Cardiac and Skeletal Muscles

Differences

Skeletal muscle	Cardiac Muscle
• Neurogenic (motor neuron-end plate-acetylcholine)	• Myogenic (action potential originates within the muscle)
• Insulated from each other	• Gap-junctions
• Short action potential	• Action potential is longer

Cardiac Muscle action potential Vs Skeletal Muscle.

Phase 0: –Depolarization phase (Na+ influx). The same in both

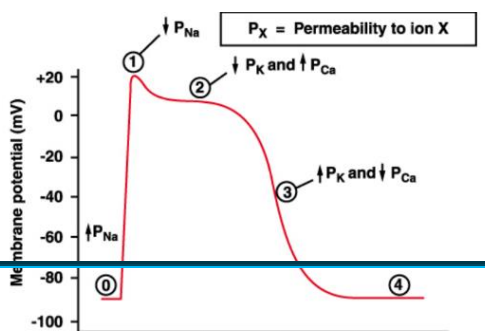
Phase 1: partial repolarization, opening of K+ channels. It is called "transient out "I_{to} " current" (Not in skeletal).

Phase 2 (very long duration): Plateau (depolarization not in skeletal) slow calcium channels Ca++ flow in, balancing K+ out flow.

Phase 3: fast repolarization phase (K+) repolarization. The same in both

Phase 4: resting membrane potential.The same in both

No plateau = no phase 1 and 2 as the myocytes



Conduction system composed of:

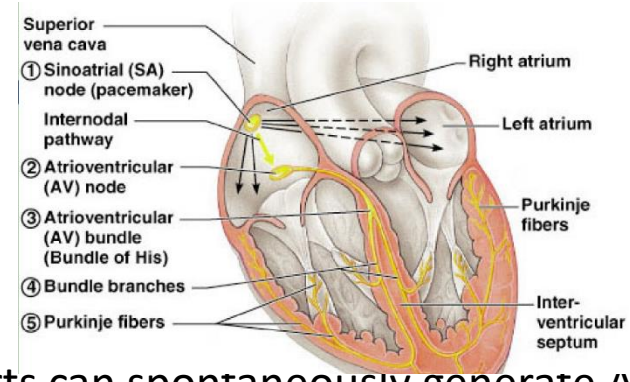
A-Sinoatrial (SA node) pacemaker

B-Atrioventricular node (AV)

C-Atrioventricular bundle (bundle of his)

D-Bundle branches

E-Purkinje Fibers.



Each of these parts can spontaneously generate APs at a given rate (they are Self-excitable) but the fastest of them is the SA node.

SA node is **the pacemaker** of the heart (it determines heart rate), because it is more leaky to Na+ more than any other cells in the heart.

Phase 4 slope of AV < SA node, so it takes longer time to reach thresholds. (Please, look at the note in the last page to clarify this point)

There is a conducting pathway from SA to AV node and both atria (From SA to left atrium through interatrial bundles. From SA to right atrium through three internodal pathways) which have very high conduction velocity to AV before it reaches threshold by itself.

AV node can not express itself, reach threshold or become pacemaker by itself, because it is over driven (suppressed by cell which has higher rate). The latent pacemakers undergo overdrive suppression. The tissue is suppressed because it is driven by other tissues. Tissue is suppressed if they are stimulated at high frequency (this include SA if receives electrical stimulus).

If we have sss (sick sinus syndrome), then the AV node will lead the heart so HR (heart rate) will be slower, almost 50 beat per minute, and we can survive with this.

If we cut AV bundle (complete AV block), we disconnected any communication between atrium and ventricles, so it become complete AV block, so the atrium will give 75 beats alone, while the ventricles be the pacemaker (30 bpm).

But 30 not enough to sustain cardiac output. This condition .necessitate the implantation of artificial pacemaker

Q = HR * SV, (SV volume blood ejected per beat)

Note: AV bundle, bundle branches, and Purkinje fibers, they are all leaky to Na+ but less leaky than SA and AV nodes. Thus, they take longer time to reach threshold, with AV node they are called latent pacemaker (hidden), they are suppressed by SA node.

We have two syncytia electrically separated by fibrous ring, the first one between the atrium (atrial syncytium), while the second one between the ventricles (ventricles syncytium). Both are **Functional Syncytia**

There are gap junctions (some sort of electrical synapses or **electrical windows**) which once you stimulate one cell the others will be stimulated in no time, they act as one cell.

Between atrium and ventricle there is no communication, they are separated by electrical insulator (fibers ring).

The only way both syncytia can communicate is via AV bundle.

There is no syncytium at the fibers in skeletal muscle, they act as separated “motor units” or motor unit as room lights; when you press a button, you turn 5 lights at once, another button will do another 5 lights, …etc. In contrast, the heart where there

is all or none, they all contact at the same time as one unit (in heart).

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Ventricle cells should not become pacemakers, the conductive pathway in the heart act as nervous system in heart, which have two function **excitation and conduction but not contraction.**
(same function of nervous system).

We call ventricular cell fast response action potential FRAP, while SA node slow response action potential SRAP (the depolarization slow that mean take along time to reach the peak).

Reach the peak to get complete depolarized in order to be able to depolarize your neighbouring cell, so we need two things, depolarized itself and electrical stimulation to contract in no time.

We don't want one part of the heart to contract while other part is relaxing, we need to contract as one unit, otherwise its inefficient contraction (it will not give you anything).

Phase zero in ventricle the slope is extremely high, it reaches depolarization very fast, fast Na+ channel work (have m gate and h gate), which works by positive feedback after stimulation, each Na+ leads more and more to influx. This happens very rapidly as the m gate opens.

(FRAP) Phase 4 stable with respect to time dv/dt is almost zero (slope). As we say to the ventricle: if you need depolarization, I will give you the order because you cannot reach threshold by yourself (cannot leak Na+ as SA/AV nodes), because if you do that, I will die. So, Na+ channels are completely close.
Stimulated by only an external stimulus (Purkinje cells).

-90 mV we need it to change the state of Na+ channels from closed inactive (as it is in -60 mV) to closed active, this cell in

ventricle once it stimulated it will utilize the fast Na+ channels they will bring the AP to the peak in no time.
Imagine that we force the SA node resting potential to increase (more negative) from -60 mV to -90 mV as in ventricular cells, by injecting it with Cl-, fast Na+ channels (channels responsible of driving the AP to the peak in no time) will open as it's stimulated.

Ectopic pacemaker: structures that can take the role of the normal pacemaker (SA node).

In case the ventricles start to be pacemaker, which causes extreme increase in the HR, so you lose the diastolic activity (ventricular fibrillation; deal with it by suppress it with a DC shock, or defibrillation=10,000 V)
If there is no transmission of action potentials from SA node to AV node, then there will be 2 pacemakers in the heart, one is the SA node, and another one (ectopic) for the ventricles as the electricity cannot reach them.

Function of heart is ejection (systole; 0.3 sec), to do that you must fill (diastole; 0.5 sec), if you want filling that means you need to relax if you not give it the chance to relax it will not fill (death).
The order is: relax, fill, contract, empty.

When multiple electrical stimuli are applied to a skeletal muscle at a sufficiently high frequency, twitches merge into higher force contractions, a process referred to as summation. Tetanus is the rigidity of muscle because of maximum summation. Tetanus is sustained contraction without relaxation. Due to the long action potential, cardiac muscle cannot be tetanized.

Sustained contraction without relaxes very painful in skeletal muscle , there are brown to tetanisation (means pain), because the AP is so short, and you can restimulate the muscle after 2ms. It is not life-threating in the lower limbs

Tetanisation for the heart means death because the sustained contraction of the heart means there is no diastolic activity. The presence of phase 2 prevent the tetanisation.

Extrinsic Innervation of the Heart:

In the medulla oblongata in CNS near to the neck.

Sympathetic ANS branch in: SA node, AV node, ventricle.

It releases of norepinephrine: increase funny current (If+) and calcium current (Ica++), increase slope for phase 4 so it reaches the threshold faster, so you shrink the cardiac cycle duration (decrease the duration), so it increases the HR (Tachycardia). This is called Positive Chronotropic effect.

Positive Chronotropic effect (increased rate of sinoatrial node or SA node discharge = increased heart rate).

Dromotropic effect (increase conduction velocity through the AV node).

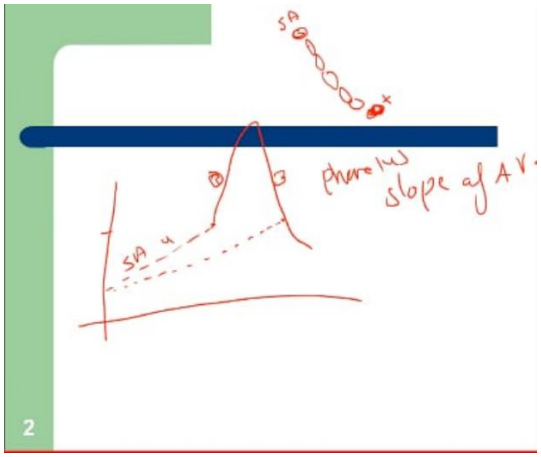
Inotropic effect: by increasing calcium current (Ica++), thus, increasing the contractility or the force of contraction in the ventricle. Acting especially on the phase 2. Increasing stroke volume

Parasympathetic ANS branch in: SA node, AV node. By Vagus (cranial tenth; X) nerve.

It releases of acetylcholine: decrease increase funny current (If+) and calcium current (Ica++) and increase K+ outflux, so decrease HR (with no effect on contractility) = negative chronotropic and dromotropic (by decreasing conduction velocity through the AV node effects.

HR if it is low or high both will be very bad.

VERY IMPORTANT NOTE: Please be careful that when we said: "Phase 4 slope of AV < SA node, so it takes longer time to reach threshold", we meant the phase 4 of the SA/AV node action potential (the first picture below), not the cardiac muscle (ventricular) potential (the second picture below).



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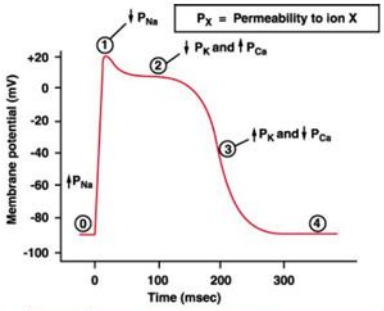

Phase	Membrane channels
0	Na^+ channels open
1	Na^+ channels close
2	Ca^{2+} channels open; fast K^+ channels close
3	Ca^{2+} channels close; slow K^+ channels open
4	Resting potential

During plateau happens at +10 mV, where the h gate (the inactivation gate) is the one the closes the Na+ channels. That means these channels will remain close even a strong stimulus is applied, thus, cannot start a second AP. It is a refractory period. This is why it prevents heart tetanisation.

Note that the negative relationship between HR and stroke volume. That makes the ventricular fibrillation very dangerous (which increases the HR as we said).

Considering what was said above, the relationship between the heart rate (HR) and cardiac output (Q) follows the bell-shaped curve below: