Cardiac cells(cardiomyocytes) within your heart:

99% : Contractile cells

They contract the heart to pump the blood

These cells contract as one unit due to the gap junctions which manage the impulse to transmit through all the neighboring cells in nearly no time and this is called : **Syncytium**

They have fast response action potential (FRAP)

No excitation unless the impulse dosen't come from the conductive cells

Phase 4: Resting Phase

- Resting membrane potential is stable at ~-90 mV.
- Maintained by the Na^+/K^+ ATPase pump and K^+ leak channels.

Phase 0: Rapid Depolarization

- Triggered by stimulus from adjacent cell via gap junction.
- Fast Na^+ channels open → Na^+ floods in → membrane rapidly depolarizes to +20 mV.

Phase 1: Initial Repolarization

- Fast Na^+ channels inactivate.
- Transient K^+ channels (Ito) open briefly → slight K^+ efflux → slight drop in voltage.

Phase 2: Plateau

- L-type Ca^{2+} channels open → Ca^{2+} enters the cell.
- At the same time, K^+ continues to exit.
- The inward Ca^{2+} balances the outward K^+, creating a plateau near 0 mV.
- This phase is unique to cardiac muscle and is responsible for prolonging the action potential → allows sustained contraction.

Phase 3: Repolarization

- Ca^{2+} channels close, but K^+ channels stay open → K^+ efflux dominates.

1%: Conductive cells

They form the intrinsic conduction system of the heart

They generate and convey the impulses by themselves to excite the contractile cells

They have slow response

Auto excitation due to their membranes are leaky for sodium ions at the resting state

Phase 4 – Pacemaker potential (Slow depolarization)

- Resting membrane potential: around -60 mV (not stable; it slowly depolarizes).
- The membrane is leaky to sodium ions through "funny" channels (If channels, not typical voltage-gated Na^+).
- These If channels allow Na^+ influx, causing slow depolarization.
- T-type (transient) Ca^{2+} channels open briefly around -50 to -45 mV, allowing some Ca^{2+} entry.

Phase 0 – Rapid depolarization

- At around -40 mV, L-type (long-lasting) Ca^{2+} channels open.
- This causes a rapid influx of Ca^{2+} → sharp depolarization.
- Membrane potential rises up to about +10 to +20 mV.

Phase 3 – Repolarization

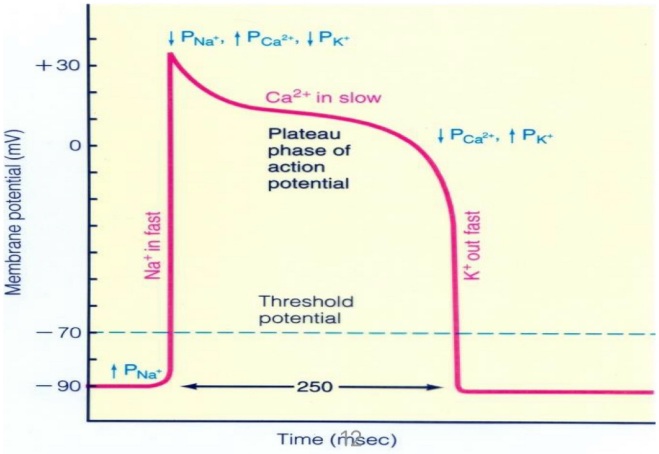
- L-type Ca^{2+} channels close.
- Voltage-gated K^+ channels open, causing K^+ efflux.
- Membrane potential repolarizes back down to around -60 mV.

Cycle Repeats

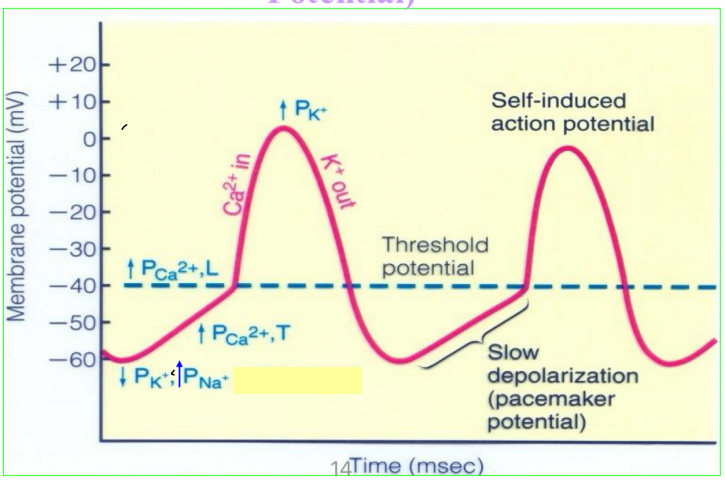
- Once back to -60 mV, If (funny) Na^+ channels reopen → cycle

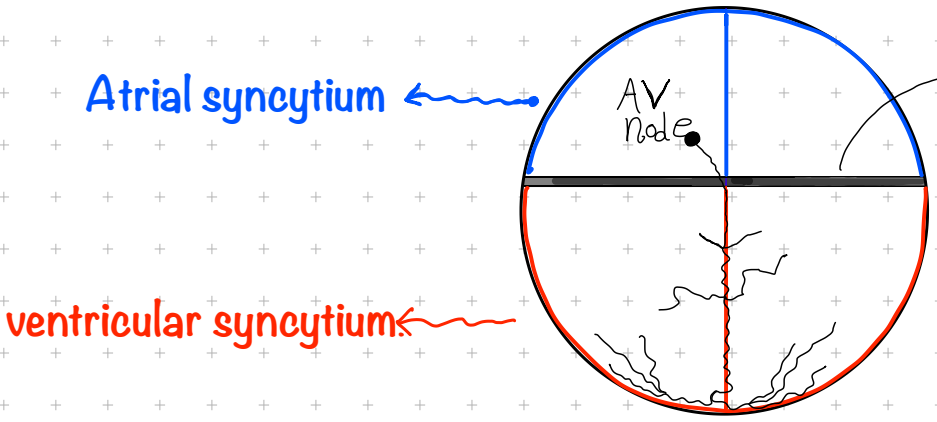
The threshold is around -40 mV, and rapid depolarization occurs afterward due to L-type Ca^{2+} channels.

Fast Response Action Potential of Contractile Cardiac Muscle Cell



Slow Response Action Potential (Pacemaker Potential)





Electrically , atria and ventricles are separated from each other by the electraical insulator and they can communicate via the **AV node** only (this is the bridge between them)

(Heart)

In syncytium's cells there are gap junctions between them so once a cell becomes excited all the neighboring cells become excited in no time!

Normal heart which it is driven by SA node , it gives a beat each 0.8 seconds

- 0.5 seconds → Ⓐ Diastole (Relaxation) → So significant to fill the ventricles by blood
- 0.3 seconds → Ⓑ Systole (contraction) → To ejaculate the blood from the ventricles

Ectopic pacemaker

It excites the cardiac muscle rapidly and successively so this reduces the duration of the relaxation (diastole) and this prevents the heart to take up the needed blood for ejaculation so this leads to many problems and this condition is called : **Ventricular fibrillation (الرجفان البُطيّني)** .This case may be treat by very very high vlotage via direct current shock (DC shock) (nearly :IOOOOV for just milliseconds) so this causes **Defibrillation**

Tetanus

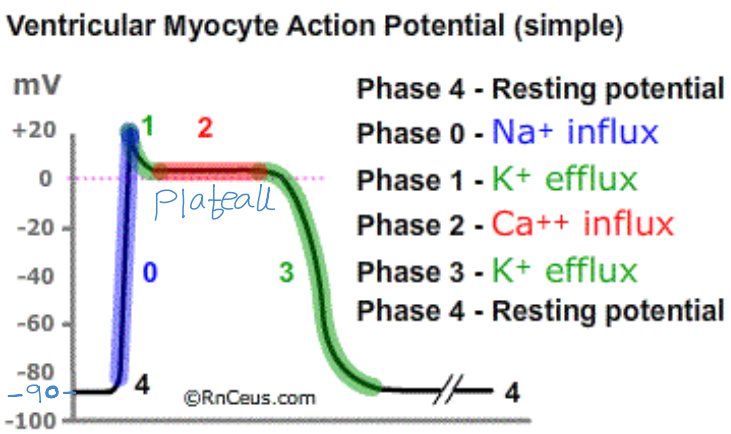
It is a condition where the sustained contraction happens for the skeletal muscle without occurring of relaxation for that muscle due to the successive receieved impulses in the muscle

The reason: The action potential in the skeletal muscle cell is very short this means that we can restimulate another action potential after the first AP by a too short duration so this doesn't manage the muscle to be relax.

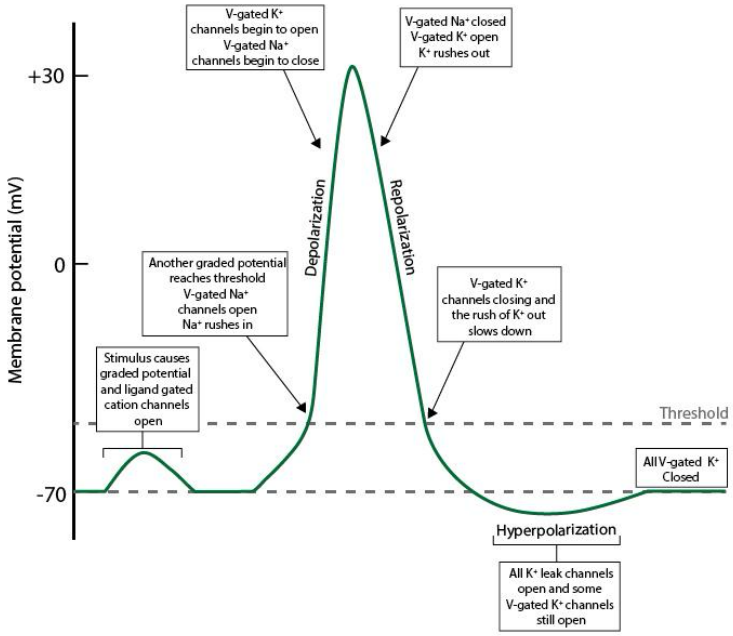
Note : The tetanisation in the heart is impossible due to the phase (2) (plateau) in the diagram of AP in the contractile cardiac muscle cell where the influx of positive charges= efflux of the positive charges .In each AP in the contractile cardiac muscle cell there is one absloute refractory period (the longest one) and the plateau is a big part of this period so this ensures fully contraction of the heart before it becomes relaxed .Another refractory period in the cardiac muscle ia : Relative refractroy period (from mid to late phase 3) it is very short but so important clinically

Tetanisation

Skeletal muscle	Cardiac muscle
Possible	Impossible



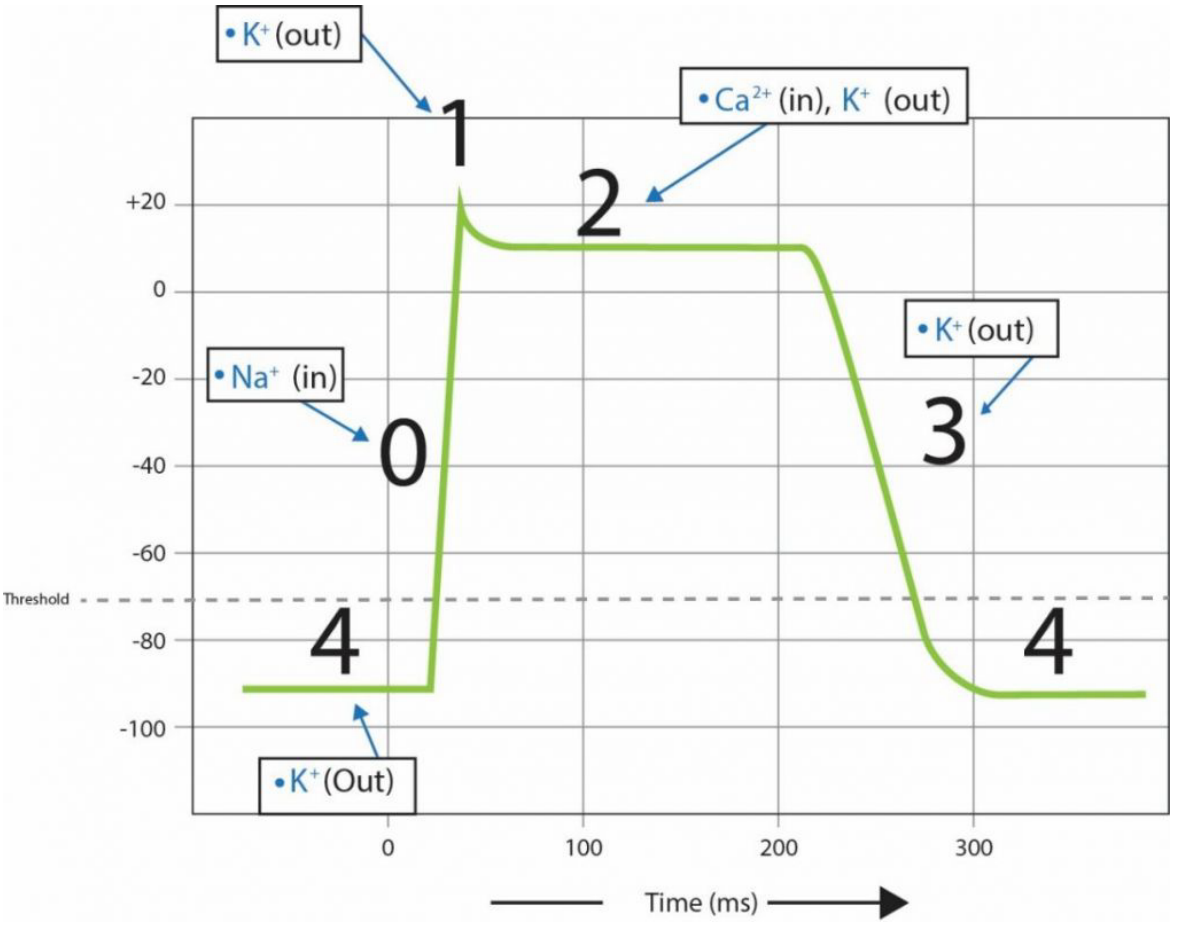
Action Potential in Skeletal Muscle.



Recall that the major phases of the action potential for skeletal muscle cells and axons, are depolarization, repolarization, and hyperpolarization. The opening of voltage-gated Na+ channels and rapid movement of Na+ into the cell, which causes the membrane potential to become more positive, is the depolarization phase. At the peak of the depolarization phase the inactivation gates on the sodium channels close, preventing further sodium entry. At roughly the same time that the inactivation gates close, the voltage-gated K+ channels finish opening and K+ rapidly diffuses out of the cell causing the membrane potential to again become negative, the repolarization phase. The relatively slow closing of the voltage-gated K+ channels results in hyperpolarization (potential below the normal resting membrane potential) and then once they close the resting potential is again restored via leak K+ channels. All of this takes place in 1-2 milliseconds.

Action potentials in cardiac muscle are significantly different from those in axons and skeletal muscle. The most significant difference is the plateau phase (phase 2) that prolongs the action potential to as long as 300 milliseconds. Another difference is that in addition to Na+ and K+, Ca2+ plays a significant role in cardiac muscle action potentials. Note in Fig 4 that there are 5 phases in cardiac muscle action potentials (phase 0, 1, 2, 3, and 4). Let's walk through

Action Potential of Cardiac Myocytes or Cardiac Muscle Cells.



Phase 4: Resting membrane potential (RMP). Note that unlike the -70 to -80 mV RMP that we are familiar with in axons and skeletal muscle, in cardiac muscle, the RMP is around -90 mV. This low resting membrane potential is due to a special group of K+ channels that open when the membrane repolarizes and close when the membrane depolarizes. Because this is essentially the lower limit for the RMP, we do not observe a hyperpolarization at the end of repolarization.

Phase 0: The depolarization phase. This phase is due to the opening of voltage-gated Na+ channels and the influx of Na+. These are the same channels found in axons and skeletal muscle and, hence, have both activation and inactivation gates. Note that when the membrane depolarizes the K+ channels mentioned in phase 4 close.

Phase 1: Rapid repolarization. At the end of the depolarization phase the inactivation gates on the Na+ close, stopping the influx of Na+. At the same time, a small number of K+ channels open and the membrane begins to repolarize.

Phase 2: Plateau. This is the phase that distinguishes the cardiac muscle action potential from other excitable tissues and is the result of the opening of voltage-gated Ca2+ channels. With the opening of these channels, Ca2+ enters the cell. Additional K+ channels also open at about the same time. During the plateau, the influx of Ca2+ essentially negates the effect of the efflux of K+. Because of the movement of these two ions, K+ out and Ca2+ in, the membrane potential remains fairly constant and does not repolarize. In addition to prolonging the action potential, the Ca2+ that is entering the cell plays a critical role in triggering muscle contraction (more on

Phase 3: Repolarization. During the plateau phase more and more K+ channels open and toward the end of the plateau phase the K+ efflux becomes greater than the Ca2+ influx and the membrane begins to repolarize. As the membrane becomes more negative the Ca2+ channels close and the membrane quickly returns to the RMP. As RMP is reached, the K+ channels close (with the exception of those mentioned in phase 4 which open as the membrane repolarizes) and the membrane is ready to respond again. This process is regulated by the ratio of Ca2+ to K+ permeability. There are at least 4 different types of voltage-gated K+ channels involved in the process, each with slightly different properties.

The prolonged nature of the action potential in cardiac muscle has at least 2 important outcomes. First, it prevents the membrane from being restimulated until the muscle has had time to contract and then relax. Stated another way, the absolute refractory period for cardiac muscle cells is much longer, preventing the muscle from being restimulated until it has time to totally relax. Recall that in skeletal muscle if the frequency of action potentials is high enough the muscle will enter a state of tetany in which the muscle remains continually contracted. If this happened in the heart, blood flow would stop, since refilling of the chambers requires that the heart relax. Second, contraction of cardiac muscle requires the contribution of extracellular Ca2+. All during the plateau phase Ca2+ is entering the cell from the extracellular fluid, contributing to total intracellular calcium concentration.

Like skeletal muscle, the signal that triggers contraction of cardiac muscle is an action potential. Action potentials are highly ordered changes in membrane potentials due to the movement of ions across the membranes through voltage-gated ion channels.

Extrinsic innervation of the heart

The heart contains an intrinsic conduction system made of specialized muscle cells, not neurons, but these cells function similarly to nerve cells in that they generate and conduct electrical impulses to coordinate heartbeat.

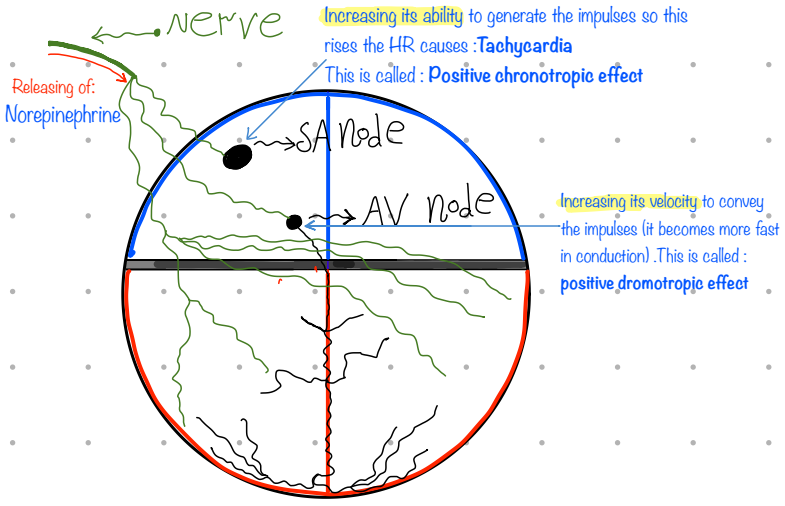
Autonomic nervous system

One of the functions of the ANS is : to control the rate of beating by its motor part and not excite it (Recall that : The heart is auto-stimulated)

Sympathetic ANS

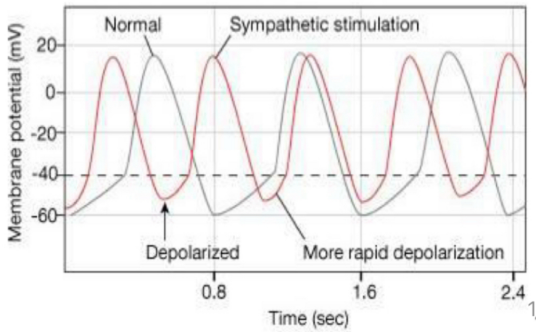
It is branching into :SA and AV nodes then in the atria and ventricles

It causes increase in the I funny and I (Ca++) so this leads to :



Increasing the contractility of the heart .This is called : **Positive inotropic effect**

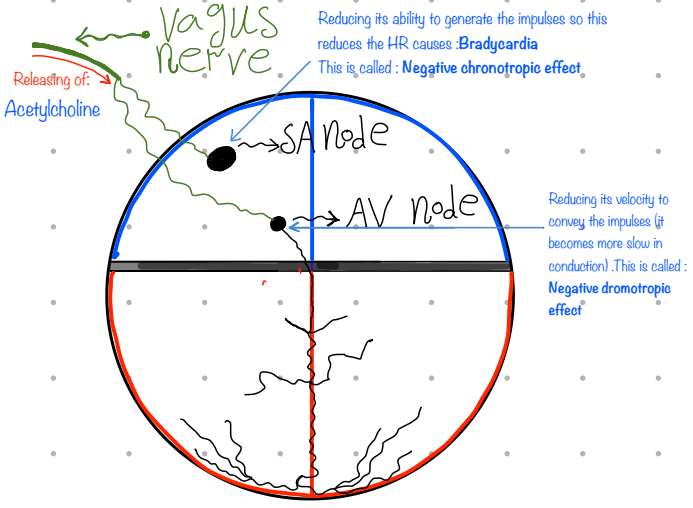
Noooote : The contractility (**inotrophy**) of the heart is coupled with the sympathetic ANS only



Parasympathetic ANS

It is branching into SA and AV nodes only

It causes reduce in the I funny and I (Ca++) so this leads to :



Note : Parasympathetic isn't involved in the inotrophy of the heart