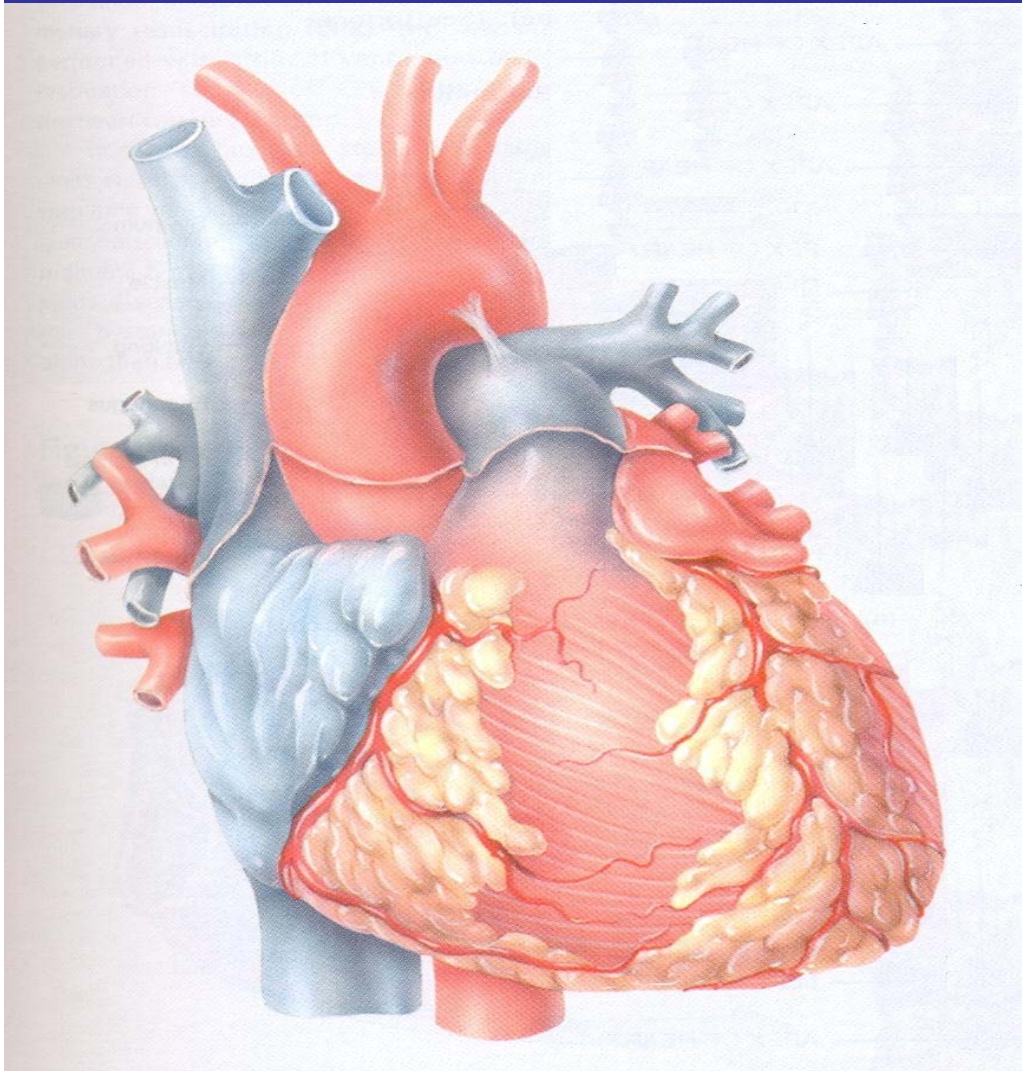
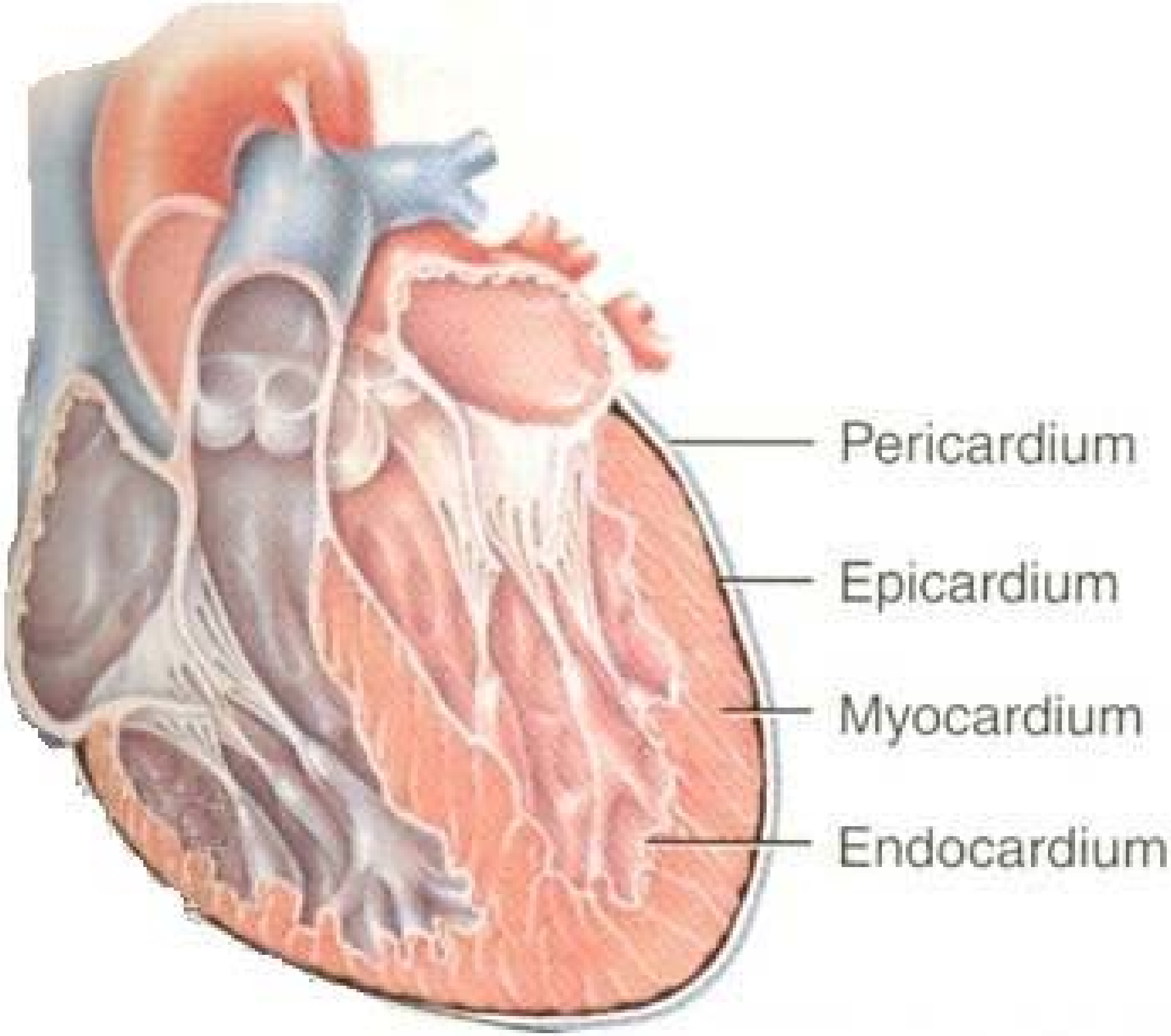
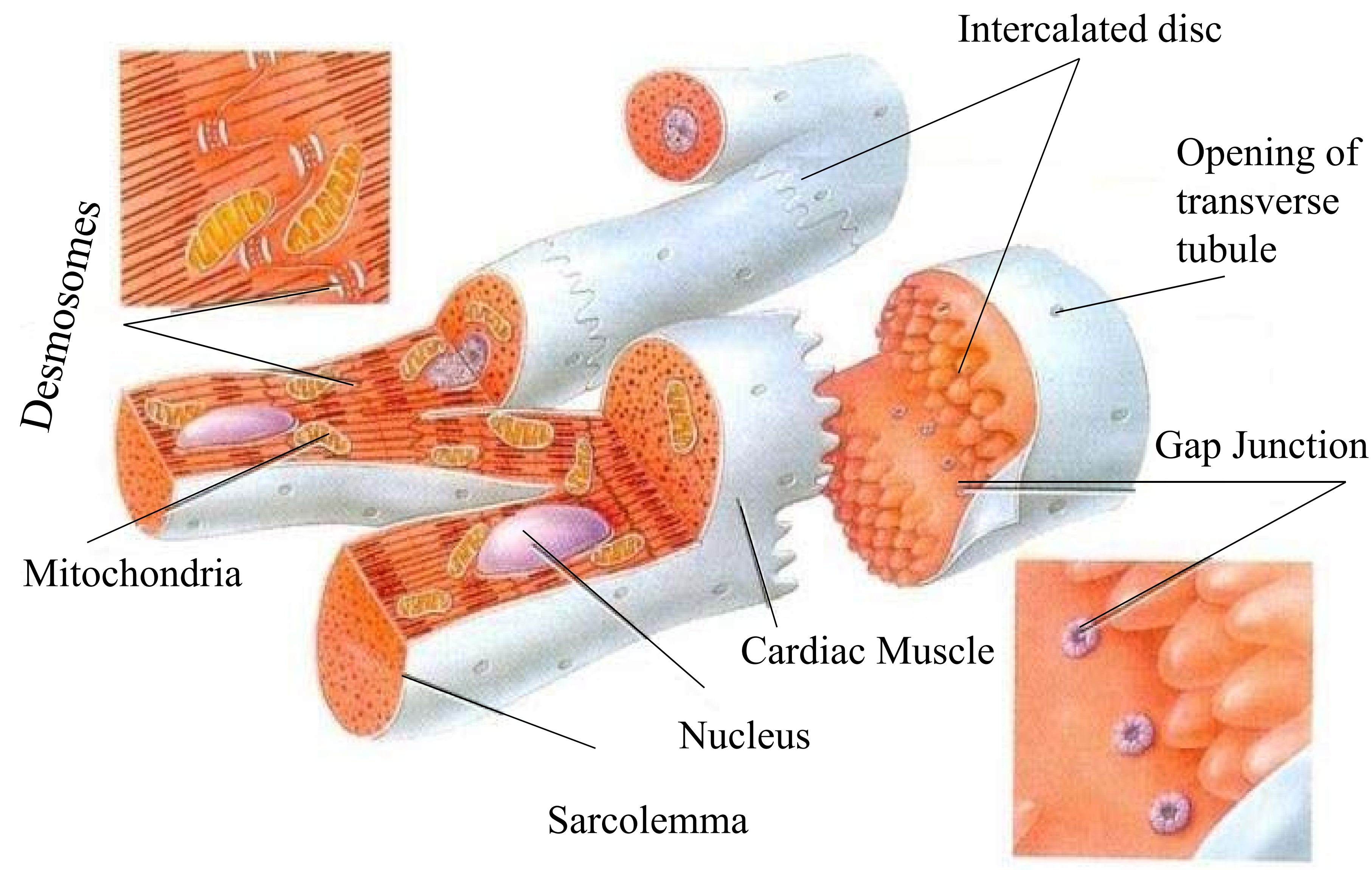
CARDIO VASCULAR SYSTEM

- Dr. Chetna Ramanuj

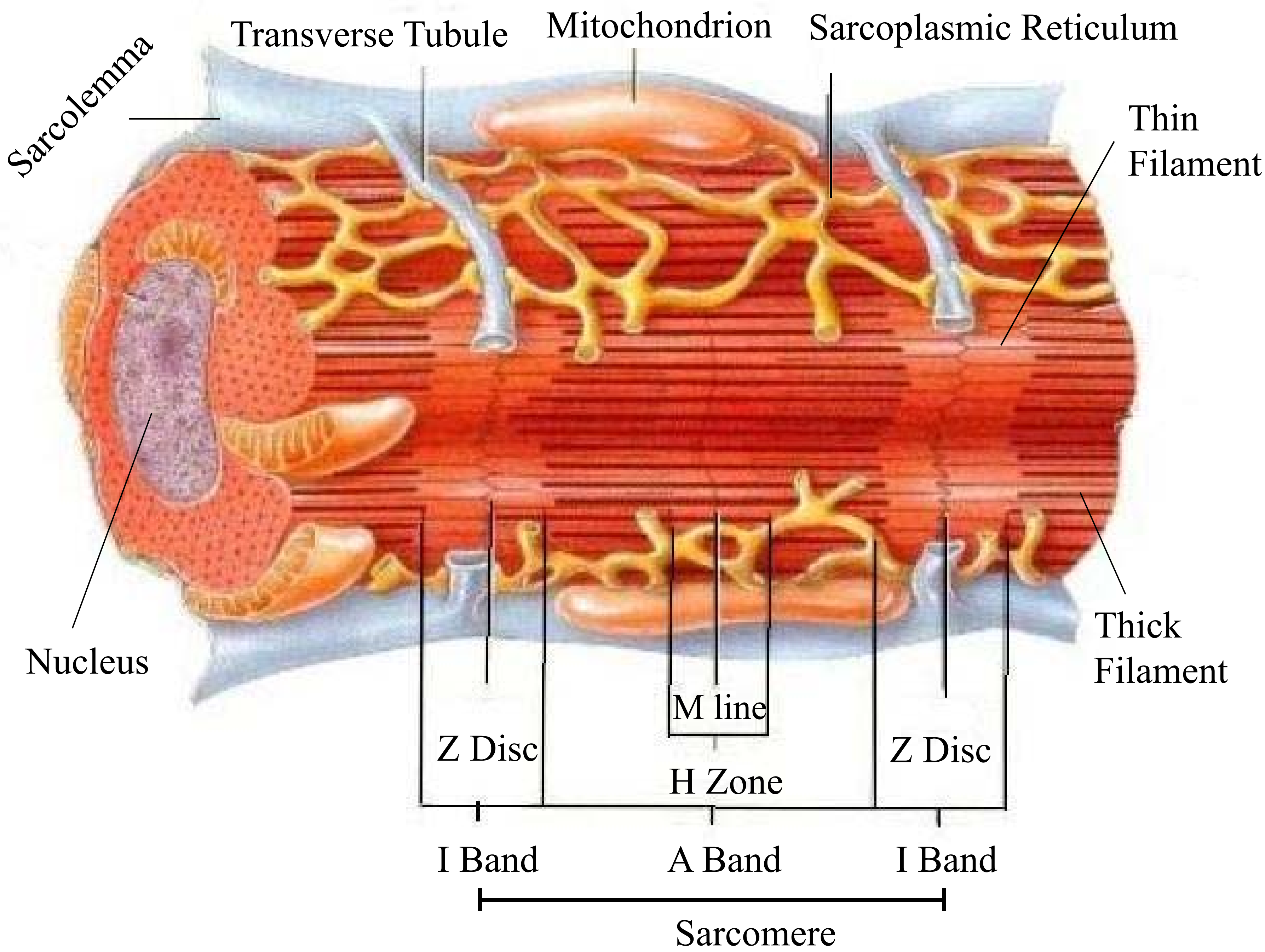




Pericardium
Epicardium
Myocardium
Endocardium



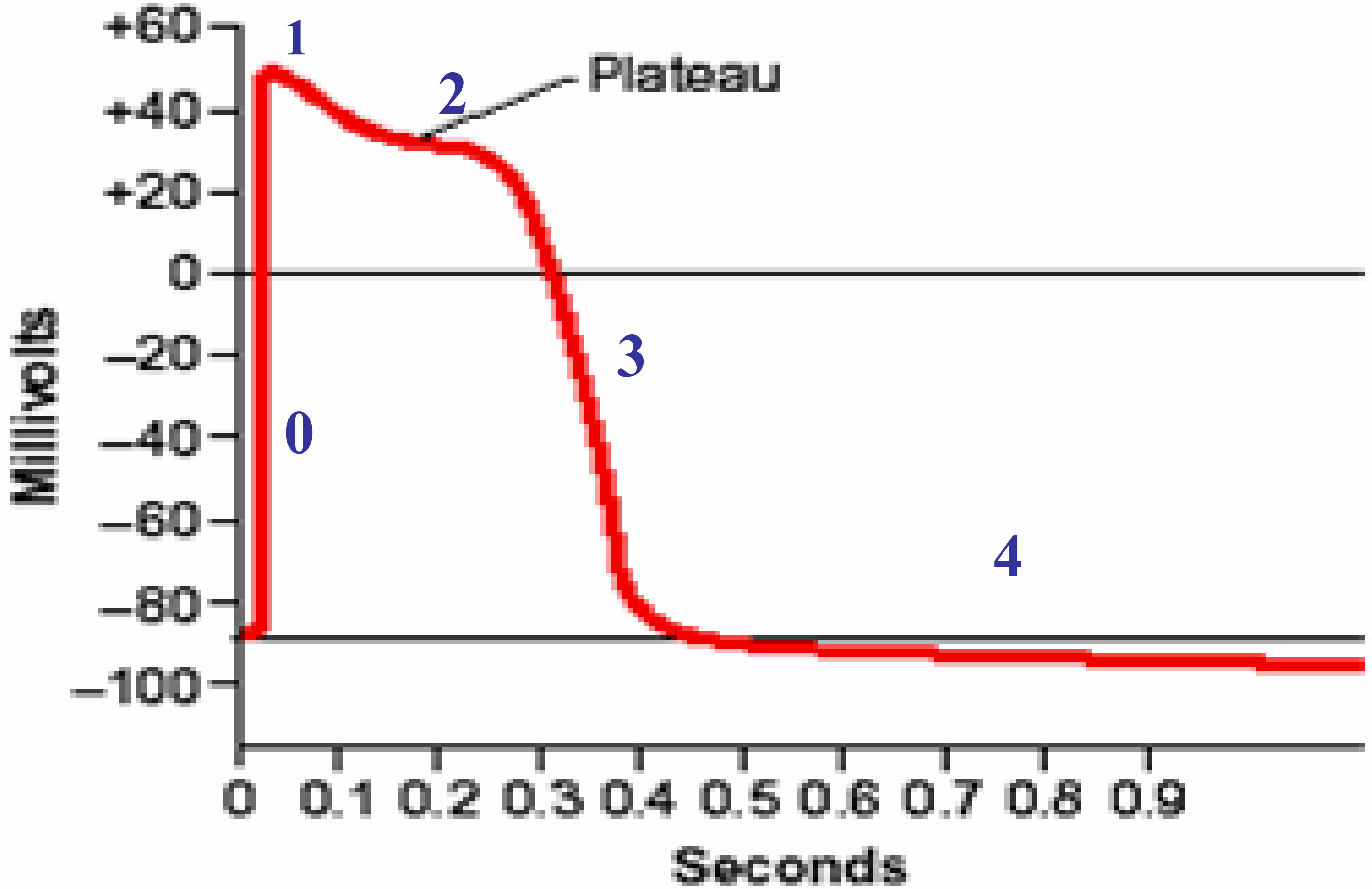
Intercalated disc
Opening of transverse tubule
Desmosomes
Gap Junction
Mitochondria
Cardiac Muscle
Nucleus
Sarcolemma

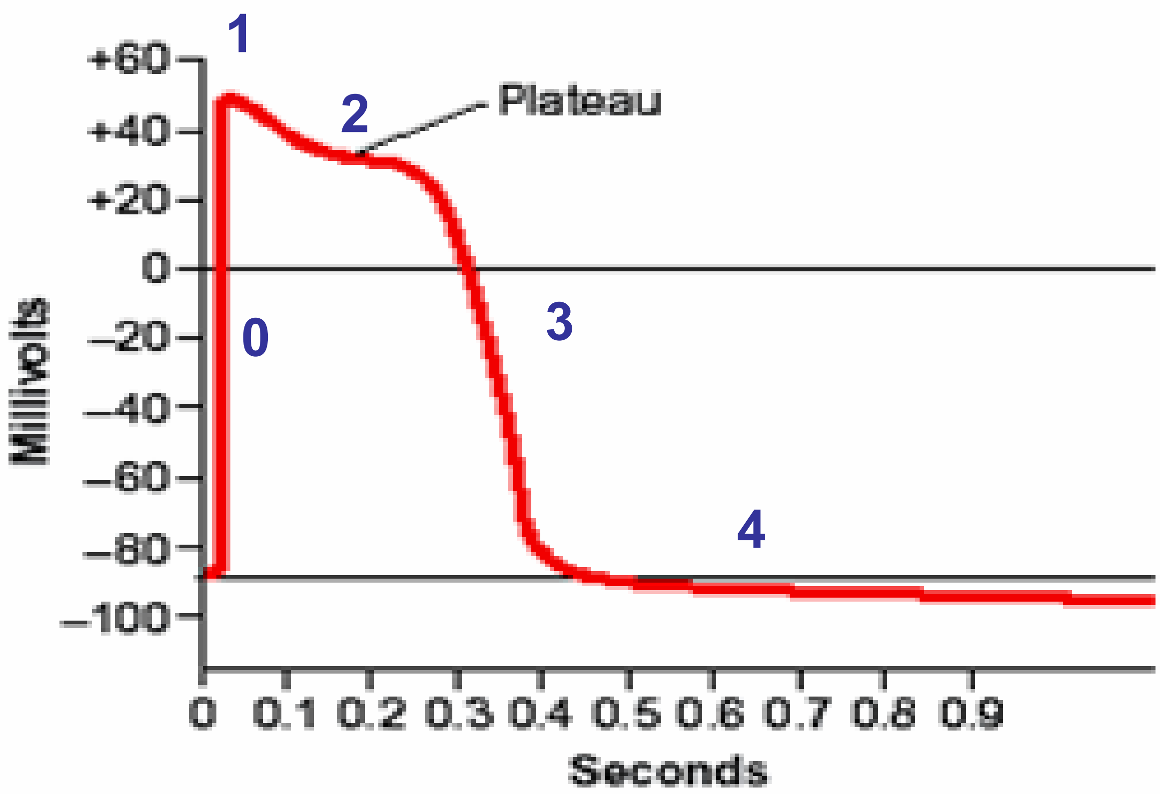

Sarcolemma
Transverse Tubule
Mitochondrion
Sarcoplasmic Reticulum
Thin Filament
Nucleus
Thick Filament
M line
Z Disc
H Zone
Z Disc
I Band
A Band
I Band
Sarcomere

PROPERTIES OF CARDIAC MUSCLE

- **Excitability**
- **Contractility and Distensibility**
  - **a) All or none law**
  - **b) Treppe or Staircase phenomenon**
  - **c) Refractory Period**
- **Automaticity**
- **Rhythmicity**
- **Coductivity**
- **Tonicity**

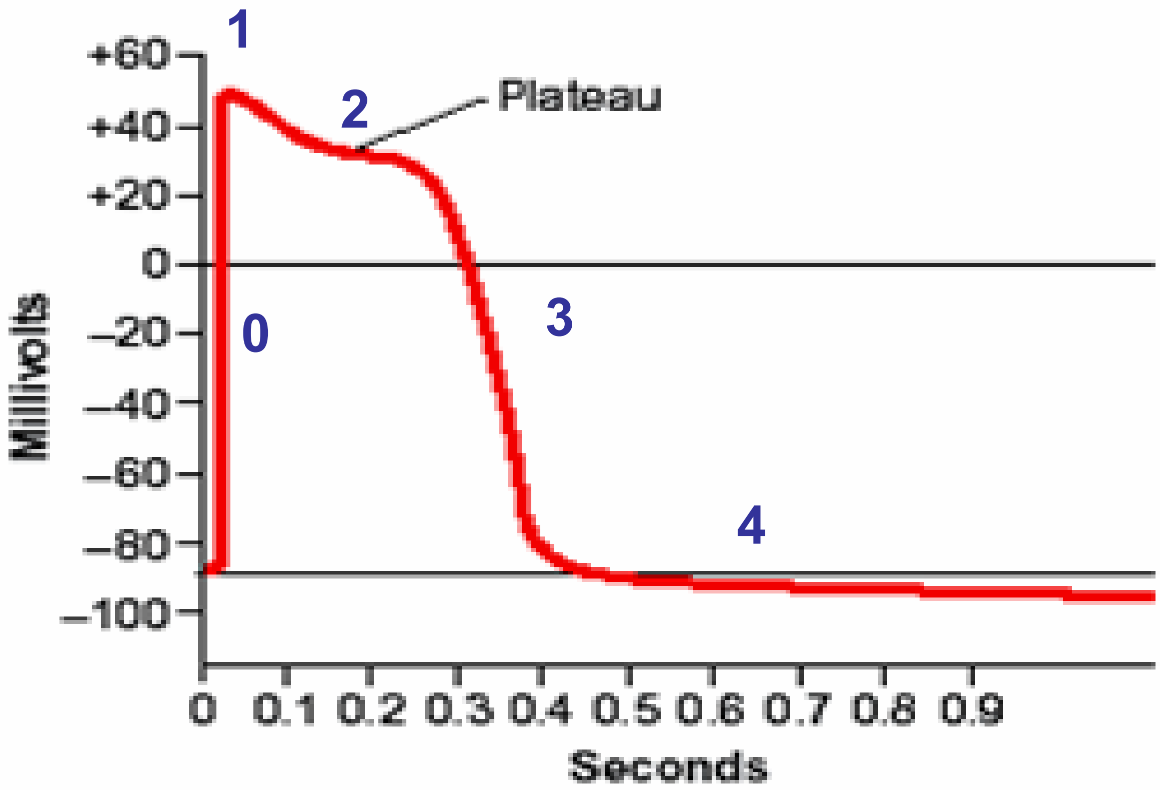
Long action potential & Plateau in cardiac muscle





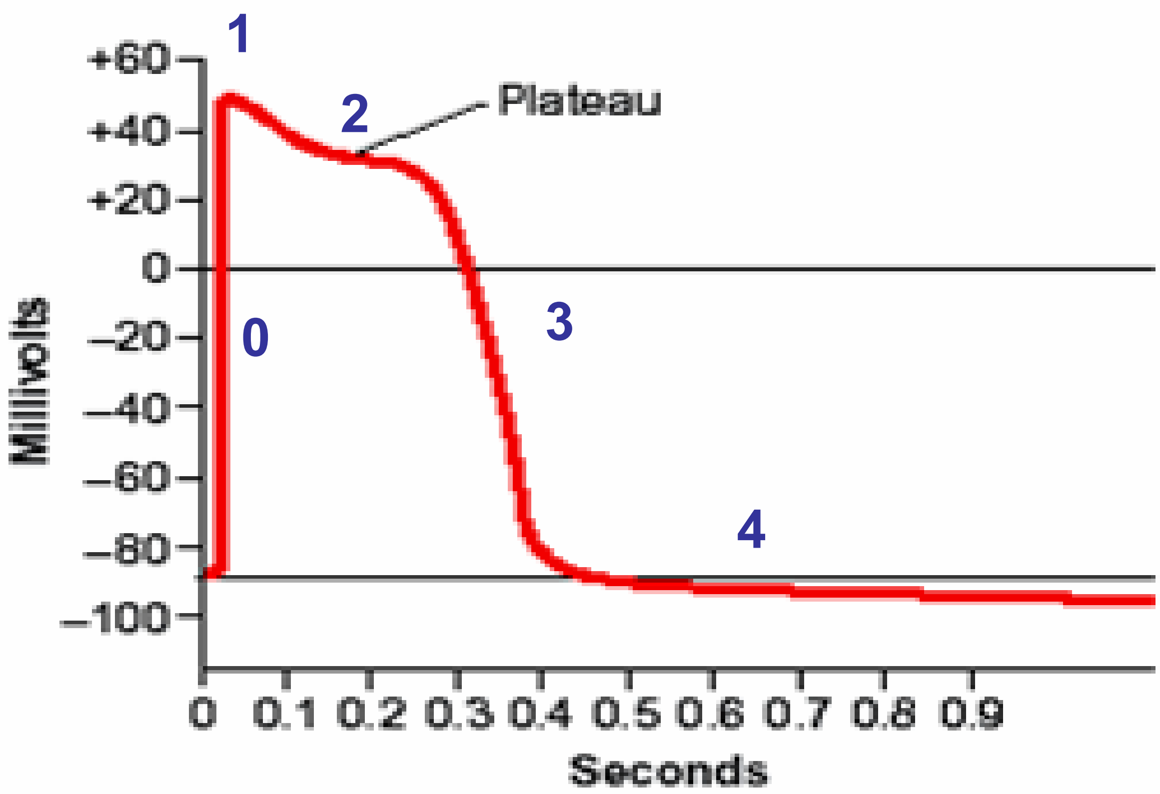
Phase 0 (Rapid Depolarization):

Entry of Na^+ in to the cardiac cell through "Fast" Na^+ channels to generate upstroke.



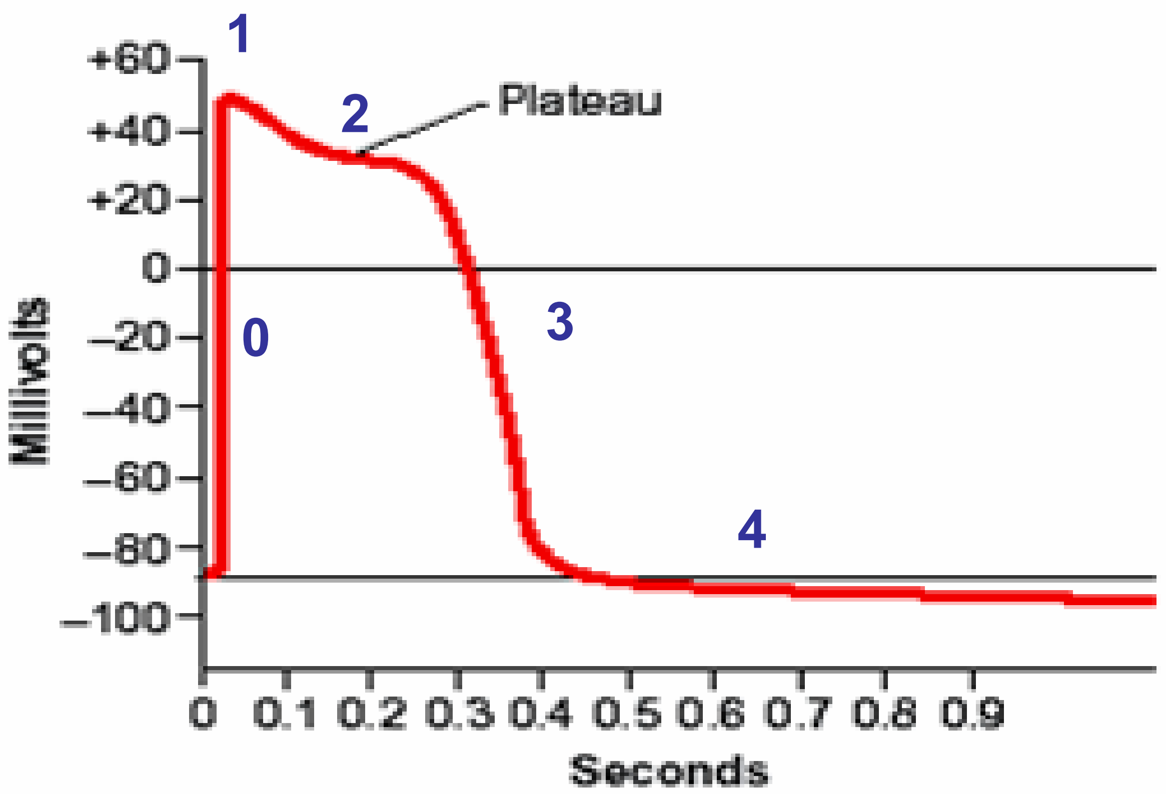
<u>Phase 1 (Initial Rapid Repolarization):</u>

Efflux of K^+ to generate early partial repolarization.



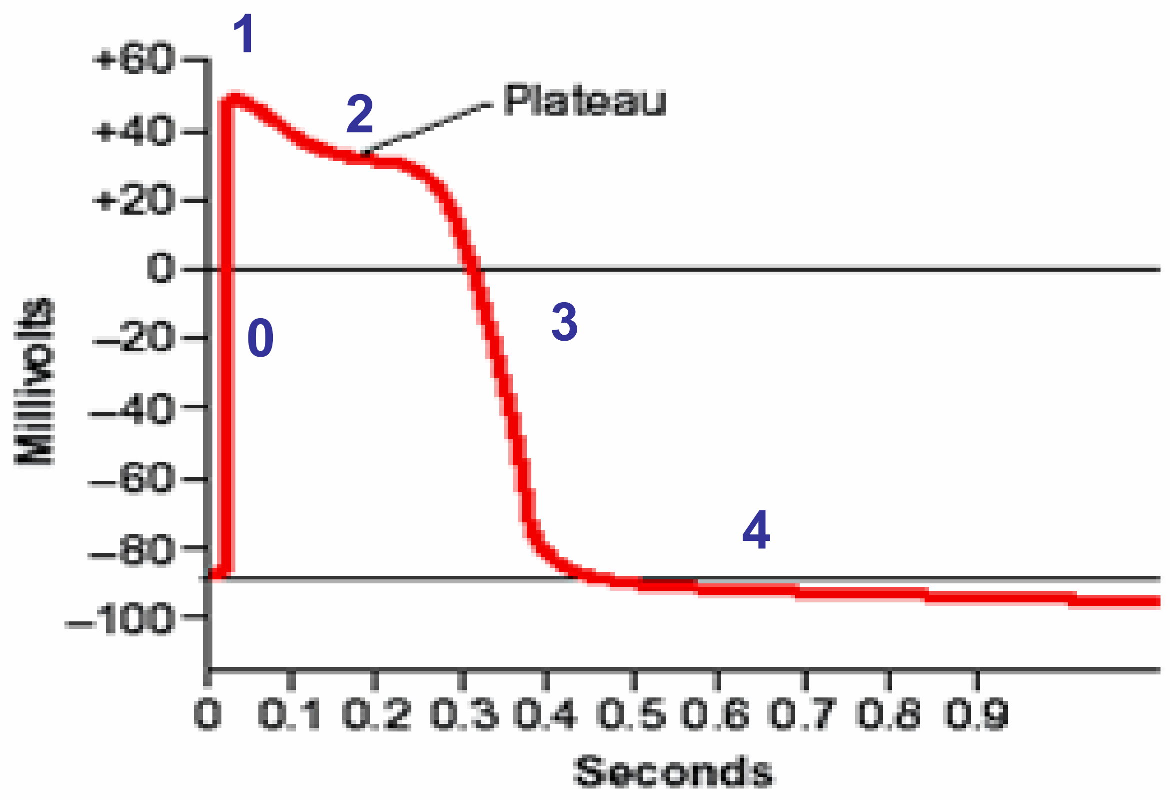
Phase 2 (PLATEAU PHASE):

Outflux /efflux of K^+ is reduced and opening of "Slow" Ca^{++}-Na^+ channels and entry of Ca^{++} and Na^+ in to the cardiac cell. This maintains a prolonged period of depolarization causing "Plateau"



Phase 3 (Repolarization):

Efflux of K^+ is accelarated.



Phase 4 (Resting Potential):

Here, Excess of K^+ going out of the cell causes "Hyperpolarization"

Excitation-contraction coupling in cardiac muscles

The sequence of events during the Excitation-contraction coupling in the cardiac muscle are similar to those in skeletal muscles except for the following :

Here, the extra calcium ions diffuse into the sarcoplasm from T-tubules without which the contraction strength would be considerably reduced.

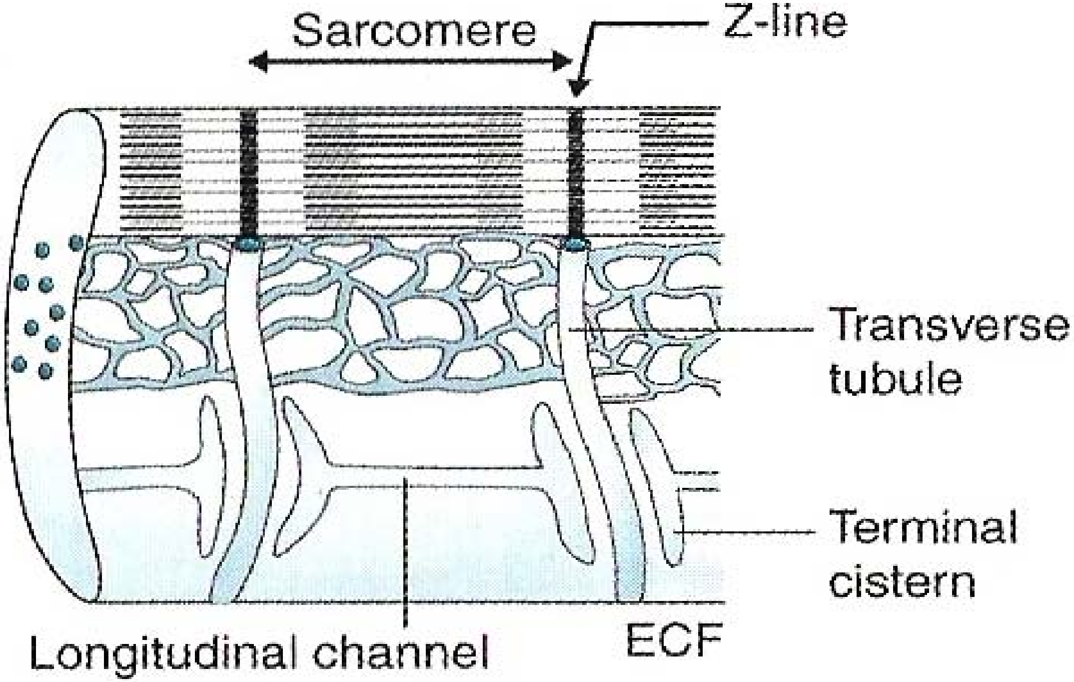


Fig. 4.1-6. *Sarcotubular system in the cardiac muscle.*

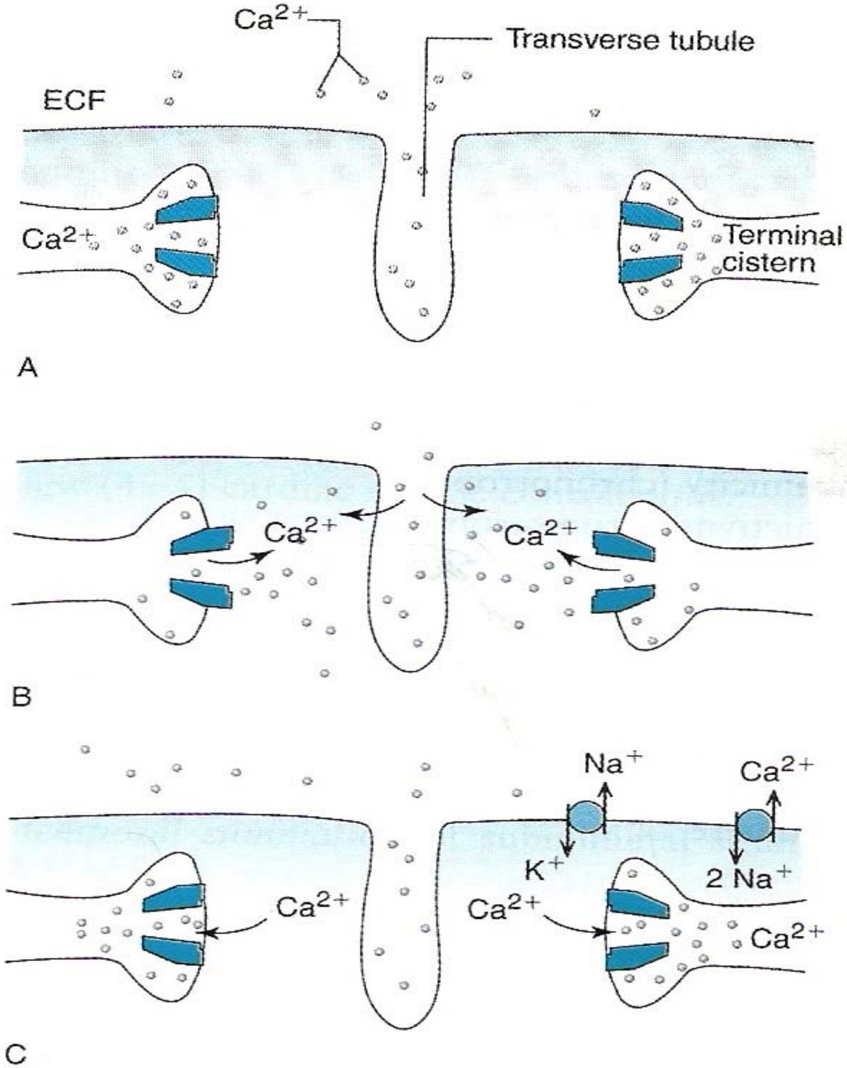


Fig. 4.1-8. *Dynamics of Ca^{2+} during excitation-contraction coupling phenomenon and relaxation in cardiac muscle: A, in resting state; B, calcium induced calcium release during excitation and contraction state; and C, during relaxation state.*

EXCITABILITY: (Bathmotropism)

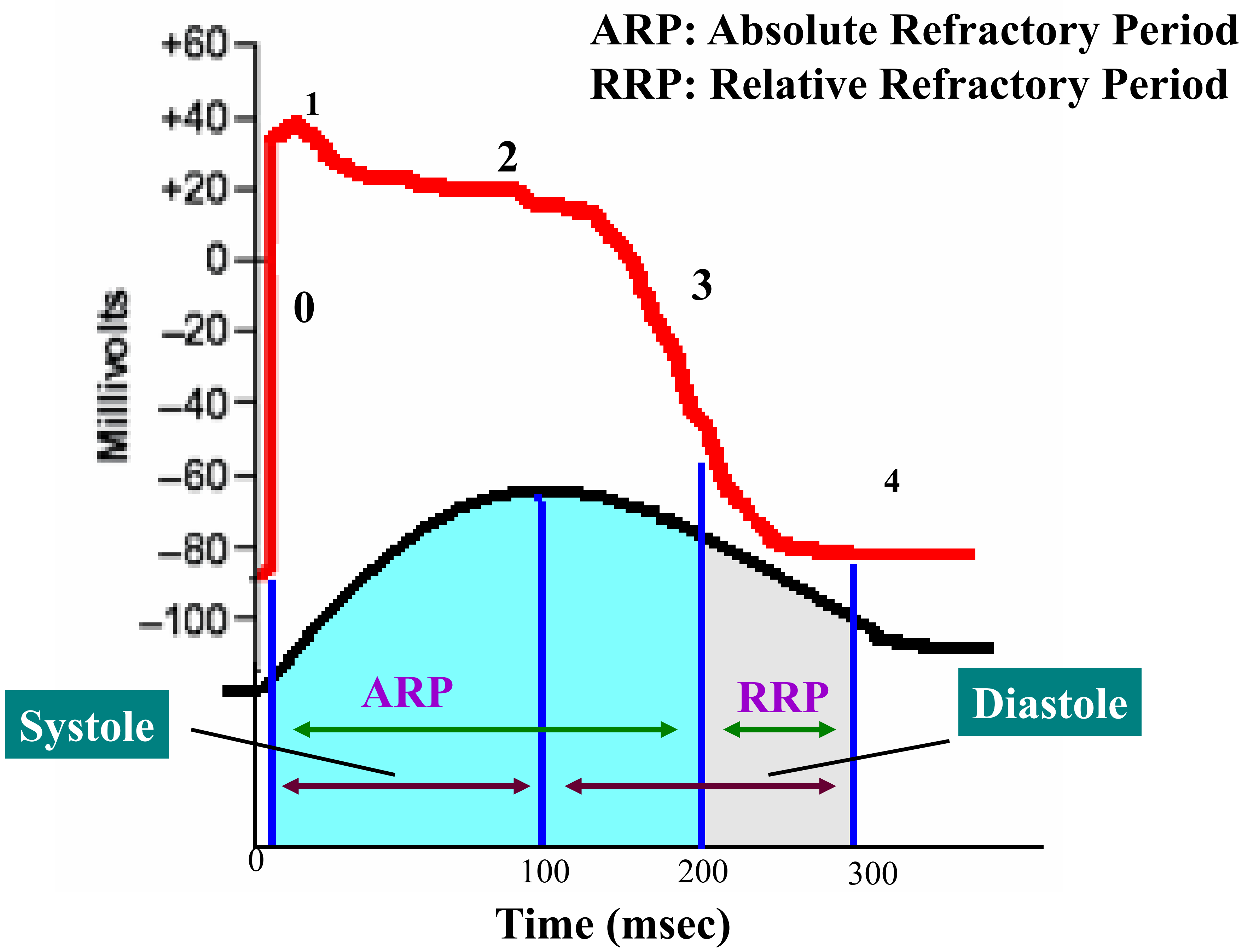
This is the property by which cardiac muscle response to stimuli by generation of electrical potential. This can be action potential of atria, ventricular or potential of pace maker tissue like S.A. node, A.V. node

Refractory Period:

Definition: it refers to the period following action potential during which the cardiac muscle does not respond to a stimulus

It can be divided into several segments.

1. Absolute Refractory Period
2. Relative Refractory Period
3. Supernormal Period
4. Full Recovery Period


ARP: Absolute Refractory Period
RRP: Relative Refractory Period
+60
+40
+20
0
–20
–40
–60
–80
–100
Millivolts
1
2
0
3
4
ARP
RRP
Systole
Diastole
0
100
200
300
Time (msec)

1. <u>Absolute Refractory Period:</u>

The excitability of cardiac muscle is nil during the period of contraction.

The stimulus however, will fail to produce any response during this phase as the muscle fiber remains in a “Depolarised State"

2. Relative Refractory Period:

- ❖ During the period of relaxation there is a gradual return of excitability. A strong stimulus is needed to bring about a response.

- ❖ Effective Refractory Period constitutes that period during which only a **local** response can be produced by a stronger than normal stimulus. Thus, cell membrane will respond to the stimulus but propagated action potential can not take place.

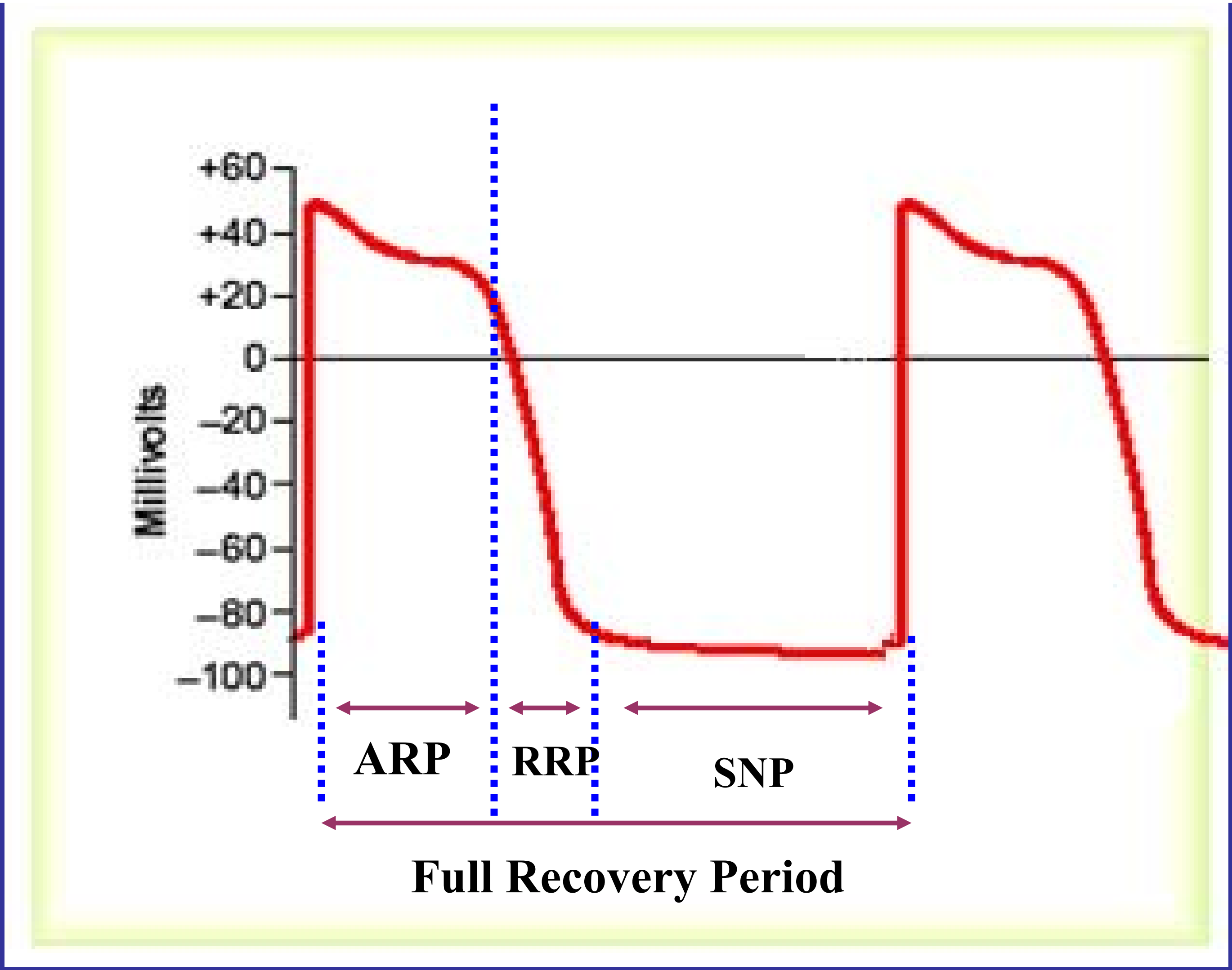
- ❖ The relative refractory period commences at the end of ERP during which propagated action potential can be generated.

3. Supernormal Period:

It is a short interval during which the cell is more excitable than normal, that is a weaker than normal stimulus can initiate a propagated action potential.

4. Full Recovery Period:

It constitutes the period from the onset of action potential to the end of supernormal period.



ARP: Absolute Refractory Period,

RRP: Relative Refractory Period

SNP: Supernormal Period

SIGNIFICANCE OF THE REFRACTORY PERIOD:

The cardiac muscle is refractory to any stimulus during the contraction phase (Systole).

Therefore, the complete summation of contraction and thus tetanus cannot be produced in the cardiac muscle.

CONTRACTABILITY and DISTENSIBILITY:

Action potential spreads to the interior of cardiac cell

⇓

Releaseof Ca^{++} intracellularly

⇓

Ca^{++} and Troponin

⇓

Troponin dislodges tropomysin from myosin reactive sites.

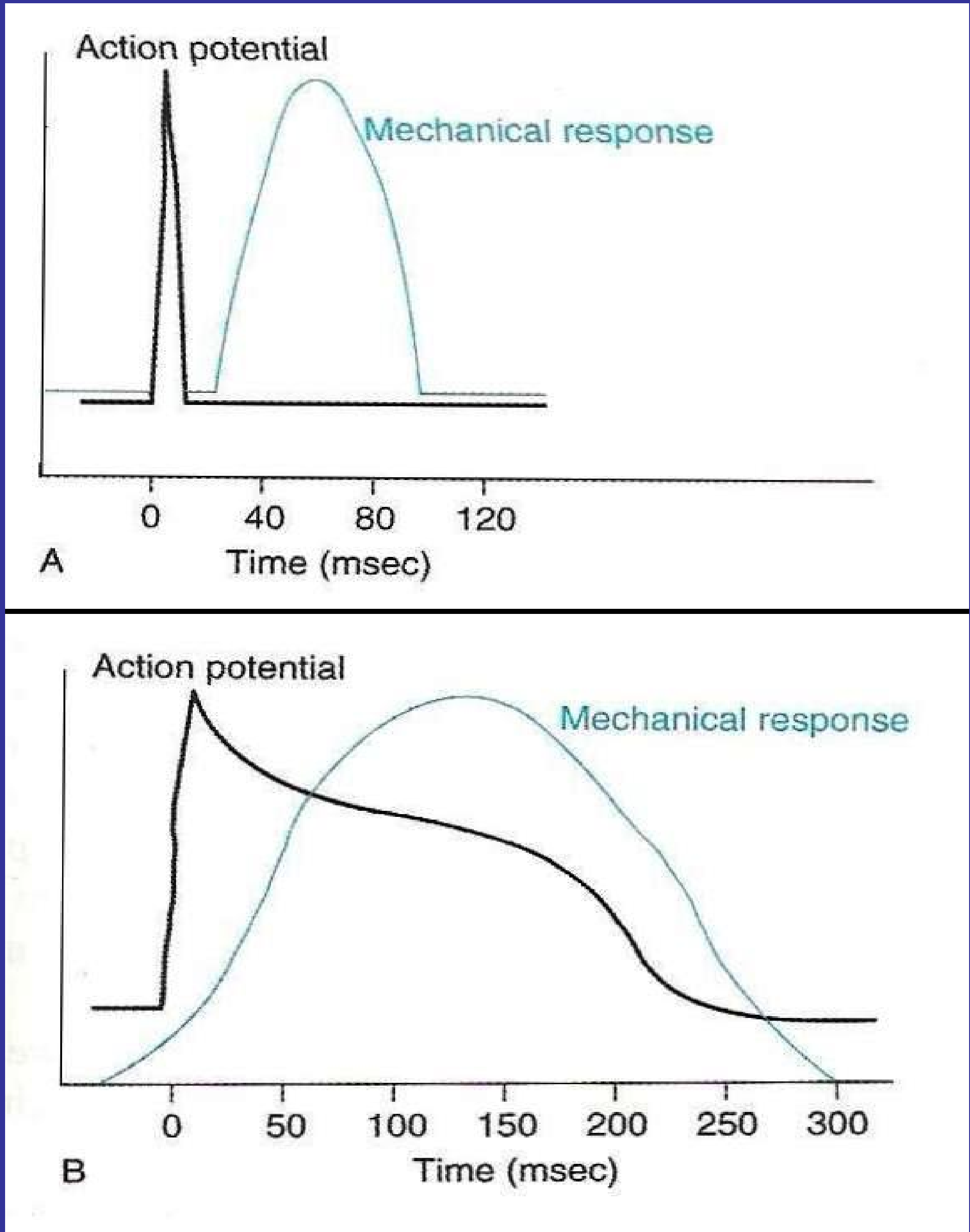
⇓

Actin and Myosin form cross bridges

⇓

Cardiac muscle contracts

- In case of cardiac muscle cell, Ca^{++} ions come from sarcoplasmic reticulum plus transverse (T) Tubules.
- This extra Ca^{++} from T-tubules (which is actually ECF Calcium) provides more strength to the contraction.
- This is in contrast with skeleton muscle contraction in which Ca^{++} is solely derived from sarcoplasmic reticulum
- Depletion of ECF Ca^{++} affects the contractility of heart but not that of skeleton muscle.



Skeletal muscle

Cardiac muscle

Factors affecting the contractile property of heart muscle

1. Intensity of the stimulus:

   Stimulus must be of threshold value to cause the contraction of heart muscle

2. Timing of the stimulus:

   If a stimulus falls during absolute refractor period – no response occur

3. Effect of initial tension:

   The heart muscle contracts better if the initial tension is high.

Factors affecting the contractile property of heart muscle

Frank-Starling Law:-

Within physiological limits- greater the heart muscle is stretched during filling, the greater will be the force of contraction.

- When the cardiac muscle is stretched to a greater length, it causes the muscle to contract with increased force because the Actin and Myosin filaments are then brought to a more nearly optimal degree of inter-digitation for force generation.

Factors affecting the contractile property of heart muscle

4. Oxygen lack:

Slight hypoxia: ↑sed contractility

Severe hypoxia: ↓sed contractility

5. Temperature:

↑ temp. :- ↑ contractility (heat causes ↑ permeability of the muscle membrane to ions)

Factors affecting the contractile property of heart muscle

6. Calcium Content:

Slight ↑ in Ca^{++} in bathing fluid : ↑ contraction

Excess calcium : Calcium rigor (Spastic Contraction of heart)

7. Nervous Effect:

Vagal stimulation :- ↓ contractility

Sympathetic stimulation :- ↑ contractility

Factors affecting the contractile property of heart muscle

8. Effect of Potassium ion:

Excess potassium in ECF

↓

↓ sed Residual membrane potential of cardiac muscle

↓

Intensity of action potential also decreases

↓

Heart becomes extremely dilated and flaccid

↓ Heart rate

All or None Law:

When a cardiac muscle fiber is stimulated by threshold stimulus, the effect will be maximum contraction AND if it is inadequate, it will not cause any response.

Treppe or Staircase Phenomenon:

In 1871, Bowditch observed in a frog’s heart, that if the heart had been inactive for a period of time, a sudden series of stimulations induce a corresponding series of contractions that increased in amplitude until a steady state was reached. This Phenomenon was called “Treppe” or “Staircase”

Period of Systole
(Inactive)

Treppe or Staircase Phenomenon:

On the other hand, when the rate of stimulations of the heart was suddenly decreased, a series of consecutively decreasing contractions was obtained until a second steady state was reached.

This Phenomenon was called "Negative Treppe" or "Negative Staircase"

Treppe or Staircase Phenomenon:

Cause of Staircase Phenomenon:

Increase of Ca^{++} ions in the cytosol because of release of more and more ions from sarcoplasmic reticulum with each muscle's "Action Potential" and failure to re-capture the ions immediately.

Treppe or Staircase Phenomenon:

Physiological Significance:

A positive staircase phenomenon may account for the improved performance of athlete after "warm up" exercise.

Treppe or Staircase Phenomenon:

REMEMBER:

Staircase Phenomenon is possible either in a skeletal muscle in the body or in an isolated heart (outside the body) because it is observed after a longtime of rest which is not possible for heart in the living body.

Effect of Preload

Preload: A load which starts acting on a muscle before it starts to contract is called Preload.

Effect: The preload increases the initial length of the muscle.

In heart muscle, the end diastolic volume forms the preload.

The Frank-Starling law of heart:

Within physiological limits the force of cardiac contraction is proportional to its end diastolic volume.

Length-tension relationship

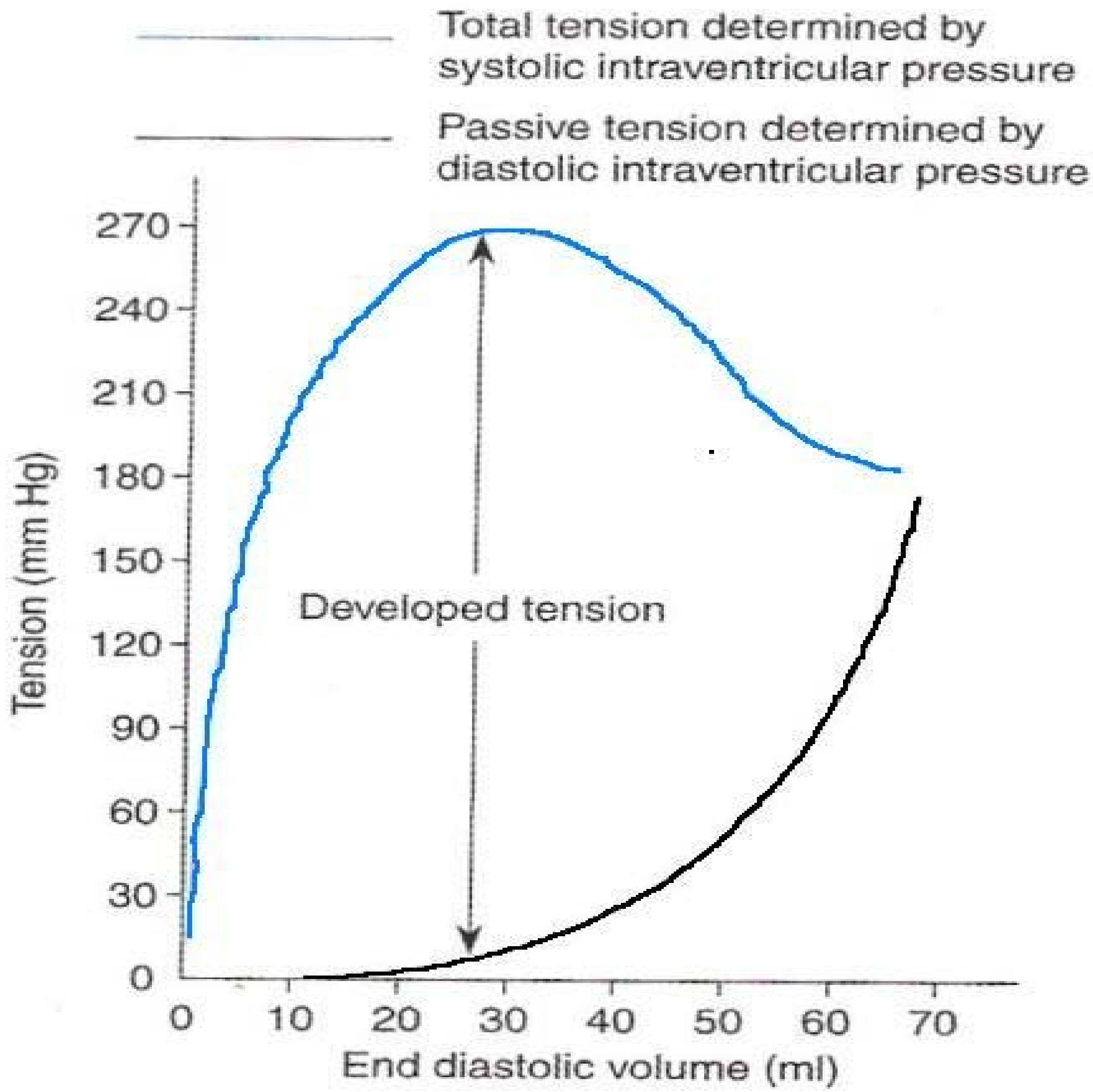


Fig. 4.1-13. *Length-tension relationship in cardiac muscle*

Length-tension relationship:

Diastolic intra-ventricular pressure represents the passive tension and it increases in end diastolic volume.

Systolic ventricular pressure represents the active tension developed which is proportionate to the degree of diastolic filling of the heart.

Effect of Afterload:

Afterload: it refers to the load which acts on the muscle after the beginning of muscular contraction.

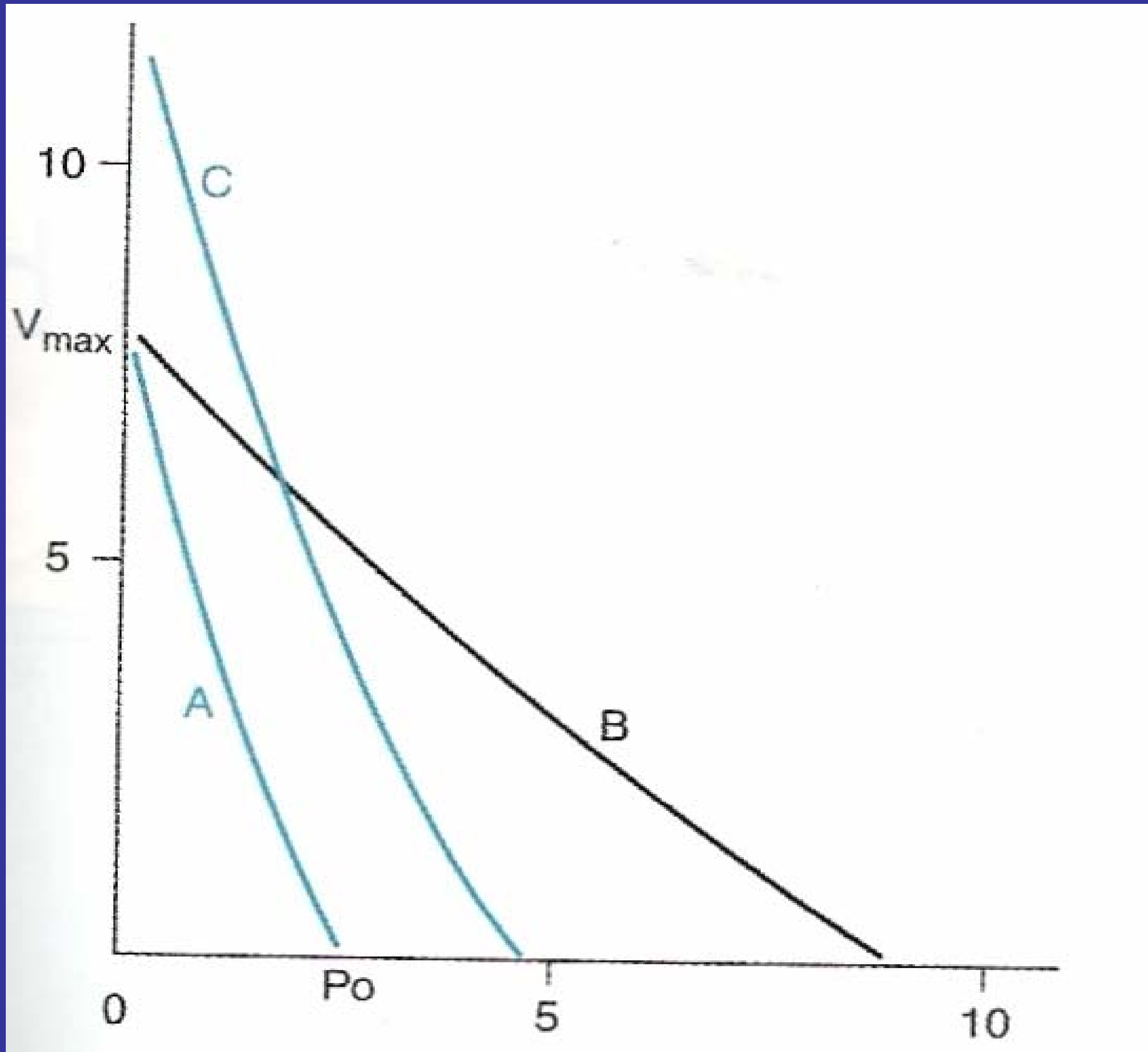
Effect: The afterload affecting the force of contraction of cardiac muscle is represented by the resistance against which the ventricles pump the blood.

Effect of Afterload:

- The afterload (resistance) for right ventricle is low in pulmonary artery due to its intrathoracic location.

- The afterload (resistance) for left ventricle is high in the aorta due to resistance to blood flow through the arotic valves and systemic blood vessels, called ***peripheral resistance.***

Force-velocity relationship



Tension (mm Hg)

End diastolic volume (ml)

Force-velocity relationship:

The force-velocity curve is plotted by noting the velocity of muscle contraction with progressively increasing load on the muscle.

In the heart, load is represented by the resistance against which the ventricles pump the blood and velocity of muscle contraction is represented by the stroke output.

Force-velocity relationship:

Inferences:

When load is zero, the muscle contracts rapidly, and the velocity of muscle shortening is maximum. (Vmax)

As load increases progressively the velocity of shortening decreases till it reaches zero. The force developed is called maximum isometric force and is represented by P_0.

DISCLAIMER

- All figures are taken from Guyton and Hall Textbook of Medical Physiology, 12^{th} Edition.