Topic 4.2 The Heart

Chapter 18

Mastering A and P and the Interactive Physiology CD will be very helpful for this topic!

J. Carnegie, UofO

4.2.1 Describe the internal and external anatomy of the heart

- simply a transport system **pump**; hollow blood vessels provide delivery routes
- enclosed within **mediastinum** of thorax
- extends obliquely for 12-14 cm from 2nd rib to 5th intercostal space; 2/3 of mass on left side; right side lying on diaphragm
- broad, flat **base** directed toward rt shoulder; **apex** points toward lt hip
- 3 layers: **pericardium**, **myocardium**, **endocardium**

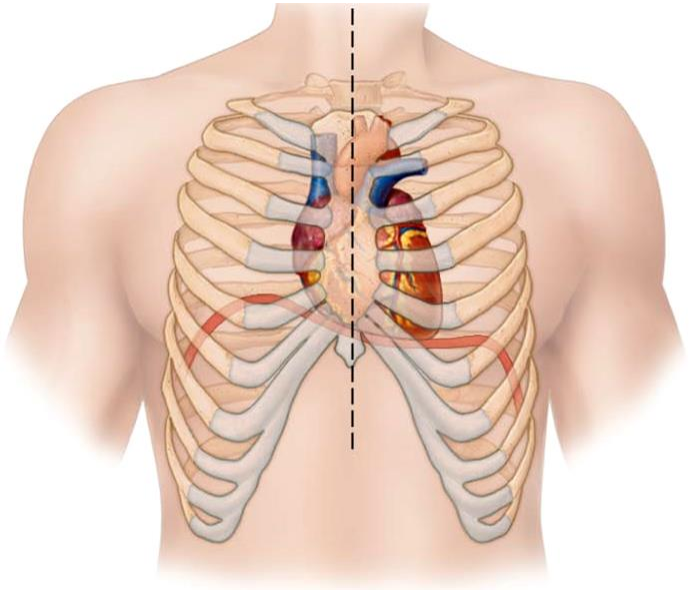
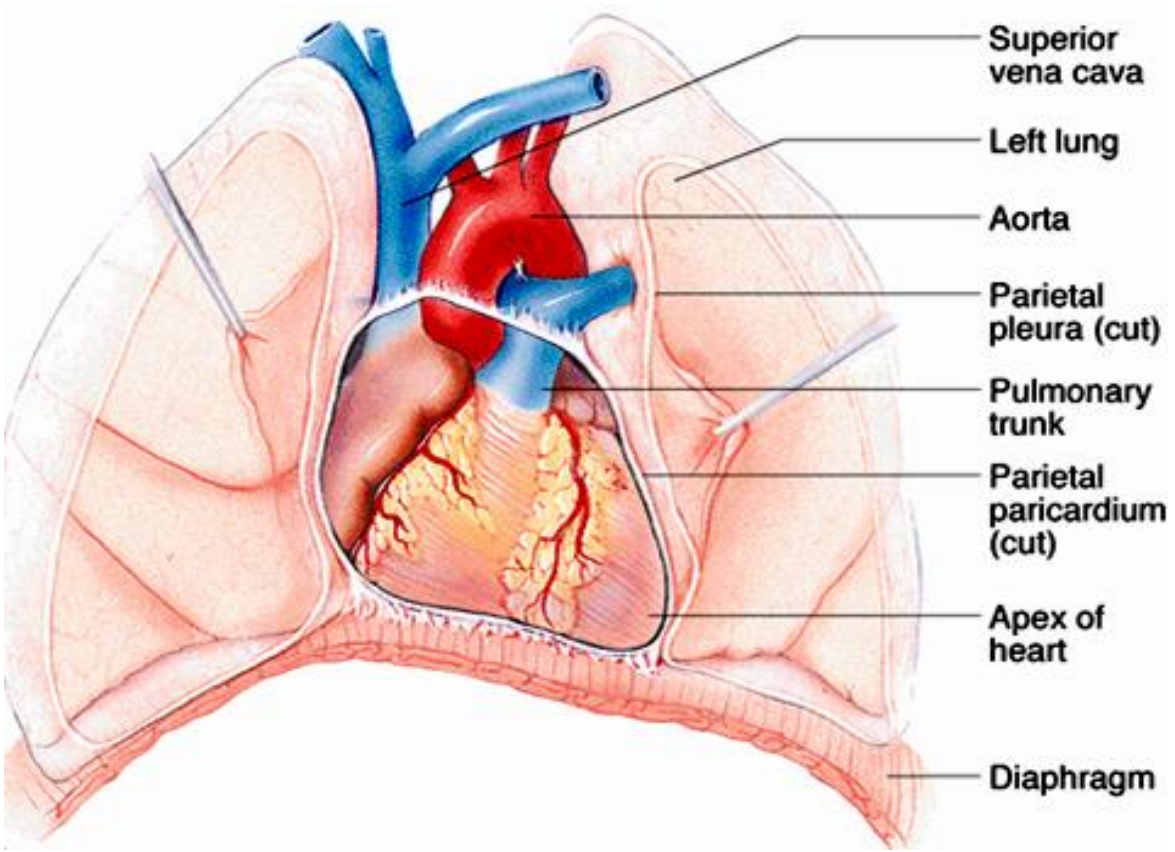


Fig. 18.2



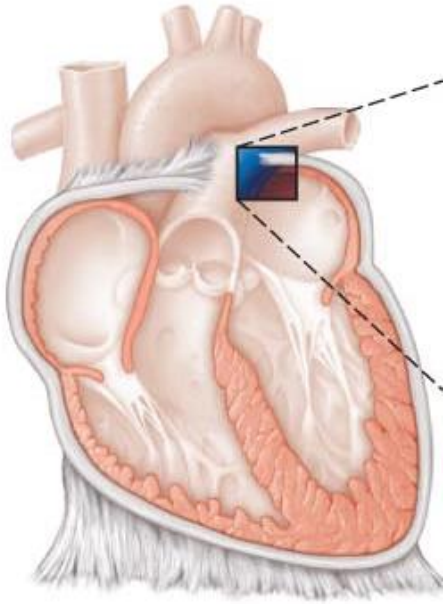
1. Pericardium

- double-walled, fibro-serous sac

a) **fibrous pericardium**

- protects heart
- anchors heart
- prevents overfilling of heart

b) **serous pericardium**:
parietal & visceral (**epicardium)** layers



fluid-filled **pericardial cavity**

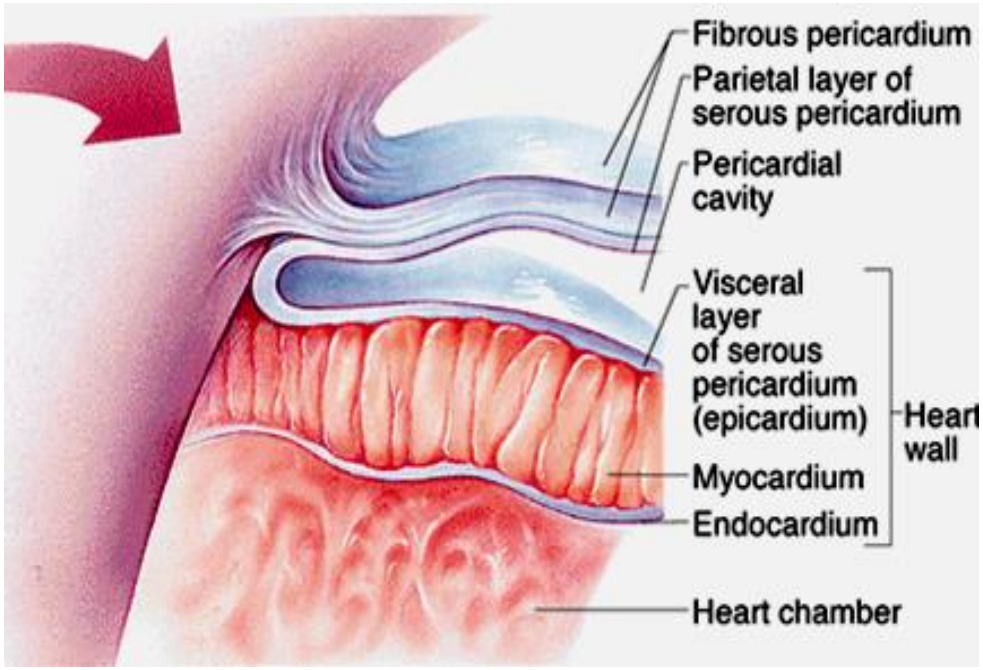
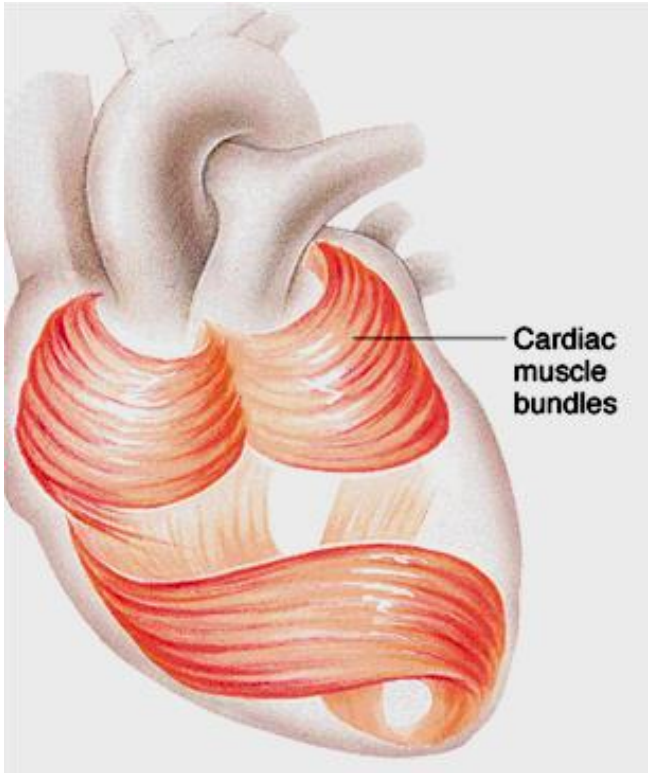


Fig. 18.3

2. Myocardium

- cardiac muscle = bulk of heart
- branching cardiac muscle cells arranged into bundles and the connective tissue wrappings of these bundles:
  - » reinforce myocardium internally & anchor cardiac muscle fibers
  - » provide additional support for great vessels & valves
  - » direct spread of action potentials across heart to specific pathways



3. Endocardium

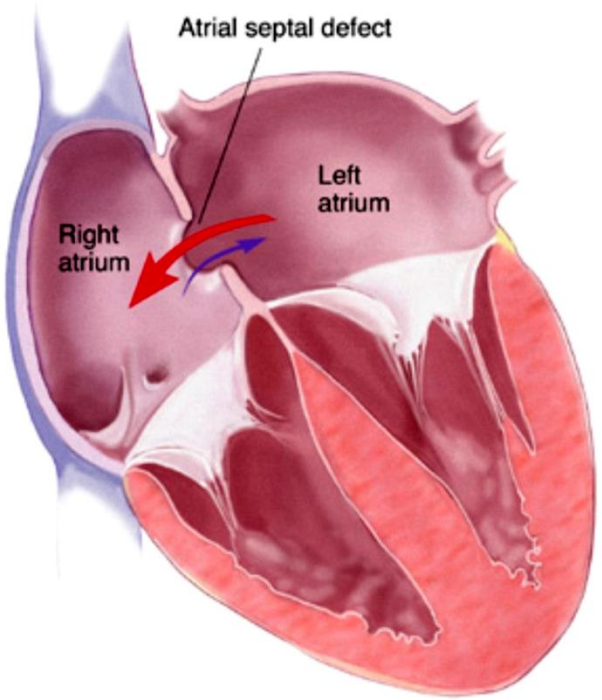
» layer of **endothelium** + CT layer on inner myocardial surface

» continuous with **endothelium** of vessels leaving & entering heart

Fig. 18.4

4.2.1.3 Locate the following on diagrams of the heart: chambers, great vessels, coronary circulation, internal muscles, etc

- 2 atria & 2 ventricles; *what are the **interatrial** & **interventricular** septa?*
- 2 exterior grooves: **coronary sulcus** (**atrioventricular groove**) & **anterior-posterior interventricular sulcus**



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Anterior view

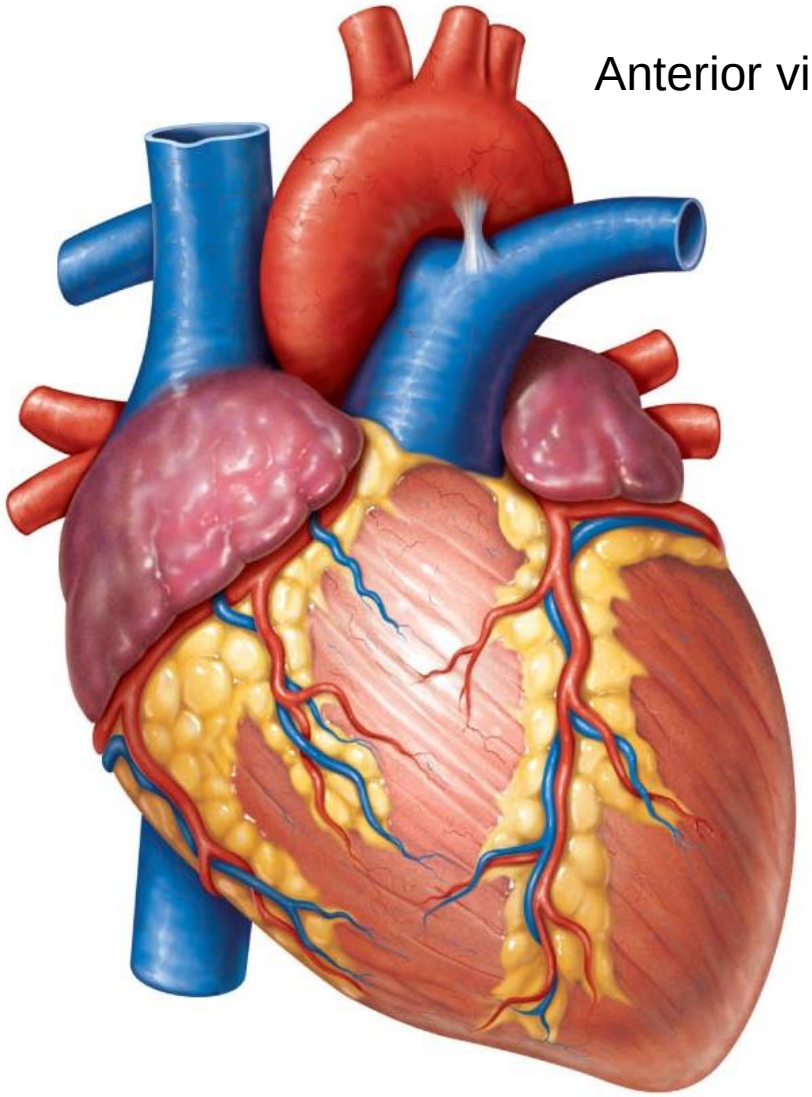


Fig. 18.5b

1. Atria: Receiving Chambers

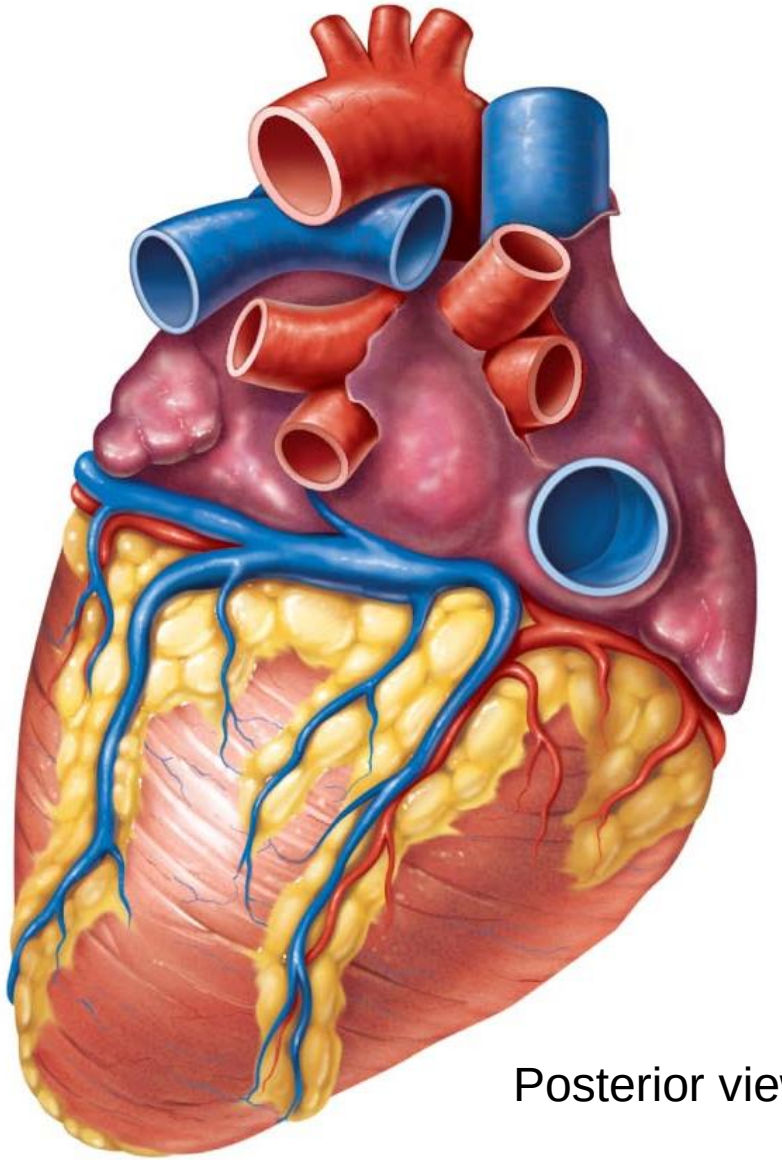
- small, thin-walled - need only convey blood to ventricles
- **deoxygenated**, systemic blood enters right atrium via:

(i) **superior vena cava** - systemic from ??

(ii) **inferior vena cava** - systemic from ??

(iii) **coronary sinus** - from myocardium

oxygenated blood to left atrium via 4 pulmonary veins



Posterior view

Fig. 18.5d

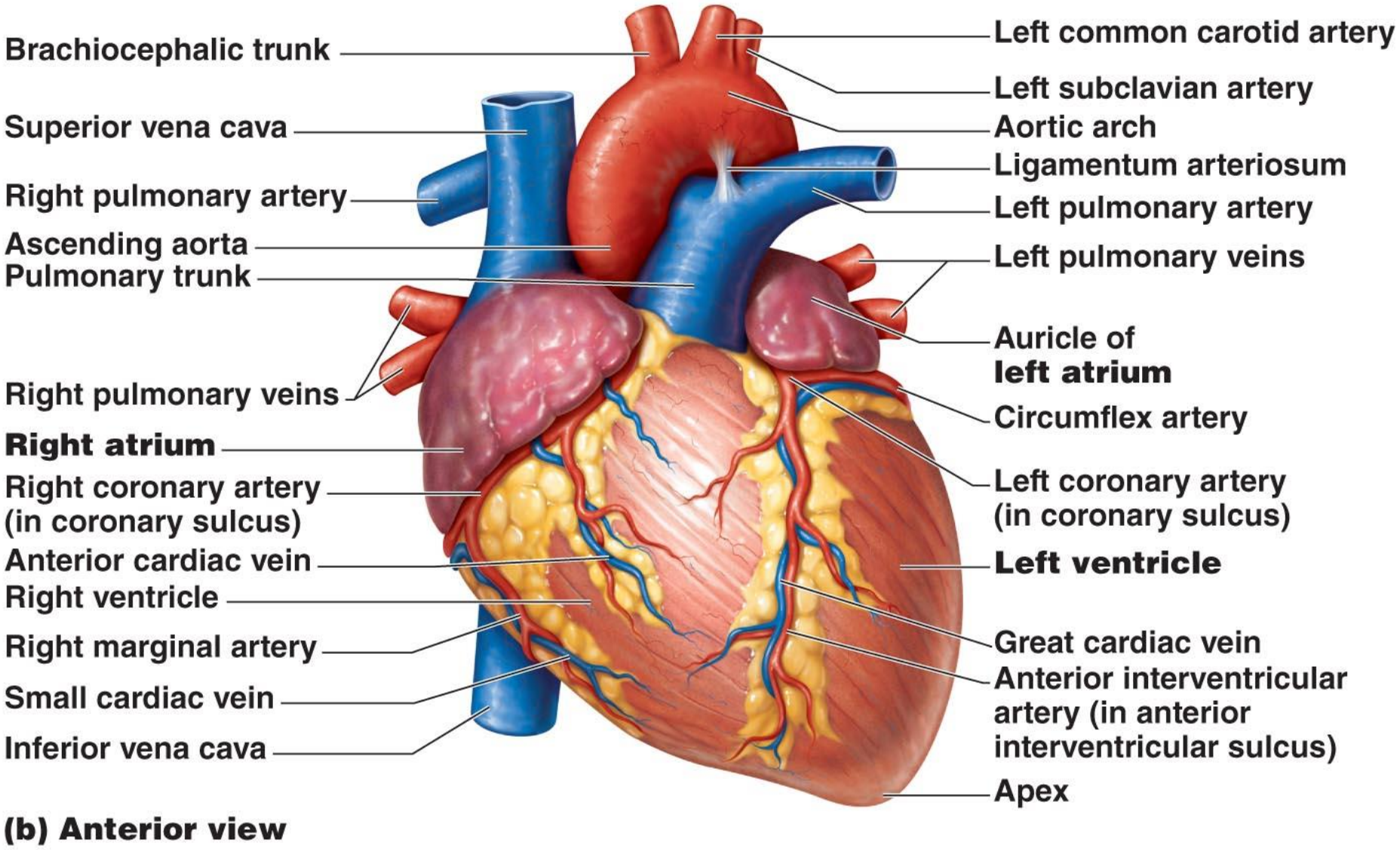
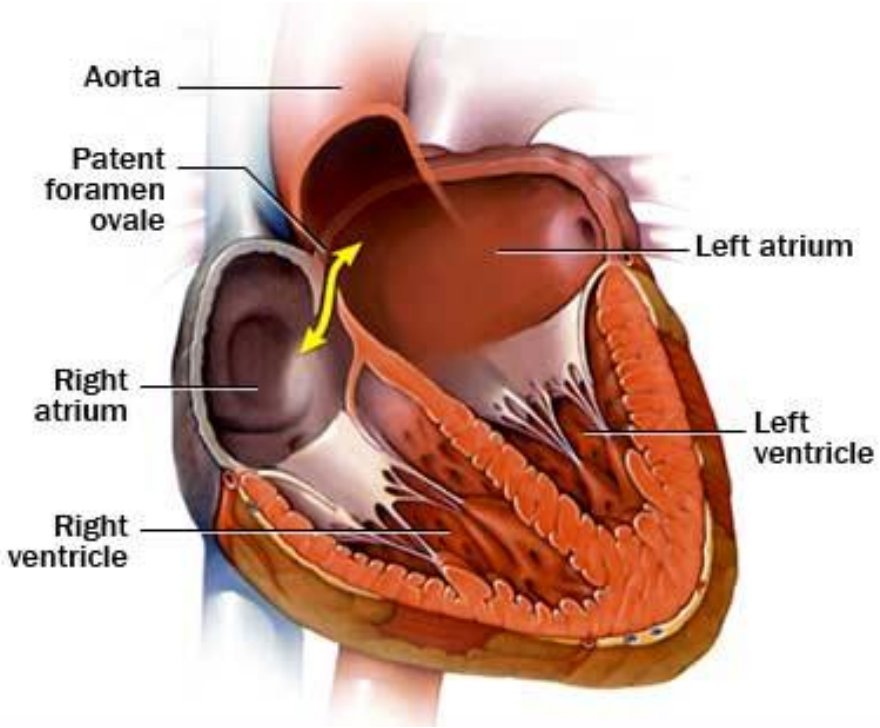


Fig. 18.5b – Gross anatomy of the heart

pectinate muscles

fossa ovalis

foramen ovale



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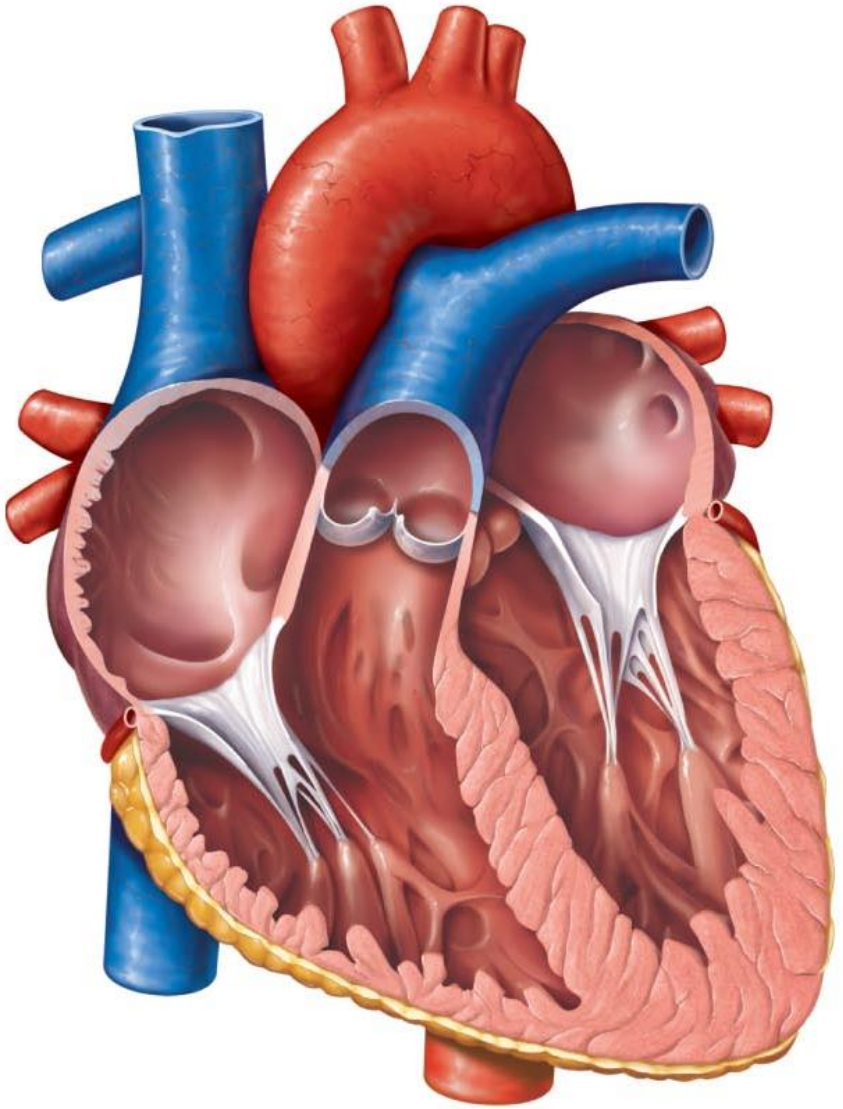
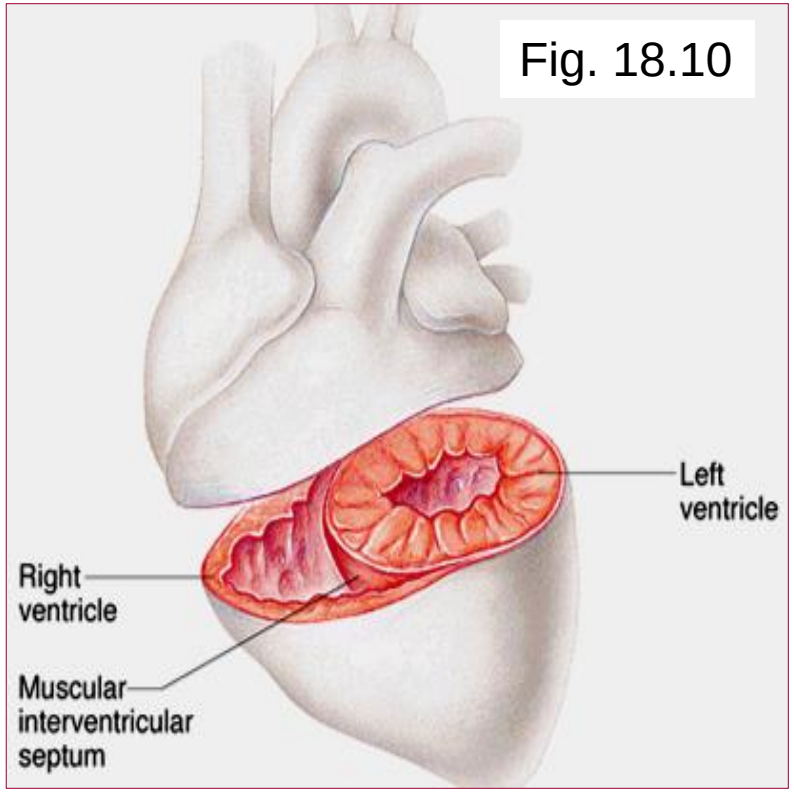
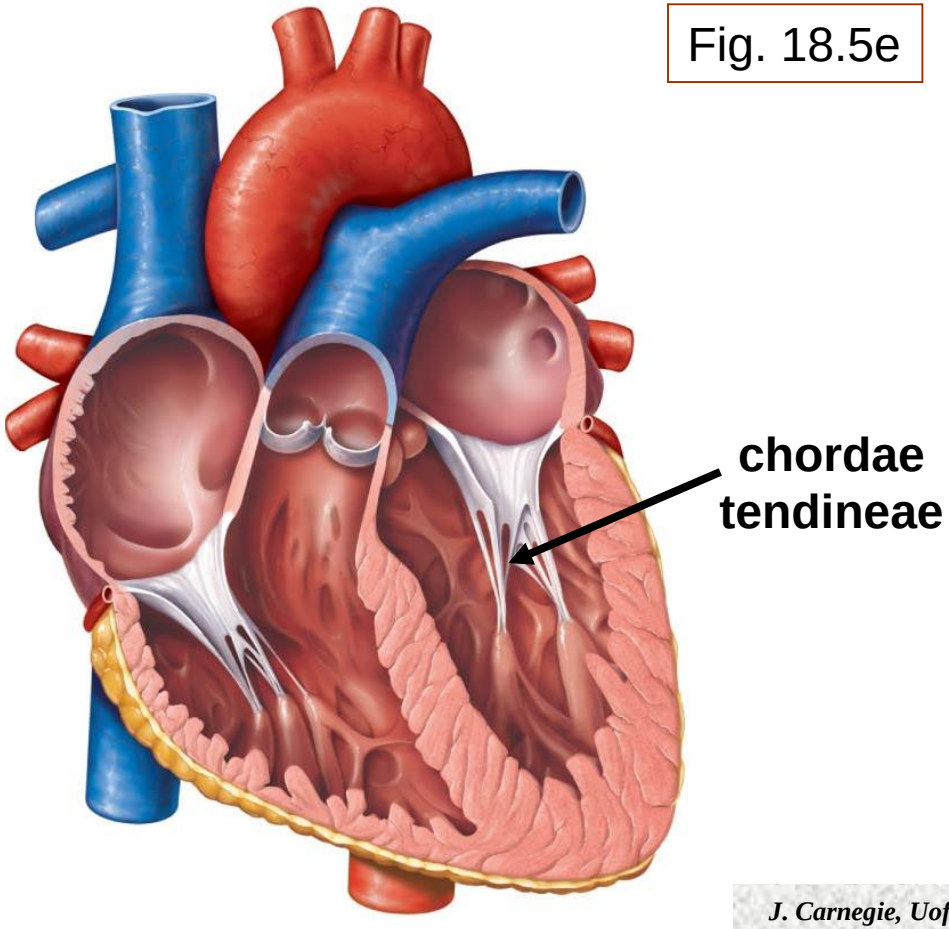


Fig. 18.5e

2. Ventricles: Discharging Chambers

- pumps of the heart; walls much thicker (esp: left ventricle)
  - (i) **right ventricle** pumps blood to pulmonary trunk
  - (ii) **left ventricle** pumps blood to aorta
- internal walls have muscle bundles: **trabeculae carneae**, **papillary muscles** (valve function)





3. Heart Valves

- blood flow is **unidirectional**; enforced by **4** heart valves
- realize importance of **chordae tendineae** & **papillary muscles**

1. Atrioventricular valves:

- paired, between ….. & …..

(i) **tricuspid valve** = right atrium to right ventricle

(ii) **mitral (bicuspid) valve** = left atrium to left ventricle

2. Semilunar valves:

- paired, from ventricles to either pulmonary or systemic circuits

(i) **pulmonary valve** = right ventricle to pulmonary artery

(ii) **aortic valve** = left ventricle to aorta

valvular **insufficiency** (incompetent valves) vs **stenosis**

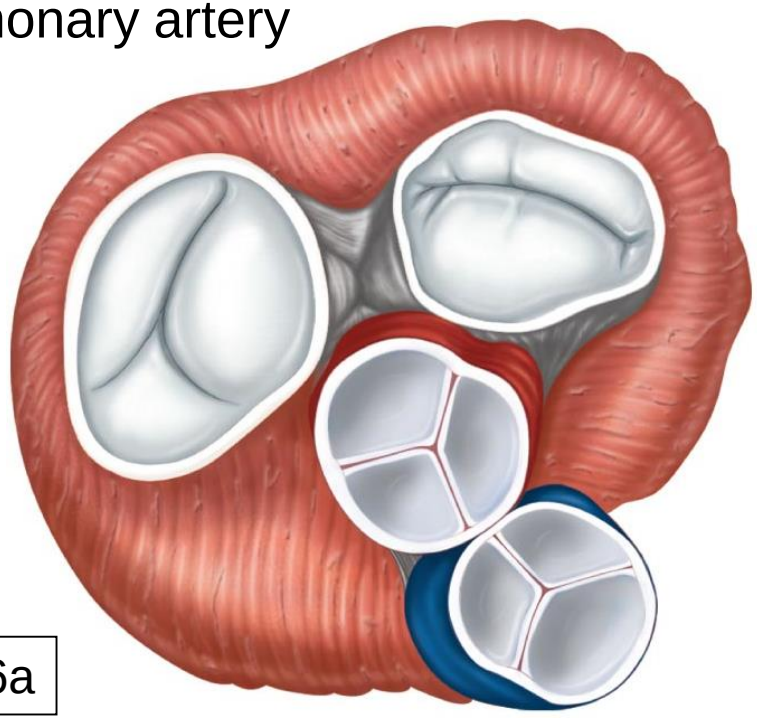
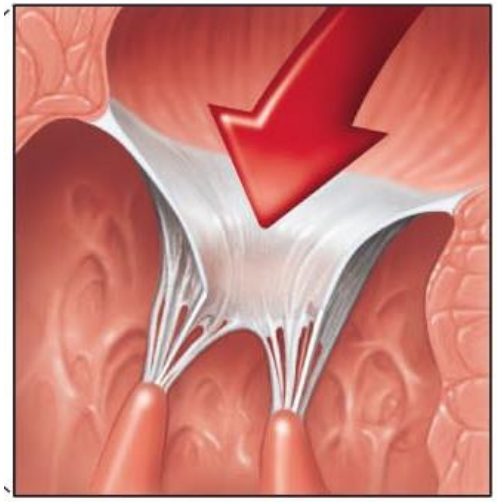
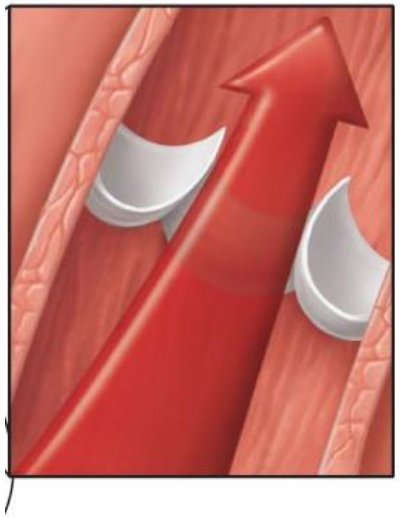


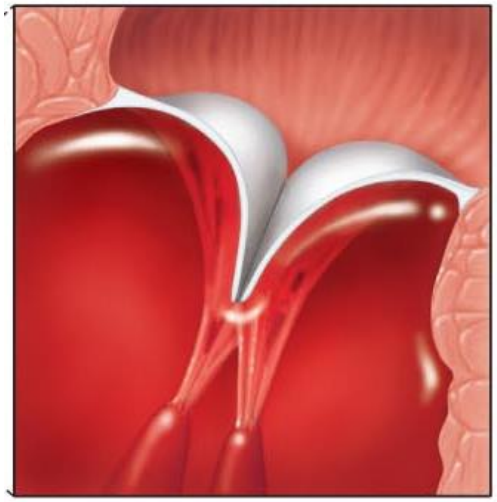
Fig. 18.6a

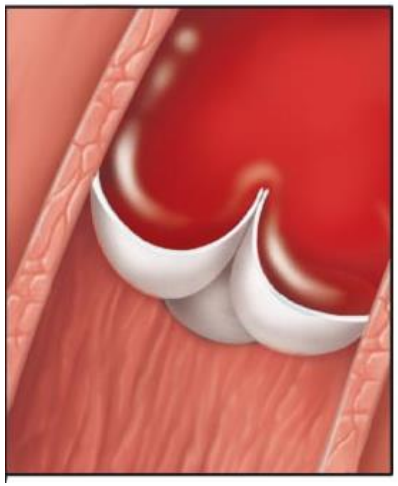


AV valves



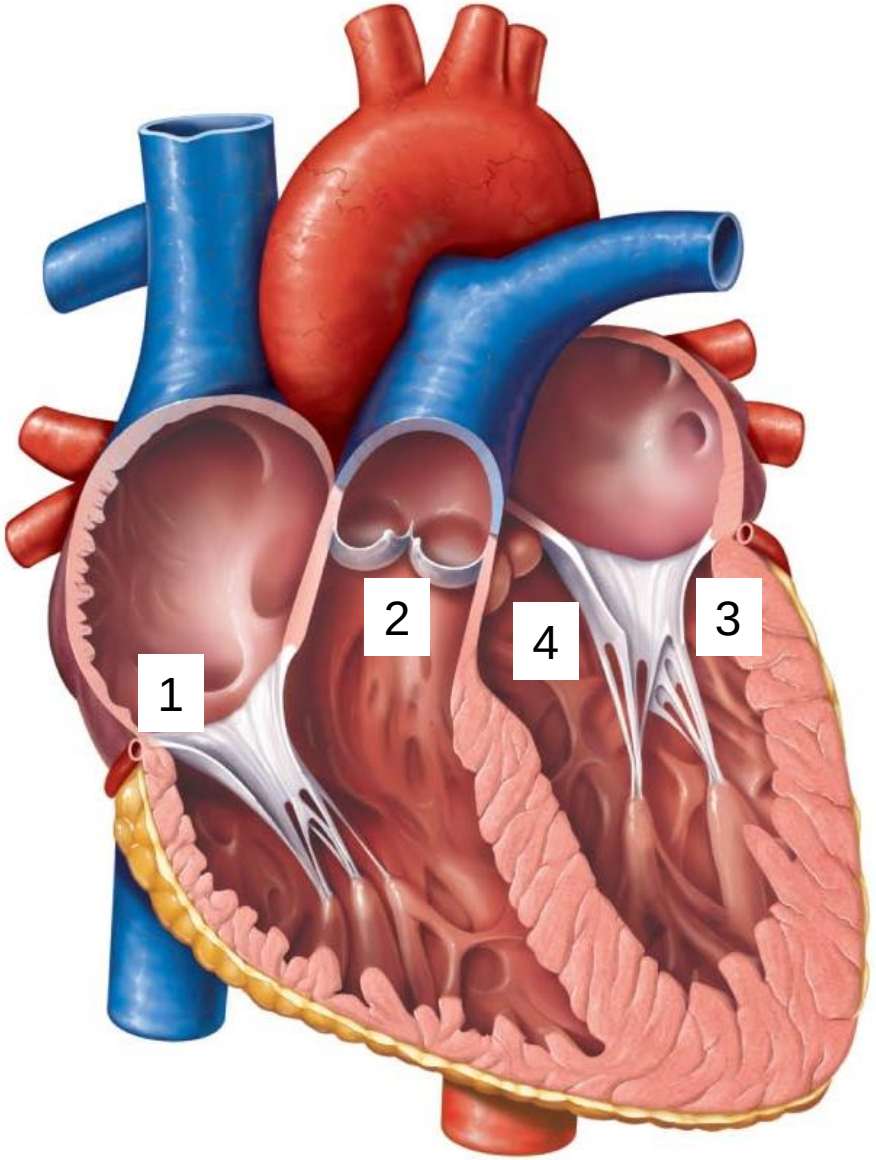
Semilunar valves





Figs. 18.7 & 18.8

4.2.2.1 Trace the pathway followed by a RBC from its entry into the heart to its exit; include all of the valves in the order encountered



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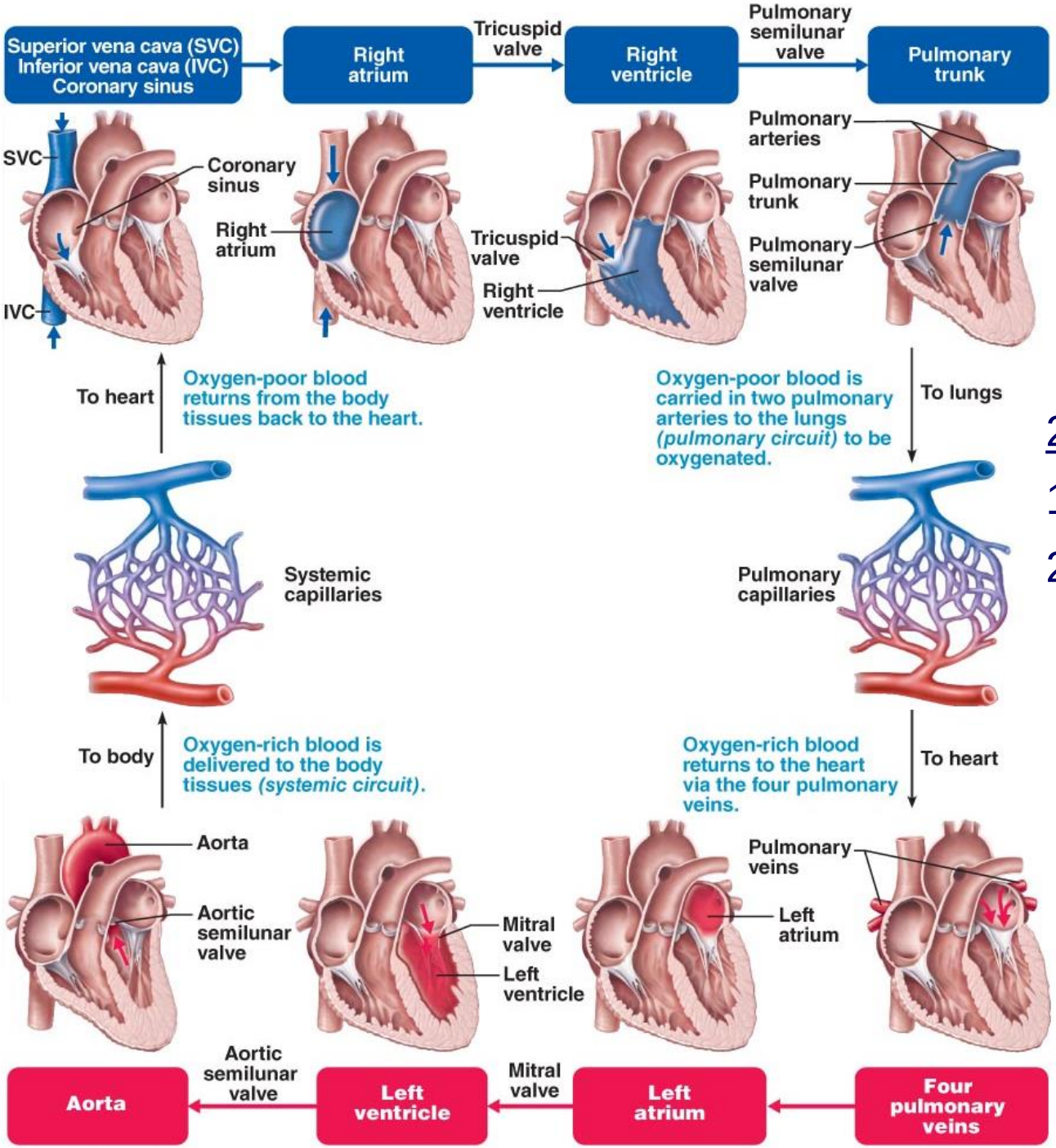


2 side-by-side pumps:

1. Pulmonary circuit
2. Systemic circuit

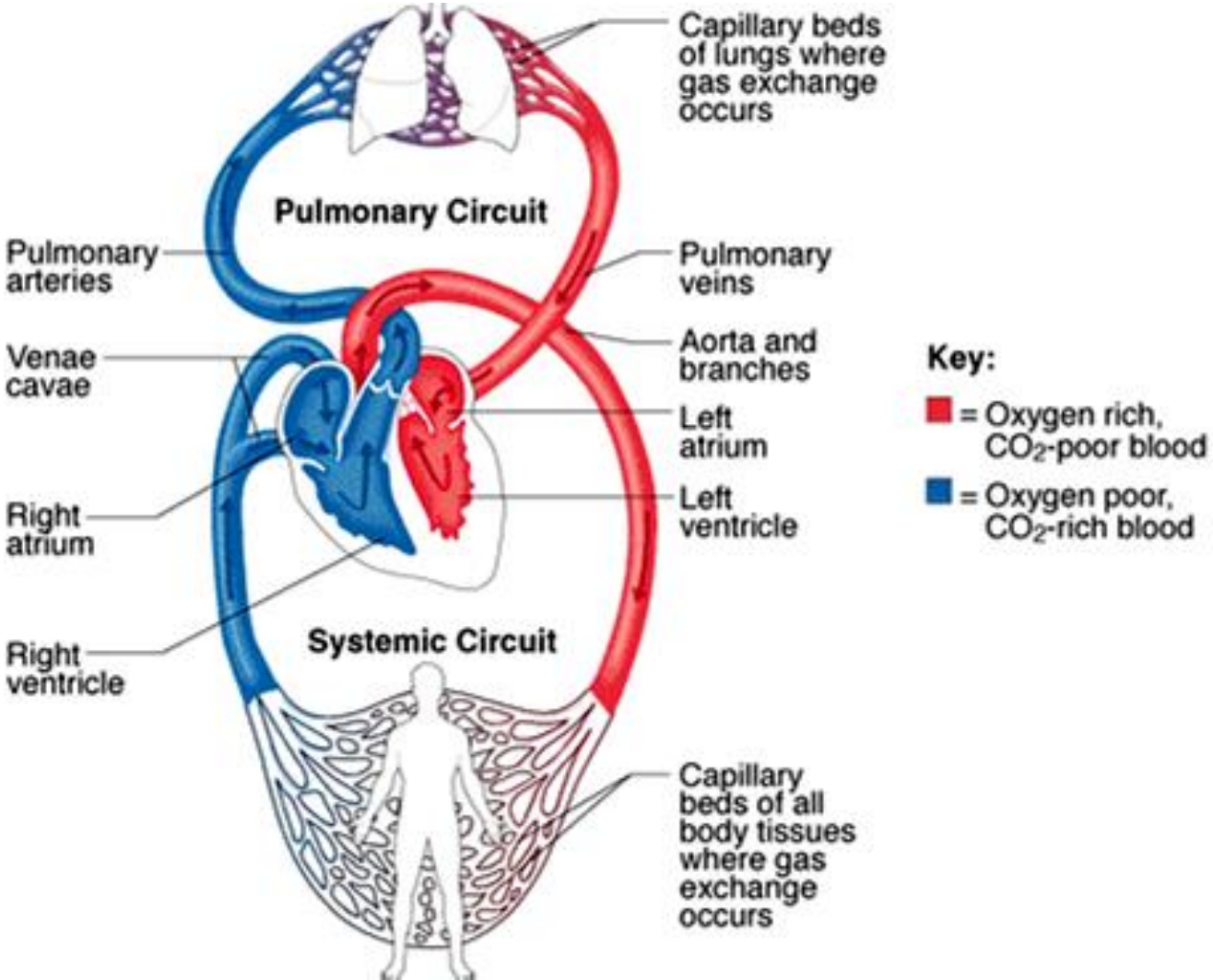
4.2.2.2 differentiate between the pathways of the pulmonary and systemic circuits: oxygenation of the blood, workload, structure of ventricular wall

equal volumes pumped into **pulmonary** & **systemic** circuits; but 2 ventricles have **unequal** workloads

(1) **pulmonary circuit** (rt ventricle): short, **low-pressure** circulation

(2) **systemic circuit** (lt ventricle):long pathway with **5X resistance**

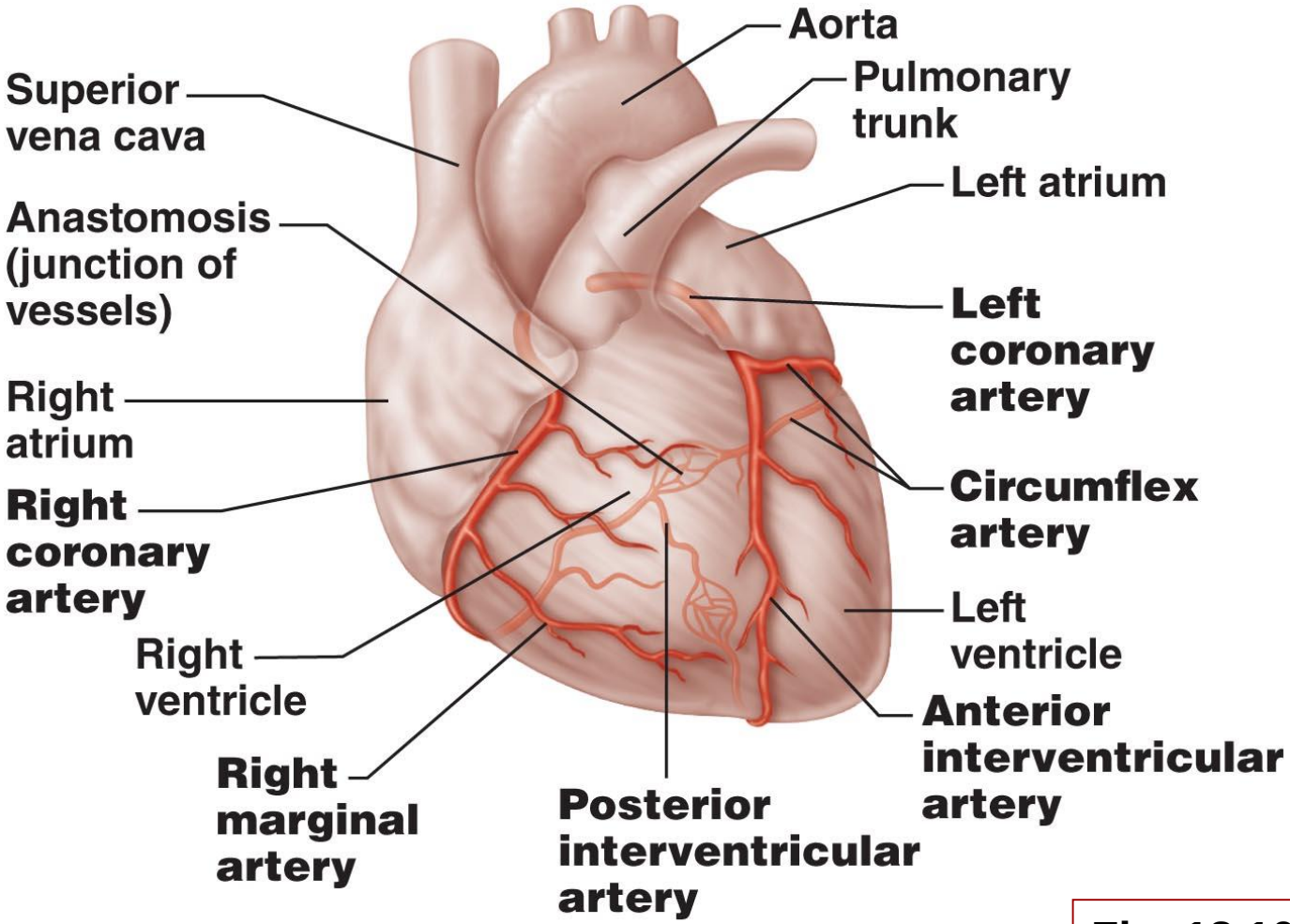
Fig. 18.5 (8th edition)



Walls of left ventricle 3X thicker than those of right.

4.2.3 Describe the coronary circulation

- shortest, but one of the most important, circulations in body
- right & left **coronary arteries** from base of aorta; encircle heart in **coronary sulcus** (**atrioventricular groove**)



(a) The major coronary arteries

Fig. 18.10a

- many anastomoses, which provide alternate routes for nourishment if a given artery begins to be occluded - **but** total occlusion means ????
  (1) actively deliver blood when heart is relaxed
  (2) largely ineffective when ventricles contracting because??
- heart ~1/200 of body but requires ~1/20 of blood supply (esp. left ventricle)

Coronary venous supply:

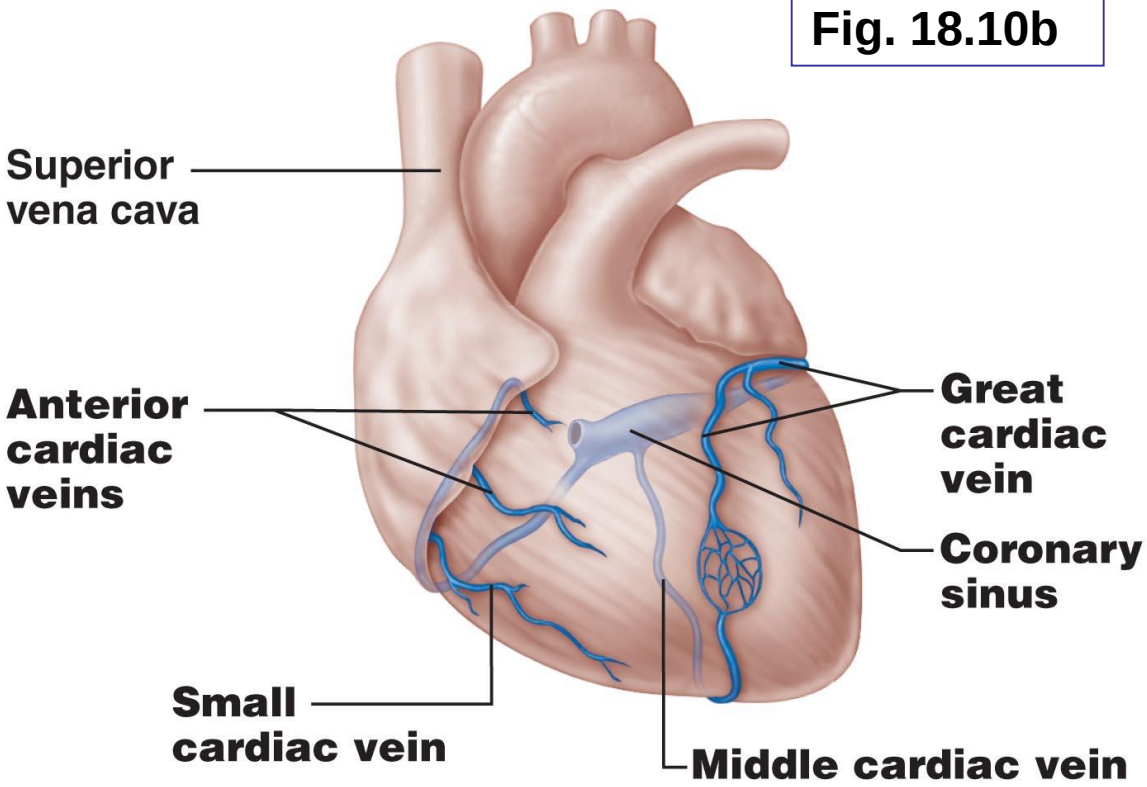
begin with capillaries:

Diseases of coronary vessels:

angina pectoris:

myocardial infarction:

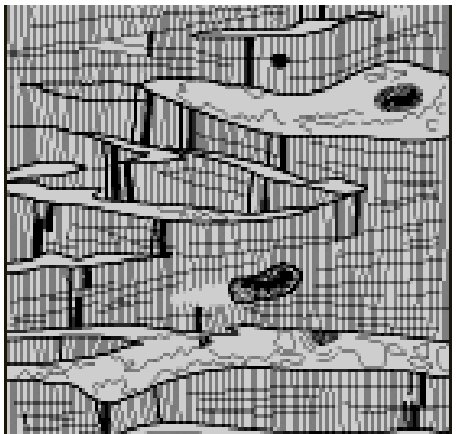
Fig. 18.10b

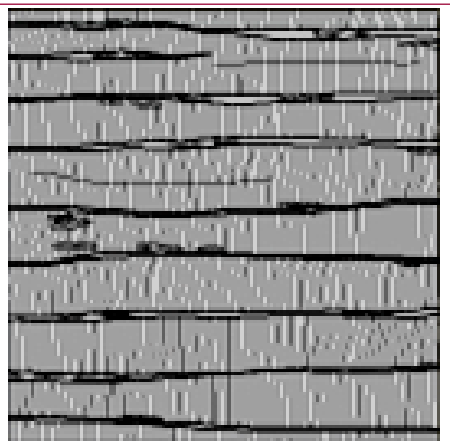


(b) The major cardiac veins

4.2.4 Compare the physiological properties of cardiac muscle fibers with those of skeletal muscle cells

Cardiac Muscle	Skeletal Muscle
shorter, fatter	longer, cylindrical
single or double nuclei	multinucleate
intercalated disks (desmosomes, gap junctions) = interdependence	structural independence; motor unit grouping
functional syncytium	functional independence
20-40% of volume is mitochondria	2-5% of volume is mitochondria
myofibril diameters vary & much branching gives indistinct striations	uniform myofibrils give distinct striations
fewer T-tubules; less elaborate Ca^{++} delivery	many T-tubules; complex Ca^{++} delivery
less developed sarcoplasmic reticulum	complex sarcoplasmic reticulum
almost exclusively aerobic metabolism	readily uses anaerobic metabolism
abundance of fuel type determines fuel used	intensity determines fuel type used





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4.2.4.2 describe intercalated discs and relate 2 aspects of their structure to the support of cardiac function

(i) **desmosomes** for strong cell-cell adhesion during contraction

(ii) **gap junctions** for electric coupling »» **functional syncytium**

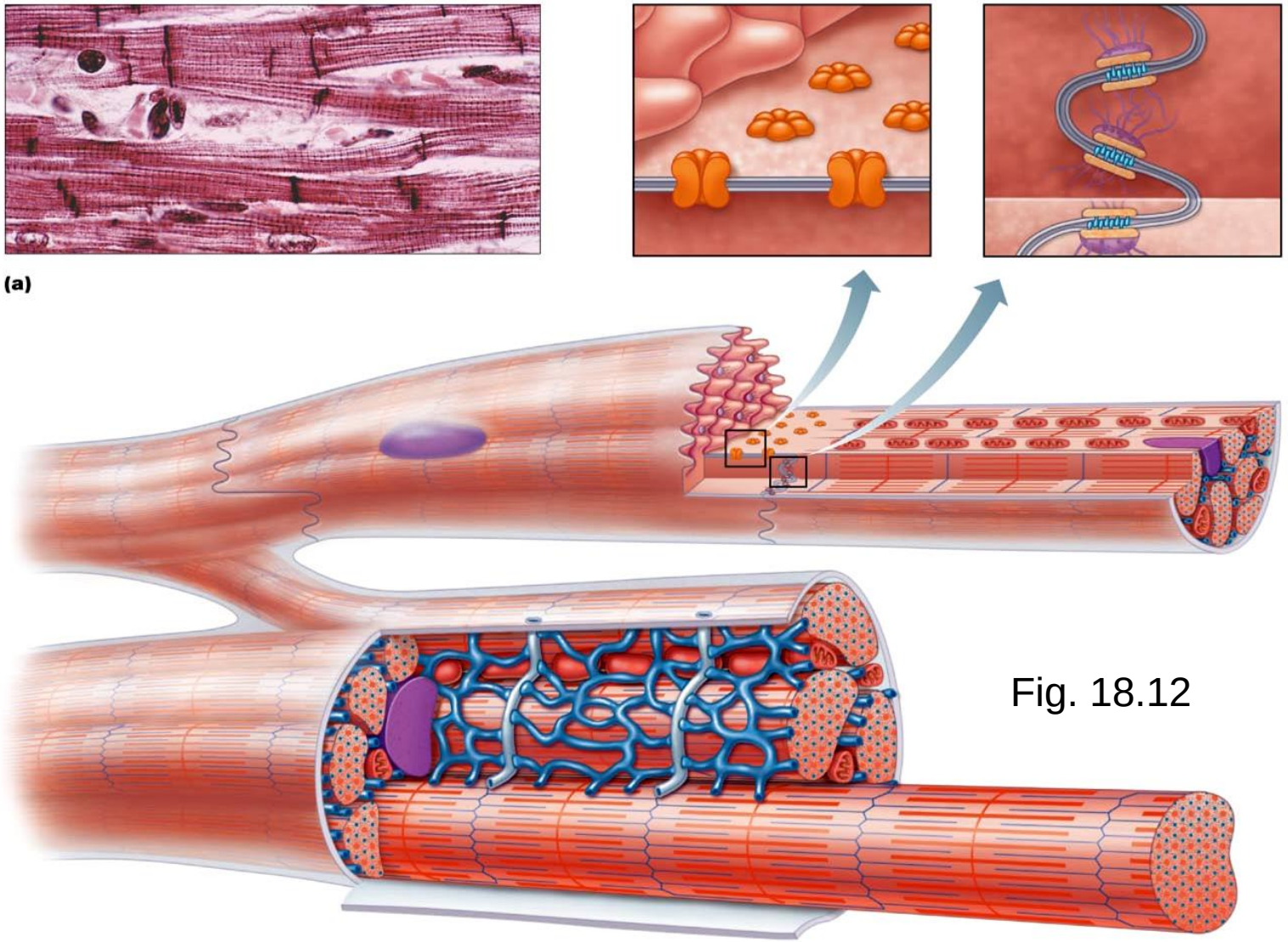


Fig. 18.12

4.2.5 Compare the electrical properties of contractile cardiac muscle cells with those of autorhythmic cardiac muscle cells

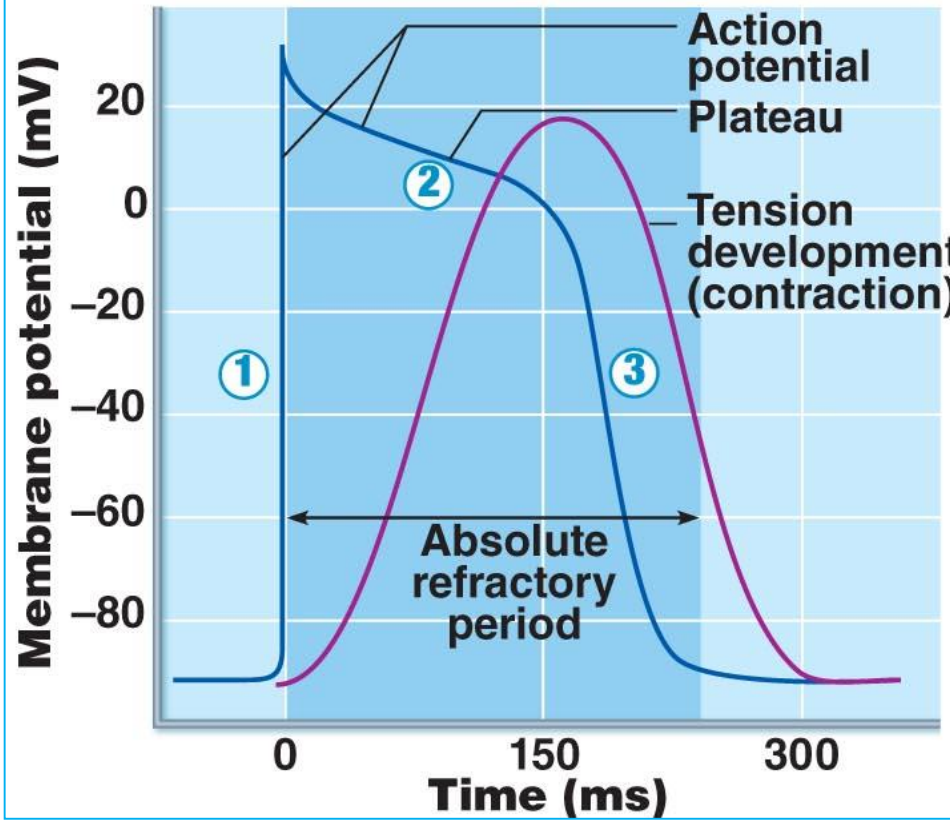
- heart contraction stimulated by action potentials: ***action potential*** = signal; *muscle twitch* = response

4.2.5.2 special characteristics of cardiac muscle cells

1) **Stimulation: autorhythmicity** of (1%) cardiac cells
2) The heart contracts as a **unit** *(all or none)*
3) Influx of Ca^{2+} from **ECF** triggers Ca^{2+} release from SR
4) **Absolute refractory period:** 250 msec vs 1-2 msec for action potential stimulating skeletal muscle – *Why is this important?*
5) Heart relies almost exclusively on **aerobic respiration**

Figure on next slide shows action potential of ventricular Purkinje fibers*; total sequence = about 300 msec.*

Fig. 18.15: Action potential of contractile cardiac muscle cells



① **Depolarization** is due to Na^+ influx through fast voltage-gated Na^+ channels. A positive feedback cycle rapidly opens many Na^+ channels, reversing the membrane potential. Channel inactivation ends this phase.

② **Plateau phase** is due to Ca^{2+} influx through slow Ca^{2+} channels. This keeps the cell depolarized because few K^+ channels are open.

③ **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its resting voltage.

- absolute refractory period almost = muscle twitch ➢➢ allows heart to fill again
- heart needs to be stimulated in just one location; whole organ responds
- activation of contraction basically as in skeletal muscle:

4.2.5.3 define "autorhythmic cell; describe cardiac autorhythmic cell properties that allow them to spontaneously depolarize

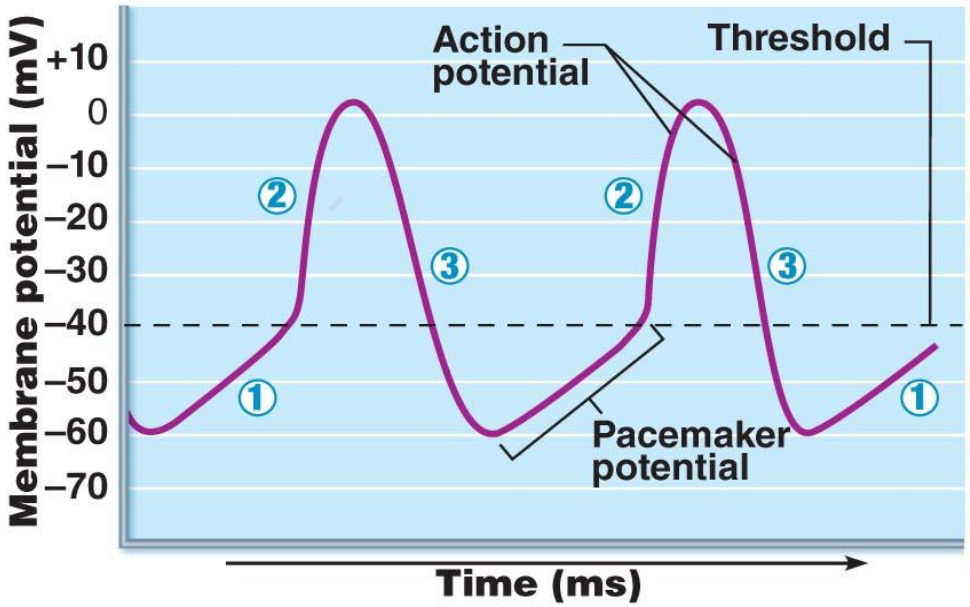
4.2.5.4 define "sinus rhythm" indicate why SA node is the pacemaker

- **pacemaker** = **SA** node because: …….
- **sinus rhythm** determines heart rate
- **autorhythmic** cells have **unstable** resting

pacemaker potentials → action potentials

- in **autorhythmic cells**, action potential due to:

Fig. 18.12



① **Pacemaker potential** This slow depolarization is due to both opening of Na^+ channels and closing of K^+ channels. Notice that the membrane potential is never a flat line.

② **Depolarization** The action potential begins when the pacemaker potential reaches threshold. Depolarization is due to Ca^{2+} influx through Ca^{2+} channels.

③ **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its most negative voltage.

4.2.6 Explain how the intrinsic conduction system of the heart allows it to function as a pump

AP generated by SA node passes to:

(2) AV node

(3) (short delay), AV bundle

(4) right & left bundle branches

(5) Purkinje fibers

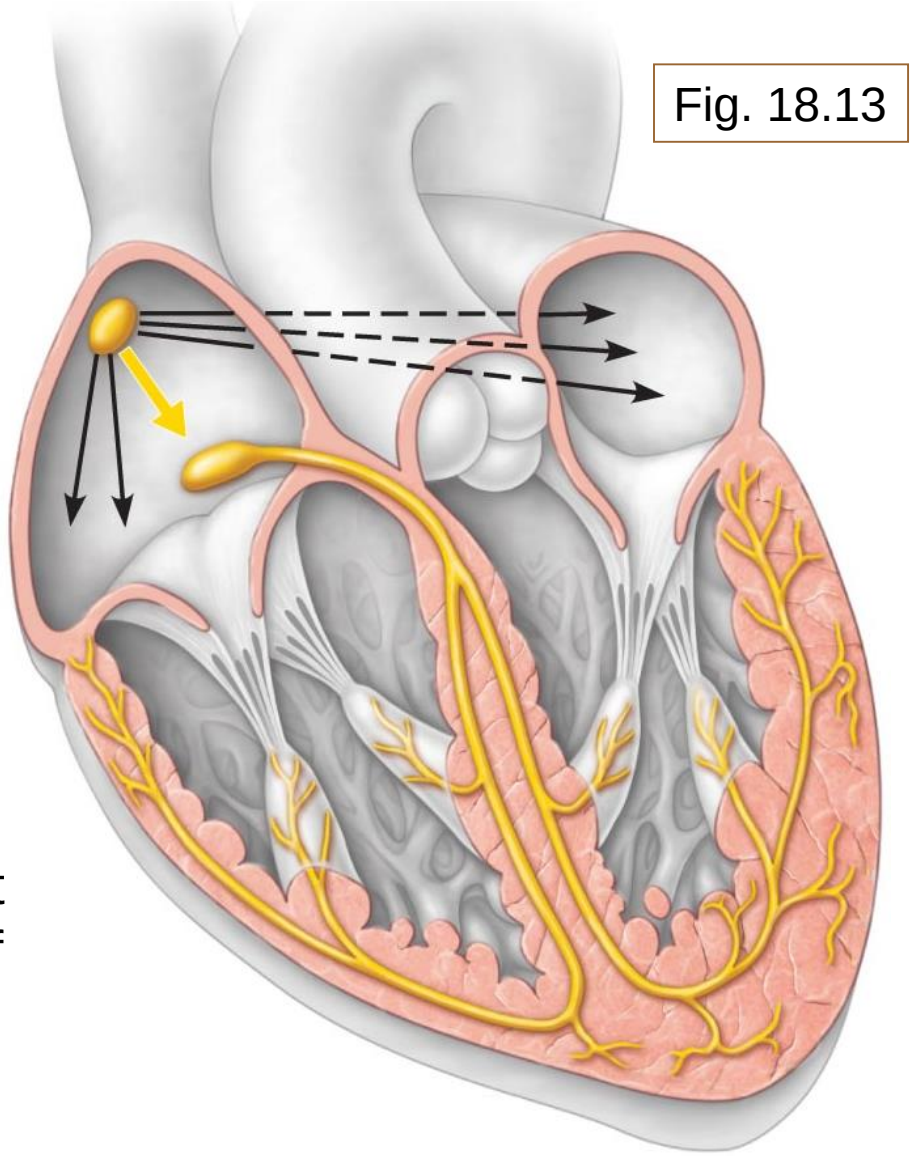
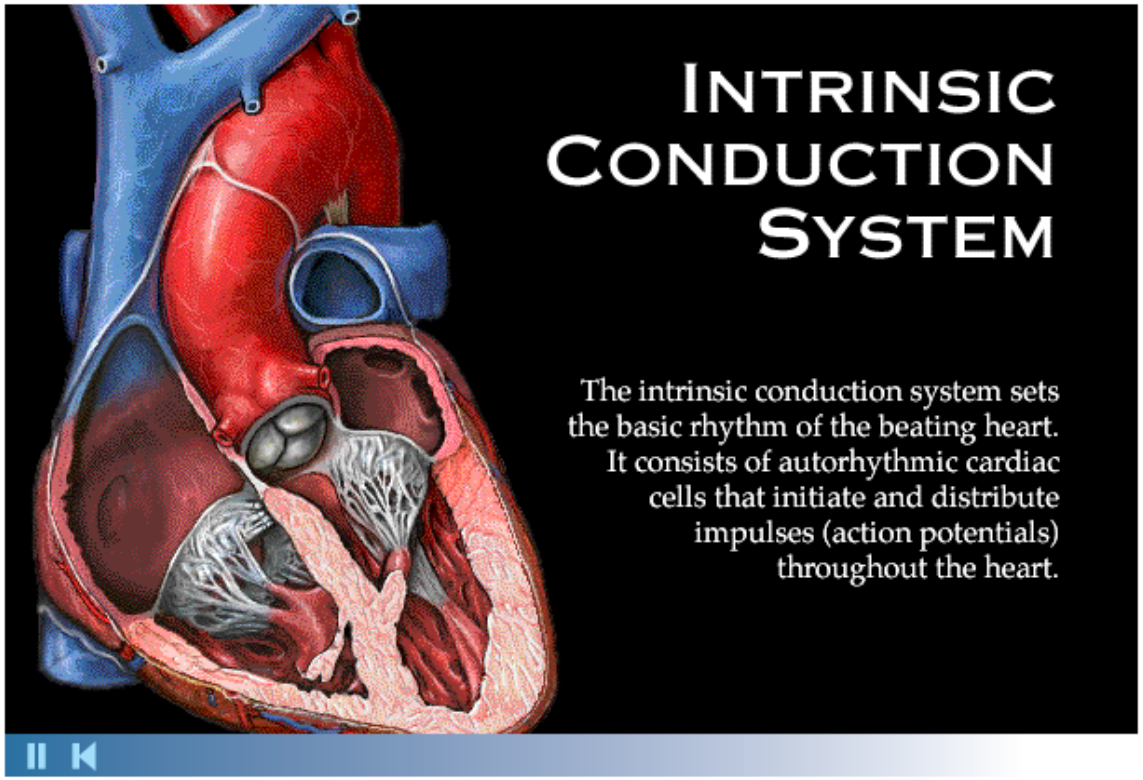


Fig. 18.13

bottleneck is from atria to ventricles

Why??

0.22 sec (220 msec) from initiation at SA node to depolarization of last of ventricular cells



Interactive physiology CD or Mastering – Chapter 18 under Heart Physiology

4.2.6.3 delineate the extrinsic innervation of the heart and contrast the influences of PNS and SNS on heart rate

Rate of SA node depolarization regulated by autonomic nervous system:

(1) **parasympathetic ns: decreases** diastolic depolarization rate

(2) **sympathetic ns: increases** depolarization & repolarization rates

Under resting conditions, **tonic** parasympathetic output to have a ***dampening*** effect on heart rate

What is bradycardia??

What is tachycardia??

What is sinus rhythm??

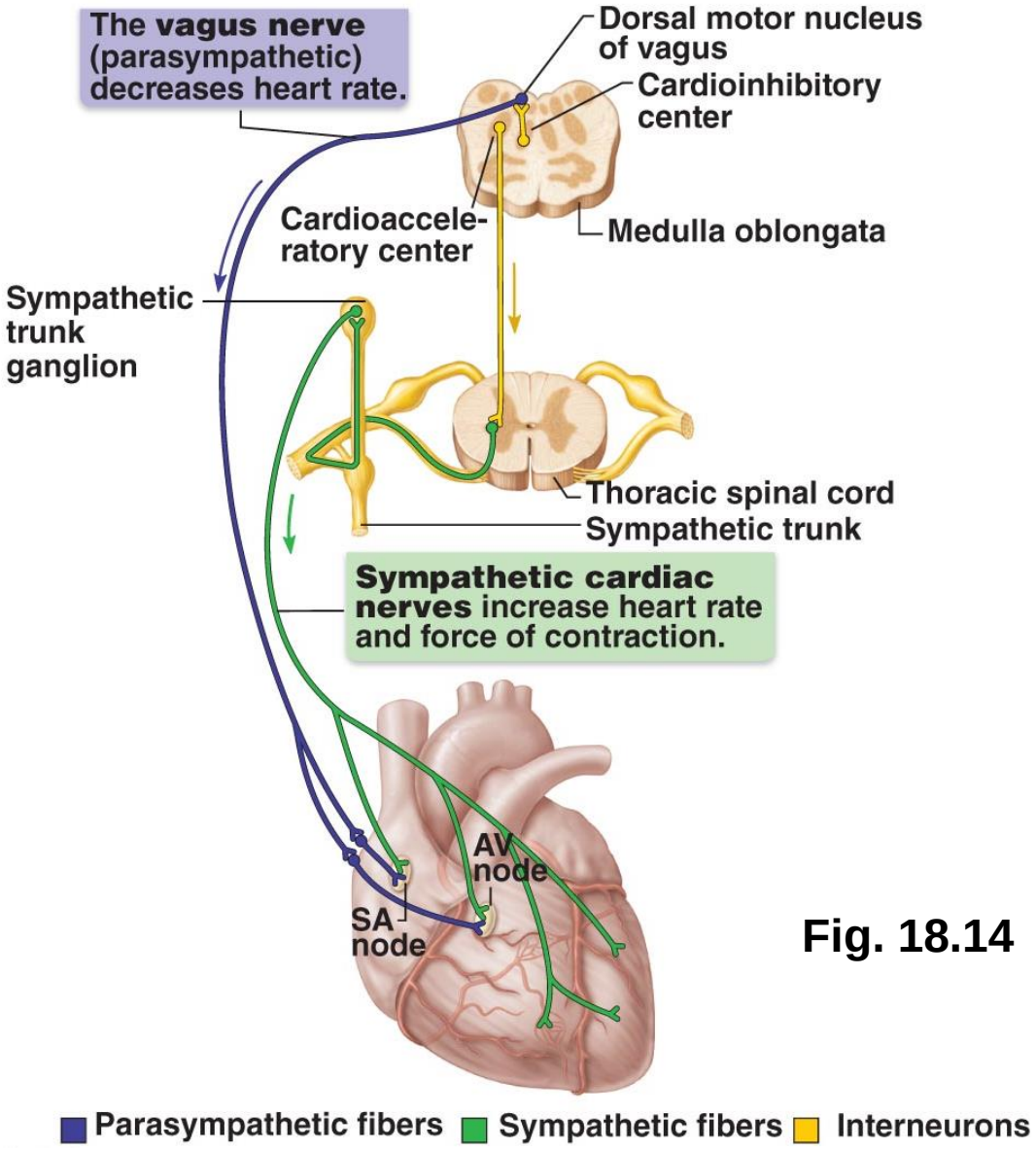


Fig. 18.14


The nodal cells of the heart, unlike cardiac contractile muscle fibers, have an intrinsic ability to depolarize _____ . This reflects their unstable _____ which drifts slowly toward threshold for firing, that is _____ . These spontaneously changing membrane potentials, called _____, are largely (but not entirely) due to reduced membrane permeability to _____ .

Ultimately, when threshold is reached, gated channels open, allowing extracellular _____ to rush into the cells and reverse the membrane potential.

From the atrium through the tricuspid valve to the _____, through the _____ valve to the pulmonary trunk to the right and left _____, to the capillary beds of the _____, to the _____, to the _____ of the heart through the _____ valve, to the _____ through the_____ semilunar valve to the _____, to the systemic arteries, to the _____ of the body tissues, to the systemic veins, to the _____ and _____, which enter the right atrium of the heart.


4.2.7 Explain what is an ECG tracing and the nature of the information it is encoding

Electrocardiogram: records electrical changes during heart activity relies on conductile activity of body fluids

(i) **P-wave** = atrial depolarization

(ii) **QRS complex** = ventricular depolarization

(iii) **T-wave** = ventricular repolarization (*where is atrial repolarization?*)

*Note: ECG records only voltage (current flow) and time; shows only **electrical** events, but from these can deduce contractile events*

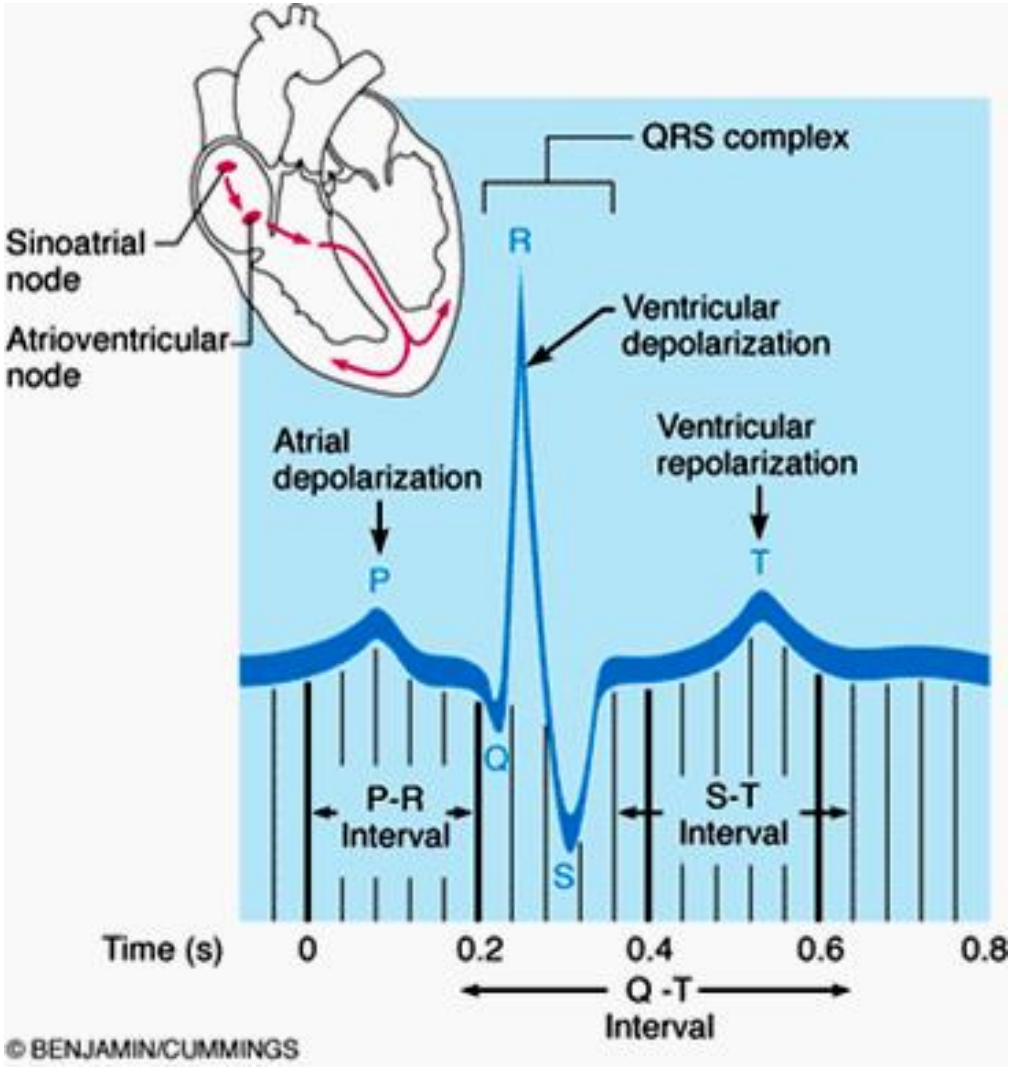
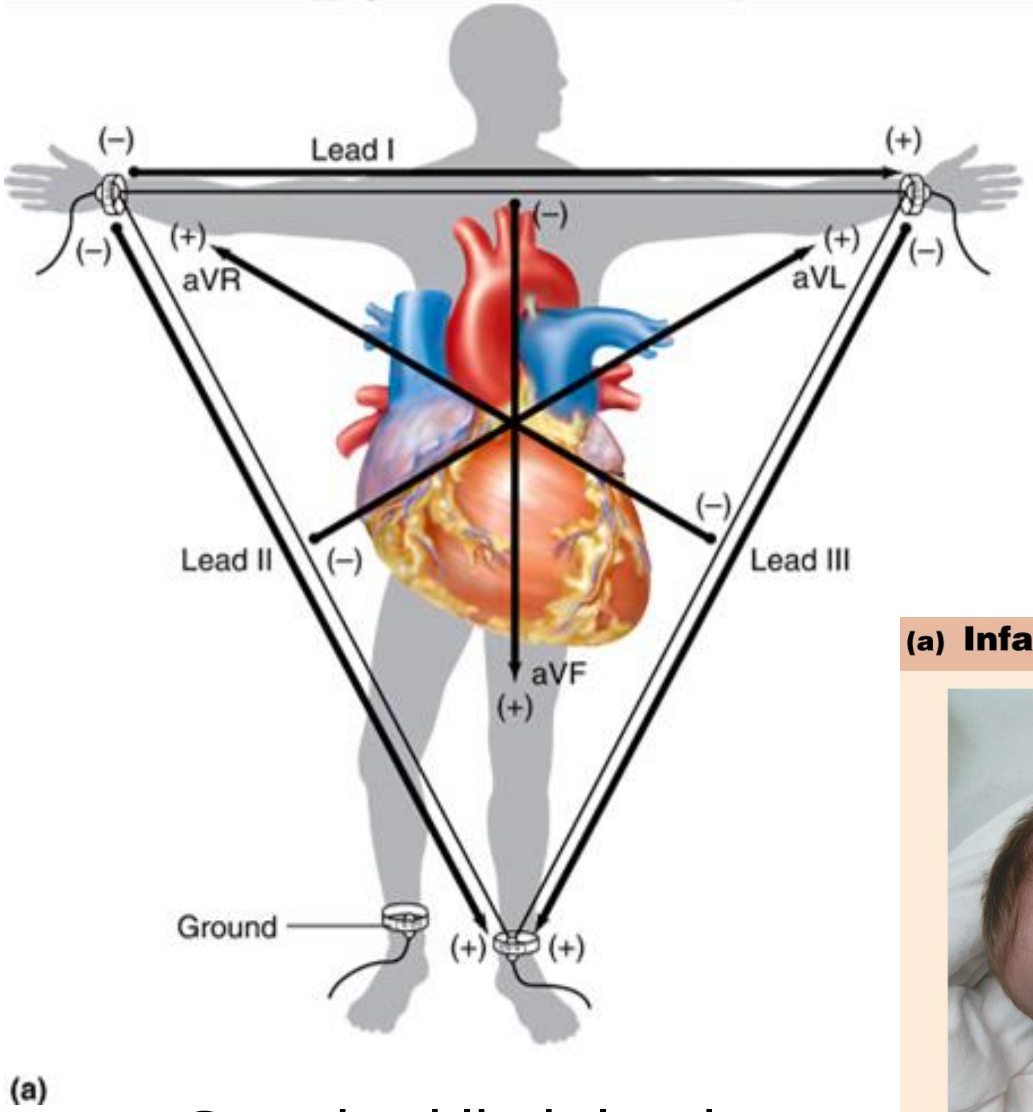


Fig.18.16



(a)

Standard limb leads for recording ECG tracings

(a) Infant undergoing an electrocardiogram (ECG)

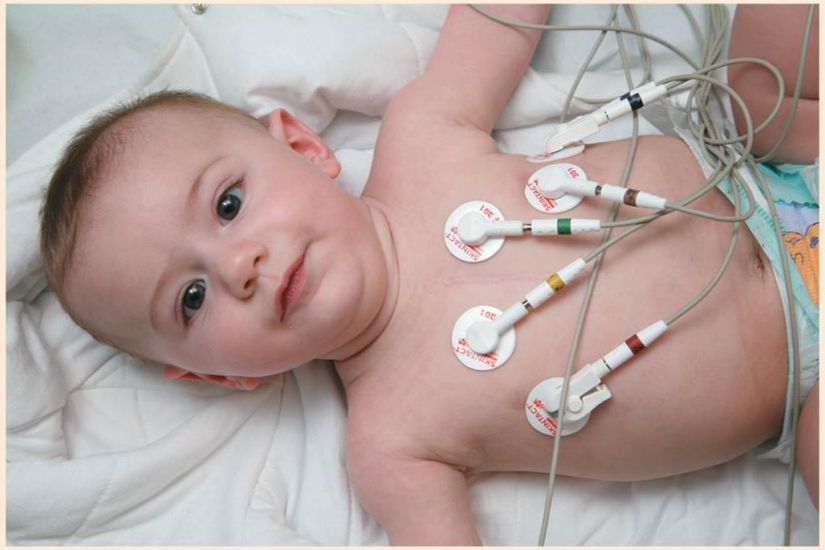


Fig. 18.18a

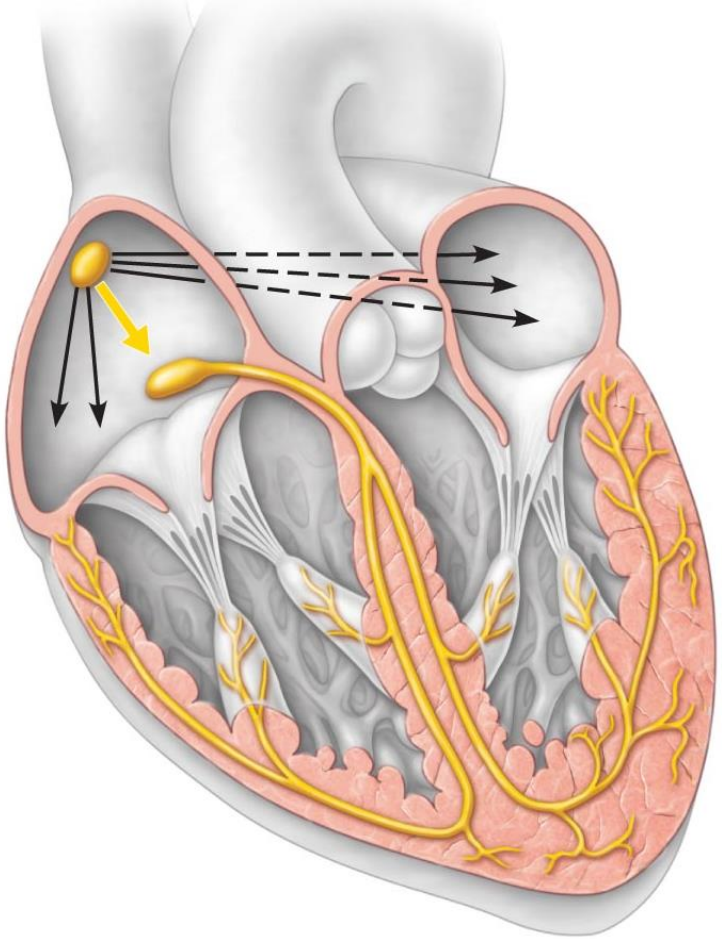
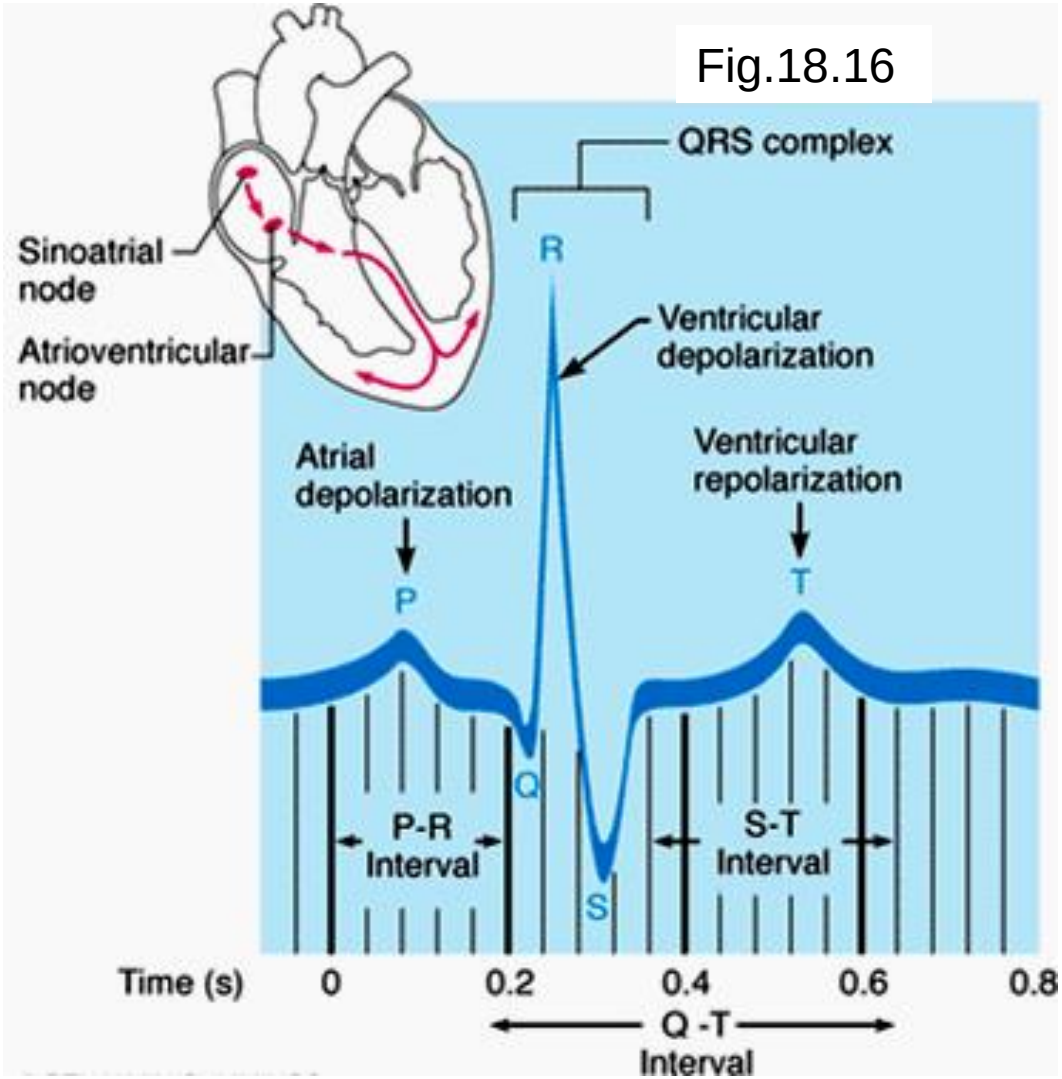


Fig.18.16


QRS complex
Sinoatrial node
Atrioventricular node
R
Ventricular depolarization
Atrial depolarization
Ventricular repolarization
P
T
Q
S
P-R Interval
S-T Interval
Time (s)
0
0.2
0.4
0.6
0.8
Q -T Interval

Abnormal Activation of the Heart

correct **sequence** of activation needed for the heart to function as a pump

Which of these ECG tracings demonstrates:

sinus rhythm

ventricular fibrillation

2nd degree heart block

nonfunctional SA node

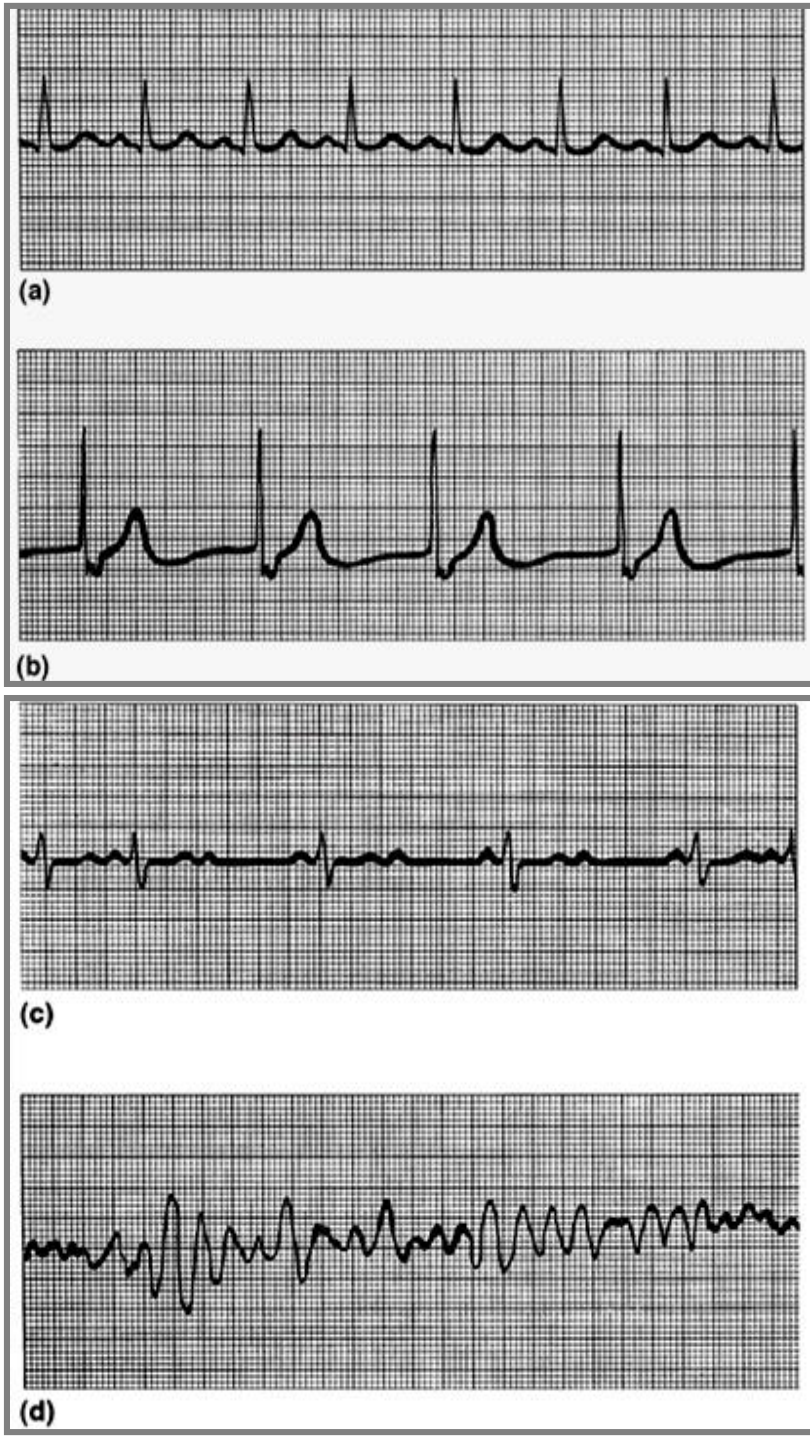


Fig. 18.18

4.2.8 Explain the events of each phase of the cardiac cycle

4.2.8.1 describe the pressure changes responsible for valve opening & closing & link these with resultant volume changes

systole: contraction of heart; pumping **OUT**

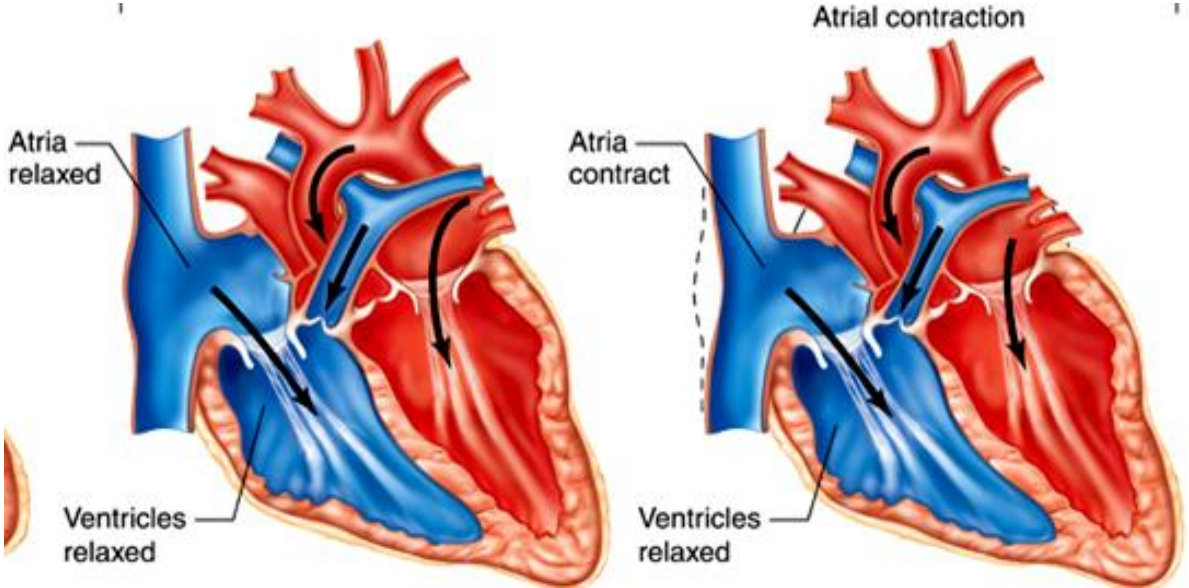
diastole: relaxation of heart; filling

cardiac cycle: **atrial** systole + diastole → **ventricular** systole + diastole

Start = mid-to-late diastole:

1. **Period of ventricular filling: mid-to-late diastole »** pressure low (but $P_{atria} > P_{ventricles}$), AV valves **OPEN**; SL valves **CLOSED**

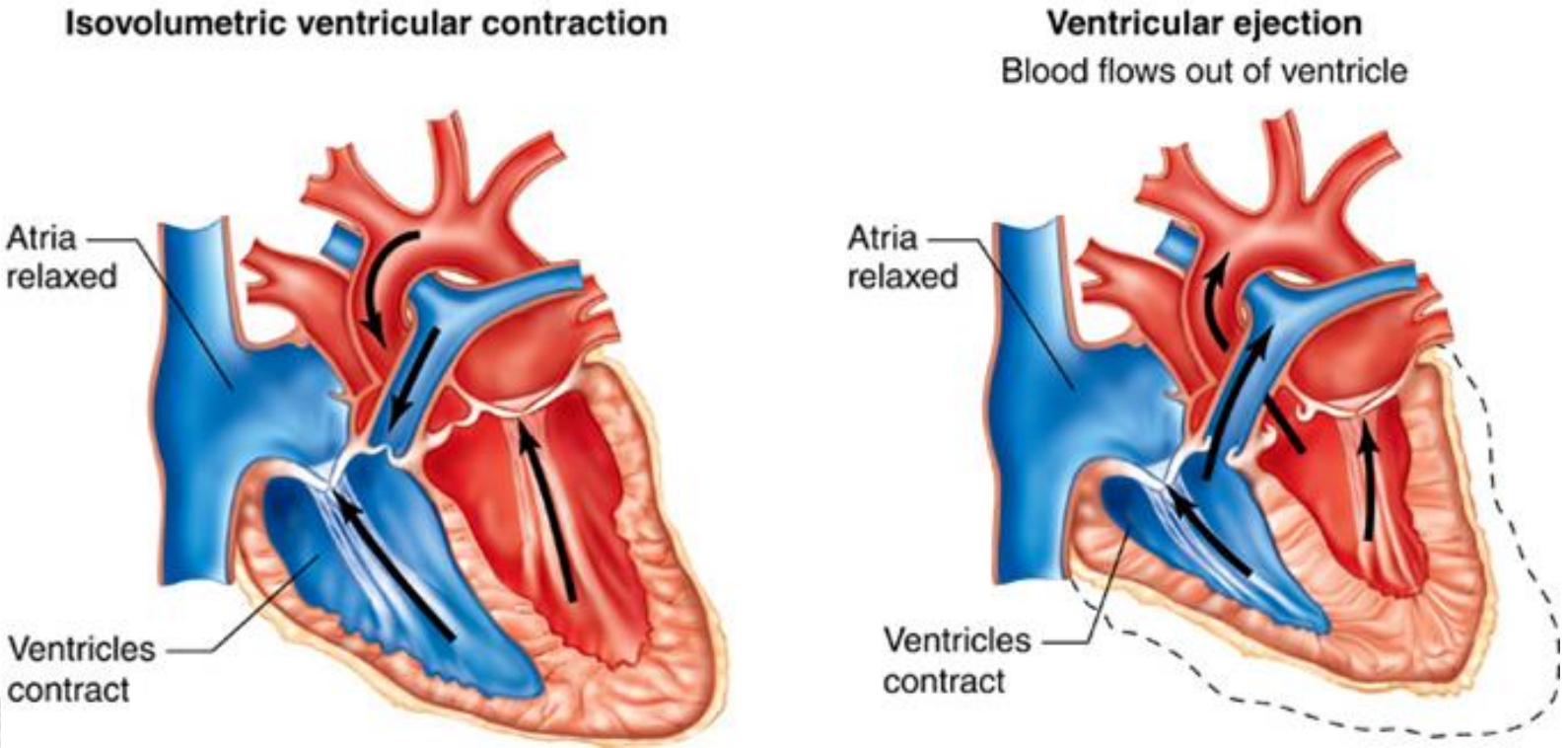
after 70% ventricular filling, AV valves **begin** to close → **P wave** & **atrial systole**; atrial pressure increases & final 30% of blood enters ventricles (**end diastolic volume** or **EDV**) → atrial **diastole** for rest of cycle



2. Ventricular systole (comprises QRS complex & T waves): ventricles begin to contract → increased pressure **closes** AV valves - period of **isovolumetric contraction** (volume constant; a closed system) → increased pressure opens SL valves → **ventricular ejection phase** (aortic pressure up to 120 mm Hg)

3. Isovolumetric relaxation: early diastole → ventricles relax; pressure decreases rapidly; backflow of aortic/pulmonary blood **closes SL valves** (**dicrotic notch**); ventricles a closed system = **isovolumetric relaxation**

4. Back to (1): atria continued in **diastole** & have been filling; when pressure > ventricular pressure, **AV valves open** and we are back to (1)



75 beat/min: each cardiac cycle=0.8 sec

atrial systole = 0.1 sec

ventricular systole = 0. 3 sec

quiescent period = 0.4 sec

2 Features driving Cardiac Cycle:

(a) Blood flow through heart controlled entirely by pressure changes

(b) Blood flows from higher to lower pressure through any available opening

** *electrical activity of left & right hearts is almost simultaneous*

4.2.8.2 define these terms in relation to a cardiac cycle: systole, diastole, isovolumetric contraction & relaxation, dichrotic notch

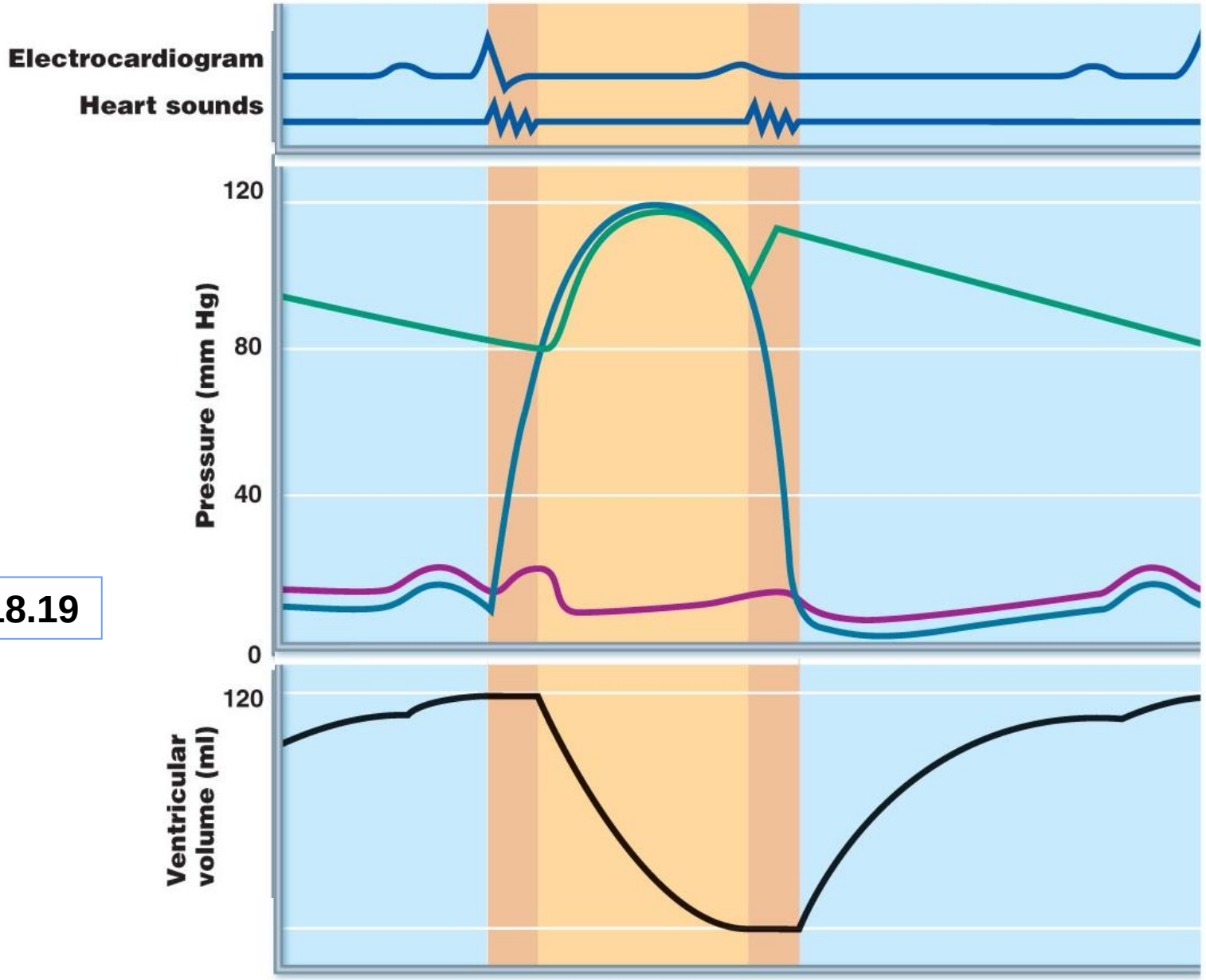
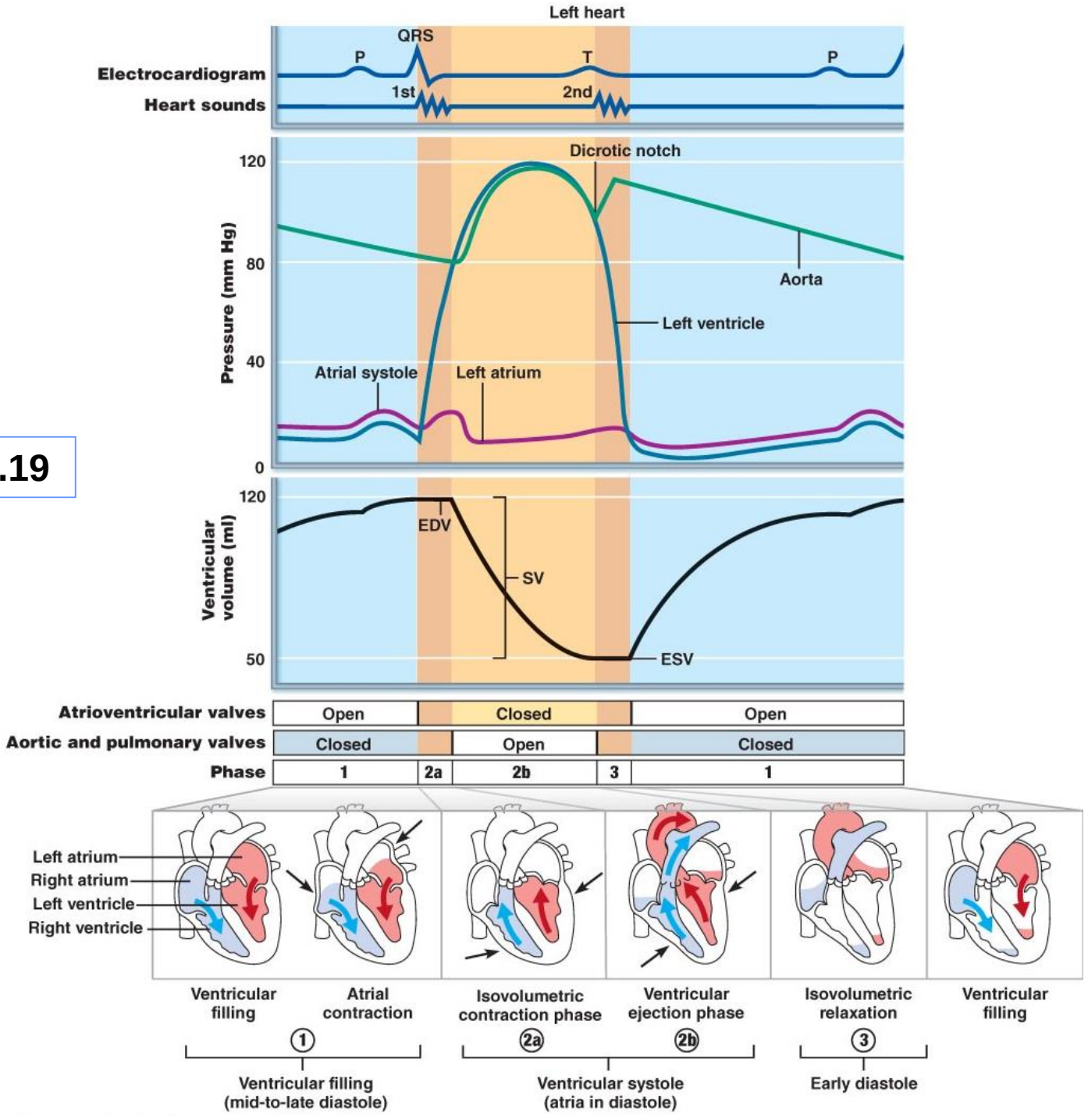
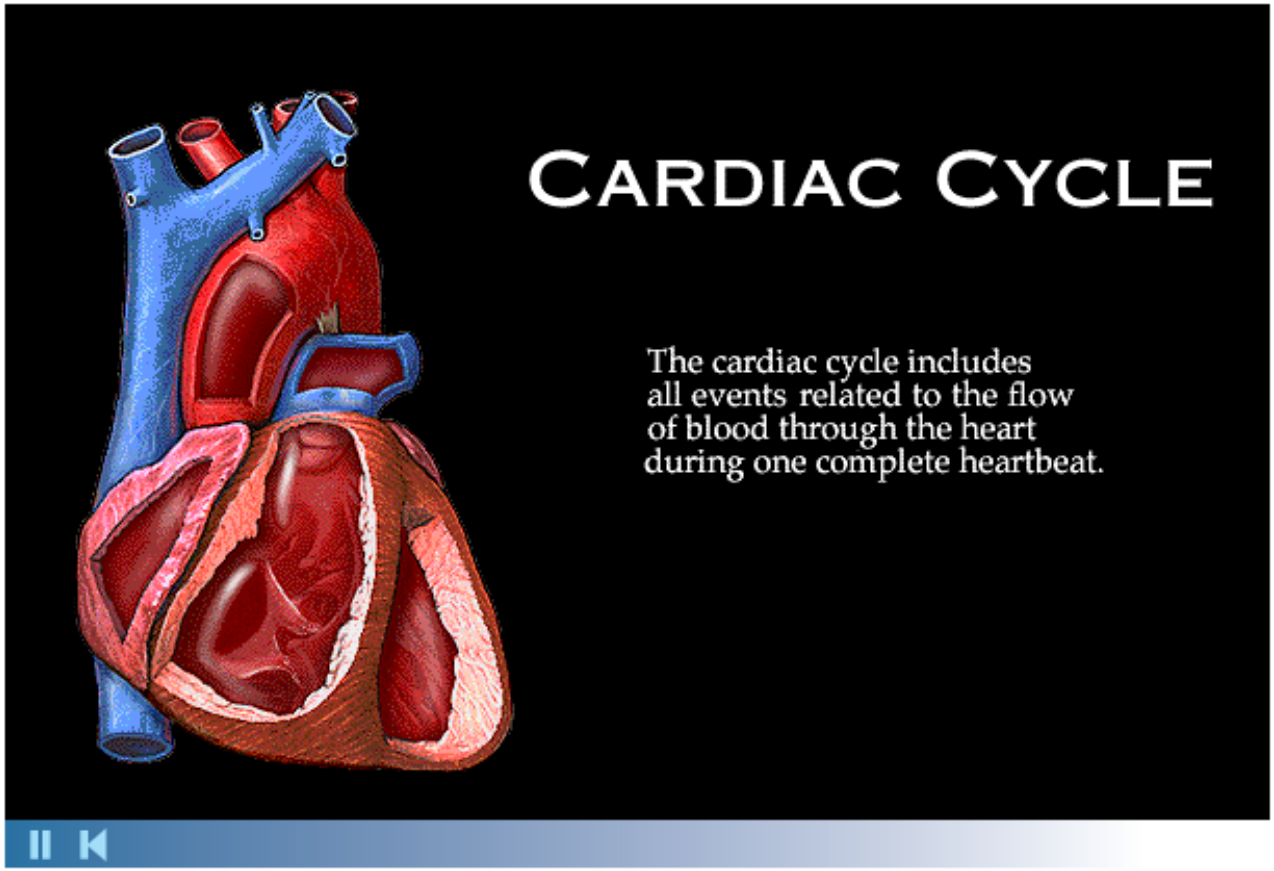


Fig. 18.19

Fig. 18.19


Left heart
Electrocardiogram
Heart sounds
QRS
P
T
P
1st
2nd
Pressure (mm Hg)
120
80
40
0
Dicrotic notch
Aorta
Left ventricle
Atrial systole
Left atrium
Ventricular volume (ml)
120
50
EDV
SV
ESV
Atrioventricular valves
Aortic and pulmonary valves
Phase
Open
Closed
Open
Closed
Open
Closed
1
2a
2b
3
1
Left atrium
Right atrium
Left ventricle
Right ventricle
Ventricular filling
Atrial contraction
Isovolumetric contraction phase
Ventricular ejection phase
Isovolumetric relaxation
Ventricular filling
1
2a
2b
3
Ventricular filling (mid-to-late diastole)
Ventricular systole (atria in diastole)
Early diastole

Introduction: Cardiac Cycle (Page 1 of 19)



4.2.8.3 indicate the physiological significance of the first and second heart sounds

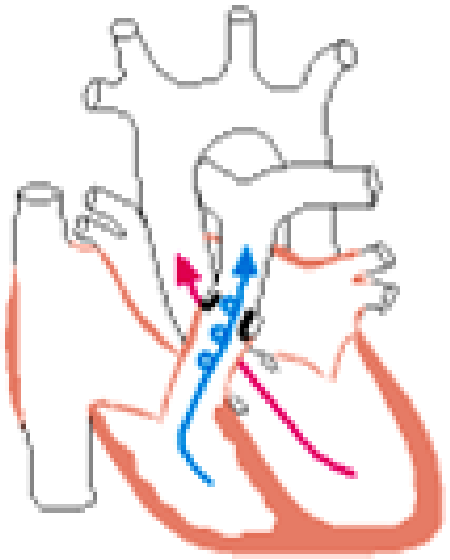
- 2 distinguishable sounds can be heard through a stethoscope
  (a) **first heart sound:** closure of AV valves = **beginning** of systole
  (b) **second heart sound:** closure of semilunar valves = **end** of systole
- heart sounds due to vibrations of heart/chest due to valve closure

heart murmurs:

(1) due to **valvular obstruction** → high velocity jet of blood through narrow opening → higher pitch of sounds

(2) due to **valvular insufficiency** → leakage of blood back causes sounds when there should be silence

Abnormal Blood Flow
In this example there is pulmonary valve stenosis. The narrowed pulmonary valve opening causes the blood to be turbulent and noisy which is heard as a heart murmur on auscultation



www.rchc.rush.edu/ rmawebfiles/heart_murmur.htm

Fill in the blanks:

The contraction of the ventricles is referred to as _____ and the period of ventricular relaxation is called _____ . The first heart sound is a result of closure of the _____ valves and closure of the _____ valves causes the second heart sound. The heart chambers that have just been filled when you hear the first heart sound are the _____. The chambers that have just emptied when you hear the first heart sound are the _____. Immediately after the second heart sound, the _____ are filling and the _____ have emptied. Abnormal sounds, or _____ usually indicate valve problems.

1. During atrial systole:
    a) the atrial pressure exceeds ventricular pressure
    b) 70% of ventricular filling occurs
    c) the AV valves are open
    d) valves prevent backflow into the great veins
    e) a and c

2. Threshold in pacemaker cells is marked by:
    a) opening of sodium gates
    b) opening of calcium slow channels
    c) opening of calcium fast channels
    d) opening of potassium gates

4.2.9 Define cardiac output (CO) in terms of heart rate and stroke volume

CO = amount of blood pumped from left ventricle into aorta/min

- average CO for resting, healthy male = 5L/min

SV = EDV-ESV *(EDV= end diastolic volume ESV = end systolic volume)*

CO = HR X SV

CO = cardiac output HR = heart rate

SV = stroke volume

Sample calculation for average male:

HR = 75 beats/min CO = 75 x 70

SV = 70 ml/beat = 5250 ml/min

- note that total blood volume for an average male is 5L → a RBC makes the complete circuit in about 1 min

4.2.9.3 indicate the influences of exercise on HR & SV

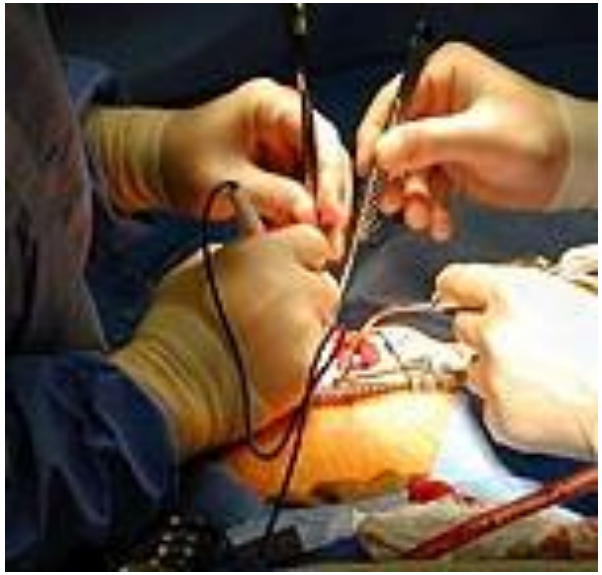
- CO ⇑ 4-5 times in a fit person
- CO ⇑ 7 times in a well-trained marathon runner
- notion of **cardiac reserve**
- combined effects on **HR** & **SV**

4.2.10 Describe in detail the mechanisms for the regulation of HR & SV

1. Delineate the effects on HR: autonomic ns (which branch dominant at rest?), epinephrine (adrenal medulla), plasma electrolytes, body temp

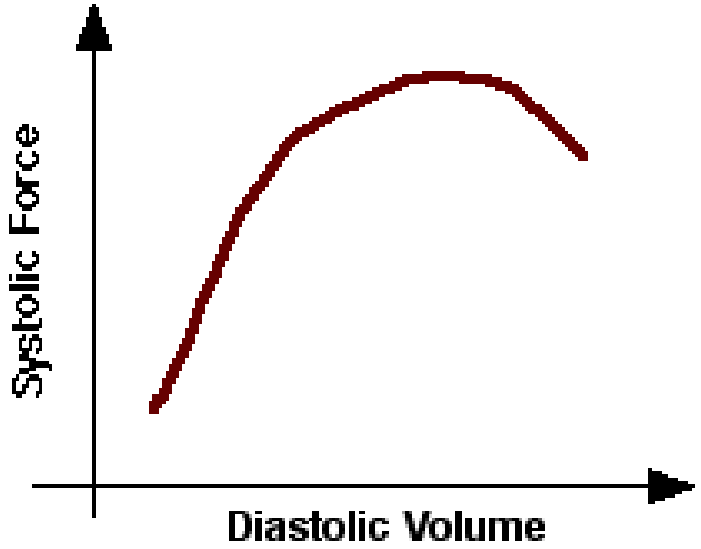
- *heart rate is determined by rate of spontaneous depolarization of* ***SA node:***
  (i) autonomic fibers innervating SA node
  (ii) circulating hormones (eg: epinephrine)
  (iii) plasma electrolyte concentration (**Ca^{++}**, Na^{+}, **K^{+}**, H^{+})
  (iv) body temperature (useful in surgery)

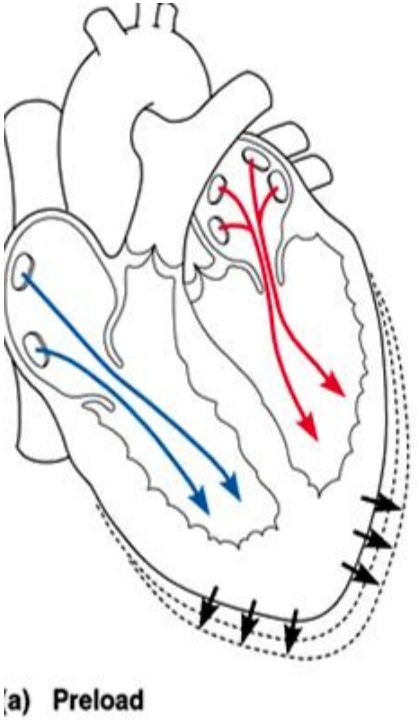
- NE (symp): **increases** rate of spontaneous depolarization → heart rate is ⇑
- ACh (parasymp ns): **decreases** rate of spontaneous depolarization (hyperpolarizes pacemaker cells) »» heart rate is ⇓
- **resting conditions: parasympathetic ns dominant (vagal tone)**



Tachycardia *(>150-170 beats/min) leads to reduced CO. Why??*

4.2.10.2 apply the Frank Starling Law of the Heart to the intrinsic regulation of SV





a) **<u>Preload:</u>** = **Frank Starling Law of the Heart:** Within defined limits, the heart will pump whatever volume of blood it receives

- over a fairly wide range, there is a proportional relationship between EDV and stroke volume

- cardiac muscle has optimal length for contraction (= *length-tension relationship*); **resting** = **shorter** than optimal length!
- each ventricle regulated independently and beat-to-beat: **FS mechanism** ensures that each ventricle pumps same volume *over a period of time*

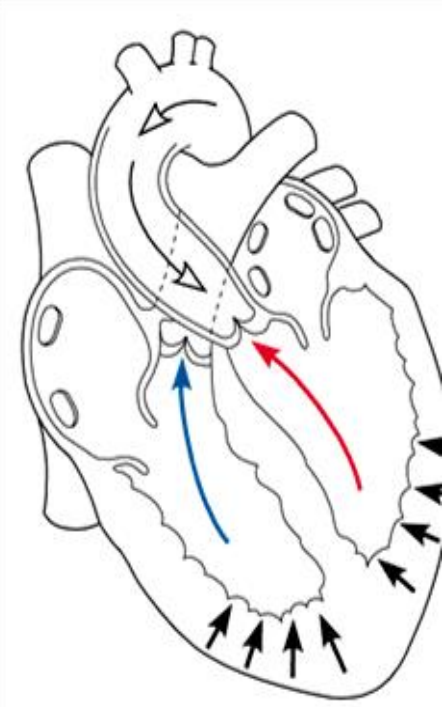
What are the effects of chronically elevated blood pressure on cardiac muscle cells?

How can this apply to both physical training or a chronic disease (e.g. pulmonary stenosis)?

4.2.10.3 define afterload and describe its influence on stroke volume

Afterload = pressure that ventricles must overcome to force open valves & eject blood from heart

- healthy: ~80 mm in aorta; 10 mm in pulmonary trunk »» not a major determinant of SV
- hypertension reduces ability of ventricles to eject blood »» ⇧ ESV and ⇩ SV



(b) Afterload

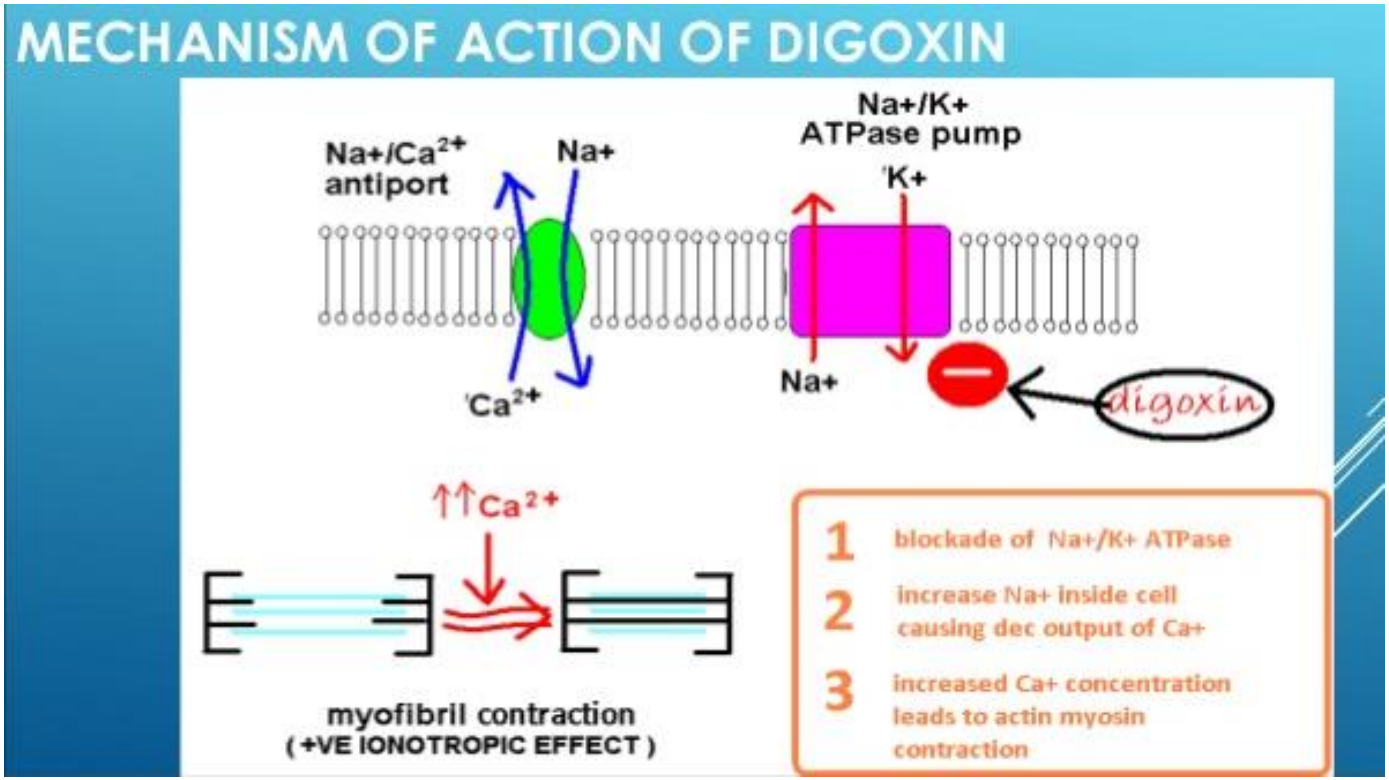
4.2.10.5 describe 2 types of extrinsic influences on stroke volume

- factors outside heart which change vigour of contraction without changing EDV = change in **contractility**
- **not** due to greater initial fiber length but involves change in strength of **contraction** due to increased Ca^{++} **influx**

(i) **sympathetic stimulation:** increases rate of contraction & relaxation

(ii) drugs such as **digoxin**: increase heart contractility

(iii) **parasympathetic ns:** antagonizes sympathetic stimulation



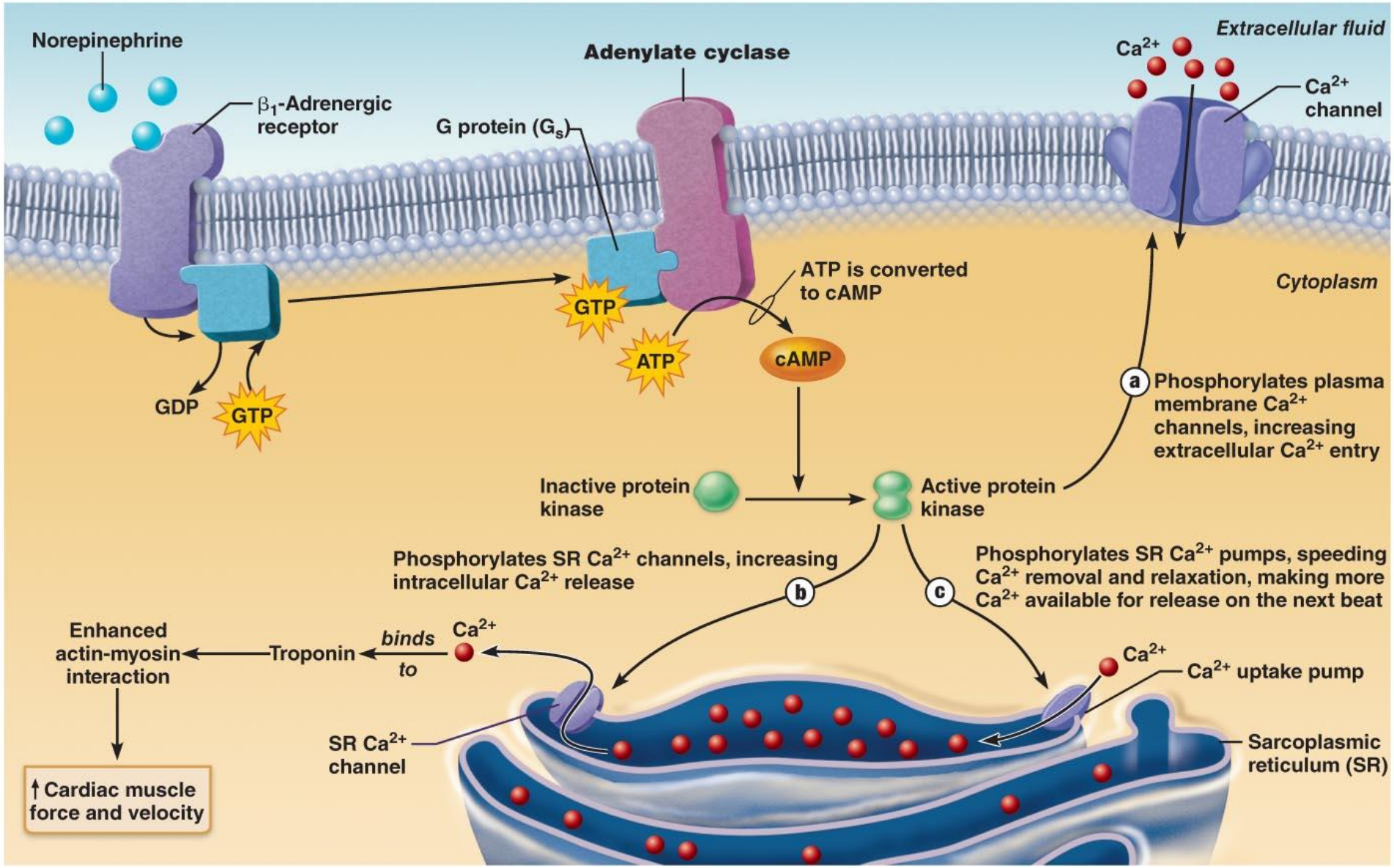


Fig. 18.23: Norepinephrine increases heart contractility via cyclic AMP

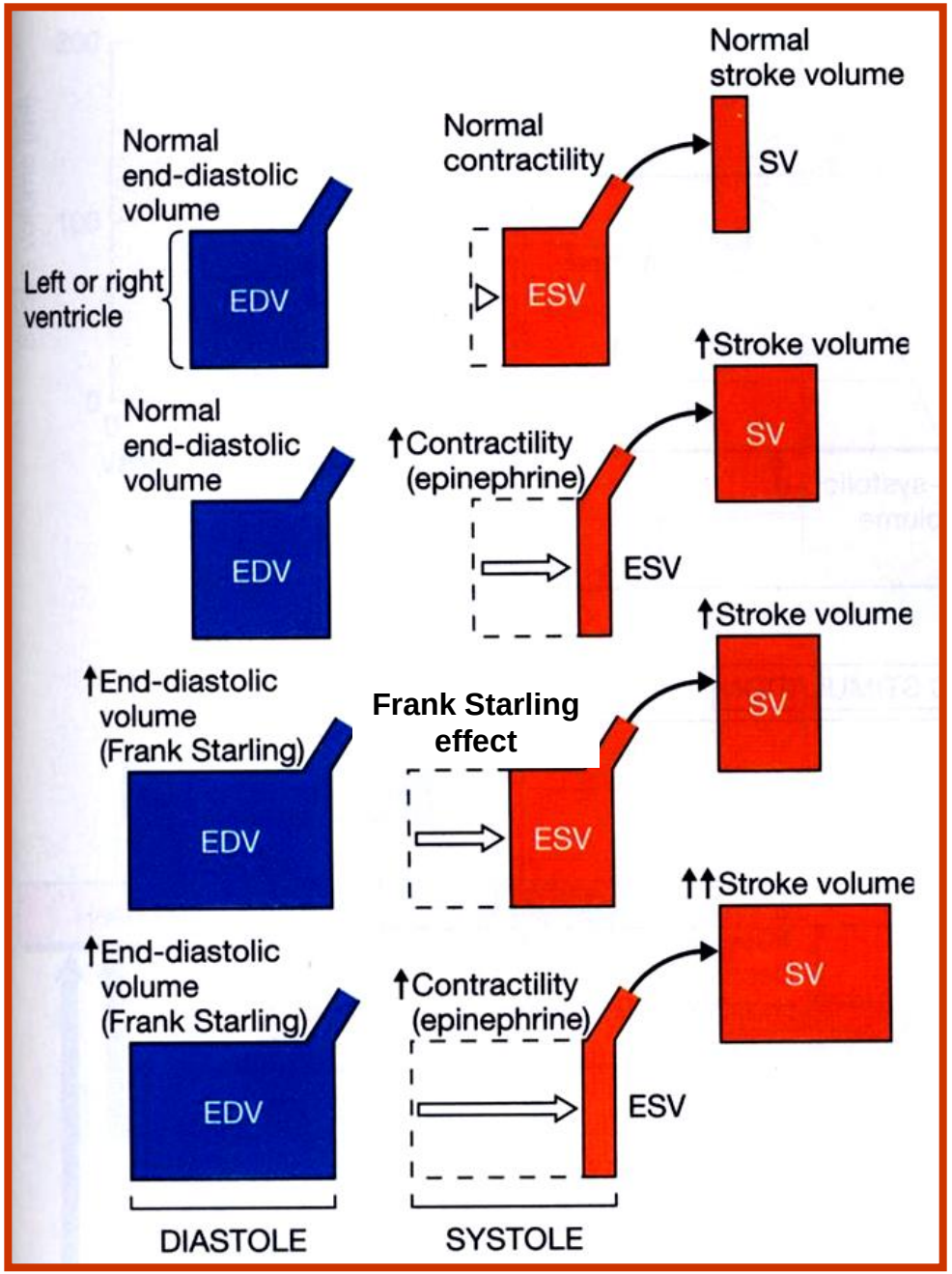


Fig. 18-23 (R&P): Interaction between contractility & Frank Starling Law to influence stroke volume