

Cardiovascular System

Outline

- Introduction
- Electrical activities of the heart
- Electrocardiogram
- The cardiac cycle
- The cardiac out put
- Law of hemodynamics
- Microcirculation
- Lymphatic system
- Control of Cardiovascular functions

Reference

Guyton and Hall, Text book of
Medical Physiology, Twelfth edition

1. Over view of cardiovascular system

- Three basic components

1. Heart

- The heart is a dual pump that:
 - drives blood in two serial circuits
 - »**pulmonary(lungs)** and the **systemic (the rest of the body)** circulations.
 - and receives blood from the rest of the body through the vena cava.

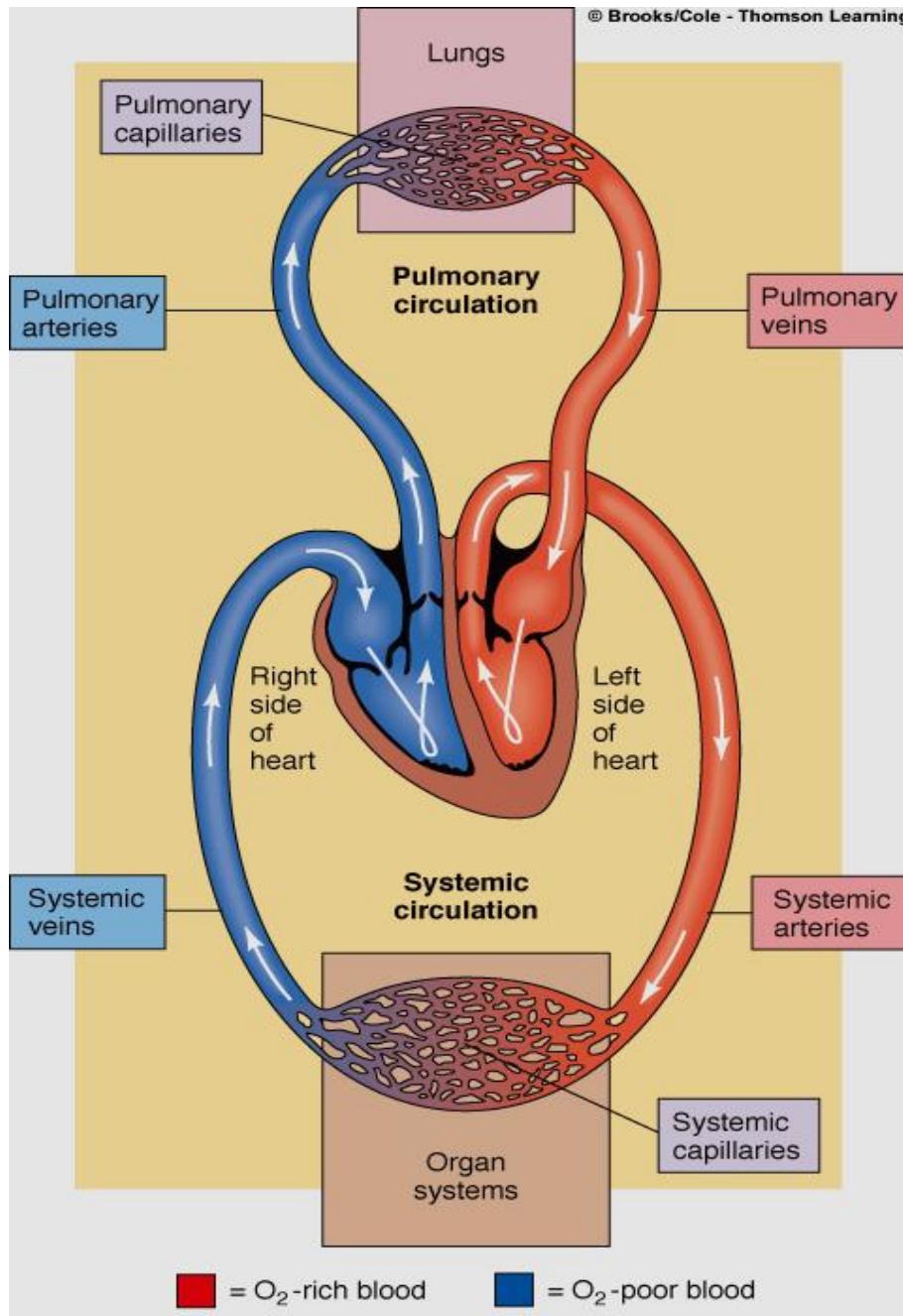
2. Blood vessels

- Passageways through which blood is distributed from heart to all parts of body and back to heart.

3. Blood:

- Transport medium within which materials being transported are dissolved or suspended.

- **Pulmonary circulation**
 - The flow of blood between heart and lungs
- **Systemic circulation**
 - The flow of blood between heart and the body systems.



Main functions of the circulatory system

- Transport and distribute essential substances to the tissues.
- Remove metabolic byproducts.
- Adjustment of oxygen and nutrient supply in different physiologic states.
- Regulation of body temperature.

Physiology of Cardiac Muscle

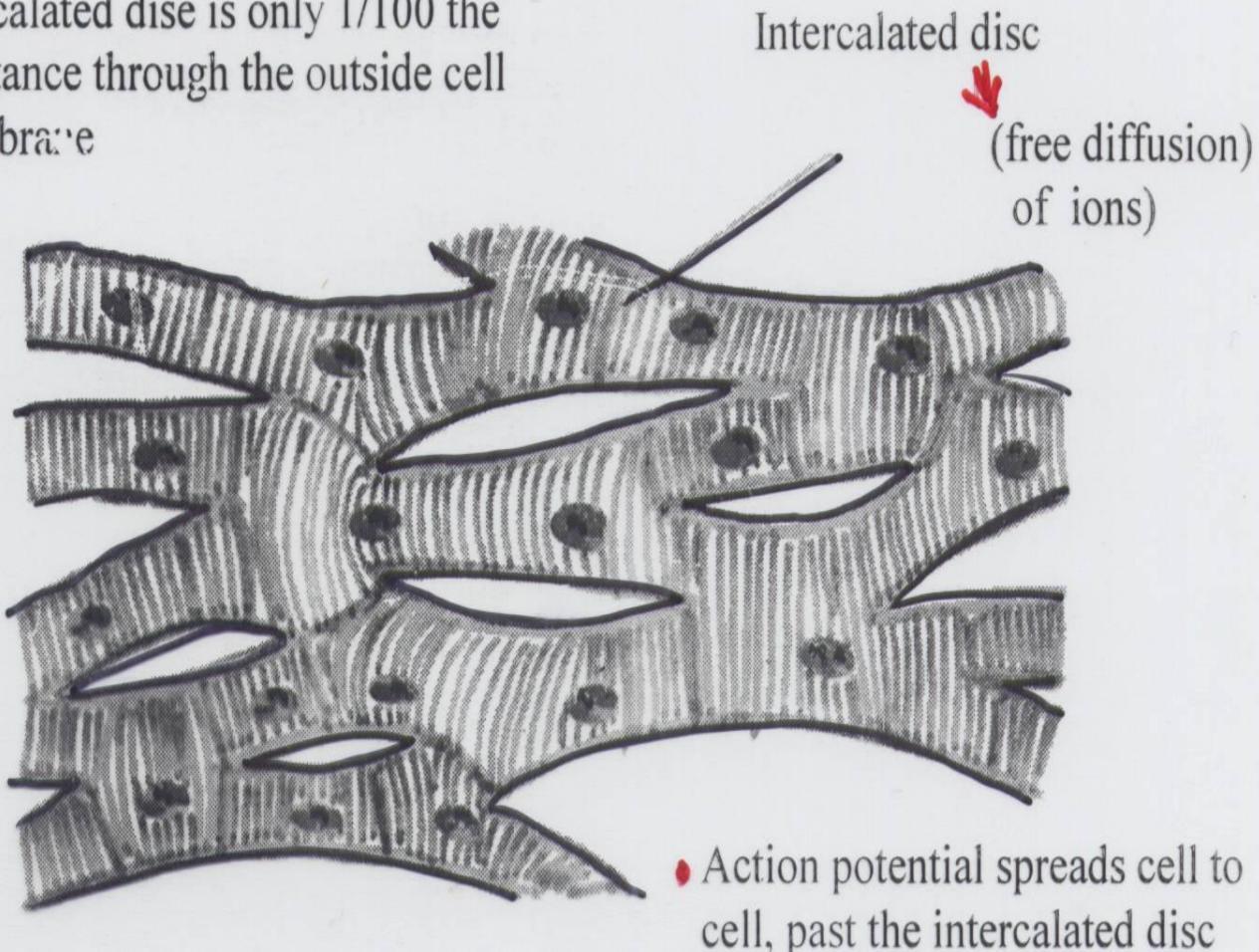
- The heart is composed of three major types of cardiac muscle: *atrial muscle*, *ventricular muscle*, and specialized *excitatory* and *conductive muscle* fibers.
 - Myocardium is made up of 3 types of muscles.
 1. Contractile unit
 2. Pacemaker unit
 3. Conducting unit



Figure: Cardiac myocytes

- The myocytes are joined end to end by thick connections called *intercalated discs*.
- *Intercalated discs* are actually *cell membranes* that separate individual cardiac muscle cells from one another.
- Action potential spreads cell to cell, past the intercalated disc.

- Electric resistance through the intercalated disc is only 1/100 the resistance through the outside cell membrane

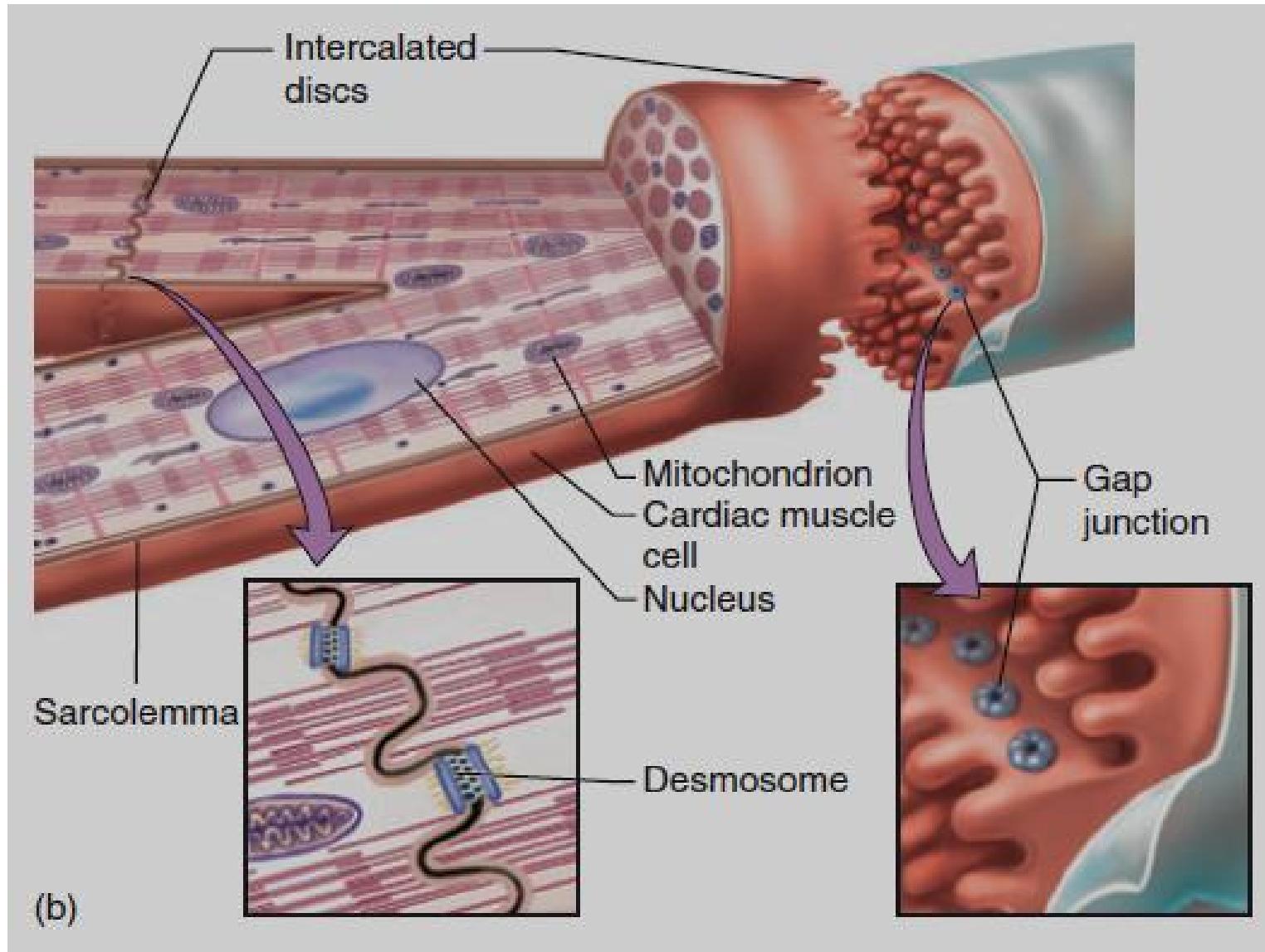


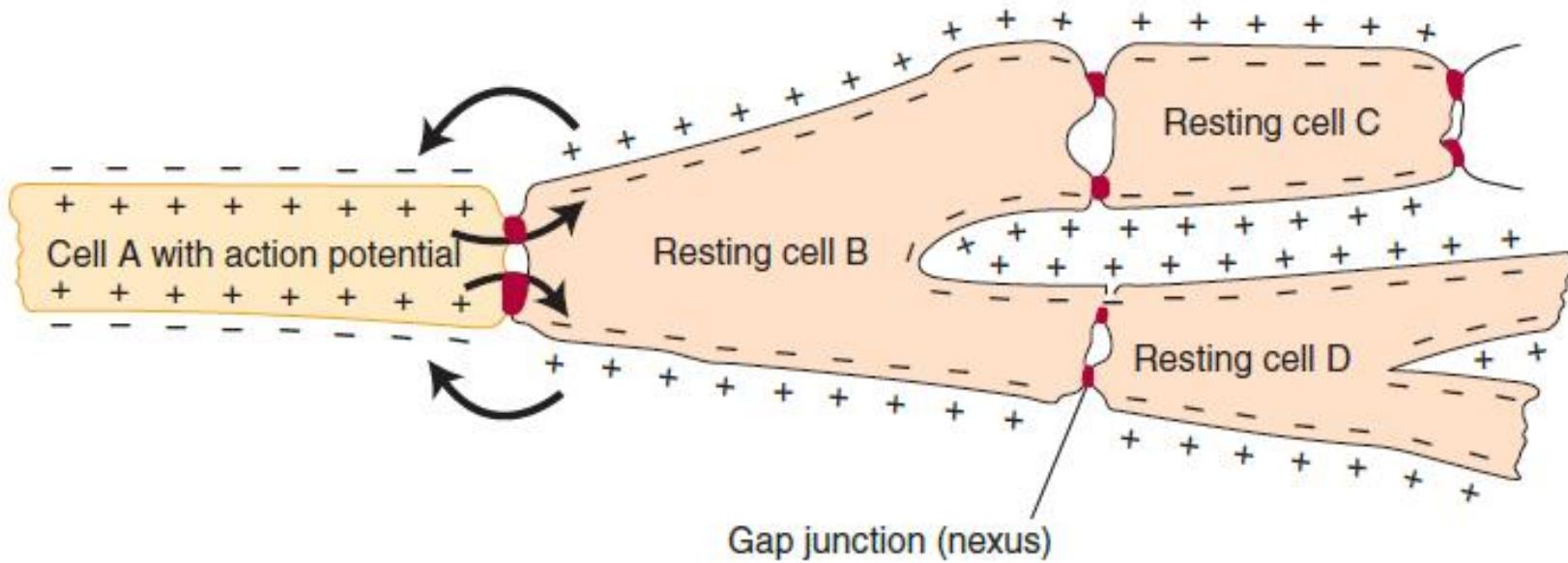
Features of intercalated disc:

1. *Interdigitating folds* - increase the surface area of intercellular contact.
2. *Mechanical junctions* (fascia adherens and desmosomes) - prevent the myocytes from pulling apart when the heart contracts.

3. *Electrical junctions* - the myocytes are electrically coupled by *gap junctions*.

- Which form channels that allow ions to flow from one cell directly into the next.
- Myocardium of the atria and the ventricles each acts almost as if it were a single cell.





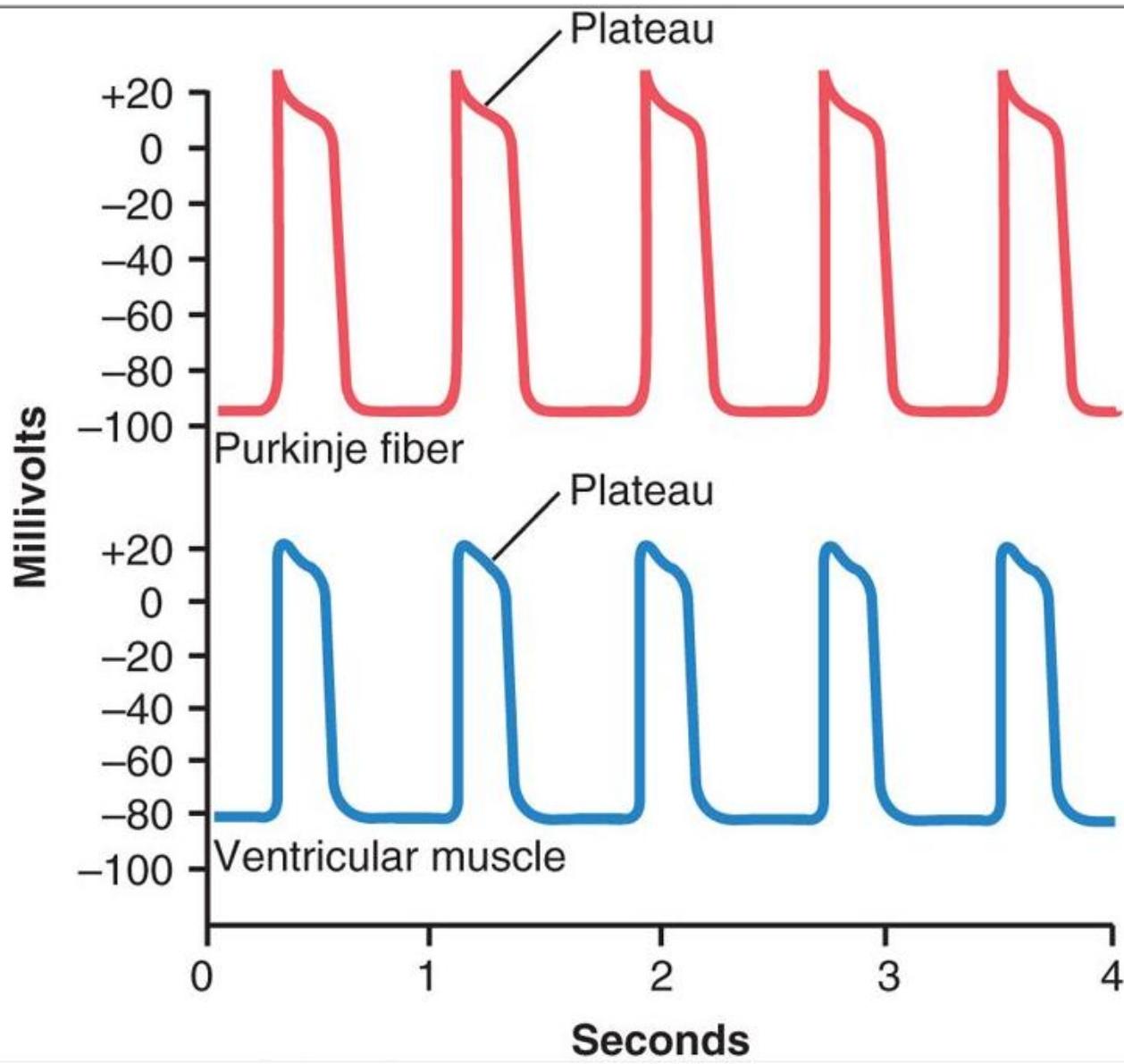
Local currents and cell-to-cell conduction of cardiac muscle cell action potentials

- The heart is composed of two syncytia:
 1. **The *atrial syncytium*** - which constitutes the walls of the two atria.
 2. **The *ventricular syncytium*** - which constitutes the walls of the two ventricles.

- The atria and ventricles are separated by ***fibrous tissue*** that surrounds the AV valve openings.
- Potentials are ***not conducted*** from the atrial into the ventricle directly through this fibrous tissue.
 - They are conducted only by way of a specialized conductive system called the ***AV bundle***.

Action Potentials in Cardiac Muscle

- In *cardiac muscle*, the *action potential* is caused by opening of two types of channels:
 1. **Fast sodium channels**
 2. **Slow calcium-sodium channels**
 - Are **slower to open** and remain open for several tenths of a second.
 - A large quantity of both **calcium and sodium** ions flows in to the cardiac muscle fiber.
 - The calcium ions that enter during this plateau phase activate the muscle contractile process.

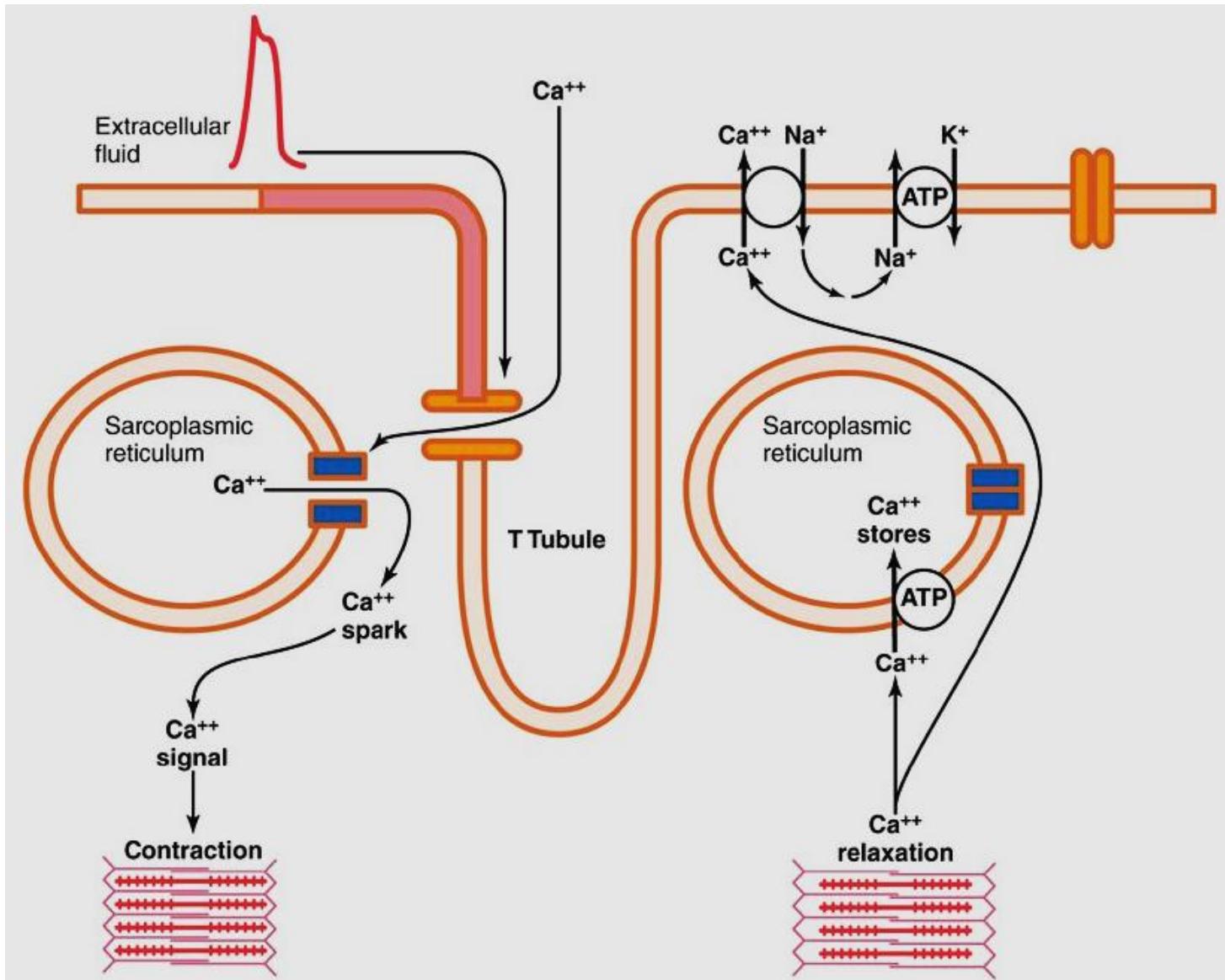


- Immediately after the onset of the action potential, the permeability of the cardiac muscle membrane for **potassium ions** *decreases.*
 - Thereby prevents early return of the action potential voltage to its resting level.

- When the slow calcium-sodium channels do **close**, the membrane permeability for potassium ions also increases rapidly.
 - This rapid loss of potassium from the fiber immediately returns to RMP level.
 - Ending the action potential.

Excitation-contraction coupling

- Mechanism of excitation-contraction coupling is the same as skeletal muscle.
- Differences
 - There is diffusion of calcium into the sarcoplasm from the **T tubules** at the time of the action potential.
 - Calcium entering the cell then activates **calcium release channels (ryanodine receptor channels)** in the SR membrane.
 - Trigger the release of calcium into the sarcoplasm.



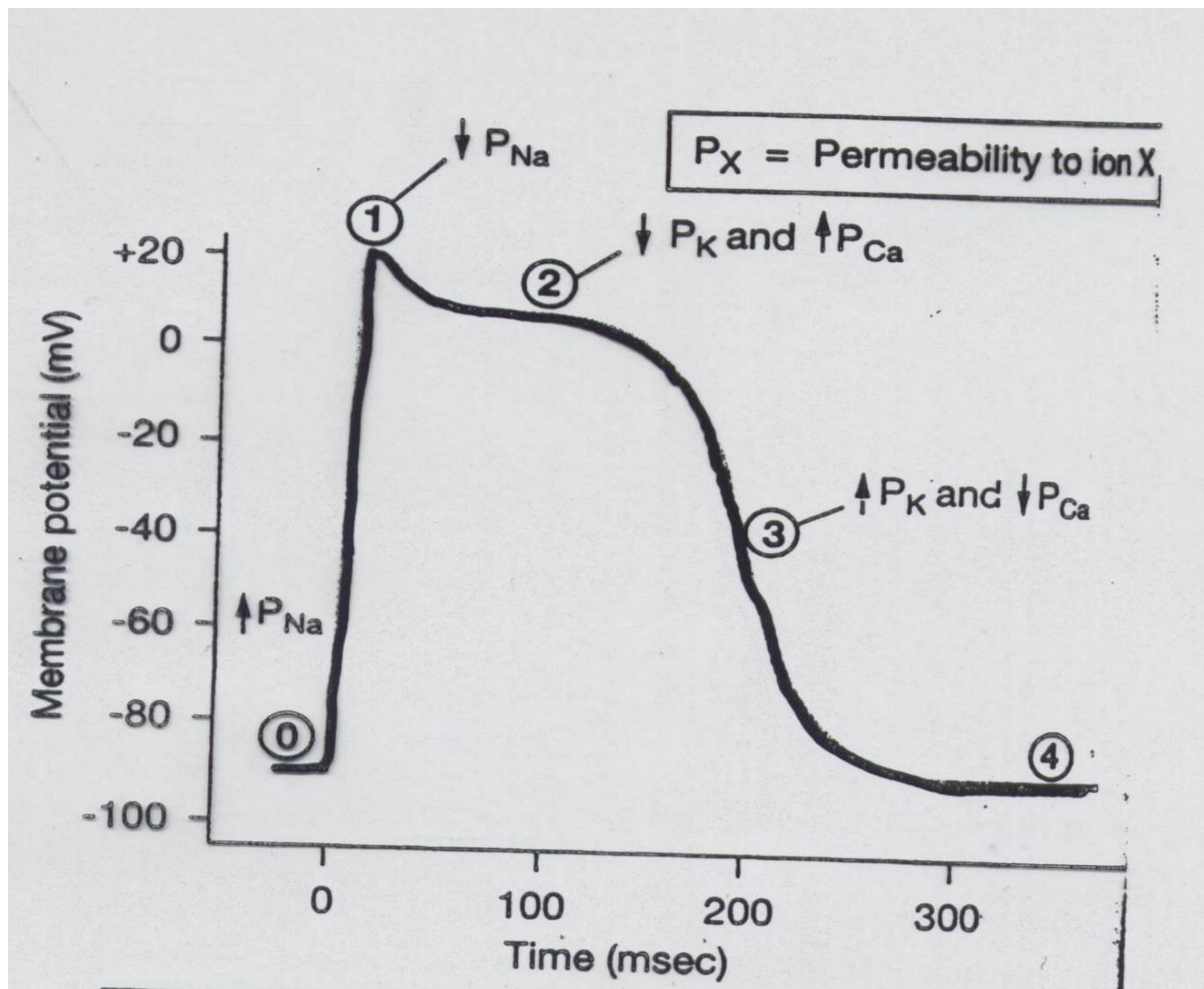
- The strength of contraction of cardiac muscle depends to a great extent on the concentration of calcium ions in the extracellular fluids.
- Because the SR of cardiac muscle is less well developed.
- The T tubules of cardiac muscle have a **diameter 5 times** as great as that of the skeletal muscle tubules.

Excitability and cardiac action potential

- Action potential of cardiac muscle, as recorded by intracellular recording, is of two types.
 - A. Fast response action potential found **in working myocardial cells.**
 - B. Slow response action potential found **in pacemaker tissues.**

A. *Fast response action potential*

- Action potential of the working myocardium.
- Fast fibers have ***functioning fast sodium channels.***
- Depolarization will be **very rapid** and action potential spreads very quickly across the surface of the cell.
- Fast fibers include **ventricular fibers, atrial fibers and purkinje fibers.**

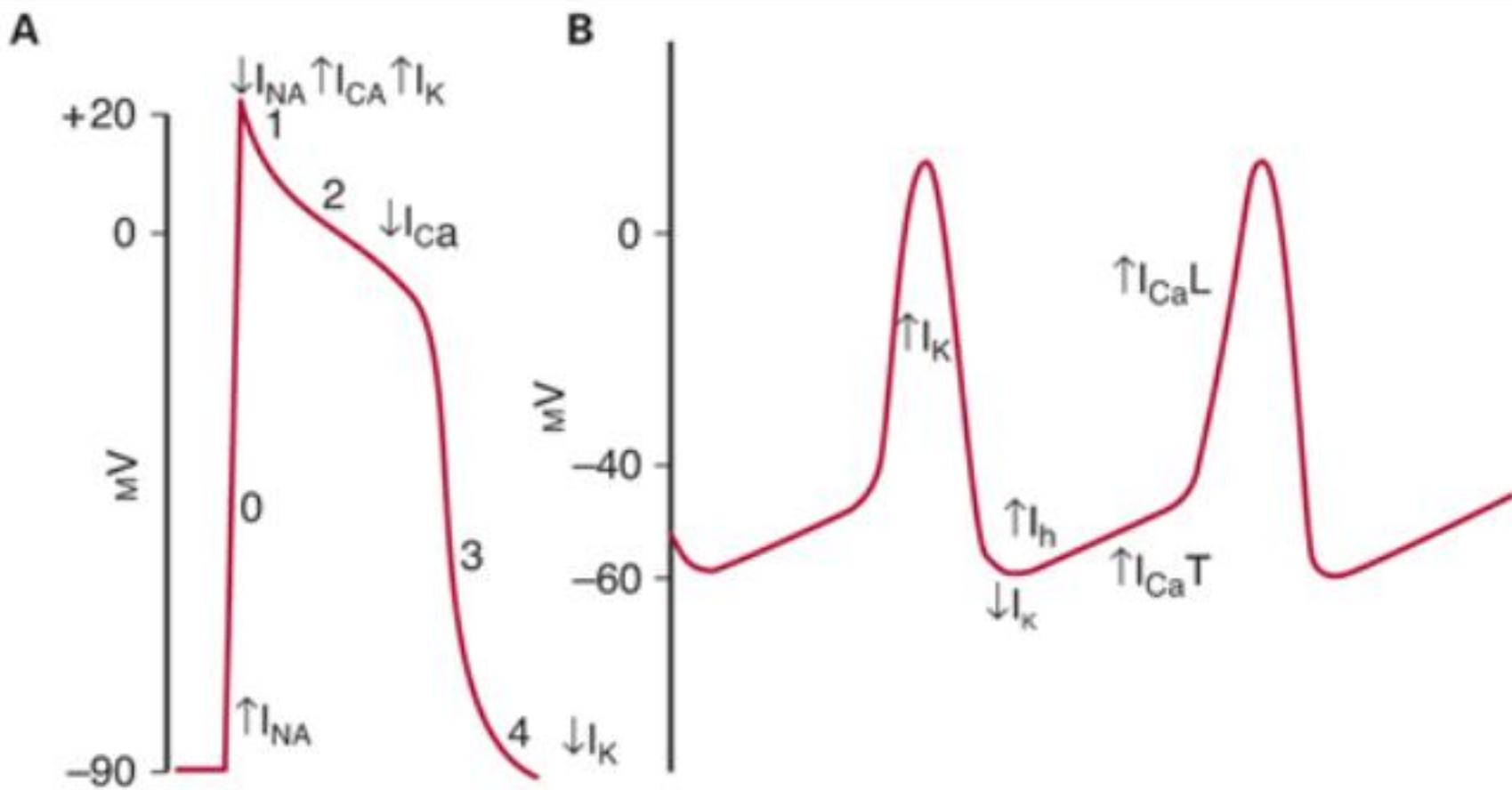


<u>Phase</u>	<u>membrane channels</u>
0.	Na ⁺ channels open
1.	Na ⁺ channels close
2.	Ca ²⁺ channels open, fast K ⁺ channels close
3.	Ca ²⁺ channels close, slow K ⁺ channels open
4.	Resting potential

B. Slow response action potential

- Slow fibers lack of functioning fast channels .
- Depolarization is **slower** and the action potential travels more slowly across the surface of the cell.
- These cells possess an **unstable RMP**.
- **Sinoatrial node and AV nodal fibers.**

- At the level of -55mV, the fast sodium channels become inactivated.
- Therefore, *only the slow sodium-calcium channels can become activated* and cause the action potential.
- After the action potential does occur, return of the potential to its negative state occurs slowly.



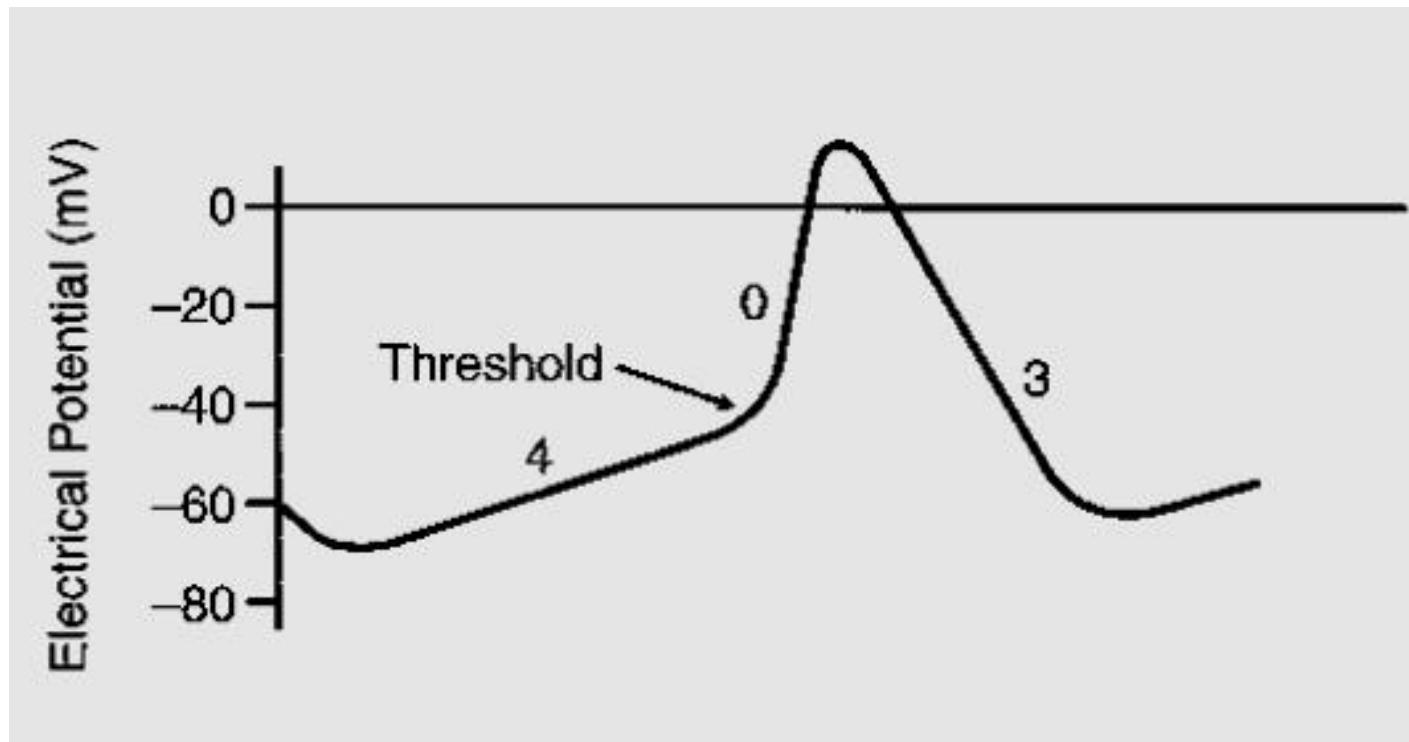
Comparison of action potentials in ventricular muscle and diagram of the membrane potential of pacemaker tissue.

The action potential of contractile and autorhythmic cell

Fast response fibers	Slow response fibers
<ul style="list-style-type: none">• REM more negative• Steep upstroke• Plateau due to calcium• Depolarize and repolarize when stimulated (not spontaneously active)	<ul style="list-style-type: none">■ REM less negative■ Upstroke more gradual■ AP amplitude smaller■ Absence of fast sodium channels.■ Presence of spontaneously opening calcium channels.

Self-Excitation of Sinus Nodal Fibers

- The inherent leakiness of the sinus nodal fibers to sodium and calcium ions causes their self-excitation.
 - Causes a slow rise in the resting membrane potential in the positive direction.
 - When the potential reaches a threshold voltage, the sodium-calcium channels become "activated."
 - Thus causing the action potential.



Nerve supply to heart

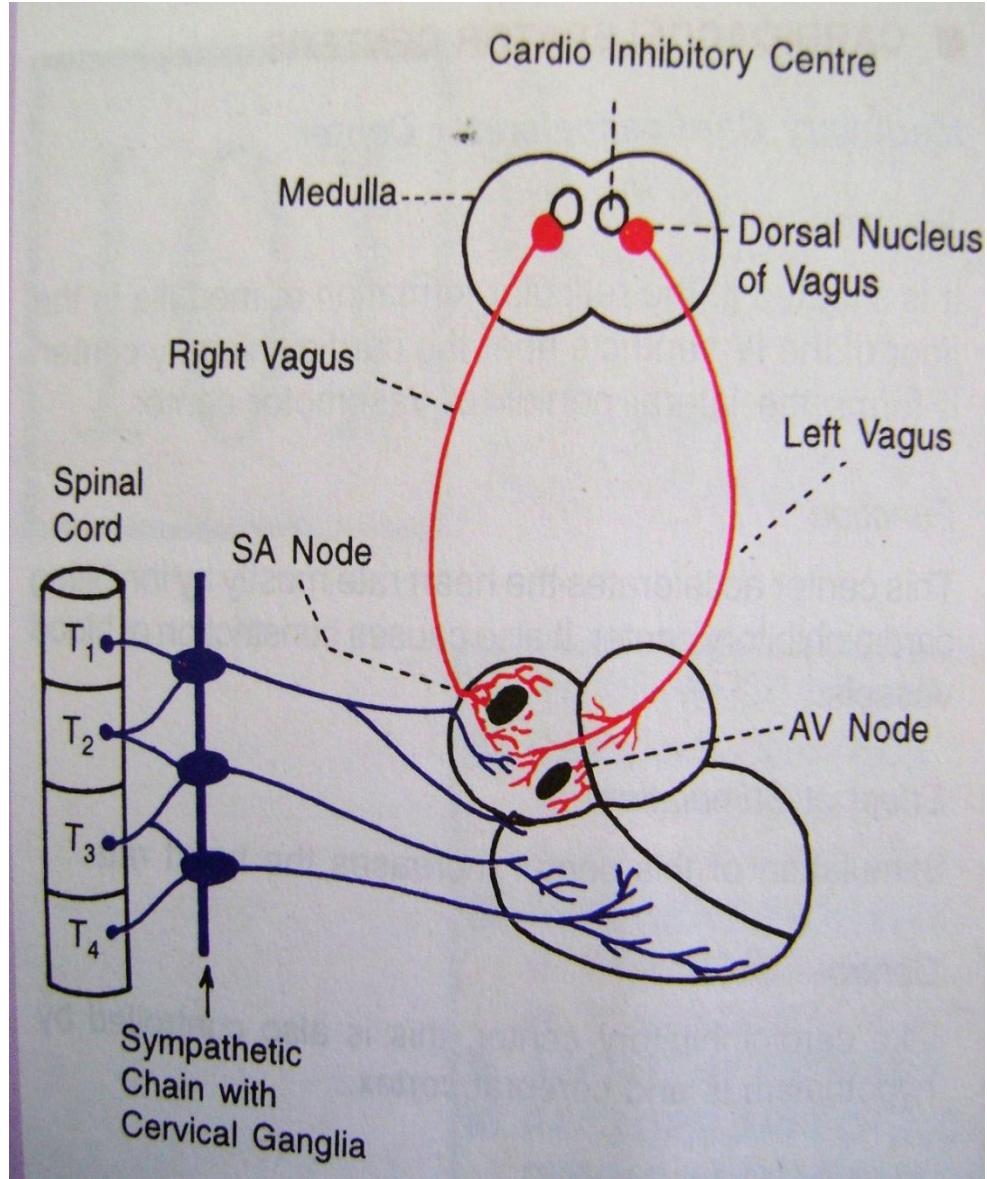
- Heart is supplied by both the divisions of autonomic nervous system.
- **Parasympathetic fibers**
 - ✓ Arise from the medulla & pass through the **vagus nerve**.
 - ✓ Parasympathetic mainly innervates **SA node, atria and AV node**.
 - ✓ No direct Ventricular innervations - Vagal escape (Ventricular escape).

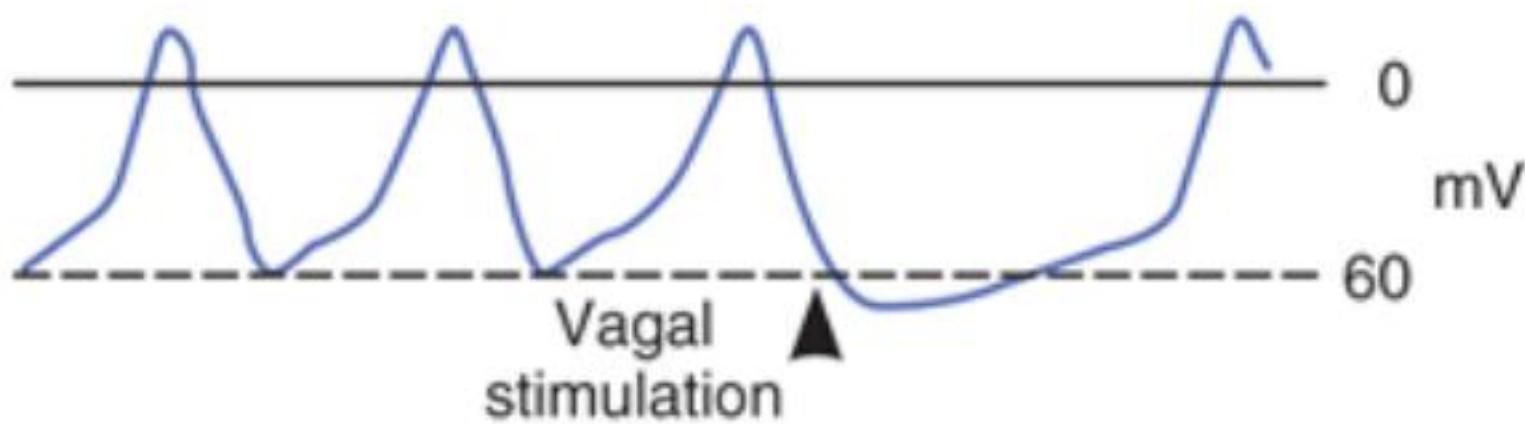
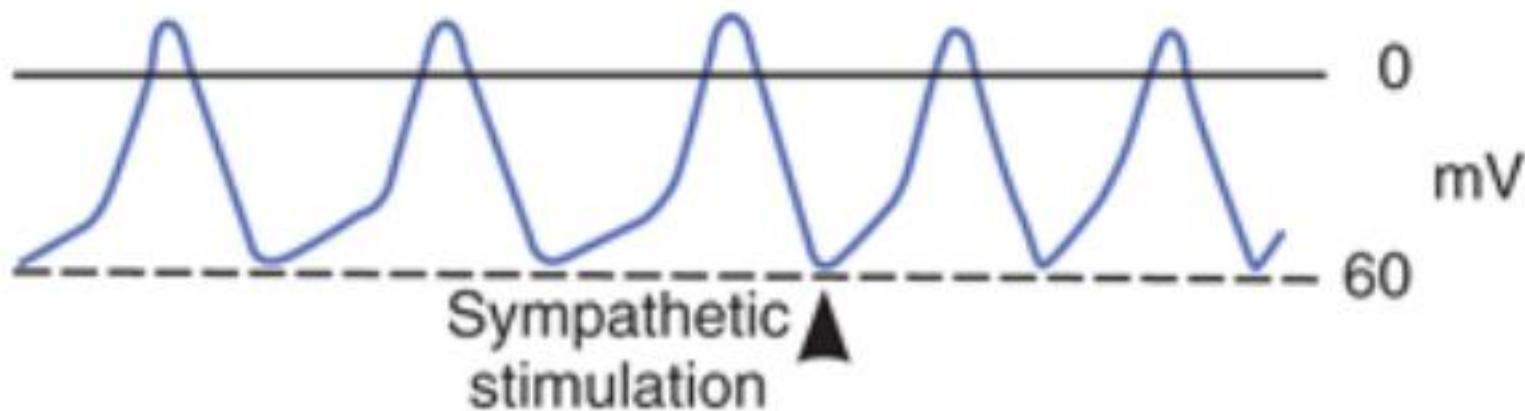
- *The sympathetic fibers:*

- Arise from the upper thoracic (T1 – T4) segments of spinal cord & pass through the sympathetic nerves.
- Can innervate *all parts* of the heart.

➤ Function:

- Parasympathetic fibers are cardioinhibitory in nature.
- Whereas Sympathetic fibers are cardioacceleratory in nature.





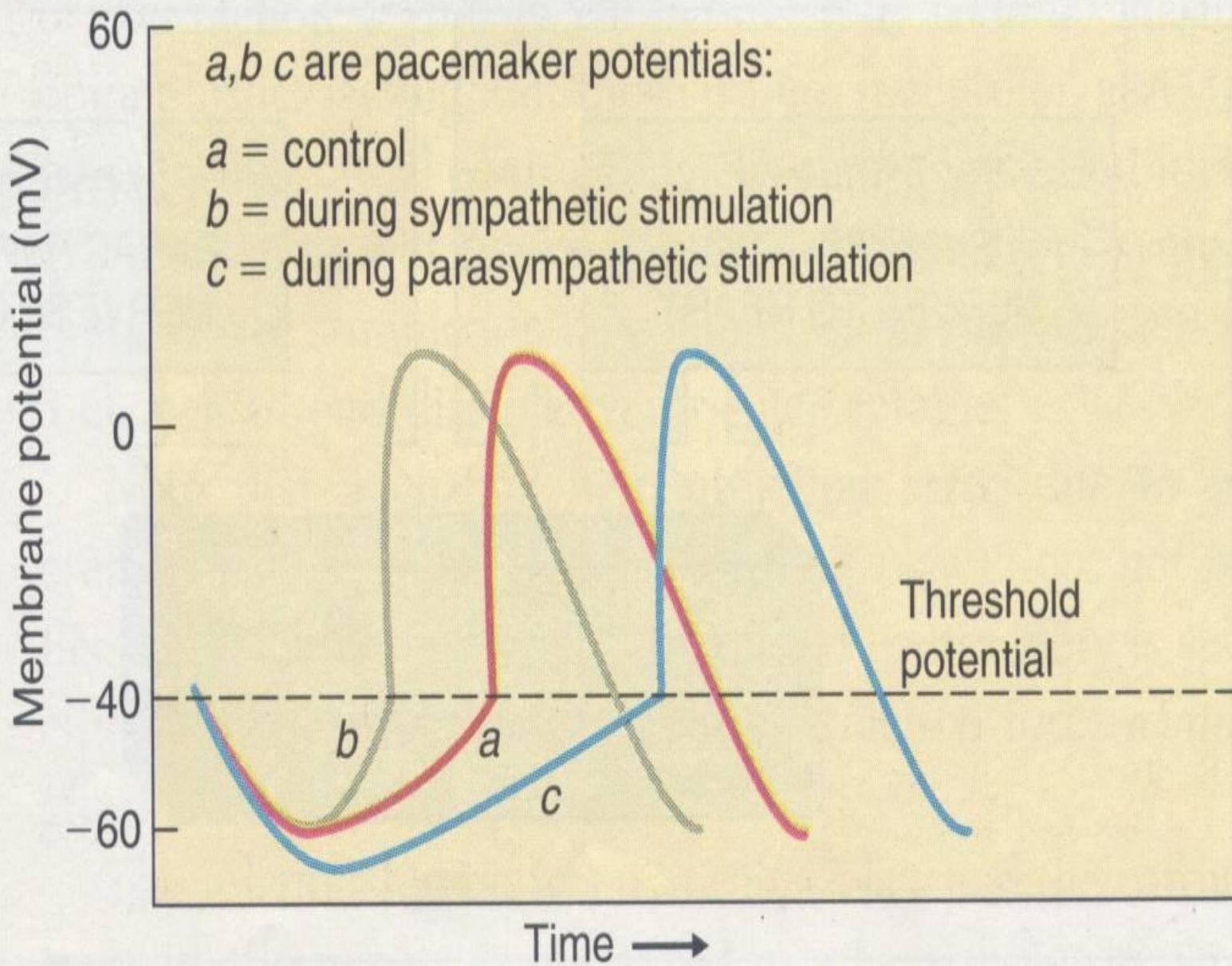
Sympathetic and Parasympathetic effects

Sympathetic stimulation

- ↑ HR (+ve chronotropic)
- ↑ force of contraction (+ve Inotropic)
- Adrenergic transmitters and sympathetic stimulation increase slope of diastolic pacemaker depolarization.
- ∴ ↑ HR

Parasympathetic stimulation

- Ach opens K⁺ channels
- Reduce slope of pacemaker potential
∴ ↓ HR
- Also hyperpolarize pacemaker potential and reduce HR
- Increases the K⁺ efflux, so slope reduced and HR ↓



Autorhythmicity:

- 1% of the myocardium cells are specialized to generate action potential spontaneously.
- The signal for contraction is **Myogenic**, originating within the heart muscle itself.
- The autorhythmic cells are also called **pacemakers** because they set the heart beat.
- S.A. node is the pace maker of the heart.

Excitatory and Conductive System of the Heart

- The conduction system consists of the following components.

1. Sinoatrial (SA) node

- Patch of modified myocytes in the right atrium, just under the epicardium.
- It is the **pacemaker** that initiates each heartbeat.
- The fibers of this node do not have **contractile elements**.
- These fibers are continuous with fibers of atrial muscle, so that the impulses from the S.A node spread rapidly through atria.

2. Atrioventricular (AV) node

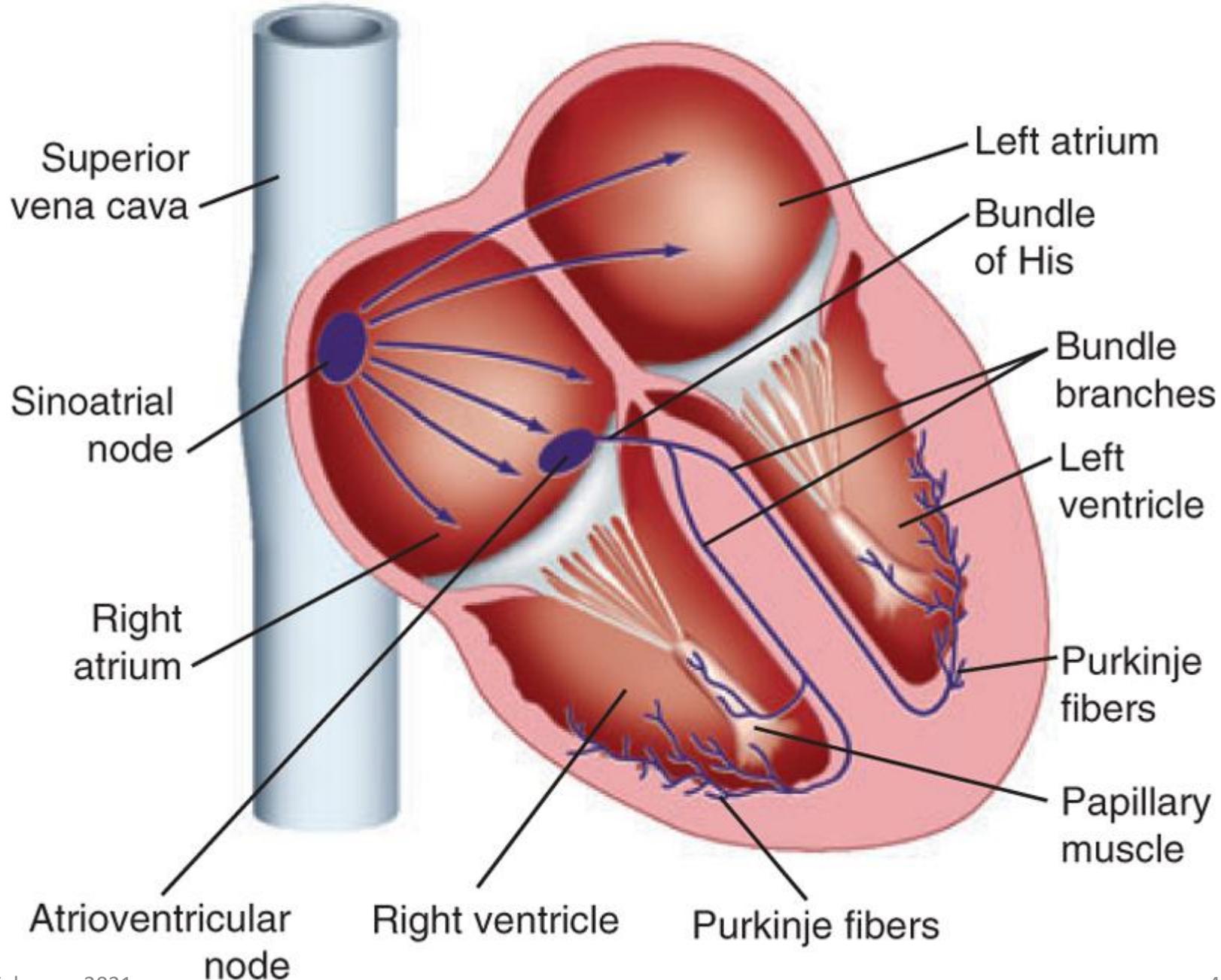
- Located near the right AV valve at the lower end of the interatrial septum.
- This node acts as an electrical gateway to the ventricles.
- Action potential is conducted from the atria into the ventricle by way of **the A-V bundle only.**
- The fibrous skeleton acts as an **insulator** to prevent currents from getting to the ventricles by any other route.

3. Atrioventricular (AV) bundle (bundle of His)

- A pathway by which signals leave the AV node.
- The **right and left bundle branches**, divisions of the AV bundle that enter the interventricular septum and descend toward the apex.

4. Purkinje fibers

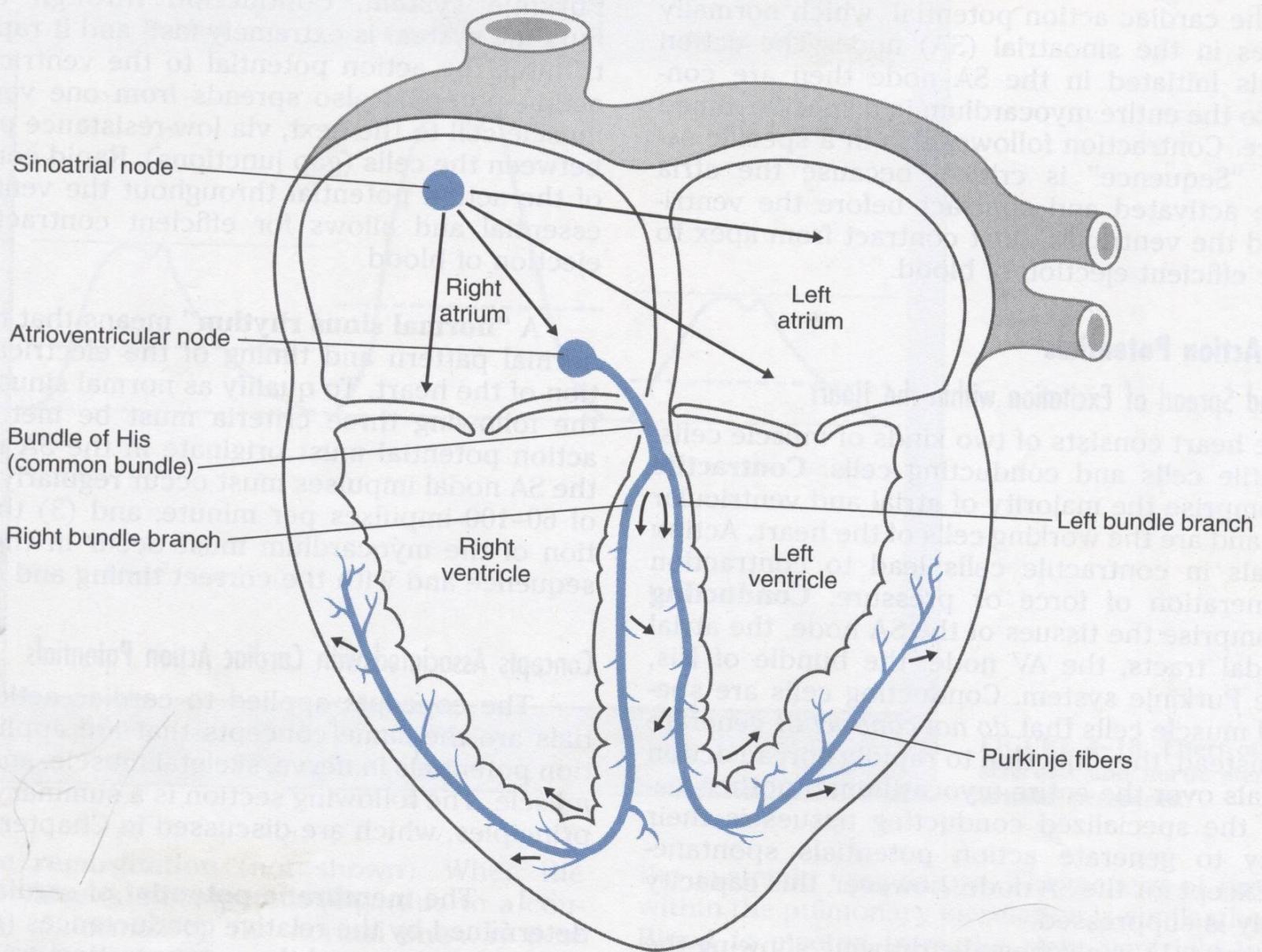
- Nerve like processes that arise from the bundle branches near the apex of the heart and then turn upward and spread throughout the ventricular myocardium.
- Purkinje fibers distribute the electrical excitation to the myocytes of the ventricles.
- ***Fastest propagation*** in the Purkinje system.



Geometry of propagation

- **Sinoatrial node (SA node)**
 - Normal pacemaker, drive heart at a rate of 70bpm (rest)
 - Excitation spreads over working myocardium of atria.
- **Atrioventricular node (AV node)**
 - Only pathway for conduction to ventricles, rest of atrioventricular boundary consist of unexcitable connective tissue.
 - Propagation briefly **delayed at AV node** (Important for ventricular filling).
 - Potential pacemaker (if SA node fails)

- **His bundle, bundle branches and purkinje fibers**
 - Propagation fast for rapid successive ventricular excitation
 - Fastest propagation in the Purkinje system



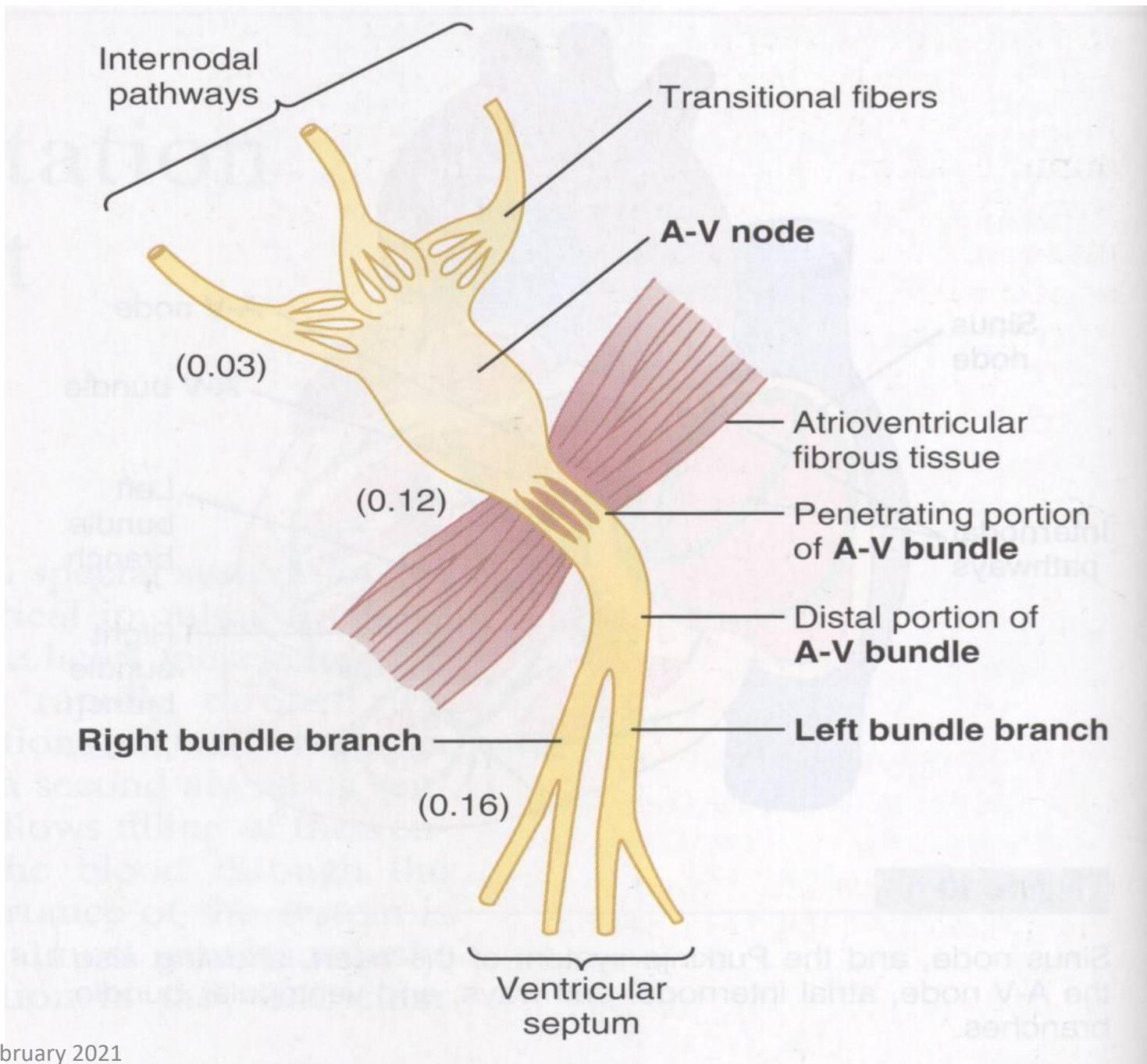
Spread of cardiac excitation

Atrial activation

- Depolarization is complete in about 0.1 second

AV nodal delay

- The conduction in AV node is slow; rate of conduction is 0.05 m/s.
- This allows atria to empty before ventricular contraction begins
- **Ventricular activation follows**



Conduction velocity

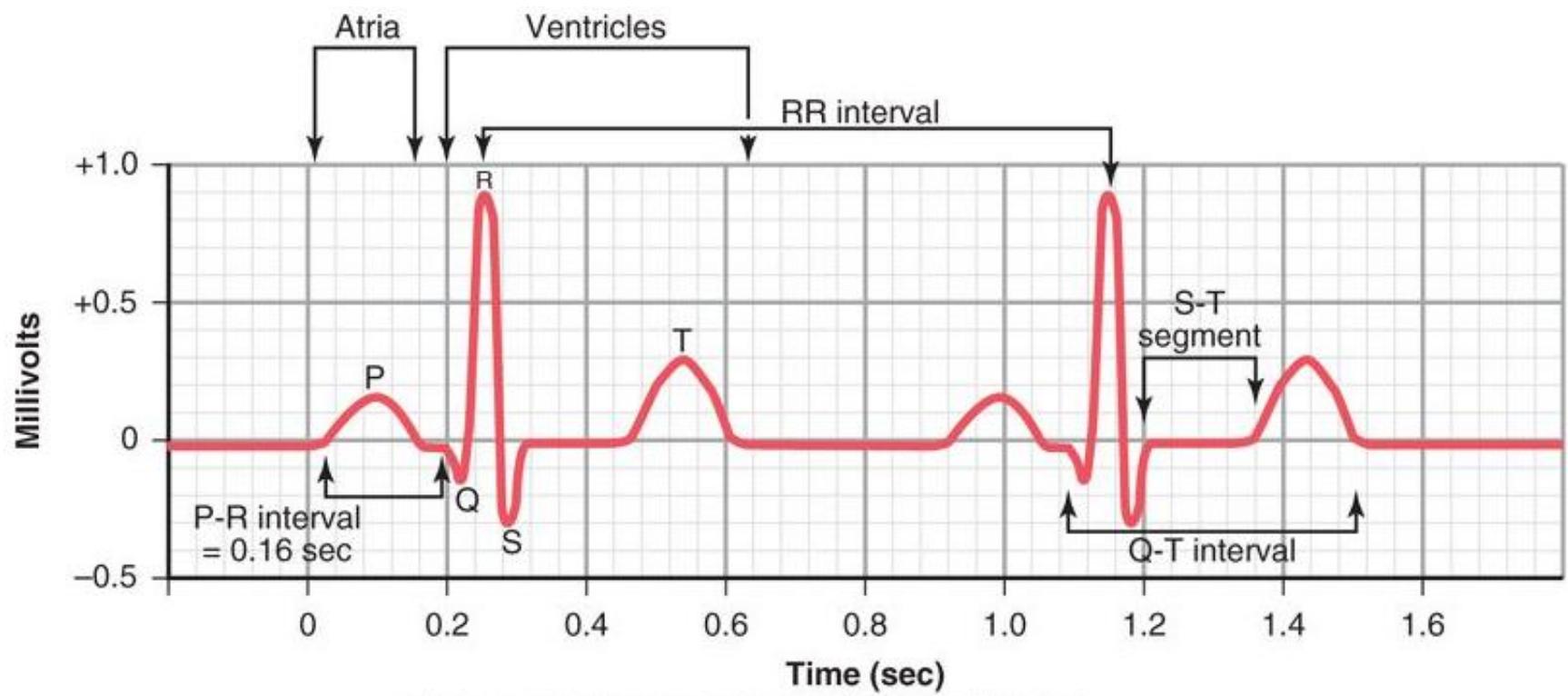
Area	Velocity of conduction (m/sec)	Rhythmicity (impulse/min)
SA node	0.05	110-120
Atrial muscle	0.3	-
AV node	0.05-0.1	40-60
A-V Bundle	2-4	35-40
Purkinje	2-4	15-40
Ventricular	0.3	-

The cardiac AP is initiated in SA node and spreads throughout myocardium

Electrocardiogram (ECG)

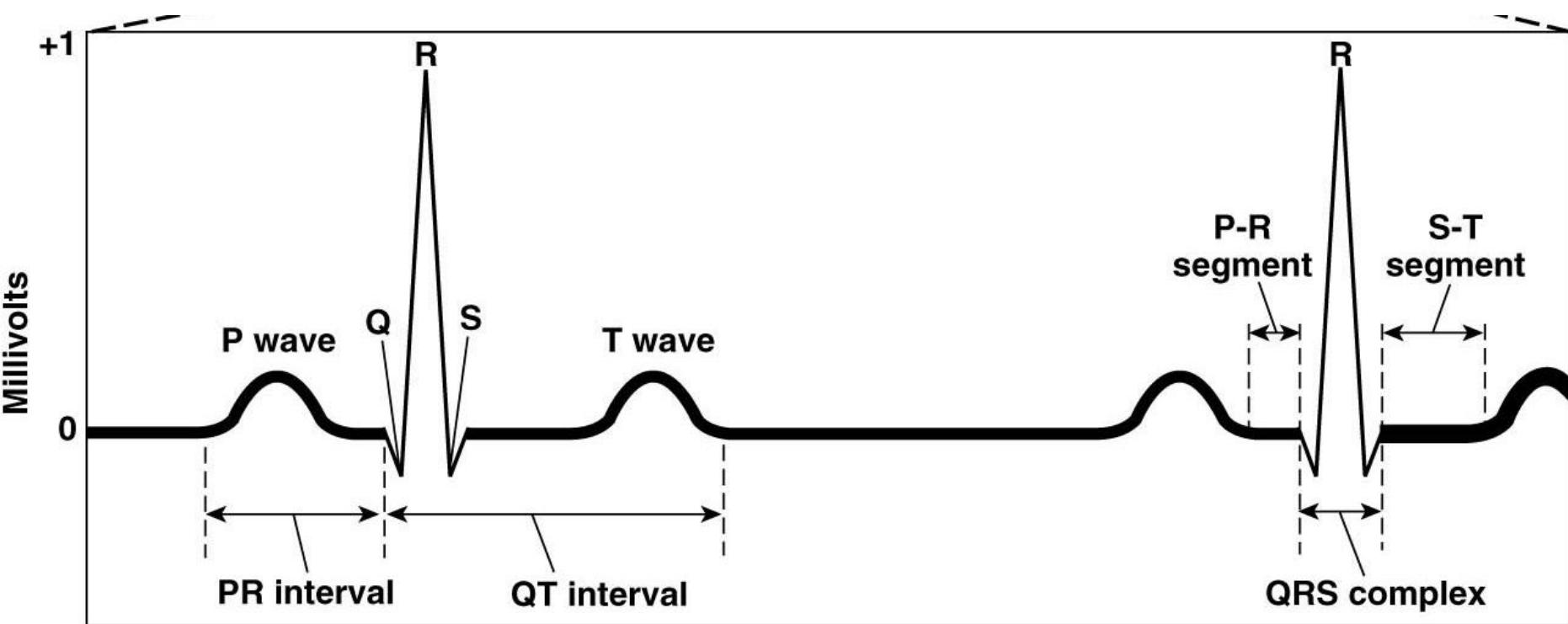
- It is a graphical representation of electrical activities of the heart during each cardiac cycle recorded from the surface of the body.
- The electrical events of the heart are conducted through out the body in all directions, as the body fluids acts as volume conductor. It is also conducted to the surface of the body.
- These electrical events can be picked up by placing a appropriate electrodes over the surface of the body.

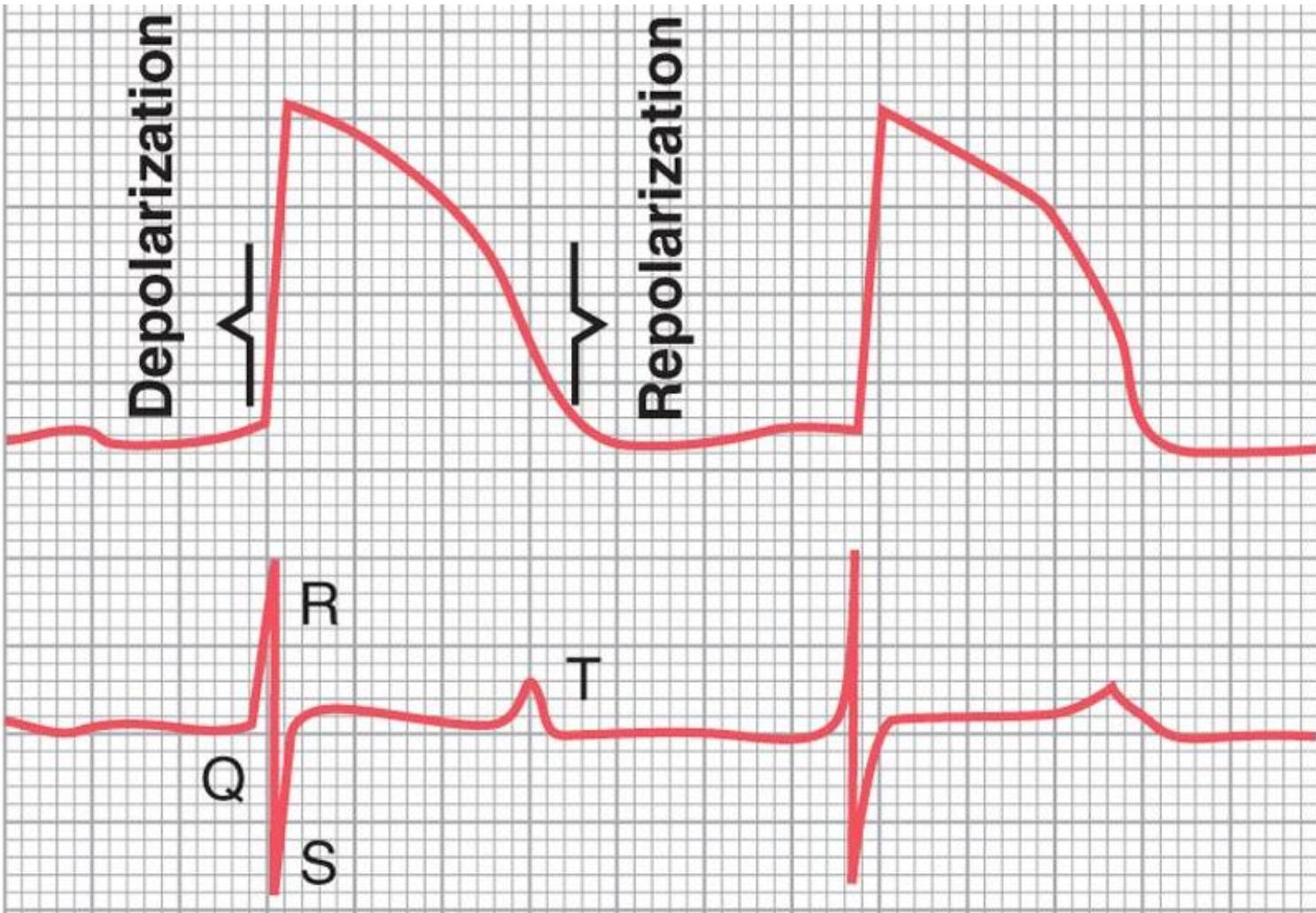
- ***Information obtained from ECG:***
 - *Anatomical orientation* of the heart.
 - *Relative size* of chambers.
 - *Rhythm and conduction disturbance.*
 - Extent, location and progress of *ischemic damage.*
 - Electrolyte disturbance.
 - Influence of drugs.
 - ***HR=1/cycle length.***
- To record ECG, the surface of the body is connected to the ECG machine by means of ECG leads.



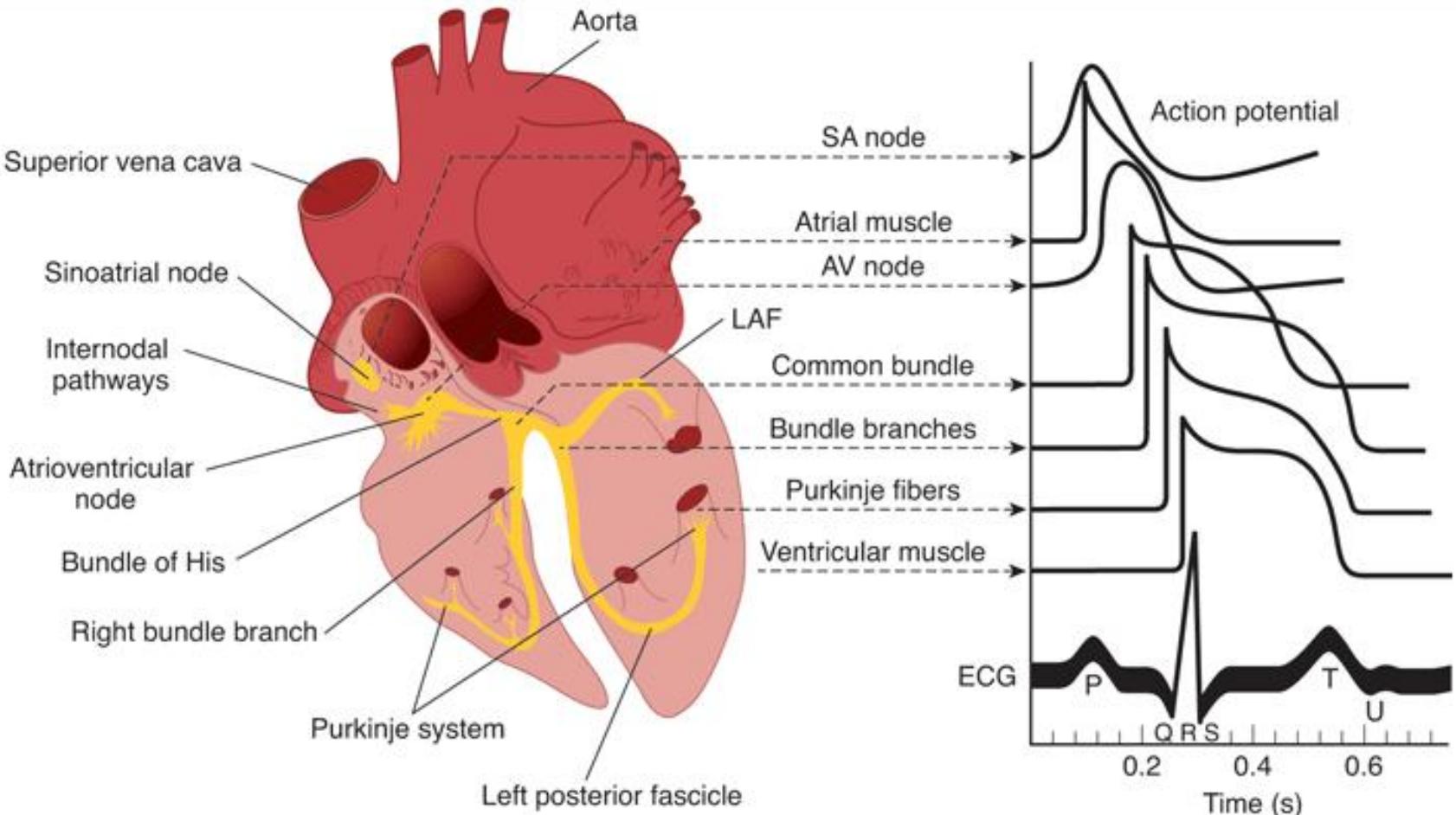
Normal electrocardiogram

Electrocardiogram ECG





Relation of the Monophasic Action Potential of Ventricular Muscle to the QRS and T Waves in the Standard Electrocardiogram



The action potentials and ECG

Atrial excitation

Ventricular excitation

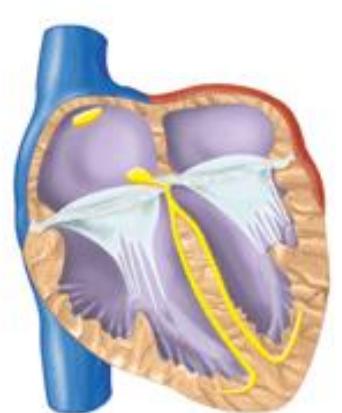
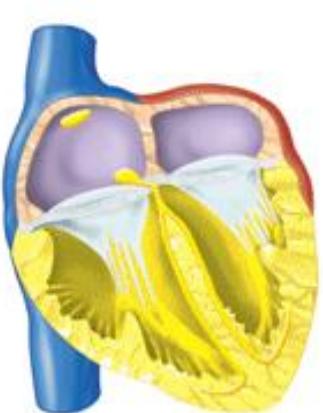
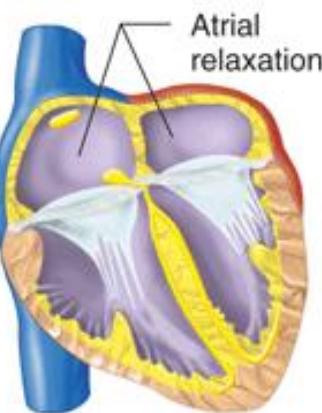
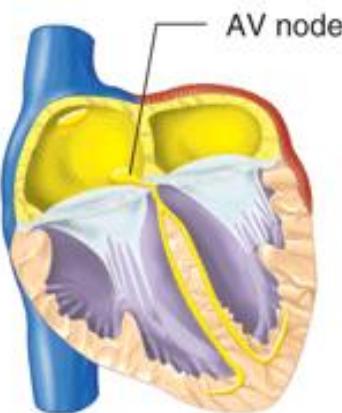
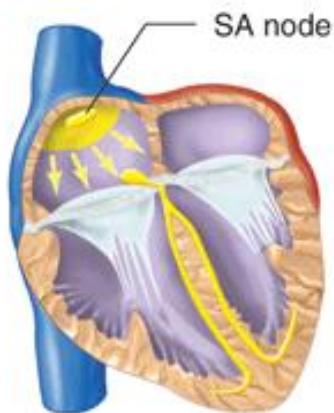
Ventricular relaxation

Begins

Complete

Begins

Complete



Time

Time

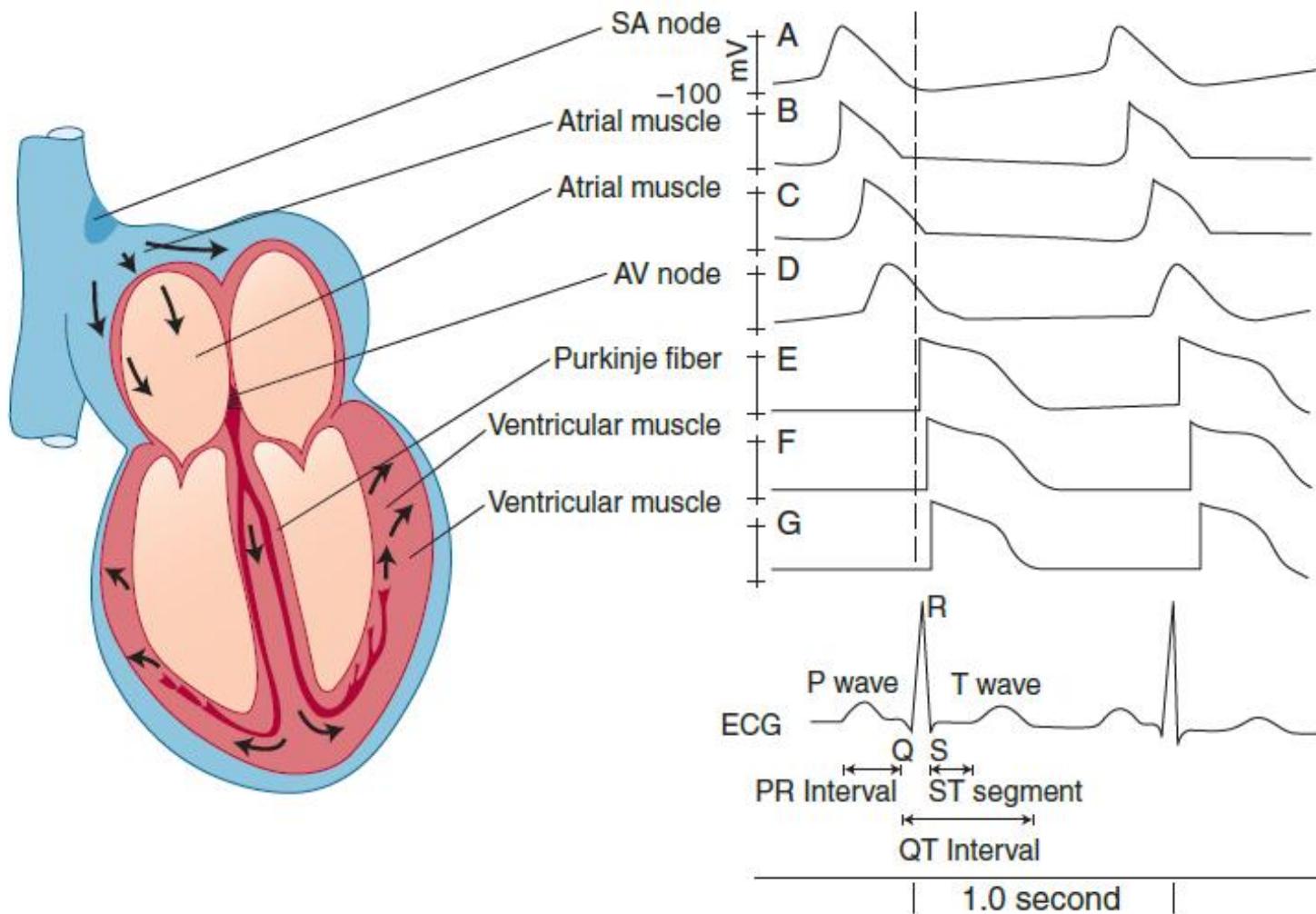
Time

Time

Time

Electrocardiogram

Normal spread of electrical activity in the heart



Intervals and segments

➤ PR intervals

- It is the time interval between the onset of P wave & the onset of Q wave.
- Arterial depolarization + conduction through AV node.
- Duration : 0.12 - 0.20 sec
- PR interval prolonged in heart block.

➤ **QT interval**

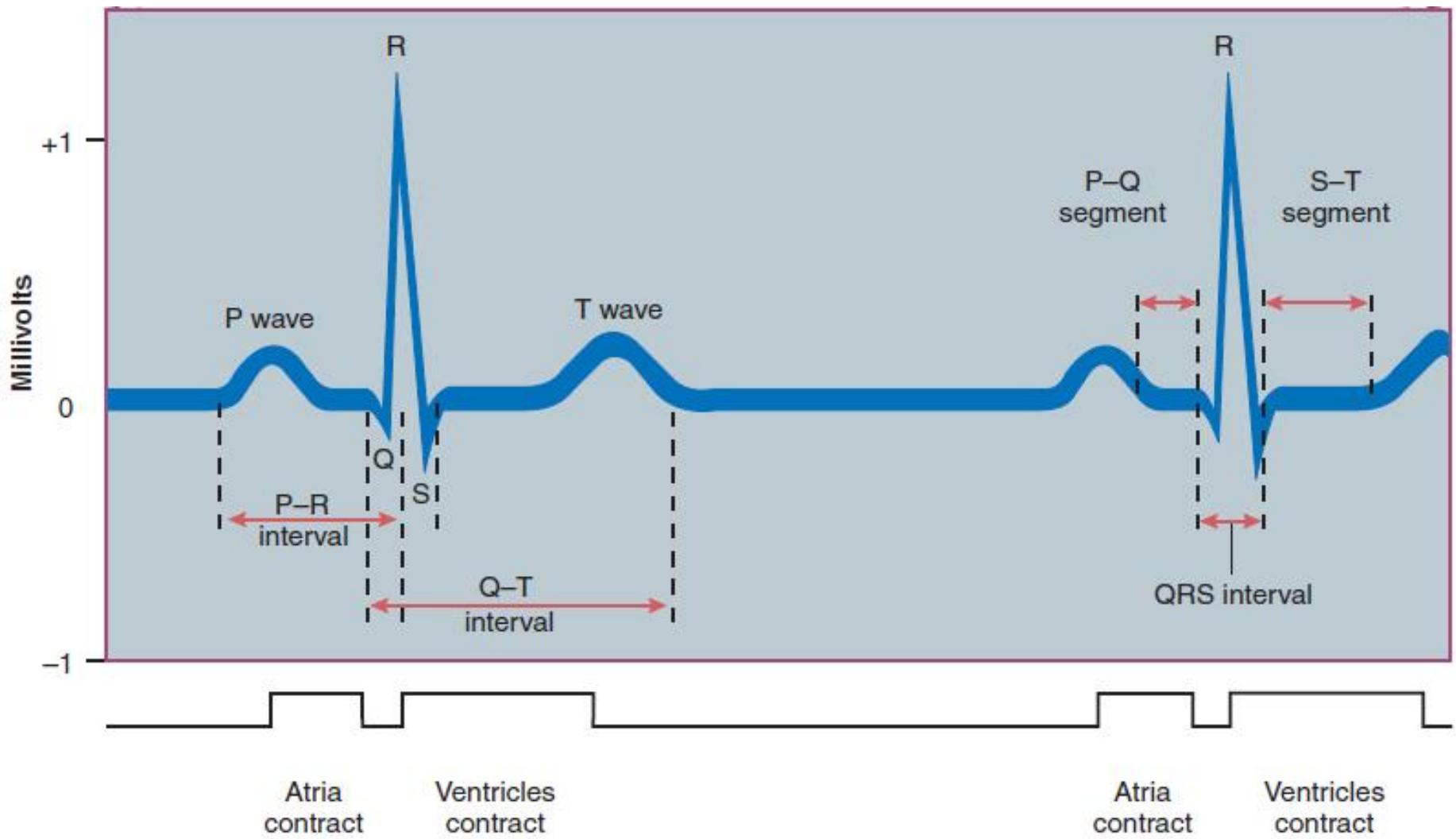
- Ventricular depolarization plus ventricular repolarization.
- Duration : 0.40 to 0.43 sec

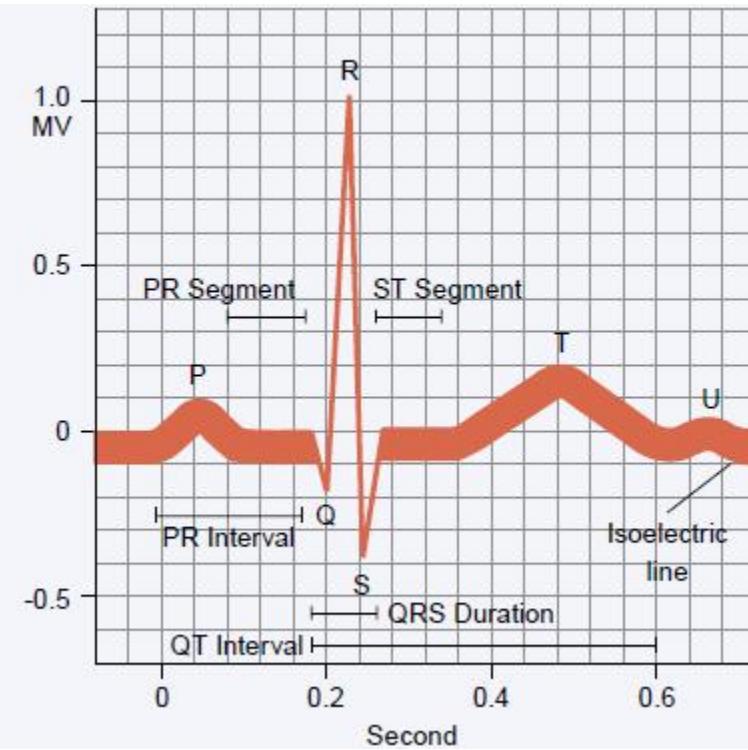
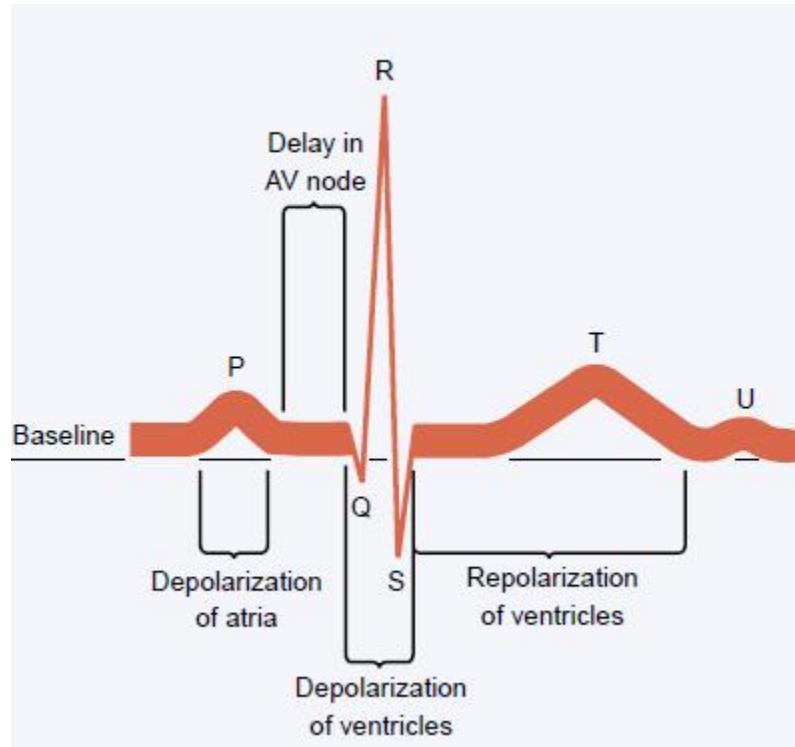
➤ **ST interval**

- Ventricular repolarization
- Duration : 0.32 sec

➤ **ST segment**

- It is the time interval between the end of S wave & the onset of T wave.

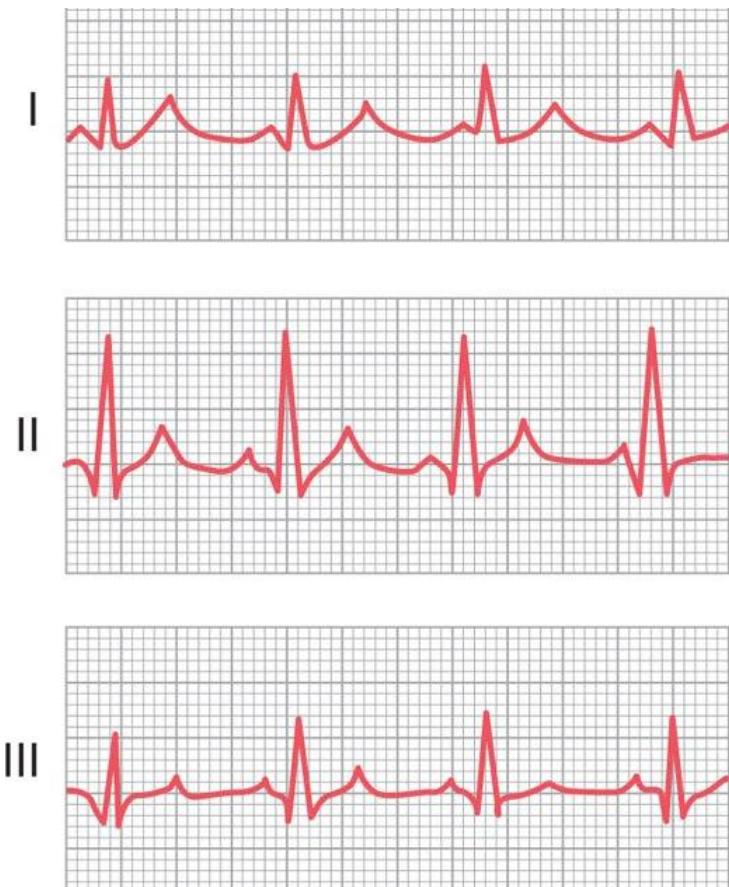
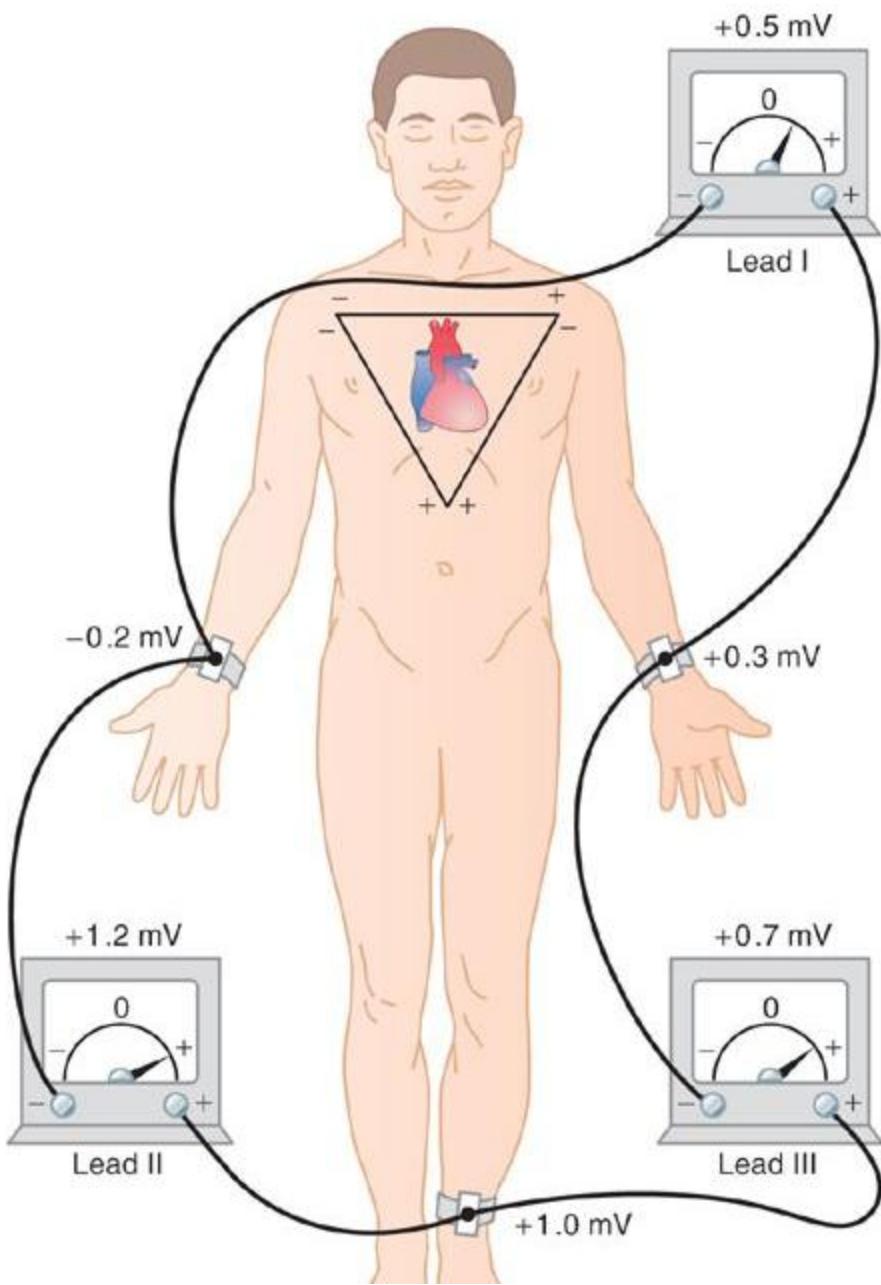




Electrographic leads

Bipolar Leads

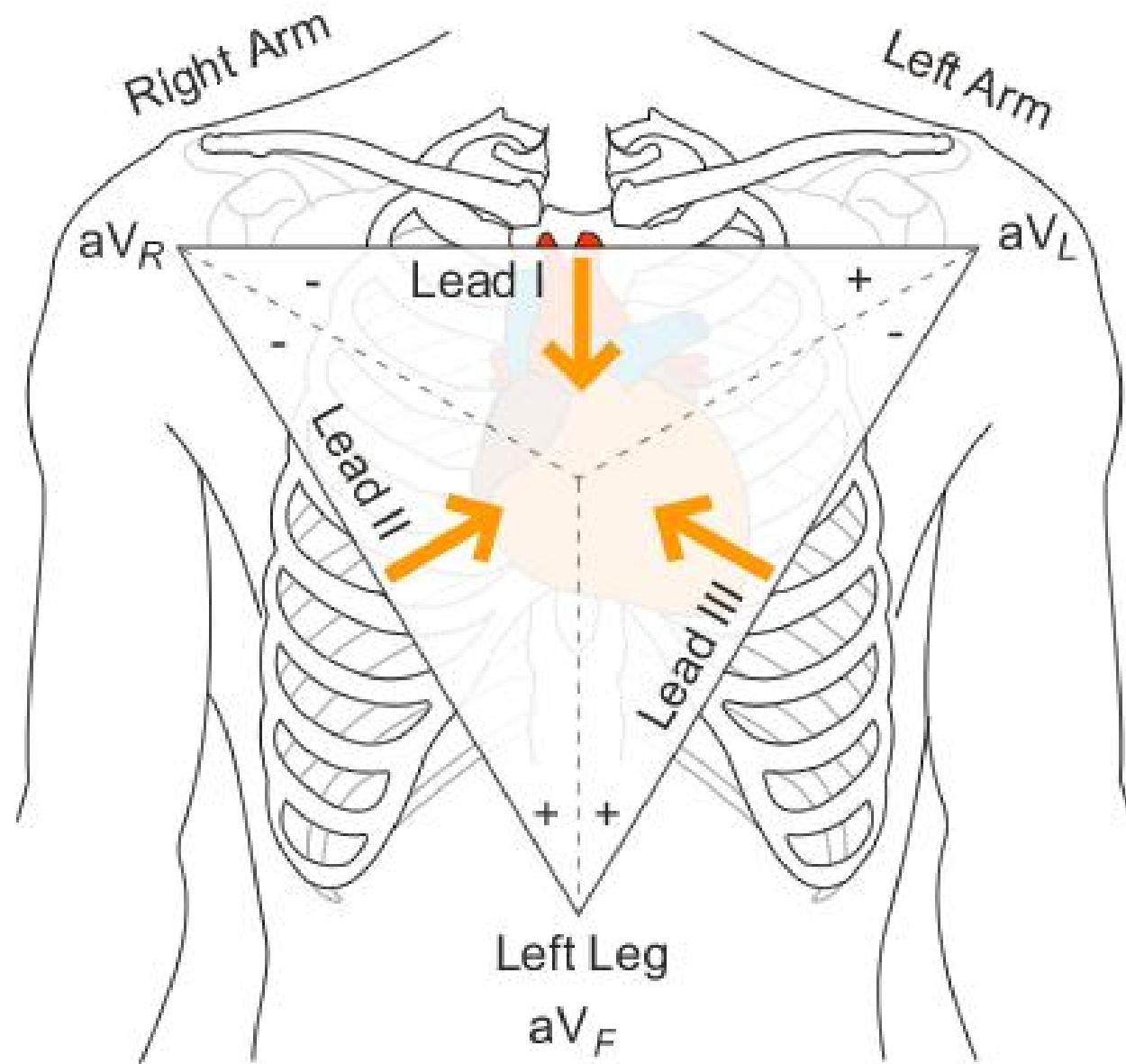
- Bipolar leads were used before unipolar leads were developed.
- The **standard limb leads**—leads I, II, and III—each record the differences in potential between two limbs.
 - Lead I – the electrodes are on the left arm and right arm.
 - Lead II - on the right arm and left leg
 - Lead III - the electrodes are on the left arm and left leg

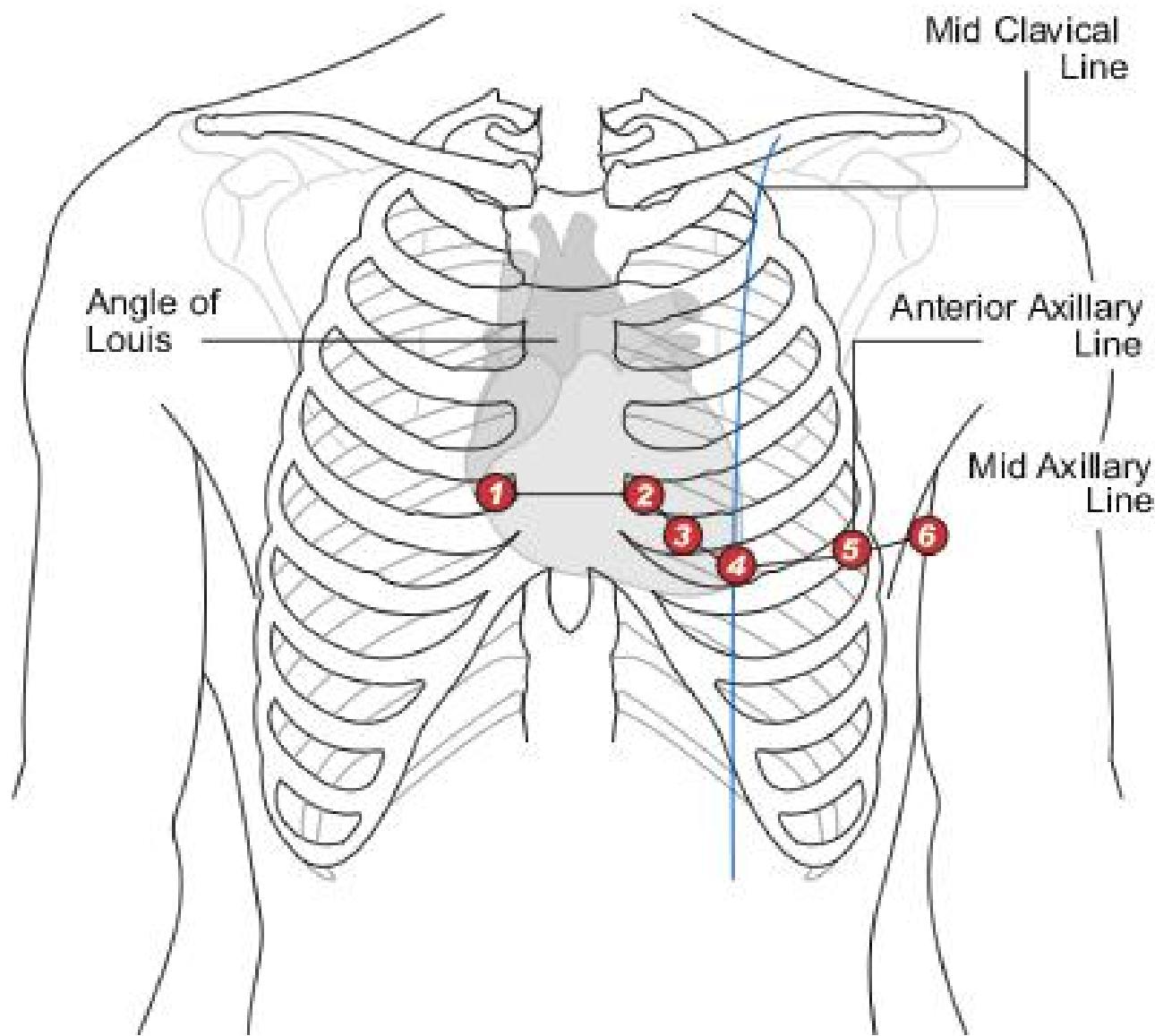


- **Unipolar Leads**

- There are six unipolar chest leads (precordial leads) designated V₁–V₆.
- Three unipolar limb leads: VR (right arm), VL (left arm), and VF (left foot).
- The augmented limb leads are recordings between one limb and the other two limbs.

Einthoven's triangle - Line of site of the bipolar leads





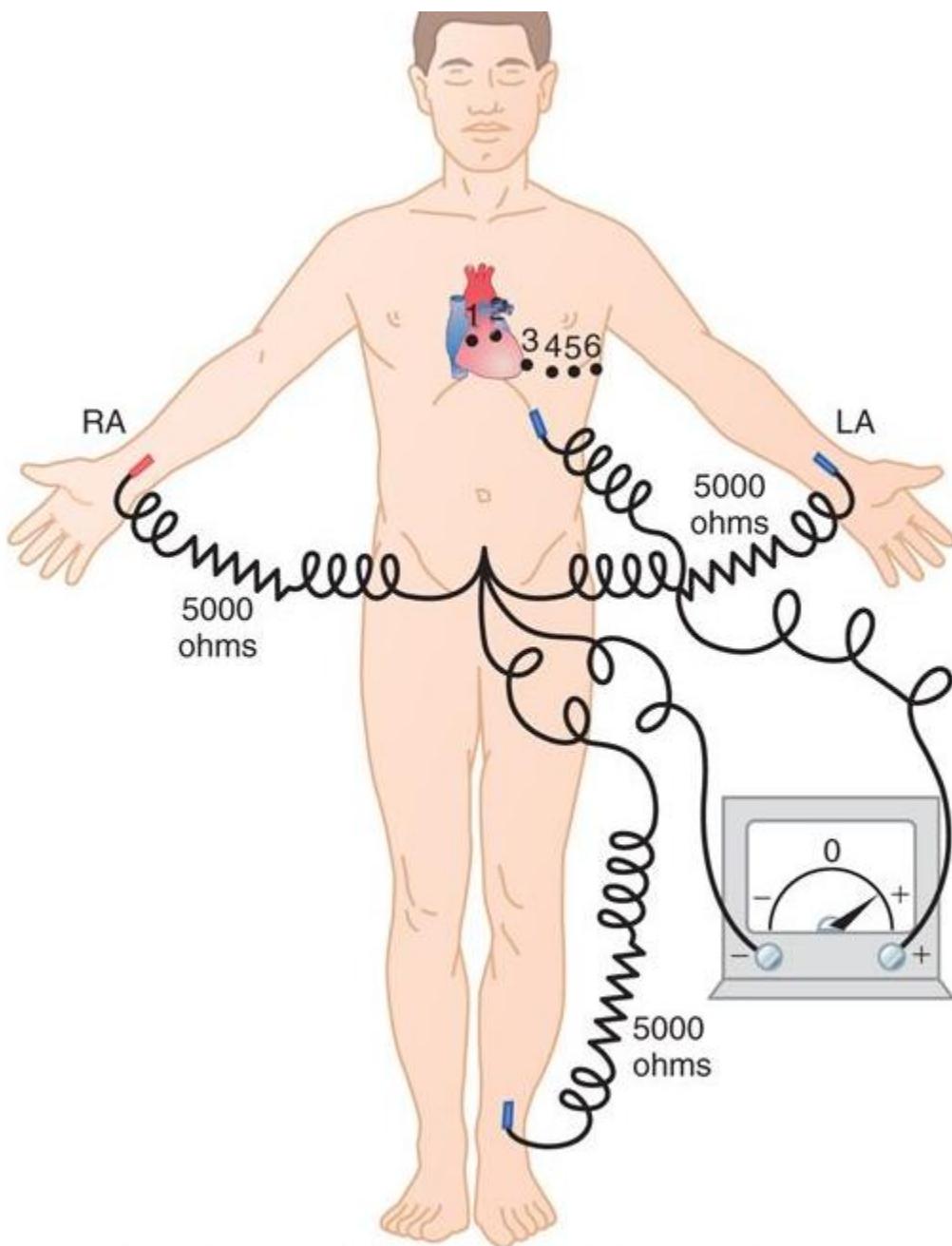
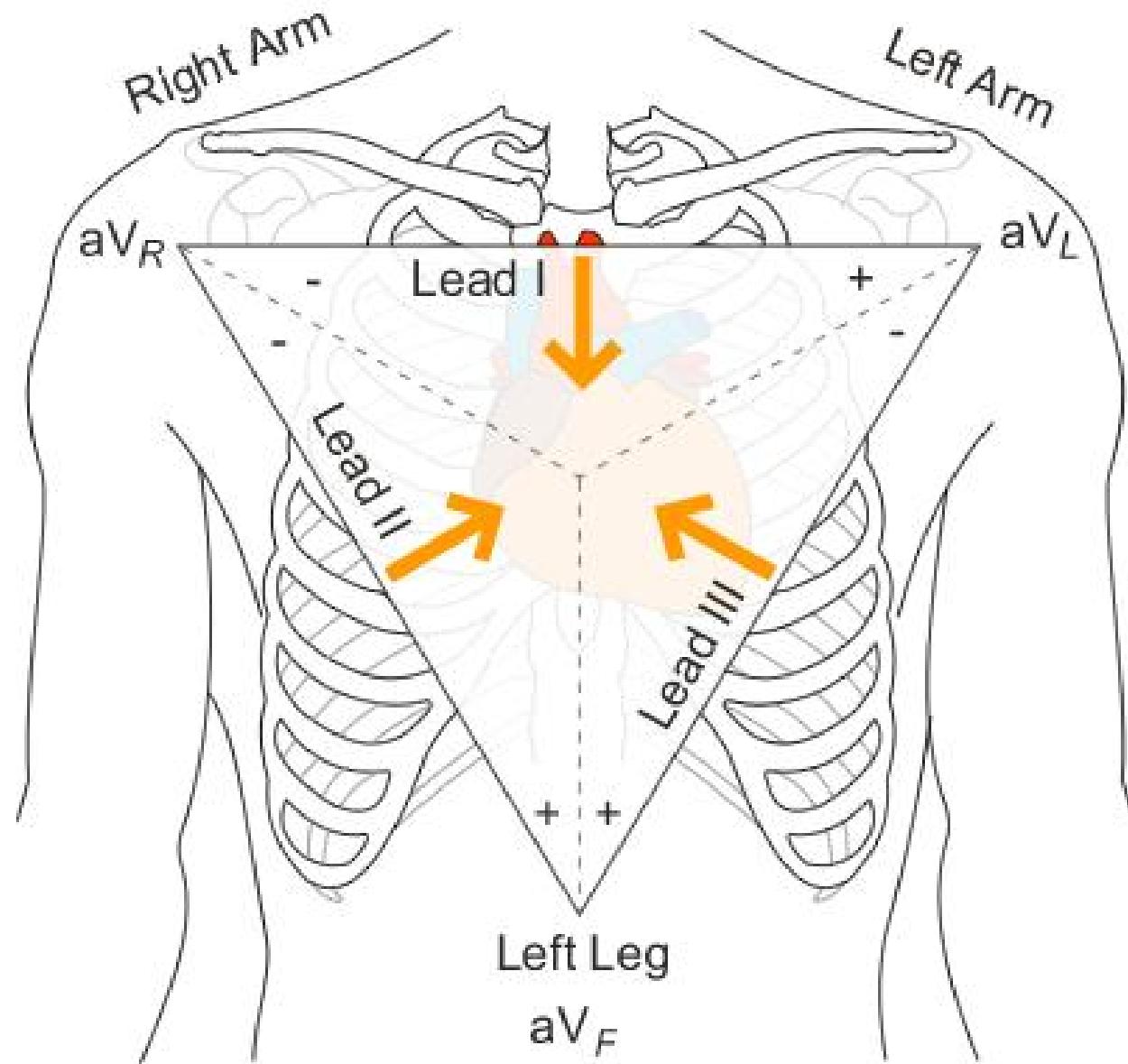


TABLE 12-2 Electrocardiography Leads

NAME OF LEAD	ELECTRODE PLACEMENT*		
<i>Standard Limb Leads</i>	<i>Reference (-) Electrode</i>	<i>Recording (+) Electrode</i>	
Lead I	Right arm	Left arm	
Lead II	Right arm	Left leg	
Lead III	Left arm	Left leg	
<i>Augmented Limb Leads</i>			
aVR	Left arm and left leg	Right arm	
aVL	Right arm and left leg	Left arm	
aVF	Right arm and left arm	Left leg	
<i>Precordial (Chest) Leads</i>			
V1	Combined limb leads	4 th intercostal space, right of sternum	
V2	" " "	4 th intercostal space, left of sternum	
V3	" " "	5 th intercostal space, left of sternum	
V4	" " "	5 th intercostal space, centered on clavicle	
V5	" " "	5 th intercostal space, left of V4	
V6	" " "	5 th intercostal space, under left arm	

Einthoven's triangle - Line of site of the bipolar leads





V₁

V₂

V₃

V₄

V₅

V₆



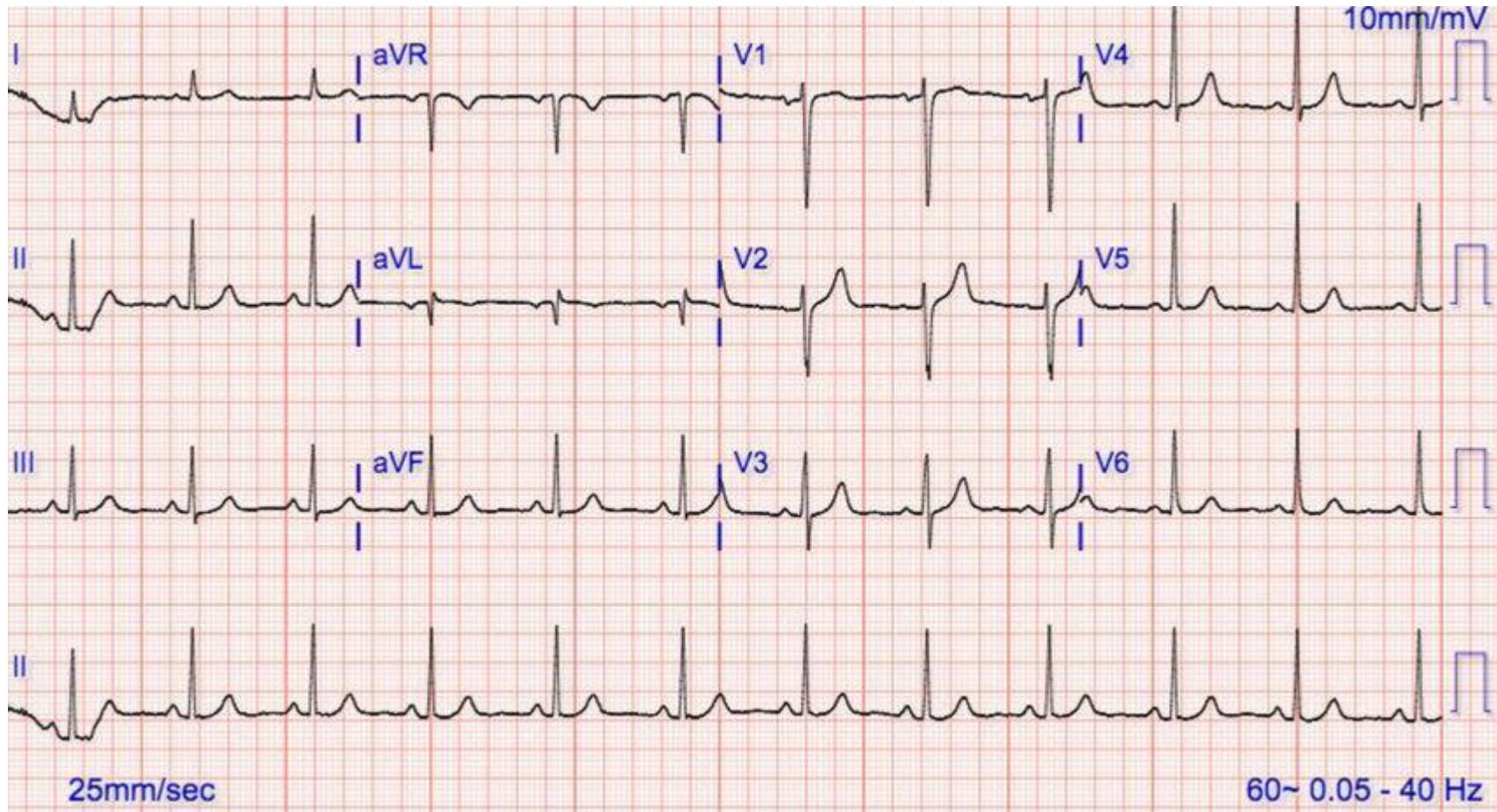
aVR



aVL



aVF



Characteristic of normal cardiac rhythms

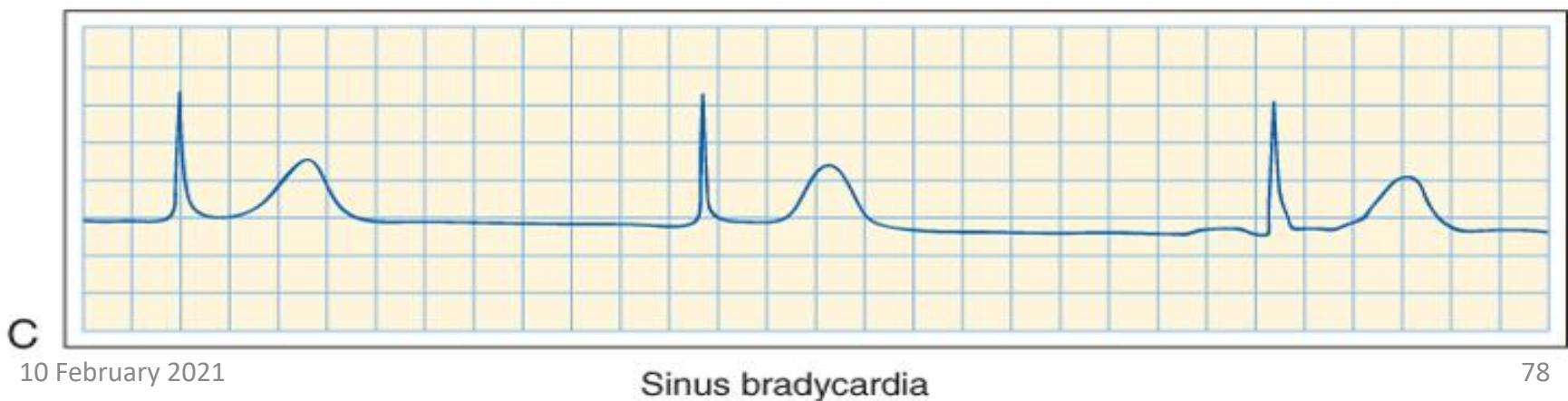
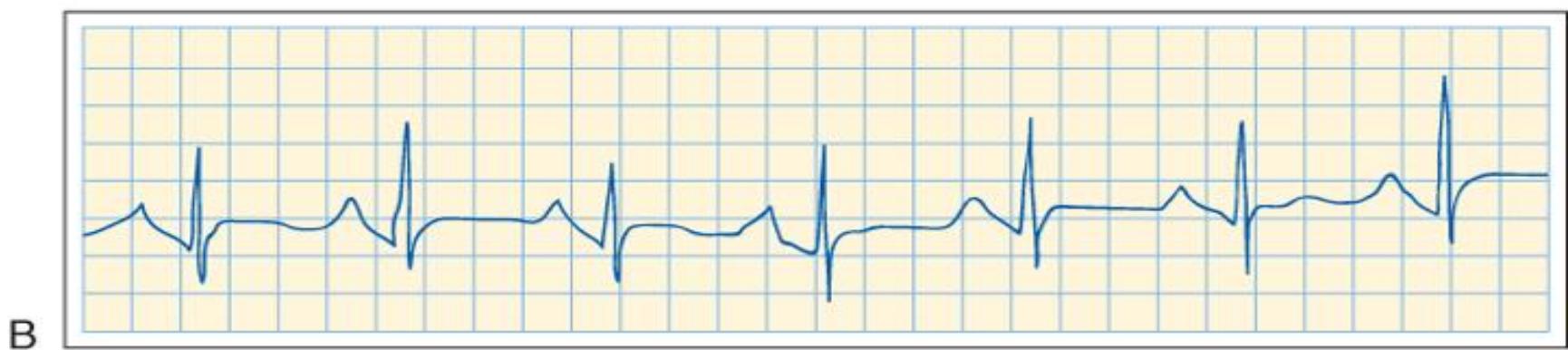
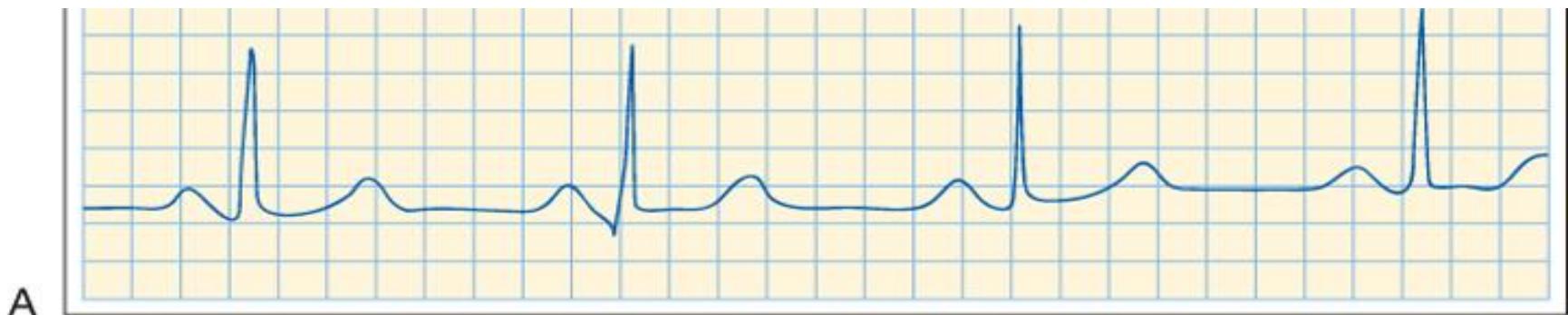
- Frequency of QRS complexes are ~ 1 per second.
- Each QRS is followed by a P wave of normal configuration indicating SA node rhythms
- PR interval is less than 200msec. indicating proper conduction delay of impulse spread through the AV node

Cardiac arrhythmias:

1. Pacemaker abnormalities
2. Conduction abnormalities (cardiac block)

Pacemaker abnormalities:

- *Sinus tachycardia*: HR above 100bpm
- *Sinus bradycardia*: HR below 60bpm
 - Is normal phenomenon in well trained athletes.
- *Ectopic beats*: originate in pacemaker cells outside the sinus



Regularity: Regularity can be done by measuring the "R-R interval". To measure the R-R interval, place the edge of a piece of paper along the line of the rhythm. Compare this measurement with the next R-R interval.



$$\text{Heart Rate} = \frac{300}{4} = 75 \text{ per minute}$$

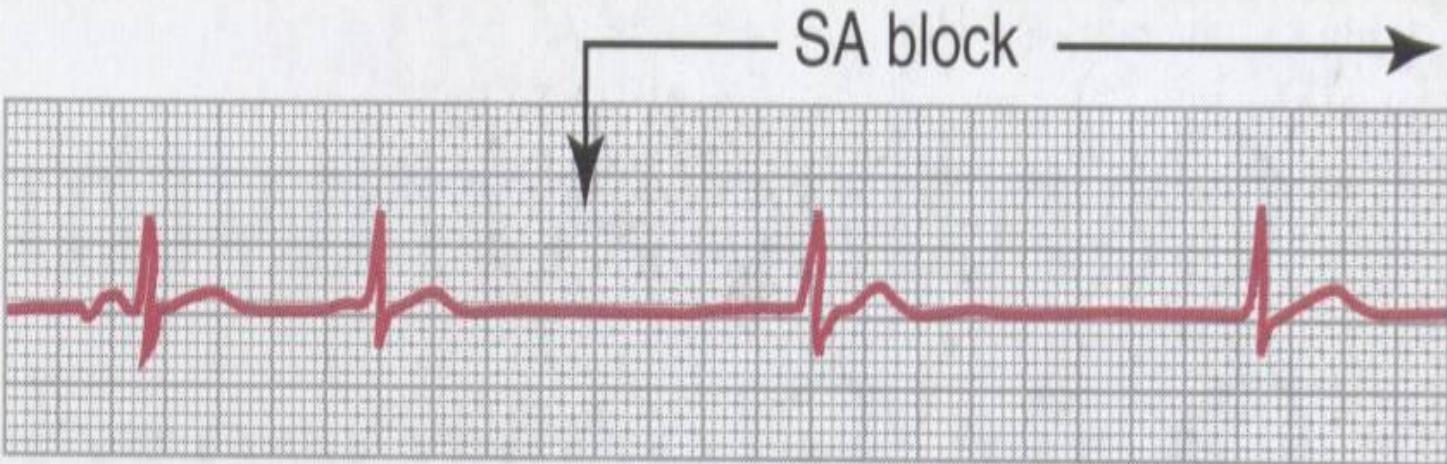


Figure 13–4

Sinoatrial nodal block, with A-V nodal rhythm during the block period (lead III)



Figure 13-5

Prolonged P-R interval caused by first degree A-V heart block (lead II).

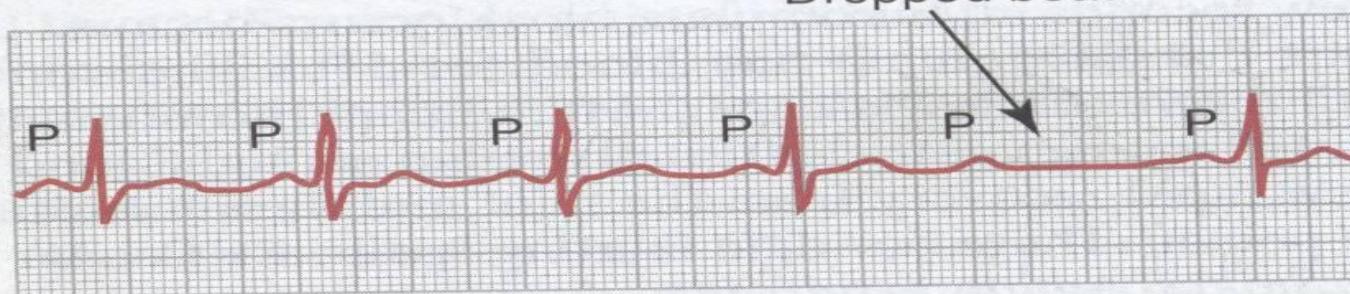


Figure 13–6

Second degree A-V block, showing occasional failure of the ventricles to receive the excitatory signals (lead V₃).

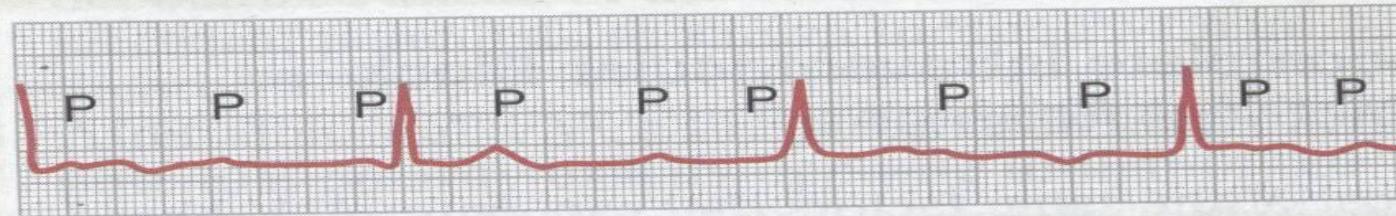


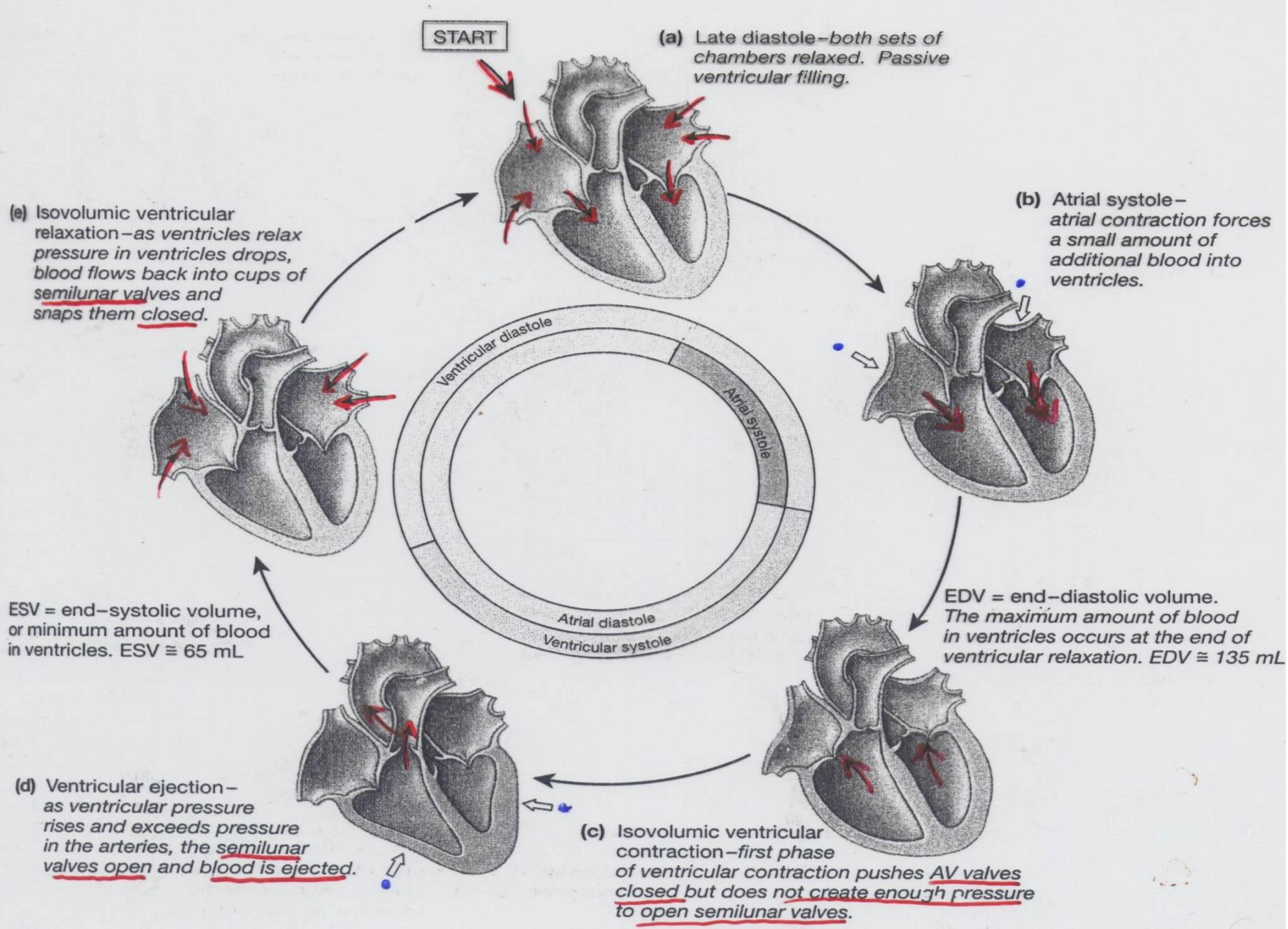
Figure 13–7

Complete A-V block (lead II).

Cardiac cycle

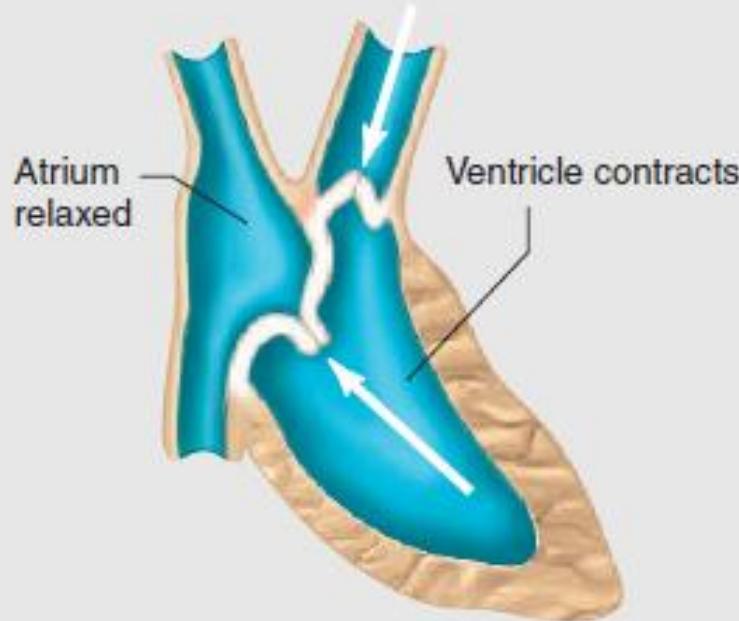
- The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the *cardiac cycle*
- The frequency of the cardiac cycle is the heart beat.
- An average normal heart beat is 70-80 beats/minute
- With a heart rate of 75 beats / min, the duration of cardiac cycle is $60/75 = 0.8$ sec.

- The cardiac cycle has two phases: **systole** and **diastole**.
- The contraction of a heart chamber is called a **systole**.
- The time during which a heart chamber is relaxing and filling with blood is termed **diastole**.



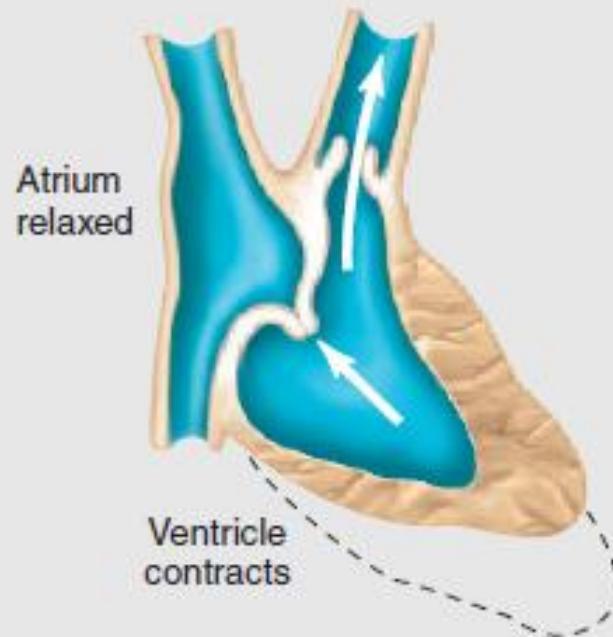
(a) Systole

Isovolumetric ventricular contraction



Ventricular ejection

Blood flows out of ventricle



AV valve:

Closed

Closed

Aortic and

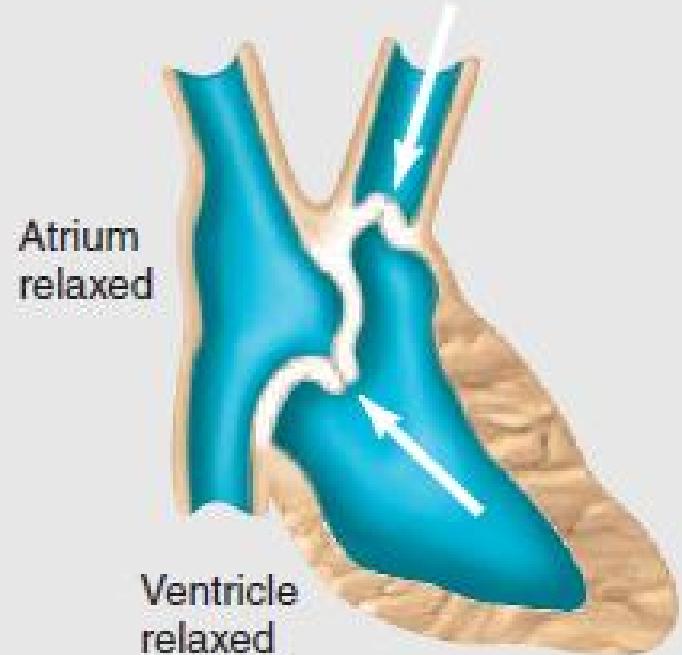
pulmonary valves:

Closed

Open

(b) Diastole

Isovolumetric ventricular relaxation

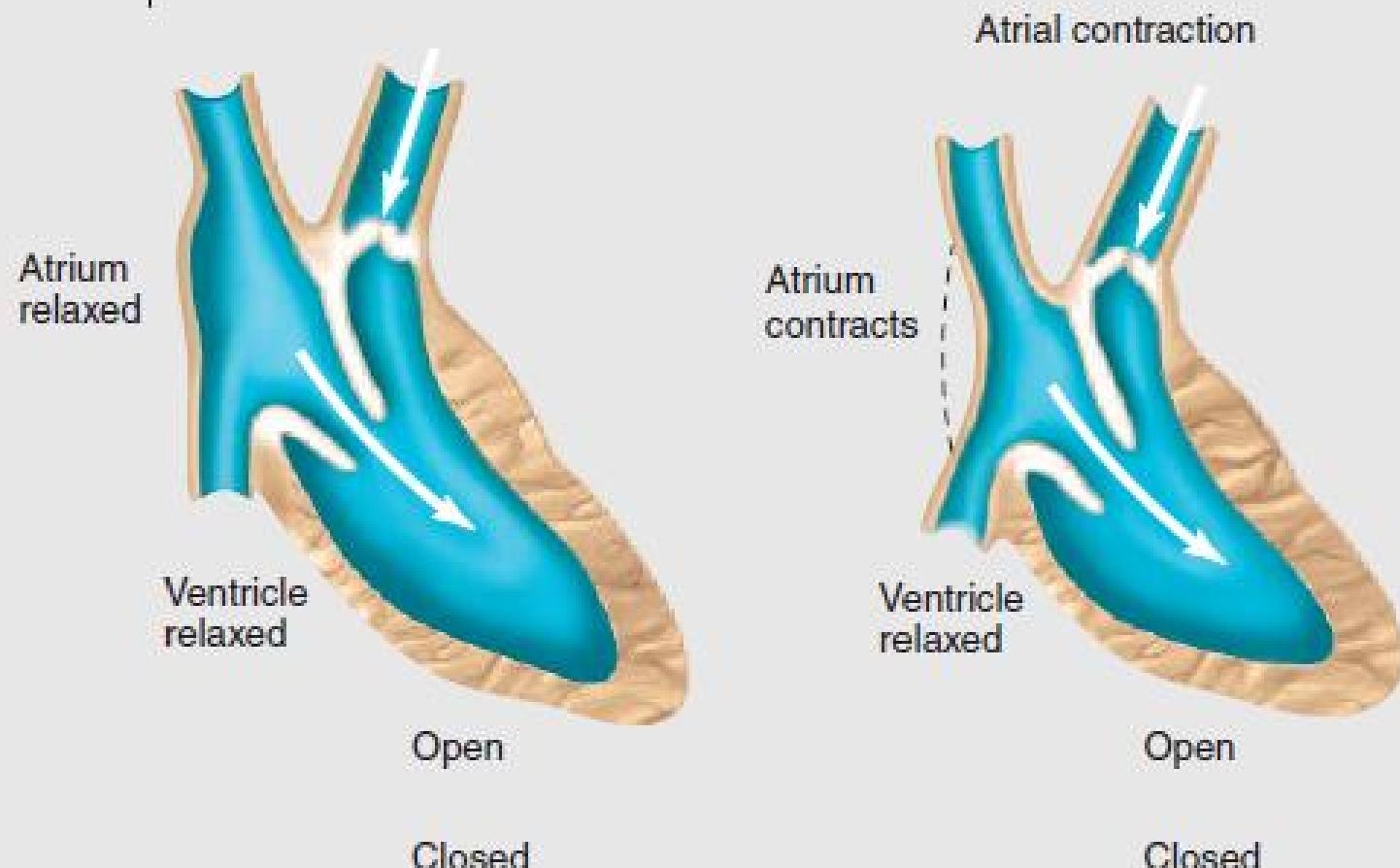


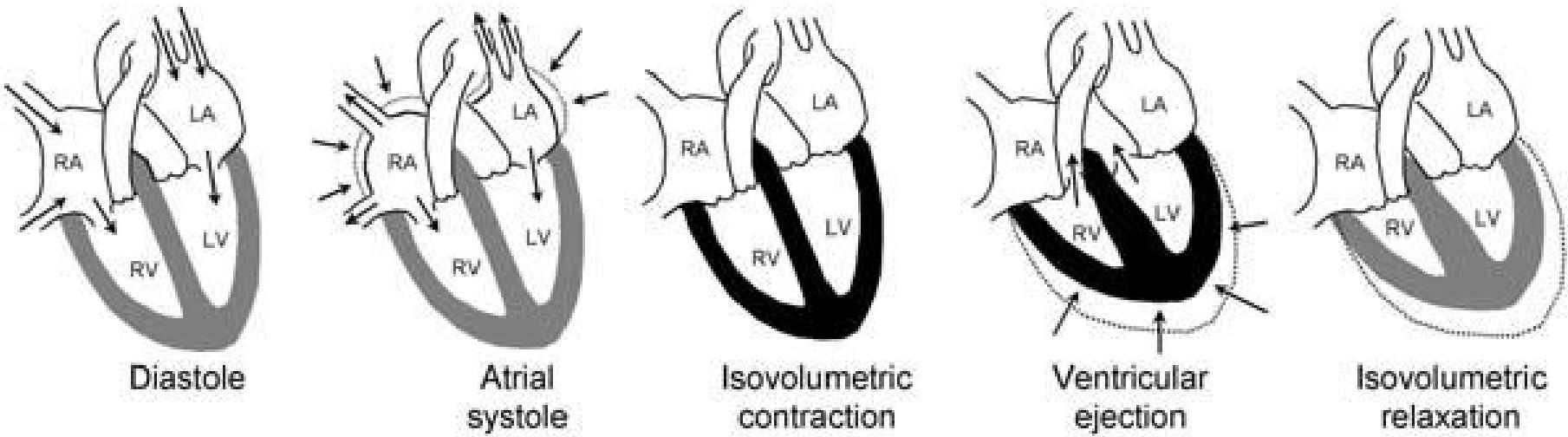
AV valve: Closed

Aortic and pulmonary valves: Closed

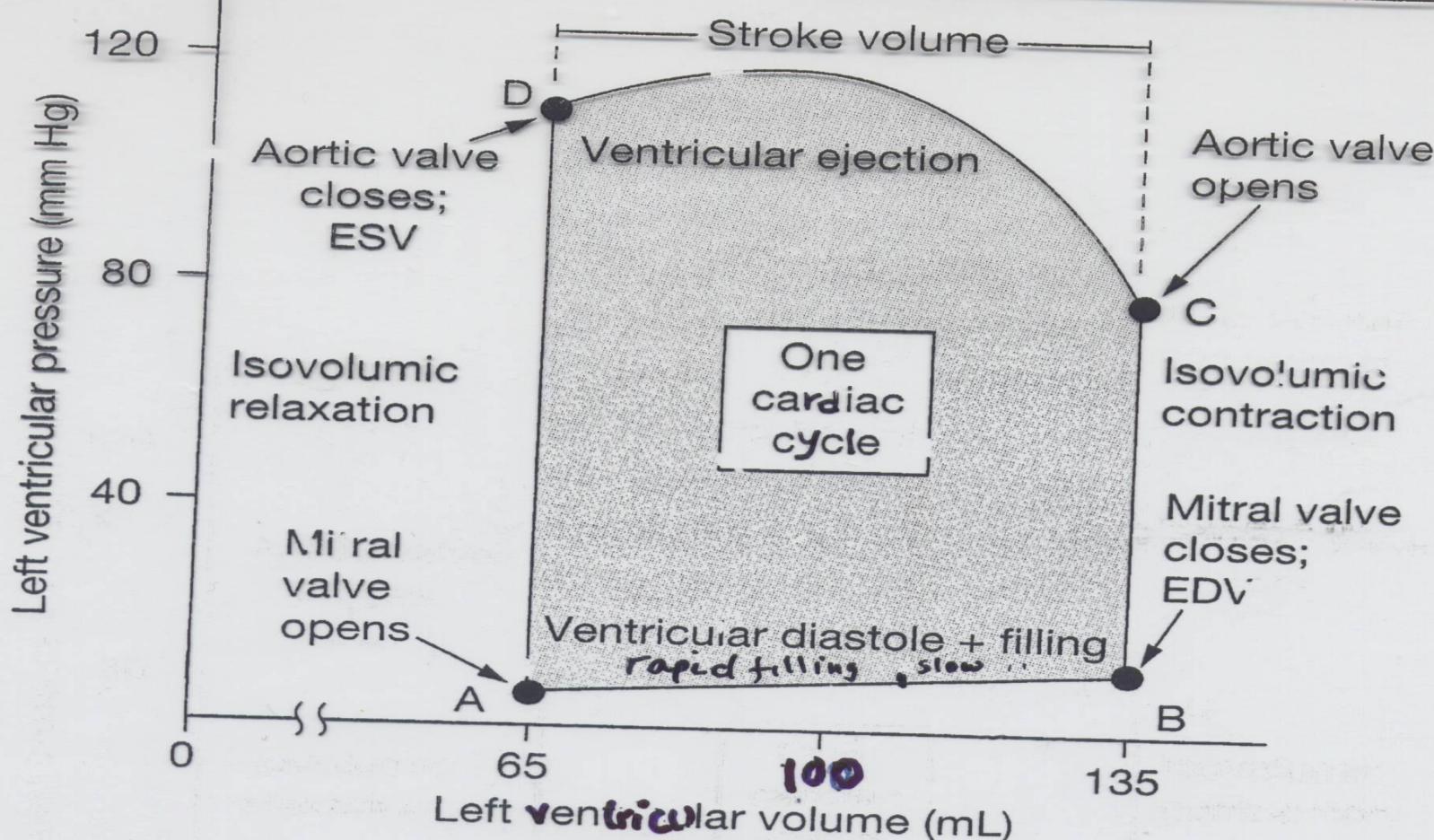
Ventricular filling

Blood flows into ventricle

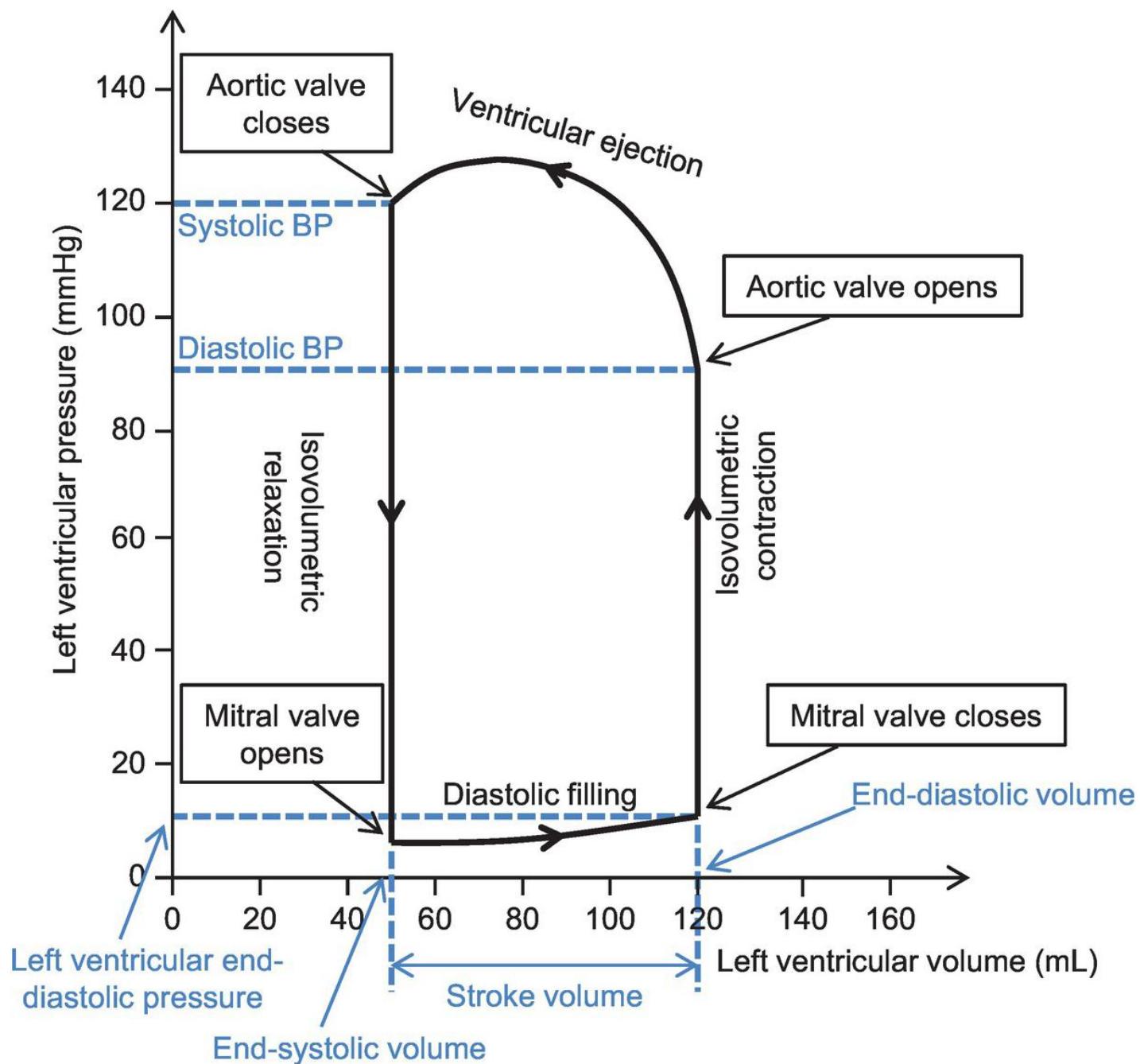


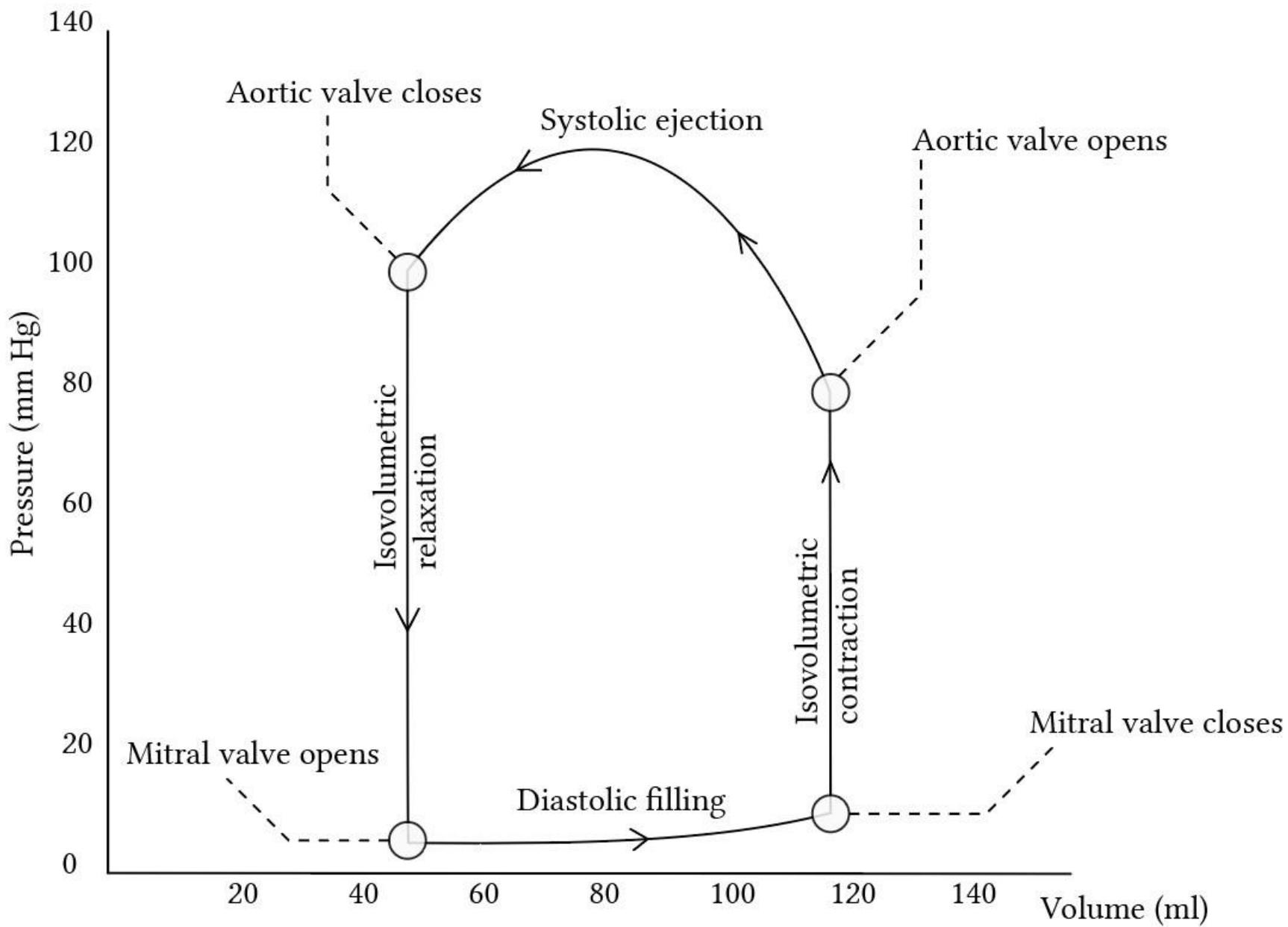


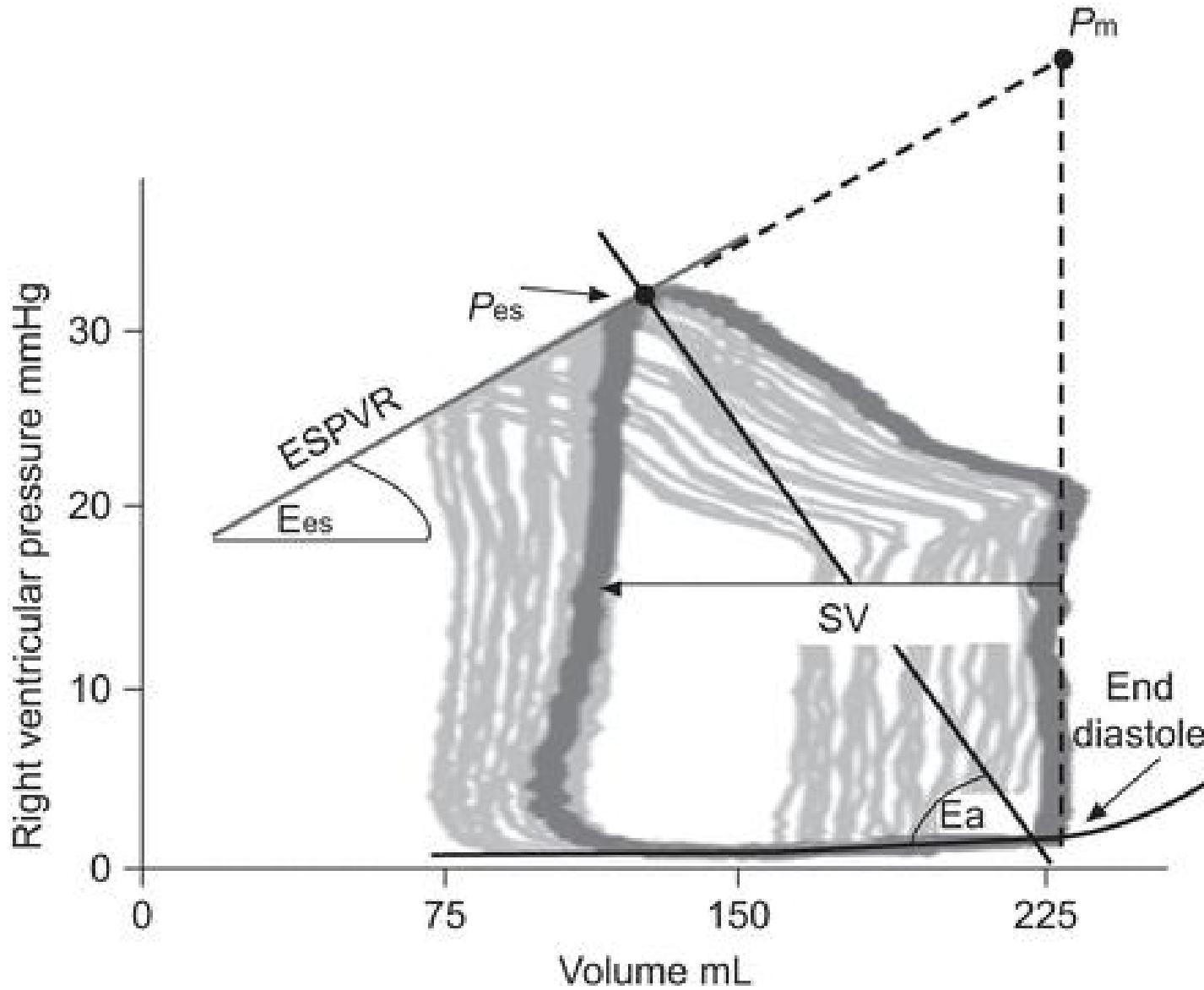
Mechanical events in the heart during a single cardiac cycle. Blood flow and wall motion are indicated by arrows.

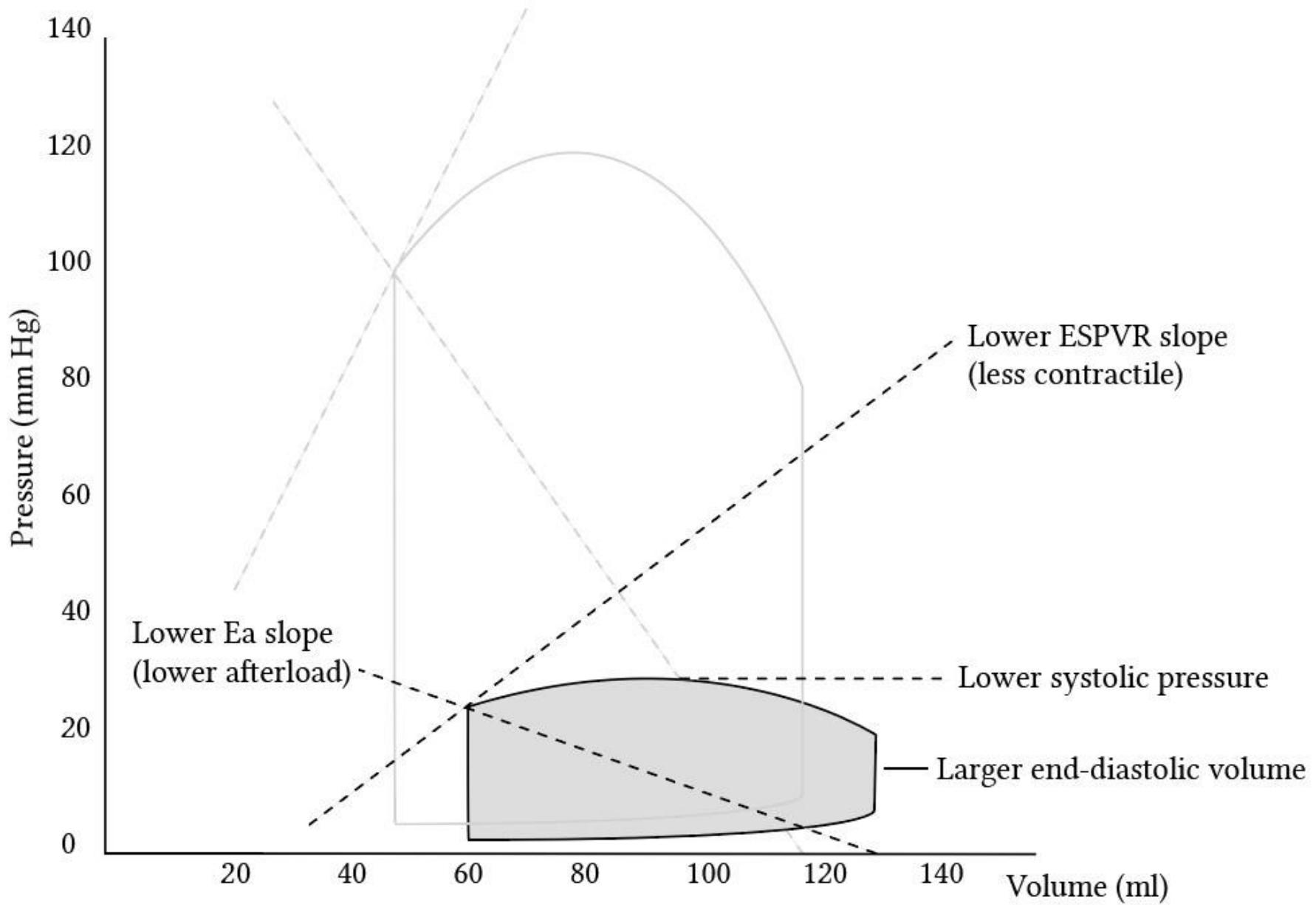


- A → B: Passive filling and atrial contraction
- B → C: Isovolumic contraction
- C → D: Ejection of blood into aorta
- D → A: Isovolumic relaxation

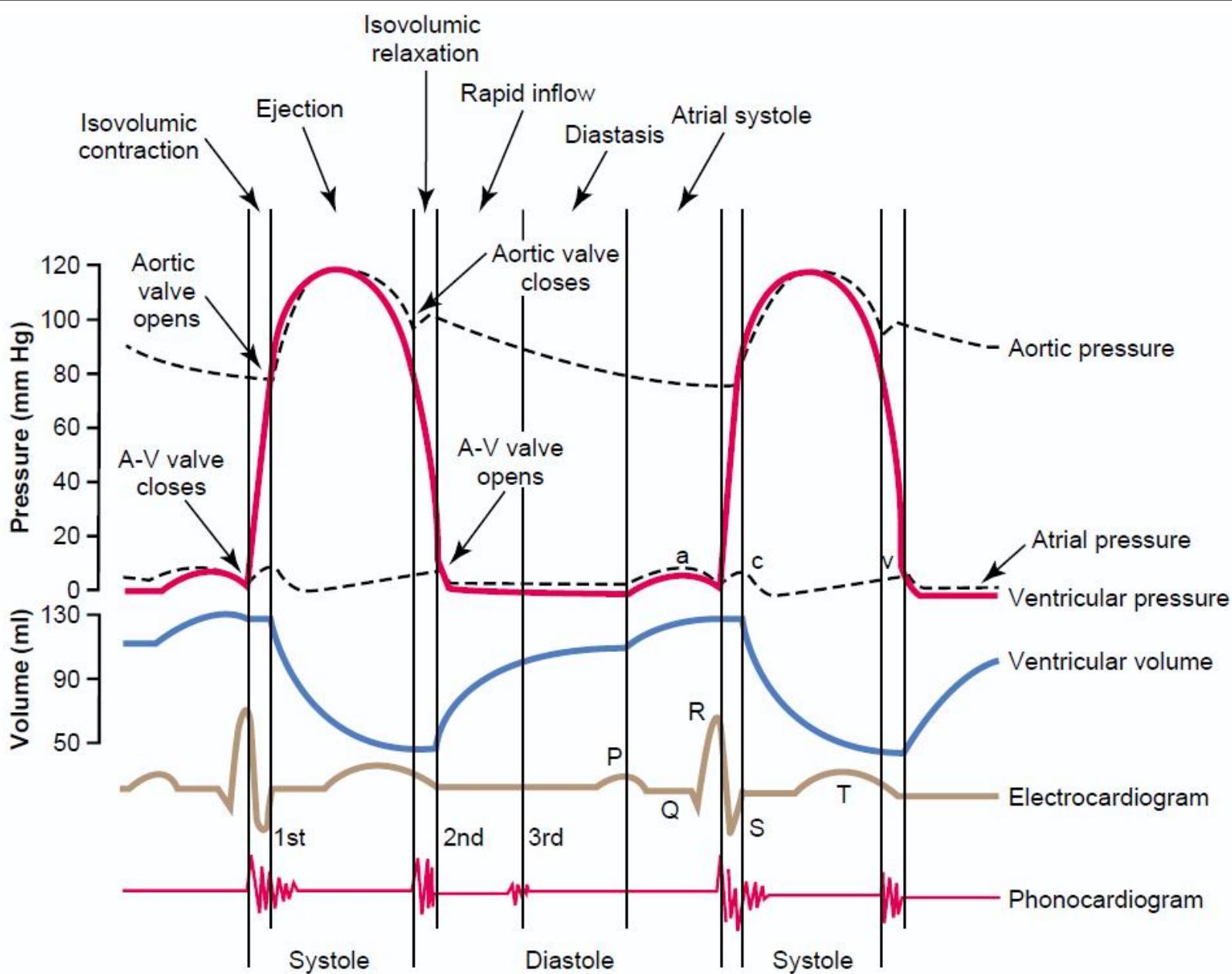








- **Heart sounds:** associated (usually) with valve closure.
 - **First heart sound** -Closure of AV valves.
Occurs at beginning of isovolumic contraction.
 - **Second heart sound** -Closure of Aortic & Pulmonic valves (Semilunar valves).
 - **Third heart sound (sometimes)** -due to rapid ventricular filling.
 - **Fourth heart sound (occasionally)**- during atrial contraction.



Cardiac Output

- *Cardiac output* is the quantity of blood pumped into the aorta each minute by the heart.
- This is also the quantity of blood that flows through the circulation.
- *Venous return* is the quantity of blood flowing from the veins into the right atrium each minute

The cardiac output (CO)

$$CO = HR \times SV$$

- SV: volume ejected during each beat , depends on venous return, can be equal to venous return
 - CO (at rest) = 5-6 L/min
 - HR=72 beats/min , SV=0.07L/min (70ml) .
- Cardiac out put is all blood pumped around circuit once each min.

- Cardiac output varies widely with the level of activity of the body.
- The following factors directly affect cardiac output:
 1. The basic level of body metabolism
 2. Exercise
 3. Age and
 4. Size of the body.

Factors controlling cardiac output

1. Intrinsic: results from normal functional characteristics of heart - **contractility, preload and after load**
2. Extrinsic: involves neural and hormonal control- Autonomic Nervous system.

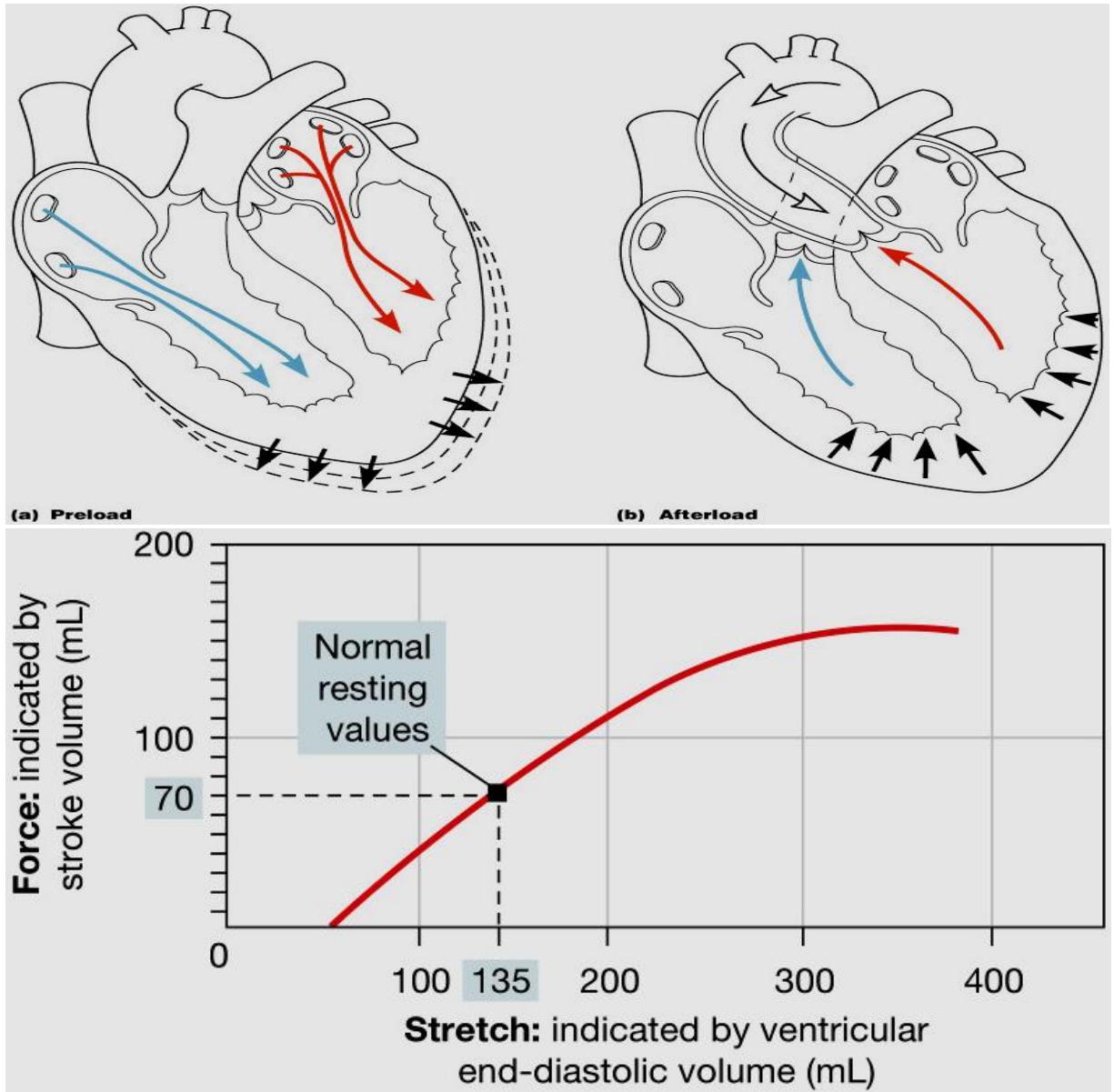
1. Intrinsic

a. Preload: the degree of myocardial stretch before contraction begins (related to sarcomere length).

- As sarcomere length increases (up to an optimum length), stretch of the ventricular wall increases, contraction of force increases and so does the stroke volume.
- This relationship between stretch and force described by Frank Starling Law.

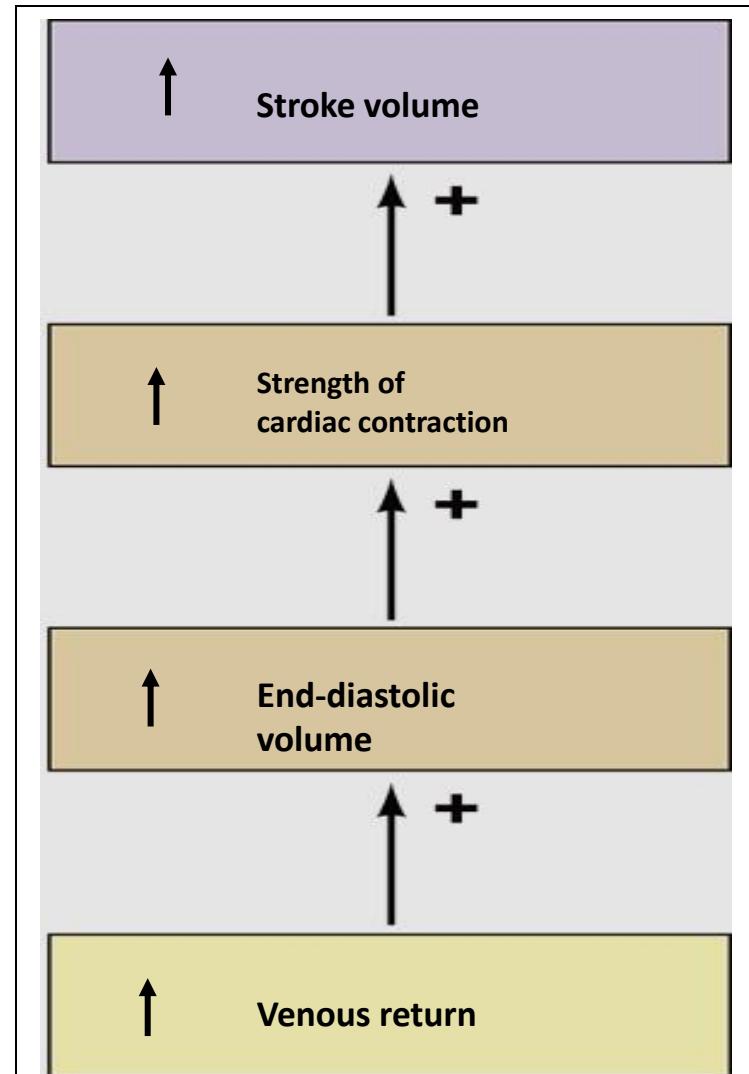
Frank-Starling Law

- ↑preload →
↑stretch of muscle →
↑force of contraction
→ ↑SV



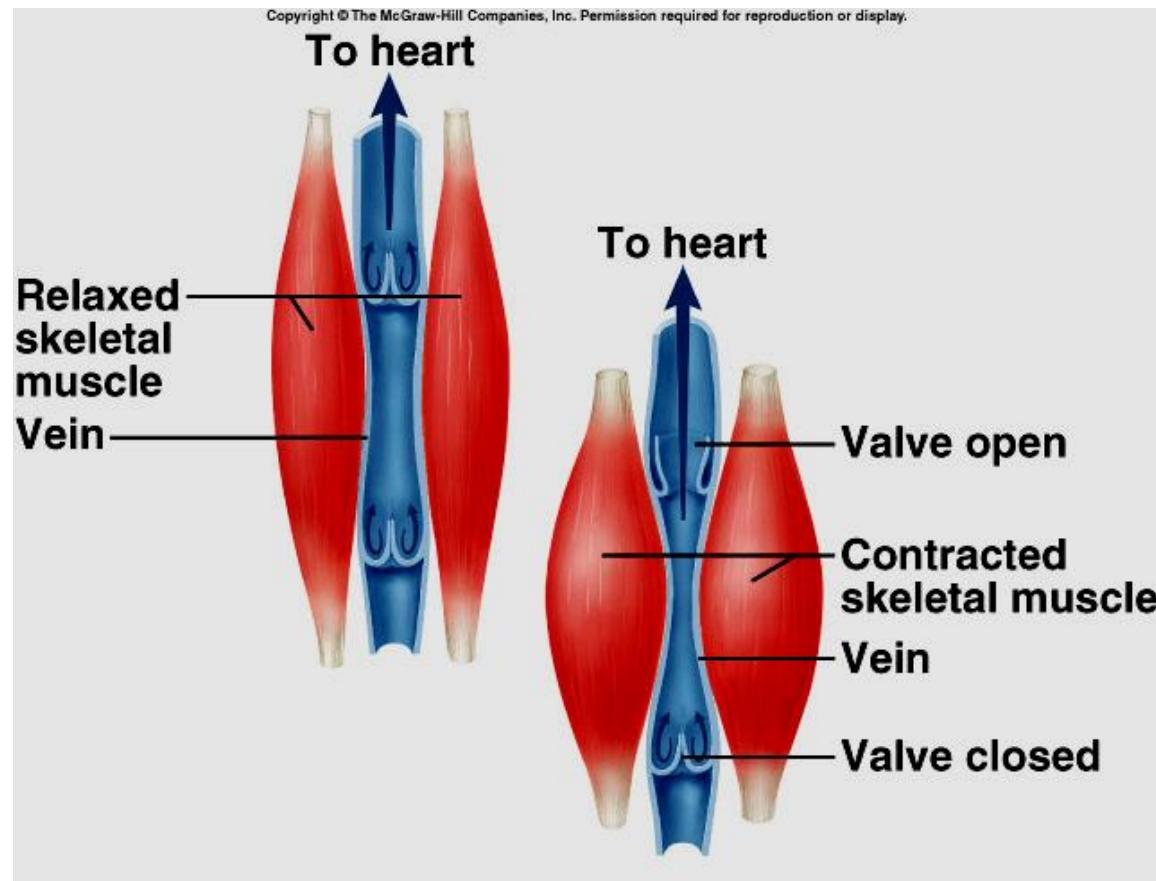
- An increase venous return, increase EDV, so that increase SV.

- **Venous Return is determined by**
 1. Pressure gradient
 2. Respiratory Pump
 3. Muscular Pump.



1. **Pressure gradient:** Because of the pressure gradient blood flows from lower parts of the body to towards right atrium.
2. **Respiratory Pump:** During inspiration intra abdominal pressure increase that compress the veins to flow blood into the Rt atrium.

3. Muscular Pump - skeletal muscle contraction that squeezes veins (particularly in the legs), compressing them and pushing blood toward the heart.



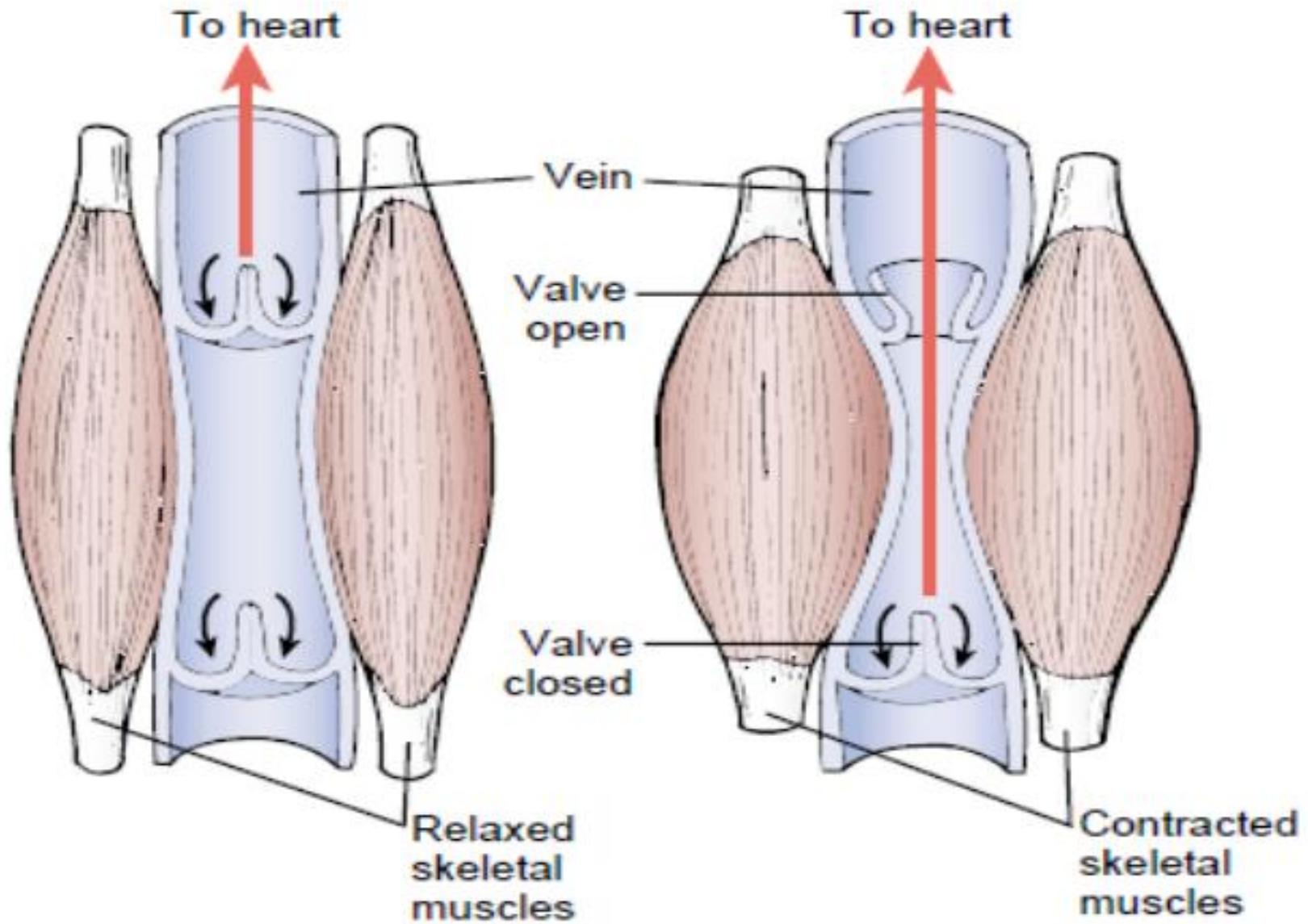
Skeletal muscle pump

A. Standing at rest:

- Valves are open
- Blood flow upward towards the heart

B. **Contraction of the muscle** :compresses the veins so that the increased pressure in the veins drives blood upwards and closes the valves at lower region just below point of muscle contraction.

C. **Immediately after muscle relaxation**, the pressure in the previously compressed venous segment fall, and the reverse pressure gradient causes the upper valve to close (to prevent back flow).

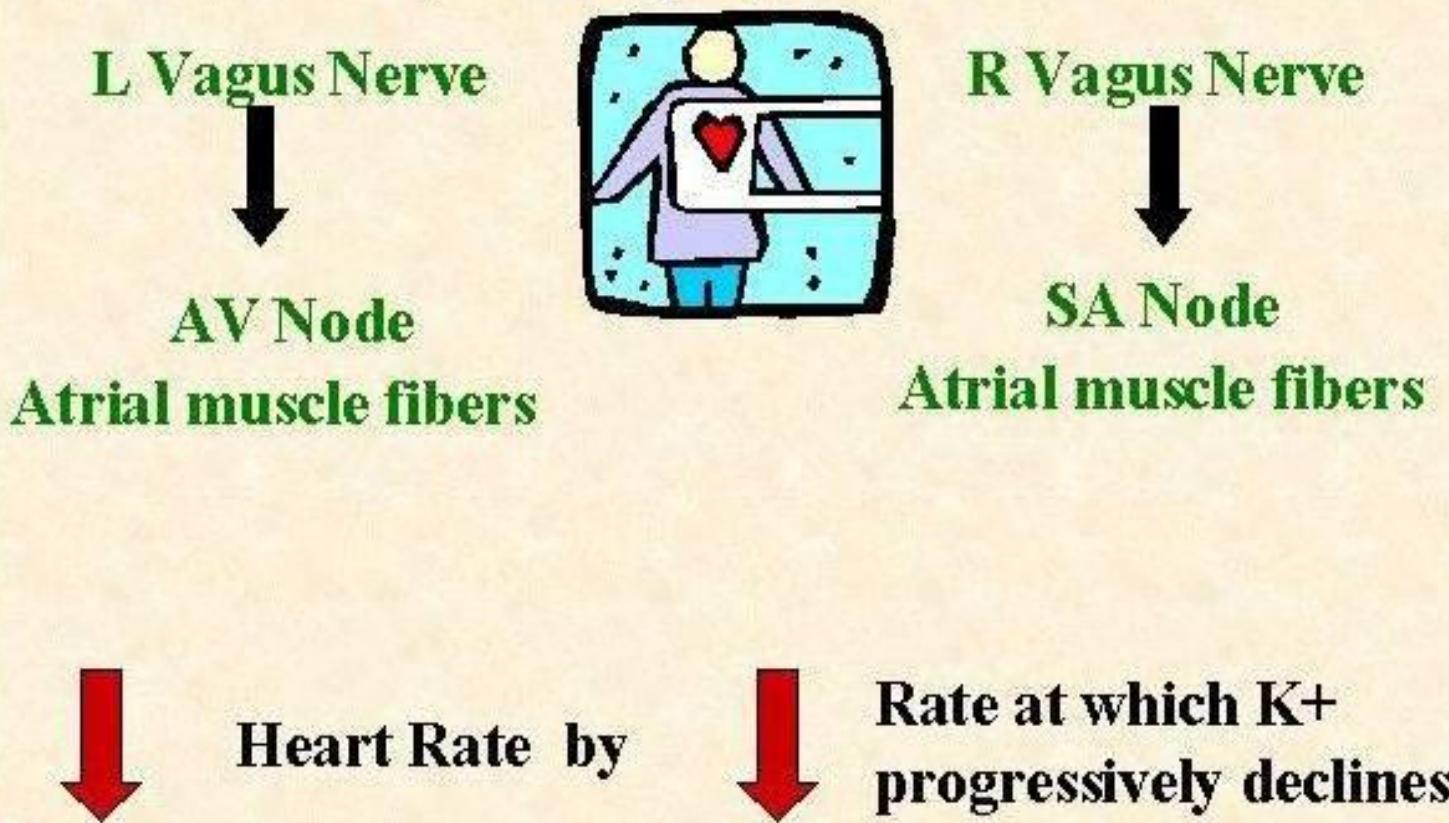


b. Afterload: it is the load against which the ventricles pump.

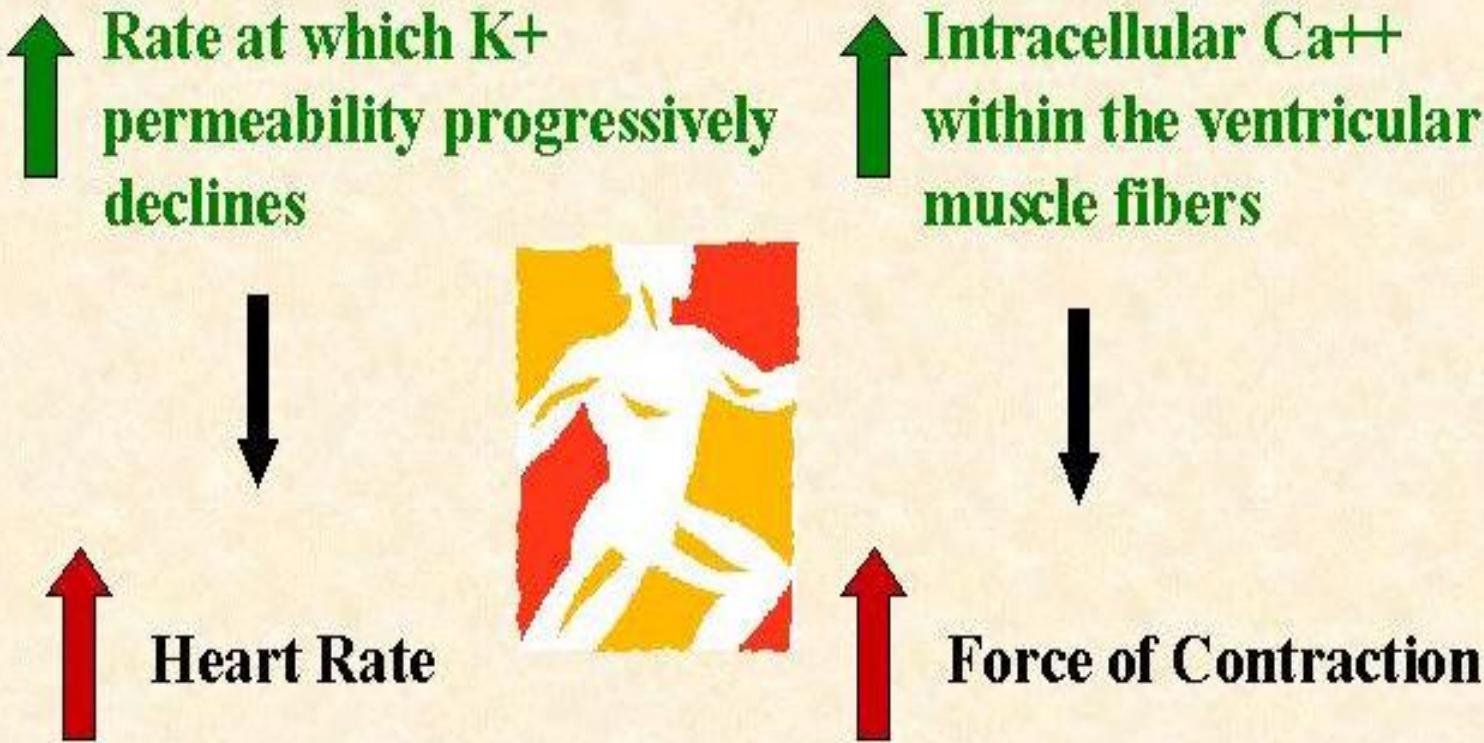
- For the left ventricle is **aortic pressure**
 - Increase in aortic pressure cause an increase in afterload on the left ventricle.
- For the right ventricle is **pulmonary artery** pressure.
 - Increase in pulmonary artery pressure cause an increase in afterload on the right ventricle.

2. EXTRINSIC

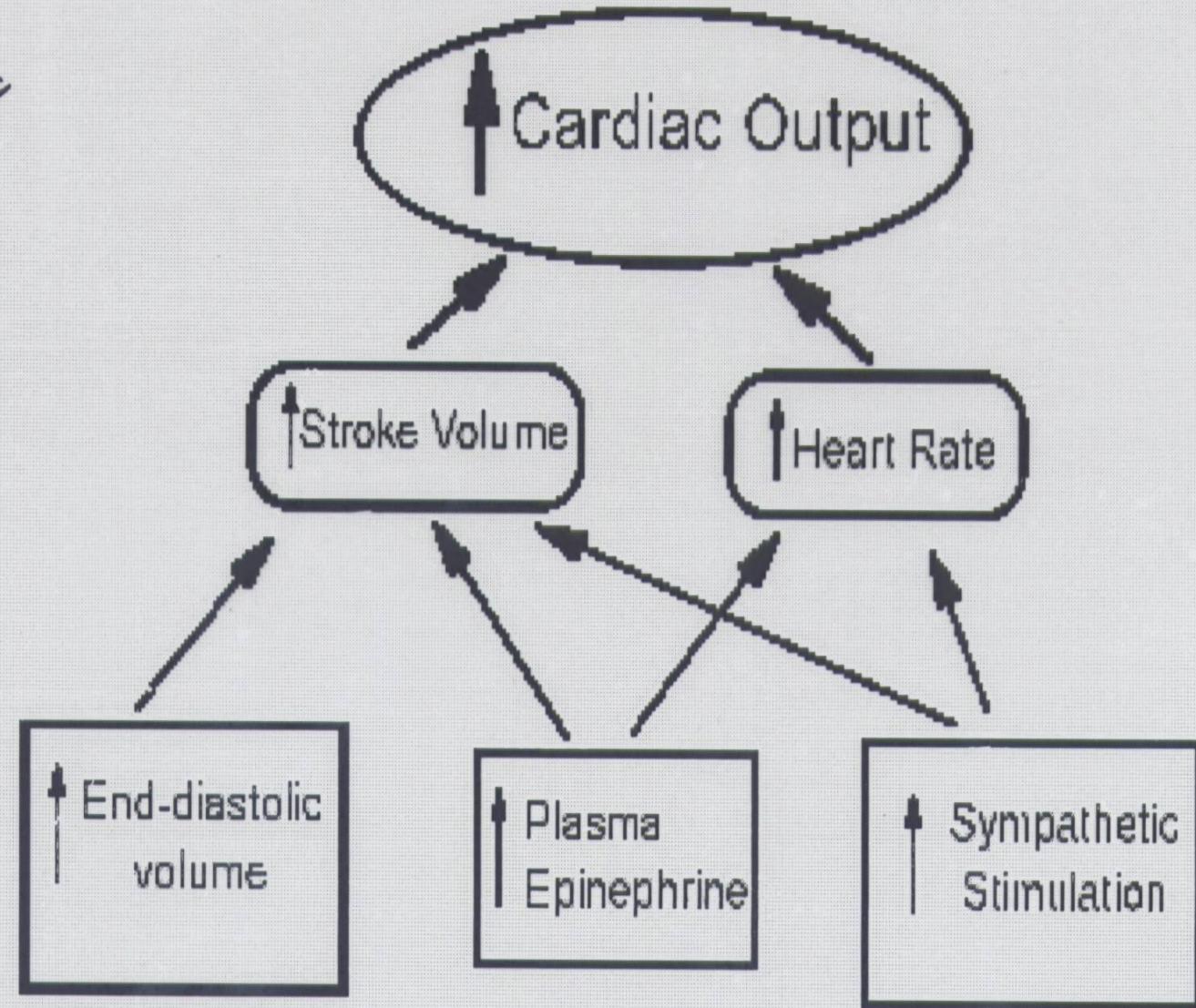
a) Parasympathetic



b) Sympathetic



Extrinsic



Vascular Physiology

Blood vessels

- Form a ***closed circuit*** of tubes that carries blood from the heart to the body cells and back again.
- The circulation is divided into the:
 - *Systemic circulation*
 - *Pulmonary circulation*

Functions of blood vessels:

- Distribution of blood
- Exchange of materials with tissues
- Return of blood to the heart

Functional categories of blood Vessels

- Types of Blood vessels
 1. Elastic (Windkessel) vessels
 2. Resistance vessels
 3. Exchange vessels
 4. Capacitance vessels

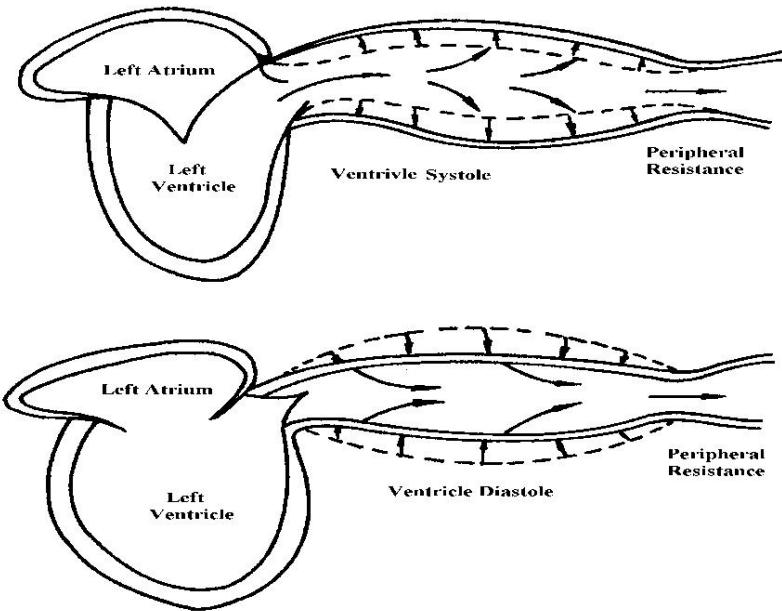
Elastic (Windkessel) vessels

- Thick-walled arteries near the heart; the aorta and its major branches.
- Large diameter, low-resistance.
- Contain lots of elastin.
- High ability of stretching and recoiling.
- Withstand and regulate large blood pressure fluctuations.

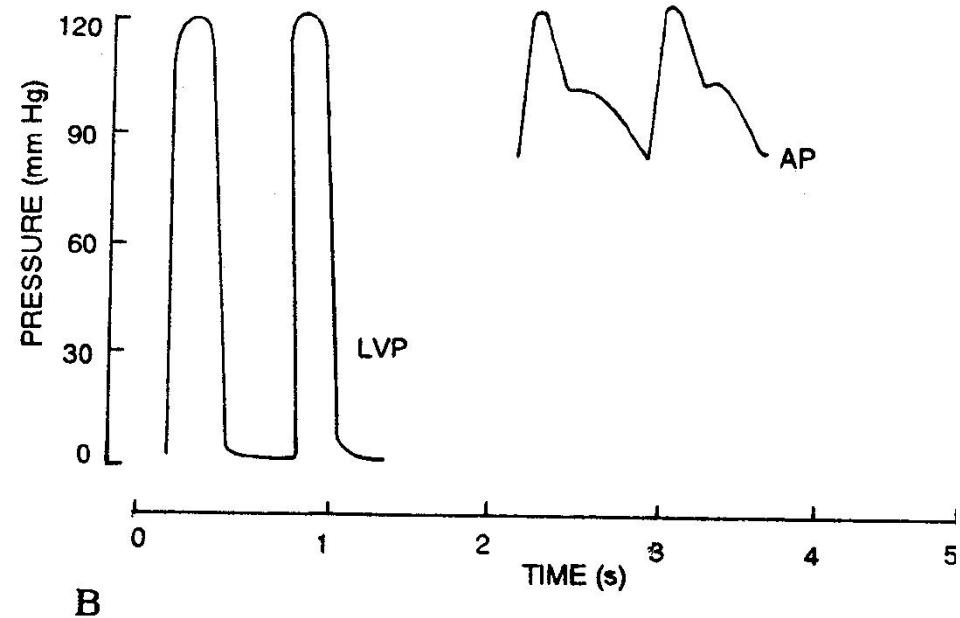
Elastic (Windkessel) vessels (continued)

- Transiently store blood during systole, and then shrink to produce onward blood flow during diastole.
- Convert the **sharp** pressure fluctuations in the left ventricle into much **smaller** pressure fluctuations in the arteries.
- Maintain continuous blood flow in the vessels.
- This function of large arteries is known as Windkessel effect.

Elastic (Windkessel) vessels (continued)



Function of the Windkessel vessel



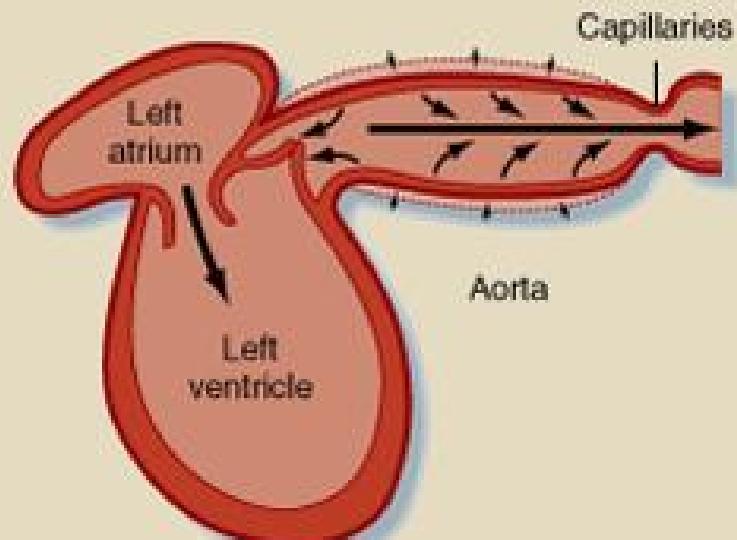
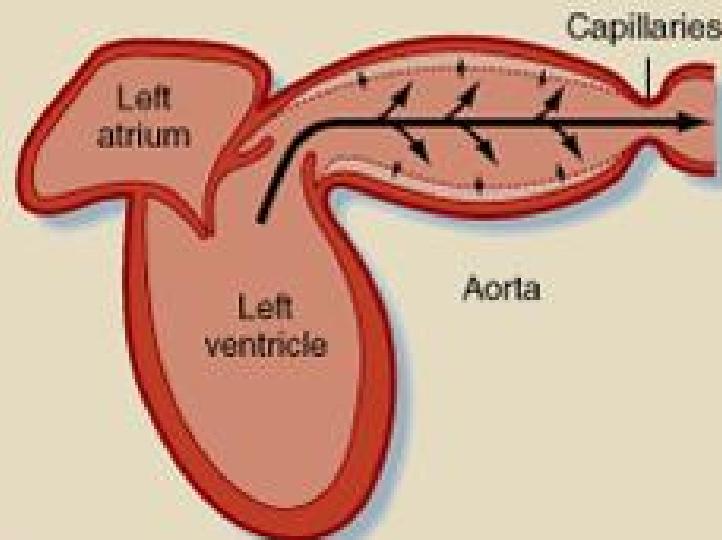
- The elastic nature of the large arteries also reduces the work of the heart.

Elastic (Windkessel) vessels (continued)

COMPLIANT

Systole Arterial blood flows through the capillaries throughout systole.

Diastole Arterial blood continues to flow through the capillaries throughout diastole.



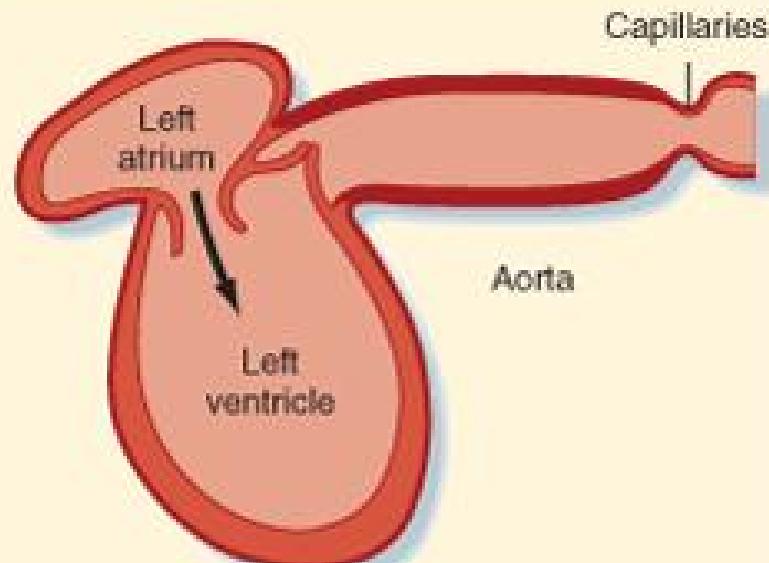
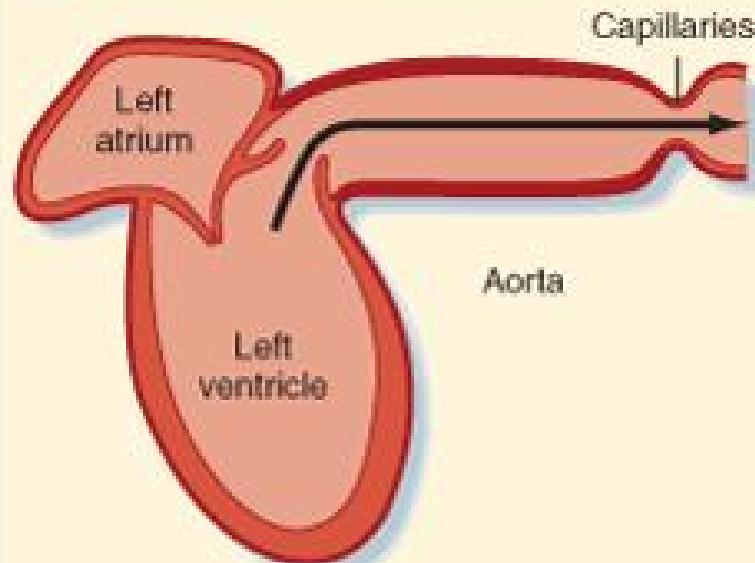
- A When the arteries are normally compliant, a substantial fraction of the stroke volume is stored in the arteries during ventricular systole. The arterial walls are stretched.

- B During ventricular diastole the previously stretched arteries recoil. The volume of blood that is displaced by the recoil furnishes continuous capillary flow throughout diastole.

RIGID ARTERIES

Systole A volume of blood equal to the entire stroke volume must flow through the capillaries during systole.

Diastole Flow through the capillaries ceases during diastole.

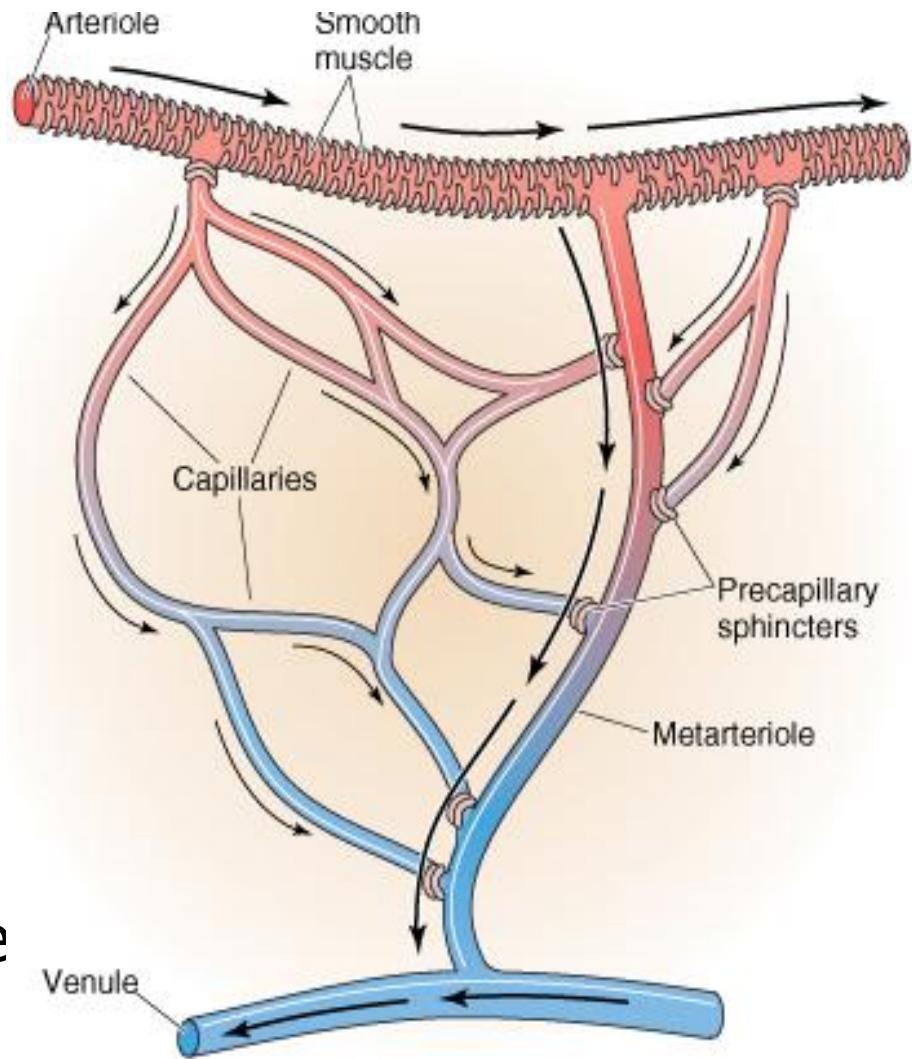


- C When the arteries are rigid, virtually none of the stroke volume can be stored in the arteries.

- D Rigid arteries cannot recoil appreciably during diastole.

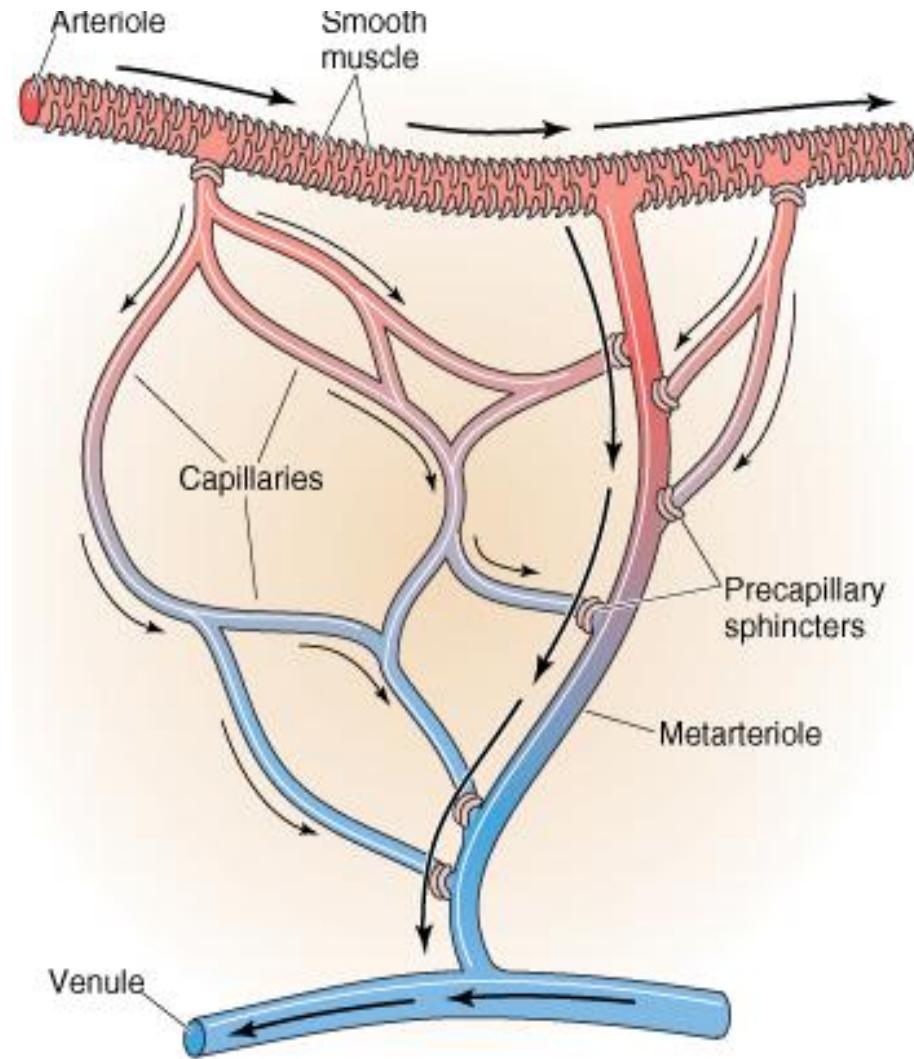
Resistance Vessels –Small arteries and arterioles

- The walls of the arterioles contain less elastic tissue but much more smooth muscle.
- The muscle is innervated by sympathetic nerve fibers.
- The arterioles are the major site of the resistance to blood flow-change TPR.



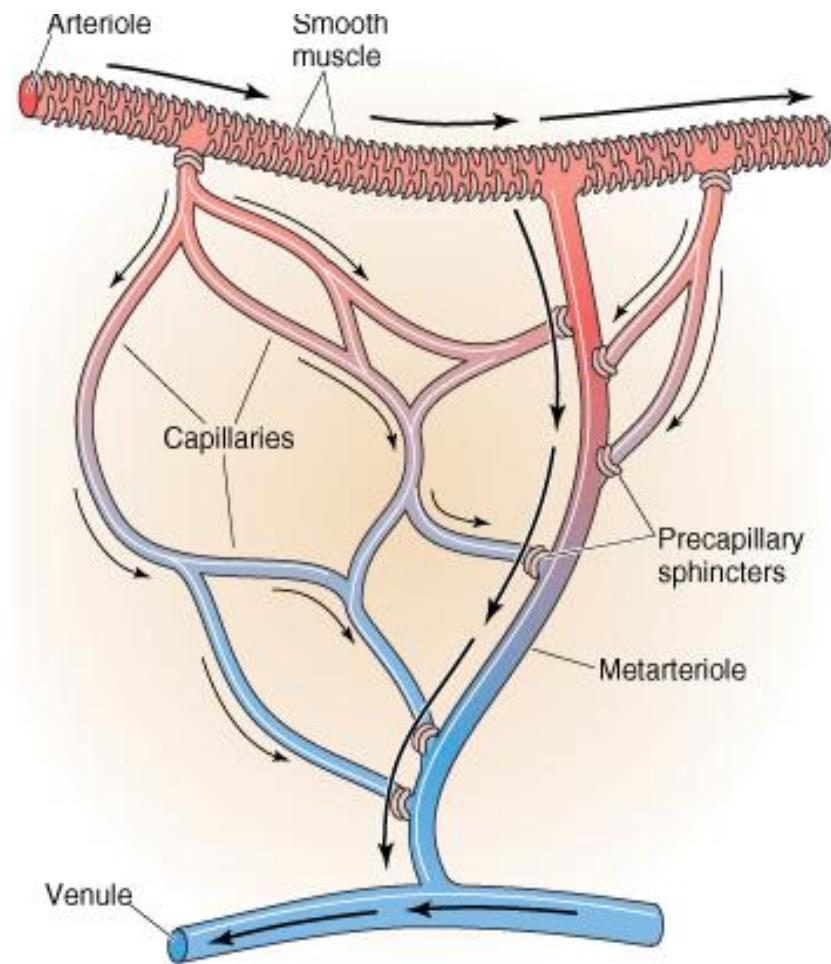
Resistance Vessels...

- Are the ***last small branches of the arterial system***- act as *control conduits* .
- Have strong muscular walls that can close the arterioles completely or can dilate the vessels several fold - have the capability of altering blood flow in each tissue.



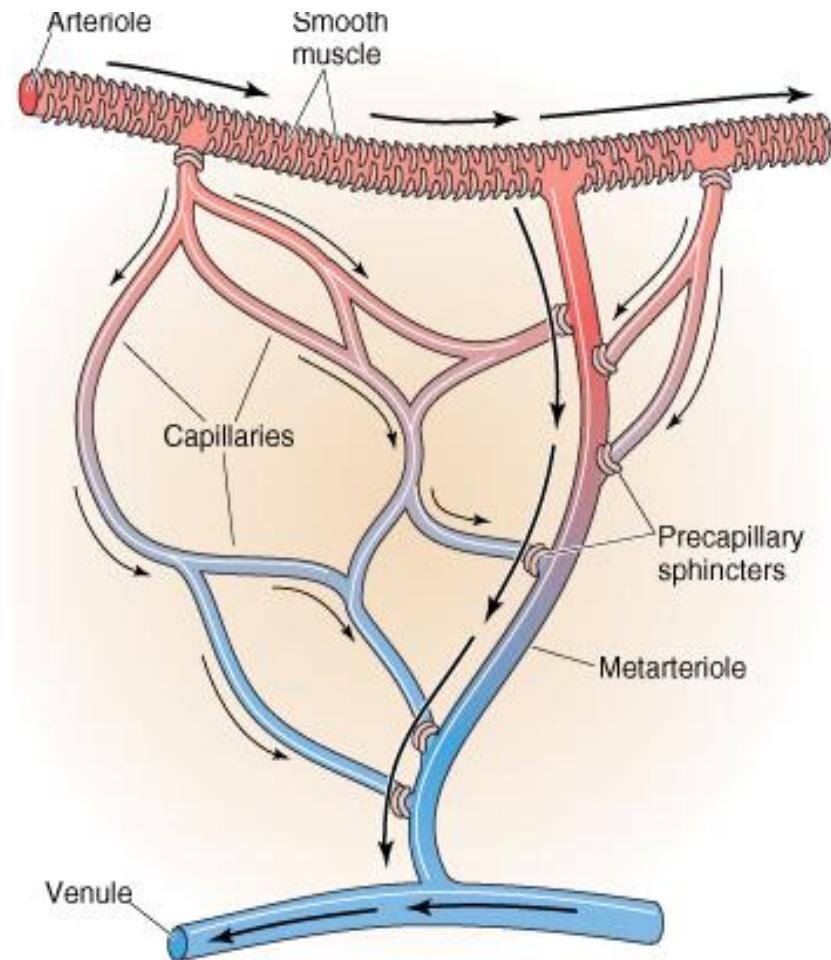
Resistance Vessels...

- Arteriolar smooth muscle possesses spontaneous contractile activity - called **intrinsic tone** (basal tone).
- It sets a baseline level of contraction that can be increased or decreased by external signals.



Resistance Vessels...

- The arterioles are the major site of the resistance to blood flow, and small changes in their caliber cause large changes in the total peripheral resistance.



Exchange Vessel – Capillary

- Their wall is composed of a unicellular layer of endothelial cells surrounded by a thin basement membrane.
- Capillary walls are very thin - permit a more rapid transport of materials between the blood and the tissues.
- Their function is to exchange fluid, nutrients... between the blood and the interstitial fluid.

4. Capacitance Vessels–Systemic veins

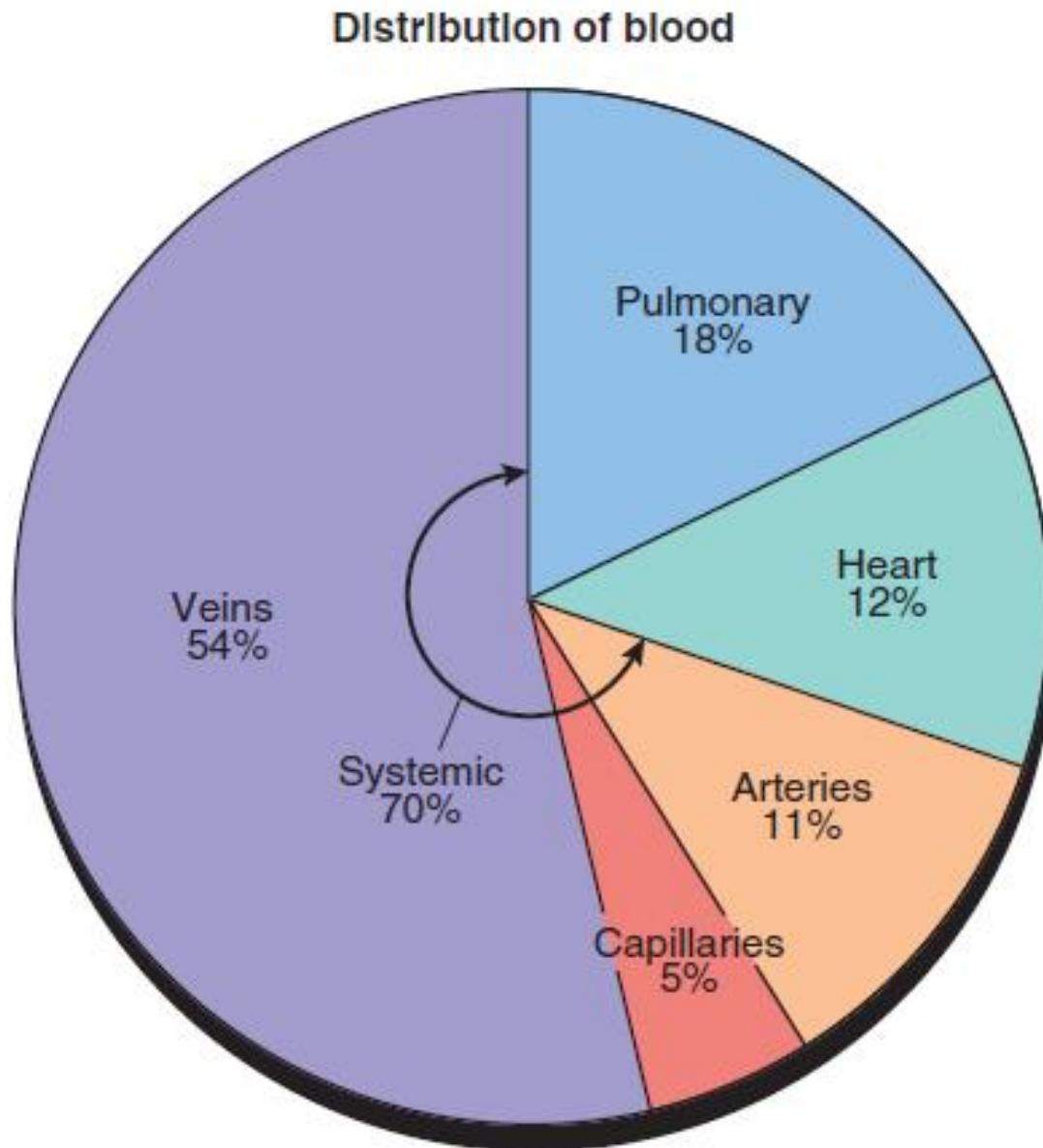
Have:

- Large diameter
- Thin walled
- Thin muscle coat
- Twice as much as the number of arteries
 - The large number and cross sectional area gives them an enormous capacity to hold blood.

Capacitance Vessels–Systemic veins (continued)

- Can accommodate large volume of blood (65% of blood volume)
- The great distensibility of veins makes their capacity adjustable too.
- In times of need, a considerable amount of blood can be squeezed from the veins to areas where it may be needed for.

Average distribution of blood in resting adult



Blood volume in high and low pressure compartment

High pressure (>30mmHg)

- Large arteries-8%
- Small arteries –5%
- Arterioles –2%

Low pressure (<30mmHg)

- Capillaries-5%
- Small veins & venules-25%
- Large veins ---34%
- Pulmonary vessels ---12%
- Heart -----9%

High pressure compartment

- Pressure reservoir
- Volume =15%

Low pressure compartment

- Volume reservoir
- Volume =85%

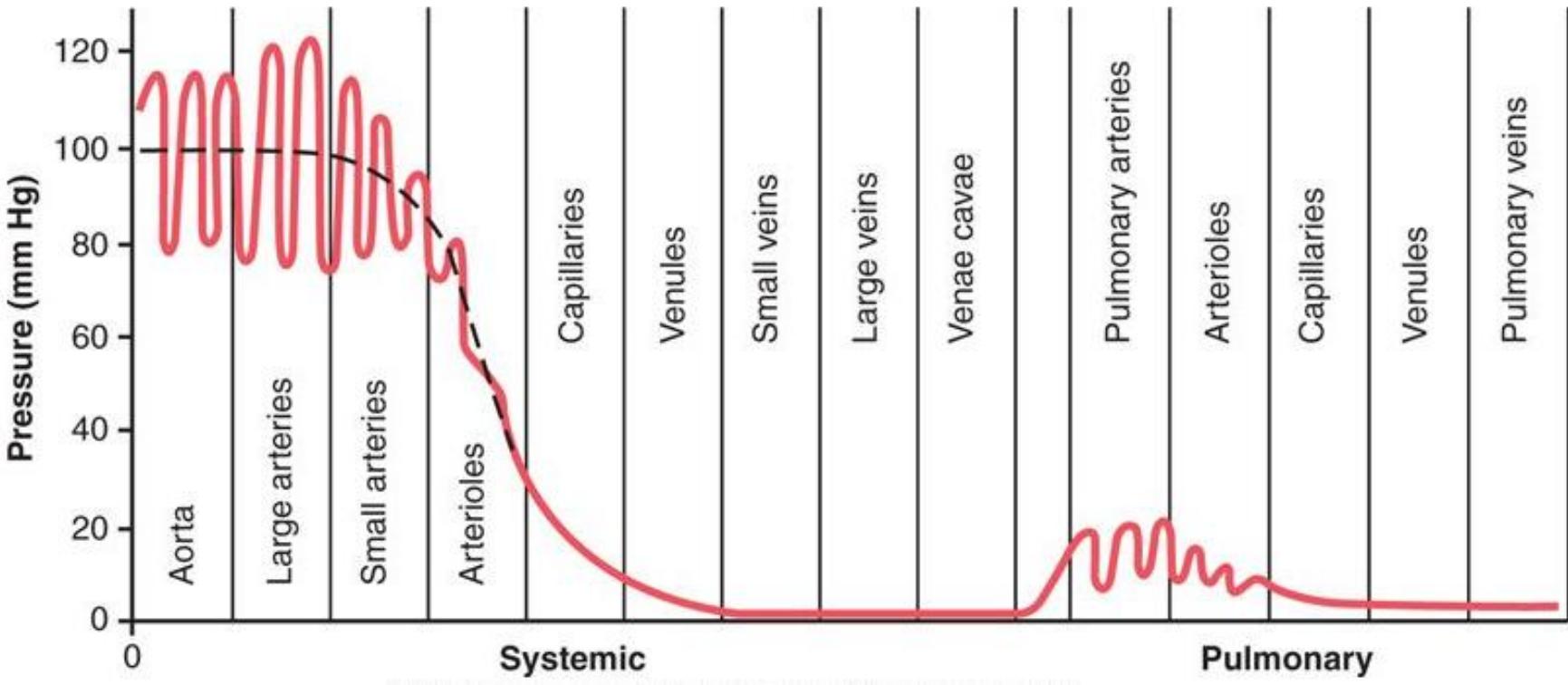


Fig: Normal blood pressures in the different portions of the circulatory system when a person is lying in the horizontal position.

Biophysics of blood flow *(Hemodynamics)*

- Relationship between blood flow, pressure and resistance.
- Blood flow is the amount of blood flowing through an organ, tissue, or blood vessel in a given time (such as L/min).

Hemodynamics *(continued)*

- *The physical principles of blood flow, (hemodynamics,), are based mainly on pressure and resistance.*

1. Pressure difference (ΔP)

- Blood flows from high pressure to low pressure.
- Q depends on $P_1 - P_2$ (ΔP) (pressure gradient)

2. Vascular resistance (R) acting along blood vessels

- It is the impediment to blood flow in a vessel.

Hemodynamics *(continued)*

Ohm's Law

$$\text{FLOW} = \frac{\Delta P}{R}$$

$$R = \frac{P_i - P_o}{Q}$$

$$P_1 > P_2$$



$$\Delta P = \text{FLOW} \times R$$

Hemodynamics *(continued)*

Poiseuille's law

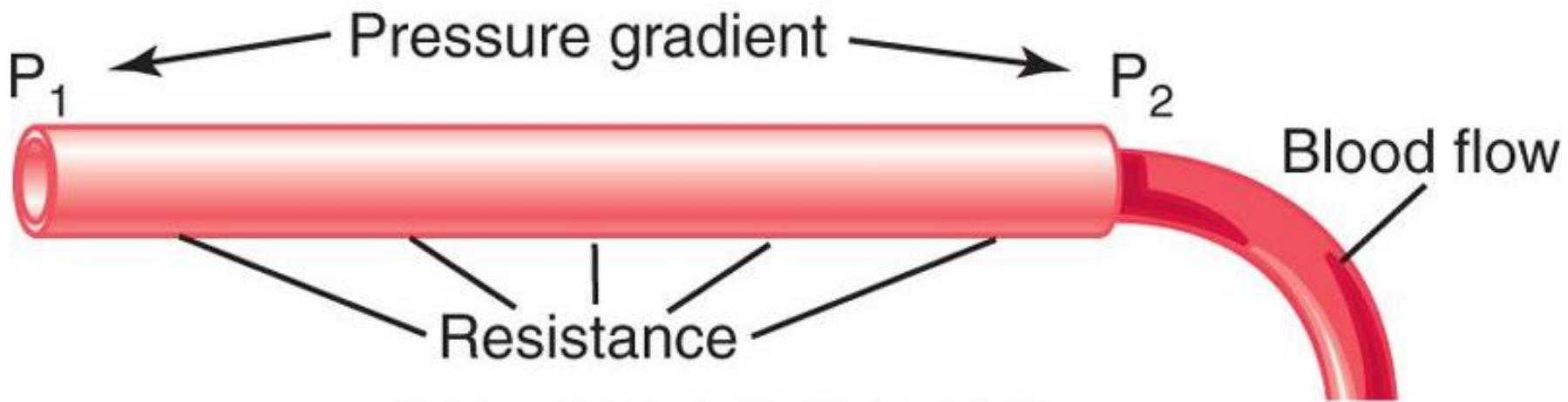
It describes the flow of fluids through cylindrical tubes in terms of flow, pressure, the dimensions of the tube, and the viscosity of liquid.

$$Q = \frac{\pi(P_i - P_o)r^4}{8\eta l}$$

$$Q = \frac{P_1 - P_2}{R} \quad R = \frac{8\eta l}{\pi r^4}$$

- Q = flow
- η = viscosity of the fluid
- r = radius of the tube
- l = length of the tube
- $P_i - P_o$ = pressure gradient from the inlet (i) of the tube to the outlet (o)

- It is the *difference* in pressure between the two ends of the vessel, *not the absolute pressure* in the vessel, that determines rate of flow.



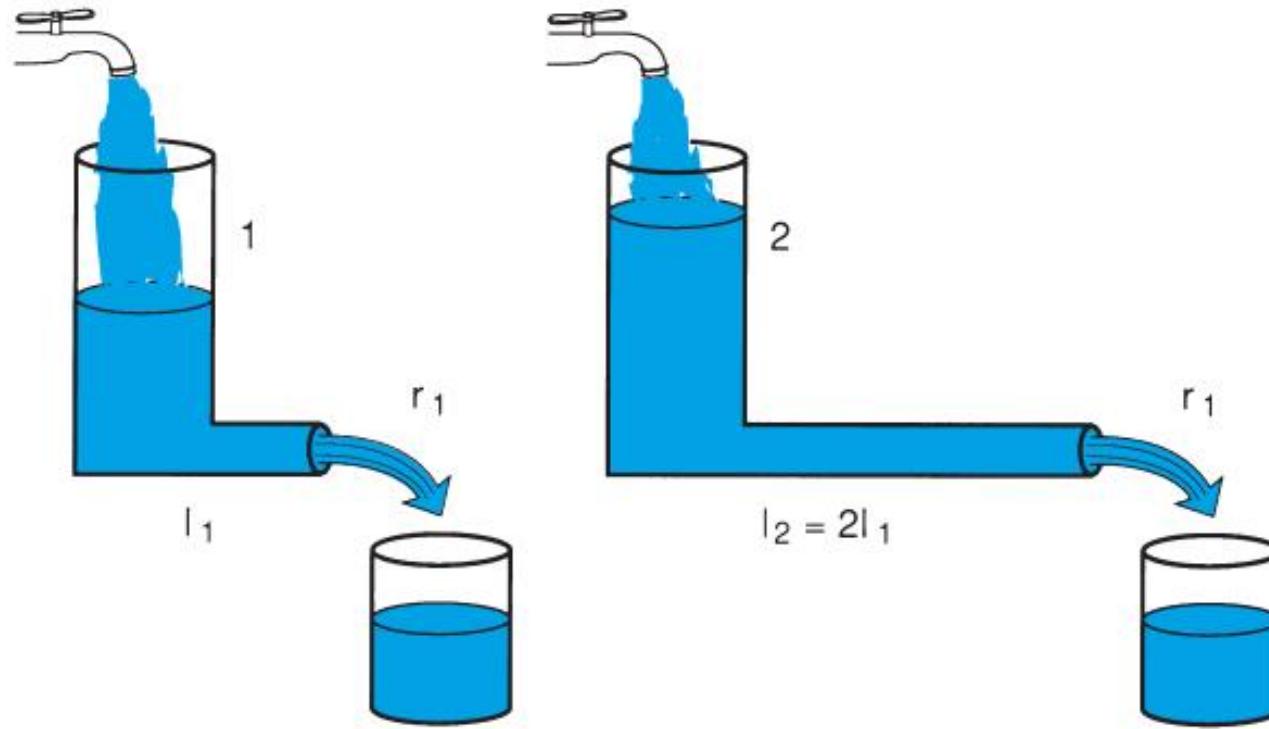
- Fig. Interrelationships of pressure, resistance, and blood flow.

Resistance to Flow

- The principal determinant of resistance to blood flow through any vessel is the caliber (radius) of the vessel.
- Normally, L and η have no change or almost no change.
 - Therefore, the diameter of a blood vessel plays by far the greatest role of all factors in determining the resistance (R) of blood flow.

- $R = 8 \eta l / \pi r^4$, π is constant
- Note that the resistance of a vessel is directly proportional to the viscosity of the blood and length of the vessel, but inversely proportional to the **fourth power** of the radius.
- Normally, L and η have no change or almost no change.
 - Therefore, the diameter of a blood vessel plays by far the greatest role of all factors in determining the resistance of blood flow.

- Factors affecting resistance to blood flow
 - Radius of the blood vessel
 - Length of the blood vessel
 - Viscosity of blood



- Fig. Fluid is added at the same rate of flow to two containers that differ only in the lengths (l) of their outflow tubes such that the length in example 2 is twice that in example 1. The radii (r) of tubes 1 and 2 are equal. Because the resistance to flow in example 2 is twice that in example 1, column height will be twice as great in example 2 at steady state.

Radius = 1 mm
Resistance = R
Blood flow = F

Radius = 1 mm
Resistance = R
Blood flow = F

Radius = 2 mm
Resistance = $1/16 R$
Blood flow = $16 F$

Radius = $1/2$ mm
Resistance = $16 R$
Blood flow = $1/16 F$

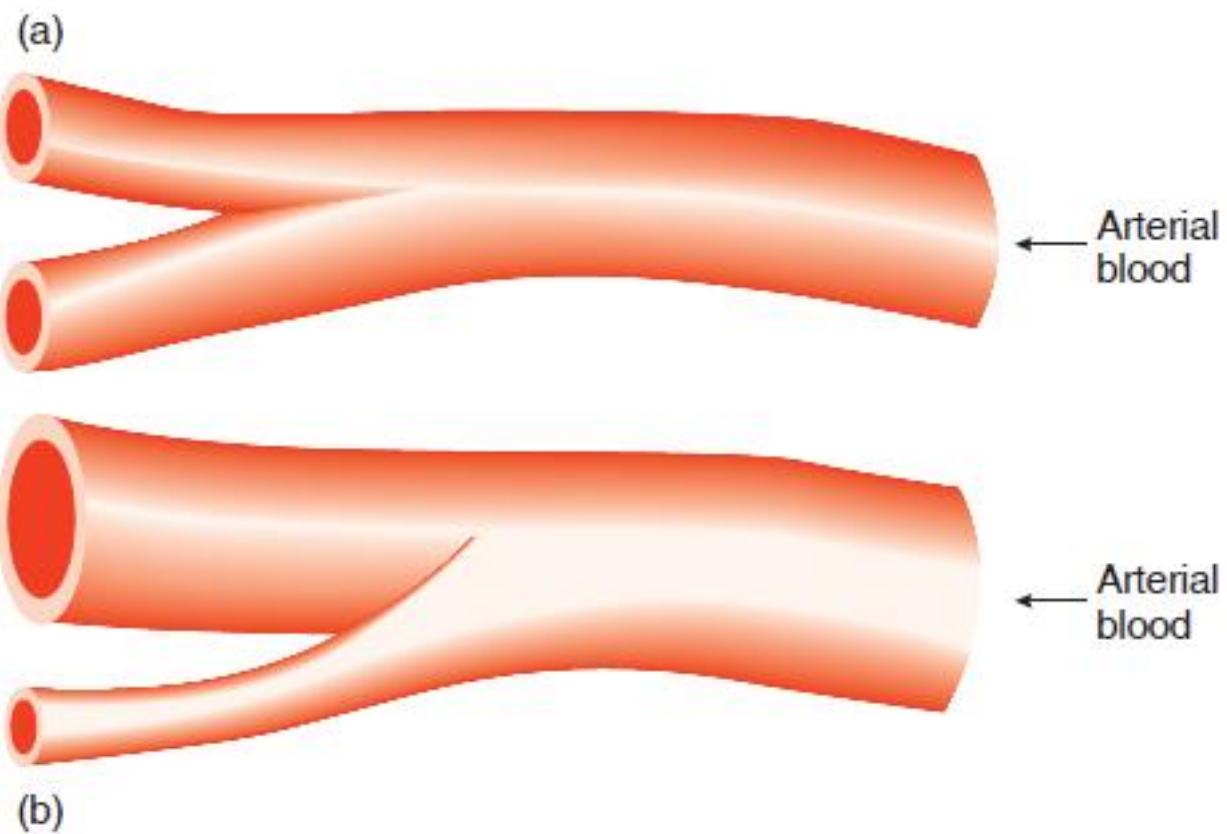


Fig: The relationships between blood flow, vessel radius, and resistance.

- The sum of all the vascular resistances within the systemic circulation is called the **total peripheral resistance**.

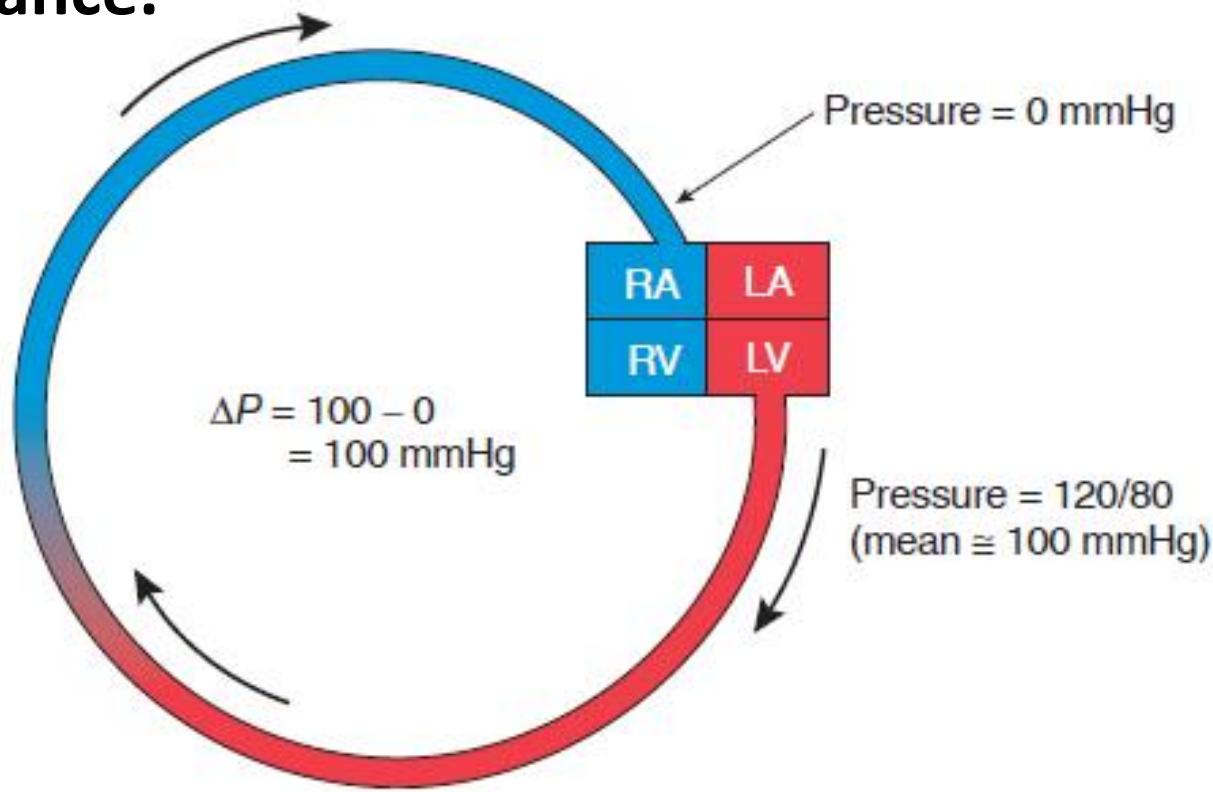


Fig: Blood flow is produced by a pressure difference.

Total peripheral resistance

Systemic circulation:

$$\text{TPR} = \frac{\text{Aortic Pressure} - \text{RAP}}{\text{Flow}}$$

$$\text{TPR} = \frac{100 - 0 \text{ mmHg}}{83.3 \text{ ml/sec (5 L/min)}}$$

$$\text{TPR} = 1.2 \text{ PRU's}$$

Pulmonary circulation:

$$\text{Pul. R.} = \frac{\text{Pul. Art. P.} - \text{LAP}}{\text{Flow}}$$

$$\text{Pul. R.} = \frac{15 - 5 \text{ mmHg}}{83.3 \text{ ml/sec}}$$

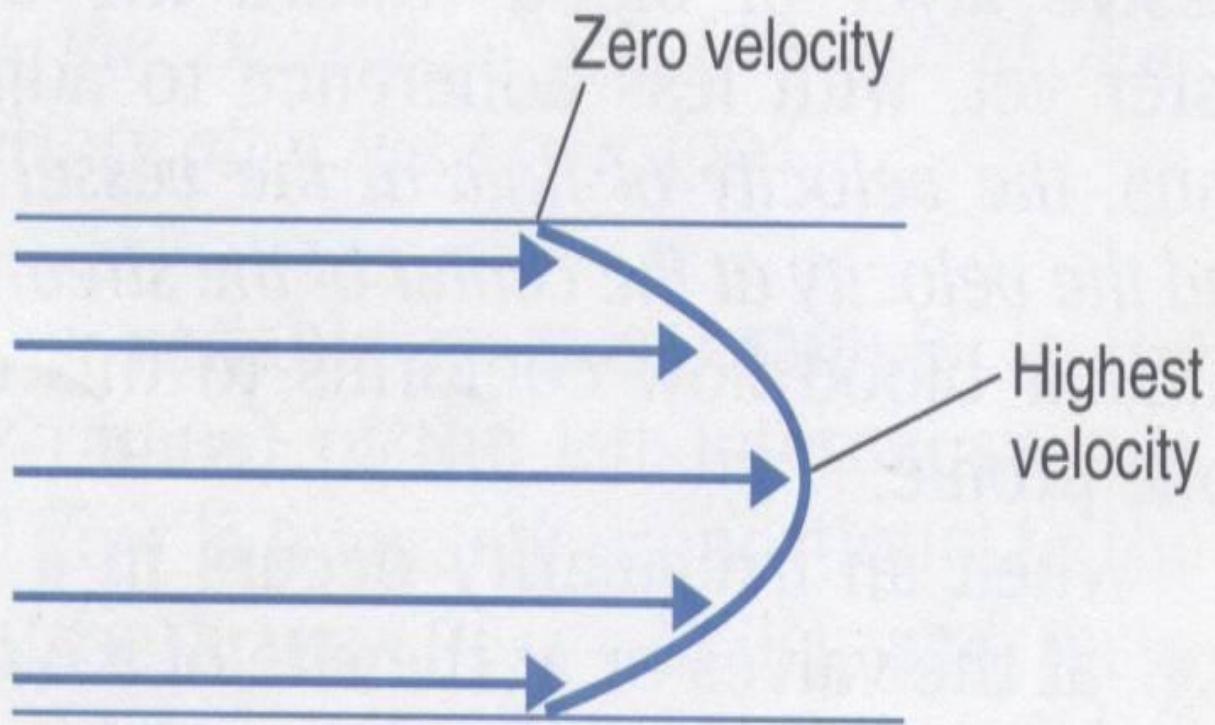
$$\text{Pul. R.} = 0.12 \text{ PRU's}$$

Types of blood flow

1. Laminar flow

- All elements of the fluid move in a stream line, that are parallel to the axis of the tube.
- Normal and noiseless.
- No fluid move in radial or circumferential direction.
- Layer of fluid in contact with the wall is motionless (thin layer, adherent to wall, hence motionless)
- Fluid that move along the axis of the tube has maximum Velocity

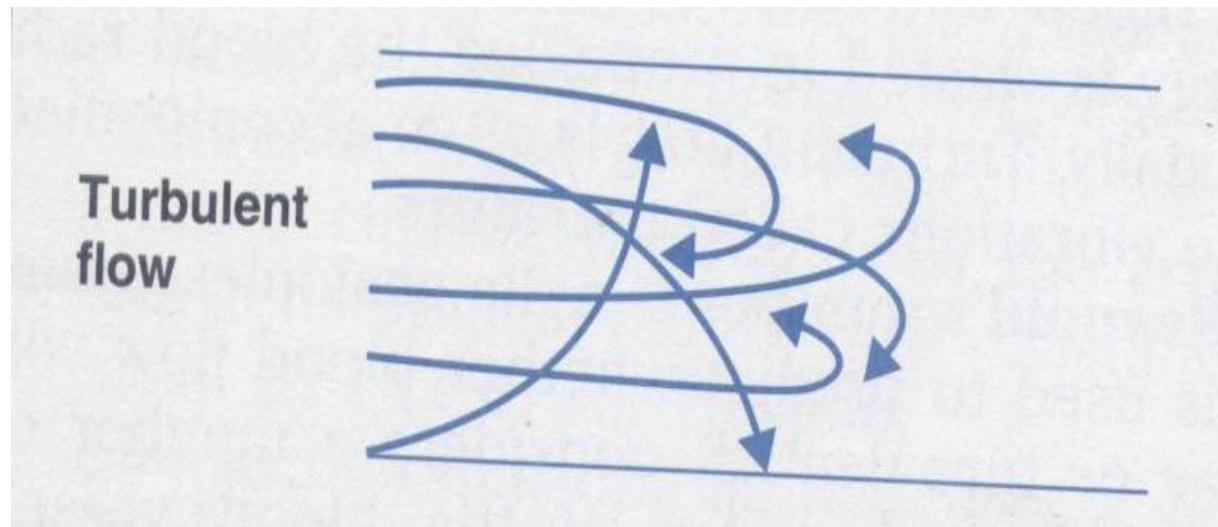
Laminar flow



2. Turbulent blood flow

- Various elements of fluid move irregularly in axial, radial and circumferential direction.
- More pressure required to drive the blood than in laminar flow.
- Energy wasted in propelling blood radially and axially
- Often accompanied by noise.
- Occur at valves and aorta (normal), at site of blood clot(pathological)

- The flow may become *turbulent when*,
 - The rate of blood flow becomes too great
 - It passes by an obstruction in a vessel
 - It makes a sharp turn
 - It passes over a rough surface



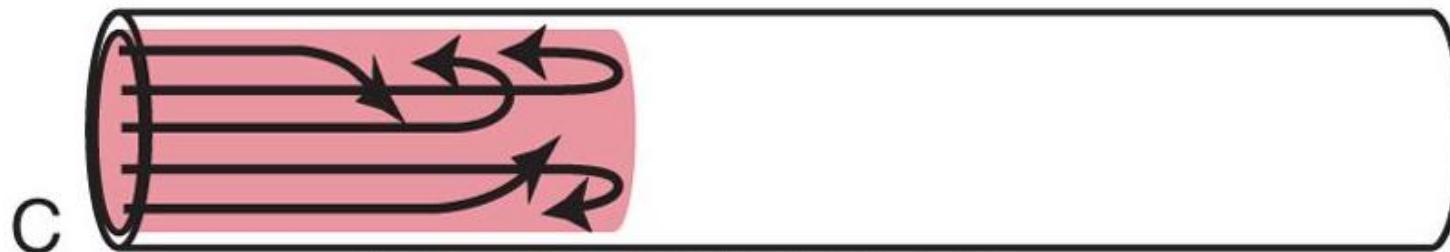
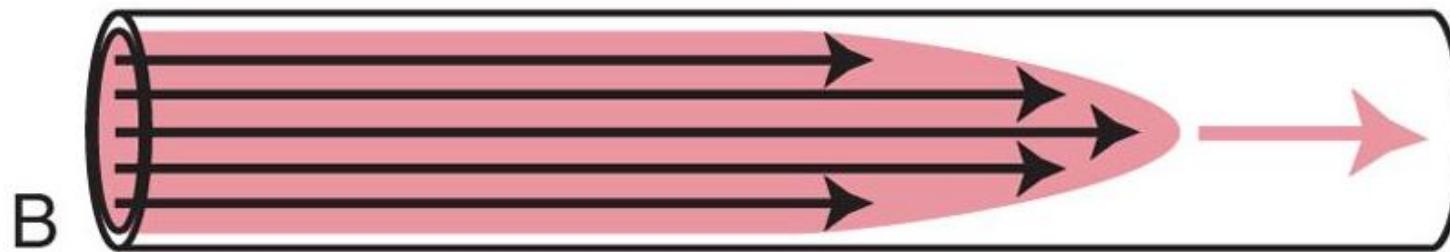


Fig: A) before flow begins; B) the same fluids 1 second after flow begins; C) turbulent flow, with elements of the fluid moving in a disorderly pattern.

Vascular distensibility

- All blood vessels are *distensible*.
- The most distensible by far of all the vessels are the veins.
- The compliance of a systemic vein is about 24 times that of artery.

- The term used to denote how easily a structure can be stretched is **compliance**.
- Is expressed as the fractional increase in volume for each mmHg rise in pressure.

$$\text{Compliance} = \Delta\text{volume} / \Delta\text{pressure}$$

- The *higher* the compliance of a structure, the *more easily* it can be stretched.

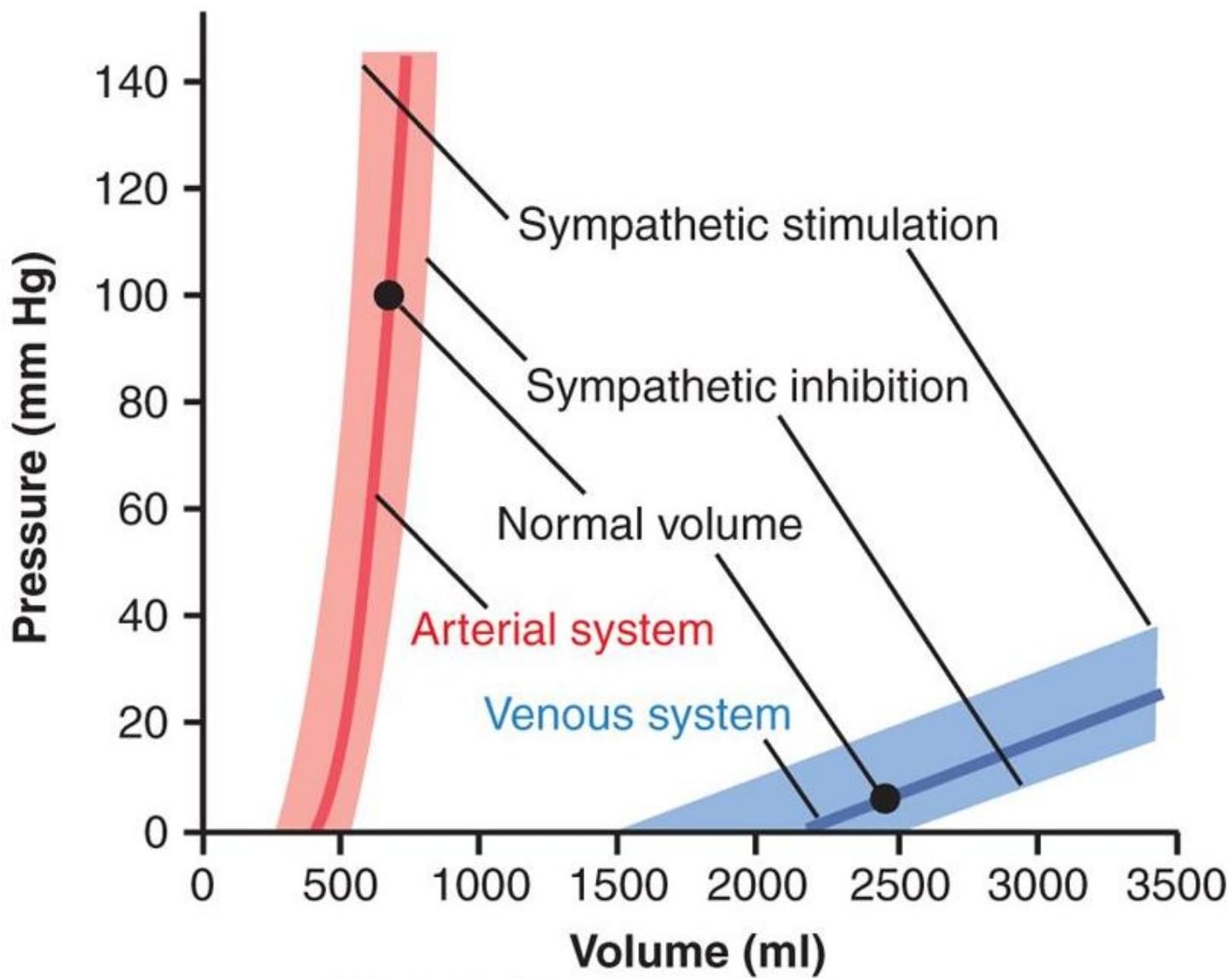


Fig: "Volume-pressure curves" of the systemic arterial and venous systems, showing the effects of stimulation or inhibition of the sympathetic nerves to the circulatory system.

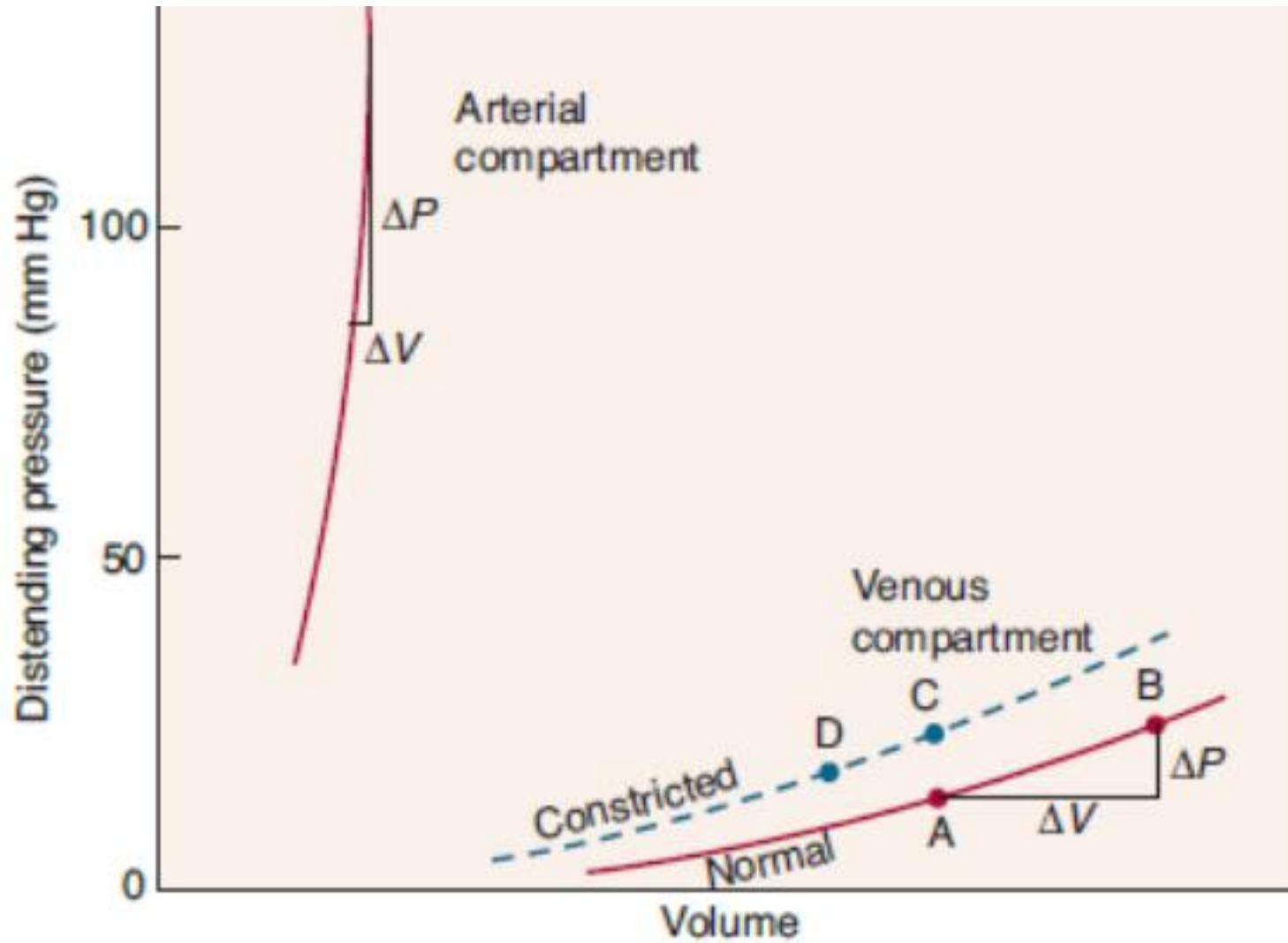
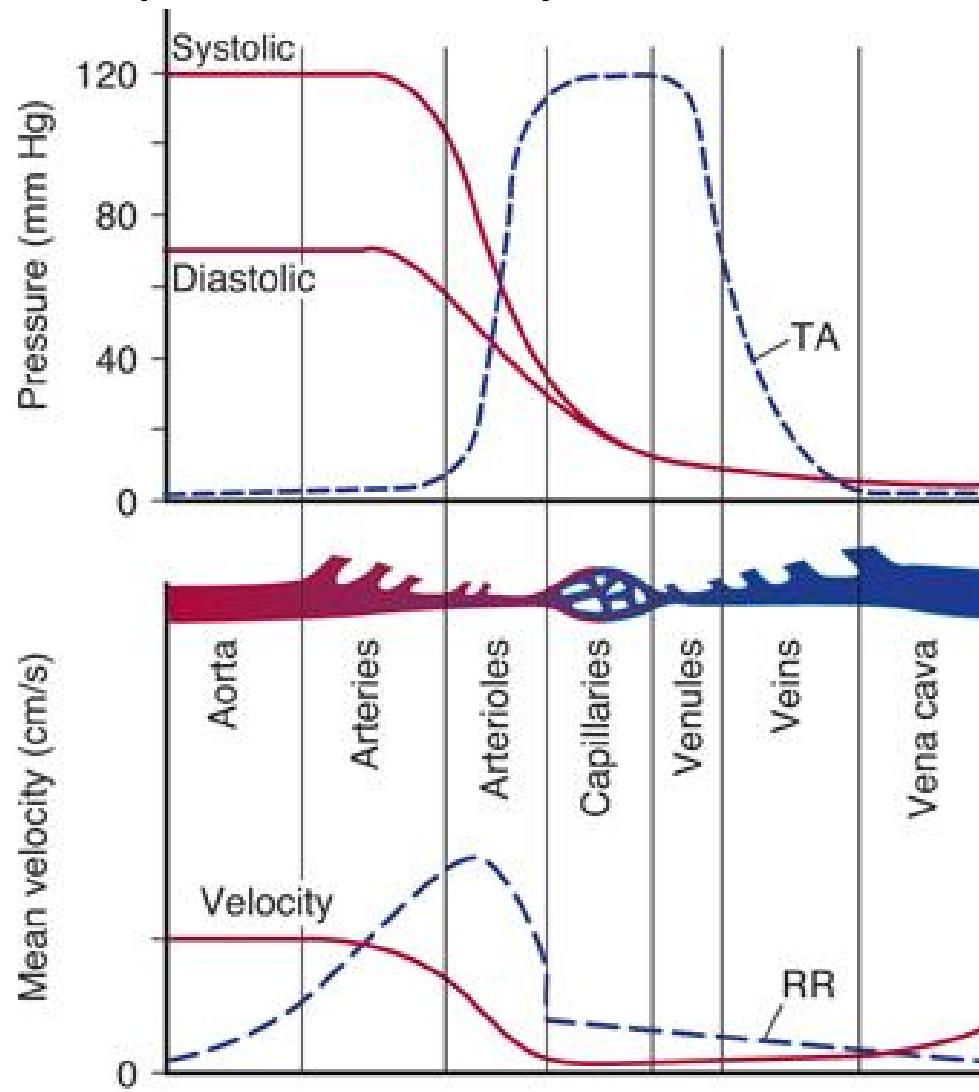


Fig: Volume–pressure curves of arterial and venous compartments.

Physical Characteristics of the Systemic Circulation

- Resistance (R), pressure (P) and flow (Q) patterns change as we go from arteries to arterioles.
- R increases as we go down from the aorta to the arterioles and hence arterioles are referred to as resistance vessels.
- Pressure decreases from aorta to capillaries and blood flow becomes steady as it passes the arterioles.

- The pressure and velocities of the blood in the various parts of the systemic circulation



- The capillaries offer much less resistance than the arterioles.
- Although the radius of each capillary is less than that of an arteriole, the capillaries as a group have a low resistance because there are so many of them in parallel.
- Each arteriole supplies many capillaries.

- One arteriole branches to 10 capillaries leading to the formation of 10 billion capillaries in the peripheral tissues.
- Capillaries, in spite of their thinness and weakness, are capable of withstanding ABP without damage because they have narrow lumen and tension developed is small.

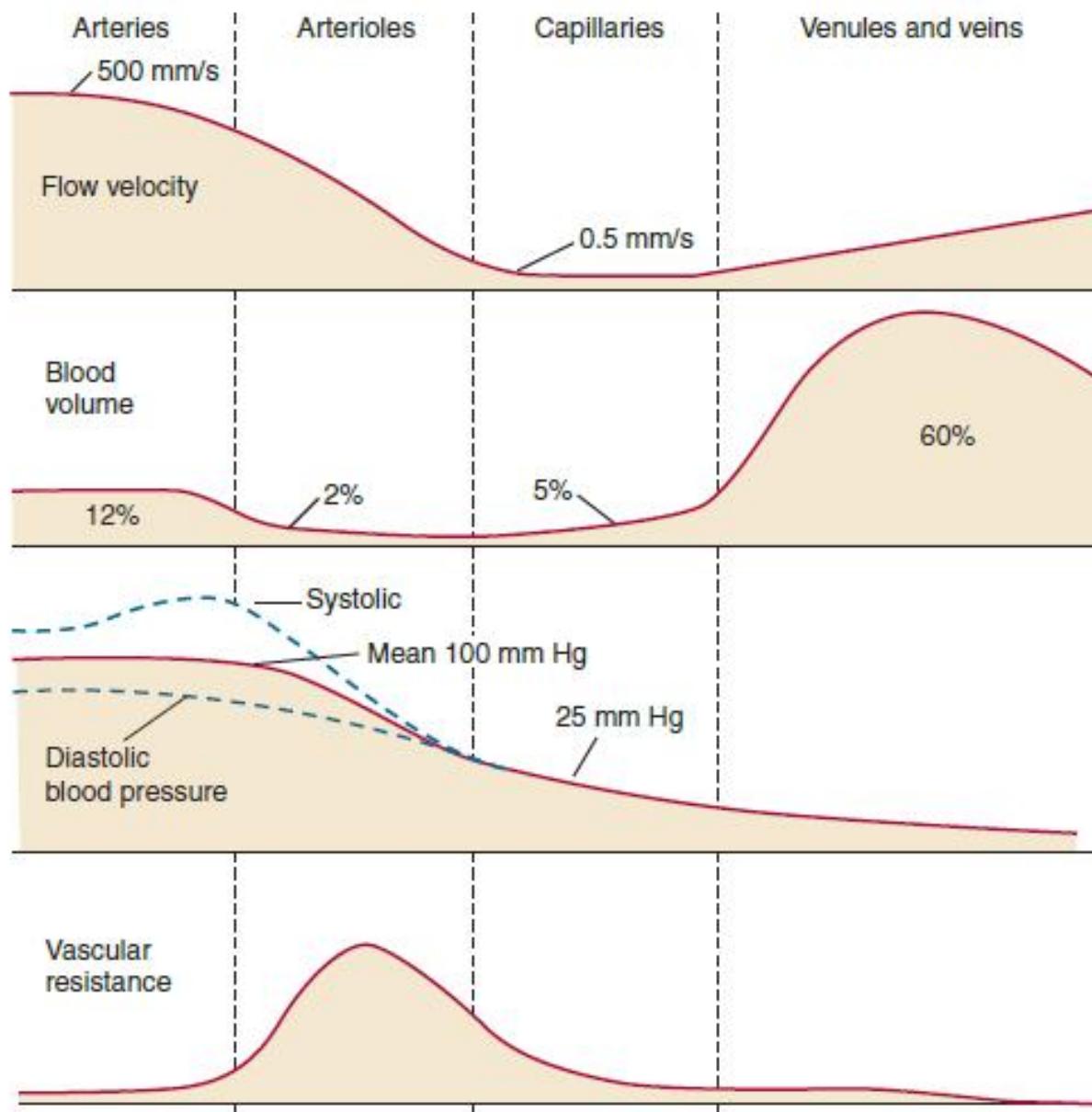


Fig: Flow velocities, blood volumes, blood pressures, and vascular resistances in the peripheral vasculature from aorta to right atrium.

The pressure is *lowest* as blood drains into **right atrium** at end of **diastole**.

R. ATRIUM
0 mmHg

GREAT VEINS
near heart
about 4 mmHg

MUSCULAR
VEINS
8 mmHg

Most of the energy imparted to blood by the heart's contraction has been spent by the time the blood reaches the venous side of circulation.

CAPILLARIES
15 mmHg ← 30 mmHg

The pressure is *highest* in the middle of **systole** as blood is ejected from **left ventricle**.

ARTERIAL SYSTEM
AORTA

Systolic Blood Pressure = 120 mmHg

Pressure is always lower in **diastole** e.g. 80 mmHg

It falls off gradually

Larger MUSCULAR ARTERIES 110 mmHg

ARTERIOLES
40 mmHg

Pulse pressure = SBP - DBP

Mean arterial pressure
 \approx DBP + $\frac{1}{3}$ (SBP - DBP)

Velocity of blood flow

- The velocity of blood flow in each segment of the circulation is inversely proportional to its cross-sectional area.

$$V = Q/\text{Cross-sectional area}$$

- The highest velocity of blood flow is at the aorta (40cm/sec at aorta Vs <2mm/sec at capillaries)
- Capillaries - largest cross-sectional area

- Velocity (v) of blood flow is directly proportional to the pressure difference (ΔP) and diameter of the vessels, but inversely related to the viscosity of blood and length of the blood vessel.

$$V = Q/\text{Cross-sectional area} \rightarrow (A = \pi r^2)$$

$$= (\Delta P \pi r^4 / 8 \eta L) / \pi r^2$$

$$V = \Delta P r^2 / 8 \eta L$$

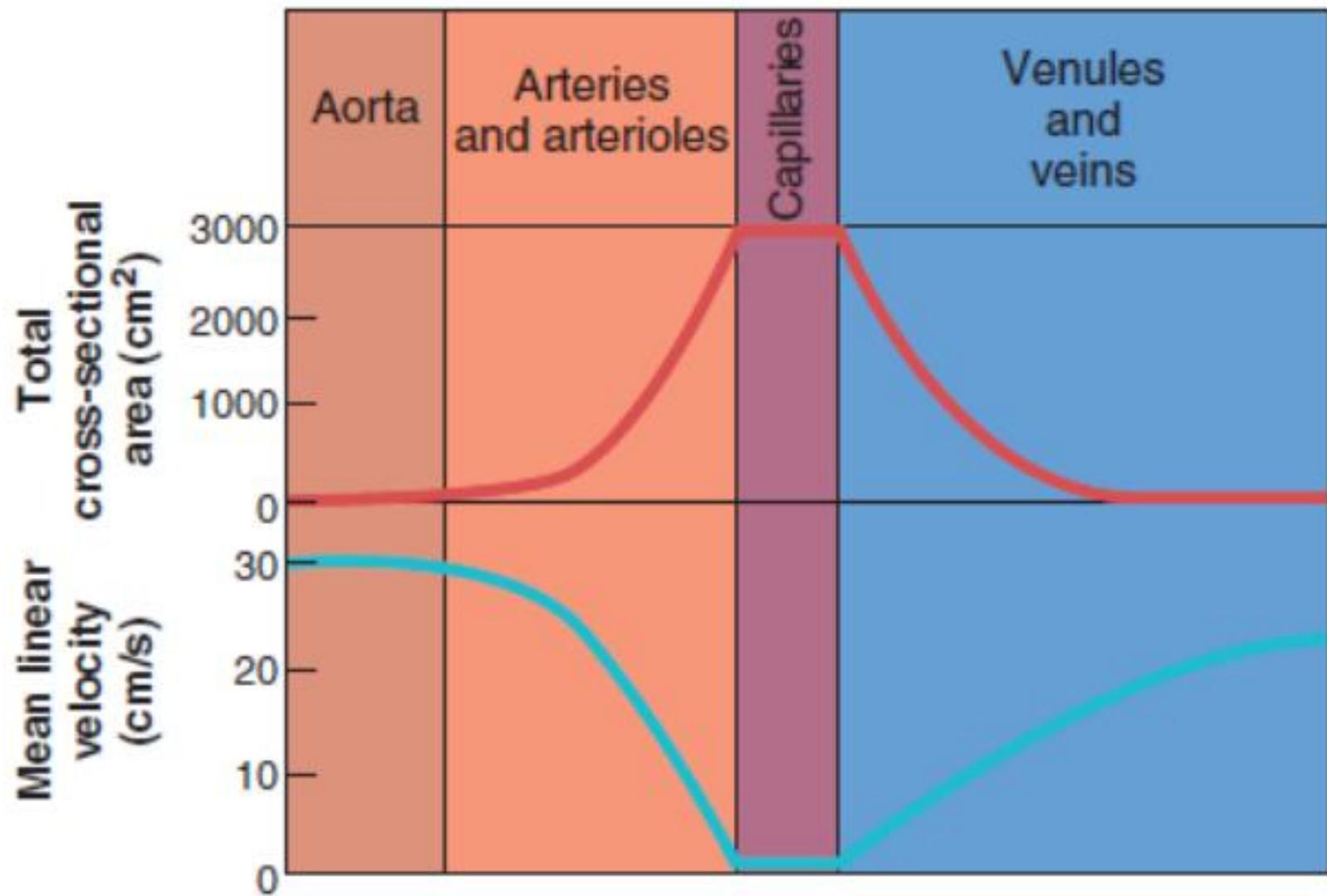
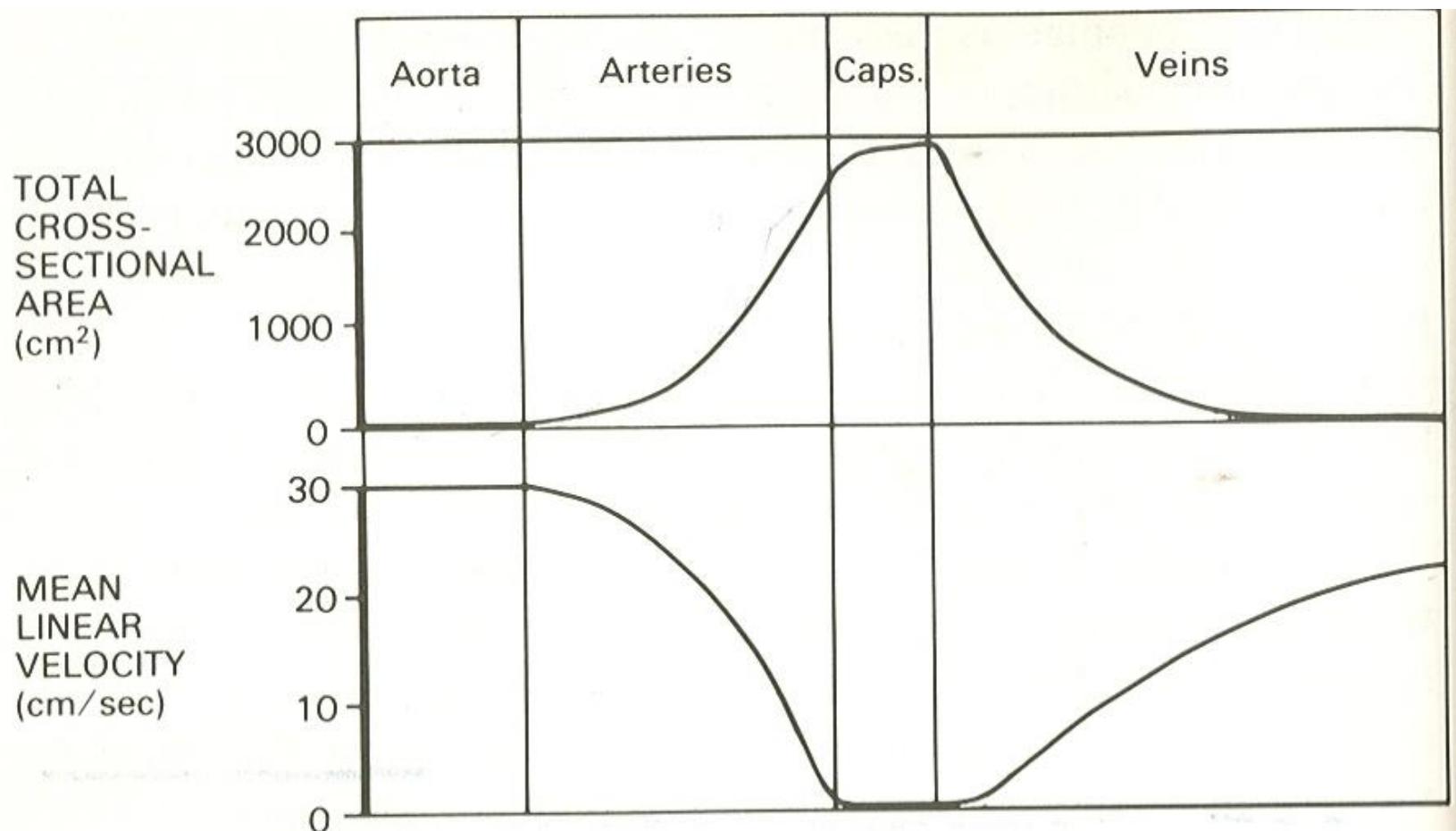


Fig: Relationship between total cross-sectional area and flow velocity in the systemic circulation.



$$\bar{V} = \frac{\text{Flow}}{\text{X-Sect. Area}}$$

Figure Relation of cross-sectional area and mean velocity of flow in the systemic circulation.

Arterial region of the systemic circulation

- Aorta branches → peripheral vascular bed → 1000 fold increase in cross-sectional area at capillaries.
- Large amount of elastin → ↑ expansion and recoil → pressure storage.

The large conducting arteries near the heart are **elastic arteries**.

SYSTOLE

When Ventricles are *contracting*

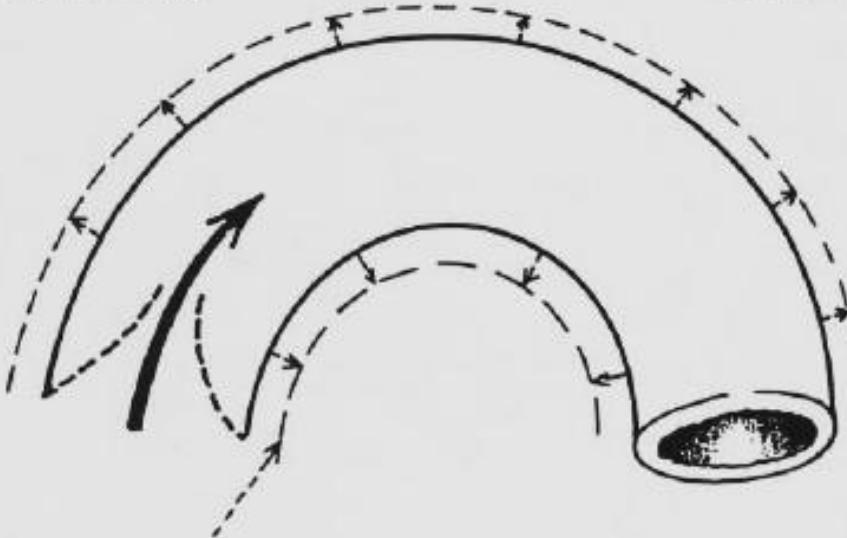
↓
pressure within
heart rises
↓

aortic valves
are forced open
↓

blood is forcibly
ejected into
aorta
↓

walls of aorta and its major
branches are stretched to
accommodate much of the blood
expelled
↓

aorta acts as a '**reservoir**'
its walls 'storing' energy
from contraction of heart



DIASTOLE

When Ventricles are *relaxing*

↓
pressure within
heart falls
↓

aortic valves
are pushed shut
↓

walls of aorta recoil to
initial position and propel
blood onwards while
heart is in diastole
↓

aorta acts as a '**subsidiary pump**'
its walls 'expending' the energy
previously 'stored' from the heart's
contraction and forcing the blood
on when the heart itself is in diastole.
This helps to 'smooth' the
pulsatile flow of blood

- A velocity of **40cm/sec at aorta** → **<2mm/sec at capillaries.**
i.e. $V=Q/A$.
- Beyond capillaries the situation is reversed and blood flow accelerates as total cross sectional area of veins decreases

Blood pressure

- Blood pressure means the force exerted by the blood against the vessel wall.
 - (or the force exerted by the blood against any unit area of the vessel wall)
- Blood pressure is a stored energy (potential energy)