The Cardio-VASCULAR System

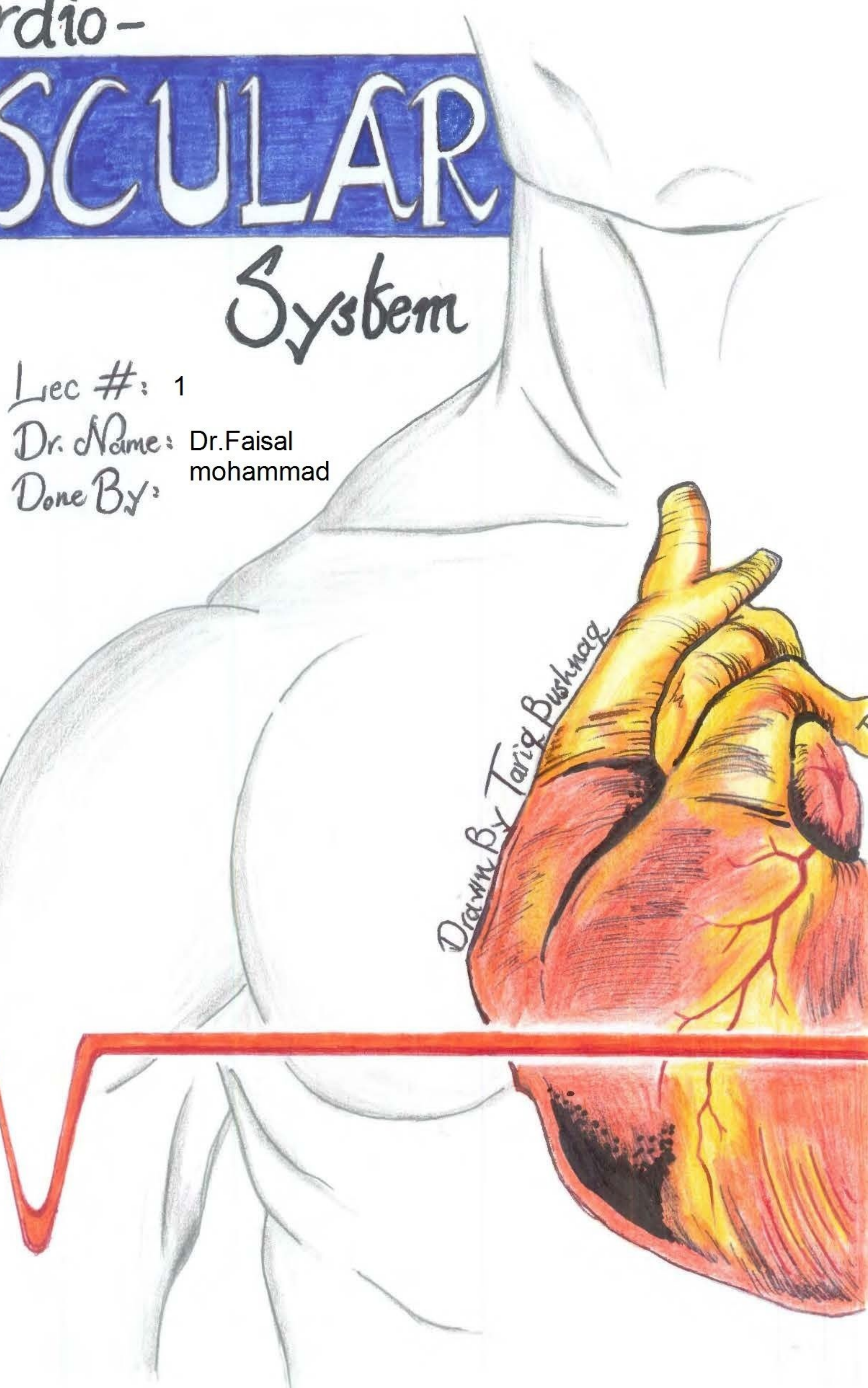
- ○ Anatomy
- ○ Histology
- ○ Pathology
- ○ Pharmacology
- ● Physiology
- ○ Microbiology

Lec #: 1

Dr. Name: Dr.Faisal mohammad

Done By:

- ○ Handout
- ○ Sheet
- ● Slide



Cadiovascular System-1

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University of Jordan

Cardiovascular Physiology for ***Medical Students***

FALL 2014-2015

Textbook: Textbook of Medical Physiology

By: Arthur C. Guyton & John E. Hall 12th Edition 2011

Lecture Topics	***Guyton11th***	**Guyton 12th**
1. Introduction	57-71,103-106	57-69, 101-104
2. Cardiac mm. Physiology	103-106	101-104
3. Conduction System of the heart	116-121	115-120
4. Electrocardiography	123-130	121-127
5. Electrocardiography	131-138	129-136
6. Electrocardiography	147-157	143-153
7. Electrocardiography		
8. Heart as a pump and cardiac cycle	106-115	104-113
9. Heart as a pump and cardiac cycle		
10.. Heart as a pump and cardiac cycle		
11. Cardiac output and venous return	232-245	229-241
12. Cardiac output and venous return		
13. Circulation / systemic	161-163	157-158, 168-175
14. Circulation / Haemodynamics	164-180	159-166, 167-168
15. Arterial System/Regulation of arterial blood pressure	204-215	201-211
16. Arterial System/ Regulation of ABP.	216-231	213-228
17. Blood flow / Tissues and is control 195-203	191-200	
18. Special circulations (coronary Muscle blood flow and exercise	246-253	243-253

Optional Readings:

1. Physiology , latest edition , by : Berne and Levy last edition
2. Physiological Basis of Medical Practice, twelfth edition , by : John B. West 1990.
3. Human physiology from cells to systems, latest edition, by: Lauralee Sherwood. Last edition

FAISAL I. MOHAMMED. MD, PhD

Clinical Problem

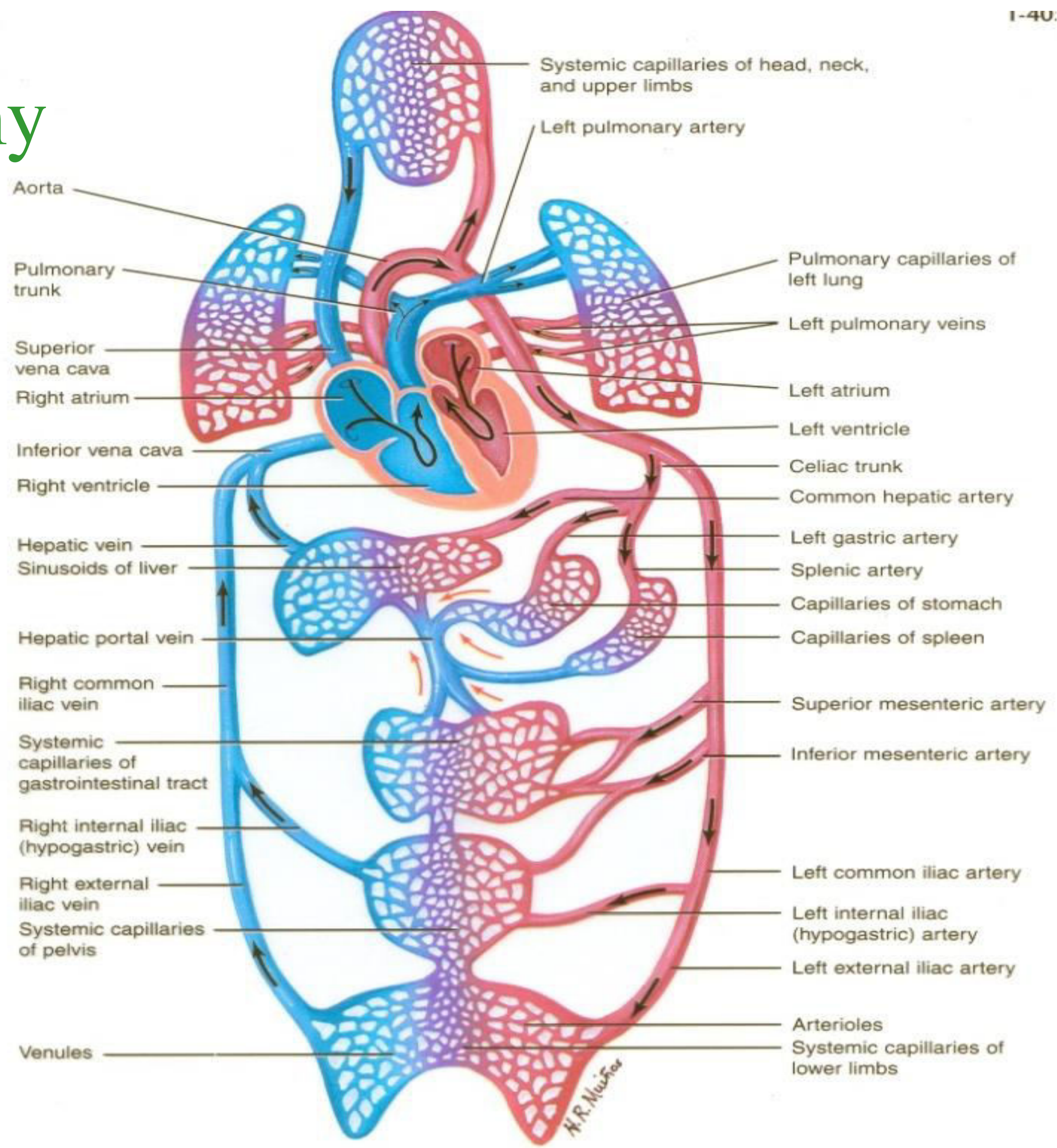
A 54 years old man seen in the cardiology clinic complaining of severe weakness, fatigue, dry cough, weight gain and difficulty in breathing. He feels severe shortness of breath while walking up stairs of his second floor apartment. He still complains of lesser severity of symptoms at rest. He states he often awakens at night feeling like he was suffocating. He is now sleeping with three pillows under his head. Lately he has taken to fall asleep while he is sitting watching T.V. He also complains of having to urinate 3-4 times per night. He was hospitalized with heart problem two months ago and was told that the efficiency of his heart is less than 30% and he needs ?? and has to wait until??. On examination his weight is 95Kg, height is 165 cm, blood pressure was 140/85 mmHg, his heart rate 90 beats/min and regular, his resp. rate is 28/min and labored.

Auscultation of the heart reveals abnormal heart sounds

Objectives:

- Introduction to the CVS physiology
- Review the anatomy of the CVS.
- List the functions of the CVS
- Comprehend the pump nature of the heart

Cardiovascular System Anatomy



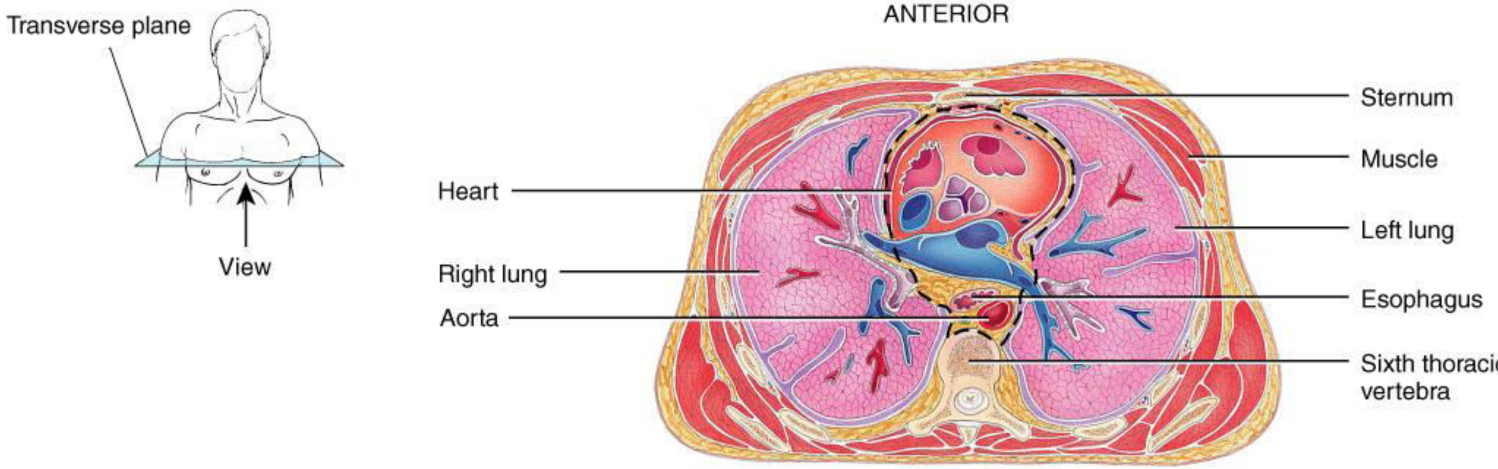
General plan of circulation

History of cardiac Transplant

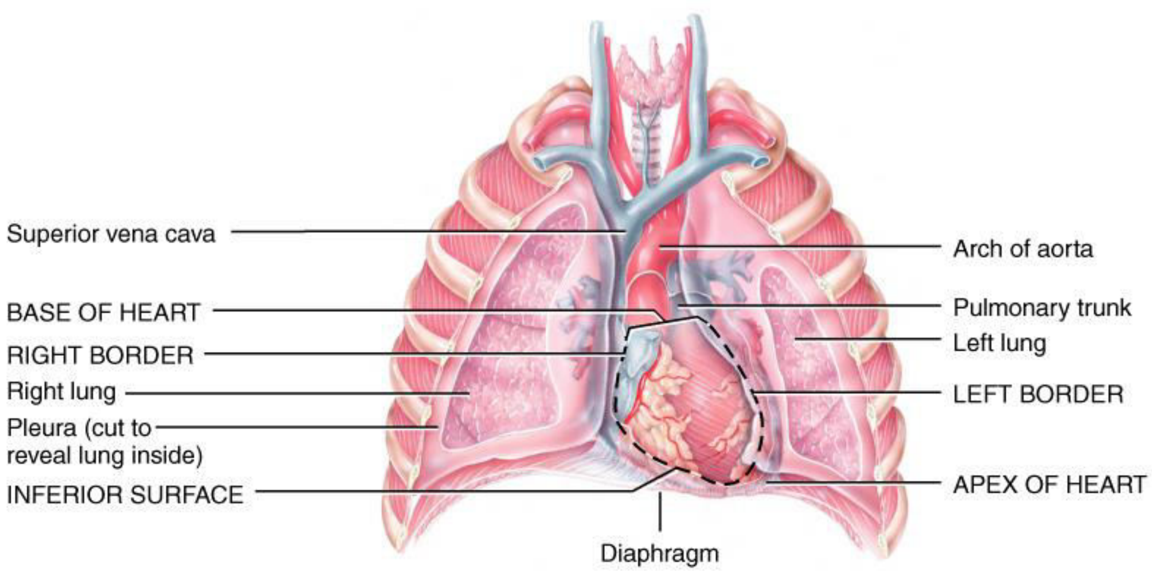
- **In 1967,** Christiaan Barnard in Cape Town, South Africa transplanted the first Human Heart removed from a 25-year-old woman who had died following an auto accident and placed it in the chest of Louis Washkansky, a 55-year-old man dying of heart damage. The patient survived for 18 days. The problem was Rejection- Cyclosporine – immunosuppressant -decreased that.
- **In 1984, the world's first successful pediatric heart transplant** was performed at Columbia on a four-year-old boy. He received a second transplant in 1989 and continues to live a productive life today.

History of cardiac Transplant…cont

- **In 1984**, in Linda Loma, California, Leonard Bailey, implanted a baboon heart into a 12-day-old girl, she survived for twenty days.
- **In 1982** in University of Utah, the first Total Artificial Heart was implanted in the chest a dentist Barney Clark by William DeVries. Clark survived for 112 days-The problem was blood clotting.



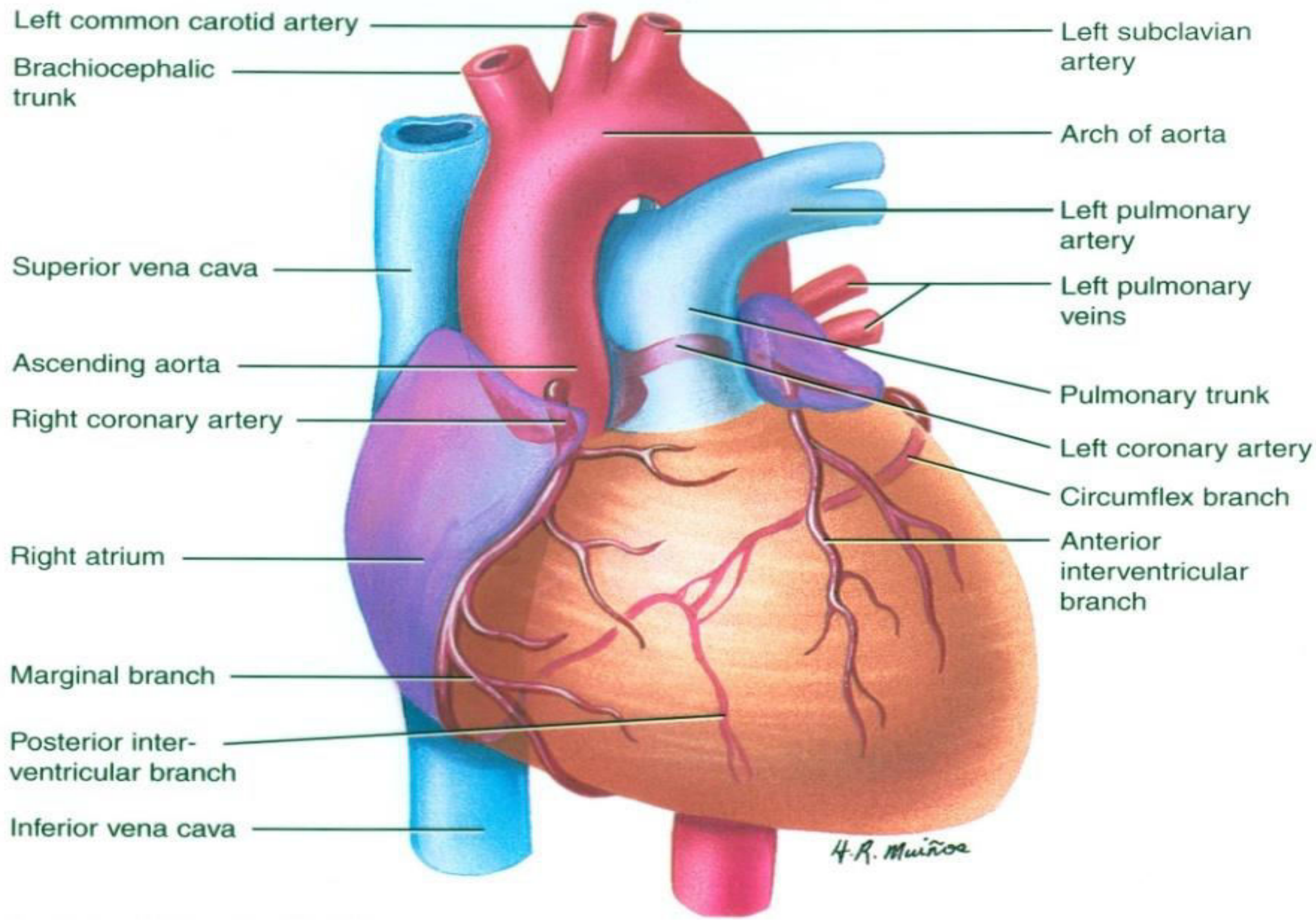
(a) Inferior view of transverse section of thoracic cavity showing the heart in the mediastinum



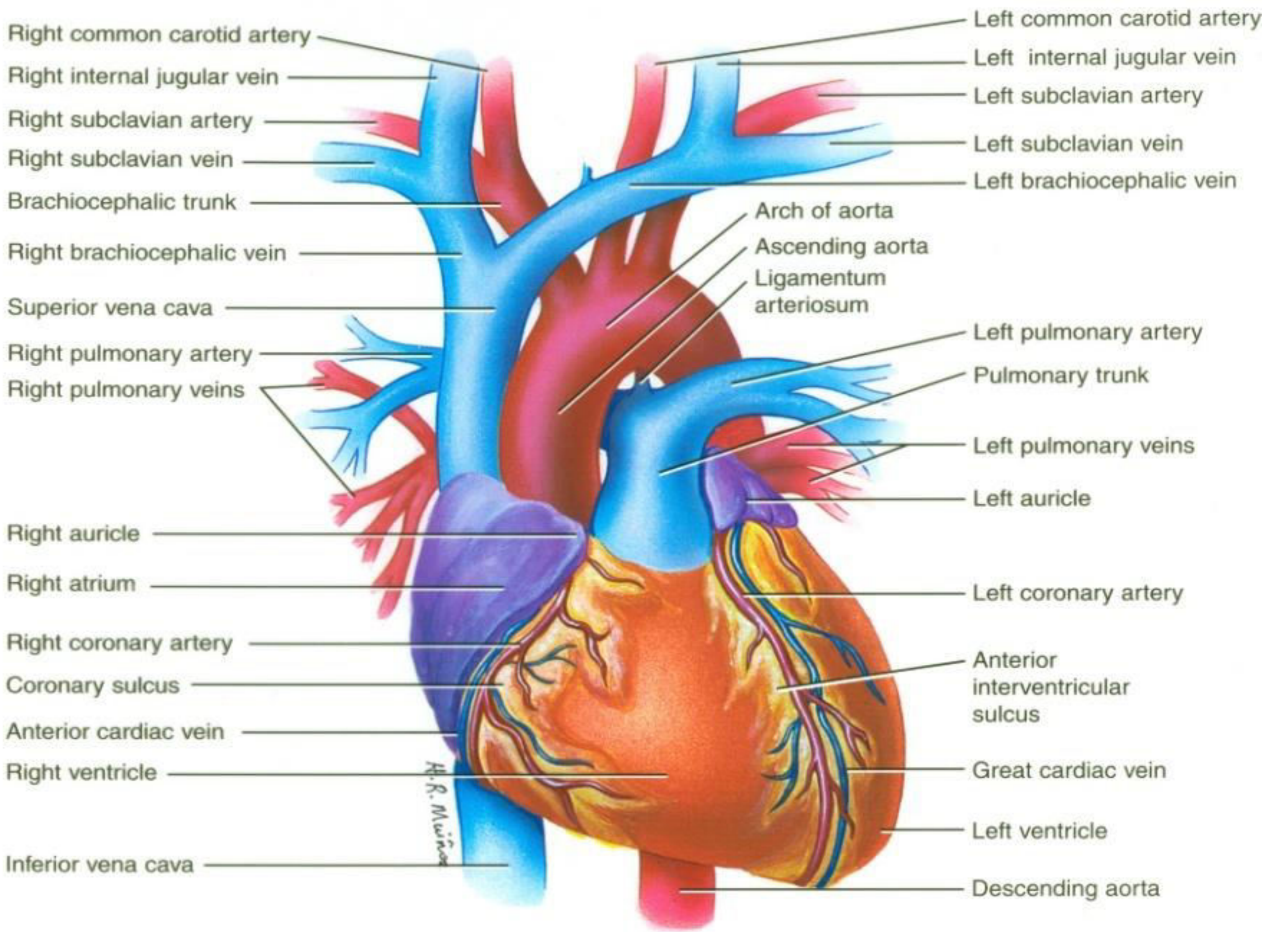
(b) Anterior view of the heart in the mediastinum

20.01

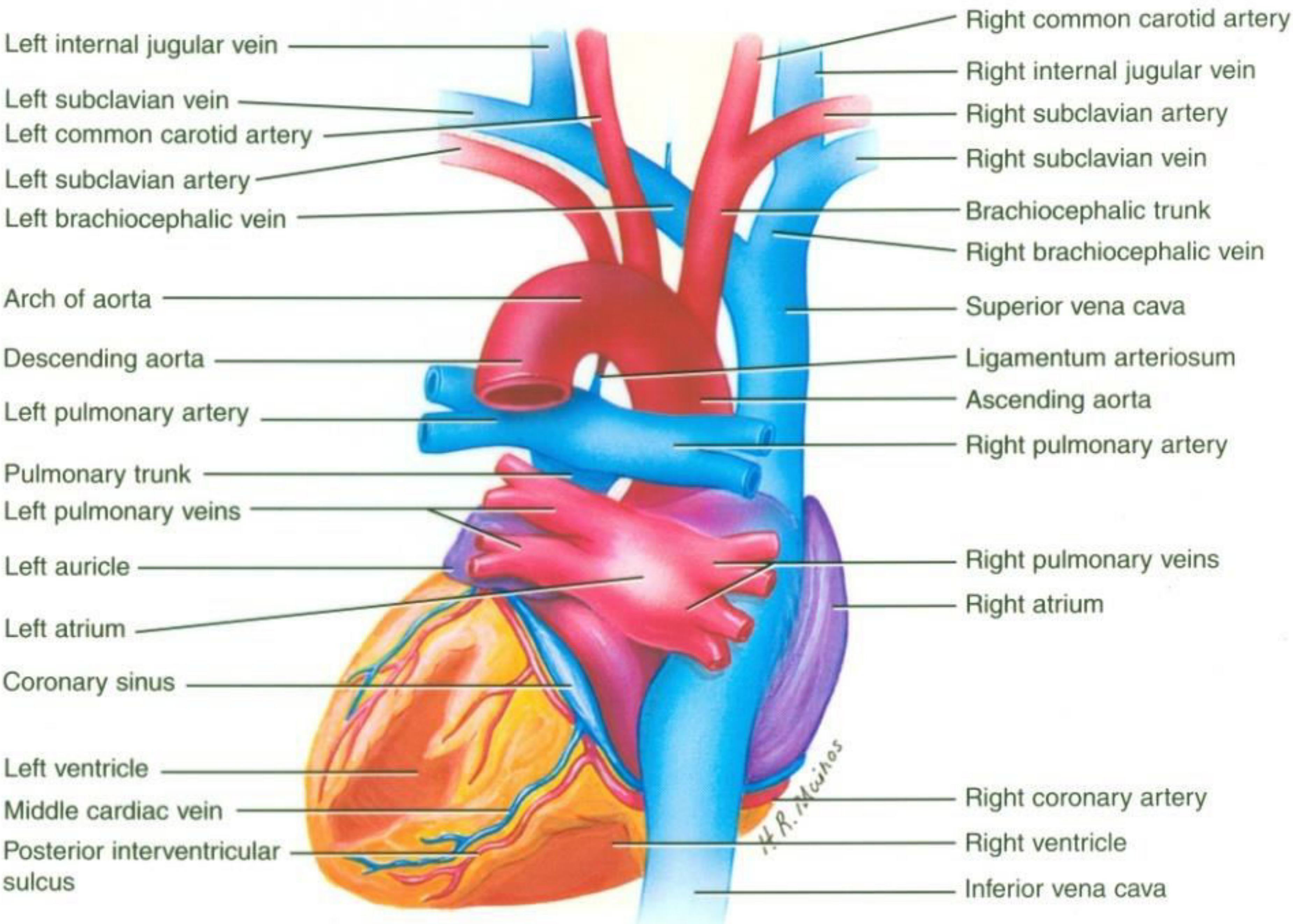
Anatomy of the heart



Anatomy of the heart

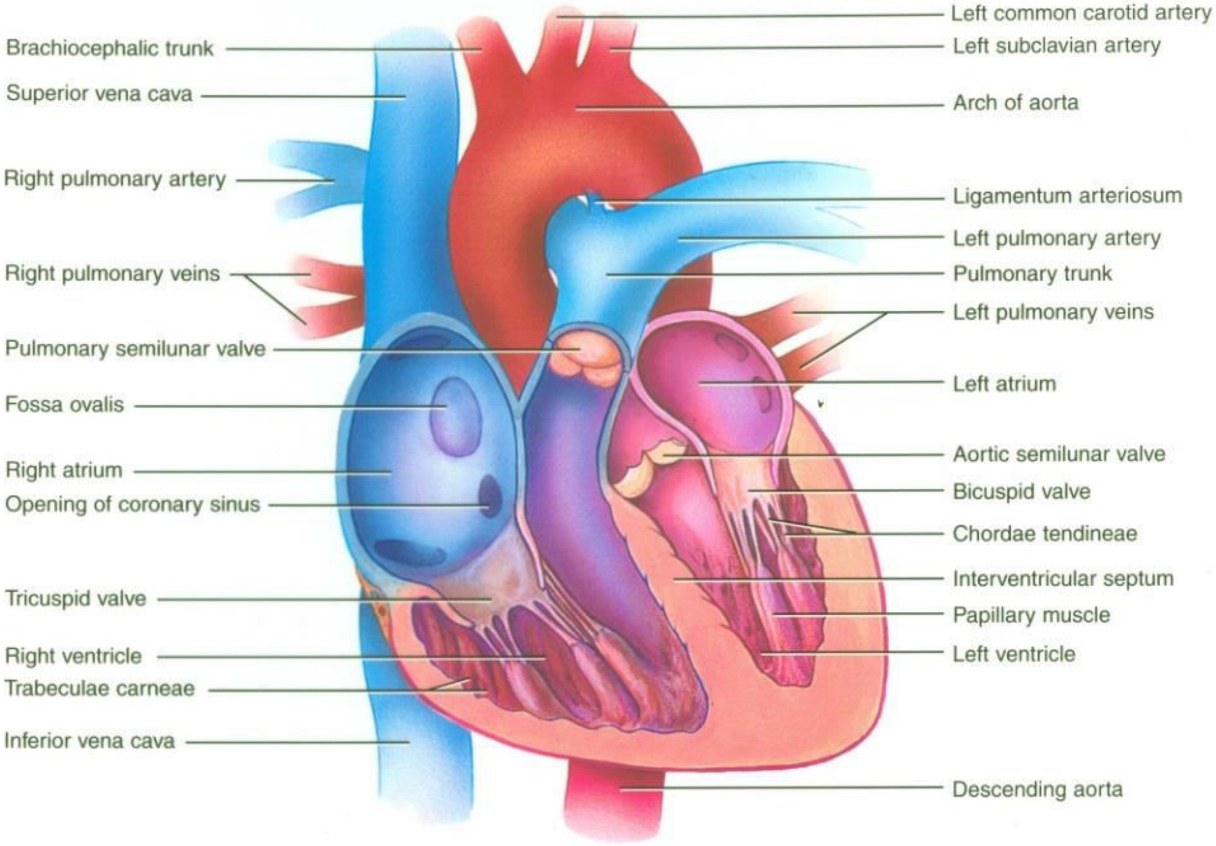


Anterior External View of Structure of Heart, Fig# 20.4a



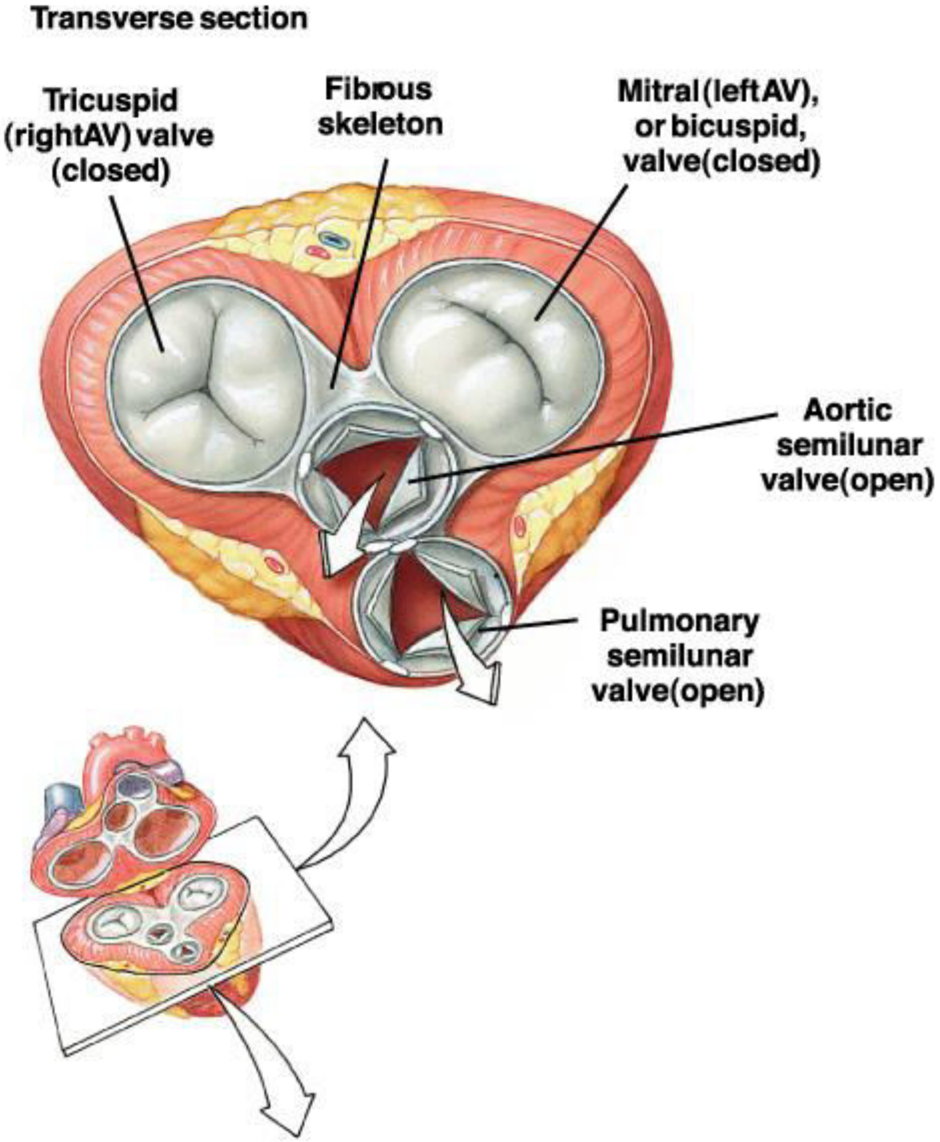
Posterior External View of Structure of Heart, Fig# 20.4c

Cardiac valves

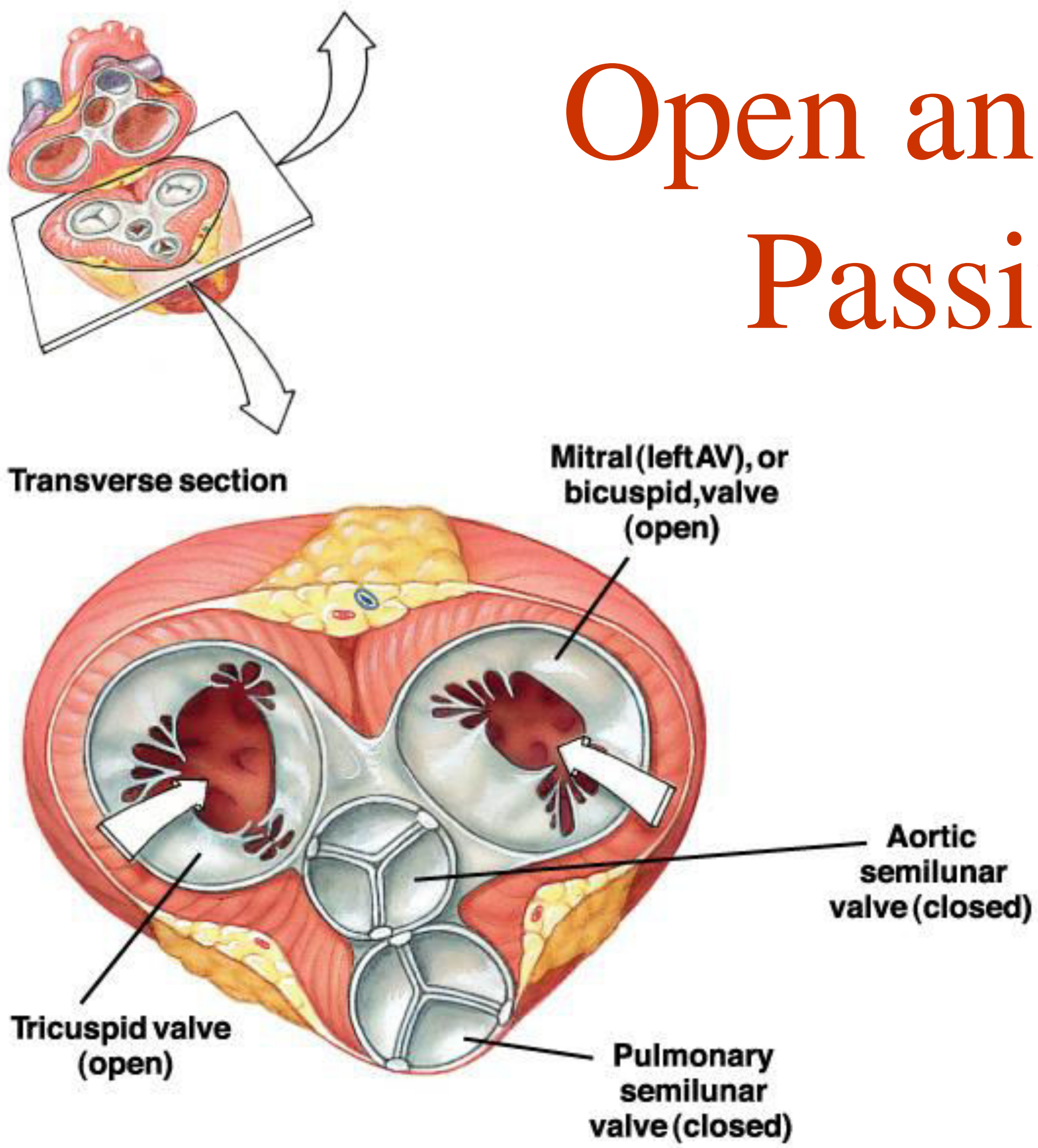


Anterior View of Frontal Section of Structure of Heart, Fig# 20.4d

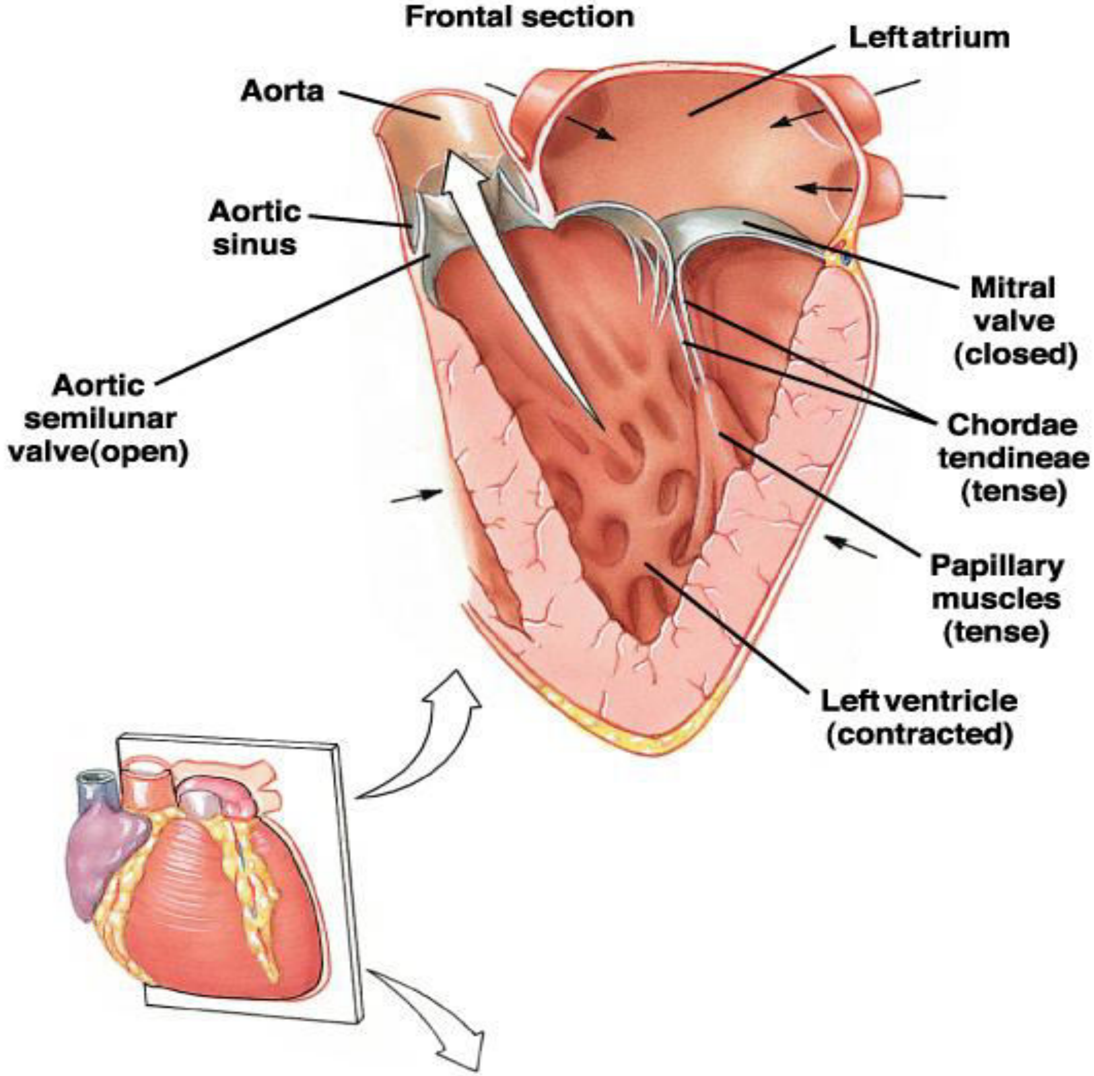
Cardiac valves



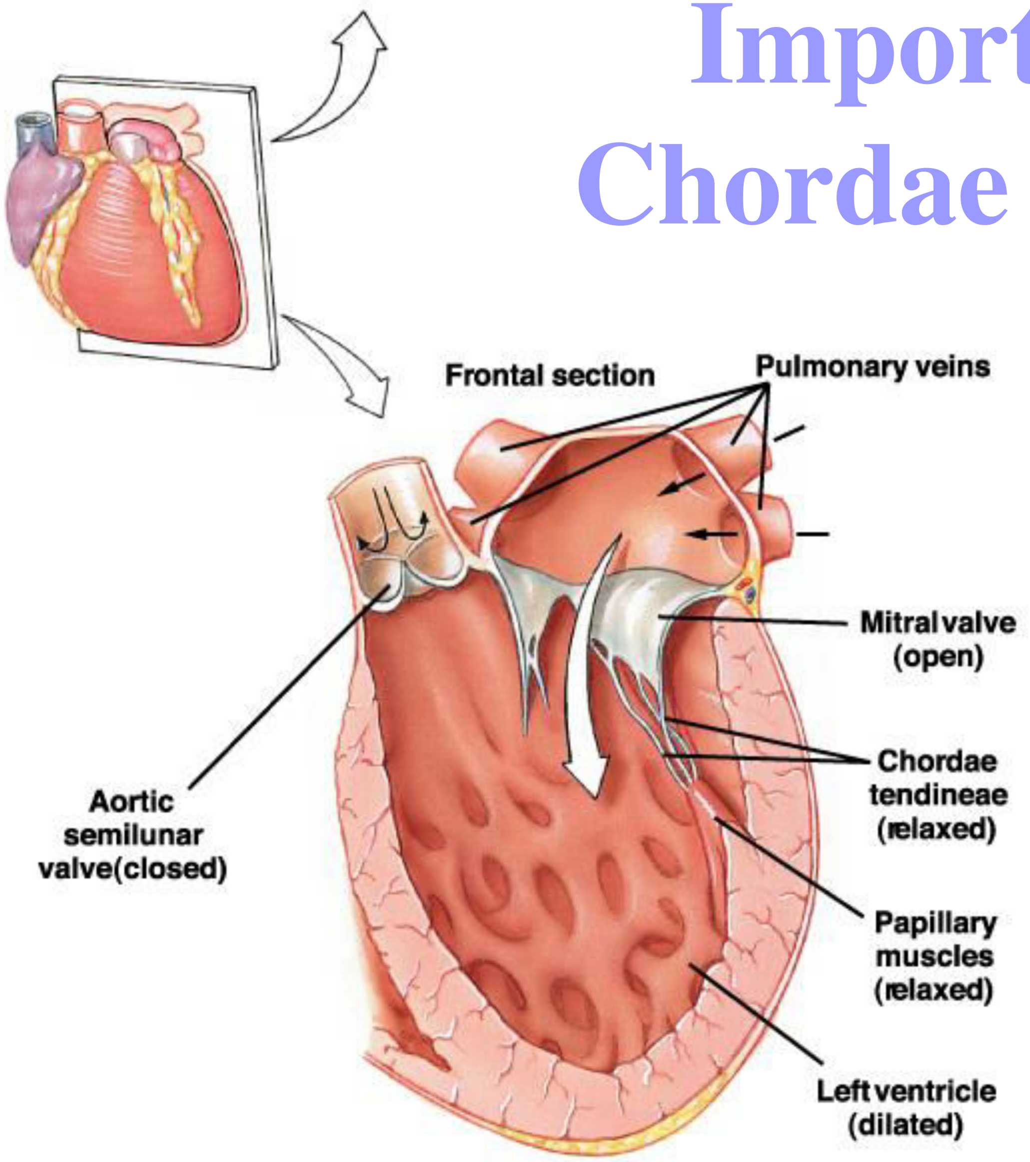
Cardiac Valves Open and Close Passively



Importance of Chordae Tendineae

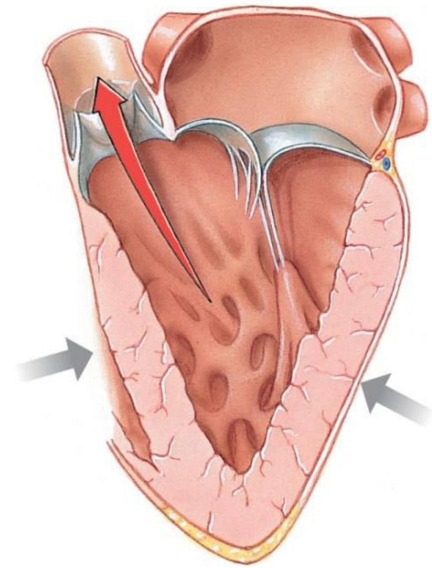


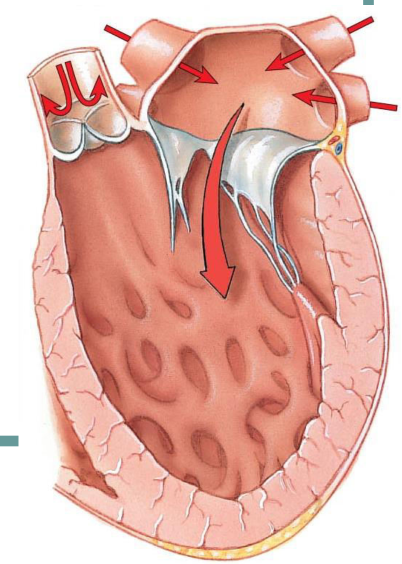
Importance of Chordae Tendineae



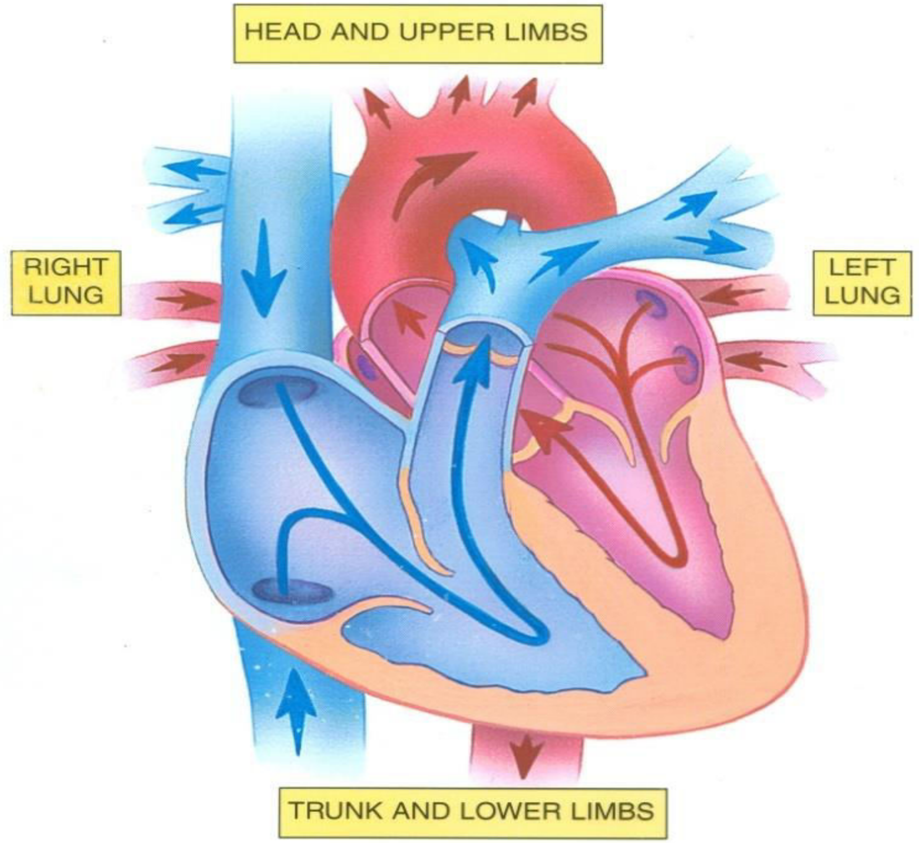
Functional Anatomy of the Heart Valves

- Function is to prevent backflow
  - Atrioventricular Valves
    - Prevent backflow to the atria
    - Prolapse is prevented by the chorda tendinae
      - Tensioned by the papillary muscles
  - Semilunar Valves
    - Prevent backflow into ventricles





Movement of blood in the heart



Blood Flow: Path of Blood Through Heart, Fig# 20.6a

Thank You

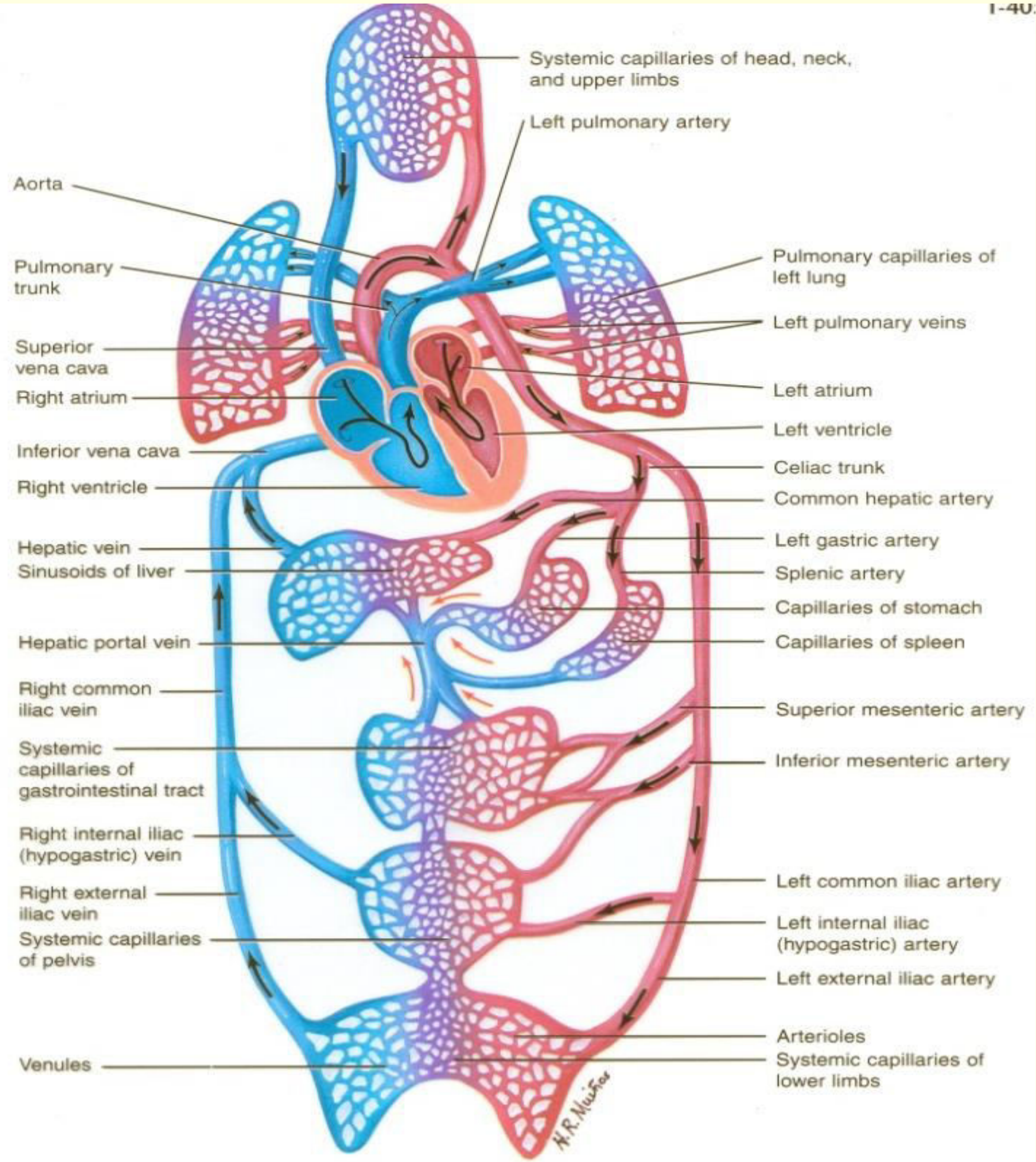
Cardiac Muscle Physiology

Faisal Mohammed, MD, PhD

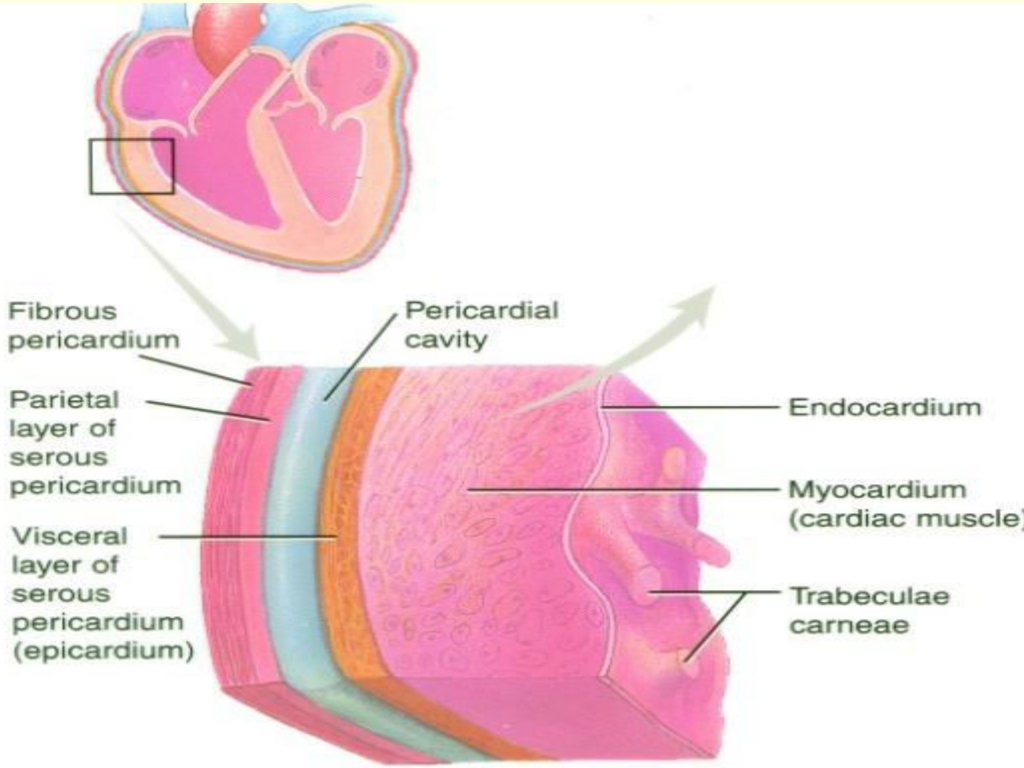
Objectives:

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

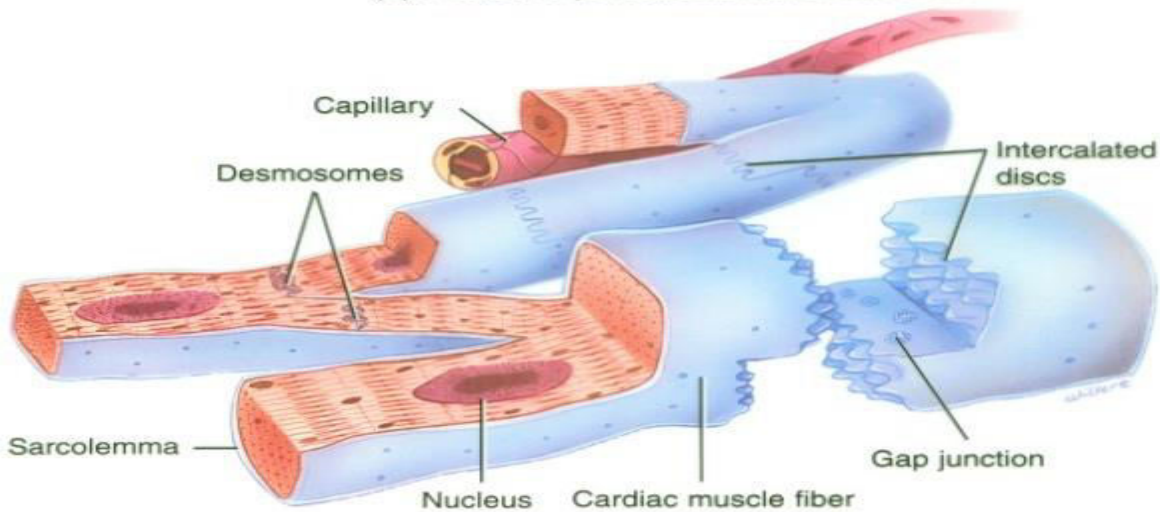
- Distinguish the cardiac muscle cell microstructure
- Describe cardiac muscle action potential
- Point out the functional importance of the action potential
- Follow the cardiac muscle mechanism of contraction
- Delineate cardiac muscle energy sources
- Outline the intracellular calcium homeostasis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



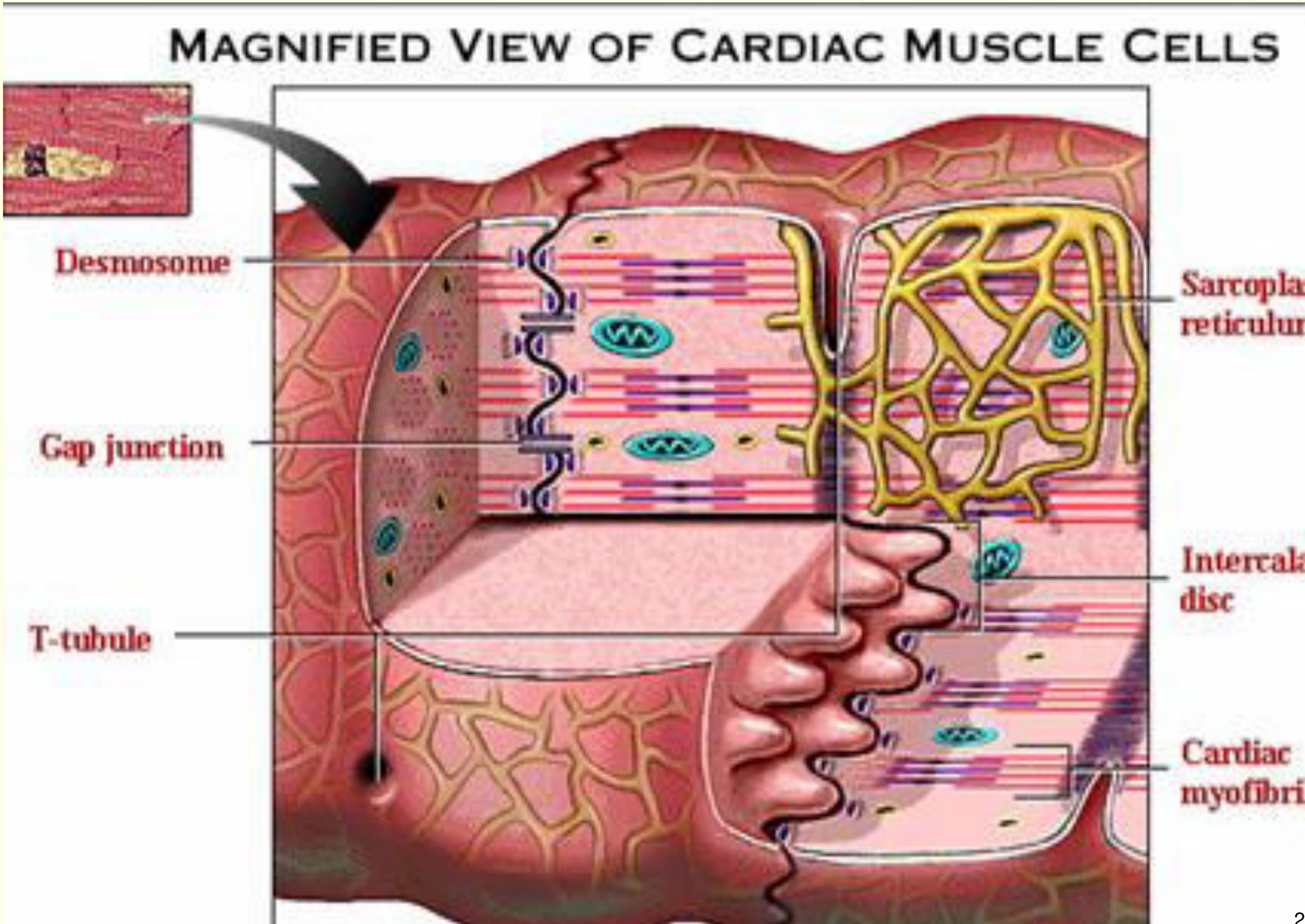
General plan of circulation

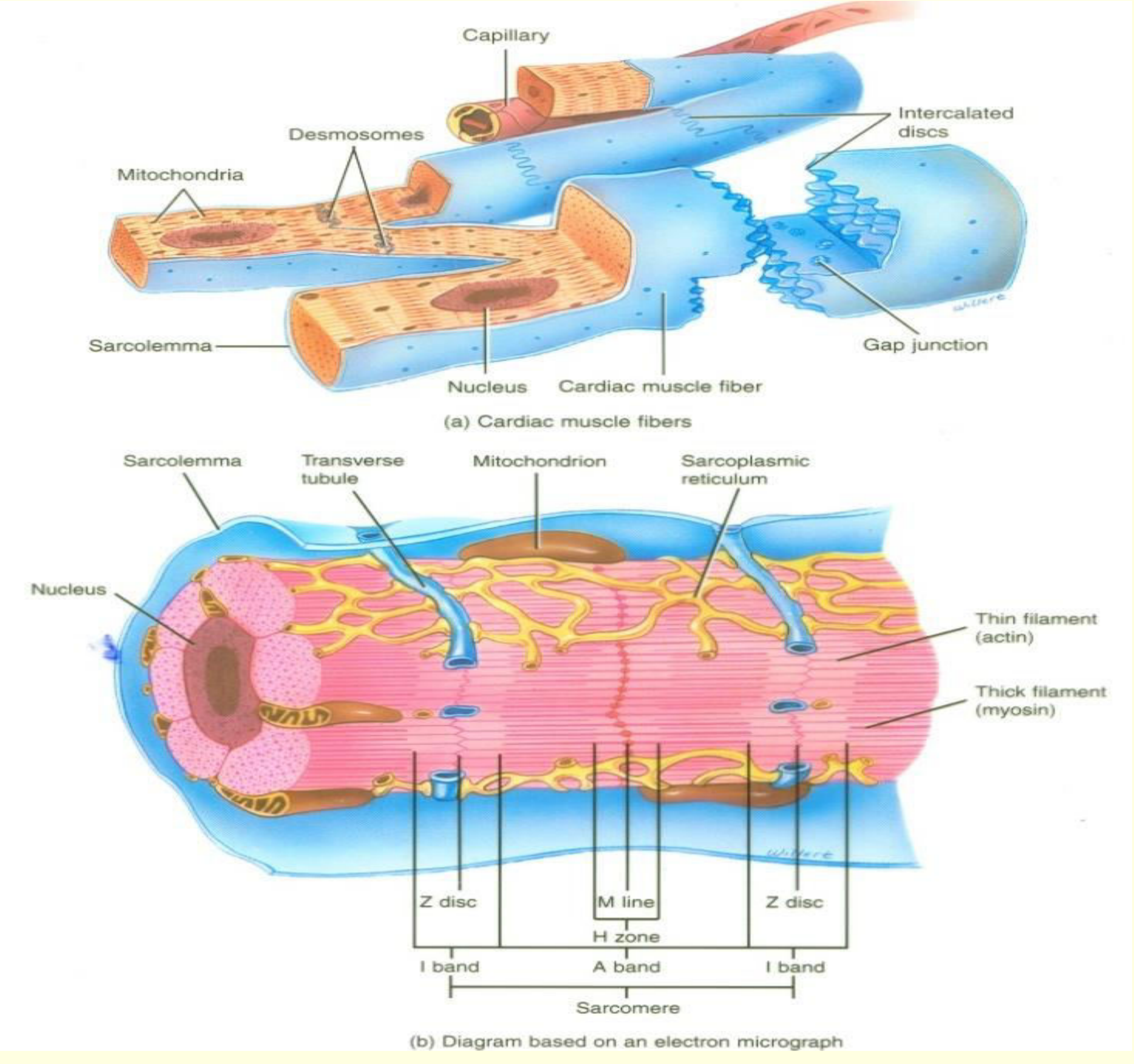


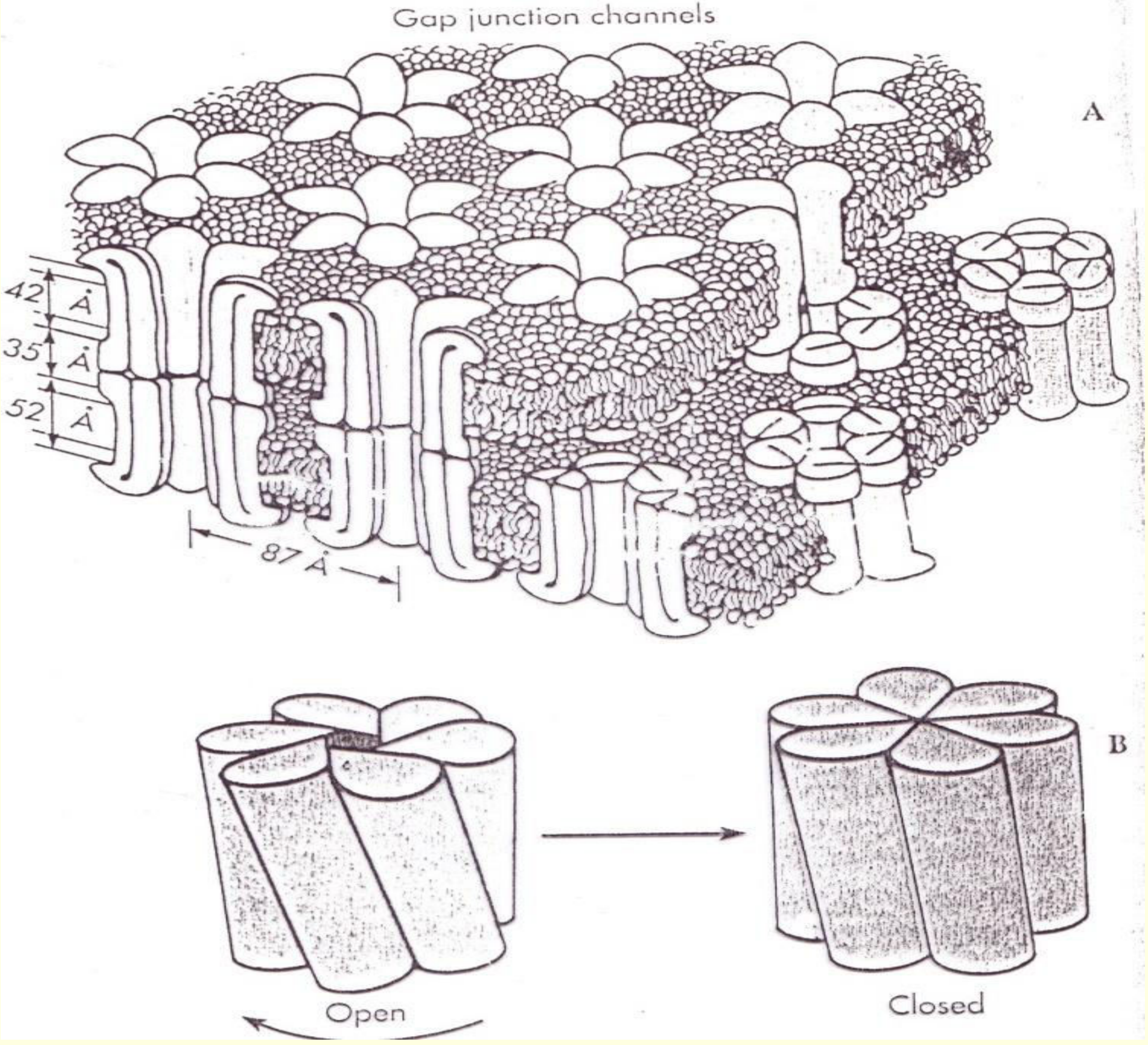
(a) Portion of pericardium and heart wall



(b) Cardiac muscle fibers


MAGNIFIED VIEW OF CARDIAC MUSCLE CELLS
Desmosome
Gap junction
T-tubule
Sarcoplas
reticulum
Intercala
disc
Cardiac
myofibril

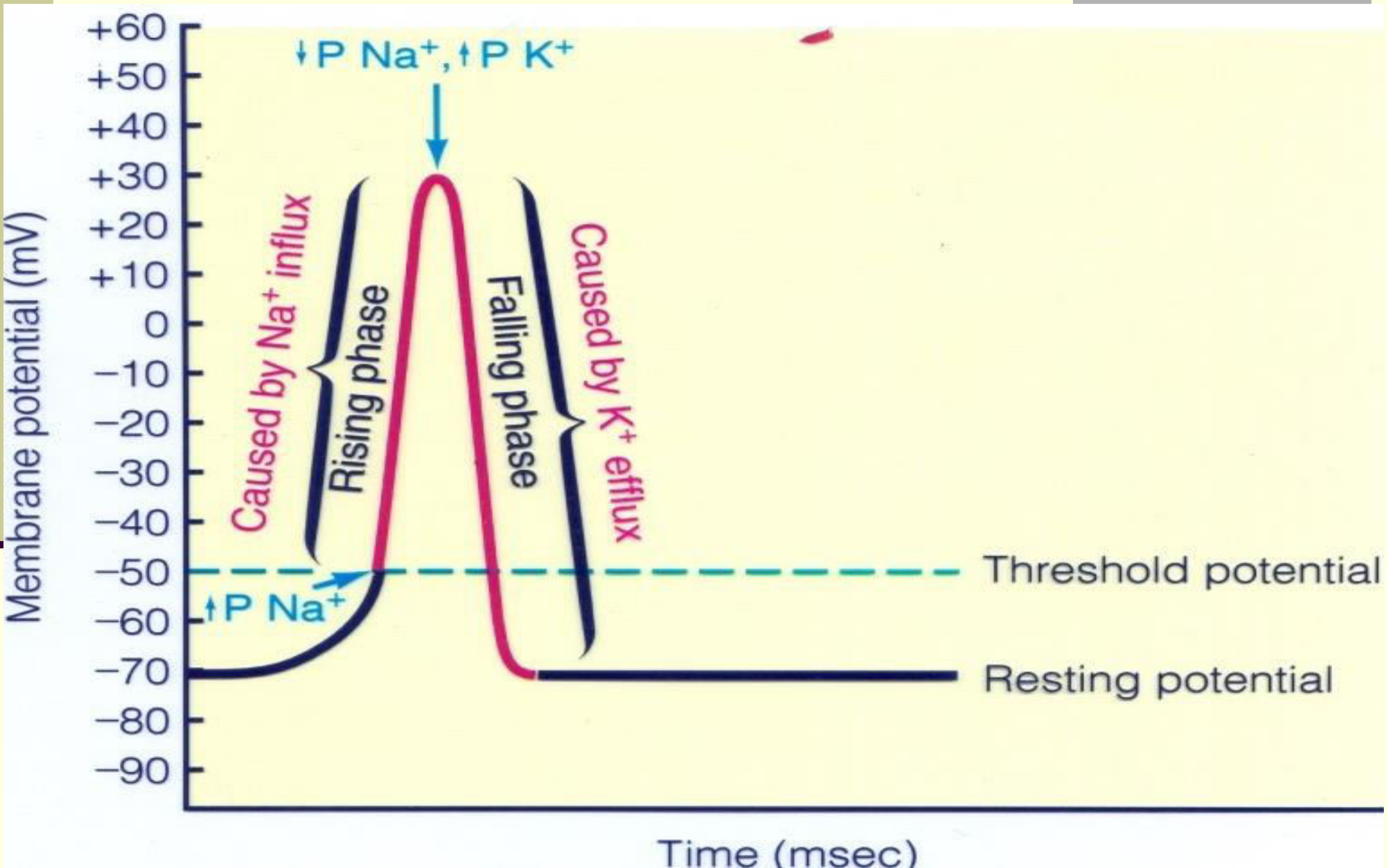

Capillary
Intercalated discs
Desmosomes
Mitochondria
Sarcolemma
Nucleus
Cardiac muscle fiber
Gap junction
(a) Cardiac muscle fibers
Sarcolemma
Transverse tubule
Mitochondrion
Sarcoplasmic reticulum
Nucleus
Thin filament (actin)
Thick filament (myosin)
Z disc
M line
Z disc
H zone
I band
A band
I band
Sarcomere
(b) Diagram based on an electron micrograph

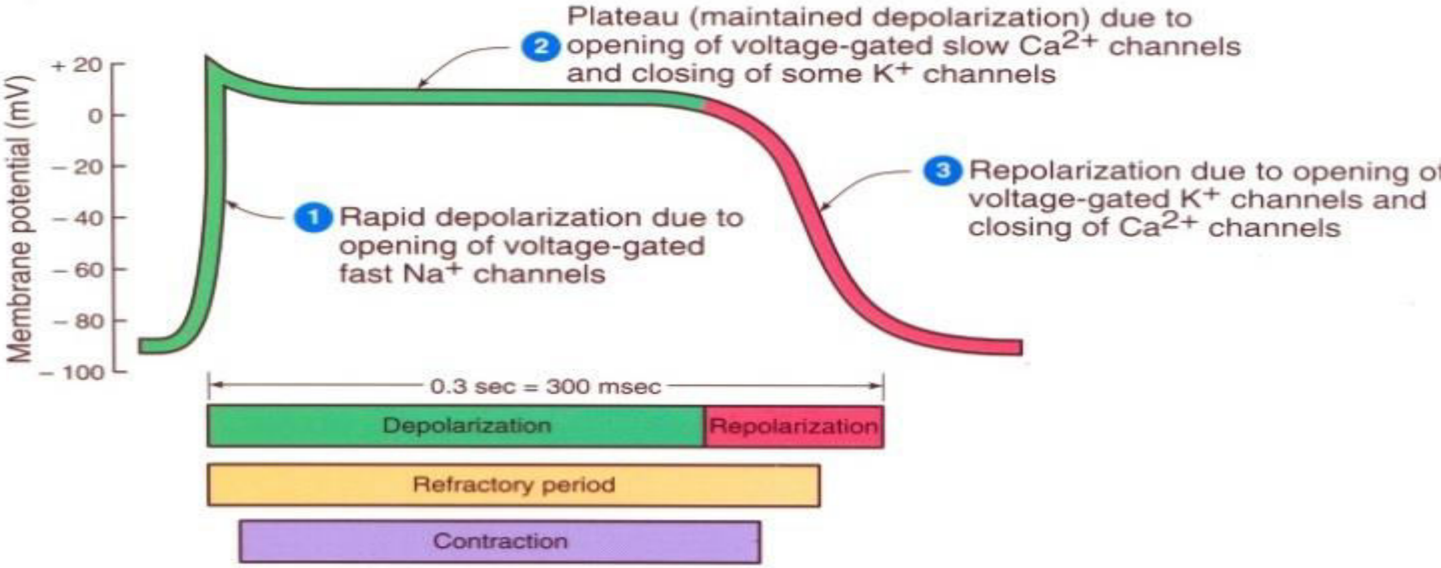

Gap junction channels
A
42 Å
35 Å
52 Å
87 Å
B
Open
Closed

Cardiac Muscle Vs Skeletal Muscle

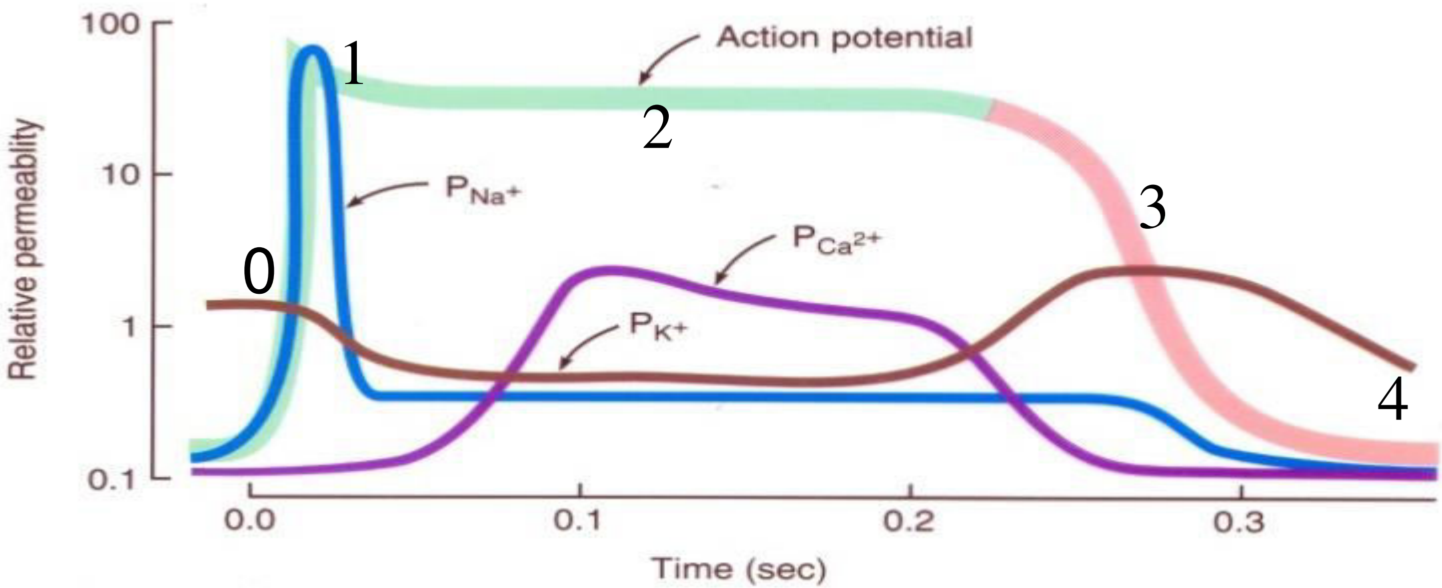
- Syncytium structure
- Gap Junction (electrical coupling) low resistance area
- Poorly developed Sarcoplasmic reticulum (SR)
- Transverse (T)Tubule on Z-line (i.e.One T-tubule per sarcomere)
- Rich in mitochondria
- Low in nuclei

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



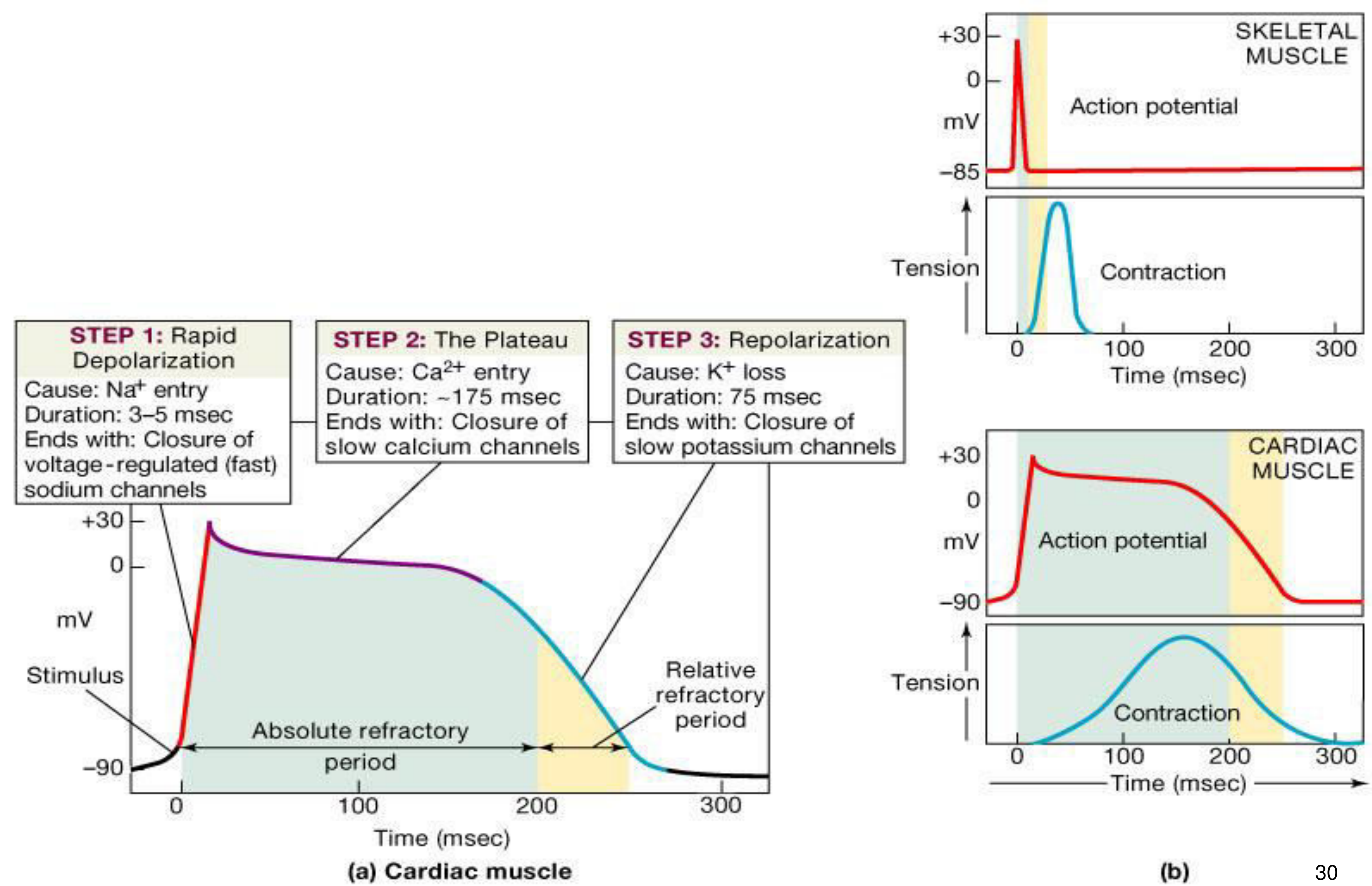


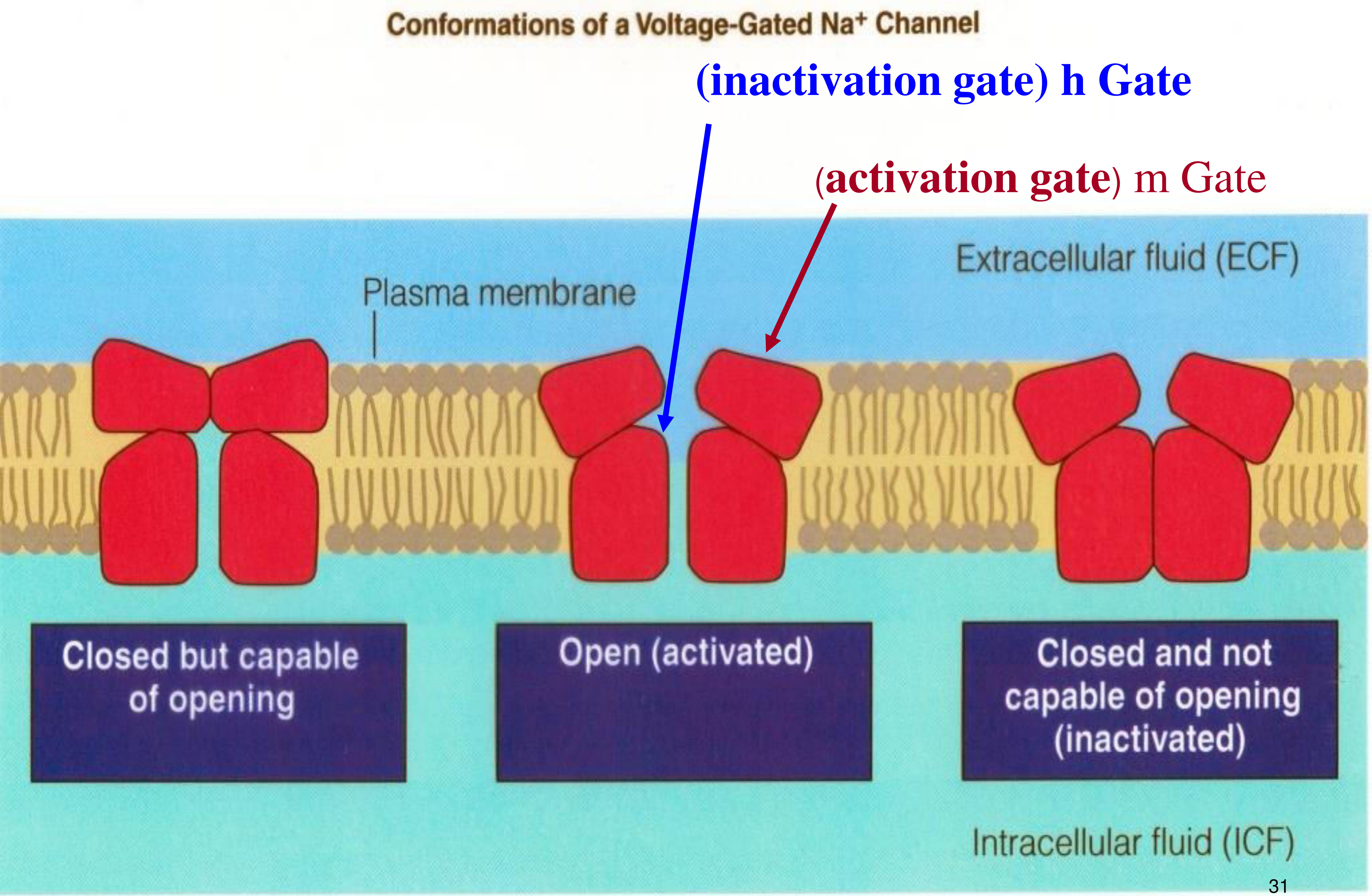
(a) Action potential, refractory period, and contraction



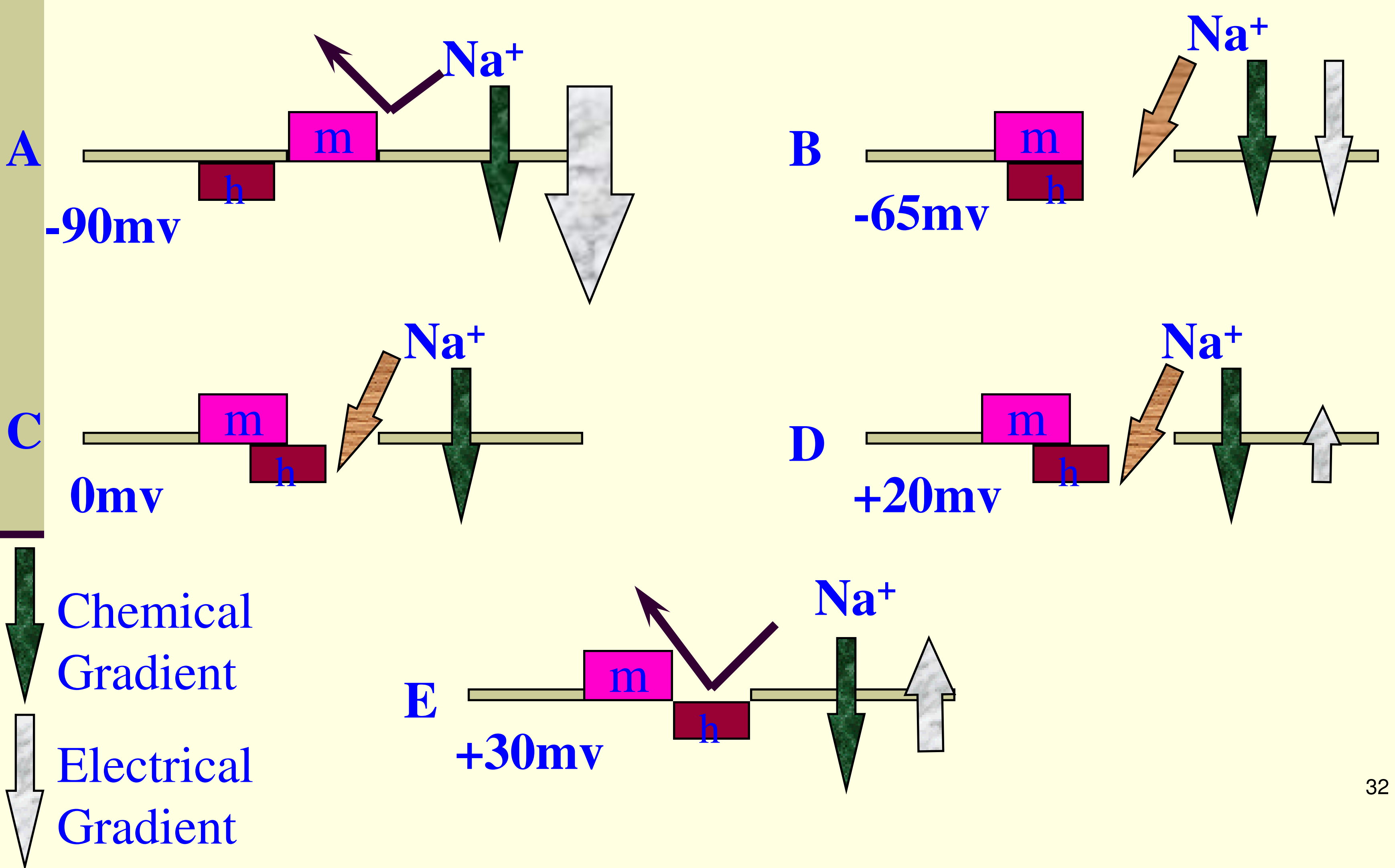
(b) Membrane permeability (P) changes

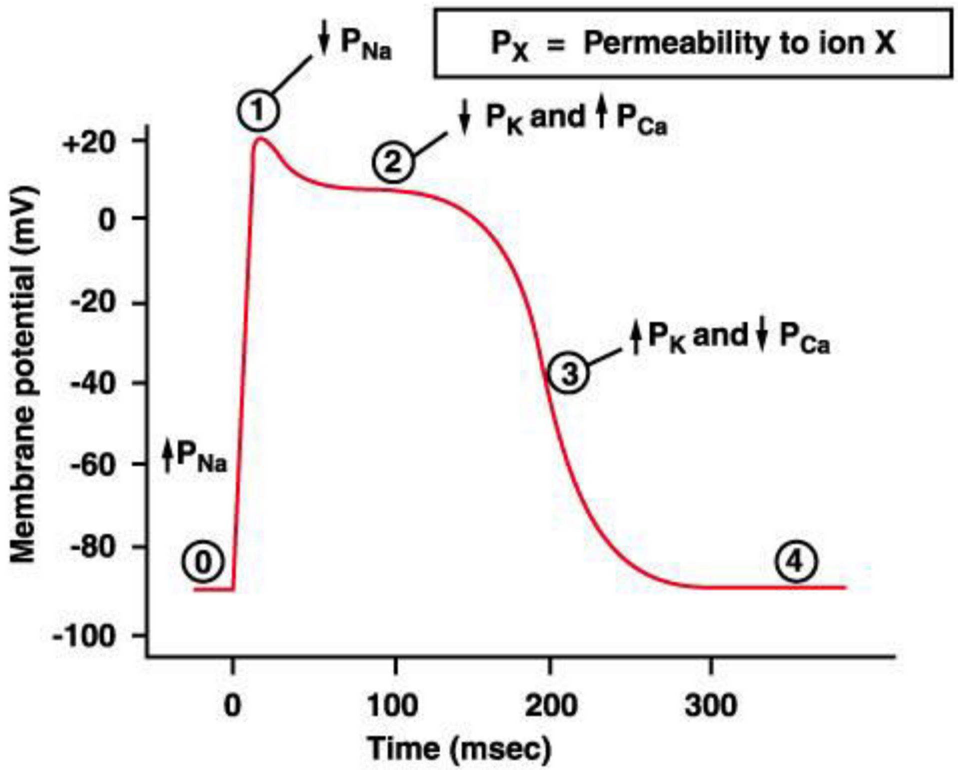
The Action Potential in Skeletal and Cardiac Muscle



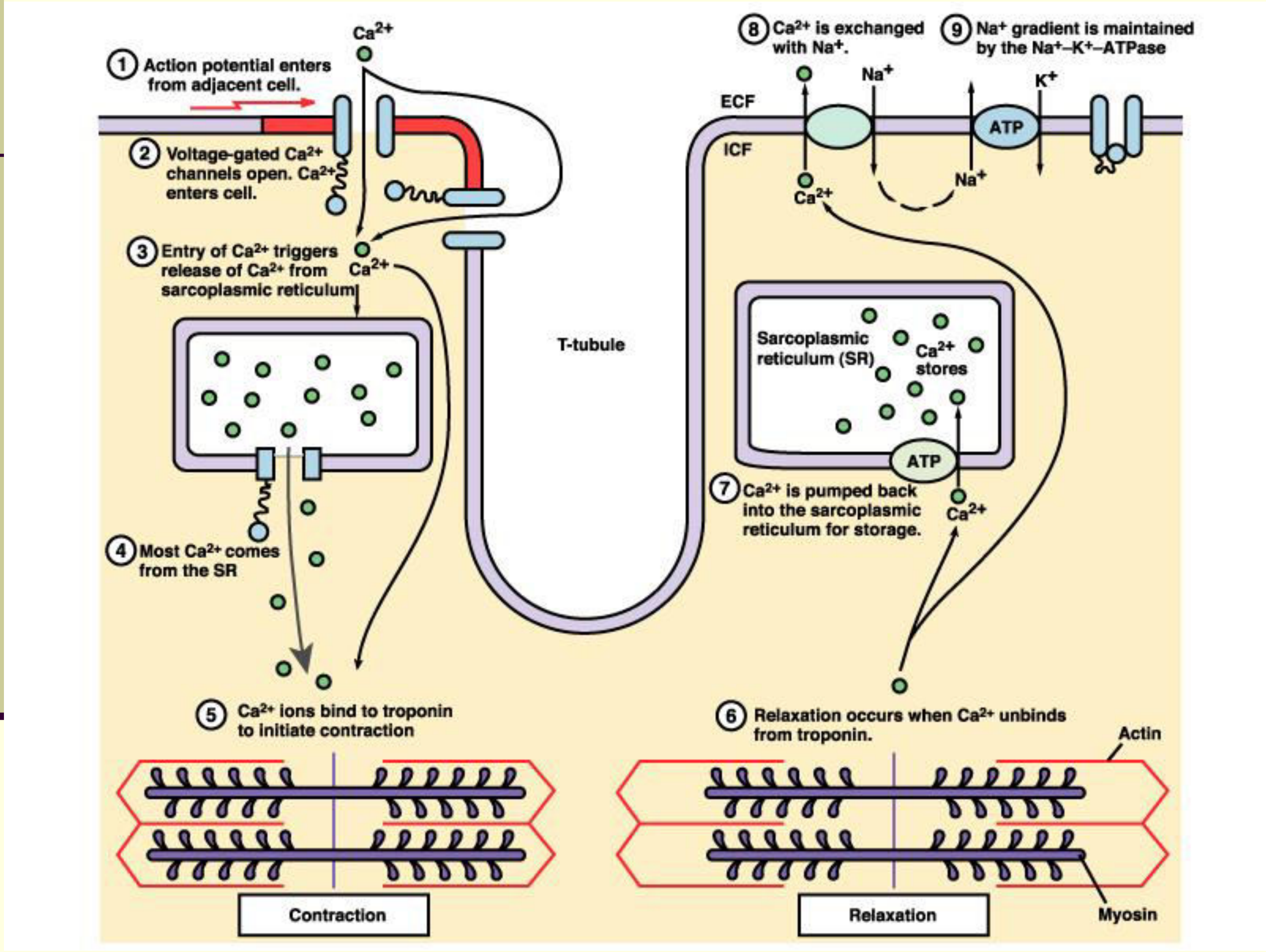

Conformations of a Voltage-Gated Na+ Channel
(inactivation gate) h Gate
(activation gate) m Gate
Extracellular fluid (ECF)
Plasma membrane
Closed but capable of opening
Open (activated)
Closed and not capable of opening (inactivated)
Intracellular fluid (ICF)

PHASE 0 OF THE FAST FIBER ACTION POTENTIAL



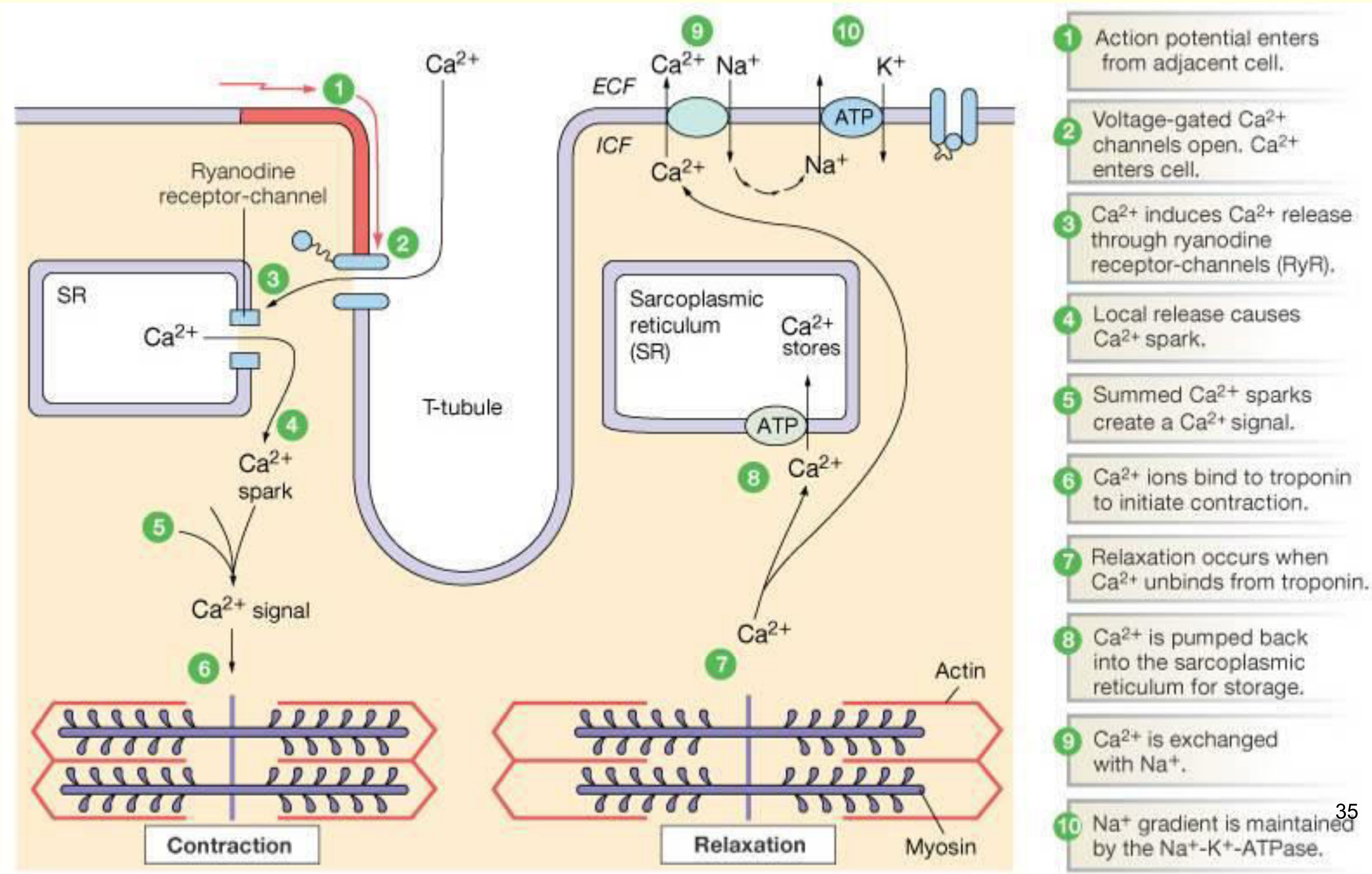


Phase	Membrane channels
0	Na^+ channels open
1	Na^+ channels close
2	Ca^{2+} channels open; fast K^+ channels close
3	Ca^{2+} channels close; slow K^+ channels open
4	Resting potential

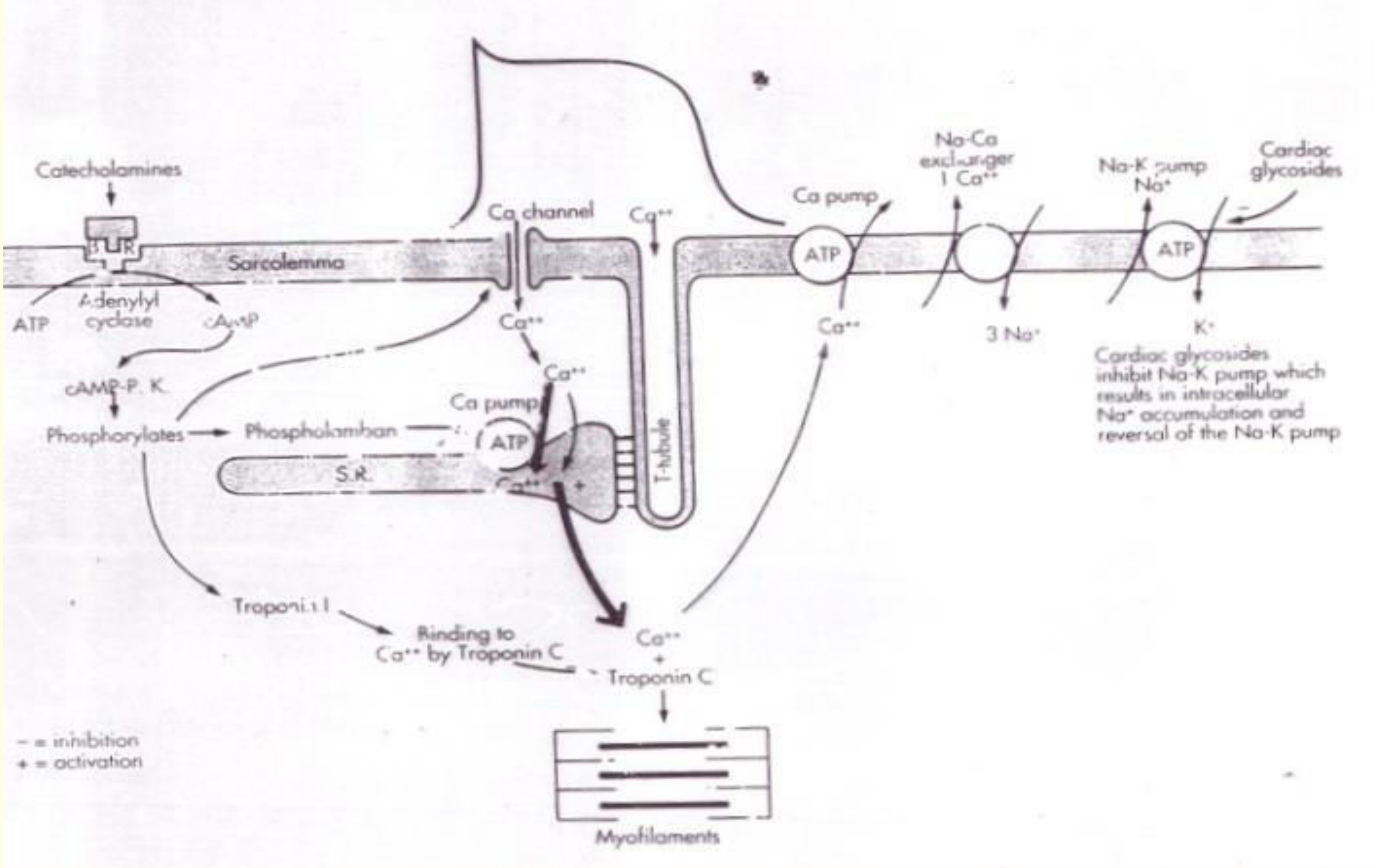


Ca2+
1 Action potential enters from adjacent cell.
2 Voltage-gated Ca2+ channels open. Ca2+ enters cell.
3 Entry of Ca2+ triggers release of Ca2+ from sarcoplasmic reticulum
Ca2+
4 Most Ca2+ comes from the SR
5 Ca2+ ions bind to troponin to initiate contraction
T-tubule
8 Ca2+ is exchanged with Na+.
9 Na+ gradient is maintained by the Na+–K+–ATPase
ECF
ICF
Na+
K+
ATP
Na+
Ca2+
Sarcoplasmic reticulum (SR)
Ca2+ stores
ATP
7 Ca2+ is pumped back into the sarcoplasmic reticulum for storage.
Ca2+
6 Relaxation occurs when Ca2+ unbinds from troponin.
Actin
Myosin
Contraction
Relaxation

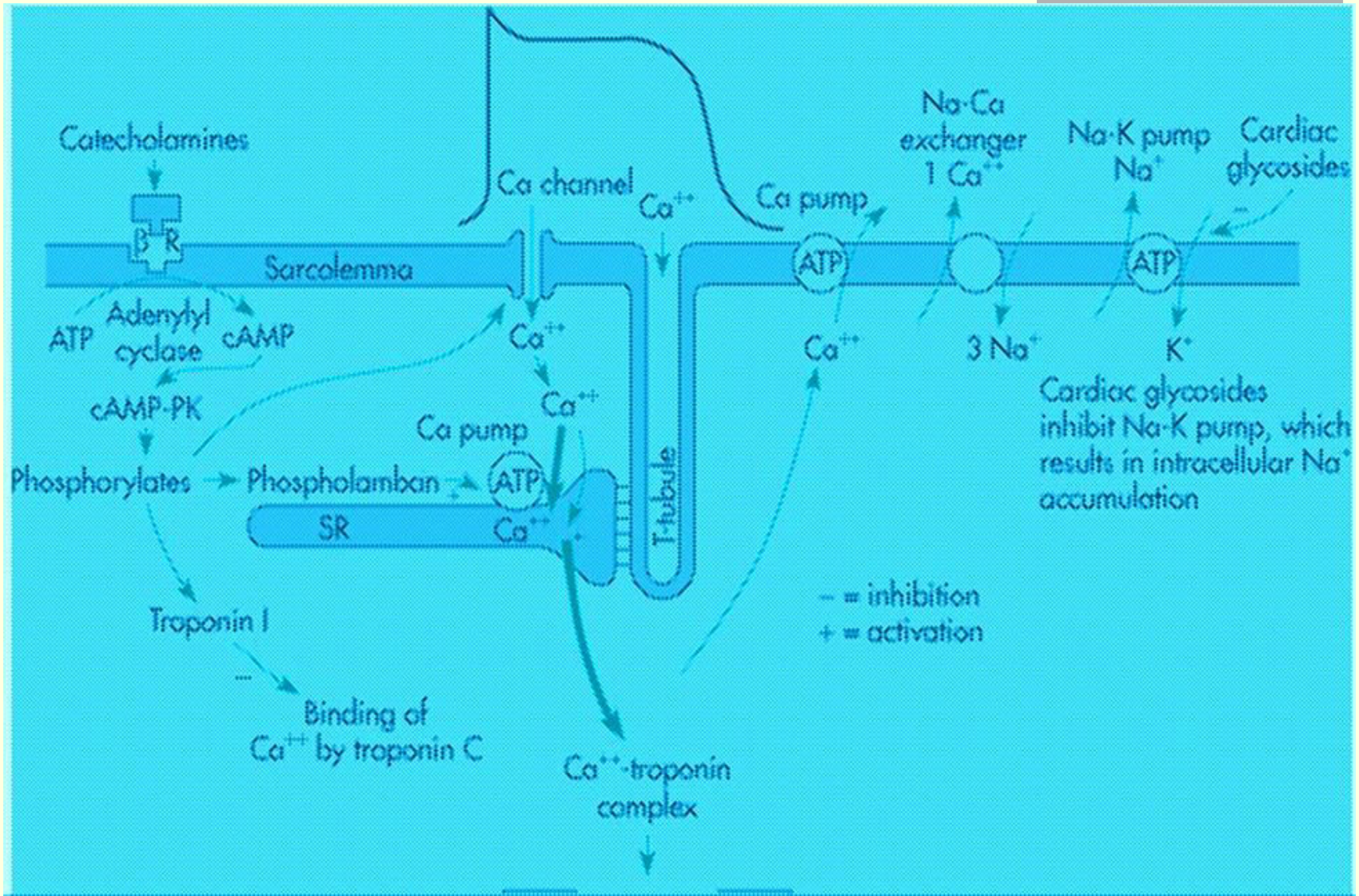
Mechanism of Cardiac Muscle Excitation, Contraction & Relaxation



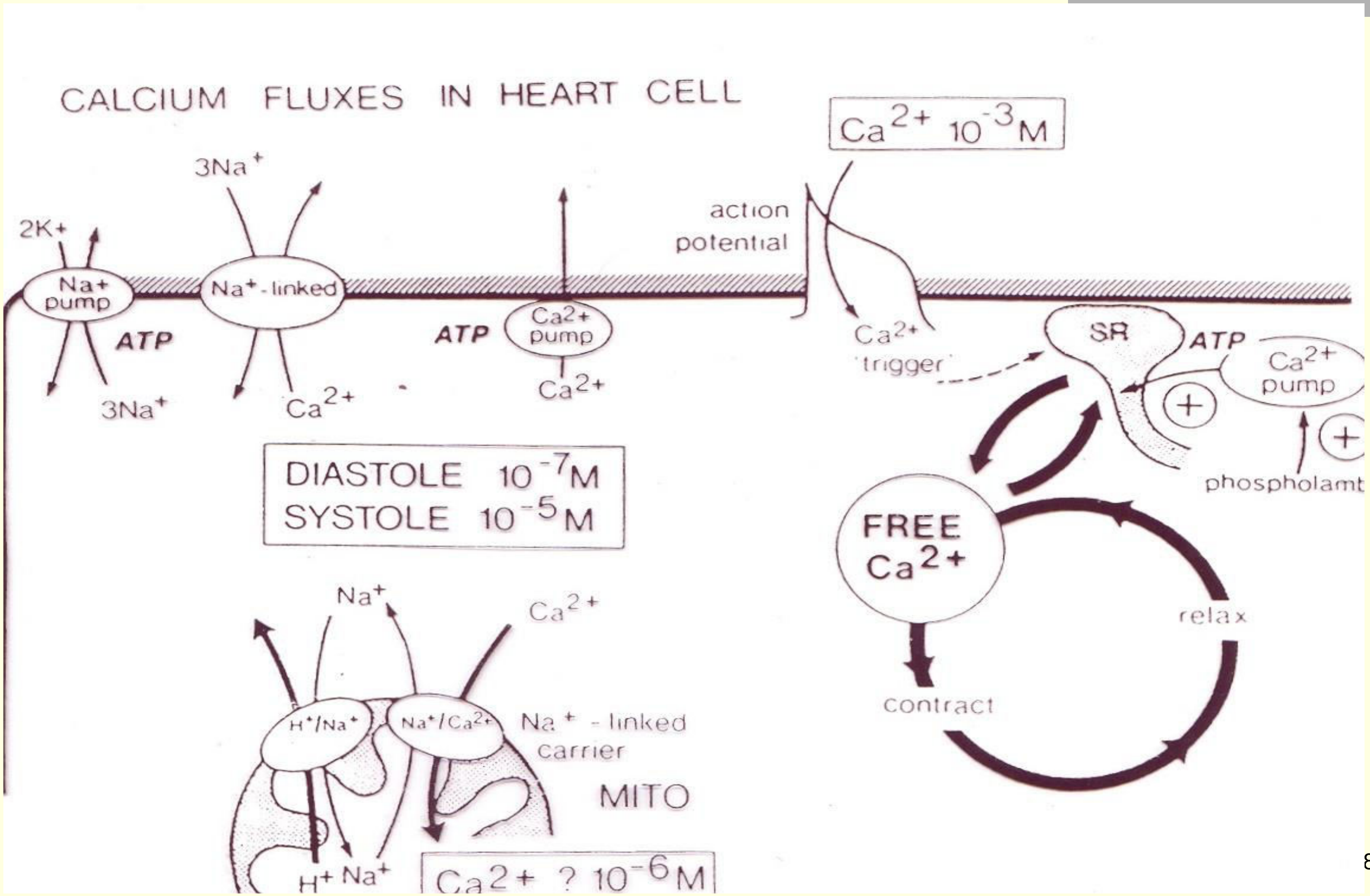
Intracellular Calcium Homeostasis…1



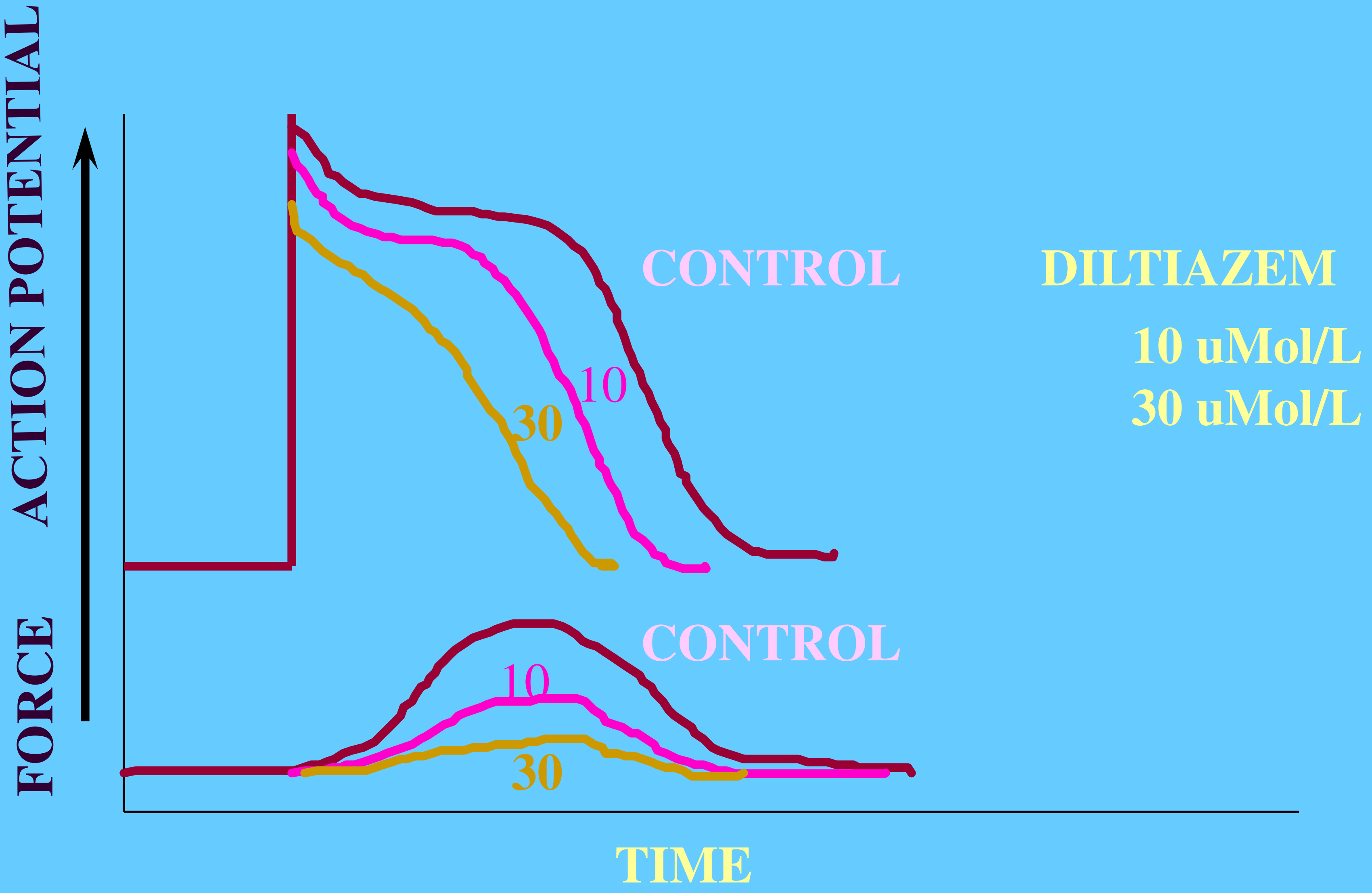
Intracellular Calcium Homeostasis…1



Intracellular Calcium Homeostasis…2

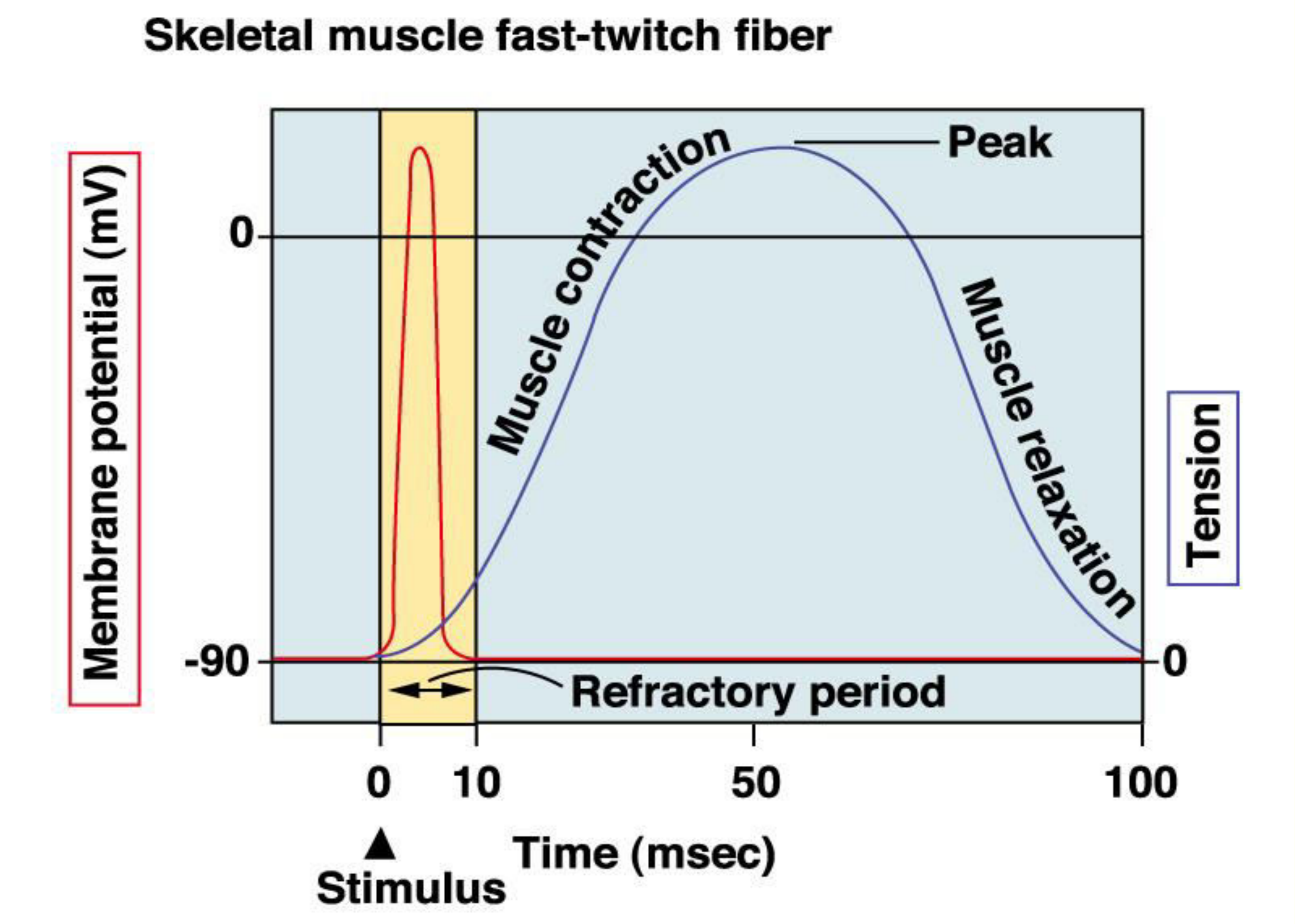


EFFECTS OF Ca++ CHANNEL BLOCKERS AND THE CARDIAC CELL ACTION POTENTIAL

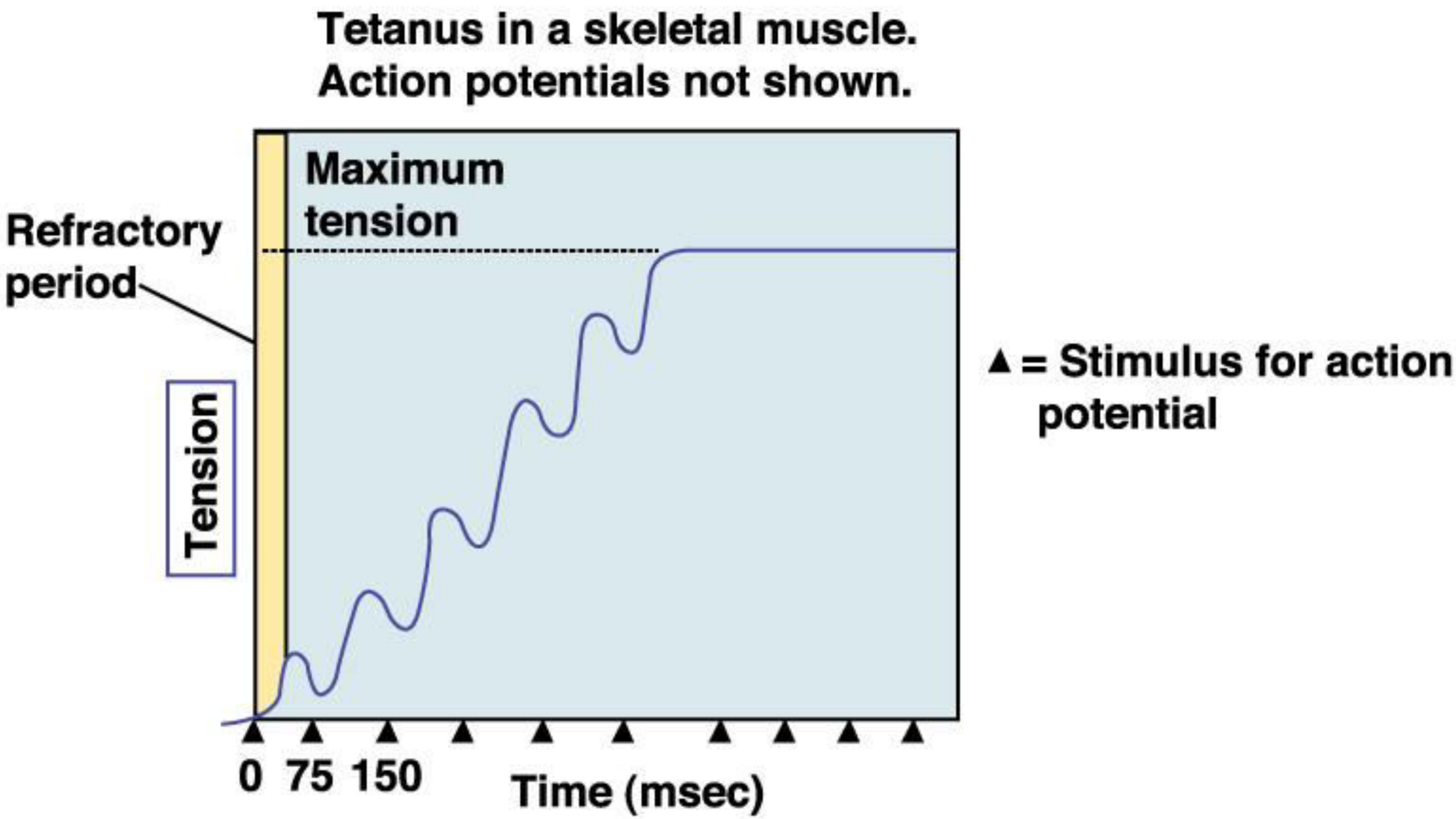


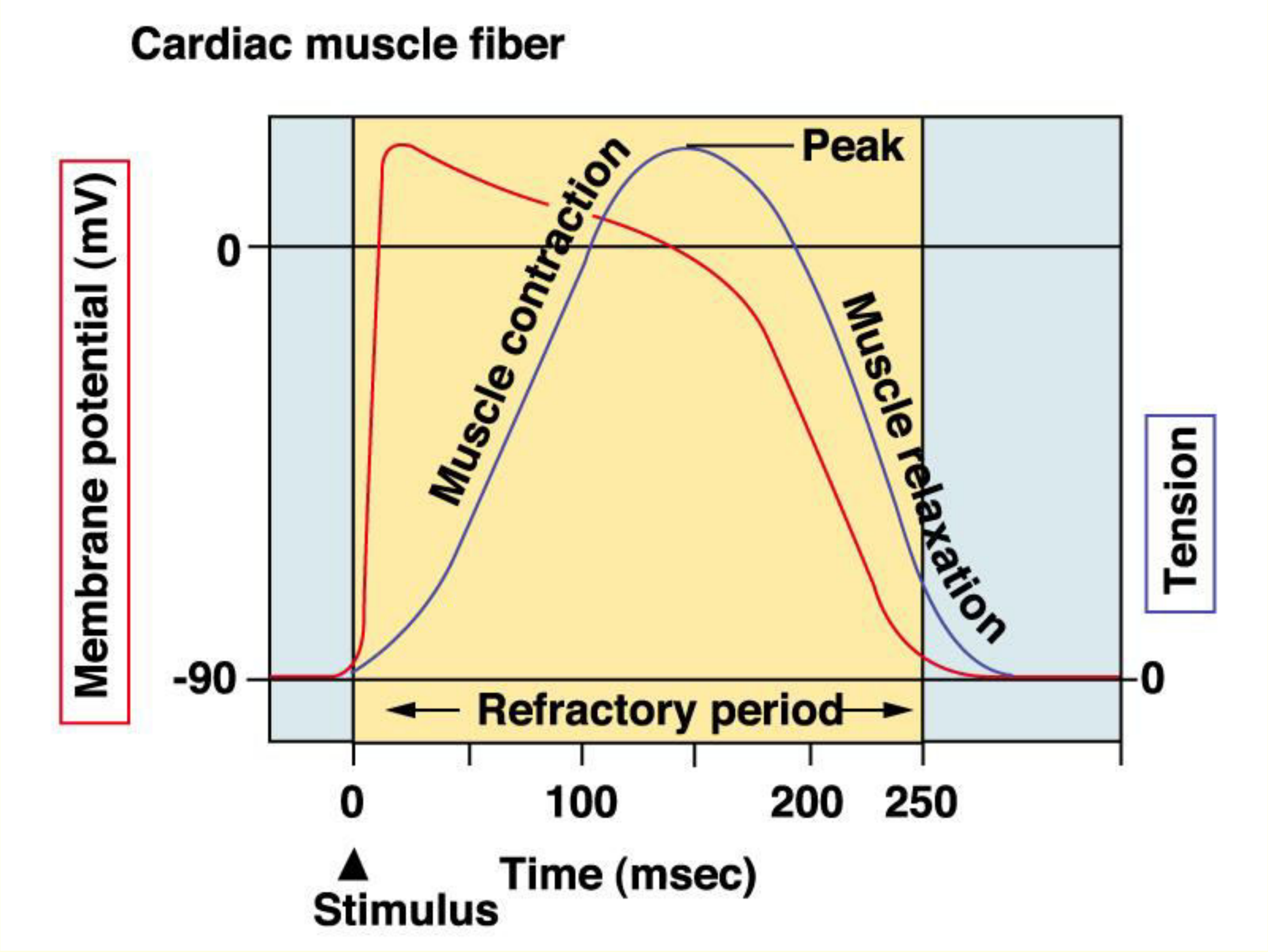
Cardiac Muscle action potential Vs. Skeletal Muscle

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

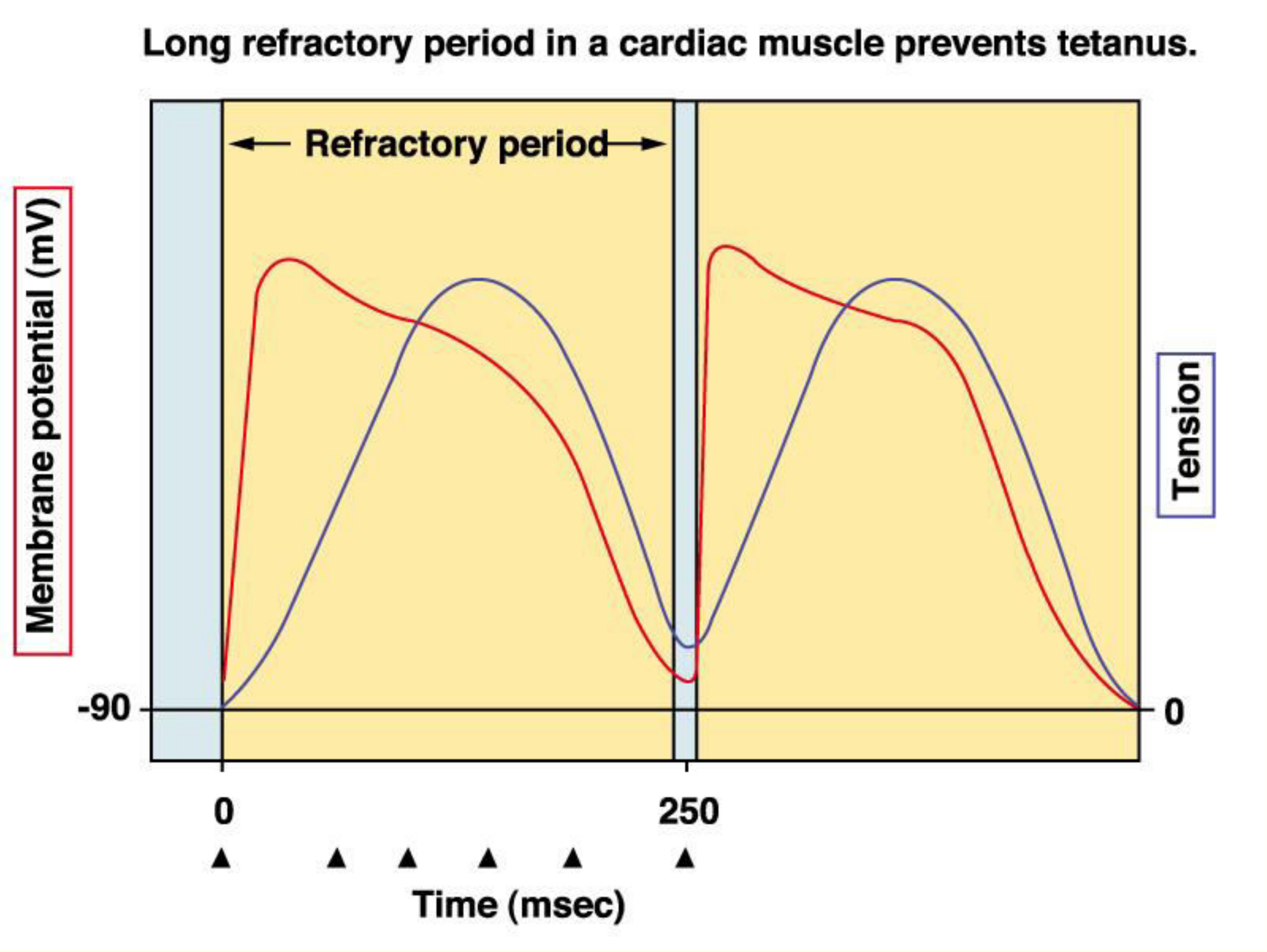


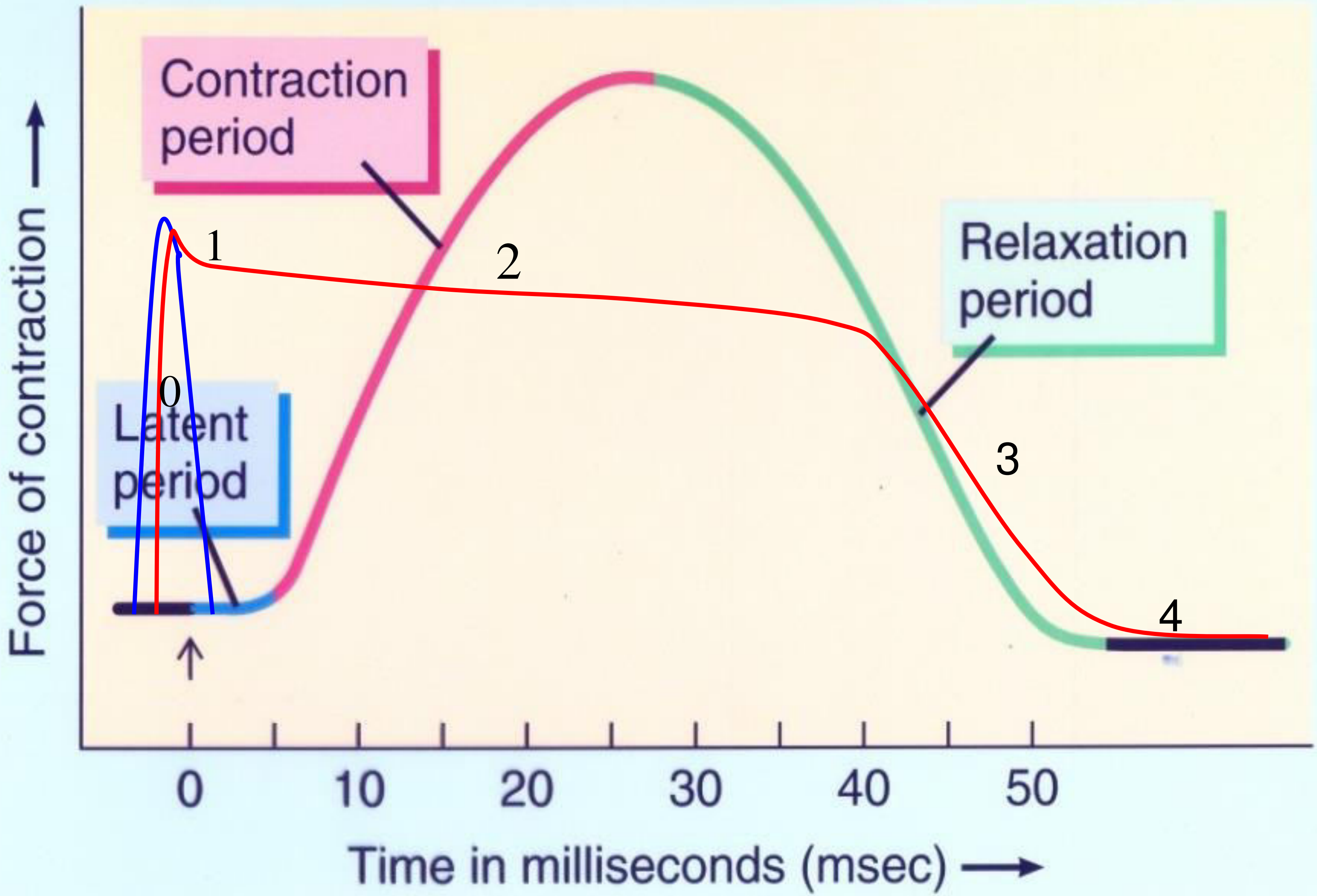
Skeletal muscle fast-twitch fiber
Membrane potential (mV)
0
-90
Muscle contraction
Peak
Muscle relaxation
Tension
0
Refractory period
0
10
50
100
Stimulus
Time (msec)


Tetanus in a skeletal muscle.
Action potentials not shown.
Maximum
tension
Refractory
period
Tension
▲ = Stimulus for action
potential
0
75
150
Time (msec)

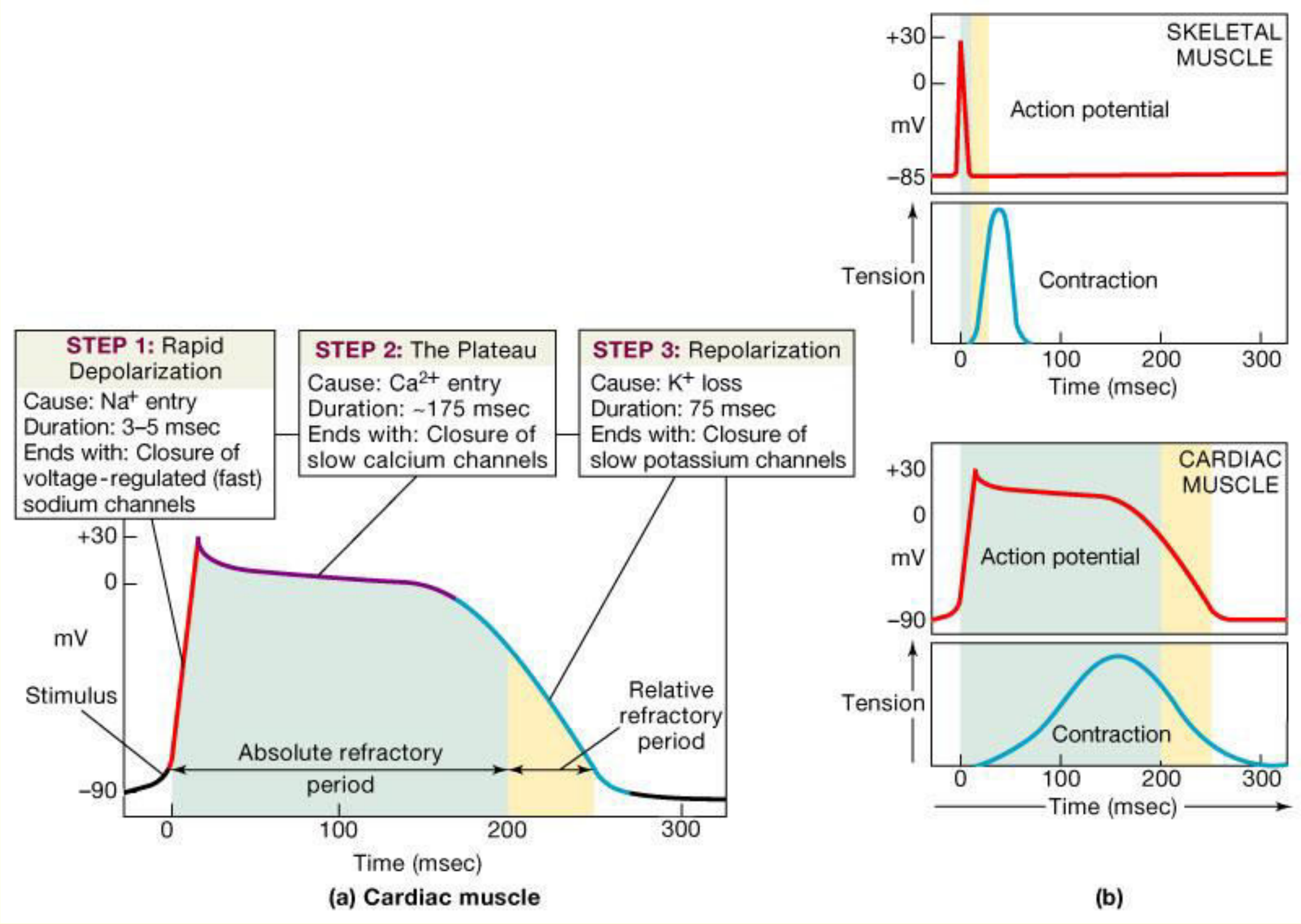


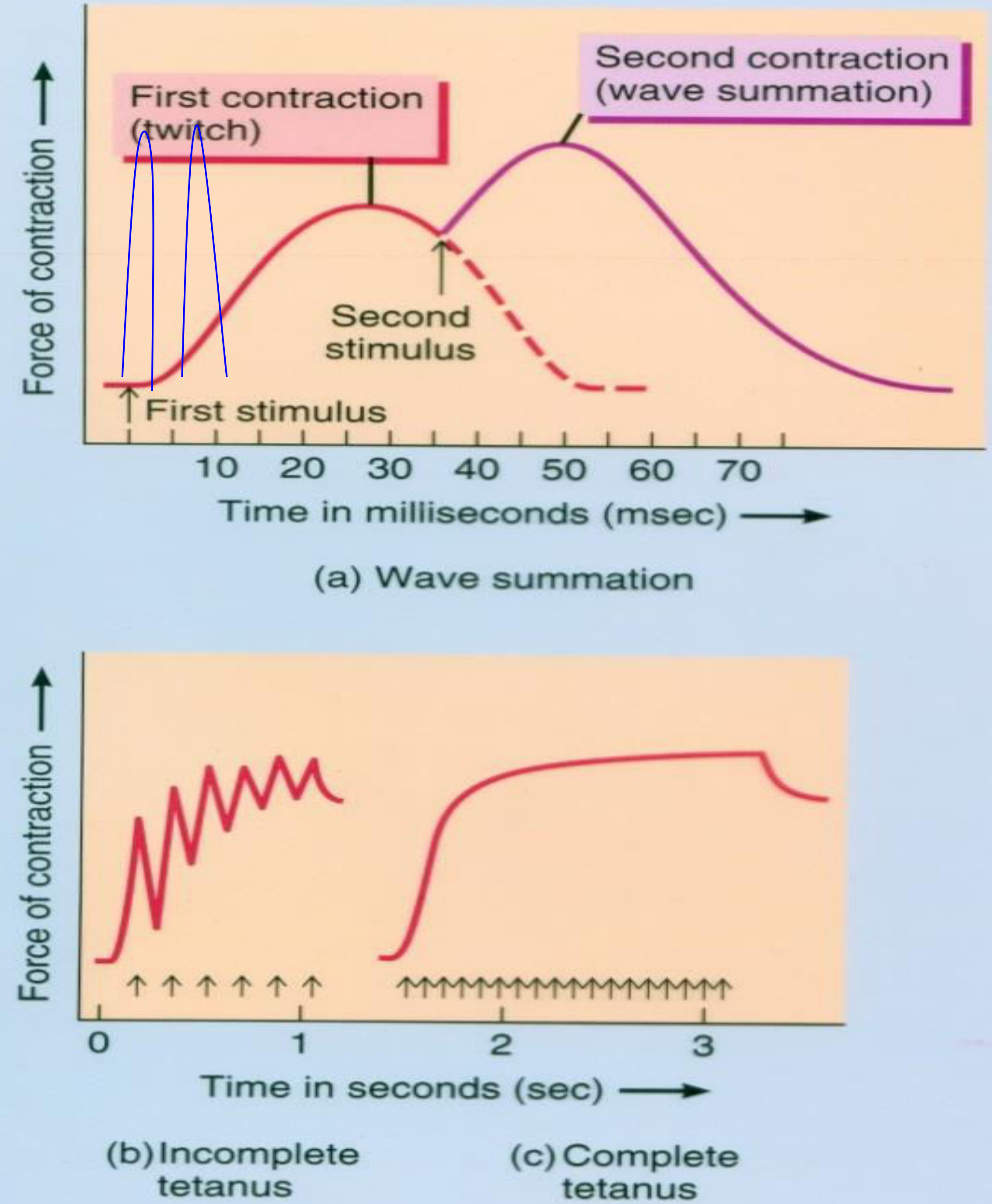
Cardiac muscle fiber
Membrane potential (mV)
0
-90
Peak
Muscle contraction
Muscle relaxation
Tension
0
Refractory period
0
100
200
250
Stimulus
Time (msec)


Long refractory period in a cardiac muscle prevents tetanus.
Refractory period
Membrane potential (mV)
Tension
-90
0
0
250
Time (msec)

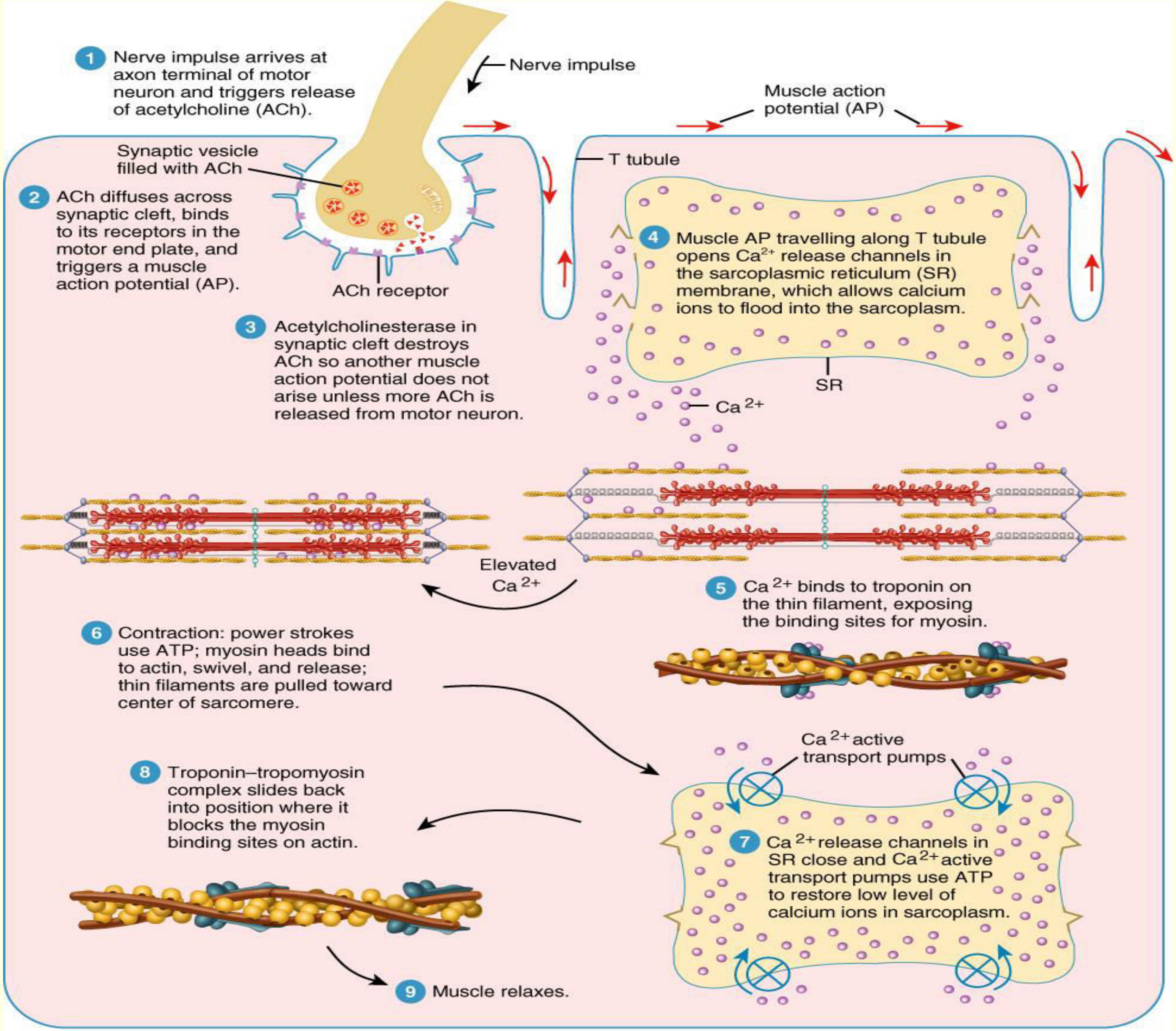

Contraction period
Relaxation period
Latent period
Force of contraction
Time in milliseconds (msec)
0
10
20
30
40
50
0
1
2
3
4

The Action Potential in Skeletal and Cardiac Muscle



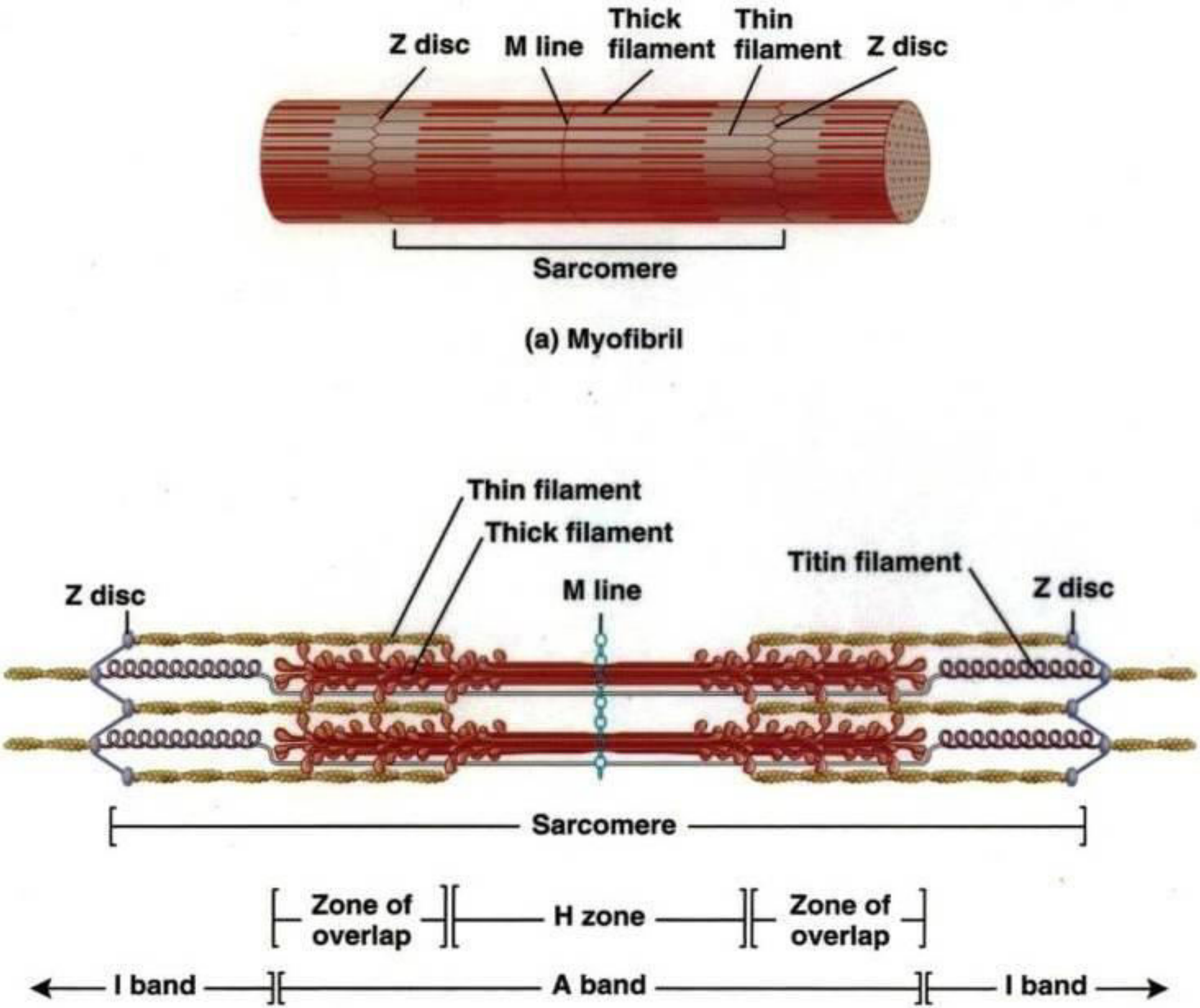


Force of contraction
First contraction
(twitch)
Second contraction
(wave summation)
Second
stimulus
First stimulus
10 20 30 40 50 60 70
Time in milliseconds (msec)
(a) Wave summation
Force of contraction
0 1 2 3
Time in seconds (sec)
(b) Incomplete
tetanus
(c) Complete
tetanus



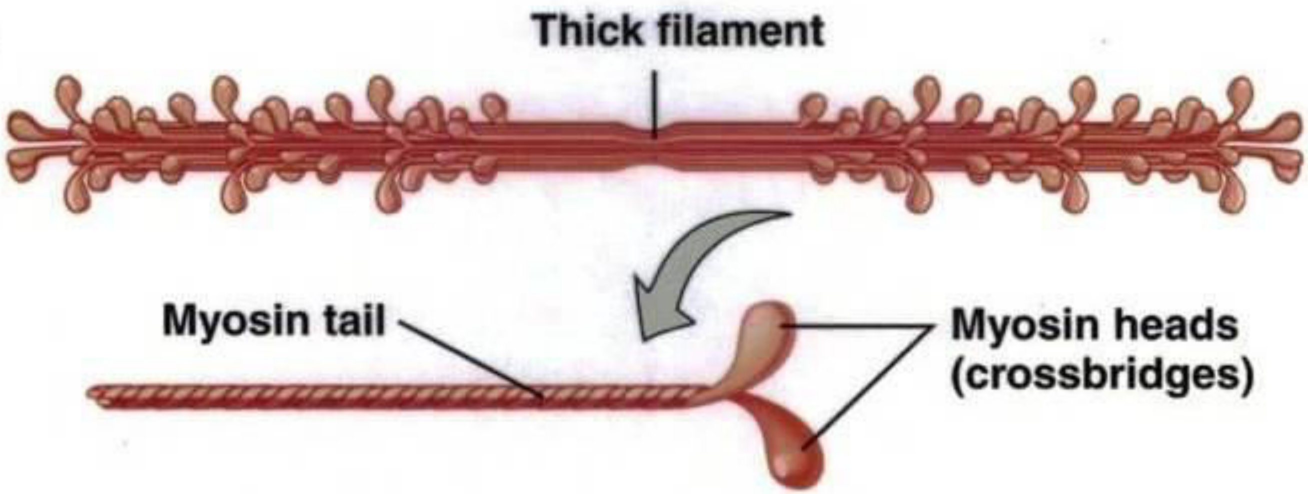
1 Nerve impulse arrives at axon terminal of motor neuron and triggers release of acetylcholine (ACh).
Nerve impulse
Muscle action potential (AP)
Synaptic vesicle filled with ACh
T tubule
2 ACh diffuses across synaptic cleft, binds to its receptors in the motor end plate, and triggers a muscle action potential (AP).
ACh receptor
4 Muscle AP travelling along T tubule opens Ca2+ release channels in the sarcoplasmic reticulum (SR) membrane, which allows calcium ions to flood into the sarcoplasm.
3 Acetylcholinesterase in synaptic cleft destroys ACh so another muscle action potential does not arise unless more ACh is released from motor neuron.
SR
Ca 2+
Elevated Ca 2+
5 Ca 2+ binds to troponin on the thin filament, exposing the binding sites for myosin.
6 Contraction: power strokes use ATP; myosin heads bind to actin, swivel, and release; thin filaments are pulled toward center of sarcomere.
Ca 2+ active transport pumps
8 Troponin–tropomyosin complex slides back into position where it blocks the myosin binding sites on actin.
7 Ca 2+ release channels in SR close and Ca 2+ active transport pumps use ATP to restore low level of calcium ions in sarcoplasm.
9 Muscle relaxes.

10.12

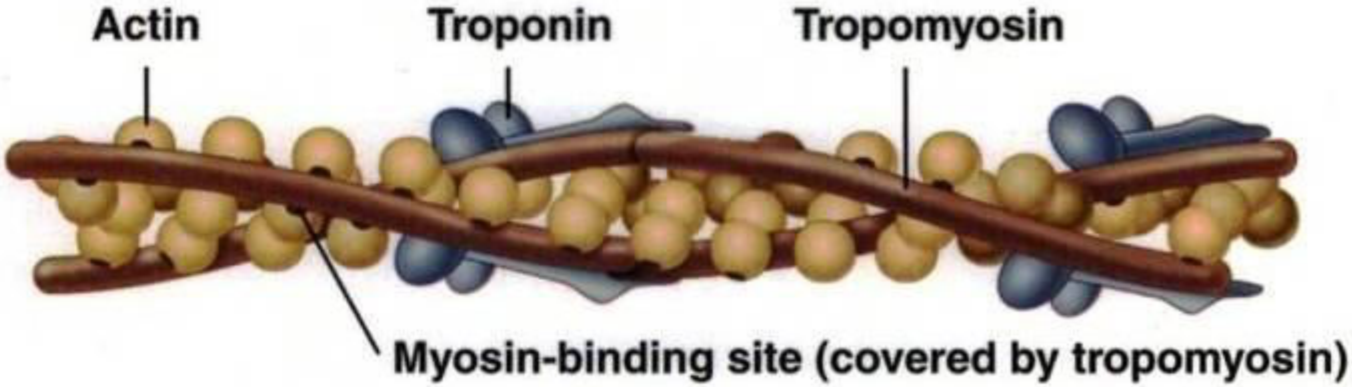


(a) Myofibril

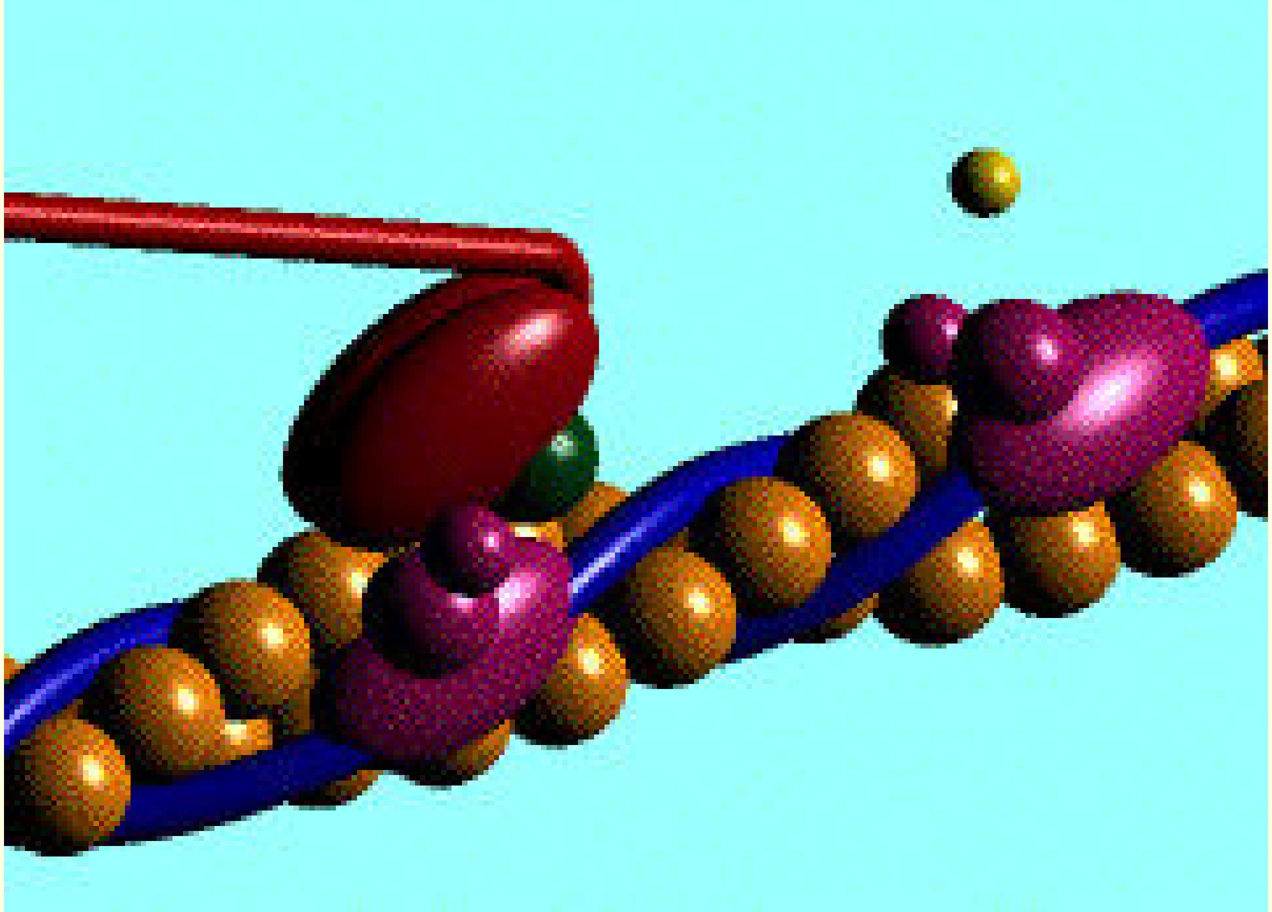
(b) Filaments

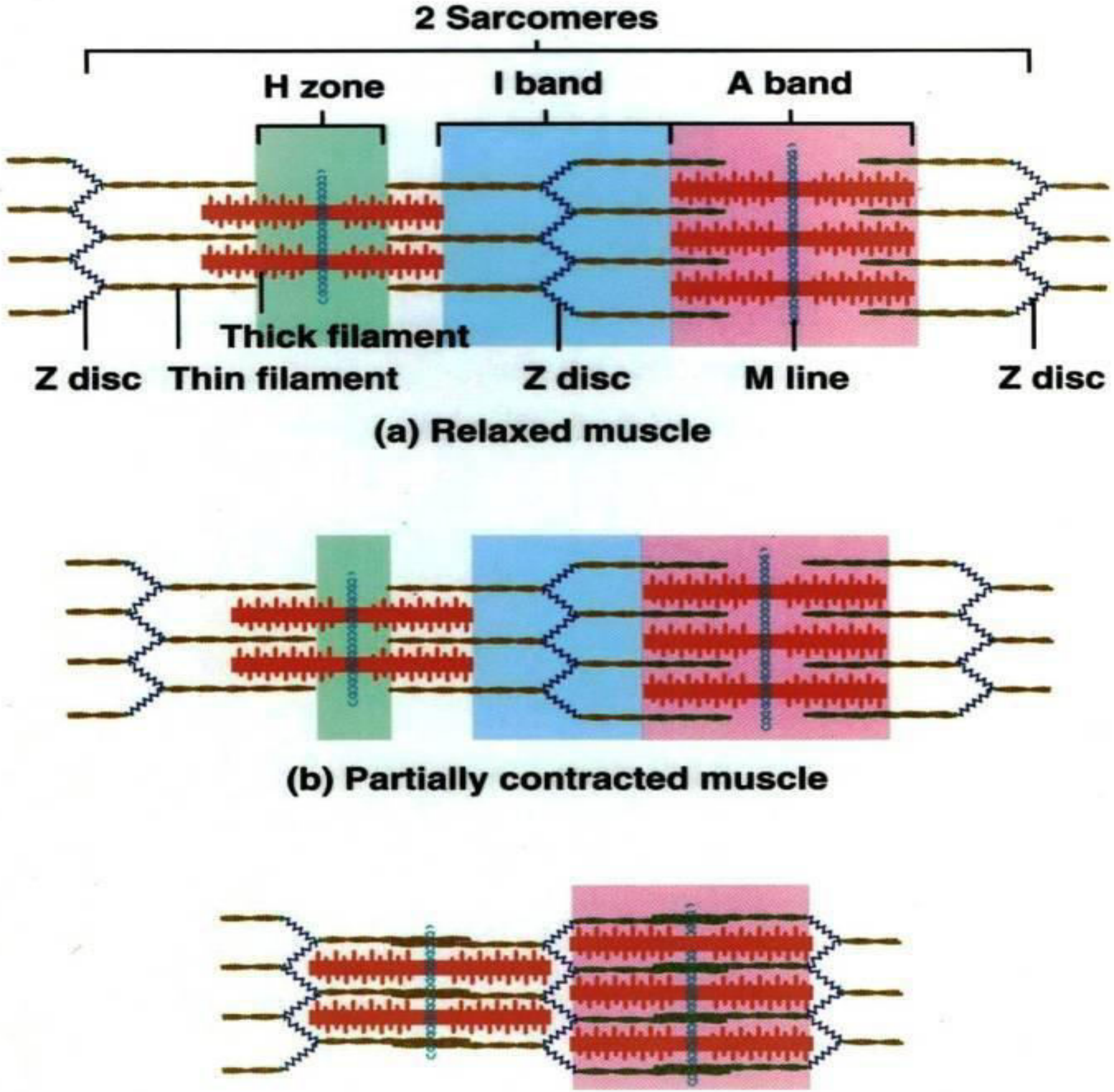


(a) One thick filament (above) and a myosin molecule (below)



(b) Portion of a thin filament

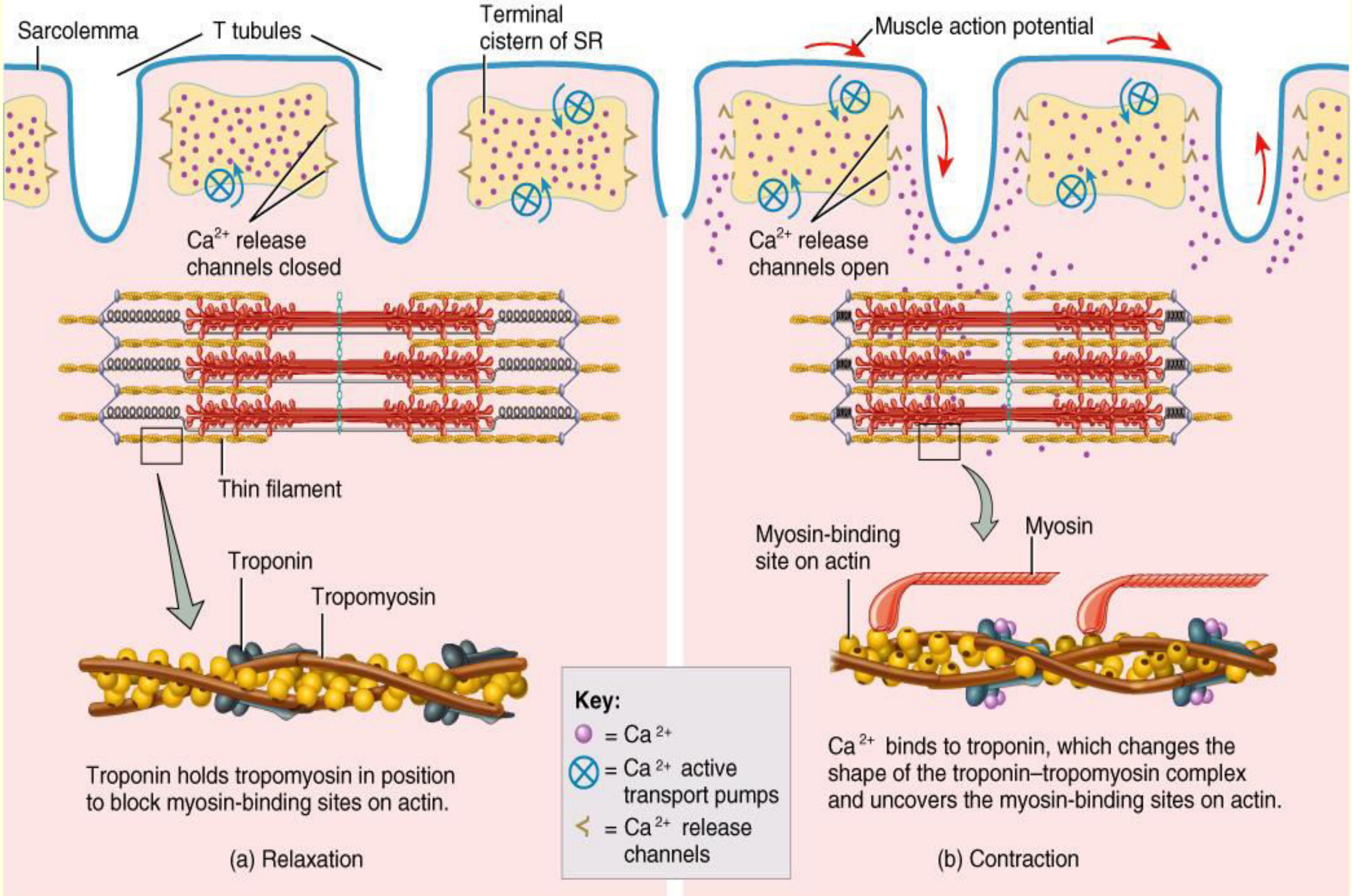




(a) Relaxed muscle

(b) Partially contracted muscle

(c) Maximally contracted muscle

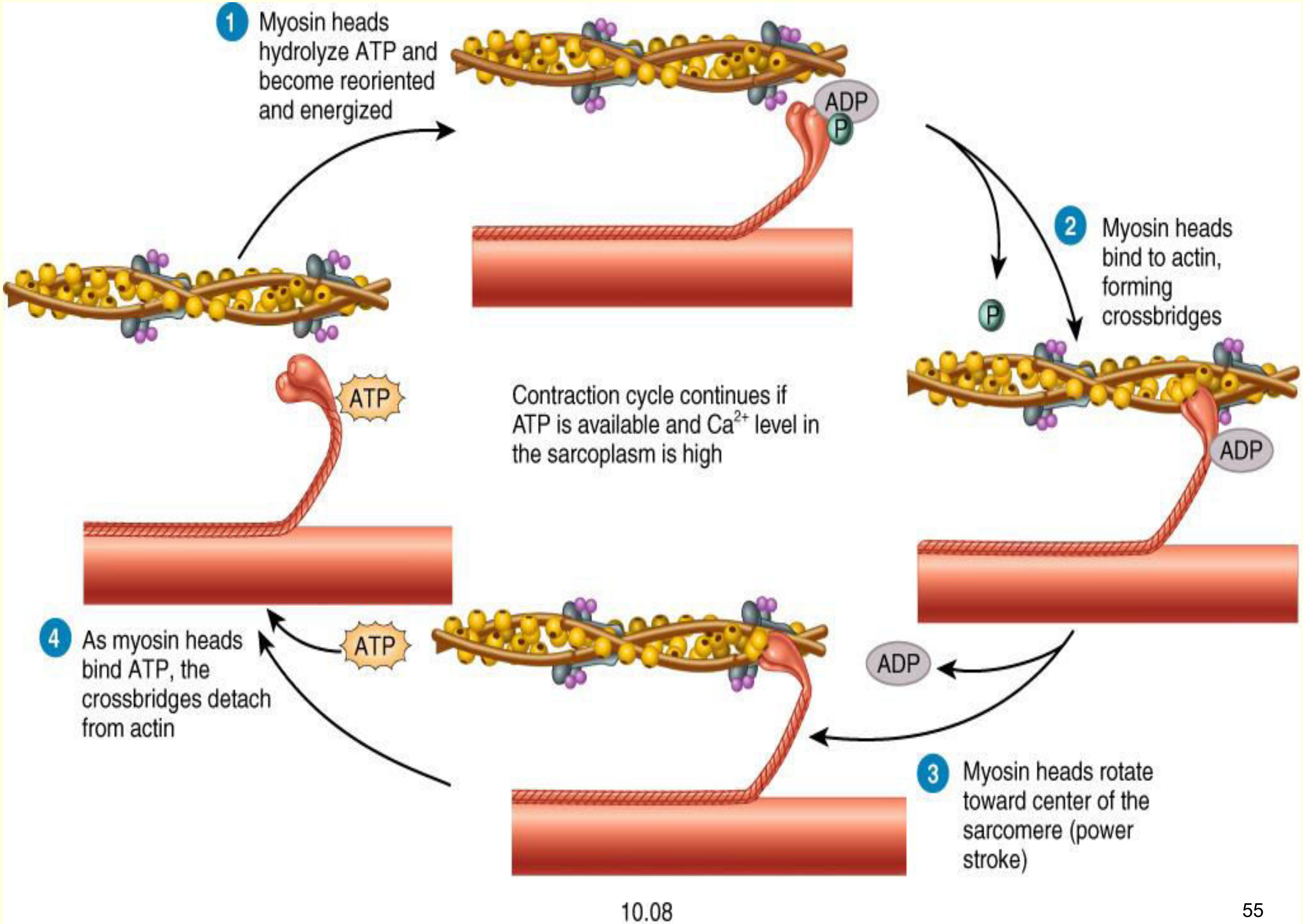


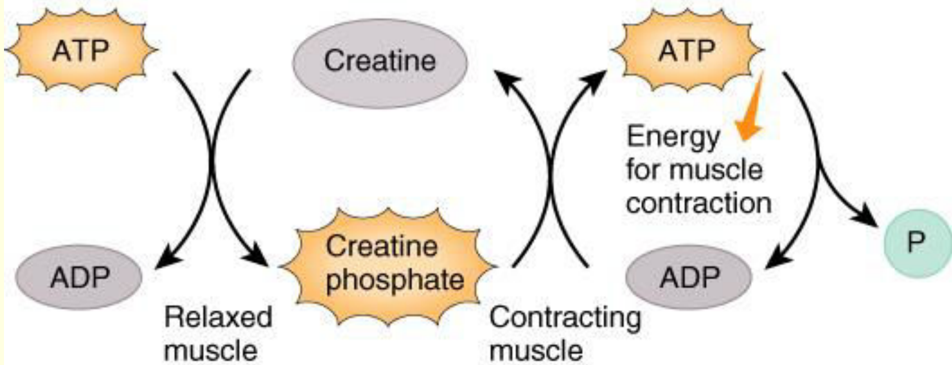
Sarcolemma
T tubules
Terminal cistern of SR
Muscle action potential
Ca2+ release channels closed
Ca2+ release channels open
Thin filament
Myosin-binding site on actin
Myosin
Troponin
Tropomyosin
Troponin holds tropomyosin in position to block myosin-binding sites on actin.
Key:
= Ca 2+
= Ca 2+ active transport pumps
= Ca 2+ release channels
Ca 2+ binds to troponin, which changes the shape of the troponin–tropomyosin complex and uncovers the myosin-binding sites on actin.
(a) Relaxation
(b) Contraction

10.09

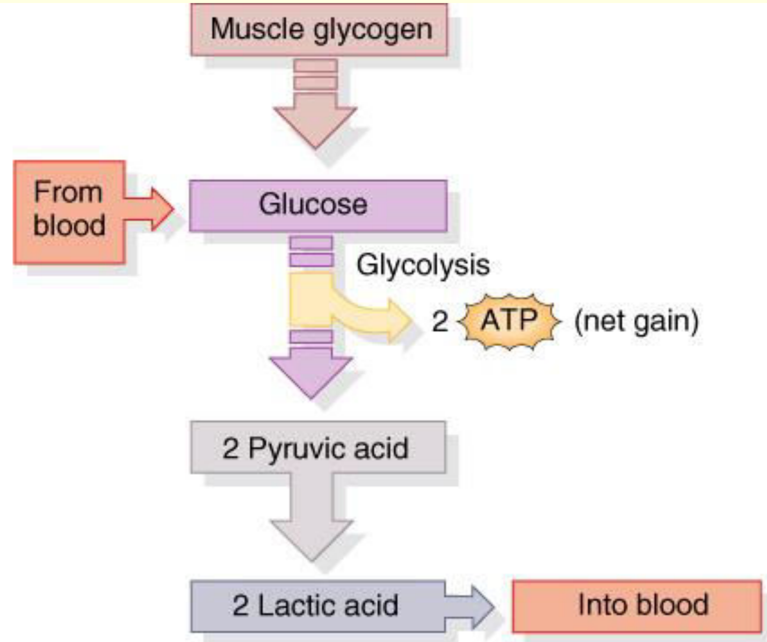
Cardiac Muscle contraction Vs. Skeletal Muscle

- 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

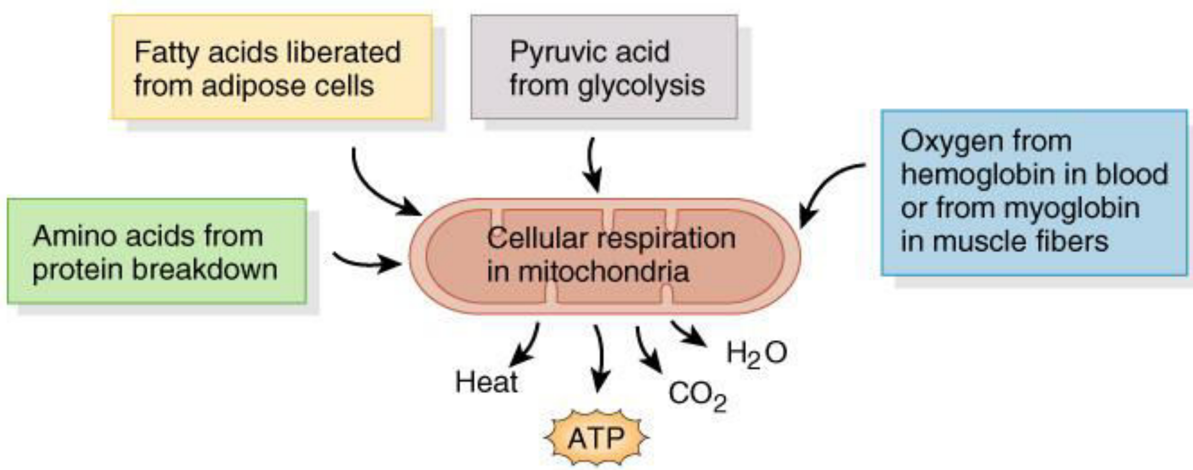

1 Myosin heads hydrolyze ATP and become reoriented and energized
ADP
P
2 Myosin heads bind to actin, forming crossbridges
P
ADP
Contraction cycle continues if ATP is available and Ca^{2+} level in the sarcoplasm is high
ATP
ADP
3 Myosin heads rotate toward center of the sarcomere (power stroke)
ATP
4 As myosin heads bind ATP, the crossbridges detach from actin
10.08



(a) ATP from creatine phosphate



(b) ATP from anaerobic respiration

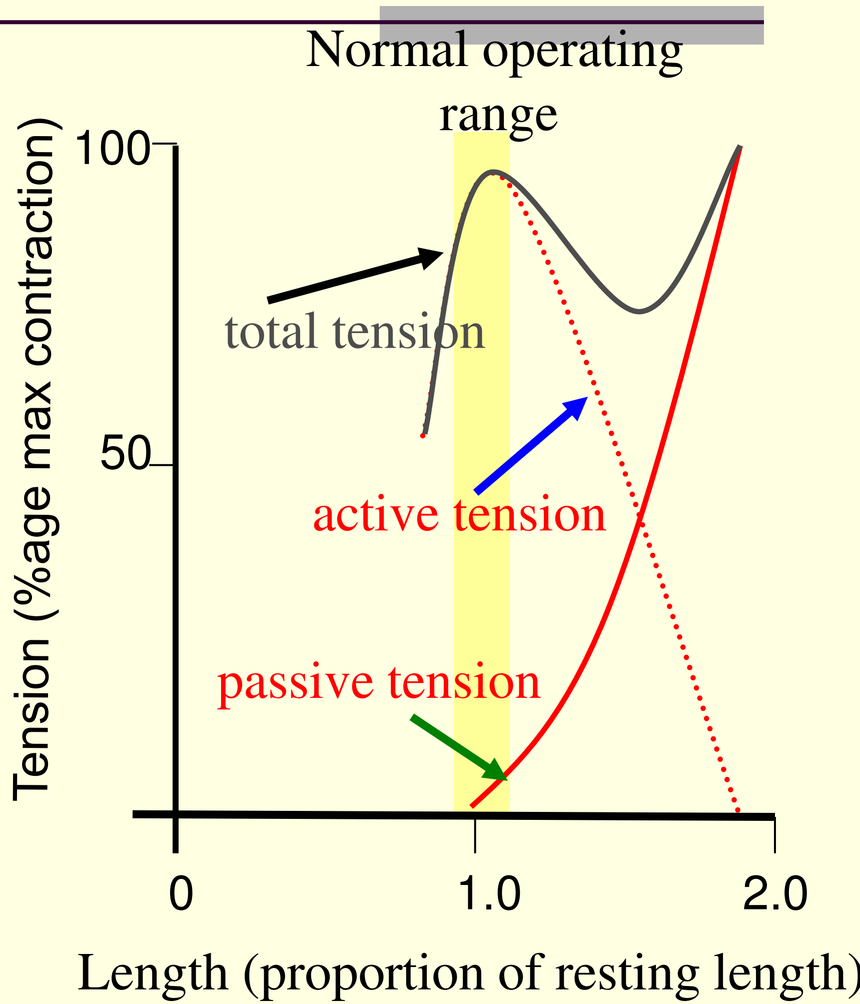


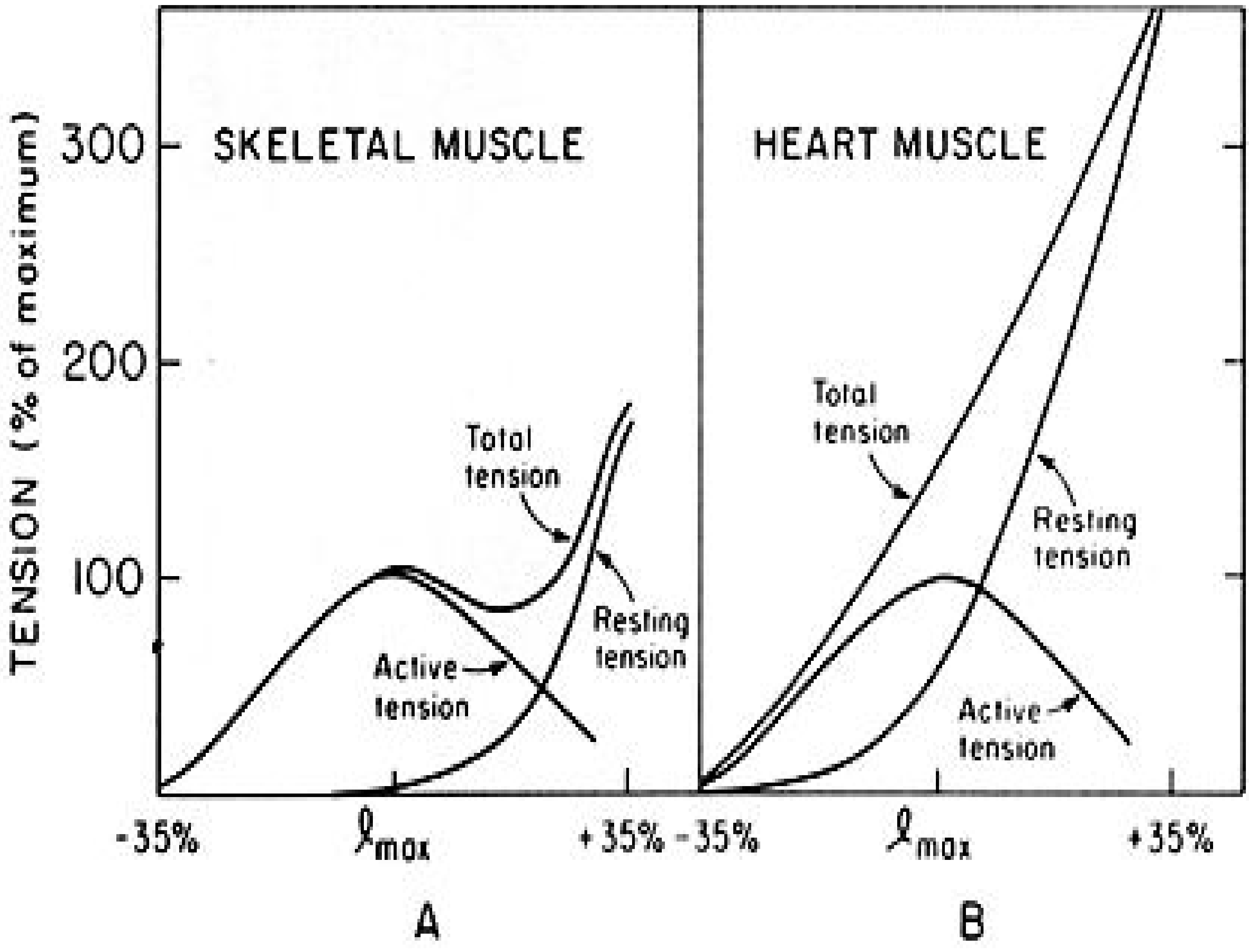
(c) ATP from aerobic cellular respiration

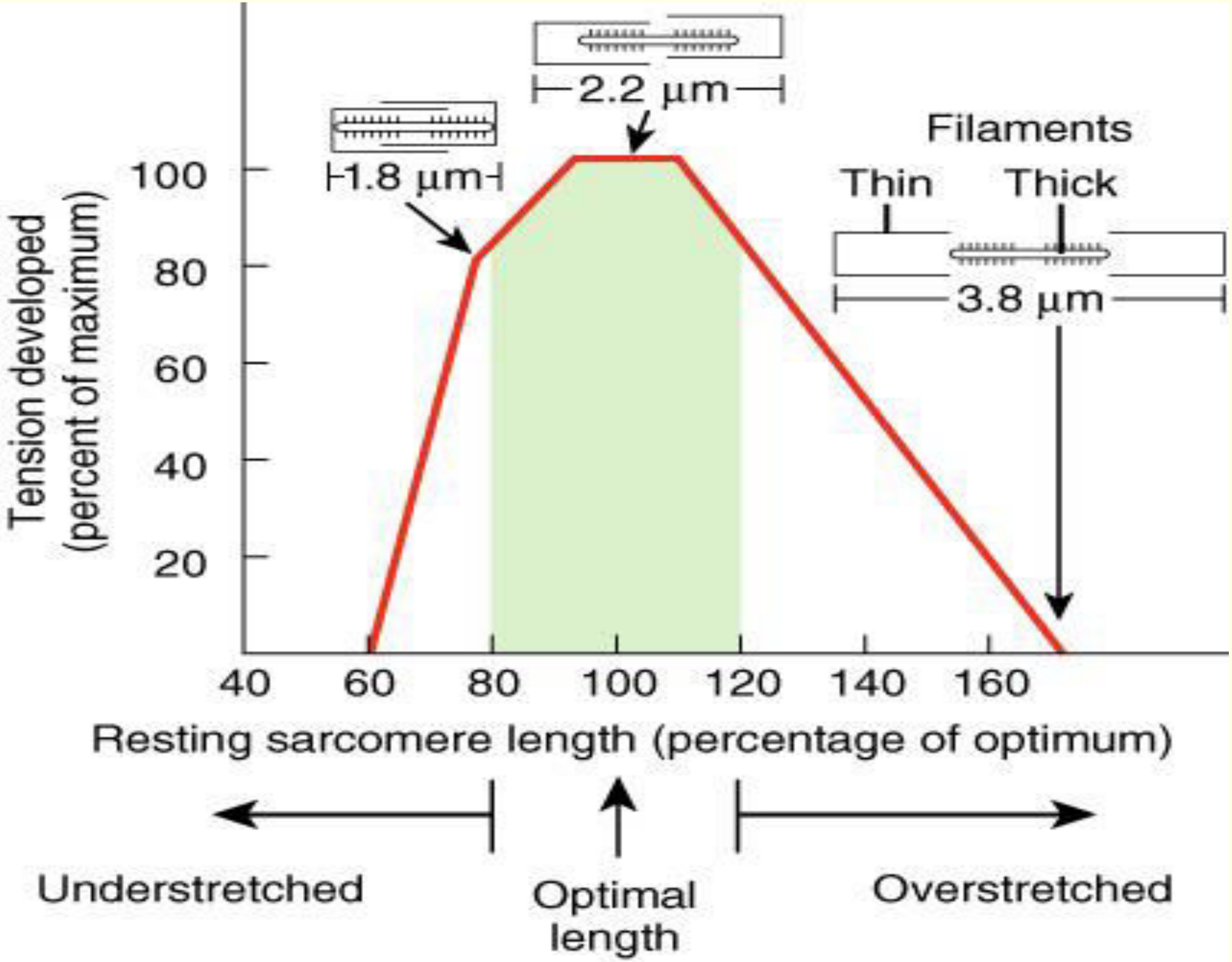
10.13

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

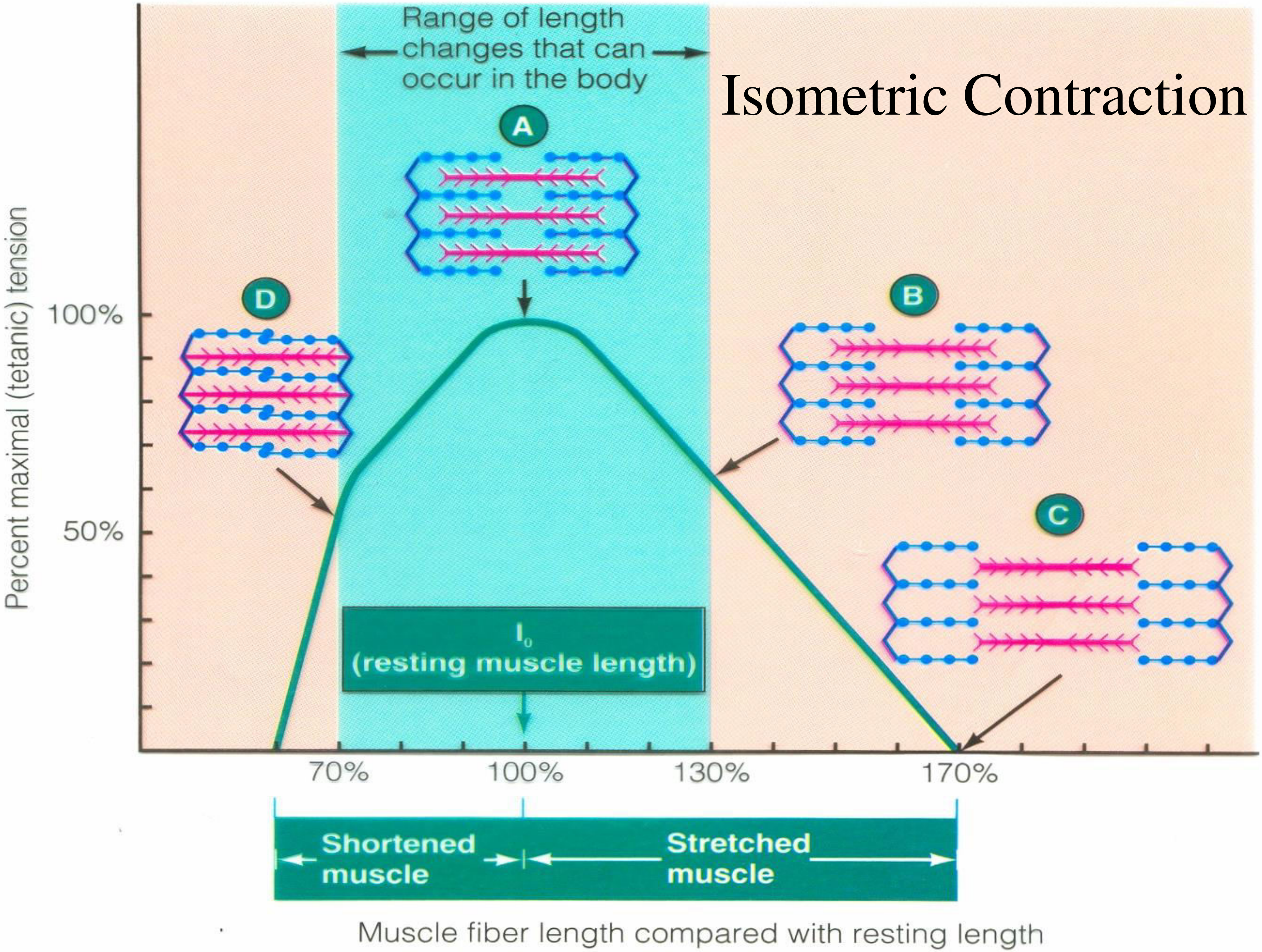


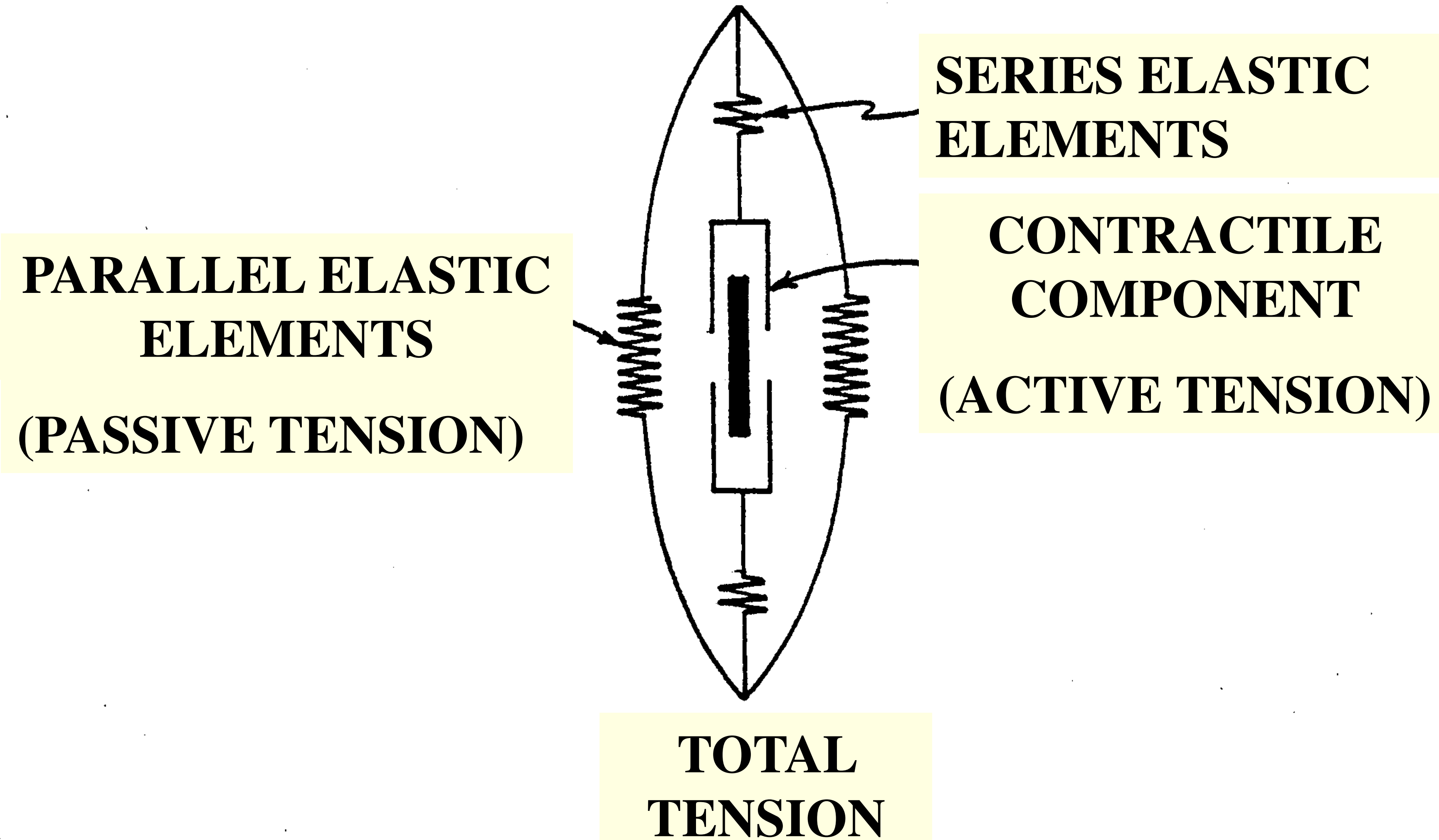

TENSION (% of maximum)
300
200
100
SKELETAL MUSCLE
HEART MUSCLE
Total tension
Resting tension
Active tension
Total tension
Resting tension
Active tension
-35%
ℓmax
+35%
-35%
ℓmax
+35%
A
B


2.2 μm
Filaments
Thin
Thick
1.8 μm
3.8 μm
Tension developed (percent of maximum)
100
80
60
40
20
40
60
80
100
120
140
160
Resting sarcomere length (percentage of optimum)
Understretched
Optimal length
Overstretched

10.10

Isometric Contraction

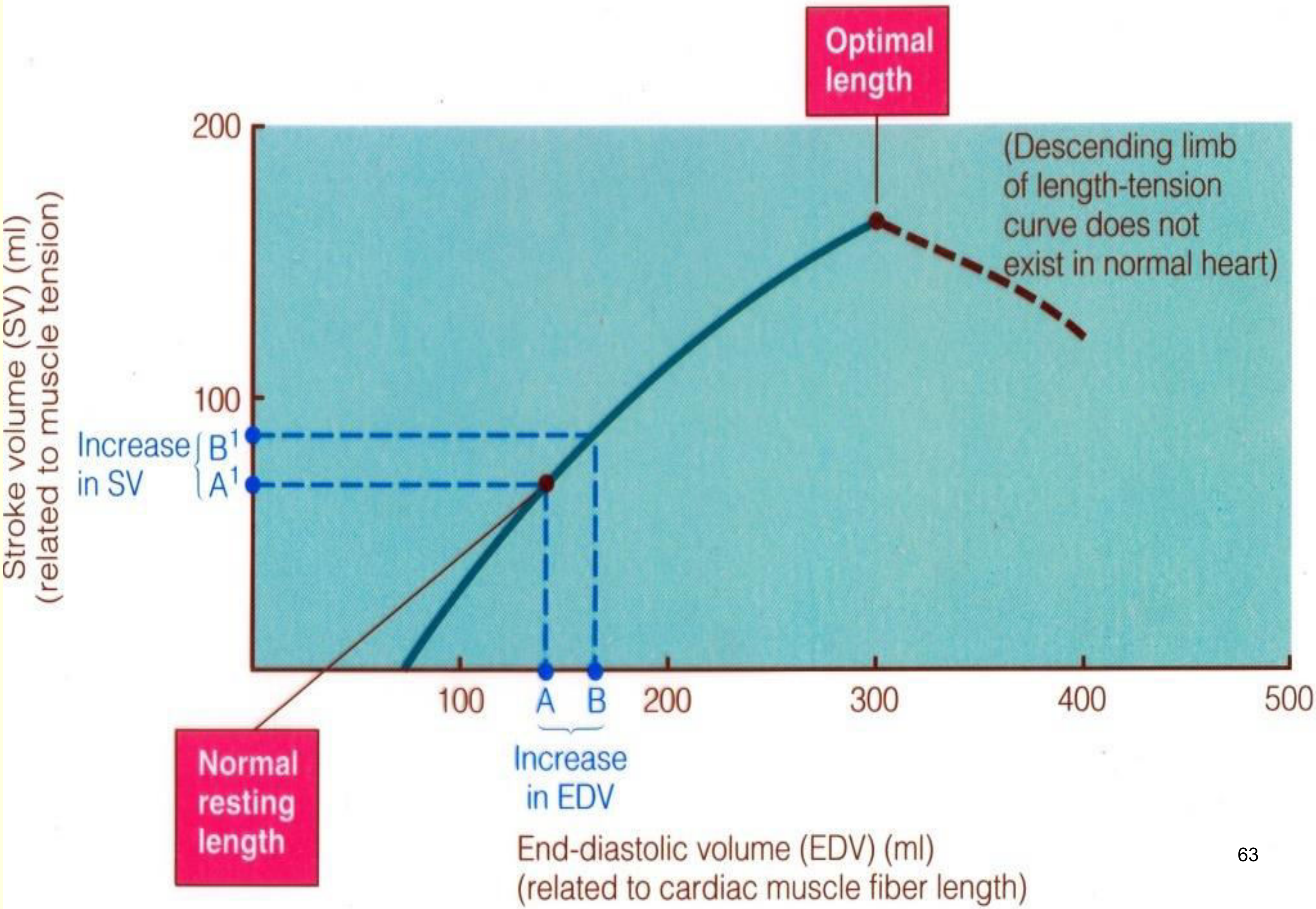



SERIES ELASTIC ELEMENTS
CONTRACTILE COMPONENT
(ACTIVE TENSION)
PARALLEL ELASTIC ELEMENTS
(PASSIVE TENSION)
TOTAL TENSION

Cardiac Muscle length-tension relationship

- 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