heart rate depends on the balance between vagal tone and sympathetic tone.

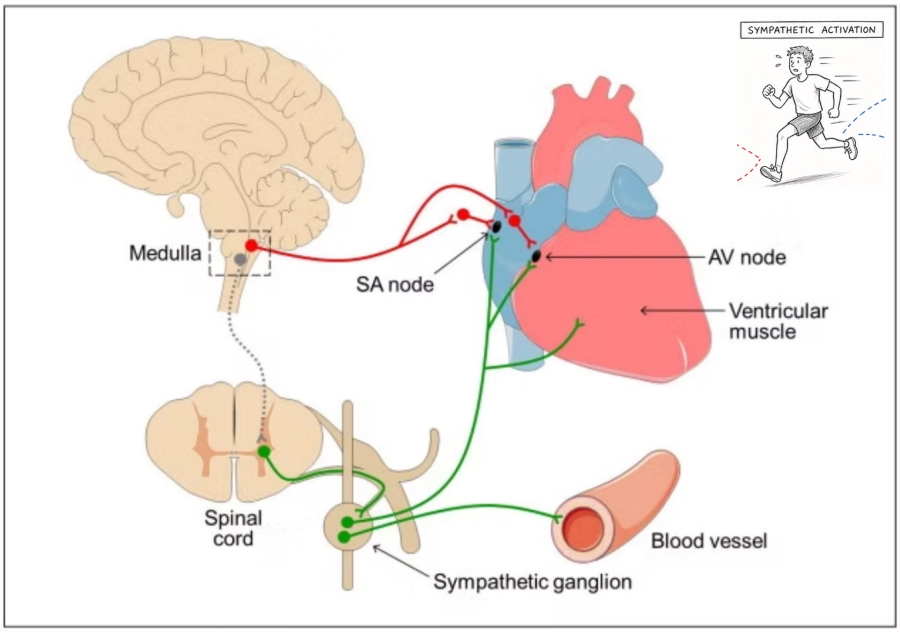
The sympathetic nervous system increases the contraction of the heart



Noradrenaline or adrenaline bind to β_1 receptors on the membrane of the cardiomyocyte, activating a Gs protein, which stimulates adenylyl cyclase.
This leads to the production of cAMP, which activates protein kinase A (PKA).
PKA phosphorylates several targets:

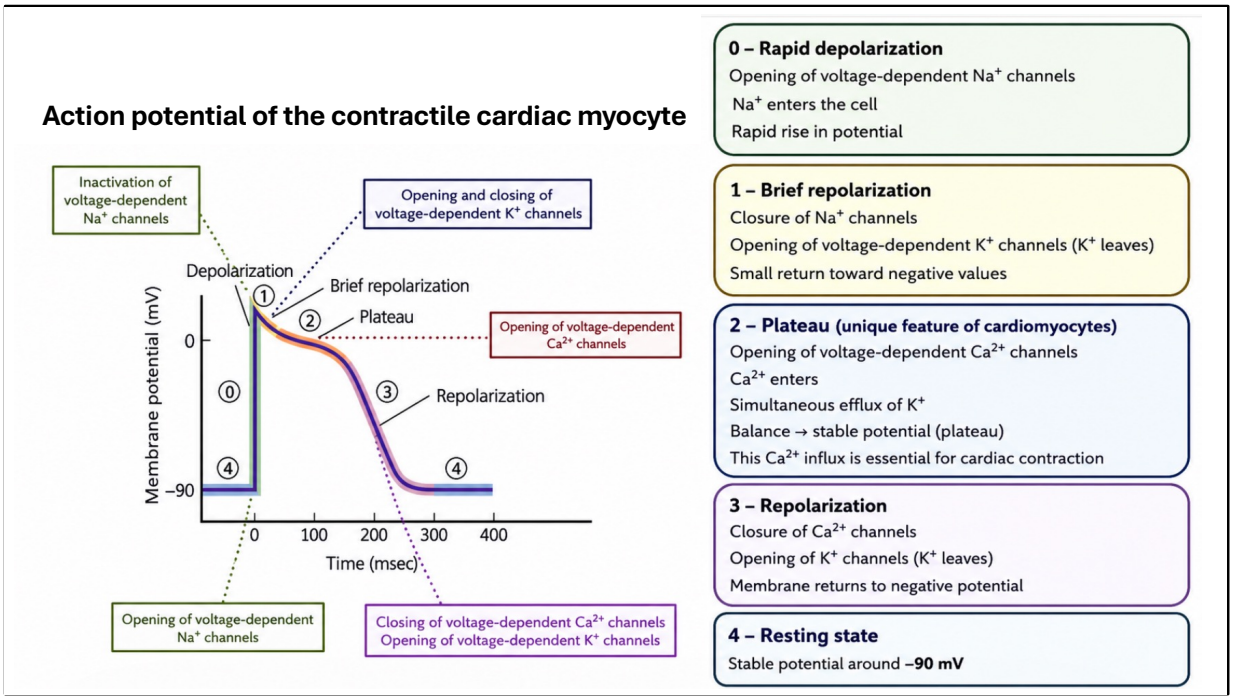
L-type Ca^{2+} channels, increasing Ca^{2+} entry
RyR channels of the sarcoplasmic reticulum, increasing Ca^{2+} release
A series of proteins involved in Ca^{2+} reuptake, including:
- the sarcoplasmic reticulum Ca^{2+}-ATPase (SERCA)
- the Na^+/Ca^{2+} exchanger (NCX)
- the plasma membrane Ca^{2+} pump (PMCA)
- phospholamban (PLB)

The increase in intracellular Ca^{2+} enhances the cross-bridge cycle between actin and myosin, increasing the force of contraction.
In addition, the increase in myosin ATPase activity accelerates the cross-bridge cycle, contributing to a faster and stronger contraction.



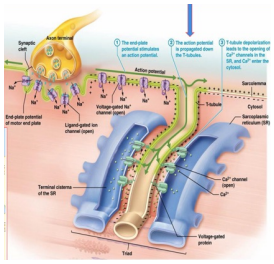
The sympathetic system does not act only on the heart, but also innervates blood vessels. This is important to regulate blood pressure and redistribute blood flow.

What does the sympathetic system do to blood vessels (in general)?
Peripheral vasoconstriction:
Sympathetic stimulation causes contraction of smooth muscle in arteries and especially arterioles of the skin, viscera, and many other regions, increasing peripheral resistance and therefore arterial pressure.
Vasodilation in “priority” areas:
At the same time, vasodilation occurs (directly through β receptors or indirectly by reducing tone) in vessels of skeletal muscles and coronary arteries, so that more blood is directed to organs involved in the “fight or flight” response.
Veins (venomotor tone):
The sympathetic system increases the tone of venous smooth muscle, reduces venous capacity, increases venous return, and therefore increases cardiac output.



Differences in action potential mechanisms between skeletal muscle and cardiac muscle:
Cardiac muscle:
Na^+ enters → depolarization
Ca^{2+} enters → plateau + contraction
K^+ exits → repolarization
Skeletal muscle:
Na^+ enters → depolarization
K^+ exits → repolarization (no plateau)

SKELETAL MUSCLE

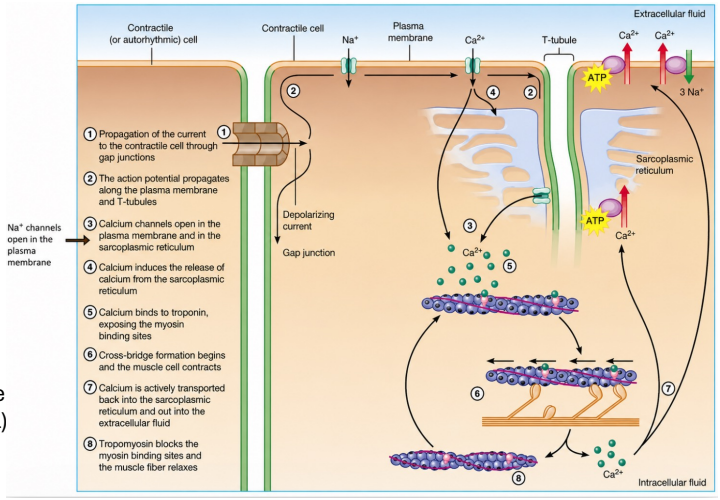


Na^+ enters through Na^+ channels

Voltage-sensitive DHPR (dihydropyridine receptors) interact with RyR (Ca^{2+} channel)

Mechanical coupling

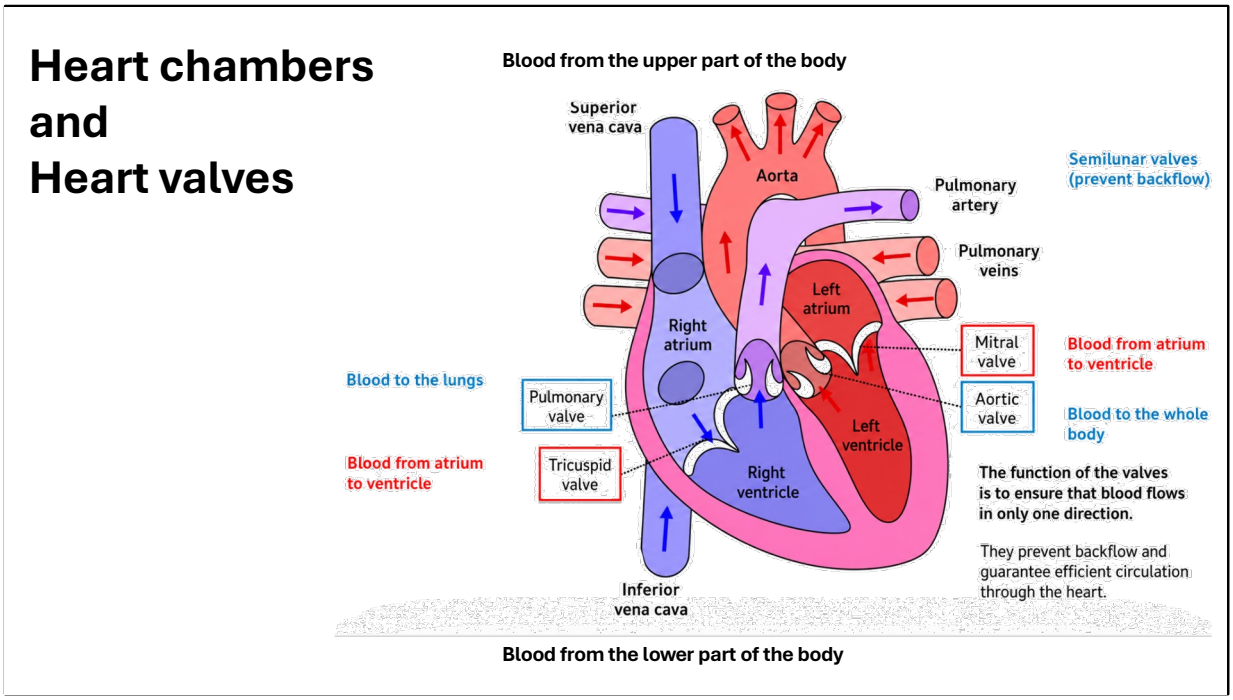
CARDIAC MUSCLE



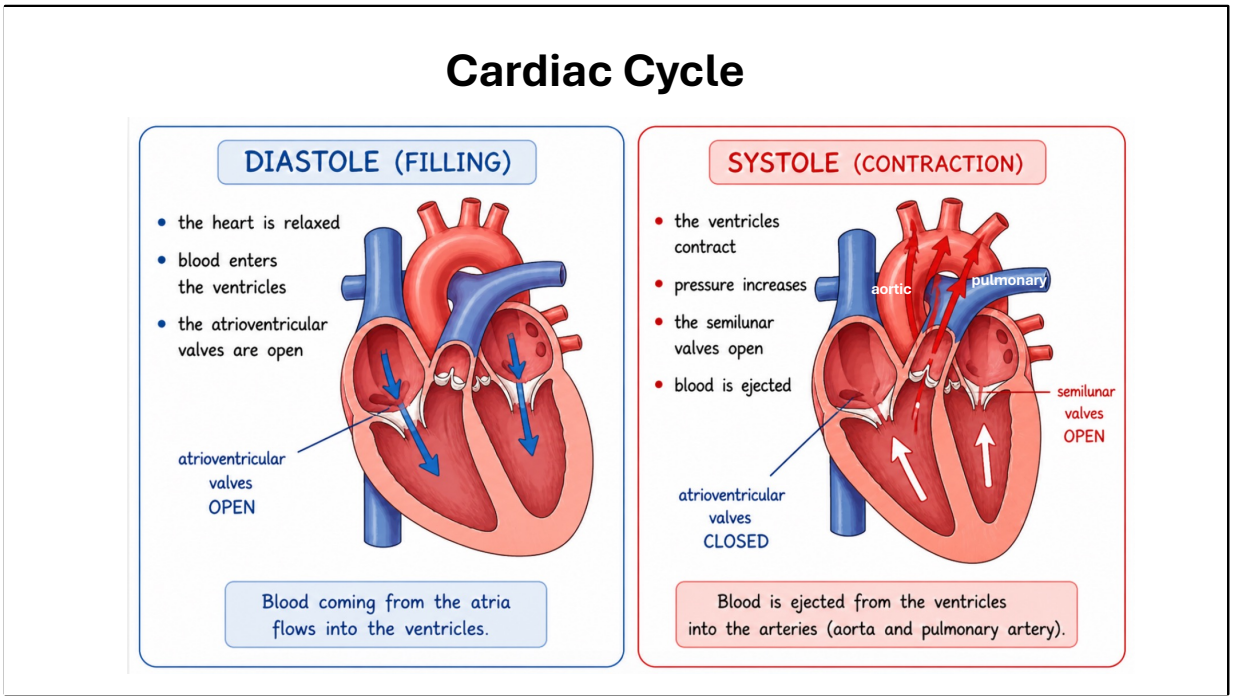
Ca^{2+} from outside enters through **Ca^{2+} channels**
Ca^{2+} directly activates **RyR (Ca^{2+} channels)**

Chemical coupling

Heart chambers and Heart valves



The contraction we have discussed is not an end in itself, but serves to generate pressure inside the heart chambers.
The heart is organized into four chambers:
2 atria (receive blood)
2 ventricles (pump blood)
These allow blood to flow in an orderly way.
Blood moves from one chamber to another following pressure gradients, but to prevent backflow, there are valves, which ensure one-way flow driven by pressure.
Blood enters the right atrium from the venae cavae, passes into the right ventricle through the tricuspid valve, and is pumped to the lungs.
From the lungs, it returns to the left atrium, passes into the left ventricle through the mitral valve, and is finally ejected into the aorta to reach the entire body.

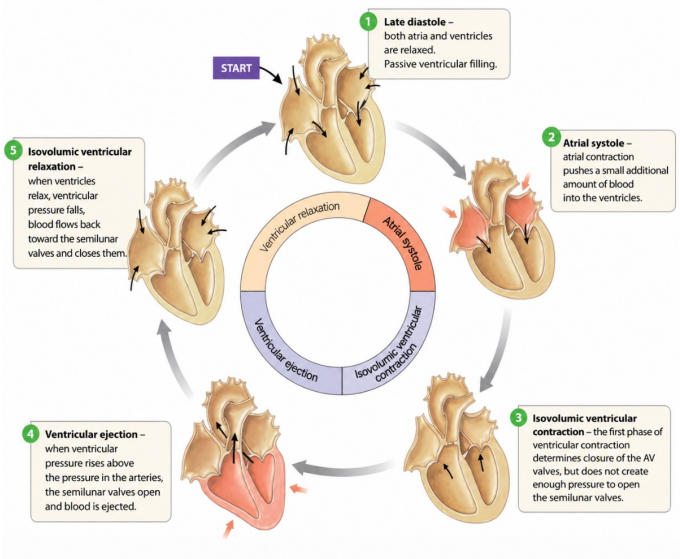


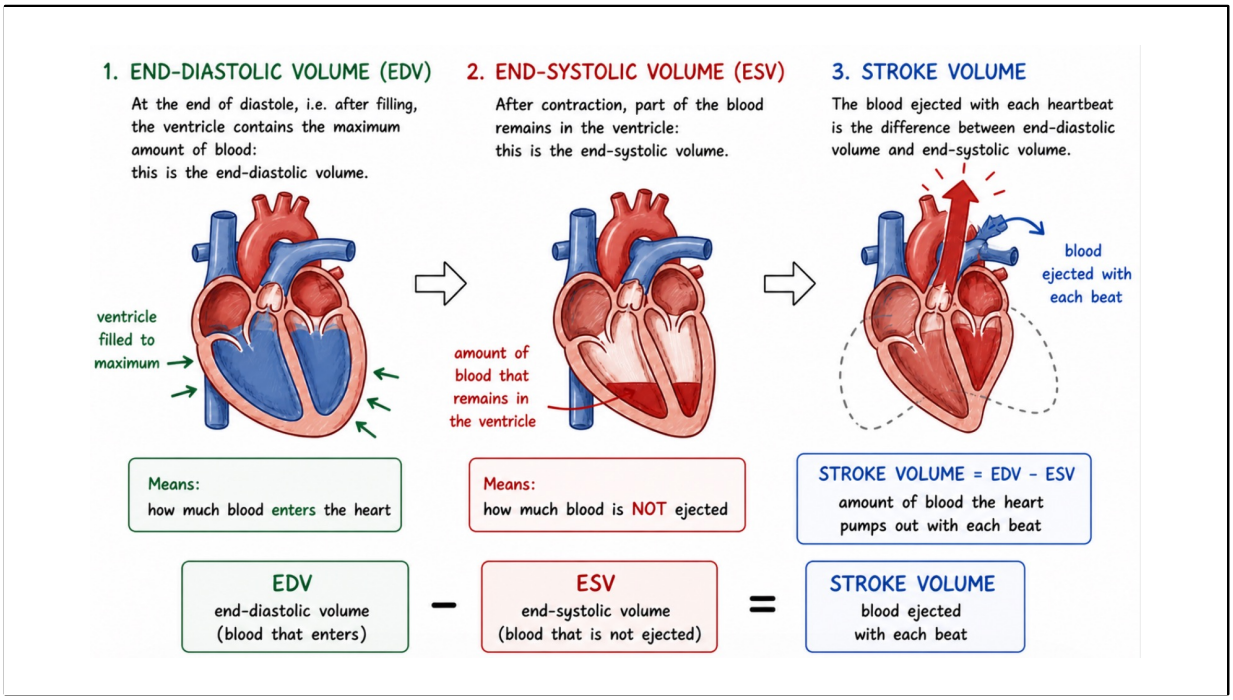
We have seen how blood flows through the heart and how the valves regulate this flow.
Now let's see how these events are organized over time, that is, in the cardiac cycle.
We can roughly distinguish two phases:

Diastole (filling):
the heart is relaxed
blood enters the ventricles
atrioventricular valves are open

Systole (contraction):
the ventricles contract
pressure increases
semilunar valves open
blood is ejected
The heart continuously alternates between a filling phase and an ejection phase, allowing the filling and pumping of blood.

Mechanical events of the cardiac cycle





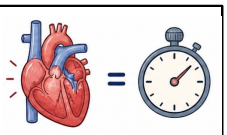
We have described the phases of the cardiac cycle, but to truly understand how the heart works as a pump, we need to quantify how much blood enters and how much is ejected.

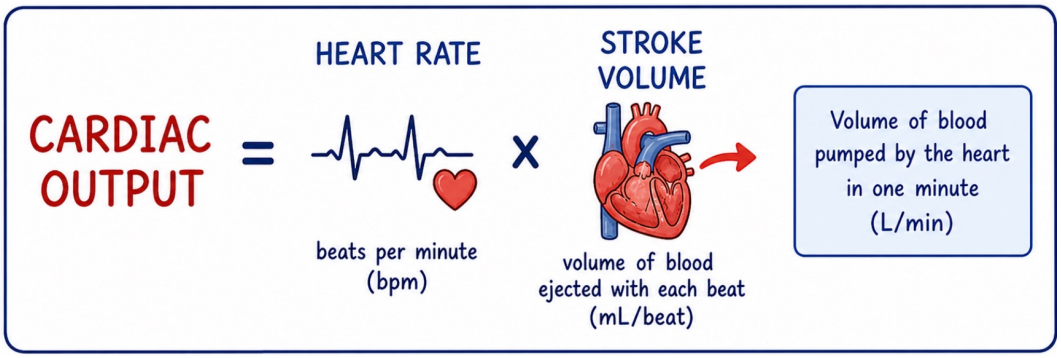
END-DIASTOLIC VOLUME (EDV)
At the end of diastole, after filling, the ventricle contains the maximum volume of blood: this is the end-diastolic volume.
It tells us:
→ how much blood enters the heart

END-SYSTOLIC VOLUME (ESV)
After contraction, some blood remains in the ventricle: this is the end-systolic volume.
It tells us:
→ how much blood is NOT ejected

STROKE VOLUME
The blood ejected by the ventricles at each beat is the difference between EDV and ESV:
SV = EDV - ESV
→ This represents how much blood is pumped out with each heartbeat

Cardiac output





65 beats/min x 70ml/beats
4550 ml/min = 4.5 L/min
Can increase up to 30-35L/min during exercise

At rest, all the blood in the body passes through the heart about once every minute.

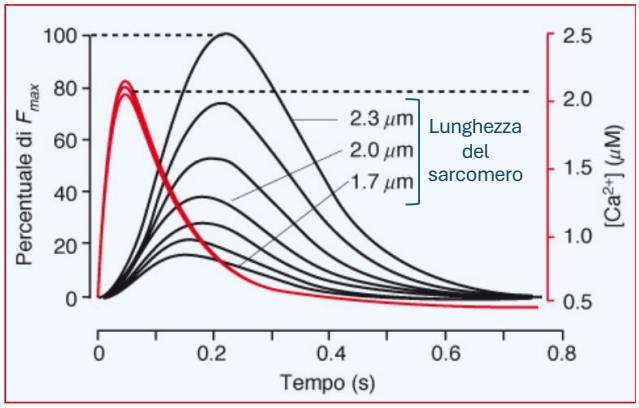
It is possible to quantify stroke volume by considering how frequently it occurs.
To do this, we refer to cardiac output, which is expressed by the following equation:

$$CO = HR \times SV$$

The total volume of blood in the body is approximately 5 liters.

The Starling law

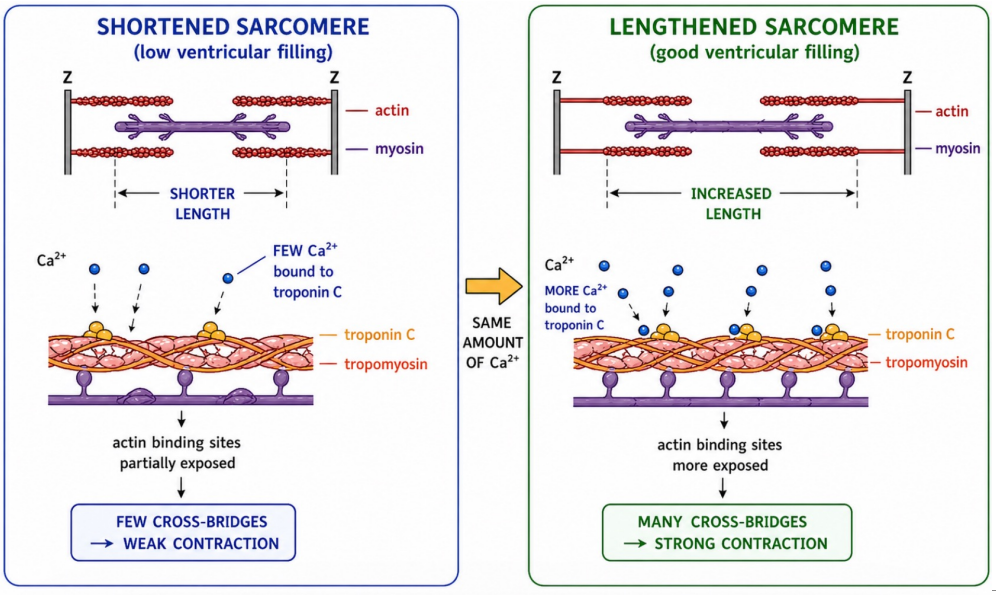
Force–length relationship



What determines the force of heart contraction?
One of the main principles that describes it is the Frank–Starling law.
This law states that:
the more the ventricle fills during diastole, the more the muscle fibers are stretched, and the greater the force of contraction.
In the graph, we can see that force increases with sarcomere length, while the amount of Ca^{2+} remains constant.
So, contraction increases not because more calcium enters, but because the muscle becomes more sensitive to Ca^{2+}.
During physical exercise, ventricular filling and stroke volume can increase up to double. This happens because venous return to the heart increases due to:
muscle contraction, which pushes blood through the veins
breathing, which helps draw blood toward the chest
activation of the sympathetic nervous system, causing venous vasoconstriction
increased heart rate
All these factors increase heart filling and therefore increase the force of contraction.

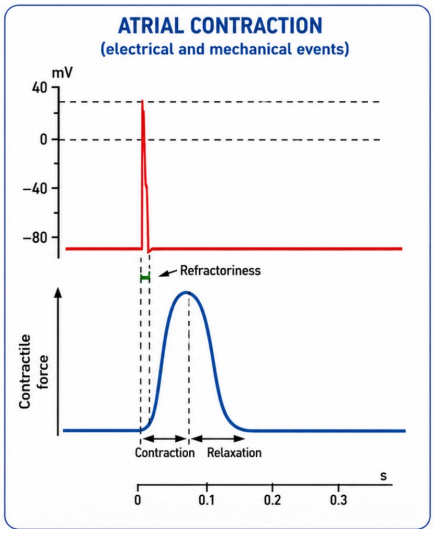
In the graph:
black curves = contraction force increases with sarcomere length
red curve = Ca^{2+} does NOT change

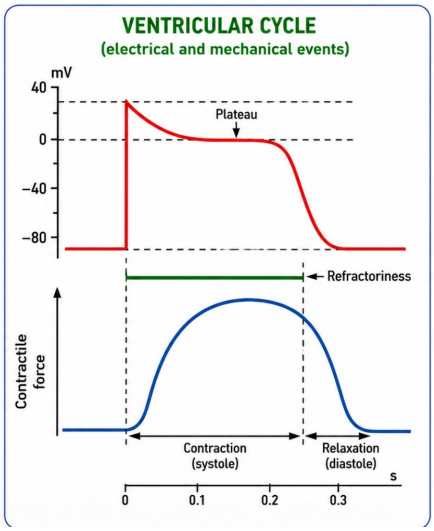
At the same intracellular Ca^{2+} level, the force of contraction increases when the sarcomere is more stretched



At the same intracellular calcium concentration, the force of contraction increases when the sarcomere is more stretched.
In the short sarcomere, calcium binds less effectively to troponin C, the sites on actin are less exposed, and few cross-bridges are formed, resulting in a weak contraction.
In the stretched sarcomere, on the other hand, the affinity of troponin C for calcium increases, more binding sites on actin are exposed, and more cross-bridges are formed, leading to a stronger contraction.
Therefore, increasing sarcomere length improves calcium sensitivity and increases the force of contraction, according to the Frank–Starling law.
The affinity of troponin for calcium changes because:
actin and myosin filaments are aligned more optimally;
steric hindrance decreases;
the structure of troponin C changes. This structural change results from mechanical tension in the sarcomere, which modifies the arrangement of the filaments, the tension on the troponin–tropomyosin complex, and also the conformational structure of troponin C.
As a consequence, in the stretched sarcomere:
troponin C assumes a conformation that (1) stabilizes binding with Ca^{2+} more effectively, and (2) causes calcium to dissociate more slowly;
(3) “calcium sensitivity” increases.
In other words, troponin C binds Ca^{2+} in a thermodynamically more favorable way.

Excitation–contraction coupling





In skeletal muscle, the action potential is very short, while the contraction lasts longer. The refractory period ends before the contraction is finished, so the muscle can be stimulated again while it is still contracted. This allows summation of stimuli and tetanic contraction.
In cardiac muscle, the action potential is long and includes a plateau due to the entry of Ca^{2+}. The contraction occurs during the action potential, and the refractory period is prolonged, almost completely overlapping with the contraction. For this reason, the heart cannot be restimulated during systole.
In conclusion, cardiac muscle cannot undergo tetanus, ensuring the alternation between systole and diastole.

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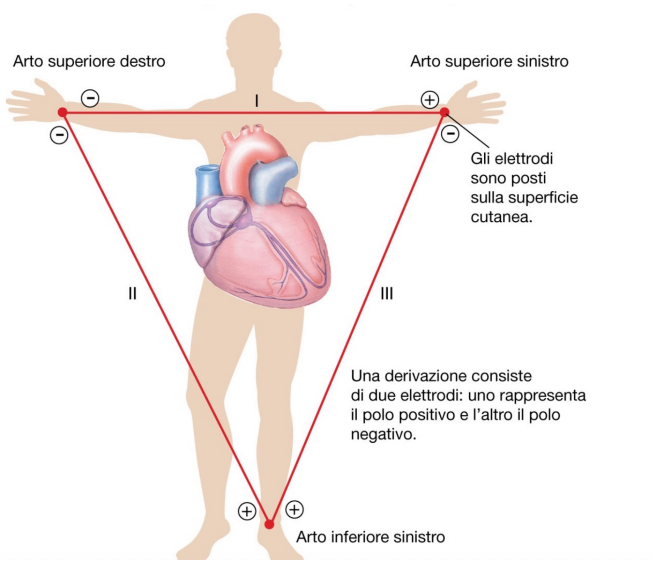
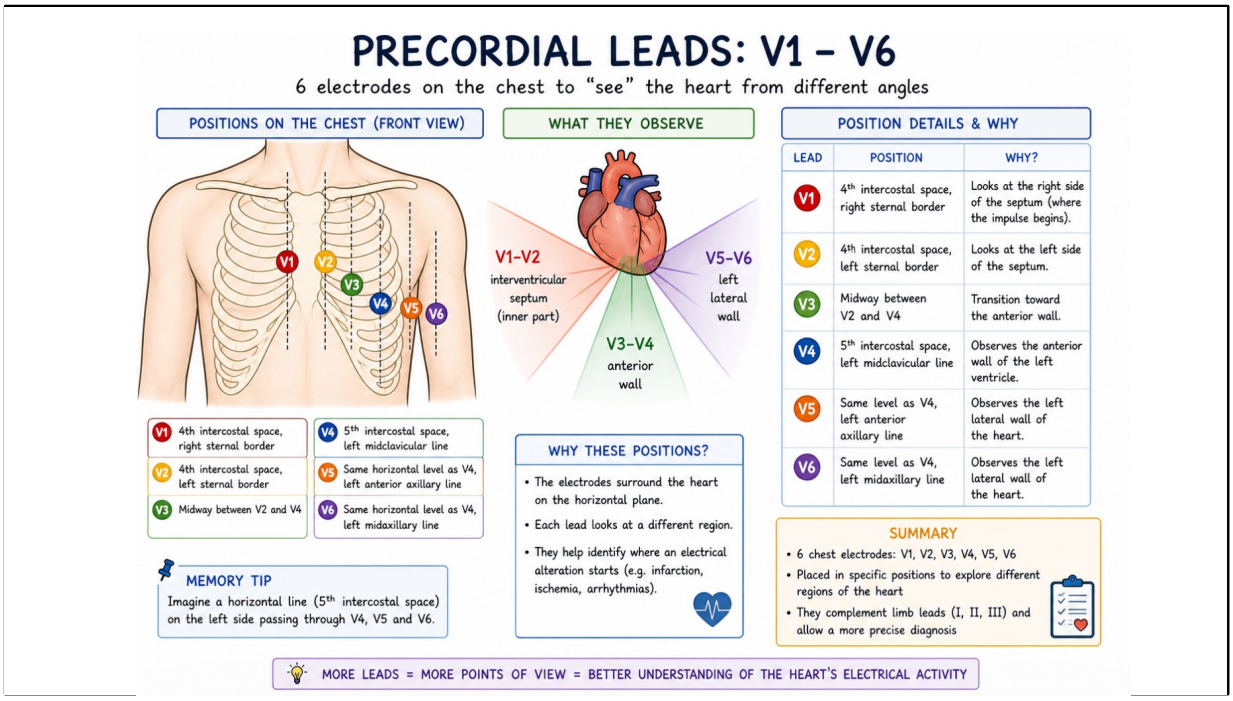
ECG



The ECG records the electrical activity of the heart.
It is the sum of the signals from all cardiac cells.

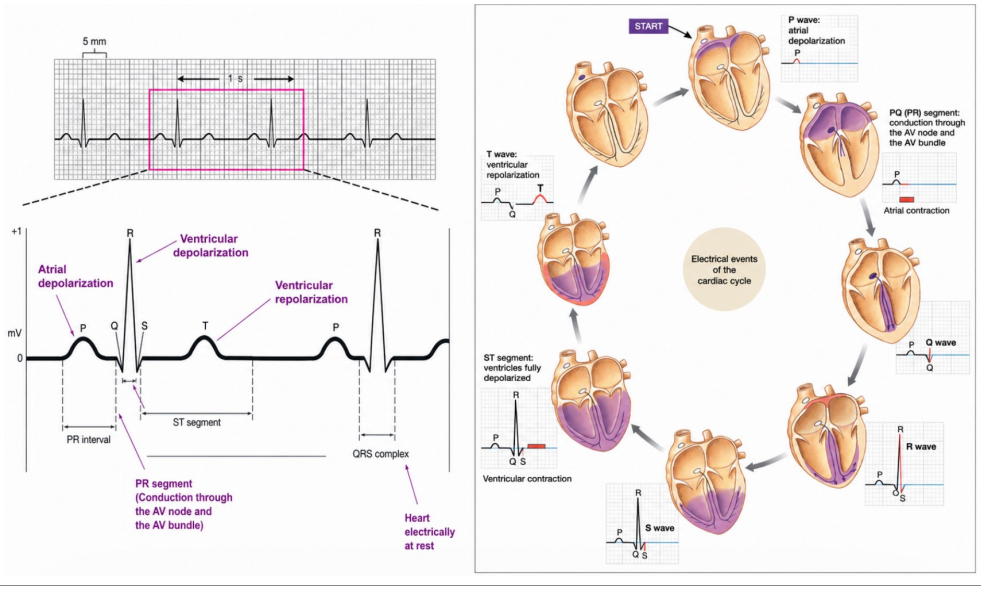
Electrodes are placed on the arms and leg.
Their arrangement forms Einthoven's triangle, which creates three different directions of observation (leads I, II, and III).

Each lead records the projection of the heart's electrical activity along that direction.



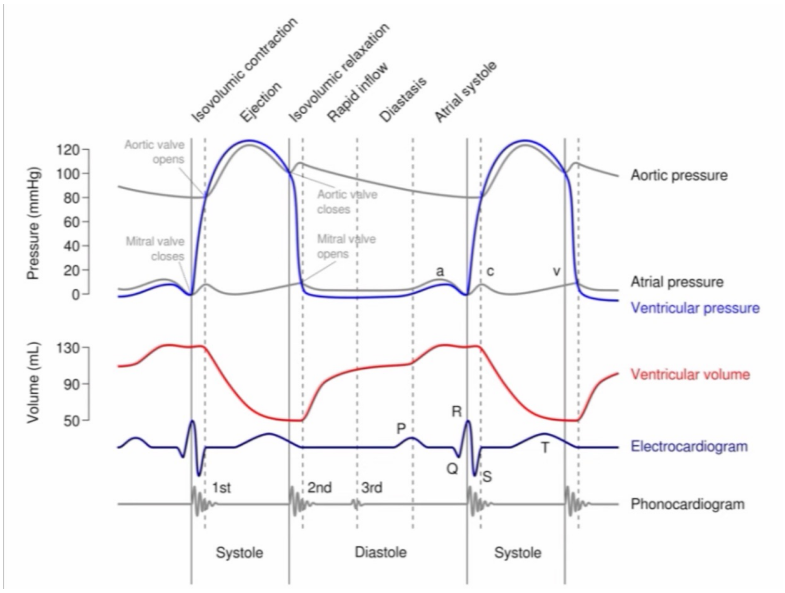
In addition to peripheral electrodes, the ECG uses electrodes that record precordial leads, positioned on the chest near the heart.
These make it possible to observe the heart on the horizontal plane.
In this case, it is as if we were looking at the heart "around the chest," studying the propagation of the electrical signal between the anterior and posterior parts of the heart, as well as between the right and left sides.
In summary:
limb leads observe the heart on the frontal plane;
precordial leads observe the heart on the horizontal plane.
The electrodes are arranged to observe the heart from multiple perspectives.

Sequence of electrical events of the heart and correlation with the ECG



The image represents the sequence of electrical events of the heart during a cardiac cycle, relating them to the ECG waves.
The cycle begins in the sinoatrial (SA) node, which generates the electrical impulse. This impulse spreads through the atria, causing their depolarization, visible on the ECG as the **P wave**.
Subsequently, the impulse reaches the atrioventricular (AV) node, where it is slowed down. This phase corresponds to the **PR segment**, which allows the atria to complete their contraction and the ventricles to fill.
After the AV node, the signal travels along the **bundle of His** and the **Purkinje fibers**, rapidly spreading through the ventricles. Ventricular depolarization generates the **QRS complex**:
the **Q wave** represents the beginning of depolarization,
the **R wave** represents the main spread,
the **S wave** represents the final phase.
During the **ST segment**, the ventricles are fully depolarized and in the contraction phase.
Finally, **ventricular repolarization** occurs, which appears as the **T wave** on the ECG. In this phase, the ventricular muscle relaxes.
Atrial repolarization is not visible because it is masked by the QRS complex.

Wigger's diagram



Every electrical event generates a mechanical event.
To understand what happens during a cardiac cycle, we can use the Wiggers diagram, which combines the ECG, pressures, volume, and valve activity.

In the Wiggers diagram, the first curve to consider is the electrocardiogram (ECG), which is mainly used as a temporal reference to identify the different phases of the cardiac cycle, particularly systole and diastole.
The QRS complex represents the beginning of ventricular systole, since it corresponds to ventricular depolarization and the onset of ventricular contraction. The T wave, on the other hand, represents ventricular repolarization and occurs during the final phase of systole, marking the transition toward diastole.
The ECG therefore makes it possible to temporally correlate all the other mechanical and pressure-related events represented in the diagram.
Another important curve is the one representing left ventricular volume. This curve shows the changes in the volume of blood contained in the left ventricle during the cardiac cycle.
At the beginning of systole, identified by the QRS complex, the ventricle starts to contract, but ventricular volume initially remains constant. This phase is called isovolumetric contraction, because both the mitral and aortic valves are closed.
When ventricular pressure exceeds aortic pressure, the aortic valve opens and the ejection phase begins. During this phase, ventricular volume decreases rapidly, as shown by the descending volume curve.
At the end of ejection, ventricular pressure falls below aortic pressure and the aortic valve closes. The phase of isovolumetric relaxation then begins, during which the ventricle relaxes but the volume remains constant, since both valves are once again closed.
When ventricular pressure becomes lower than left atrial pressure, the mitral valve opens and ventricular filling begins. Ventricular volume initially increases rapidly during the rapid filling phase, then more slowly during passive filling. Finally, atrial contraction causes a further increase in ventricular volume, known as the atrial kick, completing ventricular filling before the beginning of a new cycle.
The diagram also represents the main cardiac pressure curves: left ventricular pressure, left atrial pressure, and aortic pressure.
At the beginning of systole, corresponding to the QRS complex, ventricular pressure rises rapidly during isovolumetric contraction, while volume remains constant.
When ventricular pressure exceeds aortic pressure, the aortic valve opens and ventricular ejection begins. During this phase, ventricular pressure continues to rise and remains slightly higher than aortic pressure, a condition necessary to maintain blood flow into the aorta. Subsequently, at the end of systole, ventricular pressure begins to decrease. When aortic pressure once again becomes greater than ventricular pressure, the aortic valve closes. This event coincides with the beginning of isovolumetric relaxation.
Ventricular pressure then continues to decrease until it becomes lower than left atrial pressure. At this point, the mitral valve opens and blood can once again flow from the left atrium into the left ventricle.
During ventricular filling, atrial pressure is initially greater than ventricular pressure, favoring blood flow. Gradually, the two pressures tend

to equalize. Finally, during atrial contraction, atrial pressure rises again above ventricular pressure, contributing to the completion of ventricular filling.
The cardiac cycle therefore repeats continuously according to this coordinated sequence of electrical, pressure, and volumetric events.

Cardiovascular center in the medulla

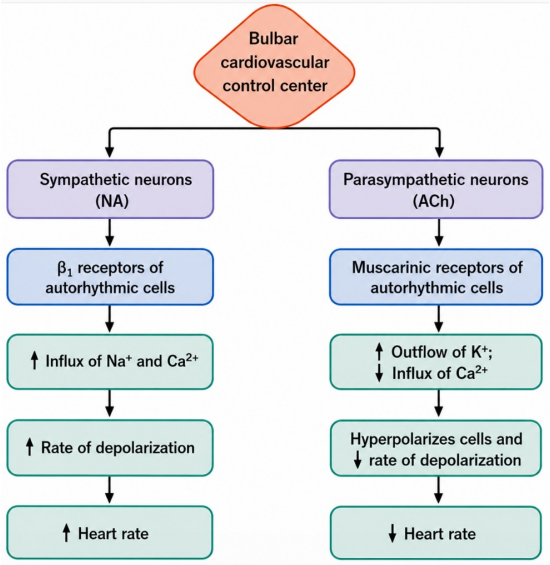
Under normal conditions, the heart is under **tonic control**, meaning a balance between:
the **sympathetic system**
the **parasympathetic system**
The tonic control of heart rate is generated by the **cardiovascular center in the medulla (oblongata)**.

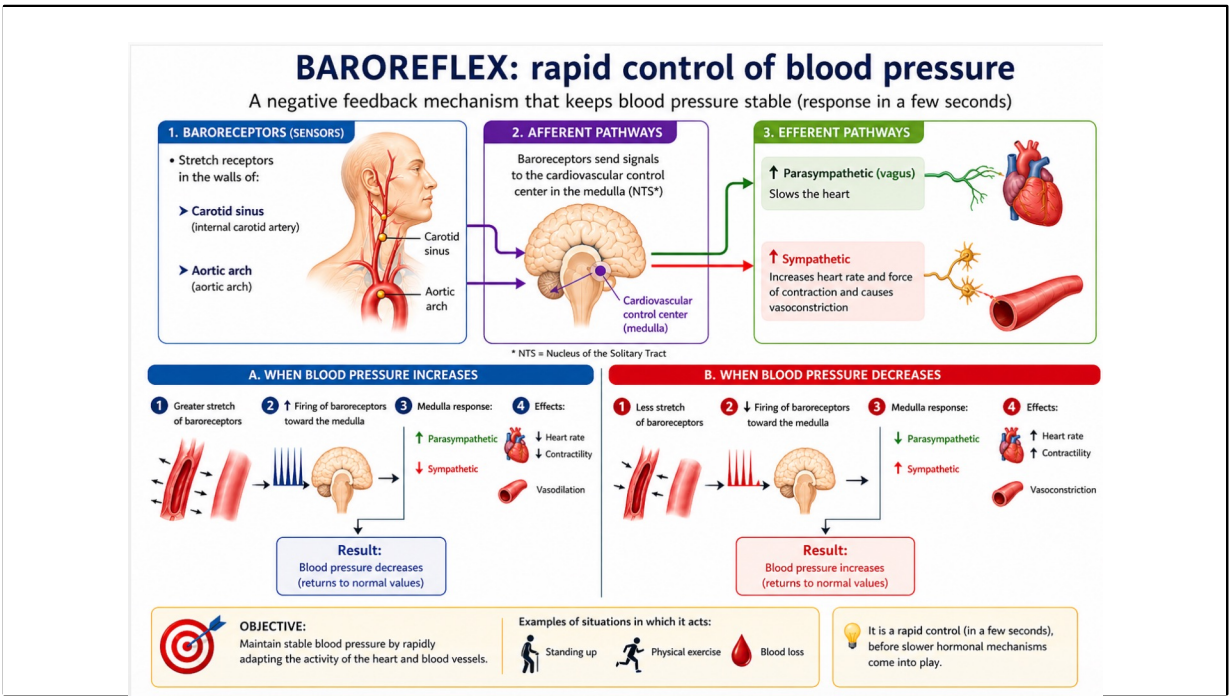
Sympathetic system (activates the heart)
"The sympathetic system, through **norepinephrine**, binds to **β1 receptors** on cardiac cells and:
increases the influx of Na^+ and Ca^{2+}
increases the rate of depolarization

Result: increased heart rate and also increased force of contraction

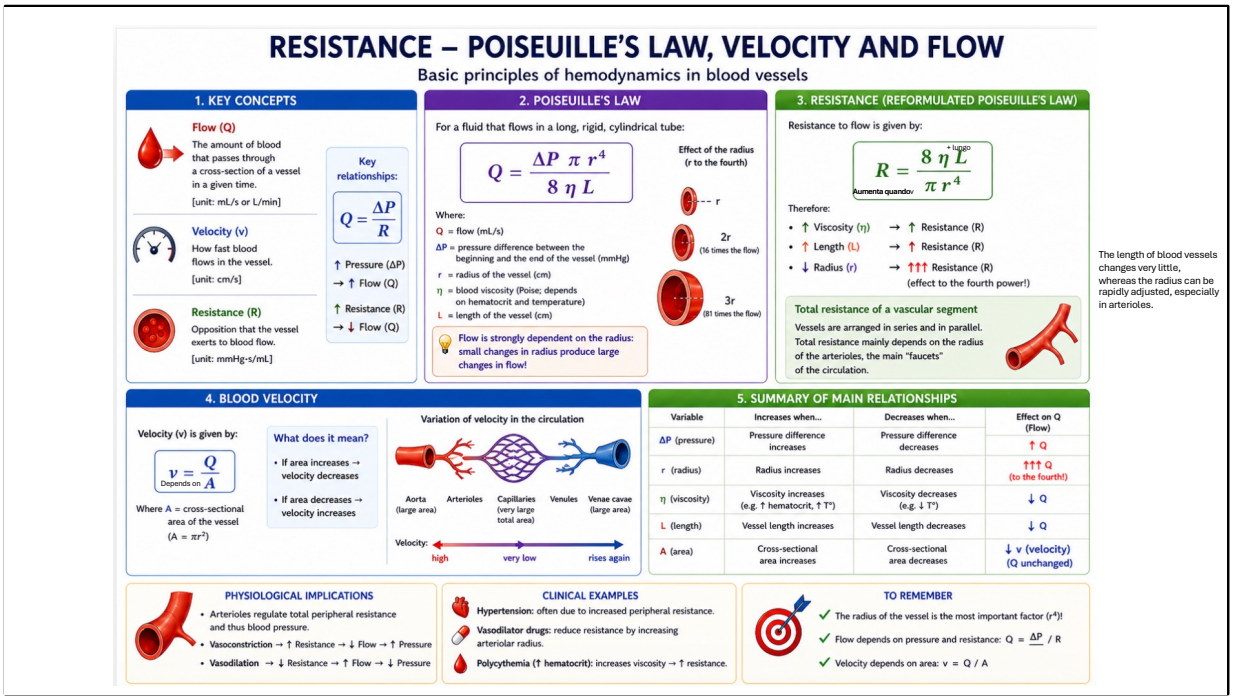
Parasympathetic system (slows the heart)
"The parasympathetic system, through **acetylcholine**, acts on **muscarinic receptors** and:
increases K^+ efflux
reduces Ca^{2+} influx
hyperpolarizes the cells

Result: decreased heart rate.





The cardiovascular center in the medulla also rapidly controls arterial pressure through the baroreflex.
The baroreflex is a negative feedback mechanism that maintains arterial pressure stable. Baroreceptors, located in the carotid sinus and the aortic arch, detect pressure changes based on the degree of stretch of the vessel walls.
This information is sent to the cardiovascular center in the medulla, which regulates autonomic nervous system activity.
When arterial pressure increases, parasympathetic activity increases and sympathetic activity decreases. This causes a reduction in heart rate, contractile force, and vasodilation, bringing pressure back toward normal values.
Conversely, when pressure decreases, parasympathetic activity decreases and sympathetic activity increases. As a result, heart rate and contractility increase, together with vasoconstriction.
The baroreflex is therefore a rapid and effective system that allows the body to adapt immediately to changes in arterial pressure.



The length of blood vessels changes very little, whereas the radius can be rapidly adjusted, especially in arterioles.

After examining the mechanisms that regulate heart rate and arterial pressure, we can now analyze the fundamental principles of hemodynamics, that is, how blood flows through vessels.

The three key concepts are flow, resistance, and velocity. Flow represents the amount of blood passing through a vessel within a given time interval, whereas resistance indicates the opposition the vessel exerts against flow. Velocity, on the other hand, describes how rapidly blood flows.

These parameters are interconnected. In particular, flow depends on the pressure difference between two points in the system and on vascular resistance: the greater the pressure, the greater the flow; the greater the resistance, the lower the flow.

The most important relationship is described by Poiseuille's law, which states that flow is proportional to the pressure difference and to the fourth power of the vessel radius, and inversely proportional to blood viscosity and vessel length.

This means that vessel radius is the most important factor: even small changes in diameter, such as those occurring during vasoconstriction or vasodilation, produce major changes in flow and resistance.

In fact, resistance increases when the radius decreases, when blood viscosity increases, or when the vessel is longer.

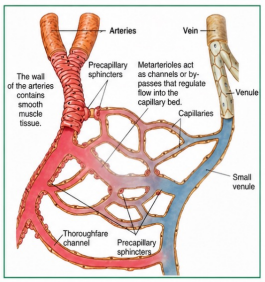
However, in our body, vessel length changes very little, whereas the radius can be rapidly modulated, especially at the level of the arterioles, which are the main regulators of peripheral resistance.

As regards blood velocity, it depends on flow and on the cross-sectional area of the vessel. In particular, velocity is inversely proportional to the area: when the total area increases, as in the case of capillaries, velocity decreases. This is essential because it allows exchanges between blood and tissues.

In summary, pressure drives blood flow, resistance opposes flow, and vessel radius is the factor that most strongly influences these processes. These principles are essential for understanding both circulatory physiology and many pathological conditions, such as hypertension or the effects of vasodilator drugs.

Function of different vessels

	Diameter (mean)	Thickness of vessel wall (mean)	Elasticity	Tissue mass	Smooth muscle mass	Fibrous tissue	
Arteries	4.0 mm	1.0 mm					
Arterioles	30.0 µm	6.0 µm					
Capillaries	8.0 µm	0.5 µm					
Venules	20.0 µm	1.0 µm					
Veins	5.0 mm	0.5 mm					



Similar to capillaries, but with a fibrous tissue comparatively.

"Volume reservoir"
Located superficially"

Arteries have thick, elastic walls and serve to carry blood at high pressure from the heart to the tissues.

Arterioles are the main resistance vessels: thanks to their smooth muscle, they regulate their diameter and therefore control blood flow and arterial pressure.

Capillaries are very thin and represent the site of exchange between blood and tissues, due to their extremely permeable walls and the low velocity of blood flow.

Venules and veins collect blood and return it to the heart; they have thinner walls and a greater capacity to stretch, which is why they function as a volume reservoir.

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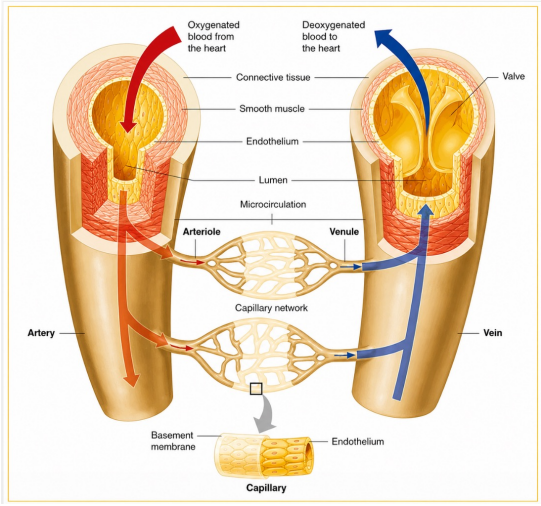
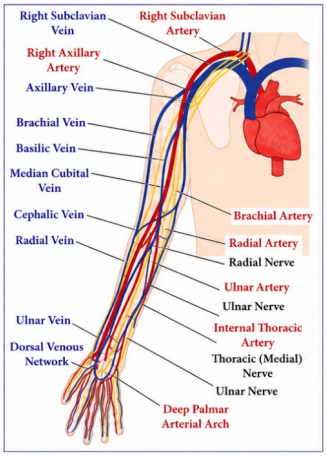
Venules and veins collect blood and return it to the heart; they have thinner walls and a greater capacity for distension, which is why they function as a volume reservoir.
Each type of vessel is specialized for a specific function: transport, flow regulation, exchange, and blood storage.

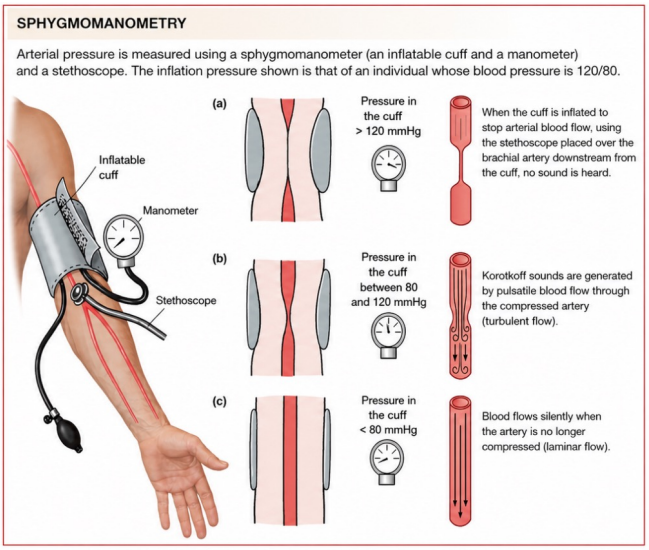
Relationships between blood vessels and direction of blood flow



This image shows the relationships between blood vessels and the direction of blood flow.
Blood leaves the heart and flows into the arteries, which have thick, strong walls because they must withstand high pressures. From there, it passes into the arterioles, which regulate blood flow to the tissues.
The blood then enters the capillaries, where the exchange of gases, nutrients, and metabolites with the tissues takes place, thanks to the very thin wall made of a single layer of endothelium.
From the capillaries, the blood passes into the venules and then into the veins, which return the blood to the heart. Veins have thinner walls and contain valves that prevent blood backflow and help venous return.

Arterial pressure and its measurement

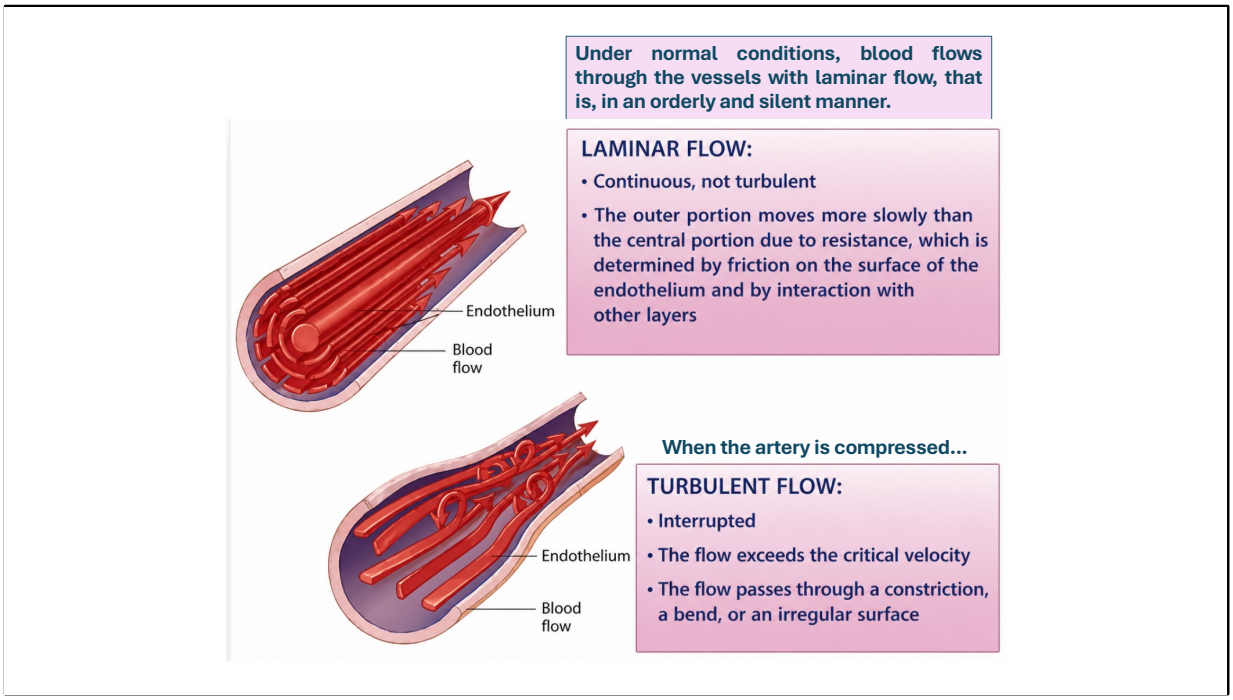




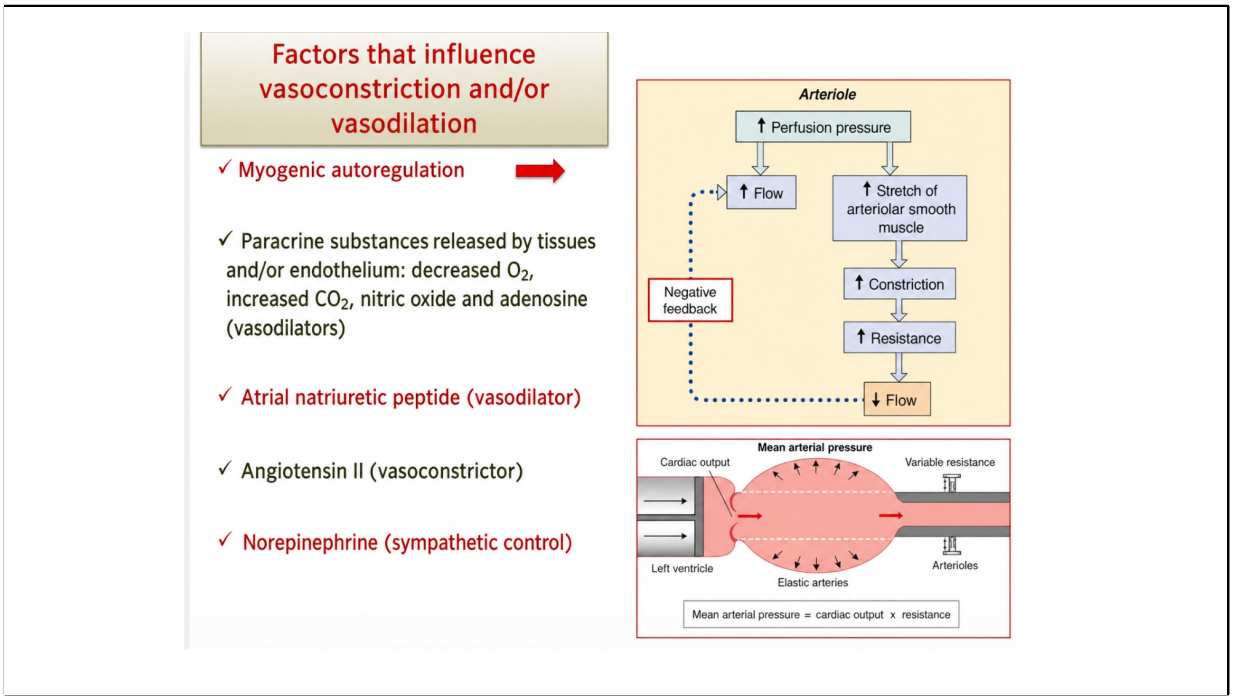
Artery closed – no flow
No "sound"

80–120 mmHg
Interval
Pulsatile "sounds"

< 80 mmHg
No "sound"

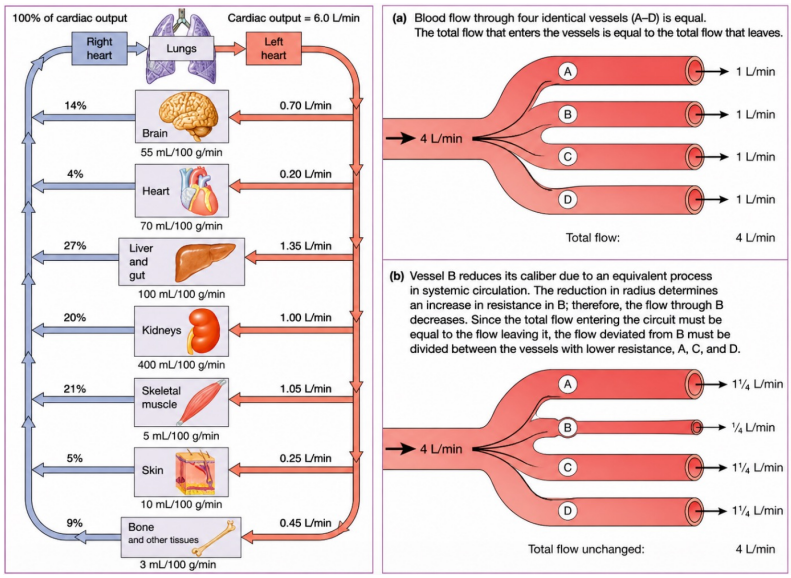


In condizioni normali, il sangue scorre nei vasi con flusso laminare, cioè in modo ordinato e silenzioso: gli strati più interni scorrono più velocemente, quelli vicino alla parete più lentamente. Questo tipo di flusso non produce rumore.
Quando invece il flusso diventa turbolento, il sangue si muove in modo disordinato, con vortici, e questo genera rumore.
Durante la misurazione della pressione:
quando il bracciale comprime completamente l'arteria brachiale, il flusso si ferma → nessun rumore
quando la pressione del bracciale scende sotto la pressione sistolica, il sangue riesce a passare ma attraverso un vaso parzialmente compresso, quindi il flusso diventa turbolento → si sentono i suoni (suoni di Korotkoff)
quando la pressione scende ulteriormente sotto la diastolica, l'arteria si riapre completamente, il flusso torna laminare → il rumore scompare.



Several factors regulate vessel caliber, that is, vasoconstriction and vasodilation, and therefore resistance and blood flow.
The first mechanism is myogenic autoregulation: when perfusion pressure increases, the wall of the arteriole stretches, and this induces a reflex contraction of the smooth muscle. In this way, resistance increases and blood flow is brought back toward normal values: this is an example of negative feedback.
There are also local factors, namely substances released by tissues or by the endothelium:
reduced oxygen, increased CO_2, nitric oxide, and adenosine → vasodilation;
these factors serve to increase blood flow where there is greater metabolic activity.
There are also hormonal factors:
atrial natriuretic peptide → vasodilator;
angiotensin II → powerful vasoconstrictor.
Finally, the nervous system:
noradrenaline, through the sympathetic nervous system, causes vasoconstriction.

Distribution of blood flow to different organs



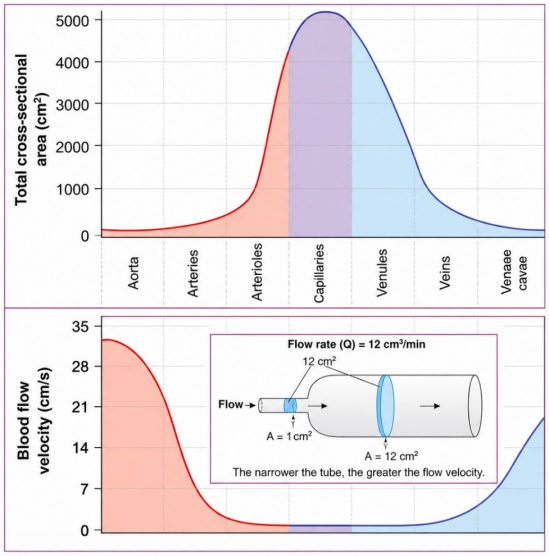
If one vascular district increases its resistance, it receives less blood, which is redistributed to the other districts.
To better understand this concept, let us look at the example in the figure.
In the upper part, all the vessels have the same resistance, so blood flow is distributed uniformly: each branch receives the same amount of blood.
In the lower part, however, one of the vessels — vessel B — becomes narrower, therefore its resistance increases.
What happens?
Less blood flows through vessel B, while the flow is diverted toward the other vessels, which have lower resistance and therefore receive more blood.
It is important to note that the total flow remains unchanged: only its distribution among the different branches changes.

Flow velocity and cross-sectional area

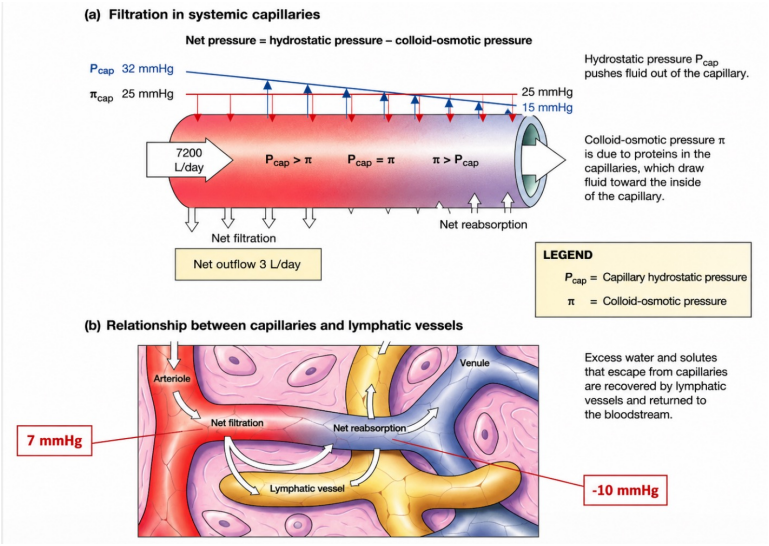
Maximum total area in capillaries → minimum velocity

Velocity of blood flow

The velocity of blood flow depends on the value of the cross-sectional area of the total section



Capillary exchange: bulk flow



After seeing that flow velocity depends on the cross-sectional area of the vessel, and that in capillaries it is very low precisely because the total area is very large, we can understand why capillaries are the ideal site for exchange.
At the capillary level, in fact, the movement of water and solutes occurs between the blood and the interstitial fluid.
This exchange is mainly regulated by two forces:
hydrostatic pressure, which tends to push fluid out of the capillary;
oncotic pressure, due to plasma proteins, which draws fluid back into the capillary.
At the beginning of the capillary, hydrostatic pressure predominates, so filtration occurs: fluid moves out toward the tissues.
At the end of the capillary, oncotic pressure predominates instead, so reabsorption occurs: fluid re-enters the capillary.
A small portion of the fluid is not reabsorbed and is drained by the lymphatic system.
In this way, a balance is maintained between fluid leaving and re-entering the capillaries, which is essential for the proper functioning of tissues.
From a clinical point of view, if this balance is altered — for example because of increased hydrostatic pressure, reduced plasma proteins, or problems with lymphatic drainage — fluid accumulates in the tissues, resulting in edema.