



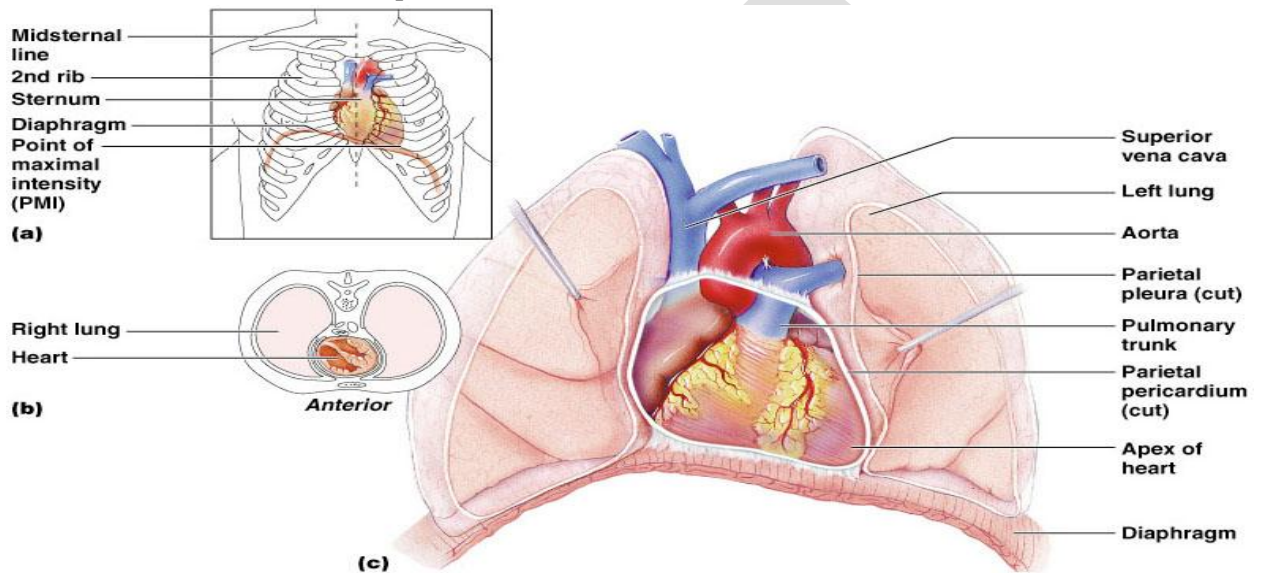
**Shree H. N. Shukla Institute of Pharmaceutical
Education and Research, Rajkot**

**B. Pharm
Semester-I**

**Subject Name: Human Anatomy & Physiology-I
Subject Code: BP101TP**

Chapter-5 CARDIOVASCULAR SYSTEM

- Approximately the size of your fist
- Location
- Superior surface of diaphragm
- Left of the midline
- Anterior to the vertebral column, posterior to the sternum



Coverings of the Heart: Anatomy

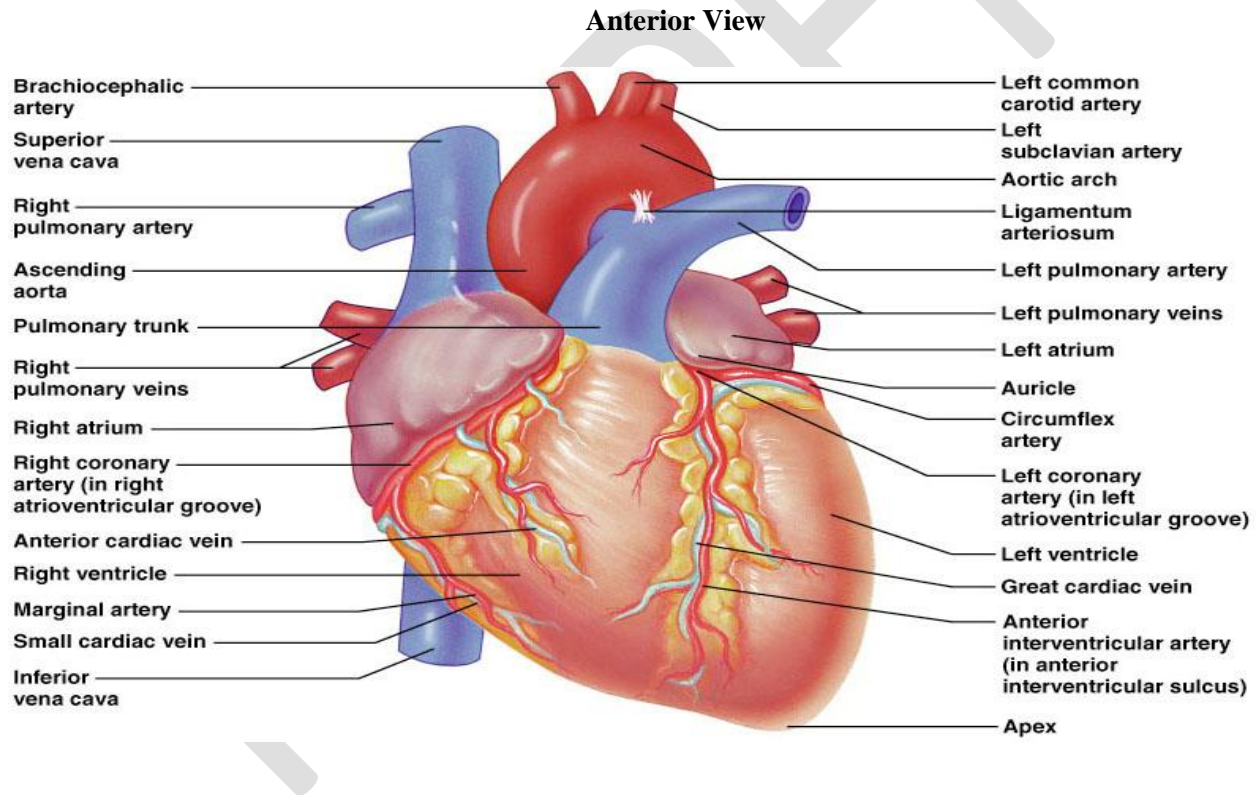
- **Pericardium** – a double-walled sac around the heart composed of:
 1. A superficial fibrous pericardium
 2. A deep two-layer serous pericardium
 - a. The parietal layer lines the internal surface of the fibrous pericardium
 - b. The visceral layer or epicardium lines the surface of the heart
 - c. They are separated by the fluid-filled pericardial cavity
- The Function of the Pericardium:
 1. Protects and anchors the heart
 2. Prevents overfilling of the heart with blood
 3. Allows for the heart to work in a relatively friction-free environment

Heart Wall

- **Epicardium** – visceral layer of the serous pericardium
- **Myocardium** – cardiac muscle layer forming the bulk of the heart
- **Fibrous skeleton** of the heart – crisscrossing, interlacing layer of connective tissue
- **Endocardium** – endothelial layer of the inner myocardial surface

External Heart: Major Vessels of the Heart

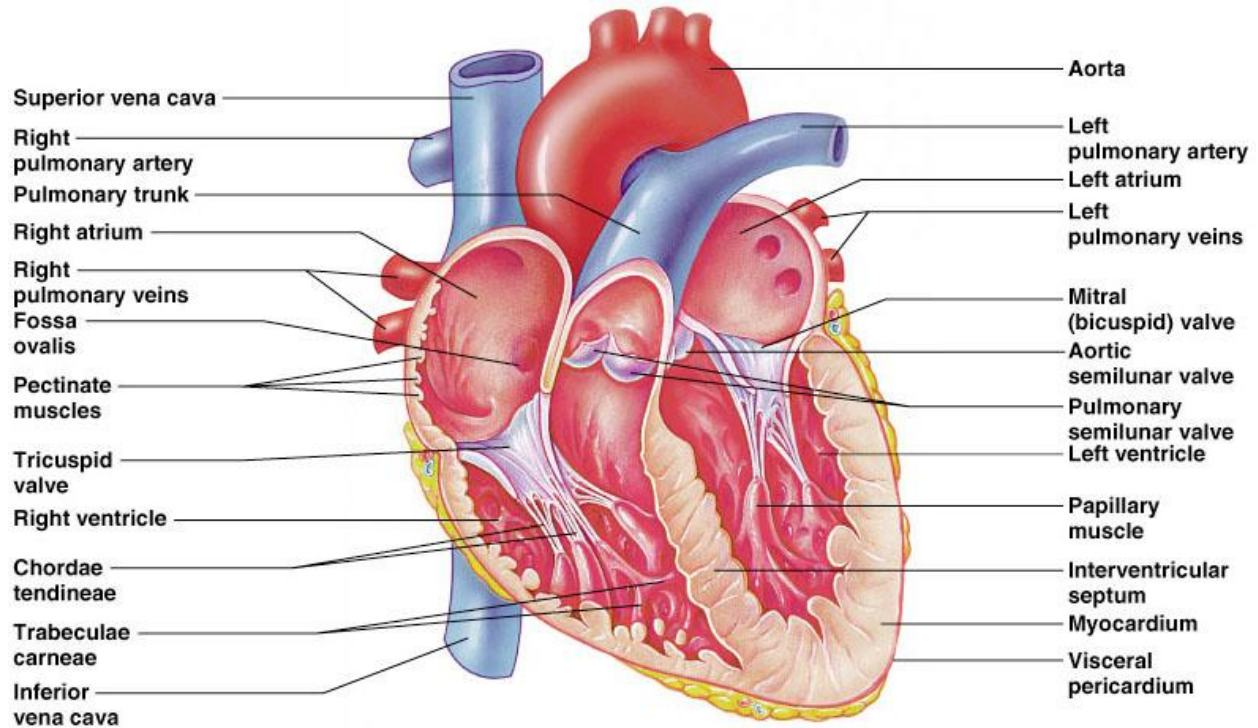
- Vessels **returning blood to the heart** include:
 1. Superior and inferior venae cavae
 2. Right and left pulmonary veins
- Vessels conveying **blood away from the heart** include:
 1. Pulmonary trunk, which splits into right and left pulmonary arteries
 2. Ascending aorta (three branches) –
 - a. Brachiocephalic
 - b. Left common carotid
- Subclavian arteries
- **Arteries** – right and left coronary (in atrioventricular groove), marginal, circumflex, and anterior interventricular arteries
- **Veins** – small cardiac, anterior cardiac, and great cardiac veins

**External Heart: Major Vessels of the Heart (Posterior View)**

- Vessels **returning blood to the heart** include:
 1. Right and left pulmonary veins
 2. Superior and inferior venae cavae

- Vessels conveying blood away from the heart include:

1. Aorta
2. Right and left pulmonary arteries



Atria of the Heart

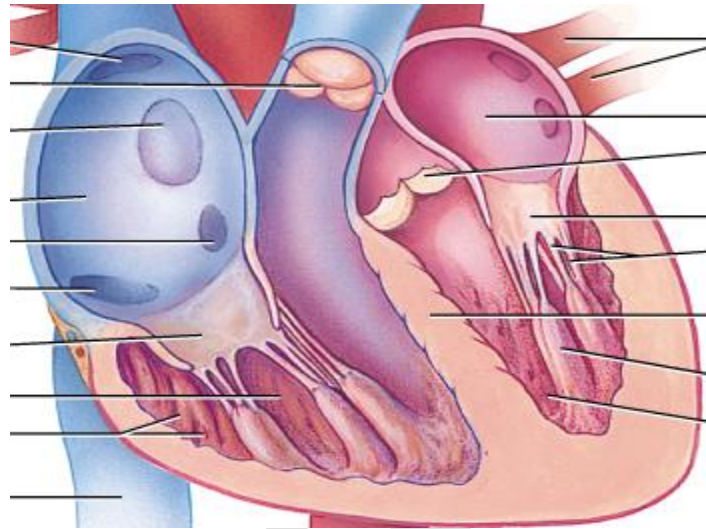
- Atria are the receiving chambers of the heart
- Each atrium has a protruding auricle
- **Pectinate muscles** mark atrial walls
- Blood enters right atria from superior and inferior venae cavae and coronary sinus
- Blood enters left atria from pulmonary veins

Ventricles of the Heart

- Ventricles are the discharging chambers of the heart
- **Papillary muscles** and **trabeculae carneae** muscles mark ventricular walls

- **Right ventricle** pumps blood into the pulmonary trunk
- **Left ventricle** pumps blood into the aorta

Myocardial Thickness and Function

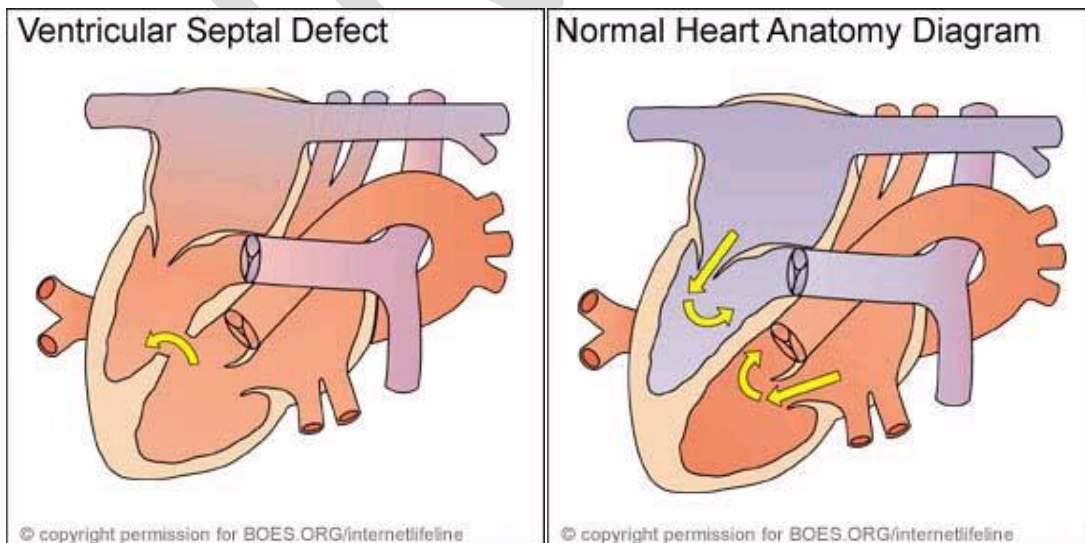


Thickness of myocardium varies according to the function of the chamber

Atria are thin walled, deliver blood to adjacent ventricles

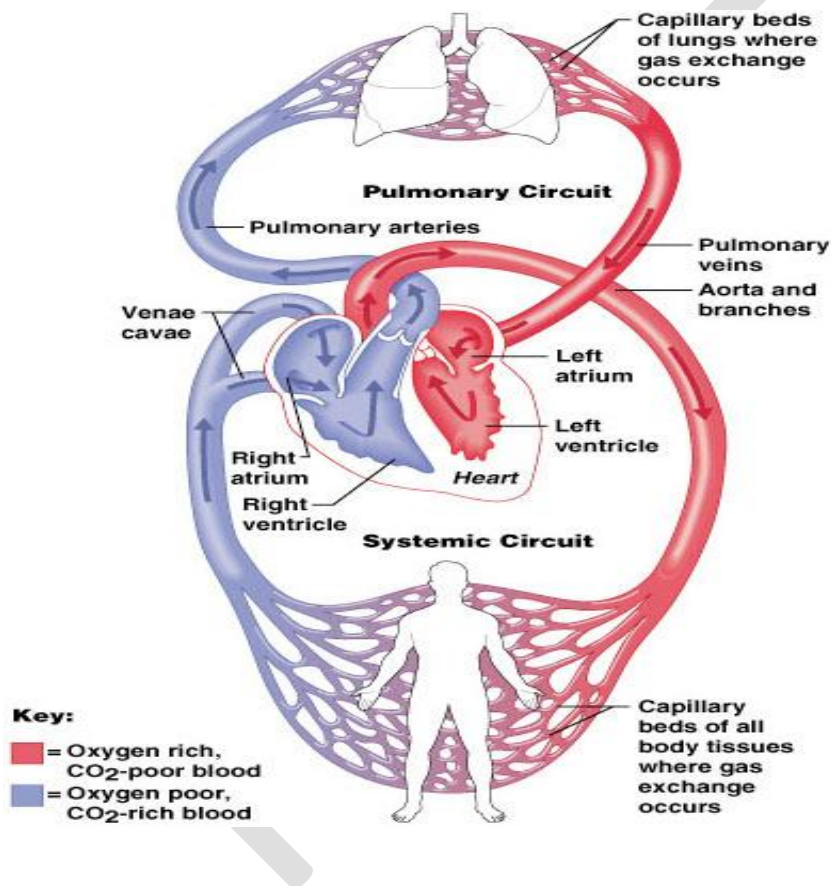
Ventricle walls are much thicker and stronger

- right ventricle supplies blood to the lungs (little flow resistance)
- left ventricle wall is the thickest to supply systemic circulation



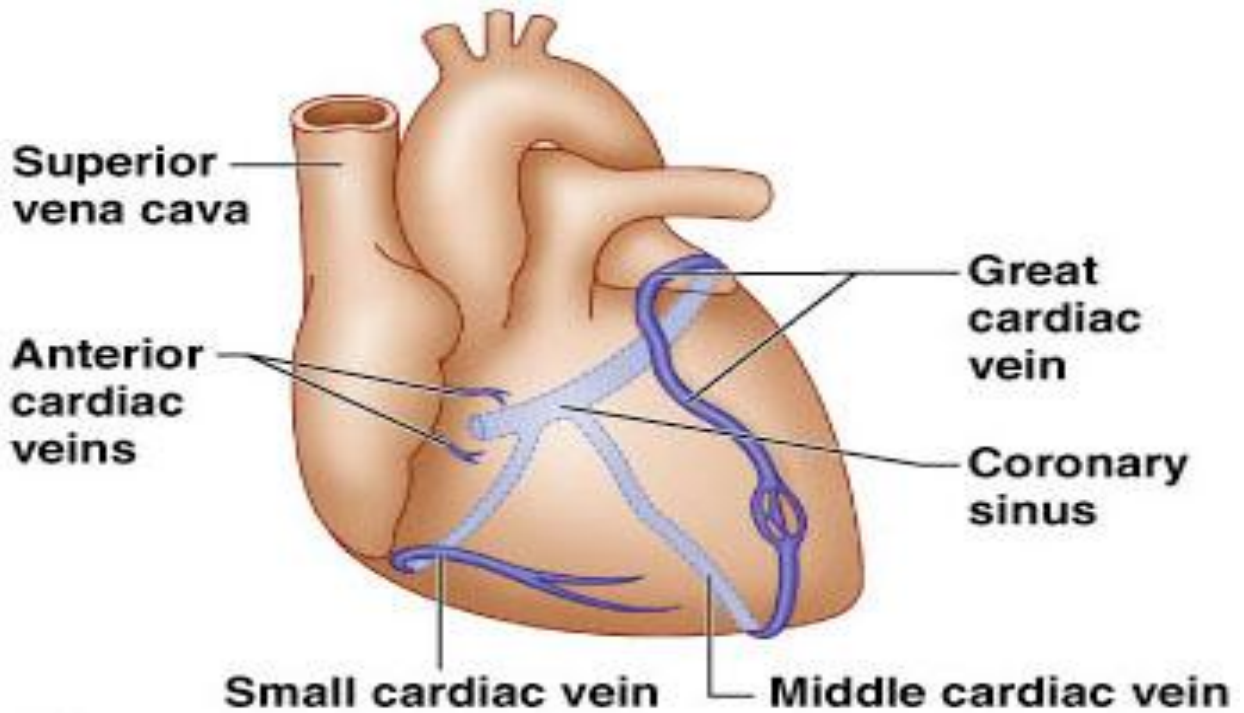
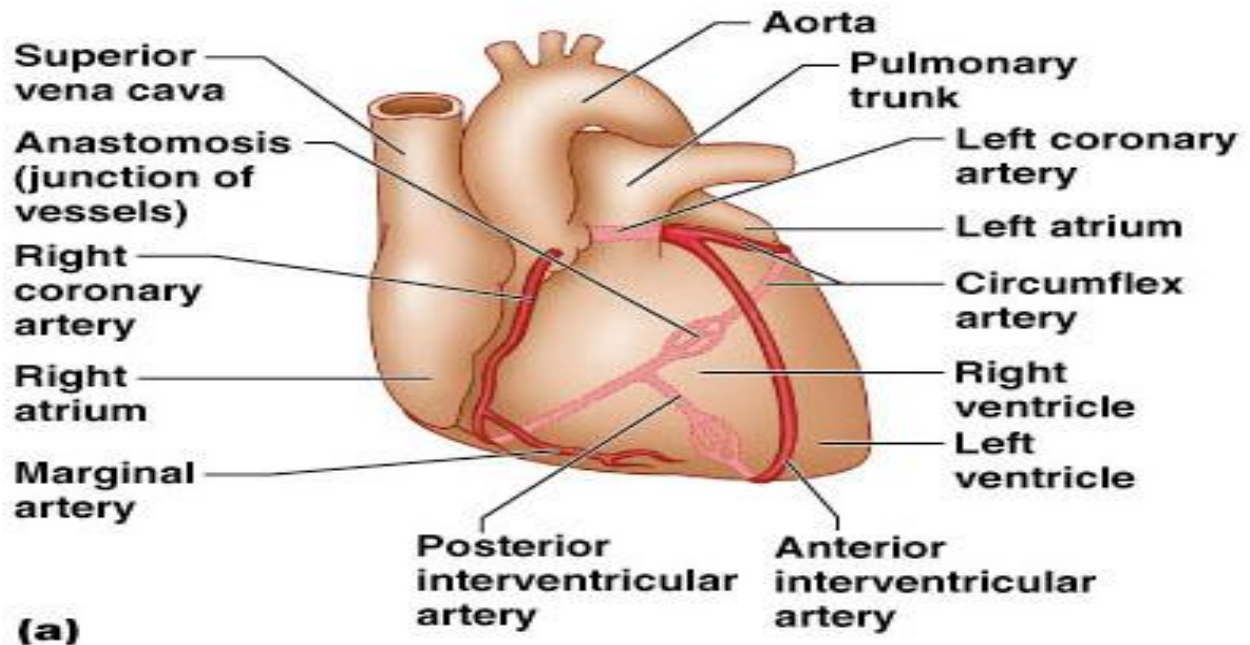
Pathway of Blood Through the Heart and Lungs

- Right atrium → tricuspid valve → right ventricle
- Right ventricle → pulmonary semilunar valve → pulmonary arteries → lungs
- Lungs → pulmonary veins → left atrium
- Left atrium → bicuspid valve → left ventricle
- Left ventricle → aortic semilunar valve → aorta
- Aorta → systemic circulation



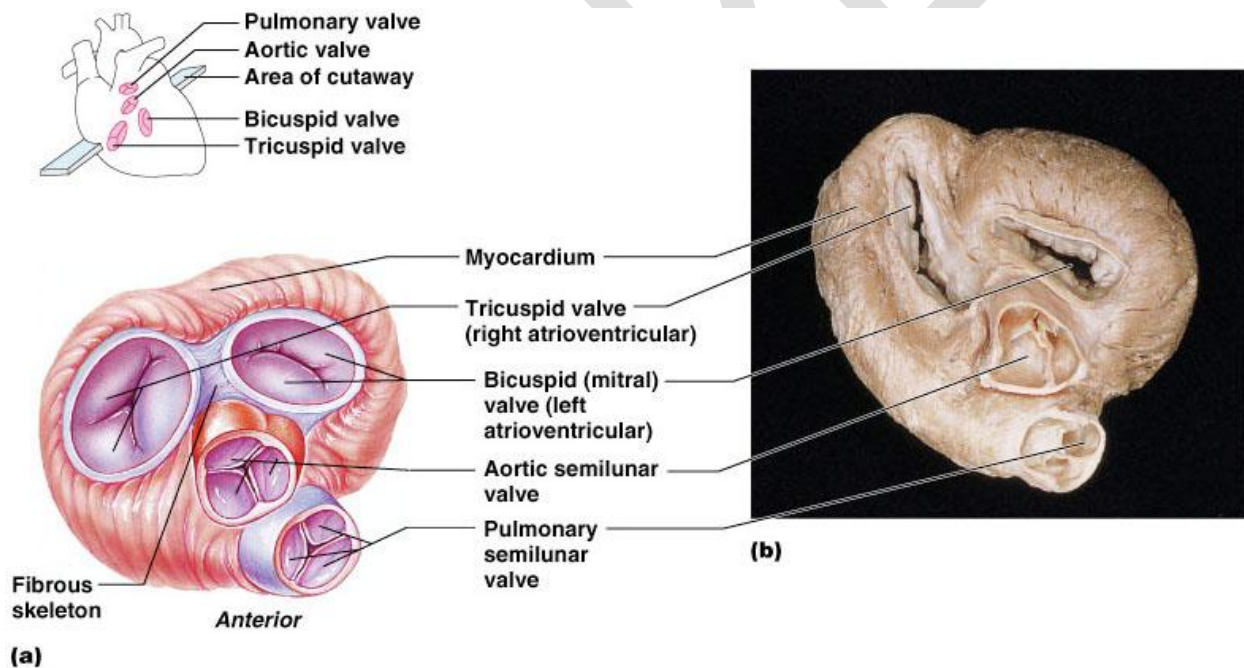
Coronary Circulation

- **Coronary circulation** is the functional blood supply to the heart muscle itself
- **Collateral routes** ensure blood delivery to heart even if major vessels are occluded



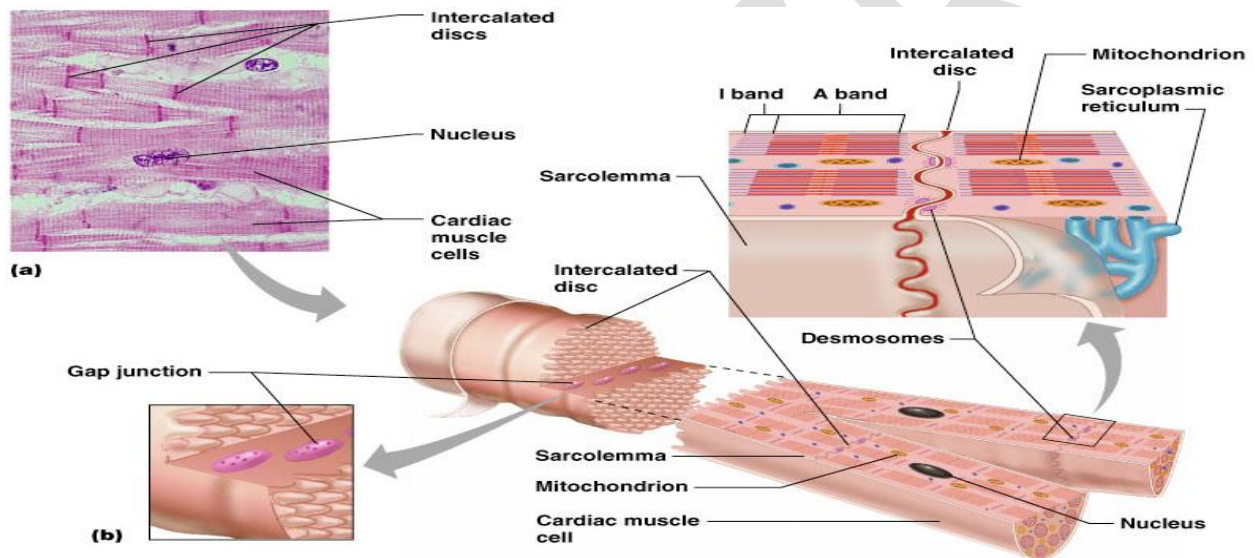
Heart Valves

- Heart valves ensure unidirectional blood flow through the heart
- **Atrioventricular (AV)** valves lie between the atria and the ventricles
 - AV valves prevent backflow into the atria when ventricles contract
- Chordae tendineae anchor AV valves to papillary muscles
- Semilunar valves prevent backflow of blood into the ventricles
- **Aortic semilunar** valve lies between the left ventricle and the aorta
- **Pulmonary semilunar** valve lies between the right ventricle and pulmonary trunk



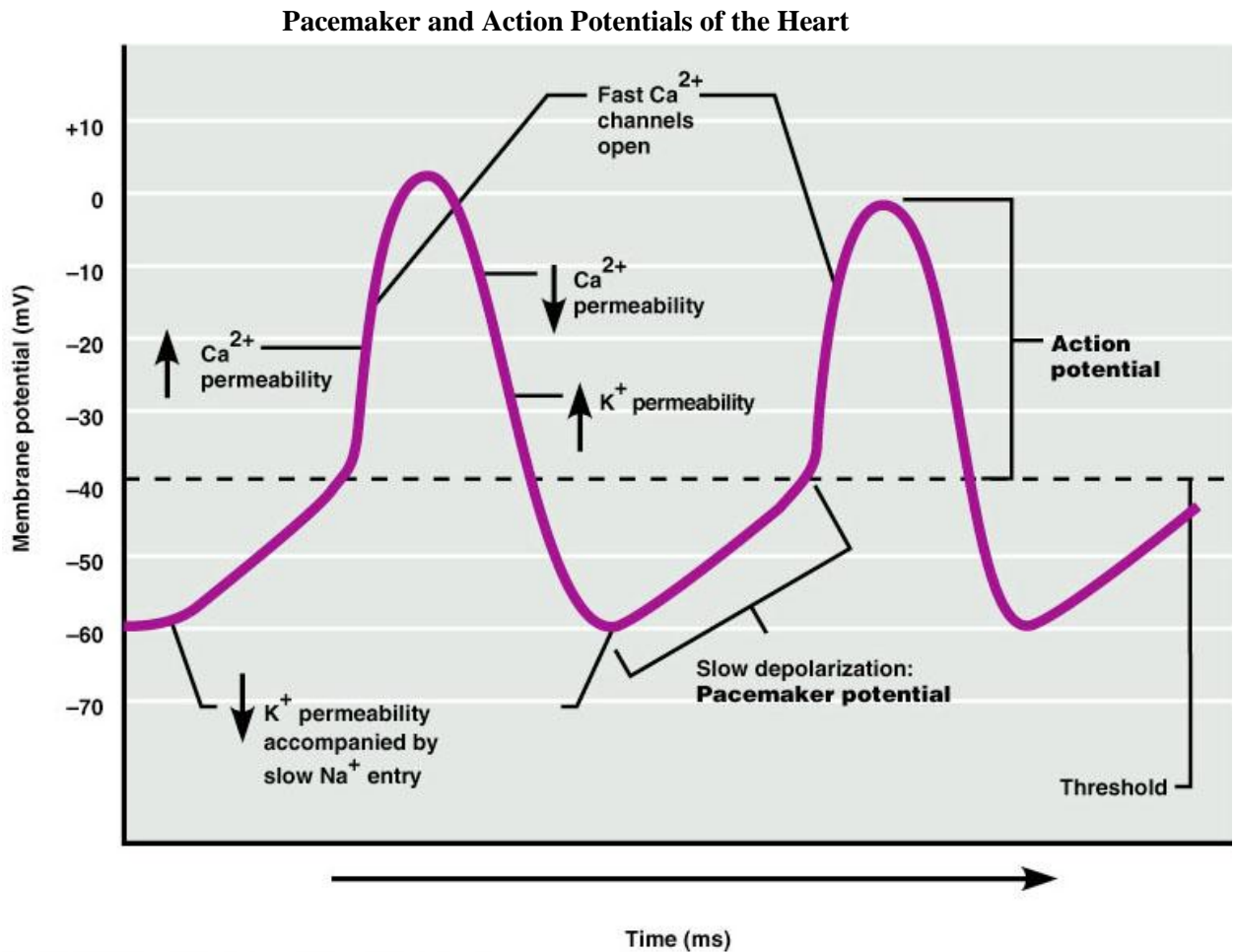
Microscopic Anatomy of Heart Muscle

- Cardiac muscle is striated, short, fat, branched, and interconnected
- The connective tissue endomysium acts as both tendon and insertion
- Intercalated discs anchor cardiac cells together and allow free passage of ions
- Heart muscle behaves as a functional syncytium



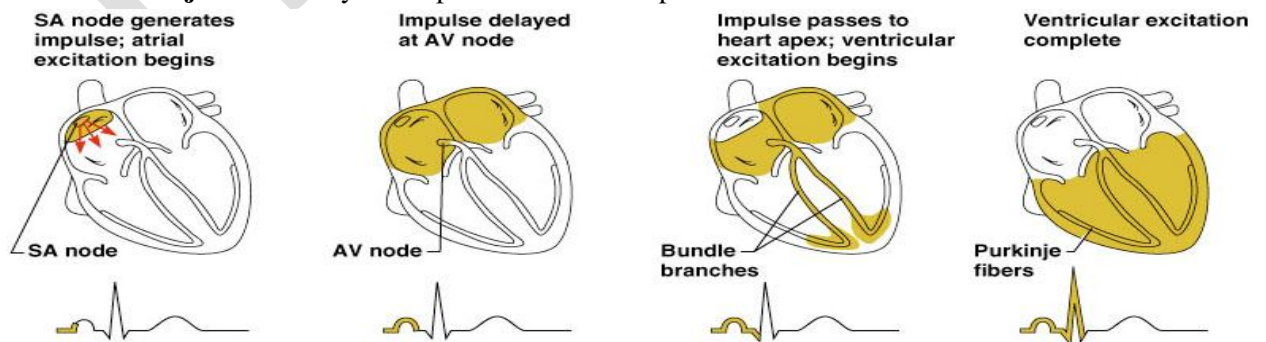
The Cardiovascular System: The Heart Physiology

- Heart muscle:
 - Is stimulated by nerves and is self-excitabile (**automaticity**)
 - Contracts as a unit
 - Has a long (250 ms) absolute refractory period
- Cardiac muscle contraction is similar to skeletal muscle contraction
- **Autorhythmic cells:**
 - Initiate action potentials
 - Have unstable resting potentials called **pacemaker potentials**
 - Use **calcium influx** (rather than sodium) for rising phase of the action potential



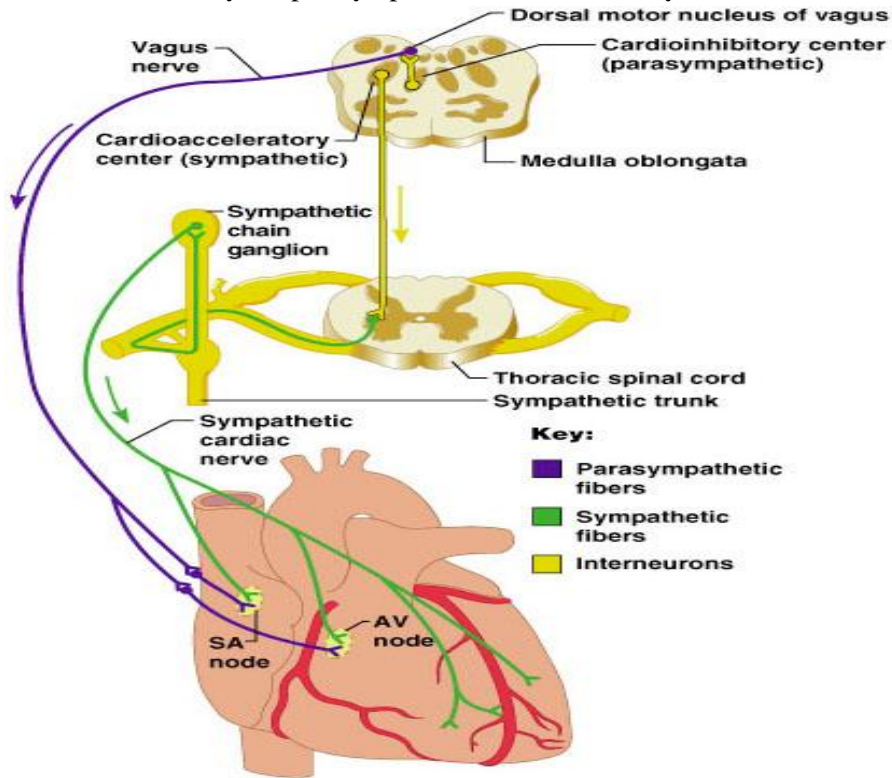
Heart Physiology: Sequence of Excitation:

- **Sinoatrial (SA) node** generates impulses about 75 times/minute
- **Atrioventricular (AV) node** delays the impulse approximately 0.1 second
- Impulse passes from atria to ventricles via the **atrioventricular bundle (bundle of His)**
 - AV bundle splits into two pathways in the interventricular septum (bundle branches)
 - **Bundle branches** carry the impulse toward the apex of the heart
 - **Purkinje fibers** carry the impulse to the heart apex and ventricular walls



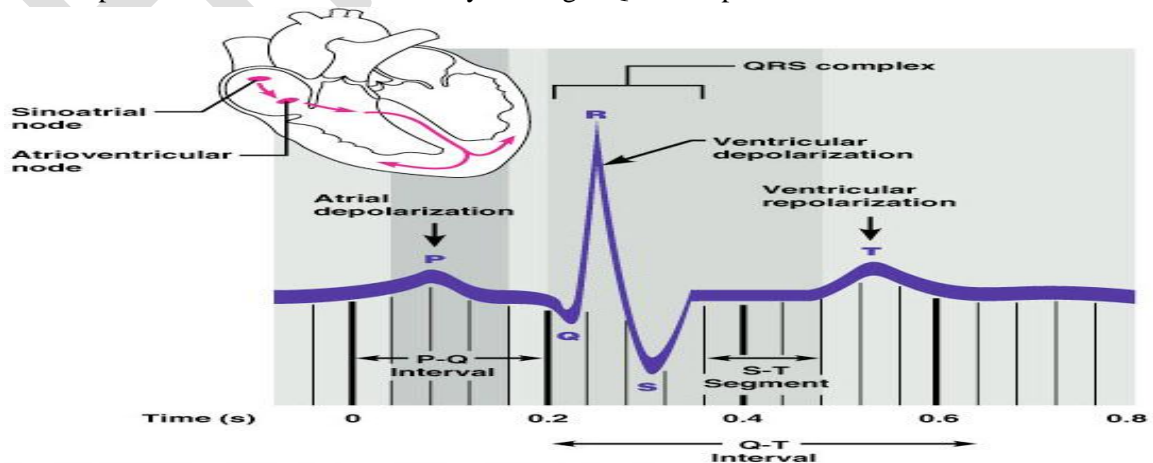
Extrinsic Innervation of the Heart

- Heart is stimulated by the sympathetic cardioacceleratory center
- Heart is inhibited by the parasympathetic cardioinhibitory center



Electrocardiograph

- Electrical activity is recorded by electrocardiogram (ECG)
- P wave corresponds to depolarization of SA node
- QRS complex corresponds to ventricular depolarization
- T wave corresponds to ventricular repolarization
- Atrial repolarization record is masked by the larger QRS complex

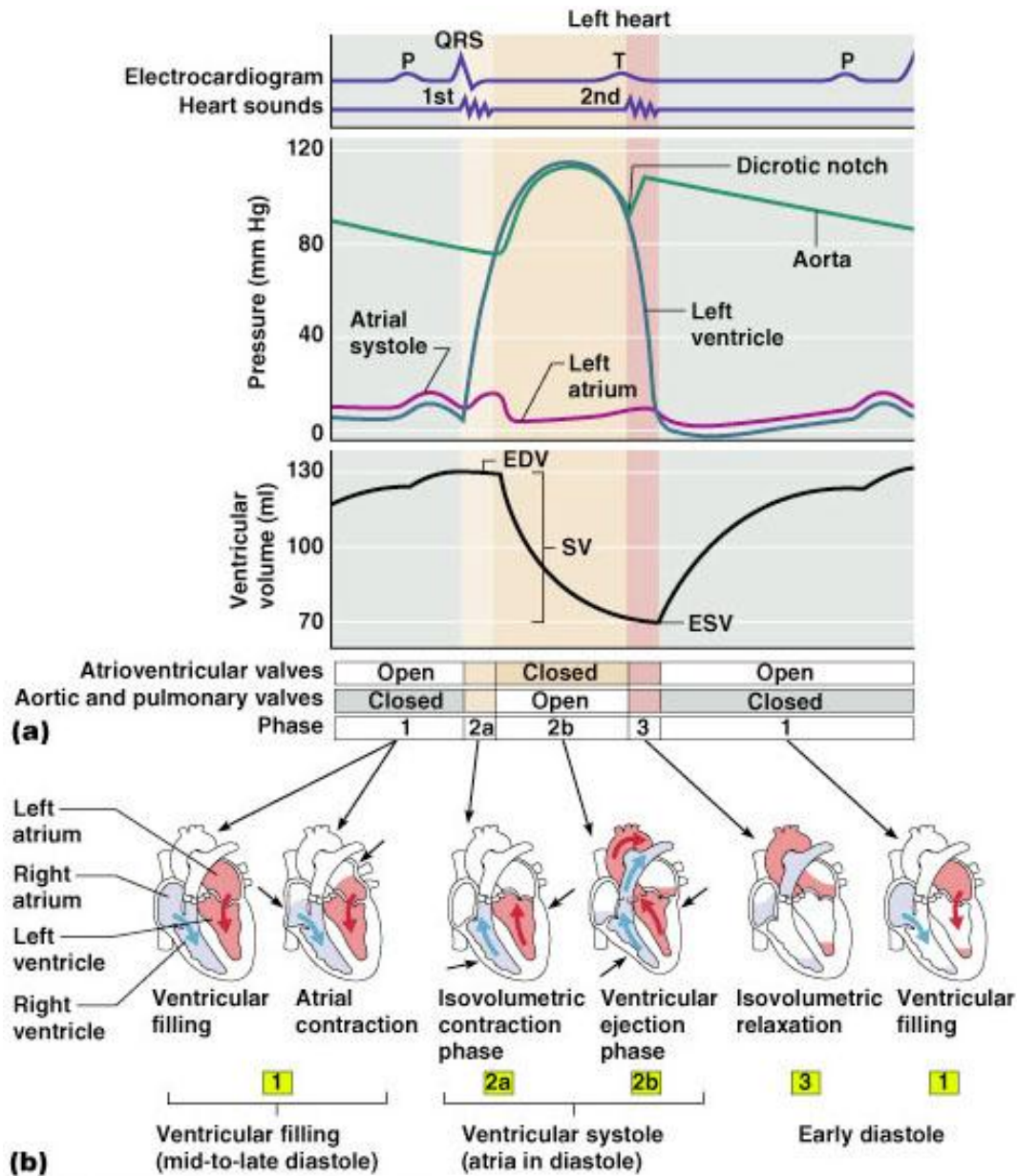


Heart Sounds:

- Heart sounds (lub-dup) are associated with closing of heart valves
 - **First sound** occurs as AV valves close and signifies beginning of systole (contraction)
 - **Second sound** occurs when SL valves close at the beginning of ventricular diastole (relaxation)

Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart
 - **Systole** – contraction of heart muscle
 - **Diastole** – relaxation of heart muscle
- **Ventricular filling** – mid-to-late diastole
 - Heart blood pressure is low as blood enters atria (passively) and flows into ventricles
 - AV valves are open, then atrial systole occurs
- **Ventricular systole (contraction)**
 - Atria relax
 - Rising ventricular pressure results in closing of AV valves
 - *Isovolumetric contraction* phase
 - Ventricular ejection phase opens semilunar valves
- **Isovolumetric relaxation** – early diastole
 - Ventricles relax
 - Backflow of blood in *aorta and pulmonary trunk* closes semilunar valves
- **Dicrotic notch** – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves



Cardiac Output (CO) and Reserve

- **Cardiac Output** is the amount of blood pumped by each ventricle in one minute
 - CO is the product of heart rate (HR) and stroke volume (SV)
 - HR is the number of heart beats per minute

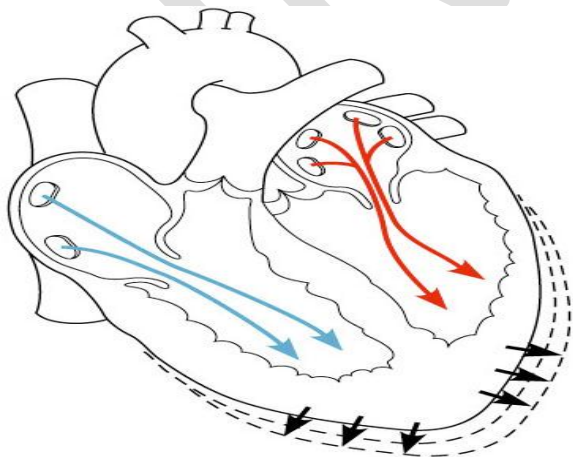
- SV is the amount of blood pumped out by a ventricle with each beat
- **Cardiac reserve** is the difference between resting and maximal CO
- $CO \text{ (ml/min)} = HR \text{ (75 beats/min)} \times SV \text{ (70 ml/beat)}$
- $CO = 5250 \text{ ml/min (5.25 L/min)}$

Regulation of Stroke Volume

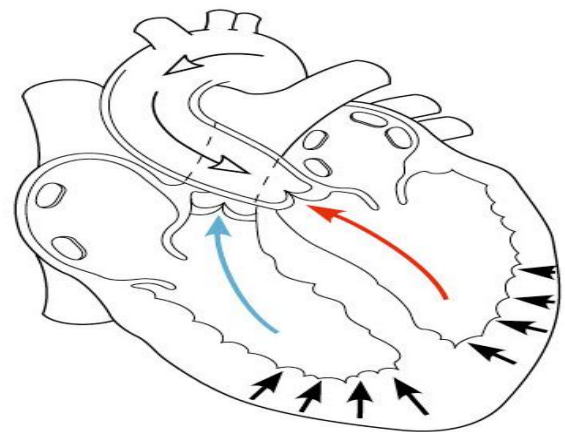
- $SV = \text{end diastolic volume (EDV)} - \text{end systolic volume (ESV)}$
 - EDV = amount of blood collected in a ventricle during diastole
 - ESV = amount of blood remaining in a ventricle after contraction

Factors Affecting Stroke Volume

- **Preload** – amount ventricles are stretched by contained blood
- **Contractility** – cardiac cell contractile force due to factors other than EDV
- **Afterload** – back pressure exerted by blood in the large arteries leaving the heart
- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume
- Slow heartbeat and exercise increase venous return to the heart, increasing SV
- Blood loss and extremely rapid heartbeat decrease SV



(a) Preload



(b) Afterload

Extrinsic Factors Influencing Stroke Volume

- **Contractility** is the increase in contractile strength, independent of stretch and EDV
- Increase in contractility comes from:
 - Increased sympathetic stimuli
 - Certain hormones
 - Ca^{2+} and some drugs
- Agents/factors that decrease contractility include:
 - Acidosis
 - Increased extracellular K^+
 - Calcium channel blockers
- **Sympathetic** stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system

Regulation of Heart Rate

- Positive chronotropic factors increase heart rate
 - Caffeine
- Negative chronotropic factors decrease heart rate
 - Sedatives
 - **Sympathetic nervous system (SNS)** stimulation is activated by stress, anxiety, excitement, or exercise
 - **Parasympathetic nervous system (PNS)** stimulation is mediated by acetylcholine and opposes the SNS
 - PNS dominates the autonomic stimulation, slowing heart rate and causing vagal tone
 - If the Vagus Nerve was cut, the heart would lose its tone. Thus, increasing the heart rate by 25 beats per minute.

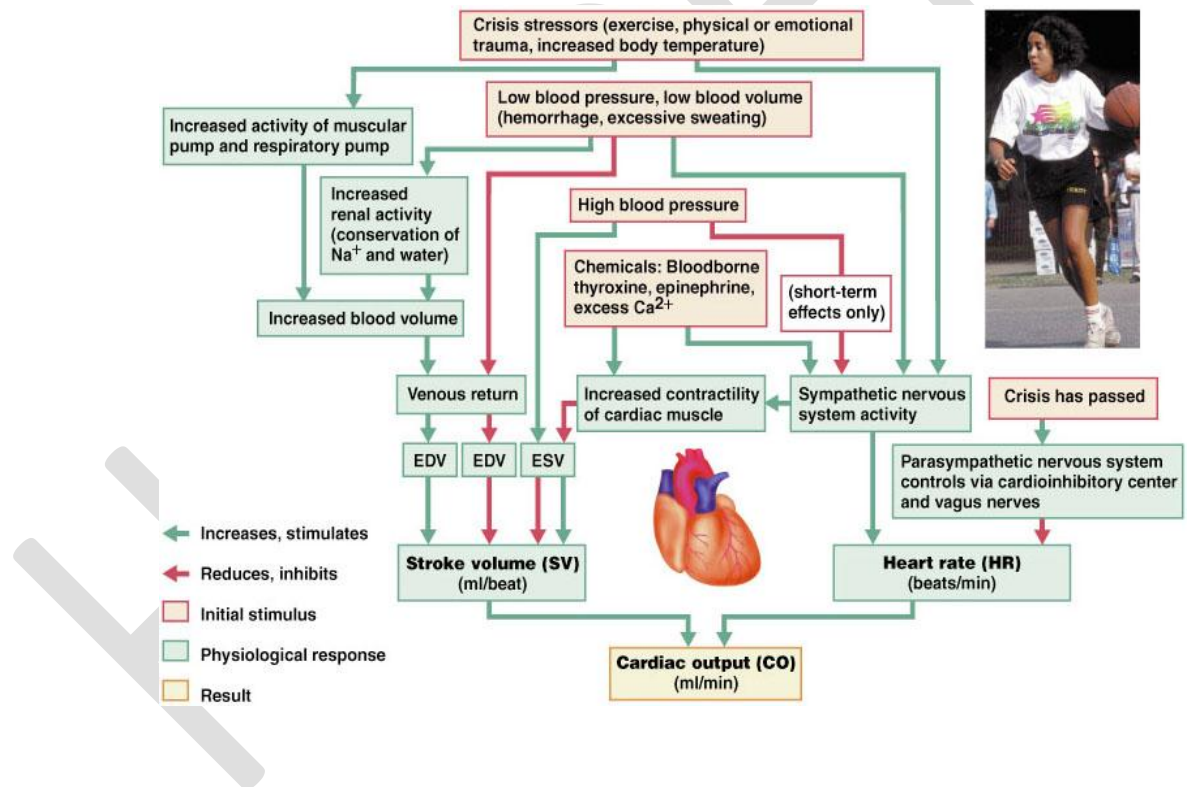
Atrial (Bainbridge) Reflex

- **Atrial (Bainbridge) reflex** – a sympathetic reflex initiated by increased blood in the atria
 - Causes stimulation of the SA node
 - Stimulates baroreceptors in the atria, causing increased SNS stimulation

Chemical Regulation of the Heart

- The hormones epinephrine and thyroxine increase heart rate
- Intra- and extracellular ion concentrations must be maintained for normal heart function

Factors Involved in Regulation of Cardiac Output



Congestive Heart Failure (CHF)

- Congestive heart failure (CHF) is caused by:
 - Coronary atherosclerosis
 - Persistent high blood pressure

- Multiple myocardial infarcts
- Dilated cardiomyopathy (DCM) – main pumping chambers of the heart are dilated and contract poorly
- Causes of CHF
 - coronary artery disease, hypertension, MI, valve disorders, congenital defects
- **Left side heart failure**
 - less effective pump so more blood remains in ventricle
 - heart is overstretched & even more blood remains
 - blood backs up into lungs as pulmonary edema
 - suffocation & lack of oxygen to the tissues
- **Right side failure**
 - fluid builds up in tissues as peripheral edema
- **MI = myocardial infarction**
 - death of area of heart muscle from lack of O₂
 - replaced with scar tissue
 - results depend on size & location of damage
- **Blood clot**
 - use clot dissolving drugs streptokinase or t-PA & heparin
 - balloon angioplasty
- **Angina pectoris**
 - heart pain from ischemia (lack of blood flow and oxygen) of cardiac muscle

Artificial Heart

