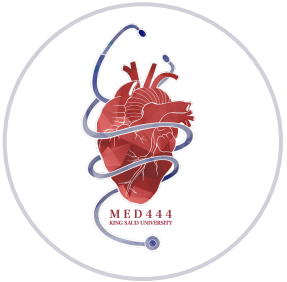
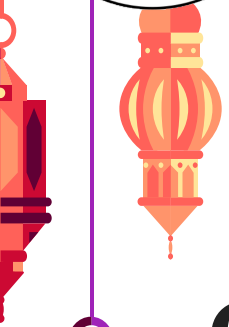




قبارك عليكم شهر رمضان

Contractile mechanism in cardiac muscle

Editing File

Main text Female's slide Male's slide Important text Doctor's note Extra

Objectives:

- Define cardiac muscle contractility & types of its contraction.
- Understand the physiology of cardiac muscle
- Understand the phases of cardiac action potential and the ionic basis
- Identify the refractory period of cardiac muscle
- Discuss the role of ca+2 in the regulation of cardiac muscle function
- Describe the mechanism of excitation contraction coupling
- Discuss factors affecting cardiac contractility

Dr Nagi
First two videos for this lecture

Female's slide

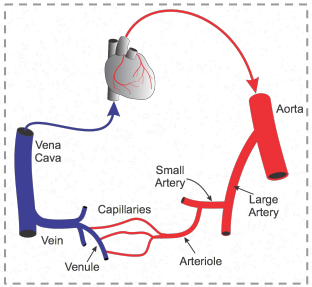
Cardiovascular system

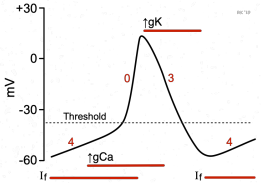
CVS

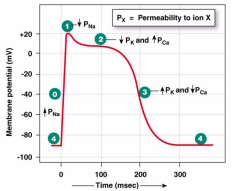
Cardio = Heart
Vascular = blood vessels

- Vascular system (Blood vessels)
  - Peripheral circulation and blood flow
- Heart (Cardiac muscle)
  - Lec 2
  - Electrical activity
    - Sinoatrial SA node (Action potential)
    - Actually contract
    - Ventricular muscle action potential
  - Mechanical activity

MECHANICAL ACTIVITY IS THE CONTRACTION AND RELAXATION OF THE CARDIAC MUSCLE, IT'S DRIVEN BY ELECTRICAL ACTIVITY.





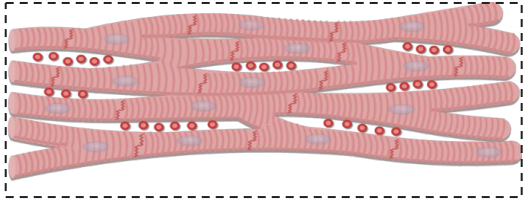


Physiologic Anatomy of Cardiac Muscle

Cardiac muscle cells are found only in the heart, are specialized to pump blood powerfully and efficiently throughout our entire lifetime.

Contractility describes the relative ability of the heart to eject a stroke volume.
Stroke volume: pump blood per contraction the amount of blood ejected in one contraction or per beat not per minute !

Characteristics of cardiac muscle tissue cells:



1. Involuntary
2. Striated
3. Intrinsically controlled
4. Branched and single centered nuclei.
5. Gaps junctions within intercalated discs
6. Abundant mitochondria

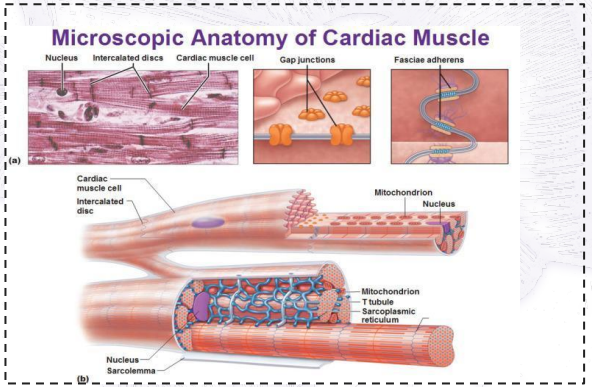
Important slide

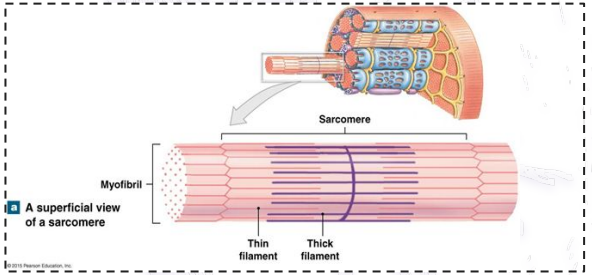
Female's slide

Physiologic Anatomy of Cardiac Muscle

Myofibril	Myofibrils are thread-like structures found in muscle cells that are responsible for muscle contraction. Each myofibril contains a series of repeating units called sarcomeres, which are the basic functional units of muscle.
Myofilaments	Myofilaments are the smaller subunits that make up myofibrils. Specifically, myofilaments refer to the individual actin and myosin protein filaments that interact to produce muscle contraction
Sarcomere	the smallest functional contractile unit of a muscle fiber, a compartment of myofibrils.
Sarcolemma	muscle fiber plasma (cell) membrane.
Sarcoplasm	The muscle fiber cytoplasm
Sarcoplasmic Reticulum	smooth endoplasmic reticulum (SR)
Transverse tubules	Tubules formed by invaginations of the sarcolemma and flanked by the sarcoplasmic reticulum. light blue, Very important for contraction

Sacro = muscle
lemma=sheath





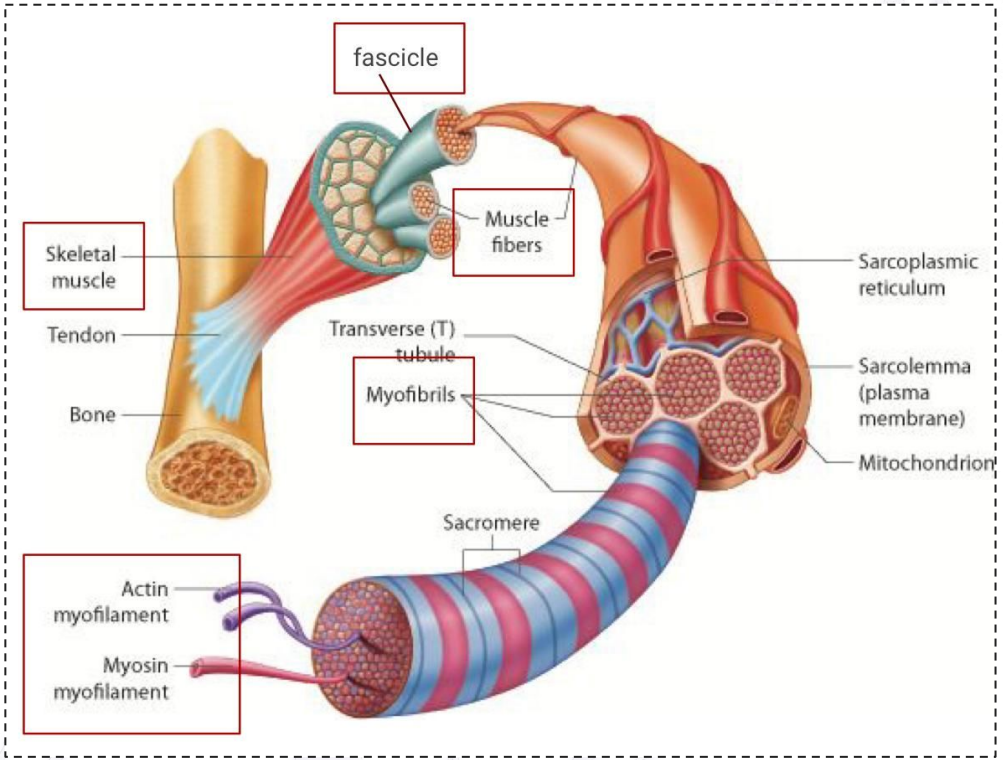
Extra slide

Physiologic Anatomy of Cardiac Muscle

Special thanks to team 443

Layers sequence :

1. Muscle
2. Fascicle
3. Myofiber (muscle fiber)
4. Myofibrils
5. Myofilament
   - Actin myofilament
   - Myosin myofilament



Physiologic Anatomy of Cardiac Muscles

Fibers are Branched and connected at intercalated discs.

Discs contain several Gap Junctions

Nuclei are centrally located

Abundant Mitochondria

Why? Because the heart is working without resting

What's the difference between cardiac and skeletal muscle ?

Atrial and ventricular muscle contract in much the same way as skeletal muscle,except that duration of contraction is much longer in cardiac muscle so refractory period is longer.

Sarcoplasmic Reticulum is less abundant than in skeletal muscle, but greater in density than smooth muscle

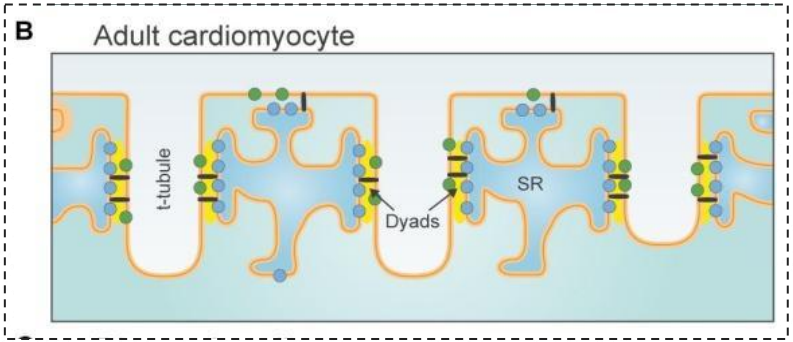
Sarcolemma has specialized ion channels (-ve voltage-gated Ca2+ channels) that skeletal muscle does not have.

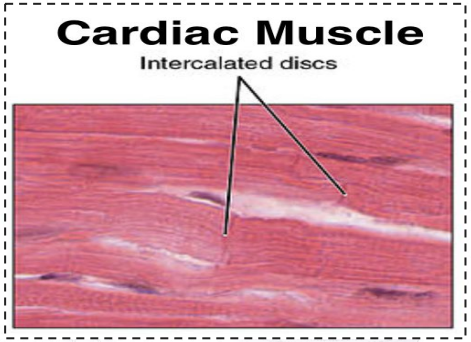
Fibers are not anchored at ends; allows for greater sarcomere shortening and lengthening

Important slide

Physiologic Anatomy of Cardiac Muscle

Dr: You may read to remind yourself it might be asked about since it's in the slides





FEATURES OF DIFFERENT TYPES OF MUSCLE

Table III-2-1. Histologic Features of Skeletal, Cardiac, and Smooth Muscle

Skeletal	Cardiac	Smooth
Striated	Striated	Nonstriated
Actin and myosin form sarcomeres	Actin and myosin form sarcomeres	Actin and myosin not organized into sarcomeres
Sarcolemma lacks junctional complexes between fibers	Junctional complexes between fibers including gap junctions	Gap junctions
Each fiber innervated	Electrical syncytium	Electrical syncytium
Troponin to bind calcium	Troponin to bind calcium	Calmodulin to bind calcium
High ATPase activity (fast muscle)	Intermediate ATPase activity	Low ATPase activity (slow muscle)
Extensive sarcoplasmic reticulum	Intermediate sarcoplasmic reticulum	Limited sarcoplasmic reticulum
T tubules form triadic contacts with reticulum at A-I junctions	T tubules form dyadic contact with reticulum near Z lines	Lack T tubules, SR controlled by second messengers
Surface membrane lacks calcium channels	Voltage-gated calcium channels	Voltage-gated calcium channels

Cardiac Muscle as a Syncytium

syncytium = unit
1- first unit is both atria
2- second unit is both ventricles

Intercalated discs:
are the dark areas crossing the cardiac muscle fibers, they are actually cell membranes that separate individual cardiac muscle cells from one another.

There are two kinds of membrane junctions Within intercalated discs:

1. Desmosomes (anchoring):Also called adherens junction, are important for mechanically coupling and reinforcing cardiomyocytes.

2. Gap junctions Essential for rapid electrical transmission between cells.

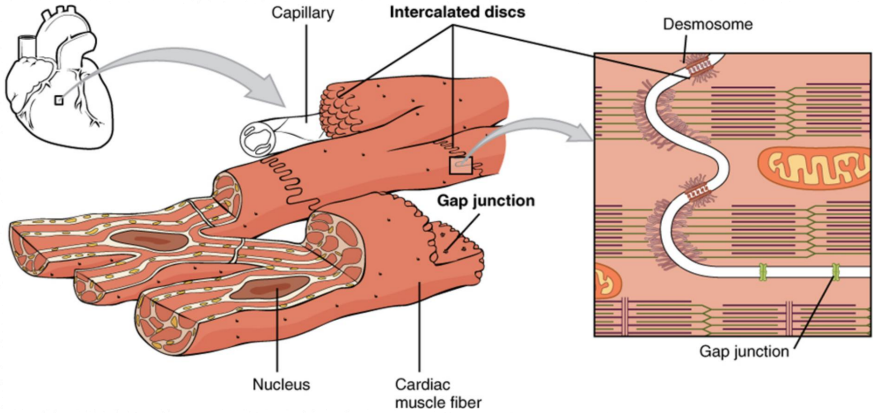
Membranes fuse with one another, and form permeable "communicating" junctions (gap junctions) which allows :

1. Action potential pass easily
2. Formation of functional syncytium
3. Allow free diffusion of ions

Cardiac Muscle as a Syncytium

Ions move with ease in the intracellular fluid along the longitudinal axes of the cardiac muscle fibers, so that action potentials travel easily from one cardiac muscle cell to the next. Thus, cardiac muscle is a syncytium of many heart muscle cells, action potential spreads to all of them.

How do gap junctions within intercalated disks aid contraction of the heart?
They allow impulses to spread from one cardiac muscle cell to another, allowing sodium, potassium, and calcium ions to flow between adjacent cells, propagating the action potential, and ensuring coordinated contractions.



Important slide

Cardiac Muscle as a Syncytium

Heart is composed of two syncytium Separated by fibrous tissue:
1. Atrial 2. Ventricular

What's the importance of this separation?
Allows atria to contract ahead (before)of ventricles.

How do action potentials reach ventricles?
Action Potentials are conducted by A-V bundle.

Nodal cell do not contract, because it conduct APs

Physiology Cardiac muscle

Important slide

Special thanks to team 443

Types of cardiac muscle cell

Autorhythmic cells

Do not contract
Generate APs
Specialized excitatory and conductive fibers

Automatic

rhythmical electrical discharge in form of APs (SA node)

Conduction

Transmit APs through heart (conductive fibers)

They specialize in initiating and conducting action potentials that are responsible for the contraction of Myocytes, rather than directly contracting themselves. Their weak contraction is due to the limited number of contractile fibers they contain.

Contractile cells

Atrial

Ventricular

99% of cardiac muscle cells, they function in mechanical work of pumping.

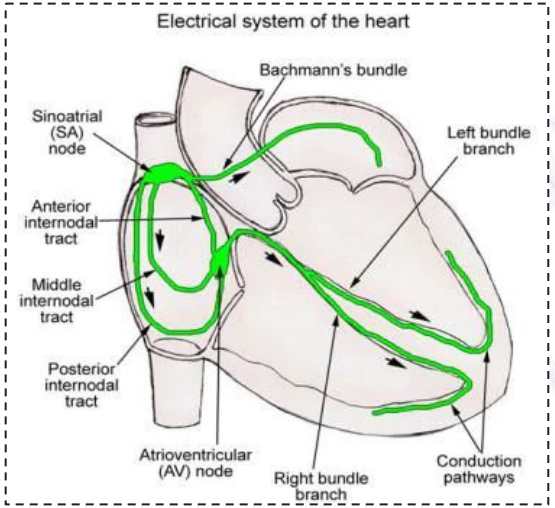
Important slide

Cardiac muscle properties

The cardiac muscle cells are responsible for electrical stimulation which leads to mechanical function. The Electrophysiological properties of cardiac muscles are shown below :

Dr: Heart does not need brain to work
modified by nerves system & endocrine system

Automaticity	Ability to spontaneously generate an electrical impulse.
Excitability	Ability to respond to an electrical impulse.
Conductivity	Allow transmission of electrical impulse to another cardiac cell.
Contractility	Ability to contract after electrical impulse response
Rhythmicity	Ability to send electrical impulses in a regularly manner



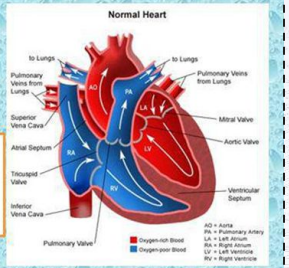
Primary function of cardiovascular system:

1. Deliver blood to and from tissues:
   - Providing essential nutrients O_2 to cells for metabolism.
   - Removing waste products.

2. The heart muscle is remarkable. At an average heart rate of 70 beats/min the physiological average is from 60 to 100 beats/min, the heart needs to contract and relax more than 100,000 times a day without stopping or tiring.

General function

- **The main function of the cardiovascular system is to deliver oxygen and nutrients and remove the waste products of metabolism.**
- **Each part of system has a specific function in helping to do this.**



- Heart
  - Pump – It is designed to push blood out through the arteries.
    - One ventricle pumps blood to the lungs to get oxygen
    - The other pumps blood to the body to deliver Oxygen and nutrients
- Blood vessels
  - Designed to carry the blood. They are hollow, and vary according to their purpose.
    - For example, arteries have thick muscular walls so that they can take the blood at high pressure from the heart, and squeeze it along to the capillaries
- Blood
  - Has a number of functions
    - Red blood cells and their haemoglobin attract Oxygen, so it can be carried around in the blood
    - White blood cells fight infection
    - Platelets help with clotting if we are injured.

Dr:

- Heart is the only muscle that should not go bigger because it will be heart failure.
- The arteries are deep, thick wall and carry oxygenated blood from the left ventricle.
- If the wall was very thick it will cause hypertension and if very thin will cause bleeding.
- The veins are superficial, thin elastic wall and carry non-oxygenated blood and has valves that direct the blood in one direction into the right atrium and then right ventricle.

لقدْ خَلقْنَا الْإِنسَانَ فِي أَحْسَنِ تَقْوِيمٍ

Cardiac muscle mechanics

Cardiac muscle goes through the same series of isometric and isotonic events as skeletal muscle. Therefore, the cardiac cycle (Heart beat) has two phases which are considered to be :

1

Isometric (Isovolumic contraction, and Isovolumic relaxation) contraction in elastic fiber without shortening in the length
Isometric Contraction: Muscle contraction without significant shortening or change in distance.

2

While all the other phases are considered to be isotonic in nature.
Isotonic Contraction: Muscle contraction without significant change in force of contraction.

Muscle Contraction

	Isometric contraction	Isotonic contraction
Length of the muscle	Remains same	Shortening of the muscle
Tension	↑ during the contraction	No change
Mechanism	Sarcomere which shorten do so by stretching those which do not	Shortening of individual sarcomeres adds up to the shortening of the whole muscle
External work	No external work down	Work down
Example	Trying to lift heavy weights (when the weights are not actually lifted)	Lifting of weights

Action potential in cardiac muscles

The ionic basis for the APs in the ventricles, atria, and Purkinje system are identical. The AP in these tissues shares the following characteristics:

1. Long duration
2. Stable resting membrane potential
3. Plateau

Heart beats rhythmically as result of action potential, it generates by itself (Auto-rhythmicity).

Important slide

Female's slide

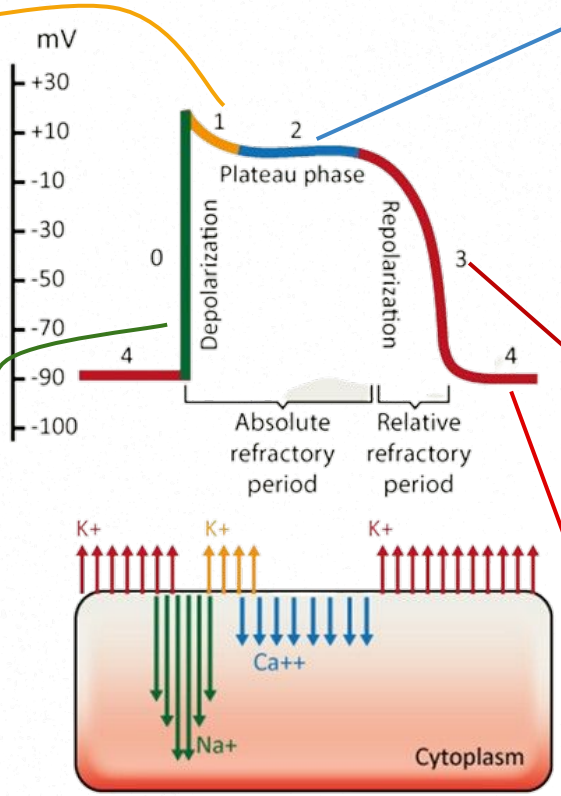
AP in cardiac muscles (ventricle)

Phase 1:

Early repolarization, K+ efflux, slow This phase occurs immediately after phase 0, and is characterized by a brief period of repolarization due to the closure of the Na+ channels and the transient opening of voltage-gated potassium channels (K+).

Phase 0:

Major phase Depolarization, fast Na+ influx, This phase is message for contraction. This phase is initiated by the influx of sodium ions (Na+) into the cell through voltage-gated Na+ channels, which causes a rapid depolarization of the membrane potential.



Phase 2:

Plateau =0.2 sec, slow Ca+2 channels open causing Ca+2 influx,the major cause of plateau is k+ out and Ca+2 in This phase is a prolonged period of depolarization that occurs due to the opening of voltage-gated calcium channels (Ca2+) and the slow influx of Ca2+ ions into the cell, balanced by the efflux of K+ ions. This plateau phase is unique to cardiac muscle and helps to maintain the prolonged refractory period necessary for the heart's proper function.

Phase 3:

Repolarization: K+ efflux, This phase occurs when the Ca2+ channels close and the K+ channels remain open, leading to the efflux of K+ ions from the cell and the return of the membrane potential to its resting state. **Rabidly message for relaxation.**

Phase 4:

Resting, K+ efflux, continuous release of k+This is the stable resting potential of the cardiac muscle cell, which is maintained by the activity of ion pumps and ion channels.**=-90**

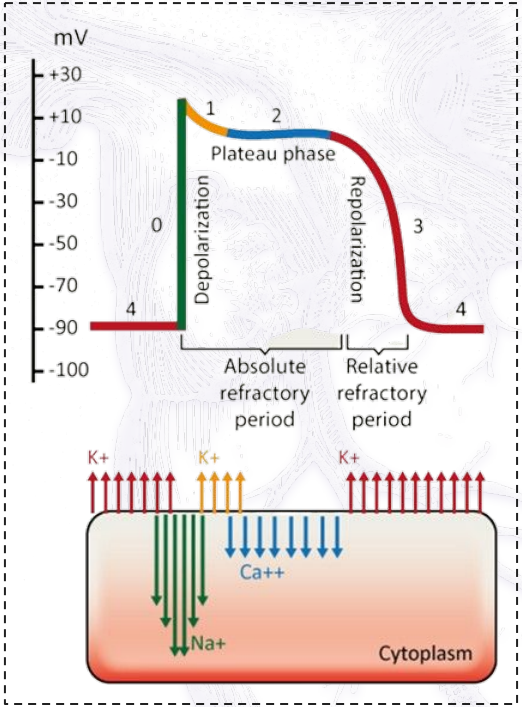
Plateau

Why plateau phase is critical to cardiac muscle function ?

Dr: To prevent too much excitation
To prevent another impulse of contraction to make the heart has the chance to pump the blood out.

1. It causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle.

2. It prevents additional impulses from spreading through the heart prematurely, thereby allowing the muscle sufficient time to contract and pump blood effectively.



What causes the plateau?

Prolonged opening of the slow calcium-channels allows calcium to enter, cause plateau. Ca+2 in and k+out

Important slide

Female's slide

How do we get this cell to rest?

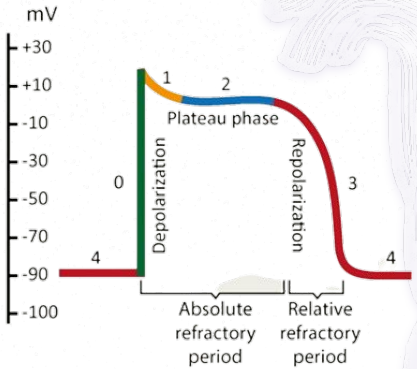
At the end of the plateau:

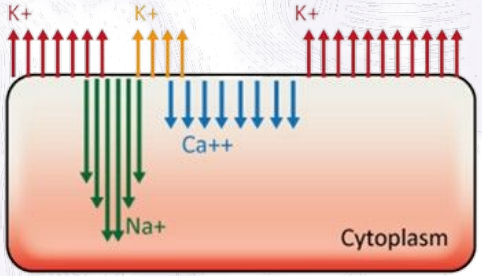
Ca+2 inside cell needs to go back to sarcoplasmic reticulum and also outside the cell.
One way to get the Ca+2 back into the sarcoplasmic reticulum is via ATP channels that pump in Ca+2 against concentration gradient and H+ out.

Other channels that do the same here is Na+/Ca+2 channel via secondary active transport as Na+ moves out from sarcoplasmic reticulum down gradient and Ca+2 against gradient.

Same two channels are used to get Ca+2 outside cell ...which all prevent contraction.
The two channels are secondary active transport

Extra:
What is the difference between primary and secondary active transport?
the main difference between primary and secondary active transport is that primary active transport directly uses ATP to move molecules across the membrane, while secondary active transport uses the energy stored in the concentration gradient of one molecule to transport another molecule against its concentration gradient.





Extra

- By Ca+2 leaves, K+ gets out aggressively (move out more).
- Phase 3 is just K+ channels efflux
- graph potential trace will show more dropping by then beyond -60 mv until it gets to resting membrane potential-90, then it will stay rested there for a bit until ions from nodal cells via gap junctions leak so it goes up to resting potential.

Important slide

Excitation-Contraction coupling

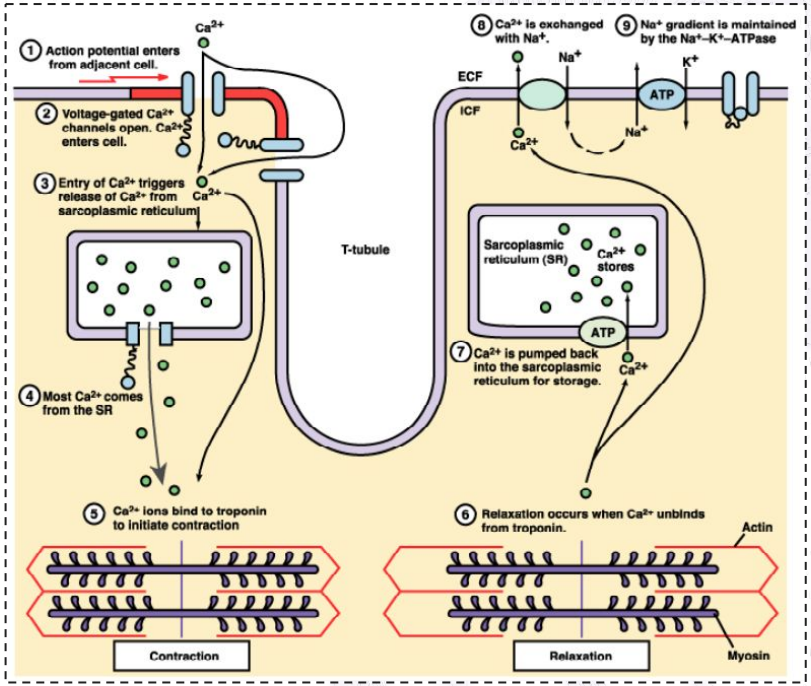
Excitation (AP)

Excitation of the heart is triggered by electrical (ionic) impulse rather than neurotransmitters.

Increase Ca++

Contraction shortening

Contraction of the heart is triggered and regulated by elevation of intracellular calcium influx.

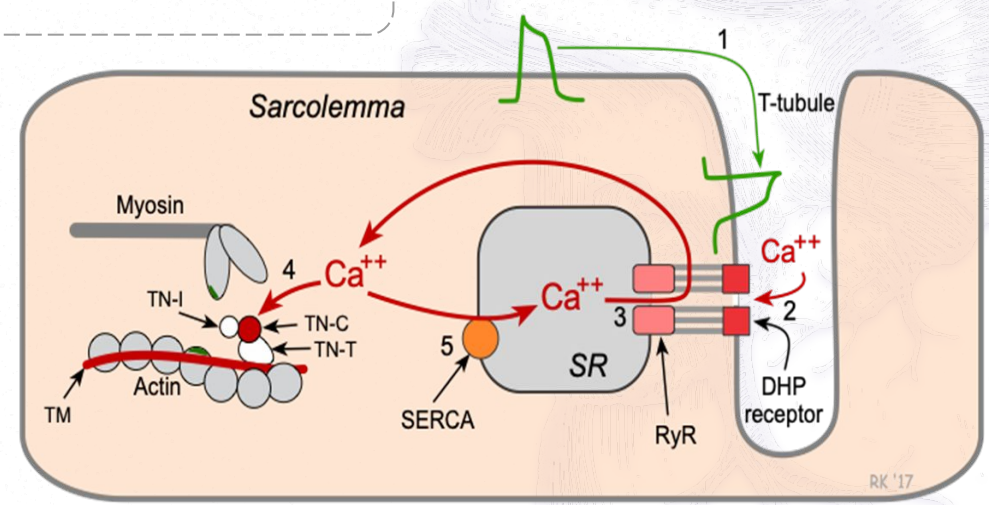


Very Important slide !!!!!

Excitation-Contraction coupling

Cardiac muscle fibers contract via excitation-contraction coupling, using a mechanism unique to cardiac muscle called calcium induced calcium release (CICR).

Calcium-induced calcium release involves the conduction of calcium ions into the cardiomyocyte, triggering further release of ions into the cytoplasm.



Entry of extracellular Ca+2 causes the release of Ca+2 from the sarcoplasmic reticulum (calcium-induced calcium release), source of about 95% of calcium in cytosol.

Steps in next slide

Important slide

Excitation-Contraction coupling

How does contraction occur? Steps for excitation-contraction coupling:

1. AP is initiated in cell membrane, and depolarization spreads to interior of cell There are invagination in the cell called T Tubule which has ve+ charge due to presence of Ca+2 there. So Ca+2 can flow in from there to cell as its concentration is high in T-tubule. Ca+2 enters cell via L-type Ca+2 channels located at the plasma membrane (sarcolemma) then activates Ca+ sensitive receptor located in sarcoplasmic reticulum called Ryanadine receptor-2.

2. Entry of Ca+2 (and can either bind to calmagulin or by itself) triggering the release of more Ca+2 from SR through ryanodine receptors-2 (RYR-2) we call this phenomenon **(Ca+2 induced Ca+2 release)**.

3. When RYR-2 opens, large amounts of Ca+2 are released from SR increases intracellular Ca+2 into the sarcoplasm. Ca+2 then binds to a special protein known as troponin, which is composed of three subunits: troponin I(where actin bind), troponin T(where tropomyosin bind), and troponin C (where Ca2+ binds). Troponin C specifically binds to Ca+2, causing it to change the conformation (shape) of troponin T, which is connected to tropomyosin (because it pulls on troponin T as it binds to troponin C because tropomyosin C is attached to tropomyosin T). This change in shape of tropomyosin removes any impediment to the binding of actin and myosin. This allows the myosin head to bind to actin, resulting in muscle contraction. The influx of Ca+2 into the sarcoplasm increases the number of cross-bridges formed between actin and myosin, leading to more contraction creating a pump action to squeeze blood.

4. **Relaxation** occurs when Ca+2 is re-accumulated in SR by **Ca+2 ATPase (SERCA**, sarco-endoplasmic reticulum calcium-ATPase).

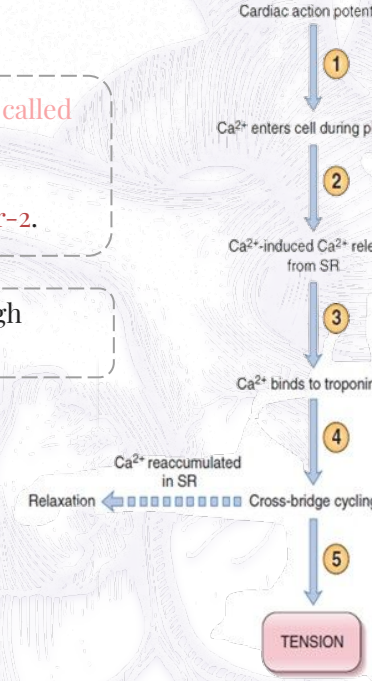


Figure 4–18 Excitation-contraction coupling in myocardial cells. See the text for an explanation of the circled numbers. SR, Sarcoplasmic reticulum.

Dr: Important picture!

Summary of Excitation Contraction coupling

1. Ca^{+2} enters the cell during depolarization and triggers release of Ca^{+2} by terminal cisternae.
2. Ca^{+2} binds to TN-C, inducing a conformational change in the troponin complex.
3. Myosin heads bind to actin, leading to cross-bridge movement (requires ATP hydrolysis) and reduction in sarcomere length.
4. Ca^{+2} is re-sequestered by sarcoplasmic reticulum by the SERCA pump.
5. Ca^{+2} is removed from TN-C, and myosin unbinds from actin (requires ATP); this allows the sarcomere to resume its original, relaxed length.

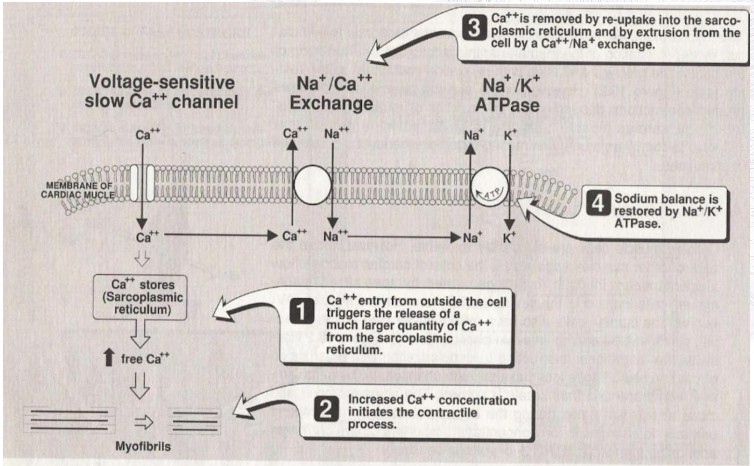


Figure 16.3
Ion movements during the contraction of cardiac muscle.

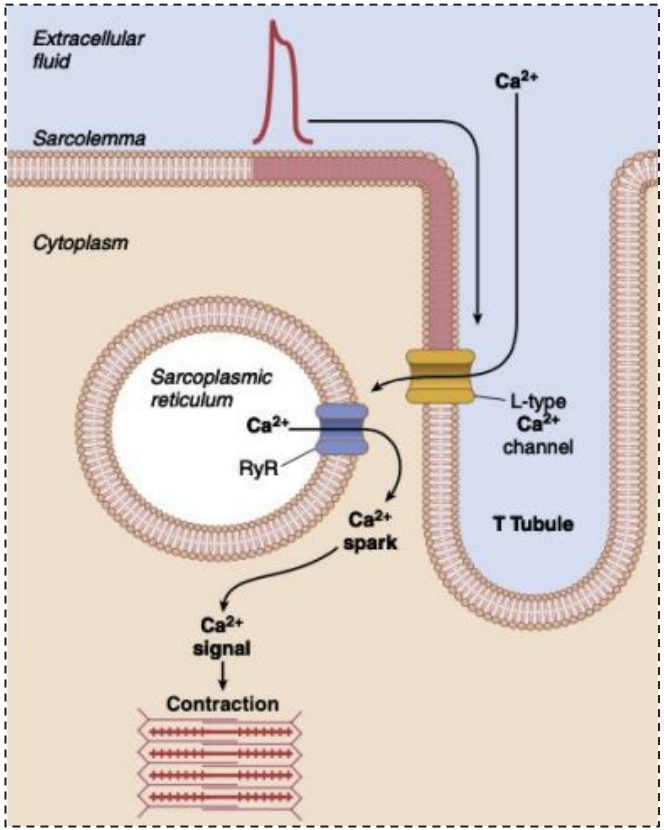
The SR cisternae are flattened, membrane-bound sacs within the sarcoplasmic reticulum that store and release calcium ions during muscle contraction. When an action potential is triggered in a muscle cell, it causes the release of calcium ions from the SR cisternae into the muscle cell, which leads to muscle contraction. After the contraction is complete, the calcium ions are actively pumped back into the SR cisternae, which allows the muscle cell to relax.

Importance of Ca+2 from T tubules

Without Ca+2 from T tubules, strength of cardiac muscle contraction would be reduced considerably because:

The sarcoplasmic reticulum is less well developed than that of skeletal muscle and does not store enough Ca^{+2} to provide full contraction. Therefore, T tubules of cardiac muscle have a diameter five times as great as skeletal muscle tubules.

Inside T tubules is a large quantity of muco-poly-saccharides that are electro-negatively charged and bind an abundant store of Ca+2 keeping Ca+2 available for diffusion to interior of cardiac muscle fiber when a T tubule AP appears



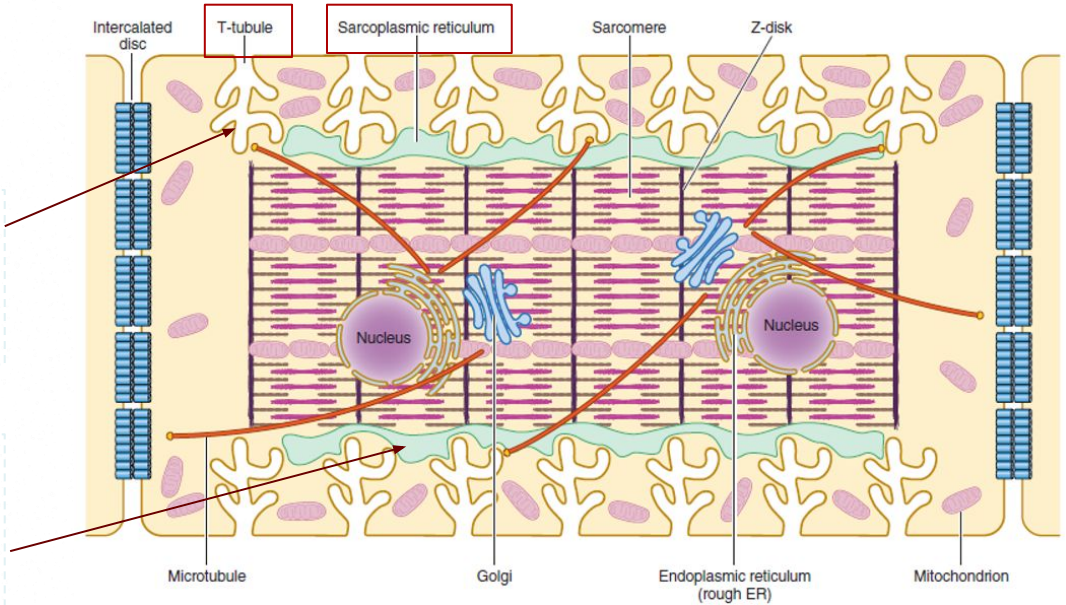
Important slide

Female's slide

illustration of the internal structures of an adult ventricular cardiomyocyte

T-tubules, which are enriched =Rich with voltage-gated L-type calcium channels.

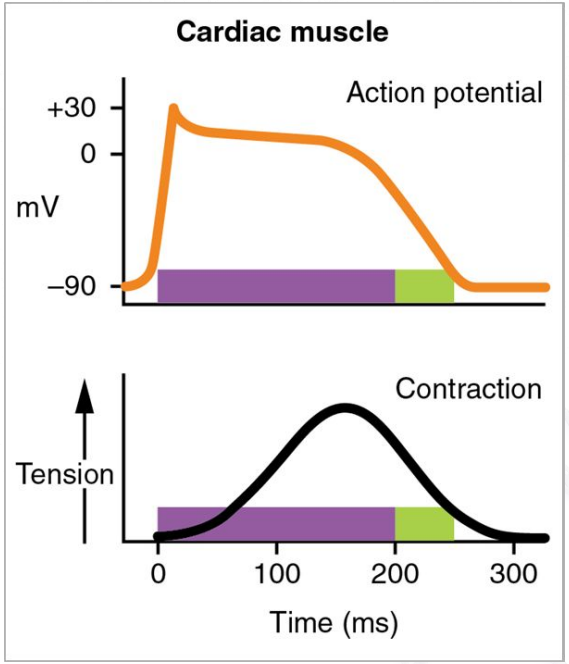
They are positioned closely near the sarcoplasmic reticulum, the primary internal calcium store.



Duration of contraction

Cardiac muscle begins to contract a few millisec after AP begins and continues to contract until a few millisec after AP ends.

The duration of contraction of cardiac muscle is mainly a function of the duration of AP (about 0.2 sec in atrial muscle and 0.3 sec in ventricular muscle).



Factors regulating contractility (inotropy)

Mechanisms for Changing Contractility:

Contractility correlates directly with the intracellular Ca^{+2} concentration.

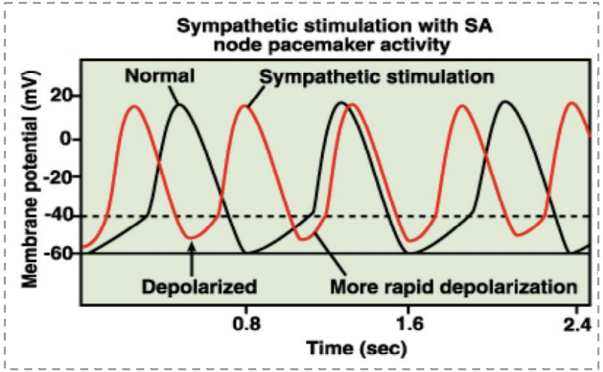
Therefore, the larger the inward Ca^{+2} current and the larger the intracellular stores, the greater the increase in intracellular Ca^{+2} concentration and the greater the contractility.

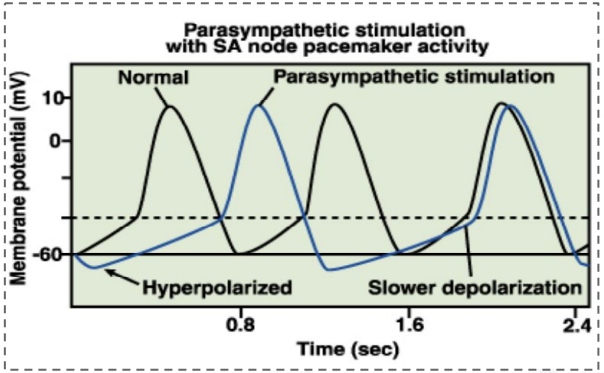
(قوة الانقباضه تعتمد على Ca^{+2})

Autonomic nervous system modulates the frequency of depolarization of pacemaker.

Sympathetic stimulation (neurotransmitter); binds to b1 receptors on the SA nodal membranes.

Parasympathetic stimulation (neurotransmitter); binds to muscarinic receptors on nodal membranes; increases conductivity of K+ and decreases conductivity of Ca^{+2}





Effects of Autonomic nerve activity on the heart:

Region Affected	Sympathetic Nerve effect	Parasympathetic Nerve effect
SA node Generate contraction	Increase rate of diastolic depolarization; increase cardiac rate (+chronotropic)	Decrease rate of diastolic depolarization; decrease cardiac rate (-chronotropic))
AV node	Increase conduction rate (+dromotropic)	Decrease conduction rate (-dromotropic)
Atrial muscle	Increase strength of contraction (+ inotropic)	Decrease strength of contraction (-inotropic)
Ventricular muscle	Increase strength of contraction (+inotropic)	No significant effect

Difference between action potential of cardiac and skeletal muscle

Dr: You can read this

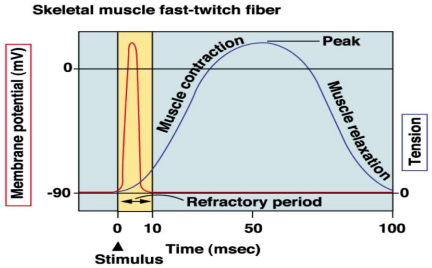
AP in Skeletal muscle	AP in Cardiac muscle
Muscle AP is caused by sudden opening of large numbers of fast Na+ channel .	AP is caused by opening of 2 channels: **1.** fast Na+ channels (same as in skeletal muscle) **2.** slow Ca^{+2} channels (also called Ca^{+2} -Na+ channels or long lasting).
At end of closure, repolarization occurs due to K+ efflux, and AP is over within a thousandth of a second.	After onset of AP, membrane permeability for K+ ↓fivefold (does not occur in skeletal muscle). Decreased K+ permeability↓ out flux of K+ during plateau and prevents return of AP voltage to resting level. When slow Ca^{+2} channels close, K+ permeability ↑ rapidly returning membrane potential to resting level, ending AP.

Difference between action potential of cardiac and skeletal muscle

AP start at first

AP in Skeletal muscle

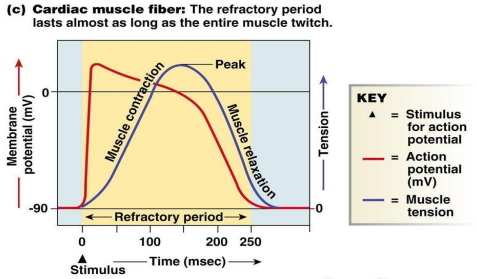
- In skeletal muscle, duration of AP is shorter than its mechanical response.
- This means that skeletal muscle can undergo tetanus via repeated stimulation.



AP in Cardiac muscle

- In cardiac muscle, duration of AP is same as duration of its mechanical response.
- This means that cardiac muscle cannot undergo tetanus via repeated stimulation.

Cardiac muscle fibre: the refractory Period lasts almost as long as the Entire muscle twitch



Q DR: Why cardiac muscle cannot have repeated excitation like skeletal muscle?

REFRACTORY (RESISTANT) PERIOD OF CARDIAC MUSCLE

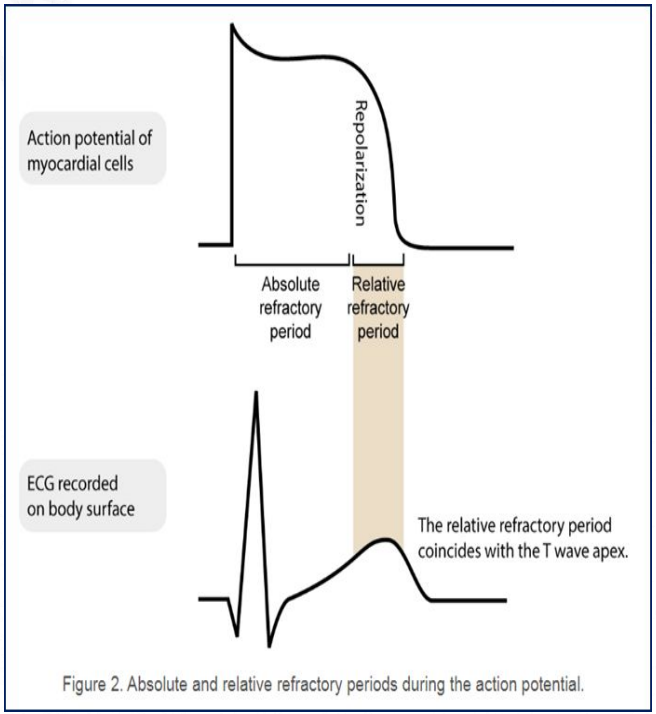


Figure 2. Absolute and relative refractory periods during the action potential.

Dr: Numbers are very important

Absolute Refractory period (ARP) of ventricle:

0.25 -0.30 sec (signal cannot re-excite an already excited area of cardiac muscle)

في هذه الفتره ماراح يكون فيه AP فيه اشاره للعضله

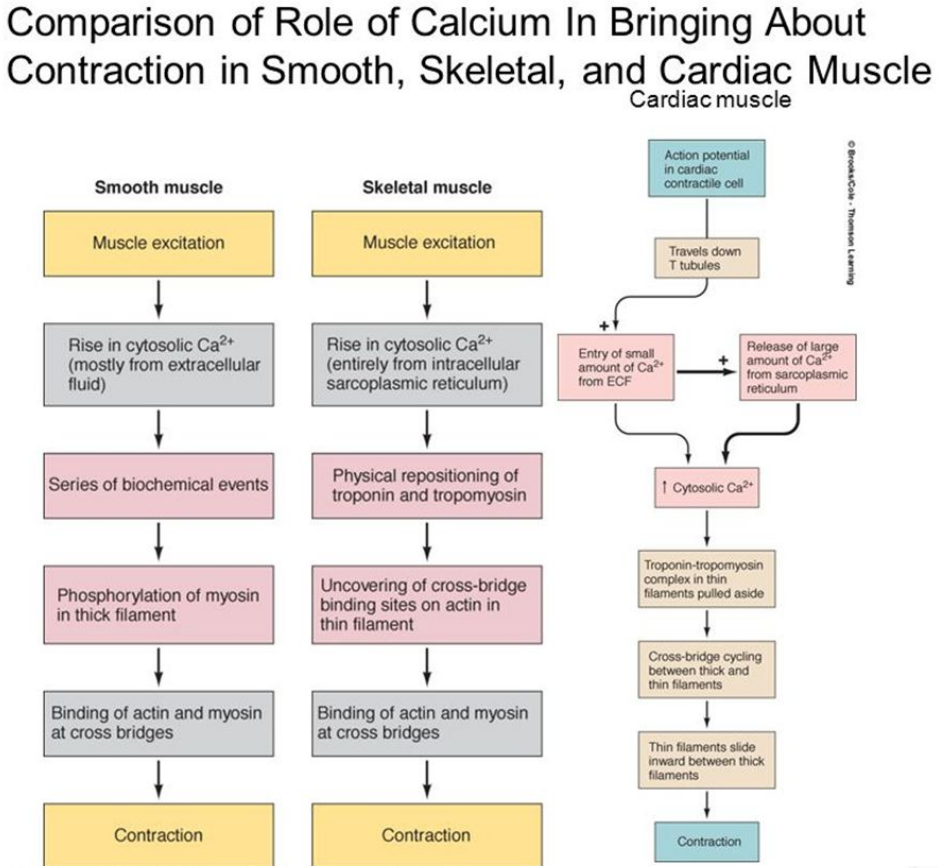
Why we need refractory period?

The ARP acts as a protective mechanism in the heart

Relative refractory period (RRP) of ventricle:

0.05 sec (muscle difficult to excite but can be excited by a strong signal "premature" contraction) not impossible

Cardiac vs skeletal



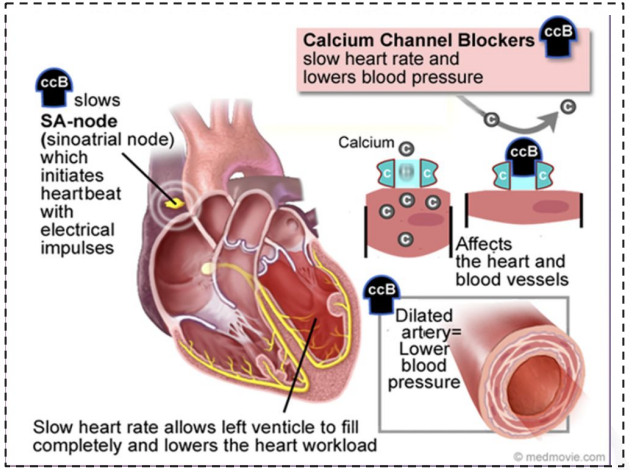
Clinical note: Calcium channel blockers

- Calcium Channel Blockers are drugs that block the movement of calcium into heart and blood vessel muscle cells, which can decrease the pumping strength of the heart and relax blood vessels.

To ↓ Ca++ so heart relax

- This causes the muscles to relax, lowering blood pressure, slowing the heart rate and decreasing oxygen demands of the heart.

- They are used to treat high blood pressure and chest pain (angina) caused by reduced blood supply to the heart muscle, as well as some abnormal heart rhythms (arrhythmias).



Angina is a type of chest pain or discomfort that occurs when the heart muscle doesn't receive enough oxygen-rich blood. It is usually caused by narrowed or blocked coronary arteries, the blood vessels that supply blood to the heart muscle.

When the heart muscle needs more oxygen than the narrowed or blocked arteries can supply, it can cause a temporary imbalance between the oxygen demand and supply. This results in a buildup of waste products in the heart muscle, which leads to pain, discomfort, or pressure in the chest, commonly referred to as angina.

team443

Check here for our summary
Highly recommended !!!!!

Sorry but if you will not cheack it راحت عليك المليون

MCQs:

Answers

For more question check our summary file!

1/A
2/C
3/A

1 Which of these contractions shorten the muscles and preserve its tension?

A	Isotonic	B	Isovolumic	C	Isometric	D	Isotension

2 A spontaneous generation of electrical impulses, known as?

A	Conductivity	B	Contractility	C	Automaticity	D	Excitability

3 What is the channel responsible for the plateau?

A	Opening of the Ca++ channel	B	Closing of the Ca++ channel	C	Opening of the Na+ channel	D	Opening of the Mg+ channel

MCQs:

Answers

For more question check our summary file!

4/A
5/A
6/C

4 what will happen at the absolute refractory phase:

A	No excitability due to complete depolarization even by a suprathreshold stimulation	B	Excitability can be achieved with strong stimulation	C	SA Node depolarizes continuously	D	Conduction is increased

5 What prevents cardiac muscle cells from tetanization?

A	Prolonged absolute refractory period	B	Short plaque period	C	Slow Ca influx channels	D	Rapid depolarization

6 The most abundant of SR is:

A	Cardiac muscle	B	Smooth muscle	C	Skeletal muscle	D	A&B

SAQ

What causes plateau?

Prolonged opening of the slow calcium-channels allows calcium to enter.

What are the two receptors or channels that get the cell to rest?

1- ATP channel that pump Ca+2 into SR and H+ proton out.
2-Na+/Ca+2 channel that moves Na+ out and Ca+2 in.

Compare between the AP in cardiac & skeletal muscle

Slide 29-30

What is the Importance of Ca+2 from T tubules in cardiac muscles

Slide 24

Finally you have arrived , we have been waiting for you !!

Meet our team !

Team leaders

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Aljoharah Alyahya

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Did you like the lecture ? we mean our work :)

Contact with us! physiology.444ksu@gmail.com