HEART

Table 14-1

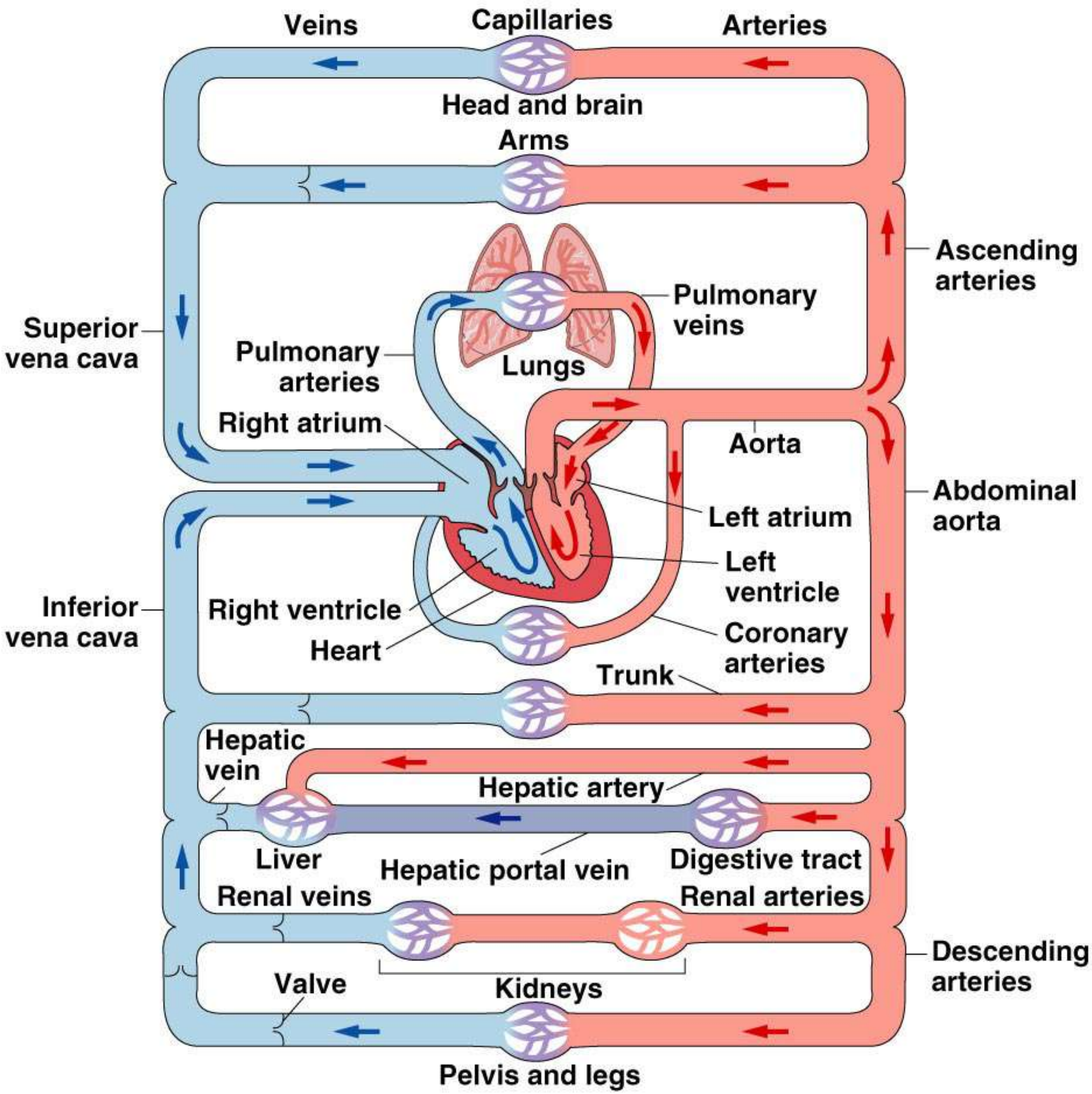
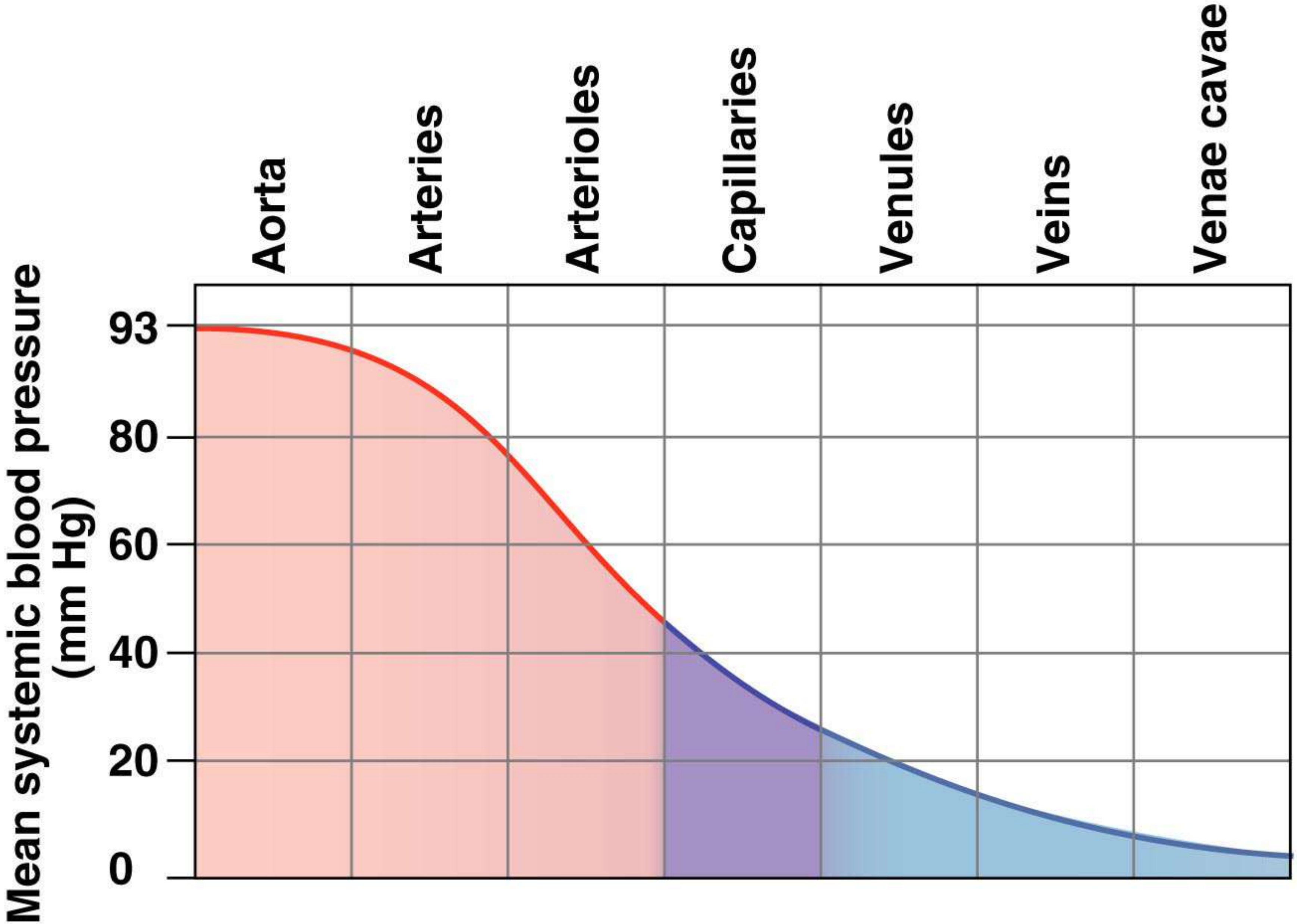

Veins
Capillaries
Arteries
Head and brain
Arms
Ascending arteries
Superior vena cava
Pulmonary arteries
Lungs
Pulmonary veins
Right atrium
Aorta
Abdominal aorta
Left atrium
Left ventricle
Inferior vena cava
Right ventricle
Heart
Coronary arteries
Trunk
Hepatic vein
Hepatic artery
Liver
Hepatic portal vein
Digestive tract
Renal veins
Renal arteries
Descending arteries
Valve
Kidneys
Pelvis and legs

Figure 14-1



Aorta
Arteries
Arterioles
Capillaries
Venules
Veins
Venae cavae
Mean systemic blood pressure (mm Hg)
93
80
60
40
20
0

Figure 14-2

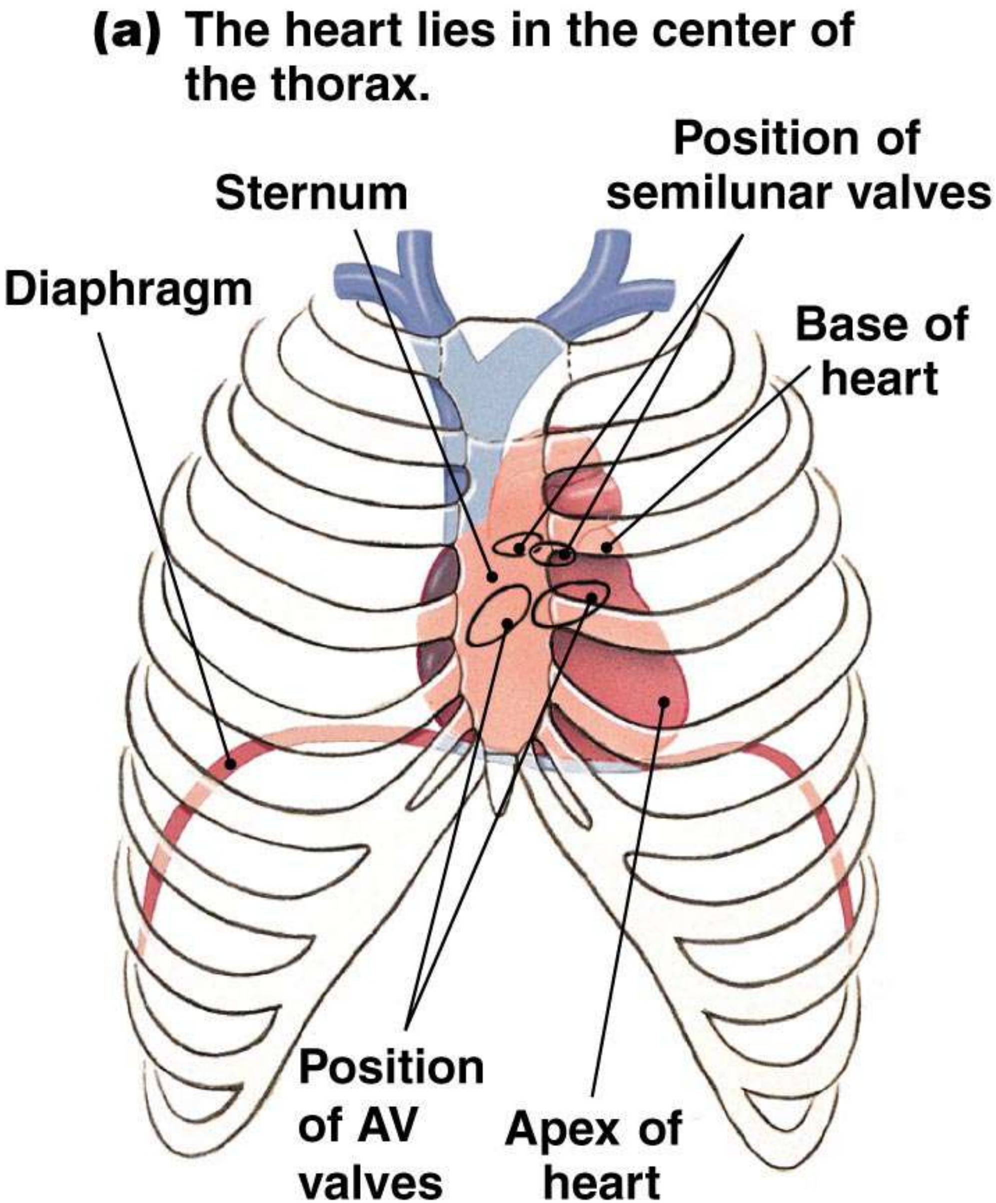
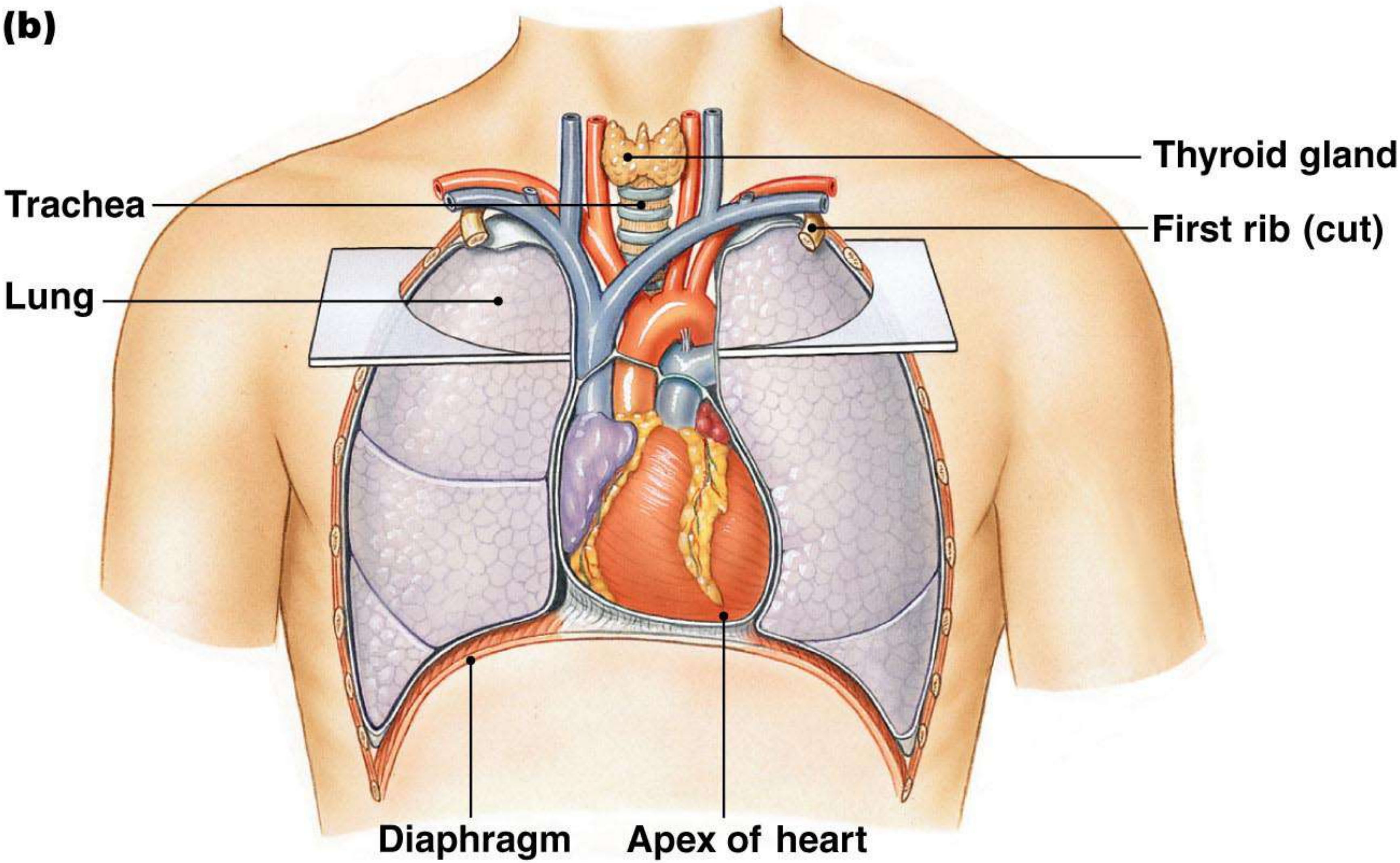

(a) The heart lies in the center of the thorax.
Position of semilunar valves
Sternum
Diaphragm
Base of heart
Position of AV valves
Apex of heart

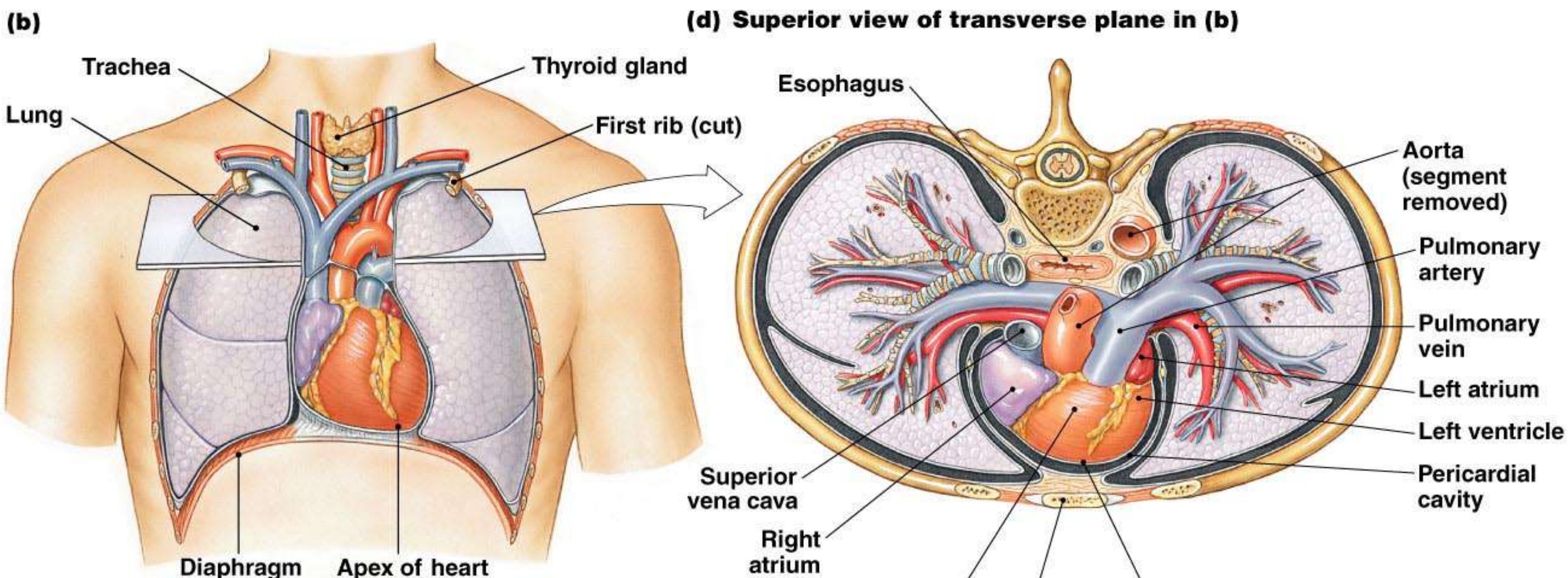
Figure 14-7a

(b)



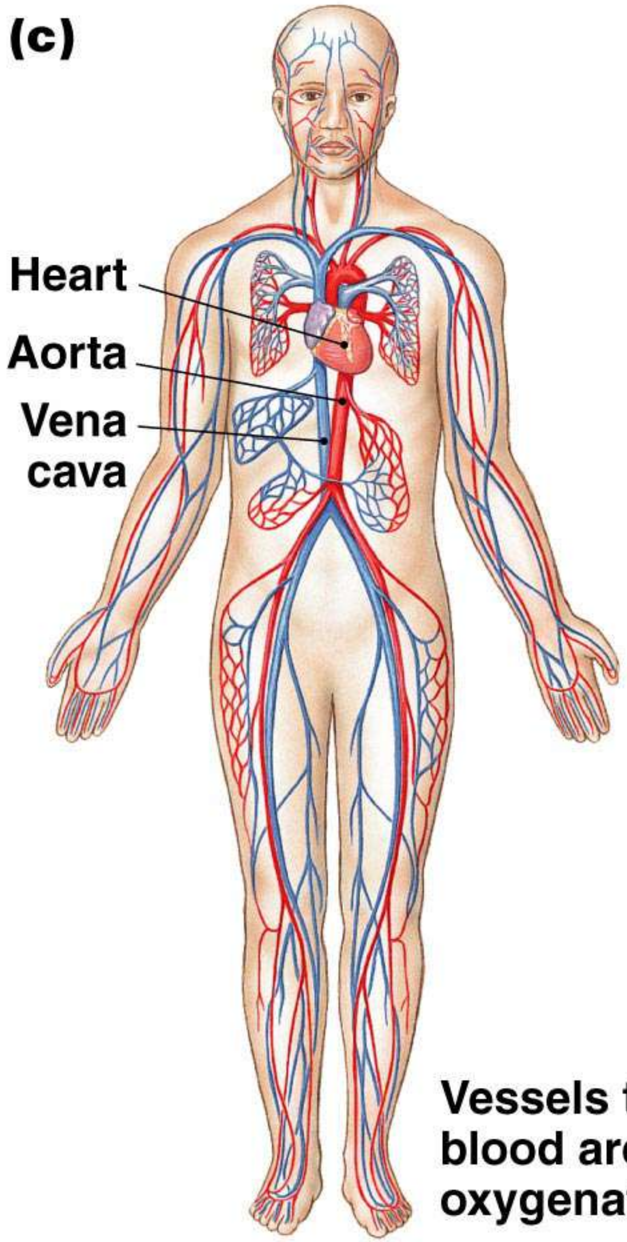
The heart is on the ventral side of the thoracic cavity, sandwiched between the lungs.

Figure 14-7b



The heart is on the ventral side of the thoracic cavity, sandwiched between the lungs.

Figure 14-7b,d



Vessels that carry well-oxygenated blood are red; those with less well-oxygenated blood are blue.

Figure 14-7c

(d) Superior view of transverse plane in (b)

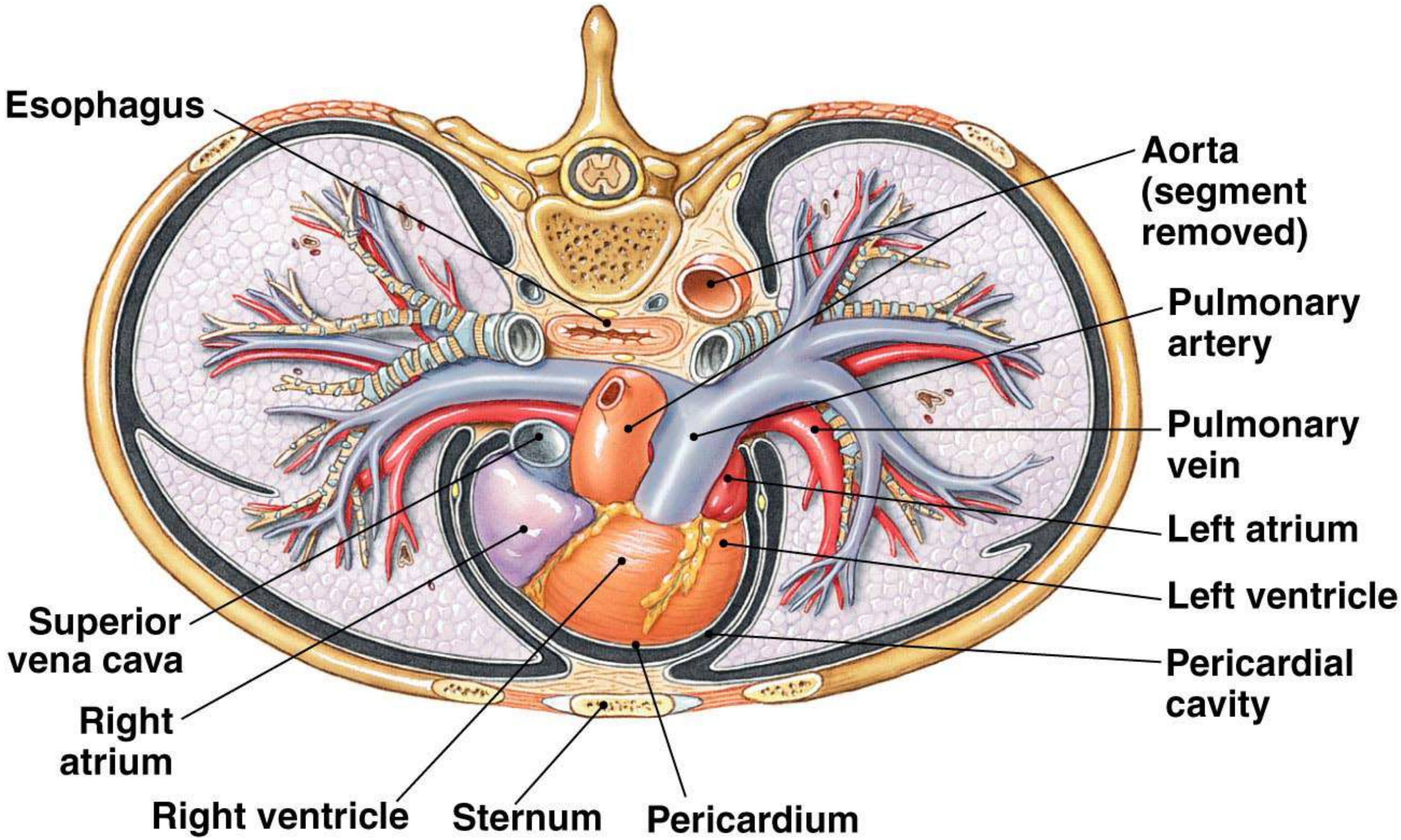
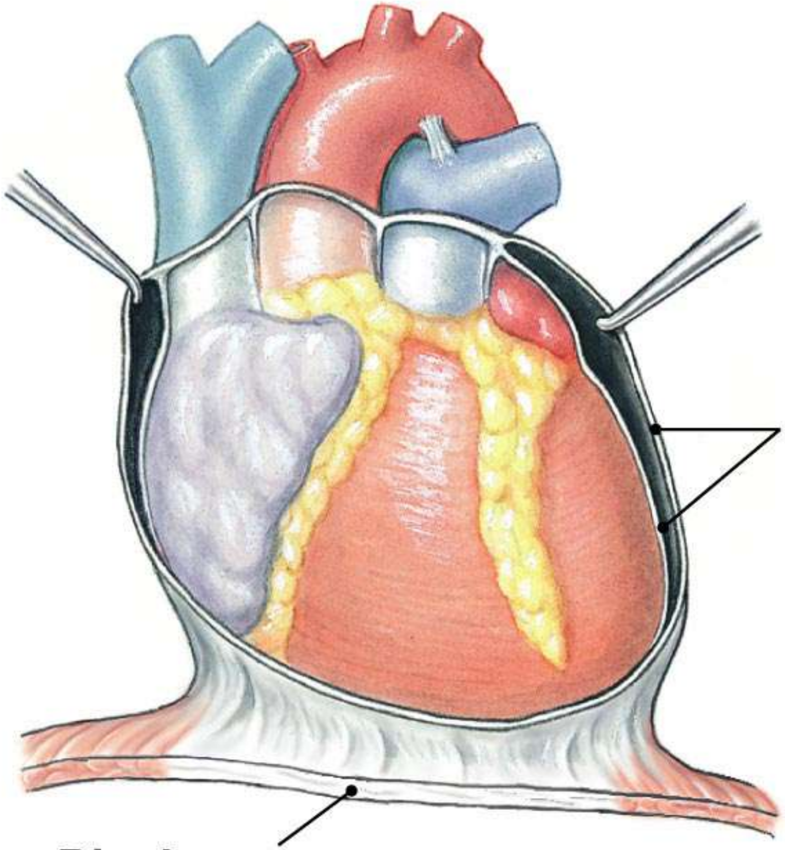


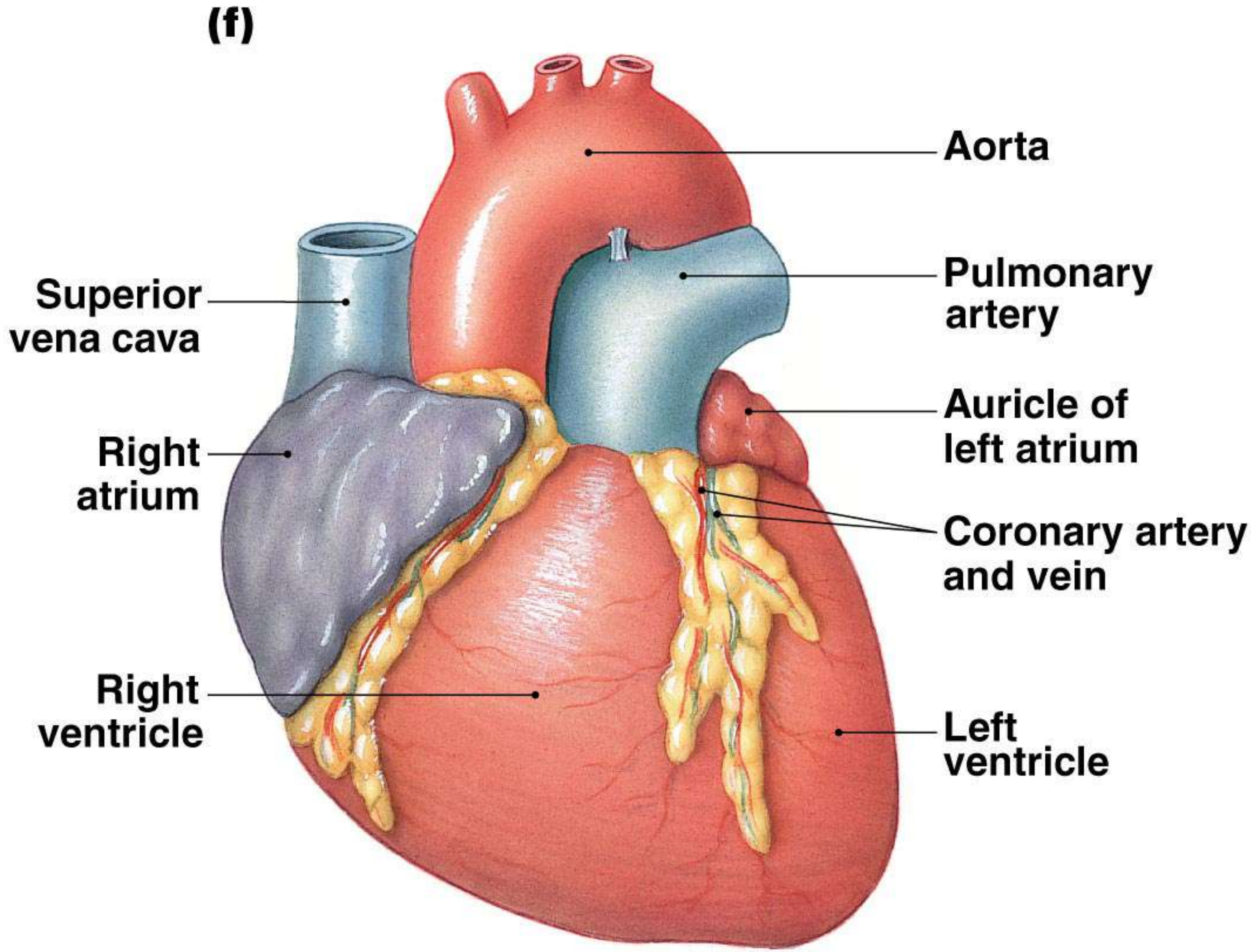
Figure 14-7d

(e)



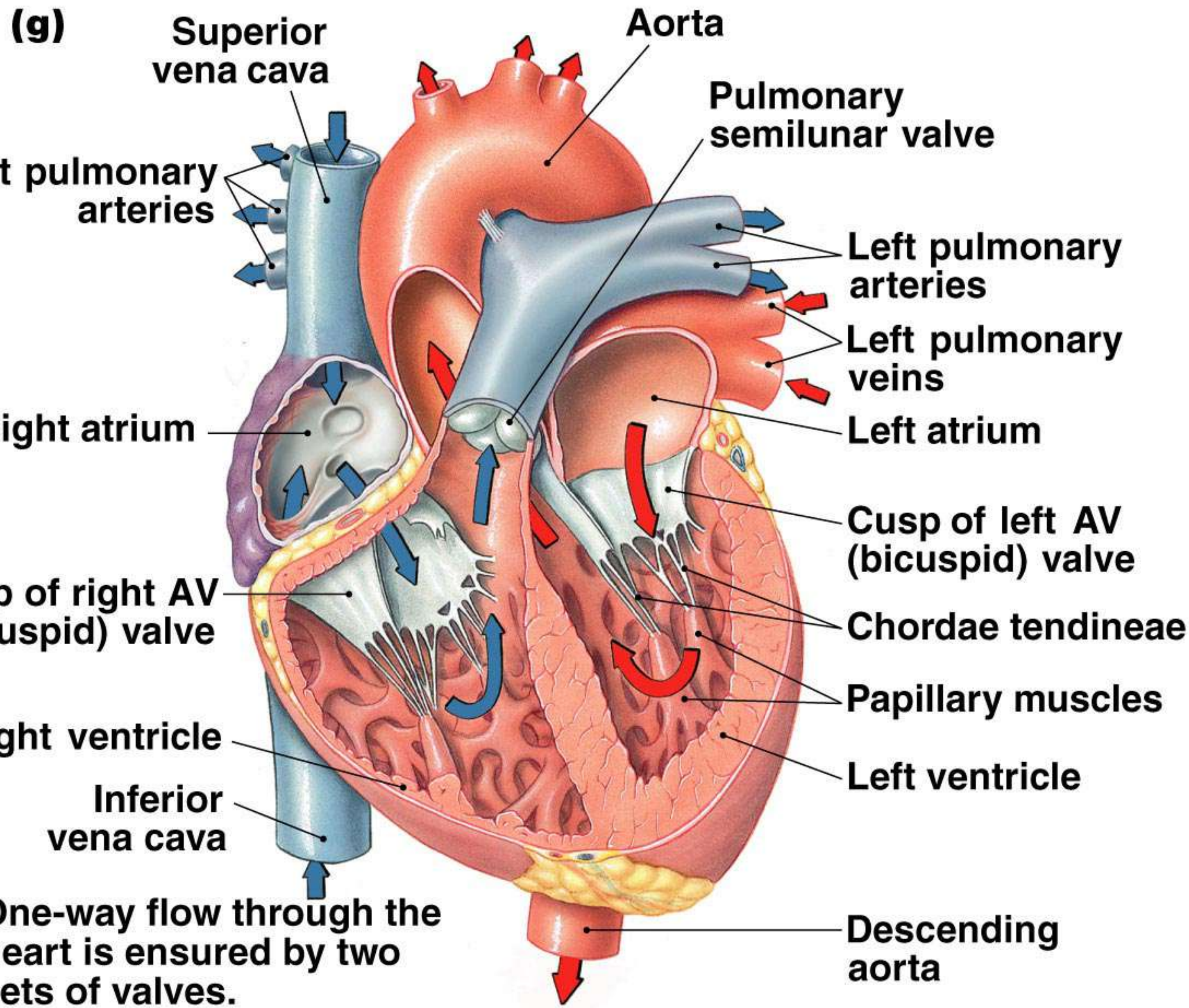
The heart is encased within a membranous fluid-filled sac, the pericardium.

Figure 14-7e



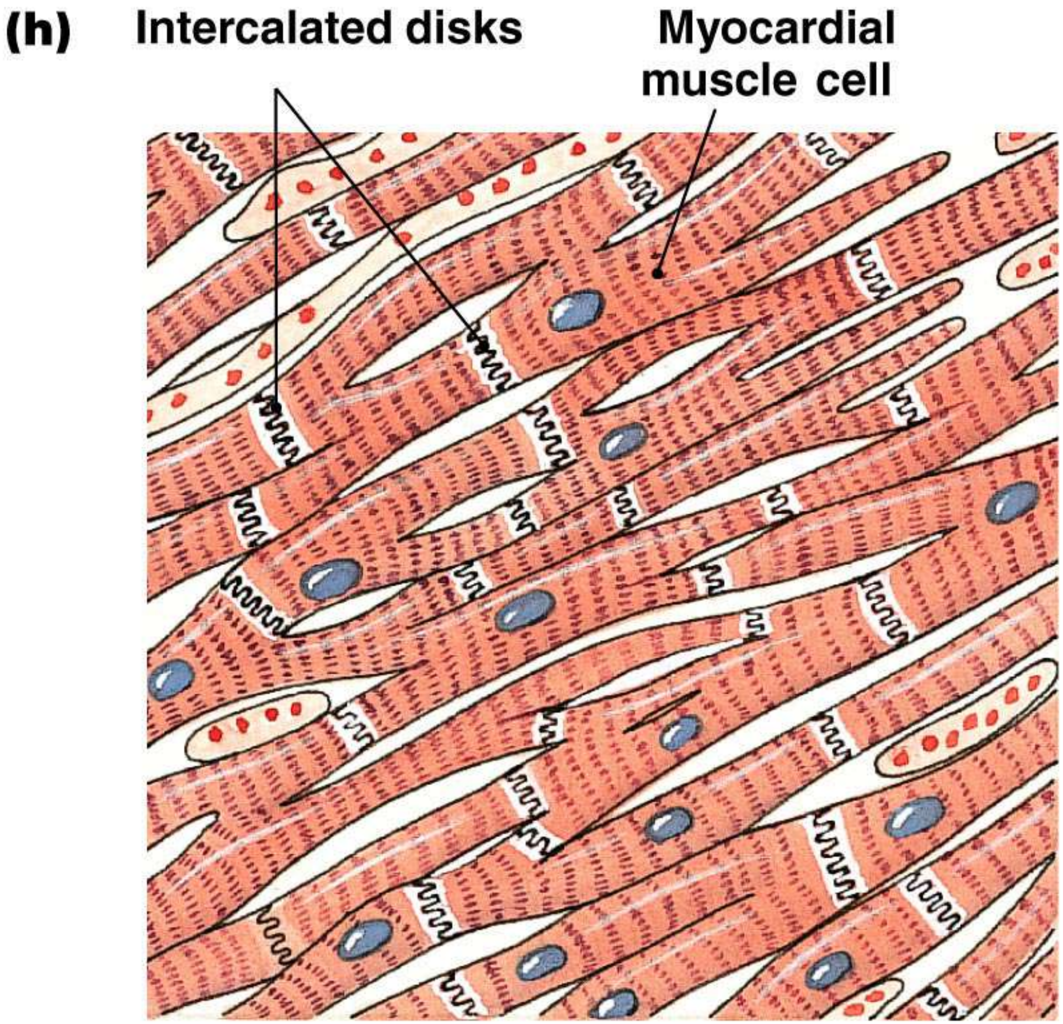
The ventricles occupy the bulk of the heart. The arteries and veins all attach to the base of the heart.

Figure 14-7f



One-way flow through the heart is ensured by two sets of valves.

Figure 14-7g



Myocardial muscle cells are branched, have a single nucleus, and are attached to each other by specialized junctions known as intercalated disks.

Figure 14-7h

TABLE 14-2 The Heart and Major Blood Vessels

Blue type indicates structures containing blood with lower oxygen content; red type indicates well-oxygenated blood.

	RECEIVES BLOOD FROM	SENDS BLOOD TO
Heart		
Right atrium	Venae cavae	Right ventricle
Right ventricle	Right atrium	Lungs
Left atrium	Pulmonary veins	Left ventricle
Left ventricle	Left atrium	Body except for lungs
Vessels		
Venae cavae	Systemic veins	Right atrium
Pulmonary trunk (artery)	Right ventricle	Lungs
Pulmonary vein	Veins of the lungs	Left atrium
Aorta	Left ventricle	Systemic arteries

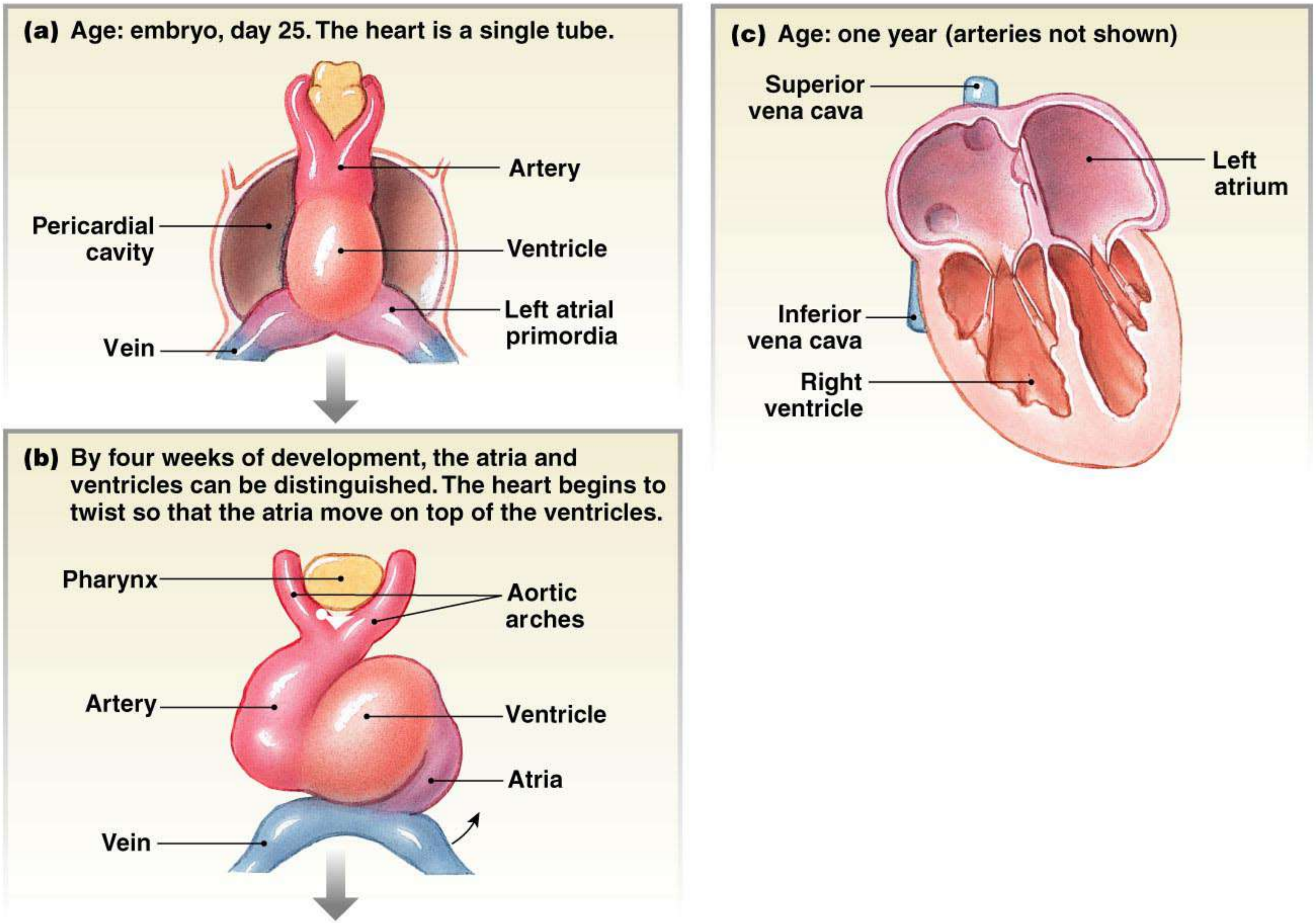


Figure 14-8 - Overview

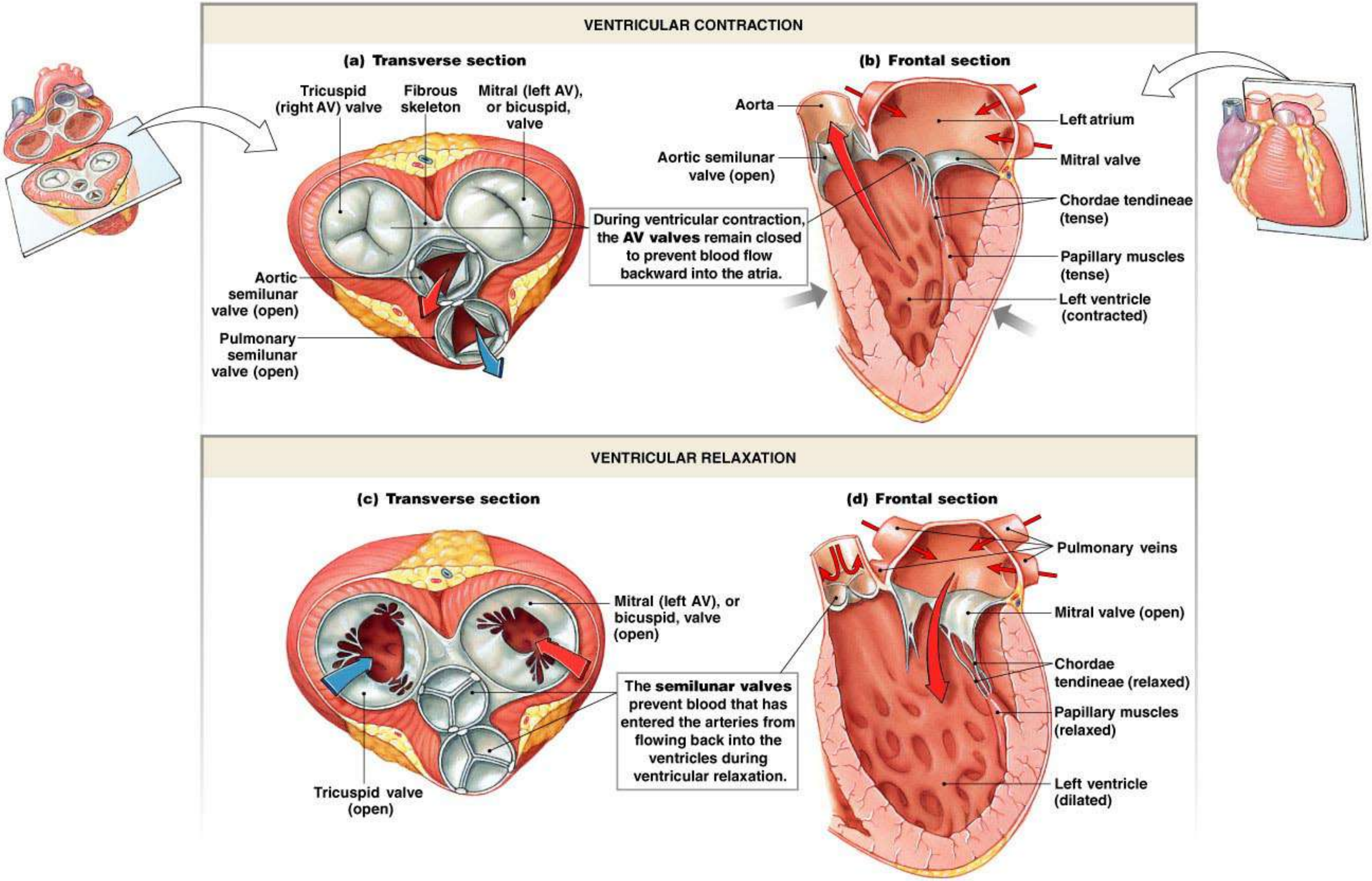


Figure 14-9 - Overview

(a) The spiral arrangement of ventricular muscle allows ventricular contraction to squeeze the blood upward from the apex of the heart.

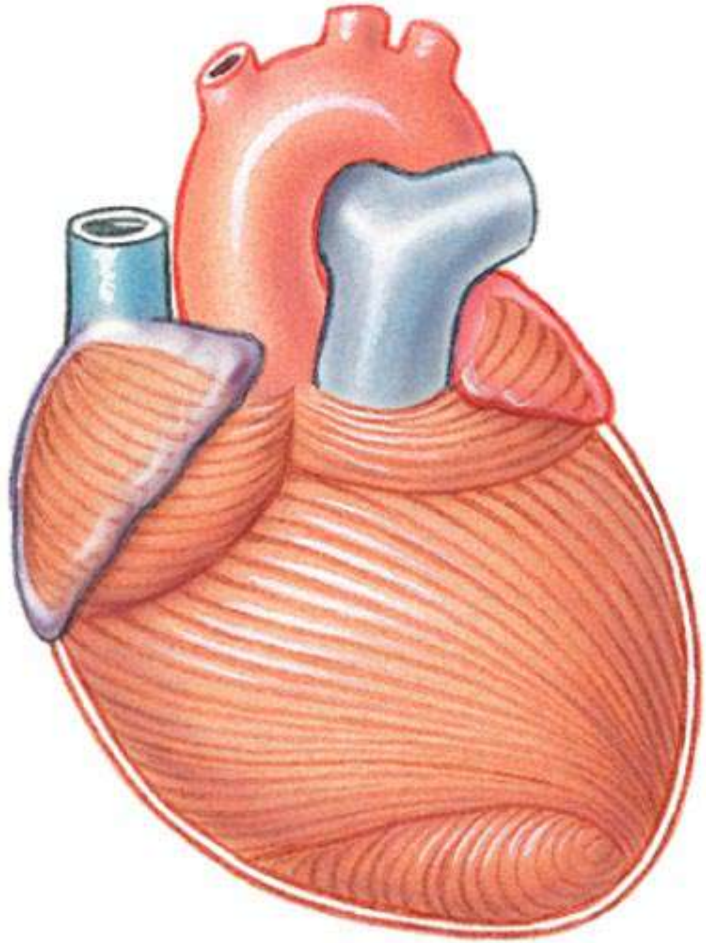


Figure 14-10a

(b) Intercalated disks contain desmosomes that transfer force from cell to cell and gap junctions that allow electrical signals to pass rapidly from cell to cell.

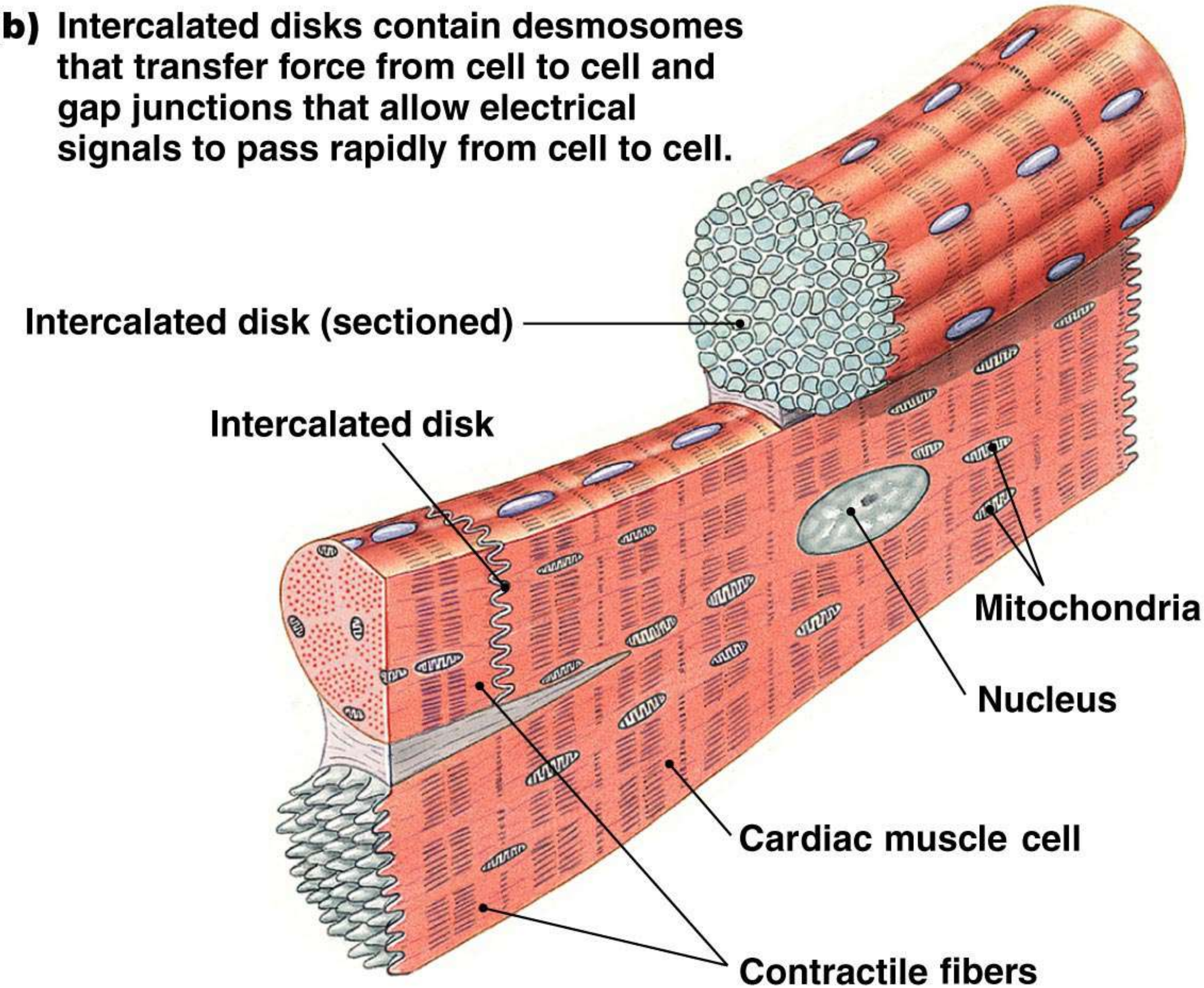
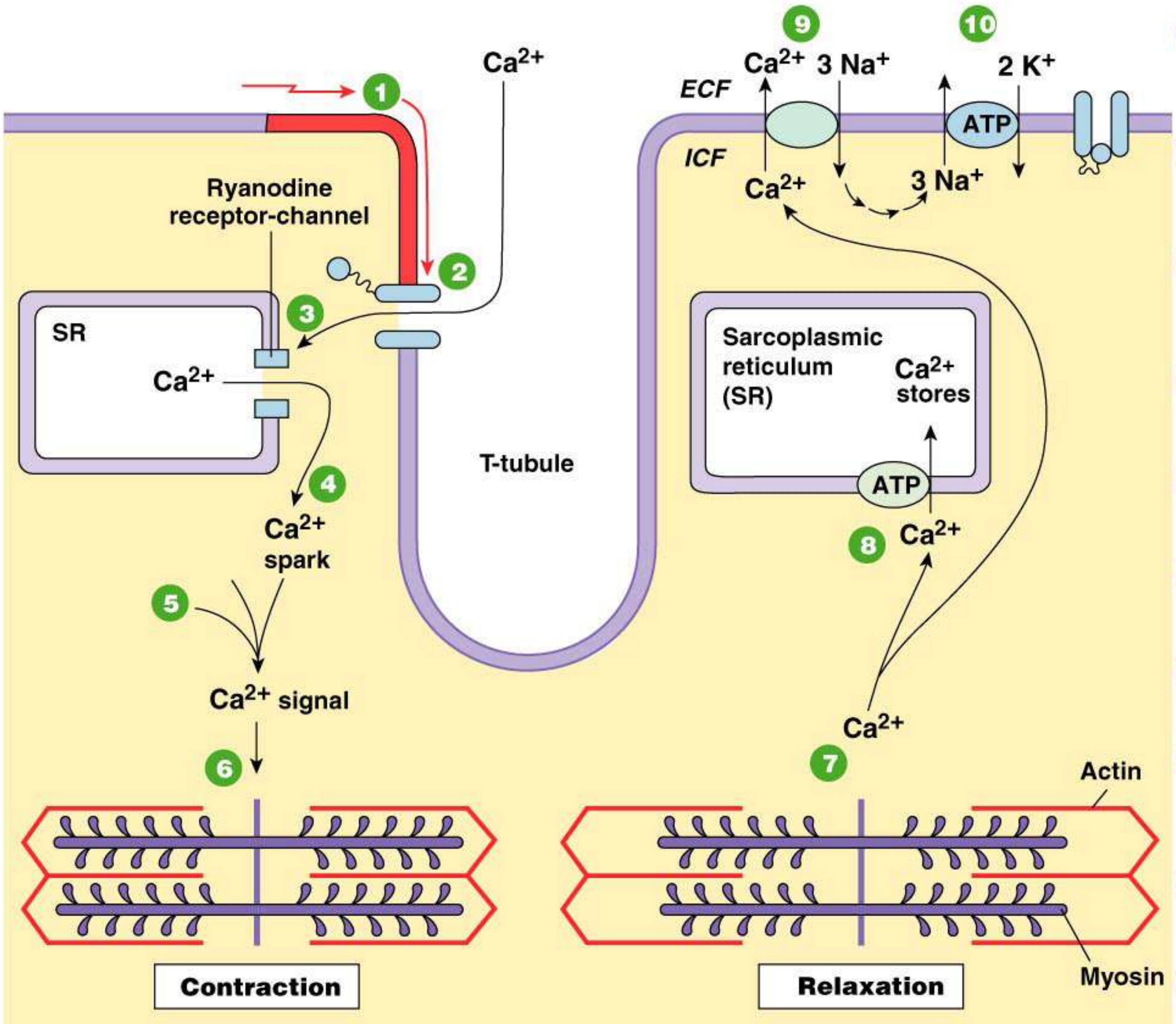
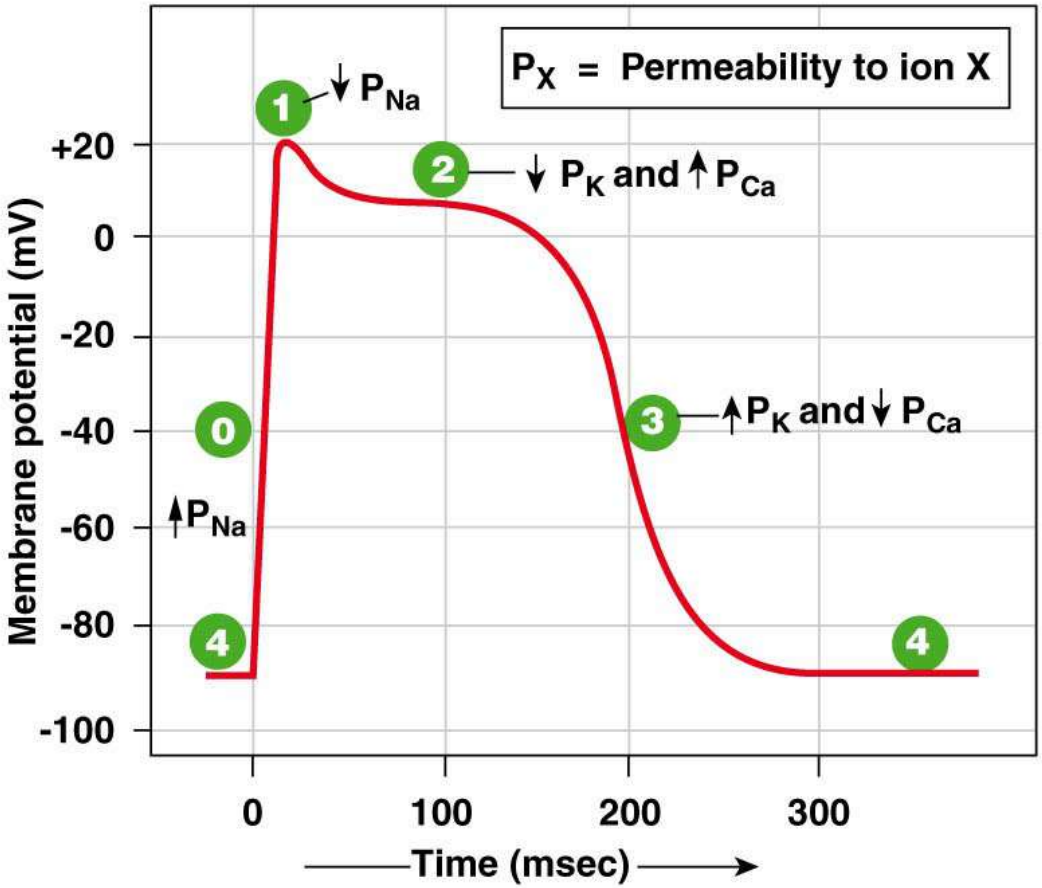


Figure 14-10b



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

Figure 14-11 - Overview



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

Figure 14-13

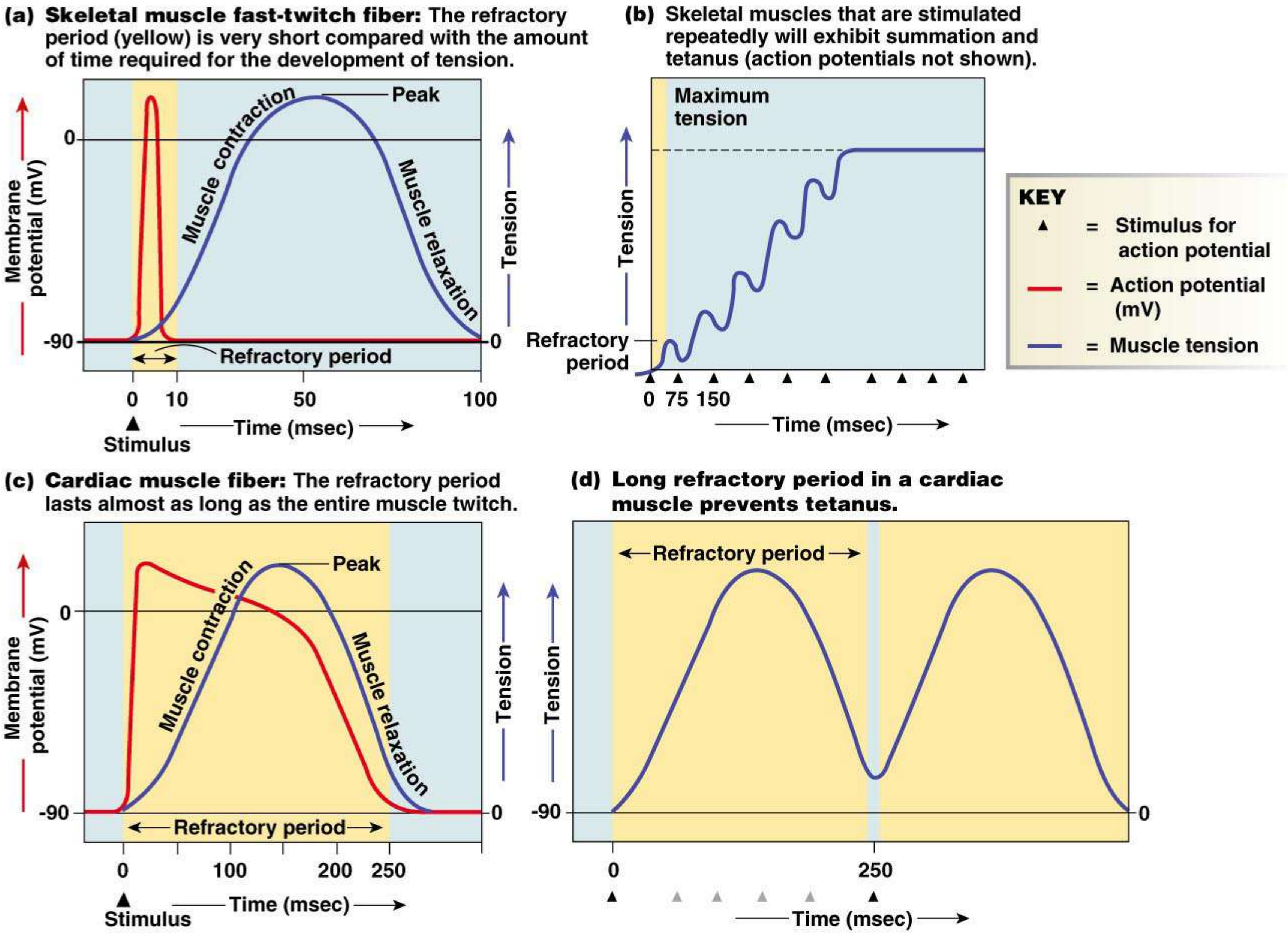


Figure 14-14 - Overview

(a) The pacemaker potential gradually becomes less negative until it reaches threshold, triggering an action potential.

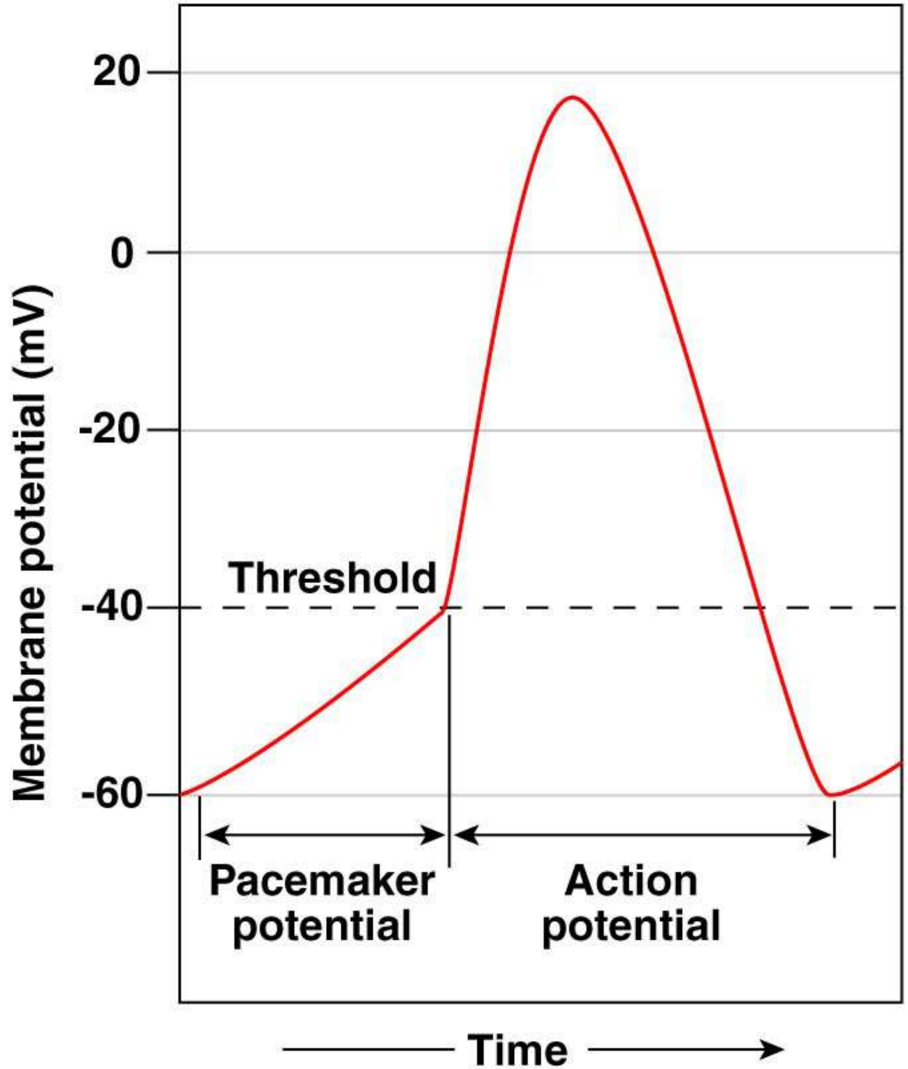


Figure 14-15a

(b) Ion movements during an action and pacemaker potential

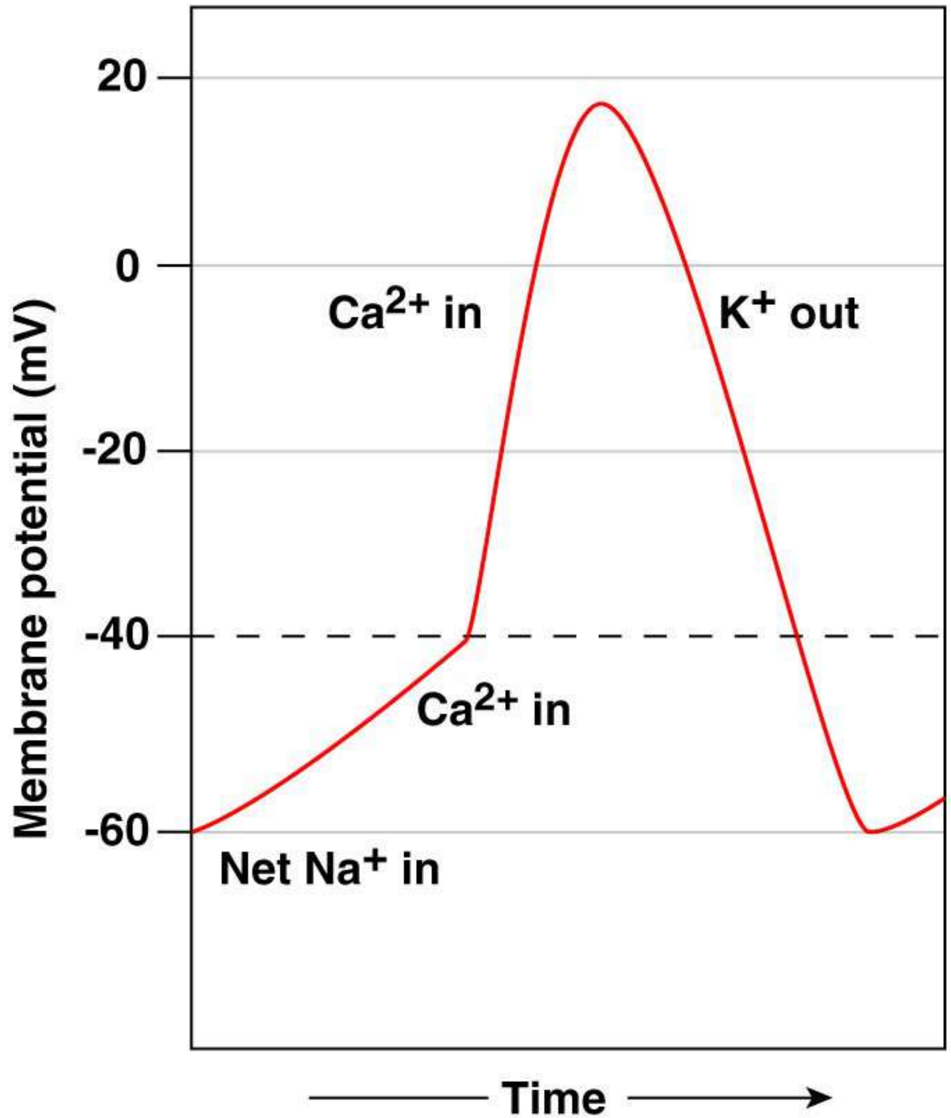


Figure 14-15b

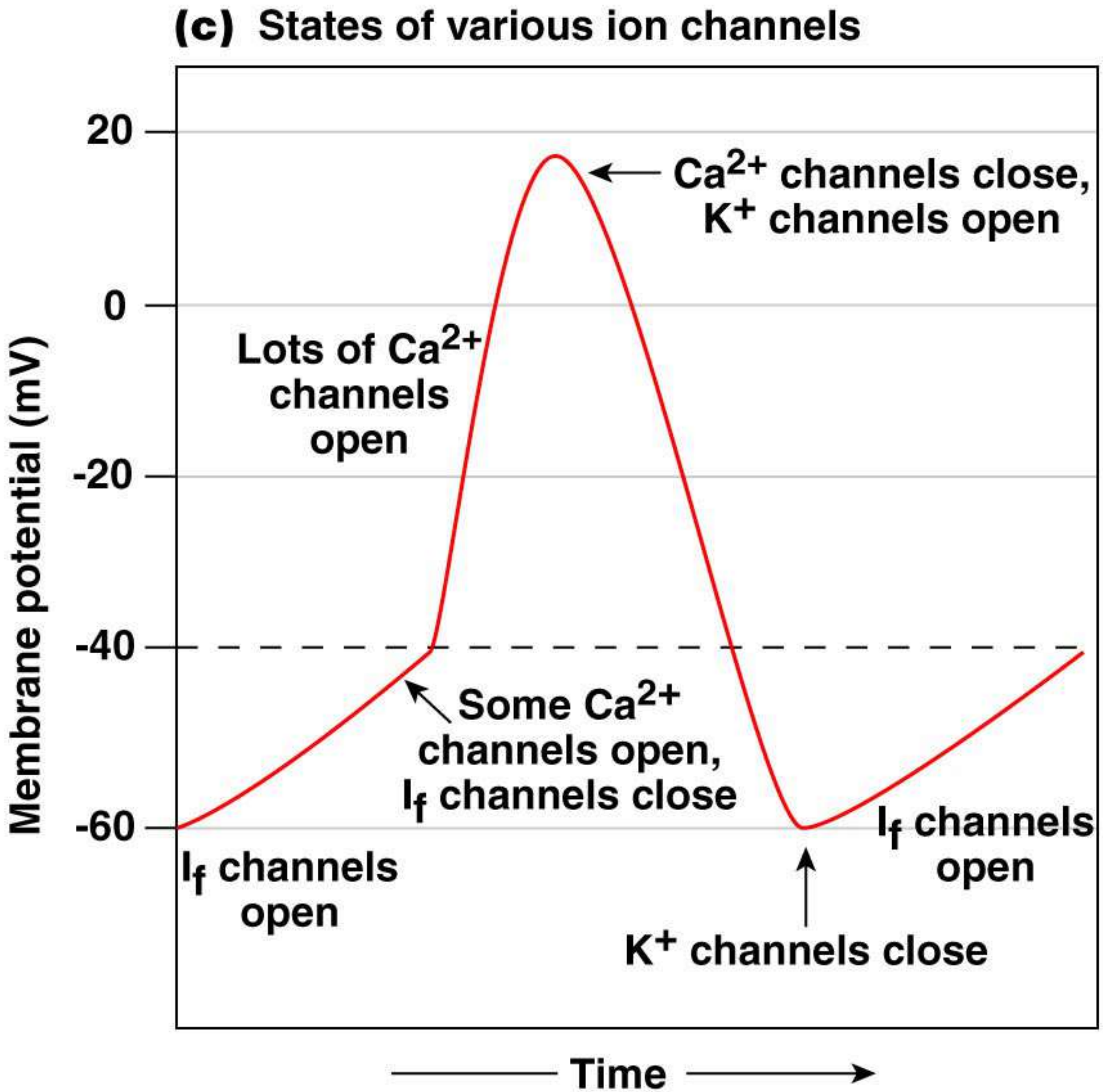


Figure 14-15c

(a) Sympathetic stimulation and epinephrine depolarize the autorhythmic cell and speed up the depolarization rate, increasing the heart rate.

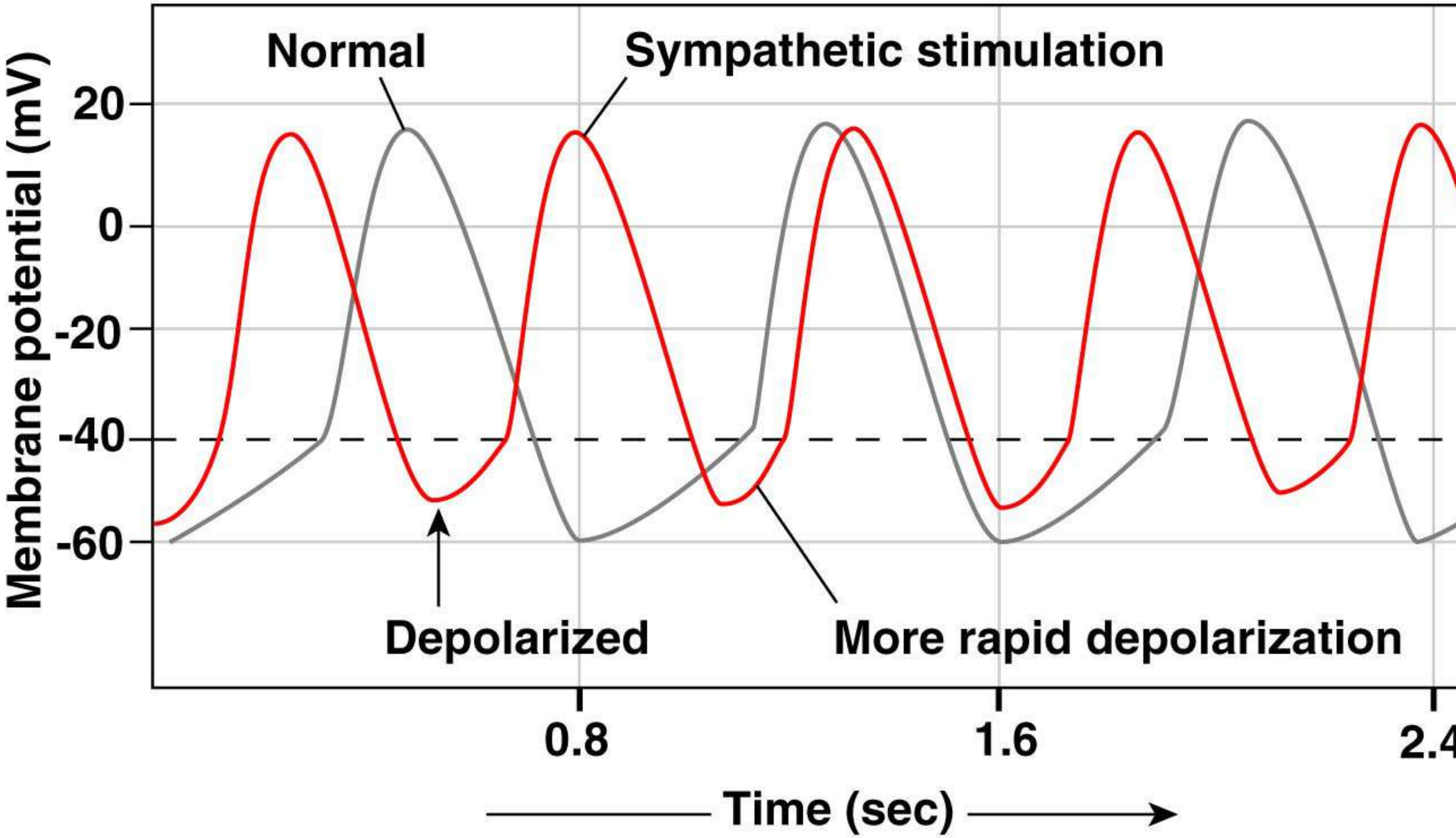


Figure 14-16a

(b) Parasympathetic stimulation hyperpolarizes the membrane potential of the autorhythmic cell and slows depolarization, decreasing the heart rate.

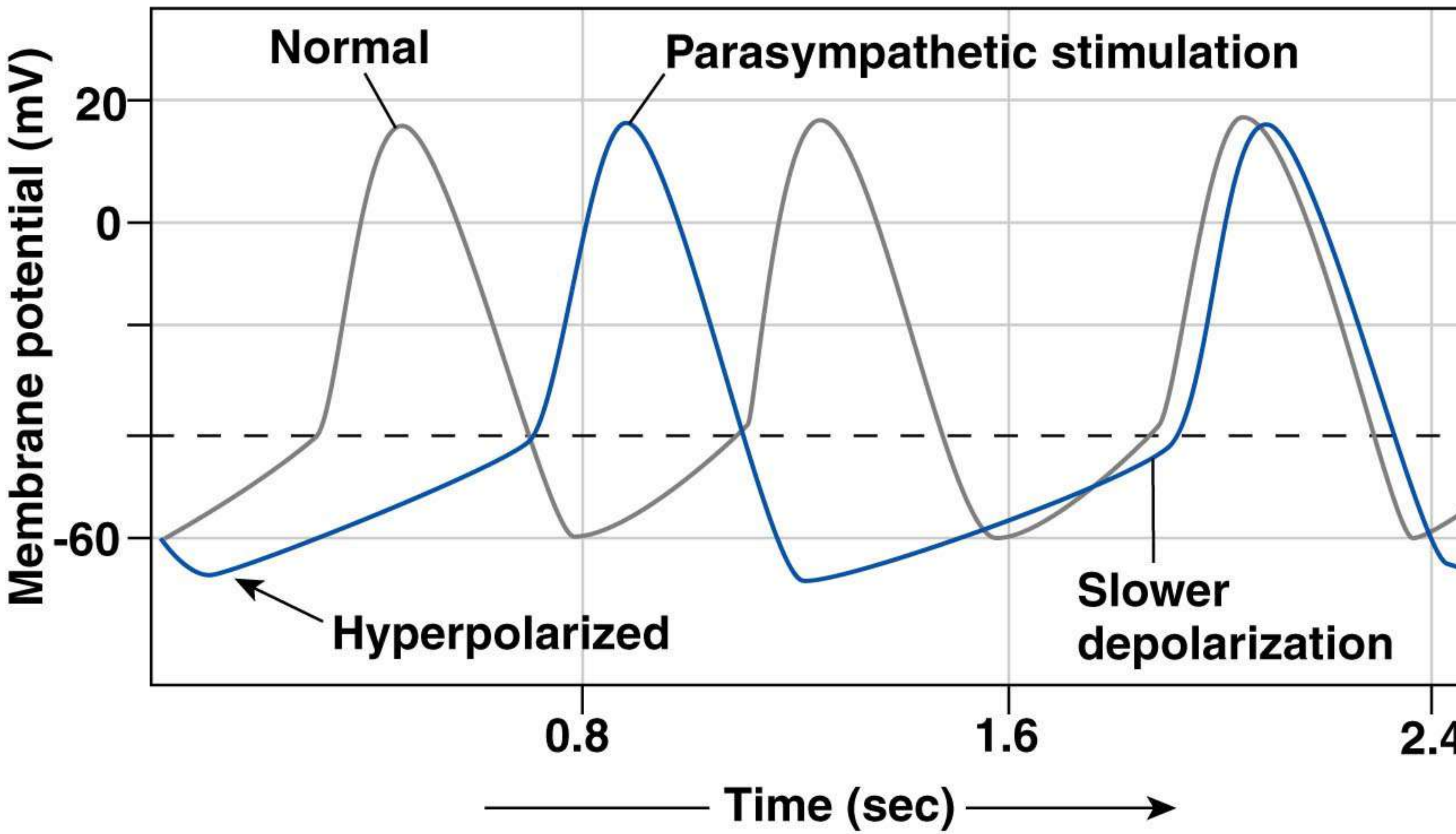
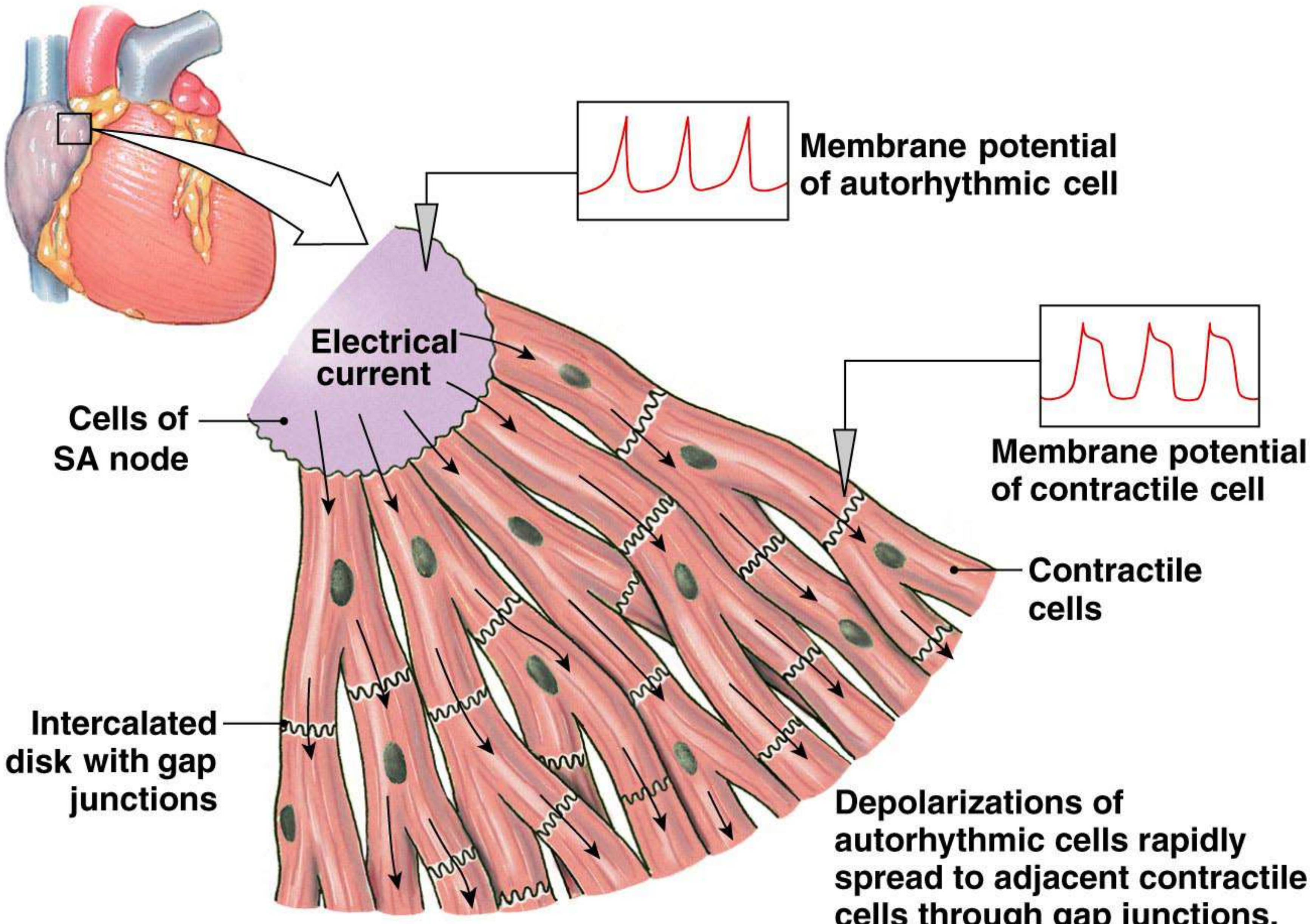


Figure 14-16b

TABLE 14-3 Comparison of Action Potentials in Cardiac and Skeletal Muscle

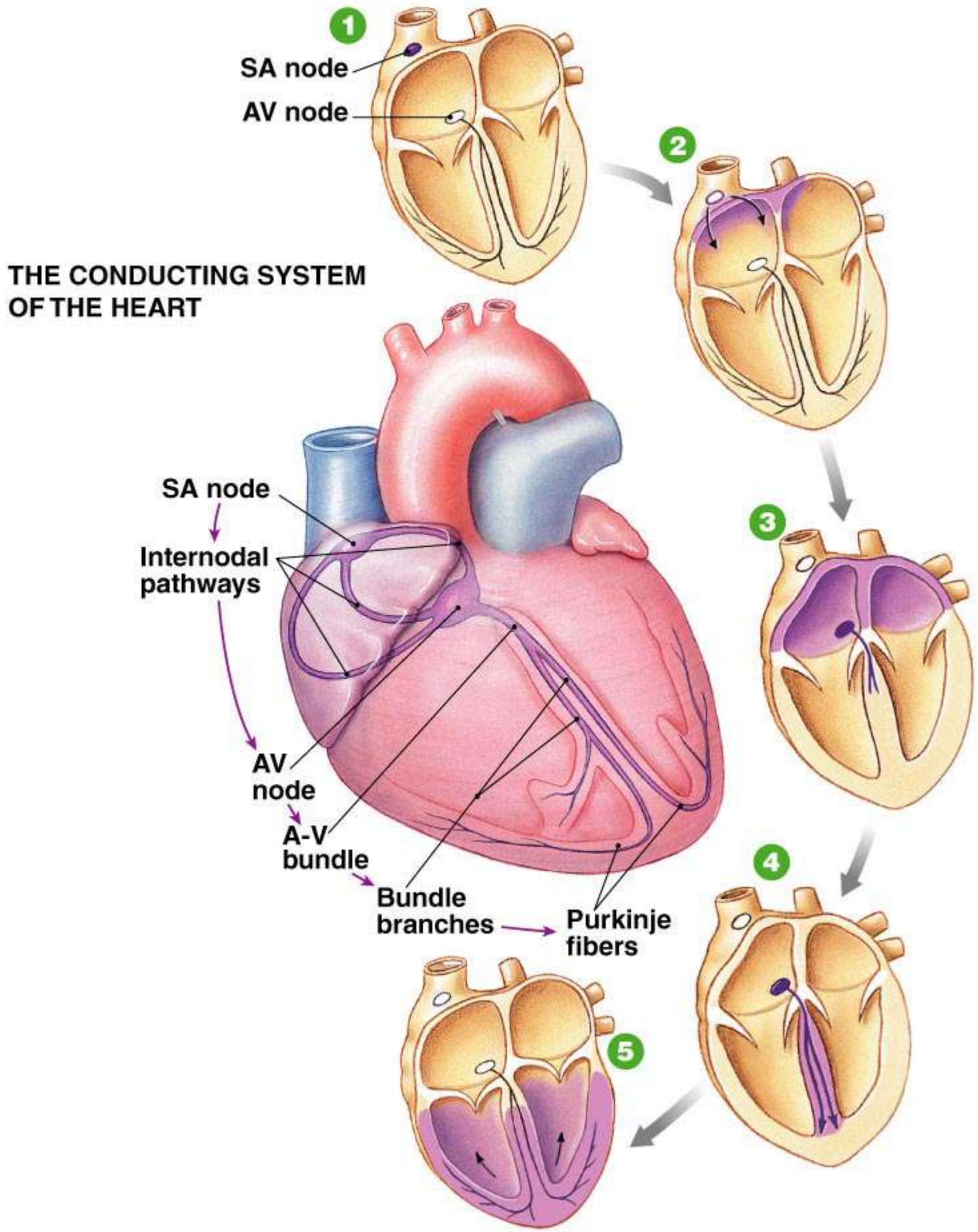
	SKELETAL MUSCLE	CONTRACTILE MYOCARDIUM	AUTORHYTHMIC MYOCARDIUM
Membrane potential	Stable at −70 mV	Stable at −90 mV	Unstable pacemaker potential; usually starts at −60 mV
Events leading to threshold potential	Net Na^+ entry through ACh-operated channels	Depolarization enters via gap junctions	Net Na^+ entry through I_f channels; reinforced by Ca^{2+} entry
Rising phase of action potential	Na^+ entry	Na^+ entry	Ca^{2+} entry
Repolarization phase	Rapid; caused by K^+ efflux	Extended plateau caused by Ca^{2+} entry; rapid phase caused by K^+ efflux	Rapid; caused by K^+ efflux
Hyperpolarization	Due to excessive K^+ efflux at high K^+ permeability when K^+ channels close; leak of K^+ and Na^+ restores potential to resting state	None; resting potential is −90 mV, the equilibrium potential for K^+	None; when repolarization hits −60 mV, the I_f channels open again
Duration of action potential	Short: 1–2 msec	Extended: 200+ msec	Variable; generally 150+ msec
Refractory period	Generally brief	Long because resetting of Na^+ channel gates delayed until end of action potential	None

Table 14-3



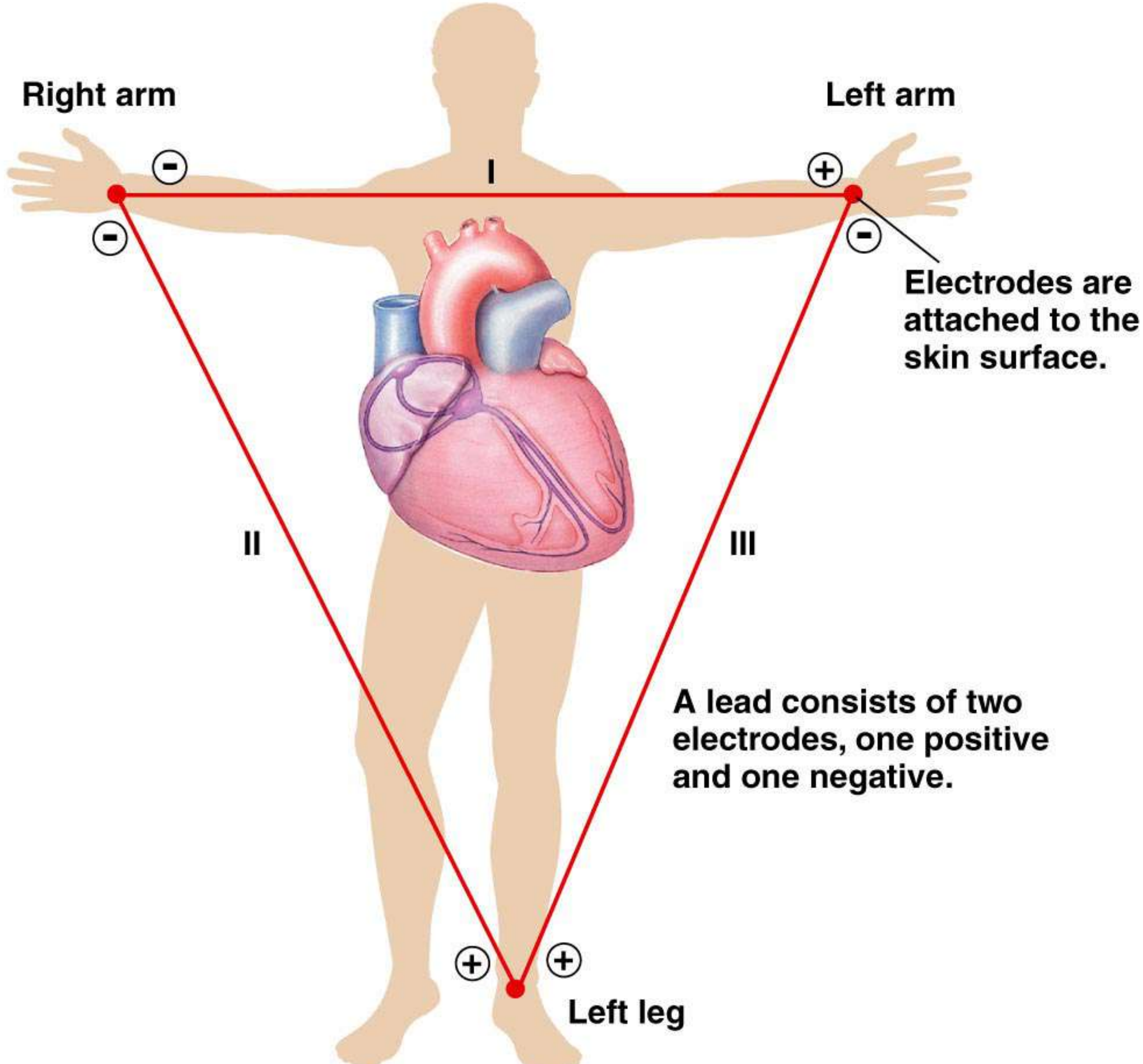
Membrane potential of autorhythmic cell
Electrical current
Cells of SA node
Membrane potential of contractile cell
Contractile cells
Intercalated disk with gap junctions
Depolarizations of autorhythmic cells rapidly spread to adjacent contractile cells through gap junctions.

Figure 14-17



1. SA node depolarizes.
2. Electrical activity goes rapidly to AV node via internodal pathways.
3. Depolarization spreads more slowly across atria. Conduction slows through AV node.
4. Depolarization moves rapidly through ventricular conducting system to the apex of the heart.
5. Depolarization wave spreads upward from the apex.

Figure 14-18



Right arm
Left arm
I
II
III
Electrodes are attached to the skin surface.
A lead consists of two electrodes, one positive and one negative.
Left leg

Figure 14-19

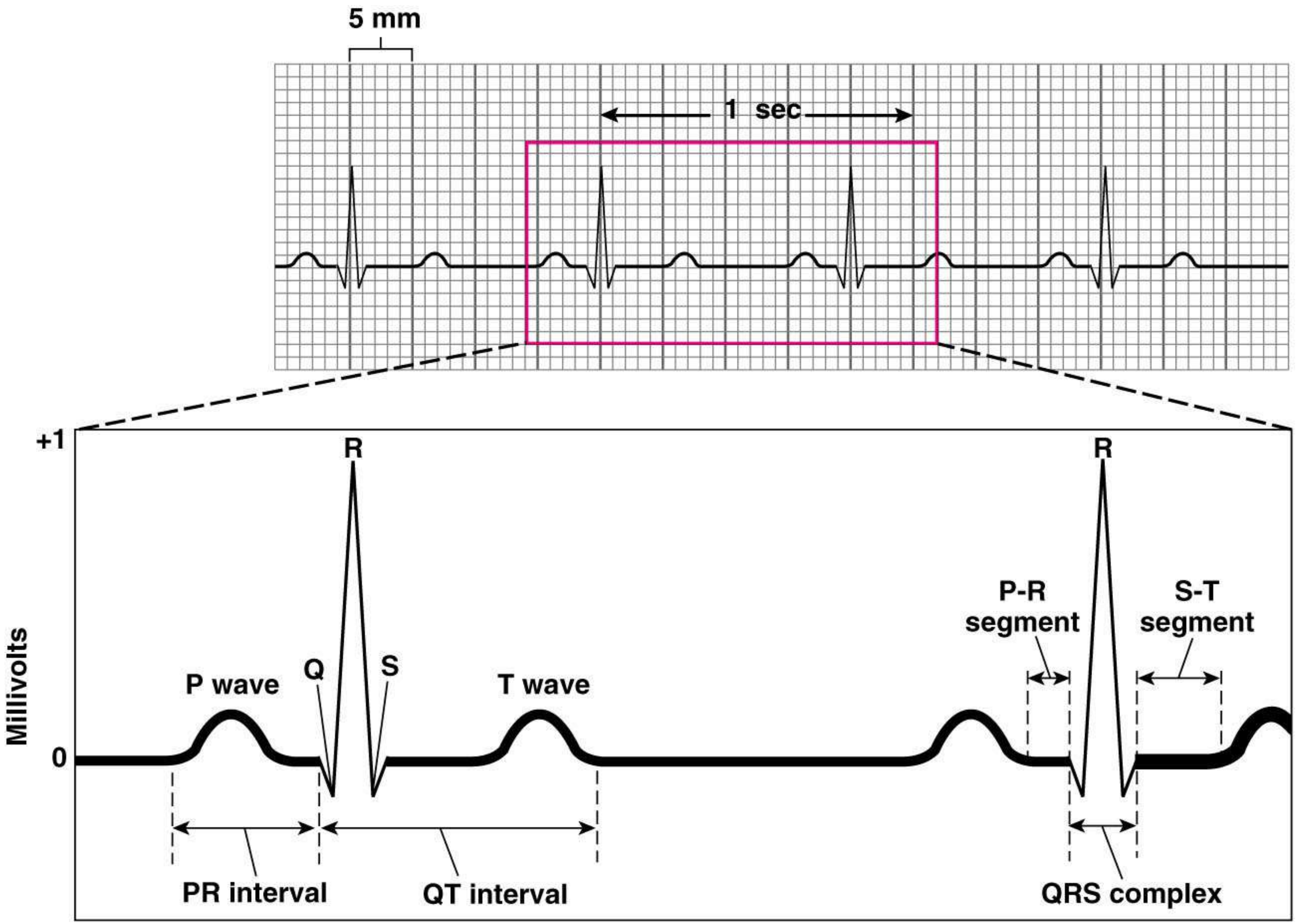

5 mm
1 sec
+1
0
Millivolts
R
R
P wave
Q
S
T wave
P-R
segment
S-T
segment
PR interval
QT interval
QRS complex

Figure 14-20

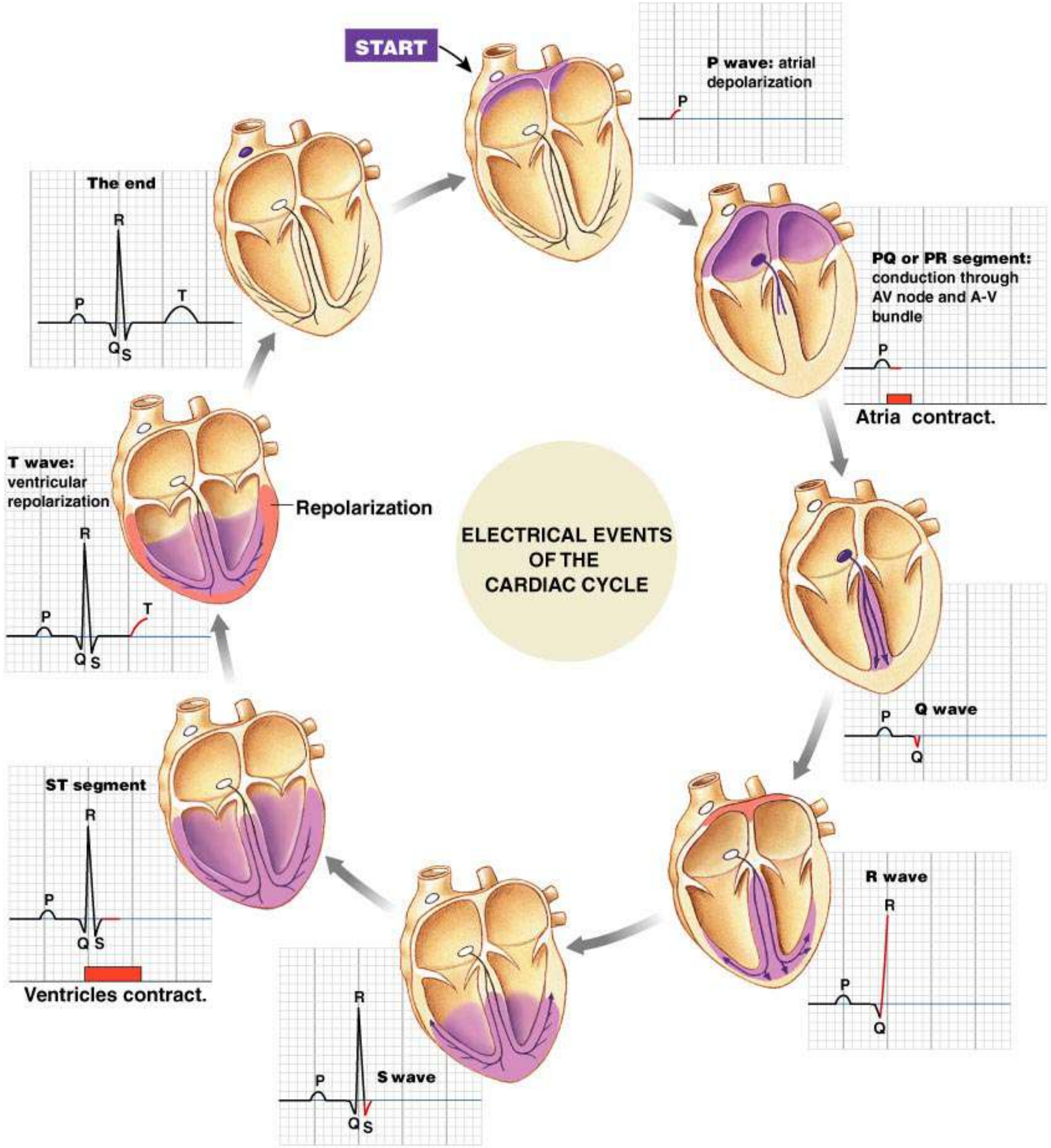
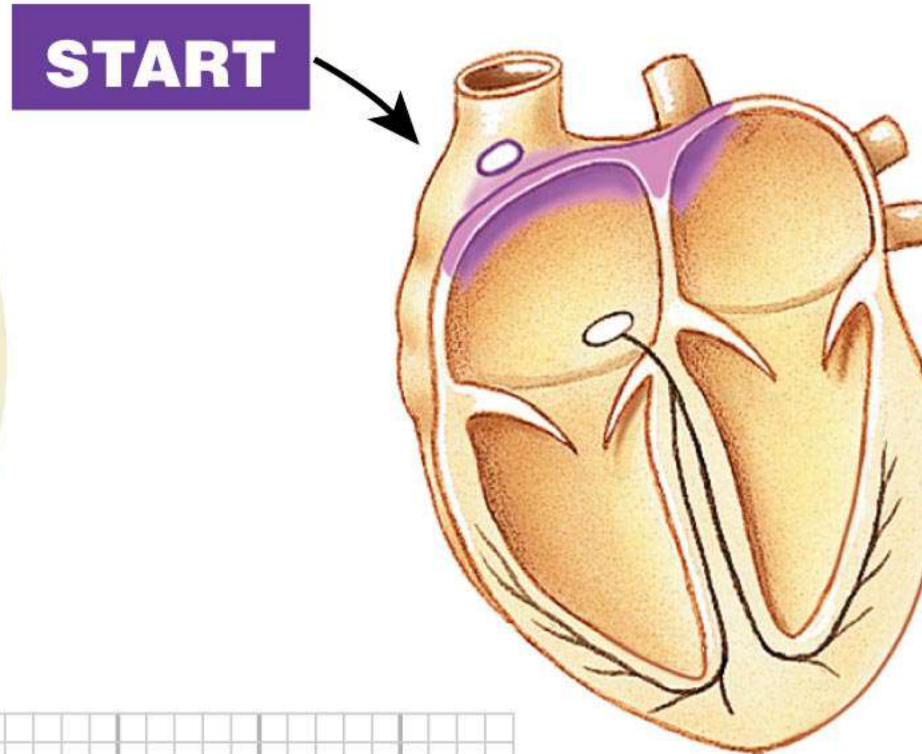


Figure 14-21 - Overview (1 of 9)

ELECTRICAL EVENTS OF THE CARDIAC CYCLE



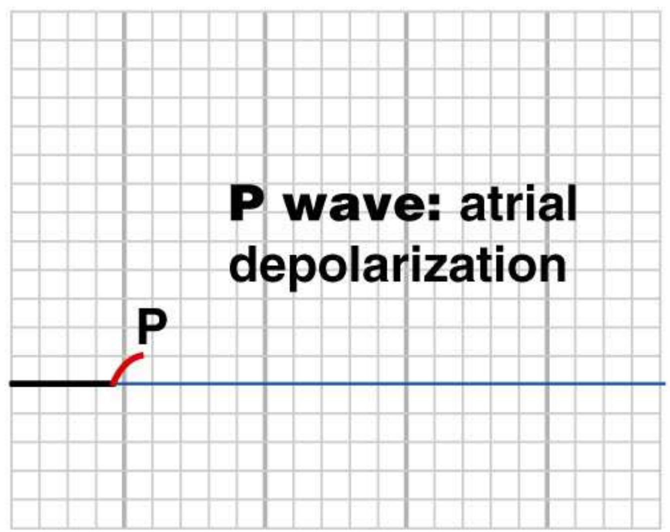
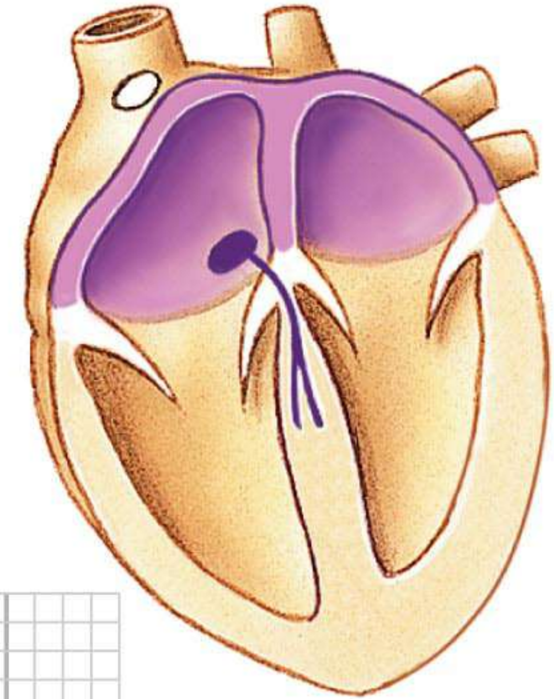
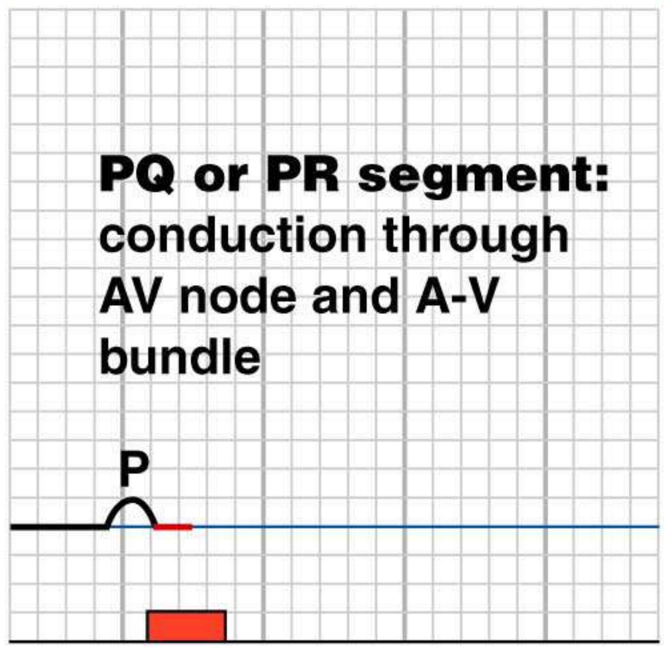


Figure 14-21 (2 of 9)

ELECTRICAL EVENTS OF THE CARDIAC CYCLE

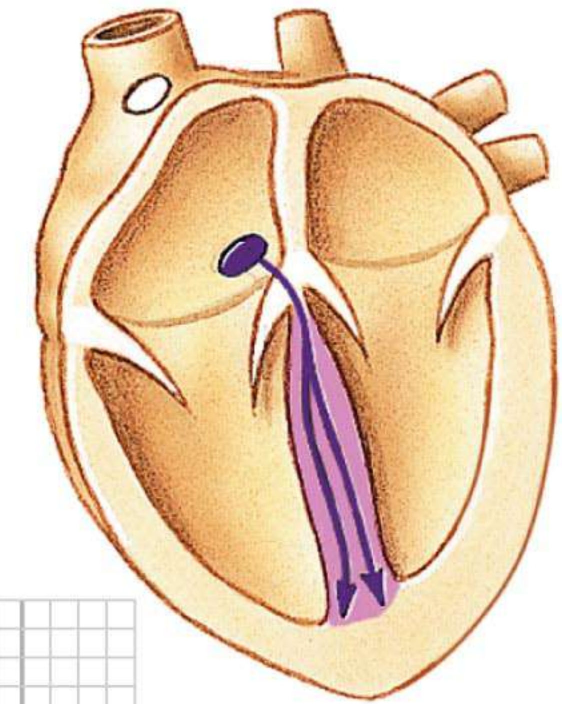




Atria contract.

Figure 14-21 (3 of 9)

ELECTRICAL EVENTS OF THE CARDIAC CYCLE



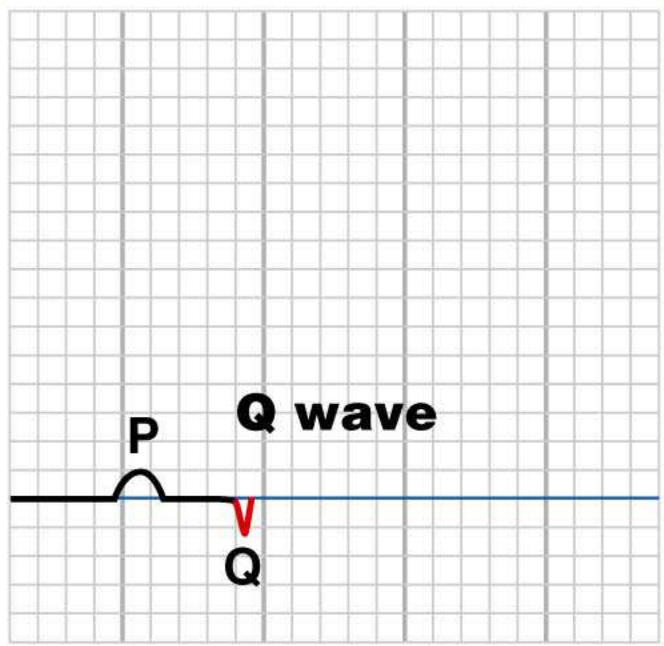
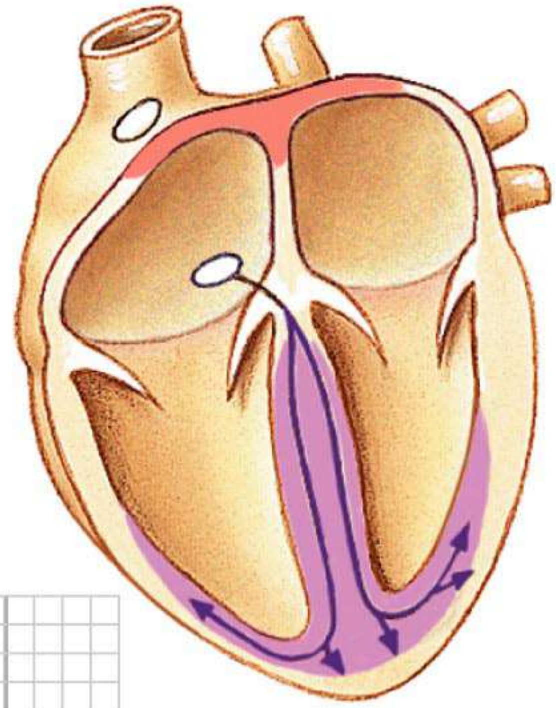


Figure 14-21 (4 of 9)

14-21CorrelateECGHeart_3_L

ELECTRICAL EVENTS OF THE CARDIAC CYCLE



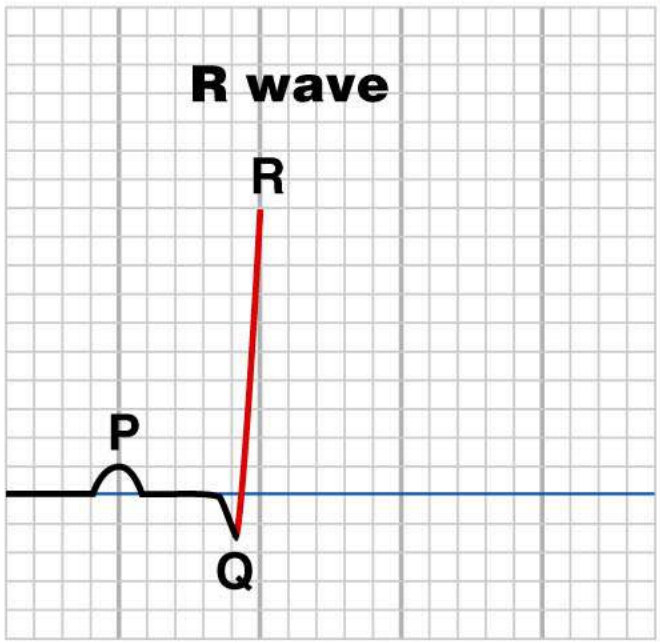


Figure 14-21 (5 of 9)

ELECTRICAL EVENTS OF THE CARDIAC CYCLE

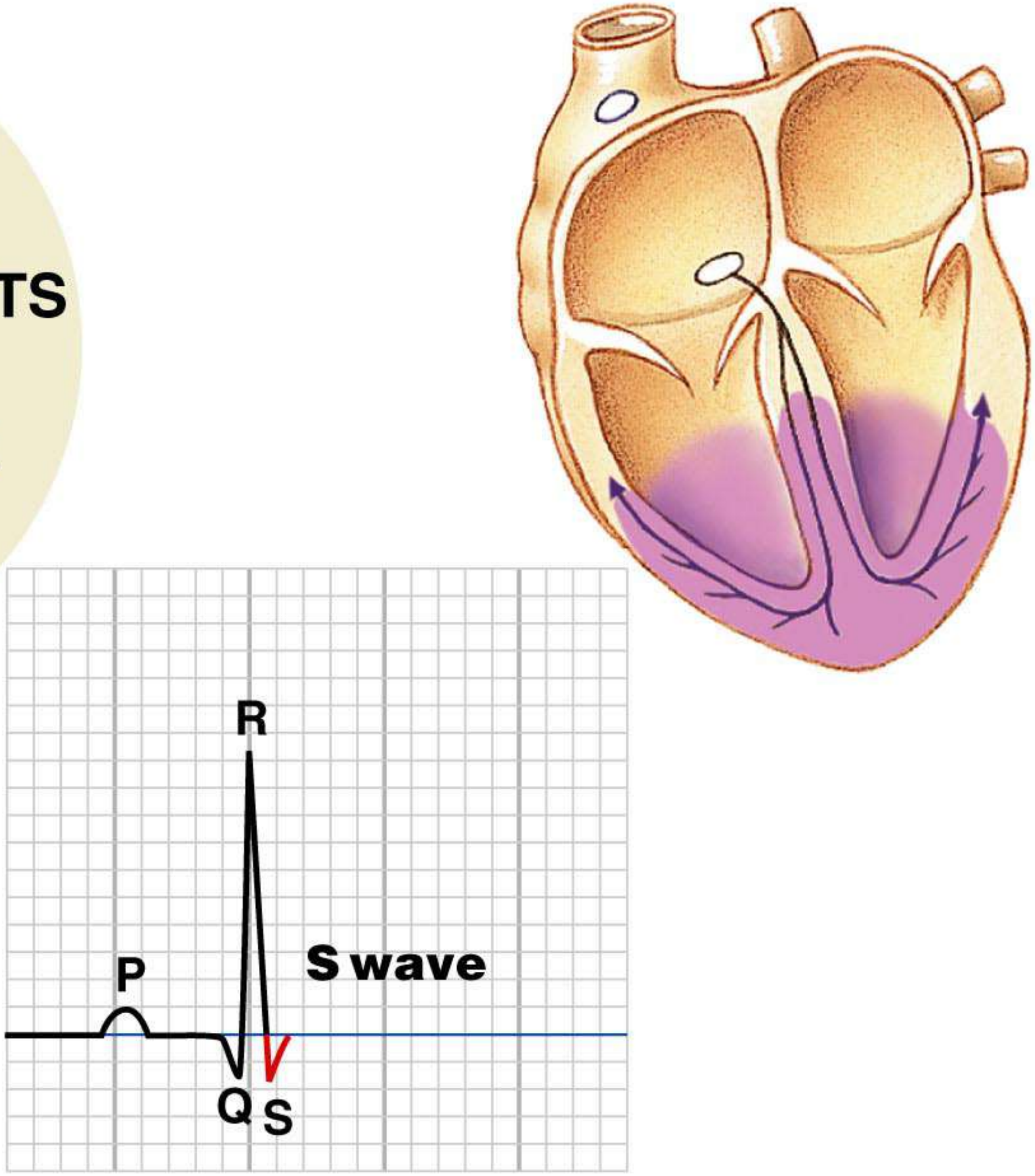
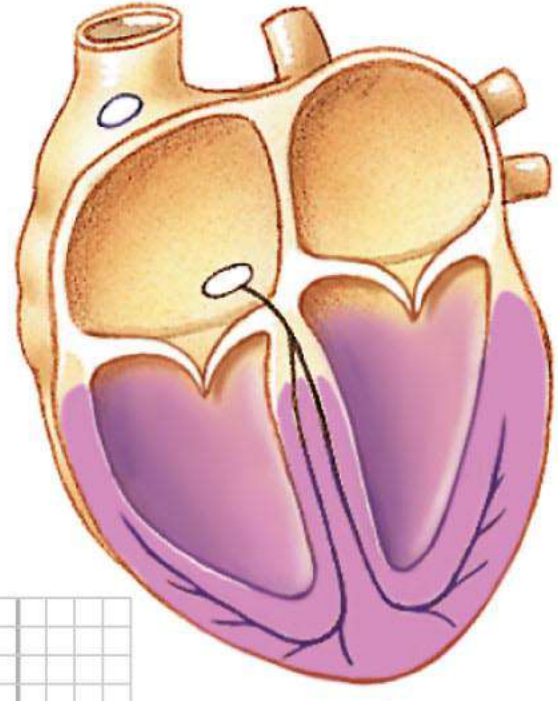
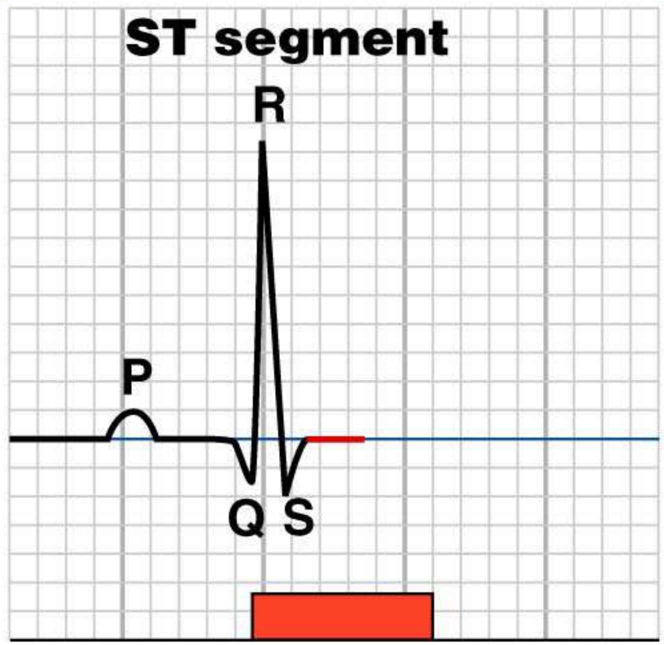


Figure 14-21 (6 of 9)

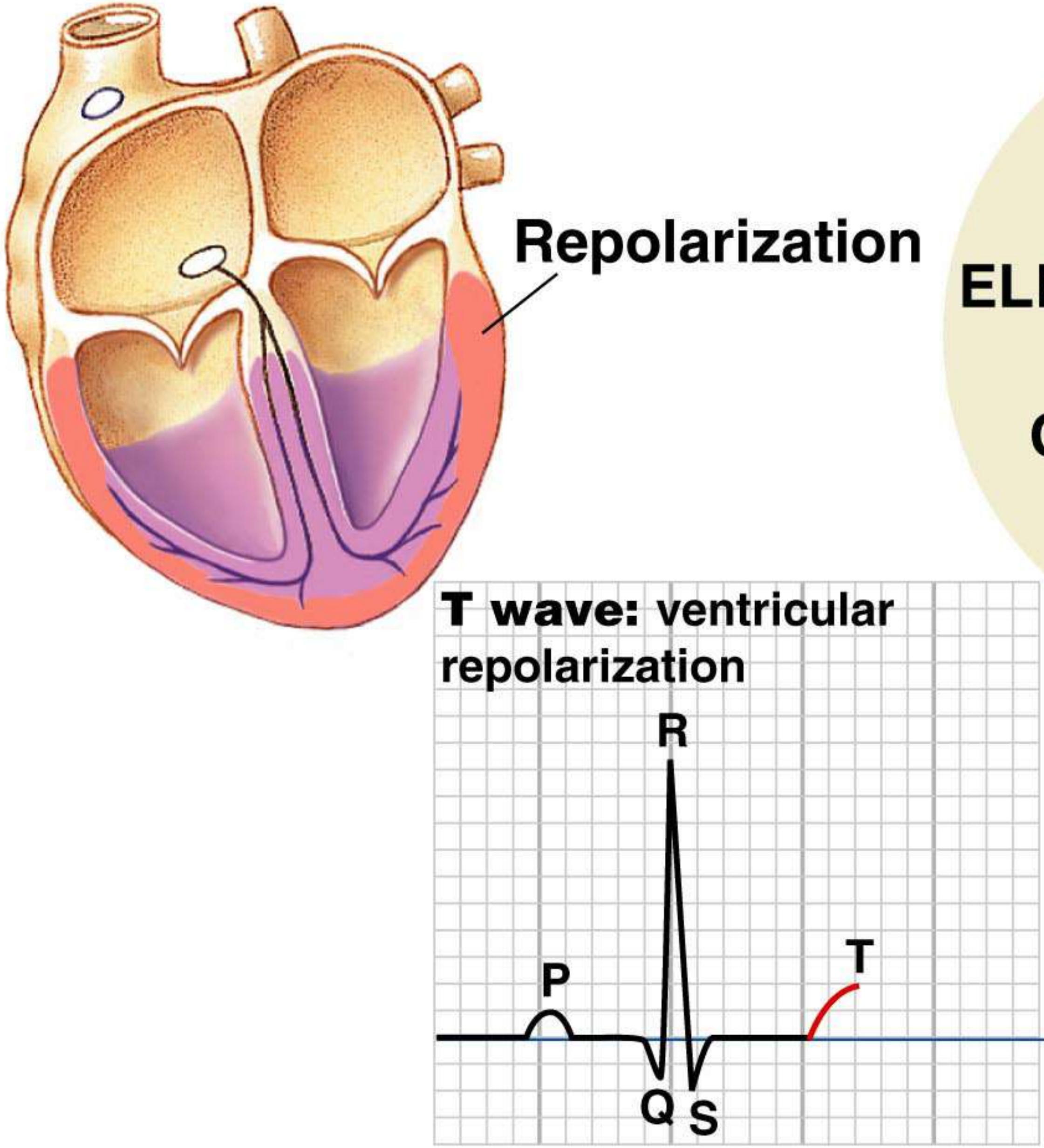
ELECTRICAL EVENTS OF THE CARDIAC CYCLE





Ventricles contract.

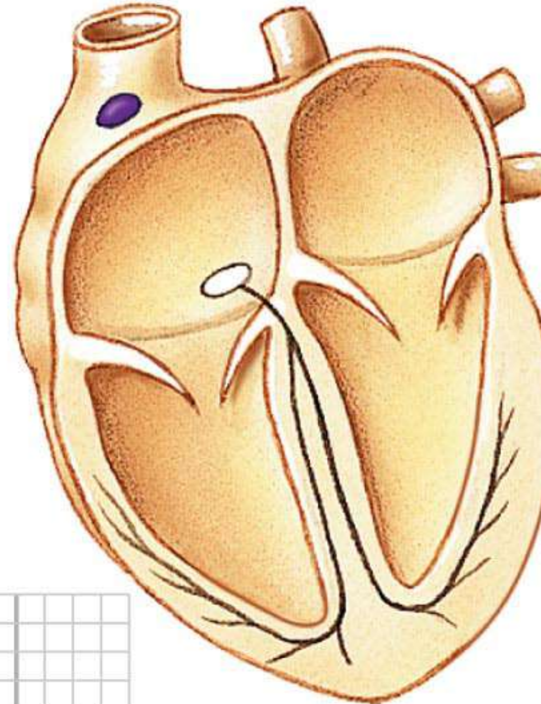
Figure 14-21 (7 of 9)



ELECTRICAL EVENTS OF THE CARDIAC CYCLE

Figure 14-21 (8 of 9)

ELECTRICAL EVENTS OF THE CARDIAC CYCLE



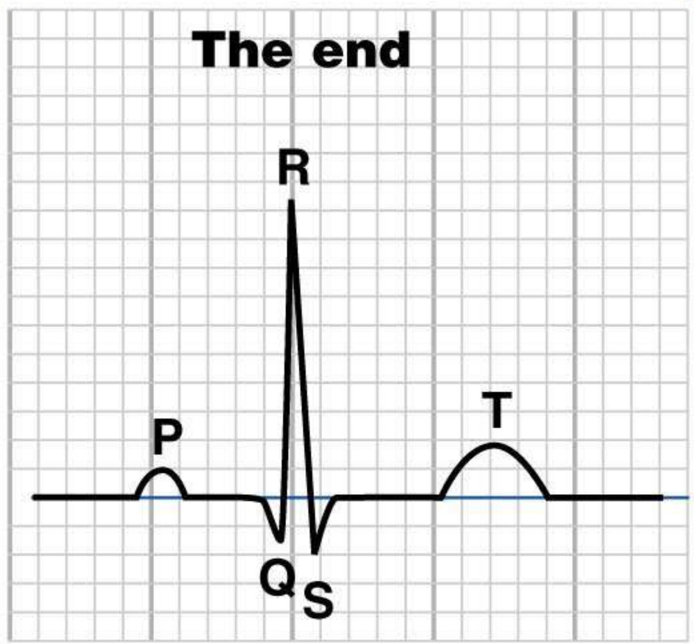
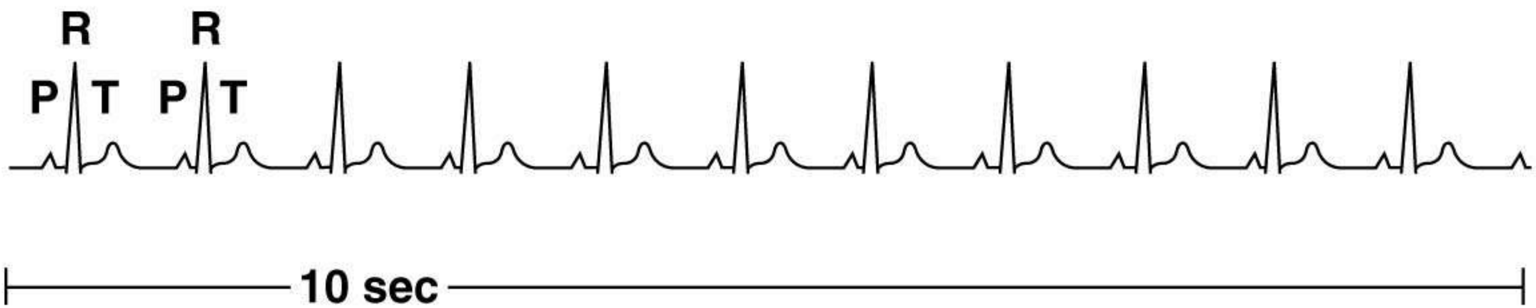


Figure 14-21 (9 of 9)

(a) Normal ECG

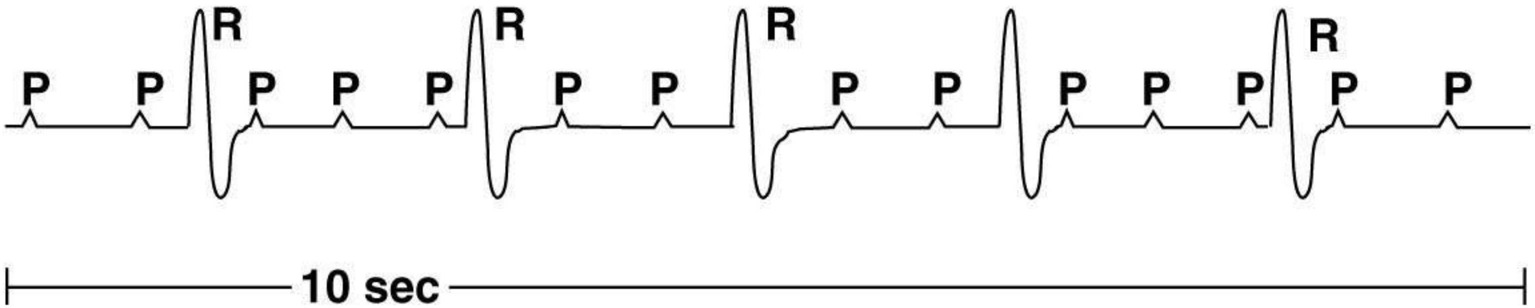


Questions to ask when analyzing ECG tracings:

1. What is the rate? Is it within the normal range of 60-100 beats per minute?
2. Is the rhythm regular?
3. Are all normal waves present in recognizable form?
4. Is there one QRS complex for each P wave? If yes, is the P-R segment constant in length?
5. If there is not one QRS complex for each P wave, count the heart rate using the P waves, then count it according to the R waves. Are the rates the same? Which wave would agree with the pulse felt at the wrist?

Figure 14-23a

(b) Third-degree block



Questions to ask when analyzing ECG tracings:

1. What is the rate? Is it within the normal range of 60-100 beats per minute?

2. Is the rhythm regular?

3. Are all normal waves present in recognizable form?

4. Is there one QRS complex for each P wave?
   If yes, is the P-R segment constant in length?

5. If there is not one QRS complex for each P wave, count the heart rate using the P waves, then count it according to the R waves. Are the rates the same? Which wave would agree with the pulse felt at the wrist?

Figure 14-23b

(c) Atrial fibrillation

Questions to ask when analyzing ECG tracings:

1. What is the rate? Is it within the normal range of 60-100 beats per minute?

2. Is the rhythm regular?

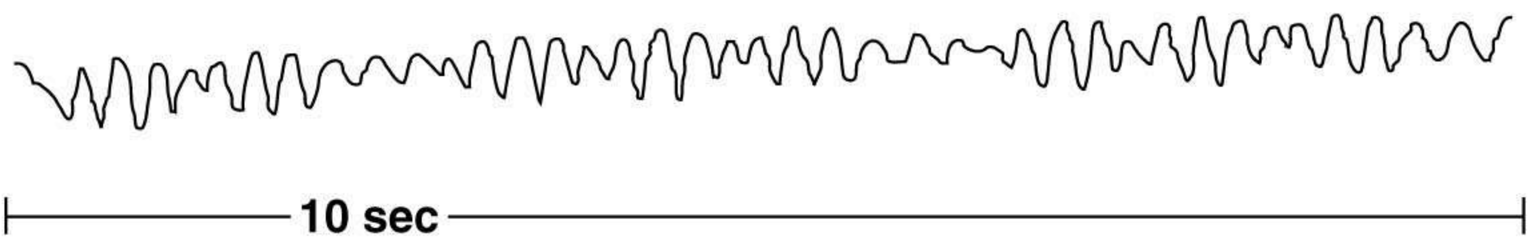
3. Are all normal waves present in recognizable form?

4. Is there one QRS complex for each P wave?
   If yes, is the P-R segment constant in length?

5. If there is not one QRS complex for each P wave, count the heart rate using the P waves, then count it according to the R waves. Are the rates the same? Which wave would agree with the pulse felt at the wrist?

Figure 14-23c

(d) Ventricular fibrillation



Questions to ask when analyzing ECG tracings:

1. What is the rate? Is it within the normal range of 60-100 beats per minute?

2. Is the rhythm regular?

3. Are all normal waves present in recognizable form?

4. Is there one QRS complex for each P wave?
   If yes, is the P-R segment constant in length?

5. If there is not one QRS complex for each P wave, count the heart rate using the P waves, then count it according to the R waves. Are the rates the same? Which wave would agree with the pulse felt at the wrist?

Figure 14-23d

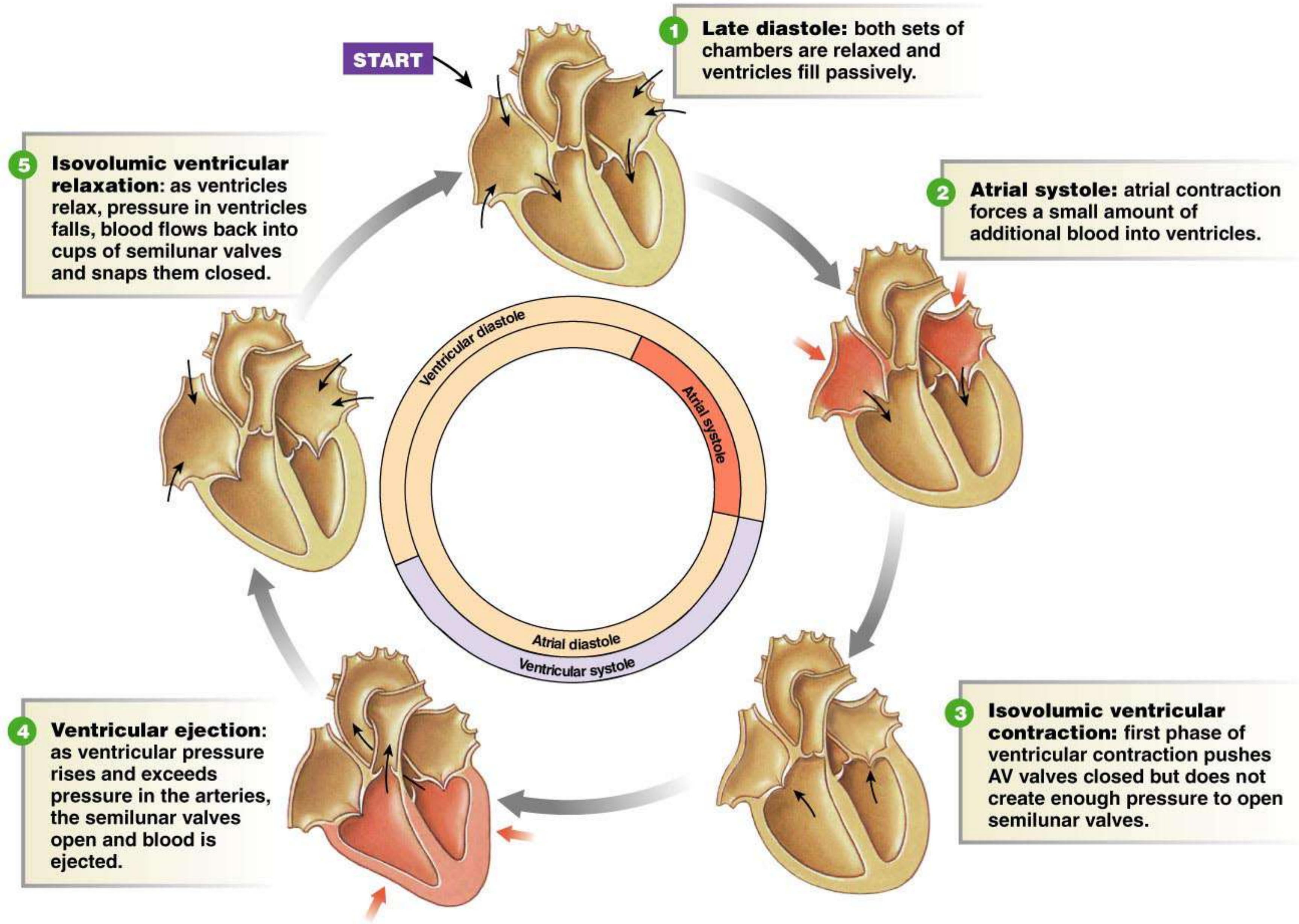
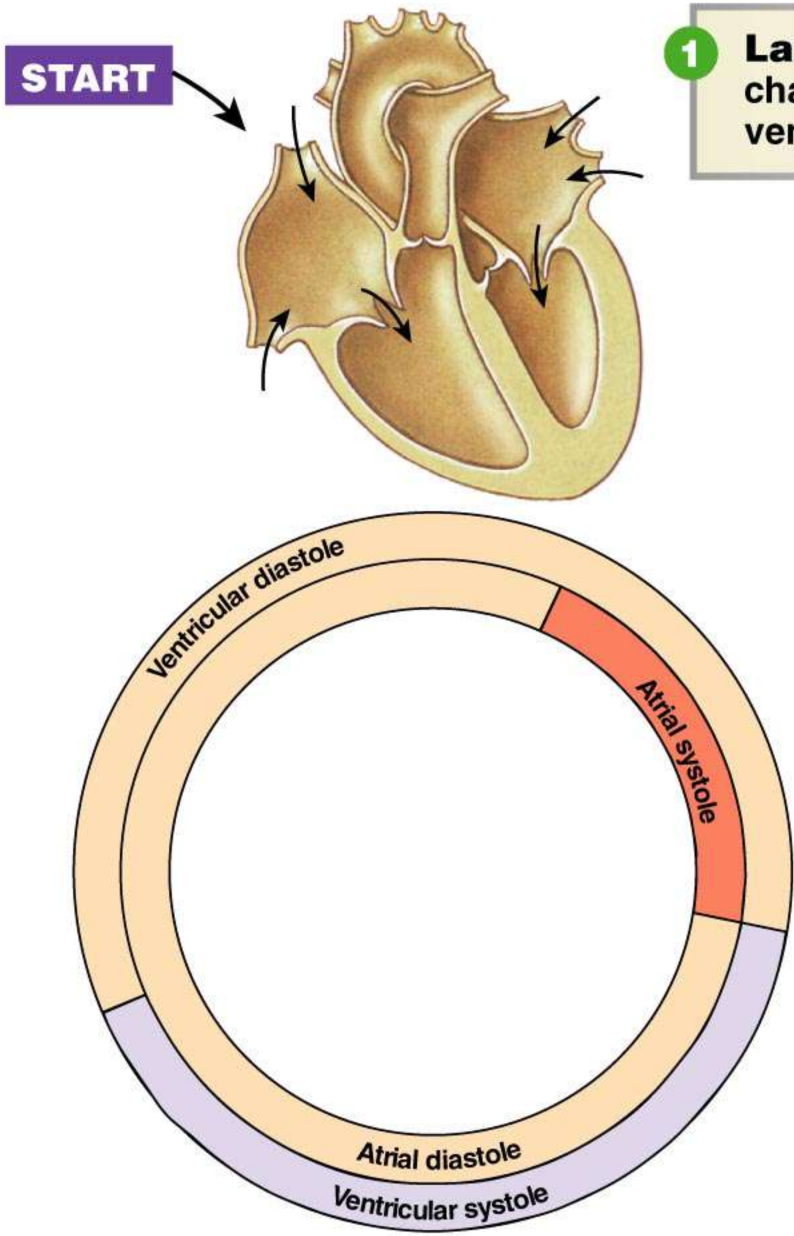
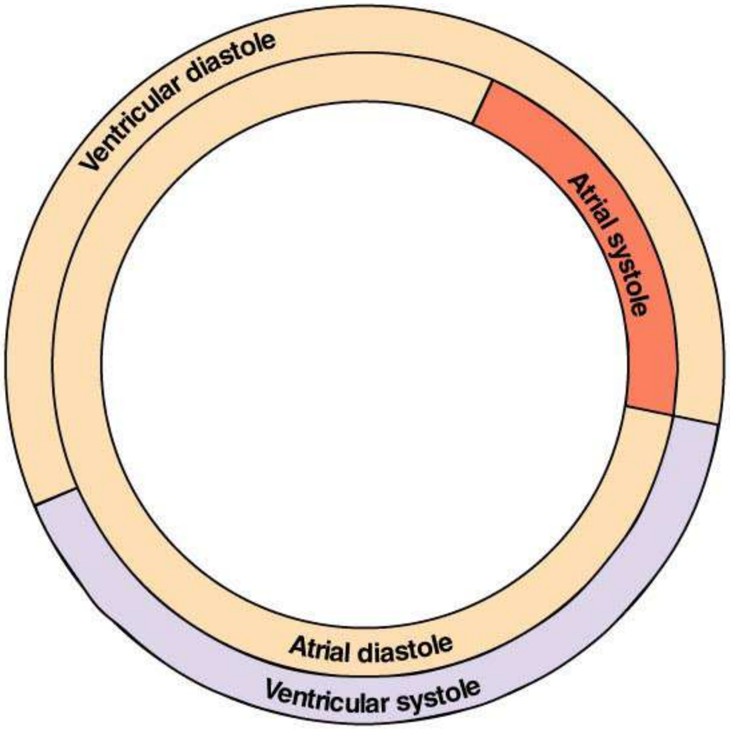


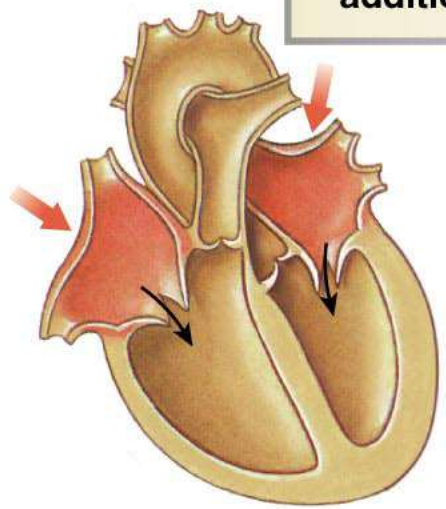
Figure 14-24 - Overview



1 **Late diastole:** both sets of chambers are relaxed and ventricles fill passively.

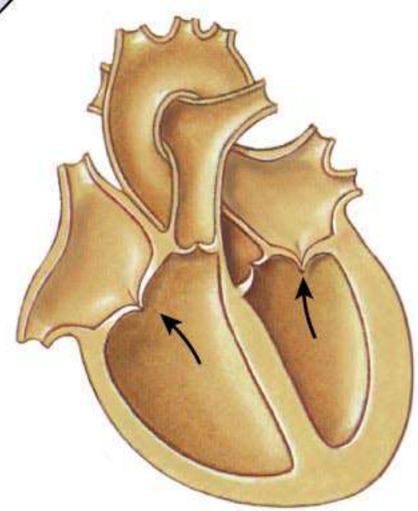
Figure 14-24, step 1





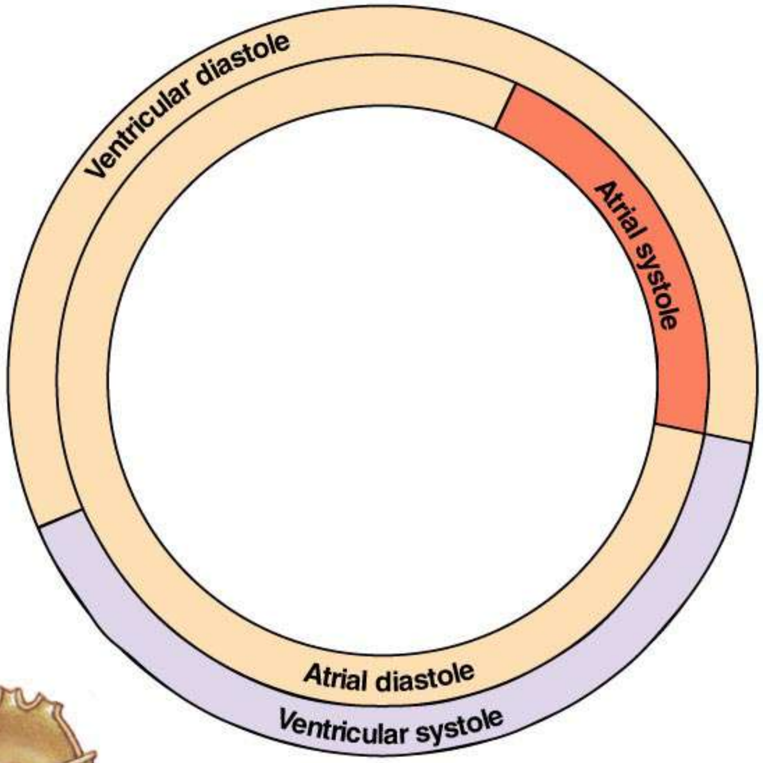
2 **Atrial systole:** atrial contraction forces a small amount of additional blood into ventricles.

Figure 14-24, step 2



3 **Isovolumic ventricular contraction:** first phase of ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves.

Figure 14-24, step 3



4 Ventricular ejection: as ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.

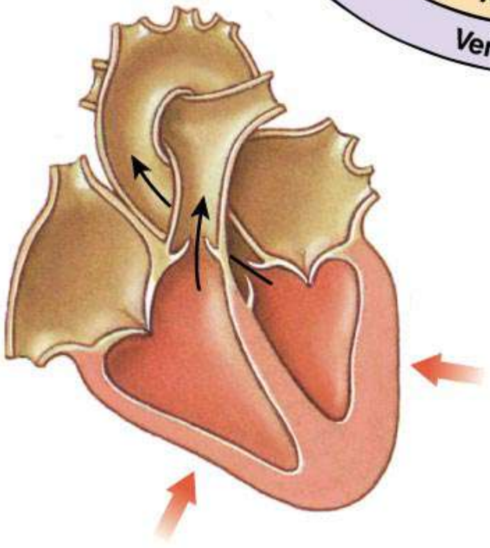


Figure 14-24, step 4

Isovolumic ventricular relaxation: as ventricles relax, pressure in ventricles falls, blood flows back into cups of semilunar valves and snaps them closed.

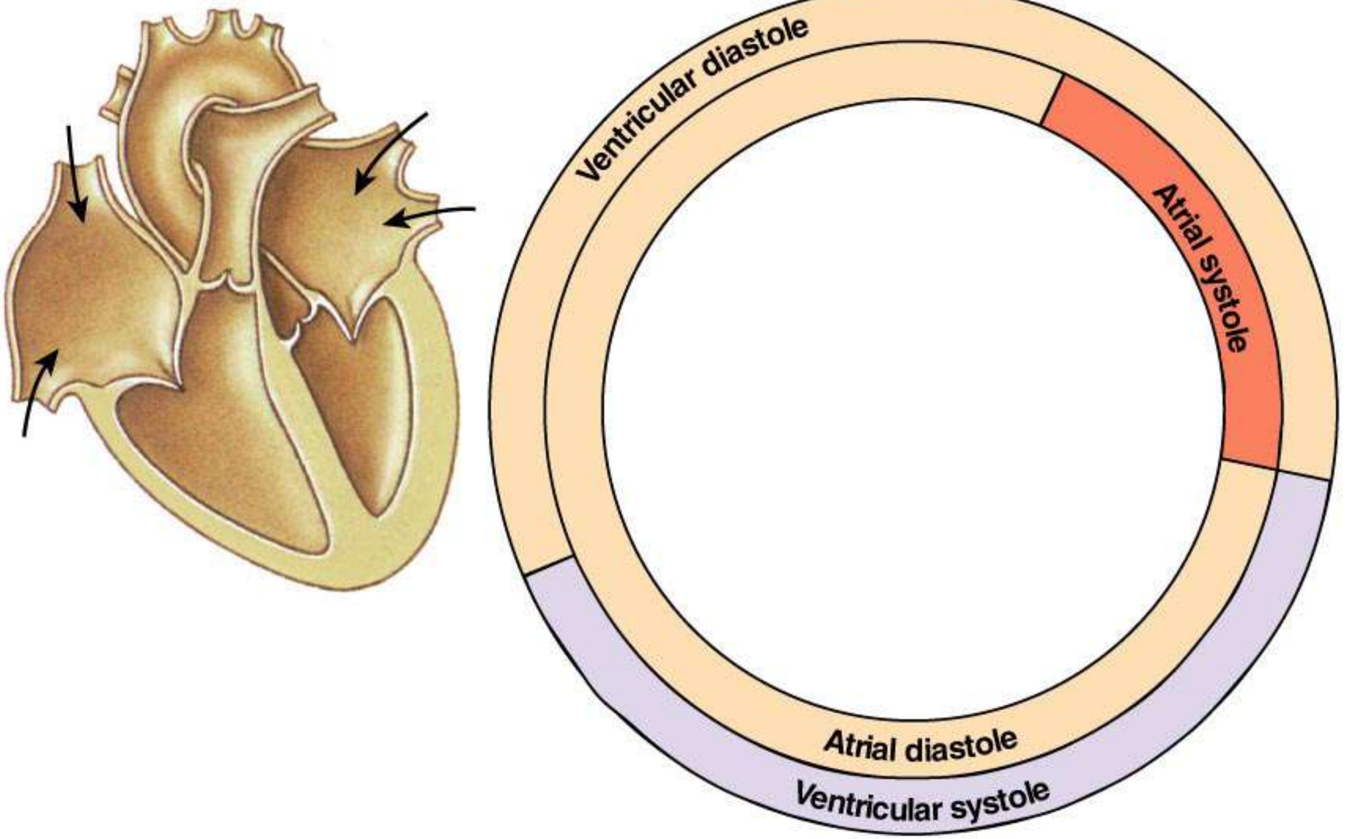


Figure 14-24, step 5

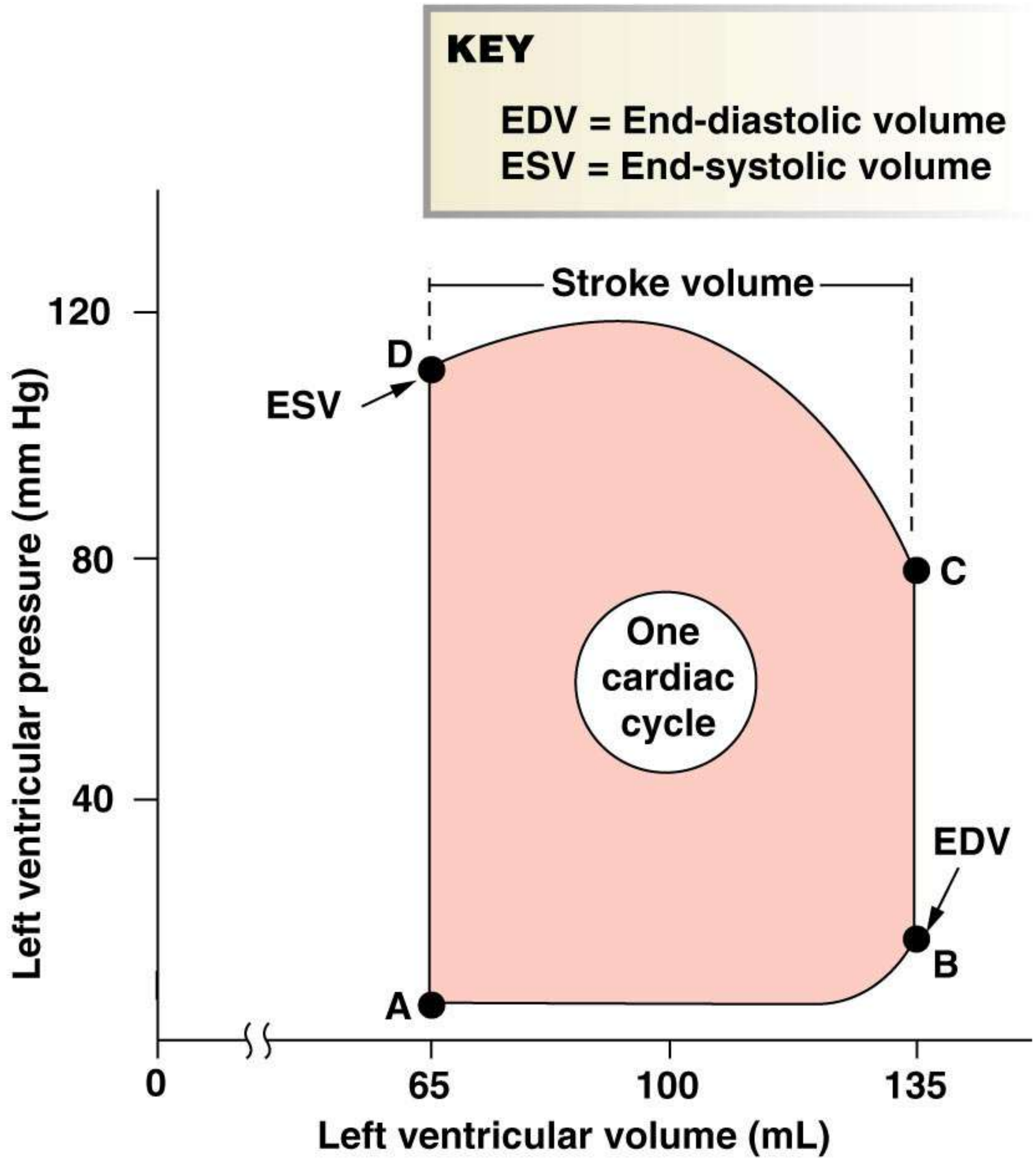

KEY
EDV = End-diastolic volume
ESV = End-systolic volume
Stroke volume
120
80
40
0
65
100
135
D
ESV
C
One cardiac cycle
EDV
B
A
Left ventricular pressure (mm Hg)
Left ventricular volume (mL)

Figure 14-25

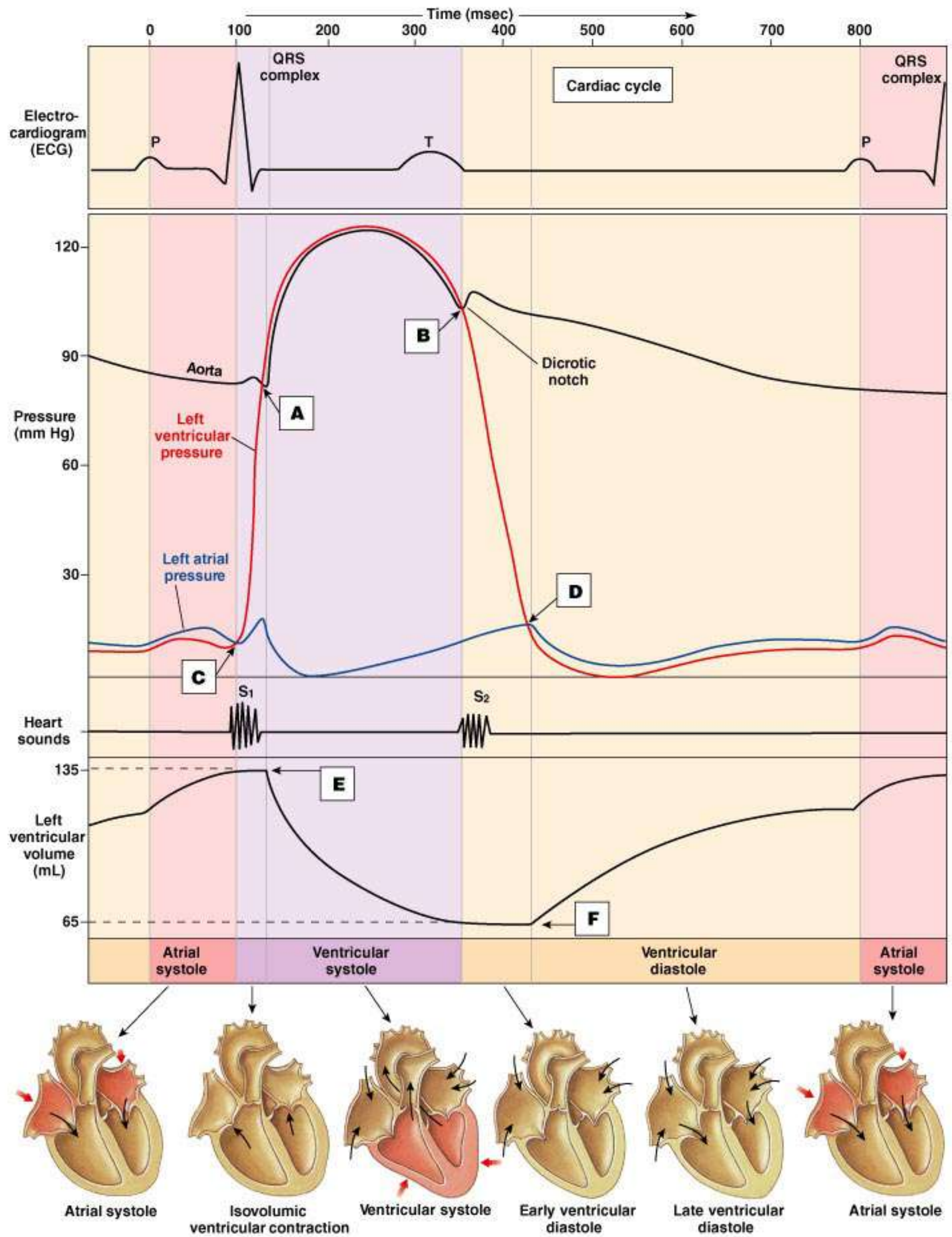


Figure 14-26 – Overview (1 of 5)

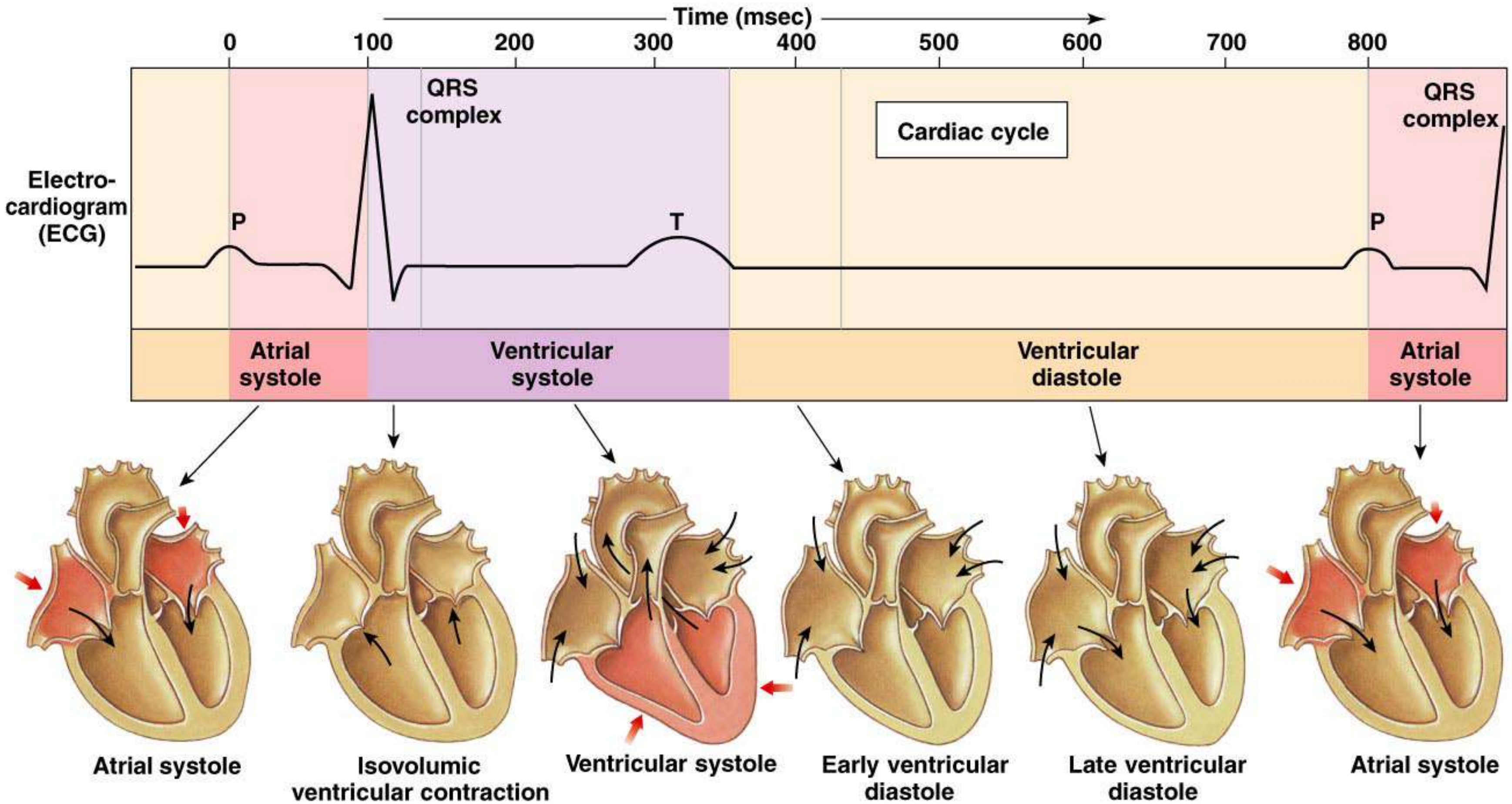


Figure 14-26 (2 of 5)

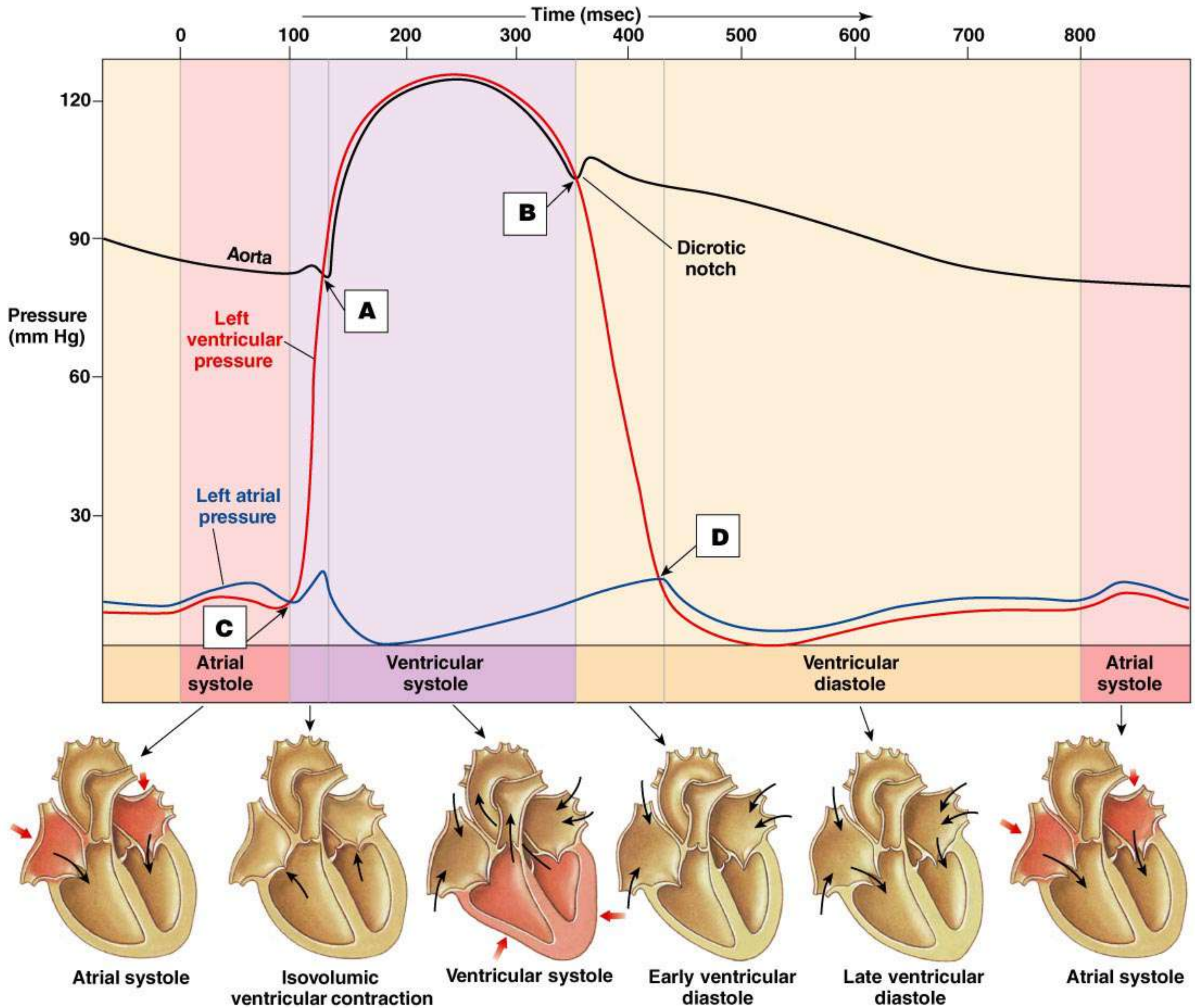


Figure 14-26 (3 of 5)

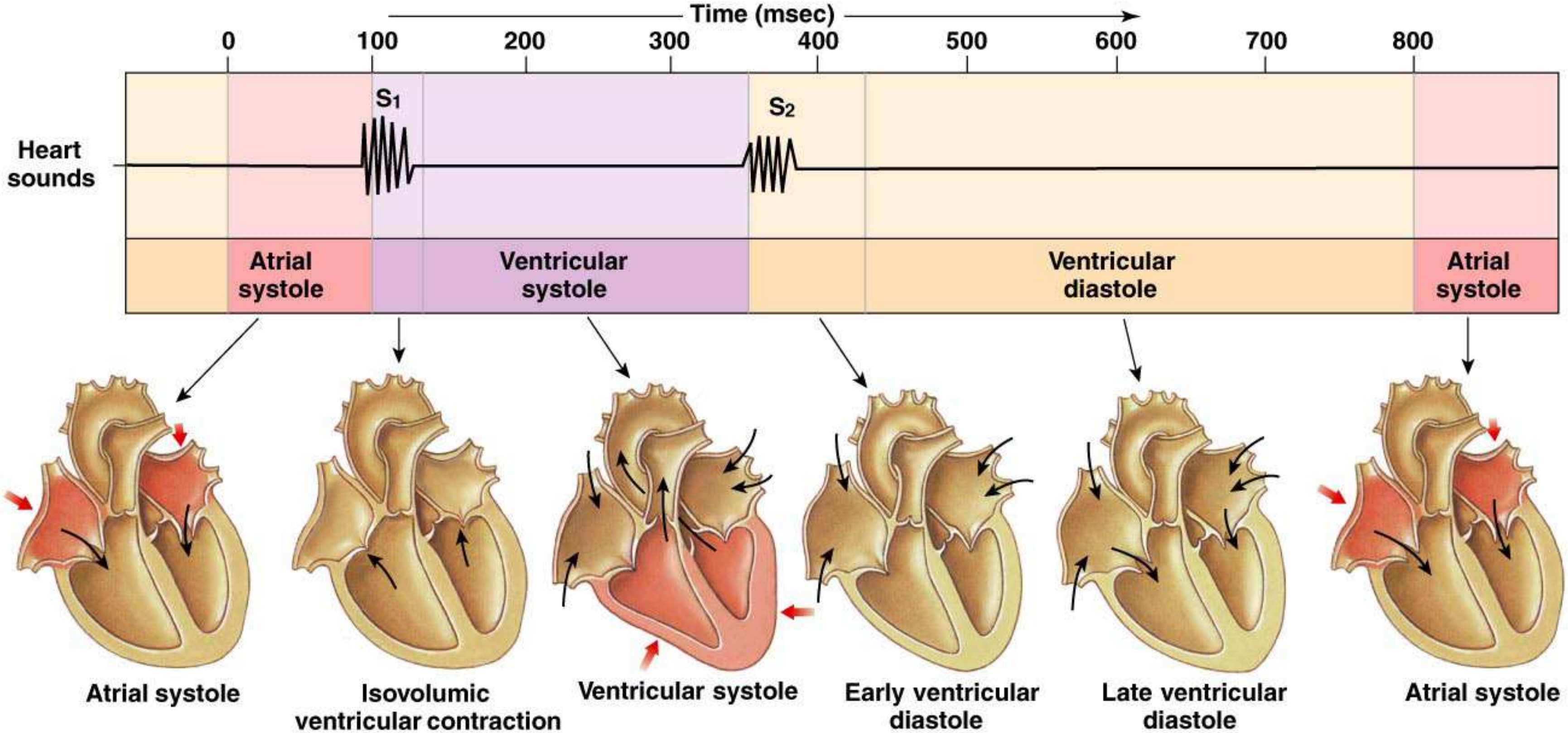


Figure 14-26 (4 of 5)

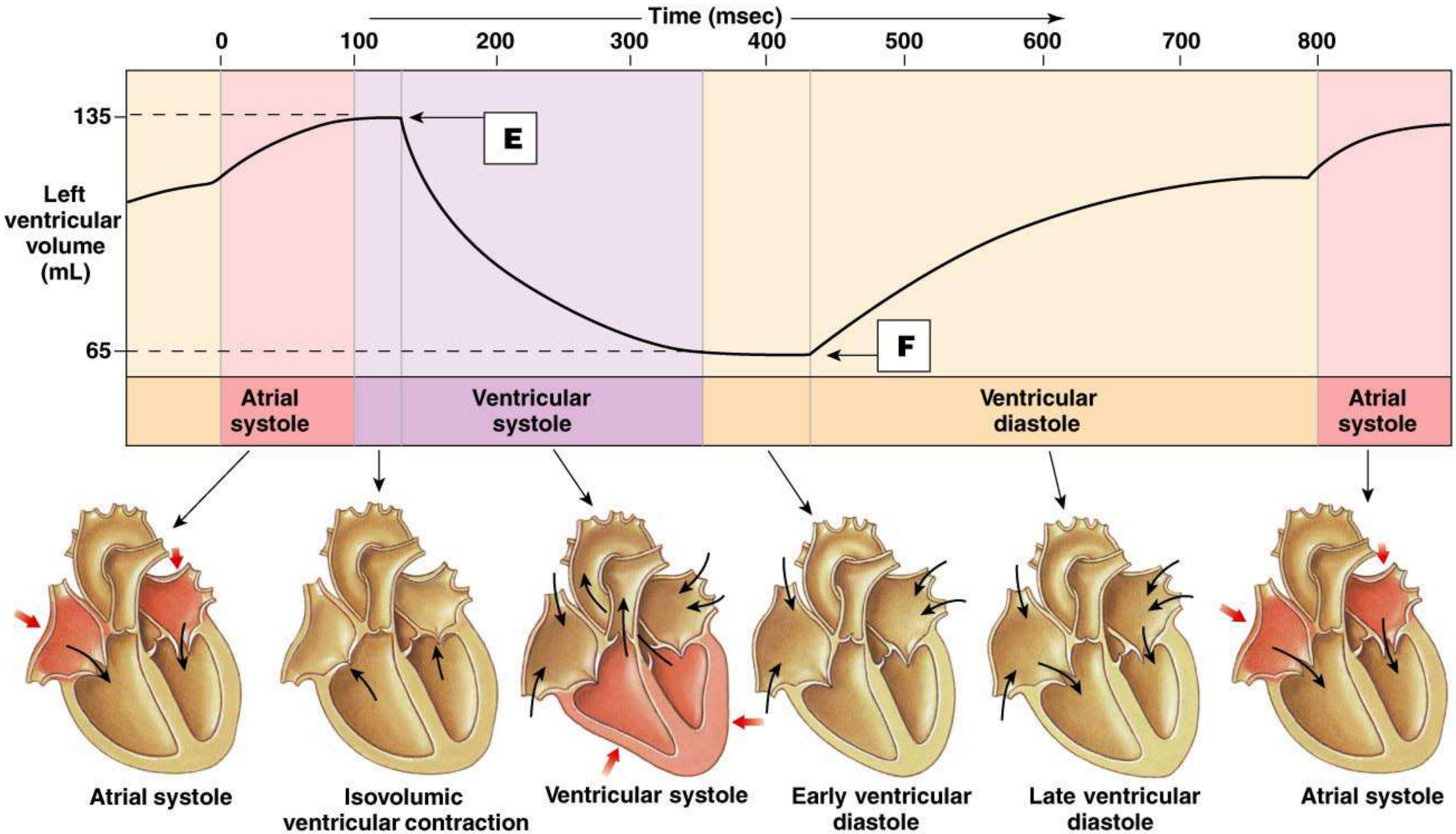
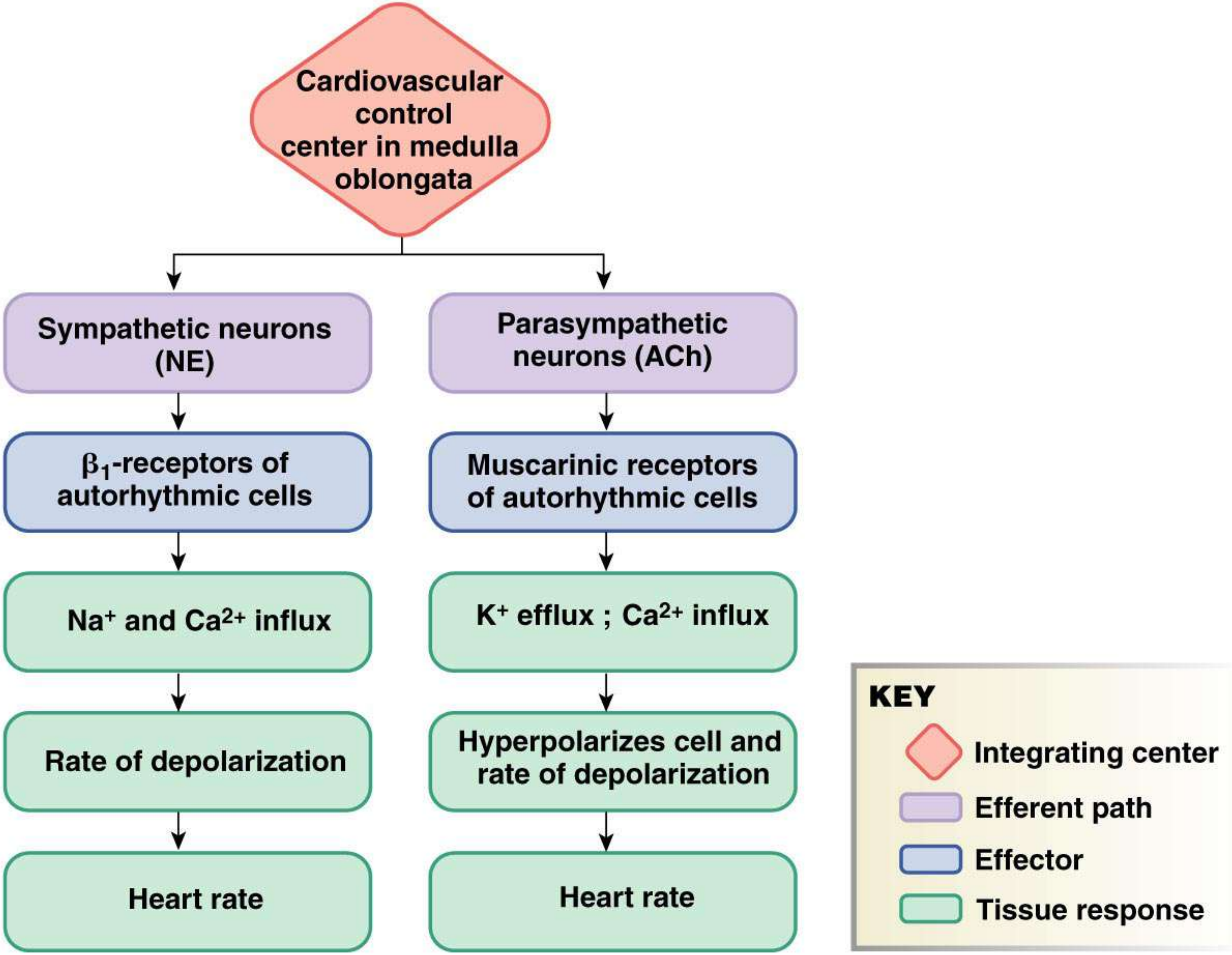


Figure 14-26 (5 of 5)



Cardiovascular control center in medulla oblongata
Sympathetic neurons (NE)
Parasympathetic neurons (ACh)
β_1-receptors of autorhythmic cells
Muscarinic receptors of autorhythmic cells
Na^+ and Ca^{2+} influx
K^+ efflux ; Ca^{2+} influx
Rate of depolarization
Hyperpolarizes cell and rate of depolarization
Heart rate
Heart rate
KEY
Integrating center
Efferent path
Effector
Tissue response

Figure 14-27

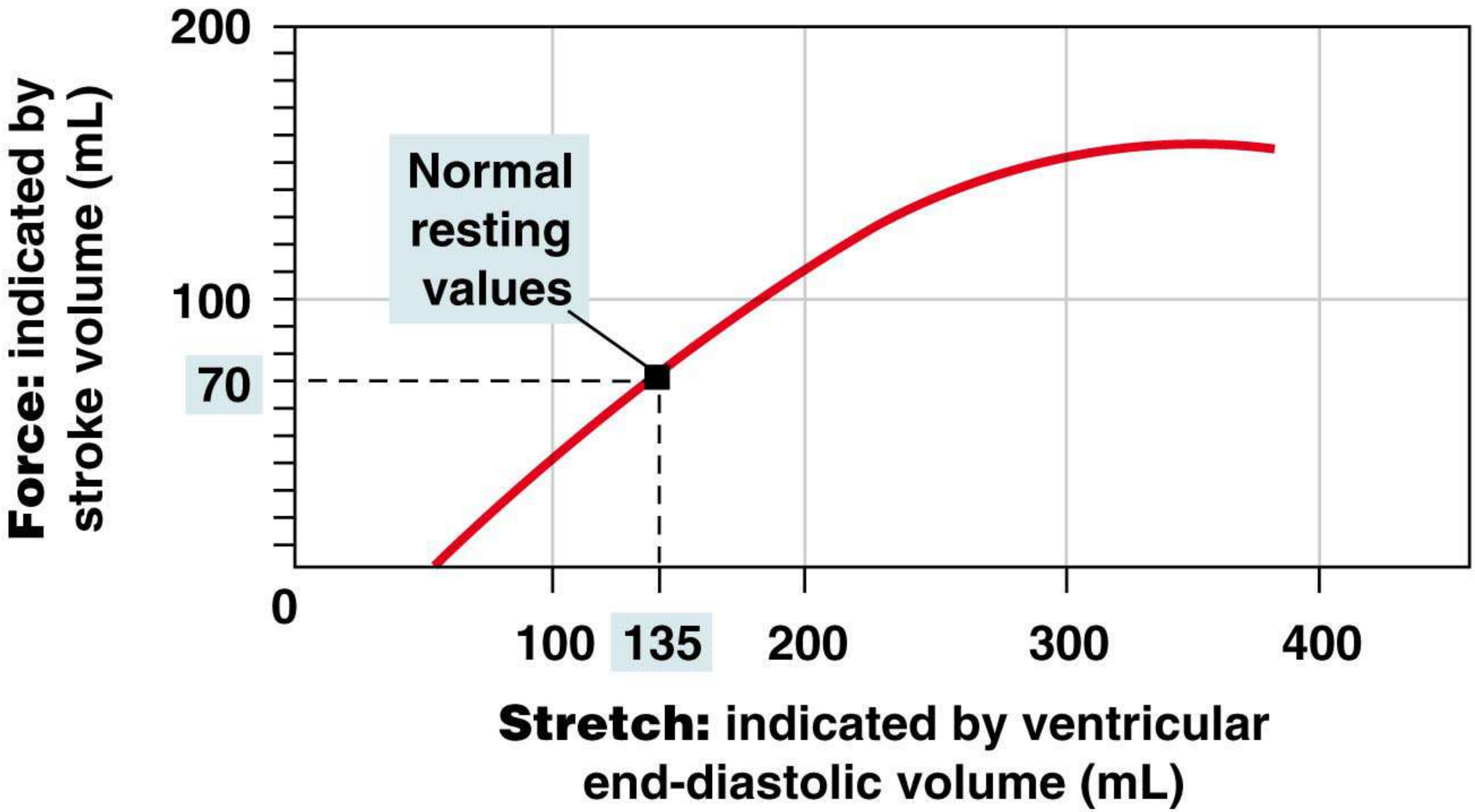

200
100
70
0
Force: indicated by stroke volume (mL)
Normal resting values
100
135
200
300
400
Stretch: indicated by ventricular end-diastolic volume (mL)

Figure 14-28

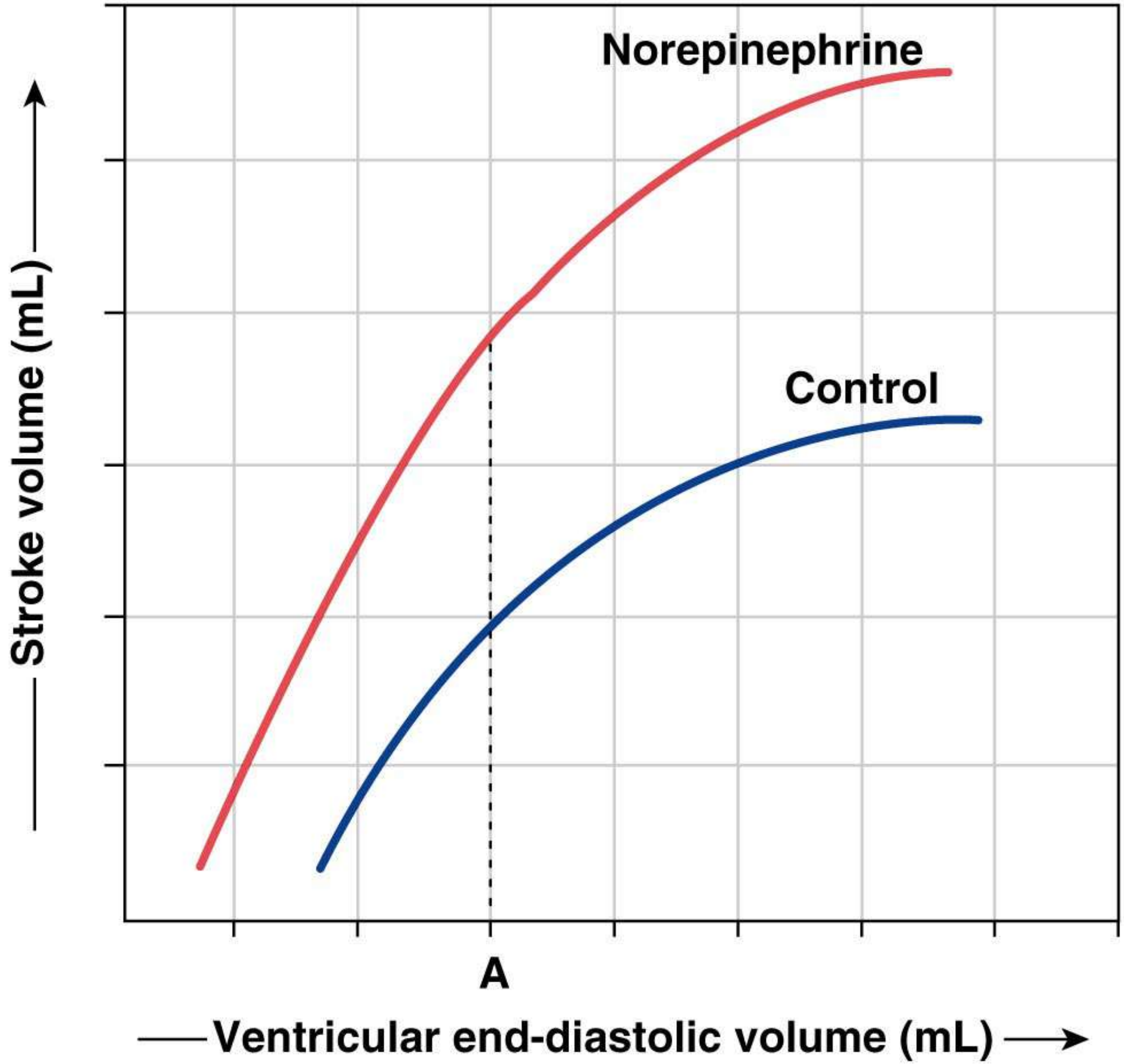

Norepinephrine
Control
Stroke volume (mL)
A
Ventricular end-diastolic volume (mL)

Figure 14-29

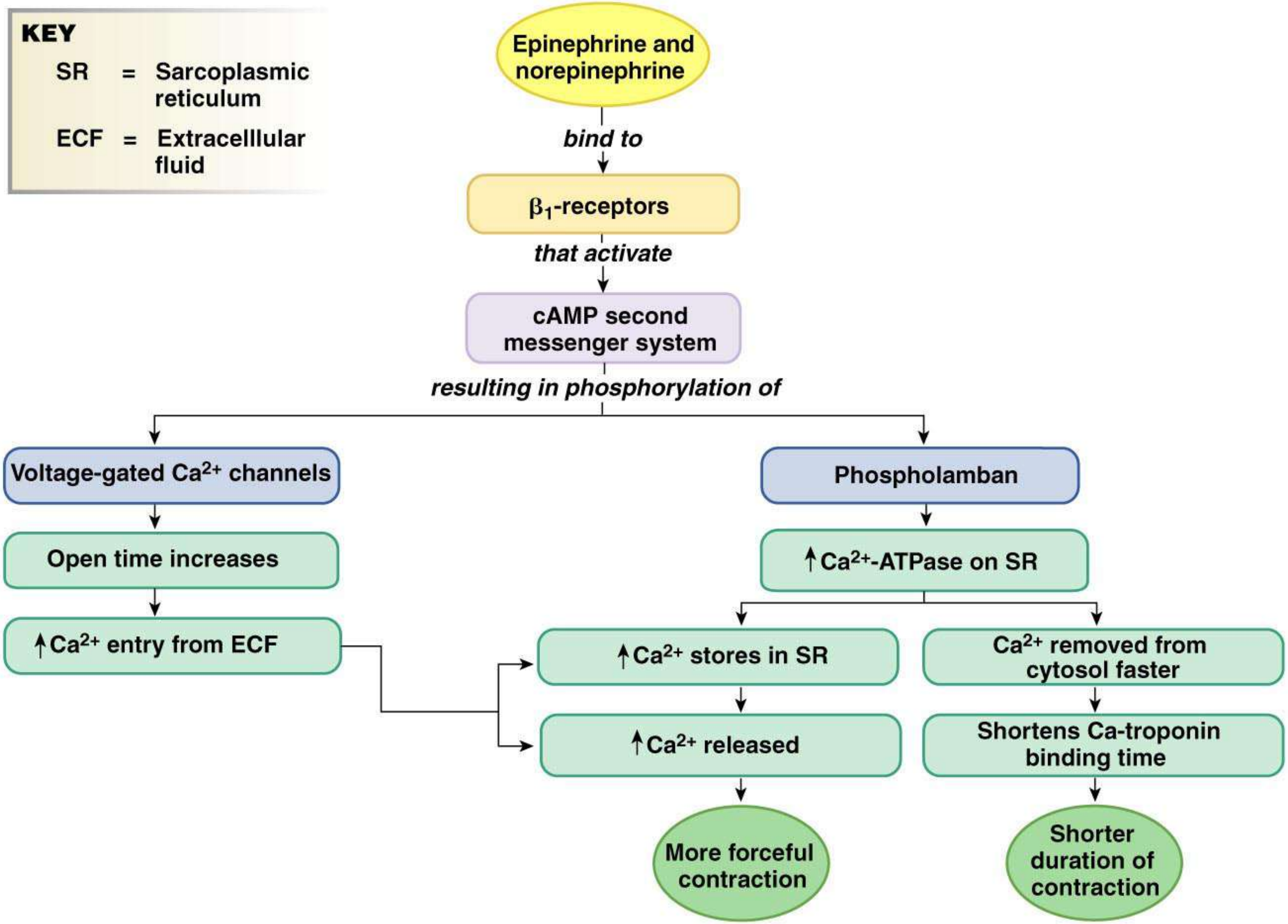

KEY
SR = Sarcoplasmic reticulum
ECF = Extracelllular fluid
Epinephrine and norepinephrine
bind to
β1-receptors
that activate
cAMP second messenger system
resulting in phosphorylation of
Voltage-gated Ca2+ channels
Open time increases
↑Ca2+ entry from ECF
Phospholamban
↑Ca2+-ATPase on SR
↑Ca2+ stores in SR
↑Ca2+ released
More forceful contraction
Ca2+ removed from cytosol faster
Shortens Ca-troponin binding time
Shorter duration of contraction

Figure 14-30

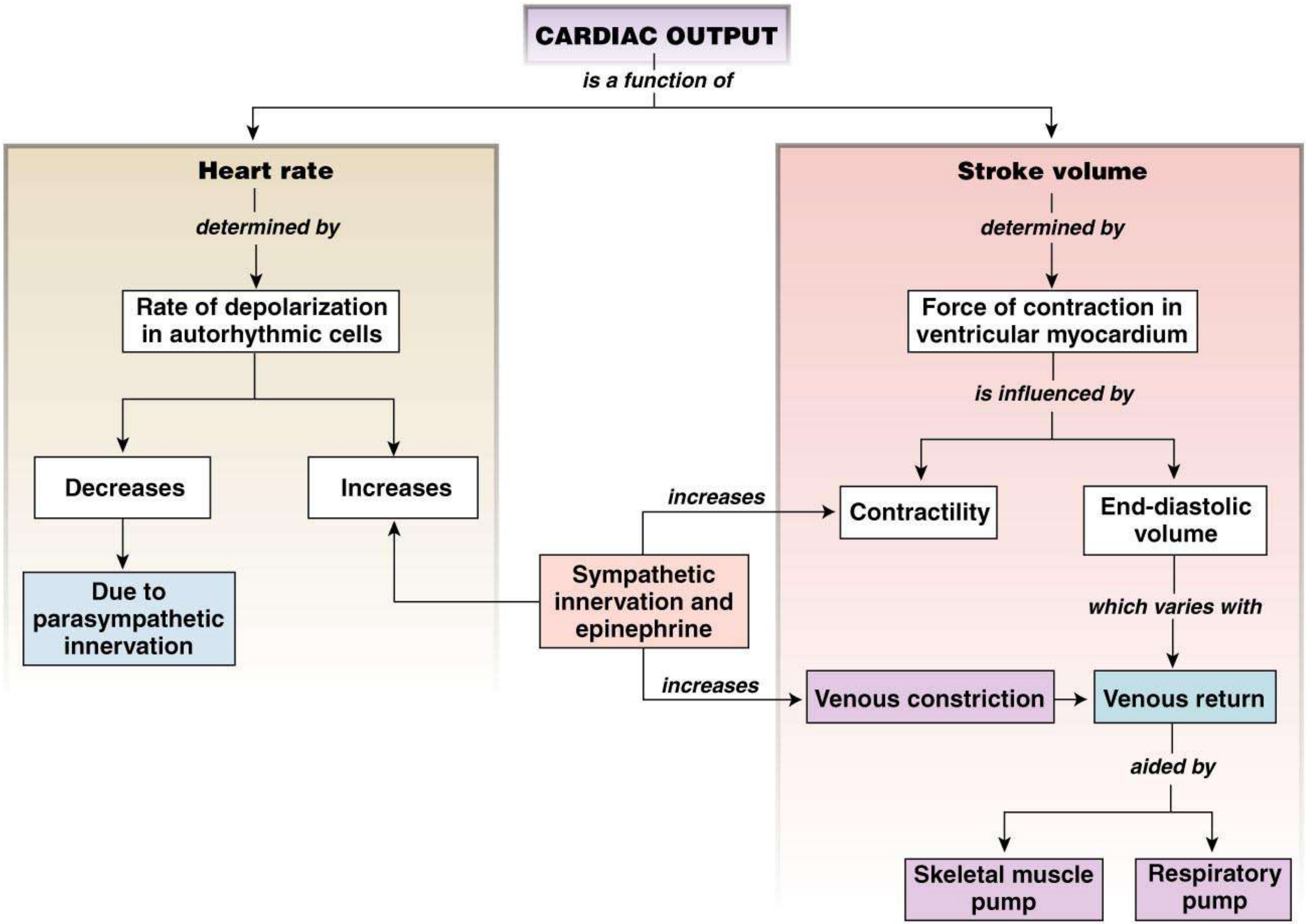

CARDIAC OUTPUT
is a function of
Heart rate
determined by
Rate of depolarization in autorhythmic cells
Decreases
Increases
Due to parasympathetic innervation
Stroke volume
determined by
Force of contraction in ventricular myocardium
is influenced by
Contractility
End-diastolic volume
increases
Sympathetic innervation and epinephrine
which varies with
increases
Venous constriction
Venous return
aided by
Skeletal muscle pump
Respiratory pump

Figure 14-31