

# Cardiac Muscle Physiology

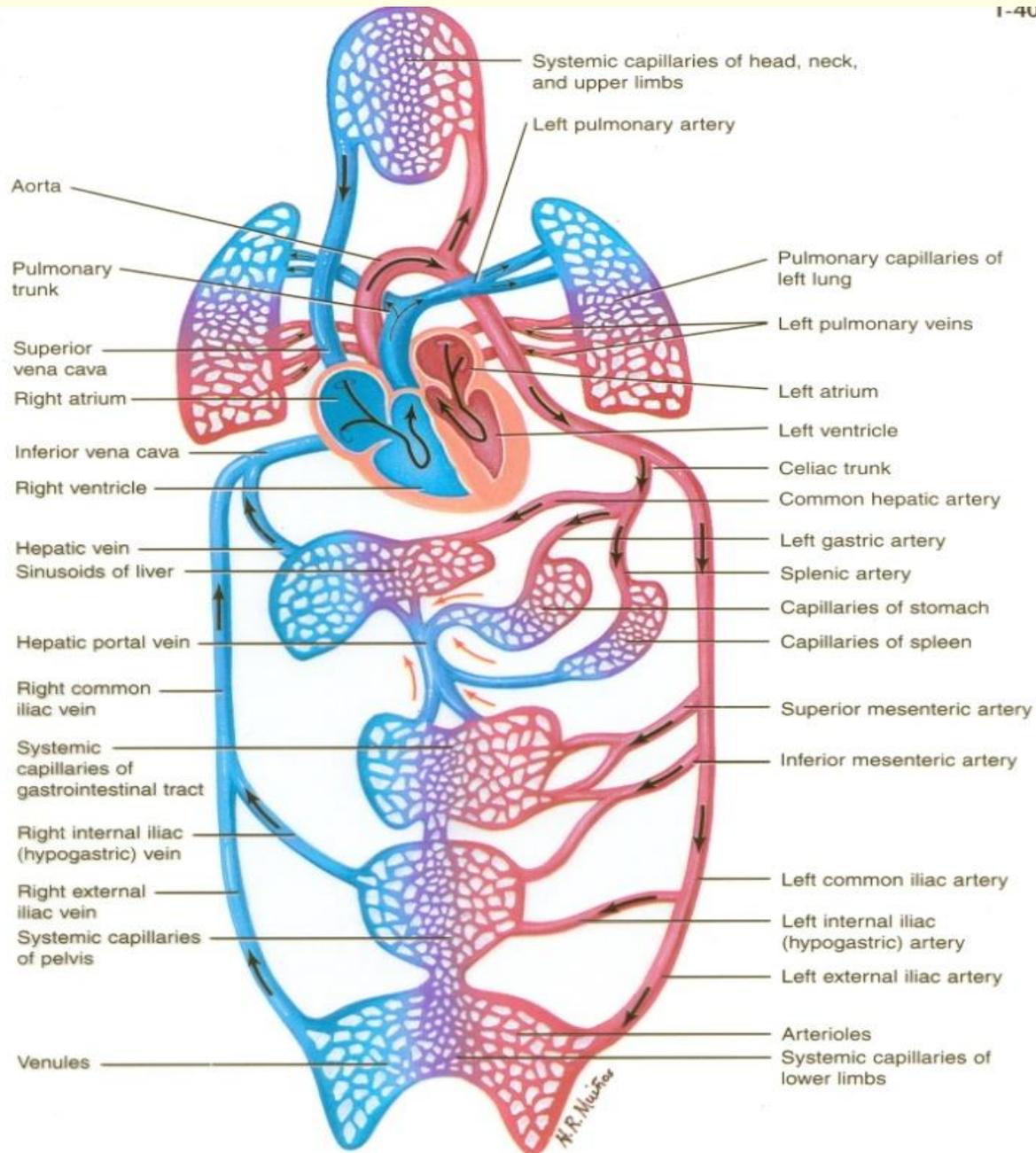
Faisal Mohammed, MD, PhD

# Objectives:

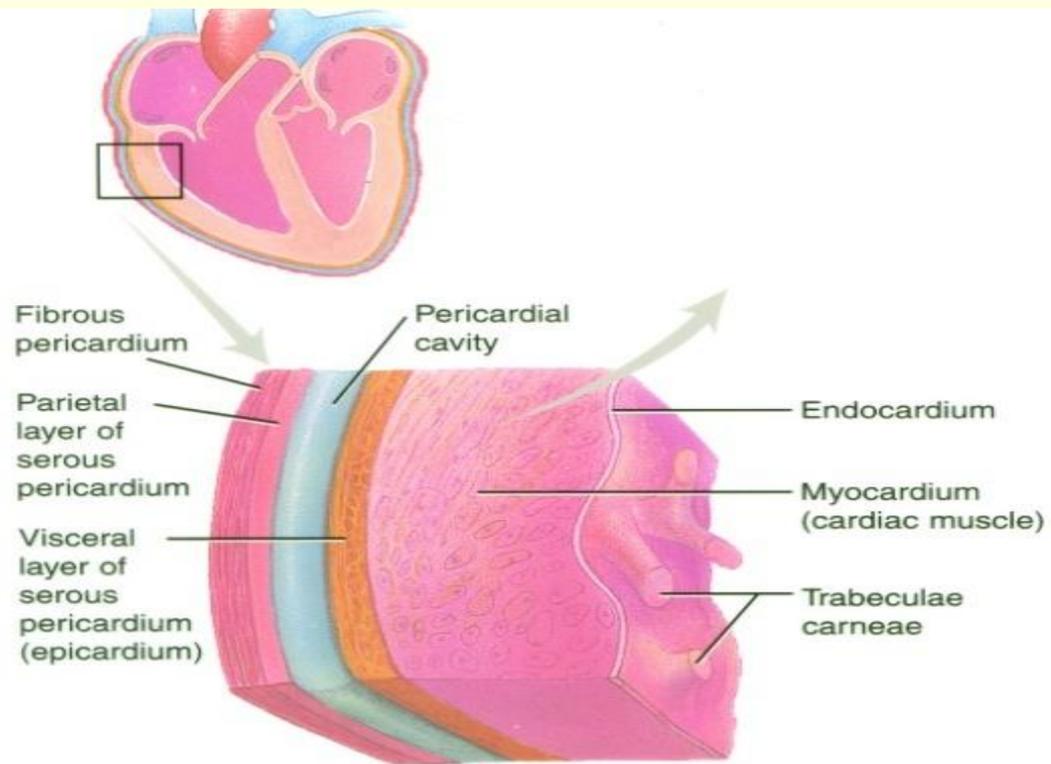
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By The end of this lecture students should be able to:

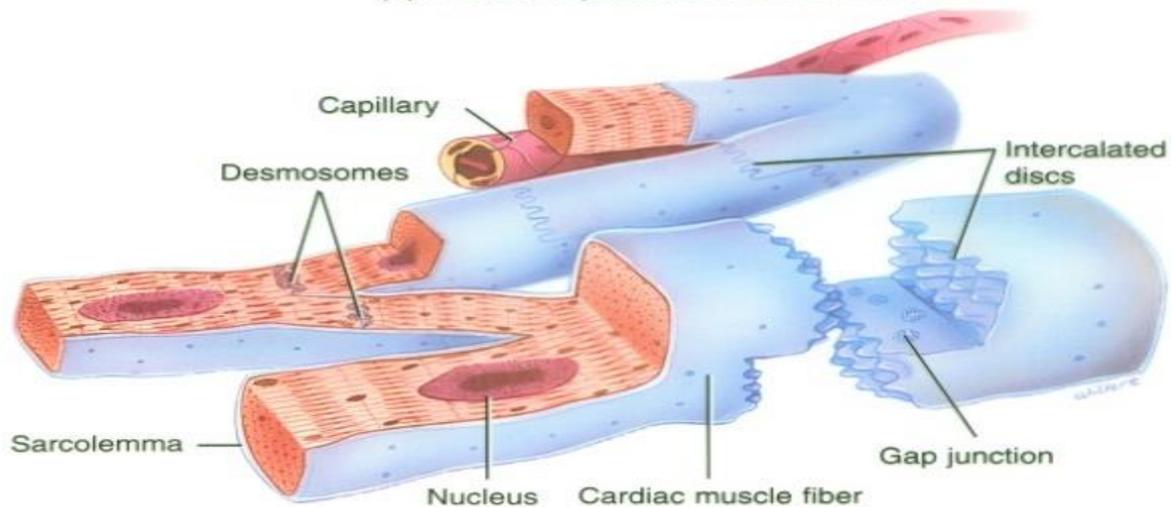
- Distinguish the cardiac muscle cell microstructure
- Describe cardiac muscle action potential
- Point out the functional importance of the action potential
- Follow the cardiac muscle mechanism of contraction
- Delineate cardiac muscle energy sources
- Outline the intracellular calcium homeostasis
- Explain the relationship between muscle length and tension of cardiac muscle (Frank-Starling law of the heart)



General plan of circulation

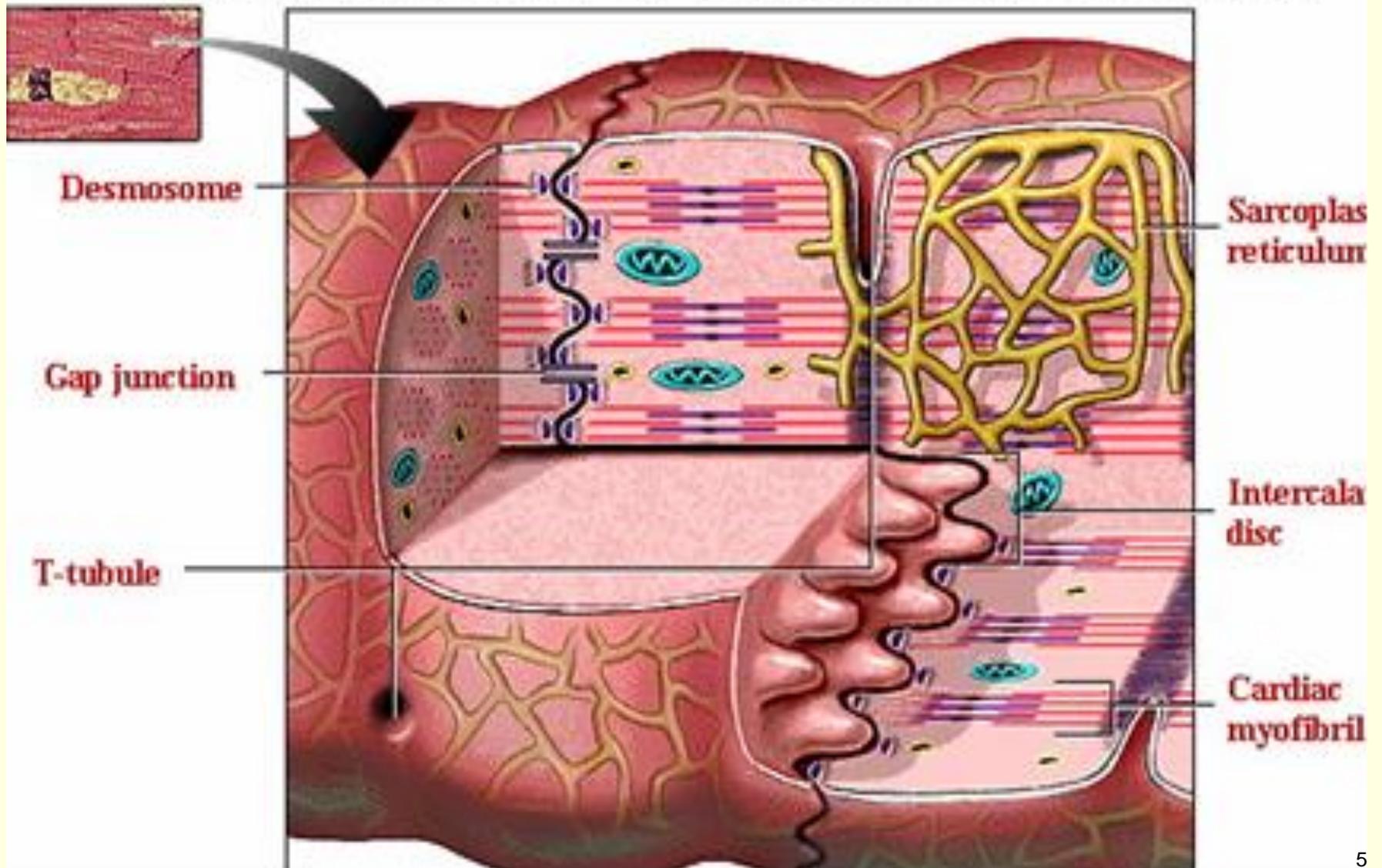


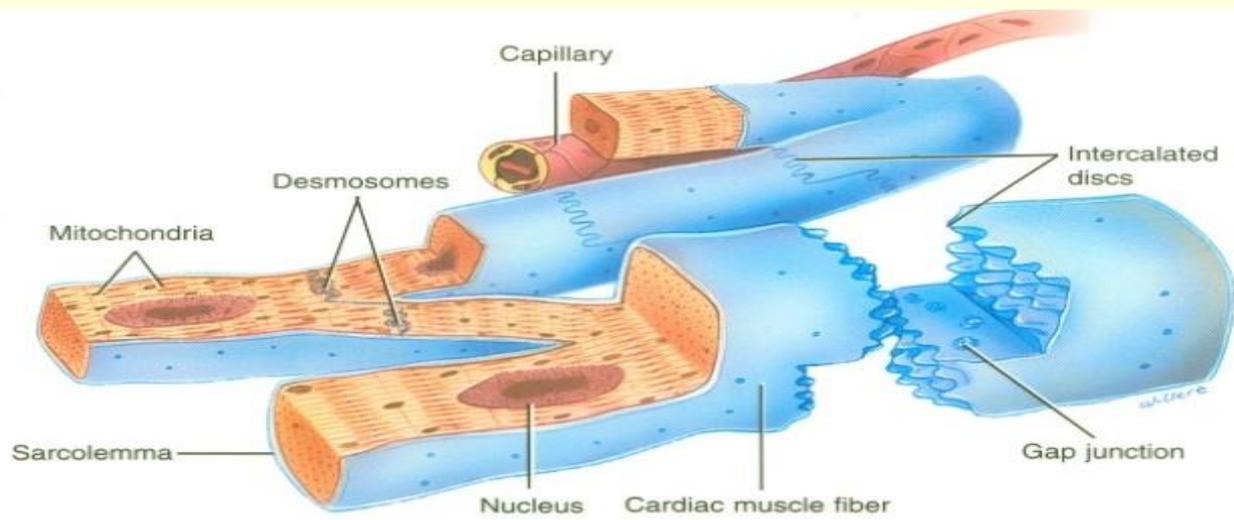
(a) Portion of pericardium and heart wall



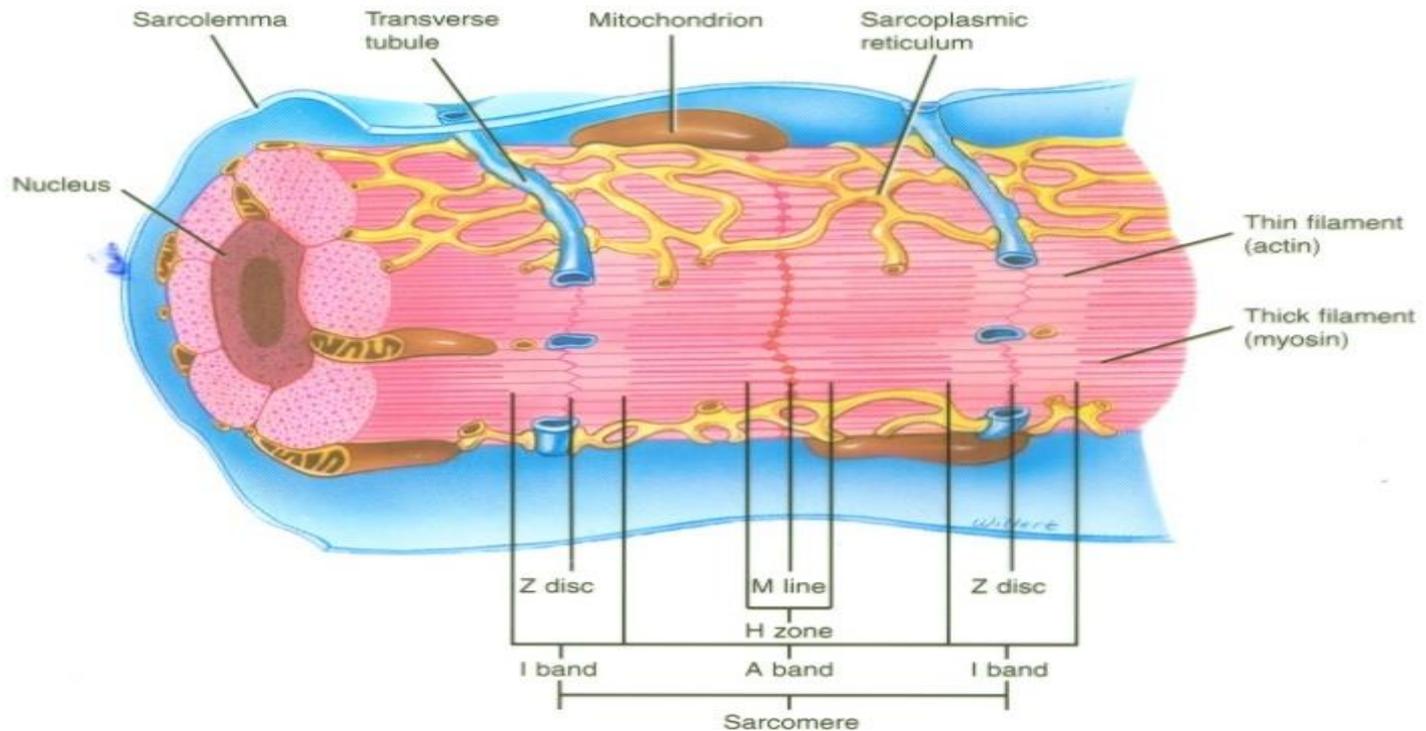
(b) Cardiac muscle fibers

# MAGNIFIED VIEW OF CARDIAC MUSCLE CELLS



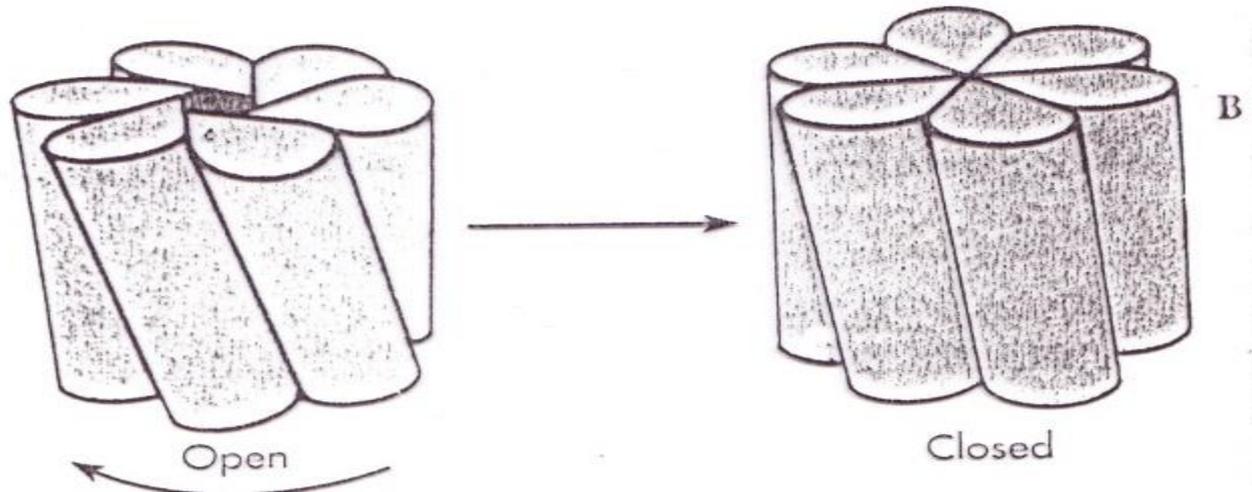
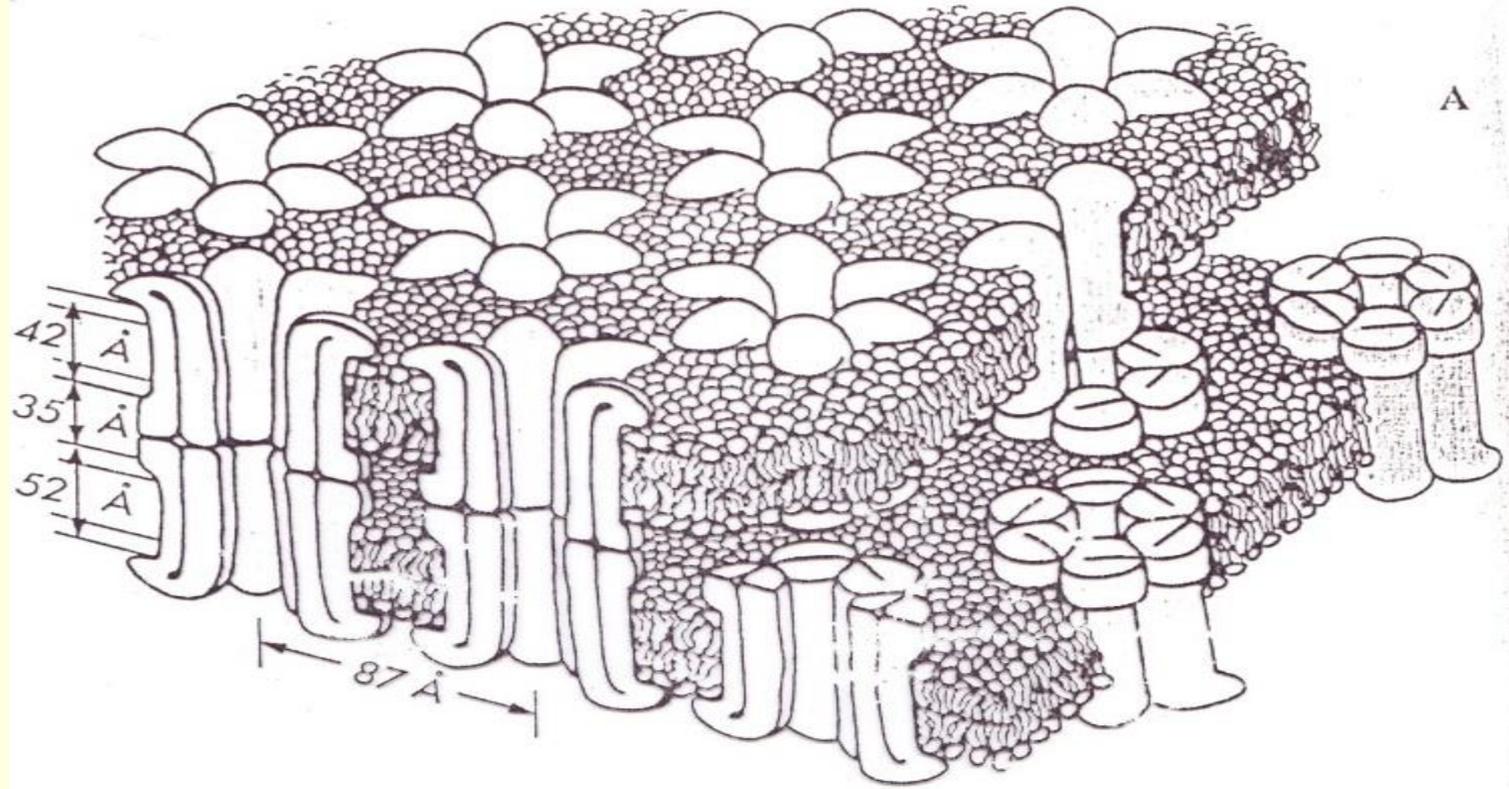


(a) Cardiac muscle fibers



(b) Diagram based on an electron micrograph

Gap junction channels

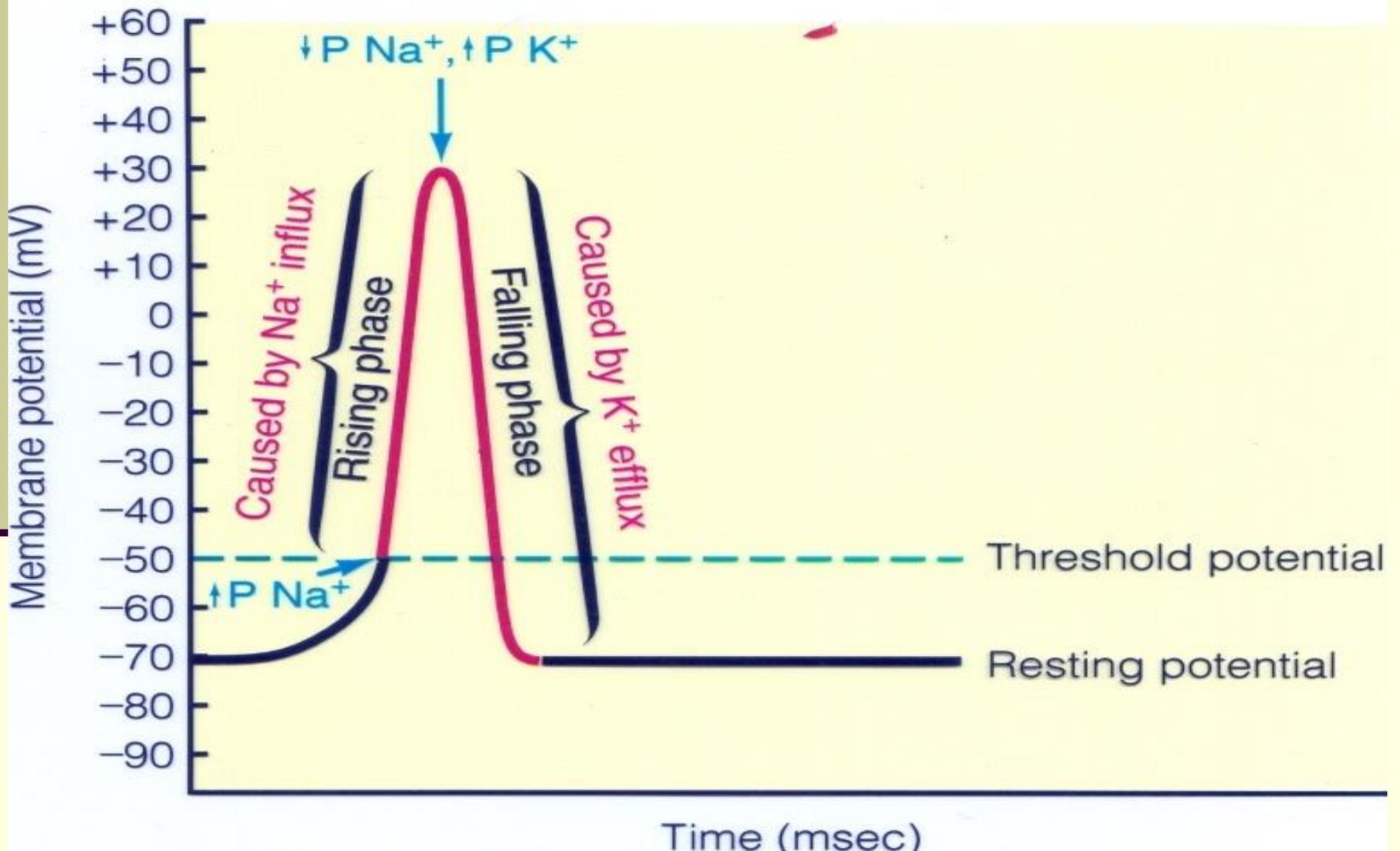


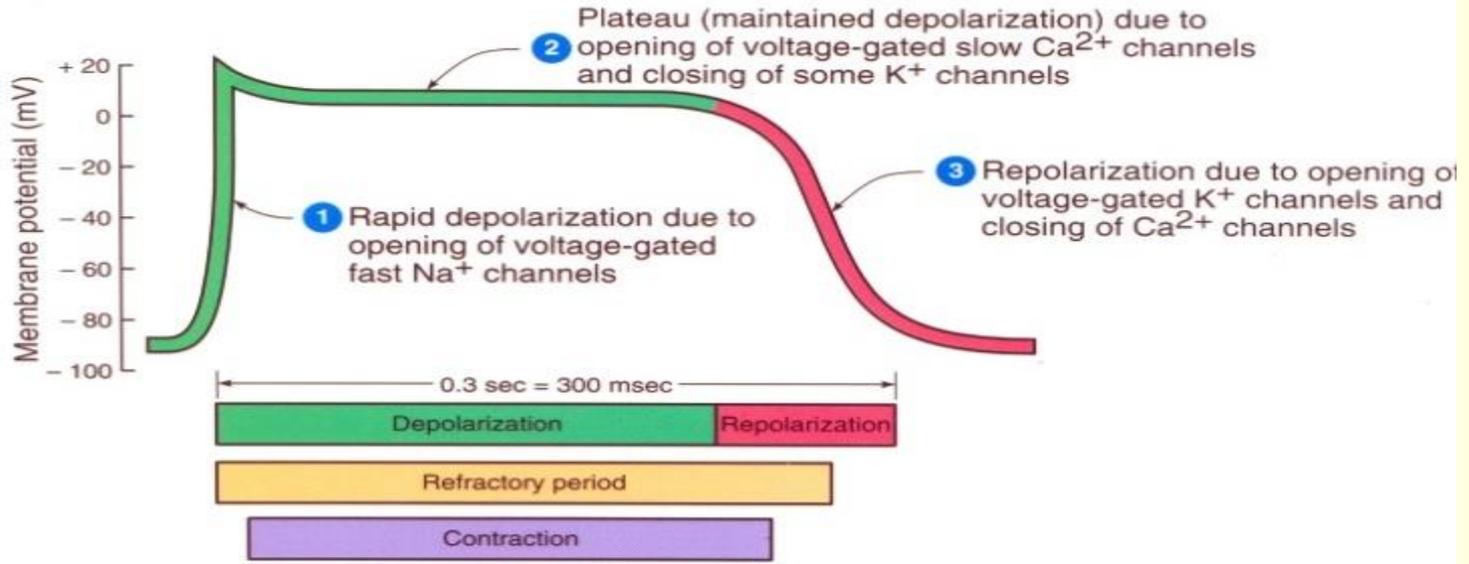
# Cardiac Muscle Vs Skeletal Muscle

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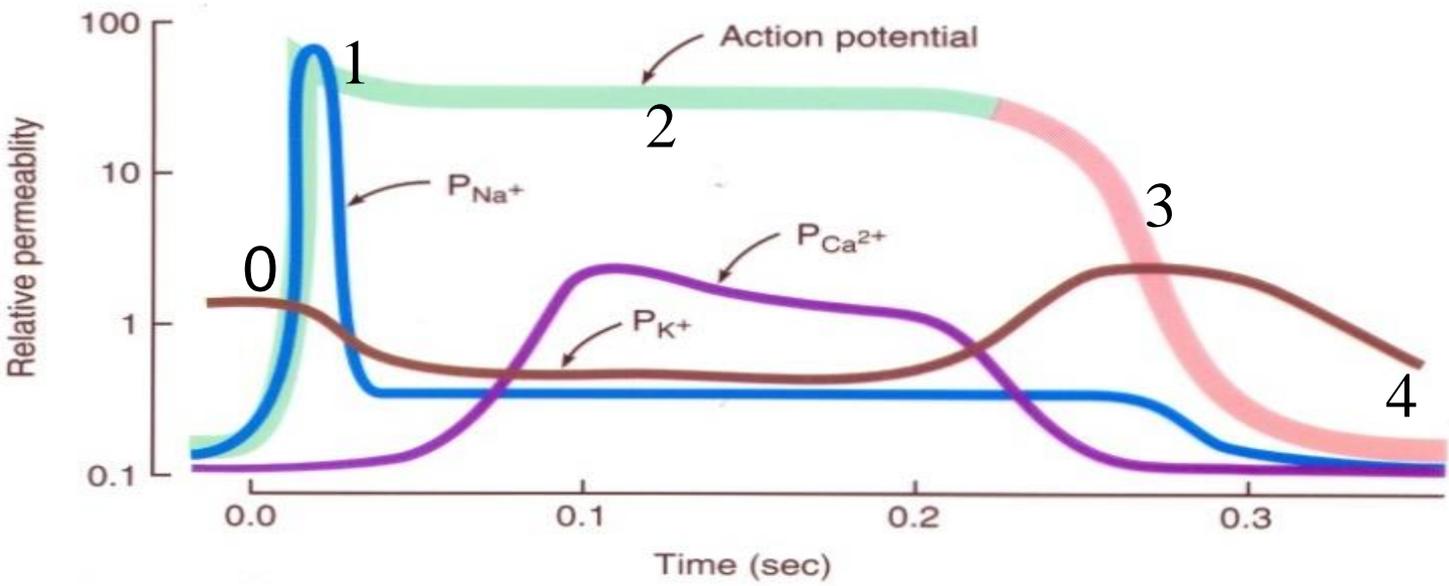
- ❖ Syncytium structure
- ❖ Gap Junction (electrical coupling) low resistance area
- ❖ Poorly developed Sarcoplasmic reticulum (SR)
- ❖ Transverse (T)Tubule on Z-line (i.e. One T-tubule per sarcomere)
- ❖ Rich in mitochondria
- ❖ Low in nuclei

# Permeability Changes and Ionic Fluxes During an Action Potential (skeletal Muscle)



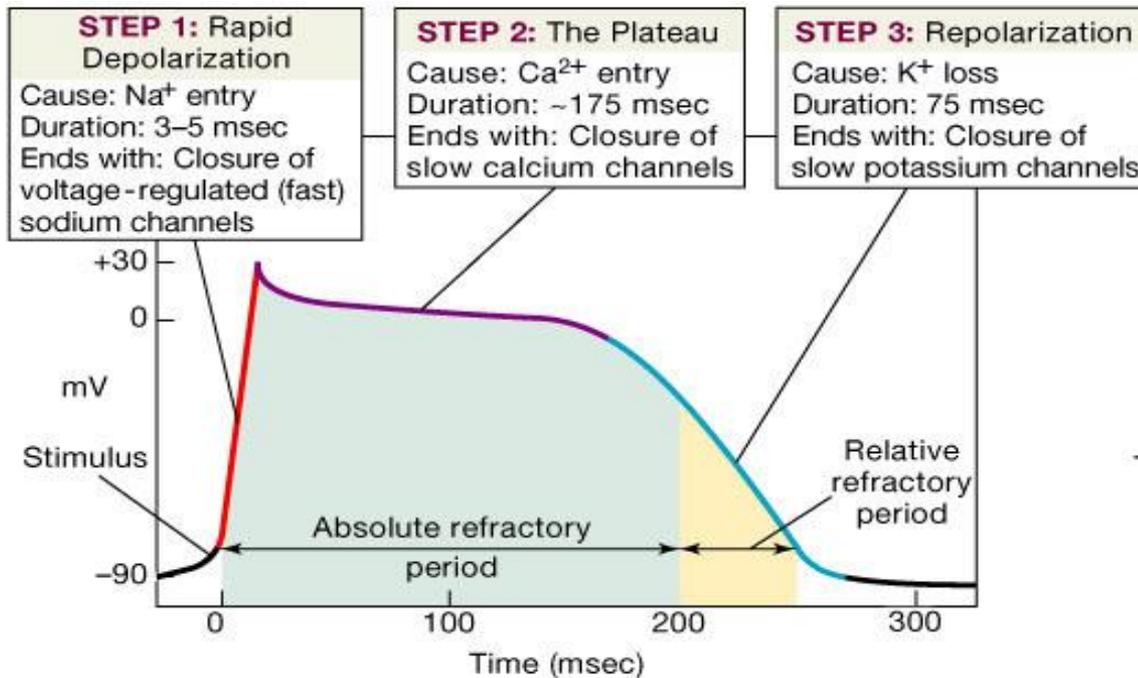


(a) Action potential, refractory period, and contraction

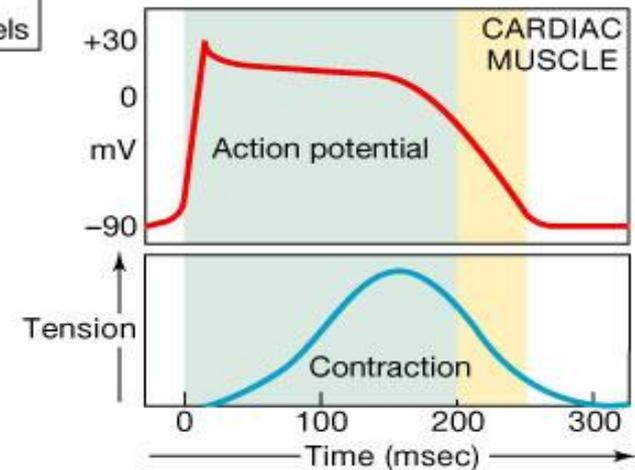
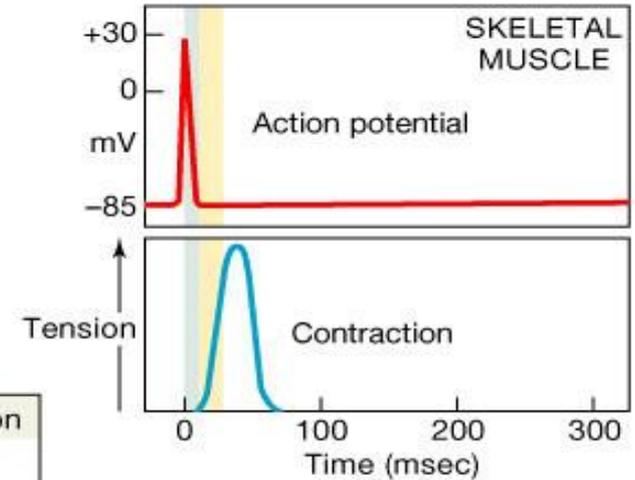


(b) Membrane permeability (P) changes

# The Action Potential in Skeletal and Cardiac Muscle



(a) Cardiac muscle

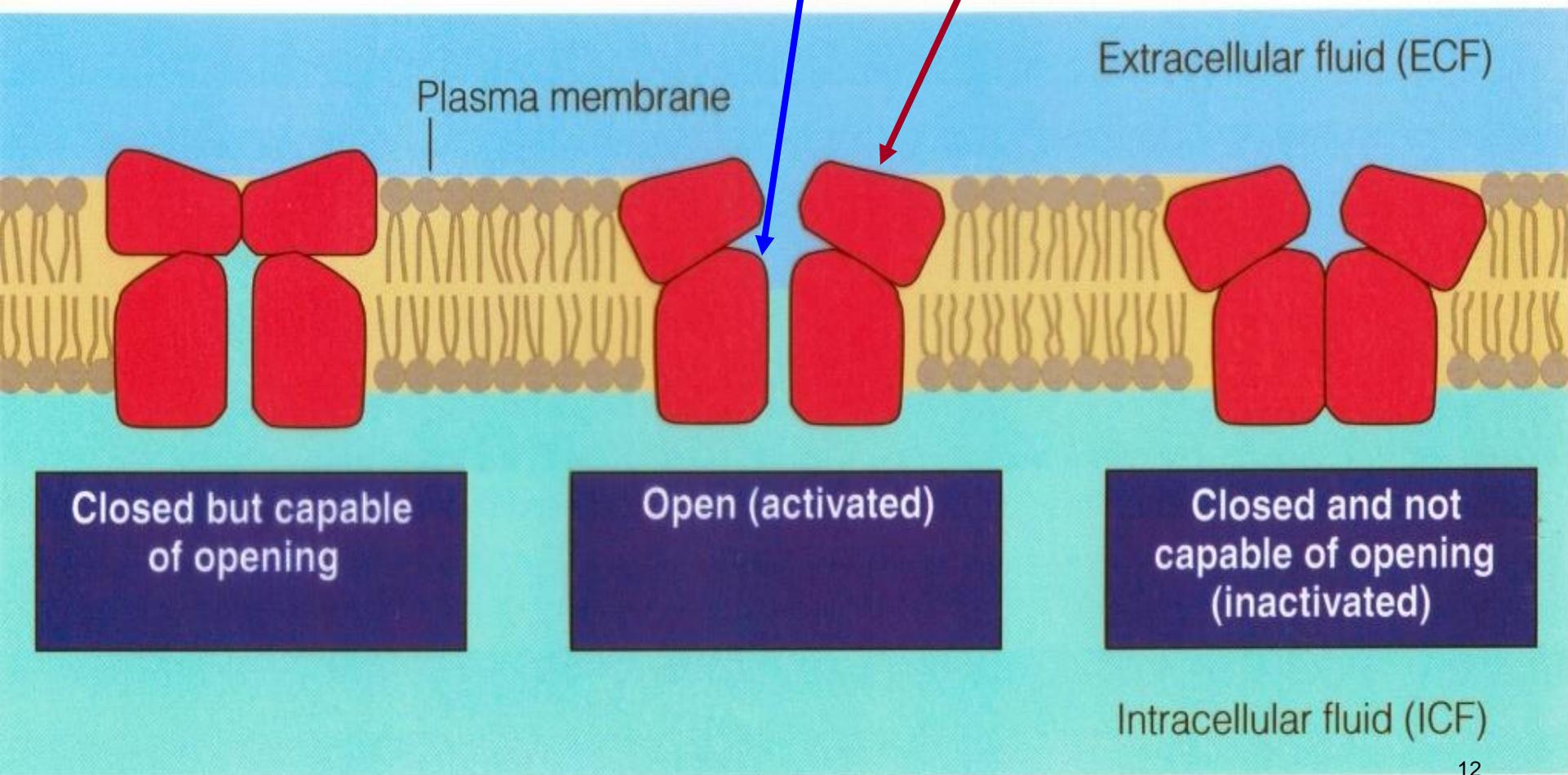


(b)

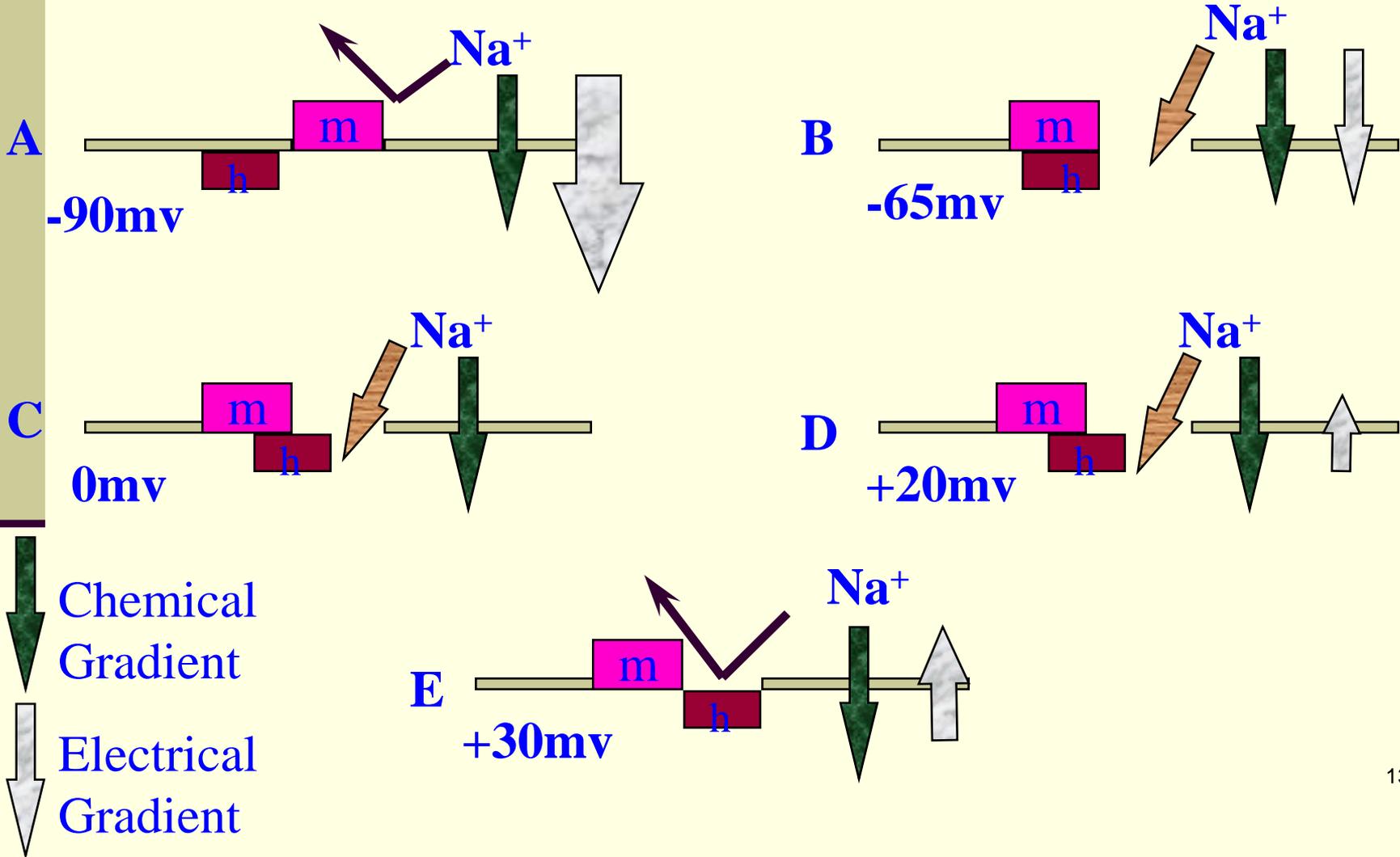
## Conformations of a Voltage-Gated Na<sup>+</sup> Channel

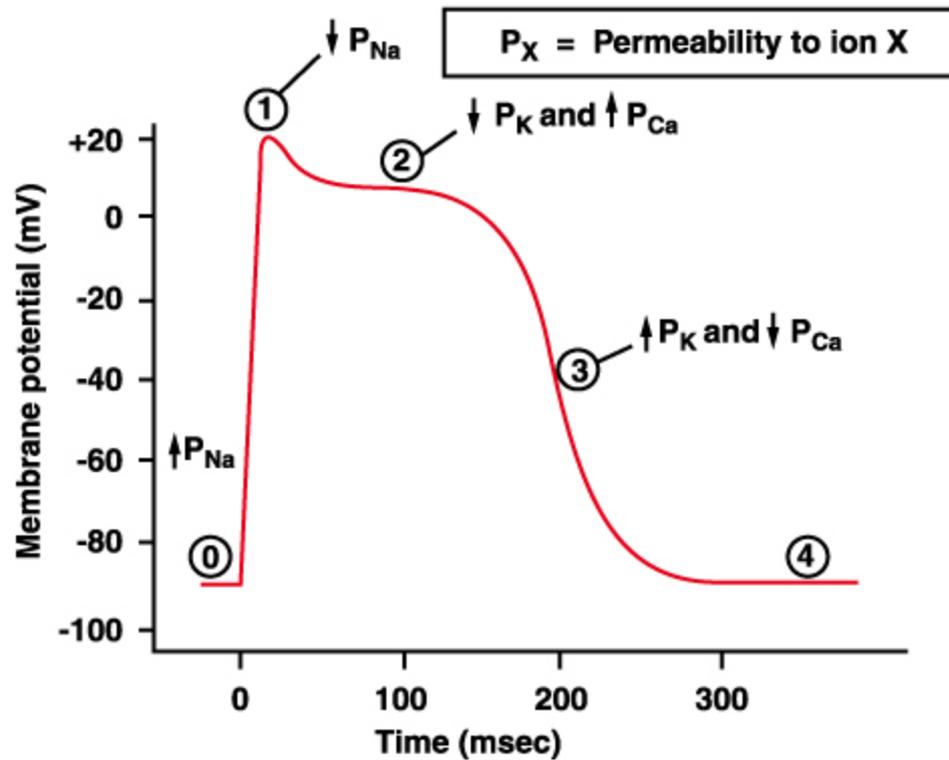
(inactivation gate) h Gate

(activation gate) m Gate

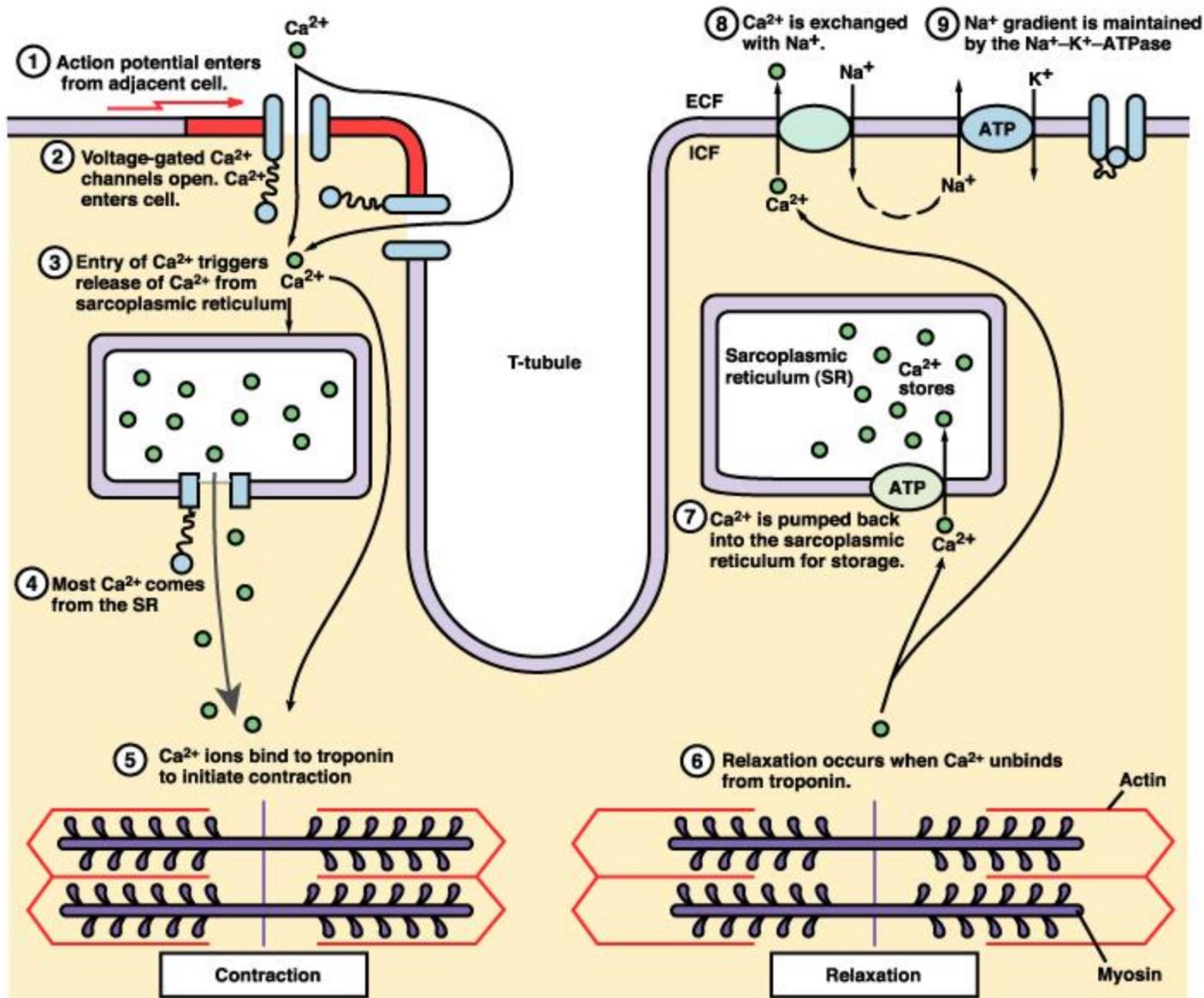


# PHASE 0 OF THE FAST FIBER ACTION POTENTIAL

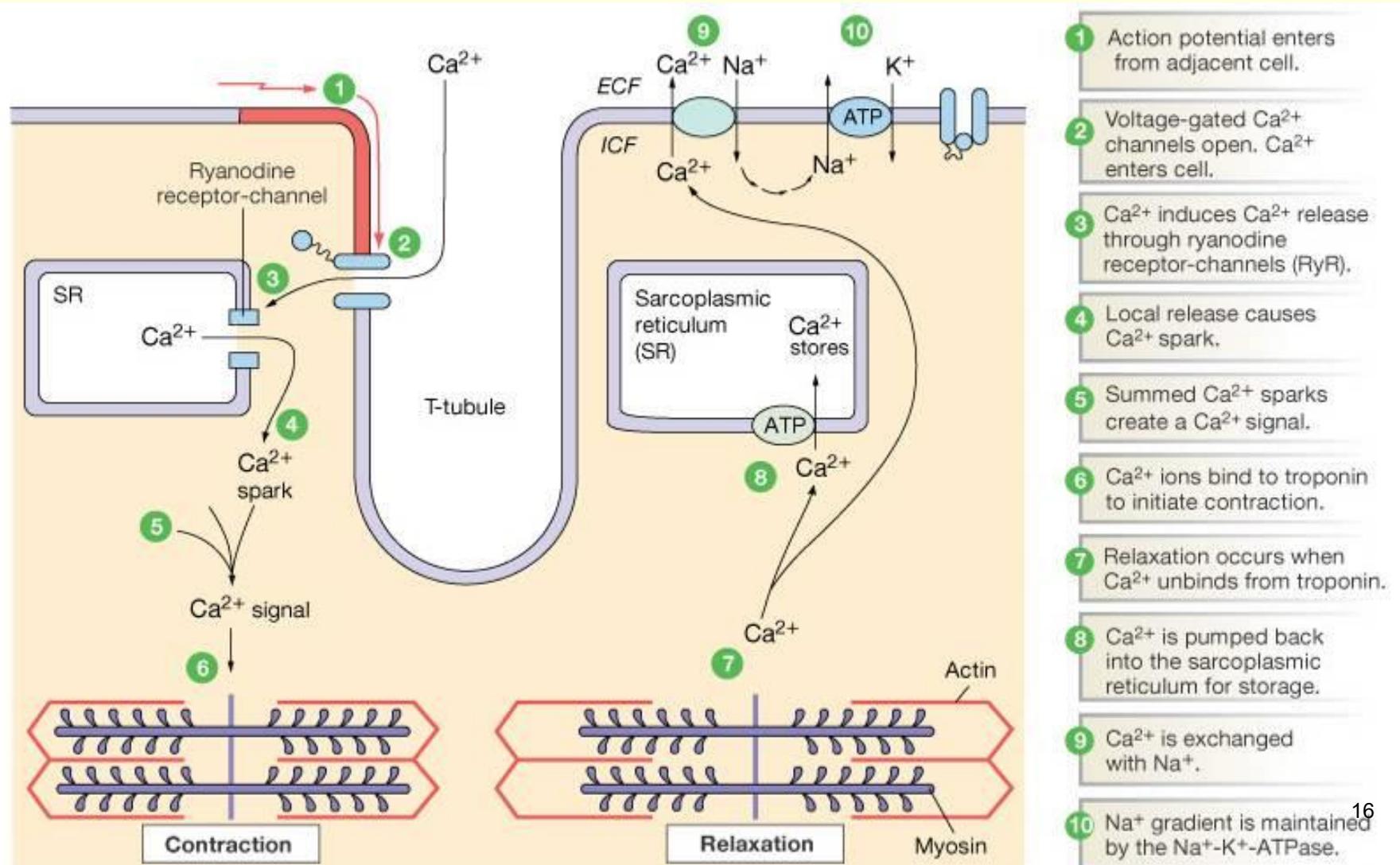




Phase	Membrane channels
①	$Na^+$ channels open
②	$Na^+$ channels close
③	$Ca^{2+}$ channels open; fast $K^+$ channels close
④	$Ca^{2+}$ channels close; slow $K^+$ channels open
⑤	Resting potential

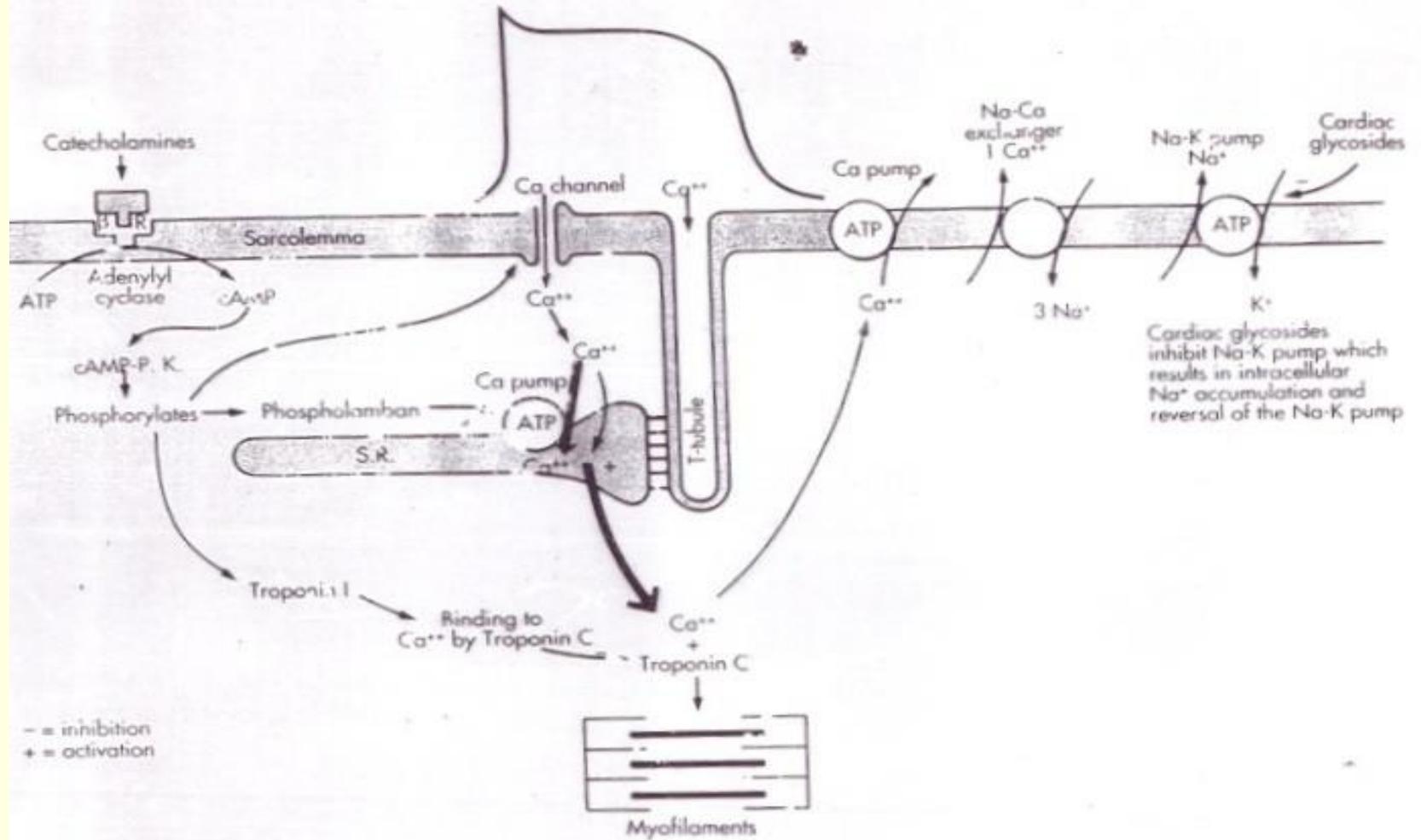


# Mechanism of Cardiac Muscle Excitation, Contraction & Relaxation

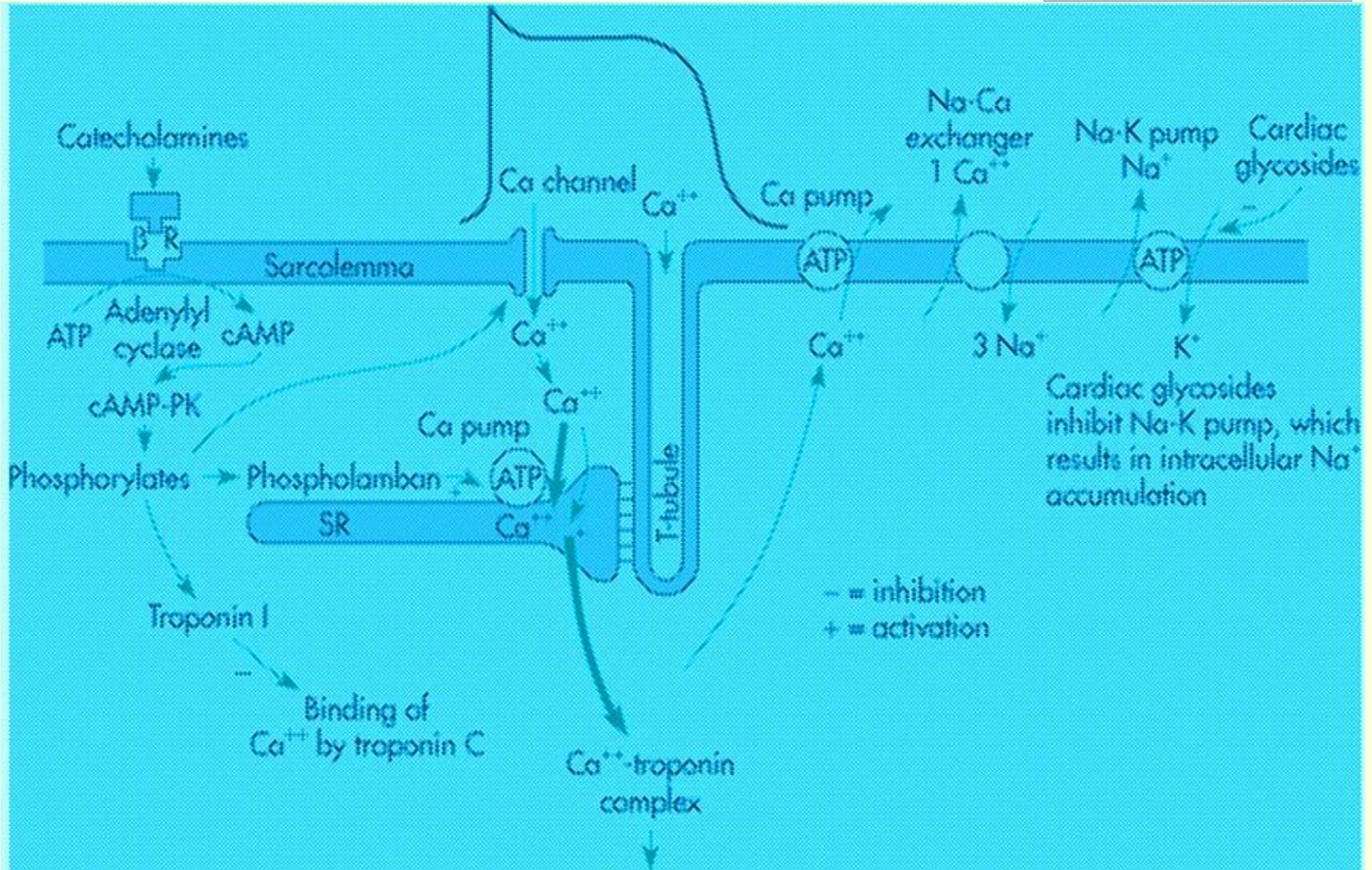


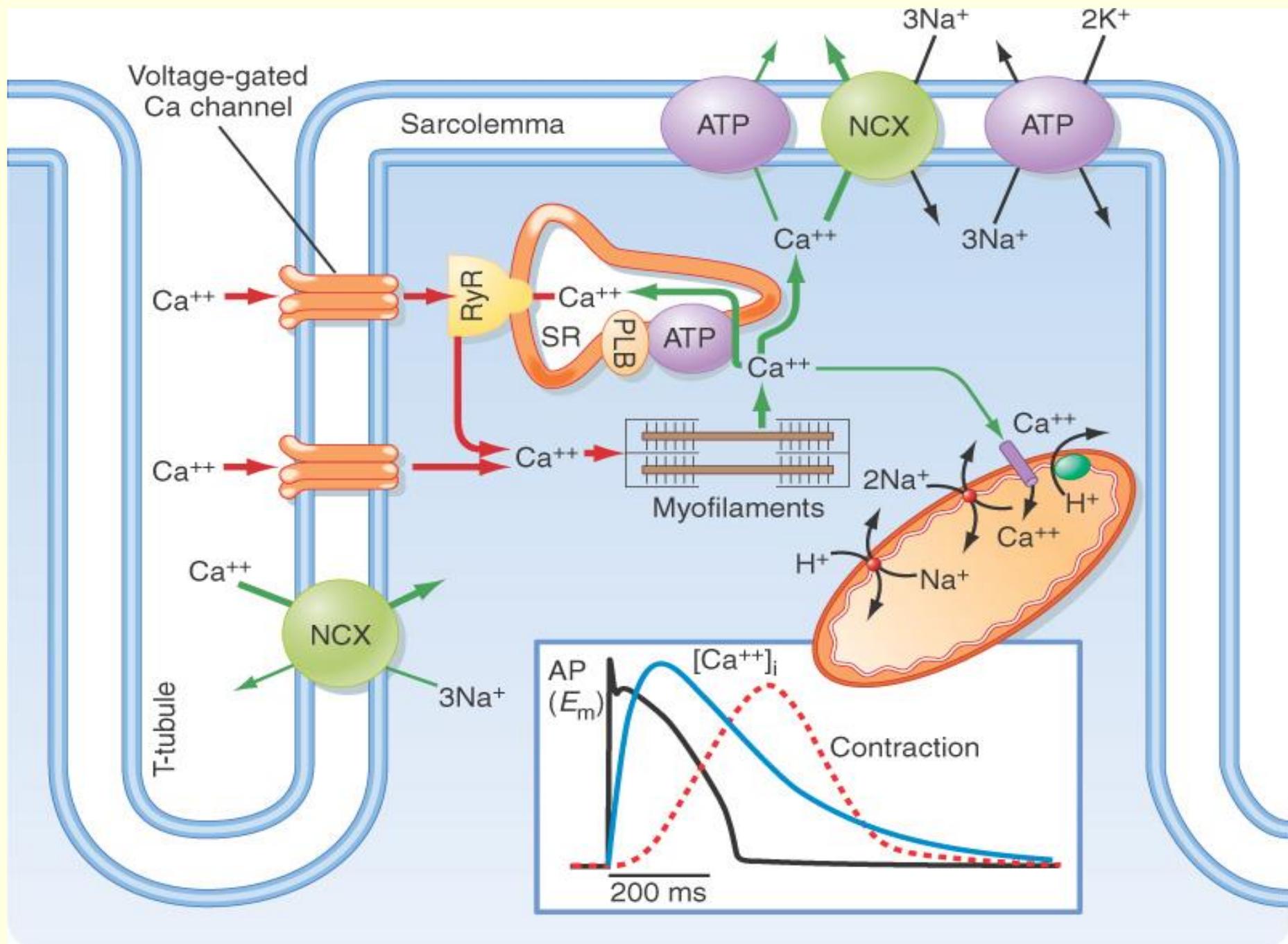
- 1 Action potential enters from adjacent cell.
- 2 Voltage-gated  $\text{Ca}^{2+}$  channels open.  $\text{Ca}^{2+}$  enters cell.
- 3  $\text{Ca}^{2+}$  induces  $\text{Ca}^{2+}$  release through ryanodine receptor-channels (RyR).
- 4 Local release causes  $\text{Ca}^{2+}$  spark.
- 5 Summed  $\text{Ca}^{2+}$  sparks create a  $\text{Ca}^{2+}$  signal.
- 6  $\text{Ca}^{2+}$  ions bind to troponin to initiate contraction.
- 7 Relaxation occurs when  $\text{Ca}^{2+}$  unbinds from troponin.
- 8  $\text{Ca}^{2+}$  is pumped back into the sarcoplasmic reticulum for storage.
- 9  $\text{Ca}^{2+}$  is exchanged with  $\text{Na}^{+}$ .
- 10  $\text{Na}^{+}$  gradient is maintained by the Na<sup>+</sup>-K<sup>+</sup>-ATPase.

# Intracellular Calcium Homeostasis...1

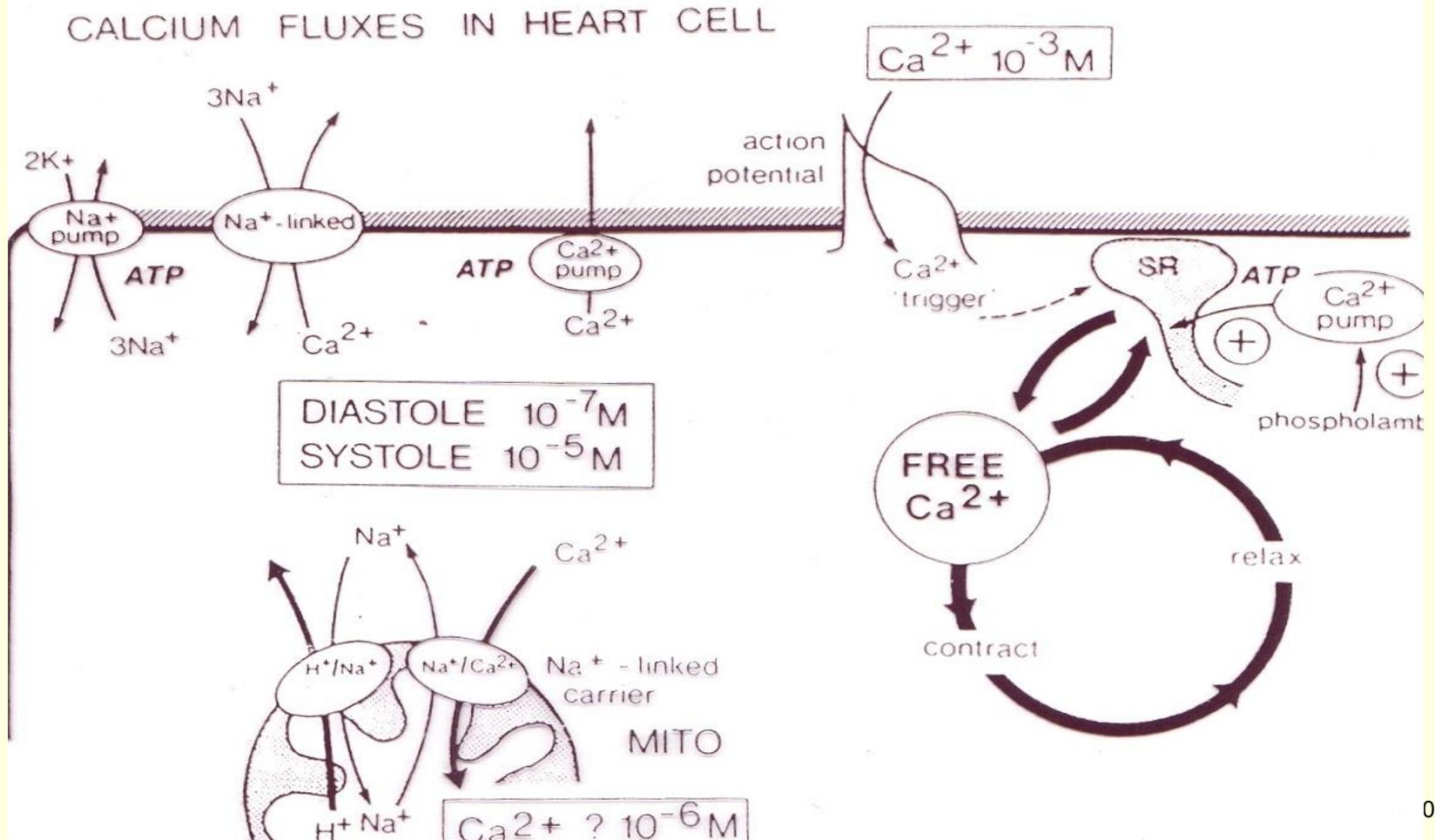


# Intracellular Calcium Homeostasis...1

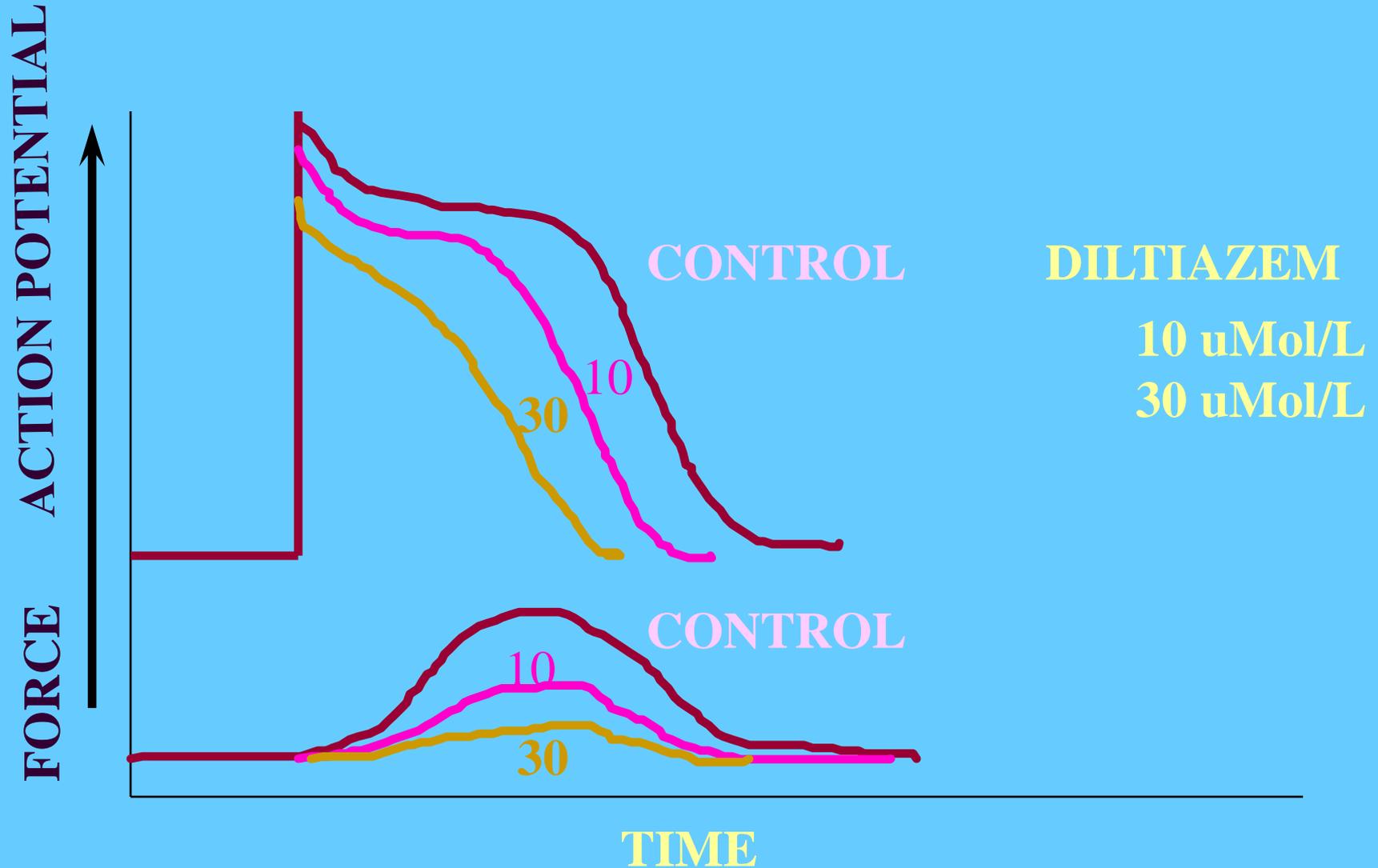




# Intracellular Calcium Homeostasis...2



# EFFECTS OF Ca<sup>++</sup> CHANNEL BLOCKERS AND THE CARDIAC CELL ACTION POTENTIAL

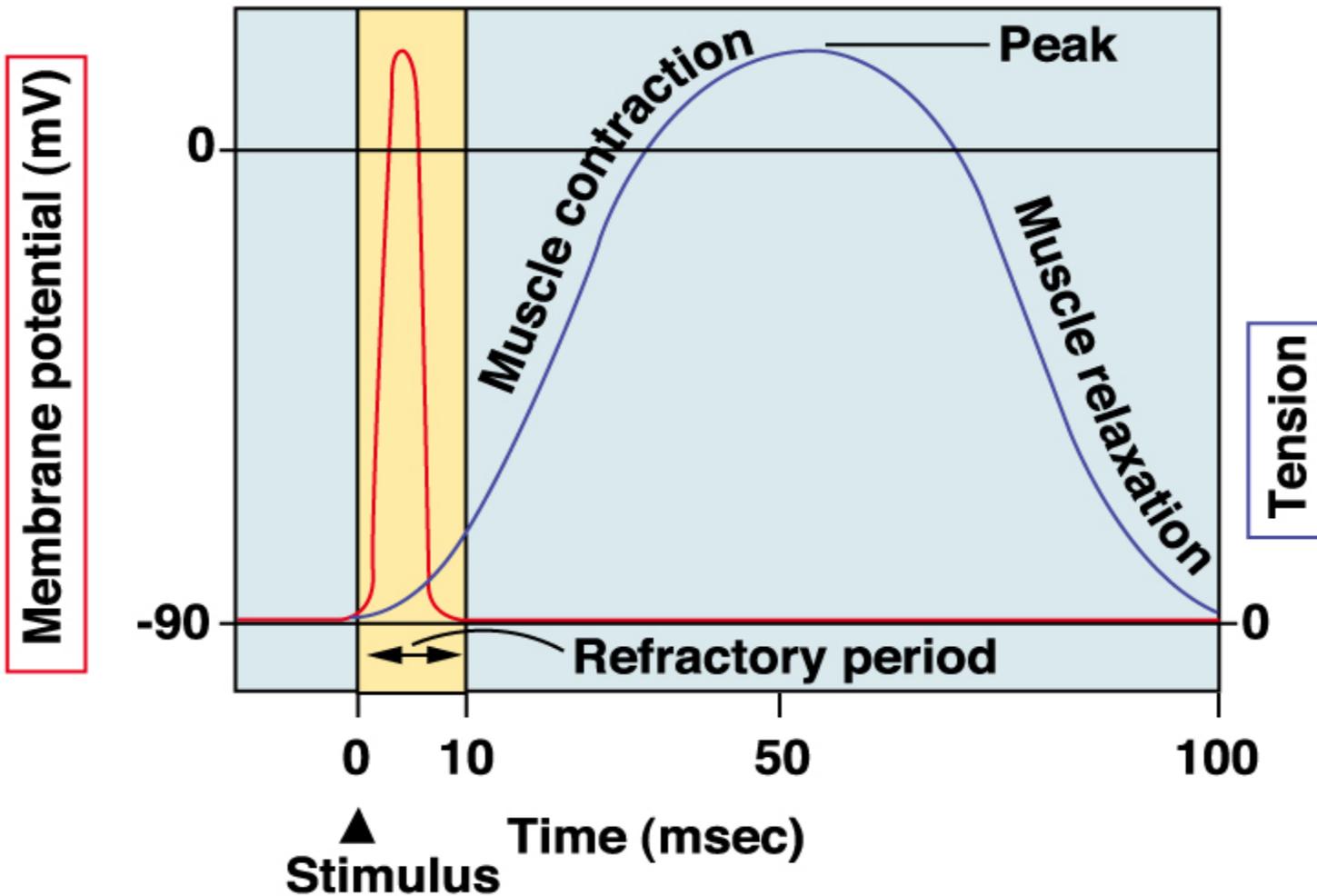


# Cardiac Muscle action potential Vs. Skeletal Muscle

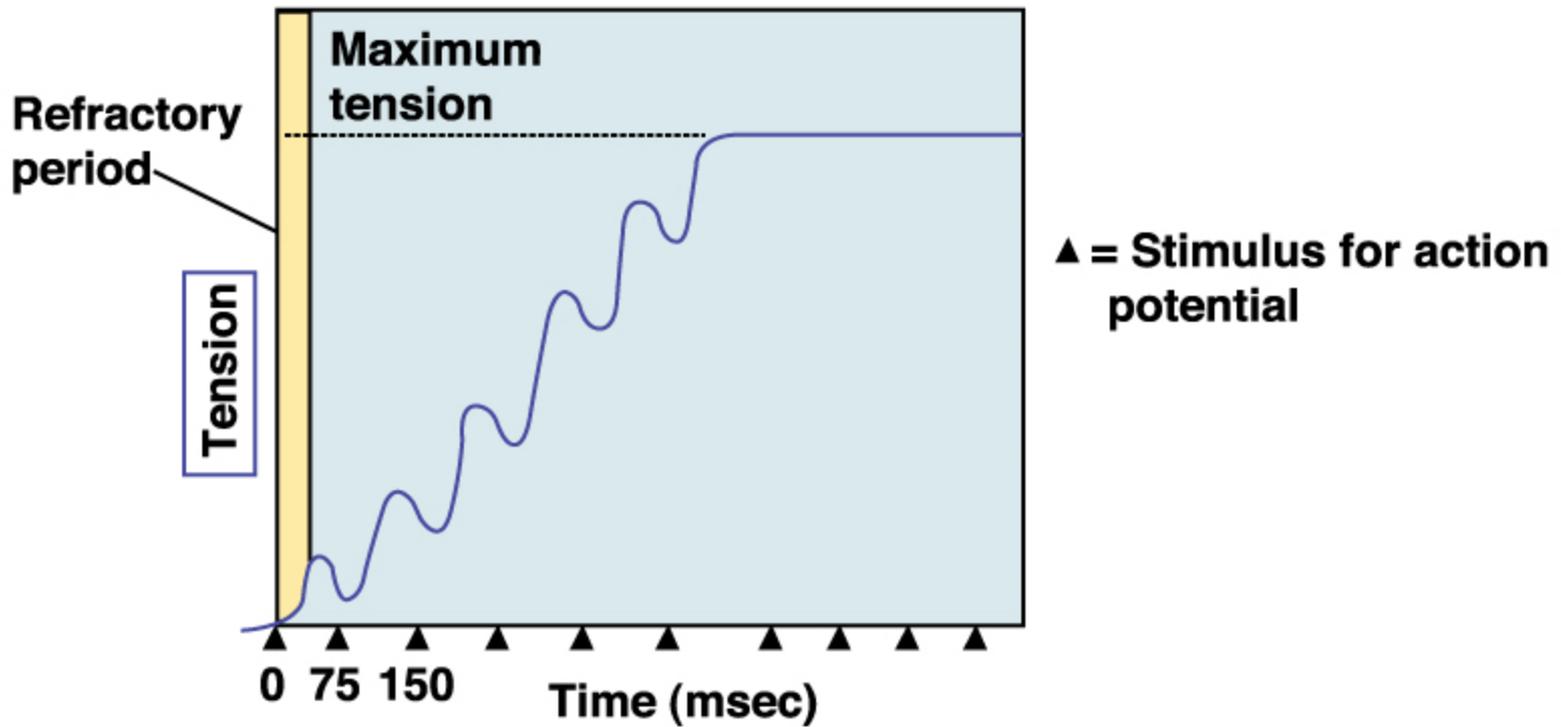
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- Phase 0 –Depolarization phase ( $\text{Na}^+$  influx)
- Phase 1 partial repolarization (Not in skeletal)
- Phase 2 Plateau (depolarization not in skeletal) slow calcium channels
- Phase 3 fast repolarization phase ( $\text{K}^+$  efflux)
- Phase 4 resting membrane potential

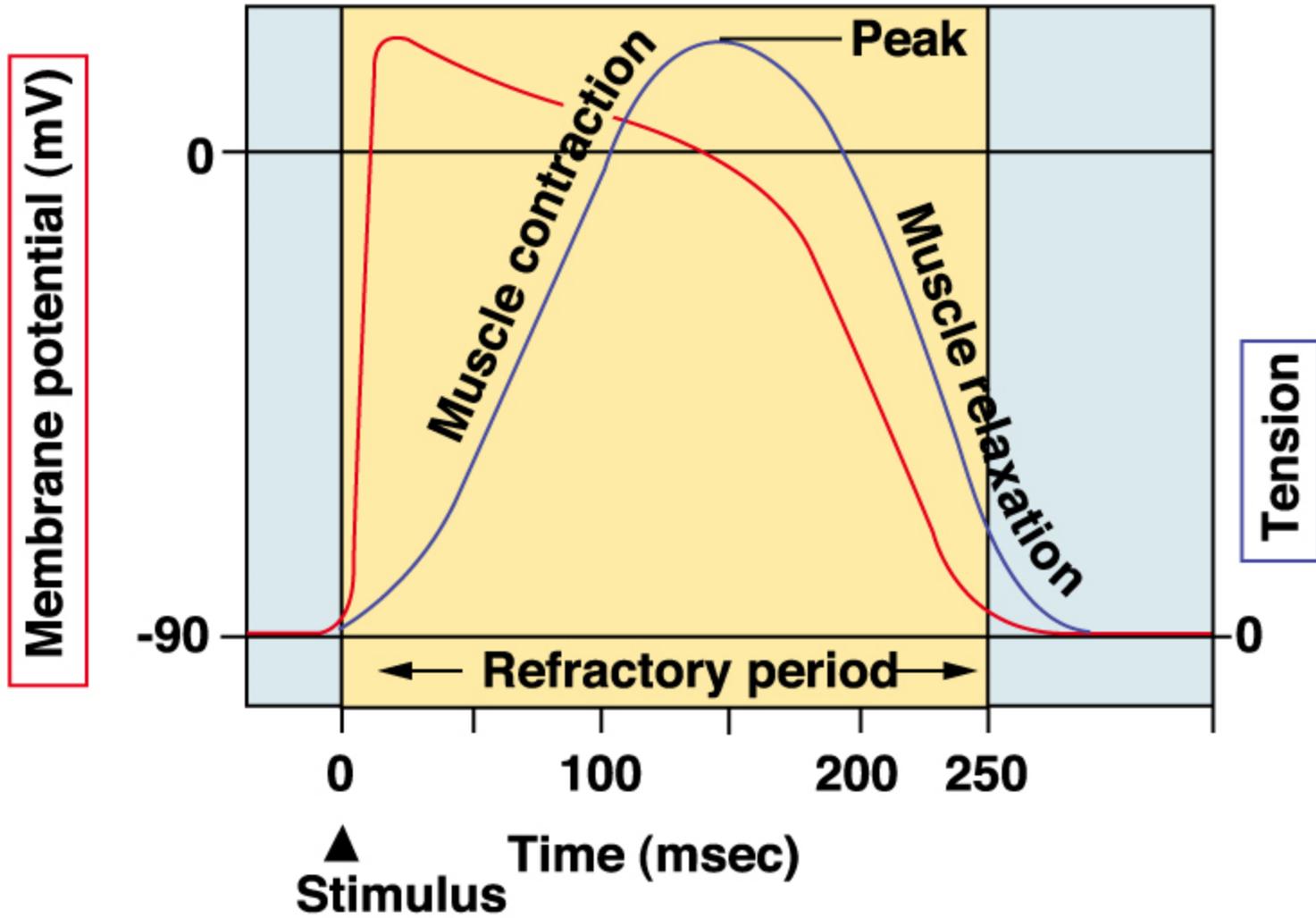
## Skeletal muscle fast-twitch fiber



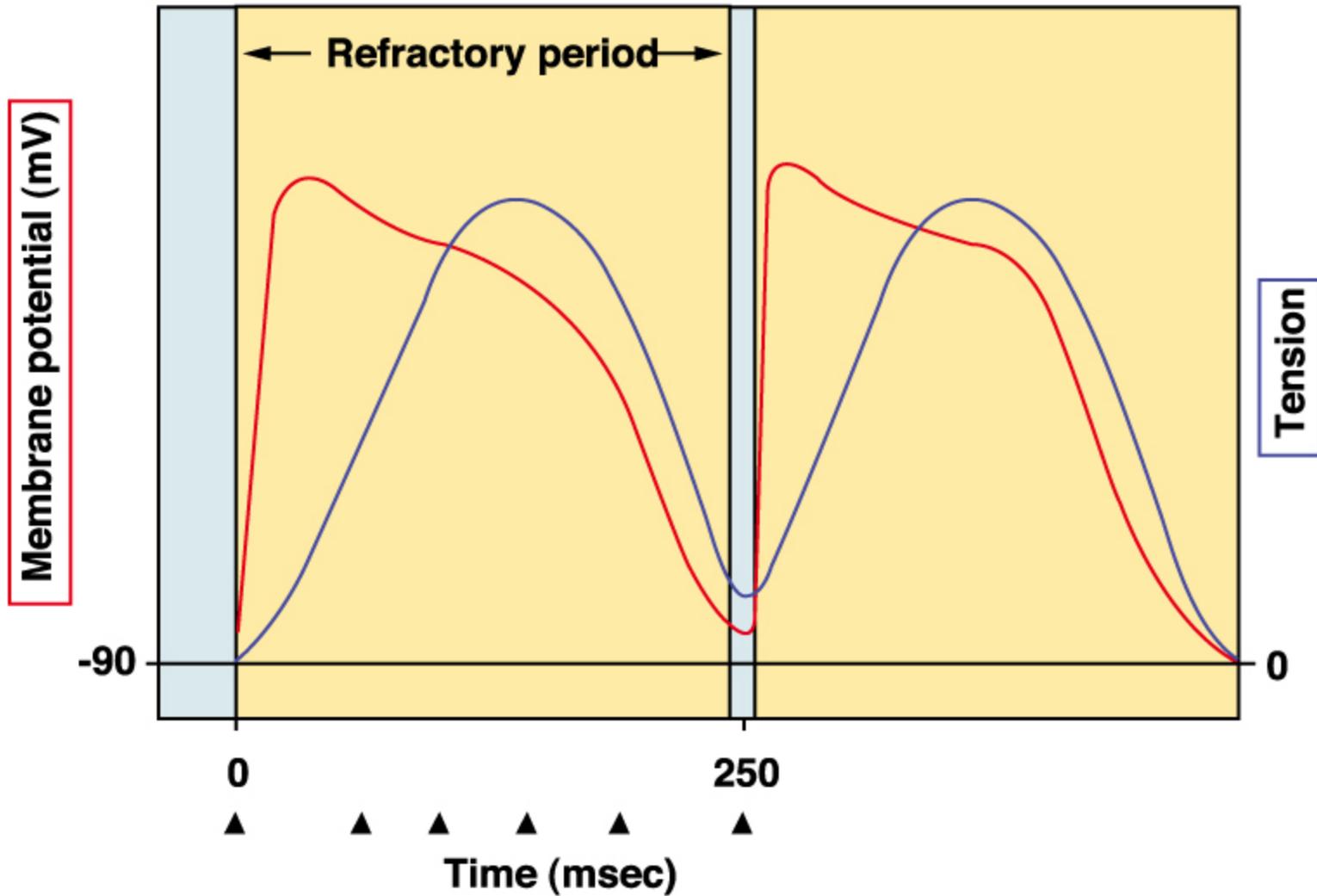
**Tetanus in a skeletal muscle.  
Action potentials not shown.**

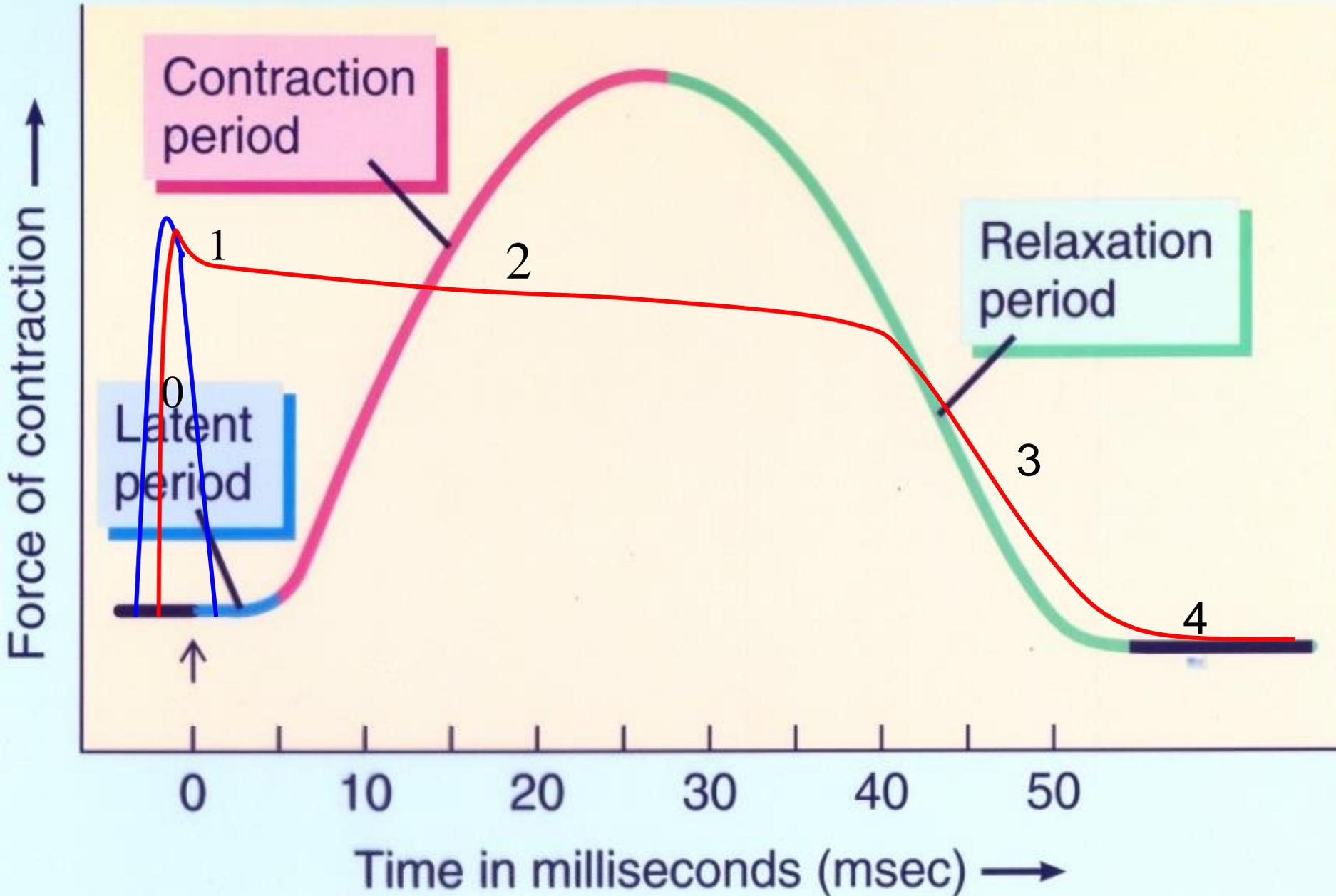


# Cardiac muscle fiber

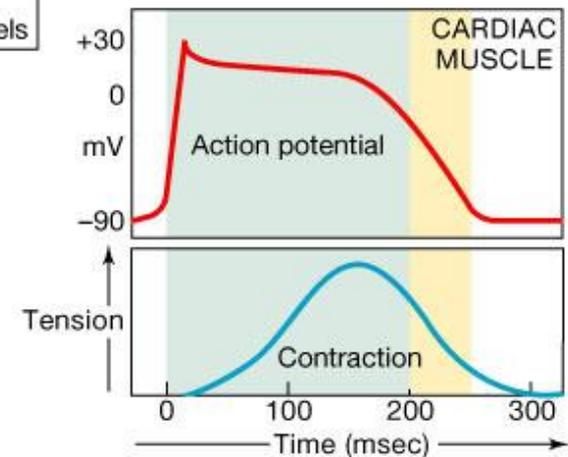
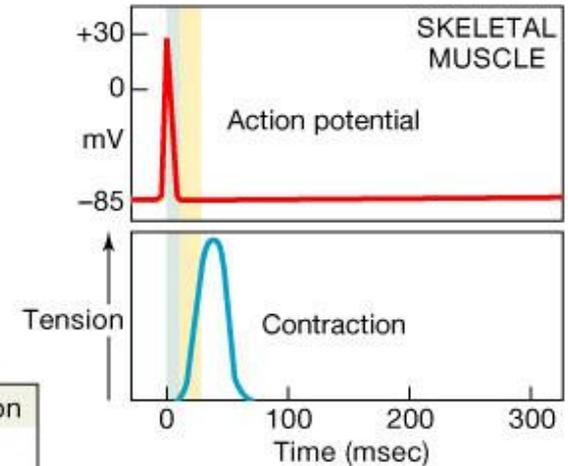
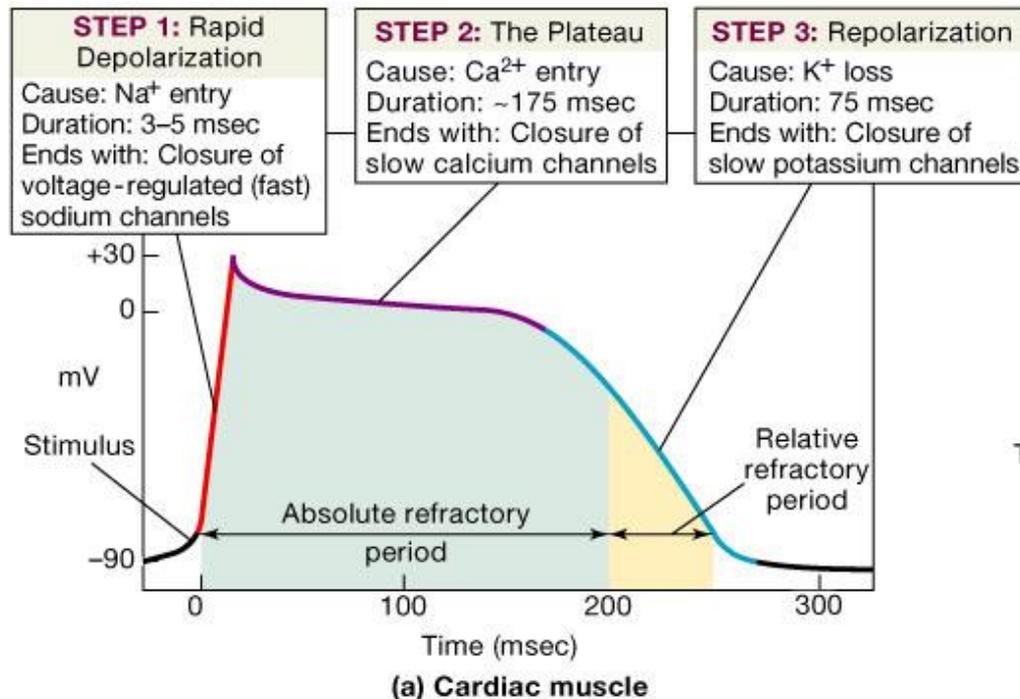


Long refractory period in a cardiac muscle prevents tetanus.

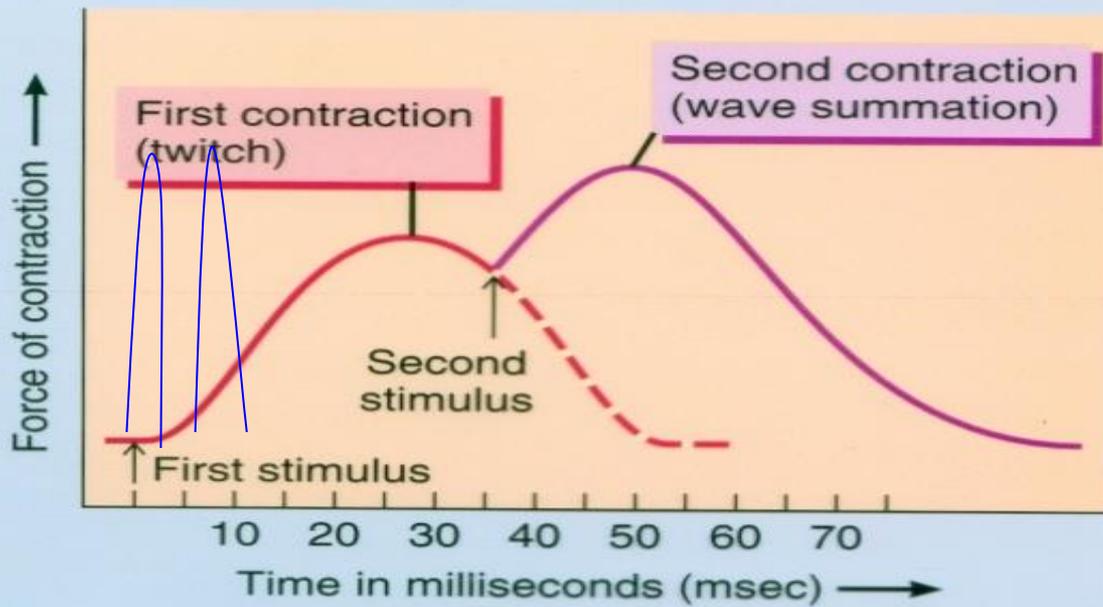




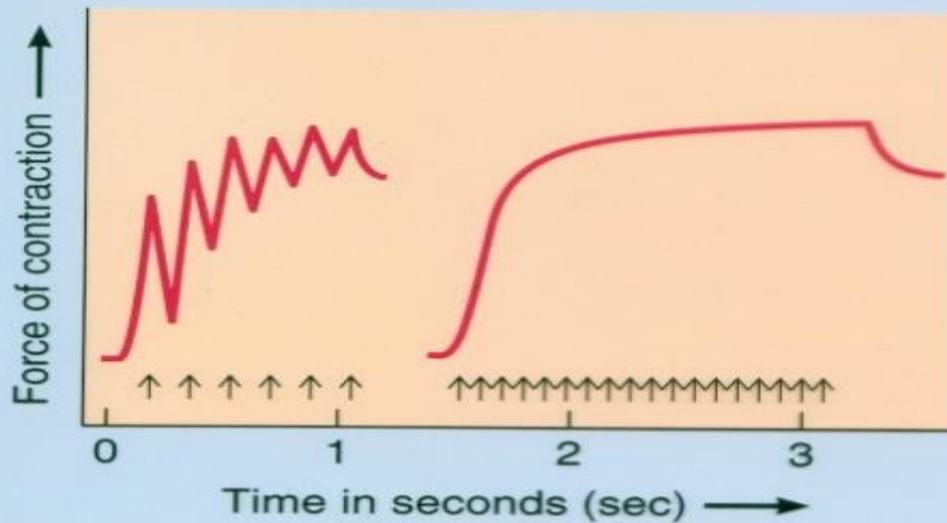
# The Action Potential in Skeletal and Cardiac Muscle



**(b)**

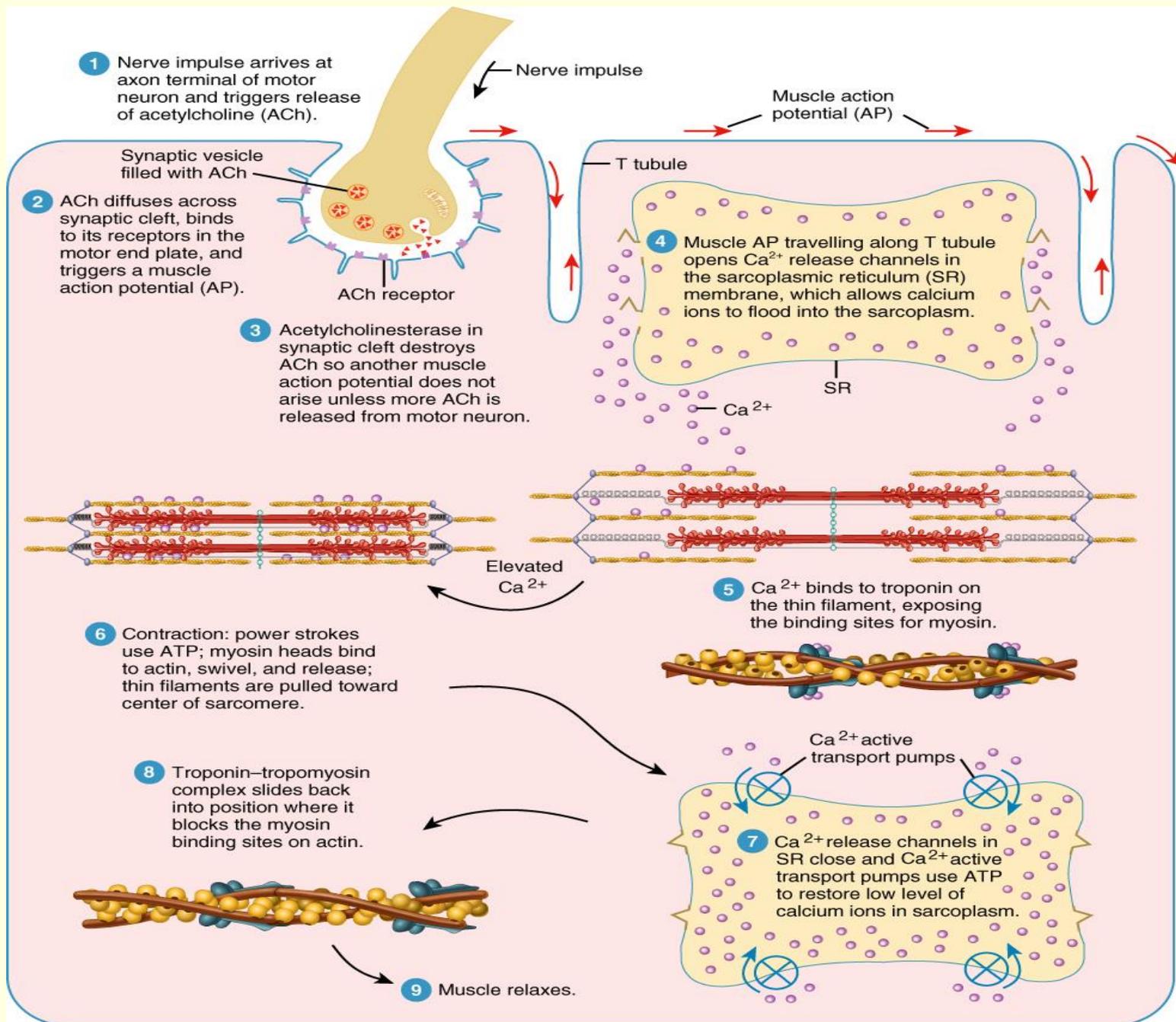


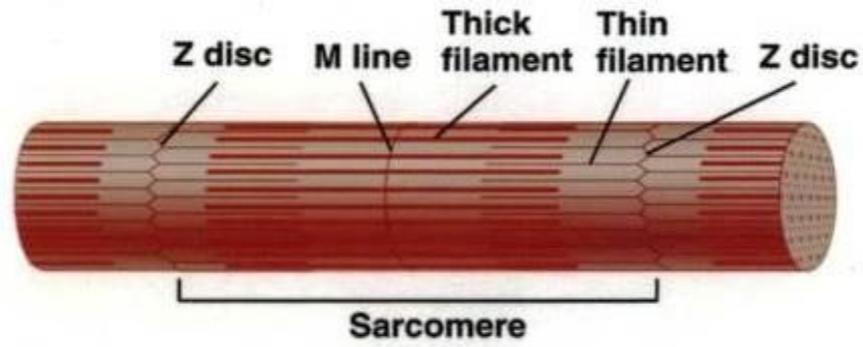
(a) Wave summation



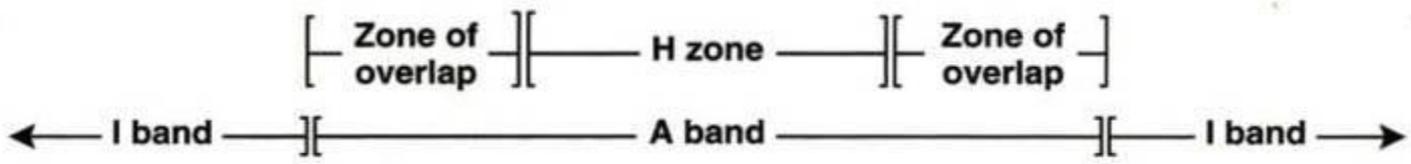
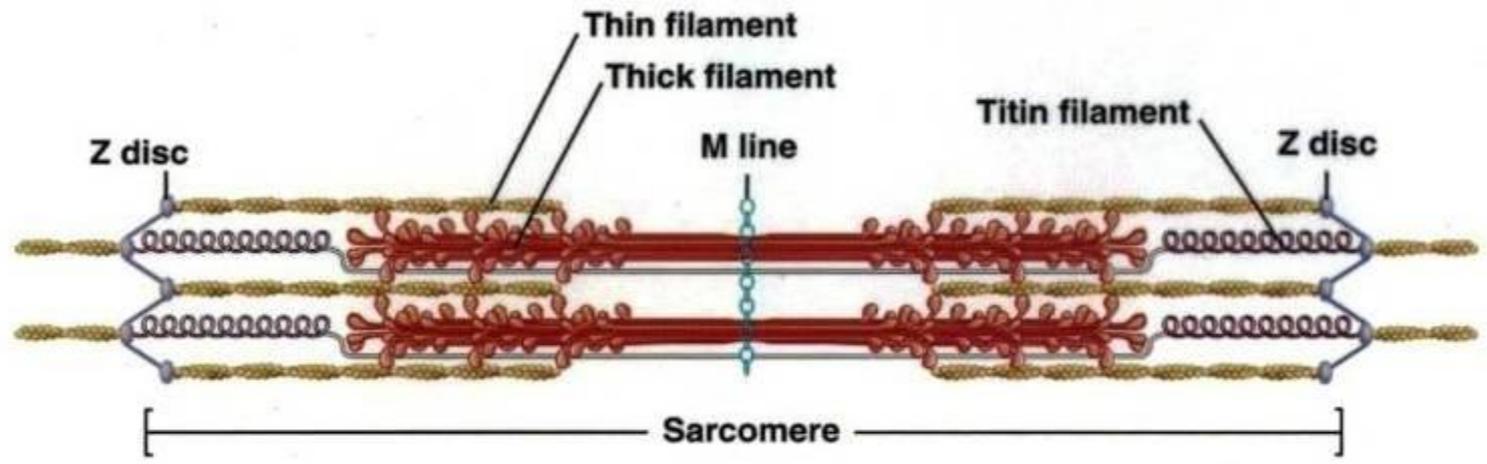
(b) Incomplete tetanus

(c) Complete tetanus

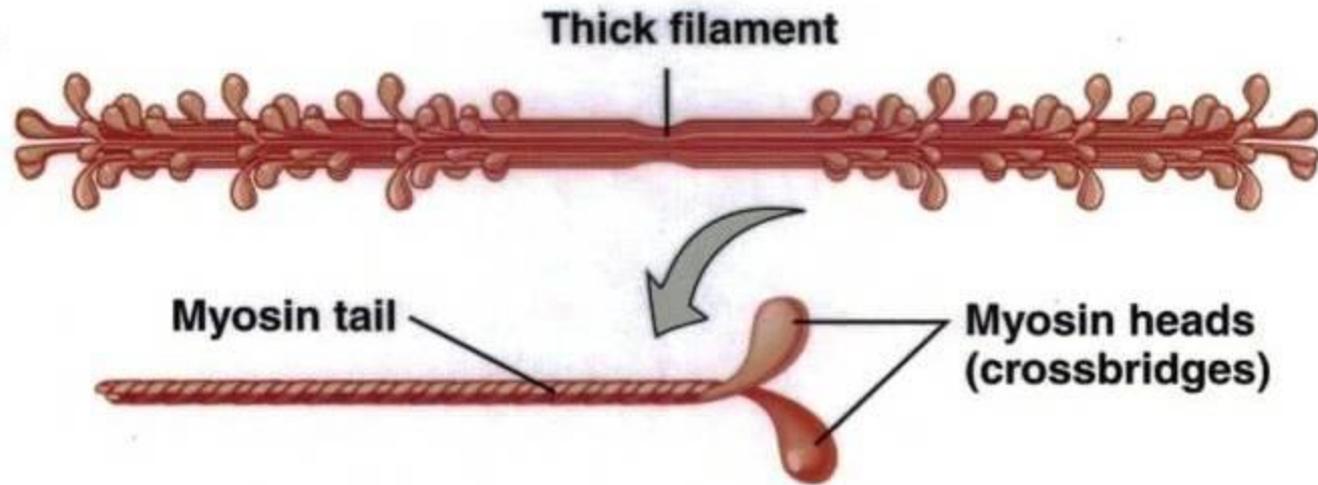




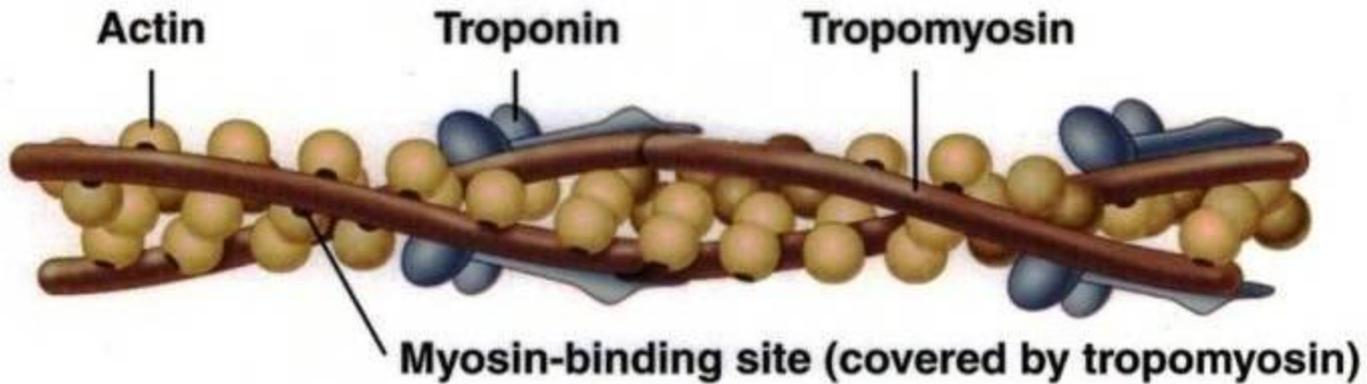
(a) Myofibril



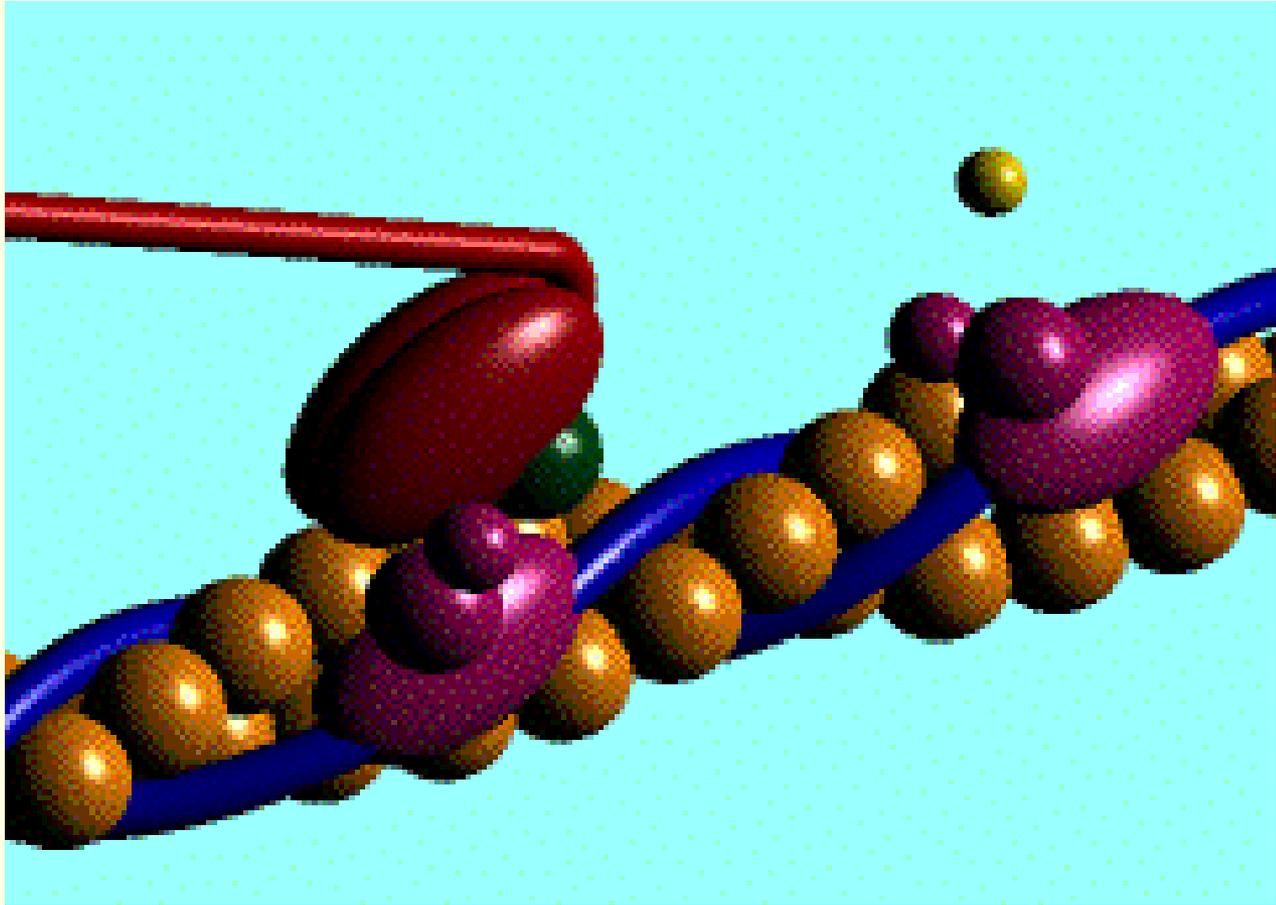
(b) Filaments

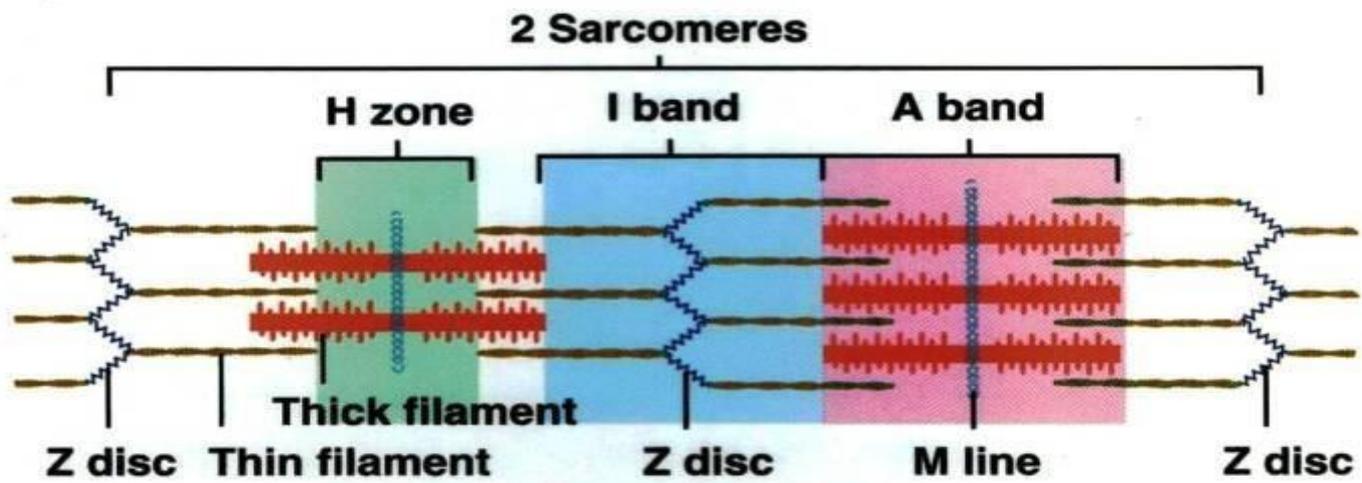


(a) One thick filament (above) and a myosin molecule (below)

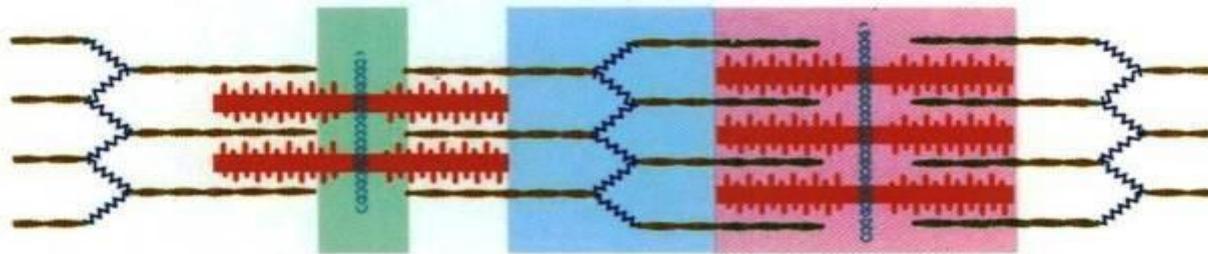


(b) Portion of a thin filament

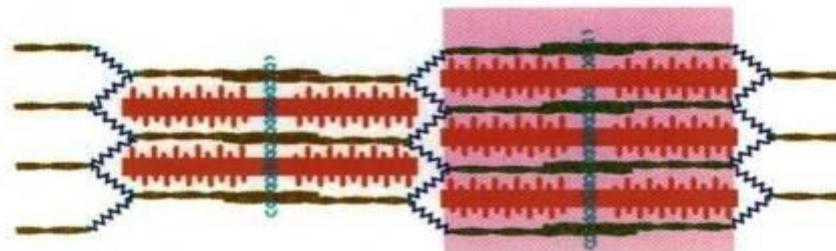




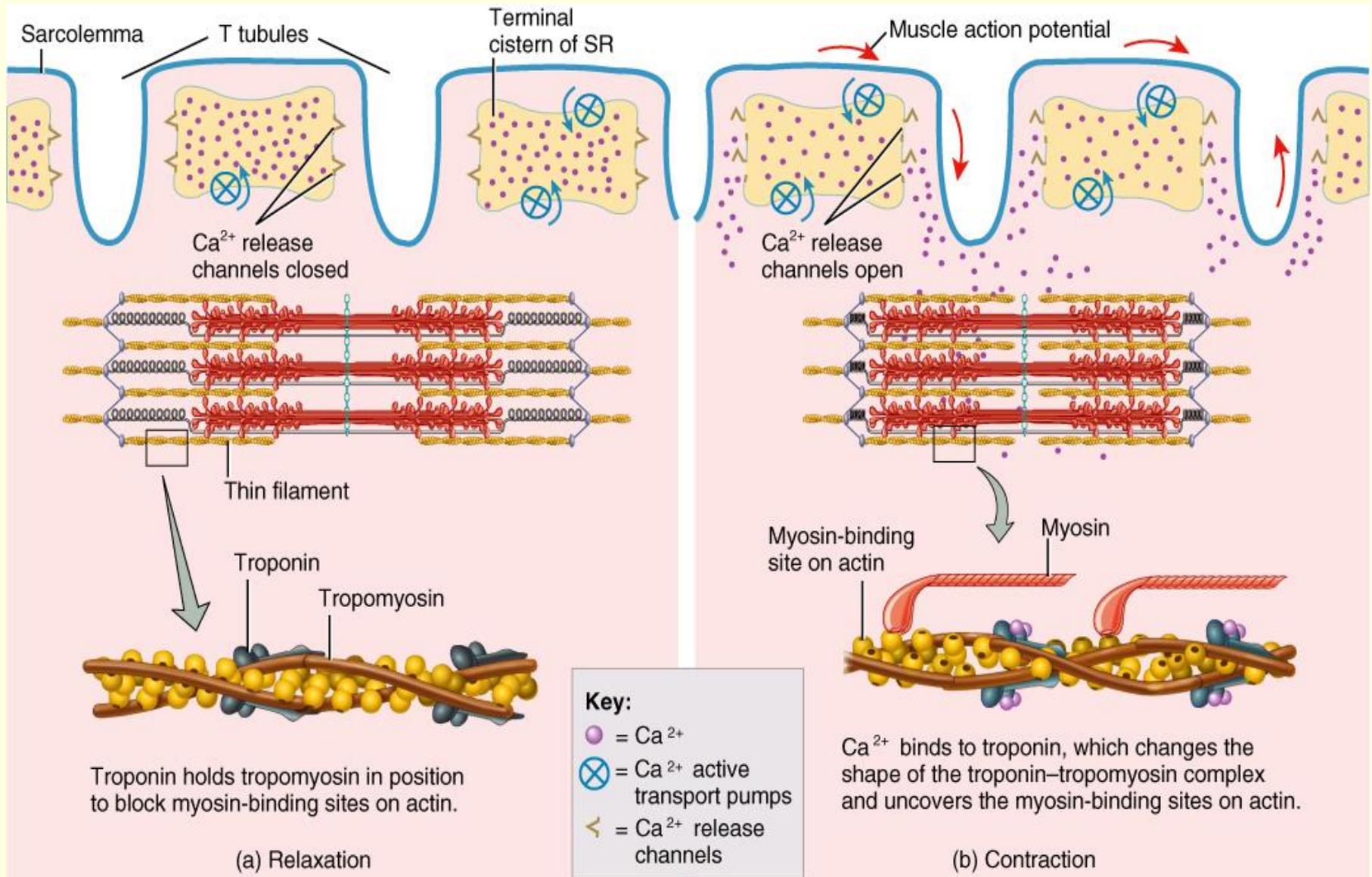
**(a) Relaxed muscle**



**(b) Partially contracted muscle**



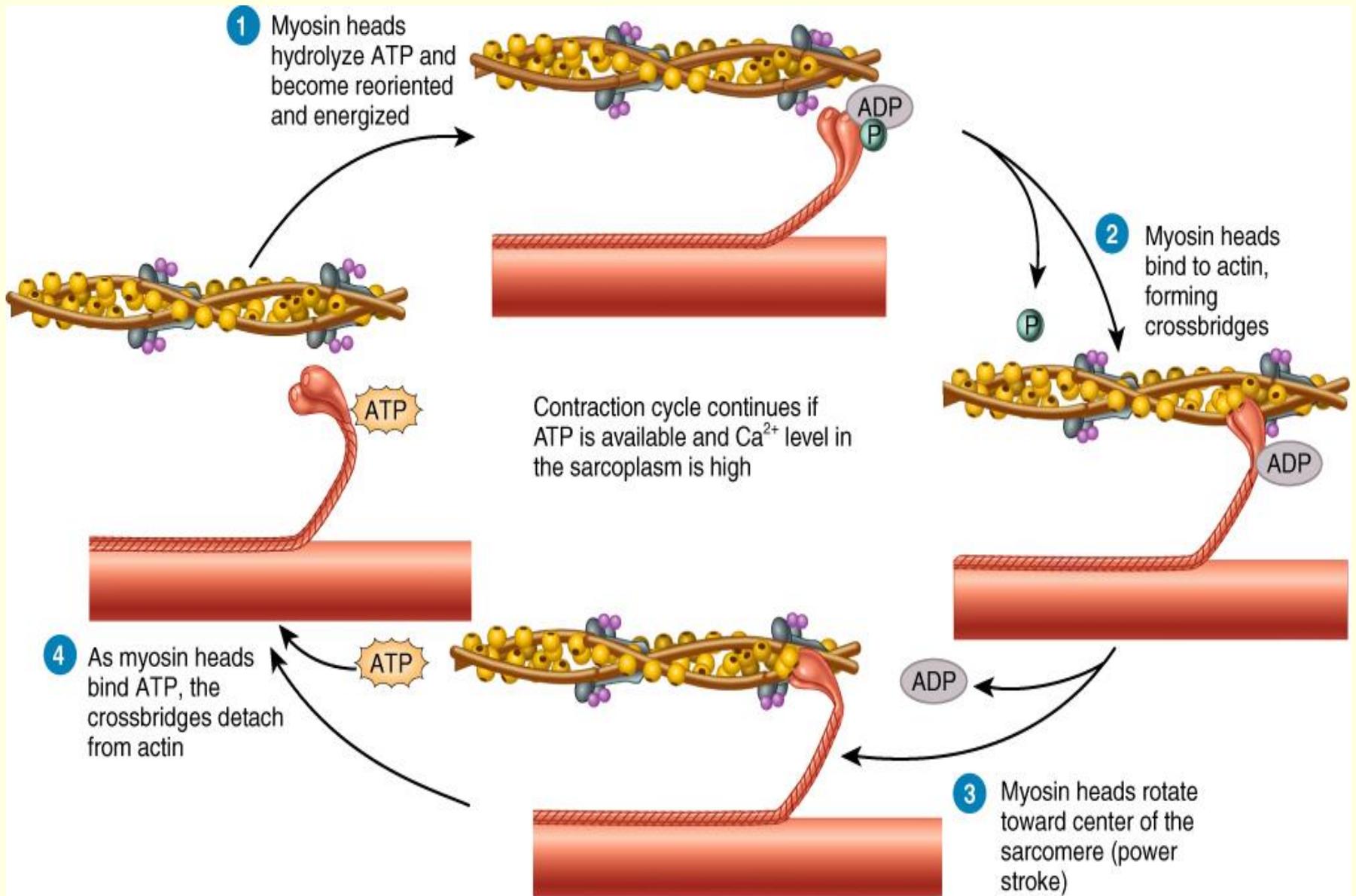
**(c) Maximally contracted muscle**

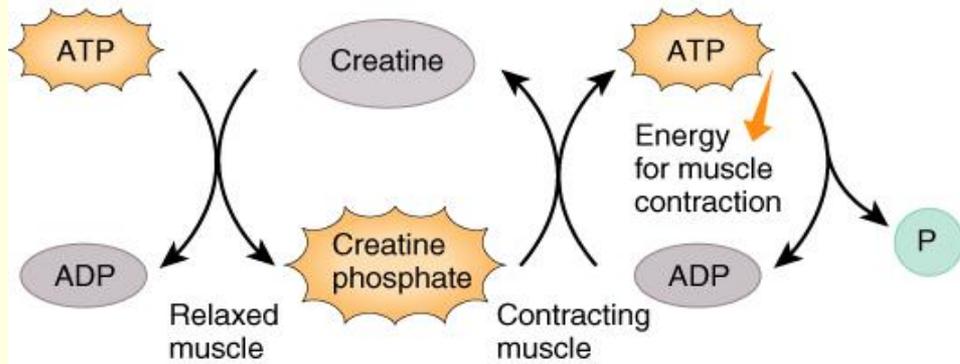


# Cardiac Muscle contraction Vs. Skeletal Muscle

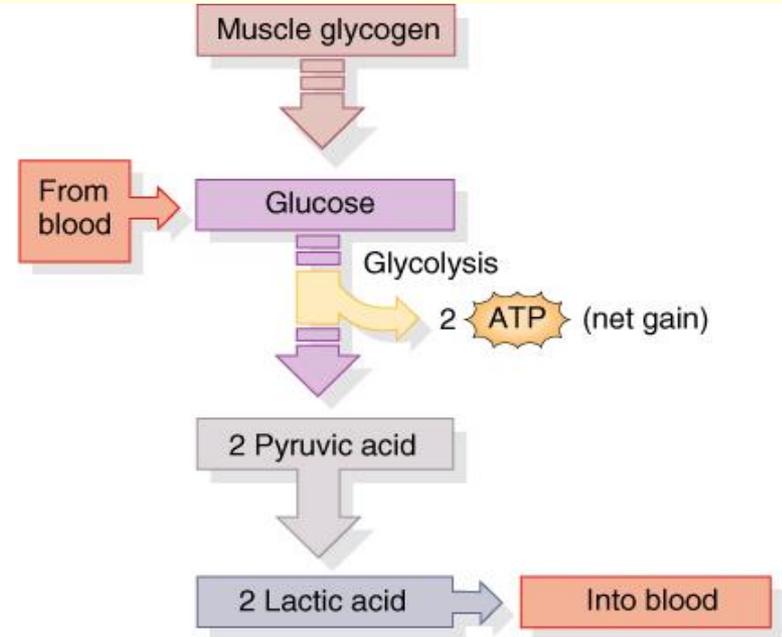
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- ⊕ Sliding filament hypothesis
- ⊕ No tetany (Long refractory period because of plateau)
- ⊕ Fatty acids main source of energy unlike skeletal muscle (Anaerobic and Aerobic)
- ⊕ Attachment and detachment cycle and ATP dependence is the same

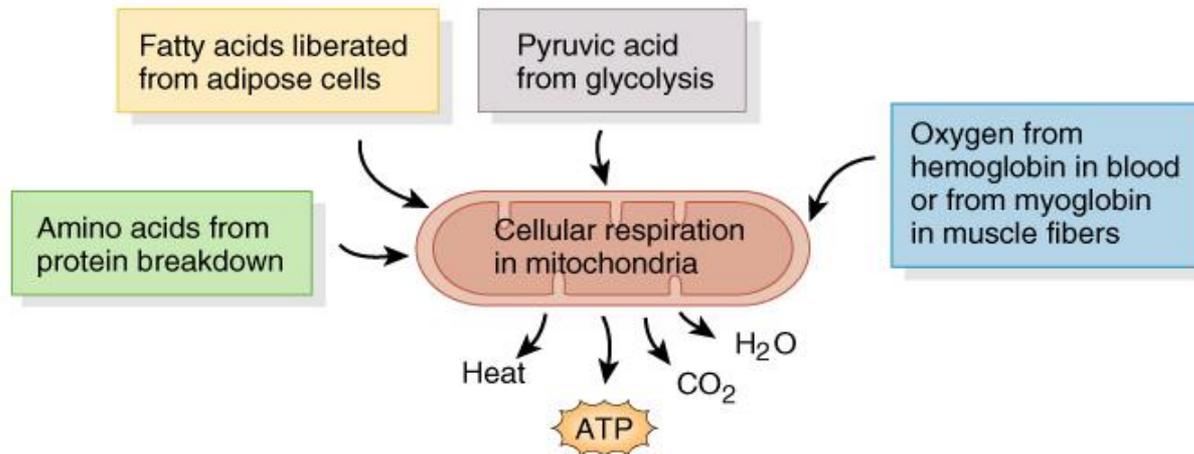




(a) ATP from creatine phosphate



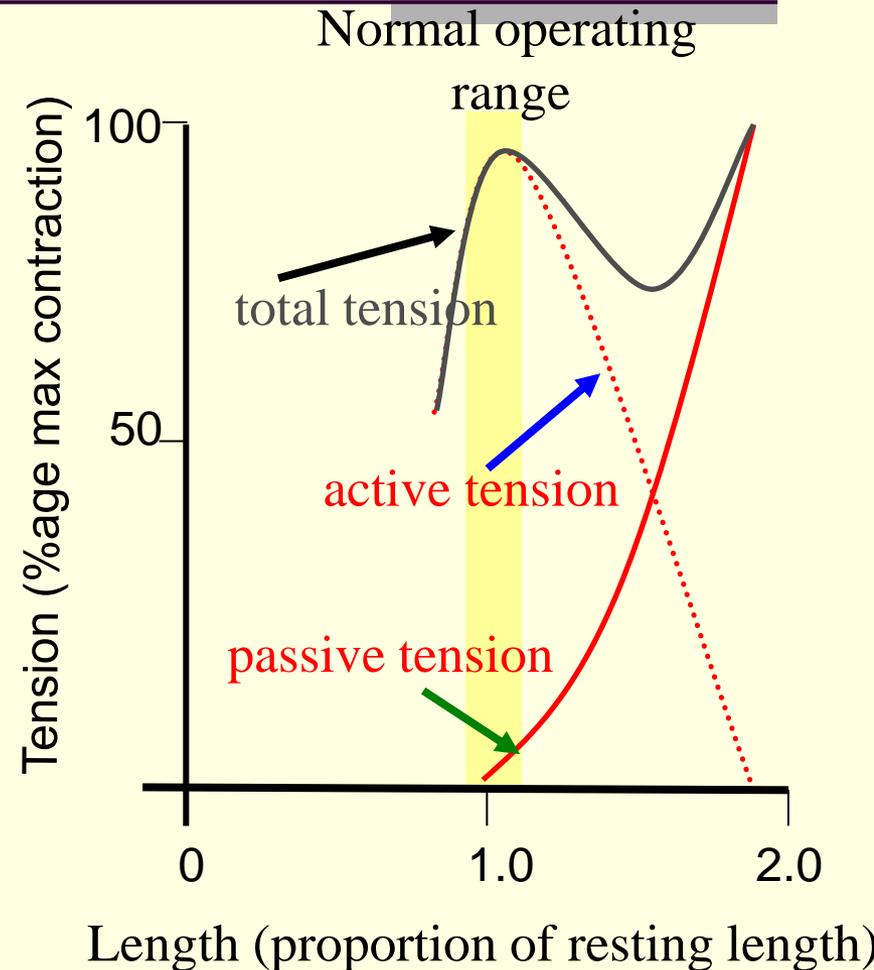
(b) ATP from anaerobic respiration

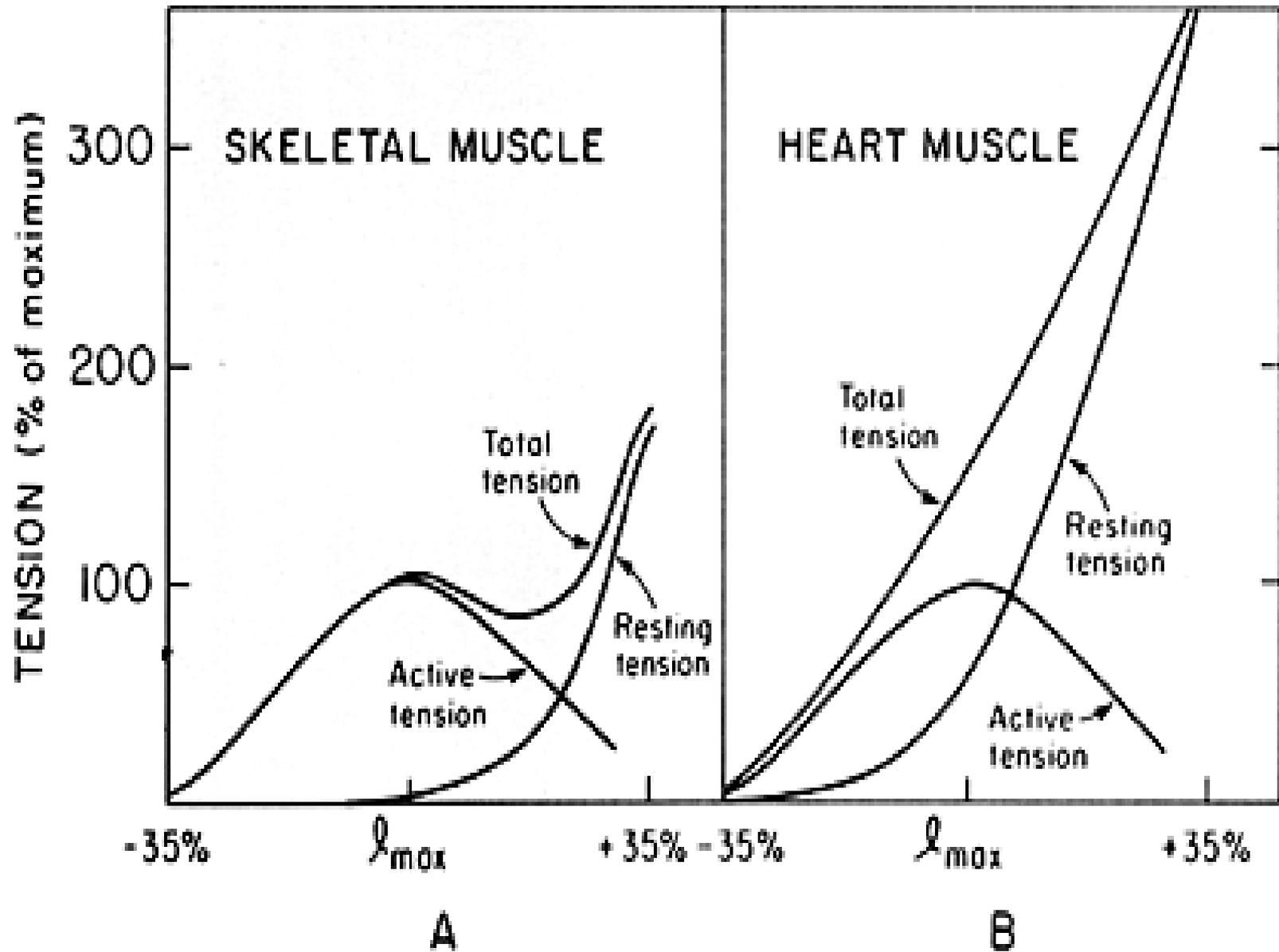


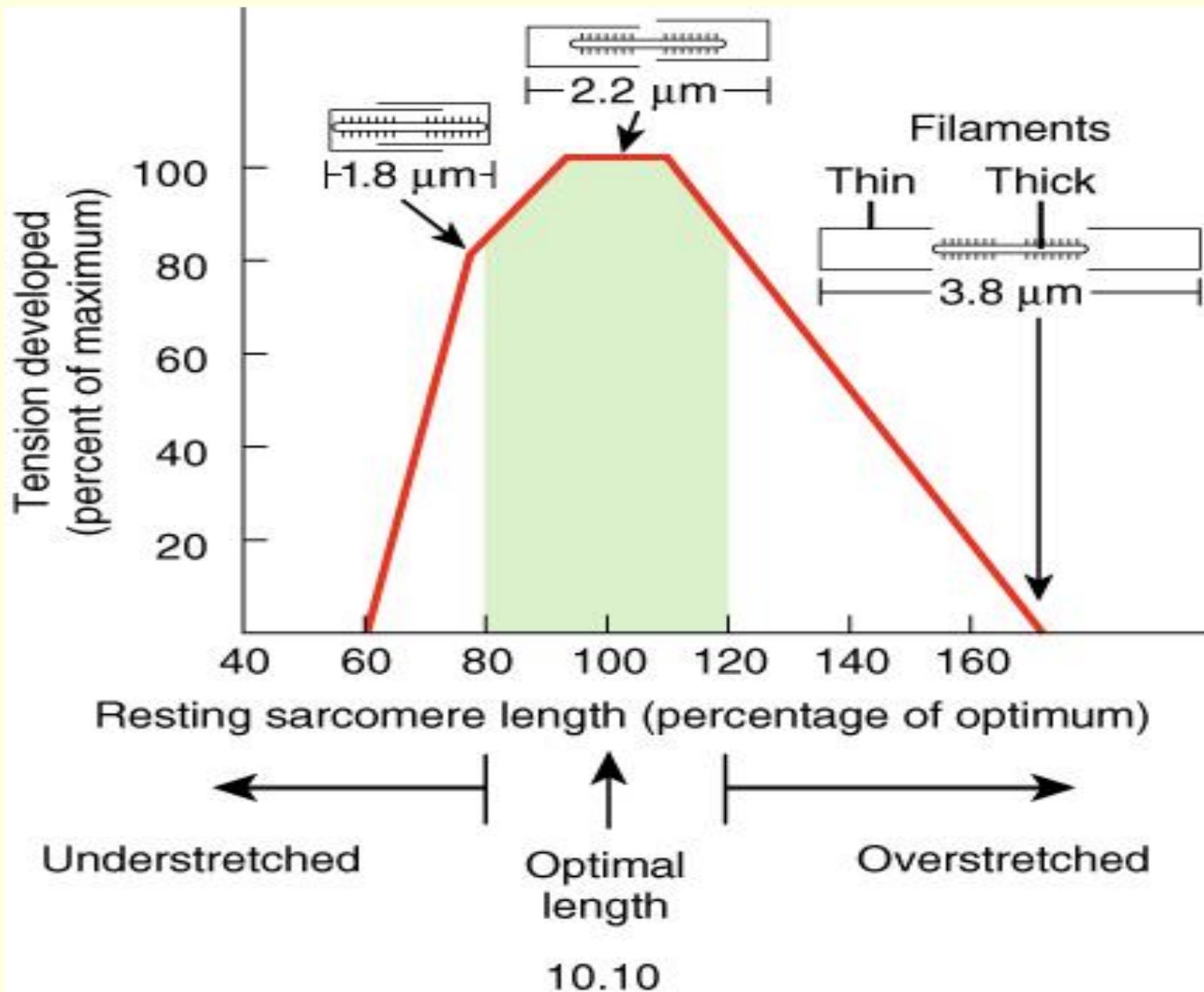
(c) ATP from aerobic cellular respiration

# Length-Tension Relation for Skeletal Muscle

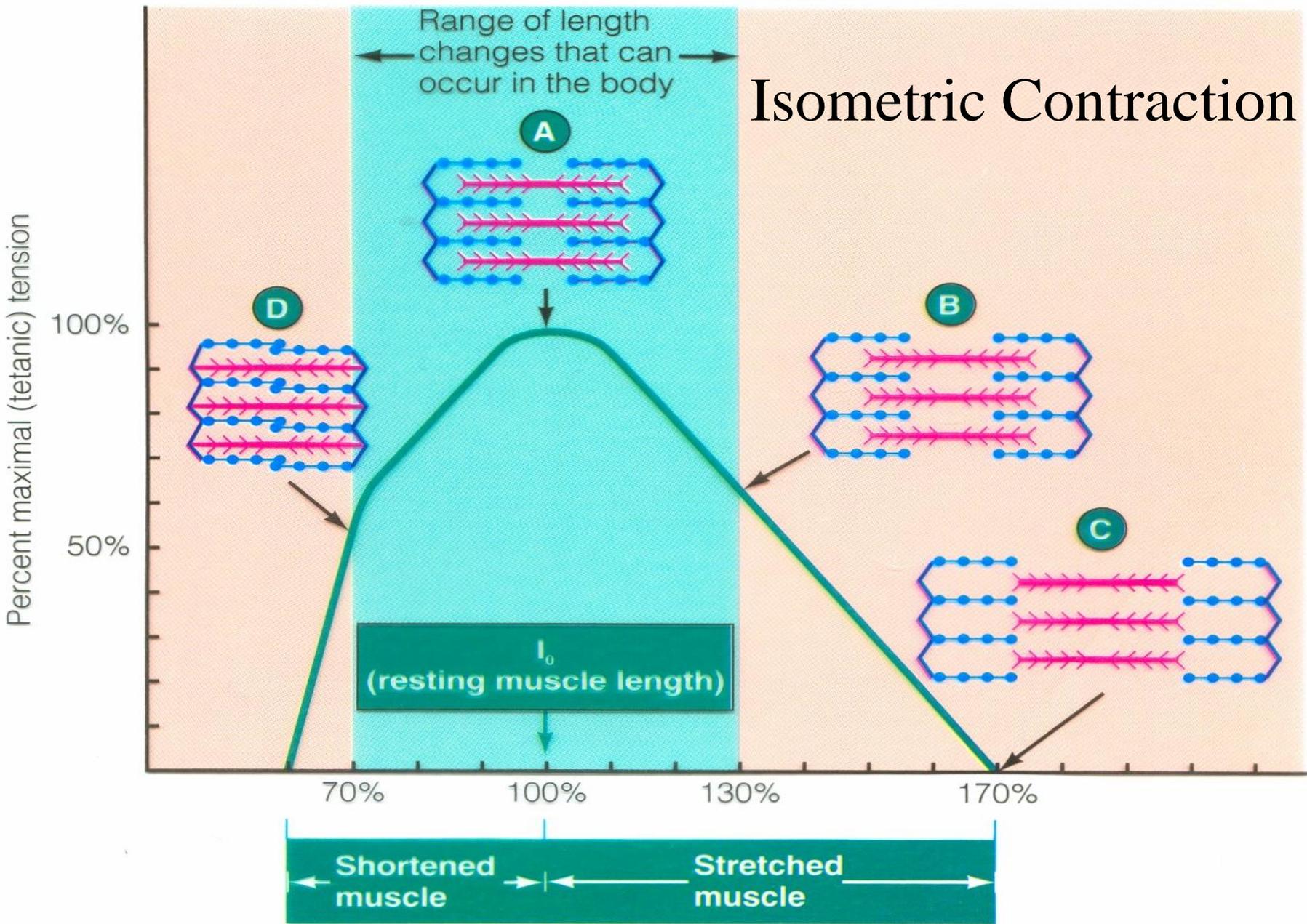
- ❖ Active tension cannot be measured directly
- ❖ What can be measured?
  - ❖ (1) passive tension - *tension required to extend a resting muscle*
  - ❖ (2) total tension - *active tension and passive combined*
- ❖ Active is calculated from 1 & 2
- ❖  $(AT = TT - PT)$
- ❖ Note that active tension falls away linearly with increasing length





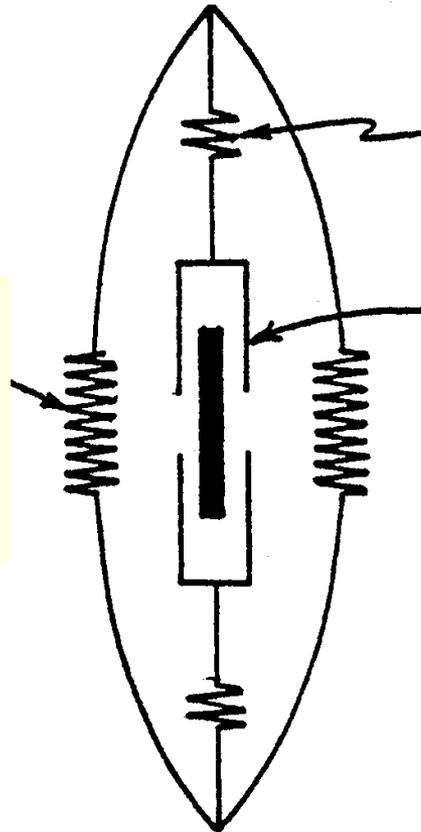


# Isometric Contraction



Muscle fiber length compared with resting length

**PARALLEL ELASTIC  
ELEMENTS  
(PASSIVE TENSION)**



**SERIES ELASTIC  
ELEMENTS**

**CONTRACTILE  
COMPONENT  
(ACTIVE TENSION)**

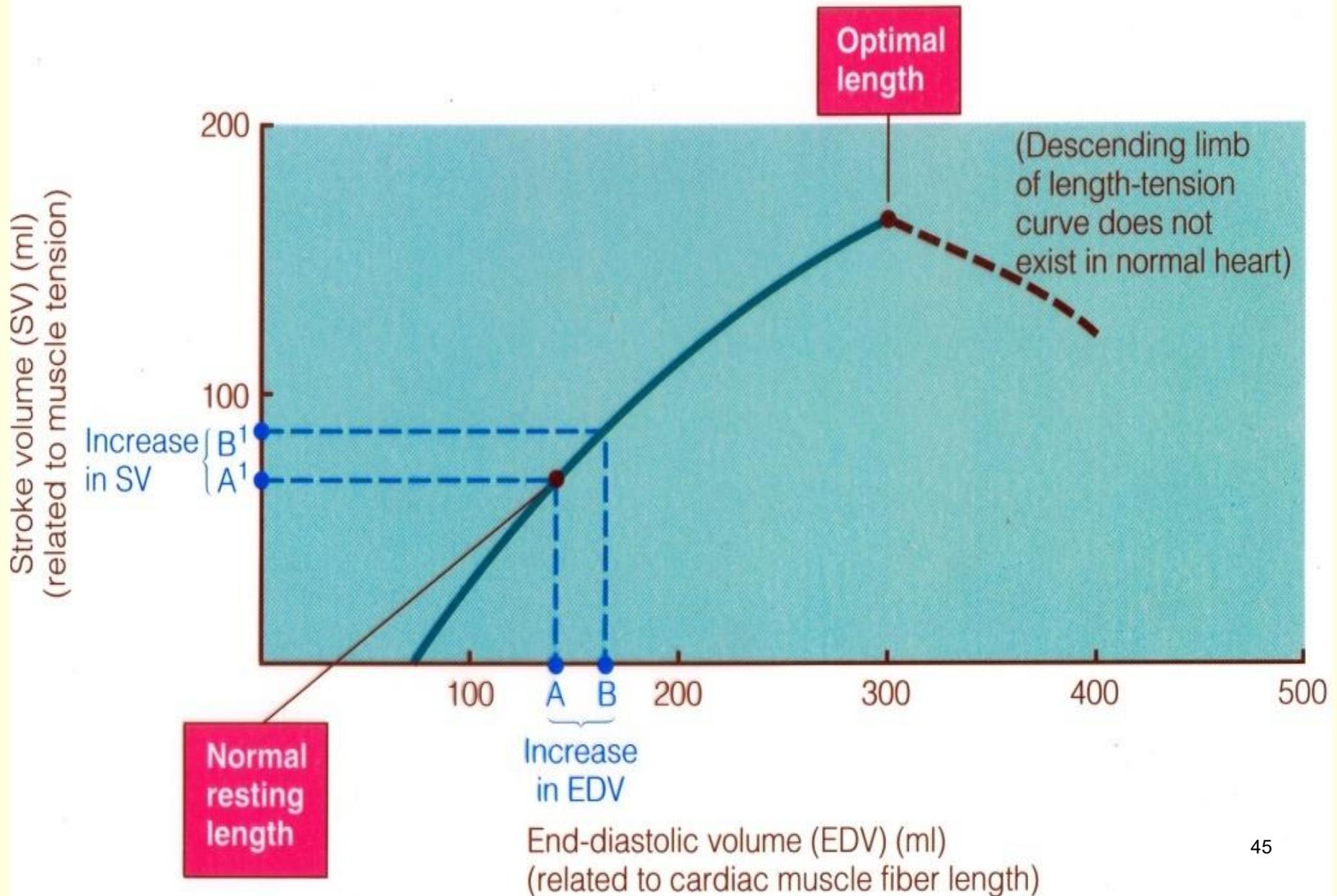
**TOTAL  
TENSION**

# Cardiac Muscle length-tension relationship

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- ☞ Cardiac muscle works at much less than its maximum length in contrast to skeletal
- ☞ Total, Active and Passive length-tension relationship differ
- ☞ Frank-Starling law of the heart

# Intrinsic Control of Stroke Volume (Frank-Starling Curve)



# Thank You

