Physiology Team 431

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Contractile Mechanism in Cardiac Muscle

★ This star means very important
Pink text = female doctor's explanation
Green text = Dr.Ashraf's explanation

Objectives:

- Define cardiac muscle contractility
- Understand the phases of cardiac action potential and the ionic bases
- Discuss the role of calcium ions in the regulation of cardiac muscle function
- Describe the mechanism of excitation contraction coupling
- Factors affecting cardiac contractility

- **Intercalated discs: cell membrane, separate cardiac muscle cells {they have gap junctions}**
- **Gap Junctions: trans-membrane channel proteins, connecting the cytoplasm of the cells**

Job:

Allow free diffusion of ions

Action potentials travel from one cardiac muscle cell to another

(Transmit impulses (excitations) so all cells contract at the same time)

- Cardiac Muscle is a Syncytium:

Stimulation of a single muscle fiber

The action potential spreads from cell to cell through the gap junctions
Contraction of all the muscle fibers

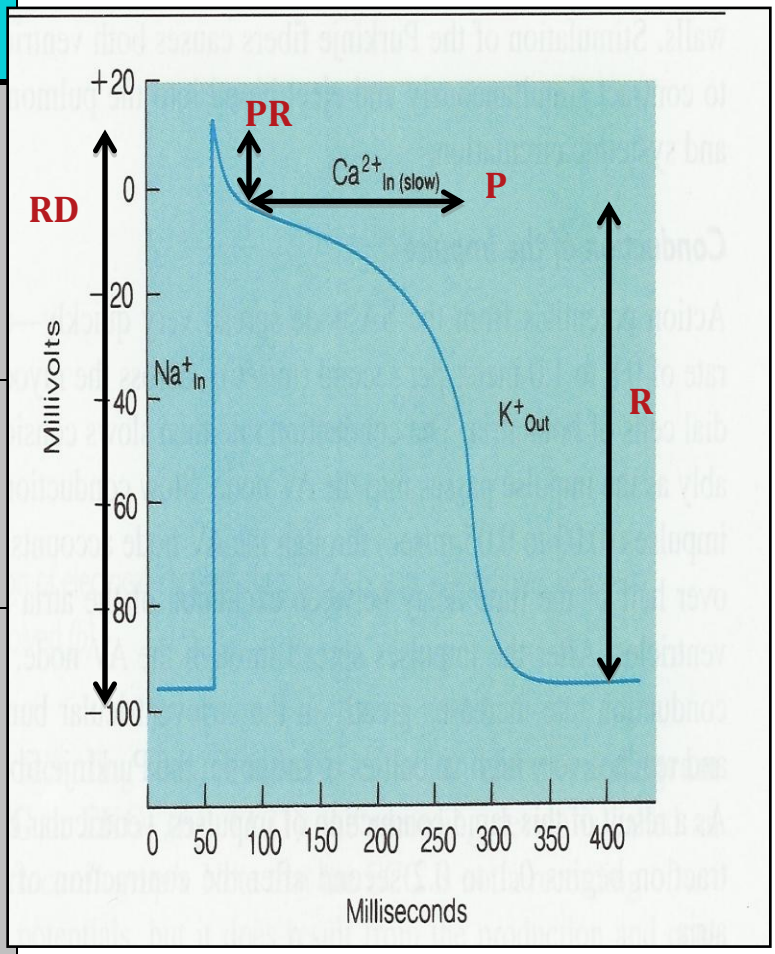
(i.e. if one cell is stimulated then all cells will be stimulated at the same time)

Action Potential in Cardiac Muscle:

- **Resting membrane potential (RMP) -85 to -95mV**
- **Phases of Action Potential in Cardiac Muscle:**

1. **Rapid depolarization (+20 mV)**
2. **Partial repolarization (5-10 mV)**
3. **Action potential plateau (0 mV)**
4. **Repolarization (back to RMP)**

Phases of cardiac Action Potential	Ionic changes
Rapid depolarization (RD) (+20 mV)	Na^+ in
Partial repolarization (PR)	K^+ out
Action potential plateau (P) (0 mV) (Cardiac muscles have slow calcium channels)	Ca^{2+} in (slow)
Repolarization (R) (back to RMP)	K^+ out



The doctor said that don't mention Na^{+} when they ask about the cause of plateau phase i.e. only Ca^{++}

What causes the Plateau in the Action Potential?

1. The fast sodium channels (as in skeletal muscle)
2. The slow calcium channels: slow to open & remain open

   Large quantity of both calcium and sodium ions flows to the interior of the cardiac muscle fiber

   Maintains prolonged period of depolarization

   Causing the plateau in the action potential

Refractory Period of Cardiac Muscle

- Absolute refractory period (ARP)
- (No response at all)

  Cardiac muscle cannot be excited while it is contracting … benefit?

  Long ARP

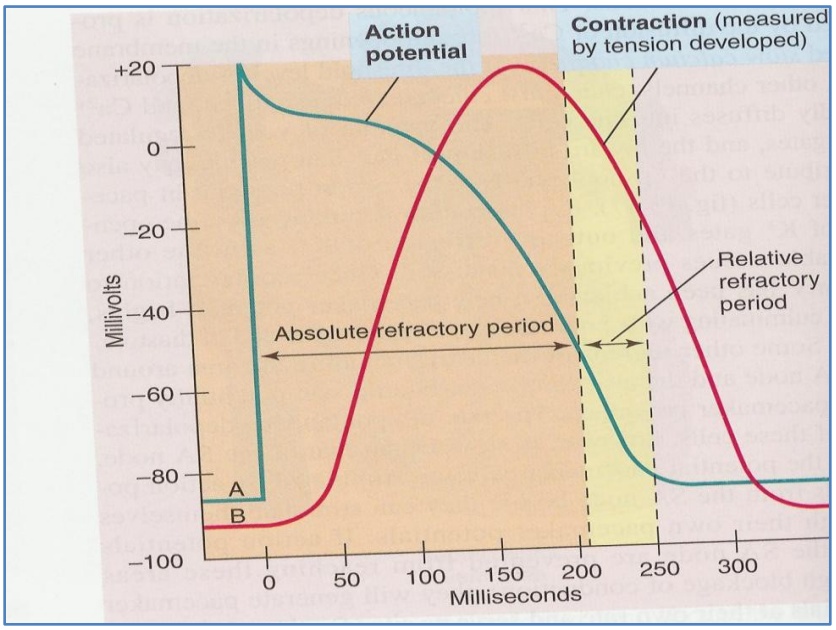
  Time: depolarization and 2/3 repolarization

  Duration: 0.25- 0.3 sec
- Relative refractory period (RFP)

  Cardiac muscle can be excited by strong stimulus

  Time: last 1/3 of repolarization

  Duration: 0.05 sec



Excitation – Contraction Coupling

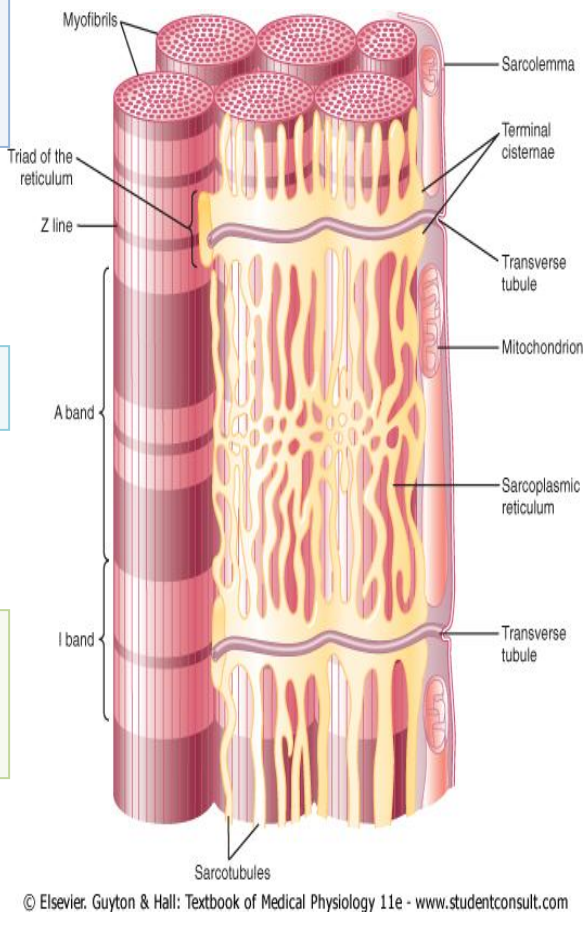
- It is the mechanism by which the action potential causes muscle contraction
- Action potential spreads to the interior of the cardiac muscle fiber along the transverse (T) tubules

Excitation – Contraction Coupling

Action Potential spreads along the T-tubules

Transverse (T) tubule-sarcoplasmic reticulum system

Release of calcium ions from sarcoplasmic reticulum and T-tubules



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Calcium ions diffuse into the myofibrils

↓

Ca^{2+} binds to troponin causing sliding of actin and myosin filaments

↓

Contraction of cardiac muscle

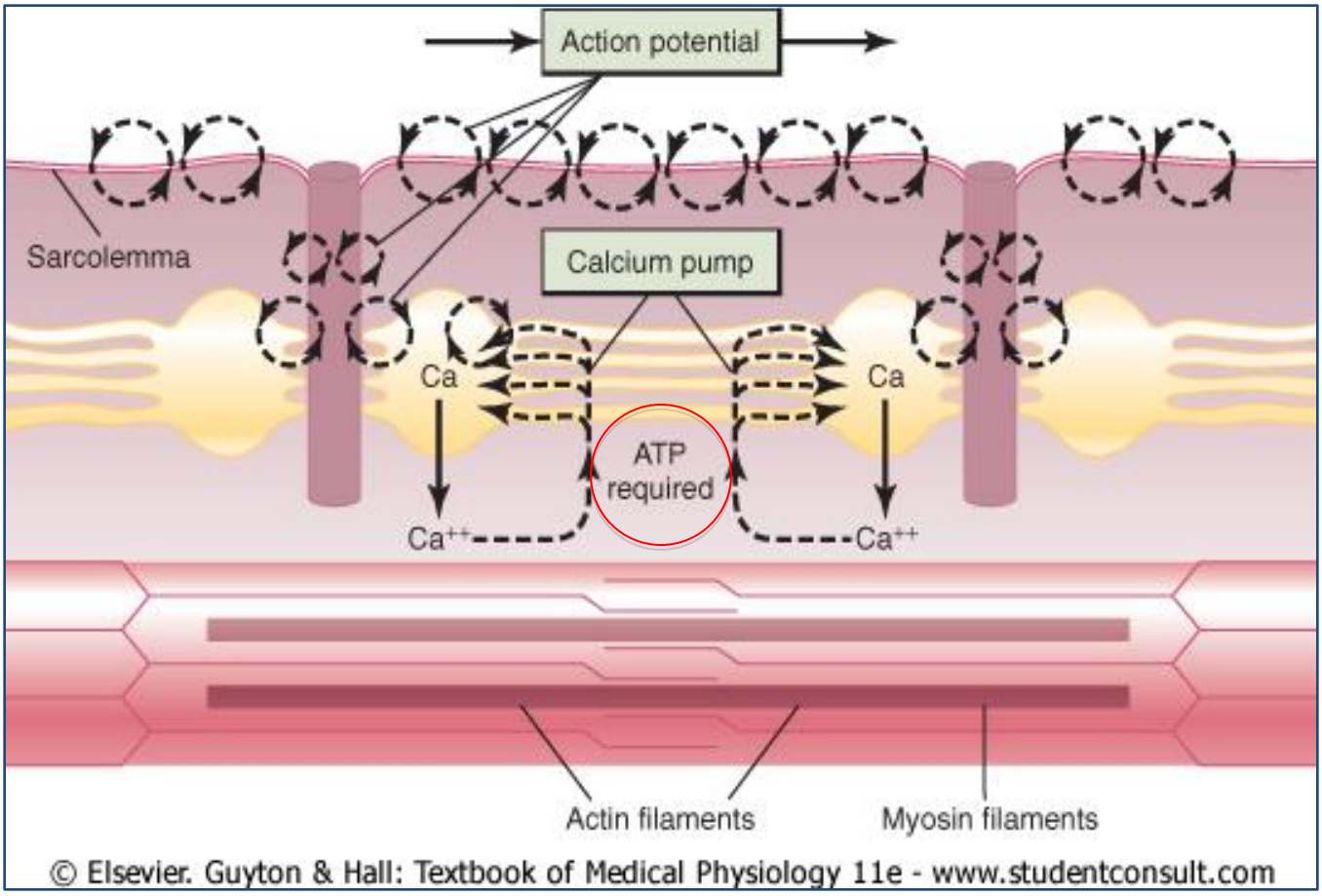
Excitation-contraction coupling in the muscle

At the end of the Plateau of the action potential

→ Calcium ions are pumped back into the sarcoplasmic reticulum and the T-tubules

→ Contraction ends (repolarization)

- Each contraction involves the hydrolysis of an ATP molecule for the process of contraction and sliding mechanism
- Cardiac muscle are continually contracting and require substantial(real) amounts of energy
- The energy is derived from ATP generated by oxidative phosphorylation in the mitochondria
- The myocytes contain large numbers of mitochondria (because of continuous need of energy)



The Contractility of the Cardiac Muscle

- **Contractility is the force of contraction of the heart**
- **It is essential for the pumping action of the heart**

Ionotropic effect:
Mechanism that affect the contractility

- ***Positive Ionotropic Effects:***
  Factors that increase the cardiac contractility

  Sympathetic stimulation
  Calcium ions

- ***Negative Ionotropic Effects:***
  Factors that decrease the cardiac contractility
  Parasympathetic stimulation
  Acetylcholine
  Vagal stimulation **(the vagus nerve lowers the heart rate when stimulated, has parasympathetic innervation)**

Cardiac muscle divided into two groups:
Atrial muscle & ventricle muscle

There is Non conductive fibrous ring between two group (AV) ring

All atrial and ventricle muscle originate and inserted in (AV) ring

All the valves: Tricuspid , pulmonary , mitral , aortic are attached to the (AV) ring

There is electrical continuity, when you stimulate atrial muscle ventricle will contract and when you stimulate ventricle the atrial will contract

There is electrical continuity called Syncytium

Stimulate move from atrial to ventricle throw Bundle of his (electrical connection) throw (AV) ring which transmit impulse from atrium to vertical

Cardiac muscle very rich in mitochondria

Intercalated disc give very little resistance because there is a gap junction (100 time less than skeletal muscle)

Syncetium (gap junction)

Nerve supply the heart through sympathetic and parasympathetic

Rhythmicity of the heart : it is continues beating of the heart

Autonomic nerve supply control the heart rate

When you stimulate sympathetic what will happen ?

*** Increased heart rate (chronotropic effect)**
*** Increased force contraction (inotropic effect)**

All parts of the heart are supplied equally by sympathetic nerve

Parasympathetic supplied AV node, SA node (vagus) , and its reduce heart rate

Parasympathetic *doesn't* supply myocardium

Force of contraction in ventricle because of sympathetic

Negative inotropic effect: less contraction (parasympathetic)

Positive inotropic effect: more contraction (sympathetic)

Where is the negative parasympathetic activity ?

In atrium, why ?

Because the SA node & AV node are situated in the atrium and its supplied by vagus nerve

Sarcolemma is the covering of cardiac muscle

T-tube is going all throw the thickness of the cardiac muscle

T-tube filled with mycopolyscaride and its rich in Ca++

T-tube of cardiac muscle is 4-5 times more product than skeletal muscle

Sarcoplasmic reticulum not fully developed in the heart , that's mean the Ca+ inside the heart is less

Intracellular Ca++ in case of heart store less because endoplasmic reticulum is not developed

Ca++ is important for any muscle contraction
For contraction we need Ca+ and ATP

In heart there are 2 sources of Ca++
1-endoplasmic reticulum (little)
2-through voltage channel

For relaxation :
80% Ca++ goes back again to endoplasmic reticulum and recycled

20% Ca+ will go through Ca+ sodium channel and then sodium pumped out and potassium move in

If we block Na-K pump what will happen ?

Increase inotropic effect

We may block Na-K pump in case of heart failure

drug : digoxin
MOA : block Na-K ATP pump in the heart and this help us for treatment of heart failure.(used in the past)

There are two sources of Ca+ inner and outer , Heart receives Ca+ from outer source.

Isovolumetric is the same as isometric contraction

Conductivity tissue of the heart it is between the nerve and muscle

SA node initiator of electrical activity in the heart (Pacemaker)

AV node can be stimulated by the interconnection between AV node and SA node (internodal connection) or in wall of atrium by way of depolarization

AV node giving impulse at rate of 80 while SA node impulse formation is 80

Right and left bundle branch coming from bundle of his
Left bundle branch has got two division is thicker than right

Purkinje fiber is the thinnest of all conductive tissue
Fastest conduction is 4 m/sec in Purkinje fiber

AV node delayed impulse 0.1 second

Questions:

In plateau Phase the value is around:

a) Zero
b) 5-10
c) 20

Negative ionotropic effect is caused by:

a) Ca++ ions
b) Ach
c) Sympathetic stimulation.

The time of relative refractory period is:

a) Last 1/3 of repolarization
b) Depolarization
c) First 2/3 of repolarization