Overview of the cardiovascular system (CVS)

CVS Function :

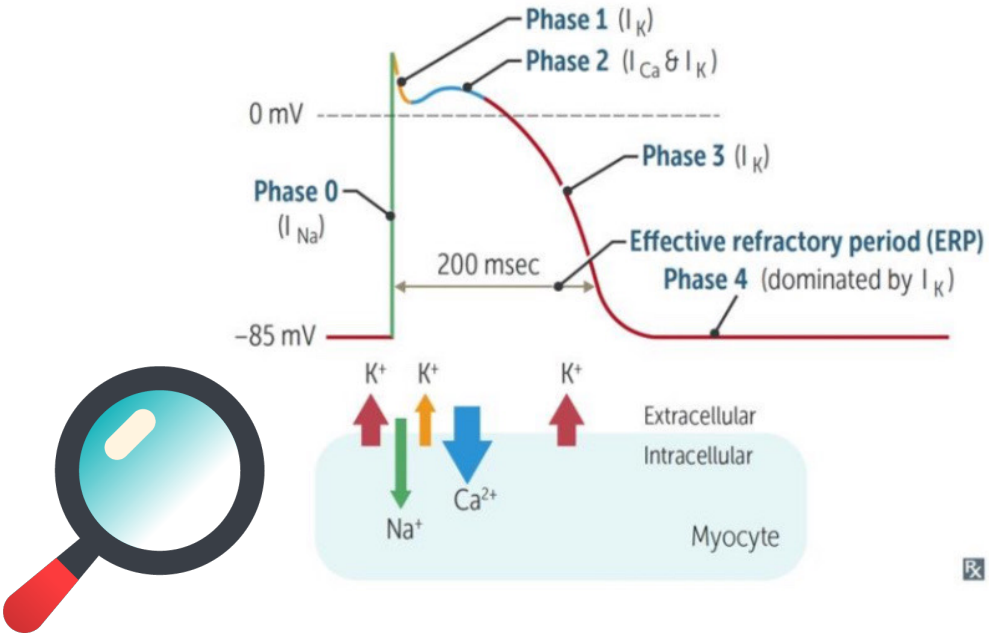
* Deliver blood to the tissues and remove waste products from the cells
* regulation of body temperature, blood pressure, hormones and so on.
* the vessels that carry blood from the heart to the tissues are arteries " which are under high pressure and contain small percentage of blood volume.
*the veins which carry blood from the tissues to the heart are under low pressure and contain the largest percentage of blood volume.
* the presence of 4 one way heart ensures unidirectional flow of blood which open and close passively because of pressure difference.

Sytemic vs pulmonary circulation

The pulmonary circulation consists of a closed loop of vessels carrying blood between the heart and the lungs and The systemic circulation is a circuit of vessels carrying blood between the heart and all body systems (except for the air sacs of the lungs, which are supplied by the pulmonary circulation

Both sides of the heart simultaneously pump equal amounts of blood ,but the left side work harder because equal amounts at a higher pressure into a higher resistance and longer system + the left side is thicker.

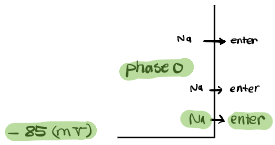
The pulmonary circulation is a low–pressure, low resistance system, whereas the systemic circulation is a high pressure, high–resistance system.



Cardiac muscle action potential

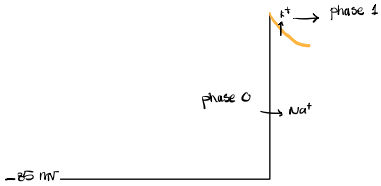
Phase 0 (Depolarization)

Fast Sodium Channels Open, so membrane potential becomes more positive reach about +20 millivolts before sodium channels close.



Phase 1 (Initial Repolarization)

The sodium channels close, the cell begins to repolarize, and potassium ions leave the cell through open potassium channels



Phase 2 (Plateau ☠️) :

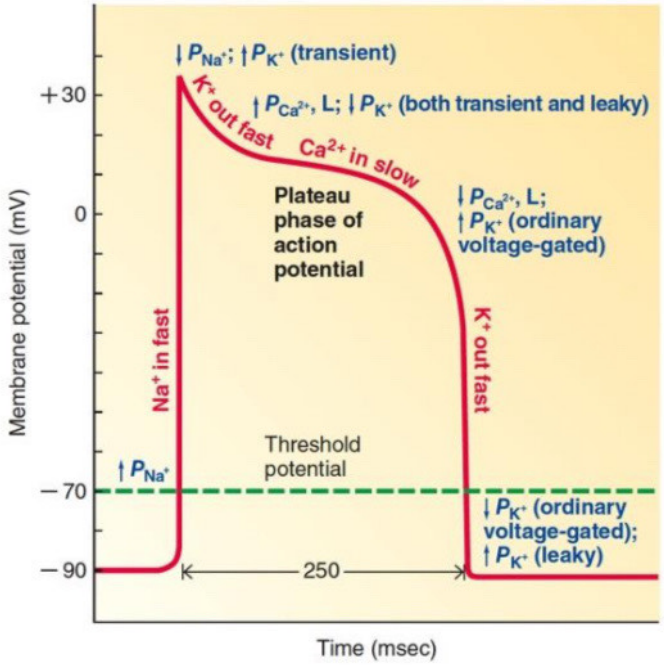
- Ion Channel Activity: Calcium channels open and fast potassium channels close, leading to a brief initial repolarization followed by a plateau in the action potential. This plateau results from increased calcium ion entry and decreased potassium ion exit.
- Calcium and Potassium Dynamics: Voltage-gated calcium channels open slowly during phases 0 and 1, allowing calcium to enter. Potassium channels close, resulting in decreased potassium efflux and sustained calcium influx, which together create the plateau in the action potential.
- Comparison with Skeletal Muscle: Unlike skeletal muscle, where fast sodium channels primarily drive the action potential, cardiac muscle involves both fast sodium channels and slow L-type calcium channels. These calcium channels open slower, stay open longer, and allow significant calcium influx, prolonging depolarization and producing the plateau.
- Calcium's Role in Contraction: In cardiac muscle, calcium ions entering during the plateau phase activate muscle contraction. This differs from skeletal muscle, where contraction is driven by calcium released from the sarcoplasmic reticulum.
- Potassium Permeability Shift: At the start of the action potential, cardiac muscle membrane permeability to potassium ions drops significantly, reducing potassium efflux and maintaining the plateau. When slow calcium channels close after 0.2–0.3 seconds, potassium permeability quickly increases, allowing potassium exit and returning the membrane potential to resting levels, thus ending the action potential.

Phase 3 (Rapid Repolarization)

Calcium Channels Close and Slow Potassium Channels Open.

Phase 4 (Resting membrane potential)

This averages about-80 to -90 millivolts



معلومة لطيفة : ينبض القلب في اليوم الواحد حوالي ١٠٠٠٠٠ نبضة أي حوالي ٣ مليار مرة في متوسط حياة الإنسان 🫶

SA node

The fibers of this node have almost no contractile muscle filaments and are each only 3 to 5 micrometers (µm) in diameter, in contrast to a diameter of 10 to 15 µm for the surrounding atrial muscle fibers, the sinus nodal fibers connect directly with the atrial muscle fibers, so that any action potential that begins in the sinus node spreads immediately into the atrial muscle

	SA node	Vs	Ventricular Muscle fiber
Resting membrane potential	-55 - 60		-85 - -90

1. less negativity
2. leaky to sodium and calcium ions, and positive charges of Ca^+ and Na^+ neutralize some of intracellular negativity.

1. more negativity

Important note

Autorhythmic cell do not have a resting potential + only the slow calcium channels can open (become activated) and thereby cause action potential (pacemaker potential)

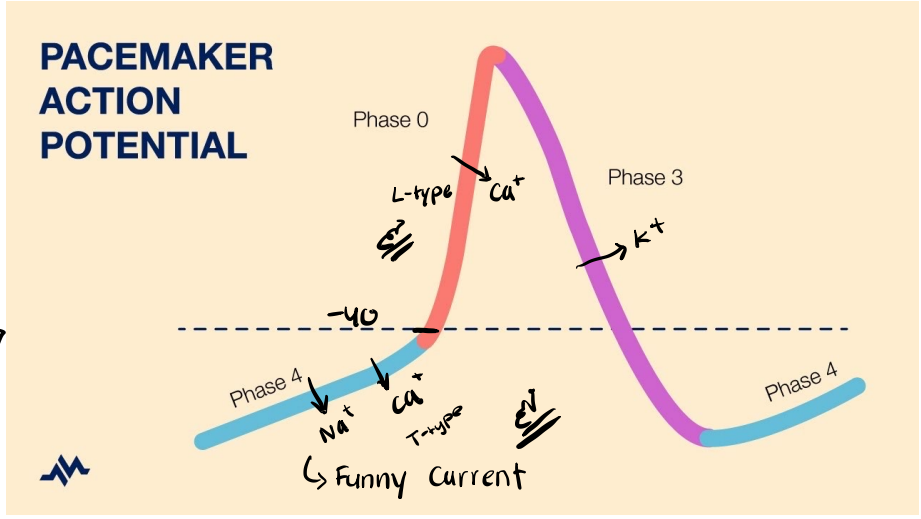
Pacemaker Potential

Hyperpolarization and Its Termination in Pacemaker Cells

1. Hyperpolarization Phase: After each action potential, the cell enters a hyperpolarized state.
2. Termination Mechanism: This state doesn't last because more potassium (K) channels gradually close after the action potential ends.
3. Resting Potential Drift: As K efflux decreases, inward sodium (Na) and calcium (Ca) currents start to exceed K outflow, causing the membrane potential to rise again.
4. Threshold and Re-excitation: The resting potential drifts upward, eventually reaching a threshold of about -40 mV, triggering the next action potential.
5. Continuous Cycle: This repeating cycle of action potential, recovery, hyperpolarization, and gradual depolarization sustains pacemaker activity throughout life.

Action Potential Mechanism in Pacemaker Cells

1. Threshold and Rising Phase: When the threshold is reached, long-lasting Ca channels (L-type) open, allowing a large influx of Ca, which drives the rising phase of the action potential.
2. Comparison with Other Cells: Unlike nerve and skeletal muscle cells that rely on Na influx, pacemaker cells use Ca influx to depolarize.
3. Falling Phase: The falling phase results from K efflux, triggered by increased K permeability as K channels open and L-type Ca channels close.
4. Next Depolarization Cycle: Slow closure of K channels after the action potential promotes a gradual depolarization, leading to the next threshold event.



Self-Excitation Mechanism in Pacemaker Cells

1. Ion Leakiness: Due to a high extracellular Na concentration and moderately open Na channels, Na ions constantly leak into the pacemaker cells.
2. Gradual Depolarization: The inflow of positive Na ions causes a slow, steady increase in the resting membrane potential between heartbeats, making it less negative.
3. Threshold Triggering: When this rising potential reaches around -40 mV, L-type Ca channels activate, causing a new action potential.
4. Cycle of Self-Excitation: This continuous Na and Ca leakiness enables pacemaker cells to self-excite, initiating each heartbeat autonomously.

Funny Na Channels (If Channels) in Pacemaker Cells

1. Initial Depolarization Phase: The initial slow depolarization to threshold is driven by Na entry through specific voltage-gated channels called “funny” or If channels, unique to pacemaker cells.
2. Activation on Hyperpolarization: Unlike typical channels that open with depolarization, If channels open when the membrane hyperpolarizes (becomes more negative) at the end of each repolarization.
3. Role in Threshold Drift: When If channels open, inward Na currents immediately begin to depolarize the membrane, moving it back toward the threshold.

Role of K Channels in Pacemaker Potential

1. Progressive K Channel Closure: In pacemaker cells, K channels slowly close at negative potentials after each action potential, unlike in nerve or muscle cells where K permeability remains stable.
2. Reduced K Efflux: The gradual closing of K channels reduces the outward flow of K, which lowers the rate of repolarization.
3. Contribution to Depolarization: This decreased K efflux, alongside Na influx through open If channels, supports the slow drift of the membrane potential toward threshold.

Role of Ca Channels in Pacemaker Potential

1. Increased Ca Influx: During the second half of the pacemaker potential, transient Ca channels (T-type) open, allowing a brief influx of Ca.
2. Further Depolarization: This Ca entry accelerates depolarization, bringing the membrane potential closer to threshold.
3. Threshold Achievement: Once threshold is reached, T-type Ca channels close, and the cycle proceeds to action potential generation.